Head Injury (GENERAL)

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1. Motor vehicle accidents - most common cause! (≥ 50%) (much more common in suburban/rural areas) – 70% MVA injuries are TBIs.

DEFINITIONS, CLASSIFICATIONS

Traumatic brain injury (TBI) - non-congenital, nontraumatic brain insult from acute external mechanical force, with associated altered state of consciousness, and temporary or permanent impairments of cognitive, physical, psychosocial functions.

“HEAD INJURY” and “BRAIN INJURY” are not identical, but cannot be easily separated. N.B. HEAD INJURY may not be associated with neurological deficits!

Scalp Injuries
C. closed – contusion.

OPEN:
1) puncture
2) laceration
3) avulsion

Skull fractures
see p. Rh5 >>

Brain injury (TBI)
Communication with outside:

A. CLOSED:
1) no scalp injury
2) no skull fracture
3) scalp injury not connected with skull fracture.
   • caused by blunt objects or no contact.

B. OPEN – scalp injury connected to skull fracture; Risk of infection!
   • caused by blunt or sharp objects.

Penetrating – with data matter injury;
   • caused by missile injuries (much more common) or impalements.

Location:
A. Diffuse:
   a) concussion (s. commotio) – mildest TBI with negative imaging.
   b) diffuse axonal injury (DAI) – microhemorrhages on imaging

B. Focal:
   a) contusions
   b) lacerations
   c) hematomas (extradural, subdural, subarachnoid, intracerebral)

Cerebrovascular injury

EPIEMIOLOGY

Brain injury – plague of modern society – despite technological progress (carriages → powerful cars; fist fighting → shootings), innate man’s aggression is not curbed.

INCIDENCE

0.2% per year (in USA, head injury occurs every 7 seconds and death every 5 minutes)

High-risk populations:

1) young people (peak 15-24 yrs. second peak > 65 yrs - due to falls)
2) men (affected 2-4 times* as often as women; approaches 1:1 as age increases - increased likelihood of TBI caused by falls)
   *motor vehicle accidents, contact sports, interpersonal violence, alcohol abuse
3) low-income individuals
4) unmarried individuals
5) members of ethnic minority groups (esp. African Americans, Native Americans)
6) residents of inner cities
7) individuals with history of substance / alcohol abuse
8) individuals with previous TBI

Morbidity

• 20-25% brain trauma cases need hospitalization
• major cause of disability!
• 100% severe TBI, 66% moderate TBI → permanent disability.
• 5-10% patients go to long-term care facility.

Mortality

• mortality ≥ 10% (higher in persons 15-24 yrs and > 65 yrs). Major cause of death in young adults!
• 5% of all patients die at site of accident (60% of fatalities occur before patients can be admitted to hospital).
   also see “Prehospital Management” below
   N.B. in gunshot TBI, mortality is 90-92% (73-76% are dead at scene; of remaining, 60% die at hospital)

50,000 individuals each year die from head injuries in USA

according to TBI severity:
1) mild TBI - mortality = 0%
2) moderate TBI - mortality = 2.5-20%
3) severe TBI - mortality = 30-50%
• if patient dies at hospital, average time to death is 2 days after trauma.

TRAUMAS IN GENERAL:
– TBI is major determinant of survival in most cases of blunt trauma.
– TBI contributes significantly for 50-75% of all traumatic deaths.
– in any traumatic cases, presence of TBI increases fatality rate 3-fold.

etiology
II. NTRACRANIAL DISORDER (due to SAH) → subarachnoid hemorrhage (esp. in child with hydrocephalus and subarachnoid hemorrhage from suicide attempt) → high brain stem compression, intoxication with alcohol / CNS depressants.

1. Head Injury (general)

II. Intracranial Disorders

1. Expanding intracranial mass (hematoma, contusions, brain edema & engorgement) → raised ICP → decrease in cerebral perfusion pressure, herniation. Cause 50% deaths!
2. Seizure
3. Vasospasm (due to SAH) → local ischemia. N.B. SAH is most common type of traumatic intracranial hemorrhage!

Very dangerous combination: ICP↑ + hypotension
N.B. injured brain is very sensitive to perfusion fluctuations – autoregulation limits become narrower (e.g. the lower limit for autoregulation may rise from 40 mmHg to 70 mmHg – here is rationale to keep CPP > 60-70 mmHg to prevent ischemia).

3. immediately after trauma, blood vessel loss and passively dilate (impaired autoregulation) → cerebral blood flow* (although metabolic demands and oxygen consumption are diminished) → ICP?

3:50 min later: postcapillary sphincters and venules constrict but arterioles remain paralyzed (maximally dilated) → cerebral blood flow* (typically less than half of normal values), cerebral blood volume* (brain engorgement) → ischemia, ICP?.

– lowest CBF values occur within first 6-12 hours after injury.
– these changes may exist for several days after injury.
– it is especially common in children.

N.B. despite disrupted autoregulation, inactivity to Pre remains (enables therapeutic hyperventilation).

Duration of cerebral autoregulation impairment significantly correlates with worse outcomes after severe TBI!


– mechanical forces and ischemia disrupt BBB for several hours (demonstrated by contrast MRI) → vasogenic brain edema → ICP?; 
– ischemia (produced by any mechanism) causes cytotoxic brain edema.

In TBI, both vasogenic and cytotoxic brain edema occur!*

PATHOPHYSIOLOGY (Biochemistry)

– immediately after trauma, bioelectrical brain activity stops → widespread neuron depolarization → release of excitatory neurotransmitters (glutamate, aspartate).

– excess neuronal firing → K- leaves cells, Na+ accumulates intracellularly → acute neuronal swelling.
– intracellular Cs+?* → generation of oxygen free radicals → membrane damage.
– metabolic activity? to restore ionic balance → release of lactate → acidosis
– excitatory neurotransmitters, lactate, oxygen free radicals are released into bruised / ischemic areas → intense inflammatory response → further brain edema.

PATHOLOGY, CLINICAL FEATURES

Head injuries and their sequelae are embedded inextricably in medicolegal system - detailed history, review of systems, complete examination, and management steps are essential - documentation should be meticulous!

External signs of trauma (scalp lacerations, abrasions, hematomas, bruising, etc) at site of impact - only confirm that injury has occurred; not always present in patient who has sustained serious brain damage!

N.B. as scalp injuries occur at site of impact - carefully explore for foreign bodies or underlying skull fractures.

– in punctured injuries, carefully document entry and exit wounds, powder burns, and foreign bodies.
– scalp may be injured with or without breach in its surface.
– injured scalp becomes markedly edematous.

Lacerations are particularly common, as scalp is readily crushed and split against underlying bone (most scalp lacerations are linear because of mechanical forces and ischemia)

scalp may be injured with or without breach in its surface.
– injured scalp becomes markedly edematous.
– lacerations are particularly common, as scalp is readily crushed and split against underlying bone (most scalp lacerations are linear because of scalp convexity).
– scalp subcutaneous layer has rich vascular supply → significant blood loss when scalp is lacerated.
– galacta is poorly fixated to underlying perosteum → large scalp flaps (scalping or degloving injuries), little resistance to hemotoma / abscess formation in subgaleal plane.

N.B. external bleeding or subgaleal blood collections may cause shock in small infant!

Alteration of consciousness (practically the most symptom for TBI!?) → inadequate functioning of brainstem or both cortices caused by:

a) primary injury (e.g. midbrain or diencephalic hemorrhages, diffuse axonal injury)
– b) ischemia (due to ICP?)
– c) hypotension
– d) hypoxia
– e) hypoglycemia

f) intoxicating substance consumed before injury → generation of excitotoxic substances (e.g. glutamate, aspartate)

Biochemistry leads cells, Na+ leaves cells, K- accumulates intracellularly → acute neuronal swelling.

The pathophysiology of brain trauma is characterized by the following events.

1) immediate brain generalized convulsion (“impact seizure”) - result from transient mechanical and neurochemical changes - most of these patients will have no additional seizures (*) and do not require long-term anticonvulsants (but require anticonvulsants for first 7 days)

* other authors state - single seizure at time of injury increases risk of post-traumatic epilepsy 10-fold
2) transient apnea, flaccidity, areflexia, dilatation of pupils (esp. in children).
3) arterial hypertension, cardiac arrest (in absence of overwhelming brain damage, recovery from arrest is rule).

RESIDUAL EFFECTS (on recovering consciousness)

1. Dizziness, nausea, emesis (common in immediate posttraumatic period, regardless of TBI degree).
2. Slight blurring of vision (transient cortical blindness may follow concussion - localized edema or vasospasm in calcarine fissure; usually resolves spontaneously within 24 hours).
3. Difficulties with concentration, mental cloudiness and confusion after full consciousness is restored (this “mental” period is prolonged, roughly proportional to degree of brain injury).
some patients are combative when they regain consciousness. 
behavioral changes (such as agitation) are most evident at night. 
difficulties with activities of daily living may continue for months. 

4. Amnesia 

(1) Anterograde amnesia (amnesia for events after trauma) - somewhat of misnomer - severe inattention in postinjury state primarily prevents retention of new information, ("posttraumatic confusional state" is more accurate term). 
(2) Retrograde amnesia (amnesia for events preceding trauma) - never occurs without anterograde amnesia, may be absent in mild TBI. 
during weeks, improvement occurs in orderly progression from most distant to recent memories (islands of absolute amnesia may remain in severe cases). 

HYSTERICAL POSTTRAUMATIC AMNESIA - tendency to recount events that cannot be recalled on later testing, bizarre affect, forgetting one's own name, excessive anterograde deficit. 

5. Headache (constant generalized or frontal; may be throbbing herniemial like migraine) 

common for days = months following trauma. H β-blockers 
persistent severe headache and repeated vomiting in context of normal alertness and no focal neurologic signs are usually benign. 

6. Early post-traumatic SEIZURES (2.5-7% clinically; 22% by EEG): within 7 days after TBI (50-80% manifest during 4th day as immediate SEIZURES) - results from cerebral edema, hemorrhagic lesions (intracerebral, subdural- epidural hematoma), penetrating injury (42% for seizures), depressed skull fractures. 
seizures are major threat - increase tissue energy requirements and cerebral blood flow by up to 400% → ICP↑. 
STATUS EPILEPTICUS may ensue! 
seizures occurring after 7 days = late post-traumatic SEIZURES (posttraumatic epilepsy) see below 

7. Facial neurologic signs - hemiplegia (9%), aphasia (6%), cranial nerve palsies, etc. - depend on extent and site of damage to intracranial structures. 
(almost) complete return of motor power and speech is 

Injury to CRANIAL NERVES: 
a) frequent complication of skull base fractures 
b) torn or stretched by brain movement within skull (esp. CN1). 
c) penetrating trauma. 

occasionally, cranial nerve palsies may not be evident for several days. 
recovery (partial or complete) is rule, but prognosis is worse for CN1 and CN2. 

CN1 (in 7% TBI cases) - anosmia and apparent loss of taste (actually loss of perception of aromatic flavors, with elementary tastes retained) occurs in 10-30% of severe TBI cases (esp. with falls on back of head or anterior fossa fractures) - results from brain displacement → shearing of olfactory filaments at cribiform plate; 
even trivial head injury (to any part of head) can result in anosmia! 
check for CSF rhinorrhea and frontotemporal contusions. 
recovery may last up to 5 years, residual hypoaesthesia is usual. 

CN2 (in 5% TBI cases): fractures of sphenoid bone may transect CN2; closed TBI → partial injuries (blurring of vision, central or paracentral scotomas, sector deficits); direct orbital injury → reversible iridoplegia (short-lived blurred vision for close objects). 
indirect optic neuropathy: observation, high-dose steroids, surgery. 
delayed onset of visual loss → surgical decompression. 
prognosis extremely variable (0.100%). 

CN3 - injured in uncal herniation. 
aberrant regeneration often occurs (e.g. lid elevation on attempted adduction). 

CN4 - fracture of lesser sphenoid wing or stretching near CNS exit site. 
TBI is most common cause of trochlear palsies! 
only 50-66% recover because of frequent nerve avulsion. 
bilateral CN4 lesions can occur - if dorsal midbrain and both 4th nerves are impacted in nicide of tentorium cerebelli; only 25% recover. 

CN5, CN6 (in facial trauma) 
hyepathpia in nerve distribution may be permanent. 

CN8 - most commonly injured ocularmotor nerve! 

CN7 - petrosus fractures (in 30-50% transverse temporal bone fractures; in 10-30% longitudinal fractures; facial palsy may be delayed 5-7 days (mechanism - progressive edema within nerve - good prognosis) 
longitudinal fractures - spontaneous recovery is usual. 
transverse fractures → nerve decompression. 

CN8 - petrosus fractures (nerve laceration in 80% transverse temporal bone fractures) → sensorineural hearing loss, vertigo, positional nystagmus immediately after injury. 
patients with low- or high-frequency hearing loss may have some recovery but those with low- and high-frequency loss usually do not recover. 
vertigo due to labyrinth concussion usually resolves within year. 

CN9-12 - fracture of occipital condyle; COLLET-SICARD syndrome. 

MIDDLE EAR TRAUMA → tympanic perforation, hemotympanum, ossicular disruption → conductive hearing loss. 
most tympanic perforations heal spontaneously, see p. Ear 38-39. 
ossicular incongruences → ossiculoplasty (if hearing loss persists > 3 months). 
INNER EAR TRAUMA 
1) cochlear concussion → sensorineural hearing loss (for high-tones). 
2) perilymphatic fistula → sensorineural hearing loss 
3) otolithic dislocation →benign paroxysmal positional vertigo 

CONCUSSION 
- biomechanically induced alteration of brain function, typically affecting memory and orientation. 
it's clinical diagnosis: 
1) immediate brief (≤ 6 hours) loss of consciousness* (dazed or "star struck"); loss of consciousness is not deep - pupillary reactions and other cranial functions are intact; extensor plantar responses may be present briefly but not decerebrate posturing).
The only objective signs of concussion (to rule out malingering) – but only within first 48 hrs:
1. Asymmetry of corneal reflexes
2. Horizontal nystagmus (may be due to alcohol intoxication!)
3. Abnormal vestibular reflexes (\( \leq 1 \) asymmetry).

• mechanism of loss of consciousness - functional disconnection of brain stem from cerebral hemispheres.

**transient electrophysiologic dysfunction of RAS** in upper midbrain caused by rotation of cerebral hemispheres on relatively fixed brainstem.

High primates are particularly susceptible to concussion; in contrast, billy goats, rams, and sheepheads can tolerate impact velocity and deceleration \( \geq 200 \) times greater than humans.

• mechanism of amnesia is not known.

• no immediate or delayed structural brain damage - no significant long-term sequelae (except postconcussion syndrome).

• biochemical and ultrastructural changes exist - that lead to traumatic encephalopathy — having ApoE-4 gene increases risk for chronic problems following concussion.

**DIFFUSE AXONAL INJURY (DAI)**

- immediate loss of consciousness lasting > 6 hours (i.e. concussion is a mild form of DAI).
- unconsciousness in resuscitated patient despite absence of any intracranial mass lesion or history of hypoxia.

N.B. D.AI is almost ubiquitous to all patterns / degrees of TBI.

• extensive generalized shearing or stretching of axons within white matter - caused by ACCELERATION / DECELERATION injury – rotational or angular forces*, but not translational forces.

* e.g. brain rotation within skull.

• N.B. magnitude of acceleration needed to produce DAI requires head to strike object (increased likelihood that DAI will be accompanied by other intracranial lesions)

• axons are either sheared off (immediate impact or degenerate soon after)
- stretch injury first affects nodes of Ranvier (blebbing of nodal axolemma).
- membrane channels open to admit toxic levels of calcium.
- numerous swollen and disconnected axons (axon retraction bulbs) throughout white matter - appear within hours of injury.

• same forces act on vessels \( \rightarrow \text{intracerebral hemorrhages (micro / macro)}. \)

Duret hemorrhages - punctate hemorrhages caused by small penetrating arteriole stretching:
- also may occur during transtentorial herniation (as secondary injury) – hemorrhages are larger than in DAI.

Subdural hematoma - caused by stretching of subdural veins.

• little cerebral swelling - no ICP! ( \( \geq 7 \) children may develop diffuse cerebral edema).

Coma lasting 6-24+ days - mild DAI (only axon stretching): 10% patients demonstrate decorticate or decerebrate posturing, but by 24 hours they are following commands.

- good prognosis (mild or no permanent disabilities, but some patients die).

Coma lasting > 24 hours - moderate or severe DAI (irreversible axon shearing, hemorrhages).

- persistent brain stem dysfunction (posturing), autonomic dysfunction (e.g. hypertension, hypopryrexia).

- poor prognosis (up to persistent vegetative state or death).

N.B. D.AI is contributing cause of death in 30-40% cases!

All patients with DAI present identically in coma - no early clinical predictor differentiates mild, moderate, or severe DAI! 

**CENTRIPETAL THEORY of A. K. ONSAYA and T.A. GANNONELL (1982)** - increases in rotation / acceleration / deceleration forces involve progressively deeper (medial) areas of brain:

1. mild DAI (grade 1) – lesions only in subcortical axons (mainly in parasagittal white matter of cerebral hemispheres).
2. moderate DAI (grade 2) – plus lesions in corpus callosum.
3. severe DAI (grade 3) – plus lesions in dorsolateral quadrants of rostral brain stem (cerebral peduncle).

• if patient survives \( \rightarrow \text{wallarian degeneration (affected areas of white matter are replaced by glial proliferation over several months)} \) \( \rightarrow \text{degeneration of involved fiber tracts (delayed neurologic deterioration).} \)

**CONCUSSION AND LACERATION**

- site of hemorrhage necrosis (hemorrhage mixed into tissue*) on brain surface** - result of CONTACT IMPACT injury.

**vs. HEMATOMA** - focal collection of blood

**vs. LACERATION** - pia-arthroclid is intact (e.g. in blunt injuries).

• blood frequently spreads under pia; if pia is lacerated \( \rightarrow \text{SAH}. \)

\[ \text{CONCUSSION selectively disrupts attention!} \]

- wide spectrum of neurologic symptoms may be described but are quickly resolved* - most patients on presentation are neurologically normal (GCS = 15) – diagnosis is usually retrospective!

* patients become normal within few minutes; others may be slightly dazed for minutes and complain of headaches for \( > 12 \) hours.

• concussions are graded:

  grade 1 - confused temporarily but does not display any memory changes.
  grade 2 - brief convulsion, antegrade amnesia of \( < 5 \) minutes’ duration.
  grade 3 - loss of consciousness for \( < 5 \) minutes and retrograde amnesia.
  grade 4 - loss of consciousness for \( > 5-10 \) minutes.
  grade 5 - loss of consciousness > 10 minutes.

According to new guidelines, concussion severity is no longer classified at time of event!

\[ \text{CONCUSSION is mild head injury!} \]
**Coronal section through frontal lobes**

**Source of pic**

**lobes:**

- Characteristic location of extensive contrecoup contusions consistent with fall backwards
- Compression of adjacent tissue → ischemia → necrosis → cyst
- 1) ICP↑, brain herniation
- 2) contusion is nidus for delayed hematoma formation (esp. in alcoholics, elderly patients or taking anticoagulants).

**LOCATION of contusions**

1) **coup contusion** - at site of impact (direct trauma or during brain acceleration):
   a) skull is sufficiently bent inward to strike underlying brain
   b) moving brain abruptly strikes fixed skull
   c) under depressed skull fractures (“Fracture contusion”)
   d) along tract of missile injuries

2) **contrecoup contusion** - in antipolar area, i.e. at point opposite impact (during brain deceleration).
   - classically, contrecoup contusion occurs when falling head strikes ground:
     - backward fall → contrecoup contusions at frontal and temporal poles; fall on side → contrecoup contusions at opposite temporal lobe.
     - forward fall: does not cause contrecoup contusions on back of brain because interior surface of skull is smooth at this point!
   - amount of energy dissipated at site of impact determines type of contusion:
     - from small hard object: most of energy is dissipated at impact site → coup contusion
     - impact from larger object - less injury at impact site since energy is dissipated at beginning or end of head motion → contrecoup contusion.
   - role of skull compliance:
     - pediatric compliant skull is easily deformed → coup contusion.
     - adults - brain is forced against bony protuberances opposite point of impact → contrecoup injury.

N.B. whatever site of injury, contusions are most severe in:

- **orbital surface of FRONTAL LOBES**
- anterior & basal portions of TEMPORAL LOBES - brain glides over ridged bony surfaces – orbital roof, sphenoid wing & petrous ridge (wrench & petrous ridge injuries)

Gliding contusions - along superior margin of cerebral hemispheres - due to sagittal angular acceleration/deceleration with abrupt stretching and tearing of parasagittal veins, arachnoid membrane, and adjacent cerebrum.

N.B. gliding contusions are result of acceleration/deceleration shear strains (as is diffuse axonal injury) - tend to be bilateral!

**HISTORY**

- **coup contusions:** - edema and pericapillary hemorrhage.
- next few hours - blood extravasation extends throughout involved tissue, across width of cerebral cortex, and into white matter and subarachnoid spaces.
- evidence of neuronal injury (necrosis, cytoplasm eosinophilia, cell disintegration) takes 24 hours to appear (functional brain injury occurs earlier). evidence of neuronal injury (nucleus pyknosis, cytoplasm eosinophilia, cell disintegration) takes 24 hours to appear (functional brain injury occurs earlier).
- axonal swellings develop in vicinity of damaged neurons or at great distances away.
- usual inflammatory response: neutrophils → macrophages. axonal swellings develop in vicinity of damaged neurons or at great distances away.

**HEALING of contusions**

- superficial lacerations - edema and pericapillary hemorrhage.
  - next few hours - blood extravasation extends throughout involved tissue, across width of cerebral cortex, and into white matter and subarachnoid spaces.
  - evidence of neuronal injury (necrosis, cytoplasm eosinophilia, cell disintegration) takes 24 hours to appear (functional brain injury occurs earlier).
  - axonal swellings develop in vicinity of damaged neurons or at great distances away.
  - usual inflammatory response: neutrophils → macrophages.

- larger areas of necrosis that extend deep heal by formation of MENINGOCEREBRAL CICATRIX (composed of glia, fibroblasts, and meninges) and larger CAVITATED LESIONS.

**CLINICAL FEATURES**

1) focal deficits (coincide with affected brain region; most often hemiparesis or gait preference) – manifest after consciousness is regained.
2) increasing ICP – manifests as progressive neurologic deterioration.
3) late posttraumatic seizures
4) contusion per se is clinically silent if in non eloquent area (e.g. anterior temporal lobes or inferior frontal lobes); but may manifest latter as expanding mass!
5) contusions in brainstem may be fatal.

**Coronal section through frontal lobes – extensive contrecoup contusions involving inferior gyri:**

**HEALING of contusions**

- Clinical symptoms: initial CT scan may be normal, but delayed scan may show evidence of contrecoup contusions:
  - large or focal defects (e.g., mass effect)
  - contusion per se is clinically silent if in non eloquent area (e.g. anterior temporal lobes or inferior frontal lobes); but may manifest latter as expanding mass!
  - contusions in brainstem may be fatal.

**Location of contusions**

- coup contusion: at site of impact (direct trauma or during brain acceleration):
  - skull is sufficiently bent inward to strike underlying brain
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  - along tract of missile injuries

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**N.B.** whatever site of injury, contusions are most severe in:

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Gliding contusions - along superior margin of cerebral hemispheres - due to sagittal angular acceleration/deceleration with abrupt stretching and tearing of parasagittal veins, arachnoid membrane, and adjacent cerebrum.

N.B. gliding contusions are result of acceleration/deceleration shear strains (as is diffuse axonal injury) - tend to be bilateral!
Old contusions - orange-brown (hemosiderin), scalloped lesion

Contrecoup contusions, mainly of right inferior frontal lobe

Extensive blunt force trauma (vehicular accident) - contusions and lacerations

Contusion of temporal poles with fresh hemorrhages
INTRACEREBRAL HEMORRHAGE (TRAUMATIC ICH, ICH)

See below >>

MISSILE (GUNSHOT) INJURY

Wounding capacity of firearm is related to kinetic energy of its missile:

\[ \text{Kinetic energy} = \frac{1}{2} \times \text{mass} \times \text{velocity}^2 \]

Types of injuries:
A. Tangential wounds - caused by impact at oblique angle relative to skull.
   - if missile has high velocity but low energy, it can travel around skull under scalp without passing through skull itself (but at site of impact depressed skull fracture can occur).
   - intracranial damage (primarily cortical contusions) occur at site of initial impact (pressure waves generated by impact).
B. Penetrating injury - projectile breaches cranium but does not exit;
   - low-velocity projectile loses energy as it penetrates skull; projectile may bounce off opposite inner table of skull and ricochet within brain.
C. Perforating injury (worst prognosis!)
   - projectile passes entirely through head, leaving both entrance and exit wounds.
   - entrance wound is smaller than exit wound.
   - brain damage is accompanied by extensive hemorrhage.

Bullets that penetrate skull do not travel in straight path:
   a) low-velocity civilian soft bullets - tend to be deflected by intracranial structures (final track is erratic and occasionally bears no relation to exit or entrance site).
   - destabilizing motions include yaw (deviation of longitudinal axis of bullet from straight line), tumbling (forward rotation of bullet around its center of mass), rotation (oscillatory motion of bullet axis around its center of mass).
   b) high-velocity military metal-jacket bullets - can project straight through tissues and easily fracture bones.

Bullets can damage brain parenchyma through 3 mechanisms:
A. Direct laceration & crushing (main mechanism of low-velocity bullets); destroyed tissue is either ejected out of entrance or exit wounds or compressed into walls of missile tract.
B. Cavitation (severe in high-velocity bullets) - produced by centrifugal effects of missile.
C. Percussion shock waves (last 5-10 msec; severe in high-velocity bullets) - cause stretch injury far from missile path (if shock wave reaches brain stem, cardiovascular and respiratory collapse can occur; shock waves can disrupt vessel walls → traumatic aneurysms).
   - all these create permanent cavity (3-4 times larger than missile diameter) and pulsating temporary cavity (as much as 30 times larger than missile diameter → diffuse damage to brain).

ICP
   - rapid increase (up to 100 mmHg) for several minutes → drop (depending on volume of secondary hemorrhage and edema).

CLINICAL FEATURES
   - loss of function of brain that is directly injured.

COMPLICATIONS:
   - hemorrhage!!!, infection!!!, post-traumatic epilepsy.
   - metal fragments may cause electrolysis, may migrate within intracranial or intraspinal compartments.
   - penetrating wounds (incl. GSW) to the head are not associated with C-spine injuries.
   - injury to vascular walls (contact or shearing forces) may lead to aneurysm formation (most commonly – pseudoaneurysm – very vulnerable to delayed ruptures).

UPPER - basic differences between skin appearances of contact, close (intermediate), and distant (indeterminate) range gunshot wound.
LOWER - wounding characteristics in skull.
Entrance wound (at left) and exit wound (at right): bullet struck at angle to produce ovoid entrance; exit wounds vary considerably in size and shape because bullet can be deformed in its transit through body (e.g. “hollow-point” bullets are designed to deform so that all their energy will be converted to tissue damage and not exit):

Slit-like exit wound (no powder or soot visible):

Contact range gunshots:

Contact gunshot entrance wound; since barrel contacts skin, gases released by fired round go into subcutaneous tissue → star-shaped laceration; note also grey-black discoloration from soot, as well as faint abrasion ring:

Contact gunshot wound - abrasion ring, formed when force of gases entering below skin blow skin surface back against gun muzzle:
Contact range gunshot wound - abrasion ring, very clear muzzle imprint:

Contact range gunshot wound - grey-black discoloration from burned powder:

Contact range gunshot wound - skull surface demonstrates heavy soot, as well as radiating fracture lines; thus direction of fire was toward back of this picture:

Contact range gunshot wound (entrance wound on skin) - black gunshot residue (red arrow) and coagulative necrosis:

Intermediate range gunshots
Intermediate range gunshot entrance wound - powder "tattooing" around entrance site:
Intermediate range gunshot entrance wound - powder “tattooing”; actual entrance site is somewhat irregular, because bullet can tumble in flight.

SELF-INFLICTED INJURIES
- injury on dominant side.
- powder burns at entrance site.
- large stellate scalp lacerations (dissection of subgaleal layer by exploding gases).
- if entrance through mouth, injury to hard palate → upper airway compromise.
- careful aim and close range → mortality ≈ 95%.
  Suicide is more lethal than homicide!

STAB INJURY (IMPALMENT)
- skull penetration is most common in thin bones of skull:
  1) orbital surfaces
  2) squamous portion of temporal bone – highest mortality (short distance to brainstem and vascular structures)
  3) cranio-cervical junction 
- 2/3 cases on left side!
- knife leaves narrow elongated “slot” fracture (in some cases, no radiological abnormality can be identified).
- cerebral damage is largely restricted to wound tract (filled with clots).
  N.B. unlike missile injuries, no concentric zone of coagulative necrosis caused by dissipated energy is present, unlike motor vehicle accidents, no diffuse shearing injury to brain occurs.
- major complications are vascular (main cause of mortality):
  1) massive intracerebral hematoma (50%)
  2) subdural hematoma (9%)
  3) contusion (31%)
  4) traumatic aneurysms (risk of rupture - H: early angiography & repair)
  5) carotid-cavernous fistula
  6) stroke (5%)

Mechanism for “defense wounds”:

Typical “defense wounds”: 
COMPRESSION INJURY

- requires significant force - skull architecture provides substantial resistance to deformation.
- multiple linear skull fractures (can be depressed if high-energy rapid compression force is applied to small area of skull).

DEGREES OF SEVERITY

- determined by GCS score after initial resuscitation**.
- additional criteria: duration of loss of consciousness, duration of antegrade amnesia.
- rarely used criteria: number of days to achieve GCS score 15, number of days to achieve GCS motor score 6, length of hospital stay, CT results.

<table>
<thead>
<tr>
<th>TBI degree</th>
<th>GCS score</th>
<th>CT</th>
<th>Loss of consciousness</th>
<th>Antegrade amnesia</th>
<th>Additional</th>
</tr>
</thead>
<tbody>
<tr>
<td>MILD (c. concussion)</td>
<td>13-15</td>
<td>negative</td>
<td>&lt; 30 min</td>
<td>minutes</td>
<td>length of hospital stay &lt; 48 hours, 3% progress to more serious injuries*</td>
</tr>
<tr>
<td>MODERATE</td>
<td>9-12</td>
<td>negative or positive (10-15% have focal intracranial lesion)</td>
<td>30 min ≤ 6 h</td>
<td>hours</td>
<td>length of hospital stay &gt; 48 hours</td>
</tr>
<tr>
<td>SEVERE</td>
<td>≤ 8 (coma)</td>
<td>positive (40% have focal intracranial lesion)</td>
<td>&gt; 6 h</td>
<td>days</td>
<td></td>
</tr>
</tbody>
</table>

Mortality according to TBI severity → see above

- patients who reach ED alive:
  - 75.80% - mild injury (but many patients do not come to ED)
  - 10.15% - moderate injury
  - 10% - severe injury (but some patients die at scene of accident or during transport)

N.B. seemingly mild TBI can rapidly degrade* into severe TBI (“speaks and dies”) – even mild TBI cases must be closely observed in acute period.

*In 75% cases due to intracranial hematoma formation.

HIGH RISK MILD INJURY

| A. External signs of trauma |
| B. Skull fracture (or palpable depressed skull fracture) |
| C. Initial GCS 13 |
| D. Loss of consciousness (> 2-5 min) |
| E. Posttraumatic confusion/amnesia (> 20 min) |
| F. Focal neurologic findings |
| G. Asymmetric pupils |
| H. Posttraumatic seizure |
| I. Repeated vomiting or vomiting for > 8 hours after injury |
| J. Persistent severe or progressively worsening headache |
| K. Second ED visit because of persistent symptoms |
| L. Multiple trauma |
| M. Serious painful distracting injuries |
| N. History of bleeding disorder/anticoagulation |
| O. Presence of cerebrovascular malformation |

LOW RISK MILD INJURY

| A. Currently asymptomatic (incl. fully awake, GCS 15, no focal neurologic findings, normal pupils) |
| B. No other injuries (incl. no evidence of skull fracture) |
| C. No loss of consciousness |
| D. Intact orientation/memory |
| E. Not intoxicated |
| F. Accurate history |
| G. Trivial mechanism |
| H. Injury > 24 hr ago* |
| I. Reliable home observers |

*in 75% cases due to intracranial hematoma formation.
P. Intoxication (→ unreliable examination)  Q. Mechanism: high-speed motor vehicle accident, fall > 8 ft  R. Unreliable / unknown history of injury  S. Suspected child abuse  T. Age > 60 or < 2 yrs

**may miss chronic subdural hematoma**

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### PREHOSPITAL MANAGEMENT

#### FACIAL INJURIES

- See p. TiH25

#### ANTERIOR NECK INJURIES

- See p. TiS21

#### SPINAL INJURIES

- See p. TiS5

---

**TRIAGE principles:**

- Thoracic, vascular, and abdominal injuries take precedence over head wounds!
- Head injuries are more urgent than spinal injuries.
- Triage of head injuries: 1. Determining patients (who are not moribund), 2. Stable patients with level of consciousness! 3. Stable awake patients

**“GOLDEN HOUR” – first hour is very important prognostically – treat hypoxia & hypotension in the field and en route to the hospital**

- 60% fatalities occur before patients can be admitted to hospital (40% at scene and 20% in ER).
- Proper management in field can make difference between normal existence or lifetime spent in total paralysis.
- Mortality for military injuries: 4.5/100 in World War II → 2.5/100 in Korea → < 1/100 in Vietnam.
  1. Rapid evacuation is given much of credit! (in civilian injuries - helicopter transport)
  2. Trained teams of rescue workers (not physicians) provide intubation, shock treatment, and other emergency measures.
- Importance of ABC – ultimate outcome of brain injury is as much (or more) dependent on early ABC as any other organ.
- Even moderate hypotension can convert reversible brain injury to irreversible isometric brain damage.

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**1. FAST (General) general**

- Should be secured immediately

  - Only means we can help injured brain in the field – supply oxygen to brain!

- Hypoxia – most common cause of prehospital death! See “secondary Injury” above

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**AIRWAYS**

1. **Clear mouth** (foreign bodies, vomitus, blood).
2. Do not extend neck! Use jaw thrust technique / chin lift maneuver.

---

3. **Airway maintenance**

   a) oropharyngeal tube
   
   b) early endotracheal intubation
      - everybody in COMA (GCS < 8)
      - everybody with PENETRATING INJURY (if physician waits for coma before intubating patient, mortality approaches 100%).
      - extensive FACIAL INJURIES
      - NECK INJURIES
      - nasotracheal intubation is preferable (no neck manipulations), but avoid in facial / skull base fractures.
      - for awake patient intubation use RSI (rapid sequence induction).
      - N.B. Failure to use paralytic agents, pharyngeal anesthesia, and barbiturate induction → massive ICP elevation! See below
      - there is some suggestion of increased mortality with prehospital intubation in patients with moderate-to-severe TBI compared with patients intubated in ED (bag-valve-mask ventilation with good technique may be of more benefit to brain injured patients than prehospital intubation!).
   
   c) endotracheal intubation
   
   d) stabilize mandibular fractures see p. TiH25

---

**BREATHING**

a) spontaneous but insufficient breathing → 100% oxygen + assisted ventilation with demand valve.

b) no spontaneous breathing → ventilation (by positive-pressure ventilation with 100% oxygen). Portable pulse oximetry should verify SaO2 > 90% (provide supplemental oxygen to achieve this level).

---

**2. BLOOD CIRCULATION**

Injured brain is extremely susceptible to lowered perfusion states!
**FLUID RESUSCITATION** (brain injury per se rarely causes hypotension!!!)
- several large-bore intravenous catheters.
- isotonic (or hypertonic*) saline to aggressively restore SBP to > 100-110 mmHg. See below >>
  *there are studies showing that 250 mL of hypertonic (7.5%) saline bolus in the field improves survival
- avoid volume overload / hypervolemia! (but fluids should not be withheld in hypotensive patient for fear of increasing cerebral edema and ICP)
- autotransfusion: leg elevation + Military Anti-Shock Trousers.

**MANAGE BLEEDING, OPEN WOUNDS**
- scalp lacerations may bleed into volume bulky; better prehospital dressing is less bulky, but with firm constant pressure!
- temporary scalp bleeding control >> see MANAGEMENT
- skull fractures – do not require special care in the field
  - do not impede liquorthea through fracture lines
  - if brain / nerves exposed – cover with dressing with sterile saline (do not push brain back into cranium) – it is largely devitalized tissue and will be removed during scalp repair.
- do not impede liquorthea/bleeding from nose / ear canal – will lead to intracranial hematoma and infection; H: place absorbent dressing without tamponade
- epistaxis is safe to tamponade only if no signs of anterior skull base fracture
- life-threatening dural sinus bleeding can be slowed by placing patient in reverse Trendelenburg position (risk of air embolism)
- penetrating objects should be left in place (stabilized with bulky fluffly bandage) - to be removed in operating room.

3. CERVICAL SPINE STABILIZATION
  >> see p. 1185 >>

4. BRIEF NEUROLOGIC STATUS
1. Level of consciousness (typically GCS)
2. Pupil size and light reactivity (asymmetry is most important)
3. Extremity motorics (asymmetry is most important) - 1 spontaneous movements, 2 following commands, 3 reaction to painful stimuli.

5. TRANSPORTATION
- rapidly transport patient with CT and definitive neurosurgical intervention*.
  *level 1 trauma centers certified by American College of Surgeons (or state trauma certification systems) have trauma surgeons in house 24/7 and neurosurgeon available within 10 minutes of notification.
- patient is moved on blue – to avoid displacing spine (or other bones), so that spinal cord (and blood vessels) are not injured.
- transport on left side, HOB elevated 15-30 degrees.
- for transport times longer than 15-20 minutes, aerial neurologic assessments should be documented every 15-15 minutes.
  - most important parameter to monitor – level of consciousness (along with pulse, BP, breathing)
  - all patients should be placed on cardiac monitor as they are transported (brainstem compression can cause cardiac dysrhythmias).
- many severely head-injured patients are initially comatose / agitated - transporting patient who is fighting against physical restraints may exacerbate physical injury, cause rise in ICP, and interfere with appropriate stabilization and management; H: prehospital sedation or paralysis (inform ED and trauma teams about patient in advance!)
- Morphone and other depressants are contraindicated during initial management!
  (short acting opioid [fentanyl] or sedative [propofol], paralytic are fine)
- early contact by telephone or radio with trauma center - medications given to patient coordinated through neurosurgeons and other health care providers at trauma center.

**DIAGNOSTIC EVALUATION**

**DIAGNOSIS IN LATIN**
Morbus traumaticus cerebri, periodica acuta/subacuta, forma levis/mediocritas periodica acuta – within 3 months.

Diagnosis is constructed by naming injuries from outside to inside:
1) scalp (e.g. Vulnus contusum reg. parietalis, Haematoma subaponeuroticum reg. frontalis) 2) fractures (e.g. F.ea aperta impressa comissura ossis frontalis, F.ea basis crani fissura mediae; F.ea ossa tibiae) 3) brain, including complications (e.g. Syn. contumationale level/medio/crave, Compressio cerebri, Haematoma epiduralis/subduralis, Contusiones multiples hemipherii sin.; Contusio cerebri lobo occipitalis, Hæmorragia subarachnoidialis),
- state syndrome first, then its cause (compressio >> haematomata).
- if there is compression or haematoma, do not mention concussion.
- Ebrietas vulgaris is mentioned at concurrent conditions.

**HISTORY**
- patient may be comatose or confused – witnesses & paramedics are of crucial importance!
3. After consciousness was regained – amnesia (last thing that can be recalled), vertigo, nausea, vision blurring, headache, other pains.
4. Prior head injuries.
5. Remote or active medication* / drug / alcohol use - risk of intracranial bleeding, cloud mental status.
6. Premorbid history of headaches, seizures, syncope, TIA & stroke, gait disturbance, psychiatric disease – all these can be the cause of TBI and should be treated!

**PHYSICAL EXAMINATION**
Examination of patient with decreased level of consciousness – see p. 330 >>

Cervical spine precautions* (10% patients with severe TBI have spinal trauma)

*xp. perform during PRIMARY SURVEY (identifying life-threatening conditions).
NEUROLOGIC EXAMINATION

1. a) awake patient → detailed neurologic examination.  
b) uncooperative/comatose patient:
   1) pupillary size & light response. (see below >>)
   2) motor response → responsive to stimuli, pathologic and deep tendon reflexes.  
   3) cranial nerves (for deeply comatose patients) - oculocephalic (oculovestibular) tests, respiratory patterns.  

   *asymmetry is most important

4. Cranial nerves & brainstem
   - pupillo: (size and response to light)  
   - corneal reflex  
   - facial asymmetry (e.g. when patient grimaces with noxious stimuli)  
   - gag reflex - chyphagia (risk of aspiration)
   - respiratory pattern

   N.B. if voluntary eye movements cannot be assessed → fast, no motion artifacts
   - pupillary size of > 4 mm
   - Fixed pupillary response < 1 mm to bright light

   pupillary asymmetry relate to intracranial injury unless proved otherwise
   unilateral dilated pupil in unconscious patient – CN3 compression (encal herniation), pinpoint pupils - ponsite lesions.
   light nonreactive pupils in mid position - midbrain tector lesions.

   eye movements
   N.B. if voluntary eye movements cannot be assessed → OCTOCEPHALIC and OCELOVESTIBULAR testing
   see p. 320 >>

   corneal reflex  
   - facial asymmetry (e.g. when patient grimaces with noxious stimuli)
   - gag reflex - chyphagia (risk of aspiration)
   - respiratory pattern

   *may also indicate pulmonary failure or infection

6. Motor examination

   N.B. if patient is not alert enough to cooperate with strength testing, motor examination is limited to assessment of motor asymmetry, pathology & deep tendon reflexes
   N.B. occult extremity trauma can make examination painful or difficult!

   Sensory examination is not reliable in patients who are intoxicated or comatose!

8. Redline cognitive testing (e.g. Mini-Mental State Examination) - to distinguish damaged and spared realms of cognitive functioning: practically never done in acute phase of TBI.  
see p. D2 >>

   - ability to lay down new memories (determines duration of anetregia amnesia via serial mental status assessments).

IMAGING

Presence / absence of cervical spine instability must be determined first! (lateral cervical spine* and chest X-rays are usually obtained in resuscitation room).  
*modern approach - cervical spine CT (at same time as admission head CT) in all but mildest TBI patients. see p. Tr5 >>

IMAGING MODALITIES

<table>
<thead>
<tr>
<th>CT</th>
<th>MRI</th>
<th>ANGIOGRAPHY</th>
<th>CRANIOGRAM</th>
</tr>
</thead>
</table>
| Pts. no motion artifacts  
2. Patient accessible for monitoring.  
3. Define acute hemorrhages, mass effects, bony injuries, hydrocephalic edema  
4. Define cerebra, leptomeningal, perivascular, posttraumatic ischemic, infarction, brainstem injuries  
5. Differentiate acute traumatic lesions.  
6. Define vascular injuries, injuries to various stroma  
7. Detect mass effects. |

ADVANTAGES

1. Usually available  
2. May help screen some patients for further imaging studies.

DISADVANTAGES

1. Artifacts from patient movement, foreign bodies  
2. Artifacts may obscure brain stem or posterior fossa.

1. Slow (long scanning time), motion artifacts  
2. Magnetic field precludes use of monitors and life support equipment.  
Does not define most acute hematogenous lesions  
4. Not useful for bony injuries  
5. Unsuitable for people exposed to ionizing effects.

Requires formal IR precheck so is done only if CTA is positive.

Does not indicate presence or absence of intracranial injury so it is obsolete study.
American College of Radiology (ACR): Appropriateness Criteria Scales

(1 = least appropriate; 9 = most appropriate; NA = not applicable)

<table>
<thead>
<tr>
<th>Clinical Situation</th>
<th>Skull X-ray</th>
<th>Cervical X-ray</th>
<th>CT</th>
<th>MRI</th>
<th>MRA</th>
<th>Angiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>mild closed TBI (GCS ≥ 13, no neurologic deficit)</td>
<td>2</td>
<td>7</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
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<tr>
<td>mild closed TBI (focal neurologic deficit)</td>
<td>2</td>
<td></td>
<td>9</td>
<td>6</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>moderate + severe closed TBI, stable</td>
<td>4</td>
<td>8</td>
<td>9</td>
<td>6</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>mild + moderate closed TBI, child ≤ 2 yrs</td>
<td>4</td>
<td>6</td>
<td>9</td>
<td>6</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>closed TBI, rule out carotid or vertebral artery dissection</td>
<td>NA</td>
<td>5</td>
<td>4</td>
<td>8*</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>penetrating TBI, neurologically intact</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>6</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>penetrating TBI, likelihood of vessel injury</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>6</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>depressed skull fracture</td>
<td>8</td>
<td>6</td>
<td>9</td>
<td>6</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>calvarial fracture</td>
<td>8</td>
<td>6</td>
<td>9</td>
<td>5</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>penetrating TBI, skull-base fracture</td>
<td>6</td>
<td>6</td>
<td>9</td>
<td>6</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>subacute TBI, late neurologic deterioration</td>
<td>NA</td>
<td>NA</td>
<td>8</td>
<td>8</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>subacute / chronic TBI, stable, normal CT, cognitive and/or neurologic deficit</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>8</td>
<td>4</td>
<td>NA</td>
</tr>
<tr>
<td>Chronic TBI, neurologic dysfunction</td>
<td>NA</td>
<td>NA</td>
<td>6</td>
<td>8</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

*5 for gadolinium-enhanced MRI

CT WITHOUT CONTRAST

- diagnostic imaging of choice! – detects skull fractures, contusions, blood, edema.
- Contrast is rarely required to outline subarachnoid / chronic hematomas or intracranial abscesses
- CT is indicated for all patients except COW-STS mild TBI:
  - for non-cow-sts mild TBI (without skull fracture), CT may be deferred if patient is hospitalized for observation (any deterioration in neurologic status or any focal signs) → CT

If CT is indicated, skull radiographs are not necessary (CT bony window shows even basilar fractures)

- more severe TBI, greater urgency for CT (exception is when other life-threatening injuries take precedence); e.g. severe TBI (esp. with lateralizing signs) → CT as soon as cardiorespiratory stability is ensured.
  - N.B. urgency for CT is function of likelihood of surgically correctable focal lesion (vs. diffuse injuries - do not benefit from acute neurosurgical intervention - precise anatomic information is not necessary for optimal management).
  - deteriorating patients should be accompanied by physician.
  - stable but seriously injured patients should be accompanied by experienced ER or trauma nurse.
  - if abdominal ultrasound or diagnostic peritoneal lavage has not been performed, most comatose patients are evaluated with CT.
  - penetrating TBI, likelihood of vessel injury
  - depressed skull fracture
  - calvarial fracture
  - penetrating TBI, skull-base fracture
  - subacute TBI, late neurologic deterioration
  - subacute / chronic TBI, stable, normal CT, cognitive and/or neurologic deficit
  - Chronic TBI, neurologic dysfunction

Most important things to look in CT:
- mass lesions (hematoma), state of basal cisterns (incl. blood inside), midline shift, ventricular size.

Basil cisterns are evaluated at the level of the midbrain.

Basal cisterns can be:
- open (all limbs open), partially closed, s. “crowded” (one or two limbs obliterated), or completely closed s. “effaced” (all limbs obliterated).

Midline shift is calculated at the level of the foramen of Monro to the septum pellucidum:

Midline shift = (A2 - A1) / B

Technique
- from base of occiput to top of vertex in at max. 5-mm increments.
- three data sets are obtained:
  1) bone windows - bony anatomy of skull.
  2) tissue windows - detailed survey of brain.
Indications for specific repeat CT:

1. GCS ≤ 1 point
2. New or aggregated focal signs
3. Persistent severe headache, frequent vomiting
4. Seizure

Indications for scheduled repeat CT:

1. Nonoperated intracranial hematomas, contusions*  
2. Coup injuries

CT is repeated within 4–24 hours of initial scan (some contusions and intracranial hematomas are brought to operating room after repeated CT).

*studies do not support SCHEDULED REPEAT CT in patients with intracranial hemorrhage and GCS13-15 and no change in status imaging do not change management and outcomes

Marshall Classification of Diffuse Brain Injury

1. Grade 1: normal CT scan
2. Grade 2: cisterns present, shift < 5 mm
3. Grade 3: cisterns compressed or absent, shift < 5 mm and no high or mixed density lesion
4. Grade 4: shift > 5 mm

Marshall Scoring of TBI

<table>
<thead>
<tr>
<th>MLS</th>
<th>Cisterns</th>
<th>High or mixed density lesion</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>None</td>
<td>None</td>
<td>No visible pathology on CT scan</td>
</tr>
<tr>
<td>II</td>
<td>&gt;=5 mm</td>
<td>Present</td>
<td>Cisterns are present with midline shift 5+ mm and/or lesions densities present, no high or mixed density lesion &gt; 25 cc, may include bone fragments and foreign bodies.</td>
</tr>
<tr>
<td>III</td>
<td>0-5mm</td>
<td>Present/Compressed/Absent</td>
<td>No visible pathology on CT scan.</td>
</tr>
<tr>
<td>IV</td>
<td>Any</td>
<td>Any</td>
<td>Any lesion surgically evacuated.</td>
</tr>
<tr>
<td>VI</td>
<td>Any</td>
<td>&gt;=25cm</td>
<td>Not surgically evacuated.</td>
</tr>
</tbody>
</table>

Grade 1 = normal CT scan (90.6% mortality)
Grade 2 = cisterns present, shift < 5 mm (13.5% mortality)
Grade 3 = cisterns compressed or absent, shift < 5 mm (34% mortality)
Grade 4 = shift > 5 mm (56.2% mortality)


PLAIN/SCull RADIOGRAPHY

- Many patients with skull fracture have no neurological sequelae and many with severe intracranial abnormalities have no associated skull fractures!

1) screening for CT in mild TBI with signs of skull fracture on physical examination when CT is not immediately available: (if skull fracture is confirmed — CT & observation for delayed complications)

N.B. yield of cranialography in low-risk mild TBI is very low (even if detected, linear fractures have limited clinical significance – adults – do not change treatment course).

2) Depressed skull fracture - use tangential shots to measure the depth of impression.

3) penetrating injury – to detect intracranial foreign bodies (small intracranial foreign bodies may be missed unless skull X-rays are taken after minor penetrating injuries).

4) additional indications for pediatric population:
   a) screening for CT in children < 5 yr (difficult clinical examination) with minor TBI
   b) suspected child abuse (skull radiograph as part of skeletal survey).
   c) suspected linear skull fracture (risk of leptomeningeal cyst) - deep scalp lacerations, large scalp hematomas.

NONENHANCED MRI

- most useful in subacute/chronic setting: prognostic use during subsequent care (assists in directing rehabilitation and goals of care).

- MRI gives superior (than CT) depiction of non-surgical lesions; some authors now advocate MRI use in neurologically stable patients with moderate-to-severe closed TBI, whereas CT is reserved for neurologically unstable patients.

- specific sequences:

  1. T2* either GRE (gradient-recalled echo) or SWI is most sensitive sequence to detect small hematomas, blood breakdown products, diffusion-weighted images - useful for cerebral infarctions
  2. FLAIR - useful for SAH

N.B. gadolinium enhancement gives no notable advantage!

Indications:

1. Subacute period of severe TBI - to depict full extent of injury to brain!
2. Suspected contusions not seen on CT (MRI is criterion standard for defining contusions*)
3. Persistent symptoms with postconcussive syndrome
4. Suspected posttraumatic ischemic infarction
5. Mental status abnormalities (unexplained by CT; MRI is more sensitive for diffuse axonal injury)
6. Subacute/chronic subdural hematomas (MRI is more sensitive than CT but normally CT is just enough).

**Contraindication:** Penetrating injuries with possible (not ruled out with X-ray) intracranial metallic fragments.

**Angiography:**
- Used only when vascular injury may be present and when endovascular intervention is anticipated:
  a) Penetrating injury (esp. due to impalement) – absolute indication if CTA shows proximity to major vessels!
  b) Carotid injuries on CTA with attributable neurologic deficits from flow limitation
  c) Skull base fracture near major arteries with unexplained neurologic deficits (esp. temporal bone fractures).
- Performed only in patients in stable condition.
- CTA/MRA can obtain similar information.
- Formerly (in pre-CT era), angiography was used to diagnose intracranial hematomas.

**Ultrasoundography:**
- Intracranial hemorrhage in infants.
- Leptomeningeal cyst (after linear skull fracture) in infants.
- Orbital soft-tissue injury.

**Conditions:**

**Concussion**
- Normal CT/MRI.
- DTI is abnormal!

Diffuse increase in mean diffusivity after remote concussions. Sagittal (A and B), coronal (C and D), and axial (E and F) slices of the tract-based spatial statistics group contrast on mean diffusivity maps (controls vs concussed in blue). The contrasts are overlaid on the mean fractional anisotropy skeleton (in green) and the standard MNI152 T1 1-mm brain template. The results are thresholded at P ≥ .05, corrected for multiple comparisons.

**Diffuse Axonal Injury**
- Often not visible (microscopic damage).
  - **CT** - Small petechial hemorrhages in white matter (most frequently juxtacortical & periventricular, corpus callosum, internal capsule and dorsorostral brainstem) – “tear” hemorrhages;
  - **DAI** can sometimes be superimposed by generalized brain swelling
  - **MRI** is more sensitive (may be positive even when CT is negative, e.g. nonhemorrhagic DAI) - diffuse, small, focal abnormalities limited to white matter tracts.
  - **SWI** is the most sensitive sequence (more sensitive than GRE)?
- Months after injury - Reduced bulk of white matter with persistent hemosiderin foci.

T2-MRI - Foci of hypointense signal representing small shear hemorrhages at gray/white junction (arrows):
SWI is more sensitive for ICH than traditional GRE sequences. A. Standard GRE image shows several small ICH in a patient with diffuse axonal injury. B. Two additional hemorrhages are identified on SWI sequence at the same slice in this patient (arrows).

QSM is able to differentiate between ICH and calcification. A. Axial GRE image shows a small hypointense lesion within the left frontal lobe subcortical white matter (white arrow). B. Coronal QSM image acquired through this region during the same MRI session shows a corresponding hyperintense lesion confirming that this is an ICH with paramagnetic properties (white arrow).

SCALP INJURIES
- CT reliably depicts subgaleal hematomas, elevated and avulsed soft tissues, scalp edema.
- air within scalp tissue suggests scalp laceration (gas detected several days after trauma - infection).
- CT hyperdense region in anterior temporal lobe:

CONTRUSION
- MRI is criterion standard for defining contusions!
- early - inhomogeneous hyperdensity* on CT, hyperintensity on MRI (T1 and T2).
- *initially (< 1-2 hours after TBI), partial volumes between dense microhemorrhages and hypodense edema can render contusions isoattenuating on CT (high initial false-negative rate!); vs. MRI demonstrate contusions from onset!
- mass effect may distort adjacent sulci and lateral ventricles.
- some degree of SAH is almost always present.
- after several hours - ring of lower density (edema surrounding contusion; very early - only edema without hemorrhages).
- after week (blood within contusions has begun to degrade, and MRI becomes more useful) - surrounding ringlike contrast enhancement.

N.B. contusions progress with time in size, number, and amount of hemorrhage within contusions – these changes are most evident over first 24-48 hours (in 25% cases delayed hemorrhage occurs in areas that were previously free of hemorrhage) – routinely repeat CT!!!

Noncontrast CT - hyperdense region in anterior temporal lobe:
Noncontrast CT - cerebellar contusion; right-sided soft tissue swelling and bone injury are also obvious:

Noncontrast CT - large, right frontal contusion with hemorrhage and surrounding edema; smaller right temporal cortical contusion (short arrow); small left frontal subdural hematoma (long arrow):

Enlargement of contusion:
A) CT on day 1 - subtle area of slightly hypodense right frontal lobe contusion (arrows)
B) CT on day 2 - large, right frontal contusion (arrow) and new, large, left temporal contusion (arrow); scalp hematomas have increased in size:

CT immediately after blunt trauma to left convexity - severe swelling of entire left cerebral hemisphere with small collection of subarachnoid blood (up arrow); right hemisphere shows contrecoup gliding contusions (down arrows):

Penetrating injuries
CT – imaging modality of choice: focal areas of increased attenuation (represent parenchymal, intraventricular, subarachnoid, or extracerebral hemorrhage).
• path of missile is often obvious from location of hemorrhage and metallic fragments (produce streak artifacts).
• coronal sections may be helpful in patients with skull base or high convexity involvement.

Plain radiographs - can be helpful in assessing bullet trajectory, the presence of large foreign bodies, and the presence of intracranial air; when CT is available, plain radiographs are not essential and are not recommended as a routine.

MRI - not generally recommended in the acute management.
• may help evaluate injuries from penetrating wooden or other nonmagnetic objects.
• in addition to artifact and image distortion, ferromagnetic missiles can also rotate and deflect in response to magnetic torque (literature has not reported any patient suffering additional injury caused by MRI).

**Angiography / CTA** – when vascular injury is suspected:
  a) wound’s trajectory passes through or near the Sylvian fissure, supracalvarial carotid, cavernous sinus, or a major venous sinus – facial, orbital, peritonsillar entry site.
  b) substantial and otherwise unexplained SAH or delayed hematoma.
• aneurysms may form in delayed fashion (initial negative CTA/angiogram does not exclude the risk).

**OTHER NEUROLOGIC TESTS**

**Lumbar puncture** is contraindicated (unless meningeal is suspected – but LP only after imaging excluded mass lesion = basal cisterns must be open on CT).
• LP can be therapeutic in SAH.

**Ophthalmoscopy** – signs of ICP (papilledema, loss of venous pulsation).

**EEG** - not emergency test!
  • if taken at some convenient time after admission, it may aid in prognosis:
    - at time of injury - suppression of electrical cortical activity: with recovery - activity returns to normal (often through phase of generalized slowing and increased voltage).
      - common feature in all patients (may persist for many weeks) - undue susceptibility of cortical activity to oxygenation
    - areas of cortical damage may show abnormal activity (slowing and spike activity) for weeks - months – risk of epilepsy

**Most useful role of EEG - diagnosis of nonconvulsive status epilepticus!**

*Most useful role of EEG - diagnosis of nonconvulsive status epilepticus!*°

°may be detected in ≥ 8% comatose patients

• N.B. sedative medications may have confounding effect!

• bilaterally absent somatosensory & auditory evoked potentials predict unfavorable outcome in ≥ 99.5% patients.

**Transcranial Doppler**
• normal average linear blood velocity (LBV) in MCA = 60 cm/s.
• LBV > 100-120 cm/s:
  a) hyperemia (parallel LBV increase in MCA and ICA) – esp. days 2-4 after TBI
  b) vasospasm (LBV in MCA exceeds LBV in ICA are least 3 times) – usually on days ≥ 5 post TBI
• low LBV (esp. early after TBI) reflects brain hypoperfusion.

**Gosling (pulsatility) index** = (systolic LBV - diastolic LBV) / mean LBV.
if > 1 – sign of intracranial hypertension

**NON-NEUROLOGIC TESTS**

**Monitor ECG** - high incidence of supraventricular tachycardia (most common dysrhythmia after TBI), ventricular dysrhythmias, ST-T wave abnormalities, prolonged QT.

**Urine toxicological screen and blood alcohol level** - interpreting patient's mental status.

**Glycemia**
• hyperglycemia correlates with poor outcome.

**Serum electrolytes & osmolality**

• Urine output↑, urine specific gravity↓

**| Sodium (alterations occur in 50% comatose patients): |
<table>
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<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>HYPONATREMIA - due to:</td>
</tr>
<tr>
<td>a) syndrome of inappropriate antidiuretic hormone (SIADH)</td>
</tr>
<tr>
<td>b) cerebral salt wasting (release of natriuretic hormone → urinary Na losses↑ → volume depletion)</td>
</tr>
<tr>
<td>hyponatremia can potentiate brain edema and cause seizures.</td>
</tr>
<tr>
<td>HYPERTERMIA - due to:</td>
</tr>
<tr>
<td>a) dehydration</td>
</tr>
<tr>
<td>b) diabetes insipidus (urine output, urine specific gravity)</td>
</tr>
</tbody>
</table>

N.B. MANNITOL can mask diabetes insipidus by producing high urine output.

**Magnesium** is depleted in acute phases of both minor and severe TBI.

H. **MAGNESIUM SULFATE IV or PO**
N.B. magnesium blocks excitotoxic response and functions as antioxidant - careful monitoring of magnesium may improve outcome!

**Coagulation studies (PT, aPTT, fibrinogen, platelet counts)**

**CBC, Hct** – monitor in case of bleeding (type and cross match are always obtained with initial orders).

**Arterial-venous blood gases** – monitor in comatose patients.
• 20-gauge Silastic catheter is inserted with tip in internal jugular bulb, usually on right - arterial jugular venous oxygen content difference can be obtained:
  - normal venous saturation ≥ 85% 
  - ≤ 50-55% - global brain ischemia
  - > 50% - ICP↑ (hypoxemia).

Maintain arterial - jugular venous O2 content difference ≤ 7 vol%.

**Volumetric indicators (CVP, PCWP)** – monitor during treatment of hypotension.

**Renal function tests & CK** – to exclude rhabdomyolysis if crush injury has occurred or marked rigidity is present.

Within 4-5 days after admission, comatose patients should have screening endocrine battery (incl. cortisol, prolactin, growth hormone, thyroid function) – screening of pituitary injury.

**MANAGEMENT**

Physicians can do nothing to either replace lost neurons or accelerate restoration of recovering neurons!
**DISPO**

**HOSPITALIZATION**

Neurologic deterioration is most common within 24 hrs after TBI

- all patients (except LOW-RISK mild TBI) must be admitted!
  - TBI (HIGH-RISK mild TBI patients should be observed for 8-24 hours*) ("heuro checks" at 30-60 min intervals - any deterioration in neurologic status or any focal signs → CT).
  - if physician decides that patient with TBI (HIGH-RISK mild TBI can be sent home, appropriate early follow-up should be arranged.

*Patiennted patients must be observed until clinical sobriety

- all severe TBI and most moderate TBI patients are admitted to ICU.
- if patient is sent home
  1) patient should be monitored at home* in quiet environment in acute period (12-24 hours) by responsible nonintoxicated adult who lives with patient (and has access to telephone).
  2) do not allow to work, drive, etc

**Goals**

1. Severe TBI patient
   - maintenance brain perfusion
   - recovery CBF
2. Seizure control
3. ICP control
4. if patient is severe TBI patients
   - fluid restriction is contraindicated in TBI
   - hyperventilation probably is used, jugular venous oxygen saturation (SjO2) is normal
   - ICP.

**TRANSFER**

Severe TBI patient:

1. ICP control - intubation, MANNITOL.
2. Seizure control - LEVETIRACETAM or PHENYTOIN.

N.B. if patient is deteriorating, perform emergency burr holes see below

**AIRWAY, VENTILATION**

- all severe TBI patients (or who have penetrating TBI) should be intubated: to prevent aspiration, to protect airway and respiratory drive, to allow PRN hyperventilation.

* Rapid sequence induction [RSI] (with adequate sedation and paralysis!!!) is recommended to avoid ICP, see p. 3905 >>
  1) ARAZTIV (cerebroprotective agent (ATTEMAT: maintains BP, lowers ICP and brain metabolism, has rapid onset and brief duration),
  2) LIDOCAINE IV (1-2 mg/kg),
  3) PARALGESIC - SCYCYCLOHEXILIDE 1-2 mg/kg.

* airway induction (→ ICP?) is blunted by MORPHINE or FENTANYL.

- hyperventilation is recommended only as a short temporizing measure for the quick reduction of elevated ICP.
- hyperventilation probably should be avoided during the first 24 hours after injury when cerebral CBF is often critically reduced (hyperoxia induces ischemia).
- if hyperventilation is used, jugular venous oxygen saturation (SjO2) or brain tissue QO2 pressure (BtO2) measurements are recommended to monitor oxygen delivery.
- pulmonary toilet.
- comat for > 5-10 days → tracheotomy.

**BLOOD PRESSURE**

- to maintain brain perfusion (cerebral perfusion pressure), see below >>

End point is CFP > 60-70 mmHg

**Goals**

- Systolic BP > 100 mmHg (IV fluids ± vasopressor agents, if necessary)
  1. **Level III recommendation:** Maintaining SBP ≥ 100 mmHg (for patients 50-69 years old) or ≥ 110 mmHg (for patients 15-49 or ≥ 70 years old) may be considered to decrease mortality and improve outcomes.
  2. do not treat hypertension < 160 mmHg until intracranial hypertension is excluded (Cushing reflex is for brain perfusion)
  3. < 140 mmHg if ongoing risk of intracranial bleeding

- N.B. albumin worsens outcome (albumin extravasates and worsens cerebral edema)

**Level II evidence**

- av PdO2 < 80 mmHg
- single episode of SBP > 90 mmHg is associated with doubling of mortality in severe TBI (≥ 2 hypertension episodes increase mortality 8-fold)

**Level III evidence**

- CVP 5-15 cmH2O
- PCWP 10-14 mmHg
- Svo2 > 80%

**BLOOD GAS**

- arterial pO2 > 60 mmHg
- arterial pCO2 < 50 mmHg
- base excess ± 3 meq/L

**RESUSCITATION FLUIDS**

- **NORMAL SALINE** is the best resuscitation fluid. HYPERONIC SALINE is not better* but is practical in battle field as takes less volume to carry in backpacks of paramedics.

*several studies of prehospital fluids in patients with TBI suggested that hypertonic solutions may restore cerebral perfusion, reduce cerebral edema, and modulate the inflammatory response that contributes to neurologic injury, and thus may benefit resuscitation of these patients.

**“Out-of-Hospital Hypertonic Resuscitation Following Severe Traumatic Brain Injury: A Randomized Controlled Trial” Bolger EM, May SJ, Bisel KD, et al JAMA, 2010;304:1489-1496-Largest randomized clinical trial of hypertonic fluid resuscitation following TBI - conclusion: initial fluid resuscitation of patients with severe TBI with either 7.5% saline + 6% dextran 70 or 7.5% saline alone is not superior to NS with respect to 6-month neurologic outcome or survival.
**Regimes, Activity**

General measures for comatose patients: see p. S30 >>

- Neurochecks every 60 mins for 6 hrs, then every hour until stable.
- Mobilize after 24 hrs; avoid direct sunlight.

**Do not overemphasize severity of injury** – unnecessarily prolongs period of invalidism!

- After severe TBI, return to active work should be deferred for 2-3 months.

**TREATMENT GUIDELINES**

- Early nutritional support can directly affect TBI outcome.
  
  **Level II A recommendation**: “Feeding patients to attain basal caloric replacement at least by the 5th day and, at most, by the 7th day post-injury is recommended to decrease mortality.”

  **Level II B recommendation**: “Transgastric jejunostomy is recommended to reduce the incidence of ventilator-associated pneumonia”
  - vs. intragastric (e.g. PEG) feeding
  - waiting 5 days to start enteral nutrition doubles risk of death at 2 weeks (waiting 7 days quadruples risk).

- Severe TBI gives 100-500-fold catecholamine surge → stress ulcers (H: PPI), MI.

**DVT PROPHYLAXIS**

TBI increases risk of DVT significantly and that correlates with TBI severity.

**Level II B recommendation**: “Low dose or low-dose unfractionated heparin may be used in combination with mechanical prophylaxis. However, there is an increased risk for expansion of intracranial hemorrhage. In addition to compression stockings, pharmacologic prophylaxis may be considered if the brain injury is stable and the benefit is considered to outweigh the risk of increased intracranial hemorrhage. There is insufficient evidence to support recommendations regarding the preferred agent, dose, or timing of pharmacologic prophylaxis for DVT.”

- Knudsen et al. found that head injury with an Abbreviated Injury Score of ≥ 3, among other factors, was an independent predictor of VTE in trauma patients.
- TBI has been associated with up to 54% incidence of deep venous thrombosis without prophylactic treatment and a 25% incidence in patients with isolated TBI treated with sequential compression devices.
- Ehreth found that DVT occurred in 1/3 of moderate and severe TBI patients with isolated head injuries, having a lower incidence than those patients with concomitant extracranial injuries. Age, subarachnoid hemorrhage, Injury Severity Score >15, and extremity injury were predictors of DVT.
- Reif et al. demonstrated a 3-4-fold increase in the DVT risk in TBI despite use of mechanical and chemoprophylaxis; VTE risk increases with TBI severity.
- Early (<24 h) initiation of VTE chemoprophylaxis in patients with traumatic intracranial hemorrhage appears to be safe.

**Hemoglobin**

Mainstream Hb > 7 g/dL.


In patients with closed head injury, neither administration of erythropoietin nor maintaining Hb > 10 g/dL resulted in improved neurological outcome at 6 months (vs. Hb > 7 g/dL). Transfusion threshold of 10 g/dL was associated with a decrease of thromboembolic events.

**NEUROLOGICAL MEASURES**

**ICP management**

- “Need to avoid cumulative injury brain” (B). N.B. in absence of any obvious signs of increased ICP, no prophylactic treatment should be initiated!

**Monitoring**

**Level II B recommendation** management of severe TBI patients using information from ICP monitoring is recommended to reduce in-hospital and 2-week post-injury mortality.

- Options (class IIH) for penetrating TBI: early ICP monitoring is recommended if: unable to assess the neurologic examination accurately / the need to evacuate a mass lesion is unclear / imaging studies (e.g. CT) suggest elevated ICP.

**Indications**

<table>
<thead>
<tr>
<th>A: GCS 3-4</th>
<th>B: Salvable patient with GCS 3-8 (after resuscitation) plus:</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) abnormally CT scan - mass lesions (contusions, hematomas) when patient is not taken to operating room for evacuation OR diffuse cerebral edema (esp. with obliteration of perimesencephalic cisterns – limited residual compliance).</td>
<td>b) Normal CT scan + any two of the following (on admission): SBP &lt; 90 mmHg, age &gt; 40 yrs, unilateral or bilateral motor paresis.</td>
</tr>
</tbody>
</table>

- Most academic neurosurgical centers monitor ICP continuously with even moderately severe head injuries.
- Even patients taken to operating room frequently have ICP monitors placed at end of operation - later brain swelling may still be a problem even after successful mass evacuation (and even after craniectomy).
- ICP monitor is ceased at midbrain level.
- If GCS is < 6, EVD is recommended over intraparenchymal monitor to drain CSF for first 12 hours postinjury (level III recommendation).
- There are studies that do not support ICP monitoring; but from practical standpoint, the ICU staff pays much less attention to brain if ICP monitor is absent.

- - systematic review and meta-analysis
- 280 patients included
- 7,107 patients studied
- 7 studies included
- - 6 retrospective (1 prospective)
- 26-247 days follow-up (1 study); 119-365 days follow-up (1 study)
- - mortality: 45.2% vs. 39.2% (P = 0.01) 34.5% vs. 30.1% (P = 0.04) 29.1% vs. 31.6% (N.S.);
- - study by Wu et al. 2015 demonstrated a lower mortality in patients with closed head injury: a systematic review and meta-analysis.
- - mortality: 45.2% vs. 39.2% (P = 0.01) 34.5% vs. 30.1% (P = 0.04) 29.1% vs. 31.6% (N.S.)
Association between ICP monitoring and mortality in patients with TBI stratified by randomization or not (shown as log [Odds Ratio] with SE). N = inverse variance.

- when ICP remains < 20 mmHg for 24–48 hours without treatment, ICP monitoring is discontinued.

**TREATMENT**

- ICP > 22* (formerly 25) mmHg for > 15 mins within 1 hour must be treated ASAP (to keep CPP > 70 mmHg). → see p. 530 >>

  * 15 mmHg after decompressive craniectomy (some would also treat > 18 mmHg for patients > 55 yrs or women of any age).

**Level II recommendation.** Treating ICP above 22 mm Hg is recommended because values above this level are associated with increased mortality.

**Level III recommendation.** A combination of ICP values and clinical and brain CT findings may be used to make management decisions. N.B. don’t guide management by ICP alone; correlate with clinical exam and CT findings!

**VENTILATION**

- in the absence of cerebral herniation, normal ventilation is the goal (PaCO₂ goal 35–45 mm Hg).
  - if hyperventilation is used, jugular venous oxygen saturation (SjO₂) or brain tissue O₂ partial pressure (PaO₂) measurements are recommended to monitor oxygen delivery.

Prophylactic hyperventilation vs. normoventilation in severe TBI

In the normoventilation group, PaCO₂ was kept 30–35 mmHg for 5 days. In the hyperventilation groups, PaCO₂ was kept 24–28 mmHg for 5 days.

Subgroup of hyperventilation received (bolus→ sustained IV infusion for 5 days)

- thrombocytopenia to minimize the effect of loss of CSF buffer during hyperventilation.

**Favourable outcome**

<table>
<thead>
<tr>
<th>3 months</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normoventilation</td>
<td>48%</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>18%</td>
</tr>
</tbody>
</table>

**Statistical significance**

p < 0.05

There were no differences at 12 months follow-up.

Prolonged prophylactic hyperventilation is deleterious in TBI

Deleterious effect of sustained hyperventilation could be overcome by THAM.

**EVD**

- two regimens of EVD:
  a) continuously monitor ICP and only intermittently drain for ICP elevations (“20 pop down to 10” protocol).
  b) continuous drainage of CSF with intermittent ICP measurements – prevalent among pediatric experts and may be more effective in lowering ICP.

**HYDROELECTROLYTE FLUIDS**

- restrict mannitol, use prior to ICP monitoring to patients with signs of transtentorial herniation or progressive neurological deterioration not attributable to extracranial causes.

ICP-guided mannitol vs. empirical mannitol


- high-quality multi-center (South and Central America). Class I RCT (N=324)

- ICP monitor vs. close clinical assessment for ICP management

- outcomes for patients managed with information from clinical assessment do not differ from those for patients managed with information from the ICP monitor – no recommendation to use either method preferentially.

- ICP monitoring alone is not sufficient to improve outcomes.
Ideal hypertensive agent should simultaneously lower ICP and maintain or improve CPP. Osmotic agents may lower CPP related to systemic effects on diuresis, intravascular volume, and cardiac output.

N.B. mannitol is a diuretic and HTS is not.

Growing evidence that hypertonic saline is better for underresuscitated patient than mannitol!

Hypertonic Saline vs. Mannitol


- prospectively collected data from the New York State THI-trace database.
- only patients received only 1 hypertonic agent – either mannitol (20%) or HTS (3% bolus) for raised ICP (patients who received both agents were excluded from data analysis to prevent erroneous conclusions).
- patients were matched (1:1 and 1:2) for factors associated with 2-wk mortality: age; GCS score, pupillary reactivity, hypertension, abnormal CT, and traumaetomy (extra-axial surgical lesions) → 25 matched pairs for 1:1 comparison and 24 HTS patients matched to 48 mannitol patients in 1:2 comparisons.
- cumulative median osmolar doses in the 2 groups were similar.
- primary endpoint - combined burden of ICP high (> 25 mm Hg) and CPP low (< 60 mm Hg).

<table>
<thead>
<tr>
<th>Combined burden</th>
<th>HTS</th>
<th>Mannitol</th>
</tr>
</thead>
<tbody>
<tr>
<td>total number of days (n)</td>
<td>20.8 ± 10.8</td>
<td>2.4 ± 2.3</td>
</tr>
<tr>
<td>percentage of days vs. total days of ICP monitoring (%)</td>
<td>8.8 ± 10.6</td>
<td>38.1 ± 26.9</td>
</tr>
<tr>
<td>total duration (hrs)</td>
<td>11.12 ± 14.11</td>
<td>30.56 ± 31.30</td>
</tr>
</tbody>
</table>

HTS bolus therapy appears to be superior to mannitol in reduction of the combined burden of intracranial hypertension and associated hyperperfusion in severe TBI. Comment (Dr. Henryk): it is important to replace fluid read losses after mannitol administration; sometimes the patient’s ICP responds better to mannitol than to HTS (mannitol is not naturally found – may have better osmotic gradient).

Mangat et al. 2014 study (class 2 evidence) indicated that HYPERTONIC SALINE may be more effective than mannitol in lowering ICP but no difference was found in short-term mortality.

2 mL/kg of 20% mannitol vs. 2 mL/kg of 7.5% hypertonic saline given when either ICP > 25 mmHg or CPP < 70 mmHg (at > 5 min)

<table>
<thead>
<tr>
<th>Hypertonic saline</th>
<th>Mannitol</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of episodes</td>
<td>ICP &lt; 25 mmHg</td>
<td>6.8</td>
</tr>
<tr>
<td>Total duration of episodes</td>
<td>62 min</td>
<td>95 min</td>
</tr>
<tr>
<td>Treatment failure</td>
<td>10%</td>
<td>70%</td>
</tr>
</tbody>
</table>

There was no significant difference in mortality or GOS between the two treatment arms.

N.B. mannitol dose was 0.4 g/kg – lower than we use routinely (1 g/kg)

- pooled data analysis from 3 trials showed continuous HTS therapy was associated with improved survival over bolus HTS therapy.

**SIDEACTIONS**
- **PEPROPON** is recommended for the control of ICP, but it is not recommended for improvement in mortality or 6-month outcomes. (N.B. high-dose propofol can produce significant morbidity).
- **high-dose BARBITURATE** is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment.

Significant positive treatment effect of pentobarbital (p = 0.04)

<table>
<thead>
<tr>
<th>BCT (best conservative treatment) vs. HTS + pentobarbital – for refractory ICP.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control of ICP in all patients (n of patients)</td>
</tr>
<tr>
<td>Control of ICP in patients with cardiovascular complications prior to randomized (n of patients)</td>
</tr>
</tbody>
</table>
Barbiturates help to control ICP but outcome is unchanged (predetermined by primary injury).

**HYPOTHERMIA (THERAPEUTIC)**

- **THERAPEUTIC HYPOTHERMIA (to treat elevated ICP) in severe TBI is effective in decreasing elevated ICP but does not improve neurological outcome and may increase mortality!**
  - hypothermia bears risks of coagulopathy, immunosuppression, and cardiac dysrhythmia.
  - it is suggested that gradual rewarming can mitigate the inherent risk of rebound intracranial pressure elevation.
  - there has been interest in localized cerebral cooling in the hopes of obtaining the desired benefits without the systemic side effects.

**CPP**

MAP by convention is calibrated to the level of the right atrium of the heart. Central venous pressure (CVP) is also calibrated to the level of the right atrium of the heart; but in TBI ICP is higher than CVP.

\[ \text{CPP} = \text{MAP} - \text{IOP} \]

- attention to pressure autoregulatory status; patients with intact autoregulation (PRx < 0.05) are best served by higher CPP values while pressure-passive patients with dysfunctional pressure autoregulation do better with lower CPP values.
- optimal CPP value may need to be tailored to individual patients.

**ADVANCED BRAIN OXYGENATION / METABOLIC STATUS MONITORING**

The goal of the medical management is to ensure that nutrient delivery to the brain is optimized through the period of abnormal physiology and brain swelling - the only way to be assured that this is being achieved is to measure brain metabolites (reassurance that the needs of oxidative metabolism are being met).

- monitor brain oxygenation, rationale – sometimes even if ICP is slightly elevated, brain oxygenation remains normal so no need to treat.
  1. **Global** – jugular venous oxygen saturation (SjO2); avoid SjO2 < 50%
  2. **Regional** – brain tissue O2 partial pressure s. oxygenation (PbrO2) = PbrO2 = PbrO2 = PbrO2 = (FIO2) – indicated for severe TBI.
e.g. “CODMAN Neurotrend Cerebral Tissue Monitoring System” – fiberoptic sensor placed directly in brain tissue monitors local brain tissue Pcri, PcriO2, pH, temperature.

- where to place? – into pia/ventricle, no need to monitor noninjured brain or dead brain areas.

  **normal brain PcriO2 = 25-45 mmHg**

First measure if brain oxygenation falls – increase FiO2; RBC transfusion and increasing CPP are secondary measures.


Threshold of BtpO2 most predictive of mortality was 29 mmHg


Threshold of BtpO2 to avoid was 20 mmHg

**Level III recommendation**: Jugular bulb monitoring of arteriovenous oxygen content difference (AVDO2), as a source of information management decisions, may be considered to reduce mortality and improve outcomes at 3 and 6 months post-injury.

**Level III recommendation**: Jugular venous saturation (SjO2) of < 50% may be a threshold to avoid in order to reduce mortality and improve outcomes.

Although patients with desaturations identified with advanced cerebral monitoring have poorer outcomes, Level II evidence showed no improvement in outcomes for monitored patients.

**BOOST-2 trial** – ICP monitor alone* vs. ICP monitor plus BtpO2:

- having brain oxygenation data available reduced the duration of brain hypoxia:
  - o condition at discharge was better in ICP monitor plus BtpO2 group.

**TCD**

- cerebral autoregulation monitoring.


There is an association between mortality and L-PRx > 0.2.

**Microdialysis**

- monitors cerebral metabolism (e.g. lactate, glutamate)

**SEDATION & ANALGESIA**

Sedative drugs should be avoided if possible (difficult to monitor level of consciousness + depress respiration) but useful for ICP control.

- restlessness may be combated with small doses of short-acting sedatives (e.g. PROMAceM, PRACTED!!!, historical drug - PARALDEHYDE)
- for agitation antipsychotics are useful when used sparingly.
- periodic withholding of sedation (“short sedation vacations”) - to allow periodic neurologic assessment.
- for analgesia – PARACETAMOL, PERCOCET, BUPROPION.
SEIZURE PROPHYLAXIS
- it is prophylaxis of EARLY post-traumatic seizures - anticonvulsant drug is administered routinely in severe TBI and/or cortical irritation but (only) for 7 days see p. E9
N.B. no prophylaxis is seizure suppressant

ANTIBIOTICS
- prophylactically indicated for:
  a) complicated scalp lacerations
  b) open skull fractures (includes penetrating head injury) – 5 days of triple antibiotics
  c) CSF leak persisting > 7 days

MEDICATIONS NEEDING FURTHER TESTING

BETA-BLOCKERS
- observational studies reveal a significant mortality advantage with β-blockers; however, quality of evidence is very low.

- severe TBI and/or cortical irritation but
- seizures are not randomized controlled trials

- some evidence is very low.

- increased ICP is due to vasodilation and cytotoxic edema (not due to vasogenic edema)

- mortality approaches 100%

- more details for posterior fossa mass lesions

- more details for IPH

- patients with severe TBI, high-dose methylprednisolone was associated with increased mortality and is contraindicated.

- case unclear but not due to infections or GI bleeding; RR for death 1.15 (p=0.0001)

NOT RECOMMENDED MEDICATIONS

1. Steroids – increased ICP is due to vasodilation and cytotoxic edema (not due to vasogenic edema);

- no human studies demonstrated benefit (e.g., in temporal or posterior fossa)

- mortality approaches 100%

- more details for posterior fossa mass lesions

- more details for IPH

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- more details for posterior fossa mass lesions

- midline shift > 5 mm (shift > 20 mm – mortality approaches 100%)

- neurodeficits (e.g., GCS < 9, GCS decrease by ≥ 2 points, focal neurologic signs, refractory ICP > 20 mmHg, effaced / compressed basal cisterns, heterogeneous clot on CT [indicates active bleeding])

N.B. in general, any neurodeficit due to extra-axial hematoma is indication for surgery!

SURGERY

MEDICATIONS

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SURGERY

SEE p. Op320

INDICATIONS FOR SURGERY


2. Certain open skull fractures: see p. TiH5

3. Certain depressed skull fractures: see p. TiH5

4. Extra-axial (epidural / subdural) or intra-axial hematoma / hemorrhagic contusion: see p. TiH5 > p. TiH3
2. N.B. (by CT criteria) must have better morbidity* and mortality outcomes vs. delayed surgery (by clinical deterioration criteria)?

3. Physical & occupational therapy

4. Cognitive rehabilitation

5. Symptomatic chronic subdural hematoma. see p. TH13 >>


7. Medically refractory intracranial hypertension → decompressive craniectomy.

METHOD TO MEASURE HEMATOMA VOLUME

X = ABC/2

- identify the CT slice with the largest area of hemorrhage (Slice 1)
- measure the largest diameter, A
- measure the largest diameter 90° to A on the same slice, B
- count the number of 10-mm slices - compare each slice with slice:
  if the hemorrhage is greater than 75% compared with slice 1, count the slice as 1.
  if the hemorrhage is 25 to 75%, count the slice as 0.5.
  if the hemorrhage is less than 25%, do not count the slice.

Add up the total C.

REHABILITATION

Guidelines by Texas Head Injury Foundation

1. Treatment should be adjusted for patient level of function.
2. Treatment should be consistent, repetitions, and structured.
3. As patient’s ability improves, difficulty of task should be increased and degree of structure should be decreased.
4. Results of performance should be consistently provided to patient.
5. Information should be presented via more than one sensory avenue.
6. Information and tasks should be relevant to needs and interests of patient.

Treatment of spasticity → see p. Mov3 >>

Aim to physical & cognitive independence (i.e. rehabilitation is continued until optimum training has been achieved or patient has become independent)

- rehabilitation is creative, cooperative effort of health-care team, patient, and family.
- family members must be kept informed of progress; they usually wish to learn rehabilitation techniques, and instruction should be provided (family will assume primary responsibility in outpatient therapy).
- rehabilitation should begin as soon as patient is stable.
- relearning old skills / learning methods to compensate for lost skills.

1. Physical & occupational therapy
- functional activities out of bed are begun as quickly as possible!

2. Cognitive rehabilitation
- cognitive enhancing medications (still experimental):
  1) AMANTADINE – efficacy proven by double-blind, placebo-controlled, randomized study – speeds up recovery but final outcome the same.
  2) METHYLPHENIDATE – improved motor outcomes and attention.
  3) DOMEPKIDE – improved visual and verbal memory as well as attentional deployment.
  4) LEVODOPA – alerting responses in vegetative or comatose patients.

* e.g. ischemic effect of prolonged cortical compression by hematoma
Bisphosphonates

5.

4.

V.

II.

Urinary Heterotopi

Spastic contractures

Malnutrition

Decubitus ulcers

Treatment with gastric coating agents (sucralfate), H2 receptor blockers, frequent antacid administration.

Deep venous thrombosis

Adequate cardiac output is essential to ensure cerebral perfusion!

GI Hemorrhage: from gastric stress erosions/ulcers. Most patients with severe TBI develop gastric erosions, but only few have clinically significant hemorrhages.

GI bleeding usually occurs in first days to 1 week after injury.

H: pylorid gastritis & treatment with gastric coating agents (sucralfate), H2 receptor blockers, frequent antacid administration.

Paralytic ileus

Bowel incontinence - caused by underlying constipation, impaired communication and mobility (H: stool softeners, laxatives, rectal suppositories).

Cardiac.

TBI (esp. with intracranial bleeding) can cause primary cardiac dysfunction - variety of rhythm, rate, conduction abnormalities - can be life threatening?

Advanced cardiac output is essential to ensure cerebral perfusion!

Hemorrhagic:

Large number of patients demonstrate mild coagulopathy.

N.B. multiple contusions detected by first CT scan = trauma-related coagulopathy that will correct itself within few hours.

5-10% (up to 90% patients with severe TBI) have various degrees of DIC.

DIC can develop within hours after any injury disrupting tissue release (of tissue thromboplastin into systemic circulation).

Decreases risk of delayed intracranial hemorrhage.

Any coagulation abnormality must be reversed.

Urinary:

1. Urinary tract infections

2. Urinary incontinence - caused by UTI, impaired communication and mobility (i.e. overflow incontinence; H: patient is taken to bathroom and given opportunity to void without instrumentation every 2 hours during day and every 4 hours overnight; if unable to void or unable to evacuate urinary bladder to completion = intermittent catheterization).

Longer term complications

1. Deep venous thrombosis, pulmonary embolism

2. Decubitus ulcers

3. Malnutrition

4. Spastic contractures - can be prevented with aggressive physical therapy.

5. Heterotopic ossification (11-76%): epycotic bone formation in soft tissue surrounding joints or bone and paraspinal muscle.

Pathophysiology: inappropriate differentiation of mesenchymal cells into osteoblasts; contributing factors - autonomic dysregulation (due to increased vascularity and venous hemostasis), humoral factors, local inflammatory mediators.

Risk factors: posttraumatic coma lasting > 2 weeks, lamb spasticity, decreased mobility.

Risk is greatest during first 3-4 months.

Clinically: joint pain, decreased range of motion*, low-grade fever & perianal swelling-Warmth-squamous.

*although ossification does not originate in joints, ankylosis occurs in ~20% cases.

Locating (in decreasing order of frequency): hips, knees, elbows, shoulders, hands, spine.

Ultrasound:

1) serum alkaline phosphatase! and ESR! = nonspecific markers in early phases.

2) triple-phase bone scanning - earliest diagnosis!!

3) plain radiography - lag behind triple-phase bone scan results by 2-3 weeks.

Sonography

Phlebitis: range of motion (ROM) exercises; unclear role - NSAIDs, low-dose radiation.

Bisphosphonates: use of bisphosphonate RCT is controversial because it is thought to be cause of heterotopic ossification, but data from human studies have not demonstrated this mechanism.

Treatment: 1) bisphosphonates - prevent crystallization of calcium phosphate (but have no effect on mature ectopic bone).

2) for functional impairment - surgical excision delayed 12-18 months to allow heteropic bone to mature (to minimize risk of recurrence).
1. Cerebral fat embolism see p. Vas3 >>
2. Epidural Hematoma see p. Th11 >>
3. Subdural Hematomas see p. Th13 >>
4. Arterial rupture, occlusion, dissection see p. Vas11 >>
   - high mortality - 23-43%.
ICA most commonly injured at skull base
   - manifests as pain, pharyngeal bleed, cranial nerve palsies, distal thrombemboli
VA most commonly during cervical motion that injures vessel intima
   - causes may be trivial (chiropractic manipulations, violent sports)
   - unilateral VA injury may be asymptomatic (but thrombosis may extend to basilar)
5. Venous sinus injuries see p. Op320 >>
   N.B. posterior fossa / occipital EDH may compress venous sinuses and imitate venous sinus thrombosis on imaging – erroneous heparin administration may cause more harm (e.g. EDH expansion).
6. Carotid-cavernous fistula see p. Td9 >>
7. SAH - most common type of traumatic intracranial hemorrhage (some blood extravasation into subarachnoid spaces is to be expected in almost any head injury; present on CT in 26-53% of severe TBI cases):
   a) tears of subarachnoid vessels (most frequently leptomeningeal vessels at vertex - greatest brain movement at impact).
   b) blood from ventricles (reached subarachnoid space with CSF flow)
   - in most cases, SAH does not indicate need for surgical intervention (except to indicate that brain has been injured) - no specific treatment is needed.
   - but SAH doubles risk of death (because of associated lesions) >>
   - detected by CT in 26-53% cases of severe TBI, repeat CT - to exclude possibility of additional intracranial occulted bleed.
   - traumatic SAH is typically located in interhemispheric or sylvian fissure. cerebral sulci (vs. SAH from ruptured cerebral aneurysm - primarily in basal cisterns).
   - differentiation of interhemispheric blood from relatively attenuating fluid cerebri may be difficult (N.B. blood extends into paramedian sulci).
   - with extensive SAH, brainstem, infratentorial, carotid branches are bathed by blood, and they may appear as filling defects. N.B. SAH may render basal cisterns isodense with brain, simulating cisternal compression!
   - traumatic basal SAH - due to blow to side of chin or jaw in alcohol-induced fistfight - ruptures vertebral artery as it enters cranial cavity (degree of traumatic force required to cause basilar SAH is less than reasonably expected).
   - complications:
     1) communicating hydrocephalus.
     2) vasospasm (most intense during 3rd week after TBI) – delayed ischemia, infarction. H: NSIMOPHR

8. Chronic cefalocedal scalp hematoma (seen chiefly in older patients prone to repeated head injuries, such as epileptics and recidivist alcoholics); underlying vault may be thickened.
9. Intraventricular hemorrhage (1.5-10%) - ruptured subependymal veins.
   - intraventricular blood is indicator of severe TBI.
   - unilateral left blood may in layer dependent part of ventricles.
   - hydrocephalus is a risk (N.B. ventricles may remain small due to compression by brain edema!)
10. Cerebral thrombosis
   - ARTERIAL THROMBOSIS - caused by injury to artery wall.
   - thrombosis of intracranial vessels is rare complication of brain distotion by epidural or subdural hematoma.
   - thrombosis occasionally develops several days after TBI in elderly patients with cerebral arteriosclerosis (it is difficult to assess role of cerebral trauma in production of these strokes).
   - VENOUS SINUS THROMBOSIS - severe ICP elevations and venous infarction.
   - treatment – anticoagulation (significant risk in those with acute head injuries!); if thrombosis progresses – direct intracranial intravenous thrombolysis.
11. Traumatic aneurysms (< 1%)
   - etiology:
     a) penetrating vessel injuries (stab wounds > gunshot wounds) → pseudoaneurysms at distal sites in intracranial circulation (can appear weeks to months after injury).
     b) blunt vessel injuries → true aneurysms (intact adventitia); examples:
       1) head hyperextension and rotation → aneurysm on ICA at skull base;
       2) closed head injury with skull fractures → aneurysm on peripheral intracranial vessel (esp. distal ACA, e.g. pericallosal artery - traumatized at falkine edge)
   - present with delayed SAH or intracerebral hematoma.
   - diagnosis - angiographically absolutely indicated ASAP in stab injuries! (intracerebral hematoma may harbor and simultaneously obscure traumatic aneurysm)
   - in CT / MRI era, angiography is rarely employed in evaluating head trauma - discovery of traumatic aneurysm is usually delayed until unexpected neurological event occurs.
   - traumatic aneurysms are unrelated to arterial bifurcations.
   - treatment (best results if still unruptured) - surgical or endovascular.
     - anticoagulation for pseudoaneurysms may minimize thrombus propagation and embolization (controversial).

Acute traumatic arterial aneurysm (carotid angiogram, lateral projection): metallic fragments are seen posteriorly;
vessels around them are stretched and narrowed, and rounded collection of contrast medium, representing traumatic aneurysm, is seen (arrow), unlinked to arterial bifurcation.
12. Intracerebral hemorrhage (ICH) (42.3%); see also p. Vas20 >>

- Differences between spontaneous ICH (sICH) and traumatic ICH (tICH):
  - sICHs are more likely to be lobar
  - tICHs are more likely to be superficial
  - tICHs are more likely to have a medium-sized volume (25–65 cc).

- **countered by**
  a) shearing forces that mechanically stretch and tear deep small-caliber arterioles – resulting small petechial hemorrhages subsequently coalesce to form ICH.
  - 85% in frontal and temporal lobes; multiple in 20% cases.
  - on occasion, ICH (ipsi- or contralateral) develops after other intracranial hematoma evacuation
  b) penetrating injury – occur wherever vessels are interrupted along tract of missile.
  - occur with all degrees of TBI; linear fracture is found in 40-80% cases.
  - most prominent in white matter (vs. contusions)
  - reach maximum size by 2-3 days following head injury.

Clinical features:
- 50% of patients sustain loss of consciousness at time of impact; some regain consciousness for lucid interval, and 1/3 have no loss of consciousness at all.
- focal brain injury (similar to contusion or extradural collections).
- brain compression → herniation.

Diagnosis - CT (hemorrhagic contusion + large blood collection) - focal area of increased attenuation*, surrounded by low attenuation (edema or contusion); irregular and may have mass effect; over time becomes well demarcated, isoattenuating and then hypoattenuating.

- ICH is made up of > 66% clots – appears more homogenous than contusion!

DELAYED HEMORRHAGE (DICH – DELAYED TRAUMATIC ICH) vs. BOLINGER’S SPATIAL APPLIQUÉ (ICH occurs in 1-7% TBI patients) – ICH that develops in the part of the brain that on the initial CT looked “normal”; may manifest as apoplectic event even after 10 days of asymptomatic interval.

Yamaki et al. (1990): only 84% of all ICHs reached the maximum size within 12 hours from initial CT scan!

- Noncontrast CT – occipital and temporal intracerebral hematomas, surrounded by mild edema and hemorrhagic contusions; small intrhemispheric subdural hematoma in posterior intrhemispheric tissue; obvious midline shift.

**TICHs**

- Associated with blood extravasation into subdural space, which occurs wherever vessels are interrupted along tract of missile.
- May manifest as apoplectic event even after 10 days of asymptomatic interval.

**Treatment:**
- a) conservative management
- b) surgical evacuation (for indications – see above) see p. Vas20 >>

Pathophysiological pros for early surgical evacuation:
- contused brain does not recover but appears as encephalomalacic brain tissue loss on convalescent phase imaging - removing TICH does not increase tissue loss.
- extravasated blood is believed to be neurotoxic, leading to secondary injury that may be avoided by surgical removal.
- larger TICHs may be associated with an ischaemic penumbra of brain tissue that could be salvaged.
- some TICHs expand to the point at which they cause mass effect with high ICP, resulting in secondary brain injury.

Surgical Trial In Traumatic intraCerebral Haemorrhage (STICH)

Gregson BA et al. Surgical Trial In Traumatic intraCerebral Haemorrhage (STICH); a randomized controlled trial of Early Surgery compared with Initial Conservative Treatment. Health Technol Assess 2015;19(70).

- randomized trial: Early Surgery (within 12 hrs) vs. Initial Conservative Treatment (delayed evacuation if it became clinically appropriate); ICH > 10 mL
- cf. STICH trial – nontraumatic ICH – see p. Vas20 >>
- 170 patients were randomised from 31 centres in 13 countries
- clinical equipoise - only patients for whom the responsible neurosurgeon was uncertain about the benefits of either treatment were eligible.
- 85% of patients had a motor score on the GCS of ≤ 5.
- volume of the largest hematoma was 10-97 ml, with a median of 23 ml.

**Early Surgery** may be a valuable tool in the treatment of ICH, especially if the GCS is 9-12 – analogy with SDH / EDH management; those with GCS of 13-15 probably can be watched carefully for any deterioration; if GCS has dropped below 8, surgical intervention appears to be less effective.

At 6 months:
- Early Surgery patients were 10.5% more likely to have a favorable outcome (absolute benefit), but this difference did not quite reach statistical significance because of the reduced sample size (63% vs. 53%, odds ratio 0.65; 95% confidence interval (CI) 0.35 to 1.21; p = 0.17).
Mortality was significantly lower in the Early Surgery group (15% vs. 33%; absolute 18.3%; 95% CI 5.7% to 30.9%; \( p = 0.006 \)).

Mathiesen et al. (1995): patients with an admission GCS of \( \geq 6 \) and a lesion volume of \( \geq 20\, \text{ml} \) who had surgery without previous neurological deterioration had significantly better outcomes than those who did not have surgery or had surgery after deterioration. None of the patients who had surgery before any deterioration died or was vegetative, as opposed to 39% of those who had surgery after deterioration and 50% of those who did not have surgery.

Choksey et al. (1993): 38% of patients with a low GCS and a volume of the TICH > 16 ml who had surgery had a poor outcome, compared with 56% of those who did not have surgery.

Zumkeller et al. (1992): poor-outcome rate in the operated patient group was 29%, compared with 59% in the non-operated group.

Other studies (observational):


Cranial hemiatrophy - left hemicranium is smaller (arrows indicate groove for superior sagittal sinus); left petrous bone is elevated, and paranasal sinuses are more extensive on affected left side.

II. INFECTION

etiology / risk factors:

1) penetrating injuries.

2) skull fractures - open depressed fractures, basilar fractures with CSF leak, linear fractures that extend into paranasal sinuses or middle ear.

3) iatrogenic - ICP monitoring, surgical interventions.

Contusions, intracerebral hematomas may show remarkable resolution, but more common end results are focal atrophy or encephalomalacia (extensive softening and cyst formation) – MRI may show evidence of past hemorrhage (prolongation of T1 and T2).– injury early in life (vascular damage rather than to direct trauma) – traumatic hemiatrophy - affects both brain and overlying skull.

Cranial hemiatrophy - left hemicranium is smaller (arrows indicate groove for superior sagittal sinus); left petrous bone is elevated, and paranasal sinuses are more extensive on affected left side.

IL INFECTION

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1. Cranial (ostomyelitis) see p. Inf73

2. Epidural (abscess) see p. Inf73

3. Subdural (empyema) see p. Inf75

4. Subarachnoid (meningitis) see p. Inf73

5. Intracerebral (abscess) see p. Inf73

- head injury (general)
III. CSF & MENINGES

1. CSF leaks
- 1.5%, but 5-10% in basal skull fractures
- p. S64

2. Post-traumatic hydrocephalus
- Communicating vs Noncommunicating — due to SAH, intraventricular hemorrhage
- p. S60
- Unexplained hydrocephalus after acute TBI — prompt thorough examination of posterior fossa (epidural or subdural hematomas may be missed on routine axial CT scans)
- Normal pressure hydrocephalus may also occur — diagnosis is difficult:
  - p. S62
  - > 85% severe TBI patients demonstrate some degree of ventriculomegaly on follow-up CT (secondary to diffuse brain atrophy)
  - TBI patients often have memory difficulties and gait abnormalities secondary to head injury
- N.B. Normal pressure hydrocephalus presents as neurologic deterioration weeks to months following TBI; high-volume lumbar puncture improves neurologic condition.

3. Subdural higroma
- p. TDI5

4. Leptomeningeal cyst
- p. TDH

5. Pneumocele
- A pneumocele:
  - Air in cranial cavity after missile wounds penetrate sinuses.
  - a) Frontal pneumocele (after fracture of frontal sinus)
  - b) Occipital pneumocele (after fracture through mastoid)
- Free air can also be sucked into penetration cavity behind projectile.
- Clinically:
  - a) Asymptomatic
  - b) Headaches, mental symptoms
- N.B. Signs of ICTP do not develop (unless pneumocele becomes infected or filled with CSF)
- Diagnosis:
  - X-ray, CT; air may not appear for several days after injury (and then only after patient sneezes or blows nose)
  - Treatment (rarely indicated per se; if spontaneous air absorption does not occur): transfrontal craniotomy → opening in frontal sinus is covered with strip of fascia lata or pericranial flap.

IV. POST-TRAUMATIC EPILEPSY

V. POST-TRAUMATIC MOVEMENT DISORDERS

VI. POSTCONCUSSION / (S. POSTTRAUMATIC) SYNDROME

- Frequent (30-50%) sequel of mild head injury
- Rare in children (but symptoms tend to be more severe)
- Diagnosed in DSM-IV as cognitive disorder not otherwise specified.

PATHOPHYSIOLOGY

1. Alterations in excitatory amino acids, catecholamines, cations (similar to migraine)
2. Subtle axial shearings lesions
3. Microscopic cortical contusions.

Even mild trauma can cause neuronal damage!

CLINICAL FEATURES

- Large array of symptoms and signs (at least 3 months duration): most common:

  Syndrome can be extremely debilitating!
  1. Headaches (30-90%):
     - Headaches are more frequent and with longer duration in mild rather than severe head injury
     - Trigeminal can abort chronic post-traumatic headaches (PTHs) attributable to mild head trauma.
     - Topiramate appears to be an effective headache prophylactic therapy in chronic PTH.
     - Low doses of tricyclic antidepressants appear to have little efficacy.
   a) Tension-type headaches (85% of all post-traumatic headaches); one cause may be temporomandibular joint injury.
   b) Migraine-like (very common) can develop in hours to weeks after mild TBI
     - Immediately after mild TBI in sports children and young adults may have first-time migraine with aura; this may be triggered multiple times after additional minor injury—turned rotor’s migraine
   c) Referred headache from neck injuries (myofascial, intervertebral disc, facet joint injury)
   d) Greater occipital neuralgia from direct blow to nerve or due to suboccipital muscle spasm (superior trapezius, semispinalis capitis).
  H. Local anesthetic nerve blocks (can be combined with injectable corticosteroids).
  2. Dizziness (34-50%) — caused by: labyrinthine concussion, benign positional vertigo, brain stem injury.
  3. Nonspecific psychological symptoms (> 50%): fatigue (43%), irritability, anxiety, depression, disruption of sleep patterns, loss of concentration and memory (20-40%), light/ease sensitivity (10%), post-traumatic stress disorder.
  - Neuropsychological testing has documented cognitive impairments (reduction in information processing speed, attention, reaction time, and memory for new information).
  - Blurred vision (14%) usually caused by convergence insufficiency.
  - Hypoesthesia (> 5%) caused by damage to efferent (flavuncular).
  - No direct correlation between TBI severity and development of PCS — symptoms may develop in patients who were only dazed (did not lose consciousness) by injury.
  - On objective examination - minimal or no neurologic abnormalities (fixed neurologic deficits are not part of PCS).

DIAGNOSIS

- Neuro-psychological testing!!!
  - MRI rule is not yet defined - symptoms may appear in absence of any imaging evidence of structural abnormality!
  - N.B. CT / MR may be warranted to rule out delayed complications of TBI (e.g. chronic subdural hematoma may mimic PCS)
  - EEG, evoked potentials are also normal.
  - When patients have unusual / persistent complaints, consider possible contributions of personality disorders, psychotic problems, or secondary gain ("compensation neurosis" or malingering).
- "N.B. patients with claims in compensation case, however, have similar symptoms that improve with time and similar cognitive test results as those without claims (for many
TREATMENT

Simple reassurance is often major treatment - inform patient that cognitive-emotional dysfunction, headaches and other somatic symptoms are not uncommon and that most patients improve after 1-6 months!

1. Supportive psychotherapy
2. Cognitive retraining (see below (REHABILITATION))
3. Physiotherapy
4. Antidepressant and antidepressant-type medications
5. Headaches often respond to

   a) PROPRANOLOL (30-60 mg/d in three divided doses), or occasionally to calcium channel blockers
   b) SSRIss
   c) tricyclic antidepressants

PROGNOSIS

- symptoms usually last 2-6 months (typically, peak 4-6 weeks following injury), but symptoms may persist for years (some are disabled permanently); risk factors for persistent symptoms:
  1) women
  2) age > 40 years
  3) prior history of head trauma
  4) neurotic symptoms before injury
  5) domestic or financial difficulties.
- 2 years after injury, 20% patients still complain of headaches.
- cognitive deficits usually resolve within 3 months after injury.

VII. POSTTRAUMATIC PSYCHIATRIC DISORDERS

Serious residual mental problems are found only after severe TBI.

- almost every patient with severe TBI shows mental changes immediately after recovery of consciousness (frequently steps to complete recovery are semistupor, bewilderment, Korsakoff-like phase, euphoria).

1. Posttraumatic agitation / aggression is common (≈ 25% patients are found to be aggressive during follow-up period of 5 years).
   - associated with frontal injuries, esp. severe TBI
   - consistently associated with depression, young age, treatment steps.
   1) first treat / eliminate agitating factors:
      - impaired recognition and inability to communicate are often agitating factors.
      - pain
      - infection
      - electrolyte imbalance
      - adverse effects of drugs (esp. avoid centrally acting drugs).
      - psychosis, insomnia.
   2) environmental modifications - tools for orientation, minimizing unnecessary stimuli (noisy rooms, bright lights, frequent visitors).
   3) drugs: high-dose β-blockers (particularly PROPRANOLOL), anticonvulsivants (e.g. LAMOTRIGINE, DEPAVPROX), antidepressants (particularly SSRIs), AMANTADINE.

   Antipsychotics are controversial - may cause excessive drowsiness, exacerbate cognitive deficits, and inhibit neuronal recovery.
   - restraints - use only as last resort to secure patient, staff, and visitor safety
   - Physical restraints often exacerbate posttraumatic agitation!
     - less restrictive restraints, such as net-covered beds (e.g. Posey or Vail beds), has become acceptable and popular.
   5) DBS (placed bilaterally in the nucleus accumbens and anterior limb of the internal capsule to modulate the prefrontal cortex) is promising.
     - DBS lead (inset) is directed through the anterior limb of the internal capsule and striatum, passing between the caudate (green) and putamen (red) with the tip in the nucleus accumbens (blue).

2. Posttraumatic depression - common sequela of TBI (major depression is found in 40-44% hospitalized patients).
   - associated with left frontal injuries.
   - may lead to cognitive decline, anxiety disorders, substance abuse, dysregulation of emotional expression, aggressive outbursts.
   - treatment:
     1) early grief reaction is better treated with supportive therapies
     2) METHYLPHENIDATE
     3) SERTRALINE
     4) AMANTADINE
   - N.B. if drugs are used, carefully consider adverse effects (esp. worsening sedation or cognitive impairment).

3. Psychosis
   - transient psychotic episodes are not uncommon, but long-continued psychosis is rare.
   - relationship of head injury to subsequent development of psychoses is medicolegal problem (severe TBI may adversely influence preexisting brain pathology and accentuate organic dementia).
   - unlikely that TBI could have any direct causal relationship to psychoses that do not have definite structural pathology (e.g. schizophrenia, manic-depressive), but indirect relationship with chemical intermediaries may be postulated.

4. Personality change sometimes results from head trauma, even in absence of obvious cognitive changes.
Hippocratic aphorism: “No head injury is so serious that it should be despised of nor so trivial that it can be ignored.”

- adults reach maximum recovery by ≈ 6 months. smaller adjustments continue for perhaps as long as 2 yrs (recovery in children takes even longer).
- common long-term deficits are cognitive (recent memory, abstract thinking, rapid information processing, etc.)
- N.B. neuropsychologic disturbances (e.g. postconcussional syndrome) are more common cause of disability in social relations and employment than are specific neurologic deficits!
- driving independently is of particular importance to most patients:
  - estimating driving ability on basis of cognitive testing is difficult (although visual spatial skills, reaction times, and awareness of deficits correlate with driving abilities).
  - actual road tests may be needed.
- moderate TBI:
  - at 3 months after trauma - 70% are unable to return to work, 90% have memory difficulties, > 90% have persistent headaches.
- severe TBI:
  - mortality decreased from 50% to 25% over the last decades.
  - only 7% have good outcome with moderate disability.
  - most are left with at least some residua (small percentage remain in persistent vegetative state); severe cognitive difficulties are rule rather than exception.

5. Most powerful factors predicting outcome

- Notably, of these five major predictors, only the hypotension is amenable to medical manipulation!

1. TBI degree (as measured by initial postresuscitation GCS score) - early predictor of patient’s overall outcome!
   - up to 85% of patients with GCS 3–4 die 24 h after injury, yet number of patients with poor initial prognosis (incl. absent pupillary light responses) survive, suggesting that aggressive management is justified in virtually all patients!
   - if the initial* GCS score is reliably obtained and not tainted by prehospital medications or intubation, approximately 20% of the patients with the worst initial GCS score will survive and 8%–10% will have a functional survival (GOS 4–5).

2. Increasing age - independent predictor of poor outcome!
   - the critical age threshold for worsening prognosis appears to be above 60 in a review of Class I and II studies (this may be an artifact of the age grouping used by various authors in converting continuous data into categorical data); some studies report outcome as a continuous function of age without threshold values.
   - aged brain:
     1) decreasing capacity for repair after TBI
     2) increasing frequency of intracranial hematomas with the largest intracerebral hematomas observed in the oldest groups.
   - type of injury that occurs frequently in each age group (e.g. increasing proportion of falls and pedestrian accidents with advancing age).
   - decline in health as one ages may predispose the aged to systemic complications after TBI.
   - children outcome is better than that of adults (patients < 20 yrs are > 3 times more likely to survive than those > 60 yrs).

3. Fixed dilated pupil; see criteria for pupillary exam >>

% Vegetative/Dead (Glasgow Outcome Scale Score [GOS] 1, 2):

<table>
<thead>
<tr>
<th>First Author</th>
<th># of Patients</th>
<th>Bilateral Reactive Pupils</th>
<th>Unilateral Unreactive</th>
<th>Bilateral Unreactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jennett '76</td>
<td>600</td>
<td>42%</td>
<td>3%</td>
<td>55%</td>
</tr>
<tr>
<td>Braakman '80</td>
<td>365</td>
<td>29</td>
<td>54</td>
<td>90</td>
</tr>
<tr>
<td>Heiden '83</td>
<td>213</td>
<td>36</td>
<td>91</td>
<td></td>
</tr>
<tr>
<td>Marshall '91</td>
<td>746</td>
<td>32</td>
<td>34</td>
<td>74</td>
</tr>
<tr>
<td>Average</td>
<td>35</td>
<td>44</td>
<td>88</td>
<td></td>
</tr>
</tbody>
</table>

% Good Recovery/Moderate Disability (GOS 4, 5):

<table>
<thead>
<tr>
<th>First Author</th>
<th># of Patients</th>
<th>Bilateral Reactive Pupils</th>
<th>Unilateral Unreactive</th>
<th>Bilateral Unreactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jennett '76</td>
<td>600</td>
<td>50%</td>
<td>3%</td>
<td>5%</td>
</tr>
<tr>
<td>Heiden '83</td>
<td>213</td>
<td>49</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Levin '90</td>
<td>259</td>
<td>53</td>
<td>17%</td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>51</td>
<td>4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*One or both pupils unreactive.

EDH vs. SDH:
- in EDH with bilateral fixed pupils mortality is only 56% compared to an average of 88% in SDH

PROGNOSIS, OUTCOME

- depends most on nature, site and severity of brain damage (than on injury to skull or scalp).

TBI may result in death, vegetative state, partial recovery, or full return to work.
patients operated on for EDH with bilaterally fixed pupils - 18% had a poor outcome (GOS 1-2) vs. 64% in SDH with bilaterally fixed pupils, delay of > 3 hours in evacuating a traumatic intracranial hematoma with bilateral pupil changes increased the chance of a poor outcome from 40% to 63%.


4. Hypotension - single measurement of SBP > 90 mm Hg.

Hypotension has 67% PPV for poor outcome (79% when combined with hypoxia).

in one study, logistic regression modeling revealed that early hypotension (injury = resuscitation) was responsible for a 15-fold excess mortality and late hypotension (intensive care unit) for an 11-fold excess mortality in severe TBI.


influence of intracranial hemorrhage episodes in patients with severe TBI who had not otherwise been hypotensive - patients with retroperitoneal hypotension had significantly worse neurologic outcomes than those without.


5. CT Classification and Outcome on Discharge

Class I evidence:

- mortality when cisterns are absent - 77% (39% when cisterns are compressed; and only 22% when cisterns are open).

- mortality is higher in acute SDH than in EDH.

- in comparison to diffuse brain swelling, however, no direct relation was seen between the status of the third ventricle and that of the basal cisterns.

- presence and type of intracranial lesions.

- presence of even one of

1) compressed or absent basal cisterns measured at the midbrain level (i.e. perimesencephalic cisterns) – the strongest independent prognostic factor.

2) SAH (on the basal cisterns, over the convexity) – the second strongest independent prognostic factor.

Presence of SAH = 2-fold increase in the risk of dying

- class I evidence: 69-72% PPV for unfavorable outcome with SAH in the suprasellar or ambient cisterns (61% PPV with convexital SAH).

- Fisher’s Grade 3 = 62% PPV toward unfavorable outcome; Fisher’s Grade 4 = 79% PPV.

3) midline shift at the level of foramen of Monro.

- PPV of 70% for mortality with midline shift > 15 mm (class II evidence).

- PPV of 78% to poor outcome with shift > 5 mm in patients > 45 yrs (class I evidence). prog.

- midline shift is more or less uniform with midline shift out of proportion to the extent of the intracranial hemorrhage.

- presence and type of intracranial lesions.

4) mortality is higher in acute SDH than in EDH.

5) mortality is more favorable in EDH than in diffuse trauma.

6) mortality volume is correlated to outcome.

- if CT is normal on admission, outcome is primarily related to concomitant extracranial injuries.

- the full extent of intracranial pathology may not be disclosed on early CT (the absence of abnormalities on CT at admission does not preclude the occurrence of raised ICP, and significant new lesions may develop in 40% of patients).


CT Classification and Outcome on Discharge (Marshall et al. 1991) Marshall classification >>

Number of Patients

Unfavorable Outcome

Favorable Outcome

(Diffuse, SAH, ICH)

(0, 35, 50)

Diffuse Injury I

52

38%

62%

Diffuse Injury II

177

65

35

Diffuse Injury III

153

84

16

Diffuse Injury IV

142

4

6

Evacuated Mass Lesion

276

77

23

Non-Evacuated Mass Lesion

36

89

11

OTHER PROGNOSTIC FACTORS

1. Penetrating TBI (vs. closed TBI – mortality 2.5 times lower), esp. gunshot TBI.

2. Multiple organ injuries - influence on the outcome of severe TBI is primarily mediated through hypotension.

3. Anticoagulants / antiplatelets - raise risk of intracranial bleeding with even trivial TBI.

Novel oral anticoagulants and trauma


- prospective observational trial across 16 trauma centers, 1,847 patients on antiplatelets / anticoagulants

- placebo control group

- 50% of patients were on antiplatelet agents, 33% on warfarin, 10% on NOAs (dabigatran, rivaroxaban, or apixaban), and 7% on combination therapy or subantigenic agents.

- patients taking NOAs were not at higher risk for ICH on univariate (24% vs. 31%) or multivariate analysis (incidence rate ratio, 1.27; confidence interval 1.13–1.43; p = 0.05).

- compared with all other agents, aspirin (99%, 81 mg; 102.5 mg) gave the highest rate (35%) and risk (incidence rate ratio, 1.27, confidence interval, 1.13–1.43; p < 0.001) of ICH.

- progression of ICH occurred in 17% of patients and was not different between medication groups.

- mortality was 7% and was not significantly different between medication groups.

- Presence of even one of APoE4 alleles - 14-fold greater likelihood of poor outcome.
GOS can be divided

GLASGOW OUTCOME SCALE

mild TBI

function)

FUNCTIONAL INDEPENDENCE

12.

11.

10.

9.

8.

7.

6.

5.

4.

3.

2.

1.

PBI versus TBI outcome distribution (on GOS scale – “J” shape vs. “U” shape):

• among survivors, the percentage of patients with favorable outcome (“moderate disability” plus “good recovery”) is equal: 74%, i.e. the main difference in outcome is the difference in mortality

N.B. in PBI, the outcome distribution differs significantly (from blunt TBI) - overwhelming majority of patients have an unfavorable outcome; mostly, death - appropriate outcome measure for PBI is mortality, not GOS score.

1. Increasing age correlates with increased mortality (Class III).
2. Suicide correlates with a higher rate of mortality than other causes of PBI (Class II); plus, suicide patients are less likely to receive aggressive resuscitation efforts.

N.B. patients who recover from attempted suicide frequently report relief over survival and express gratitude to their caregivers!
3. Perforating injuries correlate with a poorer outcome when compared with either penetrating or tangential brain injuries (Class III).
4. The effect of weapon caliber on outcome, independent of total kinetic energy, was not demonstrated in the published data.
5. Hypotension is associated with increased mortality (Class III).
6. Coagulopathy is associated with increased mortality, particularly at lower levels of the GCS (Class III).
7. Respiratory distress is associated with increased mortality (Class III).
8. In civilian patients, low GCS correlates with higher mortality and unfavorable outcome (Class I). In military injuries, fewer patients have a low GCS score; strong correlation also exists between low GCS score and unfavorable outcome in military series (Class III).
9. The presence of bilateral fixed and dilated pupils is highly predictive of mortality (Class III).
10. High ICP is predictive of higher mortality (Class II). Cisternal effacement PBI is associated with increased mortality (Class I). No relation between midline shift and outcome has been established (Class I).
11. Intraventricular hemorrhage is strongly correlated to increased mortality (Class I) - odds ratio 2.83-3.69. Subarachnoid hemorrhage is also correlated to increased mortality (Class I) - odds ratio 1.44-10.6.
12. Trajectory:

Bilateral injuries relate to increased mortality (Class II) - odds ratio 1.18-20.05, possible exception may be bifrontal injuries.
Multilobar (> 1 lobe) injuries are strongly associated with mortality (Class III) - odds ratio 3.27-84.4. Negative predictive value for mortality 77-98%.
Perhaps it should be better stated that the presence of unilobar damage is strongly correlated to survival.
Injuries with ventricular involvement have an increased mortality (Class III) - odds ratio 3.35-27.5

SCALES OF OUTCOME

FUNCTIONAL INDEPENDENCE MEASURE (FIM) (one of most widely used measures of function) - level of independence for mobility, self-care, and cognition (all these are often spared in mild TBI - FIM score is inadequate outcome measure for these patients):

Self-care

A. Eating

B. Dressing

C. Bathing

D. Dressing, upper body

E. Dressing, lower body

F. Toileting

Sphincter control

G. Bladder management

H. Bowel management

Transfers

I. Bed, chair, wheelchair

J. Toilet

K. Tub, shower

L. Walking, wheelchair

M. Stairs

Motor subtotal score:

Communication

N. Comprehension

O. Expression

Social interaction

P. Problem solving

Q. Memory

Cognitive subtotal score:

TOTAL FIM SCORE:

GLASGOW OUTCOME SCALE (GOS):

1 – DEAD

2 – VEGETATIVE

3 – SEVERE DISABILITY – conscious but disabled; dependent on others for daily support

4 – MODERATE DISABILITY – disabled but independent; can work in sheltered setting

5 – GOOD RECOVERY – normal life despite minor deficits

5 – GOOD RECOVERY – normal life despite minor deficits

lacer 1975: 1: 480– 484

GOS can be divided further into:

good outcomes (good disability, independent): 5 and 4

poor outcomes (severe disability, vegetative, dead): 1-3

Penetrating TBI

Military vs. civilian PBI – see (p. Op320)
Mostly Disability Categories:

**Total DRS Score**

<table>
<thead>
<tr>
<th>Level of Disability</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td>Mild</td>
<td>1</td>
</tr>
<tr>
<td>Partial</td>
<td>2-3</td>
</tr>
<tr>
<td>Moderate</td>
<td>4-6</td>
</tr>
<tr>
<td>Moderately Severe</td>
<td>7-11</td>
</tr>
<tr>
<td>Severe</td>
<td>12-16</td>
</tr>
<tr>
<td>Extremely Severe</td>
<td>17-23</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>22-24</td>
</tr>
<tr>
<td>Extreme Vegetative State</td>
<td>25-29</td>
</tr>
</tbody>
</table>

**Disability Categories:**

- **Arousalability**
- **Awareness and Responsivity (i.e. GCS)**
- **Communication Ability**
- **Motor Response**
- **Cognitive Ability for Self Care Activities**
- **Dependence on Others**
- **Psychosocial Adaptability**

**Motor vehicle-related TBI:**

1. Helmet by cyclists
2. Automobile seatbelts and child restraints
3. Airbags (children < 12 years should ride in back seat of car away from airbag)
4. Enforcement of drunk driving laws

**PREVENTION**

**SPECIAL ASPECTS**

**POSTERIOR FOSSA MASS LESIONS**

- parturitional hematomas in neonates (48%) involve posterior fossa
- patients can undergo rapid life-threatening clinical deterioration - limited size of the posterior fossa → brainstem compression.

**Indications for surgery:**

- a) ICH diameter > 5.3-0 cm
- b) mass effect on CT (dissolution / obstruction / obliteration of the 4th ventricle; compression of the basal cisterns; obstructive hydrocephalus)
- c) neurological dysfunction / deterioration referable to the lesion.

**Surgical treatment:**

- suboccipital craniectomy

**ALCOHOL**

- advantages - neuroprotection - alcohol use at time of injury decreases likelihood of poor outcome (alcohol impedes excitotoxicity).
- disadvantages
  1. alcohol raises risk of incurring (repeat) TBI (positive blood alcohol level is detected in 25-60% patients) - risk behavior, coordination, motor reaction time.
  2. obscured real level of consciousness.
  3. patients often aggressive, asocial.
  4. bleeding risk (thrombocytopenia, coagulopathy, brain atrophy)
  5. cardiopulmonary depression
  6. increased risk of seizures.

**LONG-BONE FRACTURES (CONCURRENT WITH TBI)**

- Femur fractures are common among TBI patients.

**TIMING OF INTERNAL fixation**

- Internal fixation with intramedullary nailing is the ideal method of treatment; however, there is no consensus regarding the optimal timing for internal fixation.

**EAST guidelines (2014):**


- Early vs. late fixation does NOT decrease mortality, infection, or VTE. Early (< 24 hr) fixation is suggested but is conditional and surgical decision must be individualized. The optimal timing remains controversial and guidelines do not replace clinical judgement.

- **no significant reduction in mortality** was associated with early stabilization (RR of 0.74 [95% CI, 0.50-1.00]), quality of evidence was rated as "low."
- **no significant reduction in infection** (RR, 0.4; 95% CI, 0.10-1.6) or VTE (RR, 0.63, 95% CI, 0.37–1.07) was associated with early stabilization; quality of evidence was rated "low."
- in the absence of a clear contraindication to surgery or anesthesis, the recommendation of this review, although conditional, should prompt early fixation. However, the surgical decision must be individualized to each patient’s needs. Delayed treatment has been associated with a reduction in adverse outcomes in patients with multiple injuries. In addition, delayed stabilization (24–48 hours) may be safer than stabilization within 12 hours for severely injured patients.

**CONCLUSION** - in trauma patients with open or closed femur fractures, we suggest early (< 24 hours) open reduction and internal fixation surgery. This recommendation is conditional because the strength of the evidence is low. Early stabilization of femur fractures shows a trend (statistically insignificant) toward lower risk of infection, mortality, and VTE. Therefore, the
American College of Surgeons (ACS) Trauma Quality Improvement Program (TQIP) best practices in the management of orthopedic trauma:

- special consideration should be given to patients with concomitant orthopedic and TBI. The management of fractures in patients with TBI represent a particular challenge in that early surgical fixation may complicate the acute management of TBI. The goals of acute TBI management are to maintain adequate cerebral perfusion, prevent hypotension, provide adequate oxygenation, avoid hypo- and hypercarbia, and maintain normothermia. Efforts should be made to adhere to each of these goals. Intraoperative monitoring of ICP should be considered to support cerebral perfusion pressure. In the context of a stable ICP and MAP, definitive fixation can be considered in the resuscitated patient, as these patients are less apt to experience transient episodes of hypotension in the operating room. The under-resuscitated patient or those patients whose ICP and CPP have not yet stabilized are best served with damage control procedures or traction.

- open fractures: historically, dogma has led orthopedists to treat open fractures with surgical irrigation and debridement within six hours of the injury or risk increased rates of infection. This practice has come to be known as the "six hour rule" in orthopedic surgery. However, it has been disproven in recent years by several high quality studies demonstrating that delaying surgical irrigation and debridement up to 24 hours does not increase infectious complications for open fractures. Based on the best available evidence, the panel does not endorse the "six hour rule."

**SPORTS**

**CHRONIC TRAUMATIC ENCEPHALOPATHY (C. DEMENTIA PUGILISTICA, PUNCH-DRUNK SYNDROME)**

- professional boxers are especially at risk (only sport with goal of deliberately injuring brain of opponent).
- grade - repeated head trauma during professional boxing.
- clinical features:
  - 1. Parkinsonism and other extrapyramidal features (tremor, ataxia, cerebellar signs).
  - manifestations begin de-4 to 16 years after starting boxing.
- pathology: hydrocephalus, thinning of corpus callosum, diffuse axonal injury, hypothalamic anomalies, degeneration of substantia nigra, neurofibrillar tangles (mainly in medial temporal areas), diffuse Aβ-positive plaques (i.e., in Alzheimer's disease), scarring of cerebellar folia.
- no therapy is actually effective.
- prevention: 1) Better protection for boxers (head protection, different gloves) 2) physician must stop match when there is evidence of brain injury (i.e. sportsman who sustained concussions, requires physician's statement to return to previous level of involvement, see below)

**MANAGEMENT OF CONCUSSION IN SPORTS**

**SHIELDEVALUATION:** Mental status testing:

2. Months of year in reverse order
3. Memory: names of teams in prior contest
4. President, governor, mayor, recent newsworthy events, three words and three objects at 0 and 5 minutes.
5. Details of contest (plays, moves, strategies, etc.)

**Exertional provocation tests:**

1. 40 yard sprint
2. 5 push-ups
3. 5 sit-ups
4. 5 knee bends

(any appearance of associated symptoms is abnormal, e.g., headache, dizziness, nausia, unsteadiness, photophobia, blurred or double vision, emotional lability, mental status changes)

**Neurologic tests:**

1. Pupils: symmetry a
2. Coordination: finger–nose–finger and tandem gait
3. Sensation: finger–nose (eyes closed) and Romberg

**GRADEING SCALE AND GUIDELINES FOR RETURN TO COMPETITION:**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Features</th>
<th>Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1</td>
<td>no loss of consciousness, confusion without amnesia</td>
<td>remove from contest → examine immediately and q/h for development of amnesia or postconcussive symptoms at rest and with exertion → may return to contest if no amnesia &amp; no symptoms appear for at least 20 minutes.</td>
</tr>
<tr>
<td>Grade 2</td>
<td>no loss of consciousness, confusion with amnesia</td>
<td>remove from contest and swallow returns → examine frequently for signs of evolving intracranial pathologic conditions → reexamine next day</td>
</tr>
<tr>
<td>Grade 3</td>
<td>loss of consciousness</td>
<td>Transport to field to ambulance (with cervical spine immobilization if indicated) to trauma hospital → emergent thorough neurological evaluation → signs of pathologic → R/O abnormality → hospitalization b) normal findings → instructions to family for overnight observation</td>
</tr>
</tbody>
</table>

**WHEN TO RETURN TO PLAY AFTER REMOVAL FROM COMPETITION:**

<table>
<thead>
<tr>
<th>Grade of Concussion</th>
<th>Time Until Return to Play*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple Grade 1 concussions</td>
<td>1 week</td>
</tr>
<tr>
<td>Grade 2 concussion</td>
<td>1 week</td>
</tr>
<tr>
<td>Multiple Grade 2 concussions</td>
<td>2 weeks</td>
</tr>
<tr>
<td>Grade 3, prolonged loss of consciousness (seconds)</td>
<td>1 week</td>
</tr>
<tr>
<td>Grade 3, prolonged loss of consciousness (minutes)</td>
<td>2 weeks</td>
</tr>
<tr>
<td>Multiple Grade 3 concussions</td>
<td>≥1 month (based on decision of physician)</td>
</tr>
</tbody>
</table>

*only after being asymptomatic with normal neurological assessment at rest and with exercise.
SECOND IMPACT SYNDROME

- Malignant cerebral edema
  - cause – one minor HI followed in short order by second minor HI in athletes who are still symptomatic from first injury.
  - mechanism - impaired cerebral autoregulation → vascular congestion → brain edema → herniation → sudden death.
  - prevention - postponed return to play for increasing lengths of time depending on concussion severity. See above.

BIBLIOGRAPHY: for ch. “Head Trauma” → follow this LINK >>

Viktor’s Notes™ for the Neurosurgery Resident
Please visit website at www.NeurosurgeryResident.net