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Brain injury

Phineas P. Gage (1823–1860) was an American railroad construction foreman remembered for his improbable survival of an accident in which a large iron rod was driven completely through his head, destroying much of his brain's left frontal lobe, and for that injury's reported effects on his personality and behavior over the remaining 12 years of his life

https://en.wikipedia.org/wiki/Phineas_Gage

TBI-related Emergency Department Visits, Hospitalizations, and Deaths

- In 2014, there were approximately 2.87 million TBI-EDHDs in the U.S., including over 837,000 of these health events among children.
- The number of TBI-EDHDs in 2014 represents a 53% increase from 2006, in which there were approximately 1.88 million TBI-EDHDs.

TBI-related Emergency Department (ED) Visits

- In 2014, there were approximately 2.5 million TBI-related ED visits in the U.S., including over 812,000 among children.
- Unintentional falls, being unintentionally struck by or against an object, and motor vehicle crashes were the most common mechanisms of injury contributing to a TBI diagnosis in the ED. These three principal mechanisms of injury accounted for 47.9%, 17.1%, and 13.2%, respectively, of all TBI-related ED visits.
- Rates of TBI-related ED visits per 100,000 population were highest among older adults aged ≥ 75 years (1,682.0), young children aged 0-4 years (1,618.6), and individuals aged 15-24 years (1,010.1).

TBI-related Hospitalizations

- In 2014, there were approximately 288,000 TBI-related hospitalizations in the U.S., including over 23,000 among children.
- Unintentional falls and motor vehicle crashes were the most common mechanisms of injury contributing to a TBI diagnosis in which the patient was hospitalized. These two principal mechanisms of injury accounted for 52.3% and 20.4%, respectively, of all TBI-related hospitalizations.
- Rates of TBI-related hospitalizations per 100,000 population were highest among older adults aged ≥75 years (470.6), those aged 65-74 years (145.5), and individuals aged 55-64 years (89.5).

TBI-related Deaths

- In 2014, there were 56,800 TBI-related deaths in the U.S., including 2,529 deaths among children.
- Intentional self-harm, unintentional falls, and motor vehicle crashes were the most common mechanisms of injury contributing to a TBI-related death. These three principal mechanisms of injury accounted for 32.5%, 28.1%, and 18.7%, respectively, of all TBI-related deaths.
- Rates of TBI-related deaths per 100,000 population were highest among older adults aged ≥75 years (78.5), those aged 65-74 years (24.7), and individuals aged 55-64 years (19.1).

Trends in TBI incidence by principal mechanism, 2006-2014

Age-adjusted rates of TBI-related ED visits increased 54% from 521.6 per 100,000 population in 2006 to 801.9 in 2014.

- 24% increase for TBI-related ED visits as a result of motor vehicle crashes (from 85.3 to 106);
- 80% increase for TBI-related ED visits as a result of falls (from 208.8 to 374.9);
- 58% increase for TBI-related ED visits as a result of being struck by or against an object (from 90.8 to 143.9);
- 60% increase for TBI-related ED visits as a result of intentional self-harm (from 0.5 to 0.8);
- 18% increase for TBIs as a result of assault (from 57.6 to 67.8).

While the number of TBI-related hospitalizations increased from 2006 to 2014, age-adjusted rates of TBI-related hospitalizations decreased by nearly 8% during that same period (from 92.2 to 84.9 per 100,000). This decrease coincides with a 34% decrease in the age-adjusted rate of TBI-related hospitalizations attributable to motor vehicle crashes (27.6 in 2006 to 18.1 in 2014).

While the number of TBI-related deaths increased from 2006 to 2014, age-adjusted rates decreased by 6% during that time period (from 17.9 in 2006 to 16.8 per 100,000 in 2014). This decrease coincides with a large decrease in the age-adjusted rate of TBI-related deaths attributable to motor vehicle crashes (5.4 in 2006 to 3.3 in 2014).

- Decreases in TBI-related hospitalizations and deaths as a result of motor vehicle crashes indicate significant progress in motor vehicle safety.
- Falls were the leading cause of injury for TBI-EDHDs in 2014, and over half of TBIs attributed to falls were in the youngest (0-4 years) and oldest (≥75 years) age groups, suggesting a need to intensify efforts related to fall prevention, particularly in these age groups.
- During the period of 2006-2014, rates of TBI-related deaths due to intentional self-harm increased 17%. This mirrors the increase in suicide rates overall in the U.S.,4 suggesting the need for expansion of comprehensive and coordinated suicide prevention efforts.

Facts about TBI

- According to the CDC, the economic cost of TBI in the United States in 2010, including direct and indirect medical costs, was estimated at $76.5 billion.
- The most common causes of TBI in the U.S. include violence, transportation accidents, construction, and sports
- Motor bikes are major causes, increasing in significance in developing countries as other causes reduce
- The estimates that between 1.6 and 3.8 million traumatic brain injuries each year are a result of sports and recreation activities in the US
- In children aged two to four, falls are the most common cause of TBI, while in older children traffic accidents compete with falls for this position
- TBI is the third most common injury to result from child abuse
- Abuse causes 19% of cases of pediatric brain trauma, and the death rate is higher among these cases.
- Although men are twice as likely to have a TBI. Domestic violence is another cause of TBI, as are work-related and industrial accidents.
- Firearms and blast injuries from explosions are other causes of TBI, which is the leading cause of death and disability in war zones

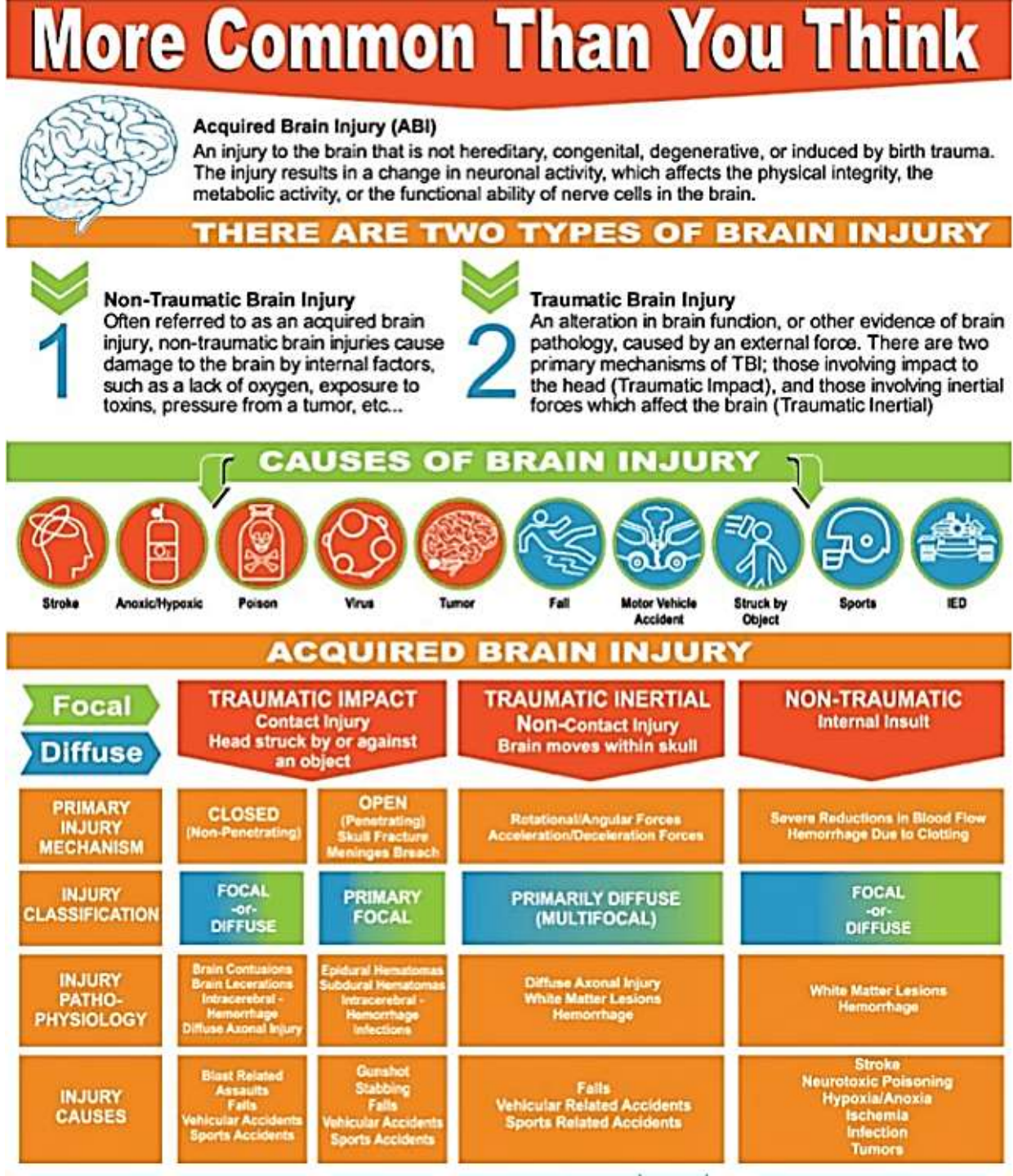
Traumatic Brain Injury

is an alteration in brain function, or other evidence of brain pathology, caused by an external force:

- falls
- assaults
- motor vehicle accidents
- sports injuries

Acquired Brain Injury

- is an injury to the brain, which is not hereditary, congenital, degenerative, or induced by birth trauma:
- stroke
- near drowning
- aneurysm
- tumor
- infectious disease that affects the brain (i.e., meningitis)
- lack of oxygen supply to the brain (i.e., heart attack)



CLASSIFICATION

Primary injury:

- Induced by mechanical force and occurs at the moment of injury by the 2 main mechanisms:
  - contact (eg, an object striking the head or the brain striking the inside of the skull)
  - acceleration-deceleration

Secondary injury:

- Not mechanically induced;
- may be delayed from the moment of impact
- may superimpose injury on a brain already affected by a mechanical injury

Silver JM, McAllister TW, Yodofsky SC, eds. *Textbook of Traumatic Brain Injury*. Arlington, Va: American Psychiatric Publishing; 2005. Arlington, VA: 27-39.

CLASSIFICATION

Focal injury:

generally caused by contact

- scalp injury,
- skull fracture,
- surface contusions

Diffuse injury:

usually caused by acceleration-deceleration forces

- diffuse axonal injury (DAI),
- hypoxic-ischemic damage,
- meningitis,
- vascular injury

Silver JM, McAllister TW, Yodofsky SC, eds. *Textbook of Traumatic Brain Injury*. Arlington, Va: American Psychiatric Publishing; 2005. Arlington, VA: 27-39.

Measures of severity

- Glasgow Coma Scale (GCS): A 3- to 15-point scale used to assess a patient's level of consciousness and neurologic functioning; scoring is based on best motor response, best verbal response, and eye opening (eg, eyes open to pain, open to command)
- Duration of loss of consciousness: Classified as mild (mental status change or loss of consciousness [LOC] < 30 min), moderate (mental status change or LOC 30 min to 6 hr), or severe (mental status change or LOC >6 hr)
- Posttraumatic amnesia (PTA): The time elapsed from injury to the moment when patients can demonstrate continuous memory of what is happening around them

Glasgow Coma Scale

Eyes
Open spontaneously **4**
Open to verbal command **3**
Open to painful stimuli **2**
No response **1**
Verbal Response
Oriented and converses **5**
Disoriented and converses **4**
Inappropriate words **3**
Incomprehensible sounds **2**
No response **1**
Motor Response
Obeys verbal commands **6**
Responds to painful stimuli by:
purposeful localization **5**
withdrawal **4**
flexor posturing **3**
extensor, posturing **2**
no response **1**
GCS Score 3 to 15

Outcome measures

- Functional Independence Measure (FIM)
- Glasgow Outcome Scale (GOS)
- Disability Rating Scale (DRS)

Disability Rating Scale (DRS)

Arousability, Awareness, & Responsivity

Eye Opening
- ❑ 0 Spontaneous
- ❑ 1 To Speech
- ❑ 2 To Pain
- ❑ 3 None

Communication Ability
- ❑ 0 Oriented
- ❑ 1 Confused
- ❑ 2 Inappropriate
- ❑ 3 Incomprehensible
- ❑ 4 None

Motor Response
- ❑ 0 Obeying
- ❑ 1 Localizing
- ❑ 2 Withdrawing
- ❑ 3 Flexing
- ❑ 4 Extending
- ❑ 5 None

Cognitive Ability for Self Care Activities

Knows how and when to feed, toilet or groom self

Feeding
- ❑ 0.0 Complete
- ❑ 0.5
- ❑ 1.0 Partial
- ❑ 1.5
- ❑ 2.0 Minimal
- ❑ 2.5
- ❑ 3.0 None

Toileting
- ❑ 0.0 Complete
- ❑ 0.5
- ❑ 1.0 Partial
- ❑ 1.5
- ❑ 2.0 Minimal
- ❑ 2.5
- ❑ 3.0 None

Grooming
- ❑ 0.0 Complete
- ❑ 0.5
- ❑ 1.0 Partial
- ❑ 1.5
- ❑ 2.0 Minimal
- ❑ 2.5
- ❑ 3.0 None

Dependence on Others

Level of Functioning

Physical & cognitive disability

- ❑ 0.0 Completely Independent
- ❑ 0.5
- ❑ 1.0 Independent in special environment
- ❑ 1.5
- ❑ 2.0 Mildly Dependent-Limited assistance
  Non-resident helper
- ❑ 2.5
- ❑ 3.0 Moderately Dependent-moderate assist
  Person in home
- ❑ 3.5
- ❑ 4.0 Markedly Dependent
  Assistance with all major activities, all times
- ❑ 4.5
- ❑ 5.0 Totally Dependent
  24 hour nursing care

Psychosocial Adaptability

Employability

As full time worker, homemaker, student

- ❑ 0.0 Not Restricted
- ❑ 0.5
- ❑ 1.0 Selected jobs, competitive
- ❑ 1.5
- ❑ 2.0 Sheltered workshop, Noncompet.
- ❑ 2.5
- ❑ 3.0 Not Employable

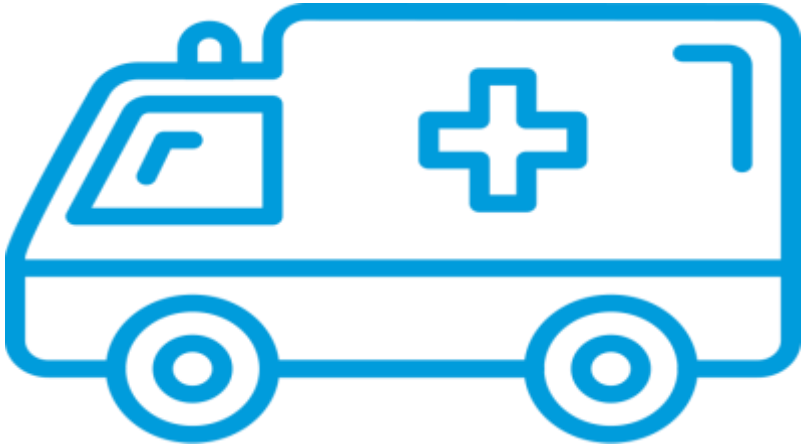
Total Score (sum all scores) _____

Complications

- Posttraumatic seizures: Frequently occur after moderate or severe TBI
- Hydrocephalus
- Deep vein thrombosis: Incidence as high as 54%
- Heterotopic ossification: Incidence of 11-76%, with a 10-20% incidence of clinically significant heterotopic ossification
- Spasticity
- Gastrointestinal and genitourinary complications: Among the most common sequelae in patients with TBI
- Gait abnormalities
- Agitation: Common after TBI
- Chronic traumatic encephalopathy (CTE)
- Long-term physical, cognitive, and behavioral impairments are the factors that most commonly limit a patient's reintegration into the community and his/her return to employment:
- Insomnia
- Cognitive decline
- Posttraumatic headache: Tension-type headaches are the most common form, but exacerbations of migraine-like headaches are also frequent
- Posttraumatic depression: Depression after TBI is further associated with cognitive decline, anxiety disorders, substance abuse, dysregulation of emotional expression, and aggressive outbursts

Cifu DX, Kaelin DL, Wall BE. Deep venous thrombosis: incidence on admission to a brain injury rehabilitation program. Arch Phys Med Rehabil. 1996 Nov. 77(11):1182-5. [Medline]
Melamed E, Robinson D, Halperin N, et al. Brain injury-related heterotopic bone formation: treatment strategy and results. Am J Phys Med Rehabil. 2002 Sep. 81(9):670-4. [Medline].
Chamelian L, Feinstein A. The effect of major depression on subjective and objective cognitive deficits in mild to moderate traumatic brain injury. J Neuropsychiatry Clin Neurosci. 2006. 18(1):33-8. [Medline].
Jorge RE, Robinson RG, Moser D, et al. Major depression following traumatic brain injury. Arch Gen Psychiatry. 2004 Jan. 61(1):42-50. [Medline]. [Full Text].
Kumar RG, Gao S, Juengst SB, Wagner AK, Fabio A. The effects of post-traumatic depression on cognition, pain, fatigue, and headache after moderate-to-severe traumatic brain injury: a thematic review. Brain Inj. 2018 Jan 22. 1-12. [Medline].
Grauwmeijer E, Heijenbrok-Kal M, Peppel L, et al. Cognition, Health-related Quality of life, and Depression Ten Years after Moderate to Severe Traumatic Brain Injury: a prospective cohort study. J Neurotrauma. 2018 Jan 17. [Medline].

Transport to hospital



- Transport patients who have sustained a head injury directly to a hospital that has the resources to further resuscitate them and to investigate and initially manage multiple injuries.
- All acute hospitals receiving patients with head injury directly from an incident should have resources, which should be appropriate for a patient's age
- A clinician with training in safeguarding should be involved in the initial assessment of any patient with a head injury presenting to the emergency department.

Criteria for performing a CT head scan

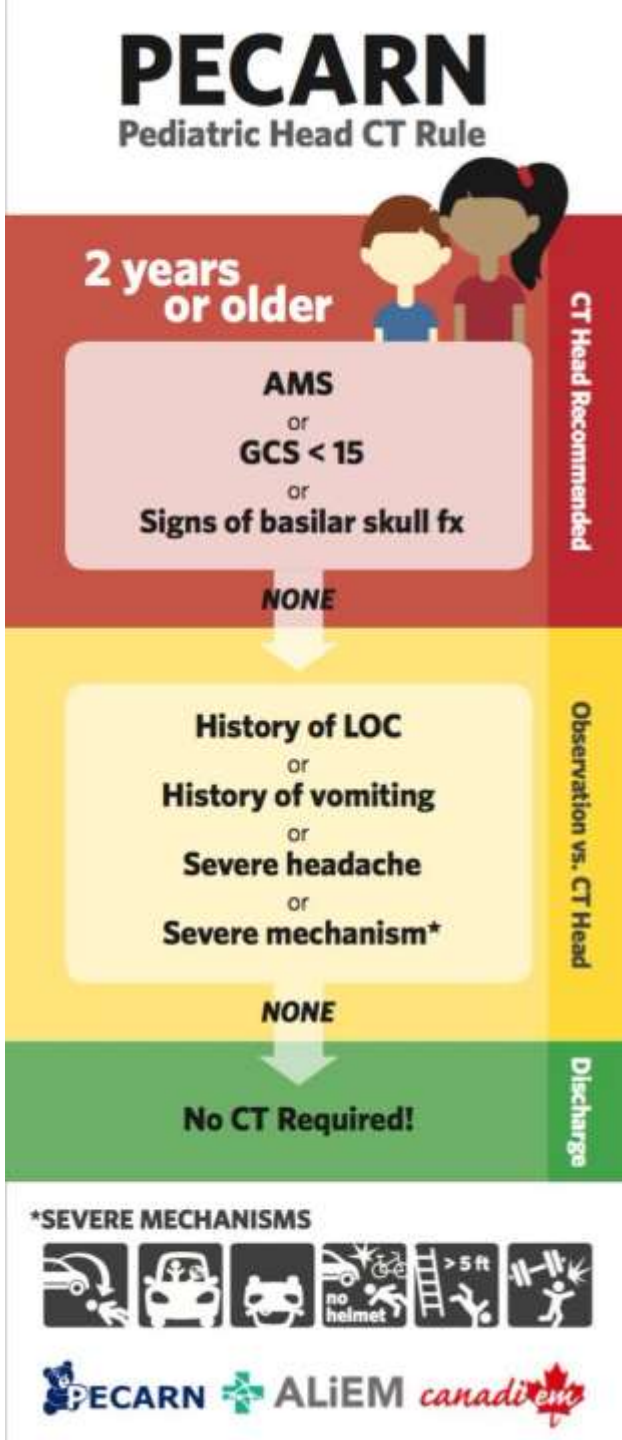
For adults who have sustained a head injury and have any of the following risk factors, perform a CT head scan within 1 hour of the risk factor being identified:

- GCS less than 13 on initial assessment in the emergency department.
- GCS less than 15 at 2 hours after the injury on assessment in the emergency department.
- Suspected open or depressed skull fracture.
- Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
- Post-traumatic seizure.
- Focal neurological deficit.
- More than 1 episode of vomiting.

Criteria for performing a CT head scan

For children who have sustained a head injury and have any of the following risk factors, perform a CT head scan within 1 hour of the risk factor being identified:

- Suspicion of non-accidental injury.
- Post-traumatic seizure but no history of epilepsy.
- On initial emergency department assessment, GCS less than 14, or for children under 1 year GCS (pediatric) less than 15.
- At 2 hours after the injury, GCS less than 15.
- Suspected open or depressed skull fracture or tense fontanelle.
- Any sign of basal skull fracture (hemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
- Focal neurological deficit.
- For children under 1 year, presence of bruise, swelling or laceration of more than 5 cm on the head.



- For patients (adults and children) who have sustained a head injury with no other indications for a CT head scan and who are having warfarin treatment, perform a CT head scan within 8 hours of the injury.
- **A provisional written radiology report should be made available within 1 hour of the scan being performed.**
- Investigating injuries to the cervical spine

For adults who have sustained a head injury and have any of the following risk factors, perform a CT cervical spine scan within 1 hour of the risk factor being identified:

- GCS less than 13 on initial assessment.
- The patient has been intubated.
- Plain X-rays are technically inadequate (for example, the desired view is unavailable).
- Plain X-rays are suspicious or definitely abnormal.
- A definitive diagnosis of cervical spine injury is needed urgently (for example, before surgery).
- The patient is having other body areas scanned for head injury or multi-region trauma.
- The patient is alert and stable, there is clinical suspicion of cervical spine injury and any of the following apply:
- age 65 years or older
- dangerous mechanism of injury (fall from a height of greater than 1 meter or 5 stairs; axial load to the head, for example, diving; high-speed motor vehicle collision; rollover motor accident; ejection from a motor vehicle; accident involving motorized recreational vehicles; bicycle collision)
- focal peripheral neurological deficit
- paraesthesia in the upper or lower limbs.

Focal neurological deficit

Problems restricted to a particular part of the body or a particular activity

- difficulties with understanding, speaking, reading or writing;
- decreased sensation;
- loss of balance;
- general weakness;
- visual changes;
- abnormal reflexes;
- problems walking.

High-energy head injury

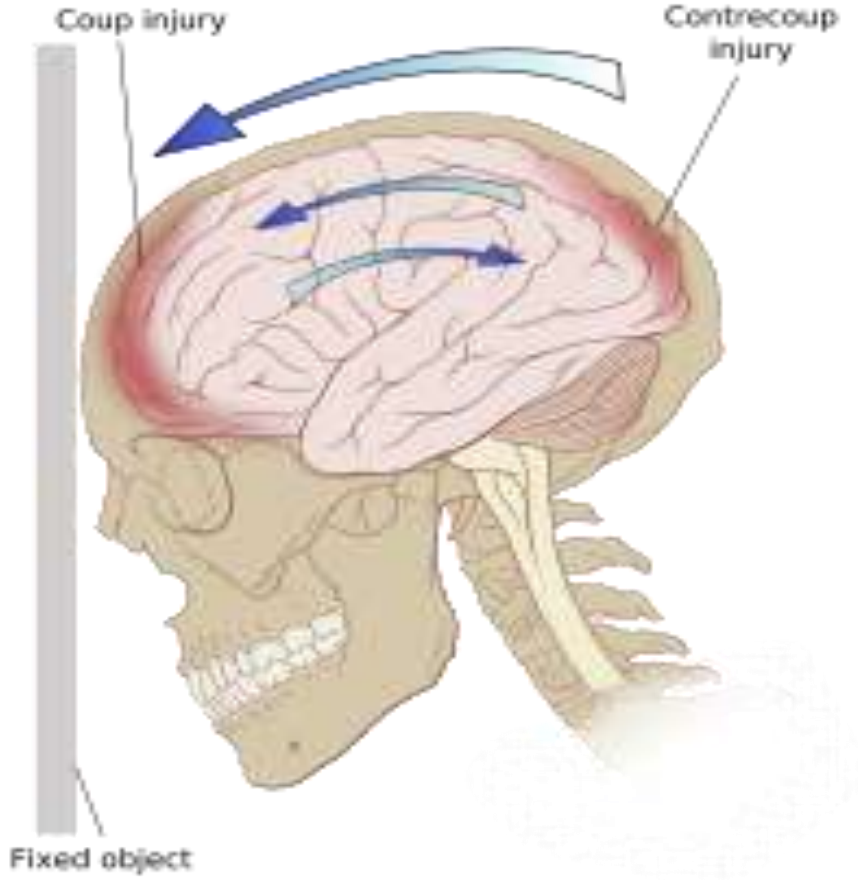
- pedestrian struck by motor vehicle,
- occupant ejected from motor vehicle,
- fall from a height of greater than 1 metre or more than 5 stairs,
- diving accident,
- high-speed motor vehicle collision,
- rollover motor accident,
- accident involving motorised recreational vehicles,
- bicycle collision,
- other potentially high-energy mechanism.

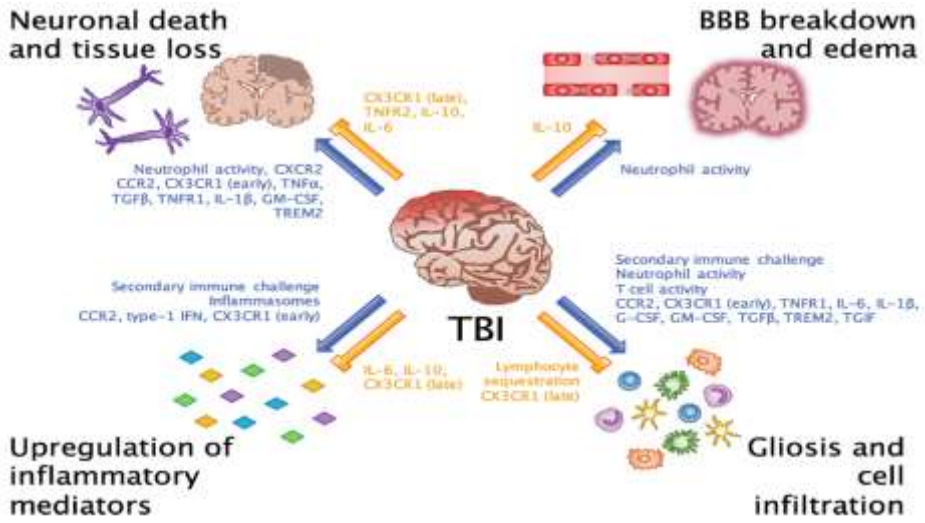
Base of open or depressed skull fracture or penetrating head injury

- clear fluid running from the ears or nose,
- black eye with no associated damage around the eyes,
- bleeding from one or both ears,
- bruising behind one or both ears,
- penetrating injury signs,
- visible trauma to the scalp or skull of concern to the professional.

Pathophysiology

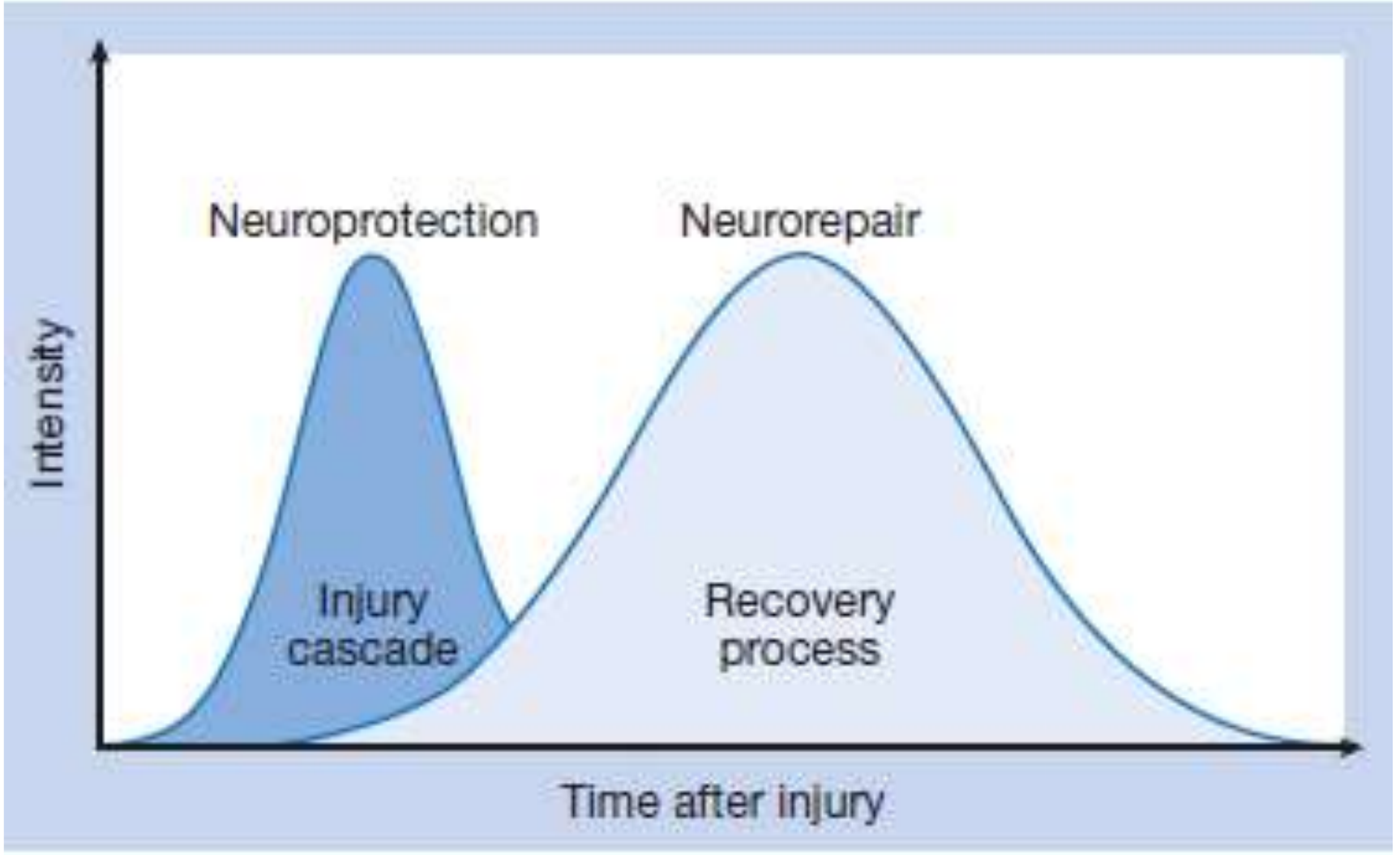
- primary injury that disrupts brain tissue and function at moment of impact;
- secondary injury through multiple biochemical cascades that propagate cellular dysfunction and lead to cell death;
- chronic degeneration, repair, and regeneration process that occurs over the long term after the injury has occurred.





continuation of injury and repair associated with pathophysiology after TBI.

The primary injury disrupts brain tissue and initiates secondary injury cascades that lead to cell dysfunction and death. Neuroprotective and neurorepair mechanisms after injury contribute to chronic degeneration, repair, and regeneration processes.



Primary Injury

Occurs at the moment of the impact and as a direct result of trauma

- Contusions and lacerations of the brain surfaces
- Diffuse axonal injury (DAI)
- Diffuse vascular injury/multiple petechial hemorrhages
- Cranial nerve injury

Responsible for the initial loss of consciousness seen in acute TBI

• Results from acceleration-deceleration and rotational forces associated with high-velocity impact (MVAs)

• The axonal injury seen in severe TBI is thought to be secondary to damage to the axoplasmic transport in axons (with ↑ Ca^{++} influx) leading to axonal swelling and detachment

Contusion—bruising of cerebral (cortical) tissue

• Occurs on the undersurface of the frontal lobe (inferior frontal or orbitofrontal area) and anterior temporal lobe, regardless of the site of impact (Figure 2–1)

• May produce focal, cognitive, and sensory-motor deficits

• Is not directly responsible for loss of consciousness following trauma

• May occur from relatively low velocity impact, such as blows and falls

Diffuse axonal injury (DAI):

• DAI is seen exclusively in TBI

• Damage seen most often in the corpus callosum and other midline structures involving the parasagittal white matter, the interventricular septum, the walls of the third ventricle and the brain stem (midbrain and pons)

Secondary

Damage that occurs after the initial trauma and as a result of the injuring event. Most secondary injury occurs during the first 12 to 24 hours after trauma, but may occur up to 5 to 10 days postinjury in very severe brain injury. Because of the delayed presentation, secondary injury may be preventable.

- A. Intracranial hemorrhage (epidural, subdural, subarachnoid and intracerebral hematoma)
- B. Brain swelling/brain edema (see below)
- C. Elevated Intracranial Pressure (ICP)
- – ↑ ICP⇒ ↓ perfusion ⇒ ischemic brain damage
- D. Brain damage secondary to hypoxia
- E. Intracranial infection
- F. Hydrocephalus
- G. ↑ release of excitatory neurotransmitters secondary to diffuse axonal injury (DAI) = excitotoxicity
- – This will increase the activity of certain brain areas and overall metabolic demand in the already injured brain
- H. Production of free-radical molecules

Other secondary causes of brain injury include:

- Hypotension
- Hyperemia
- Electrolyte imbalances
- Hyponatremia
- Anemia
- Infection
- Hyperthermia
- Carotid dissection
- Hyperglycemia
- Epilepsy/seizures
- Hypercarbia
- Vasospasm/ischemia
- Hypoglycemia

Secondary Head Injury

Brain Swelling

- Occurs after acute head injury within 24 hours.
- Identified in CT as collapse of ventricular system and loss of cerebral spinal fluid (CSF) cisterns around the midbrain
- Is due to an increase in cerebral blood volume (intravascular blood)

Brain Edema

- Occurs later after head injury (in comparison to brain swelling)
- Is due to an increase in brain volume secondary to ↑ brain water content ⇒extravascular fluid

Vasogenic edema:

– Due to outpouring of protein rich fluid through damaged vessels

– *Extracellular* edema

– Related to cerebral contusion

Cytogenic edema:

– Found in relation to hypoxic and ischemic brain damage

– Due to failing of the cells' energy supply system ⇒ ↑ cell-wall pumping system ⇒

intracellular edema in the dying cells

RECOVERY MECHANISMS

Plasticity

Brain plasticity is when the damaged brain has the capabilities to repair itself by means of morphologic and physiologic responses

Plasticity is influenced by the environment, complexity of stimulation, repetition of tasks, and motivation

It occurs via 2 mechanisms:

1) Neuronal regeneration/neuronal (collateral) sprouting

2) Unmasking neural reorganization

Neuronal Regeneration

- Intact axons establish synaptic connections through dendritic and axonal sprouting in areas where damage has occurred
- May enhance recovery of function, may contribute to unwanted symptoms, or may be neutral (with no increase or decrease of function)
- Thought to occur weeks to months post-injury

Functional Reorganization/Unmasking

- Healthy neural structures not formerly used for a given purpose are developed (or reassigned)
- to do functions formerly subserved by the lesioned area.

Consciousness

- Consciousness is a function of ascending reticular activating system (RAS) and the cerebral cortex
- RAS is composed of cell bodies in the central reticular core of the upper brain stem (mainly midbrain) and their projections to widespread areas of the cerebral cortex via both the thalamic and the extrathalamic pathways.
- Lesions that interrupt the metabolic or structural integrity of the RAS or enough of the cortical neurons receiving RAS projections can cause coma.

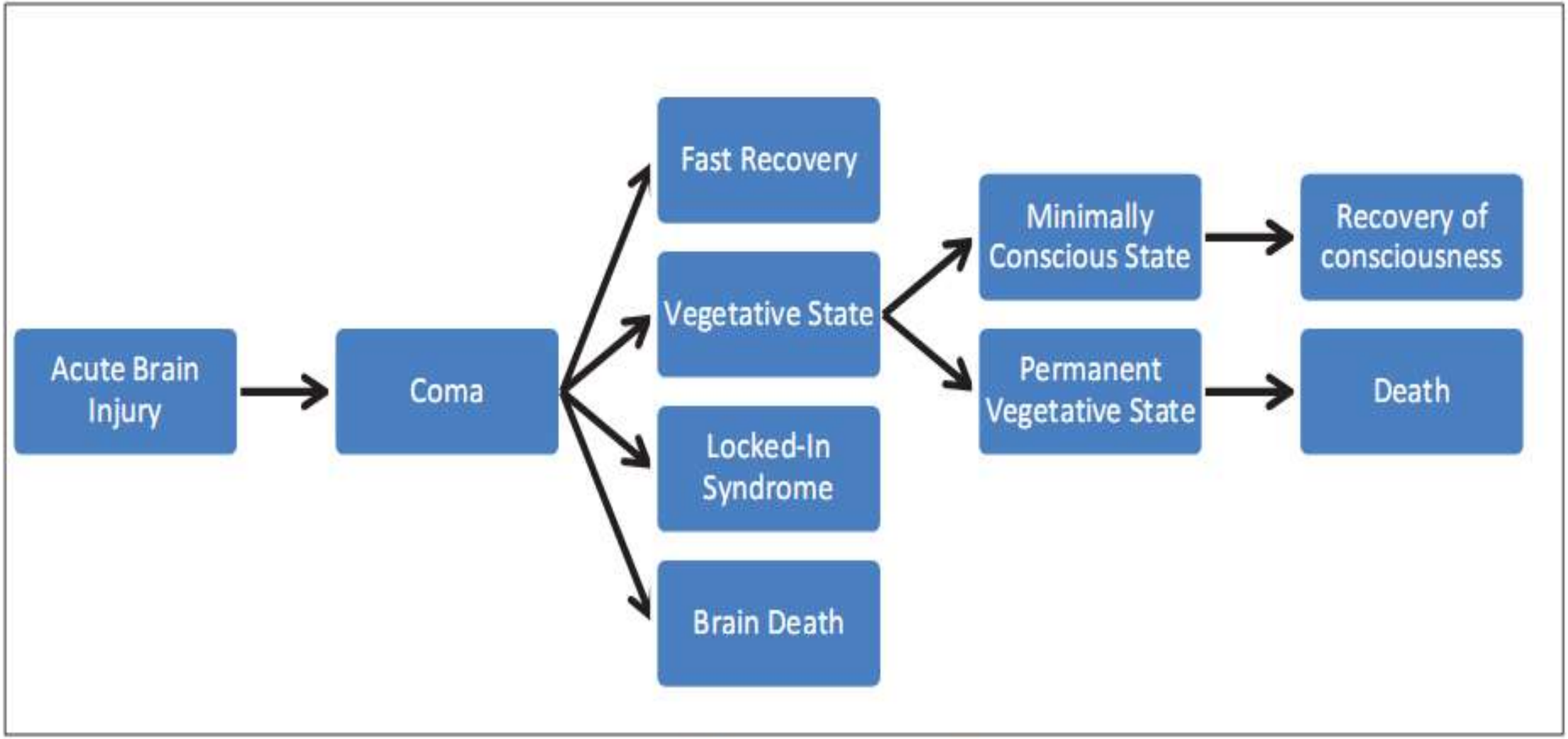
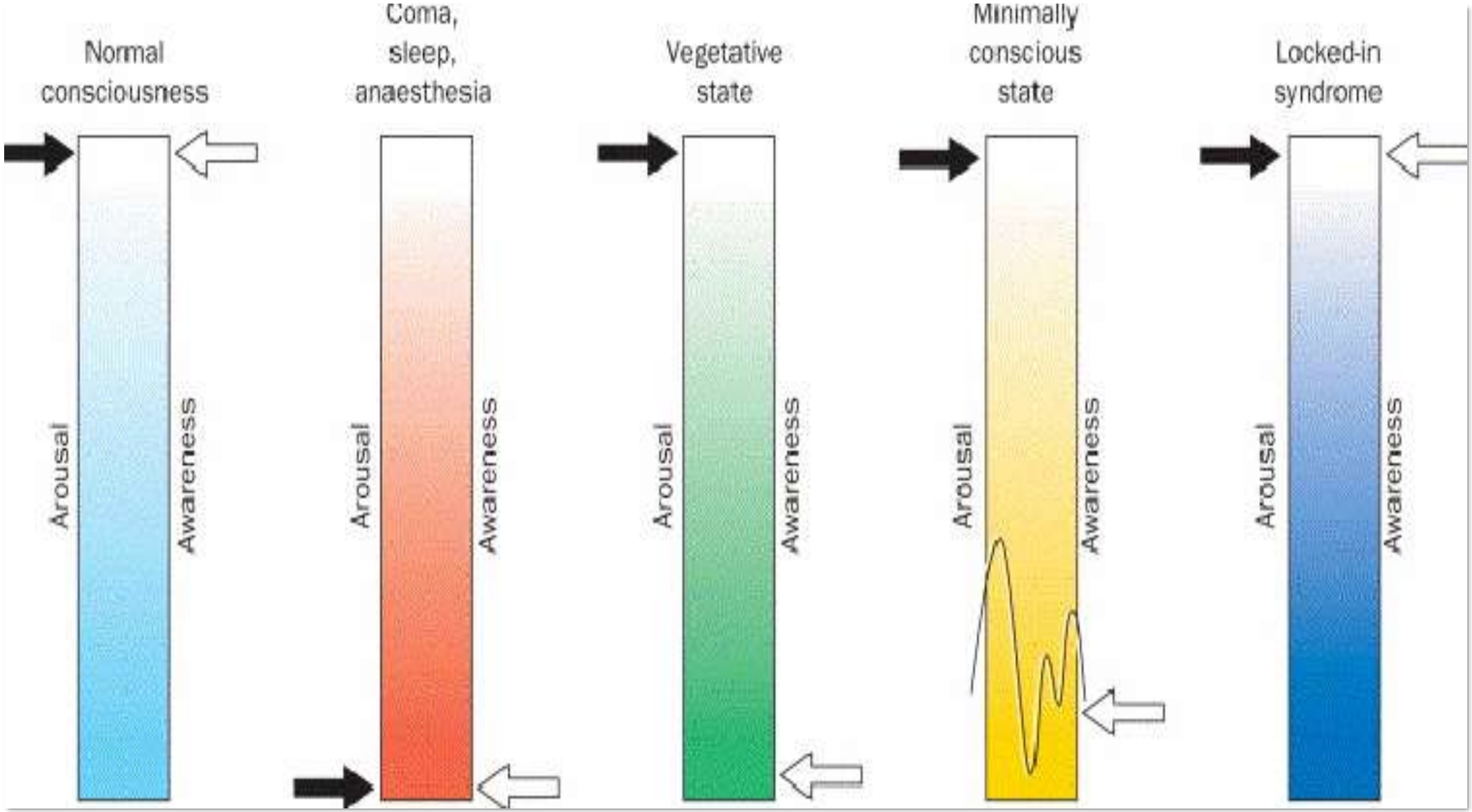


Fig. 1. Pathways of consciousness. (Modified from Laureys S. Eyes open, brain shut. Sci Am *2007; 296(5): 68 - 69.*[9]*)*


Normal consciousness
Coma, sleep, anaesthesia
Vegetative state
Minimally conscious state
Locked-in syndrome
Arousal
Awareness

Treatment

There is no evidence to support that any kind of therapy-based program (e.g., coma stimulation/sensory-stimulation program) will induce or accelerate the cessation of coma or VS

Nevertheless, an organized treatment approach to low-functioning patients permits a quantifiable assessment of responses to stimulation and early recognition of changes or improvements in response to therapeutic interventions or through spontaneous recovery

Management/Therapy Program for Patients with Disorders of Consciousness

Neuromedical stabilization
Preventive therapeutic interventions may be implemented:
– Manage bowel and bladder (B/B) function
– Maintain nutrition
– Maintain skin integrity
– Control spasticity
– Prevent contractures

Management/Therapy Program for Patients with Disorders of Consciousness

Pharmacologic treatment/intervention

- Elimination of unnecessary medicines (e.g., histamine-2 blockers, metoclopramide, pain meds, etc.) and selection of agents with fewest adverse effects on cognitive and neurologic recovery. Addition of agents to enhance specific cognitive and physical functions

– In patients emerging out of coma or VS, the recovery process may be (theoretically) hastened through the use of pharmacotherapy

– Agents frequently used include:

- Methylphenidate
- Dextroamphetamine
- Dopamine agonists (levocarbidopa and carbidopa)
- Amantadine
- Bromocriptine
- Antidepressants—tricyclic antidepressants (TCA's) & selective serotonin reuptake inhibitors (SSRIs)

– The efficacy of pharmacologic therapy to enhance cognitive function has not been proven

Management/Therapy Program for Patients with Disorders of Consciousness

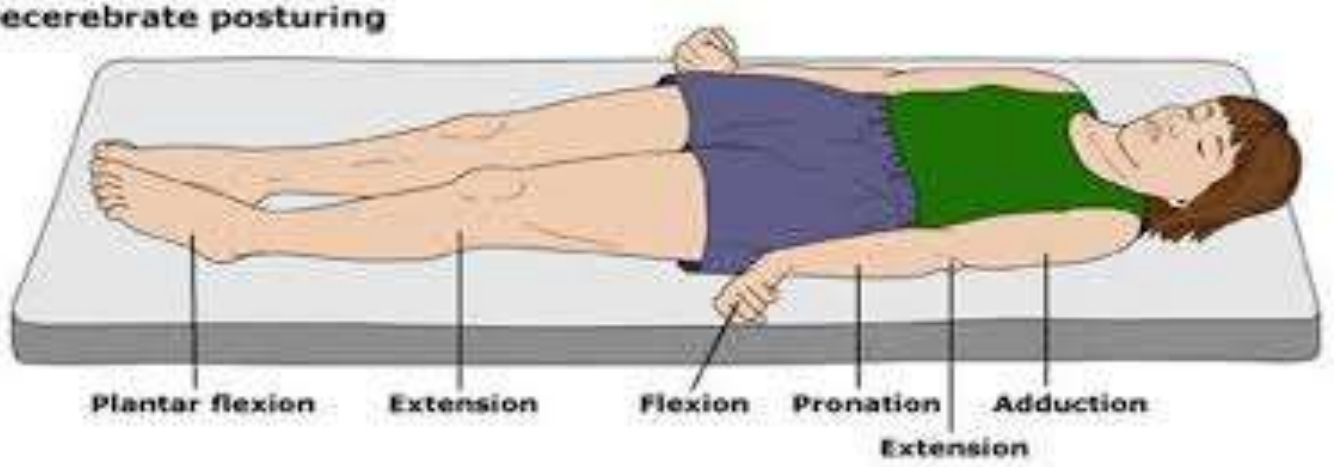
Sensory stimulation—widely used despite little evidence of efficacy as previously mentioned.

– Sensory stimulation should include all five senses, addressed one at a time, in specific therapy sessions and/or in the environmental state and developed in the room

– Avoid overstimulation (educate family)

– Patient may have adverse responses due to overstimulation, as ↑ confusion or agitation, ↑ reflex responses or avoidance reactions, which may interfere with performance

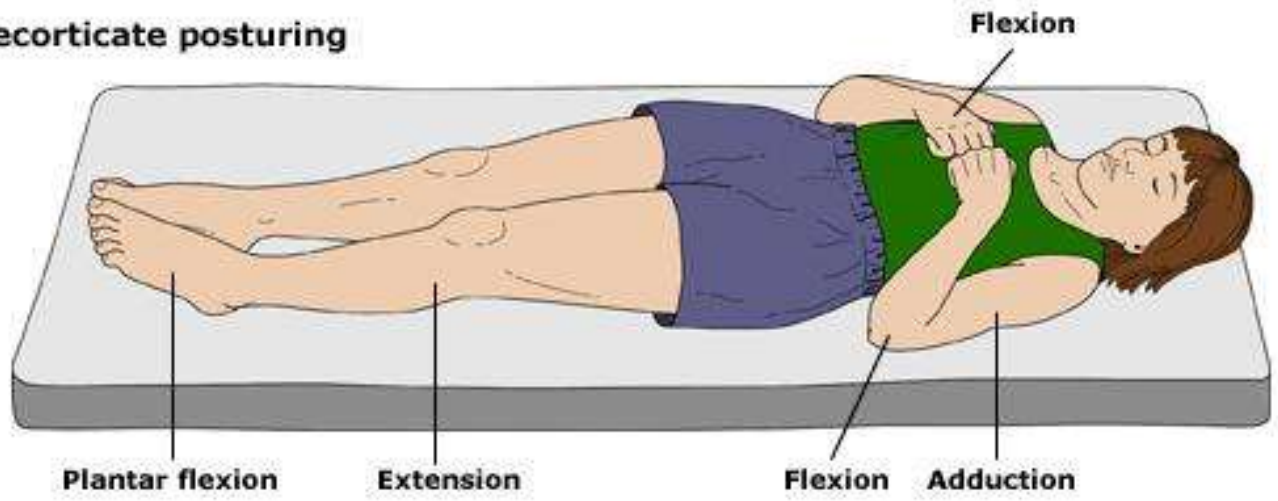
POSTURING SECONDARY TO HEAD INJURY



DECEREBRATE POSTURING

- This postural pattern was first described by Sherrington, who produced it in cats and monkeys by transecting the brain stem
- There is extension of the upper and lower extremities (hallmark: elbows extended)
- Seen with midbrain lesions/compression; also with cerebellar and posteria fossa lesions
- In its fully developed form it consists of opisthotonus, clenched jaws, and stiff, extended limbs with internal rotation of arms and ankle plantar flexion

POSTURING SECONDARY TO HEAD INJURY



DECORTICATE POSTURING

- Posturing due to lesions at a higher level (than in decerebrate posture)—seen in cerebral hemisphere/white matter, internal capsule and thalamic lesions
- Flexion of the upper limbs (elbows bent) and extension of the lower limbs
- Arms are in flexion and adduction and leg(s) extended

PREDICTORS OF OUTCOME AFTER TBI

The best Glasgow Coma Scale (GCS) score within 24 hours of injury (The *initial* GCS and the *worst* GCS (within the first 24 hours) have also been proposed as acute indicators of severity in TBI)

Length of coma

Duration of posttraumatic amnesia (PTA)

Glasgow Coma Scale

3 to 8 = severe injury

9 to 12 = moderate injury

13 to 15 = mild injury

Score	Best Motor Response 6	Best Verbal Response 5	Eye Opening 4
1	None	None	None
2	Decerebrate posturing (extension) to pain	Mutters unintelligible sounds	Opens eyes to pain
3	Decorticate posturing (flexion) to pain	Says inappropriate words	Opens eyes to loud voice (verbal commands)
4	Withdraws limb from painful stimulus	Able to converse—confused	Opens eyes spontaneously
5	Localizes pain/pushes away noxious stimulus (examiner)	Able to converse—alert and oriented	
6	Obeys verbal commands		

- GCS scores of 3–4 resulted in death or VS in 87% of patients
- Scores 5–7 = death or VS in 53% and moderate or good recovery in 34%
- Scores 8–10 = moderate or good recovery in 68%
- Score of 11 = moderate or good recovery in 87%

Posttraumatic Amnesia

Duration of PTA	Severity of Injury Category
Less than 5 minutes	Very mild
5–60 minutes	Mild
1–24 hours	Moderate
1–7 days	Severe
1–4 weeks	Very severe
Greater than 4 weeks	Extremely severe

Length of PTA	Likely Outcome
1 day or less	Expect quick and full recovery with appropriate management (a few may show persisting disability)
More than 1 day, less than 1 week	Recovery period more prolonged—now a matter of weeks or months. Full recovery possible, for most of these cases, with good management.
1–2 weeks	Recovery a matter of many months. Many patients are left with residual problems even after the recovery process has ended, but one can be reasonably optimistic about functional recovery with good management.
2–4 weeks	Process of recovery is very prolonged—1 year or longer is not unusual. Permanent deficits are likely. There must be increasing pessimism about functional recovery when PTA reaches these lengths.
More than 4 weeks	Permanent deficits, indeed significant disability, now certain. It is not just a matter of recovery but of long-term retraining and management.

Glasgow Outcome Scale (GOS)

	Category	Description
1	Death	Self-evident criteria
2	Persistent vegetative state	Prolonged unconsciousness with no verbalization, no following of commands. Absent awareness of self and environment; patient may open eyes; absence of cortical function as judged behaviorally; characterized by the presence of sleep-wake cycles
3	Severe disability	Patient unable to be independent for any 24-hour period by reason of residual mental and/or physical disability
4	Moderate disability	Patient with residual deficits that do not prevent independent daily life; patient can travel by public transport and work in a sheltered environment
5	Good recovery	Return to normal life; there may be minor or no residual deficits

Predicative Indicator	Poorer	Better
Glasgow Coma Scale score	< 7	> 7
CT scan	Large blood clot; massive bihemispheric swelling	Normal
Age	Old age	Youth
Pupillary light reflex	Pupils remain dilated	Pupil contracts
Doll's eye sign	Impaired	Intact
Caloric testing with ice water	Eyes do not deviate	Eyes deviate to irrigated side
Motor response to noxious stimuli	Decerebrate rigidity	Localizes defensive gestures
Somatosensory evoked potentials	Deficient	Normal
Posttraumatic amnesia length	> 2 wks	< 2 wks

Disability Rating Scale (DRS)

1. Eye Opening	2. Communication	3. Motor Response
0 Spontaneous	0 Oriented	0 Obeying
1 To Speech	1 Confused	1 Localizing
2 To Pain	2 Inappropriate	2 Withdrawing
3 None	3 Incomprehensible	3 Flexing
	4 None	4 Extending
		5 None

4. Feeding	5. Toileting	6. Grooming
0.0 Complete	0.0 Complete	0.0 Complete
0.5	0.5	0.5
1.0 Partial	1.0 Partial	1.0 Partial
1.5	1.5	1.5
2.0 Minimal	2.0 Minimal	2.0 Minimal
2.5	2.5	2.5
3.0 None	3.0 None	3.0 None

7. Level of functioning (physical and cognitive disability)	8. "Employability" (as full-time worker, homemaker, or student)
0.0 Completely independent	0.0 Not restricted
0.5	0.5
1.0 Dependent in special environment	1.0 Selected jobs, competitive
1.5	1.5
2.0 Mildly dependent—limited assistance (nonresident helper)	2.0 Sheltered workshop, noncompetitive
2.5	2.5
3.0 Moderately dependent—moderate assistance (person in home)	3.0 Not employable
3.5	
4.0 Markedly dependent—assist all major activities, all times	
4.5	
5.0 Totally dependent—24-hr nursing care	

Rancho Los Amigos Levels of Cognitive Function Scale (LCFS)

Level	Description
I	No response
II	Generalized response to stimulation
III	Localized response to stimuli
IV	Confused and agitated behavior
V	Confused with inappropriate behavior (nonagitated)
VI	Confused but appropriate behavior
VII	Automatic and appropriate behavior
VIII	Purposeful and appropriate behavior

Functional Independence Measure (FIM)

7 Complete Independence (Timely, Safely) 6 Modified Independence (Device)	NO HELPER
Modified Dependence 5 Supervision 4 Minimal Assist (Subject = 75% +) 3 Moderate Assist (Subject = 50% +) Complete Dependence 2 Maximal Assist (Subject = 25% +) 1 Total Assist (Subject = 0% +)	HELPER

	ADMIT	DISCHG	FOL-UP
Self-Care			
A. Eating			
B. Grooming			
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			
Locomotion			
L. Walk/Wheelchair			
M. Stairs			
Motor Subtotal Score			
Communication			
N. Comprehension			
O. Expression			
Social Cognition			
P. Social Interaction			
Q. Problem Solving			
R. Memory			
Cognitive Subtotal Score			
Total FIM			

NOTE: Leave no blanks; enter 1 if patient not testable due to risk

MEDICAL COMPLICATIONS AFTER TBI

Posttraumatic Hydrocephalus (PTH)

- Ventriculomegaly (ventricular dilation) is common after TBI , reported in 40%–72% of patients after severe TBI. However, true hydrocephalus is relatively rare; incidence is 3.9 to 8%
- Ventriculomegaly is usually due to cerebral atrophy and focal infarction of brain tissue (*ex vacuo* changes)
- Hydrocephalus in TBI is most often of the communicating or normal-pressure type
- Unfortunately, the classic triad of incontinence, ataxia/gait disturbance and dementia is of little help in severe TBI cases
- Radiographic evaluation (CT Scan) and further work-up (to rule out hydrocephalus) should be considered if there is failure to improve or deterioration of cognitive or behavioral function
- CT-Scan—periventricular lucency, lack of sulci, and uniformity in ventricular dilation favors PTH
- Initial manifestations of hydrocephalus can be intermittent HA, vomiting, confusion, and drowsiness
- Tx: Lumbar puncture, shunt placement

Elevated Intracranial Pressure (ICP)

- In a normal adult, reclining with the head and the trunk elevated to 45°, the ICP is between 2 to 5 mmHg
- ICP levels up to 15 mmHg are considered harmless
- Raised ICP: defined as ICP > 20 mmHg for more than 5 minutes
- Common after severe TBI (53% reported in a recent series)
- When ICP > 40 mmHg, there is neurologic dysfunction and impairment of the brain's electrical activity
- An ICP > 60 mmHg is invariably fatal; pressures in 20–40 mmHg area associated with increased morbidity
- 75% of the patients post severe TBI die due to deformation of tissue, shift, the development of internal hernias and secondary damage to the CNS
- If unchecked an increased ICP may cause death mainly because of deformation of tissue, brain shifts, herniation, and cerebral ischemia
- Aunilateral mass lesion causes distortion of the brain, a reduction of the CSF volume, and, in the closed skull, the formation of internal hernias (including tentorial/uncal herniation
- Increased ICP reduces cerebral blood perfusion
- It is more important to maintain an adequate cerebral perfusion pressure (CPP) than controlling only the ICP
- Cerebral perfusion is calculated by subtracting ICP from mean arterial pressure (MAP). It should remain > 60 mmHg to ensure cerebral blood flow

Factors that May Increase ICP

- Turning head, especially to left side if patient is completely horizontal or head down
- Loud noise
- Vigorous physical therapy
- Chest PT
- Suctioning
- Elevated blood pressure

Methods Used to Monitor ICP

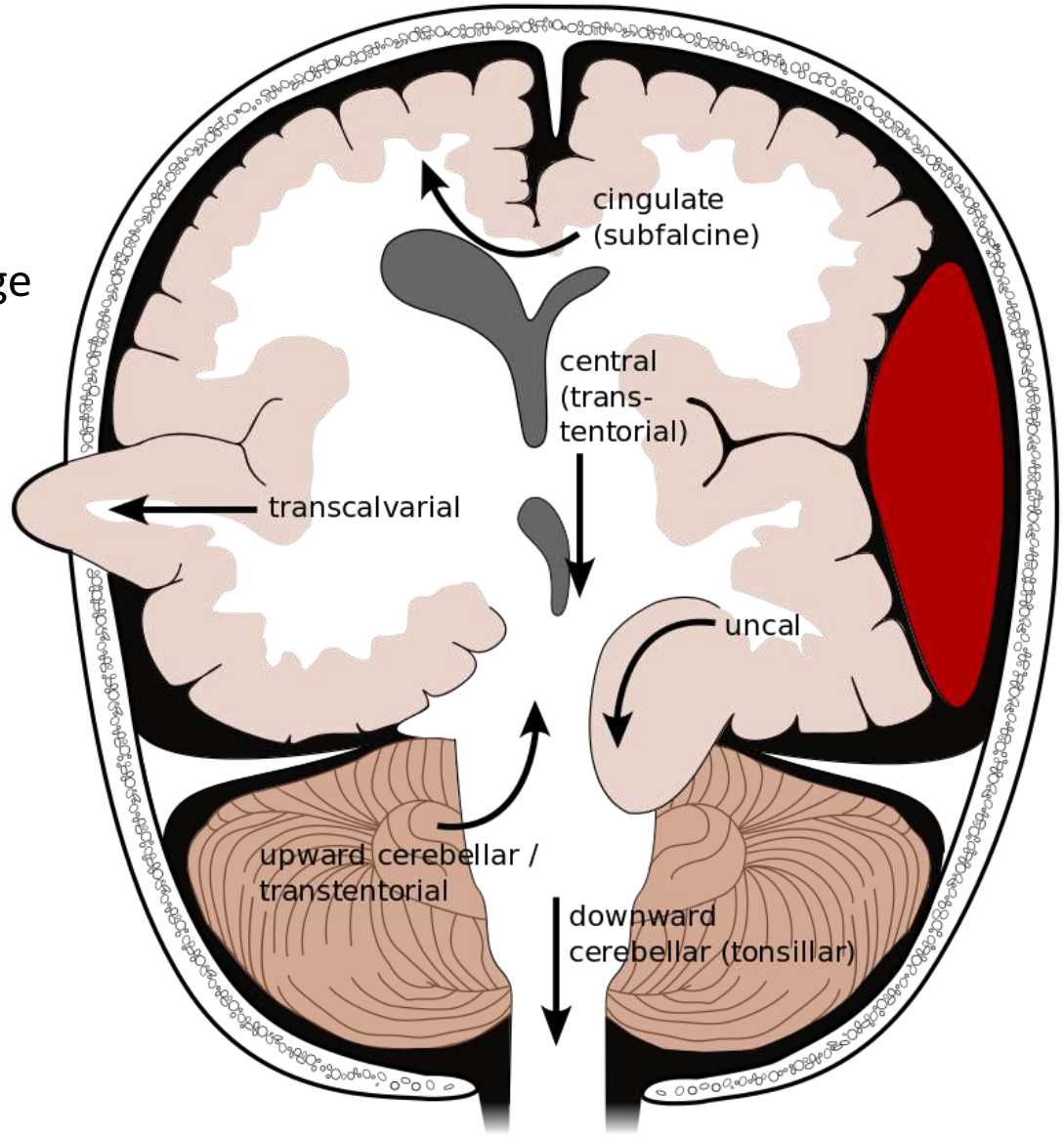
- Papilledema: papilledema is rare in the acute stage after brain injury, despite the fact that ↑ ICP is frequent
- – Usually occurs bilaterally
- – May indicate presence of intracranial mass lesion
- – Develops within 12 to 24 hours in cases of brain trauma and hemorrhage, but, if pronounced, it usually signifies brain tumor or abscess, i.e., a lesion of longer duration
- CT Scan (see earlier)—if CT-Scan equivocal, cysternography may be done
- Lumbar puncture (LP) if no papilledema (must rule out mass lesion first)
- – LP carries a certain risk of causing fatal shift of brain tissue (i.e., herniation)

Management of ICP

- Elevate head of bed 30°
- Intubation and hyperventilation: reduction of PaCO2 through hyperventilation is the most rapid mean of lowering ICP. However, it may negatively impact outcome
- – Hyperventilation should be used with caution as it reduces brain tissue PO2 this may cause brain tissue hypoxia ⇒ this may lead to ischemia ⇒ ischemia may cause further damage in the CNS tissue of the head injury (HI) patient
- – Optimal PaCO2 ~ 30 mmHg
- Osmotic agents (e.g., mannitol)—improves ischemic brain swelling (by diuresis and intravascular fluid shifts)
- Furosemide/acetazolamide may also be used
- Avoid HTN: can increase brain blood volume and increase ICP
- High doses of barbiturates (e.g., thiopental) rapidly lower ICP and suppress electrical brain activity
- Neurosurgical decompression
- Hypothermia may be used to ↓ ICP and it may protect brain tissue by lowering cerebral metabolism. Marion (1997)—treatment with hypothermia for 24 hours in severe TBI patients (GCS 5–7) associated with improved outcome
- Steroids—not proven to be beneficial management of ICP

Temporal Lobe—Tentorial (UNCAL) Herniation

- Uncal herniation results when the medial part of one temporal lobe (uncus and parahippocampal gyrus) is displaced over the edge of the ipsilateral tentorium so as to compress the third cranial nerve, midbrain, cerebral cortex, and subthalamus
- Occurs as a result of increased supratentorial pressure. It is commonly associated with
- hematoma (subdural or epidural) secondary to trauma or to a brain tumor
- • Uncal herniation of the medial temporal lobe produces:
- 1. Stretching of the third cranial nerve (oculomotor nerve) causes ipsilateral pupillary
- dilation; this may lead to complete ipsilateral third nerve palsy (with fixed pupil dilation,
- ptosis, and later, ophthalmoplegia)
- 2. Ipsilateral hemiparesis results due to pressure on the corticospinal tract located in the
- contralateral crus cerebri
- 3. Contralateral hemiparesis may result due to pressure (from edema or mass effect) on
- the precentral motor cortex or the internal capsule
- • In uncal herniation, reduced consciousness and bilateral motor signs appear relatively late.
- Central hyperventilation may also occur late in uncal herniation



Heterotopic Ossification (HO)

HO is the formation of mature lamellar bone in soft tissue
- Common in TBI, with an incidence of 11%–76% (incidence of clinically significant cases is 10%–20%)

Risk factors:
- Prolonged coma (> 2 weeks)
- – Immobility
- – Limb spasticity/↑ tone (in the involved extremity)
- – Associated long-bone fracture
- – Pressure ulcers
- – Edema
- Period of greater risk to develop HO: 3 to 4 months post injury

Signs/Symptoms
- – Most common: pain and ↓ range of motion (ROM)
- – Also: local swelling, erythema, warmth joint, muscle guarding, low-grade fever
- In addition to pain and ↓ ROM, complications of HO include bony ankylosis, peripheral nerve compression, vascular compression, and lymphedema
- Joints most commonly involved:
- 1. Hips (most common)
- 2. Elbows/shoulders
- 3. Knees

Differential Dx: DVT, tumor, septic joint, hematoma, cellulitis, and fracture

Diagnostic Tests/Labs
- *Serum Alkaline Phosphatase (SAP)*
- SAP elevation may be the earliest and least expensive method of detection of HO
- It has poor specificity (may be elevated for multiple reasons, such as fractures, hepatic dysfunction, etc.)
- *Bone Scan*
- Is a sensitive method for early detection of HO
- HO can be seen within the first 2–4 weeks after injury in Phase I (blood-flow phase) and Phase II (blood-pool phase) of a triple phase bone scan, and in Phase III (static phase/delayed images) in 4–8 weeks with normalization by 7 to 12 months *Plain X-rays*
- Require 3 weeks to 2 months post injury to reveal HO. Useful to confirm maturity of HO
- **Prophylaxis**
- ROM exercises
- Control of muscle tone
- Non Steroidal Anti-inflammatory Drugs (NSAIDs)
- Radiation—used perioperatively to inhibit HO in total hip replacement patients; concerns about ↑ risk of neoplasia limit its use in younger patient populations (e.g., TBI patients). Also, as radiation is used prophylactically to prevent HO formation of a particular joint, to use it in TBI patients would require essentially irradiation of the whole body (as HO can develop practically at any joint), which is not practical

Treatment
- Diphosphonates and NSAIDs (particularly indomethacin) have been used on patients to arrest early HO and to prevent postop recurrence, but their efficacy has not been clearly proven (TBI population)
- ROM exercises—used prophylactically to prevent HO and also used as a treatment for developing HO (to prevent ankylosis)
- Surgery—surgical removal of HO indicated only if ↑ in function is a goal (to ↑ hygiene, sitting, etc.)
- Surgical resection usually postponed 12 to 18 months to allow maturation of HO

Hypertension (HTN)

• Frequently observed post-TBI
• Estimated incidence 11%–25% post head injury
• Posttraumatic hypertension usually resolves spontaneously—long-term use of antihypertensive agents is rarely necessary
• Post TBI hypertension related to sympathetic hyperactivity usually seen in severe TBI— demonstrated by ↑ plasma and urine catecholamines levels
• Cases of HTN have been reported secondary to hydrocephalus several years after TBI
• If medication needed, propanolol recommended because:
– ↓ plasma catecholamines levels
– ↓ cardiac index
– ↓ myocardial oxygen demand
– ↓ heart rate
– Improves pulmonary ventilation-perfusion inequality

Venous Thromboembolic Disease

• Venous thromboembolic disease (VTE), including deep vein thrombosis (DVT) and pulmonary embolus (PE), are among the most significant complications of TBI as they are related to ↑ mortality in the rehabilitation setting
• The incidence of DVT in TBI rehabilitation admissions is approximately 10%–18% (Cifu, 1996)
• VTE/DVT often clinically silent in the TBI population, with sudden death from PE being the first clinical sign in 70%–80%
• DVT occurs most commonly in the lower limbs and is traditionally associated with immobility,
paresis, fracture, soft-tissue injuries, and ages > 40
• Remember Virchow's triad: venous stasis, vessel-wall damage, and hypercoagulable state

Prophylactic Regimens for DVT

• Low-dose unfractionated heparin (5000 U q 8 to 12 hours) and low-molecular-weight heparin—adequate anticoagulation generally achieved with these treatments
• Intermittent pneumatic compression—provide effective DVT prophylaxis in patients at risk of bleeding complications
• Warfarin (Coumadin®)
• Inferior vena cava (IVC) filter

Diagnostic tests

Doppler ultrasonography, impedance plethysmography (IPG), 125I-fibrinogen scanning and contrast venography

Treatment of DVT

• Anticoagulation is first initiated with IV heparin or adjusted-dose subcutaneous heparin followed by oral anticoagulation; anticoagulation continued for 3–6 months. IVC filter used when anticoagulation is contraindicated

Posttraumatic Epilepsy/Posttraumatic Seizures (PTS)

Posttraumatic epilepsy is classified as:

1. Generalized (grand mal and tonic-clonic)

2. Partial (*simple,* if consciousness is maintained, or *complex,* if not) The majority of PTS are of the partial type

Posttraumatic seizures are further classified as:

• Immediate PTS—occur within the first 24 hours post injury

• Early PTS—occur within the first week (24 hours to 7 days)

• Late PTS—occur after the first week

– Immediate PTS has better prognosis than early epilepsy; early PTS associated with increased risk of late PTS

Incidence

Varies greatly according to the severity of the injury, the time since the injury, and the presence of risk factors (see below)

• 5% of hospitalized TBI patients (overall, closed-head injury) have late *PTS*

• 4%–5% of hospitalized TBI patients have one or more seizures in the first week after the injury (early PTS) (Rosenthal et al., 1990)

Risk Factors Associated with Late Posttraumatic Seizures:

• Penetrating head injury—33%–50%

• Intracranial hematoma—25%–30%

• Early seizure (> 24 hours to 7 days)—25%

• Depressed skull fracture—3%–70%

• Prolonged coma or posttraumatic amnesia (> 24 hours)—35%

Other Risk Factors

• Dural tearing

• Presence of foreign bodies

• Focal signs such as aphasia and hemiplegia

• Age

• Alcohol abuse

• Use of tricyclic anti-depressants (TCAs)

• 50%–66% of PTS occur within one year; 75%–80% occur within two years; most PTS occur 1–3 months after injury

• 50% of patients with PTS will have only one seizure, and 25% have no more than three episodes

Posttraumatic Epilepsy/ Posttraumatic Seizures (PTS)

Diagnosis
- Clinical
- EEG
  – Standard
  – Sleep-deprived
  – 24 hour
- Prolactin level: ↑ prolactin level confirms true seizure activity (but normal prolactin levels will not exclude seizure activity)

Prophylactic Anticonvulsants
- Greater risk of development of PTS: within the first 2 years post injury
- Prophylactic use of anticonvulsants has not been proven effective in prospective, randomized, controlled studies
- Phenytoin—proven to be effective only during the first week post injury (with no benefit thereafter) at preventing early PTS. There is no proof of change in outcome with prophylactic use of phenytoin (Temkin et al., 1990)

Treatment

It has been suggested that carbamazepine and valproic acid are the drugs of choice (DOC) for the treatment of partial and generalized PTS, respectively.

Medication	Uses	Adverse Reactions
Carbamazepine	• Partial seizures • Tonic-clonic; generalized seizures • Stabilization of agitation and psychotic behavior • Bipolar affective disorder • Neuralgia	• Acute: stupor or coma, hyperirritability, convulsions, respiratory depression • Chronic: drowsiness, vertigo, ataxia, diplopia, blurred vision, nausea, vomiting, aplastic anemia, agranulocytosis, hypersensitivity reactions (dermatitis, eosinophilia, splenomegaly, lymphadenopathy), transient mild leukopenia, transient thrombocytopenia, water retention with decreased serum osmolality and sodium, transient elevation of hepatic enzymes
Gabapentin	• Partial seizures	• Somnolence, dizziness, ataxia, fatigue
Lamotrigine	• Partial seizures • Tonic-clonic; generalized seizures	• Dizziness, ataxia, blurred or double vision, nausea, vomiting, rash, Stevens-Johnson syndrome, disseminated intravascular coagulation
Phenobarbital	• Partial seizures • Tonic-clonic; generalized seizures	• Sedation, irritability, and hyperactivity in children, agitation, confusion, rash, exfoliative dermatitis, hypothrombinemia with hemorrhage in newborns whose mothers took phenobarbital, megaloblastic anemia, osteomalacia • Nystagmus and ataxia at toxic doses
Phenytoin	• Partial seizures • Tonic-clonic; generalized seizures • Neuralgia	• Intravenous administration: cardiac arrhythmias, hypotension, CNS depression • Oral administration: disorders of the cerebellar and vestibular systems (such as nystagmus, ataxia, and vertigo), cerebellar atrophy, blurred vision, mydriasis, diplopia, ophthalmoplegia, behavioral changes (such as hyperactivity, confusion, dullness, drowsiness, and hallucination), increased seizure frequency, gastrointestinal symptoms, gingival hyperplasia, osteomalacia, megaloblastic anemia, hirsutism, transient liver enzyme elevation, decreased antidiuretic hormone secretion leading to hypernatremia, hyperglycemia, glycosuria, hypocalcemia, Stevens-Johnson syndrome, systemic lupus erythematosus, neutropenia, leukopenia, red cell aplasia, agranulocytosis, thrombocytopenia, lymphadenopathy, hypothrombinemia in newborns whose mothers received phenytoin, reactions indicative of drug allergy (skin, bone marrow, liver function)
Valproic Acid	• Partial seizures • Tonic-clonic; generalized seizures • Myoclonic seizures • Absence seizures • Stabilization of agitation and psychotic behavior	• Transient gastrointestinal symptoms such as anorexia, nausea, and vomiting; increased appetite; sedation; ataxia; tremor; rash; alopecia; hepatic enzyme elevation, fulminant hepatitis (rare, but fatal); acute pancreatitis; hyperammoniemia

Withdrawal of Anticonvulsants (For Patients with Posttraumatic Seizures)

- No clear indications. It has been suggested to withdraw anticonvulsant medications after a seizure-free interval of 3 months to 6 months up to 1–2 years. (One to two-year seizure-free interval is used more often as time frame for withdrawal of anticonvulsant therapy.)
- Spontaneous resolution of PTS can occur

Posttraumatic Agitation

- Posttraumatic agitation is usually a self-limiting problem lasting 1–4 weeks
- Reported to occur in 33%–50% of patients with TBI in the acute care setting
- Posttraumatic agitation has been described as

– A subtype of delirium unique to TBI survivors, in which the survivor is in a state of posttraumatic amnesia, and there are excesses of behavior, including a combination of aggression, disinhibition and/or emotional lability/ Delirium is related to, but not sufficient for, a diagnosis of agitation.

- Often no pharmacologic intervention is required

First-line Intervention

- Patient should be maintained in a safe, structured, low-stimulus environment, which is frequently adequate to manage short-term behavior problems. Agitation may be controlled with alterations in environment and staff or family behavior
- Floor beds can eliminate need for restraints
- Use physical restraints only if patient is a danger to self or others; should be applied only to minimal degree and not as a substitute for floor bed or one-to-one or other environmental interventions

Environmental Management of Agitation

1. Reduce the level of stimulation in the environment

- Place patient in quiet private room
- Remove noxious stimuli if possible, tubes, catheters, restraints, traction
- Limit unnecessary sounds, TV, radio, background conversations
- Limit number of visitors
- Staff to behave in a calm and reassuring manner
- Limit number and length of therapy sessions
- Provide therapies in patient room

2. Protect patient from harming self or others

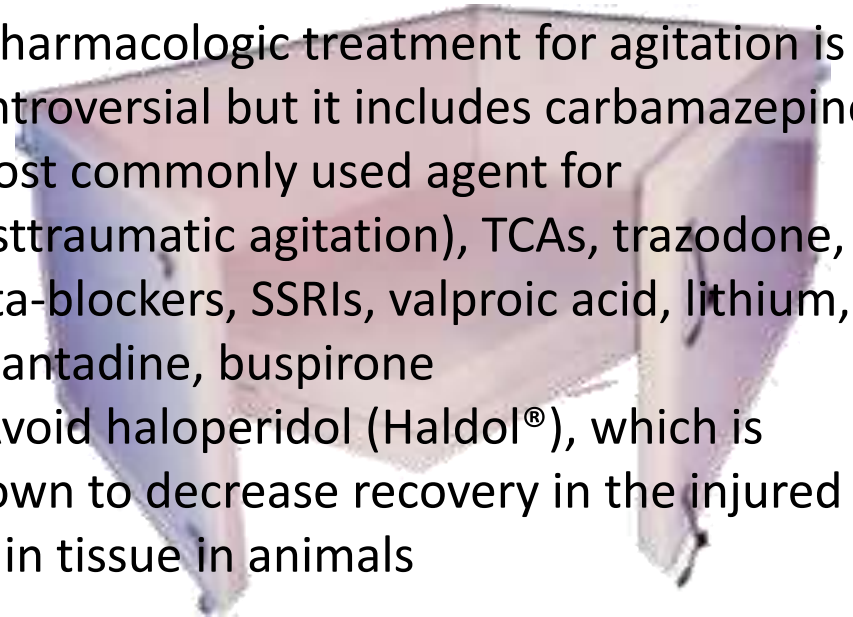
- Place patient in a floor bed with padded side panels (Craig bed)
- Assign 1:1 or 1:2 sitter to observe patient and ensure safety
- Avoid taking patient off unit
- Place patient in locked ward

3. Reduce patient's cognitive confusion

- One person speaking to patient at a time
- Maintain staff to work with patient
- Minimize contact with unfamiliar staff
- Communicate to patient briefly and simple, one idea at a time

4. Tolerate restlessness when possible

- Allow patient to thrash about in floor bed
- Allow patient to pace around unit with 1:1 supervision
- Allow confused patient to be verbally inappropriate



• Pharmacologic treatment for agitation is controversial but it includes carbamazepine (most commonly used agent for posttraumatic agitation), TCAs, trazodone, beta-blockers, SSRIs, valproic acid, lithium, amantadine, buspirone

• Avoid haloperidol (Haldol®), which is shown to decrease recovery in the injured brain tissue in animals

Urinary Dysfunction

• Neurogenic bladder with uninhibited detrusor reflex (contraction)

• TBI patients are frequently incontinent, usually presenting a disinhibited type of neurogenic bladder, in which the bladder volume is reduced but empties completely with normal postvoiding intravesicular volumes ⇒ Small voids with normal residuals

• For this type of dysfunction, a time-void program is usually helpful, in which the patient is offered the urinal or commode at a regular scheduled interval

• Anticholinergic meds (to ↑ bladder capacity) may also be used

Cranial Neuropathies

Most frequently affected cranial nerves in blunt head trauma:

• Olfactory nerve (CN I)

• Facial nerve (CN VII)

• Audiovestibular/vestibulocochlear nerve (CN VIII)

• CN affected with intermediate frequency

– optic nerve (CNII)

– ocular motor nerves (CN IV > CN III > CN VI)

• Trigeminal nerve (CN V) and the lower cranial nerves are rarely involved

CN I (Olfactory)

- Cranial nerve most often damaged by blunt head trauma
- Overall incidence ~ 7%, rising to 30% with severe head injuries or anterior fossa fractures
- Anosmia (loss of the ability to smell) is more common with occipital than with frontal blows and can result from trauma to any part of the head
- Anosmia and an apparent loss of taste result from CN I disruption thought to be secondary to a displacement of the brain with tearing of the olfactory nerve filaments in or near the cribriform plate through which they course
- Often associated with ↓ appetite/altered feeding behavior
- Associated with CSF rhinorrhea
- Recovery occurs in > one-third of cases, usually during the first 3 months

CN VII (Facial)

- Especially vulnerable to penetrating or blunt trauma to head because of its long, tortuous course through the temporal bone

CN VIII (Vestibulocochlear)

- Damage to the vestibulocochlear nerve results in loss of hearing or in postural vertigo and nystagmus coming on immediately after the trauma

CN II (Optic Nerve)

- Partial damage may result in scotomas and a troublesome blurring of vision or as homonymous hemianopsia. If nerve is completely involved or transected, patient will develop complete blindness (pupil dilated, unreactive to direct light but reactive to light stimulus to the opposite eye (consensual light reflex)

Endocrine Complications

Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)

• Water retention resulting from excessive antidiuretic hormone (ADH) secretion from the neurohypophysis secondary to multiple causes including head trauma

• In SIADH, ADH excess considered to be inappropriate because it occurs in the presence of plasma hypo-osmolality

• In SIADH, Na+ excretion in the urine is maintained by hypervolemia, suppression of the renin-angiotensin-aldosterone system, and ↑ in the plasma concentration atrial natriuretic peptide (usually > 20 mmol/L)

Signs and Symptoms in SIADH:

• In mild SIADH (with Na+ 130–135), or in gradually developing SIADH, symptoms may be absent or limited to anorexia and nausea/vomiting

• In severe SIADH (with significant hyponatremia) or in acute onset SIADH, there might be an increase in body weight and symptoms of cerebral edema—restlessness, irritability, confusion, coma, convulsions

• Edema (peripheral/soft tissue) almost always absent

Treatment

• Fluid restriction to ~ 1.0 L/day (800 ml to 1.2 L/day) (either alone or with a loop diuretic)

• Careful daily monitoring of weight changes and serum Na+ until sodium level > 135 mmol/L

• Hypertonic saline (e.g., 5% NaCl solution), 200–300 ml, should be infused IV over 3–4 hours in patients with severe symptoms as confusion, convulsions, or coma

• It is important not to raise Na+ concentration too rapidly to avoid development of serious neurological damage, pontine myelinolysis, or congestive heart failure (CHF); sodium may be corrected not more than 10 mEq/L over 24 hours until sodium reaches 125 mEq/L

• Chronic SIADH may be treated with demeclocycline, which normalizes serum Na+ by inhibiting ADH action in the kidney; lithium carbonate acts similarly but is rarely used because it is more toxic

Cerebral Salt-Wasting (CSW) Syndrome

• CSW is another common cause of hyponatremia in TBI; may probably be a more common cause of hyponatremia in TBI patients than SIADH

• Hyponatremia in TBI is generally present in a hypotonic setting with either normal extracellular volume (isovolemia = SIADH) or reduced extracellular volume (hypovolemia = CSW).

• CSW is thought to occur because of direct neural effect on renal tubular function

• In CSW, hyponatremia is not dilutional (as in SIADH)—CSW patients are, in fact, volume depleted

• *Hallmark of CSW*

– Decreased blood volume (↓ extracellular volume = hypovolemia) secondary to sodium loss (in urine) ⇒ this triggers ↑ in ADH secretion that is appropriate rather than inappropriate (differentiating this condition from SIADH)

– Signs of dehydration

• *Treatment*

• Hydration/fluid replacement and electrolyte (Na+) correction

• It is important to differentiate CSW from SIADH and to recognize that there is water depletion in this condition, because treating it with fluid restriction (adequate Tx for SIADH) may further ↓ the extracellular fluid with disastrous results to the patient

Diabetes Insipidus (DI)

• Hallmark: Deficiency of ADH (vasopressin)

• May occur in severe head injuries; often associated with fractures of the skull

• A fracture in or near the sella turcica may tear the stalk of the pituitary gland, with resulting DI (due to disruption of ADH secretion from the posterior lobe of the pituitary) in addition to other clinical syndromes depending on the extent of the lesion

• Spontaneous remissions of traumatic DI may occur even after 6 month, presumably because of regeneration of disrupted axons within the pituitary stalk

Clinical Manifestations

• Polyuria, excessive thirst and polydipsia

• Urinary concentration (osm < 290 mmol/kg, SG 1.010) is below that of the serum in severe cases but may be higher than that of serum (290–600 mmol/kg) in mild DI

• Normal function of the thirst center ensures that polydipsia closely matches polyuria, so dehydration is seldom detectable except by a mild elevation of serum Na+

• However, when replenishment of excreted water is inadequate, dehydration may become severe, causing weakness, fever, psychic disturbances, prostration, and death

• These features are associated with a rising serum osmolality and serum Na+ concentration, the latter is sometimes > 175 mmol/L

Treatment

• Hormone replacement

– DDAVP® (desmopressin acetate)—analog of antidiuretic hormone (ADH) with prolonged antidiuretic effect and no significant pressor activity

– May be given intranasally or intramuscular (IM)

• Chlorpropamide potentiates the effects of ADH on the renal tubules—used in partial ADH deficiency

Comparison of SIADH, CSW and DI

	SIADH	DI	CSW syndrome
Serum ADH (rarely done as routine lab work)	↑ (inappropriately elevated)	↓	↑ (appropriately elevated)
Diagnostic Labs			
Serum Na^+	↓	↑	↓
Serum osmolality	↓	↑	↓
Extracellular volume	Normal (isovolemic)	Normal (isovolemic)	Reduced (hypovolemic)
Urine osmolality and SG	↑ (concentrated urine with osmolality usually > 300 mmol/kg)	↓	Normal

Guidelines include the following:

Intracranial pressure (ICP) monitoring is recommended

Advanced neuromonitoring (brain oxygenation) should be reserved for patients with no contraindications to invasive neuromonitoring and patients who are not brain dead

- Targeting a threshold of under 20 mm Hg in ICP treatment is recommended
- Maintaining a minimum cerebral perfusion pressure (CPP) of 40 mm Hg is recommended
- Moderate (32-33°C) hypothermia is recommended for controlling ICP but is not recommended over normothermia for improving overall outcomes
- Decompressive craniectomy (DC) is recommended for treating neurologic deterioration, herniation, or intracranial hypertension refractory to medical management
- Initiating early enteral nutritional support (within 72 hours from injury) is recommended for decreasing mortality and improving outcomes
- Corticosteroids are not recommended for ICP

- Bolus hyperosmolar therapy (HTS) of 3% saline is recommended for patients with ICP; the recommended effective doses range from 2-5 mL/kg over 10-20 minutes
- For refractory ICP, a bolus of 23.4% HTS is recommended
- Avoiding bolus administration of midazolam and/or fentanyl during ICP crises is recommended due to risks of cerebral hypoperfusion
- Draining cerebrospinal fluid (CSF) through an external ventricular drain (EVD) is recommended for managing increased ICP
- Prophylactic treatment is recommended for reducing occurrence of early (within 7 days) posttraumatic seizures (PTSs)
- In hemodynamically stable patients with refractory ICP, high-dose barbiturate therapy is recommended

Kochanek PM, Tasker RC, Carney N, et al. Guidelines for the Management of Pediatric Severe Traumatic Brain Injury, Third Edition: Update of the Brain Trauma Foundation Guidelines, Executive Summary. *Pediatr Crit Care Med*. 2019 Mar. 20 (3):280-9.

Ventilation therapy

- Prolonged prophylactic hyperventilation is not recommended

Anesthetic agent

- Administration of barbiturates as prophylaxis against the development of intracranial hypertension is not recommended
- High-dose barbiturate administration is recommended to control refractory elevated ICP
- Propofol is not recommended for improvement in mortality or 6-month outcomes

Intracranial pressure monitor should be monitored but in low technology setting CT scan and clinical examination can be used

Cerebral perfusion pressure monitoring is recommended

Jugular Bulb Monitoring of Arteriovenous Oxygen Content Difference(AVDO2) monitoring is recommended to reduce motality

Thank you for your attention