

# Traumatic Brain Injury: A Review

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## Abstract

This article reviews issues related to traumatic brain injuries.

## EPIDEMIOLOGY

- Approximately 1.6 million head injuries occur every year in the U.S.
- Nearly 60,000 deaths from TBI yearly
- Accounts for ~50% of deaths from trauma
- Financial burden: \$100 billion annually
- Mortality: 25-35%
- Primary contributing factors: MVA, Falls, Alcohol

## PATHOPHYSIOLOGY:

### PRIMARY BRAIN INJURY:

Focal: cerebral contusions, hematomas, fractures  
Diffuse axonal injury: shearing/inertial forces

### SECONDARY BRAIN INJURY:

Due to neuronal damage from systemic physiologic responses to the initial injury  
Hypotension, hypoxia

### CEREBROVASCULAR REGULATION:

#### MONROE-KELLIE DOCTRINE:

The cranial vault is a fixed space consisting of 3 compartments:  
Parenchyma (80%)  
CSF (10%)  
Blood (10%)

Therefore, expansion of one compartment results in a compensatory decrease in another in order to maintain ICP

Cerebral Blood Flow is determined by:

CPP

PaO<sub>2</sub>

PaCO<sub>2</sub>

Cerebral metabolic demand

Normal CBF: 50 ml/min/100g of brain tissue or 60-160 mm Hg)

Autoregulation: CBF is maintained over a wide range of CPP; between 50-150 mm Hg, by autoregulation of cerebral vasculature, but becomes deranged with brain injury (CBF becomes linearly dependent on MAP)

Effects of BP, PaO<sub>2</sub>, and PaCO<sub>2</sub> on CBF:

Decreased paO<sub>2</sub>/CaO<sub>2</sub>: ↓ CBF

Cerebral vasodilation

↓ cerebral blood volume

↑ ICP

Increased paCO<sub>2</sub>: ↓ CBF

Cerebral vasodilation

CPP = MAP - ICP

Normal: 70-100 mm Hg

Used to assess the adequacy of cerebral perfusion because CBF is

Difficult to measure clinically

Evolution of CBF after TBI:

Initial hypoperfusion phase

Hyperemia (increased CBF)

Second hypoperfusion phase (cerebral vasospasm)

Recovery phase (CBF ~ CMRO<sub>2</sub>)

Begins 2-3 weeks after onset

Continues for weeks, months

ICP: Reflects the volume of: Brain parenchyma  
CSF

Intravascular blood

Normal: < 15 mm Hg

CBV + CSF + Parenchyma

Ischemia Hydrocephalus Edema

Acidosis comm.vs.noncom. Ischemia

↓ paCO<sub>2</sub> (cytotoxic)

Hyperthermia Vasogenic

↑ venous P (impaired BBB)

**CLINICAL SIGNS OF ELEVATED ICP:**

- Depressed level of consciousness
- Hypertension
- +/- Bradycardia
- Irregular respiratory pattern
- Headache
- Nausea/Vomiting
- Papilledema
- Third or Sixth CN palsies

**INITIAL MANAGEMENT AND RESUSCITATION:**

Primary survey (ABCDE's)

Monitor hemodynamic and oxygenation status

Avoid or immediately correct: Hypotension (SBP < 90 mm Hg)

Hypoxia (paO<sub>2</sub> < 60, O<sub>2</sub> Saturation < 90%)

Maintain MAP > 90 mm Hg to allow CPP > 70 mm Hg

Secondary survey (AMPLE hx)

Neurological reevaluation: GCS (pre- & postresuscitation scores important)

**EYE OPENING**

- Spontaneous 4
- Voice 3
- Pain 2
- None 1

**BEST VERBAL RESPONSE**

- Oriented 5

- Confused 4
- Inappr. Words 3
- Incompr. Sounds 2
- None 1

**BEST MOTOR RESPONSE**

- Obeys commands 6
- Localizes pain 5
- Withdraws to pain 4
- Flexion 3
- Extension 2
- None 1

- GCS Severity of TBI
- 13 -15 Mild
- 9 - 12 Moderate
- 3 -8 Severe

- Pupillary light reaction
- Oculocephalics (Doll's eyes)
- Oculovestibulars (Caloric)

**MANAGEMENT OF TBI:**

General Principles: CT of head & Neurosurgical evaluation

1. Patient positioning: HOB > 30 degrees  
Cervical collar  
Head midline
2. Fluid management: NS, LR
3. Fever management
4. Stress ulcer prophylaxis
5. DVT prophylaxis
6. Monitor electrolytes (Na, Mg)

**MANAGEMENT OF INTRACRANIAL HYPERTENSION:**

Airway Management:

Intubation-Indications: Respiratory distress Motor posturing/Absence of response to pain

Hypoxia/Hypercapnia  
GCS < 8  
Seizures  
Increased ICP  
Need for analgesics/sedatives  
Significant associated injuries

Rapid sequence intubation recommended:  
Preparation  
Preoxygenation  
Pretreatment (LOAD)  
Paralysis AFTER induction  
Protection + Positioning  
Placement with proof  
Post Intubation management

### **TREATMENT OF ELEVATED ICP: REDUCTION OF CEREBRAL BLOOD VOLUME:**

Avoid hypoxia or hypercarbia  
Hyperventilation - no longer used in the modern era

In the absence of increased ICP, chronic prolonged  
Hyperventilation therapy (paCO<sub>2</sub> < 25) should be avoided  
after severe TBI.

### **GUIDELINE:**

The use of prophylactic hyperventilation (PaCO<sub>2</sub> <35)  
therapy during the first 24 hours after severe TBI should be  
avoided due to concerns of compromising cerebral perfusion  
at a time when CBF is reduced.

### **OPTIONS:**

Hyperventilation therapy may be necessary for brief periods  
when there is an acute neurological deterioration or for  
longer periods if there is ICH refractory to sedation,  
paralysis, CSF drainage, & osmotic diuretics. Jugular venous  
oxygen saturation, arterial-jugular venous O<sub>2</sub> content  
differences & CBF monitoring may help identify cerebral  
ischemia if hyperventilation resulting in a PaCO<sub>2</sub> < 30 is  
necessary.

Promote venous drainage (elevate HOB)  
Treat severe hypertension  
Medications

### **BARBITURATE COMA:**

Guideline: High dose barbiturate therapy may be considered  
in HD stable salvageable SHI patients with ICH refractory to  
maximal medical and surgical interventions

Pentobarbital: Eisenberg RCT:  
Loading dose: 10 mg/kg over 30 minutes  
5 mg/kg q 1 hour x 3  
Maintenance: 1 mg/kg/hr

Monitor EEG for 30-60s. burst suppression pattern

### **REDUCTION OF CSF VOLUME:**

Decrease CSF production (lasix, acetazolamide)

Ventricular drainage/ICP monitoring

Indications: SHI (GCS <8 after resuscitation) + abnormal  
CT

SHI with a normal CT if >2 present at admission:

Age > 40  
Unilateral/bilateral motor posturing  
SBP <90 mm Hg

Not routinely indicated in patients with mild or moderate  
head injury

Physicians may choose to monitor ICP in conscious patients  
with traumatic mass lesions

Initiate ICP therapy at an upper threshold of 20-25 mm Hg  
Published clinical experience indicates that ICP monitoring:

1. Aids in the earlier detection of intracranial mass lesions
2. Can limit the indiscriminate use of therapies to control ICP which themselves can be potentially harmful
3. Can reduce ICP by CSF drainage
4. Helps in determining prognosis
5. May improve outcome

### **REDUCTION OF BRAIN TISSUE VOLUME:**

Administer Lasix (0.5 - 1 mg/kg IV)

Osmotic therapy:

Guidelines:

Mannitol is effective for control of elevated ICP after SHI  
Effective doses range from 0.25 - 1 g/kg  
(Effect begins within 10-20'; peaks @ 20-60'; duration 4-6 h)  
(Monitor Sna, Sosm; maintain Sosm <320)

(Maintain euvolemia by adequate fluid replacement)

Options:

Indications prior to ICP monitoring:

Signs of transtentorial herniation

Progressive neurological deterioration not attributable to extracranial pathology

Intermittent boluses may be more effective than continuous infusion

Surgical Decompression: Decompressive hemicraniectomy

**PROPHYLACTIC ANTICONVULSANTS:**

Standard: The prophylactic use of Phenytoin, Carbamazepime, or

Phenobarbital is NOT recommended for prevention of late post-traumatic seizures (> 7 days)

Options: Anticonvulsants may be used to prevent early (<7 days)

PTS in patients at high risk for seizures following head injury

Risk factors include: GCS score <10

Cortical contusion

Depressed skull fracture

SDH, Epidural hematoma

Intracerebral hematoma

Penetrating head wound

Seizure within 24 hours of injury

Phenytoin and Carbamazepime have been shown to be effective.

However, available evidence does not indicate that prevention of early PTS improves outcome following head injury.

**MEDICAL MANAGEMENT:**

Nutritional support

Patients with SHI: Hypermetabolic, hypercatabolic, hyperglycemic

Altered immune function (cellular immunity)

Altered gastric motility ( ICP, Dilantin, NMB)

Early (within first 24h) administration of enteral nutrition blunts the hypermetabolic response after injury in critically ill patients

Initial goal: 25-30 kcal/kg/day

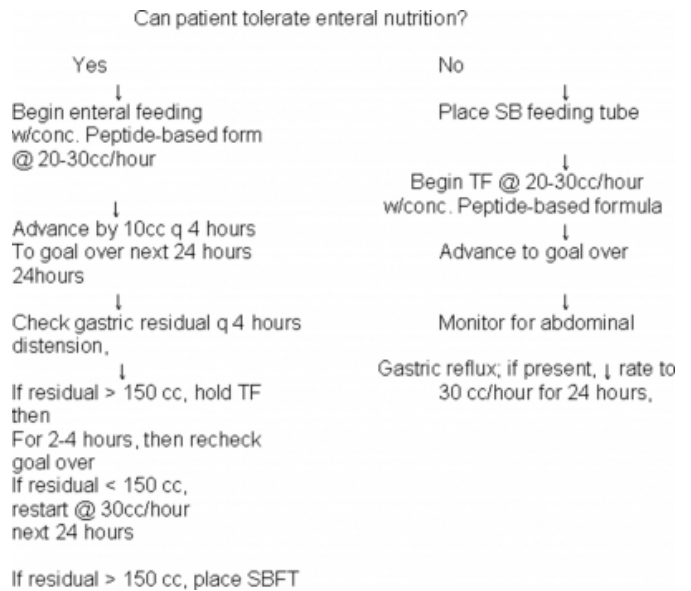
1.5-2.0 g protein/kg/day

Replace 140% of RME in nonparalyzed patients, & 100% of RME in paralyzed patients

Provide at least 15% of calories as protein by the 7th post-traumatic day.

Algorithm:

**Figure 1**



Change to intact protein/high nitrogen formula when TF tolerated for 3 days

**MEDICAL COMPLICATIONS:**

CV: MI

Pulmonary: PNA, Aspiration (peaks 5-10d post-trauma)

ARDS

Endocrine/Metabolic: SIADH, Cerebral salt wasting

DI

Skin: Pressure ulcers

Electrolyte disturbances

Hypotension

PNA\* Independent predictors of increased

Sepsis\* morbidity/mortality

Coagulopathy: Peaks during the first 3 days

Due to: Blood transfusion/resuscitation

Brain tissue injury/destruction

Medications

(Sepsis)

Associated with the systemic release of parenchymal tissue thromboplastin, etc.

DIC ~ degree of brain injury

Principles of therapy:

Routinely monitor platelets, coags

Maintain platelets > 100,000  
Treat the underlying pathology

**PROGNOSIS AND OUTCOME:**

Extent of recovery is dependent on:  
Patient's age

Severity of injury  
Type of intracranial disorder  
Recovery is often slow  
Significant disability is common  
Rehabilitation

**References**

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