# 34. Neurologic Critical Care

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*A 48-year-old female helmeted bicyclist was admitted to the ICU after a fall down a 15-foot embankment. She was intubated in the field and had a Glasgow Coma Score (GCS) of 5 upon arrival to the trauma bay. Her initial head CT scan revealed a large subarachnoid hemorrhage. A bedside ventriculostomy was placed by the neurosurgeon and the initial ICP was 28. Hyperosmolar therapy (mannitol alternating with 23.4 % hypertonic saline) was instituted. The patient was placed in a 30 degree head up position to facilitate venous drainage. Cerebral perfusion pressure was maintained with fluids and vasoactive medications.*

Neurologic problems may be present in critically ill patients either as the primary diagnosis or as a complication of the underlying process. General management principles are based on the observation that injured brain tissue poorly tolerates subsequent insults. Even apparently small decreases in blood pressure, arterial oxygen saturation, and serum sodium may result in clinical deterioration; therefore, meticulous monitoring and interventions are the mainstay of neurologic critical care. Although several monitoring techniques are listed below, the best monitor of neurologic function still consists of sequential neurologic exams by a trained clinician.

## I. Neurophysiology

A. The cranial vault has three components - brain tissue, cerebrospinal fluid (CSF), and blood all of which are housed in a non-compliant skull. Thus, an increase in volume of any of these components (cerebral edema, hydrocephalus, space occupying mass, vasodilation) will lead to increased intracranial pressure (ICP) and decreased cerebral perfusion pressure (CPP) that may, if sustained, cause neurologic damage and even death.

B. Cerebral Blood Flow (CBF)
   1. Highly regulated to match the metabolic needs of the brain.
   2. Determined by PaO2, PaCO2 and autoregulation
   3. Hypoxia and hypercapnia produce vasodilatation and increased CBF
   4. Pressure Autoregulation: in normal subjects with intact blood brain barriers, CBF is maintained constant over the normal range of mean arterial pressure (MAP: 50 – 150 mmHg).

C. Other factors that may have deleterious effects on neurologic outcome include
   1. Hyperthermia
   2. Hyperglycemia
   3. Hypoxemia
   4. Anemia

## II. Neuromonitoring

A. Neurologic examination and the Glasgow Coma Scale (GCS)

B. Neuroimaging
   1. Head CT
   2. Brain MRI
   3. Angiography

C. ICP monitoring
   1. Types
      a) Catheter-based devices (ventriculostomies) : They are more widely used and allow continuous ICP monitoring and therapeutic CSF drainage
      b) Transducer-based devices: The tip is placed in the brain parenchyma or the subdural space
   2. Indications
      a) Severe head injury (GCS < 8) and abnormal head CT finding
      b) Severe head injury (GCS < 8) and normal head CT if
         (1) > 40 years old

(2) Posturing
(3) Systolic BP < 90 mmHg

3. Complications
   a) Malfunction
   b) Infection
   c) Bleeding

D. Transcranial doppler ultrasonography (TCD)
E. Jugular venous oxygen saturation (SjvO2)
F. Brain tissue oxygen partial pressure (PbtO2)
G. Microdialysis
H. Near-infrared spectroscopy
I. Continuous EEG

## III. Disease States

### A. Altered consciousness

1. Differential diagnosis
   a) Normal sleep
   b) Pathologic: hypoglycemia, hypoxia, drug overdose, poisoning, trauma, postictal state, meningitis, encephalitis, stroke, post cardiac arrest
   c) Psychogenic coma
   d) Locked-in state
2. Diagnosis
   a) Detailed history and physical with thorough neurologic examination
   b) Neuroimaging
   c) Lumbar puncture to obtain CSF
   d) EEG
3. Management
   a) Supportive measures
   b) "Coma Cocktail" (mixture of drugs to reverse common causes of unconsciousness - dextrose, thiamine, naloxone, and flumazenil)
   c) Definitive treatment
   d) Induced hypothermia for post cardiac arrest cases

### B. Cerebrovascular disease

1. **Ischemic cerebrovascular disease (acute ischemic stroke)**
   a) Etiology
      (1) Embolic
      (2) Atherothrombotic
      (3) Lacunar
      (4) Watershed
   b) Diagnosis
      (1) History and physical exam
      (2) Neuroimaging
      (3) Echocardiography
      (4) Carotid ultrasound
   c) Management
      (1) Thrombolysis within 3 hours of onset
      (2) BP should not be aggressively lowered
      (3) Antiplatelet therapy
      (4) Supportive measures
      (5) Delayed anticoagulation for embolic stroke
2. **Intracerebral hemorrhage**
   a) Etiology
      (1) Hypertension
      (2) Rupture of cerebral aneurysm or arteriovenous malformation (AVM)
      (3) Traumatic head injury
      (4) Coagulation disorders and spontaneous bleeding
   b) Diagnosis
      (1) History and physical exam

(2) Neuroimaging
(a) CT scan
(b) MRI
(c) Angiography
(3) Lumbar puncture is contraindicated
(4) Coagulation profile

c) Management
(1) Supportive measures
(2) Antihypertensives
(3) ICP management
(4) Surgical intervention

3. **Subarachnoid hemorrhage (SAH)**

a) SAH is a systemic disease that results from a ruptured cerebral aneurysm. It carries significant neurologic morbidity and mortality.

b) Diagnosis
(1) History "worst headache ever" and physical
(2) Head CT/ CTA
(3) Lumbar puncture
(4) MRI/ MRA
(5) Angiography

c) Associated complications
(1) Rebleeding
(2) Vasospasm
(3) Hydrocephalus
(4) Fluid and electrolytes abnormalities
(5) Cardiac and pulmonary abnormalities
(6) Seizure

d) Management
(1) Supportive measures
(2) Rebleeding
(a) Angiography for aneurysm coiling
(b) Craniotomy for aneurysm clipping
(c) Antihypertensives
(d) Antifibrinolytics
(3) Vasospasm
(a) Hypertensive hypervolemic therapy (HHT)
(b) Angioplasty
(c) Calcium antagonists
(4) Hydrocephalus
(a) Ventriculostomy
(b) Shunting

C. **Traumatic brain injury**

1. Pathology
a) Primary brain injury (direct traumatic insult)
b) Secondary brain injury (due to hypotension and hypoxia)
c) Intracranial hypertension
d) Brain herniation and brain death

2. Diagnosis
a) History and physical
b) GCS
c) Neuroimaging: primarily head CT
d) ICP monitoring

3. Management
a) Supportive measures to prevent secondary injury
b) ICP management
(1) mild hyperventilation for acute elevation of ICP
(2) optimize oxygenation
(3) head of bed elevation

(4) sedation
(5) prevent hyperthermia and hyperglycemia
(6) hyperosmolar therapy
(7) CSF drainage
(8) induced coma
(9) surgical decompression

D. Traumatic spinal cord injury
1. Pathology
a) Primary spinal cord injury
b) Secondary spinal cord injury
c) Compete spinal cord injury
d) Incomplete spinal cord injury
e) Spinal shock
(1) associated with complete spinal cord injury at a high level
(2) hypotension
(3) bradycardia
(4) hypothermia
2. Diagnosis
a) History and physical
(1) level of injury
(2) completeness of injury
(3) presence of spinal shock
b) Neuroimaging
(1) Plain radiography
(2) CT scan
(3) MRI
3. Management
a) Spinal precautions
b) Supportive measures including effective management of spinal shock
c) Methylprednisolone (SoluMedrol) protocol
d) Decompressive and/or stabilization surgery

E. Status epilepticus
1. Etiology
a) Idiopathic
b) Stroke
c) Anoxic brain injury
d) Metabolic
e) Pharmacologic
f) Infection
2. Diagnosis
a) Neurologic exam
b) EEG
c) CT scan
3. Management
a) Evaluation and stabilization
b) cardiopulmonary support
c) glucose and thiamine
d) Seizure control
e) Benzodiazepines
f) phenytoin
g) fosphenytoin
h) Phenobarbital
i) Refractory status epilepticus
j) intubation and hemodynamic support
k) continuous EEG monitoring
l) pentobarbital, propofol or benzodiazepines to obtain burst suppression on EEG

F. **Guillain-Barré syndrome**
1. Pathology

a) Multifocal demyelination of cranial and peripheral nerves
b) Immune-mediated
c) Progressive weakness and areflexia
d) Autonomic dysfunction
e) Spontaneous recovery, weeks to months

2. Diagnosis
   a) History and physical
   b) CSF analysis
   c) EMG and nerve conduction studies
   d) Autoantibodies
3. Management
   a) Close observation of respiratory status
   b) Hemodynamic monitoring
   c) Immunotherapy
   d) Plasmapheresis

G. **Myasthenia gravis**

1. Pathology
   a) Autoantibodies bind to acetylcholine receptors blocking the receptors and decreasing their density
   b) Muscle weakness and fatigue
2. Diagnosis
   a) History and physical
   b) Edrophonium test
   c) Antibodies to acetylcholine receptors
   d) EMG
   e) Chest CT for evidence of thymoma
3. Management
   a) Supportive care
   b) Cholinesterase inhibitors
   c) Immunotherapy
   d) Plasmapheresis
   e) Corticosteroids
   f) Thymectomy

This chapter is a revision of the original chapter authored by C. Lee Parmley, M.D., J.D. and Steven J. Allen, M.D.

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## QUESTIONS

34.1. A 27-year-old man has had a right middle cerebral artery aneurysm uneventfully clipped 2 days following a subarachnoid hemorrhage. On the third postoperative day, he develops left-sided weakness over one hour. Of the following, which statement is most likely to be true?

A. He should be immediately returned to the OR for replacement of the clip.
B. His filling pressures should be elevated.
C. He should be hyperventilated and sedated.
D. He should receive steroids and mannitol.
E. Thrombolysis is likely to reverse these changes.

34.2. A patient with a severe headache, fever, and stiff neck is UNLIKELY to have the following finding on lumbar puncture:

A. A normal lumbar puncture
B. Elevated WBCs in the CSF
C. Blood in the CSF
D. Elevated CSF glucose
E. Elevated CSF protein

34.3. Which of the following statements is most correct about the Guillain-Barré Syndrome?
- A. It usually begins rapidly and may take months to resolve.
- B. It will affect upper extremities more frequently than legs.
- C. Bulbar involvement is almost always fatal.
- D. Plasmapheresis is the treatment of choice in mild cases.
- E. Autonomic instability is common.

34.4. The malignant neuroleptic syndrome:
- A. Is a variant of malignant hyperthermia.
- B. Is reversible with dantrolene.
- C. Can be triggered by succinylcholine in ICU patients.
- D. Can be the cause of renal failure.
- E. Is self-limited and not associated with significant morbidity.

34.5. A changing localized neurologic pattern (waxing and waning) is most consistent with:
- A. Cerebral vasospasm
- B. An expanding mass lesion
- C. A peripheral neuropathy
- D. A cerebral ischemic infarct
- E. Increased ICP

34.6. Which of the following are concerns in the comatose, head injured patient?
- A. Aspiration of gastric contents
- B. Increased neurologic damage from increased ICP
- C. Gastrointestinal hemorrhage
- D. Deep venous thrombosis and pulmonary embolism
- E. All of the above

34.7. All the following factors may have deleterious effects on neurologic outcome EXCEPT:
- A. Hypoxia
- B. Anemia
- C. Hyperglycemia
- D. Diminished autoregulation
- E. Hypothermia

34.8. The following are correct about acute ischemic stroke EXCEPT:
- A. It could be caused by an embolic event
- B. Blood pressure should be maintained lower but close to baseline
- C. Echocardiography may be required
- D. Thrombolysis should be initiated within six hours of the onset of stroke
- E. Antiplatelet therapy is recommended

34.9. Which on of the following regarding ICP is INCORRECT?
- A. ICP monitoring is indicated in a 70 year old patient with normal head CT and GCS of 7.
- B. ICP monitoring is not indicated in a 35 year old trauma patient with normal head CT and GCS of 3.
- C. Transducer based catheters for ICP monitoring are less widely used and do not allow CSF drainage.
- D. ICP monitoring is indicated in a 20 year old motorcycle crash patient with abnormal CT and GCS of 12.
- E. Complications of ICP monitoring include infection, hemorrhage and malfunction.

34.10. All the following are used to treat myasthenia gravis EXCEPT:
A. Pyridostigmine
B. Edrophonium
C. Plasmapheresis
D. Corticosteroids
E. Thymectomy

# 35. Traumatic Brain Injury

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*You are called to assess the airway of an 18-year-old male who was an unrestrained passenger in a motor vehicle crash (MVC). He open his eyes to pain, does not follows any commands and has an incoherent speech. The ED physician wants him to have a head CT SCAN first to rule out an intracranial injury as he believes the patient is just intoxicated and will get better as his EtOH levels decrease.*

I. Introduction

Severe traumatic brain injury (TBI) is a serious health problem in the US with an incidence of 1.6 million victim/yr.[1] Furthermore, TBI is associated with:

A. 60,000 deaths /year
B. 70-90,000 victims left with permanent neuro damage
C. Most common in males aged 15 to 24
D. 50% of victims have significant concurrent injuries
E. ETOH is a factor in 40% cases
F. MVCs are the most common cause in teenagers and young adults whereas falls are the most common in the extremes of ages
G. Elderly patients carry worse prognosis
H. Survivors are usually disabled and constitute a burden to their families and society
I. $100 billion/yr are expended in medical care

II. Guidelines

A. No standardization of treatment before 1995
B. Neurosurgery Guidelines Committee practice parameters (1996)[2]
C. Revised Guidelines (2000)[3]
D. Unfortunately, the guidelines have only 3 Class 1 recommendations (all 3 showing ineffectiveness of long-standing practices) and only 8 guidelines based on Class II evidence

III. Initial Assessment

A. As with any trauma patient, evaluation of airway, breathing and circulation takes precedence
B. The severity of the TBI is classified clinically by Glasgow Coma Score (GCS) - seeTable 35.1
   1. Mild 13-15
   2. Moderate 9-12
   3. Severe <8 (Mortality of 33%)
C. Careful assessment of the intoxicated patient is most important to prevent missing injuries

IV. Primary neurological injury

The primary brain injury sustained at the time of the trauma cannot be reversed. A variety of injuries can occur and will be briefly described

A. Skull fractures (Fxs)
   1. Located at cranial vault or skull base.
   2. Could be linear or stellate, depressed or not depressed
   3. Indicate that a large amount of force was applied to the patient's head
   4. Linear vault fractures are usually associated with intracranial hematomas (ICH)
   5. Basilar Fxs clinical signs: hemotympanum, Battle's sign, periorbital ecchymoses, CN palsies
B. Epidural hematomas
   1. Occur in less than 1% of all head traumas and less than 10% of comatose patients have epidural hematomas
   2. Located outside the dura and typically biconvex shape on CT scan
   3. Temporoparietal location frequent due to rupture of middle meningeal artery
   4. Initial loss of consciousness may be followed by lucent period and then late neurological deterioration
   5. Prompt intervention leads to favorable outcome
C. Subdural hematomas

1. Incidence of 30% in severe TBI
2. Results form the tearing of a bridging vein between the cerebral cortex and draining venous sinus
3. In 80% of SDH, the outcome depends on the underlying brain injury
4. CT Scan: Crescent shape blood collection with associated parenchymal contusion and midline shift

D. Intracerebral hematomas
1. Associated with moderate or severe injury
2. Frontal and temporal location most frequent
3. Produce mass effect
4. Hyperdense areas on the CT scan
5. May be a delayed injury appearing 24 hrs after insult. Late neurological deterioration mandates immediate CT scan

E. Diffuse axonal injury (DAI)
1. Shearing forces affect axons that transverse large areas of the brainstem leading to Reticular Activating System dysfunction
2. Axons are not torn at injury but suffer from sequential focal changes leading to swelling and disconnection[1]
   a) Axolemma damage allows $CA^{+2}$ influx triggering local intraxonal cytoskeletal and mitochondrial damage
   b) Presence of intra-axonal caspase 3 suggests apoptosis
   c) Disconnection downstream: disconnected fibers degenerate leading to diffuse deafferentation of target sites
3. Affected patients have a high morbidity and mortality
   a) DAI can cause immediate and prolonged unconsciousness.
   b) Persistent vegetative state is a common outcome

F. Brain edema
1. Clearly associated with acute injury
2. May worsen with surgery
3. Results in vasocongestion and mass effect

V. Management of Primary Injury
A. Rapid identification of injury by imaging and neurosurgical consultation
B. Surgery should be performed if necessary as soon as possible
C. As soon as the dura is open the ICP=0

VI. Secondary Brain Injury
A. Injury not related to the disruptive force
B. The postraumatic loss of cerebral circulation autoregulation, cerebral vasculature vasoconstriction, systemic hypotension, intracranial hypertension and hypoxia result in further ischemia and infarction
C. Other mechanisms involve include free radical formation, lipid peroxidation as the result of spillage of intracellular contents and inappropriate neurotransmitter release (glutamate, aspartate)
D. Hyperglycemia and anemia may exacerbate injury
E. Later, seizures, infection and sepsis complicates patient outcome
F. The care of TBI patients is oriented to limit the secondary brain injury

VII. Areas of Management for prevention of secondary injury
A. Hemodynamic stability - Avoid Hypotension
1. Chestnut, et al demonstrated that hypotension was associated with dramatic deterioration of favorable outcome[4]
2. Both early and late epidodes of hypotension affect outcome
3. Arterial catheter ideal for monitoring
4. Use of vasopressors while fluid resuscitation is in process might be indicated
5. Search for associated injuries that may explain the hypotension
6. Which fluid resuscitation is ideal?
   a) Avoidance of hypotension is more important than the type of fluid. Fluid restriction in an unstable trauma patient who sustained TBI is an archaic concept

b) There are data supporting aggressive resuscitation (fluid and vasopressors) does not worsen neurological injury.[5]
c) Avoidance of hypotonic fluids is optimal but no differences between NS and LR
d) There is some evidence that hypertonic saline (7.5%) with or without dextran improves outcome.[6]
e) A cohort analysis of patients from double blind trials suggests that hypertonic solution significantly improves outcome. [7]
f) Recent publication comparing hypertonic saline with LR/NS showed identical neurological function at 6 months[18]
g) If ongoing blood loss is an issue, blood might be the ideal fluid
h) Glucose containing fluids should be avoided.
i) Recent data suggest that the use of albumin in these patients increases mortality.

B. Avoidance of hypoxemia.
1. 50% of patients are hypoxic in the field. This finding is associated with increased mortality
2. Hypotension and hypoxemia are not a good mix
3. There are multiple causes of hypoxemia:
a) Airway control may be difficult
b) Concomitant thoracic injuries (pneumothorax, pulmonary contusion)
c) Trauma patients have high incidence of aspiration
d) Neurogenic pulmonary edema
e) ARDS
f) **Airway management**
(1) All patient with a GCS less than 8 must be intubated for airway protection. Hyperventilation will be discussed below.
(2) Also patients with multiple trauma and those requiring multiple diagnostic procedures should be intubated
(3) Difficulties: Elevated ICP, full stomach, C-Spine injury, airway trauma, uncertain volume status, uncooperative/combative patient, hypoxemia
(4) Rapid sequence with in-line axial stabilization
(5) Consider using Etomidate to prevent hemodynamic complications
(6) Lidocaine IV may decrease the sympathetic response and the associated ICP increase
(7) Succinylcholine is safe to use despite mild and transient increases in ICP
(8) Awake nasal intubation: Base of skull fractures may result in an intracranial intubation
(9) A note about C-spine injuries
(a) Incidence of C-spine injury is 1-3% in adults and 0.5% in children
(b) Head-first falls and high speed MVC have 10% greater risk of C-Spine injury
(c) Cross-table X-Rays may miss 20% fractures and with the addition of AP and odontoid views "only" 7%" are missed.
(10) If Mechanical Ventilation is initiated:
(a) Judicious use of PEEP
(b) Titrate $FiO_2$ to a saturation above 95% to avoid deleterious effect of oxygen therapy

C. Management of cerebral circulation
1. Cerebral circulation physiology after TBI is characterized by:
a) Reduced CBF
(1) In a series of 106 patients with TBI within 6 hrs, 1/3 had CBF<18mlx100g$^{-1}$xmin$^{-1}$ (ischemic threshold)
(2) The cerebral Arterio-Venous difference is high in the first hrs and then progressively decreases
b) Impaired cerebral pressure auto regulation
(1) In 1/3 patients CBF passively changed as CPP changed
(2) In an animal model, CBF was poor with hemorrhagic hypotension but improved with phenylephrine infusion
c) Increased ICP
2. Maintenance of Cerebral Blood flow
a) Goal: Maintain CBF=Preserve CPP

b) CPP=MAP-ICP
c) CPP> 60-70 mm Hg is ideal
d) Traditional treatment of TBI has focused in ICP reduction

3. Reduction of ICP
   a) ICP Monitoring
      (1) In cases of severe TBI, measurement of ICP is essential to monitor CPP
      (2) An ICP above 20 is considered high.
      (3) There are different methods to measure ICP and all of them have advantages or disadvantages
         (a) ***Ventriculostomy:*** Allows CSF drainage if ICP is high but it is difficult to place and carries a higher risk of infection
         (b) ***Intraparenchymal monitor:*** Easy to place, low risk of injury/infection but questionable measurement of global ICP
         (c) ***Subarachnoid Bolt***: Easy to place, low risk of injury/infection, but frequent occlusion with brain tissue
   b) How to lower ICP
      (1) Therapies to decrease ICP are added in an order that reflects the risk of complications associated with each therapy.
      (2) First line of treatment:
         (a) Intubation
         (b) Positioning: 30 degrees of head up elevation, head in neutral position to avoid venous congestion
         (c) Sedation/Paralysis to avoid valsalva maneuver
         (d) Mild Hyperventilation: see note below
      (3) Second line of treatment
         (a) CSF drainage: only possible with ventriculostomy drains
         (b) Osmotic therapy: Mannitol (0.25 -1 g/kg and titrated to specific measured osmolality or osmolar gap [measured – calculated osmolality]
      (4) Third line of treatment
         (a) Pentobarbital coma
         (b) Surgical decompression
      (5) The use of chronic and profound (25 mmHg) hyperventilation is **discouraged.**[2] Hyperventilation may decrease CBF and reduce brain oxygenation. Skippen et al using xeno-enhaced CT and CBF studies demonstrated a 2.5 fold increase in the number of regions of brain ischemia in children with TBI who were hyperventilated.[8] Muizellar et al published a prospective randomized study that demonstrated worse neurological outcome in patients that were hyperventilated. [9] A $PaCO_2$ between 30-35 seems appropriate. It should also be noted that the effects of hyperventilation are self limited and disappeared after 8-12 hours. However, it could be used in a critical situation as a lifesaving maneuver.
4. Novel theories of CPP preservation[10]
   a) Different strategies for the management of cerebral circulation during TBI have been recently advocated. They have not proved to improve outcome after TBI when compared with the traditional approach described above.
      (1) The "CPP management", has been advocated by Rosner et al.[11] According to this hypothesis, a reduction in CPP—either a decrease in MAP, an increase in ICP, or both—stimulates the cerebral vessels to dilate in an attempt to maintain CBF. This is the normal pressure autoregulatory response to a decrease in CPP. Because the increase in cerebral blood volume that accompanies the vasodilation further reduces CPP by increasing ICP, this sets up a cycle that leads to reducing CPP even more. An increase in arterial blood pressure under this circumstance has been observed to break the cycle and reduce ICP. A detailed description of this approach is given in a recent report of a clinical series. In this series of 158 patients admitted with a Glasgow Coma Scale score less than 7, mortality was only 29%, and 59% achieved a good recovery or moderate disability by 6 months postinjury. This approach was believed to be of sufficient value that it was included in the 1996 and 2000 Head Injury Guidelines as a treatment option.

(2) The "Lund" [12] therapy emphasizes reduction in microvascular pressures to minimize edema formation in the brain. The goals of this approach are to preserve a normal colloid osmotic pressure (infusion of albumin and erythrocytes), to reduce capillary hydrostatic pressures by reducing systemic blood pressures (metoprolol and clonidine), and to reduce cerebral blood volume by vasoconstricting precapillary resistance vessels (low-dose thiopental and dihydroergotamine). Treatments that would favor increasing transcapillary filtration of fluid are avoided, including cerebrospinal fluid drainage, high-dose (to burst suppression) barbiturates, osmotic diuretics, and high CPP. Decompressive craniectomy, which can also increase edema formation, is reserved as a last resort

D. Monitor brain oxygenation
   1. Measurement of the oxygen content indirectly by jugular venous oxygen saturation ($SjvO_2$) or directly by brain tissue $PO_2$ has shown to be good indicators of an injury that is worsening
   2. Jugular venous oxygen saturation
      a) Utilizes "mixed" cerebral blood that may reflect ischemia
      b) There is evidence that a decrease in ($SjVO_2$) is associated with worse outcome.[13]
      c) Limitations
         (1) Invasive
         (2) Possible malposition
         (3) Unilateral
         (4) Global (not focal) ischemia detected
         (5) Anemia may cause false negative
   3. Brain Tissue $PO_2$ provides evidence of ischemia that also correlates with neurological outcome and reflect changes in CPP
E. Neuroprotective agents/Seizure prophylaxis
   1. Phenytoin or carbamazepine are useful to prevent early postraumatic seizures (<7 days)
      a) No evidence for chronic use of anticonvulsants to prevent seizures[2]
   2. As free radicals are involved in secondary injury, use of free radical scavengers may be beneficial (i.e. Mannitol)
   3. There is no benefit in administering steroids.[2]
      a) These drugs may alter nutrition and metabolic profile (Hyperglycemia)
F. Cardiovascular system
   1. Massive catecholamine response and aggressive fluid resuscitation may unmask or worsen cardiac disease
   2. B-Blockers (labetalol/esmolol) may be used to control hypertension and tachycardia
   3. Arrhythmias are not uncommon and may be unrelated to the heart ("Brain arrhythmias")
   4. Severe ICP precipitates reflex hypertension and bradycardia (Cushing's triad)
   5. Venous thrombosis prophylaxis
      a) Anticoagulants may be contraindicated
      b) Venous compressive devices
      c) IVC filters
G. Nutrition/ Gastrointestinal
   1. TBI results in a hypermetabolic and catabolic state
   2. Caloric replacement should be at least 140% (non-paralyzed patients) and 100% (paralyzed patients) of RME by day 7. Start feedings by 72 hr
   3. Enteral feeding is the preferred route
   4. Stress gastritis protection should be provided to patients requiring ventilatory support and who are not being enterally fed. Double coverage is the norm
H. Endocrine and metabolic
   1. Avoid hyperglycemia[14]
      a) Brain glucose concentrations parallel blood levels
      b) Hypoxia or anoxia results in anaerobic metabolism and production of lactic acid
      c) The associated $H^+$ produce cellular damage by:
      d) Altering neurotransmitter release and uptake
      e) Alteration of ion homeostasis
      f) Changes in protein synthesis and activity
      g) Hyperglycemia increases mortality in the ICU[14]
      h) Avoid glucose containing fluids for resuscitation

i) Consider insulin infusion if hyperglycemia is a problem

2. Electrolyte abnormalities
   a) SIADH: Hyponatremia, UNA>30mEq/lt., Uosm>300mOsm/kg
   b) Diabetes insipidus: Hypernatremia, Uosm<290mOsm/kg
   c) Cerebral "Salt wasting syndrome": Intravascular volume contraction and negative NA+ balance

I. Hematological
   1. Coagulopathies and DIC may be present due to liberation of large amounts of brain thromboplastin
   2. Hypothermia, massive blood transfusion are also possible causes of coagulopathy in any trauma patient
   3. Monitoring of coagulation parameters is very important intraoperatively

J. Temperature
   1. Passive cooling to mild hypothermia (to about 34-35° C) decreases cerebral metabolic rate (CMR).
   2. A 7% reduction in CMR per degree Celsius decrease has been described.
   3. Reductions of CMR result in decreases in oxygen consumption and therefore hypothermia is thought to be protective of the brain. Two large studies looked into this hypothesis.
   4. Treatment of Traumatic Brain Injury with Moderate Hypothermia. Marion et al [16] Treatment with moderate hypothermia for 24 hours in patients with severe traumatic brain injury and GCS of 5 to 7 on admission hastened neurologic recovery and may have improved the outcome.
   5. Lack of Effect of Induction of Hypothermia after Acute Brain Injury Clifton, et al.[17] Treatment with hypothermia, with the body temperature reaching 33°C within eight hours after injury, is not effective in improving outcomes in patients with severe brain injury. However patients that were hypothermic upon arrival had a better outcome.
   6. It seems that hypothermia has a protective effect if it occurs at the time of the primary injury. Severe hypothermia should be avoided due its deleterious systemic effects.

*Table 35.1. Glasgow Coma Scale*

| Signs | Score |
|---|---|
| Eye opening | |
| Spontaneous | 4 |
| To verbal Command | 3 |
| To pain | 2 |
| No response | 1 |
| Best motor response | |
| Obeys verbal commands | 6 |
| Localizes pain | 5 |
| Withdraws to pain | 4 |
| Flexion response to pain (decorticate) | 3 |
| Extension response to pain (decerebrate) | 2 |
| No response | 1 |
| Best verbal response | |
| Oriented | 5 |
| Confused | 4 |
| Inappropriate words | 3 |
| Nonspecific sounds | 2 |
| No response | 1 |

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## QUESTIONS:

35.1. The CORRECT statement regarding the fluid resuscitation in the patient with traumatic brain injury is:

A. Crystalloids are better than colloids
B. Hypertonic saline is clearly superior
C. Vasopressors are clearly contraindicated
D. There is no difference in outcome at 6 months in relation to the type of crystalloid fluid

35.2. Current concepts in relation to the use of hyperventilation in the TBI patient include the following EXCEPT:
- A. Prophylactic hypercarbia is contraindicated
- B. Hypercarbia might worsen outcome
- C. Ischemia might result from the use of hypercarbia
- D. A $PaCO_2$ of 25mmHG is indicated in most TBI patients

35.3. Airway management in the TBI patient might be complicated by the following EXCEPT:
- A. Hypoxemia
- B. Unknown neck injuries
- C. Unknown intravascular volume
- D. Non combative or non intoxicated patient

35.4. Prevention of secondary injury in the TBI patient includes the following EXCEPT:
- A. CPP management
- B. Prevention of hypoxemia
- C. Immediate surgical decompression
- D. Prevention of hyperglycemia

35.5. None of the following are accepted means to care for the TBI victim EXCEPT:
- A. Aggressive hypocarbia
- B. Provide prophylactic anti-seizure drugs for 7 days
- C. Use steroids to minimize brain swelling
- D. Delay enteral feeding for 5-7 days

# 36. Management of Increased Intracranial Pressure

Jonathan R. Jagid, M.D., Matthew M. Ruel, M.D., Leo Harris, P.A, M.P.H., Miguel Cobas, M.D.

*A 32-year-old male is brought to the ED after being found unresponsive at the scene after an MVA. EMS reports that he was an unrestrained driver and was ejected from the vehicle. The patient arrives intubated, his vital signs are: HR = 52, BP = 168/95, $SpO_2$ = 100%. Injuries include multiple facial fractures, and head CT reveals a bilateral frontal contusion with diffuse brain edema. Chest and C-spine x-rays are unremarkable, and CT of abdomen/pelvis are negative for trauma related injuries. On exam, the patient is nonfocal and demonstrates decerebrate posturing to deep pain.*

I. Key Concepts / Definitions

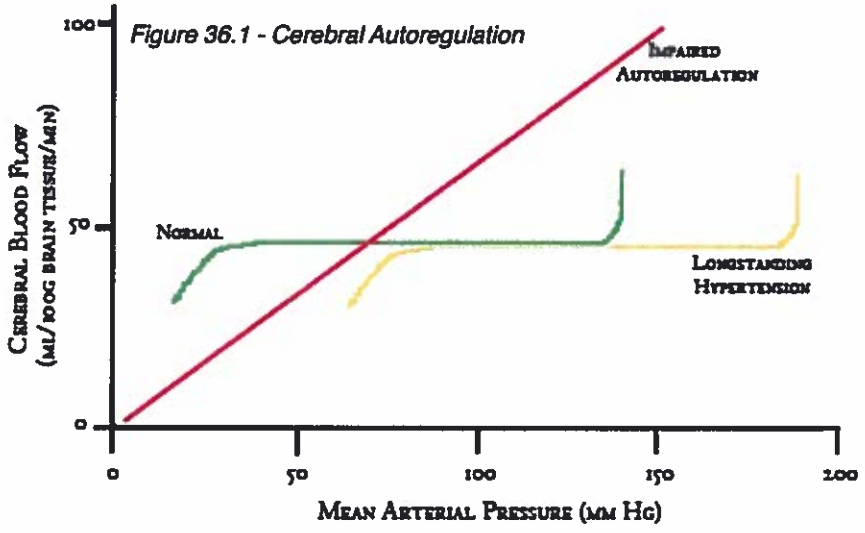
A. Anatomical/Physiological concepts unique to the brain

1. The brain is enclosed in a confined compartment, the cranium.
   (1) Monroe-Kellie Doctrine states that the cranium is a rigid compartment.
      (a) Three non-compressible components:
         (1) Brain Parenchyma 80%
         (2) Cerebral Spinal Fluid 10%
         (3) Intracranial Blood 10%
2. Any increase in volume of any of these three components, or the addition of a mass, past a threshold leads to an increase in intracranial pressure.
3. Any increase above and beyond this threshold will result in a disproportionate increase in pressure (ICP) with respect to the increase in volume. Even minute volumes (1-2 mls) can cause an exponential rise in ICP's. This is considered to be life threatening and potentially leading to herniation syndrome or "herniation."
4. The Monroe-Kellie Doctrine is the strategic foundation for the management of increased ICP's

B. Cerebral autoregulation (see Figure 36.1)

1. The ability of the brain to maintain a constant critical level of cerebral blood flow (CBF) {45-50ml /100g/min , 20ml/100g/ min in white matter to 70ml/100g/min in grey matter}over a wide range of mean arterial pressures (MAP) {60-150mmHg} in a sigmoidal pattern.
2. In the severely injured brain, there is malfunction of autoregulation termed disautoregulation. The autoregulatatory mechanism does not function and the relationship is converted to a linear pattern.

*Figure 36.1 - Cerebral Autoregulation*




3.Exact physiological mechanism are not well understood, studies suggest that the cerebral vascular bed exhibits an intrinsic myogenic regulation of vascular tone which plays a role.

4.Autoregulation is disrupted by extremes in MAP, traumatic brain injury; severe metabolic disturbances, disruption of blood brain barrier; severe hypercarbia and/or hypoxemia (see figure 36.2).

5.Chronic hypertension shifts the autoregulatory curve to the right, increasing the risk of

ischemia with relative hypotension that may fall within the normal acceptable range (MAP 60-150mmHg).

6. Disautoregulation places the onus upon the intensivist and or anesthetist to reestablish cerebral blood flow by appropriate and aggressive management of the mean arterial pressure.

C. Elastance, Compliance and the Intracranial Pressure-Volume curve
   1. The curve is a graphical representation of the nature of intracranial elastance, or the change in pressure for a given change in volume (delta P/ delta V).
   2. The concept of elastance needs to be distinguished from compliance, which is change in volume per unit pressure (think of chest wall/pleura/lung model). In the lung model we are interested in volume change per unit pressure, whereas in the cranium the change in pressure per unit volume is more relevant.
   3. An easy way to visualize these two reciprocal concepts is via a balloon analogy. Compliance relates to how easily a balloon expands when pressure is introduced, whereas elastance refers to the force the balloon exerts against a specific volume when filled.
   4. The flat portion of the curve illustrates low-elastance (high compliance), a state where incremental changes in volume are accompanied by relatively small increases in pressure. This low elastance state reflects shunting of CSF and blood to extracranial locations down a developing pressure gradient.
   5. The steeper portion of the curve is reached once this shunting mechanism is overcome and small increases in volume lead to large increases in ICP. Clinically, his high-elastance (low compliance) state can rapidly lead to catastrophic cerebral ischemia and herniation if not treated expeditiously.

D. Cerebral Perfusion Pressure (CPP).
   1. An indirect measurement of cerebral blood flow (CBF)
   2. Cerebral Perfusion Pressure = Mean Arterial Pressure- Intracranial Pressure
   3. Studies indicate that CPP should be kept above 70mmHg to ensure adequate cerebral perfusion.
   4. Recent studies have shown that if a patient's CMRO is decreased (paralytics/sedation/ barbiturates) CPP's may be allowed to drift to below 70mmHg but no less than 60mmHg.

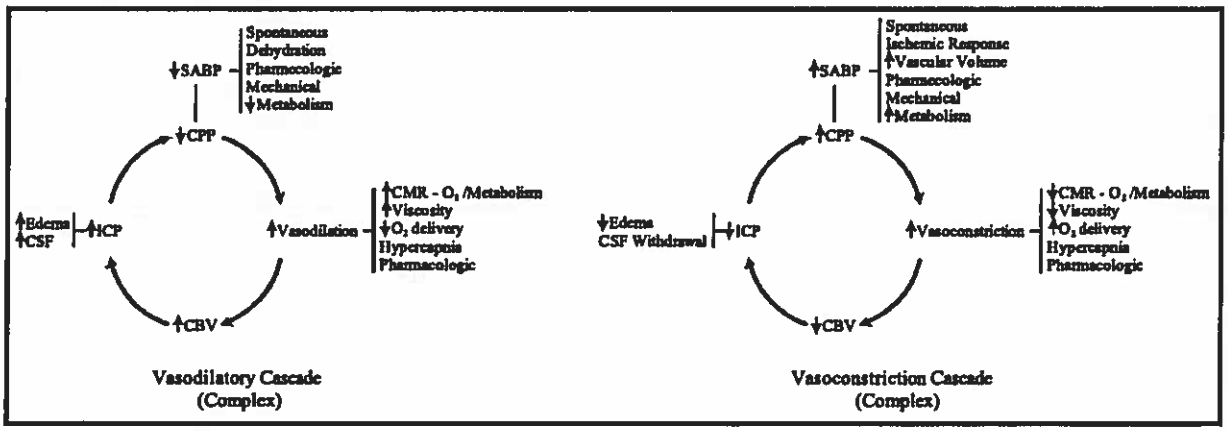



*Figure 36. 2. The proposed vasodilatory and vasoconstriction cascades model:*
*As CPP increases, cerebral vasoconstriction limits CBV and ICP. Conversely, a reduction in CPP may stimulate cerebral autoregulatory vasodilation with an increase in CBV and ICP. (Reproduced with permission from: "Rosner MJ, Rosner SD, Johnson AH: Cerebral perfusion pressure: management protocol and clinical results. J Neurosurg 83:949-962, 1995).*

II. Etiology of intracranial hypertension
   A. As stated in the Monroe-Kellie doctrine, described above, an increase in volume of any of the

three intracranial components, or the addition of a mass occupying lesion (tumor, epidural/subdural hematoma), will lead to increased intracranial pressures.

B. Increased CSF volume occurs via decreased absorption (compression of foramen of Monroe, sinus injury) or increased production.
C. Increased cerebral blood volume (CBV) may occur via vasodilation or intracranial hemorrhage
D. Increased brain tissue volume can occur in the context of enlarging neoplastic lesions, expanding intracerebral/epidural/subdural hematoma or edema.

III. Indications for ICP monitoring
A. Severe Brain Injury or Glasgow coma score ≤ 8 (after resuscitation) and abnormal CT
B. Severe Brain Injury or Glasgow coma score ≤ 8 (after resuscitation) and normal CT with ≥2 of the following:
1. age 40
2. SBP 90mmHg
3. decerbrate on motor exam
4. decorticate posturing on motor exam

IV. Methods of monitoring ICP
Continuous ICP monitoring in an ICU setting allows optimization of CPP in critically ill patients with intracranial hypertension. In present TBI practice, the intraparenchymal and ventricular catheters are considered the standard of care. A normal value for ICP in an adult is 8-15mmHg, and 3-7mmHg in pediatric patients.
A. Intraventricular (ventriculostomy) catheter, fluid coupled system
1. Gold standard for ICP monitoring
2. Allows for continuous ICP monitoring, therapeutic/diagnostic CSF drainage.
3. Risk of infection, tissue damage during placement, hematoma formation.
4. Placement can be technically challenging, as insertion is a "blind" procedure.
5. Antibiotic impregnated catheters have significantly lowered the infection risk
B. Intraparenchymal monitors
1. Directly measure brain tissue pressure using fiberoptic or strain gauge sensors.
2. Placed in cortical gray matter, risk of tissue damage during placement.
3. Solid-state technology, no tubing to kink or clog
4. Cannot drain CSF, cannot be recalibrated once placed in brain.
5. Acceptable drift that increases each day
6. Used in scenario of diffuse edema where direct ventricular access is difficult
7. Lower infection rate
8. Requires frequent re-zeroing
C. Subdural bolt
1. Hollow screw threaded into skull with tip passing into subdural space.
2. No parenchymal penetration may be placed in any location which avoids venous sinuses.
3. Cannot relieve ICP by draining CSF, infection risk similar to ventriculostomy catheter.
D. Epidural transducer
1. Places pressure-sensitive membrane in contact with dura.
2. Decreased risk of infection since no dural puncture.
3. Cannot therapeutically drain CSF
4. Placement in epidural space difficult, risk of hematoma due to venous plexus.

V. Pathophysiology
A. Ischemia is defined as a localized insufficiency of blood flow, delivering oxygen and nutrients or a mismatch of tissue demands versus blood delivery
B. The brain is the organ most sensitive to ischemic insult.
C. Ischemia results in damage to neurons as a result of sustained intracranial hypertension and is the pathopysiological mechanism by which cell death ensues.
D. Cellular metabolic consequences of ischemia
1. Sustained ischemia compromises ATP production by disrupting oxidative phosphorylation.
2. ATP deficit causes shut-down of ATP dependent ion pumps, leading to increased intracellular sodium and calcium and decreased potassium.
3. Membrane depolarization occurs which in turn causes release of excitatory amino acids

(glutamate) and oxygen free radicals.
4. This cascade of events leads to cellular necrosis and brain edema, which in turn increases ICP further and sets up a catastrophic clinical course if not managed expeditiously.

E. Precipitating factors for ischemia also know as secondary brain injury
1. Hypotension
2. Hypoxia
3. Hyperthermia
4. High ICP's

F. Hypotension and Hypoxia are two most common reasons for ischemia

G. Ischemia is not a singular event but rather cumulative, progressive event

H. Without aggressive and rapid reduction in ICP ultimately brain herniation and death will ensue.

VI. Rationale and Strategies:

A. MAINSTAY OF ICP MANAGEMENT IS PREVENTION OF ISCHEMIA
1. Maintain CBF
a) Difficult to monitor real time
(1) limited practical/reliable technology (Blood flow monitors, Xenon scans)
(2) CPP acceptable real time measure of cerebral blood flow
(a) CPP cannot be measured directly
(b) CPP has direct relationship to CBF
(c) CPP is PARAMOUNT when monitoring increased ICP's
(d) CPP = MAP – ICP
b) Arterial line, central venous access and ICP monitor are strongly urged in the moderate/severe brain injury patient.
(1) Normal MAP – 90-100mmHg
(2) NEVER lower MAP artificially in face of increased ICP's
(3) Normal ICP's
(a) Adult 8-15mmHg
(b) Pediatric 3-7mmHg
(4) Maintanence of CPP's
(a) ≥70mmHg
(b) ≥60mmHg if $CMRO^2$ (Cerebral Metabolic Rate of Oxygen Consumption) decreased with
i) Hypothermia
ii) Sedation/Paralytic
iii) Barbiturate
(5) The NeuroIntensivist and the Neurosurgeon should always devise a plan *before* initiating management of high ICP's

VII. Therapeutic Interventions

A. Positioning of patient
1. Head of Bed 30-45°
a) Promotes venous outflow of intracranial drainage (CBV) in periphery, promotes CSF flow from intracranial vault to spinal vault, ↓ ICP's
2. Neck in midline so as to not occlude carotid/jugulars vessels
3. Care must be taken to ensure MAP is maintained when in head up position.

B. Intravascular Solution
1. AVOID HYPOTONIC SOLUTIONS IN TBI patients.
2. Ideal state is EUVOLEMIA (maintain SBP) with HYPERTONICITY (↓ edema)
3. Normal Saline most frequent solution
4. Hypernatremic solutions are gaining popularity (3%-23.4%)
a) Rapid volume expansion
b) Increase tonicity of blood decreasing edema

C. Diuretics
1. Mannitol 20% (0.25-1g/kg IV) is an osmotic diuretic.
a) Should be initiated as soon as possible after neurosurgical evaluation
(1) Rapid plasma expander by increasing tonicity of blood
(a) hematocrit and viscosity improving CBF and Oxygen delivery

(b) Increased tonicity draws edema from brain tissue
(2) 15-30 minutes to take effect.
(3) Lasts 1.5-6 hours
(4) Supports microcirculation by rheology properties
(5) Therapeutic range in TBI by keeping serum 300-320 mOsm/L
(6) Bolus and Maintenance doses differ,
(a) Load is 1gm/kg, maintenance 0.5 gm/kg
(b) Frequency is usually Q4°
(7) Serum osmolarity and plasma sodium concentration should be monitored Q4, staggered with Mannitol infusion to allow for dosing changes. Renal toxicity is common with serum osmolarity greater than 320, and hypernatremia is an acceptable side effect.
(8) Must continue IVF, enteric feeding alone is not a substitute when utilizing hyperosmolar therapy
(9) Frequently the blood brain barrier is violated in brain injury patients, making it essential to maintain mannitol within the therapeutic range. If mannitol is allowed to drift to subtheraptic levels it may worsen ICP by drawing water into the cranial vault.
(10) Mannitol should be weaned to prevent rebound intracranial hypertension

2. Furosemide (0.5-1mg/kg IV alone; 0.15-0.3mg/kg in comb. with mannitol) acts at the level of the glomerulus (loop diuretic).
a) Decreases CSF production.
b) Not as effective in emergent setting as mannitol
c) Synergistic effect with Mannitol
d) Necessary to follow electrolytes closely, hypocalcemia and hypokalemia common.
e) Hold if Serum Osm ≥ 320 to prevent hyperosmolar ketoacidosis

D. Hyperventilation- $PaCO_2$ is the most potent determinant of CBF.
1. PROLONGED HYPERVENTILATION IS CONTRAINDICATED
2. HYPERCAPNIA ($PaCO_2$ ≥46) AND severe HYPOCAPNIA (≤30) SHOULD ALWAYS BE AVOIDED
3. Vasocontriction of intracranial vessels ↓CBF and CBV
4. Hyperventilation therapy is effective for acute ICP control only. After 12-24 hours compensatory metabolic mechanisms lead to decreased bicarbonate in the CSF, normalization of pH, and resolution of induced vasoconstriction.
5. For every mmHg change in PaC02, CBF changes 1-2ml/100g/min.
6. Target $PaCO_2$ 35mmHg, at partial pressures less than 25 the $O_2$-Hb dissociation curve shifts to the left and $O_2$ delivery may be compromised.
7. Hypoxemia must be avoided, at $PaO_2$ less than 50 mmHg CBF and therefore ICP will increase.
8. Be aware that PEEP may decrease venous outflow from cranial vault via increased intrathoracic pressures.
9. CBF varies directly with $PaCO_2$ over a range of 20-80mmHg.

E. CSF Diversion
1. Very Effective
2. Immediate result
3. Small volumes (5-10cc/hr) for ICP's sustained for ≥5 minutes
4. Large volumes (>10cc/hr) may result in new or increase in extra axial collections

F. Corticosteroids
1. STEROIDS ARE AN ABSOLUTE CONTRAINDICATION IN THE SEVERE BRAIN INJURY PATIENT
2. Dexamethasone requires 12-36 hours to take effect, has not been shown to be effective in the acute setting.
3. Appropriate in the treatment of vasogenic edema (tumors, abscesses) steroids have been shown to decrease vasogenic edema formation around neoplasms.
4. Edema of TBI is frequently a combination of cytotoxic, vasogenic, and interstitial
5. May help repair/stabilize disrupted blood brain barrier via exocytosis.
6. Must weigh use against risk profile in non TBI patients: hyperglycemia, GI bleed, electrolyte disturbances, increased infection risk.

G. Surgical decompression

1. Should be reserved for patients with intractable ICP's.
2. Involves removal of a portion of the skull +/- resection mass occupying lesion.
3. Studies show promise in early decompression
4. Decompressions becoming larger in size with advances in technology

H. Hypothermia
1. Studies inconclusive but subgroups have shown some benefit
2. Investigations ongoing
3. Leads to decreased CMR02 and resultant cerebral vasoconstriction
4. Target temperature should be between 33 and 35 degrees C, significant side effects with Temperatures < 33°C.

I. Barbiturate/Sedative therapy
1. Barbiturates not commonly utilized today
   a) Multi-system side effects
   b) Usually require PA catheter
2. Sedatives-Benzodiazepines and propofol
   a) Useful in lowering ICP's
   b) Lowers CMRO
   c) Shorter half life preferred to preserve rapid neurological exam
3. Paralytics
   a) Lowers CMRO
   b) Should never be used without sedative

Table 36.1: Tiered Management of Intracranial Hypertension

| Treatment | Effect | Comments |
|---|---|---|
| Head elevation | • Decreased CBV and capillary leak<br>• CSF to lumbar cistern | • Need to maintain MAP with head up |
| CSF drainage | •Decreased CSF volume | •Infection, formation of extraxial collections from overdrainage |
| Osmotherapy | • Decreased brain tissue water and CSF formation<br>• Decreased blood viscosity → decreased CBV | • Hypotension and hypovolemia<br>• Electrolyte disturbances and possibly renal failure<br>*Mannitol 20% agent of choice |
| Mild Hyperventilation ($pCO_2$ 30-35) | • Decreased CBV and capillary leak<br>• Improved CSF outflow | • No role for prolonged hyperventilation (PaCO2 ≤ 30mmHg)*<br>Decreases CBF and may exacerbate cerebral ischemia. |
| Hypothermia | • Decreased brain $CMRO_2$<br>• Cerebral vasoconstriction | • Untoward systemic side effects<br>• Less effective in GCS 3 and 4 |
| Barbiturates | Decreased $CMRO_2$ → decreased CBV<br>• Decreased capillary leak | • Immunosuppression; needs hemodynamic support and monitoring |
| Operative decompression | • Decreased brain tissue volume | • Brain tissue loss and damage |
| Steroids | Endothelial cell membrane stabilization<br>• Decreased release of cytokines | • No role in TBI *<br>• Hyperglycemia, GI bleeding<br>• Immunosuppression |

* Brain Trauma Foundation Standards

This chapter is a revision of the original chapter authored by Ahmed E. Badr, M.D., M.Sc.

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## QUESTIONS:

36.1. Desirable goals for management of acute severe head injury include all EXCEPT:
- A. Maintenance of CPP >70 mmHg
- B. Initiation of ICP treatment at an upper threshold of 20-25 mmHg
- C. Prophylactic hyperventilation to a PaCO2 of ≤35 mmHg
- D. Maintenance of adequate oxygenation with a PaO2 >60 mmHg
- E. Maintenance of euvolemia

36.2. Appropriate indications for ICP monitoring include all EXCEPT:
- A. Patients with mild to moderate head injury
- B. Patients with severe head injury (GCS of 3-8) and abnormal head CT scans
- C. Patients with severe head injury and normal head CT scans if unilateral or bilateral motor posturing is present
- D. Patients in whom barbiturate coma is used to control ICP
- E. Patients with severe head injury in whom sedation, analgesia, and paralysis are instituted

36.3. The most common electrolyte abnormality encountered with Mannitol is:
- A. Hyponatremia
- B. Hypokalemia
- C. Hypermagnesemia
- D. Hypernatremia

36.4. Upon arrival to the emergency room, the blood pressure of the patient described at the beginning of the chapter should be:
- A. Not treated
- B. Raised
- C. Lowered
- D. None of the above

36.5. Cerebral Compliance is defined as:
- A. CPP/ICP
- B. $\Delta V/\Delta P$
- C. $\Delta P/\Delta V$
- D. MAP/CPP
- E. None of the above

36.6. All of the following treatment strategies may be employed to treat intracranial hypertension EXCEPT:
- A. Mild hyperventilation
- B. Osmotic diuresis
- C. Elevation of head of bed
- D. Steroids
- E. Cerebrospinal fluid diversion

36.7. All of the following are true of Hypothermia, EXCEPT:
- A. Decreases the metabolic demand of oxygen
- B. Improves functional outcome in head injury
- C. It is associated with lowered ICP
- D. Severe systemic side effects may ensue at temperatures lower than 33°C
- E. It may be less effective in patients with GCS of 3-4

36.8. All of the following are true of Cerebral Autoregulation EXCEPT:
- A. Autoregulation is effective in the injured brain
- B. It maintains adequate cerebral blood flow over a wide range of MAP
- C. Arterial $CO_2$ is an important autoregulatory factor
- D. It is unique to the brain
- E. None of the above

36.9. Which of the following is true of mild hyperventilation:
- A. A.It should be used for long intervals of time to maintain ICP
- B. The target is a venous $CO_2$ of 28-32
- C. The target is an arterial $CO_2$ of 30-35
- D. The target is a venous $CO_2$ of 30-35
- E. It has no effect on cerebral ischemia