

Principles of Neurocritical Care

Dr. Paresh Ku. Gouda
2nd yr MCh Resident
Neurosurgery

TYPES OF BRAIN INJURY

Primary brain injury –

- DAI
- HAEMATOMA & CONTUSIONS

Secondary brain injury –

- Traumatic Brain Swelling/Intracranial Hypertension
- Hypoperfusion
- Vasospasm

PATHOPHYSIOLOGY OF TRAUMATIC BRAIN INJURY

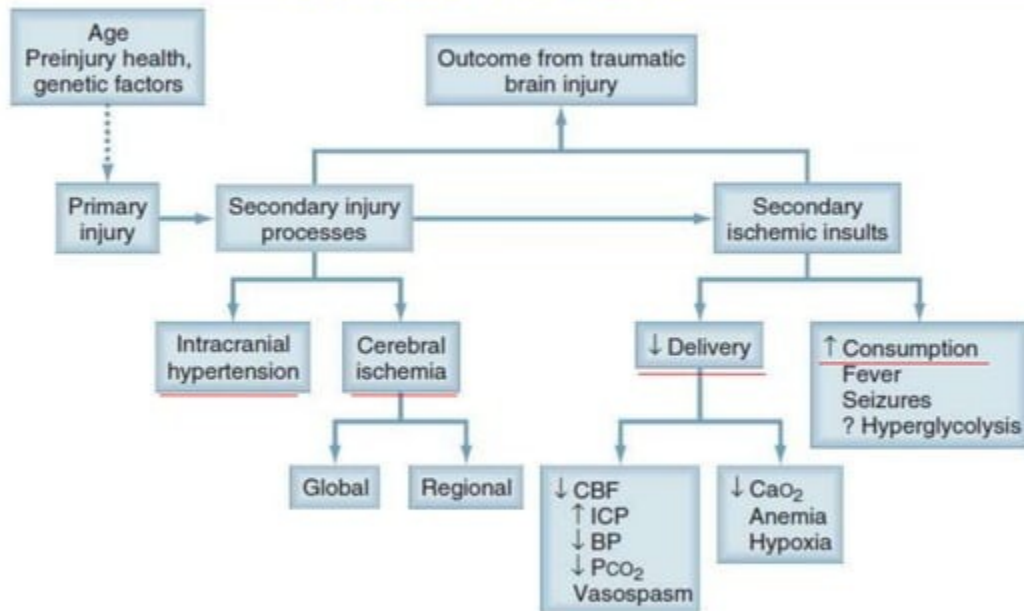


Figure 349-2. The outcome after traumatic brain injury is determined by a number of factors, including the severity and type of the primary injury, which initiates secondary injury processes and causes the injured brain to be more susceptible to secondary ischemic insults. BP, blood pressure; CaO_2 , arterial concentration of oxygen; CBF, cerebral blood flow; ICP, intracranial pressure; PCO_2 , partial pressure of carbon dioxide.

NEUROLOGICAL INTENSIVE CARE MONITORING

- The most important secondary injury processes that can be monitored, anticipated, and treated in the ICU-

INTRACRANIAL HYPERTENSION
CEREBRAL ISCHEMIA
BRAIN TISSUE HYPOXIA

NEUROMONITORING

- **MONITORING NEUROLOGICAL STATUS**

GCS,GOS, FOUR SCORE, PUPIL

- **MONITORING OF SECONDARY INJURY PROCESSES**

- INTRACRANIAL HYPERTENSION-

- 1.ICP MONITORING

- CEREBRAL ISCHAEMIA-

- 1.CEREBRAL PERFUSION PRESSURE

- **MONITORING OF SECONDARY ISCHAEMIC INSULTS**

- 1.TRANSCRANIAL DOPPLER

- 2.JUGULAR VENOUS OXYGEN SATURATION

- 3.BRAIN TISSUE PO₂

- 4.CEREBRAL MICRODIALYSIS

- **Monitoring for Cerebral Causes of Secondary Ischemic Insults**

1. INTRACRANIAL HYPERTENSION

2. SEIZURES

3. ELECTROPHYSIOLOGIC MONITORING

- **Monitoring for Systemic Causes of Secondary Insults**

1. HYPOTENSION

2. HYPOCAPNIA

3. HYPOXIA

4. ANEMIA

5. FEVER

Monitoring Neurological Status

TABLE 349-1 Glasgow Coma Scale for Assessment of Coma and Impaired Consciousness

Eye Opening	Best Motor Response	Best Verbal Response
4 = Spontaneous	6 = Obeying	5 = Oriented
3 = To speech	5 = Localizing	4 = Confused
2 = To pain	4 = Withdrawing or Normal Flexing	3 = Inappropriate
1 = None	3 = Abnormal Flexing	2 = Incomprehensible
	2 = Extending	1 = None
	1 = None	

Data from Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet*. 1974;2:81-84.

GCS SCORE

TABLE 349-3 The FOUR Score for Assessment of Coma

Eye Response	Brainstem Reflexes
4 = Eyelids opened	4 = Pupil and corneal reflexes present
3 = Eyelids open but not tracking	3 = One pupil wide and fixed
2 = Eyelids closed but opens to loud voice	2 = Pupil or corneal reflexes absent
1 = Eyelids closed but opens to pain	1 = Pupil <i>and</i> corneal reflexes absent
0 = Eyelids remain closed with pain	0 = Absent pupil, corneal, and cough reflex
Motor Response	Respiration
4 = Thumbs up, fist, or peace sign to command	4 = Not intubated, regular breathing pattern
3 = Localizing to pain	3 = Not intubated, Cheyne-Stokes breathing
2 = Flexion response to pain	2 = Not intubated, irregular breathing
1 = Extensor posturing	1 = Breathes above ventilator rate
0 = No response to pain	0 = Breathes at ventilator rate or apnea

FOUR SCORE

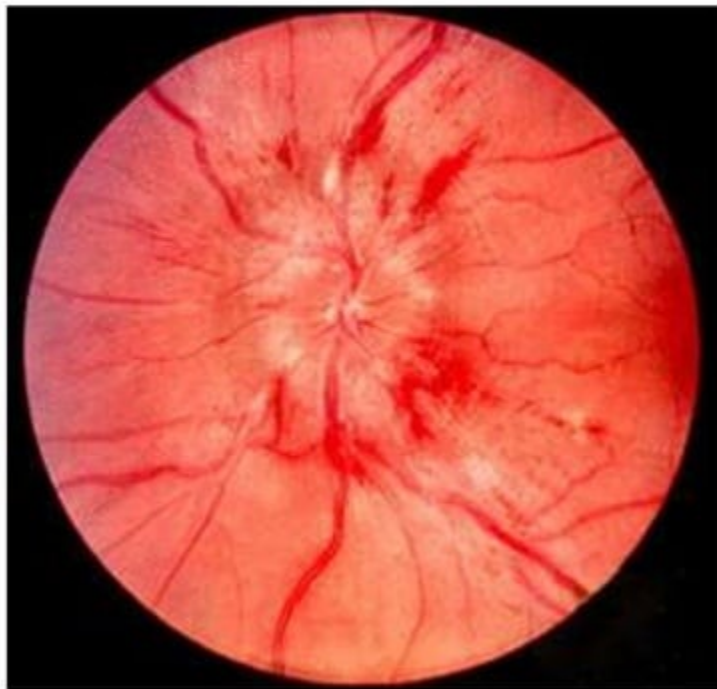
TABLE 349-2 Glasgow Outcome Scale (Original 5-Point Scale and Extended 8-Point Scale) for Assessment of Outcome

Summary	Glasgow Outcome Scale	Extended Glasgow Outcome Scale
Sleep/awake, nonsentient Conscious but dependent	1 = Dead	1 = Dead
	2 = Persistent vegetative state (PVS)	2 = Persistent vegetative state (PVS)
	3 = Severe disability (SD)	3 = Lower severe disability (Lower SD) 4 = Upper severe disability (Upper SD)
Independent but disabled	4 = Moderate disability (MD)	5 = Lower moderate disability (Lower MD) 6 = Upper moderate disability (Upper MD)
May have mild residual effects	5 = Good recovery (GR)	7 = Lower good recovery (Lower GR) 8 = Upper good recovery (Upper GR)

Data from Jennett B, Bond M. Assessment of outcome after severe brain damage. *Lancet*. 1975;1:480-484; and Wilson JT, Pettigrew LE, Teasdale GM. Structured interviews for the Glasgow Outcome Scale and the extended Glasgow Outcome Scale: guidelines for their use. *J Neurotrauma*. 1998;15:573-585.

GOS SCORE

PUPIL



ICP MONITORING

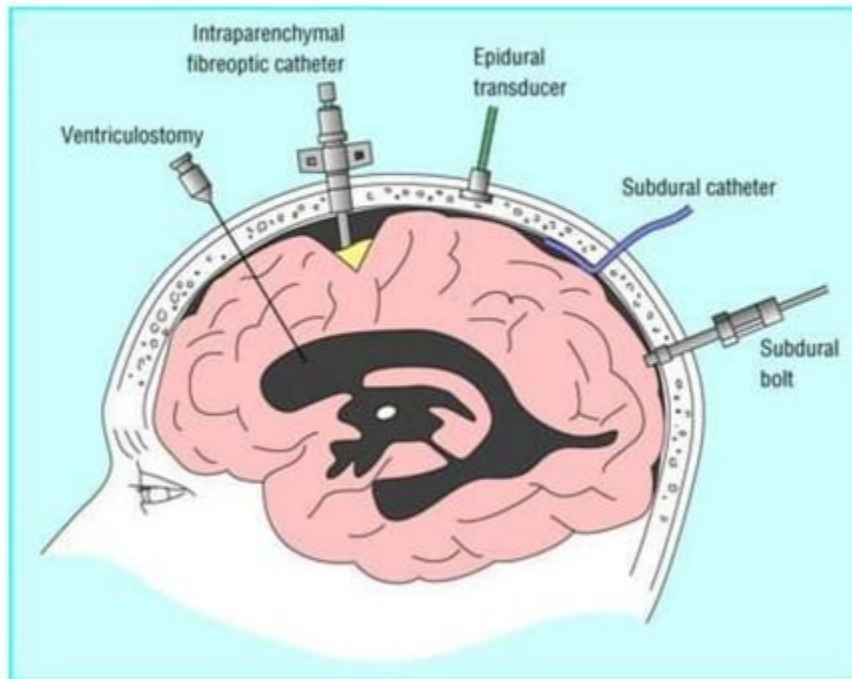
- Normally, resting ICP in adults is less than 10 mm Hg.
- A sustained ICP greater than 20 mm Hg is clearly abnormal.
- An ICP between 20 and 40 mm Hg is considered moderate intracranial hypertension.
- An ICP greater than 40 mm Hg represents severe, usually life-threatening, intracranial Hypertension.

INDICATIONS

Third Edition Guidelines for the Management
of Severe Traumatic Brain Injury, 2007

LEVEL II -Monitor ICP in all salvageable patients with a severe TBI, a GCS score of 3-8 after resuscitation, and an abnormal CT scan (with hematomas, contusions, swelling, herniation, or compressed basal cisterns).

LEVEL III-Monitor ICP in patients with severe TBI and a normal CT scan if two or more of the following features are noted at admission: age >40 yr, unilateral or bilateral motor posturing, or a systolic blood pressure <90 mm Hg.

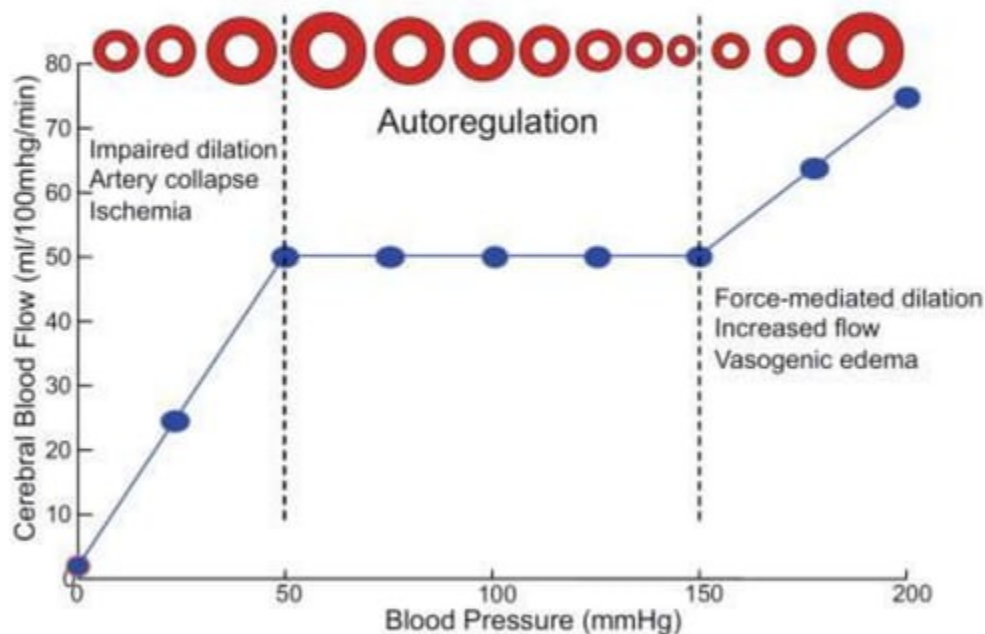




Initiate ICP
treatment at
an upper
threshold of
20 mm Hg.
LEVEL II



CEREBRAL AUTOREGULATION



CEREBRAL PERFUSION PRESSURE

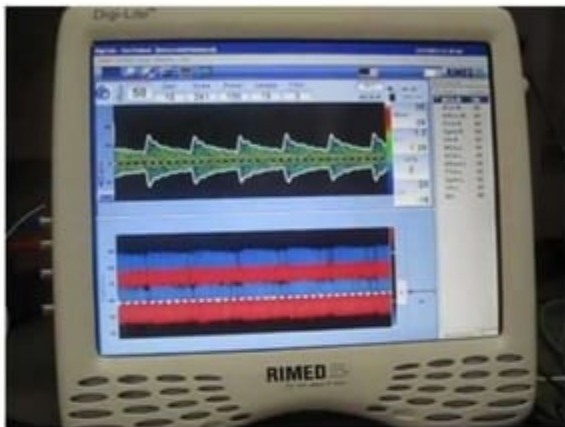
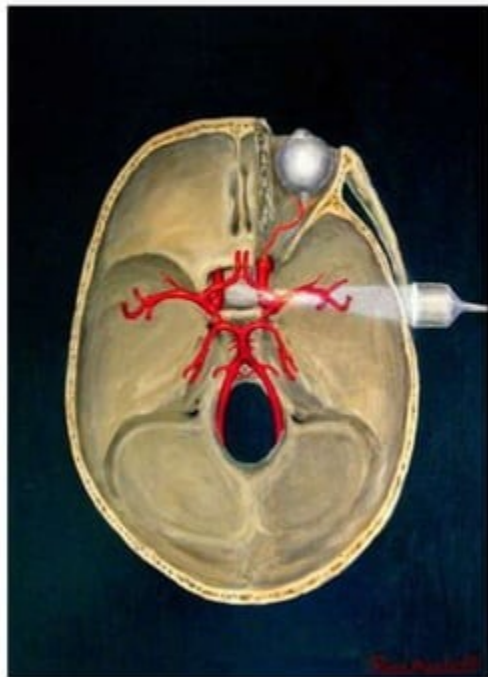
- $CPP = MAP - ICP$

LEVEL II- Avoid aggressive treatment to maintain CPP >70 mm Hg with fluids and pressors because of the increased risk of ARDS.

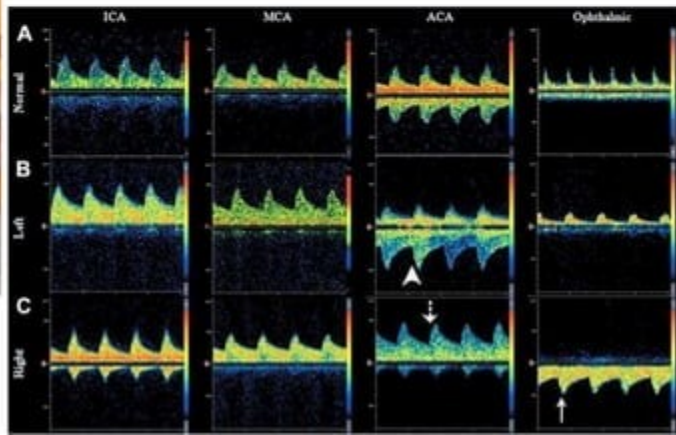
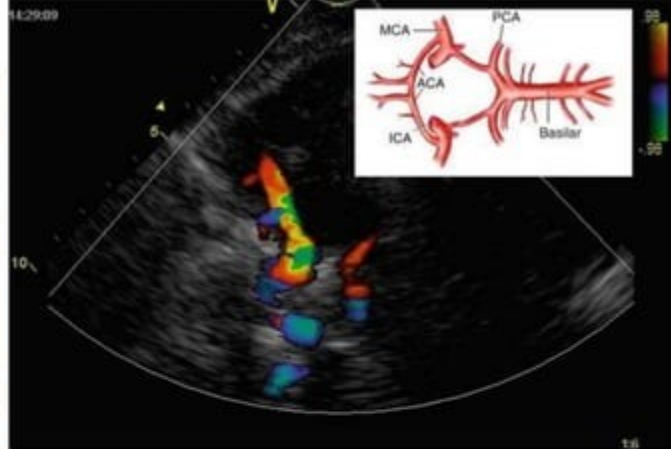
LEVEL III

- Avoid CPP <50 mm Hg.
- Optimal CPP target lies within the 50-70 mm Hg range.
- Patients with intact pressure autoregulation tolerate higher CPP values.
- Monitoring additional parameters such as cerebral blood flow, oxygenation, or metabolism facilitates CPP management.

TRANSCRANIAL DOPPLER



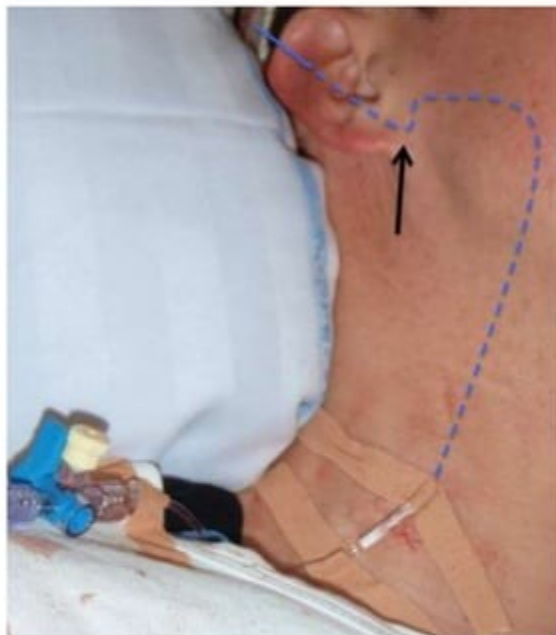
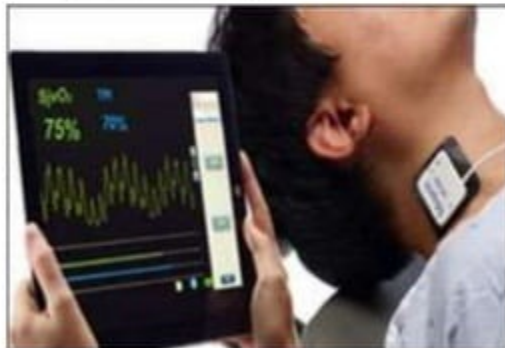
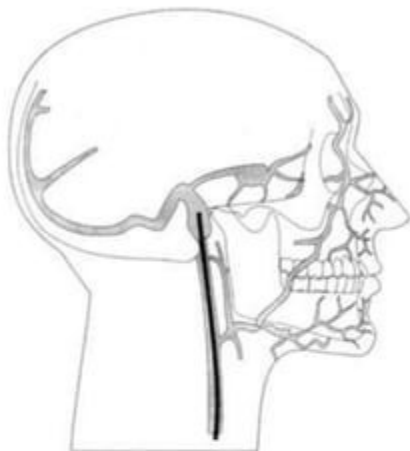
During arterial spasm, FV increases through the narrowed segment proportional to the reduction in the vessel's diameter. Severe vasospasm, with a greater than 50% reduction in vessel diameter, is associated with FV greater than 200 cm/sec



Jugular Venous Oxygen Saturation

- Robertson and associates published a series of 177 patients with severe TBI and demonstrated that 39% of those patients had at least one episode of desaturation;
- Good recovery or moderate disability occurred in 44% of patients with no episodes of desaturation, 30% of patients with one episode, and 15% of patients with multiple episodes of desaturation. Mortality was higher in patients with one episode or multiple episodes (37% and 69%), as opposed to no episodes of desaturation (21%).

Robertson CS, Gopinath SP, Goodman JC, et al. SjvO₂ monitoring in head-injured patients. *J Neurotrauma*. 1995;12:891-896.



Gibbs and colleagues

NORMAL- 55% to 71%

Gibbs EL, Lennox WG, Nims LF, et al. Arterial and cerebral venous blood. Arterial-venous differences in man. *J Biol Chem.* 1942; 144:325-332.

Normal brain metabolism can be altered at SjvO₂ values less than 40% to 50% but that values less than 20% would be required for irreversible ischemic injury

- The current TBI guidelines recommend treating an SjvO₂ less than 50% (level III recommendation).

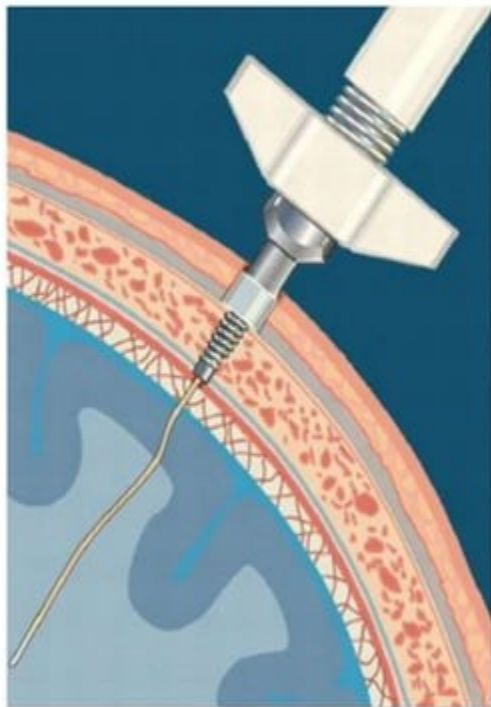
Major limitation of SjvO₂ as a monitor of CBF adequacy is that regional ischemia will not be identified

Complications-

- Site of insertion of the catheter-
 - 1.carotid artery puncture,
 - 2.injury to nerves in the neck,
 - 3.Pneumothorax,
- catheter remaining in the jugular vein-
 - 1.infection,
 - 2.increase in ICP,
 - 3.venous thrombosis.

BRAIN TISSUE PO₂

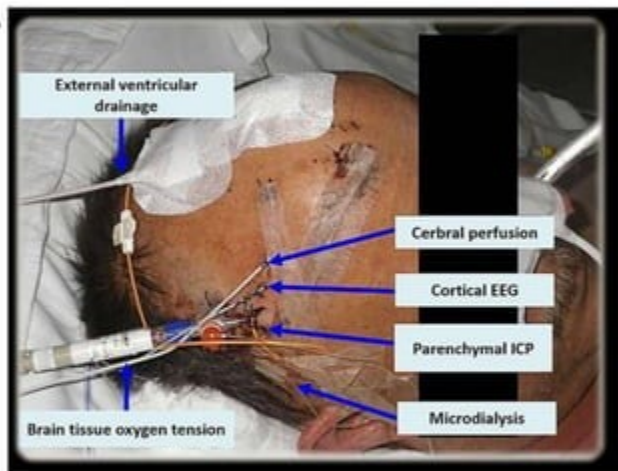
- Normal values for PbtO₂ are 20 to 40 mm Hg, and critical reductions in PO₂ are 8 to 10 mm Hg.
- The current TBI guidelines recommend treatment of PbtO₂ less than 15 mm Hg (level III recommendation).



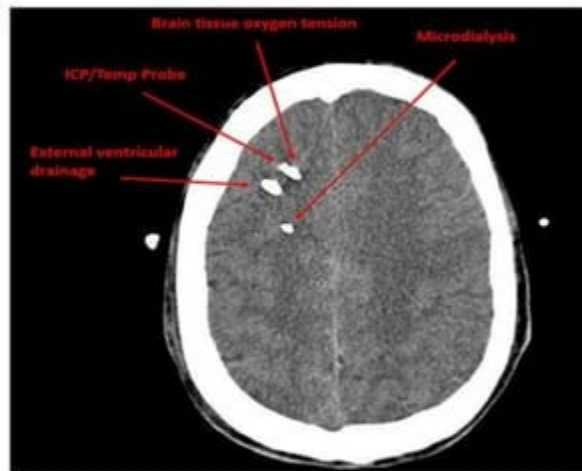
Cerebral Microdialysis

- Intracerebral microdialysis is a brain monitoring modality recently introduced for neurocritical care of brain injury patients.
- Microdialysis is a valuable technique that allows for measurement of endogenous and exogenous molecules of various sizes in repetitive intervals without tissue sampling.
- The principal biochemical markers measured using microdialysis include markers of cerebral metabolism, ischemia, or energy-related metabolism; neurotransmitters or markers of excitotoxic states; and markers of tissue damage and inflammation.

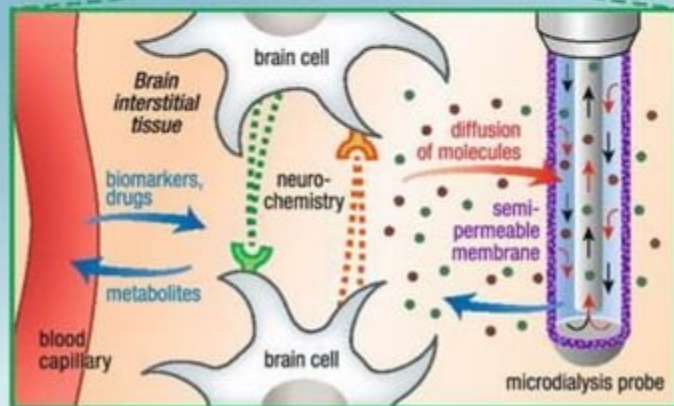
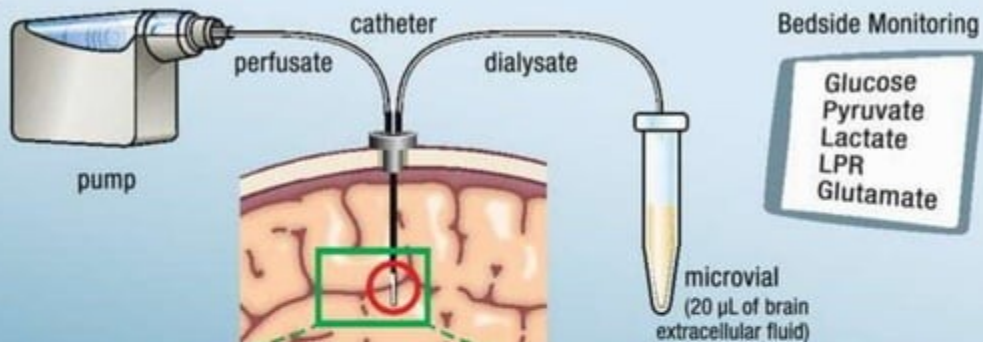
A



B



Cerebral microdialysis



Low CMD glucose (<0.8 mmol/L)

✧ Ischemia/Hypoxia

- ✧ Low CBF / PbtO₂
- ✧ Evidence of cell hypoxia
 - ✧ Elevated L/P ratio
 - ✧ Elevated lactate
 - ✧ Low pyruvate

- ✧ Elevated ICP
- ✧ Inadequate MAP/CPP
- ✧ Delayed cerebral ischemia
- ✧ Hypoxia

• Energy dysfunction

- Normal CBF / PbtO₂
- No evidence of cell hypoxia
 - Elevated L/P ratio
 - Elevated lactate
 - Normal / elevated pyruvate

- Mitochondrial dysfunction
- Cortical spreading depressions
- Hyperglycolysis

➤ *In both cases, low systemic glucose (<6 mmol/L) may further reduce CMD glucose*

* interpretation of the absolute values according to probe location (normal-appearing vs. peri-lesional tissue)

* delayed cerebral ischemia may be detected only when the probe is located within a brain area later affected by secondary infarction

- In cerebral tissue, a semipermeable dialysis membrane is introduced into the brain parenchymal area of interest. The availability of modern analytic techniques has made microdialysis a “universal” biosensor capable of monitoring essentially every small and medium-sized molecular compound in the interstitial fluid.
- The dialysis membrane at the distal end of a microdialysis catheter functions like a blood capillary.
- Chemical substances from the interstitial fluid diffuse across the membrane into the perfusion fluid inside the catheter. The recovery of a particular substance is defined as the concentration in the dialysate expressed as a percentage of the concentration in the interstitial fluid.

- **Intracranial Hypertension-** Intracranial hypertension was the most common cause of jugular venous desaturation. in a prospective study of 116 patients with severe head injury, accounting for 44% of the total number of episodes. As discussed previously, ICP should be monitored, preferably by ventriculostomy

Seizures-

- increase the cerebral metabolic rate of oxygen (CMRO₂) and of glucose.
- If CBF is marginal or uncoupled from cerebral metabolic rate, then ischemia can occur during the seizure.
- The overall risk of seizures after TBI ranges from 2.5% to 5% with all types of injury, but ranges from 7% to 39% with cortical injury and neurological sequelae.
- Seizures can significantly reduce SjvO₂ if CBF is unable to meet the increased cerebral metabolic requirements.

Electrophysiologic Monitoring

- EEG monitoring
- Bispectral index
- Evoked potentials
- Electrocorticographic monitoring

EEG monitoring can be useful to identify occult seizure activity, which has been reported in up to 15% to 18% of patients with moderate and severe TBI

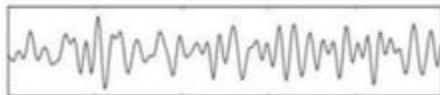




Comparison of EEG Bands



Gamma: 30-100+ Hz



Beta: 12-30 Hz



Alpha: 8-12 Hz



Theta: 4-7 Hz

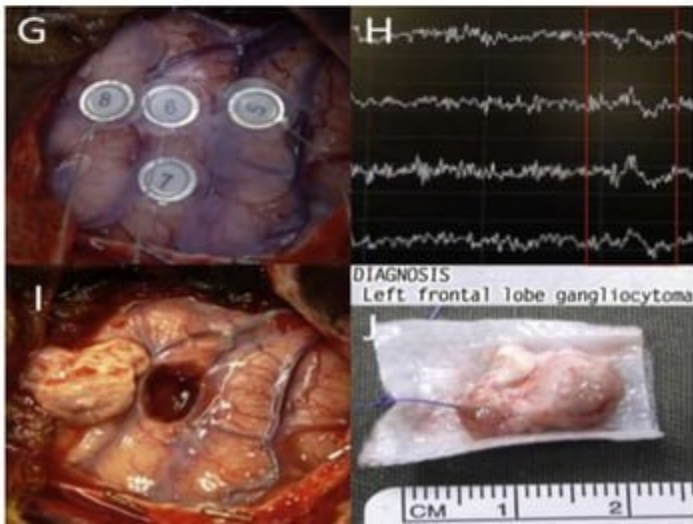


Delta: 0-4 Hz

- **Bispectral index monitoring**- is useful to track the level of sedation during anesthesia, but its added value in TBI is doubtful.
- **Evoked potentials**- may provide detailed insight into connectivity on the brainstem but pose substantial logistic problems in the “noisy” ICU setting.
- **Electrocorticography** –NEW METHOD to monitor for cortical spreading depolarizations, which are pathologic events that recur acutely in the cerebral gray matter of patients with TBI and other acute brain injuries.

ELECTROCORTICOGRAPHY

A subdural strip electrode placed directly on the exposed surface of the brain to record electrical activity from the cerebral cortex.



Hypotension-

CONSTANT BP<90 MM OF HG-worst outcome

Hypocapnia- TO BE KEPT ≥ 36

Areas of the brain that are marginally perfused have impaired CO₂ reactivity, then hyperventilation might constrict the normal areas of the brain and actually improve perfusion to the previously marginally perfused areas. This has been called the “inverse steal response.”

HYPOXIA-

- Normally, a decrease in arterial PO₂ is compensated for by increases in CBF and does not result in a decrease in overall cerebral oxygen delivery.
- TBI impairs this normal CBF response to hypoxia.
- PULMONARY CAUSES.

INITIALLY-

Hypoventilation,
Obstruction of the
airway, Aspiration
Hemo- or pneumothorax.

AFTER 24-48HRS-

Lung contusion
Atelectasis
Fat embolus, Pneumonia
Adult respiratory distress
syndrome (ARDS)

**The goal should be to maintain an arterial
oxygen saturation of 95% or greater**



- **ANEMIA-**
- In normal humans, normovolemic hemodilution sufficient to decrease the hematocrit by 26% resulted in an increase of CBF by 19%.
- As a consequence of the compensatory increase in CBF, SjvO₂ stays relatively constant with normovolemic reduction in hemoglobin concentration.
- Only when the increase in CBF is no longer able to compensate for the reduction in CaO₂ does SjvO₂ decrease.
- After head injury, however, the cerebral vasculature may not be capable of dilating in response to the drop in CaO₂ and ischemia may occur.

Hemoglobin concentration should be measured at least daily during the early phase after injury. In the multiply injured patient, more frequent monitoring is often indicated.

- **FEVER-**
- Fever increases the body's metabolic rate by approximately 10% to 13% per degree Celsius.
- Increasing temperature from 38° to 42°C increased CBF by 97% and CMRO₂ by 65%.
- Measurement of rectal temperature alone may significantly underestimate the actual brain tissue temperature.
- In a study of seven neurosurgical patients, the temperatures of the lateral ventricle, epidural space, tympanic membrane, and rectum were measured.
- Intracranial temperature has also been recorded in neurosurgical patients with a temperature probe that is incorporated into a standard ventriculostomy ICP monitor or with a separate temperature probe.
- Brain tissue oxygen probes usually include a temperature probe and therefore can be utilized to measure intracranial temperature.

In all of these studies, intracranial temperature was 0.5° to 2.0°C higher than body temperature.

NEUROLOGICAL INTENSIVE CARE MANAGEMENT

General Measures to Minimize Intracranial Hypertension/Improve Cerebral Perfusion

1. Minimize Venous Outflow Resistance: Head Elevation and Head Position
2. Sedation/Analgesia
3. Treatment of Systemic Hypertension
4. Airway Protection/Controlled Ventilation
5. Treatment of Fever
6. Prevention of Seizures

Head ELEVATION

- CONTROVERSIAL

Elevation of the head to 30 degrees reduced ICP and increased CPP, but did not change PbtO₂.

The reduction in ICP afforded by 30 degrees of head elevation is probably advantageous.

CAUTION-
Hypovolemic PATIENT
Head elevation results in hypotension

Meixensberger J, Baunach S, Amschler J, et al. Influence of body position on tissue-pO₂, cerebral perfusion pressure and intracranial pressure in patients with acute brain injury. *Neurol Res.* 1997;19:249-253.

SEDATION/ANALGESIA

- Sedative/analgesic drugs blunt the effect that the routine nursing care of patients has on ICP.
- Benzodiazepines cause a coupled reduction in CMRO₂ and CBF, with no effect on ICP.
- Narcotics have no effect on CMRO₂ or CBF but have been reported to increase ICP in some patients.
- avoid drugs with hypotensive side effects
- Hypovolemia predisposes to hypotensive side effects of sedatives

A combination of morphine and lorazepam is a frequently used regimen

TABLE 349-5 Cerebrovascular Effects of Sedative/Anesthetic Agents

	Initial IV Dose	Maintenance Dose	Effect on BP	Effect on ICP	Effect on CBF
BENZODIAZEPINES ³⁴⁰⁻³⁴³					
Diazepam	0.1-0.2 mg/kg		Decrease	No change	Decrease
Lorazepam	0.04 mg/kg		Decrease	No change	Decrease
Midazolam	0.025-0.35 mg/kg	0.05-5 µg/kg/min	Decrease	No change or decrease	Decrease
NARCOTICS ^{186, 350-354}					
Morphine	2-5 mg	2-5 mg/hr	Decrease	Increase	No change
Fentanyl	25-50 µg	50-100 µg/hr	Decrease	Increase	No change
Alfentanil	10-15 µg/kg	0.5-3 µg/kg/min	Decrease	Increase	No change
Sufentanil	0.2-0.6 µg/kg	0.01-0.05 µg/kg/min	Decrease	Increase	No change
OTHERS ^{355, 356}					
Propofol	1-2 mg/kg	5-50 µg/kg/min	Decrease	Decrease	Decrease
Etomidate	0.3-0.4 mg/kg		Decrease	Decrease	Decrease

BP, blood pressure; CBF, cerebral blood flow; ICP, intracranial pressure; IV, intravenous.

PROPOFOL INFUSION SYNDROME- potent systemic vasodilator- hyperkalemia, hepatomegaly, lipemia, metabolic acidosis, myocardial failure, rhabdomyolysis, and renal failure.

Caution - > 5 mg/kg per hour or when usage of any dose exceeds 48 hours in critically ill adults.

KETAMINE IS CONTRAINDICATED-RAISES ICP

Dexmedetomidine is an α -2-agonist that provides adequate sedation without altering respiratory drive, while facilitating frequent neurological examinations

Treatment of Systemic Hypertension

- systolic blood pressure >160 mm Hg

PREFERRED-

1.SEDATIVES

2.BETA BLOCKERS- (propranolol, esmolol, or labetalol)

3.Centrally acting α -receptor agonists (clonidine or α -methyldopa)

Reduce blood pressure without affecting the ICP

CAUTION-

vasodilating antihypertensive drugs

hydralazine

Nicardipine

Nitroprusside

consistently increase ICP

NICARDIPINE-reverse and prevent vasospasm IN MODERATE TO SEVERE TBI

Airway Protection/Controlled Ventilation

INTUBATION TO PREVENT ASPIRATION
SERIAL ABG

Mechanical ventilation- acute recovery period
-allow deeper levels of sedation to be used
-reduce the risk that periodic hypoventilation will exacerbate intracranial hypertension.

Sedation and occasionally paralysis may be necessary to control ventilation

- TREATMENT OF FEVER
- TREATMENT OF SEIZURE

The risk of posttraumatic seizure is approximately 15% with severe head injury

RISK FACTORS

Brain contusion with subdural hematoma

Skull fracture

Loss of consciousness or amnesia for more than 1 day

Age of 65 years or older

AED

Severe TBI-

in the emergency room phenytoin, 15 mL/kg intravenously at a rate that does not exceed 25 mg/min, followed by a daily maintenance dose adjusted to keep plasma levels in the therapeutic range.

- If no early seizures occur, then the phenytoin is tapered and discontinued after 1 week.

Levetiracetam- BETTER

- does not require serum monitoring or have significant pharmacokinetic interactions
- as effective as phenytoin in preventing early posttraumatic seizures
- but it was associated with an increased tendency for seizure activity in EEG analysis

Other General Measures

1. Prevention of Hospital-Acquired Infections: Ventilator-Associated Pneumonia

Antibiotic prophylaxis

Limit the duration of intubation and mechanical ventilation

2. Prophylaxis for Thromboembolism

Low-dose heparin and sequential venous compression prophylaxis

3. Prophylaxis for Gastric Ulcers

SUCRALFATE /PPI

Histamine₂ blockers in neurosurgical patients was associated with an increased incidence of thrombocytopenia- *Ecker and associates*

4. Nutritional Support

In a large retrospective study of hospitalized TBI patients, those who were not fed within 5 and 7 days after TBI had a two- and fourfold increased likelihood of death, respectively.

The amount of nutrition in the first 5 days was related to death; every 10-kcal/kg decrease in caloric intake was associated with a 30% to 40% increase in mortality rates adjusted for injury severity.

5. Management of Fluid and Electrolytes

- INITIAL RESUSCITATION
- Maintenance Fluids. After resuscitation, maintenance fluids, usually **0.9% normal saline**, should be given at **35 mL/kg per day**. Additional fluids may be needed to replace fluid losses from nasogastric aspiration and/or other drains.

HYPONATRAEMIA SYNDROMES

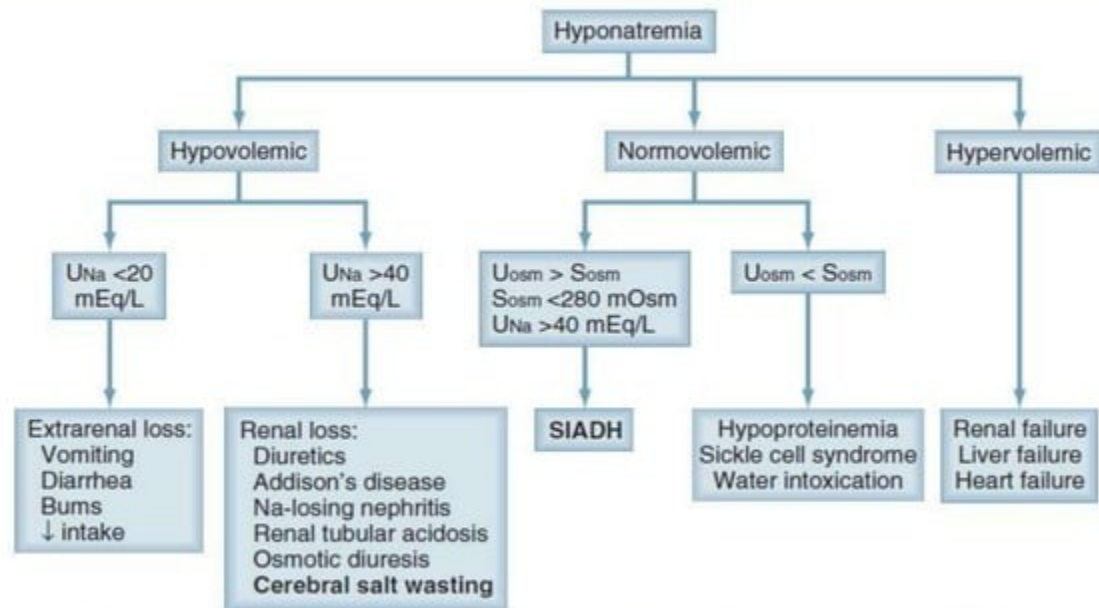


Figure 349-3. In head-injured patients, most hyponatremia can be attributed to either cerebral salt wasting or the syndrome of inappropriate antidiuretic hormone secretion (SIADH). S_{osm} , serum osmolality; U_{Na} , urine sodium; U_{osm} , urine osmolality.

- **HYPERNATRAEMIA- DI**

- ✓ MILD TO MOD.- FREE WATER

- ✓ SEVERE- aqueous desmopressin acetate 2 to 4 μg will decrease free water clearance for 8 to 12 hours

- **HYPERGLYCAEMIA- TARGET-80-110 mg/DL**

Treatment of Secondary Injury Processes: Intracranial Hypertension

- Pharmacologic Paralysis
- Hyperventilation
- CSF Drainage
- Osmotherapy-**Hyperosmolar Therapy: Mannitol versus Hypertonic Saline**
- Barbiturate Coma
- Hypothermia - LEVEL III
- Decompressive Craniectomy

PHARMACOLOGIC PARALYSIS

- ICP raised by agitation, posturing, or coughing should be prevented by narcotics and nondepolarizing muscle relaxants that do not alter cerebrovascular resistance

TABLE 349-6 Cerebrovascular Effects of Muscle Relaxants

	Intubation Dose (mg/kg)	Maintenance Dose (mg/hr)	Duration of Effect (min)	Effect on BP	Effect on ICP	Effect on CBF
DEPOLARIZING ^{167, 368}						
Succinylcholine	1		5-10	No change	Increase	Increase
NONDEPOLARIZING ^{369, 368}						
d-Tubocurarine	0.5	1.5-2	150	Decrease	Increase	Increase
Metocurine	0.4	7	150	Decrease	No change	No change
Pancuronium	0.1	4-8	120	Increase	No change	No change
Atracurium	0.5	20-50	30-45	Decrease or no change	No change	No change
Vecuronium	0.1	4-10	45-60	No change	No change	No change
Doxacurium	0.05	0.35-2.1	100-120	No change	No change	No change
Pipecuronium	0.1		120-150	No change	No change	No change

BP, blood pressure; CBF, cerebral blood flow; ICP, intracranial pressure.

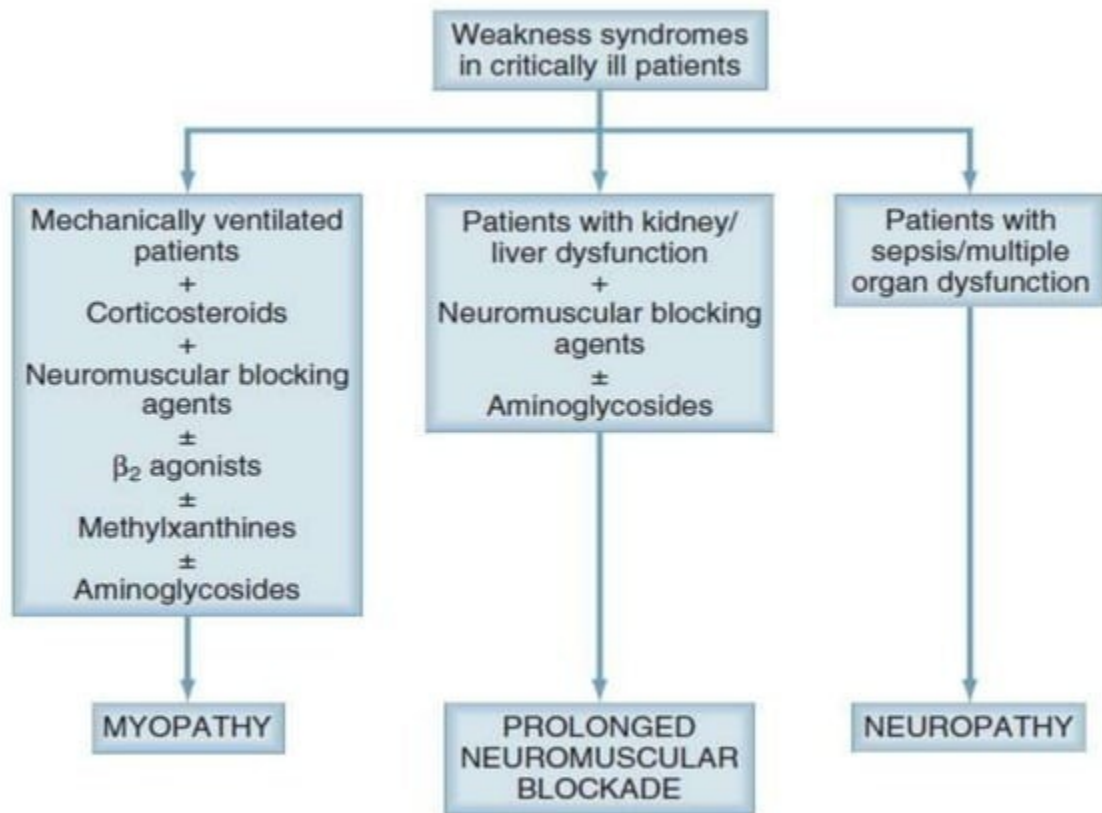
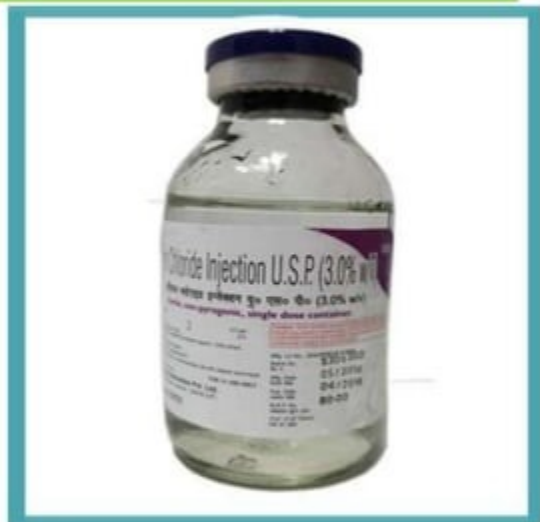


Figure 349-4. Diagram of weakness syndromes in critically ill patients.

Hyperventilation

- Routine hyperventilation (to an arterial PCO₂ of 20 to 25 mm Hg) has been shown to have a detrimental effect on outcome not recommended in the current TBI guidelines. (Level II recommendation).
- The BTF guidelines recommended using hyperventilation only in patients with intracranial hypertension rather than routinely in all head-injured patients.
- In general, hyperventilation should not be used during the first 24 hours postinjury, and should be used only if PbtO₂ or SjvO₂ is monitored to make certain that ischemia is not being produced

MANNITOL VS 3% NS



- Hypertonic saline has an advantage over mannitol in hypovolemic patients, in whom hypertonic saline augments intravascular volume and may increase blood pressure in addition to decreasing ICP.
- Hypertonic saline has been shown to reduce ICP with concurrent durable elevations in CPP, and also has been shown to increase brain oxygenation levels.
- A Cochrane database review of hypertonic saline versus mannitol in patients undergoing craniotomy also failed to show any outcome difference.
- Currently there are not enough data to recommend one therapy over the other; both are effective agents.

CSF Drainage

- Drainage of 1 to 2 mL of CSF through the ventriculostomy catheter can temporarily lower ICP. This modality can be an important adjunct therapy for lowering ICP.
- However, as the brain becomes more swollen, the ventricles collapse, less CSF is available for drainage, and the effectiveness of this modality is reduced.

BARBITURATE COMA

- In addition to lowering ICP, extracellular concentrations of lactate and excitatory amino acids are reduced after treatment with barbiturates
- Pentobarbital is given in both loading and maintenance doses. The loading dose is 10 mg/kg given over 30 minutes, followed by 5 mg/kg each hour for three doses
- Reserved for patients with intracranial hypertension resistant to other modalities

Decompressive Craniectomy

- The recent Decompressive Craniectomy (DECRA) study compared early bifrontal decompressive craniectomy with standard medical therapy for diffuse cerebral swelling and an ICP greater than 20 mm Hg for more than 15 minutes was used as the threshold for enrollment.
- The results demonstrated that, although the ICP for those patients in the surgical arm was consistently lower following surgical decompression than in the patients who received standard medical therapy, outcomes at 6-month follow-up were worse for those patients in the surgical arm of the trial.
- Randomized controlled trials in TBI—the Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of intracranial pressure (RESCUEicp) and DECRA studies—are ongoing.

SEVENTH EDITION

YOUmans & WINN

NEUROLOGICAL SURGERY



ELSEVIER

H. RICHARD WINN

THANK YOU