Neuro Anesthesia for Traumatic Brain Injury

A Review of The Basics

Michele Kolowitz CRNA, MHS
October 2014

Introduction

CDC

- 1.7 Million sustained TBI
- 52,000 Deaths
- 275,000 Hospitalizations
- 80% treated & released from ER (~1.3 million)
- Those $\geq$ 75 highest rates of TBI-related hospitalization & death

The Brain Trauma Foundation

- TBI affects 2% of the population annually
- Major cause of death & severe disability among young people
- Most important complication—intracranial hematoma

CDC 2002-2006
The BTF 2007

Objectives

- Review Cerebral Anatomy, Physiology, & Circulation
- Explore The 2007 Brain Trauma Foundation Guidelines for Management of Severe TBI
- Discuss Sodium & Water Balance after TBI
  - Central Neurogenic Diabetes Insipidus
  - Syndrome of Inappropriate Secretion of Antidiuretic Hormone
  - Cerebral Salt-Wasting Syndrome

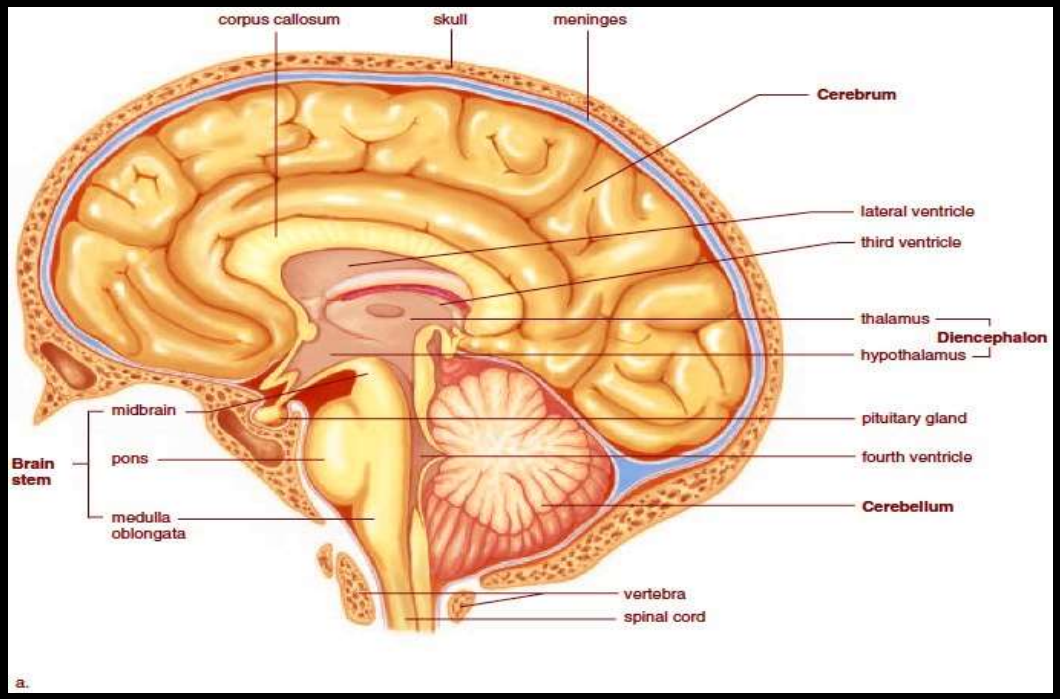
Cerebral Anatomy

Cranial Vault

- Brain 80%
- Blood 12%
- CSF 8%

Brain

- 1300 grams (3lbs)
- ~20% Cardiac Output
- High metabolic rate
- Absence of O_2 stores



Cerebral Metabolism—$CMRO_2$

Oxygen Consumption 3-3.8mL/100g/min

Average adult ~50ml/min

60% generate ATP neuronal electrical activity

***ABSENCE of significant O_2 reserves**

when O_2 tension **<30 mm/Hg**

3-8 min before ATP depleted→irreversible cellular injury

Cerebral Metabolism & Glucose

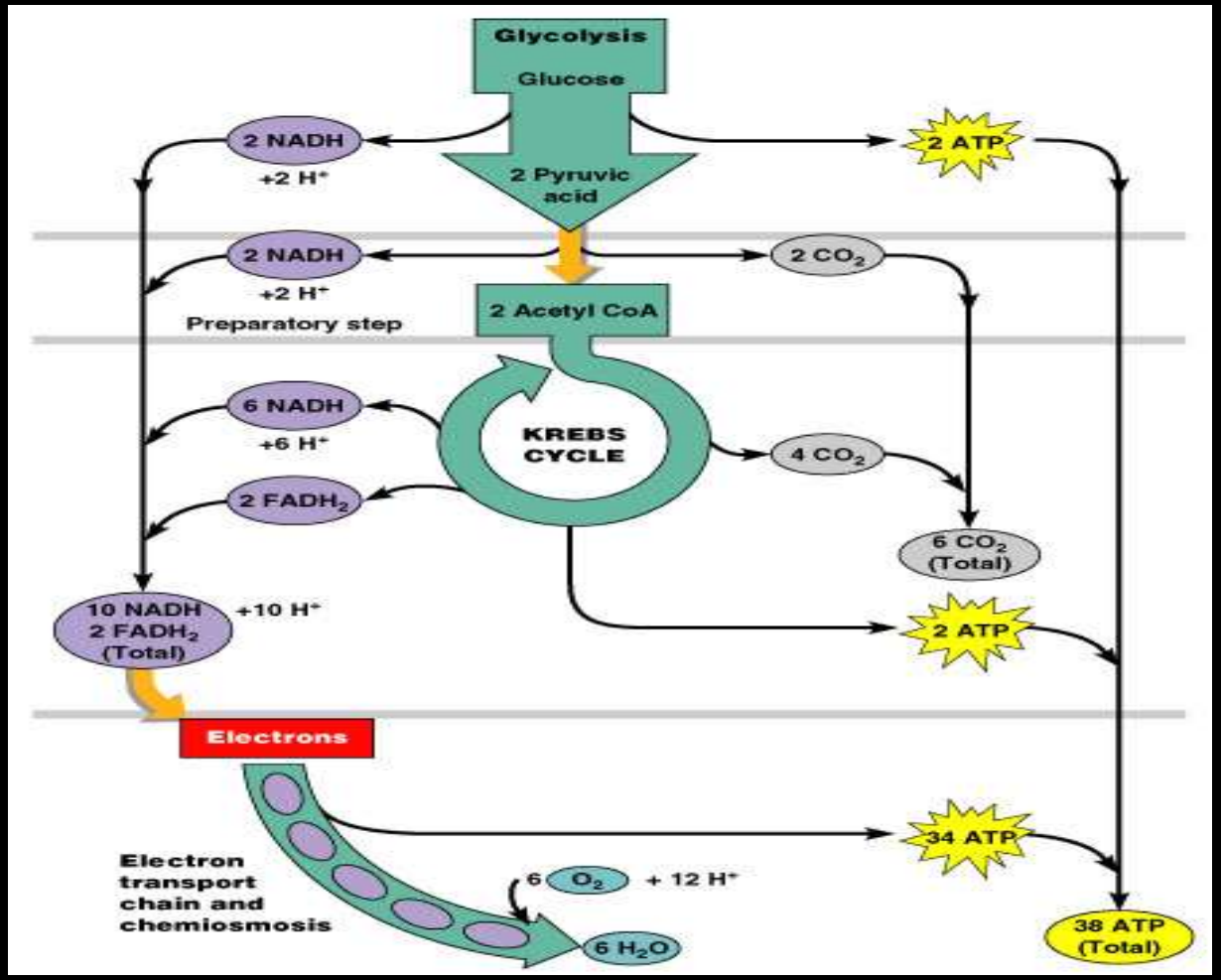
Glucose 5mg/100g/min

Average Adult ~65-70mg/min

- 90% aerobic metabolism
- $CMRO_2$ parallels glucose consumption
- Can metabolize some lactate

***Acute Sustained HYPOglycemia is equally as devastating as hypoxia**

Aerobic vs. Anaerobic Metabolism



Cerebral Blood Flow

Normal CBF→ 40-50ml/100g/min
Average adult ~750 ml/min

- Global BF & metabolic rate remain fairly stable
- Regional BF & metabolic rate can change dramatically

As metabolic rate goes up, BF goes up—Coupling

Increase [K^+ & H^+] in ECF arteriole dilation & ↑ BF

Barash 2006

Manipulating CO_2

⇧ CO_2 causes vasodilation & ⇧ Blood Flow

- ⬆CO_2 from 40 to 80 mm/Hg—DOUBLE BF
- ⬇CO_2 from 40 to 20 mm/Hg—HALVES BF

*Changes are transcient lasting ~6-8hours. BF returns to normal even if we attempt to maintain the CO_2 levels. HCO_3- level of brain ECF returns the pH to normal

Barash 2006

Low CBF Rates & Consequences

- CBF <20-25ml/100g/min
  - Cerebral impairment
  - Slowing EEG
- CBF 15-20ml/100g/min
  - Flat isoelectric EEG
- CBF 10ml/100g/min
  - Irreversible brain damage

Morgan Mikhail & Murray 2006

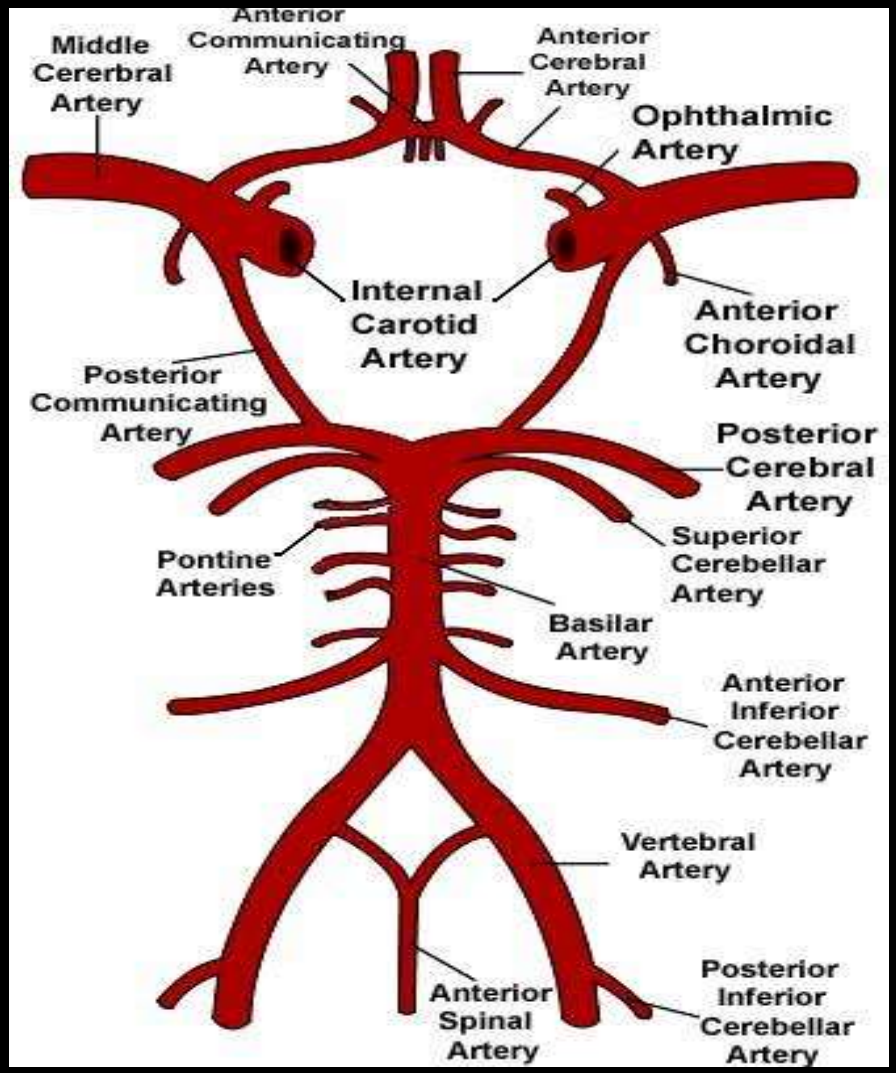
Agent	CMR	CBF	CSF Production	CSF Absorption	CBV	ICP
Isoflurane	↓↓↓	↑	±	↑	↑↑	↑
Desflurane	↓↓↓	↑	↑	↓	↑	↑↑
Sevoflurane	↓↓↓	↑	?	?	↑	↑↑
Barbiturates	↓↓↓↓	↓↓↓	±	↑	↓↓	↓↓↓
Etomidate	↓↓↓	↓↓	±	↑	↓↓	↓↓
Propofol	↓↓↓	↓↓↓↓	?	?	↓↓	↓↓
Benzos	↓↓	↓	±	↑	↓	↓
Ketamine	±	↑↑	±	↓	↑↑	↑↑
Opioids	±	±	±	↑	±	±
Lidocaine	↓↓	↓↓	?	?	↓↓	↓↓

Anatomy of Cerebral Circulation

- Originates from 2 arterial circulations—from 2 distinct systemic arteries

- Anterior Circulation
  - Carotid arteries
- Posterior Circulation
  - Vertebral arteries

Circle of Willis



Internal Carotids

- Anterior Cerebral
- Middle Cerebral

*Supply medial & lateral surfaces of cerebral hemispheres

Vertebral Arteries

- Posterior Cerebral

*Supply wide area within Brain & spinal cord; Occipital & Temporal lobes

Graphic: Vascularultrasound.net

Nagelhout & Plaus 2010

Anomalies of the intracranial collateral blood supply are COMMON in the general population

52%

Complete Circle of Willis

Drummond, Englander, & Gallo 2006

Cerebral Ischemia as an Apparent Complication of Anterior Cervical Discectomy in a Patient with an Incomplete Circle of Willis.

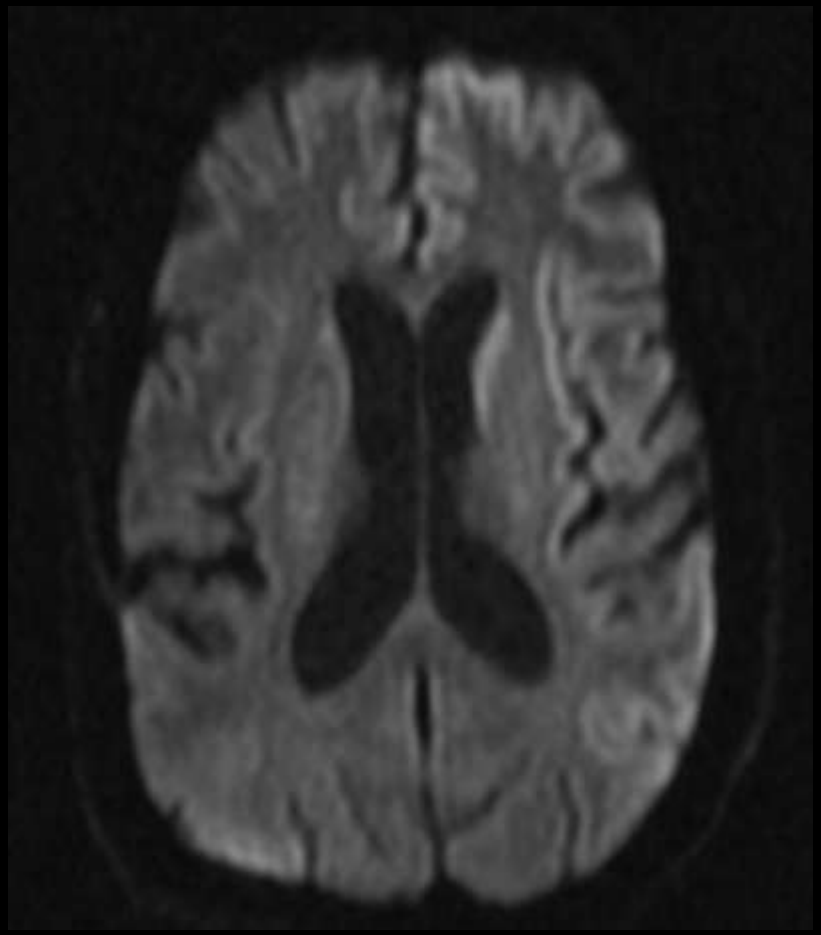
The University of California, San Diego; VA Medical Center, San Diego; Department of Neurology, Department of Neurosurgery, Sacred Heart Medical Center, Eugene, Oregon

Case Report

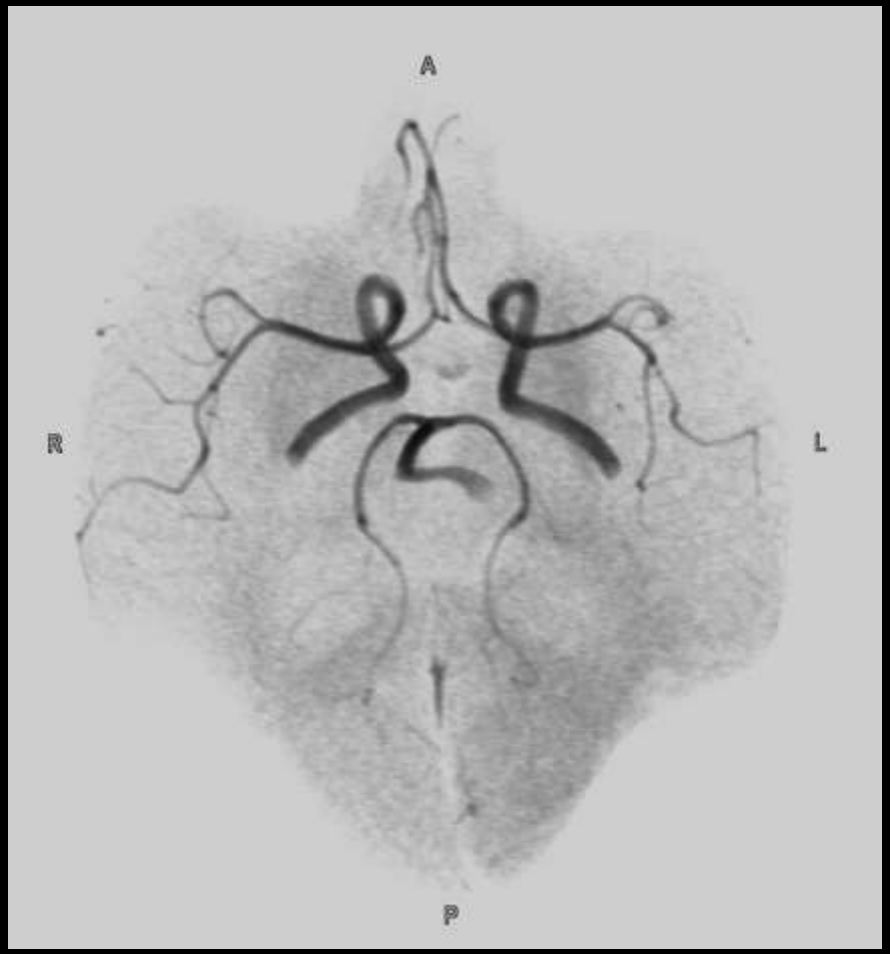
58yo Male
Ischemic injury ipsilateral to retraction
Carotid compression
Moderate arterial BP reduction
Preop MAP 99 mm/Hg; Intraop MAP ~56 mm/Hg
Extubated…Reintubated
Immediate postop—all tests normal CT, MRI, MRA, etc
POD #13 Repeated Brain MRI
MRA Circle of Willis

Drummond, Englander & Gallo 2006

Brain MRI

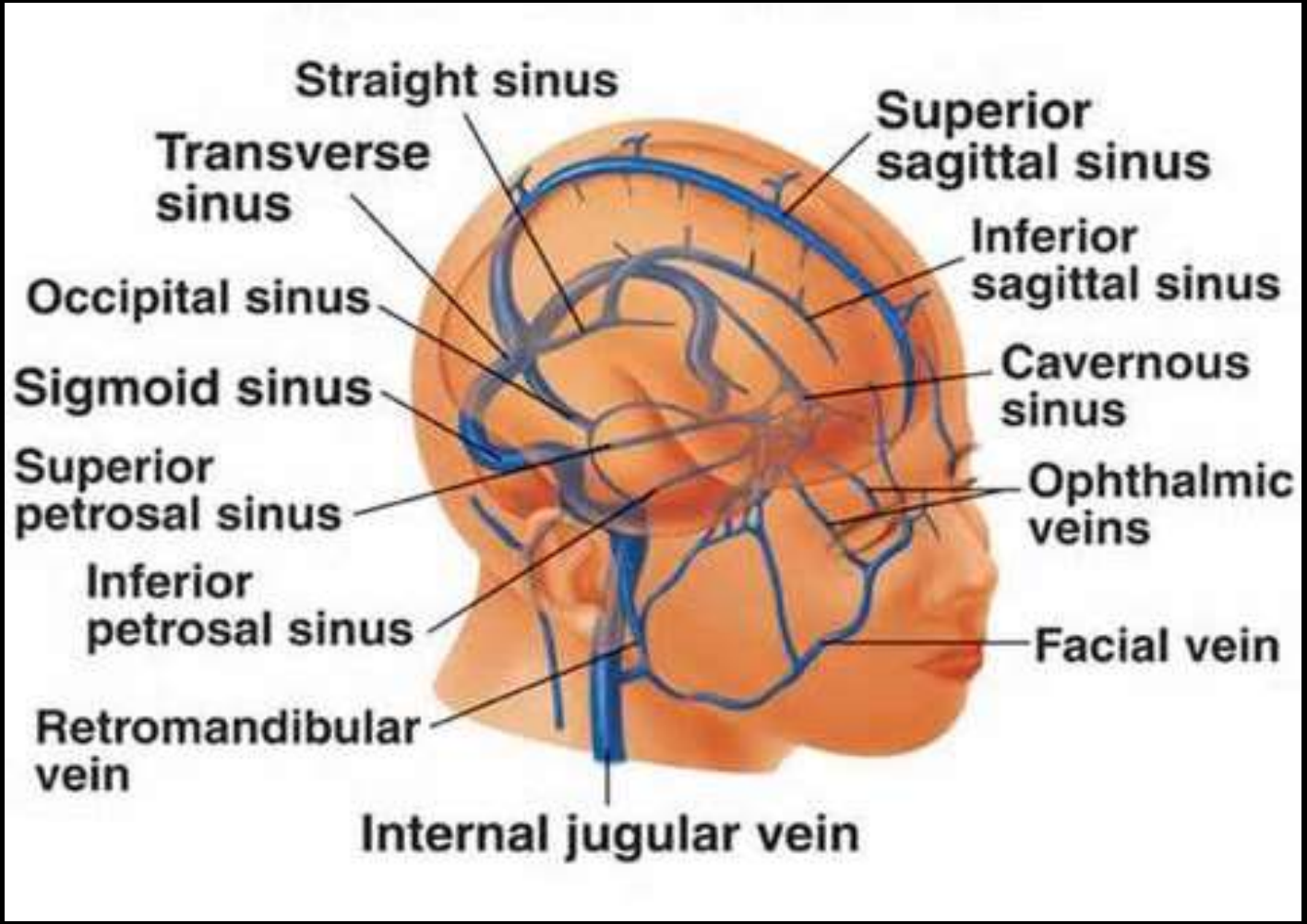


MRA Circle of Willis



Drummond, Englander, & Gallo 2006

Sinuses Drain into the Internal Jugular Vein



Graphic: rci.rutgers.edu

Cerebral Spinal Fluid

Production

- Formed Choroid Plexus
- 21ml/hr or 500ml/day
- Quick turnover rate
- ~150ml present at a given time
- Reabsorbed Arachnoid Villi

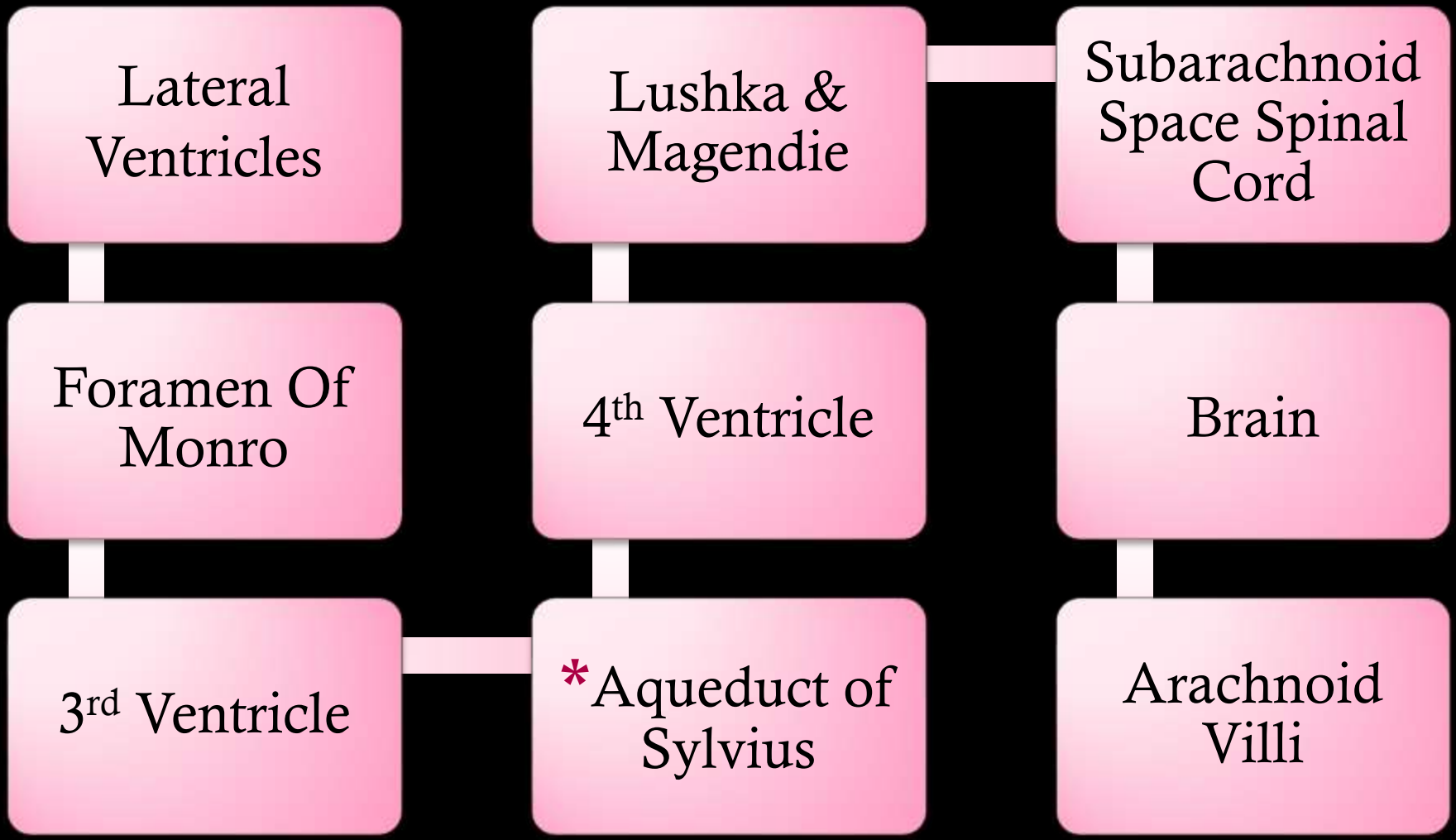
Composition

- Na^+ 141 mEq/l
- K^+ 2.9 mEq/l
- Ca^{2+} 2.5 mEq/l
- Mg^{2+} 2.4 mEq/l
- Cl^- 124 mEq/l
- HCO_3^- 21 mEq/l
- PRO 28 mg/100mL
- Glu 61 mg/100mL

pH 7.31

Barash 2006

Pattern of CSF Flow



Intracranial Pressure

ICP

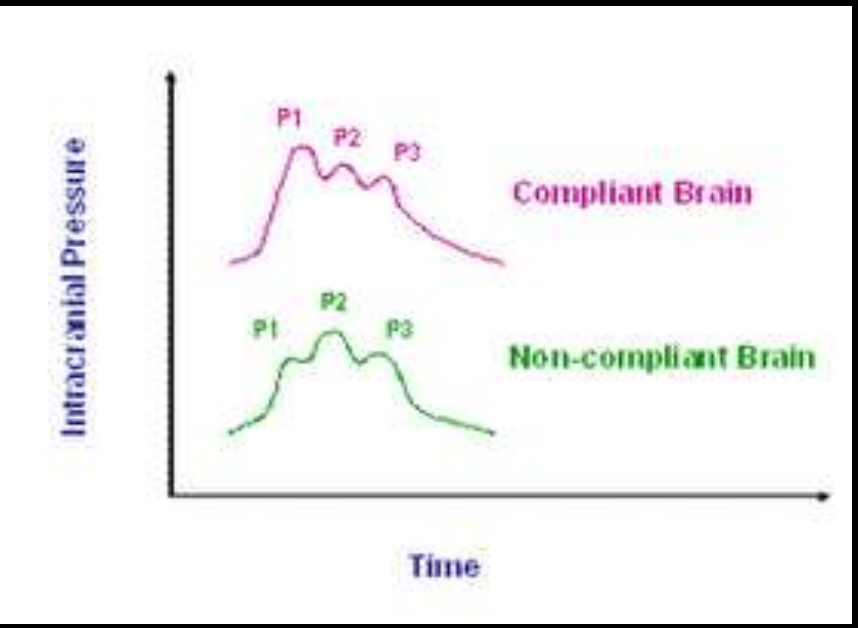
- Normal <10 mm/Hg
- ICP >15 mm/Hg →Intracranial HTN
- Most centers treat when ICP >20-25 mm/Hg

***BTF Guidelines support the initiation of treatment when ICP is ≥ 20 mm/Hg**

Marked INCREASES in ICP can DECREASE Cerebral Perfusion Pressure & Cerebral Blood Flow→ producing regional & general ischemia.

Li, Timofeev, Czosnyka, & Hutchinson 2010

ICP Waveform



Flow of 3 upstrokes in 1 wave

P1—Percussion wave
Atrial pulsation

P2—Tidal wave
Intracranial compliance

P3—Dicrotic wave
aortic valve closure

***If P2 is higher than P1→ indicates Intracranial HTN**

Graphic: eneurosurgery.com

Cerebral Perfusion Pressure

CPP= MAP-ICP

Or **CVP** (which ever is greater)

Normal CPP is 80-100 mm/Hg

- CPP <50mm/Hg—EEG slowing
- CPP 25-40mm/Hg—Flat EEG
- CPP <25mm/Hg—Irreversible brain damage

***BTF Guidelines target CPP of 50-70 mm/Hg**
No more than 70 mm/Hg

Morgan Mikhail & Murray 2006

Autoregulation at Work

- ↑ CPP—cerebral vasoconstriction
  prevents sudden ⇧CBF & CBV

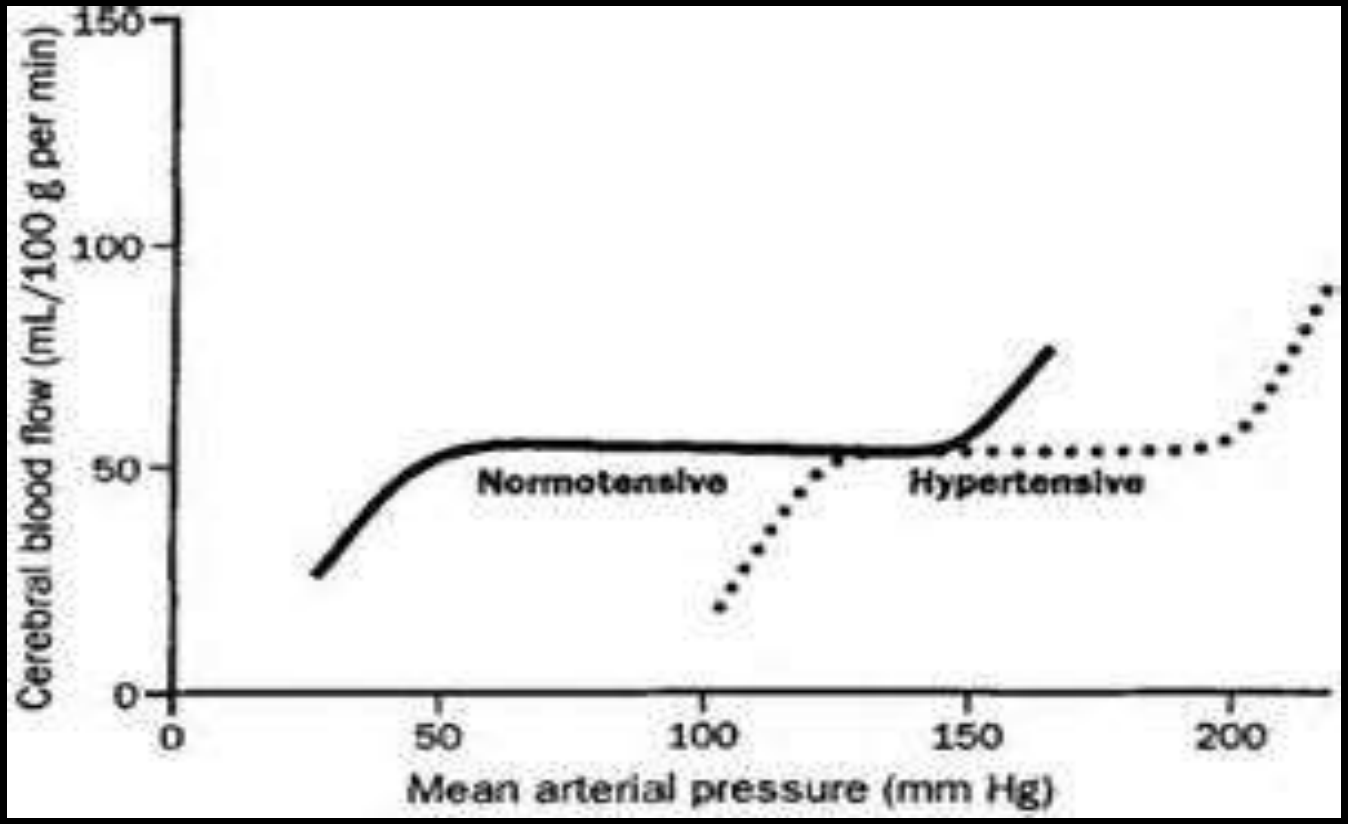
- ↓CPP—cerebral vasodilation
  maintain CBF & perfusion

*Upper limits of CPP **150-160mm/Hg**
CPP >150 Risk: Disruption of BBB
Cerebral edema
Hemorrhage

TBI capillaries become leaky & more permeable to water vessels dilate, flow increases and edema worsens

Autoregulation & Chronic HTN

***Autoregulatory curve Shifts to the RIGHT**



Graphic: jasn.asnjournals.org

Impaired Autoregulation

- Cerebral edema—Trauma
- Cerebral ischemia—Hypoxia
- Hypercarbia
- Volatile agents—Vasodilating drugs
- Subarachnoid Hemorrhage—vasospasm

Symptoms of Increasing ICP
Mild to Moderate TBI

Vague & Nonspecific

- Confusion
- Headache
- Drowsiness

Fundamental Clinical Variable

GLASGOW COMA SCALE

↓Value in MOTOR component=potential increasing ICP

Determines how the neuro status will be monitored

BEHAVIOR	RESPONSE	SCORE
Eye Opening Response	Spontaneous To speech To pain No Response	4 3 2 1
Best Verbal Response	Oriented x3 Confused Inappropriate words Incomprehensible sounds No response	5 4 3 2 1
Best Motor Response	Obeys Commands Moves to localized pain Flexion/withdrawal from pain Abnormal flexion Decorticate Abnormal extension Decerebrate No response	6 5 4 3 2 1
Total Score	Best Response **Comatose** Unresponsive	15 **≤8** 3

The Surgical Approach to the Management of Increased Intracranial Pressure After Traumatic Brain Injury;

Academic Neurosurgery unit, University of Cambridge/Addenbrookes Hospital, United Kingdom.

↑ICP within 1st 24hours of injury as well as any Secondary ↑ ICP (3-10 days posttrauma) → POOR prognosis

Secondary Causes of increased ICP

Cerebral Edema
Mass lesion
Cerebral vasodilation
Systemic HTN
Venous sinus thrombosus
Posttraumatic seizure
↑ Intrathoracic pressure
Hyperthermia

Li, Timofeev, Czosnyka, & Hutchinson 2010

Surgical Approach Cont'

Optimal approach—Anticipate the onset ↑ ICP

- Neurosurgery involved early
  - Assessment, treatment, & planning
- Treated in Neurosurgical Centers
  - Even if injury does NOT require neurosurgical intervention
- High Risk patients Neurosurgical unit w/option for Neuro ICU care

Li, Timofeev, Czosnyka, & Hutchinson 2010

The Brain Trauma Foundation Guidelines

Who is High-Risk for ⇧ ICP?

- Severe head injury (GCS score ≤ **8** after CPR)

PLUS

  - Abnormal head CT on admit **or**
  - Normal head CT PLUS any 2:
    - Age >40
    - SBP <90 mm/Hg
    - Decerebrate or Decorticate posturing

*No definitive CT feature indicates ⇧ICP

- Sedated or Induced Coma after Severe TBI
- Multisystem Injury w/altered LOC
- Receiving RX w/High volume IVF
- Postop after mass lesion removal

Li, timofeev, Czosnyka, & Hutchinson 2010

Mainstay of ICP Management

University of Cambridge/Addenbrookes Hospital, United Kingdom

Medical Management with Protocols

• Head elevation	10-15 degrees
• Adequate oxygenation	Sat $\geq$ 97%
• Fluid resuscitation	CVP 6-10
• Sedation	Propofol, versed, fentanyl
• Muscle relaxation	Atracurium 0.5mg/kg/hr
• Mild hyperventilation	
• Cooling	$\leq$ 37°C

Surgery is 2nd tier treatment
in patient's whose ⇧ ICP is refractory to maximal medical management

Li, Timofeev, Czosnyka, & Hutchinson 2010

Pharmacology
Barbiturate

- Thiopental
  - Hypnosis
  - Depression of CMR
  - ↓ CBF r/t ↑ CVR
  - Anticonvulsant property
  - Facilitate absorption of CSF

250mg boluses
up to 3-5grams
IV infusion 4-8 mg/kg/hr

"Robin Hood", reverse steal phenomenon
induces vasoconstriction in normal brain tissue, blood flow is redistributed to ischemic areas of the brain

Morgan, Mikhail, Murray 2006

Mainstay of ICP Management

University of Cambridge/Addenbrookes Hospital, United Kingdom

All Patients with or at risk for developing increased ICP

- Arterial line
- CVP line
- ICP monitor
- Rt $SjVO_2$ catheter $\geq 55\%$

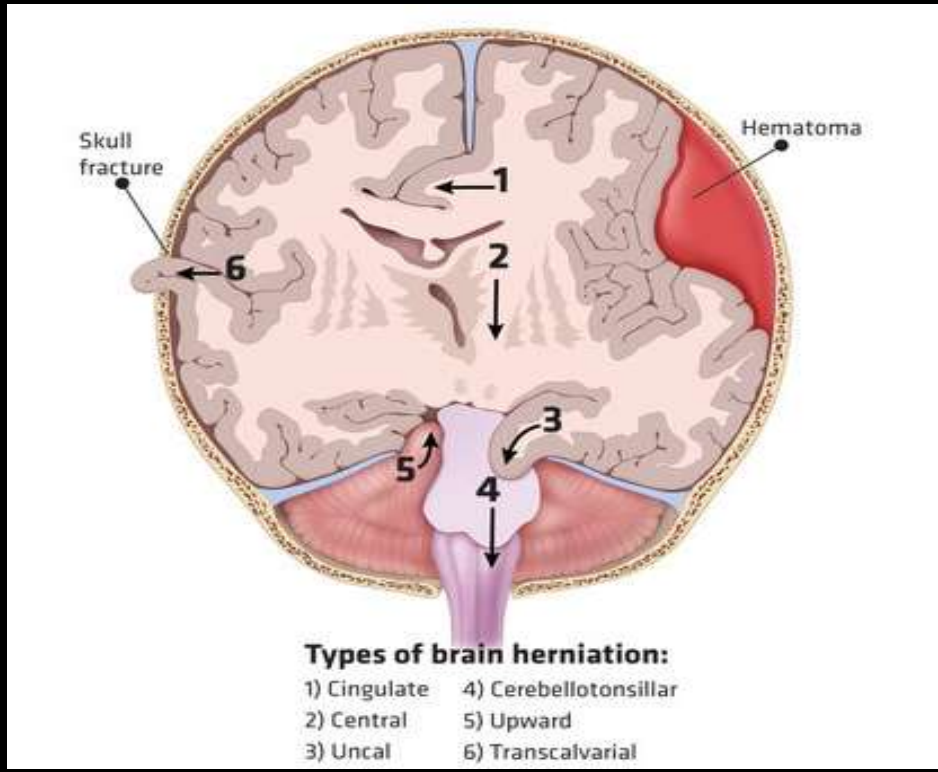
Establish transcranial doppler & multimodality monitoring within first 6hrs of Neuro ICU stay.

Li, Timofeev, Czosnyka, & Hutchinson 2010

Herniation

Elevated ICP can induce brain herniation

- Across meninges
- Down spinal canal
- Through opening in the skull
- Rapid neurological deterioration and death



Graphic: emsworld.com

Cushing's Triad
⇧SBP
Bradycardia
Abnormal breathing pattern

The Brain Trauma Foundation Guidelines

- Hypoxemia and Hypotension
- Hyperosmolar Therapy
- Indications for ICP Monitoring
- ICP Monitoring Technology
- ICP & CPP Thresholds
- Brain Oxygen Monitoring and Thresholds
- Analgesics, Anesthetics, and Sedatives

The Brain Trauma Foundation Guidelines

Hypoxemia and Hypotension

- Defining level of Hypotension is unclear
- SBP <90 mm/Hg—avoid or rapidly correct

Given the influence that CPP has on outcome:
SBP >90mm/Hg would be more desirable
esp during pre-hospital & resuscitative phases

- Hypoxia—apnea cyanosis in the field or
  PaO_2 <60 mm/Hg

The Brain Trauma Foundation Guidelines

Hyperosmolar Therapy

Agents currently in clinical use for TBI

- Mannitol
  - Single administration—short term effects
  - Prolonged therapy for ⇧ ICP

***Lack of evidence for repeated regular administration over several days**

- Hypertonic Saline
  - Current evidence is NOT strong enough to recommend use, concentration, & administration of HS in traumatic intracranial HTN

Mannitol

0.25gm/kg to 1gm/kg
Avoid SBP <90mm/Hg

Exerts beneficial effects by 2 mechanisms:

- Immediate plasma expanding effect
  - ↓ HCT and ↓ Blood viscosity
  - ↑ CBF and ↑ O_2 delivery
- Osmotic effect—delayed 15-30 minutes
  - Establishment of gradients between cells & plasma
  - Effects can persist for ~90 min to 6+ hours

BTF 2007

Hypertonic Saline

- Principle effect on ICP is osmotic movement of water across an intact BBB
- Dehydrates endothelial cells and erythrocytes
  - Increasing diameter of vessels
  - Plasma volume expansion
  - Increased CBF
- Reduces leukocyte adhesion in traumatized brain

Potential Side Effects of HS Administration

- Exclude Hyponatremia prior to HS therapy
- Pre-existing chronic hyponatremia—risk of

  Central Pontine Myelinolysis→ destruction of myelin, symptoms appear ~2-3 days

- Induce or aggravate Pulmonary edema

  Underlying cardiac or pulmonary problems

3% HS as Continuous infusion titrate:
Serum Na→145-155mEq/L
Serum Osmolality→ 310-320 mOsm/L

BTF 2007

The Brain Trauma Foundation Guidelines

Indications for Intracranial Pressure Monitoring

Hypotension and ⇧ ICP –leading cause of death in severe TBI

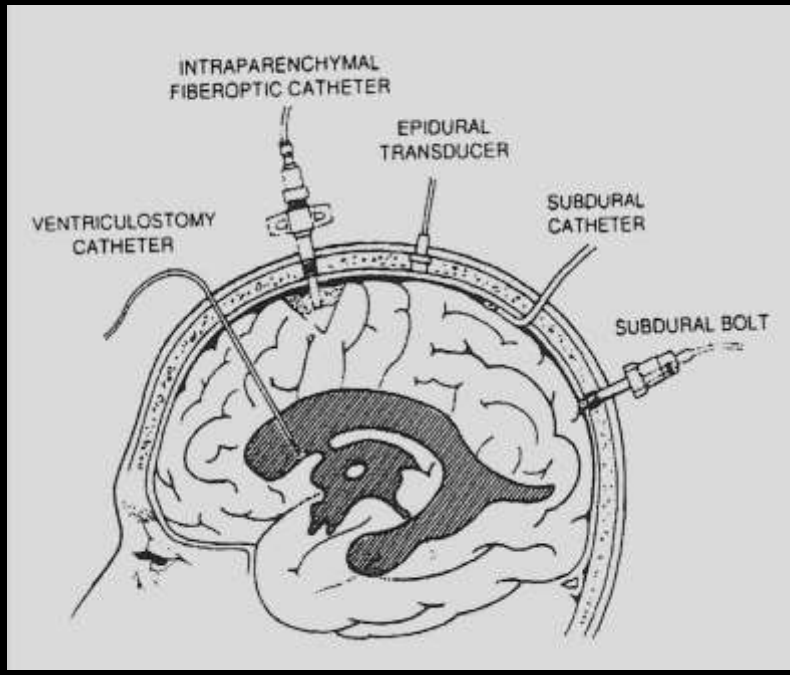
The only way to reliably determine CPP and cerebral hypoperfusion is via continuous ICP and BP monitoring

- Predict outcome & worsening pathology
- Calculate & manage CPP
- Therapeutic CSF drainage w/Ventricular ICP monitoring
- Restrict potential deleterious ICP reduction therapies

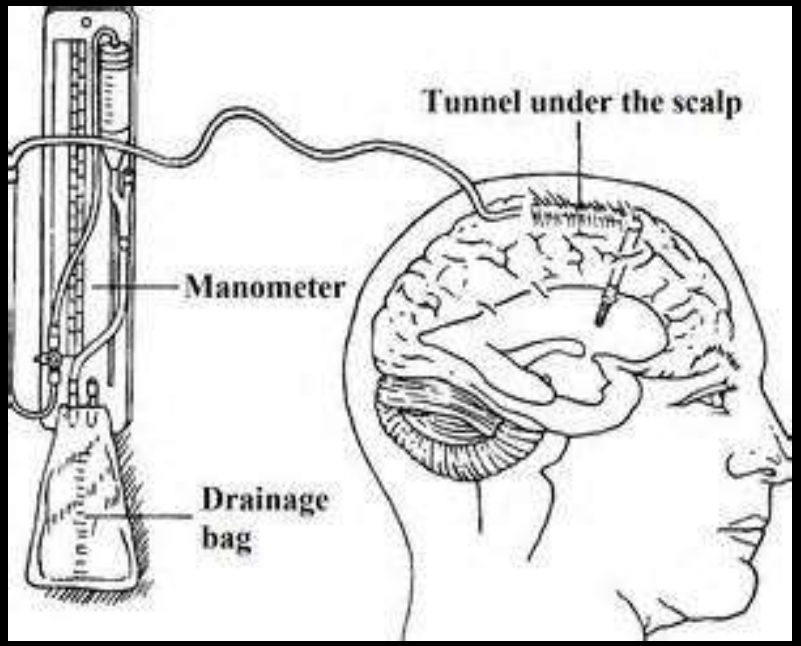
The Brain Trauma Foundation Guidelines

Intracranial Pressure Monitoring Technology

- Ventricular Catheter
  - Accurate
  - Low cost
  - Reliable
  - Recalibrated *in situ*
- Parenchymal ICP Monitors
  - Cannot be recalibrated
- Subarachnoid, Subdural, and Epidural
  - Less accurate



EVD Placement



Placed via bur hole

Frontal horn of Lateral Vent

Right sided

Patient lying at 30 degrees

External auditory meatus—
Horizontal plane as Foramen of Monro

Landmark for zeroing transducer of ICP monitor

Adjust drain height

ICP Monitoring

- **Aim of intervention is ICP control**
  - Drainage guided by effect on ICP
  - 10-15 ml per hour reasonable
- **Removal of EVD**
  - Normal ICP for 48-72 hours AFTER Withdrawal of therapy
  - Prior to removal, clamp EVD for 12-24 hours
  - Assess neuro status

Li, Timofeev, Czosnyka, & Hutchinson 2010

Complications of ICP Monitoring

- Infection
- Hemorrhage
- Malfunction
- Obstruction
- Malposition

Complications causing patient morbidity are rare

BTF 2007

The Brain Trauma Foundation Guidelines

ICP Thresholds

20-25 mm/Hg is the UPPER threshold above which treatment to lower ICP should be initiated

- Initiate treatment when ICP >20mm/Hg
- Guide patient management/treatment
  - ICP values
  - Clinical Findings
  - Brain CT findings

Brain herniation can occur at ICP <20-25 mm/Hg

The Brain Trauma Foundation Guidelines

Cerebral Perfusion Thresholds

CPP= MAP - ICP

- CPP Range 50-70 mm/Hg
  - Intact pressure autoregulation tolerate higher CPP
- Avoid CPP <50 mm/Hg
- Avoid aggressive attempts to keep CPP >70 mm/Hg with fluids and pressors
  - ARDS

The Brain Trauma Foundation Guidelines

Brain Oxygen Monitoring and Thresholds

Treatment Thresholds

- **SjVO2→** Jugular Venous Saturation <50%
  Global measurement of O_2
- **$P_{br}O_2$→** Brain Tissue Oxygen Tension <15 mm/Hg
  Local measurement of O_2

Mortality rates are HIGHER in those with episodes of desaturation

SjVO2 Monitoring

- Indirectly assesses brain's ability to extract & metabolize O_2
- Normally $CMRO_2$ coupled to CBF

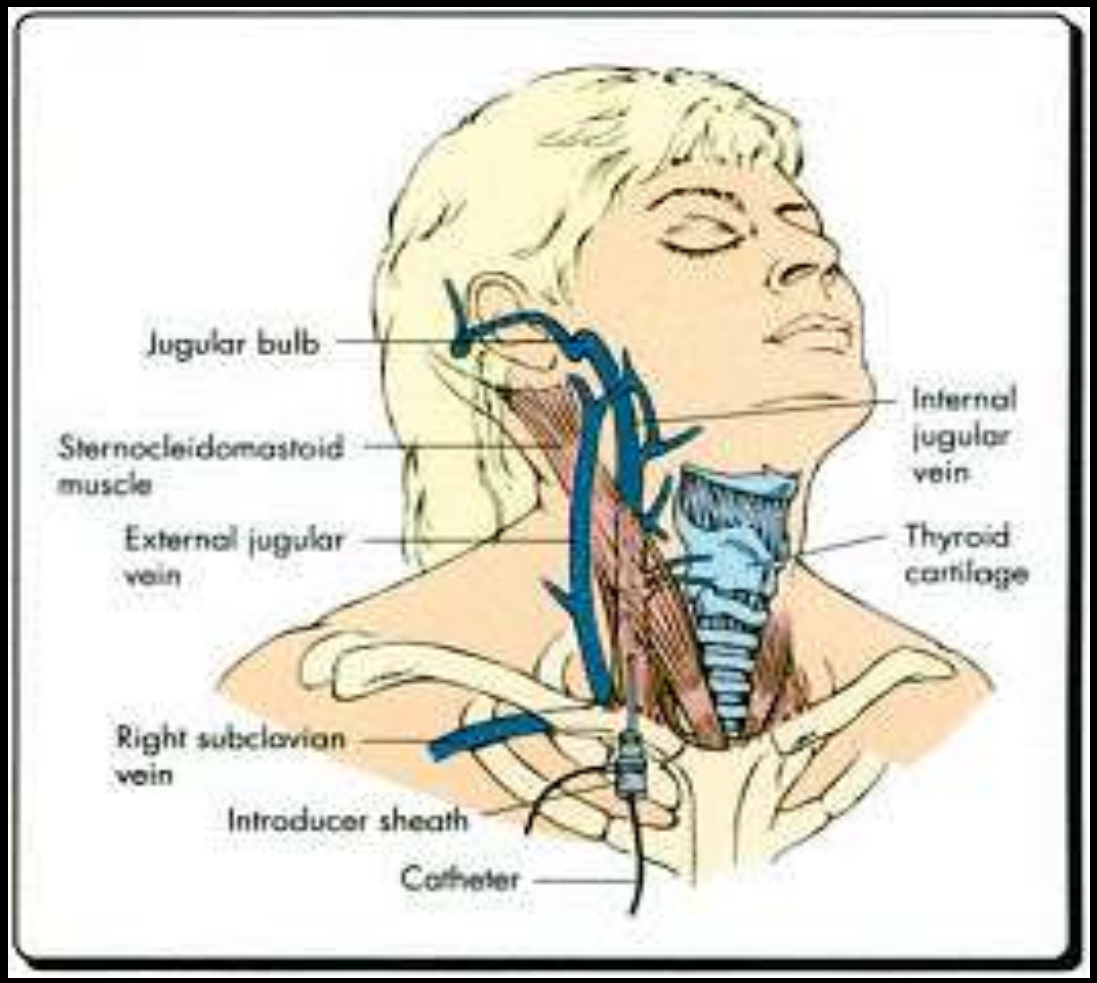
***Extraction ratio of arterial & venous blood remains constant**

- ~50% TBI patients exhibit evidence of
  - Defective cerebral autoregulation
  - Uncoupling

As long as the hemoglobin & arterial saturation remain constant the SjVO2 is an indicator of cerebral O2 demand

White & Baker 2002

S_jVO_2 Catheter Placement



$P_{br}O_2$ Monitoring

Treatment value > 15 mm/Hg

- Low PbrO2—both **Depth** & **Duration** correlated w/mortality
- 50% risk of death associated with
  - Values <15 mm/Hg
  - Lasting 4hrs or longer

***BTF Guidelines—$P_{br}O_2$ values <10-15 mm/Hg with a duration >30 minutes are associated with higher mortality**

BTF 2007

Multimodal Monitoring

Should be used TOGETHER to assess the impact of the various interventions on cerebral metabolism

- ICP
- Arterial BP
- Transcranial Doppler
- Evoked potentials
- S_jVO_2

The Brain Trauma Foundation Guidelines

Anesthetics, Analgesics, and Sedatives

- Common management strategy for ICP control
- No evidence to support their efficacy
- Have not been shown to positively affect outcome

Research Panels are looking at OUTCOME

The Brain Trauma Foundation Guidelines

Anesthetics, Analgesics, and Sedatives

- **Barbiturate**
  - Administration of high-dose to control ⇧ ICP refractory to maximal treatment

    Loading dose: 10mg/kg over 30 minutes

    5mg/kg Q1hr x3doses

    Maintenance: 1mg/kg/hr
  - Prophylactic administration of barbiturates to produce burst suppression EEG is **NOT** recommended

- **Propofol** for control of ICP
  - Not for improvement in mortality
  - High-dose can produce significant morbidity

***Do NOT EXCEED 5mg/kg/hr**

Propofol Infusion Syndrome PRIS

Common Clinical Features

- Hyperkalemia
- Hepatomegaly
- Metabolic acidosis
- Myocardial failure
- Hyperlipidemia
- Rhabdomyolysis
- Renal failure
- Death

Extreme caution must be taken when using doses >5mg/kg/hr OR ANY dose exceeding 48hr in critically ill adults

BTF 2007

Why the Increased Risk of PRIS in Acute Neurological Injury?

Negative inotropic effect creates a VISCIOUS CYCLE

- Catecholamines ↑ Cardiac output
- ↓ Propofol concentration ↑ clearance & first pass effect
- ↓ effect of propofol—reversal of anesthesia
- Propofol can depresses cardiac function→ β-receptor antagonism
- ↑ catecholamine requirements

Sabsovich, Rehman, Yunen, & Coritsidis 2007

Dosing Regimens for Analgesics & Sedatives

- Morphine 4mg/Hr continuous infusion (titrate)
- Midazolam 2mg test dose
  2-4mg/Hr continuous infusion
- Fentanyl 2mcg/kg test dose
  2-5mcg/kg/Hr
- Sufentanil 10-30mcg test dose
  0.05-2mcg/kg continuous infusion
- Propofol 0.5mcg/kg test dose
  20-75mcg/kg/min
  *Do NOT EXCEED 5mg/kg/hr

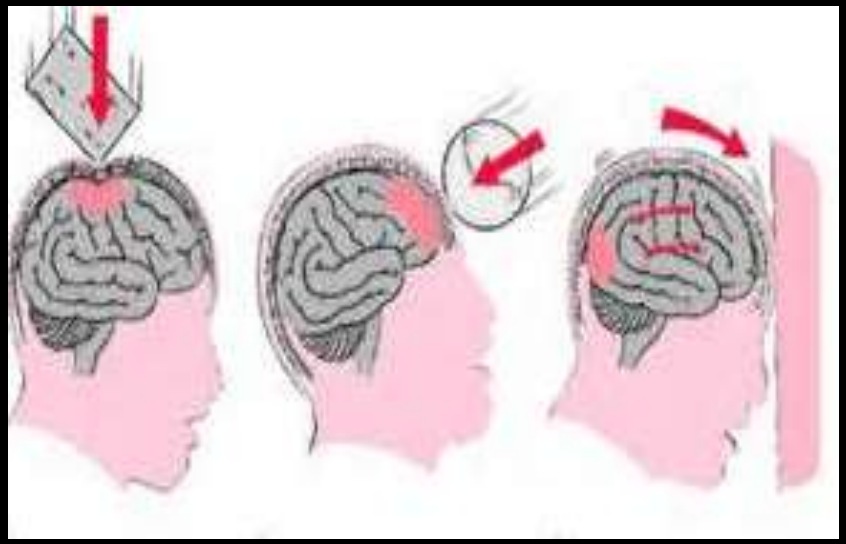
BTF 2007

Sodium & Water Balance After TBI

What Causes Electrolyte & Fluid imbalance in TBI?

Secondary Injuries r/t Force of the impact

- Cerebral edema
- Injury to hypothalamus
- Injury to pituitary gland



Hypothalamic-pituitary Dysfunction

3 common electrolyte imbalances

- Central Neurogenic Diabetes Insipidus—CNDI
  - Associated w/HYPERnatremia
- Syndrome of Inappropriate Secretion of Antidiuretic Hormone—SIADH

and

- Cerebral Salt-Wasting Syndrome—CSWS

*Associated w/HYPOnatremia

John & Day 2012

ADH

- **Osmoregulation**—maintain water balance
  - Serum Na^+ osmolality 280-295 mOsm/kg
  - Serum Os <280 ADH not secreted
  - Serum Os >295 ADH is secreted
- **Baroregulation**—changes in BV & BP
  - Located in chest, left atrium, aortic arch, carotid sinuses
  - Transmitted via vagus & glossopharyngeal nerves
  - ↑ BV & BP = ↓ ADH secretion

HYPOtension & HYPOvolemia are common in TBI
ADH secretion is INCREASED

John & Day 2012

Central Neurogenic DI

Decreased ADH secretion & Hypernatremia

- Damage to posterior pituitary—ADH is stored & secreted
- Associated with:
  - Neurosurgery & Tumors
  - Increased ICP
  - Brain death
  - CNS infections—encephalitis & meningitis
- CNDI occurs in 16% of TBI patients
- Occurs ~5-10 days after trauma

John & Day 2012

CNDI
Signs & Symptoms

- Polyuria →250 ml/hr
  *Urine specific gravity <1.005
  *Urine Os <200 mOsm/kg
- Polydipsia
- Hypovolemia
- Hypernatremia
  * Na^+ >145 mEq/L
  * Serum Os >295 mOsm/kg
- Lose 3% to 5% of body weight

*** How CNDI is diagnosed in TBI patient**

John & Day 2012

Treatment Options
Central Neurogenic DI

- Fluid replacement with 0.45% NSS
- Desmopressin

  Intranasally—5 to 2 mcg/day divided doses

  Parenterally—5 to 40 mcg/day divided doses
- Vasopressin

  Intravenous—0.5 to 2 U Q3hrs

  If U/O is >300mL/hr for 2 consecutive hrs

  Infusion– 0.2 to 0.9 U/min

Neurogenic DI
NOT
Nephrogenic DI

Pharmacology
DDAVP 4mcg/ml

- Neurogenic DI—lack of ADH
  - DDAVP is synthetic replacement—of natural hormone arginine vasopressin
  - Decreased vasopressor action
- 1 mcg DDVAP = 4 IU
- Injection is 10x the ADH effect vs. intranasal
- Mixed by pharmacy & sent "on call" r/t stability
- Administered over 10-15 minutes

Mayoclinic.com

SIADH

Increased ADH secretion & Dilutional Hyponatremia

- Renal reabsorption & water retention
- Concentrated urine
- Hyponatremia common after TBI
  - Affects ~33% of head injury patients
- Causes of SIADH in TBI
  - Traumatic Subarachnoid hemorrhage
  - Increased ICP
  - Damage to hypothalamic-pituitary region
- Body weight increase of 5% to 10%

John & Day 2012

SIADH
Signs & Symptoms

- Decreased U/O—400 to 500 ml/24 hrs
- Serum Na^+ level— <135 mEq/L
  - Seizures Na^+ <120 mEq/L
- Serum Os— <275 mOsm/L
- ↑ Na^+ in urine— >25mEq/L
- Urine osmolality > Serum osmolality

Headache, NV, fatigue, lethargy, confusion, & muscle twitching

John & Day 2012

Treatment Options
SIADH

- Fluid restriction— <1000 mL/24hr
- Slow Na^+ replacement with 3% NSS
  - Too rapid—Central Pontine Myelinolysis
  - Recommend ↑ Na^+ by 10 to 20 mEq/d
- Diuretics
- Demeclocycline hydrochloride—suppresses ADH activity
- Lithium carbonate—inhibits renal response to ADH

John & Day 2012

Cerebral Salt-Wasting Syndrome

Elevated ADH Levels & True Hyponatremia

- Lose Na^+ and ECF—plasma volume decreases
- Decrease in body weight
- Pathophysiology unclear—primary mechanism renal loss Na^+
- Occurs most often in:
  - Stroke
  - Intracerebral hemorrhage
  - Neuro surgery

***Can develop in TBI patients with ⇧ ICP**

John & Day 2012

CSWS
Signs & Symptoms

- Headache
- Increased thirst
- Dehydration
- Weight loss
- Tachycardia
- Hypotension
- Lethargy & ↓ LOC
- Seizures & coma

*Primary distinction between CSWS & SIADH volume status

CSWS→ Volume Depletion
SIADH→ Volume Expansion

John & Day 2012

Treatment Options
Cerebral Salt-Wasting Syndrome

- Replace fluids with physiologic NSS
- IV replacement with 3% NSS
- If taking PO—oral salt tablet supplements
- Fluid restriction is contraindicated
  - Cerebral vasospasm
  - Cerebral ischemia & infarction

John & Day 2012

Summary

- Neurological damage does not occur solely with the primary injury. We need to recognize & manage the secondary injuries.
- Maintain adequate cerebral blood flow, cerebral perfusion & oxygenation while the brain recovers.
- Management of ⇧ICP begins with anticipating it's development & having treatment protocols in place.
- Be aware of the electrolyte imbalances that can occur with TBI and correctly manage the cause of hyponatremia.

Thank You

References

- U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES Centers for Disease Control and Prevention National Center for Injury Prevention and Control. Traumatic Brain Injury in the Unites States, Emergency Department Visits, Hospitalizations, and Deaths 2002-2006. Accessed April 21,2013.

- The Brain Trauma Foundation. Guidelines for the Management of Severe Traumatic Brain Injury 3rd ed. The American Association of Neurological Surgeons & The Congress of Neurological Surgeons. 2007. Braintrauma.org

- Li LM, et al. The Surgical Approach to the Management of Increased Intracranial Pressure After Traumatic Brain Injury. ***Anesthesia-Analgesia.org.*** 2010;111(3):736-748.

- White H, Venkatesh B. Cerebral Perfusion Pressure in Neurotrauma: A Review. ***Anesthesia & Analgesia.*** 2008;107(3):979-987.

- Drummond JC, Englander RN, Gallo CJ. Cerebral Ischemia as an Apparent Complication of Anterior Cervical Discectomy in a Patient with an Incomplete Circle of Willis. ***Anesthesia & Analgesia.*** 2006;102:896-899.

- White H, Baker A. Continuous Jugular Venous Oximetry in the Neuro-Intensive Care Unit, a Brief Review. ***Canadian Journal of Anesthesia.*** 2002;49(6):623-629
- Barash PG, Cullen BF, Stoelting RK. Clinical Aesthesia 5th ed. 2006
- Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology 4th ed. 2006
- Nagelhout JJ, Plaus KL. Nurse Anesthesia 4th ed. 2010
- John CA, Day MW. Central Neurogenic Diabetes Insipidus, Syndrome of Inappropriate Secretion of Antidiuretic Hormone, and Cerebral salt-wasting Syndrome in Traumatic Brain Injury. ***Critical Care Nurse.*** 2012;32(2):e1-e8. ccn.aacnjournals.org
- Sabsovich I, Rehman Z, Yunen J, Coritsidis G. Propofol Infusion Syndrome: A Case of Increasing Morbidity With Traumatic Brain Injury. ***American Journal of Critical Care.*** 2007;16:82-85