Neuro Anesthesia for Traumatic Brain Injury

A Review of the Basics

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Introduction

CDC

- 1.7 Million sustained TBI
- 52,000 Deaths
- 275,000 Hospitalizations
- 80% treated & released from ER (~1.3 million)
- Those ≥ 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
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 $\rightarrow$ 40-50ml/100g/min
Average adult $\sim$ 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 & $\uparrow$ BF
Manipulating CO₂

↑ CO₂ causes vasodilation & ↑ Blood Flow

↑ CO₂ from 40 to 80 mm/Hg—DOUBLE BF
↓ CO₂ from 40 to 20 mm/Hg—HALVES BF

*Changes are transient lasting ~6-8 hours. BF returns to normal even if we attempt to maintain the CO₂ levels. HCO₃⁻ level of brain ECF returns the pH to normal
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
<table>
<thead>
<tr>
<th>Agent</th>
<th>CMR</th>
<th>CBF</th>
<th>CSF Production</th>
<th>CSF Absorption</th>
<th>CBV</th>
<th>ICP</th>
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<td>Ketamine</td>
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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
Sinuses Drain into the Internal Jugular Vein
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**
- $\text{Na}^+ \ 141 \text{ mEq/l}$
- $\text{K}^+ \ 2.9 \text{ mEq/l}$
- $\text{Ca}^{2+} \ 2.5 \text{ mEq/l}$
- $\text{Mg}^{2+} \ 2.4 \text{ mEq/l}$
- $\text{Cl}^- \ 124 \text{ mEq/l}$
- $\text{HCO}_3^- \ 21 \text{ mEq/l}$
- PRO \ 28 mg/100mL
- Glu \ 61 mg/100mL

pH 7.31

Barash 2006
Pattern of CSF Flow

- Lateral Ventricles
- Foramen Of Monro
- 3rd Ventricle
- Lushka & Magendie
- 4th Ventricle
- *Aqueduct of Sylvius
- Subarachnoid Space Spinal Cord
- Brain
- Arachnoid Villi
Intracranial Pressure

- Normal <10 mm/Hg
- ICP >15 mm/Hg \( \rightarrow \) 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 \( \rightarrow \) 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
<table>
<thead>
<tr>
<th>BEHAVIOR</th>
<th>RESPONSE</th>
<th>SCORE</th>
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<tbody>
<tr>
<td>Eye Opening Response</td>
<td>Spontaneous</td>
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<tr>
<td></td>
<td>To speech</td>
<td>3</td>
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<tr>
<td></td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No Response</td>
<td>1</td>
</tr>
<tr>
<td>Best Verbal Response</td>
<td>Oriented x3</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td>Best Motor Response</td>
<td>Obeys Commands</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Moves to localized pain</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Flexion/withdrawal from pain</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Abnormal flexion Decorticate</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Abnormal extension Decerebrate</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
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<tr>
<td>Total Score</td>
<td>Best Response</td>
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<td></td>
<td><strong>Comatose</strong></td>
<td>≤8</td>
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<tr>
<td></td>
<td>Unresponsive</td>
<td>3</td>
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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 24 hours 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 thrombosis

Posttraumatic seizure
↑ Intrathoracic pressure
Hyperthermia

Li, Timofeev, Czosnyka, & Hutchinson 2010
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

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

*No definitive CT feature indicates ↑ ICP

Li, timofeev, Czosnyka, & Hutchinson 2010
Mainstay of ICP Management
University of Cambridge/Addenbrookes Hospital, United Kingdom

Medical Management with Protocols

<table>
<thead>
<tr>
<th>Medical Management</th>
<th>Protocol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head elevation</td>
<td>10-15 degrees</td>
</tr>
<tr>
<td>Adequate oxygenation</td>
<td>Sat ≥ 97%</td>
</tr>
<tr>
<td>Fluid resuscitation</td>
<td>CVP 6-10</td>
</tr>
<tr>
<td>Sedation</td>
<td>Propofol, versed, fentanyl</td>
</tr>
<tr>
<td>Muscle relaxation</td>
<td>Atracurium 0.5mg/kg/hr</td>
</tr>
<tr>
<td>Mild hyperventilation</td>
<td>≤ 37°C</td>
</tr>
<tr>
<td>Cooling</td>
<td></td>
</tr>
</tbody>
</table>

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

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

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

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 SjVO₂ catheter ≥ 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

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 >90 mm/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 ↑ O₂ 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
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
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
Treatment Thresholds

- **SjVO₂** → Jugular Venous Saturation <50%
  Global measurement of O₂

- **P_{br}O₂** → Brain Tissue Oxygen Tension <15 mm/Hg
  Local measurement of O₂

Mortality rates are HIGHER in those with episodes of desaturation
SjVO2 Monitoring

- Indirectly assesses brain’s ability to extract & metabolize O₂
- Normally CMRO₂ 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
$S_j \text{VO}_2$ Catheter Placement
**P_{br}O_{2} Monitoring**

- 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

**Treatment value > 15 mm/Hg**
Multimodal Monitoring

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

- ICP
- Arterial BP
- Transcranial Doppler
- Evoked potentials
- $S_j VO_2$
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
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 $\uparrow$ Cardiac output
- $\downarrow$ Propofol concentration $\uparrow$ clearance & first pass effect
- $\downarrow$ effect of propofol—reversal of anesthesia
- Propofol can depresses cardiac function $\rightarrow$ $\beta$-receptor antagonism
- $\uparrow$ 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
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 & HYPOvoleemia** are common in TBI
ADH secretion is INCREASED
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
CNDI
Signs & Symptoms

• Polyuria → 250 ml/hr
  * Urine specific gravity < 1.005
  * Urine Osm < 200 mOsm/kg

• Polydipsia

• Hypovolemia

• Hypernatremia
  * Na⁺ > 145 mEq/L
  * Serum Osm > 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
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
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
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

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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

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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

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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


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