ICP
A Stepwise Protocol

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**ICP: Basic Concepts**

- **Monroe-Kellie doctrine:** skull = fixed volume
- 3 components of intracranial volume
- Normal ICP
  - $\leq 20 \text{ cm H20}$
  - $\leq 15 \text{ mm Hg}$
<table>
<thead>
<tr>
<th>Causes of Increased ICP:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Space Occupying Lesion</strong></td>
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<tr>
<td><strong>Increased CSF</strong></td>
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<tr>
<td><strong>Inc. Blood Volume</strong> (Vasogenic edema)</td>
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<tr>
<td><strong>Inc. Brain Volume</strong> (Cytotoxic edema)</td>
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</table>
Methods to Reduce Elevated ICP

• Remove Mass Lesion
  - Surgical Evacuation

• Reduce CSF Volume
  - Ventricular Drainage

• Reduce Cerebral Blood Volume
  - Hyperventilation, Barbiturates, Hypothermia

• Reduce Parenchymal Volume
  - Osmotic Diuretics
  (Mannitol, Hypertonic Saline)
Percussion wave
Dicrotic wave
Tidal wave

Normal ICP; normal compliance

Elevated ICP; reduced compliance

20 mm Hg

1 sec
ICP/CPP Management

Cerebral Perfusion Pressure (mm Hg)

Cerebral Blood Flow (ml/100 g/min)

Stephan A. Mayer, MD
CPP can influence ICP when you run out of room.
Outcome after Acute Ischemic Stroke by Admission Blood Pressure

Figure 5–5. Two classic A waves are shown (open arrows). Note that when the ICP falls after the A wave (closed arrow), it does not return to the baseline preceding the first wave.
PLATEAU (LUNDBERG A) WAVES
Indications for ICP Monitoring

- Coma (Glasgow Coma Scale score ≤8)
- CT evidence of intracranial mass effect
  - Extra-axial mass lesion
  - Midline shift
  - Effacement of basal cisterns
  - Exception: severe TBI with motor posturing
- Prognosis is such that aggressive ICU care is warranted
Clinical Signs

• Increased ICP
  – Depressed level of consciousness
  – Pressor response
  – Projectile vomiting
  – CN 6 palsies

• Brainstem herniation
  – CN 3 palsey
  – Motor posturing
  – Lower extremity rigidity
  – Loss of lateral EOMs
  – Hyperventilation
Parenchymal Micosensor

Ventricular catheter

Epidural Monitor

Richmond Bolt
ICP/CPP Treatment Thresholds

Guideline

• ICP treatment should be initiated at an upper threshold of 20 mm Hg.

Option

• Cerebral Perfusion Pressure should be maintained at a minimum of 60 mm Hg.
ICP: General Care Issues

- Elevate head of bed 30°
- **Use only isotonic fluids (0.9% saline)**
- Control fevers aggressively
- Seizure prophylaxis
- No routine steroids use

**Option:** 3% saline or mannitol for target osmolality of 300-320 mOsm/L
Emergency Treatment of Increased ICP

• Un-monitored patient with clinical signs of herniation
  – Elevate head of bed 30°
  – Normal saline 100 ml/hr
  – Intubate and hyperventilate (pCO2 30 mm Hg)
  – Mannitol 20% 1.0 to 1.5 g/kg rapid IV infusion
  – Foley catheter
  – CT scan and neurosurgical evaluation
Critical Pathway for Treatment of Intracranial Hypertension in the Severe Head Injury Patient
(Treatment Option)

- **Insert ICP Monitor**
- **Maintain CPP ≈ 70 mmHg**
- **Intracranial Hypertension?**
  - **YES**
  - **Ventricular Drainage (if available)**
  - **YES**
  - **Intracranial Hypertension?**
    - **YES**
    - **Mannitol (0.25 - 1.0 g/kg IV)**
    - **NO**
    - **Hyperventilation to PaCO2 30 - 35 mmHg**
    - **YES**
    - **Intracranial Hypertension?**
      - **YES**
      - **Repeat Mannitol if Serum Osmolarity < 320 mOsm/L & Pt euvolemic**
      - **NO**
      - **Carefully Withdraw ICP Treatment**
    - **NO**
  - **NO**

- **Other Second Tier Therapies**
- **High Dose Barbiturate therapy**
- **Second Tier Therapy**
  - **Hyperventilation to PaCO2 < 30 mmHg**
  - **Monitoring SjO2, AVDO2, and/or CBF Recommended**
Columbia Stepwise ICP Protocol

1. SURGICAL DECOMPRESSION
2. SEDATION
3. CPP OPTIMIZATION
4. OSMOTHERAPY
5. HYPERVENTILATION
6. PENTOBARBITAL
7. HYPOTHERMIA
Revised Columbia Stepwise ICP Protocol

1. **SURGICAL DECOMPRESSION**
2. **SEDATION**
3. **CPP OPTIMIZATION**
4. **OSMOTHERAPY**
5. **HYPERVENTILATION**
6. **HYPOTHERMIA**
7. **PENTOBARBITAL**
Ventricular Drainage

3-way stopcock turned off to drainage system

transpac positioned at ear level

drainage bag

100cc syringe of sterile water

LEVEL
Aneurysmal SAH with associated subdural hematoma and bilateral motor posturing
ICU Intravenous sedation

• Goal is reversibility to allow repeated neurologic assessment
• Alternatives (in intubated pts):
  – Fentanyl or Remifentanil
  – Midazolam
  – Propofol
    » Ultrashort acting
    » Allows “wake-up” in 5-15 mins
    » Reduces ICP, CMRO$_2$
    » Drawbacks: hypotension, infection
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CPP OPTIMIZATION: Dopamine infusion resulting in increased MAP and CPP, and decreased ICP.
**Lund protocol**

- Reduce CMRO$_2$ with sedation
  - Miazolam, thiopental, fentanyl
- Reduce BP and capillary hydrostatic pressure (CPP 50-70 mm Hg)
  - IV Metoprolol (.2 mg/kg/hr) and clonidine (.5 mcg/kg prn)
- Dihydroergotamine .1-.8 mcg/kg/hr
- Maintain normal hematocrit, CVP, and albumin levels
MMM: “Imaging” Vasodilatory Cascade Physiology

MMM: “Imaging” Perfusion Pressure Breakthrough Physiology

Brain tissue oxygen guided treatment supplementing ICP/CPP therapy after traumatic brain injury

- 93 severe TBI patients
  - 40 treated for CPP > 70 alone (1993-96)
  - 53 treated for CPP > 70 + PbrO2 > 10 mm Hg as needed (1997-00)
- Boosting CPP lead to less cerebral hypoxia
- Larger studies needed to see if this improve outcomes

Meixensberger JNNP 2003;74:760
Osmotherapy

- **Mannitol**
  - 0.25 to 1.5 g/kg IV wide open
  - Dose up to Q1H on an as-needed basis
  - Mechanisms:
    » Acute dehydrating effect (osmotic gradient across BBB)
    » Secondary hyperosmolality (diuretic effect)
    » Reflex vasoconstriction (viscosity effect)

- **Hypertonic Saline**
  - Varying concentrations: 3%, 7.5%, 10%, and 23.4%
  - Optimal dosing not known
30 ml 23.4% Bullets
Effect of Hypertonic Saline in CBF in SAH patients

- 10 poor grade SAH patients
- 2 mL/kg of 23.5% saline
- ICP fell 74%
- CPP rose 27%
- CBF rose 23%
- Peak effect @ 20-60 minutes
7.2% Hypertonic Saline vs 15% Mannitol Solution for Treating ICP in Neurosurgical Patients

Critical Care 2005;9:R530
Slowly raising a sodium level over days with continuous infusion hypertonic saline effectively reduces brain edema

MYTH
Changes in Sodium concentration & ICP in NICU patients treated with 3% saline solution.

Figure 1. Acute trend in mean intracranial pressure (ICP) and serum sodium concentrations in patients with head injury (top), and patients with postoperative cerebral edema (bottom). Note the reduction in intracranial pressure associated with increasing serum sodium concentration. Values are expressed as mean ± SEM. Open squares represent serum sodium; open circles represent ICP.

Evidence: 3% Saline Infusion

  - 3% saline reduced ICP and CT midline shift in patients with TBI or neoplasm
  - Effect not seen with ICH or infarction

  - 3% saline infusion reduced ICP in pediatric TBI patients
  - Effect modest: 4 mm Hg over 2 hours

3% HTS infusion has a modest and inconsistent effect on ICP
Results: HTS Use

Median (IQR) days to HTS start: 1.0 (0 – 3) days
Median (IQR) duration of HTS use: 10.5 (6 – 14) days
# Poor Grade SAH: In-Hospital Complications

<table>
<thead>
<tr>
<th></th>
<th>HTS Cases (N= 36)</th>
<th>Controls (N= 57)</th>
<th>(P)</th>
</tr>
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<tbody>
<tr>
<td><strong>Neurological</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Herniation</td>
<td>8 (23%)</td>
<td>10 (18%)</td>
<td>0.56</td>
</tr>
<tr>
<td>Infarction secondary to vasospasm</td>
<td>6 (17%)</td>
<td>13 (23%)</td>
<td>0.45</td>
</tr>
<tr>
<td>Seizures</td>
<td>10 (28%)</td>
<td>8 (14%)</td>
<td>0.11</td>
</tr>
<tr>
<td><strong>Medical</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>21 (58%)</td>
<td>14 (25%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>10 (28%)</td>
<td>3 (5%)</td>
<td>0.002</td>
</tr>
<tr>
<td>Pulmonary Edema</td>
<td>17 (47%)</td>
<td>10 (18%)</td>
<td>0.003</td>
</tr>
<tr>
<td>Congestive Heart Failure</td>
<td>7 (19%)</td>
<td>4 (7%)</td>
<td>0.07</td>
</tr>
<tr>
<td>Fever (Temp. &gt;101.5 F)</td>
<td>31 (86%)</td>
<td>47 (83%)</td>
<td>0.64</td>
</tr>
</tbody>
</table>
Hypertonic Saline: Complications

- Congestive heart failure
- Rebound brain edema
- Hypokalemia
- Hyperchloremic metabolic acidosis
  - Use 50/50 chloride/acetate solution
- Coagulopathy
- Central pontine myelinolysis
Hyperventilation

Hyperventilation is the most rapid way to reduce ICP.
Excessive hyperventilation can worsen cerebral ischemia.

Mechanism of Action:
- Hypocarbia induces serum and CSF alkalosis.
- Alkalosis induces cerebral vasoconstriction.
- Vasoconstriction reduces cerebral blood volume.

Time Course:
- ICP is reduced almost immediately.
- Peak reduction in 5-10 minutes.

Hyperventilation can have sustained effects in patients with vasodilatory vasogenic cerebral edema.
The graph illustrates the relationship between CBF (Cerebral Blood Flow), PaO₂, PaCO₂, and PRESSURE (in torr). The graph shows how CBF changes with variations in PaO₂ and PaCO₂ at different PRESSURE levels.
Hypothermia

MECHANISM OF ACTION:
PROFOUNDLY REDUCES CEREBRAL METABOLISM, AND HENCE CEREBRAL BLOOD VOLUME

TARGET
32-33°C

INDICATION
PENTOBARBITAL-REFRACTORY ICP

COMPLICATIONS:
ARRythmia AND CARDIOVASCULAR DEPRESSION
IMMUNOSUPPRESSION
COAGULOpathy
METABOLIC: SHIVERING AND REWARMING
<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>Year</th>
<th>Effect on ICP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clifton</td>
<td>42</td>
<td>1993 JNT</td>
<td>No difference compared to controls</td>
</tr>
<tr>
<td>Marion</td>
<td>43</td>
<td>1993 JNS</td>
<td>Reduced from baseline by 40%</td>
</tr>
<tr>
<td>Metz</td>
<td>10*</td>
<td>1997 JNS</td>
<td>Reduced from baseline</td>
</tr>
<tr>
<td>Marion</td>
<td>82</td>
<td>1997 NEJM</td>
<td>ICP lower (15 vs 20 mm Hg) and CPP higher; but reversed with rewarming</td>
</tr>
<tr>
<td>Jiang</td>
<td>87</td>
<td>2000 JNS</td>
<td>Reduced from baseline (29 vs 19 mm Hg) at day 7</td>
</tr>
<tr>
<td>Shiozaki</td>
<td>91</td>
<td>2001 JNS</td>
<td>No effect on concurrent therapeutic intensity for ICP management</td>
</tr>
<tr>
<td>Clifton</td>
<td>392</td>
<td>2001 NEJM</td>
<td>Reduced proportion with ICP &gt;30 mm Hg on day 2 (14% vs. 28%)</td>
</tr>
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</table>
Contemporary Management of Traumatic Intracranial Hypertension: Is There a Role for Therapeutic Hypothermia?

Matthew Schreckinger · Donald W. Marion

Table 3 Comparison of contemporary therapies for intracranial hypertension

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Total number of patients</th>
<th>Average decrease in ICP</th>
<th>Standard deviation</th>
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<tbody>
<tr>
<td>Hyperventilation</td>
<td>126</td>
<td>6.08</td>
<td>4.22</td>
</tr>
<tr>
<td>Mannitol</td>
<td>140</td>
<td>7.93</td>
<td>5.34</td>
</tr>
<tr>
<td>Barbiturates</td>
<td>167</td>
<td>8.47</td>
<td>6.71</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>367</td>
<td>9.97</td>
<td>6.66</td>
</tr>
<tr>
<td>Hypertonic saline</td>
<td>133</td>
<td>15.06</td>
<td>7.34</td>
</tr>
<tr>
<td>CSF drainage</td>
<td>72</td>
<td>15.45</td>
<td>4.67</td>
</tr>
<tr>
<td>Decompressive craniectomy</td>
<td>192</td>
<td>19.15</td>
<td>7.70</td>
</tr>
</tbody>
</table>
• **Tateshi (Neurosurgery 1998)**
  - Titrated the depth of cooling between 33-36 °C to keep ICP <20 mm Hg in 9 patients refractory to thiopental.
  - All patients responded:
    - Mean brain reduction of temperature of 2°C
    - Mean reduction of ICP from 24 to 15 mm Hg.
  - The frequency of infectious and hematologic complications in this relatively small series was still high.
High-Dose Barbituates

MECHANISM OF ACTION:
PROFOUNDLY REDUCES CEREBRAL METABOLISM, AND HENCE CEREBRAL BLOOD VOLUME

PENTOBARBITAL:
LOADING DOSE = 5-20 mg/kg IV OVER 1 mg/kg/min
MAINTAINANCE DRIP = 1-4 mg/kg/hr
GOAL: NORMAL ICP, BURST SUPPRESSION ON EEG

COMPLICATIONS:
MYOCARDIAL DEPRESSION AND HYPOTENSION
NEUROLOGICAL EXAM IS OBSCURED
ILEUS
BLOOD STREAM INFECTION
ICP CONTROL: TRAUMATIC BRAIN INJURY

- Shiozaki (J Neurosurg 1993)
  - Randomized controlled study of hypothermia (34° C) for ICP refractory to pentobarbital (N=33).
  - Hypothermia resulted in:
    » Lower ICP (36.9 to 26.5 mm Hg)
    » Increased CPP
    » Reduced CBF and CMRO2
    » Reduced arteriojugular venous oxygen differences
  - Survival was 50% in hypothermia patients compared to 18% in the control group (P<.05).
Complications of pentobarbital

- Hypotension
- Immunosuppression
- An extra 4 weeks in coma
- ICU neuromyopathy
Re-Revised Columbia Stepwise ICP Protocol

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ICP Control in Action
Neuro-ICU: July 4, 1995

- Healthy 35 yr old woman @ 34 wks gestation with pre-eclampsia and HELLP syndrome
- July 31: Rx Mg sulfate and emergent delivery
- Aug 1: At 8:00 AM, sudden HA and lethargy, progressing rapidly to deep coma
- CT SCAN: diffuse cerebral edema
Neuro-ICU: On Arrival

- **2:00 PM**: Arrival in deep coma with intact brainstem reflexes
- **4:00 PM**: BP 100/65, Rx dopamine
- **7:00 PM**: ICP monitor inserted, ICP 22 and CPP 76 mmHg
- **11:00 PM**: ICP 38, CPP 55 mmHg, mannitol started...
Early Day 2: Massive Elevation of ICP

mmHG

ICP

CPP

MANNITOL 70 g/hr

PENTOBARB 2-4 mg/kg/h
Neuro-ICU: Day 2

- **EXAM**: Sluggish pupils and corneals, no EOMs to ice water, no withdrawal to deep pain
- **EEG**: Flatline
- **TCD**: Hi velocity (90-100 cm/s), low PI
- **LABS**: Osm 401, Hct 17%, Na⁺ 121

Neuro-ICU: Day 3

- Oliguric renal failure 2° mannitol
- Hemodialysis x 1 = good response
Subsequent Course

- **Day 4**: GI bleed (Tx 5 units), LLL pneumonia with large Aa gradient
- **Day 5**: Post ATN diuretic phase, increased Na⁺ 152
- **Day 6**: ICP = 6 mmHg, light coma
- **Day 7**: ICP monitor d/c’ ed
- **Day 8**: HR 145, new inverted T waves
- **Day 12**: LUE DVT 2° central line (positive lupus A/C noted)
Later Hospital Course

- **Day 14**: Looks at a voice, grimaces and frowns, bibrachial paresis, central hyperventilation
- **Day 21**: Rarely obeys commands
- **Day 27**: EF 30% on echo
- **Day 30**: Consistently obeys commands, anarthric with pseudobulbar affect
- **Day 50**: Talking, laughing, writing messages, walking with assistance
3 Month Outcome

- Home with family and children
- Functionally independent, doing light housework, in outpatient rehab
- Mild deficits in memory, concentration, and visual-spatial function
- Mild spastic left hemiparesis
- Cerebellar dysarthria
- Mild dysmetria