Feeling the Pressure

(\textit{as in Intracranial Pressure…})

Liz Kim, MSN, ACNS-BC, FAHA
Advanced Practice Provider – Neurocritical Care
Stanford Health Care

May 15, 2017
World Live Neurovascular Conference
Disclosures

- Financial Disclosures:
  - None relevant to the clinical content being presented
  - Intermittent Stroke reviewer for The Joint Commission

- Unapproved/Usage Disclosure:
  - None
Outline

- Intracranial hemodynamics
  - CBF – Cerebral blood flow
  - CPP – Cerebral perfusion pressure
  - ICP – Intracranial pressure

- Causes of increased ICP

- Signs and Symptoms of ICP

- Treatment
  - Emergency Neurological Life Support:
    - Intracranial Hypertension and Herniation Protocol
  - Hyperosmolar Therapy
  - Decompressive Craniectomy for Malignant MCA Infarcts
Intracranial Pressure

- Skull is a fixed volume vault; skull by nature is non-compliant

- ICP = sum of 3 components to total a fixed volume in the cranial vault

- Non-compressible, but partially displaceable
Intracranial Pressure

**Monro-Kellie Doctrine**

*Sum* of the intracranial volumes of blood, brain, CSF, and other components is *constant*, and that an *increase in any one of these must be offset by an equal decrease in another*, or else pressure increases.

**Normal:**

5 - 15 mmHg

**Intracranial hypertension:**

ICP > 20mmHg sustained for more than 5 minutes
Cerebral perfusion pressure (CPP)
Difference between the force driving blood into the brain and the force resisting movement of blood into the brain

CPP = MAP - ICP

Normal: 70-100 mmHg
< 50 mmHg: Cerebral ischemia
< 30 mmHg: Brain death

MAP
CPP
ICP
Cerebral Blood Flow (CBF)
Amount of blood passing through 100g of brain tissue in 1 minute

CBF =

- Cerebral perfusion pressure
- Cerebral vascular resistance

Average: 50
Ischemia: < 18 – 20
Tissue death: < 8 – 10
Hyperemia: > 55 – 60

750ml/minute
~15% of cardiac output
50ml/min per 100g of brain tissue

Autoregulatory mechanisms maintain a relatively constant CBF, despite changes in systemic parameters.
Cerebral Dynamics: Cerebral Blood Flow

Tameen et al., 2013
## Causes of Increase ICP

<table>
<thead>
<tr>
<th>Intracranial (primary)</th>
<th>Extracranial (secondary)</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tumor</td>
<td>Airway obstruction</td>
<td>Mass lesion (hematoma edema)</td>
</tr>
<tr>
<td>Trauma (Epidural &amp; Subdural hematomas &amp; contusions)</td>
<td>Hypoxia or hypercarbia</td>
<td>Increased cerebral blood volume (vasodilation)</td>
</tr>
<tr>
<td>Non-traumatic intracranial hemorrhages</td>
<td>Posture (head rotation)</td>
<td>Disturbances of CSF</td>
</tr>
<tr>
<td>Ischemic stroke</td>
<td>Hyperpyrexia</td>
<td></td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>Seizures</td>
<td></td>
</tr>
<tr>
<td>Idiopathic or benign intracranial hypertension</td>
<td>Drug and metabolic derangements</td>
<td></td>
</tr>
<tr>
<td>Other (eg, pseudotumor cerebri, pneumoencephalus, abscesses, cysts)</td>
<td>Others (eg, high-altitude cerebral edema, hepatic failure)</td>
<td></td>
</tr>
</tbody>
</table>

*Rangel-Castello, et al., 2008*
INCREASED INTRACRANIAL PRESSURE

- Changes in LOC
- Eyes
  - Papilledema
  - Pupillary Changes
  - Impaired Eye Movement
- Posturing
  - Decerebrate
  - Decorticate
  - Flaccid
- Decreased Motor Function
  - Change in Motor Ability
  - Posturing
- Headache
- Seizures
  - Impaired Sensory & Motor Function
- Changes in Vital Signs:
  - Cushing’s Triad:
    - ↑ Systolic B/P
    - ↓ Pulse
    - Altered Resp Pattern
- Vomiting
- Changes in Speech

© Infants:
- Bulging Fontanels
- Cranial Suture Separation
- ↑ Head Circumference
- High Pitched Cry
## Signs of Increased ICP

<table>
<thead>
<tr>
<th>“EARLY”</th>
<th>“LATE”</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>Changes in level of consciousness or ↓ GCS or FOUR Score ≥ 2 points</td>
</tr>
<tr>
<td>Irritability</td>
<td>Ipsilesional change in pupillary size, shape and light-responsiveness</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Contralesional hemiparesis (new or worsening)</td>
</tr>
<tr>
<td>Photophobia,</td>
<td>Contralesional change in pupillary size and ipsilesional hemiparesis</td>
</tr>
<tr>
<td>nystagmus, diploplia</td>
<td>(Kernohan’s phenomenon)</td>
</tr>
<tr>
<td>Lethargy</td>
<td>Cushing’s triad: ↑ SBP (widened pulse pressure), bradycardia, irregular</td>
</tr>
<tr>
<td>Seizure</td>
<td>Respiration</td>
</tr>
</tbody>
</table>
How do we measure ICP?

Copyright © McGraw-Hill Education. All rights reserved.

http://accessmedicine.mhmedical.com/data/books/1340/hall4_ch86_fig-86-16.png
Herniation

- Increased intracranial compartmental pressure causing tissue shifts that compress or displace the brainstem, cranial nerves, or cerebral vasculature

Tameen et al., 2013
Treatment of Intracranial Pressure

- Think BIG – heterogeneous population
- Step wide approach
ENLS: Tier 0 – Standard Measures

ABCs
(avoid hypotension and hypoxia)

Head of bed elevated > 30 degrees and midline
(increase venous return)

Minimize stimuli or adequately sedate and provide pain relief

Normothermia, normotension, euvolemia, normonatremia, euglycemic

Treat vasogenic edema (steroids for tumors)

Intracranial Hypertension or Herniation

Tier 0: Standard Measures

Tier 1: ↓PaCO₂, Mannitol, NaCl, CSF Drainage
Consider Additional Monitoring

Tier 2: NaCl, Propofol

Head CT
Decompression

Tier 3: Pentobarbital, Hypothermia

Revise ICP/MAP Goals
Hyperosmolar therapy:
- Mannitol 0.5-1g/kg (Serum osmolality q4-6 hours)
- Hypertonic saline (Serum Na levels q4-6 hours)

Placement of external ventricular drain (EVD)
- Drain for acute rises in ICP

Hyperventilation:
Consider BRIEF (<2 hours) (PaCO2 30-35 mmHg) as temporizing measure

Other:
Brain tissue oxygenation, jugular bulb venous oximetry, cerebral microdialysis
Hyperosmolar Therapy

Mannitol:
- Osmotic diuretic, drawing water out of edematous brain tissue
- Typically given as bolus of 0.25-1g/kg
- Caution/Contraindicated: hypovolemia, hypotension, renal failure, pulmonary edema
- Over time “opens” blood brain barrier and mannitol crosses, losing efficacy
- Monitoring: Serum osmolality q4-6 hours (< 320)

Hypertonic saline:
- Causes an osmotic gradient, drawing water out of edematous brain tissue
- Can be given as bolus or infusion, ranging from 3-23.4%
- Can result in plasma volume expansion (increases blood pressure and CPP) – can be used with hypotension/hypovolemia
- Requires an intact blood brain barrier
- Overtime can lead to electrolyte abnormalities such as hyperchloremic acidosis
- Monitoring: Serum Na levels q4-6 hours (<160)

Which is better???
Difficult question limited by small number and size of trials. Possibly suggestion that HTS, but randomized trial needed.

In a true emergency, whichever you can obtain/administer the quickest!
ENLS: Post Tier 1

If ICP stabilized with Tier 1 → obtain a head CT

If not, move to Tier 2 → obtain head CT

Consider adjusting ICP, MAP and CPP based on clinical context
ENLS: Tier 2

Increase Na goal (~160mmol/L)

Increase sedation
Decompression

If failing medical management:

- Review surgical options
- Evacuation of mass lesion or decompression craniectomy

If the patient is ineligible for surgery or too unstable for brain imaging, move to Tier 3
Pentobarbital infusion (cEEG) 24-96 hours

Moderate hypothermia (32-34 degrees Celsius)

Hyperventilation to achieve mild to moderate hypocapnia (PaCO2 25-30mmHg)
Ideally with cerebral oxygen monitoring and for < 6 hours duration
Malignant Middle Cerebral Artery Infarct

- Distal ICA or proximal MCA trunk occlusion leading to a large MCA infarction (+/- ACA or PCA involvement) and poor collateral compensation
- Mortality of 78%, due to transtentorial herniation and brain death, range 2-5 days

*Hacke et al., 1996*
### Demographic & Clinical Predictors
**Malignant MCA Infarct**

<table>
<thead>
<tr>
<th>Predictor</th>
<th># patients</th>
<th>Odds ratio or Sens/Spec</th>
<th>Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Younger age</td>
<td>192</td>
<td>OR 0.4 95% CI 0.3-0.6</td>
<td>Jaramillo et al Neurology 2006</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Female sex</td>
<td>192</td>
<td>OR 8.2 95% CI 2.7-25.2</td>
<td>Jaramillo et al Neurology 2006</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p = 0.0003</td>
<td></td>
</tr>
<tr>
<td>NO prior infarcts</td>
<td>192</td>
<td>OR 0.2 95% CI 0.05-0.7</td>
<td>Jaramillo et al Neurology 2006</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p= 0.01</td>
<td></td>
</tr>
<tr>
<td>History of HTN</td>
<td>201</td>
<td>OR 3.0 95% CI 1.2-7.6</td>
<td>Kasner et al, Stroke 2001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p=0.02</td>
<td></td>
</tr>
<tr>
<td>History of CHF</td>
<td>201</td>
<td>OR 2.1 95% CI 1.5-3.0</td>
<td>Kasner et al, Stroke 2001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p=0.001</td>
<td></td>
</tr>
<tr>
<td>Admission NIHSS &gt;20</td>
<td>28</td>
<td>100% sens 78% spec</td>
<td>Oppenheim et al, Stroke 2000</td>
</tr>
<tr>
<td>(&gt;=15 for non-dom hemisphere)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nausea and vomiting 1st 24 hours</td>
<td>135</td>
<td>OR 5.1 95% CI 1.7-15.3</td>
<td>Krieger et al, Stroke 1999</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p=0.003</td>
<td></td>
</tr>
</tbody>
</table>
## Radiographic Predictors of Malignant MCA

<table>
<thead>
<tr>
<th>Predictor</th>
<th># patients</th>
<th>Odds ratio or Sens/Spec</th>
<th>Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypodensity on initial head CT &gt; 50% MCA territory</td>
<td>135 201 36</td>
<td>OR 6.1, 95% CI 2.3-16.6, p=0.0004 OR 6.3, 95% CI 3.5-11.6, p = 0.001 OR 14.0, 95% CI 1.04-189.4, p=0.047</td>
<td>Krieger et al, Stroke 1999 Kasner et al, Stroke 2001 Manno et al, Mayo Clin Proc 2003</td>
</tr>
<tr>
<td>CT Hyperdense MCA sign</td>
<td>36</td>
<td>OR 21.6, 95% CI 3.5-130, p &lt; 0.001</td>
<td>Manno et al, Mayo Clin Proc 2003</td>
</tr>
<tr>
<td>CT Anteroseptal shift ≥ 5 mm on follow up head CT &lt;48 hrs</td>
<td>135</td>
<td>OR 10.9; 95% CI 3.2-37.6</td>
<td>Barber et al, Cerebrovasc Dis 2003</td>
</tr>
<tr>
<td>MRI DWI volume &gt;145 mL within 14 hours</td>
<td>28</td>
<td>100% sens, 94% spec</td>
<td>Oppenheim et al, Stroke 2000</td>
</tr>
<tr>
<td>MRI DWI volume &gt;82 mL within 6 hours of onset</td>
<td>140</td>
<td>52% sens, 98% spec</td>
<td>Thomalla et al, Ann Neuro 2010</td>
</tr>
</tbody>
</table>

Adapted from Wartenberg, 2012
DECIMAL, DESTINY, HAMLET trials pooled their data prior to each individual results completed and published
- 93 pts randomized <48 hours

Conclusions:

Significantly more patients met the primary outcome measures mRS0-4 at one year in the surgical group, ARR 51%, p<0.0001

*Figure 1: Distributions of the scores on the mRS and death after 12 months for patients treated with or without decompressive surgery*
Neurosurgical Options: Recommendations

1. In patients <60 years of age with unilateral MCA infarctions that deteriorate neurologically within 48 hours despite medical therapy, decompressive craniectomy with dural expansion is effective. The effect of later decompression is not known, but it should be strongly considered (Class I; Level of Evidence B).

2. Although the optimal trigger for decompressive craniectomy is unknown, it is reasonable to use a decrease in level of consciousness and its attribution to brain swelling as selection criteria (Class IIa; Level of Evidence A).

3. The efficacy of decompressive craniectomy in patients >60 years of age and the optimal timing of surgery are uncertain (Class IIb; Level of Evidence C).

4. Suboccipital craniectomy with dural expansion should be performed in patients with cerebellar infarctions who deteriorate neurologically despite maximal medical therapy (Class I; Level of Evidence B).
Conclusions:

When evaluated dichotomously, the odds of discharge to institutional care and of a poor outcome did not differ at 48 hours after hospital admission, but increased when surgery was pursued after 72 hours.

**Figure.** Differences in the crude rates (and SE) in mortality, discharge to institutional care, and of a poor outcome stratified by the timing of surgery: within 48 hours (A) and within 72 hours (B). A poor outcome was defined using the Nationwide Inpatient Sample-subarachnoid hemorrhage outcome measure (death, tracheostomy and gastrostomy placement, or discharge to institutional care). Statistically significant differences from multivariable logistic regression are designated with an asterisk. DHC indicates decompressive hemicraniectomy.

Dasenbrock et al., 2017
Conclusions:

Hemicraniectomy increased survival without severe disability among patients 61 years of age or older with a malignant middle cerebral artery infarction.

The majority of survivors required assistance with most bodily needs.
Quality of Life Outcomes

Overall analysis found that DH had more quality adjusted life-years compared to medical therapy alone.

Despite moderate to severe disability including dominate hemisphere strokes, 7/8 patients had no regret for completing DH.
So.... What does this look like in practice?

Case Study
Case Study - OSH Presentation

- 59 year old male with history of Afib (on AC), HTN, BPH, GERD presenting to outside hospital with right MCA syndrome 16 hours from LKN. Transferred for higher level of care.

  - Likely stroke etiology: Cardio embolic (History of Afib, with non-compliance on Xarelto, related to recent PNA)

OSH – Non-Contrast Head CT
12/21 @ 6:36pm
Case Study - Arrival

- Arrived at 12:30am

- NIHSS at arrival: NIH of 17 (1 right gaze, 2 left VF, 2 left face, 4 left arm, 4 left leg, 2 sensory, 1 dysarthria, 1 neglect)

- Initial plan: 23% boluses q4h for goal Na of > 150 with q4 hour monitoring, OR in AM

- Around 3:30am exam deteriorated with and was taken for decompression ~ 20-24 hours post stroke

CT angiogram head and neck with and without contrast: 12/23/2016 2:38 am
CT head without contrast : 12/23/2016 11:17 am
Follow up

CT head with and without contrast: 1/30/2017 7:16 pm

Stroke Clinic:

- 5 months post stroke – living at SNF with bi-weekly rehab, will soon be moving in with son
- Full PO diet, not using PEG
- NIHSS: 11 (1 left VF, 1 left face, 4 left arm, 3 left leg, 1 sensory, 1 dysarthria)
References


ElizabethKim@stanfordhealthcare.org

THANK YOU!