Care of the Head Trauma Patient

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Objectives

1. Historical perspective in traumatic brain injury patients
2. Intraoperative hemodynamic changes during emergent decompressive craniotomy/craniectomy
3. Literature reviews and future studies

Historical Perspectives

Monro-Kellie doctrine (1824)
Intracranial volume (constant)
Historical Perspectives

Mechanism of the Pressor Response to Increased Intracranial Pressure

SIMON RODHARD AND HIROSHI SAIKI
From the Cardiovascular Department, Medical Research Institute, Michael Reese Hospital, Chicago, Illinois

Nauyn and Schreiber (1), in 1881, reviewed the literature on the effect of intracranial compression and attributed the blood pressure heightening effect to painful stimuli arising in the course of tearing of the dura. Cushing (1), at the turn of the century, carefully restudied the problem and concluded that the pressor responses were due to anemia of the brain, attendant upon an increased resistance to inflow of blood into the cranium.

Historical Perspectives


Historical Perspectives

Traumatic Coma Data Bank - Began collecting data in 1979


Secondary brain injury - Experimental research began in 1980


Historical Perspectives

Traumatic Brain Injury (TBI)

TBI is a non-degenerative, non-congenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairments of cognitive, physical, and psychosocial functions with an associated diminished or altered state of consciousness.
**Traumatic Brain Injury**

*Physical mechanisms of brain injury*
- Impact loading
- Impulsive loading
- Static or quasistatic loading

*Types of tissue deformity*
- Compressive
- Tensile
- Shear

**Types of intracranial hemorrhages**
- Epidural hematoma
- Subdural hematoma
- Subarachnoid hemorrhage
- Intracerebral hemorrhage
- Intraventricular hemorrhage

**Neurological scale of TBI**

\[ GCS = E4 + M6 + V5 \]

- Severe TBI - 3 to 8
- Moderate TBI - 9 to 12
- Mild TBI - 13 to 15

**Epidemiology**

- Annually, 250,000 TBI need hospitalization.
- Approximately 50,000 US deaths per year.
- Mortality rate in severe TBI - 33 to 55%
- Overall Mortality rate in TBI - 20 to 25%
- TBI accounts for \( \sim 35\% \) of all deaths from acute injuries in the United States.
Primary vs. Secondary Severe TBI

*Primary injury* is from bruising or penetrating objects

*Secondary injury* is from hypoxia or decrease cerebral perfusion

Peri-operative Care of Head Trauma patient

EMT → ED → Rad → ICU
EMT → ED → Rad → OR → ICU

Hypotension  •  Hypoxemia

OR Room 1 Cases @ SFGH Trauma center

- 100 - 120 cases per year; 8 - 10 cases per month
- 20% of patients required BT > 20 units for initial resuscitation (<12°)
- 25% of patients required activation of MTP
- 33% mortality rate; ~1/3 of patients had severe traumatic brain injury.
- ~30% traumatic brain injury; (10% OR mortality)
- Transfused 21 - 39 u pRBC with a 25% mortality
- Transfused > 40 u pRBC with a 52% mortality
- Complications from massive fluid resuscitation
Preoperative predictors of reduction in arterial blood pressure following dural opening during surgical evacuation of acute subdural hematoma

Kawaguchi M, Sakamoto T, Ohnishi H, Karasawa J, Furuya H

*Retrospective chart review
56 patients with traumatic acute subdural hematoma

Preoperative variables: clinical profile, hemodynamic parameters, neurological findings, and Cranial CT scan

5 min before opening the dura vs dural opening
- Group A (n = 18) MAP reduction > 20%
- Group B (n = 38) MAP within +/- 20% of baseline values

Low GCS score, absence of the mesencephalic cistern, and bilaterally dilated pupils were particularly strong predictors of this amount of blood pressure reduction. The clinical outcomes of patients in group A following dural opening during surgery were significantly poorer.

Findings suggest that blood pressure reduction following opening of the dura in patients undergoing surgical evacuation of hematoma for traumatic ASDH may be predicted by careful preoperative assessment of neurological and CT scan findings.
The Incidence & Risk Factors for Hypotension During Emergent
Decompressive Craniotomy in Children with Traumatic Brain Injury

Patrick Miller, MD*, Christ D. Mack, MS, Maria Sammer, MD, and Monica S. Vavilala, MD*
Anesth Analg 2006;103:869-875

CONCLUSION: Between 1994 and 2004, a retrospective cohort study in children (<13 yr) with traumatic brain injury (TBI) at a Level 1 pediatric trauma center to describe risk factors for intraoperative hypotension (IH) during emergent decompressive craniotomy. Overall, 56 (52%) patients had IH. ED hypotension, blood loss, CT lesion volume, and CT midline shift predicted IH. Anesthesiologists can expect children with preoperative CT midline shift 4 mm to have IH during this procedure.

Intraoperative Mortality in Severe Traumatic Brain-injured Patients

1. What is the pathophysiology of this deleterious event?

2. Can we improve the operative outcome by EGDT?
Differential Diagnosis

- Venous Air Embolism
- Cardiac dysfunction
- Volume depletion
- Hemorrhagic shock
- Neu. pulmonary edema
- Anesthetics

Volume depletion ↓
in the phase of
Mannitol (1g/kg) iv
and
Cushing’s Reflex ± D.I.

Circulating catecholamines and sympathetic activity after head injury.


Plasma NE and Dopa levels were measured during the first 7 days after head injury in 48 patients. BP, HR, temp, and GCS were recorded at the time of sampling for each patient, and the relationships of these to NE and Dopa were examined.

In patients with multiple trauma, NE levels were usually elevated, regardless of the GCS. In patients with head injury alone, however, NE was proportional to GCS. Alert patients after a brief loss of consciousness (GCS 14) had normal NE levels. Those in coma had levels as high as 7 times normal.
Circulating catecholamines and sympathetic activity after head injury


Blood pressure, pulse, and temperature were found to be elevated proportionally to elevations in plasma NE in patients with head injury.

The finding of elevated plasma NE in patients with severe head injury raises the possibility of adverse effects of sympathetic hyperactivity in patients with severe head injury, including hypermetabolism, cardiovascular abnormalities, and direct effects of catecholamines on the damaged central nervous system.

Hormonal and hemodynamic changes in a validated animal model of brain death


A Cushing reflex, followed by a hyperdynamic response and DI, occurred in every animal following brain death.

MAP  >350 mm Hg C.O.  2.8 L/min
HR  230 /min  Contractility  4200 mm Hg/sec
Catechols ↑ 15 mins after brain death
†vasopressin ↓ 15 mins after brain death
ACTH ↓ 45 mins after brain death
T3, thyroxine, and glucagon ↓ 420 mins after brain death

Conclusions:
In a validated animal model of brain death, significant decreases in the circulating concentrations of stress hormones, as well as hemodynamic instability, occurred after brain death. Measurements of plasma ACTH and vasopressin values may be useful as diagnostic predictors of brain death. Furthermore, the observed changes may contribute to organ dysfunction after brain death and may necessitate hormonal as well as inotropic and vasoactive support to maintain donor organ function in the clinical setting.
Perioperative practice changes in Severe Traumatic Brain-injured Patients, 2004-

Volume Depletion
- Mannitol iv is routinely given
- Hct is ~ normal or above normal
- BD is always within less than -10

Minimize anesthetics

Cushing’s reflex
- Hemodynamic monitoring
- CVP measurement

Perioperative practice changes in Severe Traumatic Brain-injured Patients, 2004-

OR (before dural opening)
- Established IJ or subclavian c-line, a-line
- Hemodynamic monitoring - pressors*
- Fluid resuscitation - CVP 10 or TEE 2009
- Avoid fix anesthetic agents
- O2 and muscle relaxant

OR (during dural opening)
- Well communication
- Slow decompression - fenestrations technique

Perioperative practice changes in Severe Traumatic Brain-injured Patients, 2004-

EDtrauma
- Intubated with Etomidate - avoid ↓ BP
- IV access - C-line, if possible
- Sent labs and blood samples for T&C
- ETCO₂ monitoring ( ~ 30 )
- Hemodynamic monitoring - a-line, if possible

RadCT
- Continue monitoring
- Maintain “hemodynamic”

Role of dural fenestrations in acute sub-dural hematoma

Guilburd JN, Sviri GE

One of the factors contributing to poor outcomes in cases of ASDHs could be rapid surgical decompression, owing to the severe extrusion of the brain through the craniotomy defect in response to acute brain swelling. To avoid the deleterious consequences of abrupt decompression of the sub-dural space with disruption of brain tissue, this procedure consists of creating multiple fenestrations of the dura (MFD) in a mesh-like fashion and removing clots through the small dural openings that are left open, avoiding the creation of a wide dural opening and the disruption of and additional damage to brain tissue.
Role of dural fenestrations in acute sub-dural hematoma

Guilburd JN, Sviri GE

31 patients:
26 male and 5 female with mean 32.5 yrs
16 patients (51.5%) GCS scores of 3 - 5
11 patients (35.5%) GCS scores of 6 - 8
4 patients (12.9%) GCS scores of 9 - 12

Postoperative computerized tomography scans of the brain revealed evacuation of more than 80% of the hematoma in 29 of 31 patients.
Overall mortality rate in this group was 51.6%.

CONCLUSIONS: This preliminary report of a new surgical approach for patients who have sustained ASDHs should be considered to avoid abrupt disruption of the brain and to allow the gradual and gentle release of sub-dural clots. This is especially important in cases in which there are severe midline shifts and a tight brain. Further clinical studies should be conducted in a more selected series to estimate the impact of this new procedure on morbidity and mortality rates.
## Intra-Operative Deaths Pre-Change Post-Change

<table>
<thead>
<tr>
<th></th>
<th>Pre-Change</th>
<th>Post-Change</th>
<th>p value</th>
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<tbody>
<tr>
<td>n = 20</td>
<td>n = 39</td>
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### Gender (male,female %)
70,30 | 74,26 | 0.96, 0.99

### Age
58.0 ± 20.3 | 54.1 ± 18.2 | 0.49 *

### GCS
5.0 ± 2.5 | 5.5 ± 2.0 | 0.40 *

### Pupil Abnormality (n %)
12 (60) | 19 (49) | 0.59

### CT Midline Shift (mm)(n)
14.8 ± 4.5 (17) | 12.0 ± 6.7 (35) | 0.14 *

### Time Intervals (min)
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<tbody>
<tr>
<td></td>
<td>n = 20</td>
<td>n = 39</td>
<td></td>
</tr>
<tr>
<td>EMS – ED</td>
<td>25 ± 8</td>
<td>25 ± 11</td>
<td>0.72 *</td>
</tr>
<tr>
<td>ED – Dura Open *</td>
<td>94 ± 44</td>
<td>123 ± 35</td>
<td>&lt; 0.05 *</td>
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## Mechanism of Injury (n, %)

### Type of Injury (n, %)

<table>
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<tbody>
<tr>
<td></td>
<td>n = 20</td>
<td>n = 39</td>
<td></td>
</tr>
<tr>
<td>Fall</td>
<td>9 (45)</td>
<td>17 (44)</td>
<td>0.84</td>
</tr>
<tr>
<td>Vehicular Accident</td>
<td>9 (45)</td>
<td>16 (41)</td>
<td>0.99</td>
</tr>
<tr>
<td>Assault</td>
<td>2 (10)</td>
<td>6 (15)</td>
<td>0.90</td>
</tr>
<tr>
<td>SDH</td>
<td>13 (65)</td>
<td>28 (72)</td>
<td>0.80</td>
</tr>
<tr>
<td>SAH</td>
<td>3 (15)</td>
<td>4 (10)</td>
<td>0.39</td>
</tr>
<tr>
<td>EDH</td>
<td>2 (10)</td>
<td>4 (10)</td>
<td>0.65</td>
</tr>
<tr>
<td>ICH</td>
<td>4 (20)</td>
<td>3 (8)</td>
<td>0.36</td>
</tr>
</tbody>
</table>
| Craniectomy (n, %)
13 (65) | 28 (72) | 0.80
Craniotomy (n, %)
11 (55) | 14 (36) | 0.27

### Intra-Operative Deaths Pre-Change Post-Change

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<tr>
<td>Volume Resuscitation (L)</td>
<td>6.3 ± 3.6</td>
<td>9.9 ± 5.2</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Crystalloids/Colloids</td>
<td>2.8 ± 1.4</td>
<td>4.0 ± 1.8</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Blood Products</td>
<td>1.1 ± 0.9</td>
<td>1.9 ± 1.7</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>WB (n)</td>
<td>1.7 ± 0.6</td>
<td>0 (0)</td>
<td>ns *</td>
</tr>
<tr>
<td>PRBC (n)</td>
<td>1.0 ± 0.5</td>
<td>1.1 ± 0.8</td>
<td>(35)</td>
</tr>
<tr>
<td>FFP (n)</td>
<td>0.6 ± 0.4</td>
<td>1.3 ± 0.9</td>
<td>(22)</td>
</tr>
<tr>
<td>PLTS (n)</td>
<td>0 (0)</td>
<td>0.4 ± 0.2</td>
<td>(12)</td>
</tr>
<tr>
<td>Volume Loss (L) (n)</td>
<td>3.9 ± 2.6</td>
<td>6.5 ± 3.4</td>
<td>(39)</td>
</tr>
<tr>
<td>UO (n)</td>
<td>0.8 ± 0.7</td>
<td>2.6 ± 1.5</td>
<td>(38)</td>
</tr>
<tr>
<td>EBL (n)</td>
<td>1.2 ± 0.7</td>
<td>1.5 ± 1.0</td>
<td>(36)</td>
</tr>
<tr>
<td>Fluid Balance (L) (n) *</td>
<td>1.3 ± 2.9</td>
<td>2.8 ± 4.4</td>
<td>(35)</td>
</tr>
<tr>
<td>Mannitol Dose (g) (n)</td>
<td>81.1 ± 29.0</td>
<td>98.5 ± 30.6</td>
<td>(33)</td>
</tr>
<tr>
<td>CVP (mmHg) (n)</td>
<td>0 (0)</td>
<td>15.1 ± 5.8</td>
<td>(20)</td>
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<tr>
<td>Isoflurane (n) *</td>
<td>31.4 ± 19.0</td>
<td>17.2 ± 13.3</td>
<td>(20)</td>
</tr>
<tr>
<td>Desflurane (n) *</td>
<td>60 ± 0</td>
<td>155 ± 67</td>
<td>(3)</td>
</tr>
<tr>
<td>Sevoflurane (n) *</td>
<td>10.0 ± 0.0</td>
<td>74.5 ± 97.4</td>
<td>(5)</td>
</tr>
<tr>
<td>Fentanyl (n) **</td>
<td>237.5 ± 182.7</td>
<td>176.6 ± 128.6</td>
<td>(8)</td>
</tr>
<tr>
<td>Versed (n) **</td>
<td>0 (0)</td>
<td>2 ± 0</td>
<td>(2)</td>
</tr>
<tr>
<td>Propofol (n) **</td>
<td>500 ± 0.0</td>
<td>0 (0)</td>
<td>ns</td>
</tr>
<tr>
<td>Muscle Relaxants † *</td>
<td>65%</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>Vasopressor Support † *</td>
<td>95%</td>
<td>92%</td>
<td></td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>80%</td>
<td>74%</td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>85%</td>
<td>69%</td>
<td></td>
</tr>
<tr>
<td>Nor-Epinephrine</td>
<td>0%</td>
<td>10%</td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td>20%</td>
<td>5%</td>
<td></td>
</tr>
<tr>
<td>Vasopressin</td>
<td>20%</td>
<td>10%</td>
<td></td>
</tr>
<tr>
<td>Ephedrine</td>
<td>10%</td>
<td>8%</td>
<td></td>
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![MAP vs Time Graph](image.png)

Map of ICP changes during surgery.
Perioperative Practice Changes in Severe Traumatic Brain-injured Patients - Conclusion

There is no significant difference between the two groups under study. The two groups are compatible.

The practice change group that had zero mortality received less anesthetics, early resuscitation and aggressive hemodynamic monitoring intraoperatively.

Primary vs. Secondary Severe TBI

Primary injury is from bruising or penetrating objects

Secondary injury is from hypoxia or decrease cerebral perfusion

Future Studies

Clinical
Outcome study
Cushing’s reflex - timing
Brain protection agents

Basic science
Pressor selection
Vasomotor response
Cytokines and
Catecholamine levels
Brain protection agents

Conclusion: Brain injury is reduced and neurological outcome improved after MCAO in mice lacking the $\beta_2$AR, or in wild type mice pretreated with a selective $\beta_2$AR antagonist. This is consistent with a shift away from prosurvival signaling to prodeath signaling in the presence of $\beta_2$AR activation in cerebral ischemia. Protection is associated with higher levels of Hsp72, a known antideath protein. The effect of $\beta_2$AR signaling in the setting of cerebral ischemia is complex and warrants further study.
Postischemic Brain Injury Is Attenuated in Mice Lacking the β2-Adrenergic Receptor

Ru-Quan Han, MD, PhD*(dagger), Yi-Bing Ouyang, PhD*, Lijun Xu, MD*, Rani Agrawal, PhD*, Andrew J. Patterson, MD, PhD*, and Rona G. Giffard, MD, PhD*


Conclusion: Brain injury is reduced and neurological outcome improved after MCAO in mice lacking the β2AR, or in wild type mice pretreated with a selective β2AR antagonist. This is consistent with a shift away from prosurvival signaling to prodeath signaling in the presence of β2AR activation in cerebral ischemia. Protection is associated with higher levels of Hsp72, a known antideath protein. The effect of β2AR signaling in the setting of cerebral ischemia is complex and warrants further study.

The Effects of β-Adrenoceptor Antagonists on Proinflammatory Cytokine Concentrations After SAH in Rats

Haruto Kato, MD, Masahiko Kawaguchi, MD, Satoki Inoue, MD, Katsuji Hira, MD, and Hirosi Furuya, MD


Results: In Experiment 1, CSF IL-6 concentrations in the SAH groups increased markedly and peaked at 6 h after SAH, whereas CSF TNF-α concentrations in the control groups were consistently low. In Experiment 2, CSF IL-6 concentrations in the propranolol and butoxamine groups were significantly lower compared with those in the control group (P < 0.01 and P < 0.05 for each group). Plasma IL-6, CSF TNF-α, and plasma TNF-α concentrations were comparable in all four groups.

Conclusion: CSF IL-6 concentrations increased in the acute stage of SAH and β-adrenoceptor antagonists with a β2-adrenoceptor blocking action suppressed this elevation of IL-6 concentrations after SAH in rats.

Cytokines and Catecholamines

Cytokines: TNF-α and IL-6

Catechols: Epi, NE and Dopa

Purpose of study (TBISMS)

1. To investigate the time course responses of plasma cytokines and catecholamines in the early stage of traumatic brain injury (TBI).
2. To study the serum levels of cytokines and catecholamines in response to the changes of intracranial pressure (ICP), Cushing response, and Glasgow Coma Scale (GCS).
3. To determine the relation of acute stress responses to their long-term prognosis after TBI.
Summary and Recommendations

1. Cushing reflex is a protective response to increased intracranial pressure by maintaining adequate cerebral perfusion pressure and continuing the delivery of oxygen to the injured brain tissue. (Hypertension and adequate oxygenation)

2. Catecholamines and other proinflammatory mediators tend to increase in response to the severity of brain insults. (vasomotor paralysis)

3. Mannitol administration and/or DI can cause severe hypovolemic state which in turn can be masked by the Cushing effect. (Increased Hct and U.O)

4. Preoperative BP and HR can not predict intraoperative hypotension during EDC.

5. Avoid “fix” (long-duration) anesthetic agents and use narcotics judiciously.

6. Monitor volume levels via CVP or TEE and check ABG’s frequently. Maintain CPP between 60 to 90 mmHg before and after the dural opening. (Estimation of ICP)

7. Attenuation of “reverse Cushing effect” with slow dural opening by neurosurgeon.

8. Avoid secondary injury by ultimately fulfilling the basic needs of adequate perfusion pressure and adequate oxygenation in TBI.

DR. HARVEY WILLIAMS CUSHING. (1869-1939)