The Three Ts of Brain Injury: Trauma Technology Triumph

Presented by:
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Disclosures

- Integra Neuroscience
  - Speaker’s Bureau
- Medivance/Bard
  - Honorarium
- Board of Directors
  - AANN President Elect
  - NCS
- Medical Advisory Board
  - Brain Trauma Foundation
  - Neuroptics
Managing Severe TBI

- Historical approach prior to 1995
  - ICP driven
  - Interventions
    - Hyperventilation
    - Dehydration
    - Steroids
    - Anticonvulsants (long term)
  - Outcomes poor
    - High Mortality (50%)
    - High Morbidity
Changing Practice

- **Critical Elements**
  - Evidence Based Literature
    - Publication of EBL “Guidelines for the Management of Severe Head Injury”
  - Interdisciplinary team of practitioners
    - Collaborative Practice
    - Mission Hospital SICU
  - Culture
    - Mutual respect, trust, innovation, and risk taking
    - Patient/Family Centered Care
  - Leadership/Change Agents
    - Physician/Nurse and Hospital Leaders
Critical Care Management of Severe TBI

Pathological Changes

Secondary Injury

Dynamics of Injury & Monitoring Technologies

Evidence Based Practice

Coordinated ICU Multidisciplinary Care
Etiology of Brain Injury

- **Mechanisms of Injury**
  - Trauma
    - Blunt
    - Penetrating
    - Blast

- **Primary Injury**
  - Skull integrity
  - Brain integrity
    - Focal injuries
    - Diffuse injuries
Results

- Increase in tissue volume, blood, or CSF
- Increased in contents of cranial vault
Secondary Injury: Alteration in CBF

- Numerous studies have found low CBF in early hours after TBI
- Martin et al study on CBF in TBI
  - 1st 12 to 24 hours: Hypoperfusion/decrease in CBF
  - 24 hours to Day 5: CBF exceeding CMRO2
  - Days 5/6 to 14: Slow flow due to vasospasm
- CBF altered but it must be balanced with metabolism and oxygenation

Pathophysiology of traumatic brain injury

C. Werner® and K. Engelhard

Physiologic and functional outcome correlates of brain tissue hypoxia in traumatic brain injury

Jason J. Chang, BS; Teddy S. Youn, BA; Dan Benson, BS; Heather Matteck, BS; Nicholas Andrade, BA; Caryn R. Harper, MS; Carol B. Moore, MA; Christopher J. Madden, MD; Ramon R. Diaz-Arrastia, MD, PhD

(Crit Care Med 2009; 37:283–290)
Impaired autoregulation

Pressure autoregulation: the ability of brain to maintain constant CBF in face of changing BP or CPP

CPP

- Measured with ICP in place
- \( \text{CPP} = \text{MAP} - \text{ICP} \)
- Optimal CPP differs in patients due to whether pressure autoregulation is intact
At MABP’s of <60 mmHg, cerebral ischemia develops.

At MABP’s of >140 mmHg, cerebral vascular congestion can occur.

Lassen, 1959
Cerebral Blood Flow

Autoregulation

- Vasomotor control
  - Intact: Increase in CPP causes vasoconstriction and decrease in ICP
  - Vasomotor reactivity failure: Increase in CPP causes vasodilation and increase ICP

- Flow metabolism
  - $\uparrow$ metabolism $\uparrow$ CBF

- Metabolic substances
  - PaO2
  - PaCO2
  - pH i.e., acidosis = vasodilation
Secondary Injury

- If pressure autoregulation impaired
  - Cerebral ischemia results reducing O2 delivery to brain
  - Cerebral metabolism severely altered due to
    - Loss of CBF
    - Decrease in CBF
  - Shifts metabolism from aerobic to anaerobic
Secondary Brain Injury

- Hypotension
- Hypoxia
- Hypocarbia
- Hypercarbia
- Anemia
- Fever

Fever control and its impact on outcomes: What is the evidence?
Venkatesh Aiyagari a,*, Michael N. Dringer b,1

Although there is a body of experimental data and clinical experience that relate fever to more substantial neurologic injury and worse outcome, the answer to the critical question: “Does fever control improve outcome?” is not known. This is not to indicate that absence of proof is proof of absence. The definitive study has not been performed.

Pathophysiology: Intracranial Pressure

- Theories on Brain Compartment
  - 80% brain
  - 10% blood
  - 10% CSF
- If one increases the other two decrease
- Compensatory mechanisms

- SDH
- Brain moves over
- Venous blood to heart
- CSF shunts to spine SAS
Symptoms of Increased ICP: Adults

- **Early**
  - Altered level of consciousness, restless, agitated, headache, nausea, and contralateral motor weakness
  - Cranial nerves III and VI

- **Late**
  - Coma, vomiting, contralateral hemiplegia, and posturing
  - Alteration in Vital Signs
  - Impaired brainstem reflexes
    - Pupils, dysconjugate gaze
ICP Monitors

- **Location**
  - Intraventricular - most efficient/drain CSF
  - Parenchymal - helps with trending/drifts
Intracranial Pressure

- Normal range
  - Adolescents/Adults
    0-15 mm Hg
- Abnormal ranges
  - Adolescent/Adults
    - moderate 20-40
    - severe > 40
ICP and MAP Relationship

- The brain’s ability to maintain constant blood flow in spite of fluctuations in systemic blood pressure
- Described mathematically by the Cambridge Group as Prx index
- Prx index
  - A moving correlation coefficient between MABP or MAP and ICP
Data Acquisition, PRx Calculation and Data Manipulations

All our ICU monitors (Siemens SC9000 XL) are networked. They are connected via Ethernet TCP/IP to a software (Infinity GatewayPlus, Dräger Medical) that automatically and continuously acquires and stores all parameters and waveforms. Together with the network system, Dräger supplies a software tool, named Patient Trend, that allow to visualize digitalized data on the computer screen, and also to export text data files for our purposes. Using this software, 2-h waveforms intervals were exported at 100 Hz resolution. G language software (LabVIEW, National Instruments) was developed in house to calculate PRx as Pearson correlation coefficient between 40 consecutive time averaged values of ICP and AP using a waveform time integration for 5 s intervals. Computations were repeated with a moving window of 5 s.

We validated PRx calculator software using exemplary clinical recordings obtained in NICU, Addenbrooke’s Hospital, Cambridge, UK.

The program calculates mean PRx for each 10 mmHg CPP interval and highlights the CPP corresponding to lowest PRx. The software can automatically export a printable text data file containing: 2-h mean PRx, time course of CPP, AP, ICP and PRx, and PRx-CPP distribution graph.

Every day during the observation period, PRx has been calculated over a 2-h time window as a part of clinical assessment and a printed report, containing mean PRx and CPP-PRx graph, was discussed during clinical rounds; it was accessible to ICU staff and stored in patient’s medical records. For each 2 h registration interval, the maximum and minimum ICP (ICP max and min) and the corresponding CPP and AP values (CPP at ICP max and min, AP at ICP max and min) were recorded.

We cleaned, due to the presence of obvious ICP or AP curve artefacts, a mean of 5.6 ± 11 min every 2 h (2 ± 9% of every registration periods).
ICP and CPP Relationship

- **Correlation (-1 to 0)**
  - As CPP increases, ICP decreases
  - Indicates intact cerebrovascular reactivity

- **+ Correlation (>0 to 1)**
  - As CPP increases, so does ICP
  - Indicates the loss of cerebrovascular reactivity
  - Pressure passive dilatation
Non-invasive Measurement of ICP Pupillometer
Here is a typical pupillary light response
Pupillometer


CV fell to 0.81 mm/sec when ICP trended to > 20

---

**TABLE 3**

*Pupillary measurements obtained in healthy volunteers and head-injured patients*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>healthy volunteers (310 persons, 2432 paired measurements)</td>
<td></td>
</tr>
<tr>
<td>mean maximum resting aperture (mm)</td>
<td>4.1 ± 0.34</td>
</tr>
<tr>
<td>mean minimum aperture (mm)</td>
<td>2.7 ± 0.21</td>
</tr>
<tr>
<td>mean reduction in size (%)</td>
<td>34</td>
</tr>
<tr>
<td>mean constriction velocity (mm/sec)</td>
<td>1.48 ± 0.33</td>
</tr>
<tr>
<td>mean latency duration (secs)</td>
<td>0.24 ± 0.4</td>
</tr>
<tr>
<td>head-injured patients w/ ICP &lt; 20 mm Hg (26 persons, 168 paired measurements)</td>
<td></td>
</tr>
<tr>
<td>mean maximum resting aperture (mm)</td>
<td>2.10 ± 0.16</td>
</tr>
<tr>
<td>mean minimum aperture (mm)</td>
<td>1.7 ± 0.1</td>
</tr>
<tr>
<td>mean reduction in size (%)</td>
<td>19</td>
</tr>
<tr>
<td>mean constriction velocity (mm/sec)</td>
<td>1.18 ± 0.18</td>
</tr>
<tr>
<td>mean latency duration (secs)</td>
<td>0.26 ± 0.6</td>
</tr>
</tbody>
</table>
Application Case 5 TBI

- 21 year old male sustains severe TBI
  - ICP/Brain oxygen monitors placed
    - ICP controllable first 24 hours with ICP <20
  - Pupillometer
    - Right Pupil 2.5 – 2.1mm CV 0.92 mm/sec
    - Left Pupil 2.7 -- 2.3 mm CV 1.02 mm/sec
  - Pupillometer slows 2 hours later...

![Pupillometry measurement]

<table>
<thead>
<tr>
<th>ID. No.</th>
<th>Maximum Aperture</th>
<th>Minimum Aperture</th>
<th>Latency</th>
<th>Constriction Velocity</th>
<th>Dilation Velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.340 sec</td>
<td>-0.58 mm/sec</td>
<td>0.32 mm/sec</td>
</tr>
<tr>
<td></td>
<td>2.3 mm</td>
<td>2.1 (9 %)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.340 sec</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>620779</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
21 year old male sustains severe TBI

- ICP increases to 32 mm Hg 40 minutes later
- Treated with Hypertonic Saline
  - ICP decreases
  - Constriction Velocity returns to 0.95 mm/sec and 1.05 mm/sec
**Pupillometer**

**NPI**

<table>
<thead>
<tr>
<th>ID:</th>
<th>3 [R]</th>
<th>2000/01/02 09:14:50</th>
</tr>
</thead>
<tbody>
<tr>
<td>NPI:</td>
<td>4.5 [Right]</td>
<td>4.6 [Left]</td>
</tr>
<tr>
<td>MAX:</td>
<td>4.77</td>
<td>4.19</td>
</tr>
<tr>
<td>MIN:</td>
<td>2.81</td>
<td>2.62</td>
</tr>
<tr>
<td>CON:</td>
<td>-41%</td>
<td>-37%</td>
</tr>
<tr>
<td>Lat:</td>
<td>0.23</td>
<td>0.23</td>
</tr>
<tr>
<td>CV:</td>
<td>-3.64</td>
<td>-3.72</td>
</tr>
<tr>
<td>MCV:</td>
<td>-5.40</td>
<td>-5.04</td>
</tr>
<tr>
<td>DV:</td>
<td>0.94</td>
<td>1.31</td>
</tr>
</tbody>
</table>

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![Graph showing pupillary response over time](chart)
Subjects with abnormal/nonreactive NPi™ had a peak of ICP higher than subjects with normal NPi™. The first occurrence of abnormal NPi™ relative to the time of the first peak of ICP was 15.9 hours. (CI=-28.56,-3)
Oxygenation

Delivery of oxygen to the brain dependent on Lungs
Hemoglobin and Plasma
Preload (CVP) / Cardiac Output / Afterload (SVR)

\[ CBF = CPP/CVR \]

Autoregulation
Vasomotor control

Chemical
PaCO2 / PaO2 / pH

Flow Metabolism
↑ metabolism/flow ↓ metabolism/flow
Oxygen Dynamics: Brain Tissue Oxygen Monitoring

Regional Detection
Penumbra Area

Global Measurement
Contralateral to Injury
Physiologic studies: Mitochondria needs

- Needs an mitochondrial O2 concentration of 1.5 mm Hg to produce ATP = PbtO2 15-20 mm Hg
  - Maloney-Wilensky and Leroux argue
    - Minimum threshold of 20 mm Hg is acceptable.

The physiology behind direct brain oxygen monitors and practical aspects of their use

Childs Nerv Syst
DOI 10.1007/s00381-009-1037-x
Brain Tissue Oxygen (PbtO2)

- Normal: 20-40 mm Hg
- Risk of death increases
  - < 15 mm Hg for 30 minutes
  - < 10 mm Hg for 10 minutes
- PbtO2 < 5 mm Hg
  - high mortality
- PbtO2 < 2mm Hg - neuronal death
Outcomes: TBI

Reduced mortality rate in patients with severe traumatic brain injury treated with brain tissue oxygen monitoring

Michael F. Stieffel, M.D., Ph.D., Alejandro Spiotto, M.D., Vincent H. Gracias, M.D., Alicia M. Garutte, M.S.N., Oscar Guillamondegui, M.D., Eileen Maloney-Wilensky, M.S.N., Stephanie Bloom, M.S.N., M. Sean Grady, M.D., and Peter D. LeRoux, M.D.

Department of Neurosurgery and Division of Trauma Surgery and Surgical Critical Care, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania

Brain tissue oxygen monitoring in traumatic brain injury and major trauma: outcome analysis of a brain tissue oxygen-directed therapy

Clinical article

Praeen K. Narotam, M.D., M.M.E.D., John F. Morrison, M.S., M.D., and Narendra Nathoo, M.D., Ph.D.

TABLE 3: Comparison of GOS scores between the PbO2-CCG and ICP/CPP groups

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>17 (41)</td>
<td>36 (28)</td>
<td>2.06</td>
<td>0.98-4.17</td>
</tr>
<tr>
<td>2</td>
<td>1 (2.4)</td>
<td>1 (0.8)</td>
<td>3.9</td>
<td>0.85-17.8</td>
</tr>
<tr>
<td>3</td>
<td>4 (12)</td>
<td>7 (5)</td>
<td>0.3</td>
<td>1.47-7.12</td>
</tr>
<tr>
<td>4</td>
<td>10 (27)</td>
<td>18 (14)</td>
<td>2.27</td>
<td>0.739-6.12</td>
</tr>
<tr>
<td>5</td>
<td>7 (17)</td>
<td>65 (51)</td>
<td>4.14</td>
<td>1.72-9.98</td>
</tr>
<tr>
<td>lost to FU</td>
<td>2 (4.8)</td>
<td>12 (8.6)</td>
<td>*</td>
<td></td>
</tr>
</tbody>
</table>

* Comparison reached statistical significance (p < 0.05, analysis of variance).

Fig. 1. Bar graph illustrating the mortality rates in patients who received traditional ICP/CPP therapy (Group A, 25 patients) or combined ICP/CPP and brain tissue PO2 treatment (Group B, 28 patients). *p < 0.05.
Brain tissue oxygen tension monitoring in pediatric severe traumatic brain injury

Part 1: Relationship with outcome

Anthony A. Figaji • Eugene Zwane • Crispin Thompson • A. Graham Fieggen • Andrew C. Argent • Peter D. Le Roux • Jonathan C. Peter

Table 3 PbO\textsubscript{2} parameters with adjusted Odds ratios for unfavorable outcome from multivariate logistic regression models

<table>
<thead>
<tr>
<th>Parameter</th>
<th>( p ) value</th>
<th>OR</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>PbO\textsubscript{2av} &lt;5 mmHg</td>
<td>0.004\textsuperscript{a}</td>
<td>24.6</td>
<td>2.8-214.6</td>
<td>0.561</td>
</tr>
<tr>
<td>PbO\textsubscript{2} &lt;5 for &gt;1 hour</td>
<td>0.015\textsuperscript{a}</td>
<td>27.4</td>
<td>1.9-391</td>
<td>0.54</td>
</tr>
<tr>
<td>PbO\textsubscript{2} &lt;10 for &gt;2 h</td>
<td>0.021\textsuperscript{a}</td>
<td>10.8</td>
<td>1.4-82.4</td>
<td>0.563</td>
</tr>
<tr>
<td>mPbO\textsubscript{2s} &lt;16 mmHg</td>
<td>0.062</td>
<td>8.9</td>
<td>0.9-87.5</td>
<td>0.521</td>
</tr>
<tr>
<td>Time-severity product &gt;20</td>
<td>0.002\textsuperscript{a}</td>
<td>47.6</td>
<td>4.2-543.6</td>
<td>0.564</td>
</tr>
</tbody>
</table>

\( p \) values, adjusted Odd's ratios (OR), confidence intervals, and Nagelkerke's \( R^2 \) for each multivariate model (other variables not shown). OR is reported as the odds of unfavorable outcome (severe disability or death).

\textsuperscript{a}Significant results

Table 4 PbO\textsubscript{2} parameters with adjusted ORs for mortality from multivariate logistic regression models

<table>
<thead>
<tr>
<th>Parameter</th>
<th>( p ) value</th>
<th>OR</th>
<th>95% CI</th>
<th>Confidence interval</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>PbO\textsubscript{2av} &lt;5 mmHg</td>
<td>0.016\textsuperscript{a}</td>
<td>26.9</td>
<td>1.9-387.4</td>
<td>0.464</td>
<td></td>
</tr>
<tr>
<td>PbO\textsubscript{2} &lt;5 for &gt;1 h</td>
<td>0.005\textsuperscript{a}</td>
<td>26.8</td>
<td>2.7-265.0</td>
<td>0.33</td>
<td></td>
</tr>
<tr>
<td>PbO\textsubscript{2} &lt;10 for &gt;2 h</td>
<td>0.017\textsuperscript{a}</td>
<td>20.4</td>
<td>1.7-244.7</td>
<td>0.442</td>
<td></td>
</tr>
<tr>
<td>mPbO\textsubscript{2s} &lt;16 mmHg</td>
<td>0.012\textsuperscript{a}</td>
<td>25.8</td>
<td>2.1-323.9</td>
<td>0.439</td>
<td></td>
</tr>
<tr>
<td>Time-severity product &gt;20</td>
<td>0.002\textsuperscript{a}</td>
<td>43.3</td>
<td>3.8-491.3</td>
<td>0.453</td>
<td></td>
</tr>
</tbody>
</table>

\( p \) values, adjusted Odds ratios (OR), confidence intervals, and \( R^2 \) for each multivariate model (other variables not shown). ORs are reported as the odds of mortality.

\textsuperscript{a} Significant results

Conclusion Reduced PbO\textsubscript{2} is shown to be an independent factor associated with poor outcome in pediatric severe TBI in the largest study to date. It appears to have a stronger association with outcome than conventionally evaluated measures.
Brain Tissue Oxygen Pressure and Prognosis in Spontaneous Intracerebral Hematomas

Irene Nikaina, MD, PhD,* Konstantinos N. Paterakis, MD, PhD,† Georgios M. Hadjigeorgiou, MD, PhD,‡ Vissarion Christodoulou, MD,* Apostolos Karantanas, MD, PhD,§ Antonios Karavelis, MD, PhD,† and Apostolos Komnos, MD, PhD*

Unfavorable outcome was correlated with the presence of an episode of \( \text{PtiO}_2 \) values \(< 5 \text{ mm Hg} (> 30 \text{ min}) \) and with the relative total duration of \( \text{PtiO}_2 \) values below that level. \( \text{PtiO}_2 \) monitoring in patients with spontaneous intracerebral hematomas may be a reliable tool for their prognosis.
Interventions and PbtO2

- Decreasing PbtO2
  - Hypoxia
  - Low Hemoglobin
  - Decreasing PaCO2
  - Increased ICP
  - Decreased MAP/CPP
  - Increasing temperature
  - Vasospasm
  - Systemic Causes
    - Pulmonary
    - Cardiac/Hemodynamic

- Increasing PbtO2
  - Increasing FIO2
  - Increasing Hemoglobin
  - Increasing PaCO2
  - Draining CSF -- ICP < 15 mm Hg
  - Increasing CPP/MAP
  - Decreasing temperature
  - Barbiturates
I. RECOMMENDATIONS  Level III

- Treatment thresholds
  - Jugular venous saturation (50%)
  - Brain tissue oxygen tension (15 mm Hg)
- Jugular venous saturation or brain tissue oxygen monitoring measure cerebral oxygenation (page 65)
Goal
Balance
ICP &
Brain Oxygen
Critical Care Management of Severe TBI

Pathological Changes

Secondary Injury

Dynamics of Injury & Monitoring Technologies

Evidence Based Practice
Guidelines for the Management of Severe Traumatic Brain Injury

A Joint project of the
Brain Trauma Foundation
American Association of Neurological Surgeons (AANS)
Congress of Neurological Surgeons (CNS)
AANS/CNS Joint Section on Neurotrauma and Critical Care

These guidelines are copyrighted by the Brain Trauma Foundation copyright ©2007. Copies are available through the Brain Trauma Foundation, 708 Third Avenue, Suite 1810, New York, NY 10017-4201, phone (212) 772-0608, fax (212) 772-0357. Website: www.braintrauma.org. E-mail: info@brain trauma.
I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

Blood pressure should be monitored and hypotension (systolic blood pressure < 90 mm Hg) avoided.

C. Level III

Oxygenation should be monitored and hypoxia (PaO$_2$ < 60 mm Hg or O$_2$ saturation < 90%) avoided.

II. OVERVIEW

For ethical reasons, a prospective, controlled study concerning the effects of hypotension or hypoxia on outcome from severe traumatic brain injury (TBI) has never been done. Nevertheless, there is a growing body of evidence that secondary insults occur frequently and exert a powerful, adverse influence on outcomes from severe TBI. These effects appear to be more profound than those that result when hypoxic or hypotensive episodes of similar magnitude occur in trauma patients without neurologic involvement. Therefore, it is important to determine if there is evidence for specific threshold values for oxygenation and blood pressure support.
I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

Mannitol is effective for control of raised intracranial pressure (ICP) at doses of 0.25 gm/kg to 1 g/kg body weight. Arterial hypotension (systolic blood pressure < 90 mm Hg) should be avoided.

C. Level III

Restrict mannitol use prior to ICP monitoring to patients with signs of transtentorial herniation or progressive neurological deterioration not attributable to extracranial causes.

The use of HS for ICP control was discovered from studies on "small volume resuscitation." Hyperonic saline solutions were tested in poly-traumatized patients with hemorrhagic shock. The subgroup with accompanying TBI showed the greatest benefit in terms of survival and hemodynamic parameters were restored effectively.\textsuperscript{59} The findings that HS may benefit patients with TBI while preserving or even improving hemodynamic parameters stimulated further research on the effects of HS solutions on increased intracranial pressure in patients with TBI\textsuperscript{15,18,36,40,41,46,51} subarachnoid hemorrhage,\textsuperscript{18,55,56} stroke,\textsuperscript{50} and other pathologies.\textsuperscript{14}
I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

There are insufficient data to support a Level II recommendation for this topic.

C. Level III

Pool[ed] data indicate that prophylactic hypothermia is not significantly associated with decreased mortality when compared with normothermic controls. However, preliminary findings suggest that a greater decrease in mortality risk is observed when target temperatures are maintained for more than 48 h.

Prophylactic hypothermia is associated with significantly higher Glasgow Outcome Scale (GOS) scores when compared to scores for normothermic controls.
I. RECOMMENDATIONS

A. *Level I*

There are insufficient data to support a Level I recommendation for this topic.

B. *Level II*

*Periprocedural antibiotics for intubation should be administered to reduce the incidence of pneumonia. However, it does not change length of stay or mortality.*

Early tracheostomy should be performed to reduce mechanical ventilation days. However, it does not alter mortality or the rate of nosocomial pneumonia.

C. *Level III*

Routine ventricular catheter exchange or prophylactic antibiotic use for ventricular catheter placement is not recommended to reduce infection.

A. *Level I*

There are insufficient data to support a Level I recommendation for this topic.

B. *Level II*

There are insufficient data to support Level II recommendation for this topic.

C. *Level III*

*Graduated compression stockings or intermittent pneumatic compression (IPC) stockings are recommended, unless lower extremity injuries prevent their use. Use should be continued until patients are ambulatory.*
VI. Indications for Intracranial Pressure Monitoring

I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a treatment standard for this topic.

B. Level II

Intracranial pressure (ICP) should be monitored in all salvageable patients with a severe traumatic brain injury (TBI; Glasgow Coma Scale [GCS] score of 3–8 after resuscitation) and an abnormal computed tomography (CT) scan. An abnormal CT scan of the head is one that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns.

C. Level III

ICP monitoring is indicated in patients with severe TBI with a normal CT scan if two or more of the following features are noted at admission: age over 40 years, unilateral or bilateral motor posturing, or systolic blood pressure (BP) < 90 mm Hg.

VII. Intracranial Pressure Monitoring Technology

In the current state of technology, the ventricular catheter connected to an external strain gauge is the most accurate, low-cost, and reliable method of monitoring intracranial pressure (ICP). It also can be recalibrated in situ. ICP transduction via fiberoptic or micro strain gauge devices placed in ventricular catheters provide similar benefits, but at a higher cost.

VIII. Intracranial Pressure Thresholds

B. Level II

Treatment should be initiated with intracranial pressure (ICP) thresholds above 20 mm Hg.

C. Level III

A combination of ICP values, and clinical and brain CT findings, should be used to determine the need for treatment.
IX. Cerebral Perfusion Thresholds

I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

Aggressive attempts to maintain cerebral perfusion pressure (CPP) above 70 mm Hg with fluids and pressors should be avoided because of the risk of adult respiratory distress syndrome (ARDS).

C. Level III

CPP of <50 mm Hg should be avoided.

The CPP value to target lies within the range of 50–70 mm Hg. Patients with intact pressure autoregulation tolerate higher CPP values.

Ancillary monitoring of cerebral parameters that include blood flow, oxygenation, or metabolism facilitates CPP management.

X. Brain Oxygen Monitoring and Thresholds

I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

There are insufficient data to support a Level II recommendation for this topic.

C. Level III

Jugular venous saturation (<50%) or brain tissue oxygen tension (<15 mm Hg) are treatment thresholds.

Jugular venous saturation or brain tissue oxygen monitoring measure cerebral oxygenation.
XI. Anesthetics, Analgesics, and Sedatives

I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

Prophylactic administration of barbiturates to induce burst suppression EEG is not recommended.

High-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability is essential before and during barbiturate therapy.

Propofol is recommended for the control of ICP, but not for improvement in mortality or 6 month outcome. High-dose propofol can produce significant morbidity.

XII. Nutrition

I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

Patients should be fed to attain full caloric replacement by day 7 post-injury.
I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

**Prophylactic use of phenytoin or valproate is not recommended for preventing late posttraumatic seizures (PTS).**

Anticonvulsants are indicated to decrease the incidence of early PTS (within 7 days of injury). However, early PTS is not associated with worse outcomes.

C. Level III

**Hyperventilation is recommended as a temporizing measure for the reduction of elevated intracranial pressure (ICP).**

Hyperventilation should be avoided during the first 24 hours after injury when cerebral blood flow (CBF) is often critically reduced.

If hyperventilation is used, jugular venous oxygen saturation ($S_jO_2$) or brain tissue oxygen tension ($PbrO_2$) measurements are recommended to monitor oxygen delivery.
I. RECOMMENDATIONS

A. Level I

The use of steroids is not recommended for improving outcome or reducing intracranial pressure (ICP). In patients with moderate or severe traumatic brain injury (TBI), high-dose methylprednisolone is associated with increased mortality and is contraindicated.
Managing the Severe TBI Patient
Airway and Breathing

- Assessment of airway/ventilation
  - Oxygenation
    - Titrating FIO2 as a temporary measure to benefit lungs/brain
  - Ventilation
    - Monitor CO2 constantly!
  - Modes of ventilation impact cerebral dynamics
  - Transport on ventilator to avoid inadvertent hyperventilation

B. Level II

Prophylactic hyperventilation (PaCO2 of 25 mm Hg or less) is not recommended.

C. Level III

Hyperventilation is recommended as a temporizing measure for the reduction of elevated intracranial pressure (ICP).

Hyperventilation should be avoided during the first 24 hours after injury when cerebral blood flow (CBF) is often critically reduced.

If hyperventilation is used, jugular venous oxygen saturation (SjO2) or brain tissue oxygen tension (PbrO2) measurements are recommended to monitor oxygen delivery.
Implications for Care

- Suctioning
- Bronchoscopy
- Turning vs Proning
Day 8: Lungs Worsening
Day 8: Lungs Worsening

<table>
<thead>
<tr>
<th>CO2</th>
<th>FIO2 %</th>
<th>MAP</th>
<th>ICP</th>
<th>CPP</th>
<th>PbtO2</th>
<th>Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>42</td>
<td>80</td>
<td>71</td>
<td>14</td>
<td>56</td>
<td>15.6</td>
<td>Increase Dopamine</td>
</tr>
<tr>
<td>42</td>
<td>80</td>
<td>76</td>
<td>12</td>
<td>64</td>
<td>18</td>
<td>Chest x-ray reviewed; Order to prone patient</td>
</tr>
<tr>
<td>43</td>
<td>80</td>
<td>90</td>
<td>17</td>
<td>63</td>
<td>24.5</td>
<td>4 Hours go by...sudden change in PbtO2</td>
</tr>
<tr>
<td>54</td>
<td>80</td>
<td>101</td>
<td>18</td>
<td>83</td>
<td>12.4</td>
<td>Lung sounds ↓; Supine; chest xray- Pneumo</td>
</tr>
<tr>
<td>100</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Chest tube placed</td>
</tr>
<tr>
<td>42</td>
<td>80</td>
<td>94</td>
<td>10</td>
<td>84</td>
<td>34</td>
<td>FIO2 weaned</td>
</tr>
</tbody>
</table>
Circulation

- Maintain MAP > 90 mm Hg until ICP in place
- Maintain CPP target 50-70 mm Hg
  - Find out where the right place is!
- HOW ...
  - Fluids
    - PA vs CVP thresholds
  - Vasopressors
    - Neo
    - Dopamine - frequently produces tachycardia
  - Transfusion of Packed RBCs
    - Controversial
    - Only when PbtO2 < 20 mm Hg and Hct < 33
Intracranial Pressure

- Increased ICP may cause a decreased PbtO2
- Decreasing ICP
  - Head of Bed/Neck positioning
  - CSF drainage
  - CPP optimization
  - CO2 Titration
  - Hypertonic Saline vs Mannitol
- Medications
  - Fentanyl
  - Versed
  - Propofol
  - Barbiturates
- Temperature control
- Craniectomy
Temperature

Increase in temperature

Increases oxygen consumption

Increases CBF which may lead to an increase in intracranial pressure
Temperature

- **Target**
  - 32-35 degrees Celsius
    - Protocol driven with tight control
  - 36-37 degrees Celsius
    - Ineffective
      - Acetaminophen
      - Fans / Cooling Blankets / Tepid bath / ice packs
  - Current effective methods
    - Intravascular cooling
    - Wraps / Pads
Bedside Shivering Assessment Scale (BSAS)

- Palpate masseter, pectoralis, deltoids and quadriceps muscles
  
  0 = No shivering
  
  1 = Mild shivering localized to neck and/or chest
  
  2 = Shivering involving neck and/or chest & arms
  
  3 = Intermittent generalized shivering involving all 4 extremities

Source:

Temp Control: Assessing Shivering

- **Objective:** BIS EMG Tracing
  - Picks up microshivering
**Step-Wise Management of Shivering**

<table>
<thead>
<tr>
<th>Step</th>
<th>Intervention</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Baseline</td>
<td>Acetaminophen 650–1000 mg Q 4–6 h</td>
</tr>
<tr>
<td></td>
<td>Baseline</td>
<td>Buspirone 30 mg Q 8 h</td>
</tr>
<tr>
<td></td>
<td>Baseline</td>
<td>Magnesium sulfate 0.5–1 mg/h IV Goal (3–4 mg/dl)</td>
</tr>
<tr>
<td></td>
<td>Baseline</td>
<td>Skin counterwarming 43°C/MAX Temp</td>
</tr>
<tr>
<td>1</td>
<td>Mild sedation</td>
<td>Dexmedetomidine 0.2–1.5 mcg/kg/h</td>
</tr>
<tr>
<td></td>
<td>Mild sedation</td>
<td>or Fentanyl starting dose 25 mcg/h</td>
</tr>
<tr>
<td></td>
<td>Mild sedation</td>
<td>Opioid Meperidene 50–100 mg IM or IV</td>
</tr>
<tr>
<td>2</td>
<td>Moderate sedation</td>
<td>Propofol 50–75 mcg/kg/min</td>
</tr>
<tr>
<td>3</td>
<td>Deep sedation</td>
<td>Vecuronium 0.1 mg/kg IV</td>
</tr>
<tr>
<td>4</td>
<td>Neuromuscular blockade</td>
<td></td>
</tr>
</tbody>
</table>
CBF and Shivering

07/10 07:00 to 07/12 06:59

Arrows on Arctic sun ↑↓

↑core T 0.3 C

Demerol bolus
Precedex drip
Norcuron bolus
Norcuron drip
Interventions: Systemic

- Bundles
  - VAP
  - Central Line
  - Infection control r/t ICP, foley etc

- GI:
  - OG for gastric decompression
  - Stress ulcer prophylaxis
  - Nutrition: caloric goal by day 7

- Musculoskeletal
  - ROM

- Family Support
Severe Brain Injury Algorithm

- Emergency Department: GCS 3-8
  - Oxygenate with 100%
  - Maintain in-line stabilization
  - Ventilate: PaCO2 35-45
  - RSI sequence
  - Hypertonic Saline
  - Fluid Resuscitation
  - Arterial line/Foley /OG
Severe Brain Injury Algorithm

- CT scan
- OR Priorities
  - Vent: 100% FIO2 and PaCO2 35-45
  - Place PA catheter; PbtO2; ICP
  - Optimize MAP > 90 mm Hg
    - Fluids
    - Packed RBCs
    - Correct coagulopathies
  - Propofol to reduce CMRO2 / ICP
Fluids: 9u FFP, 21u RBCs, 10u Cryo, 10 u Plts, + 4 L NS
Meds: Neo @ 200 ug/min and Propofol @ 150 ug/kg/min

- Decision to remove bone
- Neo to 200 ug
- SDH out
- Bone off
- Ortho Facial Procedures
Traumatic Brain Injury
Critical Thinking Algorithms

ICP < 20 & PbtO2 < 20

1st 24 hours: Look intracranial/alter CBF

After 24 hours: Check Systems especially the lungs

Allow the PaCO2 to rise

ICP > 20 & PbtO2 < 20

ICP > 20 & PbtO2 > 20
**PbtO² <20 & ICP>20**
- Drain CSF
- ↑ FIO₂ x 5-15 min only
- Optimize CPP
  - Fluids and vasopressors
  - Check H/H - transfuse if PbtO₂ <20 and Hct < 33
- ✓ analgesia/sedation
- Give Mannitol or HS
- Call MD
  - CT scan if ICP > 20
  - MD Decision:
    - Pentobarb Coma
    - Craniectomy
    - Cooling

**PbtO² 20-40 & ICP>20**
- Drain CSF
- ↓ CO₂ until ICP < 20; stop ↓ when PbtO₂ <20
- Optimize CPP
- ✓ analgesia/sedation
- Give Mannitol or HS
- Call MD
  - CT scan if ICP > 20
  - MD Decision:
    - Pentobarb Coma
    - Craniectomy
    - Cooling
Case: AB
Event

- 24 year old male involved in bike accident
  - Field
    - GCS 4-6-4
    - Vomiting
  - ED
    - GCS 4-6-3
    - PERRL
    - Vomiting with ? Aspiration of thick brown fluid and food
  - CT
    - Vomits again
    - Loses consciousness: GCS 1-3-1
    - Emergently intubated
Admit CT scan

TO OR
Post op
- OR
- SICU
  - ICP 20s
  - PbtO2 24 drops to 11 mm Hg
  - Pulmonary worsens

NPE
Low PbtO2 correlating with low PaO2
Progress Days 2-3

- Pulmonary Issues resolve x 4 days
- ICP controllable
- Hemodynamically improved
- Neurosurgeon elects to begin rewarm 0.05 degrees per hour on Day 4
Days 1-3
Abort

- ICP increases with attempted rewarmin
Rewarm....ICP shoots up

- Cooled for 72 hours then neurosurgical decision to rewarm
  - ICP increases to 35 mm Hg by 34.5 degrees
  - Phone conference call
  - MD decision to begin barbs
- ICP increases from 30 to 60mm Hg
  - Decompressive hemicraniectomy
  - ICP to 20s then back up
- Recool after 48 hours
Attempted Rewarm

08/12 07:00 to 08/22 06:59

Cranectomy

BPM

ICP

CPP

PetCO2

ETCO2
Recool x 7 days
Day 7
ARDS

- PaO2 50 and PbtO2 12
- Proning 4 hour down and Supine 2 hours
  - Loses effectiveness after 2 days
- Order to start Nitric Oxide
- Improvement
# Lungs worsen while on Barbs/Hypothermia ARDS

<table>
<thead>
<tr>
<th>Date/Time</th>
<th>Ventilator Support Mode</th>
<th>Pressure Control</th>
<th>Pressure Control</th>
<th>Pressure Control</th>
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<tbody>
<tr>
<td>8/21/08 09:50 thru 8/22/08 01:20</td>
<td>Inverse</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td></td>
<td>I:E Ratio On Inversed Settings</td>
<td>1.5:1</td>
<td>1.5:1</td>
<td>1.5:1</td>
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<tr>
<td></td>
<td>Ventilator Tidal Volume Setting</td>
<td>449 ml</td>
<td>440 ml</td>
<td>442 ml</td>
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<tr>
<td></td>
<td>Ventilator Tidal Volume Exhaled</td>
<td>462.0 ml</td>
<td>452.0 ml</td>
<td>451.0 ml</td>
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<tr>
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<td>Minute Ventilation</td>
<td>7.40 L/min</td>
<td>7.30 L/min</td>
<td>7.30 L/min</td>
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<tr>
<td></td>
<td>Ventilatory Support Breath Rate</td>
<td>16 bpm</td>
<td>16 bpm</td>
<td>16 bpm</td>
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<tr>
<td></td>
<td>Respiratory Rate</td>
<td>16 bpm</td>
<td>16 bpm</td>
<td>16 bpm</td>
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<tr>
<td></td>
<td>Ventilator Pressure Control Setting</td>
<td>18 cm H2O</td>
<td>18 cm H2O</td>
<td>18 cm H2O</td>
</tr>
<tr>
<td></td>
<td>Positive End Expiratory Pressure</td>
<td>15 cm H2O</td>
<td>15 cm H2O</td>
<td>15 cm H2O</td>
</tr>
<tr>
<td></td>
<td>Fraction of Inspired Oxygen (FiO2)</td>
<td>90 %</td>
<td>80 %</td>
<td>70 %</td>
</tr>
<tr>
<td></td>
<td>Peak Inspiratory Airway Pressure</td>
<td>33 cm H2O</td>
<td>33 cm H2O</td>
<td>33 cm H2O</td>
</tr>
<tr>
<td></td>
<td>Mean Airway Pressure</td>
<td>26.00 cm H2O</td>
<td>26.00 cm H2O</td>
<td>26.00 cm H2O</td>
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<tr>
<td></td>
<td>Ventilator Inspiratory Rise</td>
<td>3 %</td>
<td>3 %</td>
<td>3 %</td>
</tr>
<tr>
<td></td>
<td>Ventilator Inspiratory Time Setting</td>
<td>60 %</td>
<td>60 %</td>
<td>60 %</td>
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<tr>
<td></td>
<td>Inspiratory/Expiratory Phase Ratio</td>
<td>1.5:1</td>
<td>1.5:1</td>
<td>1.5:1</td>
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<tr>
<td></td>
<td>Sensitivity Type</td>
<td>Pressure</td>
<td>Pressure</td>
<td>Pressure</td>
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<tr>
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<td>Ventilator Circuit Temperature</td>
<td>37.0 degrees C</td>
<td>37.0 degrees C</td>
<td>37.0 degrees C</td>
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<tr>
<td></td>
<td>Resuscitation Bag/Mask At Bedside</td>
<td>Yes - connected to NO flowmeter</td>
<td>Yes - connected to NO flowmeter</td>
<td>Yes - connected to NO flowmeter</td>
</tr>
<tr>
<td></td>
<td>Bedside Pulse Oximetry</td>
<td>95 %</td>
<td>97 %</td>
<td>96 %</td>
</tr>
<tr>
<td></td>
<td>End-tidal CO2</td>
<td>33 mmHg</td>
<td>35 mmHg</td>
<td>37 mmHg</td>
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<tr>
<td></td>
<td>Gradient</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Pulse Rate</td>
<td>83 bpm</td>
<td>77 bpm</td>
<td>88 bpm</td>
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<tr>
<td></td>
<td>Partial Pressure Brain Tissue Oxygenation</td>
<td>21.5 mmHg (20.0-40.0)</td>
<td>24.9 mmHg (20.0-40.0)</td>
<td>25.0 mmHg (20.0-40.0)</td>
</tr>
<tr>
<td></td>
<td>Intracranial Pressure</td>
<td>20 mmHg (0-20)</td>
<td>23 mmHg (0-20) H</td>
<td>24 mmHg (0-20) H</td>
</tr>
<tr>
<td></td>
<td>Patient Position</td>
<td>Prone</td>
<td>Prone</td>
<td>Prone</td>
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<tr>
<td></td>
<td>Blood Pressure Assessment Label</td>
<td>170/104 mm Hg H</td>
<td>177/94 mm Hg</td>
<td>168/83 mm Hg</td>
</tr>
<tr>
<td></td>
<td>Blood Pressure Systolic</td>
<td>170 mm Hg (100-180)</td>
<td>177 mm Hg (100-180)</td>
<td>168 mm Hg (100-180)</td>
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<tr>
<td></td>
<td>Blood Pressure Diastolic</td>
<td>104 mm Hg (60-100) H</td>
<td>94 mm Hg (60-100)</td>
<td>83 mm Hg (60-100)</td>
</tr>
</tbody>
</table>
Outcome

- Nitric Oxide/Inverse x 12 days
- Prone/Supine x 14 days
- Weaned from ventilator
- Day 30
  - Opens eyes
  - Moving all 4 extremities spontaneously
- Day 45
  - To Floor
  - Ambulating/Follows commands
  - Trache downsized
  - To ARU
- Day 64: D/C Home

- Was the cause of ARDS
  - Barbs + Hypothermia
  - Posterior Fossa Injury/NPE + Aspiration