Perioperative Controversies in Neuroanesthesia

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Director, Neuroanesthesia
Beth Israel Deaconess Medical Center
Boston, MA

I do not have any financial interests or relationships to disclose
The Perfect Neuroanesthetic

*How do we get there?*

- Facilitate surgical exposure
- Provide optimal neuroprotection
- Control intracranial pressure
- Blood pressure controlled (ideally SBP <140)
- Rapid emergence to assess neurologic exam
- Avoid coughing, straining and bucking that could lead to increased ICP
- Pain is controlled
NEUROPHYSIOLOGY:
Why I love neuroanesthesia

Cottrell’s Neuroanesthesia, 5th ed.
Objectives

• Present a concise review of cerebral physiology

• Review the management of elevated intracranial pressure

• Evidence-based approach to address several controversial areas in neuroanesthesia
Case Presentation #1

Equestrian kicked in head by her horse

Transferred to our medical center for emergency evacuation of epidural hematoma
Case Presentation #2

76 year-old man with Parkinson’s disease who was found to have a large right frontal mass

- Originally admitted to the neurological intensive care unit for observation

- Now with rapidly deteriorating neuro exam and surgeons want to proceed with emergent tumor debulking
Case Presentation #3

64 year-old woman POD#1 s/p CABG found to have a fixed and dilated right pupil.

Neurosurgeons are considering a right-sided decompressive hemicraniectomy
Brain in box

BRAIN 80%
BLOOD 10%
CSF 10%
ICP (mm/Hg) vs. Intracranial volume (mL)

Compliance = $\Delta V / \Delta P$
CPP = MAP-ICP
CPP ≠ CBF
Cottrell’s Neuroanesthesia, 5th ed.
Beach Chair Position May Decrease Cerebral Perfusion
*Catastrophic Outcomes Have Occurred*
Beach Chair Position May Decrease Cerebral Perfusion

Catastrophic Outcomes Have Occurred

by David J. Cullen, MD, and Robert R. Kirby, MD

Case Presentation

A 47-year-old, healthy female underwent general anesthesia for shoulder arthroscopy. Preoperative blood pressure (BP) was 125/83 mmHg. After premedication with 50 mg of meperidine, 40 mg hydroxyzine, and 0.2 mg glycopyrrolate intramuscularly, anesthesia was induced with 200 mg propofol, 100 mg succinylcholine, and 30 mg lidocaine. Because she was hypertensive, just prior to induction, 50 mg of labetalol was given intravenously. Anesthesia was maintained with 2% isoflurane, 60% nitrous oxide, and oxygen. The patient was placed in the "barbershop" position for the surgery. Twenty minutes into the case, blood pressure decreased to 100/60 mmHg and then remained in the 80-90 mmHg systolic range for the remainder of the case. Oxygen saturation was 100% and end tidal CO₂ values were in the 30s throughout the case. Upon arrival in the post-anesthesia care unit (PACU), her blood pressure was 113/60 mmHg but she did not awaken. Naloxone 0.1 mg was given intravenously, but she remained
Cerebral Perfusion and the Beach Chair Position

Conversion Factor: 1 cm rise = 0.75 mmHg drop in MAP

MAP = 50 mmHg

20 cm = 15 mmHg rise
MAP = 65 mmHg drop in MAP
Shoulder Surgery in the Beach Chair Position Is Associated with Diminished Cerebral Autoregulation but No Differences in Postoperative Cognition or Brain Injury Biomarker Levels Compared with Supine Positioning: The Anesthesia Patient Safety Foundation Beach Chair Study

Andrew Laflam, BS,* Brijen Joshi, MD,† Kenneth Brady, MD,‡ Gayane Yenokyan, PhD,§ Charles Brown, MD,* Allen Everett, MD,‖ Ola Selnes, PhD,¶ Edward McFarland, MD,# and Charles W. Hogue, MD*

Anesth Analg 2015; 120(1): 176
Clinical and Radiographic Signs of Elevated Intracranial Hypertension: Next Steps?
ICP treatment “ladder”

- Intubation, Normocarbic Ventilation
- Increased sedation
- Hyperosmolar Rx
- Induced Hypocapnia
- Hypothermia
- Metabolic Suppression (barbs)
- Decompressive Crani

Adapted from N Engl J Med 2014; 370: 2121-2130
Algorithm for the Treatment of Increased Intracranial Pressure (ICP).

CBF as a function of CO2

Am J Physiol Heart Circ Physiol
Is there a role for hypocapnia in acute brain injury?

-imminent brain herniation
-intraoperative use in neurosurgery
Question 1: For patients with severe TBI, does monitoring ICP improve mortality and neurological function?

Question 2: For patients undergoing intracranial procedures, does the choice of hyperosmolar agent matter?
Question 1: For patients with severe TBI, does monitoring ICP improve mortality and neurological function?

Question 2: For patients undergoing intracranial procedures, does the choice of hyperosmolar agent matter?
ICP Waveform Interpretation
Guidelines for the Management of Severe Traumatic Brain Injury
3rd Edition

A Joint Project of the

Brain Trauma Foundation
Improving the Outcome of Brain Trauma Patients Worldwide

and

American Association of Neurological Surgeons (AANS)
Congress of Neurological Surgeons (CNS)
AANS/CNS Joint Section on Neurotrauma and Critical Care

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Is there a role for ICP monitoring?

A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury

Randall M. Chesnut, M.D., Nancy Temkin, Ph.D., Nancy Carney, Ph.D., Sureyya Dikmen, Ph.D., Carlos Rondina, M.D., Walter Videtta, M.D., Gustavo Petroni, M.D., Silvia Lujan, M.D., Jim Pridgeon, M.H.A., Jason Barber, M.S., Joan Machamer, M.A., Kelley Chaddock, B.A., Juanita M. Celis, M.D., Marianna Cherner, Ph.D., and Terence Hendrix, B.A.

Chesnut R. NEJM 2012;367: 2471

Intracranial Pressure Monitoring in Brain-Injured Patients is Associated With Worsening of Survival

Shahid Shafi, MD, MPH, Ramon Diaz-Arrastia, MD, PhD, Christopher Madden, MD, and Larry Gentilello, MD

Shafi S. J Trauma 2008; 64:335

Effect of intracranial pressure monitoring and targeted intensive care on functional outcome after severe head injury*

Cremer O. Crit Care Med 2005; 33:2207
A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury

Randall M. Chesnut, M.D., Nancy Temkin, Ph.D., Nancy Carney, Ph.D., Sureyya Dikmen, Ph.D., Carlos Rondina, M.D., Walter Videtta, M.D., Gustavo Petroni, M.D., Silvia Lujan, M.D., Jim Pridgeon, M.H.A., Jason Barber, M.S., Joan Machamer, M.A., Kelley Chaddock, B.A., Juanita M. Celix, M.D., Marianna Cherner, Ph.D., and Terence Hendrix, B.A.
Pressure Monitoring Group
Clinical Exam Group

1. Diencephalic lesions
2. Third nerve (uncal herniation)
3. Midbrain lesions
4. Ponsine lesions
5. Tectal lesions
<table>
<thead>
<tr>
<th>Variable</th>
<th>Pressure-Monitoring Group (N = 157)</th>
<th>Imaging-Clinical Examination Group (N = 167)</th>
<th>P Value</th>
<th>Proportional Odds Ratio (95% CI)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients assessed at 6 mo — no. (%)</td>
<td>144 (92)</td>
<td>153 (92)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary outcome‡</td>
<td></td>
<td></td>
<td>0.49†</td>
<td>1.09 (0.74–1.58)</td>
</tr>
<tr>
<td>Median</td>
<td>56</td>
<td>53</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>22–77</td>
<td>21–76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cumulative mortality at 6 mo — %</td>
<td>39</td>
<td>41</td>
<td>0.60¶</td>
<td>1.10 (0.77–1.57)</td>
</tr>
<tr>
<td>GOS-E scale at 6 mo — no. (%) ‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>56 (39)</td>
<td>67 (44)**</td>
<td>0.40¶</td>
<td>1.23 (0.77–1.96)</td>
</tr>
<tr>
<td>Unfavorable outcome</td>
<td>24 (17)</td>
<td>26 (17)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Favorable outcome</td>
<td>63 (44)</td>
<td>60 (39)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
A trial of intracranial pressure monitoring in traumatic brain injury

Samer Melhem, Lori Shutter and A Murat Kaynar

University of Pittsburgh Department of Critical Care Medicine: Evidence-Based Medicine Journal Club, edited by Sachin Yende

Melhem et al. Critical Care 2014, 18:302
http://ccforum.com/content/18/1/302

EDITORIAL

Brain in a Box
Allan H. Ropper, M.D.

The brain, despite its sophistication, resides in a rudimentary container. The rigid cranium restricts enlargement of its contents, so that intracranial pressure rises rapidly as brain volume expands. When pressure becomes greatly elevated in the intensive care unit (ICU) and aftercare may not be similar to those used in North America and Europe. A second reservation regards the devices used to measure pressure. Unlike the external ventricular drains used in many
A trial of intracranial pressure monitoring in traumatic brain injury

Samer Melhem¹, Lori Shutter² and A Murat Kaynar²*

University of Pittsburgh Department of Critical Care Medicine: Evidence-Based Medicine

Recommendation
There is insufficient evidence at this time to abandon treatment based on ICP monitoring, and further studies need to be done before changing practice. ICP monitoring must be used as part of a multimodal approach to the patient and viewed as an additional tool available to the clinician to manage patients with TBI.

a valid approach. In the future there may be other means of detecting early compression of the brain stem. Until then, clinical methods are fine.

Brain in a Box
Allan H. Ropper, M.D.

The brain, despite its sophistication, resides in a rudimentary container. The rigid cranium restricts enlargement of its contents, so that intracranial pressure rises rapidly as brain volume expands. When pressure becomes greatly elevated in the intensive care unit (ICU) and after-care may not be similar to those used in North America and Europe. A second reservation regards the devices used to measure pressure. Unlike the external ventricular drains used in many
A Consensus-Based Interpretation of the Benchmark Evidence from South American Trials: Treatment of Intracranial Pressure Trial

Question 1: For patients with severe TBI, does monitoring ICP improve mortality and neurological function?

Question 2: For patients undergoing intracranial procedures, does the choice of hyperosmolar agent matter?
Hyperosmolar Agents

Mannitol (1960’s)
Hypertonic saline (1990’s)

- Brain 80% water, so brain volume responsive to change in water content

- Gradient depends on intact blood brain barrier

- Reflection coefficient: how solute is excluded from BBB

- Both effective in reducing ICP (starts 10-15 minutes, max effect in 20-60 minutes)
## Hyperosmolar Agents

<table>
<thead>
<tr>
<th>Mannitol</th>
<th>Hypertonic Saline</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>20% solution</strong></td>
<td>Variety of concentrations (3%-23.4%)</td>
</tr>
<tr>
<td>0.25-1.0 g/kg at 2-4 hour intervals</td>
<td>Infusion/bolus</td>
</tr>
<tr>
<td>Target serum osmolarity of 300-320 mOsm/L</td>
<td>Goal sodium levels 145-150 (initially)</td>
</tr>
<tr>
<td>Check osmolar gap to redose</td>
<td></td>
</tr>
</tbody>
</table>

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*Beth Israel Deaconess Medical Center*  
*Harvard Medical School*
### COMPARISON OF OSMOTHERAPY AGENTS

<table>
<thead>
<tr>
<th></th>
<th>Sodium bicarb 8.4%</th>
<th>Mannitol 20%</th>
<th>HTS 3%</th>
<th>HTS 23.4%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equi-osmolar dose</td>
<td>1ml/kg</td>
<td>0.5gm/kg</td>
<td>2.5ml/kg</td>
<td>30ml</td>
</tr>
<tr>
<td>Osmolarity</td>
<td>2002mOsm/L</td>
<td>1098mOsm/L</td>
<td>1025mOsm/L</td>
<td>8008mOsm/L</td>
</tr>
<tr>
<td>Infusion instructions</td>
<td>IV push over 5-10min</td>
<td>IVPB over 10-15min</td>
<td>IVPB over 10-15min</td>
<td>IVPB over 10-15min</td>
</tr>
<tr>
<td>Infusion site</td>
<td>Central preferred</td>
<td>Central preferred</td>
<td>Central preferred</td>
<td>Central</td>
</tr>
<tr>
<td>Infusion effects</td>
<td>Phlebitis, metabolic alkalosis</td>
<td>Phlebitis, hypotension</td>
<td>Phlebitis</td>
<td>Phlebitis, hypotension</td>
</tr>
<tr>
<td>Other considerations</td>
<td>Readily available in nearly every unit</td>
<td>0.22 micron filter</td>
<td>Osmotic diuretic</td>
<td>Avoid medication errors</td>
</tr>
</tbody>
</table>
Re-dosing mannitol: The osmolar gap

Serum Osmolarity

\[(2 \times \text{Sodium}) + (\text{glucose}/18) + (\text{BUN}/3)\]

Osmolar Gap

Measured Osmolarity – Calculated Osmolarity
Sodium requirement (in mmoles) =
(LBW in kg x 0.5 women, 0.6 men) x
(desired Na – current Na in mmol/L)

Volume (ml) = sodium requirement
sodium concn of solution
Example: 55 kg woman
   target Na=150
   current Na=139

? Volume using 3% NaCl (513 mmol/L)

Na req (mmol) = (55 * 0.5) (150-139) = 302

Volume (ml) = 302 / 513 = 589
Mannitol VS Saline

**Mannitol**
--hypokalemic, hypochloremic acidosis
a/w volume contraction/diuresis
--replace output with normal saline
--risk of acute kidney injury with high dose (>200 g/day)

**Hypertonic Na**
--Intravascular volume expansion
--Mild acidosis, hyperchloremia, hypokalemia
--lasix if high risk for CHF
Effect of equiosmolar solutions of mannitol versus hypertonic saline on intraoperative brain relaxation and electrolyte balance.

Rozet I¹, Tontisiri N, Muangman S, Vavilala MS, Souter MJ, Lee LA, Kincaid MS, Britz GW, Lam AM.

RESULTS: There was no difference in brain relaxation (mannitol = 2, HS = 2 points; P = 0.8) or cerebral arteriovenous oxygen and lactate difference between HS and mannitol groups. Urine output with mannitol was higher than with HS (P < 0.03) and was associated with higher blood lactate over time (P < 0.001, compared with HS). Cerebrospinal fluid osmolality increased at 6 h in both groups (P < 0.05, compared with baseline). HS caused an increase in sodium in cerebrospinal fluid over time (P < 0.001, compared with mannitol).

Comparison of equiosmolar concentrations of hypertonic saline and mannitol for intraoperative lax brain in patients undergoing craniotomy

A. Raghava, Prasanna Udupi Bidkar, M. V. S. Satya Prakash, and B. Hemavathy

Results:

Brain relaxation was comparable in two groups and there was no significant difference (P = 0.633). The number of brain conditions classified as perfectly relaxed, satisfactorily relaxed, firm brain, and bulging brain in the HS group was 8, 13, 3, and 1, respectively, whereas it was 5, 17, 3, and 0, respectively, in the M group. There was no significant difference in hemodynamic variables between the two groups except CVP at 30 min (P = 0.048). Compared with mannitol, hypertonic saline caused increase in the serum osmolality at 120 min (P = 0.008) and in serum sodium at 120 min (P = 0.001). Urine output was higher with mannitol than hypertonic saline (P = 0.001).
Intraoperative Neuroprotection

- Hypothermia
- Barbiturates
- Volatile anesthetics and nitrous oxide
Hypothermia

- Conceptually interesting as cerebral metabolic rate decrease by 7% for each 1°C drop in temp
- Since CMRO2 and CBF are coupled, hypothermia will decrease CBF, and cerebral blood volume
- Despite this, no outcome data to show that hypothermia effective outside of its use for neuroprotection after cardiac arrest
Mild Intraoperative Hypothermia during Surgery for Intracranial Aneurysm

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Hypothermia Group</th>
<th>Normothermia Group</th>
<th>P Value†</th>
<th>Odds Ratio (95% CI)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. Analyzed</td>
<td>No. with Score (%)</td>
<td>No. Analyzed</td>
<td>No. with Score (%)</td>
</tr>
<tr>
<td>Score for Glasgow Outcome Scale</td>
<td>499</td>
<td>329 (66)</td>
<td>501</td>
<td>314 (63)</td>
</tr>
<tr>
<td>1 (Minor or no disability)§</td>
<td>105 (21)</td>
<td>108 (22)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 (Moderate disability)</td>
<td>35 (7)</td>
<td>47 (9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 (Severe disability)</td>
<td>1 (&lt;1)</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 (Vegetative state)</td>
<td>29 (6)</td>
<td>32 (6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rankin score</td>
<td>499</td>
<td>333 (67)</td>
<td>501</td>
<td>318 (63)</td>
</tr>
</tbody>
</table>
| Score 0 or 1 (mild or no neurologic disability) | }
Barbiturates for acute traumatic brain injury (Review)

Roberts I, Sydenham E

Summary of main results

There is no evidence that barbiturates improve outcomes in people with acute brain injury. Barbiturate therapy results in a fall in blood pressure in one in four treated patients. The hypotensive effect of barbiturate therapy will offset any intracranial pressure lowering effect on cerebral perfusion pressure.
Effect of Nitrous Oxide on Neurologic and Neuropsychological Function after Intracranial Aneurysm Surgery


Table 4. Gross Neurologic and Neuropsychometric Outcome Results: Nitrous Oxide versus No Nitrous Oxide

<table>
<thead>
<tr>
<th>Metric</th>
<th>No Nitrous Oxide Group</th>
<th>Nitrous Oxide Group</th>
<th>Univariate Analysis P Value</th>
<th>Multivariate Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>GOS at 3 months: 1 vs. &gt;1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>627</td>
<td>373</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1: Minor or no disability</td>
<td>389 (62)</td>
<td>253 (68)</td>
<td>0.073</td>
<td>0.82</td>
</tr>
<tr>
<td>GOS at 3 months: 1, 2, 3, 4, 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>627</td>
<td>373</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1: Minor or no disability</td>
<td>390 (62)</td>
<td>253 (68)</td>
<td>0.066</td>
<td>0.84</td>
</tr>
<tr>
<td>2: Moderate disability</td>
<td>140 (22)</td>
<td>73 (20)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3: Severe disability</td>
<td>54 (9)</td>
<td>28 (8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4: Vegetative state</td>
<td>1 (0)</td>
<td>0 (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5: Death</td>
<td>42 (7)</td>
<td>19 (5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rankin score at 3 months: 0 or 1 vs. &gt;1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>627</td>
<td>373</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 or 1: Mild or no neurologic disability</td>
<td>394 (63)</td>
<td>256 (69)</td>
<td>0.305</td>
<td>0.84</td>
</tr>
</tbody>
</table>

*American Society of Anesthesiologists, Inc. Copyright © 2008, Lippincott Williams & Wilkins, Inc.
Perioperative Neurosurgical Complications and US

- Intracerebral hemorrhage
- Blood pressure management
- Delayed Emergence
Post-craniotomy Hemorrhage
Perioperative BP control: Does it matter?

Relation between Perioperative Hypertension and Intracranial Hemorrhage after Craniotomy

Ayman Basali, M.D.,* Edward J. Mascha, M.S.,† Iain Kalfas, M.D.,‡ Armin Schubert, M.D., M.B.A.§

Table 3. Incidence of Intraoperative Hypertension by History of Preoperative Hypertension

<table>
<thead>
<tr>
<th>Preoperative Hypertension</th>
<th>ICH Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>56.5% (12/23)</td>
<td>53.1% (17/32)</td>
</tr>
<tr>
<td>No</td>
<td>54.3% (25/46)</td>
<td>28.1% (19/103)*</td>
</tr>
</tbody>
</table>

Fig. 1. Temporal distribution of postoperative intracranial bleeding episodes for all 69 intracranial hemorrhage (ICH) patients.
Nicardipine Is Superior to Esmolol for the Management of Postcraniotomy Emergence Hypertension: A Randomized Open-Label Study

John F. Bebawy, MD, Christopher C. Houston, MD, Jenna L. Kosky, MD, Ahmed M. Badri, MD, Laura B. Hemmer, MD, Natalie C. Moreland, MD, Louanne M. Carabini, MD, Antoun Koht, MD, and Dhanesh K. Gupta, MD
Pain control: Scalp Block

"Scalp Block" During Craniotomy: A Classic Technique Revisited

Irene Osborn, MD and Joseph Sebo, PhD

J Neurosurg Anesthesiol • Volume 22, Number 3, July 2010
Delayed Emergence: My patient is not waking up!

• Non-anesthetic causes
  – Intracerebral hematoma
  – Seizures
  – Tension pneumocephalus
  – Cerebral edema
  – Stroke
  – Vessel occlusion
  – Metabolic or electrolyte disturbances
My “typical” neuroanesthetic

- 1:1 air:oxygen and ½ mac sevoflurane
- Remifentanil 0.05-0.3 mcg/kg/min
- Dexmedetomidine 0.4 mcg/kg/HR
  - Start as soon as feasible and stop 20 min prior to extubation
- Small amount of dilaudid (0.2-0.4 mg)
- Phenylephrine gtt for upper; Nicardipine gtt for lower
- All fluids in 0.9 NS
- Lasix/mannitol/decadron
- Consider transition to propofol TIVA at end of case
- +/- scalp block for postoperative pain
The Perfect Neuroanesthetist

Did we get there?

- Facilitate surgical exposure
- Provide optimal neuroprotection
- Control intracranial pressure
- Blood pressure controlled (ideally SBP <140)
- Rapid emergence to assess neurologic exam
- Avoid coughing, straining and bucking that could lead to increased ICP
- Pain is controlled
Conclusions

-The anesthesia team plays a central role in improving the outcome associated with acute brain injury.

-A neurophysiological approach to guide therapy makes intuitive “sense” but several key tenets remain to be proven.

-Superiority of hypertonic saline vs mannitol is unclear, but the clinical condition of the patient needs to be considered.
Thank you