Course: Advanced Neurosonology – ICP mgmt. with TCD

Gregory Kapinos, MD, MS, FASN, FCCM
Neurologist, Neurointensivist, Neurophysiologist, Neurosonologist, Neuroimager.
Acute Surgical Cranial Trauma: “To drill or not to drill”

#10013 on 7/1/2015 at 11AM

Gregory Kapinos, MD, MS,
Ali Sadoughi, MD,
Jamie S. Ullman, MD, FACS, FAANS,
Raj K. Narayan, MD, FACS, FAANS.
TOOLS OF THE TRADE: NEUROMONITORING

In: SYM3 - NEUROCRITICAL CARE & NEUROSURGICAL EMERGENCIES UPDATE

Gregory Kapinos, MD, MS, FASN
Associate Professor, Neurology,
SUNY Downstate School of Medicine.
Director, Neurocritical Care Services & Critical Care Neuromonitoring,
NYC H+H Kings County.
Stylistic influences

Sub-Internships

Research: EEG/Epilepsy

Research: Stroke MRI

Clinical Fellowship: Neurocritical care

NewYork-Presbyterian

The University Hospital of Columbia and Cornell

Clinical Fellowship: Bioethics & Health Law
Disclosures

- I am a neurologist (ABPN-BC), neurointensivist (UCNS-BC), neurosurgical intensivist (CAST/SNS-BC), neurosonologist, neuroimager (UCNS-BC), with additional competencies in advanced neuroimaging, critical care EEG, neurotraumatology, neurocoagulation, as well as in neurohospital medicine, emergency medicine, palliative care & bioethics.

- I am a board member of the editorial board of the JON and of the BOD of the ASN.

- I have no financial ties to any commercializing company of any neuromonitoring probes or transcranial doppler, any neuroimaging company, or any pharmaceuticals.

- I am a non-paid advisor for a processed EEG company (Ceribell), as well as a consultant and paid speaker for a cooling company (Zoll).

- I have received honoraria from BARD for devising new venous access catheters.

- I have a commitment to a stipend and royalties in an upcoming Springer book on new frontiers in quantitative EEG.
Topic-focused Disclosures

- No financial ties to any marketed technology for trephination, TCD, neuroimaging or hemodynamic monitoring.
- No financial ties to any company marketing IVF, albumin or vasoactive drugs.
- Received <200$ honorarium from company marketing vascular access devices.
- Trademarked “Advanced Neurological Life Support (ANLS)” but not in commerce.
- Was involved in CTP research in vasospasm with PI Pina Sanelli, who was receiving AHA, BAF and NIH funding.
PIERCING THROUGH THE SKULL FOR ICP AND CPP WITHOUT A DRILL

Gregory Kapinos, MD, MS.
Key messages

- No, TCDs are not ready to replace direct ICP monitors, however...
- Compliance is more important than the ICP value
  - With pressure monitor: P2>P1, MOCAIP, etc…
  - With TCDs or regular U/S: PI, ONSD, novel index
- It is judicious to tease out the primordial inadequacy for that pt:
  - high ICP (low compliance),
  - or low CPP (low perfusion),
  - or both
  - or none
- TCD do offer advantages b/o non-invasiveness for
  - Prognosticate in ED death and poor functional outcome after TBI
  - Triaging for decision on ICU and recourse to EVD/ICP monitor
  - Teasing out better if compliance or perfusion is most compromised
  - Earlier detection of imminent crisis, as c/t using ICP/CPP threshold
  - Allocate Rx specific to the primordial inadequacy (tailored individualized goal-directed Rx for cerebral edema/ICP)
EVD not panacea

A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury


- We got it all wrong with our EVDs in the modern world???
- Or is it just an inadequate threshold to define ICP crisis?
- Should we abandon looking at ICP alone?
- Still has an adjunctive value to vasoreactivity (PRx/Mx), cerebroximetry (PbtO2) and we need to go metabolic (LPR by CMD, MRS) and neurophysiologic (qEEG, SSEPs)?
Introduction: ICP not the panacea

BARKING AT THE WRONG TREE: Analogical > Digital data...
Should we Rx ICP mean or low compliance?


What shapes pulse amplitude of intracranial pressure?

Source
Academic Neurosurgical Unit, Department of Clinical Neurosciences, Addenbrooke's Hospital, University of Cambridge, Cambridge
Compliance before ICP

Split in neurosurgeons… Which scenario would you treat? Favoring ICP mean or waveform?
Flourishing science on understanding better interaction of CSF volume onto the edematous or expanded parenchyma and the effect of each heart beat, testing vascular resistivity and parenchymal compliance:

- Pulse Amplitude, Pulsatility Index, Resistivity Index, MOCAIP…
Perfusion status on EVD WF

MOCAIP from UCLA: Beyond compliance, VD/VC state, CAR...

Complex... Let’s start simple: P2>P1

The figure visualizes four MOCAIP metrics pairs of ICP and CBFV signals, which are significantly correlated.
In France, neurosonology is embraced by neurosurgery field…

2008, Paris & Toulouse:
Triaging severe TBI in ED:
Vd<20 or PI>1.4 = Nsurgical care
When and how to use transcranial Doppler

Transcranial Doppler

Vigue et al.

Pulsatility index $PI = \frac{(V_s - V_d)}{V_m} = \frac{(V_r - V_d) \cos \alpha}{V_{mr} \cos \alpha}$
Neurosonology for TBI at NSLIJ

Good thing about PI: angle doesn’t matter…
Good thing about cosinus: we only underestimate…
PI not the panacea…

PI actually not very well correlating with gold-standard ICP by EVD…

We still do not have a reliable and validated noninvasive technique that can provide an accurate quantitative measurement of intracranial pressure (ICP) that could replace invasive quantitative measurements of ICP.

Razumovsky A, Armonda RA.

Transcranial Doppler pulsatility index: not an accurate method to assess intracranial pressure.
Behrens A, Lenfeldt N, Ambarki K, Malm J, Eklund A, Koskinen LO.

Because the correct “gold-standard” is compliance, not ICP!
Needed distinction by **extricating PI from Vd (or EDV):**

PI addresses a primary ICP (or actually compliance) issue, which is really what we care about… is there something taking too much space in the brain or is the brain too edematous…

Rx? CSF diversion, tumor/blood evacuation, craniectomy or osmotherapy.

**vs.** Vd depicts a primary CPP issue with inadequately low CPP, the beginning of compromised CBF, and if it persists, VD cascade will ensue with subsequent ischemia and rise in ICP too…

Rx? the issue is the low MAP and Rx should consist of course of raising MAP, not only to reach an optimal individualized CPP goal but also because this change by itself can intrinsically lower the ICP…
Hypertension Can Drive Elevated Intracranial Pressure

... But so can hypotension!
Now it is easy to understand why 2 schools of thoughts fight for no reason (Lund vs. Houston approaches)…

But they are both right for a specific subset of patients…

I propose to create an individualized Rx tailored to ICP vs. CPP being the preponderant issue…
I'm not a PI-advocate...

PI is the best!
It goes up in the VD cascade too...

Assessment of the lower cerebral autoregulatory threshold

1S. B. Lewis MBBS, 2M. L. H. Wong B Med Sc (Hons), 3P. E. Bannan FRACS, 3I. R. Piper PhD, 3P. L. Reilly MD

1Department of Neurosurgery, Sir Charles Gairdner Hospital, Verdun St, Nedlands 6009 Western Australia
2Department of Neurosurgery, Royal Adelaide Hospital, North Terrace, Adelaide, SA 5000, Australia

Summary Continuous transcranial Doppler ultrasonography of the middle cerebral artery (TCD-MCA) has been proposed as an alternative method for assessing cerebral autoregulation compared with the transcranial Doppler ultrasonography response curve method.

Yes, but its sensitivity for detecting an elevated ICP or a poor compliance is also its weakness...

This study conformed to the guidelines established by the National Health and Medical Research Council and was approved by the Animal Ethics Committees of the Royal Adelaide Hospital, University of Adelaide and Institute of Medical and Veterinary Science.

RESULTS

There was neither hypoxia nor sustained hyper- or hypocarbia over the experimental period for any animal. As CPP decreased, CBF was initially maintained until a sharp breakpoint occurred at a mean value of 50 ± 1.5 mmHg CPP. The grouped CBF data and the mean breakpoint are presented in Figure 2. The lower limit of autoregulation indicated by the CBF breakpoint ranged from 41 to 59 mmHg CPP. Similarly, as CPP fell, systolic Doppler flow velocity was preserved until a mean breakpoint of 50 ± 1.8 mmHg CPP (range 38–60 mmHg CPP), shown in Figure 3. In contrast, the mean diastolic Doppler flow velocity decreased to a mean breakpoint of 68 ± 3.2 mmHg CPP (range 57–76 mmHg CPP)
CPPe in landmark discoveries

- CPPe correlates to MFV/(PSV-EDV)
- PI = (PSV-EDV)/MFV
- Thus CPPe correlates to 1/PI
Estimates of ICP and CPP by Aaslid et al. and Klingelhofer et al.

- ICP/MAP correlates to RI/MFV
- Thus $ICP_e = \frac{MAP \times RI}{MFV}$
- And $CPP_e = \frac{MFV}{FV^1} \times MAP^1$
RI and EDV more relevant for CPP adequacy

Peaked my interest in FVd (EDV)!
Importance of EDV

Systolic Doppler breakpoint (50 ± 2 mmHg)
Forgotten germane parameter: EDV

Se test to detect adequacy of CPP to give CBF

Only once in the midst of the VD cascade…
Rx’ic implications: Opposite goals for MAP

Lund vs. Houston: ICP>>CPP vs. CPP>>ICP
Or a little bit of both...

The issue of Ischemia vs. Edema deserves to tease out PI from EDV...
Lumped PI and EDV...

We propose:

polytrauma → TCD with "fast echo"

Vd ≤ 20 cm/s
PI > 1.4

Increase MAP
Mannitol / hypertonic saline
CT scan / neurosurgeons

Vd > 20 cm/s
PI < 1.4

Increase MAP, Hb,
PaCO2
Follow-up +++

very worried

worried

NTD
Transcranial Doppler ultrasound goal-directed therapy for the early management of severe traumatic brain injury

<table>
<thead>
<tr>
<th></th>
<th>Abnormal admission (n = 11)</th>
<th>Normal admission (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T0</td>
<td>T1</td>
</tr>
<tr>
<td>Delay from admission (min)</td>
<td>16 ± 8</td>
<td>219 ± 96</td>
</tr>
<tr>
<td>Abnormal TCD (n)</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>Mean velocity (cm/s)</td>
<td>30 ± 6</td>
<td>43 ± 10*</td>
</tr>
<tr>
<td>Diastolic velocity (cm/s)</td>
<td>13 ± 5</td>
<td>25 ± 8*</td>
</tr>
<tr>
<td>Pulsatility index</td>
<td>2.1 ± 0.5</td>
<td>1.4 ± 0.3*</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>89 ± 15</td>
<td>105 ± 17**</td>
</tr>
<tr>
<td>ICP (mmHg)</td>
<td></td>
<td>32 ± 13</td>
</tr>
<tr>
<td>CPP (mmHg)</td>
<td>73 ± 15</td>
<td>71 ± 14</td>
</tr>
<tr>
<td>SjvO2 (%)</td>
<td>67 ± 2</td>
<td>72 ± 9</td>
</tr>
<tr>
<td>pH</td>
<td>7.39 ± 0.04</td>
<td>7.39 ± 0.02</td>
</tr>
<tr>
<td>PaCO2 (mmHg)</td>
<td>40 ± 5</td>
<td>42 ± 5</td>
</tr>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>12 ± 1</td>
<td>11 ± 1</td>
</tr>
<tr>
<td>Norepinephrine (n)</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Mannitol (n)</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Neurosurgery (n)</td>
<td>-</td>
<td>3</td>
</tr>
</tbody>
</table>

* p < 0.05 between T0 and T1
** p < 0.05 between groups
MY PROPOSAL: nuanced Rx

Rx tailored not to the primary issue (ICP vs CPP) alone, but to both issues...

Segregate pts into 2x2 simple table to allocate the best Rx
<table>
<thead>
<tr>
<th>Best Diastolic Flow Velocity / Pulsatility Index (PI)</th>
<th>PI &lt; 1.2</th>
<th>PI &gt; 1.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vd &gt; 40</td>
<td><strong>Compliant brain</strong> (unlikely to have significantly raised ICP), <strong>well perfused</strong> (likely adequate CPP): Treat by continuing current meds, current ventilator settings and same MAP goal in an <strong>Intermediate</strong> level of monitoring.</td>
<td><strong>Poorly compliant brain</strong> (likely edematous or with raised ICP), but <strong>well perfused</strong> (with likely adequate CPP): Treat with <strong>osmotherapy</strong> (prefer mannitol over HTS), consider <strong>mild hyperventilation</strong> and try mild decrease in MAP (to reduce potential edema) in NSICU.</td>
</tr>
<tr>
<td>Vd &lt; 40</td>
<td><strong>Compliant brain</strong>, but <strong>hypoperfused</strong> (with likely inadequately low CPP): Treat with <strong>no osmotherapy</strong> (but if mandated by herniation, prefer HTS over mannitol), <strong>mild hypoventilation</strong> (a.k.a. permissive hypercapnia), and definitely raise <strong>MAP</strong> goal (with volume and pressors) in NSICU.</td>
<td><strong>Poorly compliant brain</strong> (likely edematous or with raised ICP), not well perfused (with likely inadequately low CPP): Treat with <strong>osmotherapy</strong> (with HTS, not mannitol), normal ventilation and definitely raise slowly <strong>MAP</strong> goal (with volume and pressors) in NSICU.</td>
</tr>
</tbody>
</table>
**TRIAGING, TREATING AND PROGNOSTICATING outcomes**

**Marseille + Bicêtre, n=151**

Time from admission to TCD : 16±10 min

- **Pl>1.4 Vd<20 cm/s**
- **Pl>1.4 Vd>20 cm/s**
- **Pl<1.4**

![Bar chart showing outcomes](image)

**Fig. 2** Three-month Glasgow Outcome Score in patients with abnormal admission TCD (filled columns) and in patients with normal admission TCD (open columns). 1 Good recovery, 2 moderate disability, 3 severe disability, 4 vegetative state, 5 dead

Our Material & methods @NSLIJ 2013:

TCD for non-invasive ICP monitoring in 5 patients in our ICU with acute cerebral edema and risk for ICP-related secondary deterioration from global ischemia.

Deemed non-salvageable, non-surgical or at high bleeding risk for EVD.

• 1 hepatic failure with GCE
• 1 meningitis with GCE and hemispheric IPH with MLS
• 1 TBI (moderate, no surgical lesion, compensated DIC)
• 2 large hypertensive IPH with mass effect.

TCD was used to derive PI as a surrogate marker for brain compliance and diastolic velocity ($V_d$) reflecting diastolic CBF inferring adequacy of CPP.

Applied Rx’ic choices falling under the 4 described categories to specifically address the cerebral needs of each group. (Table)

Measured adequacy of Rx by appreciating the response of each patient in terms of clinical stability, normalization of derived ICP parameters, decrease in GCE and absence of secondary ischemia.
Our Results:
PROOF OF CONCEPT / FEASIBILITY

- 1 Pt had no change in mgmt b/o normal PI and Vd.
- 2 pts received HTS as favored osmotherapy along with induced HTN to alleviate the risk of ischemia due to raised ICP.
- 1 pt received mannitol and had vasopressors tapered off to address break-through pressure edema driving her ICP (Lund).
- 1 pt received hemodynamic augmentation for significant CPP amelioration, without any significant rise in PI (Houston).
- All patients had normalization of PI and Vd within our target range within an hour of the tailored therapy.
- No patient had neurological deterioration, worsening of GCE, MLS, new hemorrhage or developed infarcts within 48h of our repeated interventions.
- All succumbed to their brain injury before discharge from the ICU, except for the TBI patient.
Conclusions

- PI and waveform analysis more informative than mean ICP.
- Compliance is a better gold standard to decide on osmotherapy.
- Extracting PI from EDV may make sense to tease out ICP vs CPP issues.
- TCD easily predict/detect early ICP/CPP issues in the ED.
- My 4-category-tailored goal-directed Rx makes sense, seems beneficial and not detrimental.
- Larger feasibility study with pts with EVDs, then RCT to compare conventional monolithic ICP therapy (mainly osmotherapy and MAP augmentation for all) to this 4-category tailored ICP therapy.
- It can then be even more refined, adding ONSD, RI, novel index, or adding CAR (vasoreactivity testing), MOCAIP (VD/VC state), optimal CPP (PRx or pbtO2), LPR (CMD), OEF and spectroscopy (neuroimaging).
Enhanced accuracy:
NIRS, SVJO2, CBF
ONSD, CBF H2O CONTENT
ameliorate PI & RI, MOCAIP…

Stephan A. Mayer, MD
Arterial and arteriolar resistance

- Pulsatility Index (PI): $\frac{Vs-Vd}{\text{mean}}$
- PSV-EDV / $(2\text{EDV}+\frac{1}{3}\text{PSV})$
- Marek Czosnyka (Cambridge)

- Resistivity Index (RI): $\frac{Vs-Vd}{Vs}$
- Leandre Pourcelot (Tours)
RI better than PI?

- RIx100 correlates to shunt malfunction:
  - Normal ~50% vs. sx’ic ~70%
  - Chadduck et al.

Klingelhofer et al.: RI is important in discerning whether pts are at risk for poor CPP from VSP or raised ICP.

RI may change as a function either ICP or MFV:

At a fixed MFV, RI rises when ICP rises and falls when ICP falls. At a fixed ICP, however, RI is 1/MFV.

When RI<0.5 and MFV>120, ICP is always <20. But when ICP>20 and MFV<150, RI is always > 0.6.

“Thus, when RI<0.5, changes in MFV reflect severity of VSP and ICP is expected to be low. But if RI rose to >0.6 and MFV declines simultaneously in a pt with VSP, there is a problem with ICP rise compromising CPP.”
Refining compliance and perfusion adequacy biomarkers

- RI better than PI?
- RI/MFV?
- Trends rather than thresholds?
- Moving correlation coefficients?
- Active dynamic better than passive Mx?
- Challenges (BHI, acetazolamide, carbogen)?
Term “cerebral autoregulation”:
- Pressure autoregulation vs. CO2 reactivity
  - One does not predict the other...
- CO2 reactivity in direction of VC by HV (drop in CO2) vs. CO2 reactivity in direction of VD by apnea (BHI accumulating CO2)...
  - One does not predict the other...

“Loss vs. conserved” CAR is too Manichean:
- F/u trends gradual amenutising CO2 reactivity might predict VSP/DCI or impaired compliance even better than overt impairment of vasoreactivity
- Peak slope of decrease or EDV trends and correlation coefficient RI/MAP might correspond better to adequacy of CPP irrespective of ICP
We should do more TCDs in the Neuro-ICU:

- TCD to tailor BP in AIS to evaluate:
  - Recanalized post IV tPA but also w/o tPA (penumbral salvage by HDA)
  - RI inferring VD in capillary bed
  - CBF conservation/restoration
  - Collaterals status
  - Reserve in all ischemic pts (not only Moya-Moya) by acetazolamide
- Hyperemia, risk of severe edema and HT

In order to tailor BP and AC Rx
Revisiting Laaessen’s and Czosnyka’s curves and K:
CI replaces MAP/CPP, better PRx/Mx and ORx/pbtO2-K and then what if API replaces CBF or neuronal fxn/clinical exam...
Figure 12: Post-bleed day 15 (at the peak of vasospasm), head CT reveals no lucency to suggest any new infarcted territories, but persistent global cerebral edema.
Thank you!

kapigreg@gmail.com

Fellowships

NCC+ICUEEG+NI+Nu/s