Neurosonology in the NSICU
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Disclosures
• No disclosures to report

Outline
• Transcranial Doppler
  • Vasospasm Monitoring
  • Increased ICP
  • Brain Death
  • Emboli Detection
• Carotid Duplex
  • POCUS
• Optic Nerve Sheath Diameter
  • POCUS

Transcranial Doppler
• Insonation Windows
  • Transtemporal
  • Transorbital
  • Transforaminal

Summary of findings
Subarachnoid Hemorrhage (SAH):

<table>
<thead>
<tr>
<th>INDICATION</th>
<th>SENSITIVITY (%)</th>
<th>SPECIFICITY (%)</th>
<th>REFERENCE STANDARD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasospasm after Spontaneous</td>
<td></td>
<td></td>
<td>Conventional</td>
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<tr>
<td>Subarachnoid Hemorrhage</td>
<td></td>
<td></td>
<td>angiography</td>
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<tr>
<td>Intracranal ICA</td>
<td>25-30</td>
<td>83-91</td>
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<tr>
<td>MCA</td>
<td>39-94</td>
<td>70-100</td>
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<tr>
<td>ACA</td>
<td>13-71</td>
<td>65-100</td>
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<tr>
<td>VA</td>
<td>44-100</td>
<td>82-88</td>
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Recommendations: TCD is useful for the detection and monitoring of angiographic VSP in the basal segments of the intracranial arteries, especially the MCA and BA, following sSAH (Type A, Class I-II evidence).
More data are needed to show if TCD affects clinical outcomes in this setting (Type U).

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Summary of findings
Subarachnoid Hemorrhage (SAH)
(continued)
3. Transcranial Doppler is reasonable to monitor for the development of arterial vasospasm (Class IIa; Level of Evidence B)

Table 1. Demographic, Treatment, and Monitoring Characteristics According to TCD Examination in 4576 Patients with aSAH

<table>
<thead>
<tr>
<th>Variable</th>
<th>TCD</th>
<th>TCD +</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>49</td>
<td>50</td>
<td>0.999</td>
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<tr>
<td>Sex, female</td>
<td>3051</td>
<td>3259</td>
<td>0.031</td>
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<tr>
<td>Charlson Comorbidity Index</td>
<td>9</td>
<td>9.5</td>
<td>0.036</td>
</tr>
<tr>
<td>Location of aneurysm, posterior fossa</td>
<td>7</td>
<td>7.5</td>
<td>0.036</td>
</tr>
<tr>
<td>Discharge, discharge</td>
<td>H</td>
<td>2</td>
<td>0.019</td>
</tr>
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<td>2</td>
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</tr>
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</table>

Up to 70% of SAH patients develop angiographic vasoconstriction.

Only 20–30% develop DCI.

Cerebral infarction sometimes develops in the absence of demonstrable vasoconstriction, or in a vascular territory unaffected by vasospasm.

Prevaling hypotheses:
- Large-vessel narrowing with subsequent low flow.
- Early brain injury (EBI) [multiple physiological derangements that are thought to occur in the first 72 hours after the ictus. Initial ICP crisis and global hypoperfusion trigger glial activation, endothelial dysfunction, and inflammatory pathway.
- Microcirculatory dysfunction with loss of autoregulation.
- Cortical spreading depolarization (CSD).
- Microthrombosis.

Table 1: Components of Brain Invasibility Index (BII) (SAH day 0–14).

<table>
<thead>
<tr>
<th>Components</th>
<th>SAH day 0</th>
<th>SAH day 1</th>
<th>SAH day 2</th>
<th>SAH day 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microvasculature</td>
<td>Decreased flow</td>
<td>Decreased flow</td>
<td>Increased flow</td>
<td>Normal flow</td>
</tr>
<tr>
<td>Cerebral Blood Flow</td>
<td>Decreased flow</td>
<td>Decreased flow</td>
<td>Increased flow</td>
<td>Normal flow</td>
</tr>
<tr>
<td>Intracranial Pressure</td>
<td>Increased pressure</td>
<td>Increased pressure</td>
<td>Increased pressure</td>
<td>Normal pressure</td>
</tr>
</tbody>
</table>

Table 2: Temporospatial Grading Criteria for Bladder Artery Stenosis.

<table>
<thead>
<tr>
<th>Temporospatial Grading Criteria</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/9</td>
<td>Vesicles</td>
</tr>
<tr>
<td>2/9</td>
<td>Multifocal microvascular abnormalities</td>
</tr>
<tr>
<td>3/9</td>
<td>Normal appearance</td>
</tr>
</tbody>
</table>

TBI

1. Primary Injury – direct damage due to mechanical forces including diffuse axonal injury.
   - Hallmark of severe traumatic brain injury.
   - Diffuse involvement of adjacent regions of brain during acceleration and deceleration.
   - DCI is caused by prolonged CSD after TBI, probably due to disruption of ascending reticular activating system to cortex.
   - Angular forces + Oblique Sagittal Forces

2. Secondary Injury – cascade of cellular and molecular processes including damage due to hypotension, hypoglycemia, hypoxia, and high intracranial pressure.
Traumatic Brain Injury and Raised Intracranial Pressure

• TBI may lead to hypoperfusion (day 0), hyperemia (days 1–3), vasospasm (days 4–15), and raised ICP
• TCD can avoid use of invasive CBF measurement techniques and provide similar prognostic information.
• MCA MIV of < 35 cm/s within 72 hours of head injury has been shown to predict unfavorable outcome at 6 months
• However, on multivariate analysis, this association was significantly less with initial GCS being a stronger predictor of outcome
• 50 patients with TBI monitored in the first 7 days, the vasospasm and hyperemia groups experienced a poor outcome at 6 months (GOS 1–3).

A significant correlation between PI and ICP (correlation coefficient 0.938 \( P < 0.0001 \) ) was demonstrated in a group of 81 patients who underwent TCD MCA PI measurements combined with invasive ICP measurements
• ICP = (11.1 X PI)−1.43, which could determine an ICP via the PI within 4.2mmHg of the actual ICP, which is reasonably accurate.
• ICP of > 20mmHg could also be determined with 89% sensitivity and 92% specificity
• PI \( \geq 1.56 \) predicted 83% of patients who had a poor outcome at 6 months, whereas a PI \( \leq 1 \) identified 71% of patients with a good outcome (GOS 4–5).

Summary of findings
Increased Intracranial Pressure (ICP) and Cerebral Circulatory Arrest

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Cerebral Circulatory Arrest and Brain Death</td>
<td>91-100</td>
<td>97-100</td>
<td>Conventional angiography, EEG, clinical outcome</td>
</tr>
</tbody>
</table>

Recommendation: TCD is a useful adjunct test for the evaluation of cerebral circulatory arrest associated with brain death (Type A, Class II evidence).

What is brain death?

• Case of Melanie Bacchiochi, Connecticut
• Transcripts of the hearing
  “when the soul leaves the body”
  “the loss of our ‘personal identity’”
  “Loss of vital fluid flow”
  “Putrefaction is the only sure sign of death”
  “disintegration of individual organ and tissue”

ICP Monitoring Techniques

• Types of Monitoring Devices
  • Fluid-filled transduced ventriculostomy
  • Fiberoptic sensors
  • Microchips (internal strain-gauge devices)
  • Air pouch technologies
• Locations of Monitoring Devices
  • Ventricular
  • Parenchymal
  • Subdural

Complications
• Infection
• Hemorrhage
• Breakage
• Malfunction of the device
• Difficulty with placement
• Difficult to assess the true rate of some of these issues
• Variability of definitions used in the literature when reporting infections and hemorrhage
1968 Harvard Criteria of Brain Death

- Defined as the irreversible loss of function of the whole brain, including the brainstem.
- Brain death is a clinical diagnosis
  1) Coma/Unresponsiveness
  2) Absence of all brainstem reflexes
  3) Apnea
- Exclusion
  - Of complicating medical condition (severe electrolyte, acid base or endocrine disturbance)
  - Drug intoxication or poisoning
  - Hypothermia (core temperature ≥ 35 C or 90 F)

Pathophysiology of Brain Death

- Loss of respiratory function
- Hemodynamic instability
  - Cardiac arrhythmias
  - Loss of vasomotor control
  - Loss of temperature regulation
- Endocrine imbalances
  - ADH, T3/T4, insulin, cortisol
- Electrolyte disturbances

Consequences of Brain Death

- Arrhythmias
- Hypotension
- DIC
- Acidity
- Pulmonary edema
- Hypothermia
- Cardiovascular collapse

Brain Death

- Brain Death interval increased
  - % Donor decreased 57% - 45%
  - % Cardiac Arrest 3.9% - 7.4%
  - % Family decline 23% - 36%

Organ Donation Statistics

Transplantable organs: heart, kidneys, liver, lungs, pancreas, small intestine
Transplantable tissues: blood, blood vessels, bones, bone marrow, cartilage, connective tissues, eyes, heart valves, skin
- 123,000 adults and children are on the waiting list
- 3% of all deaths are brain dead
- 21 people on average die each day waiting for an organ
- In 2013, 14,257 Donors resulted in 28,953 transplants
- One donor can save up to 9 lives. The same donor can save/improve the lives of hundreds by donating tissues.
- Success rate is now 80-90%

"Confirmatory" test for brain death

Brain death is a clinical diagnosis
These tests are not mandatory & are options
- Cerebral angiography
- Electroencephalography (EEG)
- Transcranial Doppler Ultrasonography
- Technetium 99 brain scan
1,400 TCD examinations in 623 pts
- November 2004 to June 2010
- 603(9.6%) excluded, inadequate windows
- SAH 43.5%
- Head injury 32.5%
- Intraparenchymal hemorrhage 6.5%
- Other pathological conditions 17.5%
- TCD patterns of CCA detected in 56 pts (9%)
  - Anatomic locations by brain CT
    - 41 supra-infratentorial lesions
    - 13 supratentorial lesions
    - 2 infratentorial lesions.
  - 26/56 showed hemodynamic patterns of BD at the first TCD examination
  - 30 pts were evaluated by daily TCD until the detection of TCD patterns of BD
  - 33 TCD examinations showed an oscillating flow pattern
  - 23 examinations showed a systolic spikes pattern

TCD patterns diagnostic of irreversible CCA
- Reverberating flow with retrograde flow in diastole
- Short systolic spikes
- Complete absence of any TCD signal.

28 ICP monitoring (mean ICP 83.9 mmHg ± 26.3; range: 15–136 mmHg)
- 17 (60%) had an intraparenchymal probe
- 8 (30%) an endoventricular probe
- 3 (10%) a subdural device.
- On comparing mean ICP values with the two TCD patterns of BD, no significant differences were found between pts with reverberating flow signal and those with systolic spike pattern.
- 91% and a specificity of 100%
- In 5 pts, false-negative TCD patterns were detected, showing diastolic flow in at least one artery
  - 1 was affected by carotid-cavernous fistula
  - 4 were treated by therapeutic decompressive craniotomy.

Summary of findings
Detection of Cerebral Microemboli

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</tr>
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<tbody>
<tr>
<td>Cerebral Microembolization</td>
<td></td>
<td></td>
<td>Experimental model, pathology, magnetic resonance imaging, neuropsychological tests</td>
</tr>
</tbody>
</table>

Recommendation: TCD is probably useful to detect cerebral microembolic signals in a wide variety of cardiovascular/cerebrovascular disorders/procedures (Type II, Class II–IV evidence).
However, data at present do not support the use of TCD for diagnosis or for monitoring response to antithrombotic therapy in ischemic cerebrovascular disease in these settings (Type U).

TCD Embolus Detection
Clinical Applications in Ischemic Stroke

- **Diagnosis**
  - Cardioembolic sources
    - Atrial fibrillation
    - Myocardial infarction
    - Prosthetic heart valves
    - Atrial septal defect
    - Patent foramen ovale
  - Arterial sources
    - Carotid atherosclerotic: symptomatic/asymptomatic
    - Dissection
- **Treatment**
  - Guide to intensity of antithrombotic therapy
  - Ultrasound to guide and enhance enzymatic fibrinolysis

Identification Criteria
- Transient (usually < 300 milliseconds)
- High amplitude (> 3 dB higher than background blood flow
- Unidirectional velocity spectrum
- Audible output — “chirp,” “snap,” “moan”

- Characteristics of Artifacts
  - Rough, broad, nonharmonic sound
  - Bidirectional: signal intensity increases simultaneously in both directions
  - Coincident with probe impacts, sudden motion, or electrical switching transients
  - Appear simultaneously at two different sites
Techniques

- Types of monitoring
  - Unilateral vs. bilateral
- Duration of monitoring
  - Minutes vs. hours
- Single gated vs multigated
  - Power Mmode (PMO)

Mechanical Heart Valves

- Generate microbubbles via microcavitation
- Micro aeroemboli of no pathogenic consequence
- 5 patients, 30 minutes monitoring
- Median MES
  - Resting: 9 (per 30 min)
  - 100% O2: 0 (per 30 min)

Mechanical Heart Valves

- More MES in those with a history suggestive of stroke
- More MES with valve obstruction
- Majority of MES are gaseous
- Overall not a good indicator of stroke risk

Left Ventricular Assist Devices

- MES varies from 20% to 100%
- Novacor N100
  - MES correlate with stroke risk
  - Antiplatelet and anticoagulation reduce MES
  - Reduction in MES correlated with stroke risk
- DeBakey
  - MES not correlated with stroke risk
  - Oxygen inhalation reduces numbers

POCUS Carotid

What is POCUS?
The visual stethoscope?

Respiratory variation in carotid peak systolic velocity predicts volume responsiveness in mechanically ventilated patients with septic shock: a prospective cohort study

- Single-center, prospective, cohort study.
- Mechanical ventilation, septic shock, and hemodynamic instability for which the
- MICU/SICU tertiary academic hospital from May-October 2014.
- ACVC mechanical ventilation was performed with TV 6 mL/kg
- Fluid challenges with NS at a 7 mL/kg dose over a 30-min period
- Thermodilution before and after each challenge.
• CCA 2 cm from the bifurcation
• Maxi and min PSV obtained in a single respiratory cycle
• ΔCDPV=(MaxCDPV−MinCDPV)/(MaxCDPV+MinCDPV)/2×100
• In addition
  • TTE for stroke volume
  • Fem A: Pulse pressure variation PPV (%) = 100×(Pp max − Pp min)/(Pp max + Pp min)/2
  • The passive leg raising (PLR) test was inferior
  • Tranquility medetomidine to obtain an autoregulated SV, stroke volume variation (SVV), and other variables.
• Patients with an increase of more than 15% in the SVI after the fluid challenge were classified as “responders”, and those with an increase of less than 15% in the SVI or those with no increase were classified as “non-responders.”

Feasibility of common carotid artery point of care ultrasound in cardiac output measurements compared to invasive methods
• Single urban tertiary care academic center
• SICU and ICU
• All patients had an existing invasive monitoring device, either a PA catheter or PAMAP
• Carotid Doppler waveforms and the time average velocity 3–5 cardiac cycles
• Volume flow = Cross-sectional diameter X Time average velocity.
• Physiology studies quote a range of 15–26% for the proportion of CO that is dedicated to cerebral perfusion
• Cerebral flow comprised 20% of resting CO
• 80% of cerebral perfusion was supplied via the carotid arteries, 40% via each carotid
• CO = Carotid Doppler flow volume X 10.

Arterial trauma during central venous catheter insertion
• Approximately 7 million of CVC are installed each year in the US
• Small needle puncture 5% of cases
• Arterial misplacement of large caliber cannula have an incidence of 0.1% to 0.8%
• Carotid Artery Puncture/Cannulation
  • Of the 56 claims for CA puncture/insertion
    • 4 resulted in airway obstruction due to hematoma
    • 2 resulted in surgery with arterial repair
    • 4 resulted in case cancellation
    • In two claims, the arterial position of the catheter was unrecognized for 22 h or more
    • Neither ultrasound guidance nor pressure waveform monitoring was used for vessel localization in any of these claims
1968, Hayreh established the presence of a constant communication between the subarachnoid space of the optic nerve sheath and the intracranial cavity. Normal ONSD measured by ultrasound ranges from 4.7 to 5.0 mm in previous studies. However, in the current study, we measured the ONSD 1.1 mm, which is lower than the mean ONSD of 5.5 mm in healthy volunteers. The decrease in ONSD may be due to the patient's underlying condition, such as intracranial hypertension or optic nerve sheath dilation. The patient's ONSD remained unchanged after the increase of ICP.

As part of a previously published study, a single operator measured the ONSD in 120 healthy volunteers over a 6-month period. Utilizing the assumption that the four measurements made on each subject during this study should be equal, the relationship of within-subject variance was described using the quadratic-plateau model as assessed by segmental polynomial (knot) regression.

<table>
<thead>
<tr>
<th>Table 1 Optic nerve ultrasonography (ONUS) compared with cranial CT for evidence of signs of raised intracranial pressure</th>
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<td></td>
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<tr>
<td>Positive</td>
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<tr>
<td>Negative</td>
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</tbody>
</table>

* Sensitivity 98.4%, negative predictive value 97.3%, specificity 92.6%, positive predictive value 94.3%.

April 2006 - January 2007 Cranial CT findings were used as a reference 100 participants. Median GCS=11 Mean binocular ONSD (5.8 0.57 mm) raised ICP. Mean ONSD (3.5 0.75 mm) without.

- 35 patients were academic ER
- 14 CT results consistent with EICP.
- ONSD > 5 mm on US
- The mean ONSD for the 14 patients with CT evidence of EICP was 6.27 mm (95% CI 1/4 5.6 to 6.89)
- The mean ONSD for the others was 4.42 mm (95% CI 1/4 4.15 to 4.72)
- The difference of 1.85 mm (95% CI 1/4 1.23 to 2.39 mm) yielded a p 1/4 0.001.
- The sensitivity and specificity for ONSD, when compared with CT results, were 100% and 95%, respectively
- PPV, NPV were 93% and 100%, respectively

<table>
<thead>
<tr>
<th>Table 2 Comparison of various optical nerve sheath diameter (ONSD) and intracranial pressure (ICP) in patients with EICP</th>
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</thead>
<tbody>
<tr>
<td>Author</td>
</tr>
<tr>
<td>-------------------------</td>
</tr>
<tr>
<td>Hansen et al.</td>
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<td>Tana et al.</td>
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<td>Tanga et al.</td>
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<td>Study</td>
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<td>1989</td>
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<tr>
<td>1991</td>
</tr>
<tr>
<td>1999</td>
</tr>
</tbody>
</table>

* Sensitivity 99.0%, Specificity 98.0%
The ONSD retained high accuracy for the detection of ICP > 20 mmHg. Optimal ONSD cutoff between the two groups was 5.22 mm (95% CI 4.46–5.99 mm; p < 0.0001). ROC curve analysis demonstrated high accuracy (AUC = 0.97) with optimal ONSD cutoff 5.22 mm. The area under the ROC curve was 0.93 (95% confidence interval [CI] = 0.84 to 0.95)

- Important and unanswered question is whether ONSUS remains accurate in the setting of acutely fluctuating ICP.
- Authors identified significant acute ICP fluctuation in individual invasive ICP measurements above and below 20 mm Hg were recorded among the six attempted measurements.
- All ICP spikes to >30 mm Hg that persisted for at least five minutes, the lowest ICP in this period and the highest ICP in this period were recorded. The accuracy of ONSD measurements performed within four hours of an ICP elevation to >30 mm Hg was compared to the accuracy of ONSD measurements without any preceding spike.
- Specificity and PPV of ONSD for ICP > 30 mm Hg are substantially decreased in patients demonstrating acute fluctuation of ICP between high and normal. This may be because of delayed reversal of nerve sheath distension.
Eleven patients (7 males, 4 females) with traumatic brain injury (TBI) and GCS<9

- All patients had EVD monitors
- A total of 29 ONUS obtained with invasive ICP measurements, pulsatility indices (PI) using middle cerebral artery (MCA) and ONSD.
  - 14 ONSD was <5.0 mm, while ICPs ranged 1-18 mmHg
  - 11 ONSD one or both ONS diameters ranged 5.0-5.7mm, while ICP ranged 10-19mmHg
  - 1 ONSD 5.7 mm, with ICP 13mmHg, however, later patient developed ICP 37mmHg within 24 hours.
  - 1 ONSD 5.7mm with ICP 16mmHg, however, later developed ICP of >30mmHg within 24 hours.
  - 2 ONSD > 3.8mm with corresponding ICP >20mmHg

- There was no correlation between PI on TCD, thus TCD was not useful in this dataset.