CD monitoring in ICU

Dr. FRANK RASULO
NEURO INTENSIVE CARE UNIT - Dep. of Anesthesiology & Intensive Care,
Spedali Civili University Hospital of Brescia, ITALY
Monitoring

- Neurophysiologic
- Cerebral Hemodynamic
- Neurochemical & Metabolic
Monitoring CBF (bedside)

**Snapshot**

**Continuous**
- Needs validation
- Invasive
Transcranial Doppler Monitoring in the Neuro Critical Care Unit

- Safe
- Cheap
- Repeatable
- Bedside
- Good learning curve
- Reliable

Dr. FRANK RASULO  Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY
TCD in the ICU

- VASOSPASM
- INTRACRANIAL HYPERTENSION
- CEREBRAL CIRCULATORY ARREST
- AUTOREGULATION
- STENOSIS

- SUBARACHNOID HEMORRHAGE
- HEAD TRAUMA
- STROKE
- BRAIN DEATH

Dr. FRANK RASULO  Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY
VASOSPASM & ANGIOGRAPHY

Gold Standard

Dr. FRANK RASULO  Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY
Neurologic Complications of Cerebral Angiography: Prospective Analysis of 2,899 Procedures and Review of the Literature

39 (1.3%) neurologic complications in 2,899 procedures;
20 (0.7%) transient
5 (0.2%) reversible
14 (0.5%) permanent (strokes)

NEUROLOGIC COMPLICATIONS OF CEREBRAL ANGIOGRAPHY
A retrospective study of complication rate and patient risk factors

<table>
<thead>
<tr>
<th>Studies</th>
<th>n</th>
<th>All</th>
<th>Persistent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grzyska et al. (ref. 4)</td>
<td>1095</td>
<td>0.54%</td>
<td>0.45%</td>
</tr>
<tr>
<td>Heiserman et al. 1994 (ref. 5)</td>
<td>1000</td>
<td>1%</td>
<td>0.8%</td>
</tr>
<tr>
<td>Waugh et al. 1992 (ref. 12)</td>
<td>2075</td>
<td>0.6%</td>
<td>0.3%</td>
</tr>
<tr>
<td>Present study</td>
<td>483</td>
<td>2.3%</td>
<td>0.4%</td>
</tr>
</tbody>
</table>
TCD and Vasospasm
TCD and Vasospasm

Comparison of TCD with angiography for detection of vasospasm

- An inverse relationship between vessel diameter and TCD velocities
  
  Aaslid et al.

- $F_{Vm} > 120 \text{ cm/s} = \text{mild vasospasm}$
- $F_{Vm} > 200 \text{ cm/s} = \text{severe vasospasm}$

Correlation between $F_{Vm}$ and angiographic lumen diameter of MCA

- $F_{Vm} < 120 \text{ cm/sec} - < 25\%$ narrowing
- $F_{Vm} \text{ 120-200 cm/sec} - 25-50\%$ narrowing
- $F_{Vm} < 200 \text{ cm/sec} - > 50\%$ narrowing

Aaslid et al.
Sloan et al.
Vora et al.
Lindegaard et al.
TCD and Vasospasm

Sensitivity & Specificity
TCD and Vasospasm

Assessment: Transcranial Doppler ultrasonography
Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology

“to review the use of TCD for diagnosis”
# TCD and Vasospasm

## Vasospasm after Spontaneous 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 Subarachnoid Hemorrhage</td>
<td></td>
<td></td>
<td>Conventional angiography</td>
</tr>
<tr>
<td>Intracranial ICA</td>
<td>25-30</td>
<td>83-91</td>
<td></td>
</tr>
<tr>
<td><strong>MCA</strong></td>
<td><strong>39-94</strong></td>
<td><strong>70-100</strong></td>
<td></td>
</tr>
<tr>
<td>ACA</td>
<td>13-71</td>
<td>65-100</td>
<td></td>
</tr>
<tr>
<td>VA</td>
<td>44-100</td>
<td>82-88</td>
<td></td>
</tr>
<tr>
<td>BA</td>
<td>77-100</td>
<td>42-79</td>
<td></td>
</tr>
<tr>
<td>PCA</td>
<td>48-60</td>
<td>78-87</td>
<td></td>
</tr>
</tbody>
</table>
## TCCD and Vasospasm

### Vasospasm after Spontaneous SAH

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<thead>
<tr>
<th>INDICATION</th>
<th>SENSITIVITY (%)</th>
<th>SPECIFICITY (%)</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Vasospasm after Spontaneous Subarachnoid Hemorrhage</td>
<td>69</td>
<td>83</td>
<td>Conventional angiography</td>
</tr>
<tr>
<td>Intracranial ICA</td>
<td>100</td>
<td>97</td>
<td></td>
</tr>
<tr>
<td>MCA</td>
<td>100</td>
<td>93</td>
<td></td>
</tr>
<tr>
<td>ACA</td>
<td>71</td>
<td>85</td>
<td></td>
</tr>
</tbody>
</table>

*Dr. FRANK RASULO  Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY*

*Courtesy of Andrea Rigamonti*
Predicting Outcome after Traumatic Brain Injury:
Development and International Validation of Prognostic
Scores Based on Admission Characteristics
Ewout W. Steyerberg et al., PLoS Medicine August 2008 | Volume 5 | Issue 8 | e165

Head injury patients with SAH have a worse prognosis than patients without SAH or Vasospasm.
TCD and post-traumatic SAH

CT evidence of SAH following closed head injury occurs in up to 63% of patients

\[ \text{tSAH} \rightarrow \text{VSP} \rightarrow \text{focal DID} \]

63%  15%

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</thead>
<tbody>
<tr>
<td>Vasospasm after Traumatic Subarachnoid Hemorrhage</td>
<td></td>
<td></td>
<td>Conventional angiography</td>
</tr>
</tbody>
</table>

Data on sensitivity, specificity and predictive value of TCD for VSP after tSAH are needed.

Lee JH
Martin NA
TCD & Delayed Ischemic Deficits (DID)

*Ability of TCD to predict onset of DID’s following SAH*

DID’s occurred in **20–30%** of SAH pts. within 3–14 days after bleed. Vasospasm-related DIDs were the major cause of bad outcome. 

Kassell NF et al.

8 of 21 patients with SAH developed DID. Good correlation between TCD and DID.

Sekhar et al.
“TCD-defined vasospasm preceded the neurological deficit in 64%, therefore earlier intervention might reduce the incidence of vasospasm-related stroke.”

What about the other 36%?
Regions of cerebral ischemia are not always associated with territories of maximal vasospasm.
The accuracy of TCD to detect vasospasm in patients with aneurysmal subarachnoid hemorrhage
L. Mascia et al. Intensive Care Medicine 2003

MFv threshold > 160 cm/s

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>LR+</th>
<th>LR−</th>
</tr>
</thead>
<tbody>
<tr>
<td>TCD</td>
<td>1.00</td>
<td>0.75</td>
<td>0.72</td>
<td>1.00</td>
<td>4</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>LR+</th>
<th>LR−</th>
</tr>
</thead>
<tbody>
<tr>
<td>TCD</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

TCD  Delayed Ischemic Deficits (DID)
TCD and Vasospasm

**Vasospasm Probability Index:** a combination of transcranial doppler velocities, CBF, and clinical risk factors to predict cerebral vasospasm after aneurysmal SAH.


The index (combination of predictive factors associated with the development of vasospasm) may become a valuable tool for the clinician to evaluate the individual probability of cerebral vasospasm after aneurysmal SAH.

MCA –

✓ Fv <121 - >182 cm/s

(low risk for VSP – high risk for VSP)

MCA –

✓ Fv <120 - >200 cm/s

(NPV 94% - PPV 87%)

Vora

Gosset
TCD and Vasospasm

- Rapid rise in Fvm in a few days
- High Lindegaard ratio
TCD and Multimodal Monitoring

Journal of NeuroEngineering and Rehabilitation

Methodology

Relationship between oxygen supply and cerebral blood flow assessed by transcranial Doppler and near-infrared spectroscopy in healthy subjects during breath-holding

Filippo Molinari*¹, William Liboni², Gianfranco Grippi² and Emanuela Negri²

Brain Tissue $pO_2$ in Relation to Cerebral Perfusion Pressure, TCD Findings and TCD-$CO_2$-Reactivity After Severe Head Injury


J. Vings, J. Metxensberger, J. Amschler, B. Hamelbeck, and K. Roosen
FV decreases during the first 48 h after head trauma and subsequently increases between 48–120 h. The ability of TCD to distinguish between the two processes facilitates clinical management.
Persistently low MCA FVm is associated with poor neur. outcome

Serial transcranial Doppler measurements in traumatic brain injury with special focus on the early posttraumatic period.

Santbrink H van et al., Acta Neurochir 2002 (Wien) 144:1141–1149

Intracranial blood flow velocity after head injury: relationship to severity of injury, time, neurological status and outcome.

Chan KH et al., J Neurol Neurosurg Psychiatry 1992;55:787–791

Evaluating the Outcome of Severe Head Injury With Transcranial Doppler Ultrasonography

José A. Moreno et al., Neurosurgical Focus 1999

MCA FVm < 30 - 35 cm/sec

The most reliable indicators of low CPP are low FVd with high PI.
The gold standard for the measurement of ICP is invasive measurement. However, there are various situations where a non-invasive measurement may be useful:

- Mild and moderate head injury
- Ischemic & hemorrhagic stroke, vasospasm
- Meningo-encephalytis
- Faulty ICP catheters
- Outside the ICU (ER, OR, etc.)
The earliest sign of increased ICP is increased pulsatility.

\[ \text{PI} = \frac{(FV_{s} - FV_{d})}{FV_{m}} \]
There is a strong correlation between PI and ICP (ICP values $> 20 \text{ mmHg}$), and between PI and CPP (CPP values $< 70 \text{ mmHg}$).

Voulgaris et al.
**Transcranial Doppler Sonography**

**Pulsatility Index (PI) Reflects Intracranial Pressure (ICP)**


First **prospective** study to investigate the relationship between ICP and TCD derived PI.

---

1. Graph demonstrating a significant correlation between the CPP and PI with a correlation coefficient of r = 0.48 (p < 0.0001) and a correlation formula of CPP = 80.946 - 0.325 × PI. The correlation between CPP and PI is mainly when PI > 3. The dotted lines are the 95% confidence interval for the regression line, which can be significantly affected by outliers when PI is large.

2. Graph demonstrating a significant correlation between the ICP and PI with a correlation coefficient of r = 0.938 (p < 0.0001) and a correlation formula of ICP = 10.927 × PI - 1.284. The dotted lines are the 95% confidence interval for the regression line, which can be significantly affected by outliers when PI is large.
Admission TCD and TBI
**Admission TCD and TBI**

**Admission** FVd < 25 cm/s and PI > 1.3 is associated with a poor outcome. 

*Trabold et al.*

**Admission** velocities, FVm< 30 cm/s, were related to GCS, and correctly predicted early outcome. 

*Chan KH et al.*
24 TBI pts.  
- Anormal TCD values (group 1)  
- Normal TCD values (group 2)

3-month GOS was significantly poorer in group 1 than in group 2.
TCD, CPP and ICP

Aaslid et al

\[ eCPP = AP1 \times \frac{FVm}{FV1} + 15 \]

\[ \leq 27 \text{ mmHg} \Rightarrow 95\% \\
\leq 10 \text{ mmHg} \Rightarrow 52\% \]

Czosnyka et al.

\[ nCPP = MAP \times \frac{FVd}{FVm} + 14 \]

\[ \leq 21 \text{ mmHg} \Rightarrow 95\% \text{ delle stime} \\
\leq 10 \text{ mmHg} \Rightarrow 81\% \text{ delle stime} \]
TCD and ICP

Influence of CVA on the non-invasive estimation of CPP through TCD.
DePeri E, Rasulo F et al. Intensive Care Med (Sup.1) Sept 2005:103,
No. 389. 18th ESICM, Amsterdam

✓ 21 patients
✓ 530 samples

Dr. FRANK RASULO Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY
TCD and ICP

Monitoring of Increased Intracranial Pressure Resulting From Cerebral Edema With Transcranial Doppler Sonography in Patients With Middle Cerebral Artery Infarction  

- ICP was higher on the third day than at admission in these patients.
- Increased ICP on TCD correlated with midline shifts on CT scans.
- Early outcomes of pts who had increased ICP on TCD were poorer.
Cerebral hemodynamic changes gauged by transcranial Doppler ultrasonography in patients with post-traumatic brain swelling treated by surgical decompression
Edson Bor-Seng-Shu et al. J Neurosurg 104:93-100, 2006

\[
\text{CVR} = \frac{\text{FV}}{\text{PI}}
\]
TCD in Decompressive Craniectomy

- Male 44 yr, slurred speech, sudden hemiparesis
- First TCD in ER = Angiography
- Angiography
- left MCA Ischemic Stroke
- Thrombolysis

2° TCD
TCD in Decompressive Craniectomy

Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY

Dr. FRANK RASULO   Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY
TCD & Critical Closing Pressure

1° STARLING RESISTOR
Arteriolar resistance vessels

2° STARLING RESISTOR
bridging Veins

CPP = MAP - CCP

CPP = MAP - ICP

$P = \frac{T}{r}$

$P < \frac{T}{r}$

$P = \text{pressione transmurale}$

$T = \text{tensione di parete}$

$r = \text{raggio del vaso}$

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**Weyland A. et all**

Cerebrovascular tone rather than intracranial pressure determines the effective downstream pressure of the cerebral circulation in the absence of intracranial hypertension

J Neurosurg Anesth 2000;12:210-16
TCD and Cerebrovascular Autoregulation

CBF

MAX VASODILATION

50 mmHg

MAX VASOCOSTRICTION

ABPm

Dr. FRANK RASULO  Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY
# Cerebral Autoregulation Dynamics in Humans

Rune Aaslid, PhD, Karl-Fredrik Lindegaard, MD, Wilhelm Sorteberg, MD, and Helge Nornes, MD

*Stroke*  **Vol 20, No 1, January 1989**

## Table 1: Parameters Measured in Determining Cerebral Autoregulation Dynamics in Humans

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Hypocapnia (n=10) (22.2 mm Hg)</th>
<th>Normocapnia (n=10) (37.1 mm Hg)</th>
<th>Hypercapnia (n=6) (46.9 mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{CO_2}$ (mm Hg)</td>
<td>22.2±0.6</td>
<td>37.1±0.8</td>
<td>46.9±0.5</td>
</tr>
<tr>
<td>Control $V_{MCA}$ (cm/sec)</td>
<td>46.1±3.1</td>
<td>67.4±5.9</td>
<td>89.3±3.4</td>
</tr>
<tr>
<td>Control ABP (mm Hg)</td>
<td>84.5±4.1</td>
<td>81.6±4.2</td>
<td>88.2±7.2</td>
</tr>
<tr>
<td>$\Delta$ ABP (%)</td>
<td>21.9±1.0</td>
<td>24.1±1.1</td>
<td>21.5±2.2</td>
</tr>
<tr>
<td>RoR (sec⁻¹)</td>
<td>0.38±0.04</td>
<td>0.20±0.30</td>
<td>0.11±0.02</td>
</tr>
<tr>
<td>$\Delta$ Power (%)</td>
<td>−0.4±1.0</td>
<td>−2.55±1.2</td>
<td></td>
</tr>
</tbody>
</table>
TCD and Cerebrovascular Autoregulation

A Bedside Test for Cerebral Autoregulation Using Transcranial Doppler Ultrasound


C. A. Giller
However, clinical tests do not provide continuous assessment of CVA, which can be performed through…
Continuous Assessment of Autoregulation

CORRELATION COEFFICIENTS

Monitoring of Cerebral Autoregulation in Head-Injured Patients.

M Czosnyka, et al., Stroke, 2000

✓ (PRx)  **ABP/ICP**

✓ (Sx)  **FVs/CPP**

✓ (Mx)  **FVm/CPP**
TCD and Cerebrovascular Autoregulation

(Mx)

FVm/CPP
TCD and Cerebrovascular Autoregulation

Autoregulation and Cerebral Perfusion Pressure

![Graph showing autoregulation and cerebral perfusion pressure (CPP)]
TCD and Cerebrovascular Autoregulation

Autoregulation and Cerebral Perfusion Pressure

![Graph showing Autoregulation and Cerebral Perfusion Pressure](image)

- **Mx**
- **CPP [mm Hg]**

- **Altered CVA**
- **Normal CVA**
TCD and Cerebrovascular Autoregulation

Continuous Assessment of Cerebral Autoregulation in Subarachnoid Hemorrhage
Soehle M et al., Anesth Analg 2004;98:1133-9

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>MABP (mm Hg)</th>
<th>Mean MCA FV (cm/s)</th>
<th>Mx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>15</td>
<td>93 ± 14</td>
<td>76 ± 20</td>
<td>0.21 ± 0.24</td>
</tr>
<tr>
<td>Vasospasm</td>
<td>15</td>
<td>100 ± 13</td>
<td>148 ± 19</td>
<td>0.46 ± 0.32</td>
</tr>
<tr>
<td>p value (Student's t-test)</td>
<td>0.007</td>
<td>&lt;0.001</td>
<td>0.021</td>
<td></td>
</tr>
</tbody>
</table>

Table 3. Summary of Mx and Sx Values Obtained in Different Pathologies

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Reference</th>
<th>Pathology</th>
<th>n</th>
<th>Mx</th>
<th>Sx</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Baseline</td>
<td>Baseline</td>
<td>14</td>
<td>0.21 ± 0.16</td>
<td>-0.07 ± 0.15</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Temporal comparison</td>
<td>Intracranial normotension</td>
<td>8</td>
<td>0.07 ± 0.31</td>
<td>Preserved autoregulation</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Intracranial hypertension</td>
<td>8</td>
<td>0.80 ± 0.29</td>
<td>Impaired autoregulation</td>
<td>8</td>
</tr>
<tr>
<td>Czosnyka</td>
<td>1999</td>
<td>37</td>
<td>Head injury</td>
<td>19</td>
<td>0.15 ± 0.28</td>
<td>Baseline</td>
<td>21</td>
</tr>
<tr>
<td>Lang</td>
<td>2003</td>
<td>33</td>
<td>Head injury</td>
<td>15</td>
<td>0.21 ± 0.24</td>
<td>0.05 ± 0.21</td>
<td>15</td>
</tr>
<tr>
<td>Our study</td>
<td>2004</td>
<td>SAH</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Spatial comparison

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Reference</th>
<th>Pathology</th>
<th>n</th>
<th>Mx</th>
<th>Sx</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Contralateral side of stenosis</td>
<td>Side of stenosis</td>
<td>56</td>
<td>0.20 ± 0.17</td>
<td>0.51 ± 0.18</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Contralateral side of vasospasm</td>
<td>Side of vasospasm</td>
<td>56</td>
<td>0.20 ± 0.17</td>
<td>0.51 ± 0.18</td>
<td></td>
</tr>
<tr>
<td>Reinhard</td>
<td>2003</td>
<td>35</td>
<td>CAOD</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
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<td>SAH</td>
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<td></td>
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<td></td>
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</table>

“Mx showed impaired autoregulation in all the patients with Vasospasm and correlated with the side affected”
Mx can be estimated noninvasively with the use of a finger plethysmograph arterial blood pressure measurement instead of an invasive cerebral perfusion pressure measurement.

Proposed as a practical tool to assess CVA in patients who do not require invasive monitoring.
Cerebral Circulatory Arrest in Brain Death

Dead Brain Coral
TCD and Cerebral Circulatory Arrest

Assessment: Transcranial Doppler ultrasonography

Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology


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<th>SPECIFICITY (%)</th>
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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: Type A, Class II evidence
Transcranial Doppler Ultrasonography to confirm Brain Death: a metaanalysis

Table 2  Meta-analysis of high quality studies and sensitivity analysis of all included studies. CI confidence interval

<table>
<thead>
<tr>
<th>Primary analysis: only high-quality studies</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity (%)</td>
<td>95</td>
</tr>
<tr>
<td>Specificity (%)</td>
<td>99</td>
</tr>
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</table>

Sensitivity analysis: all studies. Velthoven et al. [24]: cerebral angiography and clinical criteria as reference test

<table>
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Sensitivity analysis: all studies. Velthoven et al. [24]: EEG and clinical criteria as reference test

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<tr>
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<tr>
<td>Specificity (%)</td>
</tr>
</tbody>
</table>
TCD in the ICU

- Vasospasmo cerebrale
- Ipertensione intracranica
- Arresto di circolo cerebrale
- Autoregolazione cerebrovascolare
- Stenosis

- Meningitis
- Encefaliti subaracnoidea
- Eclampsia
- Trauma cranico
- Liver failure
- Stroke
- Sickle cell disease
- Morbidade cerebrale
- Diabetes
- Sepsis

Institute of Anesthesiology & Intensive Care, Spedali Civili University Hospital of Brescia, Italy
Three phases of cerebral arteriopathy in meningitis: vasospasm and vasodilation followed by organic stenosis


**Vasodilation** – (vasoparalysis) due to decreased contractile energy in association with myonecrosis.

**Vasospasm** – (stimulus phenomenon) produced by the surrounding purulent material “narrowing of the the vessels caused by the inflammatory process” (arteritis)

**Stenosis** – (repair process) due to the organization of subendothelial edema with resultant intimal thickening.
TCD allowed to distinguish three phases of tuberculous meningitis related vasculopathy.

**Phase I vasculopathy** - increased Vm and normal to moderately decreased PI. In this phase reversible ischemic deficits are seen clinically and radiologically.

**Phase II** - decreased Vm. At this stage patients reveal radiological and clinical signs related to proximally evolving vasculopathy in the basal main arteries.

**Phase III** - almost absent CBF in one or more basal arteries and, accordingly, by associated brain tissue infarction and permanent severe neurological deficit or fatal outcome.
Relationship between short-term outcome and occurrence of cerebral artery stenosis in survivors of bacterial meningitis


Patients with stenosis were associated with high Fvm and a poor short-term outcome

Multimodal cerebral monitoring and decompressive surgery for the treatment of severe bacterial meningitis with increased intracranial pressure


ICP and TCD monitoring helped to decide when to perform early and late surgery (hemicraniectomy and shunting)
Do maternal cerebral vascular changes assessed by transcranial Doppler antedate pre-eclampsia?

Williams K et al. Ultrasound Obstet Gynecol. 2004 Mar;23:254-6

Maternal transcranial Doppler in pre-eclampsia and eclampsia.


Pre-eclampsia: CPP ↑, CVR ↑, CBF =

Eclampsia: CPP ↑, CVR ↓, CBF ↑
Transcranial Doppler Sonography in Fulminant Hepatic Failure


“The pattern mostly found is that of cerebral hypoperfusion.”

Therapy of Intracranial Hypertension in Patients With Fulminant Hepatic Failure

Murugan Raghavan, Paul E. Marik

*Neurocritical Care*, April 2006, Volume 4, Issue 2, pps. 179-189

“Many patients die prior to the availability of donor organs,
often because of cerebral herniation”

Cerebral Hemodynamic and Metabolic Changes in Patients With Fulminant Hepatic Failure During Liver Transplantation

G. Ardizzone, A. Arrigo, F. Panaro, S. Ornis, R. Colombi, S. Distefano, T.M. Jarzembsowski, and E. Cerruti


“A hallmark of FHF seems to be failure of autoregulation, which is linked to uncoupling between CBF and CMRO2”

Dr. FRANK RASULO  Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY
“Children with elevated velocities (≥ 200 cm/sec) have high rate of stroke. When asymptomatic children with abnormally high flow velocities demonstrated by using screening TCD are preemptively treated with maintenance transfusion, 90% of strokes are prevented.”
TCD, SEPSIS & Encephalopathy

Dysfunction of vasomotor reactivity in severe sepsis and septic shock

Christoph Terborg

Metabolic changes

SEPSIS

ENCEPHALOPATHY (70%)

Inflammatory hypothesis

Muscular proteolysis
Hepatic dysfunction
Reduced AA uptake

Cytochines
Arachidonic ac. metabolites

Dr. FRANK RASULO
Neuro-Intensive Care, Spedali Civili University Hospital of Brescia, ITALY
Cerebral Perfusion in Sepsis-Associated Delirium

David Pfister et al. *Critical Care* 2008, 12:R63

**Key messages**

- In this small group of patients, cerebral perfusion assessed with transcranial Doppler and near-infrared spectroscopy did not differ between patients with and without sepsis-associated delirium.

- We found a significant association between disturbed cerebrovascular autoregulation and sepsis-associated delirium.

- A significant correlation between higher values of C-reactive protein and increasingly disturbed cerebrovascular autoregulation suggests a harmful effect of inflammation on cerebrovascular endothelial function.

- The significant associations between sepsis-associated delirium and elevated S-100β and cortisol suggest that further investigations defining the role of these markers as aids in the diagnosis of sepsis-associated delirium are warranted.
There are no RCTs assessing the effects of jugular venous bulb saturation, **transcranial Doppler**, Intracranial pressure, or cerebral microdialysis.
Monitor Interpretation

Therapeutic Intervention

PROVING EFFECT ON OUTCOME
Monitor Interpretation

• The first step to demonstrate efficacy of monitoring is to ensure adequate training in interpretation.
Therapeutic Intervention

• A monitor cannot affect patient outcome unless it identifies a preclinical abnormality and prompts timely and appropriate therapeutic intervention.

• This abnormality must be an integral determinant of patient outcome.

• For example, intracranial hypertension cannot be reliably detected by clinical exam and has been associated with unfavorable neurological outcome.
PROVING EFFECT ON OUTCOME

Monitor Interpretation

Therapeutic Intervention

Tripple H?

- HyperVOLEMIA
- HyperTENSION
- HemoDILUTION
there is insufficient “EBM” data to recommend the use of 3-H strategy as a prophylactic treatment after SAH.
Effects of hypervolemia and hypertension on regional cerebral blood flow, intracranial pressure, and brain tissue oxygenation after subarachnoid hemorrhage. Elke Muench et al., Crit Care Med 2007 Vol. 35, No. 8

Vasopressor-induced elevation of MAP causes a significant increase of CPP and cerebral oxygenation in SAH patients.

While volume expansion results in an increase in perfusion, hypervolemia reverses the hypertension-induced benefit on PtiO2.
Central Hypervolemia with Hemodilution Impairs Dynamic Cerebral Autoregulation
Yojiro Ogawa et al. Neurosurgical Anesthesiology 2007;105:1389-96

Frequent changes in the perioperative central blood volume could affect cerebral autoregulation through alterations in sympathetic activity, cardiac output, blood viscosity, and cerebral vasomotor tone.

Although steady-state CBF velocity changes under both central hypervolemia and hypovolemia, only hypervolemic hemodilution impairs dynamic cerebral autoregulation.