Hemodynamic Monitoring in the Neurosciences ICU

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Outline

- Rationale for hemodynamic monitoring in neurocritical care.
- Invasive vs. non-invasive monitoring techniques.
- Physiologic and technical bases of invasive hemodynamic monitoring.
- Evidence for hemodynamic goal-directed therapy in neurocritical care.
Why monitor hemodynamics?

- limit systolic blood pressure (SBP) to reduce/prevent brain injury
  - subarachnoid hemorrhage with unsecured aneurysm
  - intraparenchymal hemorrhage
  - posterior reversible encephalopathy syndrome (PRES)
Subarachnoid hemorrhage:

- American Heart Association Guidelines 2009
  - Class 1: monitor and control blood pressure to reduce risk of rebleeding
  - systolic blood pressure (SBP) > 160 mmHg is a risk factor for rebleeding (Ohkuma et al. Stroke 2001)
Intraparenchymal Hemorrhage:

- American Heart Association Guidelines 2007
  - maintain SBP < 180 mmHg; ATACH (SBP < 210, 170, 140 mmHg) and INTERACT2 (SBP < 180, 140 mmHg) trials ongoing
  - SBP < 140 mmHg reduces hematoma size (INTERACT Anderson et al. Stroke 2010)
Why monitor hemodynamics?

- maintain cerebral perfusion pressure (CPP)
  - traumatic brain injury (TBI)
  - elevated intracranial pressure (ICP)
- augment hemodynamic parameters
  - triple-H therapy
Traumatic brain injury

- Brain Trauma Foundation recommendations 2007
  - Level II: Blood pressure should be monitored and hypotension (SBP < 90 mmHg) avoided.
  - Level II: ICP should be monitored in all salvageable patients with severe TBI and abnormal CT.
  - Level II: Aggressive attempts to maintain CPP > 70 mmHg should be avoided because of risk of ARDS.
  - Level III: CPP < 50 mmHg should be avoided.
Why monitor hemodynamics?

- guide treatment of hemodynamic instability
- neurogenic myocardial stunning ("Tako-Tsubo cardiomyopathy")

Why monitor hemodynamics?

- guide treatment of hemodynamic instability
  - neurogenic myocardial stunning ("Tako-Tsubo cardiomyopathy")
  - spinal shock
  - polytrauma (cardiac contusion, hemorrhagic shock)
- sepsis
- pulmonary hypertension, pulmonary embolism
- pre-existing disease
Parameters to monitor

- pressure
  - systemic arterial pressure (afterload)
  - venous pressure (preload)
  - pulmonary artery pressure
- cardiac output
- fluid status and fluid responsiveness
Invasive pressure monitoring

McGhee et al. Crit Care Nurse 2002
Pitfalls

• inappropriate reference level
• damping
• underdamping
  • long tubing
  • hyperdynamic state
  • arterosclerosis
• overdamping
  • air in tubing
  • kink or clot in catheter
  • hypotension
Cardiac output

- pulmonary artery thermodilution (pulmonary artery catheter, PAC)
- aortic transpulmonary thermodilution (PiCCO®)
- lithium indicator dilution (LidcoPlus®)
- pulse contour variation (Flo Trac®)
- echocardiography
Stewart-Hamilton equation:

\[ Q = \frac{(V_1 (T_b - T_1) K_1 K_2)}{(T_b (t) \, dt)} \]
Transpulmonary thermodilution

Pulse Induced Continuous Cardiac Output (PiCCO) Monitoring
LidcoPlus®

Stroke Volume Variation > 9.5% predicts increased stroke volume after plasma expansion (Berkenstadt et al. Anesth Analg 2001)

McGee et al. Crit Care 2007
Static vs. dynamic measures of fluid responsiveness

- volume status is critical and is difficult to assess
- change in inferior vena cava diameter (dIVC) during positive pressure ventilation predicts fluid responsiveness (CI increase > 15%) in sepsis better than central venous pressure (CVP) (Feissel et al., Barbier et al. Intensive Care Med 2004)
Static vs. dynamic measures of fluid responsiveness

**Volume loading-induced changes in cardiac output (%)**

\[ r = 0.82, \quad r^2 = 0.68, \quad p < 0.001 \]

**Feissel et al.**
*Intensive Care Med 2004*

**Barbier et al.**
*Intensive Care Med 2004*

### Table 3: Comparison of baseline values in responders and non-responders

<table>
<thead>
<tr>
<th></th>
<th>Responders</th>
<th>Non-responders</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (bpm)</td>
<td>106±25</td>
<td>92±13</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic arterial pressure (mmHg)</td>
<td>112±42</td>
<td>123±26</td>
<td>NS</td>
</tr>
<tr>
<td>CI (l/min per m²)</td>
<td>2.4±1.1</td>
<td>3.0±1.2</td>
<td>NS</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>10±4</td>
<td>9±3</td>
<td>NS</td>
</tr>
<tr>
<td>Dose of vasopressors (µg/kg per min)</td>
<td>0.14±0.19</td>
<td>0.26±0.39</td>
<td>NS</td>
</tr>
<tr>
<td>dIVC (%)</td>
<td>40±24</td>
<td>8±8</td>
<td>0.0019</td>
</tr>
</tbody>
</table>

* Mann-Whitney test
Dynamic parameters in neurocritical care

- dIVC predicts fluid responsiveness in SAH better than stroke volume variability (Moretti and Pizzi Neurocritical Care 2010)
PAC vs. PiCCO

- PiCCO-derived cardiac output is similar to PAC-derived in SAH patients with vasospasm (Mutoh, Stroke 2009). GEDI predicts fluid responsiveness better than CVP or PCOP.
PAC vs. PiCCO

Mutoh et al. Stroke 2009
PAC does not always improve outcome

- pulmonary artery catheters (PACs) increase complications, but do not affect outcome in acute respiratory distress syndrome (ARDS) (ARDS Net Investigators N Engl J Med 2006)

- PACs increase ICU days and ventilator days without changing outcome in surgical patients (Stewart et al. J Am Coll Surg 2009)
CPP-directed therapy in TBI may not be beneficial

- outcome up to 47 months was not different between a center using ICP monitoring and CPP-directed therapy vs. a center that did not monitor ICP and aimed for MAP > 90 mmHg (Cremer et al. Crit Care Med 2005)
CPP-directed therapy in TBI may not be beneficial

Table 3. Extended Glasgow Outcome Scale at follow-up

<table>
<thead>
<tr>
<th>Category</th>
<th>Center A (n = 122)</th>
<th>Center B (n = 211)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dead</td>
<td>55 (45)</td>
<td>83 (39)</td>
</tr>
<tr>
<td>Vegetative state</td>
<td>1 (1)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Lower severe disability</td>
<td>2 (2)</td>
<td>19 (9)</td>
</tr>
<tr>
<td>Upper severe disability</td>
<td>3 (3)</td>
<td>10 (5)</td>
</tr>
<tr>
<td>Lower moderate disability</td>
<td>16 (13)</td>
<td>42 (34)</td>
</tr>
<tr>
<td>Upper moderate disability</td>
<td>23 (19)</td>
<td>35 (17)</td>
</tr>
<tr>
<td>Lower good recovery</td>
<td>10 (8)</td>
<td>22 (18)</td>
</tr>
<tr>
<td>Upper good recovery</td>
<td>12 (10)</td>
<td>13 (6)</td>
</tr>
</tbody>
</table>

Table 4. Summary odds ratios for a more favorable outcome on the extended Glasgow Outcome Scale

<table>
<thead>
<tr>
<th>Analysis</th>
<th>No.</th>
<th>Crude OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intention to treat</td>
<td>333</td>
<td>0.95 (0.62–1.44)</td>
<td>1.06 (0.63–1.77)</td>
</tr>
<tr>
<td>On treatment</td>
<td>264</td>
<td>0.92 (0.58–1.46)</td>
<td>0.83 (0.48–1.43)</td>
</tr>
</tbody>
</table>

OR, odds ratio; CI, confidence interval. A summary OR >1 favors better outcome for intracranial pressure/cerebral perfusion pressure-targeted intensive care (center B); a summary OR <1 favors better outcome for supportive intensive care (center A).

*Adjusted for age, best motor score ≥4, presence of 2 nonreactive pupils, computed tomography scan category, injury cause category, and evacuation of extradural hematoma.

Cremer et al. Crit Care Med 2005
Triple-H therapy lacks evidence

- no controlled trials assess effects of triple-H on cerebral blood flow in SAH patients with vasospasm (Dankbaar et al. Crit Care 2010)
Hypertension may increase blood flow

- induced hypertension, but not hypervolemia, improved CBF and tissue oxygenation in SAH patients (Muench et al. Crit Care Med 2007)
Inotropic therapy may be helpful

- increasing MAP by 29% improves CBF in patients with vasospasm as effectively as increasing CI by 46%. Increasing CVP by 35% has no effect on CBF (Kim et al, Neurosurgery 2003).
Take home:

- arterial blood pressure is helpful when done correctly
- volume status and fluid responsiveness are difficult to assess, dynamic methods may have advantages
- invasive monitoring should be prompted by individual patient characteristics
Thank you!