CPPopt without ‘Cogitates’

can we manage patients?

teamwork
CPPopt without ‘Cogitates’

‘cogitate’
think deeply about something
meditate or reflect
CPP one size fits all?

CPP = MAP - ICP

'cogitate' = think deeply about something; meditate or reflect.

the driving force of cerebral blood flow across the microvascular capillary bed.
CPP one size fits all?

CPP above: 70? ....65?...

CPP may be low; ICP < 15 mmHg

Too low CPP: ischaemia

CPP one size fits all?

Too high CPP: hyperaemia

‘cogitate’ = think deeply about something; meditate or reflect.
the driving force of cerebral blood flow across the microvascular capillary bed.

**CPP = MAP-ICP**


“the minimum level of CPP in this instance is greater than 70mmHg and frequently higher, defined by individual circumstances”

**PRx = qMAP,ICP**


“the most important advantage of PRx is the ability to guide the management of cerebral perfusion pressure”

‘cogitate’= think deeply about something; meditate or reflect.
individualized CPP according to the autoregulation status

CPP = MAP - ICP

\[ PRx = \rho_{\text{MAP,ICP}} \]

CPPopt = \( f(\text{PRx}) \)

Luzius Steiner

‘The Cambridge Hypothesis’

CPP should be kept at the CPP where an individual patient autoregulates most efficiently

from http://cppopt.org/cppopt-calculation-visualisation/

‘cogitate’= think deeply about something; meditate or reflect.
Clinical Decision Support System approach:

- CPPopt value and curve, updated every minute, in a 4 hr calculation window
- at least 75% of time good recordings of CPP and ICP values had to be available in the 4hr calculation window
- average PRx values had to be < 0.25 the past 4hrs
- select the CPP value with most negative PRx value covered by the curve.
- U-shaped, ascending and descending curves were accepted in case the overall PRx<0.25.
**TBI: CPPopt and CPP management**

**CPP management with PRx and CPPopt:**

a) When possible, we guide CPP management using the bedside CPPopt values.

b) Management of CPPopt values with: adequate sedoanalgesia, oxygenation, ventilation, control of temperature, vasopressor therapy, fluid balance and treat intracranial hypertension.

c) When CPPopt is not available, we keep CPP between 60-70 mmHg in accordance to BFT Guidelines.
Traumatic Brain Injury and Intracranial Hypertension
NCCU protocol

all patients with or at risk of intracranial hypertension:
- EKG, SpO2, ETCO2
- Invasive ABP, CVP line
- ICP monitor with ICP wave and ICM + connection
Selected severe cases:
- NIRS
- PbtO2 and brain temperature
- TDF-CBF
- EEG and BIS

Monitoring

- 30° head up, no venous obstruction
- opt CPP or CPP=60-70 mmHg
- SpO2>97%; PaO2>90mmHg, PaCO2 35-40mmHg
- Temp<37°C; blood sugar 80-120 mg/dl
- Propofol 2% and/or midazolam: target 0<RASS<-5
- Fentanyl: target BPS < 3
- Ranitidine 50mg tid
- Norepinephrine according to CPP target
- Fluids with normal saline; 140<Na+ target<155mEq/l
- Tube feeding (orogastric, oroejunal): target 25-30Kcal/kg/d
- Control of seizures

Management I

Check ICP wave and ICP amplitude
Check / Change ICP probe

- adjust NE to CPP target if ICP<20 and CPP≠CPPopt
- 0.5-1g/kg 20% manitol if ICP>20 and S_{Osm}<320 or 2ml/kg 20% NaCl if ICP>20 and S_{Osm}>320.
- Mild hyperventilation: PaCO2=30-35 mmHg
- Mild hypothermia T 35°C
- Paralysis with rocuronium

Management II

Repeat osmotherapy bolus every 4h, if high ICP
- Moderate hypothermia 34-35°C
- Trial of tiopenthal

Management III

ICP<20
optCPP or CPP=60-70

recent CT ?
low risk of surgical lesion ?

Repeat CT

- Surgical lesion?
- CSF drainage ?
- Role for surgical decompression?

Call Neurosurgery

'CPPopt' in clinical practice at the NCCU: how do we do it with ICM+ in Porto
'CPPopt' in clinical practice at the NCCU: how do we do it with ICM+ in Porto
severe TBI and spontaneous SAH with advanced vs standard neuromonitoring

- **3 and 6M outcome** of the 2 groups of patients
- except for age there was no difference between the two groups at baseline, namely for GCS and SAPS II.
- **Advanced neuromonitoring group had a significantly better outcome (GOS) at 3 and 6 months and lower mortality.** Adjusting outcome for age, patients with advanced neuromonitoring had a lower risk of bad outcome.

<table>
<thead>
<tr>
<th>Outcome Mortality and GOS</th>
<th>Advanced Monitoring n (%)</th>
<th>Standard Monitoring n (%)</th>
<th>$p$ value</th>
<th>Odds ratio (adjusted for age)</th>
</tr>
</thead>
<tbody>
<tr>
<td>at 3 months</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good*</td>
<td>50 (75.8)</td>
<td>110 (50.0)</td>
<td>0.01</td>
<td>0.485(0.248-0.950)</td>
</tr>
<tr>
<td>Bad**</td>
<td>16 (24.2)</td>
<td>110 (50.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality</td>
<td>7 (10.6)</td>
<td>54 (24.5)</td>
<td>0.015</td>
<td>0.579(0.238-1.404)</td>
</tr>
<tr>
<td>at 6 months</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good*</td>
<td>50 (76.9)</td>
<td>119 (58.0)</td>
<td>0.006</td>
<td>0.632(0.316-1.263)</td>
</tr>
<tr>
<td>Bad**</td>
<td>15 (23.1)</td>
<td>86 (42.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality</td>
<td>9 (13.8)</td>
<td>52 (25.4)</td>
<td>0.053</td>
<td>0.798(0.346-1.838)</td>
</tr>
</tbody>
</table>

*Good = GOS 4+5   **Bad= GOS1+2+3
Optimal Cerebral Perfusion Pressure Management at Bedside: A Single-Center Pilot Study

Celeste Dias · Maria João Silva · Eduarda Pereira · Elisabete Monteiro · Isabel Maia · Silvina Barbosa · Sofia Silva · Teresa Honrado · António Cerejo · Marcel J. H. Aries · Peter Smielewski · José-Artur Palva · Marek Czosnyka

CV reactivity preserved (PRx < 0.25) \( (n=15) \)
- mean PRx = -0.04 (SD 0.13)

CV reactivity impaired (PRx > 0.25) \( (n=3) \)
- mean PRx = 0.29 (SD 0.04)

There were no differences in age, SAPSII, and Marshall scores, but patients with overall preserved autoregulation presented significantly higher GCS at admission.
**CPPopt vs CPP and outcome at 6M, 2018**

**severe TBI and spontaneous SAH**
- **6M outcome** of patients at NCCU managed according to CPPopt
- Patients at the general ICU are managed according to guidelines
- No difference between age, gender and severity scores between groups

*p < 0.001*

<table>
<thead>
<tr>
<th></th>
<th>NCCU n, (%)</th>
<th>General ICU n, (%)</th>
<th>Surgical ICU n, (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bad outcome</strong> (GOS 1, 2, 3)</td>
<td>15 (14%)</td>
<td>41 (38%)</td>
<td>52 (33%)</td>
</tr>
<tr>
<td><strong>Good outcome</strong> (GOS 4,5)</td>
<td>50 (47%)</td>
<td>35 (33%)</td>
<td>22 (21%)</td>
</tr>
</tbody>
</table>

ICM + and clinical research in the Intensive Care Department
Spontaneous Intracerebral Hemorrhage
28-day mortality and PRx, % of time of PRx > 0.25 and CPP-CPPopt

We analyzed data from 46 patients, representing a mean duration of 263±173 hours of signal records, with a median length of stay in ICU of 22 (IQR 13) days. The mean age was 62.6±11.8 years old and 24(52%) were male. EVD drainage was applied in 50% of patients and 32.6% were submitted to surgery.
**ICM + and clinical research in the Intensive Care Department**

**CPP-CPPopt along time and outcome at 3M, 2019**

<table>
<thead>
<tr>
<th>Demographic Data</th>
<th>Mean/Median (+/-sd or IQR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Patients</td>
<td>92</td>
</tr>
<tr>
<td>Age (Years)</td>
<td>53 ± 21</td>
</tr>
<tr>
<td>Gender</td>
<td>Male: 79 (86%), Female: 13 (14%)</td>
</tr>
<tr>
<td>GCS at admission</td>
<td>7 (IQR 5)</td>
</tr>
<tr>
<td>APACHE II</td>
<td>19 ± 6</td>
</tr>
<tr>
<td>Apache II mortality (%)</td>
<td>33 ± 17%</td>
</tr>
<tr>
<td>CT Marshall score</td>
<td>3 (IQR 2)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Outcome Data</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>LOS ICU (days)</td>
<td>22 ± 26</td>
</tr>
<tr>
<td>LOS Hosp (days)</td>
<td>48 ± 48</td>
</tr>
<tr>
<td>Mortality</td>
<td>14 (15.2%)</td>
</tr>
<tr>
<td>GOS at 3 months</td>
<td>3 (IQR 2)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Monitoring Data</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>ICP (mmHg)</td>
<td>11.19 ± 5.79</td>
</tr>
<tr>
<td>CPP (mmHg)</td>
<td>85.91 ± 7.37</td>
</tr>
<tr>
<td>PRx</td>
<td>0.03 ± 0.19</td>
</tr>
<tr>
<td>CPPopt (mmHg)</td>
<td>88.74 ± 8.54</td>
</tr>
<tr>
<td>CPP-CPPopt (mmHg)</td>
<td>-2.83 ± 10.23</td>
</tr>
</tbody>
</table>

While, at day 0 CPP-CPPopt is not significantly different between dead and alive, as time evolves during the first 10 days of the study, the model expects: (1) alive individuals to significantly increase CPP-CPPopt within positive range on average by 0.5 each day; (2) dead individuals to progressively lower their CPP-CPPopt values within negative range, at a rate of 0.6 per day (p=0.048).