Monro-Kellie 2.0

The dynamic vascular and venous pathophysiological components of ICP

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L.L.A. Bisschops

Monro described the skull as a rigid structure containing incompressible brain and stated that the volume of blood must remain constant unless: ‘water or other matter is effused or secreted from the blood-vessels’ in which case ‘a quantity of blood, equal in bulk to the effused matter will be pressed out of the cranium’.

Alexander Monro
1733-1817
Confirmation of Monro’s doctrine in human and animal studies: cerebral (in particular, venous) blood volume was similar no matter what the cause of death (hanging, exsanguination) was.
Cushing doctrine: sum of volume of the brain, blood and CSF is constant.
This description fails to explain the importance of volume flow
The dynamic components of ICP

Static Monro-Kellie doctrine: Equal weighting to blood and CSF misses the dynamic reality.

- Slow and steady production of CSF \((\approx 0.35\text{ml/min})\)
- Substantial, continuous blood inflow and outflow \((\approx 700\text{ml/min}, 14\% \text{ of CO})\)

Average male brain volume 1473ml (brain/CSF/blood)
Intracranial blood volume: 100-130ml
CSF volume: 75ml
The dynamic components of ICP

Normal ICP ≈ 5-15mmHg

- Greatly influenced by orthostatic position
  (ICP can be negative when standing up).
- Generally similar to cerebral venous pressures (if no distal obstruction).

Dynamic components:
Arterial influence on ICP
Venous influence on ICP
Extracranial causes of cerebral venous hypertension
The dynamic components of ICP

Arterial influence on ICP

CPP = MAP - ICP
ICP = MAP - CPP; implies no venous involvement
Guidelines: MAP > 80-90, RR_{syst} > 90 mmHg
However: Cerebral Blood Flow resulting from any MAP will differ between individuals (autoregulation, PaCO_{2})

‘static’ view of factors regulating ICP, and focus on arterial inflow alone, led to neglect of important influence of cerebral veins
The dynamic components of ICP

Venous influence on ICP

Cortical
Deeper (anterior)
Central (thalamic)

No muscular wall: vulnerable to compression
The dynamic components of ICP

A role for veins influencing ICP

Failure for (intra- and extracranial) venous efferent flow to precisely match arterial afferent flow yields immediate and dramatic changes in intracranial volume and pressure.

as CBF ↑, venous drainage ↑, with limited venous distension IVP will rise upstream, and thus ICP (similar to Monro-Kellie doctrine).
Causes of raised cerebral venous pressure

Table 1. Suggested classification of venous causes of intracranial hypertension.

<table>
<thead>
<tr>
<th>Classification</th>
<th>Location of resistance/pressure</th>
<th>Clinical examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>I a Focal extramural venous sinus</td>
<td>External compression of a significant venous sinus at a focal point</td>
<td>Depressed skull fracture, periosteal hematoma, tumor.</td>
</tr>
<tr>
<td>I b Focal intermural venous sinus</td>
<td>A focal narrowing within the sinus wall</td>
<td>Idiopathic intracranial hypertension</td>
</tr>
<tr>
<td>I c Focal intramural venous sinus</td>
<td>Obstruction within a significant venous sinus</td>
<td>Sagittal or transverse sinus thrombosis.</td>
</tr>
<tr>
<td>I d Diffuse venous compression</td>
<td>Throughout the venous tree</td>
<td>Any cause of cerebral swelling e.g. hypoxia, cerebral edema, contusions</td>
</tr>
<tr>
<td>II Extracranial venous hypertension</td>
<td>Within the neck</td>
<td>Cervical collars, hanging</td>
</tr>
<tr>
<td>II cervical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>III Extracranial venous hypertension</td>
<td>Within the thorax</td>
<td>Any cause of increased intra-thoracic pressure – Chest infection, adult</td>
</tr>
<tr>
<td>III thoracic</td>
<td></td>
<td>respiratory distress syndrome, mechanical ventilation</td>
</tr>
<tr>
<td>IV Extracranial venous hypertension</td>
<td>Within the abdomen</td>
<td>Any cause of increased abdominal pressure - obesity, obstruction</td>
</tr>
<tr>
<td>IV abdominal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V Orthostatic/gravity</td>
<td></td>
<td>Visual impairment and raised ICP/space obstruction</td>
</tr>
</tbody>
</table>
Diffuse compression of the venous system

Figure 5. CT venograms of a male patient aged 48-year-old male with refractory intracranial hypertension. (a) Transverse sinuses severely effaced with raised intracranial pressure. (b) Following bifrontal decompressive craniectomy the transverse sinus calibre increases dramatically.
Concurrent acute subdural removement and splenectomy

**Figure 6.** Brain herniation occurring at the time of abdominal closure
Relative venous outflow restriction

Intracranially
isolated
diffuse

Extracranially
<table>
<thead>
<tr>
<th>Venous cause of intracranial hypertension</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthostatic/hydrostatic</td>
<td>Avoid hypoxia. Elevate head. Avoid abdominal compression. If intubated, non-depolarising paralysis.</td>
</tr>
<tr>
<td>Cervical</td>
<td>Avoid neck vein compression (e.g. with collars) and maintain in neutral position or position where dominant jugular is optimised. Avoid “double chin” which compresses jugular.</td>
</tr>
<tr>
<td>Intracranial causes</td>
<td>Looks for dominance in transverse sinus drainage. Relieve direct sinus compression, e.g. by depressed skull fracture elevation, release of clot compressing sinus. For sinus thrombosis consider anticoagulation or interventional radiological management. Consider decompressive craniectomy.</td>
</tr>
<tr>
<td>Thoracic causes</td>
<td>Relieve causes of increased intrathoracic pressure. Minimise risk of infection/ARDS. Ventilate with no excessive positive pressure.</td>
</tr>
<tr>
<td>Abdominal causes</td>
<td>Relieve causes of intra-abdominal hypertension. Treat constipation. Place urinary catheter. In extreme circumstances, consider decompressive laparotomy.</td>
</tr>
</tbody>
</table>
Conclusion

Balance between cerebral inflow and outflow is vital

Restrictions in outflow can be as significant as mass accumulation within the cranium

Concentration of interest in ICP/CPP, neglect for venous side

ICP is a function of venous outflow: a combination of intra-cerebral resistance, and cervical, thoracic and abdominal pressures

If no resistance to venous outflow: ICP = 0. IV fluids to ‘maintain CPP’ increase CVP and can worsen ICP