13. Volume-pressure infusion tests: Typical patterns of infusion studies in different forms of CSF circulatory disorders.
Hydrocephalus is far more complex than disorder of CSF circulation

CSF circulation altered

Cerebral blood flow altered

Clinical symptoms of hydrocephalus

Which are reasons and which are effects? What are weights? Does this naive scheme depend on time? Age? Aetiology?
CSF dynamics testing - measurement techniques:
1. Perfusion study
2. Infusion test
3. Bolus injection
4. Constant pressure test
Infusion test: Measurement set-up

- Pressure Transducer
- Manometer lines filled with saline
- 2 25G Butterfly needles
- Ommaya Reservoir
- Syringe drive (50 ml syringe with normal saline)
- Computer running INFUSION TEST software
- Pressure Monitor
Clinical testing of CSF circulation

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Summary
Since shunting is almost a purely mechanical treatment that radically affects pressure-volume compensation, patients’ cerebrospinal fluid hydrodynamics compensation should be examined before a shunt is implanted. Apart from an opening pressure and a resistance to cerebrospinal fluid outflow, pulse amplitude of intracranial pressure and the content of vasogenic waves are useful to gauge cerebrospinal fluid dynamics. Infusion studies, although invasive, may help with the decision about surgery. They also provide basic information for further management of shunted patients, when complications, such as shunt blockage, under- and over-drainage, arise.

Keywords: CEREBROSPINAL FLUID; HYDROCEPHALUS; INTRACRANIAL PRESSURE; FLUID DYNAMICS.

Infusion test trolley: pressure amplifier. Infusion pump, computer+ software
What we measure: mean ICP, heart rate, pulse amplitude of ICP and RAP index
Full identification of cerebrospinal compensation: Identification of the model
Additional information: detection of the lower breakpoint of AMP/P characteristic
## Infusion test: written protocol

### Tests Results:

<table>
<thead>
<tr>
<th>Test</th>
<th>Value 1</th>
<th>Value 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSF Infusion Test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICP baseline [mmHg]</td>
<td>14.74</td>
<td>ICP plateau [mmHg]</td>
</tr>
<tr>
<td>AMP baseline [mmHg]</td>
<td>0.52</td>
<td>AMP plateau [mmHg]</td>
</tr>
<tr>
<td>Rcsf [mmHg*min/ml]</td>
<td>15.18</td>
<td>Elasticity [1/ml]</td>
</tr>
<tr>
<td>PVI [ml]</td>
<td>4.79</td>
<td>Pss [mmHg]</td>
</tr>
<tr>
<td>CSF production rate [ml/min]</td>
<td>0.17</td>
<td>Volume infused [ml]</td>
</tr>
<tr>
<td>Infusion duration [min]</td>
<td>16.18</td>
<td>Normalised Error [%]</td>
</tr>
<tr>
<td>Shunt critical ICP [mmHg]</td>
<td>16.30</td>
<td>Shunt resist. [mmHg*min/ml]</td>
</tr>
</tbody>
</table>
Different technique servo-controlled multiple stage infusion study

University of Umea, Sweden and

LIKVOR - Tools for assessing cerebrospinal fluid dynamics

»A new standard for investigation of CSF-dynamics«

Fig. 6 Curves for the intracranial pressure and external flow describing the constant pressure method. The left graph shows typical $P_{IC}$ data and accumulated infused volume versus time. The right graph is calculated from the time series to the left and shows points for mean pressure and flow determined for each steady state level. Outflow resistance is determined with linear regression and interpreted as the inverse of the slope.
Assessment of cerebrospinal fluid outflow resistance

Anders Eklund · Peter Smielewski · Iain Chambers · Noam Alperin · Jan Malm · Marek Czosnyka · Anthony Marmarou

Fig. 1 Model of the cerebrospinal fluid system described as an electrical analogy. $P_{IC}$ is the intracranial pressure, $P_d$ is the dural venous pressure, $R_{out}$ is the outflow resistance, $C$ is the pressure dependent compliance and $K$ is the elastance coefficient, $q_f$ is the CSF formation rate, $q_{ex}$ is the external infusion of artificial CSF, $q_a$ is the absorption rate, and $q_s$ is the rate of CSF storage. $P_0$ is an optional pressure source describing the reference pressure for CSF storage. In the standard model of Marmarou $P_0 = 0$. In some models an ideal diode is added to the $R_{out}$ branch to model that reverse transport is impossible ($q_a \geq 0$).
One needle infusion:

Examples of infusion studies in different types of hydrocephalus

Normal CSF circulation: $R_{csf} = 8.6$; opening pressure 6 mm Hg
NPH: normal opening pressure, increased Rcsf, slow waves, pulse amplitude well correlated with mean ICP
Atrophic brain
‘Acute’ hydrocephalus – post SAH

Infusion rate [ml/min] = 1.00  
Rcsf [mmHg*min/ml] = 6.12

ICP baseline [mmHg] = 15.93  
AMP baseline [mmHg] = 1.55

ICP plateau [mmHg] = 21.62  
AMP plateau [mmHg] = 3.13

Elastance [1/ml] = 1.00  
PVI [ml] = 2.30

Pss [mmHg] = 14.50

![Graph of ICP, HEART, AMP, and RAP over time]
Vasogenic waveforms of ICP before and during constant rate infusion study
S. Momjian et al., Cambridge 2002:

All vasogenic waveforms increased in magnitude during the test:

![Graph showing the magnitude of vasogenic waves before and during infusion. The x-axis represents AMP, RESP, and SLOW, and the y-axis represents [mm Hg]. The graph indicates significant increases in magnitude during infusion compared to before infusion for all categories.]
Test with plateau wave. Easily controlled with CSF drainage
Post-SAH: Monitoring; high dynamics of ICP

![Graphs showing ICP and RAP over time]
Non-invasive ABP during infusion test
PRx in non-shunted patients

Rcsf [mmHg/(ml/min)]

R = -0.5; p < 0.0005
TCD during infusion study – good synchronization of B waves in ICP and FV
Autoregulation is worse in patients with lower Rcsf

Cerebral Autoregulation among Patients with Symptoms of Hydrocephalus

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Near Infrared Spectroscopy during Infusion Test

18 infusion tests; 17 patients. ICP, ABP, TCD (10 good recordings) and NIRS
Average increase in ICP from 9 to 17 mm Hg, 9 tests with ICP>= 20 mm Hg
Average decrease in CPP by 3 mm Hg, NIRS and FV – no reaction
Rhythmic slow waves of ICP, NIRS, TCD and ABP (periods 20 sec to 3 minutes)

Thanks to Dr. R. Weerakkody
Coherence within bandwidth of slow waves (3 to 0.3 cycles/minute)

Thanks to Dr. R. Weerakkody
Slow waves in ICP and ABP are phase-shifted, in ICP and Hb – in phase

Phase shift between slow waves [degrees]

ICP-ABP  ICP-Hb

Thanks to Dr.R. Weerakkody
Relative change in magnitude of slow waves in ICP and Hb are correlated !!!!

Thanks to Dr. R. Weerakkody
Thanks to Mr. S. Momjian
Periventricular white matter analysis

Slices at the level of the lateral ventricles and above the caudate nucleus (no intervening grey matter to the cortex)

White matter segmented to exclude cortical grey matter

Ventricles segmented

Only the white matter on the lateral aspect of the ventricles retained

Thanks to Mr. S. Momjian
CBF in white matter in NPH decreases as a function of distance from the surface of ventricles.

In normal volunteers distribution of CBF is flat.

Autoregulation is worse closer to surface of ventricles than further away from ventricles (SRoR= static rate of autoregulation, SRoR=100%- good autoregulation; SRoR=0%- disturbed autoregulation)

Early Cushing Response during Infusion Study?

Thanks to Dr. E. Schmidt
Figure 2

\( \Delta \text{ICP} \) vs. \( \Delta \text{ABPm} \)

\( r = 0.46 \)
\( p = 0.006 \)

Thanks to Dr. E. Schmidt
Changes in ICP and other haemodynamic parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>Plateau</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICP (mmHg)</td>
<td>8.2 ± 5.1</td>
<td>25 ± 8.3</td>
<td>16.8 (+204.8%)</td>
</tr>
<tr>
<td>ABPm (mmHg)</td>
<td>106.6 ± 29.7</td>
<td>115.2 ± 30.1</td>
<td>8.6 (+8.1%)</td>
</tr>
<tr>
<td>ABPs (mmHg)</td>
<td>145.3 ± 36.4</td>
<td>156.7 ± 37.4</td>
<td>11.4 (+7.8%)</td>
</tr>
<tr>
<td>ABPd (mmHg)</td>
<td>76.5 ± 31.4</td>
<td>90.5 ± 26.5</td>
<td>14 (+18.3%)</td>
</tr>
<tr>
<td>ABPa (mmHg)</td>
<td>35.1 ± 19.3</td>
<td>38.0 ± 22</td>
<td>2.9 (+8.3%)</td>
</tr>
<tr>
<td>CPP (mmHg)</td>
<td>98.3 ± 29</td>
<td>90.2 ± 30.7</td>
<td>-8.2 (-8.3%)</td>
</tr>
<tr>
<td>HR (min⁻¹)</td>
<td>70.4 ± 10.4</td>
<td>70.3 ± 9.1</td>
<td>-0.08 (-0.1%)</td>
</tr>
<tr>
<td>HRsd</td>
<td>3.11 ± 3.31</td>
<td>4.5 ± 4.5</td>
<td>1.4 (+44.8%)</td>
</tr>
<tr>
<td>HRcv</td>
<td>0.046 ± 0.058</td>
<td>0.067 ± 0.075</td>
<td>0.021 (+46%)</td>
</tr>
<tr>
<td>FVm (cm s⁻¹)</td>
<td>55.6 ± 17</td>
<td>51.1 ± 16.3</td>
<td>-4.5 (-8.2%)</td>
</tr>
<tr>
<td>FVs (cm s⁻¹)</td>
<td>83.0 ± 22.9</td>
<td>80.7 ± 23.3</td>
<td>-2.3 (-2.8%)</td>
</tr>
<tr>
<td>FVd (cm s⁻¹)</td>
<td>37.9 ± 12.8</td>
<td>33.4 ± 12.2</td>
<td>-4.5 (-12%)</td>
</tr>
<tr>
<td>PI</td>
<td>0.828 ± 0.209</td>
<td>0.946 ± 0.257</td>
<td>0.118 (+14.3%)</td>
</tr>
</tbody>
</table>

Thanks to Dr. E. Schmidt
Message to take home:

• All parameters of CSF compensatory model may be estimated during infusion test
• CSF-CBF coupling mechanisms were identified: autoregulation, vasogenic waves, etc.
• Not a single parameter but whole profile of recorded variables characterize disorder of CSF compensation
• In NPH white matter CBF is depleted – closer to ventricles.