10. Monitoring of pressure-volume compensation - VPR and RAP
Various shapes of Pressure-Volume curves

Effects of mannitol and steroid therapy on intracranial volume-pressure relationships in patients

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The intracranial volume-pressure response was measured in 61 patients undergoing continuous monitoring of intraventricular pressure. This test, which determines the increase in intracranial pressure induced by an addition of 1 ml in ventricular CSF volume in 1 second, yields information concerning spatial compensation in patients with intracranial space-occupying processes. On the basis of variability tests, a change in volume-pressure response of 2 mm Hg/ml was accepted as significant. Pronounced enlargement of the ventricles interferes with the test. In patients with intracranial hypertension, intravenous mannitol (0.5 gm/kg) and intramuscular betamethasone (26 mg) both reduce the volume-pressure response significantly more than they reduce intracranial pressure. This suggests that these agents favorably alter the configuration of the volume-pressure curve.

Key Words: volume-pressure response - intracranial pressure - mannitol - corticosteroids

Fig. 1. Graph showing three different theoretical curves relating the volume of an expanding mass lesion to intracranial pressure (ICP). At the same resting pressure (x) the addition of the same volume (y) produces three quite different increases in intracranial pressure.
**Fig. 3.** Graph showing effect of intravenous mannitol (0.5 gm/kg) on ventricular fluid pressure (VFP) and the volume-pressure response (VPR) in eight patients. Asterisks indicate the significance of differences from control. The figures in the center refer to the significance of the differences in the percentage changes in VFP and VPR. Mean values and standard error of the mean are shown.

**Fig. 4.** Graph showing effect of steroids (intramuscular betamethasone 26 mg in 24 hours) on ventricular fluid pressure (VFP) and the volume-pressure response (VPR) in seven patients. Mean values and standard error of the mean are given before and 24 hours after starting steroids.
Volume pressure response is proportional to mean ICP but not always

Intracranial volume-pressure relationships

**Fig. 2.** Graphs showing relationship between resting ventricular fluid pressure (VFP) and the volume-pressure response (VPR) in 21 patients with normal size ventricles (left) and 23 patients with ventricular enlargement (right).
Avezaat and Ejindhoven – VPR proportional to ICP until ‘breakpoint’ level

Fig. 33. Combined plot of volume-pressure response (VPR, open circles) and CSF pulse pressure (filled circles) in single animal (no 1). Note breakpoint in both relationships above which VPR levels off and CSF pulse pressure increases more rapidly.
Compliance $= \frac{\Delta V}{\Delta P}$

$PVI = \frac{\Delta V}{\log \frac{Pp}{Po}}$

Thanks to Dr. I. Piper

Marmarou in “Head injury” Ed. Cooper 1993
• “PVI identifies patients at different risk of developing intracranial hypertension...a decrease in PVI is often associated with an increase in ICP in the following 24 hours.

• PVI may be used in order to establish the appropriate timing for ICP monitoring discontinuation.”
Manual V/P testing

Limited use in practice:
• Episodic measurements
• Labor-intensive process of obtaining repeated measurements
• Difficulties in injecting equal volumes of fluid manually at a constant, rapid rate of injection
• A potential increased risk of infection

Thanks to Dr.I.Piper
The Spiegelberg compliance monitor

**how does it work?**

- The Spiegelberg Compliance Monitor adds then removes small volumes of air to a double lumen balloon catheter placed into the ventricle of the patient.
- It measures the ICP pressure change (mmHg) resulting from the volume addition and automatically calculates the compliance (ml/mmHg) on a minute by minute readout.
Contemporary studies on brain compliance

Experimental evaluation of the Spiegelberg intracranial pressure and intracranial compliance monitor

Technical note


3 ICP devices:
• Spiegelberg Compliance
• Standard Ventricular
• Codman Intraparenchymal

Thanks to Dr. A. Spiegelberg
Intracranial compliance as a bed-side monitoring technique in severely head-injured patients.

Kiening KL, Schoening WN, Lanksch WR, Unterberg AW.
Department of Neurosurgery, Charité, Virchow Medical Center Humboldt-University at Berlin, Germany.

Fig. 1. Episode of pathological ICP. From minute 20–80, ICP increases above 20 mmHg. ICC decreases with a delay of 18 minutes below the critical threshold. \( P_{\text{O}_2} \) is not significantly affected.

Fig. 2. Regression curves of ICC vs. ICP in three different patients representing the different age classes. At a cut-off-point of 20 mmHg in ICP, mean ICC value is 0.9 in a 7 year old, 0.7 in a 35 year old and 0.6 ml/mmHg in a 75 year old patient.
Noninvasive intracranial compliance monitoring

Technical note and clinical results

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Noninvasive intracranial compliance monitoring

Fig. 1. The pressure transmitting window. Anatomical drawing of the structures involved in signal transmission from the CSF spaces to the outer ear.

Fig. 2. Two examples of simultaneously obtained noninvasive and invasive ICP traces. Note the time lag of the noninvasive trace.

HFC proportional to brain Compliance
Pressure-volume curve and relationship between pulse amplitude and mean ICP. Two regions (e.g. Region I, Region II) present the status of compensatory reserve with RAP. The transition point \( (P_z, V_z) \) implies where the curve changes from linear to exponential in shape. Upper curves: pressure-volume relationship. Redrawn from Marmarou 1978, Avezaat and Eijndhoven- 1981
Analysis of Intracranial Pressure Waveform During Infusion Test

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Typical example of change of mean ICP, pulse amplitude of ICP waveform (AMP) and RAP during a CSF infusion study in a patient with normal pressure hydrocephalus before and during infusion.

N = 74

0.45 [-0.65;0.86] Before infusion
0.86 [0.21;0.99] During infusion

P < 1.48*10^{-8}

Wilcoxon paired signed rank test

Thanks to Dr. D-J Kim
Effects of slow waves of ICP during REM phase of sleep on RAP, where time trends show the increase in RAP at the onset of the slow waves. Parameter Slow is defined as the equivalent amplitude of slow (period from 20 sec to 2 min) waves of ICP.

Changes in RAP during B waves recorded overnight

|                | N = 21 | 0.53 [-0.54; 0.83] Without B waves | 0.89 [0.23; 97] During B waves | P < 1.23*10⁻⁵ | Wilcoxon paired signed rank test |

Thanks to Dr. D-J Kim
Effects of body posture in ICP and RAP. The figure shows an increase in RAP when there are changes in body position from sitting up in bed to lying down horizontally during night sleep.

| Changes in RAP with body position during ICP monitoring | N = 20 | 0.36 [-0.71;0.45] Upright in bed | 0.57 [-0.12; 0.86] Horizontal, during night sleep | P < 0.0001 | Wilcoxon paired signed rank test |

Thanks to Dr. D-J Kim
1 y old known, IVH as premature birth
ventricles too large, AS on intial HR- MRI, floor 3rd ventricle down
HC normal,
repeat MRI at 1y: AS resolved, floor 3rd ventricle up

now: ventricles still enlarged, no S&S of raised ICP

Indication for Tx?

Thanks to Prof. M. Schuhmann
Value of Overnight Monitoring of Intracranial Pressure in Hydrocephalic Children

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B waves may overlap in time increasing mean ICP above clearly pathological threshold
RAP correlates with outcome after shunting (N=164 patients with NPH - thanks to Ms. N. Keong)
Cord injury - High pressure, RAP high, PRx fluctuates around ‘0’

Thanks to Dr. M. Werndle
Pressure-Volume curve and pulse amplitude of ICP in TBI
Upper breakpoint of AMP-P relationship and change of RAP from positive to negative.
Monitoring of Cerebrospinal Dynamics Using Continuous Analysis of Intracranial Pressure and Cerebral Perfusion Pressure in Head Injury

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Fig. 1. (a) Relationship between intracranial pressure pulse wave amplitude (y-axis) and mean ICP (x-axis) detected in deep intracranial hypertension in head injured patient. (b) Schematic relationship between the amplitude and mean ICP and dependence of RAP coefficient versus mean ICP.

Fig. 2. Time trend of intracranial pressure (ICP) and RAP coefficient in head injured patient. Plateau wave of ICP was recorded before 6 p.m. Rise in intracranial pressure was associated with significant decrease in RAP to negative values. x-axis: time in hours.
Fig. 7. (a) Amplitude of the fundamental component of intracranial pressure pulse wave (AMP) versus mean ICP. (b) RAP versus ICP

Fig. 6. (a) Amplitude of the fundamental component of intracranial pressure pulse wave (AMP) versus CPP. (b) Short term correlation coefficient between amplitude of the fundamental component of pulse wave and mean intracranial pressure (RAP) versus CPP
Fig. 6. 35-year-old male, admitted at GCS 3. Patient died 5 hours after Camino bolt was inserted. Several ICP plateau waves were recorded with temporal decreases of RAP from around +1 to 0. Before the terminal ICP elevation (15 min) the RAP decreased to 0.
Refractory intracranial hypertension

Fig. 2 (continued)
Negative RAP in patient gradually worsening

Fig. 5. Patient age 18, Glasgow Coma Score 3 on admission. Time average RAP decreased towards 0 on the 2nd day after admission, despite CPP > 65 mmHg. On the third day ICP was increasing steadily with strong vasogenic waves. Time average RAP became negative. Brain stem herniation was confirmed on 4th day after injury.

Refractory hypertension

Fig. 7. Boy, age 15, GCS 3. The RAP coefficient was oscillating around 0 from the very beginning of monitoring, despite gross intracranial hypertension. Patient died around 1 a.m.
Predicting the response of intracranial pressure to moderate hyperventilation

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Fig. 3. Predicting the ICP-response to hyperventilation. ICP Intracranial pressure, $R_{cp}$ index of cerebrospinal compensatory reserve. ICP and $R_{cp}$ are both significantly associated with ICP reduction. Multiple linear regression confirms $R_{cp}$ to be the better predictor than ICP.
True ICP = ICP*(1-RAP)

ICP was moderately elevated during the first day, then increased showing high dynamics. On third day it stabilized but remained above 40 mm Hg and CPP fell below 60 mm Hg. ‘true’ ICP indicated elevated values during the first day (!) then decreased to increase secondarily to the values well above its critical threshold. The patient died.
RTA, 17 years old female, GCS 8
Patient with low ICP and stable CPP all the time. ‘true’ ICP followed real ICP curve, carrying no additionally useful information in this case.
Outcome: Good
Relationship with outcome

‘true’ ICP shows the strongest power to separate survivors from patients who died following head injury.
Clinical observations - refractory intracranial hypertension

- ABP
- ICP
- CPP
- AMP
- RAP
- PRx
- trueICP
Example of RAP improving over time after TBI. Note improvement in PbtiO2.
Permanently elevated RAP after TBI
Initially dense brain oedema improving with successful treatment
Measuring of brain compliance is possible using
-direct (Spiegelberg compliance monitor)
-from ICP waveform analysis

Effect of decompressive craniectomy on intracranial pressure and cerebrospinal compensation following traumatic brain injury

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Difference between RAP and brain compliance (or elastance)

Compliance shows what is the slope of pressure volume curve.

RAP shows where we are relative to lower and upper breakpoints of the curve.

**FIGURE 6.** Pressure-volume curve showing that elasticity (E), the slope of the pressure-volume curve, determines how fast the curve increases within the exponential phase (above points \([V_{z2}, P_{z2}]\)). RAP indicates the relative transition points of each curve (whether the system works below or above points \([V_{z2}, P_{z2}]\), but may not give exact values of E. This implies that RAP and E are not necessarily mutually associated.
Message to take home

Brain compliance can be monitored continuously
RAP index uses cerebral pulse blood injection as an input excitation
RAP close to 0 at low ICP indicates good compensatory reserve
RAP close to 1 indicates diminished compensatory reserve
RAP <= 0 at high pressures indicates reaching critical ICP
RAP is useful adjunct parameter in hydrocephalus and after TBI