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The role of the pulsatile pressure variations in intracranial pressure monitoring

Cees J. J. Avezaat and John H. M. van Eijndhoven*

Department of Neurosurgery, Academic Hospital Rotterdam, and *Department of Electro-Neurology, Erasmus University Rotterdam, The Netherlands

Summary

The magnitude of the pulsatile intracranial pressure variations (CSF pulse pressure) is determined by the elastance of the craniospinal system and by the magnitude of the pulsatile variations in cerebral blood volume (CBV). The pulsatile change in CBV is, among other factors, determined by the compliance of the cerebral vascular bed which, in its turn, is dependent on the cerebral vasomotor tone. This concept has led the authors to devise a method for the assessment of both the elastance and the state of the cerebral vasomotor tone based on the relationship between CSF pulse pressure and intracranial pressure. This relationship was found to be of a linear nature both in clinical patients and in experimental animals. A significant, positive correlation was found between the slope of this relationship and the value of the craniospinal volume-pressure relationship: the elastance coefficient. During elevation of the intracranial pressure a breakpoint was observed in the relationship between CSF pulse pressure and the intracranial pressure above which the pulse pressure increased more rapidly. The elastance remained constant above this breakpoint. The same phenomenon was observed during plateau waves in clinical patients. Induced changes in systemic arterial pressure produced opposite effects on CSF pulse pressure and elastance coefficient. In these cases the discrepancy between pulse pressure and elastance was attributed to the pulsatile changes in CBV and this could be verified by means of electromagnetic flowmetry. The advantage of this method is that all the information is contained within the intracranial pressure signal itself, from which it can be extracted by simple means without the use of invasive tests.

Keywords: CSF pulse pressure, intracranial pressure monitoring, plateau waves, volume-pressure relationship.

1 Introduction

In coping with a volume load the craniospinal system has two mechanisms at its disposal which should be clearly distinguished: volume compensation and volume storage or buffering. Compensation means that the volume load is accommodated by a reduction in the volume of one of the normal constituents of the system, in agreement with the original Monro-Kellie doctrine. This capacity is mainly determined by the dynamics of cerebrospinal fluid (CSF) circulation. So long as the volume load is fully compensated, the total volume of the craniospinal compartment does not increase and intracranial pressure (ICP) does not rise. In volume buffering no compensation takes place and the craniospinal volume is augmented by the total amount of the volume added. The resulting rise in ICP is solely determined by the magnitude of the volume increment and by the physical properties of the system as mathematically expressed by the so-called volume-pressure relationship.

The separation of volume compensation and volume buffering is an artificial one, since in the experimental situation and even more in the clinical context both processes occur simultaneously as shown in Figure 1. However, in the range of elevated ICP the volume compensatory capacity becomes gradually exhausted and the change in ICP is more and more determined by the shape of the volume-pressure curve. That is why, since the introduction of continuous ICP recording into neurosurgical practice, the craniospinal volume-pressure relationships have been extensively investigated [4, 5, 6]. Exact knowledge of the volume-pressure relationship would enable the clinician to predict the course of ICP if a patient were exposed to further intracranial volume additions and, thereby, to select those patients who were particularly at risk. Such knowledge would thus add valuable information to the practice of ICP monitoring which only reveals the level of ICP.

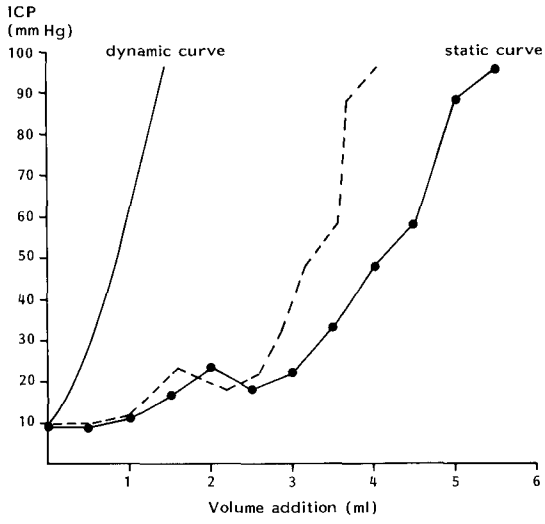

Figure 1. "Dynamic" and "static" volume-pressure curves in a single experimental animal (dog) during continuous inflation of intracranial extradural balloon (1 ml/40 min). The static curve was obtained by plotting steady-state ICP against balloon volume. The dynamic curve was constructed by means of mathematical volume-pressure model of the authors and using bolus injection technique. Volume axis of latter curve represents true increase in craniospinal volume. Dashed curve was obtained by subtracting dynamic from static curve and represents amount of compensated balloon volume at each ICP. Note that volume stored is small compared to volume compensated. Furthermore, during elevation of ICP volume compensation is still effective though gradually less so.

However, the assessment of the volume-pressure relationship requires invasive tests in which extra volume is added to the craniospinal system with all the risks involved such as infection and secondary pressure rises. Moreover, the results from bolus injections into the CSF space are variable in clinical practice, especially in restless patients, making the assessment less reliable. Finally, this method can only be used in ICP measuring techniques with access to the CSF spaces and not e.g. in epidural techniques. The authors have therefore investigated an alternative method for the assessment of craniospinal volume-pressure relationships based on the pulsatile variations of the ICP.

2 The pulsatile ICP variations

The variations in ICP of cardiac origin are caused by the pulsatile variations in cerebral blood volume (CBV). The analogy between the ICP pulsations and the pressure response resulting from bolus injection becomes immediately obvious since both are a pressure response to an increase in craniospinal volume, although the volume changes originate from different compartments, i.e., the vascular compartment and the CSF spaces. The height or amplitude of the pulsatile ICP variations, hereafter termed the CSF pulse pressure (CSFPP), is therefore determined by the magnitude of the underlying volume change, the pulsatile change in CBV, and by the elastance, defined as the slope of the volume-pressure curve at a given pressure (Figure 2). This concept holds out a prospect of using CSFPP as a measure of craniospinal elastance. A problem in this respect is that, in contrast to the uniform volume change of bolus injection, the magnitude of the pulsatile change in CBV is both unknown and perhaps variable.

It should be clearly understood that the magnitude of the pulsatile change in CBV is not equivalent to the pulsatile amount of blood supplied to the craniospinal system by its afferent vessels, since part of this volume is almost immediately compensated for by the simultaneous outflow of blood through the efferent venous system. This compensated blood volume does not contribute to an increase in total craniospinal volume and thus not to the formation of any pressure response. As a matter of fact, if the arterial inflow and venous outflow rates were identical at any point of time of the cardiac cycle, no volume change and, consequently, no pulse pressure would occur at all. The very fact that the in- and outflow curves are pulsatile and different in shape is responsible for the transient, time-dependent

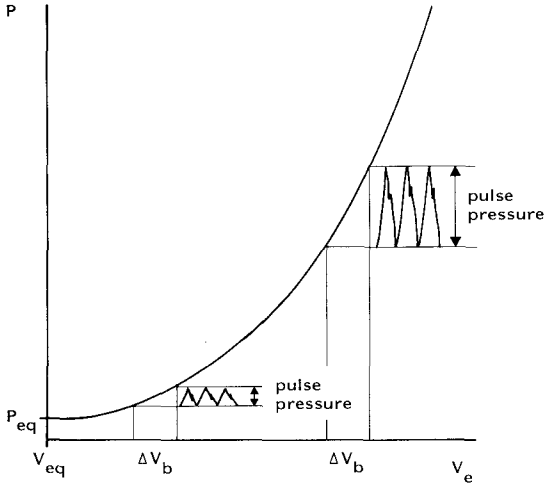

Figure 2. Amplitude of CSF pulse (pulse pressure) increases with rising ICP in accordance with exponential shape of craniospinal volume-pressure curve. Magnitude of pulse pressure is determined by shape of curve and by amount of pulsatile change in CBV (ΔV_b).

change in CBV during a cardiac cycle. The magnitude of the pulsatile change in CBV is thus determined by the temporal relationship between the pulsatile inflow and outflow of cerebral blood as shown in Figure 3.

The shape of the cerebral arterial and venous flow curves, in turn, depend on the impedances of the respective sections of the cerebral vascular bed. The inflow impedance is largely controlled by the vasomotor tone of the so-called cerebral resistance vessels which can be both actively and passively influenced. The outflow impedance can be passively influenced by means of compression of part of the venous outflow tract by an increase in ICP. CSFPP may thus be expected, through the mechanism of the pulsatile change in CBV, to be affected by those factors that influence the intracranial hemodynamics, such as the systematic arterial pessure, the cerbral blood flow autoregulatory mechanism and the ICP.

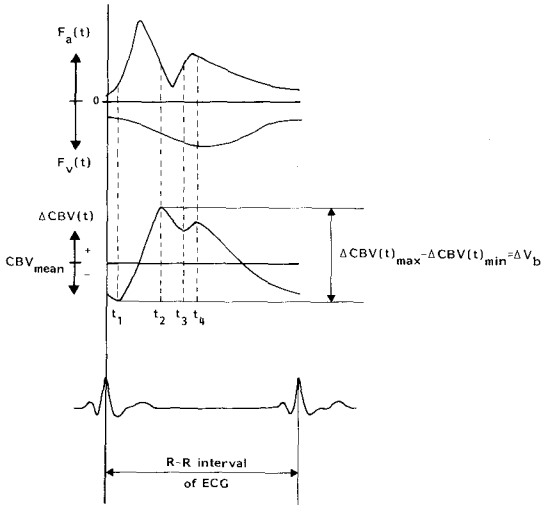

Figure 3. Schematic drawing of cerebral arterial inflow ($F_a(t)$) and venous outflow ($F_v(t)$) curves, interaction between which determines change in CBV ($\triangle V(t)$) during cardiac cycle. Maximum volume change ($\triangle V(t)_{max}$), occurring at time t_2, minus minimum volume change ($\triangle V(t)_{min}$), occurring at time t_1, is defined as pulsatile change in CBV ($\triangle V_b$).

3 Analytical method

The craniospinal volume-pressure relationship is described by a mono-exponential function, which the authors have extended with a constant term [1]:

$$P = P_1 e^{E_1 (V-V_{eq})} + P_o, \qquad (1)$$

where: P = ICP,
P_1, P_o = constant pressure terms,
E_1 = elastance coefficient,
$V-V_{eq}$ = change in *total* neural axis volume (V) with respect to the resting or equilibrium volume (V_{eq}).

The elastance of the system (inverse compliance) E, defined as the slope (dP/dV) at any one point of the volume-pressure curve, can be derived from Equation 1:

$$E = dP/dV = E_1 (P-P_o). \qquad (2)$$

Since E_1 determines the elastance at a given pressure, it is termed the elastance coefficient.
When the volume of the system increases by an amount ΔV, the ICP rises by a pressure response ΔP:

$$P + \Delta P = P_1 e^{E_1 (V-V_{eq}+\Delta V)} + P_o, \qquad (3)$$

which, utilizing Equation 1, can be rewritten as:

$$\Delta P = (P-P_o)\,(e^{E_1 \Delta V} - 1). \qquad (4)$$

This equation describes the relationship between the pressure response resulting from an increase in craniospinal volume and the ICP. The relationship is linear if ΔV is constant, since E_1 is constant by definition, in which case the slope ($\Delta P/(P-P_o)$) is determined by the product of E_1 and ΔV. P_o is equal to the intercept with the pressure axis. E_1 can be obtained by the technique of rapid bolus injection into the CSF space, in which case it is assumed that the injected volume is equal to ΔV. Utilizing Equation 4 E_1 can be computed from:

$$E_1 = \frac{1}{\Delta V} \ln \left[\frac{\Delta P}{P-P_o} + 1 \right]. \qquad (5)$$

Equation 4 can also be applied to the relationship between CSFPP and ICP, if the pulsatile change in craniospinal volume is assumed to originate from a change in the volume of cerebral blood (ΔV_b):

$$CSFPP = (P-P_o)\,(e^{E_1 \Delta V_b} - 1). \qquad (6)$$

This is a linear relationship if ΔV_b is constant in which case the slope ($CSFPP/(P-P_o)$) is a measure of both E_1 and ΔV_b. If E_1 is determined by bolus injection, ΔV_b can be calculated from:

$$\Delta V_b = \frac{1}{E_1} \ln \left[\frac{CSFPP}{P-P_o} + 1 \right]. \qquad (7)$$

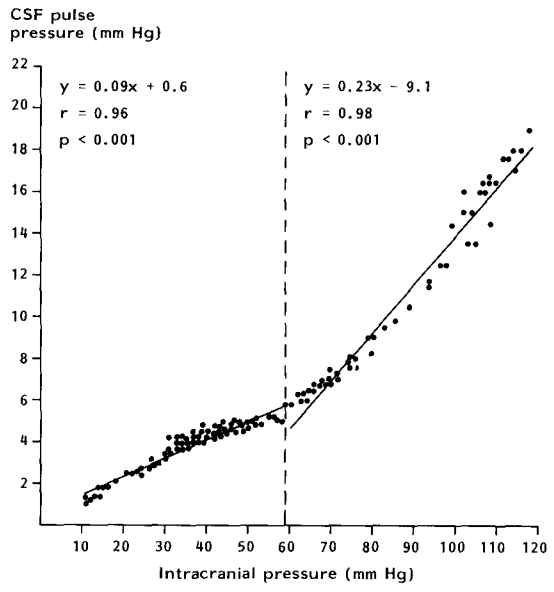

Figure 4. Relationship between pressure response following rapid bolus injection of 0.05 ml through ventricular catheter (VPR) and intracranial pressure during continuous inflation of intracranial balloon in a single dog. Regression lines and equations are given. Note linear increase of VPR with ICP up to a breakpoint above which VPR is variable but does no longer increase. ICP range above breakpoint is conceived as one with constant elastance.

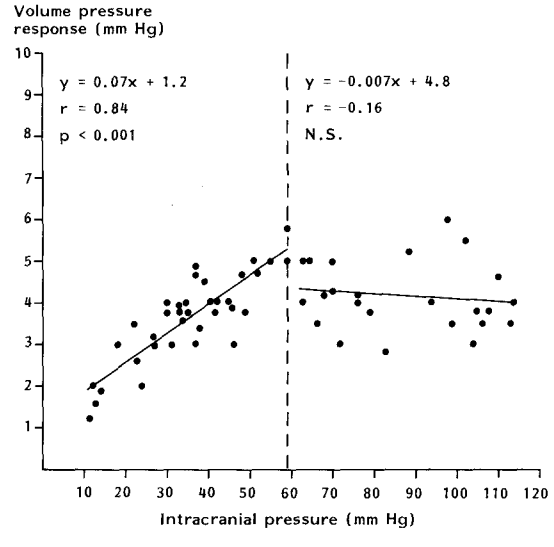

Figure 5. Relationship between CSF pulse pressure and intracranial pressure during continuous inflation of intracranial balloon in a single dog (same animal as in Figure 4). Note linear increase of pulse pressure with ICP up to a breakpoint above which CSF pulse pressure increases more rapidly. Breakpoint ICP corresponds with that in volume-pressure relationship shown in Figure 4.

4 Relationship between CSF pulse pressure and ICP

4.1 Experimental observations

Figure 4 shows the relationship between CSFPP and ICP in the dog, during elevation of the ICP by continuous inflation of an extradural balloon. It is characterized by a breakpoint dividing the relationship in two linear segments of which the one above the breakpoint has a steeper slope. The relationship between the pressure response following rapid bolus injection and the ICP shows a similar breakpoint at the same ICP (Figure 5). In a group of six animals the breakpoint occurred at a mean ICP of 60 mm HG. However, in contrast with CSFPP, the pressure response did not further increase above the breakpoint ICP but remained constant. Applying the equations of the mathematical concept to these findings several conclusions can be drawn. Below the breakpoint it follows from the linear CSFPP-ICP relationship that the pulsatile change in CBV is constant and does not change with the ICP. The slope of this relationship is determined by the magnitude of the volume change and by E_1. Above the breakpoint the volume-pressure relationship is no longer exponential in nature but linear (constant elastance). This implies that the increase in CSFPP in this ICP range must be caused by a progressive increase in magnitude of the pulsatile change in CBV.

The breakpoint in the above relationships was found to coincide with the ICP at which cerebral blood flow, as measured by electromagnetic flowmetry in the vertebral artery, began to fall. In these experiments [1] the pulsatile change in CBV was calculated from the vertebral artery pulsatile flow profile assuming a constant venous outflow. As Figure 6 shows, the reduction in flow in the high range of ICP occurs mainly in the diastolic portion of flow. As a

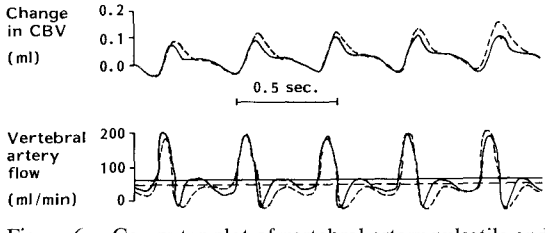

Figure 6. Computer plot of vertebral artery pulsatile and mean (straight line) flow and of change in CBV at extremely elevated ICP (dashed curves), superimposed on the results obtained at normal ICP. Flow was electromagnetically measured in the dog. Note that at elevated ICP, in spite of reduction in mean flow, the pulsatile change in CBV increases as a result of shift of flow from diastole to systole.

result, the amplitude of the systolic flow pulse relative to the mean flow level, which was assumed to be equal to the venous outflow, is increased causing an increase in the pulsatile change in CBV. The relative increase in the flow pulse amplitude due to a shift of flow from diastole to systole is in the opinion of the authors caused by a decrease in the arterial input impedance as a result of loss of the cerebral vasomotor tone (vasoparesis).

Figure 7 shows the results in a series of six animals in which the CSFPP-ICP relationship was compared with the volume-pressure relationship during elevation of the ICP and drug induced systemic arterial hypotension and hypertension. Lowering the blood pressure caused an increase in CSFPP with respect to the pressure response following bolus injection. This was due to both an absolute increase in CSFPP and a decrease in the pressure response (E_1 from 1.5 to 1.1 1/ml). It follows from these results that the increase in CSFPP must have been caused by an increase in the pulsatile change in CBV (from 0.048 to 0.085 ml). In agreement with these results the vertebral artery flow profile showed an increase in pulsatile flow due to a shift of flow from diastole to systole (Figure 8). The explanation, again, follows from the reduction in inflow impedance due to an active vasodilatory response, as part of the autoregulatory mechanism, elicited by the decrease in cerebral perfusion pressure.

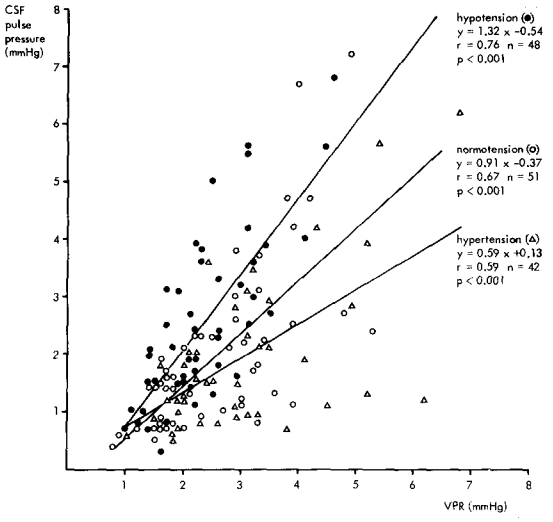

Figure 7. Composite plot of relationship between CSF pulse pressure and pressure response following intraventricular bolus injection of 0.05 ml (VPR) at three levels of systemic arterial pressure (87.5 ± 7.8 (SD) mm Hg, 132.2 ± 7.8 mm Hg and 173.2 ± 11.0 mm Hg) in six dogs during cerebral compression by inflation of intracranial balloon. The differences between the slopes of the regression lines were caused by an increase in CSF pulse pressure and a decrease in VPR during arterial hypotension and by the opposite effects during arterial hypertension.

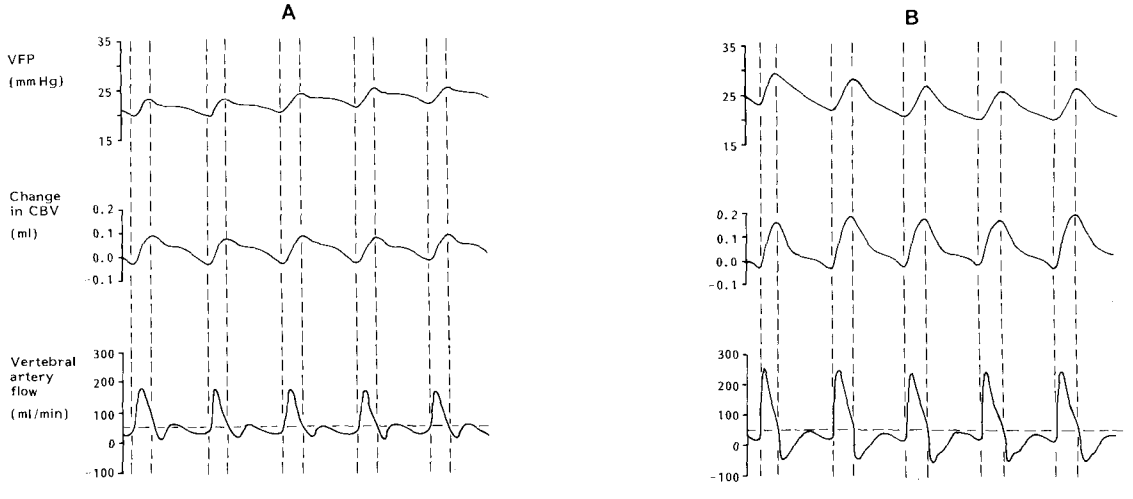

Figure 8. Computer plot of vertebral artery pulsatile and mean (dashed line) flow, change in CBV and ventricular fluid pressure (VFP) during five cardiac cycles in the dog. A = control and B = systemic arterial hypotension. Note that mean flow is not changed, whereas systolic flow is markedly increased at the expense of diastolic flow. As a result pulsatile change in CBV increased from 0.117 ml to 0.210 ml. Note also the synchronization of the extreme values of the change in CBV and CSF pulse.

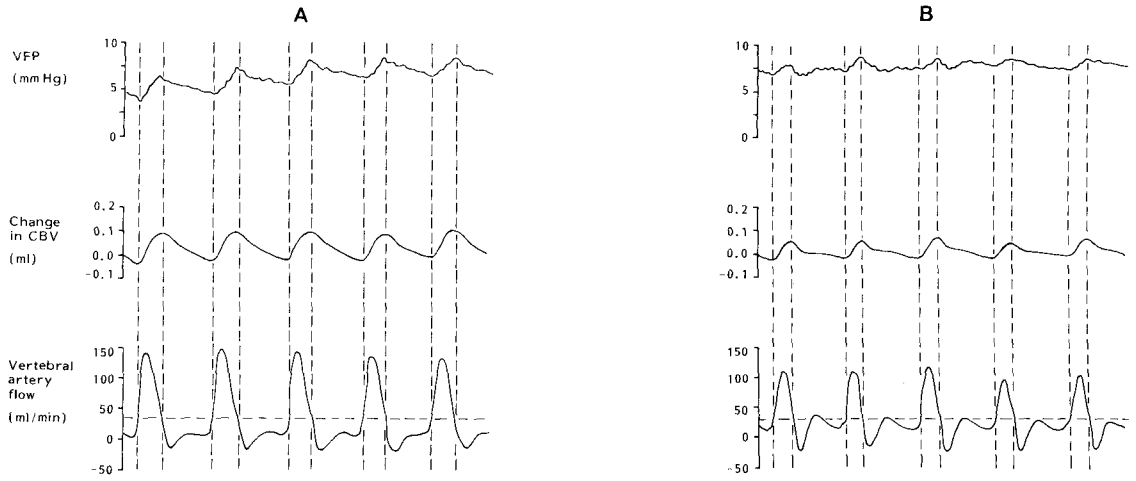

Figure 9. Computer plot of vertebral artery pulsatile and mean (dashed line) flow, change in CBV and ventricular fluid pressure (VFP) during five cardiac cycles in the dog. A = control and B = systemic arterial hypertension. Change in flow profile can be observed consisting of flow shift from systole to diastole. As a result pulsatile change in CBV decreased from 0.126 ml to 0.079 ml.

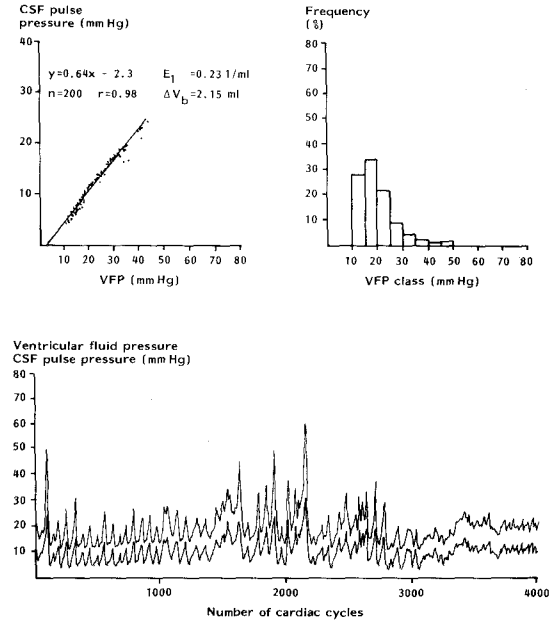

Figure 10. Computer plot of ventricular fluid pressure (VFP) recording, comprising period of 4000 cardiac cycles, in patient with supratentorial tumor. Bottom: tracings of mean VFP (upper) and CFS pulse pressure (lower). Top left: relationship between CSF pulse pressure and mean VFP. Regression equation and calculated values for elastance coefficient (E_1) and pulsatile change in CBV ($\triangle V_b$) are shown. Top right: frequency distribution of VFP represented as histogram with pressure classes of 5 mm Hg.

During systemic arterial hypertension the reverse results were obtained. E_1 was increased from 1.5 to 1.8 1/ml, whereas the pulsatile change in CBV was decreased from 0.048 to 0.027 ml with a corresponding decrease in CSFPP. Figure 9 shows the effect of raising the blood pressure on the vertebral artery flow profile.

4.2 Clinical observations

Also during clinical ICP monitoring highly significant linear relationships were found between CSFPP and ICP. Figure 10 shows a computer analysis of a recording interval of approximately one hour (4000 cardiac cycles). If E_1 is assessed by means of bolus injection, the pulsatile change in CBV can be computed from the slope of the CSFPP-ICP regression line. So long as the volume change remains constant, the slope value may serve as a guide of changes in volume-pressure relationship. In a series of 65 short-term pressure recordings. performed in a group of patients suffering from a variety of intracranial disorders, a mean slope value of 0.40 (range: 0.09–1.06) was found. The mean E_1 was 0.26 1/ml (range: 0.05–0.99 1/ml) implying a mean pulsatile change in CBV of 1.67 ml (range: 0.36–4.38 ml).

A significant positive correlation was found between the slope value and E_1 (Figure 11) indicating that the slope is indeed a measure of the craniospinal volume-pressure-relationship. However, the correlation is too weak to allow for a confident prediction of E_1 on the basis of the CSFPP-ICP relationship in individual patients. The explanation for this is that

the second determinant of CSFPP, i.e. the pulsatile change in CBV, evidently varies considerably between patients.

When so-called plateau waves occurred a breakpoint was observed in the CSFPP-ICP relationship, caused by a steepening of the regression line during the pressure wave (Figure 12). An increase in E_1, implying a steeper volume-pressure curve, seems the most plausible explanation at first sight, as it is generally believed that plateau waves arise when the elastance is high [3, 6]. However, when the pressure responses obtained at base-line ICP and at the crest of the plateau waves (usually from fluid withdrawal instead of injection) were compared, it appeared that the latter responses were generally lower than expected on the basis of linear extrapolation of the base-line data (Figure 13). This phenomenon leaves no other conclusion than that the rapid increase of CSFPP during plateau waves must be caused by an increase in the pulsatile change in CBV. The phenomenon described here bears a likeness to the breakpoint found in the experimental series suggesting that the explanation should follow the same line of reasoning. This would fit quite well into the generally accepted explanation for the origin of plateau waves, i.e. a sudden loss of cerebral vasomotor tone [7], a phenomenon which has been termed cerebral vasomotor paralysis by Langfitt et al. [3].

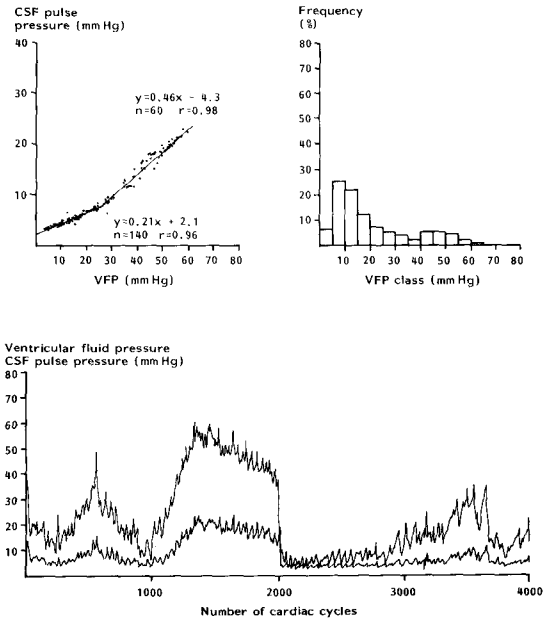

Figure 12. Computer plot of ventricular fluid pressure (VFP) recording in adult patient with hydrocephalus due to glioma of pons. Tracing shows typical plateau wave and some abortive waves. Data of base-line VFP (3–24 mm Hg) and of waves (25–60 mm Hg) were treated separately. Note breakpoint in relationship between CSF pulse pressure and VFP. During plateau wave CSF pulse pressure increased more rapidly.

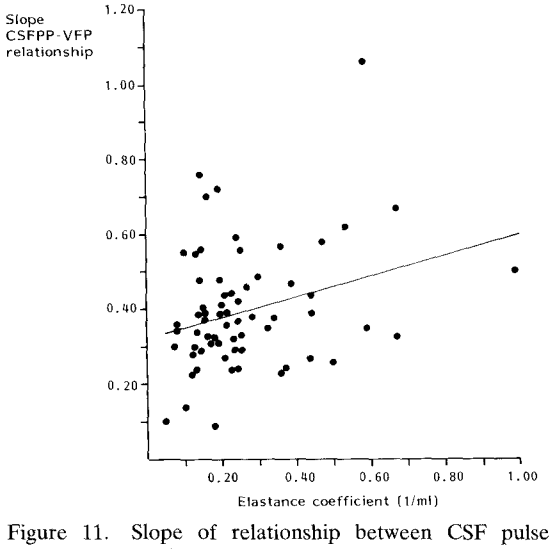

Figure 11. Slope of relationship between CSF pulse pressure (CSFPP) and ventricular fluid pressure (VFP) plotted against elastance coefficient in 65 continuous VFP recordings showing significant, positive correlation ($y = 0.27x + 0.3$; $r = 0.28$; $p<0.05$).

5 Conclusion

In view of the evidence presented it can be concluded that, under certain conditions, the relationship between CSFPP and ICP can be used as a feature of the craniospinal volume-pressure relationship. The main condition is that there are no hemodynamic factors involved which affect CSFPP through the mechanism of the pulsatile change in CBV. The hemodynamic factors involved are the cerebral blood flow autoregulatory state and the cerebral perfusion pressure, but also the carbon dioxide tension of the arterial blood and the heart rate as previously reported by the authors [2].

Because of the rather wide variation in the pulsatile change in CBV between individual patients, invasive tests are still required for the absolute assessment of the volume-pressure relationship. These tests could be performed at the beginning of each pressure recording in order to "calibrate" the slope of the CSFPP-ICP relationship as it were. So long as the hemodynamic factors remain stable, the slope value is a measure of changes in volume-pressure relation-

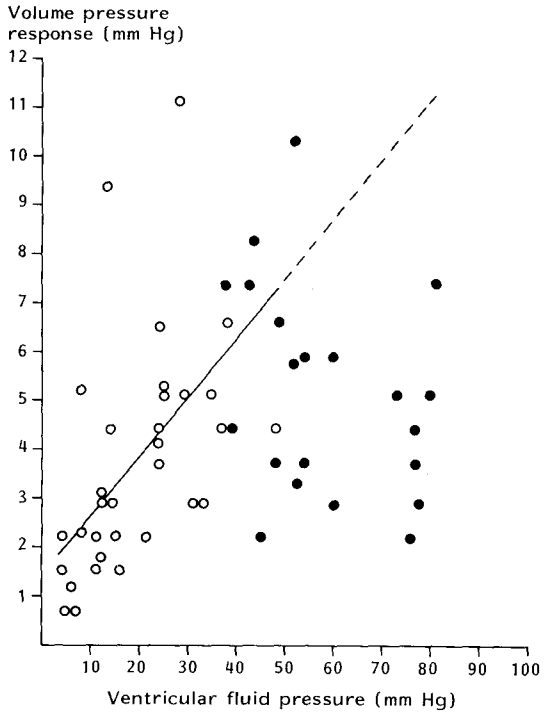

Figure 13. Relationship between pressure response following intraventricular bolus injection of 1 ml (VPR) and ventricular fluid pressure in four patients. Data were obtained during base-line ICP (open circles) and plateau waves (filled circles). At base-line significant linear relationship is observed ($y = 0.12x + 1.5$; $r = 0.51$; $p<0.01$) which is lost during plateau waves. VPR at plateau waves was generally lower than expected on the basis of extrapolation of regression line, indicating increased compliance during plateau waves.

ship. Whenever there is a change in slope during ICP monitoring, the volumetric tests may be repeated in order to establish whether the change is due to a change in E_1 or in the pulsatile change in CBV. In this way the use of volumetric tests can be rationalized and the associated risk of infection be minimized. On the other hand, on the basis of the information provided by the studies on the effects of various hemodynamic factors on the pulsatile change in CBV, it may still be possible to interpret changes in CSFPP-ICP relationship in terms of changes in volume-pressure relationship without the use of invasive tests.

The phenomenon of a relatively low elastance in contrast with a high CSFPP, observed during impairment of cerebral blood flow autoregulation and during plateau waves, suggests that under the condition of cerebral vasoparesis or vasoparalysis CSFPP is a better clinical feature than the pressure response resulting from bolus injection, as in that case the clinical state of a patient is determined by the loss of autoregulation rather than by the increased craniospinal compliance. This opens up new perspectives for CSFPP as an indicator of the integrity of the cerebral vasomotor tone.

The major advantage of this method however lies in the possibility of extracting all this information from the ICP signal itself on a continuous basis and without the necessity of access to the CSF spaces.

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Cees J. J. Avezaat, M. D.
Department of Neurosurgery
Academic Hospital Rotterdam/Dijkzigt
Dr. Molewaterplein 40
3015 GD Rotterdam
The Netherlands