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EDITORIAL (BY INVITATION) - NEUROSURGERY GENERAL

Origin of intracranial pressure pulse waveform

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Intracranial pressure (ICP) is more than a number. This slogan may mean that apart from its mean value, ICP contains many transients and waves, which are useful for describing intracranial pathology. Whoever attempts to analyse morphology of ICP waveforms or resolve the meaning of secondary indices, providing the study is scientifically sound, does a good job, as these landmarks of many intracranial pathologies are still not sufficiently understood. Recent paper of Evensen and Eide [10], describing correlation between arterial blood pressure (ABP) and ICP pulse waveforms, belongs to this family and in our opinion deserves a short editorial.

Pulse waveform of intracranial pressure (ICP) contains unique information about the cerebrospinal system. It has proven to be useful in many brain diseases like traumatic brain injury and haemorrhagic stroke but also in hydrocephalus and idiopathic intracranial hypertension. It combines markers of both cerebrovascular hemodynamic and cerebrospinal pressure-volume compensation [11]. But 'reach in information' also means that the signal is not easy to analyse, and many pulse-derived indexes proposed before never reached a wider recognition. Conventional wisdom states that increased pulse amplitude of ICP signifies worse cerebrospinal compensatory reserve [2]. Increased pulse waveform was claimed to associate with better outcome after shunting in normal pressure hydrocephalus [8]. Experimental widening of lateral ventricles after insertion of pulsating with heart rate frequency balloon [6] gave an assumption that exaggerated pulse amplitude was a possible cause of ventriculomegaly. Term 'pulsatile encephalopathy' is associated with a situation when the compliance of CSF space is low to the extent that it is unable to damp pulsations of cerebral blood volume [13].

Magnitude of pulse waveform is traditionally associated with a steepness of pressure volume (P/V) curve—the steeper it is, the lower is the compensatory reserve and greater ICP pulse amplitude. However, the most important factor is the cerebral arterial blood stroke volume; greater volume produces greater ICP pulse response. But things are not so easy as pressure-volume curve is not universally exponential. It has lower and upper breakpoints—see Fig. 1. Below lower breakpoint (at low ICP), it is linear, which means that brain compliance below this threshold does not depend on ICP. Above upper breakpoint (so-called 'critical ICP'), it bends to the right (or up, as in Fig. 1, as orientation of axes has been changed, from traditional volume (x) and pressure (y)), indicating that system gains some extra compensatory reserve, on the expense of collapsing vascular bed, which leads to a decrease in global CBF and fast-developing acute ischaemia. Analogical to a pressure-volume curve is a relationship between pulse amplitude and ICP. It also has two breakpoints. Below ICP denoting lower breakpoint of P/V curve, pulse amplitude is constant, independent on mean ICP. Above the same breakpoint, amplitude increases with mean ICP. Further, above 'critical ICP', amplitude-pressure curve decreases its slope and finally starts to decrease with mean ICP increasing further. This 'upper breakpoint' of amplitude-pressure curve is dual to 'upper bend' of P/V curve and indicates critical ischaemic ICP threshold. Slope of amplitude-pressure curve between lower and upper breakpoints is approximately equal to elasticity times cerebral arterial blood stroke volume. Increased slope was demonstrated to indicate good chance for improvement after shunting in NPH [1].

Origin of ICP pulse waveform has been discussed for quite a long time. Three phenomena have been identified: passive extension of walls of cerebral arteries due to diastole-systole arterial blood pressure (ABP) rise, increase in cerebral arterial blood volume during cardiac cycle and fluctuations of conditions for venous blood outflow.

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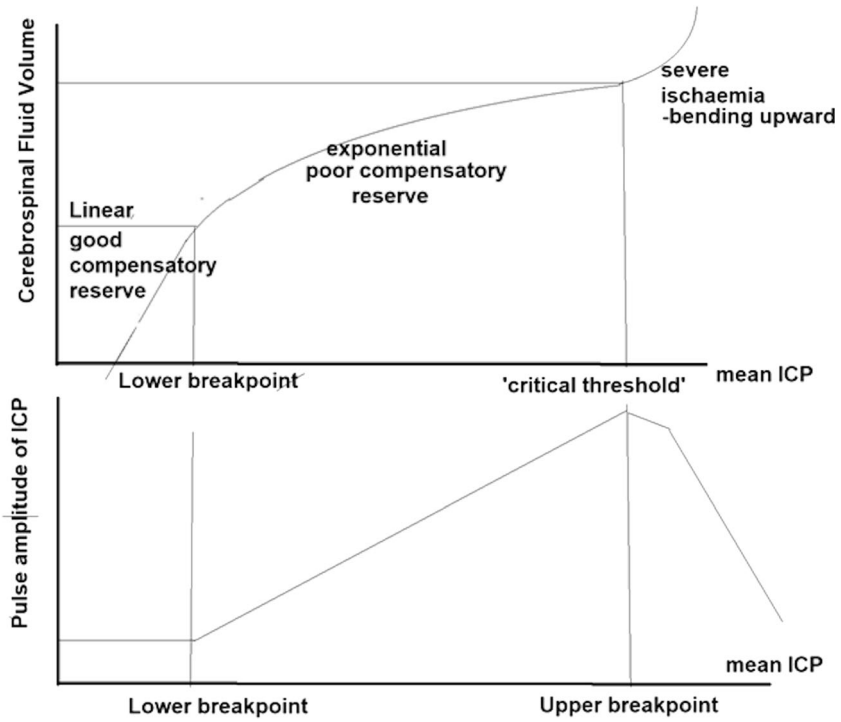

Fig. 1 Hypothetical relationship between added cerebrospinal volume (*y*-axis) and mean ICP (*x*-axis)—upper panel. On lower panel, a corresponding relationship between pulse amplitude of ICP (*y*-axis) and mean ICP(*x*-axis) is presented. Two breakpoints may be identified: lower breakpoint, at low ICP, between linear and exponential part of P/V curve and upper breakpoint which signifies 'critical threshold of ICP' above which acute ischaemia may occur

In the recent paper, Evensen and Eide [10] studied the relationship between ABP and ICP pulse waveforms. Correlation between these pulses, in most cases, occurred to be weak and in some instances at most modest. This seems to confirm previous results [9], also indicating a lack of similar correlation. Moreover, moving correlation coefficient between ABP and ICP pulses exhibited generally poor relationship to cerebrovascular reactivity PRx index. This is probably secondary to a fact that in ICP pulse, all previously mentioned components (ABP pulse, blood volume and venous outflow) commonly associated with peaks P1, P2 and P3 are present and overlap in time. P1 is synchronous with systolic peak in ABP pulse, P2 is synchronous with a peak of estimated cerebral arterial blood volume [4], and peak P3, if observed, is synchronous with venous blood outflow. Peaks P1 and P2 may change their proportions, depending on cerebrospinal compliance. Usually P1 is dominant, but when P2 starts to increase above P1, this is characteristic for diminishing compliance.

This leaves a little space to comment how we calculate both ICP and ABP pulse amplitudes. Evensen and Eide use peak-to-peak value for ICP waveform. The method is good and precise, but it does not distinguish between peaks P1 and P2. And those peaks, as we described above, have distinctively different physiological interpretation: P1 is derived from immediate distension of the walls of the arteries due to systolic rise in ABP pulse. We can infer that P1 will be well correlated to ABP pulse amplitude. P2 is correlated with increase in arterial blood cerebral stroke volume. As cerebral arteries (to the lesser extent than arterioles) are compliant, blood volume increase and peak P2 are delayed in comparison with rapid extension of peak P1. This can be studied with simultaneous recording of ABP, ICP and blood flow velocity in basal cerebral arteries using transcranial doppler velocimetry [4] (see Fig.2), or using phase-coded MRI technique [3].

We commonly use the Fourier analysis-derived amplitude of fundamental harmonic of pulse waveform [5]. It also does not distinguish, like in Eide's method, between peaks P1 and P2, but improves signal-to-noise ratio, which is helpful in any precise pulse waveform-related studies.

Another point worth to comment on is a potential change in a shape of waveform between aortic blood pressure and peripheral pulse, commonly measured in clinical practice. Transformation from recorded peripheral pulse to aortic pressure curve can be done with digital filtering [12]. Transformed aortic blood pressure is more spiky, and systolic pressure is usually lower. But we still do not have approved method on how to transform aortic pulse to cerebral pulse. Distance from the heart to the circle of Willis in adult is 30–40 cm; there are number of branching arteries and acute bends, which undoubtedly produce further change of the shape of the radial artery in comparison with arterial pulse in brain.

Finally, ICP pulse amplitude is an important indicator that ICP is monitored properly. The principle 'no pulse, no pressure' [7] is very useful in practice of pressure monitoring; however it cannot be used dogmatically. At very low ICP,

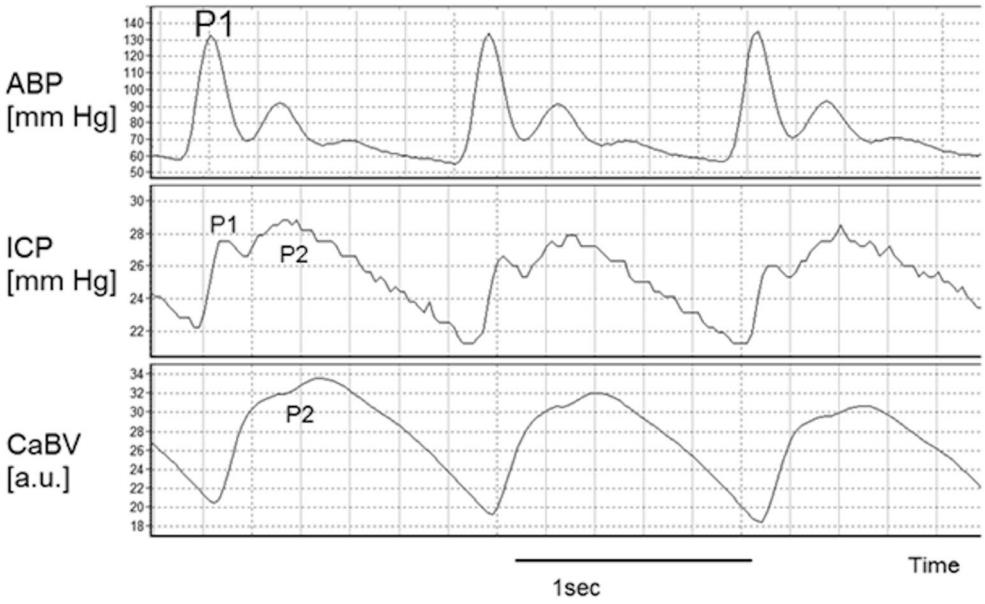

Fig. 2 Example of peaks P1 and P2 seen on ABP, ICP and cerebral arterial blood volume (CaBV) waveforms. CaBV is derived from transcranial Doppler blood flow velocity [3] within a cardiac cycle. Peak P1 in ICP is associated with systole of ABP. Peak P2 is associated with a maximum of cerebral arterial blood volume

pulse may be not very well visible, particularly hidden under the noise level in time domain or masked by dominant respiratory wave. Spectral analysis with searching for the peak associated with a heart rate is very helpful in these cases.

ICP pulse pressure contains incredibly rich information on the cerebrospinal system dynamics, and therefore its analysis deserves wider recognition.

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