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Intracranial Pressure Oscillations (B-Waves) Caused by Oscillations in Cerebrovascular Volume

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With 3 Figures

Summary

In experiments during stepwise increases in intracranial pressure, B-waves could be provoked synchronously with pial vascular diameter oscillations. The vascular oscillations are known to be a sign of myogenic cerebrovascular regulation. An increase in their amplitude indicates vessel wall relaxation and impending failure of myogenic autoregulation. Therefore, vascular oscillations provoked during intracranial hypertension can be ascribed to a fall in transmural pressure and vessel wall tension. The resulting oscillations in the cerebral blood volume are reflected by ICP oscillations and lead to synchronous variation in ICP, the so-called "B-waves" with a frequency of 0.5 to 2 per minute.

Keywords: Intracranial pressure; B-waves; cerebral blood volume; myogenic autoregulation.

Introduction

In 1960, Lundberg published a comprehensive description of ventricular fluid pressure measurements in patients, including rhythmic pressure variations and a discussion of their clinical implications. Oscillations with a frequency of 2–0.5/minute at normal and increased ICP, called "B-waves", were observed only under pathological circumstances and hence were considered as being clinically relevant even when observed at normal ICP-levels [13]. Changes in the cerebrovascular resistance during intracranial hypertension were assumed to explain the B-waves, thus suggesting variations in the cerebral blood volume [18, 19], which had already been one of Lund-

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berg's explanations [13]. It was shown by previous work from our laboratory [4, 5], that pial vessel diameters can oscillate with the same frequency as B-waves. The maximal amplitude of these vascular oscillations was observed during vessel wall relaxation at low levels of intravascular pressure.

It was therefore investigated, whether vascular oscillations appear in the situation of vessel wall relaxation produced by an elevated intracranial pressure and whether vascular rhythms would appear synchronously with rhythmic variations in the intracranial pressure which are defined as B-waves.

Material and Methods

Eleven cats were investigated under pentobarbital and nitrous-oxide anaesthesia, immobilized with 60 μgkg^{-1} pancuroniumbromide. Following endotracheal intubation the animals were ventilated with a Loosco baby-respirator. The femoral artery and vein were cannulated for the continuous recording of the mean arterial pressure (MAP), the sampling of blood for gas analysis and for drug administration. The intracranial pressure (ICP) was continuously monitored using a plastic cannula inserted into the cisterna magna and a Statham P 23 dB transducer with an HSE electromanometer. The pial vessels were observed through a closed cranial window made in the right parietal region, as described elsewhere in detail [1]. Diameter variations were recorded with a TV multi-channel videoangiometer [3] attached to a Leitz intravital microscope with Ultropak objectives [1]. Changes in cerebral blood volume were semiquantitatively recorded using a photometric technique [2] and focussing a photomultiplier on a cortical surface area free of major pial vessels and measuring the extent of light absorption by the cortical surface.

In 8 animals, intracranial pressure was increased in steps of 10 mm Hg from resting to 50 mm Hg by the intra-cisternal infusion of a mock CSF via a Y-shaped connector to the cannula in the cisterna magna (0.6–1.2 ml/minute). In 3 animals, brain oedema was produced by water intoxication, infusing 2–5 ml/minute of aqua bidestillata intravenously.

MAP, ICP, pial arterial and venous diameters were continuously recorded by a multi-channel pen recorder.

Results

In 5 out of 8 animals B-waves occurred synchronously with pial vessel oscillations during a period of increased ICP brought about by intracisternal CSF infusion. As summarized in Table 1, oscillations started at various levels of ICP, ranging from 15 to 40 mm Hg. The amplitudes varied between 2 and 12 microns in the pial vessels, between 1 and 5 mm Hg in the ICP. B-waves started together with a rise in amplitude of vascular oscillations of the same frequency, mostly 1–2 per minute. The amplitude of ICP and vascular oscillations increased with rising ICP. On several occasions, ICP and

Table 1

Experiment number	B-waves			Pial vessel rhythm
	Appearance at ICP <	Duration	Amplitude (mm Hg)	Amplitude (μm)
1	15 mm Hg	25 minutes	1 ~ 1.5	4 ~ 8
2	40 mm Hg	30 minutes	1 ~ 3	2 ~ 3
3	30 mm Hg	40 minutes	1 ~ 4	4 ~ 10
4	20 mm Hg	50 minutes	1 ~ 5	8 ~ 12
5	20 mm Hg	40 minutes	1 ~ 3	4 ~ 8

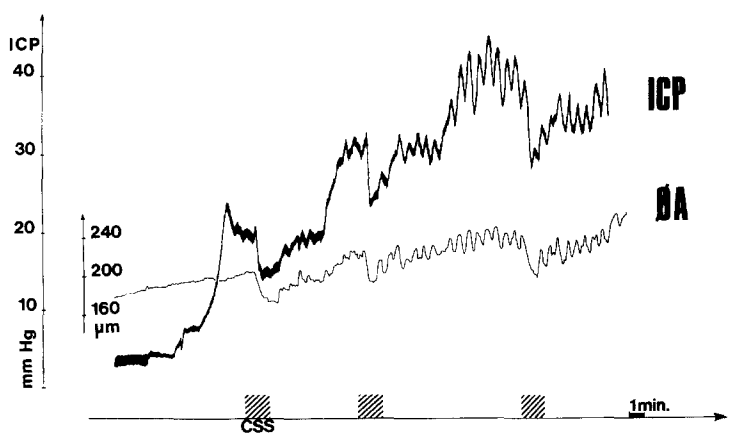

Fig. 1. During increasing intracranial pressure (*ICP*) induced by CSF-infusion, ICP and a pial artery (*ØA*) start oscillating in the frequency-range of B-waves. *CSS* = cervical sympathetic stimulation interrupts the rhythm until the constricted vessel dilates again

vascular oscillations were synchronous with oscillations in the MAP. More frequently, however, the rhythms were independent of the MAP. Single examples are given in Figs. 1–3. Rhythms of both ICP and pial vessels were sometimes superimposed by other rhythms such as the typical 5–8/minute vascular rhythm [4, 5] (Fig. 2). The latter was, however, more evident in vessel diameter curves; interferences with the B-type oscillations sometimes masked the synchronicity with B-waves in the ICP (*e.g.* Fig. 2). A rapid increase in vessel tone, as produced by sympathoadrenergic vasoconstriction [6], immediately reduces the amplitude of the B-waves and sometimes induced an increase in the amplitude of 5–8/minute vascular oscillations (Fig. 2).

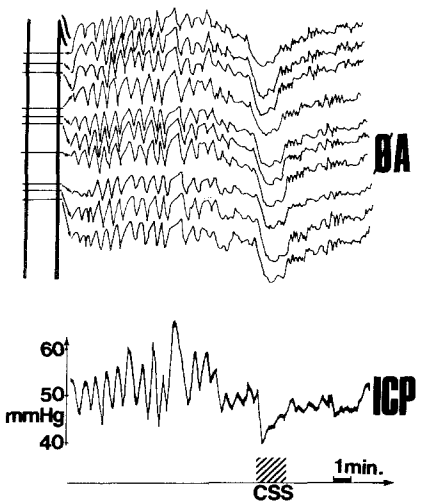

Fig. 2. B-waves in the ICP-curve at an ICP-level of around 50 mm Hg. There are synchronous oscillations in the pial arterial portions (*ØA*) as sketched on top left. *CSS* = cervical sympathetic stimulation constricts the artery, interrupts the B-type oscillation, reduces the B-waves amplitude for several minutes, and afterwards makes a 5–8 per minute vascular rhythm appear

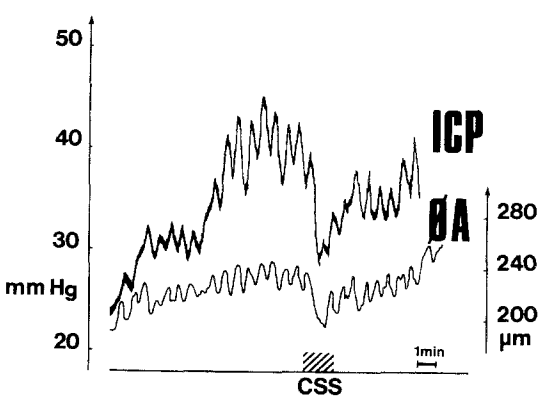

Fig. 3. B-waves on ICP-curve at ICP level between 20 and 40 mm Hg. Synchronous oscillations in the diameter curves of a pial artery (*ØA*). *CSS* = cervical sympathetic stimulation causes a transitory vasoconstriction and decrease in the rhythms' amplitude due to the rise in vessel wall tone

Discussion

The first question for the reader facing the presented data might be: why should vessel diameters oscillate and how might these oscillations become synchronous all over the brain's vasculature and hence bring about oscillations in the blood volume?

Pial vessel diameter oscillations have been described as an expression of an intrinsic myogenic regulatory mechanism [4, 5] which have also been seen in other vascular beds [10–12]. Metabolic and neurogenic regulatory influences did not affect vessel diameter oscillations except via an alteration in the vessel wall tension. The intrinsic mechanism might be explainable on the basis of the behaviour of the smooth muscle cells of the "visceral type" or "single unit type" which are present in the medial layer of cerebral arteries [8]. Their membrane potential is low ($\approx$ 50 mV) and unstable, resulting in a slow contraction-relaxation sequence. Siegel [14–17] explained this frequency by the metabolic peculiarities of these cells. The basic stimulus for smooth muscle cell contraction is stretching due to intraluminal pressure, as first described by Bayliss [7]. The slow mechanism of depolarization, typical for these "visceral type"—smooth muscle cells, leads to a slow constriction of the vessel. After this slow depolarization, the smooth muscle cells relax, and subsequently the vessel dilates. The latter leads again to stretching of the smooth muscle cells thus closing the cybernetic (autoregulatory) loop. The consequence is—again—rhythmic contraction of the vascular smooth muscle cells. Under resting conditions, the amplitude of these regulatory oscillations is very low, simulating a smooth, continuous, analogue regulatory mechanism of vessel wall tone. Near the level of complete vessel wall relaxation, an overshooting peristaltic-like oscillation starts [5], until, finally, during complete vessel wall relaxation, the oscillation stops due to a lack of constrictory stimuli. Similarly, during normotensive or hypertensive vasodilatation, overstretching of smooth muscle cells can stop oscillations, when the stretching force is stronger than the cells' contractory force.

This process, in addition, affects ICP via the changes in cerebral blood volume which necessarily result from the dilatation and constriction of the vessels. The rhythmic rise and fall in CBV causes ICP to rise and fall (oscillate). The rise in ICP during vasodilatation results in an increase in circumferential vessel pressure, *i.e.* a decrease in the transmural pressure, and further vasodilatation due to the smooth muscle cell relaxation resulting from this relative fall in the intraluminal pressure. Stretching of the smooth muscle cells causes the

vessel to constrict, ICP to fall, and start the cycle again. This oscillating ICP can be assumed to be uniform throughout the cranial cavity. Thus, ICP-oscillations, induced by the vessels' smooth muscle cells, become the mediator of the synchronicity of vessel oscillations. A brain stem centre or specialized pacemaker cells within the media-layer of vessels are no longer required to explain the synchronicity of vessel oscillations. The level of ICP at which the oscillations markedly rise in amplitude may depend on other factors influencing vascular tone, such as CO_2 and metabolic acidosis.

The conclusions drawn from the simultaneous observation of ICP and vessel-reactions correlate with data obtained by Portnoy *et al.* [14] and Chopp *et al.* [9] using the analysis of the Starling resistor model to show that the CSF-pressure wave may explain the changes in cerebral vasomotor tone, *i.e.* cerebrovascular autoregulation.

Synchronous oscillations observed in the traces of the superior sagittal sinus pressure in our experiments might directly be induced by the ICP oscillation. Increasing venous pressure then becomes a third stimulating factor causing smooth muscle cell a) relaxation, b) contraction: the increase in venous pressure increases peripheral resistance but at the some time decreases CPP by reducing the arterio-venous pressure difference.

The amplitude of vascular rhythms increases as the intraluminal pressure falls, and achieves its maximum near the lower limit of CBF autoregulation, shortly before total vasoparalysis indicates complete loss of autoregulation. These almost peristaltic oscillations of the vessels seem to express an excessive overshooting of the intrinsic myogenic regulatory mechanism. A similar situation seems to be produced by an increase in the intracranial pressure, *i.e.* an increase in perivascular pressure and a consequent fall in transmural pressure: the resulting vessel wall relaxation provokes a rise in the amplitude of the diameter oscillations. The higher the ICP, the lower is the transmural pressure and the higher the amplitude of vessel diameter oscillations. Vessel diameter variations therefore result in variations in the vascular volume, *i.e.* blood volume. Synchronously with the observed vascular diameter variations there are oscillations in the cerebral blood volume which in turn result in oscillations in the ICP. At higher ICPs, small changes in the intracranial volume become more relevant for a further rise in ICP, when considering the pressure volume relationship. Since the ICP waves during the described experiments were B-waves, as defined by Lundberg in 1960 [13], and the B-waves occurred synchronously with vascular oscillations *i.e.* an oscillation of cerebral blood volume, it can be postulated that B-waves originate from oscillations in the cerebral

blood volume. Similar suggestions have previously been made by Symon *et al.* [19] and Sørensen *et al.* [18] from experiments that indicated a progressive decrease in cerebrovascular resistance with increasing ICP up to a level around 70 mm Hg, when the cerebral blood flow sharply falls due to the critical lowering of perfusion pressure, when the ICP, reflected by the venous pressure, approaches arterial pressure. At ICP levels below 50 mm Hg, progressive pial vascular relaxation was in fact shown as net vasodilatation [6].

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