Circulatory disturbance of the venous system during experimental intracranial hypertension

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The venous drainage system during increased intracranial pressure (ICP) was studied in dogs. The ICP was gradually increased to the level of the systemic blood pressure while related arterial and venous pressures were monitored. The blood flow through the parasagittal intradural venous channels (lateral lacuna) was also measured to test the collapsibility of these vessels. The cortical venous pressure was constantly 50 to 200 mm H2O higher than the ICP regardless of the degree of elevation, while the sagittal sinus pressure remained at 50 to 75 mm H2O unless the central venous pressure was elevated by respiratory disturbance. Flow through the lateral lacuna decreased as the ICP was increased. The authors conclude that the low pressure in the sinus and the consistency of the walls of the lateral lacuna allow gradual stenosis of the lacuna during increased ICP.

Key Words · intracranial hypertension · cerebral venous pressure · cerebral blood flow · superior sagittal sinus · lateral lacuna

It is well known that elevation of intracranial pressure (ICP) causes reduction of cerebral blood flow (CBF). Kety, et al.,6 first reported the reduction of CBF when the ICP was elevated above 450 mm H2O by using the N2O clearance method. Since then, many authors have reported the reduction of CBF under increased ICP by using various methods such as radioisotope clearance,17 magnetic flow meter,2,4 and heat clearance.5 However, none of those reports has clarified the exact mechanism of the reduction of CBF under increased ICP.

Ohwada, et al.,8 observed blood flow in the cortical vessels using a fluorescein serial angiographic method under increased ICP and found that the speed of flow was markedly decreased as the dye moved from arterioles to the venous system. They concluded that the cause of the decrease in the CBF under increased ICP must have existed in the drainage system. Shulman18 recently reported a constant pressure difference between the ICP and cortical venous pressure (CVP), and suggested there might be stenosis of the venous system somewhere between the cortical veins and the dural sinuses. Risberg, et al.,11 reported an increase of regional cerebral blood volume during acute reversible intracranial hypertension.

All of these findings point to a vascular congestion as the cause. The present study was designed to investigate the behavior of the venous drainage system under increased
ICP in an attempt to find the exact site of hypothetical stenosis in that system. Our study consists of two parts. Experiment 1 concerns the pressure gradient through the various intracranial vessels as ICP is elevated. Experiment 2 concerns the collapsibility of the specific vessel in which the first experiment suggested stenosis.

Experiment 1: Pressure Gradient Determinations

Materials and Methods

Eighteen mongrel dogs unselected as to age or sex and weighing from 10 to 15 kg were anesthetized with intravenous thyanal sodium 5 mg/kg initially, supplemented by 2 to 3 mg/kg as necessary. The animals were tracheotomized and allowed to breathe spontaneously throughout the experiment; rate and mode of respiration were recorded by a thermister bridge applied to the opening of the tracheostomy. Rectal temperature was maintained at 37.5° to 38° C by a small thermoblanket. Systemic blood pressure (SBP) and central venous pressure (CVP) were recorded by passing polyethylene tubes through the femoral artery and vein so that the catheter tips lay in the intrathoracic portion of the aorta and vena cava.

Cortical arterial pressure (CAP) was measured by the following technique. After removing a temporal muscle on the left side, small openings were made in the skull and dura over the Sylvian fissure. One of the arteries with an outer diameter of 0.5 to 0.8 mm was selected for cannulation. A tapered polyethylene catheter with 0.5 mm outer diameter at its tip was inserted in a proximal direction through a small opening made in the side of the artery. The artery was ligated around the catheter to prevent leakage from the opening. The dural opening was covered by a layer of Gelfoam and then by a piece of cotton which was tightly fixed to the skull with alkyl-α-cyanoacrylate monomer. This method was a satisfactory way to reconstruct the solidity of the skull and prevent leakage of cerebrospinal fluid (CSF).

Cortical venous pressure (CVP) was measured by passing a tapered polyethylene catheter into one of the parasagittal cortical veins on the left side approximately 10 to 12 mm posterior to the coronal suture. The method of cannulation was much the same as in the method used on the cortical arteries. The catheter was inserted in a proximal direction in one group and in a distal direction in another. The tip of the catheter was placed in various positions between the cortical vein and the junction of the bridging vein with the lateral lacuna of the superior sagittal sinus.

Pressure in the superior sagittal sinus (SSSP) was measured by a polyethylene catheter small enough not to alter the venous flow in the sinus (outer diameter, 1 mm) which was inserted with its opening directed posteriorly; its tip was placed 15 mm posterior to the coronal suture. An operating microscope was used in handling these small vessels.

The ICP was measured by inserting a small condom balloon containing 0.05 cc of water into the subdural space over the left posterior parietal area. All of these catheters were connected to pressure transducers (MP-4 or LPU-0.1).* Great care was taken to prevent leakage of fluid at the level of the heart in all pressure recordings. All of the openings of the dura and the skull were closed in the same fashion as described under the measurement of CAP. To elevate the ICP, a small rubber balloon was placed in the frontal pole on the right side through a small skull opening; the balloon was inflated by injecting water while the ICP was recorded on the oscillograph.

Results

As originally reported by Shulman, the CVP was constantly higher than the ICP. In our study, the difference between these two pressures was 50 to 200 mm H2O, with a mean value of 161 mm H2O. The difference in pressure between the ICP and CVP was almost constant in each case regardless of the level of ICP. No significant difference in pressure was observed in the venous channel between the parasagittal cortical vein and the junction of the lateral lacuna with the bridging vein. The CAP correlated fairly well with the SBP; CAP was 15 to 20 mm

*Pressure transducers MP-4 and LPU-0.1 manufactured by Töyö Measuring Instruments, Co., Ltd., No. 104, 1-chome Chofuminemachi, Ota-ku, Tokyo, Japan.
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Fig. 1. Oscillograph recordings, with ICP elevated to 1300 mm H₂O. The CVP is about 50 mm H₂O higher than the ICP when the ICP is at 300 mm H₂O and increases as the ICP is elevated, maintaining a constant pressure difference. SBP = systemic blood pressure, CAP = cortical arterial pressure, CVP = cortical venous pressure, and SSSP = superior sagittal sinus pressure.

Hg lower than SBP under normal ICP, and rose with the SBP and increasing ICP.

Regardless of these changes in the CAP and CVP resulting from increased ICP, the SSSP remained at almost the same level; it decreased slightly as the ICP reached an extremely high level (Figs. 1 and 2). Although the SSSP was quite independent of the ICP, it rose easily in response to elevation of the central venous pressure caused by respiratory distress (Fig. 3).

The average values of CAP, CVP, and SSSP in relation to ICP in eight dogs indicated that the pressure gradient between the CAP and CVP gradually decreased as ICP was elevated, while the pressure gradient between the CVP and SSSP continuously increased as ICP was elevated (Fig. 4). On the basis of this evidence, we concluded that decrease in the CBF during increased ICP was mainly dependent on the decrease of the pressure gradient between the cortical arteries and veins and that the cause for elevation of the CVP with the ICP must be located between the junction of the bridging vein and lateral lacuna and the superior sagittal sinus, namely, in the lacuna close to the sinus. The second study was carried out to verify this assumption.

Experiment 2: Collapsibility of the Lacunar Venous Channel

Materials and Methods

Seven adult mongrel dogs unselected as to age or sex and weighing 10 to 15 kg were sacrificed by injecting nicotine intravenously. The superior sagittal sinus, lateral lacuna, and the cortical vein located approximately 10 to 15 mm posterior to the coronal suture were exposed through a skull opening 10 to 15 mm in diameter. The dura was opened to expose a portion of the bridging vein. A small tapered catheter with an outer diameter of 0.5 mm at its tip was inserted into a cortical vein and the tip
advanced to the orifice of the lateral lacuna through the bridging portion of the vein (Fig. 5). A ligature was applied around the bridging vein to prevent back flow. A longitudinal incision was made in the superior sagittal sinus at the outlet of the lacuna, and a catheter with an outer diameter of 1 mm was placed over the outlet and fixed to the inner wall of the sinus with alkyl-α-cyanoacrylate monomer. Methods used for measuring ICP, elevating ICP, and closing the dural and skull openings were the same as in Experiment 1.

Normal saline was continuously injected under constant pressure into the catheter on the venous side, and the outflow from the catheter on the sinus side was measured under various intracranial pressures. The height of the outlet was always maintained 50 mm above the zero level, which is equal to the normal value of SSSP in living animals. The outlet of the catheter was placed over a horizontally positioned needle of a voltmeter, and thus each drop from the outlet moved the needle, causing an instantaneous recording on an oscillograph as one vertical line for each drop (Fig. 5).

Results
Flow through the lacuna decreased as the ICP was elevated to 500 mm H₂O (Fig. 6). The rate of flow was in almost reverse proportion to the ICP regardless of the inflow pressure (Fig. 7). These findings indicate that the lacunar portion of the venous channel is quite capable of being gradually compressed by increasing ICP. Thus this may be the mechanism that maintains the cortical venous pressure at a proper level in relation to the ICP.

Discussion
Because of the stable SSSP, which is much lower than the ICP, and the high pressure in the cortical and bridging veins, there must be a large intraluminal pressure gradient between the inlet and outlet of the lacuna. Therefore, there must be a portion where the intralacunar pressure is lower than ICP. Since the difference between the ICP
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Fig. 3. Recordings with ICP elevated to 1000 mm H₂O. The SSSP elevates readily in response to the elevation of central venous pressure caused by respiratory distress, although the SSSP is quite independent of the level of the ICP. SBP = systemic blood pressure, Central VP = central venous pressure, and Resp = respiration.

Fig. 4. Graph showing the average values of CAP, CVP, and SSSP in relation to ICP in eight dogs. Pressure gradient between CAP and CVP gradually decreases as ICP is elevated, while the pressure gradient between CVP and SSSP continuously increases as ICP is elevated.

Fig. 5. Set-up used in Experiment 2. The collapsibility of the parasagittal intradural venous channels (lateral lacuna) was investigated by measuring the rate of flow through these channels while increasing intracranial pressure.
and intraluminal pressure of the lacuna at the portion close to the sinus is so great, if the wall of the vessel close to the sinus is thin and easily collapsible like that of the cortical vein, it seems likely that the closest portion is completely occluded by slight elevation of ICP, thus stopping the cerebral circulation.

It is quite important to note that the flow rate in this portion has a linear relationship with the ICP, instead of a sudden and complete stop of the flow as the ICP is elevated. As Permutt and Riley\textsuperscript{10} have demonstrated in a model simulating this condition, the most likely portion to be compressed is that closest to the side where the intraluminal pressure is low. Therefore, we believe the CVP is maintained at a proper level in relation to the ICP because the thin-walled cortical vein is not directly connected to the sinus where the intraluminal pressure is much lower than ICP. Instead, the venous blood drains into the sinus through the lacuna whose wall has just enough collapsibility to maintain the CVP at a proper level. Although the present experiment was limited to study of the drainage system around the superior sagittal sinus, anatomical examinations of the dura mater indicated that most of the bridging veins did not drain directly into the major dural sinuses, but first passed through intradural venous channels similar to the lateral lacuna of the superior sagittal sinus.

Wolf and Forbes\textsuperscript{15} in 1928 first suggested elevation of cortical venous pressures as the cause of decrease in the blood flow through the cerebral blood vessels under increased ICP. By observing dilatation of pial arteries and veins under moderately increased ICP, they postulated that the increased ICP was transmitted readily to the interior of the thin-walled veins in which the pressure was relatively low and, thus, the intracranial...
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venous pressure was raised and blood flow retarded. Wright,16 Bedford,1 and Hedges and Weinstein,3 using dogs, demonstrated dilatation of cortical vessels under increased ICP while the SSSP showed only little change. Hedges and Weinstein3 postulated that this cerebral venous stasis resulted from cuffing of the cerebral veins where they crossed the subarachnoid space and was responsible mainly for a decrease in cerebral blood flow under increased ICP.

More recently, Shulman and Verdier14 suggested that the most sensitive segment of the intracranial vasculature for change in resistance due to ICP elevation involved the subarachnoid veins, probably close to their point of entry into the dural sinuses. Langfitt, et al.,7 also believed that compression of the veins at their junctions with the dural sinuses was the cause of decreased CBF during moderate increases in ICP; the superior sagittal sinus only was compressed when intracranial pressure had become greatly elevated. Shapiro, et al.,12 demonstrated morphological changes in the superior sagittal sinus under increased ICP, and suggested collapse of the sinus as the cause of venous congestion. Osterholm9 using measured SSSP and sinography in patients with acute traumatic intracranial hematoma, suggested that the cause was stenosis of the transverse sinus.

Although it is quite possible that the sinuses may change their shape under extremely high ICP, the effect of such morphological changes on cerebral hemodynamics must be evaluated with great care. We feel that based on the data at hand, the regulatory mechanism controlling intracranial venous pressure is located in the intradural portion of the drainage system next to the major dural sinuses. During increased ICP this “intracranial venous pressure regulation mechanism” (as we have named it) prevents collapse of the thin-walled cortical vessels, but also leads to a decrease in cerebral blood flow.

References


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