Special report

Summary of First International Symposium on Intracranial Pressure, Hannover, Germany, July 27–29, 1972

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The author, at the invitation of the Journal, reports a symposium on intracranial pressure. Points of view on the various topics discussed are reduced to a consensus so that the article provides a brief summary of current views on this important subject.

—The Editor

The First International Symposium on Intracranial Pressure (ICP) was held in Hannover, Germany, July 27-29, 1972. It had been organized by Prof. H. Dietz and Dr. M. Brock under the auspices of the University of Hannover Medical School. Sixty-four papers were presented and discussed by an invited group of scientists and clinicians from all parts of the world. The scientific material was divided into several categories: 1) methodology; 2) experimental studies of the effects of focal brain damage and brain compression on ICP; 3) the effects of raised ICP on cerebral blood flow (CBF) and metabolism; 4) the relationship of ICP, CBF, and neurological status in patients with acute brain insults and hydrocephalus; and 5) the effects of various drugs on ICP, with particular emphasis on anesthetic agents. At the conclusion of the meeting the organizing committee and chairmen of the scientific sessions reorganized the material and wrote summaries that will be included in the proceedings of the symposium. The proceedings will be published by Springer-Verlag in 1973.

The interpretations and opinions expressed in this brief summary represent a consensus rather than a personal opinion.

Techniques for Measuring ICP

The only reliable method of measuring ICP is directly from the intracranial space. Cerebrospinal fluid (CSF) pressure measurements may accurately reflect ICP in the majority of patients, but increased ICP often is not fully transmitted to the spinal subarachnoid space in patients with brain swelling and intracranial mass lesions. The two principal types of instruments used for continuous recording of ICP are a catheter or cannula inserted into a CSF space, usually the lateral ventricle, and a solid state transducer placed in the epidural or subdural space or occasionally the brain tissue. The advantages of the ventricular cannula, besides its proven reliability in hundreds of cases reported, are that it provides a method for calibration of the recording system in vivo and the ability to withdraw CSF for immediate reduction of ICP or chemical analysis. The disadvantages of the method...
are the necessity of penetrating the brain, the requirement of an external fluid connection that may increase the risk of infection, and the difficulties encountered in establishing or maintaining a pressure recording in patients with small ventricles or ventricles that contain debris that tends to obstruct the cannula. The incidence of intracranial infection in experienced hands is 1% to 2%, and morbidity and mortality directly attributable to the technique appear to be virtually nil. The incidence of recording failure, especially in patients with acute head injuries and brain swelling, has not been established but probably is significant.

Several solid state transducers were described at the meeting. The basic principle in all of them, as in pressure transducers in general, is displacement of a pressure-sensitive diaphragm by a change in ICP. The movement of the diaphragm is converted to electrical energy; the electrical output is proportional to the mechanical input. Most of the transducers are a few millimeters in diameter. They are inserted through a burr hole into either the epidural or subdural space, almost always the former in the studies described at the symposium. The principal advantage of a solid state transducer compared to an intraventricular cannula is that the intradural space is not penetrated, thereby reducing the chances of brain damage and infection. Most, if not all, of the instruments are subject to baseline drift that can be large and cannot be detected during the recording period with the exception of recent models that permit calibration in vivo. Calibration is performed through a length of tubing (snorkel) from the diaphragm to the exterior.

A modification of the "transensor" was also described. It consists of an implantable glass-enclosed tuned circuit that is activated by an external oscillator. There are no external connections, and the device can be left in place indefinitely. However, there are a number of technical problems to be solved, and probably the instrument can record only pulse pressure, not absolute pressure.

Transmission of Increased ICP

During expansion of an intracranial mass lesion or brain swelling the rise in pressure in the spinal subarachnoid space tends to lag behind the increasing pressure in the intracranial space due to obstruction of subarachnoid pathways at the tentorial incisura, the foramen magnum, or both locations. This dissociation of pressures has been demonstrated in both experimental animals and man, and occasionally supratentorial pressure may equal the blood pressure at a time when lumbar subarachnoid pressure is normal.

Pressures recorded with extradural solid state transducers are equal to or slightly higher than ventricular and subarachnoid pressures under normal conditions, and as ICP is increased extradural pressure rises out of proportion to CSF pressure. The difference may reach 20 to 30 mm Hg at high pressure levels. Whether this represents a true difference in pressure across the dura or is an artifact of the recording system has not been determined. Bilateral extradural transducers were used to record temporal fossa pressures in experimental animals before and after occlusion of one middle cerebral artery. During the acute phase following the occlusion, large differences in pressure were observed across the supratentorial space. These observations explain reductions in regional CBF in or adjacent to mass lesions and focal brain swelling.

Several methods to measure brain tissue pressure were described including a catheter with a fluted tip and a catheter that contains long, fine strands of wool that protrude from the tip of the plastic tubing as a wick. Tissue pressure measured with these devices may be higher or lower than intraventricular pressure, and the change in tissue pressure with rising extracerebral pressure is uncertain. A problem with these techniques is the necessity of creating a fluid-filled cavity within the brain substance at the tip of the catheter. The cavity pressure might be artifactually high and thus not bear a predictable relationship to true changes in tissue pressures surrounding it.

Pathophysiology of Increased ICP

Pressures waves are fluctuations in ICP that may occur spontaneously or be caused by changes in systemic blood gases. There was agreement at the symposium that the original terminology for pressure waves
introduced by Lundberg\(^1\) should be retained except that the term "plateau wave" should include both plateau and A waves, and the term "A wave" should be eliminated. Plateau waves are recurring increases in ICP to values of 50 to 100 mm Hg lasting 5 to 20 minutes that generally arise from an elevated base of ICP. B waves occur more frequently (\(\frac{4}{10}\) to \(\frac{2}{2}\) min) and are of less amplitude and little clinical significance. During a series of plateau waves, both amplitude and duration tend to increase, and a terminal wave may follow in which ICP rises to the level of the systemic arterial pressure (SAP) and CBF ceases. Pressure waves are due primarily to changes in cerebral blood volume.

Blood flow through the brain is a function of the inflow and outflow pressures, the diameter of the cerebral vascular bed, and blood viscosity. Normally, the perfusion pressure across the brain is considered to be the internal carotid artery pressure minus jugular vein pressure. As ICP rises, however, cerebral venous pressure increases, and there is experimental evidence that the two pressures are nearly identical at all levels of increased ICP until the veins collapse. Thus, ICP is often considered to be the outflow pressure when it is elevated:

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\text{CBF} = \frac{\text{mean SAP} - \text{mean ICP}}{\text{CVR}}
\]

The reduction in CBF that ultimately occurs as ICP continues to rise is due to a reduction in perfusion pressure, and according to this concept CBF ceases when ICP and SAP are equal. An alternative approach is to use the equation:

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\text{CBF} = \frac{\text{mean SAP} - \text{mean JVP}}{\text{CVR}}
\]

in which circumstance the outflow pressure changes little, because it is known that the jugular vein pressure (JVP) does not change much with variations in ICP. According to this hypothesis CBF ceases when ICP equals SAP because the cerebral vascular bed has collapsed and CVR is infinite. Perfusion pressure is normal.

In experimental animals, CBF autoregulates to increased ICP produced by CSF infusion in much the same way that it autoregulates to decreased SAP produced by hypotensive agents or bleeding the animal. When autoregulation is intact and ICP is diffusely elevated, CBF does not fall until cerebral perfusion pressure (mean SAP—mean ICP in this instance) is in the 40 to 50 mm Hg range. When autoregulation is defective, CBF follows the perfusion pressure passively whether the latter is reduced by elevating ICP or decreasing SAP. In brain-injured patients, in contrast, there is not a predictable relationship between ICP and CBF whether autoregulation is intact or defective until ICP approaches or equals the blood pressure. The cause of this discrepancy between animal and clinical observations is not entirely clear. However, highly variable factors such as regional changes in pressure not reflected in measurements of ICP from only one location in the intracranial space, edema compressing the microcirculation, and paresis of the vasomotor tone of cerebral vessels secondary to the injury undoubtedly contribute. The important conclusion from these observations, one that has been well documented in clinical studies, is that the cause of increased ICP appears to be more important than the height of ICP in determining its relationship to CBF and the patient's neurological status. For example, some patients with severe head injuries exhibit neurological deterioration with rises in ICP from normal to no more than 20 to 25 mm Hg whereas other patients can tolerate pressure waves to 80 to 90 mm Hg without apparent ill effects.

Brain compression and increased ICP can cause arterial hypertension, bradycardia, and respiratory irregularities, the well-known Cushing triad. There is evidence that compression or other interference with the blood supply of a small region in the medulla is critical for the production of arterial hypertension. If intracranial hypertension is diffuse, that is, if there are no pressure gradients along the craniospinal axis, the pressor response does not occur until ICP equals or exceeds the diastolic blood pressure. In contrast, a mass lesion producing compression or distortion of the brain stem may produce a pressor response at relatively low levels of ICP. The increase in blood pressure is due to peripheral
vasoconstriction from increased sympathetic activity and to increased cardiac output.

The pulmonary complications of increased ICP are difficult to separate from those caused by a brain injury per se. The most common pulmonary complication in unconscious patients with increased ICP is alveolar obstruction. This results in ventilation perfusion abnormalities wherein non-ventilated portions of the lung continue to be perfused by venous blood. Venous admixture occurs in the pulmonary veins and left side of the heart, causing systemic hypoxemia. Increased ICP and brain compression can also cause acute pulmonary edema both clinically and in experimental animals. The mechanism is not well understood but could be due to pulmonary vein constriction or left heart failure. Acute pulmonary edema appears to be uncommon. Finally, venous admixture and systemic hypoxemia might be produced by neurogenically mediated opening of pulmonary arteriovenous (AV) shunts that bypass the alveoli.

Indications for Measuring ICP

Symposium participants agreed that continuous measurement of ICP is indicated in those patients with pathological conditions that are likely to produce brain swelling, obstruction of the CSF pathways, or a space-occupying mass or in patients who have signs and symptoms of intracranial hypertension. Thus, the decision to measure ICP is based primarily on clinical judgment. If the patient has clinical evidence of brain dysfunction and is not improving or deteriorating, and if ICP is elevated, usually it can be reduced with one or more methods of treatment to determine if the elevated pressure is the cause of the patient’s neurological condition. If the patient then improves, ICP often can be controlled continuously until the brain swelling subsides or the mass lesion is removed. Thus, continuous monitoring of ICP has proved to be useful in the management of patients with severe head injuries, spontaneous subarachnoid hemorrhage, and following craniotomy. It has been evaluated less extensively but appears to be promising in the treatment of patients with occlusive cerebral vascular disease, encephalitis and encephalopathies, hypoxic brain damage from various causes, pseudotumor cerebri with deteriorating vision, and obstructive hydrocephalus from posterior fossa tumors. In the latter circumstance, the ventricles are drained intermittently over a period of several days, and surgery is performed when ICP is normal.

The principal reason for continuously measuring ICP is to provide information that will permit better management of the individual patient. A secondary reason, but perhaps the most important one for the future, is collection of data on the incidence and clinical significance of increased ICP in patients with various types of intracranial pathology. There is abundant evidence that neither the physiological nor morphological pathology of most types of acute brain insult can be properly understood without further information on the alterations in intracranial dynamics that occur so commonly in these patients.

Reference


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