# Recent advances in cerebral monitoring

An essay submitted for fulfillment of master degree in anesthesiology

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# ABSTRACT



**Key words:** **cerebral monitoring * intracranial pressure * cerebral perfusion pressure * cerebral blood flow**



Cerebral monitoring is important for management of severe head injury and critically ill neurologic patient. It is also useful in subarachnoid hemorrhage, stroke, intracerebral hematoma, meningitis, encephalopathies, hepatic failure, after neurosurgery and in patients undergoing carotid artery surgery

The goals of cerebral monitoring are to detect changes in cerebral hemodynamics, cerebral oxygenation, intracranial pressure, cerebral metabolism and neuronal function. Advances in technology over the last few decades, have produced many new monitors of cerebral function, some of which are now available for clinical use.

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# ABBREVIATIONS

(ABI)……...……Acute brain injury

(AMP)……...……..... The amplitude of ICP waveform

(ARDS) ……... adult respiratory distress syndrome

($AvDO_2$)...…….…..……..…. Arterio-venous oxygen difference

(BBB) …….……Blood brain barrier

(BTF)…….. Brain Trauma Foundation

(CA).....…... cerebral autoregulation

($CaO_2$) …….…..... arterial oxygen content

(CFM)……..…….……. Cerebral function monitoring

(CBF) …….…….....Cerebral blood flow

(CBV) …….……. cerebral blood volume

(CMD)…….…...cerebral microdialysis

(CMR) …….Cerebral metabolic rate

($CMRO_2$)…..……...……... Cerebral metabolic rate of oxygen

(CPB) ……...…….……. Cardio-pulmonary bypass

(CPP) ……Cerebral perfusion pressure

(CSF) ……...…Cerebro Spinal fluid

(CT) …….…Computed tomography

(CVP) …….……Central venous pressure

(CVR) …….…….....Cerebral vascular resistance

(DCH) ……..…decompressive hemicraniectomy

(EEG) …….……Electroencephalography

(EP)…….…...Evoked potential

(EVD)……...……. External ventricular drain (Intraventricular)

(GCS) ……...Glasgow coma scale

(ICP)…….……Intracranial pressure

(ICA)…….…….....Internal carotid artery

(ICU)……Intensive care unit

($JvDO_2$)…….……...…….jugular venous saturation

(LDF)…… laser Doppler flowmetry

(LPR) ……..……lactate:pyruvate ratio

(MAP) …….……..…Mean arterial pressure

(MD) …….…….…… microdialysis

(MRI) …….Magnetic resonance imaging

(NICU)……...Neurointensive care unit

($PaCO_2$) …….Arterial partial pressure of carbon dioxide

($PaO_2$) ……...…….Arterial partial pressure of oxygen

($P_bO_2$)……...……Brain tissue oxygen tension

(PET)……..……...…….. Positron emission tomography

($PjvO_2$)……Jugular venous partial pressure of oxygen

(PRx) ……...…….……...…..pressure reactivity index

($PtiO_2$) ……..……..…brain tissue oxygen saturation

(QEEG)…….……quantitative EEG parameter

(RAP) ……..…..…….. Index of compensatory reserve

(RCTs )……..……...randomized controlled trials

(rCBV)…….……relative cerebral blood volume

(rCBF)…….…….... regional cerebral blood flow

(rCMRGlu)…….... relative metabolic glucose requirements

($rCMRO_2$) ……...……...….. relative metabolic $O_2$ requirements

($rSO_2$)…….…….regional oxygen saturation

(SAH)……..…….……. Subarachnoid Hemorrhage

($SaO_2$)…….……..…Arterial oxygen saturation

(SPECT)……..…single-photon emission computed tomography

($SjvO_2$) ……...…..….Jugular venous saturation of oxygen

(SSEPs)……..…….….Somatosensory evoked potentials

(TBI) ……Traumatic Brain Injury

(TCD) …….Transcranial Doppler

# CONTENTS

# Intracranial pressure monitoring

Intracranial pressure (ICP) measurement plays an important role in the management of patients with head injury and neurosurgical patients. Elevated ICP has been recognized as one of the most important factors affecting morbidity and mortality rates in patients who have had TBI; therefore, ICP monitoring has become routine in the management of severe head injuries. [1]

## Normal anatomy and physiology

The brain is a visco-elastic solid weighing about 1400 grams in air, and with the consistency and ability to withstand acceleration or deceleration forces, of a firm gelatinous mass. Sitting on a plate, it would tend to collapse under its own weight, but floating in CSF, it has an effective weight of 50 g; the brain is only 4% denser than water. In order for the brain to float, the CSF has to be constrained in a leak-proof box. [2]

The fundamental principles of raised ICP are condensed in the doctrine credited to Professors Monro (1783) and Kellie (1824), which states that (i) the brain is enclosed in a non-expandable case of bone; (ii) the brain parenchyma is nearly incompressible; (iii) the volume of the blood in the cranial cavity is therefore nearly constant; and (iv) a continuous outflow of venous blood from the cranial cavity is required to make room for continuous incoming arterial blood. [3]

It also states that CSF (100–150 ml), blood (100–150 ml), extracellular fluid (<75 ml) and brain (1200–1600 ml) are all essentially incompressible. Expansion in the volume of one component must lead to reduction in volume of another for the ICP to remain static. Unfortunately, although this doctrine is neat and tidy, the real life situation is more

complicated. The cranial cavity is not entirely enclosed, with the spinal cord, the thecal sac, and CSF passing through the foramen magnum. Although in adults the cranial cavity can be considered a rigid box of fixed volume, the lumbar thecal sac can be distended. This has a number of important implications. CSF can be squeezed out of the head to distend the lumbar thecal sac, indeed CSF flows out during systole, and flows back during diastole. Brain can also be squeezed through the foramen magnum (the basis of coning). [2]

Another consequence of the distensibility of the lumbar thecal sac is that ICP varies with body position. The normal mean ICP is 11 mmHg when supine. On standing upright, there is a hydrostatic pressure gradient from the top of the head to the lumbar region. About 3 ml of CSF flows out of the head, generating a slightly negative ICP (around −10 mmHg). In an adult, the ICP is maintained at subatmospheric pressure because of the construction of the venous system. On standing, the zero point of the venous system is a little above the right atrium. The internal jugular veins are collapsed, with no flow. Venous outflow from the brain occurs through non-collapsible veins, with walls that are held open. The dural venous sinuses anastamose with the internal vertebral venous plexus. These in turn anastamose with the intrathoracic veins. Thus, the sagittal sinus pressure is negative when upright. [4]

## ***Pathophysiology***

The intracranial compartment consists of brain approximately 83%, cerebrospinal fluid (CSF) approximately 11%, and blood approximately 6%. Under normal conditions, there are two main components of ICP, CSF and vasogenic. [5] The former is derived from the circulation of CSF and is responsible for baseline ICP. It may be deranged in pathologic

states, causing an increase in ICP, because of resistance to CSF flow between intracerebral compartments secondary to brain swelling or expansion of intracranial mass lesions, or because CSF outflow is obstructed. The vasogenic component of ICP is associated with continuous, small fluctuations of cerebral blood volume (CBV). Vasogenic increases in ICP may be caused by hypercapnia, increase in cerebral metabolism, and cerebral hyperemia. [6]

The relationship between ICP and intracranial volume is described by the non-linear pressure–volume curve (Fig. 1-1).

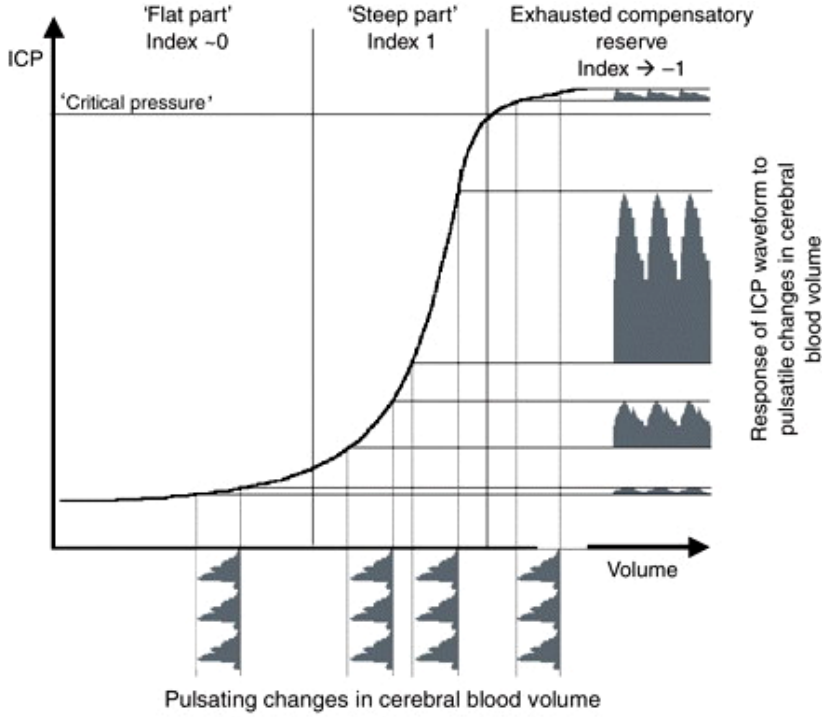


Fig. 1-1. Intracranial pressure–volume curve. Illustration of the intracranial pressure–volume curve and the relationship between pulsating changes in CBV and the ICP waveform. [7]

Classically, three parts of the intracranial pressure–volume curve are described: a flat part at lower intracerebral volumes, where good compensatory reserve is found, that is ICP remains low and stable despite changes in intracranial volume. This is attributable to compensatory mechanism, which is the reduction of the volume of cerebrospinal fluid (CSF) or intracranial blood. Once these mechanisms are exhausted, the

curve rapidly turns upwards, acquiring an exponentially rising shape. This part of the curve represents low compensatory reserve that is ICP increases considerably even with relatively small increases in intracerebral volume. Finally, at high levels of ICP, the curve plateaus again, denoting terminal disturbance in cerebrovascular responses, when cerebral perfusion pressure (CPP) is very low and ICP equals mean arterial pressure (MAP). The cerebral arterial bed cannot dilate any more and starts to collapse because of the further increase in brain tissue pressure. Therefore, the system gains some extra buffering capacity previously occupied by arterial blood volume. [8] This third part of the pressure–volume curve has been confirmed experimentally, [9] and observed clinically, although only indirectly. [10]

After TBI, increased ICP can be related to intracranial mass lesions, contusion injuries, vascular engorgement, and brain edema. Recent clinical studies have shown that brain edema, and not increased CBV as a result of vascular engorgement, is the major culprit responsible for brain swelling after TBI. [11] Vasogenic brain edema, emanating from the blood vessels subsequent to blood-brain barrier compromise, has classically been considered the prevalent edema after TBI, [12] but recent magnetic resonance imaging studies have indicated that, in patients with significant brain swelling, cytotoxic or cellular edema, occurring secondary to sustained intracellular water collection, predominates. [13] Cytotoxic edema is of decisive pathophysiologic importance, as it develops early and persists while blood-brain barrier integrity is gradually restored. These findings have implications for the treatment of TBI and suggest that cytotoxic and vasogenic brain edema are two entities that can be targeted simultaneously or independently, according to their temporal prevalence. [12] When cerebral autoregulation is absent, an increase in arterial blood pressure (ABP) causes an increase in CBV and hence in ICP. [5] An

increase in CBV and ICP may also occur in response to changes in other systemic variables, such as arterial $PaCO_2$, temperature and intrathoracic or intraabdominal pressures, or because of intracranial events such as seizures. Intracranial hypertension may also occur because of acute or chronic disturbances of CSF drainage (hydrocephalus) and other, often diffuse, pathological processes, such as cerebral edema secondary to hepatic failure. [6]

## ***Normal and Pathologic ICP***

Normal ICP varies with age, body position, and clinical condition. [14] The normal ICP is 7-15 mmHg in a supine adult, 3-7 mmHg in children, and 1.5-6 mmHg in term infants. The definition of intracranial hypertension depends on the specific pathology and age, although ICP > 15 mmHg is generally considered abnormal. However, treatment is instituted at different levels depending on the pathology. For example, ICP > 15 mmHg warrants treatment in a patient with hydrocephalus, [15] whereas after TBI, treatment is indicated when ICP exceeds 20 mmHg. [16] Thresholds vary in children and it has been recommended that treatment should be initiated during TBI management when ICP exceeds 15 mmHg in infants, 18 mmHg in children < 8 years-of-age and 20 mmHg in older children and teenagers. [17]

ICP is not equally distributed in pathologic states because CSF does not circulate freely and intracranial CSF volume may be low because of brain swelling. The assumption of one, uniform, ICP is therefore questionable and intraparenchymal pressure may not be indicative of "real" ICP, i.e., ventricular CSF pressure. [18] In the injured brain, there may be intraparenchymal pressure gradients between the supra and infra-tentorial compartments. Bilateral monitoring has revealed differential

pressures across the midline in the presence of hematomas, and in the absence of space-occupying lesions. [19]

## Methods of ICP Monitoring

ICP cannot be reliably estimated from any specific clinical feature or computed tomography (CT) finding and must actually be measured. Different methods of monitoring ICP have been described (fig. 1-2) but two methods are commonly used in clinical practice: intraventricular catheters and intraparenchymal catheter-tip, micro transducer systems. Subarachnoid and epidural devices have much lower accuracy and are now rarely used. [20]

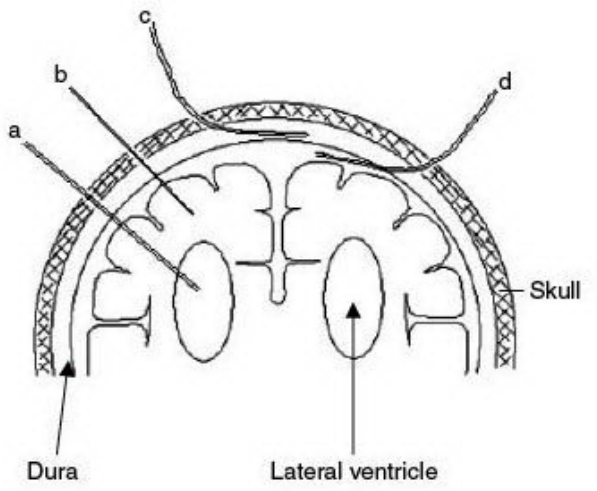


Fig. 1-2. Possible sites of ICP Monitoring: a) intraventricular drain; b) intraparenchymal probe; c) epidural probe; d) Subarachnoid probe. [8]

Measurement of lumbar CSF pressure does not provide a reliable estimate of ICP and may be dangerous in the presence of increased intracranial hypertension. [8]

## Placement

### 1. Ventriculostomy

'Gold standard' of ICP measurement (Fig. 1-3). Requires placement of a catheter into the lateral ventricle, the reference point for the external transducer is the foramen of Monroe (surface marking 2 cm above the

pterion or midpoint of a line joining the two external auditory meati). It is the simplest, most reliable and accurate system available and has the advantage of allowing drainage of CSF to lower ICP. [21]

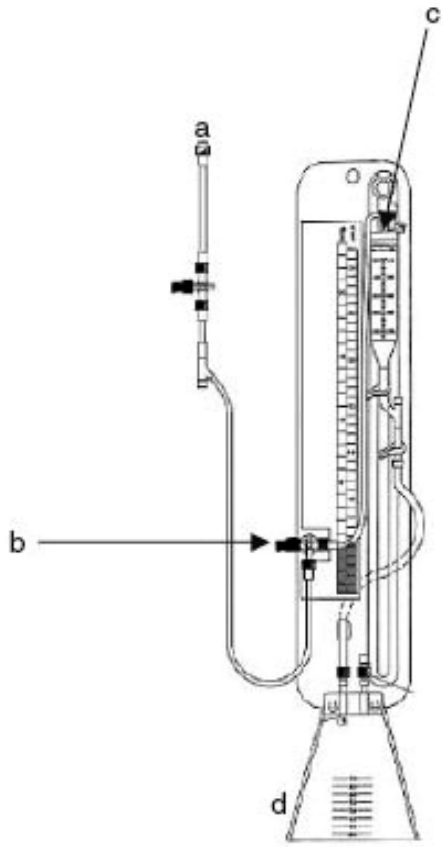


Fig. 1-3. Typical system for ICP measurement via an intraventricular drain: a: connection with drain; b: zero (should be positioned at height of patient's ear) and three-way stopcock for connection with pressure transducer; c: drip chamber, adjustable in height over zero for CSF drainage. Depending on stopcock position (b) either pressure measurements or CSF drainage are possible; and d: CSF reservoir. [8]

Disadvantages include high bacterial colonization rates defined as positive CSF or catheter tip culture (1–5% after 3 days, though ventriculitis has not been demonstrated in clinical studies), difficult placement, injury to brain tissue and potential for leaks/blocks in the system. [21]

## ***2. Parenchymal catheter***

A catheter is inserted into the brain parenchyma. It is used for monitoring ICP when ventricular access cannot be obtained or CSF