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Intracranial pressure monitoring in critically ill patients

An Essay
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Abbrev	*Full term*
ABP	Arterial blood pressure
AEP	Auditory evoked potentials
ATN	Acute tubular necrosis
ATP	Adenosine triphosphate
(A-V) DO2	Arterio-venous oxygen saturation
BAER	Brain stem auditory evoked responses
BP	Blood pressure
CaBV	Arterial blood volume curve
CAMP	Compound muscle action potentials
CAP	Carotid arterial pressure
CBF	Cerebral blood flow
CBFV	Cerebral blood flow velocity
CBV	Cerebral blood volume
CEO2	Cerebral oxygen extraction
CMRO2	Cerebral metabolic rate of O2
CN	Cranial nerve
CNS	Central nervous system
CPP	Cerebral perfusion pressure
CSA	Compressed spectral array
CSF	Cerebrospinal fluid
CT	Computed tomography
CVR	Cerebrovascular resistance
DSA	Density modulated spectral array
DTI	Diffusion tensor imaging
EAAs	Excitatory amino acids
EEG	Electroencephalogram
EMG	Electromyogram
EVD	External ventricular drain
FV	Flow velocity
GCS	Glascow coma score
ICH	Intracranial hypertension

ICP	Intracranial pressure
ICU	Intensive care unit
INR	International normalized ratio
IV	Intravenous
JVP	Jugular venous pressure
LOC	Loss of consciousness
LP	Lumbar puncture
MAP	Mean arterial pressure
MCA	Middle cerebral artery
MRI	Magnetic resonance imaging
NaCl	Sodium chloride
PaCO2	Partial pressure of carbon dioxide in arterial blood
PaO2	Partial pressure of oxygen in arterial blood
PbtO2	Perfusion and brain tissue oxygen tension
PEEP	Peak end expiratory pressure
PI	Pulsatality index
REM sleep	Rapid eye movement sleep
SaO2	Arterial oxygen saturation
SBP	Systolic blood pressure
SjVO2	Jugular venous oxygen saturation
SSEP	Somatosensory evoked potentials
TBI	Traumatic brain injury
TCD	Transcranial Doppler
tcEMEP	Transcranial electric motor evoked potentials
tcMMEP	Transcranial magnetic motor evoked potentials
VEP	Visual evoked potentials
VICA	Internal carotid artery flow velocity
VMCA	Middle cerebral artery flow velocity
VRACs	Volume regulated anion channels

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Introduction

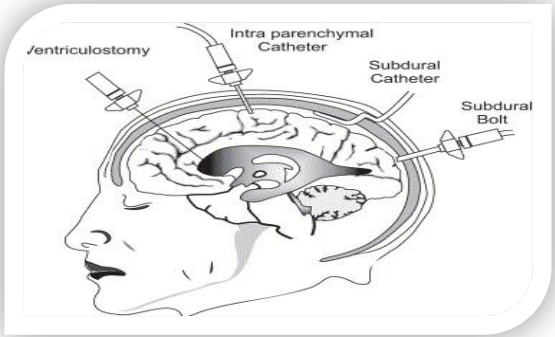
Elevated intracranial pressure (ICP) is seen in head trauma, hydrocephalus, intracranial tumors, hepatic encephalopathy, and cerebral edema. Intractable elevated ICP can lead to death or devastating neurological damage either by reducing cerebral perfusion pressure (CPP) and causing cerebral ischemia or by compressing and causing herniation of the brainstem or other vital structures. Prompt recognition is crucial in order to intervene appropriately ***(Bingaman& Frank, 2011)***.

The rapid recognition of elevated ICP is therefore of obvious and paramount importance so that it can be monitored and so that therapies directed at lowering ICP can be initiated. A raised ICP is measurable both clinically and quantitatively. Continuous ICP monitoring is important both for assessing the efficacy of therapeutic measures and for evaluating the evolution of brain injury ***(Lane et al., 2014)***.

There are four main anatomical sites used in the clinical measurement of ICP: intraventricular, intraparenchymal, subarachnoid, and epidural. Noninvasive and metabolic monitoring of ICP has also been studied, but the clinical value of these methods is unclear at present. Each technique requires a unique monitoring system, and has associated advantages and disadvantages***(Brain Trauma Foundation., 2007)***.

Aim of the work

The aim of this work is to highlight the importance, methods, indications and techniques of intracranial pressure monitoring in critically ill patients.



Neurological assessment of critically ill patients

Neurological assessment of critically ill patients

Neurologicalassessmentofcriticallyillpatients

Elevated intracranial pressure (ICP) is a potentially devastating complication of neurologic injury. Elevated ICP may complicate trauma, central nervous system (CNS) tumors, hydrocephalus, hepatic encephalopathy, and impaired CNS venous outflow (table 1) Successful management of patients with elevated ICP requires prompt recognition, the judicious use of invasive monitoring, and therapy directed at both reducing ICP and reversing its underlying cause***(Adams and Ropper,2010)***.

Table (1): Causes of intracranial hypertension***(Adams and Ropper, 2010)***

Traumatic brain injury/Intracranial hemorrhage -Subdural, epidural, or intraparenchymal hemorrhage -Ruptured aneurysm -Diffuse axonal injury -Arteriovenous malformation or other vascular anomalies
Central nervous system infections (eg, encephalitis, meningitis, abscess)
Ischemic stroke
Neoplasm
Vasculitis
Hydrocephalus
Idiopathic intracranial hypertension (pseudotumorcerebri)
Idiopathic

Intracranial pressure is normally ≤15 mmHg in adults, and pathologic intracranial hypertension (ICH) is present at pressures ≥20 mmHg. ICP is normally lower in children than adults, and may be subatmospheric in newborns. Homeostatic mechanisms stabilize ICP, with occasional transient elevations associated with physiologic events, including sneezing, coughing, or Valsalva maneuvers ***(Welch, 2009)***.

Intracranial components

In adults, the intracranial compartment is protected by the skull, a rigid structure with a fixed internal volume of 1400 to 1700 ml. Under physiologic conditions, the intracranial contents include (by volume):

- Brain parenchyma — 80 percent
- Cerebrospinal fluid — 10 percent
- Blood — 10 percent***(Monro,2008)***

Pathologic structures, including mass lesions, abscesses, and hematomas also may be present within the intracranial compartment. Since the overall volume of the cranial vault cannot change, an increase in the volume of one component, or the presence of pathologic components, necessitates the displacement of other structures, an increase in ICP, or both. Thus, ICP is a function of the volume and compliance of each component of the intracranial compartment, an interrelationship known as the Monro-Kellie doctrine***(Monro, 2008)***.

The volume of brain parenchyma is relatively constant in adults, although it can be altered by mass lesions or in the setting of cerebral edema (figure 1). The volumes of cerebrospinal fluid (CSF) and blood in the intracranial space vary to a greater degree. Abnormal increases in the volume of any component may lead to elevations in ICP ***(Fishman, 2005)***.

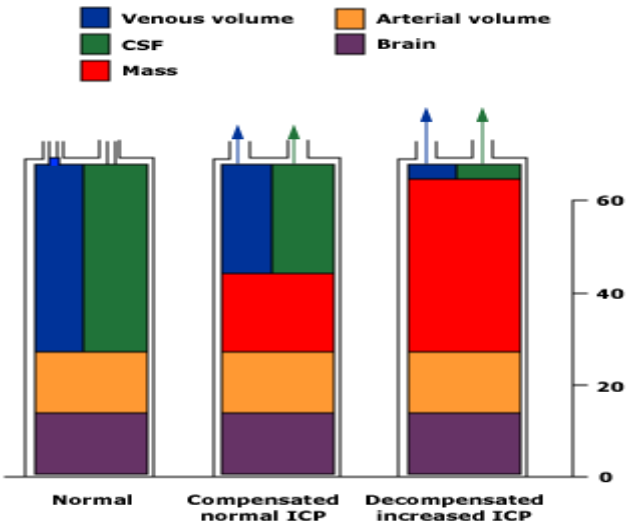


Figure (1): Intracranial compensation for an expanding mass lesion

(Fishman, 2005)

CSF is produced by the choroid plexus and elsewhere in the central nervous system (CNS) at a rate of approximately 20 mL/h (500 mL/day). CSF is normally resorbed via the arachnoid granulations into the venous system. Problems with CSF regulation generally result from impaired outflow caused by ventricular obstruction or venous congestion; the latter can occur in patients with sagittal (or other) venous sinus thrombosis. Much less frequently, CSF production can become pathologically increased; this may be seen in the setting of choroid plexus papilloma ***(Fishman, 2005)***.

Intracranial compliance

The interrelationship between changes in the volume of intracranial contents and changes in ICP defines the compliance characteristics of the intracranial compartment. Intracranial compliance can be modeled mathematically (as in other physiologic and mechanical systems) as the change in volume over the change in pressure (dV/dP) ***(Wilkins, 2006)***.

The compliance relationship is nonlinear, and compliance decreases as the combined volume of the intracranial contents increases. Initially,

compensatory mechanisms allow volume to increase with minimal elevation in ICP. These mechanisms include:

- Displacement of CSF into the thecal sac
- Decrease in the volume of the cerebral venous blood via venoconstriction and extracranial drainage***(Wilkins, 2006)***.

However, when these compensatory mechanisms have been exhausted, significant increases in pressure develop with small increases in volume, leading to abnormally elevated ICP (figure 2) ***(Wilkins, 2006)***.

Thus, the magnitude of the change in volume of an individual structure determines its effect on ICP. In addition, the rate of change in the volume of the intracranial contents influences ICP. Changes that occur slowly produce less of an effect than those that are rapid. This can be recognized clinically in some patients who present with large meningiomas and minimally elevated or normal ICP. Conversely, other patients may experience symptomatic elevations in ICP from small hematomas that develop acutely***(Wilkins, 2006)***.

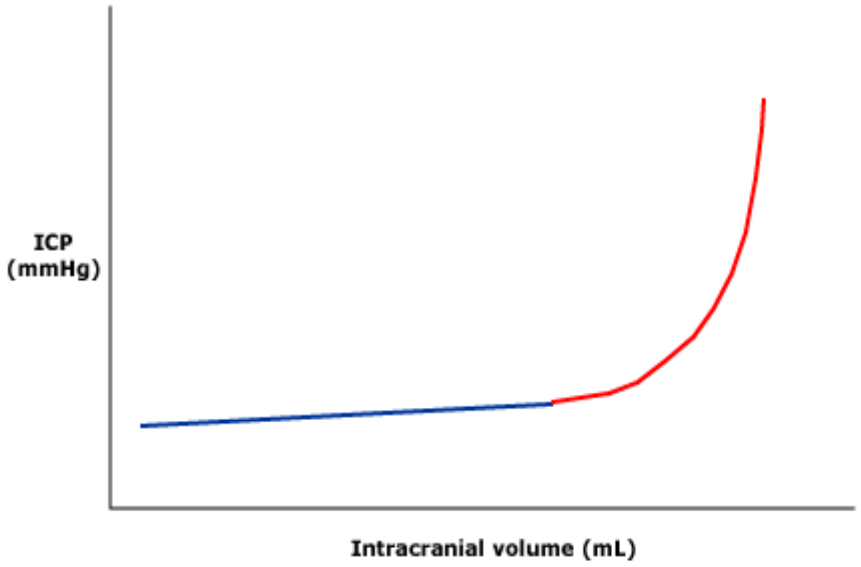


Figure (2): The relationship between intracranial volume and pressure is nonlinear.***(Wilkins, 2006)***.

Cerebral blood flow

Following a significant increase in ICP, brain injury can result from brainstem compression and/or a reduction in cerebral blood flow (CBF). CBF is a function of the pressure drop across the cerebral circulation divided by the cerebrovascular resistance, as predicted by Ohm's law:

CBF = (CAP - JVP) ÷ CVR

Where CAP is carotid arterial pressure, JVP is jugular venous pressure, and CVR is cerebrovascular resistance.

Cerebral perfusion pressure (CPP) is a clinical surrogate for the adequacy of cerebral perfusion. CPP is defined as mean arterial pressure (MAP) minus ICP.

CPP = MAP – ICP***(Wilkins, 2006)***