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Intracranial Pressure Monitoring Modalities

Ahmed Mohamed Abd El Rahim, Samia Abd-El Rahman El-Wakeel, Heba Mohamed Fathi and Khaled Mahrous Abd-El Hameed

Anesthesia, intensive care and pain management Department, Faculty of Medicine, Zagazig University, Egypt

***Corresponding author:** Ahmed Mohamed Abd El Rahim

Email: Elboraya21@gmail.com,

Abstract:

Intracranial pressure (ICP) monitoring is a fundamental component of neurocritical care, particularly in patients with severe traumatic brain injury, in whom intracranial hypertension can reduce cerebral perfusion, worsen secondary brain injury, and increase the risk of neurological deterioration and death. Clinical manifestations of raised ICP, such as pupillary abnormalities and abnormal motor posturing, may appear late; therefore, objective monitoring is essential for early detection and treatment guidance. Invasive techniques, including external ventricular drains and intraparenchymal monitors, provide direct and continuous ICP measurements. External ventricular drainage is regarded as the reference standard because it permits both pressure monitoring and therapeutic cerebrospinal fluid drainage, whereas intraparenchymal devices are easier to insert but cannot be re-zeroed after placement and may develop measurement drift. also invasive techniques are associated with complications such as infection, hemorrhage, obstruction, malposition, and mechanical failure. These limitations have encouraged the use of non-invasive methods, particularly transcranial Doppler ultrasonography, which evaluates cerebral blood-flow velocities and pulsatility index as indirect indicators of downstream cerebrovascular resistance and ICP. Other non-invasive modalities include optic nerve sheath diameter ultrasonography, neuroimaging, and tympanic membrane displacement. This review summarizes the principles, advantages, limitations, complications, and clinical applications of invasive and non-invasive ICP monitoring modalities, with particular emphasis on transcranial Doppler and pulsatility index interpretation.

Keywords: Intracranial pressure; Intracranial pressure monitoring; Traumatic brain injury; External ventricular drain; Intraparenchymal monitor; Transcranial Doppler; Pulsatility index; Cerebral perfusion pressure.

Introduction:

The accurate measurement of Intracranial Pressure (ICP) is the cornerstone of modern neurocritical care for patients with severe Traumatic Brain Injury (TBI). While the clinical examination provides valuable clues—such as pupillary dilation or changes in motor posturing—these signs often appear only after herniation is imminent. Therefore, objective, continuous monitoring is essential to guide therapy, optimize Cerebral Perfusion Pressure (CPP), and prevent secondary brain injury. This chapter reviews the current "gold standard" invasive techniques and explores the growing role of non-invasive surrogates, particularly Transcranial Doppler (TCD) ultrasonography **(1)**.

Invasive monitoring remains the definitive method for calculating ICP. The Brain Trauma Foundation (BTF) guidelines generally recommend monitoring in patients with severe TBI (Glasgow Coma Scale score of 3–8) who have an abnormal CT scan, or in patients with a normal CT scan if specific risk factors (age over 40, hypotension, or posturing) are present **(2)**.

2.1.1 External Ventricular Drains (EVD) and Parenchymal Monitors

There are two primary devices used for invasive monitoring, each with distinct advantages and mechanics **(3)**:

1. External Ventricular Drain (EVD): Often referred to as a ventriculostomy, the EVD is considered the absolute reference standard against which all other devices are calibrated. It involves inserting a catheter through the skull (typically via a burr hole at Kocher's point) into the lateral ventricle of the brain **(4)**.

- **Mechanism:** The catheter is connected to an external fluid-filled transducer system leveled at the tragus of the ear (approximating the Foramen of Monro). It measures pressure via fluid column transmission **(5)**.
- **Therapeutic Advantage:** The unique benefit of an EVD is its dual capability. It serves as both a diagnostic tool (measuring pressure) and a therapeutic tool (draining Cerebrospinal Fluid). When ICP spikes, clinicians can open the drain to divert CSF, immediately reducing intracranial volume and pressure **(3)**.

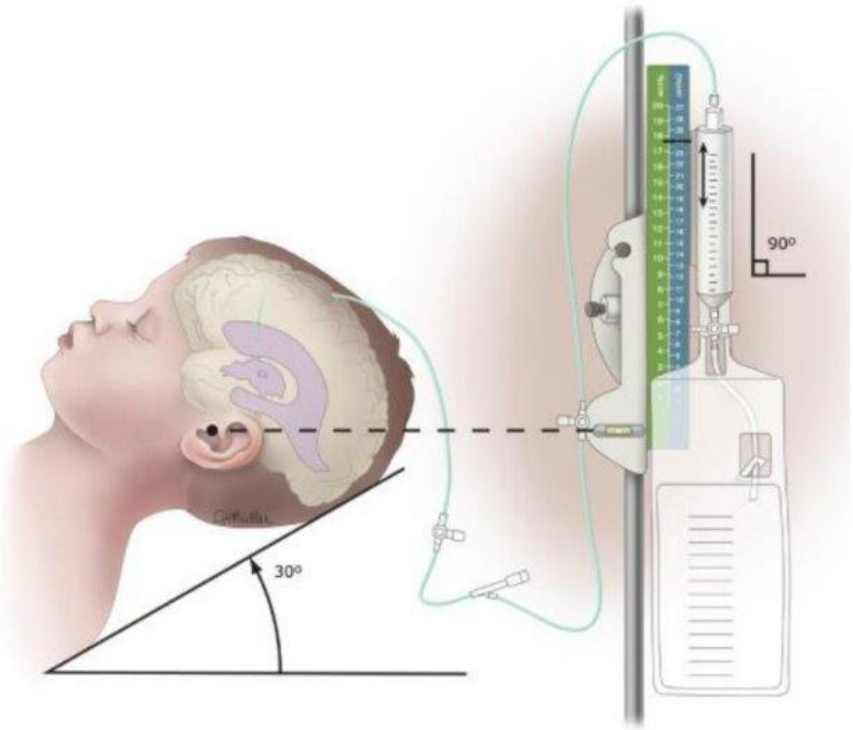

Figure 1. External ventricular drain (EVD) **(4)**.

2. Intraparenchymal Monitors: These are fiber-optic or strain-gauge devices inserted directly into the brain tissue (parenchyma), usually through a cranial bolt **(4)**.

- **Mechanism:** Most commonly, these use a micro-transducer at the tip of the catheter to measure pressure locally **(4)**.
- **Utility:** They are easier to insert than EVDs, especially when the ventricles are compressed or shifted due to brain swelling (slit ventricles). However, unlike EVDs, they cannot drain CSF for therapeutic purposes **(3)**.

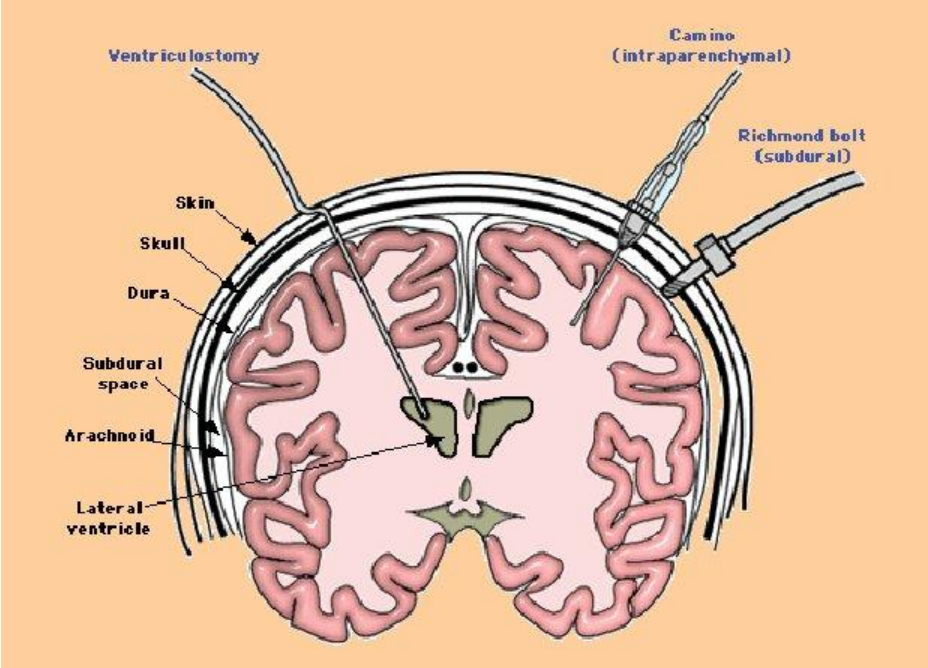

Figure 2. Intracranial pressure monitors ventriculostomy allows both ICP monitoring and therapeutic drainage of cerebrospinal fluid (CSF) **(6)**.

2.1.2 Accuracy, Calibration, and Interpretation of Waveforms

Accuracy and Calibration:

- **EVDs:** Because they use an external transducer, EVDs can be re-calibrated (zeroed) to atmospheric pressure at the bedside at any time. However, they are prone to artifacts if the tubing becomes kinked or blocked by debris/blood clots **(4)**.
- **Parenchymal Monitors:** These devices are zeroed once prior to insertion (in air). Once inserted, they cannot be re-zeroed. This leads to "drift"—a gradual loss of accuracy over days. While modern devices have minimal drift (often less than 1-2 mmHg over 5 days), this remains a limitation for prolonged monitoring **(3)**.

Waveform Interpretation: The ICP waveform is a pulsatile wave that corresponds to the cardiac cycle. Analyzing its shape provides insight into intracranial compliance (stiffness). A normal waveform consists of three distinct peaks **(5)**:

1. **P1 (Percussion Wave):** Related to the arterial pulsation transmitted through the choroid plexus. It is usually the tallest peak.
2. **P2 (Tidal Wave):** Related to brain tissue compliance. In a healthy brain, P2 is lower than P1.
3. **P3 (Dicrotic Wave):** Related to the closure of the aortic valve.

Clinical Significance of P2: As intracranial compliance decreases (the brain becomes stiffer/tighter), the P2 wave rises. When P2 becomes taller than P1, it indicates significantly reduced compliance and a brain at high risk of herniation, even if the absolute ICP value is essentially normal **(4)**.

2.1.3 Complications: Infection, Hemorrhage, and Availability

Despite their utility, invasive procedures carry inherent risks:

- **Infection (Ventriculitis/Meningitis):** This is the most significant complication, particularly for EVDs. The risk increases with the duration of monitoring (often rising significantly after 5–7 days), frequent

sampling, or leakage. Rates of catheter-related infection range widely but can be as high as 10–20% in some series without antibiotic-impregnated catheters **(7)**.

- **Hemorrhage:** Passing a catheter through the brain can injure blood vessels. While major hemorrhage is rare (<1%), minor tract hemorrhages are visible on CT in a larger percentage of patients. Coagulopathy is a relative contraindication **(3)**.
- **Malposition and Obstruction:** EVDs can be misplaced into the brain parenchyma instead of the ventricle, or the catheter can become occluded by blood or tissue debris, leading to inaccurate readings or failure to drain **(8)**.

2.2 Non-Invasive Assessment: Transcranial Doppler (TCD) Ultrasonography

Given the risks of invasive monitoring, there is immense interest in non-invasive alternatives. Transcranial Doppler (TCD) is a portable, bedside ultrasound technique that measures the velocity of blood flow through the major cerebral arteries. While it does not measure pressure directly, it assesses cerebral hemodynamics, which are heavily influenced by ICP **(3)**.

2.2.1 Relevant Anatomy: The Circle of Willis

To interpret TCD findings accurately, an understanding of the Circle of Willis is essential. This structure is a circulatory anastomosis that supplies blood to the brain and surrounding structures. It is a hexagonal arrangement of arteries located at the base of the brain, encircling the optic chiasm and infundibulum of the pituitary stalk.

The circle is formed by the anastomosis of the two internal carotid arteries and the two vertebral arteries. Key components accessible to TCD assessment include:

- **Anterior Circulation:** Comprising the Anterior Cerebral Arteries (ACA) and the Middle Cerebral Arteries (MCA). The MCA is the largest branch of the internal carotid and is the most frequently insonated vessel in TCD studies due to its accessibility and favorable angle of insonation.
- **Posterior Circulation:** Comprising the Posterior Cerebral Arteries (PCA), fed by the basilar artery.
- **Communicating Arteries:** The Anterior and Posterior Communicating Arteries link the systems, providing collateral flow in cases of occlusion or stenosis.

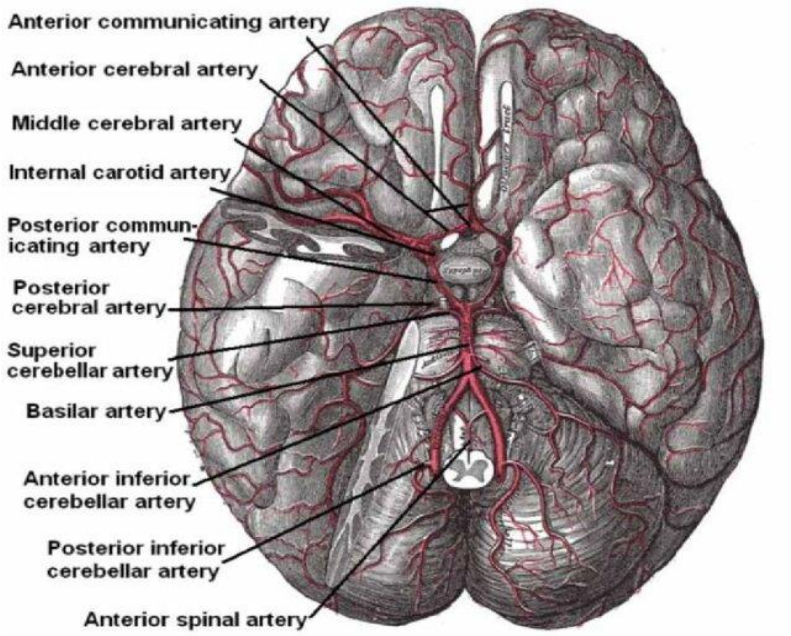

Figure 3. Anatomy of the circle of Willis at the base of the brain **(9)**.

2.2.2 Physical Principles and Insonation Windows (Transtemporal)

TCD utilizes the Doppler effect: sound waves reflected off moving red blood cells undergo a frequency shift proportional to the velocity of the blood **(5)**.

- **Frequency:** Because the skull bones are thick and absorb ultrasound, TCD requires a low-frequency probe (typically 2 MHz) to penetrate the bone **(5)**.
- **Insonation Windows:** The skull has natural "windows" where the bone is thinner or naturally open. The most critical window for ICP assessment is the Transtemporal Window, located on the temple above the zygomatic arch. Through this window, the Middle Cerebral Artery (MCA) can be insonated. The MCA is the preferred vessel for monitoring because it carries a large portion of cerebral blood flow and is directly accessible **(4)**.

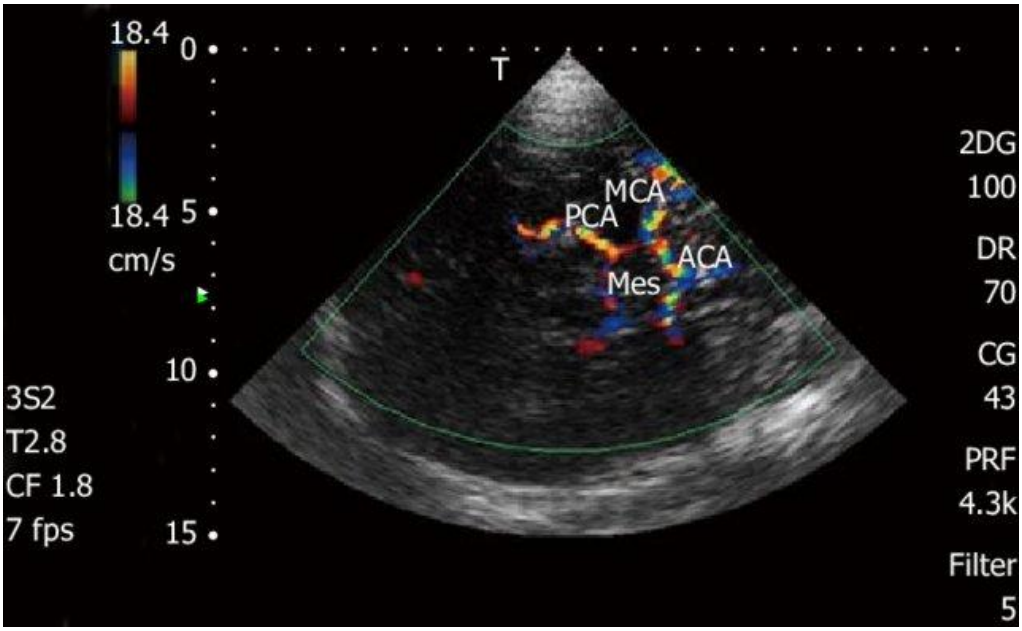

Figure 4. Transcranial Doppler color: Doppler study of intracranial arteries **(10)**.

2.2.3 Ultrasonographic Parameters: Peak Systolic and End-Diastolic Velocities

TCD generates a spectral waveform representing blood flow velocity over time. Two key velocity points are extracted from this waveform **(5)**:

1. **Peak Systolic Velocity (PSV):** The maximum speed of blood flow during heart contraction (systole).
2. **End-Diastolic Velocity (EDV):** The speed of blood flow at the end of heart relaxation (diastole).

Impact of ICP on Velocity: As ICP rises, it compresses the distal capillary beds. This resistance opposes blood flow. The effect is most pronounced during diastole when the driving pressure from the heart is lowest. Therefore, as ICP increases, EDV drops significantly, while PSV remains relatively stable or decreases only slightly. In severe cases of intracranial hypertension, diastolic flow may cease entirely or even reverse (oscillating flow), a sign of cerebral circulatory arrest **(4)**.

2.2.4 Other Methods of Non-Invasive Assessment

While TCD is a primary tool for hemodynamic assessment, other non-invasive modalities are utilized to estimate ICP, often used in conjunction with TCD to improve diagnostic accuracy.

- **Optic Nerve Sheath Diameter (ONSD) Ultrasonography:** The optic nerve is surrounded by the subarachnoid space and is distinctively continuous with the dura mater of the brain. Consequently,

increases in ICP are transmitted to the subarachnoid space surrounding the optic nerve, causing expansion of the nerve sheath. ONSD is measured using a high-frequency ultrasound probe placed over the closed eyelid. A diameter exceeding 5.0–6.0 mm (depending on the specific protocol) is widely considered indicative of raised ICP **(11)**.

- **Neuroimaging (CT/MRI):** While not continuous bedside monitors, static imaging provides critical non-invasive signs of elevated ICP. These include the effacement of basal cisterns, midline shift, sulcal effacement, and compression of the ventricles. MRI can also detect more subtle signs such as flattening of the posterior globe or empty sella syndrome (a condition where chronically elevated cerebrospinal fluid pressure forces the subarachnoid space into the sella turcica, flattening the pituitary gland against its bony walls and making the cavity appear empty on neuroimaging) **(12)**.
- **Tympanic Membrane Displacement (TMD):** This technique utilizes the perilymphatic duct, which connects the subarachnoid space to the inner ear. Changes in ICP can displace the tympanic membrane, which can be measured using specific audiometric devices, though this method is less common in acute clinical settings compared to TCD and ONSD **(13)**.

2.3 The Pulsatility Index (PI)

The relationship between systolic and diastolic velocities is quantified using the Pulsatility Index (PI). This derived parameter is the primary TCD metric used to estimate downstream cerebrovascular resistance which is indirectly related to ICP **(14)**.

2.3.1 Definition and Calculation (Gosling's Formula)

The Pulsatility Index was originally described by Gosling to describe the pulsatility of arterial waveforms. It is calculated using the following formula **(15)**:

PI = (Peak Systolic Velocity - End Diastolic Velocity) / Mean Flow Velocity

(Where Mean Flow Velocity is the time-averaged velocity over the cardiac cycle).

Normal PI values typically range from **0.6 to 1.1**. A PI value **> 1.2** is generally considered abnormal and indicative of increased resistance.

MFV in the denominator is not a simple arithmetic mean of the peak and end velocities; rather, it represents the time-averaged velocity over the entire cardiac cycle. In clinical practice, it is often estimated using the following approximation **(15)**:

Mean Flow Velocity = (Peak Systolic Velocity + (2 x End Diastolic Velocity)) / 3

Interpretation of PI Values Because the Peak Systolic Velocity (PSV) is primarily driven by cardiac contraction, it remains relatively stable. Therefore, changes in the PI are almost entirely dictated by fluctuations in the End Diastolic Velocity (EDV), which is highly sensitive to downstream resistance **(15)**:

- **Normal Range (0.6 – 1.1):** Indicates standard cerebral vascular resistance with normal intracranial compliance.
- **Low Pulsatility (< 0.6):** Suggests low distal resistance. This may be seen in conditions such as arteriovenous malformations, significant vasospasm (where high velocity occurs with low resistance), or cerebral hyperemia.
- **Elevated Pulsatility (> 1.2):** Generally considered abnormal and indicative of **increased distal resistance**. This elevation typically correlates with raised Intracranial Pressure (ICP), microvascular disease, or hypocapnia (hyperventilation), where the vascular bed constricts, reducing diastolic flow relative to systolic flow.

2.3.2 Correlation between Pulsatility Index and Invasive ICP

The correlation between PI and invasive ICP is based on the mechanical principle that external compression of the vascular bed (by high ICP) increases the pulsatility of blood flow **(16)**.

- **Linear Correlation:** Numerous studies have demonstrated a positive linear correlation between PI and ICP. As ICP rises, the gap between systolic and diastolic velocity widens (because diastole drops faster), causing the PI to increase **(16)**.
- **Estimating ICP:** Several formulas exist to estimate ICP from PI. One common simplified equation is:

  ICP = (10.93 x PI) - 1.28.
- **Limitations:** While the correlation is strong in groups of patients, the individual accuracy can vary. PI is best used as a trend monitor—a sudden rise in PI warrants immediate investigation or confirmatory invasive monitoring **(14)**.

2.3.3 Factors Affecting PI Accuracy (Vascular Resistance, Heart Rate, PaCO2)

While PI is a useful surrogate, it is not a direct measure of pressure. It reflects **Cerebrovascular Resistance (CVR)**, which is influenced by **(3)**:

1. **PaCO2:** carbon dioxide levels potently change vessel diameter. Hypocapnia (low CO2) causes vasoconstriction, which increases resistance and falsely elevates PI, even if ICP is normal.
2. **Systemic Blood Pressure:** Extreme hypotension or hypertension can alter the waveform shape, particularly if autoregulation is impaired.
3. **Heart Rate:** Bradycardia allows for a longer diastole, which can lower EDV and artificially increase PI.
4. **Vessel Spasm:** If the MCA itself is in spasm (common after trauma), the high velocities are due to narrowing of the vessel, not downstream resistance. This complicates the interpretation of PI for ICP estimation.

Table 1. Comparison of ICP Monitoring Modalities

Feature	External Ventricular Drain (EVD)	Intraparenchymal Fiber-optic Monitor	Transcranial Doppler (TCD)
Invasiveness	High (penetrates ventricle)	Moderate (penetrates parenchyma)	Non-Invasive (Ultrasound)
Gold Standard	Yes	No (but widely accepted)	No (Surrogate marker)
Therapeutic Capability	Yes (CSF Drainage)	No	No
Re-calibration	Possible (can be re-zeroed)	Not possible (drift occurs)	N/A
Primary Metric	Direct Pressure (mmHg)	Direct Pressure (mmHg)	Pulsatility Index (PI)
Key Risks	Infection, Hemorrhage, Obstruction	Hemorrhage, Drift, Mechanical failure	Operator dependent, No temporal window
Cost	Low (device) / High (hospital stay)	High (device cost)	Low (reusable equipment)

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