

Clinical review of non-invasive intracranial pressure measurement in medical cases

Shalini Nair

Abstract

Intracranial pressure (ICP) measurement plays a vital role in decision making in neurological emergencies. Invasive methods of ICP monitoring have been the gold standard. Advent of various non-invasive techniques has widened the option of ICP measurement in medical cases as well. We illustrate two cases where optic nerve sheath measurements helped in managing raised ICP and time the need for surgical intervention and review the literature on non-invasive ICP measurement.

Key words: Non-invasive intracranial pressure measurement, optic nerve sheath diameter, transcranial Doppler and pulsatility index

INTRODUCTION

Does measuring intracranial pressure (ICP) make any difference to the outcome of a patient? The question could not be answered for a long time in the absence of ethically feasible clinical trials. BEST TRIP trial^[1] was the first randomised controlled trial to answer this very question. Infact, the methodology compared invasive ICP measurement based protocol to a computed tomography (CT) based non-invasive ICP measurement and concluded that ICP based management was not better in comparison to the latter. Traditionally, ICP has been measured invasively and needs technical expertise for insertion and monitoring. Hence, its use has been limited to patients treated in neuro Intensive Care Units (ICU). The medical cohorts with intracranial hypertension as in stroke, cortical venous thrombosis,

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hepatic coma, etc., very often are not monitored to detect an early rise in ICP. In these groups use of non-invasive techniques to detect rising ICP would be extremely useful to prevent intracranial catastrophes. We discuss two such cases managed with non-invasive ICP monitoring that helped us avert intracranial herniation and present a clinical review of the available methods to monitor ICP non-invasively.

CLINICAL ILLUSTRATIONS

In the first case a 25-year-old lady with history suggestive of encephalitis presented to medical and surgical ICU (MSICU). She was extremely agitated with tachycardia (heart rate [HR] - 130/min) and hypertension (blood pressure [BP] - 180/100 mm Hg). Her CT scan was suggestive of generalised cerebral swelling [Figure 1]. After controlling her agitation, HR dropped to 75/min and BP was 170/90 mm Hg. A lumbar puncture was deferred in view of raised ICP. Six hours later, HR dropped to 45/min and anisocoria was noted. She was shifted to neuro ICU for better monitoring and anticipated surgical intervention for refractory raised ICP.

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An optic nerve sheath diameter (ONSD) measurement showed raised value of 5.8 mm [Figure 2]. Osmotherapy with 20% mannitol was initiated, and 3% saline was started to target serum Na of 140 meq/L. 3 h later ONSD had reduced to 5.0 mm and HR improved to 58/min. Over the next 2 days, she intermittently received mannitol for high ICP suggested by raised ONSD value. ONSD was carried out on the clinical discretion of the intensivist based on decreased alertness, reduced Glasgow coma scale (GCS) score or changes in cardiorespiratory parameters. She was managed non-surgically with osmotherapy based on ONSD values and clinical findings alone. By 5th hospital day, her GCS started to improve. She was transferred out to the ward on the 7th day. In the second scenario a 47-year-old gentleman with hypertension for 10 years, developed a haemorrhagic stroke and right sided basal ganglia haematoma extending up to the temporal lobe. He was admitted in MSICU with a left sided hemiplegia and left sided upper motor neuron facial palsy. He was awake and alert with GCS score of 15/15 with stable vitals. 6 h after admission he developed bradycardia. Patient was shifted to trauma neuro ICU for anticipated need of surgical intervention. Twenty-four hours since admission he was only localising to pain. An ONSD at that instance measured 5.5 mm. A repeat CT scan showed an increase in the size of the haematoma. In view of deep seated haematoma and still a good motor score, surgical intervention was deferred and osmotherapy was initiated with 3% saline and a bolus of 200 ml of mannitol. Serial hourly ONSD was performed, and 2 h after the repeat scan ONSD measured 7.0 mm. There were no further clinical deterioration, but in view of presumed increase in cerebral oedema suggested by ONSD values, despite osmotherapy a right sided decompressive craniectomy was planned. Forty-eight hours after surgery, the patient started obeying commands and was successfully extubated and transferred out with a recovering left sided hemiparesis.

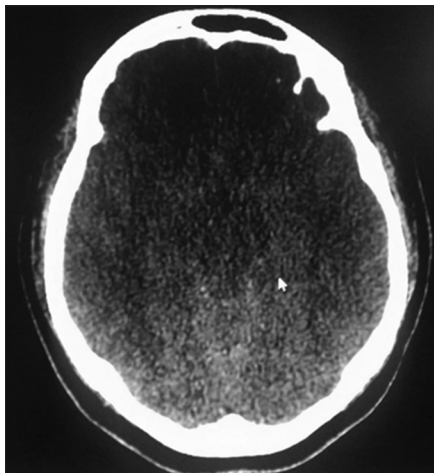


Figure 1: Computed tomography scan suggestive of raised intracranial pressure with effaced basal cisterns and obliterated ventricles

DISCUSSION

Non-invasive methods of intracranial pressure measurement

The various methods of measuring ICP non-invasively are tabulated in Table 1.

Radiological techniques

Computerised tomography

Various scoring systems have evolved based on CT findings to predict ICP and mortality.^[2-4] Effaced basal cisterns and ventricles, midline shift and loss of gray, white differentiation have been the most commonly used signs suggestive for raised ICP. However, even with a normal CT scan, the chances of raised ICP range 0–88%.^[5]

Magnetic resonance imaging

It has a better sensitivity compared to CT scan with the newer sequences like diffusion weighted image etc., Alperin *et al.*, developed an elastance index using motion sensitive magnetic resonance imaging (MRI) to measure pulsatile arterial, venous and cerebrospinal fluid flows in and out of cranial vault. This was found to correlate well

Table 1: Comparison of various non-invasive ICP measurement techniques

Methods	Sensitivity	Specificity
Radiological	Poor	High
ONSD	High	High
TCD	Poor	High
Ophthalmic vessel based	Poor	High
Tympanic membrane displacement	Poor	Poor
Pupillometer	High	High
Near infrared spectroscopy	Poor	High

ONSD=Optic nerve sheath diameter, TCD=Transcranial Doppler, ICP=Intracranial pressure

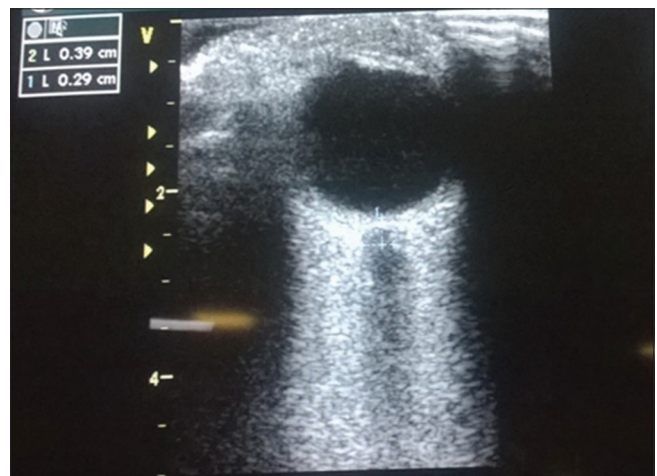


Figure 2: Optic nerve sheath diameter suggestive of raised intracranial pressure

with invasively measured ICP.^[6] However, the added time of investigation and high interobserver variation are major drawbacks.

Disadvantages of radiological techniques

- They are not bedside procedures and need the patient to be mobilised to the radiology suite
- During the transit 30° head elevation is invariably lost
- Minute to minute multimodal monitoring available in the ICU is lost.

Bedside non-invasive intracranial pressure monitoring

Transcranial Doppler based

Transcranial Doppler (TCD) is the handiest bedside tool to have in neuro ICU. It not only helps to assess blood flow [Figure 3] and severity of vasospasm, but ascertains brain death and can be used to measure ICP.

Pulsatility index

Pulsatility index (PI) has been the parameter used with TCD to measure ICP [Figure 4].

Principle

When resistance is high as in high ICP state the delta (peak systolic velocity-peak diastolic velocity) of flow in intracranial vessels is high [Figure 5] and hence PI is high. The correlation between ICP and PI was first looked upon by the Lund group.^[7] Based on the correlation between PI and ICP they computed an equation to predict ICP: $ICP = 10.927 PI - 1.284$. After the initial enthusiasm, many authors tried replicating but had mixed results. While Bellner *et al.*^[7] and Behrens *et al.*^[8] had a good correlation between PI and ICP, Voulgaris *et al.*,^[9] Homburg *et al.*^[10] and Moreno *et al.*^[11] did not find any correlation between the two variables. The possible reason could be that PI is influenced by several commonly present variants in an intubated patient [Table 2].

Though PI did not show good sensitivity to detect raised ICP, it still has remained a useful bedside tool in the ICU. The Cambridge group^[12] recently reviewed utility of PI to predict ICP and found that, any correlation between the two is seen only when the ICP rises above 35 mm Hg (area under the curve [AUC] - 0.74). They concluded that PI could be useful in predicting at the bedside possibility of a catastrophic rise in ICP.

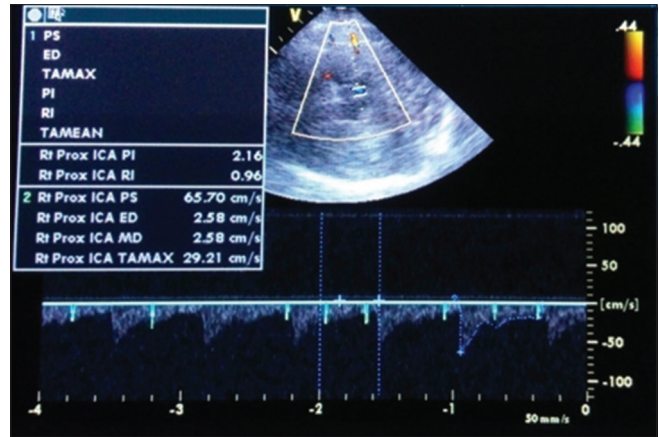


Figure 3: Transcranial Doppler assessment of blood flow and pulsatility index

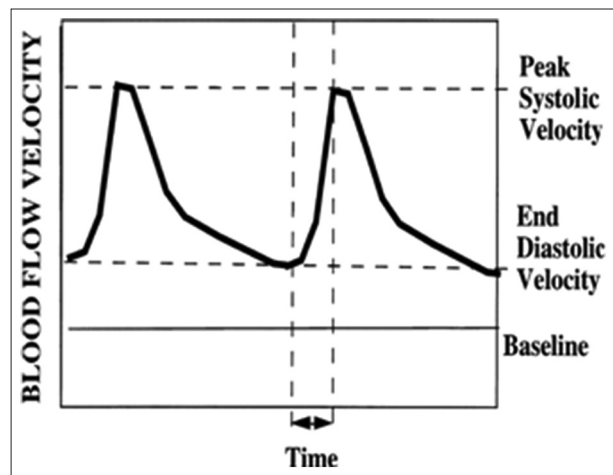


Figure 4: Measurement of pulsatility index



Figure 5: Pupillometer and its measure

Direction of flow index

With extreme high intracranial hypertension, decreased cerebral perfusion can be detected on TCD as a reversal of diastolic flow. Direction of flow index (DFI) is defined as $DFI = 1 - R/F$, where R is the velocity of the

Table 2: Factors affecting PI

Age of the patient
Disease process affecting the vasculature
Deficient autoregulation
Variation in arterial pressure
CO ₂ reactivity
PI=Pulsatility index

diastolic reverse flow and F is the velocity of the systolic forward flow.^[13] DFI <1 indicates a reversal of flow. Bandyopadhyay *et al.* used DFI to monitor the effect of sustained low efficiency dialysis on cerebral flow in hepatorenal syndrome with raised ICP.^[14]

Optic nerve sheath diameter

ONSD has been the most promising non-invasive technique for ICP measurement.

Principle

The dura mater extends along the optic nerve and the subarachnoid space beneath it. The raised ICP thus manifests as widened ONSD. The widening is most marked at 3 mm from the globe. Beyond 3 mm the presence of arachnoid trabeculations prevent widening of ONS.

ONSD is an ideal bedside measurement tool with ease to learn and quick to perform. Multiple measurements in the sagittal and coronal plane take <3 min.^[15] The procedure has a short learning curve with intensivists who are experienced in ultrasonography needing 10 (with 3 abnormal) scans to attain required skills. For novices, 20 scans are enough to start performing individually.^[16]

ONSD has a good sensitivity and specificity in predicting ICP in comparison to invasive ICP. Rajajee *et al.*, performed more than 530 ONSD measurements to define an accurate cut-off value. Having ruled out artefact measurements they established 4.8 mm cut-off as the best mark with the AUC of 0.98 (95% confidence interval [CI]: 0.96–0.99, sensitivity of 96% (95% CI: 91–99%), specificity 94% (95% CI: 92–96%), positive predictive value (PPV) 84% (95% CI: 77–89%) and negative predictive value (NPV) 99% (95% CI: 97–100%).^[15] Thus, higher cut-off increase the specificity of detecting raised ICP, but lose sensitivity thus increasing false negatives. In an elegant experimental set up Hansen *et al.*, worked on isolated human optic nerve preparations obtained from autopsies and submitted them to predefined pressure alterations, and consecutive changes in ONSD were measured by B-scan ultrasound under defined conditions.^[17] They found a linear relation of ONSD with ICP in upward and downward direction. A stepwise increase in ICP caused linear increase in ONSD with the initial pressure increase from baseline to 5 mm Hg resulting in a large change (mean 0.7 mm Hg) while later for every 10 mm Hg pressure increment there was only 0.25 mm Hg rise. The correlation was not so linear with reducing ICP. The decompression after increasingly higher pressure levels leaves higher residual ONSD.^[17]

The reliability of ONSD for dynamic ICP variation was evaluated by Rajajee *et al.*, using *post-hoc* analysis of the database from their previous study. They realised that

ONSD dropped the specificity from 98% to 74% and PPV from 90% to 76% when ICP fluctuated from normal to high and also that greater fluctuations led to greater reduction in specificity.^[18]

Launey *et al.*^[19] clinically reproduced the experimental results of Hansen *et al.* using osmotherapy with mannitol. They discovered that ONSD and invasive ICP reflected the drop in ICP after mannitol therapy at the same rate except when ONSD was wide to begin with. The dural swelling and microhaemorrhages within the dura matter was thought to delay the decrement of ONSD in comparison to invasive ICP.^[19]

Radiological measurement of optic nerve sheath diameter

To reduce technical flaws in the measurement of ONSD by ultrasonography, CT and MRI have been used. Geeraerts *et al.*^[20] used T2-weighted imaging of ONS, which correlated with invasive ICP in traumatic brain injured patients. Using a cut-off of 5.82 mm they obtained a NPV of 92%. More importantly, NPV reached 100% when ONSD was <5.3 mm.^[20] MRI may not always be available and added time of investigation is not very appealing. Hence, the possibility of using CT to measure ONSD was investigated. Sekhon *et al.*, found ONSD to correlate well with invasive ICP in severe traumatic brain injury (TBI) cohort with a low interobserver variability.^[21] Using 6 mm cut-off the AUC was 0.83 (95% CI: 0.73–0.94), PPV of 67% and NPV of 92%. They concluded that ONSD measurement by CT was a much stronger predictor of ICP (R^2 of 0.56) compared to other CT features (R^2 of 0.21).^[21] The same group also investigated if ONSD measured by CT could be used to predict mortality. Sekhon *et al.*, found that risk of in hospital mortality doubled with each 1 mm increase in ONSD (odds ratio: 2.0, 95% CI: 1.2–3.2, $P = 0.007$).^[22]

Limitations

- Interobserver variation
- Cut-off values not very specific
- Not applicable when ocular trauma present.

Pupillometer

The use of pupillometer helps to objectively measure the size, degree of reaction to light, latency and briskness of pupillary reaction to light. Based on these variables Chen *et al.*^[23] computed neurological pupillary index (NPi) with 3–5 being the normal index. They found that the NPi index dropped to <3 at an average of 15.9 h ahead of the peak in invasive ICP.^[23]

Near infrared spectroscopy

It is based on similar principle as of pulse oximeter. It detects changes in deoxyhaemoglobin and oxyhaemoglobin concentrations based on absorption of near infrared light. A pilot study showed the correlation

of low near infrared spectroscopy (NIRS) with raised ICP.^[24] A prospective observational trial looking at the possibility of predicting ICP in TBI using NIRS is underway (ClinicalTrials.gov Identifier: NCT01850069). With the current data, the international multidisciplinary consensus conference on multimodality monitoring in neurocritical care does not advocate routine use of NIRS in decision making.^[25]

Tympanic membrane displacement

Tympanic membrane displacement (TMD) is based on the proposition that an aural stimulus above acoustic reflex threshold causes a Chain of movements involving the ossicles especially the stapes which rests on the oval window of the cochlea. If the cochlear aqueduct is patent and communicates with the intracranial spaces the TMD is reflects the change in ICP. However, when Shimbles *et al.*, compared TMD with invasive ICP measurements in 36 patients, they found a high intersubject variability and a low predictive value for the technique.^[26]

CONCLUSION

The non-invasive ICP measuring tools are not surrogate for invasive ICP measurement. Clinical examination and CT findings form the basis of all neurological assessments, and the non-invasive methods are an adjunct to them. However, they are extremely important alternative tools available to follow ICP in a timely fashion and prevent intracranial catastrophes. They may be vital to triage patients with intracranial emergencies at presentation. With the ongoing volume of research around these tools, the future for non-invasive measurement of ICP looks bright.

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Conflicts of interest

There are no conflicts of interest.

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