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A DAY IN THE LIFE OF A NEUROCRITICAL CARE TRAINEE

Noninvasive ICP Monitoring by Serial Transcranial Doppler in Coagulopathic Patient

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Introduction

Intracranial pressure (ICP) monitoring is often a crucial component in the management of neurocritical care patients. However, standard methods for ICP monitoring, such as intraventricular catheters, intra-parenchymal sensors, and subarachnoid bolts, are largely invasive and their placement carries particular risk of hemorrhage in coagulopathic patients. Methods for noninvasive monitoring of ICP (nICP) are evolving to fill this need for patients who are not candidates for standard ICP monitor placement. Here, we present a case using serial transcranial Doppler (TCD) ultrasonography as a method of nICP monitoring.

Case

A 46-year-old man was admitted to the neuro-intensive care unit (ICU) postoperatively from dural AV fistula (DAVF) resection and was noted to have worsening level of arousal 12 h post-procedure. His past medical history was notable for remote traumatic brain injury (TBI), seizure disorder, and right frontal multi-compartmental hemorrhage caused by Borden grade 3 superior sagittal sinus DAVF 4/2018 [1]. At his baseline, he was ambulatory with left spastic hemiparesis. Due to continued growth of his DAVF despite partial embolization procedures, the patient underwent an elective DAVF resection. Postoperatively, the patient woke briskly to voice and followed commands. Within 12 h, the patient roused only to deep pain stimuli, stopped following commands, and was perseverative in his speech. Head computed tomography showed trace subdural blood products along the posterior falx and mild increased size of the lateral ventricles. Long-term electroencephalogram monitoring showed two brief parieto-temporal origin seizures. Diagnostic cerebral angiogram revealed complete superior sagittal sinus thrombosis, venous engorgement, and delayed arterial flow (Fig. 1a). The patient was intubated for sinus thrombectomy 24 h post-resection. The thrombectomy was attempted but unsuccessful. A low dose of fixed-rate propofol was started for sedation. The patient's venous engorgement on cerebral angiogram was concerning for impaired venous drainage and risk for high ICP—a 3% sodium acetate infusion was initiated for serum sodium goal > 145 mEq/L for prophylactic hypernatremia. A heparin drip was started for venous sinus thrombosis 28 h post-resection, on return from unsuccessful thrombectomy, with partial thromboplastin time (PTT) goal 2–2.5 times the patient's baseline PTT. The patient was ventilated to pCO_2 goal 35–40 mm Hg. Magnetic resonance imaging of the brain 48 h after resection demonstrated new edema and effacement of sulci within the parietal and occipital lobes concerning for parenchymal edema due to venous hypertension versus developing infarction (Fig. 2). A goal was determined to minimize cerebral metabolism and encourage formation of cortical vein collateralization for the thrombosed superior sagittal sinus, while monitoring intracranial pressures due to threat of impending venous infarcts. As the patient was not a candidate for standard invasive ICP monitors due to his ongoing therapeutic anticoagulation, TCD testing for noninvasive ICP monitoring (estimated $nICP = 4.47 \times PI + 12.68$ mmHg [2]) from postoperative day 3 was initiated (Table 1). Propofol infusions and hyper-osmolar therapy were titrated based on concerning trends in TCD values, as discussed below.

Management

Day 3

- *Interpretation* It globally increased pulsatility indices and mean flow velocity (MFV) borderline was low in the right middle cerebral artery (MCA) (34).

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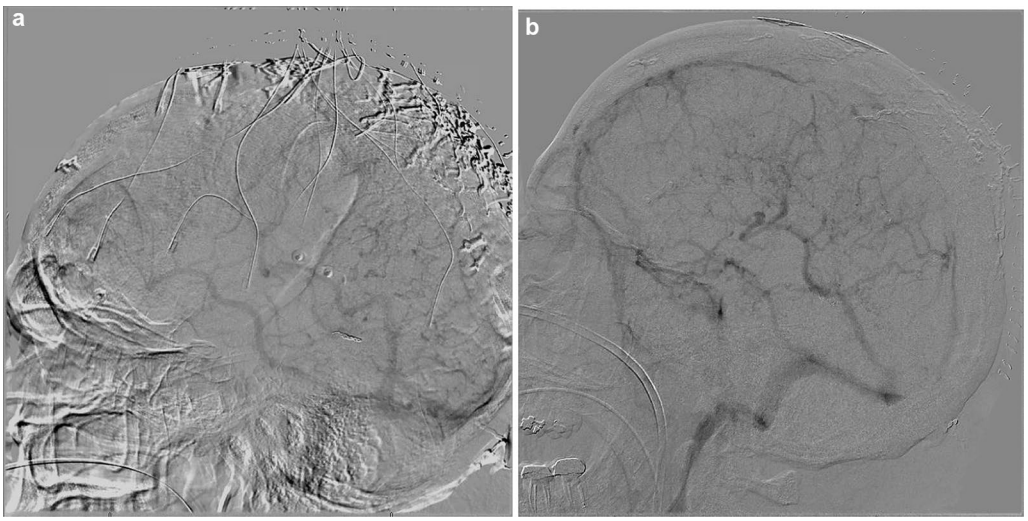

Fig. 1 Initial and follow-up angiograms. **a** (DSA #1): absence of opacification of the superior sagittal sinus (SSS) following surgical resection for multiple dural arteriovenous fistulae, concerning for SSS thrombosis, with patent straight sinus, transverse sinuses, and sigmoid sinuses. **b** (DSA #2): delayed filling of the anterior and middle superior sagittal sinus from the right. However, most venous drainage appears cortical and peri-SSS and then into the transverse sinuses or the superior ophthalmic vein on the left

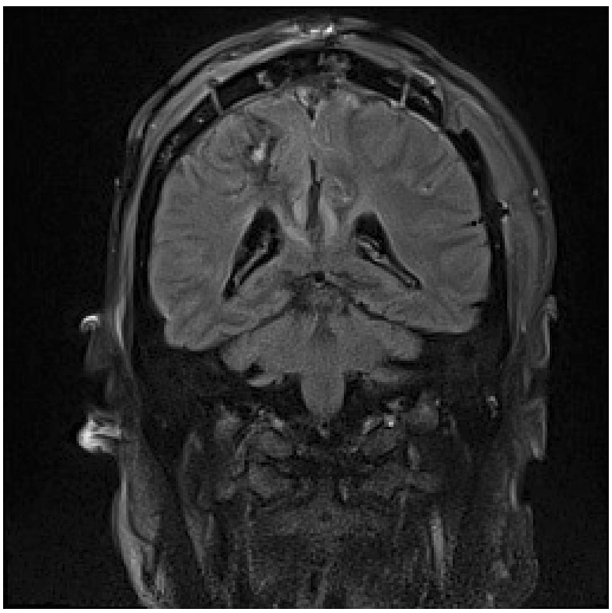

Fig. 2 FLAIR coronal image, 48 h post-surgery. Focal left posterior parietal lobe edema with additional areas of edema and sulcal effacement seen along the paramedial parieto-occipital lobes

TCD concerning for high resistance to flow suggests increased ICP, but without hemodynamic compromise (Fig. 3a).

- *Management* Propofol infusion was increased to 45–60 mcg/kg/min to decrease cerebral metabolic demand. Fentanyl injections were given as needed for ventilator dysynchrony despite propofol. A 3% sodium acetate infusion was continued for a serum sodium goal of 145–155 mEq/L.

Day 4

- *Interpretation* It increased the pulseality index (PI) (1.1–1.7), with marked decreased MFV in the bilateral terminal internal carotid arteries (ICAs), left anterior cerebral artery (ACA) and right MCA (terminal ICAs 27, 28; left ACA 33; and right MCA 38) and ongoing resistive waveforms. TCD concerning for worsening high resistance to flow suggests worsening ICP, now with hemodynamic compromise (Fig. 3b).
- *Management* Patient received 23.4% saline bolus, with mild improvement in MFVs and waveform on repeated TCD 2 h after bolus. The patient continued propofol 50 mcg/kg/min and was started on scheduled clonidine, propranolol, and gabapentin along with fentanyl infusion at 100 mcg/kg/min for episodes of neurogenic storming. Sodium acetate rate was increased to maintain serum sodium goal.

Day 5

- *Interpretation* It increased PIs 1.4–2.19, with accompanying resistive waveforms and ongoing low MFVs (bilateral terminal ICAs 23 and 30; left ACA 30).

Table 1 Progression of patient's TCD values over four days

	Day 3	Day 4 AM	Day 4 PM	Day 5 AM	Day 5 PM	Day 7 AM	Day 7 PM (midazolam wean)
MCA mean flow velocity (MFV, cm/s)	R 34 L 47	38 44	32 44	43 48	53 57	59 76	41 51
Median PIs	1.3 (0.9–1.57)	1.47 (1.12–1.7)	1.39 (1.1–1.6)	1.64 (1.4–2.2)	1.52 (1.4–1.8)	1.4 (1.1–1.6)	1.3 (1.1–1.6)
Extrapolated ICP values (mmHg)	18.5 (16.7–19.7)	19.3 (17.7–20.3)	18.9 (17.6–19.8)	20.0 (18.9–22.5)	19.5 (18.9–20.7)	18.9 (17.6–19.8)	18.5 (17.6–19.8)
Median MAPs (mmHg)	73	66	78	72	69	69	71
Median pCO_2 (mmHg)	33	35	36	34	38	36	33
Temperature (C)	36.7	37.4	36.9	37.2	36.9	37.7	37.7
Sodium levels (mEq/L)	147	154	156	156	155	155	156

ICP intracranial pressure, *MAP* mean arterial pressure, *MCA* middle cerebral artery, *MFV* mean flow velocity, *PI* pulsatility index

Worsening high resistance to flow suggests worsening ICP and continued hemodynamic compromise (Fig. 3c).

- *Management* Patient received 23.4% saline bolus, with improvement in PIs globally on repeated TCD 2 h after bolus. Propofol infusion transitioned to midazolam at 0.2 mg/kg/h in the setting of serum triglycerides 509 concerning for early propofol infusion syndrome. Propranolol was held in setting of bradycardia episodes. Scheduled clonidine and gabapentin, along with fentanyl infusion, continued for neurogenic storming episodes.

Day 7

- *Interpretation* TCD PIs trend downwards, ranging from 1.13 to 1.66, along with a less resistive waveform than previous readings. MFVs are within normal ranges. Improving compliance to flow was reassuring and prompted weaning of sedation (Fig. 3d).
- *Management* Midazolam weaned to 0.1 mg/kg/h, and fentanyl infusion decreased from 100 to 50.

Remainder of ICU Course The patient was safely extubated 12 days after surgery. There were no disruptions in the patient's anticoagulation from initation. On examination, the patient woke briskly to loud voice, followed axial and appendicular commands though was inattentive for multi-step commands, answering orientation questions (self, family, year, location) appropriately. An angiogram completed 14 days postoperatively showed partial filling of the sagittal sinus, with increased recruitment of cortical venous drainage (Fig. 1). TCD completed 21 days post-resection showed PIs ranging from 1.02 to 1.5, MFVs within normal ranges, and low resistance waveforms typical for the cerebral circulation (Fig. 3e).

Discussion

ICP monitoring and management are often limited by safety of invasive monitor placement, particularly in patients with coagulopathy. In these settings, TCD ultrasonography can be a useful bedside tool for repeated, noninvasive monitoring of cerebrovascular hemodynamics as an indirect marker for trends in ICP and cerebral perfusion (Fig. 4).

Common TCD variables are blood flow velocities such as peak systolic velocity (PSV), end-diastolic velocity (EDV), MFV, and PI. PI is calculated as the difference between systolic and diastolic flow velocities, divided by the mean velocity. It measures the variability of blood velocity in a vessel and describes distal cerebrovascular resistance. The TCD waveform itself shows a low resistance pattern characteristic of cerebral hemodynamics, with sharp systolic upstroke and slow diastolic decay [3, 4].

These TCD-derived parameters change with increasing ICP. Cerebral vessel compliance decreases with increased ICP. Decreased compliance in turn affects resistance to flow, as the waveform of blood flow velocity will show sharper systolic decay, low EDV, and decreased amplitude [3, 4]. In settings of stable systemic blood pressures and $PaCo_2$, ICP shows a positive linear correlation with PI in patients with critical brain injury [5]. TCD waveform analysis, combined with trends in MFV and PI, help serve as a technique for nICP estimation.

For our patient, we did not have a baseline TCD prior to surgery, but we were able to do twice daily TCDs at the bedside and follow trends over time. The first TCD showed normal range mean flow velocities, but the high PIs and waveforms with a rapid systolic decay were both concerning for increased resistance and ICP without hemodynamic compromise. TCDs on the following

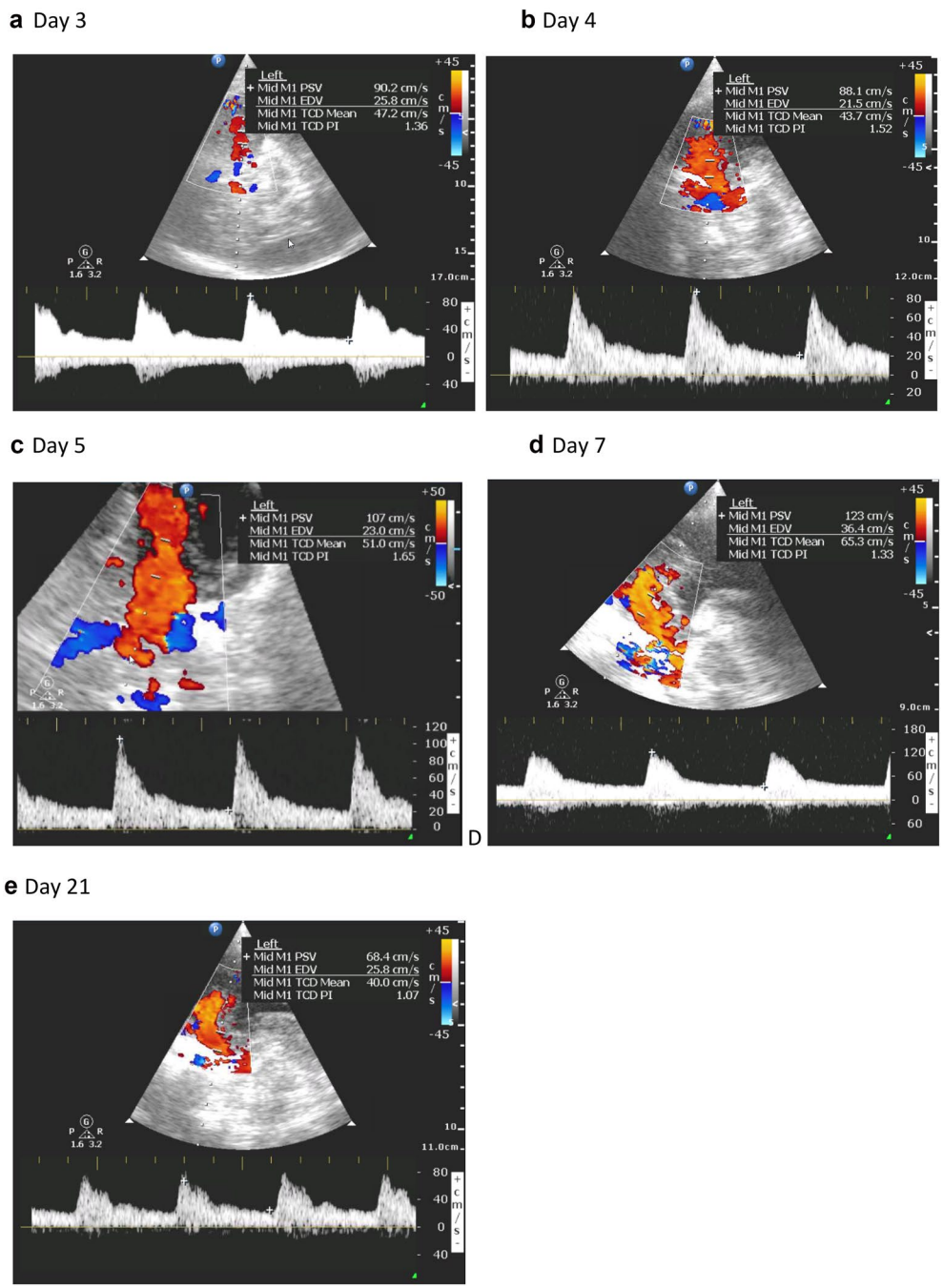

Fig. 3 TCD waveforms postoperative days 3–7 L MCA, and 21 days post-surgery (from left upper to right)

2 days showed an evolution to higher PIs and sharper waveforms with lower mean flow velocities. The changes suggested increasing resistance to flow, with evolving hemodynamic compromise. Repeated TCD waveforms after increased sedation and escalation of ICP-lowering therapies showed improvement. On postoperative day 7, waveforms showed improved compliance, MFV, and PIs, aiding the team in their decision to begin weaning sedation.

Our teaching case has some limitations. Though our patient showed clear evolution of TCD waveforms over his postoperative week, it is important to note our patient never had raised ICP based on our chosen formula for nICP derivation. Some improvement in his waveforms

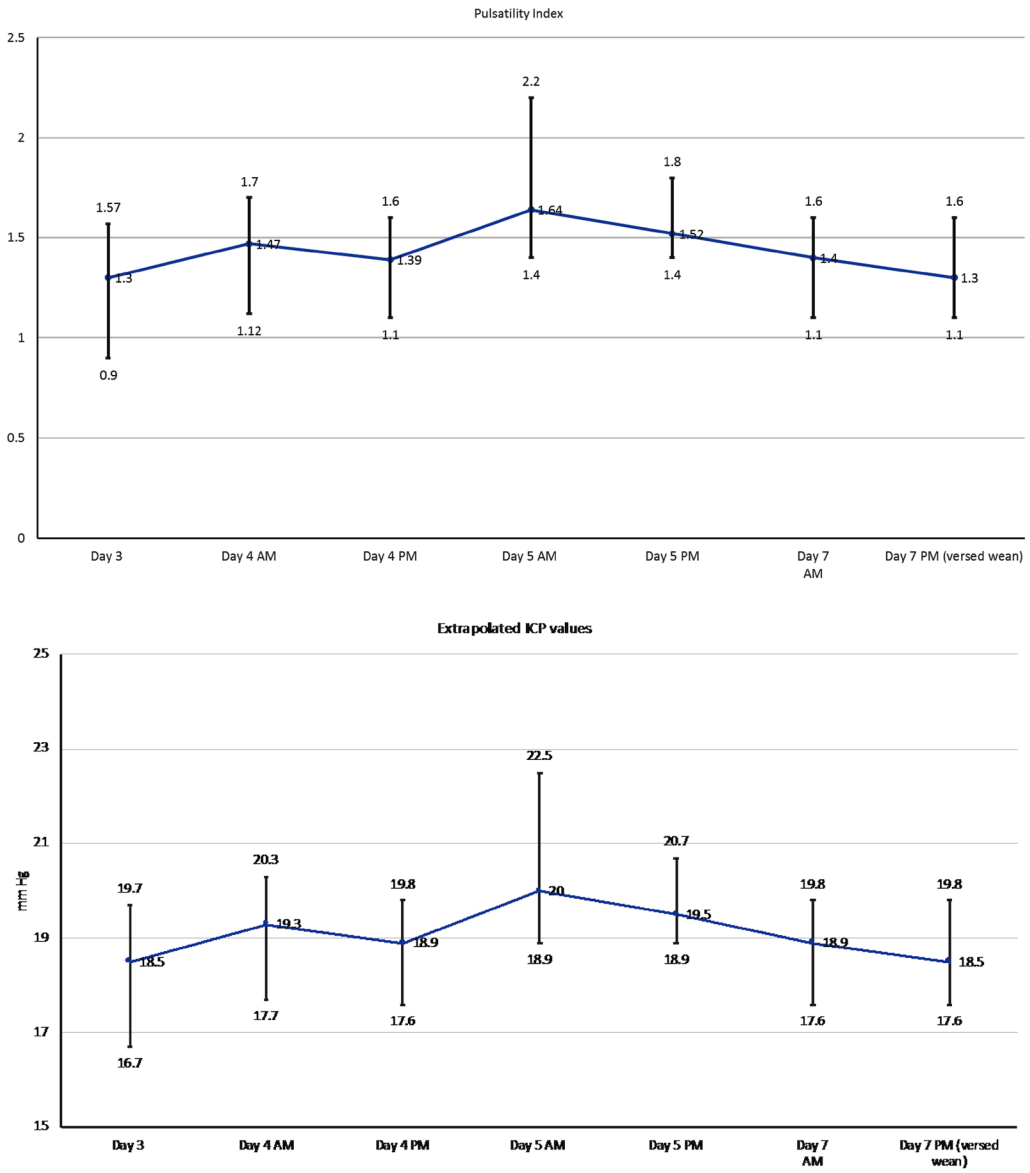

Fig. 4 Line graph of PI and extrapolated ICP over time

may have also occurred in general resolution of cerebral edema over time, in addition to our ICP-lowering therapies. We did not perform any TCDs off sedation. Due to the patient's poor ventilator synchrony and neurogenic storming, sedation was not held for prolonged periods of time to assess for other signs of clinical improvement. There are no TCD data available from day 7 until extubation.

There are many formulas for nICP measurement, based mainly on TBI pathology, with significant variation between the formulas [2, 6, 7]. Just as ICP should not be considered solely as a number but also as its waveform and compliance, TCD is best interpreted as a combination of its measured parameters as well as its waveform trends. More recent research has focused on quantitative relationships between cerebrovascular dynamics and ICP [7, 8]. There is considerable variability in the reported accuracy of these methods, in part due to differing methods for the derived calculations. Cerebral MFV and PI in particular are in reality complex functions of hemodynamic factors and are affected by several interdependent intracranial parameters [9, 10]. However, repeated TCD measurements in an otherwise hemodynamically stable patient can be extremely helpful to trend changes

in cerebral hemodynamics and compromise of flow over time, as we have found in the care of our patient.

When invasive ICP monitoring is contraindicated, TCD ultrasound is a reliable, repeatable, and noninvasive bedside method for trending cerebral hemodynamics as a surrogate for ICP. This application is particularly promising to future neurocritical care patients unable to receive invasive ICP monitors due to ongoing anticoagulation or synthetic coagulopathy in hepatic and hematologic disorders and should be a part of standard neurocritical care and vascular neurology education.

Key points

1. TCD ultrasonography is a useful bedside tool for repeated, noninvasive monitoring of cerebrovascular hemodynamics as indirect markers for trends in ICP and cerebral perfusion.
2. TCD-derived parameters change with increasing ICP. TCD waveform analysis, combined with changes in MFV and PI, help trend changes in ICP.
3. TCD is best interpreted as a combination of its measured parameters as well as waveform trends.

Author Contributions
SG, JG, and AS were involved in the creation of this manuscript.

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None.

Conflicts of Interest
All authors declare that they have no conflict of interest.

Ethical Approval/Informed Consent
IRB-approved study with patient informed consent.

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