

Transcranial Doppler for Early Cerebral Hemodynamic Assessment in Traumatic Brain Injury when Invasive Monitoring is Unavailable: A Narrative Review

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Abstract

Traumatic brain injury (TBI) can produce rapidly changing cerebral hemodynamic when invasive intracranial pressure (ICP) monitoring is unavailable or delayed. This narrative review examines the potential role of transcranial Doppler (TCD) in early goal-directed management of adults with moderate-to-severe TBI under these circumstances. Evidence is synthesized across four domains: detection of early low-flow states, assessment of intracranial hypertension and cerebral perfusion, evaluation of autoregulatory disturbance and cerebrovascular tone, and serial assessment of treatment response. Reduced flow velocity, altered diastolic flow, and increased pulsatility may identify concerning cerebrovascular patterns, but their interpretation depends on systemic pressure, PaCO₂, vascular tone, and technical factors. Serial TCD can demonstrate hemodynamic changes after osmotherapy, hemodynamic optimization, hyperventilation, or sedation. Recommended reassessment intervals remain based primarily on expert opinion. However, velocity does not measure absolute cerebral blood flow, and pulsatility cannot reliably quantify ICP. Evidence for TCD-guided therapeutic decisions remains limited, with no established improvement in patient outcomes. TCD is therefore best used as an adjunct to clinical assessment and other available monitoring modalities, not as a substitute for invasive monitoring. Prospective studies should test whether structured TCD-informed management improves clinical outcomes.

Keywords: Cerebral autoregulation, intracranial hypertension, noninvasive monitoring, transcranial doppler, traumatic brain injury

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Introduction

Traumatic brain injury (TBI) remains a major cause of death and disability. Early management focuses on preventing secondary insults, including hypoxia, hypotension, impaired cerebral perfusion, and intracranial hypertension.¹ Cerebral autoregulation is frequently impaired after TBI, so a given cerebral perfusion pressure may not produce the same cerebral blood flow across patients or over time.² Invasive Intracranial Pressure (ICP) monitoring remains central to severe TBI management, but it may be delayed, unavailable, or impractical during transport,

before monitor placement, or in resource-limited settings. This creates a period in which clinically important cerebral haemodynamic abnormalities may be present without continuous intracranial monitoring.³ Transcranial Doppler (TCD) offers a rapid, repeatable bedside measurement of cerebral arterial flow velocity and waveform characteristics.⁴ It does not measure absolute cerebral blood flow, but changes in mean and diastolic flow velocity, pulsatility, and pressure–flow relationships can provide information relevant to hypoperfusion, intracranial hypertension, cerebrovascular tone, and autoregulatory function.⁴ Interpretation remains physiological rather than purely numerical because flow

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velocity and pulsatility are influenced by arterial pressure, PaCO_2 , vascular tone, haematocrit, and other systemic and technical factors.^{4,5}

Current management provides a useful framework for defining where TCD might contribute. The Brain Trauma Foundation (BTF) recommends treatment around established ICP and Cerebral Perfusion Pressure (CPP) thresholds, while Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC) organizes ICP-directed therapy into escalating treatment tiers.^{1,6} The Brussels consensus for non-invasive ICP monitoring (B-ICONIC) extends this concept to situations in which invasive ICP monitoring is unavailable by combining clinical examination, imaging, and non-invasive neuromonitoring, including TCD.³ TCD should therefore be considered an adjunct within this framework rather than a replacement for invasive monitoring.

This review examines whether TCD can provide actionable cerebral haemodynamic information during the early management of adults with moderate-to-severe TBI when invasive monitoring is unavailable. The evidence is organized into four domains: early detection of low-flow states; assessment of intracranial hypertension and cerebral perfusion; assessment of autoregulatory disturbance and cerebrovascular tone; and serial TCD assessment of response to goal-directed therapy. The central question is how these applications may inform bedside management while accounting for the physiological and methodological limitations of TCD.

Early Detection of Cerebral Hypoperfusion

One proposed pattern of early TCD abnormality after moderate-to-severe TBI is reduced mean flow velocity (FVm) in the middle cerebral artery accompanied by a normal pulsatility index (PI). Because PI is influenced by downstream cerebrovascular resistance, a normal PI with reduced FVm may suggest that increased distal resistance is not the dominant mechanism. This pattern may therefore indicate a deficit in cerebral driving pressure, although it does not establish

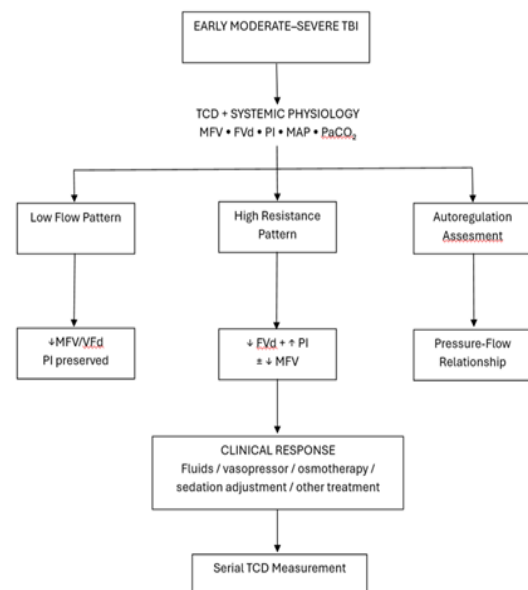


Figure 1. Conceptual framework for TCD assessment in early moderate-to-severe traumatic brain injury, integrating three physiological patterns with serial reassessment over time

the underlying cause.^{4,7} An early observational study proposed three theoretical mechanisms by which Middle Cerebral Artery (MCA) flow velocity may decrease after TBI: (1) low cerebral blood flow secondary to low cardiac output, in which both the MCA/ Internal Carotid Artery (ICA) velocity ratio and PI remain normal; (2) a larger effective diameter of the insonated vessel, in which PI falls; and (3) elevated peripheral cerebrovascular resistance, in which PI rises. The low-flow, normal-PI pattern discussed here corresponds to the first proposed mechanism. However, the authors' observed low-velocity patients predominantly had elevated PI, and the normal-PI subgroup was not separately validated.⁷

TBI patients may experience systemic contributors to reduced cerebral perfusion. Concomitant hemorrhage can reduce circulating volume, while TBI-associated cardiac dysfunction may impair cardiac output. Myocardial injury has been reported in roughly one-third of TBI patients and echocardiographic systolic dysfunction in about one-sixth.⁸ However, the relationship between these systemic disturbances and the low-FVm,

normal-PI pattern has not been directly established in TBI. No universally agreed threshold exists for defining a low-flow state on TCD. In a study involving 57 patients with severe TBI, low flow was defined as MCA FVm ≤ 35 cm/s. This finding was observed in 63% of patients and was most pronounced during the first eight hours after injury. Other studies used FVm < 30 cm/s combined with diastolic flow velocity (FVd) < 20 cm/s as part of abnormal-TCD criteria that also included elevated PI.^{9,10} Taken together, these studies suggest commonly used low-velocity thresholds of approximately 30–35 cm/s for FVm and < 20 cm/s for FVd.

However, none establishes a validated cut off specifically for the normal-PI pattern. Because this pattern may reflect a deficit in cerebral driving pressure, clinical assessment should include circulating volume, arterial pressure, and cardiac function. When hypovolemia or impaired cardiac output is identified, treatment should address the underlying disturbance rather than the TCD finding alone. Recognizing this pattern may encourage clinicians to investigate systemic contributors instead of automatically attributing reduced FVm to intracranial hypertension. However, distinguishing hypovolemia or reducing cardiac output from other causes of reduced velocity remains uncertain without supporting hemodynamic data.

Assessment of Intracranial Hypertension and Cerebral Perfusion

TCD pattern with falling mean flow velocity (FVm) accompanied by a rising pulsatility index (PI) reflects a different mechanism from the driving-pressure deficit described before. This pattern develops in a step-wise sequence as ICP rises. First, systolic velocity (FVs) increases while diastolic flow becomes blunted. If ICP continues to rise, diastolic flow reverses, oscillating to-and-fro with each cardiac cycle. With further rise, only brief, low-velocity systolic spikes remain, with no diastolic flow at all. At the extreme, no flow is detectable.¹¹ The evolution of the waveform may therefore provide useful physiological information, as

progressive changes reflect worsening cerebral perfusion rather than an isolated abnormal value. These waveform changes may raise suspicion of intracranial hypertension and impaired cerebral perfusion. However, reduced FVm with elevated PI is not specific for intracranial hypertension and should be interpreted alongside other clinical and physiological findings. TCD therefore provides supportive information rather than a definitive diagnosis of intracranial hypertension.³

CPP will decrease as ICP rises, as MAP remains unchanged.⁴ The resulting drop in transmural pressure across small cerebral arterioles compresses them, raising downstream vascular resistance.¹² FVd is affected first and most severely, since it is during diastole that intracranial pressure exerts its greatest relative effect on vessel caliber; peak systolic velocity is comparatively preserved.¹¹ This disproportionate fall in FVd relative to systolic velocity, and the consequent fall in FVm, manifests as a rising PI.^{4,11} Rising ICP reduces CPP and diastolic flow, leading to lower MFV and higher PI. This forms the basis for using PI as an indirect marker of intracranial hypertension. A recent pooled analysis across seven studies found a best cut-off PI of 1.28 (sensitivity and specificity both ≈ 0.74) for detecting Cerebral Blood Flow (CBF) changes linked to elevated ICP. Based on this, expert consensus proposes a working threshold of PI > 1.3 with FVd < 20 cm/sec to suspect or exclude these changes, though this recommendation is graded as low-level evidence.³

However, PI is not a direct substitute for ICP. It is influenced by cerebral arterial compliance, heart rate, arterial pulse pressure, cerebrovascular resistance, and CPP.⁵ Changes in PaCO₂ can also alter PI independently of ICP. In head-injured patients, PI increased during both hypocapnia, when ICP decreased, and plateau waves, when ICP increased.⁵ A rising PI therefore cannot be attributed to intracranial hypertension without considering these confounders. A reduced FVd/FVm ratio may reflect increased ICP, but it can also occur with normal ICP when MAP decreases and cerebral autoregulation is impaired.¹²

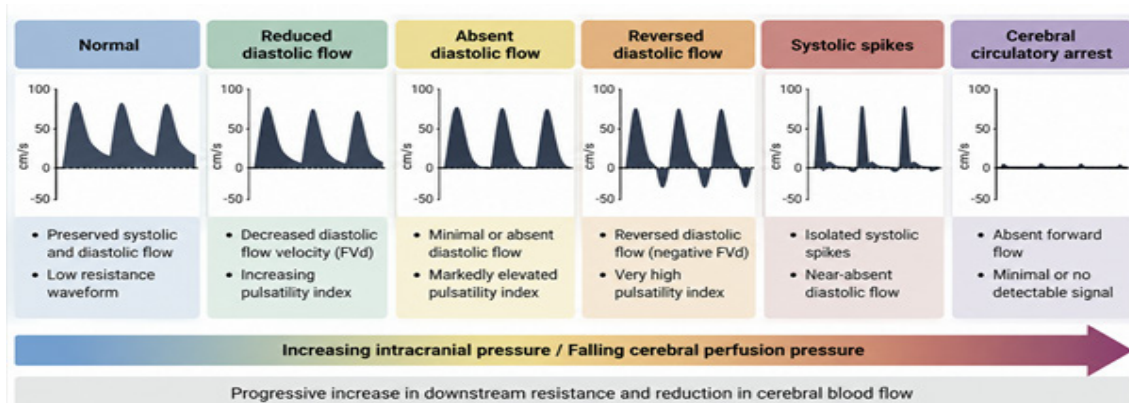


Figure 2. Schematic progression of transcranial doppler waveforms with increasing intracranial pressure and decreasing cerebral perfusion pressure. Physiological patterns based on previously published study.¹²

TCD-derived approaches to non-invasive ICP estimation have shown important limitations. In the prospective multicenter IMPRESSIT-2 study, 262 brain-injured patients provided 687 paired measurements of TCD-estimated and invasive ICP. Agreement was poor, with a concordance correlation coefficient of 0.333 and 95% limits of agreement ranging from -26.1 to +19.5 mmHg. Nevertheless, negative predictive values ranged from approximately 91% to 99%, depending on the ICP threshold.¹² These findings suggest that TCD-derived ICP estimates may have a role in screening for intracranial hypertension, but cannot reliably quantify its absolute magnitude.

Expert consensus positions TCD as a complementary adjunct, not a substitute for invasive monitoring. As one consensus states, "none of the available non-invasive intracranial pressure (nICP) tools is, at present, accurate enough to substitute invasive tools."³ TCD is most useful when invasive monitoring is delayed, unavailable, or contraindicated. This includes during transport, in resource-limited settings, or before catheter placement. In these situations, TCD can raise or lower suspicion of intracranial hypertension and support provisional decisions until definitive monitoring is available.³

Assessment of Cerebral Autoregulation

Cerebral autoregulation, the capacity of cerebral vessels to maintain relatively stable CBF across

changes in perfusion pressure, is frequently impaired after moderate-to-severe TBI. Across multiple studies, 49–87% of patients with severe TBI show absent or impaired autoregulation.² This matters because the classically taught autoregulatory plateau (MAP approximately 50–150 mmHg) is a general reference range rather than a fixed individual limit.² The upper and lower limits vary between individuals, so a fixed CPP target may lie within the autoregulatory range of one patient but outside that of another.⁴ When autoregulation fails, changes in perfusion pressure are transmitted more directly to cerebral blood flow, providing the rationale for assessing the pressure–flow relationship rather than relying on CPP alone.

TCD enables assessment of cerebral autoregulation by relating changes in cerebral arterial flow velocity to changes in arterial pressure. Intermittent and semi-intermittent methods include static and dynamic testing, which assess flow-velocity responses to deliberate pressure changes.^{13,14} Alternatively, spontaneous fluctuations in arterial pressure and flow velocity can be correlated using the mean flow index (Mx), without requiring deliberate pressure manipulation.¹⁵ Static testing transiently alters arterial pressure and compares the resulting change in cerebral flow velocity. If flow velocity changes little despite the pressure change, autoregulation is interpreted as relatively preserved; if flow velocity changes in parallel with pressure, autoregulation is

considered impaired.¹³ Static testing has important limitations. Deliberate pressure manipulation may be undesirable in the injured brain, and pharmacological or mechanical manipulation of CPP has been considered potentially inappropriate or unsafe in clinical practice.⁴ In a contemporary TBI series, 3.2% of attempted tests were stopped because ICP rose above 30 mmHg.¹³ The best-described clinical implementations are also not fully independent of invasive monitoring. Classical static rate-of-regulation methods relate changes in cerebrovascular resistance to changes in CPP.^{2,4} In a clinical TBI study using a MAP-based autoregulatory index, all patients underwent invasive ICP monitoring, and the pressure challenge was performed while ICP and CPP were observed.¹³ The applicability of this approach when invasive monitoring is unavailable therefore remains uncertain.

Dynamic testing assesses autoregulation by producing a brief reduction in arterial pressure, classically through thigh-cuff deflation, and observing the recovery of cerebral flow velocity. The pressure–flow response can be analyzed using mathematical models to derive an autoregulatory index (ARI), with lower values indicating impaired autoregulatory capacity.¹⁵ The mean flow index (Mx) assesses autoregulation by correlating mean TCD flow velocity with spontaneous fluctuations in arterial pressure or CPP over moving time windows. The MAP-based version, Mxa, does not require ICP monitoring and is therefore applicable

when invasive monitoring is unavailable.¹⁵ Values near zero indicate little correlation between pressure and flow velocity, where as increasingly positive values suggest pressure-passive flow and impaired autoregulation. Dynamic testing has been studied mainly as a way to track autoregulation over time after injury, rather than to guide treatment directly. In 122 patients with severe TBI, ARI worsened during the first 36–48 hours despite increasing cerebral blood flow and only partially recovered by day.^{10,14} Treatment followed a fixed protocol and was not adjusted according to ARI findings. These findings support the role of dynamic TCD-based autoregulation testing in serial physiological assessment, but do not establish its ability to guide individualized treatment.

Serial TCD to Guide or Assess Therapeutic Interventions

TCD's value is not limited to detecting the progression of disease; it can also track the response to management. Current severe TBI management uses ICP and CPP thresholds to guide escalation and de-escalation of therapy. BTF and SIBICC use targets of ICP <22 mmHg and CPP ≥60 mmHg. Treatment is organized in tiers, with sedation and hyperosmolar therapy used early, followed by measures such as mild hyperventilation and MAP challenge, while decompressive craniectomy is reserved for refractory cases.^{1,6} When continuous ICP monitoring is unavailable, serial TCD may provide a measurable way to follow changes in

What serial TCD can support	What serial TCD cannot support
• Direction of change in MFV, FVd, and PI • Persistence or normalization of an abnormal TCD pattern • Whether the cerebrovascular response is compatible with the expected effect of treatment • Change in the cerebral hemodynamic pattern over time	• Absolute cerebral blood flow • Exact intracranial pressure • Magnitude of ICP change from change in PI or other TCD parameters • Whether every observed change is necessarily beneficial or safe

Figure 3. Role and limitations of serial TCD during therapeutic reassessment in traumatic brain injury

cerebral hemodynamics over time and assess the response to treatment.

Osmotic therapy has the most direct evidence. In 80 patients with severe diffuse TBI, a fixed 0.3 g/kg mannitol dose reduced PI by approximately 11% and increased FVd by 30%, with maximal changes at one hour and significant effects reported at two hours.¹⁶ In a randomized comparison of equimolar mannitol and hypertonic saline, both agents reduced ICP comparably, but mannitol produced more sustained increases in FVd and FVm and a greater reduction in PI.¹⁷ The authors suggested that improved blood rheology might contribute to this difference, although this mechanism was not directly established. Together, these findings suggest that serial TCD can detect cerebrovascular responses to osmotherapy that are not fully reflected by changes in ICP. However, these responses cannot independently establish the magnitude of ICP reduction or treatment efficacy.

In practice, osmotherapy is rarely used on its own. Two further studies investigated TCD-guided early hemodynamic optimization, in which the flow pattern helped determine whether patients received an osmotic agent, a vasopressor, or both. In the first study, nine patients received norepinephrine to raise MAP, five received mannitol, and three underwent immediate surgery. Across the group, PI fell from 2.1 ± 0.5 to 1.4 ± 0.3 and FVd rose from 13 ± 5 to 25 ± 8 cm/s within a few hours.⁹ All but two patients had normalized TCD findings by the time invasive monitoring became available. A subsequent prehospital pilot study applied a similar approach, using norepinephrine when MAP was below 80 mmHg and mannitol when MAP was adequate or mydriasis was present. TCD findings normalized in five of nine treated patients, while none of the four non-responders survived beyond 48 hours.¹⁰ Guidelines treat hyperventilation cautiously, and this shapes the role of TCD during treatment. BTF discourages prophylactic hyperventilation, particularly during the first 24 hours after injury, and recommends brain oxygenation monitoring with jugular venous oxygen saturation (SjO_2) or brain tissue oxygen partial pressure ($PbtO_2$) when hyperventilation is used.¹ SIBICC permits

mild hyperventilation ($PaCO_2$ 32–35 mmHg) at Tier 2 but also emphasizes caution when brain oxygenation monitoring is unavailable.⁶ Both guidelines recognize that reducing ICP through hyperventilation may compromise cerebral perfusion and oxygen delivery.

Hyperventilation causes cerebral vasoconstriction, reducing cerebral blood flow and cerebral blood volume, which may subsequently lower ICP. In patients with acute TBI, controlled hypocapnia reduced mean flow velocity from 52 ± 17 to 45 ± 15 cm/s, while ICP decreased from 20 ± 11 to 14 ± 8 mmHg. However, changes in flow velocity correlated only modestly with changes in xenon-measured cerebral blood flow ($R^2 = 0.26$).¹⁸ TCD therefore demonstrated the direction of the response but could not reliably quantify the corresponding change in cerebral blood flow. PI is also difficult to interpret during hyperventilation. In one study, PI increased from 1.09 to 1.20 during hypocapnia despite a reduction in ICP. Conversely, PI also increased during plateau waves associated with rising ICP.⁵ Thus, a rising PI does not necessarily indicate an increase in ICP. Serial TCD can demonstrate the cerebrovascular response to hyperventilation, but it cannot independently determine whether the associated reduction in cerebral perfusion remains safe. Brain oxygenation monitoring therefore remains important when available.³

Sedation in TBI facilitates mechanical ventilation, limits coughing and straining, helps control seizures, and reduces ICP. Its ICP-lowering effects include reduced cerebral metabolic demand, cerebral blood flow, and cerebral blood volume, as well as suppression of pressure-provoking responses.¹ Because sedative agents can also influence cerebrovascular tone and systemic hemodynamics, serial TCD may help assess changes in cerebral flow velocity and PI after sedation is initiated or intensified. The cerebral hemodynamic response to sedation is not determined by anesthetic depth alone. Under propofol, cerebral flow velocity decreases progressively as Bispectral Index (BIS) falls, consistent with preserved flow–metabolism coupling. With sevoflurane, however, this

relationship becomes dissociated at deeper anesthesia: despite further reduction in BIS, FVM rises again, reflecting the increasing influence of direct cerebrovascular vasodilation.¹⁹

Beyond physiological treatment response, limited evidence suggests that serial TCD may also influence escalation of care. A retrospective study involving 71 patients in a low-resource setting without invasive ICP monitoring found that patients monitored with TCD underwent decompressive craniectomy earlier than those without TCD monitoring (6 ± 4 vs. 8 ± 3 hours, $p = 0.003$). However, mean Glasgow Outcome Scale scores were lower in the TCD group (4.3 ± 1.1 vs. 4.7 ± 1.1 , $p = 0.047$). These findings suggest that TCD may facilitate earlier surgical escalation, but do not establish improved clinical outcome.²⁰

The optimal timing of serial TCD reassessment remains uncertain. Studies have used intervals ranging from continuous monitoring to fixed 30-minute or multi-hour reassessment. The B-ICONIC consensus recommends serial TCD every 2–4 hours in severe TBI and every 4–6 hours in moderate TBI, although this is a weak recommendation based on expert opinion.³ The same consensus identifies an increase in PI of ≥ 0.5 from baseline as a finding that may support suspicion of intracranial hypertension, but advises against using changes in TCD parameters to estimate the magnitude of ICP change. No randomized trial has established whether a fully TCD-guided treatment protocol improves clinical outcomes. The BTF's fourth edition also identified insufficient evidence to support a Level III recommendation for TCD1. Thus, although intervention-specific findings are promising, the evidence remains insufficient to establish a standardized TCD-guided treatment strategy.

Limitations of Transcranial Doppler for Cerebral Hemodynamic Assessment

TCD has important physiological limitations that constrain its interpretation. Flow velocity is a surrogate rather than a direct measurement of cerebral blood flow, while PI is influenced by cerebrovascular resistance, arterial compliance,

arterial pressure, heart rate, CPP, and PaCO_2 and therefore cannot be interpreted as a direct measure of ICP.⁵ These limitations become particularly important during therapeutic interventions. Hypocapnia, for example, can increase PI despite a reduction in ICP.^{5,18} Sedative agents may alter cerebral flow velocity through different metabolic and vascular effects.¹⁹ TCD can therefore demonstrate the direction of a cerebrovascular response, but cannot independently determine its mechanism, magnitude, or clinical safety.

Serial measurement can show whether an abnormal TCD pattern persists, improves, or worsens over time. However, changes in TCD parameters should not be assumed to represent equivalent changes in cerebral blood flow or ICP. The B-ICONIC consensus specifically advises against using changes in TCD-derived parameters from baseline to estimate the magnitude of ICP change.³ TCD assessment is also operator-dependent, and adequate insonation may not be possible in patients with an inadequate temporal acoustic window.⁴ Measurements should therefore be interpreted together with systemic haemodynamic, PaCO_2 , clinical findings, imaging, and other available non-invasive monitoring rather than as isolated treatment targets.³ The evidence base also limits how strongly TCD can be incorporated into treatment algorithms. Available studies are predominantly observational, thresholds and reassessment intervals remain incompletely validated, and no randomized trial has established that a TCD-guided management strategy improves clinical outcomes^{1,3}. Current evidence therefore supports TCD as an adjunct for physiological assessment and provisional decision-making rather than as a replacement for invasive monitoring.

Across the evidence reviewed, TCD appears to be the most useful for identifying cerebral haemodynamic patterns and following their evolution over time rather than for providing absolute physiological measurements. Reduced flow velocity may suggest a low-flow state, particularly when interpreted together with diastolic velocity and PI.⁷ A combination of reduced diastolic flow and increased pulsatility may raise

suspicion of increased downstream resistance or intracranial hypertension.^{3,12} While pressure–flow relationships may provide information about autoregulatory function.^{13–15} Serial assessment adds a further dimension by showing whether these patterns persist, improve, or worsen after intervention.^{9,10,17,18} The value of TCD therefore lies primarily in physiological characterization and reassessment rather than in reproducing the information provided by invasive monitoring.^{3,12}

This distinction is particularly important when invasive monitoring is unavailable or delayed. Current evidence does not support TCD as a replacement for invasive ICP monitoring.^{1,3} A more defensible role is to provide early or provisional physiological guidance: identifying concerning haemodynamic patterns, raising or lowering suspicion of intracranial hypertension, and demonstrating the direction of response to interventions while definitive monitoring is unavailable.^{3,9} This approach is consistent with B-ICONIC, which incorporates TCD as part of multimodal non-invasive assessment rather than as an isolated substitute for invasive ICP monitoring.³ Preliminary studies of early TCD-guided management and resource-limited practice suggest that such information may influence treatment or escalation decisions, but they do not establish improved clinical outcomes.^{9,10,20}

The strength of the evidence also differs across applications. The most direct TBI evidence supports early haemodynamic assessment, screening for intracranial hypertension, and serial assessment of physiological response.^{9,12} Autoregulation testing provides additional physiological information, but available studies mainly support physiological characterization and serial assessment rather than treatment selection.^{14,15} Evidence concerning sedation-related TCD changes is less direct because the propofol–sevoflurane findings were obtained outside a TBI population.¹⁹ These differences should be considered when translating individual TCD findings into clinical decisions. Future studies should move beyond demonstrating associations between TCD parameters and physiological changes. Priorities include standardizing

acquisition and interpretation, defining clinically meaningful reassessment intervals, and testing TCD within structured multimodal treatment algorithms.³ Most importantly, prospective studies should determine whether management informed by serial TCD improves patient-centred outcomes when invasive monitoring cannot be provided.^{1,3} Until such evidence is available, TCD is best positioned as an adjunct for early physiological assessment, provisional decision-making, and serial reassessment rather than as a substitute for invasive monitoring.

Conclusion

TCD can provide useful bedside physiological information during the early management of moderate-to-severe TBI when invasive monitoring is unavailable. Its main strengths are rapid bedside availability, repeatability, and the ability to identify cerebral haemodynamic patterns and track their response to intervention. However, flow velocity is not equivalent to absolute cerebral blood flow, and PI should not be interpreted as a direct measure of intracranial pressure. TCD is therefore most useful when interpreted in clinical context, repeated over time, and combined with other available monitoring. Current evidence supports its use as an adjunct to early goal-directed management, but not as a substitute for invasive monitoring, and it has not yet shown that TCD-guided treatment improves patient outcomes. Prospective studies are needed to determine whether structured TCD-informed management can improve outcomes when invasive monitoring is unavailable.

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