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NONINVASIVE NEUROMONITORING IN TRAUMATIC BRAIN INJURY: FROM EMERGING ALTERNATIVE TO REAL PERSPECTIVE

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Abstract: Traumatic brain injury (TBI) is a major cause of mortality and neurological disability, whose clinical course is largely determined by the development of secondary brain damage. Traditionally, the management of moderate and severe TBI has been based on invasive monitoring of intracranial pressure; however, this approach has limitations in capturing the complexity and dynamics of brain physiology. In this context, noninvasive neuromonitoring has emerged as a complementary strategy aimed at the continuous assessment of different functional domains of the injured brain. This narrative review analyzes the current evidence on noninvasive neuromonitoring in TBI, addressing its pathophysiological basis, main modalities, reference values, clinical utility, and limitations. Tools such as automated pupillometry, transcranial Doppler, measurement of the diameter of the optic nerve sheath, continuous electroencephalography, and near-infrared spectroscopy allow for the early detection of alterations in perfusion, oxygenation, cortical activity, and intracranial compliance. The available evidence supports their usefulness for early detection of neurological deterioration, risk stratification, and dynamic patient monitoring, especially when integrated multimodally and interpreted in trends. Although noninvasive neuromonitoring does not replace invasive monitoring in selected patients, its application represents a real and applicable perspective in contemporary neurocritical care, particularly in resource-limited settings and in the early stages of TBI.

Keywords: traumatic brain injury, neurological monitoring, intracranial pressure.

INTRODUCTION

Traumatic brain injury (TBI) is one of the leading causes of mortality and neurological disability worldwide. Its clinical evolution depends not only on the initial primary damage, but also on the dynamic development of secondary brain damage, characterized by alterations in cerebral perfusion, vascular autoregulation, tissue oxygenation, cortical electrical activity, and intracranial compliance. These processes are often fluctuating and frequently go undetected by intermittent clinical evaluation or conventional neuroimaging(1) .

Invasive monitoring of intracranial pressure (ICP) has traditionally been the mainstay of management for moderate and severe TBI. However, an approach focused on fixed ICP thresholds has significant limitations: it provides only partial information on brain physiology, does not adequately reflect the heterogeneity of the injured brain, and its availability is limited to specialized centers. Furthermore, current evidence questions its isolated impact on clinical outcomes, reinforcing the need for complementary strategies(2) .

In this context, noninvasive neuromonitoring has emerged as a clinically attractive alternative. Modalities such as automated pupillometry, transcranial Doppler, measurement of the optic nerve sheath diameter, continuous electroencephalography, and near-infrared spectroscopy allow for repeated and multimodal assessment of brain function, with lower risk and greater accessibility. Beyond indirect estimation of ICP, these tools provide information on perfusion, oxygenation, and cortical activity, key aspects of secondary damage(3) .

The conceptual shift toward intracranial dynamics and multimodal neuromonitoring models has fueled interest in these techniques, particularly in settings where invasive monitoring is not available. However, questions remain about their validity, standardization, and real impact on clinical decision-making(4) .

This narrative review critically analyzes the current evidence on noninvasive neuromonitoring in TBI, with the aim of determining whether these technologies have transcended their status as emerging tools to establish themselves as a real and applicable prospect in contemporary clinical practice.

Pathophysiological basis of neuromonitoring in traumatic brain injury

Traumatic brain injury (TBI) is a dynamic pathophysiological process in which primary brain damage triggers a cascade of potentially preventable secondary mechanisms. These include cerebral edema, alterations in cerebrovascular autoregulation, regional hypoperfusion, tissue hypoxia, subclinical seizures, and progressive deterioration of intracranial compliance(5) .

The magnitude and interaction of these phenomena vary between patients and over time, limiting the usefulness of point-in-time assessments or single-parameter monitoring.

The classic Monro-Kellie doctrine states that the total intracranial volume consisting of brain parenchyma, blood, and cerebrospinal fluid is constant, so that an increase in one of its components must be compensated by a reduction in another

to avoid increases in intracranial pressure (ICP)(6) .

However, contemporary evidence has shown that this relationship is more complex and dynamic, incorporating concepts such as intracranial compliance, compensatory reserve, and the critical role of venous circulation and the glymphatic system. This conceptual evolution, described as "intracranial dynamics," explains why patients with normal ICP values may present significant neurological impairment and ongoing secondary brain damage(2) .

Cerebrovascular autoregulation is another key pathophysiological pillar. Under normal conditions, the brain maintains relatively constant blood flow in the face of variations in mean arterial pressure. In TBI, this capacity is often partially or completely impaired, causing cerebral blood flow to depend passively on cerebral perfusion pressure. As a result, episodes of hypotension, hypercapnia, or venous congestion can precipitate hypoperfusion or harmful hyperemia, even without obvious elevations in ICP(3) .

Likewise, the dissociation between perfusion, oxygenation, and cerebral metabolism constitutes a central mechanism of secondary damage. Tissue hypoxia can occur in the absence of macroscopic ischemia, due to microvascular alterations, perivascular edema, or mitochondrial dysfunction. In parallel, alterations in cortical electrical activity, including nonconvulsive epileptic seizures and propagated cortical depression, increase metabolic demand and aggravate energy imbalance(4) .

In this context, neuromonitoring becomes relevant not as the isolated measurement of a single value, but as a tool for exploring different domains of brain physiology: pressure, flow, oxygenation, metabolism, and neuronal function. Noninvasive neuromonitoring relies on these pathophysiological principles to provide indirect and complementary estimates of intracranial status, allowing for the identification of trends, detection of early deterioration, and characterization of physiological risk phenotypes(7) .

Therefore, the justification for noninvasive neuromonitoring in TBI lies in its ability to capture the complexity and variability of secondary brain damage, especially in scenarios where invasive monitoring is unavailable or insufficient to reflect the entirety of intracranial dynamics.

Principles of noninvasive neuromonitoring

Noninvasive neuromonitoring in traumatic brain injury (TBI) is based on the recognition that secondary brain damage is a dynamic, multifactorial, and heterogeneous process that cannot be adequately characterized by isolated measurements or single parameters. In this context, the clinical value of noninvasive neuromonitoring lies not in directly replacing invasive monitoring, but in its ability to complement neurological assessment and broaden the functional understanding of the injured brain(8) .

A central principle is interpretation based on trends rather than absolute values. Noninvasive modalities exhibit interindividual variability and depend on technical and operator factors; therefore, sequential changes over time, especially in response to therapeutic interventions, offer greater clinical relevance than an isolated cutoff point. This approach allows for the detection of

early neurological deterioration, evaluation of the effectiveness of therapeutic measures, and anticipation of intracranial decompensation events(9) .

Another fundamental principle is multimodal integration. Each noninvasive neuromonitoring technique explores a specific domain of brain physiology—pressure, flow, oxygenation, or electrical activity—and, in isolation, provides incomplete information. The combination of several modalities allows for a more comprehensive approach to the intracranial state, facilitating the physiological phenotyping of patients with TBI and the identification of predominant mechanisms of secondary damage, such as hypoperfusion, venous congestion, tissue hypoxia, or cortical hyperexcitability(10) .

Clinical contextualization is another essential pillar. Non-invasive neuromonitoring findings must always be interpreted in conjunction with neurological examination, neuroimaging, hemodynamic and respiratory status, and the stage of TBI progression. Without this integration, there is a significant risk of overinterpretation or inappropriate therapeutic decisions based on partial data(11) .

Noninvasive neuromonitoring also plays a strategic role as a screening and follow-up tool. In settings where invasive monitoring is not available, these techniques allow the identification of patients at high risk of neurological deterioration who could benefit from diagnostic or therapeutic escalation. In units with access to invasive neuromonitoring, its usefulness focuses on continuous monitoring, assessment of intracranial dynamics, and detection of physiological changes not reflected by isolated intracranial pressure(2).

Finally, non-invasive neuromonitoring aligns with a personalized, physiology-oriented model of care, in which clinical decisions are based on individual patient characterization rather than population thresholds. From this perspective, its real value depends less on the absolute accuracy of each technique and more on its correct integration into a multimodal, systematic, and clinically contextualized strategy.

Modalities of noninvasive neuromonitoring in traumatic brain injury

Non-invasive neuromonitoring allows different domains of brain physiology to be assessed using quantifiable parameters. Although no technique completely replaces invasive monitoring, the availability of reference values facilitates its clinical integration, especially when interpreted in terms of trends and pathophysiological context(1) .

➢ Automated pupillometry

Automated pupillometry objectively quantifies the pupillary response using dynamic parameters, reducing the variability of manual clinical examination. The most widely used index is the Neurological Pupil Index (NPi), calculated from multiple pupillary variables.

Reference values and warning points:

- Normal NPi: 3.0–5.0
- Abnormal NPi: < 3.0
- Critically low NPi: ≤ 2.0 (associated with high risk of neurological deterioration)

- Significant pupillary asymmetry: NPi difference ≥ 0.7 between both eyes

A progressive decrease in NPi or a sudden drop is associated with intracranial hypertension, imminent herniation, and a worse functional prognosis, even before obvious changes are seen on computed tomography. Pupillometry is especially useful for serial monitoring and early detection of brainstem deterioration(11) .

➢ Transcranial Doppler (TCD)

Transcranial Doppler indirectly assesses cerebral blood flow by measuring velocities in intracranial arteries, mainly the middle cerebral artery (MCA).

- Usual reference values (MCA):
- Normal mean velocity: 40–80 cm/s
- Hypo-perfusion: < 40 cm/s
- Hyperemia: > 100 cm/s
- Probable vasospasm: > 120–200 cm/s (depending on context)

Pulsatility index (PI):

- Normal: 0.6–1.1
- Elevated (>1.2–1.3): suggests increased distal resistance, decreased intracranial compliance, or intracranial hypertension
- Very low (<0.5): associated with hyperemia or loss of autoregulation

In TBI, an elevated PI together with decreased diastolic velocities is suggestive of cerebral hemodynamic compromise and has been correlated with a worse prognosis.

➢ Optic nerve sheath diameter (ONSD)

Ultrasound measurement of the optic nerve sheath diameter is usually performed 3 mm behind the eyeball, where distension is most sensitive to changes in intracranial pressure.

Reference values in adults:

- Normal: ≤ 5.0 mm
- Grey zone: 5.0–5.7 mm
- Suggestive of intracranial hypertension: ≥ 5.7–6.0 mm

In patients with TBI, values ≥ 5.7 mm have been associated with intracranial pressure > 20– 22 mmHg in multiple studies. However, its main use is as a screening tool and for monitoring trends, rather than as a substitute for invasive monitoring(3) .

➢ Continuous and quantitative electroencephalography (EEG/cEEG)

Continuous EEG allows the detection of subclinical electrical alterations that increase cerebral metabolic demand and aggravate secondary damage.

Clinically relevant findings:

- Non-convulsive epileptic seizures: present in 15–30% of moderate to severe TBI
- Periodic or rhythmic patterns: associated with a worse prognosis
- Absence of EEG reactivity: independent marker of poor functional outcome

Dynamic EEG changes may precede clinical and radiological deterioration(11) .

➢ Near-infrared spectroscopy (NIRS)

NIRS measures regional cerebral oxygen saturation ($rScO_2$), reflecting the balance between oxygen supply and consumption in superficial cortical tissue.

Guideline values:

- Normal $rScO_2$: 55–75%
- Relative cerebral hypoxia: < 55%
- Frequent clinical alarm: < 50% or drop > 20% from baseline

Given its regional nature and extracranial influence, NIRS is more useful for detecting sustained downward trends or significant asymmetries between hemispheres than for decisions based on an isolated absolute value(2).

➢ Emerging technologies: noninvasive ICP waveform morphology and rheoencephalography

Noninvasive analysis of intracranial pressure wave morphology assesses intracranial compliance through the relationship between pulsatile wave peaks.

P2/P1 ratio:

- Normal: < 1.0
- Compliance impairment: ≥ 1.0
- High probability of intracranial hypertension: ≥ 1.2

Recent studies have demonstrated a correlation between an elevated P2/P1 ratio, intracranial hypertension, and increased early mortality, even with invasive ICP values in the borderline range.

Rheoencephalography assesses impedance changes related to cerebral blood volume. Although promising, its use remains experimental, with values not yet standardized(7) .

Clinical evidence of noninvasive neuromonitoring in traumatic brain injury

Available clinical evidence suggests that non-invasive neuromonitoring adds value in the early detection of neurological deterioration, prognostic stratification, and dynamic physiological monitoring of patients with TBI. Although most studies are observational and heterogeneous, the findings consistently point to its usefulness as a complement to, rather than a substitute for, invasive monitoring(4) .

Early detection of neurological deterioration. Changes in non-invasive parameters precede clinical or radiological decompensation. A decrease in NPi in automated pupillometry, an increase in the pulsatility index in transcranial Doppler, and a progressive increase in the diameter of the optic nerve sheath are associated with imminent intracranial hypertension and the need for therapeutic escalation. These findings are particularly relevant in emergency departments and units without immediate access to invasive monitoring(7) .

Prognostic value. Multiple studies have identified associations between noninvasive parameters and clinical outcomes. A persistently low NPi, absence of reactivity on continuous EEG, reduced diastolic velocities on transcranial Doppler, and an elevated P2/P1 ratio in noninvasive intracranial pressure wave analysis have been correlated with higher early mortality, longer duration of mechanical ventilation, and worse function-

Modality	*Physiological domain assessed*	*"Normal"/ reference value (adult)*	*Alarm threshold (guideline)*	*Main clinical utility in TBI*
Automated pupillometry (NPi)	Brainstem function and pupillary pathway (dynamic photomotor reflex)	**NPi 3.0–5.0**	**NPi < 3.0** (abnormal); **NPi ≤ 2.0** (high risk). **Asymmetry**: ΔNPi ≥ 0.7	Early detection of neurological deterioration/imminent herniation; objective serial monitoring; prognostic stratification (trends).
Transcranial Doppler (TCD) – ACM	Cerebral hemodynamics (flow/velocity), indirect autoregulation, distal resistance	**Vm 40–80 cm/s**; **PI 0.6–1.1**	**Hypo-perfusion**: Vm < 40 cm/s; **Hyperemia**: Vm > 100 cm/s; **PI > 1.2–1.3** suggests ↑resistance/↓compliance; **PI < 0.5** suggests hyperemia/loss of autoregulation	Identify hemodynamic phenotypes (hypoperfusion vs. hyperemia); guide optimization of PPC/MAP, $PaCO_2$, and volume; monitor vasospasm and intracranial dynamics for trends.
Optic nerve sheath diameter (ONSD)	Indirect estimation of intracranial pressure and CSF dynamics	**≤ 5.0 mm**	**5.0–5.7 mm** (gray area); **≥ 5.7–6.0 mm** suggests ICH (associated with ICP >20–22 mmHg)	Rapid screening for possible ICH in the emergency room/ICU; trend monitoring (response to anti-ICH therapies); prioritize neuroimaging/mon nvasive monitoring when appropriate.
Continuous/ quantitative EEG (cEEG/qEEG)	Cortical function: electrical activity, reactivity, subclinical seizures	No "single value"; **reactive EEG** without ictal patterns is favorable	**Non-convulsive seizures** (≈15–30% in moderate-severe TBI); **absence of reactivity**; persistent periodic/rhythmic patterns	Detect subclinical seizures and guide anticonvulsants; assess sedation and prognosis (reactivity); anticipate physiological deterioration before clinical/radiological changes.
NIRS ($rScO_2$)	Superficial cortical regional oxygenation (supply/consumption balance)	**55–75%**	**< 55%** relative hypoxia; **< 50%** frequent alarm; **↓ >20%** vs baseline or sustained asymmetry	Monitoring of regional oxygenation trends; detect episodes of cortical desaturation/hypoperfusion; support decisions on ventilation, Hb, MAP/CPP, and $PaCO_2$.

Noninvasive ICP wave morphology (P2/P1)	Intracranial compliance/compensatory reserve (pulsatile dynamics)	**P2/P1 < 1.0**	**P2/P1 ≥ 1.0** (↓compliance); **≥ 1.2** (high probability of ICH/ worse prognosis)	Estimate compliance impairment even with "borderline" ICP; monitor response to anti-IHC measures; early prognostic support (trends).
Rheoencephalography (REG) (emerging)	Impedance changes linked to cerebral blood volume (global hemodynamics)	Not standardized	Not standardized (experimental use)	Potential for continuous monitoring of cerebral hemodynamic dynamics; currently under investigation/ validation, not for isolated decisions.

Table 1. Noninvasive neuromonitoring

al outcome. Although these associations do not establish causality, they reinforce their usefulness for risk stratification(8) .

Comparison with invasive monitoring. When evaluated against invasive parameters, several noninvasive techniques show moderate but clinically relevant correlations. Optic nerve sheath diameter and transcranial Doppler have demonstrated the ability to identify intracranial hypertension, while noninvasive analysis of intracranial pulse wave morphology is associated with intracranial compliance and outcomes, even in patients with borderline invasive intracranial pressure values. These findings support their role as screening and dynamic monitoring tools(10) .

Impact on decision-making. The available evidence indicates that noninvasive neuromonitoring mainly influences decisions regarding surveillance, diagnostic prioritization, and early therapeutic adjustment. Its use has been associated with earlier detection of non-convulsive seizures, Doppler-guided hemodynamic optimization, and early recognition of brainstem deterioration through pupillometry, although a robust demonstration of a direct impact on hard outcomes is still lacking(11) .

Specific clinical contexts. The greatest benefit is seen in resource-limited settings, in the early stages of TBI, and as a complement in units with invasive monitoring available. In these contexts, noninvasive neuromonitoring expands physiological surveillance and reduces reliance on intermittent assessments alone(12)allowing for early detection of complications such as increased intracranial pressure (ICP .

Limitations, challenges, and knowledge gaps

Despite the growing interest and expansion of non-invasive neuromonitoring in traumatic brain injury (TBI), its clinical implementation faces methodological, operational, and conceptual limitations that condition its widespread adoption and impact on clinical outcomes(13) .

Technical limitations and operator dependence. Several noninvasive modalities, such as transcranial Doppler and measurement of the optic nerve sheath diameter, are highly dependent on the operator and

technical quality, which introduces interobserver and interinstitutional variability. This characteristic limits the reproducibility of results and hinders the standardization of universally accepted clinical thresholds.

Methodological heterogeneity. The available evidence is predominantly observational, with heterogeneous designs, mixed populations, and variable outcomes. The reported cutoff points differ between studies and, in many cases, are extrapolated from small cohorts or specific contexts, which reduces their external validity. Furthermore, the absence of uniform acquisition and interpretation protocols limits the comparison between studies(8) .

Gap between correlation and causality. Although multiple noninvasive parameters are associated with neurological deterioration and worse prognosis, these associations do not demonstrate causality or confirm that interventions guided by these findings improve clinical outcomes(9) .

Clinical integration and cognitive load. Multimodal interpretation requires experience, specific training, and clinical time, which can increase the cognitive load on the healthcare team. Without systematic integration into clear algorithms, there is a risk of overinterpretation, unnecessary interventions, or contradictory decisions(10) .

Limitations in hard outcomes. To date, there is no robust evidence demonstrating that the isolated or combined use of noninvasive neuromonitoring reduces mortality or consistently improves long-term functional outcomes in TBI. This gap is the main challenge to its consolidation as a standard of care(11) .

From emerging alternative to real prospect

Noninvasive neuromonitoring in traumatic brain injury (TBI) has undergone a significant conceptual transition over the last decade. Initially considered a set of auxiliary or screening tools, its technological evolution and growing body of evidence have allowed its role in contemporary neurocritical care to be redefined. However, this transition is not uniform and depends on the clinical context, available resources, and how these technologies are integrated into decision-making(14) .

From a practical perspective, several non-invasive modalities have demonstrated immediate clinical utility. Automated pupillometry, transcranial Doppler, and non-invasive analysis of intracranial wave morphology provide reproducible, accessible, and relevant information for the dynamic monitoring of patients with TBI. Their ability to detect early neurological deterioration, identify alterations in intracranial compliance, and provide functional data beyond absolute intracranial pressure supports their incorporation as complementary tools in daily practice(15) .

However, non-invasive neuromonitoring should not be interpreted as a direct replacement for invasive monitoring in selected patients with moderate or severe TBI. Its greatest value is evident in specific scenarios: early stages of TBI, units without access to invasive monitoring, serial monitoring of intracranial dynamics, and physiological contextualization of borderline invasive values. In these contexts, its use helps reduce exclusive dependence on clinical and intermittent neuroimaging(12).

The "real perspective" of non-invasive neuromonitoring lies in its functional integration, rather than in the absolute accuracy of each measurement. When used in a multimodal, trend-oriented manner and linked to specific clinical decisions, these technologies allow for a more personalized approach to TBI, aligned with current models of intracranial dynamics and physiology-based medicine.

Future perspectives

The development of non-invasive neuromonitoring in traumatic brain injury (TBI) is currently in a phase of technological and conceptual consolidation, with a focus on automation, data integration, and personalized medicine. The most relevant areas of progress are centered on improving physiological accuracy, reducing operator dependence, and demonstrating clinical impact through well-designed prospective studies(12)allowing for early detection of complications such as increased intracranial pressure (ICP .

One of the main future prospects is the integration of artificial intelligence and machine learning for the continuous analysis of neuromonitored signals. These systems will enable the processing of large volumes of multimodal data in real time, the identification of subclinical patterns of neurological deterioration, and the generation of predictive alerts before the clinical or radiological manifestation of secondary damage. This approach can transform neuromonitoring from a reactive to a preventive model(16) .

Likewise, greater standardization of devices and metrics is expected, particularly in emerging technologies such as noninvasive analysis of intracranial wave morphology and rheoencephalography. Multicenter validation and the definition of agreed-upon physiological thresholds will facilitate their clinical adoption and comparison between studies(13) .

Another key line of research is the development of pragmatic clinical trials, focused not only on the diagnostic capacity of these tools, but also on their impact on therapeutic decisions and functional outcomes. The use of intermediate physiological endpoints, combined with long-term clinical results, will be essential to demonstrate their true value.(17)

Finally, noninvasive neuromonitoring has the potential to expand access to advanced neurocritical care in resource-limited settings, helping to reduce inequalities in TBI care. Its incorporation into stepwise protocols and simplified algorithms can facilitate broader and more timely brain monitoring.

Overall, the future prospects for noninvasive neuromonitoring point toward an integrated, automated, physiology-oriented model in which these technologies play a central role in preventing secondary brain damage and personalizing the management of traumatic brain injury.

CONCLUSIONS

Non-invasive neuromonitoring in traumatic brain injury has established itself as a clinically useful tool for the early detection of neurological deterioration and the dynamic assessment of secondary brain damage. By allowing the assessment of key physiological domains such as intracranial compliance, cerebral perfusion, tissue oxygenation, and cortical electrical activity,

these techniques provide complementary information that is not always captured by invasive intracranial pressure or conventional neuroimaging, especially when interpreted in a multimodal and trend-based manner.

Although noninvasive neuromonitoring does not replace invasive monitoring in selected patients with moderate or severe TBI, its value as a complement is evident, particularly in the early stages of injury and in resource-limited settings. Despite methodological limitations and the need for more evidence on its impact on hard clinical outcomes, current evidence indicates that these technologies have moved beyond their status as emerging alternatives to become a real and applicable prospect in contemporary neurocritical care.

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