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REVIEW ARTICLE

Noninvasive bedside neuromonitoring in acute brain injury. A narrative review

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Intracranial pressure;
Waveform analysis

Abstract Clinical neurological examination remains the gold standard to detect, diagnose, and follow-up responses to treatment in acute neurological conditions in the critical care setting. However, in patients with severe neurological deficits at baseline or those requiring sedatives, detecting neurological deterioration can be challenging. In this scenario, noninvasive bedside neuromonitoring as a part of multimodal strategies can be useful in the avoidance of secondary brain injury and in the selection of which patient with acute brain injury would benefit from invasive neuromonitorization.
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PALABRAS CLAVE
Lesión cerebral aguda;
Neuromonitorización no invasiva;
Cuidados neurocríticos;

Neuromonitorización no invasiva a pie de cama en la lesión cerebral aguda. Una revisión narrativa

Resumen El examen neurológico clínico sigue siendo el patrón oro para detectar, diagnosticar y realizar el seguimiento de las respuestas al tratamiento en el paciente neurocrítico. Sin embargo, en pacientes con déficits neurológicos graves al inicio o en aquellos que requieren sedantes, detectar el deterioro neurológico puede ser un reto. En este escenario, la

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Ecografía transcraneal;
Espectroscopia del infrarrojo cercano;
Pupilometría automatizada;
Electroencefalograma;
Presión intracraneal;
Análisis de forma de onda

neuromonitorización no invasiva a pie de cama como parte de estrategias multimodales puede ser útil para evitar lesiones cerebrales secundarias y para seleccionar qué paciente con lesión cerebral aguda se beneficiaría de una neuromonitorización invasiva.
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Introduction

Clinical neurological examination remains the gold standard to detect, diagnose, and follow-up responses to treatment in acute neurological conditions in the critical care setting.[1] However, in patients with severe neurological deficits at baseline or those requiring sedatives, detecting neurological deterioration can be challenging.[1] In this context, neuromonitoring techniques should be applied to support strategies directed to preserve neurological functions and avoid secondary brain injury.[2,3]

Invasive neuromonitoring, including intracranial pressure, brain tissue O_2 and cerebral microdialysis probes are commonly used in specialized centers in the context of multimodal monitoring.[2,4,5] However, there is a huge variability in their use worldwide[6] and in addition, different real-life studies[7] and randomized controlled trials[8] have shown none-to-little beneficial clinical effects, suggesting that invasive neuromonitoring is not necessary in all neurocritically ill patients. In this scenario, noninvasive neuromonitoring as a part of integral strategies[9–12] can be useful in the avoidance of secondary brain injury and in the selection of which patient with acute brain injury (ABI) would benefit from invasive neuromonitorization.[9]

In this manuscript, we review the available noninvasive, bedside, neuromonitoring techniques in the management of patients with ABI following a narrative practical approach.

Noninvasive, bedside, neuromonitoring techniques

We searched published journal articles from PubMed using the following MeSH terms «Acute Brain Injury» [MeSH] OR «Traumatic Brain Injury» [MeSH] OR «Intracerebral Hemorrhage» [MeSH] OR «Aneurysmal Subaracnoid Hemorrhage» [MeSH] OR «Status Epilepticus» [MeSH] OR «Stroke» [MeSH] OR «Cardiac Arrest» [MeSH]) OR «Intracranial Pressure» [MeSH] OR «Intracranial Hypertension» [MeSH] OR «Neuroimaging» [MeSH] OR «Neuromonitoring». The search was not limited per year but was limited to english or spanish language articles. Clinical articles, reviews and opinion papers were selected by all authors. In addition, the reference lists of all the retrieved articles were carefully examined to identify any further relevant manuscripts.

The objective of this comprehensive review was to outline the current applications of noninvasive, bedside, neuromonitoring techniques in patients with ABI, discussing its limitations and suggesting potential areas of improvement.

Transcranial ultrasound

Transcranial doppler (TCD) allows the study of the hemodynamics of the cerebral vessels. The first report was documented in 1982 by Aslid et al.[13] Technical improvements led to the capacity to analyze cerebral parenchyma in addition to the color visualization of the basal venous and arterial vessels and doppler spectral analysis, providing a noninvasive, bedside examination using transcranial color-coded duplex (TCCD) protocols.[14,15] TCCD is usually performed using a 2–2.5 MHz probe[15] and remains the most widely used nonivasive technique at bedside.[16]

In its basic form, the *bidimensional* mode allows the evaluation of midline shift and the size of the ventricular system.

To evaluate midline shift (MLS), the examination is performed from the temporal window, placing the ultrasound probe perpendicular to the walls of the third ventricle (3V), which appears as a double hyperechoic line. Then, we measure the distance from the transducer to the center of the 3V bilaterally (distances A and B). The following formula is applied: MLS = A−B/2. Whilst this can be applied to all ABI patients, it is specially useful in the follow-up of patients with malignant middle cerebral artery (MCA) stroke.[17,18]

The 3V appears in the diencephalic plane as a double hyperechoic line. The frontal horns of the lateral ventricles are then visualized after angulating the probe cephalad 10 degrees. Studies found an excellent correlation between computed tomography (CT) and TCCD in measuring the diameter of the 3V and the frontal horns,[19,20] allowing to assess the response to cerebrospinal fluid (CSF) drainage.[21]

Using the *doppler spectra* analysis the main indications of TCCD are listed as follows:

Cerebral vasospasm

The sensitivity, specificity and positive and negative predictive values in the diagnosis of cerebral vasospasm of the MCA are 66.7%, 89.5%, 93.7% and 53.4% for TCD and increase to 81.5%, 96.6%, 98.2% and 69.1% for TCCD.[22] The most commonly used cut-off points in the diagnosis of cerebral vasospasm according to mean MCA velocities are 120 cm/s and 200 cm/s, with the former being more sensitive and the latter more specific. These values are complemented by the measurement of the Lindegaard ratio (LR), the ratio between the mean velocity in the MCA and the ipsilateral internal carotid artery in its extracranial portion. This ratio allows to differentiate situations of hyperafflux (LR < 3) and severe vasospasm (LR > 6).[14,15] Of note, the evidence suporting the role of TCD/TCCD in the anterior and posterior cerebral arteries remains very limited.[15,23]

Cerebral autoregulation

TCD/TCCD allows the analysis of the static and dynamic components of autoregulation. The static rate of regulation evaluates the response of cerebral blood flow (CBF) to changes in cerebral perfusion pressure (CPP), and is measured as the ratio between the percentage change in cerebrovascular resistance (CVR) and the percentage change in CPP, where CVR = mean blood pressure/flow velocity.[24] The evaluation of the dynamic autoregulation can be made using the transient hyperemia response test, based on the compensatory arteriolar vasodilatation occuring after a brief external compression of the common carotid artery. In patients with preserved autoregulation, systolic velocity increases over 9% after carotid compression. It can also be evaluated by analyzing the spontaneous changes in blood pressure and CPP over time while measuring CBF velocity.[15]

Cerebral circulatory arrest

Cerebral circulatory arrest (CCA) occurs as a final process in patients with devastating brain injury and intracranial hypertension. Therefore, the sonographic patterns follow the evolutive changes of the increase in intracranial pressure (ICP), characterized by in crescendo high resistance patterns until the definitive cessation of CBF occurs. It is necessary to evaluate both MCA and the basilar artery (BA) in two examinations. A recent meta-analysis showed that transcranial sonography presented a sensitivity of 90% and a specificity of 98% in diagnosing CCA.[25]

The following evolutive patterns suggesting CCA are:

a) Reverberant or oscillating flow, represented by a sonogram exhibiting anterograde systolic flow with retrograde or inverted diastolic flow.
b) Systolic spikes, represented by short systolic waves (<200 ms) with low velocity (systolic peak < 50 cm/s) in the absence of any diastolic flow.
c) Lack of acoustic signal, which can be accepted only when a prior TCD/TCCD has previoulsy shown adequate acoustic window and preserved flow of the basal arteries.

Screening of intracranial hypertension. The use of transcranial ultrasound in the screening of intracranial hypertension (ICH) is particularly interesting in situations characterized by limited availability of invasive monitoring and as screening to select which neurocritical care patients really necessitates invasive monitoring.[9] Of note, the recent Brussels consensus stated that a multimodal approach including different noninvasive ICP methods (at least 2 different modalities) is necessary compared to a single method for ICP assessment.[9] It can be evaluated in different ways.

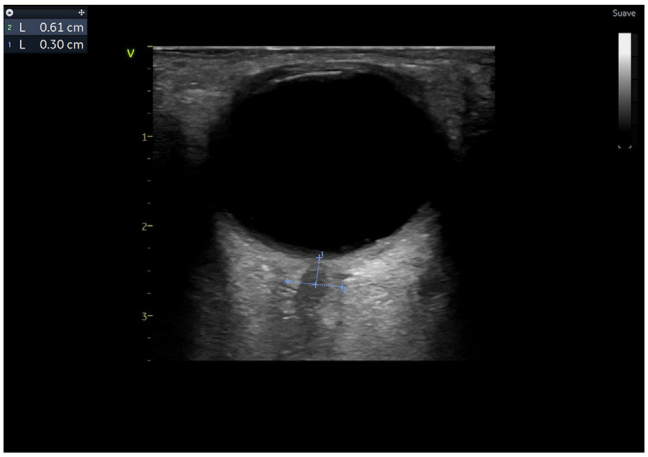

Figure 1 Measurement of the diameter of the optic nerve sheath to rule out significant intracranial hypertension. The patient had a ONSD of 6.1 mm and ICP 25 mmHg at the time the examination was performed.

Measurement of the diameter of the optic nerve sheath (ONSD). The optic nerve is covered by the meninges, as an extension of the cerebral subarachnoid and subdural space. Thus, when ICP increases, the cerebrospinal fluid displaces towards this space, increasing the diameter of the optic nerve particularly in the bulbous portion, which is more distensible and is located 3 mm from the retina and the optic disc.[3,14,15] This is the point where the ONSD must be measured (Fig. 1). Criteria for a high-quality and standardized examination have been recently developed through an international consensus.[26]

A recent meta-analysis including five hundred and forty-eight adult patients and 120 children in 16 eligible studies showed that pooled sensitivity and specificity of ONSD measurement by ultrasonography were 84% (95% CI 76%–89%) and 83% (95% CI 73%–90%), respectively.[27] Pooled area under the curve was 0.91 for adults and 0.76 for children. Optimal threshold for estimating ICH was 5.76 mm for adults and 5.78 mm for children.[27] The recent Brussels consensus recommended considering a threshold of 6 mm as a potential marker of ICH or excluding it (Strong Recommendation).[9]

Intracranial pressure according to cerebral hemodynamics. The pulsatility index (PI) has been the single most widely used hemodynamic parameter for evaluating the existence of ICH, since the circulatory flow resistances increase with rising ICP. However, PI depends on multiple physiological variables and its ability to determine increases of ICP is therefore limited.[15] The Brussels consensus recommended that to raise/exclude the suspiction of ICH, a change from basal level of PI of at least 0.5 should be considered as a sign of cerebral blood flow modification potentially related to ICP shift (Strong Recommendation).[9]

Recently, the multicenter IMPRESSIT-2 trial analyzed the estimation of ICP through insonation of the MCA in 262 neurocritical patients using the following formula and comparing it against the invasive measurement[28]:

$$\text{ICPtcd} = \text{MABP} - \text{CPPtcd; where CPPtcd} = \text{MABP} \times \text{Vd/Vm} + 14,$$

being: ICPtcd: transcranial ultrasound measurement of intracranial pressure; MABP: mean arterial blood pressure; CPPtcd: transcranial ultrasound measurement of cerebral perfusion pressure; Vd/Vm: diastolic/mean flow velocity in the middle cerebral artery.

The negative predictive value was elevated (ICP > 20 mmHg = 91.3%, >22 mmHg = 95.6%, >25 mmHg = 98.6%), indicating high discriminant accuracy of ICPtcd in excluding ICH. Concordance correlation between ICPtcd and invasive ICP was 33.3% (95% CI 25.6%–40.5%), and Bland-Altman showed a mean bias of −3.3 mmHg.[28]

Near-infrared spectroscopy (NIRS)

Brain tissue is considered one of the most sensitive to hypoxic insults, with hypoxia representing a key mechanism of secondary brain damage. However, real-world data in our environment showed that cerebral oxygenation monitoring remains infrequently used in the neurocritical care setting.[29] Near-infrared spectroscopy (NIRS) technology offers numerous advantages, including availability, potential early implementation, the capability to monitor multiple brain regions, and the absence of procedure-related complications.[30] The physical principles underlying NIRS rely on the presence of specific substances known as chromophores, which absorb light within a defined wavelength spectrum (700–1300 nm, near infrared light). Blood contains a variety of these chromophores, notably oxyhemoglobin and deoxyhemoglobin, which absorb light in the near infrared range.[30] By applying the Beer-Lambert law, NIRS devices can estimate the proportion of oxyhemoglobin and deoxyhemoglobin within the targeted cerebral tissue. Key parameters derived from this technology include the regional oxygen saturation (rSO2) and the tissue oxygenation index (TOI), both of which reflect an estimation of the percentage of oxygenated hemoglobin. The rSO2 is calculated as follows:

$$\text{HbO2}/(\text{HbO2} + \text{HHb}) \times 100\%$$

The most commonly used configuration involves two adhesive patches containing both emisors and detectors, typically placed on the skin over the frontal lobes. It is important to note that NIRS cannot distinguish between different vascular compartments, providing a mixed measurement of arterial, venous, and capillary saturations. Given that the majority of cerebral blood volume is in the venous compartment, its oxygen saturation is the predominant contributor to the rSO2 value. Normal rSO2 value is considered to range from 60% to 75%, although significant variability exists. Emphasis should be placed on monitoring trends rather than interpreting isolated values. Thresholds such as rSO2 < 55% or a 10%–20% decrease from baseline have been associated with poorer outcomes in patients with ABI.[31] Detector placement is also critical, particularly in the presence of suspected intracranial lesions such as pneumocephalus, fractures or hematomas. In traumatic brain injury (TBI), NIRS has demonstrated utility in the detection of intracranial hematomas.[32] Additionally, it has shown potential for optimizing CPP and evaluating cerebral autoregulation. Some studies have demonstrated good sensitivity for detecting severe desaturation episodes (brain tissue O2 < 12 mmHg), but its performance is less reliable for moderate events [33]. In aSAH, a recent study reported a good correlation between the extent of hemorrhage and the frequency and duration of cerebral desaturation episodes (rSO2 < 60%).[34] Additionally, NIRS could be useful for detection of delayed ischemia and maintenance of CPP in aSAH patients.[35]

However, NIRS presents limitations.[36] First, extracranial blood contamination has been documented as a potential source of measurement error. Second, no absolute treatment thresholds have been established, and inter- and intra-individual variability remains significant. Moreover, it provides data from only a few centimeters of cortical tissue. Finally, NIRS monitoring has not been consistently correlated with improved patient outcomes.

Automated pupillometry

Automated quantitative pupillometry allows for objective and accurate testing of the pupil light responses. Pupillometer devices deliver a precisely defined light stimulus and capture different variables such as maximum size, minimum size, constriction velocity, constriction amplitude, and response latency.[2] Dynamic pupil variables can be integrated in algorithm-derived indices such as the Neurological Pupil index (NPI).[2] It can be considered a validated monitor of brain function.[3]

To date, the most relevant study in patients with ABI was the ORANGE, a prospective, observational cohort study conducted at 13 hospitals in eight countries in Europe and North America.[37] The ORANGE included 514 patients (224 with TBI, 139 with aSAH and 151 with intracerebral hemorrhage). The 6-month outcome was assessed in 497 (97%) patients, of whom 160 (32%) patients died, and 241 (47%) patients had at least one recording of abnormal NPi, which was associated with poor neurological outcome (for each 10% increase in the frequency of abnormal NPi, adjusted odds ratio 1·42 [95% CI 1.27–1.64]; $P < .0001$) and in-hospital mortality (adjusted hazard ratio 5·58 [95% CI 3.92–7.95]; $P < .0001$). Therefore, automatic, repeated automated pupillometry assessment could improve the continuous monitoring of ABI progression and the dynamics of outcome prediction.[37]

However, despite initial enthusiasm,[38,39] the role of automated pupillometry in the noninvasive prediction of ICH was evaluated in a secondary analysis of the ORANGE cohort.[40] This secondary analysis included 318 adult patients who required intensive care unit admission, intubation, and mechanical ventilatory support due to acute TBI (n = 133 [41.8%]), intracerebral hemorrhage (n = 104 [32.7%]), or aSAH (n = 81 [25.5%]) and had automatic pupillometry used as part of the standard evaluation practice and ICP

Table 1 Potential use of Continuous EEG in neurocritical care.

General applications	Diagnosing disorders of consciousness
	Sedation titrating
	Neuroprognostication
	Identify impending brain tissue ischemia
Seizures and Status Epilepticus	Diagnosing nonconvulsive status epilepticus
	Identify rhythmic and periodic patterns
	Detect electroclinical improvement after treatment
	Dose adjustment and guide de-escalation of antiseizure medication
Post-cardiac arrest brain injury	Outcome prediction
Traumatic brain injury	Monitoring induced burst suppression
	Long-term clinical outcomes prognostication
Aneurysmal subarachnoid hemorrhage	Early detection of delayed cerebral ischemia
Future challenges	Improve general implementation and critical caregiver training
	Alternative techniques like quantitative EEG or intracranial EEG

monitoring.[39] No significant association between ICP and abnormal NPi (<3; odds ratio, 1.01; 95% CI, 0.99–1.03) or absent NPi (0; odds ratio, 1.03; 95%CI 0.99–1.06) was observed.[40] Therefore, in that heterogeneous population of ABI patients, the NPi values were not significantly associated with ICP values and repeated NPi measurements may not be a sufficient replacement for invasive monitoring.[40] However, the recent Brussels consensus recommended a NPi < 3 to be used (in combination with at least another tool) to suspect the presence of elevated ICP, and NPi $\geq$ 4 to rule out the presence of elevated ICP.[9]

Additionally, automated pupillometry and specifically, the NPI proved to be a promising tool in the screening of delirium,[41] need of intense neurocritical care,[42] and identifying covert consciousness.[43] More established is its role in the multimodal approach to neuroprognostication after cardiac arrest.[44] In a recent validation study including 710 survivors after out-of-hospital cardiac arrest, a NPi $\leq$ 2 predicted outcome with 0% false-positive rate (FPR) at all time points (0—72 h), and qPLR < 4% at 24—72 h. In patients with motor GCS $\leq$ 3 at $\geq$72 h, pupillometry thresholds significantly increased the sensitivity of neuron-specific enolase, from 42% (35%–51%) to 55% (47%–63%) for qPLR and 50% (42%–58%) for NPi, maintaining 0% (0%—0%) FPR.[45]

Electroencephalogram (EEG) and bispectral index scale (BIS)

The electroencephalogram (EEG) is a pivotal tool in neurocritical care, utilized primarily to assess cerebral electrical activity, detect seizures, and monitor brain function in critically ill patients.[46] EEG records spontaneous electrical activity generated by cortical neurons through electrodes placed on the scalp. It provides real-time information on neuronal function, enabling early detection of subtle brain dysfunctions, especially non-convulsive seizures or status epilepticus (SE).[2,3,46,47] Potential applications of EEG in neurocritical care are summarized in Table 1.

Continuous EEG (cEEG) monitoring has gained importance due to its sensitivity in detecting electroencephalographic abnormalities, which may remain undetected with intermittent EEG assessments.[48] However, the implementation of cEEG remains inconsistent. A Canadian multicentre observational study involving medical, surgical, trauma, and neurological ICUs revealed that out of 375 screened patients, 34% met the criteria for cEEG monitoring recommended by the European Society of Intensive Care Medicine. Yet, 63% of those patients did not receive cEEG monitoring. This included 20% of SE patients without return to baseline, 67% of intracerebral hemorrhage patients with altered consciousness, and 100% of patients undergoing targeted temperature management after cardiac arrest.[49] In addition, cEEG has been associated with reduced in-hospital mortality among mechanically ventilated patients and critically ill hospitalized patients.[50] Indeed, in patients with suspected nonconvulsive SE, initial cEEG may be necessary to support the diagnosis by identifying early sporadic epileptiform discharges and early rhythmic or periodic EEG patterns of ''ictal-interictal uncertainty''.[51]

Simplified quantitative methods such as the Bispectral Index Scale (BIS) have emerged as a form of processed EEG (pEEG). BIS, initially developed in the field of anesthesiology, processes raw EEG signals through advanced algorithms analyzing amplitude, frequency, and bispectral characteristics to generate a numeric value from 0 (isoelectric EEG) to 100 (fully awake) reflecting depth of sedation or consciousness.[52] BIS and others pEEG indexes have extended into neurocritical care as a surrogate for sedation adequacy, depth of coma, or prognosis after ABI. A recent expert consensus guideline recommended the use of pEEG to monitor the level of sedation (strong consensus), and it was recommended that all sedated patients (paralyzed or non-paralyzed) unfit for clinical evaluation would benefit from depth of sedation monitoring (strong consensus).[53]

Despite its utility, EEG presents several limitations. Interpretation requires significant expertise, often unavailable outside specialized neurological units. EEG recordings are frequently complicated by technical artifacts from muscular activity, sedation-induced alterations, patient movement, and interference from ICU equipment. Furthermore, EEG data provide a qualitative rather than quantitative analysis, complicating standardization of interpretation and clinical decision-making.[47] This underscores the need for index-based EEG methodologies to be implemented only in the populations and settings in which were validated. In this

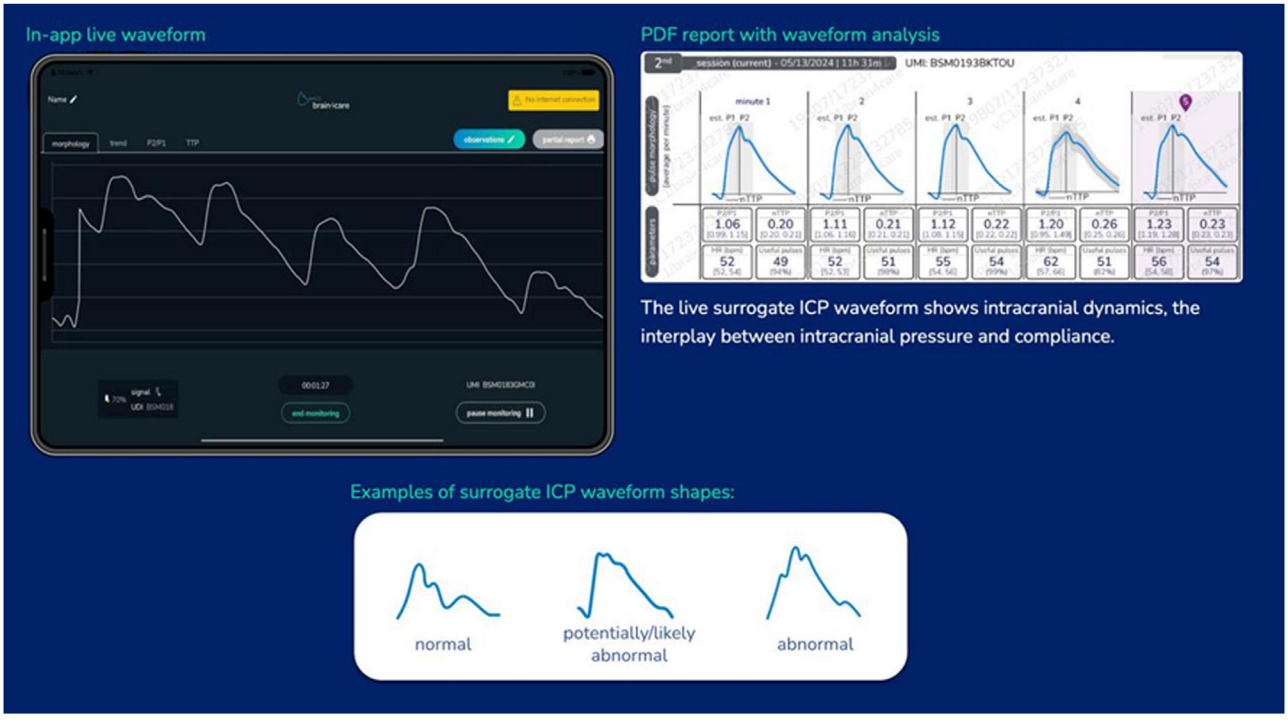

Figure 2 Noninvasive analysis of intracranial pressure waveforms.

context, point-of-care EEG systems, specifically utilizing automated computational algorithms for quantitative EEG analysis have facilitated bedside visualization for targeting timely clinical interventions by ICU staff, replacing the need for raw EEG interpretation.[54,55]

Noninvasive ICP waveform analysis

ICP waveforms (ICPW) are the representation of pulse transmission of arterial pressure via the choroid plexuses to the CSF and brain parenchyma.[56,57] The standard ICPW has three components: a) P1 ''percussion wave'' represents the arterial input; b) P2 ''tidal wave''; represents cerebral parenchyma and P3 or ''dicrotic wave'', is a reflect of venous outflow.[56,57] P1 has a sharp peak and in general its amplitude remains constant. P2 component is more variable in shape and amplitude. Usually after P3, the pressure wave decreases to its diastolic position.

Compliance refers to the ability of a mechanical system to respond to an applied force, expressed as the reciprocal of the system's stiffness.[58,59] In steady status inside of the cranial cavity, the ICPW conserve its basic morphology where P1 > P2 > P3 (stage 1 of pressure/volume curve). When the intracranial content increases and compensatory mechanisms are still preserved, the ICP ''number'' does not increase, but the waveform presents an increase in the P2 component compared to P1 (stage 2, compliance compromise without changes in ICP values)[58,59] (Fig. 2).

On the other hand, ICP pulse amplitude (difference between systolic and diastolic values) also tends to increase.[58,59] Only when the compensation capacity is exhausted, the ICP ''number'' increases and its waveform adopt a pyramidal shape with a gradual disappearance of P1, P3 and the marked ascent of P2.[58,59] Physiological studies have established that analysis of the harmonics, amplitude and variability of ICPW provide a more detailed information of what is occurring dynamically inside the skull.[58,59] Thus, the analysis of ICPW is a useful tool to gauge intracranial compliance. In summary, if P2 is sustained greater than or equal to P1, even with normal ICP values, indicates that compliance is reduced.[58,59]

Recently, a noninvasive system (Brain4care Corp, Sao Paulo, Brazil) was developed to detect and monitor the cranial cavity micro-expansions. Using a surface sensor, it allows obtaining an ICPW and the analysis of derived variables such as the relationship between the P1/P2 components and the time to reach the maximum peak (amplitude), which have shown a very good correlation with invasive ICP values.[60,61] At the same time, the analysis of the ICPW morphology allows the evaluation of cerebral compliance, making it an integrative tool for the evaluation of the intracranial compartmental syndrome[10,62] (Fig. 3).

This device was validated in different populations of ABI patients, including stroke, TBI and hydrocephalus.[60–62] A recent study using advanced machine learning models explored multiple waveform parameters to optimize mean ICP estimation. Approximately 72% of estimates from the validation sample were within 0—4 mmHg of invasive ICP values.[63] The Bland–Altman analysis revealed a mean difference (bias) between ICP and estimated ICP of −0.21 mmHg and an SD of ±3.68 mmHg. There was a moderate relationship between ICP and estimated ICP ($r = 0.43$, $P < .05$).[63] Nonetheless, the system has limitations. Patient agitation and manipulation can interfere with the sensor's ability to acquire reliable waveforms. Additional constraints include the requirement for an internet connection for waveform processing.[64]

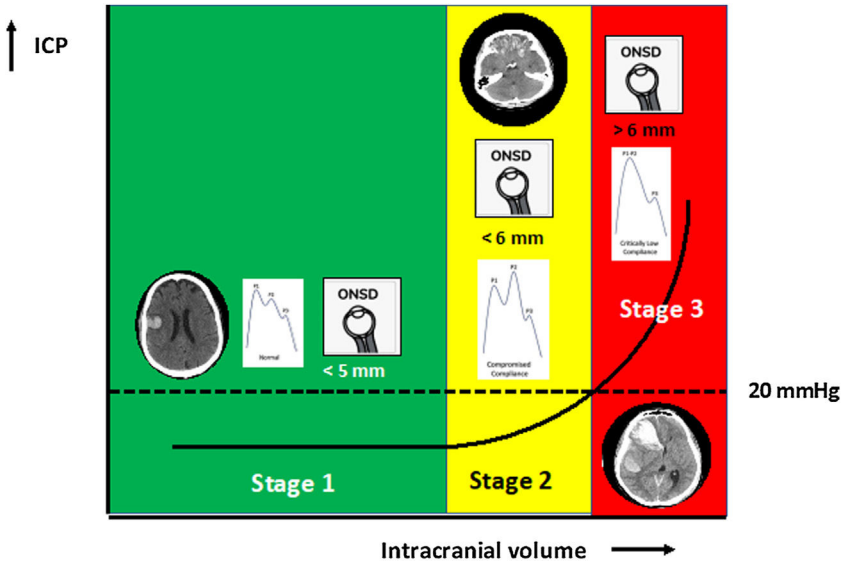

Figure 3 Stages in the development of the intracranial compartmental syndrome.

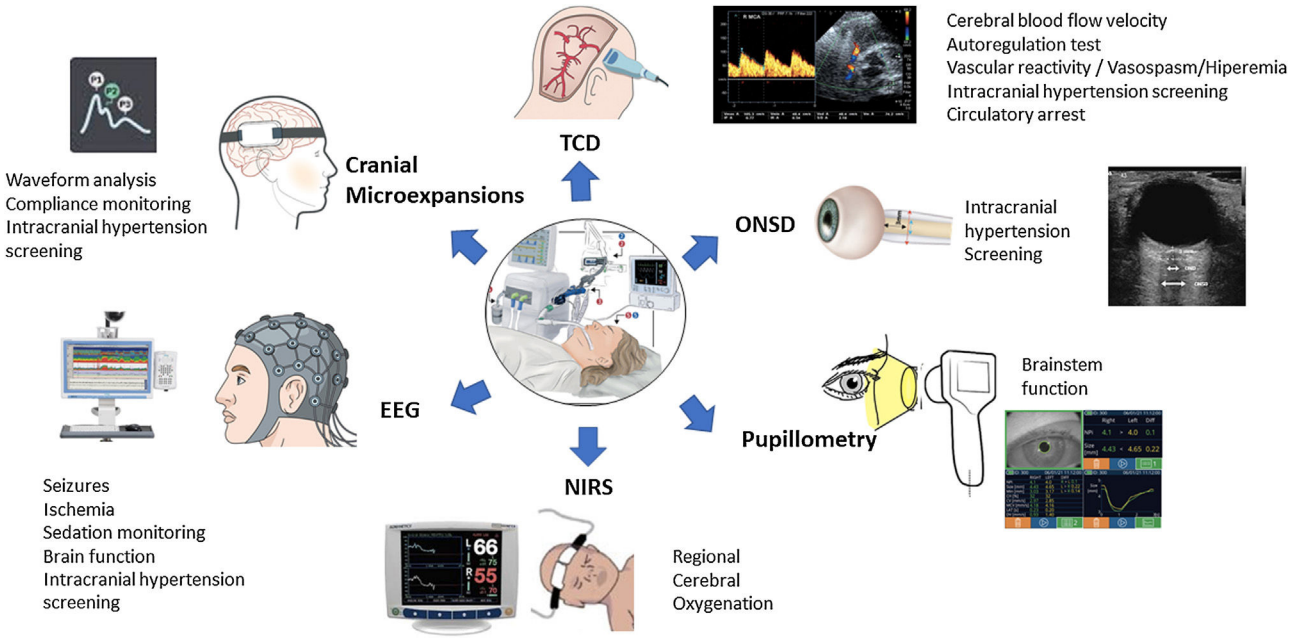

Figure 4 Summary of potential applications of nonivasive, bedside neuromonitoring techniques.

Discussion

Nonivasive, bedside neuromonitoring is increasingly being used in patients with ABI within the context of multimodal strategies. It can be used for different purposes (Fig. 4) directed to preserve neurological functions and to avoid secondary brain injury, including, but not limited to:

- Initial evaluation in the emergency room
- Need for new neuroimaging
- Categorization refinement
- Screening of intracranial hypertension and cerebral oxygenation status
- Monitoring brain function
- Indication of invasive monitoring
- Support of surgical decision

This is of special interest in low-middle income countries where access to invasive neuromonitoring is limited. However, physicians must know the advantages and limitations of all the nonivasive, bedside neuromonitoring techniques available (Table 2). Indeed, a recent article evaluated the correlation of different noninvasive neuromonitoring tools assessing intracranial hemodynamics, showing variable correlation and limited agreement, suggesting that they were not interchangeable.[65] The intensivist should be aware that

Table 2 Advantages and limitations of noninvasive, bedside neuromonitoring.

	Advantages	Limitations
Transcranial ultrasound	Bidimensional and hemodynamic monitoring No procedure-related complications	Operator-dependent
NIRS	Availability Early implementation No procedure-related complications.	Extracranial blood contamination No absolute treatment thresholds No validated bedside protocols
Automated pupillometry	Monitoring brain function Outcome preduction Promising tool in the screening of delirium, the need of intense neurocritical care, and identifying covert consciousness	Not significantly associated with ICP values alone
EEG	Early detection of subtle brain dysfunction Outcome prediction	Need of expertise Prone to technical artifacts
BIS	Sedation titrating Surrogate for depth of coma and prognosis after ABI	Developed in the field of anesthesia
ICP waveform analysis	Monitoring compliance Potential use for intracranial hypertension screening	Large skull defects Sensitivity to patient agitation Risk of signal contamination from extracranial circulation pulsations

NIRS: Near infrared spectroscopy; EEG: Electroencephalogram; BIS: Bispectral index; ICP: Intracranial pressure.

each modality captures different aspects of cerebral physiology, supporting the use of a multimodal approach.[65]

In this context, integrating (minimally invasive) determinations and trajectories of biomarkers such as neuron-specific enolase, glial fibrillary acidic protein (GFAP) and ubiquitin C-terminal hydrolase-L1 (UCH-L1) could be of help for categorization, evaluation of severity, and neuroprognostication purposes in different populations of acute brain injured patients.[66–68] However, in the author's opinion, its exact role to support daily management of patients with ABI in the neurocriticare setting remains to be determined yet.

We must acknowledge some limitations: this was not a systematic nor a scoping review. Therefore, our narrative review has limitations inherent to its design. Narrative reviews do not offer an evidence-based synthesis for focused questions, nor offer definitive guideline statements, but a readable, relevant synthesis of a diverse literature offering the authors interpretation, note gaps, and critique research.[69]

Conclusions

Nonivasive, bedside neuromonitoring techniques provide valuable information in the evaluation of patients with acute brain injury. Every noninvasive, bedside, neuromonitoring techniques have their own indications and limitations that must be recognized. These techniques may not replace invasive techniques, but can be useful to support multimodal strategies directed to preserve neurological functions and to avoid secondary brain injury.

CRediT authorship contribution statement

Conceptualization DAG and JALP; methodology DAG, JPB, FPDM, JABM, and JALP; validation DAG and JALP; investigation DAG, JPB, FPDM, JABM, and JALP; writing—original draft preparation DAG, JPB, FPDM, JABM, and JALP; writing—review and editing, DAG, JPB, FPDM, JABM, and JALP; supervision DAG and JALP; project administration DAG and JALP; funding acquisition, none. All authors have read and agreed to the published version of the manuscript.

Declaration of Generative AI and AI-assisted technologies in the writing process

The authors declare that they did not use generative AI and/or AI-assisted technologies in the writing process.

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Declaration of competing interest

The authors declare no conflicts of interest.

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