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REVIEW ARTICLE

Brain Ultrasonography in Neurocritical Care

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ABSTRACT

Ultrasonography (USG) has emerged as a vital bedside tool for diagnosis and for serial evaluation of response to the treatment in critically ill patients. In neurocritical care, brain ultrasonography offers unique advantages including the assessment of increased intracranial pressure (ICP), via midline shift measurement, cerebral blood flow measurement via transcranial doppler (TCD) and nerve sheath diameter (ONSD) monitoring. Recent technological innovations, through the incorporation of artificial intelligence (AI) and machine learning algorithms, have enhanced point-of-care ultrasound (POCUS) by improving diagnostic accuracy and reducing operator dependency. The development of portable, handheld ultrasound devices has further increased accessibility, enabling real-time decision-making even in resource-limited settings. However, the application of brain ultrasonography in neurocritical care is still limited due to a lack of knowledge and standardized training programs. This review synthesizes current evidence and provides practical guidance on integrating brain ultrasonography into neurointensive care, addressing a critical need for broader clinical adoption.

Key Words: *Brain ultrasonography, neurocritical care, optic nerve sheath diameter, point-of-care ultrasound, transcranial doppler.*

INTRODUCTION

Point-of-care ultrasound (POCUS) is being increasingly used in the critical care settings.[1] Ultrasonography (USG) can be used as a bedside tool to evaluate various pathological conditions in a quick, bedside and safe manner. In critically ill neurological patients, brain ultrasonography is commonly used to evaluate intracerebral flow velocity and perfusion using Transcranial Doppler (TCD), to assess midline shift and intracranial hemorrhage, and to diagnose raised intracranial pressure (ICP) by measuring the optic nerve sheath diameter (ONSD) and TCD derived formulae.[2,3] Despite its potential, current literature lacks a comprehensive synthesis of POCUS applications tailored to low-resource settings, emerging AI-assisted interpretations, and recent guideline updates. This review bridges that gap by consolidating evidence on the diagnostic and monitoring utility of bedside brain ultrasound in neurocritical care, with emphasis on practical implementation, technological advancements, and relevance for diverse clinical environments.

Transcranial Doppler

TCD, able to assess flow velocities in the basal cerebral arteries, was first described by Aasild in 1982.[4] It is based on the use of a low-frequency transducer (2 MHz) to insonate the intracranial vessels through selected acoustic windows. The physical principle of TCD is based on the Doppler effect, which states that reflected ultrasound waves from moving red blood cells undergo frequency shifts proportional to their velocity, generating a waveform that measures peak systolic and end-diastolic velocities. Cerebral blood flow velocity (v) is estimated based on an equation derived from the Doppler principle:

$$v = \frac{c \times f_d}{2 \times f_0 \cos\theta}$$

Where c is the velocity of emitted wave, f_d is the Doppler shift, and θ is the angle between device and the moving object. Variation in angle (θ) should be minimal for reliable measurement.

Transcranial colour-coded duplex sonography (TCCS), a technical advancement in late 1980's, enabled combined imaging of vascular structure and adjacent parenchyma.[5] TCCS offers the direct visualization of vascular structure in relation to adjacent brain parenchyma, which enables better identification of vessels. In addition, TCCS uses a phased array probe, which is commonly used in intensive care units as a part of POCUS. So, it has a logistic advantage over TCD and hence is increasingly used nowadays. TCCS is operator-dependent, limited by inadequate acoustic windows in patients with thick temporal bones and vulnerable to false-positive results with hypertension and false-negatives in severe vasospasm. It is unable to assess distal small intracranial arteries due to resolution constraints.

Acoustic windows

Skull bone is the main obstacle to the penetration of ultrasound waves. Trans-cranial ultrasound is advantageous in infants and children less than 2 years of age because of the open fontanels. In adults, areas with thin bone or foramen, the so-called acoustic windows, provide access to insonate different cerebral vessels. Four acoustic windows are commonly used.

Transtemporal window is a 3-4 cm diameter depressed area in the temporal bone cephalic to zygomatic arch, located between the ear and lateral orbital wall. Transducer is placed in this area with probe indicator pointing towards eyes.[2,6] Transtemporal window allows insonation of proximal segment (M1) the median cerebral artery (MCA), the A1 segment of the anterior cerebral artery (ACA), the posterior cerebral artery (PCA) and the final segment of the internal carotid artery (ICA).[7]

While using the *transorbital window*, the probe is placed above the closed eyelid. This approach allows visualization of ophthalmic artery, carotid siphon and optic nerve sheath.

The *suboccipital window* allows assessment of the distal segment of vertebral arteries and of the basilar artery. The transducer is placed in the upper neck at the base of the skull with ultrasound beam directed towards the bridge of the nose.

The *retromandibular window* is not an acoustic window to the brain but is used to sample the extra-cranial portion of the internal carotid artery to calculate the mean flow velocity of ICA. The ultrasound probe is placed beneath the angle of the mandible with the beam directed cranially.[2]

Doppler flow velocities and Doppler indices:

Normal spectral waveforms have certain characteristics as described in figure 1. Analysis of spectral waveform derived from TCD allows measurement of different doppler parameters as shown in the figure.

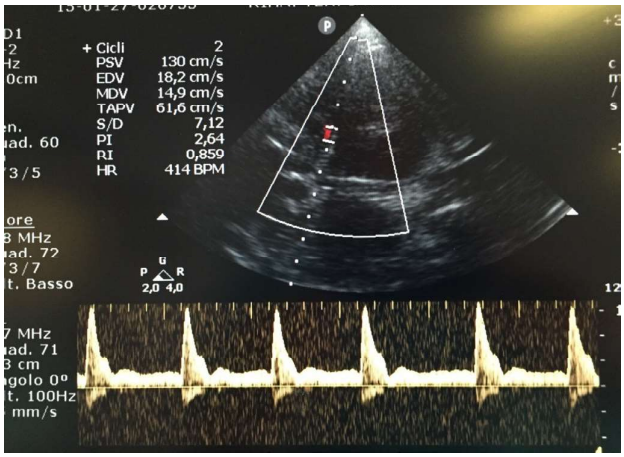

Figure 1. Transcranial Doppler; *Peak systolic velocity (PSV)*: corresponds to the highest point in the spectral waveform during each cardiac cycle. *End-diastolic velocity (EDV)*: corresponds to lowest point in the forward flow waveform. *Mean flow Velocity* (MFV): Increased MVF indicates stenosis, vasospasm or hyperdynamic flow states. MFV is decreased in hypotension, decreased cerebral blood flow or brain stem death.[8] *Pulsatility index (PI)*: calculated as the difference between peak systolic and end diastolic flow velocities, divided by the mean flow velocity.[9] PI does not depend on the angle of insonation and PI> 1.2 indicates high resistance flow.[10,11] *Resistive index (RI)*: resistance to flow distal to the insonated area of the vessel. RI is calculated as the difference between PSV and EDV, divided by PSV. Normal value is less than 0.75.[11]

Clinical applications:

A. *Detection of vasospasm:* In the patients who survive initial aneurysmal subarachnoid hemorrhage (SAH), the incidence of angiographic vasospasm is 67%, and around 30% develop delayed cerebral ischemia.[12] Vasospasm commonly develops 3 to 14 days after SAH.[13] Cerebral angiography is the gold standard for diagnosis of vasospasm but its role in monitoring is limited. TCD is increasingly being used for detection and monitoring of vasospasm. TCD is non-invasive, easily available at bedside and can be frequently performed for monitoring. Recent updated meta-analysis showed pooled sensitivity of 66.7% (55.9-75.9) for detection of MCA vasospasm. The diagnostic accuracy varies with the vessel insonated (Table 1).[14]

B. **Table 1.** Diagnostic accuracy of TCD and TCCS.[14]

	Transcranial Doppler			Transcranial color-coded duplex sonography
	MCA	ACA	BA	MCA
Pooled sensitivity	66.7% (55.9-75.9)	32.7% (10.9-65.7)	62.1% (33.3-84.3)	81.5% (67.5-90.3)
Pooled specificity	89.5% (80.3-94.7)	89.6% (48.2-98.7)	84.5% (71.1-92.3)	96.6% (93.2-98.3)
Positive predictive value	93.7% (88.9-96.9)	87.4% (57.8-98.3)	90.0% (84.6-93.9)	98.2% (96.4-99.1)
Negative predictive value	53.4% (46.7-60.9)	35.4% (24.6-50.6)	48.6% (36.5-67.6)	69.1% (56.1-80.9)

*MCA=Middle cerebral artery, ACA= Anterior cerebral artery, BA=Basilar artery. Numbers in parenthesis represent 95% CI

A. TCD- TCCS mean flow velocities are used to diagnose and categorize vasospasm. The mean flow velocity of the normal MCA is usually < 80 cm/s. Mean flow velocities of 120–159 cm/s correlates with mild vasospasm, 160–200 cm/s with moderate vasospasm, and > 200 cm/s with severe or symptomatic vasospasm.[15] These cut-offs have been derived for MCA, for other cerebral arteries, cut-offs may vary and no consensus exists. Progressive increase in MCA mean flow velocities by 21 cm/s/24 hours during first 3 days is found to be predictive of symptomatic vasospasm.[16] Mean velocities may be increased in other hyper-dynamic flow states. Lindegaard ratio (ratio of mean flow velocity in MCA to mean velocity in ipsilateral ICA) is useful for differentiating hyperemia from vasospasm.[17] Lindegaard index of > 3 is consistent with vasospasm and more than 6 is indicative of severe vasospasm (Table 2).[11] As for MCA, mean flow velocities of basilar artery (BA), when combined with the modified Lindegaard index (ratio of basilar artery to extracranial vertebral artery mean flow velocity) is useful for detection and grading of posterior circulation vasospasm (Table 2).[18,19]

Table 2. Cut-off values for MCA and BA velocity, coupled with the Lindegaard and modified Lindegaard ratio for diagnosis of vasospasm.

Parameter	Normal Values	Mild Vasospasm	Moderate Vasospasm	Severe Vasospasm
Middle Cerebral Artery				
MCA velocity (cm/s)	<120	120-159	160-200	>200
Lindegaard ratio (MCA/ extra cranial ICA)	<3	3-4.5	4.5-6	>6
Basilar artery				
BA velocity (cm/s)	<70	70-85	>85	>85
Modified Lindegaard ratio (BA/ extracranial VA)	<2	>2	>2.5	>3
Clinical correlation		Often asymptomatic	Possible neurological deficits	High risk of ischemia
Intervention	Monitor	Monitor + medical therapy	Optimize hemodynamics, consider intervention	Aggressive therapy (e.g., angioplasty, vasodilators)

B. *Non-invasive estimation of ICP:* Measurement of ICP is crucial in management of critically ill neurological patients. Invasive ICP measurement techniques are the "gold standard" but carry risk of hemorrhage, infection, technical failure and catheter displacement.[20] Various non-invasive models evaluating cerebral blood flow characteristics derived from both arterial and venous Doppler and measurement of ONSD have been used for estimation of ICP. Increased ICP is likely to affect the blood flow and pressure in major cerebral vessels which have compliant walls. This will be reflected as change in flow velocity waveform when measured by TCD. Low diastolic cerebral blood flow velocity (FDV), peaked waveform, and higher pulsatility index (PI) are seen with elevated ICP.[21] TCD-PI is probably the most studied index. Recent meta-analysis showed poor diagnostic accuracy of TCD-PI with AUROC between 0.55 and 0.718.[22] Poor diagnostic accuracy of PI is probably explained by the fact that changes in PI are also dependent on cerebral perfusion pressure (CPP), arterial blood pressure and change in CO2 level apart from ICP.[2] Schmidt et al. proposed and validated a mathematical model using MAP and cerebral blood flow velocities for estimation of non-invasive CPP (nCPP). nCPP correlated fairly with invasive CPP (R = 0.61; p = 0.003), with a 95% confidence limit range less than ± 12 mmHg, and with CPP ranging from 70 to 95 mmHg. Non-invasive ICP (nICP) was calculated by subtracting nCPP from MAP.[23,24]

Given the available evidence, non-invasive methods should not replace the invasive methods for ICP measurement. Values of non-invasive methods should be interpreted with caution if no invasive methods are available.

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C. *As ancillary test for brain death determination:* Determination of brain death is primarily clinical and ancillary test should not replace clinical examination. However, ancillary tests can complement clinical examination when some component of clinical examination cannot be performed, apnea test remains inconclusive and when confounding factors (metabolic derangements, heavy sedation, pupillary paralysis) are present.[25] The mechanistic basis for ancillary test is either absence of cerebral blood flow or absence of cerebral electrical activity. CT perfusion, CT angiogram and radionuclide scan have close to 100% specificity.[26] These techniques might not be practical in unstable ICU patients. Bed side TCD/ TCCS is a safe and non-invasive alternative. TCD confirms brain death by determining cerebral circulatory arrest (CCA), which has distinctive flow patterns: flow without forward flow progress, decrease in diastolic flow to disappearance of diastolic flow, oscillating pattern with flow reversal in diastole, short systolic spikes, and finally absence of Doppler signal (Table 3, Figure 2).[27] A recent meta-analysis looking at diagnostic accuracy of TCD for brain death determination estimated pooled sensitivity of 0.90 (95% CI, 0.87– 0.92) and pooled specificity of 0.98 (95% CI, 0.96–0.99), suggesting TCD to be an accurate ancillary test.[28] However, local regulations vary—TCD may not be a standalone confirmatory tool in most of the settings.

Table 3. Flow patterns in cerebral circulatory arrest

Flow pattern	Description
Systolic spikes	Brief, sharp peaks with absent diastolic flow.
Oscillating flow	To-and-fro movement (forward systolic + reversed diastolic flow).
Absent flow	No detectable signal

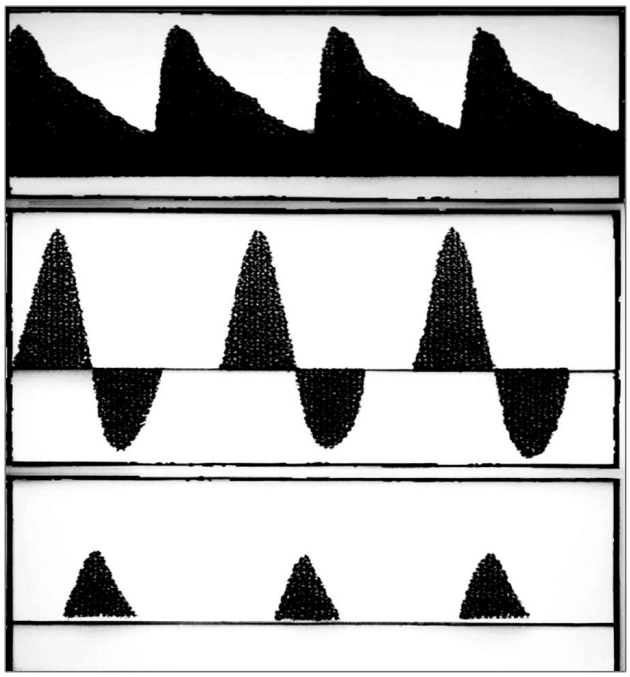

Figure 2. Transcranial Doppler (TCD) findings of flow patterns in cerebral circulatory arrest. The top panel shows normal waveform. The middle panel shows reverberating flow, characterized by alternating forward and reverse flow components within one cardiac cycle, indicating early-stage circulatory arrest. The lower panel depicts systolic spikes, representing sharp unidirectional velocity signals in early systole.

D. *Evaluation of patients with stroke:* Stroke accounts for 10% of all deaths worldwide.[29] Global lifetime risk of stroke after age of 25 years is approximately 25%.[30] CT scans and MRI scans are the main imaging modalities for diagnosis of stroke. TCCS can be used along with CT scan and MRI scans for diagnosis of arterial stenosis in ischemic stroke and identification of brain hematoma in hemorrhagic stroke. Acute intracranial hematoma (up to

5 days) appears hyper echoic whereas hematoma older than 5 days appear hypo echoic, surrounded by hyper echoic halo.[6] TCCS may be used to determine the site and size of hematoma. Maurer et al reported the sensitivity of 94% and specificity of 95% for detection of intracranial hematoma, however appropriate insonation windows were obtained in only 88% of the subjects.[31] In ischemic stroke, TCD/ TCCS has been used for screening, diagnosis, monitoring recanalization, detecting complications and for prognostication. TCD as compared with CTA for diagnosis of anterior circulation acute ischemic stroke has sensitivity of 100% and specificity of 94.5%.[32] TCCS is used safely for continuous non-invasive monitoring of thrombolysis, successful canalization and identification of early hemorrhagic transformation. Use of sonolysis and sonothrombolysis may increase the rate of recovery from stroke.[33] The abnormal middle cerebral artery blood flow, as evaluated by TCCS, in patient following mechanical thrombectomy for anterior circulation stroke is found to be predictive of poor neurological outcome at 90 days.[34]

Optic Nerve Sheath Diameter

The optic nerve sheath (ONS) is contiguous with the dura matter surrounding the brain and contains cerebrospinal fluid, which allows transmission of pressure from inside the cranium.[35] This close relationship between ICP and dilation of the orbital perineural subarachnoid space has been confirmed by various studies using ultrasound and magnetic resonance imaging.[36-39]

To measure the ONSD, a high frequency linear probe is placed over the closed eyelid after generous application of coupling gel. The optic nerve is identified as a hypoechoic structure along a regular course behind the eyeball. The diameter of the nerve is measured 3mm behind the retina perpendicular to the axis of the nerve (Figure 3).

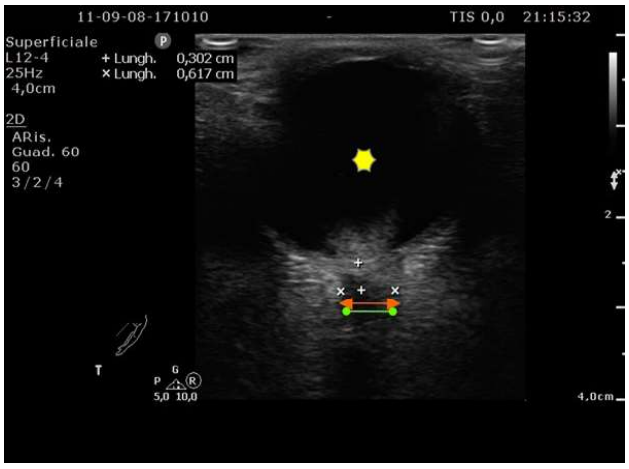

Figure 3. ONSD; the optic nerve is imaged as a hypoechoic structure extending from the retina posteriorly; the optic nerve sheath is subtly more echogenic and surrounds the nerve. Green line: optic nerve; orange line: optic nerve sheath; yellow star: posterior chamber of eye.

Recommendations for the cut-off values for detection of raised ICP have varied from 5.0 to 5.9mm with sensitivities and specificities ranging from 70 to 100% and 30 to 100%, respectively, depending on the study and the optimal cut-off value identified.[40-42] Although direct monitoring of ICP through insertion of an intracranial monitor is considered the gold standard in the diagnosis of intracranial hypertension,[43] studies have shown a good correlation between invasive intracranial pressure monitoring and optic nerve sheath diameter.[40] The 2023 European Society of Intensive Care Medicine (ESICM) consensus recommends ONSD as a Level B tool for non-invasive estimation of ICP in settings lacking invasive monitoring.[1]

Given the late onset of clinical signs in cases of intracranial hypertension, bedside measurement of ONSD may permit early detection and prompt therapeutic action, thus contributing to improved outcomes.[44,45]

The landmark systematic review by Robba et al. pooled data from 1,155 patients and reported excellent discriminative ability for detection of elevated ICP (AUC 0.93–0.94) when ONSD cut-off values of 5.0–5.7 mm was used. However, the authors highlighted critical limitations; the study had wide confidence intervals owing to heterogeneity in cut-off thresholds (range: 4.8–6.3 mm) and techniques which led to reduced generalizability. The study included mixed aetiologies (TBI, stroke, sepsis), with varying baseline ICPs and comorbidities, potentially confounding the correlation between ONSD and ICP.[46]

To address the technical variability, the 2024 consensus statement by Hirzallah et al. established evidence-based guidelines for ONSD measurement. The consensus emphasizes that adherence to standardized protocols improves reliability, particularly for serial monitoring in conditions like TBI.[47]

Based on current evidence, a cutoff of 5.5 mm is recommended for detecting elevated ICP in adults, with lower thresholds (e.g., 5.0 mm) considered in high-risk populations to improve sensitivity. Serial ONSD measurements should be prioritized over single values, as dynamic trends better reflect ICP fluctuations and therapeutic response. To enhance diagnostic reliability, ONSD should be integrated with clinical assessment and ancillary non-invasive tools (e.g., automated pupillometry or transcranial Doppler) in settings where invasive ICP monitoring is unavailable.[48]

Detection of midline shift

Brain midline shift (MLS) is a life-threatening condition requiring immediate diagnosis and treatment.[49] Alterations of consciousness following stroke was observed to be directly related to the MLS on CT scan.[50] Similarly after acute stroke MLS was an independent predictor of mortality at 15 days.[51] Midline shift (MLS) can be diagnosed with the Transcranial Sonography (TCS) in the diencephalic transverse scan. The transtemporal approach is most favoured as the low frequency ultrasound beams penetrate well through this window of thin bone.[4] Seidel et al considered third ventricle as the marker of the midline. MLS was calculated by halving the difference in the distance between the third ventricle and the temporal bone on two sides (Figure 4).[52]

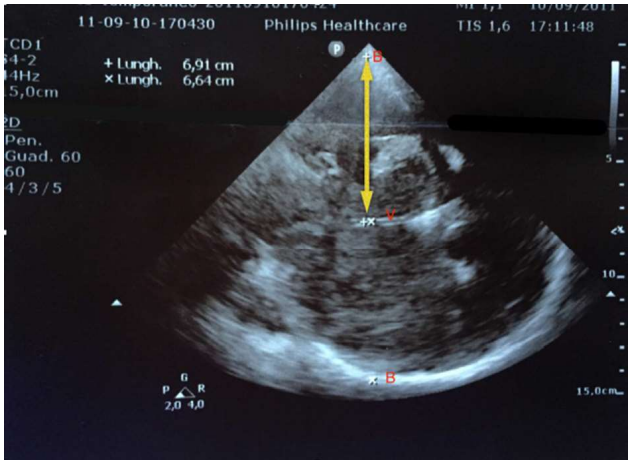

Figure 4. Midline shift (MLS) measurement by using ultrasound. B: ipsilateral and contralateral bone tables; V: the third ventricle, identified as a double hyperechogenic image over the midbrain. The distance between the external bone table and the center of the third ventricle is then measured bilaterally (yellow arrow). MLS = (B1V-B2V)/2

There might be discrepancy between the measurements based on anatomy of the brain. MLS calculated by the methods suggested by Seidel et al is only appropriate in patients with no intracranial defects, whereas for those with intracranial defects methods suggested by Carcato et al might be appropriate.[53]

As compared to other neuroimaging modalities, TCS is advantageous as it is cheap, non-invasive, readily available at the bedside and free from radiation exposure. Motuel et al have shown a good agreement between sonographic and CT findings for the assessment of MLS in neurocritical care patients.[54] TCS provides an accurate evaluation of brain parenchyma in patients with skull defects, such as in a patient who has undergone a decompressive craniectomy.[53] When TCS and CT were compared to measure MLS, only a small difference was evident between the two groups, ranging between 0.003 to 0.11cm.[55-57]

Detection of hydrocephalus

Posthemorrhagic hydrocephalus is a frequent complication after subarachnoid hemorrhage or parenchymal hemorrhage. External ventricular drainage may be necessary after severe traumatic injury to control intracranial hypertension. In these conditions, direct visualization of cerebral ventricles may be required for which TCS may be a useful tool.

Repetitive CT measurements have to be performed to monitor obstructive hydrocephalus in ICU thus US of head may be a relevant option.

Idiopathic normal pressure hydrocephalus (iNPH) is the most common form of hydrocephalus in adults. TCS can be used as screening as well as monitoring tool for iNPH. A screening test for ventricular enlargement is the Evans ratio or index which can be done with the help of brain ultrasonography.[52,58]

Intracranial Hemorrhage

TCS offers non-invasive, bedside alternatives to CT for initial assessment of intracranial hemorrhage (ICH), especially in unstable patients or in resource-limited settings.[2] Brain ultrasound provides real-time imaging with high sensitivity for detecting midline shift, hematoma volume changes, and ventricular compression—key indicators of ICH progression. Studies demonstrate that TCS can identify hyperechoic lesions corresponding to acute hemorrhage, with a reported sensitivity of 85–92% in patients with adequate acoustic windows (Figure 5).[59] Additionally, serial ultrasound examinations enable dynamic monitoring without repeated radiation exposure.[60] Despite its utility, TCS is operator-dependent and limited by skull attenuation, particularly in older patients or those with thick bone.[15] The absence of an acoustic window in 10–20% of patients reduces its universal applicability.[61] Advances in contrast-enhanced ultrasound (CEUS) and automated software for hematoma volumetry may improve accuracy, though further validation is required.[62] Future studies should focus on standardized protocols and AI-assisted interpretation to optimize diagnostic yield of TCS for detection of ICH.[63]

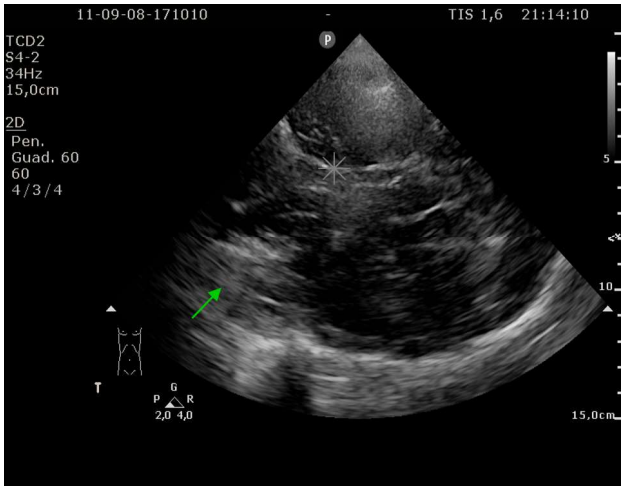

Figure 5. Transcranial sonography using the transtemporal window shows a temporal intracerebral bleed. (* sphenoid wing; Green arrow: intracerebral bleed)

Learning curve and skill levels

The learning curve for brain ultrasound varies significantly depending on the operator's prior experience and training. Basic competency in acquiring standard views (e.g., midbrain, ventricular, and parenchymal imaging) can be achieved after approximately 30–50 supervised scans, while advanced skills like Doppler assessment or interpreting complex pathologies require over 100 examinations. Skill progression is often categorized into three levels: novice (familiarity with anatomy and basic protocols), intermediate (ability to detect abnormalities like hemorrhage or hydrocephalus), and expert (proficiency in advanced techniques and integration with multimodal monitoring). Structured training programs and mentorship are emphasized to overcome diagnostic

challenges and reduce variability in image acquisition and interpretation.[3,64]

Structured training programs, such as those offered by the European Society of Intensive Care Medicine (ESICM), Society of Critical Care Medicine (SCCM), and World Federation for Ultrasound in Medicine and Biology (WFUMB), provide formal credentialing pathways to standardize skill acquisition. However, access to such programs—particularly in low- and middle-income countries (LMICs)—may be limited due to resource constraints. In these settings, feasible benchmarks (e.g., minimum supervised scans or hands-on training hours) should be adapted to local capacity while maintaining diagnostic rigor.

Incorporation of Artificial Intelligence in brain ultrasonography

There is growing evidence supporting the use of artificial intelligence (AI), especially the convolutional neural networks for the assessment of ONSD. Such applications may facilitate proper use of brain ultrasound even in setting with limited expertise.[65,66] For maximizing the benefits associated with use of AI in places with limited resources, the unique challenges inherent to these settings need to be considered and appropriately overcome.[67] To maximize AI's benefits in resource-limited settings, these hurdles must be addressed through rigorous real-world testing, transparent model interpretability, and equitable data representation.

Future research should focus on large-scale validation studies, seamless clinical integration, and the development of user-friendly AI tools that complement—rather than replace—clinician judgment. By doing so, AI can play a pivotal role in standardizing ONSD measurements and expanding the reach of neurosonography.

CONCLUSION

Brain ultrasonography has emerged as a valuable tool in critical care, offering rapid, non-invasive, and real-time bedside assessment of critically ill patients. Its diverse applications include evaluation of cerebral blood flow dynamics using TCD, assessment of ICP by measuring ONSD, detection of midline shift (MLS), and monitoring of hydrocephalus. Being an operator dependent procedure, attaining proper skill in brain ultrasonography is imperative for reliable assessment of pathologies.

While this technique holds great promise, challenges such as inter-operator variability, lack of standardized protocols, and limited accessibility in low-resource settings remain. Future research should focus on integrating artificial intelligence to enhance interpretation, establishing universal guidelines for clinical use, and expanding its applicability in low- and middle-income countries (LMICs). Clinicians and researchers must collaborate to refine training programs, validate diagnostic thresholds, and explore novel applications in neurocritical care. By addressing these gaps, brain ultrasonography can further solidify its role as an indispensable tool in improving patient outcomes worldwide.

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