



Diagnostic

Incorporation of Transcranial Doppler into the ED for the neurocritical care patient

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ARTICLE INFO

Article history:

Received 18 December 2018

Received in revised form 5 February 2019

Accepted 4 March 2019

ABSTRACT

Introduction: In the catastrophic neurologic emergency, a complete neurological exam is not always possible or feasible given the time-sensitive nature of the underlying disease process, or if emergent airway management is indicated. As the neurologic exam may be limited in some patients, the emergency physician is reliant on the assessment of brainstem structures to determine neurological function. Physicians thus routinely depend on advanced imaging modalities to further investigate for potential catastrophic diagnoses. Acquiring these tests introduces the risks of transport as well as delays in managing time-sensitive neurologic processes. A more immediate, non-invasive bedside approach complementing these modalities has evolved: Transcranial Doppler (TCD).

Objective: This narrative review will provide a description of scenarios in which TCD may be applicable. It will summarize the sonographic findings and associated underlying pathophysiology in such neurocritical care patients. An illustrated tutorial, along with pearls and pitfalls, is provided.

Discussion: Although there are numerous formalized TCD protocols utilizing four views (transtemporal, submandibular, suboccipital, and transorbital), point-of-care TCD is best accomplished through the transtemporal window. The core applications include the evaluation of midline shift, vasospasm after subarachnoid hemorrhage, acute ischemic stroke, and elevated intracranial pressure. An illustrative tutorial is provided.

Conclusions: With the wide dissemination of bedside ultrasound within the emergency department, there is a unique opportunity for the emergency physician to utilize TCD for a variety of conditions. While barriers to training exist, emergency physician performance of limited point-of-care TCD is feasible and may provide rapid and reliable clinical information with high temporal resolution.

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1. Introduction

The catastrophic neurologic emergency remains one of the most challenging presentations encountered by emergency physicians. Stress, diagnostic uncertainty, and the time-sensitive nature of the presentation lead to difficult management decisions. In these cases, a complete neurological exam is not always possible or feasible given the time-sensitive nature of the underlying disease process, or if emergent airway management is indicated. As the neurologic exam may be limited in some patients, the emergency physician is reliant on the assessment of brainstem structures to determine neurological function. This includes pupil evaluation, Glasgow Coma Scale, Cushing's reflex, cranial nerve reflexes and responses, and respiratory pattern. Physicians thus routinely depend on advanced imaging modalities such as computed

tomography (CT) or magnetic resonance (MR) imaging to further investigate for potential catastrophic diagnoses. Acquiring these tests introduces the risks of transport as well as delays in managing time-sensitive neurologic processes [1–4].

A more immediate, non-invasive bedside approach complementing these modalities has evolved: Transcranial Doppler (TCD). While TCD should not replace or delay formal imaging, it is a valuable adjunct when the patient is not stable enough to leave the emergency department (ED) for imaging tests, or in resource-limited settings where CT and MRI access is limited. It may also be used to assess for dynamic changes after the time-sensitive imaging test has already been performed.

This narrative review will provide a description of the ED scenarios in which TCD may be applicable. It will summarize the sonographic findings and associated underlying pathophysiology that may manifest in such neurocritical care patients. Lastly, an illustrated tutorial is provided along with pearls and pitfalls in this point-of-care ultrasound (POCUS) evaluation.

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2. Methods

This is a narrative review evaluating the traditional and point-of-care ultrasound (POCUS) techniques for TCD, with a discussion of their implementation in the acute care setting. A literature review of the PubMed and Google Scholar databases was performed with search date from 1980 to May 2018. Search terms included “Transcranial Doppler + ultrasound OR point-of-care ultrasound”, “intracranial pressure + ultrasound, Transcranial Doppler, OR point-of-care ultrasound”, “midline shift + ultrasound, Transcranial Doppler, OR point-of-care ultrasound”, “vasospasm + ultrasound, Transcranial Doppler, OR point-of-care ultrasound”, and “stroke + ultrasound OR point-of-care ultrasound.” The author included studies assessing the accuracy of TCD for various neurologic conditions, as well as articles detailing POCUS techniques and protocol description for TCD. Case reports, case controls, cohort studies, randomized clinical trials, and reviews were included, as determined by relevance to the narrative review. Commentaries and letters were excluded.

3. Discussion

3.1. Background

While point-of-care TCD in the emergency department (ED) and intensive care unit (ICU) is a relatively new development, it was initially described in 1982 by Aaslid et al. [5]. TCD is a noninvasive ultrasound (US) study involving the use of a low-frequency (≤ 2 MHz) transducer to detect blood flow within the intracranial circulation through relatively thin bone windows [6]. Originally, TCD was purely a pulsed wave technique guided by the depth, velocity, and audio characteristics of target vessels. However, due to the relatively recent introduction of B-mode, it is more feasible to identify the various intracranial vascular structures via TCD. Point-of-care TCD provides the emergency physician with a rapid, dynamic, and repeatable mechanism with which to monitor cerebral blood flow velocity (CBF-V) and intracranial vessel pulsatility through pulse wave Doppler (PWD) without requiring transport of a potentially unstable patient.

TCD is highly operator-dependent [7–10], however, which may limit its utility and incorporation into the ED. There is a steep learning curve to understanding the three-dimensional spatial understanding of the intracerebral arteries as they correspond to the anatomic windows [11]. Thus, a baseline knowledge of the cerebral arteries' anatomic location relative to the acoustic windows and the normal blood flow velocities within the various arteries remains critical for accurate measurements.

Literature has shown that while the large majority of patients have favorable acoustic windows, imaging is inadequate in approximately 5–20% of patients due to machine limitations, poor acoustic windows, patient compliance, and operator skill [12–14]. These anatomic difficulties may be overcome through the use of intravenous contrast agents containing microbubbles which pass through the arterial circulation, resulting in increased scattering of US waves, thereby enhancing vessel identification [15,16]. The advantages and disadvantages of TCD are provided below (Table 1):

Table 1
Advantages and disadvantages of Transcranial Doppler.

Advantages	Disadvantages
Safe, non-invasive	Time intensive
Bedside technique	Highly operator dependent
Excellent temporal resolution and repeatability	Not gold standard imaging modality
Provides useful information on cerebral vasculature	Inaccuracy due to poor acoustic window (in up to 5–20%)

3.2. Current applications of TCD

While there are a variety of indications for TCD, not all are applicable in the ED. Indications for transcranial ultrasonography and Doppler in the ED are listed below (Table 2). In particular, this review seeks to characterize the four major point-of-care applications for TCD in the ED [17–42]:

1. Midline shift
2. Vasospasm
3. Acute ischemic stroke
4. Raised intracranial pressure

3.3. Principles of spectral Doppler

The Doppler spectral pattern provides vital information regarding an arterial segment's flow characteristics. Based on the spectral patterns in various intracranial arteries, one may assess the cerebral hemodynamics in normal patients, as well as in various disease states. Spectral Doppler provides a measure of the changing velocity throughout the cardiac cycle and the distribution of velocities in the sample volume (or gate). In TCD, the sample volume (SV) is large in reference to the diameter of the intracranial arteries (SV is usually 6–10 mm, while the diameter of MCA 3–5 mm) [6]. Therefore, the Doppler spectra accurately represent the flow velocities from the center as well as from all layers of the arterial lumen, called spectral broadening [6].

The normal spectral waveform has a characteristic shape: a sharp systolic upstroke and stepwise deceleration with positive end-diastolic flow (Fig. 1). Furthermore, through the inclusion of waveform measurements derived from flow velocity readings, the emergency physician can utilize various indices, including Gosling's pulsatility index (PI) [43,44] and the Lindegaard ratio (LR) [45,46] which allow further characterization of increased cerebrovascular resistance, vasospasm, and hyperdynamic flow states. The variables of concern for TCD spectral Doppler include the following (Fig. 2) [6]:

3.3.1. Peak systolic velocity (PSV in cm/s)

PSV is the initial peak on the TCD waveform during each cardiac cycle. Most commonly, a rapid upstroke indicates an absence of severe stenotic lesions between the visualized intracranial arterial segment and heart [9].

3.3.2. End-diastolic velocity (EDV in cm/s)

The EDV should be between 20 and 50% of the peak systolic velocity values, indicating low resistance intracranial arterial flow which is seen in all structurally normal intracranial arteries [47].

3.3.3. Mean flow velocity (MFV in cm/s)

The mean flow velocity is calculated as EDV plus one-third of the difference between PSV and EDV [48]. Among the examined intracranial arteries in TCD, the MCA should have the highest MFV [6].

3.3.4. Pulsatility index (PI)

Most commonly in TCD, resistance to intra-arterial flow is assessed by using the PI, which is calculated by subtracting end diastolic velocity from peak systolic velocity and dividing the resulting value by the mean

Table 2
Indications for Transcranial Doppler.

Midline shift [17–22]
Vasospasm (including post-subarachnoid hemorrhage) [23–27]
Arterial occlusion/Ischemic stroke [28–34]
Elevated intracranial pressure [35–37]
Determination of brain death [38,39]
Monitor cerebral perfusion pressure during resuscitation [40,41]
Evaluation of thrombolysis efficacy [42]

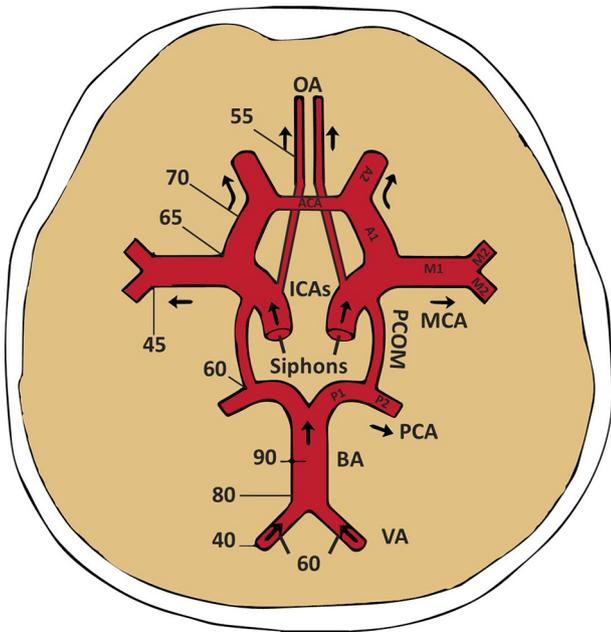


Fig. 1. Line diagram showing the major intracranial arteries and Transcranial Doppler windows. Flow directions from various arterial segments and depths of insonation (in mm) for an average human skull are shown.

flow velocity [49]. The PI is independent of the angle of insonation, and a value >1.2 represents high resistance blood flow [6,50].

Pulsatility index

$$= (\text{Peak systolic velocity} - \text{End diastolic velocity}) / (\text{Mean flow velocity})$$

or,

$$PI = (PSV - EDV) / MFV$$

3.3.5. Resistance index (RI)

Likewise, another TCD parameter used to assess flow resistance is the resistance index, which represents flow resistance distal to the area insonated [8]. RI is calculated by subtracting EDV from PSV and dividing the value by PSV, with a normal value being <0.75 [51].

Resistance index

$$= (\text{Peak systolic velocity} - \text{End diastolic velocity}) / (\text{Peak systolic velocity})$$

or,

$$RI = (PSV - EDV) / PSV$$

3.3.6. Heart rate

Heart rate may influence the aforementioned flow parameters, and is an important clinical component of TCD.

Below is a table with the insonation characteristics of selected cerebral vasculature (Table 3).

3.4. Ultrasound technique

The TCD examination is best performed using a 2-MHz frequency US transducer (Fig. 3). The higher frequency transducers routinely used to perform extracranial Doppler studies are not ideal for intracranial imaging, as the higher frequency waves are not able to adequately penetrate through the skull [5]. While there are several manuals and protocols published, all comprehensive examinations center around four 'windows' used to insonate the relevant cerebral arteries (Fig. 3) [52].

Despite each view having unique advantages for different arteries and clinical indications, a comprehensive TCD examination includes measurements from all four windows and assessment of blood flow at various depths within each major branch of the circle of Willis. These arteries' location is determined through a combination of: (1) relative direction of the transducer within the acoustic window (2) direction of blood flow relative to the transducer's orientation (3) depth of insonation (4) in difficult cases when one is able to differentiate anterior

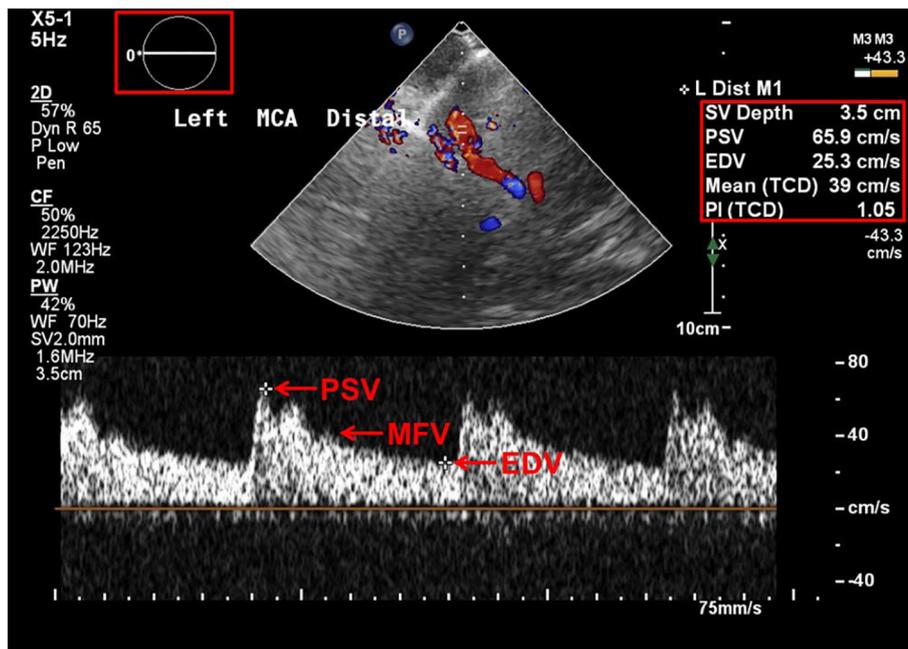


Fig. 2. Left MCA TCD waveform (bottom) with color Doppler (top). PSV = Peak systolic velocity, in cm/s. EDV = End diastolic velocity, in cm/s. MFV = Mean flow velocity, in cm/s. Note the depth and angle of insonation, as well as the Pulsatility Index (PI).

Table 3

Characteristics of selected cerebral vasculature. MFV: mean flow velocity, MCA: middle cerebral artery, ACA: anterior cerebral artery, PCA: posterior cerebral artery, BA: basilar artery, OA: ophthalmic artery.

Window	Artery	Probe angle	Depth (mm)	Flow direction	Adult MFV (cm/s)
Transtemporal	MCA	Straight/anterior-superior	30–65	Away	55 ± 12
Transtemporal	ACA	Straight/anterior-superior	60–75	Away	50 ± 11
Transtemporal	PCA-segment 1	Straight/posterior	60–70	Toward	39 ± 10
Transtemporal	PCA- segment 2	Straight/posterior-superior	60–70	Away	40 ± 10
Suboccipital	BA	Superior	80–120	Away	41 ± 10
Suboccipital	VA	Superior lateral	60–75	Away	38 ± 10
Transorbital	OA	Straight	45–55	Toward	21 ± 5

from posterior circulation, blood flow response to carotid compression or vibration [13].

However, for applications in the ED and intensive care unit (ICU) settings, a limited point-of-care protocol emphasizes measurements of the MCA due to the ease of access through the temporal window and the quality of the signal [7]. Additionally, the MCA carries approximately 50–60% of the ipsilateral carotid artery blood flow, and thus can be taken to represent blood flow to the hemisphere [49]. While reviewing a comprehensive TCD protocol is outside of the scope of this article, readers may pursue further exposure through published texts [52–54].

3.4.1. Transtemporal window

The transtemporal window view in a limited point-of-care protocol emphasizes measurements of the MCA, although a skilled ultrasonographer may be able to evaluate the ACA, PCA, and PCOM as well.

1. Using the 1–5 MHz phased array transducer with a TCD preset (or if unavailable, a cardiac preset), begin by placing your patient in the supine position with the head of bed >30°.
2. Place the ultrasound transducer (with the transducer marker oriented to screen left) over the temporal area, slightly above the zygomatic arch and immediately in front of the tragus of the ear. Orient



Fig. 3. Four acoustic windows commonly used in Transcranial Doppler examination: (A) transtemporal window (B) submandibular window (C) suboccipital window (D) transorbital window. Note the 2 MHz ultrasound transducer selection.

the transducer slightly upward and anterior (Fig. 3). Through the transtemporal window, the flow velocities in MCA, ACA, PCA, and PCOM can be obtained by pulsed wave Doppler.

3. Identify the ipsilateral and contralateral temporal bones, and the third ventricle (midline).
4. Decrease the depth to the distance of the third ventricle in the far-field and identify the cerebral peduncles and echogenic basal cisterns, approximately 3–8 cm in depth in the average adult.
5. Place a color Doppler box over the top half of the screen (the near field) just lateral to the cerebral peduncles, in order to locate the MCA. A red color signal (flow toward the transducer) between 40 and 65 mm represents flow in the ipsilateral MCA, while the blue color signal between 65 and 80 mm represents flow from the ipsilateral ACA [6]. In patients with good windows and favorable insonation, a red signal beyond 80 mm may be seen, representing flow in the contralateral A1.
6. Place the pulse wave gate on top of the MCA in order to obtain spectral Doppler waveforms. Angle correction should be utilized in order to adjust the pulse wave Doppler for the angle of insonation. MCA interrogation can be further refined, obtaining signals at 50 mm for the M1 segment, and 40 mm for the M2 segment [55].
7. Angle the transducer caudally, visualizing the terminal ICAs between 60 and 70 mm in order to obtain spectral Doppler waveforms [13].
8. After obtaining flow signals from the MCA and ACA, orient the ultrasound transducer posteriorly by roughly 10 to 30°, in order to evaluate the PCOM and PCA. In most patients, there is a loss of flow while angling the transducer posteriorly. This is followed by flow signals from the ipsilateral PCA, visualized between 55 and 70 mm, as the transducer continues to be angled posteriorly [6]. An absence of the flow gap while angling the transducer posteriorly after evaluating the MCA/ACA most often represents flow signals from the PCOM [13].
9. Repeat the above protocol for the contralateral hemisphere.

3.4.2. Submandibular window

Place the transducer laterally under the jaw anterior and medial to the sternocleidomastoid muscle, aiming the transducer upwards and slightly medially with a depth of 50 mm. The distal ICA should be visualized as a low-resistance flow signal directed away from transducer. Occasionally, the external carotid artery may be confused for the ICA, and it is important to perform a temporal artery tap to differentiate the two. The temporal artery tap consists of tapping over the ipsilateral superficial temporal artery while simultaneously assessing the carotid bifurcation for evidence of a reflected flow in the external carotid artery [56]. Note that this method alone may not reliably distinguish between the external and internal carotid arteries [57].

3.4.3. Suboccipital window

Turn the patient to one side and place the transducer just below and medial to the mastoid process, directing the transducer slightly medially toward the bridge of the nose or contralateral eye. The suboccipital window is key to obtaining flow signals from the ipsilateral vertebral artery, commonly found between 50 and 75 mm, with the signals presenting away from the transducer (appearing as blue). In patients with favorable anatomy, the basilar artery may be visualized by aiming the transducer slightly upward and medial, increasing the depth to between 75 and 110 mm. If the basilar artery cannot be visualized, one may place the transducer just below the occipital protuberance and oriented toward the nasal bridge, commonly called the transforaminal window [58]. The flow from the basilar artery is also away from the transducer.

3.4.4. Transorbital window

Transorbital insonation focuses on evaluating the ipsilateral ophthalmic artery and the ICA siphon. The transducer is placed gently over the eyelid and angled slightly medial and upward. Flow signals at a depth of <60 mm toward the transducer represent the ophthalmic

artery. In order to visualize the siphon, one must move the depth beyond 60 mm. Due the ICA siphon being a curved artery, the flow signals may vary in direction either toward or away from the transducer [59]. Additional care must be taken if the genu of the siphon is insonated, as this may present as bidirectional signals [60].

3.5. Measurement of midline shift (MLS)

Case: A 63 year-old female taking Coumadin for atrial fibrillation presents to the emergency department (ED) via private vehicle after falling from a ladder and striking her head. She did not lose consciousness and is reported to be at her baseline mental status, as per her husband. Secondary survey reveals a hematoma on her right parietal scalp. She has a Glasgow Coma Scale (GCS) of 15 with a normal neurologic exam. Initial head CT shows a small subdural hemorrhage without evidence of midline shift. Her anticoagulation is reversed, and she is admitted to the Trauma ICU for further treatment and evaluation of her injuries. However, while awaiting transfer to the ICU, the patient has further deterioration in her neurological status that is highly concerning for increasing mass effect from her intracranial hemorrhage. A bedside TCD is performed, revealing a 10 mm shift away from the previously discovered subdural hematoma. The patient is taken for emergent repeat head CT, and this midline shift is confirmed. The patient is taken to the OR emergently for evacuation.

Diagnosis of midline shift using TCD is important for both recognizing further secondary neurological injury and for neuro-prognostication in the acute setting [55]. While any amount of midline shift is abnormal, a clinically significant midline shift as little as 0.5 cm is associated with a poor prognosis [61]. Given its high temporal resolution and repeatability [19], TCD allows closer monitoring of critically injured patients. MLS calculated by use of TCD has demonstrated a high degree of correlation with CT and has been associated with predicting poor outcome secondary to midline shift in a variety of conditions, including acute stroke, hemorrhage, and traumatic brain injury [19,62–64].

There have been several methods published regarding MLS measurement by TCD, but the most reliable method includes measuring the distance from the bilateral temporal bones to the midline third ventricle [59]. Of note, distance A is measured from the ipsilateral side, whereas distance B is from the contralateral side (Fig. 4). The full length from the ipsilateral to the contralateral temporal bone should be measured. Then, MLS is calculated by using the following equation, with an example provided below:

$$\text{Midline shift (MLS)} = (\text{distance A} - \text{distance B}) / 2$$

If the MLS is positive, then the MLS is expanding away from the ipsilateral side. Conversely, if the MLS is negative, the MLS is toward the ipsilateral side. In order to reduce user error and improve repeatability, the physician should make two independent measurements from each side. There should also be an internal check for correct measurements (i.e. the sum of distance A and B should equal the full distance from the ipsilateral to contralateral temporal bones) [59]. Care must be taken, however, as measuring MLS is reliant on proper trans-temporal windows which may be challenging to obtain [15]. Likewise, there is no data correlating angle of insonation and accuracy of transcranial US midline shift measurements, although official guidelines recommend an upward angle of insonation no >10–15° [59].

3.6. Evaluation of vasospasm

Case: A 72 year-old female with a history of cerebral aneurysm is brought into a critical access hospital via EMS for the development of hemiparesis and dysarthria. Per her husband, she had complained of a severe headache one week prior to her current symptoms but had not presented to the hospital due to symptom improvement. A stroke code is called for presumed subarachnoid hemorrhage, but the CT

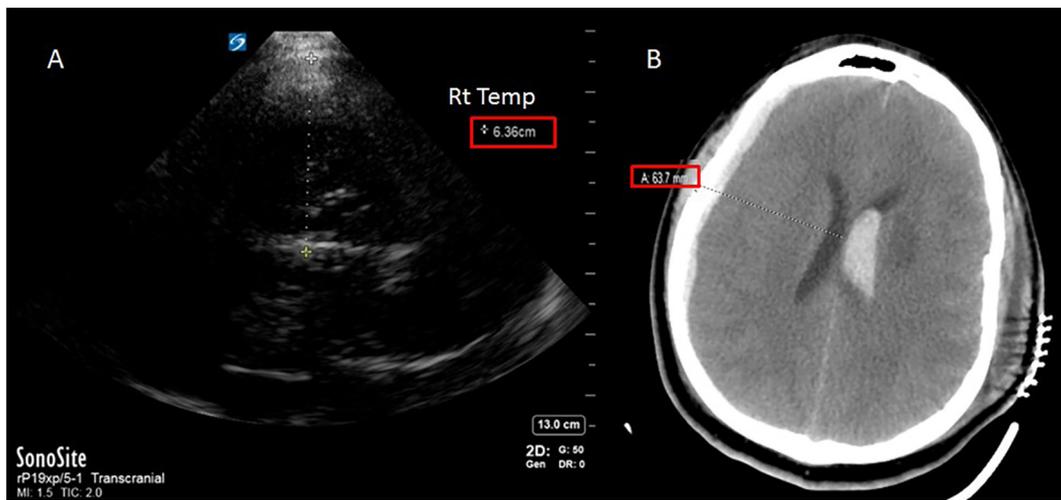


Fig. 4. Transcranial imaging for midline shift. A. Insonation from right temporal bone to third ventricle, representing distance A (6.36 cm). B. Follow-up CT scan post TCD which confirms distance from right temporal bone to third ventricle found on TCD.

scanner is not immediately available due to another critical case. A bedside TCD is performed which shows an increase in the mean flow velocity of the left MCA territory suggestive of cerebral vasospasm. This information is relayed to the neurosurgical team and antihypertensive therapy is started prior to imaging. CT angiography of the head is later performed confirming both the presence of an aneurysm in the left MCA territory as well as vasospasm in the same vessel.

TCD for vasospasm has been studied extensively and emerged as a validated screening tool, particularly in patients with subarachnoid hemorrhage (SAH) [65–67]. Post-SAH vasospasm greatly influences prognosis, leading to significant morbidity and mortality due to strokes from cerebral ischemia [68,69]. By utilizing the relationship between MCA diameter and mean blood flow velocities, the emergency physician can quantify and subcategorize vasospasm [20]. Compared to the gold standard of CT angiogram, the sensitivity and specificity of TCD for diagnosis for SAH-associated vasospasm is between 89 and 98% [70]. However, the transorbital and transforaminal windows are less reliable compared to the transtemporal window. The MCA's normal spectral Doppler profile has a sharp systolic upstroke followed by a step-wise diastolic deceleration and a normal MFV < 80 cm/s. Further categorization of vasospasm based on differing MFV is provided below (Table 4). Of note, these cutoffs were originally derived using traditional TCD transducer, not the phased array transducer common in point-of-care use. Furthermore, progressive increases in MFV early on in SAH have been shown to be predictive of vasospasm [71]. Another method of quantifying vasospasm severity is the formula known as the Lindegaard ratio, although this requires more measurements [53].

$$\text{Lindegaard ratio} = \frac{\text{Ipsilateral MCA mean flow velocity}}{\text{ipsilateral extracranial ICA mean flow velocity}}$$

As vasospasm is not the only cause of increased mean flow velocities, the Lindegaard ratio does not necessarily grade vasospasm, but rather

Table 4
Doppler characteristics of MCA vasospasm.

	Mean flow velocity (MFV)	Lindegaard ratio
Normal	≤80 cm/s	<3
Mild	81–119 cm/s	<3
Moderate	120–159 cm/s	3–5
Severe	160–199 cm/s	5–6
Critical	≥200 cm/s	>6

aids in the differentiation of hyperemia and the onset of vasospasm [59]. Grading of vasospasm by Lindegaard ratio is provided below (Table 4).

It must be noted, however, that several factors make the diagnosis of vasospasm by TCD challenging. For instance, cerebral blood flow is influenced by many factors, including PaO₂, PaCO₂, blood viscosity, and collateral flow, all of which may affect MFV. Additionally, the operator should be experienced, as improper vessel identification, various lesions proximal to the insonated area, and tortuous vessel course can lead to improper measurements.

3.7. Evaluation of acute ischemic stroke

Case: A 66 year-old male presents to a national stroke center via EMS as a field-activated stroke code whose symptoms began acutely 45 min prior to arrival. The patient arrives with severe left-sided weakness and is found to have a perfusion defect in the right MCA territory. The decision is made to begin systemic thrombolytic therapy, but following the infusion, the patient does not improve. TCD is performed, which shows no evidence of recanalization in the affected vessel. Interventional radiology is consulted and the patient is taken emergently to the IR suite for thrombectomy.

TCD is particularly useful in acute ischemic stroke as repeated TCD studies can be used to follow the course of an arterial occlusion after thrombolysis [42]. From recent studies, TCD is able to detect acute MCA occlusions with a sensitivity and specificity of >90% [72–75]. TCD detection of occlusions in the ICA siphon, vertebral, and basilar arteries has a sensitivity between 70 and 90%, and a specificity >90% [76].

Additionally, TCD plays an important role in augmenting thrombolytic-induced arterial recanalization, while simultaneously monitoring its efficacy [77]. TCD is thought to act synergistically with thrombolytic agents by delivering mechanical pressure waves to increase the amount of thrombus surface area exposed to circulating tissue plasminogen activator (tPA) [78,79]. A meta-analysis found that complete recanalization rates have been higher in patients receiving combination of TCD with tPA, 37.2% (95% CI, 26.5%–47.9%) compared with patients treated with tPA alone 17.2% (95% CI, 9.5%–24.9%) [80]. Of note, TCD-enhanced thrombolysis has not been associated with an increased risk of symptomatic intracerebral hemorrhage [80].

The presence of detectable residual flow signals on TCD prior to intravenous thrombolysis can be used to predict recanalization. Patients with detectable residual flow signals before intravenous thrombolysis are twice as likely to have early complete recanalization [81,82]. In contrast, those patients with no detectable flow signals on TCD have a <20%

Table 5
Transcranial Doppler evaluation of stenosis and occlusion.

Transcranial Doppler Evaluation of Stenosis and Occlusion			
Signs of Occlusion	Absence of signal from artery	Sonographic evidence of collateral flow	
Primary signs of stenosis	Increase in mean flow velocity at the site of luminal narrowing		
Secondary signs of stenosis	Decreased velocity upstream from the lesion	Increased pulsatility upstream from the lesion	Abnormal flow downstream from the lesion

chance for complete recanalization within two hour TCD can also be used to accurately identify these patients with incomplete recanalization. Compared to angiography (digital subtraction or magnetic resonance), TCD has a sensitivity of 91% and specificity of 93% for detecting successful recanalization after thrombolysis [42]. In the acute care setting, this finding may prompt consideration of alternative therapies, including mechanical thrombectomy or intra-arterial thrombolysis [82]. The evaluation for stenosis or acute ischemic stroke centers on the presence or absence of the following signs (Table 5) [44].

3.8. Measurement of intracranial pressure (ICP)

Case: A 22 year-old male presents to a level 1 trauma center via EMS following a motorcycle crash. Upon arrival, he was following commands with a GCS of 15. However, shortly after arrival, his condition begins to deteriorate, eventually requiring intubation. Head CT confirms the presence of traumatic brain injury (TBI) with extensive SAH and subdural hematoma (SDH). Neurosurgery is consulted, but while awaiting their evaluation, bedside TCD reveals evidence of increased ICP. The patient's head is subsequently elevated and an infusion of hypertonic saline is begun. The neurosurgical team arrives soon after and places an extraventricular drain (EVD). Following intervention, repeat TCD is performed and shows a significant decrease in ICP.

Although ICP can guide patient management in emergency care, it is not commonly monitored in many clinical conditions. This is due to the invasive nature of the standard methods for ICP monitoring (epidural, subdural, intraparenchymal, and intraventricular monitors) and their associated risks to the patient (infections, brain tissue lesions, and hemorrhage). TCD, on the other hand, provides rapid, reliable clinical information at the bedside with high temporal resolution [29]. There is extensive literature proposing TCD as a tool for noninvasive measurement of ICP, showing a good concordance between invasive ICP measurements and TCD-based ICP measurements [50,83–85].

Furthermore, in comparison to ultrasound measurement of the optic nerve sheath diameter, TCD has a higher specificity and sensitivity for detecting elevated ICP [86]. Similarly, TCD has been demonstrated to

accurately screen patients with mild or moderate TBI at risk of secondary neurological deterioration due to elevated ICP, which may play an important role in determining patient disposition and level of care [87]. For instance, multiple studies, including a recent multicenter study in 356 traumatic brain injury patients, have shown that TCD had a negative predictive value of 98% for predicting neurologic worsening over the first week in patients admitted to the hospital with mild to moderate TBI [88,89].

TCD-based ICP measurements are based on approximate semi-quantitative relationships between cerebrovascular dynamics and ICP. As ICP increases, the flow in intracranial vessels is affected. Initially, there is an increase in systolic velocity (as measured by PSV) as the increased ICP externally compresses cerebral vessels, causing the intraluminal diameter to narrow. Furthermore, diastolic flow becomes blunted, as raised ICP becomes the predominant external pressure opposing forward arterial flow during diastole [59]. In extreme cases, ICP can exceed normal forward flow during diastole, leading to diastolic flow reversal and catastrophic ischemia, as evidenced below (Fig. 5).

An increased ICP can be estimated by the Gosling's pulsatility index (PI), which reflects peripheral resistance, equal to the difference between the peak systolic velocity (PSV) and end-diastolic velocity (EDV), divided by the mean flow velocity (MFV) [52].

$$\text{Pulsatility index (PI)} = (\text{PSV} - \text{EDV}) / \text{MFV}$$

A formula has been derived to convert pulsatility index into ICP (from all causes), with an associated sensitivity of 89%, and specificity of 92% [50]:

$$\text{ICP} = (10.93 \times \text{PI}) - 1.28$$

Therefore, based on this formula, a PI of >2.13 would correlate to an ICP >22 mm Hg, which is the clinically significant cutoff for raised ICP, whereas normal PI is <1.2, corresponding to an ICP of 12 mm Hg. There are drawbacks to using this technique, however. The main limitation of utilizing TCD to measure ICP is that wide confidence intervals

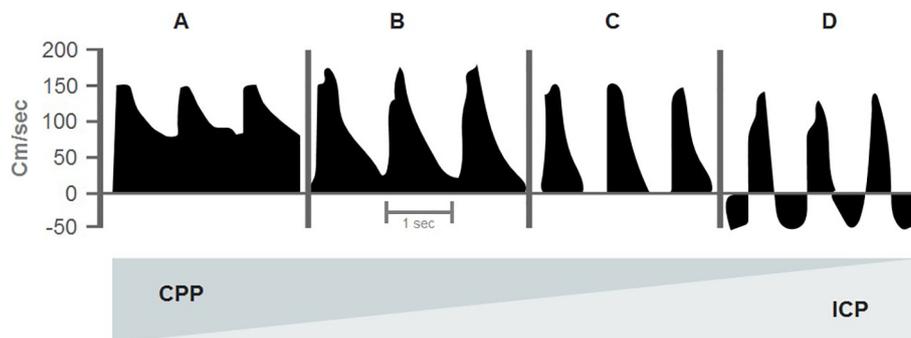


Fig. 5. Progression of Transcranial Doppler waveforms with decreasing cerebral perfusion pressure (CPP) and increasing intracranial pressure (ICP) after head injury. A. Normal systolic upstroke with normal step-down of diastolic flow. B–C. Increased peak systolic flow with decreasing diastolic flow and eventual blunting of diastolic flow. D. Diastolic flow reversal (AKA Biphasic or oscillating flow)—where diastolic flow reversal begins to approach equal size to systolic flow. This denotes one state in which cerebral circulatory arrest can be diagnosed.

have been reported when compared directly to ICP monitors [44,90]. Furthermore, cerebral blood flow and the PI are influenced by many factors, including arterial blood pressure, PaCO₂, and cerebral perfusion pressure. Likewise, lack of pulsatile flow, as is the case in patients with left ventricular assist devices, renders the systolic and diastolic ratios for pulsatility index uninterpretable. Nevertheless, TCD-based methods for ICP measurement generally present a positive degree of agreement and acceptable correlations with measured ICP while retaining a high degree of temporal resolution.

4. Conclusion

With the advent and wide dissemination of bedside ultrasound within the emergency department, there is a unique opportunity for the emergency physician to utilize TCD for a variety of conditions. The core applications include the evaluation of midline shift, vasospasm after SAH, acute ischemic stroke, and ICP. Although there are numerous formalized TCD protocols utilizing four views (transtemporal, submandibular, suboccipital, and transorbital), point-of-care TCD is best accomplished through the transtemporal window. While barriers to training exist, emergency physician performance of limited point-of-care TCD is feasible and may provide rapid and reliable clinical information with high temporal resolution.

Conflicts of interest

The author has no conflicts of interest to disclose.

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