CHEST

Better With Ultrasound
Transcranial Doppler

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VIDEO

Transcranial Doppler (TCD) ultrasound is a noninvasive method of obtaining bedside neurologic information that can supplement the physical examination. In critical care, this can be of particular value in patients who are unconscious with an equivocal neurologic examination because TCD findings can help the physician in decisions related to more definitive imaging studies and potential clinical interventions. Although TCD is traditionally the domain of sonographers and radiologists, there is increasing adoption of goal-directed TCD at the bedside in the critical care environment. The value of this approach includes round-the-clock availability and a goal-directed approach allowing for repeatability, immediate interpretation, and quick clinical integration. This paper presents a systematic approach to incorporating the highest yield TCD techniques into critical care bedside practice, and includes a series of illustrative figures and narrated video presentations to demonstrate the techniques described.

CHEST 2020; 157(1):142-150

KEY WORDS: critical care; medical education; transcranial; ultrasound

In patients who are unconscious, there is often a diagnostic gap between the yield of the bedside neurologic examination and that of other investigations that may be invasive or require patient transport.[1] In certain clinical scenarios, transcranial Doppler (TCD) may have a role in bridging this gap, effectively augmenting the physical examination and helping the bedside physician decide whether investigations such as CT scan, CT angiography (CTA), or MRI are necessary, or if intracranial pressure (ICP) monitoring or other surgical interventions are required.[2-6]

A variety of different ultrasound techniques have been described[1,7,8]; this paper aims to present a systematic approach for incorporating these techniques into bedside practice. Point-of-care TCD may be used to assess for the presence of mass effect causing midline shift (MLS), or to deploy a series of Doppler-based assessments, allowing the bedside provider to investigate for raised ICP, cerebrocirculatory arrest, or vasospasm. The combination of transcranial ultrasound imaging (bright mode) and color/spectral Doppler encompasses the modern-day applications of TCD (as used in point-of-care

ABBREVIATIONS: CTA = CT angiography; EDV = end-diastolic velocity; ICA = internal carotid artery; ICP = intracranial pressure; MCA = middle cerebral artery; MLS = midline shift; MV = mean velocity; PI = pulsatility index; PSV = peak systolic velocity; TCD = transcranial Doppler; VTI = velocity time integral

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DOI: https://doi.org/10.1016/j.chest.2019.08.2204

neurosonology and radiology applications).[1] Normal findings on TCD can also be helpful by providing reassurance in the right clinical circumstance.

Although TCD is traditionally performed by sonographers in many institutions, the same phenomenon which has brought general point-of-care ultrasound to the bedside has opened the door to intensivist-led TCD. It is crucial to emphasize, however, that TCD is more sophisticated than many other ultrasound applications and takes considerably more practice to build confidence in both image acquisition and interpretation. Despite its complexity and longer learning curve, the opportunity to expand the bedside neurologic examination to include these techniques is well worth the effort for many providers. Data obtained using these techniques should be treated as hypothesis-generating for the most part, and should always be verified with a confirmatory test. Realistically speaking, point-of-care TCD will, in resource-rich countries, serve mostly as an adjunct or precursor to definitive imaging or invasive techniques.

In this paper, the three commonly used ultrasound transducers will be described as linear (usually in the range of 10-12 MHz, most commonly used for insertion of central venous catheters), phased-array (usually in the range of 1-5 MHz, most commonly used for cardiac and thoracic ultrasound), and curvilinear (usually in the range of 2-5 MHz, most commonly used for imaging the abdomen) for the sake of brevity and simplicity. We will focus primarily on the use of the phased-array transducer for TCD because it provides the smallest footprint for insonation of the transtemporal window.[1] To address the frequent discord with respect to the correct orientation of the ultrasound image on screen, this paper will favor the radiology convention, with the orientation marker (usually a dot) to the left side of the screen as standard for transcranial applications.

The recommendations presented in this paper are our opinions. Although they represent reasonable approaches to common problems, it is important to note that there are many alternative and equally reasonable methods which may be favored by other providers. For example, optic nerve sheath diameter has been described to help predict raised ICP.[9] However, we have significant concerns related to the optic nerve sheath diameter given the small margins of error (millimeter cutoffs) for mismeasurement of this modality, its low specificity and many false-positives, and its inability to titrate its ICP measurements to cerebral perfusion pressure.[10]

Currently, there are no clear evidence-based criteria for total training time needed to become competent in point-of-care TCD; the best evidence we have is at the level of expert opinion.[11] In terms of certification processes, the best known is the Neurovascular Specialist certification examination provided by the American Society of Neuroimaging.[12] For further reviews regarding Doppler ultrasound, brain sonography, and diagnostic arterial ultrasound, there are several citations which delve deeper into the realm of formal, comprehensive TCD.[7,8,13-15]

General Advantages of Ultrasound

The general advantages of point-of-care TCD are its immediate around-the-clock availability, repeatability, noninvasive nature, and low cost.[1,4,5] Additionally, like all point-of-care ultrasound applications, it is a tool that brings the physician to the bedside to spend more time with the patient, which brings additional value in many cases. Ultrasound also provides dynamic interrogation of the neurovasculature without requiring radiation or the risk of transport for a CT scan.[2,3] In resource-limited settings, where access to other imaging modalities is scarce, TCD may be of particular utility.[1]

Technique 1: Basics of TCD Anatomy and Image Acquisition

To acquire a TCD image, start by placing the patient in a supine position with the head of bed slightly elevated (30°-45°) (Fig 1, Video 1). Although dedicated transcranial transducers exist, most point-of-care providers will use the best available substitute, the 1- to 5-MHz phased-array transducer. Most modern ultrasound machines will have a TCD preset, which will by default position the index marker to the left of the ultrasound screen. Users who do not have access to a machine with a TCD preset should use the cardiac preset, which is a reasonable substitute. However, using the cardiac preset will by default position the index marker to the right of the ultrasound screen, which can result in some confusion with respect to orientation. In such cases, we recommend that providers manually adjust the machine such that the index marker is positioned on the left side of the screen, therefore respecting the radiology convention. Three transcranial windows may be used for comprehensive TCD ultrasound: transorbital, transtemporal, and transforaminal. Because point-of-care TCD is concerned primarily with global changes to the brain, the examination relies on the transtemporal window for its

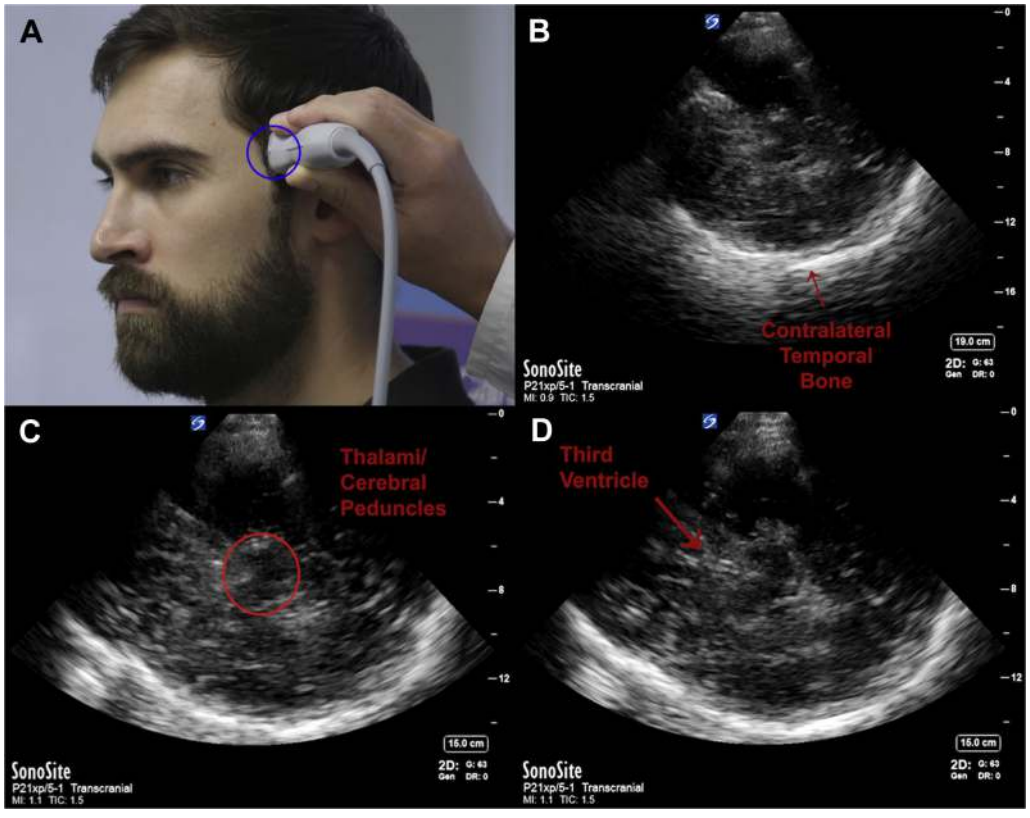

Figure 1 – *A-D, Basics of transcranial Doppler anatomy and image acquisition. A, The initial transducer position for the transtemporal window. B, Locating the contralateral temporal bone. C, Locating the midbrain. D, Locating the third ventricle.*

reliability and favorable Doppler alignment with the middle cerebral artery (MCA).

Begin the examination by finding the transtemporal window after applying ultrasound gel to the patient's temple (Fig 1A). The goal is to insonate the temporal bone at the level of the thinnest part of the skull, the pterion.[1] To find this point, place the transducer on the temporal bone at the level of the eye, just anterior to the patient's ear. With the index marker pointed anteriorly (toward the patient's eyes) (Fig 1A), use a sliding or sweeping motion to scan through the nearby brain tissue until the relevant intracranial structures (subsequently described) can be identified. These provide landmarks from which subsequent adjustments can be made.[13] A starting depth of 16 cm is typically reasonable.

The first important structure to identify is the temporal bone itself, seen in both the near and far fields. The ipsilateral temporal bone is typically seen at a depth of approximately 1 cm, whereas the contralateral temporal bone is more variable (depending on the width of the patient's skull), but often seen at a depth of 14 to 16 cm. Both project as bright, linear, hyperechoic structures, often with a slight curvature (more typical for the deep, contralateral temporal bone) (Fig 1B).

The next structure to identify is the midbrain. Two hypoechoic structures can typically be seen, the bilateral thalami and bilateral cerebral peduncles, which resemble a butterfly/heart (Fig 1C). Depending on the angle of insonation, generally the thalami can be seen (more superiorly) or the cerebral peduncles are visible (more inferiorly).

The next structure to identify is the third ventricle, a midline structure with a thin hypoechoic strip (representing cerebrospinal fluid) within thin, hyperechoic walls (Fig 1D). With both the ipsilateral and contralateral temporal bones in view, the third ventricle should be seen at exactly the midpoint of the two structures, assuming there is no MLS. In most patients, this represents a depth of 6 to 8 cm. With the temporal bones and third ventricle identified, the operator is now sure of having achieved an adequate transtemporal window and has basic landmarks available to guide the subsequent TCD examination.

Technique 2: Detecting MLS

With the basic structures identified, the first and most straightforward clinical application of TCD is to identify the presence or absence of MLS (Fig 2, Video 2).[16-18] As previously described, the third ventricle is expected to lie exactly halfway between the two temporal bones. If not, then the midline has been shifted, assuming measurements are made accurately. The MLS may be identified using two slightly different tools, both based on a similar principle. Start by placing the transducer in the transtemporal window as described in technique 1. Identify the temporal bones and third ventricle, and then adjust the transducer angle such that the ipsilateral and contralateral temporal bones appear parallel to each other on the screen. For the first tool, draw a calipered line connecting the two temporal bones. This line must bisect the third ventricle perpendicularly. Next, measure the temporal-to-temporal bone distance (Fig 2A, Video 2). Finally, measure the distance from the ipsilateral temporal bone to the middle of the third ventricle (Fig 2B). This distance should be exactly one-half the value of the temporal-to-temporal bone distance; if it is not, MLS should be suspected.

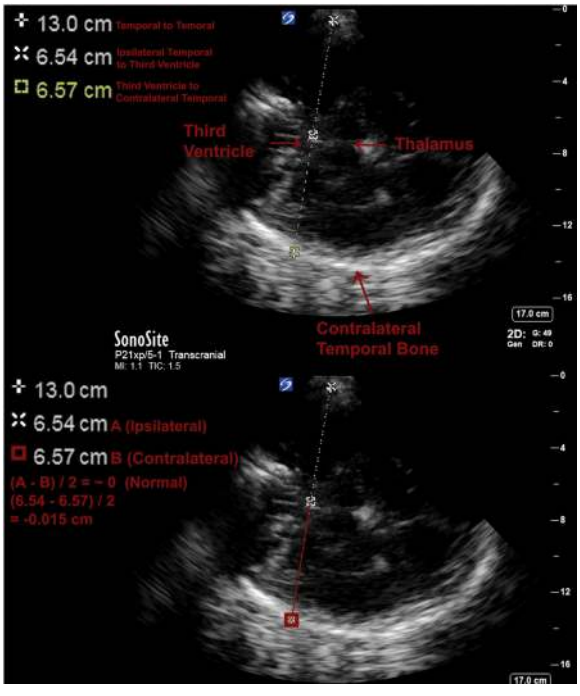

Figure 2 – *A-B, Detecting midline shift. A, First method to estimate midline shift. Measuring the distance from the ipsilateral temporal bone to the contralateral temporal bone (here 13.0 cm), and comparing it with the distance from the ipsilateral temporal bone to the third ventricle (here 6.54 cm). B, Second method to estimate midline shift. Measuring the distance from the temporal bone to the third ventricle on one side (here 6.54 cm), and comparing it with the same measurement taken on the other side (not shown).*

A second tool for detecting MLS involves making bilateral measurements. The distance from the ipsilateral temporal bone to the ipsilateral wall of the third ventricle is measured (distance A) (Fig 2B) and then compared with the same measurement on the contralateral side (distance B). The following equation is used to calculate MLS: MLS = (distance A – distance B)/2.

If the MLS value is positive, distance A is greater than distance B, and the midline is shifted away from the side where distance A was measured. If the MLS is negative, the shift is in the opposite direction. A zero value for MLS is normal, indicating no shift.

Limitations to MLS calculations, which apply to all TCD techniques in general, include thicker cranial vaults causing higher attenuation of the ultrasound by bone. Roughly 5% to 20% of patients will have difficult views leading to uninterpretable TCD images,[15,19] making this technique impossible because MLS measurements rely heavily on finding an appropriate transtemporal window. As noted initially, this technique should be considered hypothesis-generating, and findings should be confirmed by other modalities, usually CT scan, prior to altering management plans.

Technique 3: Color Doppler Interrogation of the MCA

The next set of applications involves interrogating the cerebral vasculature with color Doppler. Although all three major cerebral vessels can be interrogated using TCD, it is recommended that point-of-care providers concentrate on the MCA (Fig 3, Video 3).

Beginning from the basic view obtained in technique 1, depth of field is decreased such that the third ventricle is positioned in the far field rather than midscreen. The midbrain (specifically the bilateral thalami/cerebral peduncles) can often be detected at this point as a hypoechoic, butterfly/heart-shaped structure. The circle of Willis is located just anterior to the midbrain, on the left side of the ultrasound screen. A large color flow sampling box should be placed over the top one-half of the screen (in the near field) where the MCA is expected to be located, taking care to use a relatively low Nyquist limit (approximately 20-30 cm/s). The MCA is identified as a linear structure with blood flow directed toward the ultrasound transducer, visualized as a red color Doppler signal (Fig 3A).[1]

After identifying the MCA, pulsed-wave Doppler is used to measure the velocity of blood flow within the vessel.

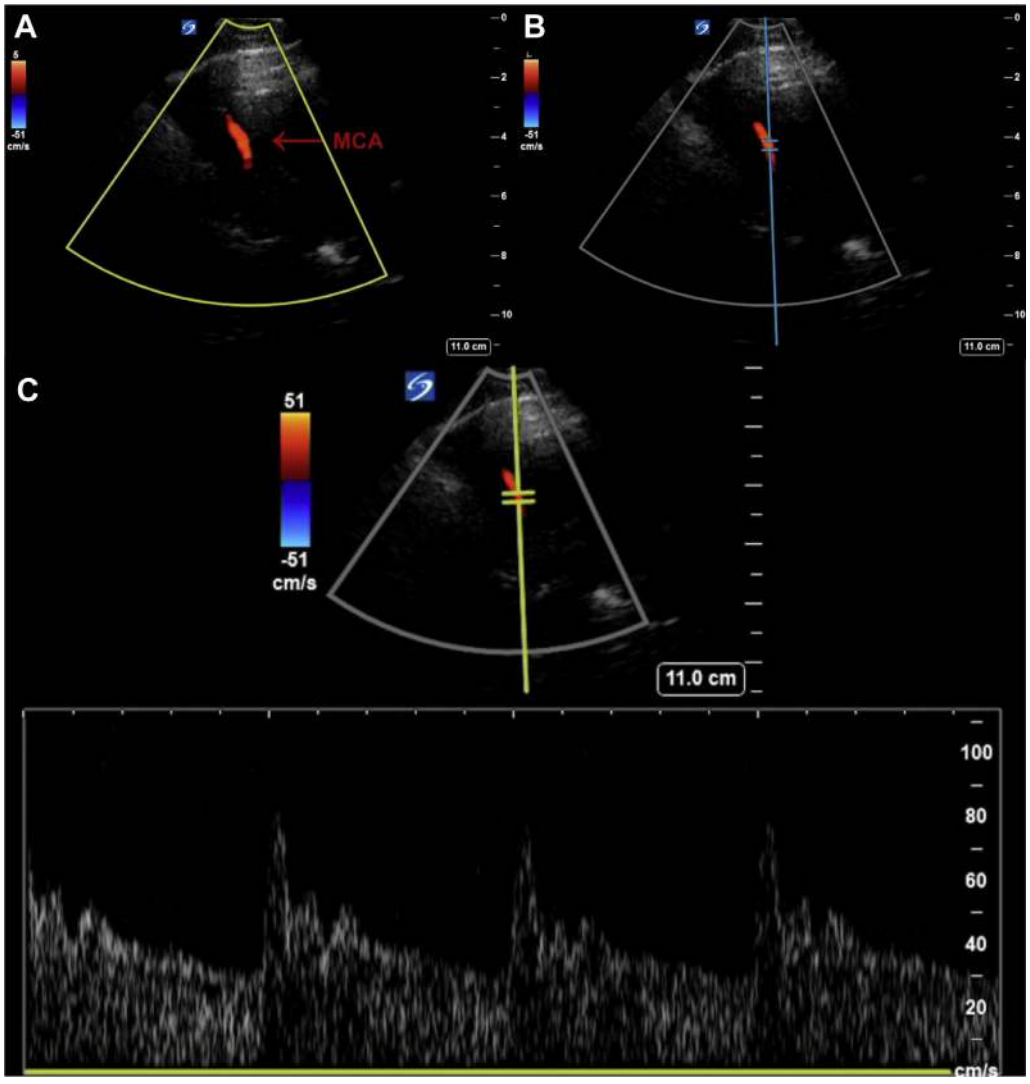

Figure 3 – *A-C, Color Doppler interrogation of the MCA. A, Locating the MCA with color Doppler, anterior (left of screen) of the midbrain. B, Interrogating the MCA with pulsed-wave Doppler. C, Resulting pulsed-wave Doppler tracing showing a steep systolic upstroke with a stepwise deceleration during diastole. MCA = middle cerebral artery.*

The Doppler sample gate is centered over the MCA's red color flow signal to obtain a spectral Doppler waveform (Fig 3B). The difference between the long axis of the MCA itself and the angle of insonation must be minimized (ideally to $< 10°$-$15°$), otherwise measured velocities will be underestimated. Some ultrasound machines offer angle correction software, but this is not recommended by professional organizations and generally should not be used.[7]

Normal MCA blood flow velocity shows a steep systolic upstroke with a stepwise deceleration during diastole (Fig 3C).[1,8,13] Now that an MCA spectral Doppler waveform has been obtained, it can be analyzed to glean clinically useful information.

Technique 4: Progression of Cerebral Circulatory Arrest and Estimating ICP

The most straightforward abnormalities to recognize are those associated with elevated ICP, which causes external compression of the cerebral vessels resulting in increased resistance to flow (Fig 4, Video 4).[1,7,20] There is a characteristic stepwise progression in the spectral Doppler tracing associated with raised ICP. The first phase is represented by an initial increase in MCA velocity during systole and a corresponding decrease in velocity, or blunting of flow, during diastole (Fig 4A). When ICP is severely elevated, diastolic flow may be absent or reversed.[20]

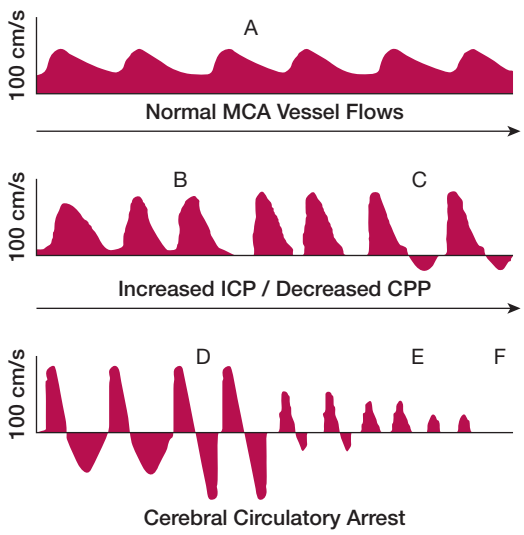

Figure 4 – *A-C, Progression of cerebral circulatory arrest. A, The progression from normal cerebral blood flow (marker A) in the middle cerebral artery. B, Peaked systolic flow (marker B) with blunting or reversal (marker C) of diastolic flow with raised intracranial pressure. C, Progression of cerebral circulatory arrest, from oscillating flow (marker D) to systolic spike flow (marker E), to complete absence of flow (marker F). CPP = cerebral perfusion pressure; ICP = intracranial pressure. See Figure 3 legend for expansion of other abbreviation.*

The end-stage of malignant intracranial hypertension is cerebral circulatory arrest and brain death. The progressive stages of cerebral circulatory arrest are illustrated in Figure 4A: (1) decreased or blunted diastolic flow; (2) oscillating flow, characterized by diastolic flow reversal roughly equal to forward systolic flow; (3) systolic spike flow, where diastolic flow is absent and systolic flow is slow (< 50 cm/s) and brief (< 200 milliseconds); and (4) complete absence of flow, in keeping with cerebral circulatory arrest.[8,20] Oscillating flow, systolic spike flow, and absence of flow are all consistent with a diagnosis of brain death.

Various methods have been described to estimate ICP from the MCA spectral Doppler waveform noninvasively.[1] To begin, interrogate the MCA with color and pulsed-wave Doppler exactly as described in technique 3. Once a spectral waveform of the MCA has been generated (Fig 4B), trace its contour to measure the velocity time integral (VTI) for one cardiac cycle (from the start of systole to the end of diastole). For ultrasound machines with TCD-specific software packages, choose the VTI preset that is often found under the calculations or measurements heading, depending on the specific machine brand. If no TCD software exists, the cardiac software package will have a VTI function (typically used to estimate stroke volume), which can also be used.

From this tracing, the ultrasound machine will derive the following measurements: peak systolic velocity (PSV), end-diastolic velocity (EDV), and mean velocity (MV) (sometimes called time-averaged velocity or time-average peak) (Fig 4B).[1,21] With values for PSV, EDV, and MV now in hand, ICP can be estimated by calculating the pulsatility index (PI) as follows[12]: $PI = (PSV - EDV)/MV$.

As PSV rises and EDV falls, PI (and therefore ICP) increases. This mirrors the expected changes in MCA blood flow pattern as discussed in technique 3. The PI can now be converted to an estimate of ICP using the following formula[20]: $ICP = (10.93 \times PI) - 1.28$.

Although PI values do not correlate exactly with absolute ICP values, in general, a PI value > 2 is pathologic, corresponding to an ICP value of > 20 mm Hg. A normal PI is typically < 1.2, corresponding to an ICP value of approximately 12 mm Hg. Unlike other measurements of MCA velocities which require on-axis measurements, the PI is relatively resistant to off-axis angles of insonation. This is because the PI is a relative ratio, meaning relative changes based on the angle of insonation for measurements should affect all measurements equally, preserving the PI ratio.[1,8,15,16]

There are important pitfalls for these techniques. The perceived absence of intracranial vascular flow could easily be because of a poor acoustic window or inappropriately interpreted images, where physicians could inappropriately misdiagnose brain death. Caution must be exercised with ICP estimation given its wide CIs (± 6 mm Hg in some studies) when compared directly with ICP monitors.[21,22] The technique is generally considered to be more useful for following trends in PI and ICP, rather than trying to measure an absolute ICP value at any given moment.[23] As such, TCD PI-derived ICP values should be considered as estimates, and an intracranial invasive monitor should be sought to confirm in the appropriate clinical setting.

These findings (particularly regarding cerebral circulatory arrest) should be substantiated with another examination (other ancillary testing such as nuclear medicine perfusion scanning, CTA, or MRI) before confirming the diagnosis. In North American jurisdictions, TCD is subservient to other modalities in confirming brain death; TCD cannot be used to confirm brain death on its own, but can help plan the optimal time for an ancillary test.[1] However, there are other jurisdictions (ie, Latin America, Spain) where TCD has been used to confirm brain death.[24,25]

Technique 5: Detecting and Quantifying Vasospasm

In many institutions, formal TCD is primarily used for the detection and quantification of cerebral vasospasm after subarachnoid hemorrhage (Fig 5, Video 5). Similar to techniques 3 and 4, this application is based on the interrogation of the cerebral vasculature with pulsed-wave Doppler. Although the anterior, posterior cerebral, and vertebral/basilar arteries can, in principle, be interrogated, we focus this article on utilization of point-of-care TCD for insonation of the MCA only as a screening test to rule-in vasospasm in the MCA. Because of the inherent lack of insonation for all arteries in cerebral circulation, insonation of the MCA alone should not comprehensive test to rule out vasospasm, and formal TCD can be sought.[1]

Start by identifying and interrogating the MCA, first with color Doppler and then with pulsed-wave Doppler, as previously described. Once a spectral Doppler tracing has been generated, perform a VTI trace of one cardiac cycle, as explained in technique 4. The ultrasound machine will then generate a series of values, the most important of which is the MV (or TAV/time-average peak).

Normal MV of the MCA is typically < 80 cm/s. Mild vasospasm MVs are in the 120 to 159 cm/s range, with velocities of 160 to 199 cm/s seen in moderate vasospasm and velocities of > 200 cm/s seen in severe vasospasm. Symptomatic clinical vasospasm is often only seen once MVs reach ≥ 160 cm/s.[26]

However, there are three major factors that may confound the interpretation of MCA MV. The first is cerebral atherosclerosis: if the vessel is chronically

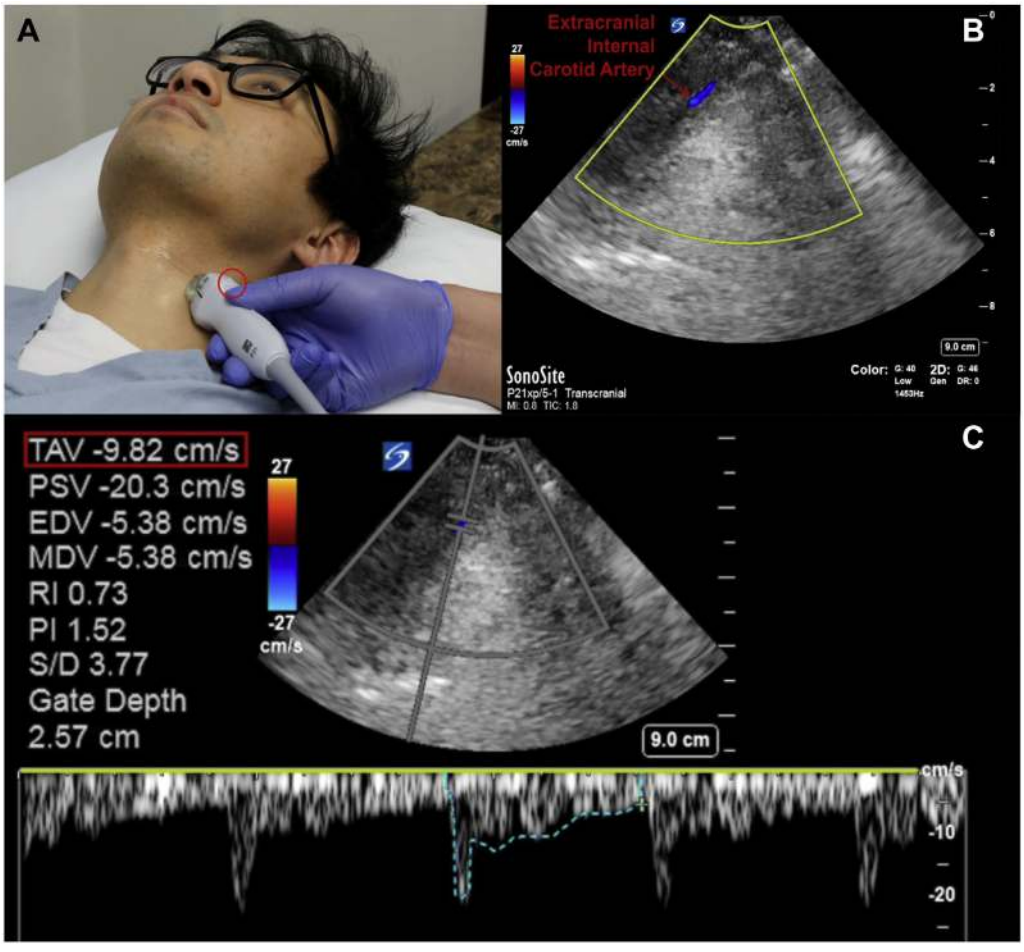

Figure 5 – *A-C, Detecting and quantifying vasospasm. A, The transducer position to measure velocity in the internal carotid artery. B, Color Doppler reveals the position of the internal carotid artery, with good alignment to measure the velocity from a parallel plane. C, Interrogation of the internal carotid artery with pulsed-wave Doppler and the resulting waveform. The machine software automatically calculates the TAV (also known as mean velocity). EDV = end-diastolic velocity; MDV = mean diastolic velocity; PI = pulsatility index; PSV = peak systolic velocity; RI = resistive index; S/D = systolic/diastolic ratio; TAV = time-averaged velocity.*

narrowed, blood flow will naturally be faster. The second factor is a hyperdynamic state: if the patient has an increased stroke volume or BP, MCA MV values will be higher. Finally, factors that cause an upstream decrease in blood flow, such as severe left ventricular systolic dysfunction, aortic stenosis, or ipsilateral carotid atherosclerotic disease, will falsely lower MCA MV.

Use of the Lindegaard ratio[27-29] can help mitigate the effect of some of these confounding factors. To calculate the Lindegaard ratio, the MV of the MCA is measured (as previously described) and then compared with the MV of the extracranial internal carotid artery (ICA) on the ipsilateral side.[1] The Lindegaard ratio formula is as follows[27-29]: Lindegaard ratio = ipsilateral MCA MV/ipsilateral extracranial ICA MV.

To measure the extracranial ICA velocity, place the phased-array transducer on the patient's neck, with the index marker pointed cranially (Fig 5A). Locate the carotid artery (noncompressible and pulsatile, compared with the compressible, nonpulsatile internal jugular vein), and slide the transducer up the patient's neck in a cranial direction until the bifurcation of the common carotid artery into the external and internal carotid arteries is identified. Bring the ICA into a position near parallel to the ultrasound beam, and then place a pulsed-wave Doppler sample gate in the middle of the ICA, while attempting to minimize the angle of insonation to $< 15°$. This is best achieved by rocking the transducer until the ICA is upright on the screen (Fig 5B). Once a spectral Doppler waveform is obtained, use the ultrasound machine's TCD software to perform a VTI trace of one cardiac cycle to generate the MV of the ipsilateral ICA (Fig 5C). This value is used as the denominator of the Lindegaard ratio.[27-29] A Lindegaard ratio > 3 is suggestive of vasospasm because this denotes an MCA MV that is increased relative to the carotid circulation. A Lindegaard ratio of 3 to 5 is consistent with mild to moderate vasospasm, whereas a ratio > 6 typically indicates severe vasospasm.[27-29]

TCD insonation for vasospasm has many of the same pitfalls and limitations as those previously mentioned for other techniques. Moreover, MCA velocity waveforms could be erroneously generated because of an incorrect angle of insonation, and it is important to minimize off-axis measurements which could underestimate the degree of vasospasm present. Findings can be confirmed with another imaging modality, typically formal TCD or CTA.

Conclusions

Specific TCD ultrasound techniques are available that can aid in the diagnosis and management of intracranial pathologies, including MLS, raised ICP, progression of cerebrocirculatory death, and cerebral vasospasm. The incorporation of these techniques by a competent provider at the point of care can significantly enhance the scope and precision of bedside neurologic care for the critically ill.

Acknowledgments

Financial/nonfinancial disclosures: None declared.

Other contributions: We thank Jack Li, MD, and Marco Balan, MD, for their contributions during filming production.

Additional information: The Videos can be found in the Supplemental Materials section of the online article.

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