Transcranial Doppler ultrasonography in neurological surgery and neurocritical care

Robert H. Bonow, MD,¹ Christopher C. Young, MD, PhD,¹ David I. Bass, MD, PhD,¹
Anne Moore, BSN, RVT,¹ and Michael R. Levitt, MD¹⁻⁴

Departments of ¹Neurological Surgery, ²Radiology, ³Mechanical Engineering, and ⁴Stroke and Applied Neuroscience Center, University of Washington, Seattle, Washington

Transcranial Doppler (TCD) ultrasonography is an inexpensive, noninvasive means of measuring blood flow within the arteries of the brain. In this review, the authors outline the technology underlying TCD ultrasonography and describe its uses in patients with neurosurgical diseases. One of the most common uses of TCD ultrasonography is monitoring for vasospasm following subarachnoid hemorrhage. In this setting, elevated blood flow velocities serve as a proxy for vasospasm and can herald the onset of ischemia. TCD ultrasonography is also useful in the evaluation and management of occlusive cerebrovascular disease. Monitoring for microembolic signals enables stratification of stroke risk due to carotid stenosis and can also be used to clarify stroke etiology. TCD ultrasonography can identify patients with exhausted cerebrovascular reserve, and after extracranial-intracranial bypass procedures it can be used to assess adequacy of flow through the graft. Finally, assessment of cerebral autoregulation can be performed using TCD ultrasonography, providing data important to the management of patients with severe traumatic brain injury. As the clinical applications of TCD ultrasonography have expanded over time, so has their importance in the management of neurosurgical patients. Familiarity with this diagnostic tool is crucial for the modern neurological surgeon.

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TRANSCRANIAL Doppler (TCD) ultrasonography is an inexpensive, noninvasive means of measuring blood flow within the arteries of the brain. First described by Aaslid and colleagues in 1982, indications for its use have expanded dramatically.² Today, TCD ultrasonography is routinely used to guide the management of patients with a range of pathologies, including occlusive cerebrovascular disease, subarachnoid hemorrhage (SAH), and trauma. The goal of this review is to outline the technology underlying TCD ultrasonography, the techniques used to perform TCD ultrasonography examinations, and common uses for the blood flow data that can be generated with TCD studies.

Technical Background

Doppler ultrasound detects motion using the difference in frequency between emitted ultrasonic waves and

the returning echoes.⁴⁹ This difference is proportional to the velocity of the moving object; if an object is stationary with respect to the transceiver, there is no Doppler shift. Importantly, only the component of velocity parallel to the ultrasound beam can be measured directly; any angle between the direction of movement and the beam must be mathematically accounted for. Inaccuracies can be introduced with this correction if the angle is incorrectly estimated, with error increasing as the angle approaches 90°. The depth of the insonated vessel can be estimated using pulsed-wave Doppler, in which the transceiver emits distinct acoustic pulses and measures the time required for their return. Using a technique termed “range gating,” modern equipment is able to selectively record signals from specific depths, allowing velocity to be recorded at distinct points along a vessel.³³

Velocities in all of the major intracranial vessels can

ABBREVIATIONS ACA = anterior cerebral artery; BA = basilar artery; CBF = cerebral blood flow; CPP = cerebral perfusion pressure; CVR = cerebral vascular resistance; DCI = delayed cerebral ischemia; eCVR = estimated cerebrovascular reserve; ICA = internal carotid artery; ICP = intracranial pressure; LR = Lindegaard ratio; MAP = mean arterial pressure; MCA = middle cerebral artery; MFV = mean flow velocity; PCA = posterior cerebral artery; PFO = patent foramen ovale; SAH = subarachnoid hemorrhage; TCD = transcranial Doppler; TEE = transesophageal echocardiography; VA = vertebral artery; VMR = vasomotor reserve.

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be measured using one of 3 “windows.” Because intracranial structures are not visualized using standard TCD ultrasonography equipment, identification of the vasculature requires the use of anatomical landmarks to properly angle the transceiver. The direction of flow at prespecified sampling depths further aids in vessel identification. Table 1 lists the parameters used in our laboratory for positive identification.

TCD findings are highly dependent on the experience and skill of the operator, who must manually position the probe to obtain measurements along the axis of the vessel by detecting the position with the highest velocity. Furthermore, variations in anatomy can make identification challenging if not impossible, particularly for the vertebral artery (VA) and basilar artery (BA). In approximately 10%–20% of patients, insonation of the terminal internal carotid artery (ICA), middle cerebral artery (MCA), anterior cerebral artery (ACA), and posterior cerebral artery (PCA) is not possible due to hyperostosis of the temporal bone.³⁴ Following a craniotomy, the temporal window may be impaired due to hemostatic agent or metal plates at the surgical site, or it may be enhanced due to bony defects.³³

Other physiological parameters can influence cerebral blood flow (CBF) velocity, including heart rate, blood pressure, hematocrit, and PaCO₂. These values should be recorded with the TCD velocities to allow for serial comparisons over time and to provide context for interpretation.

Subarachnoid Hemorrhage

A major complication of aneurysmal subarachnoid hemorrhage (SAH) is the development of vasospasm and delayed cerebral ischemia (DCI).¹² Between 30% and 70% of patients develop angiographic vasospasm after aneurysmal SAH, and half of these patients may develop symptomatic DCI,^{17,22,45} with the incidence peaking in the 2nd week after ictus.^{17,22} Hypertension, cigarette use, and SAH volume are associated with vasospasm and DCI, with the latter showing the strongest association.^{21,30}

Diagnosis of Vasospasm and DCI

Angiographic vasospasm typically precedes DCI.⁴⁵

Therefore, early detection of vasospasm may provide a window for intervention to prevent neurological deterioration.¹² In 2012, the American Heart Association guidelines for the management of SAH recommended the use of TCD ultrasonography for monitoring of development of arterial vasospasm with class IIa/level B evidence.¹²

Based on Bernoulli's principle, the velocity of blood flow in an artery is inversely related to the diameter of the artery.² During vasospasm, as the arterial diameter *decreases*, the velocity of blood flow *increases*. Therefore, changes in the velocity of blood flow can be used to estimate changes in the vessel diameter and detect vasospasm. Measured flow velocities can be compared to standard measurements and baseline values to infer changes in the vessel diameter and the presence of vasospasm.⁴⁵

Interpretation of TCD Parameters

Various parameters are measured and calculated during TCD ultrasonography to provide information regarding the vessel of interest. The mean flow velocity (MFV) is calculated from the peak systolic flow velocity (V_s) and end-diastolic flow velocity (V_d) according to the equation $MFV = (V_s - V_d/3) + V_d$ (Fig. 1).⁴⁵ In certain physiological states, intracranial velocities increase in the absence of spasm. The Lindegaard ratio (LR) normalizes the MFV of the MCA to that of the ICA to correct for this ($LR = MFV_{MCA}/MFV_{ICA}$).³⁹ In vasospasm, MFV_{MCA} increases as the MCA diameter decreases, while MFV_{ICA} remains unchanged, resulting in a higher LR. The pulsatility index can provide information regarding distal vascular resistance and intracranial compliance, although its clinical implications are less established (Fig. 2).

The correlation between TCD ultrasonography findings and angiographic vasospasm is most reliable for the MCA.^{23,38,66} An MFV < 120 cm/sec has a 94% negative predictive value, while an MFV > 200 cm/sec has an 87% positive predictive value.⁷¹ MCA vasospasm can be graded using the following criteria: mild, MFV > 120–150 cm/sec or LR 3.0–4.5; moderate, MFV > 150–200 cm/sec or LR 4.5–6.0; severe, MFV > 200 cm/sec or LR > 6.0.⁴⁵ Notably, TCD ultrasonography is less sensitive for ACA vasospasm, and patients with anterior communicating artery aneurysms are especially at high risk of false-negative

TABLE 1. Parameters used for positive identification of blood flow velocity in major intracranial vessels

Vessel	Window	Depth (mm)	Flow	Probe Angle	Normal Velocity (cm/sec)
Terminal ICA	Temporal	55–66	Toward	Anterosuperior	39 ± 9
MCA	Temporal	30–65	Toward	Anterosuperior	55 ± 12
ACA	Temporal	60–80	Away	Anterosuperior	50 ± 11
PCA-P ₁	Temporal	60–70	Toward	Posteroinferior	39 ± 10
PCA-P ₂	Temporal	60–80	Away	Posteroinferior	40 ± 10
Ophthalmic artery	Transorbital	40–60	Toward	15–20° medial	21 ± 5
ICA siphon	Transorbital	60–80	Variable	Variable	47 ± 14
BA	Transforaminal	80–120	Away	Midline	41 ± 10
Intracranial VA	Transforaminal	60–90	Away	Paramedian	38 ± 10
Extracranial VA	Inferior to mastoid	45–55	Variable	Lateral-inferior	38 ± 10
Extracranial ICA	Submandibular	30–60	Away	Posterolateral	37 ± 9

FIG. 1. Transcranial Doppler waveform showing elevated velocities in the left MCA, consistent with severe vasospasm. PI = pulsatility index.

findings.⁴⁵ ACA vasospasm is more reliably predicted by a 50% or 50 cm/sec increase in MFV over a 24-hour period.²⁶ For the PCA, difficulty with insonation renders absolute velocity readings unreliable.⁷³ Trends over time are likely more clinically relevant when assessing the ACA and PCA. As in the anterior circulation, velocities in the BA can be compared with those in the extracranial VA; BA/extracranial VA ratio > 3 is associated with significant BA vasospasm.⁵⁹ A recent meta-analysis of 17 studies including 2870 patients reported overall sensitivity of 90% and negative predictive value of 92% for the diagnosis of vasospasm with TCD ultrasonography.³⁵

Clinical Practice

At our institution, TCD ultrasonography studies are obtained in SAH patients daily until 14 days postictus. TCD ultrasonography is used as a screening tool, and its findings are correlated with the clinical examination. Patients with mild and moderate vasospasm on TCD ultrasonography used to be empirically treated with hyperemia and permissive hypertension, although recent studies have called this practice into question; prophylactic hypertension is now typically reserved for patients in whom neurological examination is difficult due to coma or intubation.²⁵ In patients with TCD ultrasonography evidence of vasospasm and new, lateralizing neurological deficit, digital subtraction angiography is often indicated for both diagnosis and treatment in the form of intraarterial vasodilators and/or balloon angioplasty. When the clinical examination and TCD ultrasonography findings are discordant or equivocal, CT angiography and CT perfusion can be used to determine candidacy for endovascular therapy.

Monitoring After Neurosurgical Procedures

Cerebrovascular Bypass

Extracranial-to-intracranial bypass may be performed to augment hypoperfused vascular territories or to replace vessels that must be sacrificed in the course of an operation.^{47,57} Low-flow bypasses have a volume flow rate of < 65 mL/min and typically involve smaller external carotid artery branches such as the superficial temporal artery directly anastomosed to intracranial vessels.⁴⁷ In high-flow bypasses, a conduit such as a radial artery or saphenous vein graft is interposed between the extracranial and intracranial vessels. Flow rates for high-flow bypasses range

FIG. 2. The pulsatility index serves as a proxy for resistance to flow and ICP. It is calculated by dividing the difference between peak systolic and trough diastolic velocities by the mean velocity. **A:** Transcranial Doppler waveform with a normal pulsatility index. **B:** Transcranial Doppler waveform with an elevated pulsatility index.

between 65 and 200 mL/min.⁵⁷ Postoperatively, brain perfusion is dependent on blood flow in the graft, and regular monitoring of flow rates can detect problems before the onset of ischemia. TCD ultrasonography is used to evaluate graft patency and quantify blood flow.⁴⁷

In low-flow bypasses, the narrow lumen frequently renders accurate assessment of cross-sectional area and thus volume flow rates challenging; peak velocities are monitored instead.⁴⁷ Duplex ultrasound is used to trace the graft from its origin to where it enters the skull, and peak velocities of the proximal, mid, and distal grafts are recorded. A focal increase in peak velocity or a slowed systolic upstroke suggests stenosis. For high-flow bypasses, the mean flow rate is around 130 mL/min.⁴⁸ Flow rates < 65 mL/min or > 200 mL/min are associated with ischemia and hyperemia, respectively, requiring further evaluation.⁴⁸

Endovascular Neurosurgery

TCD ultrasonography can be used to monitor for microembolic signals following endovascular aneurysm embolization and carotid stenting, as these signals are associated with the presence of postoperative ischemic lesions identified on diffusion-weighted imaging.^{7,11} Microemboli are high-intensity signals lasting < 0.1 second with intensity 3–60 dB above background (Fig. 3).⁶⁰ Emboli monitoring is often performed in the downstream artery after placement of an intracranial stent and after aneurysm coiling complicated by coil herniation into the parent vessel. Should a significant number of microembolic signals be detected, additional antithrombotic agents may be administered.

FIG. 3. Transcranial Doppler waveform demonstrating a high-intensity transient signal (arrow), consistent with microembolization. DM% = delta mean (monitoring mode); EDV = end-diastolic velocity; HR = heart rate; RI = resistance index; S/D = systolic/diastolic flow ratio.

Ischemic Stroke
Diagnosis and Etiology

While CT and MRI are the principal imaging modalities for the diagnosis of stroke, TCD ultrasonography can provide information about collateral flow, active embolization, subclavian steal, and even recanalization following thrombolytics.^{10,27,69} In the prehospital setting, paramedics may soon be able to use TCD ultrasonography as a screening tool for large vessel occlusions to assist with appropriate triage.⁶⁷

TCD ultrasonography may assist with identification of stroke etiology. For example, carotid artery atherosclerosis, carotid artery dissection, atrial fibrillation, prosthetic heart valves, and fat emboli have all been identified as thromboembolic sources that can cause large vessel occlusions, and these lesions are commonly associated with detection of microemboli on TCD ultrasonography.^{9,32,53} In contrast, intracranial stenosis from vasospasm or atherosclerosis elevates CBF velocities, and lacunar strokes appear to alter the pulsatility index.¹⁵ Diagnosis of these latter etiologies frequently relies principally on CT and MRI findings, but microembolic signals detected by TCD ultrasonography complement these studies.^{14,32} For example, in stroke patients in whom both carotid atherosclerosis and atrial fibrillation have been diagnosed, the presence of microemboli in multiple arterial territories suggests a cardiogenic, rather than atherosclerotic, stroke source.

Cryptogenic strokes account for 17% of ischemic strokes and tend to occur in a thromboembolic pattern.²⁸ As many as 38% of patients with cryptogenic strokes are found to have a right-to-left shunt, such as a patent foramen ovale (PFO), compared with just 18% in patients with a known

etiology.⁴⁶ These shunts can be identified by administering contrast-enhanced media, such as agitated saline, and then monitoring for bubbles on echocardiography or on TCD ultrasonography.¹⁴ Traditionally, transesophageal echocardiography (TEE) has been the gold standard in the workup of cryptogenic stroke as most shunts are PFOs and TEE allows for direct visualization of intracardiac shunts.³¹ However, TCD emboli monitoring may become the gold standard for detecting right-to-left shunts, as these studies appear to have superior sensitivity and lower cost and can detect pulmonary shunts and small PFOs missed on TEE.⁶⁸

Prognosis and Management

TCD microemboli monitoring provides prognostic value for monitoring symptomatic cerebrovascular disease. In one study, 45% of patients with symptomatic carotid artery stenosis had microembolic signals in the ipsilateral MCA, and the presence of these signals corresponded to a significantly increased risk of subsequent ipsilateral stroke and transient ischemic attacks.⁴⁴ Microembolic signals within the MCA also predict recurrent strokes caused by intracranial MCA-MCA embolization.²⁴

Microemboli monitoring on TCD ultrasonography has also been used to monitor response to medications intended as secondary prevention for stroke. For example, dual antiplatelet therapy has been shown to reduce the frequency of microembolic signals by 61% compared with medical therapy with aspirin alone.⁴² In this same study, 17 individuals (16%) had suffered recurrent stroke or transient ischemia attack by the 1-week follow-up. Although this endpoint was not significantly different between groups, the frequency of microemboli signals per hour in these 17 patients was significantly greater than the frequency in the other 90 patients in the study. Overall, these findings suggest that monitoring of asymptomatic emboli with TCD ultrasonography can be used to assess the response to medical therapy and, further, to guide escalation of treatment as needed in patients who have suffered thromboembolic stroke.

Microembolic signals are less common in patients with asymptomatic carotid artery stenosis but indicate a higher risk of ischemic stroke.³² TCD microemboli monitoring may identify a small subset of high-risk patients with asymptomatic carotid stenosis who could benefit from a carotid endarterectomy. In asymptomatic patients with at least 70% stenosis, patients with microemboli had a 2-year stroke risk of 3.6% compared with 0.7% in those without.⁴³ Similarly, TCD ultrasonography has been used in conjunction with bubble studies on TEE to quantify severity of right-to-left shunting in an attempt to identify patients who may benefit from surgical closure of a PFO.⁶⁸

In the setting of trauma, blunt cerebrovascular injury is seen in as many as 2.4% of patients admitted to the hospital with nonpenetrating injuries.^{8,61} These injuries confer a 5%–10% risk of stroke.^{19,56,62} The detection of microemboli distal to a high-grade carotid injury is associated with a 5-fold increase in the risk of stroke;⁸ TCD ultrasonography results may help to risk-stratify patients and guide the use of antithrombotic agents in this high-risk population.

Finally, arterial occlusions alter TCD waveforms in

a reliable, quantifiable manner that can yield important prognostic information. For example, improved flow signals and reduction of flow pulsatility following systemic thrombolysis can indicate recanalization of the occluded artery.⁶ These waveform properties were used to develop the Thrombolysis in Brain Ischemia (TIBI) scale as a way to quantify recanalization, and, thus, a therapeutic response, following thrombolysis.¹⁶ In comparison with the gold standard of angiography, TCD ultrasonography has been shown to be nearly 90% accurate.⁶⁹ Patients with a TIBI grade of 5, or full recanalization, within 6 hours of symptom onset are substantially more likely to be functionally independent 3 months following a stroke.⁶³ Additionally, preliminary research suggests that preservation of dynamic cerebral autoregulation, as identified by TCD ultrasonography, may predict better functional outcomes.⁴⁰

Primary Prevention in Sickle Cell Disease

Large-vessel vasculopathy can result in severe strokes following obliteration of the ICAs or MCAs in approximately 1% of children with sickle cell disease.³ Prophylactic blood transfusions can mitigate the process, but the sequelae from chronic transfusions prevent this strategy from being employed indiscriminately.⁵⁴ Peak velocities greater than 200 cm/sec confer a 10%–13% annual elevated risk of stroke,³ and prophylactic transfusions triggered by serial TCD ultrasonography data reduce the annual risk of stroke by 90%.⁵ Since peak velocities decrease after transfusion, continued TCD ultrasonography monitoring with transfusions as indicated has proven to be a reliable means of protecting high-risk children.⁴ More recently, TCD ultrasonography has been used to safely guide the transition of children from chronic maintenance transfusions to hydroxyurea for primary stroke prevention.⁷²

Cerebral Autoregulation Testing

Cerebral autoregulation refers to the brain's ability to maintain constant CBF in the face of varying cerebral perfusion pressure (CPP). In healthy individuals, the cerebral vascular resistance (CVR) varies with CPP to maintain constant CBF between mean arterial pressures (MAPs) of 60 mm Hg and 150 mm Hg.^{37,64} Assessment of cerebral autoregulation can provide information valuable in the management of a range of neurosurgical conditions, including cerebrovascular stenosis and traumatic brain injury. TCD ultrasonography is an inexpensive and noninvasive means for assessing cerebral autoregulation at the bedside. While these methods do not directly measure parenchymal CBF and autoregulation, velocity changes in trunk vessels assessed by TCD ultrasonography have been shown to correlate well with perfusion as assessed by SPECT.²⁰

Cerebral Autoregulation in Cerebrovascular Ischemia

In the setting of cerebrovascular atherosclerosis, resistance across a proximal stenosis decreases perfusion pressure to the downstream vascular territory. In order to compensate, the distal vasculature dilates, reducing overall CVR and preserving CBF. When the stenosis is critical, the distal vasculature may become maximally dilated, exhausting the ability of cerebral autoregulation to

maintain CBF in the face of further decreased perfusion. This state has been termed “misery perfusion” and has important prognostic implications. Among patients with symptomatic cerebrovascular disease, misery perfusion is associated with a 6-fold increase in the risk of subsequent stroke, highlighting the need for vigilance in the management of these patients.⁷⁴

Autoregulatory reserve can be tested by measuring blood flow velocity distal to a stenotic or occlusive lesion before and after the administration of agents that challenge the normal autoregulatory response. When autoregulation is not exhausted, resistance arterioles at the pial surface dilate, leading to an increase in CBF in the trunk vessels that manifests on TCD ultrasonography as heightened blood flow velocity. When autoregulation is exhausted, the change in velocity is blunted or even nonexistent. In severe cases, a paradoxical *decrease* in velocity may be observed due to a steal phenomenon; areas with intact autoregulation may recruit additional CBF, siphoning flow away from the region with exhausted autoregulation.³³

Inhaled carbon dioxide and acetazolamide are 2 agents that are commonly used to challenge autoregulatory reserve. Both act by decreasing the pH of extracellular fluid, which is a potent vasodilatory stimulus in the brain. Inhalation of CO₂ acts by increasing the dissolved partial pressure of CO₂ in blood, which in turn increases the concentration of carbonic acid, lowering the pH. In our laboratory, vasomotor reserve (VMR) is defined as follows: $VMR = (MFV_{\text{hypercapnia}} / MFV_{\text{baseline}}) \times 100 - (MFV_{\text{hypocapnia}} / MFV_{\text{baseline}}) \times 100$.⁵² The test is performed by administering 5% inhaled CO₂ and measuring MCA velocity after the end-tidal CO₂ increases by 10 mm Hg ($V_{\text{hypercapnia}}$). The patient then hyperventilates on room air until the end-tidal CO₂ is reduced to 25 mm Hg, and the MCA velocity measurement is repeated ($V_{\text{hypocapnia}}$). In normal individuals, MCA velocity increases by 2%–4% per mm Hg increase in PaCO₂.^{33,51} VMR < 15% is considered exhausted, 16%–38% severely reduced, 39%–69% moderately reduced, and ≥ 70% normal.

Acetazolamide is a carbonic anhydrase inhibitor that acts to block the conversion of carbonic acid to CO₂ and water, leading to a drop in pH. The drug is a potent stimulus for cerebral vasodilation and in healthy individuals can increase flow by 30%–60%. The estimated cerebrovascular reserve (eCVR) can be calculated using the following equation: $eCVR = (MFV_{\text{acetazolamide}} - MFV_{\text{baseline}}) / MFV_{\text{baseline}} \times 100$.³³

Cerebral Autoregulation in Trauma

Cerebral autoregulation impairment is present in between 49% and 87% of patients with severe TBI,⁵¹ placing these individuals at increased risk for ischemia with hypotension. In the absence of intact autoregulation, rapid increases in blood pressure may precipitate worsening cerebral edema, hemorrhage, and/or elevations in intracranial pressure (ICP). Disruptions in CBF are commonly seen in the tissue surrounding cerebral contusions, and hemispheric autoregulation tends to be more disrupted in the hemisphere containing mass lesions.^{41,55} Autoregulation impairment waxes and wanes over the course of a patient's ICU stay, with greater disruptions occurring early after in-

jury.³⁶ Impairments in autoregulation are associated with worse neurological outcome, particularly when unilateral or prolonged.^{29,50,51,70} Knowledge of autoregulation status facilitates tailored management of the TBI patient, as patients with intact autoregulation may see improvements in ICP control and brain tissue oxygenation with augmented CPP.

Two separate components of autoregulation can be assessed with TCD: the dynamic response, which considers the rate of autoregulatory compensation, and the static response, which is independent of time and measures compensation for sustained changes in CPP.

To assess dynamic autoregulation, a thigh blood pressure cuff is inflated 20 mm Hg above systolic blood pressure for 3 minutes and then rapidly deflated, prompting a transient drop in blood pressure. The dynamic autoregulatory index (dARI) can be calculated by comparing pre- and postdeflation MCA velocities using the formula: $dARI = (\Delta eCVR / \Delta T) / MAP$, where $eCVR = MAP / MFV_{MCA}$ and $T = \text{time}$.¹ In trauma patients, assessment of autoregulation by this method may be precluded by hemodynamic instability or extracranial injuries.

Static autoregulation is assessed by pharmacological augmentation of MAP. Vasoactive agents are infused to achieve a 20-mm Hg increase in CPP. The static autoregulatory index (sARI) is defined as $sARI = \% \Delta eCVR / \% \Delta CPP$.⁶⁵ When sARI is 0, cerebral autoregulation is exhausted and CBF changes linearly with CPP. Values ≤ 0.39 are considered impaired, and those ≥ 0.4 are considered intact. Static autoregulation may also be assessed using Mx, defined as the correlation coefficient between CPP and MFV; autoregulation is impaired when $Mx < 0.3$ and intact when ≥ 0.3 .¹³

Cerebral Circulatory Arrest

In some cases, determination of brain death requires ancillary studies in support of clinical assessments. Identification of cerebral circulatory arrest with TCD ultrasonography is one commonly used modality.⁵⁸ Once herniation has occurred and blood flow to the brain ceases, TCD ultrasonography may demonstrate reverberant flow, with equal antegrade systolic and retrograde diastolic velocities indicating the absence of net forward flow; narrow low-velocity systolic spikes, consistent with flow cessation at the carotid siphon; and/or absence of intracranial flow in a patient where Doppler signals had previously been detected.¹⁸ Two examinations separated by 30 minutes should be performed to confirm the diagnosis.¹⁸

Conclusions

The indications for TCD ultrasonography in neurological surgery and neurocritical care have expanded significantly since this monitoring tool was first introduced. Today, TCD ultrasonography is used routinely in the management of SAH, occlusive cerebrovascular disease, traumatic brain injury, and other diseases. Familiarity with this noninvasive monitoring technique is important to the modern neurosurgeon.

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Correspondence

Michael R. Levitt: c/o Neurosurgery Publications, University of Washington, Seattle, WA. respub@uw.edu.