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Vasospasm Surveillance With Transcranial Doppler Sonography in Subarachnoid Hemorrhage

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The use of transcranial Doppler monitoring for the diagnosis of vasospasm in subarachnoid hemorrhage is backed by national guidelines. However, it remains poorly used across neurologic intensive care units in the United States. This current practice article uses 2 clinical vignettes to illustrate the simplicity and logic behind routine daily surveillance of vasospasm with transcranial Doppler sonography in patients with subarachnoid hemorrhage, in preference to other modalities.

Key Words—neurosonology; subarachnoid hemorrhage; transcranial Doppler sonography; vasospasm

The Sound Judgment Series consists of articles highlighting the clinical value of using ultrasound in specific clinical diagnoses where ultrasound has shown comparative or superior value. In many cases, these articles support the practice of using ultrasound first. The series is meant to serve as an educational tool for medical and sonography students and clinical practitioners and may help integrate ultrasound into clinical practice.

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Abbreviations
CT, computed tomographic; MFV, mean flow velocity; PI, pulsatility index; SAH, subarachnoid hemorrhage

The use of transcranial Doppler monitoring for the diagnosis of vasospasm in subarachnoid hemorrhage (SAH) is backed by national guidelines. However, it remains poorly used across neurologic intensive care units in the United States. This current practice article uses 2 clinical vignettes to illustrate the simplicity and logic behind routine daily surveillance of vasospasm with transcranial Doppler sonography in patients with SAH, in preference to other modalities.

Case Descriptions

Case 1

A 66-year-old man underwent coil embolization of a large fusiform aneurysm of the distal right vertebral artery following a large SAH throughout the inferior basal cisterns, most prominent within the posterior fossa and perimesencephalic cisterns (Figure 1E). The patient required mechanical ventilation and external ventricular drainage. The patient's poor neurologic status (Glasgow Coma Scale score of 3) precluded a clinical suspicion or diagnosis of symptomatic vasospasm. Impaired renal function contraindicated contrasted studies. On day 6, vasospasm of the left middle cerebral artery was identified on transcranial Doppler sonography. Over the subsequent week, vasospasm of the left middle cerebral artery became severe, and it was also identified in bilateral anterior cerebral arteries and the right middle cerebral artery (Figure 1, A–D). The vasospasm responded to hyperdynamic therapy with intravenous fluids and vasopressors and resolved completely by day 12. The patient was eventually discharged to a skilled nursing facility with residual deficits secondary to delayed infarctions (Figure 1, F and G).

Case 2

A 56-year-old woman was admitted to the neurologic intensive care unit with extensive SAH in the basal cisterns (Figure 2, A–C), and a diagnostic angiogram was unrevealing. The patient was intubated and sedated on a mechanical ventilator with a poor neurologic examination (Glasgow Coma Scale score of 5). Serial transcranial Doppler studies revealed an upward trend in the pulsatility index (PI) and progressively increasing spectral resistivity from days 1 through 4 (Figure 2, E and F). The patient's neurologic examination worsened on day 6 (Glasgow Coma Scale score of 3), and transcranial Doppler sonography showed reverberating waveforms in bilateral middle cerebral arteries (Figure 2G). Other first-order vessels revealed nonreverberating, albeit

Figure 1. **A**, Mild spasm and hyperemia of the right middle cerebral artery (RMCA; >120 cm/s; Lindegaard ratio, 3.2). **B**, Severe spasm of the left middle cerebral artery (LMCA; >200cm/s; Lindegaard ratio, 6.6). **C**, Definite vasospasm of the right anterior cerebral artery (RACA; >120 cm/s; Sloan hemispheric ratio, 5.3). **D**, Definite vasospasm of the left anterior cerebral artery (LACA; >120 cm/s; Sloan hemispheric ratio, 5.5). **E**, Head CT. There is diffuse SAH throughout the inferior basal cisterns, most prominent within the posterior fossa and perimesencephalic cisterns. It is seen over the cerebral convexities more prominently anteriorly than posteriorly. **F** and **G**, Follow-up head CT showing subacute infarctions of the left frontoparietal territory.
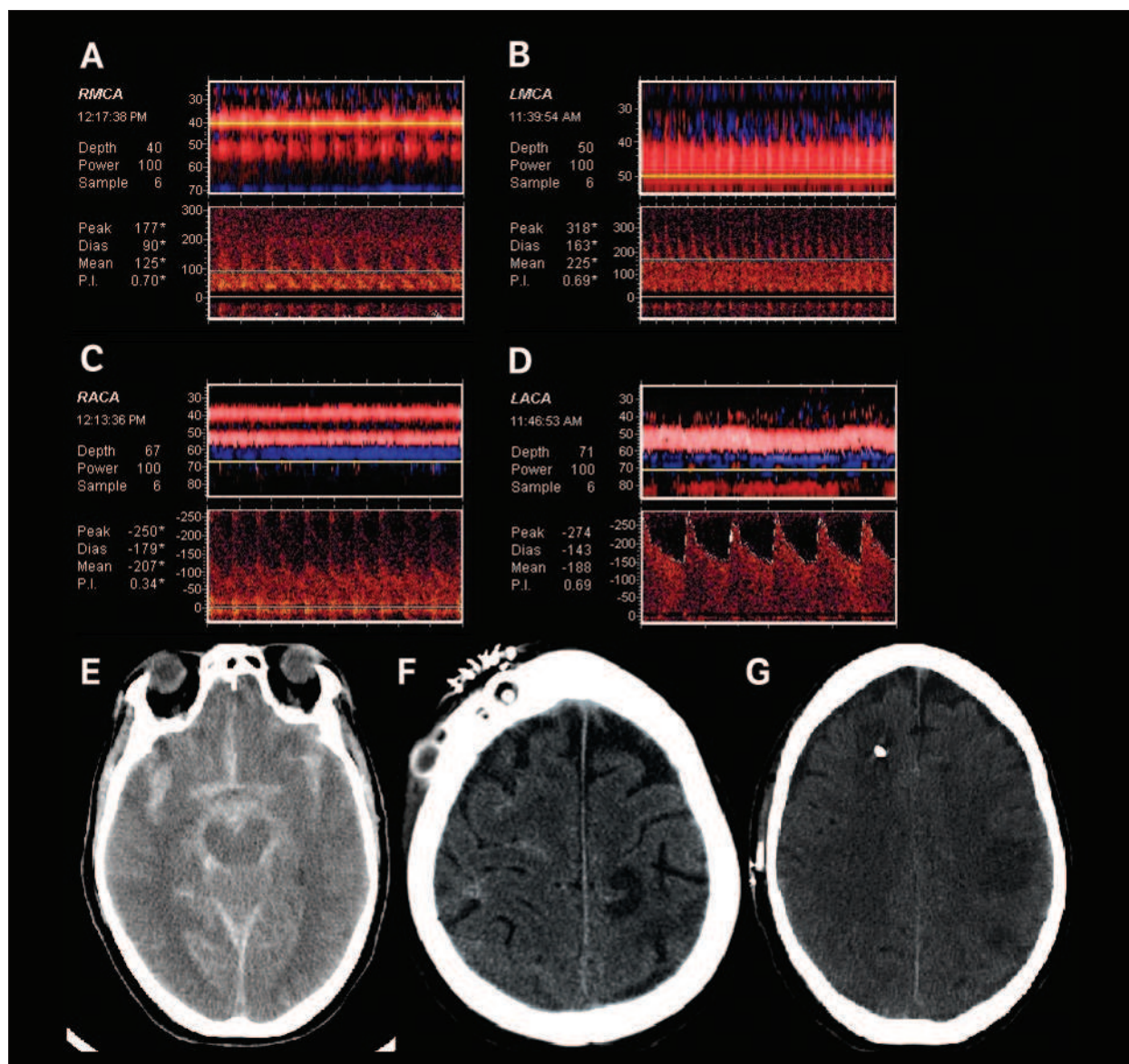
resistive, spectra. A subsequent noncontrast computed tomographic (CT) scan of the head revealed diffuse cerebral edema and ventricular compression (Figure 2D). Aggressive institution of osmotic therapy with mannitol boluses and escalation with infusion of hypertonic saline led to clinical improvement and normalization of abnormal waveforms (Figure 2H) by the next day. The patient was eventually able to be discharged to an inpatient rehabilitation facility.

Rationale Behind the Use of Daily Transcranial Doppler Monitoring in Preference to Other Diagnostic Modalities

Transcranial Doppler monitoring (available at the bedside) is an extension of the neurologic examination in patients with SAH and should not be considered optional. Alternative means of diagnosing vasospasm, such as conventional angiography and CT angiography, cannot be obtained daily, are invasive, entail nephrotoxic contrast agent administration, and require moving a critically ill patient to the angiography/CT suite. These limitations render these techniques poor surveillance tools for diagnosing vasospasm in the presymptomatic phase (when clinical suspicion is absent) and when clinical suspicion is unreliable (poor neurologic examination). Transcranial Doppler sonography, therefore, is the surveillance device of choice in patients with SAH. Even patients with poor temporal bone windows (≈10%) benefit from transcranial Doppler surveillance. Posterior vasculature (basilar artery and vertebral arteries) can be insonated through the foraminal window. The overall yield of vessel identification is improved by the use of power motion Doppler, or M-mode, imaging. This window-finding tool simultaneously displays the flow intensity and direction over several centimeters of intracranial space (see transcranial Doppler images in Figures 1 and 2). An advantage offered by this mode of insonation is to display all flow signals obtainable at a given position and direction of the transducer.

Figure 2. **A–C**, Computed tomograms on admission revealing diffuse SAH throughout the basal cisterns. There is no ventriculomegaly. **D**, Follow-up CT on day 6 revealing diffuse cerebral edema and ventricular compression. A left thalamic infarction is also noted. **E**, Normal middle cerebral artery waveforms on day 1. **F**, Resistive spectra seen on middle cerebral artery insonation on day 4. Pulsatility indices were abnormally high bilaterally in both anterior and posterior circulation vessels, suggestive of diffusely increased intracranial pressure. There was no associated clinical change. **G**, Reverberating spectra seen in the middle cerebral artery on day 6, which raised concern for impending cerebral circulatory arrest (from increased intracranial pressure), since the posterior circulation spectra, albeit resistive, were not reverberating. This appearance coincided with clinical worsening to a Glasgow Coma Scale score of 3. **H**, Normalization of spectra with aggressive institution of osmotic therapy on day 7. The patient had clinically improved.
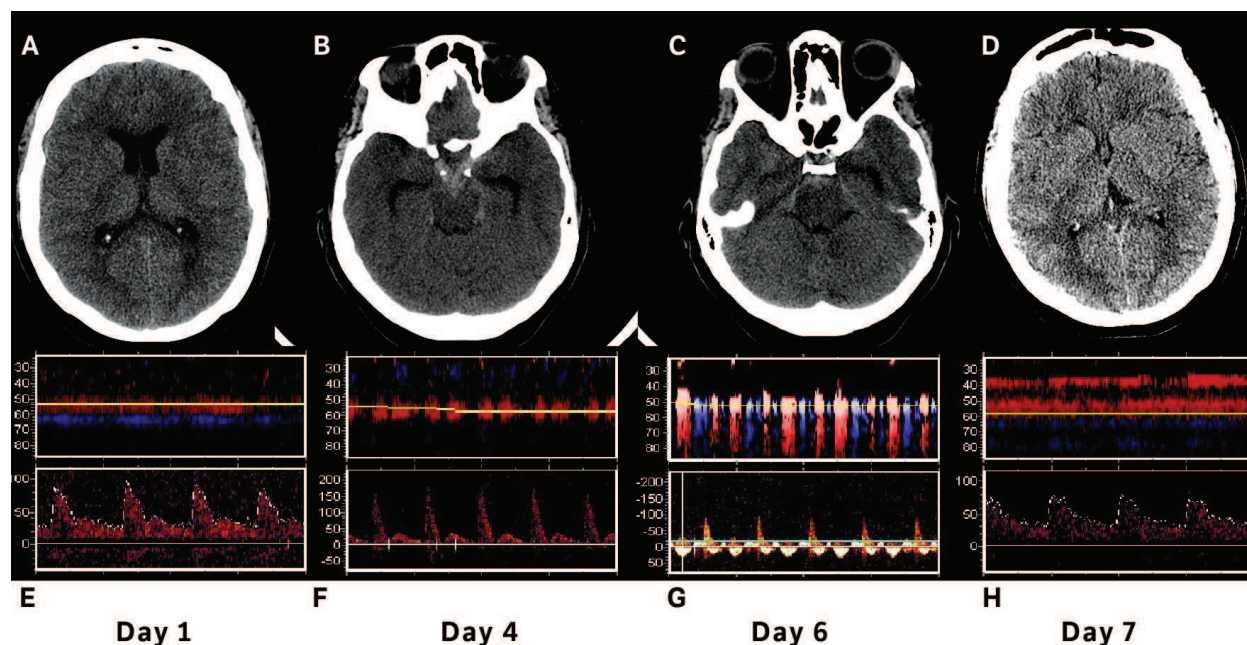
Comparison of Transcranial Doppler Sonography to a Reference Standard Is Counterintuitive

Since vasospasm is dynamic and can worsen or improve over time, it follows that a dynamic surveillance device would be the most appropriate tool for monitoring it. Neither CT angiography/CT perfusion nor conventional angiography can be used for daily routine monitoring to diagnose subclinical or clinical vasospasm or to monitor the response to treatment. Historically, the diagnostic accuracy of transcranial Doppler sonography has been compared to conventional angiography, which is often considered the reference standard. We posit that since the angiogram is but a "snapshot" in time (not repeatable like transcranial Doppler) and transcranial Doppler sonography is a dynamic monitoring device, conventional (or CT) angiography is not a valid reference standard for transcranial Doppler sonography. Absence of vasospasm on a snapshot study (conventional or CT angiography) cannot be taken as evidence of absence of vasospasm.

The physiologic information obtained from transcranial Doppler sonography provides diagnostic data that can be followed objectively on a time line, unlike the "all-or-none" diagnostic value of an angiogram. In addition, transcranial Doppler sonography also indicates other pathophysiologic consequences of SAH, such as raised intracranial pressure and cerebral circulatory arrest. Trends in PIs on successive transcranial Doppler studies can indicate changes in intracranial pressure.[1,2] The transcranial Doppler-derived PI should be considered reliable even when obtained by a relatively inexperienced neurosonographer, as it remains independent of the angle of insonation. Deterioration of waveforms into reverberating (oscillating) patterns or systolic spikes can indicate cerebral circulatory arrest.[3] Therefore, the use of transcranial Doppler surveillance fosters a holistic approach to the management of SAH, providing the treating physicians and surgeons with a continuum of clinically usable cerebrovascular hemodynamic information.

Recommended Practice

The goal of vasospasm surveillance is to improve overall patient outcomes through the detection of vasospasm before it becomes clinically manifest so that specific treatments are initiated and symptomatic ischemia prevented or timely treated, consequently preventing development of ischemic stroke, disability, and death. It is known that effective treatment of vasospasm can improve clinical outcomes,[4–8] and the challenge is to find pathways to quickly identify vasospasm as it is developing so that these therapies can be instituted, allowing their benefit to be provided before the destruction of brain tissue.[9] Daily transcranial Doppler monitoring, beginning from the day of diagnostic angiography (CT or conventional) through the day of discharge, not only successfully captures preclinical vasospasm but also guides workup and treatment of patients suspected of having raised intracranial pressure from an upward trend in PI values. A focal increase in PI, indicative of a focal increase in intracranial pressure (mass effect), is valuable in guiding follow-up CT scans and at times can lead to successful hematoma evacuation. This factor is especially true for patients in whom neurologic examination is poor and cannot guide management. It is also useful in capturing reverberating or oscillating waveforms and systolic spikes that are indicative of impending or definite cerebral circulatory arrest, allowing physicians to either escalate care or initiate a brain death evaluation.

Diagnostic Criteria for Transcranial Doppler Evidence of Vasospasm

The evidence base is stronger for the criteria used to diagnose middle cerebral artery and basilar artery vasospasm (Tables 1 and 2), and weaker for other arteries in the circle of Willis (Table 3). The Lindegaard ratio is an index used to correct for a diffuse systemic increase in flow velocities secondary to a hyperdynamic circulatory state (iatrogenic from hyperdynamic therapy or physiologic from fever, anemia, increased cardiac output, etc) in patients with SAH. To calculate the Lindegaard ratio, the mean flow velocity (MFV) of the middle cerebral artery is divided by the MFV of the ipsilateral extracranial proximal internal carotid artery, which is measured by using a 2.0-MHz transducer for insonation. This index helps distinguish global hyperemia from vasospasm, especially in the setting of hyperdynamic therapy. The Sviri ratio is the basilar artery counterpart of the Lindegaard ratio (Table 2). It is calculated by dividing the MFV of the basilar artery by an average of the time-averaged maximum MFV of both the extracranial vertebral arteries. The MFV of extracranial vertebral arteries is measured by using a 2.0-MHz pulsed wave transducer below the tip of the mastoid process at a depth of 40 to 50 mm. In practice, it is more challenging due to difficulty in discriminating the vertebral artery from the occipital artery and from the nearby internal carotid artery. Probe pressure obliterates the occipital artery, and confusion with internal carotid artery is avoided by maintaining a posterior probe position or moving anteriorly to identify the internal carotid artery as separate from the vertebral artery.

Conclusions

The use of transcranial Doppler sonography for surveillance of vasospasm in SAH is suboptimal despite recommendations by national guidelines (American Heart Association/ American Stroke Association, American Academy of Neurology, and Neurocritical Care Society).[17–22] Routine use of daily transcranial Doppler monitoring in SAH not only makes mechanistic sense but can also positively impact clinical outcomes.

Table 1. Transcranial Doppler Grading Criteria for Middle Cerebral Artery Vasospasm[10,11]

MFV, cm/s	MCA/EC ICA MFV (Lindegaard) Ratio	Interpretation
<120	≤3	Hyperemia
>80	3–4	Hyperemia + possible mild spasm
≥120	3–4	Mild spasm + hyperemia
≥120	4–5	Moderate spasm + hyperemia
>120	5–6	Moderate spasm
≥180	6	Moderate-to-severe spasm
≥200	≥6	Severe spasm
>200	4–6	Moderate spasm + hyperemia
>200	3–4	Hyperemia + mild/residual spasm
>200	<3	Hyperemia

EC indicates extracranial; ICA, internal carotid artery; and MCA, middle cerebral artery.

Table 2. Transcranial Doppler Grading Criteria for Basilar Artery Vasospasm[12]

MFV, cm/s	BA/EC VA MFV (Sviri) Ratio	Interpretation
>70	>2	Vasospasm
>85	>2.5	Moderate or severe vasospasm
>85	>3	Severe vasospasm

BA indicates basilar artery; EC, extracranial; and VA, vertebral artery.

Table 3. Transcranial Doppler Grading Criteria for Internal Carotid Artery, Anterior Cerebral Artery, Posterior Cerebral Artery, and Vertebral Artery Vasospasm[11,13–15]

| Artery | MFV, cm/s | | |
	Possible Vasospasm	Probable Vasospasm	Definite Vasospasm
ICA	>80	>110	>130
ACAa	>90	>110	>120
PCA	>60	>80	>90
VA	>60	>80	>90

ACA indicates anterior cerebral artery; ICA, internal carotid artery; PCA, posterior cerebral artery; and VA, vertebral artery.
aIn the presence of ipsilateral middle cerebral artery or internal carotid artery vasospasm, the Sloan hemispheric ratio (anterior cerebral artery/ extracranial internal carotid artery >4) is used for diagnosis instead.[16]

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