RadioGraphics

Advances in Transcranial Doppler US: Imaging Ahead[1]

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ONLINE-ONLY SA-CME

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LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Describe the advantages of gray-scale, spectral Doppler, and color Doppler flow imaging for evaluating the intracranial vasculature.
- Explain key physical principles underlying Doppler imaging.
- Identify clinical settings in which transcranial Doppler US is a useful diagnostic study.

TEACHING POINTS

See last page

Transcranial Doppler ultrasonography (US) is a noninvasive, portable technique for evaluating the intracranial vasculature. It has found its most useful clinical application in the detection of vasospasm involving the cerebral vessels after subarachnoid hemorrhage due to aneurysm rupture. The technique has become an integral part of monitoring and managing patients with subarachnoid hemorrhage in the neurologic intensive care unit. In addition, it has proved useful for evaluating the intracranial vasculature in patients with sickle cell disease, stroke, or brain death. Transcranial US originated as a "blind" nonimaging study in which pulsed Doppler technology was used. Identification of the major intracranial vessels and evaluation of those vessels for vasospasm relied on spectral waveforms obtained in each vessel and was based on the depth of the vessel from the skull, the direction of blood flow, and the orientation of the transducer. Recent advances in US technology allow the use of gray-scale, spectral Doppler, and color Doppler flow imaging to directly visualize intracranial vessels, thereby simplifying flow velocity measurements and enhancing their accuracy for vasospasm detection. In particular, measurements of peak systolic velocity and mean flow velocity and calculation of the Lindegaard ratio facilitate the identification of vessels that may be in vasospasm and help differentiate vasospasm from physiologic conditions such as hyperemia and autoregulation. Thus, gray-scale and color Doppler flow imaging offer many advantages over the original pulsed Doppler technique for evaluating the intracranial vasculature.

Introduction

Subarachnoid hemorrhage due to aneurysm rupture can lead to vasospasm of cerebral vessels, which is associated with a 15%–20% risk of stroke or death (1). Radiographically detectable vasospasm has been estimated to occur in 50%–70% of patients after aneurysm rupture, with approximately half of those affected manifesting clinical signs and symptoms usually within 3–14 days after onset of hemorrhage (2). Medical treatment of these patients consists of induced hypertension, hemodilution,

Abbreviations: ACA = anterior cerebral artery, BA = basilar artery, EDV = end-diastolic velocity, ICA = internal carotid artery, MCA = middle cerebral artery, MFV = mean flow velocity, OA = ophthalmic artery, PCA = posterior cerebral artery, PSV = peak systolic velocity, RI = resistive index, VA = vertebral artery

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Table 1
Protocol for Doppler US Spectral Measurement of the Intracranial Vasculature

Artery*	Window	Depth (mm)	Transducer Orientation	Flow Direction†	MFV (cm/sec)‡
OA	Orbital	40–50	Slightly medial	Toward	16–26
MCA	Temporal	35–60	En face	Toward	46–86
ACA	Temporal	60–75	Anterior	Away	41–76
PCA	Temporal	60–75	Posterior	Toward	33–64
VA	Transforaminal	45–75	Superior and oblique	Away	27–55
BA	Transforaminal	70–120	Superior	Away	30–57

Note.—Before gray-scale and color Doppler flow imaging techniques were developed, US evaluation of the intracranial vasculature was performed by using this complex spectral measurement technique, which did not allow direct visualization of the vessels. Vessels were identified by using information about their depth and orientation and the directionality of blood flow through the given acoustic window.
*BA = basilar artery, OA = ophthalmic artery, VA = vertebral artery.
†The directionality of flow is characterized as toward or away from the transducer.
‡Normal MFV ranges specified by Aaslid et al (9) are shown.

and hypervolemia to improve cerebral perfusion. If this initial medical therapy is ineffective, a vasodilator may be administered by intraarterial injection. Potential complications related to treatment, such as renewed bleeding from the aneurysm or into areas of infarction, increased cerebral edema, congestive heart failure, and myocardial infarction, preclude prophylactic treatment (3). Therefore, careful monitoring of patients with subarachnoid hemorrhage is necessary to detect vasospasm.

Conventional angiography permits the detection and treatment of vasospasm but carries added risks of complications associated with the invasiveness of the procedure. Computed tomographic (CT) angiography allows noninvasive assessment of vasospasm and has demonstrated good sensitivity and specificity for the detection of severe vasospasm, as well as a high negative predictive value, but its sensitivity may not be as good for mild vasospasm as for severe vasospasm (4,5). Both techniques involve potential health risks because of the use of contrast material (eg, anaphylactoid reactions, nephrotoxicity) and exposure to ionizing radiation. In addition, both of these procedures require that the patient be transported (generally from the intensive care unit) to the radiologic imaging department.

Teaching Point

Transcranial Doppler ultrasonography (US) is a noninvasive technique that allows accurate and sensitive evaluation of the cerebral vessels for vasospasm without requiring the use of a contrast medium, exposure of the patient to ionizing radiation, or transport of the patient out of the intensive care unit (6–8).

Aaslid et al (9) first demonstrated that cerebral arterial flow velocities could be measured by performing pulsed Doppler US with a transtemporal approach. The technique he developed was "blind" in that there was no direct visualization of the vessels in which flow velocities were being measured. Instead, the identity of the vessel being interrogated was determined by using a complex approach, on the basis of its depth from the skull, the transducer orientation, and the flow direction. Aaslid determined normal mean flow velocity (MFV) ranges for the middle cerebral artery (MCA), anterior cerebral artery (ACA), and posterior cerebral artery (PCA) (Table 1) (9,10). However, the inability to perform angle correction, along with the limitations imposed by the lack of direct visualization, may have led to errors in the velocities recorded with this technique.

The present article describes recent advances in transcranial Doppler US involving the use of gray-scale, spectral Doppler, and color Doppler flow imaging techniques. With the use of these techniques, direct visualization of the major intracerebral vessels is possible, allowing quick, accurate vessel identification and flow velocity measurements; in addition, color Doppler flow imaging allows easy identification of the flow direction.

Teaching Point

Transcranial gray-scale and color Doppler flow imaging not only have utility for detecting vasospasm but also show promise for evaluating the intracranial vasculature in patients with stroke, sickle cell disease, carotid artery occlusion, stenosis of intracranial vessels, and brain death (11,12).

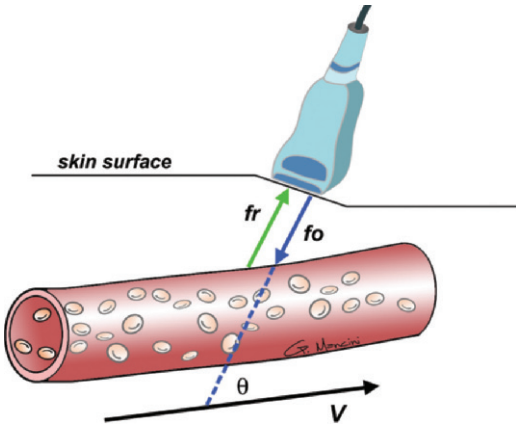


Figure 1. Diagram shows the Doppler effect. Sound waves emitted at a specific frequency *(fo)* are reflected off moving red blood cells and back to the transducer at a higher or lower frequency *(fr)*. The difference in frequencies, known as the Doppler shift, can be used to calculate the blood flow velocity *(V)* and direction. θ = angle between the incident ultrasound beam and the direction of blood flow.

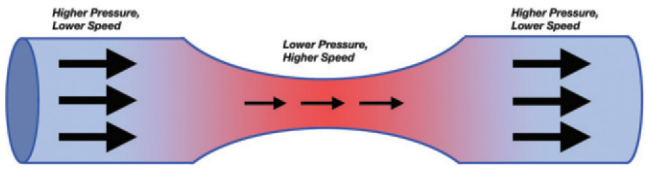


Figure 2. Diagram of the Bernoulli principle shows that as fluid flows from a conduit or vessel of greater diameter to one of lesser diameter, the velocity of flow increases and the pressure decreases to allow the same volume of fluid to pass through the narrower area. Thus, the velocity of blood flowing through areas of stenosis secondary to vasospasm in the setting of subarachnoid hemorrhage is expected to be higher than that of blood flowing through adjacent vessels.

The following sections review principles of physics that are relevant to Doppler US, techniques and protocols used to perform transcranial Doppler imaging at the authors' institution, and the various clinical entities that may be identified with these techniques.

Physics of Doppler US

Transcranial Doppler imaging, like all Doppler imaging studies, is based on two hemodynamic principles: the Doppler effect and the Bernoulli principle. The Doppler effect, first observed and described by Christian Doppler in the mid-1800s, accounts for what happens when a sound wave with a certain frequency (FO) strikes a moving object (eg, red blood cells at transcranial Doppler imaging): the sound wave is reflected from the object at a different frequency (FR). The Doppler equation describes the relation between the resultant frequency shift (ie, FD = FR – FO) and the velocity (v) of blood flow as $v = (c \cdot FD)/2 \cdot FO \cdot \theta$, where c is the speed of sound and θ is the angle between the incident ultrasound beam and the direction of blood flow within the vessel (Fig 1). (For optimal velocity measurements, θ should be less than 60°.) This equation allows one to calculate not only the velocity but also the direction of blood flow through a vessel. A shift to a higher frequency is a positive Doppler shift, indicating flow toward the transducer; a shift to a lower frequency is a negative Doppler shift, indicating flow away from the transducer.

The Bernoulli principle describes the flow of liquids through conduits of differing diameters with respect to velocity and pressure (Fig 2). As a liquid (eg, blood) moves from a conduit with a larger diameter to one with a narrower diameter, the blood flow velocity must increase (and the pressure decrease) to allow the same volume of blood to flow through the narrower area. Vessel segments affected by vasospasm are narrower; therefore, the velocities of blood flowing through these vessel segments must increase. The Doppler effect and Doppler equation allow measurement of this increase in velocity and, thereby, detection of vasospasm.

Teaching Point

A spectral Doppler waveform is a visual display of blood flow velocities within a specified area of a blood vessel (ie, sample volume) as a time-velocity curve throughout the cardiac cycle. From the waveform, peak systolic velocity (PSV) and end-diastolic velocity (EDV) can be measured (Fig 3). These values can be used to calculate the MFV with the formula MFV = PSV + (EDV · 2)/3. They also may be used to obtain the RI by using the equation RI = (PSV – EDV)/PSV. The MFV is essentially the average flow velocity over the time-velocity waveform. Threshold MFV values have been determined that correlate with the absence or presence and severity of vasospasm (see the section on "Vasospasm"). RI is a measure of resistance to the flow of blood into an organ: As the resistance increases, the flow of blood during diastole decreases; thus, the EDV drops toward zero as the RI approaches 1.0. The closer the RI is to 1.0, the greater is the resistance to blood flow.

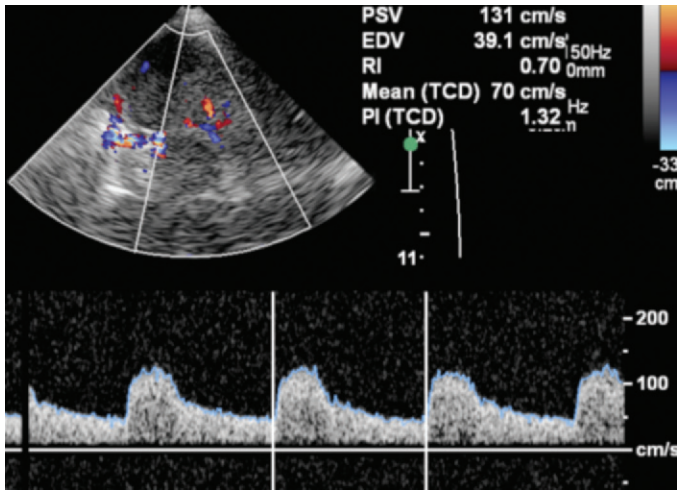
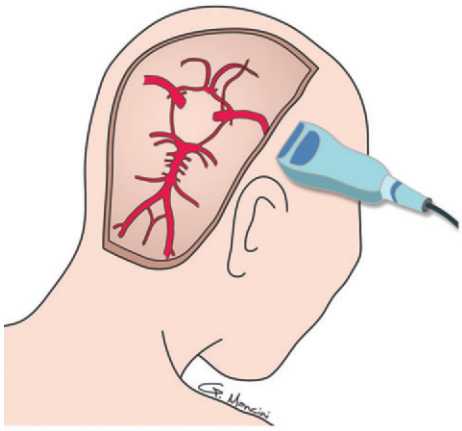


Figure 3. Duplex Doppler image and spectral waveform show the velocity of blood flowing within the PCA. The PSV, EDV, MFV, and resistive index (RI) are automatically calculated from the waveform. *PI* = pulsatility index, *TCD* = transcranial Doppler.



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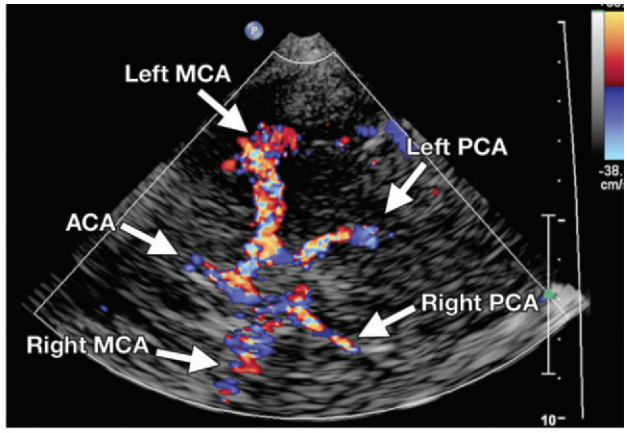

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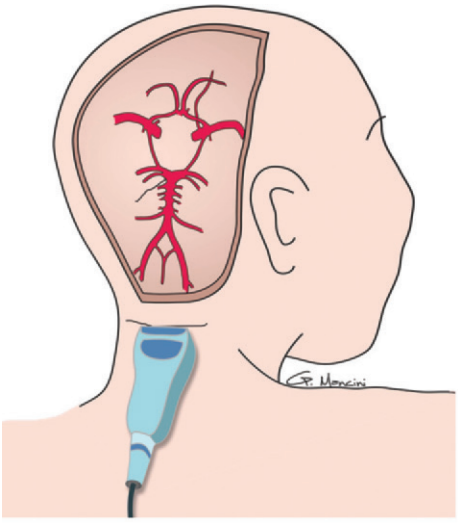
Figures 4, 5. **(4)** Drawing shows the transtemporal approach for Doppler US of intracranial vessels. The transducer is placed on the temporal bone either above the zygomatic arch and anterior to the external auditory canal or slightly more posterior, above the earlobe. **(5)** Color Doppler flow image obtained with a transtemporal window shows blood flow through the circle of Willis.

Imaging Technique

Transcranial Doppler imaging is performed at the authors' institution with a 5- to 1-MHz sector-array transducer and an Iu-22 ultrasound system (Philips Healthcare, Best, the Netherlands). Most of the imaging study is performed at the lower end of the frequency range (1–2 MHz) to allow the sound waves to penetrate the bone. Three acoustic windows are available for evaluating the intracranial vasculature: transtemporal, transforaminal or suboccipital, and transorbital. Depending on the acoustic window, different intracranial vessels are optimally visualized. Vessels are identified with gray-scale and color Doppler flow imaging. Spectral waveforms are obtained bilaterally from the proximal, mid, and distal MCA, and single measurements are obtained bilaterally in the visualized ACA, PCA, and terminal portion of the internal carotid artery (ICA). Flow velocity in the extracranial, distal ICA is also measured.

With the transtemporal approach, the transducer is placed on the temporal bone either above the zygomatic arch and anterior to the external auditory canal or slightly more posterior, above the earlobe. The transtemporal window allows optimal visualization of the circle of Willis; the MCA, ACA, and PCA; and the terminal ICA (Figs 4, 5).

With the transforaminal or suboccipital approach, the transducer is placed in the midline below the occiput and angled cephalad to allow visualization of the VA and BA in the posterior circulation (Figs 6, 7). The transorbital window allows visualization of the OA and the cavernous portion (siphon) of the ICA. With this approach, the transducer is placed so that it rests lightly on the closed eyelid (Figs 8, 9).



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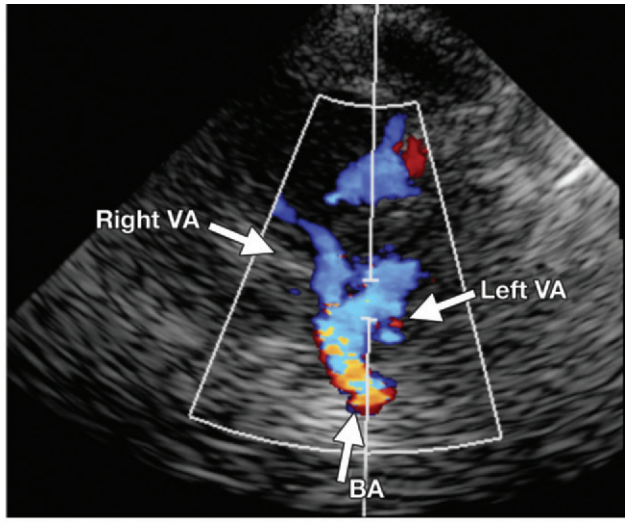
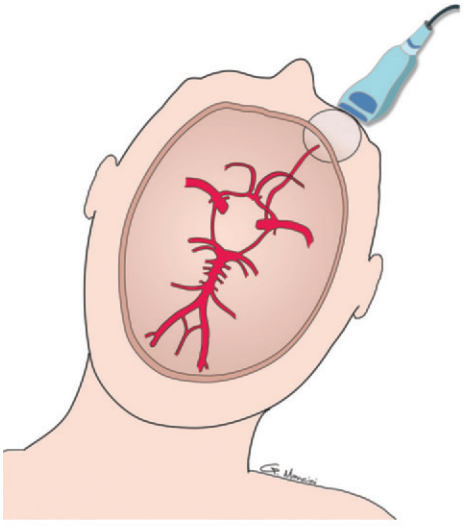


7.

Figures 6, 7. **(6)** Drawing shows the transforaminal or suboccipital approach for Doppler US of intracranial vessels. The transducer is placed in the midline below the occiput and angled cephalad. **(7)** Color Doppler flow image obtained with a transforaminal or suboccipital window shows the vessels of the posterior cerebral circulation.



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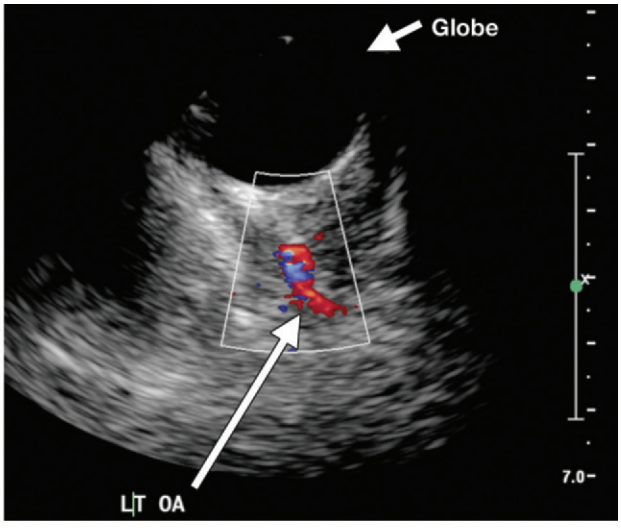
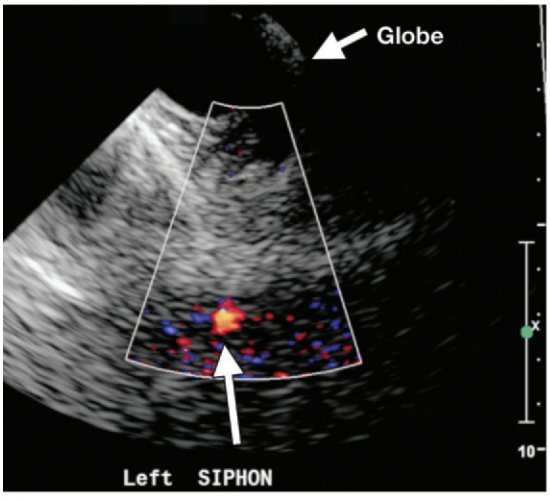


9a.



9b.

Figures 8, 9. **(8)** Diagram shows the transorbital approach for Doppler US of intracranial vessels. The transducer is placed lightly over the closed eyelid. **(9)** Color Doppler flow images obtained with a transorbital window demonstrate flow within the patent left OA **(a)** and left carotid siphon **(b)**.

Biosafety concerns related to the diagnostic use of ultrasound should be taken into account when performing transcranial Doppler imaging. Energy deposition from the ultrasound beam can result in mechanical injury or increased temperature of the tissues insonated. A guiding principle for the safe use of ultrasound is to use the lowest output power and shortest scanning time that is reasonably achievable to obtain the necessary diagnostic information (the "as low as reasonably achievable," or ALARA, principle). As a result of direct absorption of the ultrasound beam energy in bone, temperature increase is likely, with subsequent conduction of heat to the adjacent soft tissue. The eye is also susceptible to thermal injury because of its reduced ability for heat dissipation, given decreased perfusion (13).

The thermal index is an on-screen indicator of the potential increase in temperature that might occur in tissue after a long exposure time. A higher thermal index corresponds to a higher potential and higher risk for ultrasound-induced thermal damage to tissues. Specific thermal indexes can be displayed for soft tissue (ie, TIS), cranial bone (ie, TIC), and other bone (ie, TIB). Guidelines for maximum thermal indexes for specific exposure times are available in the literature (13,14).

Findings at Transcranial US

Vasospasm

As described by Spencer and Reid (15) and as indicated by the principles described earlier in this article, flow velocities are inversely proportional to the vessel diameter and directly proportional to the degree of vessel narrowing or stenosis until critical stenosis is reached, at which time flow velocities actually decrease. In general, the narrower the vessel diameter and the greater the severity of vasospasm, the higher is the flow velocity.

Teaching Point

Vasospasm following subarachnoid hemorrhage is generally absent in the first 72 hours. Vasospasm usually occurs by day 3, peaks between days 6 and 12, and resolves 15–20 days after the onset of hemorrhage (11).

The parameters used for the detection and measurement of vasospasm at transcranial Doppler imaging include the PSV, MFV, and various indexes (eg, the RI). Cutoff values that are used at our institution for specific cerebral vessels are shown in Tables 2 and 3 (1,9,16). Generally, for the MCA, an MFV of less than 120 cm/sec has proved to be a reliable predictor of the absence of vasospasm, whereas an MFV of 120–200 cm/sec is indicative of mild to moderate vasospasm, and an MFV greater than 200 cm/sec is indicative of severe vasospasm (1,17).

Table 2
Quantitative Parameters for Assessing Vasospasm of the MCA

Severity of Vasospasm	PSV (cm/sec)	MFV (cm/sec)	Lindegaard Ratio
Mild	200–250	120–150	3–4.5
Moderate	250–300	150–200	4.5–6.0
Severe	>300	>200	>6

Table 3
Quantitative Parameters for Assessing Vasospasm of the PCA and ACA

Vessel	PSV (cm/sec)	MFV (cm/sec)	Sloan Ratio*
PCA	>120	>85	NA
ACA	>120	>80	>4.0

*The Sloan ratio is calculated by dividing the MFV of the ACA by the MFV of the distal extracranial ICA to allow assessment of ACA vasospasm. NA = not applicable.

An increase in flow velocity alone is not a sufficient basis for confidently diagnosing vasospasm; physiologic conditions such as hyperemia and autoregulation, and induced conditions such as hypertension and hypervolemia, also may lead to an increase or decrease in flow velocity. To correct for and distinguish between these dynamic states, Lindegaard et al (6) proposed the use of a ratio derived from concurrent measurements of MFV in the MCA and the distal ipsilateral extracranial ICA (MFV_{MCA}/MFV_{EICA}). A Lindegaard ratio of 3–6 is indicative of mild to moderate vasospasm, and a ratio greater than 6.0 is indicative of severe vasospasm (Fig 10). Elevated flow velocities with a Lindegaard ratio of less than 3.0 are suggestive of hyperemia or another physiologic or induced state (1,6,12).

Teaching Point

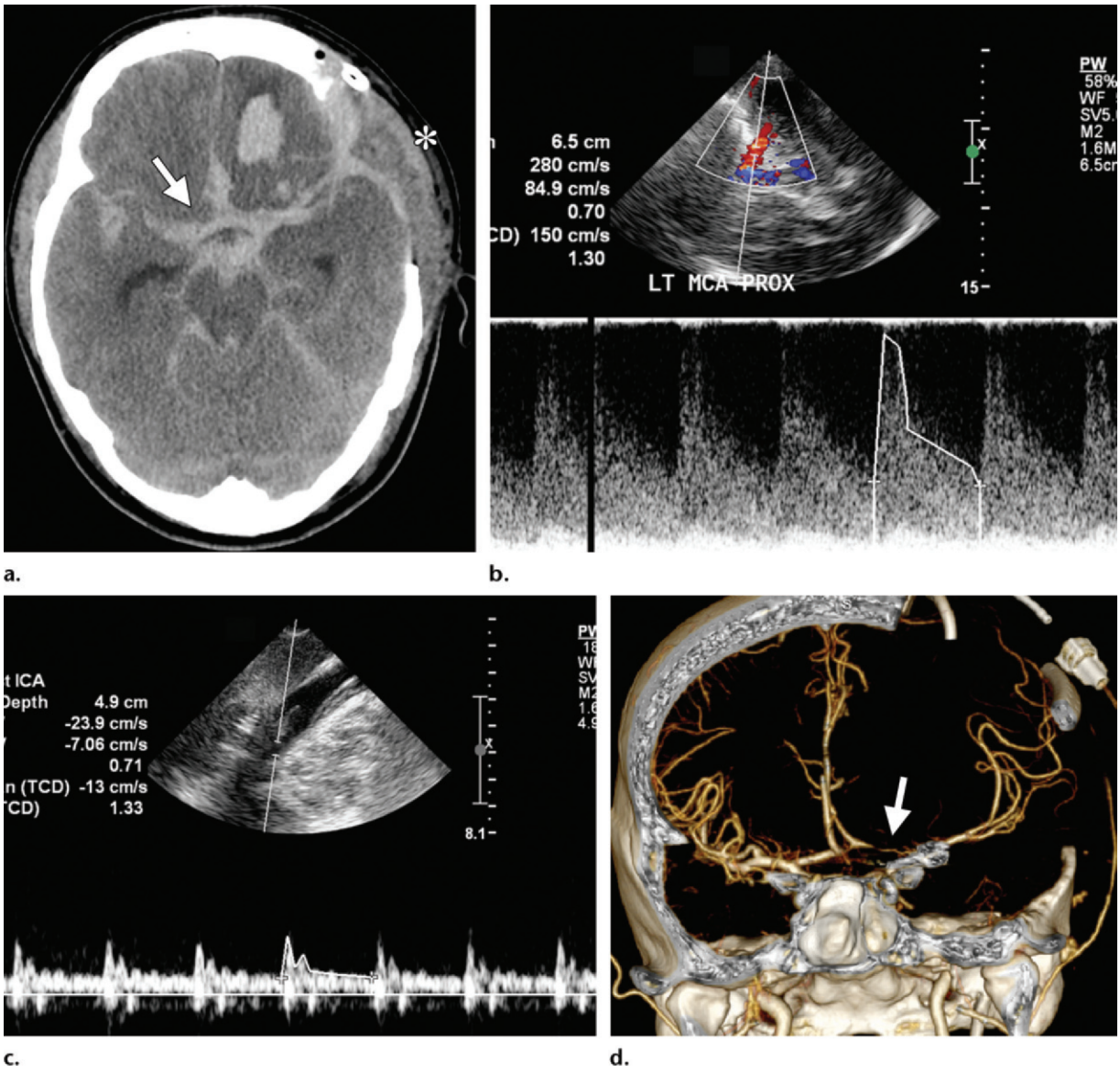


Figure 10. **(a)** Unenhanced axial CT scan of the brain depicts a subarachnoid hemorrhage (arrow) secondary to a ruptured aneurysm. Note the left frontal craniectomy (∗). **(b, c)** Doppler US images and spectral waveforms obtained in the left MCA **(b)** and ipsilateral distal ICA **(c)** show only mildly increased PSV and MFV in the left MCA (280 cm/sec and 150 cm/sec, respectively) but a markedly elevated Lindegaard ratio of 11.5, a finding indicative of severe vasospasm. **(d)** Three-dimensional reconstruction from CT angiography depicts a severe stenosis of the left MCA (arrow).

Serial Doppler US studies are often necessary, as vasospasm is not usually present immediately after the onset of subarachnoid bleeding but instead manifests later in the clinical course, usually within the first 2 weeks. A baseline Doppler US examination after radiographic detection of a subarachnoid hemorrhage is important and aids in the detection of vasospasm, since substantial elevation in flow velocities relative to those measured at baseline may be indicative of vasospasm (Figs 11, 12).

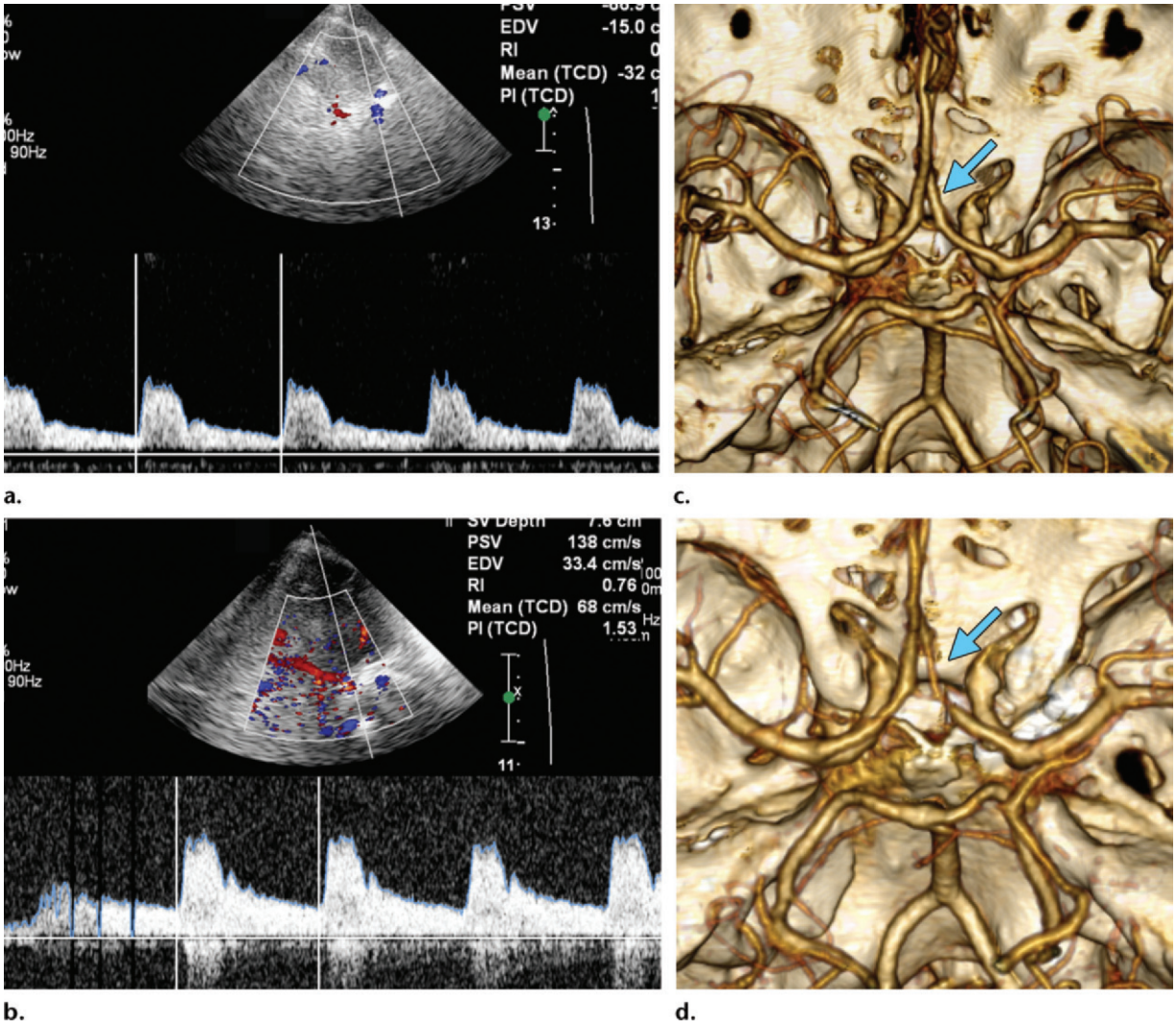


Figure 11. **(a)** Transcranial Doppler US image obtained immediately after aneurysm rupture shows normal PSV of 67 cm/sec in the right ACA. **(b)** Follow-up transcranial duplex Doppler US image obtained approximately 10 days later depicts increased PSV of 138 cm/sec, a finding suggestive of mild vasospasm, in the right ACA. The MFV is normal (68 cm/sec) but also has increased since baseline (32 cm/sec). **(c, d)** Three-dimensional images from baseline **(c)** and follow-up **(d)** CT angiography demonstrate interval development of right ACA vasospasm (arrow).

Multiple intracranial vessels may be affected by vasospasm (Fig 13). An elevated Lindegaard ratio helps distinguish between mild increases in MCA flow velocity that are due to vasospasm and increases that are due to a physiologic process such as hyperemia or autoregulation.

Although not as well studied, values and ratios have been proposed for the evaluation of vasospasm involving the posterior circulation. Analogous to the Lindegaard ratio, the ratio of the BA flow velocity to the extracranial VA flow velocity has been analyzed. A BA-to-VA ratio of 2.0–3.0, when combined with a BA flow velocity greater than 85 cm/sec, was reported to have good sensitivity and specificity for the diagnosis of mild to moderate vasospasm. A BA-to-VA ratio greater than 3.0 was found to allow accurate diagnosis of severe vasospasm (18).

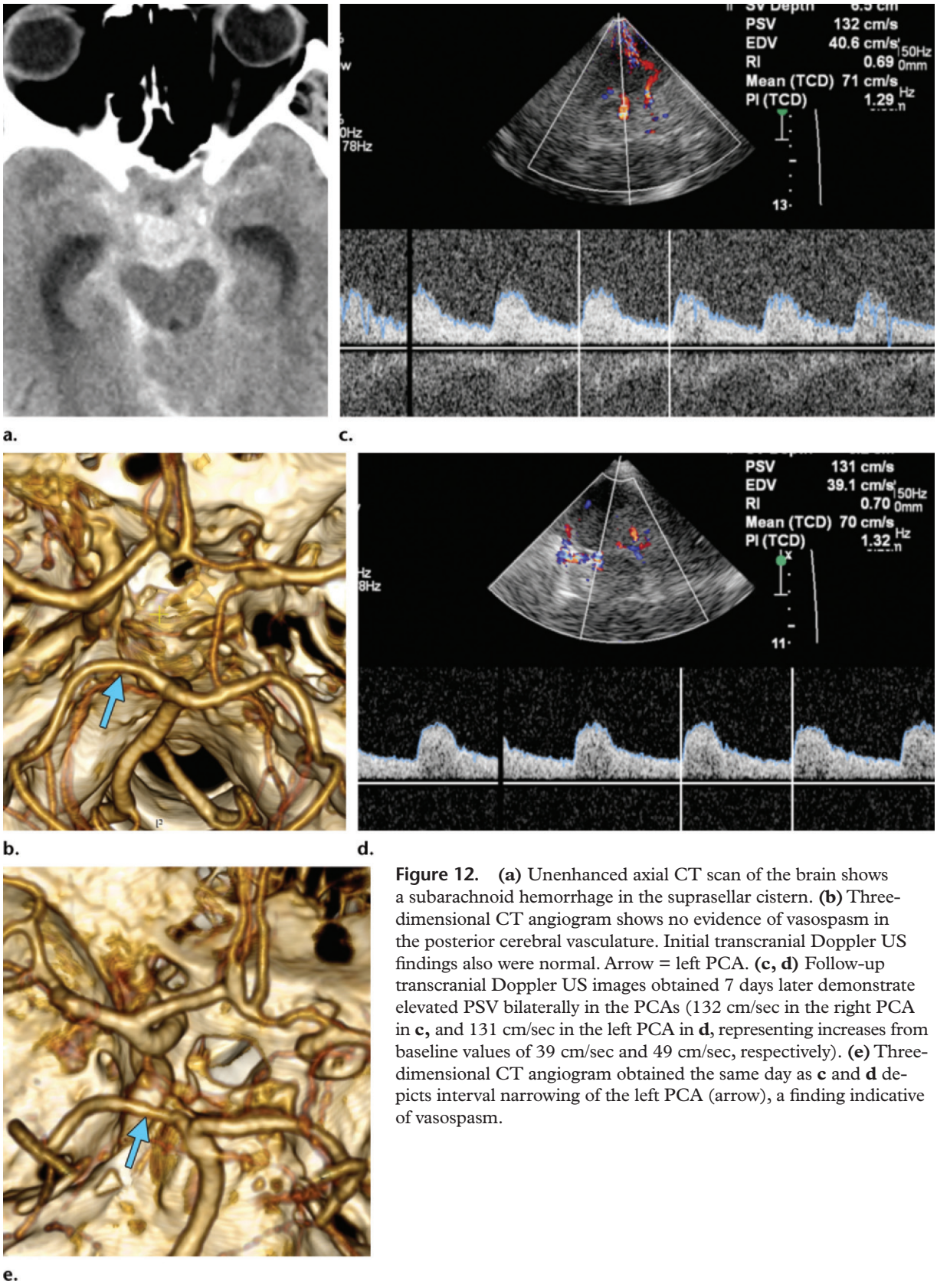
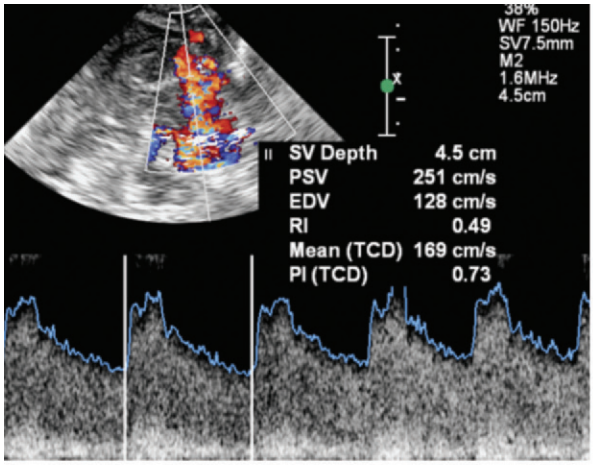


Figure 12. **(a)** Unenhanced axial CT scan of the brain shows a subarachnoid hemorrhage in the suprasellar cistern. **(b)** Three-dimensional CT angiogram shows no evidence of vasospasm in the posterior cerebral vasculature. Initial transcranial Doppler US findings also were normal. Arrow = left PCA. **(c, d)** Follow-up transcranial Doppler US images obtained 7 days later demonstrate elevated PSV bilaterally in the PCAs (132 cm/sec in the right PCA in **c,** and 131 cm/sec in the left PCA in **d,** representing increases from baseline values of 39 cm/sec and 49 cm/sec, respectively). **(e)** Three-dimensional CT angiogram obtained the same day as **c** and **d** depicts interval narrowing of the left PCA (arrow), a finding indicative of vasospasm.

Figure 13. **(a–c)** Transcranial Doppler US images and waveforms show increased MFVs of 169 cm/sec in the right MCA **(a)** and 110 cm/sec in the right ACA **(b)**, with an elevated Lindegaard ratio of 3.19. These findings are suggestive of mild narrowing, most likely secondary to vasospasm in the setting of an intracranial hemorrhage. In **c**, elevated MFV in the right terminal ICA is suggestive of vessel narrowing. **(d, e)** Cerebral angiograms in lateral **(d)** and anteroposterior oblique **(e)** projections demonstrate narrowing of the supraclinoid ICA (arrow in **d**) and the proximal right MCA (arrow in **e**) and right ACA (arrowhead in **e**).



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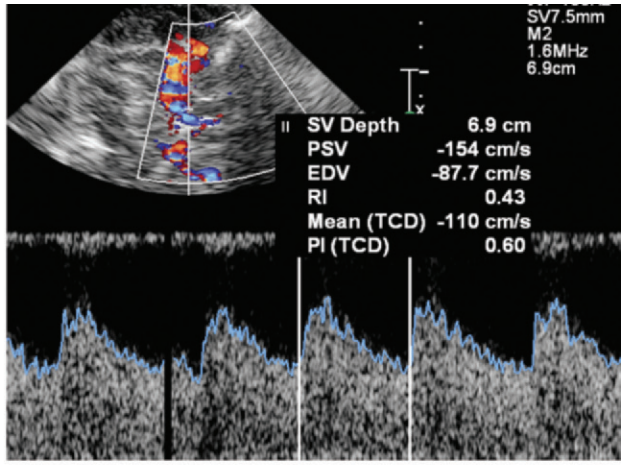

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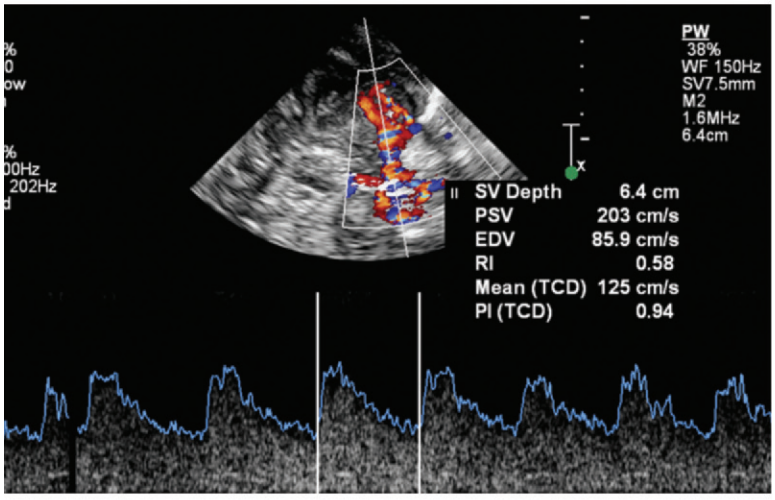

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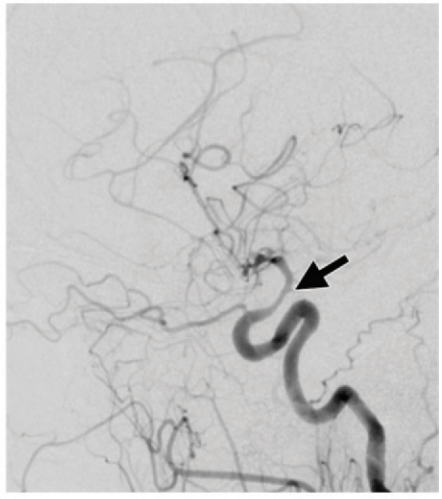

d.

Stenosis

Transcranial Doppler US has been shown to allow reliable exclusion of the presence of intracranial vascular stenosis. However, although findings at transcranial Doppler US (eg, focal elevation of flow velocities, turbulence) may be suggestive of the presence of stenosis, those findings must be confirmed with conventional angiography or CT angiography before the diagnosis can be made (19) (Fig 14).

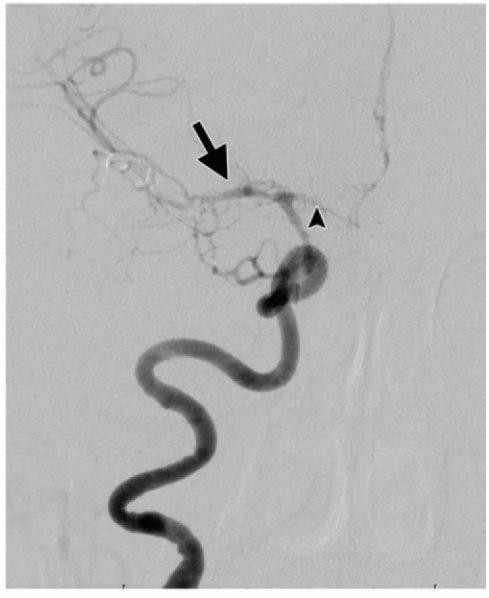

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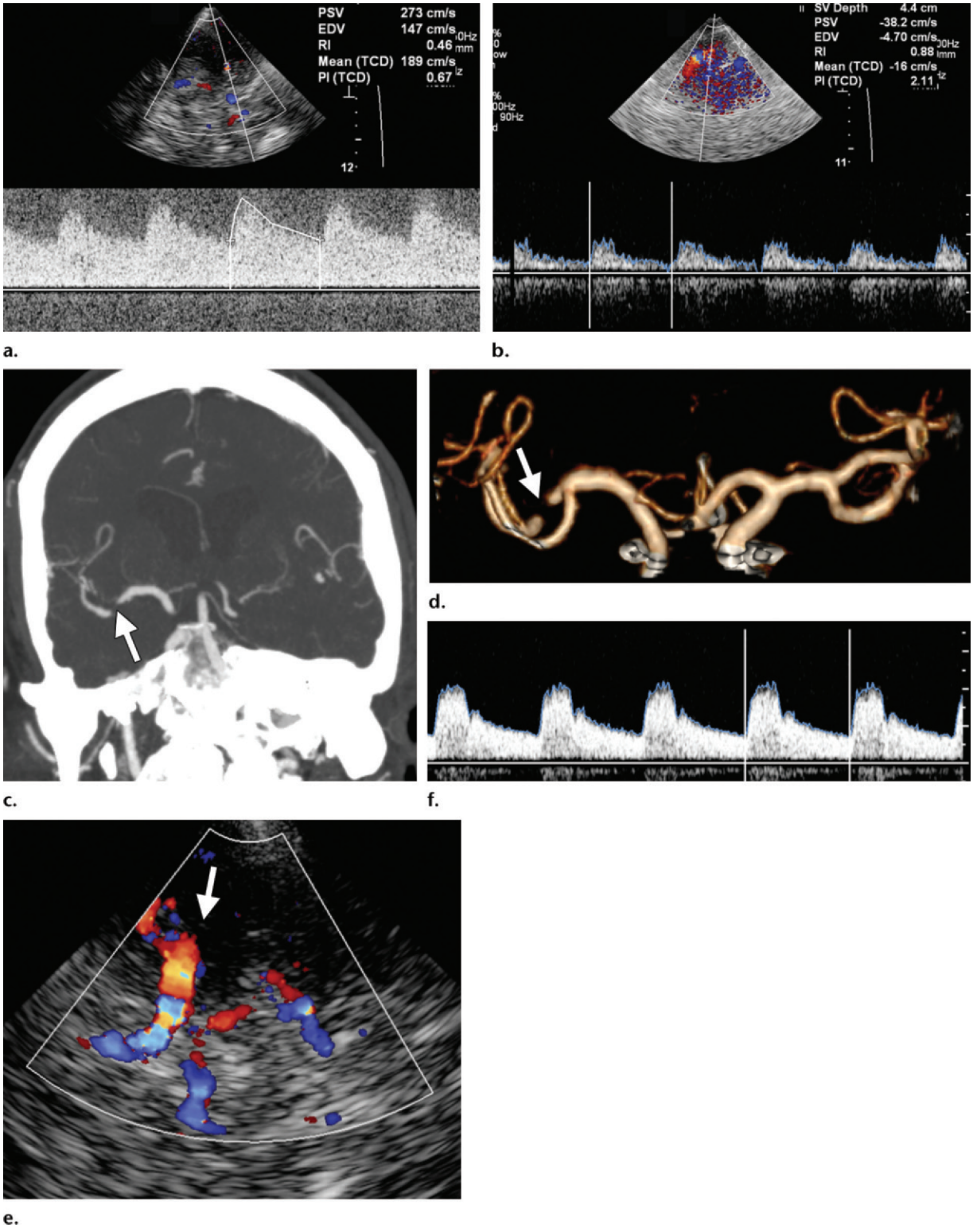
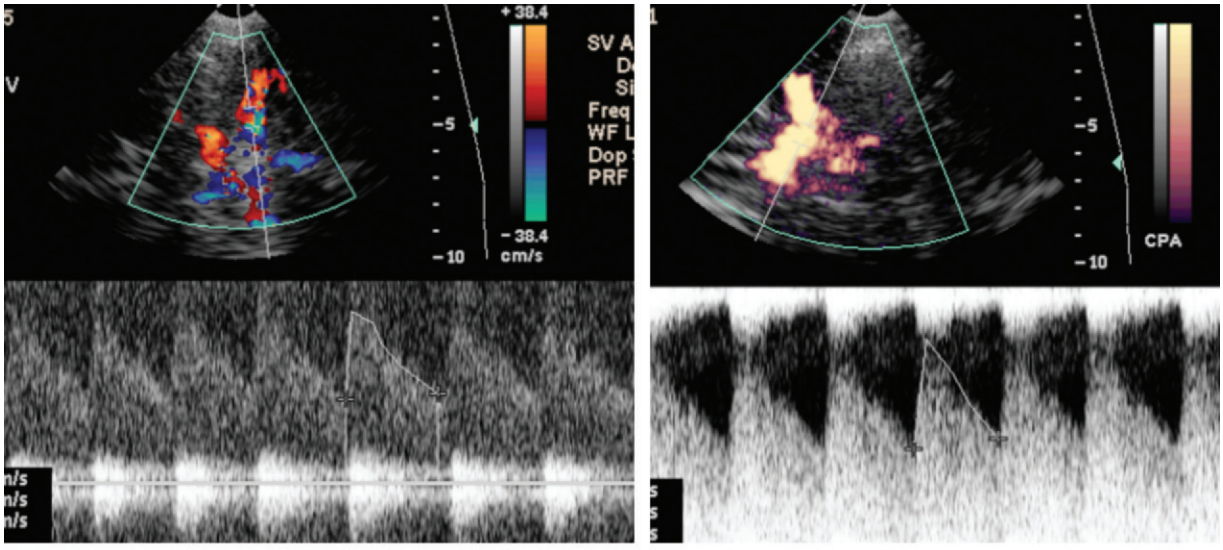
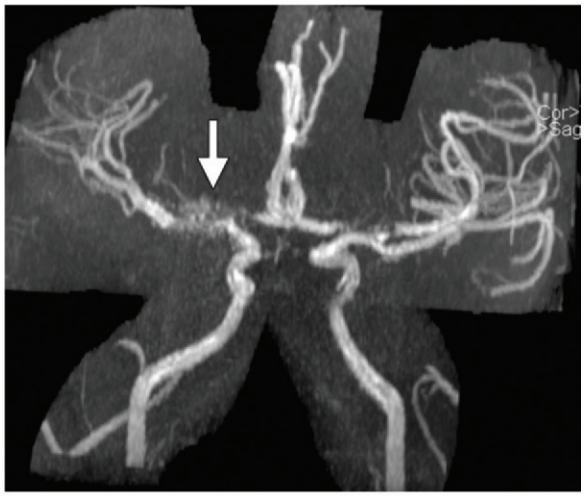
Figure 14. **(a, b)** Transcranial Doppler US images depict an increased MFV of the right MCA (**a**, 189 cm/sec) and a low to normal MFV of 16 cm/sec in the distal right ICA **(b)**, with a markedly elevated Lindegaard ratio of 11.8. **(c, d)** Coronal maximum intensity projection image **(c)** and three-dimensional volume-rendered image **(d)** from CT angiography of the circle of Willis show a short-segment severe stenosis of the M1 segment of the right MCA (arrow). Although severe vasospasm may have the same appearance, atherosclerosis was believed to be the cause of stenosis because vasospasm usually affects a longer vessel segment and because, despite clinical signs and symptoms of stroke, no subarachnoid hemorrhage was found at unenhanced CT. **(e, f)** Color Doppler US image **(e)** and waveform **(f)** demonstrate a patent left MCA (arrow in **e**) with a normal MFV (50 cm/sec).





a. b.



c.

Figure 15. **(a, b)** Duplex Doppler US of the bilateral MCAs demonstrates increased flow velocities (181 cm/sec in the right MCA in **a**, 196 cm/sec in the left MCA in **b**), raising concern about underlying vessel narrowing. **(c)** Coronal maximum intensity projection image from subsequent time-of-flight magnetic resonance (MR) angiography of cranial vessels depicts severe bilateral MCA stenosis or occlusion, more severe on the right than on the left, with the formation of collateral vessels (arrow). These findings are typical of a moyamoya circulation pattern.

Sickle Cell Disease

Children with sickle cell disease have an increased risk for stroke. The prevalence of stroke among patients with sickle cell anemia has been reported to be 11% by the age of 20 years. Stroke and infarction are usually due to occlusion of the intracranial ICA, proximal MCA, and ACA as a consequence of fibrous proliferation of the intima with resultant stenosis. Transcranial Doppler US allows the detection of intracranial lesions by depicting areas of increased flow velocity (Fig 15). Patients with an MFV of more than 200 cm/sec have a statistically significantly increased risk for stroke and have been shown to benefit greatly from transfusion therapy (20,21).

ICA Occlusion

US findings of occlusion of the extracranial ICA include echogenic material filling the vessel at gray-scale imaging and absence of flow at color Doppler flow imaging and in Doppler spectra. In addition, the ipsilateral common carotid artery waveform changes from a low-resistance to a high-resistance flow pattern. In the presence of occlusion, a collateral pathway may form in which the ipsilateral external carotid artery supplies the OA, which in turn may supply the brain via the MCA. When this pathway is present, the waveform in the external carotid artery becomes "internalized" (converts from a high-resistance to a low-resistance flow pattern) and flow within the OA reverses. Flow reversal in the OA can be demonstrated at transcranial Doppler US by using a transorbital approach (Fig 16).

Brain Death

The definition of brain death and the corresponding diagnostic criteria have evolved over time. Clinical criteria for a diagnosis of brain death include coma, absence of brainstem re-

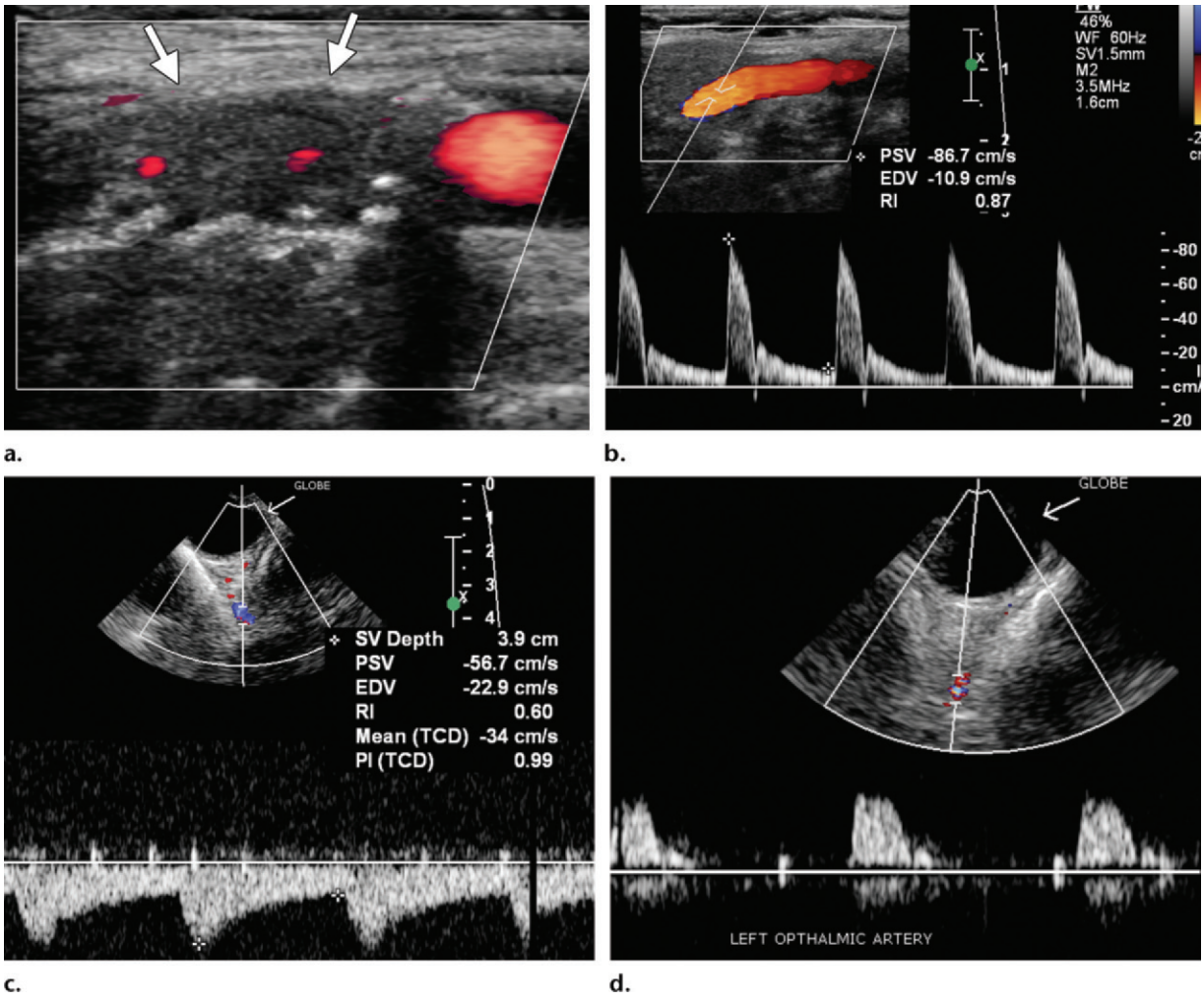


Figure 16. ICA stenosis with a collateral circulatory pathway. **(a)** Power Doppler flow US image shows echogenic material filling the right ICA (arrows). No flow was identified either with power Doppler or with spectral Doppler techniques. These findings are indicative of ICA occlusion. **(b)** Color Doppler US image and spectral Doppler waveform show low-resistance, internalized flow in the right external carotid artery. **(c)** Transcranial Doppler US image obtained with a transorbital approach shows reversal of flow in the right OA, a finding indicative of collateral flow from the external carotid artery to the MCA via the right OA. **(d)** Transcranial Doppler US image obtained in the left OA for comparison shows normal flow.

flexes, and apnea (22). Tests that may be performed to verify the diagnosis include electroencephalography, cerebral angiography, cerebral scintigraphy, and transcranial Doppler US, which demonstrates typical spectral waveform patterns. In a patient with brain death, increased intracranial pressure results in an initial loss of diastolic flow. With continuing increases in intracranial pressure, diastolic flow may reverse; this is referred to as reverberating or oscillating flow. Small, low-velocity systolic peaks or spikes are also observed in spectral waveforms. Findings suggestive of cessation of flow should be interpreted cautiously because they could result from technical factors, such as poor penetration of the skull by sound waves (23).

Conclusion

Transcranial Doppler US has become a mainstay in the intensive care unit for the evaluation and management of vasospasm secondary to subarachnoid hemorrhage. This imaging study is also useful for evaluating the intracranial vasculature in the settings of stroke, sickle cell disease, and

carotid artery occlusion. Originally, the study was based on the use of a pulsed Doppler technique that did not allow direct visualization of the vessels. Newer techniques that are based on gray-scale and color Doppler flow imaging enhance the ease and accuracy of the examination.

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Advances in Transcranial Doppler US: Imaging Ahead

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Page E2
Transcranial Doppler ultrasonography (US) is a noninvasive technique that allows accurate and sensitive evaluation of the cerebral vessels for vasospasm without requiring the use of a contrast medium, exposure of the patient to ionizing radiation, or transport of the patient out of the intensive care unit.

Page E2–E3
Transcranial gray-scale and color Doppler flow imaging not only have utility for detecting vasospasm but also show promise for evaluating the intracranial vasculature in patients with stroke, sickle cell disease, carotid artery occlusion, stenosis of intracranial vessels, and brain death.

Page E3
Vessel segments affected by vasospasm are narrower; therefore, the velocities of blood flowing through these vessel segments must increase. The Doppler effect and Doppler equation allow measurement of this increase in velocity and, thereby, detection of vasospasm.

Page E6
In general, the narrower the vessel diameter and the greater the severity of vasospasm, the higher is the flow velocity.

Page E6
An increase in flow velocity alone is not a sufficient basis for confidently diagnosing vasospasm; physiologic conditions such as hyperemia and autoregulation, and induced conditions such as hypertension and hypervolemia, also may lead to an increase or decrease in flow velocity.