# Neurosurgery

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# SECTION C

# Other Neurodiagnostic Tests

# 21 Transcranial Doppler Ultrasonography

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## History

In 1959, Satomura first reported the use of Doppler ultrasound to measure flowing blood, initially investigating the peripheral vessels.[62] Since that time, the technology has undergone significant development and refinement. Presently Doppler ultrasonography is used extensively in the evaluation of extracranial vascular disease in combination with echo imaging (duplex scanning).

In 1982, Aaslid et al. reported the ability to record flow velocities in the basal cerebral arteries using Doppler ultrasound and introduced transcranial Doppler (TCD) ultrasonography.[5] This was made possible by utilizing an optimized 2-MHz pulsed range-gated system. With the ability to record flow velocities directly from the intracranial arteries, a new dimension was added to the abilities of Doppler ultrasonography. These developments have made possible the noninvasive evaluation of intracranial stenosis due to atherosclerosis and vasospasm and have also allowed the detection of hemodynamic changes due to a variety of disorders such as extracranial occlusive disease, head injury, intracranial hemorrhage, and conditions causing increased intracranial pressure (ICP).

## Principles and Equipment

Christian Doppler, an Austrian physicist, described the Doppler effect in 1843 to explain certain astronomical observations. Briefly stated, the Doppler effect describes a shift in the frequency of a wave when either the transmitter of the wave or the receiver of the wave is moving with respect to the wave-propagating medium. Therefore, sound emanating from or reflected by an object moving toward the observer will have a higher frequency in proportion to the speed of the moving object. Conversely, sound emanating from an object moving away from an observer will have a lower frequency in proportion to the speed of the moving object. When using ultrasound to measure the velocity of flowing blood, the ultrasound is emitted by a probe and reflected off the moving blood cells, and the signal is received by the same probe. The shift in the frequency of the reflected ultrasound will be proportional to the velocity of the flowing blood, thus, blood flowing toward or away from the probe will reflect the ultrasound at a higher or lower frequency, respectively.

Doppler ultrasound is well established as a clinical tool to examine the extracranial arteries. Methods have been established using both continuous-wave Doppler and pulsed Doppler employing ultrasonic frequencies between 3 and 10 MHz. Continuous-wave Doppler constantly transmits an ultrasonic beam from a crystal source and simultaneously receives the reflected ultrasound. The receiver records the changes in frequency of the reflected ultrasound produced by moving blood throughout the path of the ultrasonic beam. Pulsed Doppler sends bursts of ultrasound at a regular interval, which is called a pulse repetition frequency. The receiver employs an electronic gate to sample the reflected pulses at certain intervals. Specifically, the gate opens at the time interval required for the ultrasound to be transmitted to and reflected back from a preselected depth. In this way pulsed Doppler is able to record from a specific sample volume at preselected targets.

Transcranial Doppler employs this pulsed range-gated design, which enables sampling of flow velocities at specific sites in and around the circle of Willis, where there is a high concentration of vessels. A 2-MHz ultrasonic frequency is used because this allows penetration through the thin portions of the temporal bone (Fig. 21-1). Studies on ultrasound transmission through the human skull have shown that transmission of up to 35 percent of the power can be achieved through the temporal bone. The diploe has a profound effect in scattering the ultrasound due to the bone spicules present. In the thin areas of the temporal bone, the inner and outer layers fuse with no diploe present, thus minimizing the absorption of ultrasound energy.

Three examination routes are available for obtaining signals from the intracranial vessels using TCD ultrasonography: the transtemporal, transorbital, and transoccipital. Through the transtemporal route, signals are obtainable from the middle cerebral artery (MCA), anterior cerebral artery (ACA), intracranial internal carotid artery (ICA), and proximal posterior cerebral artery (PCA) (Fig. 21-2). The transorbital route can be used to examine the ophthalmic artery and the ICA. Using the transoccipital approach, signals can be obtained from the vertebral arteries and basilar artery.

**Figure 21-1** Transillumination of the skull illustrates the thin portions of the temporal bone where ultrasound can penetrate.

Recording of flow velocities from the intracranial arteries has many important implications in the study of cerebral vascular disease. The recorded dimension, velocity, is not a direct measurement of flow, but proportionality does exist between velocity and flow when the arterial diameter remains constant. Lindegaard et al. have demonstrated this relationship by comparing MCA velocity to flow measurements obtained by an electromagnetic flow meter on the carotid artery during carotid surgery.[39] Bishop et al. have compared TCD velocities to cerebral blood flow (CBF) measurements obtained in human subjects using xenon 133 ($^{133}Xe$).[13] They demonstrated that although resting MCA velocity did not correlate very well with CBF, CBF changes induced by varying the $Pa_{CO_2}$ did correlate well with the change in MCA velocity. Thus when the diameter of the cerebral basal arteries remains constant, changes in the velocities can reflect accurately changes in flow.

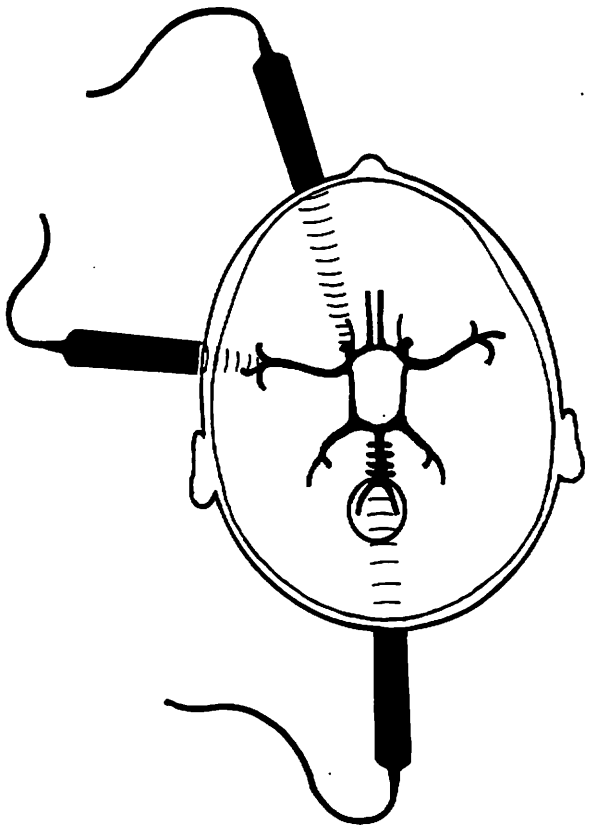


**Figure 21-2** The three windows, transtemporal, transorbital, and transoccipital (through the foramen magnum), for recording blood flow velocities from the basal cerebral arteries.

The angle of insonation (angle between the ultrasound beam and the vessel being recorded from) also needs to be considered when measuring true velocity. The true flow velocity and the observed velocity will be equal when this angle equals zero degrees. The observed velocity will decrease relative to the true velocity as the angle of insonation increases. The correction will be very small at small insonation angles and will be a product of the cosine of the angle and the true velocity.[1] Thus if the angle between the ultrasound beam and the flow vector is 15 degrees, 97 percent of the true velocity would be observed. If the insonation angle is 60 degrees, then 50 percent of the true velocity would be observed (Fig. 21-3). This angle becomes significant when examining the extracranial carotid arteries using ultrasound. The insonation angle used in transcranial applications is small for most of the arteries examined because of their anatomic positions. The change in angles between different observations is also small because of the restrictions of recording sites in the temporal region.

Stenosis produced by atherosclerosis, vasospasm, or other mechanisms will be reflected by an increase in velocity through the stenotic segment in proportion to the reduction of the cross-sectional area when the same flow is preserved. Thus, for example, if the diameter is reduced to half of the original value by vasospasm, velocity increases to 400 percent of normal.

The frequency changes that are produced by the flowing blood are converted to velocity in centimeters per second (cm/s). Increased frequency shifts indicating blood flowing toward the probe will register as velocity above the zero line on the display screen. Any decreased frequency indicating blood flowing away from the probe will register as a negative deflection on the screen. This directionality is useful in identifying specific arteries and branch points around the circle of Willis.

## Technique

As mentioned earlier, the three routes or "windows" for examination of the intracranial vessels are the transtemporal, transorbital, and transoccipital. To focus the probe on a particular artery and obtain a signal is referred to as *insonation.* In order for one to identify vessels accurately, knowledge of the anatomic position and direction of flow of the various intracranial arteries in normal and pathologic states is essential. The steps in identifying any particular artery are (1) determine the direction of flow, (2) follow the signal to various depths and determine the spatial relationship of the signal to other known arteries, and (3) determine the response to compression or vibration maneuvers.

Examination through the transtemporal route provides access to the MCA, ACA, ICA, and PCA. Ultrasound transmitting gel is applied to an area just above the zygoma and slightly anterior to the ear. The depth can initially be set at 45 to 50 mm by adjusting the range gate. Generally the strongest signal in this region is the middle cerebral artery. The depth is increased progressively, and one finds a bifurcation usually at 65 mm. A bidirectional signal

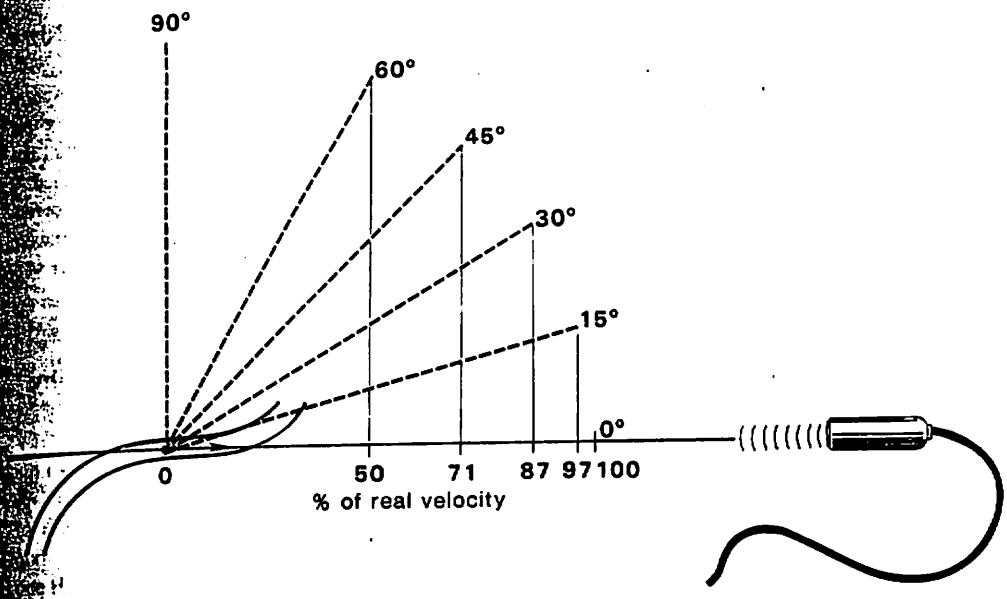



**Figure 21-3** The relationship between the angle of insonation and the percentage of the true velocity that will be observed by the recording device.

indicating simultaneous flow toward and away from the probe confirms the position on the ICA termination. The ACA signal can then be followed from this point to a depth of 70 to 75 mm. Aiming the probe inferiorly from the ICA termination will locate the MCA, and aiming it posteriorly will locate the PCA.

The access to the transoccipital "window" is obtained by flexing the patient's head forward and placing the probe just below the cervico-occipital junction. By setting the depth to 45 mm and directing the probe slightly laterally, one can usually find the vertebral artery, and under normal conditions, it will display a signal indicating blood flowing away from the probe. The depth is increased progressively to 80 to 85 mm while following the vertebral artery signal, and at this point the basilar artery is located in the midline. Signals directed toward the probe often are found at 50 to 65 mm, and represent the posterior inferior cerebellar artery.

Through the transorbital route, one can find the ophthalmic artery and also the carotid siphon. The patient's eyes are closed, and the probe is applied to the upper lid. The ophthalmic artery is located at a depth of approximately 40 to 45 mm, and it can be followed to its origin at the carotid artery.

## Subarachnoid Hemorrhage and Vasospasm

Perhaps the most promising clinical aspect of transcranial Doppler ultrasonography is its capacity to noninvasively determine the degree of vasospasm after subarachnoid hemorrhage (SAH). The most significant changes in vessel diameter induced by vasospasm usually occur in the basal arteries. As vasospasm progresses and the vessels narrow, blood flow velocities through the stenotic segments increase and velocities can exceed normal resting values by five to six times. Correlations between residual lumen diameters of the ACA and MCA on angiography and changes in velocity on TCD ultrasonography have been observed.[3] The resistance across a stenotic segment is related not only to the degree of stenosis but also to the length of the stenosis. Thus the effect of basal artery narrowing on CBF reduction after subarachnoid hemorrhage will depend on the degree and extent of vessel narrowing, the arterial blood pressure, and the ability of the cerebral circulation to autoregulate and compensate for the vasospasm. In addition, the adequacy of the collateral circulation plays a role. When vasospasm becomes severe enough to reduce CBF to critical levels, neurologic deficits will ensure, frequently in a precipitous manner.

The usefulness of TCD ultrasonography is that it can alert the clinician to the degree and extent of vasospasm and can allow institution of proper therapy before neurological deficit or cerebral infarction takes place. Another potential use of TCD ultrasonography is to aid in the decision about the timing of aneurysm surgery; it may help to avoid untimely operations in asymptomatic patients who are undergoing a rapid but silent progression of vasospasm.

The middle cerebral arteries are the most ideally suited for TCD recordings in vasospasm. They are end arteries with limited leptomeningeal collaterals under normal conditions, so there is a close correlation between the amount of spasm and the increase in velocity seen with TCD ultrasonography. The other intracranial arteries from which recordings are usually made—proximal ACA, PCA, ICA, and the vertebrobasilar system—generally have collateral branches, and depending on the degree of collateralization, the relationship between spasm and increased velocity may not be as close. If an artery with extensive distal collateral vessels narrows in vasospasm, velocity may only increase moderately because collateral channels can provide some of the blood flow demands.

The trunk of the MCA is the vessel that is most easily evaluated for vasospasm because of its location, orientation, and size. The distal ICA proximal to the carotid termination is also usually easily evaluated and is sometimes difficult to distinguish from the MCA. The region of the carotid termination and proximal MCA is the vascular location most commonly affected by vasospasm. The sensitivity and specificity of TCD for detecting vasospasm are greatest at the MCA. Lindegaard et al.[40] found an 85 percent sensitivity and a 98 percent specificity, and Sloan et al.[68] reported an 84 percent sensitivity and an 89 percent specificity for detecting angiographic vasospasm of the MCA.

Spasm of the distal cerebral vessels can, on occasion, lead to neurological deficit when angiography and TCD indicate no spasm in the proximal cerebral vessels. The frequency of this complica-

tion is unknown but it is estimated that isolated distal vasospasm occurs less than 10 percent of the time with anterior circulation aneurysms.[51]

The hemodynamic effect of vasospasm has also been examined. Hassler and Chioffi[27] found that reduced $CO_2$ reactivity in patients with significant vasospasm indicted impaired hemodynamic reserve caused by reduced distal perfusion pressure. It has also been observed that high blood flow velocity caused by vasospasm can be decreased by increased ICP.[33]

Under normal conditions, blood flow velocities in the MCA range from 33 to 90 cm/s, with an average value of 62 cm/s.[5] Velocities in excess of 120 cm/s correlate with vasospasm seen by angiography.[3] The divisions are arbitrary after this point, but mean velocities greater than 200 cm/s appear to correlate with severe spasm seen on angiography and are frequently associated with clinical episodes of ischemia and infarction.[65] Seiler et al. found in a group of 39 patients with SAH followed with TCD ultrasonography that if the blood flow velocities did not exceed 140 cm/s, no patient developed a cerebral infarct.[65] Blood flow velocities greater than 200 cm/s were associated with ischemia and infarction, but some patients remained asymptomatic.

As a secondary effect of vasospasm, between the 4th and 20th day after SAH, musical tones can sometimes be heard from the loudspeaker of the TCD recording device.[7] The maximum amplitude of these sounds is heard near the carotid termination. The cause of these murmurs is most likely the creation of pure-tone frequencies by the vibrations of the arterial walls caused by the periodic shedding of vortices in the transition between laminar flow and turbulent flow. The frequency of the tones appears to correlate with the velocity and therefore the degree of vasospasm.

## Time Course of Vasospasm

Since the original description of arterial spasm, several studies have documented the time course of vasospasm using angiography. Allcock and Drake reported that spasm was present in 45 percent of patients less than 3 days after SAH, 41 percent at 3 to 10 days, and 25 percent at more than 10 days.[9] A very detailed study was performed by Weir et al., who took careful measurements from 627 sets of angiograms from 293 patients with aneurysms.[74] These investigators found that vasospasm appeared initially on day 3 after subarachnoid hemorrhage, was maximal at days 6 to 8, and was gone by day 12. These studies, however, provide noncontinuous information in contrast to TCD ultrasonography, which has the advantage of being a noninvasive test that can be performed daily to follow the changes that occur.

Several studies using TCD ultrasonography have looked at the time course of vasospasm. In a group of 39 patients with SAH, most of whom were operated on late, vasospasm indicated by an increase in flow velocities on TCD ultrasonography was maximal between days 7 and 12.[65] If the group was broken into subgroups based on the severity of vasospasm, the patients with less severe vasospasm tended to have their maximal increase in velocities later than 7 days. In contrast, a subgroup of patients who died from cerebral infarction had large increases in velocity on days 2 and 3 and high velocities, indicating severe vasospasm, by day 5 after SAH. Harders and Gilsbach reported a series of 50 patients operated on within 72 h after SAH and treated with nimodipine.[26] They found that maximum velocities were reached between the 11th and 20th days after SAH. Figure 21-4 illustrates the time course of the TCD ultrasound velocity changes in a patient who had surgery on day 1 after SAH, received no calcium channel blocker, and did not develop any delayed neurological deficits.

Romner et al.[59] used TCD to examine patients for vasospasm within 12 h after SAH. They were unable to find evidence for significant early vasospasm during this interval. The effect of aneurysm clipping surgery on vasospasm has also been examined. It has been observed that elevations of velocities indicating progression of vasospasm were more likely to occur in patients who were operated on between 49 and 96 h following aneurysm rupture versus those operated on within 48 h of rupture.[60]

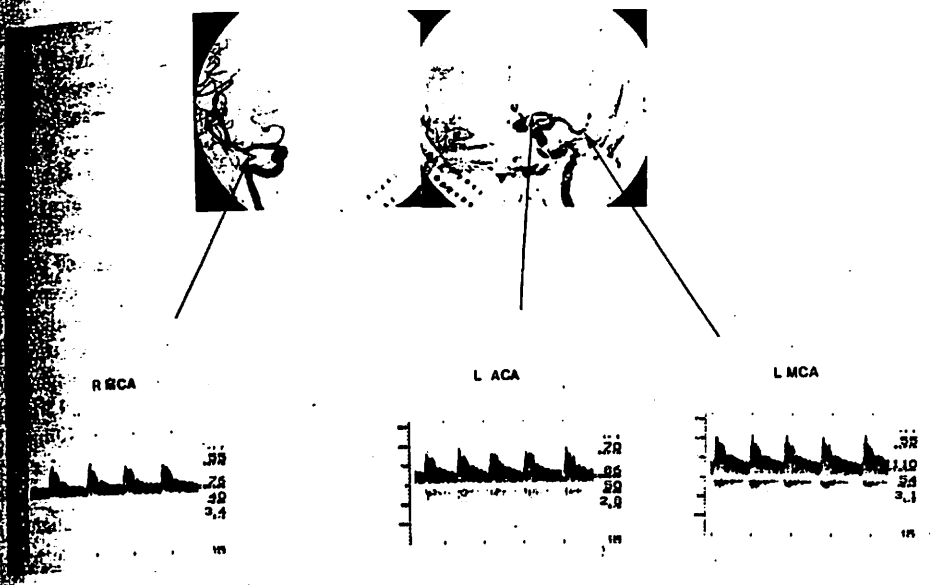
## Correlation of SAH on Computed Tomography with Velocity

Several reports have pointed out a strong correlation between the amount of blood detected in the basal cisterns on computed tomography (CT) scan after SAH and the subsequent development of vasospasm detected by angiography. Fisher et al.[19] studied a group of patients who had CT scans done within 5 days of their SAH and defined four groups based on the amount of blood seen: group 1, no blood detected; group 2, diffuse deposition or thin layers with all vertical layers of blood less than 1 mm thick; group 3, localized clots and/or vertical layers of blood 1 mm or greater in thickness; group 4, diffuse or no subarachnoid blood but intracerebral or intraventricular clots. Angiograms then done between 7 and 17 days after the SAH showed severe vasospasm in 2 of 11 cases in group 1, 0 of 14 cases in groups 2 and 4, and 23 of 24 cases in group 3. The results indicated that thick clots in the basal cisterns predispose patients to a very high incidence of severe vasospasm. Subsequently, other investigators have confirmed these findings.

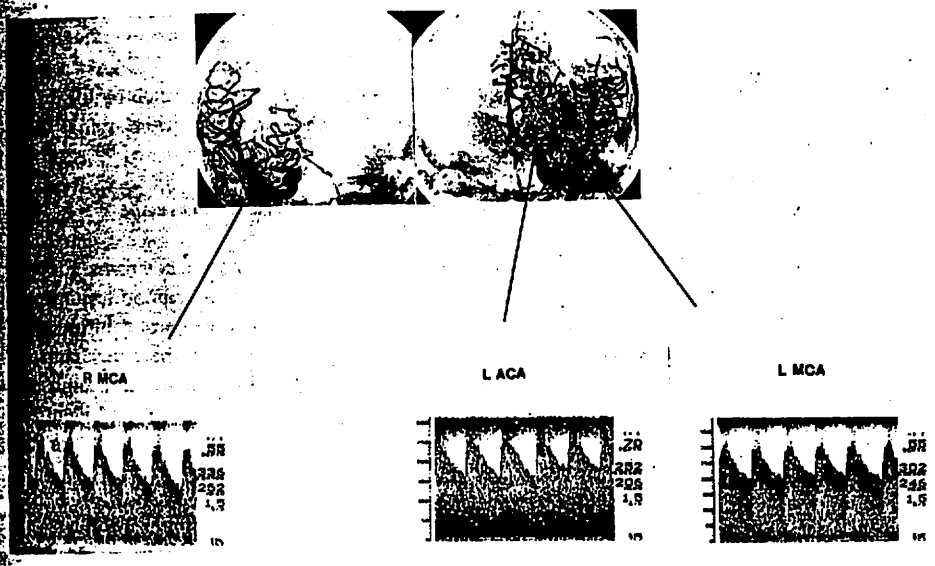
Seiler et al. performed a similar study using TCD ultrasonography instead of angiography to assess vasospasm in patients with SAH.[65] In 39 patients, CT scans were done within 5 days after SAH and assessed for the amount of blood in the basal cisterns. The patients were broken into three groups according to the criteria of Fisher et al.[19] The velocities in both MCAs were recorded daily, and the maximum velocities reached were noted. There was a strong correlation between the amount of blood in the basal cisterns and the maximum velocity reached. Eight of nine patients with no blood on the CT scan had maximum velocities below 140 cm/s. In CT group 3, with thick cisternal clots, 13 of 15 patients had maximum velocities during their hospital course of 140 cm/s or greater, and 9 of 15 had maximum velocities greater than 200 cm/s.

## CBF and Blood Flow Velocity in Vasospasm

It seems that some method of measuring or indexing CBF in combination with TCD measurements of velocity may offer the most sensitive and specific way to diagnose critical vasospasm. Ischemic deficits from vasospasm occur when the basal vessel diameter becomes reduced to the point of reducing blood flow below levels critical to maintenance of cerebral function. The velocity of blood flowing through the vessel will increase with progressive narrowing until a critical narrowing occurs, diminishing flow. This leads to a nonlinear relationship between velocity and arterial narrowing in severe vasospasm. To correct this effect, a simultaneous index of flow would be helpful. Seiler and Aaslid reported a decreased velocity in the extracranial carotid artery recorded from the neck in SAH patients with intracranial velocities exceeding 200 cm/s.[64]

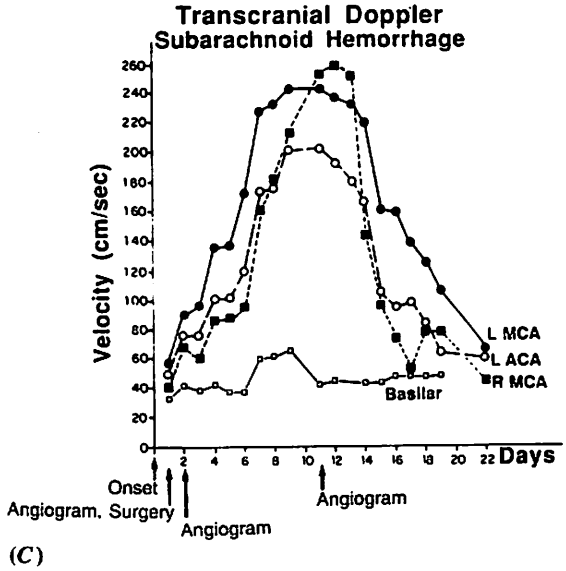



(A)




(B)

**Figure 21-4** TCD recordings from a patient with a grade I SAH from an anterior communicating artery aneurysm. This patient remained asymptomatic despite angiographic vasospasm and increased velocity on TCD ultrasonography. *A.* Angiogram and velocity recordings on day 1 following SAH. *B.* Angiogram and velocity recordings on day 11 following SAH showing vasospasm and increased TCD velocities. *C.* Graph of daily TCD velocity recordings in the same patient following SAH.




(C)

This presumably was due to a reduction in volume flow secondary to the increase in vascular resistance caused by the vasospasm. Lindegaard et al. have applied the concept of the velocity ratio of the MCA/ICA ($V_{mca}/V_{ica}$) to compensate for changes in CBF.[40] A $V_{mca}/V_{ica}$ ratio greater than 3 was found to correspond to vasospasm seen on angiography. It was also found that the $V_{mca}$ was lower when the spasm was widespread. The $V_{mca}/V_{ica}$ ratio was useful in this setting because it partially corrected for reduced velocities in the MCA caused by a decrease in flow. This ratio may prove to be useful for detecting vasospasm after head injuries because the hyperemia that often occurs may also increase flow velocities beyond the normal range.

Sekhar et al. followed a series of 21 patients with SAH from aneurysm rupture using TCD and CBF measurements performed with stable xenon CT-CBF studies and $^{133}Xe$ washout studies.[66] It was found that increases in TCD velocities preceded the onset of delayed ischemic deficits. In addition, CBF values were decreased in areas of the brain fed by vessels that had high velocities on TCD ultrasonography.

Other recent studies have examined the role of single photon emission CT (SPECT) in determining perfusion deficits due to vasospasm. Focal perfusion deficits can be seen using SPECT when vasospasm of the basal cerebral vessels reaches critical levels.[18,35,50] Perfusion deficits caused by distal vessel spasm can also be detected in cases where TCD may not indicate significant spasm.

### Effect of Treatment on Blood Flow Velocity

The effect of various treatments for vasospasm on basal vessel velocity values has also been examined. Kassell et al.[30] have reported lower velocity values by TCD as well as decreased angiographic vasospasm in patients treated with the calcium channel blocker nicardipine versus those treated with placebo. Marked persistent decreases in velocity values by TCD indicating reversal of vasospasm have been observed following transluminal angioplasty treatment of vasospasm. Newell et al.[49,50] reported persistent decreases in velocity values in patients treated by angioplasty, as well as improvements in perfusion indicated by SPECT.

## Occlusive Vascular Disease

### Intracranial Occlusive Vascular Disease

Intracranial stenosis due to atherosclerosis appears less frequently than similar lesions of the extracranial vessels but can be a source of transient ischemic attacks (TIAs). Arteriography has until now been the only way to diagnose these lesions. In patients presenting with TIAs who have normal extracranial noninvasive findings, TCD ultrasonography may be a valuable adjunct to identify those patients with intracranial stenotic lesions. In a study of 11 patients with intracranial occlusive disease, Lindegaard et al. found a clear-cut inverse relationship between the residual lumen diameter of the intracranial stenosis and velocity readings on TCD ultrasonography.[37] It was also noted that with severe stenosis of the proximal MCA, recordings distal to the stenosis revealed an abnormally low velocity with a dampened pulse wave indicating flow reduction.

Velocities that are increased above normal in a focal location, as well as a side to side difference, and sonographic evidence of disturbed flow are all indicators of intracranial stenosis.[36,37,45] Intracranial stenosis can also occur as a consequence of sickle cell anemia and result in cerebral infarction. Adams et al.[8] have established TCD criteria to identify patients with sickle cell disease who are at risk for cerebral infarction and may benefit from transfusion therapy.

MCA occlusions due to thrombosis or embolism can also be detected by TCD ultrasonography, which can reveal low velocities proximal to the occlusion and absence of a signal distal to the occlusion. The ability to detect MCA occlusions rapidly and noninvasively may have a role in identifying candidates for thrombolytic therapy or in following the time course of occlusion and recanalization and possibly to estimate the prognosis of acute stroke.

Halsey recorded MCA blood flow velocities in the hemisphere opposite to the symptoms using TCD ultrasonography in 15 patients presenting with complete hemiplegia of less than 12 h duration.[22] The mean velocities were greater than 30 cm/s in seven patients, five of whom made a complete or partial recovery. Velocities of less than 30 cm/s were recorded in the remaining patients. One patient recovered completely and the other seven patients were left with a total paralysis of the hand and arm. Brass et al.[14] have noted that in addition to an absent signal in the MCA, MCA trunk occlusion can be associated with an increased velocity of the ipsilateral ACA due to collateral flow. This positive diagnostic finding gives added confidence in determining MCA occlusion. Karnik et al.[29] have described the use of TCD to monitor the effectiveness of thrombolytic therapy for acute stroke.

### Extracranial Occlusive Vascular Disease

TIAs and stroke can be caused by a variety of mechanisms. Emboli from and hemodynamic effects of extracranial carotid lesions can both play a role. In evaluating patients with carotid bifurcation disease, it can often be difficult to tell which of these mechanisms is responsible. Doppler ultrasonography has achieved a high degree of accuracy in diagnosing extracranial stenosis and identifying hemodynamically significant lesions in the extracranial carotid arteries. The addition of TCD ultrasonography has the advantage of obtaining velocity recordings directly from the arteries supplying the major brain territories. It is therefore possible to assess the final hemodynamic effect due to the extent of extracranial occlusive disease and the adequacy of the collateral network, which is variable.

When extracranial occlusion or stenosis causes a hemodynamic change in the MCA, generally the pattern that is seen is a decrease in velocity and a dampened pulse wave ipsilateral to the lesion. Studies have thus far shown that there is a variable effect of extracranial carotid occlusion or stenosis on MCA signals.[58,63] Some patients maintain normal MCA velocities distal to a carotid occlusion, whereas other patients have reduced velocities.

Schneider et al. reported TCD findings in 39 patients with ICA occlusion and showed a statistically significant decrease in velocity and pulsatility in the MCA ipsilateral to the occlusion.[63] It was also possible in this group of patients to evaluate the sources of collateral flow from other regions of the circle of Willis.

The effects of ICA occlusion on cerebral vasomotor reactivity have also been studied. Ringelstein et al. performed a study of $CO_2$-induced vasomotor reactivity by recording the changes in MCA velocity induced by $Pa_{CO_2}$ changes using TCD ultrasonography.[58] The study included 40 normal subjects, 40 patients with unilateral ICA occlusion, and 15 patients with bilateral ICA occlusions. The results showed that in patients with a unilateral ICA lesion, vasomotor reactivity was reduced significantly in both

hemispheres but to a greater degree ipsilateral to the occlusion. In the bilateral occlusion group there was also a significant reduction in vasomotor reactivity in both hemispheres compared to normal. In addition, there was a significant decrease in vasomotor reactivity in symptomatic compared to asymptomatic patients with unilateral carotid occlusions.

In a group of patients with ICA occlusion, Halsey and Tan[25] identified a subgroup of patients on the basis of low mean velocity and pulsatility in the ipsilateral MCA who were at high risk for subsequent stroke. Similarly, Kleiser and Widder[32] identified a subgroup of patients with carotid occlusion who were at high risk for subsequent stroke because of impaired hemodynamic reserve determined by $CO_2$ reactivity testing using TCD. These studies suggest that using TCD ultrasonography in combination with physiologic testing may prove useful in identifying patients with cerebrovascular hemodynamic insufficiency.[57]

## Head Injury

Although TCD ultrasonography measures velocity and not flow directly, changes in flow can be detected and CBF changes under various conditions can be assessed. For example, Markwalder et al. found that changes in the MCA velocity ($V_{mca}$) correlated well with changes in the levels of arterial $P_{CO_2}$ in normal human subjects.[43] The $V_{mca}$ changed with expected changes in cerebral blood flow evoked by changes in $Pa_{CO_2}$. With alterations in $Pa_{CO_2}$, the distal cerebral vasculature responds by constricting or dilating, thereby changing CBF. Assuming that the diameter of the basal arteries does not change with $Pa_{CO_2}$ changes, a change in velocity readings obtained will directly reflect changes in CBF. A study by Aaslid has demonstrated rapid changes in velocity in the PCA reflecting changes in CBF induced by light and dark stimuli on the retina.[2] These changes occurred within 2.3 s of the stimulus and reflected blood flow changes to evoked cortical activity. An excellent correlation has been established between MCA velocity changes and volume flow changes in the ICA determined by an electromagnetic flowmeter, caused by moderate blood pressure fluctuations in patients undergoing carotid endarterectomy.[39] A strong correlation between CBF changes and basal artery velocity changes due to changes in ICP has also been observed in an experimental animal model.[10] Therefore, relative changes in CBF due to $CO_2$ changes, evoked cortical activity, and changes in cerebral perfusion pressure can be examined in head-injured patients. Autoregulation has been studied using TCD ultrasonography to record velocity changes induced by rapid changes in blood pressure as well as changes induced by slower spontaneous changes in blood pressure.[20] The validity of the TCD method in determining dynamic autoregulation has recently been confirmed (Fig. 21-5).[6,47] It has also been demonstrated using this method that dynamic autoregulation is impaired following head injury and can be improved in head-injured patients by hyperventilation.[72]

Continuous monitoring of MCA velocity along with other physiologic parameters has also been described in head-injured patients. Insights into circulatory changes, including those seen

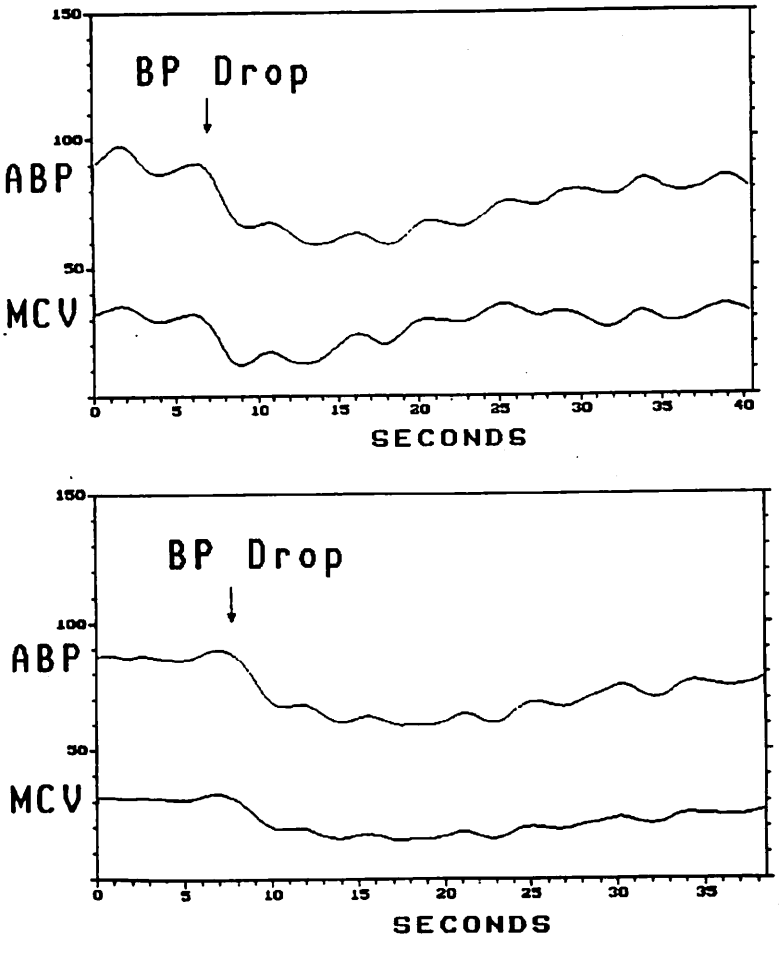



**Figure 21-5** Example of intact *(upper)* and impaired *(lower)* autoregulation on the noninjured and injured sides, respectively, of a patient with an acute subdural hematoma. The arterial blood pressure (shown in mmHg) is briefly lowered by quickly releasing cuffs around the patient's thighs. The response of the MCA velocity (shown in cm/s) as an index of flow adjustment is observed. Abbreviations: BP, blood pressure; ABP, arterial blood pressure; MCV, middle cerebral velocity.

during intracranial pressure B waves, have been obtained.[48] Future development of this technology will include simultaneous bilateral monitoring of both MCAs.[48,72]

Vasospasm has also been reported after head injury and can in some cases lead to clinical deterioration. Because either vasospasm or increased CBF can cause an increase in velocity, these two conditions must be differentiated. Distinctions between the two conditions can be made by the following: (1) With cerebral hyperemia, all vessels insonated tend to have high velocities in contrast to vasospasm, which tends to have a more focal nature. (2) If CBF studies are done simultaneously, CBF increases will parallel increases in velocity on TCD ultrasonography with cerebral hyperemia in contrast to significant vasospasm, where CBF will decrease. (3) $V_{mca}/V_{ica}$ ratios will be higher in vasospasm than in cerebral hyperemia. Grolimund et al. found that two groups exist with high velocities after head injury, one group with increased $V_{mca}/V_{ica}$ ratios and another with lower $V_{mca}/V_{ica}$ ratios.[21] It is likely that these two groups represent on the one hand, patients with mild vasospasm, and on the other hand, patients with cerebral hyperemia.

Several recent studies using TCD to detect post-traumatic vasospasm have indicated that this condition may be more common than previously appreciated.[17,44,73] It seems that post-traumatic vasospasm is associated with SAH after head injury and can cause delayed ischemic neurologic deficit and infarction.[44,50,52,73]

Although ICP levels cannot be determined directly from TCD ultrasonography, Aaslid et al. showed that in a group of patients undergoing ventricular infusion tests for hydrocephalus, decreased cerebral perfusion pressure could be detected by analysis of the TCD wave forms.[4] Chan et al.[16] have demonstrated that low cerebral perfusion pressure can be detected using TCD by examining mean velocity and pulsatility values. Low mean velocity values in the MCA have also been shown to indicate a poor outcome following head injury.[15] Kety et al.[31] were among the first to demonstrate that CBF decreases at high ICP levels, by using quantitative measurements of CBF and ICP. Several investigators using TCD ultrasonography to record from the intracranial vessels have reported a progressive decrease in the diastolic wave form with compromised cerebral perfusion pressure due to increased ICP, progressing to a reverberating pattern that occurs with cerebral circulatory arrest.[28,52] This pattern occurs when flow is obstructed at the microcirculatory level and the conducting vessel absorbs the arterial pulse wave, distending in systole and contracting in diastole (Fig. 21-6). The detection of a reverberating pattern documenting the arrest of the cerebral circulation may prove to be a useful confirmatory test in determining brain death.[28,52]

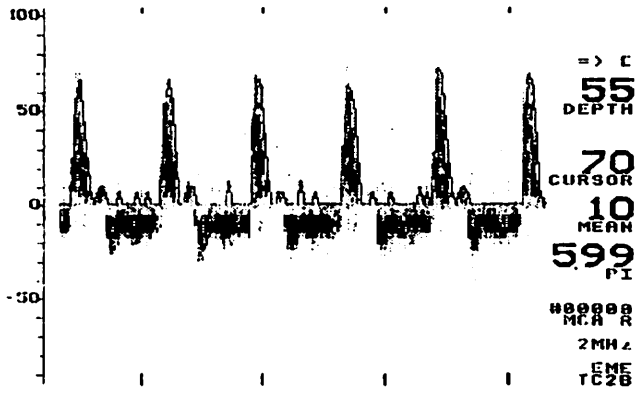



**Figure 21-6** The "to and fro" pattern seen in the MCA following cerebral circulatory arrest.

## Surgical Monitoring of CBF Changes

TCD ultrasonography has been used to monitor MCA velocities both during carotid endarterectomy (CEA) and cardiopulmonary bypass.[41,53] Using a headband and a movable probe, it is possible to obtain a continuous-velocity signal from the MCA and thus a measurement of the changes in blood flow during various manipulations such as alteration of blood pressure, onset of cardiopulmonary bypass or carotid cross-clamping, and during reperfusion.[55,56]

During carotid endarterectomy, several methods of assessing the adequacy of the CBF during cross-clamping have been used. These methods have included assessment of cerebral activity, using electroencephalography (EEG) or functional testing during surgery under local anesthesia. Blood flow has also been assessed more directly using stump pressure measurements or regional CBF studies. The results of TCD monitoring during endarterectomy have been reported by several groups.

Halsey et al. compared regional CBF using $^{133}Xe$ to TCD recordings of MCA velocity while simultaneously recording EEG in eight patients undergoing CEA.[24] It was found that there was a considerable variability in the relationship between the mean velocities and the CBF. The systolic/diastolic ratio was more sensitive in detecting changes due to cross-clamping than the mean velocity. It was concluded that while CBF measurements are reflecting cortical blood flow, the TCD tracings may be more indicative of the flow in the basal vessels reflecting blood supply to the deeper perforating vessels.

Spencer et al.[70] have recently compared MCA velocity changes to carotid stump pressures during cross-clamping for carotid endarterectomy. Comparison of carotid stump pressure to percentage drop in MCA velocity yielded an exponential curve with the velocity reaching zero at a stump pressure of 15 mmHg. It was concluded that the post-cross-clamp MCA velocity of 40 percent of the baseline value, 10 to 15 s after clamping (to allow for autoregulatory adjustment), corresponded to a stump pressure of approximately 25 mmHg. All of the hypoperfusion-related complications occurred when the MCA velocity fell below 40 percent of the baseline after cross-clamping and therefore this value represented a reasonable cut-off for the determination of a safe perfusion limit.

Halsey[23] reported the results of a multicenter study using TCD to help determine the role of shunting during carotid endarterectomy. If MCA velocity values fell below 15 percent of baseline after cross-clamping, there was a significantly higher stroke rate in those patients not shunted. If MCA velocity remained above 40 percent of baseline, there was a higher stroke rate in the shunted patients. The difference in stroke rate in shunted and nonshunted patients, when the MCA velocity dropped to between 16 and 40 percent of baseline, was not significantly different. The cross-clamp times, however, were short, so it may be prudent to shunt this group of patients if cross-clamp times approach 1 h, but this remains speculative.

## Arteriovenous Malformations

Initial experience using TCD ultrasonography to evaluate patients with arteriovenous malformations (AVMs) has revealed several findings.[38] Feeding arteries to a large AVM show high flow velocities and a low pulse pressure on TCD ultrasonography because of their conduction of high blood flow to a low-resistance vascular

Altered $CO_2$ responsiveness has been demonstrated when comparing in the same patient AVM feeding vessels to other arterial feeding areas of normal brain. The utility of TCD ultrasonography in the management of AVMs is yet to be established, but possible uses include noninvasive detection, serial follow-up in patients undergoing radiation treatment,[46] and possible use in staged obliteration.[54]

## Detection of Emboli

Detection of microemboli within the circulation is possible using Doppler ultrasound and has been used routinely by neurosurgeons to prevent excessive air emboli during neurosurgical procedures. Spencer[69] used this method to detect air emboli in humans and in animals after decompression from hyperbaric conditions. Subsequently, it has been recognized that TCD can detect intracranial air and formed element emboli under a variety of circumstances. Intracranial air emboli were first detected during carotid endarterectomy and cardiopulmonary bypass using TCD; recent studies have reported a correlation between the degree of air embolization and neurologic outcome following cardiopulmonary bypass.[71] It has also been recognized that TCD can be used to detect intracranial formed element microemboli experimentally[42,61] and in patients with TIAs or recent stroke.[34,67,69] Intracranial microemboli have also been observed using TCD in patients with prosthetic heart valves, atrial fibrillation, carotid bifurcation atherosclerosis, and other intra-arterial lesions.[69] One of the uses of this technique is to help determine the source of emboli in stroke-prone patients. This capability represents one of the new and exciting aspects of TCD technology, and the development of automated emboli detection computer software is underway. Figure 21-7 illustrates the signature of an intra-arterial embolus detected by TCD.

## Color Flow TCD

One of the recent developments in TCD technology has been the introduction of transcranial color-coded real time ultrasonography

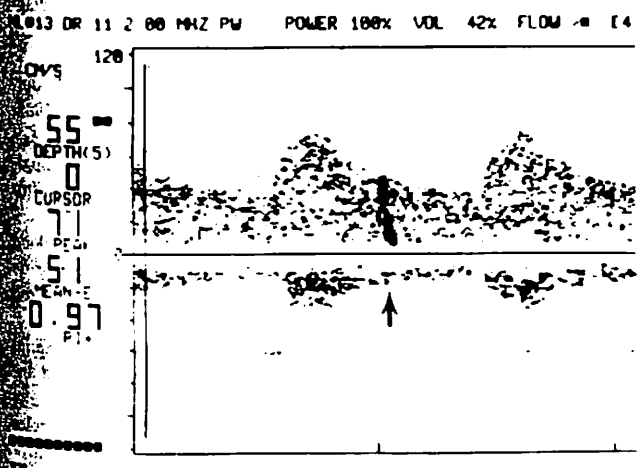



**Figure 21-7** Example of the characteristic signature *(arrow)* of an intra-arterial formed element embolus in the spectral tracing from the MCA. This patient had intracranial ICA stenosis and was having TIAs.

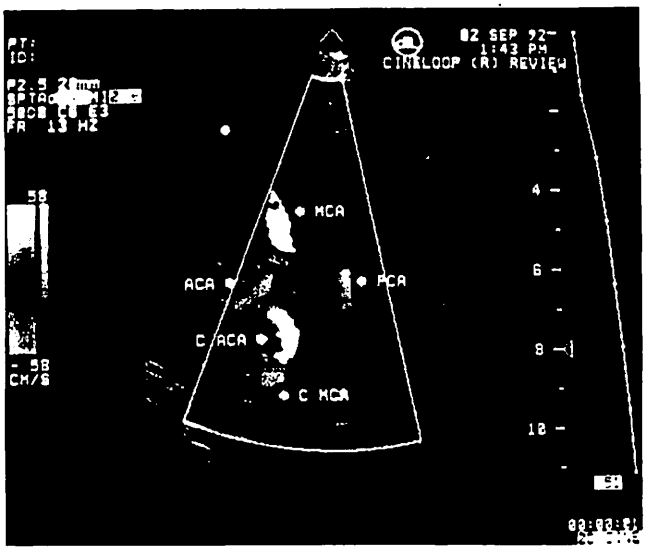



**Figure 21-8** Example of an image (printed in black and white) from a color flow TCD examination illustrating part of the circle of Willis. Abbreviations: MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; CACA, contralateral ACA; CMCA, contralateral MCA.

(TCCU).[11,12] This technique has evolved by combining transcranial B-mode imaging with color-coded displays of intravascular blood flow and standard low-frequency real-time TCD spectral analysis. The advantages of this technique include improved vessel identification and anatomic images of brain structures under normal conditions as well as during pathologic states. It has also been reported that 75 percent of aneurysms causing SAH may be detected noninvasively using this technique.[12] Figure 21-8 illustrates an image (printed in black and white) of the circle of Willis obtained using TCCU.

## Summary

TCD ultrasonography provides a new avenue to study the human cerebral circulation and seems to be very useful in the evaluation of a variety of clinical conditions. It provides for the first time a noninvasive way to assess the degree and extent of vasospasm occurring after SAH. In this way it may be useful in identifying patients at high risk for developing ischemic defects. It also has applications in the evaluation of occlusive cerebrovascular disease such as the diagnosis of intracranial stenosis and occlusion, as well as the assessment of the hemodynamic effects of extracranial disease. In addition, TCD ultrasonography seems useful in monitoring changes in CBF that occur under a variety of clinical conditions, including closed-head injury and other conditions that lead to increases in ICP.

The noninvasive assessment of cerebral autoregulation is now possible using TCD ultrasonography. Its ability to detect intracranial arterial emboli from a variety of causes will facilitate the determination of the embolic source in many patients threatened by stroke. It will also provide a means to gather much needed information on the pathogenesis of certain types of stroke and TIAs as well as a means to evaluate various treatment modalities.

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