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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.²⁵ Since that time, the technology has undergone significant development and refinement. Presently Doppler ultrasonography is extensively used 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.

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, 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. 9-1). 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 pro-



Figure 9-1 Transillumination of the skull illustrates the thin portions of the temporal bone where ultrasound can penetrate.

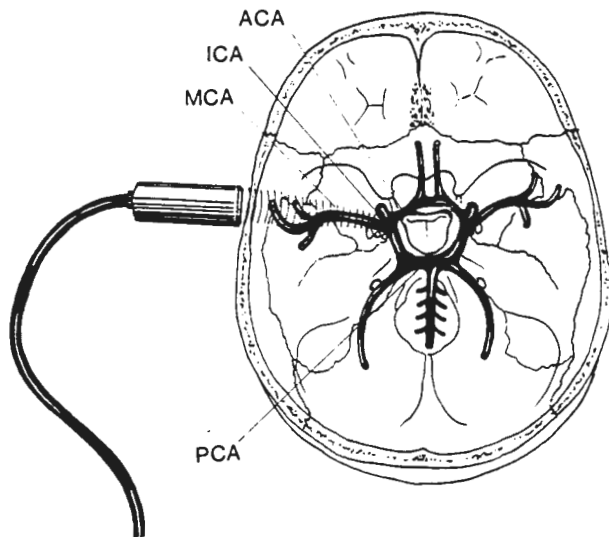


Figure 9-2 Recording from the middle cerebral artery through the transtemporal route. Signals can also be obtained from the ICA, ACA, and proximal PCA through this route.

found 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. 9-2). The transorbital route can be used to examine the ophthalmic artery and ICA. Using the transoccipital approach, signals

can be obtained from the vertebral arteries (VAs) and basilar artery (BA).

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.¹⁸ Bishop et al. have compared TCD velocities to cerebral blood flow (CBF) measurements obtained in human subjects using xenon 133.⁸ They demonstrated that although resting MCA velocity did not correlate very well with CBF, CBF changes induced by varying the P_{aCO_2} did correlate well with the change in MCA velocity. Thus when the diameter of the cerebral basal arteries remain constant, changes in the velocities can accurately reflect changes in flow.

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 is observed (Fig. 9-3). 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.

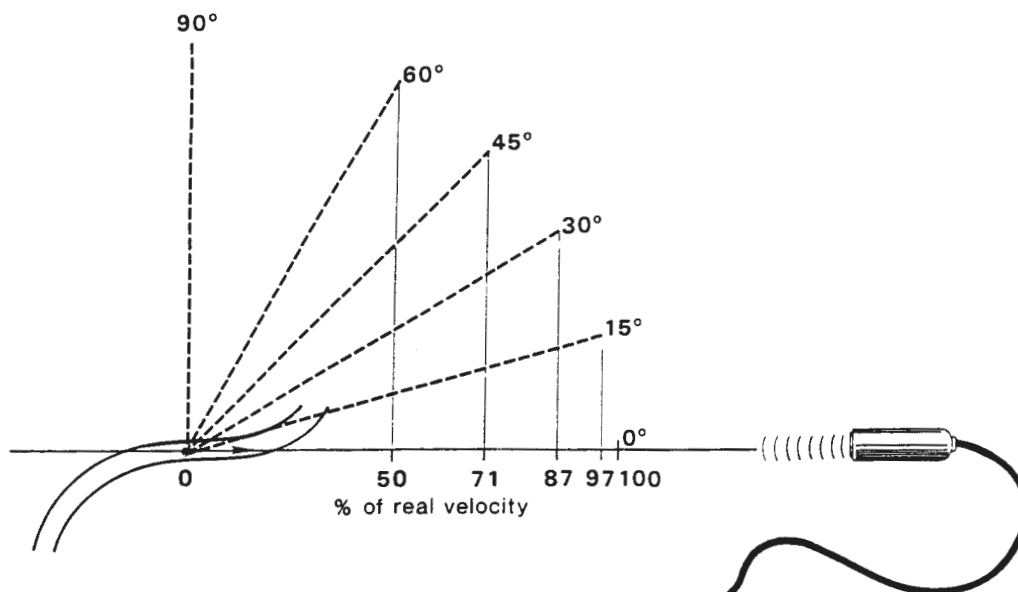


Figure 9-3 The relationship between the angle of insonation and the percentage of the true velocity which will be observed by the recording device.

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 accurately identify vessels, 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 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 ICA, 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 progressively increased 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, which 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 oph-

thalmic 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 use of transcranial Doppler ultrasonography is the 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 diameter of the ACA and MCA on angiogram and the 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 ensue, 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 neurologic 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.

Under normal conditions, blood flow velocities in the middle cerebral artery 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.²⁹ Seiler et al. found in a group of 39 patients with SAH followed with TCD ul-

trasonography that if the blood flow velocities did not exceed 140 cm/s, no patient developed a cerebral infarct.²⁹ 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.⁶ The maximum amplitude of these sounds is heard near the carotid termination. The cause of these murmurs is most likely due to 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.⁷ A very detailed study was performed by Weir et al., who took careful measurements from 627 sets of angiograms from 293 patients with aneurysms.³¹ 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 which can be performed daily to follow the changes that occur.

Several studies using TCD ultrasonography have looked at the time course of vasospasm.^{13,29} In a group of 39 patients with SAH, of whom most were operated on late, vasospasm indicated by an increase in flow velocities on TCD ultrasonography was maximal between days 7 and 12.²⁹ 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.¹³ They found that maximum velocities were reached between the 11th and 20th days after SAH. Figure 9-4 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 neurologic deficits.

Correlation of SAH on CT with Velocity

Several reports have pointed out a strong correlation between the amount of blood detected in the basal cisterns on CT scan after SAH and the subsequent development of vasospasm detected by angiography. Fisher et al. studied a group of patients who had CT scans done within 5 days of

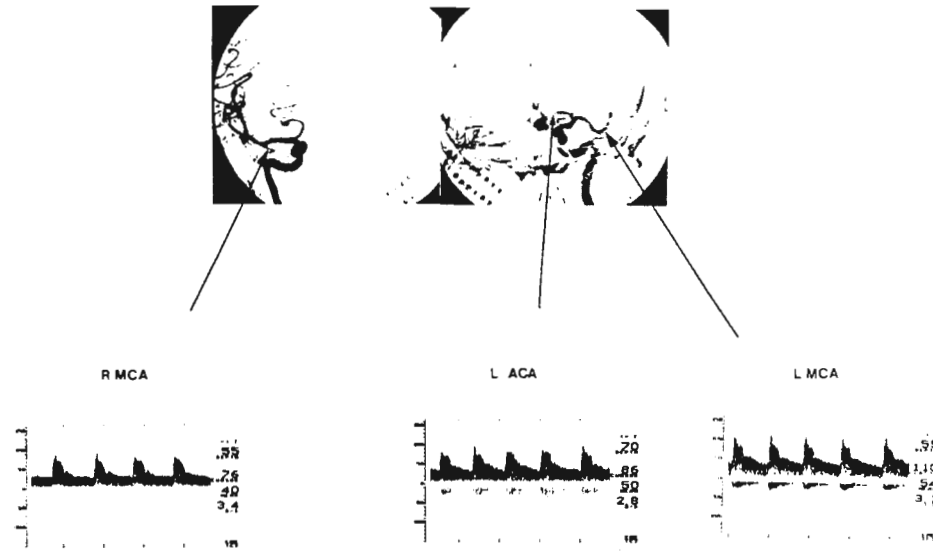
their SAH and defined four groups based on the amount of blood seen: Group 1, no blood detected; Group 2, diffuse deposition or thin layers with all vertical layers of blood less than 1 mm thick; Group 3, localized clots and/or vertical layers of blood 1 mm or greater in thickness; Group 4, diffuse or no subarachnoid blood but intracerebral or intraventricular clots.⁹ Angiograms then done between 7 and 17 days after the subarachnoid hemorrhage 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 after SAH.²⁹ 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. 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.

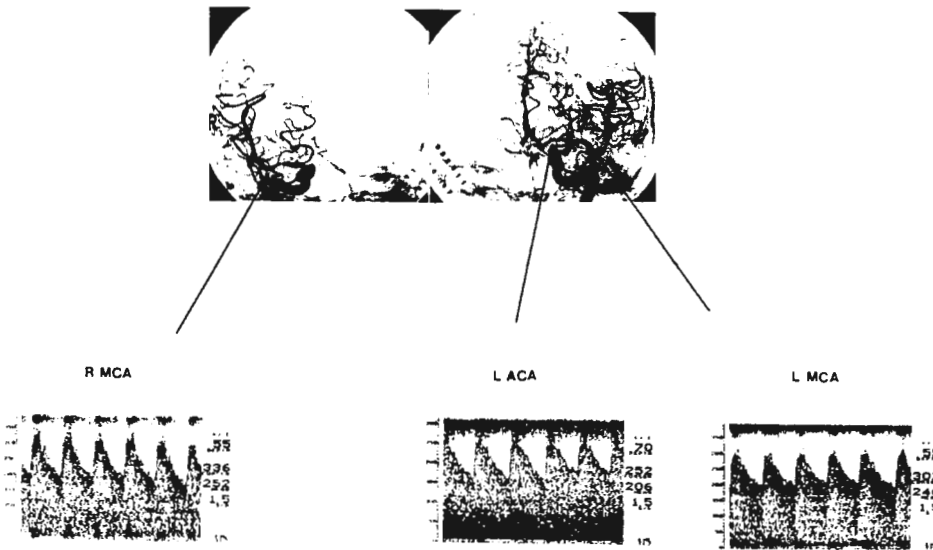
Cerebral Blood Flow and Blood Flow Velocity in Vasospasm

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.²⁸ 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.¹⁶ 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 which 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 ¹³³Xe

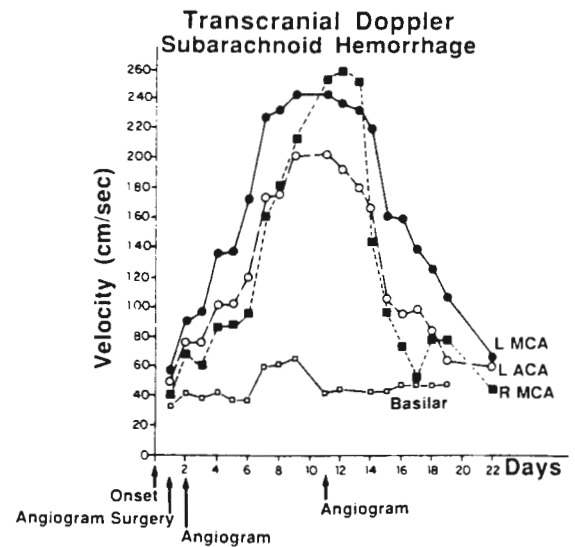


A



B

Figure 9-4 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

washout studies.³⁰ 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. It appears that some method of measuring or indexing cerebral blood flow in combination with TCD measurements of velocity may offer the most sensitive and specific way to diagnose critical vasospasm.

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.¹⁵ It was also noted that with severe stenosis of the proximal middle cerebral artery, recordings distal to the stenosis revealed an abnormally low velocity with a dampened pulse wave indicating flow reduction.

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 hours' duration.¹¹ 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.

Extracranial Occlusive Vascular Disease

Transient ischemic attacks 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 di-

rectly 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.^{24,27} Some patients maintain normal MCA velocities distal to a carotid occlusion, whereas other patients have reduced velocities. Schneider et al. recently reported TCD findings in 39 patients with internal carotid artery occlusions and showed a statistically significant decrease in velocity and pulsatility in the MCA ipsilateral to the occlusion.²⁷ 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 CO₂-induced vasomotor reactivity by recording the changes in MCA velocity induced by PaCO₂ changes using TCD ultrasonography.²⁴ The study looked at 40 normal subjects and 40 patients with unilateral, and 15 patients with bilateral, ICA occlusions. The results showed that in patients with unilateral ICA lesions, vasomotor reactivity was significantly reduced 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. There was a significant decrease in vasomotor reactivity in symptomatic compared to asymptomatic patients with unilateral carotid occlusions. These studies suggest that using TCD ultrasonography in combination with physiologic testing may prove useful in identifying patients with cerebrovascular hemodynamic insufficiency.

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 middle cerebral artery velocity correlated well with changes in the levels of arterial P_{CO₂} in normal human subjects.²⁰ The V_{mca} changed with expected changes in cerebral blood flow evoked by changes in PaCO₂. With alterations in PaCO₂, the distal cerebral vasculature responds by constricting or dilating, thereby changing CBF. Assuming that the diameter of the basal arteries does not change with PaCO₂ 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 posterior cerebral artery reflecting changes in CBF induced by light and dark stimuli on the retina.² These changes occur with 2.3 s of the stimulus and reflect blood flow changes to evoked cortical activity. Autoregulation has also been studied using TCD ultrasonography to record velocity changes induced by rapid changes in blood pressure. One advantage over other CBF measure-

ment methods is that rapid and subtle changes can be detected in humans, allowing the study of phenomena not easily studied otherwise. To use TCD ultrasonography to measure absolute blood flow in human arteries, the cross-sectional area of the artery and the angle of insonation must be known. With further technical developments, this may be possible in the future.

In severe head injury, several pathophysiologic conditions exist that may be evaluated by TCD ultrasonography such as alterations in cerebral blood flow, changes in autoregulation, vasospasm, and increases in intracranial pressure (ICP). There have been only a few reports to date on the use of TCD ultrasonography in head-injured patients, but preliminary findings suggest that it will be a useful diagnostic tool.²⁶ Obrist et al. and others have demonstrated changes in CBF which occur after severe head injury using xenon wash-out studies.²² Decreases in cerebral metabolic rate and CBF paralleled the severity of coma in general, but there was a large group of patients in whom a delayed increase in CBF was found. These increases were out of proportion to the metabolic demands of the brain tissue determined by arteriovenous oxygen differences and CBF. This condition has been termed cerebral hyperemia. There was a positive correlation between the occurrence of cerebral hyperemia and increased ICP.

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.¹⁰ 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.

Although ICP levels cannot be determined directly from TCD ultrasonography, Aaslid et al. have shown 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 Kety et al.¹⁴ 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. 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. 9-5). 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.²¹

Surgical Monitoring of CBF Changes

TCD ultrasonography has been used to monitor MCA velocities both during carotid endarterectomy (CEA) and cardiopulmonary bypass.^{19,23} Using a headband and a movable probe, it is possible to obtain a continuous velocity signal from the middle cerebral artery and thus a measurement of the changes in blood flow during various manipulations such as alteration in blood pressure, onset of cardiopulmonary bypass, or carotid cross-clamping.

During carotid endarterectomy, several methods of assessing the adequacy of the cerebral blood flow during cross-clamping have been used. These methods have included assessment of cerebral activity, using EEG or functional testing during surgery under local anesthesia. Blood flow has also been assessed more directly using stump pressure measurement or regional CBF studies. The results of TCD monitoring during endarterectomy have been reported by several groups. Halsey et al. compared regional CBF using ¹³³Xe to TCD recordings of MCA velocity while simultaneously recording EEG in eight patients undergoing CEA.¹² It

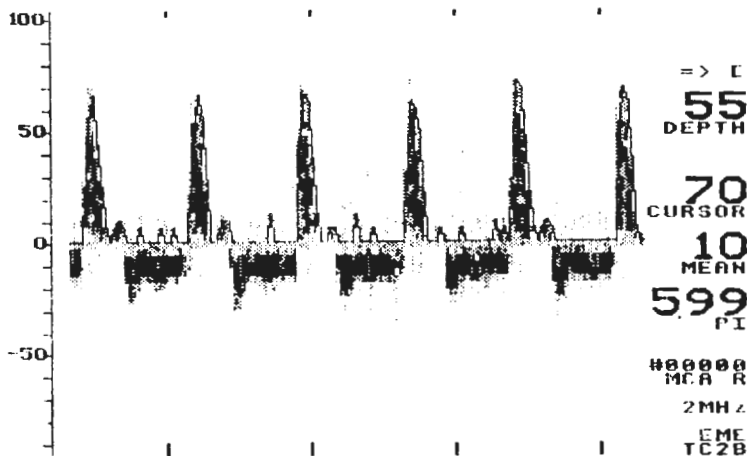


Figure 9-5 The "to and fro" pattern seen in the middle cerebral artery following cerebral circulatory arrest.

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. It remains to be determined if TCD ultrasonography will be a reliable monitoring method during carotid endarterectomy and will be able to predict the need for intraoperative shunt placement.

Arteriovenous Malformations

Initial experience using TCD ultrasonography to evaluate patients with arteriovenous malformations (AVMs) has revealed several findings.¹⁷ Feeding arteries to a large AVM show high flow velocities and a low pulse pressure on TCD ultrasonography due to their conduction of high blood flow to a low-resistance vascular bed. Altered CO₂ responsiveness has been demonstrated when comparing in the same patient AVM feeding vessels to other arteries 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, and possible use in staged obliteration.

Summary

Transcranial Doppler ultrasonography provides a new avenue to study the human cerebral circulation and appears 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. TCD ultrasonography also appears useful in monitoring changes in CBF, which occur under a variety of clinical conditions including closed-head injury and other conditions which lead to increases in intracranial pressure.

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