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Evaluation of Vasospasm Using Transcranial Doppler

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Angiographic vasospasm following subarachnoid hemorrhage (SAH) was described by Ecker and Riemenschneider in 1951. Although now well recognized as a cause of neurological deterioration after SAH, vasospasm has been difficult to diagnose precisely without cerebral angiography. The diagnosis of neurological deterioration from ischemia due to vasospasm generally has been made on clinical grounds after other causes of deterioration have been ruled out. Angiography is the most precise method of assessing the degree and extent of vasospasm after SAH, but clearly there has been a need for a test to diagnose vasospasm that is simpler and easier to repeat than angiography. In 1982, Aaslid introduced the transcranial Doppler, which for the first time permitted a noninvasive assessment of the degree and extent of vessel narrowing due to vasospasm after SAH.

Principles of Doppler Ultrasonography

The Doppler effect was first described by the Austrian physicist Christian Doppler in 1843 to explain different color shifts emitted by heavenly bodies. The wave theory of light states that color perceived is dependent on the frequency of the waves, and anything that changes this frequency would change the perceived color. The Doppler effect describes a shift in frequency of a wave when either the transmitter or the receiver of the wave are moving with respect to the wave propagating medium. If sound is emanating from or reflected by an object moving toward an observer, it will have a higher frequency in proportion to the speed of the moving object. On the other hand, sound emanating from an object moving away from an observer will have a lower frequency in proportion to the speed of the moving object. Using this principle, it has been possible to use ultrasound to measure the velocity of flowing blood. The measurement of the velocity of flowing blood in the peripheral vessels was first reported in 1959 by Satomura. The ultrasound probe emits a high frequency sound, which is reflected by the moving blood cells and transmitted back to the probe. The frequency shift of the reflected ultrasound will be proportional to the velocity of the flowing blood. By recording and processing these frequency shifts, the velocity can be calculated.

Doppler ultrasound is widely used to examine the extracranial and peripheral vessels. Both continuous wave and pulsed Doppler systems using frequencies between 3 and 10 megahertz (MHz) are used for this purpose. Continuous wave Doppler constantly transmits an ultrasonic beam and simultaneously receives the reflected ultrasound; thereby it will measure frequency shifts of flowing blood anywhere throughout the path of the ultrasound. Pulsed Doppler equipment sends bursts of ultrasound at regular intervals and also receives the ultrasound at certain precise intervals. Using this technique, pulsed Doppler equipment can record from specific targets at preselected depths. Duplex scanning combines the use of B-mode ultrasound, and by rapidly alternating between these two modes, simultaneous images are obtained from the vessels being recorded.

Transcranial Doppler

Transcranial Doppler (TCD) provides the capability of recording flow velocities directly from the intracranial vessels. The ultrasound is able to penetrate through the thin portions of the temporal bone by selecting a 2 MHz ultrasonic frequency. It employs a pulsed range gated design that permits the selection of specific recording sites on the basal vessels. There are three examination routes or "windows" that are used for obtaining signals from the intracranial vessels: the transtemporal, transorbital, and transoccipital routes. Through the transtemporal route, signals are obtainable from the middle cerebral artery, anterior cerebral artery, intracranial internal carotid artery, and proximal posterior cerebral artery.

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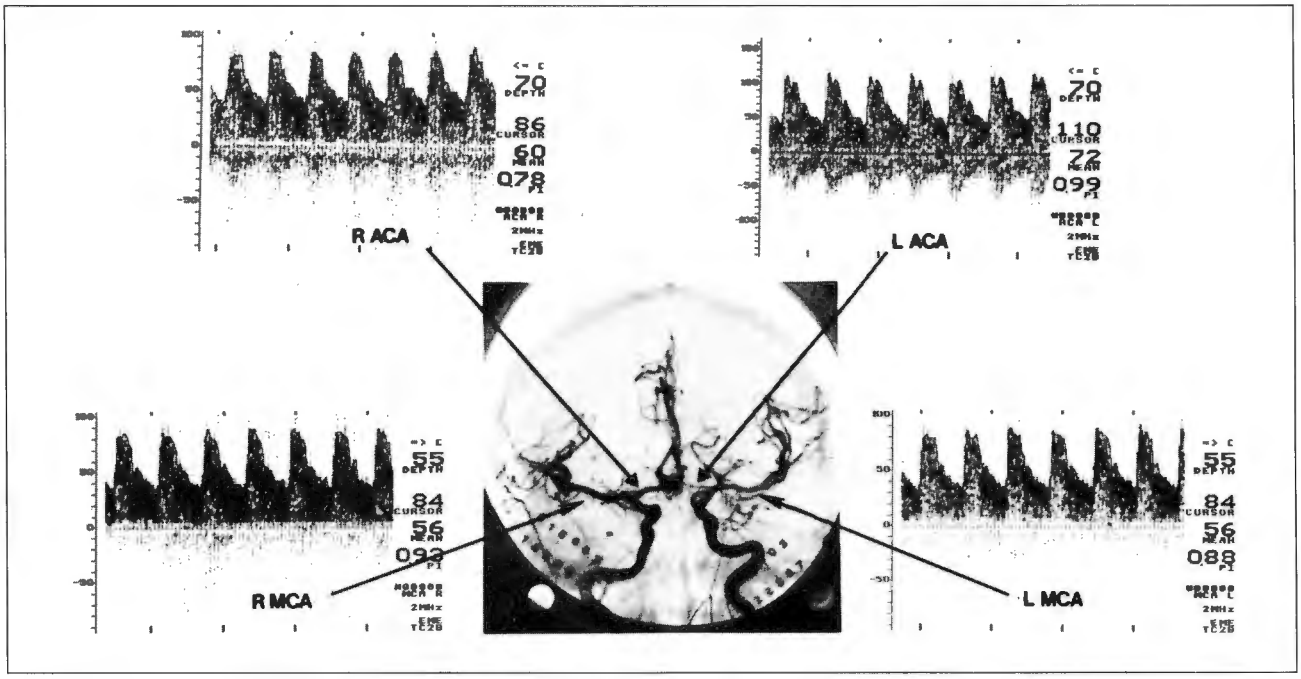

Figure 1A. Angiogram and transcranial Doppler velocity recordings on the day of subarachnoid hemorrhage in a patient with an anterior communicating artery aneurysm. *(R: right, L: left, MCA: middle cerebral artery, ACA: anterior cerebral artery.)*

The transorbital route can be used to examine the ophthalmic artery and the internal carotid artery. The transoccipital route is used to obtain signals from the vertebral and basilar arteries.

With the ability to record flow velocities directly from the intracranial arteries, TCD has broadened the applications of Doppler ultrasound. Preliminary studies indicate that TCD will be useful not only in the diagnosis of vasospasm, but also in the diagnosis of a variety of other disorders of the intracranial circulation that occur with extracranial and intracranial occlusive disease, head injury, arteriovenous malformations, and conditions that lead to increases in intracranial pressure.

Examination Techniques

In post-SAH patients, the most useful information is obtained from the transtemporal and transoccipital windows. The first step in examining the patient is to find the middle cerebral artery through the transtemporal window. The middle cerebral artery can be followed from the upwardly directed insular branches to its origin at the internal carotid termination. Ultrasound gel is applied to an area in front of the ear, just above the zygoma. The depth can be set at 45 mm and the probe is directed slightly rostral and anterior. The signal is followed to a depth of 60 to 65 mm where a bidirectional signal is found under normal conditions. This confirms the position on the internal carotid termination. From this point, the anterior cerebral artery is located at approximately 70 mm and slightly rostral. The posterior cerebral artery is located slightly posterior and the internal carotid artery is located slightly caudal to this position at 65 to 70 mm. To examine the vertebrobasilar system, the patient's neck is flexed, and the probe is placed on the posterior aspect of the neck at the level of the second cervical vertebra and aimed toward the clivus. The vertebral arteries are located on either side of the midline and can be followed from a depth of approximately 45 to 85 mm. The basilar artery can be followed in the midline from a depth of about 85 to 105 mm. The extracranial internal carotid artery also can be examined to record ratios of intracranial to extracranial velocities. The probe is placed at the angle of the mandible and directed upward toward the base of the skull with the depth setting at 45 mm.

Rationale for Using TCD to Detect Vasospasm

The most significant changes in vessel diameter induced by vasospasm usually occur in the basal arteries.

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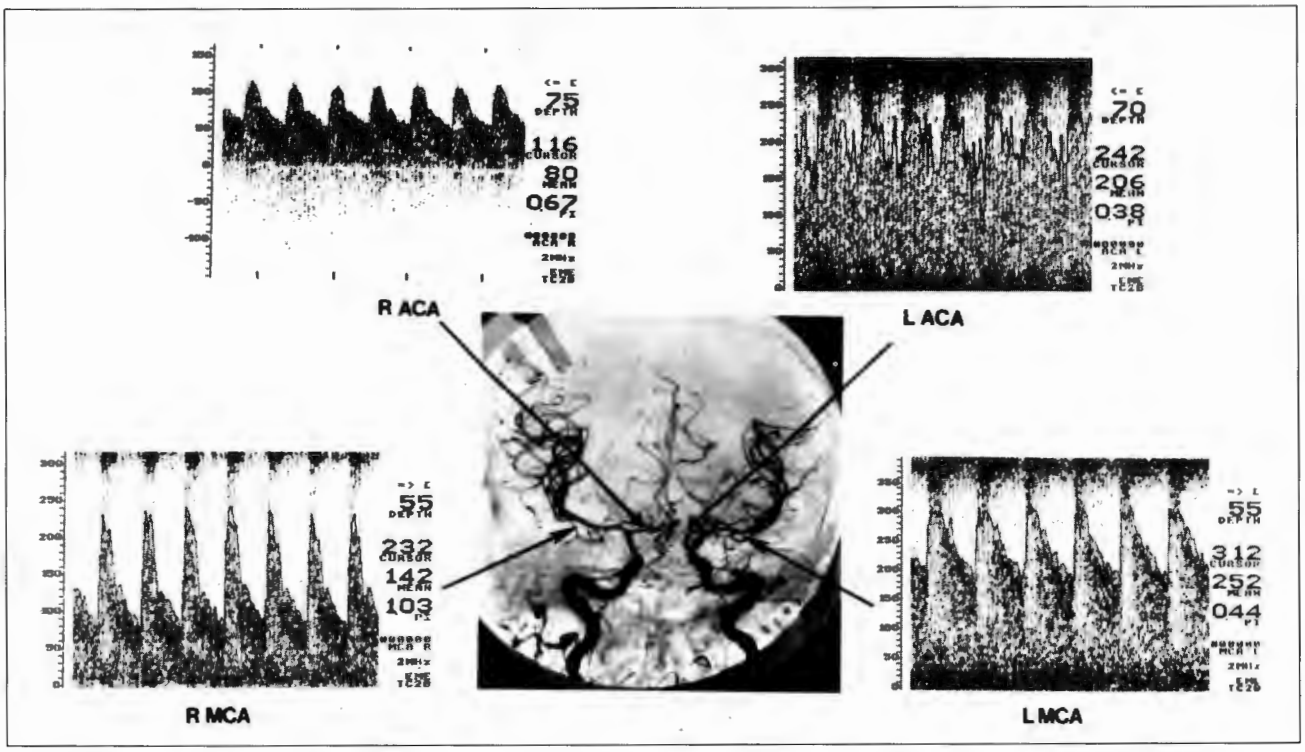

Figure 1B. Angiogram and transcranial Doppler recordings on day 6 following subarachnoid hemorrhage in the same patient. *(R: right, L: left, MCA: middle cerebral artery, ACA: anterior cerebral artery.)*

When vasospasm causes a vessel to narrow, the blood flow velocity must increase through this narrowed segment in order to maintain the same flow. In cases of vasospasm, velocities exceeding normal resting values by 5 to 6 times have been seen. The resistance across the stenotic segment is not only related to the degree but also to length of the stenosis. Thus, the effect of vessel narrowing on cerebral blood flow (CBF) reduction after SAH will depend on the degree and extent of vessel narrowing, arterial blood pressure, and the ability of the cerebral circulation to autoregulate and compensate for the vasospasm. The adequacy of the collateral circulation also plays a role. When CBF is reduced to critical levels by severe vasospasm, neurological deficits will ensue. TCD may alert the clinician to the degree and extent of vasospasm, thereby allowing institution of proper therapy before neurological deficit or infarction takes place.

Although velocity recordings can be obtained from many of the intracranial arteries, the middle cerebral arteries are the most ideally suited for TCD recordings in vasospasm. Because of the limited collateral network under normal conditions, there is a close correlation between the amount of vasospasm and the increase in velocity seen with TCD. The other major intracranial arteries generally have collateral branches, and depending on the degree of the collateral network, the relationship between increased velocity and spasm may not be as close. Under normal conditions, mean blood flow velocities in the middle cerebral artery range between 30 and 80 cm/sec with an average value of 62 cm/sec. Middle cerebral arteries that are classified as vasospastic on angiography demonstrate mean velocities of 120 cm/sec or more. Mean velocities > 200 cm/sec correspond to severe vasospasm demonstrated by angiography. Increased velocities are also commonly seen in the terminal internal carotid arteries and proximal anterior cerebral artery in vasospasm, but less commonly seen in the posterior cerebral arteries, especially with anterior circulation aneurysms. Increased velocities in the vertebral and basilar arteries indicating vasospasm have also been seen. Figure 1 illustrates velocity recordings before and during vasospasm in a patient with an anterior communicating artery aneurysm. Figure 2 shows a delayed cerebral infarction from vasospasm in the same patient.

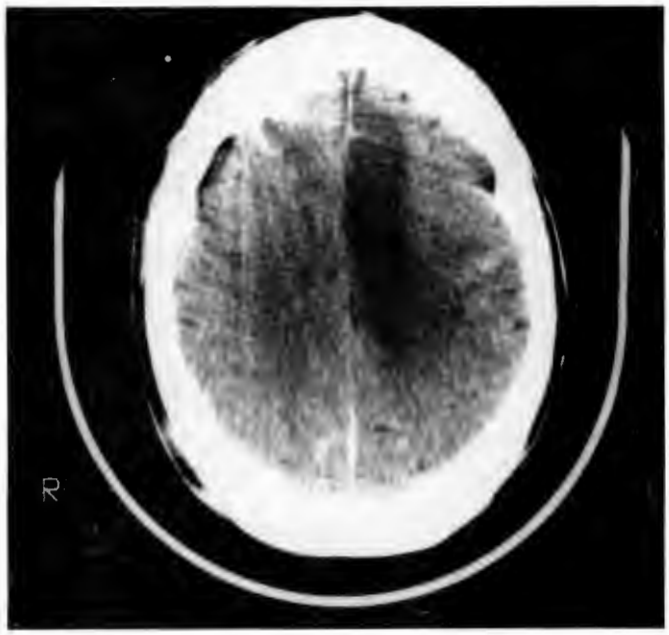

Figure 2. CT scan on the patient in Figure 1 showing a delayed left anterior cerebral artery distribution infarct.

Another reported finding in vasospasm is the occurrence of musical murmurs heard best near the carotid termination. These murmurs most likely result from the creation of pure tone frequencies caused by the vibrations of the arterial walls as the blood is in transition between laminar and turbulent flow. The frequency of the tones appears to correlate with the velocity and, therefore, the degree of vasospasm.

Correlation of Clinical Course

Although it is now well established that vasospasm is an important cause of clinical deterioration after SAH, the factors leading to ischemia and clinical deterioration are complex. It has been noted that the clinical condition of the patient does not always correlate with the degree of vasospasm found by angiography. For example, some patients can remain asymptomatic with severe vasospasm demonstrated by angiogram. Several studies have been done comparing the clinical course of patients after SAH to the TCD velocities. It has been shown that the majority of patients who developed delayed transient neurological deficits after SAH had mean velocities of > 140 cm/sec. In patients who went on to develop an infarction due to vasospasm, the majority had mean velocities of > 200 cm/sec recorded. It has been noted that patients with early rise in velocities to high levels during the first five days after SAH are at an especially high risk for developing delayed ischemic deficits (Figure 3).

Cerebral Blood Flow and Blood Flow Velocity

When vasospasm becomes severe enough to reduce blood flow below critical levels, ischemic deficits will ensue. The velocity through any particular vessel will increase through the point of narrowing until a critical diameter occurs and diminishes flow. When the flow starts to decrease, this can lead to a reduction in the velocity measurement in severe vasospasm. This effect can be partially corrected by obtaining a simultaneous index of CBF. It is possible to obtain velocity signals from the extracranial internal carotid artery in the neck using TCD. It has been found that the velocity readings recorded from this area will decrease in cases of severe vasospasm. This decrease presumably is due to a reduction in volume flow secondary to the increased vascular resistance caused by the vasospasm. One can express the velocity ratio of the middle cerebral artery to the internal carotid artery as Vmca/Vica and record these ratios. In severe vasospasm, as the middle cerebral artery velocity reaches levels high enough to be associated with reductions in blood flow, the extracranial internal carotid artery velocity reading will decrease, further increasing the value of the ratio. By recording the velocity readings in this manner, it may be possible to partially correct the effect of lowered CBF and obtain a more sensitive index of critical vasospasm. It has also been shown that if CBF measurements are obtained using Xenon 133 in cases with ischemic deficits due to vasospasm, CBF values can de-

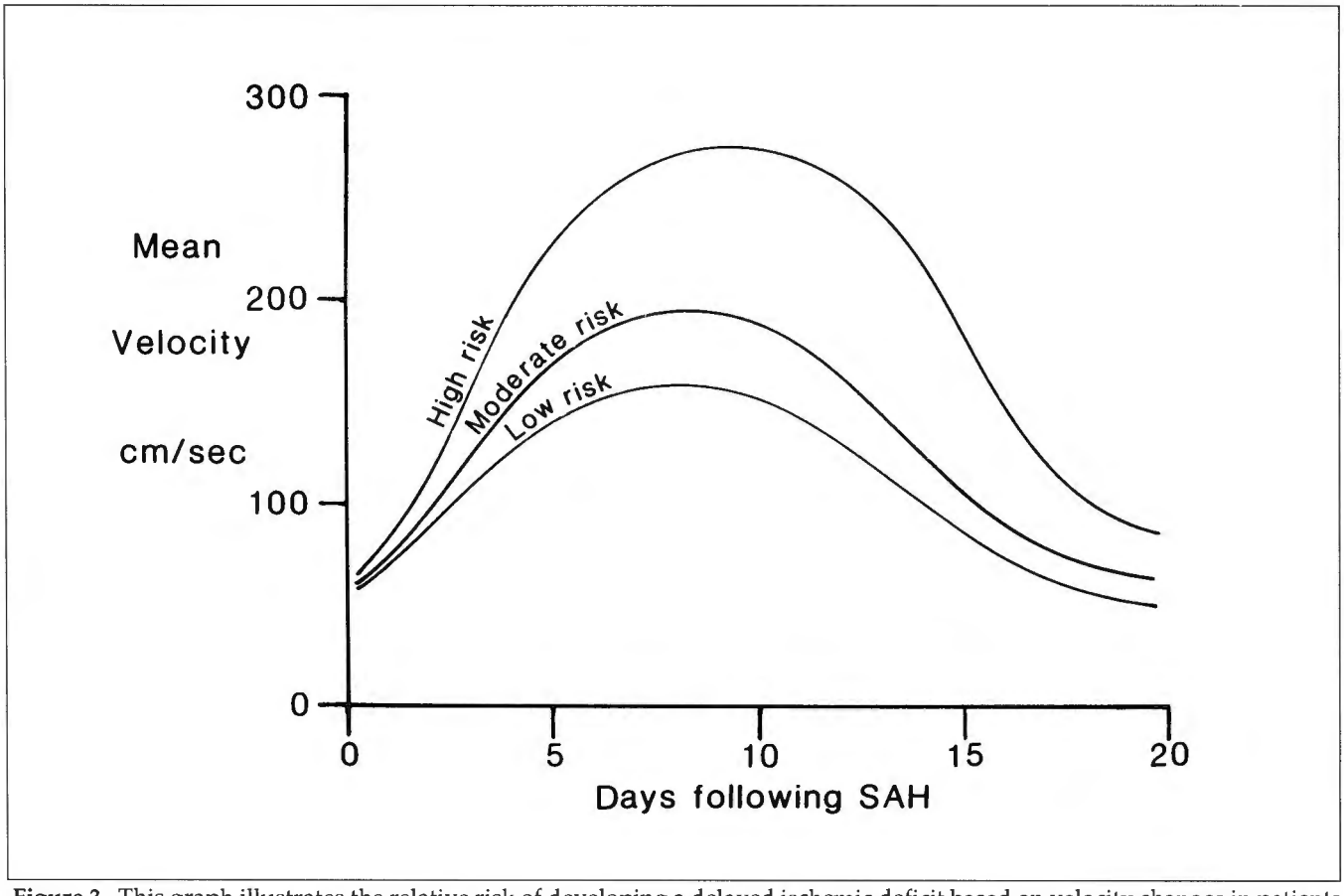

Figure 3. This graph illustrates the relative risk of developing a delayed ischemic deficit based on velocity changes in patients with subarachnoid hemorrhage.

crease in areas of brain supplied by vessels that have high velocities on TCD. The most sensitive and specific way to diagnose critical vasospasm, therefore, may be a combination of intracranial blood flow velocities combined with an index of CBF.

CT Correlations

There have been several reports showing a strong correlation between the amount of blood detected in the basal cisterns on CT scan after SAH and the development of vasospasm detected by angiography. When CT scans obtained within the first several days after SAH are examined for the amount of blood in the basal cisterns, there appears to be a greater degree of vasospasm seen on angiography and a higher incidence of delayed ischemic deficits in patients showing a large amount of blood deposited in the subarachnoid space. It has also been noted using TCD that patients with a greater amount of blood in the basal cisterns on their initial CT scan will develop higher velocities measured by TCD during their hospital course. The combination of a large amount of cisternal blood on CT scan and high velocities recorded by TCD identify patients in a high-risk category for development of ischemic deficits from vasospasm.

Time Course of Cerebral Vasospasm

Examination of angiograms performed on patients after SAH shows that the maximum degree of cerebral vasospasm generally occurs at the end of the first week after the bleeding episode. This also has been found to be the time when patients are at their highest risk for developing ischemic deficits post-SAH. Initial studies using TCD to record daily velocity readings on patients after SAH have revealed that velocities appear to reach their maximum values between days 7 and 12 after the initial bleed. It has been noted in a series of patients undergoing surgery within 72 hours after SAH who were treated with nimodipine that maximum velocities were reached between day 11 and 20. A high complication rate has been associated with surgery performed in patients after SAH, when their serial velocities are increasing rapidly. Operating on patients whose serial velocities are decreasing appears to be less risky.

Interpretation of Results

In order to gain the most information about the patient after SAH, the major vessels of the anterior and posterior circulation should be examined at regular intervals using TCD. For several reasons, it is helpful to obtain baseline readings of the velocities early after SAH before vasospasm occurs. These reasons include the following: 1) During vasospasm the vessels become smaller and therefore more difficult to locate with TCD. It is helpful for the examiner to be familiar with the anatomical configuration of the vessels without vasospasm in any given patient. 2) It is helpful to know the time course of the vessel narrowing. This information can be better utilized by plotting the daily velocity readings of each vessel on a chart or graph. High velocities in smaller branches and musical murmurs also should be noted. 3) Severe vasospasm may cause arteries to become so small that accurate quantitation of the velocity signals cannot be obtained. In this setting, faint high-frequency signals are present in a location where stronger quantitative signals were found previously. This may be an ominous sign and can indicate severe vasospasm.

. . . it appears that the most useful application of transcranial Doppler will be to identify patients at high risk for developing delayed ischemic deficits.

In order to interpret the data gained from TCD recordings in a clinically useful way, one should consider what is known about the pathophysiology of vasospasm and what has been observed using TCD. Although the velocity readings are affected by changes in flow, it appears that TCD can give a fairly accurate assessment of the severity of spasm of the basal vessels after SAH. It is known that severe vasospasm seen with angiography does not always lead to ischemic deficit because of compensatory mechanisms. Yet, delayed infarction due to vasospasm is usually associated with severe vessel narrowing. Studies done on patients after SAH using TCD indicate that the development of high velocities usually preceeds the onset of a delayed ischemic deficit. Therefore, it appears that the most useful application of TCD will be to identify patients at high risk for developing delayed ischemic deficits. This group of patients should be monitored closely and be kept well hydrated and slightly hypertensive if possible. If any clinical deterioration develops, aggressive treatment with hypervolemic hypertension can be started immediately. TCD can also be useful in identifying which patients are at low risk for ischemic deficits and who will, therefore, require less intensive monitoring.

Summary

Recently, TCD has been introduced as a method to obtain velocity measurements from the basal intracranial arteries. It has several applications in the study of disorders of cerebral circulation and appears to be especially useful in the noninvasive diagnosis of cerebral vasospasm after SAH. To date, studies using TCD to evaluate patients after SAH have demonstrated that the velocity readings show a good correlation with angiographic vasospasm and may be valuable in following the development and resolution of this condition. High velocities indicating severe vasospasm have been associated with development of delayed ischemic deficits and infarction and usually precede the onset of symptoms. The most valuable use of TCD appears to be its ability to identify a group of patients at high risk for developing ischemic deficits or cerebral infarction after SAH.

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Answer questions on response card for volume 011, lesson 18

IMPORTANT: MARK ONLY ONE OVAL AND USE NUMBER 2 PENCIL ONLY

Response card must be mailed on or before November 22, 1989

1. The type of ultrasound used for transcranial Doppler is a 10 MHz continuous wave.
True or False?

2. A bidirectional signal found at 60 to 65 mm usually indicates the carotid termination.
True or False?

3. The effect of vessel narrowing on blood flow reduction will depend on velocity/pulse pressure.
True or False?

4. Severe vasospasm seen on angiography usually corresponds to velocity readings from the middle cerebral artery of 3.5-6 times resting values.
True or False?

5. Musical murmurs caused by vibrations of the intracranial arteries are heard best at the basilar tip.
True or False?

6. CBF determinations in combination with TCD recordings may offer added information because they can detect posterior fossa vasospasm.
True or False?

7. Low CBF values during the first few days after SAH is not associated with vasospasm.
True or False?

8. Mean velocities exceeding 200 cm/sec is one of the TCD findings associated with severe vasospasm.
True or False?

9. Angiography is the most precise method of diagnosing vasospasm.
True or False?

10. The anterior cerebral artery is the most suitable for detection of vasospasm using TCD.
True or False?