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Transcranial Doppler In Cerebral Vasospasm

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The confirmation of cerebral vasospasm following subarachnoid hemorrhage, until recently, has only been possible using angiography. The introduction of transcranial Doppler (TCD) has made it possible to assess noninvasively the degree of cerebral vasospasm and also to follow its development and resolution with time.[1, 3, 4, 12, 23] Since the early description of delayed infarction of the brain caused by vasospasm following subarachnoid hemorrhage,[10] the diagnosis of vasospasm as a cause of delayed deterioration has generally been a diagnosis of exclusion. Few effective treatments for vasospasm have existed until recently; therefore, the absolute confirmation of vasospasm was not essential. With the success of induced hypervolemia and hypertension,[6, 19] calcium-channel blockers,[24, 25] and transluminal angioplasty[17] as treatments for vasospasm, accurate diagnosis and continued assessment have become more necessary.

The clinical syndrome of delayed ischemic deficit was initially believed not to correlate well with the severity and extent of vasospasm seen by angiography.[16] As more was learned about the time course and delayed onset of vascular changes, a more careful examination of the phenomenon revealed a closer correlation of angiographic and clinical changes following subarachnoid hemorrhage.[11] With an understanding of this relationship, a noninvasive method for assessing the severity and extent of vessel narrowing becomes of great value.

PRINCIPLES OF TRANSCRANIAL DOPPLER

Satomura[22] first described the use of Doppler ultrasound to measure the velocity of flowing blood in the peripheral arteries in 1959. The Doppler effect describes a shift in frequency of a wave when either the transmitter or the receiver of the wave is moving with respect to the wave-propagating medium. Using this principle, it is possible to use the change in the ultrasonic frequency reflected off moving blood cells to calculate the blood flow velocity.

Doppler ultrasound has been used extensively to measure blood flow velocity in the heart and peripheral and extracranial vessels using both continuous-wave and pulsed Doppler systems. Continuous-wave Doppler transmits ultrasound constantly from one crystal source and receives the reflected ultrasound from another. Pulsed Doppler transmits and receives bursts of ultrasound at regular intervals from the same crystal source, enabling the recording of velocity

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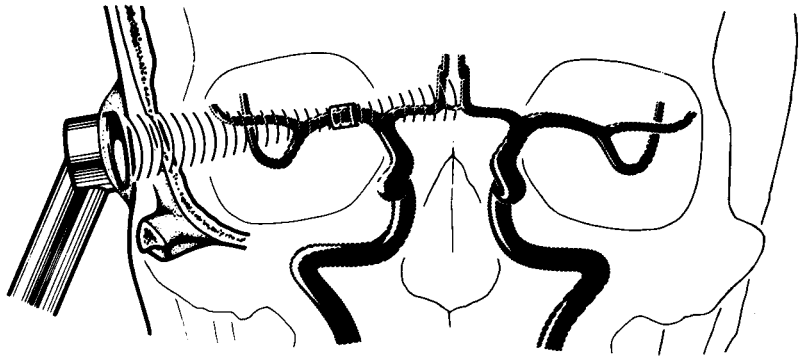

Figure 1. Representation of TCD recording from the middle cerebral artery through the transtemporal route. Cylinder represents the sample volume at a preselected depth. (*From* Aaslid R, Markwalder TM, Nornes H: Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 57:769, 1982; with permission.)

signals at preselected depths. This feature is essential for transcranial use, because it permits the differentiation of the arteries at the base of the brain.

A unique feature of transcranial Doppler is the use of a 2-MHz ultrasonic frequency versus the 3- to 10-MHz frequency used for other applications. The lower frequency allows the ultrasound to penetrate thin portions of the temporal bone to gain access to the intracranial arteries (Fig. 1). The TCD recording device receives the ultrasound wave that is reflected from the moving blood cells and processes the frequency shifts to calculate the velocity in centimeters per second (cm/sec). Increased frequency shifts indicate blood flowing toward the probe, and decreased frequency shifts indicate blood flowing away from the probe. The velocity waveforms throughout each cardiac cycle are then displayed on a spectrum analyzer. The systolic, diastolic, and time-averaged mean velocity can be noted. The mean velocity is usually used for comparative measurements, indicating a change in flow or degree of stenosis.

The recorded dimension, velocity, is not a direct measurement of flow. To calculate absolute flow, one must know the velocity and the cross-sectional area of the artery being recorded from as well as the angle of insonation (angle between the ultrasonic beam and the artery). The cross-sectional area of the vessel and the angle of insonation are not measurable with currently available TCD devices. Although absolute flow is not obtainable by TCD, changes in cerebral blood flow (CBF) are accurately reflected by changes in velocity in each individual when the arterial diameter remains constant. This proportionality was demonstrated by Lindegaard et al,[15] who compared middle cerebral artery (MCA) velocity changes to carotid artery flow changes measured electromagnetically, and also by Bishop et al,[7] who compared MCA velocity changes to CBF changes measured by xenon 133 induced by changes in pCO_2.

Stenosis caused by vasospasm, atherosclerosis, or other lesions will increase the blood flow velocity through the stenotic segment in proportion to the reduction in cross-sectional area (or radius^2) of the artery when the same flow is maintained. For example, if the diameter of a vessel is reduced to half by vasospasm, velocity increases to 400% of its original value. Theoretically, if the stenosis increases to critical levels at which volume flow is reduced significantly (>70% diameter reduction), velocity may actually decrease with further diameter reductions.[20] In practice, with very severe stenosis, the high-velocity signals become progressively weaker and more difficult to quantitate because of the reduction in reflected ultrasound owing to the decreased diameter of the vessel. In this setting, sometimes these high-frequency signals can be heard with the headphones, which should always be used when evaluating patients for vasospasm. Figure 2 illustrates the angiogram and TCD recordings from a patient on the day of subarachnoid hemorrhage and again during vasospasm.

EXAMINATION TECHNIQUES AND STRATEGIES

Three major routes of examination or "windows" exist for recording from the intracranial vessels (Fig. 3). The transtemporal

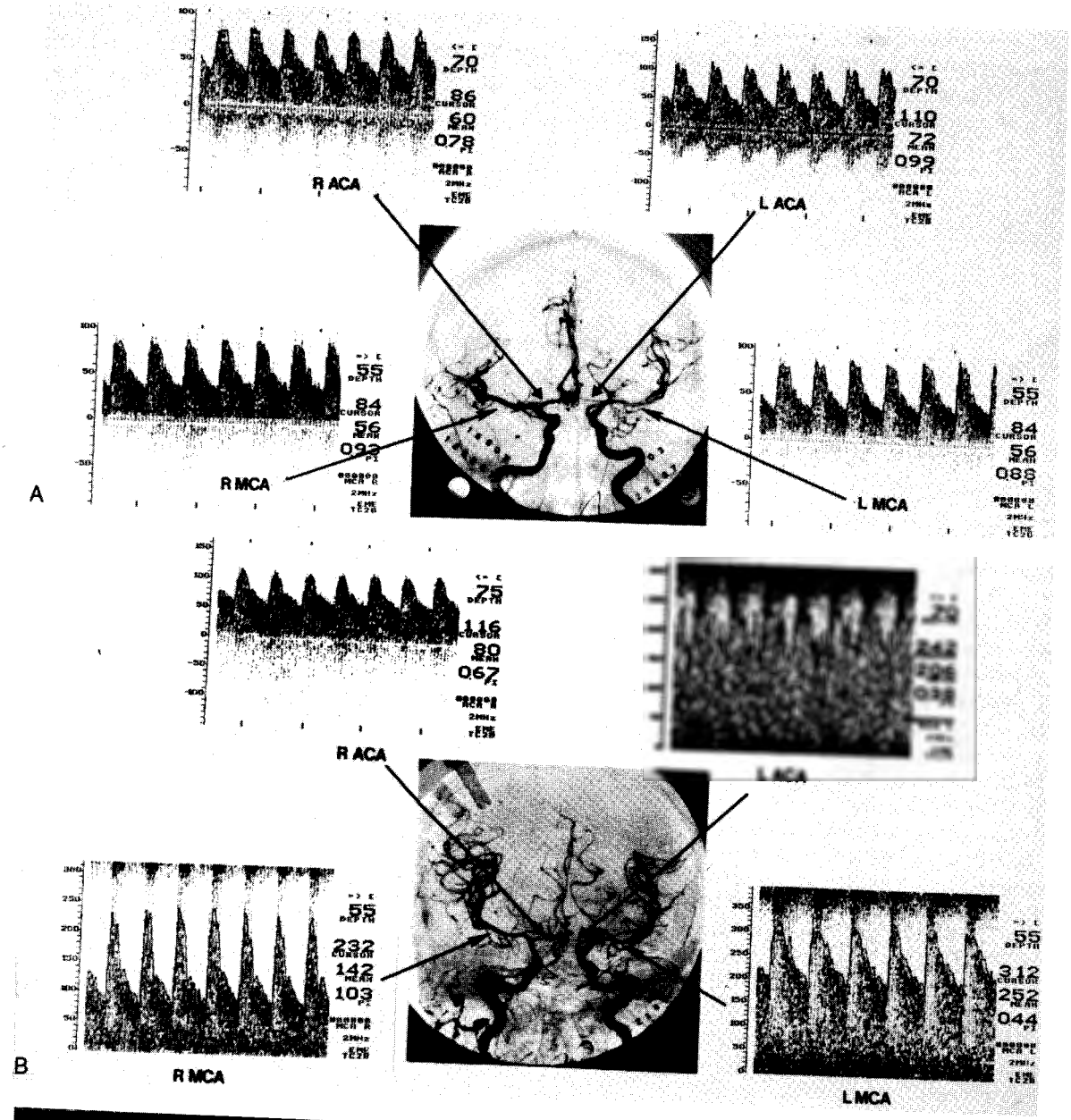

Figure 2. *A*, Angiogram and TCD recordings from a patient with an anterior communicating artery aneurysm on the day of rupture (note atretic left A1), *B*, Angiogram and TCD recording during vasospasm in the same patient reveal increased velocity signals. *C*, Subsequent infarction in the left anterior cerebral artery territory. (*From* Newell DW, Winn HR: Evaluation of vasospasm using transcranial Doppler. Contemp Neurosurg 11:1, 1989; with permission.)

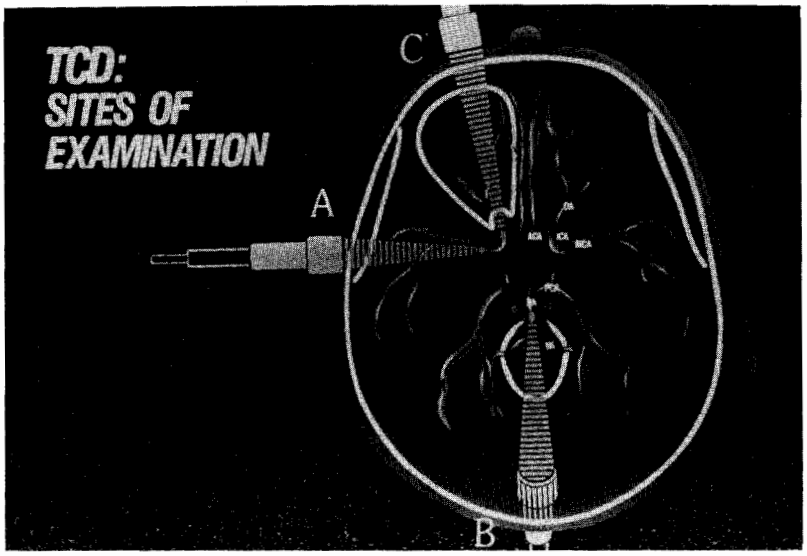

Figure 3. Schematic representation of various windows for transcranial Doppler examination. Through the transtemporal window (*A*), velocity can be determined in the middle cerebral artery (MCA), anterior cerebral artery (ACA), intracranial internal carotid artery (ICA), and posterior cerebral artery (PCA). The transoccipital window (*B*) allows examination of intracranial vertebral arteries (VA) and basilar artery (BA). Via the transorbital window (*C*), the ophthalmic artery and segments of intracavernous and supraclinoid ICA can be insonated. (Courtesy of Medasonics, Inc.)

window allows recordings to be made from the MCA, anterior cerebral artery (ACA), intracranial internal carotid artery (ICA), and proximal posterior cerebral artery (PCA). The transorbital window allows recordings to be made from the ophthalmic artery, the ICA siphon, and the supraclinoid ICA. The transoccipital route allows examination of both vertebral arteries, the basilar artery, and often the posterior inferior cerebellar artery.

The first step in examining the patient is to find the MCA through the transtemporal window. The MCA should then be examined from the upwardly directed insular branches at a depth of approximately 35–40 mm to its origin at the internal carotid termination at 60–65 mm, where a bidirectional signal is found under normal circumstances. Occasionally, a bidirectional signal is found at 50–55 mm, which usually represents a medially directed anterior temporal branch. This can be differentiated from the ACA by its failure to reverse its signal with ipsilateral carotid compression. The normal position for the ACA is at 65–70 mm, or just deep to the carotid termination. From a position on the carotid termination, the PCA is located slightly posterior and the ICA is located slightly caudal, both at 65–70 mm. The posterior circulation should also be examined routinely, although we have encountered only one case of severely symptomatic posterior circulation vasospasm in a patient with a ruptured anterior circulation aneurysm since instituting TCD. To examine the vertebrobasilar system, the patient's neck is flexed, and the probe is placed at the level of the second cervical vertebrae and aimed toward the clivus. The vertebral arteries are located on either side of the midline and can be followed from a depth of about 45 mm to the origin of the basilar artery at approximately 80–85 mm. The basilar artery can then be followed in the midline to a depth of 100–105 mm. Flow in the ophthalmic, intracavernous, and supraclinoid internal carotid arteries can be assessed through the transorbital window. Reverse flow in the ophthalmic artery frequently signals the presence of increased collateral flow to the hemisphere.

The extracranial ICA should also be examined to gain an index of change in flow and to calculate ratios of intracranial to extracranial velocity. The probe, with the depth setting at 45 mm, is placed at the angle of the mandible and directed upward toward the base of the skull. The external carotid artery usually can be distinguished by its high-resistance signal with a lower diastolic velocity.

Examination of a patient for vasospasm following subarachnoid hemorrhage should include baseline vessel measurements and repeat examinations at regular intervals to follow the development and resolution of vasospasm. A good baseline examination in

patients can be obtained during the first 24–48 hours following subarachnoid hemorrhage, when the velocity readings will usually be in the normal range.[21] There are several reasons why good baseline examinations are important following subarachnoid hemorrhage. (1) During vasospasm, the vessels become smaller and more difficult to locate with TCD. It is helpful for the examiner to be familiar with the anatomic configuration of the vessels without vasospasm in any particular patient. (2) Information on the rate of development of vasospasm is useful in determining the risk of developing ischemic deficits. (3) Severe vasospasm can cause arteries to become so narrow that the accurate quantitation of the velocity signals becomes difficult. Faint high-frequency signals that are present in a location where stronger quantifiable signals were previously found usually indicate severe vasospasm even if the mean velocity value is not in the range for severe vasospasm.

When high velocities are detected from the intracranial arteries, the highest velocity along that artery should be noted. Some patients with abnormal breathing patterns display periodic changes in the velocity measurement, which are most likely related to changes in pCO_2. In this situation, it is useful to record the highest value that occurs. In addition to quantifying the highest mean velocities, other indicators of vasospasm should be noted. These include musical murmurs, heard best near the carotid termination,[2] and low-velocity, low-pulsitility signals with a delayed systolic upstroke in distal branches of the MCA.

The sensitivity of the technique in detecting vasospasm can be affected by the experience of the examiner, the adequacy of the cranial windows, and the location of the vasospasm. Previous studies using angiography have stated that vasospasm most commonly involves the basal vessels but can occur in the distal vessels, although the exact distribution has not been well documented. Transcranial Doppler can identify spasm of the basal cranial vessels but normally not of the distal vessels. Newell et al[18] reported the proportion of basal and distal vasospasm in a group of 40 patients with vasospasm from ruptured anterior circulation aneurysms. It was found that 50% of the patients had spasm restricted to the basal vessels, 42.5% had spasm involving basal and distal vessels, and 7.5% had spasm of distal segments. Thus, in this group of patients, vasospasm should theoretically be detectable by TCD in 92.5% of cases.

CORRELATION OF VELOCITY WITH VESSEL NARROWING

As stated previously, velocity recordings can be obtained from all the major basal cerebral arteries; however, the MCAs are the most suited for TCD recordings in patients with vasospasm. Under normal conditions, the MCAs have a limited collateral network and, therefore, there is a close correlation between the amount of vessel narrowing and the increase in velocity demonstrated by TCD. The range of normal blood flow velocity in the MCA is between 30 and 80 cm/sec, with a mean of 62 cm/sec.[1] Middle cerebral arteries that are classified as spastic on angiography demonstrate mean velocities of 120 cm/sec or more. Mean velocities greater than 200 cm/sec correspond to severe spasm seen on angiography and greater than 50% vessel narrowing when sequential angiograms are studied[9] (Fig. 4). Increased velocities also are commonly seen in the terminal ICA and proximal ACA; with profound vasospasm, high-velocity signals from the proximal MCA and distal ICA can often be difficult to distinguish.

Compared with the MCA, correlations between angiographic vasospasm and TCD velocity in the proximal ACA have not been as close, although an inverse relationship between vessel diameter and velocity has been reported.[3, 12] Several possible explanations exist for the poorer correlation: (1) A significant number of ACA A1 segments will be atretic and single-diameter determinations during vasospasm compared to velocity readings may be misleading unless baseline angiograms are obtained to compare. Atretic A1 segments normally do not have high-velocity signals. (2) Owing to the collateral channel through the ACA, the relationship between vessel narrowing and increases in velocity may not be as close if the contralateral ACA is not also in spasm. (3) During

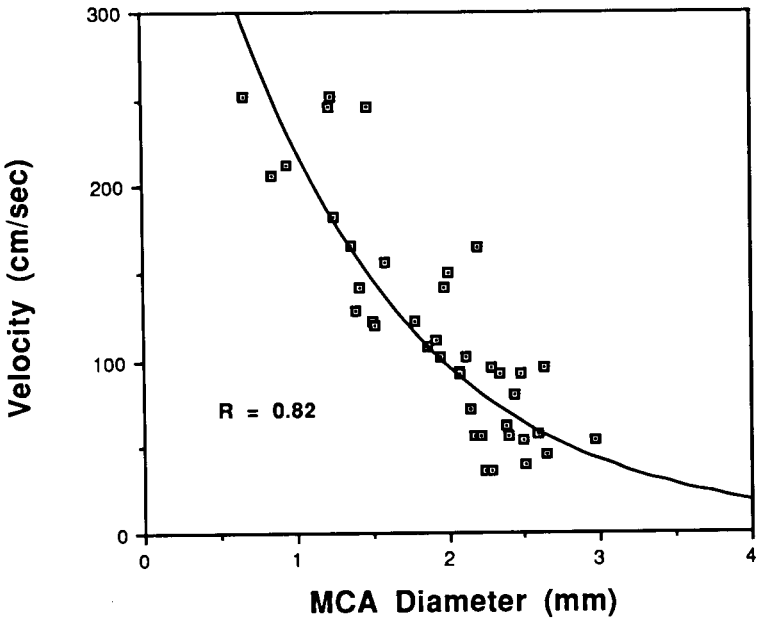

Figure 4. Correlation of MCA velocity recordings with arterial diameter in 10 patients who had two sequential angiograms.

profound vasospasm, the ACAs become very small and reflect less ultrasound, becoming more difficult to locate and more difficult to quantify velocity signals from. Despite these difficulties, TCD is still valuable in detecting proximal ACA vasospasm, although the sensitivity is probably lower than for MCA. There are few published data on the detection of vasospasm in the posterior fossa, but we have observed high velocities in cases of vertebrobasilar vasospasm documented by angiography.

CEREBRAL BLOOD FLOW AND VELOCITY

The maintenance of cerebral function during vasospasm requires the brain to use compensatory mechanisms when CBF is initially reduced by vasospasm. Some of these compensatory mechanisms include increased blood pressure, increased oxygen extraction, autoregulation of the distal vasculature, and recruitment of collateral circulation through leptomeningeal branches. The individual variability of these mechanisms, anatomic variations in the basal vessels, and the combination of vessels involved in vasospasm probably all influence a particular individual's clinical response to vasospasm.

When there is narrowing of any particular vessel, the amount of blood flow reduction through the stenosis will be related to the degree as well as the length of the stenosis. With increasing stenosis, the velocity of blood in that segment will progressively increase until a severe flow reduction occurs. At this point of hemodynamically significant stenosis, velocity should theoretically diminish owing to reduced volume flow.[20] It is unclear how significant this effect is in practical terms in patients with vasospasm. In practice, one usually sees progressive weakening of a high-velocity signal with critical vasospasm rather than reduction of velocity to lower levels.

A more sensitive method for detecting critical vasospasm would include a simultaneous index of flow as well as velocity. Aaslid et al[4] reported a decreased blood flow velocity of the *extracranial* carotid artery recorded from the neck coincident with intracranial velocity readings indicating severe vasospasm in patients following subarachnoid hemorrhage. This effect is believed to be due to a reduction in volume flow secondary to increased vascular resistance caused by the vasospasm. Lindegaard

et al[14] have applied this concept and formulated a velocity ratio by dividing the MCA velocity by the extracranial ICA velocity (Vmca/Vica). This ratio may partially compensate for the effect that CBF changes have on intracranial velocity readings. In addition to flow reductions caused by vasospasm, CBF changes can also occur because of impaired autoregulation after subarachnoid hemorrhage.[8]

Transcranial Doppler recording was combined with CBF measurements performed by stable xenon/CT CBF studies and xenon 133 washout studies by Sekhar et al.[26] Cerebral blood flow values were found to be decreased in vascular territories fed by vessels that had high-velocity readings by TCD. These studies appear to indicate that a method to measure or index CBF combined with TCD recordings may be more useful in diagnosing critical vasospasm than intracranial velocity recordings alone.

TIME COURSE OF VASOSPASM

Knowledge of the time course of the development and resolution of vasospasm in each individual patient is valuable for the physician managing patients with subarachnoid hemorrhage and can be obtained using TCD. Initial information concerning the time of development and resolution of vasospasm in humans was obtained by combining angiographic data from groups of patients. In an early study reporting the incidence of vasospasm at different time intervals, Allcock and Drake[5] reported that spasm was present in 45% of patients less than 3 days after subarachnoid hemorrhage in 41% at 3–10 days, and in 25% after 10 days. A larger, more recent angiographic study by Weir et al[28] reported that vasospasm initially appeared 3 days after subarachnoid hemorrhage was maximal at 6–8 days and minimal by day 12.

Transcranial Doppler has confirmed the delayed onset and subsequent time course of vasospasm initially recognized using angiography, but has the advantage of being able to provide serial assessment on each patient. Several studies have used TCD to examine the temporal course of vasospasm by recording the development and resolution of high-velocity signals in patients following subarachnoid hemorrhage.[12, 23] Seiler et al.[23] reported that TCD velocities rose to maximal levels between 7 and 12 days following subarachnoid hemorrhage in a group of 39 patients, in which the majority had late operations. It was noted that patients who had rapid early (day 5 or sooner) increases in velocity to levels indicating severe spasm were at high risk for developing subsequent cerebral infarction. Harders et al[12] reported that maximal velocities were reached between 11 and 20 days after subarachnoid hemorrhage in a group of 50 patients who were operated on within 72 hours of hemorrhage and were treated with nimodopine. It was also noted in this study that patients who developed high-velocity readings early in their course were at risk for developing delayed ischemic deficits.

When managing patients with subarachnoid hemorrhage, recording the daily changes in velocity during the first week after hemorrhage can help predict which patients are at high risk and which are at low risk for developing delayed ischemic deficits (Fig. 5). This information may be useful in determining how vigorous hypervolemic treatment and intensive care monitoring should be and in assessing the timing for instituting or stopping such therapies.

CORRELATION OF VELOCITY CHANGES WITH DELAYED ISCHEMIC DEFICITS

Although velocity in the basal intracranial arteries can be affected by several physiologic changes, the major cause of increased velocity readings in patients after subarachnoid hemorrhage will be decreased vessel diameter due to vasospasm. Transcranial Doppler, therefore, is a noninvasive method to determine the degree and extent of basal vessel narrowing and thus a reflection of the angiogram. It is well known that the degree of angiographic vasospasm does not always correlate with the clinical condition. For example, some patients can remain asymptomatic with severe vasospasm demonstrated by angiogram, and the incidence of angiographic vasospasm is nearly twice

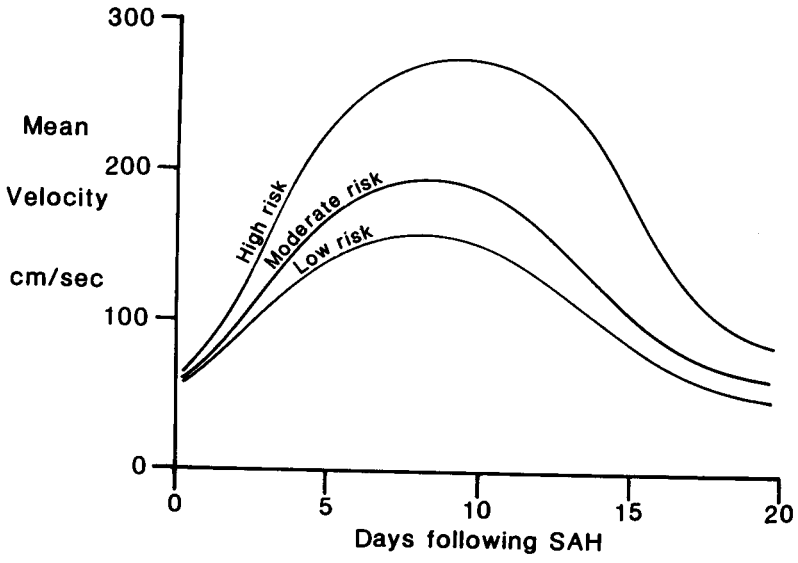

Figure 5. Graphic representation of estimated risk of developing an ischemic deficit based on the timing and magnitude of the velocity changes following subarachnoid hemorrhage. (*From* Newell DW, Winn HR: Evaluation of vasospasm using transcranial Doppler. Contemp Neurosurg 11:1, 1989; with permission.)

that of delayed ischemic deficits.[13] These patients most likely have well-functioning compensatory mechanisms and adequate collateral pathways to provide adequate CBF under these conditions. On the other hand, patients who have a true deterioration due to delayed ischemia, almost without exception, will show severe vessel narrowing by angiography.[11] When using angiography to confirm vasospasm as a cause of clinical deterioration, one must determine whether the severity and location of the vessel narrowing is appropriate to cause the clinical deficit. The same approach must be taken when interpreting the results of TCD recordings in patients following subarachnoid hemorrhage. High-velocity readings identify patients at higher risk for developing ischemic deficits, but they also may occur in asymptomatic patients. In patients who develop true delayed ischemia, careful search using TCD usually reveals the cause.

Seiler et al[23] reported clinical correlations in a group of 39 patients who were not treated with calcium-channel blockers. It was found that when serial recordings were taken, if mean blood flow velocities did not exceed 140 cm/sec, no patient developed a cerebral infarct. Mean blood flow velocities greater than 200 cm/sec were usually associated with delayed ischemia and infarction, although some patients remained asymptomatic. Recent reports indicate lower rates of development of delayed ischemic deficits compared with historical controls using prophylactic hypervolemia[27] and also using calcium-channel blockers.[23] The overall effect of calcium-channel blockers appears to be to lower velocity readings.[24] The overall effect of prophylactic hypervolemic therapy on velocity readings is unclear. Correlation between velocity levels and delayed ischemic deficits in patients prophylactically treated with hypervolemia and calcium-channel blockers is lacking.

SUMMARY

Transcranial Doppler provides a noninvasive method for recording blood flow velocity (and indirectly, diameter) in the basal cerebral arteries and therefore is especially useful in detecting vasospasm following subarachnoid hemorrhage. Vasospasm most commonly involves the basal arteries, where the changes in vessel diameter will be inversely proportional to the mean velocity measurements. Examination of patients requires that the examiner be experienced and familiar with the vascular anatomy and the various TCD indicators of vasospasm. Normal mean velocity for the MCA is 62 ± 12 cm/sec. Significant spasm on angiogram

of the MCA corresponds to a mean velocity of 120 cm/sec. Mean velocities of the MCA of 200 cm/sec or greater indicate severe spasm and correlate with 50% or greater narrowing on angiogram.

Cerebral blood flow changes that can occur after subarachnoid hemorrhage and as a result of vasospasm may affect velocity values. A simultaneous index of CBF with either direct flow measurement techniques or by recording extracranial carotid artery velocity measurements may be helpful in reflecting these changes.

Knowledge of the time course of the development and resolution of vasospasm using TCD can help the clinician predict which patients are at higher and lower risk of developing ischemic deficits, thereby guiding treatment.

Several features of TCD assessment of vasospasm are similar to angiography. High TCD velocities, like severe angiographic vasospasm, are associated with delayed ischemic deficits and infarction, although some patients can remain asymptomatic despite these changes. Delayed ischemic deficits or infarctions in patients following subarachnoid hemorrhage usually will be preceded by markedly elevated velocity or other indicators of severe vasospasm.

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