Evaluation of cerebrovascular spasm with transcranial Doppler ultrasound

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The use of an ultrasonic transcranial Doppler technique for noninvasive evaluation of cerebral vasospasm is described. Middle cerebral arteries (MCA's), classified as spastic on angiography, demonstrated blood-flow velocity between 120 and 230 cm/sec. The flow velocities in these arteries had a clear inverse relationship to the diameter as measured from angiograms in 38 patients with recent subarachnoid hemorrhage. This relationship in the proximal anterior cerebral artery (ACA) was found to be more complicated to assess, due to the collateral channels in the anterior part of the circle of Willis. The authors conclude, however, that the new method of measuring vasospasm will also detect spasm in the ACA if it has a hemodynamically significant effect upon flow resistance.

KEY WORDS ultrasound • blood flow velocity • subarachnoid hemorrhage • cerebral vasospasm • hemodynamics

SPASM of the cerebral arteries is a complication associated with subarachnoid hemorrhage (SAH), as demonstrated by Ecker and Riemenschneider in 1951. Today, angiography continues to be the standard method of assessing cerebral vasospasm. It is, however, an invasive procedure and cannot be repeated at frequent intervals to monitor the development and resolution of arterial narrowing following SAH. As previously reported,1 an ultrasonic Doppler technique was employed to record the flow velocities in the basal cerebral arteries through the intact cranium, and it was suggested then that this approach could be used to assess vasospasm.

The evaluation of spasm is based on the hemodynamic principle that the velocity of blood flow is inversely related to the lumen area.4 10 Arterial narrowing following SAH causes an increase in the velocity of flow through the affected segment, which can be detected by the increased Doppler shift of reflected ultrasound.10 For extracranial arteries, this principle has been used to quantify the degree of arterial stenosis.15 The present study reports flow velocities in the middle (MCA) and proximal anterior cerebral arteries (ACA) in patients with spontaneous SAH. These findings are compared with the diameter of the corresponding arterial lumen in the angiograms.

Clinical Material and Methods

This study was conducted on a consecutive series of 40 patients with recent spontaneous SAH. Twenty-four of these patients had evidence of an aneurysm on angiography. Satisfactory Doppler signals from the MCA were not obtained in two patients and these cases were excluded from the series. Reliable ACA recordings were obtained in 31 patients. The Doppler examination was performed 1 to 6 hours before the angiographic procedure.

Transcranial Doppler Recordings

The technique for transcranial Doppler recording has been described in detail previously.1 The temporal bone is thin enough in most subjects to allow recording with our present laboratory prototype instrument operating on 2 MHz with a focused transducer. The depth of the sampling location can be selected by the operator through range-gating.2 12

A standardized depth scanning technique was used to identify the different cerebral arteries. First, the bifurcation of the terminal segment of the internal carotid artery (ICA) was located at a depth of between 5 and 7 cm. In this region, the Doppler signals from the terminal ICA, the MCA, and the ACA can be observed with minor changes in the sampling location. The instrument and the spectrum analyzer have direction discrimination, and the MCA and proximal ACA normally produce signals on each side of the baseline. By successive reduction of the sampling depth, the signal from the MCA is scanned outward to approximately 3 cm from the probe. By this technique, it is possible to discriminate the MCA from the posterior cerebral artery.

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(PCA), which may (by less experienced investigators) initially be mistaken for the MCA. Simple anatomical considerations make it obvious that the signal from the PCA cannot be scanned further laterally than to depths of approximately 5.0 to 5.5 cm; in contrast, the MCA and its branches can be scanned up to 2.5 to 3.0 cm from the probe. In most patients a scan through the entire anterior circle into the contralateral MCA is possible, ending up at ranges of about 9 to 10 cm from the probe.

The velocities reported in the present study were obtained in proximal portions of the MCA and the ACA. Although we also obtained Doppler signals from the ICA, we will not report on the quantitative data from this artery here. The angle of incidence between the terminal part of the ICA and the ultrasonic beam is in most cases rather blunt and therefore the velocity components determined from Doppler shifts will be significantly lower than the real velocity in this artery. The MCA and the ACA, however, run at comparatively sharp angles to the ultrasonic beam. The velocities in these arteries can thus be determined without significant errors, using the relation: \[ v = 0.039 f \] where \( f \) is the Doppler shift (Hz) and \( v \) the flow velocity (cm/sec). The time-mean of the outline, or envelope velocity was determined by the cursor of the spectrum analyzer. The systolic as well as the diastolic velocities could also be determined by this procedure.

A 2-MHz pulsed Doppler instrument normally only permits recording of velocities up to about 250 cm/sec (6.25 kHz) at a depth of 5.0 cm, limited by the sampling process. By directional spectrum analysis, however, the Doppler shifts above half the pulse repetition frequency are rendered below the baseline, so-called “aliasing,” and thus velocities up to about 500 cm/sec can be measured provided interfering signals from blood flow in the opposite direction can be avoided. This was always possible in this series.

Vessel Diameter

The diameters of the cerebral arteries were measured from the angiograms using the split-image focusing technique described by Huber and Handa. Two crown glass wedges were separated by means of a micrometer adjuster from a line in such a way that the line was shifted an exactly known distance. The distances needed to produce this shift were measured on a scale. A calibration was made in such a manner that the distance of the crown glass wedges from the line to be measured corresponded on the scale exactly to the diameter of the line, when the line was shifted its own diameter to one side. The diameters were determined at the standard positions “A” and “M” corresponding to the sampling locations of the Doppler data presented, and corrected for the magnification, which was 1.16 with a film-focus distance of 120 cm.

Results

Figure 1 left shows the angiographic findings in a 46-year-old woman 7 days after SAH from a right-sided ICA aneurysm. Spasm was clearly present in the MCA and the ACA on the right side. Figure 1 right shows the transcranial Doppler findings in both MCA’s. The ve-
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Velocity on the right side was 150 cm/sec while that on the left side was 58 cm/sec, the latter being within normal range. The velocity in the spastic right ACA was 65 cm/sec as compared to 42 cm/sec on the left side (points marked “1” in Fig. 2 right). The angiograms demonstrated a filling of both pericallosal arteries from the left ICA, with only markedly reduced filling from the right ICA.

The results from the entire series are presented in Fig. 2. The flow velocities are plotted as a function of the respective diameters. For the MCA data (Fig. 2 left), two types of regression analysis were performed. A linear regression resulted in a correlation of \( r = -0.68 \); the best fit line was \( y = 193 - 42x \). Since \( a \ priori \) one would expect an inverse relationship between the velocity and the diameter squared (lumen area), a function of this type was fitted to the data. The resulting equation was \( y = 55 + 167/x^2 \), with a correlation of \( r = 0.75 \). This function is plotted in Fig. 2 left. All patients with spastic MCA’s in the angiograms also showed high flow velocities (greater than 120 cm/sec) in the Doppler recordings.

The flow velocities in the ACA’s correlated poorly with the diameters \( r < 0.3 \) when all the data were pooled. One patient with spasm of both ACA’s demonstrated definitely elevated velocities (points marked “2” in Fig. 2 right), while in another case (marked “3” in Fig. 2 right) a hypoplastic ACA showed a low flow velocity.

Another patient had high-grade spasm of both pericallosal arteries. The ACA flow velocity was moderately elevated bilaterally (90 cm/sec). The segments distal to the anterior communicating artery are beyond the reach of our present equipment. Therefore, this case of high-grade spasm could not be detected by the Doppler method.

Eighteen patients with evidence of aneurysm on angiography were divided into two subgroups according to the temporal relationship of the angiography to SAH. Eight of these patients had angiography during the first 4 days after SAH, and in this group the flow velocity in the MCA was \( 84 \pm 18 \) cm/sec (mean \( \pm \) SD) and the MCA diameter was \( 2.7 \pm 0.3 \) mm. Ten patients had angiography between the 5th and the 12th day after SAH. The flow velocity in the MCA was \( 109 \pm 45 \) cm/sec and the MCA diameter was \( 2.1 \pm 0.6 \) mm. Five of the patients in this second group had clear angiographic vasospasm, whereas no pathological arterial narrowing was seen in the first group. In comparison with a representative series of normal adults with no cerebral vascular disease, the first group had flow velocities increased by 40%. The second group demonstrated velocities 81% above the control series.

Discussion

The use of Doppler techniques to evaluate stenosis in arteries is well documented, and the principle is equally applicable when the narrowing is caused by spasm. The basal cerebral arteries are favorably located for Doppler recordings by transcranial techniques using relatively low ultrasonic frequencies (2 MHz) and focused transducers. A clear correlation between the

![Graph](https://via.placeholder.com/150)

**Fig. 2.** Left: Flow velocity in the middle cerebral arteries (MCA’s) as a function of the diameter of that section of the lumen as measured on angiography. **Triangles:** Cases without angiographic evidence of aneurysm. **Circles:** Cases with aneurysms. **Filled circles:** Cases with aneurysms and clear angiographic evidence of vasospasm. The dotted line: \( y = 55 + 167/x^2 \), was found by nonlinear regression analysis of the entire series. The correlation was \( r = 0.75 \). Right: Flow velocity in the anterior cerebral artery (ACA) as a function of the diameter of that section of the lumen as measured on angiography. Symbols as in left. The numbers refer to cases discussed in the text: “1” signifies the patient shown in Fig. 1, “2” indicates a case of bilateral ACA spasm, and “3” a case with one hypoplastic ACA.
velocity of flow and the diameter of the MCA was found in our patients, suggesting that transcranial Doppler techniques may be used to evaluate vasospasm. This series does not include any cases of high-grade spasm of the MCA’s or ACA’s, the reason being that angiography was postponed for ethical reasons if flow velocities above 200 to 250 cm/sec were observed in the Doppler investigation. Even higher velocities have been found in routine Doppler examination of patients with SAH (material to be published). We have, however, no angiographic correlates of these cases in which there probably was high-grade vasospasm.

The MCA is an end-artery. Normally there are no or very limited collateral sources of supply. Spasm of this artery is therefore a critical condition if the resistance of the narrowed segment increases beyond the compensatory capacity of cerebral autoregulation. In this case, reduction in flow inevitably ensues, with clinical neurological symptoms and, eventually, irreversible effects upon the brain.

Our experience with the Doppler method indicated that velocities of up to about 200 to 250 cm/sec in the MCA are well tolerated in most patients, depending upon the extension of the spasm as well as other factors, such as the arterial and intracranial pressure. Velocities above these values signal a potentially dangerous situation. In one of our patients we witnessed coma and ensuing death after the velocities increased beyond 320 cm/sec. The normal flow velocities in the MCA range from 30 to 80 cm/sec: therefore, there is a factor of about five separating the normal range from critical velocity levels in patients with vasospasm. The Doppler method permits monitoring of patients with SAH throughout this velocity range, thus providing a sensitive parameter for the evaluation of the development and resolution of such spasm.

The proximal ACA is not an end-channel, and an isolated spasm of one of these arteries will not be critical to the distal perfusion if the patient possesses good collateral capacity. This was the case in one of our patients, presented in Fig. 1, and indicated by “1” in Fig. 2 right. The Doppler readings showed a normal flow velocity in this clearly spastic ACA. On the other hand, if both ACA’s are affected by spasm, the situation will be similar to that in the MCA, with reduction in flow if the flow resistance of the spastic arteries increases beyond a critical level.

In one patient with bilateral ACA spasm, represented by points marked “2” in Fig. 2 right, Doppler recordings revealed high velocities through the affected segments. It follows from these considerations that the Doppler method does not disclose arterial narrowing of the ACA in all cases, but only when the spasm is causing increased flow velocities. This is, of course, also clinically relevant information, because the hemodynamic effect of vasospasm is more interesting than the arterial narrowing per se.

An extreme example of a narrow (probably hypoplastic) ACA is indicated by “3” in Fig. 2 right. The velocity in the narrow vessel was low, indicating only a minimal volume of flow, with the main supply to both pericallosal arteries coming from the contralateral side. These findings are in accordance with our intraoperative electromagnetic flowmeter recordings demonstrating much higher variability of the flow in the proximal ACA’s than in the MCA’s. It also explains the lack of correlation between diameter and flow velocity in the total series, which includes a mixture of normal ACA’s, spastic ACA’s with collateral supply, hypoplastic ACA’s, and bilateral spastic ACA’s.

In patients with combined spasm of the ICA and the MCA it was sometimes difficult to discriminate between the signals from the two arteries. The sampling volume of the present Doppler instrument is about 4 mm in diameter and 10 mm in length, and the jet from a spasm of the ICA may also intrude into the proximal MCA. In the combined signal the spectral outline, or envelope, corresponds to the highest velocity components, regardless of whether these originate in the MCA or in the terminal ICA. With improvements in the Doppler techniques we believe it may be possible to discriminate more accurately between adjacent arterial segments. The method would also give a better picture of the extent of spasm.

We have observed increased velocities in two other groups of patients with cerebrovascular disease (unpublished data). In cases of high-grade unilateral carotid stenosis or occlusion, we sometimes found high collateral flow velocities in the proximal ACA’s. The MCA velocities in such patients were often reduced and showed damped waveform on the affected side. In patients with arteriovenous malformations the feeder arteries showed high flow velocities, and we also observed increased flow velocities in the draining veins. For both of these groups we found that the Doppler-recorded velocities remained constant on a day-to-day or week-to-week basis. These findings are in contrast to the data from SAH patients presented here, which showed marked time-dependent changes in blood velocity during their hospitalization.

References

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Manuscript received June 8, 1983.
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