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Distribution of Angiographic Vasospasm after Subarachnoid Hemorrhage: Implications for Diagnosis by Transcranial Doppler Ultrasonography

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A study was undertaken to determine how frequently angiographic vasospasm occurs outside the normal access range of transcranial Doppler ultrasound in patients who have suffered a subarachnoid hemorrhage. Vasospasm located in the basal vessels is readily identifiable using transcranial Doppler ultrasound whereas spasm affecting the more distal, vertically oriented arteries is outside the standard detection range. It is therefore speculated that the sensitivity of the technique would be adversely affected by a high incidence of distal vasospasm. A total of 136 angiograms performed on 68 patients after a subarachnoid hemorrhage from anterior circulation aneurysms were reviewed to determine the typical distribution of vasospasm. Of the 40 cases that showed ≥25% vessel narrowing, 50.0% had spasm restricted to the basal vessels, 42.5% had spasm involving both basal and distal segments, and 7.5% had spasm of the distal segments only. None of the patients with distal vasospasm alone developed delayed ischemic deficits. It is concluded that most patients with anterior circulation aneurysms who develop vasospasm will have involvement of the basal vessels, but a small number of patients may develop vasospasm only in distal vessels. (*Neurosurgery* 27:574–577, 1990)

Key words: Cerebral aneurysm, Cerebral angiography, Subarachnoid hemorrhage, Transcranial Doppler sonography, Vasospasm

Previous angiographic studies have noted that the most common vessels to be affected by vasospasm after a subarachnoid hemorrhage (SAH) are the basal cerebral vessels (5–8, 12–15, 17, 18). Vasospasm has also been observed in the more distal vessels (5, 12, 14), but the relative proportion of basal and distal spasm has not been well defined. Transcranial Doppler (TCD) ultrasound was introduced in 1982 as a noninvasive method to measure blood flow velocities from the basal cerebral arteries and has been useful in detecting vasospasm (1–3, 10, 16). Blood flow velocities can be detected by TCD easily from the basal cerebral arteries, but, because of technical limitations, they cannot be detected reliably from the distal anterior and middle cerebral branches as they become vertically oriented. Moreover, examination of the posteriorly directed portion of the posterior cerebral artery is not routine. Consequently, the present study was designed to determine the extent of vasospasm occurring in the distal cerebral vessels outside the theoretical and practical access range of TCD. To study the distribution of vasospasm, cerebral angiograms were reviewed in 68 patients with SAH, in whom angiography was routinely used to check postoperative clip placement and also to diagnose vasospasm in patients who developed delayed ischemic deficits after SAH.

PATIENTS AND METHODS

A total of 136 angiograms performed on 68 patients were chosen for review from a group of patients admitted for SAH to the Harborview Medical Center, Seattle, Washington, between May 1984 and July 1988. All patients had SAH confirmed by computed tomography (CT) or lumbar puncture and the presence of aneurysm confirmed by angiography. Only patients who had anterior circulation aneurysms and were operated on within 72 hours of the hemorrhage were chosen for review. Cases were selected for review only if they had two angiograms performed, one at the time of admission and a subsequent angiogram either to confirm clip placement or to check for vasospasm. This was done because distal vasospasm cannot be accurately assessed without a baseline angiogram. The second angiograms were bilateral carotid injections in 30 patients and bilateral carotid with vertebral injections in 38 patients. There were 24 men and 44 women. The mean age was 49 years with a range of 16 to 75 years. Table 1 indicates the aneurysm location and the admission grade according to Hunt and Hess (11).

Vessels were classified as either basal or distal, dependent on the ability to be located by TCD. Basal vessels that are easily located by TCD are the following: the intracranial internal carotid artery (ICA); the middle cerebral artery (MCA) trunk and horizontal branches; the anterior cerebral artery (ACA) proximal to the anterior communicating artery; the posterior cerebral artery (PCA) proximal segment before the posteriorly directed branches; the vertebral artery; and the basilar artery. Vessels were defined as distal if they were normally not accessible for recording by TCD because of their orientation and position. These included the ACA distal to the anterior communicating artery, the MCA vertical branches in the insular region, and the posteriorly directed branches of the PCA. Figure 1 illustrates the distinction between basal and distal vessels.

The angiograms were then inspected for vasospasm. Any segments of arterial narrowing on the second angiogram were measured with digital calipers and compared with the same vessel locations on the initial angiogram. The films were corrected for magnification differences by measuring inner skull diameters or petrous ICA diameters on the first and second films and multiplying the ratio by the second value. Significant spasm was defined as a decrease of 25% or more in the diameter of a vessel between the first and second angiogram. The mean interval between the time of SAH and the second angiogram was 4.6 days, with a range of 1 to 11 days in the entire group. The patients who had vessel narrowing of 25% or more had a mean interval between SAH and the second angiogram of 6.1 days with a range of 2 to 11 days.

The resulting measurements were classified into four groups

based on the distribution of vasospasm found on the angiograms. Group 1 included patients who had less than 25% narrowing or no detectable narrowing between the first and second angiogram; Group 2 included patients who had significant spasm of the basal vessels only; Group 3 included patients who had significant spasm of basal and distal vessels; and Group 4 included patients who had significant spasm of distal vessels only without accompanying spasm of the basal vessels.

RESULTS

Inspection of the angiograms revealed that several different patterns of vasospasm could be seen. In some patients, the narrowing was very focal, either in the basal or distal vessels, and in others, focal spasm was found in both locations, skipping areas of the vessel in between. Other patients exhibited a more diffuse pattern that began in the proximal vessels and continued distally. Figure 2 illustrates several different patterns of vasospasm that were observed.

The distribution of angiographic vasospasm was analyzed for the entire population and also analyzed excluding those patients in Group 1 with less than 25% vessel narrowing. The results are illustrated in Table 2. Of the 40 patients who had significant angiographic vasospasm, 37 (92.5%) had involvement of the basal vessels, and only 3 patients (7.5%) had distal vessel spasm exclusively. The charts of these three patients were reviewed, and there was no clinical evidence of delayed neurological deterioration.

DISCUSSION

TCD has been advocated as a useful noninvasive technique for detecting vasospasm after SAH. This technique can detect increasing velocities that occur in the basal vessels when daily recordings are obtained in the post-SAH period. Velocities usually begin to rise to high levels before the onset of neurological deterioration in patients who become symptomatic from vasospasm. TCD can therefore be of value by identifying patients who are at high risk for developing delayed ischemic deficits. There are, however, several potential sources of error when using this method to detect vasospasm. For example, because of bone thickness, it may be technically difficult to measure blood flow velocities or identify specific vessels with TCD. Initial studies, however, have indicated a high success rate that increases with the experience of the examiner. For example, Gomez et al. (8) reported a 9% failure to record signals through the temporal window bilaterally in a group of 330 TCD studies. Another source of error is related to the very faint high-frequency signal emitted by vessels with profound vasospasm caused by decreased vessel diameter, thus making detection and accurate quantitation of velocities difficult. This finding usually indicates severe vasospasm and should be noted by the examiner. Lastly, vasospasm occurring in the distal rather than the basal vessels, and thus not detectable by TCD, would be another potential source of error in patients with delayed ischemic deficits.

TABLE 1
Aneurysm Location and Clinical Grade

Artery	Grade[a]					
	I	II	III	IV	V	Total
Anterior communicating	0	13	5	10	0	28
Internal carotid	1	7	4	6	3	21
Middle cerebral	1	7	2	5	3	18
Distal anterior cerebral	0	0	0	1	0	1
Total	2	27	11	22	6	68

[a] According to Hunt and Hess (11).

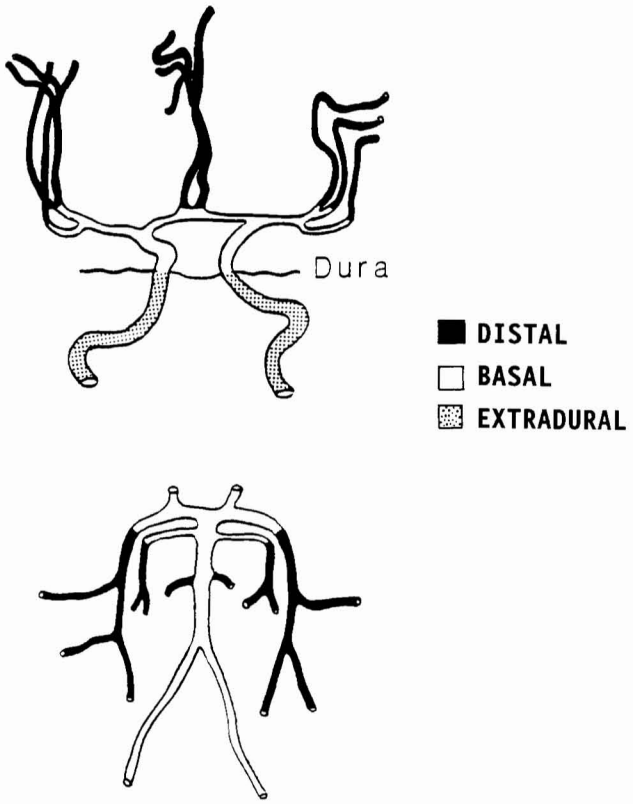

FIG. 1. Illustration of the vessels classified as basal and distal in the MCA and ACA distribution. Basal vessels are horizontal and include the intracranial ICA, proximal MCA, and proximal ACA. Distal vessels are vertical and include the MCA branches as they become vertically or posteriorly oriented and the ACA distal to the anterior communicating artery.

The present study was thus undertaken to determine the incidence of vasospasm outside the access range of TCD. The results indicate that 7.5% of patients who were found to have vasospasm on arteriography had vessel narrowing only in a distribution outside the TCD access range. Moreover, none of these patients with pure distal vasospasm had clinical signs of delayed ischemic deficit. In contrast, most patients with angiographic evidence of vasospasm had vessel narrowing of either the basal vessels alone (50%) or the distal and basal vessels together (42.5%).

Although this study suggests that a large percentage of patients with vasospasm can be detected by TCD, because they develop a pattern of vasospasm that includes the basal vessels, we cannot extrapolate our results to patients with more distally placed aneurysms. Our series did include one patient with an aneurysm of the pericallosal artery who developed vasospasms of the distal as well as the basal vessels.

Previous angiographic studies of vasospasm have generally stated that vasospasm is most common in the basal vessels, but can in some instances be noted in the distal vessels. Several studies have noted that accurate quantitative assessment of distal vasospasm is impossible without baseline angiograms for comparison. In a study that used baseline angiograms for comparison, Bergvall et al. (4) noted that 44% of the angiograms that showed central spasm actually showed peripheral artery dilatation. This is in keeping with positron emission tomography data, which show an increase in cerebral blood volume with significant vasospasm (9).

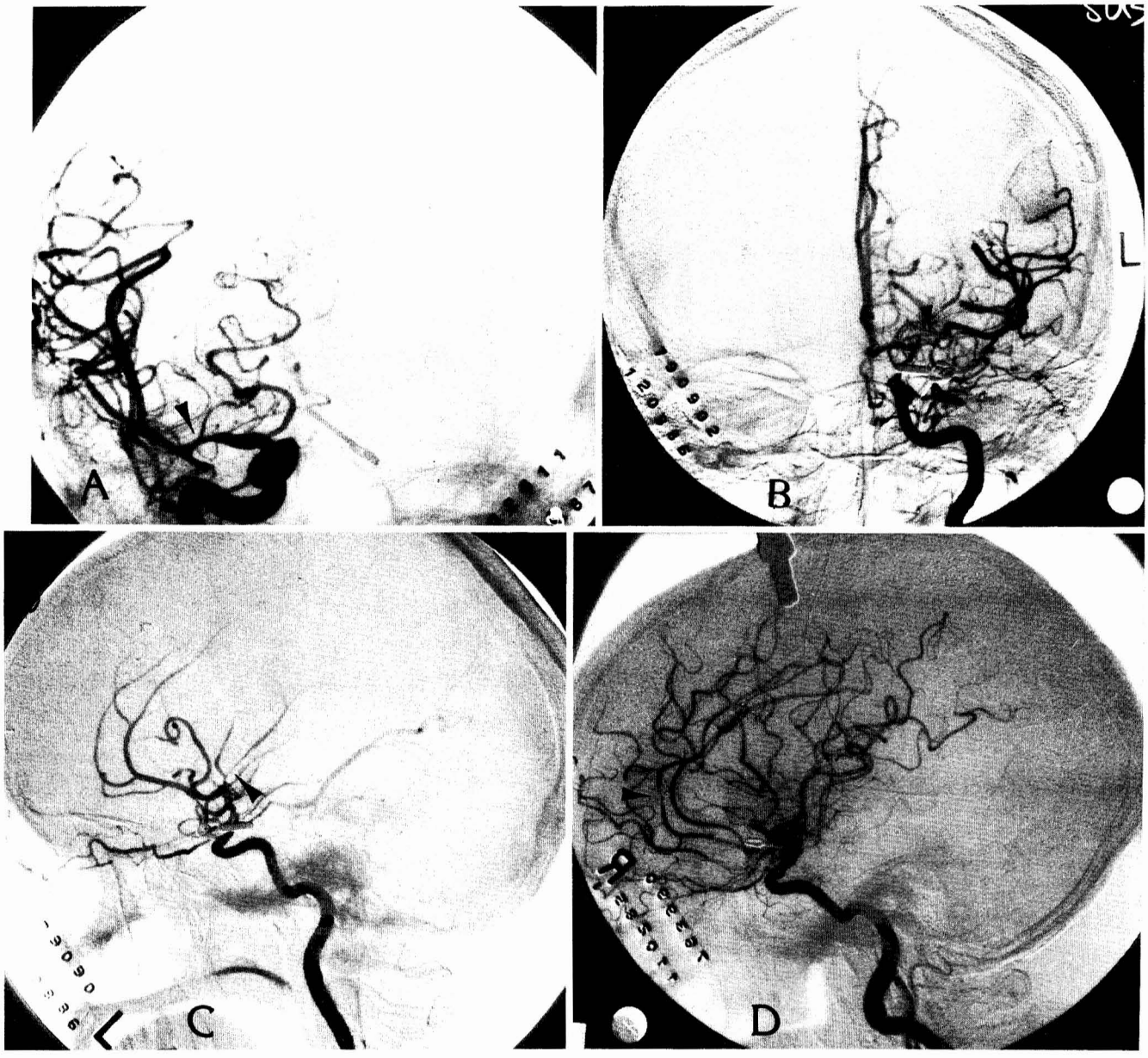

FIG. 2. Illustration of several different patterns of vasospasm that were observed. *Arrowheads* indicate areas of spasm. *A*, focal basal spasm. *B* and *C*, angiogram on another patient illustrating basal artery spasm (*B*) and distal artery spasm (*C*) of the middle artery. *D*, distal spasm only in a patient after clipping of an aneurysm of the anterior communicating artery.

TABLE 2
Location and Distribution of Angiographic Vasospasm

Group	Location and Severity	Number of Patients	Percentage of Entire Group	Percentage with Significant Spasm
1	None or mild (<25%)	28	41.2	
2	Basal vessels only	20	29.4	50.0
3	Basal and distal vessels	17	25.0	42.5
4	Distal vessels only	3	4.4	7.5

We conclude that measuring the velocities in the basal vessels using TCD can provide valuable information about the presence and severity of vasospasm. We have defined a potential small (7.5%) false-negative rate for patients who develop vasospasm only in vessels outside the access range of TCD.

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COMMENTS

The study and review by Newell and co-workers affirm, as others have, that transcranial Doppler (TCD) ultrasound should be most useful for the evaluation of cerebral vasospasm, since both the disorder and TCD methods are slanted toward the larger basal vessels. Because these arteries are rarely over 30° from a perpendicular position as seen through the temporal window, only about 15% error is to be expected from a positional standpoint.

There are many other sources of error, however, and only careful serial examinations provide the best total picture. Velocity rises when the vessel diameter at the insonated point decreases. It may fall, however, when the artery closes more proximally. Likewise, velocity may rise with hyperemic infarction, when there is loss of autoregulation, or when intravascular volume is expanded. These authors point out that any velocity change not detectable with TCD is of less clinical importance, because it occurs distally as the vessels turn cephalad.

Unlike the angiogram, TCD examinations can be carried out repeatedly with safety, and we now evaluate velocity to help decide when to operate on the candidate who will be a good risk. During the first 10 days after a subarachnoid hemorrhage, velocities of middle cerebral arteries greater than 120 to 130 cm/sec carry definitely greater risks for a poor outcome. Later, these same velocities may be better tolerated.

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The authors present a careful analysis of the distribution of angiographic vasospasm in 136 angiograms performed on 68 consecutive patients with subarachnoid hemorrhage. Only cases with anterior circulation aneurysms that were operated on within 72 hours of hemorrhage were included in this study. In this well-characterized population, 42% of the patients were found by delayed angiography to have less than 25% narrowing of one or more vessels. Only 3 patients (4.4% of the population and 7.5% of the cohort with angiographic spasm) had spasm in distal segments *only*, without spasm in more proximal vessels. None of these patients had clinical vasospasm (delayed ischemic neurological deficit).

As the authors point out, the findings are clearly relevant to the theoretical usefulness of transcranial Doppler (TCD) ultrasound as a tool for the detection and follow-up of cerebral vessel narrowing after an aneurysmal subarachnoid hemorrhage. Based on these findings, TCD should not theoretically "miss" a large fraction of patients with vasospasm, and perhaps would not "miss" any patient with clinically significant vasospasm.

The reader must be cautious not to interpret this information in terms of the sensitivity and specificity of TCD. No TCD data are presented, and no practical or direct conclusions can be reached from this study regarding "false-positive or false-negative" rates of TCD in vasospasm. Any reference to the ability of TCD to detect vasospasm in this population is speculative. As the authors carefully point out, the ability of the examiner to detect signals from all basal vessels, the quality of the signals, the reproducibility of the measurements, and other unknown factors are likely to impact on the actual sensitivity and specificity of the technique. It is clear, however, that improved equipment and increased operator experience will minimize these technical factors and push the practical applicability of TCD toward the theoretical limits defined in this study.

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