The Journal of Vascular Technology 14(3):111–115, 1990

Detection of Vasospasm following Subarachnoid Hemorrhage Using Transcranial Doppler

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ABSTRACT With the introduction of transcranial Doppler (TCD) ultrasonography, the diagnosis of vasospasm after subarachnoid hemorrhage from ruptured cerebral aneurysms can be made noninvasively. By recording daily velocities from the basal intracranial arteries, the time course and severity of vasospasm can be evaluated. This information can be used to guide therapy for vasospasm.

Recordings have been obtained over a 3-year period on 67 patients following subarachnoid hemorrhage. Correlation ($R = 0.82$) of middle cerebral artery vessel diameter seen on angiogram and TCD velocities was performed on 20 patients. We conclude that TCD is a useful technique in the diagnosis of vasospasm following subarachnoid hemorrhage.

Introduction

The most common cause of spontaneous subarachnoid hemorrhage (SAH) is rupture of a cerebral berry aneurysm. The incidence of aneurysmal SAH is approximately 10 per 100,000 population per year in the US.[1] The usual treatment of this condition includes intracranial surgery to clip the aneurysm in order to prevent rebleeding. However, a frequent sequela of SAH is the development of cerebral vasospasm.

Spasm of the intracranial vessels following subarachnoid hemorrhage was first observed using angiography by Ecker and Riemenschneider in 1951.[2] Subsequently, vasospasm has become recognized as a major cause of stroke and death during the post-hemorrhage period.[3–5] Clinical manifestations of vasospasm include aphasia, hemiparesis, mutism, and changes in consciousness that can be transient or long-lasting, depending on the severity of vasospasm. These deficits most commonly occur after a delay period, usually 5–10 days after the hemorrhage.

The pathophysiology of vasospasm is not completely understood, but is probably related to the deposition of blood around the outside of the basal intracranial arteries, where it causes a delayed narrowing. The condition has been reproduced in experimental animals, and it appears that substsances released from the blood as it breaks down injure the arteries and cause them to go into spasm.[6] This spasm acts like a stenosis hemodynamically and can impair the blood flow to critical parts of the brain.

Many treatments for vasospasm have been tried in the past without significant success.[7] Recently, three methods of treatment have shown encouraging results. The first is hypervolemic hypertensive therapy.[8] This treatment involves giving patients large volumes of intravenous fluids as well as increasing their blood pressure with medications to improve the cerebral circulation. The second treatment, which has recently gained favor, is the use of calcium channel blockers. The rationale for their use was that these agents could block the calcium-dependent smooth muscle contraction in the arterial wall. There have been some encouraging results,[9] but the mechanism of action is as yet uncertain. The third treatment, percutaneous balloon angioplasty, involves placing a small catheter inside the artery and dilating it in a manner similar to peripheral vessel angioplasty. Preliminary reports indicate that this treatment may be of great benefit in cases that are refractory to other treatments.[10,11]

In the past, neurosurgeons and neurologists have usually made the diagnosis of vasospasm in patients with SAH on the basis of clinical findings. A marked deterioration in neurological function between 5 and 10 days following hemorrhage that could not be attributed to other causes was usually ascribed to vasospasm. Angiography can be used to confirm the diagnosis, but many physicians in the past were reluctant to perform an angiogram in this setting.

For the first time, with the introduction of TCD, the diagnosis of vasospasm can be confirmed noninvasively.[12–14] TCD thus offers a technique to identify patients who are in danger of developing deficits

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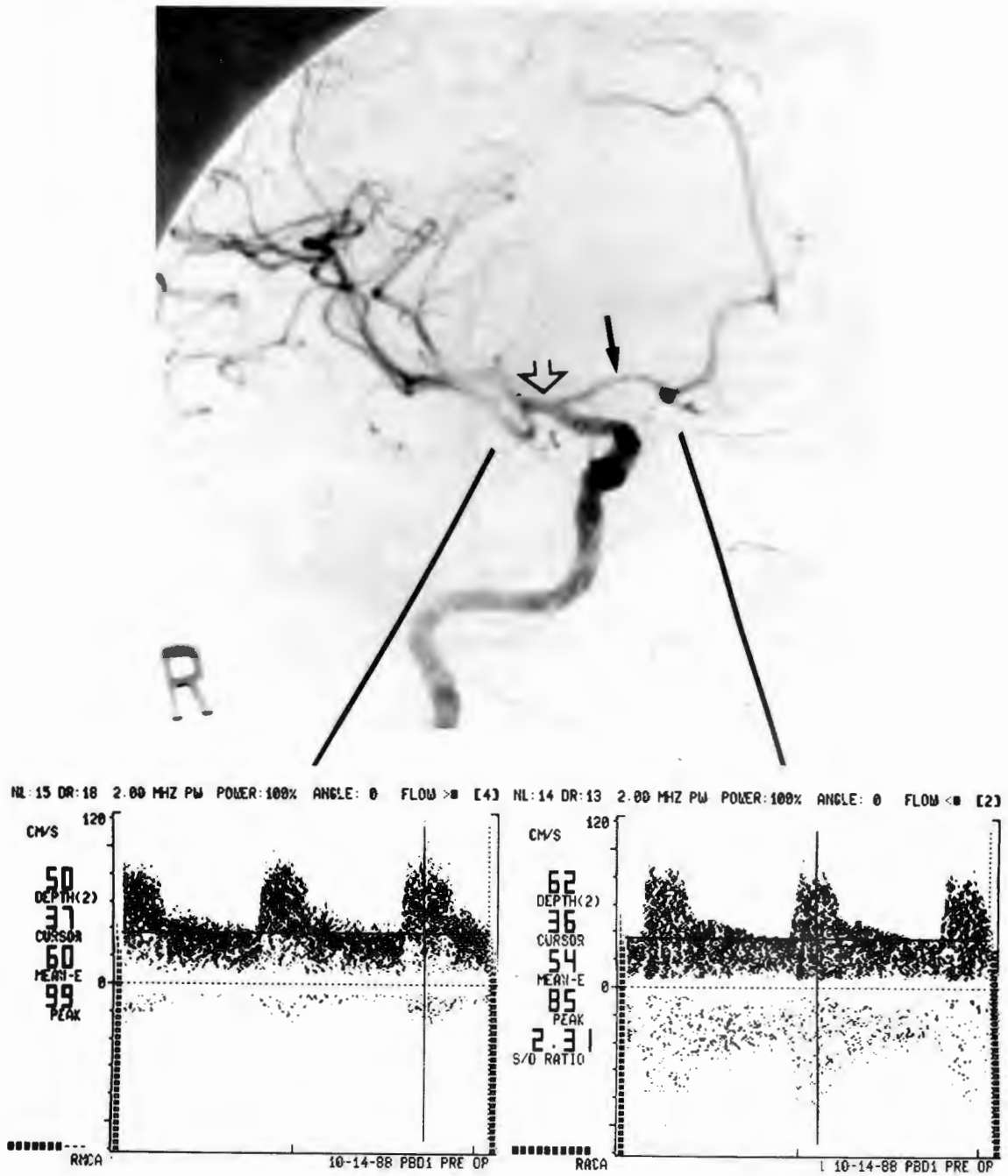

Figure 1A

Initial angiogram and TCD tracing on a patient on the day of subarachnoid hemorrhage, showing normal arterial caliber and normal TCD velocities.

from vasospasm as well as the potential to monitor therapy.

Materials and Methods

During a 3-year period, 67 patients sustaining SAH were evaluated using TCD. Equipment included a TC2-64B† and a Transpect.‡ Both units utilize a 2-MHz frequency and frequency spectrum analyzer.

† Eden Medical Electronics, Überlingen, West Germany.
‡ Medasonics Corp., Mountain View, CA.

Patients all had SAH confirmed by either CT scan or lumbar puncture, and the presence of an aneurysm confirmed by angiography. Most patients were operated on within 72 hr of their hemorrhage. Angiography was repeated following surgery in all cases to check for clip placement, and in some cases was repeated subsequently to confirm vasospasm or evaluate patients for angioplasty.

On admission, TCD recordings of all the basal arteries were obtained routinely. Readings were then obtained at regular intervals, usually daily, until 2

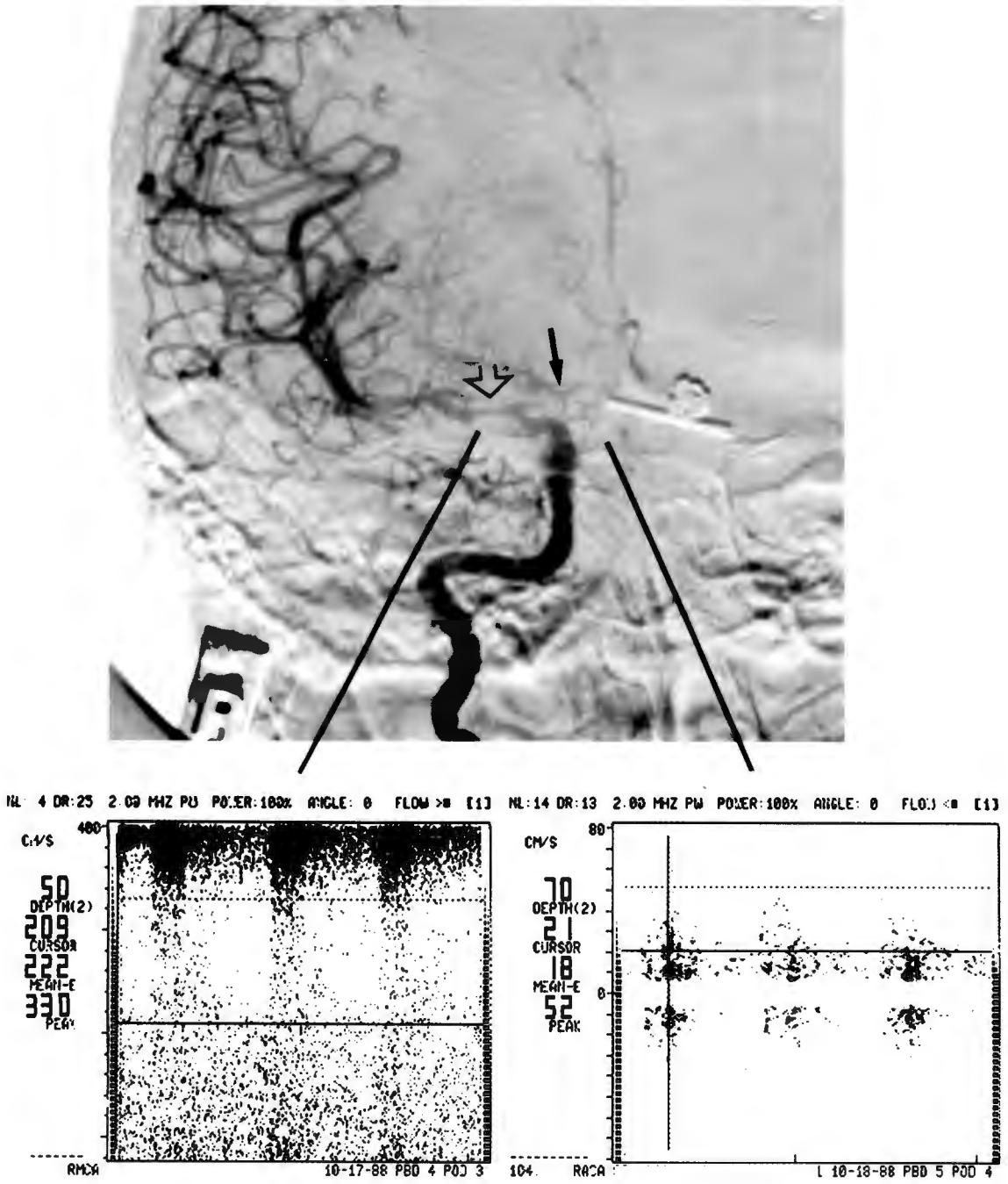

Figure 1B

Subsequent angiogram on the same patient showing severe spasm of the MCA trunk and ACA with corresponding TCD tracing (open arrow, MCA trunk; closed arrow, ACA). MCA mean velocity = 222 cm/sec. ACA shows weak signal; mean velocity = 18 cm/sec.

weeks after hemorrhage, to assess the state of the vessels. Ten patients had initial and delayed angiograms, where comparative measurements could be made, as well as TCD recordings within 24 hr of their angiograms. Measurements of the diameter of the MCA's were made from the arteriogram with calipers and compared with TCD readings. Angiograms were obtained using a standard transfemoral technique with injection of Conray 60. Biplane films were obtained and magnified an average of 2×.

Transcranial Doppler examinations were performed on all the basal intracranial arteries, when possible, on all patients via the standard transtemporal, transorbital, and transoccipital routes. The transtemporal route was used to record from the middle cerebral artery (MCA), internal carotid artery (ICA), anterior cerebral artery (ACA), and posterior cerebral artery. The transorbital route was used to record from the ophthalmic artery and the internal carotid artery siphon. The transoccipital route was used to record from both vertebral arteries and the basilar artery. When high velocities were encoun-

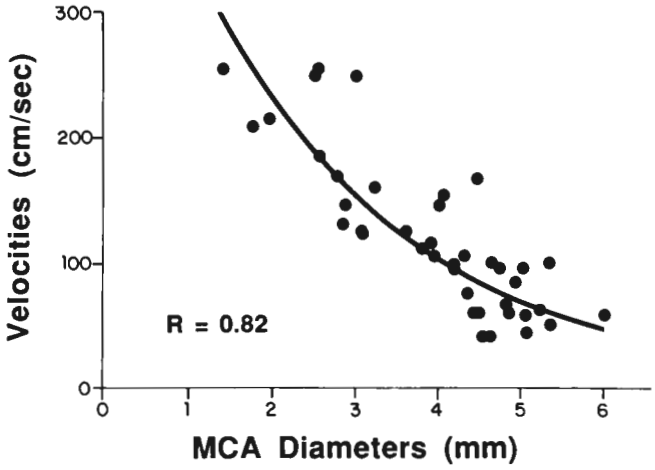

Figure 2

Graph illustrating the relationship between MCA diameters in millimeters and MCA velocities in centimeters/second. Values, uncorrected for magnification, were obtained from 10 patients who each had two angiograms: one baseline and one during vasospasm.

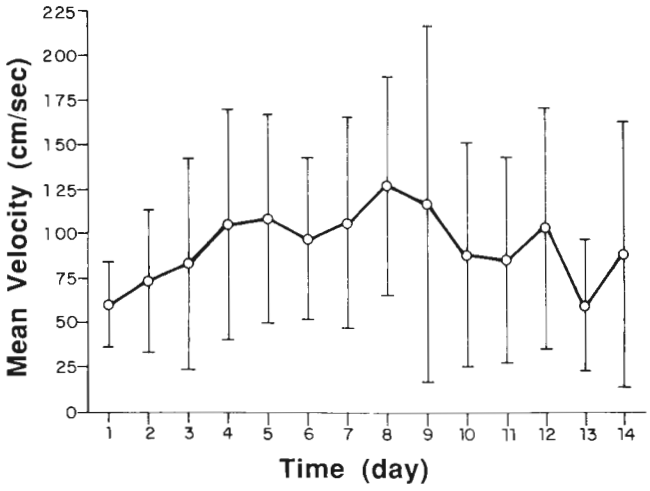

Figure 3

Time course of the MCA velocity changes in the period following subarachnoid hemorrhage in a subgroup of patients.

tered, indicating vasospasm, the highest velocity in each artery was noted and recorded.

Results

The most common locations for vasospasm as assessed by both angiogram and TCD were the intracranial ICA, proximal MCA, and proximal ACA. Measurements obtained from both sides on 10 patients on the MCA's showed good correlations between the degree of narrowing and increases in mean velocity. When high mean velocities were found in the ACA, these also correlated with vasospasm, but on some occasions mean velocities lower than baseline were seen. In certain patients with severe anterior cerebral vasospasm, serial recordings showed initial rises in velocity progressing to very weak high-frequency signals. It is likely that the true mean velocity of these signals was not registered accurately. Where angiographic comparisons were available, this finding correlated with severe vasospasm (see Figure 1).

Mean velocities up to 120 cm/sec correlated with a mild degree of narrowing by angiography, usually less than 25%. Mean velocities of 120–200 cm/sec correlated with moderate narrowing, and mean velocities of greater than 200 cm/sec indicated vessel narrowing of 50% or greater (see Figure 2).

Initial baseline recordings in a group of our patients revealed that velocities in all vessels were less than 100 cm/sec within the first 48 hr following SAH. Velocities usually began to rise on the third or fourth day following SAH, in most cases reaching a peak within 7–12 days and thereafter declining (see Figure 3).

Discussion

Transcranial Doppler is becoming an accepted method to assess the degree and extent of vasospasm following SAH. The most complete information is obtained by performing daily recordings on all of the basal vessels throughout the first 2 weeks following hemorrhage. This gives information on the severity and distribution as well as the time course of vessel narrowing. Previous studies suggest that the rate of increase in mean velocity is important in identifying patients at high risk of developing ischemic deficits.[14] Mean velocities greater than 200 cm/sec by the fifth day following SAH indicate a high risk for developing neurological deficits. It is also very helpful for the examiner to have obtained baseline values on patients to become familiar with each patient's particular anatomy. As vasospasm becomes more severe and the cross-sectional area of the vessels decreases, vessels become more difficult to locate with TCD. Faint high-frequency signals that are not quantifiable from vessels where good strong signals were found on previous recordings usually indicate severe vessel narrowing. This is especially true when recording from the anterior cerebral artery. The signals from the distal branches of the middle cerebral artery should also be evaluated for mean velocity and pulsatility. Occasionally with very severe vasospasm, proximal high-frequency MCA signals will diminish in strength as flow decreases, and the distal branches will show lower velocities with low pulsatility indicating proximal obstruction to flow. The examiner should note these indicators of spasm as well as quantifying mean velocity.

Transcranial Doppler can be a very valuable asset in the management of subarachnoid hemorrhage. It offers for the first time a way to assess intracranial vessel spasm noninvasively. Good correlations exist between vessel narrowing and mean velocity in the MCA.

Acknowledgments. We acknowledge the assistance of Patricia Midtskog in the preparation of this manuscript. This work was supported by NIH Grant NS07144.

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