# Evaluation of Vasospasm Using Transcranial Doppler

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Transcranial Doppler can record blood flow velocity in the basal cerebral arteries, making it a useful noninvasive method for detecting vasospasm. Elevated velocity readings correlate with reduced vessel diameter and usually precede the onset of delayed ischemic defects in patients with subarachnoid hemorrhage. This method is useful in following the development and resolution of vasospasm and in identifying patients who are at risk of developing delayed ischemia.

## INTRODUCTION

Transcranial Doppler ultrasonography has recently made possible the confirmation of cerebral vasospasm noninvasively (2,3,16). It has an added advantage over angiography of being able to follow the development and resolution of vasospasm with time. With the introduction of more effective treatments for vasospasm such as induced hypervolemia and hypertension (4), calcium channel blockers (7,17), and transluminal angioplasty (13,10), a method to diagnose and continually assess the degree of vasospasm is very helpful in the management of patients with subarachnoid hemorrhage.

Transcranial Doppler, by being able to assess the degree of spasm in the basal cerebral arteries can give an index of the degree and extent of cerebral vasospasm as well as its development and resolution. The clinical response to a given degree of vasospasm depends on a variety of factors and was initially believed not to correlate well with the severity and extent of angiographic vasospasm (12). A more careful examination of the phenomenon with increased knowledge about the time course and delayed onset of vasospasm has revealed a closer correlation of angiographic spasm and clinical neurologic deficits following subarachnoid hemorrhage (6). When interpreted in light of the current knowledge about vasospasm the knowledge of the degree, extent and development and resolution of basal artery narrowing is valuable in the management of patients with subarachnoid hemorrhage.

## PRINCIPLES OF TRANSCRANIAL DOPPLER

Transcranial Doppler ultrasonography was introduced in 1982 by Dr. Rune Aaslid and utilizes a 2 megahertz ($MH_Z$) pulsed ranged gated ultrasonic instrument to obtain velocity readings from the basal cerebral vessels (1). The pulsed range gated feature of this instrument allows selection of various recording depths making it possible to selectively focus on different intracranial arteries. By recording frequency shifts in the flowing blood in the intracranial arteries the velocity of flow in centimeters per second (CM/SEC) can be calculated by the equipment. Stenosis caused by vasospasm or other lesions will increase the velocity through the stenotic segment in proportion to the reduction of the cross sectional area of an artery when the same flow is maintained. By observing this phenomena one can gain an index of the degree of spasm in the basal vessels.

CORRELATION WITH ANGIOGRAPHY

Several studies have been performed in the past correlating the degree of vasospasm seen on angiography with TCD velocities found in patients following subarachnoid hemorrhage (3,8). Generally, there has been a good correlation between the degree of middle cerebral artery narrowing and the velocity readings. There are several inherent problems when correlating vessel diameters with velocity readings. Resting vessel diameters in a given population are somewhat variable and the perfusion territory of given vessels can also be variable. The cerebral blood flow in a given population following subarachnoid hemorrhage can also be variable and can be markedly reduced in patients with depressed levels of consciousness. Autoregulation can also be impaired and patients with subarachnoid hemorrhage particularly those patients with higher Hunt and Hess grades (5). It is therefore conceivable that the mean arterial pressure could significantly affect cerebral blood flow in this group of patients introducing another variable. Despite these variables we have found good correlations between cerebral artery diameter reductions and increases in mean velocity by examining a group of patients who had two serial angiograms following their subarachnoid hemorrhage. Angiograms which were obtained within 48 hours of the subarachnoid hemorrhage showing baseline values were compared to subsequent angiograms obtained to check clip placement or to document vasospasm. These angiograms were measured and compared to transcranial Doppler velocities which were obtained within 24 hours of each angiogram. By regression analysis the reduction in MCA diameters were found to correlate well with velocity increases seen on transcranial Doppler. (Figure 1a). To try to define parameters of severe vasospasm we examined patients with greater than 50% narrowing of the middle cerebral arteries between the two angiograms. We found 9 such middle cerebral arteries with greater than 50% narrowing, and all of the TCD examinations revealed mean velocities greater than 200 CM/SEC. (Figure 1b). Although the mean velocity value must be considered in light of other influences such as changes in cerebral blood flow which can occur, and differences due to age (19), a mean velocity of 200 cm per second in the middle cerebral artery generally indicates severe vasospasm defined by 50% of greater narrowing in the middle cerebral artery.

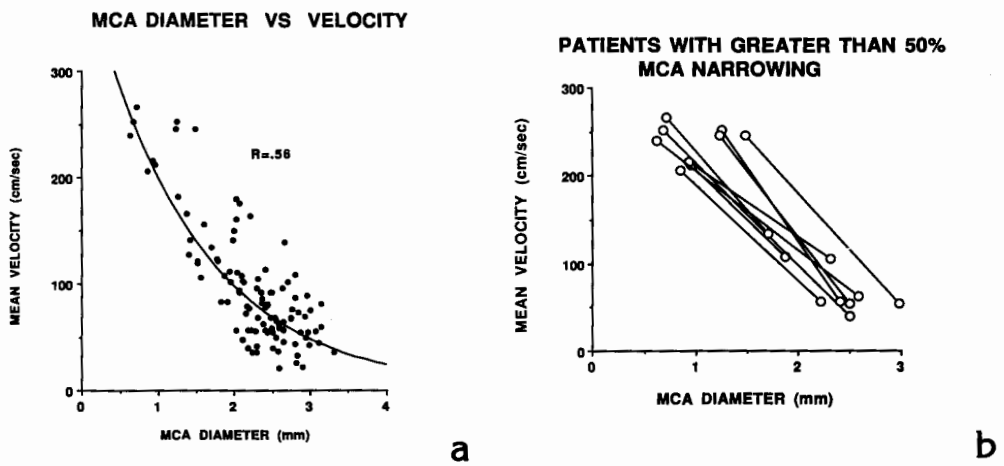


Fig. 1. a: Correlation of MCA diameter with mean velocity in patients who had 2 angiograms.
b: Elevations in mean velocity produced by 50% or greater narrowing at the MCA.

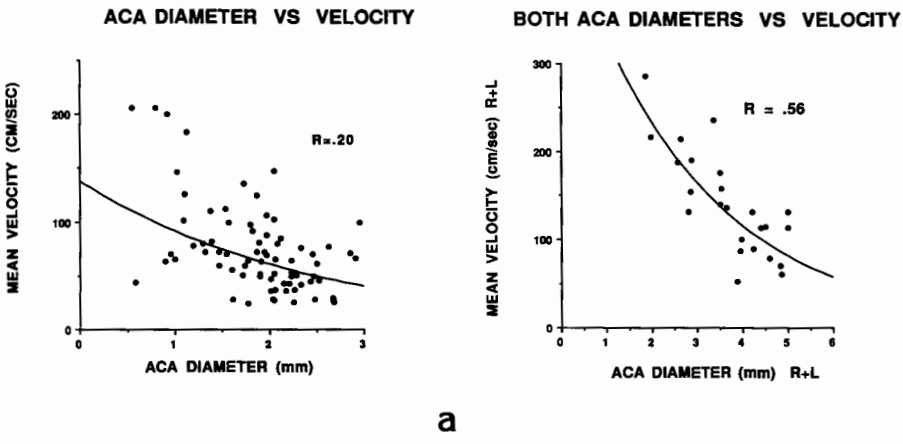


Fig. 2. a: Correlation of ACA diameter with mean velocity.
b: Correlation obtained by combining both ACA diameters and both velocities.

We also examined narrowing of the proximal portion of the anterior artery (A1 segment) in the same manner and found that if all the values were considered separately the correlation was not as close as the middle cerebral artery. (Figure 2a). Several factors can explain this poor correlation. One factor is that significant number of patients, especially those with subarachnoid hemorrhage, can have an atretic A1 segment on one side. It has been observed that during baseline examinations the velocity is usually not increased in atretic A1 segments (11) and this can lead to poor correlations if the diameter is related to the mean velocity with a regression analysis. Another factor to consider is that the anterior cerebral system has a collateral connection through the anterior communicating artery, as one anterior cerebral artery narrows in vasospasm the velocity can be increased through the contralateral anterior cerebral artery without a reduction in diameter. The data were then reconsidered, treating the anterior cerebral system as a single artery by plotting the combined arterial diameters against the combined arterial velocities. This yielded a much better regression analysis. (Figure 2b)

In order to consider the effect of cerebral blood changes following subarachnoid hemorrhage on mean velocity values, it can be helpful to obtain velocity recordings from the extracranial internal carotid arteries (2,11). This is done by using the 2 $MH_Z$ probe set at a depth of approximately 5 cm, and aimed toward the foramen lacerum from the angle of the jaw. This minimizes the angle of incidence on the artery to obtain a more true value of these velocity recordings as an index flow. It has been suggested that the values obtained can be constructed as a ratio of the velocity at the middle cerebral artery/ internal carotid artery (Vmca/Vica) to account for variabilities in cerebral blood flow in individual patients as well as compensate for decreases in cerebral blood flow which can occur with increasing degrees of vasospasm due to increases in distal cerebrovascular resistance (11). Other strategies include obtaining cerebral blood flow studies using Xenon (18) but these are often more costly and less easily repeatable.

## EXAMINATION STRATEGIES

In order to gain the most information for management of patients following

subarachnoid hemorrhage, examination should include baseline vessel measurements regular intervals to follow the development and resolution of vasospasm. Good baseline examination can be obtained during the first 24 to 48 hours following SAH when the velocity readings will usually be in the normal range (15). There are several reasons why good baseline examinations are important. 1) during vasospasm vessels become smaller and more difficult to locate with TCD. It is helpful for the examiner to be familiar with the anatomic figurations 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 accurate quantitation of the velocity signals becomes difficult. Faint high frequency signals that are present in a location were stronger quantifiable signals which were previously found usually indicates severe vasospasm even if the mean velocity value is not in the range for severe vasospasm. False positive values can occur due to hyperemia in patients with poor autoregulation although this is not generally confused with high grade vasospasm. Collateral flow through non-severely narrowed vessels can also occur through the anterior cerebral system and possibly through the middle cerebral artery which can provide collateral flow to other areas of the brain through leptomeningeal collaterals. Improved diagnostic sensitivity can be accomplished when considering the ratio of intracranial vessels over extracranial vessels (Vmca/Vica) to correct for CBF changes. False negative values for vasospasm can occur through examination error due to bone thickness or inexperience of the examiner. False negative examinations for vasospasm have also been noted to occur in occasional cases with only distal artery vasospasm which is normally inaccessible by TCD examination. It is estimated that the incidence of purely distal vasospasm is less than 10% (14).

## CORRELATION OF VELOCITY CHANGES WITH DELAYED ISCHEMIC DEFICITS

Although velocity changes in the basal intracranial arteries can be affected by several physiological 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 reflection of the angiogram. Some patients can remain asymptomatic with severe vasospasm demonstrated by angiogram and the incidence of angiographic vasospasm as nearly twice that of delayed ischemic deficits (9). Patients with severe vessel narrowing who remain asymptomatic most likely have well functioning compensatory mechanisms and adequate collateral pathways to provide adequate cerebral blood flow under these conditions. On the other hand, patients who have a true deterioration due to delayed ischemia following SAH almost without exception will show severe vessel narrowing by angiography (6). When using angiography to confirm vasospasm as the 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 SAH. High velocity readings identify patients at high risk for developing ischemic deficits, but they also may occur in asymptomatic patients (16). In patients who develop true delayed ischemia, careful search using TCD usually reveals the cause. In addition to considering the absolute velocity numbers, the rate of rise of the velocity values as well as the extent of vasospasm should be considered.

## EFFECT OF TREATMENT ON TCD VELOCITY VALUES

Recently three effective treatments have been introduced for vasospasm following SAH, hypervolemia and hypertension, calcium channel blockers, and transluminal angioplasty. There is little published data on the effects of these treatments on TCD values. The effect of hypervolemia and hypertension is not known in a large series of patients, but it is likely that the state of autoregulation will have a great influence on how this treatment influences the TCD values. Several preliminary studies of the effect of calcium channel blockers on TCD values have generally shown a reduction in mean velocity in

treated patients (7,17). Transluminal angioplasty has recently been introduced as a treatment for vasospasm and preliminary findings indicate that TCD values are markedly reduced in the basal arteries following treatment and that this reduction is sustained during subsequent serial examinations (13). (Figure 3). Several cases of increased TCD values have been observed following angioplasty in more distally located areas of spasm, when the proximal vessels are dilated.

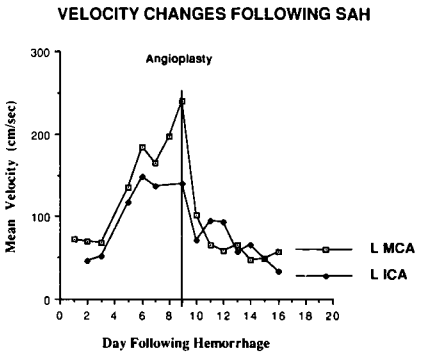


Fig 3. Serial mean velocity values in a patient before and after angioplasty of the left MCA and ICA.

## CONCLUSIONS

Transcranial Doppler is a noninvasive way to evaluate the degree of vasospasm in the basal cerebral vessels. The degree of vessel narrowing bears a direct relationship to the increase in blood flow velocity provided the blood flow remains constant. Changes in cerebral blood flow can occur following subarachnoid hemorrhage and these changes chould be considered to gain more accurate data. The clinical response to a given degree of basal vessel narrowing depends on many complex factors and similar angiography TCD can not identify with certainty an individual patient destined for clinical ischemic deficit. By following the degree as well as the development with time of vasospasm in the basal vessels patients can be categorized into high and low risk groups which can avoid unnecessary treatment in low risk groups and identify those patients at high risk for developing clinical ischemic deficits.

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