Relationship of Transcranial Doppler Flow Velocities and Arteriovenous Malformation Feeding Artery Pressures

Lauren H. Fleischer, MD; William L. Young, MD; John Pile-Spellman, MD; Buckley terPenneg, MD; Abraham Kader, MD; Bennett M. Stein, MD; J.P. Mohr, MD

Background and Purpose: Feeding mean arterial pressure immediately proximal to the nidus of arteriovenous malformations may influence the frequency of spontaneous intracerebral hemorrhage. This study assessed the usefulness of transcranial Doppler ultrasound velocities as a noninvasive estimate of feeding mean arterial pressure.

Methods: We studied 41 patients undergoing 73 staged treatments of arteriovenous malformations with endovascular embolization, surgery, or both. Before treatment during the awake state, transcranial Doppler mean and peak velocities were recorded in proximal Willisian vessels. During superselective angiography with the patient under conscious sedation or during surgery with the patient under general anesthesia, feeding mean arterial pressure was measured through a 1.5F transfemoral intracranial microcatheter or a 26-g needle by direct puncture. Measurement of insonated artery diameter was possible in 41 embolizations, and a flow velocity index (ml/min) and Reynolds’ number were estimated.

Results: Mean±SEM feeding mean arterial pressure was 38±2 mm Hg at a systemic mean arterial pressure of 77±2 mm Hg; mean velocity was 102±4 cm/s. There was an inverse correlation between feeding mean arterial pressure and parent artery mean velocity (y = -0.74x + 130, r = .75, P < .0025). The best correlation was for the first treatment in each patient (n=27) using the highest peak velocity obtainable in a Willisian vessel ipsilateral to the arteriovenous malformation (y = -1.61x + 221, r = .62, P < .0005). Flow velocity index (775±106 mL/min) did not correlate with feeding mean arterial pressure, but there was a weak correlation with Reynolds’ number (y = -12x + 161, r = .27, P = .1283). Mean Reynolds’ number was 1257±119.

Conclusions: Transcranial Doppler mean velocity is correlated with feeding mean arterial pressure but only weakly predictive. Considerations influencing the relation of distal feeding mean arterial pressure to proximal mean velocity might include the influence of other fistulae in the circuit between major inflow and outflow channels as well as turbulent flow at vascular branch points between point of insonation and the nidus, as suggested by Reynolds’ number values of more than 400. (Stroke. 1993;24:1897-1902.)

KEY WORDS • arteriovenous malformations • cerebral arteries • intracranial hemorrhage • ultrasonics

The most devastating consequence of a cerebral arteriovenous malformation (AVM) is the occurrence of spontaneous intracerebral hemorrhage (ICH). The exact pathophysiology of ICH is not known, but pressure derangements on both the arterial and venous sides of the AVM nidus are the subject of increasing investigation. In particular, recent evidence suggests that lower pressure in the distal feeding arteries may be protective, whereas higher pressures appear to increase the risk of ICH and the size of the hematoma.

A noninvasive method of determining distal feeding artery pressure could be used to screen patients for risk assessment to help guide recommendations as to the necessity of undergoing surgical, endovascular, or radiosurgical treatment for AVMs to favorably influence the natural history of ICH. Preliminary results of comparing feeding artery and draining vein pressures measured at both surgery and embolization suggested that there was a relation between feeding artery (but not draining vein pressures) and proximal transcranial Doppler ultrasound (TCD) velocities. The aim of this study was to assess the use of TCD in estimating downstream arterial pressures in patients with AVMs.

Subjects and Methods

After institutional approval and informed consent, studies were performed on 41 patients undergoing a total of 73 staged treatments of AVMs with endovascular embolization (n=26), surgery (n=11), or both (n=4). TCD (Medasonics Transpect, Freemont, Calif; EME, Uberlingen, Germany) was used to measure velocities in the proximal cisternal portion of the anterior (ACA), middle (MCA), or posterior (PCA) cerebral artery.
Pressure measurements were made before the injection of embolic material, N-butyl cyanoacrylate. Although the use of microcatheters to accurately measure systemic mean pressure has previously been confirmed,4 we verified this in each case as follows. Before the microcatheter was advanced distally, it was placed just beyond the orifice of the coaxial catheter (see Fig 1) in the extracranial ICA or VA. Pressures were recorded simultaneously at the microcatheter tip, coaxial orifice, and femoral sheath, and it was confirmed that all pressures were within 5% of each other.

At each embolization procedure, one FMAP was measured, and this value was correlated with the corresponding preprocedural TCD velocity recorded for its parent artery.

For the surgical measurements, patients were anesthetized with an isoflurane/N2O technique as previously described.5 After dural reflection, the largest feeding artery branch that fed the AVM was cannulated with a 26-g needle, and the pressures were recorded with equipment identical to that described above for embolization. Pressures were relative to the right atrium, and SMAP was taken from an indwelling radial artery catheter.

AVM size was determined by the neuroradiologist from the patients’ initial magnetic resonance imaging (MRI) or contrast-enhanced computed tomography (CT) study. In addition, parent artery diameter at the point of TCD insonation was measured from pretreatment angiograms in 41 embolization procedures, and a flow velocity index (FVI; mL/min) was calculated as follows:

\[ FVI = \pi r^2 v \times 60 \]

Arterial diameter was measured as close to the point of maximum insonation signal as was practical. This required visualization of the arterial segment in the coronal plane; therefore, measurements were made in anteroposterior projections whenever possible. Arterial diameter on film was compared with a known standard disk or ring taped to the patient’s head on both the beam entry and exit sides, and magnification was corrected on the basis of arterial position with respect to the two standards (n=10). When no interposed standard existed (n=31), bone landmarks such as clinoparietal line or sellar anteroposterior diameter were used only if digital MRI or CT could be used to scale such dimensions. Angiographic examinations without standards and ambiguous bone dimensions were excluded.

Reynolds’ number (Re) was also estimated as described by Guyton.6

\[ \text{Re} = \frac{\nu \cdot d}{\eta} \]

\[ \text{Re} = \frac{\nu}{\nu_d} \]

\[ \nu = \frac{L}{t} \]

\[ \text{Diameter} = \frac{L}{t} \]

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where \( v \) is \( \bar{v} \), \( d \) is vessel diameter, \( \eta \) is viscosity (estimated at 1/30 poises), and \( \rho \) is density (estimated at 1.05).

Parametric data were analyzed by linear regression and analysis of variance or covariance. \( \chi^2 \) test was used where appropriate. Results were considered significant at \( P<.05 \) and are reported as mean±SEM values.

**Results**

Mean patient age was 36±2 years; 15 were women, and 26 were men. Hematocrit was 41±1 (range, 30 to 49). Twelve patients had data collected at one embolization, 10 patients at two embolizations, 6 at three embolizations, and 2 at four embolizations. Eleven had measurement made at surgery only; 4 had measurements at both embolization and surgery.

Hemorrhage was the presenting finding in 14 patients (34%), 15 presented with seizures (37%), 9 with headache (22%), 1 with slurred speech and hand numbness, 1 with aneurysmal subarachnoid hemorrhage, and 1 patient was asymptomatic. Twelve lesions were fed primarily by the posterior circulation (PCA) and 29 by the anterior circulation (MCA and ACA). Left-sided lesions were predominant (22 of 41). Mean AVM size was 4.4×3.8×3.6 cm. The mean greatest diameter was 4.7±0.3 cm (range, 2.5 to 10). Primary locations were frontal (13), parietal (10), temporal (11), and occipital (7).

For the embolization cases, the ratio of mean pressure measured in the femoral artery to the coaxial catheter orifice was 0.98±0.01, with a coefficient of variation of 3%. The ratio of mean pressure measured in coaxial catheter orifice to the mean pressure at tip of the microcatheter placed just distal to it was 1.03±0.01, with a coefficient of variation of 3%.

Mean physiological values for all patients are shown in the table. Mean FMAP was 38±2 mm Hg at a SMAP of 77±2 mm Hg; 56% of patients had an FMAP ≤40 mm Hg, 36% had an FMAP between 40 and 60 mm Hg, and 8% had an FMAP of more than 60 mm Hg. Mean \( \bar{v} \) was 102±4 cm/s. MCA velocities were highest, followed by ACA and PCA. There was an inverse correlation between FMAP and parent vessel \( \bar{v} \). Feeders with the lowest FMAP exhibited the highest \( \bar{v} \) in the parent vessels (\( y=-0.74x+130, r=.35, P=.0025 \)) (Fig 2). A similar association was found for \( \bar{v} \) in the parent vessel (\( y=-0.91x+177, r=.36, P=.0017 \)). The relation was best when the highest \( \bar{v} \) in the MCA, ACA, or PCA ipsilateral to the AVM was correlated with FMAP (\( y=-1.10x+200, r=.49, P=.0001 \)) (Fig 3). No clear association was noted between parent vessel pulsatility index and FMAP. Substituting the ratios (FMAP/SMAP) and differences (SMAP−FMAP) for FMAP did not improve any of the correlations.

The largest AVMs had a tendency to have the lowest FMAP and highest TCD velocities, but these findings did not reach statistical significance. By analysis of covariance, the size of the AVM (greatest dimension) did not significantly interact with the correlation between FMAP and \( \bar{v} \) (\( P=.707 \)). Calculated flow

![Fig 2. Scatterplot of relation of feeding mean artery pressure (FMAP) to Willisian parent artery mean velocity (\( \bar{v} \)). The parent artery giving rise to the feeder in which pressure measurements were made is indicated by the key (\( y=-.74x+130, r=.35, P=.0025 \)). PCA, posterior cerebral artery; MCA, middle cerebral artery; and ACA, anterior cerebral artery.](http://stroke.ahajournals.org/)

**Table**

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<th>Pressure, Velocity, and Vessel Measurements for 41 Patients Undergoing 73 Procedures</th>
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<td>Lowest pulsatility index</td>
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<td>Diameter, mm</td>
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<td>Flow velocity index, mL/min</td>
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MCA indicates middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; FMAP, feeding mean arterial pressure; and SMAP, systemic mean arterial pressure.

*MCA>PCA, P<.05; †MCA>PCA, P<.01; ‡ACA>PCA, P<.01.
(775±106 mL/min) did not correlate with FMAP, but there was a weak correlation with Re \( (y = -12x + 1616, r = .27, P = .1283) \), as shown in Fig 4. Mean Re was 1257±119.

When examination of the relation of FMAP and TCD velocities was limited to data gathered before a patient’s first treatment (either surgery or embolization, \( n = 27 \)), the correlation between proximal velocities and distal FMAP improved \( (y = -1.61x + 221, r = .62, P = .0005) \), as shown in Fig 5.

There was no difference in any of the demographic, measured, or calculated physiological variables in patients with a hemorrhagic versus nonhemorrhagic presentation \( (P > .28) \).

**Discussion**

Several studies have reported separately on FMAP\(^1,4,7-14\) and TCD velocities in AVM patients\(^15-21\). The present study is the first report of the relation between them. Our findings for FMAP and TCD velocities are in agreement with those previously reported.

A weak but statistically significant correlation was found between FMAP and TCD measurements of the peak \( (P_v) \) and mean \( (\bar{v}) \) velocity in the “parent” vessel feeding the AVM. The correlations were best for patients before any treatment, and the correlation was weaker afterward. The correlations, although statistically significant, were too weak to permit using measurement of TCD velocities to predict the precise arterial pressure at or near the entry of feeding vessels into the nidus. TCD may be convenient and noninvasive, but our findings suggest that it is an uncertain source of insight into the dynamics of pressure changes and risks of rupture. If any inferences on pressures at the nidus should be attempted, our data suggest that the initial, pretreatment TCD measurements should be the most reliable.

Some methodological considerations may have affected the correlation of FMAP with TCD velocities. First, some of the weakness in correlation may be explained by the long distance between the sites available for TCD velocity measurements and the fistula itself. Most of the patients had AVMs that were rather distal in the circulation, fed by arterial branches that were third-order or more distal branches from the TCD measurement site (M1, P1, or A1), and this argues against the isolated significance of the distance between the TCD velocity measurement site and the fistula.

Second, measurement of arterial diameter was corrected for image magnification. The magnification standards, however, were not always in the same plane as the measured vessel, thus contributing to an error measurement estimated to be between 5% and 10%.

Third, during embolization, FMAPs were taken close to the nidus, at the point of embolic material injection; this point differed slightly across procedures. However, our experience is that pressure drops encountered along AVM feeding arteries tend to occur at major branch points\(^11\) and the FMAP was measured in all cases distal to any angiographically demonstrable major tributaries to normal regions, similar to the site of measurement during surgery. In our experience, small variations in the anatomic location of the catheter tip in the distal feeding artery do not significantly affect FMAP.

Finally, TCD velocities and FMAP measurements were not performed simultaneously, and this could have played a small role in the variance. Patients were awake during TCD measurements and moderately sedated during embolization or under general anesthesia for the surgical measurements. A role for arterial gas tension could exist. Compared with the awake state, PaCO\(_2\) was higher during embolization and lower during general

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**Fig 3.** Scatterplot of relation of feeding mean artery pressure (FMAP) to highest peak velocity \( (P_v) \) measured at the circle of Willis ipsilateral to the lesion. Whether the measurement was obtained during surgery or embolization is indicated by the key \( (y = -1.10x + 200, r = .49, P = .0001) \).

**Fig 4.** Scatterplot of relation of Reynolds’ number to feeding mean artery pressure \( (y = -12x + 1616, r = .27, P = .1283) \).

**Fig 5.** Scatterplot showing that when the examination of the relation of feeding mean artery pressure (FMAP) and transcranial Doppler velocities was limited to data gathered before a patient’s first treatment (either surgery or embolization, \( n = 27 \)), the correlation between proximal velocities and distal FMAP improved \( (y = -1.61x + 221, r = .62, P = .0005) \).
anesthesia for craniotomy. Additionally, alterations in the FMAP theoretically could occur from opening the skull at operation.

There are several physiological considerations that may have affected the correlations. First, a slightly better correlation was found when the highest $P_v$ rather than the actual measured vessel $P_v$ or even $P_v$ was used. This finding suggests that the AVM nidus exerts an influence on the lowering of pressure in all of the vessels that reach the fistula, possibly causing the branch that has the lowest pressure to influence all more proximally situated “parent” vessels. Assuming there is some degree of physiological “compartimentalization” in an AVM nidus, the measured pressure in any given feeding vessel may be determined by the lowest resistance fistula in the circuit.

FMAP did not show a strong correlation with $R_e$, a value derived by the velocity measured by TCD and the vessel diameter measured by angiography. Although $R_e$ values of more than 2000 are highly predictive of turbulent flow, and some of our observations approached this number, the mean $R_e$ was considerably less. This finding argues that the variance in the FMAP-TCD velocity correlations was not explained by turbulence along the entire length of the vessels being measured. However, the presence of a pulsatile turbulence along feeding vessels, greater at branch points, was suggested by the observation that the $R_e$ was more than 400 in virtually all of the vessels studied and that peak velocities rather than mean velocities correlated better overall with FMAP.

It was a hope of the present study to address some of the factors that explain the impression that small AVMs have a greater tendency to bleed. The reasons for this propensity to hemorrhage are unclear. Intraoperative measurements of FMAP in a recent study demonstrated that small AVMs (≤3 cm) had significantly higher pressures than did medium and large AVMs. This was offered as a reason why smaller lesions bleed more often and more aggressively, although four of the eight patients with small AVMs in that series with high FMAP were operated on for acute hematoma with mass effect, and this may have influenced the measured pressures. We could not find a significant relation between a presentation of hemorrhage and the various physiological variables, but our series consisted of a highly selected group of patients coming for staged embolization and surgery; only one of the patients had an AVM of less than 3 cm. This fact should not have affected our other findings, as the size of the lesion did not influence the relation between FMAP and TCD velocities.

Future studies with larger numbers of patients should attempt to clarify which hemodynamic factors, if any, significantly influence the pathogenesis and incidence of spontaneous AVM hemorrhage, whether it be vascular pressures or whether flow velocity itself is a determinant. Pathophysiological determinants of spontaneous ICH and the means to noninvasively estimate them are needed to assist in the risk assessment of AVMs when planning treatment course.

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References

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