Transcranial Doppler Ultrasonographic Changes After Treatment for Arteriovenous Malformations

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We performed transcranial Doppler ultrasonography on 15 patients with arteriovenous malformations before and after embolization or surgical resection to compare quantitatively the hemodynamic effects of these two treatments. Changes in mean blood velocity and pulsatility index were analyzed in 19 treated feeding arteries. Blood velocity decreased by a mean of 38.1% or 46.5 cm/sec (p<0.0001, two-tailed paired t test); decreases were greater for surgically resected arteries (46.2% or 55.9 cm/sec, p<0.003) than for embolized arteries (30.8% or 38.0 cm/sec, p<0.0003). Pulsatility index increased by a mean of 54.7% or 0.25 (p=0.0001); increases were greater for surgically resected arteries (65.8% or 0.29, p=0.0045) than for embolized arteries (44.8% or 0.20, p<0.001). The differences in the changes in blood velocity and pulsatility index between treatment groups were not significant. These data demonstrate that embolization results in hemodynamic changes that are qualitatively similar to those occurring after surgical resection of arteriovenous malformations. Transcranial Doppler ultrasonography is a reliable and convenient noninvasive method for monitoring hemodynamic effects of treatments for arteriovenous malformations. (Stroke 1990;21:260-266)

Transcranial Doppler ultrasonography (TCD) permits noninvasive measurement of the velocity of blood in the intracranial arteries.1 Qualitative studies of TCD findings in patients with arteriovenous malformations (AVMs) have been described.2-4 Feeding arteries of AVMs typically have higher-than-normal mean blood velocities with turbulence and lower-than-normal pulsatility indexes. TCD is useful in diagnosing AVMs noninvasively and in identifying major feeding arteries, minor feeding arteries, and nonfeeding arteries.2,4 TCD may also prove to be a reliable method in assessing the hemodynamic effects of therapy. To study this, we performed TCD before and after treatment for AVMs in 15 patients, comparing the hemodynamic changes in feeding and nonfeeding arteries that occur after surgical resection and embolization.

Subjects and Methods

We studied 15 patients with AVMs before and after embolization or surgical resection between February 1, 1988, and January 31, 1989. The patients ranged in age from 16 to 50 years. Table 1 summarizes the location, feeding arteries, and method of treatment for each AVM. Six patients had embolization only, four had surgical resection only, and five had staged treatment consisting of surgical resection following embolization. This combination of patients and treatments allowed us to analyze the effects of 10 embolizations affecting nine feeding arteries (one feeding artery was embolized twice, patient 15) and seven surgical resections affecting nine feeding arteries. Embolizations were performed using a superselective catheterization technique employing thrombogenic platinum coils.6 All embolizations and surgical resections, as well as the identities of the feeding and nonfeeding arteries, were verified with pretreatment and posttreatment angiography. The AVMs were completely resected in all surgically treated patients except for patient 6, in whom a small nidus of residual AVM was noted on the postoperative angiogram.

A pulsed, range-gated 2-MHz TCD (EME TC2-64B, Eden Medizinische Elektronik GmbH, Überlingen, F.R.G.) was used to measure the mean blood velocity (V) and the pulsatility index (PI) in feeding and nonfeeding arteries using the transtemporal or suboccipital ultrasonic windows as described by Aaslid7 at the same depth of insonation before and
TABLE 1. Location, Feeding Arteries, and Method of Treatment for Arteriovenous Malformations and Changes in Mean Blood Velocity and Pulsatility Index in 15 Patients

<table>
<thead>
<tr>
<th>Pt</th>
<th>Location</th>
<th>Artery</th>
<th>Treatment</th>
<th>Before</th>
<th>After</th>
<th>% change</th>
<th>Before</th>
<th>After</th>
<th>% change</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>L frontoparietal</td>
<td>L MCA</td>
<td>Surgery</td>
<td>138</td>
<td>102</td>
<td>-26.1</td>
<td>0.62</td>
<td>0.63</td>
<td>1.6</td>
</tr>
<tr>
<td>2</td>
<td>L bg/thal/parietal</td>
<td>L MCA</td>
<td>Surgery</td>
<td>100</td>
<td>78</td>
<td>-22.0</td>
<td>0.60</td>
<td>0.69</td>
<td>15.0</td>
</tr>
<tr>
<td>3</td>
<td>Cerebellar</td>
<td>BA</td>
<td>Embolization</td>
<td>72</td>
<td>52</td>
<td>-27.8</td>
<td>0.50</td>
<td>0.93</td>
<td>86.0</td>
</tr>
<tr>
<td>4</td>
<td>L frontal</td>
<td>L MCA</td>
<td>Embolization</td>
<td>108</td>
<td>82</td>
<td>-24.1</td>
<td>0.50</td>
<td>0.92</td>
<td>84.0</td>
</tr>
<tr>
<td>5</td>
<td>L parietal</td>
<td>L ACA</td>
<td>Embolization</td>
<td>132</td>
<td>94</td>
<td>-28.5</td>
<td>0.56</td>
<td>0.58</td>
<td>3.6</td>
</tr>
<tr>
<td>6</td>
<td>L temporo-occipital</td>
<td>L PCA</td>
<td>Surgery</td>
<td>86</td>
<td>38</td>
<td>-55.8</td>
<td>0.39</td>
<td>0.86</td>
<td>120.5</td>
</tr>
<tr>
<td>7</td>
<td>L frontal</td>
<td>L MCA</td>
<td>Embo.</td>
<td>100</td>
<td>100</td>
<td>0</td>
<td>0.48</td>
<td>0.63</td>
<td>31.3</td>
</tr>
<tr>
<td>8</td>
<td>L temporo-occipital</td>
<td>L PCA</td>
<td>Embo.</td>
<td>142</td>
<td>74</td>
<td>-47.9</td>
<td>0.47</td>
<td>0.72</td>
<td>53.2</td>
</tr>
<tr>
<td>9</td>
<td>R frontal</td>
<td>R MCA</td>
<td>Surgery</td>
<td>108</td>
<td>100</td>
<td>-7.4</td>
<td>0.55</td>
<td>0.68</td>
<td>23.6</td>
</tr>
<tr>
<td>10</td>
<td>L occipital</td>
<td>L MCA</td>
<td>Embolization</td>
<td>72</td>
<td>18</td>
<td>-75.0</td>
<td>0.46</td>
<td>1.09</td>
<td>137.0</td>
</tr>
<tr>
<td>11</td>
<td>L frontal</td>
<td>L MCA</td>
<td>Embolization</td>
<td>196</td>
<td>142</td>
<td>-27.6</td>
<td>0.45</td>
<td>0.59</td>
<td>31.1</td>
</tr>
<tr>
<td>12</td>
<td>L frontal</td>
<td>L MCA</td>
<td>Surgery</td>
<td>135</td>
<td>64</td>
<td>-52.6</td>
<td>0.75</td>
<td>0.90</td>
<td>20.0</td>
</tr>
<tr>
<td>13</td>
<td>L occipital</td>
<td>L PCA</td>
<td>Surgery</td>
<td>154</td>
<td>36</td>
<td>-76.6</td>
<td>0.41</td>
<td>0.94</td>
<td>129.3</td>
</tr>
<tr>
<td>14</td>
<td>Cerebellar</td>
<td>BA</td>
<td>Embolization</td>
<td>82</td>
<td>40</td>
<td>-51.2</td>
<td>0.65</td>
<td>0.82</td>
<td>26.2</td>
</tr>
<tr>
<td>15</td>
<td>R temporal</td>
<td>R PCA</td>
<td>Embolization</td>
<td>172</td>
<td>112</td>
<td>-34.9</td>
<td>0.23</td>
<td>0.34</td>
<td>47.8</td>
</tr>
</tbody>
</table>

Pt, patient; L, left; R, right; MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; BA, basilar artery; bg, basal ganglia; thal, thalamus.

after treatment (Table 1). PCO₂ was not monitored. Since the feeding and nonfeeding arteries were angiographically verified before and after treatment, we did not perform vasoreactivity testing to identify arteries. PI, an index of vascular resistance, is determined as \[
\frac{\text{systolic blood velocity} - \text{diastolic blood velocity}}{\text{systolic blood velocity}} \times 100
\] In most instances, pretreatment and posttreatment TCDs were performed within 1 week of treatment (range 1–70 days). The average time from treatment to posttreatment TCD was 8.1 days (embolization group 2.6 days, surgery group 16 days).

The two-tailed paired t test was used to assess the significance of changes in V and PI after treatment in both feeding and nonfeeding arteries. The two-tailed unpaired t test was used to compare V and PI changes in feeding arteries between the surgical resection and embolization treatment groups.

Results

One case report typifies our experience in the 15 patients studied. Patient 14 was a 16-year-old girl with an AVM of the cerebellar vermis and tectum fed by branches from the basilar artery (Figure 1, top left). Based on measurements obtained from the lateral view of the pretreatment angiogram and taking magnification into account, the origin of this patient’s basilar artery was estimated to be at a depth of 75 mm. TCD of the basilar artery at this depth before embolization demonstrated an abnormally high V (82 cm/sec; normal 31±9 cm/sec) and a low-normal PI (0.65; normal 1.2±0.6) (Figure 1, bottom left). She underwent superselective platinum coil embolization of the right superior cerebellar artery branches feeding the AVM (Figure 1, top right). After embolization, there was a 51% reduction in V in the basilar artery (to 40 cm/sec) and a 26% increase in PI (to 0.82) (Figure 1, bottom right).

For the 15 patients overall, pretreatment V in every feeding artery was abnormally high compared with published normal ranges (Table 1). Posttreatment V was decreased in each treated feeding artery, except in one feeding artery that had been embolized previously (patient 7, Table 1). The average decrease was 38.1% (range 76.6–0%) or 46.5 cm/sec (p=0.0001). Posttreatment V was normal in five of nine (55.6%: patients 6, 10, 12, 13, and 15 [R PCA], Table 1) surgically resected feeding arteries. Furthermore, mean V decrease was greater for the surgically resected feeding arteries. Furthermore, mean V decrease was greater for the surgically resected feeding arteries (Figure 2, top left; 46.2% [range 76.6–74.0%] or 45.5 cm/sec; p=0.0003) than for the embolized feeding arteries (Figure 2, top right; 30.8% [range 51.2–0%] or 38.0 cm/sec; p=0.0003), but the difference between treatment groups was not significant (p=0.19). There were no significant trends in the changes in V of nonfeeding arteries (Figure 2, bottom).

Pretreatment PI in all but two feeding arteries was abnormally low compared with published normal ranges (low-normal in patients 12 and 14; Table 1). PI increased after treatment in every treated feeding
artery (mean 54.7% [range 137–1.6%] or 0.25, p=0.0001). Posttreatment PIs were normal in eight of nine (88.9%; patients 1 [L ACA], 6, 9, 10, 12, 13, and 15 [R MCA], Table1) surgically resected feeding arteries and in five of 10 (50%; patients 2, 3, 6, 8, and 14) embolized feeding arteries. Similar to the relation for V, mean PI increase was greater in the surgically resected feeding arteries (Figure 3, top left; 65.8% [range 137–1.6%] or 0.29, p=0.0045) than in the embolized feeding arteries (Figure 3, top right; 44.8% [range 86–3.6%] or 0.20, p=0.001), but the difference between treatment groups did not reach
PFE POSTSURGERY mean velocity change = -55.9 cm/sec, p = .0029

PFE EMBOLIZATION POST
mean velocity change = -38.0 cm/sec, p = .0003

FIGURE 2. Changes in mean blood velocity after treatment of arteriovenous malformations (AVMs). Top left: Change in feeding arteries after surgical resection of nine AVMs. Top right: Change in feeding arteries after embolization of 10 AVMs. Bottom: Change in 24 nonfeeding arteries after treatment (surgical resection or embolization).

significance (p=0.34). As was the case for V, there were no significant trends in PI changes in nonfeeding arteries (Figure 3, bottom).

In four instances, AVMs with multiple feeding arteries underwent embolization of a single feeding artery (Table 2). After treatment, in two instances (patient 7 and patient 15, first embolization) V increased and PI decreased in nonembolized feeding arteries. However, in one instance (patient 5) V and PI both decreased, and in another instance (patient 15, second embolization) V and PI both increased.

Measurements were made in four nonfeeding arteries ipsilateral to the AVM (Table 3). In three instances (patients 6, 8, and 12), V increased after treatment. However, there was no significant trend in PI changes in these nonfeeding arteries.

Discussion

It has been assumed, but not previously documented, that embolization produces hemodynamic changes in AVMs that are similar to those occurring after surgical resection. Our data support this thesis.
by demonstrating that embolization consistently has a therapeutic hemodynamic effect on the embolized feeding artery similar to that seen after surgical resection (V decreases and PI increases, Figure 2, top left and top right; Figure 3, top left and top right). These qualitatively similar hemodynamic effects are not readily documented with angiography. Our data also support the commonly held assumption that surgical resection, in which the AVM shunt is completely or nearly completely removed in one step, produces greater hemodynamic changes in feeding arteries than embolization, in which the rationale of therapy is to reduce the shunt in a stepwise fashion by embolizing selected feeding branches in staged procedures over time. The changes in V and PI were more dramatic in the surgically resected feeding arteries than in the embolized arteries (Figure 2, top left and top right; Figure 3, top left and top right). Although these were consistent trends, the differences between treatment groups did not reach statistical significance, possibly because the sample size was small (10 embolized
arteries and nine surgically resected arteries). Further study of more patients will be required to confirm this trend. Our findings in this regard could have been influenced by the fact that, on average, posttreatment TCD was performed earlier after embolization (2.6 days) than after surgery (16 days). It is possible that V and PI change over time after embolization, depending on the hemodynamic behavior of the AVM feeding arteries and the thrombotic effect of the embolization coils. This possibility is the subject of a prospective study currently underway in our laboratory. Nevertheless, our TCD findings justify embolization as a therapeutic modality for AVMs, at least on hemodynamic grounds.

In patients with more than one major feeding artery, we were not able to demonstrate consistent trends in V or PI of the untreated feeding arteries after embolization (Table 2); however, we did document several dramatic changes. In two instances (patient 7 and patient 15, first embolization) V increased and PI decreased, and in another instance (patient 15, second embolization) V increased but PI did not decrease. These cases suggest that AVMs are capable of recruiting blood flow from one feeding artery when that from another feeding artery is reduced, attesting to the dynamic behavior of these lesions.

Changes in nonfeeding arteries ipsilateral to the AVM were also documented in some instances after either embolization or surgical resection (Table 3). In three instances (patients 6, 8, and 12) V increased, consistent with the possibility that the AVM had exerted a "steal" effect on these nonfeeding arteries before treatment. However, there had been no symptoms or signs suggestive of steal before treatment in any of these three patients and in no patient did neurologic function improve after treatment, suggesting that if the AVM indeed had been exerting a hemodynamic steal effect it was clinically unimportant.

All of our patients had an abnormally high V and all but two had an abnormally low PI in the feeding arteries of their AVMs before treatment, corroborating previous findings that TCD correctly diagnoses AVMs by detecting this V or V-PI profile with >87% sensitivity. More importantly, we were able to quantify consistent and significant hemodynamic trends (decreased V and/or increased PI) after embolization or surgical resection in every treated feeding artery (Figure 2, top left and top right; Figure 3, top left and top right). These data are consistent with intraoperative blood pressure and velocity measurements in feeding arteries of AVMs before and after test occlusion that have demonstrated a decrease in V and an increase in blood pressure and resistance (reflected in PI). Our findings could have been influenced by the fact that we did not control PCO2 when TCD was performed. However, the consistent posttreatment trends in V and PI of feeding arteries could not be simply artifacts of differences in PCO2 before and after treatment since these trends were not found in nonfeeding arteries (Figure 2, bottom; Figure 3, bottom). Our findings demonstrate that TCD is a convenient and reliable method to evaluate quantitatively the hemodynamic changes occurring in feeding and nonfeeding arteries of AVMs after embolization or surgical resection. As further experience is acquired with TCD in this setting, the technology may prove to be useful in planning and monitoring the effects of surgical resection, embolization, or radiotherapy of AVMs.

References

KEY WORDS • cerebrovascular disorders • cerebral arteriovenous malformations • ultrasounds
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