Pathophysiologic Assessment of Stagnating Arteries after Removal of Arteriovenous Malformations

Yoshio Miyasaka,1,3 Kenzoh Yada,1 Takashi Ohwada,2 Takao Kitahara,2 Akira Kurata,1 and Katumi Irikura1

PURPOSE: To assess the hemodynamics and pathophysiology of stagnating arteries after removal of arteriovenous malformations (AVMs). SUBJECTS: 50 patients with supratentorial pial AVMs underwent pre- and postoperative angiographic studies. RESULTS: The following characteristics were found to correlate with stagnating arteries: 1) advanced patient age, 2) large AVM size, 3) markedly dilated feeders, 4) early postoperative angiograms, and 5) delayed restoration of feeding artery diameter. CONCLUSIONS: The rate of blood flow in the former feeding arteries, expressed as \( v \times \pi \times r^2 \) (\( v \) = mean velocity, \( r \) = vessel radius), suddenly decreases after removal of AVMs. When dilatation persists postoperatively in these arteries the flow velocity decreases and stagnation takes place. Delayed postoperative restoration of feeding artery diameter may be caused by a decrease of elasticity due to long-standing hemodynamic stresses, and by increased postoperative vascular resistance of these arteries.

Index terms: Arteriovenous malformations; Angiography, postoperative; Arteries, flow dynamics

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Arteries that previously fed excised arteriovenous malformations (AVMs) often show slow blood flow, and remain visible even in the late arterial and venous phases on postoperative angiograms. These abnormal vessels have been termed “stagnating arteries” (1-4), and have been characterized hemodynamically by sluggish arterial flow and high intraarterial pressure (2, 3). Our purpose in this study was to analyze stagnating arteries and report our pathophysiologic/hemodynamic findings.

Clinical Materials and Methods

Fifty patients with supratentorial pial AVMs underwent pre- and postoperative angiography. The subjects ranged in age from 14 to 71 years (average age, 36 years). Using the classification of Waltimo (5), in which an AVM is defined as “large” if its maximum diameter is 3 cm or more and “small” if less than 3 cm, we found 38 patients to have large AVMs, and 12 small.

A total of 69 postoperative angiographic studies were undertaken in the 50 patients. The times when the postoperative angiograms were obtained are shown in Table 1: two postoperative angiograms were obtained in 16 patients, while three postoperative studies were obtained in one.

The criteria for a stagnating artery were the persistent presence of the feeding artery to an excised AVM in the late arterial and venous phases of the postoperative angiogram, and the persistence of angiographic visualization lasting for 10 seconds or more (Fig. 1) (2).

The ratio of the maximum diameter of the most dilated feeding artery (FA) to the maximum diameter of the internal carotid artery (ICA) in the cavernous portion (FA/ICA ratio) was obtained to express quantitatively the degree of preoperative dilatation of the feeding arteries. The FA/ICA

<table>
<thead>
<tr>
<th>Time of Postoperative Angiograms (Postoperative Date)</th>
<th>Postoperative Angiograms (No. of Cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Operative day</td>
<td>Initial: 24; Repeat: 0</td>
</tr>
<tr>
<td>2-7 days</td>
<td>Initial: 5; Repeat: 0</td>
</tr>
<tr>
<td>8-30 days</td>
<td>Initial: 18; Repeat: 7</td>
</tr>
<tr>
<td>30 days or more after</td>
<td>Initial: 3; Repeat: 12</td>
</tr>
<tr>
<td>Total</td>
<td>Initial: 50; Repeat: 19</td>
</tr>
</tbody>
</table>

TABLE 1: Times of postoperative angiograms

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1 Department of Neurosurgery and 2 Critical Care Medicine, Kitasato University School of Medicine, Kanagawa, Japan.
3 Address reprint requests to Yoshio Miyasaka, MD, Department of Neurosurgery, Kitasato University School of Medicine, 1-15-1 Sagamihara, Kanagawa, 228 Japan.

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Fig. 1. A, Preoperative lateral left carotid angiogram, early arterial phase, demonstrating large AVM located in the frontoparietal lobe of a 40-year-old man suffering from epilepsy. The AVM is supplied by dilated branches from the middle cerebral artery, and drains through cortical veins.

B and C, Postoperative lateral left carotid angiogram obtained on the day of surgery, late arterial (B) and early venous phases (C) showing complete removal of the AVM. The persistently dilated former feeders demonstrate sluggish arterial flow.

D and E, Postoperative left carotid angiogram obtained 28 days after the operation, lateral view, early arterial phase (D) and early venous phase (E). The former feeding arteries demonstrate reduction of their caliber (D), and no stagnating arterial flow (E).
ratio could be calculated from 44 out of 50 preoperative angiograms. Then, in order to estimate quantitatively the postoperative recovery of the caliber of a dilated feeding artery, we obtained the diminution ratio of the feeding artery by the following expression:

$$\text{Diminution ratio of feeder} = \frac{(A - B)}{A} \times 100 \%$$

where $A$ is the preoperative maximum diameter of the most dilated feeder, and $B$ is the postoperative maximum diameter of the same feeder. The diminution ratios were calculated from 54 of 69 postoperative angiograms.

A statistical analysis was performed using the $\chi^2$ test with the Yates corrections for expected values less than 10, as well as the Fisher exact probability test and the Student $t$ test.

**Results**

**Stagnating Arteries and Patient’s Age**

The mean age of the 30 patients with stagnating arteries was 38.3 ± 15.5 years (± 1 SD); whereas that of the 20 patients without such findings was 28.0 ± 15.1 years. The difference was statistically significant ($P < .05$), indicating that stagnating arteries are more likely to occur in older patients.

**Stagnating Arteries and Timing of Postoperative Angiograms**

Stagnating arteries were documented in 35 postoperative angiograms obtained from 30 patients. Twenty-three out of 29 angiograms obtained within 7 days after surgery revealed stagnating arteries, whereas only one out of 15 angiograms obtained 1 month or more after surgery showed stagnating arteries. Thus, it was found that the occurrence of stagnating arteries was significantly correlated with the time of the postoperative angiogram (Table 2). Table 2 also shows that stagnating arteries return to normal within 1 month after surgery in almost all cases.

**Stagnating Arteries and AVM Sizes**

As Table 2 shows, the sizes of the AVMs were associated with the incidence of stagnating arteries. In angiograms obtained 1 to 7 days after the operation, the incidence of stagnating arteries in large AVMs was significantly higher than that in small malformations ($P < .001$) (Table 2).

**Stagnating Arteries and Preoperative Degree of Dilatation of Feeders**

The preoperative FA/ICA ratio was 53.5 ± 17.0% (mean ± 1 SD, $n = 29$) in patients with stagnating arteries, as opposed to 43.1 ± 14.8% ($n = 15$) in those without stagnant arterial flow, the difference being statistically significant ($P < .05$). This result suggests that a markedly dilated feeder is an important causative factor of a postoperative stagnating artery.

**Discussion**

It has been suggested that early postoperative angiograms of large AVMs frequently reveal stagnant arterial flow in the feeding arteries that fed the excised malformations (1–4). According to intra- and postoperative hemodynamic studies by Hassler and Steinmetz (2, 3), stagnating arteries are characterized by high intra-arterial pressure and sluggish arterial flow. Clinically, these hemodynamic characteristics are thought to be associated with postoperative complications such as intracerebral hemorrhage and retrograde thrombosis (1, 3, 6), but we know of no discussion in relation to the pathophysiology of stagnating arteries.

Our findings on the relationship between stagnating arteries and feeder diameter diminution ratios suggest that normalization of sluggish arterial flow corresponds to postoperative recovery of size by dilated former feeding arteries. Except in very large vessels such as the aorta, arterial
blood flow shows the familiar parabolic velocity profile of laminar flow (2). In this flow pattern, the blood flow rate is expressed by the following formula:

\[ Q = \frac{1}{4} \pi r^2 \]

where \( Q \) is the blood flow rate, \( v \) is the mean flow velocity, and \( r \) is the vessel radius.

“\( Q \)” decreases rapidly after the removal of an AVM. In such a condition, as long as dilatation of the feeding arteries persists, “\( r \)” does not decrease. Therefore, a low value for “\( v \)” is obtained with this formula. This low velocity in former feeding arteries probably corresponds pathophysiologically to postoperative arterial stagnation.

Stehbens carried out a histologic study of chronic vascular changes in experimental arteriovenous fistulae (7). He found that histologic changes in the arteries proximal to the fistulae were similar to those of nonlipid-containing arteriosclerosis, and appeared to have been the direct result of induced hemodynamic stresses. In our study, the AVM feeders proximal to the nidus appear to have been affected by hemodynamic stresses, so arteriosclerotic changes similar to those described by Stehbens would be expected.

Although there is no agreement in the literature as to the effect of arteriosclerosis on the properties of the arterial wall, Farrar et al (8) found an increase in the hardening of the aorta of crab-eating macaque monkeys due to arteriosclerosis, and Pynadath and Mukherjee (9) observed a decrease in elasticity in the aorta of the rabbit, resulting from atherosclerosis. These results indicate that a decrease of elasticity may be observed in AVM feeders suffering arteriosclerotic changes.

These data suggest that postoperative development and persistence of stagnating arteries may be caused by delayed recovery of dilatation in feeders due to a decrease of elasticity. The present study shows that stagnating arteries are significantly correlated with advancing patient age, markedly dilated feeding arteries, and large AVM size. These results also suggest that pathologic changes in the AVM feeders due to long-standing hemodynamic stresses form a basis for the development and persistence of stagnating arteries.

In a study by Hassler, postoperative transcranial Doppler examinations revealed that the resistance index (peripheral vascular resistance) of former AVM feeders was noticeably higher than in normal vessels, and that an increase of the index lasted a few weeks after surgery (2). Increased peripheral vascular resistance must be considered as another possible explanation of the delayed recovery of dilatated feeding arteries.

Doppler sonographic examinations demonstrated that low flow velocity in feeding arteries persisted until the ninth postoperative day (2). This result correlates well with the large number of stagnating arteries seen in the postoperative angiograms obtained within 7 days after surgery (Table 2). Although it is still unknown how long-stagnating arteries persist in the postoperative angiograms (10), the present study indicates that such sluggish arterial flow returns to normal within 1 month after surgery in almost all cases (Table 2).

References