Evaluation of pressure changes in feeding arteries during embolization of intracerebral arteriovenous malformations

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The pressure in 47 arteries feeding 21 arteriovenous malformations (AVM's) was investigated during transarterial embolization using a Tracker-18 microcatheter. On average, systolic pressure increased by 22 mm Hg. In AVM's with single or few feeders, embolization was usually achieved well; in contrast, giant AVM's with multiple feeders and a large arteriovenous shunt were poorly embolized. However, large AVM's with well-demarcated components may be reduced by embolization to an appropriate size for surgery or stereotactic radiation therapy. It was found that the feeding artery pressure increased significantly more in well-embolized than in poorly embolized cases. Measurement of the feeding artery pressure clarified the hemodynamics of AVM's and facilitated more successful embolization.

Key Words: arteriovenous malformation • embolization • cerebral hemodynamics • normal perfusion pressure breakthrough syndrome

The hemodynamics and embolization of cerebral arteriovenous malformations (AVM's) are of increasing interest. Recently developed methods such as digital subtraction angiography, magnetic resonance imaging, and superselective angiographic catheterization have promoted a more dynamic diagnostic and therapeutic approach to difficult AVM's. In most cases, endovascular treatment reduces the size of an AVM to make it treatable by surgery or radiation therapy. However, hemorrhage, retrograde thrombosis, and migration of embolic materials into vessels supplying normal parenchyma remain major complications. The technical aspects of embolization are assumed to be the cause of most complications.20 It is imperative to assess both the volume of embolic material necessary to occlude the nidus and the end point of the procedure.

Several authors have noted that the pressure in arteries feeding the AVM is elevated considerably as obliteration is achieved.16,8-10,17 As suggested by Spetzler, et al.,24 phenomena resembling normal perfusion pressure breakthrough have been reported after surgery or embolization.11-12,21 In order to understand the hemodynamics of cerebral AVM's and the effects of endovascular therapy and to avoid complications, we measured pressure in feeding arteries before, during, and after embolization.

Clinical Material and Methods

From June, 1990, to March, 1992, pressure was measured in 47 intracranial feeding arteries in 21 patients with a cerebral AVM. A summary of patient characteristics, AVM size and location, feeding arteries, and draining veins is presented in Table 1. The mean age of the patients was 34 years (range 10 to 72 years); 15 were male and six were female. The major presenting symptom included hemorrhage in seven patients, seizure in seven, headache in six, and an ischemic attack in one.

The AVM's were classified as giant (> 60 cu cm), large (30 to 60 cu cm), medium (10 to 30 cu cm), or small (< 10 cu cm) using a volume index obtained by multiplying the length, width, and height of the AVM as measured from angiograms. Seven of the AVM's in this series were giant, six were large, three were medium, and five were small.

The pressure in feeding arteries was measured by a Tracker-18 microcatheter coupled to a transducer and a portable digital pressure monitor.9 Prior to the study, simulation with a pulsatile pump was used to compare pressures between the Tracker-18 microcatheter and other catheters to determine whether the former measured pressure correctly. In this simulation, the measured systolic pressure did not vary with the size of the catheter. However, the Tracker-18 microcatheter tended to measure a diastolic pressure that exceeded...
<table>
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<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Signs &amp; Symptoms</th>
<th>AVM Location</th>
<th>AVM Size (cm)</th>
<th>Feeding Arteries</th>
<th>Draining Veins</th>
<th>Stages of Embolization</th>
<th>Pressure Changes in Feeding Artery Pre/Post Embolization (mm Hg)</th>
<th>Obliteration Rate (%)</th>
<th>Group‡</th>
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<td>rt occipital</td>
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<td>PCA</td>
<td>ACV</td>
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<td>40/50 (38.8/45.9)</td>
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<td>2 43, M</td>
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<td>rt frontal</td>
<td>1.3 x 2.0 x 2.0</td>
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<td>ACV</td>
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<td>MCA, PCA</td>
<td>ACV</td>
<td>1</td>
<td>26/73 (17.2/47.7)</td>
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<td></td>
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<td>lt temporal</td>
<td>1.5 x 1.0 x 1.5</td>
<td>MCA</td>
<td>ACV</td>
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<td>rt temporoparietal</td>
<td>2.5 x 2.0 x 1.5</td>
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<td>vein of Labbé</td>
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<td>ACV</td>
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<td>3.5 x 3.6 x 4.0</td>
<td>MCA, PCA</td>
<td>ACV, vein of Labbé</td>
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<td>rt parietal</td>
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<td>MCA, ACA</td>
<td>ACV</td>
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<td>80/105 (58.8/85.4), 83/107 (61.5/83.6)</td>
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<td>lt temporoparietal</td>
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<td>vein of Labbé</td>
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<td>81/103 (63.2/82.4), 58/82 (47.5/73.9), 25/59 (20.5/51.3), 41/83 (28.7/56.5)</td>
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<td>bilat SCA</td>
<td>precerebellar vein</td>
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<td>lt parietal</td>
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<td>seizure</td>
<td>lt temporoparietal</td>
<td>3.0 x 4.0 x 3.0</td>
<td>MCA, PCA</td>
<td>ACV, vein of Galen, vein of Labbé</td>
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<td>seizure, migraine</td>
<td>rt temporoparietal</td>
<td>5.0 x 6.0 x 4.5</td>
<td>MCA, PCA</td>
<td>vein of Labbé, ACV</td>
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<td>46/67 (35.0/52.3), 55/66 (46.6/57.9), 78/91 (58.6/70.0)</td>
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<tr>
<td>15 20, F</td>
<td>ICH</td>
<td>lt frontal</td>
<td>4.0 x 4.0 x 4.5</td>
<td>ACA, MCA</td>
<td>ACV</td>
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<td>53/72 (47.7/62.1), 63/77 (55.8/66.4)</td>
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<td>rt temporal</td>
<td>4.0 x 5.0 x 3.2</td>
<td>MCA, PCA, ECA</td>
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<td>17 23, M</td>
<td>headache, chemoisis, exophthalmos</td>
<td>rt frontal</td>
<td>3.0 x 3.0 x 4.5</td>
<td>ACA, MCA</td>
<td>ACV, sylvian vein</td>
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<td>65/83 (53.7/65.4), 62/85 (52.1/69.7)</td>
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<td>18 14, M</td>
<td>seizure, headache, rt homonymous hemianopsia</td>
<td>lt temporoparietal</td>
<td>5.0 x 6.0 x 4.0</td>
<td>MCA, PCA, ACA, AChA, ECA</td>
<td>vein of Galen, ACV</td>
<td>4</td>
<td>81/86 (57.4/76.8), 45/66 (40.5/61.7)</td>
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<td>19 47, M</td>
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<td>MCA, PCA, ACA, ECA</td>
<td>vein of Rosenthal, ACV</td>
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<td>20 42, M</td>
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<td>rt frontoparietal</td>
<td>6.2 x 4.2 x 5.8</td>
<td>MCA, ACA, PCA, ECA</td>
<td>ACV</td>
<td>5</td>
<td>45/62 (33.6/45.9), 63/73 (49.6/55.7), 33/41 (26.2/38.7), 43/61 (42.6/47.3), 38/38 (31.7/33.0), 30/36 (25.0/33.3), 26/25 (22.4/24.3), 30/61 (23.9/47.3)</td>
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<td>21 42, M</td>
<td>seizure, headache, speech disturbance</td>
<td>rt temporoparietal</td>
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<td>MCA, PCA, ACA, ECA</td>
<td>vein of Rosenthal, ACV</td>
<td>5</td>
<td>43/51 (40.2/48.6), 45/62 (42.1/57.4), 53/56 (38.4/40.0), 52/77 (35.8/50.7), 52/57 (38.2/41.9)</td>
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* ICH = intracerebral hemorrhage; PCA = posterior cerebral artery; ACA = anterior cerebral artery; MCA = middle cerebral artery; ECA = external carotid artery; SCA = superior cerebellar artery; AChA = anterior choroidal artery; ACV = ascending cerebral vein.
† Values in parentheses denote ratio of pressure in a feeding artery to systemic blood pressure.
‡ See Fig. 7 for schematic illustration of groups.
Pressure changes in feeding arteries of AVM's

the actual one by about 10 to 20 mm Hg. This catheter is very soft and bends easily in tortuous vessels, nearly occluding the catheter lumen and thereby recording pressures that exceeded 150 mm Hg. This probably reflected pressure in the irradiation fluid bag, which was usually pressurized to 300 mm Hg.

After the Tracker-18 microcatheter was introduced, pressure was measured in each feeding artery before, during, and after embolization. Simultaneous measurements of systemic blood pressure were also made. The liquid embolic material Ethibloc† was used for the procedure;11,12,19 in contrast to other materials, Ethibloc adhered less to the microcatheter and was easier to inject repeatedly into vessels with the catheter maintained in the same position. Several injections of embolic material were needed to obliterate a large nidus (pressure was measured after each injection), whereas small feeding arteries were usually obliterated by one injection. In 17 cases arterial pressure was measured at various points in the middle cerebral artery (MCA), anterior cerebral artery, internal carotid artery (ICA), posterior cerebral artery (PCA), basilar artery (BA), and vertebral artery (VA) as the microcatheter was withdrawn after embolization. For control data, we measured pressure in the MCA and distal ICA of three patients with glioblastoma during superselective intraarterial chemotherapy.

Results

In the arteries feeding the AVM's, the average systolic pressure rose by about 22 mm Hg after embolization. The mean ratio of pressure in a feeding artery to systemic blood pressure rose by about 18% (Table 2). When several injections of embolic material were needed, the pressure increased after each injection as the nidus was gradually obliterated. A rapid and significant rise in pressure indicated nearly complete angiographic obliteration of the nidus. The pressure did not always increase as much in giant AVM's.

Pressure was recorded in the distal ICA, MCA, PCA, BA, and VA after embolization; arterial pressure gradually decreased distally (Fig. 1 left). The ratio of segments M₁ through M₃ of the MCA or P₁ through P₃ of the PCA to systemic pressure ranged from 60% to 70%; in the glioblastoma cases, this ratio was generally higher than in AVM cases (75% to 85%, Fig. 1 right), probably because the latter patients had no significant arteriovenous (AV) shunt.

Illustrative Cases

Case 6

This 40-year-old man had a right temporal AVM (Fig. 2 left). After 0.2 ml of Ethibloc was injected into the nidus, the pressure in the feeding artery immediately increased by 29 mm Hg (Fig. 2 right). The AVM was then occluded completely by adding another 0.1 ml of Ethibloc.

<table>
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<th>Degree of Embolization</th>
<th>No. of Cases</th>
<th>Factor</th>
<th>Pre-Embolization</th>
<th>Postembolization</th>
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</table>

† Ethibloc manufactured by Ethicon, Gmbh, KG, Norderstedt-Hamburg, Germany.
FIG. 2. Angiograms in Case 6, a patient with a right temporal arteriovenous malformation (AVM). Left: Superselective preoperative angiogram demonstrating the AVM nidus fed by the middle cerebral artery branch (the posterior temporal artery). Right: Angiogram after injection of 0.2 ml of Ethibloc showing subtotal obliteration of the AVM. The feeding artery pressure increased immediately by 29 mm Hg.

Case 21

This 42-year-old man had a giant right temporo-occipital AVM. In this case the microcatheter tip may have been initially situated in a small branch of the main feeder because the measured pressure was 150 mm Hg (much higher than expected in this position) and the pulse pressure was very small. Angiography showed a dense vessel in the AVM (Fig. 3 left), and we concluded that the catheter was not well positioned for embolization. Because it was dangerous to inject embolic material directly into a fine vessel, the catheter was withdrawn slightly and the pressure decreased to 50 mm Hg. The dense vessel was then not visible on angiography (Fig. 3 right).

Case 20

This 42-year-old man had a giant right frontoparietal AVM. The feeding artery pressure was very low, probably because this AVM was composed of a large AV shunt and many fistulae (Fig. 4). In an AVM such as this, with multiple feeding arteries, it was difficult to obliterate the nidus effectively, even after several stages of embolization. A number of large fistulae leading to the varix were documented during surgery.

Discussion

With the advances in microsurgical techniques, stereotactic radiation therapy, and endovascular therapy, previously inaccessible AVM's can be
Pressure changes in feeding arteries of AVM's

Fig. 4. Superselective angiogram in Case 20 showing a giant right parietal arteriovenous malformation. The feeding artery pressure was very low.

Fig. 5. Graphs showing results in well- and poorly embolized cases. Values are expressed as the mean ± standard error of the mean. Left: Systolic pressure changes in feeding arteries during embolization. Right: Ratio of feeding artery pressure to systemic blood pressure.

treated more effectively. Alone or in combination with the above-described techniques, AVM embolization should produce complete obliteration or resection with no neurological deficit. Therefore, preoperative embolization becomes much more important in cases of large or giant AVM's and in AVM's in areas of the cortex that control critical functions such as speech.

Pre- and postoperative cerebral blood flow in AVM patients has been studied using thermodilution, xenon computerized tomography, and single-photon emission computerized tomography. Pressure and velocity in feeding arteries have been measured by microvascular Doppler sonography and at direct puncture during surgery. With the advent of microcatheters, intravascular pressure can be measured both in an experimental model and in human vascular malformations, thus establishing the reliability of measuring pressure with this system. In our simulation, the agreement between small and large catheters was better for the measurement of systolic pressure than diastolic pressure.

In AVM's, there is a low-resistance pathway and high flow. In glioblastoma cases, the pressure in the MCA was higher than in AVM cases (Fig. 1). With more AV shunt flow, the arterial pressure becomes lower.

Extent of Embolization

Arteriovenous malformations can be classified as well or poorly embolized. Well-embolized AVM's may be obliterated completely or reduced to a size allowing treatment by surgery or stereotactic radiosurgery. With both embolization and surgery, the feeding artery pressure increases significantly as a vessel is occluded. If any residual nidi remains in the embolized artery, the pressure will not increase significantly. As the residual AV shunt becomes smaller, the pressure gradually increases.

In this study, well-embolized AVM's were usually small or medium in size, with an average pressure elevation after embolization of 27 mm Hg (Fig. 5, Table 2). Poorly embolized AVM's were usually giant ones with multiple feeding arteries. In these cases the average pressure increased by only 11 mm Hg (Fig. 5). This change suggests a large AV shunt through other feeding arteries, some of which increased in size after embolization. Jungreis, et al., suggested that pressure changes can be measured before they can be seen on fluoroscopy. A feeding artery should not be embolized to the extent that it prevents overflow into the normal vessels.

Pressure Changes and Systemic Blood Pressure

The pressure in feeding arteries usually changes in relation to the systemic blood pressure. In Case 9, the feeding artery (MCA) was much larger than the normal M1 trunk, which was small and had few branches (Fig. 6). From the beginning of embolization, the MCA pressure remained high and, after obliteration, the pressure of the M1 segment increased to nearly the pressure of the ICA. There may be a higher risk of brain swelling and/or hemorrhage than is usually seen with AVM embolization because the few poorly developed branches of the MCA were suddenly overloaded with a large proportion of the blood flow. In such cases, the feeding artery should be embolized slowly, while its pressure is continuously measured to avoid any abrupt increase of blood flow into the brain parenchyma that surrounds the AVM; at the same time, systemic blood pressure changes must be watched closely.

With giant and diffuse AVM's, it is often difficult to identify each feeding artery on a preoperative angiogram. In these cases, after superselective angiography, the catheter tip may migrate or become wedged into small vessels even though the catheter was placed in the proper position. Such wedging may predispose to vessel rupture during the injection of contrast media or embolic material (Fig. 3).

Classification of AVM's

Luessenhop and Gennarelli mentioned anatomical grading of supratentorial AVM's according to their operability. However, the grading system proposed by Spetzler and Martin in 1986 is useful for determining
operative indication and difficulty. This grading system considers size, location, and pattern of venous drainage. In our experience, the feeding artery is a more important factor than the venous drainage pattern in treatment by embolization. Therefore, we have categorized the AVM's in this study according to their response to embolization (Table 1).

After investigating changes in AVM feeding artery pressures during embolization, we divided the AVM's into four groups (Fig. 7). Group 1 was composed of small or medium-sized AVM's with a single or few feeding arteries that could be satisfactorily embolized. In this group the feeding artery pressure immediately and significantly increased after only one or two injections of embolus. Group 2 comprised large AVM's with several feeding arteries, detected angiographically, and a single main draining vein. Group 3 consisted of a few independent foci of minimally connected AVM's with separate feeding arteries and separate draining veins. The pressure in these feeding arteries increased progressively as the nidus was obliterated. Group 2 and 3 AVM's could be obliterated well by staged embolization, and the pressure in their feeding arteries increased remarkably after embolization. Group 4 comprised giant AVM's with entangled feeding arteries that varied in size, length, and structure. This type of AVM receives a great amount of blood and shunt flow and usually has a large fistulous component. It is often difficult to discern feeding arteries on an angiogram. Even a superselective angiogram may not show feeding arteries clearly because excessively rapid flow quickly dilutes contrast medium. Under such conditions it is also difficult to identify transit vessels. Because of the large AV shunt, the feeding artery pressure is lower than in other AVM's. It is difficult to obliterate the nidus adequately in these giant AVM's in spite of multiple attempts because there are so many pathways for blood flow. If several feeding arteries are obliterated at one stage, others soon receive a large blood flow and a shunt develops again. As a result, feeding artery pressure does not increase as much as in smaller AVM's, which makes it quite difficult to obtain good obliteration as long as multiple AV shunt pathways exist.

Conclusions
It is usually possible to predict from an angiogram whether embolization of an AVM will be difficult. The measurement of feeding artery pressure helps to clarify the immediate hemodynamic changes in AVM's, thereby making the embolization of AVM's more successful.

References

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**FIG. 6.** Internal carotid angiogram in Case 9 demonstrating a large right parietal arteriovenous malformation. The large feeding artery from the middle cerebral artery (MCA) is much larger than the normal M1 trunk of the MCA. The feeding artery pressure was very high, probably because the MCA received blood flow directly from the internal carotid artery.

**FIG. 7.** Schematic illustration of the four types of arteriovenous malformations (AVM's) in this study. Group 1: the AVM is fed by a few arteries. Group 2: the AVM is large, well demarcated, and fed by several arteries. Group 3: the AVM is large and comprises a few independent niduses that have different feeding arteries and draining veins. Group 4: the AVM is giant and has extremely tangled feeding arteries that vary in size, length, and structure (diffuse type).

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