Clinical Evaluation of Artificial Embolization in the Management of Large Cerebral Arteriovenous Malformations*

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Presented here is an interim appraisal of the potential clinical usefulness of artificial embolization in the surgical management of certain large cerebral arteriovenous malformations. In prior communications we have stated a rationale for this procedure, discussed observations of the responses of cerebral arteries to plastic emboli and described a technical modification permitting controlled catheterization of the proximal cerebral arteries. Here we will describe certain anatomical features of the lesions relating to artificial embolization and give a detailed account of our clinical experience to date. This includes observations in 15 patients, from a series of 55, in whom a total of 620 emboli were introduced.

Anatomy of Arteriovenous Malformations

The possibilities for embolization are dependent upon certain anatomical changes associated with these malformations. Since the introduction of angiography, it has been shown repeatedly that to many of the large hemispheric lesions there are enlarged arterial channels leading directly from the cervical arteries. In 1957, Hamby published a gross microscopic dissection of a hemispheric malformation indicating that these enlarged arteries terminate at the malformation by ramifying into multiple smaller interconnecting arteries before passage into a series of tortuous venous channels. This suggested that large emboli within the feeding arteries would arrest at this site which is the vascular origin of the malformation. Because the arteries to the normal surrounding brain remain of normal caliber, or smaller, a large difference in diameter between these and the feeding arteries exists. Therefore emboli sufficiently small to traverse the length of the feeding arteries could at the same time be too large for entrance into these smaller normal arteries.

For purposes of embolization we classify the large hemispheric lesions anatomically within the framework of the normal cerebral arterial anatomy. This is based upon the 55 cases referred to us since initiation of this study. By serial angiography it is usually possible to separate three distinct components of vascular alteration. These are:

1. Primary abnormality of direct artery to vein shunt.

2. Secondary enlargement of the feeding arteries.


Angiography demonstrates that the arteries and veins involved are those that normally supply and drain this vascular zone. Furthermore, the anastomotic relationship of the feeding arteries is similar to that of the normal cerebral collateral circulation, as has been described by Van der Eecken.

Normally, there is a paucity of collateral connections between adjacent cortical branches arising from each of the primary divisions of the internal carotid artery. In arteriovenous malformations this is also true, for small cortical arteries, adjacent to the feeders, pass around the malformation with no apparent participation.

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Multiple leptomeningeal arteries form the normal collateral connections of the anterior, middle and posterior cerebral arteries. When the anterior middle and posterior cerebral arteries jointly contribute to a malformation, they do so through the leptomeningeal arteries which are increased in size but not in number.

Usually there are very limited collateral connections between the penetrating arteries over the surface and the lenticulostriate arteries at the base. In arteriovenous malformations these arteries participate jointly only when the zone of abnormal shunting extends between the territories they normally supply.

The anatomical characteristics of the draining veins have been described by Kaplan et al.⁹ The cone-shaped appearance of the larger malformations conforms to the anatomical configuration of the transcerebral veins which normally join the superficial and deep venous systems but in arteriovenous malformations are transformed into much larger venous channels. When a single deep vein assumes the major burden of drainage it may enlarge into an aneurysmal sac (venous aneurysm). This is more frequently seen in the great vein of Galen or veins adjacent thereto.

The prototypes of the classification we use are shown in Fig. 1.

In Type A the lesion occupies the basal ganglia, internal capsule and thalamus and is supplied by multiple, enlarged lenticulostriate arteries. The main trunk arteries bypass the lesion. Malformations of this type cannot be treated by embolization and direct surgical attack is not reasonable.

Type B is supplied by surface arteries and small penetrating arteries. Embolization can eliminate only the surface artery contribution. However, this may be of clinical value in certain instances as suggested by one of our patients whose progressing hemiparesis was arrested for 1½ years following embolization which accomplished only this much.

Malformations of Type C are the most favorable for embolization. However, when the lesions are large there is always some contribution from one or more enlarged lenticulostriate arteries to the medially extending apex thereby precluding complete elimination of the lesion. Also the leptomeningeal anastomotic arteries from the anterior cerebral artery are potential feeders after elimination of the middle cerebral contribution. In our 3 cases with this type of lesion angiography revealed renewed filling of a portion of the lesion via these routes 1 to 3 years after embolization.

In type D, which is equally supplied by anterior and middle cerebral arterial branches, there is a good chance for elimination of the middle cerebral contribution but the anterior cerebral poses a problem. Although it is desirable that the emboli seek the anterior and middle routes equally, our observations indicate a division of about 10 to 1 in favor of the middle cerebral. The reasons for this will be discussed below.

We have not, as yet, attempted to embolize malformations of Type E in which the entire angiographic contribution is from both anterior cerebral arteries. Although these arteries may become fully as large as the middle cerebral trunks, in most of our cases this has not sufficiently altered the angles of branching at the bifurcation of the internal carotid artery to offset the more direct continuation into the middle cerebral trunk.

Occipital malformations conforming to Type F must be embolized via the basilar system. Our single experience with this will be described below.

Of the arteries proximal to the malformation, there are 2 sites of major importance in embolization. These are the internal carotid artery bifurcation and the division of the middle cerebral trunk into 2 or more principal Sylvian arteries. The most frequent alterations in arterial diameters and configurations secondary to malformations occurring at these sites are shown in Figs. 2 and 3.

Most commonly the middle cerebral trunk, in direction and size, is the anatomical continuation of the internal carotid artery; the anterior cerebral artery is half as large and arises at an acute angle. When the