Treatment of Cerebral Arteriovenous Malformations by Muscle Embolization

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Cerebral arteriovenous malformations present a difficult therapeutic problem. The methods that have been used for their treatment are: symptomatic medical therapy, x-ray therapy, superficial electrocoagulation, ligation of the arterial feeding vessels, and partial or total resection of the malformation. Although non-surgical methods have been only palliative, even surgical procedures have not solved the problem adequately except when the angiomata has been rather small and more or less peripherally situated. Total resection is not always feasible because of possible severe neurological sequelae, especially when the malformation is large or is situated in regions of specialized cortical function.

Luessenhop, et al., have devised and successfully used embolization with small plastic spheres of variable size according to the caliber of the feeding arteries. Plastic emboli are introduced into the internal carotid through the previously cut external carotid artery, with radiographic and angiographic control of the location of the radiopaque spheres.

On the other hand, Brooks in 1931 seems to have been the first to use free muscle embolization through the internal carotid for the treatment of carotid-cavernous fistulae; this idea was further developed by Hamby and Gardner, Jaeger, and others. This procedure involves intracranial occlusion of the internal carotid and, when possible, the ophthalmic artery, before using opacified muscle emboli to occlude the orifice of the carotid-cavernous communication.

We are convinced that muscle is superior to plastic spheres, first, because it is well tolerated by the brain and, second, because it can be obtained during the operation in limitless quantities and in all desirable sizes. We have used embolization on four patients with arteriovenous malformations, introducing free fragments of unopacified muscle through the external carotid into the internal carotid artery in accordance with Luessenhop’s technique. No other intracranial maneuver was carried out, in the belief that the embolus would reach the malformation because of its hemodynamic activity (diminished peripheral resistance) and finally stop at the origin of its feeding vessels.

This report presents these first four cases, two of them which are considered as to be clinically “cured” and the other two “improved” as they await further embolization.

Case Reports

Case 1. A 40-year-old woman was admitted on May 17, 1967, because of progressive right-sided headaches, a burning sensation in the left arm, and severe vertiginous attacks over the last 5 years. When she was 14 years old, she had suffered a sudden episode of left hemiplegia, vomiting, and loss of consciousness for several hours, with partial slow recovery from the hemiplegia.

Examination. There was a left homonymous hemianopsia, a left central facial paresis, a left spastic hemiparesis with distal paralysis, hyperreflexia, ankle clonus, and Babinski sign, a left hypalgesia and hypoesthesia, a diminished vibration sense and abolished position sense in the left foot, a moderate atrophy of the left limbs, and an intracranial bruit audible over the left eye and temporal region that increased with contralateral compression of the carotid artery and ceased with homolateral compression. The blood count, blood glucose and urea, and urinalysis were normal. The serology test for syphilis was negative.

Skull x-rays showed a deep right intracerebral calcification. The electroencephalogram showed absence of alpha rhythm on both sides and constant theta activity from the right hemisphere. Carotid angiography showed a deep frontoparietal arteriovenous malformation fed by branches of the middle
First operation. On June 8, 1967, a muscle embolus (3 × 3 mm) was introduced into the carotid artery. Postoperative angiography showed no changes in the malformation. On the second day after this embolization, the patient stated that the burning sensations in her left arm had stopped; however, the intracranial bruit remained as audible as before.

Second operation. On June 12, under local anesthesia, a muscle embolus (9 × 5 mm) was introduced into the carotid artery. Postoperative angiography (Fig. 1B) showed important reduction in the size of the malformation, occlusion of an abnormally enlarged anterior choroidal artery, and filling of the anterior cerebral artery which had not been visualized preoperatively.

Immediately after the second embolization, the intracranial bruit was no longer audible on the left side unless the contralateral carotid was compressed. Within 24 hours, the patient experienced greater motility at the proximal joints and an improved gait, although the distal paralysis persisted as before.

Third operation. On June 23, six muscle emboli (3 × 3 mm each) were introduced into the vessel. Angiography showed a further decrease of the malformation's size (Fig. 1C).

By the third postoperative day, motion had improved further, and new slight flexion movements of the left fingers and toes were visible, while the sensibility to all modalities had become practically normal. On the other hand, the central facial paresis had increased, and there was for the first time lingual paresis.

Six months after the third embolization, the patient has had no recurrence of right-sided headache or left-sided burning sensations; motility and strength retains the same degree of partial recovery, and sensibility is normal. The intracranial bruit can be heard only with contralateral carotid compression.

Case 2. A 23-year-old man was admitted on August 10, 1967, because of 5 years of progressively severe seizures involving the right arm and face plus residual right hemiparesis.