Mechanism of Experimental Muscle Embolization of the Carotid Cavernous Fistula and the Fate of the Emboli

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Complications or failure in the treatment of carotid cavernous fistulas with cervical or combined cervical-intracranial carotid ligation4-6,8,10,12,16 have mainly been due to the presence of the collateral circulation of the internal carotid artery, namely, the cerebral communicating branches, the ophthalmic artery, or branches of the cavernous carotid segment.9,10 The intraluminal embolization method of Brooks,1,2 advocated by Lang and Bucy,9 is simple and effective but has not been used widely because of the fear that the muscle or its fragments may pass into the cerebral circulation and obstruct the distal arterial segment. The possibility of this complication led many surgeons4,13,14 to clip the internal carotid artery intracranially before embolization. This procedure itself was not always simple or without risk.7,8 Since there has been no report on definite complications in patients treated by embolization, its failure4,11,15 seems to be due to the use of improper muscle embolus. However, Wanissorn, et al.,17 using a muscle embolus slightly larger than the caliber of the common carotid artery alone, cured four consecutive cases of carotid cavernous fistula.

The purpose of this investigation was to study the mechanism of muscle embolization of the carotid-cavernous fistula and the fate of emboli, particularly the role of intracranial ligation of the internal carotid artery.

Experimental Method

The experiments were carried out on 48 autopsied human cadavers, within 18 hours after death, aged between 10 and 68 years. The brains were removed, leaving the intracranial carotid arteries as long as possible; the dura mater over the cavernous sinus together with related cranial nerves and surrounding tissues was removed in order to expose the cavernous carotid segment. The cervical carotid arteries were also dissected, and the external carotid arteries ligated. The common carotid arteries were then cut at about 5 cm below the bifurcations and connected to water pumps which applied pressure rhythmically, simulating the living circulation (Fig. 1). The following experiments were then performed:

1. Each of the various shapes and sizes of muscle emboli obtained from the sternomastoid was inserted into the common carotid artery to find out the optimal shape and size of the muscle embolus.

2. The muscle emboli of optimal shape and size were separately inserted into each carotid artery, which had previously been fistulized at its cavernous portion. The size of the fistula of each artery varied from 1 mm upwards.

3. After each successive embolization (Step 2 above) the pressure of the water injected was increased until the fistula recurred, in order to study the fate of the muscle emboli.

Results

Carotid Arteries and Muscle Emboli. The diameter of every intracranial internal carotid artery was less than half of that of its cavernous portion, which was in turn less than half of that of its own common carotid artery.

Muscle emboli smaller than the cavernous carotid artery could be propelled quite easily through the entire carotid artery.

Muscle emboli in block shape, of the same size as the common carotid artery or a little larger (about 1½ the size of the common carotid), obstructed the artery almost

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completely at its beginning. They were propelled gradually, at each pulsation, through the entire course of the artery. They stopped for a while at two sites: first, at the base of the skull where the artery entered the carotid canal; and second, at the clinoid region where the artery turned backward and its diameter became acutely reduced. In the cavernous portion, the muscle was elongated and compressed by the wall of the artery. The latter was distended, appearing sausage-like. No water was observed to leak through the intracranial carotid end. The emboli occupied the cavernous portion about 1½ to 2 cm in length.

Muscle emboli, more than twice the size of the common carotid artery and accommodated to the artery by massage, could be propelled through the internal carotid artery. They occupied more than 3 cm of the cavernous portion, which was too long. Therefore, muscle emboli about the size of the common carotid artery or a little larger was considered to be optimal, for it obstructed the main circulation early, occluding the cavernous carotid segment tightly but not too long.

**Optimal Muscle Emboli and the Fistularized Arteries.** Regardless of the size of the fistulous opening, a part of the muscle embolus bulged out and expanded immediately on reaching the opening, while the main part remained within the lumen of the cavernous carotid segment. The muscle incarcerated in the fistulous opening acquired a dumb-bell shape (Figs. 2 B and 3 B). The bulging part acted as a hook holding the embolus in place. The embolus blocked the artery as well as the fistula tightly, and there was no movement unless significantly higher pressure was applied.

**Fate of the Muscle Emboli.** These emboli may be classified into three categories:

1. The muscle emboli stopped at the fistulous opening; with higher pressures they slipped upward into the distal cavernous carotid segment and stopped there (Fig. 2). This occurred when the fistulous openings were too small (less than 2 mm). On increment of pressure, the bulged part first appeared slightly larger while the intraluminal part was gradually migrating upward into the distal cavernous carotid segment. Finally, when the intraluminal part of the embolus was pushed beyond the fistulous opening, the bulged part was drawn back into the lumen to follow its main part, and stopped just beyond the fistula.

2. The muscle emboli stopped at the fistulous opening but with higher pressures were expelled through the fistulous opening (Fig. 3). This was found when the fistulous openings were smaller than the diameter of the cavernous carotid segments. The bulging part appeared gradually bigger and bigger, while the intraluminal part migrated a little upward into the distal cavernous carotid segment. Finally the whole muscle embolus escaped through the fistulous opening.
Fig. 2. Embolization of a small fistula and fate of the muscle embolus. A. Before embolization  B. Successful embolization; the embolus incarcerated in the fistulous opening acquired a dumb-bell shape. C. and D. Fate of the embolus on increment of pressure, the embolus stopped just beyond the reopened fistula (D).

3. The muscle emboli passed easily through the fistulous opening. This condition was found when the fistulous openings were larger than the caliber of the cavernous carotid segments.

Discussion

A muscle embolus smaller than the caliber of the artery moved freely within the arterial lumen, but a muscle embolus larger than the caliber of the artery was different; there were many factors that made the muscle embolus migrate. Besides the pulsation of the elastic arterial wall, the compressibility and accommodation of the muscle played an important role. The pulsating action of the circulation together with the taper and smooth inner arterial tube would squeeze the compressible muscle embolus forward at each pulsation.

In case of fistularized arteries, the site of the fistula was the very place where the artery had no resistance. A small as well as a large muscle embolus would plug the fistula. The small embolus obstructed only the fistula, leaving within the arterial lumen a loose segment which might be subsequently fragmented by a patent circulation. On the contrary, a large muscle embolus obstructed the fistula as well as the arterial lumen perfectly. There was no circulation through the embolized arterial segment. The muscle embolus could not be pushed forward easily because the bulging part held it in place.

The large muscle emboli never passed beyond the distal part of the fistularized internal carotid artery no matter whether the fistula was small or large.

If the fistula was small (Fig. 2), the embolus might be pushed with pressure high enough to overcome the holding action of the bulging part of the embolus; when the muscle moved into the distal cavernous carotid segment, the thrust of the circulation was diverted through the reopened fistula, and the embolus automatically stopped there. This phenomenon may happen only in experimental conditions. In live patients, the

Fig. 3. Embolization of a large fistula and fate of the muscle embolus. A. Before embolization. B. Successful embolization as in Fig. 2 B. C. and D. Fate of the embolus on increment of pressure; the embolus was expelled through the fistulous opening (D).
large muscle embolus inserted in the proximal carotid lumen will block the main circulation, and simultaneously the collateral circulation from the distal segment will be diverted down into the fistula. This diverted circulation will thrust and direct the embolus to plug the fistulous opening and be fixed there. After the fistula has been plugged, the retrograde pressure from above still persists, acting against the embolus and helping the hook action of the bulging part of the embolus. On the other hand, the acute reduction in caliber of the internal carotid artery which occurs in its supraclinoid portion more or less assists in arresting the embolus.

If the fistula was large (Fig. 3), the embolus could only be expelled through the fistulous opening into the cavernous sinus, which contains plenty of fibrous trabeculations. These trabeculations are expected to keep the embolus from slipping through the fistulous opening.

On the basis of this investigation, ligation of the intracranial internal carotid artery should not be a primary adjunct in the treatment with large muscle embolization. If one fears that the muscle may be fragmented or slip through the fistula, early ligation of the cervical carotid artery is recommended. This is performed after first checking that the fistula has been plugged, either by x-ray films or by clinical examination to see if the bruit over the affected eye has ceased.

If the embolus does not stop even temporarily at the fistulous opening, as reported by Sedzimir, et al., it is likely that not only the fistulous opening is too large but the cavernous sinus is so dilated that its trabeculae are too weak to slow up the embolus. In such cases, treatment with embolization could not cure the condition but does no harm because the embolus simply lodges in the cerebral vein or tributary of the cavernous sinus. However, this method of embolization is still valuable as a diagnostic procedure for it gives information on the condition of the fistula and the cavernous sinus. Treatment by other means such as a trapping procedure or direct transcavernous repair should also be considered.

Summary

The mechanism of muscle embolization and the fate of muscle emboli were studied in 48 autopsied human cadavers in which the cavernous carotid arteries had been artificially fistulized. A block of muscle slightly larger than the caliber of the common carotid artery was considered to be optimal, for it obstructed the fistula as well as the cavernous carotid segment perfectly. The fistula and the cavernous carotid segment were embolized mainly by the hook action of the part of the embolus that bulged through the fistulous opening.

Large muscle emboli never passed beyond the distal internal carotid arteries of the fistulized arteries, therefore, occlusion of the intracranial internal carotid artery need not be a primary adjunct. Embolization of the carotid cavernous fistula with a large muscle embolus will do no harm. It was not only a therapeutic procedure but a diagnostic tool to indicate the condition of the fistula and the cavernous sinus in case the embolization failed.

Ligation of the cervical carotid artery may be performed after successful embolization.

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References

9. Parkinson, D. Surgical approach to the


