

Middle cerebral artery embolectomy

Case report

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✓ A patient who developed an embolic occlusion of the right middle cerebral artery while undergoing a cerebral arteriogram was successfully operated on by removal of the embolus under the surgical microscope. Early postoperative cerebrovascular spasm was a factor in the transient deterioration of the patient's neurological condition. When the patient was last seen 2½ months after surgery she was almost intact neurologically with only a mild right parietal dysfunction but with total resolution of the left hemiplegia. The literature is reviewed.

KEY WORDS • middle cerebral artery embolectomy • cerebral embolism • cerebrovascular spasm • intracranial arteriotomy

DIRECT surgical treatment of occlusive intracranial vascular disease, either embolic or thrombotic, is far from well established as a treatment of choice. We were able to find only 35 cases reported in the English literature. We will report a case of middle cerebral artery embolectomy and review the previous reports on this subject.

Case Report

A 30-year-old right-handed woman was admitted to the New England Medical Center Neurology Service on July 27, 1974, for evaluation of chronic headaches of one year's duration.

Examination. Neurological examination was normal. Her previous medical history was negative except for estrogenic hormone replacement since November, 1973, following a hysterectomy. Brain scan and skull x-ray films were negative.

On July 29, 1974, she underwent retrograde femoral cerebral arteriography. The right common carotid artery was catheterized first. Prior to injection of the dye, the patient became lethargic and developed a flaccid left hemiplegia. At this point dye was injected and showed an occlusion of the origin of the right middle cerebral artery (Fig. 1). Neurosurgical consultation was obtained and approximately 2 hours later she was taken to surgery.

Operation. A small frontotemporal flap was used to open the Sylvian fissure to expose the main trunk and branches of the middle cerebral artery. The embolus appeared to be lodged at the bifurcation and also extended into the two main branches of this artery (Fig. 2 *upper left*). The embolus had moved distally when compared to its position as seen on the initial arteriogram. Two arteriotomies were made, one in each of the branches, very close to their origin from the middle cerebral artery trunk (Fig. 2 *upper right*). The emboli were removed and the arteriotomies were closed

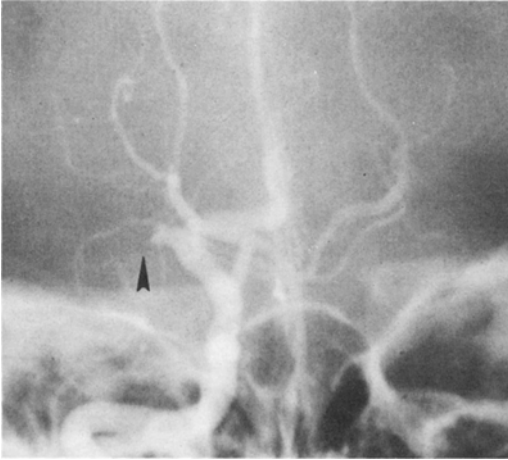


FIG. 1. Preoperative angiogram showing complete occlusion of the proximal trunk of the right middle cerebral artery (*arrowhead*).

with five to six sutures of 9-0 monofilament nylon (Fig. 2 *lower left*).

The operation was done under magnification with the microscope. The patient's blood pressure was kept artificially elevated with

neosynephrine in the 130 to 150 systolic range. She also received a high O₂ concentration. From the time of occlusion to the time when the arteriotomies were finished a period of 6 to 7 hours had elapsed.

Postoperative Course. On awakening from surgery she had some response to pain in her left extremities, the arm being more sensitive than the leg. She continued to improve gradually each day. Four days after surgery the patient's condition deteriorated over a few hours with increased lethargy and decrease in the strength of the left side. A right brachial retrograde arteriogram showed considerable spasm throughout the carotid and vertebrobasilar circulation (Fig. 3 *left*). There was also evidence of a moderate degree of cerebral edema as shown by a shift of the anterior cerebral artery and internal cerebral vein toward the left side. It was difficult to say if the arteriotomies were open because of the spasm. There was retrograde perfusion of the middle cerebral artery territory via leptomeningeal anastomosis. At that point Decadron was increased and the patient's blood pressure artificially elevated. At no

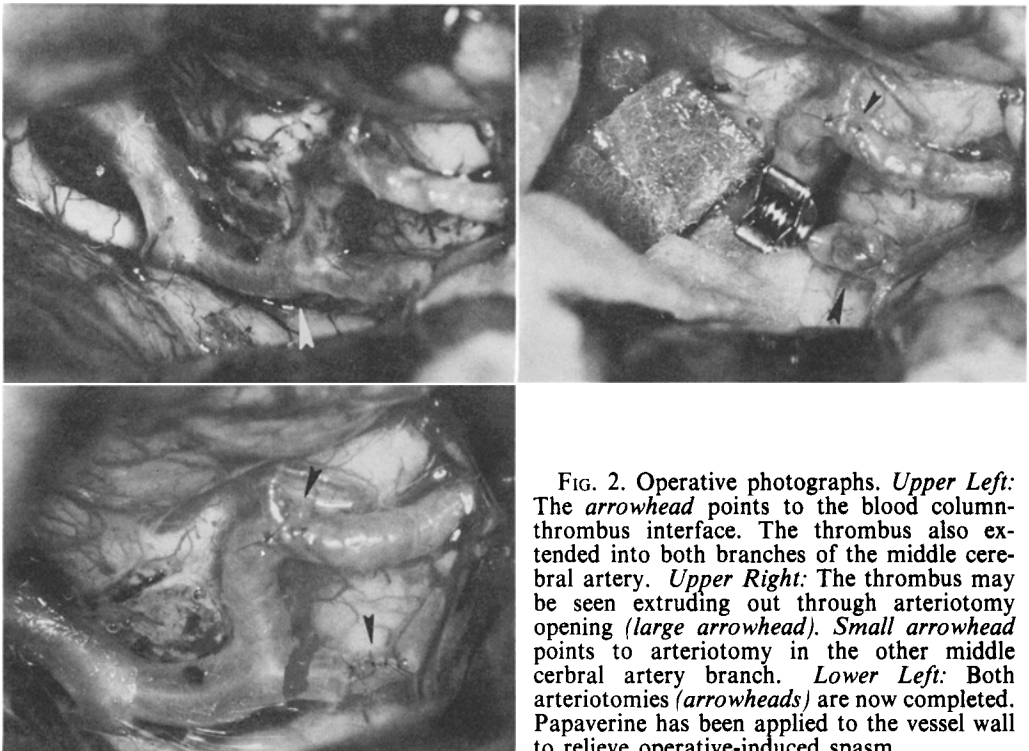


FIG. 2. Operative photographs. *Upper Left:* The *arrowhead* points to the blood column-thrombus interface. The thrombus also extended into both branches of the middle cerebral artery. *Upper Right:* The thrombus may be seen extruding out through arteriotomy opening (*large arrowhead*). *Small arrowhead* points to arteriotomy in the other middle cerebral artery branch. *Lower Left:* Both arteriotomies (*arrowheads*) are now completed. Papaverine has been applied to the vessel wall to relieve operative-induced spasm.

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time during her hospital stay was she given anticoagulants.

She subsequently made a progressive recovery. Follow-up 2½ months later showed that the patient had regained excellent motor strength and almost normal skilled movements on her left hand. There was a mild parietal lobe syndrome with inattention to the left side and decreased graphesthesia in her left hand.

A repeat right brachial arteriogram 5 months postoperatively showed the right middle cerebral artery, both main trunk and branches, to be patent. There was no evidence of collateral blood supply at this time (Fig. 3 right).

Discussion

Thirty-five other cases of intracranial arteriotomy for embolus or thrombosis have been reported in the English literature (Table 1). Eighteen out of 37 vessels operated on were patent as demonstrated on postoperative arteriograms. There was no direct evidence to indicate that those patients whose arteries were opened did better than the others whose arteries remained occluded following surgery. Postoperative intracranial hemorrhage occurred in two cases.^{9,11} In one case⁹ the com-

plication was felt to be secondary to anticoagulation therapy. Both of these patients died. Some of the patients had their operation within a few hours after the occlusion and others several days or even weeks later.

One patient developed widespread postoperative vasospasm very similar to that of our case.⁷ The cause of occlusion was embolic in the majority of cases although in some patients disease of the vessel wall and thrombosis appeared to be the primary problem. There were eight deaths in the postoperative period for a 20% mortality. Seventeen patients were improved and only one worsened postoperatively. Most of the operations were performed with the surgical microscope but some of the earlier ones were done without the microscope.

Cerebral embolism seems to carry a high mortality and morbidity. The embolus can travel to any cerebral vessel but most commonly the middle cerebral artery is involved. Carter² reported on the natural history of cerebral embolism. He collected 34 cases of his own and reviewed several other series previously reported. The cases were not broken down according to which vessel was involved. The mortality rates varied between 21% to 54% for an average mortality of 36%. In addition, a large number of patients were

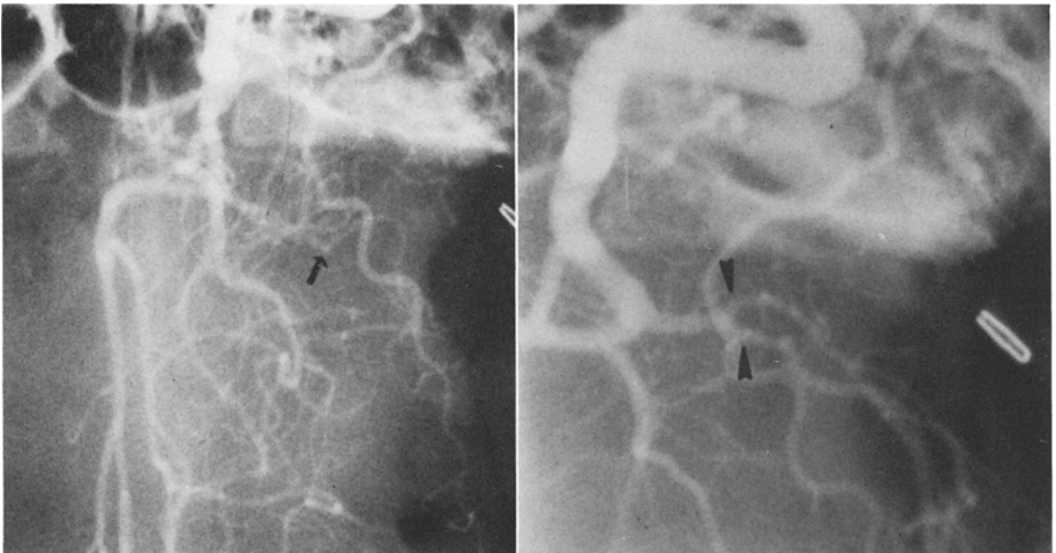


FIG. 3. *Left:* Right brachial arteriogram 4 days after surgery shows moderate degree of spasm of intracranial internal carotid artery, anterior cerebral, and proximal middle cerebral arteries. There is no significant filling of the middle cerebral artery beyond arteriotomy site (*arrow*). *Right:* Right brachial arteriogram 2½ months after surgery. The spasm has now resolved and both arteriotomies are patent (*arrowheads*).

TABLE 1

Results in 35 cases of intracranial arteriotomy reported in the English literature

Author, Year	No. of Cases	Postoperative Vessel Patency*	Improved	Unchanged	Worse	Deaths
Welch, 1956	2	1†	1	1	0	0
Jacobson, <i>et al.</i> , 1962	2	0	2	0	0	0
Chou, 1963	1	1	1	0	0	0
Donaghy, 1967	7	2	3	1	0	3
Lougheed, 1967	2	0	1	1	0	0
Shillito, 1967	6	4‡	?	1	?	3
Sundt, <i>et al.</i> , 1967	2	1	1	0	0	1
Yaşargil, 1969	11	7	6	3	1	1
Khodadad, 1973	1	1	1	0	0	0
Zlotnik, 1975	1	1	1	0	0	0
total	35	18	17	7	1	8

* Some patients did not have postoperative angiograms.

† Vessel partially opened.

‡ Two patients had two arteries operated on.

left with severe deficits (18% average). Some of the patients died from recurrent embolism and others from their primary disease that gave origin to the embolism or from other conditions. However, about one third to one fourth of the patients died as a direct result of the initial embolic insult.

Lhermitte, *et al.*,¹¹ studied the causes of ischemic accidents in the middle cerebral artery territory. They found that atherosclerosis could be incriminated only in 16.6% of cases; embolism of various sources appeared to be responsible for 40% of middle cerebral artery occlusions; 35% of the cases remained of undetermined etiology.

The prognosis in cases of middle cerebral artery occlusion, according to Lascelles and Burrows,¹⁰ is poor. They found in a series of 59 patients, followed for various periods of time, that 42% either were severely disabled or died as a direct result of the middle cerebral artery occlusion. Allcock¹ found different results on his series of 40 patients with occlusion or marked stenosis of the middle cerebral artery where only 5% of patients died and 72% were either normal or able to return to useful life. Allcock¹ performed serial angiograms on ten patients. In five of these, the follow-up angiograms showed normal vessels, in three patients the appearance was better, and in two cases there were no changes seen when compared with the original angiograms.

Dalal, *et al.*,⁶ reported seven cases of em-

bolic occlusion of intracranial arteries which were subjected to serial follow-up angiography over a period of several days to several weeks post-embolism. In every case, the follow-up angiograms showed reopening of the occluded vessel. None of the patients, however, showed any improvement of their neurological deficit.

Much experimental work has been done on middle cerebral artery occlusion.^{4,7,18,15-19} It appears, from experimental work in monkeys, that cerebral infarction does not develop immediately after occlusion of the vessel, but rather develops slowly over a matter of several hours. Cerebral edema caused by ischemia appears to aggravate the already existing ischemia by decreasing the collateral blood flow and, therefore, the process becomes self-perpetuating.¹⁵

Harvey and Rasmussen⁷ carried out temporary and permanent occlusion of the middle cerebral artery in a series of monkeys. To produce a degree of motor impairment similar to that seen on permanent occlusions it was necessary to occlude the middle cerebral artery for at least 50 minutes. No pathological changes were seen in occlusions that lasted less than 30 minutes. Sundt, *et al.*,¹⁵ found that occlusion of the middle cerebral artery in monkeys did not cause infarction consistently unless the artery had been occluded for 3 to 6 hours. Crowell, *et al.*,⁴ showed that occlusion of the middle cerebral

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artery in the monkey may be tolerated for several hours with negligible infarction. Occlusions that lasted from 6 to 24 hours were followed almost uniformly by large infarcts. It seems from the experimental results that re-establishment of flow by surgical means within a few hours after middle cerebral artery occlusion might avoid cerebral infarction.

Middle cerebral artery embolectomy and thrombectomy are presently at a very early stage of development and no definite conclusions can be made as to the indications and benefits of such procedures. A review of the prognosis of cerebral embolism and middle cerebral artery occlusions indicates that the ideal surgical candidate for this procedure is a young person in good general medical condition, in whom the embolism or thrombosis has occurred only a few hours prior to surgery.

References

1. Allcock JM: Occlusion of the middle cerebral artery: serial angiography as a guide to conservative therapy. *J Neurosurg* 27:353-363, 1967
2. Carter AB: Prognosis of cerebral embolism. *Lancet* 2:514-519, 1965
3. Chou SN: Embolectomy of middle cerebral artery. Report of a case. *J Neurosurg* 20:161-163, 1963
4. Crowell RM, Olsson Y, Klatzo I, et al: Temporary occlusion of the middle cerebral artery in the monkey: clinical and pathological observations. *Stroke* 1:439-448, 1970
5. Dalal PM, Shah PM, Sheth SC, et al: Cerebral embolism: angiographic observations on spontaneous clot lysis. *Lancet* 1:61-64, 1965
6. Donaghy RMP: Patch and bypass in micro-vascular surgery, in Donaghy RMP, Yaşargil MG (eds): *Microvascular Surgery*. Stuttgart: Georg Thieme (US dist: CV Mosby), 1967, pp 75-86
7. Harvey J, Rasmussen T: Occlusion of the middle cerebral artery. An experimental study. *Arch Neurol Psychiatry* 66:20-29, 1951
8. Jacobson HJ, Wallman LJ, Schumaker GA, et al: Microsurgery as an aid to middle cerebral artery endarterectomy. *J Neurosurg* 19:108-114, 1962
9. Khodadad G: Middle cerebral artery embolectomy and prolonged widespread vasospasm. *Stroke* 4:446-450, 1973
10. Lascelles RG, Burrows EH: Occlusion of the middle cerebral artery. *Brain* 88:85-96, 1965
11. Lhermitte F, Gautier JC, Derouesné C, et al: Ischemic accidents in the middle cerebral artery territory. A study of the causes in 122 cases. *Arch Neurol* 19:248-256, 1968
12. Loughheed WM: The surgery of intracranial vascular occlusion, in Donaghy RMP, Yaşargil MG (eds): *Microvascular Surgery*. Stuttgart: Georg Thieme (US dist: CV Mosby), 1967, pp 142-147
13. Meyer JS: Circulatory changes following occlusion of the middle cerebral artery and their relation to function. *J Neurosurg* 15:653-673, 1958
14. Shillito J Jr: Intracranial arteriotomy in three children and three adults, in Donaghy RMP, Yaşargil MG (eds): *Microvascular Surgery*. Stuttgart: Georg Thieme (US dist: CV Mosby), 1967, pp 138-142
15. Sundt TM Jr, Grant WC, Garcia JH: Restoration of middle cerebral artery flow in experimental infarction. *J Neurosurg* 31:311-322, 1969
16. Sundt TM Jr, Waltz AG: Cerebral ischemia and reactive hyperemia: studies of cortical blood flow and microcirculation before, during, and after temporary occlusion of middle cerebral artery of squirrel monkeys. *Circ Res* 28:426-433, 1971
17. Sundt TM Jr, Waltz AG: Experimental cerebral infarction: retro-orbital, extradural approach for occluding the middle cerebral artery. *Mayo Clin Proc* 41:159-168, 1966
18. Sundt TM Jr, Waltz AG, Sayre GP: Experimental cerebral infarction: modification by treatment with hemodiluting, hemoconcentrating, and dehydrating agents. *J Neurosurg* 26:46-56, 1967
19. Waltz AG, Sundt TM Jr: Effect of middle cerebral artery occlusion on cortical blood flow in animals. *Neurology* 16:1185-1190, 1966
20. Welch K: Excision of occlusive lesions of the middle cerebral artery. *J Neurosurg* 13:73-80, 1956
21. Yaşargil MG: *Microsurgery Applied to Neurosurgery*. Stuttgart: Georg Thieme, 1969, pp 101-105
22. Zatz LM, Iannone AM: Cerebral emboli complicating cerebral angiography. *Acta Radiol [Diagn]* 5:621-630, 1966
23. Zlotnik EI: Thrombectomy of the middle cerebral artery. Case report. *J Neurosurg* 42:723-725, 1975

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