

Emergency Carotid Endarterectomy for Patients with Acute Carotid Occlusion and Profound Neurological Deficits

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Emergency revascularization procedures for patients with acute stroke are controversial. Thirty-four patients with acute internal carotid artery occlusion documented at the time of emergency endarterectomy were analyzed. Before operation, all these patients had profound neurological deficits including hemiplegia and aphasia. There was a 94% success rate in restoring patency. In follow-up, nine patients (26.5%) had a normal neurological exam, four (11.8%) had a minimal deficit, 10 (29.4%) had a moderate hemiparesis, which was improved over their preoperative deficit, 4 (11.8%) remained hemiplegic, and seven (20.6%) died. The natural history of patients with acute carotid occlusion and profound neurological deficits is dismal. In comparison, 13 patients (38%) made a dramatic recovery. The surgical mortality rate compares favorably with the natural history. Good collateral flow was a good prognostic factor, while a simultaneous middle cerebral artery embolus was associated with a poorer prognosis. An emergency carotid endarterectomy may be indicated in selected patients with acute internal carotid artery occlusion with profound neurological deficits. Full preoperative angiography may identify those patients who would benefit from surgical intervention and reduce the operative mortality rate.

EMERGENCY CAROTID ENDARTERECTOMY to restore flow in patients with acute occlusion producing profound ischemic neurological deficits has generally been considered contraindicated. The lack of proven benefit, the proposed high incidence of secondary hemorrhage, and the high perioperative mortality are the basis for this opinion.¹⁻¹² A few authors suggest that if the operation is to be contemplated, flow must be restored within 1 hour from the onset of the ictus.^{9,13-15}

However, the natural history of patients with acute carotid occlusion and severe ischemic deficits is grave.¹⁶ Review of the neurological literature demonstrates that 2-12% will make a good recovery, 40-69% will be disabled by severe neurological handicaps, and 16-55% will die from infarction.¹⁷⁻²² There is now good experimental ev-

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idence supporting the concept of a critical period of reversible ischemia in which compromised neurons may be salvaged.²³⁻²⁶ The brain's tolerance to oligemia appears to be a function of both the severity and the duration of ischemia. Therefore, the extent of collateral flow following an acute carotid occlusion may provide the opportunity for reversal of the ischemic process if blood flow can be restored. In a recent report,²⁷ we analyzed 20 cases of emergency middle cerebral artery embolectomy performed for acute profound neurological deficits. In that study, a significant number of patients, seven (35%), made a dramatic recovery after restoration of flow, supporting the concept of reversible ischemic neuronal damage. This prompted us to investigate our surgical results in patients with acute carotid occlusion and profound neurological deficits who were treated aggressively with an emergency endarterectomy to restore flow. Furthermore, it is of importance to examine the information provided by clinical experience regarding the experimental concepts of tolerated periods of vessel occlusion and reversible cerebral ischemia.

Methods

Case Material

From January 1973 to November 1984, 2036 carotid endarterectomies were performed by the neurovascular surgical services at the Mayo Clinic. Out of these, 34 cases were emergency endarterectomies for acute occlusion with profound neurological deficits. Thirty-one were stable deficits and three were progressing deficits. There were 20

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males and 14 females with an average age of 68. Thirty-three patients were already hospitalized for the evaluation of cerebrovascular insufficiency, and one patient was first evaluated in the Emergency Room. In 20 patients, there was occlusion of a known high-grade internal carotid artery stenosis: 10 of these patients were off heparin awaiting elective endarterectomy, eight were on therapeutic levels of heparin, one had a documented heparin induced hypercoagulopathy, and one had polycythemia vera. In nine patients, occlusion occurred within 1 hour of cerebral angiography, eight of these being performed by the direct carotid puncture technique. In five patients, the acute neurological deterioration was due to postendarterectomy occlusion of the internal carotid artery. Fifteen patients had preoperative angiography to confirm occlusion, while, in the other 19 patients, the diagnosis of acute carotid occlusion was made on the basis of history and clinical findings (loss of bruit and a decrease in retinal artery pressure). The average follow-up was 5.4 years (range: 1–10 years).

Definitions

A. Profound ischemia. The acute onset of severe hemispheric neurological deficits including aphasia, hemiplegia, often altered sensorium, and conjugate deviation of the eyes. The ictus was attributed to an acute carotid occlusion, usually of a previously angiographically documented or clinically suspected internal carotid artery stenosis. One patient included in this group had acute severe loss of amplitude on electroencephalogram during the opening of an elective endarterectomy and was found to have an acute carotid occlusion after exposure.

B. Progressive ischemia. The patients in this group had the acute onset of ischemic symptomatology, which progressed to complete hemiplegia within 2 hours. The symptoms were progressive and nonfluctuating.

C. Carotid occlusion. All cases had intraoperative documentation of the acute occlusion with fresh thrombus causing obliteration of the lumen. There were no observed intimal dissections. Several cases had zero flow on the baseline intraoperative xenon¹³³ cerebral blood flow study, although this was not routinely done on an emergency basis.

D. Postoperative outcome. Excellent—defined as an entirely normal neurological examination without residual deficits. Good—defined as a minor deficit on examination; *i.e.*, minimal monoparesis, a decrease in fine finger movements, or a slight anomic aphasia. These patients were independent and employable. Fair—defined as a moderate hemiparesis or aphasia. This deficit was better than their preoperative deficit. These patients were independent. Poor—defined as a marked residual deficit making this individual incapable of independent living.

Surgical Technique

The surgical technique has been fully described elsewhere.^{28,29} In these cases, 33 had placement of a shunt after the thromboendarterectomy. When possible, intraoperative electroencephalogram and xenon¹³³ cerebral blood flow studies were used, although the emergency nature of these cases precluded their use in most situations.

Intraoperative angiography was performed at the surgeon's discretion for two reasons: poor backflow following thromboendarterectomy or to document vessel patency. Out of the 14 intraoperative angiograms performed, six were positive for a middle cerebral artery embolus, five of whom had an emergency embolectomy after completion of the endarterectomy.

The Fogarty catheter, either #3 or #4, was utilized to enhance thrombus removal in 10 cases. In one additional case, a distal internal carotid artery dissection secondary to the use of this catheter may have been the cause of an unsuccessful operation. Three out of the nine documented cases of a middle cerebral artery embolus occurred in patients in whom the Fogarty catheter was utilized.

In 33 patients, the arteriotomy was repaired with a saphenous vein patch graft.

Illustrative Case

The patient was a left-handed, 67-year-old woman with a history of left hemispheric transient ischemic attacks (TIAs) in the 3 months prior to admission on 3/22/82. Her episodes consisted of right arm and facial weakness and expressive aphasia occurring in an upright position lasting 2–3 minutes. In the week prior to admission, she had experienced four TIAs. Her admission neurological exam was remarkable for venous stasis retinopathy O.U., bilateral carotid bruits L > R, and bilateral decreased retinal artery pressures L > R. Significant risk factors included hypertension, obesity, and a 20 pack/year smoking history. After a normal uncontrasted CT scan, she was administered intravenous heparin with a PTT of 70. On 3/23/84 at 4:30 A.M., she had the acute onset of a flaccid hemiplegia involving her face, arm, and leg and a dense aphasia. She was unable to follow 1 step commands. This episode was more severe than her typical TIA, did not resolve, and was associated with a loss of her previously noted left carotid bruit. The patient underwent an emergency transfemoral cerebral angiogram (Fig. 1 left), which demonstrated occlusion of the left internal carotid artery and a 95% stenosis of the right internal carotid artery. The left internal carotid had reconstitution of its intracavernous portion through the ipsilateral ophthalmic artery with filling of the ipsilateral middle cerebral artery. There was also embolic occlusion of a posterior parietal branch of the middle cerebral artery. Since her neurological status was unchanged 3 hours after the onset of the ictus, she underwent a simultaneous endarterectomy and embolectomy. The time between the onset of ischemic symptoms and successful restoration of flow through both the internal carotid and middle cerebral arteries was 8½ hours. Six hours after operation, she began to demonstrate voluntary movements of both her arm and leg, followed 2 step commands, and had partial reversal of her aphasia. Postoperative angiography (Fig. 1 right) demonstrated full restoration of flow. Seven months after surgery, her neurological examination showed no motor weakness, a mild anomic aphasia, and decreased stereognosis of her right hand. A repeat angiogram at that time demonstrated an asymptomatic occlusion of the right internal carotid artery despite Coumadin® therapy. Currently, she is an active housewife and mother.

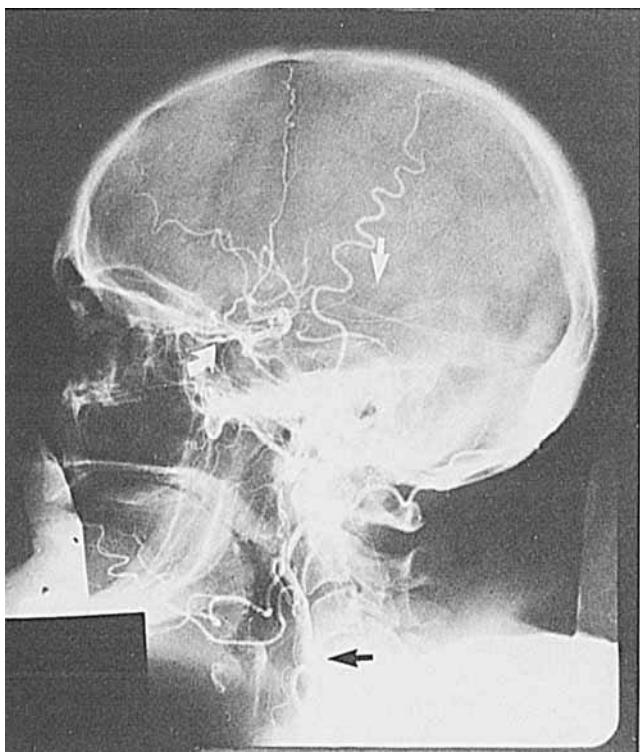


FIG. 1. *Left*. Internal carotid artery occlusion (black arrow). There is reconstitution of the intracavernous carotid through the ophthalmic artery (curved arrow) with filling of the middle cerebral artery complex except for posterior parietal branch (white arrow). *Right*. Full restoration of flow through both the internal carotid artery (large arrow) and through the middle cerebral artery complex (small black arrow).

Results

Table I lists the follow-up neurological examinations in these 34 cases. Nine patients (26.5%) returned to normal, four (11.8%) made a good recovery, 10 (29.4%) made a fair recovery, four (11.8%) had a poor result, and seven (20.6%) died. Therefore, 13 patients (38.3%) did extremely well, considering their profound preoperative neurological deficits, and a total of 23 patients (67.6%) improved after operation. Table 2 lists the outcome as related to the etiology of occlusion.

Thirty-two patients (94%) had a technically successful operation in which flow was restored. In the two cases in which flow could not be restored, death due to cerebral infarction occurred. One patient developed the syndrome

of postoperative cerebral hyperemia with a postocclusion xenon¹³³ cerebral blood flow of 120 cc/minute, over two times the normal flow. This patient was initially worse with cerebral edema and paroxysmal lateralizing epileptiform discharges (PLEDS). However, both resolved to give a long-term good result.

Out of the seven deaths, two were hemorrhagic infarctions, the time for flow restoration being 4½ hours and 6 hours. One of these hemorrhages was perhaps influenced by the need to have the patient on heparin on postoperative day #3 for multiple pulmonary emboli and brachial thrombosis.

Figure 2 compares the outcome in terms of the duration of internal carotid artery occlusion. There appears to be no good correlation between time of oligemia and out-

TABLE 1. *Follow-up Neurological Examinations*

Preoperative Deficits	Outcome				
	Normal	Good	Fair	Poor	Dead
Progressive	2		1		
Severe	7	4	9	4	7*
Total	9 (26.5%)	4 (11.8%)	10 (29.4%)	4 (11.8%)	7 (20.6%)

* Causes of death: hemorrhagic infarctions, 2; infarctions, 4 (two unsuccessful operations); pulmonary embolus, 1.

TABLE 2. *Outcome*

	Normal	Good	Fair	Poor	Dead
Occlusion of a carotid stenosis	5	3	8	0	4
Postangiographic occlusion	2	0	2	3	2
Postendarterectomy occlusion	2	1	0	1	1

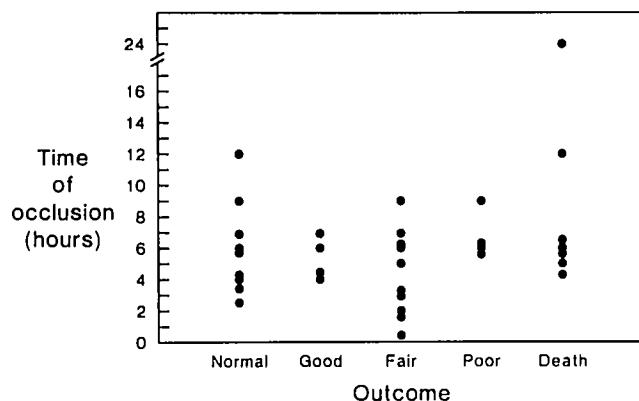


FIG. 2. Neurological outcome as a function of duration of carotid occlusion (onset of ictus to time of flow restoration *via* arterial repair or placement of shunt). Time of no flow through the internal carotid artery was not a good predictor of outcome.

come. Furthermore, many operations were performed after 6 hours without producing a hemorrhagic infarction.

Figure 3 demonstrates the poorer prognosis in nine patients with a documented middle cerebral artery embolus in association with the carotid occlusion. A simultaneous embolectomy did not improve the overall outcome. The one patient with an embolus who made a good recovery had occlusion of just the posterior parietal branch and not the main trunk of the middle cerebral artery. Of the 14 cases in which intraoperative angiography was performed, emboli were demonstrated in six, five of whom underwent an emergency embolectomy. In two patients, emboli were inferred by the CT finding of multiple infarcts within the anterior circulation, while in one patient the diagnosis of embolus was made by history alone. This patient had acute occlusion during the opening of a routine elective endarterectomy manifested by severe electroencephalographic changes. Although the time between EEG changes and placement of a shunt was just 25 minutes, this patient only made a fair recovery.

Figure 4 correlates the outcome in terms of collateral flow as ascertained from the preoperative angiograms. In 29 patients, there was sufficient preoperative angiographic data to determine if there was good collateral flow during the occlusion, while in five patients unilateral carotid angiography made this impossible. Support for good collateral consisted of the following: angiographic evidence of collateral to the ipsilateral middle cerebral artery through the ipsilateral external carotid-ophthalmic route, through the contralateral anterior cerebral-anterior communicating route, or through the ipsilateral posterior communicating artery. In the five cases of occlusion of an elective endarterectomy, intraoperative xenon¹³³ cerebral blood flows and electroencephalography were used to monitor the initial surgery. During these elective cases, an occlusion cerebral blood flow was done prior to the arteriotomy to

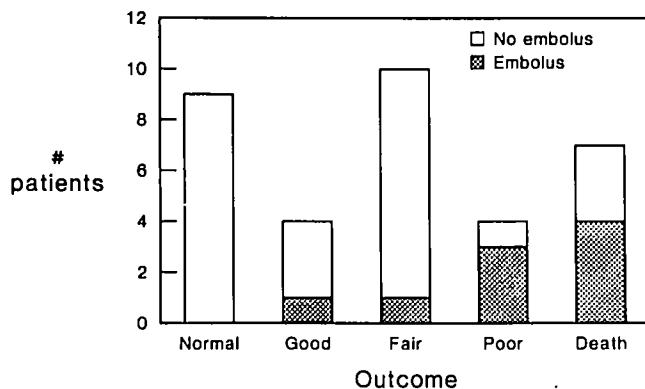


FIG. 3. Neurological outcome as a function of an associated middle cerebral artery embolus. Patients with both a carotid occlusion and middle cerebral artery embolus had a poorer prognosis. The one patient who made a good recovery had an occlusion of a parietal branch and underwent an embolectomy (see illustrative case). In the other five patients who underwent an embolectomy, there was occlusion of the proximal middle cerebral artery. These patients either died from severe infarction or hemorrhage or did poorly.

determine if a shunt would be required during the elective endarterectomy. These occlusion cerebral blood flow studies would therefore closely approximate the expected blood flow at the time of occlusion during the emergency endarterectomy. In these five cases, the cerebral blood flow studies confirmed the assessment of either good or poor collateral flow on preoperative angiograms.^{28,29} Although the surgeon routinely noted carotid backflow after the emergency thromboendarterectomy, this was not used in the classification except when the observation of either good or poor backflow collaborated with the angiographic criteria.

Figure 4 demonstrates that there was a good correlation between outcome and collateral flow. With good collateral flow there was a better surgical result, while with poor collateral flow the prognosis was poor. In the two patients

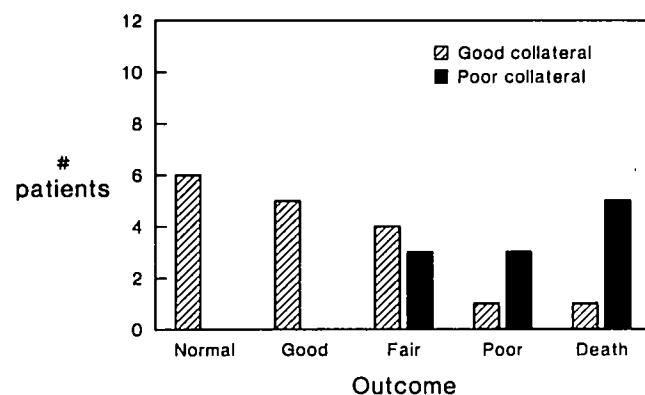


FIG. 4. Neurological outcome as a function of collateral flow as judged by preoperative angiograms. Collateral flow was associated with a good prognosis, while patients with poor collateral had a poor prognosis.

with good collateral in the poor and death outcome groups, one was associated with an embolus and one with an intracerebral hemorrhage.

Discussion

Surgical Literature

Wylie et al. in 1964,¹ presented nine cases of patients with recent strokes who underwent a carotid endarterectomy, out of which five had subsequent intracerebral hemorrhage. It is imperative to note that these patients were operated on 3 to 9 days after the ictus. Hunter in 1965² reviewed 26 patients with either carotid stenosis or occlusion. In the four patients with carotid occlusions, three had a poor surgical outcome. The Joint Extracranial Arterial Occlusive Disease Study Part IV³ reviewed 50 cases of patients with an acute carotid occlusion with neurological deficits. Although the postoperative mortality rate was 42%, the time between occlusion and operation ranged from 0-13 days.

Thompson in 1967⁴ discussed 71 patients with frank stroke, out of which 34 were described as profound. There were six (17.6%) surgical deaths. In 1970,⁵ he further analyzed 75 cases defining "frank stroke" as a neurological deficit that was either stable, improving, or worsening. The time between onset of stroke and operation was not well specified. After operation, 15 (20%) were normal, 33 (40%) were improved, 17 (27.7%) were unchanged, and five (6.7%) were worse. He concluded that it was rare for a patient with a profound deficit to improve and that there was an unacceptably high surgical mortality rate with this group.

Najafi et al. in 1971,¹³ analyzed 32 patients undergoing emergency surgery for acute neurological deficits: 10 patients had carotid stenosis, 15 patients had postangiographic carotid occlusion, and seven had postendarterectomy occlusion. In the postangiographic group, eight (53%) improved, three (20%) were unchanged, four (27%) did poorly, and one (6%) died. In the postendarterectomy group, three (43%) improved, two (28%) were "unchanged", two (28%) did poorly, and two (28%) died. Since a better prognosis was associated with a short occlusion time, they concluded that restoration of flow must be accomplished within 1 hour for a successful outcome.

In their review of carotid endarterectomy, Ojemman et al.⁶ discussed 21 cases of acute carotid occlusion in patients with either TIAs or stroke in evolution. Fifteen patients had a technically successful operation, out of which eight (53%) had marked improvement, four (27%) improved, two (13%) were unchanged, and one (6.5%) died. The six patients with a technically unsuccessful operation did worse, with only one showing improvement. They concluded that patients with a sudden profound deficit, especially if there was an altered sensorium, should not be operated on.

Kwaan et al. in 1979,¹⁴ reported on three patients with acute occlusion after an elective endarterectomy. With successful restoration of flow, two had complete resolution and one partial reversal of their neurological deficits. The time between occlusion and flow restoration was 20-45 minutes in these cases. They also discussed six additional cases in which restoration of flow was delayed for longer than 3 hours because of angiography. In these cases, two died and four remained hemiplegic. Therefore, they concluded that the operation must be performed within 1 hour and recommended no angiogram for the purpose of saving time.

Hafner et al. in 1981,⁷ reported on three patients with acute stroke, all of whom improved after emergency endarterectomy to restore flow. However, they recommended that no patient should be operated on after 2 hours or if they had an altered consciousness. DeWeese in 1982¹¹ reported on 19 patients with progressive strokes secondary to either carotid stenosis or occlusion treated with an emergency endarterectomy. Nine (47%) patients were worse and eight (37%) died after operation. He therefore recommended that no patient with profound deficits should be considered a surgical candidate. Finally, Donaldson et al.¹⁵ recently reported on six patients with carotid occlusion, three of whom presented with TIAs and three with hemiparesis. In the latter group, two improved and one was unchanged in follow-up.

There is general agreement that patients with fluctuating deficits due to acute carotid occlusion are good candidates for an emergency endarterectomy. Papers by Goldstone and Moore,³⁰ and Mentzer et al.,³¹ are examples of the improvement in patients with fluctuating deficits as compared to the natural history. Furthermore, the ability to re-establish flow if the endarterectomy is performed early after occlusion³² is well documented.

Therefore, review of the literature yields controversial views on the benefits of surgery, partially due to the lack of a well-defined preoperative neurological status, the documentation of occlusion *versus* stenosis, and the timing of surgery.

Natural History

The natural history of patients with acute carotid occlusion with profound neurological deficits is poorly defined in most studies because of the lack of angiographic documentation of carotid occlusion or a short follow-up. In 1961, McDowell¹⁷ reviewed 40 patients with angiographic or autopsy proven internal carotid artery occlusion. Twenty-two presented with severe neurological deficits, out of which one made a good recovery, nine (41%) were permanently disabled, and 12 (55%) died from the ischemic insult. The follow-up in this study was 2 years. Millikan in 1973,¹⁸ reported on 204 cases of stroke in evolution of the carotid system. However, the diagnosis

was made by history and examination without angiographic documentation, and the follow-up was only 2 weeks. He noted that 12% were normal, 5% were monoparetic, 69% were hemiparetic, and 14% died. In 1976, Jones and Millikan¹⁹ reported on 179 patients with carotid territory infarctions by examination and history but again without angiographic documentation. They found that if the patient was hemiplegic with an altered consciousness, 18 out of 44 (41%) died, but if there was normal consciousness, the mortality was only 2%. They also noted that if hemiplegia developed within 3 hours and persisted for 36 hours, there was a 90% chance of a permanent incapacitating motor deficit.

Giro and Patterson²⁰ reported on 44 patients with angiographic documented acute carotid occlusion, out of which seven (16%) died in the first 2 weeks secondary to infarction. However, in this group, 15 patients presented with TIAs, 23 with a completed stroke, and three with stroke in evolution.

Oxbury et al.²¹ reported on 93 patients with carotid territory infarction without angiographic confirmation of occlusion. Fifteen patients (16%) died in the first 3 weeks secondary to infarction.

Norrvig et al.²² reported on 59 patients with acute carotid occlusion, 54 angiographically documented and five by autopsy. Nine patients (16.6%) died in the acute stage. Unfortunately, there was no information as to the outcome of the survivors.

Therefore, if we collate the above reports recognizing their deficiencies, 2–12% make a good recovery, 40–69% have a profound deficit, and 16–55% will be dead in follow-up. In our surgical results, 26.5% were normal, and 11.8% made a good recovery with minimal deficits. Therefore, 13 patients (38%) did remarkably well and better than what the reported natural history would have predicted. A mortality rate of 20.6% compares favorably with the natural history. Therefore, it appears that an emergency endarterectomy for acute occlusion may salvage some patients without increasing the overall mortality rate. Since collateral flow on the preoperative angiogram was a good prognostic factor, while an associated middle cerebral artery embolus was a poor prognostic factor, full preoperative angiography may enhance the surgical success rate while decreasing operative mortality by preoperative identification of these factors.

Experimental Correlations

The physiological and functional integrity of neurons is dependent on the delivery of substrates for energy metabolism. The net result in oligemia is equivalent to the severity and duration of reduced flow divided by tissue vulnerability.²⁷ These three factors, namely, occlusion time, reduction of flow, and tissue vulnerability, have all been evaluated experimentally.

Multiple studies by many researchers including Sundt,^{25,33,34} Crowell,³⁵ Watanabe,^{36,37} Dujovny,³⁸ and Morawetz³⁹ using various animal models have demonstrated a permissible period of temporary occlusion of the middle cerebral artery in the production of focal ischemia. This range extends from 2 hours to 7 hours, depending on the animal model used, in which temporary occlusion will be tolerated without infarction. Furthermore, most of these studies suggest that restoration of flow after the tolerated time of occlusion increases the likelihood of hemorrhagic infarction presumably due to increased permeability secondary to ischemic damage to the endothelium. Clinically, 6 hours has been traditionally proposed as the time of permissible middle cerebral artery occlusion in humans after which infarction is likely to occur. As noted in the introduction, several authors have stated that if an endarterectomy is to be considered it must be performed within 1 hour. Although most experimental studies with middle cerebral artery occlusion are models of focal ischemia, the basic element of time of no flow or reduced flow is the same. However, as demonstrated in Figure 2, the time of no flow through the internal carotid was not a good prognostic factor. Furthermore, many of the operations were done after 4–6 hours without producing a hemorrhagic infarction. Therefore, time of no flow is a less important factor in the criteria for surgical selection.

The second element is the level of reduced flow that can be tolerated. Analysis of human and experimental models suggests that there are two thresholds of cerebral ischemia—the thresholds of electrical failure and membrane failure. Normal cerebral blood flow is approximately 50 ml/100 g/min.⁴⁰ Sundt^{28,41} and Trojanborg⁴² demonstrated in man a change in the electroencephalogram if total cerebral blood flow fell below 16 to 18 ml/100 g/min. Branston et al.⁴³ further showed in the baboon a change in evoked somatosensory potentials if flow fell below 15 ml/100 g/min. This range of flow from 18 ml/100 g/min to 15 ml/100 g/min has been termed the flow threshold of electrical failure and appears to be relatively uniform in various animal models.⁴⁴ The second threshold of membrane failure or pump failure occurs at about 10 ml/100 g/min, which experimentally is demonstrated by an increase in extracellular potassium secondary to a lack of ATP for the $\text{Na}^+ - \text{K}^+$ pump.⁴⁵⁻⁴⁷ Other metabolic abnormalities also occur, including an increase in intracellular calcium,⁴⁴ the liberation of free fatty acids,²⁶ and a profound intracellular acidosis.²⁴ The assumption is that membrane failure is irreversible cell damage. Between the threshold of electrical failure and membrane failure exists a potential zone of reversible cell damage—the “ischemic penumbra.” Our surgical results support the concept of an ischemic penumbra. Since all these patients had severe neurological deficits, they were presumably below the threshold of electrical failure. However, 13 patients (38%)

made a remarkable recovery implying that what flow existed after the carotid occlusion maintained neurons above the threshold of membrane failure. This best explains why collateral flow was an important prognostic factor. Presumably, collateral flow maintained a percentage of neurons within the ischemic penumbra, thereby enhancing the salvaging effects of an emergency carotid endarterectomy. In addition, this also explains the poorer results when there was an associated middle cerebral artery embolus, since an embolus would significantly decrease potential collateral flow. This confirms our previous report in which an associated ipsilateral carotid occlusion with a middle cerebral artery embolus was a poor prognostic factor despite simultaneous embolectomy and endarterectomy.²⁴

Analysis of Results

Although most authors have argued against an emergency carotid endarterectomy for acute occlusion with severe neurological deficits, we would suggest that in selected patients it may be a viable alternative treatment. In this series, nine patients (26.5%) made an excellent recovery and four patients (11.8%) had a very good result. Therefore, 13 patients (38.3%) had a dramatic improvement from their preoperative deficits. The 10 (29.4%) patients in the fair group are probably a better reflection of the natural history in which, after the infarct, there is a partial reversal of the neurological deficit. Our mortality rate of 20.6% approximates the expected mortality without surgical intervention.

The two most important prognostic factors were the absence of an associated middle cerebral artery embolus and the presence of demonstrated collateral flow. If the preoperative identification of these factors could be improved, then perhaps the surgical success rate could be enhanced while decreasing the operative mortality. An embolus would be a relative contraindication to surgery, whereas good collateral flow would be an encouragement. Therefore, we would recommend, if possible, complete angiography prior to surgical intervention and accept both the inherent 1-2 hour time delay and the risks of angiography in the setting of cerebral ischemia. The patients with occlusion after an elective endarterectomy may be a subgroup in which exploration should be considered early if occlusion is clinically suspected. Furthermore, these results emphasize that angiographic opacification of intracranial vessels cannot be equated with the exact level of cerebral blood flow.

In nine patients (26.5%), there was a documented middle cerebral artery embolus. Presumably the emboli originated from the carotid artery. Whether this was a primary event or secondary to surgical or angiographic manipulation is unknown. However, because of the apparent

thrombogenicity of the occluded carotid artery, great care must be exercised during the surgical procedure.

Although time was not a good prognostic factor, we would not recommend operating after a prolonged delay, as the net result must be a reflection of the time of reduced flow. Our inability to show a good correlation between outcome and time of occlusion reflects the complexity of factors involved in cerebral ischemia and infarction.

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References

1. Wylie EJ, Hein MF, Adams JE. Intracranial hemorrhage following surgical revascularization for treatment of acute strokes. *J Neurosurg* 1964; 21:212-218.
2. Hunter JA, Julian OC, Dye WS, Javid H. Emergency operation for acute cerebral ischemia due to carotid obstruction. *Ann Surg* 1965; 162:901.
3. Blaisdell WF, Clauss RH, Gailbraith JG, et al. Joint study of extracranial artery occlusion IV. Review of surgical considerations. *JAMA* 1969; 209:1889-1895.
4. Thompson JE, Austin DJ, Patman RD. Endarterectomy of the totally occluded carotid artery for stroke. *Arch Surg* 1967; 95:791-801.
5. Thompson JE, Austin DJ, Patman RD. Carotid endarterectomy for cerebrovascular insufficiency. *Ann Surg* 1970; 172:663.
6. Ojemann RG, Crowell RM, Roberson GH, Fischer CM. Surgical treatment of extracranial carotid disease. *Clin Neurosurg* 1975; 22:214-263.
7. Hafner CD, Tew JM. Surgical management of the totally occluded internal carotid artery: a ten-year study. *Surgery* 1981; 89:710-717.
8. Hugenholtz H, Elgie RG. Carotid thromboendarterectomy, a reappraisal. *J Neurosurg* 1980; 53:776-783.
9. Hunt DG, Wheeler CG, Bukhari HI, Hempel GK. Surgical management of total carotid occlusion. *Am J Surg* 1980; 139:700-703.
10. Goldstone J, Effeney DJ. The role of carotid endarterectomy in the treatment of acute neurological deficits. *Progress in Cardiovascular Disease* 1980; 6:415-422.
11. DeWeese JA. Management of acute stroke. *Surg Clin North Am* 1982; 62:3, 467-472.
12. Kistler JP, Ropper AH, Heros R. Therapy of ischemic cerebral vascular disease due to atherosclerosis (Second of 2 parts). *NEJM* 1984; 311(2):100-105.
13. Najafi H, Javid H, Dye WS, et al. Emergency carotid thromboendarterectomy. *Arch Surg* 1971; 103:610-614.
14. Kwaan JH, Connolly JE, Shaneskin JB. Successful management of early stroke after carotid endarterectomy. *Ann Surg* 1979; 5:676-678.
15. Donaldson ML, Dreznen AD. Surgery for acute carotid occlusion. *Amer Surg* 1983; 118:1266-1268.
16. Fischer CM. The natural history of carotid occlusion. In Austin GM, ed. *Microneurosurgical Anastomoses for Cerebral Ischemia*. Springfield: Charles C. Thomas, 1976; 194-201.
17. McDowell FH, Potes J, Groch S. The natural history of internal carotid and vertebral-basilar artery occlusion. *Neurology (MINN)* 1961; 11:153-7.
18. Millikan CH. Clinical management of cerebral ischemia. In McDowell FH, Brennan RW, eds. *Cerebral Vascular Disease*, Eighth Conference. New York: Grune & Stratton, 1973; 209.
19. Jones HR, Millikan CH. Temporal profile (clinical course) of acute carotid system cerebral infarction. *Stroke* 1976; 7:64-71.
20. Grillo P, Patterson RH. Occlusion of the carotid artery, prognosis (natural history), and the possibilities of surgical revascularization. *Stroke* 1975; 6:17-20.

21. Oxbury JM, Greenhall RC, Grainger KN. Predicting the outcome of stroke: acute stage after cerebral infarction. *Br Med J* 1975; 3: 125-127.
22. Norrving B, Nilsson B. Carotid artery occlusion: acute symptoms and long term prognosis. *Neurol Res* 1981; 3: 229-236.
23. Heiss WD, Hayaki T, Waltz AC. Cortico-neuronal function during ischemia. *Arch Neurol* 1976; 33:813-820.
24. Meyer FB, Anderson RE, Sundt TM Jr. Intracellular brain pH, cerebral tissue perfusion, EEG, and histology in both profound and moderate ischemia of the rabbit cortex. *J Cereb Blood Flow Metab*, in press.
25. Sundt TM Jr, Waltz AG. Cerebral ischemia and reactive hyperemia. Studies of cortical blood flow and microcirculation before, during, and after temporary occlusion of the MCA of the squirrel monkey. *Circ Res* 1971; 28:426-433.
26. Wieloch T, Siesjo BK. Ischemic brain injury: the importance of calcium, lipolytic activities, and free fatty acids. *Pathol Biol* 1982; 30:269-277.
27. Meyer FB, Piegras DG, Sundt TM, Yanagihara T. Emergency embolectomy for acute occlusion of the middle cerebral artery. *J Neurosurg* 1985; 62:639-647.
28. Sundt TM Jr, Sharbrough FW, Anderson RE, Michenfelder JD. Cerebral blood flow measurements and electroencephalograms during carotid endarterectomy. *J Neurosurg* 1974; 41:310-320.
29. Sundt TM Jr, Sharbrough FW, Piegras DG, et al. Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy. *Mayo Clin Proc* 1981; 56:533-543.
30. Goldstone J, Moore WS. A new look at emergency carotid artery operations for the treatment of cerebrovascular insufficiency. *Stroke* 1978; 9:599-602.
31. Mentzer RM, Finkelmeier BA, Crosby IK, Wellons HA. Emergency carotid endarterectomy for fluctuating neurological deficits. *Surgery* 1981; 89:60-66.
32. Kusunoki T, Roved DW, Tator CH, Lougheed WM. Thromboendarterectomy for total occlusion of the ICA, a reappraisal of risks, success rate, potential benefits. *Stroke* 1978; 9:34-38.
33. Sundt TM Jr, Grant WC, Garcia HJ. Restoration of middle cerebral artery flow in experimental infarction. *J Neurosurg* 1969; 31: 311-322.
34. Sundt TM Jr, Waltz AG. Experimental cerebral infarction, retro-orbital extradural approach for occluding the middle cerebral artery. *Mayo Clin Proc* 1966; 41:159-168.
35. Crowell RM, Olsson Y, Klatzo I, Ommaya A. Temporary occlusion of the middle cerebral artery in the monkey: clinical and pathological observations. 1970; 1:439-448.
36. Watanabe O, Bremer AM, West LR. Experimental regional cerebral ischemia in the middle cerebral artery territory in primates. Part 1. Angioanatomy and description of an experimental model with selective embolization of the ICA bifurcation. *Stroke* 1977; 8: 61-70.
37. Watanabe O, West LR, Bremer A. Experimental regional cerebral ischemia in the middle cerebral artery territory in primates. Part 2. Effects on brain water and electrolytes in the early phase of middle cerebral artery stroke. *Stroke* 1977; 8:71-76.
38. Dujovny M, Osgood CP, Barrionuevo PJ, et al. Middle cerebral artery microneurosurgical embolectomy. *Surgery* 1976; 80:336-339.
39. Morawetz RB, DeGirolami V, Ojemann RG, et al. Cerebral blood flow determined by hydrogen clearance during middle cerebral artery occlusion in unanesthetized monkeys. *Stroke* 1978; 9:143-149.
40. Kety SS, Schmidt CF. The nitrous oxide method for quantitative determination of cerebral blood flow in man: theory, procedure and normal values. *J Clin Invest* 1948; 27:476-83.
41. Sharbrough FW, Messick JM, Sundt TM Jr. Correlation of continuous electroencephalograms with cerebral blood flow measurements during carotid endarterectomy. *Stroke* 1973; 4:674-683.
42. Trojanborg W, Boysen G. Relation between EEG, regional cerebral blood flow, and internal carotid artery pressure during carotid endarterectomy. *Electroencephalography and Clinical Neurophysiology* 1973; 34:61-69.
43. Branston NM, Symon L, Crockard HA, Pasztor E. Relationship between the cortical-evoked potential and local cortical blood flow following acute middle cerebral artery occlusion in the baboon. *Exp Neurol* 1974; 45:195-208.
44. Harris RJ, Symon L, Branston NM, Bayhan M. Changes in extracellular calcium activity in cerebral ischemia. *J Cereb Blood Flow Metab* 1981; 2:203-209.
45. Astrup J, Blennow G, Nilsson B. Effects of reduced cerebral blood flow on EEG pattern, cerebral extracellular potassium, and energy metabolism in the rat cortex during bicuculline-induced seizures. *Brain Res* 1979; 177:115-126.
46. Astrup J, Symon L, Branston NM, Lassen NA. Cortical-evoked potential and extracellular K⁺ and H⁺ at critical levels of brain ischemia. *Stroke* 1977; 8:51-57.
47. Branston NM, Strong AJ, Symon L. Extracellular potassium activity, evoked potential, and tissue blood flow. Relationship during progressive ischemia in baboon cerebral cortex. *J Neurol Sci* 1977; 32:305-321.