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JOURNAL OF THE AMERICAN HEART ASSOCIATION

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Stroke 1986;17:1251-1253

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1524-4628

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Carotid Endarterectomy: How Does it Work? A Clinical and Angiographic Evaluation

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SUMMARY In a retrospective study, the clinical and angiographic results were evaluated of 100 consecutive carotid endarterectomies, carried out in 90 patients over the period 1977 to 1983. There was no operation-associated mortality; the perioperative morbidity was 5%. Angiography, carried out, on average, 4.2 months postoperatively, revealed complete occlusion of the operated carotid artery in 16 cases (18.5%); in at least 13 cases this was completely asymptomatic. Perioperative findings suggest that microembolism is, but haemodynamic insufficiency is not, an important cause of focal cerebral ischemia. Therefore, treatment for extracranial atherosclerotic cerebrovascular disease should be focused on the elimination of the source of embolism rather than on an improvement of cerebral blood flow.

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THE RELATIONSHIP between extracranial carotid disease and cerebral ischemia is well established. Over the past few decades, carotid endarterectomy has evolved as the treatment of choice for selected patients with atherosclerotic disease involving the carotid bifurcation.¹⁻³ The available data do not support a role for this operative procedure in asymptomatic patients or in patients with severe neurological deficit or with serious accompanying diseases. Several studies, however, indicate that in patients with transient ischemic attacks or mild to moderate neurological deficit due to carotid stenosis, the survival rate can be improved by carotid endarterectomy when surgery is performed by an experienced surgeon and when the combination of angiography and surgery creates a combined mortality and morbidity rate not exceeding 5%. Because the pathogenesis of ischemic attacks is still the subject of much discussion (microembolic versus haemodynamic theories), it is not known whether carotid endarterectomy is effective by removing the source of the emboli or by increasing cerebral blood flow.

The purpose of this study is to analyze retrospectively the operative findings and the clinical and angiographic results in a series of 100 consecutive carotid operations in 90 patients with carotid disease, mainly in order to evaluate any facts which may shed light upon the controversial role of embolism as opposed to haemodynamic mechanism producing symptoms in carotid artery disease.

Patients and Methods

This report includes all patients who were operated upon for carotid stenosis by one neurosurgeon (H.A.M. v. A.) during the period 1977 through 1983. Ninety patients underwent one hundred carotid operations; bilateral endarterectomy was performed in 10 patients. The patients consisted of 64 males and 26 females, with an age range at time of surgery of 44 to 70 years (mean age 61 years). More detailed patient

characteristics are given in tables 1 and 2. In order to be considered for surgery, patients had to have clinical signs of cerebral ischemia and a stenotic lesion on carotid angiography, with or without visible ulceration, in the extracranial part of the appropriate carotid artery. All patients were treated with anticoagulants, started preoperatively. All operations, which were routinely monitored with intraoperative EEG-registration, were carried out under general anaesthesia, with moderate induced hypertension and hypocapnia (pCO₂ under 30 mm Hg). No carotid shunt was placed during the period of occlusion of the artery, which in most cases was 20 minutes or less. No patch graft was used. Considering the procedures: in 94 cases, a carotid bifurcation endarterectomy was performed; in 1 case, a severely ulcerating internal carotid artery was ligated, because of the high risk of embolism after the operation; and in 5 cases of occluded internal carotid artery, an internal carotid stumpectomy was combined with an external carotid endarterectomy. Postoperatively, all patients underwent detailed neurological examinations and an EEG-study was performed at the end of the first postoperative week. Postoperative angiographic examinations (carotid angiography or intravenous digital subtraction angiography) were obtained routinely in 87 of 100 cases. For various reasons, no angiographic re-evaluation was carried out in 13 cases, including 4 in whom the internal carotid artery was already occluded, and 1 case of carotid ligation. A few patients declined postoperative angiography. Except for a number of cases, in whom angiography was indicated a few days after surgery (new signs of cerebral ischemia), the angiograms were made 3 to 6 months postoperatively.

Results

Clinical Findings

A good clinical result (no surgical complications, uneventful postoperative course, no further ischemic symptoms) was obtained after 95 operations. There was no perioperative mortality and the morbidity (when defined as any deficit during the first 30 days postoperatively that had not been present before operation) was 5%. No complications occurred during the operative procedure. Three patients suffered a new

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Received April 23, 1986, accepted June 24, 1986.

TABLE 1 *Pre-operative Angiographic Findings in 90 Patients*

Unilateral carotid stenosis	53
Bilateral carotid stenosis	32
Occlusion internal carotid artery	5

neurological deficit several hours after the operation, and in all three there was an ischemic stroke. In one patient, who showed a middle cerebral artery infarction on CT-scan, angiography revealed an occlusion of the internal carotid artery and a middle cerebral artery embolism on the operated side. In one patient, postoperative carotid angiography showed no abnormalities, and in one patient with an already occluded internal carotid artery in whom a stumpectomy was combined with an external carotid endarterectomy, there was an external carotid artery occlusion with insufficient visualization of the intracranial circulation. Two other patients developed hemiparesis a few days postoperatively. In one patient it was associated with a myocardial infarction (no postoperative angiography). In the other patient it occurred during a period of urosepsis. Angiography showed an internal carotid artery occlusion, again with inadequate visualization of the intracranial circulation.

The patient in whom a carotid ligation had been performed, was completely free of transient ischemic attacks and had no neurological symptoms after the operation.

Angiographic Findings

87 Postoperative angiograms were performed after a mean interval of 4.2 months. In 71 cases there was a 'normal' postoperative appearance (normal-sized lumen with, in some cases, small wall irregularities but without stenosis). In 16 cases there was complete occlusion of the operated carotid artery, including one case in whom an endarterectomy of the external carotid artery was performed in combination with a stumpectomy of an occluded internal carotid artery. In 13 of these 16 cases this occlusion remained completely asymptomatic. In 3 cases the occlusion was possibly related to ischemic events. As mentioned above, in one of these cases an embolism of the middle cerebral artery was also found; this was probably responsible for the clinical symptoms. In the other two cases there was no adequate visualization of the intracranial circulation to demonstrate or to exclude embolism.

TABLE 2 *Clinical Indications for Carotid Surgery*

	First operation	Contra-lateral operation
Ipsilateral carotid TIA or amaurosis fugax	58	1
Vertebrobasilar TIA	2	0
RIND or minor stroke in carotid distribution	26	0
Non-focal ischemic symptoms	0	5
Asymptomatic carotid stenosis	4	4

Discussion

It is recognized that atherosclerotic stenotic lesions of the cervical carotid artery are related to cerebral ischemia. Two mechanisms have been proposed to explain the pathogenesis of these ischemic attacks: cerebral embolism arising from mural thrombus formation in the involved extracranial carotid artery with transient or permanent occlusion of one or more intracranial arteries, and a reduction of cerebral perfusion due to severe stenosis or occlusion of the extracranial carotid artery without intracranial obstruction. It is important to establish the mechanism of cerebral ischemia to be able to choose between the different possibilities of surgical treatment (carotid endarterectomy, extracranial-intracranial bypass, carotid ligation), assuming that surgery is indicated in these cases. Different opinions on pathogenesis and therapy have been reviewed by Persin et al., Barnett and Whisnant.⁶⁻⁸

In this study some clinical and angiographic features of 100 consecutive carotid operations in 90 patients were analyzed to gather arguments for one of the above-mentioned pathogenetic mechanisms. In this series, no internal shunt was used to bypass the occluded carotid artery during the procedure. In none of these patients, including 5 cases with severe EEG-changes during carotid cross-clamping, was any neurological deficit seen due to the reduction of cerebral blood flow during surgery, even if the clamping time was longer than 20 minutes. In all cases, therefore, the collateral circulation appears to be sufficient to supply the cerebral perfusion of the relevant hemisphere during carotid clamping. This finding is in accordance with the conclusion of Ferguson, who stated that 'neither intra-operative monitoring nor the use of internal shunts is necessary to avoid intra-operative stroke in carotid endarterectomy, as the usual cause for such stroke is an embolus'.⁹ Also the fact that in one of our patients a relief of transient ischemic attacks occurred after definitive ligation of a severely atherosclerotic, but incompletely obstructed internal carotid artery, can only be explained by embolism as the cause of ischemia rather than by haemodynamic factors. Also Countee et al reported that occlusion of the carotid artery can be an effective surgical remedy for arresting embolic discharge from a seriously diseased vessel in patients in whom collateral hemispheric blood flow is believed to be sufficient to tolerate a carotid ligation.¹⁰

Another feature suggesting that the brain is well protected against haemodynamic stresses and that embolism is the more likely cause of cerebral ischemia, is the high percentage of well-tolerated carotid occlusions, detected during postoperative follow-up. In 16 out of 87 cases, in whom postoperative angiography was performed (18.5%), a complete occlusion of the carotid artery was demonstrated. The reason for this surprisingly high number is not known. Some other reports in the recent literature mention postoperative carotid occlusion rates of about 10% and combined restenosis-and-occlusion rates of up to 36%.¹¹⁻¹⁵ In other studies, the patency rate of the internal carotid artery following endarterectomy is very high.¹⁶⁻¹⁹ A

possible explanation for this discrepancy could be the fact that in some early studies only noninvasive, less accurate methods have been used to follow the postoperative course of asymptomatic patients. Another possibility is that late timing of postoperative angiography plays an important role. In our series angiography was routinely carried out 3 to 6 months after surgery.

In 13 out of 16 patients postoperative carotid occlusion was completely asymptomatic. In the other 3 cases in whom ischemic stroke had occurred, the occlusion was demonstrated shortly after operation. In one case an embolus of the middle cerebral artery was also found at angiography, in two cases, embolism was a likely cause of ischemia, but the intracranial vessels could not be visualized. In all 5 cases of postoperative ischemia, stroke occurred some hours or days after surgery, which also suggests an embolic genesis.

From these observations we conclude that there is much evidence that microembolism is the most important cause of transient ischemic attacks or cerebral infarction in a case of a stenotic atherosclerotic lesion of the cervical carotid artery. Haemodynamic mechanisms appear to play a minor role in focal cerebral ischemia. It is probable that reduced cerebral perfusion due to extracranial carotid stenosis, if present, gives rise to a compensatory mechanism which maintains blood flow by rapid development of collateral circulation. As a consequence, both carotid endarterectomy and carotid ligation can be considered as effective surgical means for eliminating the source of cerebral embolism. For the same reason, a surgical procedure, intended to improve cerebral blood flow (extracranial to intracranial bypass) would seem to be useless in the case of atherosclerotic disease, because it does not affect the source of embolism.

Acknowledgment

We are indebted to Miss Annemieke Bollen for preparing the manuscript.

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