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Prospective Analysis of Carotid Endarterectomy and Silent Cerebral Infarction in 97 Patients

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To determine the incidence of perioperative silent cerebral infarction, 97 patients who underwent carotid endarterectomy were prospectively studied with preoperative and postoperative computed tomograms. Thirty-one of 96 patients (32%) had findings of cerebral infarction on preoperative computed tomograms. Silent cerebral infarction was found preoperatively in 17 patients (18%) (lacunar infarction in 10, cortical infarction in five, both cortical and lacunar infarctions in one, and cerebellar infarction in one). Transient ischemic attacks occurred in 10 of the 17 patients with silent cerebral infarction; however, symptoms were appropriate to the site in only five of these 10 patients. Fourteen of the 17 patients with silent cerebral infarction had a hemodynamically significant carotid stenosis, and seven patients had an ulcerated plaque on preoperative angiogram. The incidence of these lesions was similar to that found in the group of 66 patients without cerebral infarction. Endarterectomy specimens revealed a higher but not significantly different incidence of ulcerated plaque in the silent cerebral infarction group. There were no perioperative deaths. Following surgery, one patient (1%) with a preoperative silent cerebral infarction suffered a transient ischemic attack, and two patients (2%) with normal preoperative computed tomograms developed permanent neurologic deficits with new cortical infarctions on postoperative computed tomograms. No new silent cerebral infarctions were found on postoperative computed tomograms in any of the 97 patients. Our data suggest that silent cerebral infarction is a common preoperative finding with an as-yet unclear etiology and that carotid endarterectomy does not appear to be a cause. (*Stroke* 1989;20:329–332)

The efficacy of carotid thromboendarterectomy (TEA) in preventing stroke has come under close scrutiny. Both its indications and complication rates have been disputed.[1–6] Recent data suggest that silent cerebral infarction (SCI) occurs in 8% of patients undergoing TEA, and, when added to clinically apparent perioperative strokes, the result is an overall stroke rate of >10% for TEA.[6] In view of the expectation that well in excess of 80,000 TEAs will be performed in the United States this year, clarifying TEA complication rates is an important challenge for all physicians caring for patients with cerebrovascular disease.[7] In an effort to clarify operative results, we studied SCI in patients undergoing TEA.

Subjects and Methods

We adopted routine preoperative and postoperative computed tomography (CT scanning) for all patients undergoing TEA performed by us or under our direct supervision during the 19 months between June 1985 and January 1987. Data were abstracted from the vascular registries at the Naval Hospital and the Veterans Administration Hospital in San Diego, California.

The indications for TEA were 1) transient ischemic attacks (TIAs) of the cerebral cortex or amaurosis fugax with significant carotid bifurcation stenosis or ulceration on the appropriate side, 2) cortical stroke with complete or near-complete recovery and significant carotid bifurcation stenosis or ulceration on the appropriate side, and 3) ≥80%

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reduction in cross-sectional area of the arterial lumen at the carotid bifurcation in asymptomatic patients without significant cardiopulmonary disease and with a sufficient life expectancy to warrant prophylactic operation.

We paid special attention to the work-up of patients with TIAs and findings of lacunar infarction in the appropriate anatomic distribution relative to the symptoms. We did not refer these patients for angiography unless noninvasive laboratory examination suggested the presence of a hemodynamically significant carotid stenosis. Our practice was based on the conclusion that lacunar infarction is usually a small-vessel thrombotic event and is rarely due to macrovascular embolic phenomena. We attempted to differentiate true cortical TIAs and transient symptoms from infarction in subcortical areas. In the presence of lacunar infarction with good recovery and significant carotid stenosis, we used a rationale for surgery similar to that used in our group of asymptomatic patients who underwent prophylactic TEA.

During the perioperative period, we paid particular attention to blood pressure management. Arterial cannulas for continuous blood pressure management were placed in all patients. Phenylephrine hydrochloride, nitroglycerin, and nitroprusside drips were available for blood pressure management and were required in 90% of our patients. We routinely injected the carotid sinus nerve with local anesthetic to block baroreceptor function. Postoperatively, we limited the use of longer-acting antihypertensive agents to prevent the hypotension caused by the cumulative and delayed effects of these drugs.

Data abstracted from the vascular registries included history, results of the physical examination and noninvasive laboratory studies, arteriographic findings, and preoperative CT scan results. Perioperatively, one of us examined endarterectomy specimens in the operating room; the presence of ulcers, intraplaque hemorrhage, and degree of stenosis were noted. The details of the operative procedure and perioperative blood pressure management were also recorded. Postoperative neurologic status was carefully examined, and CT scans were obtained in all patients a mean of 12 days after surgery.

We compared data for the groups with and without findings of SCI using Student's t test and the χ^2 distribution with significance attributed to $p<0.05$.

Table 1. Indication for 106 Carotid Thromboendarterectomies in 97 Patients

Indication	No.	%
Stroke	14	13
Transient ischemic attack	32	30
Amaurosis fugax	23	22
Vertebrobasilar insufficiency	5	5
Asymptomatic stenosis	32	30

Table 2. Operative Procedure: 106 Carotid Endarterectomies in 97 Patients

	No.	%
Resident as primary surgeon	84	79
Shunt placement	94	89
Patch angioplasty	47	44
External carotid TEA	3	3
TEA and arch vessel bypass	2	2
TEA and CABG	1	1

TEA, carotid thromboendarterectomy; CABG, coronary artery bypass graft.

Results

During the 19 months of our study, 106 TEAs were performed in 97 patients, 70 men and 27 women, with a mean age of 66 years. Patient cardiovascular risk factors included smoking in 89%, hypertension in 86%, hyperlipidemia in 18%, and diabetes in 10%. Transient ischemic events, either amaurosis fugax or TIAs, accounted for 52% of the indications for TEA. Approximately one third of the patients underwent TEA for an asymptomatic stenosis or vertebrobasilar (VB) TIA with significant stenosis. Fewer than 15% of the patients underwent TEA for the indication of stroke with good recovery (Table 1).

Shunts were routinely used at the Naval Hospital and were selectively used at the Veterans Hospital. Patch angioplasty, although not routinely performed, was liberally used at both institutions. TEA alone was performed in 94% of the operations. External carotid endarterectomy with internal carotid exclusion, TEA and carotid–subclavian bypass, or combined TEA and coronary artery bypass graft were performed in 6% (Table 2).

All but one of the 97 patients underwent preoperative CT scanning a mean of 11 days before surgery. Postoperative CT scanning was performed in all patients a mean of 12 days after surgery. Five patients initially did not keep their follow-up appointment and underwent delayed CT scanning between 4 and 7 months after surgery.

Cerebral infarction was found on the preoperative CT scan in 31 of 96 patients (Table 3). All patients with the preoperative clinical diagnosis of stroke were found to have an infarction on CT scan. The most common unsuspected preoperative finding

Table 3. Preoperative Cerebral Infarctions Documented by Computed Tomography in 96 Patients

	No.	%
Symptomatic	14	15
Silent	17	18
Lacunar	10	10
Cortical	5	5
Cortical and lacunar	1	1
Cerebellar	1	1
Total	31	32

was lacunar infarction; cortical infarction or combined cortical and lacunar infarctions were less common. One asymptomatic patient with a 95% stenosis of the carotid bifurcation was found to have a cerebellar infarction.

The indication for TEA in the 17 patients with preoperative SCI included TIA in 10, VB TIA in five, and asymptomatic stenosis in two patients. Of the 32 patients with TIA as the indication for TEA, SCI was found on the preoperative CT scan in 10 (31%); however, only five patients were found to have SCI in an area of the brain corresponding to the TIA.

When the group of 17 patients with SCI was compared with the group of 66 patients without cerebral infarction, there were no differences in the incidence of risk factors and indications for TEA. An examination of plaque morphology in the total group of 31 patients with preoperative cerebral infarction revealed ulceration in 21 (68%) and hemodynamically significant stenosis in 16 (52%). The group of 17 patients with SCI had a higher (but not significantly different) incidence of ulceration in the endarterectomy specimen compared with the group of 66 patients without cerebral infarction (71% and 54%, respectively). However, only one half of the SCIs were found in an anatomic distribution appropriate to the side of the carotid lesion.

No new SCIs were found on postoperative CT scans. Complications included early stroke secondary to carotid thrombosis in two patients (2%) who underwent TEA for TIA; both patients had a normal preoperative CT scan and developed unequivocal evidence of cortical infarction in the ipsilateral hemisphere on postoperative CT scan. One patient (1%) suffered a perioperative TIA in the distribution of a silent lacunar infarction found on preoperative CT scan; the postoperative CT scan did not show any new infarctions. There were no perioperative deaths.

Discussion

The role of TEA in the treatment of cerebrovascular disease has become controversial. Many questions regarding the efficacy of this operation arise from an imprecise definition of clinical status and a lack of close scrutiny of surgical results.[8] Reports of SCI in both the preoperative and perioperative periods are alarming, and they provide further challenges to the traditional classification of cerebrovascular symptoms and surgical outcomes. Our results provide some perspective on the importance of a careful quantification of the extent of cerebrovascular disease, its neurologic consequences, and the effects of surgical treatment.

Routine preoperative CT scanning reveals a significant number of SCIs. The overall incidence of preoperative SCI was 18% in our study. If patients with a history of stroke are excluded, the incidence is 21%. Most of these SCIs (10 of 17) were lacunar; however, fewer than half were found in the distribution of the patient's symptoms. In the 32 patients with a preoperative diagnosis of TIA, 10 (31%) were found to have SCI, but fewer than one half of these SCIs were in the hemisphere appropriate to the patient's symptoms. It must be remembered that we excluded patients with transient symptoms and corresponding lacunar infarction in an attempt to limit the designation of TIA to cortical events. Although there was a higher incidence of ulcerated bifurcation plaque in patients with SCI, fewer than one half of these lesions occurred on the side of the infarct. Therefore, the bifurcation lesion cannot be termed causative for SCI. Rather, the two processes (macrovascular plaque formation and small-vessel thrombotic events) are most likely parallel phenomena related to the risk factors present in an overwhelming majority of our patients.

The importance of routine CT scanning in evaluating patients with cerebrovascular disease who are considered candidates for surgical treatment cannot be underestimated. Without a corresponding anatomic study of the brain, clinical symptoms alone are inaccurate indicators of the presence or absence of cerebral infarction.[8] Compelling evidence suggests that the traditional classifications including TIA, reversible ischemic neurologic deficit, and stroke are insufficient to describe cerebrovascular disease without the qualifying addition of CT scan results.

Both patients with perioperative strokes had clinically obvious neurologic deficits in the early postoperative period with corresponding CT scan lesions. There were no perioperative SCIs in the 97 patients we studied. This finding differs significantly from the 8% perioperative SCI rate reported by Berguer and colleagues.[6] We compared our study with theirs in an effort to find factors that would explain this discrepancy. Their indications for surgery, incidence of preoperative SCI, and operative findings were identical to ours. Their operative technique and ours did vary, however. They combined vertebral artery reconstruction with TEA in 10 (10%) of their patients, but only one of these 10 developed perioperative SCI. We employed patch angioplasty significantly more frequently (44% vs. 13%). We used shunts in 89% of our TEAs, while Berguer et al[6] did not report the use of shunts. Although it is difficult to quantify the impact of the use of patch angioplasty and shunts, they may represent key factors in the prevention of perioperative SCI.

The details of perioperative blood pressure management in the study of Berguer et al[6] are not available. We placed special emphasis on the management of systolic blood pressure within definite limits in the perioperative period, believing that wide swings in blood pressure may be the etiology of many perioperative neurologic events. Unless carefully managed with protocols clearly stated for both house staff and nursing staff, hypotension and hypertension in the early postoperative period are often ineffectively treated. Although we cannot assess the impact of this approach to blood pressure

management in the prevention of perioperative SCI, we consider it likely to be a key factor.

Our findings suggest that although SCI is a common preoperative finding, it appears not to be a complication of carefully monitored TEA. Although an integral part of the work-up of all TEA candidates, CT scanning does not appear to be indicated following uncomplicated operations.

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KEY WORDS • endarterectomy • cerebral infarction