
A Prospective Study of Vein Patch Angioplasty During Carotid Endarterectomy

Three-year Results for 801 Patients and 917 Operations

NORMAN R. HERTZER, M.D., EDWIN G. BEVEN, M.D., PATRICK J. O'HARA, M.D., and LEONARD P. KRAJEWSKI, M.D.

From 1983 through 1985, 801 consecutive patients (mean age: 66 years) underwent 917 primary carotid endarterectomies at the Cleveland Clinic. Conventional arteriotomy closure was performed during 483 operations, while patch angioplasty using a distal segment of saphenous vein was employed in 434. Preoperative risk factors, surgical management, and antiplatelet therapy were equivalent in the vein patch (VP) and non-patch (NP) groups. Early results were evaluated by intravenous angiography (DSA) in 715 patients (89%), and 332 reconstructions (36%) have been reassessed by objective imaging during a mean follow-up interval of 21 months. Ischemic strokes occurred after 18 (1.9%) of the 917 procedures (0.7% VP, 3.1% NP; $p = 0.0084$), and symptomatic ($N = 9$) or unsuspected ($N = 8$) thrombosis of the internal carotid artery was confirmed by neck exploration or routine DSA after 1.9% of all operations (0.5% VP, 3.1% NP; $p = 0.0027$). Only ten patients (1.2%) have required reoperations for severe recurrent lesions, but the cumulative 3-year incidence of new defects ($\geq 30\%$ stenosis) documented by objective studies in the VP and NP groups was 9% and 31%, respectively ($p = 0.0066$). These results strongly suggest that VP angioplasty enhances the safety and durability of carotid endarterectomy.

CONTROVERSY CONCERNING CEREBROVASCULAR surgery was abruptly intensified in 1985, when the EC/IC Bypass Study Group concluded that medical therapy was as effective as middle cerebral revascularization in the treatment of intracranial arterial disease.¹ In response to this investigation as well as to the fact that carotid endarterectomy has become one of the most common vascular procedures performed in the United States, influential neurologists have suggested that even extracranial reconstruction may no longer be justified outside the framework of prospectively randomized clinical trials.²⁻⁴ Without exception, these criticisms were supported by the high operative stroke and/or death rates reported several years ago by Easton and Sherman⁵ in southern Illinois (21%),

From the Cleveland Clinic Foundation, Department of Vascular Surgery, Cleveland, Ohio

and more recently by Brott et al.^{6,7} in Cincinnati (6.5–9.5%).

Excessive surgical complications obviously have a legitimate influence on medical opinion. For example, Chambers and Norris⁴ calculated in 1984 that asymptomatic carotid bifurcation lesions should be corrected only if a subset of patients could be identified in which the annual incidence of stroke without operation was 5% or more. The same authors found in 1986 that the spontaneous risk of severe stenosis ($>75\%$ by Doppler ultrasonography) actually exceeded this figure (5.5%), yet they again discouraged elective intervention because four (13%) of the 30 patients in their series who underwent carotid endarterectomy had perioperative strokes.⁸ Countless other studies have demonstrated that the composite morbidity and mortality of carotid endarterectomy is less than 3% at many centers, but it is inevitable that the merit of this operation will be open to speculation until its safety is universal.

Dependable technical results are essential to the success of carotid endarterectomy. The bifurcation must be widely patent and relatively resistant to platelet aggregation in order to prevent thrombosis or microembolization, but in each of these respects, routine arteriotomy closure occasionally may be inadequate. Turnipseed et al.⁹ and Ortega et al.¹⁰ have documented postoperative internal carotid occlusions in 3–5% of patients evaluated by noninvasive methods, whereas others using intraoperative ultrasound have consistently discovered residual intraluminal defects in 7–8% of their series.¹¹⁻¹³ Early thrombosis has been recognized after approximately 2% of the carotid endarterectomies performed at the Cleveland Clinic, and neurologic deficits occurred with fewer than half of these events.^{14,15} In 1983, however, we ini-

Reprint requests: Norman R. Hertzler, M.D., Department of Vascular Surgery, the Cleveland Clinic Foundation, Cleveland, OH 44106.
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tiated a prospective study of vein patch (VP) angioplasty in an attempt to eliminate, if possible, all serious complications of an operation for which perfection appears to be the only acceptable standard. This report is a summary of the first 3 years of our experience with carotid patching.

Materials and Methods

Patch angioplasty previously had been performed on a selective basis in approximately 7% of patients who required carotid endarterectomy at the Cleveland Clinic, but one of us (N.R.H.) decided early in 1983 to apply a routine patch of saphenous vein harvested from the ankle to determine whether this precaution might influence the perioperative complication rate and/or the incidence of recurrent stenosis. Excluding late reoperations and any procedures that were limited to the external carotid artery, 801 patients underwent a total of 917 conventional bifurcation endarterectomies within the subsequent 32 months of the investigation. The remaining authors continued to patch selectively during this time, but Figure 1 illustrates that their preference for primary closure declined throughout the study period. In summary, there were 233 patients (273 operations) in the routine patch group, whereas selective patching was used in another 135 (161 operations). Only 14 entrants (1.7%) received bilateral reconstructions with a patch on one side but not on the other; demographic and survival information for this small subset was included with the non-patched (NP) group (Tables 1 and 4, Fig. 2), but the cumulative neurologic data for these 14 patients were calculated in the VP group as well (Fig. 3).

Patient Information

The VP and NP groups contained 368 and 433 patients, respectively. As indicated by the comparisons in Table 1, these two cohorts had similar clinical features.

Sex and Age

There were 563 men (70%) and 238 women (30%) in the entire series. The men ranged in age from 40 to 84 years (mean: 65 years), whereas the women were 47-87 years of age (mean: 67 years).

Surgical Indications

Unilateral carotid endarterectomy was performed in 85 patients, but 116 (14%) required bilateral procedures during the study period. Previous neurologic events had occurred in 393 patients (49%), including 41 (18%) with completed strokes followed by good functional recovery, 123 (15%) with cortical transient ischemic attacks (TIA), and 129 (16%) with amaurosis

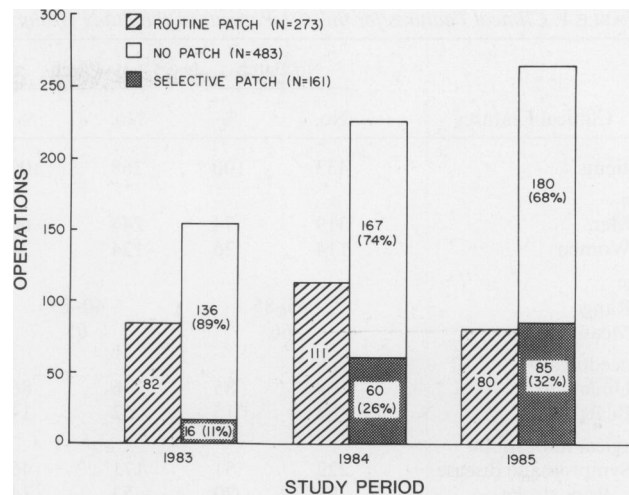


FIG. 1. Composition of the routine and selective patch groups in this series.

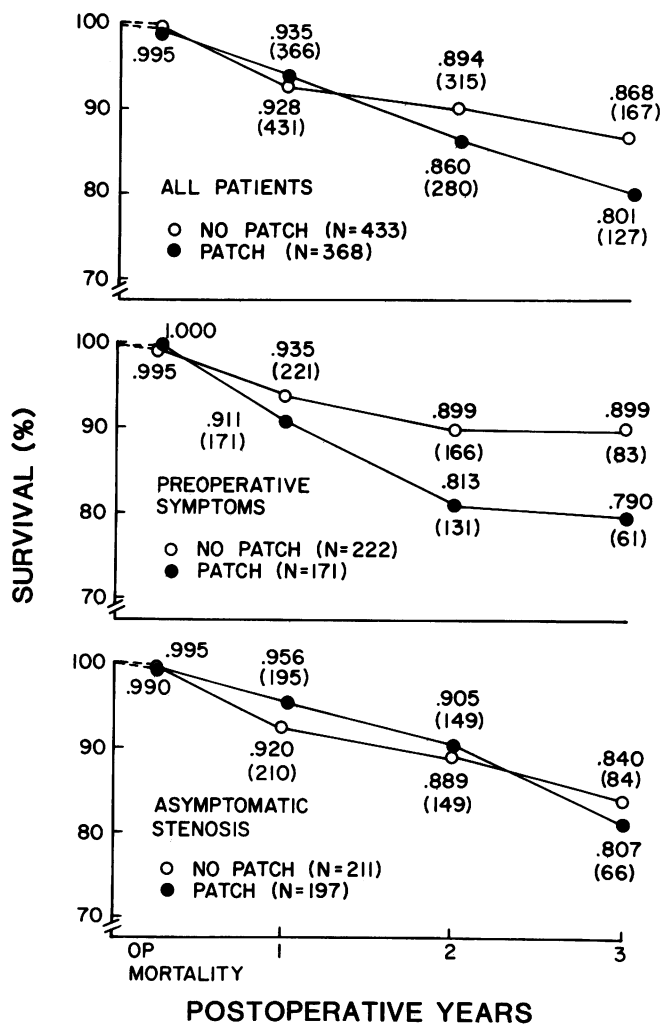


FIG. 2. Three-year life table survival according to the preoperative neurologic status. The number of eligible patients at each interval is enclosed in parentheses.

TABLE 1. Clinical Features for the 801 Patients in the Study Group

| Clinical Features | No Patch | | Patch | |
|-----------------------|----------|-----|-------|-----|
| | No. | % | No. | % |
| Patients | 433 | 100 | 368 | 100 |
| Sex | | | | |
| Men | 319 | 74 | 244 | 66 |
| Women | 114 | 26 | 124 | 34 |
| Age | | | | |
| Range | 45-85 | | 40-87 | |
| Mean | 66 | | 65 | |
| Procedures | | | | |
| Unilateral | 369 | 85 | 316 | 86 |
| Bilateral | 64 | 15 | 52 | 14 |
| Surgical indications | | | | |
| Symptomatic disease | 222 | 51 | 171 | 46 |
| Prior stroke | 88 | 20 | 53 | 14 |
| Transient ischemia | 62 | 14 | 61 | 17 |
| Amaurosis fugax | 72 | 17 | 57 | 15 |
| Asymptomatic stenosis | 211 | 49 | 197 | 54 |
| Risk factors | | | | |
| Hypertension | 300 | 69 | 254 | 69 |
| Diabetes mellitus | 90 | 21 | 70 | 19 |
| Antiplatelet therapy | | | | |
| Preoperative | 249 | 58 | 194 | 53 |
| Postoperative | 376 | 87 | 288 | 78 |

fugax or microembolic retinal infarcts. The remaining 408 patients (51%) had asymptomatic carotid stenosis (N = 385, 48%) or subtotal lesions associated with non-specific vertebrobasilar symptoms (N = 23, 3%).

Risk Factors and Antiplatelet Therapy

A total of 554 patients (69%) either had hypertension (BP \geq 180/90 mmHg) or already required antihypertensive management. Diabetes mellitus under treatment

with insulin or oral hypoglycemic agents was present in 160 patients (20%). Serum cholesterol values were available for 696 entrants (87%) and ranged from 91 to 995 mg/dL, with a mean of 245 mg/dL (NP, 240 mg/dL; VP, 252 mg/dL). Triglyceride levels were obtained for 206 patients (26%) and ranged from 35 to 4400 mg/dL with a median of 245 mg/dL (NP, 252 mg/dL; VP, 238 mg/dL).

Antiplatelet agents (aspirin and/or dipyridamole) were prescribed before surgery for 443 patients (56%), whereas 664 (83%) complied with antiplatelet therapy during the early postoperative course as well as the late follow-up interval. Another 52 patients (6%) received coumadin before their operations, and 27 (3%) have remained on formal anticoagulation after carotid endarterectomy.

Surgical Considerations

All 917 procedures in this series were conducted using general anesthesia, routine carotid shunting, and systemic heparinization that was at least partially reversed with protamine sulfate at the conclusion of each operation. The saphenous patch usually was obtained at the medial malleolus, but it was removed from the upper calf whenever the arterial circulation appeared inadequate to heal a harvesting incision near the foot. There were no wound complications with this approach, and the loss of a segment of vein close to the ankle did not interfere with subsequent *in situ* saphenous grafts in the few patients who already were scheduled for elective lower extremity revascularization at the time their carotid disease was discovered.

The carotid arteriotomy was repaired primarily during 483 operations, whereas patch angioplasty was performed in 434. Additional surgical considerations were comparable in both groups (Table 2).

Associated Procedures

Only nine (1%) of the 917 operations were necessary to correct incidental carotid lesions in patients who originally were evaluated because of other manifestations of peripheral vascular disease. Another 161 procedures (18%), however, were either staged (N = 53, 6%) or combined (N = 108, 12%) with open heart surgery in cardiac patients. Altogether, 122 (26%) of the 464 endarterectomies for asymptomatic carotid stenosis were done in conjunction with myocardial revascularization.

Angiographic Status

Preoperative angiograms demonstrated severe (\geq 70%) stenosis of the ipsilateral common carotid or internal carotid artery preceding 724 operations (79%). Severe stenosis or occlusion of the contralateral internal

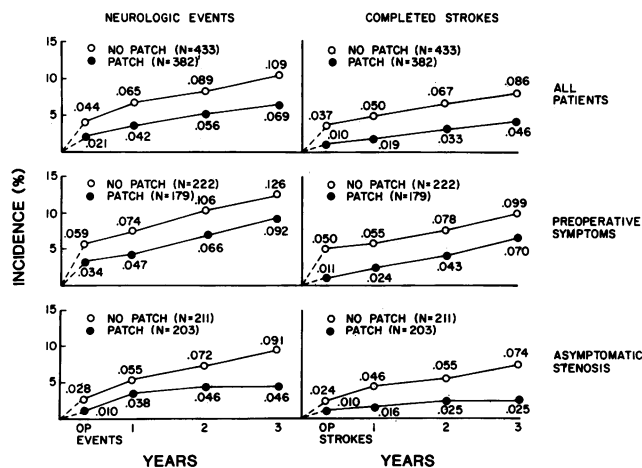


FIG. 3. The cumulative incidence of all ipsilateral neurologic events (left) and completed strokes (right) within the perioperative and late study periods.

carotid was present at the time of 215 procedures (23%), but the incidence of advanced contralateral carotid disease was similar in the VP (25%) and NP cohorts (22%).

Postoperative Assessment

Early technical results were documented by intravenous digital subtraction angiography (DSA) during the same hospital admission in 715 (89%) of the 801 patients. Although 14 operative survivors (1.8%) have been lost, late clinical results were available for the remainder of the series during a maximum follow-up interval of 40 months (mean: 21 months). Within this period, 332 (36%) of the 917 carotid endarterectomies were reassessed by DSA (N = 290, 87%) or Doppler ultrasonography (N = 42, 13%). Long-term objective imaging was obtained for 43% of the VP group and for 30% of the NP group.

Statistical Analysis

Paired data were compared using Fisher's exact test. Life table calculations were performed according to Cutler and Ederer¹⁶ and were evaluated with the method described by Lee and Desu.¹⁷

Results

Early Results

A summary of perioperative complications for all 917 operations in the series is presented in Table 3.

Operative mortality. Although 14 patients (1.7%) died as the result of cardiac or other peripheral vascular procedures that were performed within the first month after carotid endarterectomy, only four fatal complications (0.5%) were related directly to extracranial reconstruction. Two of these patients (1 VP, 1 NP) had delayed cerebral hemorrhage and another sustained a myocardial infarction following endarterectomy alone. The fourth patient experienced a brainstem stroke during simultaneous coronary and carotid revascularization and eventually developed irreversible multisystem failure. Nevertheless, early mortality was identical irrespective of whether patch angioplasty had been employed.

Ischemic neurologic deficits. Seven transient neurologic deficits (0.8%) were evenly distributed (4 VP, 0.9%; 3 NP, 0.6%) and disappeared completely within hours after recovery from general anesthesia. Ischemic strokes with persistent functional impairment occurred in conjunction with 18 operations (1.9%), and all involved the ipsilateral cerebral hemisphere. The difference between early stroke rates in the VP (0.7%) and NP groups (3.1%) was statistically significant ($p = 0.0084$).

Internal carotid occlusion. Postendarterectomy thrombosis of the internal carotid artery was docu-

TABLE 2. Surgical Considerations for the 917 Carotid Endarterectomies Performed During the Study Period

| Surgical considerations | No Patch | | Patch | |
|-------------------------|----------|-----|-------|-----|
| | No. | % | No. | % |
| Operations | 483 | 100 | 434 | 100 |
| Associated | | | | |
| Cardiac procedures | | | | |
| Staged | 29 | 6 | 24 | 6 |
| Simultaneous | 53 | 11 | 55 | 13 |
| Ipsilateral | | | | |
| Carotid stenosis | | | | |
| <50% | 47 | 10 | 28 | 6 |
| 50-69% | 63 | 13 | 55 | 13 |
| 70-89% | 198 | 41 | 144 | 33 |
| ≥90% | 175 | 36 | 207 | 48 |
| Contralateral | | | | |
| Carotid stenosis | | | | |
| <50% | 324 | 67 | 279 | 64 |
| 50-69% | 53 | 11 | 46 | 11 |
| 70-89% | 41 | 8 | 26 | 6 |
| ≥90% | 12 | 3 | 20 | 5 |
| Occlusion | 53 | 11 | 63 | 14 |

mented by immediate neck exploration or angiography in nine of the 15 patients who experienced perioperative strokes in the NP cohort, and all nine of these patients underwent urgent thrombectomy with secondary patch angioplasty. In comparison, symptomatic thrombosis did not occur in the primary patch group ($p = 0.0041$). Eight other patients (1%) were found to have asymptomatic internal carotid occlusions during routine postoperative DSA studies performed before their discharge from the hospital (2 VP, 0.5%; 6 NP, 1.2%). Thus, a total of 15 early occlusions (3.1%) were associated with standard arteriotomy closure, whereas only two clinically silent events (0.5%) were identified in the patch group ($p = 0.0027$).

TABLE 3. Operative Complications for the Complete Series of 917 Carotid Endarterectomies

| Operative Complications | No Patch (N = 483) | | Patch (N = 434) | |
|-----------------------------|-----------------------|-----|--------------------|-----|
| | No. | % | No. | % |
| Related mortality | 2 | 0.5 | 2 | 0.5 |
| Neurologic events | 19 | 3.9 | 8 | 1.8 |
| Transient ischemia | 3 | 0.6 | 4 | 0.9 |
| Completed stroke | 15 | 3.1 | 3 | 0.7 |
| Hemorrhagic infarction | 1 | 0.2 | 1 | 0.2 |
| Internal carotid thrombosis | 15 | 3.1 | 2 | 0.5 |
| Symptomatic | 9 | 1.9 | 0 | — |
| Asymptomatic | 6 | 1.2 | 2 | 0.5 |
| Miscellaneous | | | | |
| Cervical hematoma | 7 | 1.4 | 7 | 1.6 |
| Patch disruption | — | — | 3 | 0.7 |

TABLE 4. Selected Comparisons Concerning Perioperative Ischemic Stroke and Internal Carotid Thrombosis Among the 801 Patients Studied

| Patient Comparisons | N | No Patch (N = 433) | | Patch (N = 368) | |
|---------------------------------|-----|-----------------------|-----|--------------------|-----|
| | | No. | % | No. | % |
| Ischemic stroke | | | | | |
| Sex | | | | | |
| Men | 563 | 10/319 | 3.1 | 2/244 | 0.8 |
| Women | 238 | 5/114 | 4.4 | 1/124 | 0.8 |
| Neurologic status | | | | | |
| Asymptomatic stenosis | 408 | 4/211 | 1.9 | 1/197 | 0.5 |
| Previous TIA* | 252 | 7/134 | 5.2 | 1/118 | 0.8 |
| Prior stroke | 141 | 4/88 | 4.5 | 1/53 | 1.9 |
| Contralateral carotid | | | | | |
| Patent | 685 | 12/380 | 3.2 | 1/305 | 0.3 |
| Occluded | 116 | 3/53 | 5.7 | 2/63 | 3.2 |
| Perioperative thrombosis | | | | | |
| Sex | | | | | |
| Men | 563 | 13/319 | 4.1 | 1/244 | 0.4 |
| Women | 238 | 2/114 | 1.8 | 1/124 | 0.8 |
| Preoperative | | | | | |
| Antiplatelet therapy | | | | | |
| Yes | 443 | 8/249 | 3.2 | 1/194 | 0.5 |
| No | 358 | 7/184 | 3.8 | 1/174 | 0.6 |

* TIA = transient ischemic attack or amaurosis fugax.

Patch disruption. Semielective evacuation of simple cervical hematomas was necessary after 14 (1.5%) of the 917 procedures (7 VP, 1.6%; 7 NP, 1.4%). Another three patients (0.7%) in the patch group, however, developed acute false aneurysms during the early postoperative period and underwent urgent reoperations to correct this serious complication. One patient was found to have an unexplained rent in the center of the patch and awakened after its replacement with a contralateral monoparesis that resolved within the following week. The remaining two patients had anastomotic disruptions. One was repaired successfully but the other recurred because of infection 1 month later and required carotid ligation. Neither of these patients sustained neurologic deficits.

Clinical Correlations. The risk of early stroke or thrombosis is correlated with selected clinical features in Table 4. The results of patch angioplasty were measurably superior only with respect to the stroke rate among patients with patent contralateral internal carotid arteries (0.3% VP, 3.2% NP; $p = 0.0085$) and the incidence of postendarterectomy occlusion among men (0.4% VP, 4.1% NP; $p = 0.005$). Nevertheless, these data also suggested several other trends even though they could not be fully confirmed by statistical testing. Patch closure was associated with three to five times fewer perioperative strokes in women and in patients with previous neurologic symptoms. Moreover, patching appeared to discourage carotid thrombosis irrespective of whether antiplatelet agents had been administered before surgery

($0.05 < p < 0.1$). Considering all complications (death, stroke, thrombosis, patch disruption) and counting no patient twice, the combined surgical morbidity and mortality rate was 2.4% (9/368) when patches were applied and 6.0% (26/433) when they were not ($p = 0.015$).

Late Results

Survival. Cumulative survival and the number of eligible patients at each follow-up interval are illustrated in Figure 2. A total of 294 entrants were available for consideration during the third postoperative year, and the survival rates for the VP and NP cohorts at the conclusion of the study period were 80 and 87%, respectively. Attrition was comparable among symptomatic and asymptomatic surgical candidates, and none of the actuarial differences was statistically significant. Of 81 late deaths, 28 (35%) were attributed to cardiac disease, 11 (14%) to cancer, and nine (11%) to cerebrovascular events. Fatal strokes have occurred in only 1.1% of each treatment group (4 VP, 5 NP).

Neurologic events. There were 31 perioperative TIAs, strokes or deaths. Of the remaining 770 patients, 42 (5.4%) have experienced subsequent neurologic symptoms involving the ipsilateral ($N = 27$, 3.5%) or contralateral cerebral hemisphere ($N = 15$, 1.9%) during a mean follow-up interval of 20 months. Completed strokes have occurred on the same side as previous carotid endarterectomy in 11 (2.7%) of the 412 patients in the NP group and in seven (2.0%) of the 358 who received patch angioplasty. Figure 3 illustrates the cumulative incidence of all ipsilateral neurologic events in the entire series. None of the observed differences was statistically valid beyond the early postoperative period. Therefore, patching did not appear to influence the late stroke rate.

Recurrent stenosis. There were 21 perioperative deaths or internal carotid occlusions. Only ten (1.1%) of the remaining 896 endarterectomies (3 VP, 0.7%; 7 NP, 1.5%) have required reoperations for recurrent stenosis during the follow-up period (Fig. 4). As indicated in Table 5, however, objective reassessment has been obtained for just 332 arteries (37%) despite the fact that all patients were encouraged to schedule annual DSA or Doppler ultrasound examinations for at least 3 years after their original procedures. Recurrent lesions ($\geq 30\%$ of lumen diameter) have been documented with this approach in 4.8% of the VP group and in 14% of the NP group ($p = 0.0137$). Furthermore, the cumulative recurrence rate (Fig. 5) was 9% and 31%, respectively ($p = 0.0066$). The actuarial difference between the VP and NP cohorts (3.7% VP, 39% NP) was especially significant ($p = 0.0019$) when asymptomatic carotid stenosis had been the initial indication for surgical treatment.



FIG. 4. Intra-arterial digital subtraction angiogram demonstrating a high-grade "collar" stenosis (arrow) at the junction of the common carotid artery and the saphenous vein patch 1 year after carotid endarterectomy.

Discussion

Despite lingering controversy concerning its perceived overuse, countless studies have established that carotid endarterectomy is an exceedingly effective approach to stroke prevention in selected patients, provided its immediate risk meets acceptable standards. Previous reports from the Cleveland Clinic indicated that surgical treatment should be a serious consideration for either symptomatic or asymptomatic patients who have severe ($\geq 70\%$) carotid stenosis or advanced, bilateral bifurcation disease.¹⁸⁻²⁰ Moore et al.^{21,22} have presented similar data, but the fact remains that long-term benefit in appropriate candidates is greatly compromised by excessive complication rates at the centers in which their procedures are to be performed. Since acute or unstable strokes and chronic hemiplegic deficits now are universally accepted as contraindications to surgical intervention, poor results after extracranial reconstruction generally reflect imperfect surgical technique. There is abundant evidence that carotid endarterectomy *can* be done safely, and if surgeons are to respond effectively to their critics, it *must* be done safely.

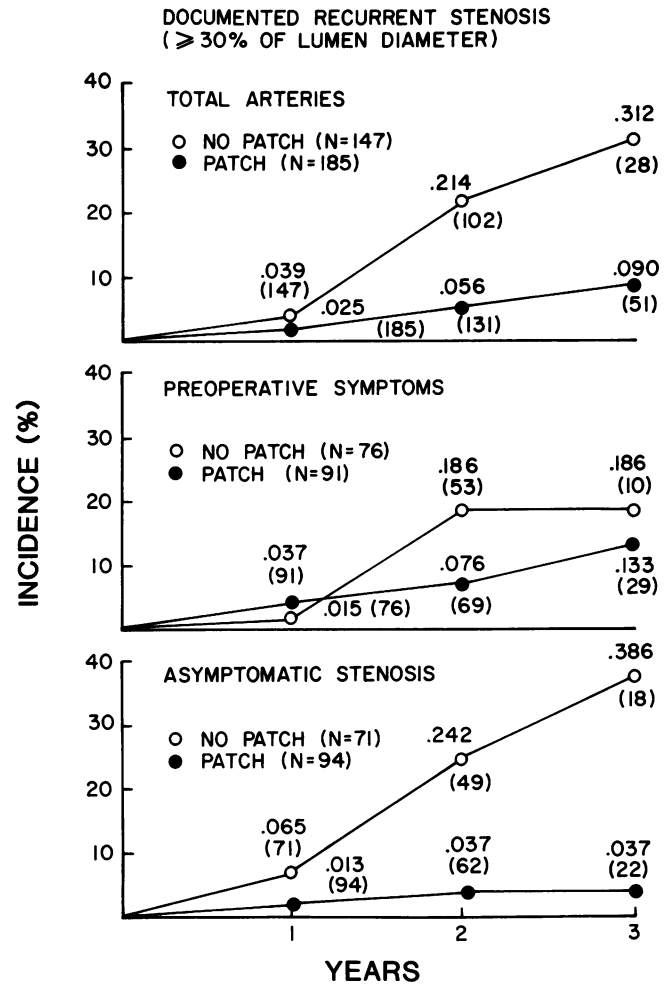


FIG. 5. The cumulative incidence of recurrent lesions ($\geq 30\%$ stenosis) in 322 arteries documented by objective carotid imaging. The number of vessels under consideration at each interval is enclosed in parentheses.

In addition to reservations about its early risk, there currently is speculation concerning the durability of carotid endarterectomy. Although large, retrospective in-

TABLE 5. Recurrent Stenosis During a 3-Year Interval Following 896 Carotid Endarterectomies*

| Recurrent Carotid Stenosis | No Patch (N = 466) | | Patch (N = 430) | |
|-----------------------------|--------------------|-----|-----------------|-----|
| | No. | % | No. | % |
| Reoperations | 7 | 1.5 | 3 | 0.7 |
| Objective follow-up imaging | 146 | 31 | 186 | 43 |
| Documented lesions | 21 | 14 | 9 | 4.8 |
| 30-49% | 5 | 3.4 | 4 | 2.2 |
| 50-69% | 6 | 4.1 | 1 | 0.5 |
| 70-89% | 6 | 4.1 | 1 | 0.5 |
| $\geq 90\%$ | 2 | 1.4 | 1 | 0.5 |
| Occlusion | 2 | 1.4 | 2 | 1.1 |

* Operative deaths and early occlusions have been omitted.

vestigations have suggested that the incidence of recurrent stenosis warranting reoperation is 3% or less, this conclusion necessarily was based on incomplete follow-up and imprecise criteria (new symptoms or cervical bruits) for diagnostic angiography.²³⁻²⁷ Since the introduction of noninvasive hemodynamic testing and carotid imaging, however, recurrence rates have been reported to be substantially higher than expected in small series of patients who were evaluated prospectively. According to several of these studies, recurrent stenosis exceeding 50% of lumen diameter occurred in 9-19% of patients within 2-5 years after conventional endarterectomy.²⁸⁻³² The vast majority of these lesions were caused by myointimal hyperplasia, and this complication appeared to be especially common in women and in cigarette smokers.^{33,34} Even if the perioperative risks of carotid endarterectomy were negligible, an unacceptable incidence of early hyperplastic recurrence undoubtedly would be cited as sufficient reason not to correct primary asymptomatic stenosis irrespective of its severity.

Sundt,³⁵ Imparato,³⁶ and their associates have long maintained that patch angioplasty was singularly important in the reduction of early morbidity and late recurrence throughout their extensive experience with carotid reconstruction. Archie³⁷ also found that vein patching eliminated neurologic complications and recurrent hyperplastic stenosis in his series of 200 prospectively randomized operations, and Deriu et al.³⁸ described similar success with prosthetic patch material following 86 procedures. In our study of over 900 endarterectomies, saphenous patch angioplasty was associated with perioperative strokes in 0.7% and internal carotid thrombosis in 0.5%. In comparison, early strokes ($p = 0.0084$) or occlusions ($p = 0.0027$) each occurred after 3.1% of routine arteriotomy closures. Although their clinical importance is not yet known, recurrent defects were objectively documented in only 4.8% of the patch group during the first 3 postoperative years, compared with 14% of the NP group ($p = 0.0137$).

This investigation has at least two unique features. First, it contains a sufficient number of patients and operations that its conclusions should be reliable. Second, neither treatment cohort was prejudiced by poor results. If VP angioplasty can make good results even better at our center, then it could substantially enhance the mediocre outcome that apparently is encountered at many others. The success of the saphenous patch probably is related to several factors. Clearly, it alters the configuration of the carotid bulb by making it larger.³⁶ In addition, its intimal surface may prevent early microembolic strokes and carotid thrombosis by discouraging platelet aggregation at the endarterectomy site. Finally, it may reduce the incidence of myointimal hyperplasia by promoting rapid endothelial coverage of the bifurcation.

The adjunctive use of a shunt is a logical consideration simply because patch angioplasty takes more time than primary closure. Accordingly, surgeons who ordinarily prefer not to shunt might choose to restrict patching to specific subsets of patients who seem to have the most to gain from this precaution. Women comprise one such group because of the small size of their carotid arteries and their propensity for hyperplastic recurrence, and patients with contralateral internal carotid occlusions are another because their risk for perioperative stroke is relatively high under any circumstances.^{39,40} Whatever the criteria for its use, our experience suggests that patch angioplasty helps to fulfill the high expectations that surgeons, patients, and critics alike have established for carotid endarterectomy: the early results must be perfect, and the late results have to stay that way.

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References

1. The EC/IC Bypass Study Group. Failure of extracranial-intracranial arterial bypass to reduce the risk of ischemic stroke. *N Engl J Med* 1985; 313:1191-1200.
2. Barnett HJM, Plum F, Walton JN. Carotid endarterectomy—an expression of concern. *Stroke* 1984; 15:941-943.
3. Warlow C. Carotid endarterectomy: does it work? *Stroke* 1984; 15:1068-1076.
4. Chambers BR, Norris JW. The case against surgery for asymptomatic carotid stenosis. *Stroke* 1984; 15:964-967.
5. Easton JD, Sherman DG. Stroke and mortality rate in carotid endarterectomy: 228 consecutive operations. *Stroke* 1977; 8:565-568.
6. Brott T, Thalinger K. The practice of carotid endarterectomy in a large metropolitan area. *Stroke* 1984; 15:950-955.
7. Brott TG, Labutta RJ, Kempczinski RF. Changing patterns in the practice of carotid endarterectomy in a large metropolitan area. *JAMA* 1986; 255:2609-2612.
8. Chambers BR, Norris JW. Outcome in patients with asymptomatic neck bruits. *N Engl J Med* 1986; 315:860-888.
9. Turnipseed WD, Berkoff HA, Crummy A. Postoperative occlusion after carotid endarterectomy. *Arch Surg* 1980; 115:573-574.
10. Ortega G, Gee W, Kaupp HA, McDonald KM. Postendarterectomy carotid occlusion. *Surgery* 1981; 90:1093-1098.
11. Barnes RW, Nix ML, Wingo JP, Nichols BT. Recurrent *versus* residual stenosis. Incidence detected by Doppler ultrasound. *Ann Surg* 1986; 203:652-660.
12. Dille RB, Bernstein EF. A comparison of B-mode real-time imaging and arteriography in the intraoperative assessment of carotid endarterectomy. *J Vasc Surg* 1986; 4:457-462.
13. Flanagan DP, Douglas DJ, Machi J, et al. Intraoperative ultrasonic imaging of the carotid artery during carotid endarterectomy. *Surgery* 1986; 100:893-898.
14. Hertzler NR, Beven EG, O'Hara PJ, et al. Early patency of the carotid artery after endarterectomy: digital subtraction angiography after two hundred sixty-two operations. *Surgery* 1982; 92:1049-1057.
15. Painter TA, Hertzler NR, O'Hara PJ, et al. Symptomatic internal carotid thrombosis after carotid endarterectomy. *J Vasc Surg* 1987; 5:445-451.

16. Cutler SJ, Ederer F. Maximum utilization of the life table method in analyzing survival. *J Chronic Dis* 1958; 8:699-712.
17. Lee ET, Desu MM. A computer program for comparing K samples with right-censored data. *Comput Programs Biomed* 1972; 2:315-321.
18. Hertzler NR, Arison R. Cumulative stroke and survival ten years after carotid endarterectomy. *J Vasc Surg* 1985; 2:661-668.
19. Hertzler NR, Flanagan RA Jr, Beven EG, O'Hara PJ. Surgical versus nonoperative treatment of symptomatic carotid stenosis: 211 patients documented by intravenous angiography. *Ann Surg* 1986; 204:154-162.
20. Hertzler NR, Flanagan RA Jr, Beven EG, O'Hara PJ. Surgical versus nonoperative treatment of asymptomatic carotid stenosis: 290 patients documented by intravenous angiography. *Ann Surg* 1986; 204:163-171.
21. Moore DJ, Sheehan MP, Kolm P, et al. Are strokes predictable with noninvasive methods: a five-year follow-up of 303 unoperated patients. *J Vasc Surg* 1985; 2:654-660.
22. Moore DJ, Miles RD, Gooley NA, Sumner DS. Noninvasive assessment of stroke risk in asymptomatic and nonhemispheric patients with suspected carotid disease. Five-year follow-up of 294 unoperated and 81 operated patients. *Ann Surg* 1985; 202:491-504.
23. Stoney RJ, String ST. Recurrent carotid stenosis. *Surgery* 1976; 80:705-710.
24. Cossman D, Callow AD, Stein A, Matsumoto G. Early restenosis after carotid endarterectomy. *Arch Surg* 1978; 113:275-278.
25. Hertzler NR, Martinez BD, Benjamin SP, Beven EG. Recurrent stenosis after carotid endarterectomy. *Surg Gynecol Obstet* 1979; 149:360-364.
26. Das MB, Hertzler NR, Ratliff NB, et al. Recurrent carotid stenosis. A five-year series of 65 reoperations. *Ann Surg* 1985; 202:28-35.
27. Piepgras DG, Sundt TM Jr, Marsh WR, et al. Recurrent carotid stenosis. Results and complications of 57 operations. *Ann Surg* 1986; 203:205-213.
28. Kremen JE, Gee W, Kaupp HA, McDonald KM. Restenosis or occlusion after carotid endarterectomy. A survey with ocular pneumoplethymography. *Arch Surg* 1979; 114:608-610.
29. Canatello NL, Cutler BS, Wheeler HB, et al. Noninvasive detection of carotid stenosis following endarterectomy. *Arch Surg* 1981; 116:1005-1008.
30. Zierler RE, Bandyk DF, Thiele BL, Strandness DE Jr. Carotid artery stenosis following endarterectomy. *Arch Surg* 1982; 117:1408-1415.
31. Colgan MP, Kingston V, Shanik G. Stenosis following carotid endarterectomy. Its implication in management of asymptomatic carotid stenosis. *Arch Surg* 1984; 119:1033-1035.
32. Ackroyd N, Lane R, Appleberg M. Carotid endarterectomy. Long-term follow-up with specific reference to recurrent stenosis, contralateral progression, mortality and recurrent neurological episodes. *J Cardiovasc Surg* 1986; 27:418-425.
33. Clagett GP, Rich NM, McDonald PT, et al. Etiologic factors for recurrent carotid artery stenosis. *Surgery* 1983; 93:313-318.
34. Thomas M, Otis SM, Rush M, et al. Recurrent carotid artery stenosis following endarterectomy. *Ann Surg* 1984; 200:74-79.
35. Sundt TM, Houser OW, Whisnant JP, Fode NC. Correlation of postoperative and two-year follow-up angiography with neurologic function in 99 carotid endarterectomies in 86 consecutive patients. *Ann Surg* 1986; 203:90-100.
36. Imparato AM, Weinstein GS. Clinicopathologic correlation in postendarterectomy recurrent stenosis. A case report and bibliographic review. *J Vasc Surg* 1986; 3:657-662.
37. Archie JP. Prevention of early restenosis and thrombosis-occlusion after carotid endarterectomy by saphenous vein patch angioplasty. *Stroke* 1986; 17:901-905.
38. Deriu GP, Ballotta E, Bonavina L, et al. The rationale for patch-graft angioplasty after carotid endarterectomy: early and long-term follow-up. *Stroke* 1984; 15:972-979.
39. Hertzler NR, Beven EG, Greenstreet RL, Humphries AW. Internal carotid back pressure, intraoperative shunting, ulcerated atheromata, and the incidence of stroke during carotid endarterectomy. *Surgery* 1978; 83:306-312.
40. Baker WH, Littooy FN, Hayes AC, et al. Carotid endarterectomy without a shunt: the control series. *J Vasc Surg* 1984; 1:50-56.