CAROTID ENDARTERECTOMY in managing extracranial occlusive vascular disease is well established. Although carotid endarterectomy does not prolong survival, several centers have demonstrated that patients who undergo surgery without neurologic morbidity or mortality have a significantly decreased risk of subsequent transient or permanent neurologic deficit compared to similar patients managed nonsurgically. However, the decision to recommend operation remains controversial among clinicians. Interest in the feasibility of operative treatment for extracranial arterial occlusive lesions was stimulated by the reports of Fisher, who called attention to the frequent association of extracranial carotid lesions with symptomatic cerebral ischemia and the probable role of these lesions in the pathogenesis of cerebral infarction. Early surgical reports noted a decrease in the instances of transient ischemic attacks (TIAs) after carotid endarterectomy, but the overall neurologic morbidity and mortality were not convincingly improved when the operative and angiographic risks were taken into account. A review of the published morbidity and mortality figures following carotid endarterectomy vary from a 0.8% permanent neurologic morbidity and a 1.9% procedure mortality in 257 operations to a 1% permanent neurologic morbidity and 19% procedure mortality in 227 operations. This disparity in results related in part to variation in the population, patient selection, change in surgical techniques, and the preoperative neurologic status of the patient. The data are, nonetheless, troubling in weighing the therapeutic alternatives for individual patients. If one compares the arteriographic and surgical risks of neurologic morbidity and mortality with the risk of stroke following nonsurgical management, and finds that the surgical and arteriographic risks are equal to or greater than those of stroke with nonsurgical therapy, then the rational of surgical therapy has to be reconsidered. The questions that must be answered are whether the older surgical series overstate the surgical morbidity and mortality that actually exist in current practice, and whether the surgical risks vary significantly with the surgeon and/or the institution.

This report reviews our own data and surveys the literature in order to evaluate the place of operation in the management of extracranial cerebrovascular disease.

Methods

The charts of 147 consecutive patients who underwent 186 carotid endarterectomies at the San Francisco VA Hospital from 1967-75 were reviewed. Patients who underwent aortic arch reconstruction and/or combined cardiovascular procedures were excluded. The patients ranged in age from 40 to 91 years (mean age 62). All but 3 were male. The indications for surgery were: amaurosis fugax, hemispheric TIA, completed cerebrovascular accidents, non-lateralizing symptoms with demonstrable extracranial occlusive lesions, or asymptomatic patients with extracranial occlusive lesions. The only contraindications to surgery were terminal disease, massive cerebral infarction, or occlusion of the appropriate carotid artery.
All patients underwent preoperative selective bilateral carotid arteriograms with intracranial views. The standard Seldinger percutaneous retrograde femoral catheter technique was utilized. Aortic arch and/or vertebral injections were performed only when the carotid arteriograms failed to demonstrate lesions which appropriately explained the neurologic symptomatology. All arteriograms included at least 2 and often 3 views of both carotid bifurcations, as well as anterior-posterior and lateral intracranial views. All cerebral arteriograms were performed by radiology residents under the supervision of a staff neuroradiologist.

During the early part of the series 36 carotid artery operations were performed under local anesthesia. The subsequent 150 operations were performed under general anesthesia. The operative techniques have been adequately described in the literature. Since 1971 all operations were performed under general anesthesia (113) and were done with the patients normotensive and normocapnic. A carotid intralumenal bypass shunt was utilized only in patients whose internal carotid artery backpressure was less than 25 mm Hg or in patients with previous ipsilateral infarction. All patients had perioperative arteriograms to assess the technical results before closure of the incision. All surgical procedures were performed by general surgery residents under supervision of a vascular surgery staff member who actively participated in every operation.

Surgical and Arteriographic Results

The morbidity and mortality from operation are summarized in table 1. One patient (1/147, 0.7%) died from a postoperative myocardial infarction. Seven patients (7/186, 3.7%) suffered postoperative strokes with mild but permanent neurologic defects, and 3 patients (3/186, 1.6%) experienced a postoperative transient ischemic event (less than 24 hours).

If the surgical data are subdivided into 2 successive time periods, 1967–70 and 1971–75, corresponding to a change in surgical technique with respect to the selective use of an indwelling carotid shunt, the surgical results of the 1971–75 period are dramatically improved in comparison to the earlier 1967–70 period. During 1967–70 there were 73 operations performed on 55 patients. The postoperative mortality and permanent neurologic complication rate were 1.4% and 8.2%, respectively. Since 1971, however, there have been 113 consecutive carotid endarterectomies in 92 patients with no postoperative deaths (0%), only one stroke (0.9%), and one transient ischemic attack (0.9%). These data are summarized in table 2.

As part of an independent multi-institutional study of the morbidity and mortality of catheter cerebral angiography, the San Francisco VA Hospital cerebral arteriogram series for 1975 was reviewed. There were 4 transient neurologic complications for a rate of 1.3%. There are no arteriographically related deaths or permanent strokes.

The combined risk of death and stroke from carotid endarterectomy and cerebral arteriography at the San Francisco VA Hospital during the period 1971–75 could be extrapolated to be 0.9%. The risk of postangiogram and postoperative transient ischemic attacks was 2.2%, for a total complication rate of 3.1% (table 3).

Discussion

Part of the decision for surgical versus nonsurgical therapy must include a review of the incidence and natural history of transient ischemic attacks and stroke. The occurrence of hemispheric transient ischemic attacks (TIAs) is helpful in identifying lesions at risk; however, not all TIAs proceed to stroke, nor are all strokes preceded by warning TIAs. Fields et al. noted that only 46% of their patients with stroke had prior TIAs; 54% with stroke had no prior neurologic symptoms. On the other hand, it has been estimated that 24–37% of the patients with TIAs will subsequently have a stroke. In a 3-year follow up of patients with active TIAs, Ziegler and Hanssen noted that 15% of the patients developed a stroke. In those patients with prior TIAs, Whisnant noted that one-third of the patients went on to have strokes, 21% of those strokes occurring within the first month after onset of symptoms, and 51% of those strokes occurring within the first year after the onset of symptomatic TIAs. In those patients with prior cerebrovascular accidents, Matsumoto et al. noted a recurrent stroke rate of 10% during the first year and 20% over the first 5 years (4% per year). It would appear that in those patients with either active TIAs

<table>
<thead>
<tr>
<th>Years</th>
<th>Number of patients</th>
<th>Number of endarterectomies</th>
<th>Neurologic transient</th>
<th>Complications permanent</th>
<th>Patient mortality</th>
<th>Procedure mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1967–70</td>
<td>55</td>
<td>73</td>
<td>2 (2.7%)</td>
<td>6 (8.2%)</td>
<td>1 (1.8%)</td>
<td>1 (1.4%)</td>
</tr>
<tr>
<td>1971–75</td>
<td>92</td>
<td>113</td>
<td>1 (0.9%)</td>
<td>1 (0.9%)</td>
<td>0 (0.0%)</td>
<td>0 (0.0%)</td>
</tr>
</tbody>
</table>

Table 2 Permanent Neurologic Morbidity and Mortality of Carotid Endarterectomy at the San Francisco VA Hospital 1967–1975.
or prior stroke, the risk of subsequent stroke without surgical treatment averages 5% per year. However, up to 21% of the strokes after TIA occur in the first month after the onset of symptoms and 40–50% of the strokes occurring after prior stroke or onset of TIAs occur within the first year after the onset of symptoms. This indicates that the first month and, certainly, the first year, represent periods of maximum risk. Therefore, while the subsequent stroke rate can be approximated at 5% per year for the first 5 years after either stroke or the onset of TIAs, the risk during the first year after the onset of symptoms is probably as high as 15%.

In a randomized study evaluating surgical and medical therapy, Fields et al. noted that the risk of permanent neurologic morbidity and mortality in the medical and surgical groups became equal at the end of 3.5 years. It can be argued, however, that these data reflect both early surgical results as well as variable results from multiple centers, and that more recent surgical results would favor surgical over nonsurgical management. In an effort to identify the trends in operative morbidity and mortality, we reviewed the published series of carotid operations from 1968 to 1977. The data are summarized in table 4. Analysis shows that 3,820 carotid endarterectomies were performed on 3,233 patients with an average permanent neurologic complication rate of 5.7%, a patient mortality rate of 3.4%, and a procedure mortality rate of 2.9%. However, a more careful evaluation of the same data reveals that both permanent neurologic deficit and mortality vary greatly, from one series to another and with technical improvements associated with the passage of time. That surgical results have improved with time is emphasized by table 5, where review of our data and that of DeWese et al. clearly demonstrates significant decrease in both permanent neurologic complications and mortality after carotid endarterectomy between 1968 and 1975. The variability of surgical results based upon the neurologic status of the patient preoperatively is shown in table 6. Review of the data by DeWese et al., Thompson et al., and Easton et al. demonstrates that the neurologic complication rate and postoperative mortality in those patients with a prior stroke or increasing neurologic deficit is at least 2–3 times higher than in patients with only TIAs, stable neurologic deficit, or asymptomatic lesions. The significance of institutional variability is demonstrated by two reports from The Second Joint Meeting on Stroke and Cerebral Circulation (Miami, FL, Feb 25–26, 1977). Fleming et al. reported 240 carotid endarterectomies from the University of Toronto with an operative mortality of 1.6% and neurologic morbidity of 1.6%. Easton and Sherman reported 228 carotid endarterectomies from 2 large community hospitals in Springfield, Illinois; in that series the risk of operative mortality and stroke were 6.6% and 14.5%, respectively, and there appeared to

**Table 4 Neurologic Morbidity and Mortality of Carotid Endarterectomy in Published Series: 1968-1973**

<table>
<thead>
<tr>
<th>Author</th>
<th>(Ref. no.)</th>
<th>Year</th>
<th>Number of endarterectomies</th>
<th>Number of patients</th>
<th>Neurologic transient complications</th>
<th>Complications permanent</th>
<th>Patient mortality</th>
<th>Procedure mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>DeWeese JA et al.</td>
<td>(3)</td>
<td>1968</td>
<td>227</td>
<td>205</td>
<td>18 (7.9%)</td>
<td>43 (18.9%)</td>
<td>23 (11.2%)</td>
<td>23 (10.1%)</td>
</tr>
<tr>
<td>Rainer WG et al.</td>
<td>(6)</td>
<td>1968</td>
<td>257</td>
<td>208</td>
<td>7 (2.7%)</td>
<td>2 (0.8%)</td>
<td>5 (2.4%)</td>
<td>5 (1.9%)</td>
</tr>
<tr>
<td>Galbraith JC et al.</td>
<td>(25)</td>
<td>1969</td>
<td>354</td>
<td>265</td>
<td>—</td>
<td>21 (6.3%)</td>
<td>10 (3.8%)</td>
<td>10 (3.0%)</td>
</tr>
<tr>
<td>Fields WF et al.</td>
<td>(5)</td>
<td>1970</td>
<td>214</td>
<td>169</td>
<td>3 (1.4%)</td>
<td>13 (6.0%)</td>
<td>6 (3.5%)</td>
<td>6 (2.8%)</td>
</tr>
<tr>
<td>Thompson JE et al.</td>
<td>(12)</td>
<td>1970</td>
<td>748</td>
<td>592</td>
<td>4 (0.5%)</td>
<td>20 (2.7%)</td>
<td>20 (3.4%)</td>
<td>20 (2.7%)</td>
</tr>
<tr>
<td>Tytus JS et al.</td>
<td>(4)</td>
<td>1970</td>
<td>44</td>
<td>44</td>
<td>8 (18.0%)</td>
<td>12 (27.0%)</td>
<td>5 (11.0%)</td>
<td>5 (11.0%)</td>
</tr>
<tr>
<td>DeWeese JA et al.</td>
<td>(11)</td>
<td>1973</td>
<td>105</td>
<td>103</td>
<td>15 (14.3%)</td>
<td>6 (5.7%)</td>
<td>1 (1.0%)</td>
<td>1 (1.0%)</td>
</tr>
<tr>
<td>Howe JR et al.</td>
<td>(13)</td>
<td>1974</td>
<td>73</td>
<td>62</td>
<td>0 (0.0%)</td>
<td>1 (1.4%)</td>
<td>0 (0.0%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Toole JP et al.</td>
<td>(26)</td>
<td>1975</td>
<td>82</td>
<td>82</td>
<td>5 (6.0%)</td>
<td>5 (6.0%)</td>
<td>5 (6.0%)</td>
<td>5 (6.0%)</td>
</tr>
<tr>
<td>Baker W et al.</td>
<td>(27)</td>
<td>1977</td>
<td>304</td>
<td>272</td>
<td>9 (3.0%)</td>
<td>5 (1.6%)</td>
<td>2 (0.7%)</td>
<td>2 (0.6%)</td>
</tr>
<tr>
<td>Matsumoto GH et al.</td>
<td>(9)</td>
<td>1977</td>
<td>130</td>
<td>130</td>
<td>6 (4.6%)</td>
<td>2 (1.5%)</td>
<td>2 (1.5%)</td>
<td>2 (1.5%)</td>
</tr>
<tr>
<td>Piroleau WH et al.</td>
<td>(8)</td>
<td>1977</td>
<td>317</td>
<td>240</td>
<td>—</td>
<td>34 (10.7%)</td>
<td>10 (4.1%)</td>
<td>10 (3.0%)</td>
</tr>
<tr>
<td>Suntz TA et al.</td>
<td>(28)</td>
<td>1977</td>
<td>331</td>
<td>331</td>
<td>2 (0.6%)</td>
<td>10 (3.0%)</td>
<td>2 (0.0%)</td>
<td>2 (0.6%)</td>
</tr>
<tr>
<td>Fleming JFR et al.</td>
<td>(24)</td>
<td>1977</td>
<td>240</td>
<td>189</td>
<td>—</td>
<td>4 (1.6%)</td>
<td>3 (1.6%)</td>
<td>3 (1.3%)</td>
</tr>
<tr>
<td>Easton JD et al.</td>
<td>(23)</td>
<td>1977</td>
<td>228</td>
<td>194</td>
<td>—</td>
<td>32 (14.5%)</td>
<td>15 (6.6%)</td>
<td>15 (6.6%)</td>
</tr>
<tr>
<td>West H et al.</td>
<td>(23)</td>
<td>1977</td>
<td>186</td>
<td>147</td>
<td>3 (1.6%)</td>
<td>7 (3.7%)</td>
<td>1 (0.7%)</td>
<td>1 (0.5%)</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td></td>
<td></td>
<td>3,233</td>
<td>3,820</td>
<td>80 (2.9%)</td>
<td>1218 (32.8%)</td>
<td>110 (3.4%)</td>
<td>110 (2.9%)</td>
</tr>
</tbody>
</table>
be no trend toward improvement from 1970 to 1976. Easton and Sherman suggested that their operative stroke and mortality rate following carotid endarterectomy was probably representative of the results from other community hospitals in the United States during the 1970's. Clearly, the risk of operation demonstrated by Fleming et al. is acceptable, and in their hands surgical treatment offers decreased morbidity and mortality when compared to medical treatment for TIA and stroke. The operative results reported by Easton and Sherman, however, are unacceptable and, in fact, their reported surgical risk is 2-3 times that of nonsurgical management.

Unquestionably, the choice of treatment must be based upon the risks of permanent neurologic deficit and/or death with or without surgical therapy. The literature demonstrates that the stroke rate for nonsurgical patients after TIA or prior stroke approaches 5% per year, and may be as high as 15% in the first year after the onset of symptoms. In order for surgery to be considered a preferred therapy, the risk of operation must be considerably less and represent an improvement over the natural history of the disease. Averaged data from published surgical results between 1968 and 1977 show the combined incidence of postoperative stroke and death to be 9.1% (table 4). In other words, the surgical risk approaches the risk of expectant treatment for one year, but it is significantly lower than the nonsurgical risk for 5 years (est. 25%). The surgical results, however, show a range in the permanent neurologic deficit of 0.8% to 27%, and in surgical patient mortality from 0.0% to 11.2%. Analysis of our own data shows that the combined risk of postarteriographic TIAs, postoperative permanent neurologic deficit, and mortality is only 3.1%, or approximately 1/5 the risk of stroke in year one for patients managed nonsurgically after the onset of active symptoms. In our institution, therefore, we are surgically aggressive when appropriate neurologic symptoms are felt to be caused by extracranial occlusive and/or ulcerative atherosclerotic vascular disease.

The message is clear, but deserves emphasis: when one considers the therapeutic alternatives for treatment of extracranial vascular occlusive disease, operation will provide considerable advantage in most patients over expectant management provided that there is careful selection of both the surgeon and the institution. It is well documented that postoperative neurologic morbidity and mortality vary from surgeon to surgeon and from institution to institution. In addition, the surgical results vary significantly with the preoperative neurologic status of the patient. As one might suspect, the surgical results have improved with time, and both the operative mortality and risk of permanent neurologic morbidity have decreased significantly from the early reports of carotid endarterectomy. Analysis of the available data shows that, depending upon which surgical series is selected as a basis of comparison, results can be used to justify either operation or no operation. However, the data from our institution and from other centers which do a large volume of vascular surgery substantiate the conclusion that, in the appropriate setting, carotid endarterectomy can be accomplished with a significant reduction in the risk of permanent neurologic deficit or mortality as compared to medical management.

### Table 5 Improvements in Neurologic Morbidity and Mortality for Carotid Endarterectomy with Time

<table>
<thead>
<tr>
<th>Author</th>
<th>(Ref. no.)</th>
<th>Year</th>
<th>Number of endarterectomies</th>
<th>Number of patients</th>
<th>Neurologic transient</th>
<th>Complications permanent</th>
<th>Patient mortality</th>
<th>Procedure mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>DeWeese JA et al.</td>
<td>(3)</td>
<td>1968</td>
<td>227</td>
<td>205</td>
<td>7.9%</td>
<td>18.9%</td>
<td>11.2%</td>
<td>10.1%</td>
</tr>
<tr>
<td></td>
<td>(11)</td>
<td>1973</td>
<td>105</td>
<td>103</td>
<td>14.3%</td>
<td>5.7%</td>
<td>1.0%</td>
<td>1.0%</td>
</tr>
<tr>
<td>West H et al.</td>
<td></td>
<td>1967-70</td>
<td>73</td>
<td>55</td>
<td>2.7%</td>
<td>8.2%</td>
<td>1.8%</td>
<td>1.4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1971-75</td>
<td>113</td>
<td>92</td>
<td>0.9%</td>
<td>0.9%</td>
<td>0.0%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

### Table 6 Differences in Neurologic Morbidity and Mortality Based Upon Pre-Operative Neurologic Status

<table>
<thead>
<tr>
<th>Author</th>
<th>(Ref. no.)</th>
<th>Number of endarterectomies</th>
<th>Number of patients</th>
<th>Neurologic transient</th>
<th>Complications permanent</th>
<th>Patient mortality</th>
<th>Procedure mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>DeWeese JA</td>
<td>(3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIA*/Asymptomatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Progressive deficit</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>or completed CVA**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thompson JE</td>
<td>(12)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td>486</td>
<td>375</td>
<td>4 (0.8%)</td>
<td>9 (1.8%)</td>
<td>5 (1.3%)</td>
<td>5 (1.0%)</td>
</tr>
<tr>
<td>Frank CVA**</td>
<td></td>
<td>262</td>
<td>217</td>
<td>0 (0.0%)</td>
<td>11 (4.2%)</td>
<td>16 (7.4%)</td>
<td>16 (6.1%)</td>
</tr>
<tr>
<td>Easton JD</td>
<td>(23)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIA*</td>
<td></td>
<td>57</td>
<td>57</td>
<td>—</td>
<td>8 (14.0%)</td>
<td>4 (7.0%)</td>
<td>(7.0%)</td>
</tr>
<tr>
<td>CVA** (Severe)</td>
<td></td>
<td>12</td>
<td>12</td>
<td>—</td>
<td>—</td>
<td>5 (41.5%)</td>
<td>5 (41.5%)</td>
</tr>
</tbody>
</table>

*Transient Ischemic Attack
**Cerebrovascular Accident
References

Comparative risk of operation and expectant management for carotid artery disease.
H West, R Burton, A J Roon, J M Malone, J Goldstone and W S Moore

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