Carotid Endarterectomy: Perioperative and Anesthetic Considerations

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Carotid endarterectomy (CEA) is increasingly performed in the United States and worldwide. In 1992, more than 90,000 CEs were performed in the United States at an average cost of about $15,000 per procedure. Because this procedure is becoming more common, anesthesiologists can benefit from a review of current practice and controversies in this field that can guide anesthetic strategy. This review discusses the natural history of carotid artery disease, current treatment modalities, and important issues in preoperative evaluation, with an emphasis on the problems posed by the patient with both severe carotid artery disease and severe coronary artery disease. Intraoperative and anesthetic management for CEA, including potential methods of cerebral and myocardial protection and the relative merits of different anesthetic techniques, as well as immediate postoperative management, are considered.

DEFINITION, PROGNOSIS, AND TREATMENT OF CAROTID ARTERY DISEASE

The most common cause of carotid artery occlusive disease is atherosclerosis, a systemic and progressive disease. It is bilateral in about half of all cases. The atherosclerotic plaque usually develops at the lateral aspect of the carotid artery bifurcation (Fig 1) and extends up into the internal and external carotid arteries (Fig 2). The plaque is composed of both cellular and acellular elements deposited in the subendothelial wall of the artery. Cellular elements principally consist of monocytes, fibrocytes, and smooth muscle cells; acellular elements are calcium, collagenous protein, lipids, and cholesterol. A fibrous cap develops at the interface between the blood and the intimal plaque. Disruption of the fibrous cap over a lipid deposition can lead to ulceration within the plaque. The embolization of thrombotic material or debris from the plaque can result in stroke or transient neurologic symptoms. The mechanisms of disease progression are not completely understood, but progressive disease is associated with increases in both cellular and acellular components of the plaque.

A number of risk factors for the development of the disease have been identified. There is a positive correlation with age; the disease is more common and more severe in patients older than 70 years. Gender also plays a role: Men are more often afflicted, whereas affected women appear to have more severe disease. Almost two thirds of all patients scheduled for CEA are hypertensive and smoke cigarettes. Elevated serum lipid levels and a history of diabetes mellitus also appear to increase the risk, but they are less powerful predictors of carotid artery atherosclerosis than cigarette smoking and hypertension. Finally, the prevalence of significant carotid artery stenosis is higher in patients with left main coronary artery disease and those with peripheral vascular disease.

In many patients, carotid disease manifests only as an asymptomatic bruit over the affected artery. Some patients may report amaurosis fugax (attacks of temporary monocular blindness) from embolization to the ophthalmic artery, the first branch of the internal carotid artery. Other patients may experience episodes of paresis and clumsiness of the extremities, often accompanied by speech problems that resolve spontaneously without sequelae after a short period of time. These symptoms are typical of the classical transient ischemic attack (TIA). The difference between a TIA and a reversible ischemic neurologic deficit is one of degree and to some extent arbitrary. A TIA is said to resolve without neurologic deficit within 24 hours, whereas reversible ischemic neurologic deficit will last longer than 24 hours before complete resolution. Unfortunately, in some patients, the first manifestation of carotid artery disease may be a stroke. The term “stroke” or “completed stroke” ordinarily describes a focal neurologic deficit with an abrupt onset that has become stable and permanent over time.

Two pathophysiologic mechanisms are believed to be responsible for the development of neurologic symptoms: embolism and hypoperfusion. In embolic disease, degenerating changes in atheromatous plaque lead to luminal thrombus formation with subsequent dislodgment of the thrombus and occlusion of the distal cerebral circulation. Alternatively, a high-grade (preocclusive) obstruction of the carotid lumen may reduce distal blood flow below a critical level, especially when systemic blood pressure is low. It is hypothesized that symptomatic patients without significant stenosis suffer from embolic disease and may be relatively tolerant of reduced systemic blood pressure.
Conversely, symptomatic patients with critical stenosis of the artery may have a pressure-dependent cerebral circulation that may put them at risk from transient bouts of hypotension.

Prognosis for Patients With Carotid Artery Disease

Patients who present with TIAs have approximately a 10% risk of stroke during the subsequent year. Of patients who have a stroke after the onset of TIAs, approximately 20% have the stroke within the first month and about 50% within a year of symptoms. In subsequent years, the stroke risk decreases to about 5% per year. In patients whose first manifestation of disease is a stroke, the risk of recurrent stroke is about 10% each year with a 5-year repeat stroke rate of about 50%. In asymptomatic patients with a stenosis of more than 75%, the stroke risk is about 5% per year. In patients with an asymptomatic stenosis of less than 75%, the risk is 1% to 2% per year. Patients with ulcerated soft plaques (plaques that contain considerable amounts of cellular debris) appear to have a relatively higher risk of stroke, which approaches 7% to 8% per year. The serious nature of cerebrovascular disease is underscored by the fact that about one third of all acute strokes are fatal, and another third result in significant residual morbidity. An isolated, cervical bruit in asymptomatic patients also appears to be associated with a higher risk of stroke, at least with regard to men, but the correlation between the location of the bruits and the type of subsequent stroke is poor. It is well documented that a bruit of itself does not define the presence of a critical carotid lesion, and, conversely, critical carotid lesions are not always associated with a bruit. As a result, further testing is performed to evaluate the precise nature of the suspected carotid lesion. At present, the most commonly used noninvasive test is the duplex scan, which combines application of beta-mode imaging and pulsed Doppler spectral analysis. The beta-mode image offers a two-dimensional image of the carotid artery, and Doppler testing evaluates blood flow within the artery. The extent of the stenosis is assessed by evaluating velocity and turbulence of the blood flow. Reportedly, the accuracy of duplex scanning reaches 95% in experienced hands when compared with angiography. Other tests that are used include oculoplethysmography, computed tomography, and magnetic resonance imaging. Although all these tests are useful and the duplex scan in particular is highly accurate, angiography remains the single best method of carotid evaluation, especially with regard to measuring the size and morphology of the atheromatous plaque.

Treatment Modalities

Pharmacologic and surgical treatments are available for carotid artery disease. In the European Stroke Prevention Study, the combined administration of acetylsalicylic acid and dipyridamole, when compared with placebo, reduced the incidence of TIAs. Numerous trials are presently being conducted to determine the value of chronic anticoagulation with warfarin in decreasing the risk of repeated stroke. However, definitive results of these trials are not yet available. For further information on this topic, the reader is referred to an excellent recent review article.

In contrast, surgical treatment (CEA), in conjunction with aspirin therapy, has been proven to be clearly indicated and superior to medical therapy alone in symptomatic
Patients with a stenosis greater than 70%. For symptomatic patients with a stenosis of less than 70%, definitive treatment guidelines are not expected until 1997 from the North American Trial.

The treatment for asymptomatic patients with a stenosis equal to or larger than 50% is less clear. The Carotid Artery Stenosis With Asymptomatic Narrowing: Operation Versus Aspirin (CASANOVA) study was not able to demonstrate that CEA was superior to medical therapy in this patient population. One randomized trial that compared medical and surgical therapy, the Mayo Clinic Trial of asymptomatic patients with severe (≥ 70%) carotid stenosis, was terminated early because of a higher incidence of myocardial infarction and transient cerebral ischemic events in the surgical group. Most of the events were not temporally connected to the surgical procedure but appeared to be associated with the absence of aspirin use in the surgical group. Therefore, the investigators recommended that patients scheduled for CEA who are taking aspirin should continue the use of aspirin throughout and beyond the perioperative period unless clear contraindications to aspirin are present. Finally, the Veterans Cooperative Study group concluded that CEA in combination with medical treatment was superior to medical therapy alone because of a reduction in the combined rate of transient and permanent neurologic events in the surgical group. However, the study has been criticized because it did not show a significant difference between the treatment modalities when stroke was the sole end point. In late 1994, a fourth trial, the Asymptomatic Carotid Atherosclerosis Study (ACAS), was terminated early because it detected a benefit from CEA as compared with medical treatment in asymptomatic patients with a stenosis greater than or equal to 60%.

In summary, rigorous scientific proof is needed that CEA is of value in asymptomatic patients with a stenosis of less than 60%. Nonetheless, many centers perform operations on such patients with good results. Of course, such operations are only justifiable if the operative morbidity and mortality are lower than the natural risk of these events in the untreated patient. For a thorough review of this area, the reader is referred to the guidelines recently published for CEA by the American Heart Association.

Predictors of Outcome After Carotid Endarterectomy

Several factors appear to be associated with increases in the morbidity and mortality of CEA. However, the true prognostic value of any single, individual factor is controversial because its significance has been both confirmed and denied by different investigators. One study found that the presence of two or more of the following risk factors was associated with a nearly twofold increase in the risk of postoperative in-hospital stroke, myocardial infarction, or death: age 75 years or older, preoperative neurologic symptom status (higher risk for patients with ipsilateral symptoms than for patients with asymptomatic or nonipsilateral symptoms), history of angina, severe hypertension with a preoperative diastolic blood pressure of greater than 110 mmHg, CEA performed in preparation for coronary artery bypass surgery, history of angina, evidence of internal carotid artery thrombus, and internal carotid artery stenosis near the carotid siphon. Another recent study of patients with ipsilateral carotid artery distribution symptoms confirmed that patients over the age of 75 had a greater surgical risk than younger ones because of a greater risk of perioperative myocardial infarction, but not of stroke or death in the older patients. Adverse outcomes were also more common in patients with complete contralateral carotid occlusions, ipsilateral intraluminal thrombus, or ipsilateral carotid stenosis. Patients with one or more of the enumerated risk factors had a twofold risk of perioperative complications. Equally important, these investigators found that the following factors were insignificant for outcome: type of neurologic symptoms (TIA or stroke), race, history of angina, recent myocardial infarction, congestive heart failure, chronic obstructive pulmonary disease, hypertension, degree of stenosis of the contralateral carotid artery, or presence of ulceration in the ipsilateral artery. Geary et al sought to specifically identify predictors of adverse neurologic events after CEA. They found that the following factors were not predictors of outcome: patient age, gender, type of cerebral protection measures during CEA, use or nonuse of a carotid artery patch, a mean carotid occlusion time of less than 30 minutes, and status of the contralateral carotid arteries. However, the risk of adverse postoperative neurologic events was significantly higher in patients with preoperative stroke as compared with patients with TIAs. Sieber et al and Musser et al showed that a history of angina, myocardial infarction, or congestive heart failure in the 6 months before surgery increased perioperative risk. Mattos et al were not able to confirm that ipsilateral carotid siphon stenosis significantly increased either long- or short-term perioperative risk. A summary of the results of the more recent investigations is listed in Table 1.

Finally, the perioperative risk may be related to the characteristics of the surgeon performing the procedure. Although there is no definitive proof, the number of endarterectomies that a surgeon performs each year appears to affect outcome in patients. Surgeons who perform more than 10 procedures annually appear to have fewer

| Table 1. Preoperative Factors Increasing Perioperative Risk in CEA |
|----------------|----------------|----------------|
| **Factor** | **Studies Supporting the Risk Factor** | **Studies Refuting the Risk Factor** |
| Age (> 70 years) | McCrory et al | Goldstein et al |
| History of angina or myocardial infarction or congestive heart failure in the 6 months before CEA | McCrory et al | Goldstein et al |
| Severe hypertension | McCrory et al | Goldstein et al |
| Severity of preoperative neurologic symptoms (TIAs, crescendo TIAs, or stroke) | McCrory et al | Goldstein et al |
| Occlusion of contralateral, internal carotid artery | Goldstein et al | Geary et al |
| Siphon stenosis on ipsilateral side | Goldstein et al | Mattos et al |
| Ver | Goldstein et al | McCrory et al | |
patients with complications. A higher complication rate in patients is associated with surgeons who did not train in a United States, western European, or Canadian medical school. The investigators recommended that referring physicians should not rely solely on a surgeon’s experience and qualifications when recommending a CEA but should also consider the individual surgeon’s and the hospital’s actual postoperative complication rate.

On the other hand, others have suggested that if preoperative angiographic, medical, and neurologic data are combined, patients can be classified into risk groups. The risk of an adverse outcome (fatal or nonfatal stroke or death) after CEA appears to be lowest in patients who are neurologically stable and have no coexisting major medical problems and low angiographic risk (risk estimate of approximately 1%). The risk increases if a neurologically stable patient with no coexisting major medical problems presents with significant angiographic risk (risk estimate of approximately 2%). It increases further if stable neurologic status is accompanied by major angiographic risks and major medical problems (risk estimate of approximately 7%). Finally, perioperative risk is highest in a patient who is neurologically unstable and has major medical and angiographic risk (risk estimate of approximately 10%).

PREOPERATIVE EVALUATION AND PREPARATION

Although the most common cause of transient ischemic attacks is occlusive carotid disease, a thorough evaluation must rule out other possible causes such as intracerebral tumor, cerebrovascular malformation, and heart diseases commonly associated with a risk for transient ischemic attacks or minor stroke. Cardiac diseases that may result in neurologic symptoms include atrial fibrillation, valvular heart disease, dilated cardiomyopathy, and a myocardial infarction with akinetic ventricular segments leading to intracardiac thrombus formation.

Of equal importance is the evaluation of a patient's cardiac reserves because the presence of coronary artery disease in this patient population is extremely high. Table 2 summarizes several studies of the incidence of coronary artery disease in patients with carotid atherosclerosis. For instance, in one study, routine coronary angiography was used before planned CEA. This test showed severe surgically correctable coronary artery disease in 37% of patients with suspected coronary artery disease by conventional clinical criteria and in 16% of patients who were without suspected coronary artery disease.

Furthermore, it has been suggested that patients with unstable or severe angina, decompensated congestive heart failure, critical aortic stenosis, or a recent myocardial infarction have a greatly increased risk of cardiac morbidity and mortality after surgery. Such patients will not normally be candidates for an elective CEA. Consequently, a thorough preoperative clinical examination to identify such patients is mandatory. In addition to a review of the patient's history and laboratory data, the authors look for signs and symptoms of impending cardiac failure such as shortness of breath on minimal exertion, orthopnea, paroxysmal nocturnal dyspnea, jugular venous distention, hepatomegaly, pretibial edema, an S1 gallop, and/or rales. The chest x-ray should be examined for cardiomegaly and the typical butterfly pattern of pulmonary edema. A recent increase in severity of angina or a change in anginal pattern and/or electrocardiogram changes suggestive of myocardial ischemia, significant arrhythmias, or new left bundle-branch block are also of concern. If there is evidence of unstable cardiac, pulmonary, or metabolic disease, the authors believe that the carotid procedure should be delayed until the patient’s condition improves. Additionally, if a myocardial infarction occurred less than 3 months ago, the authors tend to postpone the procedure. The cut-off point of 3 months is arbitrary because data do not establish conclusively how long to wait to perform carotid endarterectomy after the occurrence of a myocardial infarction. The final judgment of whether to proceed with surgery always balances a patient’s general medical and cardiac status against the severity of the neurologic symptoms.

### Table 2 Prevalence of Coronary Artery Disease in Patients With Carotid Occlusive Disease

<table>
<thead>
<tr>
<th>Study</th>
<th>Characteristics of Study Population</th>
<th>No of Patients</th>
<th>Tests Used to Confirm CAD</th>
<th>Prevalence of CAD in Percent in Patients With Suspected CAD</th>
<th>Prevalence of CAD in Percent in Patients Without Suspected CAD</th>
<th>Prevalence of CAD in Percent in Patients Without Suspected CAD But Unable to Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rokey et al</td>
<td>TIAs or small stroke</td>
<td>50</td>
<td>Stress thallium scintigraphy</td>
<td>N/A</td>
<td>58%</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Stress ventriculography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Angiography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hertzer et al</td>
<td>TIAs or asymptomatic carotid bruit</td>
<td>506</td>
<td>Angiography</td>
<td>65% overall, 37% deemed “surgically correctable”</td>
<td>16%</td>
<td>N/A</td>
</tr>
<tr>
<td>DiPasquale et al</td>
<td>TIAs or small stroke</td>
<td>190</td>
<td>Exercise ECG</td>
<td>N/A</td>
<td>23%</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Stress thallium scintigraphy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DiPasquale et al</td>
<td>Stroke</td>
<td>38</td>
<td>Thallium scintigraphy</td>
<td>N/A</td>
<td>N/A</td>
<td>60%</td>
</tr>
<tr>
<td>Urbani et al</td>
<td>Symptomatic high-grade stenosis</td>
<td>106</td>
<td>Exercise ECG</td>
<td>N/A</td>
<td>25%</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thallium scintigraphy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Love et al</td>
<td>Asymptomatic disease, TIAs, small stroke</td>
<td>60</td>
<td>Thallium scintigraphy</td>
<td></td>
<td>58%</td>
<td>33%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Angiography</td>
<td></td>
<td></td>
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</tbody>
</table>

Abbreviations: CAD, coronary artery disease; ECG, electrocardiogram
Recently, Flessher and Barash \cite{51} have correctly noted that not all patients with stable angina have the same disease process (ie, coronary anatomy, frequency of ischemia, and left ventricular function) and that patients should be classified in a more functional way. They therefore proposed a method of assessing preoperative risk based on the presentation of coronary artery disease, exercise tolerance, and extent of the surgical procedure.

Because the efficacy of a given cardiac evaluation plan has not yet been established, the decision to routinely pursue specialized cardiac evaluation such as exercise testing, Holter monitoring, or angiography in patients with stable coronary artery disease remains controversial. A positive stress electrocardiogram has been found to be predictive of adverse cardiac events by some investigators but not by others.\cite{52} The information derived from angiography is certainly useful for evaluating the status of the coronary vasculature, but the expense and morbidity associated with it limit its application.\cite{52} Similarly, although the authors' recent meta-analysis has established that Holter monitoring, radionuclide ventriculography, dobutamine stress echocardiography, and dipyridamole thallium scintigraphy are effective tests for predicting adverse postoperative cardiac events, these tests are associated with both risks and costs.\cite{53} Presumably, the rationale for a thorough evaluation of the cardiac status of patients with silent or stable coronary artery disease is to detect those patients who would benefit from coronary revascularization procedures. However, patients with carotid occlusive disease have a higher risk of sustaining a stroke or myocardial infarction after myocardial revascularization than do patients without carotid disease who undergo coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA).

Tables 3, 4, and 5 summarize the risk of perioperative stroke and mortality in patients with carotid disease undergoing major cardiovascular surgery.\cite{54,55,61} The risk of stroke in patients with symptomatic carotid disease undergoing CABG can range from 8.2% to 17%. In patients with asymptomatic carotid disease, CABG may entail a stroke risk ranging from 0.3% to 9.2%; equally important, the perioperative mortality may be as high as 13%.

Despite the high incidence of coronary artery disease in this patient population, myocardial infarction within 30 days after CEA is an infrequent occurrence, perhaps because the procedure is not normally associated with major blood loss, massive fluid shifts, or major systemic physiologic disturbance. Indeed, in a study at the Mayo Clinic, 30 days after CEA there were no significant differences between patients with or without stable coronary artery disease in the occurrence of death, myocardial infarction, or stroke.\cite{62} Table 6 summarizes the results of investigations of the risk of perioperative myocardial infarction after CEA.\cite{47,48,65}

In selected series, the overall perioperative morbidity rate (mainly because of the appearance of new neurologic deficits postoperatively) and mortality rate (mainly because of adverse cardiac events after CEA) have decreased to about 1.5% and 0.5%, respectively, with some centers reporting even lower figures.\cite{56,58} These figures are clearly lower than those reported for major cardiac procedures, even in patients free of significant carotid disease. Clearly, it is difficult to recommend a CABG procedure to a patient with symptomatic carotid disease and stable coronary disease because the combined mortality and stroke rate from CABG may be higher than that from an expertly performed CEA. This applies even more so to a patient with a high risk of stroke who, perhaps because of advanced age, might be a poor candidate for CABG surgery. Data suggest that mortality after CABG for octogenarians who have carotid disease is approximately 15%, or three times the mortality of patients without carotid disease who undergo CABG.\cite{69} The authors therefore believe that CEA can and should be safely undertaken in patients for whom

<table>
<thead>
<tr>
<th>Study/Year</th>
<th>Characteristics</th>
<th>Type of Operation</th>
<th>No of Patients</th>
<th>Risk of Stroke (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bower et al\cite{34}</td>
<td>Significant stenosis with transient ischemic attacks or recent ipsilateral stroke</td>
<td>Abdominal aortic repair</td>
<td>22</td>
<td>14%</td>
</tr>
<tr>
<td>Gerraty et al\cite{52}</td>
<td>Stenosis &gt; 50% with ipsilateral carotid territory cerebral ischemic symptoms</td>
<td>CABG</td>
<td>49</td>
<td>8.2%</td>
</tr>
<tr>
<td>Hertzer et al\cite{52}</td>
<td>Significant stenosis with transient ischemic attacks or stroke</td>
<td>CABG</td>
<td>23</td>
<td>8.7%</td>
</tr>
<tr>
<td>Kantcher et al\cite{57}</td>
<td>Stenosis &gt; 60% with lateralizing neurologic symptoms</td>
<td>CABG</td>
<td>41</td>
<td>17%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peripheral vascular surgery</td>
<td></td>
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<tr>
<th>Study/Year</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Gerraty et al\cite{52}</td>
<td>Significant stenosis without ipsilateral carotid territory cerebral ischemic symptoms</td>
<td>CABG</td>
<td>309</td>
<td>0.3%</td>
</tr>
<tr>
<td>Hertzer et al\cite{52}</td>
<td>Stenosis ≥ 70% without neurologic symptoms</td>
<td>CABG</td>
<td>58</td>
<td>6.9%</td>
</tr>
<tr>
<td>Reed et al\cite{52}</td>
<td>Carotid bruit</td>
<td>CABG</td>
<td>13</td>
<td>6.9%</td>
</tr>
<tr>
<td>Brenner et al\cite{52}</td>
<td>Stenosis ≥ 50% without neurologic symptoms</td>
<td>CABG</td>
<td>153</td>
<td>9.2%</td>
</tr>
<tr>
<td>Schwartz et al\cite{52}</td>
<td>Stenosis &gt; 60% without neurologic symptoms</td>
<td>CABG</td>
<td>130</td>
<td>3.8%</td>
</tr>
</tbody>
</table>
the operation offers benefits even if they have stable coronary artery disease. They classify patients as having stable coronary artery disease on the basis of a thorough history, physical examination, and review of results from basic laboratory tests and do not routinely use other data to define stable angina. In such patients, urgent surgery to prevent stroke should not be delayed for extensive cardiac evaluation.

However, virtually all studies to date show that the long-term risks of adverse cardiac events after CEA are much greater in those with overt coronary artery disease. Indeed, in the years after CEA, coronary rather than cerebral vascular disease becomes the most frequent cause of morbidity and mortality. In the long-term follow-up of patients enrolled in the Coronary Artery Surgery Study (CASS), patients with peripheral (including carotid) vascular disease and triple-vessel coronary artery disease randomized to receive medical therapy had decreased survival rates compared with those randomized to receive surgical therapy. 70

In summary, an aggressive preoperative cardiac work-up of patients with stable coronary artery disease may not be beneficial in the immediate perioperative period. However, further coronary evaluation should be undertaken in selected patients, perhaps after CEA has been performed and the risk of a stroke dramatically reduced, in order to improve long-term survival rates of these patients. This evaluation is particularly important in patients who suffer cardiac complications during or after surgery. Table 7 summarizes the authors' approach to preoperative assessment.

The Patient With Concurrent Carotid and Coronary Artery Disease

The management of patients with both severe coronary artery disease and carotid occlusive disease is controversial. Surgical revascularization of both arterial trees may be performed in one operation or in two. The authors recommend sequential operations when one lesion is more problematic. For example, they would perform CEA first in a patient with three-vessel coronary artery disease, good left ventricular function, stable angina, crescendo transient ischemic attacks, and an 80% carotid stenosis. If a patient had unstable angina in addition to crescendo transient ischemic attacks, a combined procedure can be performed because risk for cardiac complications is markedly increased after CEA alone.

Concurrent carotid and coronary revascularization procedures have been performed with an operative mortality rate as low as 2% and a neurologic complication rate as low as 3%. 71,72 However, other series have reported combined perioperative mortality and stroke rates ranging from 8% to 40%. 73 The reasons for these discrepancies are not clear. Indeed, the operative strategies used in concurrent operations vary among surgeons. Some surgeons begin with a simultaneous CEA and a saphenous vein harvest, initiating coronary artery bypass after these procedures have been completed. This avoids the potential threats posed to the brain by pump-associated hypotension, fluctuations in perfusion pressure, and nonpulsatile blood flow. Although hypoperfusion may be one mechanism of stroke after CABG, recent studies suggest that embolic events may predominate. Up to 15% of patients with severe carotid artery disease (>80% stenosis) have coexisting mobile protruding aortic arch atheromas with a very high embolic potential. 74 Clearly, CEA will be of no value in preventing a perioperative stroke caused by embolization of these atheromas during aortic cannulation. 75,76 This fact may explain at least some of the variation in the reported rates of neurologic complications after concurrent procedures.

Alternatively, other surgeons correct both the coronary and carotid arteries during the aortic cross-clamp period. They seek to protect the brain using systemic hypothermia of about 20 to 25°C. With this strategy, several investigators
have reported extremely low rates of perioperative strokes.\(^7\),\(^8\) Although these reports are encouraging, rigorous scientific proof for a preferred operative strategy in patients with severe coronary and carotid disease is not yet available.

In summary, in regard to concurrent carotid and coronary artery disease, if a patient with asymptomatic carotid stenosis and unstable ischemic heart disease requires operation, cardiac revascularization may be performed first and the carotid lesion evaluated after recovery. If a patient has acute TIAs and stable coronary artery disease, CEA is performed first. If a patient has both unstable carotid and unstable heart disease, CEA and coronary revascularization can be performed during one operation. Others have proposed that CEA should be performed under a cervical block and that coronary revascularization should take place immediately after the CEA is finished, which avoids the negative inotropic effects of a general anesthetic. The authors’ preference for treating the more problematic lesion first cannot yet be supported with conclusive scientific data.

INTRAOPERATIVE MANAGEMENT

The two main goals of intraoperative management must be to protect the brain and the heart. Often these two goals are in conflict. For example, increasing arterial blood pressure to augment cerebral blood flow can increase afterload and/or myocardial contractility, thereby increasing the oxygen demand of the heart. Similarly, although hypothermia may provide effective cerebral protection, it also poses a significant challenge to myocardial well-being. Thus, it is clear that compromises must be made if both goals are to be realized. Various anesthetic techniques have been proposed to protect the brain and heart during surgery.

Protecting the Brain

Normal cerebral blood flow (CBF) is approximately 50 mL/100 g/min or 700 mL/min in a 70-kg person. The cerebral metabolic rate for oxygen (CMR\(_{\text{O}_2}\)) is 3.2 mL/100 g/min or about 45 mL/min. Overall, the brain receives approximately 15% of the cardiac output but consumes 20% of the oxygen. Within the range of mean systemic blood pressure from 60 to 160 mmHg, there is little variation of CBF in healthy individuals. Conversely, within the range of 25 to 55 mmHg, CBF varies linearly with Pa\(_{\text{CO}_2}\) changing by about 3% per mmHg. High Pa\(_{\text{O}_2}\) has a minimal effect; Pa\(_{\text{O}_2}\) below 50 mmHg causes vasoconstriction. Finally, regional CBF and metabolism are highly coupled in that an increase in cortical activity will lead to a corresponding increase in CBF. In contrast, CBF in healthy persons is not appreciably affected by sympathetic tone.\(^7\)

Current attempts at cerebral protection may appear primitive. Routine aspects of anesthetic care that may affect cerebral outcome if hemispheric ischemia occurs during CEA include blood pressure augmentation, manipulation of Pa\(_{\text{CO}_2}\), and choice of fluid for intraoperative administration. Whether further interventions to reduce cerebral metabolism and cerebral oxygen requirements are neuroprotective is controversial. Recent research suggests that the outcome after cerebral ischemia may be improved by antagonizing or modifying processes that normally occur after reperfusion rather than during ischemia. Lastly, shunts may be used if cerebral hypoperfusion is suspected.

Blood pressure management. The rationale behind maintaining a stable, high-normal blood pressure throughout the procedure is based on the assumption that blood vessels in ischemic or hypoperfused areas of the brain have lost normal autoregulation. Flow in such areas is believed to be mainly pressure dependent.\(^60\),\(^82\) Under these circumstances, prolonged, severe hypotension may jeopardize brain function. Nonetheless, hypotension may not be the precipitating or sole cause of stroke; embolic events may be just as or even more important.\(^63\) In addition, the pharmacologic augmentation of blood pressure with sympathomimetic drugs is not without risk to the heart. Indiscriminate use of phenylephrine to increase blood pressure during deep general anesthesia with a volatile anesthetic increases the incidence of intraoperative segmental wall motion abnormalities detected by transesophageal echocardiography as compared with light anesthesia without the use of phenylephrine.\(^84\) Metaraminol-induced hypertension during CEA is associated with an increased incidence of perioperative myocardial infarction.\(^85\) Interestingly, the judicious, restrictive use of phenylephrine to increase blood pressure in specific instances of electroencephalogram (EEG)-detected, reversible, cerebral ischemia was not detrimental to the heart.\(^86\) Based on these studies, then, limited vasopressor use during hypotensive episodes appears defensible. This recommendation assumes that other causes of hypotension such as hypovolemia and/or excessive anesthetic depth have been excluded.

Manipulation of arterial Pa\(_{\text{CO}_2}\) level Hypercapnia dilates cerebral blood vessels, and, thus, increases cerebral blood flow.\(^87\) However, hypercapnia during CEA may be detrimental.\(^81\),\(^87\) If it dilates vessels in normal areas of the brain while vessels in ischemic brain areas, which are already maximally dilated, cannot respond. The net effect, then, is a steal phenomenon, i.e., a diversion of blood flow from hypoperfused brain regions to normal brain regions. Conversely, hypocapnia may result in a potentially beneficial “inverse steal.” Normally responsive vessels in an adequately perfused brain will constrict and thereby redirect blood to ischemic regions with unresponsive, maximally dilated vessels.\(^81\),\(^87\) Unfortunately, in patients undergoing CEA, the responses previously delineated are not always predictable.\(^81\),\(^87\) As a result, most authorities recom-
mend the maintenance of normocarbia or moderate hypocarbia at best.

**Intravenous fluid administration.** There is evidence that even moderate hyperglycemia worsens ischemic brain injury.\(^8\) Although the precise mechanism of the adverse effects of hyperglycemia is unknown, brain glucose levels increase proportionately when the blood sugar concentration increases. Increased cerebral lactic acidosis, which results from the anaerobic glycolysis of increased brain glucose stores, may be directly or indirectly responsible for the adverse effect. Because the blood’s oxygen reserve is substantially less than its glucose reserve (especially in hyperglycemia), areas of the brain experiencing severe but incomplete reduction of blood flow would be particularly affected by such a mechanism. Accordingly, the continued, albeit depressed, delivery of glycolytic substrate to hypoxic brain tissue results in accumulation of lactic acid to levels higher than those measured in areas of the brain exposed to normal or complete cessation of flow.\(^8\) Because the neuroendocrine response to surgery results in the breakdown of glycogen and the incidence of diabetes in this patient population is high, blood sugar levels will often already be mildly elevated during CEA. In this situation, the administration of dextrose-containing intravenous fluid may exacerbate hyperglycemia. A similar effect may conceivably result from lactated solutions that are metabolized to dextrose. Although there is no definite proof that the use of barbiturates in this manner improves neurologic outcome after CEA.\(^9\) Presumably, blood viscosity is reduced and attendant microcirculatory disturbances are thereby ameliorated.\(^9\)

**Anesthetic-induced reduction of cerebral metabolic oxygen requirements.** Virtually all commonly used anesthetic agents reduce cerebral metabolism, which decreases the brain’s requirements for oxygen. Under these circumstances, the brain’s tolerance for temporary ischemia may be enhanced. However, the notion that reduced cerebral metabolism is associated with cerebral protection has been challenged.\(^9\) Nonetheless, until this method of pharmacologic brain protection is refuted, there is no reason to deny its potential benefits to a patient.

Among the inhalation anesthetics, isoflurane has emerged as the volatile agent with the most potential protective effect against cerebral ischemia. Isoflurane decreases the frequency of EEG-detected cerebral ischemic changes during CEA when compared with enflurane and halothane. EEG changes suggestive of ischemia occurred at a cerebral blood flow of 8 mL/100 g/min with isoflurane and at 18 mL/100 g/min with halothane or enflurane.\(^9\) Nonetheless, outcome was not different among the anesthetic groups. Numerous other investigators have corroborated these findings.\(^9\) However, isoflurane’s protective effects are maximal only at about 2 minimum alveolar concentration (MAC). At this MAC level, potentially deleterious, systemic hypotension will occur in many patients and, therefore, maximal protection is not often clinically achievable.

Desflurane and sevoflurane may also reduce cerebral oxygen requirements at MAC values comparable with those with isoflurane\(^9\) and enable more rapid emergence and recovery than with isoflurane.\(^9\) Unfortunately, the new volatile anesthetics have not yet been evaluated for their effects on cerebral ischemia during CEA. Desflurane’s propensity to cause tachycardia may detract from its use in a patient group in whom coexisting coronary artery disease is exceedingly common.\(^10\)

Barbiturates offer a degree of brain protection during periods of regional ischemia.\(^10\) Thiopental decreases cerebral metabolic oxygen requirements to about 50% of baseline. These maximally achievable reductions in oxygen requirements correspond to a silent, ie, isoelectric, EEG. Beyond this point, additional doses of barbiturates are neither necessary nor helpful. In fact, in cases of massive global ischemia in which basal cellular metabolism has already deteriorated, even high doses of barbiturates will not improve neurologic outcome.\(^10\)

The previously described data have convinced some anesthesiologists to use thiopental not only for induction but as a continuous infusion and/or as a bolus of 4 to 6 mg/kg just before carotid occlusion. With this technique, the negative inotropic effects of the barbiturates must be considered. With excessive cardiovascular depression, inotropic support may be required. Again, there is no rigorous proof that the use of barbiturates in this manner improves neurologic outcome after CEA.

Both etomidate and propofol decrease electrical activity in the brain, and, thus, decrease cerebral oxygen requirements.\(^10\) Etomidate preserves cardiovascular stability and may be beneficial in the CEA patient population with often limited cardiac reserves. Unfortunately, etomidate has excitatory side effects that may manifest as involuntary muscle movements (eg, myocloni) detracting from its usefulness.\(^10\) During incomplete ischemia, the protective effect of etomidate has not been established.\(^10\) Like etomidate, propofol may produce excitatory side effects, but it permits rapid awakening at the end of surgery.\(^10\) After CEA, rapid awakening is desirable for early postoperative neurologic assessment. However, the studies have demonstrated both the presence and absence of brain protection with the use of propofol.\(^10\)

**Hypothermia.** Hypothermia can depress neuronal activity sufficiently to put cellular oxygen requirements below the minimum levels normally required for cell viability. At least in theory, hypothermia represents the most effective method for cerebral protection. Even a mild decrease in temperature of about 2° to 3°C at the time of arterial hypoxemia may reduce ischemic damage to the brain.\(^10\) To ensure cerebral protection, the first reported CEA was performed with the patient’s head covered by ice packs.\(^12\) Both the cumbersomeness of the method as well as the unpredictability of an individual patient’s response appear to have precluded its common use. The authors allow patients to cool passively in the operating room and do not warm the operating room, intravenous fluids, or airway gases until the carotid repair has been completed. With this method, many patients will have a temperature of 35°C.
After carotid repair, warming with forced air may counteract the adrenergic response and the increased incidence of myocardial ischemia associated with hypothermia in vascular surgery patients.

**Other pharmacologic means of cerebral protection** Outcome after cerebral ischemia may be improved by preventing adverse phenomena during reperfusion. One of the crucial events in the development of ischemic neuronal damage is activation of the neuron's excitatory amino acid (EAA) receptor. In animals, administration of the EAA receptor antagonist dextromethorphan shortly after the occurrence of a focal ischemic event decreased the extent of subsequent neuronal damage. However, to date, no conclusive data show that use of dextromethorphan improves outcome in humans.

Ischemia also leads to neuronal calcium overload, which in turn sets off a series of harmful biochemical intracellular events. Therefore, it is not surprising that much research has focused on the potentially beneficial role of calcium channel blockers. Nimodipine confers at least some benefit to patients if it is administered soon after the onset of stroke symptoms. Unfortunately, as in the case of dextromethorphan, definitive recommendations cannot yet be made. Other potentially neurotoxic agents produced during cerebral ischemia are nitric oxide and free radicals such as the superoxide and hydrogen peroxide ions. Nitric oxide synthesis inhibitors as well as free radical scavengers may play an important protective role in the future.

Because it is speculated that steroids directly or indirectly protect neuronal membranes from oxidative injury by free radicals, some centers use intravenous steroids in an attempt to protect the brain from the effects of hypoxia. In a large, randomized, controlled trial, methylprednisolone reduced the extent of spinal cord deficits when administered within 8 hours of injury.

**The Use of Shunts and the Value of Cerebral Perfusion Monitoring**

Logic appears to support the routine maintenance of ipsilateral carotid flow through the use of shunts. Temporary occlusion ("cross-clamping") of the carotid artery will acutely disrupt blood flow, even if blood flow to the ipsilateral hemisphere of the brain has been markedly diminished by severe stenosis. During carotid occlusion, the area of the brain that is isolated from its accustomed supply will depend entirely on an adequate collateral blood flow through the circle of Willis if no shunt is used. However, if carotid stenosis has increased gradually in the months and years before the CEA is performed, collaterals from the circle of Willis will have had time to develop and the cerebral circulation may not be compromised by carotid occlusion by the surgeon. Moreover, if collateral flow is compromised because of occlusive disease of the contralateral carotid artery and/or the vertebral arteries, the chances that marked hypoperfusion of the brain may occur during carotid clamping are even greater. Patients with additional contralateral disease have a higher risk of perioperative stroke than patients with only unilateral disease. Even if the various methods of pharmacologic brain protection previously described are used during the procedure, there is no guarantee of improved outcome in an individual case. The need for shunts in carotid surgery remains an unresolved issue. Therefore, some surgeons never use shunts, others use them routinely, and still others use them only selectively.

Surgeons who never use shunts usually rely on fast and expeditious surgery to avoid neurologic problems. Time is an essential factor: At a carotid artery stump pressure of 40 mmHg, neurologic dysfunction is reportedly common when the artery is cross-clamped for more than 60 minutes. Surprisingly, surgeons who choose not to place a shunt do not report worse overall outcome statistics than those who do.

A number of arguments may be made against the use of shunts. They are not risk free. Placement of a shunt is associated with an embolism-related stroke rate of at least 0.7%, from the dislodgment of thrombotic materials at the time the shunt is placed. Other technical problems of shunting such as air embolism, kinking of the shunt, shunt occlusion against the side of the vessel wall, and injury or disruption of the distal internal carotid artery must be carefully avoided. At times, the shunt may prevent easy surgical access to the artery, thereby increasing cross-clamp time. Most importantly, the use of a shunt is only beneficial if the cause of neurologic dysfunction is inadequate blood flow. However, many studies suggest that 65% to 95% of all neurologic deficits during CEA occur as a result of thromboembolic events either before or after carotid repair and not because of the period of carotid cross-clamping. Once emboli have been dislodged, for instance when the carotid artery is dissected out, insertion of a shunt will not prevent a deficit. In fact, to date, there is no rigorous proof that the routine insertion of a shunt reduces the incidence of postoperative neurologic deficits.

Surgeons who use shunts only selectively need a monitoring device of cerebral perfusion to decide rationally whether to place the shunt. To this end, a variety of cerebral perfusion monitors are used: carotid stump pressure, EEG, somatosensory evoked potentials (SSEP), and transcranial Doppler (TCD) monitoring.

**Carotid artery stump pressure.** A surgeon may simply palpate the distal end of the clamped carotid artery between the thumb and forefinger. If a pulse is detected, collateral flow is presumed to be adequate and the carotid artery can be occluded. Conversely, if there is no pulse, a shunt will be placed. The approach is reportedly successful, at least in patients without a history of stroke or contralateral carotid disease.

However, more commonly, the mean blood pressure distal to the carotid clamp (also referred to as "back pressure") is invasively evaluated. Under halothane anesthesia, a mean stump pressure greater than 60 mmHg was sufficient to prevent ischemia in most cases. If a nitrous oxide/opioid-based anesthetic is undertaken, the stump pressure should be greater than 60 mmHg for blood flow to be adequate, presumably because cerebrovascular resistance is increased with this anesthetic technique. Some surgeons will not shunt unless mean stump pressure is less
than 50 mmHg to avoid the potentially harmful complications of shunting. In some cases of seemingly adequate stump pressure, stenosis may be in the middle cerebral artery distal to the arteries that provide collateral flow and "back bleeding." In this situation, cerebral ischemia may occur despite a stump pressure greater than 60 mmHg. Finally, some studies show no correlation of stump pressure to adequacy of flow. Despite all of these limitations, stump pressure monitoring has the advantages of being inexpensive and readily available; however, its overall value in terms of better outcome has not been proven.

Monitoring with the electroencephalogram. The interpretation of information gained from the scalp-recorded EEG has been simplified considerably through the use of computerized data reduction methods. The vast amounts of information from the standard EEG have been reduced to several salient calculated variables, the changes in which are charted over time. The EEG reflects the spontaneous electrical activity of cortical (surface) neurons. With increasing levels of ischemia, electrical activity on the cortical surface decreases. Under conditions of focal cortical ischemia, the frequency of the recorded EEG waves over the affected area of the brain will slow by more than 50%, and the amplitude of the waves may decrease to a comparable extent. The appearance of a disorganized background rhythm may also be a sign of ischemia. Finally, as ischemia becomes severe, the EEG will become isoelectric. The most common manifestations of EEG ischemia are ipsilateral attenuation (39% of patients), ipsilateral slowing with attenuation (21%), and ipsilateral slowing without attenuation (14%). EEG deterioration usually begins below a cerebral blood flow of approximately 15 mL/100 g of brain tissue per minute, but cellular metabolic failure does not appear to occur until blood flow decreases below 10 to 12 mL/100 g of brain tissue per minute. Fig 3 of a processed EEG exemplifies acute cerebral ischemia after left carotid artery occlusion; the placement of a shunt in this case promptly reversed the ischemic changes.

In practice, the EEG can be obtained by using a 16-channel strip-chart recording or a processed EEG monitor with 2 or 4 channels. A 16-channel strip-chart EEG generally requires a technician to set up the monitor and to monitor and interpret the large amount of unprocessed data generated. In contrast, the 2- to 4-channel processed EEG monitors provide less data, but they can be monitored more easily. (Usually the plot of voltage versus time of the standard EEG is converted to a plot of frequency and power versus time.) Processed EEG display methods include compressed spectral array, density spectral array, pie graphs, and others. Processed EEG monitors will display both real physiologic signals and noise. Because it is not always apparent from the processed data display which data are real signals, processed EEG monitors must have the ability to display unprocessed, raw data in order to confirm that the signals monitored represent genuine brain activity and not artifacts.

Patients with contralateral occlusion and stump pressures of less than 25 mmHg routinely display major EEG changes. On the other hand, major EEG changes occur in fewer than 20% of all monitored patients. When such major EEG changes are present, they do appear to define a patient subgroup with a significantly increased risk of perioperative stroke. EEG monitoring has several limitations. For one, deep (as opposed to surface) brain structures are not monitored by EEG. In patients with preexisting neurologic deficits, strokes in evolution and/or recent reversible ischemic neurologic deficits may result in a false-negative on the EEG, ie, these patients develop perioperative strokes despite a lack of major intraoperative EEG changes. Possibly, in these patients, there are cell populations that are electrically silent or immediately adjacent to regions of infarction, and, therefore, not monitored by the EEG. Conceivably, these still-viable cell populations then progress to irreversible deterioration in the course of the operative procedure. In patients without the previously described problems, the EEG may be a reasonably sensitive detector of cerebral ischemia. EEG is not an ischemic-specific monitor because changes in the EEG may result from decreases in temperature and blood pressure, systemic hypoxemia, increases in the depth of anesthesia, or electrolyte imbalances. However, EEG changes caused by these factors are more likely to be bilateral; hemispheric ischemia is more likely to affect the electrical activity of only one side of the brain.

Thus, it is imperative that the encephalographer be kept abreast of changes in the anesthetic regimen if accurate results are to be obtained. Most importantly, the value of relatively costly EEG monitoring has not been rigorously established. Indeed, one study that correlated EEG changes and outcome demonstrated that only 2 of 10 patients with postoperative strokes had shown EEG changes intraoperatively; in addition, none of the patients with postoperative transient ischemic attacks had manifested EEG changes.

Monitoring somatosensory evoked potentials. Electrical stimuli presented to a peripheral nerve (commonly the median nerve) pass through the first- and second-order
neurons and their synapses to the brain stem and from these are transmitted to the somatosensory cortex. At this level, the stimuli are detectable as cortical potentials. Unlike the EEG, SSEP monitors evaluate deep brain structures and not only surface, cortical structures. Evoked potentials are analyzed in terms of latency and amplitude. Latency, the time from peripheral stimulation to detection of the signal at the cortex, reflects the conduction velocity characteristics of the neural pathway. Amplitude indicates the number and synchrony of the conduction fibers in the pathway. Any damage to these neural structures results in characteristic changes in the SSEP, usually a decrease in amplitude, and/or an increase in latency. Indeed, if neural damage is severe, the cortical evoked potential is completely abolished. Neural damage occurs at about one third of normal cerebral blood flow, ie, 15 mL/100 mg of brain tissue per minute.\(^{140}\)

The central challenge in SSEP monitoring is extracting the signal caused by the peripheral stimulation from the spontaneous electrical activity of the cortex. Extraction is performed by computer-assisted mathematical analysis of the temporal relationships of the peripheral stimuli and their resulting cortical electrical potentials. Even so, considerable expertise and experience are required in SSEP analysis.

There are studies that are quite optimistic about the value of SSEP monitoring in the detection of cerebral ischemia.\(^{150-155}\) However, other investigators have concluded that SSEP is neither sensitive nor specific for the detection of ischemic injury during CEA.\(^ {157}\) As in the case of the EEG, there are reports in the literature of both false-negative and false-positive results. With regard to the latter, all commonly used anesthetics lead to SSEP changes that mimic changes produced by cerebral ischemia.\(^{154-156}\) Consequently, only if a constant light plane of anesthesia is maintained are increased latencies and decreased amplitudes of evoked potentials indicative of inadequate cerebral perfusion. In addition, there appears to be a subset of neurons at risk of further deterioration that are not adequately monitored by SSEP analysis. Hence, patients may awaken with new neurologic deficits despite normal intraoperative evoked potentials.\(^ {153}\) Based on the available data, this complex and costly monitoring system cannot yet be considered essential for a safe surgical procedure.

**Monitoring with transcranial Doppler.** The TCD technique measures the mean velocity of blood flow in the middle cerebral artery; severe reductions in the mean flow rate appear to be reliable indicators of the danger of cerebral hypoperfusion with the attendant potential of ischemic neural damage.\(^ {157}\) Indeed, a 40% decrease in flow velocity corresponds with major EEG changes.\(^ {157}\) Other investigators have found that both peak and mean flow velocity in the middle cerebral artery are significantly lower in patients with stump pressures below 30 mmHg.\(^ {158}\) Reportedly, the use of TCD made it possible to detect cerebral ischemia either in advance or independent of the EEG. In one case report, investigators cited an instance in which the EEG remained normal despite a significant reduction in flow velocity. Based on this evidence of potential occlusion, the vessel was reexplored and a thrombus was removed.\(^ {159}\) TCD detects microemboli that are not usually detected by EEG.\(^ {160}\) If microemboli are detected during initial carotid dissection, the surgeon should be warned to modify his or her technique in the hope of avoiding a significant embolic stroke. Alternatively, sudden decreases in flow velocity may indicate acute thrombosis of the endarterectomy site, especially when flow velocity is reduced to zero.\(^ {161}\) With severe reductions in flow velocity to 15% of baseline or less, strokes appear to be less frequent when shunts are used.\(^ {162}\) In addition, TCD may help in identifying patients who are at risk of developing postoperative hyperperfusion syndrome.\(^ {163}\) Finally, when measurement of TCD flow velocity was compared with the measurement of regional oxyhemoglobin saturation (rSO\(_2\)) by infrared cerebral spectroscopy, severe decreases in flow velocity correlated with significant decreases in rSO\(_2\). Conversely, in patients with only minimal flow velocity reductions, no trends were observed in rSO\(_2\).\(^ {164}\) Whether cerebral spectroscopy alone or in conjunction with TCD will play a role in CEA in the future remains to be determined. Thus, although the data are certainly encouraging, definitive proof that the use of TDC will lead to improved outcome is not available.

Cerebral blood flow can also be measured intraoperatively by the intravenous or intracarotid injection of radioactive xenon or krypton. The radioactive xenon/krypton is discernible by an array of detectors placed over the patient’s head. (The more detectors used, the easier it is to localize specific areas of lower perfusion.) The xenon/krypton washout pattern is directly dependent on cerebral blood flow.\(^ {165}\) Theoretically, washout measurements can be obtained at multiple times during surgery, but it is clearly not a continuous monitor. Normally, measurements are performed just before and after carotid cross-clamping. Unfortunately, the expense and expertise required to collect and interpret the obtained cerebral blood flow data have limited its use to only a few centers. Similarly, jugular venous oxygen saturation may reflect global cerebral blood flow and oxygen consumption. However, because of interhemispheric mixing of venous blood, it is not capable of reflecting focal cerebral perfusion. In addition, because changes in regional blood flow can occur without changes in jugular venous oxygen saturation, this monitor is not generally considered adequate for ischemia monitoring.\(^ {166}\)

In summary, therefore, it must be stated that none of the previously described procedures and neurophysiologic monitors has been shown to improve outcome. This may be due to the fact that embolism and not hypoperfusion is the most common cause of perioperative stroke. In the authors’ opinion, the real value of cerebral monitoring may lie not in the prevention of stroke but in the avoidance of “blind” blood pressure augmentation with its detrimental effects on the heart.

**Protecting the Myocardium**

Besides protecting the brain, the other important intraoperative goal must be protecting the heart and preventing myocardial infarction. This area may be one in which the anesthesiologist can help the patient most.
Because the prevalence of coexisting coronary artery disease in patients scheduled for CEA is very high (Table 2), evidence of perioperative myocardial ischemia can be found in more than 50% of high-risk patients even when CEA is performed under regional anesthesia.\textsuperscript{157} Patients with intraoperative myocardial ischemia appear to be at an increased risk for postoperative cardiac events.\textsuperscript{157}

Intraoperative monitoring of patients scheduled for CEA should include the routine monitors used during all major general or regional anesthetics: precordial stethoscope, temperature probe, blood pressure cuff, pulse oximeter, and a monitor for end-tidal carbon dioxide tension. The latter is especially useful because it allows the anesthesiologist to manipulate minute ventilation to achieve and/or maintain normocarbia or mild hypocarbia.

An intra-arterial catheter for blood pressure monitoring allows detection of beat-to-beat changes in pressure and facilitates prompt treatment. The authors measure the blood pressure noninvasively before surgery in both arms because generalized peripheral vascular disease can produce striking differences between the blood pressures in the two upper extremities.\textsuperscript{165} However, it is not clear in such a circumstance whether the higher or lower blood pressure should be used to guide hemodynamic management during carotid occlusion.

The authors routinely monitor leads II and V\textsubscript{5} of the ECG for ST-T segment changes. Automated ST-T segment analysis reportedly allows easier detection of myocardial ischemia.\textsuperscript{166} ECG monitoring detects at least 60% of all myocardial ischemic events in vascular patients if systolic wall motion abnormalities are considered the reference standard for myocardial ischemia.\textsuperscript{170} Thus, in high-risk patients, the authors will also use two-dimensional transthoracic echocardiography as an additional monitor. In awake patients, the use of transthoracic echocardiography is precluded; however, the authors do not use regional anesthesia for CEA at their institution.

It is rarely necessary to use a pulmonary artery catheter, even without transthoracic echocardiography monitoring. Although earlier studies reported that an increase in pulmonary capillary wedge pressure was a sensitive indicator of myocardial ischemia, more recent studies dispute this finding.\textsuperscript{171,172} Even if increases in pulmonary capillary wedge pressure in the absence of systemic pressure changes are sensitive, they may not be specific, ie, caused by ischemia. Similarly, although v-waves on the pulmonary capillary wedge pressure tracing may be more ischemia-specific, they are not sensitive, ie, despite their absence, myocardial ischemia may nonetheless be present.\textsuperscript{173} The pulmonary capillary wedge pressure cannot be monitored continuously without causing pulmonary infarction or another grave complication. Finally, the pulmonary artery catheter would normally have to be inserted from less-favored subclavian, arm, or femoral sites because the risk of an accidental carotid artery puncture during cannulation of the internal jugular vein may be as high as 2%. Indeed, most surgeons will postpone the operation if the contralateral artery has been disturbed in this way. In summary, then, the authors use pulmonary artery catheters only under these circumstances: the rare patient with uncompensated congestive heart failure undergoing urgent CEA; the patient whose only evidence of myocardial ischemia is pulmonary capillary wedge pressure changes without ST-segment changes; or in operations in which transthoracic echocardiography is unavailable or unsatisfactory. Finally, in the authors' experience, intravenous access for volume administration can be limited to one well-secured, well-running, medium-bore intravenous line because major blood loss or fluid shifts during CEA are rare. Similarly, a central venous catheter may be helpful for determining central venous pressure and rapid administration of vasoactive substances to the heart, but the authors believe it is not normally an essential monitor for this operation. Table 8 contains a summary of the monitors needed for CEA.

Assuring intraoperative hemodynamic stability. Maintaining intraoperative hemodynamic stability begins with the preoperative visit. The patient's chart is reviewed to determine how physical status and medical problems may be optimized. Chronic medications such as ß-blockers are continued on the day of surgery because an abrupt withdrawal of these drugs may lead to well-known rebound phenomena, which may endanger the patient's myocardium. Patients who present on the same day as surgery must be reminded to take their normal antihypertensive or antianginal medicines. For omitted medications, readily available oral or parenteral substitutes are administered. Equally important, a series of blood pressure and heart rate determinations are obtained either in the preoperative clinic or during the night before surgery. These data are used to determine an acceptable range at which blood pressure and heart rate will be maintained for the patient. For most patients, the assurance provided during the interview in the preoperative clinic or during the night before surgery is sufficient to make administration of anxiolytic medication unnecessary. If sedatives are deemed indispensable, a "light" short-acting premedication will facilitate early postoperative neurologic assessment.

In the past, the authors have routinely administered an intravenous infusion of normal saline starting at midnight before surgery at a rate of 100 mL/h for a 70-kg patient to prevent or attenuate preoperative hypovolemia and to reduce the incidence of severe hypotension after the induction of general anesthesia in hypertensive patients. Now, however, with most of the patients arriving at the

<table>
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<tr>
<th>Table 8</th>
<th>Suggested Monitors for Patients Undergoing CEA Under General Anesthesia</th>
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<tr>
<td>Routine:</td>
<td>Precordial stethoscope</td>
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<tr>
<td></td>
<td>Temperature probe</td>
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<tr>
<td></td>
<td>Blood pressure cuff</td>
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<td></td>
<td>End-tidal carbon dioxide monitor</td>
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<tr>
<td></td>
<td>Intra-arterial catheter</td>
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<td></td>
<td>ECG leads II and V\textsubscript{5}</td>
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<tr>
<td>Highest-risk patients:</td>
<td>Central venous catheter</td>
</tr>
<tr>
<td></td>
<td>Transthoracic echocardiography</td>
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<td></td>
<td>Pulmonary artery catheter</td>
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hospital on the same day as surgery, the authors encourage patients to drink fluids until midnight and schedule CEA surgery as the first case of the day. Once in the operating room, the authors generally avoid administering more than 10 mL per kg body weight of crystalloid or other fluids in a typical 2-hour operation because fluid overload may contribute to postoperative hypertension.

For procedures performed under general anesthesia, thiopental, propofol, or etomidate is used for induction. Esmolol is valuable to blunt hypertensive responses to intubation in this setting.124 For patients with hypertension on entering the operating room (diastolic blood pressure > 100 mmHg) or without overnight hydration, induction of general anesthesia may result in hypotension. The authors anticipate this possibility and induce general anesthesia slowly, being prepared to augment blood pressure pharmacologically if it decreases excessively. Because the respiratory depression and sedation caused by opioids may persist into the postoperative period and may confound the results of early neurologic assessment, the authors generally restrict the use of opioids whenever possible (eg, fentanyl ≤3-5 μg/kg). A superficial cervical block (see below) before incision will decrease postoperative pain and the need for analgesics. Anesthesia is maintained with 50% nitrous oxide in oxygen and light levels of isoflurane because of its potential protective effect against cerebral ischemia. Continuous infusions of vasopressors (such as phenylephrine) to maintain blood pressure in the normal or high normal range are administered only if the patient's endogenous responses do not suffice.

Perioperative tachycardia predisposes patients to myocardial ischemia. If tachycardia or myocardial ischemia is detected intraoperatively, the authors attempt to rule out inadequate anesthesia as a cause before aggressively treating both. Beta-adrenergic-blocking drugs, α₂-agonists, and nitroglycerin may reduce intraoperative tachycardia and myocardial ischemia.175-178 With sudden onset of bradycardia and hypotension caused by surgical irritation of the carotid sinus, baroreceptor reflexes can be attenuated by chemical denervation with a topical anesthetic.179 Consequently, the authors' surgeons routinely infiltrate the carotid bifurcation with 1% lidocaine. Either vecuronium or atracurium is used to provide muscle relaxation, depending on the target heart rate. An additional dose of muscle relaxant is administered before carotid occlusion when the concentration of volatile anesthetic is decreased to augment blood pressure. The authors do not actively warm patients until after carotid reperfusion, at which time forced air warms patients who are hypothermic. With this regimen, patients can usually be extubated at the end of the surgical procedure, and an early neurologic assessment can be performed. The authors' results with this method have been good (mortality of 1%, stroke and myocardial infarction both 0.76%). Table 9 summarizes their strategy of intraoperative anesthetic management. A review of the literature shows that others have achieved similar results using a nitrous oxide/opioid technique or a continuous infusion of thiopental.180 No one general anesthetic technique has been proven to provide superior outcome.

Regional Anesthesia

Regional anesthesia is used by many centers for CEA. The necessary sensory blockade in the C2 to C4 dermatomes can be achieved in several ways: by superficial (Fig 4) or deep cervical block (Fig 5) or by subcutaneous infiltration of the peripheral nerves supplying the surgical field. These nerves have a common point of emergence around the midpoint of the sternocleidomastoid muscle and are thus relatively accessible. Alternatively, in the deep cervical block technique, the first four cervical nerves, which form the cervical plexus, can be blocked as they pass through their respective intervertebral foramina. Single or multiple injections of local anesthetic are given at the lateral side of the neck. Proponents of regional anesthetic techniques claim the following advantages: greater stability of blood pressure during surgery, inexpensive and easy cerebral monitoring, avoidance of endotracheal intubation in patients with chronic obstructive lung disease, and avoidance of negative inotropic anesthetic agents in patients with limited cardiac reserves. In addition, overall costs of regional anesthesia are also claimed to be lower.181,182

The disadvantages of regional anesthesia are that the brain cannot be protected pharmacologically with anesthetics, and that, in the case of panic, sudden loss of consciousness, or onset of seizures, control of the airway may be difficult. The need for emergency intubation with regional anesthesia is uncommon, but it may be difficult under these circumstances and complicate surgical management. Regional anesthesia requires that the patient remain highly awake.

### Table 9. Suggested Intraoperative Management of General Anesthesia

<table>
<thead>
<tr>
<th>Preanesthetic visit</th>
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<tbody>
<tr>
<td>Determine range of patient's normal blood and heart rate pressures</td>
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<tr>
<td>No or &quot;light&quot; premedication only</td>
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<table>
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<tr>
<th>Induction</th>
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<tr>
<td>Thiopental, propofol, or etomidate</td>
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<tr>
<td>Esmolol, 50-100 mg, or fentanyl, 2-5 μg/kg</td>
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<tr>
<td>Succinylcholine, 1.5 mg/kg (in the absence of neurologic deficit), or vecuronium, 0.1 mg/kg, or atracurium, 0.4 mg/kg</td>
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<tr>
<th>Maintenance</th>
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<tr>
<td>Wound infiltration with local anesthetic</td>
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<tr>
<td>Isoflurane, 0.5%-1.5%</td>
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<tr>
<td>50% O₂/50% N₂O</td>
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<tr>
<td>Vasopressors (eg, phenylephrine) only as needed</td>
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<tr>
<td>Aggressive treatment of tachycardia and severe hypertension (esmolol, labetalol, nitroglycerin)</td>
</tr>
<tr>
<td>Moderate fluid substitution only</td>
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<tr>
<td>Avoid hyperthermia (no active warming)</td>
</tr>
<tr>
<td>Maintain normocarbia</td>
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<td>No sugar-containing solutions</td>
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<tr>
<th>Emergence</th>
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<tbody>
<tr>
<td>Esmolol, labetalol</td>
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<tr>
<td>Nitroglycerin</td>
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<td>Nifedipine</td>
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<tr>
<th>Postoperative care</th>
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<tbody>
<tr>
<td>Acetaminophen first choice for analgesia</td>
</tr>
<tr>
<td>Treat hypertension as for emergence</td>
</tr>
<tr>
<td>Treat hypotension with phenylephrine or dopamine</td>
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<tr>
<td>O₂ by nasal cannula or mask</td>
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</table>
cooperative throughout the operation; sedation can be provided only to a limited extent during carotid occlusion. Some investigators have found no difference in neurologic or cardiac complications or hospital stay between patients receiving regional or general anesthesia for CEA.\textsuperscript{183}

In the authors' institution, they perform general anesthesia for CEA, supplemented by skin infiltration and tracheal administration of local anesthetics. They provide patients with a "light" general anesthetic that permits EEG monitoring and results in blood pressures in the high range of normal. However, based on the currently available evidence, they recommend that in choosing anesthetic technique, the preference of the surgeon and the experience and expertise of the anesthesiologist should be considered.

\section*{Postoperative Management}

Common problems arising after CEA include difficulties during emergence from general anesthesia, postoperative hemodynamic instability, respiratory insufficiency, and the onset of new neurologic dysfunction. Table 10 lists commonly encountered postoperative problems.

\section*{Emergence From General Anesthesia}

Because early neurologic evaluation of the patient after CEA is essential, extubating the patient on the operating table is desirable. Both acute tachycardia and hypertension may precipitate acute myocardial ischemia, and hypertension may lead to cerebral edema and/or hemorrhage.\textsuperscript{184} During extubation of the trachea, the authors are prepared to treat exaggerated cardiovascular responses. The hyperdynamic response may be attenuated by the judicious use of beta-adrenergic-blocking agents, antihypertensives, and/or antianginals. Esmolol or labetalol is most frequently used for this purpose. Calcium entry blocking drugs or nitroglycerin may control blood pressure, but they may not reduce myocardial ischemia because of reflex tachycardia.\textsuperscript{185}

Shortly before extubation, the patient is asked to move all four extremities to exclude acute thrombosis of the endarterectomy site. More than 10% of patients may suffer various cranial nerve injuries from surgical manipulation.\textsuperscript{186,187} Injury to the recurrent laryngeal nerve must be suspected in patients with a history of previous contralateral CEA or thyroid resection. In such patients, preopera-
Fig 5 Deep cervical block
Single injection of the cervical plexus can be performed at the C4 level, where the cervical nerve roots are contained in a continuous space between the scalene muscles. A single needle is inserted on the vertical line at the C4 level and directed medially and slightly caudad to contact the "gutter" of the transverse process. Possible complications include injection into the epidural space, dural sleeve, or vertebral artery (Reprinted with permission).

Recent evaluation of vocal cord function should be considered. Unilateral recurrent laryngeal nerve injury may manifest only as transient hoarseness, but bilateral injury of the nerves may lead to paralysis of both vocal cords and, hence, acute airway obstruction. About 5% of the patients may sustain injuries to the hypoglossal nerve and about 3% to the facial nerve. Damage to the hypoglossal nerve results in paresis or paralysis of the ipsilateral tongue musculature.
Hemodynamic Instability and Myocardial Infarction

Postoperative hypotension accompanied by bradycardia may also occur after CEA but is less frequent than hypertension. Surgical removal of an atheroma reexposes the baroreceptors of the carotid sinus nerve to higher levels of stimulation by increases in transmural pressure. This in turn may cause brain-stem-mediated vagal bradycardia and hypotension. Chemical denervation of the carotid baroreceptors with a local anesthetic resulted in fewer hypotensive patients and more hypertensive patients. Over time, the baroreceptors seem to adjust to the higher pressure levels, and in most cases, the hypertensive phase resolves within 12 to 24 hours. Because significant hypotension can be caused by myocardial ischemia and/or infarction, a 12-lead ECG would be obtained in addition to administering intravenous fluids and/or vasopressors such as ephedrine, dopamine, or phenylephrine to correct severe hypotension. A 12-lead ECG is routinely obtained for all of the patients in the recovery room because of the high incidence of coronary artery disease in this patient population.

Respiratory Insufficiency

Postoperative respiratory insufficiency may be caused by bilateral recurrent laryngeal nerve injury, a massive hematoma, or deficient carotid body function. Wound hematomas develop in up to 2% of patients after CEA. In the authors’ experience, small hematomas caused by venous oozing usually can be treated by reversing residual heparin with protamine or by applying gentle digital compression for a few minutes. Any expanding hematoma should be carefully and immediately evaluated because of the possibility of tracheal compression and loss of the airway. Even rapid evacuation of the hematoma may not relieve the airway obstruction if massive pharyngolaryngeal edema is present at the same time. Postoperative bleeding and hematoma formation are more common and delayed if a patch angioplasty has been performed, regardless of whether prosthetic material or saphenous vein was used. However, patch angioplasty may be used increasingly in attempts to decrease restenosis after CEA, particularly in women. Therefore, some clinicians routinely reverse heparin with protamine in patients who have had a patch angioplasty.

Surgical manipulation may also damage the nerve supply to the carotid body. Although unilateral loss of carotid body function is unlikely to be significant, a bilateral loss makes a patient unable to increase ventilation in response to a decrease in PaO₂. The authors therefore believe that the routine use of supplemental oxygen in the recovery area is justified. Similarly, drugs that depress respiratory drive should be avoided as much as possible in postoperative pain management. It is the authors’ experience that acetaminophen, particularly when skin infiltration with local anesthetic was performed in the operating room, constitutes effective pain relief in most patients.

Neurologic Dysfunction

Hyperperfusion syndrome is believed to result from blood flow to the brain greatly in excess of its metabolic needs. Postoperative hypertension resulting from sympathetic overactivity may cause brain-stem-mediated vagal bradycardia and hypotension. The baroreceptors seem to adjust to the higher pressure levels, and in most cases, the hypertensive phase resolves within 12 to 24 hours. Because significant hypotension can be caused by myocardial ischemia and/or infarction, a 12-lead ECG would be obtained in addition to administering intravenous fluids and/or vasopressors such as ephedrine, dopamine, or phenylephrine to correct severe hypotension. A 12-lead ECG is routinely obtained for all of the patients in the recovery room because of the high incidence of coronary artery disease in this patient population.

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Intensive Care Surveillance

In experienced hands, CEA is a surgical procedure that causes only minor postoperative physiologic derangements in most patients. As a consequence, the routine postoperative admission of all CEA patients to an intensive care unit is unnecessary.2°6,2°7 Although postoperative neurologic deficits may signal inadequate collateral flow, carotid thrombosis may cause postoperative stroke. Prompt surgical reexploration can produce significant neurologic improvement.2°6,2°7 Alternatively, microembolization may cause focal and minor deficits. Noninvasive assessment of internal carotid flow and anticoagulation after exclusion of a hemorrhagic brain lesion usually constitutes sufficient treatment.2°6,2°7


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CAROTID ENDARTERECTOMY


