Mechanisms of Neurologic Deficits and Mortality With Carotid Endarterectomy

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**Objective:** To evaluate the incidence and etiology of perioperative complications of carotid endarterectomy.

**Design:** Retrospective review of carotid endarterectomies performed over 13 years. Risk factors, indications, results of electroencephalographic (EEG) monitoring, and outcomes were evaluated.

**Setting:** University medical center.

**Patients:** Three hundred sixty-seven consecutive primary carotid endarterectomies were performed on 336 patients. Indications for operation included transient ischemic attack (48.5%), asymptomatic stenosis (24%), stroke (17%), nonlateralizing ischemia (9.5%), and stroke-in-evolution (1%).

**Main Outcome Measures:** Postoperative neurologic deficits (permanent and transient) and deaths were correlated with preoperative symptoms, probable mechanism of the neurologic event, intraoperative EEG changes, and the use of intraoperative shunts.

**Results:** Four new permanent neurologic deficits (1.1%) and one transient postoperative deficit were noted. Of the five deficits, three were related to undiagnosed intraoperative cerebral ischemia and two were related to perioperative embolism. Three perioperative deaths (0.8%) occurred: two of myocardial infarction and one of intracerebral hemorrhage from a ruptured arteriovenous malformation. Intraoperative EEG tracings for the most recent consecutive 175 procedures were analyzed. Shunts were used in 45 patients (26%), 38 of whom demonstrated significant EEG changes with carotid clamping.

**Conclusions:** Carotid endarterectomy can be performed with a low risk of stroke (1.1%) and death (0.8%). Stroke was due to cerebral ischemia or embolization. With meticulous surgical technique, death is due to myocardial ischemia and not neurologic events.

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Randomized clinical trials for both symptomatic and asymptomatic carotid stenosis have demonstrated a distinct benefit of carotid endarterectomy over medical management. Yet even in these well-controlled studies, involving only referral centers that routinely perform carotid endarterectomies, the risk for stroke or death at the time of operation was not insubstantial, ranging from 2% to 6%. Other studies of surgical outcomes have suggested that the incidence of perioperative stroke in community hospitals also varies quite widely.

The mechanisms of stroke associated with carotid endarterectomy have been well characterized. Ischemic neurologic deficits may result from avoidable problems such as untreated cerebral ischemia during carotid occlusion and technical errors; other events that may lead to adverse outcomes are more difficult to predict and prevent. Platelet or fibrin emboli may form even on smooth endarterectomized surfaces, and hemorrhage into previous cerebral infarctions can occasionally follow restoration of normal arterial inflow. Also, the incidence of coronary artery disease in this patient population is high; myocardial infarction is a risk irrespective of management.

Over the years, a number of strategies have been adopted to attempt to reduce the incidence of both neurologic and nonneurologic complications. Greater attention is paid to preoperative assessment of cardiac function and myocardial perfusion, and careful control of immedi-
PATIENTS AND METHODS

Data were obtained from a retrospective review of the medical records of 367 consecutive primary carotid endarterectomies in 336 patients at the University of Chicago between 1981 and 1994. Patients with recurrent carotid stenoses, carotid/subclavian disease, and nonatherosclerotic vascular disease were excluded. The patient groups were divided into two eras (1981 through 1987 [192 patients] and 1988 through 1994 [175 patients]) to assess trends in outcome.

The vast majority of patients underwent both duplex ultrasonographic imaging of the extracranial vessels and complete arteriographic evaluation with visualization of the aortic arch, carotid and vertebral arteries, and intracranial circulation. Cardiac evaluation was individualized based on history, electrocardiographic findings, and symptoms. Patients with evidence of clinically important coronary artery disease underwent echocardiography or dipyridamole-thallium stress tests followed by coronary arteriography as indicated. Simultaneous carotid-coronary operations were performed in three patients.

All operations were performed with general anesthesia. In selected patients, intraoperative transesophageal echocardiography was used to evaluate cardiac function and detect wall motion abnormalities consistent with myocardial ischemia. Digitally processed electroencephalographic (EEG) monitoring was used in nearly all patients beginning in 1983. The EEG signal from the middle cerebral artery distribution was displayed as continuous compressed spectral arrays and as histograms of the power spectrum of six frequencies: low (delta and theta), middle (alpha), and high (sigma to beta). Intraluminal shunting during carotid occlusion was selectively performed based on history (recent stroke or unstable neurologic deficit), reduced collateral circulation including contralateral carotid artery occlusion, or ischemic changes in the EEG. Our criteria for clinically important EEG changes have been previously reviewed in detail and include loss of both high and middle frequencies or a decrease in power of greater than 50% in all three frequency bands.

Postoperatively, all patients were observed in a recovery room or intensive care setting until fully awake with a stable blood pressure and heart rate (at least 6 hours). Neurologic consultation was obtained in all patients manifesting neurologic impairments to precisely characterize any deficits.

RESULTS

DEMOGRAPHICS

Of the 336 patients, 190 (56.5%) were men and 146 (43.5%) women, with an age range of 44 to 87 years. Thirty-one underwent bilateral endarterectomies. Other manifestations of atherosclerosis and contributing risk factors were prevalent and were most precisely categorized in patients in the 1988 through 1994 group (hypertension, 72%; tobacco use, 80%; symptomatic coronary artery disease, 55%; and peripheral vascular arterial occlusive disease, 46%).

Indications for operation were similar in both study periods (Table 1) and included transient ischemic attack (178 patients [48.3%]), asymptomatic stenosis greater than 75% (87 patients [24%]), stroke (62 patients [17%]), nonlateralizing ischemia (35 patients [9.5%]), and stroke-in-evolution (five patients [1%]). Twenty-eight patients (8%) had contralateral carotid occlusions.

MORTALITY

There were three perioperative deaths (0.8%). Two deaths were secondary to myocardial infarctions. One patient died of rupture of an ipsilateral arteriovenous malformation. All three deaths occurred in the first study interval (1981 through 1987). One additional patient suffered profound bradycardia associated with intraoperative manipulation of the carotid bifurcation; he was resuscitated and recovered.

NEUROLOGIC COMPLICATIONS

Permanent neurologic deficits occurred in four (1.1%) of 367 carotid endarterectomies. A transient deficit that completely resolved developed in one patient. Neurologic deficits secondary to cerebral ischemia (two permanent, one transient) developed in three of the five patients. Embolization from the endarterectomy sites accounted for the two other complications. One patient sustained a shower of platelet aggregate embolisms from the endarterectomy site at the time of carotid unclamping, and a second patient developed an embolism from a thrombus adherent to the endarterectomy site. Three of the four strokes occurred within the first 2 years of our experience when EEG was in its initial phases of use, yielding a perioperative stroke rate of 1.5% for 1981 through 1987 and 0.56% for 1988 through 1994.

Intraoperative EEG tracings for the most recent 175 consecutive endarterectomies were available for detailed reanalysis. During carotid occlusion, 38 patients (21.7%) demonstrated EEG changes consistent with cerebral ischemia that were reversed with the insertion of the intra-arterial shunt. Seven additional patients...
underwent insertion of the shunt based on historical indications, specifically recent stroke, contralateral occlusion, or unavailability of EEG monitoring. Electroencephalographic changes were more prevalent in the 11 patients with contralateral carotid artery occlusion. Five of 10 patients demonstrated EEG changes; monitoring was not used in one. Overall, ischemic changes were noted in 50% of patients with contralateral occlusions and 20% of those with patent vessels (Table 2). No neurologic complications developed in patients who underwent shunt insertion.

**CASE HISTORIES**

**CASE 1**

W. C. had an ipsilateral cerebrovascular accident 4 days prior to undergoing carotid endarterectomy that resulted in a mild deficit (left upper extremity weakness). Urgent operation was undertaken owing to a very tight carotid stenosis and the potential for complete occlusion or repeated embolization. He was monitored intraoperatively with a 16-lead unprocessed EEG; the results were believed to be unchanged throughout the procedure. No shunt was placed. Postoperatively, his preoperative deficit worsened. Postoperative carotid duplex ultrasonography of the endarterectomy site revealed no evidence of intimal defect or thrombus. The patient is presumed to have suffered an ischemic episode in the area of his recent infarct that was “electrically silent” but sensitive to ischemia.

**CASE 2**

E. K. suffered a reversible ischemic neurologic deficit 1 month prior to carotid endarterectomy. Electroencephalography was not available for this patient in 1982, but the patient was noted to have good “back bleeding” from her internal carotid artery. The plaque was smooth without ulceration and an intra-arterial shunt was not used. Postoperatively, the patient awoke with a permanent neurologic deficit. Evaluation included a normal carotid duplex and computed tomographic scan of the brain that was negative for an acute intracranial bleed.

**CASE 3**

J. M. underwent an uncomplicated left carotid endarterectomy and was neurologically intact postoperatively. Two hours into the recovery period, right hemiplegia developed. Immediate duplex ultrasonographic evaluation revealed a flow disturbance at the origin of the internal carotid artery. Surgical reexploration revealed an adherent thrombus on the endarterectomized surface unassociated with an intimal flap. The patient underwent an interposition graft but had a persistent neurologic deficit. The cause of her neurologic deficit was presumed to be secondary to embolism from the endarterectomy site thrombus.

**CASE 4**

J. A. presented 3 months after a right cerebral infarction with left hemiplegia. A carotid angiogram revealed a totally occluded right internal carotid artery and 90% stenosis of the left internal carotid artery. During left carotid endarterectomy without a shunt, she demonstrated only minor EEG changes during carotid cross-clamp (50% reduction in power) but significant bilateral EEG changes developed with the restoration of cerebral perfusion. Postoperatively, she was noted to be minimally responsive with profound bilateral weakness. Emergent surgical reexploration and an intraoperative angiogram revealed an irregular “ground glass” endarterectomy site. Upon repeat arteriometry, the endarterectomy site was lined with platelet aggregates. The vessel was irrigated with low-molecular-weight dextran. The deficit she exhibited was attributed to diffuse embolization of the platelet aggregates at the time of carotid unclamping as well as to cerebral ischemia during carotid occlusion.

**CASE 5**

The final deficit occurred early in our experience with processed EEG tracings (1983). S. N. underwent an uneventful right carotid endarterectomy but awoke with left hemiplegia, which quickly resolved. Postoperative carotid duplex ultrasonography and cerebral computed tomography were unremarkable for intimal flap, cerebral bleeding, or cerebral infarction. On retrospective review of the intraoperative EEG, it was clear that ischemic changes were present. His deficit was attributed to untreated cerebral ischemia during carotid occlusion.

None of the patients in whom a postoperative neurologic deficit developed were noted to have any significant episodes of perioperative or postoperative hypotension prior to the development of their neurologic deficit.

**COMMENT**

Based on the data available in the literature, the incidence of death and/or permanent neurologic deficit after carotid endarterectomy is low. The reported fre-
quency of adverse outcomes has decreased over the last 20 years, although concerns still remain that these published experiences reflect only the very best academic and community practices. \(^\text{13}\) Regardless of the controversy, there is no argument that documenting and maintaining excellent results are critical to the continued use of the procedure. The recent highly publicized clinical trials that supported a surgical approach to both symptomatic and asymptomatic high-grade stenoses were all carried out in institutions where surgical outcomes were closely scrutinized. Indeed, one recent multicenter study\(^\text{1}\) concluded that the principal benefit of the procedure in asymptomatic patients, a reduced 5-year risk of ipsilateral stroke in those with greater than 60% stenoses, is dependent on achieving a 3% or lower perioperative morbidity and mortality.

The results of our consecutive series of 367 primary endarterectomies performed over a 13-year period doubtless reflect subtle improvements in patient selection, anesthetic and perioperative management, and surgical technique. Both mortality and morbidity were lower in the more recent era; comparing 1981 through 1987 with 1988 through 1994, the incidence of postoperative death decreased from 1.5% to 0%, while the incidence of stroke decreased from 1.5% to 0.5%.

Unfortunately, as in most retrospective reviews, definitive analysis of the factors contributing to these improved outcomes is difficult. Clinical decision making was modified slowly over time, while management by the attending physicians was tailored to individual patients rather than dictated by a rigid protocol. Finally, the low rate of complications limited statistical power. Nonetheless, we can compare our experience with other similar series that considered the causes of perioperative death and stroke and offer specific recommendations for the conduct of the operation, which we consider to be the most critical element of care.

**CONDUCT OF THE OPERATION**

While it is our preference to perform endarterectomy under general anesthesia, there is no question that similar results can be obtained with local or regional anesthetic techniques. The critical factors that determine outcome are appropriate perioperative management of cardiac risk factors, especially hypertension; early recognition and treatment of myocardial ischemia; and meticulous technique with avoidance of both embolization and cerebral ischemia.

As emphasized by others, exposure of the vessels must be carried out in an unhurried and gentle manner, mindful of surrounding cranial nerves, especially the vagus, hypoglossal, superior laryngeal, and glossopharyngeal nerves. We administer heparin and clamp the distal internal carotid prior to any manipulation of the bifurcation. Only then is the lateral carotid bulb sharply dissected free and rotated anteromedially. The arteriotomy is made in this lateral aspect and usually extended past the distal edge of the plaque. Unhindered visualization of the end point is essential. It is also important that the proximal extent of the endarterectomy achieves a suitable nondiseased segment of vessel. If there is any ques-

tion of the distal “feather” of the endarterectomy, fine-tacking sutures are placed; such sutures should not be tied too tightly or puckering of the luminal surface may result. It has also been our practice to tack the edges of the proximal end point if a thickened intimal layer has separated from the medial layer. Intraoperatively, Doppler signals are evaluated in both the internal and external carotid arteries. Completion duplex ultrasonography or arteriography is not used routinely; however, both are employed if deemed necessary.

Recently we have applied more liberal indications for placement of a prosthetic or vein patch (extensive arteriotomy into the internal carotid artery, vessels smaller than 3 mm, female sex, or active smoking). For example, patching was performed in about 10% of patients in 1981 through 1987 and nearly one third of patients in the more recent period. The type of patch does not seem to strongly influence early or late outcomes, but care must be taken to avoid excessively enlarging the artery and altering flow dynamics.

Our practice of shunting selectively is based on our confidence in the sensitivity of the EEG and the expertise of our colleagues who aid in EEG interpretation. Application of our criteria for ischemia led us to shunt approximately one fifth of the patients. Although an argument could be made that all patients could profit from maintenance of antegrade flow, there are situations in which even the most experienced surgeon cannot safely place or secure a shunt. Furthermore, by shunting selectively, complications of shunts including poor visualization of the end points, intimal injuries, and air embolization can be completely eliminated in most patients. Nonetheless, the fact that three of the five deficits in this series could be attributed to inadequate cerebral perfusion emphasizes that shunting, which is necessary in a minority of patients, remains an important adjunct to operative management.

**CAUSES OF PERIOPERATIVE DEATH**

Death following carotid endarterectomy is infrequent and is more commonly due to myocardial infarction than stroke. In our series, two of the three deaths were attributable to acute myocardial infarction, while none of the four patients suffering perioperative neurologic deficits died. This experience is not unique. In 1981, Lees and Hertzler\(^\text{6}\) reported a total of 10 postoperative deaths in 335 patients, many of whom underwent other major surgical procedures during the same hospitalization. Six of the 10 deaths were due to myocardial infarction and only two to stroke. In the multicenter Asymptomatic Carotid Atherosclerosis Study,\(^\text{2}\) only one patient in the 825-patient surgical group died following surgery; the cause of death was myocardial infarction. In the North American Symptomatic Carotid Endarterectomy Trial report\(^\text{1}\) in 1991, of 328 patients undergoing surgical treatment of severe carotid stenoses, two deaths were noted (0.7%), one from stroke and one from myocardial infarction or arrhythmia. In the multicenter Veterans Medical Centers study of asymptomatic stenoses described by Hossen et al,\(^\text{2}\) all four surgical deaths (1.9%) resulted from myocardial infarction.
The occurrence of myocardial ischemia and infarction has been linked to hypertension. Riles et al. specifically linked the overzealous use of α-adrenergic agents to increase carotid artery "stump" pressure intraoperatively with both myocardial ischemia and infarction. In their view, the incidence of myocardial infarction was 4.9% in 284 patients with hypertension as compared with zero in 207 normotensive patients. The well-described fluctuations in systemic blood pressure in the postoperative period (including hypertension and hypotension) also contribute to cardiac morbidity. With aggressive management of hypertension and tachycardia in our more recent cohort, the incidences of myocardial ischemia or infarction and cardiac-related death have been reduced to 1.9% and 0%, respectively.

The risk for myocardial complications and death appears to increase slightly with age. Meyer and colleagues reported an overall mortality of 1.3% in 749 carotid endarterectomies performed on patients 70 years of age or older between 1971 and 1989; six of 10 deaths were due to myocardial infarction. A more recent series of 63 endarterectomies in patients 75 years or older from Perler and Williams included five major cardiac complications but no deaths.

It has been well accepted that the indications for surgery and the neurologic status strongly influence outcome. For example, in a large series of more than 1700 carotid endarterectomies, Thompson reported an operative mortality rate of 3.4% for patients with previous stroke, 1.1% for patients with transient ischemic attack, and 0% for asymptomatic patients. This trend was not evident in our patients; two deaths were cardiac in nature and the intracerebral hemorrhage was unrelated to any area of previous infarction. None of the three had had a previous stroke, although the patient with hemorrhage did present with transient ischemic attacks increasing in severity. Hence, our more recent experience and that of others supports the fact that mortality reflects cardiac status and management, while neurologic morbidity reflects patient selection and the technical aspects of the operation.

CAUSES OF PERIOPERATIVE NEUROLOGIC DEFICITS

Ischemic neurologic events in our series were due to intraoperative cerebral ischemia (two permanent, one transient) and embolism (one with reperfusion, one at 4 hours postoperatively). Another patient undergoing emergency endarterectomy for a severe stenosis of the internal carotid artery experienced a fatal intracerebral hemorrhage from an unrecognized ipsilateral arterialovenous malformation. This distribution of mechanisms is consistent with that of most other large series. Riles et al. reviewed 3062 endarterectomies and noted that intraoperative ischemia, thrombosis and embolization, and intracerebral hemorrhage accounted for 47 of the 63 deficits. Of these, thromboembolic events after endarterectomy were the most common cause of deficit, accounting for 43% of the 55 deficits where a mechanism could be determined. Perdue reported a total of 31 temporary and permanent deficits in 1023 patients; 23 or 68% were ascribed to thromboembolism.

Based on a series of 818 endarterectomies, Rosenthal et al. differentiated the mechanisms of transient and permanent deficits. They suggested that embolism from ulcerated plaques was responsible for most transient deficits, since 73% of those patients suffering reversible complications had ulcerative plaques. In contrast, 10 of 15 patients with persistent and profound deficits experienced thromboses of the endarterectomy sites. This conclusion is supported by the Cleveland Clinic series of 260 operations described by Hertzer et al., in which 92% of intraoperative deficits occurred in patients with ulcerated lesions. Overall, deficits were noted in 9.9% of patients with ulcerations but in only 0.7% of those with nonulcerated plaques.

Perhaps the best data regarding the mechanisms of neurologic complications have been obtained in patients undergoing operation while awake, so that the time of the deficit can be more exactly determined. Steed et al. studied 359 elective endarterectomies; permanent strokes occurred in 1.7%, while temporary deficits were seen in 4.3%. Intraoperative deficits developed during dissection in three patients, during carotid occlusion in one, and with clamp release in two. It is interesting that in all of these instances, the onset of the deficit was correlated with changes in the EEG. Of the additional 15 deficits occurring in the postoperative period, 14 were ascribed to emboli, reperfusion phenomena, or hypotension.

In our series, we did not note poorer outcomes in patients with ulcerated lesions. While one deficit was likely due to unclamping a friable common carotid harboring postendarterectomy platelet deposition, the remaining three intraoperative complications were due to inadequate cerebral perfusion that was either unrecognized (one patient) or untreated owing to technical problems with shunt placement (two patients).

Perhaps our analysis was hampered by the difficulty we have experienced in reliably determining whether lesions are ulcerated or merely cavitated based on angiograms or duplex ultrasonograms; it is also our concern that intraoperative assessments of ulceration are potentially flawed by observer bias. It is most likely that the observations by others regarding the prevalence of athroembolism are accurate, but that continued refinements in operative technique, especially scrupulous avoidance of any manipulation of the atheromatous carotid bulb, have reduced the overall incidence of intraoperative emboli in current practice. This trend was also noted by Riles et al. in their longitudinal analysis of more than 3000 patients treated over 30 years. Regardless of the precise incidence, embolization during dissection is a very real hazard if attention to technical detail is not maintained.

As noted above, we determined that untreated intraoperative cerebral ischemia accounted for at least three of the five total ischemic deficits. None of the three patients had contralateral carotid occlusions. The literature is divided as to whether the existence of contralateral occlusions substantially influences outcomes, although there is general agreement that the incidence of ipsilateral ischemia during clamping is significantly higher in these circumstances. In our last 176 consecutive procedures, the incidence of ischemic changes was
50% in patients with contralateral occlusion and only 20% in those with patent carotid arteries. This parallels the incidence seen by other clinicians, eg, Mackey et al used EEG criteria and shunts in 46% of patients with contralateral occlusion but in only 13.5% of others. Imparato et al, who assessed cerebral perfusion in conscious patients, noted a 27% incidence of clamp intolerance in patients with contralateral occlusion as compared with 5.4% in those with patent vessels. This disparity is consistent with the higher sensitivity of the EEG, which may demonstrate significant amplitude reduction in higher frequencies well before mentation and motor function are affected.

We did not document any serious complications referable to postendarterectomy hyperperfusion syndromes, although a large number of our patients presented with risk factors for this phenomenon (hypertension, severe stenoses, and poor collateral blood flow). While it may be that the more subtle signs of defective postoperative cerebral autoregulation, such as headache, were not always acknowledged, no strokes or seizures occurred. Since this complication is undoubtedly related to the adequate control of perioperative hypertension, it appears that intensive monitoring and the use of more reliable antihypertensive agents have decreased the incidence considerably.

Based on our experience, the mortality of carotid endarterectomy can be minimized by appropriate and early diagnosis and treatment of intraoperative myocardial ischemia and hypertension. Precise operative technique is mandatory to avoid embolization during dissection and postoperative occlusion. In our patients, cerebral ischemia during carotid occlusion and embolization from endarterectomized surfaces accounted for all deficits.

Electroencephalographic monitoring can reliably identify the onset of cerebral ischemia, even in patients with contralateral occlusion. However, as noted by others, the sensitivity of EEG does not extend to those patients with marginally perfused and "electrically silent" areas surrounding recently injured brain. Hence, intraluminal shunts should continue to be used in all patients with unstable neurologic examinations or recent strokes (within 6 months), irrespective of EEG findings or other indicators of cerebral function and blood flow.

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REFERENCES


DISCUSSION

G. A. Sicard, MD, St Louis, Mo: Carotid endarterectomy, as everybody knows, is an example of a success story in surgery. This procedure has withstood the rigor of randomized trials initiated by skeptical colleagues and neurologists. The vascular service at the University of Chicago has shown with this work that with careful attention to technical details and a good method for intraoperative monitoring brain function (in their case, EEG) perioperative stroke risk can be kept lower than 1%. Similarly, with good patient selection and preoperative appropriate medical management, you can keep the cardiopulmonary complications also at the less than 1% level. Very few, if any, major surgical procedures can be performed with such good results. It is hard to argue with success, but I have a few questions for Dr Gewertz and his colleagues.

We prefer to perform this procedure under cervical block using the awake patient as a monitor of cerebral function. When you use the EEG, who reads it? When the EEG reader or technician is not available, what do you recommend as an alternative cerebral monitoring or protection maneuver for those surgeons who perform this procedure under general anesthesia? What is the incidence of false-positive EEGs in which shunting was not technically feasible, but the patient awoke with no neurologic deficit? In the manuscript, one of your five perioperative permanent deficits was due to what you call electri-
cally silent ischemia in a patient with a recent stroke that EEG monitoring did not detect. Should all the patients who are symptomatic or have had a stroke in the previous 6 months have perirheumatic contrast CT scans to look for this peri-infarct low-flow ischaemic zone? With such good results, have you altered your length of stay? Is a 23-hour carotid endarterectomy something that is here?

Laurens R. Pickard, MD, Houston, Tex: In some of these situations, both the neurologic complications and the cardiac complications can have their source in thromboembolic problems. More of these patients whom we are seeing have been on heparin preoperatively for some period of time. There has been discussion about rebound phenomenon after heparinization has been discontinued or reversed after the carotid endarterectomy, and I think that this may present some risks. Therefore, have these hypercoagulation problems been addressed or being considered in your work?

Robert J. Hye, MD, San Diego, Calif: One of the other factors that came out in both the NASCET and ACAS studies is that arteriography in these patients is associated with significant morbidity. I noted in the presentation, despite the excellent results, there was no mention of any neurologic morbidity that was incurred as a result of arteriography, which in the ACAS study was over 1%. Based on that data, one would have anticipated three to four strokes in this patient population, depending on the number who underwent arteriography. In general, that should be considered part of the periorchirurgical morbidity. As a consequence of this type of data and improved duplex scanning criteria, we, as have many centers around the country, have moved away from arteriography and save it for selected circumstances. I wonder if the authors could comment on that issue and tell us what their current approach is toward arteriography. Did any patients have a neurologic complication of their arteriogram in this series, or was that group excluded?

James J. Peck, MD, Portland, Ore: I agree with Dr Sicard. It is hard to criticize the carotid endarterectomy study which has a less than 1% mortality and less than 2% stroke rate. However, the title of the paper was "neurologic deficits." The cranial nerve dysfunction was not addressed in these patients. In retrospective studies, the incidence is from 1% to 18% and in prospective studies it is from 15% to 25%, primarily the marginal mandibular branch of the seventh nerve and the hypoglossal nerve. What was the authors' experience with this morbidity? Secondly, there is another way you can evaluate those. None of the patients who had a shunt placed had a central neurologic deficit. Yet, three of the five patients who had neurological complications should have had a shunt and yet the shunting was felt to be not technically feasible. This study strongly recommends the routine placement of a shunt. Atraumatic placement of the shunt will be refined with practice. Further, this would avoid the expense of the EEG and the technical expertise that it involves.

James R. Deborb, MD, Peoria, Ill: It would be a mistake to take away from this paper that the authors' excellent results are due to EEG monitoring. These results are primarily due to their excellent technical performance of this operation. Forty percent of the complications the paper focused on were embolic periorchirurgical events. Have they considered using perioperative intravenous dextran and local dextran irrigation to reduce thromboembolic events from platelet aggregation?

Dr Gewertz: I address Dr Sicard's comments first. The EEG in our operating room is read by a neurology technician, and in the absence of his or her presence, the anesthesiologist feels quite comfortable reading them. But it is not a problem even if we need to read it ourselves, since it is easily displayed. If there is no access to an EEG, in general probably the safest thing to do would be to shunt. This is particularly true in patients with contralateral occlusions where the incidence of ischemia is so high or in patients with relatively recent events. The other question you asked was an insightful one about whether patients had EEG abnormalities that for whatever reason weren't shunted and woke up without deficit. Indeed that did occur in a minimum of three patients, and I remember other patients in which their EEG would slow down in the middle of the operation and we would just continue and it would come back up. In point of fact, this is fully understandable, since the EEG is hypersensitive. It does become abnormal well before brain tissue is at risk. One of the main drawbacks in addition to the one that we acknowledged in terms of the ischemic penumbra region is the fact that it is very sensitive.

Should you always obtain a CT scan in patients with recent neurologic deficits? You probably should. We do it with contrast, and those patients should be shunted irrespective of their EEG changes. Still and all, that only led to shunting in about a quarter of the patients and we do believe the operation could be done more precisely and safely in the 75% of the other patients without the shunt.

In terms of the managed care question, which seems to dominate every discussion at medical meetings, yes, we do send patients home in 23 hours. The difficulty about making that sort of a statement is that as we have seen with deliveries, that becomes the national standard to send the patients home in 23 hours, which is clearly not the best care for many of the patients. So, if the patient is appropriate, we send them home. In terms of the issues about coagulopathy, we generally do reverse anticoagulation at the termination of the procedure. We do a large number of our current carotid endarterectomies, about 25% to 33%, without an angiogram. The point made about the complications of angiogram is a fair question. The problem is, to get that true incidence, you have to enroll every single patient who undergoes an angiogram for cerebrovascular disease as opposed to just patients whom we operated upon. Since after 13 years we had no way of going back and actually pulling out every single patient in our institution who underwent an angiogram for that reason, we could not obtain an incidence. I can assure you that the incidence of angiographic stroke at our institution is going to be less than 2%.

In terms of the question about cranial nerve dysfunction, that is a significant morbidity in the procedure. There have been a number of excellent studies, one of which was prospective, in which they very carefully and objectively evaluated cranial nerve dysfunction, and the incidence is around 20% temporary and around 3% to 5% permanent.

Now, in terms of the issues broadly addressed by a number of discussants about EEG, we are not saying in this paper, nor do we wish to be misinterpreted, that the principal reason for anyone's outcomes in carotid endarterectomy is the type of monitoring they use. I would suggest, however, that doing a neurologic operation without an EEG is not terribly dissimilar from doing a coronary operation without an ECG. The EEG is not perfect, but we do believe that if you put in 100 shunts, you are apt to get 1% to 2% complications either from the insertion of the shunt, or some compromise of the technical performance of the operation. In our hands the EEG seems to be a fairly reliable and very sensitive way of determining cerebral ischemia during cross-clamping.