Benefits, Shortcomings, and Costs of EEG Monitoring

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A 5-year experience with 562 carotid endarterectomies, using electroencephalogram (EEG) monitoring and selective shunting, was reviewed. EEG changes occurred in 102 patients (18%). The frequency of EEG changes, as related to cerebral vascular symptoms, was as follows: transient ischemic attacks, seven per cent (19/259); completed strokes, 37% (36/98); vertebral basilar insufficiency, 24% (32/135); asymptomatic, 21% (15/71). Patients with contralateral carotid occlusion exhibited EEG changes in 37% (28/76) of operations. Fifteen patients suffered perioperative strokes (2.6%). Nine of the 15 were associated with a technical problem of either thrombosis of the internal carotid artery (five) or emboli (four). Technical problems were more common when shunts were used (five per cent) than when they were not (0.9%). Patients who suffered strokes prior to surgery were more at risk to develop a perioperative stroke (three per cent) than those not suffering prior strokes (0.3%). The EEG did not change in three patients who had lacunar infarcts prior to surgery and who awoke with a worsened deficit. Our series does not clearly establish the advantages of EEG monitoring, which is expensive ($375/patient) and may not detect ischemia in all areas of the brain. However, the use of shunts may introduce a risk of stroke due to technical error that is equal or greater than the risk of stroke due to hemodynamic ischemia. Since the need for protection is unpredictable by angiographic or clinical criteria, the benefit of EEG monitoring may be in reducing the incidence of shunting in those patients whose tracing remains normal after clamping. The decision to shunt, however, when there is electrical dysfunction after carotid clamping should be based not only on the EEG but also on the clinical signs and computed tomography (CT) scan. Our data does not show a net benefit in selective shunting unless the patient has sustained a stroke prior to surgery.

A CONSIDERABLE AMOUNT of time, energy, and anguish have gone into the effort to prevent immediate new neurologic deficits after carotid endarterectomy. Despite the multifactoral etiologies of these deficits, the controversy always seems to center around the use or nonuse of the temporary indwelling shunt during carotid clamping. Routine shunting, elective shunting, and no-shunting techniques each have proponents.[1-4] No technique, however, can be shown to produce superior results in comparison to the others, and each technique has its flaws. The advocacy of no shunting at all seems unacceptable since some small percentage of patients will awake with a new neurologic deficit if not shunted. Likewise, the routine use of shunts is not only unnecessary but may add to the operative risk. Unfortunately, no monitoring technique will identify only those patients who if not shunted would develop a stroke as a consequence. Each monitoring technique is oversensitive and the issue of whether any *net benefit* is derived from this selective process must be addressed.

We have reviewed our experience with 562 patients undergoing carotid endarterectomy with continuous EEG monitoring at the University of Rochester Medical Center since 1979. The first question is, "Can certain groups of patients, those who are particularly vulnerable to develop cerebral ischemia with carotid clamping, be identified either by angiographic or clinical criteria? Second, can all patients be identified who are at risk for clamp-induced strokes? Third, does selecting patients supposedly at risk for developing a stroke due to carotid clamping and shunting them actually reduce the net stroke rate? Finally, what is the cost of this selection process, and can it be justified on a broad scale in this period of cost consciousness?

Methods

Five hundred sixty-two patients underwent carotid endarterectomy at the University of Rochester Medical Center, Strong Memorial Hospital, with continuous 12-lead EEG monitoring[5] from November 1978 to January 1984. A neurologist was present in the operating room. Normocarbic general anesthesia was used except in rare situations when, for medical reasons, a superficial cervical block was employed. Indwelling arterial catheters were used whenever possible to monitor blood pressure during

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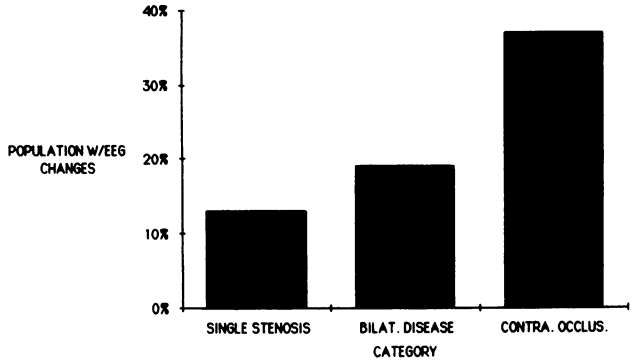

FIG. 1. The angiographic predictors of EEG changes with carotid clamping. Patients were considered to have a single stenosis when the contralateral internal carotid artery had less than a 50% diameter reduction.

the operation and around the operative period. The need for temporary shunting during clamping was determined by the EEG recording and the preference of the operating surgeon. Those EEG changes, either loss of voltage or loss of activity, that occurred less than 3 minutes after clamping were considered significant, and whenever possible a shunt was inserted. EEG recordings were continued until the patient was transferred to the recovery room. Patients who awoke with a new deficit were immediately evaluated with oculoplethysmography (OPG-Gee) and appropriate action taken.

Results

Angiographic Predictors of EEG Changes

Patients were considered to have a single carotid stenosis when the contralateral internal carotid artery had a stenosis of less than 50% diameter reduction. EEG changes were observed in 42 of the 321 patients with single carotid stenoses (13%). Changes occurred in 32 of the 165 patients with bilateral stenoses (19%). These were patients whose contralateral carotid artery had a stenosis of greater than 50% diameter reduction.

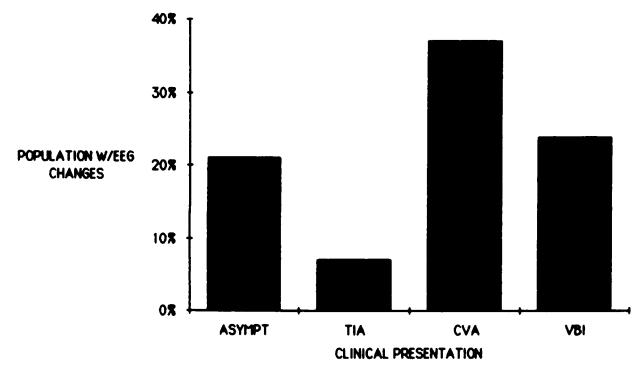

FIG. 2. Clinical predictors of EEG changes with carotid clamping.

In the group of patients with contralateral occlusions, 28 of the 76 patients developed changes with clamping (37%). The increased incidence of EEG changes in the group of patients with contralateral internal carotid artery (ICA) occlusion when compared to patients with a patent or stenotic contralateral ICA is highly significant, $p = 0.01$ (Fig. 1).

Clinical Predictors of EEG Changes

EEG changes occurred in 15 of the 71 asymptomatic patients (21%). Nine of the 15 asymptomatic patients who developed EEG changes with carotid clamping had previously undergone contralateral carotid endarterectomy for symptoms without EEG changes, and each of these patients had testing to document patency of the previously operated side. EEG changes occurred in 19 of the 259 patients operated on for transient ischemic attack (TIA) (7%). There were EEG changes in 36 of the 98 patients operated on for stroke (37%). Our policy in recommending operation to patients with stroke was to wait at least 6 weeks from the onset of symptoms unless progression was evident and a high grade stenosis was present. In those patients with reversible ischemic neurologic deficit (RIND), operation was carried out when the neurologic deficit resolved. CT scanning was used to determine the timing of the operation in patients with stroke and, in general, patients with large defects were not considered candidates for operation. EEG changes occurred in 32 of the 135 patients operated on for vertebrobasilar insufficiency (VBI) (24%). The increased incidence of EEG changes in those patients who had sustained neurologic damage prior to surgery is highly significant ($p = 0.01$) compared to all the other groups (Fig. 2).

Angiographic and Clinical Correlates of EEG Changes

If both the angiographic and the clinical classifications of each patient are considered, the likelihood of a patient developing EEG changes can be more accurately predicted (Fig. 3). Patients with either a stroke or vertebrobasilar insufficiency *and* a contralateral internal carotid artery (ICA) occlusion have a 50% chance of developing cerebral ischemia when the carotid artery is clamped. When compared to all the other groups of patients, the increased incidence of EEG changes observed in these two groups of patients is highly significant ($p = 0.01$).

Are All Patients at Risk for Stroke Identified by the EEG?

The results in patients with completely normal intraoperative EEG recordings were examined to determine if any patient with a normal EEG awoke with a new

neurologic deficit. Patients with defects due to technical problems were excluded and are dealt with elsewhere in this analysis. There were 449 patients with completely normal EEG recordings. No shunts were used in these patients. Immediate ipsilateral neurologic deficits were noted upon awakening in three of these patients (0.7%). Prior to surgery, each of these three patients had sustained lacunar infarcts confirmed by CT scan and the new deficit represented a significant worsening of the situation.

Shunting requirements

The need for shunting was determined by the EEG recording and the preference of the surgeon. There were 460 patients with normal EEG recordings (82%). No shunts were used in 449 of these patients. Eleven patients were shunted because of surgeon's preference: five because of a recent stroke, three because of multiple vessel occlusions, and three because of difficulty in obtaining distal control. There were 102 patients that had EEG changes. Shunts were inserted in 91 of these patients. Eleven patients were operated on without shunts: six because the changes occurred more than 3 minutes after clamping, four because a shunt could not be inserted, and one because of misinterpretation of the recording.

Cause of Perioperative Strokes

New or significantly worsened neurologic deficits occurred in 15 of the 562 patients (2.7%). Thrombosis of the internal carotid artery immediately after surgery resulted in stroke in five of the 15 patients (33%) with new deficits. These patients were all re-explored. In four patients, a platelet-white thrombosis deposition was found on the endarterectomized surface; in one patient, a distal intimal flap was noted. Two of the 15 patients (13%) awoke without deficit but developed a stroke 1 day and 3 days later. Noninvasive testing revealed a patent internal carotid artery and a presumptive diagnosis of embolus was made. Difficulty with insertion of the shunt was associated with stroke in two of the 15 patients (13%). In one of these cases, a piece of plaque from the proximal artery was seen traversing the shunt. In the other, the shunt injured the distal internal carotid artery. A completion angiogram, which was normal, was done in this patient. The patient awoke normally, but 4 hours later a stroke developed. It was determined by OPG-Gee criteria that the internal carotid artery had thrombosed. The EEG failed to detect ischemia in three of the 15 patients (21%). As mentioned, these patients all had suffered lacunar infarcts and awoke with significantly worsened deficits. Noninvasive studies were done in each case, and the internal carotid artery was proven open to our satisfaction. Two of these patients had

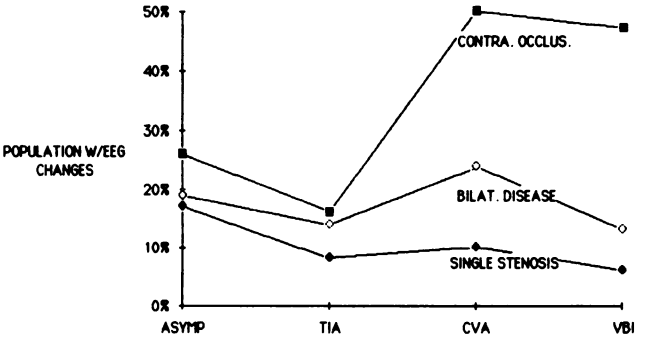

FIG. 3. Correlation of EEG changes with both angiographic and clinical criteria. Patients with contralateral internal carotid artery occlusions and either preoperative stroke or vertebrobasilar insufficiency are particularly vulnerable to carotid clamping.

recent strokes (less than 2 weeks prior to surgery). The indwelling shunt failed to provide enough flow in two of the 15 patients (13%). In one of these patients, the EEG improved with shunt insertion but did not normalize. In the other patient the EEG did improve, but the patient developed a worsened stroke. This patient had suffered multiple, bilateral strokes before surgery. The EEG was misinterpreted in one of the 15 patients (7%). This patient had suffered a stroke before surgery and was abnormal at the start of the procedure. The changes that occurred with clamping were initially felt to be similar to those already present. This patient awoke with a worsened deficit and upon review of the record it was felt that an electrical deterioration had occurred with clamping (Fig. 4).

The Causes and Effects of Technical Error

Analysis of these results reveals that of the 15 patients who suffered a stroke after surgery, nine (60%) were due to technical errors and only six (40%) were actually due to clamp-induced cerebral ischemia. There were 456

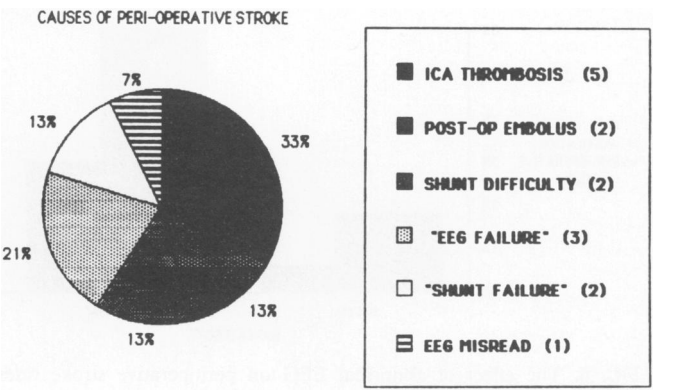

FIG. 4. The causes of perioperative strokes. Technical errors were responsible for nine of the 15 (60%) strokes in this series.

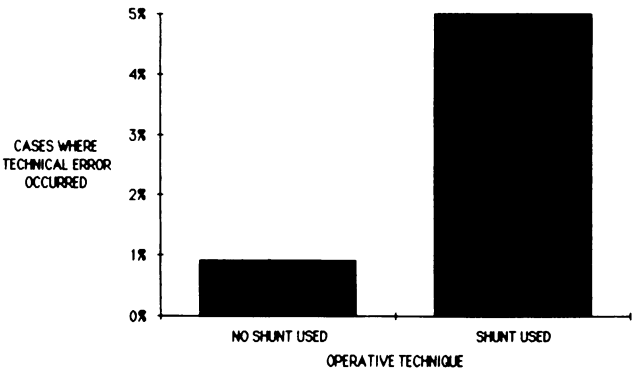

FIG. 5. The chance for technical error increased from one to five per cent in cases where a shunt was used.

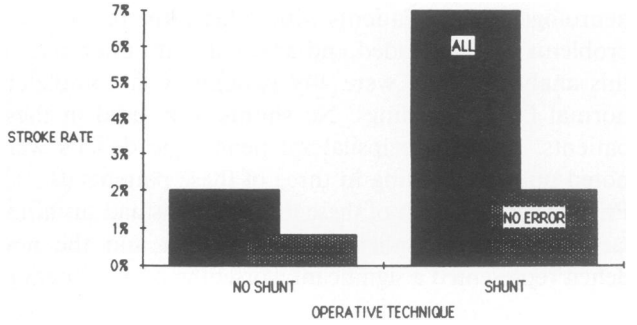

FIG. 7. The relationship between shunting and perioperative stroke. When technical error is eliminated (shaded areas), there is no difference in the stroke rate between shunted and unshunted patients.

patients operated on without shunts and four of these patients (0.9%) had technical errors which resulted in a stroke. On the other hand, 102 patients were operated on with shunts and five of these patients (five per cent) had a technical error which resulted in a stroke. The use of a shunt results in a statistically significant ($p = 0.01$) increase in the number of strokes due to technical error (Fig. 5).

Determinants of the Postoperative Stroke Rate

The effects of an abnormal EEG. The stroke rate for the 460 patients with a normal EEG is two per cent. If those cases where technical error influenced the development of a stroke are eliminated, the stroke rate in this group is 0.6%. The stroke rate for the 102 patients with abnormal EEG is six per cent. Eliminating technical error, the stroke rate for patients with an abnormal EEG is three per cent. This difference is statistically significant, $p = 0.05$. Therefore, patients with an abnormal EEG, even when successfully shunted and eliminating all technical errors, have a greater chance of developing a stroke after surgery when compared to those patients with a normal EEG (Fig. 6).

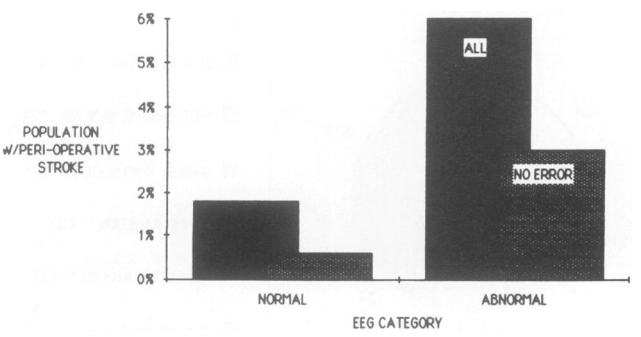

FIG. 6. The effect of abnormal EEG on perioperative stroke rates. When strokes due to technical errors are eliminated (shaded areas), there is an increase in the perioperative stroke rate in the patients with an abnormal EEG.

The effects of shunting. The stroke rate for the 460 patients operated on without shunts is two per cent. The stroke rate for the 102 patients operated on with shunts is seven per cent. This difference is highly statistically significant ($p = 0.01$). If, however, the technical error factor is eliminated, the stroke rates of one per cent for the nonshunted patients and two per cent for the shunted patients are not different (Fig. 7).

The effects of the clinical symptoms on stroke. The stroke rates based on clinical symptoms were as follows: for asymptomatic patients, 2.8%; for patients with TIA, 2.3%; for patients with stroke, 6.1%; and for patients with VBI, 0.7%. When compared to all the other groups, the patients with stroke had a significantly higher stroke rate after surgery, ($p < 0.05$). If the cases where technical errors resulted in stroke are eliminated, the true risk of stroke after carotid endarterectomy is brought out. There were no strokes in asymptomatic patients or patients with VBI when technical error is eliminated. One of 236 (0.4%) TIA patients in this category suffered a stroke after surgery, but 3 per cent of the patients with preoperative stroke, despite monitoring and shunting without error, went on to develop another stroke. This difference comparing patients with preoperative stroke to the others is highly significant, $p < 0.01$ (Fig. 8).

The results with shunting based on surgeon's preference. The stroke rate in the 449 patients with normal EEGs who were not shunted was 2 per cent. The stroke rate for the 11 patients with normal EEG who were shunted because of the surgeon's preference was 18%. A technical error that led to thrombosis of the internal carotid artery was made in each of the two patients in this group that developed a stroke. The stroke rate for the 91 patients who developed EEG changes and were shunted was five per cent. The stroke rate was nine per cent for the group of patients with abnormal EEGs who were not shunted. There is no statistical difference in the stroke rates for the patients with abnormal EEGs, whether shunts were or were not used. On the other

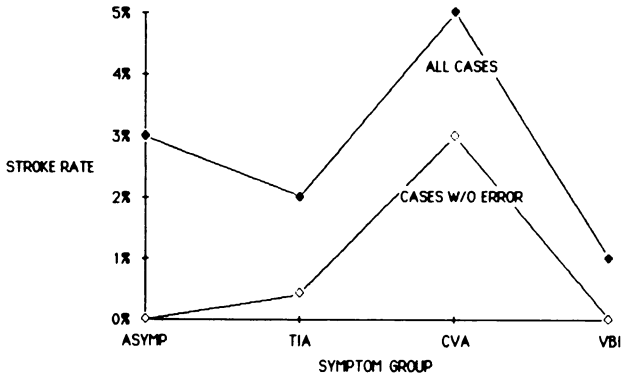

FIG. 8. The effects of clinical presentation on perioperative stroke rates. When strokes due to technical errors are eliminated (bottom line graph), there is a significantly greater risk of stroke in the patients with preoperative stroke.

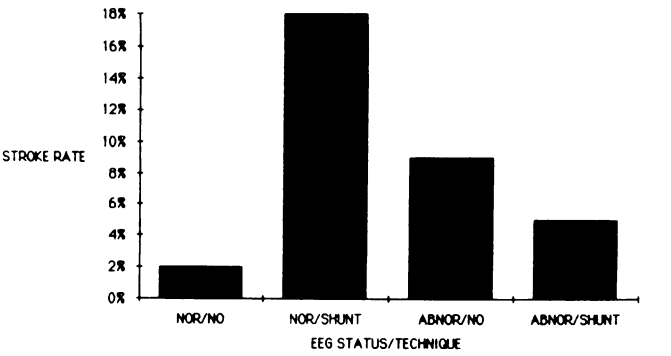

FIG. 9. Shunting based on the surgeon's preference. Patients with normal EEGs should not be shunted.

hand, the stroke rate in the patients with normal EEGs is significantly higher when shunts are used (Fig. 9).

The cost of monitoring. The costs of EEG monitoring include equipment expenses, technician salaries, and neurologist fees. These costs are currently reimbursed by third party carriers including Medicare. The average yearly expenses of routine monitoring at our institution are $42,225 and, based on 125 cases per year, it costs $375 per patient. The cost of identifying an abnormal patient is $2069. Since the equipment costs and the technicians salaries are fixed regardless of the patient volume, the cost per patient would not change significantly with a change in patient volume.

The cost of identifying a patient at risk for clamp-induced cerebral ischemia was calculated for each patient group. It costs $1775 to find an abnormal patient in the group of asymptomatic patients; $5111 to find an abnormal patient in the group of patients with TIA; $1020 to find an abnormal patient in the group of patients with stroke; and $1582 to find an abnormal patient in the group with VBI (Fig. 10).

Discussion

No technique of carotid endarterectomy has been shown to produce superior results.[1-4,6,7] This simple undeniable fact, however, does not prevent heated debate among the true believers in the various methods of operation. What can be accepted by all is that most carotid endarterectomies can be safely done without a shunt. Strokes upon awakening due to clamp-induced cerebral ischemia are a rare event, ranging from one per cent in combined series of patients who are neurologically stable to 11% in patients with the combination of stroke before surgery and contralateral occlusion.[2,8-10] The postoperative stroke rate of three per cent for those patients in our series with preoperative stroke, even when successful shunting is employed and technical error is eliminated, is significantly higher than the rate of 0.3% for all the other patients. This experience is not different from that of Sundt, who reports a 4.5% postoperative stroke rate in similar patients, again in spite of monitoring and selective shunting.[2] Identifying these high risk patients is therefore not the problem. The task confronting the proponents of monitoring and selective shunting is establishing that the net stroke rate in patients at risk for a hemodynamic ischemic stroke can be lowered by shunting.

EEG changes with carotid clamping tend to occur in those patients with more severe degrees of occlusive disease (*i.e.* contralateral occlusion) and those patients having either stroke or global ischemia due to carotid stenoses.[2,7] Kelley et al. have pointed out the high frequency of EEG changes with carotid clamping in patients operated on for VBI (24%) compared to patients with carotid territory symptoms.[11] Our findings corroborate those of the Tufts series. Unfortunately, these trends toward a higher frequency of EEG changes with increasingly severe disease are not predictive for any given patient. The fact that nine of 15 asymptomatic patients, who had previously undergone carotid endar-

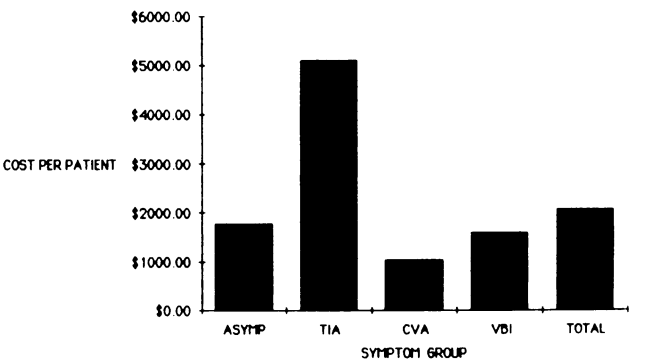

FIG. 10. The costs of EEG monitoring. It costs $2069 to find each abnormal patient.

terectomy for a symptomatic lesion without developing EEG changes, developed EEG changes when their second side was done points out the unpredictability of the situation. These are patients who should have been safe but were not. Retrospectively, we learned that several of them had suffered silent cerebral infarcts and, therefore, were not really asymptomatic. This experience has broadened our indication for CT scanning before surgery.

The unpredictability of the need for cerebral protection and the fear that a patient will awaken with a new deficit has led to the use of shunts. The major objection to selective shunting is that the present monitoring techniques are oversensitive and result in patients who are not at risk for stroke being shunted unnecessarily. This oversensitivity is due to a difference in the amount of cerebral blood flow necessary to maintain electrical function and that necessary to maintain the structural integrity of brain tissue. Electrical function deteriorates immediately at cerebral flows of less than 20 ml/100 g/min, but the development of morphologic damage is time-dependent and occurs at flows of 6 to 8 ml/100 g/min.[12,13] The risk of shunting is evident in our series. The stroke rate due to technical error in the 456 patients operated on without a shunt was 0.9%, but it was five per cent in the 102 patients operated on with shunts. In two of these patients, the poor result was directly attributable to the shunt. In the other three patients, thrombosis occurred from a platelet-fibrin clot that we feel may be due to intimal damage from the shunt insertion. Others have discussed this risk[14,15] but have not quantified the role of the shunt itself in the development of stroke. In our series the shunt could be implicated in the development of five of the 15 strokes (33%). This risk must be considered and balanced against the risk of not shunting.

In addition to being oversensitive (false-positive), the EEG may not select every patient that requires shunting (false-negative). Significantly worsened neurologic deficits occurred in three patients who had suffered lacunar infarcts before surgery; this occurred despite normal EEG recordings. This is at variance with the work of Sundt and Chiappa, who report that no patient in their series awoke with a new deficit without a clue from the EEG.[2,16] The only problem with this is that the Mayo Clinic Group also uses regional cerebral blood flow determinants and prophylactically shunts those patients with marginal blood flows to 15 to 20 ml/100 g/min with normal EEGs because they fear that the EEG may not reveal regions of focal ischemia deep in the white matter or basal ganglia. Their shunt usage rate of 44% exceeds ours of 18%, as well as the 10 to 29% range reported by others.[16,17] It is accepted that the small size and deep location of the lacunar infarct make conventional EEG diagnostic in only 34% of patients.[18] In fact, the finding of a normal EEG in the presence of neurological deficit is a criterion suggesting the diagnosis of a lacunar infarction.[19] The possibility of a falsely negative EEG in a patient with a lacunar infarction should, therefore, be recognized and addressed.

The importance of the difference between the flow threshold for electrical signs of cerebral ischemia and the threshold for morphologic damage is exemplified in our groups of patients operated on for vertebrobasilar insufficiency and stroke. EEG changes occurred more frequently with carotid clamping in these two groups, especially when the contralateral carotid was occluded where the frequency of changes approached 50%. Despite equal frequencies of clamp-induced cerebral ischemia in these two groups, the risk of stroke was quite different. There were no strokes in the group of patients with VBI when technical errors were eliminated. On the other hand, three per cent of the patients operated on with preoperative stroke developed a new or a significantly worsened neurologic deficit after surgery even when technical error was eliminated. This difference is highly significant and suggests that patients who have not sustained prior strokes can tolerate short periods of cerebral ischemia with loss of electrical function but not structure. On the other hand, those patients who have already lost structure are more susceptible to low flows and require a higher perfusion pressure to prevent increased damage. Patients who have sustained a cerebral infarct are truly at risk and the advocacy of no shunting in these patients is unacceptable, since the duration of each EEG change that can be tolerated before a neurological complication occurs is unknown.

It could be argued, therefore, that monitoring should only be done in the high risk patient since the groups without morphologic damage are unlikely to sustain a stroke, even though they might develop electrical dysfunction with carotid clamping. This point can be argued both from a financial and a medical standpoint. Our cost of $375 per patient consists of equipment costs, technician salaries, supplies, and neurologist's fees and is based on a yearly expense of $42,225. If 50% of the patients were monitored, eliminating those patients thought to be a low risk for stroke, our costs would only decrease slightly because the equipment and technical costs would not vary. If monitoring patients who are at a very low risk for stroke results in their being shunted unnecessarily, and thereby increases their risk for stroke due to technical error, then the argument can be made that only the patients at high risk should be monitored. A claim has been made that the assessment of medical and angiographic risk factors was more useful in identifying high risk patients than was the use of monitoring.[20] If one considers that silent cerebral infarcts do occur—as they did in at least five of the nine patients in our

group of asymptomatic patients who had profound EEG changes—one realses that the high risk patients are very difficult to identify by angiographic or clinical criteria. Since there is neither cost savings nor medical justification for selective monitoring, its use cannot be supported.

Our series does not clearly establish the advantages of EEG monitoring. It is expensive, oversensitive, and may not detect ischemia in all areas of the brain. On the other hand, the use of shunts (which in our experience may be hazardous and are not always clearly indicated by angiographic or clinical criteria) can be decreased if the EEG data is used wisely. We therefore feel that, despite its shortcomings and costs, EEG monitoring is an accurate method for identifying patients under general anesthesia with cerebral cortical ischemia after carotid clamping. The decision, however, of whether to shunt the patient with electrical dysfunction after carotid clamping should be based not only on the EEG but also on the clinical presentation and the CT scan of the brain. Our data do not show any net benefit in selective shunting unless the patient has suffered a preoperative stroke. In other patients, the act of shunting introduces a risk of stroke due to technical error that at least equals the risk of stroke due to hemodynamic ischemia.

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Discussion

DR. JESSE E. THOMPSON (Dallas, Texas): I enjoyed Dr. DeWeese's paper, with his usual very careful analysis of the problem. Certainly, EEG monitoring is one of the accepted methods of monitoring patients during carotid endarterectomy under general anesthesia, and his excellent results bear this out.

I have no quarrel with this approach, but we have taken the position of using a shunt routinely, and hence do not find it necessary to employ EEG monitoring. This has served us well over the years, and our incidence of neurologic deficits related to the operation has been less than two per cent.

Complications related to the shunt itself are, at least in our hands, virtually nil when one uses it all the time, and I really do not consider it hazardous.

(Slide) The advantages of the shunt are listed here. Actually, it is really quite cheap; our little shunt costs about 20¢. It is simple, quick, safe, and always available. It is effective. It allows for no-haste surgery and acts as a stent for closure. We have found it especially useful for complicated lesions and for patch angioplasty. We have not had the problem with the white thrombi with the use of the shunt as Dr. DeWeese has described.

The disadvantages of the shunt are listed on the next slide. (Slide) (Laughter) As a result of this, we have continued to use the shunt routinely, and really find it quite advantageous.

DR. DAVID ROSENTHAL (Atlanta, Georgia): Dr. DeWeese and his associates are to be congratulated for this cost/benefit analysis, and for identifying a group of patients who should be considered at high risk during endarterectomy, the poststroke, or the postRIND (reversible ischemic neurologic deficit) patient.

(Slide) As Dr. DeWeese mentioned—and I agree—the safest method for performing endarterectomy is under EEG surveillance in the so-called neurologically "stable" patient. These are patients who have experienced a TIA, amaurosis fugax event, or vertebro-basilar insufficiency symptoms. These neurologically stable patients will generally tolerate cross-clamp well, and in a previous report by our group on some 900 patients we could find no statistical difference in the incidence of postoperative neurologic deficits when endarterectomy