Continuous electroencephalographic monitoring and selective shunting reduces neurologic morbidity rates in carotid endarterectomy

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Purpose: **The role of continuous electroencephalographic (EEG) monitoring during carotid endarterectomy was evaluated in this retrospective review.**

Methods: **We analyzed data from 902 consecutive carotid endarterectomy procedures performed with vein patch angioplasty. In 591 operations from 1980 to 1988 we did not use intraoperative EEG monitoring or shunting (non-EEG group). Continuous intraoperative EEG monitoring and selective shunting were used in 311 procedures from 1988 to 1994 (EEG group). The patients' mean age was higher in the EEG group (68.8 years; range, 41 to 87 years) than in the non-EEG group (66.2 years; range, 34 to 90 years; $p < 0.001$). There was also a significantly higher incidence of hypertension (56.2% vs 41.9%) and redo operations (5.4% vs 2.54%) in the EEG group than in the non-EEG group ($p < 0.05$). The operative technique was identical in both groups. We defined a significant EEG change as a greater than 50% reduction of the amplitude of the faster frequencies, a persistent increase of delta activity, or both.**

Results: **In the EEG group, acute EEG changes occurred in 40 patients (12.8%); 31 (77.5%) unilateral and ipsilateral to the operated carotid artery, and nine (22.5%) bilateral. In five patients (12.5%) the changes correlated with an intraoperative episode of hypotension, and after normal blood pressure was restored the EEG returned to normal. In 35 procedures (87.5%) a carotid shunt was inserted. In 33 of those patients the EEG returned to baseline, in one patient there was a significant improvement, and in one patient the EEG changes persisted. Postoperative hospital strokes occurred in one patient (0.32%) in the EEG group and in 13 patients (2.19%) in the non-EEG group ($p < 0.05$). All strokes (n = 14) were ipsilateral to the operated carotid artery. Of the 13 strokes in the non-EEG group nine were major and four were minor. The one stroke in the EEG group was embolic in origin and occurred before carotid cross-clamping; it was associated with profound EEG changes that did not reverse after placement of a shunt. In the total group (n = 902), intraoperative EEG monitoring was inversely associated with postoperative stroke ($p < 0.05$).**

Conclusion: **The overall neurologic morbidity rate was significantly lower in the EEG group than in the non-EEG group, thereby demonstrating the value of intraoperative EEG monitoring in carotid endarterectomy. (J Vasc Surg 1997;25:620-8.)**

Carotid endarterectomy has been established as the treatment of choice for patients who have significant carotid artery occlusive disease, as long as the perioperative mortality and neurologic morbidity rates remain low.[1-5] Further reducing or eliminating the rate of perioperative stroke and postoperative complications will further improve the overall results of the procedure.

However, controversy persists about the pathogenesis of surgical complications and the proper use of shunting to reduce them. Several authors have performed carotid endarterectomy without a shunt,[6-11] whereas others have used shunts routinely.[12,13] Still others have proposed using shunts only in patients (detected by hemodynamic or electro-

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physiologic monitoring) in whom significant and prolonged cerebral ischemia indicated a high risk of infarction.[14-19]

Because the incidence of stroke after carotid endarterectomy is so low, no surgical group has gathered sufficient data to prove the advantage of a specific policy regarding shunts. In this series we had the unique opportunity to evaluate retrospectively the role of continuous electroencephalographic (EEG) monitoring in carotid endarterectomy. We compared two groups of patients who underwent carotid endarterectomy by the same surgeon. In one group the procedure was done without a shunt, whereas in the other group EEG data was continuously monitored and shunts were selectively used.

MATERIALS AND METHODS

Study design. We reviewed the records of 751 patients who underwent a total of 902 consecutive carotid endarterectomy procedures along with homologous vein patch angioplasty between January 1980 and December 1994. In 591 procedures (65.5%) cerebral function was not monitored during the carotid endarterectomy (non-EEG group); shunts were not used in these procedures, which were performed between 1980 and 1987. In 311 procedures (34.5%) intraoperative EEG data was continuously monitored, and shunts were used selectively on the basis of EEG changes (EEG group). These procedures were performed between 1988 and 1994. Patient records used in the study included preoperative profiles, common operative details, and postoperative in-hospital follow-up.

Operative technique. All procedures were performed with the patient under general endotracheal anesthesia and receiving systemic heparin (100 U/kg), which was reversed with protamine sulfate at the end of each operation. The choice of general anesthesia was made by the anesthesiologist and did not depend on specific clinical features. Induction was accomplished with either pentothal or etomidate. Narcotics were part of the induction in all patients (fentanyl was usually used). Anesthesia was maintained using a low concentration of isoflurane or enflurane and a variable-rate infusion of fentanyl. Each anesthetic was titrated to permit accurate EEG interpretation while maintaining systolic blood pressure within 20% of each patient's baseline value. Hypertension (an increase in systolic blood pressure >20% of baseline) was treated by increasing the volatile anesthetic, the narcotic, or both and by administering vasodilators if needed. Conversely, hypotension (a decrease in systolic blood pressure >20% of baseline value) was treated by decreasing the anesthetic or narcotic concentration and administering isotonic fluids or colloids. Vasopressors were administered if the above measures were unsuccessful. In addition, vasopressors were used before a shunt was placed when an ischemic EEG event associated with hypotension occurred and did not resolve after anesthetic manipulation and fluid administration.

EEG data. Scalp electrodes were applied during the preoperative period using collodion adhesive. Ten cephalic electrodes were placed according to the international 10-20 system of electrode placement. In addition, grounding and noncephalic electrodes were placed. Ten-channel Nihon-Kohden electroencephalographs were used.

Eight or ten EEG channels were continuously monitored during surgery using a single bipolar montage with leads from the frontal, central, occipital, and temporal regions. Additional channels were used to record the electrocardiogram and movement. The high-frequency filter was set at 35 Hz and the low-frequency filter at 0.03 Hz. The sensitivity was between 2 and 5 μV/mm. Paper speed was 5 to 15 mm/sec for the recording during the procedure.

A baseline electroencephalogram was obtained for each patient. Any preexisting voltage asymmetries, focal slowing, or other focal or lateralizing features were determined. Once the patient had been anesthetized and was asleep, additional recordings were obtained. During this period, sensitivity and paper speed settings were adjusted to enhance the appearance of activity among faster frequencies. Monitoring was maintained throughout the operation and for several minutes after the surgical procedure was complete. A clinical neurophysiologist continuously assessed the EEG throughout the monitoring period. Changes in the EEG were reported immediately to the surgeon by the attending clinical neurophysiologist, and the persistence or resolution of the changes was monitored.

We defined a significant EEG change as a reduction greater than 50% in the amplitude of the faster frequencies, an obvious and persistent increase of slow (delta) activity, or both. Changes were characterized as unilateral or bilateral. Also, all acute changes were correlated with the time of carotid clamping, any significant changes in the systemic blood pressure, and changes in the depth of anesthesia.

Statistical analysis. Data taken from charts were entered in a custom-designed database created in the 4th Dimension relational database for Macintosh, Version 2.2 (ACIUS, Inc., Cupertino, Calif.) and

Table I. Comparison between EEG and non-EEG groups by univariate analysis

	non-EEG group (n = 591)	*EEG group (n = 311)*
Sex		
male	348 (58.8%)	192 (61.7%)
female	243 (41.2%)	119 (38.3%)
Age >62 yr	423 (71.5%)	263 (84.5%)*
Smoking	408 (69%)	218 (70%)
Hypertension	248 (41.96%)	175 (56.2%)*
CAD	165 (27.9%)	106 (34%)
Diabetes	66 (11.2%)	43 (13.8%)
History of CVA	77 (13%)	53 (17%)
History of TIA	271 (45.8%)	158 (50.8%)
Nonhemispheric symptoms	58 (9.8%)	16 (5.14%)
Asymptomatic	185 (31.3%)	84 (27%)
Repeat CEN	15 (2.54%)	17 (5.4%)†
Previous CEN	136 (23.01%)	80 (25.6%)
Ipsilateral stenosis ≥75%	564 (95.4%)†	270 (86.8%)
Contralateral stenosis ≥50%	175 (29.6%)	90 (28.9%)
Contralateral occlusion	27 (4.56%)	26 (8.36%)
Postoperative CVA	13 (2.19%)	1 (0.32%)†
Postoperative death	2 (0.33%)	2 (0.64%)
Postoperative MI	3 (0.5%)	2 (0.64%)
Postoperative RF	2 (0.33%)	0 (0%)

CAD, Coronary artery disease; *CVA,* cerebrovascular accident; *TIA,* transient ischemic attack; *MI,* myocardial infarction; *RF,* renal failure.
*$p < 0.001$.
†$p < 0.05$.

were subsequently transferred to a DEC VAX 11/750 running VAX/NMS (Digital Equipment Corp., Maynard, Mass.). Univariate analysis testing included Fisher's exact text and Pearson's χ^2 test for categorical data and Student's t test for continuous variables. The BMDP statistical software 1990 package (BMDP Statistical Software, Inc., Los Angeles) was used for all statistical computations.

RESULTS

Patient data. Demographic data and preoperative clinical risk factors for both groups are shown in Table I. The mean age was higher in the EEG group (68.8 years; range, 41 to 87 years) than in the non-EEG group (66.2 years; range, 34 to 90; $p < 0.001$). There was also a higher incidence of hypertension (175 patients; 56.2%) in the EEG group than in the non-EEG group (248 patients; 41.96%; $p < 0.001$).

Eighty patients (25.6%) in the EEG group and 137 patients (23.01%) in the non-EEG group had previously undergone carotid endarterectomy ($p > 0.05$). The incidence of patients who were undergoing a repeat operation was greater in the EEG group (n = 17; 5.4%) than in the non-EEG group (n = 15; 2.54%; $p < 0.05$).

Seventy-seven patients (13%) in the non-EEG group and 53 (17%) in the EEG group had previously had complete strokes that were followed by good functional recovery ($p > 0.05$). The incidence of transient ischemic attacks was 45.8% (n = 271) in the non-EEG group and 50.8% (n = 158) in the EEG group ($p > 0.05$). Asymptomatic high-grade (≥75% diameter reduction) carotid stenosis was present in 185 patients (31.3%) in the non-EEG group and 84 (27%) in the EEG group.

Preoperative angiographic examination was performed for each of the 902 procedures. Severe stenosis (≥75% diameter reduction) of the ipsilateral internal carotid artery was demonstrated in 564 angiograms (95.4%) in the non-EEG group and 270 (86.8%) in the EEG group ($p < 0.05$). The contralateral internal carotid artery was stenosed (≥50% diameter reduction) in 175 cases (29.6%) and occluded in 27 cases (4.56%) in the non-EEG group. In 90 cases (28.9%) in the EEG group the contralateral artery had severe stenosis, and in 26 cases (8.36%) the artery was occluded ($p > 0.05$).

EEG results. There were 40 cases (12.8%) of acute EEG changes. In 30 of those cases (75%), the acute changes developed within 1 minute (<30 seconds in 27 of the 30) after carotid cross-clamping. The remaining changes developed between 1 and 9 minutes after cross-clamping. In 28 of the 40 cases (70%), the amplitude of the faster frequencies significantly decreased. The appearance or augmentation of delta activity was the only change in three procedures (7.5%). In nine procedures (22.5%), the amplitude of the faster frequencies was reduced and the delta activity was increased.

In 31 instances (77.5%), the EEG change was confined to the hemisphere ipsilateral to the clamped internal carotid artery, whereas in nine procedures (22.5%) the changes were bilateral. Of the nine patients with bilateral changes, changes were symmetrical in six and more pronounced in the hemisphere ipsilateral to the clamped carotid artery in three. Notably, seven out of nine patients who had bilateral EEG changes had had significant contralateral carotid artery disease. One additional patient who had bilateral changes had a significant hypotensive episode during the carotid clamping.

In five patients (12.5%) the EEG changes were clearly correlated with an intraoperative episode of hypotension, and after the blood pressure was restored the EEG returned to normal. Shunts were not placed in these patients. In 35 patients (87.5%) an intraluminal carotid shunt was promptly inserted. In 33 of those patients the EEG was restored to baseline, in one patient there was a significant improvement, and in one the EEG changes persisted.

The association between preoperative clinical variables and development of EEG changes during carotid endarterectomy in the EEG group is shown in Table II. Notably, none of these clinical variables proved to be significant in predicting acute EEG changes during carotid cross-clamping.

Death. Two patients (0.33%) in the non-EEG group and two (0.64%) in the EEG group died in the postoperative period ($p > 0.05$). Of the two patients in the EEG group, one had fatal complications that were related to a stroke in the territory of the endarterectomized carotid artery. The second patient died from a postoperative myocardial infarction. Neither of the deaths in the non-EEG group was attributable to complications related to postoperative strokes. One death was attributed to aspiration pneumonia that progressed into severe adult respiratory distress syndrome. A cardiac event was the cause of death in the second patient.

Perioperative complications. Postoperative hospital strokes occurred in one patient (0.32%) in the EEG group and in 13 patients (2.19%) in the non-EEG group. The difference was statistically significant ($p < 0.05$). Notably, the EEG predicted the one intraoperative stroke in the EEG group. In that patient, significant EEG changes developed before carotid cross-clamping and did not resolve after placement of an intraluminal shunt. Upon opening the carotid bifurcation, we identified a severely ulcerated carotid plaque that contained evidence of a recent hemorrhage. After the operation, the patient was found to have had a massive ipsilateral stroke. A carotid arteriogram revealed a patent reconstruction. The stroke apparently resulted from the embolization of loose atherosclerotic debris and clot from the ulcerated plaque during the dissection of the carotid artery.

In the non-EEG group, all 13 strokes were ipsilateral to the operated carotid artery. Before surgery, the majority of those patients (n = 12) had a symptomatic, critical stenosis (≥75% diameter reduction) of the ipsilateral internal carotid artery. Eleven strokes were discovered immediately or in the first few hours after the patients awoke from general anesthesia. Seven of these 11 strokes were major and resulted in significant functional impairment. The other four strokes were minor and resulted in minimal or no functional impairment on discharge. In all patients who had major strokes, the reconstructions were found to be patent by angiographic examination. All patients who had minor strokes underwent serial computed tomographic brain scans, the results of which were negative. The pathogenesis of these strokes may be attributed to either regional hypoperfusion during the carotid clamping or to microembolization. Two strokes were identified within 36 hours after the completion of the procedure. In these patients, the symptoms of a recent cerebrovascular accident grew worse after the operation. Arteriography was not obtained in these cases, and postoperative computed tomographic scans failed to identify any evidence of intracerebral hemorrhage or worsening of the preoperative radiologic appearance.

The univariate association between significant variables and postoperative neurologic morbidity (major and minor cerebrovascular accidents) is shown in Table III. The only variable associated with

Table II. Univariate association between preoperative variables and development of intraoperative EEG changes in EEG group

Variable	*EEG changes (n = 40)*
Age	
>62 yr	35/263 (13.3%)
<62 yr	5/48 (10.41%)
Sex	
Male	22/192 (11.45%)
Female	18/119 (15.12%)
Hypertension	
Yes	26/175 (14.85%)
No	14/136 (10.29%)
Diabetes	
Yes	2/43 (4.65%)
No	38/268 (14.17%)
Smoking	
Yes	28/218 (12.8%)
No	12/93 (12.9%)
CAD	
Yes	12/106 (11.32%)
No	18/205 (8.78%)
History of CVA	
Yes	6/53 (11.32%)
No	34/258 (13.17%)
History of TIA	
Yes	21/158 (13.29%)
No	19/153 (12.41%)
Asymptomatic	
Yes	11/84 (13.09%)
No	29/227 (12.77%)
Previous CEN	
Yes	12/80 (15%)
No	28/231 (12.12%)
Repeat CEN	
Yes	0/17 (0%)
No	40/294 (13.6%)
Ipsilateral stenosis ≥75%	
Yes	32/270 (11.85%)
No	8/241 (3.31%)
Contralateral stenosis ≥50%	
Yes	10/90 (11.11%)
No	30/221 (13.57%)
Contralateral occlusion	
Yes	2/26 (7.69%)
No	38/285 (13.33%)

CAD, Coronary artery disease; *CVA*, cerebrovascular accident; *TIA*, transient ischemic attack; *CEN*, carotid endarterectomy.

Table III. Univariate association between significant variables and postoperative hospital neurologic morbidity in non-EEG group, EEG group, and total group

	Non-EEG group	*EEG group*	*Total*
Sex			
Male	6/348 (1.72%)	0/192 (0%)	6/540 (1.11%)
Female	7/243 (2.8%)	1/119 (0.84%)	8/362 (2.21%)
Age			
>62 yr	8/423 (1.89%)	1/263 (0.38%)	9/686 (1.31%)
<62 yr	5/168 (2.98%)	0/48 (0%)	5/216 (2.31%)
Smoking			
Yes	9/408 (2.21%)	1/218 (0.46%)	10/626 (1.6%)
No	4/183 (2.19%)	0/293 (0%)	4/276 (1.45%)
Hypertension			
Yes	7/248 (2.82%)	1/175 (0.57%)	8/423 (1.89%)
No	6/343 (1.75%)	0/136 (0%)	6/479 (1.25%)
CAD			
Yes	4/165 (2.42%)	1/106 (0.94%)	5/271 (1.84%)
No	9/426 (2.12%)	0/205 (0%)	9/631 (1.42%)
Diabetes			
Yes	2/66 (3.03%)	0/43 (0%)	2/109 (1.83%)
No	11/525 (2.10%)	1/268 (0.37%)	12/793 (1.51%)
History of CVA			
Yes	8/77 (10.3%)*	0/53 (0%)	8/130 (6.15%)*
No	5/514 (0.97%)	1/258 (0.39%)	6/772 (0.78%)
History of TIA			
Yes	3/271 (1.10%)	1/158 (0.63%)	4/429 (0.93%)
No	10/320 (3.12%)	0/153 (0%)	10/473 (2.11%)
Nonhemispheric symptoms			
Yes	1/58 (1.72%)	0/14 (0%)	1/72 (1.38%)
No	12/533 (2.25%)	1/297 (0.33%)	13/830 (1.56%)
Asymptomatic			
Yes	1/185 (0.54%)	0/84 (0%)	1/269 (0.37%)
No	12/406 (2.96%)	1/227 (0.44%)	13/633 (2.05%)
Repeat CEN			
Yes	4/15 (26.6%)†	0/17 (0%)	4/32 (12.5%)
No	9/576 (1.56%)	1/294 (0.34%)	10/870 (1.14%)
Previous CEN			
Yes	7/137 (5.1%)	0/80 (0%)	7/217 (3.22%)
No	6/454 (1.32%)	1/231 (0.43%)	7/685 (1.02%)
Contralateral stenosis ≥50%			
Yes	4/175 (2.29%)	0/90 (0%)	4/265 (1.51%)
No	9/416 (2.16%)	1/221 (0.47%)	10/637 (1.57%)
Contralateral occlusion			
Yes	4/27 (14.8%)	1/26 (3.84%)	5/53 (9.43%)
No	9/564 (1.59%)	0/299 (0%)	9/849 (1.06%)
EEG monitoring			
Yes	N/A	N/A	1/311 (0.32%)
No	N/A	N/A	13/591 (2.20%)†
EEG changes			
Yes	N/A	1/40 (2.5%)†	N/A
No	N/A	0/271 (0%)	N/A
Postoperative MI			
Yes	3/3 (100%)*	0/2 (0%)	3/5 (60%)
No	10/588 (1.7%)	1/309 (0.32%)	11/89 (1.23%)
Postoperative RF			
Yes	5/20 (25%)	0/0 (0%)	5/20 (25%)
No	8/571 (1.40%)	1/311 (0.32%)	9/882 (1.02%)

CAD, Coronary artery disease; *CVA,* cerebrovascular accident; *TIA,* transient ischemic attack; *CEN,* carotid endarterectomy; *MI,* myocardial infarciton; *RF,* renal failure.
*$p < 0.001$.
†$p < 0.05$.

increased postoperative neurologic morbidity in the whole group (n = 902) was a history of cerebrovascular accident ($p = 0.0001$). However, continuous intraoperative EEG monitoring resulted in a significant decrease in the incidence of postoperative stroke ($p = 0.035$). A history of cerebrovascular accident ($p = 0.0001$), redo carotid endarterectomy ($p = 0.0012$), and postoperative myocardial infarction

($p = 0.001$) were predictors of neurologic morbidity in the non-EGG group. The only variable that was marginally associated with postoperative stroke in the EEG group was the presence of intraoperative EEG changes ($p = 0.042$). Because of the low perioperative neurologic morbidity rate in the latter group of patients, the results from the univariate model have to be interpreted with caution.

DISCUSSION

Prospective multicenter trials have established the value of carotid endarterectomy to prevent stroke in appropriately selected patients.[1-5] However, an overall 2% to 6% risk of perioperative death or stroke accompanies the procedure. Because the carotid artery must be cross-clamped before the arteriotomy and plaque resection, a number of complications may arise. Specifically, in patients who are not protected by adequate collateralization, cross-clamping can lead to a transient loss of blood supply to the brain. Because brain tissue has little tolerance for ischemia, significant damage may occur. An estimated 10% to 30% percent of patients who undergo unprotected carotid endarterectomy are likely to develop brain ischemia.[18-20] To decrease the chances of brain ischemia, some surgeons have routinely used an intraluminal bypass shunt in all carotid endarterectomy procedures. However, several authors have demonstrated that the incidence of perioperative neurologic morbidity in patients who did not receive shunts was equal to the incidence of stroke in patients in whom shunts were used routinely.[15] This obviously reflects the presence of a number of shunt-related complications that cancel the benefits of the protection offered by the use of a shunt.

Theoretically, selectively placing shunts would minimize procedural complications in patients who do not require shunts (the majority) and would protect the high-risk patients against cerebral ischemia. In the late 1970s, Sundt et al.[17,18,21] popularized continuous EEG monitoring as a way to evaluate cerebral function during carotid endarterectomy. The authors clearly demonstrated a correlation between regional cerebral ischemia and clamp-associated EEG changes: clamp-associated EEG changes occurred in all patients in whom regional cerebral blood flow measured less than 10 ml · 100 g^{-1} · min^{-1} but in none of the patients in whom cerebral blood flow measured greater than 25 ml · 100 g^{-1} · min^{-1}. Although EEG changes do not always imply that the unprotected patient will have a stroke, they definitely indicate a higher risk of stroke.[19,20]

It has been postulated that the incidence of stroke in most series in which no shunts were used was lower than that in those series in which shunts were selectively used on the basis of EEG monitoring.[20] Others, however, have found a significant decrease in the rate of death and stroke in EEG-monitored patients in whom shunts were used selectively.[15,16] Controversy remains about whether to use shunts selectively, to use no shunts, or to use shunts routinely. Admittedly, the stroke and death rates in most contemporary series are so small that prospective randomized trials would need a large subject pool to find valid correlations. Our series is unique because we used two specific approaches in the intraoperative management of carotid endarterectomy patients, and each approach had a sufficient number of patients to enable valid statistical analysis. In the first group, the carotid endarterectomy was completed without any means of cerebral monitoring and without shunting. The incidence of stroke in this group (non-EEG group) was similar to the incidence of stroke in other series in which unprotected carotid endarterectomy was performed.[6-9] In the second group, EEG activity was routinely monitored, and shunts were used selectively on the basis of established criteria. The two groups were not entirely similar in terms of preoperative risk factors. The proportion of patients who were older, had had hypertension, and underwent repeat carotid endarterectomy was significantly higher ($p < 0.05$) in the EEG group, placing them at a greater risk for postoperative neurologic morbidity. Despite that difference, EEG monitoring had a significant impact on the rate of perioperative neurologic complications. Whereas the incidence of stroke was 2.19% in the unprotected (non-EEG) group, it was 0.32% in the EEG group ($p < 0.05$).

Only 12% (n = 40) of the patients in this series had significant EEG changes, which confirms previous observations. In all but two of those patients the changes were reversed when a shunt was placed (n = 33) or when hypotension was controlled (n = 5). These changes were clearly related to a reduction in regional blood flow precipitated by the cross-clamping or the hypotension rather than to embolic events. In one patient the changes were partially reversed after placement of a shunt without adverse postoperative consequences. It is unclear whether an inadequate shunt or a small, clinically undetectable embolus was responsible for the partial resolution of the EEG changes. None of the above 39 patients had a neurologic deficit after surgery. Profound ipsilateral EEG changes developed in one patient before the carotid cross-clamping, which did not reverse after the placement of a shunt. He was the only patient who had a stroke in the EEG group. The intraoper-

ative findings suggested that the stoke resulted from an embolus. The association between fourteen preoperative clinical variables and the development of EEG changes during carotid endarterectomy in the EEG group was evaluated by univariate analysis. These variables included age, sex, hypertension, diabetes, smoking, coronary artery disease, history of cerebrovascular accident, history of transient ischemic attack, asymptomatic status, previous carotid endarterectomy, repeat carotid endarterectomy, ipsilateral significant carotid stenosis (≥75% diameter reduction), contralateral carotid stenosis (≥50% diameter reduction), and contralateral carotid occlusion (Table II). None of those variables was found to be a significant predictor of EEG changes during carotid clamping.

Universal shunting appears to be an appealing idea for the operative prevention of cerebral ischemia during carotid endarterectomy. The routine use of shunts, however, exposes all patients to the shunt-related complications, including an increased likelihood for embolization resulting from shunt insertion itself, as well as dissection and thrombosis of the internal carotid artery. A shunt may also limit visual exposure, thereby increasing the amount of time required to perform the endarterectomy. In several studies, shunted patients had a rate of neurologic morbidity that was equal to or greater than that in nonshunted patients.[15,22] In contrast, EEG monitoring during carotid endarterectomy enables 70% to 90% of patients to be excluded from undergoing elective shunting at all. Consequently, shunt-related complications can be reduced to an absolute minimum without compromising the safety of the operation.

An alternative approach is to perform all carotid endarterectomy procedures without intraoperative cerebral protection. Current data suggest that only 10% to 30% of patients who undergo carotid endarterectomy procedures have critical reduction of the cerebral blood flow after carotid clamping. Many of these patients apparently tolerate regional reductions in cerebral blood flow for limited periods, otherwise the neurologic morbidity rates in series with unshunted patients (including ours) would be much higher.[6-9] However, clamp-associated severe cerebral ischemia is associated with a considerable risk of postoperative stroke[20,22] that must be considered carefully, particularly when the operation is offered to asymptomatic patients. We believe that although unprotected carotid endarterectomy performed expeditiously by experienced surgeons can yield good results, the selective use of shunting on the basis of EEG changes further reduces the postoperative neurologic morbidity rate by protecting the high-risk patients.

Several other techniques have been used for the intraoperative monitoring of cerebral function during carotid endarterectomy procedures. These include carotid back pressure measurements,[23,24] transcranial Doppler monitoring,[22,25] and intraoperative neurologic assessment with regional anesthetic blocks.[26,27] These techniques may have some advantages, but each also has its own limitations.[22,25,28-30]

For the EEG to be useful as an indicator of cortical ischemia, anesthetic depth must be carefully controlled at levels that do not induce slow-wave activity. Appropriate supplementation of inhalation anesthetics with agents such as intravenous narcotics, antihypertensive medications, or both may be necessary to provide satisfactory anesthesia that does not interfere with the EEG interpretation of cortical ischemia. Also important for appropriate interpretation of the EEG are the maintenance of normocapnia, normovolemia, normal body temperature, and mean arterial pressure in the upper portion of the patient's preoperative range.[17,18]

The EEG reliably monitors cortical function, but it does not always provide useful information about significant alterations that are confined to the depths of sulci, the subcortical gray matter, or the deeper structures such as the internal capsule. These considerations have been raised by several investigators,[20,31,32] but they were not relevant in the current study because none of the patients who had unchanged EEGs had a stroke after surgery.

Disadvantages in the use of the EEG include difficulties in determining trends from the data and the need for trained personnel to interpret the raw data. In recent years, computer-assisted techniques have been introduced to address these problems. Two- and four-channel computerized EEG systems currently in use convert the on-line raw electroencephalogram into processed data that can be readily interpreted by a trained anesthesiologist.[33-36] The current results from studies using these computerized systems are conflicting, and additional research is necessary to establish their utility.[33,36,37]

The cost of EEG monitoring (technical and interpretation fee) is approximately $500 in our institution. This additional cost for the care of the patients who undergo carotid endarterectomy is relatively modest compared with the cost of caring for those patients who have postoperative strokes that could have been avoided by using EEG monitoring.

EEG monitoring can provide a rapid, highly accurate record of the effect on cerebral perfusion of various intraoperative maneuvers. By providing reliable evidence of ischemic changes during cross-clamping, EEG monitoring has resulted in the prompt identification of the patients who require a carotid shunt, and consequently it has minimized the potential of stroke in this high-risk population. EEG monitoring also enables surgeons to perform carotid endarterectomy procedures effectively and efficiently in the majority of the patients, who do not require shunts.

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ERRATUM

It has been brought to the Editors' attention that in the report entitled "Nicotine and cotinine stimulate secretion of basic fibroblast growth factor and affect expression of matrix metalloproteinases in cultured human smooth muscle cells" by C. S. Carty, P. D. Soloway, S. Kayastha, J. Bauer, B. Marsan, J. J. Ricotta, and M. Dryjski (J Vasc Surg 1996;24:927-35), an incorrect version of Fig. 5 was published. The correct Fig. 5 follows.

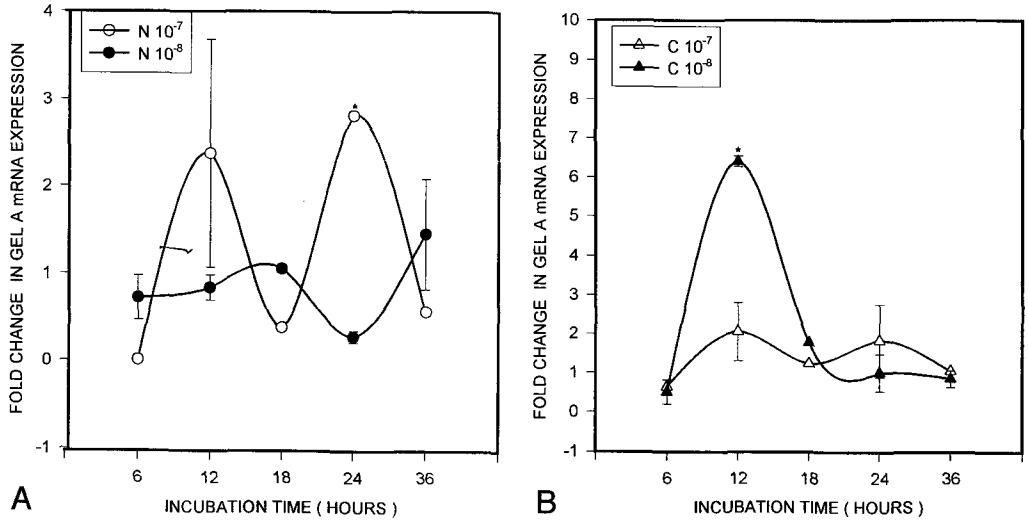

Fig. 5. Temporal induction of gelatinase A mRNA by nicotine **(A)** and cotinine **(B)**. Human SMCs were exposed to nicotine or cotinine in concentrations of 10^{-8} and 10^{-7} mol/L dissolved in albumin medium for times indicated (6 to 36 hours). Northern blot analysis (10 μg of total RNA per lane) was performed with gelatinase A cDNA probe. Blots were analyzed by autoradiography and quantitated by phosphor-imaging techniques. mRNA levels were normalized with TP1 probe and compared with levels in simultaneously analyzed control cells (exposed to albumin medium). Values represent mean of two to four experiments, with SEM indicated by *vertical bars.*