Predicting Cerebral Ischemia During Carotid Endarterectomy

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- We reviewed 86 consecutive patients undergoing elective carotid endarterectomy to determine whether preoperative clinical and angiographic data could be used to predict the risk of intraoperative cerebral ischemia during carotid occlusion. Electroencephalographic (EEG) monitoring with on-line Berg-Fourier transformation was carried out in all patients. A total of 32 patients (37.2%) underwent intraoperative shunting. Of these, 13 had no EEG changes but underwent shunting because of the surgeon’s preference, while 19 patients underwent shunting because of EEG changes consistent with cerebral ischemia. There was one permanent (1.2%) and one transient (1.2%) neurologic deficit. Angiographic findings, clinical histories, and intraoperative EEGs were retrospectively reviewed to determine which risk factors best predicted the occurrence of intraoperative cerebral ischemia. Stroke within six weeks increased the risk of intraoperative cerebral ischemia 20-fold. Intracranial disease and contralateral carotid stenosis increased the risk of ischemia 17-fold and 16-fold, respectively. Statistical summation of all risk factors yielded a probability equation for EEG change that accurately quantified preoperative risk. Prospective application of this probability equation may simplify operative decision making if EEG monitoring is not available.

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Neurologic deficits associated with carotid endarterectomy may be due to a number of factors, one of which is the temporary decrease in cerebral blood flow during carotid artery occlusion. To reverse cerebral ischemia, many surgeons employ temporary intraluminal shunts. While the complications of shunts are minimal in experienced hands, many surgeons find the device cumbersome and potentially injurious to the intima of the distal internal carotid. Recent discussion has centered not on whether the shunt is effective, but whether it should be used routinely or only selectively in those cases where cerebral blood flow is inadequate.

Our review was aimed at identifying those clinical and angiographic indicators most frequently associated with cerebral ischemia during carotid endarterectomy. We hoped to determine whether any factors or combination of factors could reliably predict the need for shunting. The degree of cerebral ischemia was assessed by continuous spectral analysis of the electroencephalogram (EEG), which has been shown to correlate well with significant reductions in cerebral blood flow.

PATIENTS AND METHODS

Eighty-six consecutive patients undergoing elective carotid endarterectomy were retrospectively reviewed. All operations were performed with the patients under general anesthesia and with continuous EEG monitoring using on-line Berg-Fourier analysis. Each patient’s history was reviewed for the following preoperative risk factors: diabetes mellitus, hypertension, coronary artery disease, smoking, and history of previous vascular procedures. Each case was also carefully assessed for prior neurologic events. The indications for endarterectomy were asymptomatic stenosis (17 patients, 20%), nonhemispheric symptoms (eight patients, 9%), transient ischemic attacks (TIAs) (35 patients, 41%), amaurosis fugax (11 patients, 13%), and distant or recent cerebral vascular accident (CVA) (15 patients, 17%).

All preoperative cerebral angiograms were independently reviewed in a blinded fashion and assessed for the following factors: (1) the presence of aortic arch disease; (2) percent stenosis of the common carotid, internal carotid, and external carotid arteries (ipsilateral and contralateral); (3) the presence of siphon stenosis and other intracerebral arterial lesions; and (4) the presence of vertebrobasilar stenosis.

Each risk factor was deemed “positive” if the angiogram disclosed any of the following characteristics: (1) greater than 75% diameter stenosis of the great vessels at their origin from the arch; (2) greater than 75% diameter stenosis of the contralateral common or internal carotid artery; (3) greater than 50% diameter stenosis of the ipsilateral carotid siphon or a major contralateral intracerebral vessel; or (4) bilateral vertebral artery stenosis of 50% or greater.

The EEG was obtained from five 9-mm gold cup electrodes affixed to the scalp with collodion. Interelectrode impedance was maintained at less than 10,000 Ω. Because the scalp distribution of EEG frequencies is only related to anatomical vascular perfusion in a general manner, we employed a single EEG channel from over the watershed area of perfusion between the anterior and posterior vascular supply of each hemisphere. The EEG signals from two channels were displayed on a monitor and digitized using a Berg-Fourier analyzer. The analyzer provided successive compressed spectral arrays (averaged over 30-s epochs) on a strip chart. Simultaneously, a histogram of the power spectrum of six preselected frequency bands was displayed on a separate monitor, updated every 3 s, and printed every three minutes. We averaged the power into three frequency bands: low (δ and θ, 0.25 to 6.0 Hz); middle (α, 6.0 to 10.5 Hz); and high (β and β, 10.5 to 16.0 Hz). The histograms were used to calculate changes in power for each of the frequency bands.

All carotid endarterectomies were performed using the same technique. The carotid bifurcation was not dissected until the common carotid and internal carotid arteries were occluded. The arteriotomy was extended above the plaque in the internal carotid artery. Care was used to ensure that all potential embolic material was removed. When necessary, the distal intima of the internal carotid artery was tacked down using 7-0 polypropylene sutures. The decision to use a shunt was made according to each surgeon’s clinical judgment. In general, shunts were used in the presence of a recent cerebral infarct (less than six weeks), or occluded con-
trilateral internal carotid artery, or if the EEG became abnormal after carotid occlusion. Our EEG criteria for cerebral ischemia were as follows: (1) diminution of high and middle frequencies by more than 50% of baseline values, or (2) the appearance of low-amplitude slow waves.

All 86 EEGs were retrospectively reviewed and the amplitudes of high frequency and middle frequency waves were measured during the course of the operation. The amplitudes immediately prior to carotid occlusions were compared with the amplitudes measured two minutes after clamps were applied. Hence, EEG changes could be assessed in all patients, even if shunts were inserted. There were no significant changes in blood pressure, oxygenation, or level of anesthesia in any patient during this time.

All risk factors, operative indications, angiographic data, and EEG changes were analyzed as independent variables by a stepwise logistic regression model utilizing the following equation:

\[ P(\text{EEG}) = \frac{\exp(B_0 + \sum B_i X_i)}{1 - \exp(B_0 + \sum B_i X_i)} \]

where \( P(\text{EEG}) \) is the probability of an EEG change, \( B_0 \) is a constant, \( B_i \) is the coefficient of each variable, and \( X_i \) is the independent variable.

This statistical method assigns a regression coefficient and standard error for each of the variables; these data are then used to calculate the relative risk of intraoperative cerebral ischemia for each independent risk factor.

**RESULTS**

**Surgical Aspects**

Fifty-four (63%) of the 86 endarterectomies had normal intraoperative EEGs and were performed without using an intraluminal shunt. Thirty-two (37%) were performed using an intraluminal shunt. In 13 patients (15%), no EEG changes were observed in the first two minutes after carotid occlusion but shunts were placed for clinical indications; the indication for shunting in ten of these patients was contralateral occlusion or intracranial disease while shunts were used in three patients because of a recent stroke. Nineteen patients (22%) underwent shunting procedures because they developed EEG changes consistent with cerebral ischemia after carotid clamping. In all 19 patients, the EEG returned toward normal within two minutes of shunt placement.

There was one permanent neurological deficit (1.2%) and one transient neurological deficit (1.2%) in the entire patient population. The single postoperative death was due to myocardial infarction (1.2%). Wound complications and transient cranial nerve injuries were documented in 4.7% of patients.

A permanent neurological deficit (hemiparesis) occurred in one patient without a shunt who underwent endarterec-

tomy for an ipsilateral hemispheric TIA. No intraoperative EEG changes occurred during carotid clamping. Postoperative noninvasive evaluation confirmed patency of the carotid artery that had been operated on. It is likely that this neurologic deficit was due to an intraoperative embolic event. One patient undergoing endarterectomy for non-hemispheric symptoms suffered a postoperative hemispheric TIA that resolved. On carotid clamping, there was global power reduction in the EEG. Because of the small caliber of the distal internal carotid artery, shunt placement was not possible and the EEG amplitude remained decreased throughout the endarterectomy. The patient awoke with contralateral hemiparesis that rapidly resolved. A computed tomographic scan disclosed no infarction and this deficit seems to be related to the decreased perfusion during carotid occlusion.

**Associated Diseases and Angiographic Features**

For further analysis, patients were divided into two groups: group I (n = 67, those with no EEG change) and group II (n = 19, those with EEG change). Forty-nine percent of the population was male and 51% was female (mean age, 66.9 years), with no difference in sex distribution or age between two groups. Significant differences were noted in associated medical problems (Table 1). The proportion of patients with shunts and with diabetes was 63%, compared with 94% for the nonischemic group (P < .05). Hypertension and smoking were also significantly more prevalent in group II than group I. Group II included more patients with a history of a recent stroke (21%) than group I (6%), while TIA was a more frequent indication for surgery in group I (group I, 45% vs group II, 26%) (Table 2). Group II had a much higher incidence of contralateral carotid stenosis and intracerebral occlusive disease (Table 3).
Electroencephalogram. Note marked decrease in all frequency amplitudes with left carotid clamping (top), followed by brisk return to near-normal amplitudes following shunt insertion. Right cerebral hemisphere electroencephalogram (bottom) was also transiently depressed with left carotid occlusion, suggesting interruption of "crossover" blood flow in this patient with right intracranial carotid stenosis.

The compressed spectral array of a typical recording is seen in the Figure. The percentage amplitude changes in the three frequency ranges were calculated from these recordings for all group II patients, as shown in the following tabulation.

<table>
<thead>
<tr>
<th>Wave Frequency</th>
<th>% Change, Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>−75 ± 27</td>
</tr>
<tr>
<td>Middle</td>
<td>−65 ± 26</td>
</tr>
<tr>
<td>Low</td>
<td>−44 ± 63</td>
</tr>
</tbody>
</table>

As expected, the highest-frequency waveforms (α and β) diminished the most as these frequencies are the most sensitive to cerebral ischemia; in ischemic patients we observed a mean decrease of 75% from the preclamp baseline.

In nine of the 19 patients with shunts there was a greater than 50% attenuation of all frequency bands (global ischemia). Eight of nine patients had shunts emplaced and responded with an increase in EEG amplitude within three minutes of restoring flow through the shunt. None of these eight patients suffered a perioperative stroke. As noted above, the single patient without a shunt in this subgroup experienced a transient deficit.

**Combined Assessment of Risk Factors**

Table 4 lists the risk factors, relative risks, and associated regression coefficients for EEG change; the higher the regression coefficient the higher the probability of cerebral ischemia. The occurrence of a recent stroke (within six weeks) had the highest relative risk for the development of cerebral ischemia during carotid clamping. Stroke within six weeks of carotid endarterectomy increased a patient's risk of intraoperative cerebral ischemia 20-fold over those patients without this specific risk factor. Intracranial disease and contralateral stenosis were also associated with increased risk of EEG change (17-fold and 16-fold, respectively).

To test the practical application of this statistical method in our clinical practice, we retrospectively calculated the $P(\text{EEG} \Delta)$ that would predict no false-negatives. That is, at what probability of EEG change would shunting have ensured that all 19 of our patients with cerebral ischemia

would have been protected? A practice of placing shunts in all patients with a calculated $P(\text{EEG} \Delta) > 0.025$ would have satisfied this requirement. Using this criterion, a total of 41 patients would have undergone shunt placement.

**COMMENT**

A number of intraoperative techniques have been used to detect cerebral ischemia during carotid surgery. The three most widely used involve monitoring of the neurologic status of conscious patients, postocclusion changes in continuous EEG recordings, or carotid stump pressure after clamping. The correlation between EEG changes and changes in cerebral blood flow has been well documented by Sundt et al. Their work has shown that a decrease in cerebral blood flow to critical limits results in EEG abnormalities that can be reversed by the placement of an intraluminal shunt. This physiologic principle is the reason most groups perform shunt procedures routinely or selectively.

The major limitation of all intraoperative methods of assessing cerebral perfusion is that they are prone to subjective interpretive errors, may be altered by anesthetic effects on systemic blood pressure, and require the availability of sophisticated equipment and personnel. We used intraoperative evidence of decreased cerebral perfusion (EEG changes) to determine retrospectively whether any preoperative measure or combination of factors could reliably predict the need for intraoperative shunting. Statistical methods allowed us to quantitate and rank the importance of each of these factors independently and in combination.

Recent, completed stroke (within six weeks) implied the highest risk of developing intraoperative cerebral ischemia. Since all 86 endarterectomies were performed at least four weeks after a stroke, the data do not allow us to draw conclusions as to whether earlier operations would increase the risk further. Our data would support the practice of those surgeons who routinely perform shunt procedures in all patients with recent, completed strokes.

Intracranial stenoses and occlusions were also found to be important prognostic indicators. The presence of intracranial disease, defined as either a severe siphon stenosis (50% lumen reduction or greater) or major intracranial vascular occlusion, increased the risk of cerebral ischemia during carotid clamping 17-fold. Contralateral internal carotid stenosis of 75% or greater increased the need of a shunt by a factor of 16 compared with those patients without contralateral stenosis.

The probability equation can be used in a prospective
manner since all required information is available preoperatively. This allows an accurate assessment of the risk of cerebral ischemia before the surgical procedure. We found that shunt placement in all patients with a probability for EEG change of $P(EEG) > 0.025$ would have ensured that all of our patients with cerebral ischemia would have been protected. Although such a policy would have required shunt placement in an additional 22 patients, we believe that this relatively small increase in shunt placement is warranted if EEG monitoring is not available.

Intraoperative monitoring techniques such as continuous EEG with spectral analysis can identify those patients with critical decreases in cerebral perfusion during carotid occlusion. However, surgeons who do not have EEG available or desire to lower costs may reliably use clinical criteria to select patients preoperatively for shunt procedures. The data presented quantitate the relative risk of historical and angiographic indicators and support selective shunting in patients with recent stroke, contralateral carotid stenosis, and intracranial occlusions.

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References


Discussion

**Victor M. Bernhard, MD, Tucson:** These authors have demonstrated clearly that they are good surgeons and, therefore, that their patients have too low a morbidity-mortality to really test their thesis. Nevertheless, there are substantiating data. There is some question of whether the EEG is reliable with the patient under general anesthesia, a point that has been noted in other studies that have attempted to determine whether or not all people who have EEG changes would necessarily have a stroke and vice versa.

Furthermore, some investigators have noted that EEG changes may appear somewhat down the line, though not within the first 2, 3, or 4 minutes, and at this point the patient is considered to be on the cusp between adequate and inadequate perfusion. Did this occur in the present study?

The authors confirm that which we have all noted and which Drs Baker and Whittemore have both described nicely: patients with contralateral occlusion and a previous stroke have a higher likelihood of intraoperative neurologic event presumably due to perfusion deficit. The questions we always have in any patient who has a deficit upon recovery from anesthesia is whether it was due to inadequate perfusion or embolism. We presume, as these authors have noted, that the cause was intraoperative embolism if the EEG was normal and the carotid plaque was friable, irregular, or ulcerated. In this regard, some cerebrovascular surgeons have argued that the presence of intracranial disease, especially at the siphon, may not correlate with postoperative deficits. Since the authors indicated that this is a significant issue, I would like to have them comment further on this factor.

I, too, like to perform carotid surgery without a shunt, and it is comforting to have an EEG tracing to increase my feeling of security that all is well when the procedure is being done with the patient under general anesthesia. However, if you want to be really safe, using the present technique and regression analysis without an EEG, almost one half of the patients in this series required a shunt. Therefore, I wonder how much these patients have really gained.

**Fletcher A. Miller, MD, St Paul:** In these days of high costs and close attention to budgets, what is the cost factor of this piece of equipment when it is prorated over the number of patients that are operated on? We all know that there are many people today, both with or without shunts, who have had acceptable results. Less than 2% of patients have these postoperative problems, and in those 2% are the problems that the authors have stated would not be detected by the EEG, eg, embolus and postoperative thrombosis after the EEG is removed. For those of us who are not in the research environment, is this a modality that we can afford to use?

**Dr Gewertz:** Anesthetic effects on the EEG are certainly a concern. We have been able to note very precisely when new bits of anesthesia are introduced into the system, and we try to minimize such alterations in anesthesia at the time of carotid clamping. However, I believe that Dr Bernhard’s point is very well taken. Both Dr Zarins and I have been concerned about that unusual patient who starts off with a well-preserved EEG and then, during the half hour or 40 minutes required for a complicated endarterectomy, demonstrates decreases in high and middle frequencies. I think that this phenomenon is a real concern and we will continue to look at it to better understand its significance.

In our opinion, it is not so much the siphon disease of the ipsilateral internal carotid that really matters, but the contralateral circulation. It seems that the intracranial A-1 segment that crosses over from the other carotid artery is the best indicator of potential collateral circulation. With all of our patients, we try to get detailed preoperative intracranial views to make an accurate assessment of the A-1 segment.

Dr Miller, I believe your comments were well justified. The cost of using EEG in our environment is very low. Neurologists work for about 20 bucks an hour as best as I can tell, but out in the community the availability of such expertise and sophisticated equipment may be less than at the university. I think that there is no doubt, as Dr Baker has emphasized many times when he has tackled this question, that the precision of the surgery is what determines the outcome much more than the gadgetry involved.