Correlation of Continuous Electroencephalograms With Cerebral Blood Flow Measurements During Carotid Endarterectomy

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Abstract: Correlation of Continuous Electroencephalograms With Cerebral Blood Flow Measurements During Carotid Endarterectomy

During an 11-month period, 81 endarterectomies under a carefully controlled level of general anesthesia were monitored with continuous electroencephalograms (EEG) and intermittent regional cerebral blood flow (CBF) measurements. There was a high correlation between the CBF (milliliter per 100 gm per minute) during carotid occlusion and alterations in the EEG: no EEG change was seen with the flow above 30 ml/100 gm brain per minute, major changes were not seen with a flow between 18 and 30 ml, and changes invariably occurred with a flow below 17 ml. The degree of EEG change reflected the severity of flow reduction but was always reversible with the placement of a shunt. The EEG at the termination of the surgery corresponded with the patient's neurological state in that all EEG tracings were normal or unchanged as compared to the preoperative tracing and no neurological worsening occurred in any patients studied. The EEG is a valuable monitoring technique that indicates when a shunt is required and informs the surgeon of the state of cerebral function not only during occlusion but also throughout the entire operative procedure.

Additional Key Words: cerebral autoregulation, halothane anesthesia, ischemic tolerance, Xenon-133, critical blood flow

This communication reviews the results of monitoring techniques during carotid endarterectomies that employ continuous electroencephalograms (EEG) and intermittent regional cerebral blood flow (CBF) measurements. Particular attention is directed to the EEGs and their correlations with CBF measurements during the surgery, the patient's course during anesthesia, and his immediate preoperative and postoperative neurological state. Conclusions regarding tolerance for ischemia of cerebral tissue and the causes of intraoperative complications will be presented and correlated with laboratory investigations. The interrelationship of the EEG and general anesthesia will be examined to some extent. The broader aspects of carotid artery surgery, including indications, technique, postoperative complications, and the relationship of CBF changes after endarterectomy to the degree of preoperative stenosis, have been previously reported in part but will be covered in detail in the future based on our total operative experience. Several excellent reports from other institutions have considered these previously.

Methods

PATIENT MATERIAL

All except emergency procedures of one surgeon's service during an 11-month period were monitored with these techniques. Each patient had a preoperative and a postoperative neurological examination, along with a retinal artery pressures (RAPs). Virtually all patients had a high-grade stenosis and were symptomatic when surgery was performed. Postoperative patency of the vessels was determined by RAPs or by angiography or by both. Before dismissal from the hospital, every patient underwent a neurological examination, and each had a follow-up examination after dismissal.

EEG TECHNIQUE

Forty-five minutes before the patient was taken to the operating room, two ear and 19 scalp electrodes were applied with collodion. The 10 to 20 international...
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System of electrode placement was employed. A 16-channel EEG recording was obtained with a Grass Model 6 machine. A baseline trace was obtained prior to induction. The EEG was then recorded continuously, beginning with induction and ending with extubation. Both routine (30 mm per second) and slow (15 mm per second) paper speeds were employed.

SURGICAL TECHNIQUE

The operative technique is essentially that described by Murphey and Miller.5 No trial occlusions were employed during the surgery, and manipulation of the carotid bifurcation was avoided. The common carotid artery was occluded with a vascular clamp, the external carotid artery with a surgical tourniquet, and the internal carotid artery with a Scoville clip. Use of a Scoville clip on the internal carotid artery allows high surgical dissection on that vessel, and it is an atraumatic means of occlusion. We employed a shunt fabricated by the Heyer-Schulte Corporation and produced in three sizes to match various-sized vessels. The distal end of the shunt was initially inserted into the internal carotid artery, and when back-bleeding through the shunt occurred, the proximal end was placed into the common carotid artery and sealed with a Rummel tourniquet. The distal end of the shunt was self-sealing, and no tourniquet was required on the internal carotid artery. The shunt was held in place by the tourniquet on the common carotid artery that sealed the shunt distal to its proximal bulbous dilatation. In many instances, removal of the plaque was completed in the internal carotid artery prior to the placement of a shunt. This was not always possible, and in instances when the shunt had to be placed immediately, this was done prior to endarterectomy. The arteriotomy was always carried distal to the furthest extension of the plaque so that, after removal of the plaque and closure of the arteriotomy, no flap of intima was present to serve as a source of emboli. The patient was given heparin prior to the arteriotomy, and the heparin was not neutralized. The arteriotomy was closed with a 6-0 arterial silk suture, but for a small vessel, a saphenous vein patch was incorporated in the closure to provide an increased diameter to the internal carotid artery at its origin from the common carotid artery.

ANESTHETIC TECHNIQUE

General anesthesia was used in all patients. Halothane was the agent of choice because it produces an increase in cerebral blood flow (decrease in cerebral vascular resistance) and a decrease in cerebral metabolic rate.*6,7 Premedication consisted of atropine with an opiate, diazepam, or both. In the operating room, a final preanesthetic evaluation a brief neurological examination, and a baseline electroencephalogram were followed by induction of anesthesia with thiopental and endotracheal intubation facilitated by intravenously administered succinylcholine. Anesthesia was maintained with halothane in a 50% nitrous oxide-50% oxygen mixture, and ventilation was controlled mechanically. Percutaneous cannulation of either a radial or dorsalis pedis artery permitted both continuous direct arterial pressure monitoring and intermittent sampling for arterial carbon dioxide tension (Paco2), oxygen tension (PaO2), and pH determinations. PaO2 was kept above 110 mm Hg. Care was taken to avoid hypotension throughout anesthesia, including the use of appropriate vasopressor therapy when indicated. Manipulation of the tissues overlying the carotid artery was minimized to avoid embolization from the intra-arterial plaque.

A semiclosed-circle absorber system was used for ventilation during both the induction and the emergence phases of anesthesia. A nonrebreathing circuit was used throughout surgery and cerebral blood flow (CBF) measurements in order to collect and exhaust to the outside atmosphere the expired gases that contained exhaled radioactive Xenon from the CBF determinations. Carbon dioxide was added to the inspired gas mixture to achieve a PCO2 between 35 and 46 mm Hg in most patients. If a patient normally had a higher PCO2 (for example, in chronic obstructive pulmonary disease), we endeavored to maintain a comparable PCO2 during surgery. (A brief discussion of the effects of CO2 on cerebral blood flow is contained elsewhere in this article.) A nondepolarizing muscle relaxant (usually gallamine) was given if needed to prevent patient attempts at spontaneous ventilation when the CO2 was added.

Heparin was given intravenously before the carotid artery was clamped. During occlusion, the arterial pressure was deliberately elevated 15% to 20%, except in patients whose hypertension (systolic pressure of 175 mm Hg and higher) made increases of this magnitude inadvisable. Some increase in arterial pressure usually accompanied the increase in PAO2. Additional pressure increases were achieved with the use of an intravenous phenylephrine drip (0.002%). After repair of the arteriotomy and completion of the CBF determinations, the use of the phenylephrine drip (if used) and CO2 was discontinued individually, and the semiclosed-circle absorber ventilating circuit was restored.

Continuous oscilloscopic display of the electrocardiogram was monitored from before induction until after emergence from anesthesia. Patients with carotid artery disease frequently have other cardiovascular problems. Ventricular extrasystoles in some patients occurred during anesthesia and surgery. If premature ventricular contractions (PVCs) were frequent, multifocal, or accompanied by a decrease in arterial pressure, the patient was given lidocaine intravenously, the halothane concentration was decreased, or a combination of the three was carried out.

At the conclusion of the procedure, the arterial needle was removed, the patient was awakened, any persistent neuromuscular blockade was reversed by the

*A nitrous oxide-narcotic-muscle relaxant technique was used in one patient (not in this series) in whom halothane was contraindicated.

†If further evaluation of arterial blood gases was desired the needle was left in place until an arterial sample was obtained in the recovery room and then removed.
use of prostigmine and atropine, the patient was extubated, and the neurological function was again evaluated. The patients received \( \text{O}_2 \) (40% unless higher concentrations indicated) in a mist via a close-fitting face mask in the recovery room and were evaluated by the anesthesiologist and surgeon before being transferred to the neurosurgical intensive care unit.

**MEASUREMENTS OF CBF**

Regional cerebral blood flow was determined from clearance curves created from the extracranial detection of intra-arterially injected Xenon-133. This radioactive indicator was injected through a no. 27 needle either into the common carotid artery with the external carotid artery occluded or into the internal carotid artery. Each injectate contained 300 to 400 \( \mu \text{Ci} \) of Xenon-133 in physiological saline and was 0.3 to 0.4 ml in volume. Customarily, three measurements were used: one prior to occlusion, one during occlusion, and one after restoration of flow. If a shunt was employed, a fourth and sometimes a fifth measurement were performed with the shunt in place. On occasion, more than one postoperative measurement was used. A single scintillation detector was used with a sodium iodide (NaI) crystal one and one-fourth inches in diameter and one-fourth inch thick. The crystal was recessed one inch behind a tapered lead collimator with an opening widening from seven-eighths of an inch to one and one-eighth inches at the surface of the crystal. The detector was placed adjacent and perpendicular to the scalp overlying the hand and face area of the motor strip.

Each clearance curve was analyzed by three techniques: initial slope, kinetic, and exponential analyses. Techniques used in this laboratory for analysis of these curves have been presented previously in detail and are based on methods of analysis relatively standard at this time for such measurements. The results of the initial slope analysis are available to the surgeon within minutes after the injection of radioactive Xenon-133. The values for CBF are calculated easily with a slide rule and those for flow rates greater than 20 ml to 30 ml/100 gm per minute are available to the surgeon within two to three minutes from the time of injection. Exponential and kinetic analyses require collection of data for at least eight or ten minutes after injection of the indicator and are computed separately after the surgical procedure. The flow values reported in this paper are determined from analysis of initial slope.

**Results**

**SURGICAL RESULTS**

No patient awoke from anesthesia with a neurological deficit that was not present before the induction of anesthesia, except for an occasional minimal weakness of the lower lip on the side of surgery related to traction on the seventh nerve at the superior end of the operative incision. Three patients (two immediately and one delayed) had low retinal artery pressures after operation. Each patient was reoperated on before a fixed neurological deficit could develop. A fresh thrombus was found in the vessel, a vein graft was placed, patency was restored with normalization of the RAPs, and the patient was dismissed from the hospital after a normal postoperative recovery. Two patients with preexisting contralateral infarcts developed seizures on the fifth to sixth day after operation, the infarcts being associated with paroxysmal lateralizing epileptiform discharges (PLEDs) on the EEG; although initially difficult to control, the seizures did not require long-term anticonvulsants. One patient died from a myocardial infarction after dismissal from the hospital.

**ANESTHETIC RESULTS**

There were no anesthetic deaths or untoward complications. The degree of transient hoarseness in some patients immediately after operation was as would be expected in patients undergoing neck surgery. There were no complications from the arterial cannulations. Cerebral blood flow measurements correlated well clinically with the \( \text{PaCO}_2 \) levels and arterial pressures, although these data were not analyzed statistically. Results of CBF recordings in those patients with EEG changes during occlusion with simultaneous measurements of systemic parameters are summarized in table 1.

**EEG RESULTS**

**Background EEG Pattern Under Anesthesia**

There were two basic patterns seen during anesthesia: a symmetric pattern and a grossly asymmetric pattern with focal changes.

The symmetric pattern was noted in all 60 cases in which the patients were free of significant preoperative neurological deficit and persistent focal findings in their waking EEG. The records were usually dominated by symmetric sustained rhythmic activity of 10 to 14 Hertz (Hz) that ranged between 25 and 100 \( \mu \text{V} \). Some records showed faster activity, of 15 to 24 Hz, but the activity was usually of lesser amplitude and persistence. In addition, with deeper levels of anesthesia, slower activity of 2 to 6 Hz, with amplitudes ranging between 50 and 150 \( \mu \text{V} \), became more apparent. All of these patterns tended to show a midline maximum in either the frontal or the central regions. Figure 1 shows a typical example of the symmetric rhythmic pattern.

Twenty-one patients had persistent focal findings and asymmetry of the background pattern throughout anesthesia. All 21 had a neurological deficit before surgery and a persistent focus in their waking EEG. The focal abnormalities were more prominent during the anesthetic state in some patients, whereas in others it was more prominent during the waking state. However, all of these patients had some persistent focal abnormality in both states. Figure 2 illustrates a typical EEG from this group. The patient had a focal neurological deficit, a persistent focus in the waking EEG, and a persistent focus in the EEG taken during anesthesia.
EEGs AND CBF MEASUREMENTS DURING SURGERY

TABLE 1
Patients With EEG Changes* During Carotid Occlusion

<table>
<thead>
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<th>Case no</th>
<th>Date</th>
<th>BP (mm Hg)</th>
<th>PaCO₂ (mm Hg)</th>
<th>PaO₂ (mm Hg)</th>
<th>pH</th>
<th>Occlusion CBF†</th>
<th>Time to place shunt (minute)</th>
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</table>

*Time from occlusion to EEG change varied from 12 seconds to three minutes; no patient had neurological complications during surgery.

†CBF determined by initial slope and reported to surgeon during occlusion.

This example shows the usual type of focal changes seen in this group—changes that consisted of reduction of normal background activity associated with variable amounts of irregular slowing.

Major Focal EEG Changes Occurring Three Minutes After Carotid Clamping

Seventeen cases had major focal EEG changes within three minutes after clamping of the carotid artery (table 1). In all 17 cases, a shunt was used.

![Typical symmetric EEG pattern during general anesthesia using both (A) slow (15 mm per second) and (B) regular (30 mm per second) paper speeds.](http://stroke.ahajournals.org/)

*Time from occlusion to EEG change varied from 12 seconds to three minutes; no patient had neurological complications during surgery.†CBF determined by initial slope and reported to surgeon during occlusion.

![Typical symmetric EEG pattern during general anesthesia using both (A) slow (15 mm per second) and (B) regular (30 mm per second) paper speeds.](http://stroke.ahajournals.org/)

Stroke, Vol. 4, July-August 1973
FIGURE 2
EEGs in a patient with preoperative neurological deficit (right hemiparesis). Waking EEG (A) shows left-sided focal abnormality which also is obvious during anesthesia (B).

and the focal changes resolved within two to seven minutes after shunting; no patient had evidence of a new neurological deficit in the immediate postoperative period. The changes were stereotyped and generally consisted of replacement of the faster background frequencies, first by higher amplitude theta components and then by lower amplitude irregular delta components. In patients with the more extreme changes, the residual EEG activity during clamping was ultimately reduced to low-voltage irregular delta activity, with total absence of faster background components. Figure 3 illustrates the typical sequence of changes seen in this group after clamping of the carotid artery.

FIGURE 3
A. Symmetric EEG pattern prior to carotid clamping. B. Major focal EEG changes began 17 seconds after right carotid clamping when blood flow was reduced to 12 ml/100 gm per minute. C. After internal shunting, EEG returned to normal symmetric pattern.
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Eleven of the 17 cases with major EEG changes, or all patients with a blood flow below 17 ml/100 gm per minute, developed changes that were extreme and became apparent within 20 seconds after clamping. Six cases had major focal change with carotid clamping and a blood flow between 17 ml and 18 ml/100 gm per minute. In these patients, the EEG change was somewhat delayed, usually developing only after 40 to 100 seconds from the time of clamping. The degree of EEG change was in general not as severe as that seen when the flow decreased below 17 ml/100 gm per minute. This type of change with occlusion occurred with equal frequency in patients with a preexisting EEG abnormality (4 of 21 cases, 19%) and those with symmetric EEG patterns prior to clamping (13 of 60 cases, 21%). There were two patients with a blood flow between 17 ml and 18 ml/100 gm per minute during carotid clamping who failed to show any EEG change during occlusion.

Four of the 81 cases showed minor asymmetry, with only reduction in amplitude and background components on the side of surgery occurring within the first three minutes after carotid occlusion. However, in these four patients, the changes returned to normal either spontaneously (three patients) or after use of a shunt (one patient). The blood flow in these four patients ranged between 18 ml and 23 ml/100 gm per minute.

Major Focal EEG Changes Not Immediately Associated With Carotid Clamping

Eight patients had major transient slowing not immediately associated with or due to carotid clamping. In four of these, the slowing occurred contralateral to the side of surgery. Three of the four patients had clear-cut evidence of contralateral carotid disease. None showed evidence of a new neurological deficit in the immediate postoperative period.

Four patients in this group had EEG changes ipsilateral to the side of surgery. These changes were not immediately associated with carotid clamping or reduction of blood flow below the critical level of 17 ml to 18 ml/100 gm per minute. In two of these patients, the changes occurred before the beginning or before the completion of the endarterectomy. No additional EEG changes were noted in these patients after closure of the arteriotomy. These two patients were free of neurological deficit in the immediate postoperative period and developed no delayed complications. In two patients, the transient changes developed after closure of the arteriotomy. In one of these, the EEG change consisted of a brief focal seizure discharge in the right central area. This patient was free of neurological complications in the first several days after operation but, on the fifth day, developed focal seizure status associated with paroxysmal lateralizing epileptiform discharges (PLEDs) ipsilateral to the side of the surgery. The patient later fully recovered. (One additional patient in this study group developed postoperative focal seizure status associated with PLEDs, but this patient had no EEG changes at surgery.) The second patient who had ipsilateral change after restoration of flow demonstrated intermittent focal slowing. Within two hours after this patient awakened from anesthesia, a diagnosis of postoperative carotid occlusion was made because of a marked unilateral reduction in retinal arterial pressure. The patient was returned to surgery, and the carotid artery was reopened and flow was restored without neurological complications. Two other patients in this series developed occlusion of the operated carotid artery after the termination of surgery. Neither of these patients, however, had EEG changes during surgery. In both of these, a very small internal carotid artery was reopened and a saphenous vein patch inserted. These patients were returned to surgery before a fixed deficit developed, and no neurological complications resulted.

Discussion

ANESTHETIC CONSIDERATIONS

The relative merits of local and general anesthesia for carotid endarterectomy have been reported.2-3, 5 Local anesthesia permits evaluation of the patient's neurological condition during surgery, but it is more uncomfortable for the patient and restricts surgical exposure. General endotracheal anesthesia provides good airway control, higher PaO2 levels, freedom from pain and anxiety for the patient, and better surgical exposure, and it permits regulation of PaCO2 levels when indicated. In addition, halothane increases cerebral blood flow (decrease in cerebral vascular resistance) and decreases cerebral metabolic rate.6, 7 Direct arterial pressure monitoring and deliberate pharmacological elevation of the pressure also are facilitated by general anesthesia.

Previously evaluation of the patient's neurological condition during surgery has been virtually impossible during general anesthesia. Our experience indicates that the EEG is an accurate indicator of cortical ischemia during general anesthesia, provided that the depth of anesthesia does not change. As the depth of anesthesia increases, the EEG becomes dominated by waves of 2 to 6 Hz, with loss of the faster components. These generalized EEG changes make recognition of focal abnormalities much more difficult. Changes in the depth of anesthesia might have contributed to previous reports that the EEG is not a sensitive indicator of cortical ischemia during anesthesia.8-12

In areas of focal cerebral ischemia, such as occur during carotid or middle cerebral artery
occlusion, cerebral autoregulation of blood flow is lost and flow becomes dependent on both perfusion pressure and blood volume. Therefore, arterial pressure should not be permitted to fall below the patient’s normal level during anesthesia and should be elevated during carotid occlusion. In addition, blood volume should be maintained by the adequate replacement of blood loss.

SURGICAL COMPLICATIONS

The most catastrophic complication that can occur from carotid endarterectomy is embolization to a major branch of the middle cerebral artery. There were no recognized major embolizations in our patients during or after the operative procedure. We attribute this to the information given to the surgeon by these monitoring techniques—information that allows him to do a precise and unhurried endarterectomy, free of uncertainty regarding the status of cerebral function. The degree of flow reduction and the EEG change indicate when and how rapidly a shunt must be placed. In the three patients with postoperative occlusion, the artery was promptly reopened (4, 8 and 18 hours) and good flow was established before a fixed neurological deficit developed. These three patients were identified before the development of severe symptoms from occlusion largely because of information provided from the CBF measurements (low postocclusion flow) and the EEG (one patient—transient ipsilateral changes) at surgery. These three patients could have had neurological complications if prompt measures had not been instituted in the immediate postoperative period.

$\text{PaCO}_2$ TENSIONS

$\text{PaCO}_2$ levels should be maintained at relatively normal levels for the patient during this procedure. Although we do not believe that the “steal” phenomenon is an important consideration in carotid artery surgery, it is nevertheless a theoretic argument against the use of hypercapnia. Our laboratory studies have indicated no appreciable protection from elevating arterial CO$_2$ levels in ischemia, although regional flow may be increased. An increased tissue acidosis in regions of ischemia from elevated $\text{PaCO}_2$ may augment the demand for CBF. Other considerations such as cardiac arrhythmias under general anesthesia with a high $\text{PaCO}_2$ are important, because coexisting cardiac disease is commonly present in this group of high-risk patients.

Although the protection, or lack of it, from an elevated $\text{PaCO}_2$ is at present unresolved, laboratory investigations of squirrel monkeys have definitely indicated a detrimental effect from a lowered $\text{PaCO}_2$. In these studies, levels of adenosine triphosphate (ATP) have been significantly lower and lactic acid determinations have been significantly higher in regions of focal ischemia when the monkeys were subjected to hypocapnia during middle cerebral artery occlusion.

The $\text{PaCO}_2$ level should be maintained near whatever is normal for the individual patient undergoing the operative procedure. In patients with chronic obstructive pulmonary disease who have a preoperative elevated $\text{PaCO}_2$, we have found that baseline flow measurements at normocapnia indicate a relative hypocapnia for the patient by reflecting blood flow measurements commonly seen when $\text{PaCO}_2$ levels are in the low 20s for normal patients. In these patients, raising the $\text{PaCO}_2$ to their normal levels results in a relatively normal baseline flow study prior to endarterectomy.

VALUE OF EEG DURING ANESTHESIA

The literature presents a confusing picture concerning the value of the EEG for detection of focal ischemic abnormalities induced by carotid occlusion during the anesthetic state. This may be the result, in part, of the sensitivity of the EEG to the depth of anesthesia. Most authors contend that the EEG is a sensitive indicator of cortical dysfunction due to acute cerebral ischemia in a conscious patient. In fact, in conscious patients with cerebral ischemia induced by carotid clamping, EEG changes often precede the clinical signs. However, the same authors, as well as others, believe that, with the patient under general anesthesia, the EEG is not a reliable indicator of cerebral ischemia. We agree with those investigators who believe that the EEG during anesthesia is useful for the diagnosis of intraoperative cerebral ischemia and for establishing the prognosis in the immediate postoperative period.

In our experience, EEG abnormalities developed in all patients in whom occlusive CBF decreased below 17 ml/100 gm per minute, and a preexisting focal abnormality on the EEG did not interfere with the detection of these ischemic changes. We caution that anesthesia must be maintained at a level which permits a stable baseline EEG and which does not mask minor alterations. The $\text{PaCO}_2$ also must be carefully controlled to avoid EEG fluctuations that have been seen with changes in the arterial tension of this gas.

In addition to identifying patients who develop cerebral ischemia on carotid clamping, some studies have suggested that the intraoperative EEG can predict whether a patient will or will not have a new neurological deficit during the immediate postoperative period. These studies have suggested that patients who do not develop a new persistent focal EEG change during anesthesia will not have evidence of a new neurological deficit in the immediate postoperative period. This is in agreement
with our experience. During anesthesia, none of our patients developed focal EEG changes that failed to resolve, and none of our patients showed any evidence of a new neurological deficit in the immediate postoperative period.

The literature also suggests that patients who, during anesthesia, develop focal EEG changes that do not resolve will show evidence of a new neurological deficit in the immediate postoperative period. We have not had any personal experience with patients developing persistent focal EEG changes during anesthesia, but we predict that these patients almost invariably would show evidence of a neurological deficit in the postoperative period. Our prediction is based on the fact that, in our study, patients with a preoperative neurological deficit and a focal EEG abnormality showed an EEG focus which persisted from the beginning to the end of anesthesia. These patients continued to show evidence of a neurological deficit and an EEG focus postoperatively. Therefore, it seems likely that, if such a persistent EEG focus developed for the first time during anesthesia, it would be associated with evidence of a new neurological deficit in the immediate postoperative period.

The exact significance of major focal EEG changes not immediately associated with carotid clamping has not been established with certainty. However, present evidence suggests the following tentative conclusions: (1) transient contralateral changes usually indicate that the patient has significant contralateral disease, and (2) transient ipsilateral changes occurring during the surgery but after closure of the arteriotomy may indicate a thromboembolic source not eliminated by the surgery, a new source created by the surgery, or an acute thrombosis at the site of endarterectomy. These types of EEG changes have alerted us to such complications and, in one patient, have led to reexploration before a fixed neurological deficit could develop. In this patient, flow was restored through an acutely occluded vessel without neurological complications, and the patient left the hospital after a normal postoperative recovery.

**ISCHEMIC TOLERANCE OF NEURAL TISSUE**

The critical cerebral blood flow for maintenance of normal cerebral metabolism in the patient under halothane anesthesia at a normal $\text{Paco}_2$ appears to be 17 ml to 18 ml/100 gm per minute. The establishment of this critical level is based on three observations: 11 of 11 patients who had blood flow below 17 ml/100 gm per minute during carotid clamping developed rapid EEG changes, six of eight patients with blood flow between 17 ml and 18 ml/100 gm per minute developed major EEG changes (in these patients, the latency of onset was usually longer and the magnitude of changes was less than that for patients with flows below 17 ml), and no major EEG abnormalities developed within the first three minutes after carotid clamping when blood flow was above 18 ml/100 gm per minute. The method of analysis used for this comparison is the technique employed in the operating room to provide the surgeon with immediate information regarding CBF, and therefore it is determined from the initial slope of the washout curve. CBF measurements by the "stochastic" method would give slightly lower values.

This critical level of blood flow, as defined by major EEG changes, correlates with the work reported by Alexander et al. In their studies, cerebral oxygen consumption decreased and cerebral glucose uptake increased when cerebral blood flow was reduced to 21 ml/100 gm per minute because of hypocapnic vasoconstriction. A recent study by Trojaborg and Boysen reported a similar value below which EEG alterations were noted during carotid occlusion for carotid endarterectomy. It is lower than the 30 ml/100 gm per minute suggested in earlier studies by Boysen and Finnerty et al.

We have not documented the duration of individual periods of ischemic tolerances in these patients for obvious reasons. A shunt was used in all patients in whom a major EEG alteration was noted in association with a cerebral blood flow less than or equal to the critical level of 17 ml or 18 ml/100 gm per minute. However, it cannot be assumed that such patients would have sustained a fixed neurological deficit had a shunt not been utilized. The shunt is not usually placed prior to adequate distal endarterectomy in the internal carotid artery because we believe that it is imperative to have a clean distal vessel without an intimal flap to serve as a source of emboli. The shunt is ordinarily placed within two or three minutes from the time an EEG change is reported to the surgeon. However, in one patient with a high bifurcation in which it was necessary to complete a very extensive internal carotid endarterectomy before placement of the shunt, an abnormal EEG was noted for ten minutes prior to restitution of flow through the shunt. The EEG in this patient promptly returned to normal when the shunt was placed, and the patient awoke from surgery without evidence of a neurological deficit.

Prior to this study, our laboratory investigations have indicated that in monkeys, after middle cerebral artery occlusion, a degree of ischemia (approximating 15 ml to 20 ml/100 gm per minute in humans) could be tolerated for as long as two hours without the uniform development of cerebral infarction. In these animals, ATP levels decreased to approximately 55% of normal at the end of two hours. Clinical experience indicates that zero cerebral blood flow as seen with cardiac arrest
cannot be tolerated for longer than four minutes without the uniform development of cerebral infarction, and animal studies indicate that after four minutes of total circulatory arrest, ATP levels decrease to 25% of normal. On the basis of these laboratory studies and clinical experience, we had previously presumed that a shunt was virtually never necessary because we believed that the severity of cerebral ischemia after carotid occlusion did not approach zero blood flow seen with cardiac arrest but rather approximated the incomplete ischemia that we had observed after middle cerebral artery occlusion in monkeys. It is now apparent that this is not true. In patients with EEG alterations, the cerebral blood flows range from virtually zero to the marginal flow of 18 ml/100 gm per minute. Within this range, the rapidity of onset and the severity of EEG changes roughly correlate with the degree of reduction of cerebral blood flow below the critical limit of 17 ml to 18 ml/100 gm per minute. At one end of the range, the tolerance to ischemia approaches the four-minute level, and at the other end, the ischemic tolerance more closely approximates the two-hour limit noted in laboratory animals.

Conclusion
The EEG serves as a valuable monitoring device for endarterectomy; EEG tracings correlate closely with CBF changes. The EEG provides moment-to-moment information regarding the patient’s cerebral function, allowing for maximal safety by indicating when a shunt is necessary, how it is functioning when in place, and the adequacy of flow after endarterectomy. This knowledge allows the surgical and anesthetic team to devote their full attention to the technical aspects of surgery without the uncertainty surrounding the possibilities of neurological complications.

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*Stroke*. 1973;4:674-683
doi: 10.1161/01.STR.4.4.674

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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