EGG Surveillance as a Means of Extending Operability in High Risk Carotid Endarterectomy

G. H. MATSUMOTO, M.D., J. D. BAKER, M.D., C. W. WATSON, M.D., B. GLEUCKLICH, M.D., AND A. D. CALLOW, M.D.

SUMMARY Some patients who have transient ischemic attacks are denied operation because severe occlusive lesions in other extracranial arteries may be inappropriately interpreted as constituting an unacceptable surgical risk, or because the lesion is so distal as to make its removal hazardous. Failure of endarterectomy is usually due to incomplete removal of the lesion or to thrombosis upon the frayed intima. Such lesions require excellent visualization and meticulous surgical technique — not always possible with a shunt. Among 130 consecutive carotid endarterectomies performed under general anesthesia, EEG changes consistent with cerebral ischemia appeared in only nine (7%). These patients required a shunt. In 11 patients normal EEG tracings were obtained during endarterectomy despite contralateral carotid occlusion. None of these patients had a neurological deficit. Continuous EEG monitoring is a reliable method of detecting changes in cerebral perfusion, permits a more meticulous endarterectomy in high-lying lesions without a shunt, and extends operability in high risk patients. Angiographical findings may be an unreliable predictor concerning risk of endarterectomy.

Introduction

CAROTID ENDARTERECTOMY for cerebrovascular insufficiency secondary to stenosis and emboli from an ulcerated atherosclerotic plaque in the extracranial internal carotid artery is a widely accepted mode of treatment. However, the methods of detection and the prevention of cerebral ischemia during endarterectomy are somewhat controversial.

Many measures to lessen the risk of carotid endarterectomy have been proposed. Regional anesthesia permits the patient’s mental status to be monitored. Increased cerebral blood flow is alleged to occur under general anesthesia. Hypercarbic general anesthesia has been proposed as a means of cerebral protection, but Boysen indicates there may be disturbances in regional cerebral blood flow although total cerebral blood flow may be increased. Routine use of an intraluminal shunt has been advocated. The insertion of a shunt may cause intimal damage and its presence may make the distal portion of the endarterectomy difficult. Cerebral embolism with a shunt in place has been reported. Because of these liabilities, some authors advocate the use of a shunt only when there is evidence of cerebral ischemia. Internal carotid back pressure has been used as an indicator of cerebral perfusion, but xenon flow studies indicate a poor relationship between back pressure and cerebral blood flow. Injected systemic hypertension increases cerebral blood flow and is used in conjunction with measurement of internal carotid back pressure. There is no agreement on the minimum pressure that is needed to protect against ischemia. Continuous EEG monitoring is utilized in this series as well as others. It can be used with general anesthesia. Studies show that left or right cerebral hypoperfusion will be detected on EEG.

In an attempt to identify those patients at high risk of incurred intraoperative cerebral ischemia or postoperative thrombosis, three potential high risk situations are investigated: (1) those patients with absent extracranial collateral vessels, (2) those with absent intracranial collateral vessels, and (3) those in whom postoperative internal carotid thrombosis or embolus, the distally extending lesion, is most likely to develop.

At the Eighth Conference of Cerebral Vascular Diseases, it was suggested that the risk of carotid surgery depended on coexisting cerebrovascular disease. At particularly high risk are those patients with stenosis of one carotid artery and occlusion of the other: 56 patients studied had an operative mortality of 16%. Another potential high risk group are patients in whom there is lack of angiographic demonstration of intracerebral communicating vessels (anterior communicating or either posterior communicating artery). If these vessels are absent or stenosed, localized ischemia may occur upon carotid cross clamping. We would like to define a third high risk group: patients whose lesion extends high into the extracranial internal carotid artery.
This is a technically high risk group in that the distal endarterectomy may be difficult to terminate and the use of an intraluminal shunt may increase this difficulty.

It is the purpose of this paper to examine these high risk groups in 130 consecutive carotid endarterectomies. There will be an attempt to determine the reliability of using preoperative arteriograms (visualization of extracranial and intracranial collateral vessels) to predict the likelihood of cerebral ischemia during carotid cross clamping.

**Methods**

One hundred thirty carotid endarterectomies from 1974 through February 1976 are reported. Prior to induction of anesthesia a ten-channel bipolar EEG is recorded from needle electrodes placed symmetrically in the frontal, central, occipital and temporal positions. The paper speed is 30 mm per second. All patients receive general anesthesia. Nitrous oxide is used with fluorothane, ethrane or narcotic technique according to the patient's pulmonary and cardiovascular status. Normocarbia is maintained and the blood pressure is kept at the patient's resting level by lightening anesthesia or use of a vasopressor.

The endarterectomy is performed without a shunt unless EEG changes indicative of ischemia appear. The different abnormal EEG patterns have been described in a prior publication. These patterns are generalized slow wave activity, lateralized slow wave activity, generalized slowing with lateralized suppression of slow activity and suppression of fast frequency without slowing. When EEG abnormalities appear in a patient with stable vital signs after cross clamping the common carotid, a shunt is inserted. When the onset of an abnormal EEG pattern accompanies bradycardia or hypotension, these are corrected and in all cases the EEG has returned to normal.

**Patients With Carotid Bifurcation Disease and Contralateral Carotid Occlusion**

Twelve patients with contralateral carotid occlusion underwent carotid endarterectomy. One had generalized slow wave activity after carotid clamping and a shunt was used. The rest had normal EEG patterns and no shunt was used. There have been no deaths in this small series and no postoperative neurological deficits. Internal carotid back pressures ranged from 35 to 95 mm Hg. Only two cases were under 50 mm (each 35 mm). The patient in whom ischemic EEG changes developed had an internal carotid back pressure of 35 mm. Although a contralateral occluded carotid has not been an absolute criterion that cerebral ischemia will develop, we consider these patients at high risk.

**Patients With No Visualization of the Circle of Willis as a Predictor of Cerebral Ischemia**

During carotid cross clamping the cerebral territory supplied by this artery must now be supplied by collateral vessels. Most of these collaterals are via the circle of Willis. If the communicating vessels are congenitally absent or insufficient due to atherosclerosis, localized ischemia may occur because other collaterals are rather small. Of the 130 operations nine patients (7%) showed cerebral ischemia by EEG criteria and required a shunt. Of these nine, eight showed no visualization of at least one communicating vessel (anterior communicating, either left or right communicating) in the intracranial portion of the arteriogram. The ninth patient had basilar artery disease. Of these nine in whom ischemic EEG changes developed and who required an intraluminal shunt, five had internal carotid back pressures of 50 mm Hg or higher and two of these had pressures of 90 mm Hg. Conversely, ischemia may not occur in cases where there is lack of visualization of intracranial communicating branches on arteriograms. An example will be given.

Case 1 (A.G.) is a patient who had one episode of left-hand weakness and confusion. Figures 1 and 2 show the right carotid artery with no communicating vessels.
visualized; figure 3 is a close-up of the ulceration. Figures 4 and 5 show stenosis and ulceration of the left carotid. Figure 6 is a left lateral view of the vertebrobasilar system. Note that none of the communicating arteries in the circle of Willis is visualized. The patient underwent staged bilateral carotid endarterectomies without EEG changes or use of a shunt. No neurological deficits were noted postoperatively.

Thus, intracranial arteriographical data can identify those patients at high risk of having cerebral ischemia. However, many patients placed in this category will not have ischemia.

Discussion

The Eighth Conference suggested that patients at high risk are those having carotid surgery with occlusion of the contralateral carotid artery. In these cases, collateral perfusion must be derived either by reversal of flow through the ophthalmic artery or more likely by way of the vertebrobasilar system. Although arteriography may indicate patency of the posterior communicating arteries, one cannot predict the capacity of these collaterals to prevent cerebral ischemia involving the territory of the patent carotid artery.

Young reported two strokes in 48 of these cases (neurologically stable preoperatively) without the use of a shunt. Bloodwell reported 92 cases with occlusion of the contralateral carotid artery operated without a shunt. Seven died, and five were made worse neurologically (unknown status preoperatively). Thompson reported 64 cases (all operated on) with a shunt with no deaths and no postoperative neurological deficits. We report 12 cases, one requiring a shunt by EEG criteria without mortality or postoperative neurological deficits. Thus, this brief clinical experience suggests the usefulness of intraoperative EEG monitoring in the
detection of cerebral ischemia. Although cerebral ischemia was manifest in only one case we must consider these patients at high risk.

Patients in whom there is lack of arteriographical demonstration of intracerebral communicating vessels (anterior communicating or either posterior communicating artery) may be at high risk during carotid cross clamping. Arteriograms which fail to demonstrate flow from either the contralateral carotid artery or the basilar artery via the circle of Willis do not allow accurate assessment of potential collateral flow. When pressure of both arteries feeding a communicating artery is equal, no crossover may be visualized, even if patent. Thus, lack of visualization of a
communicating artery may be on an anatomical or hemodynamic basis. This is suggested in the patient (Case 1) without radiological evidence of patency of any of the communicating arteries, but a shunt was unnecessary during either endarterectomy. Ischemic EEG changes were found in 7% of our cases and a shunt was used. All cases had incomplete visualization of the circle of Willis. Thus, cerebral ischemia may not develop in patients who lack visualization of intracerebral communicating vessels.

The third high risk group are patients whose lesion extends high into the internal carotid artery and which is defined as involving 3 cm or more of the internal carotid artery. Thrombosis following carotid endarterectomy usually results from incomplete removal of the atherosclerotic lesion, particularly at the most distal margin of the arteriotomy in the internal carotid artery where elevation of the distal intima can develop. Platelet thromboemboli causing transient ischemic attacks may form on frayed intima which was not removed during endarterectomy. These failures are most commonly associated with atherosclerotic lesions that extend distally into the internal carotid artery where, despite maximal mobilization of the internal carotid, a 4-cm internal carotid arteriotomy does not extend past the diseased segment. In these cases a shunt hinders the termination of the endarterectomy, hinders the placement of intimal fixation sutures should they be necessary, and may cause intimal damage or emboli. Usually these distal lesions, which can be appreciated preoperatively, extend even further than suggested by the arteriogram.

Complications of shunts also include embolization. In one case, Sundt⁶ reported a patient who incurred focal ipsilateral EEG changes while a shunt was in place and the patient awoke with a fixed neurological deficit. Marshall¹⁷ has attributed transient focal paresis occurring in the immediate postoperative period to shunts. To us, the threat of these complications contraindicates the routine use of a shunt. Our indication for shunt placement is the appearance of ischemic EEG changes. The cerebrum can tolerate at least three minutes of ischemia. Because these ischemic EEG changes do not always occur immediately after cross clamping the common carotid, there is often more than three minutes to insert a shunt. During this time one can complete the distal endarterectomy and the shunt can then be inserted without the previously stated hazards.

**Conclusion**

Patients undergoing carotid endarterectomy may fall into one or more of the following high risk categories: (1) those with potentially insufficient extracranial collateral circula-
EEG monitoring, carotid endarterectomy without the use of an intraluminal shunt can be extended into this group of patients.

Acknowledgment

The authors wish to thank Miss Reinette F. Bowker, Chief EEG Technician, New England Medical Center Hospital, for her valuable participation during intraoperative monitoring.

References

EEG surveillance as a means of extending operability in high risk carotid endarterectomy.
G H Matsumoto, J D Baker, C W Watson, B Gleucklich and A D Callow

Stroke. 1976;7:554-559
doi: 10.1161/01.STR.7.6.554

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
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:
http://stroke.ahajournals.org/content/7/6/554

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/