- Symon L, Held K, Dorsch NWC: On the myogenic nature of the autoregulatory mechanism in the cerebral circulation. *In* Fieschi C (ed): Cerebral Blood Flow and Intracranial Pressure. Basel, Karger, pp 11-18, 1972
- Sundt TM Jr, Grant WC, Garcia JH: Restoration of middle cerebral artery flow in experimental infarction. J Neurosurg 31: 311-321, 1969
  Sundt TM Jr, Waltz AG: Cerebral ischemia and reactive hyperemia: Studies of cortical blood flow and microcirculation before, during and
- after temporary occlusion of middle cerebral artery of squirrel monkeys. Circulation Research 28: 426-431, 1971
- Ames A III, Wright RL, Kowada M, et al: Cerebral ischemia II: The no-reflow phenomenon. Am J Pathol 52: 437-453, 1968
- Crowell RM, Olsson Y: Observations on the microvasculature in focal cerebral ischemia and infarction. In McDowell F, Brennan RW (eds): Cerebral Vascular Diseases. New York, Grune and Stratton, pp 77-87, 1973
- Crockard HA, Symon L, Branston NM, et al: Changes in regional cortical tissue oxygen tension and cerebral blood flow during temporary middle cerebral artery occlusion in baboons. J Neurol Sci 27: 29-44, 1976
- 31 Symon L, Ackerman R, Bull JWD, et al: The use of the xenon clearance method in subarachnoid haemorrhage. Postoperative studies with clinical and angiographic correlation. *In* Fieschi C (ed): Cerebral Blood Flow and Intracranial Pressure. Basel, Karger, pp 390-396, 1972

## EEG 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

#### 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.<sup>1</sup> 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.<sup>2</sup> Increased cerebral blood flow is alleged to occur under general anesthesia.<sup>3</sup> Hypercarbic general anesthesia<sup>4, 5</sup> has been proposed as a means of cerebral protection, but Boysen<sup>6</sup> 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.<sup>7</sup> 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.8 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,9. 10 but xenon flow 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.

studies indicate a poor relationship between back pressure and cerebral blood flow.<sup>6</sup> Induced systemic hypertension increases cerebral blood flow and is used in conjunction with measurement of internal carotid back pressure.<sup>11</sup> There is no agreement on the minimum pressure that is needed to protect against ischemia.<sup>10, 12</sup> Continuous EEG monitoring is utilized in this series as well as others.<sup>13</sup> It can be used with general anesthesia. Studies show that left or right cerebral hypoperfusion will be detected on EEG.<sup>14</sup>

In an attempt to identify those patients at high risk of incurring 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,<sup>15</sup> 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 arteriographic demonstration of intracerebral communicating vessels (anterior communicating or either posterior communicating artery). If these vessels are either 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.

Departments of Surgery and Neurology, Tufts-New England Medical Center, 171 Harrison Avenue, Boston, Massachusetts 02111 (reprint requests to Dr. Callow).

Supported in part by a General Research Support Grant #27418 and The Earl Tupper Foundation.

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.<sup>16</sup> 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 lefthand weakness and confusion. Figures 1 and 2 show the right carotid artery with no communicating vessels



FIGURE 1. Patient A. G., selective right carotid arteriogram showing lack of crossover to the opposite anterior cerebral artery and no filling of the posterior cerebral artery.



FIGURE 2. Patient A. G., lateral view of the right carotid artery showing no filling of the posterior cerebral artery.

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.

#### Patients With a Lesion Extending Distally Into the Internal Carotid

We have found that some lesions are not confined to the orifice of the internal carotid artery. When the intima does not taper to normal despite a 4-cm or more internal carotid arteriotomy, the endarterectomy is technically difficult to terminate without the risk of distal intimal elevation. An example is given.

Case 2 (S.S., age 75) is an example of a lesion that extends quite distally into the internal carotid as opposed to most lesions that are confined to the bifurcation. Expressive aphasia and blurring of vision were the presenting signs. Figures 7 and 8 show stenosis of the left internal carotid. At operation the left internal carotid lesion extended even more distally than the 3 cm shown by x-ray and a shunt would have compounded the difficulty of terminating the endarterectomy. Good visualization of the most distal intima was needed to prevent an intimal flap. Postoperative arteriograms show the extent of the endarterectomy (fig. 9).



FIGURE 3. Patient A. G., magnified view of the right carotid bifurcation revealing an ulcerated plaque.

#### Discussion

The Eighth Conference<sup>16</sup> 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<sup>5</sup> 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). Thompson7 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



FIGURE 4. Patient A. G., left carotid injection revealing no filling of the contralateral anterior cerebral artery.

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

FIGURE 6. Patient A. G., vertebrobasilar arteriogram showing no filling of either posterior communicating artery.



FIGURE 5. Patient A. G., lateral left carotid view showing no filling of the left posterior cerebral artery.





FIGURE 7. Patient S. S., left carotid injection showing stenosis of the left internal carotid artery.

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



FIGURE 8. Patient S. S., lateral view of the left carotid artery showing stenosis and ulceration; the end of the plaque measures more than 3 cm from the orifice of the internal carotid artery.

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, Sundt<sup>8</sup> reported a patient who incurred focal ipsilateral EEG changes while a shunt was in place and the patient awoke with a fixed neurological deficit. Marshall<sup>17</sup> 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-



FIGURE 9. Patient S. S., postoperative left carotid arteriogram revealing the length of the endarterectomy and leading edge of remaining intima.

tion, (2) those with potentially insufficient intracranial collateral circulation, and (3) those with the atherosclerotic lesion that extends distally into the internal carotid artery. We believe all patients with a 3-cm or more lesion extending into the internal carotid artery by arteriogram are at high risk from a technical standpoint.

Cerebral ischemia occurred in 7% of 130 patients. They had lack of visualization of one or more collateral vessels on arteriograms. However, many patients who were categorized as high risk due to potentially insufficient collateral vessels did not have cerebral ischemia. Using continuous 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

- Clauss RH, Sanoudos GM, Ray JF, et al: Carotid endarterectomy for cerebrovascular ischemia. Surg Gynec Obstet 136: 993-1000 (Jun) 1973
  Hobson RW, Wright CB, Sublett JW, et al: Carotid artery back pressure
- Hobson RW, Wright CB, Sublett JW, et al: Carotid artery back pressure and endarterectomy under regional anesthesia. Arch Surg 109: 682-687 (Nov) 1974
- Howe JR, Kindt GW: Cerebral protection during carotid endarterectomy. Stroke 5: 340-343 (May-Jun) 1974
  Young JR, Humphries AW, Beven EG, et al: Carotid endarterectomy
- Young JR, Humphries AW, Beven EG, et al: Carotid endarterectomy without a shunt. Experiences using hypercarbic general anesthesia. Arch Surg 99: 293-297 (Sep) 1969
- Bloodwell RD, Hallman GL, Keats AS, et al: Carotid endarterectomy without a shunt. Results using hypercarbic general anesthesia to prevent cerebral ischemia. Arch Surg 96: 644-652 (Apr) 1968
  Boysen G: Cerebral blood flow measurement as a safeguard during
- Boysen G: Cerebral blood flow measurement as a safeguard during carotid endarterectomy. Stroke 2: 1-10 (Jan-Feb) 1971
- Thompson JE, Austin DJ, Patman RD: Carotid endarterectomy for cerebrovascular insufficiency: Long term results in 592 patients followed up to thirteen years. Ann Surg 172: 663-679 (Oct) 1970
  Sundt TM, Sharbrough FW, Anderson RE, et al: Cerebral blood flow
- Sundt TM, Sharbrough FW, Anderson RE, et al: Cerebral blood flow measurements and electroencephalograms during carotid endarterectomy. J Neurosurg 41: 310-320 (Sep) 1974
- Hays RJ, Levinson SA, Wylie EG: Intraoperative measurement of carotid back pressure as a guide to operative management for carotid endarterectomy. Surgery 72: 953-960 (Dec) 1960
  Moore WS, Yee JM, Hall AD: Collateral cerebral blood pressure: An in-
- Moore WS, Yee JM, Hall AD: Collateral cerebral blood pressure: An index of tolerance to temporary carotid occlusion. Arch Surg 106: 520-523 (Apr) 1973
- Akl BF, Blakeley WR, Lewis CE, et al: Carotid endarterectomy: Is a shunt necessary? Am J Surg 130: 760-765 (Dec) 1975
  Hughes RK, Bustos M, Byrne JP: Internal carotid artery pressures. A
- Hughes RK, Bustos M, Byrne JP: Internal carotid artery pressures. A guide for use of shunt during carotid repair. Arch Surg 109: 494-495 (Oct) 1974
- Horton DA, Fine RD, Lethlean AK, et al: The virtues of continuous EEG monitoring during carotid endarterectomy. Aust NZ J Med 4: 32-40 (Feb) 1974
- Sharbrough FW, Messick JM, Sundt TM: Correlation of continuous electroencephalograms with cerebral blood flow measurements during carotid endarterectomy. Stroke 4: 674-683 (Jul-Aug) 1973
- Blaisdell W: Extracranial arterial surgery in the treatment of stroke. In McDowell FH, Brennan RW (eds): Cerebral Vascular Disease. Eighth Conference. New York, Grune and Stratton, 1973
  Baker JD, Gluecklich B, Watson CW, et al: An evaluation of electro-
- Baker JD, Gluecklich B, Watson CW, et al: An evaluation of electroencephalographic monitoring during carotid surgery. Surgery 78: 787-794 (Dec) 1975
- Marshall BM, Lougheed WM: The use of electroencephalographic monitoring during carotid endarterectomy, as an indicator for application of a temporary bypass. Canad Anaesth Soc J 16: 331-335 (Jul) 1969





### **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/