Results of Electroencephalographic Monitoring During 367 Carotid Endarterectomies

Use of a Dedicated Minicomputer

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SUMMARY Three hundred sixty-seven carotid endarterectomies were monitored using routine electroencephalographic (EEG) techniques. In 9.8%, changes in the EEG followed clamping of the internal carotid artery and could roughly be grouped into 6 patterns. The most common (47%) was rapid unilateral or bilateral attenuation of background anesthetic-induced fast EEG activity. Conclusions drawn from studies of the 9 patients who had immediate postoperative neurological deficits of varying degrees are presented as they illustrate monitoring techniques that are useful for early recognition of cerebral ischemia. Thirty-five patients were monitored with both routine and computerized techniques — the latter proved more useful than the former on all counts.

THE CLINICAL UTILITY of carotid endarterectomy for occlusive vascular disease has been well documented. One inescapable technical feature of the surgical procedure, cross-clamping of the carotid artery, puts the ipsilateral cerebral hemisphere at risk for ischemic damage. In some centers, carotid endarterectomies are done under local anesthesia, so that neurological function can be monitored clinically during the operation. This anesthetic technique has limitations and in most centers general anesthesia is used. Recognition of cerebral ischemia at a time when something can be done to reverse it, is, therefore, desirable. Internal carotid artery stump pressures have proven to be unreliable indicators of cerebral blood flow during the period of clamping. Regional cerebral blood flow measurements using intra-arterial xenon-133 are useful but require equipment not readily available in most departments. This method can only be used at discrete points throughout the procedure, and may be complicated by the “look-through” phenomenon.

Clinical neurophysiology laboratories have become legitimately concerned with monitoring cerebral function during the period of general anesthesia for carotid surgery. The data obtained by EEG monitoring can be used during the procedure to determine whether or not a temporary by-pass shunt is necessary to diminish the likelihood of intraoperative cerebral ischemic damage. A shunt is not used routinely because of added risk of embolic complications and because it sometimes adds to the technical difficulty of the procedure by limiting exposure of the distal portion of the plaque. Although it has been suggested that shunting is unnecessary, that is not the prevailing opinion.

There is a close correlation between cerebral blood flow and the EEG. Experience has shown that the EEG is a very reliable indicator of cerebral ischemia during carotid endarterectomies. The relationship between complications recognized in the immediate postoperative period and the presence or absence of EEG changes has been discussed by groups whose experiences differed from ours, perhaps because of differences in monitoring techniques. We present here: 1) the results of EEG monitoring during 367 carotid endarterectomies; 2) the significance of the EEG in relation to immediate postoperative neurological deficits; 3) important features of the monitoring techniques and interpretation as illustrated by our experience; and 4) the results of simultaneously monitoring 35 of these patients with a dedicated minicomputer. This latter technique proved to be of greater utility than did routine EEG methods. Abstracts of this material have been presented.

Methods

The EEG laboratory of the Massachusetts General Hospital monitored 406 carotid endarterectomies (353 patients) between January 1, 1972 and January 1, 1978. A few carotid endarterectomies were done during this period without EEG monitoring. The charts of 317 patients were available and those patients constitute the group reported here: 270 patients had 1, 44 had 2, and 3 had 3 endarterectomies, for a total of 367 endarterectomies monitored. All of the 367 intraoperative EEG’s were available in their entirety on microfilm and the original paper tracings were available for more than half. EEG’s were reviewed, described and re-interpreted without knowledge of clinical data.

The details of the usual surgical procedure have
been published previously and do not differ significantly from customary techniques. In brief, after isolation of the involved vessels, a test clamp period of 2-3 min was used to determine the necessity of a temporary bypass shunt. EEG, stump pressure, clinical, and radiological information were taken into account. The endarterectomy was then performed with or without a shunt.

The EEG electrodes were applied when the patient arrived in the operating room. In the initial part of the study, lead disc electrodes were affixed with adhesive tape in International 10-20 System positions Fp1, Fp2, C3, CZ, C4, T3, T4, 01, 02, A1, and A2, using pumice paste degreasing of the scalp and bentonite paste. In the last 18 months, Grass gold cup electrodes with a central hole were fixed in place at the same sites by a collodion-soaked gauze square and filled with standard saline gel electrode paste. The scalp beneath the cup electrodes was lightly abraded with the same blunt-tip needle used to inject the paste. With both techniques, electrode impedance was maintained below 5000 ohms. The collodion technique helped to avoid the electrodes being pulled off. Electrodes were connected to the EEG machine via a Grass Biopotential Isolator Electrode Board (Model 1MEB).

Grass Model 6 8-channel and Model 8 9-channel EEG machines were used, with a filter bandpass of 1-70 Hz, and 60 Hz notch filters in operation. The sensitivity was usually 7 microvolts per mm, but this was often increased as necessary to provide sufficient pen deflection (at least 7 mm) for easy visual recognition of attenuation changes when the activity was of a relatively low voltage. Paper speed in the initial part of the study was 30 mm/sec, but in the last 18 months this was changed to 15 mm/sec since the slower speed produced a time compression effect which made it easier to recognize amplitude changes over their usual time course of progression (5-30 sec), and accentuated slow activity. The saving in paper also is considerable.

The montage most commonly used was Fp1-C3, C3-01, Fp2-C4, C4-02, T3-C3, C3-CZ, CZ-C4, C4-T4, and ECG. On the 8-channel machines, the transverse coronal part of the montage was shortened to T3-C3, C3-C4, C4-T4 with ECG on the last channel. Slightly different bipolar and ear reference montages were also used occasionally.

Monitoring was begun at the time of anesthetic induction. At the beginning of the study, monitoring was halted during the exposure, dissection, and isolation of the carotid artery but in the last 9 months of the study monitoring was continuous. Similarly, monitoring originally was usually halted 2-4 min after the endarterectomy was finished and clamps released but in the last year of the study monitoring was continued until the patient was awake enough to cooperate in a neurological examination.

Thirty-five patients studied in 1977 were monitored with on-line computer analysis of the EEG, in addition to the usual techniques. A PDP 11/04 with 32K of processor memory, 16-channel A/D converter, dual floppy discs, Tektronix 4006 graphic storage CRT and 4631 hard copy unit were contained in a single 4-foot standard cabinet, specially fitted with 5-inch diameter electrically conductive wheels; all power for the equipment was derived from an isolation transformer. This portable system was placed in the operating room, analysis and display processes proceeded in real time, and the results were immediately available for viewing. Sampling and analysis were performed in a double-buffered mode so that no data were lost except for 10 sec at the end of each page (every 6.7 min) during screen erase and relabeling. The computer was fed the amplified signals (1-4 volts) from 4 channels of the EEG machine (Fp1-01, Fp2-02, T3-C3, T4-C4) and 128 samples/sec/channel were taken. Software Fast Fourier Transform (FFT) was performed on 4 sec epochs and the results plotted on the CRT in a Compressed Spectral Array (CSA) format with the 2 channels from each hemisphere combined by simple averaging. Thus, in figure 1 the left-most column is the CSA of activity of the left hemisphere and the next column is from the right hemisphere, with time rising vertically in 4 sec steps and 6.7 min total time per page. The third column from the left shows the frequency difference between the 2 hemispheres and is arranged so that activity present on the operated side and absent from the unoperated side (such as slow waves) shows up as a peak; conversely, activity that is present on the unoperated side and absent from the operated side shows up as a trough (attenuation of normal anesthetic-induced activity on the operated side shows up as troughs). The right-most column represents an amplitude integration of total area covered by the EEG activity in that 4 sec epoch, such that the higher the amplitude of activity, the longer the line. Activity from the left hemisphere is represented by the lines under the L that extend from the center line to the left; that from the right hemisphere is under the R and extends to the right. By visually comparing the relative lengths of the lines one can easily see...
which hemisphere has the highest amplitude activity. As each page was filled, a hard copy was automatically made, the screen erased, and a new page started. Each page was labeled with a number and the starting time. In addition, the operator could cause a numbered event marker to be placed next to an epoch (see fig. 2) simply by typing M (for mark). By keeping a suitable log, these marks could later be correlated with events and with measurements of interest during the procedure. All output and computations could easily be scaled with simple operator-typed commands. A simple algorithm suppressed display of lines contaminated by excessive artifact. The sum of the activity in each of 4 frequency bins (alpha, beta, theta, delta) and the integration values were stored on a floppy disc for each of the 4 channels for each 4 sec epoch. At the end of the procedure a summary graph was produced (fig. 4) which also automatically included the event markers.

**Results**

The EEG during general anesthesia reflects to a large degree a product of the anesthetic agent being used and PaCO$_2$. A variety of agents were used in these procedures, often in combinations, but the most common was fluothane. Thus the usual intraoperative EEG consisted of continuous rhythmic symmetrical 10-14 Hz 25-75 microvolt activity with varying amounts of slow activity superimposed; as anesthesia deepened the slow activity predominated. If the technical details outlined above are strictly adhered to, various artifacts produced by the operating or recovery room staff or equipment usually did not interfere with the EEG for sufficiently long periods to render interpretation difficult. Only 1 patient had a large amount of muscle artifact which appeared with clamping of the artery and obscured the EEG trace.

However, interpretation of the intraoperative EEGs was complicated by 4 factors: 1) patients with preoperative clinical neurological deficits usually had focal EEG abnormalities which persisted throughout the monitoring session; 2) changes in blood concentrations of the various anesthetic agents used and changes in PaCO$_2$ (which usually are not measured) both induced bilateral EEG changes, sometimes difficult to differentiate from changes secondary to events related to the carotid surgery (e.g. clamping, shunt malfunction, thrombosis) which were often bilateral also; 3) in the early part of the study the EEG machine sensitivity sometimes was not high enough to produce sufficient pen deflection to allow easy recognition of EEG attenuation; and 4) changes in level of consciousness due to "painful" aspects of surgery.

The EEG was monitored during 367 carotid end-
of the 36); the EEG did not change in 4 patients with an immediate postoperative deficit. In 36 EEGs (9.8%), a change seen during the procedure was thought to be caused by cerebral ischemia secondary to the procedure (usually associated with clamping and reversed by shunting) — 6 of these patients (16.6% of the 36) had an immediate postoperative deficit. Preoperative arteriograms showed that the opposite internal carotid was occluded in 34 patients whose EEG showed no change with surgery (10.3% of the 331) and in 13 patients whose EEG did change (36.1% of the 36); the EEG did not change in 4 patients with both ICA occluded.

Immediate Postoperative Deficits

Of the 9 complications, 2 were caused by re-thrombosis at the operative site which was not revealed by the EEG because recording was stopped immediately after unclamping and before the occlusion occurred. For example, 1 showed dramatic EEG changes during the procedure when the ICA was clamped and thus the EEG could be expected to have shown the same change with the thrombosis had the latter occurred during the period of recording. One complication was embolic in origin; the EEG showed no changes during the procedure but was not continued until recovery of consciousness. In 4 complications the etiology was uncertain and all except 1 had no permanent deficit. EEG monitoring was halted shortly after final clamp releases in all; at that time the EEG in 1 was slightly asymmetrical, in 1 it was normal, and in 1 there was no slow activity but attenuation could have been missed because of low machine sensitivity. Two showed EEG changes consistent with the clinical outcome, although in 1, because attenuation occurred while the machine was temporarily turned off (after induction of anesthesia but before the artery was dissected free), the correct interpretation was not reached until a postoperative review.

Patterns of EEG Changes

Thirty-six procedures (9.8% of the entire 367) were associated with an intraoperative change in the EEG thought to be caused by cerebral ischemia secondary to some event other than an anesthetic or ventilatory change (e.g. decreased cerebral blood flow (CBF) following ICA clamp or embolus). As noted above, 6 of these patients (16.6% of the 36) had immediate postoperative deficits, but only in 2 of these was the EEG change related to the outcome, the others to some extent being incidental in that the EEG returned to normal before monitoring was halted and the event which produced the complication occurred thereafter.

In 32 of the 36 patients, (88.9%) EEG changes were directly related to clamping and appeared within 20 sec after the clamps were applied. These changes usually persisted during the test period (1–3 min) and/or during the time taken to insert the shunt (2–10 min). Occasionally, there was a tendency for the EEG changes to reverse although this was never complete until the shunt was open. The changes were reversed by shunts except in 1 patient with a poor outcome.

One can arbitrarily categorize, in the following fashion, the EEG changes seen during these operative procedures. Although there is often a good deal of overlap such grouping helps to organize the data.

Group 1. Ipsilateral Attenuation with Clamping

Within 20 sec of ICA clamping, 10 EEGs (27.8% of the 36) showed an ipsilateral voltage attenuation of greater than 50% (e.g. fig. 7). In 1 patient, the attenuation appeared gradually over several minutes. Another patient had a marked attenuation with test clamping and rapid return on clamp release; however, with clamping for shunt insertion, there was no change; at the end of the procedure there was again ipsilateral attenuation with clamping for shunt insertion; clamping for shunt removal produced no attenuation. In all patients but 1, the attenuation was reversed within 20 sec to 3 min after shunt opening. One patient could...
Figure 6. The top 2 segments show typically rapid return of activity following release of carotid clamp (at UNCLAMP). The 10 sec segment at the bottom shows the appearance of the EEG for 35 minutes during the accelerated endarterectomy with a shunt which did not improve the EEG; the patient had a severe post-operative neurological deficit.

not be shunted and the EEG remained markedly attenuated for 13 min during the period of the greatly accelerated endarterectomy; within 6 min of clamps being released, the EEG had returned to normal and the patient had no complications. The one complication suffered in this group was in the patient whose attenuation was not reversed by shunt opening; her symptoms were mild and she recovered completely.

Group 2. Bilateral Attenuation with Clamping

Within 20 sec of ICA clamping, 7 patients (19.4%) developed bilateral voltage attenuation of greater than 50% (e.g. figs. 5 and 6). All but 1 had reversal of the attenuation within 20 sec to 3 min of shunt opening; that patient had marked attenuation for 35 min even with shunting and little return 18 min after the clamps were released — she had severe complications. One patient was not shunted; his EEG, although showing bilateral attenuation, was primarily affected ipsilaterally; 8 min after clamping his EEG began to show some return of activity, but was still at least 50% reduced in voltage 24 min later when the clamps were released; 6 min later the EEG had regained 80% of its previous voltage. He did not suffer a complication.

Group 3. Ipsilateral Slowing without Attenuation with Clamping

Within 20 sec of ICA clamping, 5 patients (13.9%) developed ipsilateral delta and theta activity but only a small degree of attenuation of fast activity; this sometimes disappeared spontaneously and always disappeared within 20 sec to 3 min of shunt opening. There was one complication in this group whose relationship to the EEG changes is unclear.

Group 4. Ipsilateral Slowing with Attenuation with Clamping

Within 20 sec of ICA clamping, 5 patients (13.9%) developed ipsilateral delta and theta activity which lasted 5 to 15 sec and was then followed by ipsilateral
voltage attenuation, the latter persisting until the clamp was released or the shunt was opened, when the changes were always reversed within 20 sec to 3 min. There was one complication in this group which was not related to the EEG changes (re-thrombosis at the operative site after monitoring was halted).

Group 5. Bilateral Slowing and Attenuation with Clamping

Within 20 sec of ICA clamping, 5 patients (13.9%) developed bilateral theta and delta activity lasting 5–20 sec and followed by bilateral attenuation. These patients usually showed some reversal of the attenuation within 4–5 min, even without shunting, although that usually reversed the attenuation completely. After shunting, 1 patient again manifested this sequence of EEG changes and it was discovered that the shunt had kinked; restoration of flow again reversed the EEG changes. There were no neurological complications in this group.

Group 6. Delayed Changes Unrelated to Clamping

Four patients (11.1%) developed EEG changes during the procedure which were not related temporally to the application of clamps. One patient had ipsilateral attenuation (seen only in the computer display) with a fall in systemic blood pressure (BP), reversed by raising the BP. One patient developed an ipsilateral attenuation and slowing 24 min after clamping (no shunt); this remained for 30 min until unclamping, at which time it rapidly returned to normal. One patient had intermittent ipsilateral slow activity throughout the procedure. None of these patients had neurological complications.

Computerized Monitoring

Thirty-five patients were monitored with the computer system as well as with the routine methods. In no instance did the routine techniques reveal a significant pattern not seen with the computer. On the contrary, the computer always rendered the EEG changes more readily apparent to all OR personnel than did the routine methods, thus aiding communication.

EEG patterns produced by anesthetic agents were clearly seen in the computer display, except for burst-suppression activity sometimes induced transiently by fast-acting barbiturates. The 4 sec epoch used for analysis produced this "smoothing" effect since the period of the burst-suppression was usually less than 4 sec. The computer was in use for 0, 2, 1, 1, 1, and 2 of the changes listed above in Groups 1–6 respectively (7 patients) and clearly demonstrated the changes in each patient. In one patient (Group 6) the computer revealed a subtle amplitude attenuation not evident on the routine paper trace — with raised BP this change disappeared (see figs. 2, 3, 4). Using the experience gained with the computer, EEG records from the remaining 332 operations, 28 with and 304 without significant EEG changes, were retrospectively examined, and it is our belief that the computer would have clearly revealed all changes.

Discussion

Studies of the relationship between CBF and cerebral cortical electrical activity (EEG) in animals establish a critical value of 16–18 ml/100 gm/min below which EEG activity becomes abnormal. Below 12 ml/100 gm/min, EEG activity is absent. These values are essentially the same as those seen in humans undergoing carotid endarterectomy. Thus, the EEG is a good indicator of adequacy of regional CBF, as opposed to stump pressures, which have been shown to be unreliable. The EEG is a useful adjunct to carotid endarterectomies because it can indicate whether or not a temporary by-pass shunt is necessary during the period of carotid clamping. Though a study has not been (and, perhaps, should not be) done to settle the matter definitely, our data lead us to believe that if clamping causes sustained slowing or voltage attenuation in the EEG, a shunt should be used.

In our patients, following the insertion of the shunt, the EEG returned to normal; in 2 where this did not occur, the patients had immediate postoperative neurological sequelae. Two patients who were not shunted but had EEG attenuation with clamping had no neurological sequelae, but 1 had purely unilateral EEG changes and was deliberately clamped for only 13 min. The other (in Group 2) showed some spontaneous return of EEG activity after 8 min of clamping, perhaps due to increased collateral blood flow. In 6 of our patients with immediate postoperative neurological complications, the EEG monitoring had either been halted before the ischemic event (in 5) or was run at insufficient sensitivity to allow recognition of attenuation and therefore could not forecast the deficit. Because of these experiences we changed our techniques. Monitoring is now continued throughout the entire procedure until the patient recovers from anesthesia and is able to cooperate during a neurological examination. EEG recording sensitivity is carefully maintained at a suitable level (usually 5 or 3 microvolts/mm). The importance of these technical details is also emphasized by the experience of Sundt, Sharbrough, and co-workers and Baker et al. who describe situations in which the EEG showed dramatic abnormalities after the arteriotomy had been closed but before the patient recovered from anesthesia; the EEG then led to early recognition and correction of problems at the operative site (patch graft or removal of embolic source). In addition, we have installed a multi-core cable from the operating room to the EEG Laboratory where a "repeater" EEG machine and an intercom allow electroencephalographers to review the record and consult with the technician and surgeon in the OR without travelling there and changing into OR dress, a time-consuming process which often renders such necessary consultations impractical.

A wide range in the percentages of EEG changes during carotid clamping is present in previously published series — 29% of 93, 11% of 213, 26% of 106, 14% of 369, 15% of 88, 27% of 29, and 27% of 52 cases — a mean of 21%, compared with our 9.8%
of 367 which is the lowest. Almost uniformly a shunt reversed all these changes. In addition, as noted above, changes unrelated to clamping can be expected; there were 4 (1.1%) in our series (Group 6) and others have reported 7 (3%)12 and 26 (7%).14 In almost all series, the EEG accurately predicted whether or not the patient would have a neurological deficit on awakening from anesthesia (although some series do not discuss the EEG recorded in patients with postoperative complications).1, 18 We agree with Sundt et al.14 that “patients who do not develop a new persisting focal EEG change during anesthesia will not have evidence of a new neurological deficit in the intermediate postoperative period and patients who develop a persisting focal EEG abnormality during anesthesia will have a new neurological deficit.”

Harris et al.1 operated under local anesthesia and noted that neurological deficits appeared after the EEG changes; they express the belief that because of the imminence of the EEG change a shunt could not safely be inserted and deferred surgery in 2 patients, operating upon another under hypothermia. Although the attenuation seen with carotid clamping in some patients signifies an extremely low rCBF there is certainly a margin of safety of several minutes and a shunt can be inserted without a permanent deficit resulting from the relatively brief low-flow state. This safety margin was also seen in monkey experiments28 where complete occlusion of the middle cerebral artery for 1-2 hrs caused no or mild neurological deficits. Most of our patients who had attenuation changes had shunts placed and this almost always reversed the EEG change within 2-3 min which seems to be the usual experience in the literature. In most series general anesthesia was used; this may impart to the cortex some degree of protection from ischemic damage, although it is probably almost insignificant at usual anesthetic levels as compared with the deep barbiturate anesthesia which was shown to be protective in experimental middle cerebral and internal carotid artery occlusions in dogs.27 This type of therapy, although tried in humans,28 has not yet been demonstrated to be helpful and the dose of barbiturate given (about 2 gr of thiopental) would induce a “burst-suppression” and/or essentially isoelectric pattern in the EEG, both of which would probably render the EEG less useful as a monitor of cerebral function.

Our human experience does not allow us to state what duration of each EEG change can be tolerated before a neurological complication will ensue. One patient in Group 1 who could not be shunted had a marked ipsilateral attenuation of the EEG following clamping which was unchanged during the 13 min taken to do a greatly accelerated endarterectomy; within 6 min of unclamping his EEG had returned to normal and there were no complications. One patient in Group 2 who could not be shunted showed bilateral attenuation more marked on the clamped side; 8 min later his EEG began to show some return of activity but was still at least 50% reduced in voltage 24 min later when the clamps were released. Six min later his EEG had regained 80% of its previous voltage and there were no complications. One patient had a markedly attenuated EEG for 35 min during clamping (shunt was not possible) and she had neurological sequelae. These examples, along with those which showed attenuation without neurological sequelae, suggest that there is a safety margin and that an attenuated EEG can be tolerated for some period of time, possibly up to 10 min, without permanent cerebral damage.

The variety of EEG changes seen with unilateral carotid clamping was striking. Although there was overlap, the first 5 groups listed in Results-Patterns of EEG Changes provide a framework for discussion. Bilateral changes were seen more often when the contralateral internal carotid artery was occluded. There was almost every possible combination of slowing and attenuation; most of these patterns have been described in the literature mentioned above, although usually not in detail. The sequence of progression through slowing to attenuation, the stage finally reached, and the speed of progression probably are related to the change in rCBF as well as its final value.

The most important EEG change seems to be voltage attenuation since it was seen in the 2 patients where the EEG predicted a poor outcome, and was not seen to that extent otherwise. This agrees with the human studies combining EEG and rCBF,9-13, 18, 29 where voltage attenuation was associated with flows of less than 10 ml/100 gm/min. We have also seen (in patients not included in this study) generalized slowing of the EEG associated with a fall in systemic BP of more than 20-30 mm Hg below the usual level for that patient; raising the BP pharmacologically abolished these changes.

Computerized analysis and display techniques have been used previously in a small number of cases (22). Our experience here also showed that this method of presenting EEG data, which renders it interpretable by all operating room personnel, was instrumental in creating enthusiasm and confidence in the results of EEG monitoring. These methods also are more effective than the traditional ones in revealing subtle changes, especially attenuation which appears to have a more serious significance than the slow activity which is more easily recognized. In addition, there is unlimited opportunity for simultaneous hard-copy display of multiple other physiological parameters, all of which have been subjected to various analytic and data reduction processes. A small, relatively cheap, microprocessor-based system is presently under development.

The ready availability of EEG and its proven accuracy in monitoring cerebral perfusion and oxygenation during carotid endarterectomy make it a useful adjunct to the procedure provided close attention is paid to details of EEG technique. Experience gained in this area suggests that a much more widespread application of EEG monitoring during any period of unconsciousness is indicated and will provide increased patient protection from the wide variety of incidents and accidents which may lead to cerebral ischemic damage in those circumstances. The EEG, as
a "final common denominator" of cerebral perfusion and oxygenation, continuously reflects changes in cerebral function within seconds, as opposed to measurements such as intracranial pressure, systemic BP, arterial blood gases, end-expired Pco₂, or anesthetic gas concentrations, whose relationships to cerebral function are more indirect, less immediate, and less all-encompassing. The addition of computerized analysis and graphic display methods using microprocessor-based devices will certainly aid in the wider use of this tool which has often been considered too complex, and therefore unreliable, as a method for recognizing cerebral ischemia at an early stage where it can be treated with maximum likelihood of avoiding a permanent deficit.

Acknowledgment

We wish to thank the vascular surgeons, neurosurgeons, and anesthetists of the Massachusetts General Hospital for their cooperation in these studies. We also wish to thank the technicians from the EEG Laboratory who spent many hours sitting in the OR.

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K H Chiappa, S R Burke and R R Young

Stroke. 1979;10:381-388
doi: 10.1161/01.STR.10.4.381

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