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Direct Cortical EEG Monitoring during Temporary Vascular Occlusion for Cerebral Aneurysm Surgery

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Anesthetic management of cerebral aneurysm clipping includes maintenance of an acceptable transmural pressure to prevent rupture of the aneurysm, especially during surgical manipulation. Systemic hypotension has been widely practiced to achieve this goal. Recently, there has been a trend toward increasing use of temporary vascular occlusion to secure surgical control of anatomically difficult lesions,[1,2] thereby avoiding systemic hypotension. Although highly desirable during vascular occlusion, monitoring of brain function with either somatosensory evoked potentials or standard scalp recording of EEG presents certain limitations during craniotomy for aneurysm clipping.[3] We have therefore begun to use direct cortical recording using subdural strip electrodes. We report here results of cortical EEG monitoring during two cases of temporary vascular occlusion and the implications for patient management.

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METHODS

The EEG data collection system consisted of a device producing a compressed spectral array (CSA) (Neurotrac®, Interspec Inc., Conshohocken, Pennsylvania) and an Apple Macintosh II computer. Either one or two channels of analog EEG data were processed and displayed. We examined multiple spectral descriptors, but present here only the ratio of power in the theta and delta bands to the power in the alpha and beta bands, (D + T)/(A + B). The equipment and analysis algorithms are similar to systems described previously.[4,5]

The EEG electrodes were Cortac® subdural strips (PMT Corp., Minneapolis, Minnesota). Each strip con-

tained a linear array of four platinum circular contacts, 5 mm in diameter, imbedded at 1-cm intervals in silastic. The electrodes are placed subdurally in the region of interest and held in place on the cortical surface with a moist cotton pledget. The placement of a typical electrode array is shown in figure 1.

Case Reports

Case 1. A 73-yr-old woman had a 2-yr history of progressive visual loss. Magnetic resonance imaging scan demonstrated a giant intracranial aneurysm in the left parasellar region. On admission, her blood pressure ranged as follows: 120–130/70–80 mmHg; physical examination was normal, except for marked loss of visual acuity bilaterally and a complete right homonymous hemianopia. Cerebral angiography demonstrated a 3-cm aneurysm in the supraclinoid segment of left internal carotid artery, distal to the ophthalmic artery.

The patient was taken to the operating room for a left frontotemporal craniotomy. General anesthesia was induced with midazolam, thiopental, fentanyl, and vecuronium and was maintained with 0.75% isoflurane in 3:2 nitrous oxide and oxygen. The Pa_{CO_2} was maintained at 28–30 mmHg. A spinal drainage catheter was inserted. The left internal carotid artery was isolated in a snare through a cervical incision for subsequent occlusion and the craniotomy was begun. The patient received 1 g/kg of mannitol. After dural reflection a strip of cortical EEG electrodes was placed on the surface of the left frontal lobe near the motor strip.

A temporary clip was placed on the ophthalmic artery. The cervical carotid artery was then occluded, and a temporary clip was placed across the carotid artery distal to the aneurysm. The blood pressure was 130/70 at this time. No EEG changes were noted with the vascular occlusions.

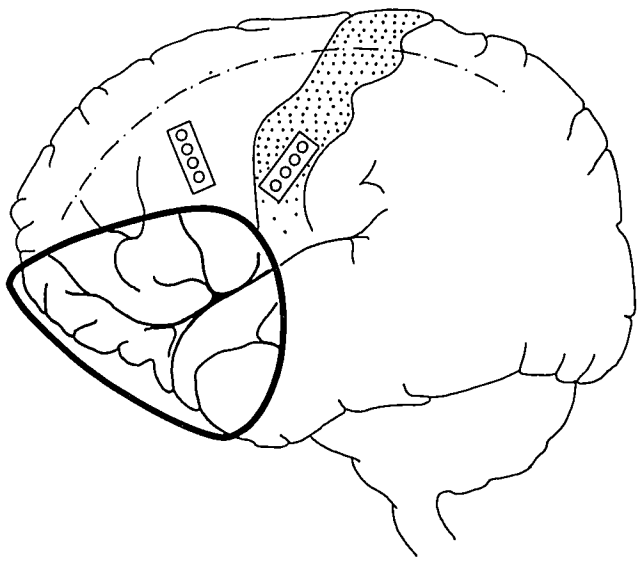

FIG. 1. Intraoperative cortical electrode placement. The edge of the pterional craniotomy site is depicted as the thick solid line. The stippled area represents the motor strip. The broken line represents the approximate demarcation of the blood flow territorial distributions for the anterior and middle cerebral arteries. After dural opening and reflection, the surgeon slips the electrodes under the edge of the craniotomy into the subdural space and places them directly on the cortical surface. The electrodes are held in place with a cotton pledget. Note that channel 2 is close to the arterial borderzone of the anterior cerebral artery. The lead placement shown was actually used in case 2. For that case the posterior lead is channel 1 and the anterior lead is channel 2 (fig. 4).

The neck of the aneurysm was difficult to dissect and had to be freed from the top of the cavernous sinus. During this dissection the blood pressure spontaneously decreased from 130/70 to 110/60. The EEG began to show a loss of higher frequency activity, consistent with cerebral ischemia (fig. 2). At this time a phenylephrine infusion was begun and the blood pressure increased to 170/90. There was a prompt reversal of the ischemic EEG changes. After the aneurysm was clipped, the carotid artery was reopened. During closure and emergence, the blood pressure was kept at 150/80 and there were no further EEG events. A summary of EEG and systemic blood pressure changes is shown in figure 3.

Postoperative neurologic examination was normal, and postoperative angiography demonstrated complete obliteration of the aneurysm and a patent carotid artery. Three months later normal visual acuity had returned.

Case 2. A 58-yr-old man with a 31-yr history of epilepsy treated with phenytoin and phenobarbital presented with increasingly frequent seizures over several months. A CT scan demonstrated a giant aneurysm in the region of the left middle cerebral artery (MCA) bifurcation. On admission, the patient was neurologically normal with a blood pressure of 130/80. Angiography confirmed the presence of an aneurysm, 2.2 cm in largest diameter, located at the first bifurcation of the left MCA.

The patient was taken to the operating room where a left frontotemporal craniotomy was performed. Anesthesia was induced with midazolam, thiopental, vecuronium, and isoflurane and maintained with 0.75% isoflurane in 3:2 nitrous oxide and oxygen. The Pa_{CO_2} was maintained at 28–30 mmHg. The patient received 1 g/kg of mannitol. Two strips of cortical EEG electrodes were applied to the lateral surface of the frontal lobe near the motor strip (fig. 1).

The patient's blood pressure was 130/70 at this time. The Sylvian fissure was split and the aneurysm was mobilized from its arachnoid attachments. This permitted identification of the proximal middle cerebral artery (M1), its two major branches, and the neck of the aneurysm. The blood pressure was increased to 170/90 with a phenylephrine infusion and a temporary clip was placed across the M1 segment, proximal to the aneurysm. Within 10 s, the posterior EEG became nearly isoelectric (fig. 4). The clip was removed with subsequent recovery of EEG activity within 10 s. Because this trial occlusion had "failed," a titration of thiopental sufficient to result in burst suppression was begun (400 mg). The blood pressure decreased from 170/90 to 150/80 with thiopental administration.

The aneurysm was then isolated by placing temporary clips on the middle cerebral artery and each of the two major distal MCA branches. The blood pressure during these maneuvers was 150/80. The aneurysm was deflated and a permanent clip was placed across the neck of the aneurysm. Further dissection of the aneurysm indicated that the clip did not completely occlude the neck; therefore, a second permanent clip was passed just distal to the initial clip.

At this point the EEG burst–suppression pattern had begun to recover (fig. 4*B*). Twenty minutes after placement of the first clip, slightly different burst–suppression ratios for the two sets of frontal electrodes and different burst amplitudes were evident. After an initial inspection of the clip placement, it appeared that the clip was properly positioned. However, persistence of the asymmetric return of electrical activity prompted re-inspection of the initial clip, and it was found to be partially occluding one of the main branches of the middle cerebral artery. The offending permanent clip was repositioned and within several minutes the EEG became symmetric (fig. 4*C*).

Postoperatively, the patient was neurologically normal. He was slightly somnolent and this was attributable to the intraoperative dose of thiopental. The patient was discharged home on the sixth postoperative day.

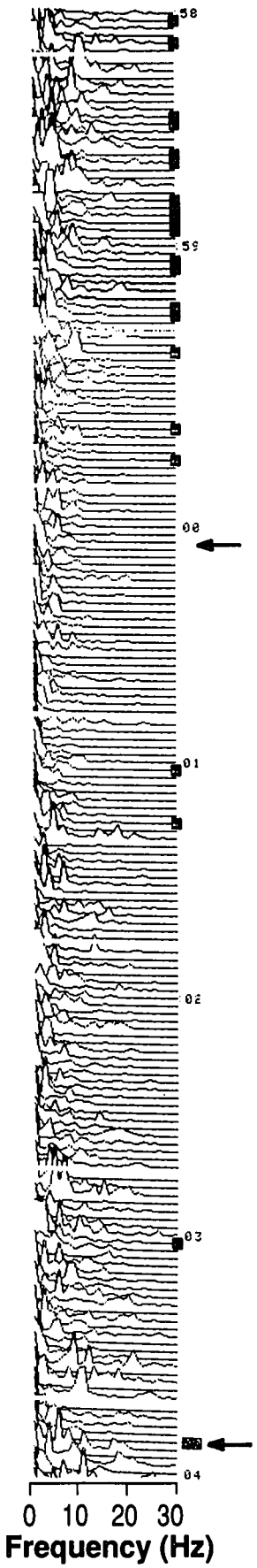

FIG. 2. Compressed spectral array tracing obtained during ischemic episode during case 1. Each line represents a 2-s epoch across a frequency spectrum of 0–30 Hz. The numerals to the right of the CSA trace are minute markers (relative to internal clock of the CSA device). The carotid occlusions took place prior to the beginning of the tracing shown. A gradual loss of higher frequency power can be seen, reaching a nadir toward the end of minute marker 00. At this time phenylephrine infusion was begun (top arrow), which brought about a reversal of the EEG changes, almost completely by minute marker 03. The cervical and intracranial carotid arteries were unoccluded after successful clipping of the aneurysm neck (*bottom arrow*).

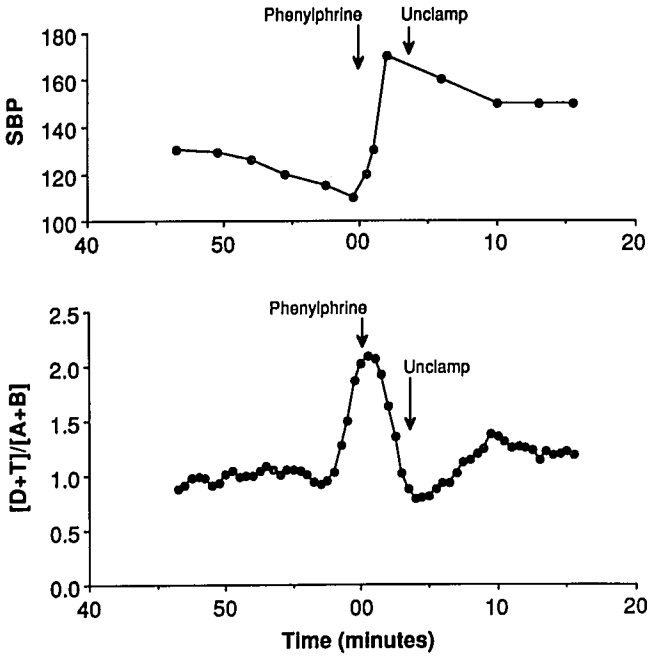

FIG. 3. Relationship of systemic blood pressure to ischemic EEG changes during case 1. Time in minutes is on the x-axis, corresponding to the CSA tracing in figure 2. Systemic arterial blood pressure (mmHg) is on the y-axis and is shown in the upper curve. The lower curve is the ratio of delta plus theta to alpha plus beta EEG activity ([D + T]/[A + B]), in relative units normalized to baseline conditions. The blood pressure drifted spontaneously from 130/70 to 110/60 with an abrupt rise in the ratio of ([D + T]/[A + B]). A phenylephrine infusion was begun and the pressure increased to 170/90 with prompt reversal of EEG changes. The cervical and intracranial carotid arteries were unoccluded after clipping of the aneurysm neck.

DISCUSSION

These two cases illustrate the feasibility of using cortical EEG monitoring in the setting of aneurysm clipping where EEG information can be used to guide the conduct of blood pressure augmentation during periods of temporary vessel occlusion and to assess a "trial occlusion" of a major vessel and then be used to follow the course of pharmacologic brain protective therapy.

Temporary occlusion techniques require some modification of the traditional anesthetic management of cerebral aneurysm clipping. During temporary vascular occlusion of a major intracranial artery, not only must systemic hypotension be avoided but blood pressure augmentation may also be useful. Some form of monitoring for cerebral ischemia such as EEG is desirable, although there is no clear consensus on the best methods or the reliability of the information gained. In the series of Jones *et al.*,[3] four of 21 patients suffered new postoperative neurologic deficits after aneurysm clipping under controlled hypotension, even though the EEG showed no sustained changes in pattern intraoperatively. They ascribed this failure of scalp EEG to predict neurologic outcome to 1) an etiology of deficit due to something other

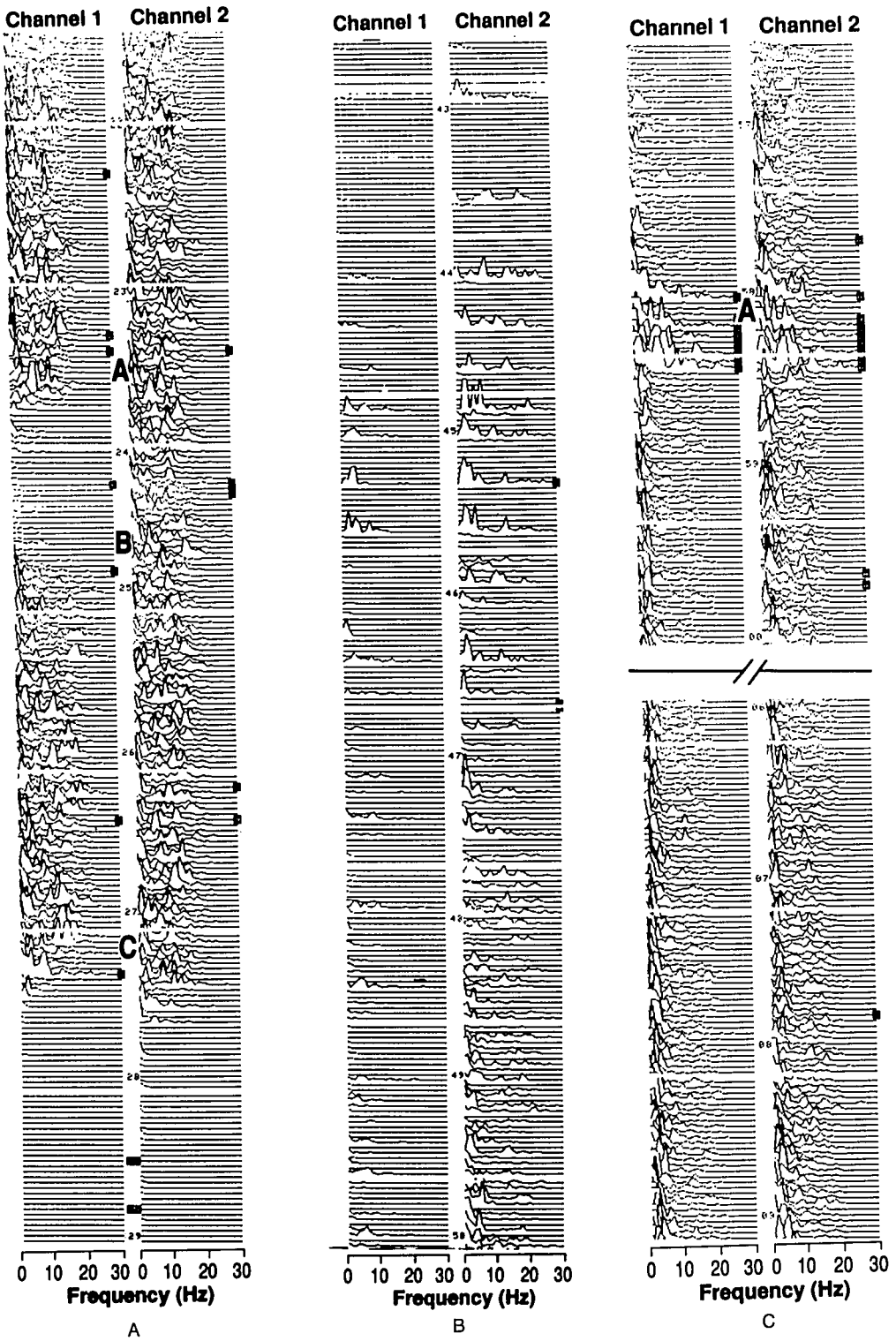

FIG. 4. The following three compressed spectral array tracings (CSA) were obtained from case 2. Channel 1 is posterior and channel 2 in anterior as depicted in figure 1. Each line represents a 2-s epoch across a frequency spectrum of 0–30 Hz. The numerals between CSA channels are minute markers. *A.* A trial occlusion of the main branch of the middle cerebral artery was done at the point indicated as A. The decrease in EEG power in the posterior lead, and not the more anterior one which was only several centimeters away, was probably due to collateral irrigation of the cortex under the anterior lead from the anterior cerebral artery distribution. At B the artery was opened followed by a rapid return to the baseline EEG pattern within 20–30 s. At C 400 mg thiopental was given. The two dark bars between CSA channels represent the times at which the proximal and distal middle cerebral arteries were occluded to isolate the aneurysm's blood supply. Note that this trace ends at minute 29. The middle cerebral artery was opened at approximately minute marker 34. *B.* This is a continuation of the CSA shown in figure 4*A*, beginning just after minute 42. The thiopental effect has begun to dissipate, with appearance of a burst–suppression pattern. However, the burst pattern in channel 1, the one previously ischemic during test occlusion as seen in the previous panel, is of considerably lower amplitude than in channel 2. By minute marker 46, it is becoming evident that both the amplitude and frequency of the burst–suppression pattern is asymmetric. By minute marker 48–50, the asymmetry is unequivocal. The surgeons were informed, and initial examination of the clip placement showed good occlusion of the aneurysm neck. *C.* The continued EEG asymmetry prompted a careful look at clip placement. One of the permanent clips across the neck of the aneurysm was found to be causing a partial occlusion of one of the main middle cerebral artery branches. The clip was repositioned starting at minute 56–57 and completed at A. Almost immediately, the asymmetry began to resolve. By minute marker 06, the EEG is again symmetric.

than hypotension (*i.e.*, edema, spasm) or 2) lack of information from the area at risk, specifically the operative field, because craniotomy may require placement of scalp electrodes at some distance from ischemic brain areas.

Surgical access to an aneurysm is greatly facilitated by a combination of techniques that produce brain relaxation, including anesthetics that are not cerebral vasodilators, modest hypocapnia, osmotherapy, spinal drainage, and careful head positioning. The result of these maneuvers is to introduce a large air space between the dura and the arachnoid and to increase the distance between the scalp electrodes and the underlying cerebral cortex on the nondependent side of the head. This causes attenuation and distortion of the EEG signal. In addition, there is a preferential shunting of EEG activity across the scalp from more distal cortical sites that are closer to the inner table of the skull, producing a signal that does not originate from the immediate underlying cortex.[6] These problems are overcome by using electrodes placed directly on the cerebral cortex. Such cortical EEG electrodes are commonly used for localization of epileptogenic foci in the surgical management of epilepsy.[7]

The MCA territory, which includes the lateral surfaces of the frontal and temporal lobes, forms the region at greatest risk for ischemia during temporary occlusion of the vessels in the anterior (*i.e.*, carotid) circulation. Cortical electrodes are easy to place on these surfaces after pterional craniotomy. The placement of cortical EEG electrodes can be accomplished safely and does not interfere with surgical access. Thus, direct cortical EEG monitoring is advantageous as a relatively sensitive means of assessing cerebral cortex at risk for ischemia during temporary vascular occlusion. Because the majority of currently available EEG monitors available for use by anesthesiologists in the operating room have only 2–4 leads, a montage that monitors the regions at highest risk for ischemia is appealing. The time-consuming process of placing a full complement of scalp electrodes is not widely applied in a busy practice.

A disadvantage of both cortical and scalp electrodes is the lack of information about ischemia resulting from interruption of blood supply by small perforating arteries that may supply deep structures, such as the internal capsule and basal ganglia. Although highly sensitive, the use of cortical EEG electrodes in the manner presented is not absolutely reliable as a specific indication of cortical ischemia because it only supplies information about those few specific brain regions monitored. There may be discrete areas not monitored that experience critical reductions in cerebral blood flow during vessel occlusion. If desired, a small switching device could be used to periodically monitor between different electrode arrays, increasing the size of the cortical sample from which EEG is obtained. Ideally, one would utilize a complement of scalp electrodes in addition to a larger cortical electrode array to circumvent some of the stated disadvantages of using the electrode array described in this report. This would also allow comparison of hemispheric data for analysis of side-to-side asymmetry.

Some additional points are worthy of mention regarding the influence of the information gained from EEG monitoring on patient management. The first case demonstrates the importance of maintaining adequate cerebral perfusion pressure in the face of temporary vascular occlusion and the usefulness of blood pressure augmentation to treat the development of cerebral ischemia. This has implications for patients with simple hemodynamic ischemia from vascular occlusion as well as in the treatment of patients with symptomatic vasospasm from aneurysmal subarachnoid hemorrhage,[8] who may have impaired autoregulatory capacity.

Augmentation of systemic arterial pressure enhances flow to ischemic regions of brain that have lost the ability to autoregulate to changes in perfusion pressure.[9–11] It is common to observe improvement in ischemic signs and symptoms during blood pressure augmentation in the awake patient.[10] However, under anesthesia one must resort to some form of cerebral function monitoring to help guide blood pressure management. This is analogous to temporary arterial occlusion during carotid endarterectomy where the EEG may guide the decision to employ temporary bypass shunting.

In the first case, the patient's arterial blood pressure was maintained at a level we believed was sufficient to ensure adequate cerebral perfusion. The blood pressure decreased spontaneously to a level that, had we not had the benefit of the EEG, may have been clinically acceptable. With the aid of the EEG, we were able to successfully increase MAP to treat the developing cerebral ischemia (figs. 2 and 3). In this case, fortunately, it was possible to unocclude the carotid artery shortly after induced hypertension corrected the ischemic EEG changes. However, it is not unlikely that a similar set of circumstances could arise at a time when it would not be advantageous to unocclude the vessel, *i.e.*, during a difficult approach to the neck of the aneurysm. Therefore, use of adequate EEG monitoring permits diagnosis of an ischemic event followed by appropriate therapeutic action.

Barbiturates are effective cerebral protective agents when used in the setting of focal cerebral ischemia.[12] Use of pharmacologic protection during intracranial vascular occlusion in humans has been described by several authors using thiopental[13] and etomidate,[1] although no randomized clinical studies have documented the efficacy of such therapy. In the second case, there was a near-complete loss of the EEG power in the posterior lead, despite moderate blood pressure augmentation. We therefore elected to induce EEG suppression with the addition of thiopental

to the isoflurane anesthetic after a trial occlusion of the MCA resulted in EEG evidence of ischemia. An interesting finding in this case was the fact that there was a significant asymmetry in the return from burst suppression between the two cortical EEG electrode strips (fig. 4*B*). The asymmetry in the two EEG signals prompted the surgeon to examine the placement of the clip, which was otherwise considered to be properly positioned. The finding that a major MCA branch was kinked led to correction of clip placement that might have otherwise resulted in a profound neurologic deficit from MCA stroke. One of the perceived disadvantages of using barbiturates or etomidate for cerebral protection is that a suppressed EEG is not generally considered to be useful as an indicator of ischemia. The findings in the second case suggest that asymmetry between two previously symmetric regions, in terms of return from burst suppression, is likely to be a result of cerebral ischemia.

Temporary vascular occlusion during cerebral aneurysm clipping is becoming a standard management option. Based on our experience with these initial cases, we conclude that cortical EEG monitoring is a useful adjunct to monitoring patients undergoing temporary vascular occlusion during the course of aneurysm clipping.

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