Use of computerized electroencephalographic monitoring during aneurysm surgery

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The clinical usefulness of intraoperative electroencephalographic (EEG) monitoring of cerebral perfusion during aneurysm surgery has received little attention, primarily due to problems with electrode placement over the operative site, which is the area of maximal risk. In this report, 27 patients undergoing surgery for anterior circulation aneurysms were monitored intraoperatively with a two-channel computerized EEG complex using a bilateral frontal-occipital montage. In 16 patients, a normal EEG pattern was observed throughout surgery; all 16 awoke neurologically intact and their postoperative angiograms did not reveal cerebral vasospasm. In the other 11 patients, one of two patterns of persistent EEG abnormalities was identified. 1) In six of these patients a marked attenuation of EEG activity was observed ipsilaterally which coincided with various intraoperative events including brain retraction, hypotension, and aneurysm dissection/clipping. Five of these six patients awoke with new neurological deficits which persisted beyond 12 hours in two, both of whom had angiographically proven vasospasm 24 hours after surgery. 2) In the remaining five patients, a distinct abnormal EEG pattern consisting of marked hyperactivity in the delta, theta, and alpha frequency ranges was observed ipsilaterally. Four of these five patients had a poor neurological outcome and vasospasm on their angiogram 24 hours after surgery. Thus, EEG monitoring which spans the operative area during aneurysm surgery is practicable and appears to be of value in the detection of compromised cerebral perfusion during aneurysm surgery. The possible significance of the two abnormal EEG patterns identified in this report is discussed.

KEY WORDS - cerebral ischemia - computerized electroencephalography - vasospasm - aneurysm

Cerebral ischemia during aneurysm surgery continues to be a major cause of morbidity and mortality in the perioperative period. Early recognition and therapy of compromised cerebral perfusion has been shown to be the most important factor in the recovery from cerebral ischemia.10,11,23 Unfortunately, during general anesthesia for aneurysm surgery, cerebral blood flow (CBF) is not routinely monitored. Thus, some patients will awaken from surgery with a new focal neurological deficit due to undetected intraoperative cerebral ischemia from a variety of causes such as brain retraction, cerebral vasospasm, surgical damage to a small perforating vessel or major artery, or hypotension.18,25

Electroencephalographic (EEG) monitoring with conventional eight- to 16-channel apparatus is a reliable means of evaluating cerebral perfusion during carotid endarterectomy (CEA),21,24,26 cardiopulmonary bypass procedures,22 and controlled hypotension.1,6 Intraoperative EEG monitoring during aneurysm surgery has received little attention, however, primarily due to problems with electrode placement, particularly over the operative region.6

Recent advances in electronic technology have simplified the use of intraoperative EEG monitoring. Commercially available computerized monitors that process the EEG signal into a concise form utilizing fast-Fourier analysis have been developed to simplify the recognition and interpretation of EEG changes during anesthesia.2,28 Several studies have shown that these one- to four-channel monitors, which require fewer electrodes for proper use, are reliable indicators of significant reductions in CBF during CEA,2,15,16,20 cardiopulmonary bypass procedures,14,16 and cardiac arrest.28 These findings and the advantages associated with computerized EEG monitoring led us to the routine use of a two-channel monitor intraoperatively in an effort to minimize the drawbacks and increase the practicality of real-time EEG monitoring during aneurysm surgery. This report summarizes our findings with regard to the
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<table>
<thead>
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<th>TABLE 1</th>
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<tr>
<td>Characteristics of 27 patients monitored by computerized EEG</td>
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<td>during aneurysm surgery*</td>
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<td>Factor</td>
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<td>-------------------------------</td>
</tr>
<tr>
<td>mean age (yrs)</td>
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<tr>
<td>age range (yrs)</td>
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<tr>
<td>sex (M:F)</td>
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<tr>
<td>cases with SAH</td>
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<tr>
<td>mean interval from SAH to surgery (days)</td>
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<tr>
<td>vasospasm prior to surgery</td>
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<tr>
<td>Hunt &amp; Hess grade (at surgery):</td>
</tr>
<tr>
<td>Grade 0</td>
</tr>
<tr>
<td>Grade I</td>
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<tr>
<td>Grade II</td>
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<tr>
<td>Grade III</td>
</tr>
<tr>
<td>aneurysm location</td>
</tr>
<tr>
<td>posterior communicating artery</td>
</tr>
<tr>
<td>internal carotid artery</td>
</tr>
<tr>
<td>anterior communicating artery</td>
</tr>
<tr>
<td>middle cerebral artery</td>
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<tr>
<td>posterior cerebral artery</td>
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</table>

* EEG = electroencephalography, SAH = subarachnoid hemorrhage.

early detection and therapy of intraoperative cerebral ischemia due to vasospasm and other causes.

Clinical Material and Methods

Patient Population

Computerized EEG monitoring was performed during intracranial surgery for anterior circulation aneurysms in 27 patients treated at the Washington University Medical Center in St Louis. Twenty-four of the 27 patients suffered a subarachnoid hemorrhage (SAH) prior to admission. The characteristics of these patients are summarized in Table 1. Preoperative clinical grading was performed according to the scale of Hunt and Hess. All patients underwent a complete neurological examination prior to surgery, in the immediate postoperative period, and at the time of discharge from the hospital. In addition, cerebral angiographic studies were performed 24 to 48 hours prior to surgery and in the postoperative period. The majority of patients who awoke from surgery with a new deficit had an angiographic study repeated within the 1st postoperative day, while those who were neurologically intact had a repeat angiogram performed 3 to 4 days postoperatively. Angiograms were assessed for the presence of cerebral vasospasm, for patency of intracranial vessels, and for obliteration of the aneurysm.

Anesthesia

A deep level of surgical anesthesia was induced with etomidate and fentanyl. Endotracheal intubation was performed after complete neuromuscular blockade was achieved with atracurium. Maintenance of anesthesia included: delivery of nitrous oxide (60%) in oxygen, fentanyl infusion, administration of low-concentration isoflurane (< 0.25%), and atracurium infusion. Ventilation was mechanically controlled, and PaCO₂ was maintained between 26 and 30 mm Hg. Direct systemic blood pressure recordings were obtained via radial arterial cannulas. Volume status was assessed using a pulmonary artery or central venous pressure catheter, and normovolemia or modest hypervolemia was maintained throughout surgery. Controlled hypotension, when indicated, was induced with low-dose sodium nitroprusside.

Electroencephalography

Prior to induction of anesthesia, a commercially available computerized EEG monitor with compressed spectral assay (CSA) and power band (PB) histogram capabilities was employed. This monitor displays two channels (left and right cerebral hemispheres) of raw EEG, CSA, and PB data (Fig. 1) recorded from five subdermal fine-gauge platinum needle electrodes placed in a symmetrical frontal-occipital montage as follows. A group electrode was placed in the middle of the forehead just superior to the nasion. Channels 1 and 2 were connected by their positive (G2) terminal to left and right electrodes, respectively, placed on each side of the forehead at the lateral brow. In addition, these two channels were connected by their negative (G1) terminal to left and right electrodes, respectively, placed on or posterior to the mastoid. Interelectrode distance was at least 10 cm and interelectrode impedance was 3 kOhms or less. In order to produce satisfactory tracings, the sensitivity was adjusted to between 30 and 80 µV. After the patients were positioned for the routine frontotemporal flap incision, tincture of benzoin was applied to the skin, and the electrodes were protected from the surgical preparation solution with transparent adhesive dressing. Acceptable impedance was reconfirmed after completion of surgical draping and intermittently throughout the procedure.

Throughout each surgical procedure, a continuous on-line two-channel raw EEG tracing was obtained with a simultaneous printout of the CSA plus intermittent recordings of the PB histogram. Baseline tracings were obtained prior to and soon after induction of anesthesia. With the use of fast-Fourier analysis, raw EEG data over a 2-second epoch is digitized to be separated into its component sine waves. Squaring the amplitudes of the individual sine waves generates the power spectrum plotted for each channel as a PB histogram in each of the four classical frequency bands: delta (1–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), and beta (13–30 Hz) (Fig. 1). The CSA display mode presents the power spectrum concisely in the form of a three-dimensional graph. With frequency as the x-axis, successive 2-second epochs of spectral analysis are represented as vertically

* EEG monitor manufactured by Interspec-Neurotrac, Conshohocken, Pennsylvania.
† Electrodes manufactured by Grass Instrument Co., Quincy, Massachusetts.
FIG. 1. Normal electroencephalographic (EEG) pattern under anesthesia in a 56-year-old woman with a left middle cerebral artery aneurysm. The tracings were recorded soon after clipping. Channel 1 = ipsilateral (operative) hemisphere; Channel 2 = contralateral hemisphere; sensitivity = 80 μV.

A: Normal symmetrical raw EEG tracings under anesthesia.

B: Normal symmetrical compressed spectral array tracings under anesthesia. The frequency range is 1 to 30 Hz. The black boxes represent the spectral edge frequency (the highest significant frequency present in the current EEG spectrum).

C: Normal symmetrical power band histograms under anesthesia. Total power is 125 picowatts (pW) in Channel 1 and 122 pW in Channel 2. The numbers “1” and “2” below each band represent Channels 1 and 2 in each of the four frequency ranges studied. The numbers above each band indicate the power in each frequency range for either channel. In this case, power was primarily in the theta frequency range with some delta and alpha contributions.

Results

Electroencephalographic Patterns

Three EEG patterns were observed, defined as follows. 1) The normal EEG pattern (Fig. 1) included symmetrical EEG tracings consistent with the awake and anesthetized states. The awake EEG was composed of predominantly fast activity (20 Hz). The EEG pattern under anesthesia consisted of predominantly theta activity with some delta and alpha contributions (10 Hz). 2) Attenuation of the EEG activity pattern (Fig. 2) consisted of a greater than 33% decrease of amplitude and power in the ipsilateral (dependent) hemisphere, with or without a decrease in SEF. 3) The EEG hyperactivity pattern (Fig. 3) showed a greater than 50% increase of amplitude and power in the ipsilateral (dependent) hemisphere, predominantly in the delta, theta, and alpha frequency ranges.

EEG Patterns Correlated with Neurological Outcome

The results are summarized in Fig. 4. Sixteen of the 27 patients monitored had normal EEG patterns throughout surgery. All 16 awoke from anesthesia neurologically intact, and postoperative angiography performed in all 16 patients did not reveal any evidence of cerebral vasospasm. Preoperatively, 13 of these 16 patients had been classified in clinical Grade 0 or I according to the Hunt and Hess scale.

In the remaining 11 patients, one of the two patterns of persistent EEG abnormalities defined above was identified. Preoperatively, eight of the 11 patients had been classified in either Grade II or III. These 11 cases are summarized in Table 2 and were categorized as follows:

In six of the 11 patients, the attenuation of EEG activity pattern was noted in the ipsilateral hemisphere. None of the six had either focal deficits or angiographic evidence of vasospasm in the immediate preoperative period. In three of these six patients, the onset of this EEG abnormality occurred during dissection and aneurysm clipping. The EEG tracing did not recover throughout the remainder of surgery in two of these cases: both patients (Grade III), who were drowsy prior to surgery, awoke confused with new focal deficits which persisted postoperatively. Angiography 24 hours after surgery revealed vasospasm in both. One patient recovered gradually over 1 week, while the other suffered a permanent deficit. In the third patient, the
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**Fig. 2.** Attenuation of electroencephalographic (EEG) activity pattern in a 38-year-old woman with a right posterior communicating artery aneurysm. The tracings were recorded soon after clipping. Channel 1 = ipsilateral (operative) hemisphere; Channel 2 = contralateral hemisphere; sensitivity = 60 µV. For further description see legend to Fig. 1. A: EEG voltage attenuation in Channel 1 postaneurysm clipping. B: Compressed spectral array tracings demonstrating loss of amplitude in Channel 1 without change in spectral edge frequency. C: Power band histograms showing a 50% decrease of the total power in Channel 1 (49 picowatts (pW)) compared to Channel 2 (98 pW) distributed equally in the delta, theta, and alpha frequency ranges.

**Fig. 3.** Electroencephalographic (EEG) hyperactivity pattern in a 42-year-old woman with a right posterior communicating artery aneurysm. The tracings were recorded soon after clipping. Channel 1 = ipsilateral (operative) hemisphere; Channel 2 = contralateral hemisphere; sensitivity = 60 µV. For further description see legend to Fig. 1. A: EEG tracings demonstrating a large voltage increase in Channel 1 compared to Channel 2. B: Compressed spectral array tracings revealing a marked amplitude increase in the delta, theta, and alpha frequency ranges of Channel 1 compared to Channel 2. C: Power band histograms showing a greater than 50% increase of the total power in Channel 1 (218 picowatts (pW)) compared to Channel 2 (105 pW) distribution primarily in the delta, theta, and alpha frequency ranges.
PATIENTS UNDERGOING ANEURYSM SURGERY (N = 27)

NORMAL EEG (N = 16)
- No neuro deficit
- Angio-postop: no spasm

ATTENUATION OF EEG ACTIVITY PATTERN (N = 6)
- (N = 3) AFTER ANEURYSM CLIPPING:
  - (n = 1) Permanent neuro deficit
  - Angio-postop: spasm
  - (n = 1) Neuro deficit recovered gradually
  - Angio-postop: spasm
  - (n = 1) Vasospasm visualized in operative field
  - Recovery of EEG with volume expansion
  - Neuro deficit recovered in 24 hours
  - Angio-postop: refused

- (N = 2) DURING HYPOTENSION:
  - Recovery of EEG with normotension
  - Neuro deficit recovered in 24 hours
  - Angio-postop: no spasm

- (N = 1) DURING BRAIN RETRACTION:
  - Recovery of EEG after retractor repositioning
  - No neuro deficit
  - Angio-postop: no spasm

EEG HYPERACTIVITY PATTERN (N = 5)
- (N = 3) PRIOR TO INDUCTION OF ANESTHESIA AND THROUGHOUT SURGERY:
  - Angio-preop: spasm
  - Neuro status unchanged postop with 2 permanent deficits and one death
  - Angio-postop: spasm

- (N = 2) AFTER ANEURYSM CLIPPING:
  - (n = 1) Permanent neuro deficit
  - Angio-postop: spasm
  - (n = 1) No neuro deficit
  - Cardiac arrest postop resuscitated successfully
  - Angio-postop: refused

Fig. 4. Summary of perioperative electroencephalographic (EEG) recordings, postoperative angiographic findings, and clinical outcome in all 27 patients in this series.

Attenuation of EEG activity coincided with vasospasm of the left internal carotid artery (ICA) observed by the surgeon during dissection. Induced hypotension was avoided and the aneurysm was clipped, after which augmentation of cardiac output with intravascular volume expansion was instituted. The EEG tracing slowly recovered within 90 minutes to findings comparable with those recorded prior to the onset of attenuation. The patient awoke with a new right hemiparesis which resolved in 12 hours. Postoperative angiography was refused.

In the other three of the six patients whose intraoperative EEG demonstrated ipsilateral attenuation of EEG activity pattern, the onset of this EEG abnormality coincided with certain intraoperative events. In two of these three cases, just prior to aneurysm clipping, the attenuation of EEG activity was associated with hypotension due to aneurysmal bleeding in one case and pharmacologically induced hypotension in the other. Normal blood pressure was restored within 10 minutes in both patients, and their EEG tracings slowly recovered within 20 and 60 minutes, respectively, to findings comparable with those recorded prior to the onset of attenuation. Both awoke with a transient deficit which recovered in 6 and 12 hours, respectively. In the third patient, the attenuation of activity was detected during application of a brain retractor. Her EEG tracing rapidly recovered to baseline within 10 minutes after the retractors were repositioned and she awoke neurologically intact. Angiography performed 3 to 4 days postoperatively did not reveal vasospasm in any of these three patients.

In the remaining five of the 11 patients with persistent intraoperative EEG abnormalities, the EEG hyperactivity pattern was noted in the hemisphere ipsilateral to the disorder. Angiographic evidence of vasospasm was demonstrated 24 to 48 hours prior to surgery in three of the five cases. In these three patients, this pattern of EEG hyperactivity was noted prior to anesthetic induction and remained unchanged throughout surgery. In one Grade II patient clipping was not performed due to severe brain swelling which precluded aneurysm exposure. He awoke without deficit, but 10 hours postoperatively his condition worsened rapidly. A repeat angiogram 24 hours later revealed severe vasospasm. He died 11 days later from aneurysm rebleeding. In the other two patients, both impaired neurologically prior to surgery (Grade III), the postoperative neurological examinations and angiograms 24 hours after surgery were unchanged.

In the remaining two patients, whose intraoperative EEG tracings demonstrated the hyperactivity pattern,
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**TABLE 2**

<table>
<thead>
<tr>
<th>Preoperative Status</th>
<th>Location of Aneurysm</th>
<th>Surgery</th>
<th>EEG Pattern</th>
<th>EEG Pattern at End of Surgery vs. Baseline</th>
<th>Postoperative Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>38, F; Grade III</td>
<td>rt PCoA: SAH, no spasm</td>
<td>uneventful clipping</td>
<td>ipsilateral attenuation during dissection/ clipping</td>
<td>persistent attenuation</td>
<td>confused, new rt monoparesis; angiogram: spasm; recovered gradually over 1 wk</td>
</tr>
<tr>
<td>31, M; Grade III</td>
<td>lt PCoA: SAH, no spasm</td>
<td>uneventful clipping</td>
<td>ipsilateral attenuation during dissection/ clipping</td>
<td>persistent attenuation</td>
<td>alert, new lt hemiparesis; angiogram: spasm; persistent deficit at discharge</td>
</tr>
<tr>
<td>33, F; Grade II</td>
<td>lt PCoA: SAH, no spasm</td>
<td>lt ICA spasm visualized during dissection</td>
<td>ipsilateral attenuation during dissection</td>
<td>recovered within 90 min with volume expansion</td>
<td>alert, new lt hemiparesis; recovered in 12 hrs with maintenance of volume expansion; angiogram: refused</td>
</tr>
<tr>
<td>36, M; Grade I</td>
<td>rt ICA: SAH, no spasm</td>
<td>hypotension due to aneurysm bleeding</td>
<td>ipsilateral attenuation</td>
<td>recovered within 20 min with normotension &amp; volume expansion</td>
<td>obtunded, new lt monoparesis; recovered in 12 hrs; angiogram: no spasm</td>
</tr>
<tr>
<td>56, F; Grade 0</td>
<td>rt ICA: no spasm</td>
<td>controlled hypotension</td>
<td>ipsilateral attenuation</td>
<td>recovered within 60 min with normotension &amp; volume expansion</td>
<td>alert, no deficit; angiogram: no spasm</td>
</tr>
<tr>
<td>58, F; Grade I</td>
<td>rt MCA: SAH, no spasm</td>
<td>difficult brain retraction</td>
<td>ipsilateral attenuation</td>
<td>recovered within 10 min after retractor repositioning</td>
<td>persistent hyperactivity</td>
</tr>
<tr>
<td>58, F; Grade III</td>
<td>lt ICA: SAH, spasm</td>
<td>uneventful clipping</td>
<td>ipsilateral hyperactivity preanesthesia</td>
<td>obtunded, persistent impairment of cognitive function at discharge; angiogram: spasm</td>
<td></td>
</tr>
<tr>
<td>14, M; Grade II</td>
<td>lt ICA: SAH, spasm</td>
<td>brain swelling, clipping not attempted</td>
<td>ipsilateral hyperactivity preanesthesia</td>
<td>persistent hyperactivity</td>
<td>drowsy, no focal deficit; angiogram: spasm; expired 11 days later from rebleeding</td>
</tr>
<tr>
<td>49, F; Grade III</td>
<td>rt PCoA: SAH, spasm</td>
<td>uneventful clipping</td>
<td>ipsilateral hyperactivity preanesthesia</td>
<td>persistent hyperactivity</td>
<td>confused, lt hemiparesis; angiogram: spasm; persistent deficit at discharge</td>
</tr>
<tr>
<td>42, F; Grade III</td>
<td>rt PCoA: SAH, no spasm</td>
<td>uneventful clipping</td>
<td>ipsilateral hyperactivity during dissection/ clipping</td>
<td>persistent hyperactivity</td>
<td>confused, new lt hemiparesis; angiogram: spasm; persistent deficit at discharge</td>
</tr>
<tr>
<td>28, F; Grade II</td>
<td>rt PCoA: SAH, no spasm</td>
<td>uneventful clipping</td>
<td>ipsilateral hyperactivity during dissection/ clipping</td>
<td>persistent hyperactivity</td>
<td>alert, no deficit, followed by sudden cardiac arrest (successfully resuscitated); angiogram: refused</td>
</tr>
</tbody>
</table>

* EEG = electroencephalographic; SAH = subarachnoid hemorrhage; PCoA = posterior communicating artery; ICA = internal carotid artery; MCA = middle cerebral artery.

the onset of this pattern occurred soon after aneurysm clipping and persisted throughout the remainder of surgery. One patient awoke with a new left-arm monoparesis which did not improve postoperatively. His angiogram, performed 24 hours after surgery, revealed severe vasospasm. The second patient, extubated and neurologically intact, suffered a sudden cardiac arrest precipitated by ventricular fibrillation 1 hour after surgery from which she was successfully resuscitated. This patient, who refused a repeat angiogram, did well postoperatively and was discharged neurologically intact.

**Discussion**

Operations for intracranial aneurysms carry risk to the perfusion of the neural tissue supplied by the aneurysm-bearing artery. With or without vasospasm, SAH causes significant decreases in CBF and cerebral metabolic rate of oxygen. This, therefore, CBF in these patients may be both pressure- and volume-dependent, because autoregulation is impaired. In addition to this possible alteration in autoregulation, there are other intraoperative events which can further increase the risk of neuronal ischemia. These include vasospasm, surgical damage to a small perforating vessel or major artery, brain retraction, and hypotension, either pharmacologically induced or secondary to aneurysmal rupture. The demonstration in recent years that computerized EEG monitors are reliable indicators of significant decreases in CBF during CEA and cardiopulmonary bypass prompted us to employ this technique of electrophysiological monitoring during aneurysm surgery in an attempt to increase the safety of this procedure.

The results reported here appear to indicate that computerized EEG monitoring during aneurysm surgery can be of value in the early detection of intraoperative cerebral ischemia due to various causes. Excluding the three patients with angiographically proven vasospasm preoperatively (in whom the EEG hyperactivity pattern was noted prior to anesthetic induction), 24 of...
of the 27 patients monitored had a normal EEG pattern at the onset of surgery. In 16 of these 24 cases, the EEG pattern remained normal throughout surgery and all awoke neurologically intact. In the remaining eight patients, new persistent EEG abnormalities, thought to be caused by cerebral ischemia secondary to certain events, were first detected intraoperatively. Six of these eight patients awoke with new neurological deficits. In an earlier report, Jones, et al., using conventional eight-channel EEG monitoring during aneurysm surgery, were unable to demonstrate a similar association between persistent EEG abnormalities and new postoperative neurological deficits. In their investigations, the EEG monitor failed to identify intraoperative ischemia in three of the four patients with postoperative focal deficits. Differences of EEG electrode placement may explain their high incidence of false negatives. To avoid interference with the surgical field, they restricted their electrode placement to the posterior areas of the scalp and the unoperated, contralateral side. Therefore, the area of potential maximum vulnerability, related to the aneurysm-bearing artery, was not monitored in their study. In contrast, we were able to obtain satisfactory EEG tracings using a bilateral frontal-occipital montage, which enables our EEG coverage to span the operative site.

Two patterns of persistent EEG abnormalities were identified in this report. The ipsilateral attenuation of EEG activity pattern, detected intraoperatively in six patients, appears to be similar to previously described EEG abnormalities associated with critical decreases in regional CBF during carotid cross-clamping. Thus, it is not surprising that the onset of this pattern demonstrated a temporal relationship with various intraoperative events: hypotension in two cases, brain retraction in one, and aneurysm dissection/clipping in three others. In these latter three cases, it may be possible that the EEG attenuation detected during dissection/clipping was precipitated by vasospasm, as this was observed intraoperatively in one patient and on angiography performed 24 hours later in the other two.

Interestingly, a second persistent EEG abnormality, described as an ipsilateral increase of amplitude/power in the delta, theta, and alpha frequencies, was also identified in five cases. This EEG hyperactivity pattern was first identified during aneurysm dissection/clipping in two of the patients, and prior to anesthetic induction in the three patients who were known to have angiographic vasospasm 24 to 48 hours prior to surgery. After detection, this pattern persisted throughout surgery, and four of these five patients had angiographically proven vasospasm 24 hours postoperatively. The fifth patient, without angiographic vasospasm preoperatively, awoke neurologically intact, but suffered a cardiac arrest 1 hour after surgery, from which she was successfully resuscitated. Unfortunately, this patient refused an angiogram after surgery. Life-threatening arrhythmias have been described previously in patients with SAH and are believed to be a result of sympathetic stimulation triggered by the intracranial hemorrhage. In addition, although this patient remained intact neurologically, it has been shown that ischemic symptoms develop in only approximately 50% of patients with angiographic vasospasm. In view of these previous reports, it is possible that had an angiogram been performed postoperatively, it may have also revealed vasospasm in this patient.

We can offer no ready explanation as to why two distinctly different abnormal EEG patterns were identified in this report of 11 cases of apparent intraoperative cerebral ischemia. In one study, which analyzed conventional EEG findings in patients with SAH on Days 4 and 7 prior to surgery, unilateralized high voltage slow waves were found in 85% of patients with angiographically confirmed vasospasm. Also, mild to moderate hypoxia is known to produce an increase in EEG amplitude and frequency. In another investigation that utilized CSA analysis of the EEG tracings, an asymmetrical pattern of CSA amplitude increase was described in two conscious patients with prolonged vasospasm following SAH. To our knowledge, this appears to be the only report of a computerized EEG hyperactivity pattern similar to ours. We can only suggest, based on these results, that the EEG hyperactivity pattern appears to be more indicative of vasospasm and a less favorable clinical outcome. Conversely, attenuation of EEG activity pattern is perhaps more often associated with various intraoperative causes of ischemia.

Some patients will awaken from aneurysm surgery with a new focal deficit due to undetected intraoperative ischemia. It would be expected that the intraoperative use of an electrophysiological monitor, such as an EEG system, would lead to earlier recognition and therapy which has been demonstrated to be the most important factor in recovery from cerebral ischemia. This was illustrated in four patients in the present series: attenuation of EEG activity was associated with hypotension in two, brain retraction in one, and possible vasospasm of the left ICA was observed by the surgeon in another. In all four cases, the EEG tracing demonstrated either complete or almost complete recovery to its preischemic baseline after institution of therapy (volume expansion), and the patients awoke either intact or with a short-lived deficit which resolved in less than a day. Of course, one cannot assume from these observations that patient outcome would have been different without EEG monitoring; however, it is possible that these patients may have been helped by early therapeutic action based on EEG warning signs of ischemia.

This report shows that computerized EEG monitoring is practicable during intracranial aneurysm surgery. It does not interfere with the operative field and allows monitoring of cerebral function in the region of the aneurysm-bearing artery. The evidence presented here suggests that this technique may be of help in the detection of intraoperative cerebral ischemia during
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aneurysm surgery and in some cases may allow early therapeutic intervention. Further experience is needed regarding the exact significance of the two abnormal EEG patterns identified in this report and their relationship to both angiographic findings and clinical outcome.

References


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