EEG Monitoring for Induced Hypotension for Surgery of Intracranial Aneurysms


SUMMARY EEG was monitored at bilateral scalp sites outside the operative field during hypotensive aneurysm surgery in 21 patients. Mean arterial blood pressure at axillary level was 50–60 mm Hg (average 55 mm) for 1.9–5.3 hours (average 3.6). Four new deficits were noted immediately post-operatively, all related to the operated site: these were attributable to intra-operative rupture with forced vascular clipping, vasospasm, or edema. In no instance was hypotension solely responsible for a new deficit. EEG showed persistent slowing in relation to surgery in only 1 patient, where aneurysmal rupture led to severe hypotension, forced clipping of 1 posterior cerebral artery, and subsequent brain stem infarction. In the 3 other patients with fresh focal postoperative deficits, no persistent intraoperative EEG changes were observed. EEG monitoring did not detect ischemia in these 3 patients because 1) hypotension was moderate and did not per se cause new deficit, and 2) EEG electrodes did not survey the area at maximal risk, namely the operative field. EEG scalp electrodes near but outside the operative site do not seem useful for monitoring cerebral function in the region of aneurysm surgery. Epidural or cortical electrodes in the operative field may prove to be more useful.

RUPTURE OF an intracranial aneurysm during surgery remains a threat even in the era of microsurgery. Profound controlled hypotension reduces the risk of rupture and aids dissection. Hypotension "defuses" the aneurysm by markedly decreasing wall tension. As one reduces cerebral perfusion pressure, however, the lower limit of autoregulation is approached, and risks of cerebral ischemia increase. This is particularly true when subarachnoid hemorrhage or surgical manipulation has already compromised autoregulation. Therefore, reliable techniques are needed for monitoring the CNS response to lowered blood pressure during aneurysm surgery.

Since the electroencephalogram (EEG) continuously assesses certain aspects of electrophysiologic function of the brain, this test seemed potentially useful for monitoring hypotension during aneurysm surgery. EEG monitoring during carotid endarterectomy during cardiopulmonary bypass can likewise warn of cerebral ischemia during hypotension. In a series of 75 patients undergoing cardiopulmonary bypass, intraoperative EEG accurately predicted postoperative neuropsychiatric deficits and in several instances ischemic EEG changes were reversed by raising mean arterial blood pressure. Therefore, we have studied the clinical usefulness of intraoperative EEG as a monitor of the CNS response to induced hypotension during aneurysm surgery.

Methods

Patients

Twenty-one patients undergoing aneurysm surgery were studied. Characteristics of these patients are presented in table 1. Most patients had sustained subarachnoid hemorrhage more than one week before surgery and were in good clinical condition (Botterell Grade 1).

Anesthesia

In almost all patients, after premedication with fentanyl and droperidol (Innovar), anesthesia was induced and maintained with low dose halothane (≤1%), supplemented by 50% nitrous oxide and 50% oxygen. Neuromuscular blockade was achieved with intravenous pancuronium bromide or curare. Continuous systemic blood pressure recordings were obtained in all patients via radial arterial cannulas standardized at the anterior axillary line. Arterial blood gases were sampled serially. Controlled hypotension was induced with low dose sodium nitroprusside.

Electroencephalography

The EEG was recorded by scalp electrodes applied to areas of the head not involved directly in the sur-

<table>
<thead>
<tr>
<th>Table 1 Patient Characteristics (N = 21)</th>
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<tbody>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>SAH</td>
</tr>
<tr>
<td>Interval from SAH to surgery</td>
</tr>
<tr>
<td>Spasm (at any time)</td>
</tr>
<tr>
<td>Botterell Grade I (at surgery)</td>
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From the Neurosurgical and Neurology Services, Massachusetts General Hospital, and Harvard Medical School.
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surgery. This restricted coverage to the posterior areas bilaterally and the unoperated side. Gold cup electrodes, located according to the International 10-20 system, were applied with bentonite paste and collodion-soaked gauze to the scalp prepared with pumice paste degreasing. All electrode impedances were below 5 kilohms. Both bipolar and monopolar montages were used. The monopolar reference was a neck-chest linkage (spinal process of C7 to right clavide) balanced through a 50 K potentiometer to reduce ECG artifact. The recordings were made on a Grass model 6 eight-channel instrument with a sensitivity setting of 10 microvolts/min, a bandpass of 1 to 35 or 70 Hz (approximately .2 dB down), and a paper speed of 30 mm/sec. Notations about surgical maneuvers, anesthetic changes, and blood pressure levels were made directly into the EEG recording by the technician. All recordings were analyzed independently by 2 electroencephalographers who had no prior knowledge of clinical outcomes.

Results
Hypotension and Postoperative Deficits

For the whole group, the average mean arterial blood pressure (MABP) was $55 \pm 5.43$ mm Hg during induced hypotension with an average duration of $3.6 \pm 1.72$ hours. These values were not significantly different in those 4 patients awaking with a new deficit (see table 2). In these 4 patients, deficits were related to forced vascular clipping, vasospasm, or edema.

EEG and Postoperative Deficits

Persistent EEG abnormalities were not usually correlated with postoperative deficits. Three of the 4 immediate deficits were not associated with lasting EEG changes (see table 2). Only one of the 21 intraoperative EEGs showed persistent changes indicative of ischemic damage (see table 2, patient H.C.). Continuous low voltage delta with burst suppression was registered during and after intraoperative hypotension. This patient had a precommunal posterior cerebral artery aneurysm which ruptured intraoperatively leading to brief, profound hypotension and forced clipping of posterior cerebral artery. Postoperatively, the patient was comatose with bramstem signs and left hemiplegia. During hospitalization, she improved to the point of ambulation. Her deficit seems attributable to posterior cerebral artery occlusion rather than hypotension per se.

Discussion

Drug-induced intraoperative hypotension aids the neurosurgeon in approaching intracranial aneurysms. In view of variable loss of autoregulation in patients with aneurysm, however, hypotension could unpredictably cause cerebral ischemia. A practical method is needed to monitor the CNS response to hypotensive stress. EEG successfully detects ischemia during carotid endarterectomy and cardiopulmonary bypass and, therefore, might be expected to detect ischemia during hypotensive aneurysm surgery. In addition, EEG might be expected to detect ischemia caused by various intraoperative problems, for example, vasospasm or forced vascular clipping. Though maximal EEG changes would be anticipated in the operative field itself, nearby scalp electrodes might also be expected to detect substantial ischemic events. Were EEG to detect ischemia, the surgeon could take steps to restore cerebral perfusion pressure. During hypotension, the blood pressure would be raised to restore perfusion. For vasospasm, blood pressure could likewise be raised since hypertension has been shown to improve perfusion and clinical outcome. In case of vascular clipping, the clip might be adjusted to restore vascular continuity. EEG and clinical responses would be correlated with these therapeutic adjustments.

Hypotension in this series was never the clear cut sole cause of a deficit. This experience corresponds to the absence of sequelae after hypotension noted by

<table>
<thead>
<tr>
<th>Postoperative status</th>
<th>Operation</th>
<th>Hypotension (MABP x Duration)</th>
<th>EEG</th>
<th>Postoperative status</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.C. 26F SAH, Grade I. PCoA anr., no spasm-</td>
<td>Uneventful clipping</td>
<td>$53 \text{ mm} \times 3 \text{ hours}$ ($33 \text{ mm norm}$)</td>
<td>No changes</td>
<td>Confused; R leg 6/10 power. Angio—spasm &amp; 4 mm shift. Recovered gradually</td>
</tr>
<tr>
<td>J.S. 43 M SAH, Grade I. LICA apex. anr., spasm present mild</td>
<td>Spasm of LICA</td>
<td>$62 \text{ mm} \times 6 \text{ hours}$</td>
<td>$47 \text{ mm} \times 14 \text{ min}$ ($86 \text{ mm norm}$)</td>
<td>No changes</td>
</tr>
<tr>
<td>E.P. 59F SAH, Grade I. LMCA anr., modest spasm.</td>
<td>Brain swelling required bone flap out.</td>
<td>$53 \text{ mm} \times 3 \text{ hours}$</td>
<td>$40 \text{ mm} \times 1 \text{ hour I}$ ($78 \text{ mm norm}$)</td>
<td>No change</td>
</tr>
<tr>
<td>H.C. 57F SAH, Grade I. LPFCa anr., minimal spasm.</td>
<td>Rupture forced LPCA clipping.</td>
<td>$53 \text{ mm} \times 2 \text{ hours}$</td>
<td>$42 \text{ mm} \times 2.5 \text{ hours}$ ($90 \text{ mm norm}$)</td>
<td>Persistent loss of 10-12 Hz after hypotension</td>
</tr>
</tbody>
</table>

Anr. = aneurysm. PCoA = posterior communicating artery. ICA = internal carotid artery. ACaA = anterior communicating artery. MCA = middle cerebral artery. PFCa = pre-communal posterior cerebral artery. DIC = disseminated intravascular coagulation.
Drake and colleagues in several hundred patients with Grade I aneurysm. In the present study, 4 new postoperative deficits appeared after rupture with forced vascular clipping, vasospasm, or edema. These postoperative deficits might have been exacerbated by hypotension, which was sometimes protracted.

In this study, EEG did not identify intraoperative local ischemia in 3 patients with immediate postoperative focal deficits. Several reasons may explain the failure of EEG to predict these deficits. Most importantly, infarction caused by hypotension was not identified in these patients, and thus we lacked a clear-cut hypertensive stress for detection by EEG surveillance. Moreover, standard EEG electrodes could be placed only in the area over the unoperated scalp and, therefore, the area of potential maximum vulnerability was not monitored.

EEG with scalp electrodes near but outside the surgical site does not seem helpful for monitoring cerebral function in the region of aneurysm surgery. Preliminary studies in our operating room suggest that electrocorticography over the operative area will be more revealing.

References

Dissecting Aneurysms of the Basilar Artery in 2 Patients

C. Bruce Alexander, M.D., Peter C. Burger, M.D., and John A. Goree, M.D.

SUMMARY An uncommon consequence of intracranial vascular disease is the intramural dissection of blood, or “dissecting aneurysm.” A 69-year-old man with chronic subarachnoid hemorrage from a posterior fossa mass lesion and a 30-year-old man with migraine and a brain stem stroke illustrate the diverse etiologic, clinical, radiographic, and pathologic characteristics of this unusual lesion.

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“DISSECTING ANEURYSMS” of the cerebral arteries warrant consideration in the differential diagnosis of subarachnoid hemorrhage, occlusive cerebrovascular disease, and intracranial mass effect. By means of 2 illustrative cases affecting the basilar artery, this report reviews the clinical, pathological, and radiologic expressions of this unusual cerebrovascular lesion.

Patient I

Clinical History

The patient was a 69-year-old white male admitted to Duke University Medical Center because of a 2-year history of progressive intellectual deterioration. One month prior to admission, when incontinence, paraparesis, and episodic nausea and vomiting made continued home care unfeasible, he was admitted to another hospital. A history of progressive bilateral hearing loss was obtained. The cerebrospinal fluid was xanthochromic, and contained 6,000 red blood cells per cubic millimeter and 920 mgm protein and 41 mgm of glucose per deciliter. An aortic arch study with cerebral angiography visualized the carotid and basilar systems and disclosed slight irregularity of the basilar artery lumen and gross posterior displacement from the clivus. Pneumoencephalography revealed moderate bilateral lateral ventricular dilatation and a mass lesion in the lower pre-pontine cistern which could not be distinguished from the brain stem. There was slight posterior displacement of an otherwise unremarkable fourth ventricle.

Upon transfer to Duke University Medical Center,
EEG monitoring for induced hypotension for surgery of intracranial aneurysms.
T H Jones, K H Chiappa, R R Young, R G Ojemann and R M Crowell

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