the loss of sound energy that occurs with increasing lengths of conducting tubing, even in low-frequency sound transmission.

The effect of learning response upon test performance possibly may have biased the results of this study. The order of testing was uniform, such that the longer tubing lengths were tested at the end of each study session. The test subjects, therefore, may have gained some improvement in hearing level by improving their testing skills over the 15-min session.6 This could obscure an actual decrease in sound transmission using the longer tubing lengths. The magnitude of this learning response is not well defined in the audiology literature. In the experimental setting described here (left motivation to perform well and a short test period), it is probably no more than 1–2 db, which borders clinical significance (1 db being the smallest change in sound level detectable by the human ear). Also, the finding that the bilateral stethoscope earpieces provide a 5 db gain in hearing level over the monaural earpiece, which is in agreement with previously reported data, occurred too soon in the session (less than 5 min from the beginning) to be significantly biased by learning response.

I conclude that the use of an appropriately sized stethoscope bell and binaural spring-loaded earpiece allows optimum transmission of low-frequency sounds in the range normally encountered in clinical practice. The use of a stopcock plus iv or oxygen tubing extensions of the normal sort commonly employed in clinical practice do not adversely affect the anesthetist's ability to monitor these critical sounds.

Implications of these findings, which have been borne out in clinical practice, are that if heart and Korotkoff sounds are inaudible with the commonly used monaural molded earpiece, then auscultation may be significantly improved utilizing the binaural stethoscope earpieces. The cost of doing this is loss of input from the rest of the operating room. Under these circumstances, a reasonable compromise is the intermittent use of the stethoscope earpieces. Perhaps of greater usefulness is the finding that, as opposed to previous recommendations, increasing the length of the connecting tubing between the patient and the earpiece (up to 120 cm, a generous length) does not significantly decrease the anesthesiologist's ability to hear, while mobility and convenience are greatly facilitated.

REFERENCES

Intracranial Subdural Gas: A Cause of False-positive Change of Intraoperative Somatosensory Evoked Potential

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Scalp-recorded somatosensory evoked potentials (SEPs) are useful in preventing brain injury during intracranial operations.1–4 A decrease in amplitude and increase in latency of SEPs characteristically occurs in both animals and humans during hypoxia within neural tissue.1,7–8

We present two patients in whom intraoperative

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Fig. 1. The decrease in amplitude of scalp recorded SEPs in response to median nerve stimulation in patient 2 is shown. Brain retractors were assessed but not repositioned in response to change in SEPs. Cranial x-ray in sitting position showed subdural air under the parietal electrodes (C3, C4), but not midline electrode (C2). SEP determinations, separated by 2 min without change in anesthetic gases, were made in sitting and supine positions at end of operation. Cranial x-ray in supine position showed frontal subdural air. The bilateral increase in amplitude of SEPs with change in position can be seen.

scalp-recorded upper-extremity SEPs changes occurred paralleling those seen during hypoxia, however, both patients awakened promptly without neurologic deficit. In the second patient, lower-extremity SEPs were unchanged. Cranial radiographs in both patients showed moderately large quantities of subdural air. We feel that these gas collections subjacent to the electrodes (or beyond) may result in changes in SEP during intraoperative monitoring (upper extremity). Monitoring of lower extremity SEPs in the sitting position appears less affected by subdural air and may prevent the erroneous conclusion of intraoperative neurologic injury. Plain skull roentgenograms will demonstrate intraoperatively when subdural air is subjacent to the SEP electrodes.

REPORT OF TWO CASES

Patient 1. A 14-year-old girl had a suboccipital craniotomy performed in the sitting position. During the operation, SEPs in response to each median nerve stimulation progressively decreased in amplitude and were unrecoverable at the end of the operation (sitting position). Brain retractors were repositioned to minimize pressure on the brainstem, without improvement of SEPs. The patient awakened rapidly following surgery without neurologic deficit. Skull roentgenograms 1 h following surgery (supine) demonstrated bilateral subdural gas mainly beneath frontal bone.

Retrospectively, we feel that subdural air had acted as an insulator under the SEP electrode.

Patient 2. A 38-year-old woman underwent intradural exploration of the cervical spinal cord in the sitting position under fentanyl–enflurane–oxygen anesthesia. SEPs were evaluated using a four-channel signal averager (Nicolet Med-800, Nicolet Biomedical, Madison, Wisconsin). Electrodes were placed 2 cm posterior to C3, C4, and C2 (International 10–20 system) and were designated C3, C4, and C2, respectively, and an electrode was placed over the second cervical vertebra. All electrodes were referenced to Fpz. Sterile needle stimulating electrodes were placed near each median and posterior tibial nerve, and a stimulus intensity three to four times motor threshold was used. SEPs in response to stimulation of each extremity were monitored frequently throughout the procedure. Waveforms were evaluated for both latency and amplitude. A progressive decrease in amplitude of cortical SEPs in response to stimulation of each median nerve stimulation occurred without a change in waves recorded over second cervical vertebra (fig. 1). The amplitude of the lower-extremity SEPs were not decreased (fig. 2). Because of the location of surgical manipulation (cervicomedullary junction), retractors displaced the cerebellar hemispheres laterally without apparent traction on the brainstem. Retractor position was evaluated and felt not to be injurious to the brainstem and therefore were not adjusted in response to the change in SEPs. During the period of decrease in SEP amplitude, enflurane concentration, patient temperature, and PaCO2 were maintained constant. Evoked potential equipment function was normal and electrode impedance was adequate (<2 kohm). Following completion of surgery, anteroposterior and lateral projection radiographs of the skull were taken with the patient anesthetized (0.5% enflurane) in the sitting position and then 2–3 min afterward in the 30-degree head-up position without a change in anesthetic level and without a change in PaCO2 or end-tidal PaCO2. SEPs were determined in sitting position and in the 30-degree head-up position approximately 2 min later. The amplitude of the upper-extremity SEPs was increased immediately following position change (fig. 1E). Radiographs obtained with the patient in the sitting position showed both subarachnoid and subdural gas. Subdural gas was underlying the parietal electrodes (C3, C4) without extension to midline, thus sparing the C2 electrode. The location of the gas (parietal) was seen in both frontal and lateral projections. Radiograph obtained immediately thereafter with the patient supine showed that...
the subdural gas had shifted to the frontal region and was no longer beneath the parietal electrodes. The anesthetic gas then was discontinued, and the patient awakened rapidly without neurologic deficit.

FIG. 2. Lower-extremity SEPs in the second patient are shown. The amplitude of the response increased with a decrease in latency during surgery while amplitude of upper-extremity SEPs was decreased.

**DISCUSSION**

We present two patients who had intraoperative changes in SEPs similar to those previously associated with transient or permanent brain injury\(^1\) who awakened without neurologic deficit. The presence of intracranial gas interfering with conductivity was suspected in the first patient, and the presence of intracranial gas was confirmed by postoperative skull radiographs. We hypothesized that subdural gas between the somatosensory cortex and the scalp recording electrodes would dampen and even ablate the amplitude of the scalp recorded waves. We further assumed that in the sitting position the upper-extremity SEPs recorded over the parietal convexity might be more affected than the lower-extremity SEP (recorded over the vertex), since the patient’s operative position would favor gas collecting laterally.\(^2\) In the second patient, the amplitude of lower-extremity SEPs was retained, despite a precipitous decline in amplitude of upper-extremity SEPs. A change from the sitting to 30-degree head-up position caused an immediate increase in amplitude of the depressed upper-extremity SEPs amplitude, associated with movement of the subdural gas from under the parietal electrodes (\(C_3\), \(C_4\)).

In the patients presented, surgical and anesthetic procedures were not changed despite changes in SEPs similar to those previously reported evidencing neurologic injury.\(^1\) In our cases, all other aspects of anesthesia and surgical management were found acceptable and SEP changes due to intradural gas were considered. Thus, we did not ignore alterations in SEPs but ruled out the usual causes of changes in SEPs (brain retraction, arterial hypotension, and excessive anesthetic). Both anesthesiologist and neurosurgeon concurred that, with the exception of changes in SEPs, care seemed satisfactory according to other criteria and we did not effect major changes in the surgical procedure. Nonetheless, in both patients, the possible source of neurologic change, brainstem retraction, was evaluated and thought satisfactory. Therefore, in both patients, the only alternatives were to completely remove the retractor and continue surgery at a severe disadvantage (lack of surgical exposure), discontinue surgery, or disregard the otherwise unsubstantiated changes in SEPs. The first two alternatives involve substantial risks.

Preservation of lower-extremity SEPs in the second patient concurrent with dramatic depression of upper-extremity SEPs suggests that monitoring both upper- and lower-extremity SEPs may prevent the erroneous
conclusion of neurologic injury when changes in upper-extremity SEPs are caused by subdural gas. Intraoperative skull roentgenograms obtained in response to unexplained changes in the upper-extremity SEPs affirmed the presence of subdural gas. This simple technology is readily available. Since the upper- and lower-extremity SEPs are transmitted in the medial lemniscus of the brainstem, it is unlikely that one portion of the lemniscus would be affected profoundly by brainstem retraction and the adjacent lamellation spared. Whether SEP changes associated with subdural gas are due to an insulator effect caused by the gas interface or whether they are a result of injury from tension pneumocephalus is difficult to ascertain. We have previously shown that patients who become symptomatic from tension pneumocephalus do so a variable time after surgery.10 Since SEP amplitude changed prior to dural closure in this setting, injury from pneumocephalus is less likely to account for the SEP change.

In conclusion, we believe that subdural gas can accumulate and alter upper-extremity SEPs in patients in the sitting position. Preservation of lower-extremity SEPs in the presence of such subdural air affirms the benignity of this condition. Intraoperative skull radiography can demonstrate subdural gas under the parietal electrodes and obviate a potentially dangerous alteration in standard surgical technique.

Prevention of Electroconvulsive Therapy-induced Dysrhythmias with Atropine and Propranolol

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Prophylactic antidysrhythmia treatment for electroconvulsive therapy (ECT) should inhibit adverse alterations in cardiac rhythm while not interfering with the therapeutic efficacy of the seizure. While atropine sulfate fulfills these requirements and is routinely used as a premedication for ECT, ventricular dysrhythmias frequently develop in patients at risk despite atropine pretreatment.1-3 Although lidocaine inhibits ventricular ectopy associated with ECT, the seizure duration is reduced on a dose-related basis, thereby decreasing the therapeutic efficacy of ECT.4-7 Intravenously administered propranolol suppressed ventricular dysrhythmias during ECT in a patient who had previous seizure inhibition associated with lidocaine pretreatment,10 but in our case described below, it failed to prevent severe bradycardia with ventricular premature contractions (VPCs), bigeminy, and coupling of ectopic beats. The combined pretreatment regimen of atropine with propranolol appears to be a logical and effective approach.

REPORT OF A CASE

A 61-year-old man was scheduled to receive a series of ECT treatments for major depression resistant to maximal medical therapy.

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