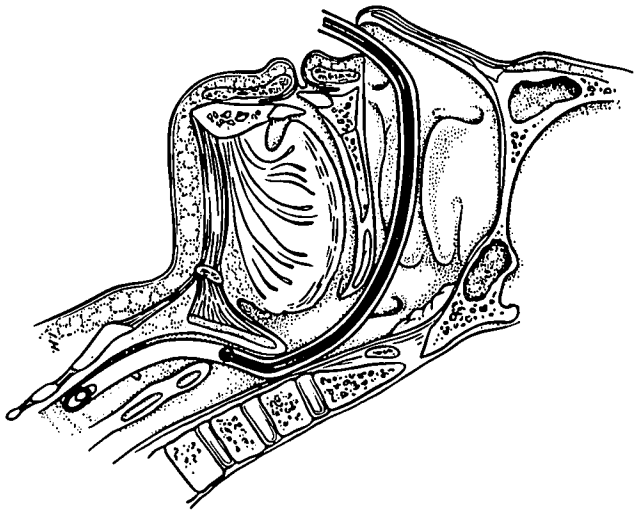

FIG. 3. Nasotracheal tube advanced off stylet into larynx and trachea.

and start over. The other technique avoids this problem. The stylet is preshaped in a duplicate tube. Using a stylet blindly without measuring the correct length may be risky, since the stylet may be longer than the nasotracheal tube, resulting in damage to the larynx, trachea, or esophagus.

Various types of stylets have been used in this technique. The two main types are copper for the larger tubes and a moderately rigid wire for the smaller pediatric tubes. An 8-gauge stylet is used in larger tubes and a 14-gauge stylet in the smaller pediatric tubes. Pliability in the stylet is necessary, so that it will advance through the endotracheal tube and make the bend in the nasopharynx, while at the same time maintaining the upward bend of the anterior tip.

DISCUSSION

This technique was designed primarily to be used in those patients in whom all other techniques at intubation had failed. In general, these patients were anesthetized with general anesthesia and were spontaneously breathing. The technique has been used approximately 30 times with one failure. There have been no complications. This technique for intubation of the trachea may well be valuable during difficult intubations in a paralyzed patient whose anterior larynx can be visualized, but because of inadequate room to use the forceps, intubation proves difficult. This is particularly true in patients with a very small mouth and in infants. The technique also has been used in the emergency room in several patients after attempts at blind nasotracheal intubation had been unsuccessful.

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Seizure Associated with Induction of Anesthesia with Isoflurane

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All previous studies have concluded that, although isoflurane may cause high-voltage synchronous spikes in dogs and cats,[1–4] it does not produce electroencephalographic (EEG) or clinical seizures in humans, even when administered in high concentrations and in association with hypocapnia and photic or auditory stimulation.[5–9] We present a patient who twice had seizures during the induction of anesthesia using isoflurane and nitrous oxide.

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Key words: Anesthetics, volatile: isoflurane. Complications: convulsions. Monitoring: electroencephalography.

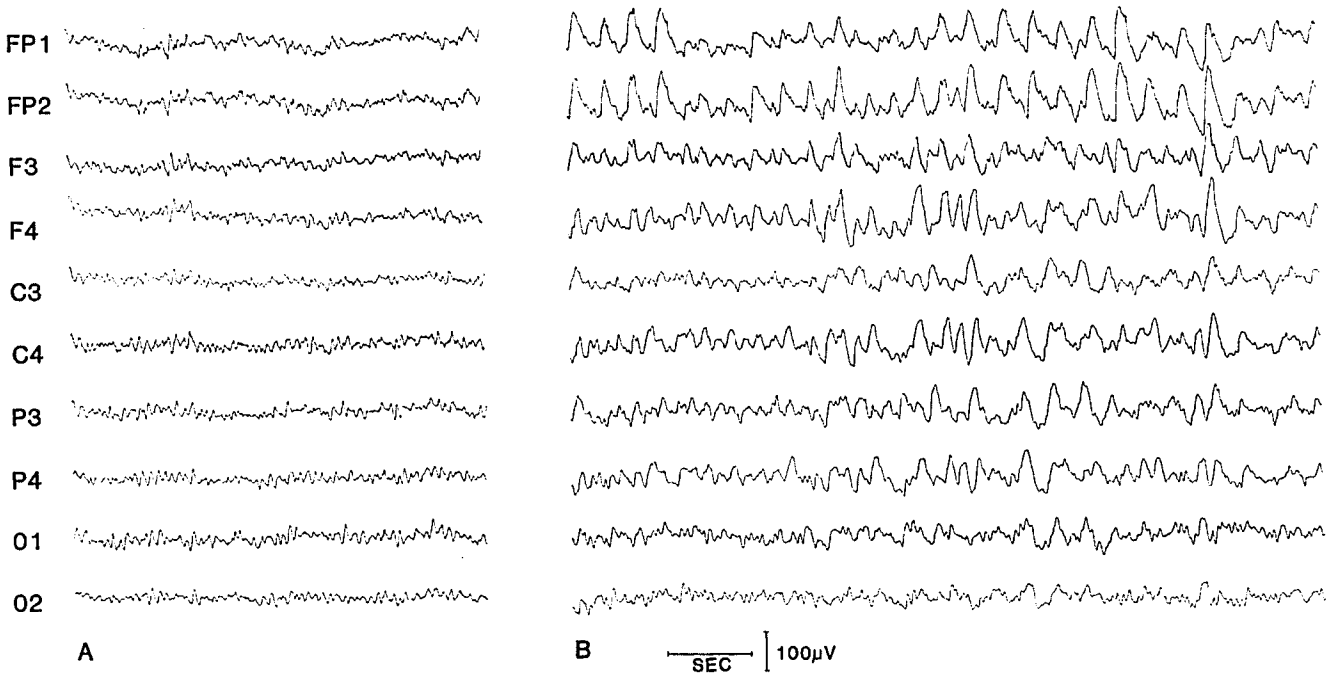

FIG. 1A. 1021 hours: Normal resting EEG immediately before anesthesia, no premedication. B. 1024 hours: EEG pattern during induction of anesthesia 40 s before the beginning of the seizure discharges. Note: An ipsilateral ear reference montage was used throughout the recording; the input 1 electrodes are indicated at the left of the tracings; the input 2 electrodes were alternatively A1 and A2. To save space, tracings 11–16 from the temporal areas have been deleted from all figures. They showed essentially the same symmetric patterns as seen in the medial convexity tracings. Amplification 7 μV/mm.

REPORT OF A CASE

A 24-year-old Hispanic man sustained an injury to his left lower leg when it was pinned between the bumpers of two cars in a parking lot. The patient was previously in excellent health, and his injuries were limited to the involved extremity, which was crushed at the level of the upper tibia. He had never been in a hospital previously and had no history of head trauma or a seizure disorder. He was physically active and worked 40–60 hours weekly, doing hard manual labor.

During the eight months after the injury, the patient underwent general anesthesia uneventfully 11 times for debridement of tibial osteomyelitis and for skin grafting. Each time, anesthesia was induced with thiamylal administered iv between 30 and 40 min after premedication. Premedication included im meperidine and hydroxyzine on six occasions and oral diazepam on the five other occasions. Maintenance of anesthesia was with isoflurane on nine occasions, with enflurane once, and with nitrous oxide/oxygen and narcotic once. No movements of the body of the patient were noted during induction or maintenance of these anesthetics.

Nine months after his original injury, the patient came to the operating room unpremedicated for his 12th anesthetic for debridement and skin grafting. He was anxious and requested that anesthesia be induced by inhalation to avoid the discomfort of having an iv infusion started prior to loss of consciousness. Anesthesia was induced with 60% nitrous oxide in oxygen, followed 2 min later by the introduction of isoflurane, 0.5%. The concentration of isoflurane then was increased in stepwise fashion (approximately 0.5% increment every 40 s). After approximately 4 min, the delivered concentration of isoflurane was 4.0% as indicated on vaporizor dial. The patient breathed spontaneously with an apparent large tidal volume throughout the induction, which was not complicated by breath-holding or coughing. Arterial blood pressure was 110/70 mmHg and the heart rate 110 bpm when he suddenly had tonic flexion at the hips and elbows and then had clonic seizure activity (approximately three cps) involving the facial masculature and arms, which lasted for 30 s. At that time, pH_a was 7.38, Pa_{CO_2} 46 mmHg, and Pa_{O_2} 240 mmHg. Anesthesia was continued with isoflurane (1.5–2.0%) and nitrous oxide (60%) in oxygen. The remainder of the anesthetic was uneventful, the surgery was completed, and the patient awoke without problems.

In anticipation of the patient's return for an additional procedure 2 days later, approval was obtained from the institutional human experimentation committee, and informed consent was obtained from the patient to duplicate his anesthetic with continuous EEG monitoring. A complete neurologic examination of the patient was within normal limits. A baseline EEG recording, including both awake and sleep tracings as well as photic stimulation and hyperventilation, was recorded 1 h before the patient was taken to surgery. This EEG was entirely within normal limits. EEGs were recorded using a 16-channel Grass® Model 8 EEG instrument.

Immediately prior to induction of anesthesia, the EEG showed alpha activity of moderate voltage, at 8–10 per second, mild beta activity, and some random diffuse theta activity (fig. 1A). Anesthesia again was induced with 50% nitrous oxide and oxygen without premedication. There was no change in the EEG after 60 s. At that time, isoflurane 0.5% was added to the mixture, with increases in the concentration by 0.5% every 30 s. The induction again was tolerated well with no coughing or breath holding. During the first 100 s of isoflurane administration, there was a slight increase in alpha frequency. At 110 s, theta activity became more prominent and bursts of higher voltage rhythmic activity in the delta frequency band began to appear and become more prolonged and progressively increased in voltage (fig. 1B). Two hundred seconds after the beginning of administration of isoflurane, the patient suddenly flexed

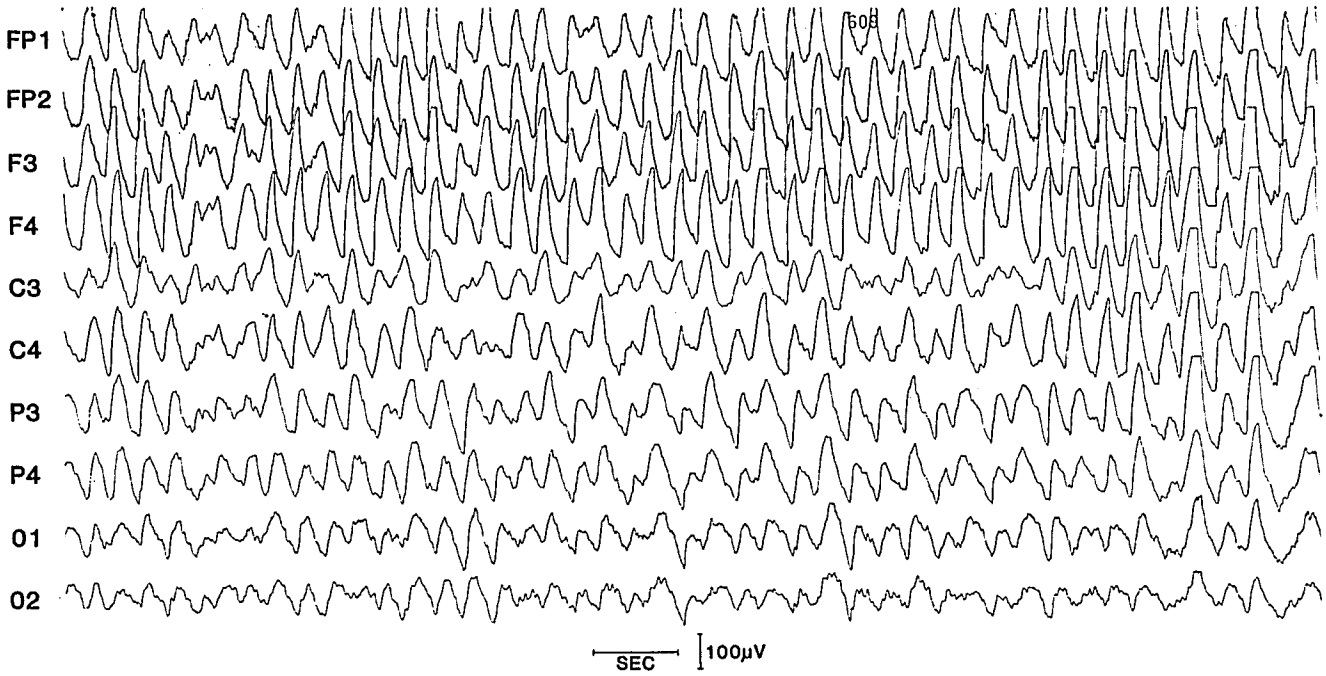

FIG. 2. 1025 hours: Early seizure discharge, 15 s after flexion at hip and neck.

at the hip and neck. Coincident with that sudden movement, high-voltage rhythmic 2.5/s waves appeared diffusely in the EEG with a short burst of muscle potentials. This rhythmic slow activity rapidly increased in voltage to 200–300 μV (fig. 2) and varied in frequency, between 2 to 3/s. During the next 80 s the frequency became more regular and gradually slowed to 1.5/s (fig. 3), at which time jerking movements of both shoulders were observed, followed by blinking of the eyes, jerking of the legs, and finally, clonic movements of the entire body. After a further 60 s, during which body jerking continued, the slow activity became less regular with mixed frequencies, variable amplitudes, and somewhat more irregular wave forms. Amplitude of the slow waves increased to 400–500 μV. Since no

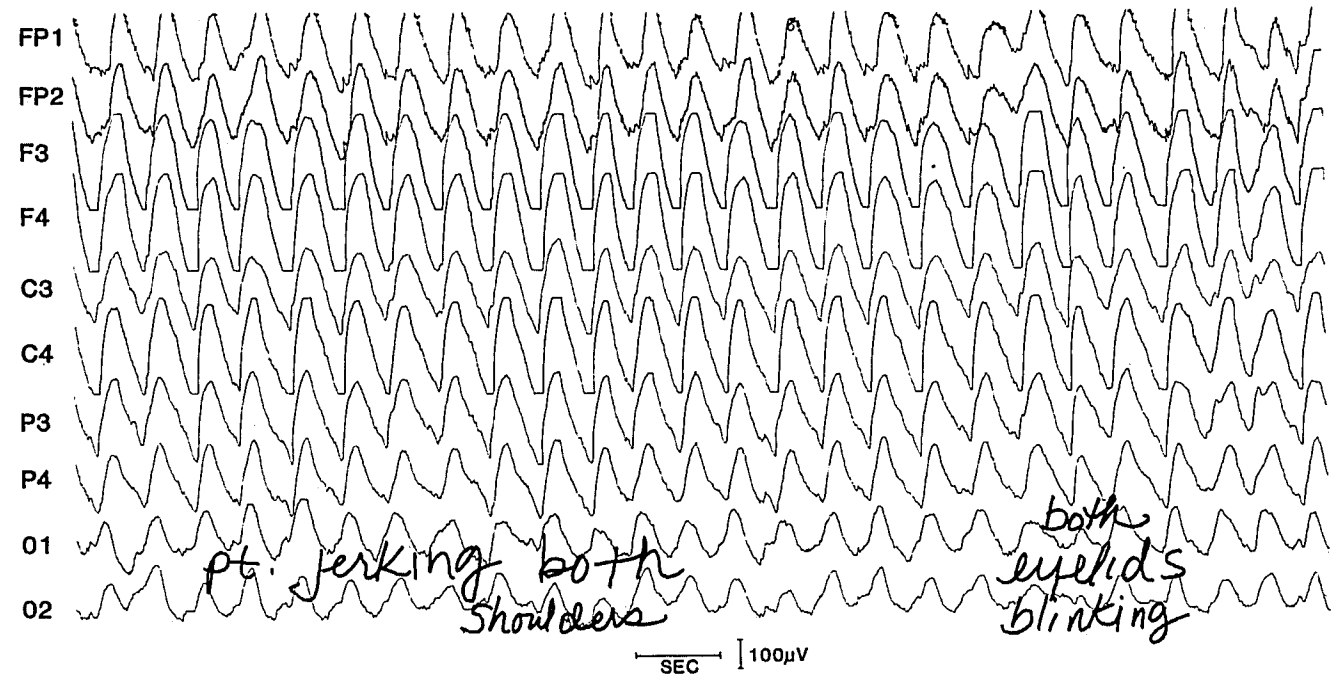

FIG. 3. 1026 hours: Start of the clinical seizure, technician's observations indicated. Amplifcation 10 μV/mm.

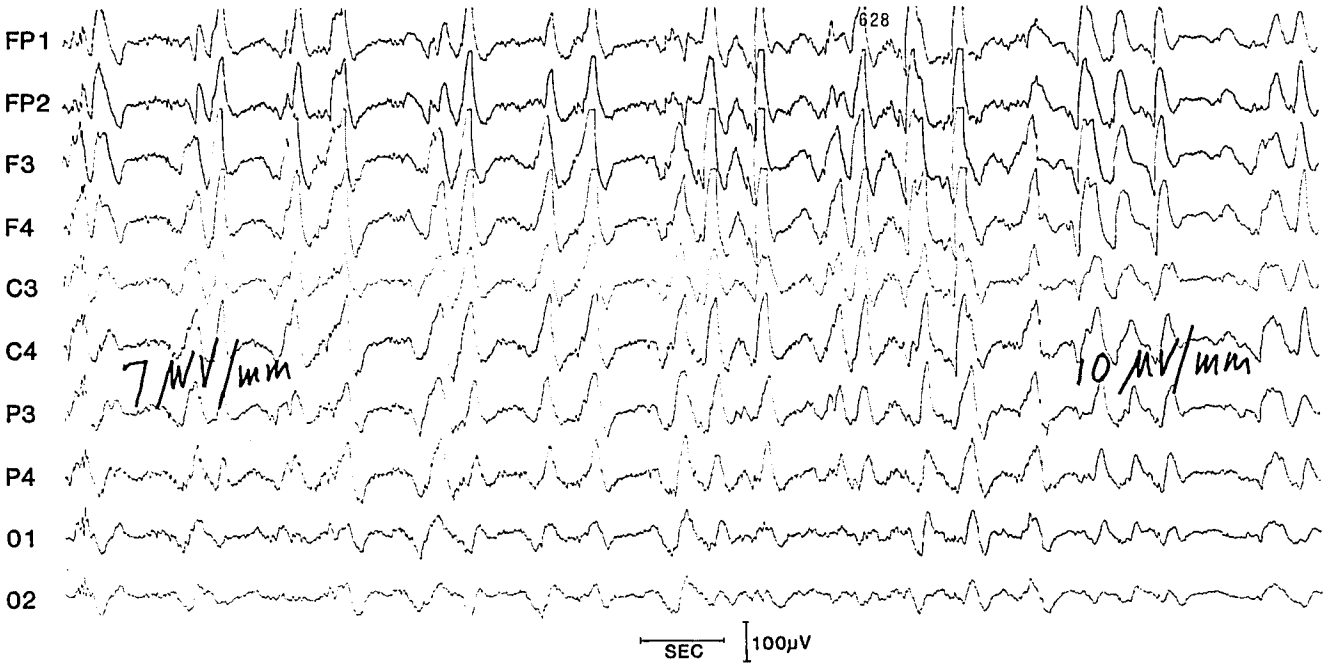

FIG. 4. 1029 hours: Generalized body jerks continuing. A short time constant (0.035 s) was in use, producing a relative distortion of slow wave forms and reduction of slow component amplitude, thus revealing some spike and wave complexes. The amplification had been restored to 7 μV/mm but again was reduced to 10 μV/mm 1 s before the notation "10 μV/mm" at the right.

spike activity was seen, the time constant was reduced to 0.035 s to reduce the relative amplitude of the slow components in order to search for spikes; a few spike and wave forms then were seen (fig. 4). Only a few more spike and wave complexes were seen intermittently during the remainder of the recording.

The body jerking gradually decreased and ceased 4.5 min after it began. Twitching of the eyes continued for another 40 s. At that time, 50 mg of thiamylal was administered iv and the clinical seizure activity ceased. Precordial auscultation confirmed adequate assisted spontaneous ventilation throughout the seizure. High-voltage irregular

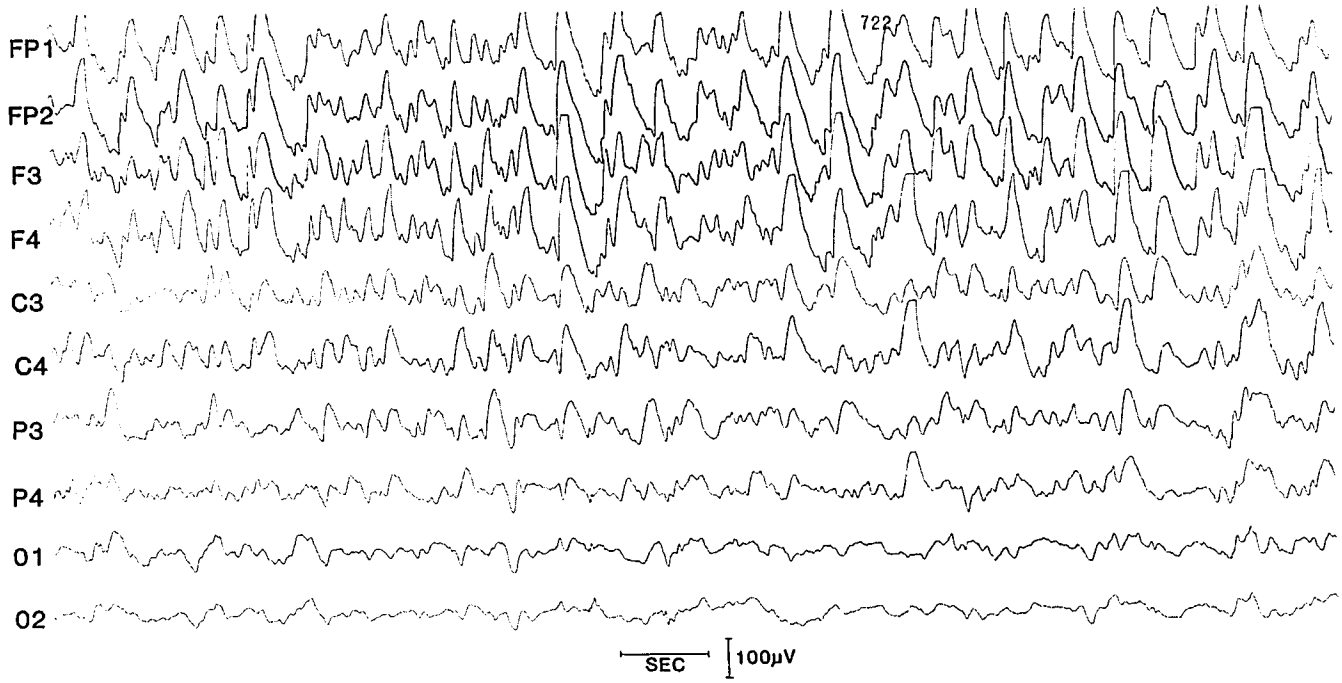

FIG. 5. 1044 hours: Pattern after seizure, similar to fig. 1*B*, but delta components are more prominent.

slow bursts continued in the EEG for another 4–5 min, gradually giving way to a pattern of mixed-frequency high-voltage delta waves with rhythmic theta waves and some activity in the alpha frequency band, more characteristic of typical EEG recordings during general anesthesia (fig. 5). Thus, the seizure discharge, consisting predominantly of very high voltage smoothly contoured delta waves, began 95 s before clinical seizure manifestations began (excepting the early isolated flexor jerk) and continued for 4–5 min after they ended. Because of the gradual change in pattern, it is not possible to say precisely when the seizure discharge stopped, but its total duration was at least 10 min.

The patient awoke uneventfully following conclusion of the 60-min procedure. Repeat neurologic examination the following day was entirely within normal limits. The patient left town shortly after this anesthetic and is lost to follow-up.

Discussion

The administration of a variety of ethers has been associated with seizure activity. Clinically, enflurane may cause seizures in humans, especially when given in relatively high concentrations during hyperventilation.[10] Since isoflurane is a structural isomer of enflurane, several investigators have examined its EEG effects and concluded that neither seizure activity nor classical EEG "spiking" patterns appears in humans during the administration of isoflurane.[5–9] Our patient clearly had seizures on two occasions during the induction of anesthesia using isoflurane and nitrous oxide, one of which was confirmed by EEG.

Why should a seizure occur reproducibly in a single patient during induction of anesthesia using isoflurane and nitrous oxide when a large body of clinical experience and a significant number of monitored anesthetics in research settings failed to disclose such an occurrence? Perhaps our patient had a preexisting subclinical seizure diathesis unmasked only during induction of anesthesia. The probability of that being the case is reduced by the normal baseline EEG with photic driving and voluntary hyperventilation.

Did our patient inadvertantly receive enflurane rather than isoflurane? Although no gas analyses were done, the two anesthetics were administered in two different rooms using two different anesthesia machines with two different vaporizers. Thus, we reject that possibility.

Might the observed seizure activity have been caused by nitrous oxide, which can cause overt seizures in mice[11,12] but only minor EEG changes in humans?[13] Because of this and because of the uncounted millions of uneventful anesthetics using nitrous oxide during the past 100 years, we do not consider that a possible explanation.

The two anesthetics during which seizures occurred were distinguished from his other uneventful isoflurane anesthetics by our omission of both premedication and of barbiturates during induction of anesthesia. Homi *et al.*[5] monitored the EEG during isoflurane anesthesia for 20 procedures. No seizures were observed. They did not specify whether those patients received premedication, nor did they detail the manner in which anesthesia was induced. Thus, perhaps all of their patients monitored by EEG received a barbiturate for premedication or induction of anesthesia. Clark *et al.*[6] studied five healthy unpremedicated adult volunteers during induction of anesthesia with nitrous oxide and isoflurane in a manner similar to that used with our patient. None of the experimental subjects had EEG or clinical evidence of seizure activity.

Pauca and Dripps[9] reported the clinical anesthetic courses of 100 patients anesthetized with isoflurane. Of the 100, 38 underwent an inhaled induction using nitrous oxide and oxygen or oxygen alone as the accompanying gas. Of the 38 patients, 20 were premedicated with a narcotic and 18 with a barbiturate, diazepam, chloral hydrate, or atropine alone. None of those patients had clinical evidence of seizures. Although, in the same study, the EEG was monitored in 55 patients, it is not possible to ascertain the percentage of those who were premedicated and the percentage who received an inhalation induction of anesthesia. Those investigators did report "spike-like wave complexes" lasting 1–2 s and of up to 200 μV amplitude in five of six patients in whom end-tidal isoflurane was increased above 2.5% and/or in whom end-tidal carbon dioxide concentration intentionally was lowered. Finally, Eger *et al.*[7] anesthetized seven healthy unpremedicated adult volunteers by inhalation with isoflurane in oxygen. In none of these subjects was clinical or EEG evidence of a seizure apparent, with a maximum alveolar concentration of isoflurane of 2.5%. This may be higher than the alveolar concentration achieved in our patient.

Thus, few unpremedicated patients have been studied by inhalation of isoflurane using inspired concentrations in excess of 2.5%. The appearance of "spike-like wave complexes" of relatively large amplitude in five of the patients studied by Pauca and Dripps[9] may be significantly related to the EEG and clinical phenomena of our patient. Moreover, perhaps the pungency and respiratory responses of isoflurane makes it unlikely that many inhalation inductions with isoflurane in premedicated patients occur.

Isoflurane produces spike waves in cats[1,2] and in dogs.[3] Pauca and Dripps[9] documented the production of high-amplitude spike-like waves in humans during isoflurane administration. These facts combine with our observations in one patient to suggest that the systematic search for clinical or EEG evidence of seizure activity in unpremedicated humans ought to be expanded. Our case report indicates that isoflurane can be associated with seizures in humans, even if only rarely.

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