EFFECTS OF ENFLURANE ON VISUAL EVOKED POTENTIALS IN HUMANS

O. Z. CHI AND C. FIELD

SUMMARY
Effects of enflurane on visual evoked potentials (VEP) were studied in eight healthy female patients. Control VEP were recorded from the occipital electrode (Oz-Cz), and prominent negative (N70) and positive (P100) peaks were observed following flash light stimulation. After induction of anaesthesia with thiopentone, VEP were recorded at expired concentrations of 1.2, 1.8, 2.4 and 2.7% of enflurane in 100% oxygen. The amplitude of the P100 was decreased and the latency of the N70 was increased significantly from the control with all concentrations of enflurane. No significant difference was found between different concentrations of enflurane. The latency of the P100 was increased significantly only at concentrations above MAC and at 2.7% when it was significantly longer than those at 1.2 and 1.8%. We conclude that VEP are altered significantly with enflurane in clinically used concentrations.

KEY WORDS
Anaesthetics volatile: enflurane. Monitoring brain: visual evoked potentials.

All general anaesthetics affect electrical activity of the brain, including the electroencephalogram (EEG) and the evoked potentials. Unlike other frequently used general anaesthetics, enflurane induces episodes of paroxysmal spike activities and periodic suppression in the EEG with inspired concentrations exceeding 3%. This phenomenon occurs especially in children [1,2]. Enflurane produces dose related reduction in the amplitudes of the cortical components of the somatosensory evoked potentials (SSEP) and early cortical components of the brainstem auditory evoked potentials (BAEP). Latencies of these evoked potentials are increased with enflurane in a dose related pattern [3,4].

Even though recording of the visual evoked potentials (VEP) is vulnerable to various factors during surgery, it has been used in monitoring function of the central nervous system during cardiopulmonary bypass, as disturbance of visual function is one of the most common complications following cardiovascular surgery [5-7]. Others used VEP in monitoring integrity of the visual pathways during neurosurgical procedures [8,9]. The VEP are elicited by flashes from light-emitting diodes mounted on goggles placed over the patient’s closed eyes. They have prominent negative and positive peaks at about 70 and 100 ms after stimuli, termed N70 and P100, respectively. The P100 is thought to arise in the striated and parastriated visual cortex. The origin of N70 is not clear. Changes in the latencies and amplitudes of these peaks are affected by several factors, including anaesthetic agents and surgical stimuli.

The effects of enflurane on the VEP are not well known and this study was designed to examine this in further detail.

PATIENTS AND METHODS
This study was approved by our Institutional Review Board. Informed consent was obtained from eight female patients (ASA physical status I or II, age 21–45 yr) undergoing non-neurosurgical operations. No patient had ophthalmological or neurological disorders. No premedication was given.

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Silver chloride disk electrodes were fixed to the scalp with collodion according to the 10-20 international system [10]. The VEP were recorded from the occipital electrode (Oz) with reference to the vertex electrode (Cz) using the mastoid electrode as a ground. Impedance was kept less than 3000 Ω. The bandpass filter was set at 1–200 Hz. Sensitivity was set at 100 μV full scale. A pair of goggles lined with light-emitting diodes was placed over the patient’s closed eyes. Binocular flash stimuli were given at a rate of 1.9 Hz and a duration of 10 ms. Each averaging epoch was 500 ms, with a total of 100 repetitions. A Nicolet Pathfinder I (Nicolet Biomedical Instruments, Madison, WI) was used to record the evoked potentials.

The VEP were recorded before induction of anaesthesia as a control. Anaesthesia was induced with tubocurarine 0.05 mg kg\(^{-1}\), thiopentone 4–5 mg kg\(^{-1}\) and suxamethonium 1.5 mg kg\(^{-1}\) i.v. Anaesthesia was maintained with various concentrations of enflurane in 100% oxygen. Vecuronium was given as needed to maintain neuromuscular block and controlled ventilation. End-expiratory enflurane concentration was measured with an Engstrom Emma gas analyser equipped with a Flextube Humidifier. End-expiratory partial pressure of carbon dioxide was monitored using a Datex CO\(_2\) Monitor, and maintained at 4.7–5.3 kPa. Body temperature was measured with a Stat-Temp temperature sensor, and maintained at 35–37 °C. Systolic arterial pressure was kept within ±25% of the value before operation with enflurane and an i.v. infusion of lactated Ringer's solution. Twenty minutes after induction, VEP were recorded at 1.2, 1.8, 2.4 and 2.7% of enflurane. Each end-expiratory enflurane concentration was maintained for 10 min before recording. At least two individual VEP were recorded at each enflurane concentration. The order of administration of each concentration was randomized. To avoid the effects of surgical stimuli, the VEP were recorded when there was no or minimal stimuli. Latencies of the N70 and the P100 were measured, and the amplitude of the P100 was measured as the vertical distance between the N70 and the P100.

For data analysis, repeated measures analysis of variance and the Student-Newman-Keuls multiple comparison test were used. \(P < 0.05\) was considered statistically significant.

![FIG. 1. The VEP recording from a patient with inspired concentrations of enflurane from 1.2% (E1.2%) to 2.7% (E2.7%). C = Control.](http://bja.oxfordjournals.org/)

### Table I. Mean (sd) latencies and amplitudes of the VEP at control and at different concentrations of enflurane. Statistically significant: * compared with control; † compared with 1.2% and 1.8%

<table>
<thead>
<tr>
<th>Enflurane Concentration</th>
<th>N70 (ms)</th>
<th>P100 (ms)</th>
<th>N70-P100 (μV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n = 8)</td>
<td>78.8 (10.3)</td>
<td>110.1 (19.5)</td>
<td>11.21 (5.45)</td>
</tr>
<tr>
<td>1.2% (n = 8)</td>
<td>92.0 (9.0)*</td>
<td>116.9 (11.3)</td>
<td>7.56 (3.25)*</td>
</tr>
<tr>
<td>1.8% (n = 8)</td>
<td>89.5 (7.6)*</td>
<td>118.4 (10.1)</td>
<td>6.60 (2.69)*</td>
</tr>
<tr>
<td>2.4% (n = 8)</td>
<td>91.3 (8.4)*</td>
<td>122.8 (10.5)*</td>
<td>5.95 (3.08)*</td>
</tr>
<tr>
<td>2.7% (n = 6)</td>
<td>98.0 (6.3)*</td>
<td>132.0 (12.7)*</td>
<td>4.87 (3.09)*</td>
</tr>
<tr>
<td>(P&lt;0.0005)</td>
<td>&lt;0.0025</td>
<td>&lt;0.0025</td>
<td>&lt;0.0025</td>
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</tbody>
</table>
EFFECTS OF ENFLURANE

RESULTS

VEP were obtained in a reproducible manner in all patients. Because of clinical constraints, however, 2.7% enflurane was not administered to two patients. In figure 1, typical VEP waveforms from one patient are shown. Duplications of the waveforms are superimposed. Mean values for latencies and amplitudes at various end-expiratory enflurane concentrations are shown in table I. The latency of the N70 at each enflurane concentration increased significantly from control. Differences between various concentrations of enflurane were not significant. Latencies of the P100 increased significantly only at 2.4 and 2.7% compared with control. The latency of the P100 at 2.7% was significantly longer than those recorded at 1.2 and 1.8%. Otherwise, no significant difference was found between various concentrations of enflurane. The amplitude of the P100 at each enflurane concentration diminished significantly from that of control. No significant difference was found in the amplitude recorded at various concentrations of enflurane.

DISCUSSION

Many factors in addition to anaesthetics may affect recording of evoked potentials. Hypothermia increases the latency [11]; in this study, body temperature was maintained within a reasonably narrow range. We minimized fluctuations in end-expiratory carbon dioxide by controlled ventilation in order to avoid the effects of carbon dioxide tension on evoked potentials [12]. Premedication was not given and, to minimize the effects of thiopentone, we waited at least 20 min after induction of anaesthesia before recording the VEP. Systemic arterial pressure was maintained within ±25% of the value before operation [13].

Our study showed that the amplitude of the P100 significantly decreased with administration of enflurane. This finding is similar to that of Raitta, Karhunen and Seppalainen, who demonstrated that 2% enflurane and 66% nitrous oxide in oxygen decreased the amplitude of the VEP [14]. They found that latencies of the VEP increased with 2% enflurane in 66% nitrous oxide, although the increase was not statistically significant. In figure 1, decreased amplitudes of the VEP peaks during enflurane administration, especially those of longer latencies, make the VEP recording look different from that of the control.

However, the peaks of longer latencies beyond P100 are thought to have little significance in interpretation of the VEP. Our study showed that the latency of the N70 increased with enflurane. The latency of the P100 increased also with enflurane in concentrations greater than the MAC value. Effects of enflurane on the VEP appear to be similar to those recorded using isoflurane. Chi and Field reported that isoflurane increased the latencies and decreased the amplitudes of the VEP [15]. When compared with a high dose of fentanyl (up to 60 µg kg⁻¹) which causes a 30% decrease in amplitude of P100 without significant changes in latencies [16], VEP is vulnerable to the effects of enflurane and isoflurane. Marked flattening of the VEP peaks during enflurane or isoflurane anaesthesia make interpretation of the VEP difficult. This supports clinical use of opioid anaesthesia during surgery in preference to inhalation anaesthesia when VEP is monitored.

Keenan and colleagues monitored VEP during cardiopulmonary bypass with hypothermia to 18.8 °C in spite of a major decrease of amplitudes and increase of latencies when opioid anaesthesia was used [5]. If enflurane is used, the VEP changes caused by enflurane will be added to the changes of hypothermia which will make interpretation of the VEP difficult. Detection of awareness during cardiopulmonary bypass is also probably not possible with VEP monitoring. Changes in latencies and amplitudes of the VEP with various enflurane concentrations are not significant except for the latency of the P100 at a concentration of 2.7% compared with those at 1.8% and less. Therefore, VEP would not be useful in monitoring depth of enflurane anaesthesia. We did not see any increase in the amplitudes of the VEP as reported by Burchiel and colleagues [17], possibly because of the lower concentrations of enflurane administered in our study.

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REFERENCES


