EFFECTS OF HALOTHANE ON MOTOR EVOKED POTENTIAL RECORDED IN THE EXTRADURAL SPACE

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Somatosensory evoked potentials (SEP) recorded from the extradural space are used frequently for monitoring spinal cord integrity during scoliosis surgery [1]. However, neurological deficits may still occur in spite of unchanged SEP [2]. Recent experimental data suggest that changes in neurological function induced by spinal cord ischaemia are detectable in motor pathways before sensory pathways [3, 4].

In 1980 Merton and Morton described a technique for stimulating the motor cortex in intact man [5] and this has been used to assess motor conduction velocities in several neurological conditions [6, 7]. The technique has been used also to monitor motor pathways during scoliosis surgery, for which it was thought to be a potentially useful adjunct to conventional monitoring of SEP [8]. The effects of anaesthetic agents on the motor evoked potential (MEP) have not been investigated, although they are considered to have little influence [8]. In the present study we have examined the effects of halothane on the MEP recorded in the extradural space.

PATIENTS AND METHODS

We studied 16 patients admitted to the Royal National Orthopaedic Hospital for corrective surgery for idiopathic adolescent scoliosis. All patients had a scoliosis which was concave to the left and with a Cobb angle between 30° and 70° assessed by plain x-rays. The study was approved by the Hospital Ethics Committee and written informed consent was obtained from the patients, or parents, as appropriate.

All patients were premedicated with papaveretum 0.3 mg kg⁻¹ and hyoscine 0.006 mg kg⁻¹ i.m. 90 min before induction of anaesthesia. When the patient arrived in the anaesthetic room two standard EEG electrodes were fixed to the scalp with collodion and used to stimulate the motor cortex. One electrode was placed at the vertex and the other 5 cm lateral and 1 cm anterior, to act as the cathode and anode, respectively. Anaesthesia was induced with propofol 2.5 mg kg⁻¹ and intubation of the trachea facilitated with pancuronium 0.08 mg kg⁻¹. The lungs were ventilated with 70% nitrous oxide in oxygen and a radial artery cannulated for measurement of arterial pressure. The patients were placed in a prone position on a Montreal mattress and a bipolar recording electrode.
inserted into the extradural space at the C7-T1 space as described previously [9].

The correct position of the electrode within the extradural space was confirmed by recording the SEP to alternate right and left posterior tibial nerve stimulation [1]. The polarity of the recording electrode was reversed and a Digitimer D180 stimulator triggered by a Medelec MS 91 used to stimulate the motor cortex. This system had a maximum output of 750 V with a peak current of 500 mA and a time constant of 50 ms. Because it was difficult to measure the output of the stimulator directly, the stimulus intensity was graded as a percentage of the total nominal output. At least three responses were averaged and a range of intensities, at 10% increments, studied from 30 to 100% of the total output. In this manner a stimulus intensity was selected which was supramaximal. Further details of the electrophysiological techniques were described by Boyd and colleagues [8]. After the measurement of the baseline MEP approximately 20 min after induction of anaesthesia, patients were allocated randomly to receive either halothane (n = 8) or an infusion of propofol (n = 8). In the halothane group the lungs were ventilated to an end-tidal concentration of 1 MAC (Datex) corrected for age and nitrous oxide concentration [10]. Stimulation of the motor cortex was repeated 15, 30, 40 and 50 min after introduction of halothane. In the propofol group a three-stage infusion of propofol was administered as follows: 21 mg kg⁻¹ h⁻¹ for 5 min, 12 mg kg⁻¹ h⁻¹ for 10 min and 6 mg kg⁻¹ h⁻¹ for 35 min. This infusion regimen has been shown to result in blood concentrations of propofol 4 µg ml⁻¹ after 30, 40 and 50 min of infusion [11]. Cortical motor stimulation was undertaken 15, 30, 40 and 50 min after the start of the infusion and blood samples also were collected at each time point for measurement of propofol concentrations by HPLC with fluorescence detection [12].

Mean arterial pressure was measured at the same time as each MEP was recorded. Ventilation was adjusted to an end-tidal concentration of 4.0–4.5% carbon dioxide and rectal temperature remained greater than 35 °C throughout the study. At the end of the investigation the patient was taken into theatre and surgery commenced. The extradural electrode was used for the routine monitoring of the SEP during corrective surgery.

A typical MEP recorded in the extradural space is shown in figure 1. A well defined short latency, negative potential was observed in all patients. In a few patients there were two later lower amplitude components (I waves). The potentials were analysed for peak latency and peak-to-peak amplitude and the results expressed as median values and ranges. Statistical evaluation was undertaken using the Wilcoxon signed rank test, Mann-Whitney U test and Kendall's rank correlation coefficient (r) as appropriate.

**RESULTS**

Mean (SD) age and body weight were similar in the two groups (halothane 17.5 (3.0) yr and 61.5 (7.5) kg; propofol 16.2 (1.8) yr and 52.6 (6.3) kg). There was no significant change in amplitude or latency with time in each group, or a significant difference between groups (table I). Mean arterial pressure declined similarly in both groups, from 89 mm Hg in the baseline period in the halothane group to 73 mm Hg after 50 min, and from a baseline of 94 mm Hg to 80 mm Hg after 50 min in the propofol group. There was no significant correlation between the amplitude of the MEP and mean arterial pressure (r = −0.05).

Mean (SD) blood concentrations of propofol after 15, 30, 40 and 50 min were 7.14 (3.65), 4.36 (1.67), 4.23 (1.90) and 4.54 (1.82) µg ml⁻¹, respectively.

**DISCUSSION**

The results show clearly halothane 1 MAC had no significant effect on the amplitude and latency of the MEP recorded in the extradural space following electrical stimulation of the motor cortex. The derivation of this potential was examined in detail by Boyd and colleagues [8], who concluded that it represented the descending nerve volley in response to activation of corticospinal fibres.
asynaptic nature of the pathway was evident from the short latency (table I) and the potential did not appear to have a contribution from ascending sensory pathways.

During the course of this study, Katayama and colleagues [13] described the characteristics of the MEP recorded extradurally after direct stimulation of the motor cortex exposed during intracranial surgery. They stated that the response was resistant to anaesthesia and neuromuscular blocking drugs, although no details were given of the anaesthetic drugs used. The apparent lack of effect of anaesthesia on the MEP measured extradurally is an important advantage over the use of a peripheral recording method such as the EMG. The effects of anaesthetic agents on neuromuscular transmission are well recognized, and such interactions must alter the EMG response to motor stimulation. A possible improvement is the development of a modified "wake up" test during surgery in which the EMG is used to monitor motor pathways after cortical stimulation (Hugon, personal communication), but this requires further evaluation. At present, during surgery, the more direct method of recording motor cortical stimulation in the extradural space is preferable.

Most investigations have used electrical stimulation of the motor cortex. The magnetic stimulator was described first in 1985 by Barker, Jalinous and Freeston [14] who found that it was possible to produce an EMG in peripheral muscle by magnetic stimulation of the cortex. Magnetic stimulation is pain-free and so tolerated more easily than electrical stimulation in an awake patient. It has been found that the mean conduction time from scalp to muscle is shorter for electrical than magnetic stimulation [15], and it has been suggested that electrical stimulation results in both direct (D) and indirect (I) waves, whereas magnetic stimulation results in only later I waves [16]. The suitability of the magnetic stimulator for monitoring motor function during anaesthesia has yet to be evaluated. In the present study we observed I waves occasionally after electrical stimulation; they were of small amplitude and apparently unrelated to the anaesthetic technique.

In conclusion, the use of halothane up to 1 MAC end-tidal concentrations is unlikely to alter the interpretation of the MEP recorded in the extradural space. It is probable that other inhalation agents at equivalent concentrations behave similarly.

### REFERENCES


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**Table I. Median (range) amplitude and latency in patients receiving halothane or propofol**

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