It has been suggested that evoked potentials may be used to assess depth of analgesia/anaesthesia [1,2]. The late SSEP (> 50 ms) show close correlation with individual pain perception after the administration of centrally acting analgesics [3, 4]. We have evaluated the extent of afferent nociceptive input to higher cortical structures during various stages of prolonged (> 2 h) anaesthesia with nitrous oxide in oxygen plus propofol.

METHODS AND RESULTS

After obtaining informed written consent, we studied 15 patients (mean age 60 yr, mean weight 63 kg) undergoing elective abdominal surgery. Premedication comprised pethidine 0.5 mg kg\(^{-1}\), promethazine 1.0 mg kg\(^{-1}\) and atropine 0.01 mg kg\(^{-1}\) i.m, 45 min before anaesthesia. Anaesthesia was induced with propofol 2 mg kg\(^{-1}\) i.v. followed by suxamethonium 1.0 mg kg\(^{-1}\) to facilitate tracheal intubation; maintenance consisted of an infusion of propofol 100 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) (6 mg kg\(^{-1}\) h\(^{-1}\)) and 66% nitrous oxide in oxygen. Pancuronium was used to maintain neuromuscular block and ventilation was controlled to maintain normocapnia. Core temperature was measured by a probe in the oesophagus to verify that there was no decrease below 36°C.

Ten minutes before the end of anaesthesia, the propofol infusion was discontinued and the lungs ventilated with 100% oxygen.

For measurement of evoked potential from the right or left median nerve, silver-silver chloride cup-electrodes were placed at C\(_3\)-Fpz or C\(_4\)-Fpz, respectively (EEG 10/20 system). The ground electrode was placed around the right or left arm. Impedance was maintained less than 2 K\(\Omega\) at all times, by cleaning and abrading the skin at the site of application, the use of freshly chlorided silver-silver chloride cup-electrodes and application of sufficient electrode gel into the dome of the electrode.

The median nerve was stimulated at the wrist with a rectangular pulse of 5 Hz, 0.2 ms duration and a constant current 1 mA above motor threshold (Digi Stim II, Neuro Technology, Houston) using conventional ECG pregelled stick-on electrodes. Two hundred and fifty-six impulses were amplified, filtered (30-15000 Hz) and averaged with a Lifescan EEG monitor (Neurometrics, San Diego) using a sampling rate of 3.1 KHz.

After base-line preinduction values were recorded and replicated, median nerve evoked potentials were assessed during different intra-operative stimuli in prolonged (> 2 h) abdominal operations. SSEP were used to evaluate the extent of block of sensory nerve conduction at the following stages: preinduction; during steady state anaesthesia; during traction of the mesentery; 10 min after anaesthesia. Propofol 100 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) and nitrous oxide in oxygen anaesthesia induced a significant decrease in amplitude of the SSEP; noxious stimulation resulted in an increase in afferent nerve transmission and a concomitant increase of amplitude of the late evoked potential. After operation, impulse transmission recovered rapidly and the amplitude was similar to control at 10 min after anaesthesia.
FIG. 1. Mean amplitude height (stippled columns) and latency change of the $N_{100}$ peak (——) of somatosensory evoked potentials at different episodes. Significance compared with control.

potentials were recorded repetitively. The following episodes were compared: before induction (control); during steady state anaesthesia; during traction of the mesentery; and 10 min after anaesthesia.

The major negative deflection of the late evoked potential wave ($N_{100}$) was analysed in terms of peak to base amplitude ($\mu$V) and latency (ms) changes.

Data were analysed statistically by Friedman's rank analysis of variance followed by pairwise comparison of Wilcoxon and Wilcox. Significance was defined as $P < 0.05$.

Cortical responses during anaesthesia were depressed markedly as there was a significant ($P < 0.01$) decrease in amplitude height from a mean (SD) of 3.6 (2.7) to 1.4 (0.8) $\mu$V (fig. 1). Traction of the mesentery resulted in a significant increase in amplitude compared with steady state anaesthesia ($P > 0.05$) with a mean of 2.8 (1.1) $\mu$V and little change in latency. This increase was not significant in comparison with the amplitude height of the preoperative control period (fig. 1).

Ten minutes after the end of anaesthesia, there was a return of amplitude of evoked potential towards control (4.0 (2.5) $\mu$V) (fig. 1).

COMMENT

Our results demonstrate the effects of propofol-nitrous oxide in oxygen anaesthesia on SSEP signals, and the benefit of using the amplitude of the evoked potential to indicate reduction in depth of anaesthesia during the course of anaesthesia. Increase in amplitude during traction of the mesentery resulted from generalized CNS activation (arousal). Activation offsets the effects of anaesthesia/analgesia, and is followed by an increase in nerve traffic reaching higher cortical centres. As an increase in noxious stimulation increases the amount of afferent nerve potentials from the median nerve, the evoked peaks result in an increase in amplitude height at the cortical site. Thus evoked potentials demonstrate insufficient anaesthesia/analgesia.

The use of evoked potentials as a guide to depth of anaesthesia has been advocated by Sebel [1, 2] and others [4, 5]. Our data obtained 10 min after anaesthesia confirm this view. At this stage, amplitude height of the SSEP reached preanaesthetic values, at a time when the anaesthetic action was terminating.

The present data also demonstrate the potential benefit of propofol for prolonged (> 2 h) use. SSEP of patients after propofol reflected fast recovery of nervous conduction and little or no residual depressant effect in the immediate postoperative period. This recovery coincided with the time when patients became co-operative and orientated in time and space, in contrast to enflurane anaesthesia [6].

REFERENCES


