Processed electroencephalogram during combined extradural and general anaesthesia

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Summary

The processed electroencephalogram (pEEG) was monitored in eight patients undergoing gynaecological laparotomy under combined extradural and nitrous oxide–isoflurane anaesthesia. Pre-incisional mean spectral edge frequency 95 percentile (SEF95) and median frequency (MF) were 11.67 (SD 1.63) Hz and 3.74 (0.24) Hz, respectively. After skin incision, both SEF95 and MF decreased to 6.61 (2.04) Hz and 2.72 (0.32) Hz, respectively ($P<0.001$). An increase in mean arterial pressure after incision suggested inadequate depth of anaesthesia. After introduction of extradural analgesia, these variables returned to pre-incisional values (SEF95 11.65 (1.73); MF 4.02 (0.41)). Reduction of end-tidal isoflurane from 1.0% to 0.5% after extradural analgesia did not cause significant pEEG changes. pEEG may assist anaesthetists to recognize adequacy of combined general–extradural anaesthesia. (Br. J. Anaesth. 1997; 78: 751–753).

Key words


Extradural analgesia has often been combined with light general anaesthesia in major surgery. The sympathetic block produced by extradural analgesia with the negative inotropic action of general anaesthetics may cause further cardiovascular depression. Therefore, it is advisable to avoid a high dose of general anaesthetics in order to prevent haemodynamic derangements.

However, even under adequate extradural analgesia, it is not known if patients remain unconscious during light general anaesthesia. Despite anaesthetists’ concerns about intraoperative awareness, particularly in paralysed patients, there have been no studies of intraoperative EEG under combined general–extradural anaesthesia.

Methods and results

After oral premedication with ranitidine 150 mg, 2 h before anaesthesia, an extradural catheter was sited at L2–3 or L3–4. Lignocaine 1% (2 ml) was used for local infiltration anaesthesia. Extradural test doses consisted of 3 ml of 1% lignocaine with adrenaline 1:200 000. No further local anaesthetic was given before induction of anaesthesia with thiopentone 5 mg kg$^{-1}$ i.v. Vecuronium was administered to facilitate tracheal intubation and surgical exposure. Positive pressure ventilation was adjusted to maintain normocapnia (end-tidal carbon dioxide partial pressure of 4.5–5.0 kPa), measured with an anaesthetic gas monitor (Capnomac Ultima, Datex, Helsinki, Finland). Non-invasive arterial pressure, electrocardiogram, oxygen saturation, urine output and rectal temperature were monitored.

After induction, the lungs were ventilated with 1.0% end-tidal isoflurane and 65% nitrous oxide in oxygen. We waited 15 min after intubation before the start of surgery (phase 1). During the first 15 min after incision, anaesthesia was continued with the same concentration of inhalation agents as above (phase 2). Then 1.5% plain lignocaine 20 ml were administered extradurally in 5-ml incremental doses in 6 min and inhalation anaesthesia was maintained as above for another 15 min (phase 3). The end-tidal concentration of isoflurane was then reduced to 0.5% with 65% nitrous oxide in oxygen and 15 min elapsed until the end of the study (phase 4).

A Dräger pEEG monitor (Dräger AG, Lübeck, Germany) was used to monitor continuously pEEG. Four disposable electrodes were placed over the frontal and mastoid areas bilaterally. A reference electrode was placed in the frontal midline. Impedance of the electrodes was measured every 3 min and maintained below 5 kΩ throughout the study. Conventional frequency bands were used to describe power spectrum variables. Epoch length of EEG acquisition was 2 s. pEEG data were, recorded onto a PC-compatible computer showing the trend of spectral edge frequency (SEF) and raw EEG waveform, and on postoperative replay the trace of raw EEG was inspected to ensure that artefacts caused by diathermy were excluded in the off-line analysis.

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For statistical analysis, the pEEG data were averaged from 10 consecutive epochs in the last 5 min of each study phase. Mean arterial pressure and heart rate were expressed as percentage of baseline values.

One-way repeated measures ANOVA followed by Tukey’s multiple comparison was performed using StatView 4.0 (Abacus Concepts, Inc., Berkeley, CA, USA).

There were no differences between the four phases for end-tidal carbon dioxide concentration, oxygen saturation or rectal temperature.

After surgical incision, SEF95 and MF decreased in all eight patients (table 1). After extradural analgesia, these pEEG variables returned to pre-incisional values. There was no significant change in pEEG variables after reduction of end-tidal isoflurane concentration from 1.0% to 0.5%.

The only significant haemodynamic change was an increase in mean arterial pressure after incision.

**Comment**

pEEG shifted towards slower waves after incision in all eight patients. Intraoperative EEG slowing has been considered to reflect increased depth of anaesthesia. However, a significant increase in arterial pressure after incision suggests that anaesthesia at that time was not adequate enough to suppress sympathetic responses.

EEG slowing associated with clinical arousal phenomena caused by surgical stimulation is termed “paradoxical arousal”.1 Controversy exists as to the characteristic EEG changes induced by surgery. Sensory stimulation may induce a shift of the dominant EEG frequency towards faster waves,2,3 which is similar to what occurs during emergence from anaesthesia.4 On the other hand, Bimar and Bellville described that EEG patterns, as either increases in high-frequency or low-frequency activity, may represent intraoperative arousal.5 EEG responses with increases in low-frequency activity were most prominent at frontal areas.6 In our study, electrodes were placed in the bilateral fronto-mastoid areas, thus reflecting mostly electrical activity in the frontal cortex. This may be the reason why “paradoxical arousal” patterns were identified clearly.

All pEEG variables returned to pre-incisional values after extradural analgesia. It may be criticized that it is impossible to assess the efficacy of extradural analgesia in anaesthetized patients. However, with the general–extradural technique, anaesthetists rely on haemodynamic changes to decide on the timing of extradural dosing and assess its effectiveness. Mean arterial pressure decreased significantly after administration of extradural lignocaine. Mean blood loss at that point was less than 50 ml. Therefore, it is likely that the decrease in arterial pressure in phase 3 resulted from effective extradural analgesia. Our results suggest that the “paradoxical arousal” phenomenon disappeared after achievement of analgesia.

Although there was a slight increase in SEF95 after reduction of end-tidal isoflurane from 1.0 to 0.5%, the difference was not statistically significant. Drummond and colleagues identified a threshold value for SEF of 13.6 Hz which provided separation from surgical anaesthesia to pre-arousal state during emergence from isoflurane–nitrous oxide anaesthesia.4 Mean SEF95 in phase 4 was less than this threshold value and none of our patients had intra-operative recall. In view of the duration of effect of 1.5% lignocaine, it is unlikely that extradural anaesthesia was wearing off in phases 3 and 4. We assume that when afferent noxious stimuli are interrupted by extradural analgesia, a modest reduction in dose of inhalation agent may not cause significant changes in pEEG variables, although the possibility of low statistical power resulting from small numbers of subjects cannot be excluded.

Cardiovascular variables have been the only variables available to assess the efficacy of extradural analgesia in anaesthetized, paralysed patients. There was a significant change in mean arterial pressure after incision and also after extradural analgesia. It may thus be argued that in these healthy patients, pEEG did not provide any additional information that was not readily apparent from haemodynamic observations. However, intraoperative haemodynamic state can also be affected by other factors.
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apart from depth of anaesthesia, such as volume status and concomitant cardiac medications. Consequently, it may be of clinical significance that pEEG variables showed significant changes after incision and extradural analgesia.

Acknowledgement

We thank Dräger Japan Ltd for loan of the pEEG monitor.

References


