EFFECTS OF 2% LIGNOCAINE ON SOMATOSENSORY EVOKED POTENTIALS RECORDED IN THE EXTRADURAL SPACE

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SUMMARY

We have studied the effects of extradural administration of 2% lignocaine at the L3-4 interspace on somatosensory evoked potentials recorded in the cervical extradural space before corrective surgery for idiopathic adolescent scoliosis. Eight patients in whom the equivalent volume of 0.9% sodium chloride solution was administered into the lumbar extradural space acted as a control group. Lignocaine 2% resulted in a decrease in overall amplitude, but its main effect was a significant decrease in the amplitude of the second and third peaks, suggesting an action on the dorsal columns of the spinal cord. A significant increase in latency was found in both the lignocaine and sodium chloride groups. We conclude that the use of extradural 2% lignocaine in patients undergoing scoliosis surgery may interfere with the intraoperative monitoring of somatosensory evoked potentials.

KEY WORDS


Monitoring of the somatosensory evoked potentials (SEP) to posterior tibial nerve stimulation is the method of choice for assessing the integrity of the spinal cord during scoliosis surgery [1]. Despite the reliability of this method over several years [2, 3], there are instances of neurological damage occurring without any change in the SEP [4]. Thus a condition of anaesthesia for scoliosis surgery is that the patient is awake and cooperative soon after completion of the surgery so that any neurological deficit may be detected and appropriate action taken. Additional requirements include the provision of controlled hypotension and alleviation of severe postoperative pain.

The use of extradural analgesia would meet these anaesthetic considerations for scoliosis surgery if local analgesics did not interfere with the intraoperative monitoring of the SEP. Therefore, in the present study we have examined the effects of 2% lignocaine 10 ml on the SEP to posterior tibial nerve stimulation recorded in the extradural space. We chose this large dose of lignocaine, 200 mg, to determine the maximal effect on the SEP.

PATIENTS AND METHODS

We studied 24 patients (21 female, three male) 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 with a Cobb angle between 30° and 70° as assessed by plain erect x-rays. The study was approved by the Hospital Ethics Committee and written, informed consent was obtained from the patients, or the parents, as appropriate. Patients were allocated randomly to one of three groups. A control group received a general anaesthetic without extradural analgesia. A second group received the same general anaesthetic together with 2% lignocaine extradurally and a third group received the same...
general anaesthetic and extradural 0.9% sodium chloride solution.

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. Anaesthesia was induced with propofol 2.5 mg kg\(^{-1}\) and tracheal intubation facilitated with pancuronium 0.08 mg kg\(^{-1}\). The lungs were ventilated with 70% nitrous oxide in oxygen and a radial artery cannulated for the measurement of arterial pressure. The patients were placed in a prone position on a Montreal mattress and the extradural space located at the C7–T1 interspace using a 15-gauge modified Tuohy needle and the hanging drop technique. A 3-French gauge bipolar recording electrode was passed through the needle cephalad and connected to the preamplifier of a Medelec MS 91 electromyograph recorder.

The right and left posterior tibial nerves were stimulated alternately via skin surface electrodes using square-wave impulses of 0.2 ms duration at an intensity of 150 μV. The stimulation frequency was 20 s\(^{-1}\) and a minimum of 750 responses were averaged with a resolution of 0.033 ms. After the measurement of the baseline SEP those patients in the extradural groups received either 2% lignocaine 10 ml at the L3–4 interspace (extradural lignocaine group) or 0.9% sodium chloride 10 ml at the L3–4 interspace (extradural saline group) using a single-shot technique. A three-stage infusion of propofol was started in all patients: 21 mg kg\(^{-1}\) h\(^{-1}\) for 5 min, 12 mg kg\(^{-1}\) h\(^{-1}\) for 10 min and 6 mg kg\(^{-1}\) h\(^{-1}\) thereafter. This infusion regimen has been shown to result in blood concentrations of propofol of approximately 4 μg ml\(^{-1}\) at 30–50 min after the start of the infusion [5].

SEP measurements from the cervical extradural space to alternate posterior tibial nerve stimulation were made 30, 40 and 50 min after the extradural injection (extradural lignocaine and saline groups) or start of infusion of propofol (propofol control group). At each time point three SEP, each of 750 responses, were made from each leg and mean arterial pressure was measured. Ventilation was adjusted to maintain an end-tidal carbon dioxide concentration of 3.5–4.0% and the rectal temperature remained greater than 35 °C.

In the extradural lignocaine group, Hartmann’s solution was infused i.v. to maintain a mean arterial pressure greater than 70 mm Hg. At the end of the investigation the patient was taken into theatre and surgery commenced. The extradural electrode was then used to monitor the SEP during the corrective surgery.

The evoked potentials were analysed for first peak latency, overall amplitude and amplitude of the first, second and third peaks. The neurophysiological data are presented as medians and ranges; because of the skewed distribution of the data, statistical evaluation was undertaken using the Wilcoxon signed rank test and the Mann–Whitney U test.

**RESULTS**

Mean (SD) age and body weight were similar in the three groups: extradural lignocaine 18.5 (4.4) yr and 52.7 (4.0) kg; extradural saline 16.7 (3.2) yr and 51.3 (10.5) kg and propofol control 18.4 (4.2) yr and 54.8 (7.9) kg.

There was no significant difference between the extradural saline and propofol control groups for any of the neurophysiological variables. For the sake of clarity, therefore, data are presented only for the extradural lignocaine and saline groups.

Typical SEP after the extradural administration of 2% lignocaine 10 ml are shown in figure 1. The important changes were a decrease in overall amplitude and second and third peak amplitudes. These were present after 30 min and sustained at 0.4 μV for 3 ms after 40 min.

![Fig. 1. Effects of 2% lignocaine 10 ml on the somatosensory evoked potential to posterior tibial nerve stimulation recorded in the cervical extradural space. A: Baseline; B: 30 min after lignocaine; C: 40 min after lignocaine; D: 50 min after lignocaine.](http://bja.oxfordjournals.org/)

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40 and 50 min. There was little change in first peak amplitude.

**Overall amplitude**

Overall amplitude decreased after administration of lignocaine (table I), but this change was significant only for the right leg at 30, 40 and 50 min \( (P < 0.05) \). Extradural saline had no effect on overall amplitude; a significant difference between the extradural groups was found for the right leg only at 30 and 50 min \( (P < 0.05) \).

**Individual peak amplitudes**

There was no significant effect of lignocaine on the amplitude of the first peak and no significant differences between the groups (results not shown). Extradural lignocaine resulted in a significant decrease in second peak amplitude in both
legs (left leg, \( P < 0.05 \) at 30 min and \( P < 0.01 \) at 40 and 50 min; right leg, \( P < 0.05 \) at 30 and 40 min) (table II). There was a significant difference between the extradural lignocaine and saline groups for the left leg at 40 and 50 min (\( P < 0.05 \)) and for the right leg at 30, 40 and 50 min (\( P < 0.05 \)). A similar pattern of change was seen for the third peak amplitude (table III). Lignocaine produced a significant decrease in amplitude for the left leg at 30 and 50 min (\( P < 0.05 \)) and 40 min (\( P < 0.01 \)), but only at 50 min (\( P < 0.05 \)) for the right leg. Again this resulted in a significant difference between the groups at 30, 40 and 50 min for the left leg (\( P < 0.01 \)) and for the right leg only at 40 min (\( P < 0.05 \)).

Latency increased significantly (\( P < 0.05 \)) throughout the study in all groups at 30, 40 and 50 min and there was no significant difference between the groups at any time (table IV). Mean arterial pressure decreased similarly in the three groups. During the period of measurement of the SEP mean arterial pressure was between 76 and 79 mm Hg in the extradural lignocaine group and between 80 and 83 mm Hg in the extradural saline group. There was no significant difference between the groups.

### DISCUSSION

The major finding of the study was that administration of 2% lignocaine 10 ml into the lumbar extradural space resulted in a decrease in the SEP recorded in the cervical extradural space. The L3-4 site was chosen so that the maximal effect of lignocaine on the stimulation of the posterior tibial nerve could be studied. Although overall amplitude decreased, the most important feature was a decline in amplitude of the second and third peaks. The first peak of the SEP represents conduction in the anterolateral tracts at 65-80 ms^{-1}, while the later two peaks represent slower transmission in the dorsal column [2]. Our results suggest that extradural lignocaine depressed conduction in the dorsal column selectively. This is surprising, as dorsal column transmission is largely asynaptic, whereas the first peak, which was unaffected, involves some synaptic transmission. It is possible that the apparent selectivity of lignocaine for the dorsal column reflected a direct effect of the drug on superficial sites in the spinal cord. It is noteworthy that 2% lignocaine did not abolish the SEP in any patient, indicating that afferent sensory transmission was preserved even when sufficient lignocaine was given to achieve good clinical analgesia.

The only comparable clinical work is that of Saugbjerg and colleagues [6], who investigated the effects of 0.5% bupivacaine 10 ml injected into the lumbar extradural space on the SEP, recorded using scalp electrodes, to posterior tibial nerve stimulation. Their results suggested that bupivacaine suppressed the early components of the SEP and increased the latency of the first peak. As the first peak of the SEP recorded cranially is considered to represent dorsal column activity [7], our data on amplitude are compatible with the findings of Saugbjerg and colleagues [6]. However, they studied only six patients and did not include a control group. The lack of a control group is of particular importance, as these authors noted an increase in first peak latency after bupivacaine. In the present study, we observed a similar increase in latency in all three groups, which was related possibly to the slow cooling of
the spinal cord [8]. Thus changes in latency of the SEP cannot be ascribed to the actions of a local analgesic in the absence of a suitable control group.

We observed a greater effect of lignocaine on the second and third peak amplitudes after left leg rather than right leg stimulation (tables II and III). This laterality of effect was noted in a previous study examining the action of halothane on the SEP [9] and has been described by others also [10]. It is likely that this finding reflects the surgical model studied, idiopathic adolescent scoliosis, in which the spinal curve is almost invariably concave to the left. The possible occurrence of this laterality in scoliosis patients is missed if only one leg is stimulated or both legs stimulated concurrently [11].

The most likely explanation for the effects of lignocaine in modulating the SEP is that the local analgesic inhibits afferent transmission in the nerve roots. It is possible that lignocaine also alters the evoked potential by decreasing spinal cord blood flow. Experimental studies in dogs have yielded equivocal data, with either no effect or an increase in cord blood flow after subarachnoid lignocaine [12, 13], and a decrease in blood flow after extradural lignocaine [14]. In the study of extradural lignocaine Mitchell and colleagues [14] found that a loss of the SEP occurred concurrently with a decrease in cord blood flow in the lumbar region. However, their data also show that there was no difference in cord blood flow between dogs given either extradural lignocaine or saline. We consider it unlikely, therefore, that our results may be attributed to a direct effect of lignocaine on spinal cord blood flow.

For extradural lignocaine to be used in patients undergoing scoliosis surgery, this technique must not interfere with the intraoperative monitoring of the SEP. A loss of more than 50% of overall amplitude, or the complete loss of one peak, is used commonly to indicate neurological damage [1]. The peaks were always present after lignocaine, although significantly decreased, but three of the eight patients showed a decrease in overall amplitude greater than 50% at some point during the study. It is probable that this represents the maximal effect of extradural lignocaine 200 mg on the SEP to posterior tibial nerve stimulation, and that lignocaine may have less effect at smaller concentrations and other sites such as the mid-thoracic region.

REFERENCES