


Sir,—Previously, we have used both laser and thermodc stimuli and concluded that the distinct, first pain perception, evoked by brief argon laser stimuli differs in quality and intensity from the pain elicited by a slowly heating thermode. We have compared the effect of local anaesthetics on both laser and thermode thresholds and found a different effect on the two thresholds [1]. This discrepancy may be explained by: activation of different nerve fibre populations (Aδ- and C-fibres); a different number of receptors activated (spatial summation); a different duration of receptor activation (temporal summation); and the fact that lasers do not cause concurrent activation of mechanosensitive afferents as a touching thermode. Furthermore, brief cutaneous laser stimuli cause a very fast increase in intracutaneous temperature which is known to generate high frequency bursts of activity from nociceptors. From a physiological point of view, lasers seem to be adequate for cutaneous pain stimulation. Traditional pinpricks also cause bursts of neuronal activity, but this stimulus cannot be controlled or quantified. Pain perceptions evoked by laser and pinprick stimuli are similar in quality, but different from pain elicited by thermodcs. It is, therefore, not surprising that different results are obtained by different stimulation techniques.

The characteristics of the long latency pain-evoked potential are similar to long latency (vertex) potentials evoked by other sensory modalities. These vertex potentials are sensitive to changes in vigilance and hence the degree of sedation. The modulation of pain-evoked potentials by thiopentone or propofol therefore, may be caused by both an anaesthetic and a sedative effect, and the contribution of the individual factors could not be separated. It can be argued that an overall measure which is sensitive to both anaesthetic and sedative effects may be a good correlate with clinical practice, in which pain alleviation often is obtained by a combination of both factors. The purpose of the present study was to elucidate if single subhypnotic doses of thiopentone or propofol caused hyper- or hypoalgesia to acute pain. If hyperalgesia was obtained, we would expect to see an increase in the size of the potential [2].

The latency of the laser-evoked potential is not affected by sedation [3] and therefore we have proposed this variable for quantification of peripheral and central conduction properties along the pain pathway [4, 5]. As this information cannot be obtained by threshold determinations, we always use both evoked potentials and thresholds for assessment of anaesthetic efficacies.

The use of laser-evoked potentials and pain thresholds in anaesthesia is a fairly new approach, and investigation of how these measures respond to various interventions along the pain pathway enables us to determine their applicability and validity for testing and comparing anaesthetic procedures and substances.

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REFERENCES


INCREMENTAL SPINAL ANAESTHESIA AND CAESAREAN SECTION—RELEVANCE TO THE TEST DOSE FOR EXTRADURAL ANALGESIA

Sir,—It is possible to administer an otherwise catastrophic dose of bupivacaine into the subarachnoid space of a pregnant woman at term without ill effect, provided she is kept static. Kestin and colleagues demonstrated this in their study of incremental spinal anaesthesia for Caesarean section [1]: one of their mothers required bupivacaine 37 mg intrathecally to achieve effective anaesthesia. In the next issue of the Journal, Randall and colleagues, in their study on spinal anaesthesia for Caesarean section obtained a large number of high blocks with bupivacaine 12.5 mg [2]. Their mothers were subjected to a change of posture after a single spinal injection: the mothers were moved from full left lateral through supine to 15° right tilt. This manoeuvre produces a brief period of inferior vena caval (IVC) compression sufficient to cause acute engorgement of the extradural veins, and a cephalad surge of CSF results. Whatever the extent of this surge, it is enough to add a significant impetus to the spread of administered drugs intrathecally. It has been shown that IVC compression is essential to produce good filling of the extradural veins during lumbar extradural venography [3].

All the evidence [1—3] continues to support Russell’s hypothesis that sudden diversion of blood into the vertebral venous system decreases the space available for CSF at the caudal end of the subarachnoid space [4]. This dynamic effect of IVC compression is in addition to any influence on drug distribution of the reduced lumbosacral CSF volume caused by sustained congestion of the adjacent extradural veins. In this regard, it should be noted that IVC compression is not necessarily relieved adequately by tilt in some individuals.

The above observations suggest that the test dose for accidental intrathecal placement of the extradural catheter