A703

TITLE: A SPINAL MODEL STUDY OF LOCAL ANESTHETIC DISTRIBUTION: AN EXPLANATION FOR CAUDA EQUINA SYNDROME FOLLOWING CONTINUOUS SPINAL ANESTHESIA.

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We recently described four cases of cauda equina cauda equina syndrome following continuous spinal anesthesia.[1] In all four, a dose of local anesthetic greater than that usually used with a single-injection technique had been administered incrementally to extend a predominantly sacral block to achieve adequate anesthesia. We postulated that these deficits resulted from maldistribution of anesthetic, causing a direct neurotoxic effect of the local anesthetic. The present studies were conducted to determine the concentration of local anesthetic which might result if the anesthetic were injected through a catheter which was directed sacrally in the subarachnoid space.

A model of the subarachnoid space was constructed from an acrylic tube (I.D. 1.8 cm). Sampling ports were placed along the "ventral" surface of the model, and one port at the sacral end. A single injection port was placed on the "dorsal" surface at the peak of the lumbar lordosis. The model was filled with artificial cerebrospinal fluid (CSF). The injected solution was a 20:1 mixture of 5% lidocaine hydrochloride (Astra Pharmaceutical) and 1% methylene blue. A 28-gauge catheter (CoSpan, Kendall Healthcare) was placed in the model and advanced 3.5 cm sacrally. One ml of the local anesthetic solution was injected over 60 s. Three min later, 0.3-ml samples of CSF were aspirated from 8 sample ports at intervals of 4 cm, beginning at the sacral tip (port 1). After refilling the model with CSF, three sequential 1-ml injections were made 5 min apart. Three min after the last injection, CSF was again sampled. The absorbance of each sample was measured at 675 nm and compared with the absorbance of the injected solution. The samples from the most sacral port were also assayed for lidocaine concentration using an immunoassay technique.

The pattern of distribution following the single and the three sequential injections was restricted sacrally with the highest levels occurring at the most sacral ports. The peak lidocaine concentration from the single injection was 1.4%, and from the sequential injections, 3.4%.

Administration of hyperbaric local anesthetic through a sacrally-directed catheter resulted in a limited distribution of anesthetic with a relatively high peak concentration. Clinically, such restricted distribution would result in a limited sacral block. If additional doses of local anesthetic are administered with the same technique, local anesthetic will distribute in the same pattern, reinforcing the area with the highest concentration. The concentration achieved after three sequential injections of lidocaine exceeded that capable of causing neurotoxic damage in some animal models.[2]

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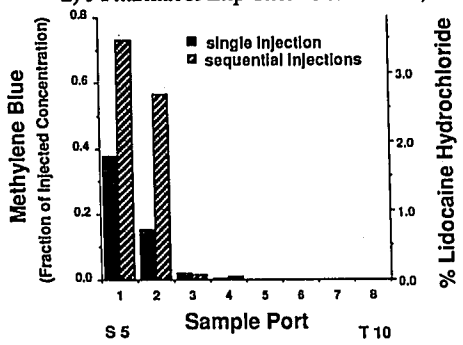
References: 1) Anesth Analg 72:275-81, 1991
2) J Pharmacol Exp Ther 250:406-413, 1989



A704

TITLE : **Continuous spinal anesthesia : the rationale for associating meperidine to lidocaine during the induction phase.**

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Continuous spinal anesthesia (CSA) allows to adequatly control the level of the sensory and motor blokade and decreases the likelihood of cardiovascular instability during induction (1). It was also postulated a synergistic analgesic effect of local anesthetic agents and opiates (2). The aim of this study was to compare the quality of CSA with 1.6 lidocaine when induction is performed with or without meperidine.

After obtaining institutional approval, 24 elderly patients (80.5 ± 6.2 years) who underwent orthopedic surgery for fracture of the neck of the femur were randomly assigned in a double blind fashion into tow groups. In the first group (n=10) CSA (22 G pencil point needle, 27 G catheter, L4-L5 space) was induced with lidocaine 1.6% alone whereas in the second one (n=14) it was associated with meperidine 1%. The adequate induction dose (ie : sensitive block at L2 level (pin-prick) and motor block grade II-III (Bromage Scale)) was determined by an initial injection of 1 ml followed by additional doses of 0.5ml injected iteratively every 5 minutes. Then every 45 minutes one third of the initial volume of the induction dose was reinjected using exclusively 1.6% lidocaine in the two groups. Blood pressure (BP) and heart rate were recorded every 5 minutes and blood gas were analyzed before and one hour after induction. Bleeding was exactly compensated with a 4% albumine solution. Ephedrine was injected when the amplitude of the fall in systolic BP was greater than 20% of the preoperative control values. Finaly post operative analgesia was assessed from the delay before requirement for pain medication. Results are given as mean ± SD and compared using unpaired Student t-Test.

There was no statistical difference between the groups with respect to age, weight, bleeding, duration of surgery and change in PaO2 and PaCO2 values. In the lidocaine + meperidine group ephedrine was required for 5 patients (see table) because of impairments in haemodynamic stability. In the lidocaine group, ephedrine was not used at all but in 8 out of the 10 patients, earlier reinjections of drug were administered because of pain (mean delay between injection : 35 ± 9 min). In addition, drowsiness (13/14) was observed only in the lidocaine + meperidine group.

It is concluded that although association of meperidine with 1.6% lidocaine during the induction of CSA slightly impairs haemodynamics stability it also i) allows to decrease the initial induction dose ii) increases the delay of reinjection iii) produces a satisfactory drowsiness iv) induces a long lasting pain relief.

	lidocaine Group n=10	lidocaine + meperidine Group n=14
Induction dose(mg)		
lidocaine	36.8±43	28±3.9*
meperidine	0	17.8±10.2
Ephedrine requirement (number of patients)	0/10	5/14
• during induction	0/10	3/14
• during reinjection	0/10	3/14
Delay before pain medication (hours)	2.4 ± 2	13.3 ± 5.8*

*p < 0.01

1) Anesth. Analg. ; **70** : 97-102, 1990.
2) Anesth. Analg. ; **67** : 943-8, 1988.