

TITLE : EFFECT OF CLONIDINE PREMEDICATION ON POSTANESTHETIC RECOVERY

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Clonidine given preoperatively has been reported to reduce anesthetic requirements and to improve intraoperative hemodynamic stability (1). Since clonidine has sedative properties due to its alpha 2 agonist effect (2), one may be concerned that postanesthetic recovery would be prolonged after clonidine administration. The present study was designed to assess this point.

48 ASA I-II patients scheduled for thyroid surgery were included in a double blind study after institutional approval and informed consent. Patients were allocated randomly in three groups. 2 hours before surgery they received as oral premedication, either 1 mg flunitrazepam (group 1) or 150 mcg clonidine (group 2) or 1 mg flunitrazepam plus 150 mcg clonidine (group 3). Anesthesia was induced with thiopental (5mg.kg⁻¹) alfentanil (20 mcg.kg⁻¹) and vecuronium (0.1mg.kg⁻¹) and maintained with nitrous oxide 70 % in O₂ and 0.6 % end-tidal isoflurane. Additional alfentanil bolus doses (0.1 mg.kg⁻¹) were administered if systolic arterial blood pressure (SAP) increased up to 180 mmHg while isoflurane was discontinued if SAP decreased below 90 mmHg. Recovery was assessed with mean motor reaction time to 50 consecutive auditive stimulations (MART) measured with a micro-computerized device (Canon X07) and electro-oculographic measurements (EOG) (Gould Universal Amplifier : 13-4615-

58) as previously described (3). Measurements were performed prior and 30, 60, 120, 240 min after arrival in the recovery room. Statistical analysis used Wilcoxon test and paired t-test. Sixteen patients were included in each group. Demographic data and surgical procedures were comparable in the three groups. 19 Patients experienced high SAP which required alfentanil supplementation (10 in group 1, 3 in group 2 and 6 in group 3), and 3 patients (1 in each group) experienced low SAP leading to discontinue transiently isoflurane. Results are reported in table 1.

This study documents that 150 mcg preoperative clonidine does not result in delayed recovery compared to flunitrazepam premedication.

Table 1:

	J-1	30min	60min	120min	240min
MART I (s.10 ⁻²)	26.2±9.05	39.2±10.2	33.3±8.5	29.9±7.0	25.9±6.9
	24.9±5.5	33.7±9.5	30.5±10.3	29.2±9.1	25.6±9.2
	22.6±4.3	44.4±13.8*	35.8±10.7	28.1±7.1	26.4±9.2
EOG I meantSD II (°.sec ⁻¹)	266±52	135±58	161±63	193±59	219±49
	267±16	199±64	189±54	203±40	229±35
	249±38	162±69	165±46	190±63	214±39

*significant difference from group 1

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TITLE: IMPLANTATION OF SPINAL CORD STIMULATORS USING SPINAL ANESTHESIA

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The site of action of spinal anesthetics on the neuraxis is speculative. The phenomenon of differential axial blockade suggests a predominant effect of local anesthetics on spinal rootlets(1), however, this does not exclude a direct effect on the spinal cord. We assessed effects of spinal anesthesia on cord conduction while implanting spinal cord stimulators (SCSs) for treatment of chronic low back and leg pain.

We report results with 19 patients (mean age 54 years) who had SCSs placed using spinal anesthesia. Heretofore, spinal anesthesia has not been used for SCS implantation, due to the widespread belief that it will abolish stimulation-induced paresthesias, which are used intraoperatively to refine electrode position. To be effective, paresthesias produced by the stimulator must coincide with the area of chronic pain. Patients were admitted to the hospital for elective implantation of Neuromed or Medtronic stimulators. Written informed consent was obtained from all patients and the study protocol was approved by our institutional review board. Spinal anesthesia was used for SCS lead tunneling and generator placement. Lidocaine was administered intrathecally, in single or divided doses (mean 110 mg); one patient received 7.5 mg of tetracaine. Isobaric and hyperbaric formulations were used. Anesthetics were well-tolerated by all patients and resulted in complete motor blockade of the legs and anesthetic sensory levels extending to upper thoracic dermatomes (T8 to T1). Operations were carried out in the prone position after the anesthetics

were established. SCS electrodes were positioned at T10 under fluoroscopy.

All patients felt trial stimulator-induced paresthesias during spinal anesthesia. In 8 patients, cortical evoked potential (CEP) monitoring with a Nicolet CA 1000 or Pathfinder II was used to further assess cord conduction during spinal anesthesia. CEPs and simultaneous paresthesias induced by direct cord stimulation were obtained, despite an anesthetic sensory level 3 to 9 segments cephalad to the stimulating electrode. Tibial nerve evoked potentials recorded at the scalp or cord (recording from the SCS electrode) were abolished. In all but one patient, spinal anesthesia temporarily abolished the chronic pain. Fourteen patients have had good results with SCS (average follow-up of 9 months).

A predominant site of action of spinal anesthetics appears to be at spinal rootlets since tibial nerve evoked potentials recorded at the scalp or spinal cord were abolished and induced paresthesias (thought to represent dorsal column function) were maintained in the presence of spinal anesthesia. However, an effect on the dorsal root entry zone can not be excluded(2). In addition, there can also be a direct but less pronounced anesthetic effect on the cord itself, since blunting of cord to scalp CEPs was observed in 3 patients.

Disappearance of chronic pain under diagnostic spinal anesthesia does not prove that pain originates in the cord(3), but pain persisting after CEPs are abolished would suggest a supersegmental level of origin. Use of spinal anesthesia for SCS implantation improved patient comfort and cooperation, reduced the need for sedation and facilitated electrode positioning.

References:

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