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TITLE: COMPARISON OF INTRA-ARTICULAR BUPIVACAINE AND MORPHINE FOR ANALGESIA FOLLOWING ARTHROSCOPIC KNEE SURGERY

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Behavioral and neurophysiological studies in animals have shown that the local administration of opiates at an inflammatory site results in analgesia.[1] We wished to determine if such a peripheral opiate receptor-mediated analgesia could be demonstrated in man. We compared the analgesic efficacy of local anesthetics and morphine administered intra-articularly (ia) at the end of arthroscopic knee surgery.

Forty-seven patients scheduled for arthroscopic knee surgery by a single surgeon participated in this institutionally approved, randomized, double-blind study. Anesthetic regimen consisted of lumbar epidural analgesia using 2% lidocaine with 1:200,000 epinephrine (E) to a mean sensory level of T_{10} (T_8 to T_{12}). No systemic or epidural narcotics were administered in the intraoperative period. Patients were randomized to receive 20 ml ia of 0.9% saline with 100 ug E [Group (Gp) 1,n=16], or 0.25% bupivacaine with 100 ug E [Gp 2,n=15], or 3 mg morphine sulfate (MS) and 100 ug E in normal saline [Gp 3,n=16]. Postoperative pain (using visual analog scores, VAS) and the level of residual sensory and motor blockade were monitored by a blinded observer at hourly intervals until discharge. Subsequent VAS of pain were completed by patients for 24 hr at 2-4 hr intervals. Patients' first request for pain medication and analgesic use during the 2-3 day post-op period were monitored. A repeated-measures ANOVA was used to compare VAS scores and the least significant difference method used for comparison of means.

The patient demographics were similar in all 3 groups. VAS scores were similar in the groups preoperatively and on arrival in the recovery room. In the first two postoperative hours the residual sensory blockade was minimal and similar in the 3 groups, but the bupivacaine group [Gp 2] had significantly lower ($p < 0.05$) VAS scores than the morphine group [Gp 3]. Subsequent VAS scores were not statistically significantly different in the 3 groups (see figure, values are mean ± sem). While only 3 of 15 patients in Gp 2 requested analgesics during the first 2 postoperative hours, ten of 16 patients in Gp 3 required systemic analgesics. Oral analgesic (acetaminophen-oxycodone) consumption during the 48-72 hours postop. was similar in the 3 groups.

These data suggest that, following arthroscopic surgery under epidural anesthesia, intraarticular morphine failed to provide significant analgesia. Intraarticular local anesthetics may provide better pain relief in the immediate postop. period.

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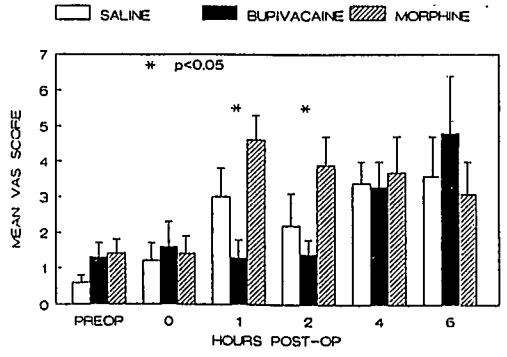

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TITLE: ABSORPTION OF LOCAL ANESTHETICS VIA VASCULAR WALL: *IN VITRO* STUDY

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Lidocaine is considered to have a high therapeutic index and it is very commonly used in the practice of anesthesiology for local infiltration, nerve blocks, regional blocks and arrhythmia management. Large quantities of lidocaine are instilled around major neurovascular bundles during nerve blocks. The drug is absorbed and eliminated without subsequent effects most of the time[1,3]. Side effects are related to the serum levels of lidocaine and can vary from mild sedation to convulsions, coma and cardiovascular collapse[2,4]. Loss of consciousness and/or convulsions have been occassionally observed during or soon after injection in the head and neck region[4]. This has been commonly attributed to rapid venous absorption and/or direct intravascular injection.

We observed a few instances where such symptoms developed transiently during an interscalene block. There was no apparent intravascular injection nor were they associated with elevated serum lidocaine levels. The possibility of direct absorption through the carotid vertebral arterial system leading to a transient increase in lidocaine concentration in the brain was entertained. We found no data available in the scientific literature in this regard.

The aim of the study was to evaluate the absorption of lidocaine through arteries and veins in an *in vitro* model.

The study was approved by the Institutional Research Committee. Carotid arteries and jugular veins were harvested from dogs used in other experiments and immediately placed in physiological solution at 37°C. From 2.5cm to 3cm segments of each vessel were isolated and incorporated into a closed loop perfusion systems with a roller pump. The vascular segments were perfused and immersed in Kreb's physiological solution and bubbled with oxygen. The perfusion was maintained at 80-120 ml/minute avoiding undue distention of the vessels. Forty-five minutes were allowed for stabilization. The bathing fluid was replaced with 2% lidocaine hydrochloride. Samples (3ml) were withdrawn from the perfusion system at 5 min, 15 min and 60 min. An equal volume of physiological solution was injected into the perfusion system each time a sample was collected. Lidocaine levels of the samples were determined by commercial laboratories.

Table I

Mean ± SEM of Lidocaine levels (μgm/ml) absorbed via isolated canine vascular wall

Time	Carotid artery	Jugular vein
5 min	0.1 ± 0.02	7.9 ± 0.1
15 min	1.5 ± 0.07	19.2 ± 0.6
60 min	7.2 ± 0.09	79.8 ± 1.03

There was no significant absorption of lidocaine through the arterial segment at 5 minutes (0.1 microgm/ml) as compared to the venous segment (7.9 micrograms/ml). Subsequent sampling shows increased arterial absorption but still much less than the venous absorption (approximately 1/10).

The very poor absorption of lidocaine through the arterial wall during the initial period may be due to arterial constriction[5]. Subsequently arterial absorption increased but never approached the venous levels. This is probably related to thick layers of the arterial wall. The role of hydrostatic pressure in the vessel remains to be studied. Our results indicate that arterial absorption is very unlikely to be a significant factor in the immediate toxic manifestations of lidocaine following large volume injections in the head and neck area.

References

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