Comparing Sodium Bicarbonate with Normal Saline for Reversing of Epidural Anesthesia with Plain 2% Lidocaine

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In this study we investigated whether washout of local anesthetic with sodium bicarbonate compared with 0.9% NaCl through an epidural catheter could provide a faster recovery of motor and sensory block in patients undergoing 2% plain lidocaine epidural anesthesia. In a randomized and double-blinded clinical trial, 60 ASA classes I and II male patients scheduled for elective knee or ankle orthopedic procedures underwent epidural anesthesia with 2% plain lidocaine were enrolled. A T4 dermatome level of analgesia was maintained intraoperatively. Following surgery, patients were randomly allocated to receive no epidural bolus (control), 1.5 mL Normal Saline (NS) or 0.4 mL sodium bicarbonate plus 1.46 mL normal saline (BC) postoperatively through epidural catheter. Assessment of motor and sensory block was performed at 5 min intervals until complete motor and sensory block recovery. Times to complete sensory and motor block recovery were significantly less in NS and BC groups compared with control group (p = 0.012) but there were no significant differences between NS and BC group (p = 0.08). The results suggest that recovery of sensory and motor function of the lower extremities in patients undergoing epidural anesthesia with plain 2% lidocaine can be accelerated with epidural administration of 1.5 mL NS or 0.4 mL Sodium bicarbonate plus 14.6 mL NS.

**Key words:** Epidural anesthesia, lidocaine, recovery, sensory block, motor block
INTRODUCTION

Epidural anesthesia has many advantages over general anesthesia for numerous surgical and obstetrical procedures (Sitzman et al., 2001). Prolonged postoperative sensory and motor block following neuraxial blocks (spinal or epidural block) after short surgical procedures is a major disadvantage of this technique (Rodriguez et al., 2001). Delay in motor function, which is necessary for postoperative revival increase Post Anesthesia Care Unit (PACU) time and expense (Gissen et al., 1980) and patient dissatisfaction (Sitzman et al., 2001).

Increasing the speed of recovery from epidural anesthesia, will improve patient acceptance of epidural anesthesia, as well as potential cost savings. Gissen et al. (1980) revealed that neural block can be readily reversed by washout of local anesthetic from isolated nerve preparations using crystalloids in vitro.

For the first time Johnson et al. (1990) reported that epidural motor block could be reversed by epidural injections of crystalloid solutions (Ringer Lactate and Normal Saline). Their study included 26 obstetrical patients undergoing elective cesarean section delivery with epidural anesthesia by 0.75% bupivacaine. Three 15 mL crystalloid solution boluses with 15 min intervals were administered postoperatively. There was no significant difference in dermatomal sensory regression time between the groups. However, full motor function recovery of the lower extremities took more than twice as long in the control group than in the crystalloid groups (Johnson et al., 1990).

Several studies confirmed that using numerous epidural washout solutions accelerated motor recovery and reduced PACU stay (Chan et al., 1999; Brock-Utne, 1998; Caponga, 1995).

All these previous studies used normal saline or Ringer solutions for washout of local anesthetic.

The efficacy of alkalization of local anesthetic solutions and different regional blocks in prolongation of sensory block were determined that alkalization produced the best results with lidocaine and bupivacaine for epidural block (Brown et al., 2005). Some investigations have shown that carbonation of the local anesthetic may help a more rapid peak blood level of the drug as a consequence of higher blood entry to be achieved (Covino and Wildsmith, 1998). We hypothesized that alkalization of washout solution may enhances lidocaine washout and faster recovery period. The objective of this study was to compare the effect of epidural normal saline (0.9% NaCl) with sodium bicarbonate for reversing sensory and motor block of epidural anesthesia using 2% plain lidocaine.

MATERIALS AND METHODS

This clinical trial was performed in Dr. Shariati Hospital of Tehran University of Medical Sciences in 2005. After the institutional review board approval and informed consent were given, 60 ASA physical statuses I and II male patients aged 25-65 years scheduled for elective knee or ankle orthopedic procedures under epidural anesthesia were enrolled in the study.

All patients were premedicated with meperidine 1 mg kg⁻¹ and midazolam 1 mg intravenously. Under standard monitoring, lumbar epidural anesthesia was performed with the patient in the lateral position, using a 3.5 inch, 17-gauge Tuohy needle inserted midline at the L₃-L₄ intervertebral space. The epidural space was identified using the loss of resistance technique. Epidural catheters were inserted 3 to 4 cm into the epidural space. Following a negative aspiration for blood or Cerebrospinal Fluid (CSF), a 3 mL test dose containing 2% lidocaine with 5 µg epinephrine was administered via the catheter to rule out intravascular or subarachnoid placement.

Epidural anesthesia was induced by administering 2% lidocaine in sufficient volume to obtain an analgesic level up to T4 dermatome (nipple) in all patients. This sensory level was determined by a decreased perception of toothpick prick at the T4 dermatome level bilaterally when compared with an unblocked dermatome, such as the shoulder.

The sensory level was evaluated every 10 min during the surgical procedure and maintained at the T4 dermatome level using intermittent 5 mL boluses of local anesthetic.

At the end of surgery, each patient was assigned randomly using a table of random numbers to one of three groups (n = 20 in each group).

Group I (NS) received no epidural solution. The epidural catheter was removed 15 min following completion of surgery.

Group II (NS) received an epidural bolus of 15 mL NS at the end of the surgical procedure. Group III (BC) received an epidural bolus of 15 mL Sodium bicarbonate solution (0.4 mL Sodium bicarbonate plus 14.6 mL NS with pH of 8.4). In NS and BC groups the catheter was removed 15 min after the epidural bolus injection.

All epidural boluses were prepared by a negative aspiration for blood and CSF and were infused over a 1 min period. All the boluses solutions were preservative free. Patients were blinded to their group assignment as were the investigators assessing postoperative motor and sensory recovery data. Assessment of motor and sensory block was performed in the PACU at 5 min intervals.
following completion of surgery by an independent investigator blinded to group assignment. The level of motor block of the lower extremities was assessed at 5 min intervals until complete motor (intact hip, knee and ankle flexion) and sensory (sacral dermatome sensation) block were recovered (Sitzman et al., 2001).

**Statistical analysis:** Sample size was calculated to detect 12 min difference with standard deviation of 13 min in the time to full motor recovery with $\alpha = 0.05$ and statistical power of 80%. Normality of distribution was tested by Kolmogorov-Smirnov test. Parametric data were analyzed using one-way analysis of variance (ANOVA). When overall between-group significance was present, Post Hoc Tukey test was performed. Statistical analysis was performed with SPSS ver. 12. Differences were considered significant at $p<0.05$.

**RESULTS AND DISCUSSION**

There were no significant differences in patients demographic and clinical data between the study groups (one way ANOVA, $p>0.05$) (Table 1).

Times to complete sensory and motor block recovery were significantly less in the patients receiving epidural NS or BC boluses compared to the control group (ANOVA, $p = 0.012$) but there were no significant differences between NS and BC groups (Post Hoc Tukey test, $p = 0.08$) (Fig. 1).

No patients in any group experienced back discomfort or other complications during or after epidural NS or BC boluses.

**Discussion:** This study demonstrates that there is no significant difference for using epidural 0.9% NaCl or Sodium Bicarbonate to accelerate the recovery of motor and sensory block following epidural anesthesia by 2% plain lidocaine.

Previous studies have utilized epidural NS or crystalloids rather than sodium bicarbonate for washout in patients undergoing epidural anesthesia with local anesthetics (Sitzman et al., 2001; Johnson et al., 1990; Mather et al., 1976; Eisenach et al., 1987; Capogna et al., 1995).

Sodium bicarbonate is commonly added as an adjunct to epidural local anesthetic solutions to alkalize the local anesthetics for increasing onset speed, more complete block and higher block quality (Arakawa et al., 2003a, b; Strichartz et al., 2005; Bromage et al., 1963).

It has been shown that addition of bicarbonate or carbon dioxide to local anesthetic solution to block an isolated nerve, accelerates the onset time and decreases the minimum Concentration (cm) required for conduction blockade (Covino et al., 1998). Also, it has been suggested that the addition of bicarbonate for increasing the pH of local anesthetic solution, increases the concentration of non ionized free base, which theoretically increases the rate of intraneural diffusion of the drug and more rapid penetration of connective tissue surrounding the nerve trunk and speed of the onset of block.

The exact mechanism of redistribution of drugs from the epidural space to their site of action (e.g., intradural spinal nerve root, dorsal horn of the spinal cord) is not well understood.

It was thought that the mechanism of action of epidural anesthesia was block of intradural spinal nerve roots by local anesthetic diffusion through the spinal nerve root dural cuff. However, experimental studies have shown that diffusion through the spinal meninges is the primary mechanism (Bromage et al., 1963, 1975; Cousins et al., 1998; Bernards et al., 1999).
Likewise, how an epidural bolus (i.e., washout) alters the pharmacokinetics of epidural administered local anesthetics, to accelerate motor and sensory block recovery is not entirely clear. There are several possible anatomic and physiochemical explanations. The epidural space is filled with loose adipose tissue, lymphatics and venous plexuses. If the epidural space serves as a reservoir of local anesthetic agent, which subsequently diffuses across the meninges to reach its ultimate site of action, then an epidural administered bolus would result in a dilution of remaining local anesthetic reservoir. Additionally, rostral and caudal spread of the dilute local anesthetic solution within the epidural space could result in the exposure of local anesthetic to a larger venous and lymphatic surface area and hence, greater vascular uptake.

Theoretically increasing the pH of washout solution might significantly lessen the sensory and motor block duration by increasing the concentration of non ionized free base and more rapid diffusion rate of the drug to adjacent tissues, including blood and results in higher peak levels (Sitzman et al., 2001).

This study failed to demonstrate such hastening. Our results show motor and sensory function recovery trends similar to those of previous epidural crystalloid washout studies using plain local anesthetic solutions. It may be caused by separate phenomena. Firstly, alkalization of washout solution may enhance the block by improving the diffusion of ionized lidocaine to non-ionized form and consequently aggregate the block rather than improving its dilution.

Secondly, it is possible that the epidural administration of 0.9% NaCl, which is slightly acidic at a pH of 5.0, could lower the pH of the epidural lidocaine solution and shift its equilibrium toward the charged cationic form of lidocaine, (Steven et al., 1989) which would result in less redistribution from the epidural space to the spinal site of action.

Clinical studies have demonstrated a more prolonged onset of epidural analgesia and anesthesia when the pH of local anesthetics has been adjusted away from the physiologic range (Difazio et al., 1989; Chan et al., 1999). Chan et al. (1999) found similar venous plasma lidocaine concentrations following 1, 20 and 40 mL of NS epidural flush, suggesting that the volume of epidural NS flush cannot influence vascular absorption of epidural lidocaine. Also, it may be suggested that vascular absorption of local anesthetic has an uncertain role (Brock-Utne et al., 1998).

Previous studies involving epidural crystalloid administration following epidural anesthesia have shown the safety of a 15 mL NS volume over a 1 min period which was chosen for this study.

A limitation of this study is that the venous plasma lidocaine concentrations were not measured in patients. No patient in our study complained of back pain during or after the epidural NS boluses. There was no evidence of sensory level progression above the T4 dermatome in any patient. The lack of adverse effects of epidural NS or BC flushes in our and previous studies, support that epidural sodium bicarbonate or 0.9% NaCl washouts can be safely used in clinical practice to accelerate the recovery of sensory and motor block following epidural anesthesia.

In summary, this study demonstrates that the recovery of sensory and motor function of the lower extremities in patients undergoing 2% lidocaine epidural anesthesia can be accelerated by epidural administration 15 mL of NS or 0.4 mL of Sodium bicarbonate plus 14.6 mL of NS at the end of surgery.

REFERENCES


