

**TITLE:** PATIENT-CONTROLLED EPIDURAL ANALGESIA: EFFICIENT AND EFFECTIVE POSTOPERATIVE ANALGESIA!

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To combine the efficacy of epidural opiate administration and the short response time of patient-controlled analgesia, we developed a method of administering patient-controlled epidural sufentanil (PCES). We sought to determine if PCES would provide relief of postoperative pain comparable to intermittent epidural administration of morphine.

After Institutional Review Board approval, consenting patients scheduled for abdominal or lower extremity operations were randomly assigned to receive PCES (n=20) or epidural morphine (n=20). The visual analog scale (VAS, 0-100 mm) was used to quantitate postoperative pain. The VAS and use of the PCA device were explained to the patient, preoperatively. After the operation was completed, while the patient was still under general anesthesia, a lumbar epidural catheter was inserted. In patients who received PCES, sufentanil (20 µg in 5 ml normal saline) was injected into the epidural catheter, followed by an infusion of 5 µg/h.

Additional 5 µg doses were available on demand, with a 20 min lockout. If the requests for medication exceeded 200% of the allotted dose during any two-hour period, 20 µg of sufentanil was given, and the basal rate was increased by 5 µg. Patients who were randomly assigned to receive epidural morphine were given 5 mg of preservative free morphine sulfate every 12 h. To achieve quick relief of breakthrough pain, 20 µg sufentanil was given epidurally. Visual analog scale scores were recorded daily. Three scores were requested of the patient: level of pain at the time of assessment, the least pain experienced, and the worst pain felt during the previous 24 hours. Data were statistically evaluated using the Mann-Whitney rank-sum test.

Pain was well controlled in both groups. No significant differences were found between the groups in the intensity of pain. No instances of respiratory depression were noted in either group. No significant differences were found in the incidence of pruritus, nausea, vomiting, and urinary retention. However, a 4.5 fold difference was noted between the two groups in the number of interventions required to maintain therapy. Eleven patients who received intermittent epidural morphine required 36 separate interventions for the treatment of breakthrough pain. In contrast, only eight interventions were required for the treatment of breakthrough pain in eight patients who received PCES.

Our results demonstrate that PCES provides analgesia comparable to epidural morphine, but requires far fewer interventions by physician and nursing staff.

**TITLE:** Potentiation of Local Anesthetic Action of Lidocaine Following Bicarbonate Buffering.

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It has been common practice to add bicarbonate to local anesthetics in an attempt to increase the rate of onset and potency of their action, even though efficacy has been unproven in clinical or laboratory controlled trials. This study was designed to evaluate the effect of raising the pH of Lidocaine on the rate of onset and potency of the anesthetic action in anesthetized, normocapnic and normoxic rats.

Anesthesia was induced and maintained with an intraperitoneal injection of 50 mg/kg sodium pentobarbital. Animals were intubated and ventilated so as to maintain normal PaO<sub>2</sub> and PaCO<sub>2</sub> levels. The common carotid and external jugular were cannulated for continuous arterial and venous pressure measurement, discontinuous arterial blood gases, and intravenous infusion of balance electrolyte. EKG and temperature were also monitored continuously. Femoral skin incisions were made exposing both femoral nerves. Bipolar recording and stimulating electrodes were positioned on both nerves for monitoring of action potentials. A twenty six gauge needle tipped catheter was inserted medial to each femoral nerve sheath.

Fifty microliters of unbuffered 1.7% Lidocaine pH 6.22 was injected on one side simultaneously with the injection of 50µl of buffered 1.7% Lidocaine pH 7.22 to the opposite side. Action potentials were measured prior to Lidocaine injection and at 1min intervals for 15min followed by 5min intervals until recovery. Mean action potential

amplitudes were compared using paired t tests with p<0.05 as significant.

Both rate of onset and depth of the Lidocaine nerve block were potentiated by the addition of bicarbonate. Maximal action potential amplitude reduction to 55% of control occurred by 8min in the unbuffered group (n=10). The action potential amplitude was reduced to below 30% by 8min (n=10) in the buffered group and continued to a maximal depression below 25% by 14min. Duration of block did not appear to be significantly affected.

The addition of bicarbonate to the Lidocaine injectate alkalizes the solution liberating CO<sub>2</sub>. Increasing the pH increases the concentration of the neutral form of the anesthetic which is available for crossing the lipid membranes into the nerve. The effect of increased CO<sub>2</sub> levels was not assessed in this study, however, the addition of high levels of CO<sub>2</sub> into the tissue upsets the CO<sub>2</sub>, H<sub>2</sub>CO<sub>3</sub>, HCO<sub>3</sub> equilibrium driving it to the right in favor of the formation of additional bicarbonate further buffering the system.

