TITLE: EPIDURAL MORPHINE DECREASES PAIN FOLLOWING TRANS-STERNAL THYMECTOMY IN PATIENTS WITH MYASTHENIA GRAVIS

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INTRODUCTION: Following trans-sternal thymectomy, patients with myasthenia gravis may have respiratory insufficiency because of their underlying disease and because of inadequately treated post-operative pain. Epidural narcotics have been shown to provide superior analgesia to intravenous narcotics following conventional thoracotomy and improve respiratory mechanics.[1] We tested the hypothesis that pre-operative administration of morphine into the lumbar epidural space provides superior post-operative analgesia and post-operative pulmonary mechanics compared to intravenous narcotics.
METHODS: This prospective, double-blinded, randomized study was approved by the hospital committee on clinical investigation. All patients gave informed consent upon entering the study. Prior to induction of anesthesia patients received 14 ml. of either morphine (0.5 mg/ml) or sterile saline into the lumbar epidural space. Anesthesia was induced with thiamylal and maintained with an inhalational agent. Patients were given intra-operative intravenous narcotics at the discretion of the anesthesiologist. No patient received intravenous muscle relaxants. Post-operative heart rate, blood pressure, respiratory rate, negative inspiratory force, forced vital capacity, duration of post-operative intubation and ventilation, analgesic requirements, and visual analogue pain scale (PS) were monitored.
RESULTS: Nine patients received epidural morphine and 11 epidural saline. Demographic variables were similar in both groups. No complications resulted from epidural administration of drug or placebo. There was no difference in intra-operative dose of intravenous narcotics. Immediately following surgery, patients receiving epidural morphine had significantly lower pain scores (PS: 3.5±1.2 vs 7.3±1.2), greater forced vital capacity (FVC:43±5 vs 30±6% of control), and less tachypnea (RR 14±2 vs 22±2 breaths/min) as compared to patients receiving saline. Patients receiving morphine required much less supplemental narcotic post-operatively than patients receiving placebo (0.12±0.04 vs 0.22±0.03 mg/kg in the first 8 post-operative hours). There were no differences between groups for duration of post-operative intubation or ventilation, heart rate, blood pressure, or negative inspiratory force.
DISCUSSION: Epidural morphine provides post-operative analgesia which is superior to that of intravenous narcotics with improved respiratory mechanics. We feel that aggressive pain management can be accomplished in myasthenics with epidural morphine without significant morbidity.
REFERENCE: 1. Anesthesiology 61:569-575, 1984.

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TITLE: PENTOXIFYLLINE INHIBITS HYPOXIA-INDUCED PULMONARY HYPERTENSION IN THE PIG.

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Pentoxifylline (PTX) has been shown to inhibit endotoxin-induced pulmonary hypertension (PH) and pulmonary edema in the pig, and appears to be a potent pulmonary vasodilator. The effect of PTX on hypoxia-induced PH in the pig is studied.

Twenty-seven in-situ, isolated blood perfused porcine lung preparations were studied: Group 1, Time control (n=6), lungs were ventilated with 5% CO_2 in compressed air; Group 2, Hypoxia control, (n=7) lungs were ventilated with an hypoxic gas mixture (5% CO_2 + 5% O_2 + 90% Nitrogen); Group 3, (n=7), PTX-pretreatment + hypoxia, lungs were pretreated with PTX (15 mg/kg bolus + 0.1 mg/kg/min infusion) prior to hypoxia challenge; Group 4, (n=7). Hypoxia + PTX post treatment, lungs were given PTX at the peak of PH. Pulmonary arterial pressure (Ppa) was followed for 4 hours after hypoxia challenge. Lung wet/dry (W/D) weight ratio was used as an index for lung edema formation.

Hypoxic ventilation caused sustained PH (Fig. 1) and moderate edema formation (Fig. 2). Pretreatment with PTX prevented hypoxia-induced PH and edema. With PTX posttreatment Ppa decreased precipitously and remained below the baseline value. PTX posttreatment also prevented edema formation.

The present study showed that PTX is a potent inhibitor of hypoxic pulmonary vasoconstriction. Since this effect is similar to the effect on endotoxin induced PH, the vasodilatory action of PTX may mediate through a common mechanism. The mechanism could be related to an increase in cAMP and/or prostaglandin synthesis.

Fig. 1.

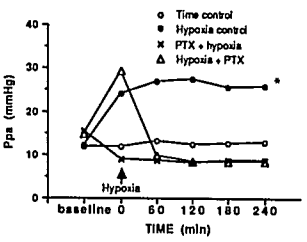

Fig. 2.