TITLE: EPINEPHRINE IMPROVES THE QUALITY OF BUPIVACAINE SPINAL ANESTHESIA

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Previous studies suggest that epinephrine may not prolong the duration of bupivacaine spinal anesthesia (1). This study is designed to evaluate epinephrine's effect on anesthetic quality.

After approval by our institutional review board, 61 patients undergoing TURP were prospectively randomized into two groups: Group I received 6 mg of bupivacaine, 66 mg dextrose, and 0.2 ml of sterile water (total vol 1.0 ml). In Group II, 0.2 mg of epinephrine (in 0.2 ml) was substituted for the water. Observers were blinded. Spinal technique was standardized. Sensory (pin prick) and motor blockade (sum of hips, knees, & ankles with complete blockade) were assessed at standard intervals. The incidence of pain at the operative site was noted. Differences were evaluated by Fishers exact test, Chi-Square, and unpaired t-test.

Addition of epinephrine resulted in a significant decrease in the incidence of pain at the operative site (Table 1). All episodes of pain were mild in nature and resolved with intravenous fentanyl. There were no differences between groups in age, height, weight, ASA status, dose of premedication, or surgery time. Time to 2-segment regression was not different (I = 77 ± 25, II = 81 ± 29 min, mean ± SD).

Although epinephrine did not prolong duration, it improved the quality of spinal anesthesia. Addition of 0.2 mg of epinephrine reduced the incidence of painful sensations from 16% to 0%. Increasing the dose of bupivacaine would probably produce similar results. However, similar studies with tetracaine suggest that increasing the dose of local anesthetic also increases the incidence of hemodynamic perturbations (2).

Table 1. Comparison of Epinephrine and H20 Groups

Group	Highest Sensory Level	Maximum Motor Blockade	Onset to T10 (min)	Painful Sensations (yes/no)
I (H2O)	T6 ± 2	4 ± 2	2.7 ± 1.5	5/26*
II (Epi)	T5 ± 2	5 ± 2	3.3 ± 4.0	0/30*

*P=.03

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TITLE: INTRATHECAL CLONIDINE AND THE RESPONSE TO HEMORRHAGE

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INTRODUCTION: Intraspinally administered clonidine represents a new approach to pain therapy in obstetric patients. Clonidine may decrease blood pressure by diminishing sympathetic nervous system activity, and this effect could interfere with maintenance of blood pressure during hemorrhage.

METHODS: At least 5 days following surgical insertion of arterial and intrathecal catheters, 5 sheep received lumbar intrathecal injections of saline or clonidine, 300 μg. Fifteen min later, 20% of estimated blood volume was removed over 10 min. Arterial blood was sampled prior to injection, immediately prior to and at the end of hemorrhage, and at 10 and 60 min following the end of hemorrhage and analyzed for blood gas tensions, atrial natriuretic peptide, catecholamines, plasma renin activity, and vasopressin. Blood pressure and heart rate were continuously monitored throughout. Blood was reinfused 1 hr following hemorrhage and the companion experiment (saline or clonidine) performed at least 48 hrs later. Groups were compared by one-way ANOVA, with $P < 0.05$ significant.

RESULTS: Compared to saline, clonidine decreased blood pressure at rest (by 10±2%) but increased blood pressure following hemorrhage (Fig. 1). Plasma norepinephrine increased following hemorrhage in both groups, but this increase was less in the clonidine-treated ewes (plasma norepinephrine following hemorrhage 0.45±0.13 in clonidine group vs. 1.10±0.57 ng/ml in saline group; $P < 0.05$). Groups did not differ in other parameters.

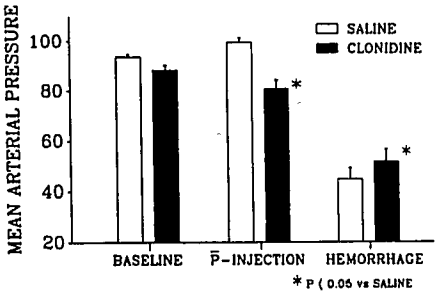

FIG. 1: Blood Pressure Response to Hemorrhage

DISCUSSION: Clonidine exerts opposing actions on blood pressure, decreasing it by inhibiting sympathetic nervous system activity, and increasing blood pressure by peripheral vasoconstriction. In the resting state, clonidine-induced sympathetic inhibition predominates and blood pressure decreases. In contrast, during periods of near maximal hemodynamic stress, such as occur following acute, major hemorrhage, clonidine minimally alters hormonal responses, but supports blood pressure by peripheral vasoconstriction.[1]

REFERENCES:

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Supported in part by GM35523.