LOCAL ANESTHESIA AND PAIN I

TITLE: COMPARISON OF PLASMA BETA-ENDORPHIN LEVELS DURING SPINAL VERSUS GENERAL ANESTHESIA

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Introduction. Surgical and anesthetic stresses of regional versus general anesthesia have long been debated. Pflug and Halter showed that plasma levels of epinephrine, norepinephrine, growth hormone, and cortisol are elevated in patients undergoing general but not spinal anesthesia. They attributed this to spinal anesthetic suppression of neural afferent from sites of tissue injury. Roizen et al. found that only at high anesthetic concentrations (well above MAC) were adrenergic responses to general anesthesia obviated. Recently, circulating beta-endorphin has been suggested as another marker of both physical and psychological stress. The present study compared operating room (OR) and recovery room (RR) levels of plasma beta-endorphin during and following spinal and general anesthesia.

Methods. With informed consent and Human Subjects Committee approval, eleven patients undergoing cystoscopy and transurethral resection of prostate or bladder were studied. Diazepam 10 mg orally was given preoperatively. The general anesthesia group received thiopental, succinylcholine, and enflurane, and their tracheas were intubated. Tetracaine (12-14 mg) was used for all spinal anesthetics, and patients received no inhalation or intravenous supplementation. Plasma beta-endorphin samples were drawn through an indwelling venous catheter immediately prior to entry to the OR, 5 minutes after anesthetic induction, one hour into the surgical procedure, on entering the RR, and finally on discharge from the RR. Plasma beta-endorphin levels were determined by radioimmunoassay using New England Nuclear Beta-endorphin RIA kits. Data was analyzed using Student's t-test for grouped data with significance defined at p < 0.05.

Results. There was no significant difference between groups with regard to age, height, weight, ASA classification, or pre-anesthetic plasma beta-endorphin level. The percent rise in plasma beta-endorphin level with general anesthesia was significantly higher through the surgical procedure and into the RR period (Figure).

Discussion. Beta-endorphin is a naturally occurring peptide released into the circulation by the posterior pituitary, but may also be secreted by other tissues. The present study utilized circulating plasma beta-endorphin as a marker of stress. The rise seen in plasma beta-endorphin was significantly higher at all time points during general anesthesia as compared to spinal. Plasma beta-endorphin increased immediately after induction and remained elevated one hour into the surgical procedure. This differs significantly from a recent report that showed no elevation in beta-endorphin with induction of general anesthesia, but high levels after surgical stimulus. The absence of a continued rise in plasma beta-endorphin levels with surgical stimulus in the present study may be due to a greater anesthetic depth at this time point (1 hour) in the general group. The largest rise in beta-endorphin occurred during awakening from general anesthesia and had not returned to normal by the time of RR discharge. Patients receiving spinal anesthesia showed no elevation of plasma beta-endorphin even after the anesthetic effects had dissipated.

Association of plasma beta-endorphin with stressful situations has been documented. Our results implicate general anesthesia with enflurane as being more stressful than spinal anesthesia, as measured by plasma beta-endorphin.

![Graph showing change in plasma beta-endorphin](image)

Fig. The percent change in plasma beta-endorphin with respect to immediate pre-anesthetic levels. *p < 0.05