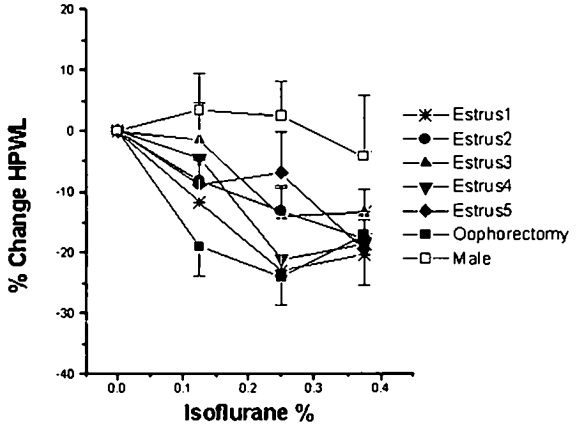
GERTIE MARX

GM-1

THE EFFECT OF OVARIAN HORMONES ON ISOFLURANE HYPERALGESIA *Flood, P. Daniels, D. Anesthesiology, Columbia University, New York, NY* Volatile anesthetics increase pain sensitivity at the low concentrations that are present on emergence in humans and animals [1-3]. Gender differences in pain sensitivity and in pharmacodynamic responses to drugs used to treat pain have been widely reported (reviewed by Berkley, 1997)[4]. To determine whether there was a gender difference in the hyperalgesic response to isoflurane, we measured hind paw withdrawal latency in male and female mice with and without isoflurane. Females had a more prominant hyperalgesic response to isoflurane than males (Figure 1). We tested female mice for HPWL in each stage of their estrus cycle, after oophorectomy and with exogenous estrogen replacement. Hyperalgesia was more prominant in stages 1 and 4 of the cycle (low estrogen and progesterone)and after oophorectomy. Estrogen replacement was not protective against isoflurane hyperalgesia. A combination of estrogen and progesterone may be required to mimic the isoflurane response found in estrus stages 2,3 and 5. Hyperalgesia from low isoflurane concentrations may be particularly problematic in females, but may not be a problem in pregnancy. *1. Zhang, Y, et al.; Inhaled anesthetics have hyperalgesic effects at 0.1 minimum alveolar concentration. Anesth Analg, 2000. 91462-6. 2. Dundee J and Moore J; Alterations in response to somatic pain associated with anesthesia IV. The effect of sub-anesthetic concentrations of inhalational agents. British Journal of Anaesthesia, 1960. 32:453-9. 3. Flood P et al.; Isoflurane hyperalgesia is modulated by nicotinic inhibition. Anesthesiology (in press). 4. Berkley KJ; On the dorsal columns: translating basic research hypotheses to the clinic. Pain, 1997.70:103-7.*



Females have more hyperalgesia from isoflurane than males. Hyperalgesia is pronounced at low estrogen, low progesterone stages of the estrus cycle and after oophorectomy.

GM-2

PEAK POINT CORRELATION DIMENSION: A NOVEL PREDICTOR OF ADVERSE HEMODYNAMIC RESPONSE TO SPINAL ANESTHESIA. *Chamchad, D. Arkoosh, V.; Buxbaum, J.; Horrow, J.; Nakhamchik, L.; Kresh, J. MCP Hahnemann University, Philadelphia, PA*
Introduction: Cardiovascular instability often complicates spinal anesthesia (SPA) for Cesarean Section (C/S). Early detection of cardiovascular imbalance would enable prompt response or preemptive treatment. This study observed changes in point correlation dimension (PD2), an index of Heart Rate Variability (HRV), before and after SPA. **Methods:**With IRB approval and consent 22 non-laboring parturients with a single fetus, scheduled for C/S under SPA, received spinal bupivacaine-morphine following intravenous prehydration. An adverse response was defined as hypotension (SBP≤75% baseline), nausea or vomiting. The ECG, obtained in LUD position before and 10 min after SPA, was stored in analog form. This was converted off-line (DataQ,Akron,OH) to digital RR intervals. Peak PD2(pPD2) analysis extracted the nonlinear parameters embedded in HRV.[1] Hemodynamic and pPD2 data were compared before and after SPA (paired Student's t-test). The median pPD2 value before SPA separated the 22 patients into two groups of 11 (LO,HI). **Results:** pPD2 decreased after SPA [3.22(0.89)(mean(SD)) vs. before SPA 4.05(0.64) p=0.0003]. Median pPD2 measured 3.9 before SPA. Baseline heart rate was higher in the LO group [95/min (10.2)] vs. the HI group [81.4(9.6)]. No HI group patient pPD23.9 before SPA experienced an adverse response, whereas adverse responses occurred in all 11 LO group patients pre-SPA pPD2<3.9 p=0.0000028, fig1. Groups LO and HI did not differ in age, height, weight, bupivacaine-morphine dose, initial BP or volume of IV prehydration. **Discussion:**Because adverse responses to SPA are frequent (50% in our series), prior risk identification should advance patient safety and comfort. pPD2 successfully stratified patients undergoing C/S into risk cohorts. Additional studies are required to validate pPD2=3.9 as a risk discriminant and to investigate the use of this measurement to stratify patients for prophylaxis. *1. Integ Physi & Beh Sc 1994; 29(3):217-35*

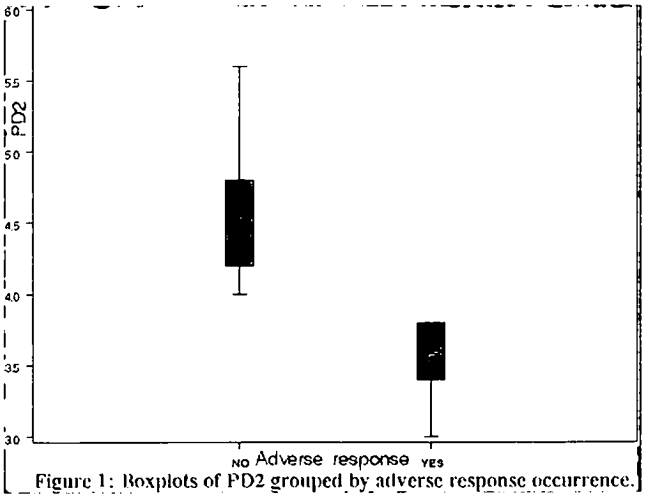


Figure 1: Boxplots of PD2 grouped by adverse response occurrence.