POSTER REVIEW #2

A50 (Poster 40)

PHOSPHODIESTERASE (PDE5) RECEPTOR INHIBITION MODERATES THE VASOPRESSOR EFFECT OF 5-HYDROXYTRPTAMINE (5-HT) ON THE FETOPLACENTAL VASCULATURE. *Downing, J.W.; Minzter, B.H.; Ramasubramanian, R.; Paschall, R.L.; York, J.J.; Johnson, R.F. Anesthesiology, Vanderbilt University, Nashville, TN* The intracellular neucleotides (ICN) second messenger signaling pathways regulate numerous important physiological processes. Cyclic ICN receptors have been identified in human placental tissue[1]. Cyclic guanine monophosphate (cGMP), a potent physiological vasodilator, is an ICN normally expressed through the release of nitric oxide. But inhibition of its enzymatic breakdown by PDE5 can also increase the local tissue content of cGMP. In this study, the fetoplacental vascular response to PDE5 inhibition after exposure to 5-HT, a potent vasoconstrictor, was investigated. Six placentae were collected from healthy women with written informed consent and IRB approval. They were perfused using established protocol[2]. 5-HT at a concentration of 3 μM was added to the fetal perfusate. Increasing concentrations (0.015, 0.15, 1.65, 4.5, and 10.5 μM) of a PDE5 inhibitor were then presented to the fetal circuit at 20 minute intervals. 5-HT alone significantly increased mean fetal umbilical artery pressure (FAP) from 66.2$\pm$2.5 mmHg to 96.6$\pm$5.6 mmHg ($p = 0.009$). No changes were observed with the two lower concentrations of PDE 5 inhibitor. Significant ($p = 0.005$) reductions in mean FAP were observed with PDE5 inhibitor concentrations of 1.65 μM (84.4$\pm$2.9, $p=0.0129$), 4.5 μM (79.0$\pm$2.2, $p=0.009$), and 10.5 μM (71.3$\pm$2.5). This study demonstrates that the vasoconstrictor effect of 5-HT is significantly obtunded by placental PDE5 inhibition. **Reference:** 1 Anesthesiology 2000;92S:A11 2 Anesthesiology 1995;82:459-468.

A51 (Poster 41)

PHOSPHODIESTERASE (PDE5) PLACENTAL RECEPTOR INHIBITION OBTUNDS HYPOXIC FETOPLACENTAL VASOCONSTRICTION (HFPV) IN THE HUMAN PLACENTA. *Downing, J.W.; Minzter, B.H.; Ramasubramanian, R.; Paschall, R.L.; York, J.J.; Johnson, R.F. Anesthesiology, Vanderbilt University, Nashville, TN* PHV (pulmonary hypoxic vasoconstricion) and HFPV[1] are physyiological responses to hypoxemia. Cyclic guanine monophosphate (cGMP) expression by nitric oxide (NO) produces pulmonary arterial vasodilatation under hypoxemic conditions. We hypothesize that cGMP modulates HFPV in a similar manner and that inhibition of accessible placental PDE5 receptors responsible for the enzymatic breakdown of cGMP will obtund HFPV. In this study, the effect of PDE5 inhibition on HFPV was investigated using the dual perfused, isolated human placental cotyledon[2]. Five placentae were collected from healthy women with written informed consent and IRB approval. Perfusion pressures for both circuits were measured and recorded every minute using in-line pressure transducers. The fetal and maternal circuits were exposed in sequence for 30 minutes to 21% 0_2 (normoxia), 0% 0_2, (hypoxia) and hypoxia in the presence of a PDE5 inhibitor (10.5 μM). Hypoxia significantly ($p = 0.007$) increased the fetal umbilical arterial perfusion pressure (FAP) from 69.8$\pm$1.6 mmHg to 90.0$\pm$3.9 mmHg. Subsequent PDE5 inhibition was associated with a significant ($p = 0.007$) reduction in FAP to 71.8$\pm$2.0 mmHg. This study demonstrates that the inhibition of accessible placental PDE5 receptors obtunds HFPV. **Reference:** 1 Am J Obstet Gynecol 1987;157:1261-66. 2 Anesthesiology 1995;82:459-68.

A52 (Poster 42)

PRIMARY PULMONARY HYPERTENSION: SUCCESSFUL C/S USING TIVA, NO AND TEE *DeBalli, P.; Habib, A.; Grocott, H.; Olufolabi, A. Anesthesiology, Duke, Durham, NC* **Introduction:** Primary pulmonary hypertension(PPH) in the parturient with a peripartum mortality of 30-50%, presents with significant challenges. **Case report:** A 113kg, 21yr old presented at 26wk with lightheadedness, increasing dyspnea, and left hemiparesis. An EKG revealed rapid AF which was restored to normal sinus rhythm using diltiazem. There was no prior history of sleep apnea, smoking, congenital heart disease, hypercoagulability, thromboembolism, or diet drug use. A prior pregnancy and C/S were unremarkable. A spiral thoracic CT, head scan and lower limb Doppler were all normal. Transthoracic echo revealed normal valvular and LV function but enlargement of the RA and RV with a peak systolic pressure of 58 mmHg. In the absence of other causes, a diagnosis of PPH was made. Diltiazem and heparin therapy was commenced. At 30wk, increasing dyspnea prompted early C/S. **Anesthesia:** Following anti-reflux prophylaxis and arterial line placement, monitors including a Bispectral Index (BIS) were applied. RSI was performed using fentanyl 500 mcg, etomidate 13 mg, and succinylcholine 120 mg. Anesthesia was maintained with propofol (20- 50mcg/kg/min), titrated to a BIS index of 55 to 65. O2 in air with 10 ppm nitric oxide was delivered via IPPV. A central venous cannula was placed. TEE was used to monitor cardiac status intraoperatively. A 6lb 3oz male infant was delivered (Apgar scores 5,7). The patient was transfered to ICU and extubated after 3hr. She was discharged home on the 10th PPD. **Discussion:** This is the first reported successful use of nitric oxide, TEE, TIVA with BIS in a PPH parturient undergoing C/S. GA allowed the intraoperative use of TEE. TEE allowed for the continuous assessment myocardial function and volume status. TIVA and BIS allowed for optimization of anesthesia requirements. The intraoperative management of the PPH parturient requires multidisciplinary approach and selection of appropriate anesthetic monitoring and technique.

A53 (Poster 43)

THE USE OF NATIONAL CQI DATA TO IMPROVE HOSPITAL ANESTHESIA OUTCOMES *Kaul, B.; Vallejo, M.A.; Ramanathan, S.; Mandell, G.L. Department of Anesthesiology, Magee-Womens Hospital, Pittsburgh, PA* The National Perinatal Information Center (NPIC) is a monitor of CQI data in USA. It provides comparative CQI feedback to hospitals. Data submitted by each member institution is combined to create the Perinatal Center Database from which reports are generated. Anesthesia complications include problems arising from general anesthetia, analgesia or other sedation in labor and delivery.Pulmonary complications (aspiration, collapsed lung, respiratory distress/shortness of breath due to anesthetia), cardiac complications (arrest, congestive cardiac failure, hypotension,bradycardia, central nervous system (CNS) complications (cerebral anoxia, meningitis)are reported. The complications are reported to the NPIC by auditors at our hospital. The NPIC data reports showed that the number of anesthetic complications went up in 1998 (Table) to fall outside the upper 95% confidence interval for NPIC data.Investigation of the complications showed a number of these were a result of high spinals in patients who had a spinal block following an inadequate epidural for cesarean section. We have modified our clinical practice by avoiding spinals and redoing the epidural blocks, resulting in a fall in complication rate to an acceptable levels. Conclusion: Continuous quality improvement programs, which include the use of national CQI data, can improve anesthesia outcomes.

Complications	ICD Codes	1996	1997	1998	1999
Pulmonary	668.01,668.02	1	3	3	3
Cardiac	668.11	4	3	5	1
CNS	668.22	1	1	1	0