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IN VITRO INVESTIGATION:DURAL TRAUMA PATTERNS,CSF LEAK AND EPIDURAL NEEDLE PUNCTURE *Angle, P. Kronberg, J.; Thompson, D. Anesthesia, Sunnybrook and Womens College HSC, Toronto, ON, Canada*

The effect of epidural needle design, angle of puncture and bevel orientation on dural trauma patterns and CSF leak was examined. For each study phase human cadaveric dura (L1/2-L4/5) was cut into approximate 2cm square specimens and mounted on a cylindrical model of the dural sac. The model was pressurized to 15cm with artificial CSF (left lateral decubitus pressure) and dura punctured with an epidural needle according to a pre-determined randomization schedule. The pressure was then raised to 25cm (labor/semi-sitting pressure) and leak measured over 15 minute intervals x4. A micromanipulator was used to ensure precise needle angle, bevel orientation and advancement at the time of puncture. Dural trauma patterns were examined using Scanning Electron Microscopy (SEM). Every comparison involved use of dura from the same cadaver. Part 1: addressed the effect of gauge/tip design using 6 epidural needles: 17G Husted; 17G Tuohy; 18G Tuohy; 18G Special Sprotte; 18G Crawford; and 20G Tuohy (10 cadavers). Punctures were made at 90deg to the long axis of the dura with the bevel parallel where applicable. Part 2: The effect of needle angle (30 vs 90 deg) was examined for each of 2 needle types: 18G Tuohy (bevel parallel, 10 cadavers) and the 18G Special Sprotte needle (6 cadavers). Part 3: The effect of bevel parallel vs perpendicular bevel orientation was examined using the 18G Tuohy (10 cadavers). Statistical analysis using RMANOVA was blinded with $p < 0.05$ considered significant. We found a large (3-5 fold) statistically significant reduction in CSF leak/15 minute interval between the 20G Tuohy and each of the other needles examined in Part 1 (reported as mean gm +/- SD per 15 minutes; lgm = 1ml; p values comparison with the 20G Tuohy): 17G Husted (516 +/- 319, $p = 0.002$); 18G Tuohy (420 +/- 191, $p = 0.002$); 17G Tuohy (405 +/- 209, $p = 0.002$); 18G Special Sprotte (359 +/- 208, $p = 0.016$); 18G Crawford (356 +/- 121, $p = 0.0001$); 20G Tuohy (99.5 +/- 112). Part 2: CSF leak for the 18G Tuohy at 30deg was 401 +/- 135 vs 485 +/- 215 at 90deg ($p = 0.31$). Leak for the 18G Special Sprotte at 30deg was 408 +/- 205 vs 401 +/- 208 at 90deg ($p = 0.96$). Leak after puncture with an 18G Tuohy with a perpendicular bevel was 367 +/- 119 vs 485 +/- 216 with the bevel parallel ($p = 0.12$). Characteristic dural trauma patterns were identified on SEM for each needle type, orientation and angle of puncture. This study suggests that a large statistically significant reduction in CSF leak occurs with the 20G Tuohy needle compared with larger epidural needles. Large reductions in leak were found with the Tuohy at a 30deg angle vs 90deg that did not reach statistical significance. Angle of puncture made no difference in leak for the Sprotte epidural needle. Large reductions in leak, not achieving statistical significance were found with a perpendicular Tuohy bevel orientation when compared to a parallel orientation.

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SODIUM NITROPRUSSIDE (SNP) INHIBITS HYPOXIC FETO-PLACENTAL VASOCONSTRICTION (HFPV) IN THE DUAL PERFUSED, SINGLE ISOLATED HUMAN PLACENTAL COTYLEDON *Downing, J.W., Ramasubramanian, R.; Minzter, B.H.; Paschall, R.L.; E, L.; Johnson, B.; Johnson, R. Anesthesiology, Vanderbilt University, Nashville, TN*

Nitric oxide (NO), a potent vasodilator, plays a major role in modulating vascular resistance¹. The dual perfused, single isolated human placental cotyledon is a well-established in-vitro placental model². The model has been used to demonstrate the existence of Hypoxic Feto-Placental Vasoconstriction (HFPV) and its modulation by a variety of vasoactive agents^{3,4}. This study examines the ability of SNP, a NO donor, to counter HFPV in the human fetoplacental circulation. Six placentae were collected from healthy women with their written informed consent and IRB approval. Both the fetal and maternal sides of a single cotyledon were perfused with KRB equilibrated with air (21% oxygen) using the open (non-recirculating) model. Perfusion pressures for both circuits were measured and recorded at one minute intervals. The pH of both circuits was maintained at 7.4 by the addition of CO₂ in the gas mixture (air or nitrogen). Each placenta was allowed a 30 minute normoxic interval to rest and establish its baseline fetal arterial pressure (FAP). Thereafter, the perfusate was rendered hypoxic by substituting nitrogen for air in the gassing media. Hypoxic perfusion was continued for the 30 minutes. FAP increased due to HFPV during this episode of hypoxic equilibration. SNP (50 μM) was then added to the fetal circuit. Thirty minutes later (total duration of hypoxia = 1 hour), the cotyledon was again exposed to the original aerated perfusate for another thirty minutes. In keeping with earlier experiments⁴, removing oxygen from the placental perfusate significantly increased mean (± sem) FAP (66.7 ± 4.2 vs. 78.2 ± 5.3 mmHg, $p = 0.002$). The addition of SNP (50 μM) to the hypoxic preparation rapidly decreased FAP back to control levels (78.2 ± 5.3 mmHg vs. 65.2 ± 4.9 mmHg, $p = 0.0008$). FAP levels remained at baseline during the 30 minute recovery period on air. This study demonstrates that SNP reverses HFPV in the dual perfused, single isolated human placental cotyledon and suggests that NO plays a vasodilatory role in countering HFPV in the human placenta. 1) *Am J Obstet Gynecol* 1991; 164:687-692. 2) *Anesthesiology* 1995; 82:459-468. 3) *Anesthesiology* 2001; V94, No A1, Apr: A51. 4) *Am J Obstet Gynecol* 1987; 157:1261-66.