1348 IN-VIVO CALIBRATION IMPROVES THE RELIABILITY OF TRANSCUTANEOUS (Tc) OXYGEN MONITORING. Richard W. Krouskop, Eresvita E. Cabatu, Bhaktharaj P. Chelliah. (Spon. by Edwin G. Brown). Mount Sinai School of Medicine, Mount Sinai Medical Center, Department of Pediatrics, New York.

We have found a wide variability in correlation between $P_{Tc}O_2$ and PaO_2 under usual clinical conditions; r=.75 for 432 data pairs in 14 sick infants. We felt this poor correlation resulted from patient variation (range of r=.43 → .98 for each infant).

To test this hypothesis we devised and evaluated the usefulness of an in-vivo calibration procedure. Prior to each 4 hour application to the skin, the Tc sensor was calibrated in room-air per manufacturer's instructions. PaO_2 was measured > 20 min. < 1 hr. after application. The resulting error in estimation of PaO_2 by $P_{Tc}O_2$ was calculated [Error = $(PaO_2 - P_{Tc}O_2)/PaO_2$]. If this was >.15, the Tc monitor was calibrated by adjusting its gain to a new value (calibrated $P_{Tc}O_2$) calculated from the present $P_{Tc}O_2$ reading (raw $P_{Tc}O_2$) and the Error [calibrated $P_{Tc}O_2$ = raw $P_{Tc}O_2$/(Error -1)]. Subsequent PaO_2 and $P_{Tc}O_2$ data pairs comprised the study data. Control data for each infant was obtained during alternative time periods by following the same initial procedure, but no in-vivo calibration was performed. In 10 infants, the PaO_2 vs $P_{Tc}O_2$ correlation for study data was 0.93, while only 0.80 for control data. 34.4% of all applications were > our 15% error tolerance. Consequently, in-vivo calibration utilizing one arterial blood gas within each 4-hour application provides a more accurate estimate of PaO_2 from the transcutaneous monitor. However, an increased work load is imposed on nursery staff.

1349 CONGENITAL ABSENCE OF DUCTUS ARTERIOSUS (ADA) Samuel J. Lacina, Otto G. Thilenius, Wade T. Hamilton, Saroja Bharati, Rene A. Arcilla; Pritzker School of Medicine, The University of Chicago Hospitals and Clinics, Department of Pediatrics, Chicago, Illinois

Newborn infants (NB) with cyanotic heart disease and reduced pulmonary flow are often ductus-dependent and improve with prostaglandin (PGE) infusion. However, PGE may fail if the ductus arteriosus is absent. The angiograms of 19 cyanotic NB (mean age 2.7 d) were analyzed. Group A consisted of 5 with ADA, and group B of 14 with patent ductus (PDA). All had pulmonary atresia complex with or without VSD. The diameters of aorta (ascending [AA], descending [DA], at diaphragm level [DDA]), right pulmonary artery (RPA), and left pulmonary artery (LPA) were obtained. Aortic size in A did not differ from those in B at all 3 levels. However, RPA and LPA were smaller in A than in B shown by mean pulmonary/aortic ratios: RPA/AA = 0.21 vs 0.389, RPA/DA = 0.38 vs 0.628, RPA/DDA = 0.41 v 0.64, LPA/AA = 0.20 vs 0.36, LPA/DA = 0.36 vs 0.60, LPA/DDA = 0.39 vs 0.605 (p value in all < 0.005), indicating impaired pulmonary artery growth in utero in pulmonary atresia or severe pulmonary stenosis with ADA. The angiograms in B showed PDA in all, and no increase of bronchial collaterals. In contrast, bronchial flow was abundant in A. Aortic valve stenosis was present in 3/5 in A but in 0/14 in B. ADA may be suspected clinically in cyanotic NB with reduced pulmonary flow if there is no response to PGE infusion and/or if aortic valve stenosis is also present. The diagnosis may be established angiographically based on non-visualization of the ductus, diminutive pulmonary arteries and abundant bronchial flow.

● 1350 EFFECTS OF RATE OF VOLUME EXPANSION ON BRAIN BLOOD FLOW AFTER ASPHYXIA AND HYPOTENSION IN THE PIGLET. A. Laptook, B.S. Stonestreet, W. Oh, Brown Univ. Prog. in Med., Women & Infants Hosp., Dept. of Ped., Providence, RI

The rate at which the systemic arterial blood pressure (SABP) is restored after asphyxial and hypotensive insults may be a factor in the pathogenesis of perinatal central nervous system lesions. To examine this, brain blood flow (BBF) was determined in 16 newborn piglets during asphyxia, superimposed hypotension and subsequent volume expansion (15 ml/kg, plasmanate). Asphyxia (A) was produced by a respiratory dead space and hypotension (H) by phelbotomy. Group 1 received plasmanate in 3 min., Group 2 in 30 min. and Group 3 received none. BBF (microsphere technique) was measured with each insult and at 5 and 30 min. after the start of the volume expansion and removal of the dead space. Results were (M±SEM):

Group	Control (C) BBF (ml/min/gm)	(% Change in BBF from C) A	A+H	5 min.	30 min.
1, n=6	1.11±.1	217±30*	25±21	85±34+	75±18*
2, n=6	0.96±.15	180±30*	8±17	33±14+	99±22*+
3, n=4	1.10±.12	122±26*	-13±11	-22±11	17±23

*p<.02 as compared to control, +p<.05 as compared to Group 3. SABP was reduced (p<.05) in Group 3 at 5 min. after A and H. All groups had significant reductions in hematocrit from control at the completion of the study. At autopsy, intracranial hemorrhage was not observed. The data indicate that 1) the rate of volume expansion did not influence the magnitude of BBF and 2) the higher BBF at the completion of volume expansion probably reflects compensation for anemia to achieve adequate oxygen delivery.

● 1351 CENTRAL NEURAL MATURATION OF THE EXCITATORY RESPONSE TO CAROTID SINUS NERVE STIMULATION. Edward E. Lawson, Albert G. Haddad & Walker A. Long (Spon. by H.S. Harned), Univ. of N. Carolina, Dept. of Pediatrics, Chapel Hill.

The biphasic respiratory response (hyperpnea followed by hypoventilation) of the newborn human has been attributed to central hypoxic depression of the respiratory centers. Recently, we have shown that, in the absence of hypoxia, the excitatory effect of continued carotid sinus nerve (CSN) stimulation fatigues in piglets 4-14 days of age (The Physiologist 23:140, 1980). To study maturation of the excitatory effects of the CSN without confounding blood gas changes, we compared changes in respiratory output (RO=f × neural tidal volume) during electrical CSN stimulation in young and older piglets (7 piglets 4-14 days; 7 piglets 20-34 days). The piglets were anesthetized, vagotomized, paralyzed and ventilated (FIO_2=1.0). End-tidal CO_2 was kept constant with a servo-controller (mean end-tidal CO_2=30.2mmHg). RO was quantified by moving average of the rectified phrenic neurogram. The RO of all piglets increased (p<.01) at the onset of CSN stimulation due to change in both frequency and neural tidal volume. However, by 50-60s, the average RO had decreased to 54±7% (±SEM) of peak RO in young piglets whereas in the 7 older piglets respiratory output was 78±5% of peak. This difference was significant (p<.025). Again, change in both frequency and neural tidal volume accounted for the changes in RO. These findings demonstrate that central neural mechanisms control the biphasic respiratory response to hypoxia. We conclude that neural maturation during the first 4 weeks of life permits the more sustained response seen in the older animals.

1352 INCOMPLETE SUPPRESSION OF NEONATAL GLUCOSE PRODUCTION BY EXOGENOUS GLUCOSE. Rosemary D. Leake; Carolyn Waters; Irene E. Karl; and Dennis M. Bier; Departments of Pediatrics, Harbor-UCLA Medical Center and Washington University, Los Angeles and St. Louis.

Incomplete suppression of glucose production (GP) during exogenous glucose infusions has been reported in newborns within the first 3 hours of life (Ped. Res. 14:576, 1980) and in prematures (Ped. Res. 14:586, 1980). To further evaluate the effects of birth weight, gestational age, and postnatal age on this process, we infused eight infants with [6,6-$^2H_2$]glucose prior to and after beginning an infusion of exogenous glucose at a rate approximately equal to normal neonatal hepatic glucose output (5 mg kg^{-1}-min^{-1}). The study subjects ranged from gestational ages of 32-40 weeks, had birth weights between 1320 and 4140 grams, and were studied between 14 hours and 5 days after birth. None was hypoglycemic prior to or during the study. Basal GP averaged 5.02 ± 0.43 mg·kg^{-1} min^{-1}. Exogenous glucose infusion produced incomplete suppression of GP in all infants averaging 50 ± 9% (range 3-72%). The degree of suppression did not correlate with gestational age, birthweight, or with postnatal age. However, there tended to be an inverse relationship between the degree of suppression of GP and the difference between the plasma glucose concentration before and after the exogenous glucose infusion. Thus, failure of complete suppression of GP in the newborn is present throughout the immediate postnatal period. Also, those infants with less suppression of GP tend to become more hyperglycemic during such infusions.

1353 FEEDING INDUCED BRADYCARDIA IN A PRETERM INFANT Lewis A. Leavitt, James N. Ver Hoeve, David N. Sheftel (spon. by R. Hong), University of Wisconsin-Madison, Department of Pediatrics, Madison, Wisconsin

An infant born at 31 weeks gestation and 1785 grams developed bradycardia (50 to 80 bpm) associated with feeding. At gestational age 39 weeks her physical and neurologic exam was normal. A chest X-ray and electrocardiogram were within normal limits. An EEG performed during a feeding-induced bradycardic episode was within normal limits. A radionucleitide study revealed no evidence of gastroesophageal reflux. Complete blood count, electrolytes and calcium were unremarkable. The infant was normoglycemic by Dextrostix during a bradycardic episode. A sleep pneumogram revealed no apneic episodes greater than 3 sec. Non-nutritive sucking was unassociated with bradycardia.

Bradycardia during feeding could be reliably produced by inflating a small rubber balloon at the level of the distal esophagus to a pressure of 50 mm of Hg.

Administration of atropine abolished the esophageal pressure induced bradycardia. This unusual case demonstrates a neurogenic, clinically significant bradycardia elicited by feeding and mediated via the vagus. The cardioinhibition induced by esophageal distention is treatable with anticholinergic medication.