● 1336 SIMULTANEOUS BLOOD PRESSURE AND ANTERIOR CEREBRAL ARTERY FLOW VELOCITY WAVE FORM ANALYSIS IN THE PRE-TERM INFANT. Alfred Karickhoff (Spon. by Louis Gluck)

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 Pourcelot's index of resistance as adapted by Bada into the pulsatility index (PI) (ratio of systolic & diastolic blood velocities) is suggested as a means of identifying changes in neonatal cerebral blood flow. Abnormality of PI in the anterior cerebral arteries, as detected by anterior fontanelle transcutaneous Doppler technique is suggested for anticipating & identifying intraventricular hemorrhage/intracerebral hemorrhage (IVH/ICH). Since PI reflects a ratio of relative flow velocities without absolutes for correlation, a study was done combining simultaneous recordings of flow velocity wave forms from left & right anterior cerebral arteries with direct umbilical artery catheter blood pressure wave forms. Forty-one studies were performed at 1 to 126 hr of life on 21 pre-term infants (Birthwts 880 to 4300 gm; gest. age 27 to 37 weeks). Analysis of tracings showed that near term and pre-term infants systolic and diastolic flow velocities have direct relationship to arterial blood pressure. PI data was obtained by analysis of beat-to-beat simultaneous blood pressure and flow velocity wave forms. PI data produced in this fashion has in all cases had absolute correlation with the presence or absence of IVH/ICH, verified independently by real time ultrasound brain studies. The technique is a simple, non-invasive bedside procedure.

1337 EFFECTS OF CHANGES IN CIRCULATION ON TRANSCUTANEOUS PCO₂ (TcPCO₂). Sudha Kashyap, Mark Stefanski, Karl Schulze and L. Stanley James, Div. of Perin. Med., Dept. of Ped., Columbia Univ., NY.

We compared TcPCO₂ with arterial CO₂ (PaCO₂) & continuous measures of heart rate (HR) & blood pressure (BP) (T°) in 14 infants (mean GA=32.7 weeks, mean wt.=2011 gms & mean postnatal age=1.76 days).

The Novamatrix heated electrode was mounted on the chest after calibration (mean calibration time=16.5 min.). The equilibration time (mean=12.15 min.) was directly correlated with GA (r=.74 p <.005). The correlation coefficient for TcPCO₂ vs PaCO₂ (n=29) was 0.76. The slope of the regression line was 1.15±0.18 with an intercept of 19.18±8.56.

HR & BP, indirect measures of tissue perfusion, were related to TcPCO₂ using time series & cross correlation analysis. Mean BP, accumulated HR & TcPCO₂ were calculated for 3 min. intervals in 4 infants. The relationship between changes in variables was examined following correction for biologic & methodologic lags. The change in TcPCO₂ between successive 3 min. intervals was strongly correlated with the changes in HR (Δ TcPCO₂ vs Δ HR), range r = -.58 to -.76 & occasionally with the change in BP range r = -.09 to -.46.

These results indicate that much of the variability in TcPCO₂ can be accounted for by changes in circulation. Variation in tissue perfusion occurring independently of changes in PaCO₂ could make estimates of PaCO₂ from TcPCO₂ highly misleading.

1338 HYPERACTIVE PMN CHEMOTAXIS (CX) AND RANDOM MIGRATION (RM) IN STRESSED NEONATES WITH SEPSIS. Abdul J. Khan, Warren N. Rosenfeld, Ramesh Jhaveri, Mathew Varghese, Parvin Khan and Hugh E. Evans. Department of Pediatrics, Jewish Hospital and Medical Center, Brooklyn, New York.

Normal newborn (NB) infants have defective PMN CX. PMN CX in sick NB infants has not been well studied. We determined the chemotactic index (CI) and RM in 10 stressed NB infants with "clinical sepsis" including 3 with bacterial pathogens isolated and compared them with 10 term normal NB. 8 of 10 patients had asphyxia with meconium aspiration; one congenital heart disease and 2 necrotizing enterocolitis. The patients had a mean age of 3 days, birth weight, 2817 gm and gestation, 38 weeks and all were receiving antibiotics. CI and RM were determined by a semimicro modification of Boyden's technique. PMNs placed in the upper compartment of the chamber were tested simultaneously in turn against 1) endotoxin activated serum (EAS), 2) E. coli derived chemotactic factors (ECF), 3) Hank's Solution alone, placed in the lower compartment. The ratio of migrated and total PMNs in first 2 sets were CI and in the third RM. Mean (SE) are presented (table). CIs and RM were significantly elevated in patients

	EAS	ECF	RM	compared with normal NB
patients	174 (35)	171 (44)	82 (19)	infants. The results
normal NB	65 (7)	69 (7)	26 (2)	suggests PMNs of NB may
P value	<0.002	<0.02	<0.002	become hyperactive during

stress compared with PMNs of healthy neonates. The basis for this may include factors such as prematurity, infection, asphyxia, drugs acting alone or in combination.

1339 FAILURE FOR CATCH-UP GROWTH AT 3 YEARS IN PRETERM INFANTS ≤ 1500 GM. Keith Kimble, Ronald L. Ariagno, David K. Stevenson and Philip Sunshine, Dept. of Pediatrics, Stanford Univ. School of Medicine, Stanford, Calif.

Growth charts based on data from normal children are commonly used to assess the growth of children who were premature. We examined the growth of 62 infants (EGA 30±2 wks and birthweight 1205±198 gm) who were born in 1975-77, followed prospectively for 3 years and who had normal developmental histories, physical exams and no apparent sequelae of prematurity at age 3 years:

	Age (mo)*	Wt (kg)	Length (cm)	OFC (cm)
Boys	10.0±1.4	8.3±1.0 (10)	69.9±3.0 (10)	45.4±1.2 (30)
girls	10.1±1.3	7.9±1.0 (15)	69.7±3.7 (25)	44.6±1.7 (40)
boys	21.6±1.6	11.1±1.2 (20)	81.8±3.0 (10)	48.3±1.2 (35)
girls	21.9±1.4	10.5±1.2 (20)	82.7±3.4 (25)	47.6±1.8 (50)
boys	33.8±1.7	13.0±1.4 (20)	91.3±3.5 (15)	49.6±1.5 (25)
girls	34.7±1.7	12.7±1.6 (20)	92.0±4.4 (25)	48.9±2.0 (45)

*chronological age corrected for prematurity; mean ± S.D.

Number in parentheses= National Center for Health Statistics

percentile of mean.

We concluded that growth charts prepared from data from normal children are not appropriate for children who had birthweights ≤ 1500 gm (except perhaps for head circum.) and that little catch-up growth occurs between one and 3 years in such infants. What factors are operative and whether this pattern is significant or whether there will be later catch-up growth is unknown.

1340 TRANSCUTANEOUS OXYGEN MONITORING, HYPOXIC RANGE AND CORRELATION WITH PROSTAGLANDIN E₁. Keith J. Kimble, Ronald L. Ariagno, David K. Stevenson and Philip Sunshine, Dept. of Pediatrics, Stanford Univ. School of Medicine, Stanford, California.

Poor correlation between transcutaneous PO₂ (tcO₂) and arterial PO₂ (PaO₂) during hypoxemia is a common clinical problem. Vasoactive drugs such as prazosin may also adversely affect correlation, even during normoxemia. No information exists, however, as to the effect of prostaglandin E₁ (PGE₁) administration on the relationship between tcO₂ and PaO₂. We, therefore, analyzed 201 simultaneous tcO₂-PaO₂ determinations in 6 infants, 32-38 wks gest. 1-18 days old (1 infant 137-141 days) receiving PGE₁ for ductus-dependent cyanotic congenital heart disease and 208 paired determinations from 4 infants with pulmonary disease of similar postnatal and gestational ages who were selected as controls. None of the infants were hypotensive or on vasoactive drugs other than PGE₁. TcO₂ measurements were made using a Litton Oxy-monitor[®]. The electrode (temp. 44°C) was placed on the anterior chest or abdomen. The PaO₂ was <60 torr in 83% of the paired determinations in the PGE₁ infants and 38% from the controls. The relationship between PaO₂ and tcO₂ was linear even during hypoxemia in the PGE₁ infants (r=0.83); however in the control infants it was curvilinear. A polynomial regression gave the best correlation (p<0.01) for the control infant data. TcO₂ monitoring in infants receiving PGE₁ provides relatively accurate estimations of PaO₂ even during hypoxemia. We speculate that in control infants changes in local blood flow induced by hypoxemia may result in a non-linearity between PaO₂ and tcO₂.

1341 WHOLE BLOOD PLATELET VOLUME MEASUREMENTS IN NEONATES Samuel L. Kipper, Lance Sieger and Michael Lubran. UCLA School of Medicine, Harbor-UCLA Medical Center. Departments of Pediatrics and Pathology. Torrance, California.

The MPV(mean platelet volume)and FLP(fraction of large platelets)were determined for 31 non-thrombocytopenic and 11 thrombocytopenic neonates. Samples were obtained by heel stick, by venipuncture, from cord blood, or from indwelling catheters. 0.1 ml unfractionated anticoagulated whole blood samples were analyzed using a Coulter model S+ cell counter, which determines MPV by direct measurement. The FLP was calculated by determining the fraction of platelets on a platelet volume histogram between 13 and 28μm³. There was no difference between MPV and FLP of capillary blood compared to catheter arterial blood samples. The platelets of 4 thrombocytopenic premature infants(< 100,000) were significantly larger(MPV=9.80, FLP=21.45) than the platelets from 7 non-thrombocytopenic premature infants (MPV=8.20, FLP=10.11). The platelets of 7 thrombocytopenic full-term infants were also significantly larger(MPV=9.61, FLP=28.34) than the platelets of 25 non-thrombocytopenic full-term infants (MPV=8.51, FLP=11.90) Nine of the thrombocytopenic neonates had proved or suspected sepsis; 1 had DIC with severe acidemia; and 1 had aortic thrombosis. This simplified method of platelet volume determination is particularly useful in neonates because of the small volume of blood required and suggests that thrombocytopenia associated with neonatal sepsis is due to increased destruction rather than decreased production.