1531 CARDIOMYOPATHY ASSOCIATED PERSISTENT PULMONARY HYPERTENSION (PPHN).

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Persistent pulmonary hypertension often occurs in infants with meconium aspiration syndrome or asphyxia. There is a subgroup of infants with PPHN in whom myocardiopathy predominates. We reviewed the course of 7 infants treated for PPHN who had increased heart size on chest x-ray, and/or an EKG with right ventricular hypertrophy. All patients had documented right-to-left shunting by echocardiogram either through PDA or foramen ovale. Mean B.W. was 3.13 ± .49 kg SD (range 2.32 to 3.74), G.A. was 38.85 ± 2.38 weeks SD, Apgars mean of 4.71 ± 2.49 SD at one minute and 6.57 ± 2.43 SD at five minutes. All patients required >90% inspired oxygen concentration, with 6 patients on 100% requiring mechanical ventilation. The following were mean ventilator settings on day one: FiO_2 98.57% ± 3.77 SD, rate 87 ± 34.95 SD bpm, IP 38.66 ± 12.35 SD cmH_2O, PEEP 5.5 ± 1.64 SD cmH_2O. The mean critical PCO_2 on day 1 was 23.85 ± 8.7 SD mmHg, with a mean PO_2 of 128.85 ± 109.02 mmHg SD. Six of 7 infants were treated with intravascular volume replacement and dopamine (mean maximum dose 18.58 ± 18.05 SD mcg/kg/min). In 4 surviving infants on dopamine and intravascular volume, the maximum dose of dopamine was associated with a reduction in inspired oxygen within a range of 25-40% within 48 hours followed by entrance into the transitional phase (decrease of 30% inspired O_2). The mean day of entry into the transitional phase was 6.4 ± 3.30 SD days of life. These infants represent a subgroup of neonates with PPHN. Cardiomyopathy in these infants appears to benefit most from intravascular volume replacement and dopamine therapy.

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†1532 PERIODIC BREATHING IN PRE-TERM INFANTS AS A PREDICTOR FOR SIGNIFICANT APNEA OR LIFE-THREATENING APNEA.

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Periodic breathing has been associated with "near miss SIDS" events in term infants. At present, studies have not demonstrated the relationship between periodic breathing in pre-term infants to prolonged apnea. The purpose of this study was to determine the predictive value of periodic breathing with regards to significant and/or life-threatening apnea. Periodic breathing was defined as three or more apneic pauses of greater than 3 sec. but less than 10 sec. within periods of normal respiration of 20 sec. or less. Significant apnea was defined as 3 pauses of greater than 10 sec. or any pause greater than 15 sec. with or without associated bradycardia. Life-threatening apnea was defined as any apneic event (inpatient or home monitor infants) which required vigorous stimulation and/or mouth-to-mouth resuscitation. Pneumograms for 153 pre-term infants were reviewed. All infants were studied prior to starting theophylline.

RESULTS:

	All Pre-term	Preterm w/o Apnea	Preterm with Significant Apnea	Preterm w/Life-threatening Apnea
No. Patients	153	91	62	8
Mean % P.B.	7.93	7.27	9.35	9.56
SEM	.94	1.17	1.56	3.48
GA (corrected)	31.7	31.6	31.9	35.8

8/62 infants (12.9%) with subsequent life-threatening apnea had initial pneumograms which showed significant apnea. We therefore conclude that periodic breathing alone is not predictive of apnea or life-threatening apnea. The presence of significant apnea on pneumogram tracings appears to be more predictive of subsequent life-threatening apnea.

†1533 EFFECT OF CLOSING THE DUCTUS ARTERIOSUS (DA) AT AGE 10 MIN IN PRETERM LAMBS.

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Clinical respiratory distress (RD), serial pulmonary blood flow (PBF), and 4 hr lung water in 6 preterm lambs after mechanically closing the DA at age 10 min (Group A) were compared with those in 8 lambs in which the DA was not closed (Group B). During fetal surgery at 128-132 d, an electromagnetic flow probe was placed on the postductal segment of the common pulmonary artery in both groups; a polyethylene snare was placed loosely around the DA in Group A. The lambs were delivered by C-Section 8 days later. PBF rose rapidly in both groups. The snare was tightened at age 10 min to close the DA (Group A) during which little clinical change was noted. PBF in Group A peaked close to the time of DA closure (median=10.6 min), earlier than the PBF peak in Group B (median=25.0 min; $p < .05$). Peak PBF as well as the PBF at 2 and 3 hr was lower in Group A ($p < .05$). Clinical RD was similar in both groups. Although lung water tended to be lower in Group A, this was not statistically significant.

Left-to-right DA shunting thus appeared to contribute significantly to the high PBF during and after the initial surge but had minimal or no effect on lung water. These data together with our previous experiments (Ped. Res. 17:124A,1983) suggest that the high PBF associated with ovine RD is a secondary phenomenon and point to defective pulmonary vascular resistance as the primary cause for the pulmonary edema found in this disease.

1534 DO VENO-VENOUS (VV) AND VENO ARTERIAL (VA) EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO) CAUSE DIFFERENT MODIFICATIONS OF HEMODYNAMICS AND PROSTANOID HOMEOSTASIS IN NEWBORN LAMBS?

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Prolonged VA or VV ECMO, acceptable modes of perfusion for neonatal respiratory failure, may alter hemodynamics and prostaglandin metabolism in unanesthetized newborn lambs. 48° after pulmonary/femoral artery and L atrial catheter placement, 14 lambs were placed on either partial VA or VV bypass at 80-100cc/kg/min for 21°, then weaned. Blood samples were obtained before, during, after ECMO. Serum thromboxane A_2 (TxB_2) and Prostacyclin ($PGF_{1\alpha}$) were determined by RIA. 1) VA ECMO caused a rise in mean systemic pressure from 94±5mmHg to 127±9mmHg ($p<.05$) at 4min with return to baseline by 15min. VV ECMO caused a rise in mean systemic pressure from 90±1mmHg to 111±1mmHg ($p<.01$) at 4min with return to baseline by 8min. 2) VA ECMO caused a rise in mean pulmonary artery pressure from 23±2mmHg to 51±6mmHg ($p<.02$) with a return to baseline by 30min. VV ECMO caused a rise in pulmonary artery pressure from 21±1mmHg to 61±1mmHg at 4min with a return to baseline at 30min ($p<.05$). 3) Thromboxane B_2 increased in the first 30min of VA bypass from 304±133pg/ml to 1206±272pg/ml ($p<.01$) and in VV bypass from 132±33pg/ml to 873±394pg/ml ($p<.05$) before returning to baseline for the remainder of ECMO. 4) 6-keto-prostaglandin $F_{1\alpha}$ increased in the first 30min of VA bypass from 320±191 pg/ml to 778±385pg/ml ($p<.05$) before returning to baseline. With VV ECMO there was no significant generation of 6-keto-$PGF_{1\alpha}$. Both VA and VV ECMO cause significant alteration in cardiopulmonary hemodynamics and prostanoid homeostasis in newborn lambs.

●1535 A CONTROLLED TRIAL OF GRANULOCYTE TRANSFUSIONS IN NEUTROPENIC NEONATES.

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To determine if granulocyte transfusions(Grtx) improve survival of neutropenic infants, we prospectively randomized 25 critically ill infants to +Grtx and -Grtx groups, matching for BW <1500 gm(n=8) or >1500gm(n=6) or necrotizing enterocolitis(NEC) (n=11). All infants had 2 absolute neutrophil counts(ANC) <1500 at presentation. Bone marrow neutrophil storage pools(NSP) were determined in 23/25. Grtx($0.1-0.9 \times 10^9$Gr/kg) were given daily until ANC >1500, yet only 5 infants with NEC needed >1 Grtx.

(x±SD)	n	GEST(wks)	BW(kg)	ANC	I:T	NSP≤7%	SURVIVAL
+Grtx	12	30.9±3.8	1.3±.6	888±120	.83±.17	3	7
-Grtx	13	30.3±3.4	1.4±.6	559±432	.82±.10	6	9

Blood CSF or peritoneal cultures were positive in 17/25 infants. 6/9 +Grtx and 9/12 -Grtx infants with a peripheral immature: total(I:T) granulocyte ratio ≥.8 survived. 1 +Grtx and 4 -Grtx infants with bone marrow NSP depletion ≤7% survived. 2 infants had a decreased PaO_2 after a 3rd Grtx without X-ray changes. Neither an I:T ≥.8 nor an NSP ≤7% predicted survival. Also, Grtx did not improve the survival of the total 25 infants or the 17 infants with positive cultures. 4/9 deaths related to neurologic compromise, while 4/16 survivors have permanent neurologic deficits. In addition, of the 6 +Grtx infants with NEC, 2/5 perforations occurred after transfusion, while 4 infants developed strictures and 5, short gut syndrome. We conclude: 1) neither an I:T ≥0.8 nor an NSP ≤7% predict mortality, and 2) Grtx do not improve survival. Repeated Grtx may increase the risk of respiratory compromise.

†1536 NEONATAL HYPERTENSION: A NATIONAL SURVEY.

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Umbilical arterial catheter(UAC) placement has been implicated in the etiology of neonatal hypertension(HBP). 119/163(73%) of NICUs responded to a survey regarding UAC care and placement and the incidence and outcome of HBP. Respondents identified 581 HBP among 70,186 admissions, defined as BP 95/63(mean 71 mmHg) for infants >1500gms, and 82/51(mean 60) for those <1500gms who accounted for 25% of HBP. 94% of HBP had UAC prior to or during HBP which complicated 0.82% of admissions(range 0 to 8%). HBP incidence was calculated according to UAC site and whether maintained with constant infusion(CI) or intermittent flushes(IF).

	CENTERS	HBP	IF	CI	IF vs. CI p value
High UAC	47%	0.97%	2.4%	0.7%	0.001
Low UAC	45%	0.75%	0.4%	1.1%	0.001
p value	NS	0.01	0.02	NS	

A comparable number of units preferred high UACs (T6-T10) to low UACs (L3-L5). Thromboembolism of the renal artery was stated as the most common etiology of HBP(63%), with heart failure the most common manifestation(44%), followed then by renal failure, respiratory distress, encephalopathy and retinopathy. 68% of centers initiated treatment for HBP alone, the rest required evidence of systemic symptoms. A favorable long term outcome was reported for 90% of HBP. These data suggest that both UAC position and its specific care influence the incidence of HBP, the greatest risk occurring with high UAC and intermittent heparin flushes.