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THE SAFETY OF ARGININE VASOPRESSIN (AVP) IN DONOR 1549 BLOOD FOR NEWBORNS. <u>Amaury Velez</u>, <u>Guillermo</u> Zeballos, David Clark, Julian Stewart, Harry S.Dweck. New York Medical College, Westchester County Medical Center Valhalla, New York.

Sick newborns have been found to have significantly elevated levels of AVP. The frequency of blood transfusions in sick neonates and the multiple physiological actions of AVP prompted

neonates and the multiple physiological actions of AVP prompted us to determine the content of AVP in donor blood. In keeping with standard blood banking practices, 80 ml of blood were drawn from each of eight donors, mixed with 7 ml of anticoagulant (CPD) in standard blood bank bags and refrigerated at 4°C. Daily samples were drawn on each of five successive days. The plasma was acidified and frozen at -20°C until the time of assay. AVP was extracted by bentonite procedure modified from Skowsky technique in duplicate and measured by radioimmunoassay in triplicate. Only 2 of 8 donors had higher than normal values (6 & 7 pg AVP/m1) at the time of the initial sample. AVP levels decreased from day 1 to day 5 with the greatest fall in the first 24 hours.

	AVP LEV	ELS (pg.	/ml) (N	1 = 8)			
	(1 pg = 2.5 microunits)						
DAYS							
	1	2	3	4	5		
mean (pg/ml)	3.63	2.85	2.72	2.22	2.08		
(S.E.)	(0.70)	(0.28)	(0.47)	(0.43)	(0.21)		
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We conclude that the use of banked donor blood in newborns is not likely to result in excessive infusion of AVP.

***1550** MARKEDLY ELEVATED SERUM CALCITONIN (CT) REFRACTORY TO SERUM CA CHANGES AND CA-RESPONSIVE SERUM PARATHYROID HORMONE (PTH) IN HYPOCALCEMIC VERY LOW BIRTH WEIGHT (VLBW) INFANTS. P.S.Venkataraman, K.E.Blick, H.D.Fry, R.K.Rao. Dept. Pediatr., Univ. Okla., Okla. City (Spon. O.M.Rennert). In VLBW infants, we studied the these that in early peopatal †1550

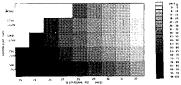
Okla., Okla. City (Spon. O.M.Rennert). In VLBW infants, we studied the theses that in early neonatal hypocalcemia, I)serum PTH would rise, 2)serum CT would decline, and in response to IV Ca infusion, 3)serum PTH would be lowered, 4)serum CT would rise. Fifteen AGA infants, gestation <32 wks., bt.wt. <1500 gms. were enrolled in the study. In 8 infants whose serum Ca declined <6.0 mg/dl, changes in serum Ca,Mg,P,PTH,CT and blood iCa were evaluated at 8 ±4 hrs.(baseline), when serum Ca declined to < 6.0 mg/dl (pre-Ca), immediately post-infusion of 18 mg/kg of Ca⁺⁺ as Ca gluconate (post-Ca). Serum Ca declined from 7.9 ± 0.6 baseline (mean ± s.e.) to 5.2 ± 0.2 mg/dl pre-Ca. p < 0.025 and rose to 9.17 ± 0.74 mg/dl post-Ca. p < 0.025 and pre-Ca, p < 0.025, and rose to 9.17 ± 0.74 mg/dl post-Ca, p < 0.025 and 7.1 ± 0.5 mg/dl at 8 hr.post-Ca. Whole blood iCa declined from 4.82 ± 0.24 7.1 ±0.5 mg/dl at 8 hr.post-Ca. Whole blood iCa declined from 4.82 ±0.24 to 3.72 ±0.19 mg/dl pre-Ca, p < 0.025, and rose to 6.68 ±0.32 post-Ca, p < 0.025 and 4.12 ±0.21 mg/dl at +8 hr. post-Ca. Serum Mg and P did not change significantly. Serum PTH (RIA, mid-molecule, N 29 to 85) rose from 116±17 to 204 ±34 p mol/l pre-Ca, p < 0.01 and declined to 149±22 post-Ca, p < 0.01, and was 187 ±28 p mol/l at +8 post-Ca. Serum CT (RIA, adult N 32 to 102) was 751 ±150, 738±115, 895 ±206 and 551 ±94 pg/ml at these times, p < 0.005 vs. term infant values, changes not significant. Thus, in infants <32 wks. gestation, serum PTH rises in early neonatal hypocalcemia and is suppressed by IV Ca infusion; serum CT is markedly elevated and is not lowered in early neonatal hypocalcemia, and does not rise further in response to IV Ca infusion in VLBW infants. We suggest that hypercalcitonemia occurs in VLBW infants, and serum CT concentrations are unresponsive to changes in serum Ca.

THE INFLUENCE OF GESTATIONAL AGE(GA) VS. BIRTHWEIGHT (BW) ON NEONATAL MORTALITY(NM) OF PREMATURE INFANTS: 1551 **1551** A DUTCH NATIONWIDE STUDY.<u>S.P.Verloove-Vanhorick,J.H.</u> <u>Ruys,R.Brand</u>.Leiden University Hosp, Dept of Ped and Dept of Med

Statist, Leiden, The Netherlands (sponsored by William Oh). During the year 1983, NM data were collected prospectively on all liveborn infants with a GA of 32 weeks(wks) and/or a BW of <1500 gr, admitted to 102 neonatal wards(level 1,2 and 3 hospi-tals) in the Netherlands. The 1208 infants, admitted to the study (133 infants excluded because of lethal congenital malfor-mations or uncertain GA) represent 94% of the total liveborn population of this category in the Netherlands. Results were:

Neonatal Mortality

1203 22% total 28% (range 83%~25 wks-8%~31 wks) <32 wks 906 (1500 gr 983 24% (range 63% 500-749 gr-11% 1250-1499 gr) By fitting these data to a loglinear model (figure) we were able to demonstrate that GA has a significantly larger influence on NM than BW. We conclude that GA rather than BW should be used in NM statistics of premature infants:



COMPARISON OF TWO ANIMAL MODELS IN ARTIFICIAL SUR-FACTANT THERAPY FOR HYALINE MEMBRANE DISEASE (HMD). †1552 Dharmapuri Vidyasagar, Haruo Maeta, Hiroo Matsuda, Tonse Raju, Rama Bhat, Mark Anderson, Margaret Go, Urmila Dahiya Eunice John, Yvette Roberson. University of Illinois Hospital, Department of Pediatrics, Chicago, Illinois. Preterm baboon (B) and lambs (L) have been used as models for

different surfactant studies. Varying responses have been record-ed. We studied and compared the responses of these models to S-TA therapy. Animals delivered prematurely (8:76% and L:83% term) by C-section were used. All had HMD. S-TA 100 mg/kg was instilled into trachea at 2 hrs. of age. Controls received no S-TA. Sequential a/APO2 data are shown. Treated B had sustained improvement in

		a/APU2 Baboon VS Lamb					
	n	pre-surf.	Post 1 hr.	Post 4 hr.	Post 6 hr.		
	0.20+1.10	0.53+0.05***	0.44+0.11***	0.42 <u>+</u> 0.08***			
	0.23+0.11	0.23+0.06	0.13+0.05	0.13+0.05			
Lambs			0.27+0.08**	0.22 + 0.12*	0.06+0.01		
Lambs (5)	0.10 ± 0.05	0.10 ± 0.02	0.07 ± 0.04	0.03±0.01			
() = r	1 1	Table: x±S.	.D., *p<0.05,	**p<0.005, ***	p<0.001		

(-) = n Table: x15.D., *p<0.05, *xp<0.000, *xp<0.001a/AP02 up to 16 hrs. and mean airway pressure dropped from 14.5<u>+</u> 1.1 to 10.2<u>+0.5</u> cmH₂0 p<.001. In lambs improvement in a/AP02 was significant but transient lasting only 2-3 hrs. with deterioration by 4-5 hrs. Pressure vol. curves at autopsy showed significantly larger hysteresis in treated baboons than lambs. Two other treated baboons could be maintained alive for 36 hrs. We conclude: 1) Differences in response to S-TA therapy may be species related, 2) Baboon HMD model may be better for studies of long term effects of surfactant therapy than the lambs.

EARLY INDOMETHACIN (IND) DOES NOT PREVENT

EARLY INDOMETHACIN (IND) DOES NOT PREVENT **1553** BRONCHOPULMONARY DYSPLASIA (BPD) IN A RANDOMIZED CLINICAL TRIAL M.J. Vincer, A.C. Allen, D.A. Stinson, J.R. Evans, C.G. Nwaesei, E. Rees, A. Fraser, Dalhousie University, Dept. of Pediatrics, Halifax, Nova Scotia Thirty infants < 1500 g at birth on IPPB or CPAP by 12 hours were entered in a randomized double blind controlled trial to test the efficacy of IND in preventing BPD. 15 treated infants received IND at 12, 24 and 36 hours of age while 15 control infants received normal saline at these times. The groups were infants received normal saline at these times. The groups were similar for birth weight, gestational age and sex.

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		BPD		IRDS		IVH GRADE				
	NUMBER	MILD	CYSTIC	MOD	SEV	182	3&4	SPDA	DIED	
Indomethacin	15	5	3	5	5	7	5	1	3	
Control	15	7	1	5	8	5	4	5	2	

Although there was a 65% probability of detecting a 50% reduction in BPD, there was in fact no difference in the incidence of BPD between the two groups. The incidence of IRDS and IVH was similar in the two groups. There was a tendency towards reduced incidence of symptomatic PDA (SPDA) in the IND group but it did not reach significance. There were no cases of NEC in either group and the incidence of pneumothorax and RLF was similar. Length of stay, length of ventilator support and length of oxygen therapy did not differ between the two groups

IND may reduce the incidence of SPDA. However, when given within 12 hr of birth, IND does not reduce the incidence or severity of BPD, or the need for ventilation or oxygen.

EFFICACY OF THEOPHYLLINE FOR PREVENTION OF

•1554 EFFICACY OF THEOPHYLLINE FOR PREVENTION OF POST-EXTUBATION RESPIRATORY FAILURE IN INFANTS LESS THAN 1250 GRAMS. <u>Rose M. Viscardi,</u> <u>Roger G. Faix, Thaddeus H. Grasela, Joanne J. Nicks, University of</u> <u>Michigan, Departments of Pediatrics, Pharmacy, and Respiratory</u> Therapy, Ann Arbor. (Spon. by SM Donn).

Because methylxanthines modulate apnea and enhance intercostal and diaphragmatic contractility, we hypothesized that administration of the ophylline to infants $<1250~{\rm gm}$ on low ventilatory settings would of theophylline to intants < 1250 gm on low ventilatory settings would facilitate extubation and prevent post-extubation respiratory failure. A prospective, randomized, blinded, placebo-controlled trial was performed. Twenty-five infants [11 placebo (P), 14 treated (T)] who were < 1250 grams on assisted ventilation with FiO₂ \leq .30, PIP \leq 20, IMV \leq 10 were enrolled. Electrolyte abnormalities and anemia were corrected before entry and absence or stability of IVH was confirmed by cranial sonography. Prior to extubation, T infants received 6 mg/kg theophylline loading dose IV or PO and then a maintenance dose 2 mg/kg q 12h for 5 doses. Control infants received equal volumes for mg/kg q 12h for 5 doses. Control infants received equal volumes of normal saline. No significant differences existed between groups for birthweight, GA, duration of ventilation, and peak FiO₂. Nine of 14 T infants and only 1 of 11 P infants were successfully extubated for at least 5 days (p=0.01). Of 5 T infant failures, 2 had congestive heart failure with patent ductus arteriosus and 3 had severe apnea. Of 10 P infant failures, 8 had recurrent apnea and 2 failed to wean from the respirator during the study period. Seven of 9 P infants were later successfully extubated with theophylline. We conclude that theophylline therapy prior to extubation of infants < 1250 gms is effective in preventing osst-extubation respiratory failure. effective in preventing post-extubation respiratory failure.