†1483 HUMAN MILK LIPASE ASSOCIATED WITH BREAST MILK JAUNDICE: SUBSTRATE SPECIFICITY.

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Human milk samples that inhibit bilirubin-UDP-glucuronyl transferase (UDPGT) activity in vitro are associated with the syndrome of breast milk jaundice. We have reported that the inhibitor appears to be non-esterified fatty acid and the milk samples associated with the clinical syndrome exhibit significantly higher unstimulated lipase activity ($P<0.01$) and specific activity ($P<0.001$) than controls using a pH-stat assay with tributyrin as substrate. (Pediatr. Res. 14:1328, 1981).

The present study was done to test whether that lipolytic activity was due to a specific lipase or to a non-specific esterase. Two water soluble substrates were tested: p-nitrophenyl (p-NP) acetate (for non-specific esterase) and p-NP butyrate (a fatty acyl ester). Assays with these substrates were performed simultaneously along with the above tributrin assay using 23 human milk samples as sources of enzyme. Enzyme units are defined as 1 μM of substrate released by 1 ml of milk per minute. Lipase activity (tributrin assay) ranged from 3.1 to 11.1 units. Activity vs. p-NP butyrate (fatty acyl ester) ranged from 1.1 to 23.7 units and activity vs. p-NP acetate (non-specific ester) ranged from 0.3 to 2.6 units. Tributyrin assay activity correlated positively with p-NP butyrate activity ($r=0.66$, $P<0.001$) but did not correlate with p-NP acetate activity ($r=0.07$, N.S.). These results are consistent with the hypothesis that the unstimulated enzyme activity we described associated with breast milk jaundice is produced by a lipase and not a non-specific esterase.

1484 PERIVENTRICULAR (PVH), INTRAVENTRICULAR (IVH), PARENCHYMAL HEMORRHAGE (PH) AND VENTRICULAR DILATATION (VD) IN TWIN INFANTS.

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It is generally expected that the second-born twin may have an acute distress due to the adverse effects of intrapartum period and the small twin is usually a subject for intrauterine chronic distress which may both lead to an intracranial bleed. We studied 21 pairs of low birth weight (LBW) twin pregnancies to see whether the incidence and severity of US abnormalities, such as PVH, IVH, PH and VD, are different between the pairs. The study population had a gestational age of ($\bar{X}$) 32.8 wk, birth wt of 1649 g, 5 minute Apgar score of 7.9 and the postnatal age at first US examination of 45.6 h. Comparison between twin (A) and (B) and also twin Small (S) and Large (L) follows:

		US-#1			US-#2			US-#3	
	n	VD	PVH,IVH,PH	n	VD	PVH,IVH,PH	n	VD	PVH,IVH,PH
Twin-A	21	4	2	9	4	0	8	4	0
Twin-B	21	5	2	11	1	1	8	4	0
Twin-S	21	4	2	9	4	0	7	4	0
Twin-L	21	5	2	11	4	1	9	4	0

The entire comparison also shows no significant difference between the sequence and size based groups as they relate to cranial US and other relevant clinical parameters. There appears to be no evidence of relative increase in the incidence or severity of PVH,IVH,PH and VD in twin infants during immediate, early and late neonatal period.

1485 MATURATION OF RESPIRATORY PATTERNS IN VERY LOW BIRTH WEIGHT (VLBW) INFANTS: INFLUENCE OF POST-CONCEPTIONAL AGE (PCA).

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Periodic breathing (PB) and prolonged sleep apnea (>15 sec) (PSA) often are present in VLBW infants, may be secondary to CNS immaturity and can result in serious complications. Early reports show the incidence of PB peaks during the second and third week of life; our observations indicate a different pattern in VLBW infants. Ten VLBW infants (BW=1130±0.21 gms, GA=29.9±1.4 wks) were studied weekly from birth. A total of 70 pneumograms (each > 300 min. sleep) were analyzed for % PB, PSA, and bradycardia (B) (<80 BPM). All infants were off mechanical ventilation by day 4 of life. Eight infants received aminophyllin (A) for a mean of 5.9±2.2 wks, but only 4 were on A in the first wk of life. No attempt was made to control A administration since the study was not designed to dictate clinical care.

Percent PB, PSA, and B were assessed in relation to postnatal age (PNA), and PCA. Significant PB (>5%) increases with PNA, but does not occur prior to 33 wks PCA.

PNA(wk)	1	2	3	4	5	6	7	8	9	10	
%PB>5	0	11	20	22	50	12	67	50	60	67	
n	8	9	10	9	8	7	6	4	5	3	
PCA(wk)	29	30	31	32	33	34	35	36	37	38	39
%PB>5	0	0	0	0	11	29	56	38	44	60	75
n	2	3	5	8	9	7	9	8	9	5	4

In contrast to PB, B and PSA episodes decrease with PNA and PCA.

Increased PB occurs in VLBW infants despite A and was present only after 32 wks PCA. This finding, requiring further evaluation, may be important to understanding maturation of respiration in VLBW infants.

†1486 TIN PROTOPORPHYRIN (TP) AND THE EXCRETION RATE OF CO (VeCO) IN ANTIBIOTIC-TREATED NEONATAL RATS WITH ARTIFICIAL HEMATOMAS.

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Previous studies showed that TP inhibits tissue heme oxygenase activity (HO) and lowers serum total bilirubin levels [B], but does not effect the VeCO of neonatal rats with and without artificially created hematomas (H). It is possible that intestinal bacteria play a role in this model by metabolizing excreted heme to CO. Rat litters, with intestines sterilized by antibiotics, were divided into 3 groups of 3 rats each. At 12 h postpartum (t=0), the TP/H rats were injected with TP (65 μmoles/kg). Hematomas were given to H and TP/H rats (t=45 h).

	VeCO (mean ± SE, μl/kg/h, n=8)					
GROUP	t=43	t=52	t=63	t=77	t=88	t=112
Saline(S)	48±1	50±3	51±2	47±2	47±2	38±2
Hematoma(H)	51±2	74±3	87±4	81±2	71±2	47±3
TP/Hematoma(TP/H)	49±2	53±2	69±4	77±3	80±3	51±2

Rats were sacrificed at t=113 h. The [B] of the TP/H rats was not reduced, but hepatic HO activity was suppressed ($p<.005$) when compared to the H control group. The overall average VeCO of H rats was increased by ∿ 45% over S rats and TP was effective in delaying and suppressing this increase; the inhibition of average VeCO was ∿ 25%. The results suggest that in antibiotic-treated rats with artificial hematomas: 1) single dose TP was effective in lowering VeCO and thus total bilirubin formation and 2) intestinal bacteria may contribute significantly to VeCO, if endogenous heme degradation is inhibited.

1487 PROLONGED HYPERCALCEMIA IN A NEONATE FOLLOWING RHABDOMYOLYSIS AND ACUTE RENAL FAILURE (ARF).

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Hypercalcemia occurs in about 1/3 of adults during the diuretic phase of rhabdomyolysis associated ARF. This etiology of hypercalcemia in neonates has not been reported previously.

An asphyxiated (Apgars 1 and 3) postmature baby, born by emergency Caesarian section, developed oliguric ARF. Urine on day 3 was heme positive without r.b.c. and serum CPK was 4855 U/L (CPK MB=301 U/L). During the 10 day oliguric period, 587 mg/kg of intravenous Ca gluconate was used to maintain normocalcemia, and peritoneal dialysis was required. Two weeks after diuresis began, hypercalcemia developed. Serum Ca persisted between 13 and 16 mg/dl for 6 weeks. Postulated mechansims for hypercalcemia following rhabdomyolysis and ARF include 1) elevated serum calcidiol, 2) inappropriate release of PTH, and 3) resorption of muscle calcifications. In this case serum PTH was low normal despite the marked hypercalcemia. Calcitriol level was 131 pg/ml and calcidiol level was 27 ng/ml (both WNL).

Late hypercalcemia can occur in stressed neonates with signs of myoglobinura associated ARF. The hypotheses of inappropriate PTH release and resorption of muscle calcifications are supported by this case.

†1488 PROPHYLACTIC INDOMETHACIN (I) FOR THE PREVENTION OF PATENT DUCTUS ARTERIOSUS (PDA).

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To assess the efficacy of prophylactic I therapy in decreasing the incidence of PDA, premature infants < 1400 g birthweight with respiratory distress (O_2 or ventilator (IMV) requirement at > 6 hrs. of age) were randomized to receive intravenous I or placebo (P). Infants were pair-matched for birthweight, gestational age and severity of respiratory distress at the time of randomization: one of each pair received I, the other P. I (0.2 mg/kg) was given intravenously at < 24 hrs. of age and every 12 hrs. thereafter for 3 doses total. After initial 3 doses, I was prescribed in any infant who developed a hemodynamically significant PDA. Courses of study infants have been analyzed for PDA and severity of respiratory disease (time on IMV and on O_2). Sequential analysis has been used to monitor possible adverse effects of therapy: intraventricular hemorrhage (IVH), death. Forty-two babies have been entered into the study and 16 pairs have been completely analyzed:

Group	PDA (%)	O2 (days)	IMV (days)	IVH (%)	IVH (grade)	Death (%)	
Indo	6.2	15	11	93	2.6	19	
Placebo	43.8	15	12	62	1.5	19	
p	<.05*	NS	NS	NS*	NS*	NS	*(χ^2 Yates)

Results: Prophylactic I decreases incidence of PDA but has no effect on respiratory disease. The trend toward increased incidence and severity of IVH is not statistically significant. Ongoing study monitored by sequential analysis will determine if trend toward increased incidence and/or severity of IVH is real.