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EFFECT OF PHOTOTHERAPY (Px) AT 3 BILIRUBIN (bili) THRESHOLDS IN TERM NEONATES WITH PHYSIOLOGIC HYPERBILIRUBINEMIA (H-B). Andrew C Argent, Alan D Rothberg, Peter A Cooper (Spon by M. Jeffrey Maisels), Witwatersrand Univ., Johannesburg Hospital, Dept Pediatrics, Johannesburg, S. Africa.

The risk of bili-encephalopathy following non-hemolytic H-B in term neonates has not been fully evaluated. Prophylactic Px is frequently used to "protect" neonates from possible sequelae of H-B. We studied the effect of Px on peak bili levels and duration of hospitalisation in 92 neonates with physiologic H-B. Infants were randomised for Px at bili levels (in mg/dl) of 10 (Gp A), 15 (Gp B) or 17.5 (Gp C). Px was continued until bili <10 (Gp A) or <15 (Gps B, C). The results were as follows:

Group	A (n=32)	B (n=32)	C (n=28)
Bili on entry (Mean $\pm$ SD)	11.7 $\pm$ 2.5	11.4 $\pm$ 2.5	10.6 $\pm$ 2.5
No. treated with Px (%)	31 (97)	15 (47)	5 (18)
Max bili (Total Gp)	13.2 $\pm$ 2.2	13.9 $\pm$ 2.9	12.6 $\pm$ 3.3
(Mean $\pm$ SD) (Px Gp)	13.4 $\pm$ 1.9	16.5 $\pm$ 1.2	18.6 $\pm$ 0.9
Days in hospital (Total Gp)	5.8 $\pm$ 1.8	5.6 $\pm$ 1.2	5.4 $\pm$ 1.4
(Mean $\pm$ SD) (Px Gp)	5.9 $\pm$ 1.3	6.2 $\pm$ 1.1	7.2 $\pm$ 1.8
Discharge bili (Total Gp)	9.1 $\pm$ 2.0	9.6 $\pm$ 3.0	10.3 $\pm$ 3.0
(Mean $\pm$ SD) (Px Gp)	9.1 $\pm$ 2.0	11.1 $\pm$ 2.8	13.1 $\pm$ 5.8

"Complications": 2 in Group C - 1 exchange / 1 readmission (H-B)

Early Px for bili >10 was associated with fewer bili's of 15-18 (p<.05), but did not significantly shorten hospitalisation and resulted in Px to $\pm$ 100% of the group. Late Px may result in "complications". Prophylactic Px for non-hemolytic H-B is arguable, but when used, a threshold of 15 appears appropriate.

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EARLY INTERVENTION FOR AT RISK PREMATURE NEONATES. Alan D Rothberg, Muriel Goodman, Peter A Cooper, Jennifer D Cartwright (Spon by M. Jeffrey Maisels), Witwatersrand Univ., Johannesburg Hospital, Depts of Pediatrics & Physiotherapy, Johannesburg, South Africa.

In a prospective study of premature neonates of birthweight <1700gm, a) we validated a 12 point neurodevelopmental assessment score (NDAS) assigned at 3, 6, 9 and 12 months corrected age, against a Griffiths developmental quotient (DQ) at 12 months corrected age; b) we assessed the value of early physiotherapy intervention during the first year. According to the NDAS at 3 mths, infants were classified as normal (score 0-4), at risk (5-9) or probably impaired (10-12). Normal (N) and at risk (R) infants were randomised into intervention (+) and control (-) groups. All infants with scores of 10-12 were treated. N+ and R+ infants received physical therapy as outpatients and at home. A physical therapist assigned the NDAS at 3, 6, 9 and 12 mths; the Griffiths test was performed at 1 year by a psychologist blinded for infant group. Fifty one infants have been tested (28N, 23R). Infants classified as R according to the NDAS had a mean DQ of 95.9 $\pm$ 9.9 at 1 yr vs 102.6 $\pm$ 8.7 for N infants (p<.02). Intervention had no effect in N or R infants: N+ and N- had similar NDAS's of 2; 2; 1; 0 at 3, 6, 9 and 12 mths resp. Similarly R+ and R- had similar scores (6; 3; 2; 1 vs 6; 4; 2; 1 at 3, 6, 9, 12 mths). Our NDAS appears to predict poorer outcome as assessed by the Griffiths DQ at 1 year, while early intervention has no apparent effect on development over the same time period.

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A-NEONATAL STATUS SCORE OF TRANSPORTED NEWBORNS AS A PREDICTOR OF SHORT TERM MORBIDITY AND MORTALITY. Rita L. Saldanha, Grant W. Somes, Lillian M. Ruckman, Cathy J. Conklin and Arthur E. Kopelman (Spon. by Jean F. Kenny), East Carolina Univ. Med. Sch., Pitt Mem. Hosp., Dept. of Peds., Greenville, NC.

A Neonatal Status Score (NSS) was used to evaluate neonates at the referring hospital (T₁). Temperature, heart rate, blood pressure, response to noxious stimuli and dextrostix were scored 0-2, 0 being the worst score. The same items were scored again after arrival back at our hospital (T₂).

Two hundred and five infants were transported between Sept., 1980 and Dec., 1982. The mean NSS at T₁ was 12.3 and at T₂ was 13.7. T₂ was significantly higher than T₁ (p<.001). There was a strongly positive correlation between the NSS (at either T₁ or T₂) and survival (p<.001 for each). There was no correlation between the NSS at T₁ and neurologic morbidity (ICH, seizures, hydrocephalus, or abnormal neurologic examination at discharge). A low score at T₂ correlated with the occurrence of seizures.

One hundred sixty-three transports were done by neonatal nurse clinicians and 42 by residents. There was no difference in the NSS before or after transport between infants transported by neonatal nurse clinicians or residents, suggesting that they performed comparably with equivalently sick patients.

The NSS is easily scored and is an accurate predictor of mortality. As an objective measure of an infant's condition and risk of mortality prior to transport, the NSS can be used to compare the prognosis of groups of infants transported or treated in different ways.

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ENERGY PROTEIN UTILIZATION AND GROWTH IN LOW BIRTH WEIGHT FORMULA (LBWF) AND HUMAN MILK (HM) FED PRETERM INFANTS (PT). Guy Putet, Jacques Senterre, Jacques Rigo, Bernard Salle (Spon. by Francis H. Glorieux). INSERM U34 and Neonatal Depts, Lyon, France and Liège, Belgium.

Influence of pooled human milk and LBWF feeding on growth, fat and protein accretion in PT was studied by the combined techniques of 3 days nutrient balances and indirect calorimetry. Two groups of PT, LBWF (n=6; BW:1353 $\pm$ 255 g; GA:30.3 $\pm$ 1.0 wks) and HM (n=6; BW:1318 $\pm$ 115 g; GA:30 $\pm$ 1.5 wks) were studied twice. One study was performed at 33 (I) and the second at 36 wks of postconceptional age (II). Between studies weight gain was 13.5 and 22.9 g/kg/d in HM and LBWF groups respectively (p < 0.001); length gain 1.0 $\pm$ 0.3 (HM) and 1.4 $\pm$ 0.2 cm (LBWF) (p < 0.05); head circumference gain 1.0 $\pm$ 0.2 (HM) and 1.1 $\pm$ 0.1 cm (LBWF) (NS). Energy and protein balance results are shown in the table (m $\pm$ s.d.):

Study		Energy (Kcal/kg/d)		Protein (g/kg/d)	
		HM	LBWF	HM	LBWF
I	Absorbed intake	87 $\pm$ 11	117 $\pm$ 5	2.11 $\pm$ 0.4	2.81 $\pm$ 0.1
	Oxidized	46 $\pm$ 6	57 $\pm$ 3	0.5 $\pm$ 0.1	0.5 $\pm$ 0.1
	Stored	41 $\pm$ 8	60 $\pm$ 6	1.6 $\pm$ 0.4	2.3 $\pm$ 0.2
II	Absorbed intake	102 $\pm$ 11	122 $\pm$ 3	2.0 $\pm$ 0.2	2.9 $\pm$ 0.1
	Oxidized	51 $\pm$ 3	63 $\pm$ 4	0.5 $\pm$ 0.1	0.8 $\pm$ 0.2
	Stored	51 $\pm$ 9	59 $\pm$ 3	1.5 $\pm$ 0.2	2.1 $\pm$ 0.2

Fat deposition accounted for 25% (HM) and 23% (LBWF) of the weight gain in Study I and for 29% (HM) and 26% (LBWF) in Study II. In conclusion, these data show that the post-natal protein retention was higher in the LBWF than in the HM fed PT and fat deposition was important for weight gain.

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OXIDATIVE METABOLISM OF ¹³C MEDIUM CHAIN TRIGLYCERIDE (MCT) FED TO PRETERM INFANTS (PT). Guy Putet, Alice Thelin, Michel J. Arnaud, Guy Philippoussian, Jacques Senterre, Nahed Fahmy and Bernard L. Salle (Spon. by: Francis H. Glorieux). INSERM U34 and Neonatal Depts, Lyon, France and Liège, Belgium, and Nestlé Res. Dept., Vevey, Switzerland.

Fat malabsorption is frequent in preterm infants fed either breast or formula milk and improves when part of milk fat is replaced by MCT. The metabolic fate of MCT is not well known in PT. Five PT (BW: 1771 $\pm$ 100 g; GA: 34 $\pm$ 1 wks; age at study: 24 $\pm$ 4 d) fed a formula containing 50% of fat as MCT were given a known amount of ¹³C-trioctanoin on the second day of a 3 day balance. Continuous indirect calorimetry was performed during the 24 hours following the ¹³C-MCT ingestion and energy expenditure, total carbohydrate, lipid and protein oxidation were determined. Continuous sampling of expired CO₂ allowed ¹³C/¹²C ratio measurements over the 24 hour period and the amount of MCT oxidized was then derived. Results are shown in the table (per kg/d, m $\pm$ s.d.):

	CHO (g)	Fat (g)	Protein (g)	Energy (Kcal)
Absorbed	12.4 $\pm$ 5.3	4.9 $\pm$ 0.2	2.5 $\pm$ 0.3	107 $\pm$ 5
Oxidized	11.7 $\pm$ 4.0	1.6 $\pm$ 0.6	0.6 $\pm$ 1.1	64 $\pm$ 6
Stored	0.7 $\pm$ 1.3	3.3 $\pm$ 0.8	1.9 $\pm$ 0.1	43 $\pm$ 10

Elimination curves of ¹³C in expired CO₂ were similar in all infants but were different in amplitude. Mean MCT oxidation was 27 $\pm$ 14% (range 6-43%) and accounted for 31% (range 7-47%) of the total fat oxidation. In conclusion, MCT oxidation shows considerable variations between PT infants. Furthermore no correlation with energy expenditure and with total fat oxidation is observed.

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CARDIOVASCULAR RESPONSE TO HYPOXIA IN PUPPIES DURING NATURAL SLEEP. Janis I. Schaeffer, Gabriel G. Haddad. Dept. of Pediatrics Columbia Univ.-Coll. of P&S, NY.

To examine the maturation of the cardiovascular response to hypoxia (FiO₂=.15) during natural sleep, we studied 48 chronically instrumented unanesthetized beagle puppies at 15 and 30 days of age. We measured the RR interval and mean aortic blood pressure (MAP). In addition, in a subset of 10 puppies, we measured cardiac output (CO) by the Fick principle. After baseline measurements, puppies were exposed to hypoxia for 1-2 hrs. During normoxia, at 15 days of age: MAP was lower (mean 10%, p<.025) and CO higher (mean 20%, p<.05) in REM than in quiet sleep. There was no difference in RR interval between the 2 sleep states. In the older puppies, similar results were obtained except that there was no difference in CO between REM and quiet sleep. In response to hypoxia at 15 days of age, there was a decrease in CO (mean 30%, p<.05), an increase in MAP (mean 8%, p<.025) and no change in RR interval during REM sleep. In quiet sleep, MAP and RR interval did not change; CO could not be accurately measured as periods were usually less than 3 min. in duration. In the older puppies, hypoxia induced a decrease in CO in both sleep states (mean 30%, p<.025), a decrease in RR interval in quiet sleep and an increase in MAP during REM sleep. We conclude that in puppies: 1) CO is dependent on sleep state during normoxia at 15 but not at 30 days of age; with hypoxia, CO is reduced in both sleep states; 2) systemic vascular resistance is lower in REM sleep when compared to quiet sleep in normoxia and increases with hypoxia in both sleep states and 3) stroke volume is smaller in quiet than in REM sleep in normoxia and decreases with hypoxia in both sleep states.