

RESPIRATORY WATER LOSS AND OXYGEN CONSUMPTION IN NEWBORN INFANTS DURING PHOTOTHERAPY.

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Phototherapy has been considered to increase insensible water loss. Water loss from the skin can not explain this increase (Ped Res 26:514,1989).

In the present study respiratory water loss (RWL, ml/(kg·min)) was measured together with oxygen consumption ($\dot{V}O_2$, mg/(kg·min)) in eleven fullterm newborn infants before and during phototherapy.

The method for determination of RWL and $\dot{V}O_2$ is based on an open flow-through-system with a mass-spectrometer for measurement of gas concentrations.

The study was made with the infant nursed in an incubator with a controlled environment as to temperature, ambient humidity and air velocity. The infants were calm during the measurements and body temperature and respiratory rate were stable.

Results: RWL was 4.40 ± 0.66 (SD) and $\dot{V}O_2$ was 5.91 ± 0.90 (SD) before phototherapy. During one hour of phototherapy the corresponding values were 4.34 ± 0.75 (SD) and 5.74 ± 1.05 (SD).

Conclusion: No significant change in respiratory water loss or oxygen consumption is seen in thermally stable infants during phototherapy.

Treatment of Neonatal Sepsis - A Multicentre, International Study.

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1316 neonates with clinical signs of sepsis were randomised to receive ceftazidime (CAZ) or gentamicin plus ampicillin (GENT + AMP) in standard doses between Feb '88 and Nov '89. Those centres with a high incidence of Listeria or enterococcal infection added AMP to the CAZ arm. Some centres substituted tobramycin or amikacin for GENT dependant on resistance patterns. 176 (13.4%) had bacteriological proven infection and 489 (37.2%) had strong clinical evidence including an abnormal WBC band count ≥ 2 or CRP ≥ 20 mg/L. The remaining 651 (49.4%) neonates with weak evidence of infection were analysed for safety. Comparisons of the demographic and baseline data were similar. Cure rates for the bacteriological and clinical populations were 119/146 (82%) for CAZ, 169/184 (92%) for CAZ + AMP and 278/335 (83%) for GENT + AMP (or alternative aminoglycosides). The major pathogens isolated from CSF and blood were Group B streptococci, *S. epidermidis* and *S. aureus* and those from urine were *E. coli*. Drug related adverse events were low for both treatment regimens with only one patient (GENT) requiring to be withdrawn. 8 patients receiving CAZ or CAZ + AMP and 7 patients receiving GENT + AMP died of their infection (23 and 27 neonates respectively died due to their underlying disease). Neonatal sepsis can be efficaciously treated with CAZ, a safe and alternative to GENT + AMP.

HUMAN INTRAVENOUS GAMMAGLOBULIN (HIVIG) REDUCES THE IN VITRO CYTOTOXIC ACTIVITY OF KAWASAKI SYNDROME (KS) SERA ON INTERLEUKIN (IL-1 α) TREATED CULTURED HUMAN UMBILICAL VEIN ENDOTHELIUM (HUVE)

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Sera from patients with acute KS cause complement-mediated lysis of IL-1 α stimulated HUVE (D.Y.M. Leung et al. I. Exp. Med. 164. 1958-72, 1986). In vitro cultured HUVE monolayers were labelled with ⁵¹Cr and treated with hu rIL-1 α (10 U/ml) for 4 hours. Addition of KS sera (1:2.5) to the culture caused a 28.4 \pm 7.3% (n=7) ⁵¹Cr release in the presence of complement. The addition of HIVIG (Gammalimmune 0.5%) to the culture reduced the HUVE lysis to 12.3 \pm 5.2%. Sera from patients in the convalescent phase of KS (n=4) had no cytotoxic effect. We conclude that cytotoxic antibodies in acute KS sera directed to IL-1 α inducible endothelial cell antigens may compete for receptor sites with antibodies present in HIVIG preparations that fail to fix complement. This competition may result in the decrease of the cytotoxicity by KS sera and in consequence in the beneficial effect of HIVIG in KS.

RESTING METABOLIC RATE IN OBESE CHILDREN AND ADOLESCENTS DURING DRASTIC WEIGHT LOSS AND DURING 8 MONTHS FOLLOW UP.

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Obese individuals often return to their obese state after weight loss. Changes in metabolic rate may develop during caloric restriction and may contribute to the inability to maintain weight loss. To assess the effect of drastic and longterm weight loss on resting metabolic rate (RMR) in obese children and adolescents the RMR of 18 obese individuals, aged 10.2 to 13.1 years (mean \pm SD:11.8 \pm 0.8 yrs) were measured by indirect calorimetry (SensorMedics 2900) before weight loss, during a three weeks drastic weight reduction (700 kcal mixed diet) and bimonthly during 8 months follow up. Body composition was assessed by body impedance method (BIA 109, AKERN/RJL).

Results	kg body weight	BMI	LBM (kg)	RMR (kcal/d)	kcal/kg LBM
baseline	82.3 \pm 16.2	28.8 \pm 2.2	44.6 \pm 3.4	2009 \pm 182	45.2 \pm 4.0
3 weeks	75.0 \pm 14.3	26.3 \pm 1.9	40.1 \pm 2.6	1466 \pm 162	35.8 \pm 3.4
2 months	76.2 \pm 17.2	26.7 \pm 2.3	42.3 \pm 2.3	1627 \pm 293	36.8 \pm 4.4
8 months	76.4 \pm 16.7	27.3 \pm 2.5	42.9 \pm 3.0	1887 \pm 356	44.3 \pm 4.4

After the three weeks drastic weight reduction RMR significantly decreased for 27% (p<0.01). During the 8 months follow up RMR and kcal/kgLBM increased concomitantly with the weight gain. Individuals who maintained their weight, however, increased their RMR parallel with increasing LBM. Those, who rapidly gained weight because of increased caloric consumption showed baseline RMR after 2 months.

HYPOXANTHINE (Hx) CONCENTRATIONS IN VITREOUS HUMOR (VH), CEREBROSPINAL FLUID (CSF), AND PLASMA, TOGETHER WITH URINARY EXCRETION IN TWO HYPOXEMIC GROUPS OF YOUNG PIGS

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We investigated how hypoxemia before death influences Hx levels in the VH post mortem, and the relationship between Hx accumulation in the VH and levels found in other extra-cellular fluids. Hx was measured with an HPLC method, in 2 groups of hypoxicemic young pigs. Group 1: $FiO_2=0.08$, ($PaO_2:2.3-3.0$ kPa) and group 2: $FiO_2=0.11$, ($PaO_2:3.0-4.0$ kPa). The hypoxemia lasted until death; which occurred in group 1 after 175 ± 52 min. and group 2 after 283 ± 118 min. Mean \pm SD are given. *: $p<0.02$ compared to values before hypoxemia. #: $p<0.02$ compared to values in other group.

Group	Before hypoxemia		120 min hypoxemia		Death	
	1(n=7)	2(n=7)	1	2	1	2
VH (umol/l)	12 \pm 6	8 \pm 2	26 \pm 5*	25 \pm 18	16 \pm 2	20 \pm 9*
CSF (umol/l)	18 \pm 4	15 \pm 3	61 \pm 24**	31 \pm 12	103 \pm 52**	27 \pm 23
Plasma (umol/l)	25 \pm 4	20 \pm 3	43 \pm 30*	36 \pm 22*	30 \pm 34	15 \pm 9
Urin (nmol/kg/min)	21 \pm 15	13 \pm 8	43 \pm 30*	36 \pm 22*	30 \pm 34	15 \pm 9

In both groups, CSF Hx increased until death, while in 4 pigs plasma Hx reached a peak followed by a fall towards death.

In conclusion: CSF Hx reflects hypoxemia, partly independent on degree and duration. Plasma Hx reflects the degree better than the duration of hypoxemia. Post mortem VH Hx may however, reflect the duration of hypoxemia before death better than the degree.

METABOLIC RESPONSE TO EARLY INTRODUCTION OF IV LIPID (IVL) IN SICK VLBW INFANTS.

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IVL permits extra calorie intake in VLBW infants. AIMS: a) To test whether provision of extra oxidative fuel improves glucose(G) homeostasis, b) to evaluate tolerance of early IVL. METHOD: NICU infants <1500 g received isocaloric protein + G regime from d1 either with IVL 1g/kg from d1 to 3g/kg from d3 (Gp1, n=16) or IVL added only from d8 (Gp2, n=13). Blood glucose, gluconeogenic precursors (lactate, pyruvate, alanine), serum NEFA and insulin were determined daily. RESULTS: No adverse effects of IVL were seen in either group. Weight loss was less in Gp1 at 7d. There was no difference in mean blood G or in frequency of hyperglycaemic or hypoglycaemic episodes. Mean FFA and triglyceride levels were similar in both groups even in SGA infants, suggesting that lipid clearance was not impaired. No difference in gluconeogenic precursors or inverse relationship between glucose and FFA levels observed (as expected if the G-FA cycle was operating to reduce G utilisation). COMMENT: In these sick infants hormonal effects on metabolism due to stress and exogenous administration of metabolic fuels may have obscured the expected G-fat relationship. VLBW infants can tolerate and utilise lipid from the first day of life.