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PERINATAL BRAIN DAMAGE: POOR PREDICTIVE VALUE OF METABOLIC ACIDOSIS AND APGAR SCORE  
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Asphyxia is usually defined by Apgar score, but brain damage is poorly predicted by even the 5 min score (A5). To assess the prognostic value of umbilical arterial pH, base excess, and lactate, we correlated them with A5 and adverse outcome (10 died, 60 had cerebral palsy or developmental delay at 1 yr) in 982 consecutive deliveries. Normal range ( $\pm$  2SD) was derived from 106 cases with no maternal, pregnancy, delivery, neonatal, or developmental problems. Low pH ( $<$ 7.16) was found in 95 cases (10%), 9 of them had adverse outcome. Low A5 ( $<$ 8) was found in 29 cases (3%), 9 had adverse outcome. Combination of low pH with low A5 was seen in only 9 cases, 1 of them had adverse outcome. Of the 15 infants with severe damage, 2 had low A5, 2 had low pH, and 1 had both. Base excess or lactate, singly or in combination, did not improve the predictive accuracy of A5 or pH. We conclude that although the specificity of A5 in relation to brain damage is high (98%), its low sensitivity (13.4%) or predictive value (34.6%) is not improved by measurement of pH or related variables in umbilical arterial blood at birth.

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PHYSIOLOGICAL AND BEHAVIOURAL EFFECTS OF NONNUTRITIVE SUCKING ON NEONATES. R. Paludetto, A. Stagni, C. De Fusco, M.G. Corbo. Dept. of Peds., Univer. of Naples, Italy.

This study was performed to evaluate possible immediate physiological and behavioural changes during non-nutritive sucking bursts (NNSBs) in neonates (n.). 26 n. in room air and without severe perinatal complications (Minde  $s <$  4) were included in the study (G.A.wk  $34 \pm 3$ ; B.W.  $2030 \pm 590$ ; Apgar s. 5m.  $8.6 \pm 1$ ). Behavioural states (B.S.), transcutaneous oxygen tension (TcPO<sub>2</sub>), heart rate (H.R.), respiratory rate (f.) and NNSBs were monitored during 3m. of nonnutritive sucking opportunity offered to n. by a pacifier. Data were sampled at 10' intervals. N. sucked  $58 \pm 35$  per cent of the time available for sucking. Variables were compared between periods without and with NNSBs (p.t. Tests). B.S. went from  $2.7 \pm 0.8$  to  $2.5 \pm 0.5$  (n.s.), TcPO<sub>2</sub> from  $71 \pm 12$  to  $70 \pm 10$  (n.s.), H.R. from  $139 \pm 3$  to  $138 \pm 12$  (n.s.). Only f. increased from  $42 \pm 11$  to  $46 \pm 13$  (p < .05). Our data document an immediate interaction between NNSBs and respiratory rate suggesting an effect on the central respiratory control mechanism. Moreover, they indicate that the decrease in respiratory rate reported in a similar group of neonates during feeding should not be interpreted as an effect of sucking per se.

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AGE-DEPENDENT MATURATION OF  $\beta$ -ADRENOCEPTORS ON B- BUT NOT ON T-CELLS

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Therapeutic use of  $\beta$ -adrenoceptor ( $\beta$ -AR) agonists in infants is often ineffective. Since  $\beta$ -AR on lymphocytes (Ly) are low in infants, it has been suggested that this might be due to a relative deficiency of  $\beta$ -AR. However, low numbers of  $\beta$ -AR on different Ly subpopulations might mimic a  $\beta$ -AR defect in infants. Therefore we studied  $\beta$ -AR binding on B- and T-Ly enriched fractions (separated by F-rossetting, called B- and T-Ly) in infants (aged 4 to 18 months) and children (aged 7 to 15 years) using 125 iodocyanopindolol (ICYP). Receptor (R) density and affinity were calculated by Scatchard plots. Resolution of  $\beta$ -AR into those with high and low affinity state was obtained from inhibition curves with salbutamol using the Hofstee plot.  $\beta$ -AR density on B-cells of infants was nearly the same as on T-cells, whereas in children it was 2 to 3 fold higher on B- than on T-cells. Affinity of the  $\beta$ -AR to the antagonist ICYP was much lower on T-cells than on B-cells of infants, whereas no difference was observed among the children. However, using salbutamol displacement curves it appeared that  $\beta$ -AR on T-cells were at a higher affinity state compared to those on B-cells. No differences were observed between infants and children.

In conclusion,  $\beta$ -AR deficiency postulated in infants may be a result of low  $\beta$ -AR density on B-cells, indicating a maturation process on B-cells during the first year of life.

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POSTNATAL CHANGES OF AVP IN PREMATURE INFANTS WITH RDS.  
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The mechanism responsible for the spontaneous diuresis that occurs prior to the recovery from RDS is unknown. We studied the possibility for AVP-mediated diuresis in 10 consecutive premature infants requiring ventilation for RDS (Mean  $\pm$  DS, BW  $1885 \pm 827$ ; GA  $32 \pm 2$  wk; Apgar scores  $5 \pm 1$  and  $6 \pm 2$  at 1' and 5'). No alteration was made in either the administration of fluid or medications ordered by the infant's physician. During the first five days of life, the changes of AVP were serially determined by RIA method (Buhlman Lab., Basel) and related to the variations in creatinine clearance (C cr), osmolality, output/intake (O/I), body weight and pulmonary function.

DAY	AVP (pg/ml)	O/I (%)	FiO <sub>2</sub> /PaO <sub>2</sub>	OSMOLALITY (mOsm/l)		WEIGHT Wt(%)	C cr (ml/min)
				PLASMA	URINE		
1	$18 \pm 26$	78	1.24	$282 \pm 5$	178+63	-8.8	$12.0 \pm 8$
3	$11 \pm 8$	80	0.70	$285 \pm 4$	200+53	-9.0	$18.0 \pm 13$
5	$11 \pm 13$	70	0.65	$284 \pm 14$	184+93	-9.0	$19.4 \pm 10$

Even if our study concerns infants with different RDS severity, a temporal relationship was found between diuresis, loss of weight and the improvement of pulmonary functions, without significant change in creatinine clearance. Throughout all three study periods, osmolality remained unchanged and the urine produced was hypotonic compared with plasma. A decrease in the high AVP concentration is an unlikely explanation for the excretion of a dilute urine described in the present study.

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MECHANICAL LOAD OF BIRTH AND INTRAVENTRICULAR HEMORRHAGE. von Loewenich, V., Halberstadt, E. Dept. Neonatology and Dept. Obstetrics University-Hospital, Frankfurt a.M., F.R.G.

We made the clinical observation that VLBWI's covered by hematomas after birth ("bruised babies") had a relative high incidence of Intraventricular Hemorrhage (IVH). To quantify this observation one of us (E.H., obstetrician) classified in 132 VLBWI's (Gestational Age below 211 days, mean GA 28 w + 1 d, mean birth weight 1,069 gr) the birth mode into mechanically difficult or easy, without knowing the outcome. The two groups of infants were compared in respect to frequency of IVH I° to IV°. The overall-rate of IVH was 23%. IVH was found in 68% (19 out of 28) after difficult and in 12% (12 from 104) after easy delivery (p below  $10^{-6}$ ). After vaginal delivery the ratio was 100% vs. 4% (8 from 8 vs. 1 from 24; p below  $10^{-6}$ ), after cesarean section 55% vs. 14% (11 from 20 vs. 11 from 80; p below 0.000,1). There were no differences in GA between infants with difficult or easy delivery (27 w + 6 d vs. 28 w + 2 d) nor between infants with or without IVH (28 w + 0 d vs. 28 w + 1 d).

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DIAGNOSIS, TREATMENT AND FOLLOW-UP OF TRANSIENT MYOCARDIAL ISCHEMIA OF THE NEWBORN

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52 newborns with Transient Myocardial Ischemia (TMI) were admitted to our Department between August 1979 and August 1986, (1,5% of all admissions). Gestational age ranged from 29 to 41 (mean  $38,9 \pm 3,22$ ) weeks, birth weight from 1050 to 4400 (mean  $2920 \pm 850$  g); there was a history of complicated pregnancy in 23 (57,5%) patients and of birth asphyxia in 23 (57,5%). Symptoms started within the first 48 of life in 35/52 varying from severe cardiogenic shock to heart murmur usually due to mitral or tricuspid insufficiency, and/or electrocardiographic Q or ST/T abnormalities. Metabolic acidosis in 14/32, hypoxia in 16/52, were observed. 3 patients died: only 1 because of myocardial ischemia. 14 newborns required assisted ventilation, 6 oxygen therapy, 15 digitalis and/or diuretics, 8 atropin and 9 isoproterenol. Symptoms subsided within 2 to 43 (mean 14) days in 39 subjects. The ECG picture reverted to normal in 6-108 (mean 45) days in 38 cases. 25 newborns were followed up for 2-19 (mean 7,4) months. In 20 the clinical signs and the ECG became normal; in one the ECG was normal after 5 months but showed again signs of myocardial necrosis 4 months later, one patient with normalized ECG had a syncopal episode and bradycardia at 4 months; 3 patients had normal ECG with murmurs (1 IVD, 2 PDA). 1 patient with myelomeningocele died at 12 months of age because of pulmonary infection. In our experience TMI is associated with birth asphyxia in only 44,3% of cases, and with hypoxia in 50% of cases. It is probably underestimated. In the majority of cases the prognosis is favourable. A long long-term follow-up in order to detect possible sequelae related to the neonatal myocardial damage is mandatory.