91 RENAL ACIDIFICATION AND PROGNOSIS OF VENTILATED NEONATES.

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Titratable acidity /TA/ and ammonium /NH₄/ of 67 neonates requiring IPPV or CPAP ventilation were daily determined from continuously collected urine for 92 hours. Irrespective of birth weight and maturity, an increase in TA and NH₄ excretion by the 3rd postnatal day was found in 57 infants. All these babies survived after 5 to 21 days of ventilation therapy. Eleven infants died on the 5th to 12th postnatal day. In these cases the urinary TA and NH₄ excretion gradually decreased, and the mean values on the 3rd and 4th day were significantly lower than those of the survivors.

It is concluded that an early decline of TA and NH₄ excretion predicts a rather poor prognosis of the ventilated neonate.

92 DIAGNOSTIC AUDIT OF PLAIN CHEST X-RAYS IN THE DIAGNOSIS OF SYMPTOMATIC PDA IN NEWBORN INFANTS

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The aim of this study was to assess the value of plain chest x-rays in the diagnosis of symptomatic patent ductus arteriosus (s.PDA). This was done for the first time in an unselected population of preterm and term newborn infants who had been ventilated for at least ten days. The study was done retrospectively in 111 patients. 65 infants had a clinically established diagnosis of s.PDA which was combined with RDS in 48 cases. Of 46 infants without s.PDA 23 had RDS. Method: Chest films taken during the first ten days of life were analyzed if the following radiological signs were present: 1) Perihilar lung edema & pulmonary vascular congestion 2) More intensive lung edema hiding vascular structures 3) Radiolucency decreasing on the 4/5th day in initially normal lungs 4) No improvement of radiolucency in initially underaerated lungs on the 4/5th day. Radiological and clinical diagnoses were compared. Results: 1. Radiological sign 1 or 2 combined with sign 3 or 4 was found in 36 of the infants with clinically established s.PDA (sensitivity = 85%). The same signs were also found in 16 infants without s.PDA (specificity = 65%). These radiological signs had a positive predictive value of 78%. The negative predictive value was 80%. 11. In 26 of 55 infants with the clinical and radiological diagnosis of s.PDA radiological signs were found on average 1 day earlier than the clinical symptoms. Conclusion: In an unselected population of ventilated newborn infants the studied radiological signs had a low specificity and moderate sensitivity. In the early diagnosis of s.PDA, however, the radiological method was more sensitive than clinical examination in about half the cases.

93 HEAD NARROWING IN PRETERM INFANTS.

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The marked narrowing of the head which develops in many preterm infants often causes concern to their parents, who may worry that the unusual shape will persist or that it may be associated with intracranial pathology and developmental delay. The biparietal (BP) and anteroposterior (AP) diameters were measured weekly in 203 preterm infants during their stay in NICU, and at regular intervals up to three years of age. The AP/BP ratio was calculated (Baum J D and Searls D, Dev Med and Child Neurol. 1971 13: 576-581). This ratio rose from a mean of 1.36 in the first week to a mean of 1.48 at six weeks and fell to a mean of 1.42 by 13 weeks. Less mature babies and those with lower birth weights showed more flattening but this was not statistically significant. The presence of intracranial pathology detected by ultrasound did not affect head shape. 11% of babies showed marked early flattening (AP/BP ratio 1.55). These were compared with the rest of the group. At three years there was no significant difference in AP/BP ratio or in Griffiths Development Quotient. We conclude that by the age of 3 years the babies showing very marked early head flattening did not differ from the rest of the cohort in the areas examined.

(NICU = Neonatal Intensive Care Unit)

94

THE GROWTH OF LOW BIRTH WEIGHT 3 YEAR OLDS

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177 children (81% of a cohort of 218 weighing 2000g or less at birth) were measured regularly until the age of 3 years. At 3 years the weights of 36% (53% SGA and 26% AGA) were <10th centile, and of 11% (5% with handicap) <3rd centile. The heights of 23% (31% SGA, 18% AGA) were <10th centile. When height was corrected for prematurity only 11% were <10th centile, suggesting that many children's heights were close to this centile. If they continued on their 3 year old centile their adult height would be close to the tenth centile. Categorising them as "below the 10th centile" gives a misleading pessimistic impression. Comparison with the weight centile at 6 months showed that 33% crossed the 10th centile by 3 years. The predictive value of the weight at 6 months was better for boys than girls. The study confirmed the findings of previous workers and showed that predictions about growth cannot reliably be made at 6 months. Furthermore, many of those whose height was below the 10th centile at 3 years may well attain an acceptable adult height. SGA = Small for Gestational Age, AGA = Appropriate for Gestational Age.

95

AUDITORY BRAINSTEM RESPONSES IN JAUNDICED NEWBORNS

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This study was designed to determine whether Auditory Brainstem Responses (ABR) may reveal brainstem dysfunction caused by hyperbilirubinemia, whether exchange transfusion (ET) may have a beneficial effect on this dysfunction and if ABR is a reliable test for assessing the risk of bilirubin encephalopathy. In 18 full term jaundiced infants who needed an ET a total of 96 ABRs were evoked: 33 before and 33 after the ET; 14 ABRs (9 neonates) one week after the ET and 16 ABRs (9 neonates) at a mean age of 6 months. The pre-ET bilirubin values were not very high (23.2±2.6mg/dl). From the 33 pre-ET ABRs 23 (16 newborns) were abnormal: only 2 out of the 18 newborns had normal pre-ET ABRs bilaterally. From the 23 abnormal pre-ET ABRs 19 (13 newborns) became normal immediately after the ET; 3 ABRs (2 newborns) were normal one week after the ET and the remaining ABR (newborn) was still abnormal at the age of 6 months. At that age this baby was hypertonic. An abnormal ABR showed absence of waves V and I and an increased brainstem transmission time (5.10±0.33ms before vs 4.86±0.51ms after ET, p<0.005). Our data indicate that a) hyperbilirubinemia even at levels not very high may cause brainstem dysfunction which in most cases is reversible with ET b) ABR may detect this dysfunction and c) babies with an abnormal post-ET ABR are at high risk for developing bilirubin encephalopathy.

96

HYPEROXIA DOES NOT INCREASE BILIRUBIN DEPOSITION IN RAT BRAIN.

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The entry of bilirubin into brain is facilitated by displacing agents, and by factors affecting the permeability of the blood-brain barrier. It has been suggested that hyperoxia may increase bilirubin entry into brain. We have investigated the effects of short term (1 hr) and long term (27-30 hrs) hyperoxia on the entry of bilirubin into rat brain. Bilirubin 20 mg/kg/hr was infused for 3 hrs into awake, 5-6 weeks old male Sprague-Dawley rats. Prior to the infusion each rat was given 10-20 uCi ¹²⁵I-human serum albumin. In group 1 (n=8) the rats breathed room air at all times. In group 2 (n=8) the rats were exposed to 80% oxygen during the last 60 minutes of the bilirubin infusion. In group 3 (n=10) the rats were kept in 70-80% oxygen for 24-27 hrs prior to, as well as during the bilirubin infusion. After 3 hrs the rats were sacrificed and the brain perfused in situ with icecold saline 20 ml/min for 3 minutes. Brain albumin was estimated by counting the activity in one half brain and in a serum sample. Brain bilirubin was determined by chloroform extraction from the other half. Results:

Group	Brain albumin (ug/g)		Brain bilirubin (ug/g)	
	Mean	S.E.M.	Mean	S.E.M.
1	161	+/- 41.6	1.46	+/- 0.370
2	133	+/- 33.3	1.58	+/- 0.710
3	150	+/- 14.5	0.89	+/- 0.145

None of the differences are significant at the 5% level. We therefore conclude that hyperoxia does not increase bilirubin deposition in rat brain.