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SERUM BILIRUBIN: A POOR PREDICTOR OF KERNICTERUS
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Kernicterus was found in 20 infants autopsied between Jan '72 and June '77 at Kings County Hospital-Downstate Medical Center. Most of the infants were very premature; mean gestational age was 31.5(27-37)wks with mean birth weight of 1430(670-2910)g. Age at death ranged from 41 hrs. to 5 mos. with a median of 130 hrs. Peak serum bilirubin values ranged from 6.5 to 20.6 mg% (mean, 11.5mg%). Only 2 infants had values greater than 15mg%; both of these infants received exchange transfusion and 11 others had phototherapy.

Since the bilirubin levels were so low, we analyzed the clinical and laboratory features which may have contributed to the development of kernicterus. Hypoalbuminemia ($<2.5g\%$) was found in 6/9. In 4 of 5 infants in whom HABA dye binding studies were done, the values were very low ($<25\%$). Anemia was present in all infants with evidence of hemolysis in 9/14. Internal bleeding was found in all, including 15/20 cases of intracranial hemorrhage. Respiratory distress with acidosis and hypoxia was present in all cases. There was evidence of infection in 13/20 infants. A greater proportion of the larger ($>1950g$) infants had infection (5/6) than did the smaller infants (8/14). Although most of the infants did not exhibit all the classical signs of kernicterus clinically, they did have evidence of non-specific CNS involvement such as apnea, bradycardia and cardiorespiratory arrest.

On the basis of these findings, it is difficult to define a safe level of serum bilirubin below which kernicterus cannot occur. It would appear that in infants with extreme prematurity, infection, acidosis and hypoxia, we must either find a means of preventing even modest bilirubin accumulation or a new index to assess the state of bilirubin diffusibility.

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INTRACEREBRAL HEMORRHAGE IN HIGH RISK PREMATURES

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Subependymal (SEH) and intraventricular hemorrhage (IVH) in infants less than 35 weeks gestation, requiring intensive care for 24 hours or longer, were studied prospectively. Initial computerized tomographic scan (CT scan) was obtained, and, if positive for blood, head circumference, clinical course, and serial scans were followed until ventricular size was normal.

29/58 infants were shown to have SEH and/or IVH, 26 by CT scan, 1 by ventricular tap, 2 on autopsy. 8 infants died, 6 of these had marked IVH, 3 shown by CT scan, 1 by ventricular tap, 2 by autopsy. Acute hydrocephalus of only mild to moderate degree occurred in the 3 fatalities with positive scans. The 23 survivors with positive scans had follow-up scans. 18/23 did not show progressive hydrocephalus, and of these 4 had only SEH. Of the others, 10 had mild, 3 moderate, and 1 marked IVH. 5 survivors with IVH developed severe progressive hydrocephalus. 2/5 had only mild IVH, which resolved spontaneously. 3/5 required treatment. IVH was moderate in 1, marked in 2. Serial head circumference was not predictive of need for treatment.

The incidence of SEH and IVH was 50% in study infants and was not related to gestational age. The quantity of blood may be prognostically significant. No infant with SEH or mild IVH required treatment. Progressive hydrocephalus developed in 2 and resolved spontaneously. 7/10 infants with moderate to marked IVH survived. 3 required treatment.

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EFFECT OF POSITIONING AND HEAD BANDING ON INTRACRANIAL PRESSURE IN THE PREMATURE INFANT. Niki Kosmetatos and Margaret L. Williams. Dept. of Peds.,

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The intracranial pressure of 12 infants, birthweight 720-1800 gm., who had no evidence of intracranial hemorrhage, was measured over the anterior fontanelle by the Ladd ICP monitor. Pressures were determined before and one minute after the release of encircling phototherapy eye patches or nasal cannula apparatus. The measurements were performed twice on each infant in three different positions -- head up 30°, horizontal, and head down 30°. A significant decrease in pressure was noted immediately after release of the encircling band in all three positions in most infants. The average decrease was 3.75 (range 0-14.5), 3.73 (range 0-10), and 3.45 (range 0-9) cm H₂O in the head up, horizontal, and head down position respectively. Three of the four infants who demonstrated the smallest decrease in intracranial pressure had been exposed to constricting apparatus for long periods of time prior to their determinations suggesting possible compensation. The magnitude of the decrease in pressure was not different in the various positions studied, although the pressures tended to increase as the infant was placed in the head down position. The changes noted in intracranial pressure both with positioning and banding raise questions as to the effect of such infant care practices on the brain of infants with compressible skulls. Increases in intracranial pressure induced by head banding may play a role in the high incidence of intracranial hemorrhage in the very small infant.

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QUANTITATIVE NON-INVASIVE METHOD OF MEASURING CEREBRAL

BLOOD FLOW (CBF) IN THE NEWBORN. F. Leahy, K. Sankaran

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We devised a quantitative method to estimate CBF based on the principle of jugular occlusion. With gentle and short compression of both jugular veins there is an increase in head circumference (OFC) which reflects CBF. To make measurements quantitative we differentiated regression of OFC on skull volume: $Y = 59.1(X) - 1,608$, where Y = skull volume and X = OFC. The formula obtained was $CBF = 355/Y \cdot dx/dt$ in ml/100g/min. Method entails measurement of (1) OFC, using a mercury strain gauge. Change in OFC is measured as a slope when jugular veins are compressed. (2) Skull volume, using polystyrene cast of the infant's skull filled with water, and volume measured. Comparing values to autopsy brain weights it is accurate within $\pm 4\%$. (3) Monitoring intracranial venous and arterial flow by Dopplers taped over superior sagittal sinus and nearby artery. When veins are completely occluded flow stops. Alveolar CO_2 (P_{ACO_2}) is simultaneously recorded. When values from complete occlusions and no interference with arterial flow are taken, results are reproducible. In 8 preterm infants (G.A. 34wk ± 2 S.D.; B.W. 1.8kg ± 6 S.D.; age 9.4 days ± 7.9 S.D.) the mean CBF was 67.1 ± 13.6 S.E. ml/100g/min. The values are similar to those reported previously using the N_2O method. During administration of 2% to 3% CO_2 , CBF increased to 97 ± 24.1 S.E. Mean P_{ACO_2} increased from 38.7 ± 1.2 to 43.4 ± 1.1 S.E., therefore the increase in CBF per mmHg P_{ACO_2} was 6.9 ml/100g/min. The 10% increase in CBF is double that reported for adults. We suggest that CBF in preterm infants is more sensitive to changes in P_{ACO_2} than adults.

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SERIAL MATURATION OF AUDITORY BRAINSTEM EVOKED POTENTIALS IN PRE-TERM INFANTS. Allan Krumholz, Philip J. Goldstein, Jacob K. Felix, Robert F. Carr, and Dorothy

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The auditory brainstem evoked response to clicks matures to adult values within the first two years of life. To evaluate this procedure as a measure of nervous system maturation in premature infants, serial testing was done in 20 infants of 35 weeks or less gestational age. The children were tested at weekly intervals. Gestational age was determined by Dubowitz's criteria. Thirty normal fullterm infants served as controls. All subjects were tested with clicks of 80 dB SL at a stimulus rate of 9-12/sec and averaged by computer. Responses were obtainable in all infants over 30 weeks. The earliest waves noted were I, III, and V. Waves II, IV, and VI were unstable in early recordings. However, waves II, IV, and VI were present in over 50% of patients as they approached 40 weeks. The latency of wave V gradually decreased at a rate of about .2msec. per week to fullterm. However, waves I and III approached relatively mature latencies earlier, at 34 to 37 weeks. The standard deviation for the latency of wave V was large. This value, contrary to previous reports, is a relatively insensitive measure of gestational age and may be influenced by perinatal factors. The appearance of the auditory brainstem evoked response at 30 weeks and the rapid serial decrease in its latency from 30 to 42 weeks gestational age suggest that this procedure can be useful in monitoring the maturity and serial development of the nervous system in the premature infant.

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QUALITY OF LIFE AFTER PROLONGED NON-TRAUMATIC COMA IN

CHILDREN: A REAPPRAISAL. Lewis A. Margolis, Bennett A. Shaywitz, Dept. Ped, Yale U. Sch. of Med. New Haven.

Recent advances in medical technology have resulted in a reduced mortality of critically ill children, but quality of life in these survivors remains ill-defined. Such information is of particular importance in an era when physicians are frequently called upon to decide whether and when to discontinue extraordinary measures of life support. We report the outcome (follow-up 1-7 yrs) in 16 children ages 6 mos-16 yrs in coma of non-traumatic origin for longer than 5 days. The 8 girls and 8 boys remained in coma for periods of 6-180 days (median 17.5 days) and 12 children remained in deep coma 1-35 days (median 10.9 days). Major neurological residua (seizures, developmental retardation, blindness) occurred in 4, minor residua (MBD, minimal motor deficits, personality disorder) in 7, and no deficits were observed in 5 children. After recovery 6 were average or superior in intelligence and 2 additional children exhibited no decrement in I.Q. The likelihood of sequelae appeared related to etiology and occurred in all cases of anoxia (5/5), frequently after encephalitis (5/8) but in none of the children with Reye Syndrome (0/3). Duration of deep coma as well as the presence of increased intracranial pressure for more than 2 days appeared to influence outcome. However seizures, the necessity of assisted ventilation, or the duration of light coma was not of any predictive value. Our results suggest that a significant proportion of children surviving prolonged coma of non-traumatic origin are able to attain a meaningful quality of life.