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COMPARISON OF INCANDESCENT AND FLUORESCENT LIGHT SOURCES IN PHOTOTHERAPY. T.R.C. Sisson, M. Ruiz, K-T Wu, O. S. Afuape (Spon. by A. DiGeorge), Temple University School of Medicine, Department of Pediatrics, Philadelphia, Penna.

The purpose of this study was to determine the efficiency of an incandescent light source of high intensity in phototherapy of neonatal jaundice in comparison with a standard fluorescent source. Forty well infants with physiologic jaundice (1810-3320 gm.) were assigned at random to two groups of 20 each: Gp. A under a quartz-halide tungsten filament lamp (6.5 $\mu\text{W}/\text{cm}^2/\text{nm}$) 80 cm. above skin surface, next to a radiant warmer; Gp. B under a canopy of 8 standard blue (F20T12/B) lamps (4.1 $\mu\text{W}/\text{cm}^2/\text{nm}$) 45 cm. above skin surface. Phototherapy was begun when serum bilirubin concentration exceeded 10.0 mg/dl in first 72 hr of life. In Gp. A, phototherapy was stopped when conc. <80 mg/dl. In Gp. B, this was done in two-thirds of infants, but stopped $\bar{p}$ 36 hr. in one-third because of failure of phototherapy. In Gp. A mean hr. Rx = 41.5, mean bili. drop 1.92 mg/dl/24 hr.*

We conclude that the quartz-halide lamp placed high above the infant is more effective in the clinical use of phototherapy than the standard blue fluorescent light source. It has spatial advantage and lack of light scatter. Body temperatures in Gp. A were not increased by use of the quartz-halide lamp.

*In Gp. B mean hr. Rx = 46.7, mean bili. drop 0.48 mg/dl/24 hr.

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NEONATAL NECROTIZING ENTEROCOLITIS (NEC): 100 NEW CASES David K. Stevenson, C. Benjamin Graham, and John K. Stevenson (Spons. by J. Johnson) U. of Wash. School of

Medicine, University and Children's Orthopedic Hospitals, Seattle 100 infants between Aug., 1969, and Oct., 1976, with radiologic, surgical, and/or pathologic diagnosis of NEC since this institution's last report of 43 cases were analyzed, representing an incidence of 2-3% of all intensive care nursery admissions. Strict X-ray criteria were used to confirm the diagnosis: pneumatosis intestinalis (92 cases), hepatic portal venous gas (HPVG, 31), and pneumoperitoneum (12). 20% of cases were diagnosed after 2 weeks, the latest at 51 days. 30 infants with highly suggestive clinical signs for 24 or more hours had initial X-rays which were nondiagnostic. 4 intestinal perforations were not manifested by free gas on X-ray. The diagnosis was not made antemortem in 9. The overall mortality was 46% compared to 37% in the last series. 67 infants were managed medically, and 33 underwent surgery with mortalities of 37% and 64%, respectively. 18 who had operations were <1400gms. None succumbed during surgery, and 9 were judged at the time of operation to have such extensive disease as to not warrant resection and/or diversion procedure. Failure of medical management (surgery and/or death) occurred in 58. Mortality was increased with low birth weight, low gestational age, HPVG, and surgery. Of 11 infants ≥ 2500 gms, the mortality was 27%. Infants with confirmed NEC continue to have a high mortality, suggesting that advanced disease is already present when these strict criteria are met. Methods for early, reliable identification of infants at risk to develop NEC are needed to reduce the incidence, morbidity, and mortality of this condition.

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MASSIVE HEMORRHAGE IN THE NEWBORN LAMB

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We studied responses of newborn lambs to acute hemorrhage. Seventeen lambs, aged 4-70 days, were bled either 45-52% or 58-65% of their measured blood volumes. We measured red cell volumes with cesium chloride labeling and fluorescent excitation analysis. We assumed an Fcell ratio of .85 to calculate blood volume. We used only local anesthesia and bled at a rate of 1.4-3.0% blood volume per minute.

In the group hemorrhaged 45-52%, hematocrit, total serum solids, heart rate, and blood pressure fell significantly by the end of the hemorrhage. Respiratory rate and paO_2 increased, while paCO_2 decreased. pH remained unchanged. Nine of 12 animals survived more than 24 hours post hemorrhage with no IV replacement. In contrast, in the lambs bled 58-65%, heart rate increased significantly, while other variables showed changes similar to the other group. In this group, however, only 1 animal survived, while 4 died within 90 minutes after the end of the hemorrhage ($P < .01$).

We conclude that newborn lambs can tolerate approximately 50% acute loss of blood volume. Further decrease in blood volume significantly increases mortality.

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INCREASED PLATELET PROSTAGLANDIN FORMATION IN INFANTS OF DIABETIC MOTHERS, Marie J. Stuart, Haim Elrad, David O. Hakanson, Janet E. Graeber, and Mary K.

Barvinchak, Depts. of Pediatrics and Perinatology, SUNY, Syracuse, N. Y.

Infants of diabetic mothers have an increased incidence of thrombosis. In an attempt to evaluate the role of the platelet in the etiology of this thrombotic tendency, platelet rich plasma (PRP) from 15 control maternal-neonatal pairs was compared to PRP from 6 pairs in whom maternal diabetes mellitus was present. Platelet Malonyldialdehyde (MDA) formation (n moles per 10^6 platelets) in the presence of N-ethyl maleimide (NEM) or thrombin was measured as an indicator of platelet prostaglandin synthesis. Control maternal MDA values in the presence of NEM or thrombin were 3.23 ± 0.31 (1SD) and 1.30 ± 0.17 respectively, with control cord blood values being 2.46 ± 0.61 and 0.90 ± 0.19 . There was a significant increase in platelet MDA to 3.74 ± 0.49 (NEM) and 1.51 ± 0.35 (thrombin) in the diabetic mothers. This finding was associated with platelet hyperaggregability in 3/6. Platelet MDA formation was also significantly increased in the infants of these diabetic mothers to 3.66 ± 0.53 (NEM). Platelet hyperaggregability was present in 3/6 infants and was absent in all 15 control neonates. Platelet sizing did not differ significantly between the control or patient groups. Platelet prostaglandin synthesis appears increased both in the diabetic mother and her infant. The observed platelet hyperaggregability and increased Prostaglandin synthesis may contribute to the thrombotic tendency in the infant of the diabetic mother.

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EFFECT OF INCREASED Ca, P INTAKE ON BONE MINERALIZATION IN PRETERM AGA INFANTS. Jean J. Steichen, Tari L. Gratton, Julie A. Russell, Stephen D. Minton, Reginald C.

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The effect of Ca supplementation in prematures on bone mineral content (BMC) has not been quantitated in vivo. BMC (radius and ulna) was measured by photon absorptiometry in 42 term and 39 preterm AGA infants. Preterm infants were divided into 2 groups: "A" (n=30) 29-36 wks. gestation, fed Similac 20 cal/oz; and "B" (n=9) 28-36 wks. fed a modified Similac 20 cal/oz (126 mg Ca and 63 mg P/dl). Ionized Ca (iCa) total Ca, 25 hydroxy vitamin D, parathyroid hormone, Mg and P were measured every 2-4 wks. for 12 wks. In term and preterms, BMC at birth correlated with gestation ($r = .821$, $p < .001$). The regression line of BMC vs. gestation in these infants represents "normal" "intrauterine" bone mineralization (IUBMC). In "A", postnatal BMC was less than IUBMC (covariance $F = 12.5$, $p < .001$). In "B", BMC increased significantly with postnatal age ($r = .627$, $p < .05$) and did not differ from IUBMC ($p > .05$); at 37-38 wks. postconceptual age BMC was $.072 \text{ gm}/\text{cm}^2 \pm .002$ SEM, similar to BMC in infants born at 37-38 wks. In "B", daily Ca intake was $157 \pm 22 \text{ mg}/\text{kg}/\text{day}$ in the first wk. and 250 ± 7 in the 2nd and subsequent wks. Serum total Ca levels were $9.8 \pm .3 \text{ mg}/\text{dl}$; iCa levels were $4.4 \pm .1 \text{ mg}/\text{dl}$. No clinical complications were observed in "B". We conclude that 1) in premature infants on routine formula, extrauterine bone mineralization lags significantly behind intrauterine bone mineralization and 2) increased Ca and P intakes might result in bone mineralization rates similar to intrauterine mineralization rates.

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RISK OF RESPIRATORY DISTRESS SYNDROME AND MATERNAL INFECTION AFTER PRENATAL DEXAMETHASONE TREATMENT. H.W. Taeusch, Jr., F. Frigoletto, J. Kitzmiller, M.E.

Avery, A. Hehre, B. Fromm, E. Lawson, R.K. Neff, Harvard Medical School, Boston, MA.

A prospective randomized double blind trial was carried out to test whether prenatal glucocorticoids could reduce risk of RDS in prematurely born infants. Mothers (< 33 weeks gestation) with premature labor or with premature rupture of membranes, were treated with dexamethasone- PO_4 (4mg IM Q8H x 6) or saline (1ml IM Q8H x 6). Risk factors for RDS or infection were not different in steroid and placebo groups. Prophylactic antibiotics were not used.

	DEX	PLACEBO	P
Risk of RDS	2/30	14/69	< 0.05*
(Dex 6 doses vs. placebo)	(7%)	(20%)	
Risk of severe RDS	1/30	11/69	< 0.05*
(Dex 6 vs. placebo)	(3%)	(16%)	
Maternal Infection	14/52	8/66	< 0.05
(Dex vs. placebo)	(27%)	(12%)	

* one-tailed

In this study the duration of rupture of membranes and the duration of the interval from entry into study to delivery did not affect risk of RDS. We conclude that glucocorticoids 1) are effective in lowering incidence and severity of RDS, and 2) may be associated with increased risk of infection (usually endometritis).