VITAMIN E PROPHYLAXIS IN NEONATAL PHYSIOLOGIC AND 1493 GGPD DEFICIENCY HYPERBILIRUBINEMIA. Arthur I. Eidelman, Matti Ehrlichman, Joseph D. Schulman, and Larry Corash. Department of Neonatology, Shaare Zedek Medical Center and Sect. Human Bioch. and Develop. Genetics, NICHD and

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To study the efficacy of parenteral vitamin E in minimizing neonatal hyperbilirubinemia in both G6PD deficient and normal term infants, a prospective controlled study was performed. Parenteral vitamin E, 20 units/Kg/day for 4 days, was administered from birth. No significant difference in gestational age, nor birth weight between Group A (G6PD-Rx), Group B (G6PD-no Rx), Group C (Normal-Rx) nor Group D (Normal-No Rx) were noted. Mean peak bilirubin was:

	n	Bilirubi		
Group A	$(2\overline{1})$	9.76		
Group B	(20)	9.82		
Group C	(26)	10.75		
Group D	(29)	10.58		

Similarly the percentage of infants requiring phototherapy or exchange transfusion did not differ significantly. No complications from vitamin E therapy were noted.

Thus parenteral prophylactic vitamin E was not efficacious in minimizing either neonatal physiologic hyperbilirubinemia or neonatal jaundice associated with GGPD deficiency in Israeli subjects. Noteworthy also is the lack of excessive hyperbilirubinemia in G6PD deficient compared to normal subjects in this study population.

RATIONAL MANAGEMENT OF ASSISTED VENTILATION. Karl F.

1494 Schulze, Mark Stefanski, Julia Masterson, Andrew Schenkman, Michael Graff & L. Stanley James. Columbia Univ., Coll. P & S, Bables Hosp., Div. Perin., Dept. Ped., N.Y. Total ventilation (VIT) of artifically ventilated infants equals mechanical ventilation (VIM) plus infant ventilation (VIB) or VIT=VIM+VIB. Since ventilation is the product of tidal volume (VT) and frequency (f) then VTTxfT=(VTMxfM)+(VTBxfB). To evaluate any change in ventilatory support it is necessary to measure all the factors in the above equation. We examined the effects of reduced PEEP on the mean values and trends in these factors with continuous measurements of ventilation during alternate 30 min. periods of PEEP at 6cm H₂O, or 2cm H₂O. Ventilator rate, FiO₂ and peak pressure (PIP) were constant. Ten studies were performed on 6 infants weighing 740 to 1000g. All breaths were analyzed, x=4020 breaths/hour/infant.

			Kesults		•			
PEEP	VIT	v_{TT}	f_{T}	Ϋ _{IM}	v_{TM}	v_{IB}	v_{TB}	$f_{\mathbf{B}}$
6cm H20	157	2.59	61.1	63.7	5.95	89.7	1.85	48.2
2cm H ₂ 0	165	2.20	73.0	90.2	7.94	75.1	1.16	60.0
p<	N.S.	N.S.	.01	.02	.02	.10	.05	.01

At PEEP 2cm H2O VTM increased immediately due to a 4cm H2O increase in ΔP , then progressively decreased in all patients over time (\bar{x} decrease =15%). Thus at PEEP=2 \dot{v}_{IB} is less efficient (smaller v_{IB} & increased fg) & contributes less to \dot{v}_{IT} .

Optimal management of infants on assisted ventilation is not possible without continuous measurements of the above ventilatory parameters.

TRANSCUTANEOUS BILIRUBINOMETRY OLD AND NEW. Robert 1495 E. Schumacher and Gary R. Gutcher. Univ. of Wisc., Madison, WI (Spon. by William E. Segar).

As a screening device for hyperbilirubinemia, we compared the Minolta Janudice Meter (M) with the 30 year old Ingram icterometer (Ic). The M is a fiberoptic reflectometer marketed as a device to identify infants whose degree of jaundice indicates the need for chemical serum bilirubin estimation. The inexpensive Ic (appros. \$10) consists of a strip of transparent plexiglass on which are painted 5 yellow stripes of precise and graded hue. In use, the color of the baby's skin is matched with the stripes and a jaundice score assigned. 120 serum bilirubin values were obtained by A.C.A. Dupont Analyzer because of visible jaundice in a population of term, healthy caucasian newborns. Under controlled, artificial light, first the Ic then the M was used on each baby. Defining hyperbilirubinemia as > 12.9 mg/dl the sensitivity and specifity of each device was determined. For 120 trials the Ic had a sens. of .86 and a spec. of .75. The M had a sens. of .95 and spec. of .77. 2 of 3 false negatives with the Ic occurred in the first 4 measurements, a time when familiarity with the insturment was being obtained. The last 116 measurements produced a sens. of .95, spec. of .74 for the Ic and a sens. of .95, spec. of .77 for the M. Although serial individual assessment was consistent, interobserver variation was significant. With the Ic as with the M, we would suggest that individuals establish their own action levels prior to reliable usage. We conclude that in spite of the availability of more complex and expensive instruments, for the individual clinician, the Ic continues to serve as a sensitive, cost effective screening device for hyperbilirubinemia.

CIRCULATING SEROTONIN LEVELS IN PREMATURE INFANTS: Robert E. Schumacher, E. Burt Olson, Jr. and Philip 1496

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1496

Robert E. Schumacher, E. Burt Olson, Jr. and Philip
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We measured serotonin (5HT) levels in platlet-poor plasma (PPP) from premature newborns. 5HT is a neurotransmitter and a powerful vasopressor with specific vascular receptor sites as well as a-adrenergic receptor activity. 5HT is actively taken up and metabolized by the pulmonary vascular endothelium. This specific pulmonary 5HT uptake has been demonstrated in adult humans as well as in fetal and newborn animals. Animal models suggest that the 5HT uptake ability of the lung is greatest in the newborn period. 5HT is present in neuroendocrine cells (NEC) which are most numerous in the lung during the perinatal period, and animal studies suggest that 5HT may be released from NEC in response to alveolar hypoxia. Imbalances in circulating 5HT levels have been associated with changes in vascular hemodynamics. 46 samples (0.2 cc PPP) were obtained from 29 premature newborns (27-36 wks gestation). Samples were taken on days 2-3 and 6-7 of life. 17 of these represent paired serial samples. 5HT levels were measured using a precolumn sample enhancement technique followed by ion exchange HPLC with electrochemical detection. The average 5HT on days 2-3 was 1.67 + 0.65 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% con. limit], and on days 6-7 was 0.74 ± 0.26 ng/ml [mean ± 95% c

REDUCTION OF LBW WITH ENHANCED ANTENATAL CARE. † 1497 Kenneth E. Scott (spon. by Richard B. Goldbloom).

Dalhousie University Faculty of Medicine, Department of Pediatrics, Grace Maternity Hospital, Halifax, Nova Scotia.

A blind trial of enhanced antenatal care was carried out, involving 11,000 pregnancies. The purpose was to decrease (a)LBW, (b) need for antenatal referral and neonatal intensive care, and (c)mental handicap. Group allocation was by stratified randomization using previous LBW scores. Physicians used a risk appraisal system, and referred pregnant women to Community Health Nurses. The program emphasized prenatal class attendance, and home visiting for counselling on nutrition, exercise, food finances, support, coping with stress, smoking and alcohol.

Sub-group analysis excluding a non-compliant area showed a decrease in LBW , a 25% decrease <2000g (16.2vs21.7/1000, α =.043), and a 43% decrease in 1500-1999g (7.4vs12.9/1000, α =.007) in the

compliant enhanced care areas.

In the 25% of smokers still smoking 10+c/d at first home visit the LBW rate was 9.8%, in light smokers 4.0%, in non-smokers 2.8% 75% of BW variance with study socio-lifestyle factors was attributed to smoking. In heavy smokers only unmarried showed a higher incidence (α =.01) of LBW. Smokers accounted for 60% of LBWs

and heavy <u>smoking</u> accounted for 35% excess.

In interviewed non-smokers LBW showed a relation to inadequate nutrition (2.1vs3.8%, α =.03), lack of exercise (2.5vs4.9%, α =.05), and insufficient food finances (2.5vs6.4*, α =.01). Inadequate nutrition at first home visit accounted for 24* excess LBW, lack of exercise 12%, insufficient food finances 8%. Programs to reduce these detrimental factors decrease LBW.

PROPHYLACTIC INDOMETHACIN AND INTRAVENTRICULAR HEMORRHAGE (IVH) IN THE † 1498 INTRAVENTRICULAR HEMORRHAGE (IVH) IN THE PREMATURE: ES Setzer, BM Morse, RN Goldberg, M Smith, and E Bancalari, University of Miami, Jackson Memorial Hospital, Departments of Pediatrics and Radiology, Miami, FL.

have previously shown significant hemostatic derangements

We have previously shown significant hemostatic derangements associated with IVH and have questioned the use of indomethacin [I], a potent platelet anti-aggregating agent in infants vulnerable to IVH. During a prospective study of [I] prophylaxis for patent ductus arteriosus (PDA), we assessed the impact of [I] upon the incidence of IVH. Sixty one inborn infants were randomized by birth weight (Group A: <900 gm; Group B: 900-1300 gm) to receive either placebo [P] or [I]. The first dose (0.2 mg/kg), given within 12 hours after birth, was followed by two q 12 hourly doses (0.1 mg/kg). Cranial ultrasonography was performed prior to dose # 1, daily for 3 days and at 1 week. Abnormal scans were repeated weekly. The most severe grade of IVH (Papile criteria) sustained by each infant is shown below: (Papile criteria) sustained by each infant is shown below:

ı٧ 0 11 Ш Group A: [Pi (n=10) [I] (n=10) 0 0 Group B: [P] (n=22) 12 2 13 0 [] (n=19)

In Group A, 2 of 10 (20%) of infants who received I developed IVH (Grades II-IV) in contrast to 8 of 10 (80%) of infants given [P], p < 0.025. In Group B, there was no significant difference in IVH between [I] and [P]These preliminary data suggest that prophylactic [1] may diminish the incidence of IVH in infants < 900 grams, perhaps by preventing clinically significant PDA or by altering prostaglandin-related central nervous system vascular phenomena.