

1414 RETROLENTAL FIBROPLASIA (RLF) II - ASSOCIATION OF OXYGEN THERAPY. Malini Satish, Gerald Katzman, Venkatesan Krishnan, Daniel Marcus, Jerald Bovino,

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50 neonates with RLF identified between 1/75 and 12/79 were compared with a group of matched controls without RLF. There was a significantly greater number of hours of oxygen exposure in the RLF group over the control group. Analysis of duration of exposure to FiO₂ above room air is presented. No significant association was found with duration of exposure to FiO₂ > .5. RLF neonates were exposed to significantly longer periods of FiO₂ less than .5 than controls. Increased duration of oxygen exposure at any FiO₂ seems to influence RLF development more than the actual magnitude of the FiO₂.

	RLF		CONTROL		p*
	̄hrs.	S.D.	̄ hrs	S.D.	
Total Duration					
Oxygen Therapy	974	755	386	510	.001
100% Oxygen	5.11	10.9	8.13	44.3	NS
71-100% Oxygen	16.66	38.7	8.8	29	NS
51- 70% Oxygen	50.9	127.2	22.7	75.2	NS
41- 50% Oxygen	97.3	212.2	25.8	74.2	0.05
31- 40% Oxygen	198	231	72	124	0.01
21- 30% Oxygen	589	404	279	334	0.001

*t test also confirmed with Cochrane test

1415 STUDIES IN RETROLENTAL FIBROPLASIA (RLF) I - ASSOCIATION OF ARTERIAL PaO₂. Malini Satish, Gerald Katzman, Venkatesan Krishnan, Daniel Marcus, Jerald Bovino,

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Of 569 oxygen-treated neonates examined by two retinologists between 1/75 and 12/79, 50 had RLF. The severest grade in either eye is indicated in Table I. A comparison of the duration of exposure to several PaO₂ levels was made between RLF neonates and 50 weight-matched controls without RLF. Linear trending of the PaO₂ between individual PaO₂ determinations was assumed in order to estimate the time each patient was exposed to a given PaO₂ range. Only the period when arterial blood gases were obtainable from indwelling catheters was analyzed. Short periods of exposure to high PaO₂'s do not seem to have a significant relationship to the genesis of RLF. However, even modestly elevated PaO₂'s (80-99) for significant durations may influence RLF development.

TABLE #1	NUMBER	%
#Examined	569	
#RLF	50	8.8
Grades: I	29	5.0
II	10	1.75
III	5	0.9
IV	1	0.2
V	5	0.9

TABLE #2	̄	Duration Exposure	S.D.	P Value
PaO ₂ Range	RLF	Control	RLF/C	
150	1.56	1.67	1.26 1.74	NS
100-150	10.84	6.71	9.18 4.94	NS
80-99	24.02	10.62	21.61 7.37	.01
Peak PaO ₂	̄	̄	81 60	NS
	192.9	174.3		

1416 RETROLENTAL FIBROPLASIA (RLF) III - ASSOCIATION OF PHYSIOLOGIC STATE AND THERAPEUTIC MODALITIES OTHER THAN OXYGEN. Malini Satish, Gerald Katzman, Venkatesan Krishnan, Daniel Marcus, Jerald Bovino.

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50 neonates with RLF and their matched controls were studied. 16 of 34 parameters analyzed had significance with RLF and are presented. These suggest that compromised neonates are susceptible to RLF.

TABLE #1	̄	S.D.	CONTROL ̄	S.D.	P Value
TMV - hours	526.6	506.31	185.9	324.5	< .001
CFAP - hours	136.3	129.34	55.1	84.4	< .01
PaO ₂ < 50 (hours)	6.55	7	3.7	4.5	< .02
*Bradycardic spells	6.2	10.2	1.42	2.82	< .001
*Apneic spells	2.1	2.8	1.06	1.94	< .005
Peak PCO ₂	58	11.25	49.4	8.8	< .005

TABLE #2	RLF (50)		Control (50)		X ²	p
	Yes	No	Yes	No		
Exchange transfusion	22	28	8	42	8	< .005
FF Plasma Given	11	39	0	50	8.6	< .005
Abd. Distention	35	15	22	28	5.8	< .025
Ileus & NPO > 10 days	18	32	7	43	5.3	< .025
PDA-LA/AO > 1.3	15	35	5	45	5	< .005

Analysis of therapeutic modalities also seem to reflect the need for greater support in these patients. Therapies to improve tissue perfusion and oxygenation may paradoxically add to RLF risk.

1417 HYPOXANTHINE (HX) CONCENTRATIONS AS INDICATOR OF HYPOXIA. O.D. Saugstad, Bruce Kessel, Brian Saunders, Louis Gluck.

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The HX concentration is a specific indicator of tissue hypoxia. The plasma level of this purine metabolite can be used to assess intracellular energy status. Extensive animal studies, but less clinical data have evaluated monitoring this metabolite in routine clinical work. In the present study plasma HX has been determined according to the micromethod previously described (Saugstad, 1975). Umbilical cord blood from normal delivery & after asphyxia (by Apgar score & cardiocographic tracings) were studied. The mean HX concentration in non-asphyxiated babies was 9 μmol/l (±2SD=0-22 μmol/l). In babies with moderate asphyxia, the HX level was elevated (26-40 μmol/l). Arterial plasma HX from neonates with & without tissue hypoxia (by clinical signs & acid-base status) in 18 samples showed linear correlation between plasma HX & base deficit (BD): (BD=0.44HX -1.8 r=0.66 p<0.01). BD ranged between -6 & 21 μmol/l. The relation between HX & pH was: (pH= -0.006HX + 7.35 (r=0.46, p=0.05). No baby received sodium bicarbonate.

Conclusion: The present results demonstrating a good correlation between HX & BD is in agreement with animal studies. (Saugstad et al, 1978, Thiringer et al, 1980). Hypoxanthine determination is rapid & simple. HX more specifically than BD or lactate reflects tissue hypoxia. We therefore suggest that HX be measured routinely in clinical neonatology for assessment of hypoxia.

1418 INCREASED INCIDENCE OF EARLY ONSET HYPERBILIRUBINEMIA IN BREAST FED VERSUS BOTTLE FED INFANTS. Kenneth L. Saul and David Warburton (Spon. by Alan B. Lewis).

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We performed a 9 month retrospective study on 1351 full term healthy neonates (874 breast fed; 477 bottle fed) to compare the incidence of early onset hyperbilirubinemia (EOH) (defined as serum bilirubin >8 mg/dl within 48 hours after birth and/or >12 mg/dl after 48 hours but before 7 days). Excluded from the study were premature (<2500 gms), infants with direct hyperbilirubinemia and/or Rh or ABO blood group incompatibility. 379 (43%) breast fed and 182 (38%) bottle fed infants had serum bilirubin levels measured because of visible jaundice (p > 0.5). EOH was found in 293 (34%) breast fed and 109 (23%) bottle fed infants, (p < 0.001). In addition, marked EOH (>15 mg/dl) was found in 40 (5%) breast fed and 4 (1%) bottle fed infants (p < 0.001).

We have found a significantly greater incidence of EOH in breast fed as compared with bottle fed infants. Possible etiologies include dehydration and/or inadequate nutrition while the breast milk supply is coming in, or contributory factors in the breast milk itself. Breast fed infants should be carefully monitored for EOH in the first seven days of life.

1419 QUICK EVALUATION OF CARDIOPULMONARY ADAPTATION OF NEWBORNS BY OXYGEN-CARDIORESPIROGRAPHY. Harald Schachinger,

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Blood gas and acid base values in cord blood are often not predictive of neonatal cardiopulmonary adaptation. We employed oxygen-cardiorespirography to measure beat-to-beat heart rate, respiratory rate, thoracic impedance and transcutaneous Po₂ in 337 newborns. 16 infants had severe cardiorespiratory problems in the first hours (11 respiratory distress, 5 congenital heart disease); two of these had a cord blood pH below 7.15, and one showed a low Po₂. In all 16 infants oxygen-cardiorespirography was abnormal in at least one of the parameters. In clinically abnormal infants the tcPo₂ was low, and frequently a decrease of long-term variability of the heart rate was seen. In 10 of 16 infants, a pathological pattern was recognized by oxygen-cardiorespirography before clinical symptoms appeared.

A hyperoxia test adds additional information. Within 2 minutes following hyperoxia, a distinction can be made between healthy newborns, respiratory problems and congenital heart disease. Shunting through an open ductus arteriosus can be shown by a difference in tcPo₂ measured simultaneously from the thorax and abdomen.

Oxygen-cardiorespirography allows continuous multiparametric data collection and permits the early recognition of important trends in neonatal cardiopulmonary adaptation.