1213 AUGMENTATION OF PULMONARY SURFACTANT RELEASE BY LUNG EXPANSION AT BIRTH.

Edward E. Lawson, Robyn L. Birdwell, Pearl Huang, H. William Taeusch. Harvard Med. School, Children's Hosp. Med. Ctr., Dept. of Pediatr., Boston, MA.

Pulmonary stability increases within 30 minutes of birth in newborn rabbits, suggesting enhanced surfactant release after birth (J. Appl. Physiol. 37:809, 1974). The mechanism for this is unknown but circulating catecholamines and lung expansion per se (Fed. Proc. 32:1018, 1973) have been implicated. To study the effect of lung expansion we delivered by hysterotomy 7 litters of rabbit pups at 30 days gestation and divided them into 3 groups which were sacrificed: a) after 30 minutes air-breathing, b) after 30 minutes nitrogen-breathing, or c) after 30 minutes tracheal occlusion. Each group was compared to a littermate group sacrificed at birth. Groups b) and c) continued respiratory efforts for 30 minutes despite progressive asphyxia. Following sacrifice alveolar sufactant was recovered by saline lavage and measured quantitatively on a surface-tension balance as described by Clements (Science 169:603, 1970). Surfactant concentration at birth was 140±44µg/0.1gm dry lung and rose to 192±40µg after 30 minutes air-breathing ($p<.03$). Surfactant also increased in the nitrogen-breathing pups (312±108 vs 185±70 in littermate controls; $p<.05$) but not in the occluded group (178±41 vs 177±52). These results confirm the hypothesis that surfactant release is enhanced at birth. Furthermore, the data suggest that lung expansion, rather than circulating humoral factors associated with stress, is responsible for augmented surfactant release following birth.

1214 MORPHOLOGICAL ANALYSIS OF THE PULMONARY VASCULAR BED (PVB) IN CONGENITAL LEFT-SIDED DIAPHRAGMATIC HERNIA (CLDH).

Daniel L. Levin. (spon. by David Fixler) Univ. of Texas Health Science Center, Dept. of Ped., Dallas.

The contribution of an abnormal PVB to R→L shunting in patients with CLDH and alveolar hypoplasia has not been defined. In 3 infants we analyzed lungs fixed by perfusion. Serial sections were prepared and 5th generation (resistance) vessels identified. Left lung volumes (ml/kg birth weight) were 1.7, 6.5, and 4.0 (control =11.7) and right lung volumes 5.7, 11.5, and 9.8 (control=14.3). Compared to control, medial width/external diameter ratios (m/d) were increased in one lung from each patient: left, m/d=0.25±0.01 (S.E.)(n=50)($p<0.01$), m/d=0.21±0.01(n=51), m/d=0.28±0.01(n=43)($p<0.001$); right, m/d=0.21±0.01(n=49), m/d=0.24±0.01(n=48)($p<0.05$), m/d=0.22±0.01(n=48); control, m/d=0.21±0.01(n=138). The m/d ratio in the left was greater than right lung in 2/3($p<0.01$). We counted the total small muscular vessels in 10 to 30 randomly selected sections from each lung and calculated number of vessels/cm^2 lung. In each case there were fewer than control; left 112±8(S.E.), 66±5, 136±8; right 132±12, 197±19, 218±18; control 341±18 vessels/cm^2 ($p<0.001$). There were significantly fewer vessels/cm^2 in the left than in the right lung in 2/3($p<0.001$). In these patients tolazoline did not improve oxygenation. The data suggest pulmonary vasodilatory therapy may not be effective in these infants since the elevated pulmonary vascular resistance is due to decreased total size of the PVB and reduced number of vessels per unit lung rather than increased vasomotor tone. Supported by UT Grant 5-S01-RR-05426-14, AHA Grant 76-878, and AHA Texas Grant 53940.

1215 AIRWAY REACTIVITY IN CHILDREN FOLLOWING CROUP,

G.M. Loughlin* and L.M. Taussig, Department of Pediatrics, Arizona Health Sciences Center, Tucson, Arizona.

Pulmonary function was studied in 28 asymptomatic subjects, 9-18 years with a history of laryngotracheobronchitis (croup) at least 5 years previously. The subjects were separated into 2 groups, croup--no allergies (CNA) and croup--allergies (CA), based on allergy history, skin tests, and eosinophil counts. A control group of 10 children was also studied. Lung volumes, peak expiratory flow rates (PEFR), forced expiratory volume in 1 second (FEV_1), maximum mid-expiratory flow (MMEF), and maximum flow at 25% and 50% of the vital capacity ($\dot{V}max_{25}$ and $\dot{V}max_{50}$) were measured pre and post-exercise and post-isoproterenol inhalation. The response of flow rates to a helium-oxygen gas mixture was also measured. Baseline flow rates did not differ amoung the 3 groups. The table shows the number with >10% drop in flow rates post-exercise:

	Δ PEFR		Δ MMEF		Δ $\dot{V}max$ 50		Δ $\dot{V}max$ 50	
N	1/10 (10%)		1/10 (10%)		1/10 (10%)		2/10 (20%)	
CNA	4/11 (36%)	45%	3/11 (27%)	34%	6/10 (60%)	53%	6/11 (55%)	60%
CA	9/11 (54%)		7/17 (41%)		7/15 (46%)		11/17 (65%)	

Changes in PEFR, $\dot{V}max_{50}$ and $\dot{V}max_{25}$ for the CA and CNA groups were significantly different from normals at $p<.05$. None of the subjects had a history of exercised-induced bronchospasm and only 4 had a history of wheezing. This data suggests that patients with a past history of croup have normal resting bronchomotor tone but have an increased prevelance of both large and small airway hyperreactivity, which occurred irrespective of allergies.

1216 HELIUM MIXING INDEX (MI) IN INFANTS AND YOUNG CHILDREN,

G.M. Loughlin* and L.M. Taussig, Arizona Health Sciences Center, Tucson, Arizona.

Routine testing of lung function is difficult in young children. Ventilation distribution can be assessed by measuring the number of breaths to 90% equilibration while breathing a helium gas mixture in a closed circuit system used to measure functional residual capacity (FRC). The ratio of predicted breaths (Bates, 1953) to the observed is the MI. The MI was calculated in 77 normal children (N), 2-72 mos. of age. Similarly, 118 determinations were performed in 86 infants 2-24 mos. of age recovering from respiratory distress syndrome (RDS). The mean MI for N was 73% with a lower 97.5% confidence limit of 44%. The MI was independent of age or sex and there was no correlation between MI and respiratory rate (RR). 29/118 FRC determinations in the RDS group were abnormal. 49/89 of those with normal FRC had an abnormal MI. RR was >45/min in 43/49. In six infants only the MI was abnormal. There was an inverse correlation in the RDS group (-.24, $p<.005$) between MI and RR, suggesting a frequency dependence of helium equilibration. Resting arterial blood gases were obtained in 9 of the infants with normal FRC and abnormal MI. 4 who had MI <30 had $PaCO_2$ ≥40 mm Hg., despite RR >50/min. The other 5 had abnormal MI but relatively normal blood gases, possibly reflecting a perfusion adjustment to this ventilatory disturbance. In conclusion, the MI is a simple, convenient, and noninvasive test of lung function in a group in which few tests are applicable on a routine basis. It may be a more sensitive test of ventilatory dysfunction than blood gases.

1217 DESIGN OF DUAL ELEMENT SENSORS, ELECTRONIC SYSTEMS AND CALIBRATORS FOR THE TRANSCUTANEOUS MEASUREMENT OF PCO_2

Robert A. Macur, Ponthenkandath Sasidharan, John P. Grausz
Spon. by Fredrick M. Blodgett
Medical College of Wisconsin, Milwaukee County Medical Complex
Department of Pediatrics, Milwaukee, Wisconsin

A complete system suitable for continuous measurement of transcutaneous CO_2 clinically is described. The sensor is based upon dual Pd-PdO pH electrodes in a Severinghaus PCO_2 sensor. The isolated electronic system provides for two channel measurement of CO_2, displays interface temperature and the power (mW) required to maintain the interface temperature at 43°C. Alarms and lock-outs on the heater system are activated on high temperature or high power. Design of a free standing three gas calibration system is also described. System response time on a volunter was 2.5 minutes for a hyperventillatory 10 mm Hg PCO_2 change. Overall system drift at 50 mm Hg in the system calibrator was 0.5 mV/hr and 0.25 mV/hr for each channel. Electrode response to an O_2 change from 150-708 mm Hg showed an almost linear upward drift of 1.7 mV/hr. The effect of topical pressure on power (relative blood flow) is described. Thermal gradients and water balance problems are described. System evaluation on neonates is under way.

1218 IN UTERO LUNG LECITHIN METABOLISM IN TERM FETAL RABBITS.

Frank Mannino, Alan Jobe and Louis Gluck. Univ. of Calif. San Diego, Dept. of Pediatrics, La Jolla.

Fetal rabbits at 30 days gestation received lecithin precursors ^{14}C-palmitic acid, ^{3}H-choline and ^{32}P simultaneously by injection of pregnant does. Fetuses were delivered by cesarean section from 11 min to 18 hrs after isotope injection. Part of each litter was sacrificed at birth, and the remainder allowed to air breathe for 40 minutes. Alveolar wash surfactant was recovered by lavage from the newborn lungs. The lungs were homogenized, an aliquot was reserved for parenchymal lecithin analysis, and microsomal and lamellar body (LB) fractions were isolated from the remainder by density gradient centrifugation. Lecithin labeled with palmitic acid and choline appeared very rapidly in microsomal and parenchymal fractions while ^{32}P labeled lecithin appeared in the same fractions after approximately 1/2 hour. The appearance of labeled lecithin in the LB or alveolar wash fractions was delayed 3 hours, a delay identical to that found in other studies in newborn rabbits. The specific activity (SA) of LB lecithin exceeded the SA of alveolar wash lecithin at each time point, and maximal SA in these fractions was not achieved by 18 hours. The SA of lecithin in the lung fractions from the newborns allowed to air breathe approximated the SA of the lecithin from the non-breathing animals. This data for in utero term rabbits and previous studies with newborn rabbits indicate that newly formed lecithin slowly appears in the alveolar space - after a 3 hour delay - kinetics that are uniquely characteristic of lung phospholipid metabolism in the perinatal animal.