†1810 APNEA CAUSED BY REGURGITATION IN INFANTS. P. Aravindaksha Menon, Bradley T. Thach, Washington Univ. Sch. Med., Dept. Peds., St. Louis 63110.

Regurgitation (R) of gastric contents is presumed to cause apnea in some infants. The mechanism of such apnea is unclear and polygraphic documentation is lacking. We recorded nasal airflow (flowmeter), abdominal movements, oral CO_2 and EKG in 13 infants. Group A (9 preterm infants, age 2-4 wk and 1 term, age 1 wk) had a history of idiopathic prolonged apnea and ordinary post-feeding R episodes ("spitting up"); Group B (3 term infants, age 3 mo) had R only. R episodes were recorded in all infants (43 episodes in Group A and 10 in Group B). In 37 of 53 R episodes pharyngeal pressure and pH were recorded to aid in detection of R. In Group A most R episodes (92%) were immediately followed by either a short apnea (S-apnea = absent airflow $\geq$3 sec) or prolonged apnea (P-apnea = absent airflow for $\geq$20 sec or absent flow with heart rate $\leq$100 or cyanosis). Although most of the 100 P-apneas recorded in Group A were unrelated to R episodes, 8 P-apneas (occurring in 4 preterm infants) coincided with R (a statistically significant association in 3 infants: $p<.01$, $p<.05$, $p<.05$). All Group A infants had S or P-apnea, or both, during R. There were no S or P-apneas during R in Group B. Most P-apnea (83%) and S-apnea (78%) consisted of obstructive inspiratory efforts or breathing pauses combined with obstruction. We conclude: 1) regurgitation has little effect on breathing in healthy term infants but often causes S-apnea and, occasionally, P-apnea in infants with a past history of apnea; and 2) the mechanism of this apnea involves both airway obstruction and respiratory depression. Funding: NIH HD 10993.

1811 CORRELATION OF $paCO_2$ WITH EXPIRED CO_2 (P_ECO_2) MEASURED VIA MASS SPECTROMETRY IN NEONATAL CARDIOPULMONARY DISEASE. Robert G. Meny, Abdul M. Bhat, Christine DiGiovanni, David Dickey, Fatma Yehia (Spon. by Shih-Wen Huang), Hurley Med. Cen., Dept. of Pediatrics, Flint, MI.

Measurement of arterial carbon dioxide tension ($paCO_2$) is an invasive technique for determining the adequacy of ventilation and, therefore, is associated with possible morbidity. Consequently, a non-invasive means for estimating $paCO_2$ is desirable. A Chemetron mass spectrometer (MS) with a response time of 0.68 sec to 63% of a step change in CO_2 and analytic accuracy for CO_2 of ± 2% was used to measure P_ECO_2 of neonates on IPPV/CPAP. Gas was continually sampled from 1.0 cm beyond the plastic adapter of an oral ET tube via a catheter with inner diameter 0.38 mm. 9 neonates (mean BW 2182g, range 870-3440; mean GA 33.8 wk, range 26-40) with cardiopulmonary diseases (5 HMD, 2 aspiration, 1 each PDA, CHD, BPD, pneumothorax) were studied at an initial mean age of 82 hrs (6/9 < 72 hrs). 75 paired comparisons of P_ECO_2 and $paCO_2$ had a correlation of 0.69 ($p < 0.001$) and a mean gradient ($paCO_2$-P_ECO_2) of 8.3 torr (range +23 to -3). 61.3% of the gradients were $\leq$ 10 torr. We conclude that the MS is a useful monitoring device but that the correlation between $paCO_2$ and P_ECO_2 was adversely affected by the relatively long response time of the total system. We speculate that reduction of this parameter would enhance the usefulness of the MS by increasing the correlation and decreasing the gradient.

●1812 IMMUNE COMPLEX FORMATION IN RESPIRATORY DISTRESS SYNDROME (RDS). David Strayer, T. Allen Merritt, Mikko HALLMAN. Dept. of Pathol. Yale University, New Haven, Univ. of Calif., San Diego, Dept. of Ped., La Jolla, CA

To determine whether serum immune complex formation occurs following intratracheal instillation of exogenous human surfactant in infants with severe RDS, an ELISA assay for surfactant-anti-surfactant immune complexes was developed using rabbit anti-human surfactant antibodies followed by horseradish-peroxidase anti-human Ig. Anti-surfactant antibody formation during the first 3 months following surfactant instillation was measured & C_{50} & C_3 levels & urinalysis were performed to determine whether the classical pathway was activated. Seven infants receiving exogenous human surfactant & 11 infants with severe RDS receiving only intermittent mandatory ventilation (IMV) were compared. Serum surfactant-anti-surfactant immune complex formation was detected shortly after surfactant instillation & peaked in concentration (O.D.490) 7 days after treatment versus a peak in surfactant-anti-surfactant immune complex formation 12 days after birth in infants with severe RDS receiving only IMV. Complexes could be detected for 2 months following surfactant administration & for 25 days in IMV treated infants. C_{50} levels in surfactant treated infants was 90 ± 15 vs. 105 ± 22 U/ml & C_3 levels were similar in both groups & urinalysis did not reveal proteinuria. Although some variation in time course & peaks was observed, a consistent pattern of surfactant-anti-surfactant complexes was found in infants with RDS. These findings underscore the need for caution in administration of xenogenic biologically active substances to human infants.

1813 SYSTEMIC CARDIAC OUTPUT & BLOOD FLOW DISTRIBUTION IN SURFACTANT DEFICIENT, HIGH FREQUENCY OSCILLATED RABBITS. Robert Mirro, Tasio Kawano, Massa Tamura (Spon. by Pamela M. Fitzhardinge). Univ. of Toronto; Mount Sinai Hospital & Hospital for Sick Children. Depts. of Pediatrics and Respiratory Physiology, Toronto.

High frequency oscillation (HFO) appears to be an alternate, less traumatic mode of ventilating surfactant deficient patients. Conventional mechanical pressure limited ventilation (CMV) is known to compromise cardiac function at high mean airway pressures. Our objective was to compare systemic cardiac output and its distribution using these two modes of ventilation. We studied ten adult rabbits rendered surfactant deficient by pulmonary saline lavage and alternated on both modes of ventilation. Cardiac output and organ blood flow were measured using the radionucleotide labelled microsphere technique during HFO(f=15Hz) and CMV(Bournes BP-200), both at mean airway pressures of 15cm H_2O and F_IO_2=1.0. Cardiac output did not differ from HFO (353±10ml/min) to CMV (340±8ml/min). In addition, organ perfusion was similar during both modes of ventilation. (table)

Organ blood flow (ml/min/100gm tissue)

	HFO	CMV	
brain	193±19	223±25	
heart	327±23	333±28	paired-t N.S. at $p<0.05$
liver	5.9±1.3	4.8±1.4	
kidneys	194±20	176±12	(all data as mean±SEM)

At similar mean airway pressures there is no difference in systemic cardiac output from CMV to HFO. Furthermore this study found no evidence of organ ischemia during HFO.

1814 FETAL BREATHING MOVEMENTS AND LUNG HYPOPLASIA: PRELIMINARY HUMAN OBSERVATIONS. Harold E. Fox and Adrien C. Moessinger (Spon. by L. Stanley James), Depts. of Ob-Gyn and Pediatrics, College of Physicians and Surgeons of Columbia University, New York, N. Y.

Hypoplasia of the fetal lungs (L.H.) is found in association with long standing oligohydramnios. In the animal model, interference with fetal breathing (F.B.) activity by phrenectomy or curarization has led to lung hypoplasia. It has been suggested that L.H. associated with oligohydramnios is due to inhibition of F.B.

Four patients with oligohydramnios (2 intact membranes, 2 prolonged rupture) were studied in a total of eleven 60-minute sessions from 27-38 weeks. F.B. and other fetal movements (OFM) were recorded using linear array sonography. For each patient study session a gestational age matched control with normal amniotic fluid volume was studied. Three of the four study subjects had fetal lung hypoplasia. F.B. was observed in all four cases. For the 2 cases with intact membranes (Renal Agenesis and Polycystic Kidneys) F.B. was seen during 0.25±0.048 S.D. [L.H.] of the 60 min observation (control 0.06±0.02). With ruptured membranes F.B. was seen (0.02±0.05 and 0.069 [L.H.] of the time with control of 0.19±0.024). No difference in other OFM was noted.

From these preliminary observations we conclude that FB does occur in cases of oligohydramnios associated with lung hypoplasia. It is therefore unlikely that lung hypoplasia is merely the result of absent FB.

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1815 MEASUREMENT OF TOTAL THORACIC IMPEDANCE (TI) AND SPECIFIC CONDUCTANCE (SG) IN NORMAL INFANTS BY FORCED OSCILLATION. Wayne J. Morgan, Robert Tepper, Kathy Cota, Lynn M. Taussig, and GHMA Pediatricians, Dept. Ped. and Div. Resp. Sci., Arizona Health Sciences Center, Tucson, AZ.

We studied 56 normal infants (NL) and 16 infants with bronchopulmonary dysplasia (BPD) to determine alterations in TI and SG with growth in health and disease. TI was measured at 6 and 10 Hz by a standard forced oscillation technique. Functional residual capacity (FRC) was measured by helium dilution technique. The ages, FRCs, and lengths (L) were not different between groups. In both groups there was a significant decrease in TI with increasing size (NL: $r=-.53$, $p<.001$; BPD: $r=.54$, $p<.02$). The mean TI values were significantly higher for the BPD group:

	Normal	BPD	p Value	
TI-6Hz	34.4 ± 1.5*	46.8 ± 6.9	< .01	*Mean ± SE
TI-10Hz	27.6 ± 1.0	34.2 ± 3.9	< .05	

TI at 10Hz was significantly lower in both groups than TI at 6Hz ($p<.001$). SG also decreased with growth despite the decrease in TI (NL: $r=-.50$, $p<.001$; BPD: $r=-.38$, $p<.10$). There was no difference in SG between groups. We conclude that TI is easily measured in infants by forced oscillation and demonstrates the expected decrease with growth and a frequency dependence. Finally, SG decreases with growth suggesting a relative dysanapsis in lung growth with lung volume increasing more rapidly than airway size.

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