†1741 CORRELATION OF PLASMA ZINC AND CLINICAL STATUS IN PATIENTS WITH CYSTIC FIBROSIS. Jane D. Carver, James M. Sherman (Spon. by Barness) College of Medicine, Univ. of So. Florida, Dept. of Pedatrics, Tampa, FL

Plasma zinc levels have been variably reported as low or normal in patients with cystic fibrosis (CF). Zinc is a co-factor for more than 60 enzymes including the Δ6 desaturase enzyme, and may be required for the mobilization and transport of vitamin A, reported to be low in patients with CF. Zinc is also important in immune function. For these reasons, zinc is a potentially important mineral in patients with CF. We measured zinc and vitamin A levels in 25 patients with CF (ages 5-29) and compared them to controls. CF patients have significantly lower plasma zinc, 70.12 ± 2.66 and vitamin A, 30.13 ± 1.51, than do controls (94.11 ± 3.21 and 47.85 ± 4.41, respectively, $p<0.001$, $p<0.001$). Plasma zinc but not vitamin A levels corresponded to the degree of pulmonary involvement (normal or mildly affected 78.72 ± 4.34, moderately to severely affected 63.65 ± 1.89, $p<0.001$), but not to the presence of pancreatic insufficiency. Six patients were supplemented with zinc gluconate for 6 weeks with 3 mg/kg/day. Plasma zinc levels rose signficantly, although there was no obvious change observed in their clinical status. Plasma copper levels, which have a reciprocal relationship with plasma zinc, decreased significantly but remained within the normal range. A longer period of supplementation may be required to bring about a change in clinical status. Previous conflicting reports of zinc status in CF may reflect heterogeneity of pulmonary involvement between groups of patients studied.

1742 TREATMENT OF AIDS ASSOCIATED LYMPHOID INTERSTITIAL PNEUMONITIS WITH INTRAVENOUS GAMMAGLOBULIN AND PREDNISONE. Morris Charytan, Ben Zion Krieger, Andrew Wiznia, Larry Bernstein, Bernard Silverman, and Arye Rubinstein, Albert Einstein College of Medicine, Department of Pediatrics, Microbiology and Immunology, Bronx, New York.

6 children with Acquired Immunodeficiency Syndrome and biopsy proven lymphoid interstitial pneumonitis were treated on a protocol of intravenous gammaglobulin and corticosteroids. Indications for therapy were a clinical history of severe and/or recurrent episodes of respiratory distress responsive to intravenous antibiotics and hypoxemia persisting after resolution of the acute illness. Hypoxemia was defined as a $pO_2 < 70$ torr on three determinations. Therapy consisted of an initial period of loading with intravenous gammaglobulin. Duration and dosage of loading varied with the severity of the clinical circumstances. Corticosteroids were then initiated at 1-2 mg/kg/day and tapered to 0.75-1.0mg/kg on alternate days within 6-8 weeks. Prior to therapy the mean alveolar-arterial oxygen gradient ($AaDO_2$) was 47 torr and the mean pO_2 was 52 torr. After one month of therapy the mean $AaDO_2$ was 21 torr and pO_2 was 80 torr. At three months follow-up $AaDO_2$ was 15 torr and pO_2 79 torr. In the two patients treated for twelve months, $AaDO_2$ and pO_2 remained stable at 16 torr and 89 torr respectively. In vitro immunologic responses were not suppressed by the alternate day corticosteroid treatment.

1743 MECONIUM FREE FATTY ACIDS INDUCE ALVEOLAR COLLAPSE. David A. Clark, Gary F. Neiman, Jeffrey E. Thompson, Andy M. Paskanik, John E. Rokahr, Carl E.Brendenberg Departments of Pediatric and Surgery, Upstate Med. Ctr.Syracuse, N.Y., Neonatal ICU, Westchester Co. Med. Ctr., NYMC, Valhalla, N.Y..(Spons. by Harry S. Dweck).

Free fatty acids of meconium alter surface tension of lung extract in vitro. We examined meconium and its primary free fatty acids (oleic, palmitic, stearic) instilled in vivo into the trachea of 15 experimental and 8 control mongrel dogs who were anesthetized, placed on a piston ventilator and subjected to a left thoracotomy. The lungs were lavaged with meconium alone, a petroleum ether extract of meconium, or a suspension of free fatty acids in saline. Cardiac output, venous and arterial blood gases and femoral, pulmonary and left atrial pressures were monitored for two hours. Static lung compliance was calculated.

Mean airway pressure increased and static lung compliance decreased in both meconium and meconium extract groups. ($p<.05$). P_aO_2 decreased significantly ($p<.01$) without recovery to the baseline in all experimental groups. There were no changes in pH P_aO_2 or any hemodynamic parameter. The dogs were sacrificed and the surface tension of lung extract was measured in a Wilhelmy balance. Although atelectesis and copious airway foam were seen, the surface tension minimum of crude lung extract and airway foam was less than 10 dynes per centimeter. We conclude on the basis of the significant changes in lung compliance, increased airway pressure and decreased P_aO_2 that the free fatty acids of meconium may induce alveolar collapse by displacing surfactant from the alveolus.

1744 CHRONIC RESPIRATORY ACIDOSIS DOES NOT EFFECT P_sCO_2 ESTIMATION OF P_aCO_2. Aaron J. Cohen, (Spon. by Mary Ellen Wohl). Department of Newborn Medicine, Brigham & Women's Hospital and Division of Respiratory Diseases, Children's Hospital, Boston, MA.

To determine whether chronic respiratory acidosis effects the accuracy of transcutaneous estimation (P_sCO_2) of P_aCO_2, 27 paired measurements were obtained from 21 subjects with Cystic Fibrosis (CF) and 4 subjects with other chronic lung diseases. 8 subjects with CF were chronically hypercarbic PCO_2 (mean±sd)= 55.6 torr±8.4, mean pH_a=7.40. Age (mean±sd) was 25 yr.±9.9 and 20±8.3 for hyper- and normocarbic subjects respectively; age range: 10-44 yr. Blood was obtained under local anesthesia from the radial artery, iced, and analyzed within ½ hour. P_sCO_2 was measured at 45° using a prototype O_2/CO_2 combined sensor (Severinghaus/Radiometer) with corrections made for temperature and metabolic effects. P_sCO_2/P_aCO_2 (mean±sd) was 0.98±0.03 and 1.03±0.04 for normo- and hypercarbic subjects. Linear regression of P_sCO_2 on P_aCO_2: $P_sCO_2 = 1.05\ P_aCO_2 - 2.27$, (Syx-1.58, Sxx=3172.41). Using these data, estimates of P_aCO_2 (P_aCO_2) from a new P_sCO_2 measurement may be made. (See Table).

P_sCO_2	P_aCO_2	95%C.I.
60	59.2	±3.2
50	49.7	±0.7
40	40.2	±0.7
30	30.7	±1.0

Chronic respiratory acidosis has no clinically important effect on P_sCO_2 estimation of P_aCO_2.

1745 THE HYPERCARBIC VENTILATORY RESPONSE TEST: NEAR-MISS SIDS, SIBLINGS OF SIDS, AND SUBSEQUENT APNEA. Michael Coleman, Christine A. Reardon, Mark C. Mammel, Stephen J. Boros, Children's Hospital, St. Paul, MN

Hypercarbic ventilatory response (HVR) tests were administered to 65 near-miss SIDS victims, 78 siblings of SIDS victims, and 31 controls. HVR values were compared, then correlated with the incidence of subsequent apnea. HVR tests used a steady-state, breath-by-breath technique. HVR results were expressed as changes in exhaled minute volume per change in $PACO_2$ (ml/min/kg/mmHg $PACO_2$). Twenty-three near-miss SIDS victims (35%) had subsequent apnea; one died of SIDS. Seven siblings of SIDS victims (9%) eventually developed apnea; two died of SIDS. HVR values were similar in the three patient groups. HVR values were not different from controls in either those infants with previous apnea or those who developed subsequent apnea. Surprisingly, resting $PACO_2$ values were lower in the mear-miss group ($P<.05$). When all infants who developed subsequent apnea (both near-miss and siblings) were compared to all those who did not, those with subsequent apnea also had lower $PACO_2$ values ($P<0.001$) along with higher HVR values ($P<.05$).

Conclusions: 1) HVR values were not depressed in near-miss SIDS or siblings of SIDS victims; 2) Resting $PACO_2$ values were lower in near-miss SIDS victims; 3) Infants who developed subsequent apnea had higher HVR values and lower $PACO_2$ values than those who did not.

=1746 MORPHOMETRY OF OLIGOHYDRAMNIOS-INDUCED FETAL LUNG HYPOPLASIA. Margaret H. Collins, Adrien Moessinger, Jerome Kleinerman, William Blanc. Coll. of P & S, Columbia Univ., Presb. Med. Ctr., Depts. Path. & Pediatr.; Mt. Sinai Sch. Med., Dept. Path., New York.

In fetal guinea pigs oligohydramnios (OH) causes lung hypoplasia which is more severe the earlier the onset and the longer the duration of OH (Ped. Res. 18:336A, 1984). We have quantitated the structural alterations in the lungs of fetal guinea pigs subjected to OH from 45 to 50 days gestation (term 67 days). OH fetuses (n=5) compared to littermate controls (n=4) have:

	OH	Control	P
Lung/Body Weight Ratio ($X10^{-2}$)	2.8	3.2	<.006
Lung Volume (ml)	1.17	1.34	<.04
Volume Density Parenchyma	.83	.90	<.025
Total Number of Saccules ($X10^6$)	46	69	>.05
Internal Surface Area (cm^2)	698	974	<.04
Total Length Parenchymal Elastic Tissue (M)	504	974	<.0025
Length Elastic Tissue (M/mm^3)	.51	.81	<.025

Even a brief period of OH during the late canalicular-early saccular phases of lung development is sufficient to markedly reduce fetal lung growth and to cause profound structural changes. The disproportionate effect of OH on elastic tissue may be related to the fact that this tissue first appears in the parenchyma of the fetal guinea pig lung on day 45; this adverse effect might impede the ability of the lung to recover and could have long term sequelae.