†1789 LUNG DISEASE AND OTHER DETERMINANTS OF ENERGY NEEDS IN C.F.: L. Levy, V. Stallings, P. Pencharz, M. Corey Department of Paediatrics, Research Institute, The Hospital for Sick Children, Toronto, Canada.

Energy requirements in CF patients have been estimated to allow for malabsorption. Energy needs may also be increased due to the extra work of breathing as lung disease worsens. In order to find out which factors relate to energy needs, we conducted sixteen, 75 min. calorimetry studies on 15 patients (Mean age(y)=16 .4±4.9(SD),range,9-26y). Standard open circuit indirect techniques were used. Subjects were fasted x 16hr., and were afebrile. Ten subjects were recovering from chest infection within the preceeding two weeks. Lung disease ranged from mild to severe ($\bar{X}FEV_1$ =40±16%predicted). Twelve subjects were underweight ($\bar{X}$ weight as a percentage of ideal=84±10). Factors studied in relation to energy expenditure were fat free mass, %fat, $W^{0.75}$, Wt.%ideal, pulm. flow rates, arterialized blood gases, ±Pseudemonas cepacia in sputum, and infection within the preceeding two weeks. Actual energy expenditure ranged from 92-149% of predicted values as determined from the Harris-Benedict equation. The variables most closely linked to energy expenditure were, in order of significance, $PaCO_2$(r^2=0.60), infection(r^2=0.36), FVC(r^2=0.27), FEV_1(r^2 =0.24), $W^{0.75}$ (r^2=0.22). We conclude 1)That the energy needs of some CF patients are higher due to increased energy expenditure. 2)That increased energy expenditure is primarily related to lung disease. 3)That the most sensitive indicator relating lung disease to energy expenditure is $PaCO_2$.

1790 Donald E. Lighter, William G. Perkins (Sponsored by Sachchida N. Sinha); Department of Pediatrics, University of Tennessee, Knoxville Unit. Use of a Spreadsheet Program to Calculate Pediatric Pulmonary Function Normal Values.

The spreadsheet program Multiplan was used to configure a worksheet for calculating pulmonary function normals on a Fortune 32:16 computer. The worksheet used linear and multiple regression formulas found in the pediatric literature to calculate pulmonary function normal values based on patient sex, race, weight, and height. The program output included: Forced Vital Capacity, Residual Volume, Total Lung Capacity, Forced Expiratory Volume, Maximum Midexpiratory Flow Rate, Wright peak Flow Rate, Peak Expiratory Flow Rate, Maximum Expiratory Flow Capacities, Airway Resistance, and Airway Conductance. The worksheet consisted of a block for user input of patient parameters, a calculation block that calculated normal values using the regression equations, and an output block that yielded specific normal values for the patient using the Boolean logic functions of the spreadsheet program. The user can enter patient pulmonary function test results into the output block to obtain a percentage comparison with the calculated normal values. Since the multiple regression formulas contained complex mathematical constructs (e.g. fractional exponential terms), manual calculation of the normal values consumed 20 minutes of technician time; using this spreadsheet, the time is reduced to 1 - 2 minutes. Development of the spreadsheet took approximately 1.5 hours, and development of a program in Applesoft BASIC that performed the same calculations took nearly 6 hours. Spreadsheet programs such as Multiplan can reduce development time for calculation intense clinical applications.

1791 Lymphoid Interstitial Pneumonitis (LIP) in children with Acquired Immunodeficiency Syndrome (AIDS) prodrome. Marina I. Liscano, L. R. Laraya-Cuasay, and Lawrence D. Frenkel. (Spon. by Lawrence T. Taft); UMDNJ-Rutgers Medical School, New Brunswick, NJ.

Three children aged 11 mos. to 3 yrs. with LIP diagnosed by open lung biopsy had clinical and laboratory features of AIDS prodrome. No opportunistic organisms were isolated from the lungs or morphologically demonstrated. Two of the patients are black and one is biracial (black-Caucasian). One girl's father had AIDS and died of PTB and Pneumocystis pneumonitis. Her mother is on INH for PTB. The 2nd child's mother is mentally handicapped, sexually promiscuous, and alcoholic, and a possible drug abuser, while the father is unknown. The 3rd patient's mother has no risk factors but the father is a Vietnam war veteran who was treated for PTB. None received blood transfusion prior to presentation with recurrent respiratory infections, failure to thrive, persistent Candida infection, generalized lymphadenopathy and hepatosplenomegaly. Roentgenogram showed diffuse reticulonodular interstitial densities. All have $pO_2 <$ 80 mm.Hg in room air. Immunologic studies showed marked hypergammaglobulinemia, depressed cellular immunity, inverted T-cell subset ratios, elevated circulating immune complexes, normal chemotaxis, depressed natural killer and bactericidal cell functions, positive $HTLV_3$ serology and elevated $alpha_1$ thymosin. None of the patients were treated with steroids. One receives gamma globulin. All three received antibiotics intermittently. All have been clinically stable during the 5-24 months of follow up. Steroids need not be given in children with LIP in AIDS prodrome.

†1792 TRACHEAL INFLAMMATION AND HIGH-FREQUENCY VENTILATION. Mark C. Mammel, Janice P. Ophoven, Margaret J. Gordon, Mary C. Sutton, Stephen J. Boros, Children's Hospital, St. Paul, and University of Minnesota, Minneapolis, MN.

Tracheobronchial histopathology following conventional positive-pressure ventilation (CPPV) was compared to that following high-frequency positive-pressure ventilation (HFPPV) and two types of high-frequency jet ventilation. Twenty-six cats were each ventilated for 16 hours. Seven received CPPV via an infant ventilator (Bourns BP200) cycling at 30 bpm. Seven received HFPPV using the same ventilator cycling at 150 bpm. Six received HFJV via an IDC VS600 ventilator (HFJV1) cycling at 250 bpm. Six received HFJV using a Bunnell ventilator (HFJV2) cycling at 400 bpm. A 4-point, 9-variable histologic scoring system graded tissue changes at four levels of the tracheobronchial tree. High-frequency ventilation produced more inflammatory tracheal injury at the endotracheal tube tip when compared to CPPV ($P < .05$). CPPV produced more histopathology at the carina and brainstem bronchi ($P < .05$). There were no significant differences in injury patterns produced by HFJV1 or HFPPV; HFJV2 resulted in less lower airway damage than either HFJV1 or HFPPV ($P < .05$).

In this study, high-frequency ventilation produced inflammatory injuries near the endotracheal tube tip. CPPV produced different and greater histopathology distally. These differences suggest that CPPV and high-frequency ventilation have different injury mechanisms. Tracheal inflammation seen with HFPPV suggests that frequency, not ventilator type, may be at least partially responsible for this lesion.

†1793 FATTY ACID BINDING TO LUNG CYTOSOLIC PROTEINS. William M. Maniscalco and Jacob N. Finkelstein. University of Rochester School of Medicine, Strong Memorial Hospital, Department of Pediatrics, Rochester, NY.

In tissues with a high rate of fatty acid utilization, intracellular free fatty acids are bound to a low molecular weight (12000 daltons) fatty acid binding protein (FABP). By differential binding of fatty acids, FABP may compartmentalize different fatty acid species and regulate cellular free fatty acid utilization. To determine if lung contains cytosolic proteins that bind free fatty acids, we prepared a 100,000xg supernatant of whole rabbit lung and incubated it with radiolabeled palmitate. After gel filtration on a calibrated Sephadex G-75 column, two peaks of radioactivity were noted. The first eluted in the void volume and probably represents high molecular weight aggregates of fatty acids. A second peak eluted at the volume calibrated to be approximately 12000 daltons. Similar studies on supernatants prepared from pure adult type II pneumocytes showed a similar elution profile. No peak in this region was observed following incubation of the cytosol with radiolabeled phosphatidylcholine. Incubation with equimolar concentrations of radiolabeled oleate produced a smaller peak than observed with palmitate. These studies suggest that whole lung and pure type II cells contain an intracellular binding protein for fatty acids which may be similar to FABP. (Supported by a grant from the March of Dimes--Birth Defects Foundation 1-923).

†1794 SURFACTANT TREATMENT IN NEONATES WITH SEVERE RDS: IMPROVES OXYGENATION WITHOUT IMPROVING DYNAMIC LUNG COMPLIANCE ($dynC_L$). Frank Mannino, T. Allen Merritt, Mikko Hallman, Howard Schneider. UCSD Medical Center, School of Medicine, Department of Pediatrics, San Diego and Children's Hospital, Helsinki, Finland.

We determined the $dynC_L$ on six neonates (wt. 1.0±0.1kg) with severe RDS before and after successful human surfactant treatment (mean age 6.6 hr). Transpulmonary pressure was measured with a differential transducer comparing the pressure at the proximal ET tube to pressure from an esophageal balloon. Flow was measured by a pneumatochygraph and volume determined from the area of the flow tracing. The $dynC_L$ during spontaneous and ventilator breaths was calculated as the ratio of volume to transpulmonary pressure change between points of no flow. Blood gases and ventilator settings were recorded at the time the pulmonary studies were performed (mean 25 min before, 36 min after surfactant treatment). With treatment the mean F_iO_2 required for adequate oxygenation decreased from 0.89 to 0.46 ($P < .001$); there was no change in PaO_2 or ventilator settings. The $dynC_L$ before treatment was 0.45±0.11 ml/cmH_2O for spontaneous breaths and 0.26±0.16 ml/cmH_2O for ventilator breaths; after treatment the $dynC_L$ was 0.37±0.06 ml/cmH_2O and 0.17±0.16 ml/cmH_2O respectively. On chest radiographs lung volume was increased after treatment. Surfactant therapy did not alter acutely the $dynC_L$ in severe RDS. We speculate that surfactant treatment increases FRC thereby improving $\dot{V}/\dot{Q}$ inequality. The poor compliance characteristic of RDS may be due to factors other than surfactant deficiency.