1777 NEW LIGHT ON STUDIES OF CHEST WALL DISTORTION. Gregory P. Heldt (Spon. by Richard D. Bland). Cardiovascular Research Institute and Dept. of Pediatrics. University of California, San Francisco.

Chest wall distortion (CWD) has been studied with magnetometers, strain gauges, and inductance plethysmography - all methods for estimating the volume excursion of the chest wall (CW). To quantitatively describe the movement and volume changes of the CW, we developed a system to optically map the CW of small infants. We project a grid onto the torso, and view the infant with a video camera from an angle of 45° from the axis of the projected grid. The video information is later played back frame-by-frame, and a cursor is superimposed on the intersections of the grid to determine their positions. The viewing angle and the calibration of the projector and camera are used to calculate 130 X-Y-Z coordinates for the surface. We studied an infant with this system, weighing 1980g. The right half of the CW was divided into four axial slices, 1.4 cm thick, located in the upper, middle, lower, and costal portion of the CW. Volume changes of each slice were calculated at 4 equally spaced times during 5 breaths, expressed in ml, as the difference from end-expiration. The total volume of the slices represents the overall CW volume change. The uncertainty of the volume measurements was ±3.6%, based on measurements of test objects. The variability of the volumes from breath to breath averaged ±5.4% of the volumes shown.

Slice	End Exp	Early Insp	Late Insp	Early Exp	Late Exp
Upper	0	-1.77	-1.28	0.42	1.16
Middle	0	-1.14	-2.01	-0.84	-0.87
Lower	0	1.15	-1.76	0.51	2.21
Costal	0	1.45	2.75	1.65	-0.05
Total CW	0	-0.31	-2.30	1.74	2.45

The upper and lower CW moved paradoxically during inspiration, with an overshoot in expiration, whereas the middle CW moved paradoxically throughout breathing. The costal portion had no paradox. The total CW volume is consistent both in pattern and volume change with estimates made with the inductance plethysmograph.

†1778 BRONCHODILATOR RESPONSE IN INFANTS WITH CYSTIC FIBROSIS. P.Hiatt, R.Tepper and H.Eigen. (Spon. by J.Lemons) Indiana Univ. School of Medicine, Indianapolis.

Cystic fibrosis infants (CFI) develop airways obstruction which is frequently treated with bronchodilators although no studies have yet documented their efficacy. The purpose of our study was to evaluate the response of CFI to the inhaled bronchodilator metaproterenol (BD). We studied 14 CFI, mean age of 20.5 months (range 3-39). Functional residual capacity (FRC) was measured by helium dilution. Maximal flows at FRC, VmaxFRC, were obtained by the rapid chest compression technique and expressed as size corrected flows, $\dot{V}$maxFRC/FRC. For CFI, a paired t-test revealed no significant change compared to baseline in $\dot{V}$maxFRC/FRC ($P>.15$) after NS, but an increase ($P<0.01$) after BD, of the group mean by +30%. Six of 14 increased by >30% and 8 < 30% but baseline $\dot{V}$maxFRC/FRC of these 2 groups were similar (.94 vs 1.00) ($P>.7$). We assessed interpatient variability of BD response by testing 5 normal infants, mean age 6.7 months (range 4.5-8), whose baseline $\dot{V}$maxFRC/FRC was similar to CFI (1.05 vs .98) ($P>.4$). None had a change of > 30% with either NS or BD and only 2/5 had an increase post BD. In contrast 13/14 CFI increased flows post BD. Two of 4 CFI < 1 year and 5/10 < 2 years of age increased flows > 30% post BD. We conclude that CFI can demonstrate a significant improvement in lung function after inhaled BD. The magnitude of response is not related to age or baseline function.

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†1779 EPITHELIAL FLUX AND EPIDERMAL STORAGE OF WATER IN CYSTIC FIBROSIS. Richard E. Honicky, Thomas Adams and Mahlon C. Smith (Spon. by Marshall Klaus). Michigan State University, Depts. Ped. and Hum. Devel., Physiol. and Mech. Engin., East Lansing, MI.

Comparatively little attention has been given to the direct measurement of water in secretory processes and products in CF. Using a newly developed non-invasive technique, we have quantified under conditions of stable thermal and mass transfer coeff. both steady state as well as dynamic hydration and dehydration phenomena in 11 disease-free persons randomly selected for age, sex and race, and in those with CF. Preliminary data indicate that the mean steady state transcutaneous water loss rate (Ess) from the intact skin of the medial forearm of 2, 6 y/o twins (Swachman scores: 73 and 75) was twice that of the disease-free group (mean ± SE: 19.1 ± 1.1 and 9.3 ± 0.6 $\mu g \cdot cm^{-2} \cdot min^{-1}$, resp.). Also, time-controlled (T:min) hydration tests using water-impermeable barriers at the skin surface indicate mean epidermal water storage (Vol; µg) follows the pattern: $Vol=122.5+4.2T+0.01T^2$ ($r=0.995$) for these children, and $Vol=64.3+1.9T-0.01T^2$ ($r=0.988$) for the control group. These data are consistent with the observation that unimpeded transepidermal water diffusion is higher in CF. Older children (9 and 18 y/o; Swachman scores: 64 and 80, resp.) show little difference in Ess (each was 10.0 $\mu g \cdot cm^{-2} \cdot min^{-1}$) compared to the control group, however, they may also hydrate their skin more rapidly ($Vol=55.9+6.9T-0.1T^2$ [$r=0.988$] and $Vol=57.7+3.6T-0.1T^2$ [$r=0.991$], resp.) for currently unknown reasons. We suggest that transcutaneous water diffusion and storage rates are elevated in CF, and that these measurements may provide new and useful information about epithelial transport abnormalities.

=1780 HUMAN SURFACTANT ASSOCIATED APOLIPOPROTEIN A: MOLECULAR COMPOSITION AND PRIMARY TRANSLATION PRODUCTS. William Hull, Timothy Weaver, Gary Ross, Jeffrey Whitsett. University of Cincinnati College of Medicine, Cincinnati, Ohio.

Surfactant apolipoprotein A (apo A) is a major lipid binding protein and an active component of pulmonary surfactant. Apo A was purified from human alveolar lavage, amniotic fluid and alveolar proteinosis fluid. Two major forms were identified by silver stain and immunoblot: apo A_2 (Mr=34,000), apo A_1 (Mr=28,000). Larger forms were reduced to Mr=28,000 by treatment with endoglycosidase F demonstrating complex N-linked oligosaccharide. Isoelectric point was increased by neuraminidase demonstrating presence of sialic acid. Poly A mRNA isolated from adult human lung was translated in vitro; primary translation products were identified at Mr=28,000. Homology between Mr=34,000 and 28,000 was confirmed by analysis of 2D tryptic peptide maps of apo A_1 and A_2 which were identical, thus providing evidence that apo A results from processing of the Mr=28,000 precursor by addition of carbohydrate. Amino acid composition of purified apo A was rich in glycine and contained a large portion of collagen-like sequence. Apoproteins A were identified in surfactant from amniotic fluid, normal adult lung lavage, human cadaver lavage and material obtained from a patient with alveolar proteinosis. Apo A was identified in all of these samples by silver stain and immunoblot analysis. Alveolar proteinosis fluid contained acidic aggregates whose peptide maps were identical to apo A from normal human lung lavage. These studies clarify the identity of human apo A, a complex N-linked glycoprotein derived from a polypeptide of Mr=28,000.

1781 MORPHOMETRY OF LUNGS FROM FETAL LAMBS TREATED WITH CORTISOL. June Z. Kendall, Jeffrey Lakritz, Allison J. Weir, Charles G. Plopper, Geoffrey C. Randall, Gail E. Richards and Manubai Nagamani. (Spon.by George T. Bryan). U.T.M.B. Depts Ob-Gyn & Pediat. Galveston, Tx., Univ. of Calif. Dept of Anat. Davis, Ca. and A.D.R.I. Ottawa Canada.

In the ovine fetus a surge in cortisol (F) at term is considered to mature the lung for birth. Infusion of F at 0.9 of gestation enhances surfactant production and lung stability but does not promote maximum distensibility. To assess whether cortisol promotes structural maturity of immature lungs we infused (Fx) 4 fetal lambs (130d GA) until labor appeared to be established (57.5 ±3.2hrs). 3 sham operated fetuses served as 131d controls (C). 4 intact fetuses were delivered at term. Lungs were perfused with Karnovskys solution, processed for LM and evaluated using stereology. Plasma F and tracheal fluid F increased in all Fx fetuses; maternal plasma P_4 declined up to 50% of basal values. Lung vol. and lung vol/body wt. were similar in Fx and C. Compared to C Fx induced the following changes; a decrease in the % of parenchymal tissue ($p=.05$), an increase in abs. vol. of parenchyma ($p=.05$) and nonparenchyma ($p=.04$) and increases in parenchymal airspace ($p=.05$). These differences from C were also seen in term animals.

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1782 DIETHLYCARBAMAZINE(DEC)DOES NOT BLOCK HYPOXIC PULMONARY VASOCONSTRICTION (HPVC) IN AWAKE LAMBS. Thomas J. Kulik, Carrie Balcom, James E. Lock, Harvard Medical School, The Children's Hospital, Department of Cardiology, Boston

We and others have reported that certain leukotriene(LT)antagonists abolish HPVC. If LTs are necessary for HPVC, all LT antagonists should block HPVC. We therefore studied the LT synthesis blocker DEC. Six lambs(7-14 dy old)were chronically instrumented for cardiac output(CO;l/min.), pulmonary artery(PA), left atrial (LA;n=4), and aortic(Ao)pressures(P;mmHg). Five days later, with the lambs awake, PAP,LAP,AoP, and CO were measured in normoxia(N), in hypoxia prior to DEC(H), in H after IV boluses of DEC (H+DEC; 1,3,10, and 20 mg/kg), and after hypoxia was stopped (N_2).

We found: 1) DEC caused an early(+10 sec.), transient (lasting <30 sec.), small, fall in PAP(19%), AoP(26%), and CO(19%)(these values after 10 mg/kg; all $p>.05$). At no dose was DEC even a transient pulmonary vasodilator. 2) These parameters, and pulmonary and systemic vascular resistance(PR and SR;units),measured after 45 min. of H and a total of at least 34 mg/kg of DEC, were not different from those in H prior to DEC. Pulmonary arteriolar resistance (n=4) was also unchanged (22.7 vs. 22.1 units).

Condition	PAP	AoP	CO	PR	SR all ±S.D.
N	18±5	87±5	1.11±.50	18±6	98±55 *p<.05 compared
H	33±12*	83±17	1.37±.59	28±14	76±54 with N (ANOVA)
H+DEC	32±7	72±19	1.42±.62	28±16	64±47 **p<.05 compared
N_2	20±7**	71±18	1.04±.37	20±6	72±16 to H+DEC (ANOVA)

Hence, DEC does not block HPVC in the awake lamb, unlike other LT antagonists. This data does not support the hypothesis that LTs are necessary for the generation of HPVC.