

**1171** CORRELATION OF RESPIRATOR DIAL "COMPLIANCE" WITH MEASURED LUNG COMPLIANCE. Gordon B. Avery, Joan Conry, and Cheryl M. Naulty. George Washington Univ. Dept. Child Health and Dev., and Children's Hosp. Nat. Med. Center, Washington, D.C.

In clinical practice, lung compliance is often inferred from pressure and volume readings on the respirator dials. These estimates differ from measured lung compliance ( $C_L$ ) in that both pressure and volume are diminished by gas compression, flexing of the tubing and any leaks. Lung compliance was measured using a pneumotach and esophageal balloon and compared with  $\Delta V/\Delta P$  from the dials of the Bourne respirator. 102 data pairs from 25 prematures with HMD at varying stages of the disease were compared. Infants ranged from 14 hours to 48 days in age. The overall correlation between true and calculated compliance was poor,  $R = .539$ . The calculated values from the respirator were neither systematically above or below the actual  $C_L$  values. The problem was random variation. Sources of error include high peak pressures which cause increased compression in the tubing and low apparent  $C_L$  leaks in the tubing or respirator, which cause high apparent  $C_L$ , and inaccuracies in the respirator dials themselves. These data suggest that inferences about lung mechanics from the respirator readings are inaccurate and can be misleading.

**1172** WHAT IS BEST PEEP? Gordon B. Avery, and Cheryl M. Naulty. Children's Hosp. Nat. Med. Ctr. and George Wash. Univ. Sch. of Med., Dept. Child Health and Dev., Washington, D.C.

The interrelationships of compliance ( $C_L$ ),  $O_2$  transfer,  $CO_2$  excretion, and pulmonary circulation have not been explored in prematures with HMD. Using an esophageal balloon and pneumotach,  $C_L$  was measured at intervals during titration of PEEP, together with arterial blood gases, in 7 prematures with HMD varying from 1h to 9d in age. As PEEP was varied stepwise upwards and downwards, an optimal value was found for  $C_L$ ,  $PaO_2$ , and  $AaDO_2$ . A 15 min. equilibration time was allowed at each PEEP setting, and the entire titration spanned 2-3 hrs. "Best PEEP" was different for  $C_L$ ,  $PaO_2$ , and  $AaDO_2$ , although best  $PaO_2$  and best  $AaDO_2$  were very similar. Interestingly, best  $PaO_2$  was at a significantly higher PEEP than best  $C_L$  (mean 5 vs. 7,  $lcm H_2O$ ,  $P < .05$ ).  $PaCO_2$  rose at PEEPs of 6-9 in 4 patients, but was not limiting to "best PEEP." The titration itself yielded an apparently beneficial "opening up" effect in 3 babies, in that repeat measurement of  $PaO_2$  and  $C_L$  at the same PEEP showed improvement. Clinical estimations of PEEP were often not the same as recommended best PEEP, based on these titrations. In 3 cases PEEP was increased, in 2 it was decreased, and in 2 the previous setting was considered optimum. These data suggest that no single parameter defines "best PEEP," but rather it must be a judicious compromise.

**1173** BETAMETHASONE INDUCTION OF LECITHIN SYNTHESIS AND PHOSPHATIDIC ACID PHOSPHATASE IN FETAL LUNG. Philip L. Ballard, Arlette Brehier, Bradley J. Benson, Mary C. Williams and Robert J. Mason. Spec. Center of Res. in Pulm. Dis., Cardiovas. Res. Inst. and Depts. of Ped., Anat., and Med., Univ. of California, San Francisco.

We studied the effect of maternal glucocorticoid treatment on lecithin synthesis by lung slices and on the activity of three key enzymes (phosphatidic acid phosphatase (PAPase), choline phosphotransferase and phosphatidylglycerolphosphate synthetase) involved in surfactant synthesis. Pregnant rabbits (n=23) were injected with betamethasone (acetate plus phosphate) at 0.25 mg/kg IM (1 or 2 doses) or with saline (controls), and fetal tissues were obtained at 26 days gestation 12-48 hr after treatment. Glucocorticoid activity in maternal and fetal plasma reached peak levels of 129 and 33  $\mu g/dl$  cortisol equivalents, respectively, at 1-2 hr and returned to preinjection levels (4.2 and 1.4) between 24 and 48 hr. Treatment significantly increased the rate of choline incorporation into lecithin (pmol/mg tissue/hr) and the activity of PAPase (nmol P/mg protein/min) in lung, but not liver, homogenate. Mean  $\pm$ SE values are:

	Control	12 hr	24 hr	48 hr	24+48 hr
Choline Uptake	3.25 $\pm$ .11	4.13 $\pm$ .26	4.97 $\pm$ .23	4.07 $\pm$ .42	5.33 $\pm$ .21
PAPase (lung)	6.6 $\pm$ 0.5	7.4 $\pm$ 0.3	9.5 $\pm$ 0.5	10.8 $\pm$ 0.7	12.8 $\pm$ 0.6
PAPase (liver)	7.2 $\pm$ 0.6	—	5.6 $\pm$ 0.2	5.9 $\pm$ 0.5	5.0 $\pm$ 0.2

There was no significant change in the other lung enzymes. We conclude that betamethasone, at a dose equivalent to that used in human prenatal therapy, stimulates both lecithin synthesis and the specific activity of PAPase in fetal rabbit lung.

**1174** PRENATAL BETAMETHASONE FOR THE PREVENTION OF IDIOPATHIC RESPIRATORY DISTRESS SYNDROME. R. A. Ballard, S. Sniderman, P. L. Ballard, and P. Granberg, Depts. of Ped., Mt. Zion Hosp. and Med Ctr. and Univ. of Calif., San Francisco.

In a retrospective study, we examined the effect of betamethasone (Celestone Soluspan), 12 mg in 2 doses 12-24 hours apart given to women in premature labor at 26-34 wks gestation on incidence of idiopathic respiratory distress syndrome (IRDS). We compared 84 treated babies with 125 untreated infants, all weighing 750-1750 gm and < 35 wks gestation. IRDS occurred in 63/125 (50.4%) untreated vs 24/84 (28.6%) partially or fully treated babies and survival was 78.4% vs 85.7%, respectively. We examined this by time from first treatment to delivery:

TIME (N)	0-24 hrs(19)	24-48 hrs(25)	2-10 days(40)	control(125)
IRDS	7 (36.8%)NS	10(40%)NS	7(17.5%)p<0.005	63(50.4%)
SURVIVAL	16(84.2%)NS	18(72%)NS	38(95.0%)p=0.05	98(78.4%)

We compared the 2-10 day treated group with the untreated group and found no significant difference in sex ( $\sigma^1$  50.0% vs 47.2%), mean birth weight (gm) (1351 $\pm$  44 vs 1334 $\pm$  26), gestational age (30.1 $\pm$  0.4 vs 30.8 $\pm$  0.2), incidence of intrauterine growth retardation (2.5% vs 4.8%), or Cesarean section deliveries (22.5% vs 21.6%). In babies with IRDS survival was 7/7 (100%) vs 37/63 (59%) NS. The length of time membranes were ruptured had no effect on the incidence of IRDS in either group. Therapy was not effective in babies < 750 gms. We conclude that the incidence of IRDS is decreased and survival enhanced in small preterm infants born 2-10 days after treatment with betamethasone.

**1175** CONTINUOUS MONITORING OF ARTERIAL  $PaCO_2$  BY NON-INVASIVE TRANSCUTANEOUS METHOD. Anthony V. Beran, Joseph J. Munoz, Gordon Y. Shigezawa, and Robert F. Huxtable. (Spon. by Thomas L. Nelson) University of CA College of Medicine, Department of Pediatrics, Irvine, CA.

Antimony oxide (Sb-SbOx) and pH-sensitive glass electrodes, combined with reference electrodes, surrounded by electrolyte and covered with gas permeable membrane (gpm) were used for transcutaneous  $PCO_2$  ( $P_{TC}CO_2$ ) measurement. A servo-controlled heater unit maintained sensor temp. and produced local hyperemia. A thermistor was placed under the gpm to measure true sensor temp.  $O_2$  sensitivity of Sb-SbOx electrode was reduced greatly in the 65-175 mm Hg  $PO_2$  range by cell loading, but below 65 mm Hg,  $O_2$  sensitivity was significant. The pH-sensitive glass electrode was preferred due to its insensitivity to  $O_2$ . After the sensor was placed on the skin of rabbits, it was calibrated *in situ* and its temp. coefficient established. This was repeated at the end of the 6-hr. experiment. For the best correlation between  $PaCO_2$  and  $P_{TC}CO_2$ , the skin surface temp. was maintained between 40-42°C, as lower temps. made  $P_{TC}CO_2$  higher. Based on *in situ* calibration, the fitted regression line had a slope of 1.029, intercept of -4.39, and SE of 4.14 mm Hg  $PCO_2$ . In the range of 21-75 mm Hg,  $P_{TC}CO_2$  was 2.2 - 3.8 mm Hg below  $PaCO_2$ . Based on *in situ* calibration with one point  $PaCO_2$  calibration, the line had a slope of 1.0, intercept of 0.6, SE of 3.4 mm Hg  $PCO_2$ , and linear regression coefficient of 1.0. These data indicate the usefulness of  $P_{TC}CO_2$  monitoring to indicate  $PaCO_2$  changes under normal physiologic conditions.

**1176** LUNG COMPOSITION AND STRUCTURE IN CONGENITAL UNILATERAL DIAPHRAGMATIC HERNIA. Will R. Blackburn, Phyllis Logsdon, and John A. Alexander. University of South Alabama College of Medicine, Department of Pathology, Pediatric Pathology Section, Mobile, Alabama.

The lungs of five infants dying with unoperated diaphragmatic hernia were studied by morphological and biochemical methods. The hypoplastic "H-Lung" was greatly reduced in size and weight ( $P < .001$ ) as compared to the contralateral control "C-Lung". DNA analyses suggested a marked reduction in cell number within the H-Lung (23.5 mg/HL vs 103.3 mg/CL). Glycogen levels were variable but tended to be elevated in H-Lungs. H-Lungs contained less total lipid (1.12 mg/mg DNA) than C-Lungs (1.56 mg/mg DNA). The phospholipid fraction was only slightly depressed (0.46 mg/mg DNA vs 0.55 mg/mg DNA). Phosphatidylcholine (PC) was significantly ( $P < .02$ ) decreased in H-Lungs (0.22 mg/mg DNA vs 0.37 mg/mg DNA). Electron Microscopic studies revealed mild retardation in pneumocyte cytodifferentiation in the C-Lung (as compared to the lungs of age matched infants without hernia); the H-lung showed severe growth and developmental retardation at the substructural level. Comparisons of H- and C-Lungs revealed no significant alterations in the ratio of type II pneumocytes/non-type II pneumocytes. Collectively these findings offer an explanation for the observed susceptibility of the H-Lung to hyaline membrane disease and edema following surgical correction. Supported by USPHS NIH Grant HL-17328-03.