1676 CPAP DOES NOT ALTER CO2 SENSITIVITY. A.Krauss,R.Goldstein,V.Alfero,W.Frayer,P.Auld. Perinatology Cent., Dept. Pediatrics, Cornell Med. Center, New York, N.Y.

CPAP has been proposed as an effective treatment for the apnea of prematurity. Its mechanism of action is unknown. Twentyfour preterm infants receiving CPAP for the treatment of respiratory distress were studied at 0,5,and 10cm H2O CPAP. These infants were allowed to breathe 2%,5%,and 8% CO2 for 3 minutes each. During the 3rd minute, recordings of tidal volume,respiratory rate, and esophageal pressure were made. From these recordings, minute volume,inspiratory time (Ti),expiratory time (Te), compliance, duty cycle (Ti/Ttot), tidal volume/inspiratory time ratio (Vt/Ti) and CO2 sensitivity were calculated. Although CO2 sensitivity rose from 3.9±6.8 cc/min x mm Hg^{-1} at 0 cm H2O of CPAP to 4.8±8.0 at 10 cm H2O, this difference was not statistically significant. Significant differences in respiratory rate, Ti, Te, Vt/Ti, and Ti/Ttot were not observed when values for all 3 levels of CPAP were compared. Measurements of intraesophageal pressure indicated that 62% of the applied airway pressure was transmitted to the mediastinal structures at 5 cm H2O and 54% of applied pressure at 10 cm H2O. These results suggest that in a group of premature infants with poor CO2 sensitivity that CPAP does not either improve CO2 sensitivity or the drive to breathe, and that other explanations, such as improvement in arterial blood gases or stabilization of the chest wall must account for the relief of apnea in infants receiving CPAP therapy.

1677 USE OF HIGH FREQUENCY POSITIVE PRESSURE VENTILATION (HFPPV) IN CHRONIC VENTILATORY FAILURE IN NEONATES. Venkatesan Krishnan, Malini Satish, Gerald Katzman, Jose Urrutia, Sidney Kripke, Irwin Weinfeld, P.L.S. Amma. (Spon. by M. G. Robinson) Medical College of Ohio, The Toledo Hospital, Dept. of Ped., Toledo, Ohio.

HFPPV (ventilator rates 100-150/minute; I.E. ratio 1:2) was used in 4 infants of gestational ages 26-29 weeks, birth weight 700-1000 gms. Age at commencing HFPPV was 25-90 days. All were on mechanical ventilation with radiological and clinical bronchopulmonary dysplasia. The Baby Bird ventilator was used. Duration of HFPPV 3-4 weeks.

	PCO_2	FiO_2	$TcPO_2$	$A\text{-}adO_2$	PIP/PEEP
	$\bar{x}$	$\bar{x}$	$\bar{x}$	$\bar{x}$	$\bar{x}$
Before HFPPV	65	100%	43	613	22.75/4.5
1-6 hrs. on HFPPV	33	95%	61	575	22.75/4.5
4-6 days on HFPPV	44	56.5%	--	330	15.5/4.5
Off HFPPV to ventilator rates 20-40 per minute	59	47.5%	--	365	17.25/4.5

3 infants were successfully weaned off ventilators. The fourth infant who had mechanical ventilation discontinued had no evidence of pulmonary barotrauma on autopsy. HFPPV can be used with success in chronic ventilatory failure in neonates.

1678 PRESSURE PLUS TIME: REQUISITES FOR INFLATION OF IMMATURE LUNGS. Ashok Kumar, Bella C. Clutario, Cheryl Doyle and Emile M. Scarpelli. Pediatric Pulmonary Division, Albert Einstein College of Medicine, Bronx, N.Y. 10461.

Functional anatomy of 0.87 gestation (immature) rabbit fetal lungs was studied by direct stereomicroscopic observation as the first air volume-pressure (VP) diagrams were recorded. Lungs were airless, but contained normal volume of fetal pulmonary fluid (FPF) prior to air-inflation. Volume was recorded 0, 15 and 120 sec after stepwise changes of 5 cm H_2O pressure. Inflation pressures up to 25 cm H_2O (P_{25}) produced large, time-dependent, volume changes in which the conducting airways were distended and FPF moved to the periphery of the parent airway before lateral branches were inflated. Characteristically, there was no saccular aeration below P_{25}. At pressures greater than P_{25}, but below P_{max}, subpleural saccules were the first to aerate; repeated inflation-deflation cycles in this pressure range produced preferential aeration of the same saccules, which thereby became vulnerable to rupture. As pressure was raised from ~P_{25} to P_{max} (i.e., P_{35}-P_{40}), saccules were recruited by both time- and pressure-dependent processes and recruitment continued, but at a slower rate, during early deflation. Time-dependent changes predominated at high pressures and were related to FPF flow through terminal ducts, formation of transient labile bubbles, and progressive enlargement of saccules. During deflation from P_{25}, derecruitment of saccules was largely time-dependent and air-trapping by FPF locks in the airways accounted for most of the residual air at P_0. We conclude that time can be as significant a factor as pressure in the initial inflations of immature lungs.

1679 PHYSIOLOGIC SEQUELAE OF NO_2-INDUCED BRONCHIOLITIS. Carin Lam, Meyer Kattan, Alexander M. Collins, Jerome Kleinerman (Spon. by Kurt Hirschhorn) Depts. of Pediatrics & Pathology, Mt. Sinai Sch. of Med., N.Y., N.Y.

To test the hypothesis that injury in the developing lung may lead to chronic obstructive lung disease, we studied the effects of NO_2-induced bronchiolitis in newborn hamsters. Three day (d) Syrian hamsters were exposed continuously to 30-35 ppm NO_2 23 hrs daily for 7 d. Pressure-volume and flow-volume curves during forced expiration at -50 cm H_2O and minimal volumes (MV) were determined on excised lungs at 3 and 9 weeks (wk) of age. In NO_2 animals studied at 3 wk (n=5), maximal expiratory flow at 40% total lung capacity (TLC) and elastic recoil pressure at 40% TLC were significantly lower than in control group (c) (n=4) ($p<.02$ and $p<.04$, respectively), while MV was significantly increased ($p<.02$). The upstream resistance (mean ± SEM) in the 3 wk NO_2 group (.275 ± .058 cm H_2O/ml/sec) was greater than in (c) (.209 ± .010) (p=NS). All measurements in 9 wk NO_2 animals (n=4) were not significantly different from (c) (n=6). The decreased flow rates and increased MV in the NO_2 group suggest small airways obstruction which may be related to decreased elastic recoil, increased airways resistance or both factors. The reversibility after 8 wk may result from 1) lung growth producing an increase in airways diameter, 2) alteration of the elastic properties of the lung associated with an increase in alveolar number during growth, or 3) physiologic accommodation to the effects of NO_2. These data do not support the concept that acute lung injury is a progenitor of chronic lung disease. (Supp. by Amer. Lung Assn.)

Abstract Withdrawn

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