CHEST PHYSIOTHERAPY IN THE NEONATE WITH RESPIRATORY 1189 DISTRESS, <u>Neil N. Finer</u>, <u>Michael G. Grace & Jan Boyd</u> (<u>Spons. by D. Schiff</u>) University of Alberta, Royal (Spons. by D. Schill) University of Alberta, noyal Alexandra Hospital, Dept. of Pediatrics, Edmonton, Alberta Chest physiotherapy is a frequently practiced but poorly docu-mented form of therapy. Twenty neonates (mean age=43 hr) were studied with a variety of respiratory disorders to assess the ef-fect of complete chest physiotherapy on arterial blood gases. This was compared to the alteration of blood gases following suc-tioning of the airway. Nine were receiving added oxygen alone, six were receiving continuous distending pressure and five were intubated receiving mechanical ventilation. Complete chest physio-therapy treatment consisted of postural drainage with percussions and vibrations in each drainage position followed by suctioning of the airway. The treatment was performed for a specific area of lung involvement if present. For generalized lung disorders, drainage was performed with an emphasis on the basal segments of the lower lobes. Each infant had arterial blood gases performed five minutes prior to and twenty minutes following suctioning, and similarly before and after physiotherapy (average interval be-tween the two pairs=1.8 hr) There was a mean increase of 20.8 mm Hg in the pO_{1} following physiotherapy, $(p\langle 0,01)$ with no significant change observed following suctioning. There was no significant change in the pH or pCO_{2} with either procedure. Analysis of covariance revealed no significant correlations between pO_{2} and sex, age, body weight, gestation, inspired oxygen concentration, or the order of receiving the procedures. In neonates with respi-ratory distress, oxygenation is improved following physiotherapy.

1190 PULIONARY IRRITANT REFLEXES IN NEWBORN INFANTS. The Hospital for Sick Children, Toronto, Canada. It has been well established that slowly adapting vagal stretch receptors (the Hering-Breuer reflex) are active in new-born infants, while the vagal rapidly adapting (irritant) re-ceptors have not been studied. We have tested the activity of the irritant receptors in two ways by 1. rapid lung deflation with negative pressure (-5 cm) applied via a face mask; and 2. direct stimulation of the bronchial mucosa with a fine catheter in intubated infants. Rapid lung deflation in 8 infants (gesta-tion 29-40 weeks) failed to stimulate respiration in all (control frequency 46+16; frequency during deflation 43+16, meanFSD). Direct bronchial mucosal stimulation in 8 intubated babies Direct bronchial mucosal stimulation in 8 intubated babies (gestation 29-39 weeks) failed to stimulate respiration in 5 infants less than 35 weeks gestation. In 3 infants, 35-39 weeks, respiratory frequency markedly increased or the infant coughed. The failure to elicit a deflation reflex in all infants, and the absence of response to mucosal stimulation in some infants suggests that vagal irritant reflexes are weak or absent in human newborns. This may be one reason why aspiration is common

in this age group.

11191 A NEW APPROACH FOR MEASURING FUNCTIONAL RESIDUAL CA-PACITY (FRC) IN THE INTUBATED INFANT. William W. Fox, Jacob G. Schwartz, Thomas H. Shaffer (Spon. by Jean A. Cortner) Univ. of Pa. Sch. of Med., Depts. of Peds. and Physiol. and The Children's Hospital of Philadelphia, Philadelphia, PA. Measurement of FRC in distressed neonates on continuous posi-tive airway pressure (CPAP) with endotracheal (ET) tubes presents special problems. Since neonatal ET tubes are uncuffed they per-mit leakage of gas around the tube during respiration and, there-fore, introduce errors in FRC determination. The present study evaluates a new 60 second closed circuit helium (He) dilution technique for determination of FRC independent of small gas leaks. By analytically relating the fall in He concentration due to mix-ing with that due to leakage it is possible to predict the final equilibration concentration of He and, therefore, correct for ET tube leaks. The system (120 ml) contains an air pump, He meter, breathing bag in cylinder, a strip chart readout, and solenoid valve. CPAP or ventilator pressure can be applied during testing. Fifty in vitro measurements of FRC ranging from 5-50 cc in both leak and non-leak models were accurate to 7.2 and 5.2% respective-ly. Seventy-two FRC measurements were performed on 15 neonates (700-4400 g) on CPAP with ET tubes. Leak rates were significant-ly higher (P<.COI) on 3 cm H20 CPAP compared to 0 cm H20 CPAP resulting in a mean (+SEM) total helium loss in 60 seconds of 16.2 (+ 1.1)% and 9.1 (+ 0.5)% respectively. The mean measured FRC was 53.5 ml at 3 cm H20 CPAP and 46.3 at 0 cm H20 CPAP. If leak calculations were not considered the error in FRC could have been as high as 76.7 and 42.7%. This method permits accurate calculation of FRC in neonates on CPAP with ET tubes.

A NEW APPROACH FOR MEASURING FUNCTIONAL RESIDUAL CA-

1192 ALTERATIONS IN NEONATAL RESPIRATORY FUNCTION FOLLOWING CHEST PHYSIOTHERAPY. William W. Fox, Jacob G. Schwartz, Thomas H. Shaffer (Spon. by Jean A. Cortner) Univ. of Pa. Sch. of Med., Depts. of Peds. and Physiol. and The Children's Hosp. of Phila., Philadelphia, PA. Percussion, suctioning and hyperventilation have been recom-mended for airway management in neonates requiring endotracheal tubes. To investigate physiological alterations in respiratory function due to chest physiotherapy, we measured arterial blood gases, respiratory patterns, lung mechanics, and functional resi-dual capacity (FRC) in 10 neonates, weights (1.25 to 3.20 kg) during the control period, post vibration of the chest and suc-tioning, after hyperventilation, and 2 hours post suctioning. Post suctioning compared to controls mean (+ SEM) Pa02 decreased 75.2 (+ 9.3) to 44.0 (+ 3.1) mm Hg (P<0.01) while lung compliance and FRC were unchanged. There was a decrease in inspiratory re-sistance (RI) from 81.8 (+ 14.6) to 59.0 (+ 10.6) cm H20/L/sec. (P<0.02) and I:E ratio increased from 0.93 (+ 0.04) to 1.04 (+ 0. 06) (P<0.07). Hyperventilation compared to post suctioning re-sistance (RI) from 81.8 (+ 0.500 the 32.0 (+ 0.04) to 1.04 (+ 0. 06) (P<0.07). Hyperventilation compared to post suctioning re-(P<0.02) and I:E ratio increased from 0.93 (+ 0.04) to 1.04 (+ 0. 06) (P<0.07). Hyperventilation compared to post suctioning resulted in an increase in mean (+ SEM) PaO2 to 63.9 (+ 10.0) mmHg (P<0.01), and RI to 98.3 (+ 22.0) cm H20/L/sec. (P<0.05). FRC, CL, VT were unchanged. Two hour followup values for all parameters studied were similar to control values except for a trend toward increased compliance. There were no significant differences at any stage of the study for PaCO2, pH, base excess, VT, or minute ventilation. This study indicates that there is a significant decrease in PaO2 after hyperventilation but these changes do not appear to be related to alterations in lung volume.

1193 FACTORS AFFECTING LUNG VOLUME IN POST EXTUBATED NEONATES. <u>William W. Fox</u>, Jacob G. Schwartz, Thomas <u>H. Shaffer</u> (Spon. by Jean A. Cortner) Univ. of Pa. Sch. of Med., Depts. of Peds. and Physiol., and The Children's Hospital of Phila., Philadelphia, PA.

Hospital of Phila., Philadelphia, PA. Extubated neonates previously treated with endotracheal con-tinuous positive airway pressure (CPAP) maintain arterial oxygen tension (PaO2) and functional residual capacity (FRC) equivalent to values at 2-3 cm CPAP without alteration in inspired oxygen concentration. To determine the factors that maintain PaO2 and FRC after extubation we studied arterial blood gases, lung me-chanics, and FRC in 15 neonates weighing 1.4 to 4.4 kg. Intra-esophageal pressure, air flow, and tidal volume provided for calculation of lung compliance (CL), and lung inspiratory (RI) and expiratory (RE) resistance. Following extubation at zero CPAP, increases occurred in mean (+ SEM) PaO2 from 59.3 (+ 5.8) to 79.5 (+ 4.0) mm Hg (P<0.05); FRC from 53.9 (± 6.0) to 67.3 (± 10.0) ml; RE from 148.0 (+ 17.3) to 167.5 (± 25.7) cm H2O/L/ second, and CL from 1.70 (+ 0.33) to 2.09 (+ 0.4) ml/cm H2O. I:E ratio decreased from 0.95 (± 0.05) at zero CPAP to 0.83 (+ 0.07) after extubation (P<0.05). A significant correlation existed between FRC and expiratory resistance for all patients (P<0.01). No significant alterations occurred in PaCO2, base excess, minute ventilation, tidal volume, respiratory rate and heart rate. The changes in FRC with changes in expiratory resistance after ex-tubation indicate that upper airway resistance , possibly at the laryngeal level may have a major role in maintenance of an adequate FRC after extubation.

1194 OXYGEN TOXICITY: COMPARISON OF LUNG BIOCHEMICAL RESPONSES OF NEONATAL AND ADULT RATS. Lee Frank, John Yam, and Robert J. Roberts, The University of Iowa, Depts. Of Pediatrics and Pharmacology, Iowa City, Iowa. Although immature animals have long been known to be less susceptible to 0, toxicity than adults, the basic mechanism(s) to explain this phenomenon remains unresolved. Two biochemical mechanisms for protection of the lung from 0,-induced injury have been proposed: the glutathione (GSH) system in reducing toxic lipid peroxide anion. Experiments were carried out to determine whether these lung defense systems respond differently in neonatal and adult rats exposed to toxic concentrations of 0,. Neonatal rats (4 to 7 days old) and adult rats showed extensive pulmonary edema and 65% died within 3 days of 0, exposure. Neonatal rats, however, all survived up to 5 days of 20, exposure without gross evidence of lung edema. During the course of exposure animals were sacrificed for biochemical analysis and the data expressed on a per lung basis as % of control values. After 72 hours of 0, exposure neonatal rats showed increased activity of GSH (171%), GSH-peroxidase (GP) (126%), GSH-reductase (GR) (120%), glucose-6-phosphate dehydrogenase (G-6-PD) (139%), and SOD (114%). Adult rats, however, failed to show increases in pulmonary GSH, GP, GR, and SOD activity. Thus, the resistance of the neonatal lung to 0_2 -induced injury may be due to the augmented activity of these protective enzyme systems. (Supported by GM 12675 and NIH 1F32 HL05415.)