DISTRIBUTION OF DIAPHRAGMATIC BLOOD FLOW DURING 1855 INSPIRATORY RESISTIVE LOADED BREATHING IN PIGLETS. Jon F. Watchko, Thomas A. Standaert, David E. Department of Pediatrics, University of Washington, Woodrum. Seattle, WA.

Recently, it has been suggested that the diaphragm is composed of two separate muscles, its costal and crural compo-nents, that serve distinctly different functional roles depend-ing on the demands of the respiratory system. Because blood Ing on the demands of the respiratory system. Because blood flow to skeletal muscle is proportional to the muscular effort expended (Cir. Res. 10:94, 1962), an examination of the distri-bution of blood flow to the costal and crural components should be an accurate means of assessing the partition of effort within the diaphragm. We examined costal ( $Q_{c}$ ) and crural ( $Q_{c}$ ) dia-phragmatic blood flow on 5 anesthetized spontaneously breath-ing piglets (age 15-23 days, wt. 2.8-4.2 kg) in order to assess partition of offort during coordinates and the diaphrage. In pipelets (age 13-2) days, wt. 2.6-4.2 kg) in order to assess partition of effort during inspiratory resistive loaded breath-ing (IRL).  $Q_{c}$  and  $Q_{cr}$  were measured using radionuclide labelled microspheres during quiet breathing and after 30' of IRL.  $Q_{co}$  and  $Q_{cr}$ , expressed in cc/100 g tissue/min (±SD) in-creased significantly above baseline values after 30' of IRL. There were no significant differences between Q and Q during the baseline and IBL periods the baseline and IRL periods. Q 14.0±4.0 Q 14.5±3.7 BASELINE

IRL X 30' IRL X 30' 41.7±14\* 45.9±17\* We conclude that diaphragmatic muscular efforts, as reflected by changes in Q and Q , increase during IRL and that the partition of effort is equally distributed between costal and crural components. (\*Compared to baseline, paired t test p<.05).

end poly (A) mKNA Was isolated from adult rat lungs. In vitro translation resulted in mRNA-dependent incorporation of 'S-methionine into TCA-precipitable proteins. Immunoprecipitation of these translation products, with rabbit antisera directed against rat Apo(s) A, identified a protein with Mr=26,000; this protein was not precipitated by non-immune rabbit sera. Specificity of the immunoprecipitated protein was verified by competition experiments: addition of rat lung Apo A completely inhibited immunoprecipitation of Mr=26,000; addition of BSA or DPPC had no effect. Further, Mr=26,000 was not detected in in vitro translated rat liver poly (A) mRNA. Two-dimensional SDS-PAGE demonstrated that Mr=26,000, pl=4.3, co-migrated with Apo A<sub>1</sub> from rat lung lavage and also with protein immunoprecipitated from 'S-methionine-labelled Type II epithelial cells. Addition of tunicamycin to Type II cell cultures resulted in appearance of Apo A<sub>1</sub> and A<sub>2</sub> were identical. Collectively, these observations demonstrate that Mr=26,000 is the intracellular precursor to rat pulmonary surfactant Apo(s) A and that larger molecular weight forms result from extensive N-linked glycosylation of the primary translation product.

EFFECT OF ALMITRINE ON HYPOGLOSSAL AND PHRENIC •1857 ELECTRONEUROGRAMS. Debra E. Weese-Mayer, Robert T. Brouillette, Linda Klemka, and Carl E. Hunt. Northwestern University, Children's Memorial Hospital, Department of Pediatrics, Chicago, IL.

Admitrise increases breathing by stimulating peripheral chemo-receptors. Previous studies suggest clinical usefulness in adults with COPD but few data are available to decide whether almitrine would be helpful in diseases involving pharyngeal airway obstruction such as apnea of prematurity or obstructive sleep apnea. We investigated the effect of intravenous almitrine on hypoglossal (HG, an upper airway nerve), and phrenic (PHR) neural activity in eight chloralose-urethane aneshetized, paralyzed, vagotomized, ventilated cats. Recordings were made of raw and integrated HG and PHR electroneurograms (ENGs),  $P_ACO_2$ , of raw and integrated HG and PHR electroneurograms (ENGs),  $P_ACO_2$ , PaO<sub>2</sub>, arterial blood pressure and rectal temperature. We found that: 1) in a dose-response study (N=3 cats) at doses of 0.1 -4.0 mg/kg, almitrine doses as low as 0.1 mg/kg increased both HG and PHR ENG activity, with a maximum effect at 1.0 mg/kg; 2) holding PaCO<sub>2</sub> at 40 mmHg, almitrine markedly increased HG and PHR ENG activity at all PaO<sub>2</sub> values from 35-175 mmHg (N=5 cats); 3) holding PaO<sub>2</sub> above 150 mmHg, almitrine increased HG and PHR ENG activity at all PaCO<sub>2</sub> values from 30-70 mmHg (N=4 cats); 4) in a ventilatory parameter timing study almitrine increased  $V_T/T_i$  and decreased  $T_i/T_{tot}$  at normoxia and eucapnea (N=6 cats). If the finding that almitrine increases upper airway-maintaining activity can be confirmed in unanesthetized sleeping animals, activity can be confirmed in unanesthetized sleeping animals, almitrine may be useful in obstructive sleep apnea and apnea of prematurity.

DETERMINANTS OF TIDAL VOLUME(VT) DURING HIGH-FREQUENCY JET VENTILATION(HFJV). SA Weisberger, †**1858** 

used to monitor dr(peak minus end expiratory pressure).  $v_{\rm T}$  was measured via a pneumotachometer placed on the expiratory arm of the ventilator and validated with a body plethysmograph. The integrated flow signal was reset by the jet solenoid at onset and end of inspiration. At f of 240 and 480/min there was a signifiend of inspiration. At f of 240 and 480/min there was a signifi-cant inverse relationship between  $T_{\rm I}$  and the  $\Delta P$  required to de-liver constant  $\nabla_{\rm T}({\rm ANOVA~p<.05})$ . At  $T_{\rm I}$  <50ms there was at least a 3-fold increase in  $\Delta P$ . Despite the longer  $T_{\rm I}(62{\rm ms})$  at f=480/min and I:E=1:1,  $\Delta P$  was still increased because the shortened expir-atory time produced marked air trapping. At a rate of 120/min,  $T_{\rm I}$  did not significantly influence  $\Delta P$ . Net air flow at the pneu-potent during the op phone of the int much was in the number notach during the on phase of the jet cycle was in the outward direction indicating lack of gas entrainment. In summary: 1) as  $T_{\rm I}$  was shortened during HFJV higher  $\Delta P$  was necessary to main- $T_1$  was shotched during hist higher Ar was necessary to main-tain  $V_T$  and 2) in this model, gas entrainment did not contribute to the delivered  $V_T$ . Duration of  $T_1$  is therefore critical for optimal  $V_T$  delivery if barotrauma is to be minimized. Further-more, the delivery of humidification and  $0_2$  during HFJV cannot be dependent on gas entrainment. Supp. ALA-Ohio, ALANO

1859 LUNG COMPLIANCE( $C_L$ ) AND EXPIRATORY TIME( $T_E$ ) INFLUENCE AIR TRAPPING(AT) DURING HIGH FREQUENCY JET VENTILATION(HFJV). SA Weisberger, WA Carlo, JM Fouke, RL Chatburn, T Tillander, RJ Martin. CMRU, Dept.Feds, Cleve, OM At high ventilating frequencies,  $T_E$  may be insufficient for passive lung deflation, resulting in AT. To document AT during HFJV, we monitored inadvertent positive end expiratory pressure (inadv PEEP) with a 16 gauge catheter placed distally in a 3.0 ET tube and change in functional residual capacity( $\Delta$ FRC) with a body plethysmograph. In 9 adult rabhirs Tr was varied by emp body plethysmograph. In 9 adult rabbits, T<sub>E</sub> was varied by em-ploying frequencies(f) of 120, 240 and 480/min at I:E ratios of ploying frequencies(f) of 120, 240 and 480/min at I:E ratios of l:1,1:3,1:5 and 1:9 in lungs of normal(NC<sub>L</sub>) and decreased( $+C_L$ ) compliance (after saline lung lavage). Peak airway pressure was varied to achieve a wide range of tidal volumes(V<sub>T</sub>). At f of 240 and 480/min and V<sub>T</sub> of 2.5-3cc/kg (approx dead space) there was a significant effect(analysis of variance) of T<sub>E</sub> (p<01) and lung compliance (p<.01) on inadv PEEP(see table).  $\Delta$ FRC was ac-cordingly influenced by T<sub>E</sub> and C<sub>L</sub>.

| freq:480/min        |          | Inadv PEEP(cmH20),n=9 |          | $\Delta$ FRC(cc/kg), n=4 |      |  |
|---------------------|----------|-----------------------|----------|--------------------------|------|--|
| T <sub>E</sub> (mse | ec)(I:E) | NCL                   | +CL      | NCL                      | +CŢ. |  |
| 62                  | (1:1)    | 12±4                  | $10\pm4$ | 25±6                     | 8±8  |  |
| 94                  | (1:3)    | 7±3                   | 5±2      | 17±4                     | 5±4  |  |
| 104                 | (1:5)    | 6±2                   | 4±2      | 14±6                     | 3±3  |  |
| 112                 | (1:9)    | 5±2                   | 3±2      | 9±5                      | 3±3  |  |
|                     |          |                       |          |                          |      |  |

At f of 120/min, inadv PEEP occurred almost exclusively at an At 101 120/main, induction of contract and the second second at an at 125 of 11. AT further increased at greater  $V_T$ . We conclude that AT during HFJV is accentuated with 1)lungs of normal compliance, 2)decreasing  $T_E$  and 3)increasing  $V_T$ . Therefore, as  $C_T$  improves with resolving lung disease, appropriate changes in  $T_E$  and f must be made to prevent air trapping. (ALA, Ohio)

PULMONARY ASSESSMENT OF CHILDREN AFTER CHLAMYDIAL POLMONARY ASSESSMENT OF CHILDREN AFTER CHLAMYDIAL
PNEUMONIA OF INFANCY. Steven G. Weiss, Richard W. Newcomb, Marc O. Beem. The University of Chicago
Hospitals and Clinics. Department of Pediatrics. Chicago. We evaluated the pulmonary status of 18 children 7 to 8 years after their hospitalization for chlamydial pneumonia of infancy.
Pulmonary function tests (PFT) and respiratory questionnaire Pulmonary function tests (PFT) and respiratory questionnaire results on this group (CT) were compared to those of a control group (CR) comprised of 19 age, race and sex comparable children from the same community, or to values that other investigators have reported for normal children. Significant limitations of expiratory airflow were found in the CT group mean values compared to CR group (FEV<sub>1</sub> p = .01; FEV<sub>1</sub>/FVC p  $\lt$  0.03; PEF p = .04; and FEF<sub>2.757</sub> p = .009). CT group plethysmographic results revealed abnormally elevated volumes of trapped air (ightarrow 2 SD from reference means) present in 3 of 18 FRC and 13 of 18 RV/TLC ratios. These obstructive patterns were responsive to inhaled isoproterenol. Similarly, the CT group also had a significantly greater number of children with physictan-diagnosed asthma than the control group (6/18 vs 1/19, p  $\lt$ .03). The obstructive PFT abnormalities could not be accounted for by recognized risk factors such as exposure to smoking at home The obstructive FFT abhormatifies could not be accounced for by recognized risk factors such as exposure to smoking at home (11/18 vs 12/19 p = NS) or family history of atopy (6/18 vs 4/19 p = NS). Our results show that chlamydial pneumonia of infancy is associated with PFT and respiratory symptom abnormalities 7 to 8 years after recovery from the acute illness.