PRESSURE-FLOW BEHAVIOR OF A BRONCHO-PLEURAL FISTULA DURING MECHANICAL VENTILA-TION. Julio Pérez Fontán and Andrea O. Ray (spon. by G. Lister). Yale Univ. Depart. of Pediatr. New Haven, CT.

For a broncho-pleural fistula to resolve, the impedance of the fistulous pathway must increase until flow ceases and the leak seals. We examined the effect of ventilatory pattern on bronchopleural pathway impedance in 7 anesthetized, paralyzed 6-week old lambs. A wedge of subpleural lung tissue was removed and a peripheral bron-chus (ID < 1mm) sectioned to create an artificial broncho-pleural pathway. We estimated the pathway's average impedance (\overline{Z}) from the mean pressures in a regional bronchus measured with a retrograde catheter (\overline{P}_{br}) and at the pleural inlet of the chest tube draining the pleural space $(\overline{P}_{\text{Dut}})$, and the mean flow through the close studies $(\overline{V}_{\text{leak}})$ as $\overline{Z} = (\overline{P}_{\text{Dr}} - \overline{P}_{\text{out}})/\overline{V}_{\text{leak}}$. Tidal volume (V_T) was measured with a jacket plethysmograph calibrated in absolute volume by He dilution. We determined \overline{Z} while ventilating the lungs with rates of 20, 40, 60, and 80 breath/min and inspiratory times of 1.0, 0.6, 0.3, and 0.2 s. FRC, V_T , and mean lung volume were first kept constant at 40, 5, and 2.5 ml/kg. In a second experiment, V_T was adjusted to maintain P_ACO_2 constant, without changing FRC or mean volume. \overline{Z} ranged from 0.172 to 5.930 cm $H_2O/ml/s$ and was independent of ventilatory rate even when V_T was Increased. Repeated stepwise inflation/deflation maneuvers revealed a biphasic static \overline{Z} -lung volume relationship, with \overline{Z} decreasing at low volumes and increasing slowly at volumes greater than FRC. Our results indicate that \overline{Z} is 1) unaffected by ventilatory pattern at conventional ventilatory pattern at conventional ventilatory rates and 2) sensitive to lung volume only at low volumes. At high lung volumes, peribronchial pressures may exceed the pressure inside the fistula, thus creating a choking effect which increases \overline{Z} . The same effect may occur if \overline{P}_{out} is lowered by applying negative pleural pressure.

> HIGH FREQUENCY VENTILATION DECREASES THE BAROREFLEX. Erik A. Hagen, George A. Gregory. Univ. of Califor-nia, CVRI and Anesthesia Dept., San Francisco. Effects of high frequency ventilation (HFV), such

†165 as apnea, are mediated by the autonomic nervous

tem. To further define these effects, we studied an important autonomic reflex, the baroreflex, during HFV and con-ventional ventilation (CV) of 5 young adult male rabbits anesthetized with chloralose and paralyzed with metocurine. To keep FRC constant during change from CV to HFV, we continuously Keep FRC constant during change from CV to HFV, we continuously measured FRC by jacket plethysmograph, adjusting jet ventilator driving pressure as necessary. Ventilation (P_aCO_2) was also kept constant by adding CO_2 to inspired gas as needed. We tested the baroreflex by increasing blood pressure (BP) with phenylephrine and plotting change in BP against change in R-R interval obtained from EKG. Measurements were made during baseline CV (pressure 25/4, rate 45), HFV (rate 600), and after return to CV. PD CV. BP and heart rate were not affected by changes in ventilator frequency. The slope of the line of BP vs R-R interval was 31% to 62% less during HFV compared to CV (P<0.04). The slope of the baroreflex returned to baseline when CV was re-instituted. Since FRC and ventilation were constant, we speculate the decrease in baroreflex results from rapid pressure changes (dp/dt) during HFV stimulating pulmonary mechanoreceptors causing centrally mediated baroreceptor inhibition. We conclude that close cardiovascular monitoring of patients during HFV is warranted, especially when there is cardiovascular compromise (shock, hypovolemia, sedation or anesthesia). Neonates are especially at risk when the baroreflex is diminished because their relatively fixed stroke volume makes cardiac output heart rate dependent.

ESOPHAGEAL ATRESIA WITH TRACHEO-ESOPHAGEAL FISTULA--A REVIEW OF CRITICAL CARE, ANESTHETIC AND SURGICAL MANAGEMENT. <u>Debra Y. Hamilton, Kumar G. Belani, Arnold</u> <u>S. Leonard, John E. Foker, Theodore R. Thompson</u> (Spon. by Milliam Krivit) University of Minnesota Hospital,

Minneapolis, MN We reviewed the critical and perioperative management of 57 newborns and infants with esophageal atresia (EA) and tracheo-esophageal fistula (TEF) during the last 16 years (1970-1985). The common anomaly of proximal EA with distal TEF was present in 54; 3 had the less common H type fisutla. The major preoperative problem was pneumonia + atelectasis. There was no relationship between intubation technique and subsequent ventilatory problems. Even though <u>serious</u> ventilatory problems after an <u>initial</u> gas-trostomy have been reported, no such problem was found in this review. Invasive monitoring (arterial + central venous lines) review. Invasive monitoring (arterial + central venous innes) was used more often in recent years. When compared to previous reports the overall one-year mortality was significantly better for babies weighing < 2.5 kgs (< 2.0 kgs = 50%; 2-2.5 kgs = 87%). Survival for babies > 2.5 kgs continues to be good (97%). Improved survival maybe related to better monitoring, improved surgical technique, cardiac and nutritional support. Prophylactic esophageal dilation of the repaired atretic segment was done routinely and may have decreased the incidence of chronic aspira-tion and mortality. However, there was a difference in mortality and morbidity between primary and staged repairs. With the lat-ter, the mortality and morbidity was 8.9% and 89% respectively. With primary repair only 3.6% and 57%. The patients who had staged repair were of younger gestational age, weighed less and had a higher incidence of preoperative pulmonary problems. FATIGUE OF THE INSPIRATORY MUSCLES OF THE RIB CAGE

IN HUMANS Marc B. Hershenson, Yoshihiro Kikuchi, Mary Ellen B. Wohl, Robert K. Crone, Stephen H. Loring. Harvard Medical School, The Children's Hospital, Departments of Anesthesia and Pulmonary Medicine, and The Harvard School of Public Health, Department of Physiology, Boston, MA 02115. 167

Department of Physiology, boston, MA U2113. Studies on respiratory muscle fatigue have focused on the diaphragm (DIA). We have found, however, that maximal static inspiratory pleural pressure (Pi max) is limited by the strength of the inspiratory muscles of the rib cage (RC). Failure to of the inspiratory muscles of the rib cage (RC). Failure to maintain Pi max during inspiratory endurance tasks might there-fore result from fatigue of the RC muscles rather than DIA. To fore result from fatigue of the RC muscles rather than DIA. To test this hypothesis, 5 trained subjects repeated maximal inspiratory efforts (duty cycle, 0.5) until Pi max could not be maintained. Subjects performed such efforts with a minimum of abdominal muscle recruitment. Before and after inspiratory efforts, DIA muscle strength was evaluated by measuring the transdiaphragmatic pressure generated during maximal expulsive efforts with an open glottis (Pdi max). (During expulsive efforts the DIA is maximally activated; thus, the Pdi obtained is a true index of DIA strength). Results (x±SD, *p<.01, paired t): $\frac{Before fatigue}{97\pm13} = \frac{After fatigue}{68\pm4*}$ Pdi max (cmH2O) Pdi max (cmH2O)

Pi max (cmH20) 9/±13 Pdi max (cmH20) 189±13 183±20 We conclude that (1) Pi max is not limited by DIA strength, and (2) because DIA strength was unchanged, failure to maintain Pi max in these subjects appeared to be due to fatigue of the inspiratory muscles of the RC. RC muscle fatigue may be important in the pathogenesis of respiratory muscle failure. Supported by HL 07633.

PENTOXIPHYLLINE (PENT) AND HYPOTHERMIA (HT) REDUCE THE SUSCEPTIBILITY TO HYPOXIA IN ANESTHETIZED DOGS Marc B. Hershenson, James A. Schena, Paul A. Lozano, <u>Robert K. Crone</u> (spon. by Ann R. Stark). Harvard Medical School, The Children's Hospital, Departments of Anesthesia and Pulmonary Medicine, Boston,MA 02115 •168

Hypothermia (HT) has been used to reduce 02 consumption ($\dot{V}02$) Hypothermia (HT) has been used to reduce 02 consumption (\dot{V} 02) in critically ill children. HT, however, does not reduce the critical level of Pa02 required to maintain \dot{V} 02, because cardiac output (CO) and 02 extraction (02 ext) are also decreased. We hypothesized that PENT, by increasing RBC deformability and vaso-dilation, would increase CO and 02 ext during HT, reducing the critical levels of 02 delivery (D02) and Pa02. 15 adult beagles were exposed to hypoxic hypoxia during normothermia (38° C), HT (30° C) and PENT plus HT. We measured Hb, %sat, p02, CO, blood viscosity and \dot{V} 02, and calculated D02 (CO-Ca02), SVR, PVR, 02 ext and critical D02 and Pa02. 38° C 30° C PFNT+ 30° C

	38°C	30°C	PENT+30°C
VO2(cc∕kg/mn)	6.6±0.5	3.5±0.4**	3.7±0.5**
CO(cc/kg/mn)	144±46	86±22*	140±35
SVR(dyn/s/cm ⁵)	3178±620	5485±687*	2735±642
critical DO2	13.0	7.1	5.3
critical PaO2	18	17	10
02 ext	.54	.39	.78
Recause of its	Affects on CO	and 02 ovt UT alone	did not noduos

Because of its effects on CO and O2 ext, HT alone did not reduce critical PaO2. PENT reversed HT-induced changes in CO and O2 ext without changing VO2, allowing reductions in critical DO2 and PaO2. When combined, PENT and HT reduce the susceptibility to hypoxia in dogs by reducing VO2 while maintaining DO2 and O2 ext. PENT and HT may be useful in severe, intractable hypoxemia. Supported by HL 07633.

EFFECT OF HEMOGLOBIN CONCENTRATION [Hb] ON CRITICAL (crt) CARDIAC OUTPUT (CO) AND O TRANSPORT (SOT). Felipe Heusser, Andrea O. Ray, John T. Fahey, George Lister. Yale Univ Sch Med, Dept Ped,

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We previously reported that 4 week old lambs have the least tolerance to reduction in CO. We hypothesized that this the least tolerance to reduction in CO. We hypothesized that this was due to their relative anemia, because the low [Hb]: 1) decreased SOT (SOT=COxCaO₂) or 2) impaired O₂ extraction (extr). To test these hypotheses, we lowered CO in 4 week old lambs by incremental stepwise inflation of a balloon tipped catheter (Foley) in the right atrium. We determined the point where O₂ consumption (VO₂) decreased or arterial lactate increased, which defined: crt CO₂ or t fractional CO (crtFCO=crtCO/basalCO) and crt SOT. We performed 12 experiments in 5 lambs at various [Hb], obtained by isovolemic exchange transfusions with sheep packed red cells or plasma. The data (mtSD) are: Intermediate intermediate (mtSD) are the planet of the cell or the construction of the cell or the cel

[***]		Duouroo	Dusur 109	01001	cin co
(g7df)		(ml/min/Kg)	(ml/min/Kg)	(ml/min/Kg)	
7.6±.37	5	155±28.5	7.5±1.3	8.1±1.2	0.53±0.12
10.5±.3	5	120±12.4	6.7±0.8	8.2±3.2	0.45±0.12
13.7	2	147.5	8.3	8.8	0.39
(13.4 - 14.0)		(141-154.3)	(7.8-8.7)	(7.4 - 10.2)	(0.29 - 0.38)

These findings further confirm the postulate that the crt SOT is These findings further contribution the postulate that the crit SOT is independent of the manner in which the SOT is altered (no difference in the crt SOT among the 3 groups). Furthermore the anemic lambs had the least tolerance for reduction in CO, because of decreased O_2 carrying capacity rather than impaired maximal O_2 extr (no difference in VO₂/crtSOT). These data have important clinical implications for maintaining SOT in subjects with limited CO. with limited CO.

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