1780 "INADVERTENT PEEP" WITH THE USE OF A CONVENTIONAL VENTILATOR IS UNDETECTED BY A PROXIMAL AIRWAY PRESSURE MONITOR.

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"Inadvertent PEEP" develops during high frequency ventilation. This effect is not as well recognized using a conventional ventilator at high frequencies. A neonatal lung model with respiratory compliance of 3.3 ml/cm H_2O and a pressure tap to measure alveolar pressure (Palv) was ventilated with a Baby Bird through a 2.5 mm ID neonatal ET tube at 20 to 100 breaths per minute (BPM) using a variety of I:E ratios, peak pressures, and end expiratory pressures. Proximal airway pressure (Paw) was measured at the ET tube adaptor. End tidal Palv increased as frequency, peak pressure and I:E ratios were increased even if the ventilator was set at zero PEEP. This inadvertent increase in Palv was not detected by the proximal airway pressure monitor even though the "inadvertent PEEP" (Palv-Paw) was as great as 9.8 cmH_2O at 100 BPM. "Inadvertent PEEP" is the result of insufficient expiratory time at high ventilator rates, and its magnitude is decreased by increasing expiratory time.

				INADVERTENT PEEP	
	Paw PEEP*	Palv		Palv-Paw	
I:E		20 BPM	100 BPM	20 BPM	100 BPM
1:1	0	0	9.8	0	9.8
	4	4	10.6	0	6.6
	8	8.4	15.0	0.4	6.6
1:2	0	0	4.8	0	4.8
	4	4	7.2	0	3.2
	8	8	10.6	0	2.6

*Peak Inspiratory Pressure 24 cmH_2O

1781 MECHANICAL SIMILARITIES OF A HIGH FREQUENCY JET VENTILATOR AND A CONVENTIONAL VENTILATOR.

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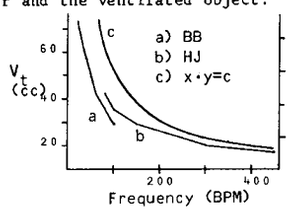
The outputs of a Healthdyne Jet (HJ) and a Baby Bird (BB) ventilator were compared by ventilating a neonatal lung model with physiological resistance and compliance. We measured alveolar pressure (Palv), change in lung volume (ΔLV) and tidal volume (V_T) over the frequency (f) range of each ventilator (80-900 BPM and 20-100 BPM respectively) using a variety of I:E ratios, peak pressures, end expiratory pressures, and flow rates. The relationships of Palv, LV and V_T to f were similar in both ventilators. Palv and LV increased similarly by increasing frequency. This effect was magnified by increasing I:E ratio or flow rate. V_T decreased in both ventilators according to a hyperbolic relationship ($x \cdot y$=constant). A conventional and jet ventilator have very similar mechanical output and pressure and volume relationships over their frequency ranges. This similarity derives in part from similarity of design and in part from interaction between the ventilator and the ventilated object.

	I:E	f	Palv	ΔLV
	1:1	20	3.4	11.1
BB		100	10.6	34.5
	1:2	20	3.6	11.7
		100	7.2	23.4
	1:9	100	1.6	5.2
HJ		900	14.6	47.1
	1:4	100	9.6	31.0
		900	36.0	116.1



†1782 MEASUREMENT OF EXPIRED ETHANE AND N-PENTANE DOES NOT PREDICT OXYGEN INDUCED DAMAGE TO THE LUNGS OF LAMBS.

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Measurement of the rate of excretion of ethane and n-pentane in expired gas has been suggested as a method of detecting oxygen induced injury to the lung. To test this theory, we made daily measurements of pulmonary arterial (PA) and left atrial (LA) pressures, lung lymph flow, the lymph:plasma protein ratio and concentrations of ethane and n-pentane in expired gas in 6 23±3 day old lambs as they continuously breathed 100% oxygen.

Results: Mean ± S.E. (* different $p < 0.05$)

Day in O_2	PA (torr)	LA	Lymph Flow (ml/hr)	Lymph/Plasma Protein	pH	P_aO_2 (torr)	P_aCO_2
First	15±1	2±1	1.63±0.26	0.62±0.02	7.40±0.01	390±8	47±2
Last	16±1	1±1	4.42±0.44*	0.66±0.02*	7.08±0.06*	174±26*	113±11*

(pm/100g/min)	Day 1	Day 2	Day 3	Day 4	Day 5
Ethane	1.46±0.25	1.68±0.33	1.38±0.31	1.52±0.27	1.14±0.39
Pentane	0.47±0.12	0.45±0.10	0.53±0.11	0.57±0.11	0.29±0.10

After 3-4 days in oxygen lung lymph flow and the lymph:plasma protein ratio increased demonstrating that pulmonary microvascular permeability to protein had increased presumably as a result of oxygen induced damage to the endothelium. Daily excretion of ethane and n-pentane did not change throughout the course of the experiment even though all 6 lambs died of respiratory failure from oxygen toxicity. We conclude that the measurement of excretion of ethane and n-pentane in expired gas is not a reliable indicator of oxygen induced damage to the lungs of lambs.

●1783 FUROSEMIDE DECREASES VENTILATION IN NEWBORN RABBITS.

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Furosemide improves lung mechanics in infants with bronchopulmonary dysplasia (BPD). However, in some infants we found that even if lung mechanics improve, $PaCO_2$ rises and ventilation falls during furosemide therapy. Is this due to a furosemide-induced metabolic alkalosis with a secondary respiratory adjustment? Because the acid-base status of BPD patients is complex, we studied normal rabbit pups. For the first 9 days of life, 10 pups received intraperitoneal injections of furosemide (2mg/kg bid) and 10 pups received saline. On day 10, we inserted a catheter into one carotid artery and placed each pup in a plethysmograph. We measured body temperature; blood pressure; arterial gases and pH; serum Na, Cl, K; respiratory frequency and tidal volume. We calculated ventilation ($\dot{V}_E/g$) and base excess (BE) and measured post-mortem static lung compliance (C_L). Variables which were significantly different by unpaired t-test are shown below (means only, $p<.05$):

	Na	Cl	pH	PO_2	PCO_2	BE	$\dot{V}_E/g$	C_L
SALINE	137	99	7.39	88	41	-1	.570	0.100
FUROSEMIDE	126	84	7.46	72	55	+11	.390	0.139

Furosemide treated pups developed a primary alkalosis and a secondary respiratory acidosis; in addition, lung compliance was improved. KCl injection to alkalotic pups increased $\dot{V}_E$ and decreased pH. We conclude that furosemide 1) causes a metabolic alkalosis and reduces ventilation and 2) increases the $PaCO_2$ which reflects changes in acid-base status and not changes in lung function.

●1784 EFFECT OF BREATHING PATTERN ON OXYGENATION DURING SLEEP IN PREMATURES.

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Periodic breathing (PB) occurs commonly in ostensibly healthy preterm infants, yet its significance is unknown. To elucidate the effect of PB on oxygenation, we studied 13 AGA 32 wk infants with a mean BW of 1744 gms at a postnatal age of 7 days. Infants in good condition were selected and morbid conditions such as asphyxia at birth were specifically excluded. Impedance pneumography, R-R interval, $P_{tc}O_2$ and $P_{tc}CO2$ were continuously recorded for up to 8 hrs. The O_2 and CO_2 electrodes were calibrated at a temperature of 43.5 and 44°C, respectively. Sleep state was determined using the behavioral criteria of Prechtl. Periods of regular breathing (RB) and PB were identified for both active (AS) and quiet sleep (QS). PB was defined as repetitive pauses of 3-5 sec. duration separated by 10-15 sec. of RB. After stabilization in each sleep state, the first 2 min. of RB and PB were selected for determination of $P_{tc}O_2$ and $P_{tc}CO_2$ levels. For analysis, samples were taken every 10 sec. and averaged over the 2 min. period. No difference in mean $P_{tc}CO_2$ levels occurred in either breathing pattern or sleep state. $P_{tc}O_2$ was lower during PB than RB in both sleep states. Marked variability of levels occurred during AS with only occasional fluctuations during QS, but there were no differences in the mean levels between AS and QS. Since respiratory rate was reduced in PB, the relative hypoxia during PB under normal conditions is determined by respiratory rate and not by sleep state.

1785 DAY-TO-DAY PNEUMOGRAM VARIABILITY.

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To determine day-to-day variability in respiratory pattern, we have obtained 188 comparisons of pneumograms performed in two successive 24-hour intervals. The respiratory pattern values calculated were total duration of brief apnea (apnea density), periodic breathing episodes, longest apnea, number of apneas > 11 sec, and number of apneas > 15 sec. For Day 1 apnea density (A_6/D%), periodic breathing and longest apnea values of < 0.6%, < 1.5 episodes/100 min and < 20 sec, Day 2 values fell outside that range in 10%, 8% and 1% of comparisons, respectively. The theoretical prediction of Day 2 results based on the observed Day 1 values was assessed by calculation of confidence and prediction intervals. We can be 95% confident that a Day 1 A_6/D% of 0.5% will be less than 1.2% on Day 2, 1.5 episodes of periodic breathing/100 min on Day 1 will be less than 3.2 on Day 2, and a Day 1 longest apnea of 15 sec will be less than 22 sec on Day 2. These prediction intervals are too wide to be clinically useful. Part of this variability, however, is not random but is related to adaptation, a systematic tendency for the Day 2 value to be less than the Day 1 value. For each parameter, the Day 2 range is lower, slope of the Day 2 vs Day 1 regression line is significantly less than one ($p < .01$) and the y intercept is significantly greater than zero ($p < .01$). Knowledge of day-to-day pneumogram variability in general, and of the adaptation phenomenon specifically, should be helpful in interpreting individual pneumogram results and in assessing the clinical usefulness of pneumograms.