25 DECLINE IN RESPIRATORY COMPLIANCE DURING THE FIRST WEEK OF LIFE IN INFANTS ≤ 1000 g.

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Single measurements of respiratory compliance (Cr_s) in premature infants have been reported; however serial recordings have not been published. Monitoring respiratory mechanics could improve ventilation techniques. Using a non-invasive single breath occlusion technique (LeSouef et al: Am Rev Resp Dis 129:521, 1984) Cr_s was measured serially in 10 ventilated infants ≤ 1000 g. 2 infants with very low Cr_s $\leq .25$ ml·cm H₂O⁻¹ died on day 1 from respiratory failure. In a further 2 infants with severe lung disease (Mean airway pressure [MAP] 12-15 cm H₂O on day 2) no change in Cr_s occurred throughout the first week ($.4-.5$ ml·cm H₂O⁻¹). Six infants

showed a striking decline in Cr_s (Table).

No	BW (g)	GA (wks)	Day 1-2	Day 3	Day 5-6	Day 7-8	Max Δ Cr _s	Outcome
1	575	23	.79	.67	.54	.35	.44	BPD
2	550	24	.90	.55	.31	.27	.63	BPD
3	680	24	1.0	.93	.33	.24	.76	BPD
4	750	25	1.0	.93	.54	-	.46	BPD
5	780	26	.52	.52	.32	.22	.30	Died
6	1000	26	1.2	.51	.63	.64	.69	BPD

This fall in Cr_s was also seen in those infants ventilated mainly for apnoea: MAP in cases 1-3 ranged from 3.5-7.2 cm H₂O during the first week, and FIO₂ from .21-.34. We conclude that even modest mechanical ventilation in infants ≤ 1000 g may be associated with an early, rapid and progressive decline in Cr_s.

26 AIRWAYS PRESSURES AND ARTERIAL BLOOD GASES AND pH AT INHIBITION OF INSPIRATORY ACTIVITY DURING POSITIVE PRESSURE VENTILATION. T. Norsted, A. Jonzon and G. Sedin. Dept. of Physiology and Medical Biophysics, Uppsala University, Biomedical Center Uppsala, and Department of Paediatrics, University Hospital, Uppsala, Sweden.

One of the most important characteristics of high-frequency-pressure ventilation (HFPPV) is the almost instantaneous inhibition of breathing when HFPPV is started. With HFPPV spontaneous breathing against the ventilator can be avoided also in neonatal intensive care.

This experimental study in anesthetized cats was made to determine whether the ventilatory pattern in terms of ventilatory frequency, insufflation time and PEEP influences the blood gas level at which breathing is inhibited. The airway pressures were measured at the minute ventilation causing inhibition of inspiratory activity as reflected by the phrenic nerve activity.

The inspiratory activity could be inhibited both at high and at low ventilatory frequencies but a much lower PCO₂ was needed to achieve inhibition at a ventilatory frequency of 15 b.p.m. than at higher frequencies. The larger tidal volumes used in the lower frequency range could evoke inspiratory activity with phrenic nerve bursts at the same rate as the insufflations given by the ventilator. At ventilatory frequencies between 30-120 b.p.m. inhibition could be achieved at a higher PCO₂ still within the normal range. The inhibition of phrenic nerve activity tended to be less stable when PEEP was added during ventilation with a long insufflation period.

CONCLUSIONS: Inhibition and excitation of inspiratory activity is in the first place determined by the chemical regulation of respiration. At low ventilatory frequencies with concomitant very high ventilatory pressures a low PCO₂ is needed to achieve inhibition.

27 REDUCTION OF BAROTRAUMA IN VENTILATED NEONATES BY INCREASE IN VENTILATION FREQUENCY - FIRST RESULTS OF A PROSPECTIVE COLLABORATIVE AND RANDOMIZED TRIAL OF TWO DIFFERENT VENTILATORY TECHNIQUES

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Two ventilatory techniques were investigated prospectively for the production of extraalveolar air leakage (eal) in a collaborative study from 8 children's hospitals (eal: interstitial emphysema, pneumothorax, -mediastinum, -pericardium, -peritoneum). Ventilated four hours old neonates with normocapnia and an oxygen requirement of 40% or more were entered into the study. Within three gestational age classes (<28, 29-30, 31-32 weeks gestation) each subject was randomly assigned either ventilatory technique A or B. A: frequency 60/min, I:E=1:2; ventilation was increased or decreased only by changing ventilatory pressure. B: frequency 30-(40)/min, I:E=1:1-(2:1), ventilation could be increased or decreased by changing ventilatory pressure, frequency and (<40 min) I:E ratio (<2:1). Standardized ap chest x-rays were assessed blind for the presence of eal between the 4th and 24th hour of life and on the 3rd, 5th and 7th day of life. A closed sequential test design was used for the statistical analysis. Results: Pairs without eal =13; pairs with eal =16; pairs with eal using ventilatory technique A =10; pairs with eal using ventilatory technique B =25. By 1/4/85 it was possible to say that ventilatory technique A, which meant a higher ventilation frequency and a lower I:E ratio, causes less eal than B with a probability of error less than 10% and close to 5%.

28 ARE FAST RATES AN EFFECTIVE ALTERNATIVE TO PARALYSIS?

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We have previously demonstrated that infants who actively expire against positive pressure inflation at "conventional" ventilator rates develop pneumothoraces and amongst such infants pancuronium significantly reduces the incidence of air leaks (1) However pancuronium is not without side-effects, therefore this study investigated if fast rates could, as effectively, suppress spontaneous respiration and so possibly prevent pneumothoraces. 17 infants were demonstrated as actively expiring at ventilator rates of 30-40/minute by recording oesophageal pressure, tidal flow and volume simultaneously with ventilator pressure changes. The respiratory response of these infants to ventilator rates of 60 and 120/minute was then studied for 20 minute periods. The results are shown in the table.

	30-40	60	120
response:-			
active expiration:-	17	9	2
"incoordinated resp":-			7
synchrony:-	-	7	5
apnoea:-	-	1	3

The 9 infants who remained actively expiring or had incoordinated respiration at fast rates were subsequently paralysed, only one developed a pneumothorax during ventilation. The 8 infants who breathed in synchrony or were apnoeic at fast rates were not paralysed and remained on increased rates, 4 later developed air leaks. We conclude that fast rate ventilation appears to have only limited success (18%) in suppressing respiratory activity in infants actively expiring against the ventilator. (1) Greenough et al Lancet 1984,i,1-4

29 MYOINOSITOL SUPPLEMENTATION REDUCES SEVERE COMPLICATIONS OF THE RESPIRATORY DISTRESS SYNDROME (RDS).

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Breast milk is among the richest dietary sources of myoinositol (INO). Little if any INO is present in intravenous fluids given to sick preterm infants. Since INO promotes hormone-induced lung maturation in animals (Biochim Biophys Acta 795:67, 1984), effects of INO supplementation (quantity similar to that present in breast milk) in RDS was studied. Sixty preterm infants received double-blind either 150 mg/kg/day of INO or identical quantity of glucose in isotonic solution during ten neonatal days. All infants weighed <2000 g and required mechanical ventilation for at least two neonatal days to treat RDS.

INO supplementation increased the already high levels of serum INO present at birth in RDS, whereas serum INO tended to decrease among controls who received little breast milk. There was no difference in birth weight (1334±289g), gestational age (29.8±2.1w), or severity of RDS at birth among the two groups. However, RDS was less severe in INO group during days 5 to 9. Four INO-infants and 8 controls had bronchopulmonary dysplasia (BPD). Two INO-infants and 7 controls died. The incidence of death and/or BPD was higher in controls than in INO-group (p=0.04). There was no detectable difference in the incidence of intraventricular hemorrhage, patent ductus arteriosus, necrotizing enterocolitis, or infections. The long-term follow-up continues.

According to present results INO supplementation to small preterm infants favorably modifies the course of RDS. INO may accelerate the neonatal development of the lung, and potentiate the effect of glucocorticoid on the lung.

30 NON-PROTEIN CARBON BALANCE, GLUCOSE AND FAT UTILISATION IN TERM INFANTS ON FAT-FREE PARENTERAL NUTRITION.

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Glucose is the main energy source for infants on total parenteral nutrition. In order to study the impact of increasing levels of glucose intake we performed continuous open circuit indirect calorimetry during 4 hrs on 38 AGA term infants. The energy intakes ranged between 13.1 and 105.0 kcal/kg.d and the glucose intakes between 3.5 and 24.1 g/kg.d. Glucose oxidation increased significantly with increasing glucose intake (r=0.90; p<0.001), but was only 80% of the intake at all levels of intake up to 24 g/kg.d. Endogenous fat oxidation decreased significantly with increasing energy intake (r=-0.80; p<0.001) and with increasing glucose oxidation (r=-0.95; p<0.001). The increase in glucose intake resulted in a significant increase in energy expenditure (r=0.82; p<0.001), in non-protein carbon dioxide production (r=0.87; p<0.001) and non-protein oxygen consumption (r=0.66; p<0.001). Non-protein carbon balance was negative at low glucose intakes becoming positive with increasing levels of glucose infusion.

CONCLUSIONS: 1) Indirect calorimetry shows no upper limit in glucose oxidation in newborn infants on fat-free parenteral nutrition. 2) 1/5 of the glucose carbon atoms are stored as fat, glycogen or in an unidentified tissue carbon pool (e.g.: glycerol, cholesterol, non-essential amino acids, glycoproteins, etc.), depending on the level of energy intake. 3) At high glucose intakes, there is increasing lipogenesis which wastes energy and produces more carbon dioxide than it consumes oxygen; this might impose a respiratory stress on the neonate with borderline lung function.