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The effect of PEEP on lungmechanics and bloodgases in mechanically ventilated newborn infants.
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Because little is known about the effect of small PEEP changes on bloodgases and lungmechanics and its impact on respiratory care in the individual patient, we investigated the effect of PEEP of 2, 4 and 6 cm H2O on compliance of the respiratory system(Crs) and bloodgases in 17 mechanically ventilated neonates(GA 28-39 wks , BW 600-3400 g, mainly one day old). The PEEP levels were randomly altered after half an hour, keeping other variables constant. At each PEEP level compliance was measured as described by Simbruner G (J Pediatr 1986; 108:589) and pO2, pCO2 determined from an arterial blood sample. PEEP increased pO2 (10 mm Hg/cm H20). In 11/17 patients a PEEP 2 yielded the highest, and in 8/17 PEEP of 6 cm H2O yielded the lowest Crs. At PEEP settings, yielding the highest Crs [optimal PEEP], Crs was on the average 36% (in individuals up to 100%) higher and CO2 2 mm Hg (in individuals up to 10 mm Hg)lower compared to those PEEP levels, yielding the lowest Crs[worst PEEP]. We conclude that small PEEP changes can significantly influence Crs of individual patients. Setting optimal PEEP in individals would allow gas exchange at lower pressures without much decrease of pO2. In future Crs would have to be referred to the PEEP level at which it was measured.

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Does gas trapping occur during high frequency positive pressure ventilation (HFPPV)?
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HFPPV, using rates of 120/min is associated with improvement in oxygenation, but this beneficial effect has only been demonstrated in non-paralysed infants. Critics claim that, particularly amongst paralysed infants, such fast rates may have an important disadvantage of gas trapping as a result of short expiratory times. The aim of this study was to determine the occurrence of and factors influencing gas trapping at fast rates in infants ventilated during the acute stage of RDS. 2 groups were studied all less than 4 days of age, 20 paralysed (mean gestational age, GA, 29.1 weeks, range 26-36) and 31 non-paralysed (mean GA 29.3 weeks, range 24-36). The infants were ventilated at rates of 60 and 120 breaths/min; (I:E ratio 1:1.2). At each rate the expiratory and inspiratory volumes were measured and reported as the average of 10 regular breaths. No infant had evidence of gas trapping at rates of 60/min. 11 infants (9 paralysed $p<0.004$) had gas trapping at rates of 120/min. Gas trapping was commoner in the more mature infants ($p<0.05$).
We conclude that rates as fast as 120/min can be safely and advantageously used in non-paralysed infants. In paralysed infants, more mature than 30 weeks, however, ventilator rate should be reduced to 60/min if gas trapping is to be consistently prevented.

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AIRWAY PRESSURES DURING IPPV BEFORE AND AFTER LUNG INJURY. Anders Jonzon, Torgny Norsted, Gunnar Sedin. Uppsala University, Departments of Paediatrics and Physiology, Uppsala, Sweden.

Seven cats were ventilated with intermittent positive pressure ventilation (IPPV) at 15 and 60 b.p.m. without and with superimposed oscillations (SO), before and after lung injury. The lung was injured by instillation of xanthine oxidase (xo) into the airways. Comparisons were made at inhibition of inspiratory activity (phrenic nerve).

Before lung injury airway pressures were lower during IPPV without SO at 60 b.p.m. than at 15 b.p.m. ($p<0.02$). Before lung injury SO lowered airway pressure during IPPV at 15 b.p.m. ($p<0.05$).

After lung injury airway pressures were lower during IPPV at 60 b.p.m. than at 15 b.p.m. with and without SO ($p<0.05$). After lung injury SO did not lower airway pressure during IPPV at 15 or 60 b.p.m.

Arterial PCO_2 was always lower after than before lung injury ($p<0.1$ - 0.01) at inhibition of inspiratory activity.

We conclude that SO lowers airway pressure during IPPV at low frequencies but not at high frequencies. After lung injury SO does not lower airway pressures. The lower PCO_2 necessary to inhibit inspiratory activity after lung injury may indicate an altered inhibitory input to the respiratory center.

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High frequency oscillation in treatment of severe hyaline membrane disease and of persistent pulmonary hypertension of the newborn. O. CLARIS, G. PUTET, B.L. SALLE Department of Neonatology, Hopital Edouard Herriot 69437 LYON Cedex 03

Between January 1986 and December 1988, 49 premature infants with severe hyaline membrane disease (HMD) and 28 premature and full-term infants with persistent pulmonary hypertension of the newborn (PPHN) were ventilated with high frequency oscillation (HFO) when conventional ventilation (CV) could no longer ensure sufficient blood oxygenation ($PaO2<6,5$ KPa despite an $FiO2>0,6$) before 12 hours of age. a) In the HMD group, the mean birth weight was 1300 g (range 720-2700 g) and the mean gestational age was 30 weeks (range 26-36 weeks). Thirty-five (71%) babies survived, and intercurrent complications during HFO were the following : pneumothoraces 26%, bronchopulmonary dysplasia 17%, intraventricular hemorrhage 26%, periventricular leukomalacia 6%, necrotizing enterocolities 9% and patent ductus arteriosus 6%. Mean PaO2/PAO2 raised from 0.10 before HFO to 0.26 at 24 hours and to 0.39 at 48 hours of age. b) In the PPHN group, 25 (89%) infants survived. HFO seems to be very effective in the treatment of infants with PPHN and to be encouraging in the treatment of HMD, but randomized controlled studies should be carried out to demonstrate that HFO improves survival in premature infants with HMD.

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THE EFFECT OF CPAP ON THE EXPIRATORY BREATHING PATTERN IN PRETERM INFANTS
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The infant can defend or even elevate dynamically the functional residual capacity by additional respiratory muscles work: 1. early expiratory airflow($\dot{V}$) may be retarded by postinspiraton inspiratory muscle activity and/or laryngeal narrowing; 2. inspiration can start before expiration has been completed to the relaxation volume. To study the effect of CPAP on both phenomena we elevated the airway pressure stepwise in 23 infants (BW 1746±417 g). A computerized bedside $\dot{V}$-volume(V) technique was used for evaluation. In 16 "responders" early expiratory braking decreased and "preterm inspiratory interruption" was postponed at an appropriate CPAP. The linear segment (relaxation line) of the $\dot{V}$-V-loop was lengthened with the expiratory time reaching a maximum. Elevation of CPAP beyond this level again produced a rapid, shallow pattern - often combined with $\dot{V}$-acceleration late in expiration (recruitment of expiratory muscles). These latter signs of excessive airway pressure occured in the remaining 7 infants (nonresponders) already at the lowest CPAP-levels applied during the "titration trials". Respiratory rate without CPAP was different between responders (84±17/min) and nonresponders (46±11). This approach to deciding the CPAP level might reduce the risk of respiratory muscle fatique.

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LUNG VENTILATION AND PERFUSION SCINTIGRAPHY IN THE FOLLOW-UP OF REPAIRED CONGENITAL DIAPHRAGMATIC HERNIA. JL Demarquez, B Lambert, T Lamireau, RI Galperine, AJ Brendel.

To evaluate the effects of congenital diaphragmatic hernia (CDH) and pulmonary hypoplasia on subsequent lung development, we performed lung Xenon 133 ventilation and M.A.A. - Tc 99m perfusion scintigraphies (LVPS) in 16 patients (pts) 2-3 months of age who had had surgical repair of a left CDH within the first 12 hours of life. 10 of these 16 pts were reevaluated at 1-2 years and again at 5-6 years in 6 of them. On the initial lung scintigraphies, ventilation (V) to the hernia side was decreased in 8/16 pts and $\Delta[V_R-V_L]$ averaged 12.6 ± 13.7. In 9/16 pts, there was a trapping of Xenon 133 at the left lung base. Perfusion (P) to the hernia side was decreased in 11/16 pts and $\Delta[P_R-P_L]$ averaged 26.2 ± 23.2. Correlation between V and P was good : $r = 0.70$, $p<0.01$. After 1 year, V to the hernia side became normal except in 2/10 pts ($\Delta V = 5.2 \pm 4.9$), but P to the hernia side remained low in 9/10 pts ($\Delta P = 21.2 \pm 7.9$). After 5 years, V was normal in all of the 6 pts studied ($\Delta V = 2.6 \pm 3.1$); however P to the hernia side was still reduced in 5/6 pts ($\Delta P = 18.6 \pm 5.4$). These results show a progressive improvement and normalization of V and a persisting reduction of P to the lung of the hernia side, suggesting a primary vascular pulmonary hypoplasia in CDH. LVPS is a unique non invasive method that provides quantitative and qualitative informations on P and V, which are very helpful in the follow-up of repaired CDH, during the first years of life when physiologic studies are most difficult to obtain.