

POST-TRANSFUSION DETERIORATION OF PULMONARY FUNCTION IN PRETERM INFANTS. P Sasidharan, R Heimler, J Pelegano. Medical College of WI, Milwaukee County Medical Complex, Dept of Peds, Milwaukee, USA.

We hypothesized that treatment of anemia by packed RBC transfusion in preterm infants will increase the mean inspiratory flow. Pulmonary mechanics were studied using an esophageal balloon and mask during the quiet state in 10 preterm anemic infants (Hct- 28±3) with a mean GA 29.2±2.4 wks at a postnatal age of 42±21 days, before and after (16 hrs) transfusion with 10 cc/kg of packed cells.

	Pre Tx	Post Tx	P
Resp rate	66	74	NS
Compliance (dyn) ml/cmH <sub>2</sub> O	2.80	2.066	<.001
V <sub>T</sub> ml/kg	7.24±1.4	6.96±1.2	NS
Ti sec.	.42	.38	NS
V <sub>T</sub> /Ti	30.7	33.3	NS
Resist. cm H <sub>2</sub> O/l/sec	49.7±27	66.5±26	NS

These results indicate that there is a significant deterioration in lung compliance, after transfusion. The increase in inspiratory impedance may have blunted the anticipated rise in mean inspiratory flow. Whether these changes are related to the volume of blood infused, remains to be determined.

AN INFANT VENTILATOR TO PROVIDE HIGH FREQUENCY VENTILATION NEAR THE RESONANT FREQUENCY OF THE RESPIRATORY SYSTEM. Andreas Schulze, Peter Schaller, Bernd Behrhardt, Dieter Gayrek (spon. by Hans Versmold), Med. Academy, Dept. Pediatrics, Dresden DDR.

By a microcomputerized ventilator system with feedback loops for flow( $\dot{V}$ ) and pressure(P) it was possible to generate a positive P during inspiration and a negative P during expiration in a constant proportion to the instantaneous  $\dot{V}$  of spontaneous breathing. The ratio P/ $\dot{V}$  represents a negative ventilator resistance(NVR), is adjustable (0-15 kPaxs/l) and produces a resistive unloading of the respiratory system. We found a strong linear correlation between the NVR and the resultant drop in transpulmonary resistance (39 trials in 8 rabbits after intra- or extrapulmonary airway obstruction,  $r=0.93$ ,  $p<0.001$ ). Similarly, there was a close relationship between the NVR and the decrease in inspiratory resistive work per tidal volume of spontaneous breathing ( $n=39$ ,  $r=0.85$ ,  $p<0.001$ ). If NVR exceeded the total resistive load of the respiratory system, the combined ventilator-ETtube-patient-system started to oscillate continuously near its resonant frequency(RF) as it was no longer damped. Oscillatory amplitudes and mean airway pressure were controlled independently of each other. This new principle proved useful for high frequency ventilation in rabbits with normal ( $n=5$ , RF=4-6Hz) and surfactant deficient lungs ( $n=5$ , RF=6-10Hz).

ELASTASE-PROTEINASE INHIBITOR IMBALANCE IN PROLONGED VENTILATION (PV) OF VLBW INFANTS ± BPD. H.Walti\*, P.Saugier\*, L.Gerbault\*\*, C.Tordet\*\*\*, NICU & Biochimie\*\*CHU Cochin Port-Royal Paris; Biologie Cellulaire CNRS\*\*\*, Ivry, France.

Acute imbalance between elastase (el) and  $\alpha_1$ -proteinase inhibitor ( $\alpha_1$ -Pi) may contribute in RDS and BPD; persistent imbalance in PV of VLBW infants has not been addressed. Weekly (4-8 wks) serum (S) and bronchoalveolar (Bal) samples were taken in 14 VLBW infants ventilated from birth: 9 had BPD (5 RDS, 4 others); 5 controls had no BPD (apnea). G.A. and B.W. were similar ((29.8-29.8 wks; 1180-1104 g).  $\alpha_1$ -Pi,  $\alpha_2$ -macroglobulin ( $\alpha_2$ -M), albumin (Alb) were measured in S and Bal by immunonephelometry, free el-activity by SAPNA in Bal. S and Bal results were similar at all times in controls and were pooled. Statistical study: ANOVA; \* $p<0.05$ ; \*\* $p<0.25$ ).

	Controls	BPD 4 w	5 w	6 w	7 w	8 w
S $\alpha_1$ -Pi g/l	2.1	2.5	2.5	2.6*	2.3	2.
S $\alpha_2$ -M g/l	3.	3.5	3.9*	3.8**	3.6	2.7
Bal $\alpha_1$ -Pi/Alb $\mu$ g/mg	116	71	72	79	69	89
Bal $\alpha_2$ -M/Alb $\mu$ g/mg	84	74	66	71	72	73
Bal Alb mg/l	35	75*	55	49	87	46
Bal el/2 $\alpha_1$ -Pi ng/2 $\mu$ g	0.2	0.6	0.5	0.6*	0.6**	0.4**

These results suggest a local destruction of  $\alpha_1$ -Pi and  $\alpha_2$ -M, and a persistent imbalance (increased el/ $\alpha_1$ -Pi molar ratio).

POSTNATAL CHRONIC HYPOXIA CHANGES THE OXYGEN SENSITIVITY IN THE CAROTID BODIES. Jens B. Grøgaard, Urban Selstam, Jean-Michel Hascoet and Hakan W. Sundell, Vanderbilt Univ., Dept. of Peds, Nashville, TN (Spon. by HD22712-01)

The ventilatory response to acute hypoxia is mediated mainly by the carotid body (CB) chemoreceptors. These are active in the last trimester of the fetal lamb. The normal rise in PaO<sub>2</sub> occurring at birth silences the CB during the first postnatal days before the postnatal reset of O<sub>2</sub> sensitivity has occurred. We hypothesized that the postnatal reset mechanism of the CB oxygen sensitivity can be reversed by longterm exposure to hypoxia. Minute ventilation (Vmin) was studied in 6 lambs at 2 weeks of age during normoxia and during hypoxia (FiO<sub>2</sub> 0.14). The results were compared to Vmin after 8 days in chronic hypoxia (FiO<sub>2</sub> 0.14). Vmin decreased 53% during chronic hypoxic exposure compared to Vmin prior to hypoxia. The acute hypoxic ventilatory response to a change in FiO<sub>2</sub> from 0.21 to 0.14 was decreased compared to that prior to chronic hypoxia (+3% vs +53%,  $p=0.002$ ). The hypoxic ventilatory response after chronic hypoxia corresponds to our previously reported hypoxic ventilatory response to CB denervated lambs. We conclude that the decreased hypoxic ventilatory response demonstrated is due to a reversal of the CB oxygen sensitivity. This finding might have implications for the ability to abort apnea in infants at a time period when CB is an important factor in the control of breathing.

CHEMOREFLEX RESPONSES TO ALTERNATIONS OF FiO<sub>2</sub> IN THE NEWBORN INFANT.

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We wondered whether infants would respond to alternations of inspired oxygen fraction (FiO<sub>2</sub>) with an alternating reflex respiratory response. Five infants born at term and aged 2-3 days were studied in quiet sleep. Respiration was measured by inductance plethysmography (Resptrace). Respiratory variables were measured on-line by a computer, which also controlled the delivery of inspired gas at 1 l/min via a nasal catheter, switching between two lines supplying air or a hypoxic gas (FiO<sub>2</sub> 0.16) at the start of alternate expirations. Thus 2-breath alternations of air and hypoxia were delivered for 50-100 breaths (test runs). For control runs air was delivered in both gas lines. Alternations in respiratory variables were quantified for the second of each pair of breaths using a modification of the method of Metias et al (1981). The results showed a significant increase in the alternation of tidal volume, frequency and ventilation during test runs in comparison to control. Thus newborn infants respond to 2-breath alternations in FiO<sub>2</sub>, presumably via a peripheral chemoreceptor reflex. We are currently developing the method for the assessment of peripheral chemosensitivity.

References:

Metias E.F., et al (1981). Pflugers Arch. 389, 243-250.

FAMILIAL SEX CHROMOSOMAL MOSAICISM. Richard C. Juberg, Daniel J. Holliday, and Victoria S. Hennessy. Wright State University, School of Medicine, Departments of Pediatrics and Obstetrics/Gynecology, Department of Biological Sciences, and Ultrachem Biomedical Laboratories, Dayton, OH, U.S.A.

Familial mosaicism has rarely been reported. Its recognition poses problems in prognosis, especially prenatally. A 36-year-old white woman, gravida 1, following Clomid stimulation had an amniocentesis at 19 weeks. Of 65 spreads, 82% were 45,X and 18% 46,XX. A second amniocentesis 3 weeks later showed 73% 45,X and 27% 46,XX in 37 spreads. Thorough genetic counseling with discussion of the Turner mosaic phenotype preceded a decision to continue the pregnancy. At term the female infant had normal mensurations, no stigmata of the 45,X syndrome, and blood study showed 38% 45,X, 60% 46,XX and 2% 47,XXX in 40 spreads.

The phenotypically normal mother had blood studies 2 months apart: (1) N = 41 with 10% 45,X, 83% 46,XX, and 7% 47,XXX; (2) N = 100 with 5% 45,X, 94% 46,XX, and 1% 48,XXXX. The phenotypically normal maternal grandmother, 74, showed 5% 45,X, 90% 46,XX, and 5% 47,XXX in 80 spreads, and the phenotypically normal maternal uncle, 39, showed 2.5% 45,Y, and 97.5% 46,XY in 40 spreads.

Thus, 3 generations of females showed sex chromosomal mosaicism. The possibility of familial mosaicism reaches critical importance in interpreting prenatal mosaicism.