

† 1798 BRONCHIAL HYPERREACTIVITY IN LONG TERM SURVIVORS OF THE NEONATAL RESPIRATORY DISTRESS SYNDROME (RDS). Ian B. Maclusky, Joshua Zarfin, Karen Pape and Henry Levison. The Hospital for Sick Children, Department of Pulmonary Medicine, Toronto, Ontario, Canada.

Recent reports have shown residual bronchial hyperreactivity in long term survivors of bronchopulmonary dysplasia and Wilson Mikity syndromes. Our aim was to extend these studies to survivors of uncomplicated RDS. The study group consisted of 7 males and 7 females born between 1974 and 1976. Measurements of static and dynamic lung volumes were made and a methacholine challenge (MCH) was performed on each child. Expiratory flow rates for the total group were statistically significantly reduced ($p < .01$). Pulmonary function was significantly lower in the MCH positive group when compared with the MCH negative group. There was a positive correlation between duration of IPPB and log dose MCH, $R = -0.78$, $p = 0.02$. There was no significant differences between either group for age, sex, height, weight or age at onset of mechanical ventilation. The MCH positive group had received a significantly longer duration of IPPB during the course of the neonatal respiratory failure. Thus, even in survivors of RDS without major sequelae, there is evidence of residual airway hyperreactivity and abnormal pulmonary function. We consider this increased airway reactivity and reduced pulmonary function to be one of the risk factors in the development of chronic obstructive pulmonary disease later in life.

1799 EMERGENCY MANAGEMENT OF ASTHMA: FREQUENT LOW DOSE NEBULIZED SALBUTAMOL. Colin Robertson, Freda Smith, and Henry Levison. The Hospital for Sick Children, Department of Pulmonary Medicine, Toronto, Ontario, Canada.

The bronchodilator response to nebulized salbutamol in stable asthmatics reaches a peak at 20-30 mins and is maintained for 60 mins. However, clinical observations suggest that in acute asthma, the duration of this peak effect is shorter. The aim of this study was to examine the effect of frequent administration of low doses of salbutamol during the initial management of acute asthma. 22 children aged 6-17 years who presented to the Emergency Department with acute asthma were assessed by measurement of FEV₁ initially and at 20 minute intervals for 2½ hours. All children received an initial dose of 0.15 mg/kg salbutamol in 2 ml of normal saline via a nebulizer. Thereafter, those in group A (8 patients) received the same dose at 60 min. intervals for 2 further doses and those in group B (14 patients) received 0.05 mg/kg salbutamol at 20 min. intervals for 6 doses. Mean FEV₁ (% predicted) for both groups were similar initially (A=33, B=29) at 20 min. (A=46, B=45), and at 2½ hours (A=54, B=57). FEV₁ in group A deteriorated between doses and maximal bronchodilation was not achieved until 2½ hours. Whereas FEV₁ in group B was maximal at 60 min. and failed to show further improvements in spite of additional medication. Air flow limitation in acute asthma is multifactorial and it would appear that maximal bronchodilator effect of salbutamol can be achieved by 60 minutes and maintained by frequent administration of small doses.

1800 CAN RESPIRATORY MUSCLE STRENGTH AND VITAL CAPACITY SERVE AS INDICES FOR THE DEVELOPMENT OF HYPERCAPNEIC RESPIRATORY FAILURE IN DOUCHEENNE'S MUSCULAR DYSTROPHY? Amir Szeinberg, Sandra England, Catherine Mindorff and Henry Levison. Department of Pulmonary Medicine, The Hospital for Sick Children, Toronto, Ontario, Canada.

Patients with muscular dystrophy (MD) have proximal myopathy and develop a progressive restrictive defect in lung function leading to respiratory failure. A group of 11 wheelchair bound MD patients, without complicating lung disease was studied to determine whether a reduction of vital capacity (VC) and respiratory muscle strength (RMS) below 55% and 30% (of predicted) respectively was associated with hypercapnic respiratory failure. These criteria were established by Braun et al who studied another group of proximal myopathy patients (Thorax 1983; 38:616). Maximal expiratory and inspiratory pressures were measured using the technique described by Black and Hyatt (ARRD 1969; 99:696) and were expressed as percentage of predicted (% MEP; % MIP respectively). Percentage RMS was the mean of % MEP and % MIP. Despite significant reduction in VC (39 ± 9) and % RMS (33 ± 13) arterialized Pco₂ was in the normal range (40 ± 3 mmHg). This group differed from the one studied by Braun et al in that the % MEP was significantly reduced in comparison to % MIP ($P < 0.001$) suggesting a more prominent weakness of the expiratory muscles. In conclusion, the criteria of VC < 55% and RMS < 30% for developing hypercapnea should not be applied to patients with non uniform distribution of respiratory muscle weakness.

1801 THE EFFECT OF CHANGING RATE AND I:E RATIO USING DIFFERENT HIGH FREQUENCY JET VENTILATORS. Patrick K. Lewallen, Mark C. Mammel, Margaret J. Gordon, and Stephen J. Boros, Children's Hospital, St. Paul, MN

Eighteen healthy cats were anesthetized, paralyzed, and ventilated with either the IDC VS600 or Bunnell Lifepulse (BLP). Mean airway pressure (Paw) and rates were constant as I:E increased from 1:8 to 1:1. Next, Paw and I:E were constant as rates increased from 100-600 bpm. The two machines behaved similarly in terms of I:E and quite differently in terms of rate. As I:E increased, both ventilators maintained normal pH and PCO₂ values until 1:2, then hypercarbia and acidemia developed. As rates increased, the VS600 maintained normal pH and PCO₂ values until 250 bpm; the BLP did so until 600 bpm. Above these rates, hypercarbia and acidemia developed. At all rates and I:E ratios, AaDO₂ were higher with the BLP; Paw was higher with the VS600, but not significantly so.

	BLP - 200 bpm - VS600		BLP - 600 bpm - VS600	
pH	7.47 ± 0.14	7.33 ± 0.1	7.38 ± 0.12	7.04 ± 0.09*
PCO ₂	21 ± 9	25 ± 6	28 ± 10	75 ± 13*
AaDO ₂	375 ± 214	68 ± 41*	446 ± 198	90 ± 35*
Paw	3.6 ± 3.7	7.5 ± 3.2	4.3 ± 3.1	7.5 ± 3.3

*P < 0.01

Conclusions: 1) The VS600 produced better arterial oxygenation and somewhat higher Paw. 2) The BLP produced better ventilation over a wider range of rates. 3) Different high frequency jet ventilators perform differently. The conclusions from the studies of one system cannot, and should not, be applied to any other.

† 1802 ALKALOSIS ATTENUATES HYPOXIC PULMONARY VASOCONSTRICTION IN NEONATAL LAMBS. RK Lyrene, KA Welch, A Dew, JB Phillips (Spons. by G Cassidy) Univ. of Alabama in Birmingham School of Medicine, Dept. of Pediatrics Birmingham, AL 35294.

Hyperventilation (respiratory alkalosis) is an important treatment for persistent pulmonary hypertension in neonates. The precise way that hyperventilation attenuates hypoxic pulmonary vasoconstriction is unclear. We studied the effect of alkalosis on hypoxia-induced pulmonary vasoconstriction in 13 acutely instrumented neonatal lambs. We specifically examined the relative roles of a metabolic alkalosis (MA) vs a respiratory alkalosis (RA). After stabilization in each experimental situation, the lambs were made acutely hypoxic by a 1-minute inhalation of 5% O₂-95% N₂. The control pulmonary vasoconstrictor response to hypoxia was measured at both the beginning and end of each experiment and was compared to the responses observed after hyperventilation (RA) and after bicarbonate infusion (MA-normal PaCO₂). The table gives the mean changes before and during hypoxia.

	Control 1	RA	MA	Control 2
ΔPpa	11±4	8±6*	9±3	11±6
ΔPVR	16±7	11±9*	8±3**	15±6

$\bar{x} \pm SD$, ΔPpa = mm Hg, ΔPVR = mm Hg. L⁻¹ min, *p<.025, **p<.005.

Data consider different from control 1 when p<.05 by 2-way ANOVA.

Hypoxic pulmonary vasoconstriction was significantly milder whenever the animal was alkaleotic - regardless of whether the alkalosis was respiratory or metabolic. Although the rise in PVR was smaller during MA than during RA, there was no significant difference between the effects of the two types of alkalosis. In summary, the elevated pH rather than decreased PaCO₂ during hyperventilation appears to be the major factor in blunting hypoxic pulmonary vasoconstriction.

1803 DEXAMETHASONE REDUCES VENTILATOR DEPENDENCE IN BRONCHOPULMONARY DYSPLASIA. Mark C. Mammel, Claudia L. Nachtsheim, Michael Coleman, and Stephen J. Boros, Children's Hospital, St. Paul, MN

Eight ventilator-dependent infants with clinical and radiographic BPD (birth weight 924 ± 118 grams; age 22 ± 7 days) were treated with IV dexamethasone (Dex), 0.5 mg/kg/day, for 7 days. Dex was then tapered over 2 weeks and discontinued. These infants were compared to 8 controls (birth weight 986 ± 138 grams) with improving lung disease. At study entry, treated infants had significantly increased ventilator requirements compared to controls at 14 days. Controls were extubated at age 13.3 ± 14 days. FiO₂, rate, peak inspiratory pressure (PIP), and alveolar arterial oxygen differences (AaDO₂) were compared before and after 7 days of Dex and to controls at age 28 days, using two-tailed Student-t test.

	FiO ₂	Rate (bpm)	PIP (cm H ₂ O)	AaDO ₂ (torr)
Pretreatment	0.43 ± 0.12*	18.1 ± 5.6	16 ± 1.7	172 ± 72*
Dex (7 days)	0.32 ± 0.09	2.5 ± 3.8	5.4 ± 7.5	103 ± 51*
Control (28 days)	0.25 ± 0.04	1.5 ± 3.2	3.75 ± 7.0	50 ± 19

*P < 0.05 +P < 0.01

All infants were extubated 6.1 ± 2 days after Dex treatment. Compared to 28 day controls, treated infants had higher AaDO₂ ($P < 0.05$). FiO₂, ventilator requirements, and infection rates were similar in both groups. At one year of age, all Dex treated infants were alive, well, living at home, receiving neither oxygen nor diuretic therapy. Conclusion: Early Dex therapy significantly reduces ventilator dependence and may improve prognosis in developing BPD.