1682 AEROSOL INHALATION TECHNIQUE FOR ASTHMATIC CHILDREN

Haesoon Lee (Spon. by Lawrence R. Shapiro). New York Medical College, Lincoln Hospital Center, Department of Pediatrics, Bronx, NY

42 children with asthma who had been taking aerosol medication from canister nebulizers for longer than 6 months were evaluated for aerosol inhalation technique. Proper inhalation technique is a simple procedure. Patient should close lips around the mouthpiece of the nebulizer and exhale first. As the canister is actuated he should inhale deeply by mouth and breathhold for a few seconds before exhaling. The mouthpiece of a canister nebulizer was modified so that actuation of the canister and inhalation through the mouthpiece could be recorded on paper. The age range of the patients was 7 to 15. They were either on bronchodilator or steroid aerosols or a combination of both. Of the 42 tested, 24 had correct technique and 18 (43%) did not. 11 simply squirted the aerosol into the mouth without inhaling. 6 inhaled first, then actuated the aerosol. One inhaled only a whiff of air (50ml) with the actuation. Those who inhaled incorrectly were taught the correct technique. 11 learned it within 5 minutes of instructions and demonstrations. 7 required a teaching aid. When retested 1-2 weeks later, 15 out of 18 had retained the correct technique. 2 others learned it after the second lesson, but 1 could not be taught even after 6 lessons. Incorrect inhalation technique is a frequent finding among asthmatics taking aerosol medications. The physician starting this form of therapy should be certain to teach each patient the correct inhalation technique and also check those already on it.

1683 ORAL VERSUS AEROSOL ADRENERGIC BRONCHODILATORS IN ASTHMA

Haesoon Lee (Spon. by Lawrence R. Shapiro). New York Medical College, Lincoln Hospital Center, Department of Pediatrics, Bronx, NY

17 children attending asthma clinic who had moderate airways obstruction and who were in clinically stable condition were tested for bronchodilator response to aerosol and oral adrenergic agonists in a double blind randomized sequence on 4 different days. 4 regimens were: Aerosol- metaproterenol aerosol (Alupent) 1 puff (0.65mg) at the start and at 20 and 40 minutes plus placebo tablets, Oral- metaproterenol tablets 10-20mg or terbutaline 2.5-5mg by mouth based on weight plus placebo aerosols, Combined- a combination of both actives, and Placebo-placebo tablets and aerosols. Forced vital capacity and forced expiratory volume in 1 second (FEV₁) were measured before and after the therapy at 20, 40, 60, 120, 180, and 240 minutes. Side effects, blood pressure, and pulse rates were checked before and after the therapy. Oral therapy produced a slow improvement in FEV₁ which reached the maximum (26% above the baseline) at 120 minutes and started to decline at 240 minutes. Aerosol therapy produced a prompt, marked increase in FEV₁ at 20 minutes which reached the maximum (39%) at 120 minutes and started to decline at 180 minutes. FEV₁ response on the aerosol therapy was significantly greater than on oral therapy from 20 to 120 minutes. The combined therapy produced a slightly, but not significantly, greater response than the aerosol therapy throughout the test period. Side effects were frequent on the oral and combined therapy and infrequent on the aerosol regimen. Aerosol route is superior overall.

1684 ABNORMAL PROSTAGLANDIN (PG) METABOLISM AND PLATELET FUNCTION IN CYSTIC FIBROSIS (CF) PATIENTS.

Richard J. Lemen, Betty Revsin, James J. Corrigan, Claire M. Payne, Mary A. Moon and Chris M. Hokans. Departments of Pediatrics Physiology and Pathology, University of Arizona, Tucson, Arizona.

Our previous studies (Lemen, et al. Am.Rev.Resp.Dis., 117:639-46, 1978) reported abnormal plasma PG concentrations in CF patients; but, the physiologic significance of these observations is unknown. We correlated plasma PGs to ADP induced platelet aggregation (PA) in 8 normal and 11 CF subjects. Platelet proaggregators (e.g. thromboxane (Tx)A₂ as TxB₂ and PGE₂) and antiaggregators (PGI₂ as 6-keto-F₁α) were measured by radioimmunoassay. With parallelism between standards and plasma and with the within-sample coefficient of variation <9%, 6-keto-F₁α was significantly (p < 0.05) reduced in CF patients (X̄±SE 408 ± 105 pg/ml) compared to normal subjects (X̄±SE 708 ± 196 pg/ml). PGF₂α was significantly (p < 0.05) elevated in CF patients (X̄±SE, 282 ± 106 pg/ml) compared to normal subjects (X̄±SE, 173 ± 65). TxB₂ and PGE₂ were not different in CF patients (X̄±SE, 327 ± 84 and 793 ± 239 pg/ml, respectively) compared to normal subjects (X̄±SE, 318 ± 92 and 995 ± 495 pg/ml, respectively). ADP induced PA was inhibited in 13/18 studies in CF patients. Platelet electromicrographs showed decreased dense bodies/platelet profile in 4/8 studies, and PA correlated with dense bodies in 4/8 studies. PA was not correlated with drug therapy including antibiotics or vitamin E. We conclude that abnormal PG metabolism may result in abnormal platelet function in CF patients. Supported in part by grant HL 23773 and the Cystic Fibrosis Foundation.

1685 UPPER AIRWAY OBSTRUCTION (UAO) DURING SLEEP: A CLINICAL SPECTRUM.

S. Levine, M. Bose, M. Wegmann, R. Beckerman J. Smith (Spon. by J. Lewy), Dept. of Pediatrics, Section of Pulmonary Diseases, Tulane Univ. School of Medicine, New Orleans.

Sleep disturbance (SD) is often the presenting complaint in children who have UAO of various etiologies. We recently evaluated 9 children who presented with symptoms of SD and/or signs of UAO during sleep. They were studied with noninvasive techniques including sleeping airway fluoroscopy, respiratory gas exchange, airflow and impedance pneumography for evidence of UAO and apnea.

N	Group 1		Group 2	
	5		4	
x Age (Yrs)	2.7		2.9	
Predisposing Factors	(T&A)		Tonsils Adenoids 5/5	
Hypopharyngeal Collapse	3/5		Micrognathia Glossoposis 3/4	
Cor Pulmonale	0/5			
Alveolar Hypoventilation	1/5			
Apnea	1/5			
Delayed Milestones	1/5			

We found dynamic fluoroscopy of the UA to be a valuable and non-invasive test for children with symptoms of SD and/or obstructive apnea. The site of obstruction was localized by fluoroscopy to the hypopharynx in 6/8 of cases studied. The abnormalities seen in group 1 were reversed by simple T&A. Group 2 required more complex surgical procedures. We feel children presenting as moderately severe SD should be evaluated with tests of airway function and airway fluoroscopy prior to and after surgery.

1686 CHANGES IN MEAN AIRWAY PRESSURE: SIGNIFICANCE IN NEONATAL AIRLEAK SYNDROME.

Willard A. Litzenger, M.Douglas Cunningham, Nirmala S. Desai, Dept. of Pediatrics, College of Medicine, Univ. of Kentucky, Lexington (Sponsored by Jacqueline A. Noonan)

The increased occurrence of pulmonary interstitial emphysema, pneumothorax, pneumomediastinum, and pneumopericardium during mechanical ventilation is well known. However, the mechanical relationships during ventilation between time, pressure and neonatal airleak syndrome (NAS) are poorly understood. 13 of 56 infants (23%) ventilated with pressure-cycled ventilators (Bournes BP200 set to deliver a square respiratory wave form) developed roentgenographic findings of NAS. 13 similarly ventilated infants (controls) who did not develop NAS were matched with the NAS patients for birth weight (X̄=1485), gestational age (X̄=31 wk), initial mean airway pressure (Paw) and initial alveolar-to-arterial gradient (A-aDO₂). Infants who developed NAS had positive changes in Paw (+ΔPaw) until 60 hr of life, while controls showed -ΔPaw after 12 hr of life. The ΔPaw for NAS patients in the first 24 hr of life was +3.5 cm H₂O vs -0.4 cm H₂O for controls (t=2.76, p<0.02). Average Paw for 108 hr was 11.9 cm H₂O ± 1.0 SEM for airleak infants vs 8.3 cm H₂O (t=3.0, p<0.01). We conclude: 1) infants who develop NAS while undergoing pressure-cycled ventilation receive significantly higher Paw than infants without NAS, and 2) the increase in Paw during the first 24 hr of ventilation is more rapid in infants who develop NAS; therefore, we anticipate an airleak if the Paw must be increased more than 3.5 cm H₂O in first day of mechanical ventilation.

1687 PULMONARY FUNCTION TESTS, PULMONARY EDEMA (PE) AND BRONCHIAL OBSTRUCTION IN CHILDREN WITH BRONCHOPULMONARY DYSPLASIA (BPD).

M.M. Logvinoff, R. Lemen, L.M. Taussig, and B. Deatin, Arizona Health Sciences Center, Dept of Pediatrics, Div of Respiratory Sciences, Tucson, Arizona.

In 6 BPD (3 to 43 m) measurements of intraesophageal pressure, airflow and tidal volume allowed calculations of total pulmonary resistance (R_L) and dynamic compliance (C_L). On distinct days for each drug, changes of R_L and C_L with diuretic, Furosemide (1mg/kg IV), inhaled bronchodilators, Isoproterenol (1:200, .2cc) Atropine (.05mg/kg) were assessed. Each patient was his own control on the following "placebo" day. A two-tailed paired test was used to compare baseline values prior drug study, versus baseline values the following "control" day. The same T test was used to compare change of R_L, C_L in each group (Furo, Isop, Atr.) versus change during control day. Baseline values (n=26) revealed: On 23 instances, R_L was between 150% and 450% of the predicted value; C_L was below 75% of the predicted value in 5 instances. There was no significant difference in baseline parameters (p > .05) prior drug study versus control day. Diuretic improved C_L (p=.01, n=6), sympathomimetic improved R_L (p=.07, n=5), parasympatholytic improved C_L (p=.06, n=4). The study indicated that BPD had marked alteration in large airway (law) mechanism (R_L). Small airway (Saw) function (C_L) appeared normal in 13 instances, pointing out that only specific C_L per liter of lung volume will assess Saw; however, changes of C_L reflected Saw changes. PE and Law bronchospasm appeared coexisting factors in BPD. PE may also contribute to Saw (C_L) through a vagal reflex.