CAN SPECTRAL ANALYSIS OF POLYGRAPHS DISTINGUISH HIGH †1759 RISK FOR SIDS FROM NORMAL INFANTS? J. P. Finley, S.T. Nugent (Spon. by R.B. Goldbloom, Dalhousie Univ. Dept. of Pediatrics, Halifax, N.S., Canada)
Despite widespread use of cardio-respiratory polygraphs to

screen infants at high risk for SIDS, analysis for apnea frequency and periodic breathing often fails to find abnormalities in high risk infants, even those who subsequently die. The possibility that abnormal cardio-respiratory control might alter the frequency spectrum of heartrate (HR) and respiratory (resp.) pattern led us to study prospectively 14 infants at risk for SIDS (7 near miss, 7 siblings) ages  $7.0\pm3.4$  wks. Two healthy comparison groups were used: 14 infants aged  $12.2\pm3.4$  wks. and 10

other aged 0.6 ± .6 wks. All infants are alive at six months.

Infants were studied in hospital during daytime naps, with recording of heartrate, strain gauge respirogram and sleep state. Power spectra were computed for HR and resp. during quiet sleep for analysis of peaks at the resp. frequency (RF) and at low frequency (LF) (.05-.15Hz) previously described (J. Physiol. 285:85, 1978, Can J. Physiol. 61:329, 1983).

We found no significant difference in RF or LF amplitude or frequency for either HR or resp., between high risk and controls: 
 Resp. LF(Hz)
 RF(Hz)
 LF amp.
 HR:LF(Hz)
 RF(Hz)
 LF amp.

 Risk
 .083
 .59
 11.7 + 16
 .074
 .59
 4.69 + 3.4

 0.6 wk.Con.
 .093
 .61
 16.1 + 19
 .093
 .63
 3.78 + 3.4

 12 wk. Con.
 .089
 .65
 23.4 + 22
 .096
 .66
 5.30 + 4.5

 CONCLUSION:
 Spectral
 analysis
 does not discriminate between high
 risk and normal groups, although our groups were small. Age variations in the spectra were not significant in normals.

BRONCHIAL REACTIVITY IN PREMATURE INFANTS WITH BPD. •1760 Maria Fort, Etsuro Motoyama, Kenneth Klesch, Rebecca Mutich, Robert Guthrie, University of Pittsburgh School of Medicine, Pittsburgh, PA

Bronchodilators generally are not considered useful in infants,

Bronchodilators generally are not considered useful in infants presumably because of poor airway smooth muscle development. On the other hand, bronchial reactivity has been suspected in infants with chronic lung disease such as BPD. We investigated to see when such a reactivity develops postnatally in patients who are mechanically ventilated in the neonatal ICU. In 22 prematurely born infants (BW: 1118 ± 350(SD)gm; GA:27.7 ± 2.4 wk; PNA: 46.5 ± 30.7 days) with RDS/BPD, maximum expiratory flowvolume curves by forced deflation (DFVC; Pediatr Res 11:220,1977) were obtained on 63 separate occasions. At each testing DFVC were examined in 3 conditions: baseline (BL), after saline ne bulization (NSS) as a placebo, and after 10 minutes of 0.25% isoetharine nebulization (bronchodilator BD). At BL all infants showed decreased maximum expiratory flow at low lung volumes (Vmax25), indicating lower airway obstruction. Forced vital capacity (FVC) was also decreased (29.3 ± 12.8 ml/kg). Following NSS and lung inflation 12% of infants showed an increase (>20%) in FVC, indicating recruitment of airspaces. After BD 79% showed an increase (>20% of NSS) in  $\hat{V}$ max25. The mean response in  $\hat{V}$ max25 at the same lung volume from TLC was 320 ± 249% of NSS (maximum: 1270%) (p<.001), indicating marked bronchodilation. The youngest responder was a 17-day-old and the most premature who responded was 28 weeks PCA. Thus the course of a majority of premature infants with chronic ventilatory failure is complicated by marked bronchial hyperreactivity. (Supported in part by grants: NIH HL25810; ALA of PA).

ANATOMIC DISTRIBUTION AND † 1761 CHANGES IN GUINEA PIG PULMONARY B-RECEPTORS IN EXPERIMENTAL ASTHMA. Catherine Gatto, Thomas P. Green, Dana E. Johnson. University of Minnesota Medical School, Department of Pediatrics, Minneapolis, Minnesota. Quantitation using tissue homogenates has demonstrated a decrease in pulmony of recorders in corporate and the state of the sta

in pulmonary B-receptors in experimental asthma. However, techniques using disrupted tissue have not permitted precise identification of the (A) was produced in guinea pigs by the sc injection of oxalbumin then daily exposure to ovalbumin aerosol for 4-5 weeks. Animals initially developed dyspnea during the aerosol exposures, but showed a tolerence to increasing ovalbumin concentrations over time. Using to increasing ovalbumin concentrations over time. Osang 3H-dihydroalprenolol, 8-receptors were radioautographically localized and quantitated in lung sections of saline control (C) (n=6) and (A) (n=9) guinea pigs. Scatchard analysis showed a single class of binding sites with a Bmax of  $368\pm32(\text{C})$  and  $258\pm15(\text{A})$  fmole/mg protein (p<.005). Binding was of high affinity  $K_d=0.89\pm.09(\text{C})$ ,  $0.77\pm0.07(\text{A})$  nM (N.S.). A 25-30% decrease in  $\beta$ -receptor number in alveolar, bronchiolar and bronchial epithelium (E) and bronchiolar smooth muscle (SM) (pc.001) appeared to be responsible for the 30% decrease in total (A) lung \(\beta\)-receptors. No decreases were noted in bronchial, arterial or venous SM. Despite the decrease in \(\beta\)-receptors no significant differences were noted in tidal volume, dynamic compliance and airway resistance between (C) and (A) animals in response to antigen challange as determined by body plethysmography immediately prior to sacrifice. These data suggest that decreases in \( \mathbb{B}\)-receptor number can occur without changes in pulmonary function and decreases in E rather than SM B-receptors account for the majority of the observed change.

CONGENITAL DIAPHRAGMATIC HERNIA- CONTRIBUTION OF **•**1762 PULMONARY ARTERIAL REMODELLING TO PERSISTENT PULMON-ARY HYPERTENSION. Robotzt L. Geggel, John D. Murphy, David Langleben, Lynne M. Reid. Spon. by Thomas J. Hougen. Harvard Medical School, Children's Hospital Center, Department of Research Pathology, Boston.

Some infants with congenital diaphragmatic hernia (CDH) who die after surgical correction have a transient postoperative period during which oxygenation is adequate("honeymoon"(H) period) while others have persistent hypoxemia. To explain the different clinical course, lung development and pulmonary arterial structure of 7 neonates with CDH who came to autopsy were analyzed using morphometric techniques. Three infants comprised the H group, transiently having PaO2>150 torr in the descending aorta (DAo) while receiving 100% oxygen. Four infants comprised the no-honeymoon (no-H) group and never had PaO<sub>2</sub>>85 torr in the DAo. All lungs were small for age, the ipsilateral being smaller in weight and volume. Three of the 4 infants in the no-H group had more reduced ipsi- and contralateral lung weight and ipsilateral lung volume compared with the H group. The contralateral lung volumes were similar suggesting relative overinflation of lung substance in the no-H group. Each member of the no-H group had abnormal structure of intraacinar arteries with muscle extending precociously to alveolar wall vessels. In addition, compared with the H group, the luminal area of pre- and intraacinar arteries in the no-H group was decreased by reduced external diameter and/ or increased medial thickness in both lungs. The different post-operative course of infants with CDH is based on both severity of pulmonary hypoplasia and extent of pulmonary arterial remodelling

RHINOMANOMETRY AND NASAL AIRWAY RESISTANCE (Rn)
MEASUREMENTS IN NORMAL AND PREALLERGIC CHILDREN.
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Winston-Salem, N.C.
Rhinomanometry is used primarily to measure nasal airflow
(Y), transnasal driving pressure (P) and Rn in adults and
gives an objective quantification for the common symptom of
nasal congestion. This study compares the usefulness of 3
rhinomanometric methods and Rn measurements in non-atopic
children (negative allergy skin tests) and preallergic
children (positive skin tests, no allergic symptoms). Rn was
determined at a low airflow (V) and at the recommended
reference of -1.5 cm H.O P and pressure (P).
Anterior rhinomanometry (AR), posterior rhinomanometry (PR)
and forced oscillatory rhinomanometry (FOR) were done in 9
pre-allergic and 9 age-and-sex matched normal children (7-15
yys.). The V and P measurements were obtained for 2-4 hours.
Most children had 1-3 additional times performing
rhinomanometry.
Mean total Rn ranged from .214 to 9.84 cm H<sub>2</sub>O/1/sec at the
low reference and 1.50-20.55 at the 1.5 reference. All 18
children could perform AR whereas only 10 could reliably do PR
and 9-FOR. At the low reference, the preallergic children had
higher unilateral Rn compared to the normal children (P.C.05)
when analyzing the more congested nasal passage. There was no
difference in total Rn obtained by the 3 methods (D.10). At
the 1.5 reference, the preallergic children had higher total
Rn using the AR and FOR methods (D.10 and P.C.05) in addition
AR is the best suited method of rhinomanometry to use in
children. Preallergic children exhibit subtle differences in
Rn which precede the appearance of symptoms and changes in the
masal mucosa. Rhinomanometry may assist the physician in
differentiating allergic children from normal children.

CHANGES IN PULMONARY MECHANICS WITH GROWTH IN INFANTS

CHANGES IN PULMONARY MECHANICS WITH GROWTH IN INFANTS TIME CHOOSE TO THE CHOOSE THE CHANGE OF THE CHANGE THE CHANGE OF THE CHANG to 4 years of age, were studied as controls. Tidal volume was measured by pneumotachography, esophageal pressure with a water filled feeding tube, and FRC by N2 washout. Because infants with CLD had a slower growth pattern, results could not be compared chronologically. FRC was therefore related to weight by linear regression analysis. This correlation was very good and nearly identical for both groups, (r=0.94 in CLD; 0.98 in controls) suggesting normal growth of lung volume in CLD. FRC was therefore suggesting normal growth of lung volume in CLD. FRC was therefore used as independent variable and  $C_L$  and  $G_L$  were related to FRC by linear regression analysis.  $C_L$  was closely correlated to FRC in both groups,(r=0.93 in CLD;0.96 in controls) with an intercept that was nearly identical. The slopes however were different indicating a lower specific  $C_L$  in CLD than in the controls (60 vs 75 ml/cmH<sub>2</sub>0/FRC).  $G_L$  was also closely related to FRC (r=0.77 in CLD;0.91 in controls). The slopes were nearly identical, but the intercept in infants with CLD was only half of that in the normal controls. This indicates that infants with CLD have a reduced  $G_L$  in the newborn period that gradually increases with growth and in the newborn period that gradually increases with growth and approaches normal values by age  $3\ \mathrm{years}$ .