

BREATH SOUND MONITORING FOR APNEA. Robert C. Becker-

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Impedance respiratory monitoring is incapable of detecting obstructive apnea. We compared a microphone breath sound detector to an impedance monitor in 10 sleeping infants and children in order to determine its ability to detect normal respirations, central, and obstructive apneas and alarm for apneas greater than 10 seconds. Air flow was used as a standard for all measurements. Breath sounds detected 98.4% and impedance 98.9% of all normal respirations. There were a total of 42 central apneic pauses. Breath sound detected 40 and impedance 41 of these apneas yielding 95 and 98% respective rates of detection. Breath sound monitor alarmed for 5 of 7 central apneas and impedance monitor for 2 of the 7. There were 75 obstructive apneas. Breath sound detected 58 or 77% detection rate. The breath sound monitor alarmed for 5 of 13 obstructive apneas and the impedance monitor none. There were no statistical differences in either breath or central apnea detection between the two monitors. Air flow was statistically better than breath sound for the detection of obstructive apnea ($p < .05$). Breath sound was statistically better than impedance for obstructive apneas ($p < .025$). Our study suggests that a breath sound monitoring system may offer a practical alternative to impedance respiratory monitoring for the detection of normal respiration, central and obstructive apneas in infants.

PULMONARY SEQUELAE OF PROLONGED AMNIOTIC LEAK. Vinod

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Fetal compression, pulmonary hypoplasia, and oligohydramnios have been previously associated with prolonged amniotic leak (PAL) syndrome. Eight fetuses were identified with PAL duration of 2 to 14 weeks. These fetuses were evaluated postnatally for pulmonary complications. In addition, prenatal real time ultrasound examination was utilized to measure the ratio of thoracic circumference to abdominal circumference (TC:AC). These neonates were beyond -1 S.D. of previously established mean values (normal mean \pm SEM values of TC:AC = 0.944 \pm 0.050). At birth there was evidence of oligohydramnios and compression; the mean \pm SEM values were: birthweight, 1233 \pm 110 gm; gestational age (GA), 29.1 \pm 0.78 wks; onset of PAL, 22.6 \pm 1.9 wks GA; duration of PAL, 6.5 \pm 1.5 wks. Pulmonary hypertension, right to left shunting, severe respiratory acidosis and need for high peak inflation pressures (40 to 65 cm H₂O) were observed in 5/8 neonates. Of these, 3 died and pulmonary hypoplasia was diagnosed at necropsy (mean lung weight 14.3 gm). Two of the survivors developed significant bronchopulmonary dysplasia. No long-term sequelae were observed in 3/8 neonates except the immediate need for ventilatory support. Both morbidity and mortality were best correlated to GA at onset of PAL and the GA at birth. These observations emphasize the need for intensive prenatal evaluation to predict fetal thoracic compression and postnatal pulmonary sequelae. Evidence of fetal thoracic compression may prove to be a useful determinant for optimal time of delivery with the PAL syndrome.

VENTILATORY RESPONSE OF GROWING PRETERM INFANTS TO COMBINED INSPIRATORY AND EXPIRATORY RESISTIVE LOAD. Vinod K. Bhutani, Soraya Abbasi, Emidio M. Sivieri,

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Ventilatory responses to combined inspiratory and expiratory (I & E) loading were evaluated in 5 growing preterm neonates. Mean \pm SEM values were gestational age: 29.6 \pm 0.7 weeks, birthweight: 1178 \pm 138 gm; study age: 54 \pm 13 days; study weight: 1661 \pm 440 gms. Pulmonary functions, tidal volume (V_T), minute ventilation (MV), peak inspiratory flow (V_I), and expiratory flow (V_E), inspiratory time/total respiratory time (T_I/T_{tot}), respiratory frequency (f) and work of breathing (WOB) were measured before and after application of the resistance (R). A variable resistor was used to administer a separate load of 50 (R₁) and 100 (R₂) cm H₂O/L/sec during I & E flow for 60 seconds. Mean \pm SEM control pulmonary function values were: pulmonary compliance 3.4 \pm 0.26 ml/cmH₂O; pulmonary resistance: (I) = 44 \pm 14 and (E) = 69 \pm 13 cmH₂O/L/sec, respectively; V_T = 13.7 \pm 2.1 ml and MV = 894 \pm 79 ml/min; V_I = 2.75 \pm 0.23 and V_E = 2.85 \pm 0.26 L/min; T_I/T_{tot} = 0.48 \pm 0.04 and f = 61 \pm 4 breaths/min. Application of R₁ resulted in a significant change ($p < 0.05$) immediately and for a duration of 60 secs in the V_T (16% decrease) and in both V_I and V_E, a 20.4 and 21.6% decrease, respectively. The MV only decreased (25%, $p < 0.01$) at initial application, and compensation in MV and work was noted by 60 sec. A similar, but greater magnitude change was observed with R₂. These observations show an initial decreased ventilatory response to the load and an ensuing adaptation by 60 seconds.

EFFECT OF HIGH FREQUENCY JET VENTILATION ON NEONATAL

1741 TRACHEAL MECHANICS: Vinod K. Bhutani, Thomas H. Shaffer, Alan R. Spitzer, Frank W. Bowen, & William W. Fox, University of Pennsylvania, Dept. of Pediatrics, Temple University School of Medicine, Dept. of Physiology, Philadelphia, Pa.

Preterm neonates have recently been provided adjunctive ventilatory support with high frequency jet ventilation (HFJV). Neonatal airways are more compliant and susceptible to pressure-induced deformation. Thus, these airways may react differently when exposed to the rapid oscillations (10 - 15 Hz) and changes in intratracheal pressures during HFJV. To study this effect, changes in in-vitro tracheal dimensions and mechanics were determined in preterm (0.85 term gestation) and term rabbits pups after HFJV. Length, diameter, volume (V) and pressure-volume (P-V) relationships (by liquid plethysmography) were measured for both groups (n = 6 each) before and after HFJV. For a duration of 60 min. HFJV was administered to excised tracheal segments at peak airway pressures of 20 cm H₂O; end distending pressure, 3.5 to 3.8 cm H₂O mean airway pressure, 6.7 to 6.8 cm H₂O; and the rate, 600/min. In the preterm there was a significant ($p < 0.001$) increase in the mean tracheal length and diameter. Mean \pm SEM values of V increased from 0.80 \pm 0.16 ml to 1.65 \pm 0.32 ml (109.4%; $p < 0.001$). Similarly in the term group, the mean V increased 60.5% ($p < 0.001$). The P-V relationships were altered such that the mean \pm SEM tracheal specific compliance decreased significantly from 0.0359 \pm 0.0015 to 0.0155 \pm 0.0011 cm H₂O ($p < 0.001$) in the preterm; and, from 0.0295 \pm 0.0017 to 0.0208 \pm 0.0016 cm H₂O, ($p < 0.05$) in the term groups. These data indicate significant dimensional and mechanical alterations in the tracheal behavior after HFJV for both groups.

CHARACTERISTICS OF THE LATE RESPONSE IN EXERCISE-INDUCED ASTHMA. C. Warren Bierman, Stephen G. Spiro

Exercise-induced asthma (EIA) occurs after exercise and subsides spontaneously within 2 hours in most individuals with asthma. However, a subpopulation of patients has a late asthmatic reaction 4 to 12 hours later. An increasing number of papers have documented this late reaction. This study further characterizes the late pulmonary reaction to exercise.

Nine young adult subjects who developed asthma after a standard treadmill exercise test were studied. Pulmonary function tests were performed before and after exercise and serially for at least 6 hours afterwards. The subjects performed pulmonary function tests while breathing room air and after 3 inhalations of an oxygen-helium mixture with each test. Although pulmonary function returned to normal within 3 hours after exercising in all subjects, 8 of the 9 had a late asthmatic reaction 3 to 5 hours afterwards. In general, the late response was less severe than the initial. Two subjects, however, noticed clinical asthma. In the immediate response, 8 of 9 were helium "responders" suggesting that the immediate response involved both large and small airways. In the late response, all were helium "nonresponders" suggesting that it occurred primarily in small airways.

One could speculate that the immediate reaction to exercise is due both to airway cooling which would affect primarily large central airways and mediator release which would affect smaller peripheral airways while the late response is due solely to mediator release and affects primarily small airways.

EFFECTS OF LABOR AND AIR-BREATHING ON CATION EXCHANGE

1743 IN LUNG EPITHELIAL CELLS OF BABY BUNNIES. R D Bland and C A R Boyd. Cardiovasc Res Inst, Dept Pediatr, Univ California, San Francisco; Dept Human Anat, Univ Oxford, UK.

Absorption of fetal lung liquid before and soon after birth is essential for the normal transition from placental to pulmonary gas exchange. We recently found that granular epithelial cells from lungs of adult and newborn rabbits actively extrude Na⁺ in exchange for K⁺; this process, which may be important in regulating lung fluid balance, was considerably slower in cells derived from fetal lungs. To test the hypothesis that either air-breathing or labor, or both, might activate this process, we harvested granular pneumonocytes from lungs of fetal and newborn rabbits and measured 86Rb⁺(K⁺) uptake by these cells in the presence or absence of 10⁻⁴ M ouabain (ø), which inhibits Na⁺-K⁺-ATPase. Fetal rabbits, preterm (28 d) and term (31 d), experienced neither labor nor air-breathing. Newborn rabbits, born by cesarean section (cs) or vaginally (v), breathed for up to 2 h after birth. Pups born operatively did not experience labor. Results ($\bar{x} \pm s_x$):

	86Rb ⁺ (K ⁺) Uptake nmol/10 ⁶ cells/h	Fetuses	Newborns
Total	6 ± 1	6 ± 1	7 ± 3
ø-Sensitive	5 ± 1	5 ± 1	5 ± 1
ø-Insensitive	1 ± 1	1 ± 1	3 ± 1

We conclude that air-breathing stimulates passive (ø-insensitive) 86Rb⁺ uptake, whereas labor influences both active (ø-sensitive) and passive cation exchange in granular pneumonocytes derived from term rabbits. These effects may hasten clearance of fetal lung liquid and help keep air spaces dry after birth.