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ALL NEONATES ARE NOT OBLIGATE NOSE BREATHERS. M.J. Miller, R.J. Martin, W.A. Carlo, A.A. Fanaroff, Dept. Peds., Case Western Res. Univ., RB&C Hosp., Cleve, OH

Unlike adults, neonates are considered obligate nose breathers, hence entirely dependent on a patent nasal airway for ventilation. To fully explore their respiratory response to nasal obstruction, we simultaneously monitored nasal and oral ventilation during sleep and in response to multiple 15 sec nasal occlusions. Ten healthy term infants (mean BW 3600±190g, age 1.7±0.6 d) were studied for 60 min periods. Nasal and oral airflow (via two resistance-matched pneumotachometers), heart rate (HR), TcPO<sub>2</sub> and PetCO<sub>2</sub> and sleep state were continuously recorded.

Five of 10 infants initiated and sustained effective oral breathing without arousal during 36±32% of occlusions independent of sleep state. In these infants, arousal occurred during 31±27% of occlusions, and no response in 34±23%. In the remaining 5 infants who did not exhibit oral breathing, arousal occurred in 42±34%, and no response in 58±34% of occlusions. Once begun (within 0-6 sec) oral breathing could be sustained for at least 1 min of continuing nasal occlusion. Tidal vol., resp. rate, HR, TcPO<sub>2</sub> and PetCO<sub>2</sub> did not change when oral breathing occurred during nasal occlusion, although minute ventilation decreased from 265 to 199 cc/min/kg, p<0.05. Moreover, two of 10 infants exhibited spontaneous combined nasal/oral ventilation while in undisturbed sleep. These results clearly demonstrate that not all infants are obligate nose breathers. Effective oral ventilation can now be included among the neonate's physiologic defenses against life threatening nasal obstruction.

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THE EFFICACY OF FACE MASK RESUSCITATION

Anthony D. Milner, Harish Vyas and Iris E. Hopkin Dept. of Child Health, City Hospital, Nottingham, UK.

Although the physical characteristics of hand-held face mask resuscitation devices used by most units in the first line therapy for asphyxia at birth are well known, there have been no studies published on their efficacy. We have, therefore, measured inflation pressure and tidal exchange using a Laerdal Resuscitator in 9 babies asphyxiated at birth (L). Their birth weight ranged from 1.96 to 3.8 Kg (mean, 3.26) and gestational ages from 34 to 40 weeks (mean, 39.0). Results were compared with a matched group of 9 babies (I) resuscitated by intubation. The Apgar scores immediately prior to resuscitation were 5.0 (L) and 3.3 (I). Tidal exchange and inflation pressures were measured using the same pneumotachograph/pressure transducer systems. Only expiratory volumes could be analysed with the Laerdal system due to leak around the face mask. Both systems produced similar first inflation pressures (L, 31.4; I, 28.2 cm/H<sub>2</sub>O) but expiratory volumes were strikingly different (L, 2.7 ml; I, 14.3 ml). Similar results were in the next two breaths. Throughout resuscitation the Laerdal system rarely produced adequate exchange (dead-space X 2). The failure appears to be due to the short duration of the inflation pressure (L, .3 to .5 sec; I, >1.0 sec) in a situation where the airway is fluid-filled and highly viscous. We conclude that face mask resuscitation does not produce adequate tidal exchange but relies on stimulating the baby to commence spontaneous respiration. Performance could be improved by prolonging the inflation time.

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OLIGOHYDRAMNIOS (OA)-INDUCED LUNG HYPOPLASIA (LH): INFLUENCE OF TIMING AND DURATION, (ANIMAL MODEL). Adrien C. Moessinger, Margaret H. Collins, William A. Blanc, Jerome Kleinerman, L. Stanley James, Depts. of Ped. & Path Columbia Univ. & Mt. Sinai Sch. of Med, N.Y.

Having documented that OA leads to significant LH in the fetal guinea-pig, we looked to see if the effect varied with the timing of onset and the duration of OA. OA was induced by creating amnio-peritoneal fistulas in 4 experimental groups: I, long term OA early (d 40-50); II, long term OA late (d 45-55); III, short term OA early (d 45-50); IV, short term OA late (d 50-55). For each group, untouched littermate or gestational age-matched fetuses were used as controls. OA was documented by lack of amniotic fluid (AF) at sacrifice in each experimental animal (N=23) whereas controls had normal AF volumes (N=23). Lung DNA content was used as an index of cell number. Since there was a slight but not significant difference in body weight, we expressed the values as lung DNA per gram of fetal weight. Significant differences (p<.005) were found in groups I, II, III. The "magnitude" of the effect, (i.e. the percentage difference between experimental and control values) was as follows: I:-38%; II:-26%; III:-24%; IV:-14%. This experimental study confirms the clinical impression that the earlier the onset of OA and the longer its duration, the greater is the impact on lung growth. Morphometric studies of 4 lungs in group III showed that the experimental lungs had lower volumes (83%), lower volumetric density of lung parenchyma (95%), less total # of alveolar-like structures (74%), lower ISA (84%) and a disproportionate reduction in total length of elastic tissue (47%).

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IS THEOPHYLLINE MORE EFFICIENT THAN CAFFEINE IN CONTROLLING APNEA IN PREMATURE INFANTS? I. MURAT, G. MORIETTE, C. BROUARD, B. FLOUVAT, J.P. REITZ sponsored by A. MINKOWSKI, Service de Médecine Néonatale, Hôpital Port-Royal, Paris, France.

To compare the efficiency of theophylline vs caffeine in controlling idiopathic apnea of premature infants, we studied 16 infants with 3 or more severe apneic attacks (i.e. apnea > 10 seconds with H.R. < 80 beats/min. > 30 sec.) during a 24 hours cardiorespiratory recording. These infants were randomly assigned to the theophylline treated (group I : n = 8 ; GA = 30.5 ± 0.4 wk ; BW = 1.27 ± 0.07 kg ; PNA = 11.7 ± 1.9 days) or the caffeine treated group (group II : n = 8 ; GA = 30.5 ± 0.7 wk ; BW = 1.46 ± 0.10 kg ; PNA = 11.6 ± 2.8 days) (means ± SEM). The corresponding treatment was started immediately using currently accepted doses and schedules.

Recordings immediately before (day 0) and after randomization (day 1), and four days later (day 5) allowed to calculate and to compare the apnea indices (AI) (i.e. average number of severe apneic attacks per 100 minutes) in the two groups. The AI on day 0 day 1 and day 5 were similar in groups I and II (d0 : 1.02 ± 0.4 vs 1.42 ± 0.7 ; d1 : 0.12 ± 0.04 vs 0.13 ± 0.1 ; d5 : 0.06 ± 0.02 vs 0.07 ± 0.02). Significant decreases (p<0.001) of AI were observed from d0 to d1 and from d0 to d5 in the two groups.

We conclude that theophylline and caffeine demonstrate similar efficiency in the treatment of apnea. We suggest therefore that caffeine which is easier to use and potentially less toxic than theophylline should be preferably chosen.

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PROSTAGLANDIN E<sub>1</sub> OPENS THE DUCTUS VENOSUS IN THE NEWBORN LAMB. Frederick C. Morin (Spon. by Donald L. Shapiro). University of Rochester School of Medicine, Strong Memorial Hospital, Department of Pediatrics, Rochester, NY.

The ductus venosus plays an important role in determining the distribution of umbilical venous blood flow in the fetus. The mechanisms by which blood flow across the ductus venosus are regulated are only partially understood. The following experiments were performed to examine one mechanism by which the ductus venosus may maintain patency. Four lambs had catheters placed in the umbilical vein under inhalational anesthesia. A baseline portal angiogram was performed to assess patency of the ductus venosus. In each lamb, the ductus venosus was closed before the infusion began. The awake lamb then received an infusion of prostaglandin E<sub>1</sub> at a rate of 1 mcg/Kg/min for a period of 2-4 hours into the umbilical vein. The portal angiogram was then repeated. In 2 lambs studied at 24 hours of life, the ductus venosus was open following the infusion of prostaglandin E<sub>1</sub>. In 2 lambs who were studied at 2, 4 or 6 days, the ductus venosus remained closed following the infusion of prostaglandin E<sub>1</sub>. During the infusion, the sheep breathed spontaneously and showed no overt ill effects. Prostaglandins of the E series may be important in maintaining patency of the ductus venosus as they are in maintaining patency of the ductus arteriosus.

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PULMONARY VASCULAR EFFECTS OF POSTOPERATIVE ANESTHESIA IN CONGENITAL DIAPHRAGMATIC HERNIA John D. Murphy, Robert K. Crone, Joseph P. Vacanti (Spon by: Thomas J. Hougen) Harvard Medical School, Children's Hospital, Depts. of Cardiology, Anesthesia, and Surgery, Boston, MA.

Because of persistent high mortality of patients (pts) with congenital diaphragmatic hernia (CDH), symptomatic within hours of birth, from persistent fetal circulation (PFC); we designed a protocol to assess the physiologic effects of general anesthesia (GA) after repair of CDH. In 12 months 10 pts, presenting at < 7 hrs of age, underwent repair of left sided CDH. Cardiac catheterization was performed immediately after surgery. Postoperatively GA was maintained with fentanyl and pancuronium using rapid ventilation (2Hz-FiO<sub>2</sub> 1.0). Pulmonary artery (PA) and aortic pressures and pre-post ductal PaO<sub>2</sub>s were continuously recorded. The ratio of shunt to total systemic blood flow (Q<sub>s</sub>/Q<sub>t</sub>) was calculated and recorded throughout the postoperative period.

Three pts with severely hypoplastic lungs never improved and died from hypoxia and acidosis. Seven pts initially achieved a PaO<sub>2</sub> 150 torr after surgery; 6 (86%) survived; overall survival 60%. All 7 pts who entered the 'honeymoon' period had a low pulmonary vascular resistance (PVR); 5 later developed suprasystemic PVR and PFC. In all but one, who died at 14 days of age, the PVR was controlled by fentanyl and ventilatory adjustments without the use of vasodilators. The Q<sub>s</sub>/Q<sub>t</sub> was initially greater than 50% in all but 2 pts decreasing to less than 15% by 48hrs in all survivors. While closely monitoring the PA pressure and PaO<sub>2</sub> the pts were gradually weaned from pressure ventilation and oxygen.

These preliminary data suggest that those pts with CDH who enter the 'honeymoon' period after repair have sufficient lung tissue to survive. Prolonged GA with fentanyl may blunt the reactivity of the PVR allowing postnatal maturation of the pulmonary circulation, obviating the need for potentially dangerous vasodilators. When coupled with careful monitoring these techniques may improve the survival of pts with CDH.