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CONTRIBUTION OF LEFT-TO-RIGHT (L→R) DUCTUS ARTERIOSUS SHUNTING (DUCT SHUNT) TO PULMONARY BLOOD FLOW (PBF) IN PREMATURE LAMBS WITH AND WITHOUT HYALINE MEMBRANE DISEASE (HMD) DURING THE FIRST HOURS OF LIFE. Dora A. Stinson, Alexander C. Allen, Hugh M. MacDonald, Paul M. Taylor. Dalhousie Univ., Halifax, N.S. and Univ. of Pittsburgh, Dept. of Peds.

We tested the hypothesis that net L→R duct shunt contributes to PBF surge in premature lambs during the first hour of life (Pediat. Res., 8:433, 1974) and to high PBF seen later in lambs with severe HMD (Pediat. Res., 9:393, 1975). Total PBF, right ventricular output (RVO) and duct shunt were measured during first hours of life in 1 lamb with and 1 without HMD. At 129-130d gestation, a pre-calibrated electromagnetic flow transducer with non-occlusive zero was implanted on the main pulmonary artery (PA) to measure RVO; a similar transducer was implanted on the postductal portion of the common PA to measure PBF. Duct shunt was calculated as the difference between PBF and RVO. Lambs were returned to the amniotic cavity for 7-8d and then delivered by C-Section at 137d gestation. In each lamb, PBF rose sharply from < 50 to peaks of 487 and 473 ml/kg/min, respectively, at 22 and 29 min of age and then stabilized at lower levels by 1 hr of age. RVO stayed at high levels until after the PBF surge, then fell to stable levels by 1 hr of age. During the first 4 hr, net L→R duct shunt accounted for 28 to 68% of PBF (means 54 and 52%). These data suggest that L→R duct shunt contributes appreciably to PBF during the PBF surge and at age 1-4 hr. L→R duct shunt may be a major source of the high PBF previously reported in lambs with HMD.

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LONGITUDINAL ASSESSMENT OF THE EFFECTS OF UPPER RESPIRATORY TRACT ILLNESSES (URI) ON LUNG FUNCTION. L.M. Taussig, G.M. Loughlin, and K.A. Cota, Department of Pediatrics, Arizona Health Sciences Center, Tucson, Arizona.

The acute and chronic effects of URIs on lung function have not been adequately studied in children. Four normal (N) and 10 allergic (A) children, 9-15 years in age were studied prospectively at baseline, at the time of URI and 2, 4, and 9 months following URI. Lung function studies included maximal expiratory flow-volume curves and the response of flows to a helium-oxygen gas mixture (Visoˇ), exercise and isoproterenol. At baseline, 1 N and 5 A had minor abnormalities in flows measured after 50% (ˇmax 50) and 75% (ˇmax 75) of the vital capacity had been exhaled. At the time of the URIs, 12/14 subjects had elevated Visoˇ; 8 had a fall in FEV_1, ˇmax 50 and ˇmax 75; 9 subjects demonstrated a greater than 10% fall in flows with exercise (this included all 4 N), but Visoˇ was abnormal in only 3/12 subjects post-exercise. With isoproterenol, FEV_1 did not change in any subject but ˇmax 50 and ˇmax 75 increased in 10 subjects; Visoˇ was abnormal in 4 subjects. Two months following URI, small airway obstruction and bronchial hyperreactivity were still evident in nearly 1/2 of the subjects, but the abnormalities had lessened. By 4 months, lung function had returned to normal in most subjects. These results suggest that URIs produce large and small airway hyperreactivity which may contribute to the airway obstruction observed following URIs; these changes may persist for months. Supported by NHLBI Grants #14136 and #17153. G.M.L. was a Fellow of the American Lung Association.

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SKIN METABOLISM AND BLOOD FLOW MEASUREMENT: ROLE IN $TCPo_2$ ANALYSIS. A.M.Thunstrom, M.Stafford and J.W. Severinghaus (Spon.by W.H.Tooley) CVRI,U.C.San Fran.

The effect of heating by a tc Po_2 electrode on skin metabolism and blood flow, and on the relationship of Pao_2 to $tcPo_2$ was determined in 3 normal adults. While breathing O_2, skin metabolism was measured as O_2 solubility x $\Delta tcPo_2/\Delta t$ (torr/min), at 37° and 45° after arterial cuff occlusion. Using this, skin blood flow and diffusion gradients for O_2 and heat between capillaries and surface were measured by solution of simultaneous equations for $tcPo_2$ vs Pao_2 from 6 steady state correlations at 2 temperatures(43°,45°) and 3 Po_2s (75,150,600), using the changes of O_2 dissociation and solubility thereby induced. Heating efficiency, (Tc-37)/(Te-37) averaged .84 (c=capillary,e=electrode). (c-e)Do_2 was 28±3 torr at 44° Te. Skin O_2 consumption and blood flow were .0043±.0001 ml O_2 and .75±.10ml blood per gm per min. From these parameters, $tcPo_2$ was computed for $0<Pao_2<700$ defining a sigmoid relationship. At Te=44°, $tcPo_2$ nears 0 at Pao_2=20, approximates Pao_2 when $60<Pao_2<130$, and parallels Pao_2, (about 50 torr lower) when $Po_2>300$. $TcPo_2$ was most dependent on blood flow at high O_2. The data yield algorithms for "correcting" $tcPo_2$ measurements to obtain Pao_2. Skin metabolism increases 7%/°C, while blood flow (after 30 min. heating at 45°C) fell only 1.2%/°C with brief reduction of Te to 43°C. Skin blood flow, independently determined from $tcPo_2$ washin time constant after release of occlusion while breathing O_2, averaged about 33% higher than above, possibly due to post-ischemic hyperemia, and uncertainty regarding skin O_2 solubility. Similar analysis will be done in children.

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DISATURATED PHOSPHATIDYLCHOLINE AND PRENATAL PREDICTION OF PULMONARY MATURITY. J.S. Torday and Edward E. Lawson (Spon. by H.W. Taeusch, Jr.) Harvard Medical School, Boston Hospital for Women, Dept. of Pediatrics,Boston, MA.

Respiratory Distress Syndrome (RDS) in premature infants is associated with a deficiency of pulmonary surfactant. Disaturated phosphatidylcholine (DSPC) is the major phospholipid component of surfactant. We have utilized the osmium tetroxide (OsO_4)technique described by Mason et al. (J. Lipid Res. 17:282, 1976) to isolate DSPC from amniotic fluid. The DSPC fraction is quantitated on thin layer chromatography by spectrodensitometry. The L/S ratio and DSPC concentration were determined for 100 consecutive uncontaminated amniotic fluid samples. Both correlated with gestational age ($p < .01$) and with each other ($r=0.70$, $p < .01$). Using the L/S ratios and DSPC concentrations from 60 uncontaminated samples obtained within 72 hours of delivery enabled us to predict pulmonary maturity; however, the table below demonstrates that DSPC predicts RDS more accurately than does the L/S ratio:

	L/S			DSPC	
	< 1.5	1.5 - 1.9	> 2.0	≤ 150µg/dl	> 150µg/dl
RDS	6	2	2	10	0
NO RDS	3	8	39	3	47

DSPC also provides: 1)specificity for pulmonary lecithin, circumventing spurious results due to non-pulmonary lecithins, including blood and meconium contamination and 2)ready adaptability to pre-existing instrumentation for determining the L/S ratio with no added expense.
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STAGES OF FORMATION OF HYALINE MEMBRANES IN CONTROLLED EXPERIMENTS WITH PREMATURE LAMBS. Bernard Towers, Forrest H. Adams, Machiko Ikegami and Alan B. Osher, UCLA School of Medicine, Dept. of Pediatrics, Los Angeles, Calif.

In ten successive pairs of premature (118-122 days) twin fetal lambs endotracheal instillation of natural surfactant in suspension prior to the first breath of one twin prevented the appearance of RDS, and the lungs at autopsy (120 mins. after delivery by C-section) were well-aerated and compliant (Ped.Res. in press). Each control twin (on an alternate basis) received only diluent (water or saline) endotracheally. Each pair of twins was treated identically on volume-respirators (5ml/kg) on room air. All controls died between 23 and 49 minutes, except for two that survived 91 and 97 minutes. The histological development of hyaline membranes proceeded as follows (with some overlap) according to survival time: 1. appearance of shrunken cells with pycnotic nuclei lying free in alveoli; 2. clumping of dead and dying cells in lumina of respiratory and terminal bronchioles; 3. distention of small airways, usually proximal to the epithelial clumps; 4. outpouring of glycogen by bronchiolar epithelial cells; 5. hyaline membranes, initially with extrusion of remaining nuclear debris. Characteristic membranes were present only in animals that survived for more than 90 minutes. Two sets of triplets were studied during the series; each third member was sacrificed prior to the first breath: no lung pathology was found. Of the 10 twins that received NS only one showed two small segments of hyaline membranes; histochemical examination showed that NS had not penetrated into those peripheral units.

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PULMONARY FUNCTION FOLLOWING ACUTE MECONIUM INSUFFLATION. Tran,N.,C.Lowe,E.Sivieri,T.Shaffer(Spon.N.Huang Temple U,Sch of Med,Depts Physiol & Neonatol,Phila,PA

To investigate the acute airway obstruction phenomenon seen in neonates with meconium aspiration syndrom,pulmonary function was studied in 7 rabbits(mean2.6kg) after insufflation of meconium-saline fluid(1-2ml/kg). Animals were anesthetized,cannulated,intubated and mechanically ventilated with 100% O_2. Measurements of transpulmonary pressure,flow,volume and functional residual capacity(FRC) enabled calculation of dynamic(C_L)and specific(C_S) lung compliance and inspiratory(R_I)and expiratory(R_E) lung resistance before(control) and after(15,60,120min) insufflation of meconium-saline mixture. Arterial blood gases,pH,and ΔA-a were also done. For all animals mean control values were: C_L=1.40±0.24SE ml/cmH_2O kg; C_S=0.098±0.025SE ml/cmH_2O/ml; R_I=12.1±3.3SE cmH_2O/L/sec; R_E=18.1±3.0SE cmH_2O/L/sec; FRC=33.3±1.7SE ml/kg; PaO_2=496±12SE mmHg; $PaCO_2$=41.1±2.8SE mmHg; pH=7.37±0.03SE; ΔA-a=175±12SE mmHg. At 15 min post insufflation mean values of R_E($p<0.005$),FRC($p<0.05$), $PaCO_2$($p<0.005$),ΔA-a($p<0.005$) increased; and C_L($p<0.005$),C_S($p<0.05$) PaO_2($p<0.005$) and pH($p<0.005$) decreased relative to controls. At 60 & 120min these changes persisted with the exception of C_S and FRC which returned to control level. R_I was greater($p<0.025$) than control only at 60min post-insufflation,and at all times less($p<0.01$) than R_E. Meconium was found in distal airways(0.2mm) of all rabbits including 2 that died at 15min post-insufflation. These data suggest that acute meconium insufflation produces a check valve effect($R_E >> R_I$)resulting in increased FRC. Blood gas and ΔA-a results reflect ventilation-diffusion abnormalities secondary to lung mechanics dysfunction. (Supp. by USPHS HL19402).