

mean PO_2 dropped from 102 to 35 mm Hg, and mean Cl prot. increased 38 % (range 15 to 49 %) over control values. Cl prot. returned to control values on return to 21 % O_2 ventilation in 3 pups, and remained elevated in 2. Arterial pH was maintained at normal levels throughout the experiments. Transpulmonary artery wedge pressure was similar during periods of ventilation with 21 % and 10 % O_2 . Thus a hemodynamic basis for the increase in Cl prot. associated with hypoxemia is unlikely. These data indicate that PCP, which is already increased as a handicap of immaturity in pups, is further increased by severe hypoxemia. Similar studies are in progress on adult dogs. (SPR)

- 4 *Effect of Alternations of Pulmonary Arterial and Alveolar Gas Tensions on the Pressure-volume Curve and Surface Tension of Dog Lungs.* EDMUND E. FARIDY* and VICTOR CHERNICK, Dept. of Peds. and Physiology, Univ. of Manitoba, Winnipeg, Canada.

The effect of acute alterations of pulmonary blood flow (Q_p), pulmonary arterial blood gas tensions ($PVCO_2$; PVO_2) and alveolar gas tensions on the mechanical properties of lungs has been studied in left lower lobes of open-chest dogs. Static deflation pressure-volume curves and the stability ratio (SR) of bubbles expressed from the lung were measured. In non-ventilated lobes, the absence of circulation for 4 hours did not produce a change in the percent of maximum air volume remaining at a transpulmonary pressure of 10 cm H_2O ($V_{\%10}$). In non-ventilated lobes, perfusion with hypercapnic hypoxic blood ($PPVO_2 > 60$ mm; $PVO_2 < 45$ mm) caused a significant decrease in $V_{\%10}$. In lobes ventilated with 100 % nitrogen and perfused with hypercapnic hypoxic blood, $V_{\%10}$ decreased more than in the non-ventilated lobes. Change in $V_{\%10}$ could be reversed by perfusing with normal $PVCO_2$ and PVO_2 for 3 hours. In contrast, $V_{\%10}$ was unaltered in lobes perfused with blood having normal PV gas tensions and ventilated with 100 % nitrogen, even when Q_p was decreased to 15 to 20 % of control values. However, $V_{\%10}$ decreased in lobes perfused with blood having normal gas tensions but ventilated with 10 % CO_2 in nitrogen. A decrease in $V_{\%10}$ correlated with a decrease in SR. These studies suggest that the presence of normal surfactant depends upon the intimate gaseous environment of the alveolar cell. Both hypercapnia and hypoxia are required for the production of increased retractive forces on lung deflation. Reversal to normal deflation characteristics is possible by reverting to normal gas tensions. (Supported by the Medical Research Council of Canada) (SPR)

- 5 *Effect of Magnesium on Pulmonary Vasomotor Response to Hypoxia.* GERD J. CROPP*, Univ. Colo. Med. Ctr., Denver, Col. (introduced by Frederick C. Battaglia).

Magnesium relaxes constricted smooth muscle in systemic arterioles and bronchioles, and alveolar hypoxia causes pulmonary vasospasm. Since pulmonary vasoconstriction strains the right ventricle and causes shunting of blood away from the lung in the neonatal period, prevention of hypoxic pulmonary vasospasm would be beneficial; drugs used up to date to overcome this vasospasm have had undesirable side effects. We therefore determined the effect of intravenous infusions of isotonic, buffered $MgCl_2$ in blocking hypoxic pulmonary vasospasm. Anesthetized dogs breathed 100 % O_2 , followed by 7 % O_2 , 10 % O_2 or 10 % O_2 6 % CO_2 . Systemic and pulmonary arterial pressures, heart rate,

cardiac output (CO), respiration, plasma Mg and arterial pO_2 , pCO_2 and pH were measured during high and low O_2 breathing, before and following infusion of $MgCl_2$. All dogs showed a marked rise in pulmonary vascular resistance (PVR) during hypoxia at normal blood Mg levels. As Mg-concentrations increased the rise in PVR during hypoxia lessened. At levels above 10 mEq/l hypoxic pulmonary vasoconstriction was usually absent. [Mg] of less than 13 mEq/l did not decrease resting CO or the CO response to hypoxia and did not cause hypotension or hypoventilation. [Mg] above 15 mEq/l produced hypoventilation and areflexia. It is concluded that controlled elevation of blood Mg to 10–12 mEq/l will block hypoxic pulmonary vasoconstriction without causing deleterious changes in hemodynamics or pulmonary ventilation. (Sponsored by American Heart Association) (SPR)

- 6 *Ventilation/Perfusion Relationships in the Lungs of Children with Congenital Heart Disease.* MARTIN H. LEES*, R. CLIFTON WAY* and BENJAMIN B. ROSS*, University of Oregon Med. Sch., Portland, Ore. (introduced by Richard Goldbloom).

The large increases and reductions of pulmonary blood flow (Q_p) observed in congenital heart disease must challenge the lungs in their function of maintaining physiologic respiratory gas tensions in arterial blood. If ventilation (V_A) and Q_p are not well matched throughout the lung, alveolar-arterial respiratory gas tension differences result. We have examined the extent of uneven V_A/Q_p ratio distribution by the alveolar-arterial O_2 difference (A-a DO_2) breathing air and 100 % O_2 (10 subjects) and the steady-state air-breathing arterial-alveolar N_2 difference (a-ADN $_2$) (21 subjects). Arterial N_2 tension was assessed by gas chromatographic determination of urine N_2 tension (PuN $_2$). Normal subjects had air breathing A-a DO_2 of 10 ± 2.7 mm Hg and 16 ± 10 mm Hg breathing 100 % O_2 . Those with pulmonary overperfusion had A-a DO_2 of 22.4 ± 8.6 mm Hg on room air and 135 ± 48 mm Hg on 100 % O_2 suggesting that intrapulmonary venous admixture rather than V_A/Q_p unevenness was the major cause of lowered arterial O_2 tension. Urine-alveolar N_2 difference (U-ADN $_2$) in normal subjects was 7.6 ± 4.3 mm Hg. U-ADN $_2$ was 3.3 ± 6.4 mm Hg in those with overperfused lungs suggesting very even V_A/Q_p ratio distribution. Children with underperfused lungs had U-ADN $_2$ of 14 ± 7.7 mm Hg indicating considerably increased V_A/Q_p ratio unevenness. We conclude that pulmonary overperfusion is associated with rather even matching of ventilation to perfusion throughout the lung. Diminished pulmonary blood flow (with low pulmonary arterial pressure) results in poor matching of ventilation to perfusion, probably because of selective perfusion of lower lung zones and overventilation of upper zones—an exaggeration of the normal effect of gravity. (SPR)

- 7 *Airway Obstruction in Cystic Fibrosis.* ROBERT B. MELLINS*, O. ROBERT LEVINE*, ALFRED P. FISHMAN* and CAROLYN R. DENNING*, Columbia-Presbyterian Med. Ctr., New York, N.Y. (introduced by Edward C. Curnen).

The ineffectiveness of cough in cystic fibrosis (CF) has been attributed to the viscosity of the bronchial secretions. In the present study, the contribution of abnormal lung mechanics to airway obstruction was investigated in 13 patients with CF, using 8 normal