# *Literature Briefs*

*C. Philip Larson, Jr., M.D., Editor*

*Literature Briefs were submitted by Drs. A. Boutros, D. R. Bucchel, W. Mannheimer, D. H. Morrow, F. C. McPartland, J. W. Pender and H. Roe. Briefs appearing elsewhere in this issue are part of this column.*

## Circulation

**EXTRACELLULAR FLUID** Administration of furosemide to 17 hypertensive subjects resulted in a decrease in mean arterial pressure (17 per cent), a diuresis of 1,920 ml, and a decrease in plasma volume (10 per cent). Infusion of 5 per cent glucose in excess of the volume of urine formed was required to restore blood pressure and responsiveness to norepinephrine. The excess glucose solution reexpanded the extracellular space without correcting negative sodium volume or plasma volume deficit. These results further document the importance of the extracellular fluid in the regulation of arterial pressure. (*Davidov, M., and others: Relation of Extracellular Fluid Volume to Arterial Pressure during Drug Induced Saluresis, Circulation 50: 349 (Sept.) 1969.*)

## Respiration

**PULMONARY ARTERY LIGATION** The amount of hemorrhagic pulmonary consolidation following unilateral pulmonary artery ligation was much less in dogs which breathed 5 to 6 per cent carbon dioxide in air for as long as ten days postoperatively than in control dogs which breathed air or which, in addition, received continuous infusions of isoproterenol. Carbon dioxide inhalation maintained bronchodilatation and increased ventilation, minimizing the incidence of atelectasis, which predisposed to hemorrhagic consolidation. Surfactant activity and pressure–volume characteristics of surviving lung were not affected by ligation of the pulmonary artery. (*Edmunds, L. H., and Holm, J.: Effect of Inhaled $CO_2$ on Hemorrhagic Consolidation due to Unilateral Pulmonary Artery Ligation, J. Appl. Physiol. 26: 710 (June) 1969.*)

**BREATH-HOLDING** A previous model for the control of breath-holding demonstrated that the total drive to resume breathing has two dynamic components, one of which is linearly related to the increasing $P_{CO_2}$ without any threshold, while the other is a time-dependent non-chemical component arising from the absence of normal respiratory movements. A more complicated interaction is now suggested. The relationships between initial and breathing-point $P_{CO_2}$ values and breath-holding times at different lung volumes in ten subjects were estimated. Alveolar $P_{CO_2}$ was set at different levels by periods of rebreathing from a spirometer filled with 5 to 8 per cent $CO_2$ in oxygen. The subject held his breath to a breaking point at the appropriate lung volume at a $P_{O_2}$ that was always higher than 180 torr. In most instances, the overall relationship between initial and breaking point $P_{CO_2}$ values and breath-holding times were nonlinear. A plot of breath-holding time against alveolar $P_{CO_2}$ demonstrated an inflection point, which may represent a threshold for $CO_2$ comparable to the one seen in steady-state ventilation, below which $CO_2$ makes no contribution to the drive to breathe. (*Patrick, J. M., and Reed, J. W.: The Interaction of Stimuli to Breathing during Breath-holding, J. Physiol. 203: 76P (July) 1969.*)

**DISTRIBUTION OF INSPIRED GAS** Effects of varying inspiratory flow rates on intrapulmonary distribution of inspired gas were studied in healthy volunteers by measuring concentration gradients of $^{133}Xe$ down the lung using external counters and also by recording shape of the alveolar plateau during exhalation. When inspiration was begun from residual volume, inspired gas was distributed preferentially to the apices of the lungs; this effect was much more pronounced during slow inspiration. When inspiration started at higher lung volumes (*e.g.*, 40 to 50 per cent of vital capacity) slow inspiratory flow rates caused a uniform increase in $^{133}Xe$ concentration down the lung, with basal segments having higher