# BRIEFS FROM THE LITERATURE

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Briefs were submitted by Drs. John Adriani, C. M. Ballinger, Norman Bergman, Peter Bosomworth, Gaylord Buchanan, M. T. Clarke, Deryck Duncalf, J. E. Eckenhoff, Martin Helrich, J. J. Jacoby, S. J. Martin, S. R. Oech, R. E. Ponath, A. D. Randall, R. W. Ridley, Wallace Ring, and H. S. Rottenstein. Briefs appearing elsewhere in this issue are a part of this column.

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**CARBON DIOXIDE** Factors producing a change in expiratory carbon dioxide levels in infants and children include elevation and depression of body temperature, alkalosis, and acidosis. Changes in pulmonary blood flow are reflected by changes in the carbon dioxide level of expired gas. Reduction in pulmonary blood flow with return toward normal was noted when acute severe hemorrhage with subsequent transfusion occurred. Changes in cardiac output may be followed by continuous monitoring of expired carbon dioxide. Respiratory factors likely to produce changes in expired carbon dioxide include increased dead space in the anesthetic equipment, exhausted soda lime, ineffective valves, overdose of drugs, abdominal distention, collapsed lung, atelectasis and obstructed airway. (*Leigh, M. D., and others: Expired Carbon Dioxide as Continuous Guide of Pulmonary and Circulatory Systems During Anesthesia and Surgery, J. Thor. Cardiov. Surg. 41: 597 (May) 1961.*)

**pH AND CARBON DIOXIDE** Increasing the alveolar carbon dioxide tension from 44 to 50 mm. of mercury caused an increase in the minute volume of ventilation. When the alveolar carbon dioxide tension was maintained at 50 mm. of mercury while the resulting acidemia was reversed by intravenous infusion of bicarbonate, 45 per cent of the respiratory stimulation was removed. The residual respiratory stimulation was correlated with *p*H changes in the spinal fluid and possibly other extravascular sites. Although respiratory changes do occur when blood *p*H is altered without change of blood $P_{CO_2}$, comparable stimulant effects of molecular carbon dioxide cannot be demontrated without producing a simultaneous alteration of *p*H at some site. Cerebral blood flow index varied directly with changes in blood $P_{CO_2}$ and did not respond to any change in *p*H not caused by a change in blood $P_{CO_2}$. (*Lambertsen, C. J., Semple, S. J. G., Smyth, M. G., and Gelfand, R.: $H^+$ and $P_{CO_2}$ as Chemical Factors in Respiratory and Cerebral Circulatory Control, J. Appl. Physiol. 16: 473 (May) 1961.*)

**ALVEOLAR-CAPILLARY BLOCK** In 7 patients with varying degrees of alveolar capillary membrane block, a good correlation was found between the respiratory impairment and the circulatory impairment. The changes included elevation in pulmonary artery pressure during exercise, inadequate augmentation of cardiac output when compared with oxygen uptake, and some evidence of increased pulmonary arteriolar resistance. (*Tabakin, B. S., and others: Circulatory and Ventilatory Dynamics in Alveolar-Capillary Membrane Block, Amer. Rev. Resp. Dis. 83: 194 (Feb.)1961.*)

**AIRWAY RESISTANCE** In a series of 14 normal subjects and 14 patients with chronic pulmonary disease, measurements were made of airway resistance, functional residual capacity, various partitions of lung volume and the maximal mid-expiratory flow. Increases in air resistance to twice normal were produced in both control patients and those with pulmonary disease by inhalation of constrictive aerosols. Inhalation of a sympathomimetic