Literature Briefs

John W. Pender, M.D., Editor

Briefs were submitted by Drs. C. M. Ballinger, Peter P. Bosomworth, M. T. Clarke, Deryck Duncalf, J. E. Eckenhoff, A. Fayard, Martin Helrich, G. Hohmann, J. J. Jacoby, F. C. McPartland, W. H. Manheimer, S. R. Oech, A. S. Paterson, R. E. Ponath, Alan D. Randall, Norman Rosenbaum, P. H. Sechzer, W. H. Ring, and H. S. Rottenstein. Abstracts of Japanese articles were prepared by Excerpta Medica Foundation. Briefs appearing elsewhere in this issue are part of this column.

PROFOUND HYPOXIA Brief profound hypoxia was induced by voluntary overventilation during nitrogen breathing in three healthy men aged 33 to 38. Unconsciousness ensued when this procedure was performed for longer than 16 seconds. Voluntary overventilation with nitrogen for 16 seconds reduced end-tidal oxygen tension to below 10 mm. of mercury for 8 seconds. Minimal arterial oxygen tension was calculated to be 16 mm. of mercury. Therefore, there was a reversal of the normal alveolar-arterial oxygen tension difference. Venous blood flowing through the jugular bulb, the femoral vein, and pulmonary artery were analyzed and recorded continuously for oxygen saturation and *p*H. Oxygen tension of the jugular blood exhibited the most rapid and profound reduction when nitrogen was inhaled. Femoral vein oxygen tension exhibited only a very transient and slight fall. Oxygen tension of blood flowing through the pulmonary artery exhibited a moderate fall. (*Ernsting, J.: Effect of Brief Profound Hypoxia Upon the Arterial and Venous Oxygen Tension in Man, J. Physiol. 169: 292 (Nov.) 1963.*)

RESPIRATORY FAILURE Increase in pulmonary ventilation in response to hypoxia is mediated by peripheral chemoreceptors. Renal adaptation occurs over several days during which the acid-base balance may be shifted to the alkaline side in acute hypoxia, or to the acid side, when there is also carbon dioxide retention. The cardiovascular adjustments to hypoxia include: (1) an increase in cardiac output and an increase in blood flow through dilated coronary vessels, which satisfies the demand of the myocardium for extra oxygen; (2) changes in the caliber of blood vessels, the majority of which dilate. Cerebral, coronary and limb blood vessels increase in caliber; however, the combination of hypoxia and increased carbon dioxide constricts pulmonary arterioles. The adrenal medulla liberates increased amounts of catecholamines, which in turn stimulate respiration and the heart. Many tissues adapt by metabolic changes involving a process of anaerobic glycolysis producing lactic acid, which in turn stimulates respiration and the heart. Carbon dioxide retention may be a major factor in producing a breakdown in the respiratory response to hypoxia. First evidence of respiratory failure is usually cerebral with a variety of mental changes including irritability, confusion and delirium. Eventually, convulsions may occur with coma and death. This may further strain the heart, leading to heart failure and a precipitous fall in blood pressure, usually very difficult to treat. Renal failure and liver damage may result from either hypoxia or carbon dioxide retention. Patients with chronic respiratory disease obtain relief from intolerable dyspnea with oxygen. (*Penman, R. W. B.: Hypoxia in Respiratory Failure, Geriatrics 18: 807 (Nov.) 1963.*)

CO_2 DRIVE IN HYPOXIA The threshold of alveolar-arterial P_{CO_2} as a respiratory stimulant was examined by observing phrenic nerve discharges in the presence of acute hypoxia in anesthetized dogs. It was found that the threshold for P_{CO_2} decreased as the P_{O_2} decreased. The relationship was curvilinear. Furthermore, in the absence of an adequate P_{CO_2} drive, there was no hypoxic drive; thus

during intense hypoxia, the respiratory drive continues to be a consequence of the P_{CO_2} drive, even when the P_{CO_2} has been reduced far below normal. (*Honda, Y., and others: P_{CO_2} for Respiratory System in Acute Hypoxia of Dogs, J. Appl. Physiol. 18: 1053 (Nov.) 1963.*)

CARBON DIOXIDE Twenty-one subjects were exposed to 1.5 per cent carbon dioxide in air for 42 days. Respiratory minute volume and alveolar P_{CO_2} were increased throughout the exposure to carbon dioxide. Carbon dioxide retention with uncompensated respiratory acidosis persisted for the first 23 days of the experiment. After transition back to air, the respiratory minute volume decreased while the P_{CO_2} remained elevated for nine days. Carbon dioxide excretion was increased over normal during the nine day recovery period indicating a release of carbon dioxide from stores. (*Shaefer, K. E., and others: Respiratory Acclimatization to Carbon Dioxide, J. Appl. Physiol. 18: 1071 (Nov.) 1963.*)

HYPERVENTILATION Intermittent positive-negative respiration in 10 dogs resulted in a consistent reduction in arterial carbon dioxide content and rise in blood *p*H. This was accompanied by a fall in cardiac output and blood pressure which remained at a constantly depressed level. An immediate rise in renal arterial blood flow resulted which later fell below control values indicating a marked preferential increase in renal blood flow. (*Pollock, L., and others: Influence of Hyperventilation on Cardiac Output and Renal Blood Flow, Surgery 55: 299 (Jan.) 1964.*)

CONTROLLED VENTILATION Utilizing an open circuit nitrogen washout method, distribution in the lung of inspired gas was compared during spontaneous ventilation in the conscious and the anesthetized patient and also in the artificially ventilated, anesthetized, paralyzed patient. Induction of anesthesia or substitution of artificial ventilation for spontaneous ventilation caused no change in the uniform distribution of inspired gas. (*Bergman, N. A.: Distribution of Inspired Gas During Anesthesia and Artificial Ventilation, J. Appl. Physiol. 18: 1085 (Nov.) 1963.*)

COMPLIANCE AND RESPIRATORY RATE Measurements of pulmonary compliance were made at different respiratory frequencies in adult man. It was found that compliance decreased with an increasing respiratory frequency. The effect was greatest at low lung volumes, least in middle lung volumes, and decreased again at greater lung volumes. (*Mills, R. J., Cumming, G., and Harris, P.: Frequency-Dependent Compliance at Different Levels of Inspiration in Normal Adults, J. Appl. Physiol. 18: 1061 (Nov.) 1963.*)

RESPIRATORY EFFICIENCY Anesthetized paralyzed dogs were given intermittent positive pressure ventilation to a constant maximum inflating pressure using four separate pressure profile curves. The four profiles were: (1) rapid inspiration and rapid expiration; (2) slow inspiration, rapid expiration; (3) rapid inspiration, prolonged expiration; and (4) prolonged inspiration, prolonged expiration. The smallest alveolar-arterial gradients, both oxygen and carbon dioxide, occurred when the mean pressure during the respiratory cycles was high, and it was noted that decreases in mean pressure were consistently associated with increases in both oxygen and carbon dioxide gradients. A significantly high mean pressure during the respiratory cycle might be beneficial and lowering of the mean pressure during intermittent positive pressure breathing in an attempt to minimize circulatory effects might compromise respiratory efficiency. (*Bergman, N. A.: Effects of Different Pressure Breathing Patterns on Alveolar-Arterial Gradients in Dogs, J. Apply. Physiol. 18: 1049 (Nov.) 1963.*)

PULMONARY EFFUSION Pleural effusion reduces chest wall compliance slightly. Pleural thickening with effusion is accompanied by marked reduction in chest wall and lung compliance and reduction in lung volumes and diffusing capacity. Resorption of fluid leads to increasing lung volumes. Decortication may or may not reduce the changes due to pleural thickening. In patients with effusion and thickening, arterial blood gas values at rest are normal. (*Yoo, O. H., and Ting, E. Y.: Effects of Pleural Effusion on Pulmonary*