BRIEFS FROM THE LITERATURE

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VENTILATION DURING SLEEP The changes in alveolar carbon dioxide tension, pulmonary ventilation, respiratory gas exchange, mechanics of breathing and blood *p*H were studied in normal subjects during sleep. Physiologic sleep in the normal subject is associated with a significant fall in total and alveolar ventilation. There was a concomitant significant rise in alveolar carbon dioxide tension and a fall in carbon dioxide production and oxygen consumption. The ventilatory response to carbon dioxide is also reduced. A fall in blood *p*H with a considerable incidence of periodic breathing was observed. There was no significant alteration in alveolar oxygen tension, arterial oxygen saturation, respiratory exchange ratio or the mechanics of breathing during normal sleep. Carbon dioxide retention of sleep is related to a depressed respiratory center sensitivity. The speed of onset and reversal of this depression of the respiratory center sensitivity suggests that it is probably mediated neurogenically. The theory of the genesis of sleep based on changes in blood gas constituents is probably untenable. (*Robin, E. D., and others: Alveolar Gas Tensions, Pulmonary Ventilation and Blood pH During Physiologic Sleep in Normal Subjects, J. Clin. Invest. 37: 981 (July) 1958.*)

PULMONARY FUNCTION Alveolar hypoventilation was associated with myxedema or lung disease. Most of the obese persons in this study with normal lungs, normal respiratory muscles and an apparently normal respiratory center did not have alveolar hypoventilation. Obese persons with pulmonary insufficiency are benefited by weight loss. This improvement is caused by reduction in the work of breathing. (*Bedell, G. N., Wilson, W. R., and Seebohm, P. M.: Pulmonary Function in Obese Persons, J. Clin. Invest. 37: 1049 (July) 1958.*)

PULMONARY INSUFFICIENCY Patients with chronic pulmonary insufficiency show a rise in hematocrit but not in hemoglobin levels. The phenomenon seems to be explained by the entrance of water into the cells. (*Grant, J. L., and others: Red Cell Changes in Chronic Pulmonary Insufficiency, J. Clin. Invest. 37: 1166 (Aug.) 1958.*)

CARBON DIOXIDE The changes in tissue carbon dioxide content during acute respiratory acidosis was studied in albino rats. These animals were exposed to 24 per cent carbon dioxide in air for periods ranging from one-half to 40 hours and measurements were made of *p*H and carbon dioxide content of blood and tissues. A profound respiratory acidosis with high plasma carbon dioxide and a plasma *p*H of 6.92 appeared after one-half hour of exposure. This was followed after 7 to 15 hours by a further slow rise in plasma carbon dioxide and a rise in *p*H which reached 7.10 after 48 hours. The rate of rise and the absolute level of tissue carbon dioxide was higher in brain than in muscle. A profound acidosis developed rapidly in both brain and muscle. In contrast to plasma, however, there was no evidence of adaptation with return of *p*H toward normal levels up to 48 hours of exposure. (*Nichols, G. Jr.: Serial Changes in Tissue Carbon Dioxide Content During Acute Respiratory Acidosis, J. Clin. Invest. 37: 1111 (Aug.) 1958.*)

HYPERVENTILATION In 20 individuals, hyperventilation regularly caused the appearance of signs and symptoms of tetany, but the ECG showed only minor alterations of the T