BRIEFS FROM THE LITERATURE

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PULMONARY RECEPTORS To clarify the presence and nature of pressoreceptors in the pulmonary vascular bed, the left lung of the intact dog was perfused with normal saline under variable pressures and flow rates. About 6 to 7 seconds after acute hypertension was induced in the pulmonary vascular bed, bradycardia, systemic hypotension and depression of respiration occurred simultaneously. The presence of an intact vagus seems necessary as the response was not elicited following ipsilateral (but not contralateral) vagotomy. Also required is an impedance to flow in the distal pulmonary venous bed, suggesting that the majority of the receptors are located in the distal vasculature. (*Downing, S. E.: Reflex Effects of Acute Hypertension in Pulmonary Vascular Bed of Dog, Yale J. Biol. & Med. 30: 43 (Sept.) 1957.*)

PULMONARY EMPHYSEMA Changes in minute volume, *p*H, oxygen and carbon dioxide tension in arterial blood, physiologic dead space and alveolar ventilation were determined following the inhalation of pure oxygen in 20 normal subjects and in 20 emphysematous patients. In normal subjects, the changes are in the range of normal. Following oxygen inhalation by emphysematous patients, the minute volume decreased; the oxygen saturation was or became normal; the carbon dioxide tension increased with concomitant decrease in *p*H; the respiratory dead space increased in proportion to the reduced alveolar ventilation. Progressive increase in the respired oxygen concentration and use of artificial respirators that create a positive pressure during the initial phase of the inspiration are suggested as therapeutic measures. (*De Coster, A., and Denolin, H.: Respiratory Changes Following Inhalation of Pure Oxygen in Normal Subjects and in Patients with Pulmonary Emphysema, Rev. fr. clin. biol. 2: 129 (Feb.) 1957.*)

PHYSIOLOGIC RESPIRATOR Design of an electronic device for the control of mechanical respirators in accordance with the physiologic need of the individual is presented. Primary muscles of respiration stimulated by the respiratory center itself were found to be most effective in triggering the electronic device, but even minor accessory muscles such as the platysma or the ala nasi have been used successfully for many hours in anesthetized animals. Partially paralyzed patients have been able to rely upon the electronic control for their respiration for periods of more than 24 hours at a time, including long periods of normal sleep during which it was not necessary to adjust the skin electrodes or the controls of the electronic circuit. (*Batson, R., and others: Electronically Controlled Respirator, Science 126: 819 (Oct. 25) 1957.*)

OBESITY DYSPNEA The basic pathologic physiology of the well-documented clinical entity "cardiorespiratory syndrome of extreme obesity" is apparently the increased mechanical work of breathing caused by large deposits of fat on the chest wall and in the abdomen. Alveolar hypoventilation leads to arterial hypoxemia and hypercapnia. The hypoxemia in turn produces cyanosis, secondary polycythemia and pulmonary hypertension, usually with electrocardiographic evidence of right ventricular overload. The hypercapnia leads to somnolence and decreased sensitivity