BRIEFS FROM THE LITERATURE

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Briefs were submitted by Drs. C. M. Ballinger, L. E. Binder, M. T. Clarke, R. A. Devloo, Cody Eames, D. W. Eastwood, J. E. Eckenhoff, S. J. Martin, R. E. Ponath, R. W. Ridley, and H. S. Rottenstein.

PULMONARY PHYSIOLOGIC DEAD SPACE The anatomic and physiologic dead space was measured in 25 patients with moderate to severe emphysema. Exercise increased the mean resting physiologic dead space of 333 ml. to 443 ml., and decreased the mean resting anatomic dead space of 203 ml. to 200 ml. A large physiologic dead space indicates a marked decrease of the volume of the alveolar vascular bed with increased velocity of pulmonary capillary blood flow in the presence of normal cardiac output. (*Wilson, R. H., and others: Pulmonary Physiologic Dead Space as Index of Effective Alveolar Perfusion, Am. J. M. Sc. 234: 547 (Nov.) 1957.*)

POSITIVE PRESSURE BREATHING High levels of positive pressure breathing in the dog produced substantial decrease in central blood volume as well as in cardiac output and arterial pressure. The administration of metaraminol improved these lowered values while positive pressure breathing was maintained. Beneficial effects were attributed to the drug influence on venous and arteriolar tone and myocardial contractility. (*Braunwald, E., and others: Alteration in Central Blood Volume and Cardiac Output Induced by Positive Pressure Breathing and Counteracted by Metaraminol, Circulation Res. 5: 670 (Nov.) 1957.*)

PULMONARY GAS EXCHANGE Pulmonary gas exchange occurs in three steps: exchange within the alveolar gas, exchange across a pulmonary membrane, and diffusion and chemical combination within the pulmonary capillary blood. This review deals with the relative importance of these three steps, methods of measurement and the normal values of some of their parameters, and the application of these methods to disease of the lung. (*Forster, R. E.: Exchange of Gases Between Alveolar Air and Pulmonary Capillary Blood: Pulmonary Diffusing Capacity, Physiol. Rev. 37: 391 (Oct.) 1957.*)

PULMONARY EDEMA Unilobar pulmonary edema was produced in the dog by infusion of blood into a lobe the arterial inflow and venous outflow of which were occluded. High levels of pulmonary venous pressure often were reached before pulmonary edema supervened. No constant relationship between pulmonary venous pressure and the development of edema was evident under these experimental conditions. These findings support the concept that pulmonary edema should not be considered entirely in terms of left ventricular failure and changes in pulmonary venous pressure. (*Shedd, D. P., and Gray, F. D., Jr.: Experimental Unilobar Pulmonary Edema, Yale J. Biol. & Med. 30: 110 (Nov.) 1957.*)

VENTILATION STIMULUS Two different theories give an interpretation of the increase in ventilation observed at high altitudes in subjects: Haldane states that the low oxygen tension is the constant factor causing the hyperventilation, while Bjurstedt and Rahn state that only initially low oxygen tension causes the hyperventilation, and once the $BHCO_3/CO_3$ relationship has adjusted itself again to the normal ratio, ventilation returns under the control of the carbon dioxide stimulus, and the low oxygen tension plays only a minor role. The effects of hyperoxygenation were investigated in three subjects at 11,000 feet altitude after a few days of acclimatization. After twenty seconds of in-