(*Parmley, L. F., Jr., North, R. L., and Pickens, G. E.: Pulmonary Embolism as a Cause of Systemic Hypotension and Shock, Amer. J. Cardiol. 15: 333 (Mar.) 1965.*)

HYPERBARIC OXYGENATION Hyperbaric oxygenation was used in the treatment of purpura gangrenosa occurring in a 19 month old Negro child. Within three days, eight five-hour exposures were given at two atmospheres absolute pressure. The child was placed in a pediatric high humidity oxygen tent, and this was in turn placed inside the treatment compartment of the hyperbaric chamber. During pressurization the oxygen tension of inspired air exceeded 1,400 mm. of mercury. The tent was usually opened during the midpoint of each five-hour period for 15 to 20 minutes for medication, nursing care, and to interrupt pulmonary exposure to extreme hyperoxia. Respiration of oxygen at increased atmospheric pressures for prolonged periods is associated with a specific, progressive picture of pulmonary insufficiency terminating in death. However, patients have not demonstrated recognizable symptoms of pulmonary toxicity when breathing oxygen at two atmospheres pressure. (*Waddell, W. B., and others: Purpura Gangrenosa Treated with Hyperbaric Oxygenation, J.A.M.A. 191: 971 (Mar. 22) 1965.*)

PULMONARY EMBOLISM The right heart of dogs was bypassed using a reservoir-pump system. Following experimental pulmonary embolization with glass microspheres or autologous clots, pulmonary artery pressure and pulmonary vascular resistance increased while arterial oxygen saturation decreased. Calculated venous admixture increased to 30 per cent of cardiac output. Imposition of 20 cm. of water resistance to exhalation restored saturation and admixture to pre-embolization values. Decrease in hemoglobin saturation in pulmonary embolism is caused by perfusion of poorly or nonventilated areas of lung caused by collapse of respiratory units due to presence of edema fluid. Pulmonary hypertension following pulmonary embolization may be due to vasoconstriction in addition to mechanical blocking of pulmonary vessels. (*Caldini, P.: Pulmonary Hemodynamics and Arterial Oxygen Saturation in Pulmonary Embolism, J. Appl. Physiol. 20: 184 (Mar.) 1965.*)

EMBOLI During extracorporeal circulation a wide variation in tolerance to air emboli was found in dogs. Air passed rapidly through the coronary capillaries in some while forming a permanent obstruction in others. Carbon dioxide emboli, though causing much less injury than air, were not completely innocuous. The most effective method for prevention of air emboli is the use of induced ventricular fibrillation in combination with decompression of the left ventricle. If emboli have occurred, increasing the perfusion pressure and manually massaging the heart is usually effective. (*Spencer, F. C., and others: Significance of Air Embolism During Cardiopulmonary Bypass, J. Thor. Cardiov. Surg. 49: 615 (Apr.) 1965.*)

PULMONARY EMBOLI Transient pulmonary hypertension lasting up to an hour can be produced in dogs by the introduction of air into the pulmonary artery. This hypertension is due to mechanical increase in pulmonary vascular resistance by the surface tensions of the many bubbles in small vessels, which also cause temporary decrease in compliance and decrease in pulmonary function. A major portion of the vascular bed needs to be obstructed by air emboli before any pressure change is apparent. Pulmonary air embolism during open heart surgery, although tolerated in a person with normal lungs, may result in significant increase in pulmonary hypertension at the critical time of bypass termination in patients with preoperative pulmonary vascular changes. (*Anderson, R. M., and others: Pulmonary Air Emboli During Cardiac Surgery, J. Thor. Cardiov. Surg. 49: 440 (Mar.) 1965.*)

HYPOXIA In chronic hypoxia at high altitudes cardiac output is not increased because of a compensatory increase in oxygen-carrying capacity of blood. Increased volume of ventilation was achieved entirely by greater tidal volume; however, the number of moles of air ventilated actually decreased. Basal oxygen uptake increased slightly. With exercise, at any given level of oxygen uptake, the volume of air ventilated was greater at high altitude.

At high altitude significant pulmonary hypertension presumably causes better perfusion of the normally poorly perfused apical segments thereby facilitating oxygen transfer from alveoli to blood. (*Grover, R. F.: Effects of Hypoxia on Ventilation and Cardiac Output, Ann. N. Y. Acad. Sci. 121: 662 (Mar.) 1965.*)

ACIDOSIS Carbon dioxide breathing caused a greater increase in minute ventilation than acute metabolic acidosis in normal subjects and in patients with obstructive disease of the lung. Augmentation of alveolar ventilation in normal subjects during carbon dioxide breathing becomes relatively greater with increasing hypoxia. Hypercapnea causes diffuse reflex bronchoconstriction in animals which may permit an optimal balance between anatomic dead space and resistance to efficient breathing. Blood hydrogen ion concentration, by altering vasomotor tone of the pulmonary vasculature, affects the distribution of perfusion of the lungs at least in those conditions where there is considerable initial nonhomogeneity of distribution and oxygen transport. (*Enson, Y.: Effects of Acidosis on Respiratory Function, Ann. N. Y. Acad. Sci. 121: 674 (Mar.) 1965.*)

RESPIRATORY DEAD SPACE During bronchospirometry after introduction of a Carlens tube in conscious patients breathing spontaneously, the tube leading to one lung was lengthened by another tube thus creating a dead space of 25 to 50 ml. Alveolar hypoventilation with increased P_{CO_2} and decreased P_{O_2} was found on the dead space side, while the other side showed hyperventilation. (*Hertz, C. W.: Research on Gaseous Exchange in the Functional Inhomogenous Lung After Introduction of an Artificial Dead Space on One Side, Deutsch. Arch. Klin. Med. 210: 183 (Mar.) 1965.*)

ALVEOLAR PERMEABILITY Studies were performed on isolated perfused canine lungs to determine the permeability characteristics of the alveolar membrane to several substances. The test substances, which appeared to cross the membrane largely by diffusion rather than active transport mechanisms, were added to the fluid perfusing the vascular bed of the lung, their rate of diffusion into the alveolar spaces being a function of alveolar membrane permeability. Calculated permeability coefficients were potassium 56, sodium 7, urea 23, glucose 3 and dinitrophenol 400 (a fat soluble substance). This selective permeability is similar to that of usual cell membranes in other tissues, but quite unlike simple capillary membranes. (*Taylor, A. E., Guyton, A. C., and Bishop, V. S.: Permeability of the Alveolar Membrane to Solutes, Circulat. Res. 16: 353 (Apr.) 1965.*)

EMPHYSEMA Electron microscopy of emphysematous human lungs shows capillary lesions identical with those found as precursors to the earliest alveolar lesions in rabbit lungs. This strongly suggests that the earliest lesion in emphysema is in capillaries. (*Martin, H. B., and others: Electron Microscopy of Human Pulmonary Emphysema, Amer. Rev. Resp. Dis. 91: 206 (Feb.) 1965.*)

PULMONARY NORMALS In a large total population study, all male cigarette smokers showed a greater decline with age in one-second forced expiratory volume, forced vital capacity and peak expiratory flow rate than nonsmokers or those smoking less than half a pack a day, suggesting that pulmonary standards for nonsmokers be used as representative of normal. (*Ferris, B. G., and others: Prediction Values for Screening Tests of Pulmonary Function, Amer. Rev. Resp. Dis. 91: 252 (Feb.) 1965.*)

CORD PARALYSIS Following injury of cervical portion of spinal cord there is flaccid paralysis of the muscles of the trunk and extremities despite a normally moving diaphragm. This may produce normal or enhanced excursions of the abdomen with paradoxical inspiratory retraction of the rib cage. Since descent of the diaphragm normally accomplishes only 40 per cent of the tidal volume, ventilation may be inadequate. Not infrequently intermittent cyanosis may develop and during acute infections and other forms of stress, respiratory acidosis may occur. Vital capacity and maximum breathing capacity are decreased. In some subjects lowered arterial oxygen tension and elevated arterial carbon dioxide potential are found, indicating alveolar hypoventilation.