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.