or two segments removed and was reduced in those having 3-5 segments removed. Average maximal breathing capacity, per cent of rapid vital capacity expired in one second, the 7 minute alveolar nitrogen and arterial oxygen saturation were essentially unchanged. (*Miller, R. D., and others: Pulmonary Function Before and After Pulmonary Resection in Tuberculous Patients, J. Thoracic Surg. 35: 651 (May) 1958.*)

OXYGEN INTAKE The maximal oxygen intake is dependent on both cardiac output and arteriovenous oxygen difference. The widening of the arteriovenous oxygen difference was due principally to diminution in mixed venous oxygen content. There was no significant change in arterial oxygen tension from rest to heavy work; the slight decrease in oxygen saturation that was observed can be explained by the pH change of the blood and the resulting shift in the oxygen dissociation curve. The venous oxygen tension showed no significant change, even though the venous oxygen content and saturation fell appreciably. The end result of the phenomenon is to maintain an adequate oxygen tension gradient from capillary to cell. In ascertaining the physiologic meaning of the maximal oxygen intake, the relative importance of cardiac capacity and increase in arteriovenous oxygen difference must be determined. It is probable that in the normal individual the ability to increase cardiac output is the more important of the two factors. (*Mitchell, J. H., Sproule, B. J., and Chapman, C. B.: Physiological Meaning of Maximal Oxygen Intake Test, J. Clin. Invest. 37: 538 (April) 1958.*)

HYPOXEMIA On hypoventilation with air CO_2 retention develops and hypoxemia progresses at an increasingly rapid rate. However, significant hypoxemia only begins to appear when the arterial CO_2 tension is over 60 mm. of mercury. Hypoxemia of this sort may be caused by increased metabolism without a concomitant increase in ventilation or by diminished alveolar ventilation. The latter may occur from a depressed total ventilation or by an increased dead space ventilation such as might occur with an inefficient anesthetic apparatus. The ventilatory needs of a patient during anesthesia or in a mechanical respirator may be predicted by calculating the alveolar ventilation necessary to maintain the normal CO_2 tension, measuring or assuming a normal dead space, and then calculating the required total ventilation. (*Williams, M. H., Jr.: Quantitative Relationships Between Hypoxemia and Disorder of Pulmonary Function, Yale J. Biol. & Med. 30: 306 (Feb.) 1958.*)

PULMONARY DIFFUSION The function of the lungs depends upon two phenomena: alveolar ventilation and alveolar diffusion. The diffusing capacity of normal resting lung is sufficient to supply about three times the normal resting O_2 uptake, but this capacity must increase in order to deal with the O_2 uptake required by even moderate exercise. Thus the reserve of diffusing capacity for O_2 is small and respiratory failure may occur if diffusing capacity is impaired by disease. By contrast, CO_2 diffuses over twenty times more rapidly than O_2 so that elimination of CO_2 is never limited by diffusion, and CO_2 retention, when it occurs, is due to inefficient ventilation, not to impaired diffusion. Resistance to pulmonary diffusion is provided by the alveolar membrane which consists of the alveolar epithelium, a complex basement membrane and the capillary epithelium. Any increase in thickness of this membrane or reduction in the number of functioning alveoli or capillaries will reduce diffusing capacity. Normally the alveolar membrane offers about 70 per cent of the total resistance to diffusion of O_2 while the resistance of uptake into the red cells accounts for about 30 per cent. Methods for measuring diffusing capacity utilize carbon monoxide gas (D_{CO}), since measurements of D_{O_2} presents a number of technical difficulties. (*Marshall, R.: Methods of Measuring Pulmonary Diffusing Capacity and Their Significance, Proc. Roy. Soc. Med. 51: 101 (Feb.) 1958.*)

RESPIRATORY INSUFFICIENCY Of those who survive an accident involving coma and head injury, the main cause of death is respiratory insufficiency and anoxia. The anoxia is due to central disturbances of the control of respiration, and reduction of compliance of the lungs by