atelectatic or otherwise nonventilated or that permitted no oxygen diffusion. The findings of increased venous admixture that persisted during oxygen breathing and decreased with deep inspiration are in keeping with the opinion that atelectasis is the major cause of hypoxemia in obesity. The slight or absent fall in venous admixture observed in patients with obstructive emphysema or diffuse pulmonary fibrosis following deep breathing suggests that relatively little or no atelectasis was present. (*Said, S. I., and Banerjee, C. M.: Venous Admixture to the Pulmonary Circulation in Human Subjects Breathing 100 Per Cent Oxygen, J. Clin. Invest. 42: 507 (Apr.) 1963.*)

VENTILATION-PERFUSION Abnormalities in the ventilation-perfusion relationships occurred in 12 patients after limited excisional surgery for pulmonary tuberculosis. Changes were most pronounced in the first postoperative week and lasted no longer than the second week. Likely causes of hypoxia were splinting due to pain, localized increase in airflow resistance due to secretions, and regionally decreased compliance. Elastic attributes of the remaining lung tissue are changed by limited resection or transient edema. Until these responses to trauma resolve, variations in ventilation between different parts of the lung are inevitable, for air entering the bronchial tree will be preferentially distributed away from the less compliant portions of the lung. (*Di Benedetto, A., and others: Effects of Limited Pulmonary Resection on Ventilation-Perfusion Relationships in the Post-operative Period, J. Thor. Cardiov. Surg. 45: 312 (Mar.) 1963.*)

ACID-BASE REGULATION Primary lung disease and hypercapnia may produce alkalotic rather than acidotic values in plasma *p*H. Rapid mobilization of carbon dioxide by forced hyperventilation, hypochloremia and body chloride depletion along with therapeutic administration of alkalizinng agents may account for the mechanism of this paradoxical alkalosis. Laboratory measurements do not necessarily serve to distinguish primary metabolic alkalosis. When the carbon dioxide tension is greater than 95 mm. of mercury in a patient with severe carbon dioxide retention he is invariably acidotic, indicating a fairly sharp upper limit of rise of plasma carbonate concentration. Patients who have moderate chronic elevations of carbon dioxide tension are frequently acidotic, suggesting that the compensatory mechanisms for acid-base regulation under these circumstances must be studied as specific intracellular areas responsible for acid-base regulation. (*Robin, E. D.: Abnormalities of Acid-Base Regulation in Chronic Pulmonary Disease, With Special Reference to Hypercapnia and Extracellular Alkalosis, New Engl. J. Med. 268: 917 (Apr. 25) 1963.*)

EMPHYSEMA Ten subjects with emphysema were studied by cardiac catheterization at rest and during intermittent positive pressure respiration (IPPB) with air. Mean pulmonary artery pressure fell in all but one during the IPPB. The cardiac output increased in four out of five subjects, and there was evidence of a fall in pulmonary vascular resistance in these four subjects. With IPPB the arterial oxygen saturation increased in most of the subjects, but hypoxia was not fully corrected in any of them. The arterial P_{CO_2} decreased in eight of nine subjects. Hemodynamic changes were more closely related to the decrease in arterial P_{CO_2} than to the increase in arterial oxygen saturation. (*Daly, J. J., and Duff, R. S.: Effect of Intermittent Positive Pressure Breathing on Pulmonary Circulation in Emphysema, Brit. Heart J. 25: 47 (Jan.) 1963.*)

DYSPNEA Nervous mechanism of dyspnea remains uncertain, but the simplest arrangement appears to be one in which information concerning chest volume and pressure developed within the chest is correlated. Changes in the chest volume may be appreciated by the individual as a length change, perhaps in terms of the length of the muscle fibers or of the angular rotation of the ribs. Likewise, change in the intrathoracic pressure may be recognized as an alteration in the tension within the muscle fibers. If the length change is inappropriate to the change in tension, dyspnea results. This may be expressed as "length-tension inappropriateness." (*Bennett, D., Jayson, M., and Rubenstein, D.: Perception of Dyspnea, Dis. Chest 43: 411 (Apr.) 1963.*)