

arterial oxygen pressure gradient. Breathlessness brings work to a halt at these altitudes. Breathing oxygen at altitude restored work performance, including cardiac work, almost to normal. Roentgenographic and electrocardiographic studies offered evidence of right ventricular hypertrophy secondary to pulmonary hypertension. Red cell count, hematocrit, and hemoglobin and red cell mass continually increased during three months at high altitude. Water turnover is increased by about one-third due to the increased water loss from the lungs because of hyperventilation of dry air. An altitude of 15,000 to 17,000 feet is probably the maximum that can be tolerated by acclimatized plainsmen. (Pugh, L. G. C. E.: *Physiological and Medical Aspects of the Himalayan Scientific and Mountaineering Expedition, 1960-61*. *Brit. Med. J.* 2: 621 (Sept. 8) 1962.)

THERAPEUTIC HYPOXIA Experimental ventricular tachycardia produced in 20 dogs by ligating the anterior descending coronary artery was ameliorated in all but two animals by exposing them to an hypoxic atmosphere. Inhalation of 5 to 10 per cent oxygen concentrations caused a significant tenfold increase in the percentage of supraventricular beats, while higher concentrations were ineffective. Concentrations below 5 per cent commonly led to ventricular fibrillation. These findings lend indirect support to Brofman's oxygen-gradient theory as the explanation for post-infarct arrhythmias. This hypothesis holds that the ventricular ectopic arrhythmias following myocardial infarction are due to the electrical gradient set up between normally perfused and ischemic heart muscle. The corollary is that a uniformly hypoxic heart is electrically stable. Hazards of applying this concept to man are possible. (Jacobson, E. D., Scheiss, W., and Moe, G. K.: *Effect of Hypoxia on Experimental Ventricular Tachycardia*. *Amer. Heart J.* 64: 368 (Sept.) 1962.)

PULMONARY FUNCTION Before pulmonary surgery, blood gas analyses are indicated in patients whose pulmonary reserve seems diminished. Bronchspirometry is de-

sirable when a decrease in pulmonary reserve is indicated by routine function tests. Right heart catheterization is desirable when pulmonary hypertension is found. "Functional pneumonectomy" by pulmonary artery occlusion of the lung to be removed demonstrates whether or not the patient can survive without the lung. (David, D., and Correll, N.: *Value of Cardiopulmonary Studies in the Evaluation of Patients for Lung Resection*, *Surgery* 52: 523 (Sept.) 1962.)

PULMONARY COMPLIANCE Compliance of the lung in patients undergoing surgery for disease in one segment was determined immediately after the chest was opened and again after resection. The patients were ventilated either manually or with an automatic intermittent positive-pressure ventilator set to deliver a predetermined volume. The preresection compliances of manually and automatically ventilated patients did not differ significantly. Postresection compliances fell equally in both groups. The compliance after ventilation with double tidal volume was usually higher than that with tidal volume plus 200 ml. (Karlson, K. E., and others: *Effect of Volume-Cycled Automatic Ventilation on the Elastic Recoil of the Lung*, *J. Thor. Cardio. Surg.* 44: 189 (Aug.) 1962.)

CHEST COMPRESSION Chest compression was produced in human subjects by inflation of balloons under a tightly fitting corset about the chest. During chest compression functional residual capacity fell 1 liter and decreased lung compliance, increased respiratory rate and alveolar hyperventilation occurred. After release of chest compression, values did not return to normal until a deep breath was taken, suggesting the necessity of re-expanding alveoli which had collapsed during compression. Complex changes occurred in blood gas tensions which could not be explained by any single factor. The hyperventilation observed was probably reflex and related to decreased lung volume. (McIlroy, M. B., Butler, J., and Finley, T. N.: *Effects of Chest Compression on Reflex Ventilatory Drive and Pulmonary Function*, *J. Appl. Physiol.* 17: 701 (July) 1962.)