determined in a series of both normal subjects and cardiac patients. Diffusing capacity was significantly lowered in severe mitral stenosis with pulmonary congestion but was insignificantly affected by mild or moderate cardiac lesions. Capillary blood volume was elevated in severe mitral stenosis and in interatrial septal defects. Aortic valvular lesions had little effect on either of these parameters. (*Flatley, F. J., and others: Pulmonary Diffusing Capacity and Pulmonary Capillary Blood Volume in Normal Subjects and in Cardiac Patients, Amer. Heart J. 64: 159 (Aug.) 1962.*)

PULMONARY EXCHANGE During anesthesia with barbiturates or barbiturates combined with ether there was in some subjects a decrease of arterial oxygen pressure and an increase in the alveolar-arterial oxygen difference. A slight respiratory acidosis developed but there was no significant change in bicarbonate. Functional dead space increased independent of respiratory volume. During anesthesia there may be marked disturbance of distribution and pulmonary shunting in some parts of the lung. (*Reichel, G., and Harrfeldt, H. P.: Investigations Concerning the Disturbances of Gas Exchange in the Lungs Caused by Anesthesia, Der Anaesthesist 11: 231 (July) 1962.*)

APNEA Intravenous administration of a barbiturate causes a short period of apnea if 100 per cent oxygen is being breathed but very little respiratory depression with absence of apnea when room air is being breathed. Sodium bicarbonate, given intravenously, has the same alkalinity and shows identical results. Respiration is not influenced for a short time when the oxygen binding power of the hemoglobin is blocked by carbon monoxide or sodium nitrite but respiration stops as soon as the physically dissolved oxygen has been used up. This shows the importance of the physically dissolved oxygen which together with blood carbon dioxide regulates respiration. If a strongly alkaline solution is given intravenously, blood carbon dioxide is bound. If at the same time by breathing pure oxygen the plasma carries five times more oxygen than normal, then two substances which stimulate respiration are removed. (*Lenggenhager, K.: Explanation of a Strange Phenomenon During Narcosis, Der Anaesthesist 11: 181 (June) 1962.*)

RESPIRATORY CHEMOREFLEXES Existence of a respiratory chemoreflex mechanism requires the demonstration of (1) an anatomical receptor structure and afferent fibers in the brain stem, (2) sensitivity of this receptor in response to normal chemical changes in its environment, and (3) ventilatory changes which may be induced by blocking conduction in afferent fibers. On the basis of studies in both animals and adult humans it is probable that the fall in ventilation during transient hyperoxia and the ventilatory increase during transient hypoxia indicate that there is a chemoreceptor drive of ventilation. In humans, in short-term altitude acclimatization or acute hypoxia a powerful ventilatory chemoreflex oxygen drive exists. Although both chemoreflex carbon dioxide and pH drive have been established, it is not presently possible to differentiate this effect from their centrogenic drive. (*Dejours, P.: Chemoreflexes in Breathing, Physiol. Rev. 42: 335 (July) 1962.*)

EFFECTS OF ALTITUDE At an altitude of 25,000 feet, barometric pressure was 288 mm. of mercury, the inspiratory oxygen tension of warmed saturated air was 50 mm. of mercury, the oxygen tension of alveolar air was 33 mm. of mercury and that of carbon dioxide, 14 mm. of mercury. Arterial oxygen saturation was about 57 per cent at rest. The basal metabolic rate and the ventilatory equivalent for oxygen were the same as at sea level. Cheyne-Stokes respiration was prevalent during sleep. Even after three months at 19,000 feet the main respiratory drive was still furnished by hypoxia. With increase in altitude maximum work, maximum oxygen intake, maximum heart rate and cardiac output declined though maximum work of ventilation increased even though air density falls about 20 per cent at 20,000 feet. In subjects working to exhaustion at 19,000 feet, arterial oxygen saturation fell to 44 to 50 per cent and was associated with impaired oxygen diffusion at the low alveolar oxygen tension and with a large (20 to 30 mm. of mercury) alveolar-

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. Bronchospirometry is desirable 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. Cardiov. 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.*)