EEG A patient with a convulsive disorder showed slow activity in the electroencephalogram. There was also a high blood carbon dioxide tension and somnolence. Sodium bicarbonate produced improvement. These findings suggest that the brain lesion causing the convulsions also caused the respiratory acidosis. It is postulated that electroencephalographic changes (slow activity) produced by voluntary hyperventilation are the result of hypocarbia directly and not anoxia secondary to cerebral vasospasm and that slow activity may result with either hyper- or hypo-carbia. (*Tarlau, M.: EEG Changes in Neurogenic Chronic Respiratory Acidosis, Electroencephalography and Clinical Neurophysiology, 10: 724 (Nov.) 1958.*)

CARBON DIOXIDE RESPONSE On study of the respiratory response to carbon dioxide in 65 subjects exposed to various concentrations it was found that the individual differences in response to carbon dioxide could be related to the basic respiratory pattern of the individual. Those subjects with a low ventilatory response to carbon dioxide had a larger tidal volume, inspiratory reserve and vital capacity; reduced adrenal sympathetic response to carbon dioxide; and fewer symptoms incident to carbon dioxide inhalation, and thus appears to be better physiological risks for training as underwater swimmers or aviators. (*Schaefer, K. E.: Respiratory Pattern and Respiratory Response to Carbon Dioxide, J. Appl. Physiol. 13: 1 (July) 1958.*)

SPIROMETRY Forced vital capacity and one second forced expiratory volume were studied in 534 patients with obstructive airway disease, heart disease, or pulmonary fibrosis. Both the one second F.E.V. and F.V.C. fell with loss of effort tolerance in each disease. In obstructive airway disease, the one second F.E.V. fell more than the F.V.C. In heart disease, the two fell more or less proportionately. With loss of effort tolerance, the forced expiratory ratio $\left(\frac{\text{F.E.V.}}{\text{F.V.C.}}\right)$ therefore fell little in patients with heart disease, but fell notably in patients with obstructive airway disease. The F.E.R. may best distinguish the two conditions and point to the disease which is the major cause of disability when both are present in the same patient. Heart disease and pulmonary fibrosis could not be distinguished from each other by the method. (*Capel, L. H., and Smart, J.: Spirometry and Effort Tolerance in Diseases of the Heart and Lungs, Lancet 2: 771 (Oct. 11) 1958.*)

BRONCHODILATORS Bronchospasm is one of the most intractible features of chronic bronchitis. The effectiveness of bronchodilators was therefore tried in a group of 39 patients. Isoprenaline, 20 mg., was administered sublingually, but this produced palpitation, dizziness and tremor in many patients. No similar complaints resulted from the inhalation of isoprenaline powder. Practically all patients showed an improvement in forced expiratory volume after inhalation of the powder. In 19 additional patients, the bronchodilator, mepyramine, was injected intramuscularly in 25 mg. doses. Forty-seven per cent of the patients demonstrated an increase in one second forced expiratory volume. (*Robinson, W., Woolley, P. B., and Altounyan, R. E. C.: Bronchodilators in Chronic Bronchitis, Lancet 2: 821 (Oct. 18) 1958.*)

CARDIAC ARREST Hypercapnia, usually associated with anoxia, and the ensuing respiratory acidosis, undesirable parasympathetic stimuli, patients' sensitivity to drugs or the presence of certain circulatory disturbances, particularly coronary insufficiency, lead to asystole. Myocardial ischemia and the overdosage or inadequate selection of the anesthetics employed give rise to ventricular fibrillation. Predisposing general factors: fever, electrolytic unbalance, anemia and malnutrition. Exhaustive pre-operative care of patients and careful operative preventive measures lower the incidence of this unfortunate complication. (*Perez Alvarez, J. J.: Prevention and Treatment of Cardiac Arrest in the Operating Room, Revista de la Confederacion Medica Pan-Americana 3: 15 (Jan.) 1956.*)

TACHYCARDIA Coronary hemodynamics, myocardial metabolism and cardiac efficiency were studied in the intact dog during tachycardia induced by an electrical stimulator. This induced tachycardia did not profoundly

alter general hemodynamics but it greatly increased coronary blood flow, cardiac oxygen consumption and carbon dioxide production. In the intact anesthetized dog it appeared that the heart rate had a close relationship to coronary flow volume when the heart is electrically accelerated. In the circumstances described, a profound fall in heart efficiency occurs. It seemed that, within limits, it is more efficient to deliver the same amount of cardiac work at a slow heart rate than at a fast one. In the latter circumstance, energy is inadequately converted to useful work and must appear in some other form, e.g., heat. (*Maxwell, G. M., and others: Induced Tachycardia: Its Effect Upon the Coronary Hemodynamics, Myocardial Metabolism and Cardiac Efficiency of the Intact Dog, J. Clin. Invest. 37: 1413 (Oct.) 1958.*)

CORONARY CONSTRICTION Intra-coronary administration of epinephrine or norepinephrine into the beating, fibrillating or potassium-arrested dog heart, indicate that the primary action of these drugs is vasoconstriction. The secondary action of vasodilation is due in large part to the resultant hypoxia of the heart muscle. (*Berne, R. M.: Effect of Epinephrine and Norepinephrine on Coronary Circulation; Circulation Res. 6: 644 (Sept.) 1958.*)

CORONARY HEART DISEASE Twenty-four patients with coronary heart disease and without valvular disease or hypertension were studied by cardiac catheterization. Patients with previous infarction, enlarged heart and dyspnea all displayed evidence of left ventricular failure, increased pulmonary capillary and artery pressure, depressed mixed venous blood saturation, and elevated right atrial pressure. By contrast, patients with previous infarction, normal heart size, and angina showed normal hemodynamics at rest and when free of pain. During exercise in this latter group varying degrees of left ventricular failure occurred in 9 of 11 patients, and in all 6 patients in whom anginal pain was precipitated. Following administration of nitroglycerine in 4 patients exercise was tolerated without evidence of left ventricular failure. (*Muller, O. and Rorvik, K.: Haemodynamic Consequences of Coronary Artery Disease, Brit. Heart J. 20: 302 (July) 1958.*)

CARDIAC FAILURE Patients in various degrees of left ventricular failure as a rule showed respiratory alkalosis and low oxygen tension. Patients with severe pulmonary edema had variable findings, including severe acute respiratory acidosis. Patients with heart disease and pulmonary emphysema tended to have respiratory acidosis. (*Carroll, D.: Oxygen Tension and Acid-Base Adjustment in Cardiac Failure, Bull. Johns Hopkins Hospital 103: 242 (Nov.) 1958.*)

VENTRICULAR FIBRILLATION During closure of an atrial septal defect under hypothermia, a ten year old white female developed ventricular fibrillation lasting 60 minutes. Despite eight electrical shocks and adrenalin injected into the pulmonary artery fibrillation continued. The rhythm spontaneously reverted to normal sinus rhythm and then to atrial fibrillation when the body temperature rose to 32.5 C. The aortic arch was occluded distal to the left subclavian artery for 78 minutes, and the patient developed muscle weakness of the legs presumably due to spinal cord anoxia. With careful massage and rewarming, ventricular fibrillation of long duration can be overcome. (*Martinez, J. B., and others: Factors Involved in the Recovery of a Patient After Prolonged Ventricular Fibrillation During Hypothermia, J. Thoracic Surg. 36: 749 (Nov.) 1958.*)

RIGHT HEART PRESSURE In cats under chloralose anesthesia there was frequently a fall in systemic blood pressure and heart rate when the inflow of blood to the right atrium was increased so that venous pressure rose. Increase in the output resistance of the right heart resulted in no changes in the circulation until the right heart output began to decline and venous pressure rose. These changes were probably dependent on the integrity of the vagus nerves. (*Barer, G. R., and Kottegoda, S. R.: Changes in Heart Rate and Blood Pressure of the Cat in Response to Increased Pressure on the Right Side of the Heart, J. Physiol. 143: 1 (Aug. 29) 1958.*)