**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