The larger the temperature gradient between left and right ventricular myocardia, the higher the temperature at which fibrillation occurred. Fibrillation was readily produced by induction of temperature gradients during nonuniform rewarming in hearts which had been uniformly cooled to substantially lower temperatures without fibrillation. At increasingly lower temperatures, fibrillation occurred with progressively smaller gradients; and below 20° C., fibrillation sometimes occurred with no measurable gradient. (*Mouritzen, C. V., and Andersen, M. N.: Myocardial Temperature Gradients and Ventricular Fibrillation During Hypothermia, J. Thor. Cardiov. Surg. 49: 937 (June) 1965.*)

ISCHEMIC HEART DISEASE Response of the circulation to a change in posture (tilt table) was studied in normal subjects and in patients with ischemic heart disease. These measurements were combined with the determination of cardiac output, stroke output, heart rate, and blood pressure. The normal response was an increase in cardiac output mainly due to an increase in stroke output, with minimal change in heart rate or blood pressure. After myocardial infarction most patients were unable to respond in a normal fashion and in some patients the cardiac output and stroke output dropped. When this occurred the compensatory mechanism was an increase in heart rate. This test would appear to have a place in clinical practice and evaluation of the extent of existing damage in patients who have sufferred myocardial infarcation. (*Thomas, M., and Shillingford, J.: Circulatory Response to a Standard Postural Change in Ischaemic Heart Disease, Brit. Heart J. 27: 17 (Jan.) 1965.*)

POSTURAL CHANGE When the postural change was made under general anesthesia a severe hypotension often occurred in cardiac patients with right-sided heart failure. This was most marked when halothane was used and least marked with cyclopropane. A correlation was present between intracardiac pressures in the right side of the heart, rise of systemic venous pressure and degree of blood pressure fall. Measurement of basal positional pressure prior to administration of anesthesia is of little significance in assessing the cardiovascular responses to postural change under general anesthesia in cardiac patients. (*Imazato, I.: Cardiovascular Response to Postural Change Under General Anesthesia (Japanese) J. Kurume Med. Assoc. 27: 195, 1964.*)

CIRCULATORY STIMULANT H 835, a mixture of two amino-alcyl-theophylline derivatives, is a new circulatory stimulant. Given intravenously to human beings, it caused long lasting increase in blood pressure and decrease in heart rate. Cardiac output increased without change in peripheral resistance. Indication for clinical use is hypotension not due to hypovolemia. (*Fischer, F., and Weis, K. H.: Experimental and Clinical Evaluation of the Circulatory Effects of Two Theophylline Derivatives (German), Der Anaesthesist 14: 147 (May) 1965.*)

MYOCARDIAL INFARCTION Hemodynamic changes following the administration of oxygen to patients with acute myocardial infarction were studied. Measurements included the determination of cardiac output and brachial artery pressure. Oxygen administration resulted in a rise in arterial blood pressure and a fall in cardiac output. The hemodynamic response to oxygen may be a result of vasoconstriction associated with an increase in blood pressure followed by a fall in cardiac output due to the rise in peripheral resistance. Therapeutic implications of this work could not be identified. (*Thomas, M., Malmcrona, R., and Shillingford, J.: Hemodynamic Effects of Oxygen in Patients with Acute Myocardial Infarction, Brit. Heart J. 27: 401 (May) 1965.*)

ANAPHYLACTIC SHOCK Anaphylactic shock produces myocardial damage extending over a period of several days. Myocardial cell injury was identified by the appearance of slowly evolving ST changes resulting from a current of injury and marked lengthening of the corrected Q-T duration. Selective heart chamber enlargement also was documented and presumed to be related to the profound bronchospasm associated with hypoxia and increased right ventricular work. (*Carmichael, D. B.: Human Electrocardiogram in Anaphylactic Shock, Dis. Chest 47: 564 (May) 1965.*)