FIBRINOLYSIS More bleeding occurs after extracorporeal circulation than after other thoracic surgical procedures. No specific defect in the coagulation mechanism has been identified, and fibrinolysis may be responsible. Activation of fibrinolysis occurs during stress, with obstetrical complications, with extensive neoplastic disease, cirrhosis, and following surgery. One half of the patients in this study developed fibrinolysis during the latter part of the perfusion. This was not related to acid-base balance, platelet count, fibrinogen level, or duration of perfusion. The only potential factors identified were the duration of storage of donor blood, fresh blood being better, and the type of blood container, siliconized bottles being better than plastic bags. (*Andersen, M. N., and Mendelow, M.: Fibrinolysis During and After Extracorporeal Circulation, Arch. Surg. 86: 649 (Apr.) 1963.*)

CAROTID BODIES An investigation was made of the effect of stimulation of the isolated perfused carotid body chemoreceptors on heart rate, blood pressure and cardiac output. Measurements were carried out by the dye-dilution method in anesthetized dogs breathing room air. Chemoreceptors were stimulated by changing the perfusate from oxygenated to hypoxic blood. In dogs with natural respiration, stimulation of the carotid bodies caused an increase in cardiac output and a reduction in total peripheral vascular resistance. When changes in pulmonary ventilation during stimulation of the carotid bodies were prevented by applying artificial respiration, bradycardia, a diminution in cardiac output and an increase in peripheral vascular resistance invariably occurred. These results show that the cardiovascular effects occurring in the spontaneously breathing dog are due, not to direct or primary reflex effects from chemoreceptors, but largely to secondary mechanisms arising from the concomitant reflex hyperventilation. Two mechanisms giving rise to secondary effects on the cardiovascular system and evoked by reflex stimulation of respiration are an inflationary reflex from the lungs and a reduction in arterial blood P_{CO_2}. (*Daly, M. B., and Scott, M. J.: Cardiovascular Responses to Stimulation of the Carotid Body Chemoreceptors in the Dog, J. Physiol. 165: 179 (Jan.) 1963.*)

CARDIAC CONTROL A study was carried out in intact dogs, except for various types of cardiac denervation, to clarify the nature of cardiac adaptation to exercise. The normal response to mild exercise is characterized by a slight increase in end-diastolic volume, increase in stroke volume, increase in stroke work and increase in pulse rate. Decrease in end-diastolic volume is usually but not invariably seen in exercise at this level. The decrease in end-diastolic volume during exercise suggests that the ventricular reservoir is a significant source of blood at the onset of exercise. In dogs with cardiac sympathectomy, vagectomy, or total cardiac denervation, the left ventricle remains able to respond to exercise by an increase in cardiac output and integrated minute work. Intrinsic myocardial factors, whereby these animals respond to exercise, are not solely a property of the isolated heart-lung preparation. Response of the left ventricle to exercise under these circumstances appears to be initiated by increased ventricular filling. (*Bruce, T. A., and others: Role of Autonomic and Myocardial Factors in Cardiac Control, J. Clin. Invest. 42: 721 (May) 1963.*)

VENTRICULAR VOLUME Effects of sudden changes in heart rate on left ventricular volumes were studied in dogs. Fast rates were produced by electrical pacing and slow rates, by efferent stimulation of the right vagus nerve, with and without slow pacing. The left ventricular stroke and the end-systolic and end-diastolic volumes were measured by an indicator dilution method in which temperature was the indicator. Cooled blood was injected into the ventricle, and aortic thermodilution curves were recorded by a thermistor catheter. Left ventricular volumes decreased during moderate tachycardia. At the fastest rates produced, the fall in stroke volume was proportionately greater than that in end-systolic and end-diastolic volumes. At slow rates stroke volume increased proportionately more than the other two volumes. The left ventricle in these experiments did not function with a constant end-systolic or residual volume. However, the directionally similar changes in end-systolic volume were of lesser proportion than the stroke volume alterations. (*Bristow, J. D., and others: Influence of Heart Rate on*