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*

Left Ventricular Volume in Dogs, J. Clin. Invest. 42: 649 (May) 1963.)

VENOMOTOR CONTROL In anesthetized open-chest dogs on cardiopulmonary bypass with a fixed cardiac output, venomotor function was observed by measuring venous return to the oxygenator, a large return indicating reduction in the systemic venous bed (venoconstriction) and a reduced return indicating an enlarged bed (venodilatation). Neurohumeral stimuli affected the capacity of the systemic venous bed profoundly. Pressure changes in the carotid sinus and the cardiac chambers, variations in arterial oxygen and carbon dioxide, and vasoactive drugs (norepinephrine, epinephrine, trimethaphan), as well as antihypertensive drugs (reserpine, guanethidine), all significantly altered venomotor tone under these experimental conditions. Further, in the intact human, additional studies with oral guanethidine and reserpine indicated these agents block reflex venoconstriction. Such alterations of venous return undoubtedly play an important role in regulating cardiac output and arterial pressure. *(Braunwald, E., and others: Reflex Control of the Systemic Venous Bed. Effects on Venous Tone of Vasoactive Drugs, and of Baroreceptor and Chemoreceptor Stimulation, Circulat. Res. 12: 539 (May) 1963.)*

RESPIRATION AND AORTIC PRESSURE In pentobarbital anesthetized dogs, inspiration resulted in a drop in aortic blood pressure. This drop was found to be dependent upon both a reduction in the left ventricular stroke output and the transmitted fall of intrathoracic pressure. During cardiac tamponade and airway obstruction the fall was accentuated, in the first instance by a greater fall in left ventricular output, and in the second by a greater fall of intrathoracic pressure. *(Shabetai, J., Fowler, N. O., and Gueron, M.: Effects of Respiration on Aortic Pressure and Flow, Amer. Heart J. 65: 525 (Apr.) 1963.)*

OXYGEN REFLEXES Whereas acetylcholine increases pulmonary blood volume by actively dilating regions within the pulmonary circulation, inhalation of 100 per cent oxygen decreases pulmonary blood volume. Since, at the same time as the volume decreases, the pulmonary artery and left atrial mean pressures and, hence, pulmonary vascular distending pressure tend to fall, a passive mechanism of action for 100 per cent oxygen must be invoked. Possibly oxygen reflexes produce systemic venodilation with a consequent redistribution of blood from the pulmonary to the systemic compartment. Inhalation of 100 per cent oxygen may improve the status of patients with pulmonary edema, not only by increasing arterial oxygen content, but also by decreasing the amount of blood in the lungs, thereby relieving pulmonary congestion. *(Glick, G., and others: Effects of Inhalation of 100 Per cent Oxygen on Pulmonary Blood Volume in Patients with Organic Heart Disease, Circulation 27: 554 (Apr.) 1963.)*

HYPOXIA Samples of blood were drawn from brachial arteries and the renal veins of patients being investigated for disorders of kidney function. Following control determinations, 8.5 per cent oxygen in nitrogen was administered for 25 minutes. The oxygen saturation of arterial blood fell to between 74 per cent and 87 per cent, that of renal vein blood to between 72 per cent and 80 per cent. Urine flow decreased to one-half or less during hypoxia. The PAH-clearance and inulin clearance also were reduced during hypoxia. *(Duner, H., and Granberg, P.: Effect of Induced Hypoxia on Renal Function in Man, Acta Chir. Scand. 125: 253 (Mar.) 1963.)*

CORONARY PERFUSION Cardiac function was studied after regional perfusion of the coronary arteries with oxygen unsaturated blood or isotonic colloidal solutions (dextran) at a pressure level equal to that in systemic arteries. Perfusion of 4 ml/minute during 30 minutes into the anterior descending coronary artery did not alter the rate of heartbeat, the aortic or left atrial pressures. Rapid perfusions of 20 ml/minute during or after the above perfusions, also did not alter heartbeat or pressures. The retropressure and backflow increased during these perfusions. Gradual exsanguination of the animals during dextran perfusion permitted total exsanguination with-