Induced Hypotension, Der Anaesthesist 10: 72 (Mar.) 1961.)

HEMODYNAMICS Effects of alterations of intracardiac pressure on total venous return, systemic vascular volume and peripheral vascular resistance were measured during perfusion of the systemic vascular bed using an extracorporeal circulation while pressures were independently varied in an innervated, but hemodynamically isolated heart. Elevation of intracardiac pressure in all four chambers or only in the left side of the heart resulted in an augmentation of systemic vascular volume. Venous return declined by an average of 25 per cent from the control levels. Significant decreases in systemic vascular resistance were observed when left ventricular systolic pressures were elevated, when left ventricular diastolic and mean left atrial pressures were also elevated or when extrasystoles occurred. Only minimal alterations in vascular resistance were observed with elevation of right heart pressures alone. *(Ross, J., Frahm, C. J., and others: Influence of Intracardiac Baroreceptors on Venous Return, Systemic Vascular Volume and Peripheral Resistance, J. Clin. Invest. 40: 563 (Mar.) 1961.)*

SPLANCHNIC CIRCULATION Estimated hepatic blood flow, splanchnic blood volume, calculated splanchnic vascular resistance, and mean arterial pressure did not change significantly or consistently during light general anesthesia with thiopental, nitrous oxide and succinyl choline. Hypercapnia under these circumstances results in an increase in mean calculated splanchnic vascular resistance. Estimated hepatic blood flow decreases, remains unchanged or rises depending upon the arterial pressure. The average value for circulating splanchnic blood volume decreases significantly. These changes are ascribed to a combination of arteriolar and venous constriction. Sulfobromophthalein clearance and extraction by the liver decrease significantly during hypercapnia although unaffected by anesthesia. *(Epstein, R. M., and others: Effect of Hypercapnia on Estimated Hepatic Blood Flow, Circulating Splanchnic Blood Volume, and Hepatic Sulfobromophthalein Clearance During General Anesthesia in Man, J. Clin. Invest. 40: 592 (Mar.) 1961.)*

HEPATIC BLOOD FLOW Hepatic plasma flow during thiopental, cyclopropane, ether, local, and spinal anesthesia was measured in 68 patients using the tagged chromic phosphate method. Premedication with pentobarbital and atropine produced only slight changes. During thiopental-nitrous oxide anesthesia, in 11 patients, mean hepatic plasma flow fell from 829 to 531 ml. per minute after induction of anesthesia. When thiopental was supplemented with succinylcholine for muscular relaxation, the accompanying depression of liver plasma flow was considerably less. With cyclopropane anesthesia, in 13 patients, mean hepatic plasma flow fell from 710 ml. to 507 ml. per minute. During thiopental and during cyclopropane anesthesia, changes in hepatic flow were consistent and statistically significant. During ether anesthesia the changes were variable and of smaller magnitude. Mean hepatic plasma flow was 903 ml. before induction, and 810 ml. during induction. There were no demonstrable correlations between changes in blood pressure, pulse, or cardiac output and hepatic plasma flow nor between the anesthetic level, *p*H, carbon dioxide tension, and hepatic plasma flow. *(Levy, M. L., and others: Hepatic Blood Flow Variations during Surgical Anesthesia in Man Measured by Radioactive Colloid, Surg. Gynec. & Obstet. 111: 289 (Mar.) 1961.)*

BLOOD-COAGULATION By means of a battery of simple tests the changes in various clotting elements were studied in 30 experimental and 8 clinical perfusions of under 1½ hours duration and in 1 patient perfused for 2¼ hours. The complete neutralization of heparin was achieved in all cases by use of Polybrene in a dose of 1 to 1.5 mg. for each milligram of the heparinizing dose which was 2 mg. per kilogram. In perfusion under 1½ hours the average platelet count after bypass was about 70 per cent of the pre-perfusion level and the fibrinogen concentration about 85 per cent. Prothrombin time was little changed. The changes bore no direct relationship to the duration of the perfusion but were influenced by the effect of massive

transfusion of donor-blood during perfusion. Freshly collected donor-blood is preferred for perfusion. The patient perfused for 2½ hours developed abnormal bleeding due to fibrinogen destruction, but was successfully treated by the infusion of human fibrinogen. Plasma hemoglobin after perfusions of less than 1 hour was 50 mg. per cent or less. (*Rothnie, N. G., and others: Changes in Blood-Coagulation Due to Perfusion for Cardiac Surgery, Brit. J. Surg. 48: 272 (Nov.) 1960.*)

SHOCK Several factors which may contribute to circulatory collapse following cardiovascular surgery are blood loss, airway obstruction, partially relieved valvular obstruction, hemopericardium, ball valve thrombosis, dehydration, cardiac arrhythmias, reactivation of rheumatic carditis, myocardial infarction, and infection. Adequate management is dependent entirely on the correct diagnosis of the cause. (*Redo, S. F., and Arditi, L. I.: Causes and Treatment of Arterial Hypotension, Circulatory Collapse and Shock Following Cardiovascular Operations, Surg. Clin. N. Amer. 41: 309 (Apr.) 1961.*)

HEMORRHAGIC SHOCK Hepatic metabolism and hemodynamics have been studied in a series of unanesthetized dogs whose major hepatic vessels had been previously catheterized. Two types of hemorrhage were studied. Blood was suddenly or acutely withdrawn in one group of dogs in amounts sufficient to lower blood pressure to 60 to 40 mm. of mercury. These findings were compared with those from another group of dogs following slow, protracted hemorrhage. After hemorrhage there was a progressively increasing hepatic glucose output, hepatic potassium output, and hepatic sodium uptake, and an increased hepatic venous resistance. The latter was intensified after retransfusion of the withdrawn blood. In previous studies, the authors had found that comparable conditions were produced by the infusion of epinephrine to unanesthetized dogs and also that the adrenal gland output of catechol amines was increased by hemorrhage. This suggests that many of the hepatic, metabolic, and hemodynamic effects that follow hemorrhage are mediated by epinephrine. (*Shoemaker, W. C., Walker, W. F., and Turk, L. N.: Role of Liver in Development of Hemorrhagic Shock, Surg. Gynec. & Obstet. 111: 327 (Mar.) 1961.*)

TRANSFUSION THERAPY If a transfusion reaction is suspected 20 ml. of venous blood should be drawn, and a specimen of urine collected. The blood should be utilized in the following tests: (1) complete blood grouping and cross-matching using pretransfusion and post-transfusion samples from the recipient and the donor blood from the container; (2) direct Coomb's test on the red cells of the recipient; (3) testing of the recipient serum against the panel of known group O cells to determine the presence and specificity of any antibody; (4) estimation of free hemoglobin and serum bilirubin in the post-transfusion sample from the recipient and the recipient's urine; (5) Gram stain and culture from the original container if available. (*Grove-Rasmussen, M., Lesses, M. F., and Anstall, H. B.: Transfusion Therapy (concluded), New Engl. J. Med. 264: 188 (May 25) 1961.*)

HYPOTHERMIA Ventricular fibrillation occurring during the combined use of extracorporeal circulation and hypothermia in dogs is easily reversible when controlled by a pump oxygenator. Its occurrence does not necessarily indicate anoxemia or myocardial damage. Recovery of normal ventricular function is not altered by cold combined with 10 to 60 minutes of myocardial ischemia at temperatures of 7 C. Studies reveal that effective heart action ceases at 25 C. Others reveal that potassium-magnesium-prostigmine cardioplegia appears to be a safe technique. (*Sealy, W. C., and others: Observations on Heart Action during Hypothermia Induced and Controlled by Pump Oxygenator, Ann. Surg. 153: 597 (May) 1961.*)

HYPOTHERMIA Observations on the distribution of infused potassium in dogs in moderate hypothermia (mean temperature 27.9 C.) indicate that there is probably no remarkable alteration in the exchange of potassium in the hypothermic state. Probably there is a net loss of potassium from the cells as hypothermia progresses. Although the se-