CARDIOGENIC SHOCK Intraaortic balloon pumping was used in the treatment of 16 patients who were in shock due to myocardial infarction. All were refractory to ordinary therapy and were considered to be in terminal condition. Pumping duration varied from 1.5 hours to 55 hours. Diastolic pressures rose 5 to 42 per cent. Three patients had cardiac output determinations; these averaged 2 l/min before, and 4 l/min during, pumping. Pulmonary edema decreased, while peripheral blood flow and urinary output improved. Shock was reversed in all patients. Three patients died immediately, during interruptions in the pumping. Six patients died hours or days after pumping was discontinued. Seven lived and were discharged from the hospital. The results in this small group are encouraging, and indicate that the method merits further study. *(Kantrowitz, A., and others: Mechanical Intraaortic Cardiac Assistance in Cardiogenic Shock, Arch. Surg. 97: 1000 (Dec.) 1968.)*

SHOCK Both dopamine and isoproterenol exert prominent positive inotropic effects on the heart. The amines differ in their actions on peripheral blood vessels: isoproterenol produces beta-adrenergic vasodilatation; dopamine lacks this action but does produce selective renal dilatation. The hemodynamic response to intravenous infusion of these amines was examined in dogs subjected to hemorrhagic shock. In the doses selected, both agents increased thoracic and abdominal aortic blood flow. Splanchnic and renal blood flows were increased by both amines, but significantly so by dopamine only. The cardiac output was increased by isoproterenol only. *(Gifford, R. M., and others: Changes in Regional Blood Flows Induced by Dopamine and by Isoproterenol during Experimental Hemorrhagic Shock, Canad. J. Physiol. Pharmacol. 46: 847 (Nov.) 1968.)*

INTRAVASCULAR COAGULATION Detailed analyses of blood coagulation were made in 36 pediatric patients with septicemia. Various changes in the clotting mechanism were found, irrespective of infectious agent but apparently related to blood pressure. The most frequent single abnormality was thrombocytopenia, found in 61 per cent of all cases. Multiple coagulation defects, regularly noted in patients with hypotension or shock, were interpreted as being secondary to diffuse intravascular coagulation. Similar changes were not seen in patients with normal blood pressures. The most reliable laboratory guides seemed to be reduced platelet count, low factor-V levels in plasma, and fibrinolytic-split products in serum. Heparin (50 to 100 units /kg body weight) was given every four hours to all patients with hypotension suspected of having the defect. Most patients with septicemia and low blood pressure apparently have coagulation defects. *(Corrigan, J. J., and others: Changes in the Blood Coagulation System Associated with Septicemia, New Eng. J. Med. 279: 851 (Oct.) 1968.)*

ABTRACTER'S COMMENT: Disseminated intravascular coagulation is now recognized as being responsible for most of the acutely acquired hypofibrinogenemic disorders. It has been hypothesized, and supported by experimental work, that endotoxin activates Factor XII, thereby initiating the clotting mechanism. In the process, bradykinin, the most potent vasodepressor known, is released. If the activation of Factor XII could be inhibited by drugs, such as protamine, as suggested by Nossel, perhaps the consumption coagulopathy and the shock seen in septicemia could be prevented.

BLOOD TRANSFUSION In massive blood transfusions, consideration must be given not only to total quantity of blood being administered, but also to the rate of administration. Possible dangers from excessively rapid administration include citrate intoxication, hypocalcemia, hyperkalemia, acidosis, and hypothermia. Hypothermia is, without doubt, the most important problem in massive blood transfusion, and may persist long into the postoperative period. However, any of the above changes, alone or in association with movement of the patient, alteration of posture, reduced ventilation or administration of sedative drugs, may precipitate cardiac arrest long after the period of hemorrhage has ceased. *(Churchill-Davidson, H. C.: Some Problems of Massive Blood Transfusion, Proc. Roy. Soc. Med. 61: 681 (July) 1968.)*