tem is used to minimize ischemic loss of heart muscle during a heart attack. A tube is inserted into one of the patient's leg arteries and passed into the abdominal aorta. The pump, synchronized to the heartbeat with an electrocardiograph, withdraws blood from the aorta during cardiac systole, thus lowering systolic pressure and diminishing the work load of the heart. The blood is then rapidly returned to the aorta during diastole, thus raising the diastolic coronary perfusion pressure as well as maintaining systemic pressure. The pump's action forces open some of the vascular 'detours' in the heart, nourishing the area that is being deprived of blood by the blockage of a main vessel. If started soon enough—within three hours after the patient is stricken—the pump, which has thus far been used satisfactorily on five human patients as well as on animals, can restore a myocardial blood supply before damage becomes irreversible. (*Medical News: Counterpulsating Pump Described at Forty-seventh College of Surgeons Congress, J. A. M. A. 178: 26 (Oct. 28) 1961.*)

**MYOCARDIAL AUGMENTATION** A new method has been developed in dogs to provide mechanical assistance in the management of central circulatory insufficiency. Postsystolic myocardial pressure was augmented with the aid of an electronic circuit and the QRS complex of the electrocardiogram. Normal circulatory dynamics were approximated by this method in dogs with experimentally induced dilated hearts and circulatory hypotension. (*Watkins, D. H., and Duchesne, E. R.: Postsystolic Myocardial Augmentation: I. Effect Upon an Induced Shock State, Proc. Soc. Exp. Biol. Med. 107: 659 (July) 1961.*)

**SHOCK** Intravascular coagulation results from the intra-aortic injection of E. coli endotoxin in dogs. This is indicated by (1) a decrease of blood clotting factors, (2) presence of thrombi in tissue sections, (3) finding of focal necrosis and infarcts, and (4) prevention of these findings by preheparinization. The immediate (reversible) fall in blood pressure after endotoxin injection is caused by a decrease in cardiac output. This is due to a decreased venous return to the left heart caused by a cor pulmonale and associated vasospasm following blockage of pulmonary capillaries by thrombi. It is also due to a decreased venous return to the right heart by the damaging effect of thrombi in the portal system. The secondary (irreversible) fall in blood pressure and death appears to be greatly assisted by loss of blood and serum into the bowel lumen, secondary to the necrosis and sloughing of the superficial gastrointestinal mucosa. (*Hardaway, R. M., and others: Endotoxin Shock. A Manifestation of Intravascular Coagulation, Ann. Surg. 154: 791 (Nov.) 1961.*)

**SHOCK** Serum level serotonin in dogs falls to 22 per cent of normal values during lethal endotoxin shock. Plasma catecholamine concentrations rise after endotoxin administration. Epinephrine returns to normal levels but norepinephrine appears slightly elevated. The release of histamine, serotonin, epinephrine, and norepinephrine cause hemodynamic changes which lead to endotoxin shock. It is believed that a temporary depletion of peripheral catecholamine store occurs resulting in vascular instability, vasodilatation, pooling, decreased cardiac output, and shock. After hemorrhagic hypothermia, peripheral venous levels of endogenous epinephrine increases 90 fold over control values. (*Rosenberg, J. C., and others: Studies on Hemorrhagic and Endotoxin Shock in Relation to Vasomotor Changes and Endogenous Circulating Epinephrine, Norepinephrine and Serotonin, Ann. Surg. 154: 611 (Oct.) 1961.*)

**SHOCK** Prevention of serious effects from hemorrhage is best accomplished by early and adequate blood transfusion. Experiments on dogs in severe shock from bleeding show that hydrocortisone and adrenergic blocking agents promote survival, but that noradrenaline does not. (*Hakstian, R. W., Hampson, L. G., and Gurd, F. N.: Pharmacological Agents in Experimental Hemorrhagic Shock, A. M. A. Arch. Surg. 83: 335 (Sept.) 1961.*)

**ADJUSTMENTS TO HEMORRHAGE** By analyzing the volume-flow-pressure relationship in sympathectomized dogs, the various compensatory adjustments to hemorrhage