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

can be judged. When the blood volume is reduced by less than 10 per cent, the arterial pressure is maintained primarily by the non-neural factors. With a volume reduction between 10 and 25 per cent, the sympathetic activity, the inhibition of vagi, and the non-neural factors are of about equal importance. When the reduction of blood volume is greater than 25 per cent, the sympathetic activity becomes the most significant compensatory factor. (*Chien, S., and Billig, S.: Effect of Hemorrhage on Cardiac Output of Sympathectomized Dogs, Amer. J. Physiol. 201: 475 (Sept.) 1961.*)

AURICULAR PRESSURE In five dogs and one human being, right auricular, intrathoracic (esophageal), intratracheal, and arterial pressures were measured during spontaneous respiration, manually controlled respiration, IPBB, and positive-negative phase respiration. Right auricular pressure is low during spontaneous inspiration, and elevated during expiration. The pressures in the right atrium show a direct relationship to those in the respiratory tract. With high insufflation pressures, the pressure in the right atrium is markedly increased and venous filling is diminished. The addition of a negative phase during expiration causes restitution to almost physiological conditions. The right auricular pressure curves resemble those obtained during spontaneous respiration. (*Hanquet, M., and Lefèbvre, L.: Respiration Contrôlée et Pression Auriculaire Droite, Acta Anaesth. Belg. 11: 381 (Dec.) 1960.*)

VENTRICULAR TACHYCARDIA Quinidine and procaine amide are the most effective drugs for treatment of ventricular tachycardia, and the majority of episodes respond to these agents. However, in large doses, these drugs significantly depress myocardial contractility with resultant heart failure and shock. The object of electric countershock, whether the underlying arrhythmia is ventricular tachycardia or fibrillation, is to achieve simultaneous, uniform depolarization of all parts of the ventricle, thereby promptly extinguishing ectopic foci and permitting the sinus node to resume as pacemaker. When countershock is applied directly to the exposed heart, relatively small amounts of current (50 to 150 volts) are employed; greater current, usually around 350 to 440 volts, is necessary when countershock is applied externally. The obvious advantages of electric countershock are that it is easily administered, the results are immediate, there is no lasting depression of cardiac function, and there are no serious after-effects. Combined with the safety factors of effective external pacemakers and external cardiac massage, this new technique may be the treatment of choice for ventricular tachycardia when anti-arrhythmic drugs have proven ineffective. (*Alexander, S., and others: Use of External Electric Countershock in the Treatment of Ventricular Tachycardia, J. A. M. A. 177: 916 (Sept. 30) 1961.*)

STARLING'S LAW Changes in effective left ventricular end-diastolic pressure were determined by measuring left ventricular pressure with a catheter introduced through the atrial septum, and intraesophageal pressure with a balloon. The activity of the autonomic nervous system was reduced with an infusion of trimethaphan administered at a constant rate. The reduction in circulatory reactivity was indicated by the absence of arterial pressure response to the cold pressor test. Measurements of cardiac output, stroke volume, left ventricular work, power, tension-time index, and the duration and mean rate of left ventricular ejection were carried out before, during and upon completion of a transfusion of 1,500 ml. of the subject's own blood. Transfusion resulted in a significant elevation of left ventricular end-diastolic pressure in each subject. As this occurred, left ventricular performance also became augmented. These data are consistent with the hypothesis that the end-diastolic pressure is an important determinant of the characteristics of ventricular contraction and that Starling's Law of the heart is applicable to man. (*Braunwald, E., and others: Studies on Starling's Law of the Heart. V. Left Ventricular Function in Man, J. Clin. Invest. 40: 1882 (Oct.) 1961.*)

BARORECEPTORS The relationship between pulmonary arterial pressure and the impulse activity recorded from pulmonary arterial baroreceptor fibers has been studied in