benefit. Denervation of the mesenteric sympathetics is effective only if performed prior to the onset of shock. (*Berger, R., and others: Surgical and Chemical Denervation of Abdominal Viscera in Irreversible Hemorrhagic Shock, Ann. Surg. 162: 181 (Aug.) 1965.*)

SHOCK Anesthetized dogs were subjected to either severe (35 mm. of mercury mean blood pressure for 5 hours) hemorrhagic or *E. coli* endotoxin shock. Both pretreatment with adenosine triphosphate (ATP) or its administration late in hemorrhagic shock resulted in a significant increase in survival rates in paired animals to whom either ATP or a placebo was given by random selection. In the pretreated group, survival was 83 per cent as compared with 23 per cent in controls; in the late-treated group, there was a 70 per cent survival rate. ATP pretreated animals shocked with endotoxin had a 40 per cent survival rate as compared with 20 per cent in nontreated. The apparent beneficial effect of ATP in shock may be attributed to vasodilatation, energy transfer, anticoagulation or a combination of these mechanisms. (*Massion, W. H.: Value of High Energy Compounds in the Treatment of Shock, Amer. J. Surg. 110: 342 (Sept.) 1965.*)

SHOCK Bedside hemodynamic evaluation is proposed as the criterion for clinical management of shock. Three basic causes of clinical shock are identified: hypovolemia, cardiac insufficiency, and deficient vascular tone. Two types of observations are utilized: central venous pressure and arterio-venous oxygen difference. Hypovolemia and cardiac insufficiency both cause reduced cardiac output, manifested by increase in the arterio-venous oxygen difference. Hypovolemia is accompanied by low or normal venous pressure, while cardiac insufficiency causes elevated venous pressure. Deficient vascular tone is associated with normal cardiac output, and normal arterio-venous oxygen difference, or hypotension out of proportion to any decrease in cardiac output. For each type of shock the appropriate therapy can be selected: blood volume expansion, cardiac support, or vasoconstrictor-drug administration. The limiting factor in survival is cellular metabolic failure. (*Wilson, J. N.: Rational Approach to Management of Clinical Shock, Arch. Surg. 91: 92 (July) 1965.*)

CARDIAC MECHANICS Myocardial force-velocity relations were investigated in 13 patients employing a cineradiographic technique. A constant relation was established between intraventricular pressure and myocardial wall tension. When afterload was augmented with methoxamine or was decreased by impeding venous return with a balloon distended in the inferior vena cava, force and velocity varied inversely. Norepinephrine, isoproterenol, increasing heart rate by electrical pacing, and paired electrical stimulation all augmented velocity at any given pressure. In conscious man the heart displays the same reciprocal relation between velocity of shortening and generation of force observed in isolated papillary muscle, and a change in the contractile state of the human heart is manifested by a shift in the force-velocity relation. (*Glick, G., Sonneblick, E. H., and Braunwald, E.: Myocardial Force-Velocity Relations Studied in Intact Unanesthetized Man, J. Clin. Invest. 44: 978 (June) 1965.*)

A-V REFRACTORY PERIOD The refractory period of the atrioventricular conduction system was measured in man employing a bipolar electrode catheter. The time interval during which A-V conduction failed was taken as the A-V refractory period. The average refractory period in 20 unanesthetized patients, studied in the basal state, was 350 msec. In 9 patients, atrial tachycardia induced by rapid electrical stimulation of the right atrium prolonged the refractory period. In 7 patients, muscular exercise shortened the refractory period by an average of 10 msec. Isoproterenol and atropine caused reductions of the refractory period similar to those observed during exercise. Elevation of arterial pressure with methoxamine or phenylephrine prolonged the refractory period by an average of 252 msec. Since this prolongation could be prevented by atropine, it was considered to result from reflex vagal stimulation. In anv given patient the A-V refractory period may be affected by many influences including sympathetic and parasympathetic stimuli. (*Linhart, J. W., Braunwald, E., and Ross, Jr., J.: Determinants of the Duration of the Refractory Period of the Atrioventricular Nodal System in Man, J. Clin. Invest. 44: 883 (June) 1965.*)