pacing to the pre-nitroglycerin angina-producing rate. Compared with pre-drug studies, aortic, pulmonary arterial, right atrial and pulmonary capillary wedge pressures all were reduced. Cardiac output and stroke volume were reduced, as was end-diastolic heart size and the tension–time index. Anginal pain was not produced in ten of 13 patients. Nitroglycerin allowed the heart to be driven at higher rates without production of pain. These effects reflect a decreasing oxygen requirement of the heart and are consistent with the pain-relieving properties of the drug. (*Frick, M. H., and others: Hemodynamic Effects of Nitroglycerin in Patients with Angina Pectoris Studied by an Atrial Pacing Method, Circulation 37: 160 (Feb.) 1968.*)

CARDIAC ARREST ACIDOSIS Acidosis secondary to cardiac arrest was studied in 22 patients. The ten patients with predominantly respiratory acidosis were those with pulmonary problems. The *p*H range in eight members of this group was 6.86 to 7.09; hypercapnia was prevalent, alkali therapy seemed less effective and improved ventilation appeared the major therapeutic objective. Most of the eight patients with predominantly metabolic acidosis had myocardial infarction. The *p*H range in this group, 7.15 to 7.35, appeared to be benefitted by sodium bicarbonate, with or without hyperventilation. (*Chazan, J. A., Stenson, R., and Kurland, G. S.: The Acidosis of Cardiac Arrest, New Engl. J. Med. 278: 360 (Feb.) 1968.*)

EXPERIMENTAL HEMORRHAGE Nine young male volunteers underwent removal of about 15 per cent of their blood volumes at either slow or rapid rates, by either venous or arterial routes. There was an insignificant decrease in cardiac index and a significant decrease in stroke volume, as reflected by arterial pressure and pressure pulse contours. Pulse rates increased significantly. The calculated total peripheral resistance decreased slightly in seven subjects and increased in two. Central venous pressure decreased during or following hemorrhage. Rapid bleeding caused briefer and sharper changes in measured parameters than slow bleeding. More profound and prolonged falls in blood pressure resulted from venous than from arterial hemorrhages. Valsalva maneuver performed after hemorrhage produced evidence of temporary impairment of right and left ventricular outflows in all subjects. Inspiratory breath-holding resulted in decreases in cardiac index, blood pressure, and total peripheral resistance. (*Skillman, J. J., and others: The Hemodynamic Effect of Acute Blood Loss in Normal Man, with Observations on the Effect of the Valsalva Maneuver and Breath-holding, Ann. Surg. 166: 713 (Nov.) 1967.*)

CARDIAC SHOCK Experimental cardiogenic shock produced by intracoronary artery microsphere embolization in the dog and clinical cardiogenic shock in man following myocardial infarction or open-heart surgery give similar pictures. The decreases in cardiac output and blood pressure trigger the release of epinephrine and norepinephrine via the baroreceptor reflex response. This increased alpha-adrenergic activity increases total peripheral resistance by causing vasoconstriction in the splanchnic (including renal), pulmonary, and cutaneous beds. The resultant decreased perfusion of these areas leads to metabolic acidosis, compensatory hyperventilation, reduced renal blood flow, and a further increase in vascular resistance. The damaged heart frequently is unable to tolerate the increased work, and death follows. Alpha-adrenergic blockade with phenoxybenzamine or chlorpromazine increases the survival rate from 25 to 60 per cent in dogs with experimental myocardial infarction. Patients with cardiogenic shock following open-heart surgery responded well to phenoxybenzamine or chlorpromazine in doses of one mg./kg., provided central venous pressure was maintained above 10 cm. H_2O with volume expanders. The improvement was manifested by decreased vasoconstriction, improved renal function, lessening of the acidosis, and ultimate recovery of the patient. (*Dietzman, R. H., and Lillihei, R. C.: The Treatment of Cardiogenic Shock, Part IV, Amer. Heart J. 75: 136 (Jan.) 1968.*)

CARDIOGENIC SHOCK Massive doses of glucocorticosteroids have proved helpful in the treatment of septic shock both experimentally