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

and clinically. Similar massive doses also may help in the treatment of cardiogenic shock. In both conditions steroids reduce peripheral resistance and vasoconstriction, thus improving tissue blood flow. Although definite alpha-blocking activity has not been proven, the response is similar to alpha-adrenergic blockade. In addition, steroids help maintain the integrity of cell membranes and help prevent the release of proteolytic enymes from lysosomes. With steroid treatment, survival rate in dogs with experimentally-produced myocardial infarction rose from 25 to 65 per cent. Patients with cardiogenic shock also responded well. Doses of 30 mg./kg. of methylprednisolone (Solu-Medrol), 6 mg./kg. of dexamethasone (Decadron), or 150 mg. of hydrocortisone (Solu-Cortef) were necessary to produce this effect. (*Dietzman, R. H., and Lillihei, R. C.: The Treatment of Cardiogenic Shock, Part V, Amer. Heart J. 75: 274 (Feb.) 1968.*) ABSTRACTOR'S COMMENT: The increase in the survival rate of dogs receiving alpha-adrenergic blockade or steroids is impressive. There is not sufficient data to evaluate the benefits in human cardiogenic shock. The clinical impression is that they might be of some value.

BLOOD VOLUME Plasma volume increased 21 per cent following one week's administration of guanethidine to normal men. A similar increase in plasma volume was seen after phenoxybenzamine. There was no associated sodium retention. Forearm venous sympathetic reflexes were attenuated. The sympathetic nervous system may provide a means whereby blood volume is regulated. (*Weil, J. V., and others: Plasma Volume Expansion Resulting from Interference with Adrenergic Function in Normal Man, Circulation 37: 54 (Jan.) 1968.*)

HEMODILUTION At the terminal stage of bypass the hemodilution technique imposes a time limitation on the prevention of severe metabolic acidosis during bypass. The time limit may be 60 minutes with high hemodilution, 120 minutes with moderate hemodilution, and 180 minutes with low hemodilution. With prolongation of total cardiopulmonary bypass time, care must be exercised in the treatment and stabilization of all acid–base parameters during and following bypass. The administration of additional Tris buffer at the midpoint of the perfusion and one hour after the operation; adequate counter-measures to prevent respiratory distress; and frequent observation of the acid–base equilibrium is recommended. (*Taguchi, K., and others: Clinical Experiences with Hemodilution in Total Cardiopulmonary Bypass, Surgery 63: 252 (Feb.) 1968.*)

BLOOD STORAGE The addition of adenine and inosine to stored acid–citrated blood can increase the useful storage period to eight weeks. The increased survival is correlated with the adenosinetriphosphate content of the blood. The useful life of bank blood could be extended by the addition of adenine and inosine as the customary expiration date of three weeks approaches. (*Strumia, M. M., and others: The Preservation of Blood for Transfusion. VI. Effect of Addition of Adenine and Inosine on ATP and Posttransfusion Survival of Red Cells of Stored Blood, J. Lab. Clin. Med. 71: 138 (Jan.) 1968.*)

ERYTHROCYTE PRESERVATION Criteria for determining clinical acceptability of preserved erythrocytes include viability, mode of removal of nonviable red cells, and quantity of supernatant hemoglobin in the blood. Other measurements include toxicity of additives, sterility, pH, and extracellular potassium concentration. Supplementation of ACD solution with purine nucleosides increases acceptable storage time. Frozen blood is essentially packed erythrocytes. If coagulation factors are needed, fresh frozen plasma should be used. Preservation of erythrocytes by freezing is more costly than the standard method that uses ACD liquid preservation. Freezing of erythrocytes may be indicated to store rare types of blood, for autotransfusions, to establish a supply of selected red cells lacking the antigens to which recipients are most commonly sensitized, and as a stockpile of selected erythrocytes in anticipation of a military or civilian disaster. (*Valeri, C. R.: Preservation of Human Red Blood Cells, Bull. N. Y. Acad. Med. 44: 1 (Jan.) 1968.*)