response to isoproterenol was unchanged. This differential response to the three sympathomimetic amines employed was attributed to the increased levels of circulating catecholamines found in acidosis. This conclusion was supported by the finding that the chronotropic response to tyramine in hemorrhagic hypotension, a condition known to produce increased levels of circulating catecholamines, was also potentiated in the low dosage range. (*Ford, G. D., Cline, W. H., Jr., and Fleming, W. W.: Influence of Lactic Acidosis on Cardiovascular Response to Sympathomimetic Amines, Amer. J. Physiol. 215: 1123 (Nov.) 1968.*)

HEMORRHAGIC SHOCK Hypotensive shock was produced in rats by removal of blood and maintenance of blood pressure at 30 or 40 mm Hg. While the degree and duration of hypotension were the most critical factors determining the severity of shock and probability of survival, other conditions, such as high environmental temperature, a fasted state, and lack of previous exercise, also lessened the capacity of the rat to withstand the stress of hypotension. (*Steinman, R., and Denstedt, O. F.: Experimental Production of Hemorrhagic Shock in the Rat, Canad. J. Pharmacol. 47: 305 (March) 1969.*)

KREBS-CYCLE METABOLITES Since specific Krebs-cycle intermediates stimulate anaerobic ATP synthesis, treatment with such metabolites might influence 24-hour survival of rabbits subjected to hemorrhagic shock. Anesthetized rabbits were bled to a mean blood pressure of 35 mm Hg and the blood retransfused after 90 minutes of hypotension. Metabolites were administered intravenously during the hypotensive period and for an additional two hours following the beginning of blood replacement. The metabolites were diluted with 0.75 M sodium chloride so that all infused solutions had the same toxicity as 1.5 M glucose. Animals receiving either fumarate or a combination of oxalacetate and α-ketoglutarate had a significantly greater survival rate than untreated or 0.75-M NaCl-infused animals. Treatment with either oxalacetate or α-ketoglutarate alone or with glucose failed to produce statistically significant increases in survival. No correlation between survival and *p*H or bicarbonate levels of arterial blood was observed. Administration of Krebs-cycle metabolites may alter the lethal course of the hemorrhagic shock syndrome by stimulating high-energy phosphate production under conditions of tissue hypoxia. (*Chick, W. L., and others: Influence of Krebs-cycle Intermediates on Survival in Hemorrhagic Shock, Amer. J. Physiol. 215: 1107 (Nov.) 1968.*)

SHOCK Experimental myocardial infarction and shock in dogs are associated with marked peripheral vasoconstriction, reduced tissue perfusion, lactic acidemia, and a mortality rate of 70 per cent. Survival and acute hemodynamics are significantly improved among dogs made tolerant to epinephrine (survival is 100 per cent) by reduction in the magnitude of the vasoconstrictor response in the face of an amount of myocardial damage equal to that which produces 70 per cent lethal shock in nontolerant dogs. (*Dietzman, R. H., and others: Prevention of Lethal Cardiogenic Shock in Epinephrine-tolerant Dogs, Surgery 65: 623 (April) 1969.*)

SODIUM REABSORPTION The hematocrits of anesthetized dogs were decreased with no changes in blood volume by exchange transfusion using a reservoir containing artificial plasma. Fractional reabsorption by the proximal tubule was decreased significantly, while sodium excretion was changed insignificantly after equilibration with the reservoir. Expansion of the blood volume with blood previously equilibrated with the dog resulted in a significant drop in sodium reabsorption by the proximal tubule and increased sodium excretion. Both dilution of the blood and expansion of the blood volume independently may depress sodium reabsorption by the proximal tubule. (*Knox, F. G., and others: Effect of Dilution and Expansion of Blood Volume on Proximal Sodium Reabsorption, Amer. J. Physiol. 215: 1041 (Nov.) 1968.*)

STORED BLOOD HEMOGLOBIN FUNCTION Serial oxygen dissociation curves were made for blood preserved in ACD,