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,

ACD-adenine, and ACD-adenine-inosine. Dividing blood from a single donor into two or more bags allowed direct comparison of these preservatives. During the first week of storage in ACD, a progressive increase in oxygen affinity was observed. Thereafter, little further change was noted. Oxygen affinity increased even more rapidly during initial storage in ACD-adenine. However, with inclusion of inosine as a preservative, oxygen affinity remained unaltered during the first two weeks. Increases in oxygen affinity correlated well with decreasing levels of erythrocytic 2,3-diphosphoglycerate (2,3-DPg) during storage. No significant accumulation of ferrihemoglobin was detected. When blood stored 20 days in ACD or ACD-adenine was incubated with inosine for 60 minutes at 37 C, 2,3-DPG and adenosinetriphosphate were resynthesized, and oxygen affinity decreased. The distribution of 2,3-DPG in fresh and stored erythrocytes appeared to influence experimental values for heme–heme interaction. (*Bunn, H. F., and others: Hemoglobin Function in Stored Blood, J. Clin. Invest. 48: 311 (Feb.) 1969.*)

VOLEMIA Measurement of left and right atrial and pulmonary arterial pressures in healthy, anesthetized, closed-chested dogs and in dogs subjected to shock provides no reliable index of the state of volemia. Decreases observed with the withdrawal of 40 per cent of the blood volume and increases noted with transfusions of similar magnitude are of brief duration. The dog apparently has an extraordinary species capacity for pooling of blood. (*Waddell, R., and Shumacher, H. B.: Vascular Pressure Changes during Hemorrhage and Transfusion, Surgery 65: 617 (April) 1969.*)

FLUID THERAPY Eight dogs (average weight 23 to 25 kg) were anesthetized with pentobarbital and allowed to breathe spontaneously through an endotracheal tube. Following cannulation of one carotid artery and one jugular vein, erythrocytic volume (RCV) and extracellular fluid (ECF) were measured by ^{51}Cr-tagged erythrocytes and ^{35}S-tagged sodium sulfate, respectively. The dogs were then bled to a mean arterial pressure of 50 mm Hg. Repeated sampling for ^{51}Cr and ^{35}S was started 90 minutes later and continued for 180 minutes and 210 minutes, respectively. Results showed a 5.7 per cent decrease in ECF and 46 per cent and 45 per cent decreases in RCV and PV. These results contrast sharply with those of Shires, who reported a 30 to 40 per cent deficit in ECF under similar circumstances. The differences are attributed to inaccuracies in the techniques used by Shires. The use of large volumes of lactated Ringer's solution during operative procedures and in shock is unwarranted and leads to unnecessary expansion of ECF space. (*Roth, E., Lex, L. C., and Maloney, J. V., Jr.: Ringer's Lactate Solution and Extracellular Fluid Volume in the Surgical Patient—A Critical Analysis, Ann. Surg. 169: 149 (Feb.) 1969.*) ABSTRACTER'S COMMENT: The authors of this paper explained in great detail their investigative methods and the objections they have to techniques used by previous workers in this field. If these objections are valid, it is possible that the practice of administering large volumes of lactated Ringer's solution to patients undergoing major surgical procedures or suffering from shock is unjustified. It will be interesting to see what previous workers, whose techniques are criticized, have to say about this paper.

pH AND LUNG VESSELS The effects of *p*H, P_{O_2} and P_{CO_2} on pulmonary vessels were studied in isolated cat lungs perfused at constant flow with autologous blood at 37 C. Each animal was ventilated with 20, 10, 5 and 2.5 per cent oxygen. P_{CO_2} was either zero or 60 mm Hg. Airway and left atrial pressures were constant. Lactic acid or sodium bicarbonate was infused to alter *p*H. In six lung preparations with P_{CO_2} zero and *p*H 7.6, pulmonary arterial pressure (P_{pa}) responses to hypoxia were attenuated or absent. Reducing *p*H to 7.0, at an oxygen tension 120 mm Hg, caused P_{pa} to increase from 17 to 25 mm Hg and augmented the pressor responses to hypoxia. In six lung preparations with P_{CO_2} at 60 mm Hg and *p*H about 7.6, P_{pa} increased from 14 to 26 mm Hg, with severe hypoxia ($P_{O_2} = 20$ mm Hg); lowering the *p*H to about 7.0, with oxygen tension 120 mm Hg, caused P_{pa} to increase from 14 to 16 mm Hg, and