ume. The hematocrit was then decreased to 17 per cent. The third bleeding removed another 15 per cent of the erythrocyte mass (total 75 per cent removed), and the hematocrit was then 10 per cent. It was replaced with 17 times its volume. Cumulative replacement ratio was 4,880 ml Ringer's solution to 590 ml blood, or 8.3:1. All animals survived but became very edematous and gained 30 to 50 per cent in weight. Diuresis and diarrhea removed 80 per cent of the fluid in 24 hours, with final retention of less than one liter of excess fluid. Urinary output was the best clinical indication of the adequacy of vascular re-expansion. (*Moss, G.: Fluid Distribution in Prevention of Hypovolemic Shock, Arch. Surg. 98: 281 (March) 1969.*)

ENDOTOXIN SHOCK Forty-three dogs were each given 2 mg/kg of *E. coli* intravenously. The animals developed markedly decreased oxygen consumption, increased A-V oxygen difference, increased hydrogen-ion concentration, increased serum lactate and pyruvate concentrations, increased hematocrit, decreased cardiac output and decreased blood volume. Treatment with phenoxybenzamine alone caused death before the two-hour experiment ended. Treatment with phenoxybenzamine plus dextran 70, or levarterenol, or isuprel, increased the oxygen consumption, but after treatment ceased it declined to below pretreatment levels. Isoproterenol was considered to be the most beneficial treatment, although there were no striking differences in effects among the agents studied. (*Anas, P., Neely, W. A., and Hardy, J. D.: Effects of Vasoactive Drugs on Oxygen Consumption in Endotoxin Shock, Arch. Surg. 98: 189 (Feb.) 1969.*)

DEXAMETHASONE IN SHOCK Oligemic shock produces decreased perfusion and anaerobic metabolism, resulting in increased lactate:pyruvate ratios and metabolic acidosis. Corticosteroids enhance gluconeogenesis from lactate, and reduce the amount of free hydrogen ion released by the lactic-acid dissociation. Splenectomized monkeys were bled into shock and treated with pharmacologic doses of dexamethasone. The results were increased survival, less lactate production and less acidosis. (*Schumer, W.: Dexamethasone in Oligemic Shock, Arch. Surg. 98: 259 (March) 1969.*)

STEROID ANTISHOCK EFFECT Corticosteroids given in high doses seem to be beneficial in the treatment of shock, whether endotoxic, traumatic, epinephrine or hemorrhagic in origin. The mechanism of this protective effect is unclear. Both cortisol and dexamethazone given intravenously prior to induction of hemorrhagic shock in the cat prolonged survival significantly after reinfusion of the shed blood. High doses of aldosterone and cortisol were ineffective in prolonging survival when administered at the time of reinfusion. Plasma activity of a myocardial-depressant factor (MDF) was increased in saline-, aldosterone-, and cortisol-treated cats at reinfusion time but was decreased in glucocorticoid-pretreated animals. Thus, an inverse correlation between MDF activity and survival time was found. MDF was found to be a peptide produced in the ischemic intestine, with a molecular weight of approximately 500–1,000. Glucocorticoids may prevent the disruption of lysosomes and/or prevent proteases from being released into the blood. This would prevent cleavage of peptide fragments from plasma proteins, one or several of which may actually be MDF. (*Lefer, A. M., and Martin, J.: Mechanism of the Protective Effect of Corticosteroids in Hemorrhagic Shock, Amer. J. Physiol. 216: 314 (Feb.) 1969.*)

BED REST AND FLUID BALANCE The hemodynamic and metabolic effects of two weeks of absolute bed rest were evaluated in 20 healthy volunteers. Post-recumbency tilting resulted in a more profound decrease in stroke volume and cardiac output than had been present prior to bed rest. This was coupled with failure to augment stroke volume and cardiac output to pre-recumbency levels during a standard exercise after bed rest. A large diuresis and saluresis occurred during bed rest. In spite of this, plasma volume was at pre-recumbency levels after the bed rest. The fluid loss must have been derived from

extravascular compartments, with resultant lowering of tissue pressure. It is likely that a large transudation of plasma water into lower-extremity tissue spaces occurred during post-recumbency tilting. The resultant decrease in plasma volume during tilt would then account for the decrements in stroke volume and cardiac output observed during tilt and exercise. *(Hyatt, K. H., and others: Extravascular Dehydration as an Etiologic Factor in Post-recumbency Orthostatism, Aerospace Med. 40: 644 (June) 1969.)*

INTRAVASCULAR COAGULATION Two patients with disseminated intravascular coagulation secondary to incompatible blood transfusion were studied. Intravascular coagulation may occur more frequently than has been appreciated; hence, laboratory study of hemostasis should be a part of the evaluation of patients who have received incompatible blood. Prompt diagnosis, along with heparin therapy, may prevent the development of a generalized bleeding diathesis. *(Rock, R. C., and others: Heparin Treatment of Intravascular Coagulation Accompanying Hemolytic Transfusion Reaction, Transfusion 9: 57 (March) 1969.)*

CEREBRAL DEATH The effects of ischemic anoxia on the spontaneous electrical activities of the cerebral cortex and the medullary reticular formation were studied in six adult cats immobilized with gallamine. Arterial blood pressure was monitored throughout the experiment. Cerebral ischemia was induced by occlusion of cerebral circulation. Following the occlusion of circulation for four to ten minutes, changes in the electrical activity of the cerebral cortex began before the medullary reticular formation changed. Following occlusion of longer duration (15 min), the cortical activity remained isoelectric, but the reticular activity persisted. Concurrent with the disappearance of the electrical activity of the medullary reticular formation was a decrease in arterial blood pressure; the sustained medullary activity and the maintenance of the arterial pressure coincided. The electrical seizure activity due to ischemia concurred not with the extreme degree of anoxia but rather with the period of recovery from it. *(Fujita, M., and others: An Experimental Study of the Cerebral Death, Jap. J. Anesth. 18: 420 (May) 1969.)*

ADRENAL CATECHOLAMINES Catecholamine secretion from acutely-denervated, perfused cat adrenal glands was studied. Glucose-deprivation plus anoxia caused an increase in catecholamine output from adrenals perfused with normal Locke solution; this was abolished by removal of calcium from the perfusion medium. Anoxia plus glucose-deprivation did not depress the secretory response to repeated exposures of a low concentration of acetylcholine, but did depress the response to a higher concentration of acetylcholine. Cyanide potentiated the secretory response to calcium in the presence of glucose, but when glucose was omitted from the perfusion fluid, cyanide caused a gradual decrease in calcium-evoked secretion. The glycogen content of medullae was profoundly depleted under anoxic conditions. Energy is required for the secretory action of medullary chromaffin cells. This energy may be derived from glycolysis or oxidative metabolism. The alteration in the percentages of adrenalin and noradrenalin secreted during anoxia indicates that anoxia may regulate catecholamine secretion through a peripheral as well as through a central mechanism. *(Rubin, R. P.: The Metabolic Requirements for Catecholamine Release from the Adrenal Medulla, J. Physiol. (London) 202: 197 (May) 1969.)*

INSULIN In dogs the characteristic rise in blood glucose in early hemorrhagic shock is associated with a significant increase in plasma insulin. As shock continues, the levels of both insulin and glucose decrease. An infusion of glucose after prolonged hypovolemia, when metabolic and physiologic functions of the organism have started to deteriorate, elicits another increase in plasma insulin. Possibly, hormonal mechanisms for energy metabolism are preserved after prolonged shock, though changes may be prevented by circulatory effects. *(Bauer, W. E., and others: Insulin Response during Hypovolemic Shock, Surgery 66: 80 (July) 1969.)*