gen consumption. No significant differences in these parameters were demonstrated in this small group of patients. The authors believe that uncomplicated, primary hyperthyroidism is not accompanied by an obligatory increase in activity of the sympathetic nervous system. It is suggested that sensitization of the cardiovascular system, and the metabolic responses, occur in hyperthyroidism when the catecholamine levels are normal. (*Harrison, T. S., and others: Adrenergic Reactivity in Hyperthyroidism, Arch. Surg. 94: 396 (March) 1967.*)

HEMOLYSIS Silastic appears to be the best available surface for conduit tubing with a roller pump, when lipemic blood is used. Occlusive pump settings resulted in considerably higher rates of hemolysis than the nonocclusive settings with all materials except Silastic. (*Bernstein, E. F., and Gleason, L. R.: Factors Influencing Hemolysis With Roller Pumps, Surgery 61: 432 (March) 1967.*)

PHAGOCYTOSIS AND SHOCK Phagocytosis of colloidal carbon by reticuloendothelial cells was studied in rats subjected to intestinal ischemic shock. Three hours after shock, phagocytic activity was depressed in controls, but was increased more than 100 per cent in saline and norepinephrine treated rates and 230 per cent in animals treated with 2-phenylalanine-8-lysine vasopressin (PLV-2). Twenty-four hours after shock, surviving controls and angiotensin and PLV-2 treated rats exhibited increased phagocytosis, while saline and norepinephrine treated animals showed decreased phagocytosis. (*Altura, B. M., and Hershey, S. G.: Use of Reticuloendothelial Phagocytic Function as an Index in Shock Therapy, Bull. N. Y. Acad. Med. 43: 260 (April) 1967.*)

CORTISOL A study of splenectomized dogs before and after adrenalectomy determined the functions of cortisol during stress. With adrenals intact 50 per cent of the plasma deficit was restored within 18 hours. Without adrenals, hemorrhage was followed by a progressive fall in plasma volume and often by death. Without adrenals, but maintained on DOCA and cortisol, only a minimal restoration of plasma volume occurred in 18 hours. However, increased cortisol above the maintenance dose in adrenalectomized dogs enhanced the ability to restore plasma volume to the level of a dog with intact adrenals. (*Marks, L. J., and others: Physiological Role of Cortisol in the Plasma Volume Response to Hemorrhage, Surgery 61: 422 (March) 1967.*)

SHOCK Hemorrhagic shock was produced in dogs and the effects on blood uric acid concentrations caused by infusion of norepinephrine, phenoxybenzamine or isoproterenol were measured. In one hour blood uric acid rose 7.5-fold with norepinephrine, 3.5-fold with isoproterenol, and no appreciable change occurred with phenoxybenzamine. When compared with results in untreated animals, isoproterenol had no significant effect, whereas norepinephrine greatly increased and phenoxybenzamine greatly reduced the blood uric acid concentration. (*Cowsert, M. K., Jr., and Carrier, O., Jr.: Effect of Some Autonomic Drugs on the Uric Acidemia of Hemorrhagic Shock, Canad. J. Physiol. Pharmacol. 45: 169 (Jan.) 1967.*)

SHOCK AND HYPERBARIC O_2 Following control measurement of P_{O_2}, glucose, lactate and pyruvate in femoral arterial and femoral, hepatic, portal and renal venous blood, two groups of dogs were bled to a mean arterial blood pressure of 50 mm. of mercury and maintained at this level for one hour. During the period of hemorrhagic shock, one group breathed oxygen at atmospheric pressure while the second group breathed oxygen at three atmospheres. Control values were comparable in both groups. After one hour of hemorrhagic shock, venous P_{O_2} from all sites was significantly lower than control in both groups. Arterial "excess lactate" was present in both groups during shock and was not significantly decreased by hyperbaric oxygen. Without other treatment to support perfusion, hyperbaric oxygen did not prevent the stagnant hypoxia of hemorrhagic shock. (*Cain, S. M., and Connolly, J. M.: Tissue Oxygenation During Hemorrhage in Dogs Breathing 1 and 3 Atmospheres of Oxygen, J. Appl. Physiol. 22: 255 (Feb.) 1967.*)