ADRENOCORTICAL RESPONSE Clinical studies on 21 male patients have confirmed animal studies that major surgical procedures under ether anesthesia can produce maximal pituitary adrenocortical activation. The administration of ACTH immediately after surgery produced a further rise in plasma hydroxycorticosteroid values despite the fact that post-operative steroid levels were already elevated. The increase in adrenocortical capacity to respond to ACTH after surgery may last as long as ten days after operation. It may be due to a possible relationship of the secretions of endogenous ACTH to postoperative alterations. (*Estabrooks, R. A., and others: Effect of Surgical Operation on Adrenocortical Response to ACTH, Ann. Surg. 150: 941 (Nov.) 1959.*)

ADRENOCORTICOSTEROID METABOLISM Studies of adrenal vein blood in various animals show that cortisol, corticosterone and aldosterone are the major corticosteroid secretory products. In man cortisol is the major glucocorticoid steroid, and aldosterone very probably is the major mineralocorticosteroid. Corticosterone, which man secretes in small quantities is the major adrenal steroid of certain other species (*e.g.*, rodents). These steroids exist in blood both in aqueous solution and bound to protein. Most of plasma cortisol and corticosterone is bound. About 70 to 80 per cent of aldosterone is bound by serum albumin. If the bound steroid represents an inactive transport complex, only the free steroid is biologically active and capable of diffusion into the extravascular space and cells. Protein binding may also prevent glomerular filtration of the steroid and protect it from excessively rapid transformation by the liver. Protein binding thus may affect distribution, transport, excretion and thus, biotransformation of the steroid. Metabolism by the liver is the most important influence on the rate of disappearance from plasma. The rate of disappearance is decreased in liver disease, myxedemia, pregnancy, old age, starvation, and estrogen therapy. (*Peterson, R. E.: Metabolism of Adrenocorticosteroids in Man, Ann. New York Acad. Sc. 82: 846 (Oct. 14) 1959.*)

DEEP HYPOTHERMIA Dogs were anesthetized with thiopental and nitrous oxide, their tracheas intubated, their chests opened and hearts exposed, and extracorporeal circulation established by means of a Melrose type pump-oxygenator. Cooling was effected by placement of a specially developed heat exchanger distal to the oxygenator. In one group of dogs, body temperature was lowered rapidly to 3 to 5 C. (hepatic). The animals were then rapidly rewarmed. In the first 10 of this group rewarming was discontinued at 37 C. All of these animals showed a tendency to hemorrhage and failed to breathe spontaneously. In the next 8 animals, rewarming was continued to 40 C. Six of these dogs survived. In another group, 22 dogs were rapidly cooled to hepatic temperatures of 3 to 5 C. All extracorporeal circulation was discontinued and the blood volume was drained into the oxygenator. The animals were kept in a state of suspended animation for 30 to 40 minutes. Rewarming was then started after the blood was pumped back into the corporeal circulation. Twelve of these dogs died of metabolic acidosis. When the acidosis was treated by an infusion of potassium chloride, calcium gluconate and isotonic bicarbonate solution most of the remainder survived. (*Kenyon, J. R., and others: Experimental Deep Hypothermia, Lancet 2: 41 (July 18) 1959.*)

HYPOTHERMIA The capability of the left ventricle to perform work after elective cardiac arrest with potassium citrate has been investigated. Measurements were made with body temperature at 37 C. and at 28 C. Measurements were obtained of cardiac output, heart rate, mean arterial blood pressure, and mean left atrial pressure. At 28 C., after cardiac arrest with potassium citrate, the left ventricle could perform more than 15 gram-meters of stroke work, whereas at 37 C. the left ventricle was unable to exceed 8 gram-meters of stroke work. These results indicate that hypothermia offers partial protection to the myocardium subjected to cardiac arrest with potassium citrate. (*Cooper, T., and others: Myocardial Function after Elective Cardiac Arrest during Hypothermia, Surg. Gynec. & Obst. 109: 423 (Oct.) 1959.*)