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, Acrospace 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.*)