the time required for half-removal of intravenously injected protein was found to be prolonged 4–5 times. It is concluded that administration of ACTH and cortisone lowers normal vascular permeability. (*Mednik, G. L.: Influence of ACTH and Cortisone on Vascular Permeability, Probl. Endokr. 2: 40 1957.*)

SERUM SODIUM Simultaneous measurements of serum sodium concentration, serum osmolarity, total exchangeable sodium, total exchangeable potassium and body water were made in a group of chronically ill patients. Serum osmolarity correlates closely with serum sodium concentration. Serum sodium concentration is only partially or poorly correlated with total exchangeable sodium, total exchangeable potassium, and total body water as related to body weight. There is a high degree of correlation between serum sodium concentration or "corrected" serum osmolarity and the ratio of sodium plus potassium divided by total body water. Body water appears to be passively distributed in proportion to osmotic activity. All or almost all of the body potassium is osmotically active. (*Edelman, I. S., and others: Interrelations Between Serum Sodium Concentration, Serum Osmolarity and Total Exchangeable Sodium, Total Exchangeable Potassium and Total Body Water, J. Clin. Invest. 37: 1236 (Sept.) 1958.*)

POTASSIUM EXCESS Rats were subjected to potassium loading which increased stepwise from a normal intake level of 1.6 to as high as 36.8 mEq. per 100 Gm. of body weight per day. These animals showed no signs of toxicity, electrocardiographic changes or elevation of plasma potassium concentration values until the intake exceeded 25.6 mEq. Of the animals taking more than this amount, one-half died with signs of potassium intoxication within three days. Measurements on the surviving half of the group disclosed pathologic elevation of plasma potassium concentration and characteristic electrocardiographic changes, but no significant changes in total carcass potassium content or concentration values and a significant decrease in cardiac intracellular potassium concentration. These findings were in contrast to those in animals on an ordinary dietary intake in which some intracellular "storage" of potassium may be observed. It is suggested that the restoration of cellular potassium values to normal following an acute load and the maintenance of these values at normal levels even in the presence of sustained hyperkalemia under conditions of chronic loading may be mediated by adrenocortical hormones. (*Drescher, A. N., and others: A Study of the Effects of Excessive Potassium Intake Upon Body Potassium Stores, J. Clin. Invest. 37: 1316 (Sept.) 1958.*)

ALDOSTERONE Decreased secretion of aldosterone has been reported in: (1) low potassium diets; (2) desoxycorticosterone acetate, prednisone, amphernone and pitressin administration; (3) renal hypertension; (4) atrial stretching; and (5) manipulation of certain central nervous system functions in various animals. Increased output has been reported in: (1) sodium deprivation, potassium administration, and low chloride intake; (2) choline deficiency; (3) ouabain administration; (4) thyroid dysfunction; (5) blood loss or contraction of body water; (6) changing from horizontal to upright position; and (7) surgery. From these observations and from data derived from decapitation and from serial cranial sections, a diencephalic or mid-brain center seems to control the secretion of a humoral tropic factor (possibly in part via the pituitary) which stimulates aldosterone secretion. The center receives stimulating or inhibitory impulses from unidentified peripheral receptors, parts of the brain stem, and higher centers (emotion, anxiety). It responds primarily to the level of serum electrolytes, which may act on it directly or by way of chemoreceptors, and to volume receptors located in the atria and possibly the great veins. The volume receptors normally seem to play a minor role in controlling aldosterone secretion, but with extreme reduction in body water, the volume receptors provide the more important stimuli. (*Farrell, G.: Regulation of Aldosterone Secretion, Physiol. Rev. 38: 709 (Oct.) 1958.*)

MONOAMINE OXIDASE Monoamine oxidase is the enzyme which is responsible for the oxidative deamination of such monoamines as epinephrine, butylamine and tyramine. It