

Antidiuresis in Dogs, Ann. Surg. 147-375 (Mar.) 1958.

DIURETICS AND ARRHYTHMIAS

Mercurial diuretics and carbonic anhydrase inhibitors influence heart rhythm indirectly by aiding in the re-establishment of cardiac compensation. In some cases they suppress arrhythmias, particularly those caused by the increased burden imposed on the failing heart muscle. However, possibly by increasing potassium excretion and thereby enhancing digitalis effects, they may also be instrumental in the causation of various arrhythmias. On the other hand, chlorothiazide seems not to cause an overt hypokalemia; and in patients in whom chlorothiazide has been substituted for other diuretics, the electrocardiographic signs of digitalization often diminishes. (Pines, I., Sulazar, E., and Lopez, T.: *Antiarrhythmic Properties of Chlorothiazide, Ann. New York Acad. Sc. 71: 380 (Feb.) 1958.*)

ELECTROLYTE EXCRETION

Sodium is probably reabsorbed continuously throughout the length of the tubule. Proximally, this process is manifested by a reduction in volume and the fluid remains isotonic with the plasma. In the loop of Henle and in the distal tubule the removal of sodium results in dilution. Limited observation on potassium suggests that in the proximal tubule reabsorption is about proportional to volume flow. More potassium is apparently added to the urine at some unidentified point in exchange for sodium. The amount of sodium presented to the "exchange mechanism" is a critical determinant of the rate of secretion. The secretion of hydrogen ion is also involved in the exchange for sodium. Hydrogen ion secretion also seems to be responsible for the reabsorption of bicarbonate and for the acidification of urine. Finally, chloride reabsorption begins in the proximal tubule and continues into the distal segment, approximately paralleling that of sodium. (Berliner, R. W.: *Renal Transport of Electrolytes, Ann. New York Acad. Sc. 71: 324 (Feb.) 1958.*)

KRYPTON 85 Krypton 85, a radioactive form of a harmless inert gas was used to detect cardiac left to right shunts in

100 patients. The radioactive gas was introduced into the left side of the heart by having the patient breathe a mixture of the gas and air from an ordinary anesthesia bag. Samples of blood for analysis were obtained from the right side of the heart through a catheter. Radiation from Krypton 85 is nonpenetrating and elaborate precautions during its use are not necessary. (Sanders, R.: *Use of Radioactive Gas (Kr^{85}) in Diagnosis of Cardiac Shunts, Proceed. Soc. Exper. Biol. & Med. 97: 1 (Jan.) 1958.*)

HYDROXYDIONE

Hydroxydione (Vindril) in four times the dose of thiopental was less depressant to the cardiovascular and respiratory systems of anesthetized dogs and cats. Hydroxydione produced pronounced relaxant and antispasmodic effects on ileal and tracheal smooth muscle. (Das, P. K., and Arora, R. B.: *General pharmacological Properties of 21-Hydroxy-Pregnanedione Sodium Succinate, Intravenous Anesthetic Agent, J. Pharmacol. & Exper. Therap. 121: 149 (Oct.) 1957.*)

STRESS

Ether anesthesia suppressed the increase in prothrombin time which usually followed electroshock of rats. (Morgenson, G. J., Fisher, L. M., and Jaques, L. B.: *Effect of Stress and Adrenalectomy on Response to Dicumarol in Rats, Canad. J. Biochem. and Physiol. 36: 51 (Jan.) 1958.*)

ETHER

Urinary adrenaline and noradrenaline estimations in the rat can serve as indications of an alteration of sympathetic activity following administration of a drug or some other deviation from normal. Use of this method revealed that ether anesthesia probably had no effect on noradrenaline output but caused a significant rise in adrenaline output. A single dose of morphine produced no significant rise in sympathin excretion but four doses at hourly intervals did cause a significant rise. Rats from which the adrenal medullas had been removed showed an increased urinary excretion of noradrenaline but not adrenaline after four doses of morphine. No adrenaline was detected in the urine of demedullated rats after ether anesthesia. (Crawford, T. B.