34 (1616)

The potassium content of normal and some pathological human bloods.

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Our interest in the potassium content of human blood was aroused some time ago by the observation of Smillie[1] that poisoning may result from the administration of potassium salts to nephritic patients, and later confirmed in experimental uranium nephritis.

Observations on the potassium content of serum and whole blood have been made in fifteen cases, including four normal subjects, several cases of nephritis with marked nitrogen retention and a few miscellaneous cases. The potassium estimation was carried out by the cobaltinitrite method of Drushel essentially as described by Myers[2] in 1909 for spinal fluid.

The results obtained for serum in the four normal cases were somewhat less than 20 mg. per 100 c.c. calculated as K, and for whole blood about 8 to 10 times this amount. In five cases of nephritis with marked nitrogen retention the figures for the serum varied from 10 to 19 mg., in one mild case the potassium was 28 mg. while in a case of double polycystic kidney 35 mg. were found. The potassium content of the whole blood in these cases ranged from 52 to 148 mg. with an average of 100 mg. per 100 c.c. In all of these cases there was an associated secondary anemia and the total solids were diminished. No significant variation was found in a case of pure hypertension or a case of diabetic coma. Figures obtained for whole blood in two cases of pernicious anemia were low owing to the diminution in the red cell content.

In general the potassium content of whole blood tends to vary directly with the red cell content and the percentage of total solids. The few observations reported on cases of nephritis with marked nitrogen retention do not appear to support the suggestion that possibly some of the symptoms of uremia are due to a potassium poisoning as a result of retention of this element. Serum

[1] Smillie, *Arch. Int. Med.*, 1915, xvi, 330.

[2] Myers, *Jour. Biol. Chem.*, 1909, vi, 115.

appears preferable to plasma for potassium determinations since hemolysis seems to be much more readily prevented.

35 (1617)

On the elimination of phenolsulphonephthalein in acute mercuric chloride intoxication.

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In two recent publications [1,2] on the toxic effect of mercuric chloride in normal and in naturally nephropathic animals, observations have been made concerning the relationship between the development of an acid intoxication and the acute kidney injury. In these animals the poison was given in large doses, 15 mgs. per kilogram, which eliminated the study of the development of the intoxication in the early stages.

The following preliminary note has as its object a study of the early stages of the development of an acid intoxication from mercuric chloride and the relationship of such a disturbance to renal function as is shown by the elimination of phenolsulphonephthalein.

Fourteen normal dogs have been used in the study. The animals were kept in metabolism cages, fed on bread with a small amount of cooked meat and given 500 c.c. of water by stomach tube daily. The animals were catheterized once a day and the urine examined for albumin and glucose. Centrifugalized samples were examined for casts. The phenolsulphonephthalein test was conducted according to the technique of Rowntree and Geraghty. Blood urea determinations were made by the method of Marshall as modified by Van Slyke and Cullen. The reserve alkali of the blood (R.p.H.) was determined by the method of Marriott. After the commencement of the intoxication the urine was ex-

[1] MacNider, Wm. deB., "A Study of Acute Mercuric Chloride Intoxication In The Dog with Special Reference To The Kidney Injury," *Jour. Exp. Med.*, Vol. XXVII, 519, 1918.

[2] MacNider, Wm. deB., "The Susceptibility of Naturally Nephropathic Animals to Acute Mercuric Chloride Intoxications," *Jour. Med. Research*, Vol. XXXIX, 461, 1919.