did not give an indication of a change in the distribution of the various acid-soluble phosphate compounds of muscle. Since the zero value of the hydrolysis curves represents the sum of inorganic and phosphocreatine P, there remained the possibility of an increase in phosphocreatine at the expense of inorganic phosphate. In 2 experiments on cats (Table I) and 3 experiments on rats (Table II) an increase in phosphocreatine P could not be detected though there was, at least in 2 experiments, a decrease in inorganic phosphate.

Summary. The decrease in the inorganic phosphate content of plasma following the injection of glucose plus insulin is not accompanied by a detectable increase in the hexosemonophosphate, adenosinetriphosphate or creatine phosphate content of muscle.

9102

Changes in Renal Blood Flow in Relation to Changes in Pressure in Urine Formation.

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In conjunction with certain experiments on the mechanism of chloride reabsorption by the tubules of the kidney of the dog in a heart-lung perfusion system as described by Verney and Starling,[1] we have had occasion to observe some interesting relations between renal blood flow, perfusion pressure, chloride concentration and total chloride excreted in the urine.

Winton[2] has pointed out that the rate of blood flow through the kidney is a factor of secondary importance, as compared with the glomerular filtration pressure, in determining the character and quantity of urinary secretion. Numerous observations have indicated to us an indubitable relation, nevertheless, between renal blood flow and urinary secretion, particularly in the case of spontaneous changes in the former.

Figure 1 shows graphically the results of a type of experiment in which concomitant with a rising renal blood flow, from 110 to 210 cc. per minute, while the perfusion pressure was kept substantially constant for 35 minutes, there was an increase in urine flow from

1 Verney, E. B., and Starling, E. H., *J. Physiol.*, 1922, **56**, 353.

2 Winton, F. R., *J. Physiol.*, 1931, **73**, 151.

0.35 cc. to +.8 cc. per minute, and an increase in chlorides from 50 to 150 mg. %. This sort of behavior has been noted in a number of instances after the kidney has begun to secrete well.

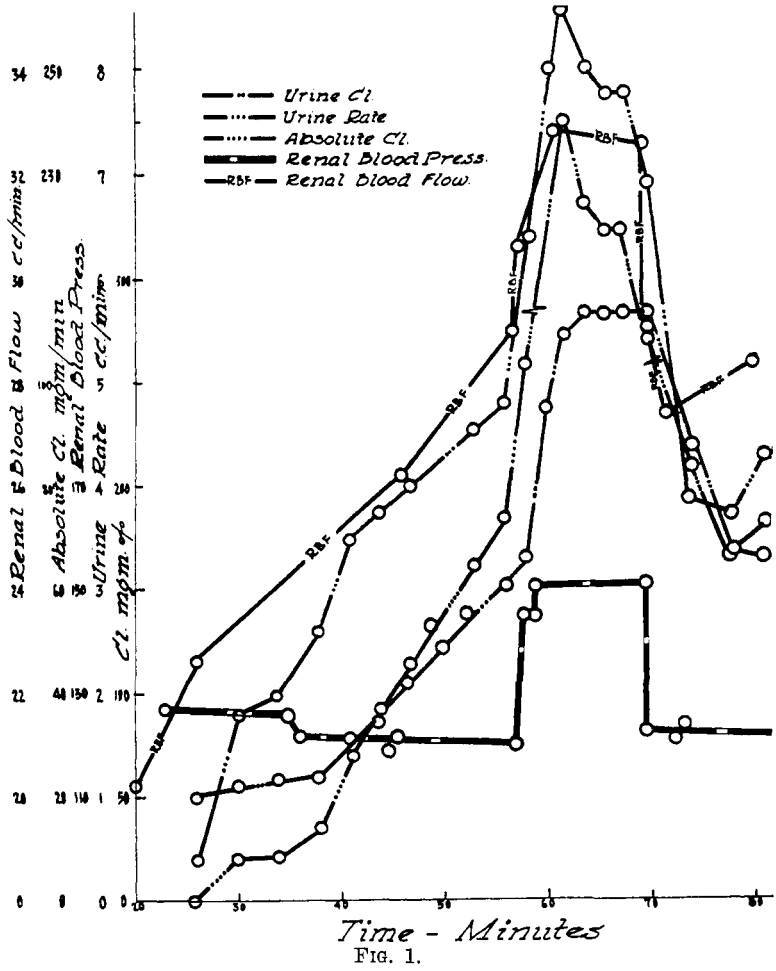

FIG. 1.

These observations are not in any sense contradictory to those of Winton, because he was dealing with changes induced in the rate of blood flow by altering the pressures in the renal vein and the ureter. Moreover, we believe that our observations can be interpreted best by ascribing the results to changes in the state of constriction of certain portions of the vascular bed in the kidneys resulting in changes in glomerular filtration pressure as outlined below.

An increase in the rate of renal blood flow at constant perfusion pressure could result only from a vasodilation in some regions of the

vascular bed of the kidney. This could conceivably be in the afferent arterioles, the efferent arterioles or in the veins.

The evidence from the changes in urinary secretion gives a clue as to the site of the vascular dilatation. With the increase in renal blood flow there were observed large increases in the volume of urine formed and in its chloride content. These increases bespeak an increased glomerular capillary pressure, bringing about augmented filtration. Tubular activity is largely independent of blood flow and pressure (Winton[2]), therefore with increased glomerular filtration the chloride content of urine is increased. With constant renal arterial pressure an increase in glomerular capillary pressure along with an increased blood flow could not result from dilatation of any blood vessels in the kidney except the afferent arterioles. Dilatation of either the efferent arterioles or the veins would result in a decreased glomerular capillary pressure. Thus the changes observed, seem to be due primarily to a dilatation of afferent glomerular arterioles.

In the portion of the experiment shown in Fig. 1 from the period 55 to 70 minutes, the perfusion pressure was increased from 80 to 150 mm. Hg. This resulted in an increase in urine flow from 4.8 to 8.5 cc. per minute, and in chloride concentration, from 150 to 280 mg. %. One sees illustrated here the fact described by Canny, Verney, and Winton[3] that with increased rates of glomerular filtration the tubular reabsorption of chloride does not keep pace, and consequently the chloride concentration in the urine rises. The reabsorption of chloride falls behind the net rate of reabsorption of water, otherwise only the absolute chloride elimination and not the chloride concentration would rise.

The absolute chloride excretion rose 300%, considerably more than the chloride percentage, but the latter increased nearly 100%. The tubules obviously do not have a capacity to maintain constant concentration ratios for chloride at varying rates of formation of glomerular filtrate.

These observations on the effect of increased arterial pressure on the composition and volume of the urine are in support of some of the preceding arguments, in particular regarding the interpretation of simultaneous increases of volume and chloride content.

Twenty-five successful heart-lung kidney experiments have been conducted and the type of spontaneous change described has been encountered in one-fourth of the cases. The interpretation of these spontaneous changes is particularly important, we believe, because

[3] Canny, A. J., Verney, E. B., and Winton, F. R., *J. Physiol.*, 1930, **68**, 333.

the extreme hypotonicity of heart-lung-kidney urine, and its low chloride content, have been difficult to interpret. The urine in this preparation differs in chloride content so markedly from that in the intact animal that the reasons for the difference must be elucidated before the isolated perfused kidney results can be applied to normal renal physiology. The findings reported here indicate that the hypotonic urine may be due, in part, to a relatively low glomerular filtration rate caused by greater constriction in afferent than in efferent arterioles.

9103

A Comparison of Some Methods for the Extraction of Vitamin B_1 from International Standard Acid Clay.

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Williams and co-workers[1] in their description of an improved method for obtaining crystalline vitamin B_1 from rice polish have stated that by using barium hydroxide to extract the vitamin from activated fullers' earth no more than a 50% recovery could be obtained. For this reason they chose to use quinine sulfate to displace the vitamin from fullers' earth. Recently Kinnersley and Peters[2] have reported that by the use of a baryta extraction method, vitamin B_1 can be quantitatively removed from the acid clay used as the international standard. However, they made no comparison of the baryta method of extraction with the quinine extraction method proposed by Williams. In this communication we present the results of our comparison of the Williams' extraction method with other proposed methods together with some data on the potency of crystalline vitamin B_1 in terms of international units.

The method of assay used throughout this work was the Ammerman and Waterman[3] modification of the Smith curative procedure. In most cases the test substance was injected subcutaneously since as reported by Kinnersley and Peters and confirmed by us there is no appreciable difference in the response of the animal to oral or

1 Williams, Waterman and Keresztesy, *J. Am. Chem. Soc.*, 1934, **56**, 1187.

2 Kinnersley and Peters, *Biochem. J.*, 1936, **30**, 985.

3 Ammerman and Waterman, *J. Nutrition*, 1935, **10**, 25.