value of liver glycogen being that found in the liver sample before the administration of the glucose, adrenalin, or insulin.

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Studies of Renal Metabolism.

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A kidney is drawn out of the body cavity of a dog and sutured under the skin of the back, with the renal vein located in such a position that it can be punctured through the skin with a needle. The metabolism of the kidney has been studied by simultaneous analyses of arterial and renal bloods and of urine secreted during the observation periods. The rate of blood flow through the kidney has been estimated by comparing the amount of urea removed from a unit volume of blood in passing through the kidney with the amount excreted per minute. In most of our experiments only one kidney at a time has been studied; one is brought out under the skin, and the other is removed, in order to simplify the experiments. In a few controls in which one kidney was brought out and the other left *in situ* excretion was similar in both.

Through a single kidney in dogs of 15 to 20 kilos weight the blood flow is in the neighborhood of 200-250 cc. per minute. Increasing the urea content of the blood as much as 10-fold does not significantly accelerate the blood flow. The urea excretion increases 10-fold, but the increase is due to the fact that the proportion of blood urea removed during perfusion of the kidney remains constant, so that the amount removed per liter of blood passing through the kidneys rises in proportion to the amount present in the blood. This explains the manner in which the blood urea clearance is kept constant during wide fluctuations in blood urea content.

The proportion of oxygen removed from the blood by the kidney is rather low, 10 to 20% of the arterial oxygen content as a rule. Increasing the work of the kidney in the form of urea excretion does not markedly affect the oxygen consumption.

Occasionally we have noted temporary cessation of removal of urea from the renal blood. Such cessation apparently occurs as the result of trauma in tapping the renal vein. The cessation appears to be an all or none phenomenon. When it occurs the urea content of the renal vein rises as high as, and sometimes higher than, the urea content of the arterial blood.

The occurrence of renal blood urea contents markedly higher at times than arterial urea contents appears to be proof of the occurrence of reabsorption of urea from the kidneys.

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Production of Anatomical Lesions in the Isolated Organ.

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Since the frog's kidney has proved such a suitable organ for the investigations of the physiologist it seems likely that an examination of its structure and activity under abnormal conditions might be of value. The present report concerns one aspect of such a study. We have previously described the lesions in the frog's kidney when renal toxic agents, including potassium bichromate, corrosive sublimate, uranium nitrate and snake venom, are injected into the living animal.[1] The next step has been the elaboration of a method to test their functional activity, and in this procedure it was found advisable to isolate the organ and perfuse it with a modified Locke's solution.[2] While it was functioning normally under these controlled conditions the same toxic agents were administered by way of the perfusion fluid and the resulting disturbance of functional activity noted. These will be described at another time.[3] The tissues were then fixed and examined histologically.

In this study our first procedure was to determine that normal perfusion of the organ produced no structural changes. It was found that after 6 hours of such treatment the most delicate cytological structures such as the brush border of the epithelial cells, the mitochondrial elements and the achromatic spindle of mitotic figures, which are occasionally found in the frog's kidney, were entirely normal in appearance.

In the tissues of the damaged kidneys all the pathological lesions that had developed in the kidneys of the living animal after poisoning with the same toxic agents were observed, except those which

[1] Oliver, J. and Smith, P., *J. Exp. Med.*, 1930, **52**, 181.

[2] Oliver, J. and Shevky, E., *J. Exp. Med.*, 1929, **50**, 15; *Am. J. Physiol.*, 1930, **93**, 363; MacKay, E., and Oliver, J., *J. Exp. Med.*, 1930, **51**, 161.

[3] *J. Exp. Med.*, in press.