11477

Respiration of Kidney Cortex in High Potassium-Low Sodium Ringer's Solution.*

ALVIN E. LEWIS AND JOHN FIELD, 2D.
From the Department of Physiology, Stanford University.

Carr and Beck[1] have shown that in the brief life span of an albino rat after bilateral adrenalectomy there is a gradual decrease of about 25% in the basal metabolic rate, when the animal is maintained on a "normal" diet. The experiments reported here are part of a series designed to determine the cause and effect relationship of this decreased metabolic rate in the syndrome of adrenal insufficiency. In this instance an attempt was made to see whether the typical serum electrolyte picture in advanced adrenal insufficiency would reduce the respiration of kidney slices from normal rats. The kidney was chosen for study because of its importance in producing the adrenal insufficiency syndrome[2] and because it has been shown by Crismon and Field[3] that there is a decrease in kidney respiration in the adrenalectomized rat of about 38%.

Kidney slices were prepared with the Terry razor microtome which proved very satisfactory for this purpose.[4] The oxygen consumption of kidney cortex was measured by the Warburg method.[5] The suspension medium used in control experiments was the mammalian Ringer's of Dickens and Greville,[6] hereinafter termed D.G.-Ringer's. For the experimental series the medium was a high potassium-low sodium modification of mammalian Ringer's solution osmotically balanced with glucose, hereinafter called A.I.-Ringer's. The concentrations of electrolytes in this medium were those reported for blood of adrenalectomized animals by Grollman[7] and Hegnauer and Robinson.[8] The compositions of these solutions are summarized in Table I.

* Supported in part by a grant from the Rockefeller Fluid Research Fund of the Stanford University School of Medicine.

1 Carr, C. J., and Beck, F. F., *Am. J. Physiol.*, 1937, **119**, 589.

2 Loeb, R. F., *Glandular Physiology and Therapy*, Chicago, The American Medical Association, 1935, Chapter 20.

3 Crismon, J. M., and Field, J., *Am. J. Physiol.*, 1940, in press.

4 Terry, B. T., *Am. J. Clin. Path.*, 1937, **7**, 69.

5 Field, J., Belding, H. S., and Martin, A. W., *J. Cell. Comp. Physiol.*, 1939, **14**, 143.

6 Dickens, F., and Greville, G. D., *Biochem. J.*, 1935, **29**, 1468.

7 Grollman, A., *The Adrenals*, Baltimore, Williams and Wilkins Co., 1936, 184-188.

8 Hegnauer, A. H., and Robinson, E. J., *J. Biol. Chem.*, 1936, **116**, 769.

TABLE I.

	D.G.-Ringer* g per liter	A.I.-Ringer* g per liter
NaCl	7.00	5.4
KCl	0.18	0.36
$MgCl_2 \cdot 6H_2O$	0.1627	0.1956
$CaCl_2 \cdot 2H_2O$	0.25	0.25
Glucose	2.00	9.80

*Both solutions were buffered at pH 7.4 with sodium phosphate in final concentration of M/150.

Thirty-two determinations were made in D.G.-Ringer's solution and 39 in the A.I.-Ringer's solution. Since the tissues were obtained from 9 white rats (Slonaker-Wistar strain), the arithmetic means of the results obtained on each animal were analyzed statistically as paired data. The mean oxygen consumption, N.P.T., per mg (dry weight) in one hour was 16.954 cu mm in D.G.-Ringer's solution and 16.098 cu mm in A.I.-Ringer's solution. The mean difference was 0.855; the standard deviation was 1.29; the standard error was 0.489, and the value of "t" (Fisher, 1936) was 1.7485. This indicates that this small difference in oxygen consumption could occur by chance more than 5 times in a hundred. Thus, the difference observed is not statistically significant. Although the mean difference observed may actually exist, as might possibly be shown by a more lenient statistical method, this observed fall in oxygen consumption, even at its maximum, is not of the order of magnitude of that observed for tissues of adrenalectomized animals.

It can readily be seen that this experiment represents a very limited reproduction of the situation occurring in adrenal insufficiency. Although the electrolyte content of the A.I.-Ringer's solution approximates the blood picture occurring in fairly extreme cases of adrenal insufficiency, there are certain aspects of the total picture which have necessarily been omitted. Thus, for example, the time factor has been completely neglected. Whereas the electrolyte change occurring in adrenal insufficiency is slow and chronic, this experiment, as a first approach to the evaluation of the electrolytic factors, necessarily represents an acute situation.

To the extent that the experimental situation produced here *in vitro* is comparable to the electrolytic imbalance occurring in adrenal insufficiency it would appear that the changed electrolyte content does not have a direct rôle of significant magnitude in the depression of the oxygen consumption of the kidney cortex.