

cellular fluid of the inflammatory reactions of the skin of the guinea pigs tested did not appear to be associated with the loss of ascorbic acid, as reported in previous work.¹ Comparable losses were found in the ascorbic-acid concentration of the skin lesions induced by diphtheria toxin and by heat, but the change in the Eh of the extracellular fluid of the skin in the reaction induced by heat was in the direction of a more positive potential while that of the toxin reaction was toward a more negative potential than was observed in the normal animals.

11819

Nature of the Change in Resistance of Red Cells to Hemolysis.

PRISCILLA M. PORTER. (Introduced by Eric Ponder.)

From the Biological Laboratory, Cold Spring Harbor, N. Y.

It is known in hemolysis experiments that a suspension of red cells varies in its resistance to hemolysis, becoming more resistant the longer it stands. Whether this change is due to an inhibitory substance which is given off, or whether the cell membrane is altered, is not known.

We therefore did experiments to determine: (1) whether the phenomenon has any temperature dependence, and (2) whether an inhibitory substance can be detected in the supernatant saline of a red cell suspension which had been standing at 39° C for about 3 hours.

In the first experiment, a standard suspension (1 cc rabbit red cells in 20 cc of 1% saline) was hemolysed by varying dilutions of saponin (1-10,000 to 1-50,000) at 16-18°C and at 39°C immediately after making the suspension and again after the suspension had stood for one hour.

In the second experiment, a standard suspension was left in a water bath at 39°C for 3 hours. It was then centrifuged, and the supernatant saline was used as the 0.8 cc of saline in the hemolysis of a fresh cell suspension.

The first experiment showed that the resistance of the cell suspension increased by varying amounts (from 0.1% to 17%) during the hour experimental period. These values might be indirectly a

measure of the quantity of an inhibitory substance given off by the cell suspension. This loss, however, does not depend on the temperature at which the suspension was hemolysed.

In the second experiment, there was essentially no difference in the hemolysis time between the experimental and the control tubes.

These results show that the resistance of a red cell suspension increases, but as no appreciable inhibitory substance can be recovered from the saline of a suspension which has been standing, this increased resistance is probably due to a change in the cell membrane. Such a change, possibly of the nature of disorientation in a highly oriented cell membrane, is not unlikely to occur.

11820

Effect on Body Growth of Small Doses of Testosterone Propionate Administered at Different Seasons.

H. S. RUBINSTEIN AND M. L. SOLOMON.

From the Research Laboratory, Surgical Division, Sinai Hospital, Baltimore, Maryland.

Since the observation that castration in the male albino rat was followed by growth inhibition,¹⁻⁶ studies have been carried out to determine whether growth could be stimulated by the injection of male sex hormones. The first attempts made with daily injection of 1 mg of testosterone propionate resulted, surprisingly enough, in growth depression.⁷ This seeming paradox was considered to have resulted from the inhibition of the pituitary growth function induced by excessive hormonal dosage.⁷⁻¹⁴

¹ Rubinstein, H. S., Abarbanel, A. R., and Kurland, A. A., *Endocrinol.*, 1939, **25**, 397.

² Donaldson, H. H., and Hatai, S., *J. Comp. Neurol.*, 1911, **21**, 155.

³ Evans, H. H., and Simpson, M. E., *Anat. Record*, 1937, **35**, 36.

⁴ Korenchevsky, V., *J. Path. and Bact.*, 1930, **33**, 607.

⁵ Commings, W. D., *J. Exp. Zool.*, 1932, **63**, 573.

⁶ Lawless, J. J., *Anat. Rec.*, 1936, **66**, 455.

⁷ Rubinstein, H. S., Kurland, A. A., and Goodwin, M., *Endocrinol.*, 1939, **25**, 724.

⁸ Hamilton, J. B., and Wolfe, J. M., *Endocrinol.*, 1938, **22**, 360.

⁹ Steinach, E., and Holzknacht, G., *Arch. f. Entwicklungsmech. d. Organ*, 1917, **42**, 490.

¹⁰ Bugbee, E. P., and Simmond, A. E., *Endocrinol.*, 1926, **10**, 360.

¹¹ Spencer, J., D'Amour, F. E., and Gustavson, R. G., *Endocrinol.*, 1932, **16**, 647.