present work the animals were not infected until 2 to 3 weeks after operation at a time when the general resistance is not as severely affected but the spleen and lymphoid tissue have undergone pronounced atrophy (Perla[8]). The results of these experiments is further proof of the effect of hypophysectomy on the functional response of splenoid tissue.

Summary and Conclusions. Hypophysectomy in rats was followed by a decrease in resistance to a protozoan infection such as *Trypanosoma lewisi.* The infection was prolonged and the intensity of the infection exceeded that of the controls. The administration of alkaline extracts of anterior lobe, rich in spleen-stimulating factor restored the resistance to the normal level.

It is likely that the altered resistance to protozoan infection in hypophysectomized rats is in part attributed to impairment of the function of the splenoid tissue.

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Comparative Experimental Studies on Symptomatology and Anatomical Changes Produced by Anoxic and Insulin Shock.

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Thirty-eight rabbits were exposed to anoxia for short periods during which the oxygen percentage in the mixture with nitrogen was so slowly decreased as to avoid excitement, though in some instances the anoxia was prolonged until respiration ceased. This slow deprivation of oxygen is termed anoxic shock. The rabbits were exposed to from 1 to 51 anoxic shocks during a period of from 1 to 60 days. The duration of a single shock varied from 15 to 60 minutes. Twenty-nine other rabbits were injected with insulin sufficient to produce coma and convulsions.

The symptoms of the insulin and anoxic shock were similar except that those due to anoxia occurred on a more rapid time scale. Anoxia was characterized by increase of the respiratory rate, dilatation of the ear arteries, muscular weakness, somnolence and finally decrease of the respiratory rate and complete cessation of respiration, though the heart continued beating. In insulin shock muscular weakness and somnolence also occurred before convulsions and coma. Here, too, respiratory arrest was observed. With

anoxia 2 types of convulsions might be distinguished. A too rapid diminution of the oxygen percentage precipitated convulsions. A more interesting type of convulsion was observed following the resaturation of the blood with oxygen. Characteristically these convulsions could be suppressed by lowering the oxygen percentage again.

The anatomical changes produced by both types of shock, the anoxic and insulin shock, were also much the same in the central nervous system as well as in the heart, skeletal musculature and liver. In the central nervous system they consisted of from slight to severe degenerations of ganglion cells and reactive glial proliferation. The frequency of shocks, and in the insulin rabbits the individual susceptibility and the severity of the clinical response to the administered insulin were the decisive factors on which depended the severity of the anatomical changes. These changes were especially impressive wherever large pyramidal cells were present, but were by no means confined to them. The cerebral cortex, the Purkinje cells in the cerebellum, the cornu Ammonis, the medulla and the spinal cord were usually definitely involved. No particular nuclei were specifically affected, but more or less numerous ganglion cells in various areas. In a few instances that had shown exceptionally severe clinical responses almost all ganglion cells of circumscribed areas of the cortex or the cornu Ammonis had perished, as described in experiments of Stief and Tokay.[1] An adverse vascular reaction during seizures might be answerable for these localized damages. The more evenly distributed changes were similar to those recently reported by Weil, *et al.*[2] In insulin shock they are best explained by the deprivation of the ganglion cells of carbohydrate which is, according to Himwich, *et al.*,[3] their foremost nutritional material; in anoxic shock by the lack of oxygen[4] which is equally indispensable for the maintenance of their metabolism. Though the same ganglion cells were adversely affected either by insulin or anoxic shock, there was, nevertheless, a difference in the morphological appearance of those cells. The ganglion cells severely affected by insulin shock faded away leaving a "bleached" area in stained preparations;

[1] Stief, A., and Tokay, L., *Z. f. d. ges. Neurol. u. Psych.*, 1932, **139**, 434.

[2] Weil, A. E., Liebert, E., and Heilbrunn, G., *Arch. Neurol. and Psychiat.*, 1938, **39**, 467.

[3] Himwich, H. E., Bowman, K. M., Wortis, J., and Fazekas, J. F., *Science*, 1937, **86**, 271.

[4] Himwich, H. E., Bowman, K. M., Fazekas, J. F., and Orenstein, L. L., PROC. SOC. EXP. BIOL. AND MED., 1937, **37**, 359.

in anoxic shock they were shrunken and darkly stained, as if they were calcified.

Both types of shock produced in the cardiac musculature smaller or larger areas composed of pale staining swollen muscle cells. Only in exceptional cases were a few muscle cells definitely destroyed as was also indicated by the formation of reactive cellular proliferation. Its absence in most of the animals even if they had been frequently treated evidenced the reversibility of the changes described above. The skeletal musculature showed the same changes, but to a more marked degree, rupture of muscle fibres being visible in the iliopsoas muscle and in the musculature of the thigh. Here a definite reactive cellular proliferation was also noted. In the liver hydropic changes of hepatic cells were observed after exposure to insulin or anoxic shocks. Definite focal necroses of the liver were found only in those rabbits which had shown severe responses to insulin or anoxic shock. Fatty infiltration in the parenchymatous organs was not noted following anoxic shock, obviously because of the relatively short duration of a single shock period.

The hydropic changes in the heart, skeletal musculature and liver are usually reversible. In the brain, however, single ganglion cells of various areas are destroyed, and this perhaps is a necessary step in the effective therapy of schizophrenia. The great similarity of the symptomatology and anatomical changes produced either by insulin or anoxic shock might warrant a test as to the curative results obtainable by anoxic shock in schizophrenia.

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The Antigonadotropic Factor. The Quantitative Aspects of the Prolan-Antiprolan Reaction.

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The question as to whether or not the neutralization of prolan by antiprolan is subject to quantitative laws is of interest since it permits of certain conclusions in regard to the mechanism of this reaction. We know that fermentive processes are not subject to quantitative laws. On the other hand, adsorptive processes (*e. g.*, the toxin-antitoxin reaction) are so, subject to the deviation known