ment. It also melted at 80.3°. The 2 in admixture gave no depression of melting point. These melting points disregard the persistence of a "ghost" which, in the grass alcohol lasted for nearly 3°, and in the insect wax alcohol for about 2° above the figures given. The alcohol from grass solidified at a slightly lower temperature than that from insect wax, and it was also slightly less soluble in ether. Both specimens showed only general absorption in the ultraviolet region, that from grass being somewhat the more opaque. In other respects the 2 were indistinguishable. It appears that the ceryl alcohol from *Agrostis* is essentially the same substance, perhaps with a slightly higher mean carbon content, as that from the 2 grasses first investigated.

7029 C

Inhibition of Hypoglycemic Perspiration by Spinal Anesthesia.

HARRY KOSTER AND ARTHUR I. MIRSKY.* (Introduced by W. S. Collens.)

From the Research Laboratories of the Crown Heights Hospital.

When the blood sugar falls to a critical level, many manifestations of sympathetic excitation present themselves in the so called hypoglycemic syndrome. The investigations of Cannon, McIver and Bliss[1] with the denervated heart, of Abe[2] with the denervated iris, of Houssay, Lewis and Molinelli[3] with their cross-circulation preparations, and others,[4] indicate that insulin hypoglycemia is associated with a discharge of epinephrine from the adrenal medulla. Inhibition of the sympathetic adrenal mechanism by any means (sympathectomy, splanchenectomy adrenal medulla ablation, ergotamine poisoning) prolongs the hypoglycemia. This indicates that epinephrine plays an important rôle in the regulation of and in compensation for any abnormal diminution of the blood sugar.

In man, we have no direct evidence that the phenomena observed during hypoglycemia are the result of excitation of the sympathetico-adrenal mechanism; our evidence is only inferred from the animal experimentation. During the course of studies on hypo-

* Richard Morton Koster Fellow.

1 Cannon, McIver and Bliss, *Am. J. Physical*, 1924, **69**, 46.

2 Abe, *Arch. Exp. Path. Pharm.*, 1924, **103**, 73.

3 Houssay, Lewis and Molinelli, *Compt. rend. Soc. Biol.*, 1924, **91**, 1011.

4 MacLeod, Carbohydrate Metabolism and Insulin.

glycemia experimentally produced in man, we observed that one of the earliest and prominent symptoms of a marked fall in the blood sugar, was profuse perspiration. The sweat glands receive nerve fibres which are entirely sympathetic in origin (Langley[5]). It is therefore not surprising that a mechanism apparently which excites the sympathetico-adrenal mechanism should produce profuse perspiration. That this is a "cause and effect" relationship can only be inferred. We could find no direct proof in the literature. To test the hypothesis we attempted to inhibit the sympathetic nervous system, employing spinal anesthesia for the purpose.

Spinal anesthesia is associated with many manifestations of sympathetic inhibition, *viz.*, vasodilatation, intestinal contractions, hypotension, decreased heat rate, etc. The anesthesia *per se,* does not induce perspiration. We therefore employed the following procedure: 0.1 unit of insulin per kilo of body weight was administered to subject and the extent of perspiration noted. The sweating usually began within a half hour. On the following day the patient was given a spinal anesthesia through the intratracheal administration of neocaine. As soon as anesthesia was complete, insulin was administered and the extent of sweating again noted. In some cases, blood samples were taken in oxalate at intervals throughout the experiment for blood sugar determinations. In every instance where the skin anesthesia was below the level of the fifth dorsal segment ("low anesthesia") perspiration occurred only above the area of anesthesia. The anesthetic area always increased in temperature but showed no detectable perspiration even in the presence of profuse large beads of sweat in the non-anesthetized area. In some cases, the hypoglycemia occurred at the same time as the level of skin anesthesia diminshed. In these instances, the area of perspiration gradually increased.

These observations indicated that a paralysis of the sympathetic nervous system, *per se,* may be responsible for the lack of perspiration in the anesthetized areas, and that an increase in circulating epinephrine was not a direct factor in the peripheral manifestations of the hypoglycemic syndrome.

To further elucidate this question, a normal male was given 4 units of insulin intravenously and in 18 minutes he began to perspire. Beads of perspiration became visible over the whole skin surface. On the following day 300 mg. of neocaine was administered intraspinally and a "high" anesthesia was obtained. The anesthetic area extended to just above the fifth dorsal segment. Insulin was

[5] Langley, *J. Physical,* 1891, **21**, 347.

then administered intravenously. The subject was watched carefully for 2 hours and though his blood sugar fell to 43 mg. %, no visible perspiration was noted anywhere. In this case the site of origin of all the sympathetic fibres was affected by the spinal anesthetic. However, the experiment does not permit the presumption that adrenalin secretion is not responsible for the hypoglycemic perspiration. It is probable that the splanchnic nerves to the adrenals were anesthetized and that therefore these glands were denervated functionally. As a consequence, the hypoglycemia could not stimulate adrenalin secretion through these nerves. If the latter is true it is still impossible to explain on this basis the absence of perspiration in the anesthetized areas during a "low spinal anesthesia."

Adrenalin stimulates "nerve-endings", hence any increase of this hormone in the circulating blood should be associated with peripheral manifestations of its action even if the nerve supplying the part is removed. To test this concept we studied 7 patients who had been subjected to lumbar sympathectomies for various diseases. The following case is illustrative of the general results. M. G., a male, aged 28, suffering from early thromboangiitis obliterans was given an intravenous injection of 0.1 units insulin per kilo of body weight. In 23 minutes he began to perspire profusely, both feet showing a similar degree of moisture. He was subsequently subjected to a unilateral lumbar sympathectomy. Two weeks post operation, he was again subjected to the above procedure but no perspiration was noted in either leg. Subsequent studies revealed that 2 or 3 times the preoperative dose of insulin was necessary to produce the hypoglycemic syndrome. The degree of perspiration was similar on both sides in the majority of instances but several cases showed more sweating on the normal side.

It is therefore apparent that insulin hypoglycemia can induce a secretion of the sweat glands even in the complete absence of the sympathetic nerve to the glands.

These studies are reported because they indicate a definite rôle of the sympathetic nervous system in the insulin hypoglycemic syndrome.

Numerous explanations of the above observations may be made. However, two possible mechanisms merit further discussion. The low and high anesthesia studies suggest that hypoglycemia induces a stimulation of the sympathetic nervous system caudal to the spinal cord, probably in the hypothalamus; that the stimulation is transmitted via the splanchnic nerves to the adrenals and that the consequent increase in adrenalin secretion produces the observed

peripheral effects. This hypothesis does not explain all the observations. The other possible mechanism suggested, is a primary, direct stimulation of the adrenals by the low blood sugar, with a consequent increase in circulating adrenalin which in turn stimulates the sympathetic nervous system centers in the midbrain. Anesthesia of the sympathetic nerves prevents the transmission of these impulses to the sweat glands and perspiration does not occur. When the amount of circulating adrenalin is markedly increased then the effects are produced by peripheral stimulation of the nerve endings.

7030 C

Direction of Amoeboid Movement of Leucocytes on a Glass Surface in an Electric Field.

WILLIAM B. WARTMAN AND MORTON MC CUTCHEON.

From the Department of Pathology, University of Pennsylvania School of Medicine.

It is well known that in inflammatory areas the hydrogen ion concentration is frequently increased.[1] According to Abramson,[2, 3] the chemical changes in tissues incidental to injury would give rise to differences in electric potential sufficient to account for emigration of leucocytes. Migration in such an electric field might be due in part to electrophoresis, in part to amoeboid movement of which the direction was determined by the electric field.[4]

The present report is concerned only with the latter manner of progression, amoeboid movement in an electric field, a phenomenon that has been termed, somewhat loosely, galvanotaxis. It seemed of some interest to those of us who are concerned with the general pathology of inflammation and especially with the mechanism of chemotropism to learn whether the amoeboid motion of leucocytes can in fact be directed by an electric current, and, if so, toward which pole the cells move.

Only two papers on galvanotaxis of leucocytes have been found in the literature. Mendelssohn[5] reported that the cells moved

[1] Schade, H., Neukirch, P., and Halpert, A., *Z. Exp. Med.*, 1921, **24**, 11. For brief review of literature see Loos, H. O., *Z. Exp. Med.*, 1931, **75**, 463.

[2] Abramson, H. A., *J. Exp. Med.*, 1927, **46**, 987.

[3] Abramson, H. A., *J. Gen. Physiol.*, 1928, **11**, 743.

[4] Abramson, H. A., in Alexander's Colloid Chemistry, New York, 1928, **2**, 701.

[5] Mendelssohn, M., *Comptes rend. Acad. des Sciences*, 1916, **162**, 52.