THE EFFECT OF INSULIN HYPOGLYCEMIA ON THE CONTRACTION OF THE NICTITATING MEMBRANE OF THE CAT DURING STIMULATION OF PREGANGLIONIC SYMPATHEITIC NERVE FIBERS

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The working of the nervous system, and in particular of its autonomic division, at different levels of the blood sugar is one of great interest and a large number of investigations have been devoted to it (see the summary by Hellhorn [12]. In recent years several investigations on this subject have been carried out in our own laboratory [1-7]. The majority of workers point out that during slight hypoglycemia excitation of the sympathetic division of the nervous system predominates, but as hypoglycemia increases the parasympathetic division becomes more excited. However there is an almost complete absence of a differential analysis of the part played in these reactions by the cells of the various levels of the sympathetic division; in particular little work has been done on the study of the sympathetic ganglia during hypoglycemia.

McIntosh [14] discovered that during perfusion of the superior cervical ganglion of the cat with Ringer's solution not containing glucose, stimulation of the preganglionic nerve prevents the transmission of impulses (as shown by diminution of the contraction of the nictitating membrane) and causes a fall in the acetylcholine content; during perfusion with the same solution containing glucose, according to Brown and Feldberg [11] even prolonged stimulation causes no change in the acetylcholine content of the ganglion tissue.

Kahlson and McIntosh [13] showed that during perfusion of the superior cervical ganglion with a solution not containing glucose, with prolonged stimulation a more rapid suppression of the transmission process appears (tested by the contraction of the nictitating membrane) than when glucose is present in the perfusion fluid; however the reaction of the ganglion to acetylcholine injected from outside is not affected. Perry and Reinert [15] found that there is a falling off in the reaction to injected acetylcholine.

Conditions of perfusion and total deprivation of glucose are far from natural; for this reason it is of interest to investigate the function of the superior cervical ganglion in the intact animal in a state of insulin hypoglycemia.

The aim of this investigation was to trace the changes during insulin hypoglycemia of the threshold of excitation of the preganglionic nerve fibers and also the transmission of submaximal stimuli. The indicator used was the contraction of the nictitating membrane of the cat.

EXPERIMENTAL METHOD

Experiments were carried out on cats which were starved for 24 hours. The first group of experiments was performed under chloralose anesthesia. Ringer's solution, containing 0.8% chloralose, was injected (after induction with ether) into the femoral vein in a dose of 100 mg of anesthetic per kg body weight of the animal. The cervical sympathetic nerve was dissected out, ligated and divided; the peripheral end was laid on
Fig. 1. The effect of insulin hypoglycemia on the threshold of stimulation of the preganglionic cervical sympathetic nerve, as indicated by contraction of the nictitating membrane of the cat. a) During chloralose anesthesia (experiment on January 15, 1957), b) during section of the spinal cord (experiment on April 13, 1957); 1) value of the threshold of stimulation; 2) level of the blood sugar in mg %. Along the axis of the abscissa — time in hours; along the ordinate axis; figures on the left — blood sugar level in mg %, figures on the right — value of the threshold of stimulation in divisions of the potentiometer scale. The first arrow indicates the moment of injection of insulin, the second and third — the moments of injection of glucose.

Immersible electrodes. Gauze drainage strips were placed in the wound, soaked in Ringer's solution. To prevent displacement, the electrodes were fixed with a clamp attached to the stand. As a result of many hours of exposure on the electrodes, despite great care in some of the experiments the nerves were nevertheless damaged; these experiments were not considered. The nictitating membranes were grasped in clips and their contractions recorded on the kymograph by means of Swedish levers. The animal was kept warm throughout the experiment. When preparations were completed an interval of 1-2 hours was allowed, and then measurement of the thresholds of stimulation of the nerve was begun. Stimulation was carried out by means of an induction coil with an alternating current of a frequency of 50 cps. Into the secondary circuit of the coil was introduced a potentiometer with a scale graduated in 100 divisions. In the records and on the graphs the voltage of the stimulating current is expressed not in absolute values but in divisions of the potentiometer scale. When the threshold was being determined the stimulus lasted 3 seconds, with an interval of not less than 3 minutes between stimuli. The submaximal stimulation lasted 15 seconds and was given not more often than once in 15 minutes. In all the experiments blood was taken at definite intervals for estimation of the blood sugar content by the Hagedorn-Jensen method. After the threshold of stimulation of the nerve had remained