activity of liver proteins and decrease the rate of incorporation into the connective tissue and muscles.

Kurt Schreier
Corinna Kazassis

Universitäts-Kinderklinik,
Luisenheilanstalt,
Heidelberg.

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PHYSIOLOGY

Dextran and Insulin in Rats

While testing the effects of insulin on the circulatory reaction of laboratory rats to dextran, we found that many animals died after combined doses of the two agents which were not lethal when administered separately. The validity of this observation was put to experimental test. Male Sprague-Dawley rats weighing at least 150 gm. and fasted 24 hr. were subcutaneously injected with zinc insulin ('Iletin') at two dose-levels, 7·5 or 10 u./kgm. body-weight. An hour later dextran was administered by vein in a total volume of 10 ml./kgm. 0·85 per cent sodium chloride containing either 60 or 600 mgm. dextran ('Expandex'). Control animals received saline without dextran. Fatalities due to these amounts of dextran have not occurred in our work. The results of eight experiments are summarized in Table 1, which shows the incidence of survival after 24 hr. Animals that died exhibited convulsions usually within 3 hr. of insulin administration. Few signs of the dextran reaction characteristic of this species were noticed.

Table 1. Effect of Intravenous Administration of Dextran on Survival of Fasted Rats after Insulin

Insulin u./kgm.	No. survived/total number: Dextran (mgm./kgm.) 0	60	600
7·5	16/20	5/20	13/20
10	13/26	4/26	20/26

The smaller dextran dose caused more stress than the larger at both insulin-levels. Comparison between the insulin doses (2 × 3 table) showed the results were not significantly different, so they were combined. The data were tested in 2 × 2 tables using χ-square with Yates's correction. Although not independent, these tests indicated that the decreased incidence of survival after 60 mgm./kgm. dextran was highly significant when compared with controls or with the number surviving after 600 mgm./kgm. The incidence of survival in the latter groups did not differ significantly from each other.

Aggravation of the dextran reaction in rats measured by œdema and blueing has been reported to follow insulin pretreatment[1,2]. We have found no previous mention, however, of the effect of dextran on this species' reaction to insulin, possibly because the observations were not continued long enough, or the insulin dose was too small. The occurrence of fatal convulsions after insulin doses which without dextran are subconvulsive may be due to indirect effects of dextran. One of these is severe and prolonged vasodepression[3], which would accentuate the already low glucose-level in the central nervous system. This situation could also prevail if vasoconstriction comparable to that seen in the mesoappendix and skin of the rat after intravenous administration of dextran[4] occurs likewise in the small vessels of the brain. Possible direct effects of dextran on resistance to insulin include changes in cellular permeability and blood sugar. Insulin in amounts too small to change the level of blood-sugar significantly has been reported to enhance œdema formation after subcutaneously administered dextran[5]. Our investigations of the manner in which dextran administered by vein affects insulin hypoglycæmia and the incidence of convulsions are continuing.

L. H. Marshall
C. H. Hanna

Laboratory of Physical Biology,
National Institute of Arthritis
and Metabolic Diseases,
National Institutes of Health,
Bethesda 14, Maryland.

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Summative Action of Acetylcholine with Physiological Stimulus on the Generator Potential in the Lateral Eye of the Horseshoe Crab

The action of acetylcholine on the electrical activities of various receptors has been studied by many authors[1]. Most of the investigations of its action were carried out on the responses of receptors in terms of impulses they discharge. In this communication the effect of acetylcholine on the retinal slow potential, which is regarded as a generator potential[2], was studied by means of intracellular micro-electrodes. The electrical activities arising within single ommatidia of the excised lateral eye of the horseshoe crab (*Tachypleus tridentatus*), which was immersed in the physiological saline solution[3], were led to a cathode follower through a micro-electrode filled with 3 *M* potassium chloride.

Having obtained the responses by illumination of a given intensity, duration and interval as controls (*a* in Fig. 1), the solution made by dissolving acetylcholine chloride in physiological saline solution was added by a micropipette to the external solution of a known volume. Then the responses by the same stimuli were successively recorded. The concentration of acetylcholine was calculated afterwards.

As seen in Fig. 1*b*, a remarkable increase in the amplitude of both the initial maximal and the successive less but steady depolarization was induced by the addition of acetylcholine. Moreover, prolongation of the falling phases of the initial maximal depolarization and the phase of repolarization following cut-off of the stimulus also occurred.