Liberation of Potassium by Acetylcholine in the Central Nervous System

SIMILARITIES in the physiological activities of potassium and acetylcholine have been frequently pointed out[1]. As we know that potassium directly affects the functions of the cells, one should consider whether acetylcholine works through a liberation of ionic potassium or not. This might be the case, as in such organs like muscles and the central nervous system a combined form of potassium has been actually detected. Consequently, the liberation of potassium under the influence of acetylcholine was the first thing to look for.

As an object for our inquiry, we took the central nervous system of the Hungarian Esculenta. The whole nervous system was carefully isolated and cut into two homologous longitudinal portions. Both were soaked in isotonic saline (1 c.c.), one being kept for a control, the other being submitted to the action of the drug. After a while (about one hour), nervous tissue and surrounding saline were treated in a mortar in a final concentration of 96 per cent alcohol, and free, uncombined potassium was estimated in the liquid after centrifuging. The following data have been gathered:

(1) Neither eserine alone nor acetylcholine alone affected the content of free potassium.

(2) Acetylcholine, when eserine had been previously supplied for thirty minutes, increased the ratio of free potassium (12–56 per cent, with an average of 23 per cent).

(3) A previous treatment with atropine inhibits the effect of acetylcholine.

Thus it is demonstrated that acetylcholine liberates potassium from some compound. Considering our experimental conditions are purposely unphysiological in the sense that nervous cells turn rapidly inactive in the absence of oxygen, it is highly probable that the effect of acetylcholine should not be considered as the consequence of a functional alteration due to it, but rather to its primary and direct effect.

What adrenaline would do under the same circumstances is now the matter of our investigations.

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Influence of Sodium Chloride on Glucose Absorption from the Intestine of Iodoacetate-poisoned Rats

IODOACETATE poisoning decreases the rate of absorption of glucose and the 'actively absorbed' hexoses from the intestine of rats by about 60 per cent, whereas the absorption of xylose is not affected by this poison[1]. Adrenalectomy has the same effect on sugar absorption[2]. According to Meyerhof[3], iodoacetate acts on glycolysis by suppression of the oxido-reduction between pyruvic acid and triosephosphate. It has been suggested[4] that adrenalectomy influences the glycolytic metabolism in a similar way. Now it has been shown by various American workers that adrenalectomy is followed by a severe disturbance in the excretion and distribution of electrolytes, and that by means of a diet containing no potassium and much sodium it is possible to maintain life with adrenalectomized animals indefinitely. It was therefore tried whether the effect of iodoacetate on sugar absorption also depends on the electrolyte composition.

Female rats of 150–200 gm. were used. Six hours before the absorption experiment they were given subcutaneously 1·5 c.c. 1 per cent NaCl + 0·5 c.c. N/10 Na_2CO_3. Two hours before the absorption this dose was repeated and 1½ hours before the absorption the animals were poisoned with 0·1–0·12 mgm. iodoacetate 1 gm. body weight. The experiments were carried out in urethane narcosis using the same methods as in the above-mentioned previous work[1]. Simultaneously with the sugar, a third dose of sodium chloride (0·5 c.c. 1 per cent subcutaneously) was given. The experiments showed indeed that the inhibiting effect of iodoacetate on sugar absorption is suppressed after sodium chloride administration. The accompanying table shows the experimental data (including experiments in which Na_2CO_3 was replaced by NaCl, and which show the same type of result).

AMOUNT OF GLUCOSE ABSORBED IN 45 MINUTES (PER CENT OF TOTAL AMOUNT INJECTED).

Normal rats	Iodoacetate-poisoned rats		
	Without NaCl	After $NaCl+Na_2CO_3$ administration	After NaCl administration
83·5	26·0	62·0	73·0
49·3	25·0	75·5	63·2
72·6	28·0	73·0	65·5
62·8	31·0	69·0	81·5
61·5	31·0	58·0	
Average: 65·9	28·2	67·5	70·8

The general toxic effect of iodoacetate on rats is counteracted by sodium chloride in a similar way. Female rats of 90–110 gm. die 12–16 hours after injection of 0·05 mgm. iodoacetate/gm. body weight, whereas after administration of sodium chloride in a similar dose as above they survive after a dose of 0·08 mgm./gm.

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June 19.

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Nicotinic Acid, Pentose-nucleotides and Anæmia

IN a recent issue of NATURE[1], Caspersson and Schultz stress the importance of the occurrence of pentose-nucleotides in the nucleus and especially in the cytoplasm of cells in rapid mitotic division. Their observations would appear to have a distinct bearing on the subject of the pathogenesis of pernicious anæmia and other blood abnormalities of kindred mode of origin.

Some time ago[2], the suggestion was made that, in these conditions, the normal formation of erythrocytes by the mitotic division and subsequent ripening of the erythroblast was in abeyance: and that the red blood cells were here formed, heteroplastically and directly, from the corresponding hæmocytoblasts (primitive or definitive as the case might be) after multiplication had occurred by an amitotic process.