qu'en absence de potassium, si on double la teneur en Ca^{++}, l'action potentiatrice de l'adrénaline est aussi doublée. Cette action favorisante du calcium sur l'effet de l'adrénaline est aussi vraie pour l'adrénochrome pur[1], la *l*-nor-adrénaline[2] (fig. 2) et la *N*-isopropylnor-adrénaline (aleudrine). Remarquons que cette dernière, surtout inhibitrice du muscle lisse[3], se comporte sur le muscle strié comme l'adrénaline et la nor-adrénaline. Dans le tyrode sans potassium, l'adrénaline $1{,}10^{-6}$ produit une potentiation de 5 à 10%; le maximum d'action est atteint en moins de cinq minutes, après quoi la potentiation, un moment étale, décline progressivement. Au contraire dans le tyrode sans potassium, avec une dose de calcium double de la normale, l'action de l'adrénaline $1{:}10^{-6}$ se prolonge pendant 15 à 20 minutes et le maximum de potentiation atteint 50%. Après lavage,

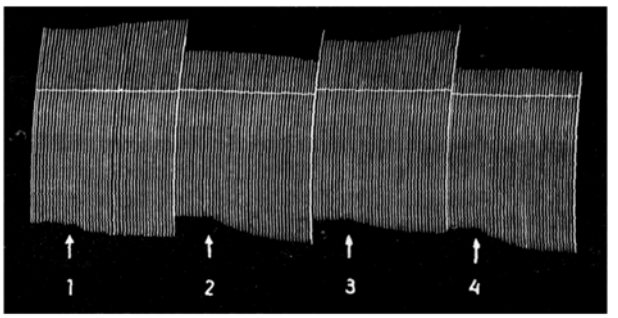

Fig. 2. – Potentiation du twitch maximal d'une préparation isolée diaphragme-nerf phrénique de rat par la nor-adrénaline lévogyre 1.10^{-6}. Contraction vers le bas. En 1 et 3, la nor-adrénaline est ajoutée au bain de tyrode sans K^+ où se contracte le muscle. En 2 et 4, la nor-adrénaline est ajoutée à du tyrode sans K^+, la dose de $CaCl_2$ étant de 0.4 g $^0/_{00}$.

la contraction rejoint lentement sa valeur initiale. On constate les mêmes faits avec la *l*-nor-adrénaline et l'isopropyl-nor-adrénaline. Un excès de calcium a donc un effet favorisant tant sur la durée que sur l'intensité de l'action de quelques amines sympathicomimétiques sur le muscle strié.

En résumé: L'action potentiatrice de l'adrénaline sur le «twitch» maximal du muscle non fatigué de mammifère se retrouve avec l'adrénochrome, la *l*-noradrénaline et l'isopropylnoradrénaline. Cette action est favorisée par un excès d'ions calcium. Ces amines sympathicomimétiques, ou leurs dérivés, agissent sur la fibre musculaire striée elle-même, stimulée directement, après curarisation complète.

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Summary

l-Adrenaline, *l*-nor-adrenaline, isopropyl-nor-adrenaline and adrenochrome potentiate the maximal twitch of the unfatigued mammalian muscle. This phenomenon is increased by an excess of calcium ions in the surrounding fluid. These substances act directly on the muscle fibre, in a completely curarized preparation.

[1] M. GOFFART, C. R. Soc. Biol., (1949, sous presse).

[2] M. L. TAINTER, B. F. TULLAR et F. P. LUDUENA, Science *107*, 39 (1948).

[3] D. F. MARSH, M. H. PELLETIER et C. A. ROSS, J. Pharmacol. Exp. Therapy *92*, 108 (1948).

DISPUTANDA

Diabetes Renalis in Diabete Mellito and the Sugar-proof Kidney[1]

GERGELY denies that *Ag* is a variable value and a function of the blood sugar concentration.

It was not our intention to discuss the theory of *Ag* in all its details. This has been done in another place. We only pointed briefly to the essential facts about *Ag* and discussed whether these could be of use for the solution of a clinical problem. Neither could we go into detail concerning the theory of *Tmg* (SHANNON and FISHER)[2].

GERGELY's two errors are due to a false interpretation of the concepts of *Ag* and *Tmg*. He confuses *Ag* with the "threshold". *Ag* is actually not a fixed, but a *variable* value. Furthermore, GERGELY confuses *Tmg* with the quantity of re-adsorbed glucose (= *Rg*); the maximum of *Rg* is designated as *Tmg*. GERGELY's view that no glycosuria arises as long as *Tmg* is not reached is incorrect. All these questions are discussed extensively in LOVATT-EVANS, *textbook of physiology*[3].

GERGELY's deductions are based on erroneous presuppositions. It is therefore not possible to discuss them in detail. When he – erroneously and arbitrarily – sets the *constant* value *Tmg* instead of the *variable* value *Rg* in the formula for *Ag*, then he must necessarily arrive at the false conclusion that *Ag* is a constant.

From the data of SHANNON and FISHER, GERGELY erroneously concludes that *Ag* is a constant value. That is to be explained as follows:—

(1) GERGELY takes into consideration only the second to fourth period of the experiment. In this *Tmg* is already reached; *Ag* is accordingly already constant.

(2) GERGELY overlooks the first period, in which glycosuria already exists but *Tmg* has not yet been reached. *Ag* then amounts to 127·6 mg%. *Ag* is not even a constant in this experiment as it increases from 127·6 to 240 mg%. The example is not at all well chosen as *Tmg* is already reached in the second period.

The footnotes to GERGELY's remarks are the least comprehensible to us. The author checked the data of H. W. SMITH[4] and found that *Ag* varies from 189–330. Instead of conceding that our values fall within the same range, he unexpectedly calculates an averagé value of 289 mg% for *Ag*. (SMITH's values are not asserted, as he claims of ours, to be based on "technical errors".) GERGELY is then surprised to see that this number lies considerably higher than the average *Ag* value of the literature – 180 mg%. There he again confuses *Ag* with the "threshold".

In summary we can say that GERGELY's remarks are founded on errors. We must reject them completely. On this occasion we call attention to an error which occured in the formula for *Ag* and has only been discovered after publication[5]. The corrected formula reads:

$$Ag = -0{\cdot}00121\ Pg^2 + 1{\cdot}3\ Pg - 24$$

M. FÖLDI, G. SZABÓ, and S. ZSOLDOS

First Medical Clinic, University of Budapest, September 1, 1948.

[1] Reply to J. GERGELY's *Some remarks on the aglucosuric blood sugar concentration*, Exper. *4*, 198 (1948), concerning our paper in Exper. *3*, 329 (1947).

[2] I. A. SHANNON and S. FISHER, Amer. J. Physiol. *122*, 765 (1938).

[3] W. C. LOVATT-EVANS, *Principles of human physiology* (London; Churchill, 1945).

[4] H. W. SMITH, *Lectures on the kidney* (Kansas, 1943).

[5] Exper. *3*, 330 (1947).