

manageable source of pigment than the eggs. Although such protein complexes are widely distributed among Crustacea, they do not seem to be common in molluscan eggs. It is hoped to pursue the embryology of the pigment, which gives an excellent vital-staining effect; material in formol would be welcome, owing to the difficulty of obtaining supplies of *Pala* in Britain.

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### Effect of *l*(+)Glutamic Acid in Hypoglycæmia

OXIDATION of *l*(+)glutamic acid by brain tissue was first demonstrated *in vitro* by Quastel and Wheatley<sup>1</sup>. This work was developed by Krebs<sup>2</sup> with reference to the function of glutamin and glutaminase. Weil-Malherbe<sup>3</sup> suggested that *l*(+)glutamic acid fulfils a specific function in relation to carbohydrate metabolism in cerebral tissue. Latterly, Waelsch and his collaborators<sup>4</sup> observed that the oral administration of *dl*-glutamic acid hydrochloride reduced the number of petit mal attacks in epileptics while it was without effect upon major seizures. Zimmerman *et al.*<sup>5</sup>, confirming these observations, have claimed a favourable influence of the orally administered *l*(+)glutamic acid upon the intelligence of defective children.

This communication is a preliminary report of a series of observations designed to reveal whether *l*(+)glutamic acid could, *in vivo*, replace glucose as a substrate for brain metabolism. The subjects of the experiment were otherwise healthy schizophrenics undergoing Sakl's insulin coma treatment. The preparation of *l*(+)glutamic acid used was a solution prepared by the neutralization of *l*(+)glutamic acid hydrochloride with sodium hydroxide and adjusted to a pH of 7.2. This preparation contained the equivalent of 26 gm./100 ml. of *l*(+)glutamic acid and between 8 and 9 gm./100 ml. of sodium chloride. Sterilization was effected by filtration through a Ford 'Sterimat' grade SB.

Each subject was allowed to enter hypoglycæmic coma in the usual way and was thereafter left undisturbed for a period of fifteen minutes. After a preliminary blood-glucose determination, 80 ml. of solution (20 gm. *l*(+)glutamic acid) was injected intravenously over a period of two minutes. A series of blood-glucose determinations was made while the subject was observed for signs of returning consciousness.

In twelve out of fourteen cases, after a period which varied between five and ten minutes, the subjects became sufficiently conscious to answer questions and to drink of their own accord. The two remaining subjects showed signs of decreasing depth of coma without being fully awake. During the period of awakening the blood glucose rose from 10 mgm. ( $\pm 2$ )/100 ml. to 30 mgm. ( $\pm 4$ )/100 ml.

Using the same subjects, control observations were made after (1) the injection of a similar quantity of a sodium chloride solution containing 9 gm./100 ml. ;

and (2) the administration of 200 gm. of sucrose by nasal tube into the stomach.

In the first series of controls the injection of sodium chloride solution was without effect upon either consciousness or blood-glucose level. In the second series of controls consciousness was restored at a blood-glucose level of 52 ( $\pm 3$ ) mgm./100 ml.

It will be seen that, after the intravenous injection of *l*(+)glutamic acid, consciousness is restored at a considerably lower level of blood glucose than when glucose or sucrose alone is employed. We have previously shown<sup>6</sup> that a blood-glucose level of 50 mgm./100 ml. is necessary before sufficient glucose is available to the cortical cell for the restoration of consciousness, and it is unlikely that sufficient glucose would be so available at the levels of blood glucose obtained after the injection of *l*(+)glutamic acid. It therefore seems probable that the *l*(+)glutamic acid was acting as a substrate for the metabolism of the brain cells.

The problem arises of how to explain the rise in blood glucose after injection of *l*(+)glutamic acid. There would seem to be two possibilities: it may be synthesized in the body from the glutamic acid injected, or it may be remobilized from the reserves of glycogen. This problem is being investigated further.

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<sup>2</sup> Krebs, H. A., *Biochem. J.*, **29**, 1951 (1935).

<sup>3</sup> Weil-Malherbe, H., *Biochem. J.*, **30**, 665 (1936).

<sup>4</sup> Price, J. C., Waelsch, H., and Putman, T. J., *J. Amer. Med. Assoc.*, **122**, 1153 (1943).

<sup>5</sup> Zimmerman, F. T., Burgemeister, B. B., and Putman, T. J., *Arch. Neur. and Psychiat.*, **56**, 489 (1946).

<sup>6</sup> Mayer-Gross, W., and Walker, J. W., *Brit. J. Exp. Path.*, **26**, 81 (1945).

### Use of Filter Paper Pellets in the Spectrochemical Analysis of Solutions

THE Ramage method<sup>1</sup> for the spectrochemical analysis of solutions has been developed by other workers<sup>2</sup>. In this method, filter paper is impregnated with the solution and is introduced into the flame or arc in the form of a roll or strip. Mechanical devices have been designed to ensure the steady combustion of the sample<sup>2</sup>.

We have found that if filter paper (quantitative, 'ashless') is disintegrated with concentrated hydrochloric acid and well washed, the resulting pulp can be easily compressed into small cylindrical pellets which serve as a convenient supporting medium for solutions that are to be analysed by arc methods. The pellets, after forming, are dried in an oven and then impregnated with a measured volume of the solution by touching the pellet with the tip of a micro-pipette. The impregnated pellets are dried again, and 'arced' in the crater of a carbon or graphite arc.

Our original pellets did not burn satisfactorily; but this difficulty was overcome by adding dilute potassium nitrate solution to the original pulp. If potassium is to be determined analytically, another oxidizing agent can be substituted for potassium nitrate.