A Specific Method for the Determination of Glycosides Containing Desoxymethylpentose

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The methods proposed for the chemical estimation of cardiac glycosides depend upon (1) the unsaturated lacton ring, or (2) the steroid skeleton, or (3) the sugar moiety. These tests, however, can scarcely be regarded as specific for cardiac glycosides. The reactions based on the lacton ring or those based on the steroid skeleton codetermine not only steroid derivatives other than cardiac glycosides but also other fairly common compounds which have an active H. The reactions based on the sugar moiety also proved to be unsatisfactory: in some cases they were given even by other non-specific sugars being present in the sample analyzed, in other cases they were not strictly quantitative.

Since the sugar moiety of the cardiac glycosides consists of highly specific desoxysugars, a possibility is afforded of detecting glycosides by utilizing the specificity of the sugar component. The data recorded show that desoxymethylpentoses, *i.e.* the specific sugar compound occurring only in the cardiac glycosides, give a reaction with orcinol which may be used for the quantitative determination of these glycosides. Although a great variety of carbohydrates react with orcinol, the test, in this modified form, seems to be highly specific for desoxymethylpentoses. This permits the detection of minute amounts of cardiac glycosides even in several thousandfold excess of common sugars. All the primary and secondary cardiac glycosides of digitalis give the reaction, as do *k*-strophanthin and oleandrin. Ouabain and the squill glycosides, both containing a methylpentose, do not react, nor do other methylpentoses or desoxypentoses. Aglycones, saponins and other steroid derivatives do not interfere with the reaction. A further advantage of the test is that all the glycosides react in relation to their theoretical sugar content. The method may be used for the quantitative determination of desoxymethylpentose in amounts of 0.5–60 μ g; this amount corresponds to about 1–100 μ g of glycosides. The experimental error of a double determination is ± 1.51 %.

On the Effect of Substituted Barbiturates* on Mitochondrial Respiration

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Substituted barbiturates have long been known to affect the cellular respiratory system. In 1949, Eiler and McEwen¹, working with brain homogenates, showed that the oxidation of pyruvate and fumarate could be blocked by pentobarbital while that of succinate was unaffected. Later investigations, performed on isolated mitochondria, did not lead, however, to a generally valid interpretation of these findings as to the mode of barbiturate action. Brody and Bain² reported a decrease of the phosphorylation/oxidation ratio and attempted to interpret the barbiturate action in terms of an uncoupling effect rather than a respiratory inhibition. This view, however, could not be shared by Johnson and Quastel³. These authors found, as an effect of barbiturates, a decreased capacity of brain mitochondria to activate acetate, but came to the conclusion that this effect might be a secondary one.

From the above mentioned results of Eiler and McEwen it may be envisaged that the effect of barbiturates must be of a rather fundamental character and may therefore also be studied in a system metabolically better defined than brain, *e.g.* liver mitochondria. In experiments with this system we have found that amytal, in a concentration of $1.8 \cdot 10^{-3}$ *M* totally inhibits the oxidation of a series of substrates (Table 1); a clearly marked exception is formed by succinate, the oxidation of which is not at all influenced by amytal. This finding — which is consistent with, and an extension of, the early results by Eiler and McEwen — indicates that the action of barbiturates is concerned with the transfer of hydrogen *via* the pyridine nucleotides.

In some preliminary experiments we were also able to obtain an indication of a correlation between narcotic power and inhibition of respiration. This is shown in Table 2 where

* pentobarbital = 5-ethyl-5-(1-methylbutyl)barbiturate; amytal = 5-ethyl-5-isoamylbarbiturate; hydroxy-amytal = 5-ethyl-5(3-hydroxy-isoamyl)barbiturate.

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