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TUBERCULOSIS AND DIABETES.*

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The classical conception of the frequent association of pulmonary tuberculosis with diabetes is so fixed that phthisis is generally considered to be one of the most frequent, as it is one of the most serious, complications of diabetes. Of recent years, however, there has taken place a certain re-orientation of our ideas in regard to both these diseases, of such nature as to lead to a change in our views on the relations which exist between the two diseases. In practice, the remarkable improvements in our anti-diabetic therapy, thanks to the development of the insulin treatment, in addition to the success obtained with newer methods of treatment in tuberculosis, have tended rather to lessen our conception of the gravity of tuberculous infection in diabetic subjects; the frequency of association of the two diseases seems capable of reduction by an increase in the knowledge of prophylactic measures amongst patients. We are accordingly perhaps on the eve of a revision of the classical notion alluded to in our opening statement. But it is indispensable that we should first of all make a rapid résumé of the more recent developments in our knowledge and treatment of both diabetes and tuberculosis.

Turning first to the subject of the diabetic, the outstanding fact remains that if etiological and anatomo-pathological investigations have thrown but little light on the problem, studies in pathological physiology have succeeded in finding the guiding thread through its many aspects, and have permitted us to formulate a scale of gravity by the division of our diabetic patients into two classes. Henceforth, from this time forward, it will be possible to “draw up a balance sheet” for the diabetic and from it to forecast the evolution of his case. With Marcel Labbé, we now distinguish two types:

1. Diabetes without denutrition. Here we have to deal with a simple alteration in carbohydrate metabolism, due to a disturbance of the sugar-regulating mechanism. Hyperglycaemia and glycosuria result; incidentally, we recollect that the normal proportion of sugar in the blood varies between 1 and 1.5 grammes per litre. In this relatively simple type of diabetes the patient

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preserves a certain degree of tolerance for carbohydrates, and as this index of tolerance varies between individuals, the essential problem in practice comes down to the determination of the personal coefficient.

2. Diabetes with denutrition. Here we have added to a disturbed carbohydrate metabolism a disturbance of the protein metabolism. The carbohydrate tolerance has disappeared: even on a diet from which all carbohydrate has been eliminated, we find no diminution of the glycosuria. Sugar is formed by the destruction of protein material (and doubtless also of fats), and as has been well said, these patients "manufacture sugar from everything that they ingest."

The disturbance of protein metabolism is revealed by the alterations in the urinary analyses, by the appearance of abnormal proteins in the urine, and above all by the disturbed normal acid-base equilibrium and the appearance of acidosis, due to the presence of o-oxybutyric acid, diaetic acid and acetone.

Our better knowledge of the diabetic process would have served us but little if we had remained as poorly armed against it as of old: but dietetic cures in themselves have become a valuable therapeutic aid since the day when, under the guidance of these physio-pathological concepts, it became possible to adjust the diet to each individual case.

In diabetes without denutrition, a reduced carbohydrate regime will cause the disappearance of glycosuria; from this point, by progressive and judicious adjustment of the diet, we can arrive at the estimation of the patient's threshold of carbohydrate tolerance; there remains nothing then but the fixation of the regime of tolerance.

The serious difficulties arise in diabetes with denutrition; these cases call for a reduction of carbohydrates and also of proteins in the diet, although the actual protein wastage requires an increased protein intake; furthermore, the proteins are the main source of the ketone bodies. In such cases, we should rely chiefly on the fats in the diet, which will act as "sparers" of the proteins.

In this fashion, we have come to the meticulous regulation in practice of the appropriate dietary in each individual case of diabetes, and even to its suitable variation as we follow the fluctuations of the carbohydrate and protein metabolism in the same patient.

But if we have thus acquired a definite improvement on older methods, the discovery of insulin has endowed us with a weapon of incalculable value. This pancreatic derivative, isolated at Toronto by Banting and Best in 1922, has changed the entire outlook in severe forms of diabetes. Insulin, we know, acts at the same time on carbohydrate metabolism and on the ketogenic bodies and ketones: on the other hand, it so far diminishes hyperglycaemia as even to suppress it completely (this action, however, is merely temporary, rarely lasting for more than 8 to 10 hours), and, on the other hand, it reduces or abolishes aceto-