THE CARBOHYDRATE METABOLISM IN ABDOMINAL TUBERCULOSIS

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It has long been taught by eminent clinicians, notably the late John Thomson, that cases of abdominal tuberculosis, especially where there is much diarrhoea, or loss of weight, do best on a diet containing a liberal allowance of protein with some restriction of carbohydrate. It was in the hope of throwing some light on this more or less empirical fact that the work to be described was undertaken.

At the outset, the obvious suggestion was that there might be a defective power to absorb carbohydrate from the bowel, just as in some of these cases there may be a relative inability to absorb fat. Such a defect is not so readily demonstrable as a steatorrhoea, the examination of the faeces for carbohydrate residue being an unsatisfactory procedure. One suggestive fact was already available, namely that the oral glucose tolerance curve may be flat in these cases. It was decided to try to ascertain the significance of this curve by carrying out intravenous glucose tolerance tests as well.

The test employed consists in the injection into a vein of the fasting patient of 10 gm. of pure dextrose as a 20 per cent. solution in normal saline, the time taken for the injection being from one to three minutes according to circumstances. Specimens of blood are taken from a prick in the warmed lobe of the ear at intervals of two minutes at first, and later of five or ten minutes, the whole observation extending over an hour.

Results and discussion.

Three cases were examined in this way. In all three, oral curves of a fairly flat type were found (fig. 1), the fasting level in two of the patients being notably low (64 and 66 mgm. per cent. respectively). The rises from the fasting level to the peaks of the curves were 72, 29 and 68 mgm. per cent., figures of which one only is strikingly low, and which might escape comment. The intravenous curves, however, were all of a high type (fig. 2) indicating gross impairment of tolerance.

There was thus presented the seeming anomaly of oral curves tending to be flat with intravenous curves of the opposite form, and there appears to be only one ready explanation of this, namely an absorptive defect. If such a defect exists, then the low oral is obviously explained. Further, according to present beliefs the glucose tolerance of a healthy individual is determined solely by the amount of carbohydrate the diet has contained for the period preceding the test. An absorptive defect should produce the

FIG. 1.—Flat blood sugar curves obtained after giving 30 gm. of glucose orally to each of three cases of abdominal glandular tuberculosis.

FIG. 2.—High intravenous blood sugar curves obtained from the same three cases as the oral curves in fig. 1. A small section of a normal curve is included for comparison.

same effect as carbohydrate deprivation in this regard, and hence the intravenous curve should be high. The anomaly is explained on this basis.

A further point of rather a confusing nature should be cleared up at this stage. As mentioned earlier, the rise from fasting level to peak following an oral test dose of sugar was not strikingly small in two of the cases examined, and this might be thought at once to rule out any absorptive defect. Actually, however, the curves obtained in a case of moderate absorptive deficiency must reflect the resultant of two opposing factors. On the one hand, the fact that only a small part of the sugar given actually reaches the blood might be expected to give a low curve. On the other, if the patient is otherwise healthy the deprivation of carbohydrate must be expected to lead to impaired tolerance and a high curve, there being an abnormal rise in blood sugar level in response to each moiety of sugar actually reaching the blood. It is the interaction, it is suggested, of these two opposing factors which may lead to a curve in some respects approaching normal.

It may be raised as a criticism that diarrhoea alone might amount to a sufficient absorptive defect. Opportunity arose to re-examine one of these cases during a period of severe diarrhoea and again during a remission and the curves show little, if any, significant difference.

All of these cases were of the glandular type with easily palpable abdominal glandular masses, and in two of the cases calcification of some of the glands was revealed by x-ray. Opportunity recently offered to study similarly an ascitic case, in which no gross glandular involvement was discovered, and there had been no diarrhoea, though wasting was gross. The result here was an interesting contrast, the curves (fig. 3 and 4) being in all regards comparable to those of a normal patient on a very low carbohydrate diet, i.e. both oral and intravenous curves were high, and it is inferred that there was little or no absorptive defect. What then, was the cause of the impaired tolerance? In this respect it is dangerous ground to suggest that the causal agent was the tuberculous toxæmia. Evidence that this was present included a fairly high temperature and rapid pulse rate. It may be mentioned that the same tests have been carried out on two other tuberculous patients—one suffering from coxalgia, the other from a large retroperitoneal mass of tuberculous glands. The former was clinically ill and toxic-looking and showed similar curves of much milder degree. The latter was clinically well and gave a perfectly normal intravenous curve.

Treatment.

The bearing of these observations upon treatment was next considered. According to current ideas, degrees of glucose tolerance or intolerance are caused by the presence in circulation of more or less 'insulin-kinase,' a third factor in the insulin-glucose reaction which is probably prepared for the

FIG. 3.—Oral curves from an ascitic patient. The solid line represents the original condition; the dashed line the effect of giving 2 c.c. of Campolon intravenously prior to the test; the dotted line the condition after withholding liver treatment for eleven days; the crosshatched line the effect of giving one-half ounce of liquid extract of liver by mouth twice a day for eleven days.

FIG. 4.—Intravenous curves obtained from the same patient as the curves in fig. 3 under the same conditions. The dotted line represents the original condition; the dashed line the effect of Campolon; the cross-hatched one the reversion on withholding liver treatment; the solid one the effect of liver treatment by mouth.

greater part in the liver. Previous work had led to the belief that this substance was present in 'Campolon.' Accordingly, the worst of the three glandular cases was given 2 c.c. of 'Campolon' intravenously six minutes before a further intravenous tolerance test. The resulting curve (fig. 5) was strikingly altered towards normal, and in fact was for practical purposes normal. The patient was then left untreated for four days and again tested without 'Campolon.' The curve had reverted to its previous level. He was then given one ounce of liquid extract of liver orally daily, and curves

FIG. 5.—Curves obtained from a severe glandular case. The upper one represents the state before treatment with Campolon, and again after cessation of liver treatment for four days; the lower one was obtained after intravenous Campolon and again after two weeks and after twelve weeks on liver extract by mouth.

obtained in two weeks and in twelve weeks, practically overlie that obtained with 'Campolon.' An almost identical series of tests was carried out on the ascitic case, and showed a corresponding improvement of tolerance, both intravenously and orally (fig. 3 and 4).

As to clinical results, the treatment proved most striking. Both patients had the advantage of rest in bed and of diets which were adequate even if the carbohydrate were disregarded. The patient with glandular enlargement was admitted to hospital with the gravest possible prognosis from a convalescent home of which he was no longer a proper inmate. He

had been bedridden for months up to the commencement of treatment and was shockingly wasted. In three months' treatment he gained $11\frac{1}{2}$ lbs. in weight and improved to the point of being up and actively running about. The patient with ascites who was also given an early fatal prognosis on admission, has lost all her ascites, gained eleven pounds notwithstanding, and, while still in bed, feels and looks perfectly well. In neither patient was there any blood disorder more grave than a mild anaemia of secondary type which improved slightly under treatment. There is no evidence that the absorptive defect in the glandular type of case is materially improved by liver treatment.

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