But even if we admit that orations are but empty words, there exists a far more vital evidence that the torch of inspired enthusiasm has been faithfully handed down from his day until now.

First, there was that dear old man Clift, who like a watch-dog guarded his master's treasures after his death, when it seemed that nobody wanted them. For seven years he lived on seven shillings a week, whilst he kept his trust, and then later became the first formal custodian of the Museum. The watch-dog growled and showed his teeth, but, alas! could do no more when his master's precious papers, many of which Clift had himself helped to write, were carted away and secretly destroyed by Home. Clift's daughter married Richard Owen, who succeeded him in the guardianship of the Hunter Museum and became the greatest palaeontologist and comparative anatomist of his day. He was later transferred to the Natural History Museum at South Kensington. Owen was followed by Paget, a most notable torchbearer, whose ceaseless labours added so much to surgical pathology, and whose silver-tongued eloquence is remembered by many now living. Then came the greatest of Hunter's followers, Lister, whose name is held in honour all the world over as the one whose work has saved more lives and suffering than have been sacrificed by war. Lister, it is true, never held high office in this College, though he was a member of Council for eight years. His gentle soul did not love fighting with his detractors, and he preferred to pursue his quiet research in the laboratory and at the bedside. And lastly, we are fortunate in having to-day, in the person of our beloved Curator, Sir Arthur Keith, one on whom the mantle of John Hunter seems perfectly to rest, the depth of whose learning and the eloquence of whose tongue fit him so well to be the guardian of Hunter's heritage and the exponent of Hunter's inspiration.

Hunter may truly be said to have raised the idea of the scientific pursuit of truth to the level of that compelling influence in men's lives which is called religion. For he had a vivid faith in his quest, an unquenchable enthusiasm for his ideal; he left an inspiration for all who come after him, and a real hero for us to worship.

ADVANTAGES TO THE DIABETIC OF A DIET RICH IN CARBOHYDRATES.*

BY

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WHEN Banting revolutionized the treatment of diabetes by the introduction of insulin he provided a means whereby the diabetic can metabolize carbohydrate. It was unfortunate that this discovery should have come at a time when Allen had revived Guelpa's method of treating diabetes by starvation. Because the "starvation" method unquestionably became grafted on to the insulin method of treatment, with the result that, in addition to the necessary evil of having to endure daily hypodermic injections, the diabetic is often subject to deliberate and, in my opinion, unnecessary degrees of undernutrition. The high fat dietary is another survival from pre-insulin days.

The practice of feeding diabetics with an excess of protein was more readily dropped when insulin was introduced, although many advertised diabetic foods still claim as a merit their high protein content. I cannot conceive that in these days there is any advantage to the patient in offering as a substitute for carbohydrate an excess of fat or of protein. For, to speak plainly, if a diabetic deprived of carbohydrate does not convert the fat or protein of his food into glucose or glycogen he dies. There are a few people so constituted that they can derive from fats and protein an adequate glycogen supply—the Eskimos as a race seem capable of this physiological *tour de force*. In our race or our climate the healthiest of us mostly fail miserably if we try to accomplish it. The diabetic, with

or without insulin, is equally liable to fail; it is almost a miracle when he succeeds.

The question I propose to consider is whether a diabetic need nowadays ever be asked to attempt either the task of deriving his glucose exclusively from fats and proteins or the alternative task of living cheerfully and working usefully whilst balancing on a sort of knife-edge of starvation.

The importance of keeping the blood sugar low if a diseased pancreas is to have a chance of recovery is universally admitted, but the prospects of recovery are decreased if the low blood sugar is accompanied by a general starvation. A starved pancreas will not regenerate, even though the blood sugar is low; new tissues are not built up by such means.

Before, however, the regeneration of the pancreas is considered, there may come the far more pressing question of keeping the diabetic patient alive. The first fear is that diabetic coma may supervene. Insulin treatment has reduced the mortality from coma to such an extent that one can say "a death from diabetic coma is an unnecessary death." Nevertheless diabetic coma is not an unreal danger even to-day, and it is only by constant supervision on the part of the doctor and intelligent co-operation on the patient's part that this danger is averted.

We know that the chief factor in the production of diabetic coma is incomplete oxidation of fats. These fats may be either exogenous, the fats of food, or endogenous, the fats of the body. If insufficient carbohydrate is ingested, the body endeavours to derive its caloric wants from the fats; in any case a certain amount of carbohydrate is required to secure complete oxidation of the fats. If the oxidation is incomplete ketosis ensues, and finally such a degree of ketosis is reached as produces coma and death. Thus if a patient with diabetes, in whose urine acetone bodies are present, comes under observation the immediate aim of treatment is not to regenerate the pancreas, it is to prevent coma. The treatment of coma when established is admitted to be the administration of glucose rendered usable by insulin. No one urges that the least particle of fat is needed when the patient is comatose.

When treating a diabetic I am never satisfied so long as any trace of ketone bodies can be detected in the urine. I know that Rothera's sodium nitro-prusside test is unduly delicate, but it seems to me that any patient who has had sufficient ketosis to give a positive ferric chloride (Gerhardt's) response, may as well be cleared completely, even to the point where Rothera's test becomes negative. In like manner, when Rothera's test becomes positive in a diabetic where previously it was negative, it is better to push the patient back on the safe side and render the urine completely negative, rather than let him continue on a path, whose width we cannot presume upon, which divides a positive Rothera's reaction from a negative Gerhardt's.

In a patient showing even slight ketosis there seems no sound reason for administering fats at all. The first object is to eliminate the ketones, and we only know one practical way of doing this—namely, the administration of carbohydrate. When the subject of ketosis is not a diabetic we give glucose, either by the mouth or intravenously, but if diabetes is present then insulin must be added to enable the ketones to be oxidized with the carbohydrate. If the diabetic patient should chance to have a high blood sugar it may be that insulin alone will enable the blood sugar to be utilized, but there is no way of determining whether the blood sugar all by itself is sufficient to counteract the ketosis when insulin has been given.

One may meet with a patient in diabetic coma with hypoglycaemia, because the insulin has enabled the blood sugar to be exhausted and yet was insufficient in amount to burn up all the ketones. Obviously this can only be avoided by giving carbohydrates freely with the insulin and ignoring glycosuria. As the majority of patients threatened with coma are under-nourished, we need not fear that their weight will increase immediately. Make them safe first, then consider their nutrition.

The patient with ketosis, however slight, does not stand in immediate need of fat. On the contrary, a diet rich in fat but poor in carbohydrates produces fat deposit rather than glycogen in the liver, and where fat has got in there

* Presidential address to the Bath and Bristol Branch of the British Medical Association, 1929

is apparently no room for glycogen. Now a respectable glycogen deposit in the liver provides to the diabetic who is having insulin a definite safeguard against hypoglycaemia.

A diet poor in fat and rich in carbohydrates is the only way to ensure glycogen storage. So I advocate the withholding of fats for a time in patients with any degree of ketosis, and being generous with the carbohydrate. I give the patient an adequate maintenance diet, replacing the fats as far as possible with their equivalent of carbohydrate—that is, 1 ounce of fat replaced by $2\frac{1}{4}$ of carbohydrate. I recently had a small boy sent into Bristol Royal Infirmary verging on coma. I ordered my house-physician to give him all the sugar he could eat, and sufficient insulin to restore him to consciousness without bothering about the glycosuria. Also a baby of 18 months was treated with insulin whilst kept on a milk diet, with no added fat.

Diabetic children can rarely do without a generous ration of carbohydrate. Even the normal child is easily upset by a surplus of fat over carbohydrate, which accounts for a certain number of cases of cyclical vomiting, acetonæmia, or acidosis in children. No child should be compelled to eat more fat than he has the taste for. Avitaminosis is not a real danger. Vienna experience showed how little fat added to the diet would avert xerosis of cornea, rickets, etc.

There is another class of diabetic in whom a fat-free or fat-poor diet is an advantage—that is, the patient who requires a surgical operation or an anaesthetic. No surgeon or anaesthetist likes to embark on an operation upon a patient who has not a reasonable store of glycogen. Funk said: "Let us not forget that sugar (glycogen) is the food, and the only food, upon which muscle can live. Especially to be remembered as regards the heart." Fats are no help in the preparation of any patient for operation, less still do they help the diabetic.

Diabetic atheroma is another condition in which a good carbohydrate supply is definitely beneficial and a fat-rich diet is harmful. Joslin remarks that every dose of insulin defers arterio-sclerosis and postpones old age; for it has been observed that the diabetic who develops arterio-sclerosis at an abnormally early age is the severe diabetic kept alive upon a low carbohydrate, high fat diet.

There is another point to be considered in reference to arterio-sclerosis: insulin has been administered in order to relieve arterial spasm, and has proved beneficial in some cases of Raynaud's disease and gangrene; but it has been observed that insulin-hypoglycaemia may actually precipitate an attack of angina pectoris in a patient with a diseased heart. The sclerotic heart, as van den Bergh remarked, may even demand a high blood sugar level.

Pregnancy in the diabetic woman is another state which surely calls for great caution in the administration of protein and fat; I know no solid objection to allowing her all the carbohydrate she asks for. There is a grave doubt whether a high cholesterol content in the blood, such as excessive fats produce, may not damage the kidneys as well as prevent glycogen storage in the liver—a combination of evils which is especially dangerous in pregnancy.

Sansum has been advocating for some time a more generous use of carbohydrate, and Joslin's only criticism is that it may lead to undue gain of weight. Nearly every writer on diabetes advises that the diabetic shall be kept at a weight 10 per cent. below the average for his age and build. I believe the best weight is that at which a person feels fittest and is most active in mind and body.

I always feel sorry for the diabetic who has to take his necessary calories in the more condensed form, say, of butter and cream, and stay the pangs of hunger with cellulose and similar indigestible roughage. Which of us, if given our choice, would not prefer 1 oz. of bread to $\frac{1}{2}$ lb. of cabbage *plus* 5 oz. of brussels sprouts and 5 oz. of lettuce? Our patients are advised to eat Ryvita; four pieces of Ryvita contain more carbohydrates than 1 oz. of white bread. Porridge is not everybody's meat, yet a plate of porridge made with $\frac{3}{4}$ oz. raw oatmeal contains the same carbohydrates (15) and protein (3) as 1 oz. of white bread, and 3 grams of fat in addition.

Most children like milk, and it is not so fearfully dangerous; 1 pint contains 30 grams of carbohydrate, 20 of protein, 20 of fat, and in a palatable digestible form.

Three ounces of potatoes contain only 20 grams of carbohydrate.

Bear in mind that for every ounce of fat that is omitted $2\frac{1}{4}$ oz. of carbohydrate may be substituted. So that if we are content with $\frac{1}{4}$ oz. of butter instead of the 1 oz. allowed in the Bristol Royal Infirmary basal metabolic diet of 1,679 calories, our meal may contain at least $\frac{1}{2}$ oz. more white bread. My experience is that if fats are limited and sugar is omitted few patients will voluntarily exceed a 3,000 calorie diet which contains ordinary carbohydrate and meat. They must, of course, be careful not to take sugar or jam in any form, raw or cooked. That means giving up marmalade for breakfast, sweets for dinner, and jam for tea.

The advantages that may be claimed for allowing diabetic patients a diet rich in carbohydrates appear to be:

Contentment and satisfaction are secured.

The regimen is more readily observed.

The danger of ketosis is removed.

Arterio-sclerosis is delayed.

Most important of all, a fat-poor diet increases carbohydrate tolerance and the prospects of pancreatic regeneration.

MISSED DIAGNOSES.*

BY

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KNOWLEDGE of pathological processes and their manifestations grows steadily, and with it has developed a finer discrimination of disease; but we must confess that at times diagnosis eludes us, not always because we are lacking in skill or in experience, but because our knowledge is still insufficient. Missed diagnoses are not to be regarded as errors. The medical museum and the post-mortem room offer proof that in many cases a missed diagnosis, often unavoidable, has occurred.

Our nomenclature is sometimes at fault, suggesting an artificial classification of disease into separate entities; thus, when an anatomical diagnostic term is used it tends to narrow our field of view, and we may fail to realize that disease may be a widespread process affecting the whole organism. Occasionally a diagnostic term may serve as an expedient for concealing ignorance. When we speak of a deficiency of hormones are we in fact more lucid than were our predecessors, when they said a patient was suffering from the spleen?

Disease Without Symptoms.

Variations from the normal structure may occur without associated disturbances of function. The presence of unilateral kidney may remain unsuspected during the lifetime of the patient and be revealed for the first time in the post-mortem room; before the introduction of modern methods of genito-urinary investigation cases occurred in which the unilateral kidney was excised in the operating theatre with fatal results. The patient with a polycystic kidney may not present symptoms till he has reached middle age, and the analogous condition of congenital cystic liver may remain unsuspected till its increasing size creates a diagnostic problem.

A woman, aged 43, had an enormous liver, with cystic swellings that had been slowly developing for many years; tests for suspected echinococcus disease gave negative results; laparotomy revealed simple cystic disease of the liver.

Congenital morbus cordis may remain unsuspected, and I have known an absent interventricular septum to be found accidentally at a necropsy on an adult woman. Advanced cancer of the stomach may occur without the association of dyspeptic symptoms, and surgeons have demonstrated that patients whose stomachs have been removed may afterwards suffer little or no inconvenience.

In acquired disease compensatory changes, or encroachment on the physiological reserve, may mask the condition

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