

THE
Journal
OF
Nervous and Mental Disease

Original Articles

THE DIET IN EPILEPSY.

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It is undoubtedly a well-known fact that diet has an important influence on the manifestations of epilepsy, and medical literature contains numerous statements concerning the effect of one or the other kind of food upon the frequency and severity of the seizures. Most authorities agree that meat in large quantities is harmful to epileptics. Consequently, dietaries prescribed for such patients include, as a rule, a relatively scant allowance of meat, and that given usually in one of the daily meals only.

The effect of varying, not the kind of food but its proximate composition, has not been subjected to systematic study directly. Some interesting data may, however, be found in a well-known communication by Merson.¹ To test a hypothesis which had been advanced by Hughlings Jackson, according to which a palliative for epilepsy might be found in the substitution of phosphorus for some of the nitrogen in the chemical make-up of cortical tissue, Merson kept a group of twelve epileptic patients for four weeks on a "farinaceous diet," poor in nitrogenous principles, but containing a normal amount of fats and a large amount of carbohydrates (proteids, 74 grams; fats, 92 grams; carbohydrates, 312 grams, daily); these patients were receiving, in addition, ten drops of phosphorated oil daily. A similar group of patients was kept on a "nitrogenous diet," containing an excess of nitrogenous material, but insufficient in fats and carbohydrates (proteids, 202 grams; fats, 45 grams; carbohydrates, 215 grams, daily). The effect of the farinaceous diet was an unmistakable

¹J. Merson. The West Riding Lunatic Asylum Medical Report, 1875.

decrease, that of the nitrogenous diet—a marked increase, in the frequency of convulsions. The same effect was obtained when, in a second experiment, the first group of patients received the nitrogenous, the second group the farinaceous diet.

Ample experience since has shown that phosphorus is entirely worthless in the treatment of epilepsy. Merson's results, therefore indicate that either the reduction of nitrogenous matter or the reduction of meat has a beneficial influence in epilepsy.

Turning from the empirical results of dietetic investigations to the recent chemical-pathological studies of epilepsy, there is good ground for the assumption that the various manifestations of this disease are probably dependent upon a disorder of nitrogenous metabolism, and upon nothing else. This disorder is supposed to consist in the formation of a poisonous substance, which is either not formed at all in the normal state or exists only in harmless quantities, and which in epilepsy accumulates in the body more or less rapidly, causes vertigo, convulsions and psychic equivalents, and during the seizures is converted into urea—the final product of proteid metabolism. In other words, in epileptics, at a certain stage in the process of conversion of proteid material into urea there is a hitch, and abnormal, often violent, activity of the nervous and muscular tissues becomes necessary for the completion of this process of conversion.

That a substance capable of producing convulsions and other phenomena of epilepsy actually exists in the blood of patients in the status epilepticus has been proved by the researches of Krainsky who, by injecting blood obtained from patients in that condition into guinea pigs, produced convulsions and paraplegia. The same observer also found in the blood of patients in status epilepticus large quantities of ammonium carbamate; and he has shown that by injecting this substance into rabbits epileptic seizures can be produced.²

Ammonium carbamate is said to be present in the blood normally in very small quantities and is supposed to be a precursor of urea. Its conversion into urea occurs chiefly in the liver, and Krainsky finds further corroboration of his theory, according to which the phenomena of epilepsy are due to the periodical accumulation of ammonium carbamate in the circulation, in the experiments of Hahn, Massen, Nencki and Pawlow. These

² Binswanger, "Die Epilepsie," p. 238, *et seq.*

observers have shown that when in animals an Eck's fistula is established (an artificial communication between the portal vein and the inferior vena cava), the portal circulation is thrown out of action and the ammonium salts formed in the course of proteid catabolism, being no longer completely converted into urea, accumulate in the circulation, presumably in the form of the carbamate, and give rise to convulsions, ataxia, somnolence and coma before the animal dies.³

In the light of these experimental facts, it seemed to me safe to interpret Merson's observations in the sense that it is not the kind of food, not meat or farinaceous material as such, but the absolute quantity of proteid matter, irrespective of its origin, that constitutes the dietetic factor of influence on the frequency and severity of epileptic manifestations. In view, however, of the great practical importance of the question, I decided to subject this interpretation to the test of direct experiment.

Eleven patients suffering from "genuine" epilepsy of old standing, but presenting no complications, were kept on five different diets consecutively, the kind and content of the food being systematically varied. For the purpose of eliminating the influence of exercise on metabolism, the patients were kept in bed during the entire duration of the experiments. For a similar reason they received absolutely no medication. A careful record was kept of the weight and mental condition of the patients, as well as of the number of convulsions. Since the frequency of the seizures in any case of epilepsy is apt to vary without any apparent cause, I thought it wise to compare the total weekly average number of convulsions under each diet, and not, for instance, the number for each patient for each week. Following are the results:

EXPERIMENT I. REGULAR DIETARY OF THE HOSPITAL.

Breakfast: Oatmeal, wheat flakes, or farina with syrup or milk; bread, butter, coffee with milk and sugar.

Dinner: Beef, mutton, veal or pork; potatoes, vegetables, bread.

Supper: Pudding, cake, baked beans or macaroni; fruit, bread, butter, tea with milk and sugar.

³ Hahn, Massen, Nencki and Pawlow. "Die Eck'sche Fistel zwischen der unteren Hohlvene und der Pfortader und ihre Folgen für den Organismus," *Archiv für experimentelle Pathologie und Pharmakologie*, Vol. 32, p. 18.

This dietary is somewhat variable, and the quantity of food received by each patient is not accurately measured; there is, however, no important variation in the proportions of the different proximate principles. The amount of proteids is small, that of the carbohydrates large, and that of the fats moderate.

This experiment was carried on for ninety-one days (from Dec. 1, 1903, to Feb. 29, 1904). During that time the patients' mental condition presented no unusual feature; their weights remained nearly stationary, and the total average of convulsions per week was 14.3.

EXPERIMENT II. VEGETABLE DIETARY.

Breakfast: Oatmeal, wheat flakes, or hominy with syrup; bread, butter, coffee with sugar, but without milk.

Dinner: Beans or peas, rice, sago or tapioca, potatoes, vegetables, bread, butter.

Supper: rice, cornmeal or bread pudding, fruit, bread, butter, syrup, tea with sugar, but without milk.

This dietary differs from the regular hospital dietary only in the substitution of vegetable proteids for those of animal origin, the quantities of the proximate principles being practically the same in both. This experiment was carried on for forty-six days (from May 1, 1904, to July 15, 1904). During that time no change from the usual mental condition of the patients was observed. Seven patients gained in weight from three to eight pounds; one patient lost three pounds, and the weight of the remaining three patients remained constant. The total average of convulsions per week was 14.3.

EXPERIMENT III. DIETARY WITH INSUFFICIENT QUANTITY OF PROTEIDS.

Breakfast: Rice, 3 oz. (weighed before cooking); milk, 8 oz.; sugar, 1 oz.; coffee.

Dinner: Rice, 3 oz.; milk, 8 oz.; tea.

Daily quantities of proximate principles received by each patient: Proteids, 62 grams; carbohydrates, 336 grams; fats, 41 grams.

This experiment was carried on for forty-seven days (from July 16, 1904, to Aug. 31, 1904). During that time the patients were restless and irritable; their weights remained stationary; the total average of convulsions per week was 17.6.

EXPERIMENT IV. DIETARY WITH EXCESSIVE AMOUNT OF PROTEIDS.

At each meal, 2 eggs; milk, 24 oz.; bread, 3 oz.; sugar, 1 oz.; tea or coffee.

Daily quantities of proximate principles received by each pa-

tient: Proteids, 148 grams; carbohydrates, 305 grams; fats, 116 grams.

This experiment was carried on for thirty days (from Sept. 1, 1904, to Sept. 30, 1904). All the patients gained in weight from four to twelve pounds. Mentally they were restless and irritable. The total average of convulsions per week was 18.2.

During October, 1904, the regular diet was resumed. The total weekly average of convulsions then fell to 14.

EXPERIMENT V. DIET WITH LARGE EXCESS OF PROTEIDS AND VERY DEFICIENT IN CARBOHYDRATES (THE DIABETIC DIETARY OF THE HOSPITAL).

Breakfast: Eggs, bread, butter, coffee.

Dinner: Meat, cabbage, lettuce or celery, bread, butter, coffee.

Supper: Eggs, stew or cheese, bread, butter, tea.

No milk was used. Saccharin was used with the tea and coffee. Where bread is called for on the dietary bran bread or gluten bread was used.

This experiment was carried on for twenty-one days (from Feb. 13, 1905, to March 5, 1905). Most of the patients' weights were diminished from two to five pounds. Mentally, they became restless, irritable, noisy, destructive to their clothing, and assaulting. The total weekly average of convulsions was 23. The increase in the number of convulsions persisted for about two weeks after the regular diet had been resumed.

To sum up the results: 1. Experiments I and II prove that the effect of a mixed diet in epilepsy differs in no way from that of a vegetable diet containing the same quantities of proximate principles. Consequently any lingering belief that animal food has any effect as such is to be definitely discarded.⁴ 2. Experiments III and IV prove that the quantity of proteids in the diet has a decided influence on the manifestations of epilepsy; if it is either above or below the indispensable minimum the severity of the disease is increased. 3. Experiment V shows that if the diet of an epileptic is made to contain a large excess of proteid and practically no carbohydrates, so that the organism is compelled to use proteid material in place of carbohydrate material, the number of convulsions increases enormously and there is a general aggravation in the physical and mental condition of the patient.

It seems, then, that *just as the organism of the diabetic is unable to properly utilize carbohydrates, so the organism of the epileptic cannot take care of proteid material as it is taken care*

⁴ In this connection it may be interesting to note the fact that epilepsy is common in the herbivora.

of by the normal organism. The epileptic is, however, the worse off of the two, for while the diabetic can without serious harm leave out carbohydrates from his diet, substituting for them fats and proteids, the epileptic can only partly substitute proteids by the other proximate principles, being compelled to take a certain minimum amount to sustain life.

Possibly the analogy between diabetes and epilepsy can be carried still further. The cases upon which my experiments were made are of old standing, with the "epileptic habit" deeply rooted in the organism, with mental complications, and possibly with secondary organic changes in the nervous system; they may be compared to "severe" cases of diabetes in which even the most rigid diabetic restrictions fail to cause a total disappearance of the manifestations of the disease. On the other hand, there are mild cases of epilepsy, which are not of long duration, in which the seizures are infrequent and which are not complicated with mental disorders; these may be compared to "mild" cases of diabetes in which it is unnecessary to exclude completely carbohydrate substances from the diet, a mere reduction in this quantity being sufficient to remove all the evidences of the disease. These mild cases of epilepsy may be reasonably expected to recover completely under appropriate dietetic treatment in accordance with the principle laid down above.

The therapeutic indication is clear. Carbohydrates and fats are to a certain extent capable of replacing the proteids in the diet.⁵ The epileptic patient, then, should receive the largest amount of carbohydrates and fats that he can assimilate without inconvenience, and the smallest amount of proteids which is compatible with the preservation of the nitrogenous equilibrium; that is to say the amount of nitrogen ingested with the food must not be allowed to fall below the amount excreted, for then the thing is overdone, a condition of proteid starvation is established, the general health of the patient suffers, and his disease becomes aggravated.

⁵ Herter, "Lectures on Chemical Pathology," p. 150.