

EPILEPSY, ITS DIET AND DRUG RELATIONS

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Since the problem of the etiology of epilepsy remains so obscure that we cannot even estimate the rôle which endogenous and exogenous factors play, there is a certain justification in following up mere conjectures in order to prove or disprove their merits. The fact that fasting produces a cessation or at least a diminution in the number of seizures in many cases was brought out through the conjecture of an osteopath in 1921. The ketogenic, or acid-producing, diet was worked out afterward and was calculated to cause changes in the body of the epileptic similar to those produced during starvation.

During fasting there is a distinct amelioration of symptoms in the epileptic, with a reduction in the alkali reserve of the body, a decrease in blood sugar, an increase in uric acid and in the hydrogen ion concentration of the blood, as well as an increase in excretion of ammonia and a titratable acidity of the urine. Through the administration of the ketogenic diet similar results are produced.

The ketogenic diet consists of a normal amount of proteins, a very much reduced quantity of sugar and starches, and an excess of fats in the form of rich cream, butter, mayonnaise, bacon, oil, etc. The fat is only incompletely broken up in the absence of carbohydrates, and intermediary products of digestion are formed belonging to the acetone groups, which are highly acid in reaction. It is thought that this production of acid during digestion is the essential thing in obtaining results, although there are probably some additional factors still unknown.

The ketogenic diet must be estimated and adjusted for each individual case, every patient having his own peculiar metabolism. It is interesting to note how much difference there is in the requirements for acid production in the various patients. While one patient is markedly benefitted by a diet producing only a minor degree of acidity, another patient needs a very high degree of acid production. This peculiarity is not constant in the same patient

but has shown a good deal of variation at certain times. The same is true in the variability of acid required in different patients at various times in order to cause a change in the hydrogen ion concentration of the body fluids. It might be stated here that this difference in acid requirement in various individuals is one reason for the conflicting results obtained in this method of treatment.

The administration of acids as well as acid-producing drugs has proved beneficial, but their action is much more transitory than the ketogenic diet and is, therefore, of value largely as a supplement to the diet. It cannot be denied that in fasting or in the administration of the ketogenic diet we have found for the first time a method to control epileptic seizures without the use of depressing drugs. Whether the result is obtained because of the dehydration of the body or because of the anaesthetic action produced by the acetone bodies, or on account of the neutralizing effect upon the excessive alkalinity in the body, is as yet undetermined. One must also take into consideration the possibility of detoxifying and stabilizing process of the body produced by the ketogenic diet.

Our work this last year has been carried on at the Chicago State Hospital in the cottage for female epileptics with eight inmates. In the course of time we found that about forty per cent of the total number of patients were amenable to the dietary treatment. Of the forty per cent about one-half of them showed a marked improvement and the other half a moderate improvement.

Patients with marked mental deterioration were found to be unsuitable, also those with organic brain defects or with physical disease. Patients who were markedly underweight or those with pulmonary or renal disease were rejected.

We have had a few patients who were exceedingly interesting. One woman, forty-five years old, for twenty-eight years an inmate at the institution, has had daily major convulsions since infancy. She had a surprisingly small degree of mental enfeeblement and was physically in a good condition. Within less than a week after the administration of the ketogenic diet, her convulsions ceased. Her physical appearance changed a good deal, her complexion became clearer, her eyes brighter, her walk more elastic, and her interest in her surroundings more keen. She had not a single convulsion for several weeks until she broke her promise and succumbed to the temptation of a piece of candy; then she had a number of severe convulsions. We again put her on a diet, and the

seizures stopped until we made a little change in the diet which again caused some convulsions. This patient needed a highly restricted diet.

Another patient, about twenty-five years old, showed a cessation of convulsions while on a very moderate ketogenic diet. The restriction of carbohydrates was hardly large enough to cause acetone bodies to appear in the urine, but yet there was a marked improvement. When the diet was discontinued, she had from two to sixteen convulsions daily. The improvement in physical appearance was the first change noticeable, the greater mental alertness followed, and a little later the influence on the number as well as on the severity of convulsions could be demonstrated.

The cooperation of the patient is essential, since even a small error in diet will upset the results obtained after weeks of treatment.

In conclusion it might be stated that the ketogenic diet is not a cure-all in epilepsy, that it is of value only where constant individual observation by a physician is possible. We have found, however, a basis of attack on the disease and a starting point for a new direction of research.