THE EFFECT OF VITAMIN B_1 ON DIGESTIVE GLAND SECRETION

COMMUNICATION I. GASTRIC GLAND SECRETION IN DOGS WITH EXPERIMENTAL VITAMIN B_1 DEFICIENCY

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(Received March 31, 1955. Presented by Prof. V. N. Chernigovsky, Active Member of the AMS, USSR)

One of the most important questions in research concerning the mechanisms regulating digestive tract activity and their normal and pathologic states is the role played by vitamins in digestive gland activity. In spite of the numerous works on this subject, the exact effects of the various vitamins on the digestive glands has not yet been definitely established.

Many of our colleagues have studied the effect of various vitamins on the activity of gastrointestinal tract organs and tissues (N. V. Asmayan, A. V. Gubar, O. F. Sharovatova, S. I. Filippovich, Yu. N. Uspensky, N. G. Shchepkin and others). For the past few years, we have been making a study of experimental B_1-avitaminosis.

In research prior to this, B_1-avitaminosis with typical clinical symptoms and metabolism disturbance has been produced in dogs fed various diets (synthetic and autoclave-processed). It has also been established that the pathologic body condition caused by B_1-avitaminosis can be reversed by timely treatment with vitamin B_1. The experimental model obtained made it possible to proceed with the study of digestive gland (salivary and gastric) secretion in dogs with B_1-avitaminosis.

We found a series of authors in the literature (Tsitovich and Levinson, Levinson, Bikkel, Never and others) who came to conflicting conclusions after studying gastric secretion in dogs with polyavitaminoses and an isolated miniature stomach. Some authors found no changes of gastric secretion in B_1-avitaminosis, while others observed great changes in the secretory function.

Among the experimental works directly associated with our study, the unpublished work of N. A. Izmailova, conducted in the laboratory of Prof. M. P. Brestkin, should be mentioned. Her experiment showed that the reflex phase of gastric secretion diminished with B_1-avitaminosis and that the humoral phase of gastric secretion at first, grows, and then, during the last days before the death of the animal, diminishes greatly.

In our work, we studied the dynamics of gastric gland secretion in dogs during the development of B_1-avitaminosis and during the subsequent vitamin therapy.

EXPERIMENTAL METHODS

Gastric secretion was studied in dogs with an isolated miniature stomach.

The weight, diuresis, concentration of pyruvic acid in the urine, temperature, pulse and number of respirations were systematically determined, and the amount of food and water consumed daily was ascertained.

After a general background and a background of gastric secretion to the stimuli employed had been established, the animal was put on one of the experimental diets. During the development of experimental B_1-avitaminosis in the animals, we systematically recorded the changes in the secretion indices of the gastric glands and compared them with the clinical symptoms. The method and general and dietary conditions used to obtain experimental B_1-

avitaminosis in the dogs we have already described in detail in a previous article.

We used 100 g of autoclave-processed meat or 100 ml of 10% alcohol as stimuli (the alcohol was administered to the dogs rectally). The experiments were done daily; the amount of secretion, acidity and the digestive power of the juice were determined according to Mett.

The results given in this work were obtained from 4 dogs, 3 of which (Boy, Beetle and Topper) had a stomach isolated according to Brestkin-Savich, while the other (Daisy) had a Pavlov's miniature stomach and a gastric fistula. Two of the dogs (Boy and Beetle) were fed a casein saccharide diet, and the other two (Daisy and Topper) were fed an autoclave-processed diet. Over 500 experiments were conducted.

EXPERIMENTAL RESULTS

The first signs of avitaminosis sickness (diminished appetite, periodic refusal to eat and loss of weight) appeared in the experimental animals on the 15th-20th day after the beginning of the experimental diet. Deviations from normal gastric gland secretion appeared at the same time and intensified as the avitaminosis developed.

Experimental results are given in Figs. 1 and 2.

A slight qualitative change in secretion was observed in the animals during the first month of the vitamin deficient diet. Later, secretion became more profoundly disturbed; secretion fluctuated greatly to both stimuli, and gastric juice was secreted on an empty stomach.

In the experiments with the 100 g of autoclave-processed meat (Fig. 1), the amount of gastric juice was observed to fluctuate, the amounts primarily increasing, more and more as avitaminosis developed.

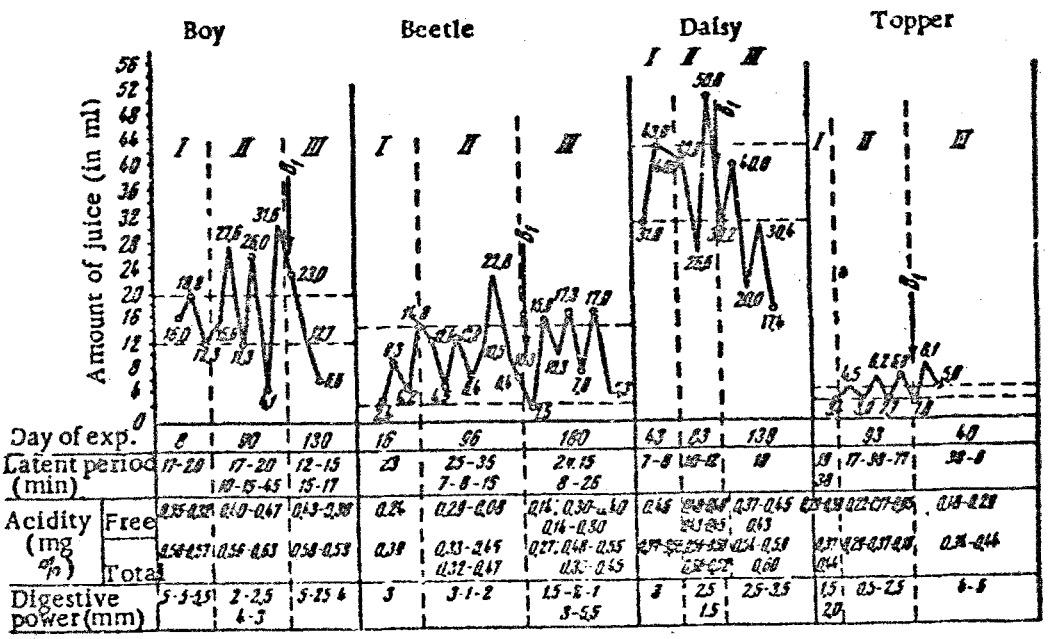

Fig. 1. Gastric secretion to 100 g of autoclave-processed meat. The fluctuation limits of gastric juice secretion in milliliters to the given stimulus are marked by the two horizontal dotted lines. Along the abscissa - duration of the period in days. Along the ordinate - amount of juice in milliliters (each number represents the arithmetical mean of fluctuations from several experiments in the given period). Numbers near dots on graph - amount of secretion in one experiment.
The periods are divided by the vertical dotted lines: I) background, II) development of avitaminosis, III) restoration.
Arrows signify the critical day of the disease and the first vitamin B_1 injection.

In the dog Boy, as Figure 1 shows, the amount of gastric juice, normally 18.8 ml, increased from 27.6 to 31.6 ml. In Beetle, the secretion, normally 14.8 ml, reached 22.8 ml. In Topper, the juice secreted during the development of avitaminosis was twice the normal amount. For example, juice secretion, normally 3.4 ml, increased to 6.2 ml, then sank sharply to 1.8 ml on the critical day of the disease. Secretion in the dog Daisy fluctuated at a level slightly lower than the norm (26.6 ml), with a sharp incline to 51 ml 12 days before the crisis.

Secretion fluctuations were also observed in the experiments with the rectal introduction of 100 ml of 10% alcohol, but the amount of gastric juice secreted was considerably less than normal.

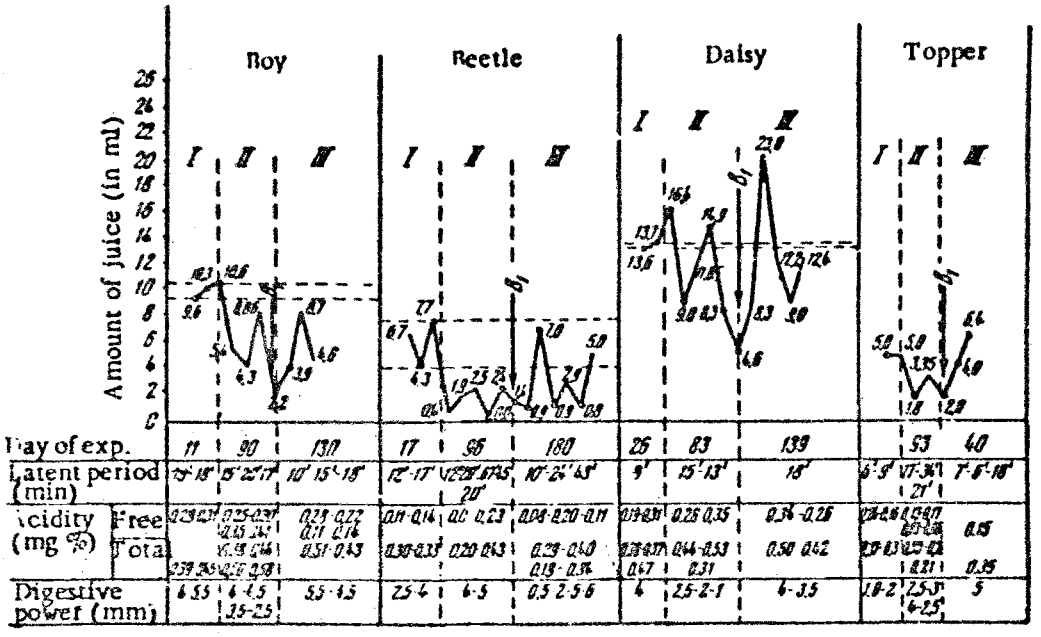

Fig. 2. Gastric secretion to 100 ml of 10% alcohol.
The symbols are the same as in Fig. 1.

Figure 2 shows that in the dog Boy, fluctuations of secretion, normally 9.6-10.6 ml, were observed during the development period of avitaminosis which were 4.3-8.8 ml, with a decrease to 2.2 ml on the critical day of the disease. As the avitaminosis condition became more serious in the dog Beetle, secretion sharply declined, reaching 2.5 and 0.0 ml in the acute stage of the avitaminosis, when the normal secretion was 4.3-7.7 ml. Secretion fluctuations were also lower than the norm in Topper (from 1.8 to 3.3 ml).

As the disease developed in the dog Daisy, sharper, intermittent secretion fluctuations above and below normal limits were observed. Thus, secretion, normally 13 ml, increased to 16 ml, then fell to 9 ml, increased again to 14 ml and then steadily decreased until the critical day of the disease when it was 4.6 ml.

Therefore, the experiments conducted showed that fluctuations occur in gastric secretion (hypersecretion or hyposecretion) to meat and alcohol during the development of avitaminosis.

Besides the changes which occurred in the amount of gastric juice during the development of avitaminosis and the subsequent vitamin therapy, changes were also observed in the latent period of secretion, the acidity and the digestive power of the juice to both stimuli (meat and alcohol).

Figure 1 shows the changes in the latent period, acidity and digestive power according to the periods (extreme limits of fluctuation are given). As the graph shows, the acidity of the gastric juice changes in the same manner as the amount of gastric juice, but not as much.

In some of the dogs, during the acute stage of the disease, we observed a divergence between the degree of acidity and the amount of gastric juice, which seemed to be connected with the changed trophic function of the glands.

During the development period of avitaminosis, the digestive power of the juice decreased, returning to normal, or sometimes to above-normal, only after long treatment with vitamin B_1 (Figs. 1 and 2). For example, the digestive power of the juice to meat in the dog Boy, Normally 5-6 mm according to Mett, had decreased by the first week of the vitamin-deficient diet to 2-2.5 mm, and only returned to normal after a month of treatment. During the development period of avitaminosis, the digestive power of the juice in Beetle fluctuated between 3 and 1 mm (the normal being 3 mm) and remained 2 mm for a month before the crisis, returning to normal only after 2 months of vitamin therapy. In Daisy, the digestive power returned to normal a week after vitamin therapy.

It is evident from the graphs that the latent period during the development of avitaminosis was variable-sometimes being longer, sometimes shorter. In some experiments, however, it became longer to both stimuli. For example, the latent period in the dog Boy, normally 17-20 minutes, became as long as 45 minutes. In Beetle, the latent period, normally 23 minutes, decreased to 7-8 minutes, then lengthened to 2 hours, 15 minutes. In the dog Topper, it lengthened to 77 minutes, i.e., became twice as long as the normal latent period.

Vitamin B_1 was injected intramuscularly (10 mg thiamine hydrochloride on the critical day of the disease when the animal was in a serious condition. Fifteen to twenty minutes after the vitamin B_1 injection, the general condition of the dog improved, vomiting ceased, and the complete refusal to eat was replaced by an extraordinary appetite. Thereafter, the sick dog was injected daily with 5-10 mg of thiamine hydrochloride until his complete recovery.

Vitamin therapy caused gastric secretion to become almost normal. At first, sharp secretion fluctuations were also observed during the treatment; there was still some decrease in secretion to meat and increase in secretion to alcohol in three of the dogs, Daisy, Boy and Beetle.

Therefore, the data obtained show that B_1-avitaminosis causes considerable distrubance in gastric gland secretion and also that there is a definite relation of gastric secretion to the degree to which the pathologic process occurring in the body with the development of B_1-avitaminosis is expressed.

Microscopic examination of the mucous membrane of the stomach in dogs with B_1-avitaminosis (Rudik-Gnutova) showed considerable hyperemia of the vessels of the mucous and submucous layers. The mucosa itself retained its normal structure, while a loss of secretory granules and vacuolization of the protoplasm could only be observed in the chief and cover cells, which could relate to the functional changes connected with the secretion of gastric juice.

The numerous experimental works and the mass of clinical literature treating vitamin B_1's effect on the nervous system show the close relation between thiamine and the function of the nervous system.

Vitamin B_1 is also known to play an extremely important part in body metabolism and in the metabolism of some body tissues — nervous, glandular, etc. Consequently, one can propose that a vitamin B_1 deficiency in the body can cause changes and distrubances in gastric secretion because of the metabolic changes occurring in the glandular tissue, in the nerve plexi of the digestive tract, in the central nervous system and in its highest division — the cerebral hemispheres.

A comparison of the physiological data we obtained with the data from our histological examinations, which showed an absence of organic distrubances in the glandular cells of the gastric mucosa, led us to propose that the distrubance of gastric gland secretion which occurs in B_1-avitaminosis is functional and depends on neural regulation.

Which of the divisions of the nervous system regulating gastric secretion is the most affected by B_1-avitaminosis is a subject for further investigation.

SUMMARY

The quantity of secretion, its acidity and power of digestion was studied in experiments on dogs with isolated Paubu's stomach in condition of B_1 insufficiency. Mett's method was employed. It has been established that vitamin B_1 insufficiency causes pronounced disturbances in digestive glands. The power of digestion of the stomach juice drops from 5-6 cc to 2-2.5 cc. The quantity of stomach juice increases up to 31.6 cc, the normal being 18.8 cc.

LITERATURE CITED

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