

EFFECT OF THE UNCONDITIONED FOOD REFLEX ON THE RHYTHM
OF CARDIAC ACTIVITY UNDER PATHOLOGICAL CONDITIONS
(AGAINST THE BACKGROUND OF DIPHTHERIA INTOXICATION,
ACUTE DISTURBANCES OF CORONARY CIRCULATION,
AND PHARMACOLOGICAL EFFECTS)

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In the clinical and experimental investigations by a number of authors [1, 4, 8, 10, 15, 21] the possibility has been established of marked changes of cardiac activity under the effect of reflexes from the organs of the digestive tract.

The effect of food reflexes (conditioned and unconditioned), simulated feeding, swallowing movements, and hunger contractions of the stomach on the rhythm of cardiac activity has been studied by many investigators [2, 9, 11, 12, 13, 22, 23]. It was observed that the food reflex causes an acceleration of the rhythm of cardiac contractions. Direct stimulation of the stomach may exert its effect on cardiac activity, depending on the character of action. Introduction of warm water into the gastric cavity causes a slight deceleration of the cardiac rhythm [6].

Under conditions of cardiac denervation, physiological and emotional reactions, including those induced by food stimuli, are not accompanied by changes in the cardiac rhythm [11, 12].

Of special interest is the problem of the effect of food reactions on the cardiac rhythm under conditions of pathological cardiac activity. There have been cases encountered in the clinic where the vomiting reflex was accompanied by a normalization of the impaired rhythm of cardiac activity [3, 7, 14].

Cases have been described where swallowing movements provoked disturbances of the cardiac activity and, inversely, where they contributed to the normalization of cardiac activity [16]. G. F. Lang [7] pointed out that the nature of these influences is still obscure.

Our work has been carried out with the aim of tracing the effect of unconditioned food and drink reflexes on the rhythm of cardiac activity under various types of its disturbance.

METHODS OF EXPERIMENTS

Experiments were conducted on dogs. Changes of the rhythm of cardiac activity connected with the food reflex were studied on healthy animals, animals with isolated N. vagus, isolated sympathetic cardiac innervation, and in various types of cardiac arrhythmia.

Diphtheria intoxication was produced via single and fractional injections of the toxin in nonlethal and lethal doses. The maximum duration of the disease with a lethal outcome following a single injection of the diphtheria toxin exceeded a month. Strophanthin poisoning was induced via repeated intravenous administrations of the preparation.

Disturbances of coronary circulations were obtained via application of 3 to 5 ligatures to the large branches of the coronary arteries.

The electrocardiograms were recorded by means of "Siemens" and "EKP" electrocardiographs and the "4 PFD" ink-recording device.

In selecting the food stimuli we considered the possibility of a marked reduction of food excitability of the animals in the course of the disease, and employed strong stimuli in our experiments (meat and meat-sugar powder, sausage products, sour cream, sugar, and milk). In cases of marked reduction of food excitability the animals often drank water greedily.

RESULTS OF EXPERIMENTS

Over 1000 observations of dynamic changes of the cardiac activity rhythm in the course of food reactions of healthy animals against the background of normal cardiac activity indicate a monotype character of these changes. During the first few seconds of the unconditioned food reflex the cardiac rhythm becomes accelerated. The extent of acceleration depends on the food excitability of the animal. At a high food excitability, the cardiac rhythm rate at the beginning of the food reflex exceeds by 150-200% the initial rate. The maximum acceleration takes place during the first 6-9 seconds, less frequently, in the first 12 seconds of the food reflex. Starting with the 6th to 12th second, the rhythm of cardiac contractions slows up perceptively, but remains above the initial level up to the end of the food or drink reflex.

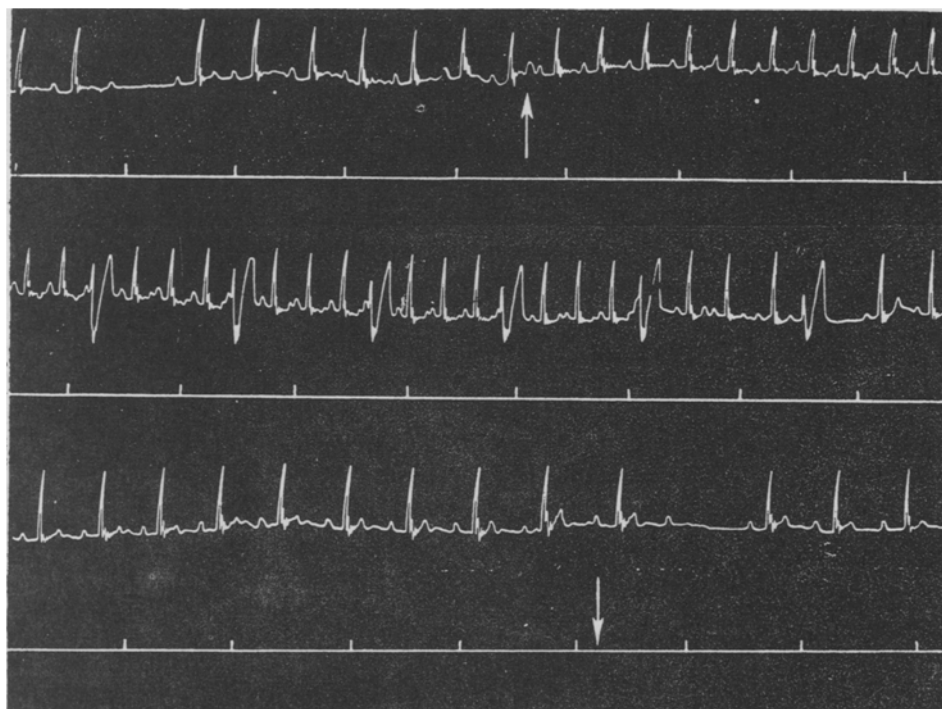


Fig. 1. Changes of the rhythm of cardiac activity in the course of an unconditioned food reflex against a background of prolonged, chronic strophanthin poisoning (the dog drinks milk). The start and termination of the reflex are indicated by arrows. Prior to the start of the unconditioned reflex and following its termination, an incomplete atrio-ventricular stage II block is noted. The food reflex is accompanied by acceleration of the rhythm of cardiac contractions, improvement of atrioventricular conductivity, and the emergence of ventricular extrasystoles at the height of maximal acceleration of the cardiac rhythm. Designation of curves (from top downward): EKG; annotation of time (one second).

Experiments with food reflexes against a background of partial denervations of the heart were staged on 12 dogs.

The desympathization of the heart was performed by separating as much as 10 pairs of the anterior radicles of the thoracic segments of the spinal cord, starting with the 8th cervical. Following this intervention, the cardiac rhythm acceleration at the start of the food reflex became less pronounced. The cardiac components of the food reflex did not revert to the initial figures until 9 months after the operation.

After a high separation of N. vagus on the neck, the reduction of food excitability and dispeptic phenomena (eructation of food, etc.) hamper the study of the cardiac components of the food reflexes. Against the background of marked reduction of food excitability in the vagotomized animals, perceptible changes of the cardiac rhythm may be absent in the course of the food reflex. When a higher food excitability of vagotomized dogs was present in the course of the food reflex, a distinct, though slight, acceleration of the cardiac rhythm by 10-20 contractions per minute was observed.

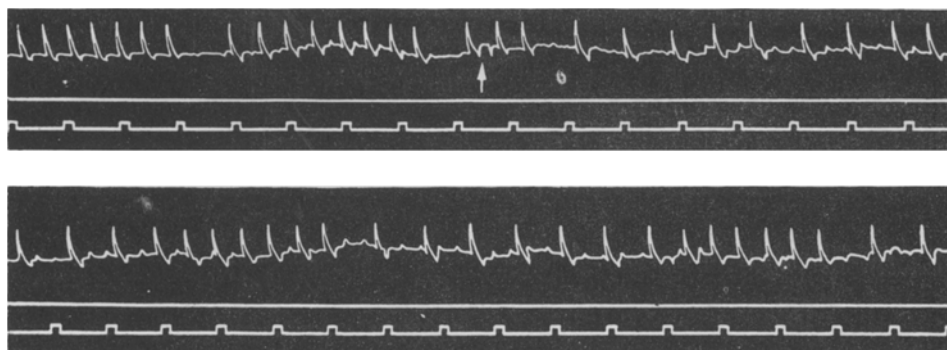


Fig. 2. Acceleration of the atrial rhythm and worsening of atrioventricular conductivity along the path of the unconditioned drink-reflex against the background of severe strophanthin poisoning. The start of the reflex is indicated by an arrow. Designation of curves—as in Fig. 1.

Following exclusion of N. vagus via subcutaneous injection of atropine (from 2 to 4 ml of 0.1% solution), any subsequent acceleration of the cardiac rhythm in the course of a food reflex is possible only at a fairly high food excitability of the animal (we used milk in these experiments). Within 25-30 minutes after atropine injection, with the background of cardiac rhythm at 215 contractions per minute, we observed its acceleration to 250 per minute at the start of the food reflex, and to 270 contractions against a background of 250. (The rate of cardiac rhythm is always indicated in our recalculation per contractions per minute, though in reality we speak of rhythm changes at the start of the food reaction for time segments of 12 seconds' duration).

In diphtheria intoxication (experiments on 9 dogs), during the first days of the disease the cardiac components of the unconditioned food reflex are well expressed. Against a background of stage II of an atrioventricular block at the start of the disease and in the course of an unconditioned food reflex we observed a distinct improvement of conductivity.

This fact indicates the important role of the neurogenic component in the disturbances of atrioventricular conductivity in the early stages of diphtheria.

During the subsequent course of the development of the disease, against the background of marked acceleration of cardiac contractions, the food reflexes are accompanied only by slight changes of cardiac activity analogous to its changes in vagotomized animals.

In chronic strophanthine poisoning (observations on 6 dogs), the changes of the cardiac activity rhythm in the course of an unconditioned food reflex depend on the stage of development of the pathological process.

In the most severe forms of intoxication, against a background of a paroxysmal ventricular tachysystolia, the food excitability of the animals was markedly reduced, whereas the drink reflexes induce no distinct changes of the rhythm under these conditions. In less severe forms of intoxication against the background of an incomplete stage II atrioventricular block, the food reflex is accompanied by an acceleration of the rhythm of cardiac contractions most clearly expressed during the first few seconds of the unconditioned reflex. In the majority of instances this is accompanied by a distinct improvement of atrioventricular conductivity and a normalization of the cardiac rhythm. However, diverse variants are possible, depending on the degree of intoxication.

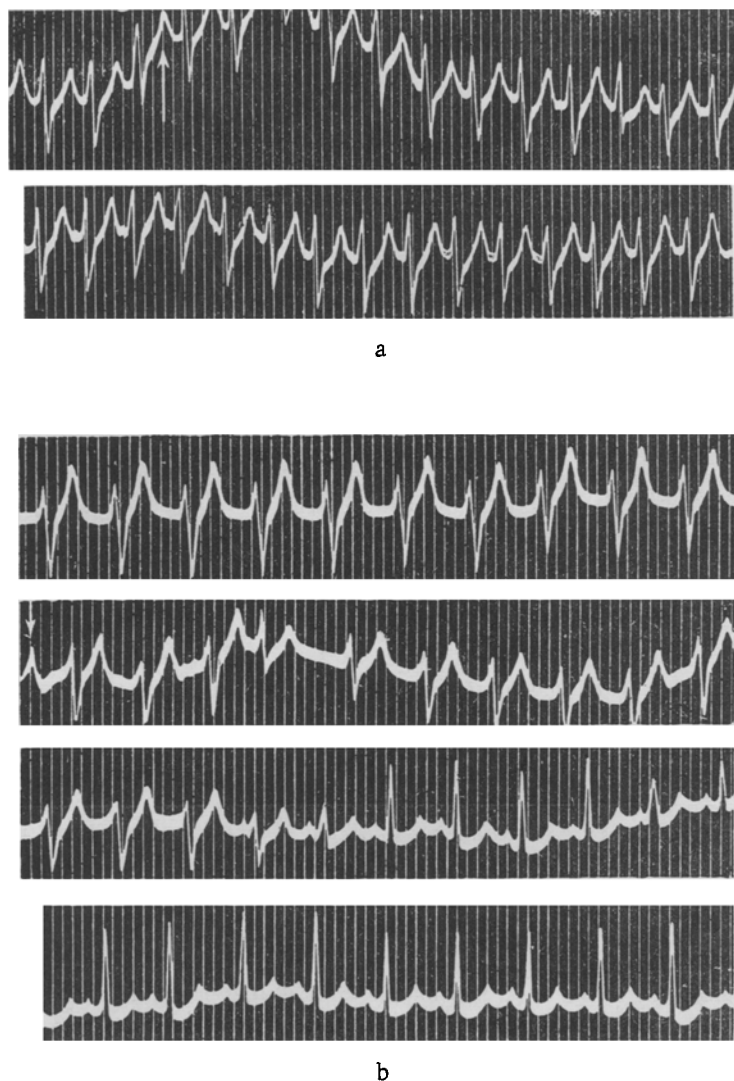


Fig. 3. Unconditioned food reflexes against the background of acute disturbances of the coronary circulation. a) Unconditioned food reflex within 24 hours after ligation of the coronary arteries against the background of a severe ventricular tachysystole (240 contractions per minute) is not accompanied by substantial changes of the cardiac activity rhythm; b) unconditioned food reflex in the same dog within 48 hours after ligation of the coronary arteries against the background of ventricular tachysystole (170 contractions per minute) is accompanied by normalization of the cardiac rhythm within a few seconds after the start of the reflex. The start of the unconditioned food reflex is marked with an arrow.

In one of our dogs which has been receiving toxic doses of strophanthin for six years, the unconditioned food reflex (the dog drank milk) was accompanied not only by an acceleration of the sinus rhythm and improvement of conductivity, but also by the emergence of ventricular extrasystoles. They appeared during the first half of the food reaction, at the height of the maximal acceleration of the cardiac rhythm. The extrasystoles in this dog disappeared after the first 12 seconds of the unconditioned food reflex (Fig. 1).

The emergence of ventricular extrasystoles simultaneously with a maximal acceleration of the sinus rhythm and an improvement of atrioventricular conductivity offers a basis for the conclusion that they are connected with an increased tonus of the sympathetic cardiac nerves at the start of the food reflex. The food reflex revealed under these conditions latent foci of intensified activity of heterotopic ventricular automatism.

Acceleration of the sinus rhythm and the improvement in atrioventricular conductivity in the course of the food reflex must be connected principally with the reduction of the tonus of cardiac centers of *N. vagus*. We observed the improvement of conductivity during the unconditioned food reflexes in five out of six dogs. We encountered an opposite phenomenon in only one of our dogs at the height of severe strophanthin poisoning. A marked acceleration of the atrial rhythm was accompanied in the course of the drink reflex not by an improvement, but by a worsening of atrioventricular conductivity (Fig. 2). In our opinion, this should be regarded as the manifestation of profound parabiologic changes in the conducting system of the heart: the acceleration of impulses which were directed toward the atrioventricular node intensified the parabiologic state of the latter.

In acute disturbances of coronary circulation (experiments on nine dogs), the changes in the rhythm of cardiac activity along the path of food and drink reflexes depended on the time elapsed since the ligation of the coronary arteries and on the initial background of cardiac activity. In paroxysmal ventricular tachysystole of over 200 contractions per minute, the unconditioned food and drink reflexes were not accompanied by substantial changes of the cardiac activity rhythm (Fig. 3, a).

In paroxysmal ventricular tachysystole under 200 contractions per minute, the food and drink reactions were accompanied by distinct changes of the cardiac rhythm. The heterotopic ventricular rhythm changes to a regular sinus rhythm within a few seconds after the start of the unconditioned food reaction (Fig. 3, b).

At a low activity of the foci of heterotopic ventricular automatism against a background of solitary or group ventricular extrasystoles, within 3-8 days, less frequently two days following ligation of the coronary arteries, the unconditioned food reflex produced at once a normalization of the cardiac rhythm in connection with the increase in the activity of the sinus node.

In order to explain the mechanism of the effect of the food reflex on the cardiac activity rhythm in various types of its disturbances, it is expedient to compare the observed changes of cardiac activity with the peculiarities of its regulation at various stages of the pathological process.

The literature [5, 20] and our previous reports [17, 18, 19] point out the effect of extracardial nerves' separation on the initial functional state of the cardiac activity. We observed a stable normalization of the cardiac rhythm resulting from the separation of *N. vagus* in the disturbances of atrioventricular conductivity and in ventricular rhythms of relatively low frequency. We observed no normalization of the cardiac rhythm, if *N. vagus* had been separated against the background of ventricular tachysystolia with a rate of over 200 contractions per minute. Analogous results were elicited in the study of the effect of the food reflex on the heterotopic ventricular rhythm.

In evaluating the character of changes of the cardiac components of food reflexes after surgical and pharmacological interventions on cardiac nerves, as well as the peculiarities of the effect of these reflexes on the cardiac rhythm in various types of disturbances, one arrives at the conclusion that the changes of the rhythm of cardiac contractions in the course of a food reflex are connected principally with the reduction of the tonus of the central centers of *N. vagus*. An increased tonus of the sympathetic cardiac nerves is clearly manifested during the first few seconds of the unconditioned food reflex if the animal possesses sufficiently high food-excitability.

The results of our investigation indicate the possibility of utilizing food and drink reflexes as harmless physiological tests which would permit us to ascertain the peculiarities of the neural regulation of impaired cardiac activity, and to a considerable extent reveal the possibility of its normalization under the effect of surgical neural intervention.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
