CHANGES IN THE SECRETORY AND MOTOR FUNCTION OF THE STOMACH IN EXPERIMENTALLY INDUCED GASTRITIS

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The use of surface coagulation of the gastric mucosa by 10% solution of $AgNO_3$ for production of inflammatory changes accompanied by inhibition of gastric secretion was developed in the laboratory of I. P. Pavlov [1, 4]. In recent times this method has been used for the study of blood and cardiovascular changes associated with gastritis [3, 5, own observations].

EXPERIMENTAL METHOD

In the course of the present work the secretory-motor function of the stomach was investigated in 2 dogs with gastric fistulas. Inflation of a rubber balloon introduced into the stomach was used as a stimulus. Gastric juice was collected every 15 minutes during the first hour of the experiment; each specimen was examined for amount of juice, total acidity and free hydrochloric acid. The motor activity of the stomach was recorded manometrically during a period of 2 hours; the gastric tonus was judged from the water manometer readings. The experiments were carried out 18-19 hours after the last meal. Experimental gastritis was produced by irrigation of the gastric mucosa, by way of the fistula, with 1000 ml 10% solution of $AgNO_3$ over a period of 5 minutes.

EXPERIMENTAL RESULTS

The character of gastric secretion of the dog Reks in response to mechanical stimulation under normal conditions and during experimentally induced inflammation of the mucosa is presented in the table and in Fig. 1.

Changes of Gastric Secretion in the Dog Reks Before and After Surface Coagulation of the Mucosa

Secretory indices	Before coagulation of the mucosa	After coagulation of the mucosa		
		1st week	2nd week	2nd month
Hourly output in ml	16—60	0	10—12	4—14
Free HCl in %	0,226—0,378	0	0—0,164	to 0,124
Latent period in minutes	5—10	0 (mucus in large quantities)	15—60	10—30

As can be seen from the table, during the first few days following surface coagulation there was no secretion; only considerable amounts of mucus, sometimes bloody, were observed. A prolonged period of hyposecretion set in from the second week, and lasted about 2 months. Gradual restoration of secretion in response to mechanical stimulation was only seen during the 3rd month after surface coagulation of the mucosa.

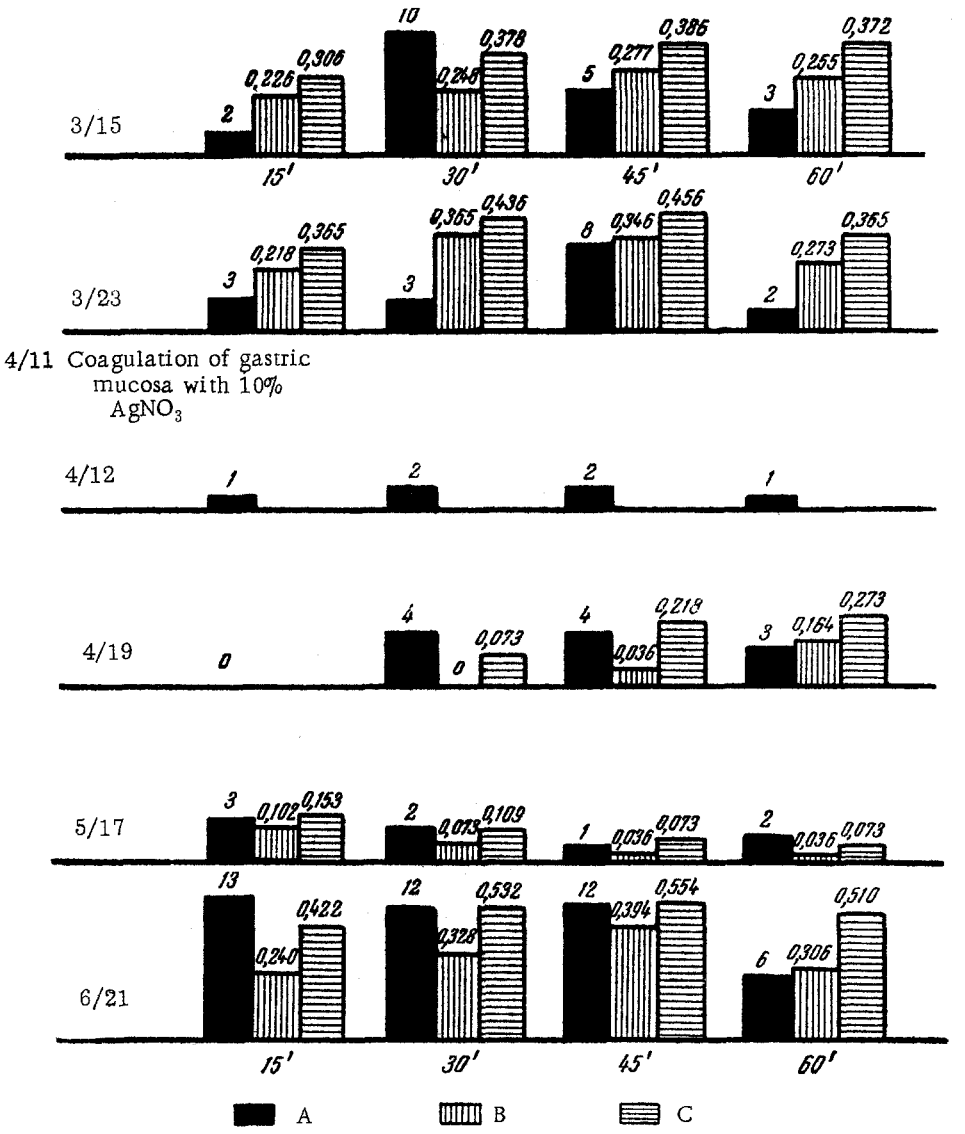

Fig. 1. Changes in gastric secretion in experimentally induced gastritis, dog Reks. A) Amount of gastric juice in milliliters; B) free hydrochloric acid in percentages; C) total acidity in percentages.

A similar sequence of changes was noted in the case of the dog Dichka with the difference that there was no complete inhibition of secretion during the first week.

In the normal state the motor activity of the stomach continued throughout the time of mechanical stimulation. The dog Reks showed rhythmic contractions of 10-12 seconds' duration 1-5 minutes after the onset of stimulation. By approximately the end of the first hour prolonged increase in gastric tonus (to 36-40 cm H_2O compared to the initial 20-24 cm), lasting 40-60 seconds with pauses up to 1 minute. This character of gastric movement was maintained until the end of recording. In the case of Dichka, the latent period was longer and contractions more frequent, seen against a background of moderately increased gastric tonus (from 24-26 to 30-32 cm H_2O). These contractions were only infrequently replaced by slower waves.

The day following surface coagulation of gastric mucosa both dogs showed profound inhibition of gastric motor activity, despite the same strength of mechanical stimulation. This occurred against the background of increased gastric tonus (30-32 H_2O). During subsequent days (first week) the motor activity increased gradually. Low amplitude waves with long intervals between them appeared after a prolonged latent period of 20-30 minutes.

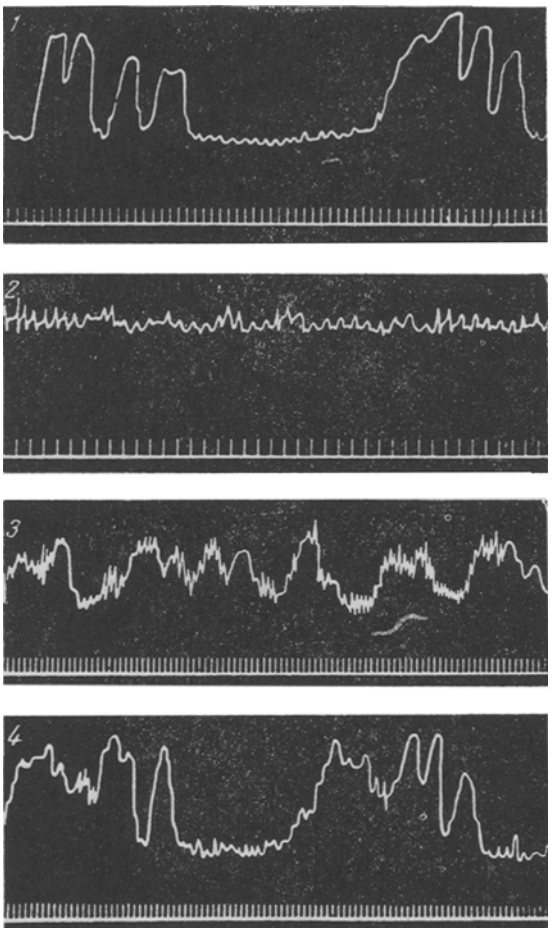

Fig. 2. Changes in motor activity of the stomach in experimental gastritis in the dog Reks. All the gastrograms recorded during the middle of the 2nd hour of the mechanical stimulation; 1) Normal motor activity, 2) 2nd day of gastritis, 3) 10th day of gastritis, 4) recovery of normal motor function 1 month after surface coagulation of mucosa. Time marker – 3 seconds.

This type of motor activity resembles "fasting periodicity." Seven-ten days after surface coagulation the motor activity assumed a haphazard character. This type of curve may be likened to the "arrhythmic" type described clinically in chronic gastritis [2].

The initial character of motor activity was restored in both dogs by the end of the first month after coagulation of the mucosa.

The dynamics of the described changes are presented in Figs. 2 and 3.

The damaging effect of an experimental pathogenic factor on the motor function of the stomach thus proved to be less marked and more transient than its effect on the secretory function.

The uniformity of secretory impairment, viz., inhibition, indicates that the model of gastritis employed cannot reflect fully all the multiplicity and variety of disturbances of the secretory process in inflammatory diseases of the stomach.

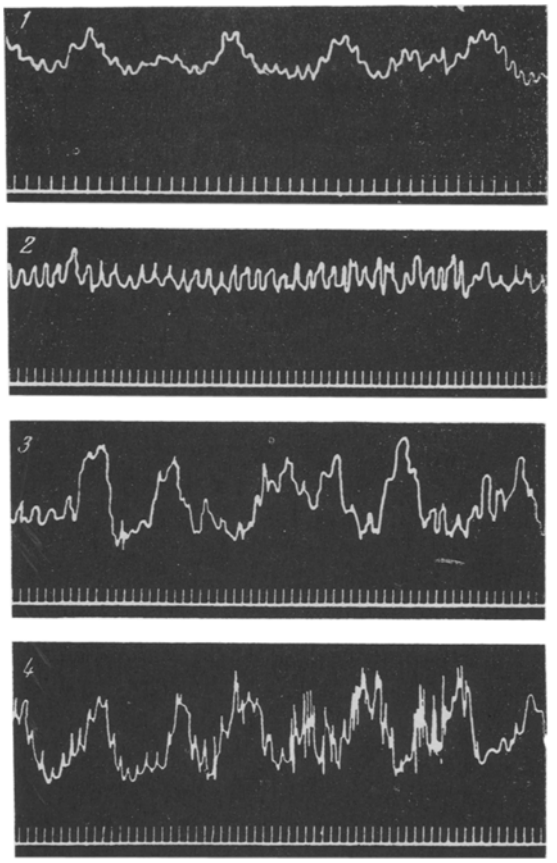

Fig. 3. Changes in gastric motor activity in experimental gastritis in the dog Dichka. All gastrograms recorded in the middle of the first hour of mechanical stimulation. 1) Normal motor activity, 2) 2nd day of gastritis, 3) 4th day of gastritis, 4) 20th day of gastritis. Time marker — 3 seconds.

SUMMARY

Experimental gastritis was induced in dogs by surface coagulation of the gastric mucosa with 10% silver nitrate solution. This gastritis was characterized by depression of the secretory function, inhibition of gastric peristalsis and increased tone of the stomach. The secretory function was re-established during the third month, the motor activity was restored at the end of the first month after coagulation of the gastric mucosa.

The secretory disturbances in experimental gastritis were found to be more considerable and lasting than derangement of gastric motility. This shows that the injury of the secretory glandular apparatus of the stomach was more pronounced.

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