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The essential basis of our bloodless method of reducing gastric secretion is that after determination of the nature of the lesion (duodenal ulcer) the secretory zones of the stomach are identified by the method of intragastric local pH measurement through an endoscope with staining of the gastric mucosa with Congo red. Freshly prepared hypertonic glucose solution is injected through a separate channel of the endoscope, by means of an injector needle, into the submucosa of the secretory zone of the stomach. By infiltrating the secretory zone of the stomach with this solution, hydrochloric acid production is inhibited.

In this paper we give the results of an experimental study of the acid-producing function of the stomach before and after infiltration of the secretory zones of the stomach (from 2 weeks to 1 year).

The investigation was conducted on 35 mongrel dogs. To study the secretory function of the stomach before and after infiltration of the acid-producing zones an experimental model was created. Under hexobarbital anesthesia gastrostomy was performed on all the animals by K. P. Sapozhkov's method. The dog was fixed to a Pavlov frame and normal gastric function studied by the following methods: determination of the volume of gastric juice and total hydrochloric acid production, acidity of the gastric contents, the rate of hydrochloric acid production, acid and alkaline components of secretion, and intraventricular pH-metry.

Since peptic ulcer is accompanied by excess activity of the gastric glands, the investigation always began with determination of basal secretion. The stimulated phase of gastric secretory function was studied by means of maximal histamine stimulation by Kay's method. The study of the quantitative composition of the gastric secretion was essential in order to determine the mass of the parietal cells.

The rate of hydrochloric acid production in unit time was studied by measuring the hourly quantity of basal secretion (BAO) and the hourly secretion of hydrochloric acid after histamine stimulation (MAO).

The gastric juice obtained by aspiration contained a wide range of impurities, with an alkaline reaction (mucus, bile, etc.), which necessitated making allowance for the alkaline component of the secretion and determining the true rate of hydrochloric acid production. Besides catheterization studies of the acid-producing function of the stomach we also used an electrometric method of pH measurement. This investigation also was carried out in the fasting state and after Kay's maximal histamine test. By comparing basal and stimulated phases of gastric secretion, the relative importance of functional and organic factors in the genesis of the secretory disorders was assessed.

To study the intensity of acid formation before and after stimulation, Noller's alkaline test was used.

After determination of the normal state of gastric secretion in the animals an operation was performed with the aim of acting on the secretory zone of the stomach. The operative technique consisted of laparotomy and gastrostomy, followed by identification of the secretory zones of the stomach by local intragastric pH-metry and chromoscopy, based on the ability of a 0.3% solution of Congo red, on contact with the secretory zones of the stomach, to change its color from red to dark violet. After precise identification of the boundaries of the secretory zones of the stomach, this zone was then infiltrated with hypertonic glucose solution.

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TABLE 1. Mean Values of BAO (in meq/liter) and MAO (in meq/liter) before (a) and after (b) Infiltration of Gastric Secretory Zones

Parameter	BAO		MAO	
	a	b	a	b
Rate of HCl production	1.0±0.34		7.9±0.25	1.5±0.11
True rate of HCl production per hour	1.25±0.25		9.0±0.30	2.0±0.35

TABLE 2. Mean Values of Intra-gastric Local pH-Metry before (a) and after (b) Infiltration of Gastric Secretory Zones

Part of stomach	Curvature of stomach				Wall of stomach			
	greater		lesser		anterior		posterior	
	a	b	a	b	a	b	a	b
Antral	7.0±0.10	7.8±0.31	6.8±1.02	7.6±0.21	7.6±0.31	7.0±0.4	6.8±0.18	7.0±0.40
Lower third of body	6.0±0.15	7.8±0.41	6.6±0.60	7.6±0.30	4.2±0.11	7.4±0.35	4.0±0.10	7.5±0.35
Middle third of body	4.7±0.25	7.8±0.22	5.5±0.45	6.8±0.17	1.8±0.03	7.8±0.27	1.6±0.01	6.8±0.20
Upper third of body	6.0±0.50	6.8±0.35	4.3±0.35	6.4±0.17	4.8±0.09	6.8±0.17	2.0±0.04	7.0±0.16
Subcardinal	2.8±0.44	6.4±0.80	4.3±0.53	6.7±0.20	2.4±0.10	6.5±0.20	2.6±0.01	7.4±0.23

The following normal values for the state of gastric secretory function in the dogs were obtained by the methods described above: total quantity of gastric juice produced during a 1-hour investigation of basal secretion varied from 7 to 10 ml, the content of free HCl in it from 110 to 130 titration units, the rate of HCl production ($M = m$) was 1.0 ± 0.34 meq/liter, and its true rate 1.25 ± 0.25 meq/liter. After maximal stimulation by histamine the volume of gastric juice varied from 75 to 80 ml, the content of free HCl was from 110 to 130, i.e., the rate of HCl production ($M \pm m$) was 7.9 ± 0.25 meq/liter, and the true rate of production 9.0 ± 0.30 meq/liter (Table 1). Investigation by intra-gastric pH-metry showed that the basal pH varied from 2 to 1, acid formation was continuous, the alkaline test result was 10 min, alkalification did not reach the level of anacidity, evidence of the marked intensity of acid production. Stimulation by Kay's method was accompanied by a hyperacid reaction, with the pH falling to 0.9-0.8. The low pH values remained at this same level for more than 1 h. The difference in the readings of the antral and cardinal electrodes was nearly always negligible and did not exceed 1.0 (Table 2).

After intra-gastric inhibition of secretion, the acid-producing function of the stomach was investigated by the methods described above. The total volume of fasting gastric juice was extremely small, and the juice consisted of mucus with a neutral reaction, and with absence of free HCl. The HCl production rate correspondingly could not be determined. During the study of gastric secretory function after stimulation the volume of gastric juice varied from 21 to 23 ml, the acidity of the gastric juice after maximal stimulation was from 60 to 65, i.e., the rate of HCl production ($M \pm m$) was 1.5 ± 0.11 meq/liter and the true rate 2.0 ± 0.31 meq/liter.

Investigation by the method of intra-gastric pH-metry showed that the basal pH varied from 7 to 5.5 in different parts of the stomach, evidence of absence of acid formation. The alkaline test could not be determined. After histamine stimulation the pH fell by 3.0-2.5 units after 40-60 min. The readings of the antral electrode under these circumstances were virtually unchanged.

Histological and histochemical investigations of the stomach wall of the dogs were carried out before and after the suggested method. The following results were obtained.

Between 3 and 4 weeks relative integrity of the parietal cells was observed, and in the upper levels the glands of the mucosa were formed entirely from parietal cells, whereas in the lower (basal) levels they contained degeneratively changed chief and accessory cells: hypertrophy of the parietal cells was observed in these zones, and in some cells the number of nuclei was doubled.

Between 6 and 8 months the boundaries of all layers of the wall became clearer, with focal sclerosis of the mucosa and atrophy of the glands in the fundal region of the stomach. marked degenerative changes of vacuolation type were visible in the preserved parietal cells.

Between 9 and 12 months degenerative and necrobiotic changes affected all types of cells and were manifested chiefly as intensive vacuolation of the cytoplasm, amounting in some cases to balloon degeneration, karyorrhexis, and lysis followed by cytolysis. After staining by Van Gieson's method (with picrofuchsin) there was a clear tendency toward atrophy of the mucosa, evidently linked with denudation of the reticular basis of the glands as the result of death of functionally active cells.

Analysis of the results thus demonstrated the development of considerable inhibition of gastric secretory function, as shown by absence of free hydrochloric acid in the basal secretion. According to the results of maximal histamine stimulation, there was a clear decrease in excitability of the gastric glands with the development of a hyporeactive type of gastric secretion. The causes of these changes were revealed by morphological investigations. They were severe atrophy of the gastric glands, disturbances of the proportions of their cellular composition, and the appearance of functionally defective chief and accessory cells.

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EFFECT OF STREPTOZOCIN ON TEMPERATURE VARIATIONS IN RATS

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According to recent reports in the literature, the blood sugar level is not a perfect criterion for determining the state of diabetics: normoglycemia can coexist with ketonuria, debility, malaise, and worsening of retinopathy [5, 6, 10]. There is thus the need to discover new tests for the diagnosis and evaluation of the state of diabetics. At the present time, the state of these patients is determined on the basis of a whole range of parameters, both chemical and physical [1, 3, 4, 7, 8]. Physical methods of investigation are attractive because they may be noninvasive. One such method is thermometry. It reflects the state of the body's energy metabolism. In diabetes this type of metabolism can be assumed to be disturbed because 70% of it is maintained at the expense of carbohydrates [9]. This hypothesis is supported by observations showing changes in body temperature in hypo- and hyperglycemic coma [8]. It is also reasonable to expect disturbance of temperature reactions to various procedures.

The aim of this investigation was to test experimentally the effect of a disturbance of carbohydrate metabolism on the time course of the temperature reactions of the body at rest and during variations of the ambient temperatures. To do this, the difference of the core temperature of the body and the temperature of its surface layers was measured over a period of time at room temperature, and during cooling and heating of the body, under normal conditions and after administration of streptozocin.

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