

lows: 1) In the early stages (6 h) the scale of destruction of the pancreas is reduced, zones of injury are demarcated, hemorrhages, inflammation, and vascular changes disappear, and the secretory activity of the pancreas is depressed; 2) after 1-3 days the zones of necrosis become smaller, demarcated, and later cleared of debris, and show evidence of initial repair; 3) after 7 days complete healing of the zones of injury takes place with signs of substitutive sclerosis of the pancreas and of incomplete regeneration; 4) the time course of the parameters of pancreatic activity corresponds completely to the morphologic data.

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MORPHOLOGIC STUDY OF THE EFFECT OF VAGOTOMY ON THE MUCOSAL MICROFLORA OF THE STOMACH AND DUODENUM

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In the modern view, besides the bacterial flora of the lumen, the luminal or L-flora in the small intestine of higher animals and man, there is also a flora which is directly connected with structures of the mucous membrane, the mucosal or M-flora [4-6]. It has been shown that the conditions of life of laboratory animals, and also various pathological processes involving the human digestive organs affect the qualitative and quantitative characteristics of both the L-flora and the M-flora. In particular, in duodenal ulcer, the quantity of both the L- and M-flora is increased [5, 6].

A widespread method of surgical treatment of duodenal and gastric ulcer with a hypersecretory syndrome at the present time is vagotomy [2, 3]. The reduction of the acidity of the gastric contents resulting from vagotomy is bound to affect the state of the microflora. However, no morphological investigations into the effect of vagotomy on the state of the M-flora and its interaction with the epithelial cells of the gastric and duodenal mucosa have yet been undertaken. The aim of the investigation described below was to study these problems.

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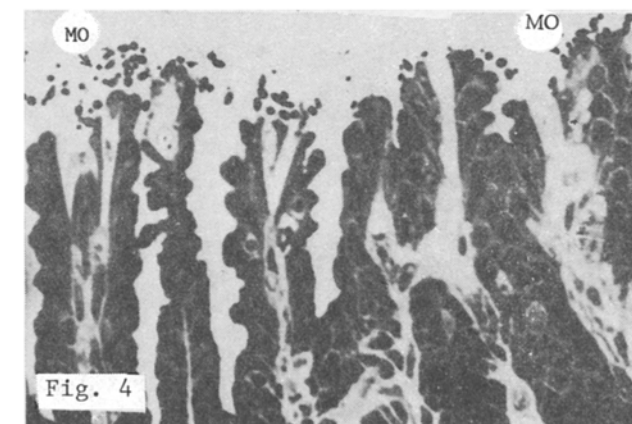
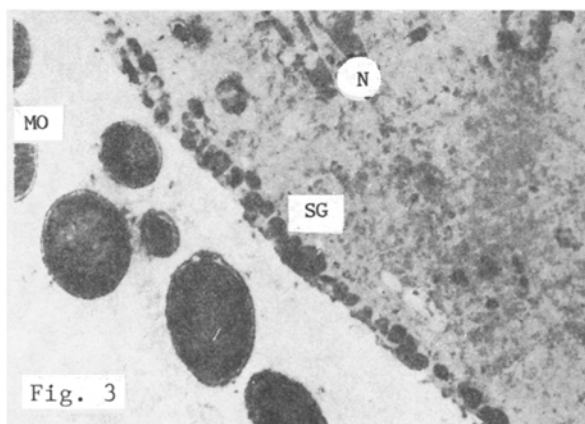
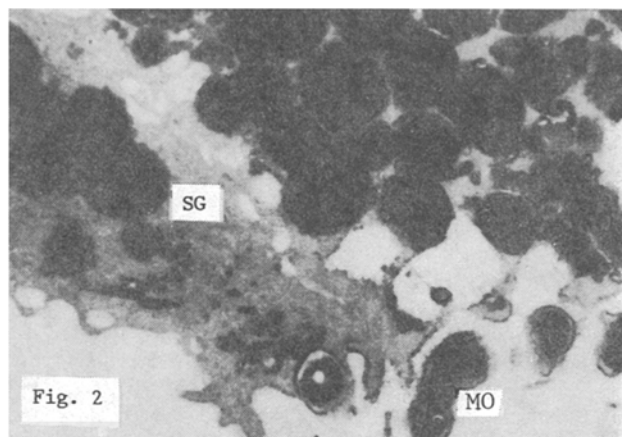
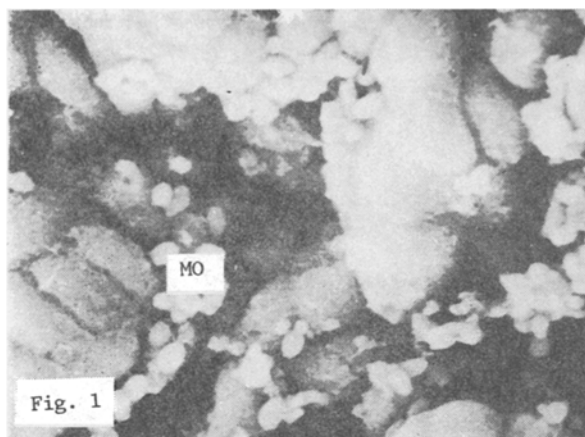


Fig. 1. Microorganisms (MO) on surface of mucosal cells of pylorus 30 days after vagotomy (SEM, 4000 \times).

Fig. 2. Contact between microorganisms and surface of pyloric gland cells of stomach 60 days after vagotomy (TEM, 15,000 \times). SG) Secretory granules.

Fig. 3. Microorganisms (MO) on surface of pyloric gland cells of stomach 60 days after induction of chronic ulcer (TEM, 7500 \times). N) Nucleus.

Fig. 4. Microorganisms (MO) on surface of pyloric cells 60 days after induction of ulcer and 30 days after vagotomy (semithin section, 200 \times).

EXPERIMENTAL METHOD

The effect of vagotomy on the mucosal microflora or "contact microflora," (by analogy with the term "contact digestion" [7]) was studied in 72 Wistar rats. Subdiaphragmatic trunk vagotomy was performed on 18 rats. In 40 animals chronic gastric ulcers were induced by the method in [8], described in detail in the Soviet literature [1]. In 18 of them, 30 days after induction of the ulcers, trunk vagotomy was performed. The control group consisted of 14 rats. Samples of gastric and duodenal mucosa were washed twice for 30 sec each time in sterile physiological saline and then treated by the usual methods for study by transmission and scanning electron microscope (TEM and SEM, respectively). Semithin sections were stained with methylene blue and fuchsin. The relative volume of the microflora in the region of the epithelium and in the juxtamural zone was determined stereometrically. The juxtamural or contact zone was taken to be a distance of not more than 40-45 μ from the surface of the epithelial cells in different parts of the stomach and duodenum, corresponding to the average height of the epithelial cells.

TABLE 1. Effect of Vagotomy on Relative Volume (in %) of Gastric and Duodenal Mucosal Microflora

Time of investigation	Fundus of stomach		Pylorus		Duodenum	
	L	M	L	M	L	M
Control	94,4±0,6	5,1±0,6	90,4±0,2	9,6±0,2	94,4±0,6	5,6±0,6
After vagotomy						
30 days	86,1±0,6	14,9±0,6	73,4±3,3	26,7±3,3	67,3±0,3	32,7±0,3
P		<0,01		<0,01		<0,01
60 days	94,3±0,1	5,7±0,1	80,4±0,1	19,6±0,1	79,7±1,8	20,3±1,8
P		>0,1		<0,01		<0,01
After induction						
30 days	85,3±0,2	14,7±0,2	79,8±0,8	20,2±0,8	86,9±0,8	13,1±0,8
P		<0,01		<0,01		<0,01
60 days	90,9±0,3	9,1±0,3	81,9±0,4	18,1±0,4	90,1±0,7	9,9±0,7
P		<0,01		<0,01		<0,01
90 days	95,3±0,1	4,7±0,1	92,9±0,5	7,1±0,5	95,2±0,3	4,8±0,3
P		0,1		<0,01		>0,1
60 days after induction of ulcer and 30 days after vagotomy	94,9±0,1	5,1±0,1	65,7±0,2	34,3±0,2	86,2±1,1	13,8±1,1
P		>0,1		<0,01		<0,01
90 days after induction of ulcer and 60 days after vagotomy	91,1±0,5	8,9±0,5	82,8±0,3	17,2±0,3	94,1±0,5	5,9±0,5
P		<0,01		<0,01		>0,1

Legend. L) Lumen, M) mucosal microflora.

EXPERIMENTAL RESULTS

Light-optical studies and also investigations by the SEM and TEM showed that vagotomy leads to a marked increase in the relative volume of mucosal microorganisms in the parts of the stomach and duodenum studied (Figs. 1-4). Stereometric calculations show that the mucosal microflora in the regions of the gastric and duodenal mucosa examined in the control animals occupied the greatest volume in the pylorus, which accounted for $9.6 \pm 0.2\%$ of the relative volume. The greatest increase in volume of the mucosal microflora occurred on the 30th day after vagotomy (Table 1). The results showed that the greatest increase took place in the relative volume occupied by the mucosal microflora in the gastric pylorus. The relative volume occupied by the mucosal microflora fell 60 and 90 days after vagotomy in all parts of the stomach and duodenum investigated. Whereas the relative volume of the mucosal microflora in the fundus was actually a little lower than in the control, in the pylorus and duodenum it was significantly greater than in the control animals. The presence of a chronic gastric ulcer led to an increase in the relative volume of the mucosal microflora in all parts studied. The volume occupied by the mucosal microflora was increased particularly substantially in vagotomized animals with chronic gastric ulcers (Table 1).

The results of investigation with the TEM and SEM showed that microorganisms of the *Escherichia coli* type are found most frequently on the surface of the epithelial cells, together with various cocci (Figs. 1-3). Often at sites of close contact between microorganism and epitheliocytes, changes of a degenerative type took place in the latter, in the form of edema of the apical parts of the cells and rupture of the plasma membrane. Under these circumstances the release of secretory granules was increased. In the connective-tissue proper of the mucosa the number of eosinophils, mast cells, and plasma cells was increased. Release of secretory granules by mast cells and eosinophils and migration of these cells into the epithelium also were increased.

The investigations thus showed that the maximal volume of the mucosal microflora is found in the glandular part of the rat stomach in the pylorus. Chronic gastric ulcers cause an increase in volume of the mucosal microflora. Vagotomy leads to a marked increase in the relative volume of the mucosal microflora in the duodenum, the fundus of the stomach and, in particular, the pylorus.

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ELECTRON-CYTOCHEMICAL AND MORPHOMETRIC INVESTIGATION OF CEREBRAL CORTICAL SYNAPSES DURING POSTMORTEM AUTOLYSIS

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KEY WORDS: cerebral cortex; synapses; postmortem autolysis

A close study of the structure of interneuronal junctions in the brain after death is essential in order to elucidate early postmortem autolytic changes in the CNS. The time course of ultrastructural changes in synapses during postmortem autolysis has been described [1-3]. However, the state of the system of neurofilamentous subsynaptic units (SSU) which, as we know, plays an important role in maintaining the integrative function of synapses, was not examined in the investigations cited. The ability of the SSU to change rapidly under various influences has been demonstrated [6, 7].

The aim of this investigation was to study structural changes in SSU of cerebral cortical synapses of rats during postmortem autolysis.

EXPERIMENTAL METHOD

Experiments were carried out on 12 male albino rats weighing 170-230 g, anesthetized with ether, at a temperature of 20°C. Three intact animals served as the control, and nine rats were killed by clamping the intubation tube. Cardiac arrest took place 5 min 30 sec after the beginning asphyxia. The brain was perfused with a mixture of 4% formaldehyde solution and 1% glutaraldehyde solution in phosphate buffer, pH 7.4, with sucrose (5%). Pieces of sensorimotor cortex for orientation purposes were embedded in a plane-parallel arrangement in Araldite 30 and 90 min and 6 h after death. To reveal paramembranous synaptic concentrations of neurofilaments, at the dehydration stage in 100% ethanol the material was stained in a 5% solution of phosphotungstic acid (PTA) for 3 h. Ultrathin sections of the molecular layer of the neocortex were cut in a tangential plane and studied in the EMV-100LM electron microscope. Twenty random fields of neuropil from one animal were photographed under standard magnification of 15,000. Morphometry was carried out on negatives on a "Belarus'-2" enlarger with final magnification of 30,000. The total number of PTA-positive contacts and the number of definite and indefinite synapses, symmetrical relative to the SSU system and of asymmetrical contacts per 100 μ^2 of neuropil were determined. Depending on the abundance of dense projections (DP) of the presynaptic grid (PG) all the definite synapses were divided into four types: A, B, C, and D [5]. Synapses with a straight and curved active zone of contact (AZC) also were distinguished. The numerical results were subjected to statistical analysis.

EXPERIMENTAL RESULTS

On staining of the cortex with PTA mainly a paramembranous specialization of the neurofilaments of interneuronal synapses was observed in the form of SSU (Fig. 1a). In asymmetri-

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