THE ROLE OF GASTROINTESTINAL HORMONES IN THE PATHOLOGY OF THE DIGESTIVE SYSTEM

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Annotation: *The functioning of the digestive system, the conjugation of motility, secretion and absorption are regulated by a complex system of nervous and humoral mechanisms. There are three main mechanisms of regulation of the digestive apparatus: central reflex, humoral and local. Gastrointestinal hormones play an important role in the humoral regulation of digestive functions. These substances are produced by endocrine cells of the mucous membrane of the stomach, duodenum, pancreas and are peptides and amines. Gastrointestinal hormones are involved in the regulation of secretion, motility, absorption, trophism, release of other regulatory peptides, and also have general effects: changes in metabolism, activity of the cardiovascular and endocrine systems, and eating behavior.*

Keywords: *regulatory peptides, metabolism, endocrine systems.*

Gastrin is synthesized by G-cells located in the mucous membrane of the antrum of the stomach (in the middle zone of the pyloric glands) and in the crypts, villi, Brunner glands of the duodenum. In the catabolism of gastrin, the small intestine and kidneys play a significant role, and the liver plays a much less important role in the degradation of natural gastrin. Along with the main type of action of gastrin on the secretory activity of the stomach - by direct stimulation of the parietal and chief cells after binding to their receptors - in recent years, the effect of gastrin mediated by the central nervous system on the functions of the stomach has been discussed.

Almost all researchers do not doubt the prevailing role of the endocrine type of the mechanism of action of gastrin, i.e. direct influence of gastrin synthesized by G-cells and entered into the blood on target tissues (stomach, pancreas). An increase in intragastric pH is a physiological stimulus for gastrin incretion.

Gastrin and its synthetic pentapeptide (pentagastrin, reproducing essentially all the effects of antral hormone) significantly increase the functional activity of the mass of parietal and chief cells of the fundic mucosa, cause an increase in the debit of hydrochloric acid and pepsin, depending on the rate of incretion of the endogenous

hormone or the dose of gastrin (pentagastrin) administered from the outside . Since the blood supply to the gastric mucosa largely ensures its functional activity, it should be noted that not only in animal experiments, but also in human studies, the regular increase in blood flow in the fundic part of the gastric mucosa with pent gastrin should be noted. Gastrin enhances the flow of prostaglandin E2 into the gastric juice both in animals and in humans after the administration of gastrin or pent gastrin.

This fact complements the information about the trophic effect of gastrin on the gastric mucosa. Gastrin and pent gastrin increase the tone of the lower esophageal sphincter, enhancing the barrier function of this barrier to gastroesophageal reflux. The trophic effect of gastrin on the exocrine tissue of the pancreas has been shown. With intravenous administration of gastrin and pent gastrin to animals and humans, a significant increase in the concentration and flow rate of pancreatic bicarbonates and enzymes is noted. According to the Dnipropetrovsk Research Institute of Gastroenterology (1977), gastrin and pent gastrin have analgesic and antiasthenic morphine-like effects in diseases of the digestive system, lasting from 5 hours to 2-3 days after intravenous, intramuscular, intranasal or sublingual administration of the drug.

With hypergastrinemia develops: a tumor of the islets of Langerhans of the pancreas; a sharp increase in the secretion of hydrochloric acid by the stomach; diarrhea (due to the formation of an acidic environment in the duodenum, unfavorable for the action of pancreatic and intestinal enzymes; inhibitory effect of gastrin on the absorption of water and salts in the small intestine; gastric metaplasia in the mucous membrane of the small intestine; multiple gastroduodenal ulcers, often accompanied by hemorrhages, perforation, penetration into neighboring organs; Violation of gastrin incretion is noted in chronic gastritis, chronic duodenitis, gastric and duodenal ulcers, dumping syndrome and some other diseases of the gastrointestinal tract.

Back in 1928, Ivy (Ivy) and Oldberg (Oldberg) designated the term "cholecystokinin" extractable from the intestinal mucosa hormonal factor that causes contraction of the gallbladder. Fifteen years later, Harper and Raper reported that an extract from the small intestine mucosa stimulated the secretion of pancreatic enzymes and named the hormone pancreozymin responsible for this effect. Classical studies on the purification of cholecystokinin and pancreozymin preparations carried out in 1964 (Jorpes, Mutt) revealed their structural identity: this led to the designation "cholecystokinin-pancreozymin". The hormone is found in the endocrine I-cells of the duodenal, jejunal and, to a much lesser extent, ileal mucosa, and is naturally detected in the brain. Its molecule consists of 33 amino acids. The leading effects of HCP are a powerful increase in gallbladder motility and a significant stimulation of pancreatic secretion of enzymes. The relaxation of the sphincter of Oddi, synchronous with the contraction of the gallbladder, after the introduction of CCP intravenously or intraduodenal administration of implementers of endogenous CCP incretion (fatty and peptide components of food, as well as bile acids) contributes to the flow of bile into the

duodenum. HCP-stimulated pancreatic enzymes are also secreted there, creating optimal conditions for the breakdown of food Without affecting the pancreatic release of bicarbonates by itself, HCP potentiates (albeit moderately) the specific stimulating effect of secretin on this process.

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