lipid complex of the superficial gel. The thickness and viscosity of gel is hereby reduced despite its increased production. **HP is a strong, possibly exclusive source of gastric urease**. The latter catalyses the hydrolysis of urea, thus producing alkaline microenvironment which protects the mucosa from the effect of gastric acid.

Helicobacter pylori is an extremely world-wide spread infection. However, it brings about gastritis or ulcerative disease only in a small group of infected persons. On the basis of its limited incidence it is possible to state that the disease develops when supported by the presence of certain decisive factors. Unconfirmed, but still appealing are the hypotheses explaining the liability to succumb the disease on the basis of more aggressive HP strains, higher bacterial density, or exaggerated response of organism. A certain critical density of HP is assumedly inevitable to enable the neutralisation of its surrounding by urease and generation of sufficiently strong chemotactic signals supporting the active inflammation. The impacts of acid, pepsin and other impairment-inducing factors are of secondary importance.

Helicobacter pylori is alleged to be responsible for a majority of chronic active gastritis. Histologically it is moderate superficial gastritis. The superficial area of lamina propria comprises neutrophils and mononuclear leukocytes. Children develop lymphoid hyperplasia. The most common sign resides in a moderate reduction of mucus.

There is a link between the peptic ulcer and HP. The presence of HP in the antrum was found in 90 % of patients with duodenal, and 70 % of patients with gastric ulcer. HP is considered to be an important factor in the pathogenesis of peptic ulcer. However, it still remains to be clarified as to whether HP represents the causal factor of the development of peptic ulcer, or a merely auxilliary and modificating factor.

7.6 Exocrine pancreas

The pancreas is localised retroperitoneally behind the stomach. The pain coinciding with pancreatitis therefore spreads from the epigastric area into the back. The pancreatic head is in the duodenal flexure and its tail touches the spleen. Pressure on the spleenic vein (vena lienalis) can cause varicous dilatation of gastric veins. An impaired drainage of the lymph from the stomach can be caused by e.g. reactive inflammation of the pancreas. The pancreas has an outstanding position among glands in organism as it comprises both endocrine and exocrine glands.

The exocrine pancreas consists of acini which produce enzymes and alkaline fluid. Acinal cells are arranged spherically around small outlets. Pancreatic juice is drained via the major pancreatic duct which enters the duodenum together with the common bile duct (ampulla Vateri). **Anatomical conditions** in this area vary. In some individuals the pancreatic juice is drained by the Santorini's duct which is independent from the major pancreatic duct, and enters the duodenum via the minor duodenal papilla. The secretory pressure in the efferent ducts reaches approximately 200 mm H_2O. The lumen of ducts is lined with one-layer epithelium which is permeable neither for bile and trypsin, nor for E. coli suspension. At rest, the pancreas secretes approximately 2 ml of juice, after stimulation its amount reaches 5 ml. The insular apparatus is localised predominantly in the pancreatic tail. It is separated from the exocrine parenchyma by connective tissue. Endocrine pancreatic agents get into circulation via portal veins.

The arterial blood is brought into pancreas via branches of the truncus coeliacus and from the arteria mesenterica superior. The venous blood is drained from the pancreatic head into the portal veins and from the pancreatic tail into the spleenic vein.

Postganglionic fibers stimulate the enzymatic and hormonal secretions. The sympathetic postganglionic fibers from the coeliac ganglion and superior mesenteric plexus innervate the blood vessels and stimulate vasoconstriction. In general, the sympathetic nervous system inhibits the pancreatic secretion and the parasympathetic nervous system stimulates it.

Pancreatic juice is isoosmotic. It contains potassium, sodium, the most important anions include bicarbonates and chlorides which participate in a reverse ratio (an increase in bicarbonates causes a decrease in chlorides and vice versa). The potassium and zinc levels in the pancreatic juice is twice as high as those in the serum, the contents of proteins cor-

relates with the contents of enzymes. A markedly alkaline pancreatic juice neutralises the acid duodenal contents of the stomach. The alkaline environment is optimal for the impact of digestive enzymes. The secretory cells in acini transport H^+ into blood and bicarbonates into the acinal lumen. Bicarbonates are transeferred into acini by active transport. Potassium and chlorides are excreted by diffusion. Water transgresses into acini iso-osmotically. Slow production of pancreatic juice brings about an exchange of bicarbonates to chlorides. In consequence of fast production there is not enough time for this exchange and therefore the juice is more alkaline. This is brought about during the intake of food.

Pancreatic enzymes (trypsin, chymotrypsin and carboxypeptidase are able to hydrolyse proteins, saccharides, and fat. These enzymes are secreted in acini and transported into the duodenum in an inactive form (trypsinogen, chymotrypsinogen, and procarboxipeptidase). Pancreas must be protected from the impact of the organism's own enzymes. Therefore the pancreas produces an inhibitor of trypsin which hinders the activation of proteolytic enzymes, providing they are present in the pancreas, or in its draining ducts. As soon as the pancreatic juice reaches the duodenum, the proenzymes are activated by enterokinase which is produced by duodenal mucosa: enterokinase activates the conversion of trypsinogen to trypsin. Trypsin in turn stimulates the conversion of chymotrypsinogen to chymotrypsin and the transformation of procarboxipeptidase to carboxipeptidase. Each of these enzymes splits specific peptide bonds and reduces polypeptides to smaller peptides. **The central role in activation of enzymatic precursors is performed by trypsin** which activates not only the precursors of other proteases, but yet it is able to activate itself (autocatalysis). Trypsin splits also its own inhibitory complex in the small intestine and in this way it activates itself. This inhibitory system procures the protection of ducts from autodigestion.

The pancreatic alpha-amylase is secreted in an active form. The optimal pH for alpha-amylase is approximately 6.9. The pancreatic lipase hydrolyses triglycerides to free fatty acids and monoglycerides. The pancreatic juice includes also cholesterol-esterhydrolase and phospholipases A and B. These enzymes have its optimum pH ranging from 7 to 9.

Enzymatic secretion of acini is maintained and stimulated by cholecystokinin and acetylcholine. Cholecystokinin is released in the duodenum. Its release is stimulated by amino acids and fatty acids which are present in the gastric chyme. The activation of pancreatic enzymes in the duodenum and small intestine hinders the release of cholecystokinin. In this way, a feedback involving several pancreatic enzymes is applied. Acetylcholine is released from the pancreatic branches of the vagus nerve. An increased release of acetylcholine takes place during the cephalic phase of digestion. The pH of gastric chyme (pH 3.5–4.5) stimulates S-cells (secretin-producing cells) to release secretin. Secretin is absorbed in the intestine and transported to the pancreas by blood. Secretin within the pancreas stimulates the duct and acinar cells to secrete alkaline fluid. The effects of secretin include also an inhibition of gastrin which results in a decrease of gastric secretion and motility.

7.6.1 Classification of pathological states of the pancreas

1. Impairment of the exocrine function of pancreas
2. Inflammatory diseases of pancreas
   - acute pancreatitis
   - recurrent acute pancreatitis
   - chronic pancreatitis
   - recurrent chronic pancreatitis
3. Tumours of pancreas
   - tumours of draining ducts
   - tumours originating from acinous cells
   - tumours of pancreatic islets
4. Traumatic impairment of pancreas
   - blunt non-penetrating injuries
   - penetrating injuries
5. Diseases of genetic origin
   - cystic fibrosis
   - hereditary pancreatitis