of the oesophagus are therefore weakened and this condition is accompanied by incompetence of the lower oesophageal sphincter. **Achalasia** is a condition caused by ganglionic cellular degeneration of the Auerbach's plexus. Consequently, this degeneration decreases the tension and deteriorates the relaxation of the lower oesophageal sphincter. The peristalsis is frequently absent as well. The particular cause of the diffuse oesophageal spasms is not known. Application of immunosuppressive substances can bring about oesophageal infection by herpes virus or by Candida albicans. Radiation oesophagitis appears in coincidence with the therapy of mediastinal and pulmonary malign processes. It is more pronounced in combination with chemotherapy.

7.4 Stomach and duodenum

Owing to the gastro-oesophageal and pyloric sphincters, the stomach can retain the received food for the period which is necessary for the small intestine to become prepared to digestion. During this stage of gastric storage, the food is mixed with gastric juice. Thereafter it is released into the duodenum in small bulks.

The structure of gastric mucosa is highly specialized. The **blood supply** is performed via branches of the arteria coeliaca. The largest arteries are localised along the major and minor curvatures. The stomach possesses a rich collateral circulation. Assumedly, this fact aids to the origin of ischaemic changes in the gastric wall. The venous blood is drained away from its right half into the vena gastroepiploica dextra (vena mesenterica superior) and the blood from the left half of the stomach is drained into the vena gastroepiploica sinistra and venae gastricae breves (vena lienalis). The sympathetic and parasympathetic innervation systems are subordinated to local effects and are controlled by brain centres. The muscular layer of the gastric mucosa (muscularis mucosae) contains "plexus submucosus Meissneri" and the muscular layer of the stomach (tunica muscularis) contains a "plexus myentericus Auerbachi". The subserous tissue contains a "plexus subserosus".

An **empty stomach** contains approximately 50 ml of fluid. The tension of its walls is low. At the beginning, the intake of food causes relaxation of the gastric fundus. This relaxation is perfectly coordinated. This so-called receptive relaxation is positively affected by gastrin and cholecystokinin. Peristaltic movements proceed from the fundus to the antrum. Usually, three peristaltic waves are observed per minute. Gastrin and the vagus nerve enhance the contractions. Cholecystokinin hinders gastric motility and consequent evacuation of the stomach. The velocity of peristaltic waves is assessed by muscular cells which act as pacemakers.

The mixing of food and evacuation of the stomach takes several hours. When the bolus gets to the antrum, the contraction becomes stronger. If the pylorus does not open, the chyme returns to the gastric corpus. This activity is referred to as retropulsion and enhances proper mixing of food. Each peristaltic movement results in shifting a small bulk of prepared chyme via the pylorus into the duodenum. The pylorus, in its narrowest portion is 1.5 cm in length. This portion contains a permanent aperture, only 2 mm in diameter.

Evacuation of the stomach is a complex process which depends on the conditions within the stomach and duodenum, including the chemical conditions. The first products of fat digestion together with bile and pancreatic juices stimulate the secretion of cholecystokinin. The duodenum contains osmoreceptors which are sensitive to the changes in osmotic pressure in the duodenal contents. Both hyperosmotic and hypoosmotic gastric chymes delay the gastric evacuation in order to maintain the isoosmotic conditions within the duodenum. Secretions of the duodenal mucosa, pancreas and liver neutralise the acid gastric contents within the duodenum. Evacuation of the stomach is subordinated to these conditions.

The ingestion of food stimulates the **secretion of gastric juice**. The gastric mucosa produces various substances. Parietal cells of the gastric mucosa produce hydrochloric acid and the intrinsic factor. The chief or central cells form pepsinogen. The composition of gastric juice depends on the velocity of its production. Slow production results in low concentrations of hydrogen and chlorine ions, and the concentration of sodium ions is high. Fast production brings about reverse conditions. Potassium is always released into the gastric juice in an amount exceeding

its concentration in the plasma. In general, the gastric secretion in the morning is low and increases in the afternoon. It also decreases due to disagreeable odour or taste, anger, fright and pain. These sensations and emotions are applied via the sympathetic impulses and by inhibition of the parasympathetic impulses. Inversely, aggressivity and hostility induce an increase in secretion thus participating in the origin of pathological changes in the stomach.

The **main task of the gastric acid** is to dissolve food fiber, to extinguish bacteria brought into the gastrointestinal tract together with food and to convert pesinogen to pepsin. The production of hydrochloric acid by parietal cells is principally based on the transport of hydrogen and chlorine ions into the lumen of gastric glands. High secretion of acid causes that the bicarbonates originating within the production of hydrogen ion, return into the venous blood. This process can become manifestant as late as by alkaline reaction of urine.

Gastric secretion is stimulated by acetylcholine, gastrin and histamine. The vagus nerve releases acetylcholine and stimulates the secretion of gastrin. Histamine is stored in enteroendocrine cells of the gastric mucosa. The gastric mucosa contains also histamine receptors (H_2). Antagonists of H_2 receptors can significantly decrease the secretion of gastric acid in patients with gastric ulcer. Acetylcholine and gastrin stimulate the chief cells to release pepsinogen during meal. The conversion of pepsinogen takes place as soon as pH drops below 5.0. The optimal pH value for conversion is 2.0. When the gastric content reaches the alkaline environment of the duodenum, the effects of pepsin are inactivated.

The gastric mucosa is protected from autodigestion by mucus. The adhesion of mucus to gastric epithelial cells is very tight. The mucous barrier is impermeable for gastric acid. Prostaglandins participate in the formation of the barrier by stimulating the production of mucus and bicarbonate, and by inhibiting the secretion of gastric acid. An impairment of the barrier (by e.g. aspirin, ethanol, regurgitated bile, or ischaemia) can lead to inflammation and ulcer development.

The secretion of gastric acid is liable to a large variety of effects. The **cephalic phase** is based on sensory stimuli – smell, sight, and taste of food, the process of mastication and deglutition. The cephalic phase is mediated by vagal activation via the myenteric plexus. The release of acetylcholine stimulates parietal and chief cells to produce HCl and pepsinogen. The G-cells in the antrum release gastrin into the blood. The transport of gastrin into the gastric glands thus stimulating the secretion of gastric acid. The secretion of gastric acid precedes the arrival of food into the stomach. Insulin represents another strong stimulus of gastric secretion. Owing to insulin, the secretion of gastric acid is mediated via the vagus nerve from a special hypothalamic sensor. This sensor reacts to a decreased level of glucose caused by insulin. The recovery of the glucose level to the standard suppresses the gastric secretion caused by insulin.

The **gastric phase** of stomach secretion begins as soon as the food arrives into the stomach. The arrival of food represents the strongest stimuli for secretion. Basically, they are the distension of the stomach and the presence of digested proteins which activate the process. The distension of the stomach activates the mechanoreceptors, and the stimulation of secretion is performed by the vagus nerve. The activation is mediated by acetylcholine. Therefore it can be blocked by atropine. Protein chains which get into contact with the pyloric mucosa stimulate the release of gastrin from G-cells in the antrum. An increase in gastric pH up to 3.0 causes an inhibition of the gastrin release.

The release of gastrin is inhibited also by somatostatin which is produced in the antral gastric mucosa by endocrine cells (D cells). Somatostatin reduces gastric acid secretion by inhibiting gastrin release, and at the same time it directly inhibitits the secretion of the neighbouring parietal cells.

The **intestinal phase** of gastric secretion is induced when the chyme reaches the duodenum. The gastric secretion in this phase is stimulated by absorption of amino acids in the small intestine. A reverse situation, i.e. a sufficient amount of acid chyme in the duodenum inhibits the gastric secretion and gastric motility. The acid reaction of chyme in the duodenum stimulates the release of these intestinal peptides which via the blood inhibits the secretion of gastric acid thus restricting the intestinal phase of gastric secretion.