

quent bulks of chyme from the stomach into the distended small intestine is inhibited. The intestinointestinal reflex procures the hindrance of intestinal motility in cases when some part of the intestine is extremely distended. The gastroileal reflex arises in coincidence with increased gastric motility and secretion. It causes an increase in the motility of ileum. In this way, a more rapid evacuation of the ileum is procured, in order to continue the acceptance of more chyme. The gastroileal reflex is probably regulated by neurotensin. During long-term fasting, or sleep, a slow contraction proceeds from the stomach to the terminal ileum. In this way this reflex procures the evacuation of gastric and intestinal contents into the large intestine. The gastrocolic reflex (urgency to defecate after meal) is pronounced especially in childhood.

The ileocecal junction is procured by a valve (sphincter) which is enclosed at rest. Its opening is stimulated by peristaltic waves. After the transition of a small part of chyme it encloses again. This mechanism inhibits both regurgitation of chyme and extreme distension of the large intestine.

7.9 The large intestine

The large intestine is approximately 1.5 m in length. It is constituted by the caecum, appendix, colon (ascendent, transverse, descendent, sigmoid), rectum and the anal canal. The caecum accepts the chyme from the ileum. The vermiform appendix might have its significance in elimination of unusual components of food present in the bowel. The sphincter in the distal part of the sigmoid colon controlling the transport of chyme into the rectum is in comparison with the ileocecal sphincter less demarcated. It is an intestinal part which fulfils the task of a sphincter of a longer section. The anal canal encloses the internal sphincter constituted by smooth musculature and the external sphincter which is constituted by striated muscles.

The longitudinal musculature of the large intestine constitutes three stripes referred to as colic taeniae (taeniae coli). The fact that they are shorter than

the bowel thus determines its typical shape. The circular musculature is separated into sections which are referred to as haustra. Muscular contractions reduce and enlarge the haustra.

The large-intestinal mucosa contains cylindrical cells which absorb water and electrolytes. Mucus-producing cells create a protective layer on the mucosal surface.

The **motor and secretory activities** of the large intestine are regulated from the myenteric plexus which is to a great extent independent from other systems. The parasympathetic innervation of the caecum, ascendent colon and the first part of the transverse colon originates from the vagus nerve which in this area of the large intestine stimulates rhythmic contractions. The distal colon is parasympathetically innervated from the pelvic nerves. The internal anal sphincter is innervated by the sympathetic nervous system (the sympathetic system enhances contractions) and parasympathetic system (the parasympathetic system supports relaxation). It is permanently in state of contraction. It relaxes only for a short time in coincidence with the distension of the rectum and at defecation. The external sphincter is innervated by sacral branches of spinal nerves. The sympathetic innervation originates in the coeliac and superior mesenteric ganglia. Destruction of the lower part of medulla spinalis paralyses the external sphincter whereas the internal sphincter does not respond. The sympathetic activity in the area of the large intestine modulates intestinal reflexes, somatic sensations and pain.

Basic movements of the large intestine are represented by **segmental contractions** which always involve an entire haustrum. **Peristaltic movements** involve several haustra. They procure the transition of the faecal mass aborally to the rectum. The gastrocolic reflex enhances peristaltic movements of the large intestine after the stomach filling, which stimulates defecation. It is probable that gastrin participates in this reflex.

Fluid is absorbed in the large intestine by diffusion or by active transport. Under the influence of aldosteron, the diffusion of sodium into the cells and its active transport through the basolateral membrane into the interstitial fluid increase. Together with sodium, also chlorides – as complementary anions – are absorbed. They enter the cells on the basis of exchange for bicarbonates. The active transport

of sodium currently increases the gradient for the diffusion of potassium from cells into the lumen. The substances being absorbed in the large intestine include also the chains of fatty acids which are produced during fermentation. Neither saccharides, nor amino acids are absorbed in the large intestine.

Under physiological circumstances, the gastric contents, owing to the presence of HCl, is almost sterile. The bacterial reproduction in the duodenum is inhibited by bile and antibodies. Duodenum contains only a minimal amount of aerobic, and no anaerobic microbes. Anaerobic microbes harbour the more remote part of the ileum – the ileocecal area. The intestinal flora mostly includes *Escherichia coli*, *Clostridium Welchii* and streptococci. Bacteria significantly participate in the metabolism of bile acids, namely in the reabsorption of bile components, elimination of toxic metabolites from bile and the breakdown of fibrous material. The intestinal bacterial flora participates in the metabolism of oestrogens, androgens, lipids, various nitrogen substances and drugs.

7.10 Malabsorption

The main function of the gastrointestinal tract is to digest and absorb the nutrients serving as the source of energy. In addition to the latter, an organism needs small amounts of other significant substances for the optimal procurement of specific functions.

In a precise pathophysiological sense, the term **malabsorption** refers to a defective absorption of nutrients by the intestinal mucosa, whereas an impaired hydrolysis of nutrients is referred to as **maldigestion**. However, as the processes of digestion and absorption are very closely associated, all aspects of impaired digestion and absorption are in clinical practice commonly referred to as malabsorption syndrome.

7.10.1 Inadequate digestion (maldigestion)

The malabsorption syndrome often develops in con-

sequence of defective digestion. The accepted food must be initially processed down to basic components. These activities enable further steps to be carried out with subsequent resorption. The process takes place within the lumen, on the mucosal surface (contact digestion). A significant role in the process of digestion is carried out by the pancreas. It produces lipase, colipase and proteases, especially trypsin. Therefore malabsorption is the cardinal symptom of chronic pancreatitis. In order to procure the optimal digestion of fat, the chyme must contain a sufficient amount of bile acids which are necessary for normal lipolysis and formation of micelles. A decreased production of bile salts can develop in consequence of its decreased synthesis in the liver, however more often it is caused by cholestasis in coincidence with hepatic cirrhosis. A further cause resides in an increased deconjugation of bile acids caused by bacterial overgrowth.

Bile acids deficiency may be caused also by diseases of the ileum or ileal bypass by leading to insufficient recirculation of bile acids. The liver in these cases is not able to synthesize bile acids *de novo* in a sufficient amount. This condition results in **malabsorption of fat** and deficiency of fat-soluble vitamins. These global disturbances can be simultaneously present in specific defects of digestion. Thus, e.g. lactase deficiency on the surface of enterocytes deteriorates the absorption of saccharides. This condition can result in intolerance to milk and clinical symptoms as flatulence, intestinal distension and diarrhea. It occurs more frequently in black people. It can occur as a consequence of a diffuse defect of mucosa in coincidence with other diseases.

7.10.2 Inadequate absorption (malabsorption)

The inadequate absorption can appear in normal intact digestion. Most frequently it is caused by a reduced surface for absorption of substances. It develops after intestinal resection due to mesenteric infarction, bypass due to morbid obesity, or due to Crohn's disease.

In other cases, the surface necessary for absorption may be sufficiently large, but insufficiently involving the processes of absorption *per se*. These impairments are caused by the defects in mucosal cells (inborn or acquired). Examples of this condi-