the shape of a cast of the intestinal lumen. The cause of this state is not clear. Hypersecretion of mucus occurs in vegetatively stigmatized individuals. Some consider it to be an impact of allergy.

The factors leading to the onset of irritable bowel syndrome are classified as local or overall. The overall factors include an increased vegetative irritability and instability. The corticovisceral theory explains its commencement as being a consequence of functional disturbances, cortex-subcortex relations with an origin of parasympathotonia resulting in spasms, hypermotility and hypersecretion.

Out of the locally effective factors, the influence of food must be taken into consideration, as well as that of laxatives, or other drugs, and the reflexive impact of the surrounding tissues.

7.12.3 Congenital and acquired megacolon

The congenital megacolon occurs in childhood. It is characterized by an enormously distended large intestine, and constipation. Children have a large, tympanic abdomen. Stool is absent for days, even weeks. Evacuation of bowels is seldom, but the weight of stool may attain the amount of 10 kg.

The disease was described for the first time by Hirschsprung. He believed that the disease involved a congenital intestinal anomaly, e.g. abnormal plicae which enclose the intestine in a valvular manner. Later it was believed that the predominance of the sympathetic innervation decreases the intestinal motility with the development of the above described clinical picture.

The current conception apprehends that this condition involves a state which is analogic to achalasia of the cardia. It was found out that the afflicted intestinal segment (most frequently the sigmoid colon and rectum) is narrower in comparison with the rest of the intestine due to congenital absence of the intramural neural plexuses. The intestine above the afflicted portion is dilated and its wall is hypertrophic. Such defect of the autonomic innervation is manifestant by an inability to arise peristalsis within this portion, thus causing functional obstruction. It seems that this disease is incompatible with life. If it is not treated by replacement of the rectum and the lower part of the sigmoid colon, ileus develops with subsequent ulceration and perforation. Currently we distinguish:

1. **Megacolon congenitum** (Hirschsprung's disease) is caused by the absence of ganglia in the rectal Meissner's and Auerbach's plexuses. The aganglionic segment is most frequently in the area of the junction of sigmoid colon with rectum. This **aganglionic segment is narrowed** down, the peristaltic movements cease before it, while the segment lying proximally to it (in the oral direction) is dilated and its wall is hypertrophic. This condition can even lead to a subileous state, resorption impairment, vomiting and weight loss.

2. **Idiopathic megacolon** is difficult to be distinguished from the congenital form. It is a disease assumedly acquired in consequence of inappropriate defecatory habits and the main role is played by psychogenous factors. The rectum is overfilled with stool, and its distension extends as far as to the anal sphincter.

3. **Symptomatic megacolon** develops in consequence of acquired organic disorders, e.g. narrowment of the rectum due to its strictures entailed by injury, infection, etc.. It also can intervene in consequence of dilatation developed due to ulcerative colitis.

7.13 Intestinal obstruction

7.13.1 Terminology, classification, clinical impact

Intestinal obstruction refers to a **situation when the intestinal contents cannot be forced further in aboral direction**. Transit of intestinal content depends not only on an intact state of intestinal lumen, but also on peristalsis. Intestinal obstruction can be therefore caused by two principally distinct mechanisms:

- **mechanical obstruction** (occlusion, obturation) of the intestinal lumen, or

- **paralysis of the intestinal musculature** (intestinal pseudoobstruction). The clinical symptomatology of this state yields symptoms of a severe obturation, however, it is impossible to detect the mechanical obstacle disabling the propulsion of the intestinal contents.

Both mechanisms (mechanical obstacle and inhibition of intestinal motility) can take place independently, subsequently, as well as simultaneously. They can cause a partial or complete obstruction which in turn can be of temporal or permanent character, acute or chronic. A simple mechanical obstruction (single obstacle in single site) is rarely complicated by strangulation, i.e. impairment of mesenteric vessels and nerves. However strangulation is a common complication if the bowel is occluded at least in two sites (obstruction of an enclosed loop) as e.g. in incarcerated hernia or volvulus. The obstructed intestinal loop can be evacuated neither in aboral direction, nor by progressive dilatation of the bowel proximal to the obstacle. The enclosed intestinal loop develops high intraluminal pressure, which in turn decreases the inflow of arterial blood and hinders the venous outflow. In cases of pseudoobstruction, strangulation can develop secondary to the compression of mesenterium by the distended bowel. If the obturation lasts for a period which is sufficiently long to enable an impairment of mucosa by autodigestion, a **severe state is developing which is even now incurable – the ileus**. This clinical syndrome is characterized by **intestinal inactivity, autodigestion of the intestinal mucosa and breakdown of the internal environment**.

Regarding its causes, ileus can be classified as being post-obstructional when caused by a mechanical obstruction, and pseudo-obstructional when developed in consequence of the cessation of intestinal peristalsis. Keeping its pathogenesis in mind, ileus is an irreversible, incurable clinical syndrome. Hence, great attention must be paid to early diagnosis and treatment of all states which enable its development. Mechanical intestinal obstruction frequently induces an acute abdominal pain and requires urgent examination which is often immediately followed by surgical intervention.

The identification of the cause of intestinal obstruction is not always simple. Owing to the fact that individual pathomechanisms of intestinal obstruction require principally distinct therapies, early distinguishment of the mechanical and paralytic forms is of great value. Regarding the quantitative aspect, mechanical obstruction represents a severe problem as it is the cause of approximately 20 % of all urgent cases where surgical intervention is necessary. 95 % of cases yield an affliction of the small intestine, most frequently in consequence of abdominal adhesions. Abdominal adhesions can develop spontaneously, or in consequence of intraperitoneal inflammation. The majority of adhesions causing intestinal obstruction is however caused by preceding operations. Approximately 5 % of abdominal operations are complicated by intestinal obstruction, in a number of cases as late as some years after surgery. Post-operative scars detectable by inspection are therefore a significant diagnostic indicator. Most frequent causes of the small intestine obstruction include internal and external hernias, and tumours. Mechanical obstruction of the large intestine occurs more frequently in the elderly population, and is caused by tumours, inflammatory processes and volvulus.

Historical aspects

First documentation describing the clinical symptoms associated with intestinal obstruction are dated far back to the past. They can be found, e.g. in the studies of Hippocrates. The success rate of therapy, either surgical (Praxagoras, 350 B.C., enterocutaneous fistula) or conservative (reposition of incarcerated hernias, analgetic therapy by opium, mercury or lead p.o. aimed at reopening of the enclosed intestine, gastric irrigation, etc.) was until the 20th century minimal. **Had ileus developed in consequence of intestinal obstruction, the mortality rate reached 100 %. An improvement took place after 1912**, when Hartwell and Hoguet noticed that infusion of the physiological solution prolonged the survival of dogs with intestinal obstruction. Infusion is even nowadays a part of the therapy of intestinal obstruction. The second chief therapeutic principle – suction by nasogastric or intestinal tubes – was gradually introduced into clinical practice in the 1920's (McIver, 1926). Antibiotics began to represent a standard part of therapy algorithms of intestinal obturation in the 1940' and 1950's. Infusion therapy, intestinal suction, antibiotics and somatostatin together with improved surgical and anaesthetic techniques have substantially reduced the mortality rate, especially that of the simple mechanical obstruction. Other causes of intestinal obstruction, such as strangula-

tive intestinal obstruction or generalised shock have even now a very bad prognosis and remain a serious clinical problem.

7.13.2 Etiology

The **causes of mechanical intestinal obstruction** can be localised in the intestinal lumen (polypoid tumours, intususception, bezoars, meconium, bile stones, etc.), intramural (congenital anomalies – e.g. atresia, stenosis, Meckel's diverticulum, tumours, post-inflammatory structures – e.g. in Crohn's disease, radiation enteritis, chronic intestinal ischaemia, lesions induced by KCl enteral tablets, posttraumatic states – e.g. duodenal haematoma), or in the intestinal surroundings (congenital, but especially post-operative adhesions, incarcerated external hernias, internal hernias, volvulus, compression of intestine by abscess, tumour, etc.). **Acute pseudoobstruction** can accompany the conditions after laparotomy and orthopedic operations, diseases of abdominal organs (intestinal ischaemia, pancreatitis, pyelonephritis, peritonitis, intraperitoneal abscess), thoracic organs (pneumonia, acute myocardial infarction), and overall diseases (sepsis, shock, polytrauma, decreased level of plasma potassium). **Chronic pseudoobstruction** is caused by primary (familial visceral myopathies), or secondary (collagenoses, muscular dystrophies, amyloidoses, radiation impairment) diseases of the smooth intestinal muscles, diseases of the myenteric plexus (familial visceral neuropathy, paraneoplastic degeneration, Chagas' disease, intestinal agangliosis, neuronal intestinal dysplasia, myotonic dystrophy), diverticulosis of the small intestine, coeliac sprue, jejunoileal bypass, some neurologic diseases (Parkinson's disease, tumours in the brain stem), endocrine and metabolic disorders (myxoedema, feochromocytoma, hypoparathyreoidism, acute intermittent porphyria), and drugs (opiates, phenothiazines, clonidine, tricyclic antidepressive drugs, vincristine).

7.13.3 Pathogenesis

Initial pathogenesis and clinical manifestation of intestinal obstruction vary as they are determined by mechanism, localisation, and the stage of obstruction, actual state of the gastrointestinal system and organism per se. Each intestinal obstruction impairs in a specific manner the intestinal transport, secretion, digestion, absorption and immunity functions. In addition, **intestinal obstruction generally leads to hypoxia of the intestinal mucosa** which represents the primary mechanism in the pathogenesis of ileus. Hypoxia decreases the resistance of intestinal mucosa and thereby enables autodigestion, which is the secondary mechanism leading to ileus.

The correct function of intestinal mucosa is determined by a continuous supply of oxygen, either by blood, or by aerophagia (alimentary respiration). Bearing this in mind, hypoxia of intestinal mucosa can be formally caused by intestinal hypoperfusion, hypoxaemia and hypoventilation of the intestinal lumen.

Luminal hypoventilation represents a common pathogenetic mechanism of mechanical intestinal obstruction and pseudoobstruction. Hypoventilation is caused by every mechanical obstacle disabling the transport of intestinal contents and by all changes leading to the cessation of peristalsis.

Intestinal motility depends on the functional state of intestinal musculature, its supply by oxygen and its nervous and humoral regulation. The etiology of intestinal pseudoobstruction is therefore of heterogenous character. All impairments of the contractile apparatus of the intestinal smooth musculature and excitation-contraction coupling, local impairments of intestinal circulation, all situations associated with activation of the sympathetic system and centralisation of circulation, as well as all hypoxaemic states can halt the peristalsis, thus causing intestinal pseudoobstruction.

Negative impacts of hypoperfusion and hypoxaemia on the intestinal supply by oxygen are of two kinds. They either cause a deficit in blood-delivered oxygen within the intestinal mucosa, or decrease the intestinal motility thus leading to luminal hypoventilation. A great variability in the clinical course of mechanical intestinal obstruction and especially that of pseudoobstruction can be ascribed to the possibility of mutual compensation of oxygen supply by intraluminal air and blood, as well as to varying aggressivity of the intestinal contents depending on the amount and composition of digestive juices.

The pathogenesis of intestinal obstruction is characterised by several positive feedbacks which cause its progression, and since the moment of ileus development, also the irreversibility of this clinical syndrome.

Vitality of intestinal mucosa and primarily its absorptive function react very sensitively to the deoxygenation of the intestinal contents, especially in cases which include reduction in the delivery of oxygen by blood. Organism strives to get rid of the deoxygenated intestinal contents (vomitus, diarrhoea) and to compensate the physiological level of oxygen in the intestinal lumen by aerophagia. Failing to perform this, absorption ceases as the first of physiological functions, later the mucosa afflicted by autodigestion develops inflammation with a pronounced exudation, and finally the mucosa succumbs to necrosis. These processes impair the immunity functions of the intestinal wall, the state of which enables the development of migrating peritonitis. **Reduced absorption, increased secretion and exudation lead to the accumulation of fluid in the intestinal lumen**. Together with vomiting and reduced oral intake of food, the oedema of the intestinal wall and splanchnic vasodilatation can evoke extreme hypovolaemia, or even hypovolaemic shock. These mechanisms can deduct as much as 6 l of fluid from the circulation system, especially in cases of proximal or complete obstruction. During the acute compensatory response to hypovolaemia, a part of which resides in centralisation of circulation, the bowel is hypoperfused thus making mucosal hypoxia more profound.

In addition to hypoperfusion of intestinal musculature, **hypovolaemia causes also the hypoperfusion of respiratory muscles**. Hypoperfused respiratory muscles reduce the ventilation of the lungs, and contributes in this way to the development of pneumonia. Pneumonia intensifies the hypoxaemic condition and thereby profounds the hypoxic impairment of intestinal motility and that of mucosa. In addition to the intestinal obstruction, hypovolaemia and hypoxaemia are also important components of shock. Both these clinical syndromes therefore often mutually combine. Shock can occur as either cause or consequence of intestinal obstruction. The possibility of autodigestion and the tendency to limitate intestinal perfusion within the course of the general adaptation reaction cause that the bowel becomes liable to irreversible damage during the circulatory shock, and can influence negatively the prognosis of all hypovolaemic and hypoxaemic states.

Hypoxia of intestinal mucosa stimulates aerophagia which delivers air into bowel. However, under the condition of prolonged intestinal obstruction, aerophagia is an inappropriate mechanism, as it does not procure the exchange of gases. In addition, aerophagia gradually leads to intestinal distention. Later, when the oxygen contained in the swallowed air is depleted, hypoxia becomes more profound as the epithelial cells occur in anoxic environment of pure nitrogen. The distention per se enables the development of further positive feedbacks. The intestinal distention limitates the movement abilities of the diaphragm which has a negative impact on the exchange of gases by the lungs, thus contributing to hypoxaemia. In addition to the latter, the increased contents of gases inside the intestine brings about **intestinal angularities** which not only enclose the intestinal lumen, but also comprime the mesenterium, thus deteriorating the intestinal perfusion, and increasing the congestion of the intestinal wall.

Acid gastric juice, IgA antibodies and normal peristalsis maintain low bacterial contents (0–10^3 bacteria/mm^3) in the proximal part of the gastrointestinal tract. The bacterial count increases in the aboral direction, and in the terminal ileum it attains 109–1012 bacteria/mm^3. The effects of intestinal antibacterial mechanisms substantially decrease during the intestinal obstruction. Bacteria (primary enteric aerobic bacteria) proliferate out of control until their count reaches that in the large intestine. These bacteria can enter the mesenteric lymph and portal circuit and assumedly participate in the severe, deadly course of intestinal obstruction. **A significant role in the pathogenesis of intestinal obstruction is taken by bacterial endotoxins**. They directly incur damage to intestinal mucosa, but can also transgress the walls of the obstructed bowel. Endotoxins stimulate the elimination of vasoactive intestinal peptide, other intestinal peptides and various prostaglandins (PGI2, PGF2) which mutually cause abnormal hypersecretion in the obstructed bowel.

The intestinal myoelectrical and motor activities in pseudoobstruction are impaired primarily. The impairment brought about by the mechanical intestinal obstruction is of secondary character. The phases of hyperperistalsis and antiperistalsis are followed by a reduction in the electrical activity of myocytes and the migrating myoelectric complex is substituted by conglomerates of action potentials and contractions which are not able to bring about luminal transport.

7.13.4 Pathophysiological principles of ileus prevention

Intestinal obstruction is curable until ileus develops. Several positive feedbacks which progressively intensify both intestinal and overall impairments, disable the successful therapy of ileus. The therapy, or prevention of intestinal obstruction include therefore the measures disabling ileus to develop. Preventive antiileous algorithms are based on the pathogenesis of ileus and are aimed at the procurement of luminal ventilation, removal of digestive juices from the intestinal lumen, and the procurement of sufficient intestinal perfusion by oxygenated blood. In a majority of cases, the **mechanical intestinal obstruction** requires surgical therapy (assessment of the localisation and cause of obstruction, resection of the necrotic intestine). The significance of the preventive measures resides in the fact that an entirely resuscitated patient is able to tolerate better the state of anaesthesia and surgical intervention per se.

In some cases, especially if partial obstruction of the small intestine is involved, the intestinal transport can be renewed solely by means of conservative therapy. **Intestinal pseudoobstruction** often requires a long-term conservative therapy lasting even for several weeks, and its supplementation by parenteral nutrition. An exclusively conservative therapy of intestinal obstruction, however, increases the risk of the development of intestinal strangulation. Therefore it should be applied solely in cases when the risk of intestinal strangulation is low. Inversely, if the assumed cause of complete obstruction resides in intususception, volvulus or incarcerated hernia, it will be necessary to operate immediately, with no regard to the clinical state.

Non-standard, although frequent is the use of various antimicrobic interventions. Administration of wide-spectrum antibiotics can restrict the growth of intestinal bacteria. An analogue of somatostatin – octreotide is administered with the aim to restrict the secretion of intestinal mediators of inflammatory reaction induced by bacteria, or endotoxins.

A **primary significance of prevention** is ascribed to those techniques of abdominal operations which reduce the risk of the development of adhesions. A pronounced protective effect resides in the preservation of the omentum, inhibition of both contamination and the drying out of the peritoneum, and avoidance of extensive peritoneal ligatures.

Alterations within the internal environment evoked by dehydration or circulatory shock quickly succumb to changes; therefore it is necessary to monitor continuously the serum electrolytes, acid-base balance and haemogram. Azotaemia and a pronounced increase in haematocrit give evidence of a severe decrease in intravascular volume. Metabolic acidosis and leukocytosis are frequently present in coincidence with intestinal strangulation and advanced ileus. No biochemical parameters can be however used for unambiquous assessment of the diagnosis of intestinal strangulation.

OVERVIEW OF THE ILEUS PREVENTION

A. Procurement of luminal ventilation
   1. Gastrointestinal aspiration
   2. Intraluminal insufflation of oxygen

B. Removal of digestive juices
   1. Gastrointestinal aspiration
   2. Fasting prior to operation
   3. Parenteral nutrition
   4. Fasting after operation until the reappearance of intestinal motility

C. Procurement of intestinal perfusion by oxygenated blood
   1. Infusion therapy – maintenace of the volume and capacity of blood to transport oxygen. Correction of the deficit in fluids and electrolytes
   2. Optimalisation of the cardiopulmonal and renal systems
   3. Anaesthesia without hypoxaemia
   4. Spinal anaesthesia

7.14 Inflammation and other intestinal diseases

Inflammatory bowel diseases represent two clinically distinct diseases, namely ulcerative colitis and