an overall disease. It is necessary not to exclude the patient's sexual orientation from diagnostical considerations. Homosexuals yield a high incidence of intestinal infections.

Chronic diarrhea can occur in several forms. Secretory chronic diarrhea is usually induced by drugs, hormones, bile or fatty acids. Osmotic chronic diarrhea is usually induced by drugs, laxatives, or malabsorption. Inflammatory intestinal diseases (ulcerative colitis, Crohn's disease), ischaemic colitis, parasitic invasions, motility impairments, sclerodermia and diabetic neuropathy can also bring about chronic diarrhea.

7.12 Constipation

Constipation is a prolonged period of the passage of intestinal contents through the large intestine, dense intestinal contents, less frequent defecation with a small amount of stool and an impairment of the defecatory reflex. Regarding the wide range of normal habits of bowel evacuation, it is difficult to define obstipation accurately. Obstipation is defined as a reduction in frequency of defecation down to less than 3 times per week.

The amount of stool depends on many factors, such as the amount and quality of food, velocity of its intestinal passage, resorption, bacterial flora, intestinal excretion etc.. Neither frequency, nor the amount of stool represent signs indicating to obstipation. However, many patients yield normal frequency, but they must develop excessive strain in order to achieve bowel evacuation. Their stool is hard and they have a sensation of fullness in the hypogastrium and as well as that of incomplete evacuation. Therefore, when stating the definition of constipation, it is necessary to take into consideration both **objective** and **subjective** criteria.

Constipation can be divided according to several aspects. The most important is the classification of obstipations that is based on causality. Such classification justifies the distinguishment of symptomatic (secondary) and independent (primary) obstipation. On the basis of its duration, obstipation can be either acute or chronic; according to the origin, obstipation can be organic or functional.

Symptomatic (secondary) obstipation represents merely a symptom that accompanies other primary pathological states, most frequently being the diseases of the digestive tract or other organs. It can occur in acute or chronic forms.

The acute form, i.e. cessation of natural and habitual defecatory manners can be due to strangulation, obturation and paralytic ileus. A retention of gases and stool, meteorism, colicky pain, intestinal hyperperistalsis above the site of obstruction, emesis and shock can arise.

The chronic form occurs in coincidence with incomplete stenosis of the large intestine in consequence of slow tumour growth or of adhesions. It occurs in cases of lead toxicosis, morphinism, hyperacidity, cholelithiasis, hypothyreosis etc.

The independent (primary) obstipation represents rather a clinical entity than a symptom. It is referred to as **habitual obstipation**, which according to its pathogenesis can be divided into **simple obstipation** which is caused by an inhibition of the defecatory reflex, and **spastic obstipation** which involves a neuromuscular disturbance of the large intestine and is one of the forms of the irritable bowel syndrome.

According to the prior classification, the types of obstipation were divided into spastic and atonic forms. Later, the classification took into consideration the aspect of the site of obstipation. Yet, another classification takes into consideration anatomical and physiological aspects. According to the latter three following forms are distinguished:

1. obstipation caused by impaired motility of the large intestine (spastic form),
2. obstipation caused by impaired mechanisms of defecation (dyschezia),
3. obstipation in consequence of decreased motility (atonic form).

Spastic form

Spastic obstipation usually represents merely a symptom arising in coincidence with various impairments of the large intestine. Most frequently it occurs as a symptom within the irritable bowel syndrome.

Spastic obstipation is characterized by a combination of several types of motility impairments,

initially by hypertonic-hypokinetic and dyskinetic forms of obstipations. Hypertonia (spasmus) is most frequently localised in the descendent and sigmoid colons, while the coecum and the ascendent, or transverse colons are dilated (hypotonia), hence the intestinal contents is forced through the contracted portions of intestine with excessive effort and in small bulks. In consequence of a prolonged stasis in the spastic area, the stool becomes dense and comminuted due to resorption of water. Such consistency of stool does not represent an adequate stimulus able to evoke defecation.

Dyschezia (pelvirectal obstipation, torpor recti, proctogenous obstipation).

Stool accumulates in the rectal ampulla without evoking defecation. The cause assumedly resides in an increase in the threshold of irritability neccessary for the arousal of the defecatory reflex and abnormal capability of adaptable relaxation of the rectum.

Atonic form

X-ray pictures in this form of obstipation display a dilated atonic large intestine, enlarged in its volume. It does not involve any congenital inferiority of the colonic musculature. Hypotonia is caused by several further factors, e.g. food poor in dietary roughage (incapable of representing a sufficient signal for the accomplishment of peristalsis) and fat, insufficient intake of fluids, deficiency in B-complex vitamins and some mineral deficiencies. The above described picture is referred to as **diffuse hypokinetic obstipation**.

A transversoptotic form is characterised by sluggishness of the transverse colon which is consequently suspended within the abdomen as a garland. It occurs more frequently in women. The cause assumedly resides in slackened abdominal walls.

7.12.1 Etiology and pathogenesis

From the pathophysiological point of view, constipation represents a general **consequence of impaired transition of the intestinal contents through the large intestine or impaired anorectal function** caused by primary impairment of motility, in association with some drugs or a large number of systemic diseases afflicting the gastrointestinal tract. Constipation may exarcerbate during chronic disease leading to either physical or psychical deteriorations, and to inactivity or immobilisation. Supportive factors can include: deficiency in food fiber, overall weakness, stress and anxiety. When judging the acute constipation, obturation should always be regarded as a possible cause of this state. In addition to neoplastic processes in the large intestine, strictures induced by ischaemia, diverticulosis, or inflammatory intestinal diseases, foreign bodies and anal strictures may be involved. Spasms of the anal sphincter caused by painful haemorrhoids or fissures can also suppress the need of bowel evacuation.

If no obstacle is present, the obstruction can be simulated by the **impairment of motility of the large intestine**. Disruption of the parasympathetic innervation of the large intestine in coincidence with injuries, or other diseases of the lumbosacral spine or sacral nerves can induce constipation with hypomotility, colon dilation, decreased tonus and sensitivity of the rectum resulting in defecatory disorders.

Patients with sclerosis multiplex may develop constipation (in coincidence with neurogenous dysfunctions of other organs). Constipation can be associated also with CNS disturbances caused by parkinsonism or cerebro-vascular episodes. The Chagas's disease – parasitic trypanosoma infection which occurs in South America, evokes constipation by damaging the ganglionic cells of the myenteric plexus.

Hirschsprung's disease – aganglionosis is characterized by an absence of myenteric neurons in the distal segment of the colon closely proximal to the anal sphincter. The diagnosis of Hirschprung's disease in a majority of patients is assessed up to the age of 6 months, in special cases the symptoms are so moderate that the disease remains undiagnosed until adulthood.

The **drugs** inducing constipation include especially those **with anticholinergic effect, antidepressants, antipyretics, codein** and other narcotic drugs. Further noteworthy substances include aluminium or calcium containing antacids, sucralfate, iron containing drugs, and calcium antagonists.

In patients with some endocrinopathies, such as hypothyreosis or diabetes mellitus, the constipation is usually moderate and affectable. Patients with myxoedema rarely develop megacolon that would endanger their lives. Constipation can be an accompanying disturbance in pregnancy, assumedly in consequence of the changes in levels of progesteron and estrogen which decrease the passage of stool through

intestines. Vascular collagenoses can be accompanied by constipation. Constipation can be an especially pronounced symptom in progressive systemic sclerosis. In this disease, the delay in intestinal passage is induced by fibrosis of the smooth colonic musculature. **In a majority of patients, the cause of constipation is not obvious**. An important role in the pathogenesis of idiopathic constipation in childhood is ascribed to psychological and physiological factors. Afflicted children suffer from retardation in stool passage. It is assumed that voluntary suppression of the call to stool, or abnormal anorectal function are involved. Young and middle-aged women can suffer from severe constipation with scarce defecation, extreme strain at defecation with low effect of laxatives and rigidity of this condition even after diet rich in fiber. 70 % of these cases display slow passage of stool via the large intestine (inertia coli). This state can be proved by administration of x-ray contrast markers which visualize the slow passage through the proximal colon. However, in 30 % of patients, the passage through the large intestine is normal and abnormalities are proved in the anorectal sensoric and motoric functions. This state is referred to as **outlet obstruction** or anismus and arises assumedly in consequence of insufficient intestinal relaxation or inappropriate contraction of both puborectal muscle and external anal sphincter. It is held that this dysfunction of rectal sfincter is an acquired or learned habit rather than an organic or neurogenous disease. Chronic strain at defecation per se can lead to descending perineum and stretching of the pudendal nerve thus impairing the anal sphincter muscle which in turn leads to faecal incontinence, or disability to retain stool. Rectocele is a rectal hernia (bulge) which can affect defecation as it fills up with stool especially during the attempts to defecate.

Chronic **idiopathic pseudo-obstruction** is a scarce impairment in which the episodes of intestinal obstruction are not accompanied by visible evidence of the mechanical blockade. This impairment can have a familial occurrence in consequence of neuropathy and myopathy of the intestines, and sometimes also of the urinary bladder.

Idiopathic megacolon or **megarectum** is a dilated large intestine or rectum, possibly due to constipation and extreme effort at defecation. This condition is ascribed to neurogenous dysfunction.

Young and middle-aged persons most frequently develop constipation in coincidence with the **irritable bowel syndrome**. In contrast to the syndromes of idiopathic obstipation, the irritable bowel syndrome is typical by concomitant abdominal pain, especially that in the hypogastrium with hard pellet-like stool, sensation of incomplete evacuation and extreme effort at defecation. Patients can complain of meteorismus, inflated abdomen, heartburn, nausea, difficulties at deglutition, back pain and urogenital symptoms. The pathophysiological mechanism of the symptoms is not precisely clear.

A precise description of symptoms and their duration are data of great importance in case history. Constipation lasting from birth or early childhood is assumedly of congenital origin, whereas its later development suggests an acquired character of the disease. Diagnostic considerations should always include the possibility of obstruction being caused by neoplastic processes.

Especially valuable is the information on the precedent use of laxatives and modes of their application. It is very important to evaluate sensitively and carefully the signs of anxiety and the use of mood affecting substances.

7.12.2 Irritable bowel syndrome

Irritable bowel syndrome (IBS) foremostly involves disturbances of motoric and secretory functions of the large intestine. The assumed pathogenetic mechanism of this impairment proceeds as follows: increased irritability of the neuromuscular intestinal apparatus impairs the normal peristaltic rhythm, the condition of which results in constipation alternating with diarrhea.

IBS is typical by **painful dyspepsia**. Basically, it coincides with an uncertain colicky pain varying in localisation and developing on the basis of dyskinesia. A particular intestinal portion is afflicted by spasm which hinders the propulsion of the intestinal contents. In the attempt to overcome the obstacle, the section above the spasm develops an increased activity which distends the intestinal wall. Owing to the wall distension, the patients undergo the sensation of pain.

It is frequent in this condition that mucus appears in stool. An especially typical symptom arising in coincidence with an atack of **mucous colic** is that the patient defecates pure mucus. The stool has often

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