juice in order to be able to bind with the intrin-
sic factor)

d) increased consumption of cobalamin in coinci-
dence with bacterial overgrowth

**Malabsorption can occur in many systemic dis-
eases.** Patients with hyperthyroidism often display moderate steatorrhea. Its main cause assumedly re-
sides in a high intake of fat in consequence of hy-
perphagia and faster motility of the digestive tract.
Furthermore, steatorrhea can be enhanced by gas-
tric, biliary and pancreatic secretions and by changes
in the jejunal morphology.

Malabsorption is less frequently associated with hy-
pothyreosis. Malabsorption in hypothyreosis is
ascribed to pancreatic impairment. Patients with
diabetes mellitus can develop malabsorption in co-
incidence with the bacterial overgrowth which ap-
pears in consequence of delayed passage caused by
autonomous neuropathy. Systemic sclerosis may be
accompanied by bacterial overgrowth together with
consequent malabsorption. Malabsorption in sys-
temic vasculitis can be determined by partial atrophy
of the villi, in rheumatoid arthritis by amyloidosis.

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**7.11 Diarrhea**

Diarrhea is a state of abnormally liquid faeces. The
amount of stool exceeds 200 g per day. An in-
creased number of bowel evacuations can occur, ac-
companied with a sensation of defecatory urgency
and perianal discomfort. Faecal incontinence can be
present.

The intestine receives 8 to 10 l of fluid daily. Out
of this amount, 1500 to 2000 ml are included in food.
The remnant is constituted by the secretions of sali-
vary, gastric, pancreatic, biliary and intestinal secr-
tory glands. Out of this amount, the small intestine
absorbs approximately 1 l. The large intestine ab-
sorbs 90% of fluid. A volume of 100 to 150 ml of
fluid per day is eliminated by stool.

**Absorption of water** depends on absorption of so-
lutes. Basically, the water within intestines is moved
passively in the direction of the osmotic gradient.

Therefore, e.g. the presence of weakly absorbable and
osmotically active solutes in intestinal lumen deter-
s absorption of water, or even causes secretion of wa-
ter.

The mechanisms responsible for **absorption of so-
lutes** vary in individual parts of the intestine. In
general however, salts and other substances are ab-
sorbed by specific mechanisms. Several substances
are absorbed in dependence on the sodium transport.
The latter involves both the entrance of substances
into, and their elimination from enterocytes. The
sodium transport involves a large amount of com-
ponents including protons, chlorides, glucose, amino
acids, bile acids and other substances. Secretion of
bicarbonates is associated with the sodium-hydrogen
exchange. The chyme in the ileum is moderately al-
kaline. Jejunal and ileal contents attain significant
values of ions as follow: $\text{Na}^+ - 1140 \text{mmol}$, $\text{K}^+ -
6,0 \text{mmol}$, $\text{Cl}^- - 100 \text{mmol}$, $\text{HCO}_3^- - 30 \text{mmol}$.

The **absorption of solutes in the large intestine**
takes place in a partially different manner. It is lim-
ited by the presence of electrolytes. Absorption is
carried out through specific sodium channels where
the electric potential is developed in the membranes.
The membrane potential is the basis of chlorides ab-
sorption and potassium secretion. The large inte-
state contents yields high concentration of potassium.
Organic acids produced by colonic bacteria and bi-
carbonates release organic anions and $\text{CO}_2$ from non-
absorbable saccharides and fat. The fluid in the large
intestine contains: $\text{Na}^+ - 40 \text{mmol}$, $\text{K}^+ - 90 \text{mmol}$,
$\text{Cl}^- - 15 \text{mmol}$, $\text{HCO}_3^- - 30 \text{mmol}$ and organic anions
- 85 mmol.

In addition to the above mentioned processes,
the small and large intestines simultaneously secrete
electrolytes and water. The secretion in small in-
testine is associated with the entrance of chlorine
anions. Sodium and water are thereafter passively
transported in accordance with the electric and os-
motic gradients.

Diarrhea can occur in consequence of several
changes:

1. decreased absorption of solutes and water
2. increased secretion of electrolytes
3. the presence of hardly absorbable and osmoti-
cally active solutes in the intestinal lumen
4. abnormal intestinal motility
5. intestinal inflammation with exudation and production of mucus, haemorrhage and the presence of pus.

Basically, the diarrhea may be divided into three groups: secretory diarrhea, osmotic diarrhea and diarrhea due to abnormal intestinal motility.

### 7.11.1 Secretory diarrhea

The secretory diarrhea is caused by abnormalities in both secretion and absorption of electrolytes. The central role in these impairments is played by an increase in cAMP which has two basic effects:

1. inhibits neutral NaCl absorption
2. stimulates the secretion of chlorides without impairing other transport mechanisms.

The impact of the cyclic AMP is evident e.g. in cholera. In cases of cholera, the intestinal mucosa binds the toxin which stimulates adenylate cyclase to produce the cyclic AMP. Since other transport mechanisms are intact, rehydration can be achieved by oral application of the solution of glucose with sodium. Hypersecretion can be associated also with an increased intracellular calcium or cyclic GMP. Impairments of the small intestine which cause atrophy of microvilli are associated with an increased secretion of electrolytes. Non-absorbable bile acids and fatty acids can stimulate the secretion of ions in the mucosa of the large intestine. The secretory diarrhea has the character of a copious profuse diarrhea which lasts for two days. The diarrhea fluid contains prevalingly water and electrolytes.

### 7.11.2 Osmotic diarrhea

Osmotic diarrhea is caused by an accumulation of weakly absorbable solutes in the intestinal lumen. It can appear:

1. after an intake of weakly absorbable solutes, such as lactulose, Mg\(^+\), SO\(_4\)\(^-\), or PO\(_3\)\(^-\),
2. due to malabsorption,
3. in specific impairments of absorption of such substances as lactose.

Osmotic diarrhea caused by the presence of weakly absorbable solutes is typical by the fact that it ceases after abstaining from their intake. The value of stool pH in malabsorption of saccharides is significantly shifted to the acid side.

### 7.11.3 Diarrhea due to abnormal intestinal motility

Abnormal motility can enhance bacterial overgrowth within the intestine. Increased motility shortens the contact period of chyme with mucosa. Therefore in consequence of this change, the volume of chyme in the large intestine increases which causes faster evacuation. The consequent abbreviation of contact of chyme with mucosa results in a decrease in water absorption. Diarrhea due to abnormal intestinal motility is usually present in coincidence with irritable bowel syndrome, after gastrectomy, vagotomy, diabetic neuropathy, sclerodermia and thyreotoxicosis.

In general, patient’s information on subjective sensations concerning defecation and stool are of important diagnostic value. Voluminous stools give evidence of an impairment either in the small intestine, or in the proximal part of the large intestine. A small volume of stools and frequent defecatory urge give evidence rather of an impairment in the descendent colon, or rectum. Presence of blood in stool indicates to mucosal inflammation and other pathological conditions. Foamy stool gives evidence of malabsorption of carbohydrates. Fatty stool, its offensive odour, or the presence of visible admixture of fat (steatorhea) signifies malabsorption of fat. It is very important to keep in mind that antibiotics and a number of drugs can induce diarrhea on the basis of various mechanisms. In addition to antibiotics, antacids, antihypertensives, thyroxine, digitalis, propranolol, quinidine, colchicine, lactulose, ethanol and laxatives can be involved.

Acute diarrhea is caused by infection, drugs or develops after consuming particular substances in food. Intestinal ischaemia is not associated with acute diarrhea.

Faeces in acute diarrhea contain pus and leukocytes. In order to assess precisely the cause, it is necessary to distinguish which of the pathological states is involved. Inflammation, malabsorption, structural changes or abnormal intestinal motility must be taken into consideration. Each case of diarrhea must be judged regarding the possible presence of
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 obstructions that is based on causality. Such classification justifies the distinction 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,