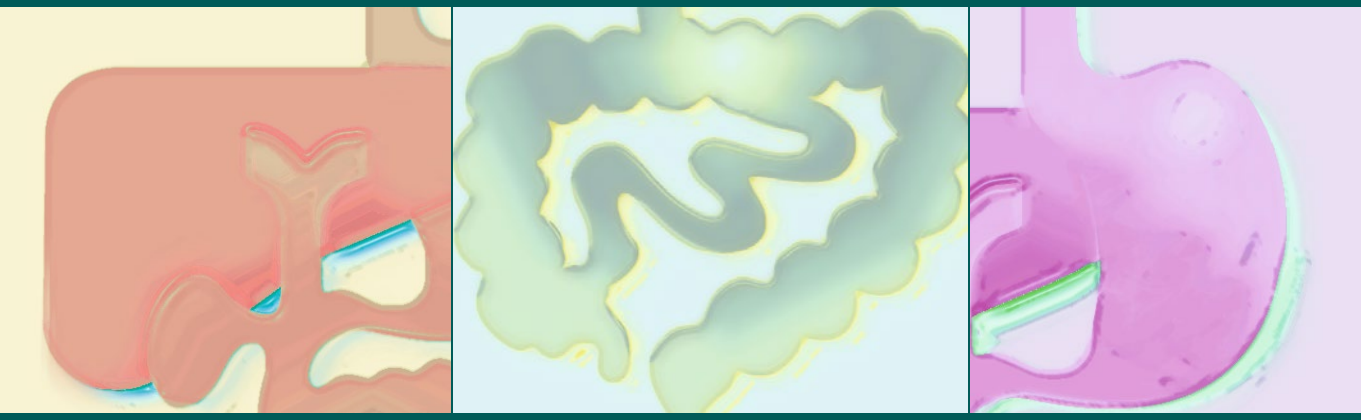
Gastroenterology
From Symptom to Diagnosis

A Guide for Hospital and Practice



T. Kucharzik, K. Kannengießer, P.G. Lankisch
Städtisches Klinikum Lüneburg (Germany)

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A Guide for Hospital and Practice

T. Kucharzik, K. Kannengießer, P.G. Lankisch
Städtisches Klinikum Lüneburg (Germany)

Authors' address:

Prof. Dr. T. Kucharzik
Dr. K. Kannengießer
Prof. Dr. P.G. Lankisch
Klinik für Allgemeine Innere Medizin
und Gastroenterologie
Städtisches Klinikum Lüneburg
Bögelstr. 1
21339 Lüneburg
Germany

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"Gastroenterology – From Symptom to Diagnosis" is intended as a guideline for the rapid clarification of gastrointestinal symptoms. It has been deliberately limited to the essentials and does not claim to be a guide to comprehensive differential diagnosis. The cited references are intended to provide greater depth of information in the text, or to support individual statements. This is not intended as a comprehensive review of the literature on the various symptoms.
This guide is intended for physicians and students who are interested in gastroenterology.

T. Kucharzik, K. Kannengießer, P.G. Lankisch
Klinik für Allgemeine Innere Medizin und Gastroenterologie,
Städtisches Klinikum Lüneburg (Germany)

1 Fetor ex ore and halitosis

Definition

- *Fetor ex ore:* foul odor from the mouth (disorder in the mouth)
- *Halitosis (= bad breath):* foul odor on the breath, even when mouth is closed
- *Parosmia:* hallucinatory disorder of the sense of smell that is associated with some neurological/mental health conditions

Possible causes

The most common cause of fetor ex ore is bacterial plaque, a biofilm made up of microcolonies of bacteria that are embedded in a matrix or glycocalyx. The quantity, composition, and specific metabolic activity of the microorganisms determine the strong-smelling and unpleasant metabolic products that are formed – such as hydrogen sulfide. In the oral cavity, plaque can be found predominantly on tooth surfaces, but it is also found in the gingival sulcus/gingival pocket, and on the tongue dorsum in the architecture of the papillae of the tongue.

Fetor ex ore is usually attributable to a cause originating in the upper oropharynx or nasopharynx, whereas halitosis is usually attributable to a cause originating in deeper organs such as the lungs and upper gastrointestinal tract. Overlapping is possible. About half of those affected do not notice their own bad breath.

Causes of fetor ex ore

- Smoking: Heavy smoking causes the development of a very fuzzy tongue that can trap food remnants and components of the inhaled tobacco substance. When combined with reduced saliva production, these trapped elements create a foul-smelling odor in the mouth, as well as halitosis (see below)
- Poor oral hygiene: Bacteria encounter a bounty of food, which provides them with ideal living conditions. Protein-rich foods such as milk, and fat-rich dairy products or fish encourage halitosis
- Plaque and food residues between the teeth
- Cavities
- Poorly positioned dental prostheses, loose bridges and crowns
- Heavy, mostly crust-like coatings on the dorsoposterior surface of the tongue and on the oral mucosa
- Acute necrotizing ulcerative gingivostomatitis (periodontal disease characterized by necrotizing ulcers of the interdental papillae which spread along the gingival margins)

- Acute and chronic inflammation, usually caused by bacteria, such as rhinitis, Vincent's angina (foul odor), diphtheria (sweet odor), or inflammatory mucous membrane changes caused by cytostatic agents
- Disintegrating tumors (for example disintegrating tissue masses from extensive squamous cell carcinomas) that have been colonized by anaerobes that produce organic hydrogen sulfide compounds, thiols or mercaptans, which as gases are notable for their unpleasant odor
- Xerostomia (reduced saliva flow) when breathing via the mouth, depression, salivary gland disease (such as Sjögren's syndrome), anticholinergic medication (atropine derivatives and psychotropic drugs)
- Mouth odor in the morning (low saliva production during the night, saliva does not have a sufficient deodorizing effect)

Causes of halitosis

- Diseases of the respiratory tract
  - Heavy smoking (see above), and exhalation of tobacco components that have been deposited on the mucous membranes of the upper respiratory tract
  - (Predominantly chronic) lung infections, such as purulent bronchitis, bronchiectasis, pneumonia, lung abscesses
  - Tumors of the larynx and the lungs
- Diseases of the digestive tract
  - Retention of food in the upper gastrointestinal tract, for instance in the case of esophageal diverticula or esophageal carcinoma, achalasia, gastric outlet obstruction, ileus, or foreign bodies
  - Helicobacter pylori colonization of the stomach
- Absorption of odorous substances in the intestines and exhalation via the lungs
  - Specific foods, such as garlic, leeks, and onions (exhalation of sulfur compounds)
  - Intoxication with phosphorus, arsenic, parathion, selenium, tellurium (garlic smell)
  - Medication
  - Hunger or fasting state (it is likely that the odor is caused by breakdown products of the body's own fat and proteins, ketone bodies)
- Metabolic diseases such as diabetic coma (sweet, fruity, acetone smell), hepatic coma (smell of raw liver), uremia (ammonia, urine-like smell)
- Unexplained halitosis: exhalation of malodorous fatty acids of unknown etiology

Practical approach

Medical history

- Detailed history of food intake and medication
- Objective assessment of the mouth odor by the physician

Physical examination

- Inspection of the oral cavity and tonsils, and if necessary, X-ray of the paranasal sinuses, plus ENT consultation if required

Further diagnostic procedures

- Esophagogastroduodenoscopy to exclude organic causes originating in the upper gastrointestinal tract
- X-ray of the chest to detect chronic lung disease
- Dental examination should be performed to detect any residues behind/underneath the edges of crowns, and should also be performed in the case of fillings with rough surfaces and/or gingival pockets. The examination should be combined with professional tooth cleaning.

Nota bene (NB)

- An objective assessment of bad breath is very important because some patients feel that they have bad breath even though they do not (halitophobia). A psychiatric consultation may be appropriate in such cases.
- Check whether oral hygiene really is being adhered to (brushing teeth at least twice a day, flossing to clean between the teeth at least every second day, plus it may be advisable to recommend an additional brush or scraper for the tongue in the case of heavy tongue coating).
- Always arrange a dental examination in the case of diseases in the oral cavity.

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Kapoor U, Sharma G, Juneja M, Nagpal A. Halitosis: Current concepts on etiology, diagnosis and management. Eur J Dent. 2016;10(2):292–300.

2 Taste disorders

Definition

- *Total ageusia:* complete lack of ability to taste or inability to distinguish between sweet, salty, bitter and sour
- *Partial ageusia:* the inability to taste some of the aforementioned flavor qualities
- *Specific ageusia:* the inability to taste certain substances
- *Hypogeusia:* reduced ability to taste, may be either total or partial – i.e. can affect either some or all flavor qualities
- *Dysgeusia:* perception of a different taste than the one presented at taste testing, or perception of a taste when no taste is presented
- *Parageusia:* incorrect taste perception in the form of altered perception of one or more flavor qualities, often combined with an impaired sense of smell
- *Cacogeusia:* subjective perception of a bad taste when none is present

Possible causes

A prerequisite for a sense of taste is sufficient production of saliva – the saliva brings the substances that produce the taste to the tasting organ. The tasting organ is the collection of taste buds that are mainly found on the tongue dorsum, margin of the tongue, and the base of the tongue, but also on the palatopharyngeal arches. Taste buds are bud-like chemoreceptors found in the epithelium of the vallate papillae and foliate papillae of the tongue, and some isolated taste buds are also found in the fungiform papillae. They are made up of cylindrical sensory cells whose microscopic hair-like protrusions (microvilli) protrude out from the taste pores on the epithelial surface.

From there, they send taste stimuli to the brain. The taste fibers from the anterior two thirds of the tongue run via the lingual nerve, the chorda tympani, and the facial nerve (intermediate nerve), and those from the vallate papillae (posterior third of the tongue) and foliate papillae run via the glossopharyngeal nerve.

A taste disorder can occur at various levels (Table 2/1):

- Local
  - Xerostomia (dry mouth): The substances that produce the taste are not transported to the taste buds
  - Damage to receptors: The receptors cannot receive the substances that produce the taste
- Central
  - The taste signal cannot be transmitted to the CNS

1. Taste disorders linked to signaling

- Medications
- Heavy metal intoxication
- Radiation therapy
- Sjögren's syndrome
- Xerostomia

2. Taste disorders linked to sensory impairment

- Aging process
- Candidiasis
- Medications (thyrostatic agents, cytostatic agents)
- Endocrine causes
- Carcinoma in the oral cavity
- Pemphigus
- Radiation therapy
- Viral infections (especially with herpes viruses)

3. Central taste disorders

- Diabetes
- Hypothyroidism
- Carcinoma in the oral cavity
- Oral and maxillofacial surgery procedures
- Radiation therapy
- Kidney disease
- Stroke and CNS conditions
- Trauma
- Infections of the upper respiratory tract, e.g. common cold

Table 2/1
Causes of taste disorders
(modified according to Lalwani 2015)

In addition, there are a number of endocrine causes and other causes.

Taste disorders by localization

- Taste disorders of the anterior two thirds of the tongue:
  - Facial nerve disorders (in the case of processes in the brain stem, skull base region, or pertosal region, and in the case of idiopathic facial nerve processes)
  - Disorders of the chorda tympani (in the case of processes in the middle ear, following temporomandibular joint fractures and electrocoagulation of the geniculate ganglion)
  - Disorders of the lingual nerve (following tonsillectomy)
- Taste disorders of the posterior third of the tongue:
  - Glossopharyngeal nerve disorders (in the case of processes in the brain stem, skull base region, and jugular foramen, and in the case of tonsillar abscesses, tonsil tumors, and following tonsillectomy)
- Single-sided taste disorders affecting all three thirds of the tongue:
  - Disorders of the central gustatory pathway (in the case of infarctions, bleeding, trauma, inflammation, or tumors)

Practical approach

Medical history

- General medical history (prior radiation therapy, trauma, surgery)
- Detailed medication history

Physical examination

- Taste testing (if appropriate, testing can be done to clarify whether the taste disorder may have an anatomical cause – see taste disorders according to localization):
  Taste sensation testing for
  1. sour using diluted vinegar or citric acid
  2. sweet using sugar solution
  3. salty using saline solution
  4. bitter using quinine solution (as the final test)
- Electrogustometry if appropriate
  (neurological consultation required for this examination)

Further diagnostic procedures

- Exclusion of the aforementioned hormonal or metabolic disorders (see Table 2/1)

NB

- Taste disorders are not a serious gastrointestinal symptom, but they are unpleasant for patients.
- Older people generally only report that they have a taste disorder when they are asked about it. In this population, taste disorders are one of the causes of reduced food intake and the resulting weight loss that sometimes occurs in old age (see section 15: "unintentional weight loss").
- Also consider mental health conditions when taking the history!
- Xerostomia can have various causes, but one of the most common ones is the general aging process. Xerostomia is often associated with a poor self-cleaning function of the oral cavity and insufficient oral hygiene. This damages the oral mucosa, and the damage can in turn cause taste disorders.
- Xerostomia induced by antibiotics or glucocorticoids is particularly prone to causing Candida overgrowth when the affected person also has impaired immunity. In the case of a patient with a taste disorder who is also generally at risk of fungal overgrowth in the upper gastrointestinal tract, it is advisable to attempt treatment using nystatin or another antifungal drug.

- Patients with kidney disease have an elevated threshold for the perception of a sweet or sour taste, but this normalizes after dialysis.
- Important in terms of the history: Dysgeusia is often observed following trauma to the chorda tympani branch of the facial nerve during surgical procedures in the middle ear region, or following extraction of the third molar (wisdom tooth – trauma to the lingual nerve with chorda tympani).
- Also important: distinguishing between "nothing tastes good to me anymore" (e.g. in the case of a lack of appetite) and "I can't taste anything" (e.g. in the case of a taste disorder).

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3 Singultus (hiccups)

Definition

Incontrollable involuntary contraction of the diaphragm leading to a quick inspiration that ends with abrupt closure of the glottis, producing the typical "hic" sound.

Possible causes

The precise mechanism that causes singultus is unknown. It seldom occurs as a cardinal symptom. Rather, it almost exclusively occurs as a concomitant symptom in various diseases.
The diaphragm is the effector of the following reflex arc: Parts of the vagus nerve, sensory fibers of the phrenic nerve, and sympathetic fibers of the lower thoracic segments Th6 to Th12 form the afferent limb, and the phrenic nerve and cervical nerve (C5–C7) and thoracic fibers (Th1–Th11) of the sympathetic nervous system form the efferent limb. Coordination probably takes place in the medulla oblongata or in the cervical medulla. A singultus may be triggered by the irritation of individual sections of this reflex arc. The causes may be functional, psychological, systemic toxicity-related, or organic in origin.

The duration of a singultus varies depending on its cause. Usually, a distinction is made between self-limiting singultus that lasts less than 48 hours, and persistent singultus, which last longer. Whereas in the first type of singultus (self-limiting and short-lived type), the cause is usually functional in origin, in the case of the second type (persistent type), the cause may be intoxication or more serious underlying organic conditions (see Tables 3/1 and 3/2).

Circumstances that could trigger singultus:	
– Stretching of the stomach	• Eating large quantities or eating very quickly • Aerophagia • Carbonated beverages • Air insufflation during a gastroscopy
– Sudden temperature change	• Eating very cold or very hot food • Sudden change in ambient temperature • Cold showers
– Psychological factors	• Sudden excitation • Emotional distress

Table 3/1
Singultus < 48 hours (likely due to a functional cause) (modified according to Kawan et al. 2003)

Systemic toxicity-related causes

- Uremia
- Electrolyte disorders (hyponatremia, hypokalemia, hypocalcemia)
- Sepsis
- Alcohol intoxication
- Medication side effects, e.g.:
  - Benzodiazepines
    - Diazepam
    - Chlordiazepoxide
  - Short-acting barbiturates
  - Dexamethasone
  - Dopamine agonists

CNS conditions

- Cerebrovascular insufficiency
- Cerebral tumors
- Hydrocephalus
- Encephalitis
- Meningitis
- Parkinson's disease
- Postoperative states

Diseases of the neck region

- Goiter
- Lymphadenopathy (inflammatory, metastatic)
- Pharyngitis/laryngitis

Diseases of the thoracic organs

- Lung
  - Bronchitis
  - Pleurisy
  - Central lung carcinoma
  - Tuberculosis
- Heart and blood vessels
  - Myocardial infarction
  - Myocarditis
  - Pericarditis
  - Aortic aneurysm
- Esophagus
  - Reflux esophagitis
  - Esophageal carcinoma
- Mediastinum
  - Mediastinitis
  - Mediastinal tumor

Diseases of the abdominal organs

- Esophagus/stomach
  - Paraesophageal hernia
  - Gastric carcinoma
  - Peptic ulcer disease
- Liver
  - Liver abscess
  - Cirrhosis of the liver
  - Hepatocellular carcinoma
  - Subphrenic abscess
- Bile
  - Cholecystitis
  - Cholelithiasis
- Pancreas
  - Pancreatitis
  - Pancreatic pseudocyst
  - Pancreatic carcinoma
- Peritoneum
  - Peritoneal carcinomatosis

Mental health conditions

- Neuroses
- Anorexia nervosa

Table 3/2
Singultus > 48 hours (likely due to an organic cause) (modified according to Kawan et al. 2003 and Steger et al. 2015)

Practical approach

Medical history

- History of the concomitant circumstances that triggered the singultus
- History with regard to warning signs of malignant diseases such as B symptoms, loss of appetite, etc.
- Detailed history of alcohol intake and medication

Physical examination

- Thorough physical examination, paying particular attention to whether any tumor is present (anemia, enlarged lymph nodes, hepatomegaly, pathological resistance in the abdomen)

Further diagnostic procedures

- Chest X-ray or chest CT and echocardiography to rule out any diseases of the lungs, heart, and aorta
- Sonography and esophagogastroduodenoscopy to rule out diseases of the upper gastrointestinal tract, liver, gallbladder, and pancreas
- Cerebral computed tomography (CT) or nuclear magnetic resonance imaging of the skull
- Neurological consultation if required
- Psychiatric consultation if required

NB

- There are many postulated causes of persistent singultus, and the "home remedies" and medication-based treatments that it is thought may help are also very numerous, but there is no clear evidence for any of this. Small studies and case series have shown positive effects for baclofen, metoclopramide, gabapentin, and chlorpromazine, but there is a lack of large-scale controlled studies on these.

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Steger M, Schneemann M, Fox M. Systemic review: the pathogenesis and pharmacological treatment of hiccups. Aliment Pharmacol Ther. 2015;42(9):1037–50.

4 Nausea and vomiting

Definition

- *Nausea:* subjective sensation of needing to vomit. Unpleasant feeling in the upper abdomen that is associated with loss of appetite and is accompanied by a feeling of pressure or cramping in the hypopharynx. In severe cases, hypersalivation occurs, along with a feeling that vomiting is imminent.
- *Retching:* involuntary contraction of the abdominal, thoracic and pharynx musculature, often occurring before or during vomiting
- *Vomiting:* forceful expulsion of the stomach contents via the mouth, often occurring after nausea and retching. During vomiting, the peristaltic movements of the small bowel that propel the contents onwards stop and retroperistalsis occurs, along with contraction of the abdominal, thoracic and pharyngeal muscles, and hypersalivation.
- *Regurgitation:* not the same as vomiting. It is when the contents of the esophagus return upwards, for instance in the case of web stenosis, diverticula, achalasia, or esophageal carcinoma.

Possible causes

Possible causes of nausea and vomiting include medications and toxins, as well as many different gastrointestinal and non-gastrointestinal causes (see Table 4/1).

General: Nausea and vomiting may be concomitant reactions occurring in the presence of severe diseases, with or without fever and with or without pain, and sometimes with a combination of fever and pain (see Tables 4/2 and 4/3).

Practical approach

Medical history

- Detailed history, paying particular attention to medications and the time when vomiting occurred

Physical examination

- Thorough investigation of the condition of the entire body, paying particular attention to abdominal findings (ileus? pain/abdominal guarding as a manifestation of inflammation?)
- If required, investigation of neurological status, including assessment of the ocular fundus
- ENT consultation if required

Intra-abdominal causes

- Disruption to intestinal transit (benign/malignant)
  - Pyloric stenosis
  - Ileus of the small/large bowel
  - Mesentericoduodenal obstruction
  - Efferent loop syndrome following Billroth II resection of the stomach
- Inflammatory diseases (with or without visceral pain)
  - Infectious gastroenteritis
  - Peptic ulcer
  - Acute cholecystitis with or without biliary colic
  - Acute pancreatitis
  - Acute appendicitis
  - Acute hepatitis
  - Peritonitis
- Motility disorders
  - Reflux disease
  - Irritable stomach
  - Diabetic gastroparesis
  - Intestinal pseudoobstruction
- History of radiation therapy in the abdominal region
- Gastrointestinal tumors
  - Pancreatic carcinoma (including without duodenal stenosis)
  - Metastases with intestinal lumen obstruction or, for instance, peritoneal carcinomatosis with formation of ascites/obstruction of passage

Extra-abdominal causes

- Cardiovascular diseases
  - Cardiomyopathy
  - Myocardial infarction
  - Hypertensive crisis
  - Vasovagal syncope
- CNS diseases
  - Migraine
  - Increased intracranial pressure
    - Tumor
    - Bleeding/infarction
    - Meningitis
    - Congenital malformation
    - Hydrocephalus
    - Pseudotumor cerebri
  - Labyrinth disorders
    - Motion sickness
    - Menière's disease
    - Vestibular neuronitis
- Mental health conditions
  - Anorexia nervosa/bulimia
  - Depression
  - Psychogenic vomiting
- Postoperative vomiting
- Endocrine/metabolic causes
  - Pregnancy
  - Uremia
  - Acute liver failure
  - Diabetic ketoacidosis
  - Thyroid and parathyroid diseases
  - Adrenal insufficiency

Medications and toxins

- Medications
  - Chemotherapy drugs
  - Antibiotics
  - Antiarrhythmic drugs
  - Digoxin
  - Oral antidiabetic drugs
  - Oral contraceptives
  - Opiates
  - Theophylline
- Toxins
  - Ethanol
  - Fungi
- Food poisoning (e.g. staphylococci)

Table 4/1
Possible causes of nausea and vomiting (modified according to Hasler 2015)

Laboratory tests

- Electrolytes (potassium, calcium, sodium, chloride)
- Blood glucose
- Acid-base balance
- Inflammation parameters
- Iron status: iron deficiency anemia?
- Liver, kidney, and pancreatic values: acute hepatitis? renal failure? acute pancreatitis?
- Pregnancy test
- Special endocrinological examination if required
- Determination of digoxin/theophylline levels if required

Further diagnostic procedures

- Sonography, and if necessary, abdominal survey radiography with the patient standing up or lying on their left side: ileus?

If these examinations do not provide enough information, if appropriate, perform:

- Esophagogastroduodenoscopy
- Colonoscopy
- Assessment of passage through GI tract using water-soluble contrast medium or MRI enterography (Sellink technique MRI)
- Computed tomography of the abdomen
- Gastric emptying scintigraphy and/or small bowel manometry
- Laparoscopy to rule out or treat adhesion ileus or peritoneal carcinomatosis
- Cerebral computed tomography/MRI examination of the head

NB

- The clinical constellation in which vomiting occurs often permits conclusions to be drawn regarding the cause. The medical history plays a key role here. Factors such as the duration of the vomiting and the vomit's appearance, taste, and smell, are as important as the concomitant circumstances.
- Blood in the vomit?
  – If yes, the source of bleeding is proximal to the ligament of Treitz
- Concomitant symptoms:
  – Fever: inflammation
  – Weight loss: serious cause, obstruction or carcinoma
- Vomiting without prior nausea: cause originating in the CNS
- A thorough history of the patient's medication must be taken. Almost any medication can trigger nausea and vomiting – it is often simply a question of the dose and the patient's individual tolerance.

Cardinal symptom: Acute vomiting with nausea		
Indicative	**Causes**	**Confirming the diagnosis**
Medical history	• Medication • Intoxication	Medical history, determination of levels, toxicological tests (serum, urine, stomach contents)
Abdominal pain		
Mostly without fever	• Gastritis	Esophagogastroduodenoscopy* (with histology)
	• Duodenal ulcer	Esophagogastroduodenoscopy*
	• Peptic ulcer	Esophagogastroduodenoscopy* (with histology; Cave! carcinoma)
	• Biliary colic	Sonography, laboratory tests, endoscopic ultrasound or MRCP
	• Renal colic	Sonography, urinalysis, intravenous urography/CT of the abdomen
	• Myocardial infarction	ECG, laboratory tests (troponin-T, CK)
Mostly with fever	• Gastroenteritis	(Possibly) Detection of microorganisms in the stool
	• Acute hepatitis	Laboratory tests (hepatitis serology, transaminase levels)
	• Cholecystitis	Sonography, laboratory tests (CRP, ALP, gamma-GT, WBCs)
	• Appendicitis	Clinical signs, sonography, laboratory tests (WBCs), surgery
	• Pyelonephritis	Urinalysis, detection of microorganisms in the urine
With fever and signs of an acute abdomen	• Ileus	Sonography, and if appropriate, abdominal survey radiography, CT of the abdomen, surgery
	• Pancreatitis	Sonography, laboratory tests (serum amylase and/or serum lipase)
	• Mesenteric ischemia	Abdominal survey radiography, sonography, CT angiography
Dizziness		
Peripheral vertigo with or without tinnitus	• Vestibular neuronitis	Clinical signs, ENT consultation
	• Menière's disease	Clinical signs, ENT consultation
	• Motion sickness	Medical history
Severe headache		
One-sided or diffuse headache	• Migraine	Clinical signs
	• Hypertensive crisis	Blood pressure measurement
Severe headache around the eyes and forehead "Pink eye"	• Glaucoma	Intraocular pressure measurement
		* with detection of Helicobacter pylori

Table 4/2
Differential diagnosis of acute vomiting (modified according to Zeh et al. 2003)

Cardinal symptom: Acute vomiting with nausea		
Indicative	**Causes**	**Confirming the diagnosis**
Possibly decreased consciousness, usually **without** focal neurological deficits	• Heat stroke • Concussion • Meningoencephalitis	Medical history Medical history CSF examination, possibly CT or MRI
Possibly decreased consciousness, usually **with** focal neurological deficits	• Cerebral tumor • Cerebral hemorrhage • Cerebral infarction	CT, MRI
Reduced state of consciousness		
Metabolic disorders, usually **without** severe headache	• Intoxication • Hypotonic dehydration, hypotonic hyperhydration • Hypercalcemia • Uremia • Diabetic ketoacidosis • Liver cell failure	(Third party) medical history, toxicological tests Laboratory tests (sodium in serum and urine, osmotic concentration in serum and urine) Determination of serum calcium, clarification of causes Laboratory tests (retention values in serum, acid-base status) Laboratory tests (blood glucose measurement, acid-base status) Laboratory tests (ammonia)
Adynamia		
Hypotension, hypoglycemia, hyperkalemia	• Addison's disease	Laboratory tests (electrolytes, acid-base status, determination of cortisol and ACTH levels)
Heat intolerance and weight loss		
Tachycardia Systolic hypertension	• Hyperthyroidism	Laboratory tests (thyroid values, thyroid antibodies) Thyroid scintigraphy, thyroid sonography
Abbreviations: ENT: ear, nose, and throat; CSF: cerebrospinal fluid; CT: computed tomography; MRI: magnetic resonance imaging; ACTH: adrenocorticotropic hormone		

Continuation of Table 4/2

Cardinal symptom: Chronic vomiting *with* nausea		
Indicative	**Causes**	**Confirming the diagnosis**
Vomiting, partially independent of food intake (e.g. occurring in the morning while still in fasted state)	• Early stage of pregnancy	Medical history, gynecological examination, sonography, pregnancy test
	• Chronic alcoholism	Medical history, liver damage? (Note: determination of MCV and gamma-GT)
	• Use of medication	Medical history, attempt treatment discontinuation
	• Radiation therapy	Medical history
Vomiting with longer intervals between eating and vomiting, usually marked weight loss	• Gastric outlet obstruction	Esophagogastroduodenoscopy
	• Gastric carcinoma	Esophagogastroduodenoscopy
	• Afferent loop syndrome	Medical history, upper gastrointestinal X-ray series
	• Pancreatic carcinoma	Sonography, CT, endosonography, with fine needle aspiration if necessary, ERCP
	• Autonomic (diabetic) polyneuropathy	Medical history, neurological examination, diabetes diagnosis, evidence of delayed emptying of the stomach
Signs of right-sided cardiac insufficiency	• Heart failure	Clinical picture, auscultation of heart and lungs, chest X-ray, echocardiography
Cardinal symptom: Chronic vomiting *without* nausea		
Indicative	**Causes**	**Confirming the diagnosis**
Young female patients (15–25 years) Amenorrhea Disturbances of food intake Neuroticism	• Anorexia nervosa	Psychiatric investigation
	• Bulimia	
Abbreviations: MCV: mean cell volume; GI tract: gastrointestinal tract; CT: computed tomography; ERCP: endoscopic retrograde cholangiopancreatography		

Table 4/3
Differential diagnosis of chronic vomiting (modified according to Zeh et al. 2003)

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5 Heartburn

Definition

A burning pain that climbs up from the epigastrium and is felt predominantly in the retrosternal region, but sometimes also in the pharyngeal and/or epigastric region.

Possible causes

Heartburn is the cardinal symptom of gastroesophageal reflux disease (GERD), and the most common cause of this disease is a functional disorder of the lower esophageal sphincter.

- In terms of differential diagnostics, heartburn can also occur
  - in the case of non-acid reflux (e.g. bile reflux)
  - as a variant of irritable stomach or hypersensitive esophagus
  - as a concomitant symptom of a motility disorder or hypermotility
  - in the case of secondary reflux disease due to disorders of the emptying of the stomach (atonia in the case of neuropathies, gastric outlet obstruction)
  - in the case of polyneuropathies or neuromuscular diseases (e.g. muscular dystrophy)
  - in the case of collagen diseases (e.g. scleroderma)
  - in the case of esophageal thrush
  - in the case of viral esophagitis
  - in the case of eosinophilic esophagitis
  - after chemical burns
  - in the case of radiation-induced esophagitis
  - in the case of esophagitis of other etiology

Practical approach

Medical history

- Thorough medical history, paying particular attention to questions about triggering factors
- It is also important to ask about "upper" and "lower" reflux symptoms (it is possible for the patient to be affected in both regions):
  - *Reflux symptoms in the upper region:* burning sensation in the pharynx, acidic eructation, non-acidic eructation (= regurgitation, often incorrectly described as vomiting!), dry cough/slight cough in the morning, husky voice, hoarseness, asthmatic attacks
  - *Reflux symptoms in the lower region:* heartburn, retrosternal chest pain, dysphagia (difficulty swallowing), in rare cases odynophagia (pain when swallowing)

Physical examination

- A thorough physical examination is always required, but it rarely provides information to guide the diagnosis.

Further diagnostic procedures

- Esophagogastroduodenoscopy in order to classify lesions visible in the endoscopy in the case of GERD and to rule out other esophageal diseases (Table 5/1)
- If the cause of the symptoms remains unclear, use long-term pH-metry or impedance measurement if appropriate
- Possibly manometry and/or contrast medium examination of the esophagus with reflux testing for the purpose of differential diagnosis (e.g. to rule out achalasia or motility disorders/ hypermotility)
- ECG/stress test to rule out coronary artery disease
- Chest X-ray and, if appropriate, lung function test to rule out bronchopulmonary diseases
- ENT examination in the case of suspicion of acid-related laryngitis, if appropriate in combination with two-point acidity measurement to rule out a proximal reflux

NB

Symptoms

- In terms of sensitivity, heartburn is the best indicator of GERD. If heartburn is reported as the leading clinical symptom, it is highly likely (> 75%) that reflux disease is present (Koop et al. 2014).
- Information about the nature, intensity and frequency of reflux symptoms and when they occur (when fasting, after eating, at night) cannot be used to determine the severity of GERD (e.g. to determine whether esophagitis is present and to what extent) (Koop et al. 2014).
- However, it is thought that individual risk of developing adenocarcinoma of the distal esophagus increases in line with increasing frequency of reflux symptoms, occurrence of reflux symptoms at night, and with increasing length of the history of reflux (Lagergren et al. 1999).
- Many patients are unable to say with certainty whether their pain is a pain felt as pressure, a cramping pain, or a stabbing pain. The quality of the pain is not very helpful in the differential diagnosis of retrosternal symptoms.
- It is the extent of the symptoms, and not the extent of the lesions that is decisive in terms of how the patient is able to cope with their professional and personal life, and to how far

their overall quality of life is affected (Labenz and Borkenstein 2003). In light of this, it is important to remember that non-erosive reflux disease (NERD) can in some cases cause more problems than the erosive form (ERD).

- In the case of proven reflux, extra-esophageal symptoms may occasionally be the predominant symptoms – for instance laryngitis, asthma, and chronic bronchitis (microaspiration!) – perform further diagnostics if necessary. However, extra-esophageal symptoms of GERD are quite rare overall and almost never occur without heartburn occurring at the same time.
- Medications that are used in the treatment of lung diseases (theophylline, β_2 sympathomimetic drugs) or heart diseases (nitrates, calcium antagonists), as well as psychopharmaceuticals (tricyclic antidepressants) can lower the pressure in the lower esophageal sphincter and worsen the esophageal clearance by inhibiting the motor function of the tubular esophagus.
- Foodstuffs such as fat, chocolate, alcohol and peppermint also lower the pressure in the lower esophageal sphincter, protein increases it.

Grade A	One (or more) mucosal break no longer than 5 mm that does not extend between the tops of two mucosal folds
Grade B	One (or more) mucosal break more than 5 mm long that does not extend between the tops of two mucosal folds
Grade C	One (or more) mucosal break that is continuous between the tops of two or more mucosal folds but which involves less than 75% of the circumference
Grade D	One (or more) mucosal break which involves at least 75% of the esophageal circumference

Table 5/1
The Los Angeles classification of gastroesophageal reflux disease

Diagnosis

- Even in the case of clear reflux symptoms and absence of alarm symptoms (e.g. dysphagia, weight loss, signs of bleeding), endoscopy at an early stage is advisable. Endoscopy allows primary diagnosis of reflux esophagitis while at the same time allowing the level of severity to be determined. This facilitates treatment planning and precise therapy monitoring. It is useful in diagnosing Barrett's esophagus and in detecting complications (e.g. ulcer, stricture). It also serves to rule out diseases that must be treated immediately, such as malignant tumors.

- In order to classify lesions that are visible in the endoscopy in the case of GERD, the Los Angeles classification should be used in accordance with the current DGVS guideline (DGVS = German Society for Gastroenterology, Digestive and Metabolic Diseases).
- In terms of the patient's prognosis, Barrett's esophagus diagnostics are of crucial importance, and the diagnosis is usually determined according to the Prague classification. Two measurements are combined for this: Firstly, the length of the changes that extend across the entire circumference of the esophagus (circumferential metaplasia) is noted in centimeters together with the designation "C", and secondly, the additional, mostly tongue-shaped extensions are also noted in centimeters with the designation "M" for the maximum extension.
- A single negative 24-hour pH-metry does not rule out reflux disease, nor does a normal esophagogastroduodenoscopy.
- Manometry and contrast medium examinations of the esophagus are used for differential diagnosis (see above). They cannot be used to diagnose GERD.
- Attempting symptomatic treatment using proton-pump inhibitors (ex-juvantibus diagnostics) is permissible, provided that you are aware that the following applies, even if there is an improvement in the symptoms:
  – Many diseases exhibit a periodic disease pattern with spontaneous improvement.
  – The treatments that are available are non-specific. For example, an acid inhibitor can also improve the symptoms of a malignant gastric ulcer.
  – Both the patient and the physician making the judgment are often susceptible to the placebo effect.

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6 Dysphagia

Definition

- *Odynophagia:* painful obstruction of the act of swallowing
- *Dysphagia:* painless obstruction of the act of swallowing. The term "dysphagia" is generally used as a generic term for all painful and painless swallowing disorders.
- *Globus pharyngeus (hystericus):* a feeling of having a "lump stuck in the throat", but swallowing can be done without any difficulty

The different disorders that lead to dysphagia can be classified according to their localization:

- *Oropharyngeal dysphagia:* disturbed transport of the bolus from the mouth to the esophagus. The mouthful remains in the mouth during the swallowing attempt or passes into the nose or trachea.
- *Esophageal dysphagia:* obstruction of passage in the esophagus. The patient only feels that something is wrong after the end of the voluntary part of the act of swallowing.

Possible causes

Oropharyngeal dysphagia (modified according to Koop 2013)

- *Neurological diseases:*
  - CNS: history of cerebrovascular insult, Parkinson's disease, hypoxic encephalopathy, etc.
  - Peripheral: neuropathy
  - Muscle diseases: muscular dystrophy, metabolic, myasthenia gravis (early symptom)
- *Tumors of the oropharynx* including tumors of the proximal esophagus
- *Infection of the oropharynx* including abscesses, tuberculosis
- *Disorders of the upper esophageal sphincter*
- *Congenital deformity of the pharynx*

Esophageal dysphagia

- *Reflux disease*, possibly even without peptic stenosis
- *Eosinophilic esophagitis*
- *Motility disorders* (achalasia, scleroderma, diffuse esophageal spasm)
- *Carcinomas* (esophageal and extra-esophageal)
- *Diverticula*
- *Benign stenosis* (benign tumors, ring or web stenosis, anastomotic strictures)
- *Compression from outside* (blood vessels, spinal spur), *foreign bodies*
- *Postoperative* (fundoplication)
- *Functional*

Practical approach

Medical history

- Detailed medical history: provides the diagnosis in about 80% of cases.

Physical examination

- Inspection of the oral cavity and pharynx to rule out inflammation (e.g. pharyngitis or thrush) and tumors
- Inspection of the skin and the extremities: scleroderma?
- Palpation of the thyroid (enlargement affecting the esophagus?)
- Auscultation of the chest: aspiration pneumonia?

Oropharyngeal dysphagia

Question	Response	Indicates
Other neuromuscular disorders?	Yes	History of cerebrovascular insult, difficulty swallowing in the context of neuromuscular diseases (e.g. myasthenia)
	No	Hypopharyngeal cancer
Improved by drinking?	Yes	Dry mouth
	No	Neuromuscular

- ENT consultation if required
- Neurological consultation if required

Further diagnostic procedures

- Esophagogastroduodenoscopy with random biopsies
- X-ray (esophageal examination using barium swallow)

Esophageal dysphagia

Question	Response	Indicates
For how long?	For several months	Achalasia
	For weeks	Malignant tumor
	Sudden	Foreign bodies, bolus aspiration
	For a few days	Infectious or medication-induced esophagitis
At every meal?	Yes	Organic
	No	Functional
Problems with both solid and liquid food?	Yes, from the beginning	Achalasia, spasm
	No, initially only with solid food	Organic stenosis
Is it painful when food gets stuck?	Yes	Organic stenosis, spasm
	No	Achalasia
Additional symptoms?	Continuous pain	Malignant tumor
	Cough	Aspiration, fistula
	Hoarseness	Recurrent laryngeal nerve paralysis, aspiration

- CT of chest and abdomen
- Esophageal manometry
- Fiberoptic endoscopic evaluation of swallowing (FEES) in the case of suspicion of oropharyngeal dysphagia

NB

- In the case of myasthenia gravis, often, even in the early stages, it is difficult for the patient to push the mouthful of food into the pharynx using the tongue. In addition to the muscles of the mouth, the eye muscles (eyelid lifting muscles) and the muscles of the neck and larynx are also affected. Rapid fatigue of the affected muscle groups and the short-term recovery after pausing to rest are characteristic signs.
- In the case of the "drying out" of the oral cavity that has persisted for some time, Sjögren's syndrome should be considered as a possible cause in addition to medication. Difficulty swallowing can also be caused by xerostomia.
- In the case of scleroderma, dysphagic symptoms are not obligate symptoms, and they usually occur at a late stage. At this point, the characteristic symptoms of scleroderma cannot be overlooked.
- In case of irritable cough after eating, the possibility of esophageal carcinoma should be considered (tracheo-esophageal fistula).
- If after a large mouthful, and especially during a meat meal, further swallowing of solid or liquid food is impossible, consider the possibility of "steakhouse syndrome"/"backyard barbecue syndrome". The trapped (meat) bolus can usually be recovered by endoscopy. Causes of the "steakhouse syndrome" include: pathological ring formation, reflux esophagitis with stricture formation, postoperative strictures in the anastomosis area, eosinophilic esophagitis.

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7 Acute abdomen

Definition

Acute abdomen is one of the most important conditions in visceral medicine. The term refers to a serious condition with very severe abdominal pain, which requires both immediate investigation and rapid treatment due to its tendency to worsen rapidly.

Cardinal symptoms

The cardinal symptoms of acute abdomen are:
- Severe abdominal pain (circumscribed or diffuse)
- Peritoneal symptoms (abdominal guarding)
- Acute circulatory disorder, up to and including circulatory shock
- Disturbance of intestinal peristalsis (meteorism, nausea, vomiting)

In the case of acute abdomen in elderly people, some of the cardinal symptoms may be absent, or they may be less pronounced.

Pain

In the everyday clinical setting, the precise nature of pain is of critical importance as a cardinal symptom. Colic-like pain is more indicative of mechanical obstructions. This visceral pain is usually dull or diffuse and is characterized by distension of the hollow organs, muscle contractions, spasms, or abrupt stretching of organ capsules. However, constant pain is more indicative of inflammatory processes. This somatic pain is often a burning pain and is often easy to localize, originating from the parietal peritoneum, mesentery, mesocolon or retroperitoneal space. Important clues can be derived from the kinetics of the pain – sudden onset versus slow onset, constant worsening versus spontaneous clearing of symptoms (**Cave!** "silent interval" in the case of mesenteric ischemia for example). The localization of the pain provides further clues to the possible causes of the symptoms.

Abdominal guarding

Abdominal guarding may initially be localized in the region of the diseased organ, but can also be present in the entire peritoneum (peritonism), both at the beginning or diffusely later on. Signs of inflammation such as fever and changes in the blood count as well as peritonism often herald subsequent peritonitis.

The following often lead to peritonitis:
- Intra-abdominal bleeding
- Perforation
- Ileus
- Visceral perfusion disorder

- Inflammation of an abdominal organ that extends into the peritoneum

The abdominal guarding that is normally present in the case of acute abdomen may be absent or diminished in the case of:
- Inflammatory processes in the lesser pelvis
- Muscle weakness in the abdominal wall
- Prior high dose cortisone therapy
- Prior administration of antispasmodic drugs or analgesics
- Polyneuropathies
- Patients on artificial respiration (sedatives? muscle relaxants?)
- Dialysis patients
- Very young or very old patients

Important causes of acute abdomen

When looking for the causes of acute abdomen, abdominal and extra-abdominal causes as well as metabolic disorders must be considered (Table 7/1; modified according to Silen 2015)

Diagnosis

Acute abdomen is an emergency situation within the field of visceral medicine. With this condition, there is little time for diagnostic measures, and the relevant diagnostic measures can sometimes only be performed after the treatment for circulatory shock (which gets to work immediately) has been administered. The time available for diagnostic procedures depends on the severity of the condition. Rapid interdisciplinary diagnostics are crucial to the patient's prognosis.

Medical history

It makes sense to ask targeted questions about:
- **When the pain started:** For instance, in the case of colic caused by a ureteric calculus, patients give a precise answer to the question "What were you doing when the pain started?" ("I was just opening the car door"), but in the case of appendicitis, the response is vague ("The pain started in the afternoon").
- **Prior events that may help guide the diagnosis:** Trauma to the upper abdomen for instance points to a rupture of the liver, spleen, or intestines.
- **Nature of the pain:** Depending on the localization, colic-like pain may indicate biliary colic, colic caused by a ureteric calculus, or mechanical ileus. Women who have been through childbirth often describe "labor-like pains". Persistent, frequently increasing pain indicates an inflammatory process, for example appendicitis, pancreatitis, or cholecystitis. If the pain symptoms flare up again after an initially se-

Abdominal causes	Extra-abdominal causes	Metabolic disorders
• Peritonitis (parietal peritoneum) – Bacterial inflammation, e.g. in the case of perforated appendix, inflammation of the pelvic region – Chemical irritation, e.g. in the case of perforated ulcer, acute pancreatitis • Mechanical obstruction – Ileus of the small/large bowel – Choledocholithiasis – Ureteric calculus – (Intestinal pseudoobstruction) • Vascular causes – Embolism or thrombosis – Aortic dissection – Sickle-cell anemia – Rectus sheath hematoma • Trauma including intra-abdominal bleeding • Gynecological causes – Tubo-ovarian abscess – Ectopic pregnancy – Ovarian cyst torque or ruptured ovarian cyst	Diseases of the... • ...chest, e.g. pleuropneumonia, pulmonary embolism, myocardial infarction • ...genitals, e.g. testicular torsion • Paroxysmal nocturnal hemoglobinuria (PNH)	• Exogenic causes – e.g. lead poisoning • Endogenic causes – Uremia – Diabetic ketoacidosis – Acute intermittent porphyria – Addisonian crisis – Hemolytic crises – C1 esterase inhibitor deficiency – Mediterranean fever

Table 7/1
Important causes of acute abdomen (modified according to Silen 2015)

vere pain has subsided ("stage of illusion"), it is crucial to consider whether this is due to a perforation (e.g. ulcer or gallbladder) or due to a vascular process such as mesenteric ischemia, and whether the flare-up may be heralding the onset of peritonitis. In the case of mesenteric ischemia, gangrene of the small bowel is usually present, and this requires prompt surgical resection of the affected bowel segment.

- **Initial/current pain localization:** There is a large amount of temporal variability and variability between individuals when it comes to localization of the pain – for example, projection of biliary colic pain into the epigastrium is often observed, whereas the initial pain symptoms of acute appendicitis are classically located in the epigastrium and later shift to the right lower abdomen. In addition, the development of localized pain into diffuse pain may indicate secondary development of peritonitis.

Physical examination

Inspection: The patient's posture can help guide the diagnosis:

- Restless, rolling back and forth: Biliary colic/ colic caused by a ureteric calculus?
- Curved protective posture: Pancreatitis, peritonitis?

Auscultation: Auscultation should always be performed before palpation and percussion because the latter two measures may stimulate bowel peristalsis and thus obscure, for example, a subileus:

- Normal bowel sounds: normal peristalsis
- Increased bowel sounds: enterocolitis? mechanical (adhesion) ileus? inflammatory or tumorous intestinal diseases (holosystolic murmur)?
- Reduced/absent bowel noises: paralytic ileus, reflectory in colic caused by ureteric calculus, pancreatitis, metabolic disorders. In this case, differential diagnosis should be performed with suspicion of ischemia and peritonitis.

Palpation/percussion: Is abdominal guarding detectable? If yes, the localization may help guide the diagnosis: The irritation of the parietal peritoneum may correspond to the organ boundaries in the abdominal cavity. In the case of diffuse (rubber-like) abdominal guarding, acute pancreatitis should be suspected. The localized abdominal guarding may allow conclusions to be drawn regarding the affected organ. Hernial orifice examinations serve to rule out an incarcerated hernia.

In the case of suspicion of abdominal wall pain: The very simple Carnett's test (Carnett 1926) that was initially described for chronic pain makes it possible to distinguish between intra-

abdominal pain sources and pain sources in the abdominal wall, which is often difficult when using other means. If the patient is able to indicate the location of the pain by indicating the exact point with the finger, or if they can limit it to only a small area of pain, this test should be performed. Palpation of the location of maximum pain before and after tensing the abdominal wall should then be done. If the pain symptoms have increased or are still present in the same place after the abdominal muscles have been tensed, the cause lies in the abdominal wall; meaning that the Carnett's sign is positive. If the pain is less or if it has even disappeared, an intra-abdominal cause can be assumed, meaning that the Carnett's sign is therefore negative.

Testing for special clinical signs: Before the introduction of imaging techniques, physicians relied solely on medical history and clinical findings (inspection, auscultation, palpation and percussion). Some of these physicians have described signs that are now associated with their names and are still valuable today as indications of organ diseases (Table 7/2). Investigations into the sensitivity and specificity of these signs are rarely done.

Further diagnostic procedures

Laboratory tests

The indication for laboratory testing depends on the suspected condition (see Table 7/3). At a minimum, the following investigations should be done:

- C-reactive protein (CRP)
- Blood count
- Serum amylase and/or serum lipase
- Cholestasis parameters
- Creatinine
- Electrolytes
- Blood glucose
- Lactate
- Urinary sediment

Imaging procedures

Medical history and clinical examination should precede the establishment of an indication for examination using an imaging procedure. If two or more imaging procedures are of equivalent value for diagnosis, the fastest and most economical procedure that places the lowest possible burden on the patient should be selected.

Psoas test *Positive:* pain when moving the thigh, especially when overextending the hip joint (→ appendicitis? diverticulitis?)
Blumberg's sign (M. Blumberg, 1873–1955, surgeon in Berlin) *Positive:* crossed rebound tenderness with right lower abdominal pain during rapid removal of pressure on the left (→ appendicitis?)
Rovsing's sign (T. Rovsing, 1862–1927, surgeon in Copenhagen) *Positive:* increased pain in the ileocecal region upon retrograde palpation of the colon (→ appendicitis?)
Boas sign (I.I. Boas, 1858–1938, gastroenterologist in Berlin) *Positive:* hyperesthetic zone on the back in the paravertebral region between T11 and L1 (→ cholecystitis?)
Obturator sign Rotation of the thighs and hip joints causing tensing of the obturator muscle (→ appendicitis? salpingitis? abscess in the pelvis/ retroperitoneum?)
Murphy's sign (J.B. Murphy, 1857–1916, surgeon in Chicago) Left hand is placed on right costal arch, and the spread thumb is pressed into the region of the gallbladder. While inhaling, the gallbladder is pressed against the thumb by the diaphragm, which is moving downward. *Positive* in the case of → cholecystitis? hepatitis?
Kehr's sign (H. Kehr, 1862–1916, surgeon in Berlin) *Positive:* pain in the left shoulder in the case of acute abdomen (→ splenic rupture?)

Table 7/2
Additional examinations in the case of suspicion of defined organ disease (Aldea et al. 1986; Bree 1995; Ralls et al.1982; Urbano and Carroll 2000)

Ultrasound examination (including duplex sonography)

The ultrasound examination is the continuation of the physical examination of the acute abdomen. The procedure is non-invasive, requires no preparation, and can be repeated at any time. The time required for a systematic examination of the abdominal organs should be no more than 10–15 minutes.

If an ultrasound examination can confirm a diagnosis of pneumoperitoneum or ileus, in many cases, it is possible to dispense with the usual abdominal survey radiography with the patient in a standing or left-sided position. There is a lack of studies on how long free air under the crus of diaphragm can still be detected after perforation of a hollow organ in the abdominal cavity. However, postoperative examinations have shown that a pneumoperitoneum can be detected by computed tomography up to 18 days after surgery (Gayer et al. 2000), and that this imaging method is superior to abdominal survey radiography with the patient on their left side (Earls et al. 1993).

Computed tomography (with use of oral and/or rectal, and possibly intravenous contrast medium)

Ask about contrast medium allergies when arranging a contrast CT. Care must be taken when using contrast media in the case of:

- Renal failure
- Hyperthyroidism
- Plasmacytoma
- Metformin treatment

Generally speaking, the spectrum of diagnostic possibilities using computed tomography is similar to that of sonography. The time required with the 16-row devices is 3–5 minutes. No special preparation of the patient is required. The not inconsiderable radiation exposure should be taken into account when establishing the indication.

A complete computed tomography examination should be carried out if the medical history and physical examination, sonography and, if applicable, native X-ray imaging surveys have not led to a diagnosis, or if a questionable diagnosis needs to be clarified further. This applies in particular to cases where ability to assess by sonography is limited, or where precise topographic classification of lesions, gas accumulations, soft

Question/ suspected diagnosis	Laboratory tests	Machine-aided diagnostics
Acute pancreatitis	Lipase/amylase, if necessary, ALP, gamma-GT, AST, bilirubin, BC, CRP	Sonography
Acute cholecystitis	BC, CRP, if necessary, ALP, gamma-GT, AST, bilirubin	Sonography
Choledocholithiasis	BC, CRP, ALP, gamma-GT, AST, bilirubin	Sonography, in further course endoscopic ultrasound/ERCP
Acute appendicitis	BC, CRP	If necessary, sonography
Ileus	BC, CRP, lactate	Sonography, X-ray of the abdomen, CT of the abdomen
Perforation of hollow organs	BC, CRP, lactate	Sonography, if necessary, CT of the abdomen
Mesenteric ischemia	BC, CRP, lactate	(Angio-) CT of the abdomen
Intra-abdominal bleeding	BC, INR, PT	Sonography, if necessary, CT of the abdomen
Intra-abdominal abscess	BC, CRP	Sonography, if necessary, CT of the abdomen
Ureteric calculus	Urinalysis, BC, creatinine, urea	Sonography, if necessary, CT of the abdomen
Ectopic pregnancy	Pregnancy test	Sonography, gynecological consultation
Tubo-ovarian abscess	BC, CRP	Sonography, gynecological consultation
Ruptured ovarian cyst/ovarian cyst torque	BC, CRP	Sonography, gynecological consultation
Aortic aneurysm/dissection	BC, INR, PT, D-dimer	Sonography, if necessary, CT of the abdomen
Myocardial infarction	Troponin, CK, LDH, AST	ECG
Diabetic ketoacidosis	Glucose, ABG, urinalysis	

Abbreviations:
ALP: alkaline phosphatase; gamma-GT: gamma glutamyl transferase; AST: aspartate aminotransferase; BC: blood count; CRP: C-reactive protein; INR: international normalized ratio; PT: prothrombin time; CK: creatinine kinase; LDH: lactate dehydrogenase; ABG: arterial blood gas; CT: computed tomography; ECG: electrocardiogram

Table 7/3
Laboratory diagnostics and machine-aided measures according to suspected diagnosis

tissue structures, calcifications, and foreign bodies has to be performed.

CT angiography is indicated in cases of suspected mesenteric perfusion disorders, and possibly also in cases of lienal or renal perfusion disorders. Since mesenteric perfusion disorders are still too often detected too late, this disease should be given generous consideration and the indication for CT angiography should be established accordingly. The procedure is also suitable for diagnosing otherwise undetectable sources of bleeding in the gastrointestinal tract, and bleeding from kidney injuries.

Magnetic resonance imaging

The question of the diagnostic value of magnetic resonance imaging (MRI) for acute abdomen remains open.
In pregnant women with acute abdominal and/or pelvic pain or acute abdomen, MRI is recommended if the cause of the symptoms cannot be clarified using an ultrasound examination (Birchard et al. 2005). Ultimately, the fact that the quick access to the method is limited may be a limiting factor for this method.

X-ray diagnostics

A chest X-ray in two planes can rule out an extra-intestinal cause of an acute abdomen, such as basal pleuropneumonia. To detect pneumoperitoneum or ileus, abdominal survey radiography with the patient in a standing or left-sided position will serve.

Endoscopy

Endoscopy is of limited value in emergency diagnostics for acute abdomen. In the case of acute abdominal pain of unclear etiology, an esophagogastroduodenoscopy can be used to establish or rule out a peptic ulcer or duodenal ulcer as the cause of the pain.
If the patient is willing to have surgery, esophagogastroduodenoscopy is permissible in the case of suspicion of a sealed perforation, if this perforation has not previously been evidenced by other examinations. If the perforation is confirmed, but the location is unclear, an esophagogastroduodenoscopy can be performed to detect the perforation site.

Electrocardiogram

Myocardial infarction is one of the most important differential diagnoses for acute abdomen. An electrocardiogram should therefore be per-

formed in every case of acute abdomen where it is not clear from the medical history that the cause is definitely located in the abdomen.

NB

- **Interdisciplinarity:** Patients with acute abdomen are often first presented to the internist and not to the surgeon. Since the clinical picture of acute abdomen can often change dramatically within a short period of time, it is important for the internist to consult their surgical partner immediately to assess the patient's clinical picture in order to ensure that the surgeon has more than just a "snapshot" of the situation, but rather is familiar with "the whole film", and can therefore effectively cooperate in making the decision about the further procedure, or take over the treatment of the patient.
- **Auscultation:** The auscultation of the abdomen can be disappointing: The bowel sounds may be completely normal despite an urgent indication for surgery, for instance in the case of strangulation ileus or perforated appendicitis.
- **Pain localization:** The localization of the pain does not always correspond to the limits of the affected organ; for example, basal pneumonia can cause pain in the right upper abdomen, and sometimes even in the midabdomen. A raised, inflamed appendix can cause pain in the upper abdomen.
- **Physical examination is more difficult in the case of**
  - **Pregnant women:** Acute appendicitis is the most common cause of acute abdomen in pregnancy (1:6000). During pregnancy, uterine enlargement displaces the appendix from the lower right quadrant to the upper right quadrant, thus pushing it away from the abdominal wall, which may have the result that there is no longer any irritation of the parietal peritoneum.
  - **Young women:** Consider the possibility of gynecological causes (ectopic pregnancy, ruptured ovarian cyst/ovarian cyst torque, acute salpingitis, tubo-ovarian abscess), arrange gynecological consultation if necessary. Precise menstruation history (last period? how heavy was the last period?) is essential; both questions can provide clues as to whether there is a pregnancy with possible complications.
  - **Very young or very old patients:** Children exhibit less abdominal guarding because the muscular development of the abdominal wall is not yet complete. Elderly people exhibit less abdominal guarding due to increasing muscular atrophy and the reduction in physical defensive reactions which can often be

found in old age. Fever and leukocytosis are only of limited diagnostic value because they occur in many diseases affecting children and are rare in elderly patients.

– **Patients under immunosuppression:** Signs of peritonitis (pain, abdominal guarding, rebound tenderness) are less pronounced, and fever develops less frequently.

- **Laboratory tests:** Laboratory values alone are never sufficient for evaluation of acute abdomen. The leukocytosis result, for example, should never be the decisive factor in whether surgery is indicated or not. Leukocytosis of over 20,000/µl can occur in the case of peritonitis caused by perforation of a hollow organ, but it can also occur in acute pancreatitis, acute cholecystitis, and inflammatory processes in the pelvic region that do not necessarily require surgery.
- **Serum amylase/lipase values:** The severity of acute pancreatitis is independent of serum enzyme levels in the case of hospital admission (Lankisch et al. 1999).
- **Diabetic ketoacidosis:** In diabetic ketoacidosis, serum amylase is often elevated. This is partly due to an increase in total amylase, but it is also due to pancreatic/salivary isoamylase – it does not necessarily mean that acute pancreatitis is present. The cause of the elevation remains unclear in many cases. However, diabetic ketoacidosis often also leads to hypertriglyceridemia, which in turn can trigger acute pancreatitis. Acute pancreatitis is present in 10–15% of cases of diabetic ketoacidosis (Nair et al. 2000).
- **Incarcerated ureteric calculus:** In the case of acute abdomen as a result of an incarcerated ureteric calculus, the ultrasound findings may still be normal when symptoms begin because urinary retention with dilatation of the renal pelvis on the affected side can only be detected later. Initially, erythrocyturia may also be absent. Intravenous urography – until a few years ago the gold standard for clarification of calculus – is increasingly being replaced by native spiral computed tomography, which has better sensitivity and specificity than urography (Spencer et al. 2000).
- **Analgesia:** Several studies have shown that early analgesic treatment does not hinder diagnostic certainty and does not have any relevant effect on the resulting treatment decisions/surgical indications (among others, Attard et al. 1992; Gallagher et al. 2006; Vermeulen et al. 1999).

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8 Meteorism

Definition

Objective or subjectively perceived increase in intestinal gas with the feeling of being bloated, increased intestinal activity and noise, and possibly also expulsion of air via the anus (flatulence).

- *Aerophagia: swallowing of air*, however, excessive air swallowing is what is meant by this term. Most of the air that is transported to the stomach is expelled through eructation (burping). In some patients, repeated, voluntary eructation leads to increased aerophagia, thus causing a vicious circle. During eructation, gastric acid often enters the esophagus along with the air, and this can lead to reflux symptoms and, due to the triggering of a swallowing stimulus, to renewed aerophagia.
- *Flatulence:* expulsion of air via the anus. Frequency varies widely between individuals and depends on nutrition to a large degree. Daily gas expulsion varies between 500 and 1500 mL.

Possible causes

- *Aerophagia:*
  - Each day, about 2–3 L of air enter the stomach due to the act of swallowing (2–3 mL of air per swallow), through deep inhalation (1–2 mL air per inhalation), or through food containing air. The average transit time from stomach to anus for gases is 35 minutes, for solids, the average transit time is 30–40 hours.
- Increased net production:
  - Carbon dioxide (CO_2) is produced by carbonated beverages and by the neutralization of HCl and fatty acids by the bicarbonate from the digestive glands. CO_2 is absorbed in the small bowel and exhaled through the lungs.
  - In the large bowel, gas is produced during the breakdown of carbohydrates by bacteria and enzymes. The extent of gas production depends on the amount of undigested carbohydrates (e.g. cellulose and other fibrous substances) and the diet (e.g. fiber-rich food, wholemeal bread, muesli, fruit, vegetables, legumes; onions in particular cause increased gas production in the colon due to breakdown by bacteria).
  - N_2, O_2, CO_2, H_2, and CH_4, the five main odorless gases, account for 99% of the volume of gastrointestinal gases. About 30–50% of all humans have anaerobes in the colon that can form methane from H_2 and CO_2. The smell of some intestinal gases is caused by bacterial fermentation of unabsorbed protein products with trace formation of H_2S, NH_3, indole, skatole, and volatile fatty acids, etc.

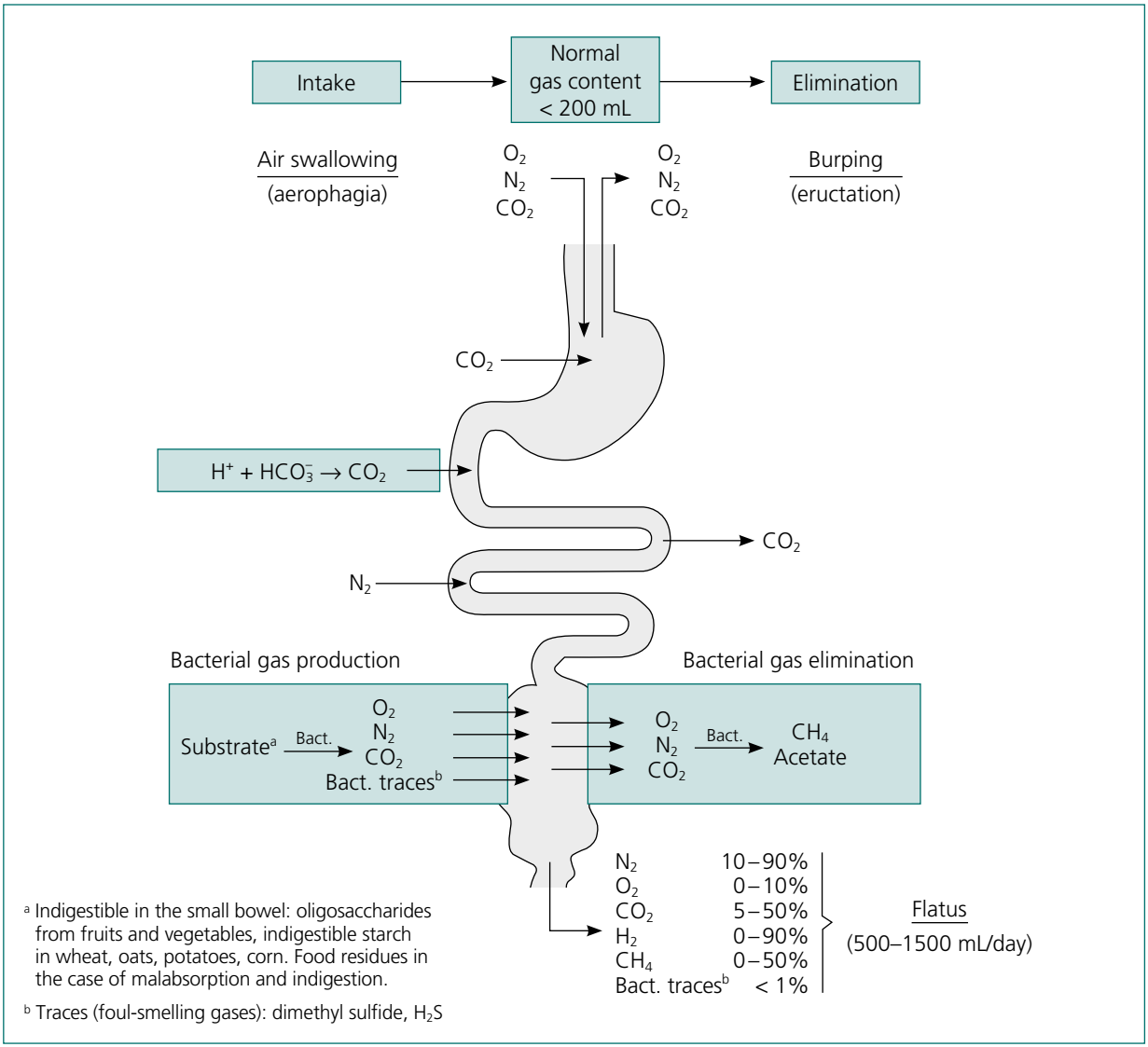

Figure 8/1
Gastrointestinal gas balance (from Koelz et al. 1995)

– Increased gas production in the gastrointestinal tract can be caused by
  – increased saliva production in the mouth, for example when chewing gum
  – incorrect eating habits, eating and drinking too quickly, too many carbonated drinks
  – increased intake of indigestible or non-absorbable (e.g. fiber-rich) foods, or
  – carbohydrates that can only be absorbed to a limited extent (e.g. lactose and fructose)

• *Causes of pathological intestinal gas production or gas production that is perceived as pathological:*
  – *Acute meteorism:*
    – Ileus (paralytic or mechanical)
  – *Chronic meteorism:*
    – Aerophagia (increased air swallowing)
      – Neurotic behavioral disorder (most common)
      – Stress, feeling anxious
      – Dry mouth, e.g. caused by medication
      – Tracheostoma
    – Increased intestinal gas production
      – Increased amount of available substrate for the colonic flora
      – Intake of indigestible/non-absorbable carbohydrates: cellulose, dietary fiber, lactulose, sorbitol, xylitol. Sorbitol, which occurs in fruit, diabetic diet products, and chewing gum is only absorbed to a small extent and causes the production of gas in the colon.
      – Intake of carbohydrates that can only be absorbed to a limited extent, such as lactose or fructose
      – Lactase deficiency
        In the case of lactase deficiency, undigested lactose enters the colon, leading to the formation of H_2, CO_2 and lactic acid, and possibly causing diarrhea.
      – Tube feeding
      – Celiac disease
      – Exocrine pancreatic insufficiency (foul flatus odor due to volatile fatty acids)
      – Short bowel syndrome
    – Bacterial overgrowth of the small bowel (e.g. in the case of history of ileoascendostomy, blind loop syndrome, or intestinal stenosis)
    – Giardia lamblia infection
  – Insufficient removal of CO_2 and reduced H_2 consumption by the colonic flora
    – Portal hypertension
    – Right-sided cardiac insufficiency
    – Antibiotic treatment
  – Gastrointestinal motility disorders
    – Irritable bowel syndrome (most common)
    – History of abdominal surgery

  - Hormonal disorders such as hyperthyroidism
  - History of fundoplication
- Very rare causes, such as pneumatosis cystoides intestinalis (cysts containing H_2 along the colon)
  - Elevated visceral sensitivity

Practical approach

Medical history

- Detailed history, paying particular attention to eating and nutrition habits, as well as use of medications

Physical examination

- Attempt to objectively assess meteorism by inspection, palpation, and percussion. Rectal examination (sphincter tone?)

Further diagnostic procedures

- General laboratory screening
- Examination to exclude organic gastrointestinal diseases (e.g. esophagogastroduodenoscopy with deep duodenal biopsies and examination of the duodenal juice for Giardia lamblia and colonoscopy, possibly additional abdominal sonography [signs of right-sided cardiac insufficiency or portal hypertension?])
- Malassimilation diagnostics: lactose H_2 breath test, D-xylose test, elastase in stool, possibly determination stool weight and stool fat content
- Exclusion of pathological small bowel colonization (glucose H_2 breath test)
- Psychiatric consultation, if appropriate

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9 Ascites

Definition

Accumulation of fluid in the free abdominal cavity, with or without an increase in abdominal girth.

Possible causes

In most cases, ascites is a complication of a disease that is already known, for example cirrhosis of the liver, severe heart failure, nephrotic syndrome, or peritoneal carcinomatosis. In other (rarer) cases, the clarification of the cause poses a diagnostic problem.
The cause of ascites formation often becomes clear following diagnostic paracentesis. Ascites can be further classified according to its causes by determining whether the ascitic fluid obtained during paracentesis is transudate or exudate (Table 9/1), or by taking the history with regard to whether other indicative symptoms or findings are present (Table 9/2).

Practical approach

Medical history

- Weight gain
- Feeling full due to increase in body circumference?
- Heartburn or shortness of breath (especially when lying down) because of increased intra-abdominal pressure due to ascites?
- Alcohol consumption?
- Prior jaundice?
- Changes to typical bowel movements?
- Peripheral edema?
- Kidney disease?

Physical examination

- Jugular venous congestion?
- Peripheral edema?
- Skin changes due to liver disease? (including spider naevi, palmar erythema, Dupuytren's contracture)
- Palpable lymph nodes (especially supraclavicular lymph node enlargement, Virchow's node) or metastases? (e.g. hard, periumbilical nodes, Sister Mary Joseph's nodule)
- In the case of suspicion of ascites:
  – Ballottement/fluid wave or
  – Flank dullness and shifting dullness when position is changed or
  – Percussion in knee-elbow position

Parameter	Transudate	Exudate
– Specific gravity – Protein concentration – Serum-ascites albumin gradient	< 1016 g/L < 3.0 g/dL > 1.1 g/dL	≥ 1016 g/L ≥ 3.0 g/dL ≤ 1.1 g/dL
Differential diagnosis 1. Portal and cardiac ascites (80% of cases)	• Cirrhosis of the liver • Right-sided cardiac insufficiency • Budd-Chiari syndrome • Constrictive pericarditis	
2. Malignant ascites		• Tumors: Ascites often hemorrhagic Cytology Tumor markers, such as CEA Ascitic fluid cholesterol Ascitic fluid/serum LDH
3. Inflammatory ascites		• Bacterial peritonitis: WBCs ↑, polymorphonuclear cells ↑ Positive culture (bacteria) PCR and culture (tuberculosis)
4. Pancreatic ascites		• Pancreatitis, pancreatic fistula: Amylase, lipase ↑
5. Hypoalbuminemia ascites	• Nephrotic syndrome • Protein-losing enteropathy	

Table 9/1
Differential diagnosis of the ascites by determining whether the ascitic fluid obtained during paracentesis is transudate or exudate

Indicative secondary symptoms and findings	Suspected diagnosis
– Signs of right-sided cardiac insufficiency	• Constrictive pericarditis • Valve defect • Birth defects • Coronary artery disease (CAD) • Inflammatory myocardial diseases • Cor pulmonale
– Signs of portal hypertension with indication of primary chronic liver disease	• Cirrhosis of the liver • Congenital liver fibrosis
– Signs of portal hypertension without indication of primary chronic liver disease	• Portal vein thrombosis • Schistosomiasis • Granulomatous diseases • Myeloproliferative diseases • Leukemia
– General tendency towards edema with diarrhea	• Indigestion-malabsorption syndrome
– General tendency towards edema without diarrhea	• Nephrotic syndrome
– General tumor signs (weight loss) – Possibly palpable liver (with formation of nodules) – Bloody ascites	• Liver metastases • Primary hepatocellular carcinoma
– General tumor signs (weight loss) – Possibly formation of palpable nodules – Bloody ascites	• Peritoneal carcinomatosis
– Sacroiliac pain – Ischiadic nerve symptoms – Gynecological palpation, and possibly dyspnea (pleural effusion right > left)	• Ovarian tumors
– Mucous aspirate during ascites puncture	• Pseudomyxoma
– Postoperative:	• Biliary/pancreatic leakage • Intestinal leakage and peritonitis

Table 9/2
Differential diagnosis of ascites according to symptoms and findings (modified according to Fehring and Jipp 1994)

Indicative secondary symptoms and findings	Suspected diagnosis
– Signs of portal hypertension without indication of chronic liver disease (hepatosplenomegaly)	• Veno-occlusive disease • Budd-Chiari syndrome
– Abdominal pain with signs of chronic liver disease and signs of portal hypertension	• Spontaneous bacterial peritonitis in the case of known cirrhosis of the liver (However, painless in 50% of cases)
– Jaundice – Splenomegaly – Fever – Flapping tremor – Fetor hepaticus – Hypotension	• Fulminant hepatitis • Decompensated cirrhosis of the liver
– Acute abdomen	• Suspected intra-abdominal bleeding, e.g. following splenic rupture
– Acute abdomen, possibly with mostly left-sided pleural effusion – History of alcohol intake – Cholecystolithiasis	• Acute pancreatitis
– Recurrent attacks of pain in the central abdomen and the left upper abdomen – Weight loss – Possibly left-sided pleural effusion	• Chronic relapsing pancreatitis

Continuation of Table 9/2

Other examinations

- Sonography
- In determining the cause of ascites formation, diagnostic paracentesis (50–100 mL) and simultaneous therapeutic paracentesis if necessary, as well as follow-up sonography are useful (see Tables 9/1 and 9/2)
- Ascites examination
  - Inspection: clear, cloudy, sanguinolent, dirty-brown, chylous?
  - Laboratory diagnostics are obligatory
    - Protein/albumin concentration with determination of the serum-ascites albumin gradient
      - High gradient: > 1.1 g/dL (protein concentration < 3 g/dL)
      - Low gradient: ≤ 1.1 g/dL (protein concentration mostly ≥ 3 g/dL)
    - Detection of microorganisms in ascites: inoculate aerobic and anaerobic blood culture bottles
    - Cell count (WBCs, RBCs, granulocytes, possibly lymphocytes)
  - Laboratory diagnostics are optional
    - LDH
    - Gram staining
    - Cytology: high specificity, but unfortunately relatively low sensitivity
    - Specific gravity
    - Lipase, amylase, creatinine, bilirubin
    - Fibronectin

Differential diagnosis

The crucial factor in the diagnosis of ascites is distinguishing between malignant ascites and benign ascites, and between infected ascites and non-infected ascites.

Malignant ascites

Since the specificity of the cytological examination of the ascites aspiration material is almost 100%, false-positive results are almost impossible. Unfortunately, the sensitivity is limited, meaning that it is advisable to be generous when deciding whether there is indication for diagnostic laparoscopy in the case of unclear ascites.

Infected ascites

The greatest risk for patients with hepatogenic ascites is the development of spontaneous bacterial peritonitis (frequency 10–20%, mortality up to 50%). The cardinal symptoms are pain, fever and jaundice in the context of hepatic decompensation. Asymptomatic progression is not unusual (Rimola et al. 2000).

The following mechanisms are decisive in terms of the pathophysiology (Wiest and Schölmerich 2006):

- Bacterial overgrowth of the gut with gram-negative bacteria
- Disorder of the mucosal barrier with increase in intestinal permeability
- Reduction in humoral and complement-mediated defenses against infection

Practical approach

In the case of suspicion of infected ascites, microbiological culturing should be performed (at least 10 mL ascitic fluid in anaerobic and aerobic blood culture bottles). If it is not possible to wait for the culture result, the number of neutrophils in the ascites should be determined; > 250/mm³ defines spontaneous bacterial peritonitis.

NB

- The lower limit of detection for clinical examination is approx. 1000–1500 mL, the lower limit of detection for sonography is approx. 50 mL.
- The serum-ascites albumin gradient is the best parameter for the detection of portal ascites and for differentiation between a neoplastic or an inflammatory etiology. A difference between serum albumin and ascites albumin concentration of > 1.1 g/dL indicates a hepatogenic etiology after exclusion of a cardiac cause (Wiest and Schölmerich 2006).
- If the etiology of the ascites remains unclear, laparoscopy is indicated.
- If a patient with known cirrhosis of the liver has hemorrhagic ascites, primary hepatocellular carcinoma is present in about 25% of cases.
- Taking account of the circulatory situation and possibly substitution with regard to albumin in the case of large-volume paracentesis (> 5 L), complete removal of the ascites by paracentesis is justifiable as long as no hepatorenal syndrome is present.
- In the case of spontaneous bacterial peritonitis, for the most part, only one pathogen, usually a gram-negative bacterium (e.g. Escherichia coli), is detectable. However, secondary peritonitis, which is often caused by intestinal perforation, is usually triggered by several microbes, and these are usually also anaerobes. Further diagnostic features which may be helpful for the detection of secondary peritonitis include concentration of alkaline phosphatase in ascites > 240 U/L, ascites CEA > 5 ng/mL, and/or a lower glucose concentration in ascites (equivalent to < 50 mg/dL) compared to the

serum as well as absence of a decrease in cell count in ascites after 48 hours. In the case of very high WBC counts in ascites, the differential diagnosis of secondary peritonitis should also be considered.

- **Cave!** Hepatorenal syndrome: restriction of renal perfusion and glomerular filtration rate (GFR) as well as oliguria in the presence of advanced liver disease and already frequent refractory ascites without evidence of primary kidney disease. This syndrome is often a complication of excessively aggressive diuretic therapy.

  A distinction is made between hepatorenal syndrome with rapidly progressive kidney failure (type I), and not rapidly progressive kidney failure (type II):

  Type I: progressive impairment of renal function with doubling of initial serum creatinine to > 2.5 mg/dL or decrease of GFR by 50% to < 20 mL/min in less than 2 weeks.

  Type II: slow serum creatinine increase to > 1.5 mg/dL or decrease of GFR to < 40 mL/min. A typical characteristic is also an only negligible improvement in renal function (reduction of serum creatinine to ≤ 1.5 mg/dL or absence of increase of 24-hour creatinine clearance to at least 40 mL/min) after discontinuation of diuretics and volume substitution of, for instance, 1.5 L glucose 5% or an electrolyte solution.
- **Cave!** Hepatopulmonary syndrome: lung perfusion disorder, sometimes with hypoxemia (rare).

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10 Diarrhea

Definition

Diarrhea is defined as stool frequency of > 3/day or stool weight ≥ 200 g/day. The stool consistency is reduced or liquid (water content > 80%). All three parameters are influenced by dietary fiber content, medication, stress and extreme sports (e.g. marathon running).

- *Acute diarrhea:* < 4 weeks duration
- *Chronic diarrhea:* > 4 weeks duration
- Acute diarrhea may signal the onset of chronic diarrhea

Special forms

- *False diarrhea:* increased stool frequency with normal stool weight (e.g. irritable gastrointestinal syndrome, proctitis, anal incontinence)
- *Paradoxical diarrhea:* liquid stool caused by bacterial fermentation of the stool, by wall stretching prior to stenosis, by stasis in the distal colon (carcinoma), and occurring in the case of immobile patients
- *Nosocomial diarrhea:* diarrheal disease occurring in hospitalized patients more than 72 hours after admission

Acute diarrhea

Possible causes

Acute diarrhea is usually the result of intoxication, infection, allergic reaction or psychovegetative dysregulation.

Practical approach

Medical history

The most important medical history questions help in the diagnosis of acute and chronic diarrhea (Tables 10/1 and 10/2).

Physical examination

The physical examination also includes an assessment of hydration and body temperature, an abdominal palpation, and a rectal examination.

Further diagnostic procedures

Most acute diarrhea is mild and self-limiting. Disease pattern and time of limitation (duration of disease < 96 hours except under special conditions; Table 10/3).

Leading symptom besides diarrhea	Pathophysiology/microorganisms
Nausea	**Toxins** • *Endotoxins* – Bacillus cereus, Staphylococcus aureus, Clostridium perfringens • *Enterotoxins* – Vibrio cholerae, enterotoxic E. coli, Klebsiella pneumoniae, Aeromonas species, norovirus (Norwalk virus), Rotavirus
Abdominal pain	**Intestine-adherent bacteria/protozoa** • Enteropathogenic or enteroadherent E. coli, Giardia species, Cryptosporidium **Cytotoxin-producing bacteria** • Clostridium difficile • Enterohemorrhagic E. coli (EHEC)
Fever	**Invasive organisms** • *Inflammation to varying extents* – Salmonella, Campylobacter, Aeromonas species, Vibrio parahaemolyticus • *Severe inflammation* – Shigellae, enteroinvasive E. coli, Entamoeba histolytica
Blood in the feces	• *Enteroinvasive pathogens* – Salmonella, Shigellae, Campylobacter, enteroinvasive E. coli, Yersinia, EHEC, Campylobacter, Entamoeba histolytica First manifestation of IBD Ischemic colitis NSAID-induced colitis Radiogenic colitis

Table 10/1
Summary of the causes of acute diarrhea according to symptoms (modified according to Hecht et al. 2015)

- Time when illness started?
- Abdominal pain?
- Vomiting?
- Stool volume?
- Stool consistency?
- Stool frequency?
- Suspicious substances in feces (e.g. blood or pus)?
- Time of defecation (e.g. does it also occur at night)?

Table 10/2
Important medical history questions for acute and chronic diarrhea

- Profuse diarrhea leading to dehydration
- Bloody stools
- Fever ≥ 38.5 °C
- Duration of diarrhea > 48 hours with no clinical improvement
- Several patients with acute diarrhea in the area
- Associated with severe abdominal pain
- Older patients ≥ 70 years, small children and/or immunocompromised patients
- Prior stay abroad, especially in southern, mostly subtropical/tropical countries
- Employees in industries where food is a relevant factor, or in the health sector

Table 10/3
Acute diarrhea: Indication for further diagnostics

Clostridium difficile and norovirus should be considered as possible infectious causes of nosocomial gastroenteritis.

If microbiological stool tests and/or bacteriological blood cultures do not lead to a diagnosis, a colonoscopy should be considered, depending on the clinical course, especially in the case of bloody diarrhea, in order to differentiate between an initial manifestation of inflammatory bowel disease, NSAID colitis, and ischemic colitis (Table 10/4). Stool cultures should include Campylobacter and Salmonella, among others, as well as Clostridium difficile and enterohemorrhagic E. coli (EHEC) if there is a corresponding risk constellation. In the winter months, noroviruses and rotaviruses (children) should be given greater consideration in the case of group diseases.

Diarrhea	Measures
Bloody	Microbiological stool examination, ultrasound of the bowel; colonoscopy
Feverish	Microbiological stool examination, blood cultures, ultrasound of the bowel; consider colonoscopy
During or after antibiotic treatment	Discontinue antibiotics, perform stool culture to test for Clostridium difficile including testing for detection of toxins, ultrasound of the bowel; rectoscopy/colonoscopy (pseudomembranous colitis?)

Table 10/4
Differential diagnostic procedure in the case of acute diarrhea

Chronic diarrhea

Possible causes

Chronic diarrhea can be classified into the following categories according to pathophysiological aspects (see Table 10/5)

- osmotic
- secretory
- inflammatory
- motility disorder-induced
- self-induced

In addition, various medications can cause chronic diarrhea.

However, pathophysiological classification of diarrhea reaches its limit when more complex relationships are involved, for example when there is a mixed clinical picture of osmotic diarrhea and secretory diarrhea. For instance, this occurs in the case of the following types of chronic diarrhea with weight loss:

In **celiac disease** the absorption disorder in the presence of villous atrophy dominates the clinical picture at first glance. At the same time, however, there is a digestive disorder, for example a disorder of digestion of disaccharides. A fat digestion disorder due to a lack of endogenous stimulation of the pancreas (secondary exocrine pancreatic insufficiency) and an emulsification disorder due to a lack of gallbladder contraction are added to this. Weight loss can be aggravated by lack of appetite or intestinal symptoms.

In **Crohn's disease**, and especially in the case of colon involvement, at first glance, inflammatory diarrhea dominates the clinical picture. In case of extensive involvement of the lower small bowel and after resection of the terminal ileum, a loss of bile acid occurs, which cannot be compensated by the liver increasing its de novo bile acid synthesis. This decompensated bile acid loss syndrome leads to steatorrhea (malabsorption of fat) with weight loss. In the case of **stenosis**, bacterial overgrowth with subsequent steator-

Classification	Clinical signs	Disease
Osmotic diarrhea	Decrease in diarrhea due to fasting, massive stools, weight loss, deficiency symptoms, stool osmotic gap	Carbohydrate malabsorption, lactase deficiency, lactulose therapy, sorbitol (chewing gum abuse), fructose and sorbitol intolerance, celiac disease, exocrine pancreatic insufficiency
Secretory diarrhea	Watery diarrhea, does not decrease with fasting, dehydration, possibly systemic effect of hormones, absence of stool osmotic gap	Enterotoxins such as Vibrio cholerae and E. coli, laxatives, bile acid loss syndrome, microscopic colitis Endocrine diseases – Neuroendocrine tumor with carcinoid syndrome (increased secretion of secretion-increasing biogenic amines such as serotonin, etc.) – Gastrinoma (increased secretion of gastric juice plus acid-induced inactivation of pancreatic lipase in the duodenum) – VIPoma (increased secretion of vasoactive intestinal peptide) – Medullary thyroid carcinoma (increased calcitonin secretion)
Inflammatory diarrhea	Fever, abdominal pain, bloody stools	Inflammatory bowel diseases (Crohn's disease, ulcerative colitis, infectious), radiation enterocolitis, eosinophilic gastroenteritis, AIDS-associated infections
Motility disorders	Postoperative diarrhea Alternating diarrhea/constipation Diarrhea with weight loss	History of gastrectomy/vagotomy Irritable gastrointestinal syndrome Hyperthyroidism (usually associated with mild steatorrhea)
Self-induced	Frequently affects women, watery diarrhea with hypokalemia, often general weakness and edemas	Laxative abuse

Table 10/5
Classification of chronic diarrhea

rhea and diarrhea may occur in the prestenotic bowel segment due to a motility disorder. In addition, during inflammatory episodes, diarrhea can be osmotically aggravated in the case of lactose intake. Finally, **lactase deficiency** may occur during and several weeks after an acute disease flare-up. The resulting diarrhea is osmotic in etiology.

Acute radiation enterocolitis is usually the result of acute inflammation of the mucous membrane caused by radiotherapy. Further causes may be associated with the selected irradiation field. If the pancreas was in the irradiation field, the cause of diarrhea may be exocrine pancreatic insufficiency. If the terminal ileum was in the irradiation field, diarrhea is the result of bile acid malabsorption and, if this malabsorption is significant, also subsequent fatty acid malabsorption. Impaired intestinal motility can lead to bacterial overgrowth and thus increase steatorrhea. Finally, 20% of all patients irradiated in the pelvic area suffer from fecal incontinence, which can present as chronic diarrhea.

Practical approach

Medical history

A detailed general medical history (impairment of general condition? flare-ups of fever? weight loss?) and medication history (antibiotics, laxatives [also hidden use], cytostatic agents, non-steroidal anti-inflammatory drugs [NSAIDs] theophylline and digitalis) are required. Other relevant medical history aspects include: duration, frequency, consistency, volume, color, admixtures (blood and mucus?), concomitant symptoms (abdominal pain?), persistence of diarrhea (at night? when abstaining from eating?) (Table 10/6). These questions often allow a tentative diagnosis to be made.

Physical examination

Physical examination is not limited to the abdomen. Indications of anemia, pathological resistance in the abdominal region, and skin symptoms (erythema nodosum, pyoderma gangraenosum, hyperkeratosis, parakeratosis, acrodermatitis, alopecia, etc.) should be taken into consideration. A rectal examination is an essential part of any diagnosis of chronic diarrhea (blood or mucus on the examining finger? stool consistency? sphincter tone? anal fissure?)

Question	Suspicion of...
Small amounts of stool, frequent elimination of small amounts of stool, mucus and blood deposits?	... disease of the distal colon or sigmoid/rectum
Large amounts of stool?	... diseases of the small bowel or pancreas
Watery stools without visible blood, sometimes undigested food residues?	... disease of the small bowel, microscopic colitis
Greasy and glossy, voluminous, foul-smelling stools?	... exocrine pancreatic insufficiency or celiac disease
Diarrhea after consuming milk and dairy products?	... lactase deficiency
Persistent diarrhea after fasting or at night?	... secretory or exudative diarrhea, neuroendocrine diarrhea, laxative-induced diarrhea, bacterial infections, viral infections, inflammatory bowel disease
Decrease of diarrhea after fasting or at night?	... osmotic diarrhea, steatorrhea, food allergy, bile acid loss syndrome, incontinence, possibly bacterial overgrowth
Medication leading to diarrhea?	... drug-induced diarrhea

Table 10/6
Important medical history questions for clarifying the cause of diarrhea

Further diagnostic procedures

The differential diagnostic procedure then depends on the symptoms and the suspected diagnosis (Figures 10/1–10/4; Lankisch et al. 2006).

Tips and tricks

The following list of tips for everyday clinical use does not claim to be exhaustive, but it is intended to help in some difficult situations.

Medical history

Before starting a diagnosis, incontinence ("stool smear") must be addressed.

Chronic diarrhea, such as in the case of celiac disease, does not always start slowly, acute forms of diarrhea do occur!

Osmotic gap
The stool at the end of the intestinal tube is approximately isotonic to the serum (normal stool osmolality approx. 290 mOsm/kg). Osmolality is mainly determined by the electrolytes (Na^+ and K^+ with their respective anions Cl^- or HCO_3^-). In this way, stool osmolality can be roughly estimated: Na^+ and K^+ x 2.

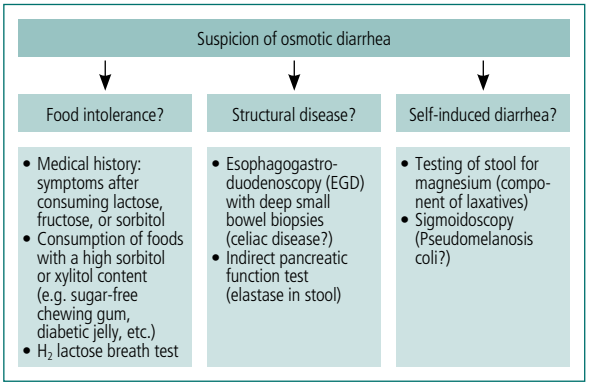

Figure 10/1
Diagnostics in the case of suspicion of osmotic diarrhea

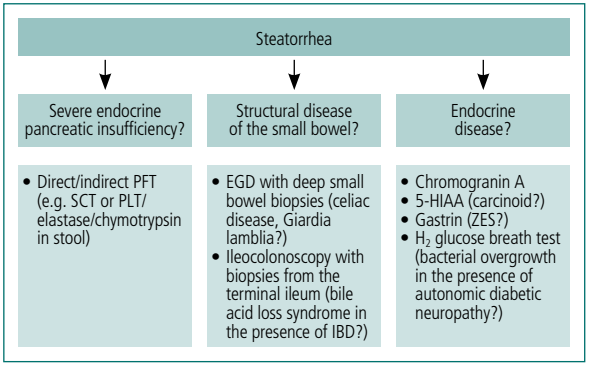

Figure 10/3
Diagnostics in the case of proven steatorrhea (stool fat content > 7 g/day, mean value from three tests)

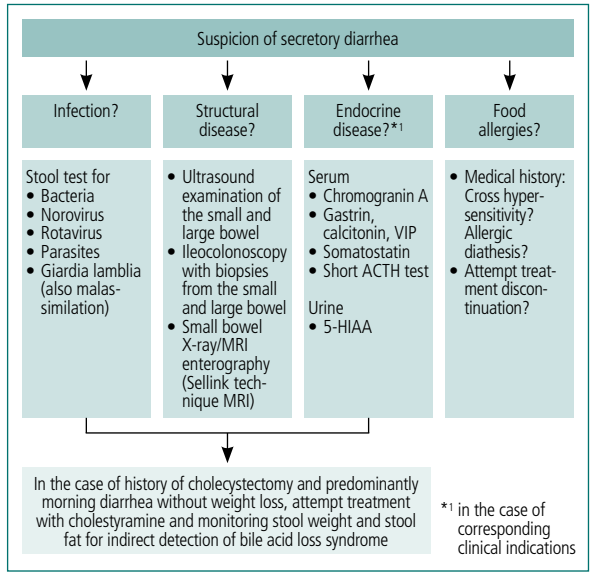

Figure 10/2
Diagnosis in the case of suspicion of secretory diarrhea

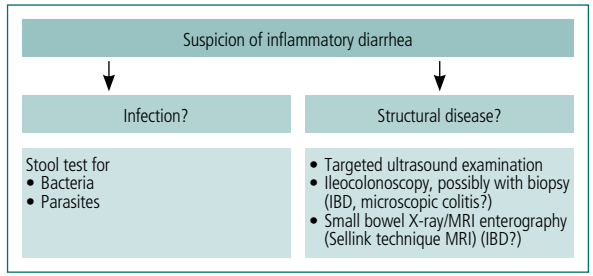

Figure 10/4
Diagnostics in the case of suspicion of inflammatory diarrhea

The *osmotic gap* is the difference between the osmolality of the stool estimated in this way and that measured directly in the laboratory (< 50 mOsm/kg). If the osmotic gap is increased, it can be assumed that unphysiological osmotically active substances are present, and thus malabsorption or use of osmotic laxatives can be assumed. Unfortunately, this valuable examination is dying out in laboratories.

Visual stool examination
Using a "visual stool examination" to detect steatorrhea (normal stool fat content ≤ 7 g/day) is unreliable – only massive steatorrhea (stool fat excretion > 15 g/day) can be detected with the naked eye (Lankisch et al. 1996). However, the examination is useful for the assessment of other factors (e.g. incorrect assessment by the patient, admixtures).

Antibiotic-induced diarrhea

The following forms are possible:

Osmotic diarrhea – frequent, soft, and voluminous stools, typical when taking ampicillin, for instance. The altered colonic flora cannot sufficiently break down carbohydrates in the colon into absorbable short-chain fatty acids.

Secretory diarrhea – dihydroxy bile acids that are not dehydroxylated to secondary bile acids by anaerobic bacteria exert a secretagogue effect in the large bowel.

Pseudomembranous colitis – frequency clearly increasing, can be life-threatening, Clostridium difficile toxins A and B positive in stool (false-negative findings possible in up to 20% of cases). Isolation is expedient (spore flight); hygiene measures!

Segmental hemorrhagic colitis – only after penicillin and penicillin derivatives (probably a hypersensitivity reaction). Acute onset with tenesmus; later hematochezia; characteristic sonographic picture. Restitutio ad integrum in a few days.

Other forms

Diarrhea in the case of many years of poorly controlled **diabetes**:
In the case of visceral neuropathy, two different pathophysiological mechanisms may be at work:
- Stasis symptoms with bacterial overgrowth (antibiotic-responsive) or
- A disorder of postganglionic alpha-adrenergic fibers resulting in secretory diarrhea (secretion > absorption); in the best-case scenario, clonidine therapy will be successful.

Food-induced diarrhea – Sugar substitutes (sorbitol, xylitol, partly also fructose) as well as alpha-glucosidase inhibitors (acarbose, miglitol) can cause osmotic diarrhea (carbohydrate malabsorption) in the case of inadequate dosage or if the sugar intake is inadequate (corresponding H_2 breath tests can be carried out to exclude sorbitol or fructose malabsorption).

Laxative abuse – In Pseudomelanosis coli diagnosed by colonoscopy, there is usually a history of prolonged intake of laxatives (anthraquinones) (**Cave!** also in the case of "liver protection" preparations). If there is a suspicion of laxative abuse as the cause of diarrhea of unclear etiology, but there is no Pseudomelanosis coli, a stool water analysis test for magnesium and bisacodyl can be performed.

Suspicion of **motility-related diarrhea** – Motility-related diarrhea very rarely occurs solely as a result of hypermotility, for example, in the case of irritable colon or infectious enteritis. Other important forms of intestinal dysmotility include pancreaticocibal asynchrony in the case of partial gastric resection, Roux-en-Y anastomosis, and intestinal hypomotility, which always leads to bacterial overgrowth and can be the cause of diarrhea.

Giardia lamblia-induced diarrhea – For the detection of Giardia lamblia, duodenal biopsy and duodenal juice (must be tested warm!) are highly specific and sensitive. The direct detection of Giardia lamblia in the stool using ELISA is particularly indicative in the case of liquid stools. Giardia lamblia should also be taken into consideration in the case of chronic diarrhea and steatorrhea (malassimilation syndrome).

Diarrhea with weakened defense against infection – In the case of acute onset of diarrhea in patients undergoing chemotherapy (toxic intestinal damage vs. infectious genesis) or those with otherwise impaired body defenses against infection, such as hypogammaglobulinemia/agammaglobulinemia, IgA deficiency, AIDS and immunosuppression, infectious diarrhea should be taken into consideration. Please note: Prolonged disease courses are particularly common in these cases. In this case, Cryptosporidium should be given particular consideration. In the case of neutropenic colitis: blood culture.

Medication-induced diarrhea (common in elderly patients), for example induced by antibiotics, metformin, digoxin, colchicine, non-steroidal anti-inflammatory drugs, magnesium-containing antacids, proton-pump inhibitors, HMG-CoA re-

ductase inhibitors, laxatives, selective serotonin reuptake inhibitors, tyrosine kinase inhibitors, immunosuppressants, cytostatic agents.

Microscopic colitis – Especially in the case of watery, non-bloody diarrhea and inconspicuous colonoscopy findings, biopsies are still necessary in order to avoid overlooking microscopic colitis (an umbrella term for collagenous and lymphocytic colitis). Both forms are histologically characterized by an increase in intraepithelial lymphocytes, but in collagenous colitis there is also subepithelial deposition of collagen. Since the disease often manifests in the right side of the colon, random biopsies should also be taken from the right side of the colon.

Celiac disease – The formerly classic symptoms, fatty diarrhea and weight loss, have now faded into the background, but ultimately the symptoms can be very variable and there is almost no symptom that excludes celiac disease. Often, anemia is in the foreground. To be on the safe side, in the case of anemia that cannot otherwise be explained, especially in younger patients, a celiac disease should be ruled out by esophagogastroduodenoscopy with small bowel biopsies (six biopsies from the deep duodenum and the bulb).

The laboratory test for anti-transglutaminase antibodies (or anti-endomysial antibodies) is quite specific in the detection of celiac disease, but duodenal biopsies are mandatory for the diagnosis of *manifest* celiac disease and for the assessment of the extent of the actual architectural disorder.

Account must be taken of the fact that even in specialized institutions, up to 5% of the chronic diarrheal diseases presented remain unexplained, meaning that examinations during the course of the disease may be useful. In such unexplained cases, the possibility of Münchhausen syndrome or factitious diarrhea should also be considered from a differential diagnosis point of view.

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11 Constipation

Definition

- Acute constipation is the absence of defecation for several days after previously regular bowel movements.
- The old definition of chronic constipation: "less than three defecations per week", only does justice to a minority of constipated patients and should no longer be used.
- Chronic constipation is also present if the following additional/other complaints are subjectively reported: insufficient stool volume, hard stool, heavy straining necessary when defecating, feeling of incomplete bowel evacuation.

Possible causes

Pathological mechanisms that lead to constipation can be found at various levels, for example nutrition, intestinal motility and defecation (Table 11/1).

Practical approach

Medical history

History of defecation. Stool diary (e.g. also using the Bristol Stool Form Scale), stool evacuation difficulties? Certain clues in the history may give rise to the suspicion of forms of constipation such as lack of colonic filling, slow colon transit, and functional anorectal obstruction (Table 11/2).

Physical examination

- *General:* complete physical examination, especially digital rectal palpation, evaluation of sphincter tone (resting and voluntary contraction). Search for resistances, judge vial, check whether stool-filled or empty. If appropriate, determine the condition of the stool by visual stool examination. Bowel evacuation disorders can usually be diagnosed by a thorough rectal examination.
- *In particular:* If pelvic floor dyssynergia or functionally effective rectocele is suspected, have the patient bear down. Rectocele often becomes visible or palpable when the pelvic floor is lowered. Internal prolapse also often palpable when the patient is bearing down. In the case of anismus, retraction of the anus when bearing down. In the case of Hirschsprung's disease, wide rectum, no relaxation when bearing down.

Type of constipation and causes	Examples
Acute constipation	
• Obstruction or stricture in the colon	Carcinoma, stricture after e.g. diverticulitis or inflammatory bowel disease (ulcerative colitis, Crohn's disease)
• Anal sphincter spasm	Anal fissure, painful hemorrhoids
• Temporary or situational constipation	Special living conditions: feverish illnesses, being confined to bed, change of diet while traveling, unclean toilets
Chronic constipation	
• Underlying diseases	Hypothyroidism, neuropathy, stenosis, tumor...
• Functional constipation	Irritable bowel syndrome with constipation
• Severe motility disorders	Hirschsprung's disease, inert colon
• Medications	Opiates, calcium antagonists, cytostatic agents (e.g. vinca alkaloids)
• Evacuation disorder	Functional (pelvic floor dyssynergia), structural: e.g. rectocele

Table 11/1
Possible causes of constipation in adults

Laboratory tests

In the case of suspicion of…
- *Inflammation:* CRP, blood count, if necessary differential blood count, calprotectin in stool
- *Electrolyte disorder:* sodium, potassium, calcium (tumor hypercalcemia?)
- *Renal failure:* serum creatinine
- *Endocrine disorder:* blood glucose, HbA_1 (diabetes?), TSH (hypothyroidism?)

Further diagnostic procedures

- Colonoscopy
- CT colonography in the case of incomplete colonoscopy
- Measures that are only occasionally necessary:
  – Colon transit time measurement with radiopaque markers (Hinton test)
  – Anorectal manometry in the case of suspicion of sphincter dysfunction

Clues in the history	Form of constipation
• Alarm symptoms (weight loss, rectal bleeding) present	Tumor-related stenosis (immediate clarification necessary!)
• Low-fiber diet in the diet history	Lack of colon filling
• No spontaneous urge to defecate, abdomen often bloated with a feeling of fullness – Long history – Signs of endocrine or neurological diseases – Drugs that cause constipation – Fiber is ineffective	Slow colonic transit
• Feeling of incomplete evacuation of the rectum – Feeling of a blockage when bearing down – Heavy bearing down despite urge to defecate and soft stool – Manually-aided bowel evacuation, if necessary from the vagina in the case of rectocele – Removal with a finger is often necessary	Functional anorectal obstruction (e.g. rectocele, internal prolapse, anismus)
– Constipation since birth	Ultrashort-segment Hirschsprung's disease

Table 11/2
Clues in the history and form of constipation (modified according to Müller-Lissner 1996)

– Defecography (X-ray or MRI) in case of suspected evacuation disorder
– Rectal barostat in the case of suspicion of hyposensitivity
– Small bowel manometry in case of suspicion of generalized motility disorder
– Colonic manometry in the case of suspicion of inert colon

NB

- The shorter and more alarming the history, the more likely the cause is organic, and the more urgently clarification is required!
- In the vast majority of patients (probably more than 90%) there is no underlying disease responsible for their constipation, and constipation can be dealt with using general measures such as sufficient hydration, exercise, and treatment with bulking agents such as wheat bran or Plantago ovata seed husks (10–30 g/day over 2–4 weeks).

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12 Fecal incontinence

Definition

Involuntary release of rectal contents:
- Grade I: incontinent for flatus
- Grade II: incontinent for liquid stool
- Grade III: incontinent for solid stool
- Special form: isolated stool smearing = release of very small amounts of stool
- Urge symptoms: needing to go to the toilet immediately if there is an urge to defecate, otherwise the stool will escape in an uncontrolled manner due to the insufficient retention function

Possible causes

The prevalence of fecal incontinence is between 0.3 and 1.5%, making this symptom a relevant medical problem. Damage to the continence organ can occur at various levels (see Table 12/1).

Practical approach

(modified according to Madoff et al. 1992; Pehl et al. 2000; Scheurlen et al. 2000)

Medical history

– Stool frequency? Stool consistency?
– Classification of severity of fecal incontinence?
– Are there urge symptoms and/or does involuntary defecation occur?
– Prior conditions?
– Surgery, injuries?
– Prior treatments?

Physical examination

- *Inspection:*
  – Are clothes soiled with stool?
  – Injuries, scars, fissure, fistula, tumor, malformations?
  – Anocutaneous reflex?
- *Digital rectal examination*
  – Circumscribed pain?
  – Resting pressure? Pressure during voluntary contraction?
  – Sphincter defect?
  – Lowering of the pelvic floor?
  – Rectocele, rectal prolapse?
  – Pathological resistance?

Further diagnostic procedures

- *Proctoscopy/sigmoidoscopy*
  – Inspection of the anal canal and the lower rectum, dynamic examination (resting pressure, pressure during voluntary contraction), exclusion of organic diseases such as fistulae, scars, tumors, prolapse, proctitis

Anal sphincter weakness	Traumatic: Birth trauma Surgery (e.g. hemorrhoidectomy, fistulotomy, sphincterotomy) Non-traumatic: Scleroderma, thinning of the internal anal sphincter of unclear etiology Sphincter atresia Neuropathic: Stretch damage, birth trauma, diabetes Medication-induced: Anticholinergics, spasmolytics, calcium antagonists, nitrates, alpha receptor blockers, benzodiazepines
Anatomical changes in the pelvic floor	Fistulae Rectal prolapse Descending perineum syndrome
Anorectal inflammation	Crohn's disease Ulcerative colitis Radiation proctitis Anorectal infection
CNS disorders	Dementia Stroke Cerebral tumors Spinal cord damage Multiple sclerosis Multiple system atrophy (Shy-Drager syndrome)
Intestinal changes	Irritable bowel syndrome Post-cholecystectomy diarrhea Constipation with or without stool impaction Pseudodiarrhea
Postoperative/neoplastic	Anal carcinoma Low-positioned rectal carcinoma Coloanal/deep colorectal anastomosis

Table 12/1
Possible causes of incontinence (modified according to Bharucha et al. 2015)

- *Rectal manometry:* Using a special rectally-inserted catheter, anal pressure and rectal sensory perception, coordination and compliance can be determined. In particular, the following is examined:
- Resting pressure, pressure during voluntary contraction
  – Rectoanal coordination
  – Anorectal sensitivity
  – Rectal compliance
- *Rectal endosonography:* Using a transanal ultrasound transducer with a frequency between 6.5 and 10 MHz, defects of the anal sphincters can be visualized:
  – Externus muscle defect
  – Internus muscle defect
  – Internus muscle myopathy

Further diagnostics

- *Defecogram:* Nowadays, in defecography, the emptying of the bowels with luminal contrast medium is mostly visualized and analyzed by means of MRI.
  In particular, the following is checked:
  – Outflow of contrast medium
  – Emptying function
  – Anal canal length and width
  – Anorectal angle
  – Pelvic floor position
  – Rectocele? Intussusception (= internal invagination of the rectal wall)?
  – Pelvic floor dyssynergia
- *Magnetic resonance imaging (MRI):* MRI is well able to detect pathological changes in anorectal structures, whereas computed tomography does not play an important role
- *Ileocolonoscopy:* This should be performed in particular in cases of difficulty distinguishing between diarrhea and fecal incontinence

NB

- *Medical history:* Involuntary defecation indicates a lesion of the internal anal sphincter muscle (internus) or a sensory disorder; the presence of urge symptoms indicates a disorder in the region of the external anal sphincter muscle (externus).
- *Physical examination:* A normal digital rectal examination does not rule out incontinence.
- *Rectal manometry:* The most common finding is a reduction in the maximum pressure upon voluntary contraction as a manifestation of a disorder of the externus muscle, less frequently there is a reduction in the resting pressure as an indication of an internus muscle disorder.

- *Rectal endosonography:* Approximately two thirds of all fecal incontinence patients exhibit muscular defects in the endosonography. One third of patients with neurogenic fecal incontinence have a muscular defect as an additional cause. The most frequent causes of sphincter defects are injuries during childbirth and surgical interventions in the anal canal. A small number of patients have an internus myopathy.
- *Defecography/MR defecography:* diagnostics of bowel evacuation disorders. Not intended as routine examination.
  Disadvantages:
  – High operator dependency
  – Findings such as rectocele, lowering of the pelvic floor, or internal intussusception are also found in many symptom-free individuals.
  – Wide range of normal values for rectal evacuation
  – Poor correlation of rectal emptying time with symptoms, colon transit or anorectal manometry

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13 Gastrointestinal bleeding

Definition

Bleeding from the gastrointestinal tract can occur in the form of five different clinical manifestations:

- *Hematemesis:* vomiting of "red" blood or "coffee ground"-like substance
- *Melaena:* tarry stool, black, glossy, sticky, foul-smelling stool
- *Hematochezia:* Bloody stool or release of light to dark red blood in the stool, characteristic of a source of bleeding from the lower gastrointestinal tract, also occurs in massive upper gastrointestinal bleeding
- *Occult gastrointestinal bleeding:* no manifest gastrointestinal bleeding. Detection of blood in stool test procedure
- *Symptoms of blood loss or anemia:* feeling dizzy, syncope, angina pectoris and/or dyspnea

Possible causes

In gastrointestinal bleeding, about 85% of the causes are in the upper gastrointestinal tract and about 10% are in the lower gastrointestinal tract (colon). In about 5% of cases, the cause of the bleeding is in the post-duodenal small bowel. The following differentiation has proven to be effective for the localization of bleeding in the everyday clinical setting:

- *Upper gastrointestinal bleeding:* localization is proximal to the ligament of Treitz
- *Middle gastrointestinal bleeding:* localization between the ligament of Treitz and the ileocecal valve
- *Lower gastrointestinal bleeding:* localization in the colon and anus/rectum
- **Common causes of bleeding from the upper gastrointestinal tract** *(anatomically arranged from top to bottom):*
  - Esophageal varices
  - Erosive reflux esophagitis
  - Mallory-Weiss syndrome
  - Gastric varices
  - Gastroduodenal erosions
  - Gastroduodenal ulcers
  - Malignoma
- Rare:
  - Dieulafoy's lesion: arterial bleeding from a solitary small ulcer, mostly located in the gastric fundus with erosion of a submucous artery
- **The most common causes of bleeding from the post-duodenal small bowel, i.e. from the middle gastrointestinal tract (alphabetical order):**
  - Angiodysplasia (most frequent cause: age > 50–60 years)

- Diverticulum, Meckel's diverticulum (most frequent cause of bleeding from the lower gastrointestinal tract in children)
- Inflammatory bowel disease (Crohn's disease)
- Invagination
- NSAID-induced small erosions and ulcers
- Small bowel involvement in the case of infections
- Small bowel ischemia
- Tumors, for example adenocarcinoma, GIST, leiomyoma, lymphoma, benign polyps, neuroendocrine tumors, metastasis
- Varices in the small bowel
- Vasculitis

• **Causes of bleeding from the large bowel (alphabetical order):**
- *Above the anal region*
  - Angiodysplasia
  - Diverticular bleeding
  - Infectious enteritis, for example Campylobacter jejuni and salmonellosis
  - Inflammatory bowel disease
  - Ischemic colitis
  - Malignoma
  - Polyps
  - Rectal ulcer
  - Varices
- *Perianal*
  - Anal fissures
  - Hemorrhoids

To a certain extent, classification by the probability of the cause of acute middle and lower gastrointestinal bleeding can be done according to the age of the patients (Table 13/1).

Children/adults < 25 years	Adults 26–60 years	Adults > 60 years
Inflammatory bowel disease (ulcerative colitis > Crohn's disease)	Diverticulosis	Angiodysplasia
Polyps	Inflammatory bowel disease (ulcerative colitis > Crohn's disease)	Diverticulosis
Heterotopic gastric mucosa in Meckel's diverticulum	Polyps	Carcinoma
	Carcinoma	Polyps
	Infectious colitis	Ischemic colitis
	Angiodysplasia	

Table 13/1
Classification of the causes of acute middle and lower gastrointestinal bleeding according to the age of the patients

Practical approach

Medical history

In an emergency, the history must be taken particularly quickly and in a very targeted manner. Main questions:

- Are any anemia symptoms such as weakness, dizziness, thirst, etc. present?
- To a certain extent, conclusions about the localization of the bleeding can be drawn from the history and – if possible – from inspection of the vomit or stool (Table 13/2)
- Did any symptomatic hypotension/syncope or presyncope occur during the bleeding event?

Physical examination

- *Immediate measurement of vital parameters, especially blood pressure and pulse rate:* In the case of light bleeding, the patient is usually asymptomatic and the circulation is usu-

Medical history	Possible, but by no means certain localization
Upper gastrointestinal bleeding	
• Alcohol abuse	
– with nausea → vomiting → hematemesis?	Mallory-Weiss syndrome
– without nausea or vomiting?	Erosive gastritis (approx. 20% of cases)
– known liver disease?	Esophageal varices in the presence of cirrhosis of the liver?
• Pain when fasting?	e.g. duodenal ulcer
• Heartburn?	Erosive reflux esophagitis
• Medication, especially NSAIDs for joint pain?	Approx. 50% of all patients receiving constant NSAID medication have erosions, and approx. 15–30% have ulcers. They can occur in the esophagus, stomach, and/or duodenum. They may also occur in the post-duodenal small bowel and in the colon
• Trauma, burns, surgery?	Stress ulcer
• Billroth II resection of the stomach (more than 10 years ago)?	Anastomotic ulcer (malignant degeneration possible)
Hemorrhage in the middle or lower GI tract	
• Known ulcerative colitis or Crohn's disease?	Bleeding in the small or large bowel
• Known diverticulosis?	Diverticular bleeding
• Prior stay abroad?	Amebic colitis
• NSAIDs?	Erosive or ulcerative mucosal changes

Table 13/2
Medical history and possible source of bleeding

ally stable. In the case of moderate bleeding, paleness, weakness and orthostatic reactions occur. In the case of heavy bleeding, shock symptoms may occur. A vasovagal syncope with bradycardia can also occur with less bleeding.

- *Determine shock index:* ratio of pulse rate to systolic blood pressure, used to estimate the volume deficit (Table 13/3)

Further diagnostic procedures

- *Laboratory tests*
  - Blood count (hemoglobin, hematocrit)
  - Coagulation status
  - Renal function
  - LFT values
  - Draw blood sample for cross-matching for transfusion if necessary

Value	Blood loss	Evaluation
0.5	< 10%	No shock
1	10–30%	Imminent shock
1.5	> 30–50%	Manifest shock

Table 13/3
Shock index and bleeding

- *In case of suspected upper gastrointestinal bleeding:*
  - Emergency esophagogastroduodenoscopy in the case of clinically relevant bleeding, if necessary after circulatory stabilization, in non-urgent cases routine endoscopy
  - Classification of bleeding activity according to Forrest (F) (Table 13/4)
- *In the case of suspicion of hemorrhage in the middle or lower gastrointestinal tract:*
  - *Ileocolonoscopy* (after colonic lavage if possible, otherwise angiodysplasia may be overlooked)
  - *Push endoscopy* (use pediatric colonoscope if necessary)

FI	Active bleeding	Ia Ib	Spurting arterial hemorrhage Oozing hemorrhage
FII	Inactive bleeding	IIa IIb IIc	Non-bleeding visible vessel Adherent clot Flat pigmented hematin on ulcer base
FIII	Lesion without signs of recent hemorrhage or fibrin-covered clean ulcer base		

Table 13/4
Endoscopic classification of bleeding activity according to Forrest (F)

– *Enteroscopy* (single balloon or double balloon enteroscopy)
– *Selective angiography* in the case of active bleeding (detection and if appropriate, treatment of the source of bleeding – only if blood loss of > 1 mL/min is detected during the examination)
– *Capsule endoscopy* if appropriate (patient swallows a miniature camera – 66% success rate in the case of gastrointestinal bleeding that could not be diagnosed using other methods) (Ell et al. 2002). Suspicion of strictures is a contraindication because the capsule could get stuck, in which case it would then have to be surgically removed. In the case of signs in the history or clinical signs, perform MRI enterography (Sellink technique MRI) prior to capsule endoscopy if appropriate
– If none of the above examinations lead to detection of a source of bleeding, *intraoperative endoscopy* should be considered

• *In the case of occult gastrointestinal bleeding:*
– In the case of symptoms in the upper gastrointestinal tract: *esophagogastroduodenoscopy* in the first instance
– In the case of symptoms in the lower gastrointestinal tract: *ileocolonoscopy* in the first instance, followed by *esophagogastroduodenoscopy*
– If neither of these endoscopic examinations allow detection of a source of bleeding, *capsule endoscopy* or *balloon enteroscopy* should be considered

NB

Medical history

• What appears to be gastrointestinal bleeding may not be:
– Tarry stools after eating blueberries or liquorice, or after taking medications such as activated charcoal, iron or bismuth
– "Red stools" after eating beetroot
• *Bleeding characteristics:*
– Hematemesis is not an obligate symptom of upper gastrointestinal bleeding. When hydrochloric acid hematin occurs, this simply means that blood has come into contact with hydrochloric acid. Therefore, the blood does not necessarily come from the stomach – it may have entered the stomach from the nasopharyngeal cavity. In the case of massive bleeding or achlorhydria, no coffee ground appearance!
– Melaena is usually the result of bleeding from the upper gastrointestinal tract, but bleeding from lower bowel sections can also cause the stool to turn black. All that is re-

quired for melaena to occur is for the blood to remain in the bowel for a sufficient amount of time (> 8 hours) because the black color is caused by the breakdown of the blood by bacteria.
– The darker the blood that is discharged in the stool, the higher up the source of bleeding is located.
– Bleeding from the upper gastrointestinal tract is more likely to be acute, have effects on the circulatory system, and be life-threatening. Bleeding from the lower gastrointestinal tract is more likely to be chronic and less likely to be an acute threat.
– In the case of a heavier rectal blood loss, and especially in the case of circulatory depression, there is also a strong indication for gastroscopy in addition to sigmoidoscopy.
– Diverticular bleeding begins suddenly and painlessly, and often manifests as severe. Diverticula in the right side of the colon are particularly at risk of bleeding. Diverticular bleeding often stops spontaneously, but recurs in 20–25% of cases.
– Thin, watery, tarry stool indicates severe bleeding, possibly arterial bleeding. Prognosis is serious! Well-formed tarry stool indicates less extensive bleeding. The prognosis in this case is less serious.

Physical examination

- Rectal examination is extremely important, both initially and in the further course!
- Shock index: A value < 1 does not rule out hemorrhagic shock.

Further diagnostic procedures

- *Laboratory tests:* The hemoglobin value does not fall immediately in the case of gastrointestinal bleeding. Initially, the patient bleeds "whole blood", i.e. RBCs and plasma volume fall to the same extent. Only once the extravascular fluid enters the blood vessels to compensate for the loss of volume does the hemoglobin level drop; this can take up to 72 hours after the onset of bleeding.
- Elderly patients often have angiodysplasia, often located in the ileocecal region and in the ascending colon.
- Never be satisfied with the suspected diagnosis "bleeding hemorrhoids", always insist on a complete diagnosis (ileocolonoscopy) of rectal bleeding.

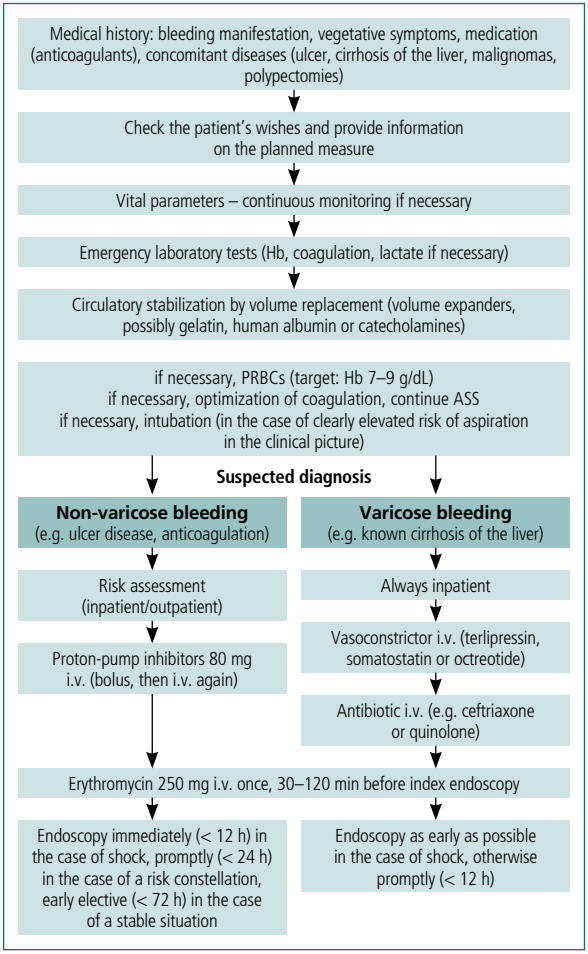

Figure 13/1
Algorithm for pre-endoscopic management (Source: Götz et al. 2017. Reprint with the kind permission of the DVGS and the authors.)

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14 Jaundice

Definition

Yellowing of the skin, mucous membranes, and sclera due to deposition of bilirubin in the tissue. Scleral icterus becomes visible when the total bilirubin increases to more than 2 mg/dL, skin jaundice becomes visible when the total bilirubin increases to more than 3 mg/dL. Skin coloring and light conditions influence these limit values.

Possible causes

For practical purposes, the division into pre-, intra- and post-hepatic jaundice has been proven useful (see also Beckh 2013; Pratt and Kaplan 2015).

Causes of jaundice

Pre-hepatic jaundice

- Increase in unconjugated (indirect) bilirubin:
  - Bilirubin overproduction
    - Hemolysis
    - Dyserythropoiesis
    - Absorption of hematomas

Intrahepatic jaundice

- Isolated increase in unconjugated (indirect) bilirubin:
  - Reduction in hepatocellular bilirubin uptake:
    - Gilbert's syndrome
  - Reduction in hepatocellular bilirubin conjugation:
    - Crigler-Najjar syndrome type I and II
    - Gilbert's syndrome
    - Neonatal jaundice
- Isolated congenital increase in conjugated (direct) bilirubin:
  - Reduction in hepatocellular bilirubin excretion:
    - Dubin-Johnson syndrome
    - Rotor syndrome
    - Benign recurrent intrahepatic cholestasis (Summerskill-Walshe-Tygstrup syndrome)
- Disorders with an increase predominantly in direct bilirubin:
  - Acute (Table 14/1) and chronic liver diseases
    - Toxic (alcohol, other toxins, medications) (Table 14/2)
    - Infectious
    - Autoimmune
  - Malignant infiltration of the liver
  - Intrahepatic cholestasis of pregnancy
  - Metabolic diseases (Wilson's disease, hemochromatosis, alpha$_1$-antitrypsin deficiency)

– Postoperative status
– Total parenteral nutrition
– Primary/secondary biliary cholangitis
– Acute/chronic congested liver

Post-hepatic jaundice

- Choledocholithiasis
- Compression of the bile duct
  – Acute or chronic pancreatitis
  – Pancreatic carcinoma
  – Lymphoma or metastasis of the portal lymph nodes
- Cholangiocellular carcinoma
- Papillary adenoma/carcinoma

In addition:

- Bile duct stricture
- Biliary atresia
- AIDS cholangiopathy
- Parasites (clonorchiasis, ascariasis)

Practical approach

Answering the following questions is crucial:

- Is there an isolated serum bilirubin elevation?
- If yes, is it an elevation of the unconjugated (indirect) or conjugated (direct) bilirubin?
- If other liver values exhibit abnormalities in addition to hyperbilirubinemia, is the jaundice hepatocellular or due to a bile flow disorder?
- If cholestasis is present, is it intrahepatic or extrahepatic in nature?

The following procedure should be used to answer these questions:

Medical history

- Detailed medical history, especially with regard to medication (medications prescribed by the physician or acquired by the patient themselves [over-the-counter]), handling of environmental toxins, parenteral nutrition, transfusions, evidence of intravenous drug abuse, tattooing, sexual intercourse with frequently changing partners, recent stays abroad, living with other jaundice patients, increased alcohol consumption, additional symptoms such as joint pain, myalgia, skin rash, loss of appetite, weight loss, abdominal pain, fever, itching, discoloration of stools and urine, diarrhea

Physical examination

- Signs of chronic liver disease (e.g. spider naevi, palmar erythema, gynecomastia, caput medusae, Dupuytren's contracture, parotid enlargement, testicular atrophy, ascites)?
- Signs of a tumor in the abdominal region (e.g. enlargement of the Virchow's gland in the left supraclavicular region, metastasis in the um-

bilical region [Sister Mary Joseph's nodule, Courvoisier's sign])?
- Signs of right-sided cardiac insufficiency with liver involvement (e.g. congested jugular veins, liver and/or spleen enlargement, ascites, edema)?

Further diagnostic procedures

- *Laboratory tests*
  - *General:* total bilirubin, if appropriate, differentiation between direct and indirect bilirubin, transaminase levels, alkaline phosphatase, gamma-GT, albumin, and prothrombin time. Determination of medication levels if applicable
  - *In the case of hepatocellular damage:* hepatitis serologies. Ceruloplasmin if the patient is under 40 years of age (Wilson's disease?). Iron metabolism (hemochromatosis?)
  - *In the case of suspicion of autoimmune hepatitis:* detection of typical autoantibodies (ANA, SMA, SLA, LKM1) and IgG immunoglobulins
  - *In the case of suspicion of primary biliary cholangitis (PBC):* antimitochondrial antibodies (AMA) and IgM immunoglobulins
  - *In the case of suspicion of primary sclerosing cholangitis (PSC):* p-ANCA

Machine-aided diagnostics

- Ultrasound examination of the liver, with contrast medium if appropriate
- *In the case of liver damage of unknown origin or staging of liver disease:*
  - Hepatic puncture (percutaneous and guided by sonography, or during a laparoscopy)
  - If appropriate, laparoscopy with hepatic puncture or transjugular hepatic puncture
- *If etiology is still unclear*
  - Contrast-enhanced CT
  - Magnetic resonance cholangiopancreatography (MRCP) (especially in the case of PSC)
  - Endosonography of the pancreatic biliary system
  - Endoscopic retrograde cholangiopancreatography (ERCP). Generally only in the case of a therapeutic indication
    Cave! Increased risk of bacterial cholangitis in the case of primary sclerosing cholangitis

NB

- In the case of differential diagnosis of jaundice, the following should be noted: High carotin intake leads to cutaneous icterus, whereas quinacrine can also lead to scleral icterus. The handling of phenols can also lead to cutaneous icterus.

Hepatotropic viruses	HAV, HBV, HCV, HEV, possibly HDV
Non-hepatotropic viruses	HSV, CMV, EBV (VZV, HHV-6, Parvovirus-B19, adenovirus)
Hemorrhagic fever	e.g. yellow fever, dengue fever
Non-viral infections	e.g. leptospirosis, malaria
Immunological	autoimmune hepatitis
Foreign substances	toxins, medications, herbs, lifestyle preparations
Hereditary	Wilson's disease
Perfusion disorders	acute Budd-Chiari syndrome, arterial macrocirculatory disorders (e.g. thrombosis) or microcirculatory disorders (e.g. sickle cell anemia, HUS/TTP)

Abbreviations:
HAV: hepatitis A virus; HBV: hepatitis B virus; HCV: hepatitis C virus; HEV: hepatitis E virus; HDV: hepatitis D virus;
HSV: herpes simplex virus; CMV: cytomegalovirus; EBV: Epstein-Barr virus; HHV: human herpes virus;
HUS: hemolytic uremic syndrome; TTP: thrombotic thrombocytopenic purpura

Table 14/1
Differential diagnosis of acute hepatocellular damage (source: Bahr 2013)

- Clues in the history are not specific, but they can be useful. For example, if jaundice is associated with a sudden pain in the right upper abdomen and chills, this is suggestive of choledocholithiasis and ascending cholangitis. In the case of joint and muscle pain prior to the start of jaundice, the possibility of hepatitis (e.g. hepatitis caused by a virus, medication, or autoimmune conditions) should be taken into consideration.
- In the case of patients with jaundice due to chronic hemolysis, consider the fact that in these patients, pigment (calcium bilirubinate) gallstones occur frequently, and these can also lead to hyperbilirubinemia in the case of choledocholithiasis.
- Patients with Gilbert's syndrome are common. The syndrome is thought to occur in 3–7% of the population, and affects men more frequently than women.
- Patients with liver cell damage mostly respond with a higher increase in transaminase levels compared to alkaline phosphatase levels. Patients with cholestasis exhibit reverse enzyme

Isoniazid	Labetalol
Pyrazinamide	Amiodarone
Sulfasalazine	Allopurinol
Statins	Dapsone
Propylthiouracil	Methyldopa
Imipramine	Abacavir
Ciprofloxacin	Efavirenz
Doxycycline	Valproic acid
Nitrofurantoin	Carbamazepine
Terbinafine	Phenytoin
Ketoconazole	Diclofenac/ibuprofen
Itraconazole	MDMA (ecstasy)
Combination preparations with higher toxicity:	
Trimethoprim sulfamethoxazole	
Rifampin isoniazid	
Amoxicillin clavulanic acid	

Table 14/2
Some medications that can trigger an idiosyncratic reaction, up to and including acute liver failure (adapted from AASLD Position Paper: The Management of Acute Liver Failure: Update 2011)

behavior. Total bilirubin can be elevated under both conditions, and is not helpful in differential diagnosis.

- *Albumin:* A low albumin level is indicative of a chronic process such as cirrhosis of the liver or carcinoma. In the case of normal albumin levels, an acute process such as viral hepatitis or choledocholithiasis is more likely.
- *Prothrombin time:* A prolonged prothrombin time either indicates a vitamin K deficiency (due to longstanding jaundice as a consequence of malabsorption of vitamin K), or a significant liver cell disorder. If the prothrombin time cannot be corrected even with parenteral substitution of vitamin K, the patient has severe liver cell damage.

- In the case of suspicion of primary biliary cholangitis: Diagnosis is established through positive detection of antimitochondrial antibodies in 95% of cases.
- In the case of suspicion of primary sclerosing cholangitis: If detected, remember that about 75% of patients have inflammatory bowel disease, and its course is often subclinical, with primary sclerosing cholangitis occurring at the same time.

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15 Unintentional weight loss

Definition

Clinically relevant if weight loss of 5–10% of total weight is lost within 3 to 6 months (Table 15/1).

Possible causes

The causes of weight loss can be classified according to energy consumption or energy requirements and reduced food intake, with or without increased calorie requirements.
Another option is to classify the cause of the disease according to whether appetite is normal, increased, or decreased, and whether eating is associated with pain (sitophobia), which often leads to reduced food intake and thus weight loss.
Although the causes of weight loss in old age are basically the same as the causes in younger people, special illnesses and conditions need to be taken into account when clarifying the cause of weight loss in older people (Table 15/2).

1. Elevated energy consumption
- Hyperthyroidism
- Cushing's syndrome
- Pheochromocytoma
- Intestinal parasites

2. Elevated energy requirements
- Diabetes
- Malabsorption (celiac disease, chronic pancreatitis, inflammatory bowel disease, short bowel syndrome)

3. Reduced food intake, with or without increased calorie requirements*
- Adrenal insufficiency (primary/secondary)
- Chronic infections (including tuberculosis, endocarditis, AIDS)
- Chronic liver diseases
- Chronic obstructive pulmonary disease
- Dementia
- Depression
- Eating disorders
- ENT and dental disorders
- Gastrointestinal diseases with sitophobia (e.g. angina abdominalis, intestinal ischemia, chronic pancreatitis, pancreatic cancer, gastric carcinoma, peptic ulcers, gallbladder and bile duct diseases, inflammatory bowel disease: Crohn's disease, ulcerative colitis)
- General weakness
- Hypercalcemia (tumors, hyperparathyroidism, sarcoidosis)
- Medication, including chemotherapy (conditioned loss of appetite, conditioned nausea)
- Pernicious anemia
- Poverty
- Severe cardiac insufficiency
- Tumor-related stenosis in the gastrointestinal tract
- Tumors
- Uremia

* Alphabetical order

Table 15/1
Possible causes of weight loss
(Lankisch 2002; Löser et al. 2007; Reife 2015)

1. Dentition
2. Dysgeusia
3. Dysphagia
4. Diarrhea
5. Disease chronic
6. Depression
7. Dementia
8. Dysfunction (social status)
9. Drugs
10. Don't know (unknown cause)

Table 15/2
The nine Ds: Main causes of weight loss in the elderly (Robbins 1989)

Practical approach

- The physician's first task is to determine whether weight loss has really taken place and whether it is really significant according to the definition.
- The focus should be on searching for an additional symptom that allows a relationship with an organ to be found. As a general rule, the following should be considered:
  – Medical history (e.g. eating habits, social status, abdominal pain – especially after eating, diarrhea, etc.)
  – Physical examination
  – Standard laboratory tests including blood count, differential blood count, inflammation parameters, electrolytes, urinary excreted substances, blood glucose, thyroid parameters, protein electrophoresis, and urinalysis
  – Abdominal ultrasound
  – Chest X-ray
  – Esophagogastroduodenoscopy
  – Ileocolonoscopy
  – In the case of weight loss and deterioration or manifestation of diabetes, consider whether pancreatic neoplasia may be present, and if necessary, perform a CT of the abdomen and/or an endosonography of the pancreas
  – In the case of B symptoms, in order to clarify whether lymphoma may be present, if appropriate, perform chest and abdomen CT, as well as lymph node sonography
  – Gynecological consultation, mammography if appropriate

In addition, functional examinations to rule out malassimilation as the cause of weight loss may be useful.

Further examinations with corresponding indications in the history and/or pathological findings:

- If diarrhea is reported, a diagnosis of malassimilation should be ruled out:
  - Duodenoscopy with six duodenal biopsies (from the descending duodenum and duodenal bulb) in order to rule out celiac disease
  - Clarification of whether exocrine pancreatic insufficiency is present
  - D-xylose absorption test (jejunum) if appropriate and vitamin B_{12} absorption test (terminal ileum)
  - If appropriate, perform imaging procedures such as imaging of transit through the small bowel and/or MRI enterography (Sellink technique MRI), video capsule endoscopy, push-and-pull enteroscopy, in order to rule out inflammatory disease in the small bowel
- In unclear cases, a psychiatric examination should be arranged, because a disease from this field (usually depression) may be responsible for the unintentional weight loss. This applies in particular to elderly patients.

NB

- If the information given is unclear on whether weight loss has in fact taken place, it is often helpful to ask whether clothing from before still fits, or whether it is now too big.
- In elderly patients with weight loss, it is essential to check the patient's dental status and oral cavity, because food intake may be inadequate due to the patient having few of their own teeth or having damaged teeth, or a poorly fitted or inadequately cleaned set of dental prosthesis, or due to taste disorders (dysgeusia).
- If a patient has difficulty chewing due to oral problems, it is not uncommon for them to switch from solid food to liquid food, which can lead to diarrhea or weight loss.
- In elderly patients, a bland, but not very tasty diet without any spices that is entered into with the idea that it is healthier can lead to a deterioration of the sense of taste, and thus to a reduced intake of food.
- In elderly patients in particular, it is very important to take a medication history (Table 15/3), because medications such as antibiotics can cause taste disorders, which can in turn lead to reduced food intake.

Side effect	Medication or additive
Anorexia	Amantadine, amphetamines, antibiotics, anticonvulsants, benzodiazepines, digoxin, gold, levodopa, metformin, neuroleptics, nicotine, opiates, selective serotonin reuptake inhibitors (SSRIs), theophylline
Dry mouth	Anticholinergics, antihistamines, clonidine, loop diuretics
Dysgeusia and/or dysosmia	ACE inhibitors, acetazolamide, alcohol, allopurinol, amphetamines, antibiotics, anticholinergics, antihistamines, calcium antagonists, carbamazepine, chemotherapy drugs, chloral hydrate, cocaine, cromoglicic acid, etidronate, gold, hydralazine, hydrochlorothiazide, iron, L-DOPA, lithium, metformin, methimazole, nitroglycerin, opiates, penicillamine, pergolide, phenytoin, propranolol, selegiline, spironolactone, statins, terbinafine, tobacco, triazolam, tricyclic antidepressants
Dysphagia	Alendronate, antibiotics, anticholinergics, bisphosphonates, chemotherapy drugs, corticosteroids, gold, iron, L-DOPA, non-steroidal anti-inflammatory drugs, potassium, quinidine, theophylline
Nausea and/or vomiting	Amantadine, antibiotics, bisphosphonates, digoxin, dopamine agonists, hormone replacement drugs, iron, L-DOPA, metformin, metronidazole, nitroglycerin, opiates, phenytoin, potassium, SSRIs, statins, theophylline, tricyclic antidepressants

Table 15/3
Side effects of medications and additives (in alphabetical order) that may contribute to weight loss (modified according to Alibhai et al. 2005)

- In the elderly, weight loss is one of the most common symptoms of hyperthyroidism, and can be quite extreme.
- In terms of the history, it is crucial to know the patient's social background. The patient should be asked about their ability to supply themselves with adequate nutrition.

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FALK FOUNDATION e.V.

Leinenweberstr. 5
79108 Freiburg
Germany

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