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**Fig. 9.4:** Flow chart for use in clinically suspected “liver tumour” with positive sonographic findings. • Imaging procedures (→) which may be indicated include power-Doppler sonography (PDS), computer tomography (CT), magnetic resonance imaging (MRI) and scintigraphy (SC). • Histological diagnosis is indicated in some cases in order to confirm or exclude imaging diagnosis. (---→)

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**Tab. 9.2:** Indications for hepatobiliary sequential scintigraphy

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**Tab. 10.9:** Assessment criteria for the number-connection test (NCT; part A)

**Tab. 10.10:** Assessment criteria for the line-tracing test and star-construction test

# Symptoms and syndromes

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**Tab. 12.1:** Localization and developmental mechanisms of the various types of jaundice

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**Tab. 12.6:** Main colouration of duodenal bile, stools and urine as well as the results of bile pigment tests in the urine of jaundice patients (Ø = negative)

**Tab. 12.7:** Important laboratory parameters for the differential diagnosis of jaundice

## 13 Cholestasis

**Fig. 13.1:** The hepatocyte as a polar unit. • *Major hepatocellular transport systems:* CM = canalicular membrane, BM = basolateral membrane, TJ = tight junctions, BS<sup>-</sup> = bile salts, OA<sup>-</sup> = organic anions, OC<sup>+</sup> = organic cations, GSH = reduced glutathione, AE<sub>2</sub> = ATP-dependent anion exchange (Cl/HCO<sub>3</sub><sup>-</sup>; GSH), BST = ATP-dependent bile acid transporter, NTCP = sinusoidal Na<sup>+</sup>-dependent taurocholate cotransporting protein, OATP<sub>1</sub> = sinusoidal Na<sup>+</sup>-independent organic anion (and cation) transporter protein, BSEP = bile salt export pump for monovalent bile salts, MRP<sub>2</sub> = canalicular multispecific organic anion transporter (= MOAT), MDR<sub>1</sub> = ATP-dependent organic cation transporter, MDR<sub>2</sub> = ATP-dependent phospholipid transporter (= flippase), MRP<sub>1</sub> = sinusoidal multidrug resistance-associated protein

**Fig. 13.2:** Chronic cholestasis with granuloma-like accumulation of lipid-laden macrophages (xanthoma cells) (HE)

**Fig. 13.3:** Scratch marks resulting from pruritus in recurrent intrahepatic cholestasis

**Fig. 13.4:** Obstructive jaundice with feathery degeneration of hepatocytes (→) (HE)

**Fig. 13.5:** Bilirubinostasis with bile droplets (→) in hepatocytes and canaliculi. Clinical diagnosis: extrahepatic obstructive jaundice (HE)

**Fig. 13.6:** Smooth, brownish-green sprinkled liver surface (due to azathioprine)

**Fig. 13.7:** Flow diagram of cholestasis and jaundice (N = normal, Ø = not present) (s. fig. 9.3)

**Fig. 13.8:** Acholic fatty stool in recurrent intrahepatic cholestasis

**Tab. 3.1:** Forms of cholestasis

**Tab. 3.2:** Causes of extrahepatic obstructive cholestasis (with or without initial jaundice; however, consecutive jaundice may develop later)

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**Tab. 13.8:** Ultrasound criteria for the differentiation of cholestasis (or jaundice)

**Tab. 13.9:** Efficiency of diagnostic procedures in cholestasis: determination of cholestasis (= sensitivity), differentiation between intrahepatic and extrahepatic cholestasis (= specificity)

**Tab. 13.10:** Histological findings as criteria for intrahepatic or extrahepatic cholestasis

**Tab. 13.11:** Treatment of functional cholestasis and pruritus

## 14 Portal hypertension

**Fig. 14.1:** Pathogenesis of portal hypertension

**Fig. 14.2:** Slight lobular inflammation. Periportal/perisinusoidal delicate fibrosis as a result of chronic vitamin A intoxication (same patient as in fig. 14.3) (Ladewig)

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**Fig. 14.4:** Anatomical systematics of portal hypertension

**Fig. 14.5:** Forms, localization and systematics of portal hypertension (resistance-related and volume-related hypertension can occur either as primary or secondary forms)

**Fig. 14.6:** Dilation and meandering of the fine peritoneal vessels in the initial stage of portal hypertension

**Fig. 14.7:** Splenic tumour in portal hypertension following post-hepatic liver cirrhosis

**Fig. 14.8:** Diagram showing the formation of oesophageal varices in portal hypertension (modified from H. EPPINGER, 1937) • OV = oesophageal veins; SC = superior vena cava; SV = splenic vein; PV = portal vein; 1 = coronary gastric vein; 2 = perioesophageal venous plexus; 3 = submucous venous complex in the lower oesophagus; 4 = short gastric veins; 5 = hemiazygous vein; 6 = azygous vein; 7 = venous plexus of the central oesophagus

**Fig. 14.9:** Moderately dilated oesophagus with irregular surface. Numerous, differently sized filling defects as a result of varices

**Fig. 14.10:** Oesophagogastrosocopy: pronounced varicosis (degree of severity III) in the lower third of the oesophagus

**Fig. 14.11:** Postoperative adhesions in the region of the abdominal wall with "spontaneous Talma effect" resulting from portal hypertension

**Fig. 14.12:** Dilation and convolution of the small veins in the region of the round ligament (= teres) with recanalization of the umbilical vein resulting from portal hypertension (s. fig. 6.7)

**Fig. 14.13:** External caput Medusae in liver cirrhosis. • The thick paraumbilical vein (diameter 2 cm) is shown subcutaneously at the exit of the vessel from Glisson's capsule. The colour-encoded vessel with a varicose enlargement at the exit point of the paraumbilical vein from Glisson's capsule is visible immediately below the ventral layers of the abdominal wall

**Fig. 14.14:** Portal hypertensive gastropathy showing linear and patchy erosions

**Tab. 14.1:** Forms and localization of portal hypertension

**Tab. 14.2:** Forms and causes of prehepatic portal hypertension

**Tab. 14.3:** Causes of elevated presinusoidal resistance in intrahepatic portal hypertension (with some references)

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**Tab. 14.6:** Causes of posthepatic portal hypertension

**Tab. 14.7:** Sonographic criteria in portal hypertension

**Tab. 14.8:** Characteristic morphological, haemodynamic and pathophysiological sequelae of portal hypertension

**Tab. 14.9:** Haemodynamic and clinical findings in the 5 localized forms of portal hypertension (elevated hepatovenous pressure gradient = >9 mm Hg; increased risk of oesophageal varix bleeding = >12 mm Hg) (N = normal; Ø = not present)

**Tab. 14.10:** Possible formation of portacaval collaterals

**Tab. 14.11:** Substances for lowering portal hypertension



## 15 Hepatic encephalopathy

**Fig. 15.1:** Branched-chain amino acid cycle and glutamate cycle in the brain (A = astrozyte, N = neuron, BBB = blood-brain barrier, GNT = glutamate neurotransmitter, BCAA = branched-chain amino acids, BCKA = branched-chain keto acids) (137)

**Fig. 15.2:** Diagram illustrating the development of hepatic encephalopathy due to diuretics and/or hypokalaemia

**Fig. 15.3:** Self-monitoring by the patient using a documentation programme for the early detection of subclinical hepatic encephalopathy or onset of oedema (s. fig. 10.1) (s. p. 311)

**Tab. 15.1:** Preconditions for hepatic encephalopathy and its development as a synergy of multiple pathogenic factors

**Tab. 15.2:** Causes of hepatic encephalopathy and hyperammonaemia (frequency in %) (s. fig. 15.2)

**Tab. 15.3:** Clinical forms of hepatic encephalopathy (HE)

**Tab. 15.4:** Normal results in latent (subclinical) stage of PSE

**Tab. 15.5:** Diagram of the stages of hepatic encephalopathy (subclinical and latent forms and manifestation stages I–IV) as well as their respective symptomatology

**Tab. 15.6:** Recommendations for therapy in various forms and stages of HE (Ø = not recommended, + = recommended or important in individual cases, ++ = important)

## 16 Oedema and ascites

**Fig. 16.1:** Fluid spaces and exchange of water (blood plasma + interstitial fluid = extracellular space, intracellular fluid = intracellular space)

**Fig. 16.2:** Pronounced anasarca in portal ascites as a result of alcoholic cirrhosis

**Fig. 16.3:** Fluid exchange between plasma and interstitial tissue (mm Hg)

**Fig. 16.4:** Lymphostasis in the region of the liver capsule of the left liver lobe (lower part) and the falciform ligament

**Fig. 16.5:** Numerous, partially ruptured lymphocysts (light red, dot-like ruptured openings) on the liver surface with extravasation of protein-rich lymph (= *liver weeping*) in alcoholic cirrhosis

**Fig. 16.6:** Diagram of the main pathogenic mechanisms in the formation of ascites according to the four different theories

**Fig. 16.7:** Massive refractory ascites with large umbilical hernia. Muscular atrophy and loss of subcutaneous fatty tissue

**Fig. 16.8:** Right-sided hepatic hydrothorax in liver cirrhosis

**Fig. 16.9:** Chylous ascites with pronounced portal hypertension as a result of posthepatic (HBV) coarse nodular cirrhosis

**Fig. 16.10:** Diagnostic steps used to differentiate between portal, infected, malignant and pancreatogenic ascites (modified from J. SCHÖLMERICH, 1990)

**Fig. 16.11:** Tapping ascitic fluid (1672) (German National Museum, Nürnberg)

**Fig. 16.12:** Diagram of the positioning of a peritoneovenous shunt (with Denver valve)

**Fig. 16.13:** Enormous refractory ascites in alcoholic cirrhosis. Bilateral inguinal hernia with scrotal oedema. Muscular atrophy. Hepatic encephalopathy (II–III) (same patient as in fig. 16.14)

**Fig. 16.14:** Retrogression of ascites and oedema, increasing stabilization of biochemical and physical findings 16 weeks after placement of a LeVeen shunt (→) (survival time 45 months with two shunt recanalizations). (same patient as in fig. 16.13)

**Fig. 16.15:** Placement of a TIPS (according to M. RÖSSLE et al., 1989)

**Fig. 16.16:** Sonographic evidence of a TIPS (arrow) in a hypo-echoic hepatic mass with the typical texture of cirrhosis (VP = portal vein; RL = right liver lobe)

**Fig. 16.17:** Steps in conservative and invasive or surgical treatment for hepatogenic ascites

**Tab. 16.1:** Constituents of the most important electrolytes (in mval/l) in extracellular and intracellular fluid

**Tab. 16.2:** Causative factors of oedematization

**Tab. 16.3:** Mechanical factors in the formation of ascites

**Tab. 16.4:** Stages of lymph vessel insufficiency with cirrhosis (s. fig. 16.4)

**Tab. 16.5:** Predominant changes in the concentration of biochemical factors in the blood and urine in portal ascites (N = normal)

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**Tab. 16.8:** Differentiation of ascites according to its severity

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**Tab. 16.11:** Basic therapy of ascites (stage I)

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**Tab. 16.13:** Pathogenetic or causal factors which may explain apparent resistance to ascites therapy

**Tab. 16.14:** Indications for a peritoneovenous shunt

**Tab. 16.15:** Absolute and relative contraindications for peritoneovenous shunt implantation

**Tab. 16.16:** Early and late complications following peritoneovenous shunt implantation (with some references)

**Tab. 16.17:** Risk reduction criteria for PVS

**Tab. 16.18:** Necessary measures for the patient and medical check-ups after placement of PVS

**Tab. 16.19:** Surgical attempts to eliminate refractory ascites (s. also tab. 19.7!)

## 17 Hepatorenal syndrome

**Fig. 17.1:** Hypothetic pathogenesis of the hepatorenal syndrome in cirrhosis

**Tab. 17.1:** Synopsis of the activity of biochemical factors in the liver, plasma and kidneys or urine relating to the hepatorenal syndrome (with some references)

**Tab. 17.2:** Risk and predictive factors in patients with cirrhosis and ascites regarding the development of HRS

**Tab. 17.3:** Severe liver diseases with the occurrence of a hepatorenal syndrome (with some references)

**Tab. 17.4:** Urine findings for the differential diagnostic definition of the hepatorenal syndrome – provided that there has been no previous diuretic therapy. • Generally, however, this has proved necessary, which could be a reason for the considerable differences in the factors and figures reported in the literature

**Tab. 17.5:** Coexistent disease of the liver and kidneys (comorbidity), so-called pseudohepatorenal syndrome (H. O. CONN, 1973)

**Tab. 17.6:** Forms of secondary kidney damage in the course of hepatobiliary diseases

## 18 Hepatopulmonary syndrome

**Fig. 18.1:** Possible mechanisms in the pathogenesis of the hepatopulmonary syndrome. (VEGF = vascular endothelial growth factor, iNOS = inducible nitric oxide synthetase, TNF = tumour necrosis factor)

**Tab. 18.1:** Liver diseases associated with the hepatopulmonary syndrome (with some references). • Portal hypertension is considered to be an essential factor in the pathogenesis of HPS.

**Tab. 18.2:** Biological effects of endothelin

**Tab. 18.3:** Main properties of bosentan

## 19 Coagulopathy and haemorrhage

**Fig. 19.1:** Exogenous and endogenous plasmic coagulation cascade (with inhibition by antithrombin III and protein C<sub>a</sub>) and the fibrinolysis pathway through plasminogen-plasmin. Proteolytic plasmin principally hydrolyzes the cross-linked fibrin clot into high molecular weight fragments and D fragment dimers (Ph = phospholipids) (s. tab. 5.12)

**Fig. 19.2:** Schematic course of consumptive coagulopathy

**Fig. 19.3:** Haemorrhagic erosions in the stomach as the cause of bleeding

**Fig. 19.4:** Haemorrhagic gastritis in thrombopenia

**Fig. 19.5:** Angiodysplasia in the duodenum

**Fig. 19.6:** Oesophageal varices with red wall signs

**Fig. 19.7:** Endosonographic detection of fundus varices (↑)

**Fig. 19.8:** Fresh bleeding of the oesophageal varices (first bleeding)

**Fig. 19.9:** *Torsade de pointes*: The QT interval represents the phase of myocardial spread of stimulus and repolarization. Excessive QT lengthening may be caused by certain drugs or electrolyte imbalance. In addition, a U wave can occur, whereby its amplitude exceeds the T wave in  $V_4$ – $V_6$ . Subsequently, a potential life-threatening arrhythmia of type *torsade de pointes* may develop. Clinical symptoms include vertigo and syncope. This arrhythmia can spontaneously disappear, but also pass into ventricular fibrillation and thus end fatally

**Fig. 19.10:** Large varix in the gastric cardia

**Fig. 19.11:** Pronounced oesophageal varicosis: indication for ligation

**Fig. 19.12:** Pronounced oesophageal varicosis: condition after ligation

**Fig. 19.13:** Sengstaken-Blakemore tube (R. W. SENGSTAKEN, A. H. BLAKEMORE, 1950): 1. oesophageal tube, 2. gastric tube, 3. gastric balloon tube, 4. oesophageal balloon tube

**Fig. 19.14:** Linton-Nachlas tube (R. R. LINTON, 1953; M. M. NACHLAS, 1955; L. BERTRAND, H. MICHEL, 1969): 1. oesophageal tube, 2. gastric tube, 3. balloon tube

**Fig. 19.15:** Flow diagram: therapeutic spectrum for acute upper gastrointestinal bleeding, including bleeding oesophageal and gastric varices

**Fig. 19.16:** Colonic varices in alcohol-toxic cirrhosis

**Tab. 19.1:** Haemostasis parameters showing the normal range and corresponding changes in the course of hepatogenic consumption coagulopathy up to decompensation (s. tab. 5.12)

**Tab. 19.2:** Therapy of haemostasis disorders

**Tab. 19.3:** Forms and classification of upper gastrointestinal haemorrhage, including the classification according to FORREST

**Tab. 19.4:** Causes of upper gastrointestinal bleeding (with some references and frequencies)

**Tab. 19.5:** Significant negative prognostic risk factors in upper gastrointestinal bleeding

**Tab. 19.6:** Relevant parameters as distinct risk factors for assessing the haemorrhagic tendency of oesophageal varices

**Tab. 19.7:** Overview of semi-invasive and surgical procedures for bleeding oesophageal varices and portal hypertension in chronological order (1874–1994). For reasons of simplification, only the first authors are named in each case. (see also tab. 16.18!)

## 20 Acute and chronic liver insufficiency

**Fig. 20.1:** Sugillations, ecchymoses and petechial haemorrhages in liver cirrhosis (with “paper money skin” and white nails) (s. figs. 4.8, 4.19)

**Fig. 20.2:** Extensive purpura in the abdominal area with bleeding into the cholecystectomy scar

**Fig. 20.3:** Centrilobular, two-week-old liver cell necrosis resulting from paracetamol intoxication (HE)

**Fig. 20.4:** Diagram showing the molecular adsorbent recirculating system (MARS)

**Fig. 20.5:** Diagram showing a bioartificial liver (BAL) (98)

**Tab. 20.1:** Characteristics and prognosis of acute liver failure and its subtypes (modified according to J. G. O’GRADY et al., 1993) (N = normal)

**Tab. 20.2:** Various causes of acute liver failure (with some references)

**Tab. 20.3:** Criteria for a poor prognosis in acute liver insufficiency

**Tab. 20.4:** So-called “minor signs” of chronic liver insufficiency

**Tab. 20.5:** Albumin-bound substances (ABS) relevant in acute liver failure

## Clinical aspects of liver diseases

### 21 Clinical and morphological principles

**Fig. 21.1:** Non-specific reactive hepatitis in sepsis due to cervical lymph node abscess: sparse single cell necrosis and periportal inflammation (HE)

**Fig. 21.2:** Adaptive changes of hepatocytes due to hyperplasia of the smooth endoplasmic reticulum. Several binuclear hepatocytes (BH). Small Kupffer cell nodule following cell necrosis (↑) (HE)

**Fig. 21.3:** Lipofuscinosis (metachromatic-red cytoplasmic pigments) following abuse of analgesics of the phenacetin type (Ladewig)

**Fig. 21.4:** Ballooned hepatocytes with intracellular bilirubino-stasis (Prp) (HE)

**Fig. 21.5:** Cholesterol ester storage disease (CESD). Micro-/macrovesicular fat droplets in hepatocytes and foam cells in a portal tract (Sudan III)

**Fig. 21.6:** Brownish ceroid (“decomposition pigment”) in nested macrophages together with posthepatic late-phase nodule (HE) (s. p. 424)

**Fig. 21.7:** Carbon pigment deposits in portal macrophages. Clinical diagnosis: pronounced anthracosis (HE)

**Fig. 21.8:** Peliotic sinus dilatation in Osler’s disease with small liver trabeculae (►) (HE)

**Fig. 21.9:** Sarcoid granulomas (miliary type, up to the size of a lentil) on the liver surface (right liver lobe)

**Fig. 21.10:** Fibrosing epithelioid cell granuloma containing giant cells in sarcoidosis (HE)

**Fig. 21.11:** Degenerated hepatocyte with either an intracellular lymphocyte, which is often surrounded by a narrow clear halo (= *emperipolesis*), or a lymphocyte invaginated in the cell membrane (= *peripolesis*) – it is perhaps a T lymphocyte. This condition points to immunologically induced liver damage in florid virus hepatitis B (HE)

**Fig. 21.12:** Biochemical causative mechanisms of hepatocellular degeneration and cell death due to oxidative stress and disruptions of cellular calcium homeostasis (similar to a vicious circle)

**Fig. 21.13:** Atrophy of left liver lobe due to acute viral hepatitis B

**Fig. 21.14:** Periportal and septal fibrosis following severe acute viral hepatitis B: clearly disrupted liver architecture; older collapse fields with condensed reticular fibres (Gomori’s reticulin stain)

**Fig. 21.15:** Pericellular trabecular fibrosis with a wire mesh pattern (= *chicken-wire fibrosis*) due to chronic alcohol abuse (Sirius red)

**Fig. 21.16:** Perivenular and perisinusoidal fine-meshed fibrosis and discreet cellular inflammatory reaction as a result of chronic alcohol abuse (ASH) (CV = central vein) (Ladewig)

**Tab. 21.1:** Possible causes of the formation of granulomas in the liver (s. figs. 21.9, 21.10; 24.3, 24.4, 24.8, 24.14; 29.6, 29.7)

**Tab. 21.2:** Some cytokines and peptides as well as chemical or herbal substances with profibrogenetic and antifibrogenetic effects

**Tab. 21.3:** Classification of regenerative lesions

**Tab. 21.4:** Classification of dysplastic lesions

**Tab. 21.5:** Classification of primary malignant liver tumours

**Tab. 21.6:** Classification of secondary malignant liver tumours

### 22 Acute viral hepatitis (A–SEN)

**Fig. 22.1:** Acute viral hepatitis A: Periportal inflammation and periportal cell loss (HE)

**Fig. 22.2:** Subsiding acute viral hepatitis A with lytic loss of hepatocytes and round-cell infiltrates (HE)

**Fig. 22.3:** Kupffer cell nodules (arrow) in subsiding acute viral hepatitis B; hydropic hepatocytes, often binuclear (HE)

**Fig. 22.4:** Feathery degeneration (↑) of ballooned hepatocytes, massive liver cell oedema and canalicular bilirubinostasis. Clinically: cholestatic course of acute viral hepatitis B (HE) (s. fig. 13.4)

**Fig. 22.5:** Numerous multinuclear giant cells (centre of picture) with bile duct proliferation (arrow) in giant-cell hepatitis (HE)



**Fig. 22.6:** Syndrome of acute hepatitis (s. tab. 5.16)

**Fig. 22.7:** HBsAg in the cytoplasm of hepatocytes (arrows). Clinically: HBsAg carrier (former drug abuse) with moderately increased transaminases (immunoperoxidase reaction). These findings correspond to the ground glass cells in HE (s. figs. 5.7; 22.8)

**Fig. 22.8:** Immunohistochemical detection of HBcAg in the nuclei of liver cells shown by monoclonal antibody. Hepatocytes with ground glass-like homogenization of the cytoplasm, so-called ground glass cells (arrow). HBsAg in the cytoplasm is not presented immunohistochemically in this case (s. figs. 5.7; 22.7)

**Fig. 22.9:** Screening of pregnant women in the final trimester with indications for simultaneous vaccination of the neonate

**Fig. 22.10:** Clinical courses of disease in the HBsAg carrier status (s. tabs. 5.17, 5.18) (s. p. 122)

**Fig. 22.11:** Preliminary tests and measures in the event of exposure to or infection with HAV or HBV

**Fig. 22.12:** Preliminary tests prior to active vaccination against hepatitis B (s. tab. 5.17)

**Fig. 22.13:** Late stage of acute viral hepatitis B: moderate round-cell infiltration of the lobular parenchyma with small aggregates of Kupffer cells and Kupffer cell activation; slight disarray of liver cell trabeculae; minimal canalicular cholestasis (→); liver cell mitosis (►) and binuclear hepatocytes (HE)

**Fig. 22.14:** Scarred area with fragmented parenchymal islets (→) after confluent necrosis in the wake of viral hepatitis B (HBsAg+). Localized round-cell infiltration in the remaining parenchymal areas (HE)

**Fig. 22.15:** Large sunken scar plate in the area of the left lobe of liver (about 6 × 4 cm) subsequent to massive dystrophic liver parenchymal necrosis due to severe viral hepatitis B

**Fig. 22.16:** Massive atrophy of the left liver lobe with pronounced capsular callosity following severe acute viral hepatitis B. This finding was completely misinterpreted when using sonography. (s. fig. 21.13; 35.1, 35.17)

**Fig. 22.17:** Diagram of the genetic organization and structure of HCV

**Fig. 22.18:** Structure of the hepatitis D virus

**Fig. 22.19:** Diagram of the immune status in the course of coinfection and superinfection with HDV

**Fig. 22.20:** Possible phylogenetic scheme of the HCV (*Hepaciviridae*) and GB viruses (especially GBV C/HGV) as well as other members of the *Flaviviridae* (I): DV = Dengue virus, WNV = West Nile virus, JEV = Japanese encephalitis virus, YFV = yellow fever virus, or *Pestivirus* (II): BVDV = bovine viral diarrhoea virus, HCHV = hog cholera virus (mod. J.N. SIMONS et al., 2000)

**Tab. 22.1:** Morphological changes with acute viral hepatitis

**Tab. 22.2:** Extrahepatic manifestations with acute viral hepatitis A (with some references) (s. tabs. 22.6, 22.8)

**Tab. 22.3:** Geographical distribution of HBV genotypes (areas of predominance)

**Tab. 22.4:** Known HBV mutants and their possible biomolecular repercussions

**Tab. 22.5:** HBsAg and HBV-DNA detection in body secretions and excretions (with some references)

**Tab. 22.6:** Spectrum of laboratory parameters in acute viral hepatitis B, depending on the degree of severity and specific courses of disease (N = normal)

**Tab. 22.7:** Extrahepatic manifestations in viral hepatitis B (with some references) (s. tabs. 22.2, 22.9)

**Tab. 22.8:** Main geographical distribution of the HCV genotypes

**Tab. 22.9:** Extrahepatic manifestations in viral hepatitis C (with some references) (s. tabs. 22.2, 22.7)

## 23 Acute concomitant viral hepatitis

**Fig. 23.1:** Agglomeration of activated Kupffer cells (partially beaded), especially in the sinusoidal vessels, with single-cell necrosis. Clinical diagnosis: hepatitis mononucleosa (HE)

**Fig. 23.2:** Acute herpes simplex virus hepatitis with intranuclear bodies (Cowdry type A) (→) (HE)

**Fig. 23.3:** Herpes zoster. Pronounced concomitant hepatitis: GPT 186 U/l, GOT 132 U/l, GDH 8.3 U/l, γ-GT 56 U/l, cholinesterase ↓; alkaline phosphatase and bilirubin normal

**Fig. 23.4:** Portal inflammation and acute parenchymal necrosis (N) due to shock (only periportal hepatocytes are intact). Clinical diagnosis: yellow fever (HE)

**Fig. 23.5:** Portal (left) and parenchymal (right) round-cell inflammation. Clinical diagnosis: Ebola fever (HE)

**Tab. 23.1:** Secondary hepatotropic viruses causing viral hepatitis. • In Germany, **obligation for notification** is given in cases of suspicion (S), disease (D) or exitus (E). • But this varies from country to country. *If in doubt*, contact the Public Health Department!

**Tab. 23.2:** Significant exotic hepatotropic viruses which can cause hepatic damage. • In Germany, the **obligation for notification** is given in cases of suspicion (S), disease (D) or exitus (E). • This can, however, vary from country to country. *If in doubt*, contact the Public Health Department!

## 24 Bacterial infections and the liver

**Fig. 24.1:** Markedly altered, swollen hepatocytes. Focal intralobular accumulation of Kupffer cells, histiocytes and neutrophilic leucocytes. Clinical diagnosis: streptococcal sepsis (HE)

**Fig. 24.2:** Fitz-Hugh-Curtis syndrome: perihepatitis with violin string-like adhesions in gonorrhoeal infection

**Fig. 24.3:** Granuloma-like, lymphohistiocytic infiltrate in an area of parenchymal loss. Clinical diagnosis: typhus abdominalis (HE)

**Fig. 24.4:** Tuberculous epithelioid cell granuloma with Langhans' giant cells and small central necrosis (N); additional steatosis of hepatocytes (HE)

**Fig. 24.5:** Small nodular hepatic tuberculosis: foci, the size of a millet seed up to that of a lentil, in the right lobe of liver

**Fig. 24.6:** Old intrahepatic tuberculoma. Encapsulated eosinophilic necrosis with marked caseation (HE)

**Fig. 24.7:** Acid-fast bacilli of mycobacterium tuberculosis in the liver of an AIDS patient (Fite staining)

**Fig. 24.8:** Lepira: granuloma-like, lymphohistiocytic infiltration in the liver parenchyma (HE)

**Fig. 24.9:** Enlargement of hepatocytes with large nucleoles (arrow), distinct icterus with (green) bile thrombi in the biliary capillaries and hepatocytes. Multivacuolar steatosis of isolated liver cells. Groups of histiocytes (left) with phagocytized nuclear material and lipofuscin. Clinical diagnosis: Weil's disease (HE)

**Fig. 24.10:** Lues connata: Severe inflammatory infiltration (especially left of picture); sinusoidal fibrosis; irregular liver cell plates (far right of picture). Also called "interstitial syphilitic hepatitis" (HE)

**Fig. 24.11:** Syphilis, secondary stage: hepatitis syphilitica. In the liver parenchyma, massive amounts of corkscrew-shaped syphilis pathogens, 6–10 μm in length, are visible (= treponema pallidum) (silver impregnation)

**Fig. 24.12:** Hepatic gumma from syphilitic hepar lobatum. Intrahepatic necrotic zones surrounded by a thin layer of granulation tissue (Sirius red)

**Fig. 24.13:** Dense, granulocytic infiltration in the environment of fresh hepatocellular necrosis (right lower half of the picture). Clinical diagnosis: listeriosis (HE)

**Fig. 24.14:** Two intraparenchymal granulomas, one of which has the appearance of a lipogranuloma (→). Clinical diagnosis: Q fever (HE)

**Tab. 24.1:** Pathomechanisms of liver involvement in different bacterial diseases

**Tab. 24.2:** Morphological reactions of the liver following various bacterial infections

**Tab. 24.3:** Various bacterial organisms causing liver damage. In Germany, **obligation for notification** is given in cases of suspicion (S), disease (D), exitus (E), or perinatal infection (P). But this varies from country to country. *If in doubt*: contact the Public Health Department!

## 25 Parasitic infections and the liver

**Fig. 25.1:** Ultrasonographic visualization of an amoebic abscess in the right lobe of liver (s. fig. 9.2)

**Fig. 25.2:** Amastigotes of *Leishmania Donovanii* in swollen Kupffer cells (→) (HE)

**Fig. 25.3:** Malarial pigment: marked haemozoin deposition in the hepatic macrophages (HE)

**Fig. 25.4:** Granuloma close to a portal field. Clinical diagnosis: Toxoplasmosis (HE)

**Fig. 25.5:** Egg of *Ascaris lumbricoides* in bile fluid (obtained by means of a nasogastroduodenal probe)

**Fig. 25.6:** X-ray with contrast medium showing two ascarids in the duodenum

**Fig. 25.7:** Passage of the ascaris pair and three young ascarids (not shown by the X-ray above) through the intestine following anti-helminthic treatment

**Fig. 25.8:** Sonographic visualization of an *Ascaris lumbricoides* in the common bile duct (longitudinal/transverse imaging). Clinical findings: colicky pain, cholestasis and slight jaundice

**Fig. 25.9:** Egg of *Clonorchis sinensis* in bile fluid (obtained by a nasogastroduodenal probe)

**Fig. 25.10:** Chinese liver fluke (*Clonorchis sinensis*)

**Fig. 25.11:** *Fasciola hepatica* (large liver fluke), 2.9 × 1.1 cm (× 3).  
• Ovum of *Fasciola hepatica*, approx. 0.13 × 0.07 mm, in bile fluid (obtained by means of a nasogastroduodenal probe) (× 400)

**Fig. 25.12:** Large intestinal fluke (*Fasciolopsis buski*)

**Fig. 25.13:** Schistosomiasis: cicatricial granulomas with lamellar walls; perifocal lymphohistiocytic inflammatory rim (HE)

**Fig. 25.14:** *Schistosomiasis*: portal fibrosis (so-called clay pipe-stem fibrosis) (HE)

**Fig. 25.15:** Morphology of a hydatid cyst (*E. cysticus*) in the liver

**Fig. 25.16:** *Echinococcus cysticus* (stage IIB): bizarre conglomerate with peripheral daughter cysts

**Fig. 25.17:** CT showing *Echinococcus cysticus* in the liver

**Fig. 25.18:** Calcified *Echinococcus cysticus* in the liver

**Fig. 25.19:** Laparoscopic view of a large *Echinococcus cysticus* hydatid in the right lobe of liver: irregularly thickened hypervascular capsule. Fibrin deposition at the adjacent peritoneum. Distended elastic consistency

**Fig. 25.20:** Hydatid cyst of *Echinococcus cysticus* in the spleen

**Fig. 25.21:** Laparoscopic view of multiple *Echinococcus alveolaris* hydatids in the right lobe of liver

**Fig. 25.22:** *Echinococcus alveolaris* hydatid: pseudocyst surrounded by a rim of radially arranged histiocytes. Markedly damaged adjacent liver parenchyma. Small parasitic membranes in the bright lumen of the pseudocyst (arrow) (HE)

**Tab. 25.1:** Parasitic diseases caused by protozoiasis and helminthiasis which may lead to liver involvement or concomitant liver disease. The **obligation for notification** in Germany is given in cases of suspicion (S), disease (D), exitus (E) or perinatal infection (P). But this varies from country to country. *If in doubt, contact the Public Health Department*

**Tab. 25.2:** Key features of imaging techniques for the diagnosis of biliary ascariasis

## 26 Mycotic infections and the liver

**Tab. 26.1:** Predisposing factors for mycosis of the liver and biliary tract

**Tab. 26.2:** Fungal species causing hepatobiliary organ mycosis in the presence of predisposing factors

**Tab. 26.3:** Morphological findings in mycosis of the liver and biliary tract

## 27 Liver abscess

**Fig. 27.1:** Multiple hypoechoic microabscesses (A) in the right liver lobe (K = kidney)

**Fig. 27.2:** CT scan shows multiple pyogenic liver abscesses in segment 4 (a and b) following perforation in diverticulitis (*E. coli*). Full recovery

**Tab. 27.1:** Various access routes leading to the development of liver abscesses

**Tab. 27.2:** Main pathogens (bacteria, protozoa, helminths, fungi) of liver abscesses and microabscesses (with some references) (see chapters 24, 25 and 26)

**Tab. 27.3:** Underlying diseases and other causes relating to the development of liver abscesses (with some references)

**Tab. 27.4:** Laboratory parameters in liver abscess(es) depending on the degree of severity, course of disease and involvement of the biliary tract

## 28 Alcohol-induced liver damage

**Fig. 28.1:** Important alcohol-related effects via lipid peroxidation and acetaldehyde leading to the development of different forms of ALD

**Fig. 28.2:** Mallory-Denk bodies: immunohistochemical reaction with an antibody against ubiquitin (arrow)

**Fig. 28.3:** Hydropic-degenerated hepatocyte with Mallory-Denk body (→) (HE)

**Fig. 28.4:** Hepatocyte with megamitochondria (→) in alcoholic fatty liver (HE)

**Fig. 28.5:** Siderogranular deposits (↑) in hepatocytes (siderosis) in chronic alcohol abuse (HE)

**Fig. 28.6:** Massive fatty liver (4,750 g hepatic wet weight) after acute alcohol intoxication ("brandy drinking bet"); exitus four days later

**Fig. 28.7:** Alcohol-induced periportal and centrilobular fibrosis, partially spider leg-like (Sirius red) (s. fig. 21.14)

**Fig. 28.8:** Meshwire fibrosis in alcoholic steatohepatitis (Sirius red)

**Fig. 28.9:** Micronodular alcohol-induced liver cirrhosis

**Fig. 28.10:** Chronic moderate periportal and portal inflammatory reaction with septal fibrosis and centrilobular steatosis in chronic alcoholic liver damage (DD: mild chronic viral hepatitis C!) (van Gieson)

**Fig. 28.11:** Septate liver fibrosis without cirrhotic transformation in chronic alcohol abuse (misinterpreted, however, as cirrhosis in sonographic examination)

**Fig. 28.12:** Spectrum and course of alcoholic liver diseases (—→ fulminant course)

**Fig. 28.13:** Alcoholic steatohepatitis. Satellitosis of neutrophilic granulocytes surrounding hydropic-degenerated hepatocytes with Mallory hyalin (→) (HE)

**Fig. 28.14:** Chronic alcohol-induced fatty liver hepatitis: scattered light reflection due to irregular surface; brick-red colour with red-dish-speckled pattern; reticular fibrosing process with enhanced vascularization of the capsule

**Fig. 28.15:** Alcohol-related complete liver cirrhosis: sonographically spotted coarsening of the structure with a distinctly wavy edge (arrows) (A = ascites)

**Fig. 28.16:** Alcoholic cirrhosis with portal hypertension: stenosis of the portal vein (see arrow) with stagnation of blood flow in the portal vein (VP) and portal flow reversal (blue = hepatofugal) as well as enhanced arterial flow (red). Arterial signals are visible in the flow profile. Inhomogeneous liver structure

**Fig. 28.17:** Alcoholic cirrhosis with parenchymal steatosis and slight cholestasis (Sirius red)

**Fig. 28.18:** Alcohol-induced cholestasis in a fatty liver

**Fig. 28.19:** Severe alcohol-induced cholestasis syndrome in alcoholic hepatitis. Dilated lymph vessel due to lymphostasis (↓)

**Tab. 28.1:** Types of alcoholism (E.M. JELLINEK)

**Tab. 28.2:** Significant alcohol-induced metabolic disturbances and changes in biochemical reactions in the liver

**Tab. 28.3:** Causes of interaction between alcohol and xenobiotics, particularly medicinal preparations

**Tab. 28.4:** Pathogenetic factors responsible for alcoholic liver disease

**Tab. 28.5:** Laboratory parameters in chronic alcohol-induced liver disease (N = normal)

**Tab. 28.6:** Biomarkers (plus detectability time) and laboratory parameters giving evidence of a single intake of alcohol (5-HTOL = 5-hydroxytryptophol; 5-HIES = 5-hydroxyindolacetic acid)

**Tab. 28.7:** Laboratory parameters (some of them with detectability time) giving evidence of long-term alcohol consumption

## 29 Drug-induced liver damage

**Fig. 29.1:** Diagram illustrating potential pathogenetic mechanisms related to drug-induced toxic liver damage

**Fig. 29.2:** Necroinflammatory (“toxic”) hepatitis after halothane intoxication (HE) (s. tab. 29.11)

**Fig. 29.3:** Mixed (intracellular and canalicular) cholestasis following the use of dextropropoxyphene hydrochloride (HE) (s. tab. 29.11)

**Fig. 29.4:** Pronounced bilirubinostasis and cholate stasis as well as Kupffer cell cholestasis (see arrow) following administration of thiamazole (HE) (s. tab. 29.11)

**Fig. 29.5:** Slightly florid, destructive cholangitis (see arrow) resulting from an ACE inhibitor (HE) (s. tab. 29.11)

**Fig. 29.6:** Epithelioid cell granuloma resulting from sulphonyl urea therapy (HE) (s. tab. 29.11)

**Fig. 29.7:** Collidon storage: resorptive reaction of Kupffer cells and multinuclear macrophages to collidon deposits ten months after administration of plasma expander volume. *Insert:* Collidon granuloma (HE)

**Fig. 29.8:** Hepatic impairment with monocellular infiltration following administration of carbamazepine (HE) (s. tab. 29.11)

**Fig. 29.9:** Budd-Chiari syndrome with obliterated radicular vein (see arrow) after administration of contraceptives (van Gieson) (s. tab. 29.11)

**Fig. 29.10:** Intima proliferation and thrombosis of a (radicular) hepatic vein resulting from Senecio alkaloids (HE) (s. tab. 29.11)

**Fig. 29.11:** Adenoma (approximately 5 cm in diameter) following long-term use of oral contraceptives (s. tab. 29.11)

**Fig. 29.12:** Focal nodular hyperplasia in the left liver lobe following seven years' use of oestrogen (same patient as in fig. 29.13)

**Fig. 29.13:** Focal nodular hyperplasia with parenchymal nodules between fibrotic areas resembling portal zones (Goldner) (same patient as in fig. 29.12)

**Fig. 29.14:** Liver cell adenoma after 21 years' use of oestrogens, with subcapsular focal bleedings and malignant degeneration (hepatocellular carcinoma) (s. tab. 29.10)

**Fig. 29.15:** Chronic toxic hepatitis after ten months chaparral (“creosote bush”) automedication. • Laparoscopy: marked acinar structure, irregular chagreen-like surface (splintered light reflex) and extremely fine fibrosis. Histology: single cell necrosis, slight inflammatory infiltrations and moderate steatosis

**Tab. 29.1:** Pathogenetic criteria of hepatotoxic xenobiotics and the basic types of liver damage induced by foreign substances

**Tab. 29.2:** Xenobiotic-induced acute and chronic types of morphological liver damage (with fundamental and individual reservations) (s. tab. 29.11)

**Tab. 29.3:** Some medicaments that may provoke ductopenia (= vanishing-bile duct syndrome) in isolated cases. (s. tab. 29.11)

**Tab. 29.4:** Some medicaments that may provoke idiosyncratic fulminant or protracted liver failure in isolated cases (with some references) (s. tab. 29.11)

**Tab. 29.5:** Diagnostic measures in suspected fulminant liver failure

**Tab. 29.6:** Morphological reactions or laboratory findings resulting from hepatic damage caused by drug-induced toxicity (s. tab. 29.11)

**Tab. 29.7:** Check-list for the detection of possible hepatotoxic xenobiotics

**Tab. 29.8:** Factors causing reduced drug metabolism in increasing age (>55 years)

**Tab. 29.9:** Certain errors or risks involved in the preparation of herbal remedies

**Tab. 29.10:** Medicinal plants with reported or suspected facultative hepatotoxicity (ALF = acute liver failure, R = rare) (with some references)

**Table 29.11:** Table of 314 (selected) medicaments potentially causing liver damage. (6, 84, 89, 108, 141) • → Drugs which may cause acute liver failure or lethal liver damage. (We would be grateful for any suggestions, corrections or additions)

## 30 Liver damage due to toxic substances

**Fig. 30.1:** Haemangiosarcoma in a case of disease induced by vinyl chloride. Tumorous endothelial proliferation with blood cavities and vascular fissures containing erythrocytes (HE)

**Fig. 30.2:** Thorotrast liver: dark brown colouring of the liver surface with reticular bright white fibrosis

**Fig. 30.3:** Thorotrastosis: deposits of thorotrast in portal and perisinusoidal macrophages; periportal fibrosis and inflammation (HE)

**Tab. 30.1:** Factors influencing the extent and type of liver damage caused by toxic substances

**Tab. 30.2:** Pathogenic and pathophysiological mechanisms of liver damage caused by toxic substances

**Tab. 30.3:** Table of important, mainly industrially used, toxic substances (selection). (+ = causes very severe liver damage, severe toxic hepatitis and acute liver failure) • We would appreciate any corrections, supplements or additions

## 31 Metabolic disorders and storage diseases

**Fig. 31.1:** Glycogenated nuclei (so-called glycogen vacuolations of the nuclei) in diabetes mellitus. *Insert:* nucleus strongly laden with glycogen (↑) (PAS) (s. pp 402, 540, 605, 613, 630, 639)

**Fig. 31.2:** Large-droplet (coarse-vacuolar) fatty liver in diabetes mellitus (preliminary stage of fat cyst formation) (HE)

**Fig. 31.3:** Pronounced mixed-droplet fatty liver (Sudan red)

**Fig. 31.4:** Steatosis in periportal liver parenchyma. Clinical diagnosis: chronic phosphorus poisoning (HE)

**Fig. 31.5:** Centroacinar steatosis. Clinical diagnosis: chronic alcohol abuse (Sudan red)

**Fig. 31.6:** Fatty liver with pronounced, thumb-sized fatty changes at the periphery of the left lobe (so-called “yellow spot”)

**Fig. 31.7:** Non-alcoholic steatohepatitis: Hydropic degenerated hepatocytes with Mallory's hyaline (←); lymphocytic and granulocytic infiltration as well as activated Kupffer cells (HE)

**Fig. 31.8:** Non-alcoholic steatohepatitis with massive steatosis and fibrosis. A 10-year-old boy with diabetes mellitus type 2

**Fig. 31.9:** Liver cirrhosis in non-alcoholic steatohepatitis. A 24-year-old adipose woman with insufficiently treated diabetes mellitus type 2 (Sirius red)

**Fig. 31.10:** Reye's syndrome (newborn): fine-droplet fatty changes of hepatocytes as well as glycogen depletion; no signs of inflammation (PAS)

**Fig. 31.11:** Microvesicular fatty changes of liver cells in acute fatty liver of pregnancy (Sudan red)

**Fig. 31.12:** Serologically confirmed  $\alpha_1$ -antitrypsin deficiency type PiZ in a juvenile patient (homozygosity). Globular  $\alpha_1$ -antitrypsin deposits in the hepatocytes; PiZ immunohistochemistry with PiZ antibody ATZ11

**Fig. 31.13:** Liver amyloidosis: Perisinusoidal amyloidosis with atrophic hepatocyte trabeculae (HE)

**Fig. 31.14:** Gierke's disease: phytocyte-like hepatocytes surrounded by a delicate network of fibrosis (Ladewig)

**Fig. 31.15:** Cholesterol ester storage disease. Fine-droplet fatty changes in the hepatocytes. Widely extensive small and larger lipid vacuoles in the liver cells and foam cells of the portal field (Sudan black) (s. fig. 21.5). Same patient as in fig. 31.16

**Fig. 31.16:** Light yellowish-red, smooth surface in fatty liver due to cholesterol ester storage (13-year-old girl). Same patient as in fig. 31.15

**Fig. 31.17:** Gaucher's disease. Gaucher cells (sphingolipid-storing macrophages) within the liver parenchyma (arrow), shown here as pale-blue cells with an internal structure similar to cigarette paper (PAS)

**Fig. 31.18:** Skin changes (face, front part of the neck) in porphyria cutanea tarda (s. fig. 4.13)



**Fig. 31.19:** Chronic active hepatitis in PCT: dispersed light reflection, capsular fibrosis with pronounced net-like fibrosis. Spider-like subcapsular neovascularization (s. fig. 33.13)

**Fig. 31.20:** Chronic porphyria: uncharacteristic lobular hepatitis histologically correlating to increased liver enzyme levels (HE)

**Fig. 31.21:** Chronic hepatic porphyria: Sonographically, multiple, ring-shaped foci with marginal hyperechoic ring and central hypoechoic reflexes. (Completely reversible after alcohol abstinence)

**Fig. 31.22:** Chronic hepatitis in Wilson's disease. Pronounced "simian cleft" with barely recognizable hepar succenturatum (s. p. 19)

**Fig. 31.23:** Implanted liver with micronodular (regenerative-weak) cirrhosis in Wilson's disease (18-year-old woman presenting with acute liver failure) (Sirius red)

**Fig. 31.24:** Cirrhosis in Wilson's disease. Numerous copper deposits in periportal liver epithelia (Rhodanine)

**Fig. 31.25:** Active progredient liver cirrhosis in Wilson's disease

**Fig. 31.26:** Postulated pathogenetic and morphological cascade of damage in HC

**Fig. 31.27:** Haemochromatotic cirrhosis: micronodular, slate-grey to brownish discolouration, rich in septate fibrosis, marked neovascularization

**Fig. 31.28:** Micronodular cirrhosis due to hereditary (HFE-related) haemochromatosis (Berlin blue)

**Fig. 31.29:** Cirrhosis in hereditary haemochromatosis. Massive siderosis in hepatocytes and in epithelia of preformed bile ducts (arrows) (Berlin blue)

**Fig. 31.30:** Siderosis of Kupffer cells in haemolytic anaemia (Berlin blue)

**Tab. 31.1:** Pathogenesis of liver steatosis and fatty liver

**Tab. 31.2:** Formation, localization and various courses of liver steatosis resulting in the development of fatty liver (WW = wet weight of liver)

**Tab. 31.3:** Causes of microvesicular fatty degeneration (with some references)

**Tab. 31.4:** Some drugs causing phospholipidosis

**Tab. 31.5:** Acquired causes of liver steatosis or fatty liver (including some references)

**Tab. 31.6:** Congenital causes of liver steatosis or fatty liver (so-called thesaurismoses)

**Tab. 31.7:** Liver steatosis or fatty liver due to medication, chemical substances or toxins (s. tab. 29.11!)

**Tab. 31.8:** Diagnosis of fatty liver or NASH, involving determination of the degree of severity, differential diagnosis and course

**Tab. 31.9:** Possible complications of fatty liver

**Tab. 31.10:** Primary (erythropoietic, hepatic, hepatoerythropoietic) porphyrias

**Tab. 31.11:** Secondary (symptomatic, acquired) disorders of porphyrin metabolism

**Tab. 31.12:** Constellation of findings in erythropoietic porphyrias (v = variable, N = normal)

**Tab. 31.13:** Drugs which are able to trigger acute hepatic porphyria (AIP, VP, HCP) with differing degrees of risk (s. tab. 31.10)

**Tab. 31.14:** Porphyrin and porphyrin precursor content in urine and faeces for the differentiation of hepatic porphyrias (v = variable, N = normal) (s. tab. 31.10)

**Tab. 31.15:** Decisive laboratory criteria for the diagnosis and follow-up of Wilson's disease

**Tab. 31.16:** Diagnostic measures for detecting the type and extent of organ involvement in Wilson's disease

**Tab. 31.17:** Classification of haemochromatosis and haemosiderosis (WW = wet weight)

**Tab. 31.18:** Biochemical findings in hereditary haemochromatosis

## 32 Autoimmune hepatitis

**Fig. 32.1:** Acute AIH type 1 (ANCA +) with pronounced centrilobular parenchymal loss. Rapid therapy success (clinically, biochemically) with immunosuppressives (HE)

**Fig. 32.2:** Lymphoplasmocytic interface hepatitis in autoimmune hepatitis type 1 (HE)

**Fig. 32.3:** AIH type 1 (so-called *lupoid hepatitis*). The left lobe of liver shows an irregularly rippled surface (scattered light reflex) with salmon-pink and yellow colouring; patchy red marking due to highly inflammatory parenchymal zones; fine vascular multiplication and whitish scarred areas with diffuse fibrosis

**Fig. 32.4:** Autoimmune hepatitis in remission under treatment: septal fibrosis and disarranged lobular structure (HE)

**Fig. 32.5:** Liver cirrhosis due to AIH: flat-nodular liver surface, highly cicatrized furrows, local neovascularization, signs of mild inflammation

**Fig. 32.6:** Active autoimmune liver cirrhosis type 1 with enlarged periportal inflammatory lymph nodes (arrows) (VP = portal vein)

**Tab. 32.1:** Differentiation of autoimmune hepatitis (s. tab. 5.20). • (AHA = anti-histone antibody; anti-GOR = anti-specific nuclear antigen in HCV) (see text for other abbreviations). –/(+) = no or occasional slight increase

**Tab. 32.2:** Main endocrine and non-endocrine disorders in APS type 1

**Tab. 32.3:** Diseases or immunopathies which may be associated with autoimmune hepatitis (s. tabs. 22.2, 22.7, 22.8; 33.3, 33.6)

## 33 Cholangitis and cholangiodysplasia

**Fig. 33.1:** Cholesterol-calcium-pigment stone: a rare specimen showing a striking coloured/chemical development (here: a so-called *tiger-eye stone*) (*at our disposal*). Diagnosis: obstructive cholangitis due to *Mirizzi's syndrome*

**Fig. 33.2:** Ascending, suppurative, destructive, relapsing cholangitis with abscess formation; the loose periportal fibre cuff (arrow) points to previous cholangitic episodes (HE)

**Fig. 33.3:** Unusual form of slight fibrosis with septated pattern. Clinically: history of previous bile-duct inflammation (Sirius red)

**Fig. 33.4:** Chronic cholangitis. Periportal fibrosis; dark red/brownish discolouration of the liver with greenish patches: finely nodular surface (= scattered light reflection)

**Fig. 33.5:** Biliary cirrhosis following chronic relapsing and abscess-forming cholangitis; green and grey "dirty" colouration of the deformed micronodular surface. (Chronic cholecystitis with formation of a shrunken gall bladder)

**Fig. 33.6:** Aerobilia: Sonography shows echogenic, strand-like gas bubbles in the respective bile ducts (↓)

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