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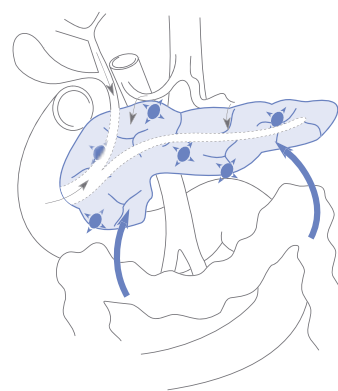


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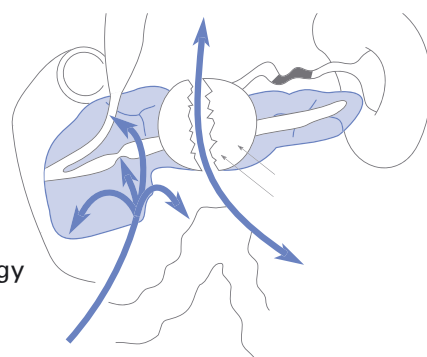
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- Pathogenesis and Pathophysiology
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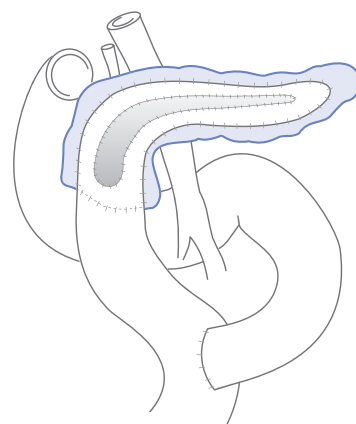


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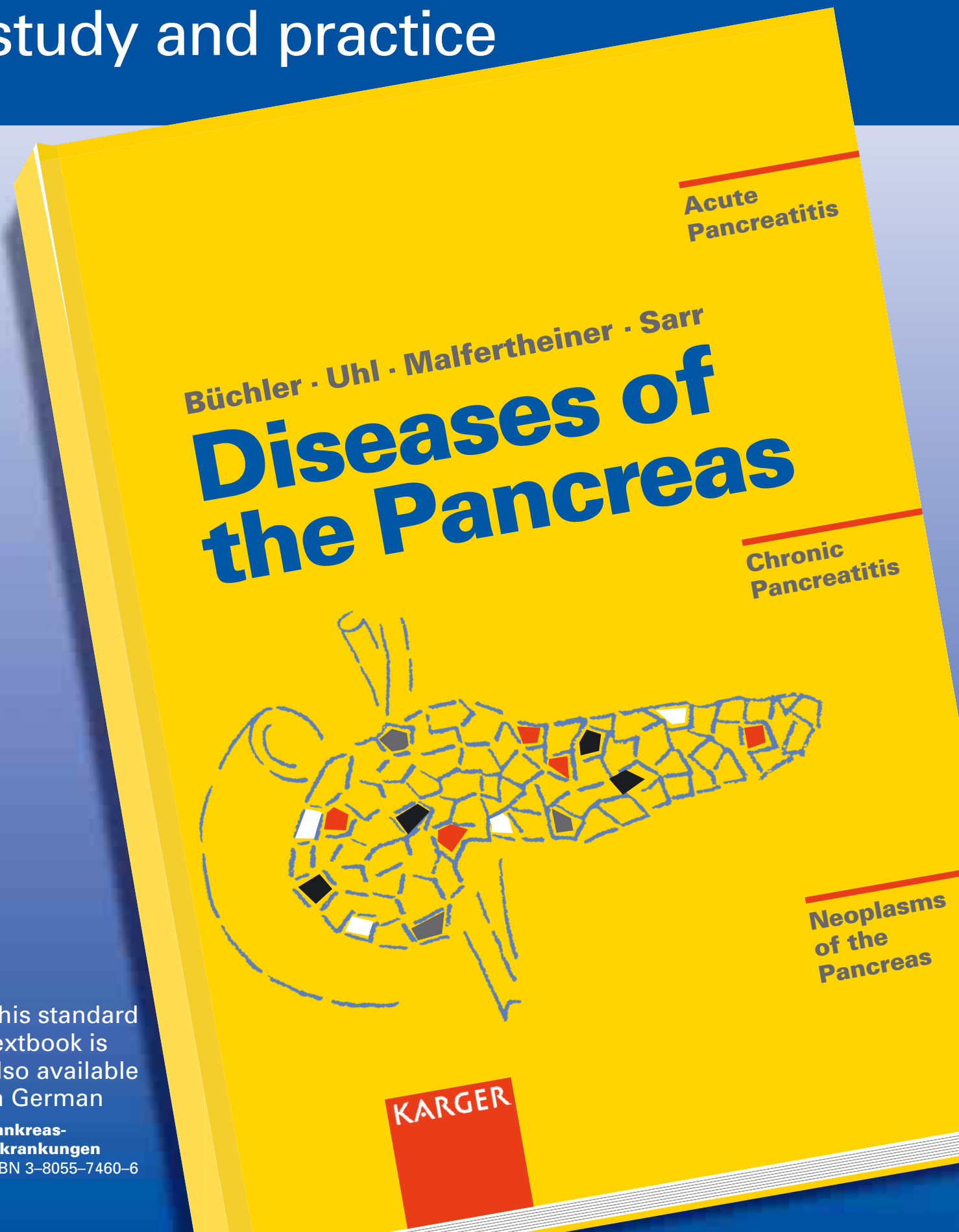
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Diseases of the Pancreas

Sample pages from part 1
Acute Pancreatitis

Fields of Interest:
Gastroenterology,
Surgery, Oncology,
General Medicine,
Internal Medicine,
Endocrinology,
Pediatrics, Pathology,
Pharmacology,
Radiology

Clearly structured text, presented with the corresponding illustrations on a two-page spread

Compiled by a team of authors with many years' experience in the field of diseases of the pancreas, this book is an ideal combination of evidence-based literature and the authors' expertise. An interdisciplinary approach to the basis, diagnosis and treatment of diseases of the pancreas has been chosen, and the succinct and clearly structured text is consistently linked with the corresponding illustrations on a two-page spread. The division into three major parts (acute pancreatitis, chronic pancreatitis and pancreatic tumors) further enhances the clarity of the text.

In the chapter 'Acute pancreatitis', along with the new markers of necrosis, several major topics of imaging via nuclear magnetic resonance and the different randomized controlled studies addressing treatment are covered. In the section on chronic pancreatitis, clinical aspects, pancreatic function and imaging, and recent clinical studies receive special attention. Finally, in the section on tumors of the pancreas, particular emphasis has been placed on

Acute Pancreatitis

Pathogenesis and Pathophysiology

Depending on the causal factor, the pathogenesis of acute pancreatitis develops most probably from direct acinar cell damage, as one would expect, especially with metabolic causes, or possibly via intraductal activation of enzymes and the passage of the latter into the interstitial region of the pancreatic tissue. Through either of these mechanisms, the pathologic process results in morphologic damage. With certain etiologic factors, there may be a combination of impaired intraductal permeability and direct damage to the acinar cells, each potentiating the other. Our knowledge to date of the pathogenesis of acute pancreatitis is almost wholly derived from animal experiments. Acute pancreatitis has been triggered in various diverse models (rat, mouse, opossum, cat, dog) by increasing the pressure in the pancreatic duct in combination with activated pancreatic enzymes or intraductal toxins (bile), with impairment of ductal permeability, via direct acinar cell damage by cellular toxins, or interestingly overstimulation with pancreatic secretagogues (CCK, cerulein).

The mechanism of direct cell damage proceeds via disruption of the normal cell compartmentalization with disruption of intracellular trafficking of zymogen granules. Whereas under physiologic conditions, precursors of the pancreatic enzymes (the inactive proenzymes) are protected against early activation and resultant autodigestion by intracellular protective mechanisms (compartments), this intrinsic intracellular mechanism of protection is disrupted by the specific triggering toxins or secretagogues. This pathologic process results in fusion of the zymogen granules containing the inactive enzyme precursors with the lysosomes (so-called 'colocalization')

Pathogenesis and Pathophysiology

Pathophysiology (Acinar Cells)

1 = zymogen granules
2 = hydrolases

Intracellular injury resulting from enzyme activation (crinophagy)

Triggering factor

- Obstruction of intracellular trafficking of zymogens
- Systemic toxic injury of the acinar cells
- Changes in cell and tissue compartmentalization (colocalization)
- Intracellular activation of intrapancreatic enzyme
- Inhibition of zymogen release from acinar cell

Defense mechanism

- Synthesis of enzymes as inactive zymogens (pro-enzymes)
- Storage of zymogen granules
- Inhibitors of protease activity (acinar cells)
- Serum antiproteases
- Unimpeded drainage of secretions and lymph
- Blood perfusion of organs

Sample pages from part 2
Chronic Pancreatitis

Chronic Pancreatitis

Complications

Pancreatic pseudocysts, stenosis of the common bile duct, and stenosis of adjacent structures (duodenum, portal or splenic vein, colon) also occur in chronic pancreatitis. If pseudocysts of 2 cm in size are included, these are found on ultrasonography or CT in almost half of all patients with chronic pancreatitis. Unlike the acute peripancreatic fluid collections found in patients with acute pancreatitis that often resolve, pseudocysts occurring in the setting of chronic pancreatitis rarely regress. On the other hand, these cysts initially do not represent a danger to the patient unless they reach sizes >6 cm that may lead to complications, such as compression of the duodenum or the bile duct. Chronic pancreatitis of many years' standing as well as the familial form involve a substantially higher risk of the development of pancreatic carcinoma. The incidence of adenocarcinoma in patients with chronic pancreatitis over a 10-year period of follow-up is approximately 5%.

Stenosis of the common bile duct is an important finding, the incidence of which varies from 10 to 40% depending on the definition used. The extent of stenosis differs widely; it is caused by fibrotic stenosing reaction of the pancreatic tissue surrounding the intrapancreatic portion of the common bile duct, by external compression due to an inflammatory tumor in the head of the pancreas, or by a pseudocyst. This stenosis may be evident only by a rise in alkaline phosphatase or bilirubin, but occasionally it appears as clinical jaundice. Stenosis of the intestinal tract occurs predominantly in the duodenal region; stenosis of the colon occurs in extremely rare cases. Extra-

Complications

Possible Complications

- Hemosuccus pancreaticus
- Hemobilia
- Pleural effusion
- Splenomegaly
- Rupture of the spleen
- Hemorrhage
- Splenic vein thrombosis with portal hypertension (varices)
- Infection of a pseudocyst
- Bleeding into a pseudocyst
- Retroperitoneal spread
- Ascites
- Hemoperitoneum

Stenosis

- Common bile duct → Obstructive jaundice, cholangitis
- Pancreatic duct → obstructive pancreatitis
- Duodenum → vomiting
- Colon → colonic obstruction (partial)
- Portal system → portal hypertension

As a result of pseudocyst or compression/spread of chronic inflammation

Duodenal or Common Bile Duct Stenosis

Passage of contrast medium with duodenal stenosis in a patient with a chronic inflammatory tumor of the head of the pancreas.

ERC showing common bile duct compression complicating a chronic inflammatory tumor of the head of the pancreas.

Sample pages from part 3
Neoplasms of the Pancreas

Neoplasms of the Pancreas

Endocrine Pancreatic Neoplasms

Endocrine pancreatic neoplasms are classified as benign or malignant neoplasms of the neuroendocrine system of the pancreas, i.e. so-called neuroendocrine or islet cell neoplasms. For the classification of these neuroendocrine tumors (NET), the functional classification has proved most useful according to the hormones that are clinically active in the neoplasm.

The two most frequent endocrine neoplasms of the pancreas are insulinoma (approximately 75% of patients) and gastrinoma (approximately 20% of patients). All other endocrine pancreatic neoplasms are extremely rare and are found in the literature, usually, as case reports.

Insulinoma

Approximately 75% of all endocrine pancreatic neoplasms present clinically as insulin-producing islet cell neoplasms. Over 90% of insulinomas are benign. A third of insulin-producing neoplasms arise in the body of the pancreas, a third in the head of the pancreas, and a third in the tail of the pancreas. Approximately 80% of insulinomas occur singly, with a mean tumor size of 1–3 cm.

Clinical Picture and Diagnosis

The symptoms of an insulinoma are generally characterized by neurologic signs caused by the hyperinsulinemic hypoglycemia. Very frequently, patients present with loss of consciousness, which may masquerade as a primary neurologic disease. It is not unusual for an insulinoma to be mistaken for epilepsy.

The diagnosis of insulinoma is made through a 72-hour fasting test. The patient has to fast for up to 72 h, with simultaneous and continuous

Classification

Type	Incidence %	Mutation rate, %	Hormone	Extrapancreatic location, %
Insulinoma	75	<10	insulin	1
Gastrinoma	15–20	>50	gastrin	20–40
Vipoma	1–2	>50	vasoactive intestinal polypeptide	5–20
Glucagonoma	1–2	>70	glucagon	rare
Somatostatinoma	<1	>50	somatostatin	frequent
PPoma	<1	?	pancreatic polypeptide	?
Carcinoid	?	?	serotonin	?
Corticotropinoma	<1	>99	melanocyte-stimulating corticotropin	?
Hyperparathyroidism	<1	>99	?	?
Neurotensinoma	?	?	neurotensin	?
Calcitoninoma	?	?	calcitonin	?
Nonfunctional neoplasms	<5	>50	–	?

Clinical Features

Insulinoma	Frequency, %
Neurologic symptoms (dizziness, absence attacks, apathy, coma)	92
Cardiovascular symptoms (episodic palpitations, precordial pain)	17
Gastrointestinal symptoms (hunger attacks, nausea, vomiting)	9

Site of Operation and Macroscopic Appearance

Intraoperative site in the case of an insulinoma in the tail of the pancreas (arrow).

Macroscopic appearance of an insulinoma.