ACUTE PANCREATITIS
&
PANCREATIC NEOPLASMS

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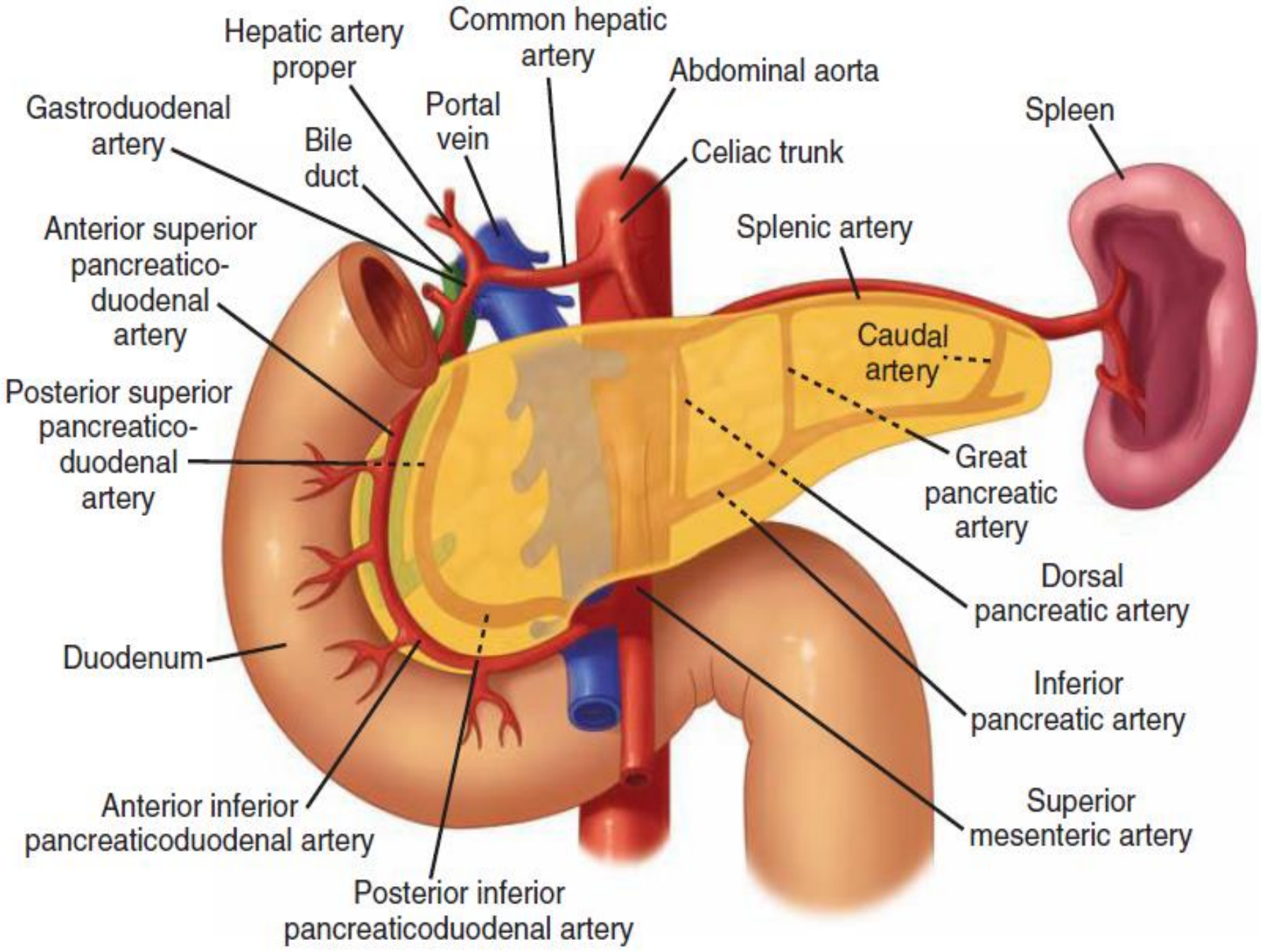
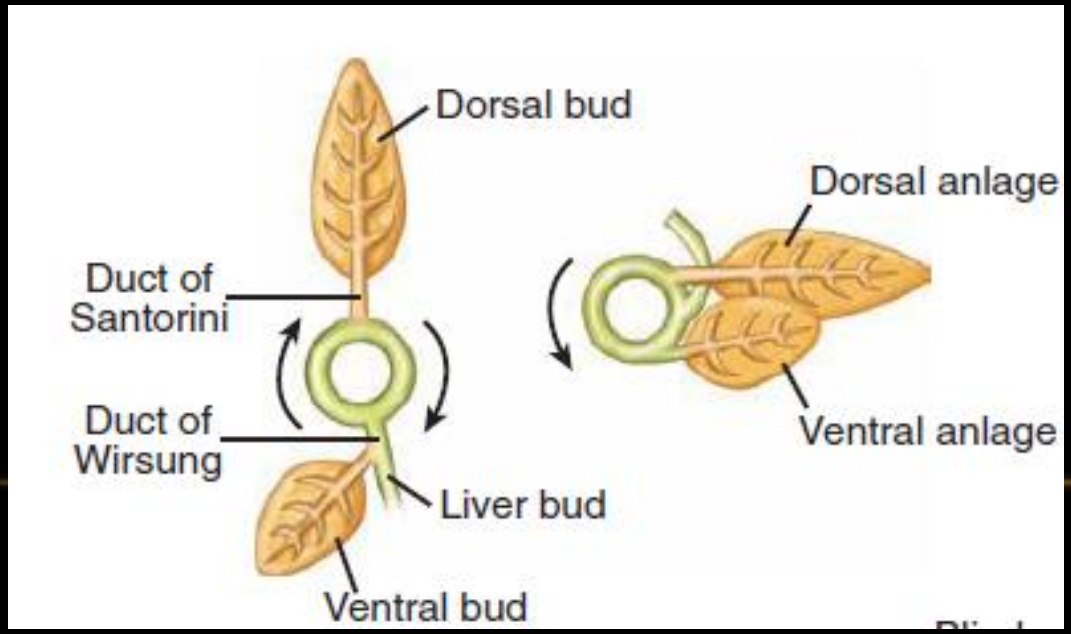
ANATOMY

- The pancreas is perhaps the most unforgiving organ in the human body, leading most surgeons to avoid even palpating it unless necessary.
- The pancreas is a retroperitoneal organ that lies in an oblique position.
- In an adult, the pancreas weighs 75 to 100 g and is about 15 to 20 cm long.
- The neck of the pancreas is anterior to the vertebral body of L1 and L2.


Hepatic artery proper
Common hepatic artery
Abdominal aorta
Gastroduodenal artery
Portal vein
Bile duct
Celiac trunk
Spleen
Anterior superior pancreatico-duodenal artery
Splenic artery
Caudal artery
Posterior superior pancreatico-duodenal artery
Great pancreatic artery
Dorsal pancreatic artery
Duodenum
Inferior pancreatic artery
Superior mesenteric artery
Anterior inferior pancreaticoduodenal artery
Posterior inferior pancreaticoduodenal artery

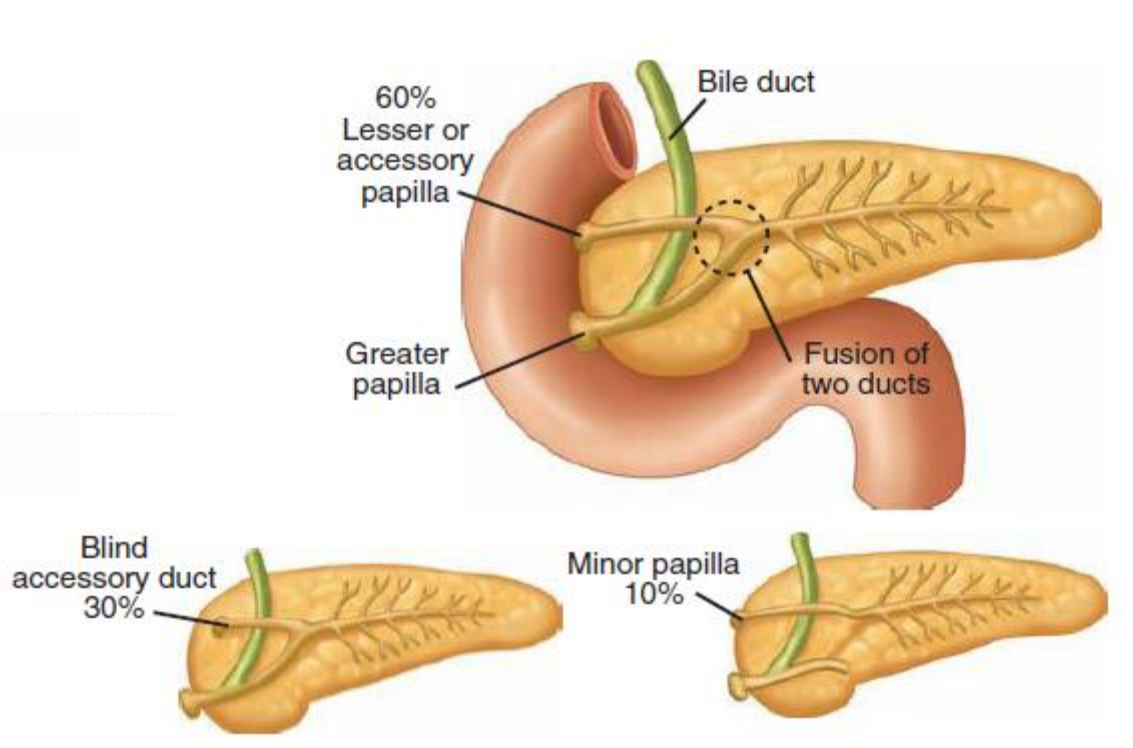
ANATOMY : DUCT

- The pancreas is formed by the fusion of a **ventral** (arises from the hepatic diverticulum) and **dorsal** (arises from the duodenum) bud.



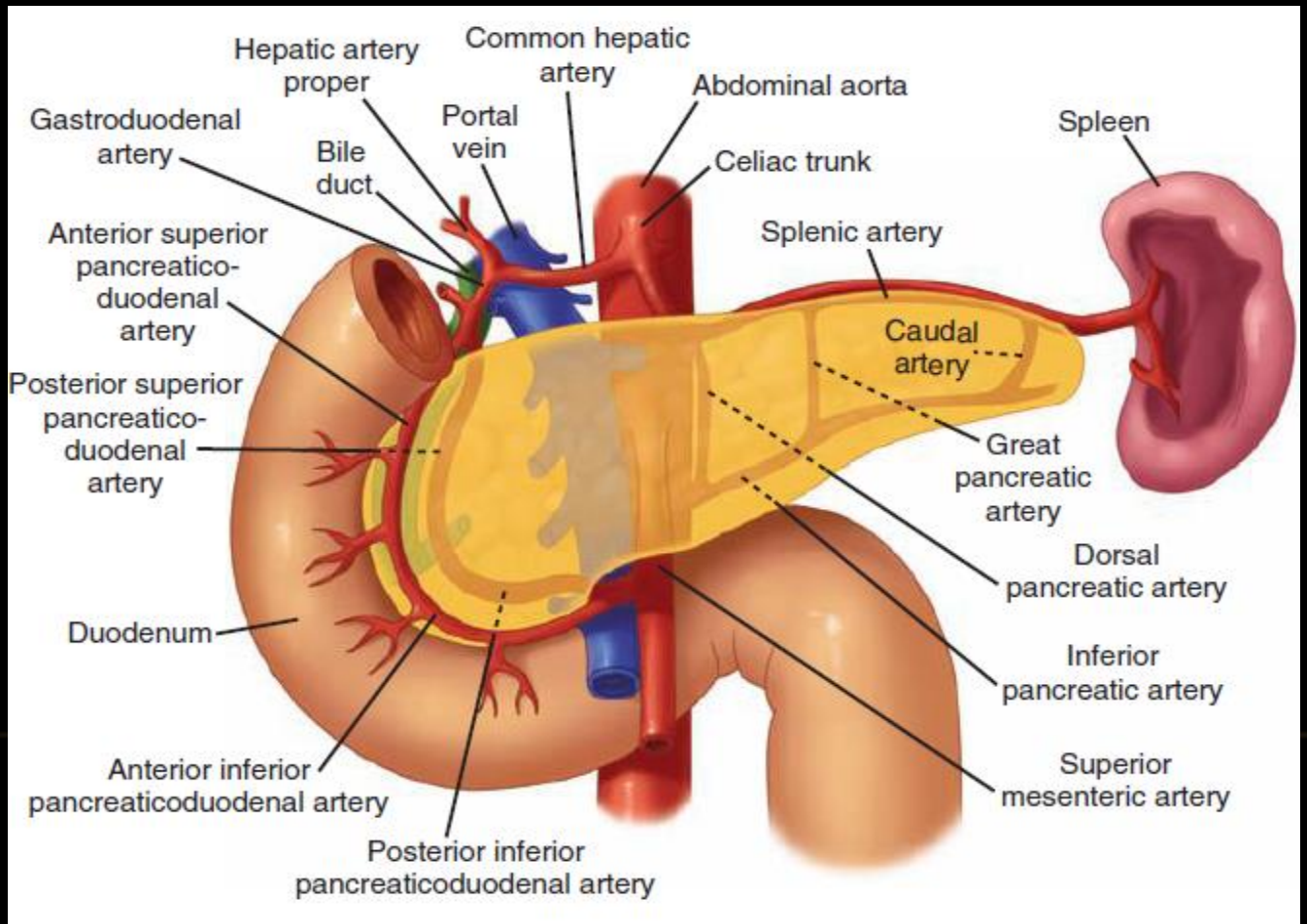
ANATOMY : DUCT

- The duct from the smaller ventral bud, connects directly to the common bile duct (**Wirsung**).
- The duct from the larger dorsal bud, drains directly into the duodenum (**Santorini**).



VASCULAR ANATOMY

- The blood supply to the pancreas comes from multiple branches from the celiac and superior mesenteric arteries .



ANATOMY

- The venous drainage of the pancreas follows a pattern similar to that of the arterial supply.
- The lymphatic drainage from the pancreas is diffuse and widespread .
- The parasympathetic system stimulates endocrine and exocrine secretion and the sympathetic system inhibits secretion.
- The pancreas also has a rich supply of afferent sensory fibers, which are responsible for the intense pain associated with advanced pancreatic cancer, as well as acute and chronic pancreatitis. These somatic fibers travel superiorly to the celiac ganglia .

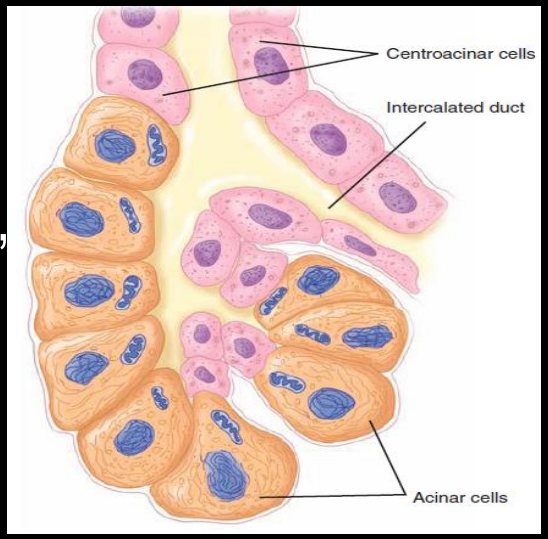
PHYSIOLOGY

- The exocrine pancreas : 85% of the pancreatic mass
- Extracellular matrix : 9-10%
- Blood vessels and the major ducts : 3-4%
- Endocrine tissue: 2% .

- Although patients can live without a pancreas when insulin and digestive enzyme replacement are administered, the loss of this ***islet-acinar coordination*** leads to impairments in digestive function.

PHYSIOLOGY

- **Exocrine Pancreas** : The pancreas secretes approximately 500 to 800 mL per day of colorless, odorless, alkaline, isosmotic pancreatic juice.



- The acinar cells secrete **amylase**, **proteases**, and **lipases**, enzymes responsible for the digestion of all three food types: carbohydrate, protein, and fat.
- Amylase is the only pancreatic enzyme secreted in its active form

PHYSIOLOGY

- The proteolytic enzymes are secreted as proenzymes that require activation. Trypsinogen, Chymotrypsinogen.(enterokinase activates trypsinogen)
- Elastase, carboxypeptidase A and B, and phospholipase are also activated by trypsin
- Pancreatic lipase is secreted in an active form. Colipase is also secreted by the pancreas, changing its molecular configuration and increasing its activity.
- The centroacinar and intercalated duct cells secrete the water and electrolytes present in the pancreatic juice.

PHYSIOLOGY : ENDOCRINE

- There are nearly 1 million islets of Langerhans in the normal adult pancreas.

- Most islets contain 3000 to 4000 cells of five major types:

- **alpha** : glucagon
- **β-cells** : insulin
- **delta cells** : somatostatin
- **epsilon cells** : ghrelin
- **PP cells** : PP

Pancreatic islet peptide products

- Insulin β (beta cell) Decreased gluconeogenesis, glycogenolysis, fatty acid breakdown,and ketogenesis/Increased glycogenesis, protein synthesis, and glucose uptake
- Glucagon α (alpha cell) Opposite effects of insulin; increased hepatic glycogenolysis and gluconeogenesis
- Somatostatin δ (delta cell) Inhibits GI secretion/Inhibits secretion and action of all GI endocrine peptide/Inhibits cell growth
- Pancreatic polypeptide PP (PP cell) Inhibits pancreatic exocrine secretion and section of insulin/Facilitates hepatic effect of insulin
- Ghrelin (epsilon cell) Decreases insulin release and insulin action

ACUTE PANCREATITIS

DEFINITION, INCIDENCE, AND EPIDEMIOLOGY

- It ranges from a mild self-limiting inflammation of the pancreas to critical disease characterized by infected pancreatic necrosis, multiple organ failure and a high risk of mortality.

- Worldwide the incidence of acute pancreatitis ranges from 5 to 80/100,000 population with the highest incidence recorded in Finland and United States.

ETIOLOGY

- The most common causes are **gallstones** and **alcohol**, accounting for up to 80% of cases but it is not uncommon to diagnose acute pancreatitis in the absence of these etiological factors.

Etiologies of acute pancreatitis
Alcohol
Biliary tract disease
Hyperlipidemia
Hereditary
Hypercalcemia
Trauma
External
Surgical
Endoscopic retrograde cholangiopancreatography
Ischemia
Hypoperfusion
Atheroembolic
Vasculitis
Pancreatic duct obstruction
Neoplasms
Pancreas divisum
Ampullary and duodenal lesions
Infections
Venom
Drugs
Idiopathic

ETIOLOGY

- ***Gallstones***: "**common channel" hypothesis**, transient incompetence caused by the passage of a stone through the sphincter might allow duodenal fluid and bile to reflux into the pancreatic duct, ductal hypertension.

- ***Alcohol***: Ethanol is a metabolic toxin to pancreatic acinar cells.

- ***Drugs***: thiazide diuretics, furosemide, estrogens, azathioprine, methyldopa, the sulfonamides, tetracycline, procainamide, nitrofurantoin, valproic acid.

PATHOPHYSIOLOGY

- pancreatitis begins with the activation of digestive zymogens inside acinar cells, which cause acinar cell injury.

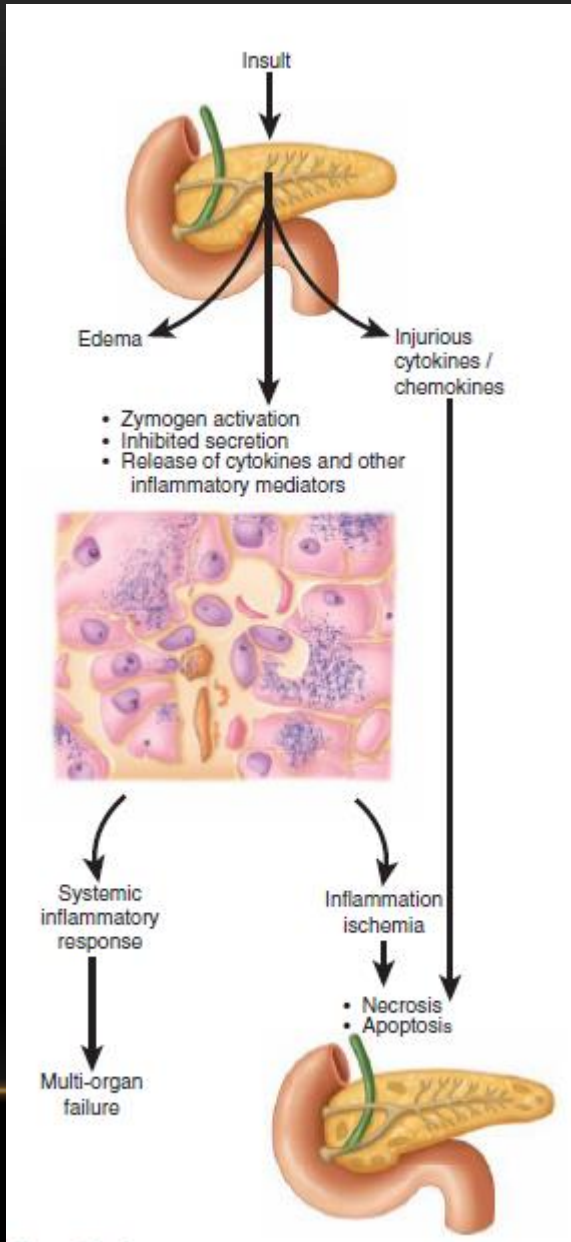


Figure 33-10. Pathophysiology of acute pancreatitis.

MANAGEMENT OF THE PATIENT

- Patients with suspected acute pancreatitis should be admitted to hospital.

- ***The risk of mortality*** : 1% for those with mild disease, 10% for those with moderate disease, but for severe and critical disease the mortality risk is much higher (20% to 40% and greater than 50%, respectively).

DIAGNOSIS

- ***Acute onset of a severe constant epigastric pain which often radiates through to the mid back and the elevation of serum amylase or lipase (>3 times upper limit of normal)***

- *Imaging (usually by contrast enhanced CT scanning) is only required for the diagnosis of acute pancreatitis when these diagnostic criteria are not met.*

- The serum amylase concentration increases almost immediately with the onset of disease and peaks within several hours. It remains elevated for 3 to 5 days before returning to normal. There is no significant correlation between the magnitude of serum amylase elevation and severity of pancreatitis; in fact, a milder form of acute pancreatitis is often associated with higher levels of serum amylase compared with that in a more severe form of the disease.

DIAGNOSIS

- **Hyperamylasemia** can occur in : small bowel obstruction, perforated duodenal ulcer, or other intra-abdominal inflammatory conditions.

- In contrast, a patient with acute pancreatitis may have a normal serum amylase (hyperlipidemia) .
- In many cases, urinary clearance of pancreatic enzymes from the circulation increases during pancreatitis; therefore, ***urinary levels may be more sensitive than serum levels***.

DIAGNOSIS

- In some patients (about 1%), the blood from necrotizing pancreatitis may dissect through the soft tissues and manifest itself as a bluish discoloration around the umbilicus (Cullen's sign) or in the flanks(Grey Turner's sign).

- The severe fluid loss may lead to prerenal azotemia with elevated blood urea nitrogen and creatinine levels. There also may be hyperglycemia, hypoalbuminemia, and hypocalcemia sufficient in some cases to produce tetany.

PREDICTING SEVERITY

- Accurately predicting acute pancreatitis severity : making triage decisions, and in making decisions about fluid therapy, whether an ERCP is indicated, and other issues.

- At 24 hours after admission an APACHE II score of 8 or more or a serum C-reactive protein level of >150mg/dl has a similar accuracy in predicting severity as Ranson's criteria.

PREDICTING SEVERITY

- Scoring systems such as Ranson's Criteria identify high-risk patients .

Ranson's prognostic signs of pancreatitis	
Criteria for acute pancreatitis not due to gallstones	
At admission	During the initial 48 h
Age >55 y	Hematocrit fall >10 points
WBC >16,000/mm^3	BUN elevation >5 mg/dL
Blood glucose >200 mg/dL	Serum calcium <8 mg/dL
Serum LDH >350 IU/L	Arterial PO_2 <60 mm Hg
Serum AST >250 U/dL	Base deficit >4 mEq/L
	Estimated fluid sequestration >6 L
Criteria for acute gallstone pancreatitis	
At admission	During the initial 48 h
Age >70 y	Hematocrit fall >10 points
WBC >18,000/mm^3	BUN elevation >2 mg/dL
Blood glucose >220 mg/dL	Serum calcium <8 mg/dL
Serum LDH >400 IU/L	Base deficit >5 mEq/L
Serum AST >250 U/dL	Estimated fluid sequestration >4 L

AST = aspartate transaminase; BUN = blood urea nitrogen; LDH = lactate dehydrogen-ase; PO_2 = partial pressure of oxygen; WBC = white blood cell count.

Note: Fewer than 3 positive criteria predict mild, uncomplicated disease whereas more than 6 positive criteria predict severe disease with a mortality risk of 50%.

PREDICTING SEVERITY

Bedside **I**ndex for **S**everity of **A**cute **P**ancreatitis (**BISAP**) :

BUN (> 25 mg/dl),

Impaired mental status (GCS <15),

Presence of systemic inflammatory response syndrome (SIRS){Two or more of following criteria are met:Temperature ≥38°C (100.4°F) or ≤36°C (96.8°F)Heart rate ≥90 beats per minute/Respiratory rate ≥20 breaths per minute or $Paco_2$ ≤32 mmHg or mechanical ventilation/White blood cell count ≥12,000/⌈L or ≤4000/⌈L or ≥10% band forms}

- Age >60 years, and pleural effusion.

THE SEVERITY OF ACUTE PANCREATITIS

- The key determinants of severity are local complications (absent, sterile or infected) and systemic complications (absent, transient organ failure, or persistent organ failure)

Four categories of acute pancreatitis severity based on organ failure and local complications[61]

DETERMINANTS	NO LOCAL COMPLICATIONS	STERILE LOCAL COMPLICATIONS	INFECTED LOCAL COMPLICATIONS
NO ORGAN FAILURE	MILD	MODERATE	SEVERE
TRANSIENT ORGAN FAILURE	MODERATE	MODERATE	SEVERE
PERSISTENT ORGAN FAILURE	SEVERE	SEVERE	CRITICAL

MANAGEMENT

- **Pain control (** NSAIDS, OPIOIDS**)**
- **Fluid Resuscitation**
- **Nutritional Support**
- **ERCP**
- **Antibiotics**
- **Managing Local Complications**
- **Managing Organ Failure**
- **Cholecystectomy**

FLUID RESUSCITATION

- Fluid therapy to restore and maintain circulating blood volume is the most important intervention in the early management of acute pancreatitis

- It is probably best to resuscitate with a balanced crystalloid and to restore normal blood volume, blood pressure, and urine output. On the basis of recent data it appears that ***lactated Ringer's solution*** may be superior to normal saline in reducing the systemic inflammatory response.

NUTRITIONAL SUPPORT

- It is no longer acceptable to “rest the pancreas” by avoiding enteral nutrition, and parenteral nutrition should only be offered if the patient’s calculated nutritional requirements cannot be achieved by the enteral route.

- **Enteral nutrition should be commenced after initial fluid resuscitation and within the first 24 hours of admission.**

- In predicted mild acute pancreatitis the transition from oral fluids to food is usually timed to when the patient’s abdominal pain has resolved, but the trend is toward allowing patients to resume intake ad libidum (i.e., patient controlled nutrition).

ERCP

- More recent evidence has suggested that early ERCP confers no benefit in the absence of concomitant cholangitis, as the offending common duct stone usually passes before ERCP can be performed.

- If symptoms and chemical abnormalities persist or increase after intial presentation, magnetic resonance cholangio-pancreatography (MRCP) can detect a filling defect in the distal common bile duct, and may be used as prerequisite for attempted ERCP.

ANTIBIOTICS

- Although the use of broad-spectrum antibiotics to treat established infection in acute pancreatitis is a well-established practice, there has been considerable controversy surrounding the use of prophylactic antibiotics.

MANAGING LOCAL COMPLICATIONS

- This means close monitoring of the patient by serial examination, supplemented by regular measurement of inflammatory markers (e.g., C reactive protein) and a pancreatic protocol CT scan if a local complication is suspected and intervention considered warranted.

- Uncommon before 3 to 4 weeks from the onset of symptoms.

- An important emerging approach is the increasing use of percutaneous catheter drainage in patients with suspected infected complications.

MANAGING LOCAL COMPLICATIONS

- The management of an acute noninfected pseudocyst is usually conservative, as about half of these will resolve spontaneously.

- When symptoms of pain or inability to eat persist or infection occurs, intervention is required. Pseudocysts persist because of communication with the main pancreatic duct and/or distal ductal stenosis.
- **Percutaneous drainage should be avoided** in this situation because of the risk of external pancreatic fistula. EUS guided internal drainage into stomach, duodenum, or transpapillary pancreatic duct stenting is the preferred approach.

MANAGING ORGAN FAILURE

- The early identification of organ dysfunction and failure is important because it is a key determinant of severity and outcome and to facilitate the timely transfer of the patient to an intensive care unit to optimize management, provide organ support, and allow more intensive monitoring.

CHOLECYSTECTOMY

- Index cholecystectomy, done in the same admission and prior to discharge, appears safe and can almost always be accomplished laparoscopically.
- But index cholecystectomy is not suitable for all patients, particularly some who have had local pancreatic complications which includes a large phlegmon which extends into the porta hepatis. These patients may require an interval cholecystectomy after resolution of the inflammatory process. If surgery is required for the management of local complications, then a cholecystectomy is often performed at that time.

Complications of acute pancreatitis

I. Local
- A. Pancreatic phlegmon
- B. Pancreatic abscess
- C. Pancreatic pseudocyst
- D. Pancreatic ascites
- E. Involvement of adjacent organs, with hemorrhage, thrombosis, bowel infarction, obstructive jaundice, fistula formation, or mechanical obstruction

II. Systemic
- A. Pulmonary
  - 1. Pneumonia, atelectasis
  - 2. Acute respiratory distress syndrome
  - 3. Pleural effusion
- B. Cardiovascular
  - 1. Hypotension
  - 2. Hypovolemia
  - 3. Sudden death
  - 4. Nonspecific ST-T wave changes
  - 5. Pericardial effusion
- C. Hematologic
  - 1. Hemoconcentration
  - 2. Disseminated intravascular coagulopathy
- D. GI hemorrhage
  - 1. Peptic ulcer
  - 2. Erosive gastritis
  - 3. Portal vein or splenic vein thrombosis with varices
- E. Renal
  - 1. Oliguria
  - 2. Azotemia
  - 3. Renal artery/vein thrombosis
- F. Metabolic
  - 1. Hyperglycemia
  - 2. Hypocalcemia
  - 3. Hypertriglyceridemia
  - 4. Encephalopathy
  - 5. Sudden blindness (Purtscher's retinopathy)
- G. Central nervous system
  - 1. Psychosis
  - 2. Fat emboli
  - 3. Alcohol withdrawal syndrome
- H. Fat necrosis
  - 1. Intra-abdominal saponification
  - 2. Subcutaneous tissue necrosis

Algorithm for the evaluation and management of acute pancreatitis

1. Diagnosis
   - History of abdominal pain consistent with acute pancreatitis
   - >3x elevation of pancreatic enzymes
   - CT scan if required to confirm diagnosis
2. Initial assessment / management (first 4 hrs)
   - Analgesia
   - Fluid resuscitation
   - Predict severity of pancreatitis
     - Ranson's criteria
     - HAPS score
   - Assess systemic response
     - SIRS score
     - SOFA (organ failure)
3. Reassessment / management (4 to 6 hrs)
   - Assess response to fluid resuscitation
     - mean arterial pressure
     - heart rate
     - urine output
     - hematocrit
   - Determine etiology
     - Ultrasound for gallstones/sludge
     - History of alcohol consumption
     - Laboratory evaluation of other causes
   - MRCP and/or Urgent ERCP if concomitant cholangitis is present
     - not for cholestasis or predicted severe disease per se
   - Transfer to ICU or specialist center as needed
     - Deterioration or failure to respond to initial management
     - Intensive support for persistent organ failure
   - Commence enteral nutrition
     - Once normovolemia restored (usually after 6 hours)
     - Commence via NG tube if no gastric stasis
   - No prophylactic antibiotics or probiotics
4. Conservative management and monitoring (at least daily)
   - Clinical evaluation
     - Assess cardiovascular, respiratory, and renal function
     - Detect peritonitis and abdominal compartment syndrome
   - Daily C-Reactive Protein
   - Classify severity (mild, moderate, severe, critical)
   - Detect intolerance of NG EN
     - Advance tube for NJ feeding if needed
     - Consider supplemental Parenteral Nutrition by day 4
5. Indications for "pancreatic protocol CT scan" (rarely in 1st week)
   - For significant clinical deterioration and elevated CRP
   - For suspicion of local pancreatic complications
   - For suspected bowel ischemia
   - For acute bleeding (CTa) (if stable enough & consider embolization)
   - For abdominal compartment syndrome
6. Invasive intervention
   - For deteriorating patient with suspected infected local complication
   - "Step up approach" with initial drain guided by current CT scan (percutaneous or endoscopic drainage)
   - Delay for 3 to 4 weeks with intensive care support, if possible
   - If failure to respond or secondary deterioration, repeat CT scan, and select appropriate minimally invasive technique based on available expertise and equipment
     - Video-assisted retroperitoneal debridement or percutaneous nephroscopic debridement
     - Endoscopic transluminal debridement
     - Ongoing large bore drainage and irrigation
7. Indication for laparotomy
   - Failed "step-up approach" for further debridement/ drainage
   - Acute abdomen (perforation or ischemia)
   - Severe abdominal compartment syndrome (rarely)

PANCREATIC NEOPLASMS

- Neoplasms of the Endocrine Pancreas
- Neoplasms of the Exocrine Pancreas

NEOPLASMS OF THE ENDOCRINE PANCREAS

- The cells of the endocrine pancreas, or islet cells, originate from neural crest cells.
- Some pancreatic endocrine neoplasms are functional, secreting peptide products that produce interesting clinical presentations
- The key to diagnosing these rare tumors is recognition of the classic clinical syndrome; confirmation is achieved by measuring serum levels of the elevated hormone.
- Localization of the tumor can be a challenging step, but once accomplished, the surgery is relatively straightforward.

- As with pancreatic exocrine tumors, the initial diagnostic imaging test of choice for pancreatic endocrine tumors is a multidetector CT scan.

- **EUS** also can be valuable in localizing these tumors, which can produce dramatic symptoms despite their small (<1 cm) size. In contrast to pancreatic exocrine tumors, many of the endocrine tumors have somatostatin receptors (**SSTRs**) that allow them to be detected by a radiolabeled octreotide scan.

INSULINOMA

- ***Whipple's triad***: symptomatic fasting hypoglycemia, a documented serum glucose level <50 mg/dL, and relief of symptoms with the administration of glucose.

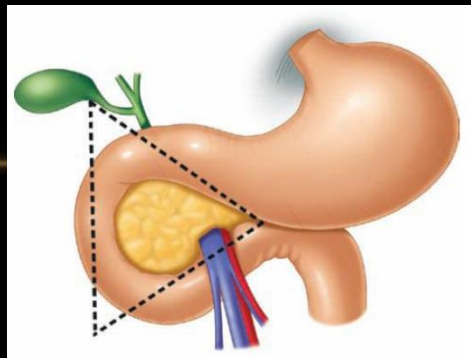
- Serum insulin levels are elevated. C-peptide levels should also be elevated. Unlike most endocrine pancreatic tumors, the majority (90%) of insulinomas are benign and solitary .

GASTRINOMA

- Zollinger-Ellison syndrome, secretes gastrin, leading to acid hypersecretion and **peptic ulceration+diarrhea**.

- The diagnosis of ZES is made by measuring the serum gastrin level. It is important that patients stop taking proton pump inhibitors for this test.

- In most patients with gastrinomas, the level is >1000 pg/mL. In 70% to 90% of patients, the primary gastrinoma is found in Passaro's triangle



- **Vasoactive Intestinal Peptide-Secreting** :The *vasoactive intestinal peptide-secreting tumor* (*VIPoma*) *syndrome* is also called the *WDHA syndrome* due to the presence of *w*atery *d*iarrhea(5 L/d) , *h*ypokalemia, and *a*chlorhydria.
- **Glucagonoma**: Diabetes in association with dermatitis. Glucagon is a catabolic hormone, and most patients present with malnutrition.
- **Somatostatinoma**:inhibits pancreatic and biliary secretions, patients with a somatostatinoma present with gallstones due to bile stasis and diabetes due to inhibition of insulin secretion
- **Nonfunctioning Pancreatic Endocrine Tumors**

NEOPLASMS OF THE EXOCRINE PANCREAS

- Overall, pancreatic cancer has the worst prognosis of all malignancies with a 5-year survival rate of only 6%.
- Pancreatic cancer is more common in the elderly with most patients being >60 years old
- risk factors: obesity and diabetes, smoking, family pos, diets high in fat and low in fiber, chronic pancreatitis

World Health Organization classification of primary tumors of the exocrine pancreas
A. Benign 1. Serous cystadenoma (16%) 2. Mucinous cystadenoma (45%) 3. Intraductal papillary-mucinous adenoma (32%) 4. Mature cystic teratoma
B. Borderline 1. Mucinous cystic tumor with moderate dysplasia 2. Intraductal papillary mucinous tumor with moderate dysplasia 3. Solid pseudopapillary tumor
C. Malignant 1. Ductal adenocarcinoma 2. Serous/mucinous cystadenocarcinoma (29%) 3. Intraductal mucinous papillary tumor

- About two-thirds of pancreatic adenocarcinomas arise within the head or uncinate process of the pancreas; 15% are in the body, and 10% in the tail, with the remaining tumors demonstrating diffuse involvement of the gland.

- Tumors in the pancreatic body and tail are generally larger at the time of diagnosis, and therefore, less commonly resectable.
- Tumors in the head of the pancreas are typically diagnosed earlier because they cause obstructive jaundice.
- Ampullary carcinomas, carcinomas of the distal bile duct, and periampullary duodenal adenocarcinomas present in a similar fashion to pancreatic head cancer but have a slightly better prognosis, probably because early obstruction of the bile duct and jaundice leads to the diagnosis.

- The most critical deficit in the ability to treat pancreatic cancer effectively is the lack of tools for early diagnosis.
- Ultimately, the majority of patients present with pain and jaundice.
- On physical examination, weight loss is evident and the skin is icteric; a distended gallbladder is palpable in about one-fourth of patients. More fortunate patients have tumors situated such that biliary obstruction and jaundice occurs early and prompts diagnostic tests.
- Unfortunately, however, the vast majority of patients are not diagnosed until weight loss has occurred—a sign of advanced disease.
- Most patients do experience pain as part of the symptom complex of pancreatic cancer, and it is often the first symptom.

- A low threshold for ordering a CT scan with “pancreatic protocol” should be maintained for elderly patients with unexplained, persistent, although vague, abdominal pain. As mentioned above, new-onset diabetes in an elderly patient, especially if combined with vague abdominal pain, should prompt a search for pancreatic cancer.

- Direct hyperbilirubinemia and elevated alkaline
- Prothrombin time will be prolonged due to a depletion of vitamin K
- CA19-9

- In patients presenting with jaundice, a reasonable first diagnostic imaging study is abdominal ultrasound. If bile duct dilation is not seen, hepatocellular disease is likely.
- Demonstration of cholelithiasis and bile duct dilation suggests a diagnosis of choledocholithiasis, and the next logical step would be ERCP to clear the bile duct.

- In the absence of gallstones, malignant obstruction of the bile duct is likely, and a CT scan rather than ERCP would be the next logical step. For patients suspected of having pancreatic cancer who present without jaundice, a CT scan should be the first test.

- EUS can be used to detect small pancreatic masses that could be missed by CT scanning and is commonly used when there is a high suspicion for pancreatic cancer but no mass is identified by the CT scan.

- EUS has the added advantage of providing the opportunity for transluminal biopsy of pancreatic masses, although a tissue diagnosis before pancreaticoduodenectomy is not required.

PALLIATIVE SURGERY AND ENDOSCOPY

- For the 85% to 90% of patients with pancreatic cancer who have disease that precludes surgical resection, appropriate and effective palliative treatment is critical to the quality of their remaining life.

- In general, there are three clinical problems in advanced pancreatic cancer that require palliation:

- Pain : oral narcotics, A celiac plexus nerve block
- Jaundice: bile duct stent
- and duodenal obstruction.

SURGICAL RESECTION: PANCREATICODUODENECTOMY

- In a patient with appropriate clinical and/or imaging indications of pancreatic cancer, a tissue diagnosis before performing a pancreaticoduodenectomy is not essential.

CYSTIC NEOPLASMS OF THE PANCREAS

- A cystic neoplasm needs to be considered when a patient presents with a fluid-containing pancreatic lesion.

- However, some of these neoplasms slowly undergo malignant transformation and thus represent an opportunity for surgical cure, which is exceedingly uncommon in the setting of pancreatic adenocarcinoma.
- The dilemma for the surgeon is an accurate assessment of the risk-benefit ratio of resection vs. observation of these lesions in individual patients.
- Radiologic features including the size of the lesion and its growth rate, the density of the lesion, characteristics of the wall such as nodules, septations, or calcifications, and the relationship between the lesion and the pancreatic duct can help categorize these lesions.

- Although a thorough history and radiographic findings often suggest a particular diagnosis, EUS-guided FNA and analysis of cyst fluid or ERCP provide useful additional information to guide clinical decision making. Cysts that contain thick fluid with mucin, elevated carcinoembryonic antigen (CEA), or atypical cells must be treated as potentially malignant

- As discussed in “Complications of Chronic Pancreatitis,” the diagnosis is usually straightforward from the clinical history. Although not usually necessary, analysis of pseudocyst fluid would reveal a high amylase content. The danger comes in mistaking a cystic pancreatic neoplasm for a pseudocyst and incorrectly draining a cystic neoplasm into the GI tract rather than resecting the neoplasm. For this reason, biopsy of the pseudocyst wall is a common step in the management of pancreatic pseudocysts.