ACUTE PANCREATITIS

PANCREAS

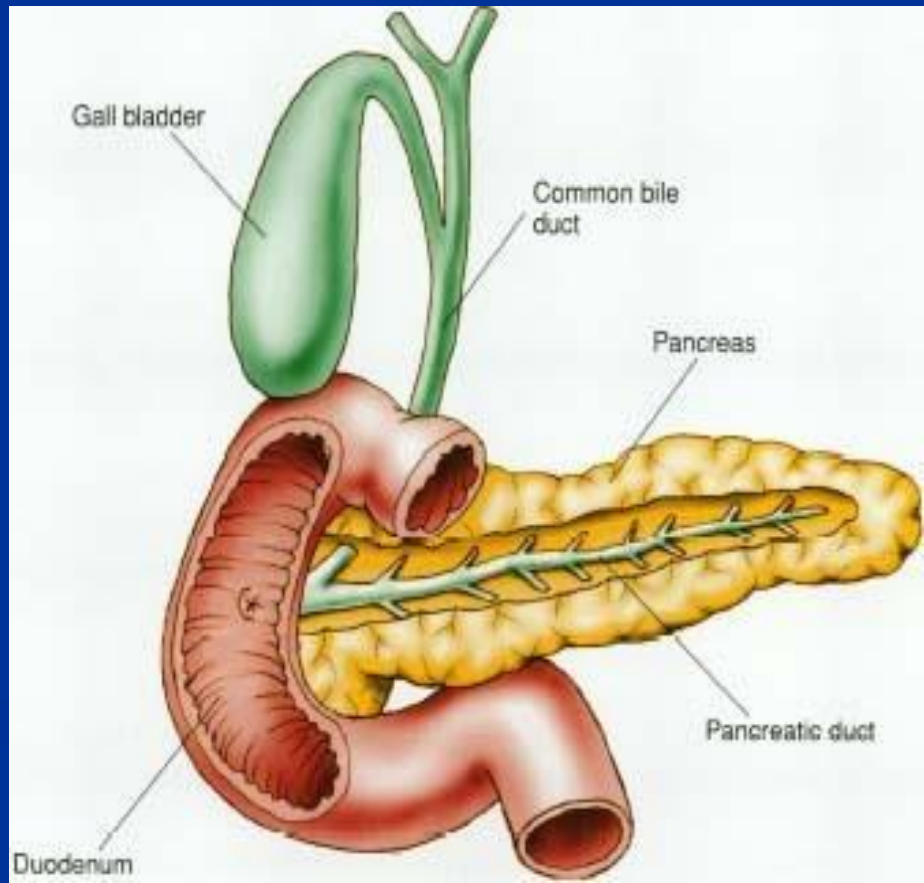
History and anatomy

History :

It was first referred to as the “finger of the liver” in the Talmud, written between 200 bc and 200 ad

Galen named it “pancreas” Pan – All , Kreas - Flesh

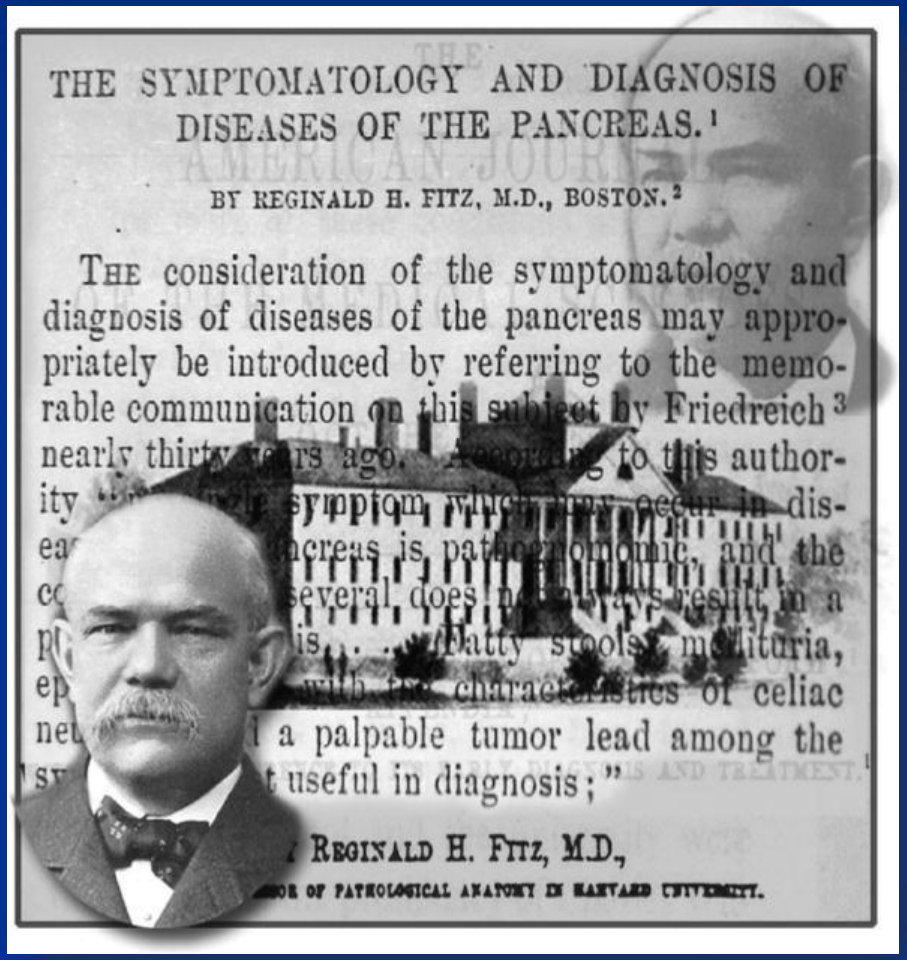
The description of acute pancreatitis by Fitz in 1889



History of the pancreatic surgery

- Johann Georg Wirsung (1589-1643) –describe the anatomiy of the pancreatic duct
- Claude Bernard (1813—1878) – describe the exocrine function of the pancreas
- P.Langerhans (1869) - describe Langerhans cells and endocrinic function of the pancreas.
- Banting and Best (1922) – descoverz insulin (Historical Vignette)
- Eduard Klebs (1870) describe acute pancreatitis
- Reginald Fitz (1899) describe necrotic pancreatitis

Reginald Fitz in 1889 described 3 forms of acute pancreatitis (hemorrhagic, suppurative, and gangrenous) and proposed that fat necrosis was a sequel of severe pancreatitis



THE SYMPTOMATOLOGY AND DIAGNOSIS OF DISEASES OF THE PANCREAS.[1]

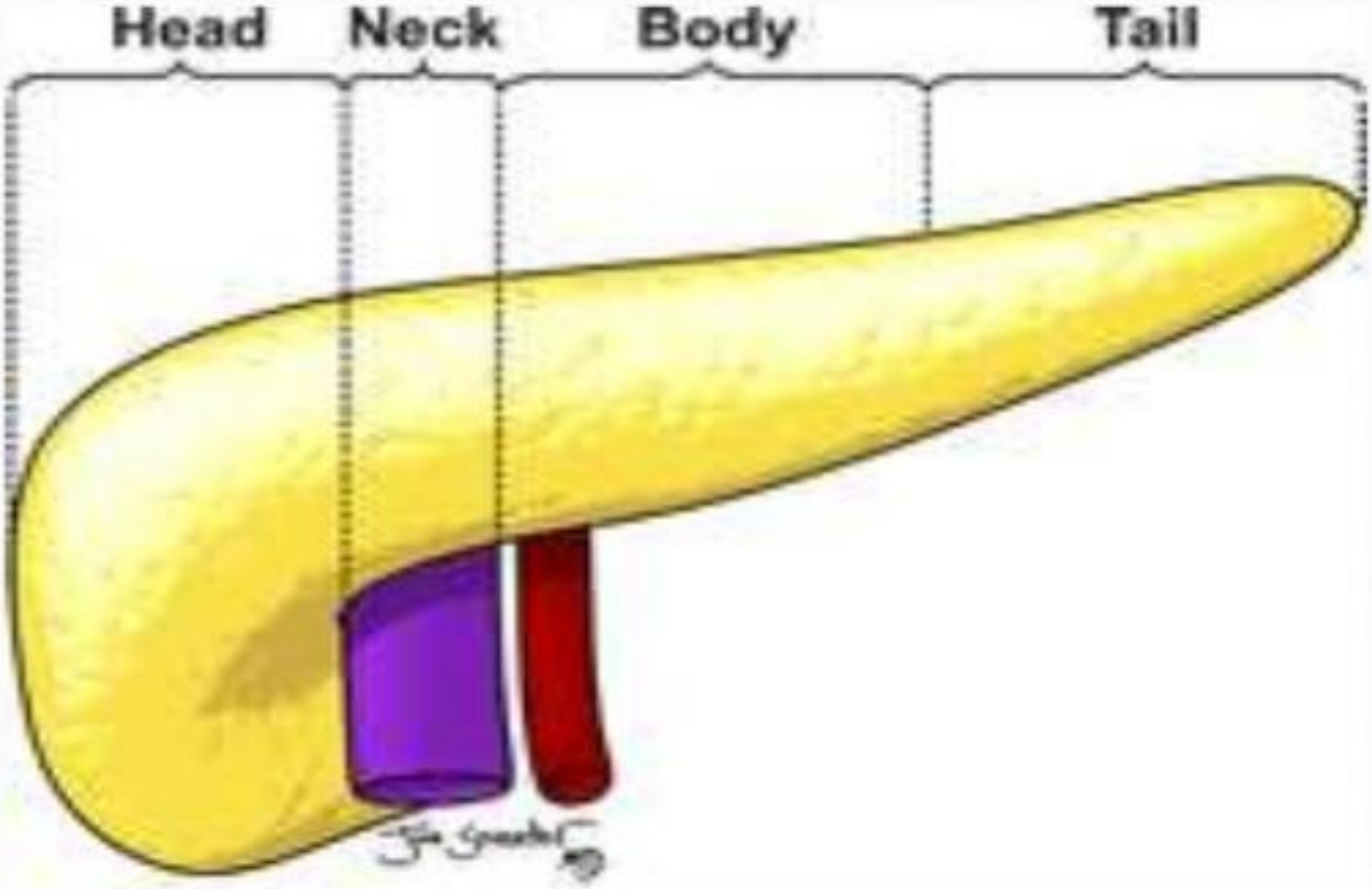
BY REGINALD H. FITZ, M.D., BOSTON.[2]

THE consideration of the symptomatology and diagnosis of diseases of the pancreas may appropriately be introduced by referring to the memorable communication on this subject by Friedreich[3] nearly thirty years ago. According to this authority "... symptom which may occur in disease of the pancreas is pathognomonic, and the combination of several does not always result in a ... Fatty stools, melituria, ... with the characteristics of celiac ... a palpable tumor lead among the ... useful in diagnosis;"

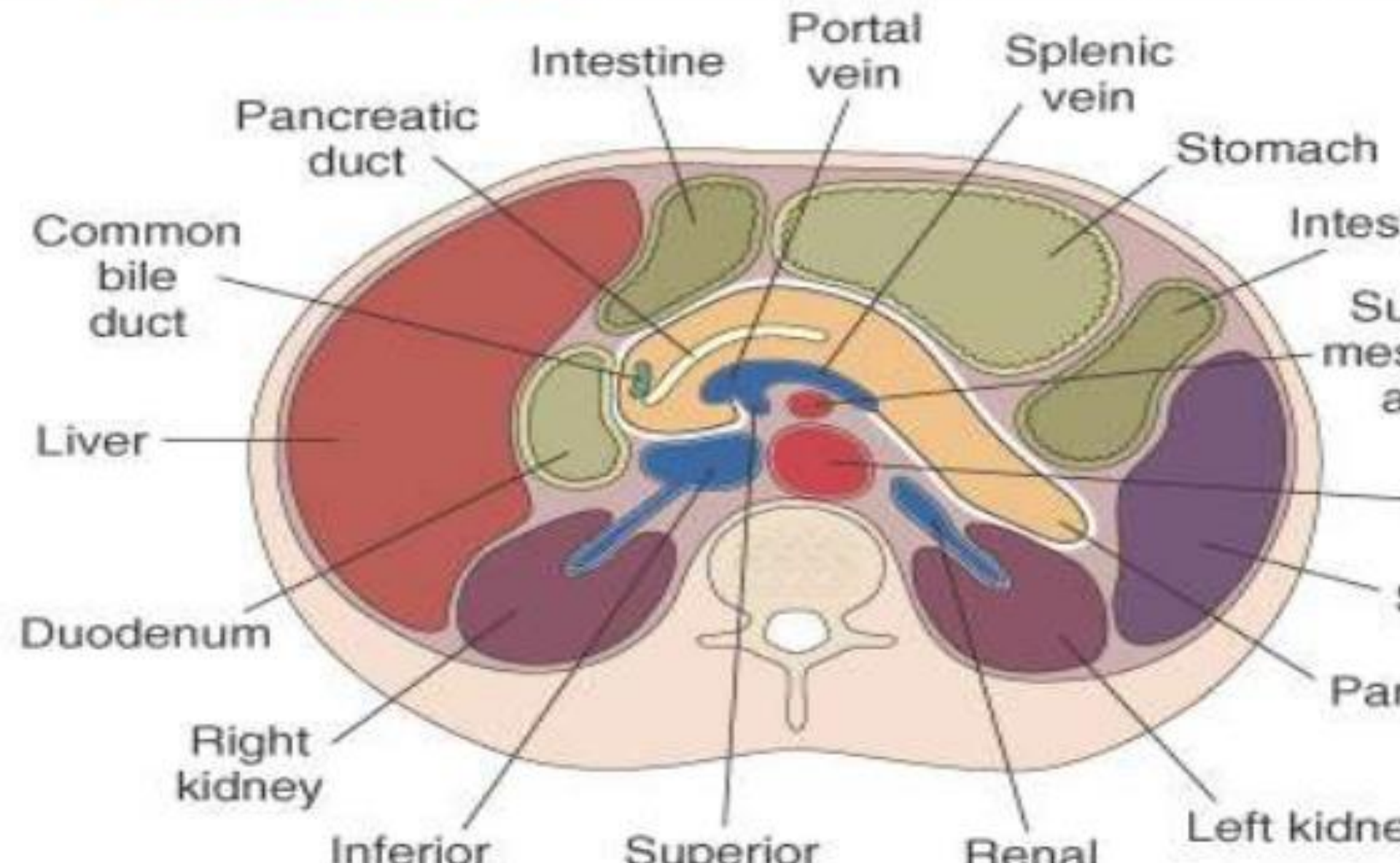
REGINALD H. FITZ, M.D.,

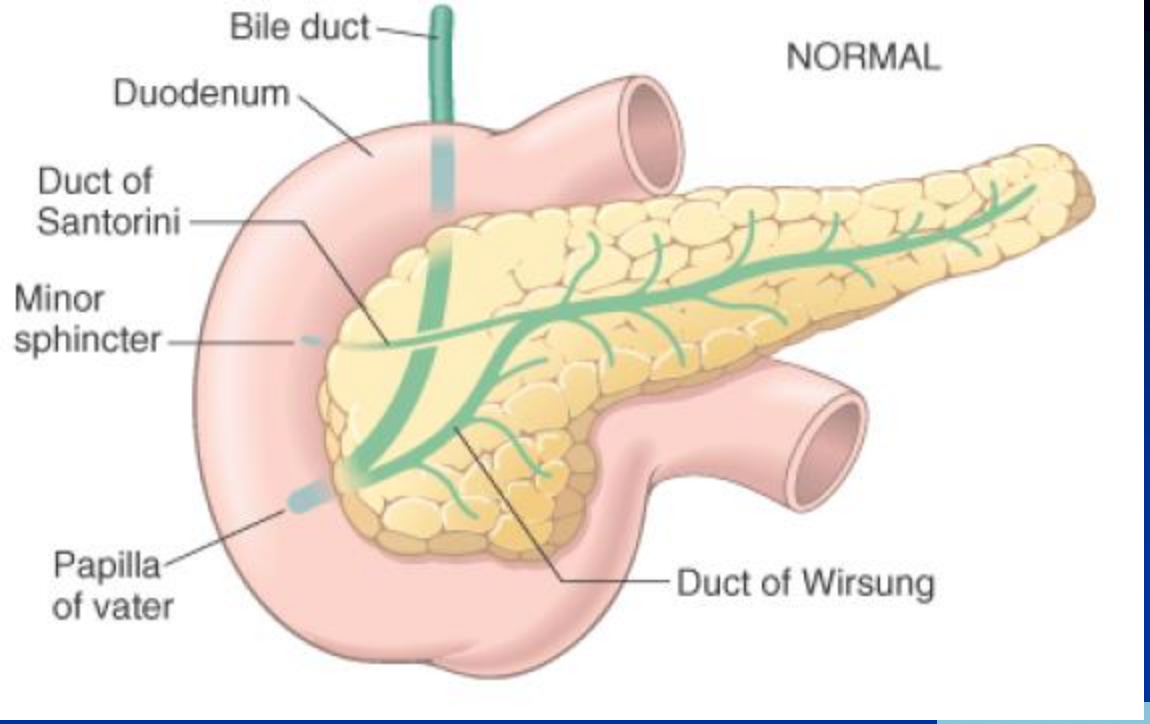
... OF PATHOLOGICAL ANATOMY IN HARVARD UNIVERSITY.

110 gm; 12-15 cm
C-loop



Relationship to surrounding structures:

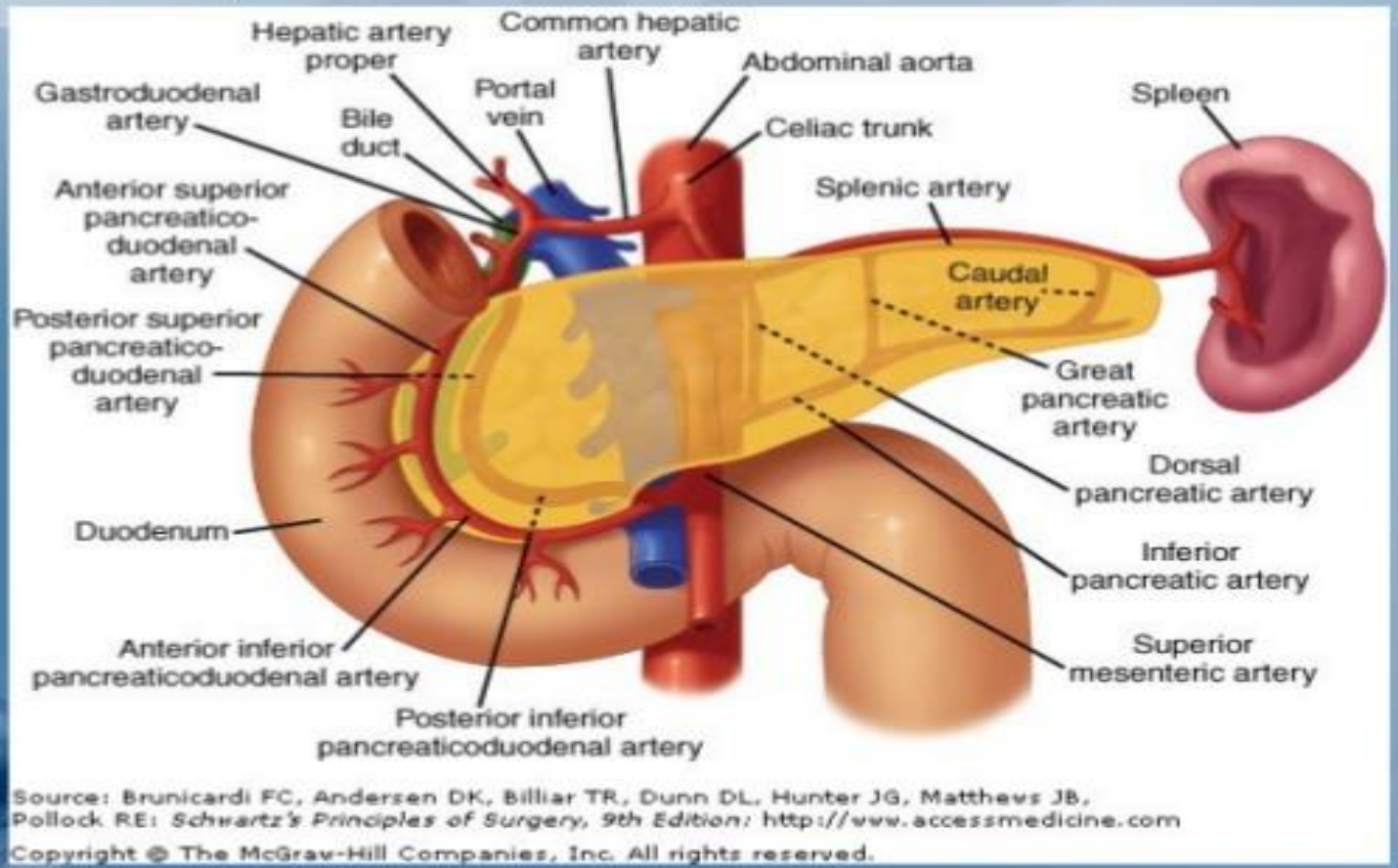




- The duct **of** Wirsung unites with the CBD and drains into the major papilla.
- The duct **of** Santorini, or accessory duct, drains the anterior and superior portion **of** the head into the minor papilla.
- The distal CBD and duct **of** Wirsung traverse the sphincter **of** Oddi (which consists **of** three separate smooth muscles) to enter the duodenum.

Blood supply

Anatomy



Source: Brunicardi FC, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, Pollock RE: *Schwartz's Principles of Surgery, 9th Edition:* http://www.accessmedicine.com

Pancreas – Function

- Exocrine 85%
- bicarbonate ← secretin
  - neutralize HCl
  - activation of pancreatic enzymes
- pancreatic enzymes ← CCK
  - lipase
  - amylase
  - protease
  - phospholipase
- Vagal stimulation

Endocrine 2%
Islets of Langherans
Cell types :- glucagon
insulin
Somatostatin
Pancreatic polypeptide

Enzyme Secretion :

- Enzyme Secretion
- The acinar cell (Latin term meaning “berry in a cluster”) - zymogen granules, lysosomes, or other cell compartments Amylolytic enzymes - such as amylase
- The lipolytic enzymes include lipase, phospholipase A, and cholesterol esterase
- Proteolytic enzymes include endopeptidases (trypsin, chymotrypsin), which act on internal peptide bonds of proteins and polypeptides exopeptidases (carboxypeptidases, aminopeptidases), which act on the free carboxyl- and amino-terminal ends of peptides, respectively; and elastase

Enzyme Secretion :

- Enterokinase, an enzyme found in the duodenal mucosa, cleaves the lysine-isoleucine bond of trypsinogen to form trypsin
- Trypsin then activates the other proteolytic zymogens in a cascade phenomenon

Epidemiology

- Acute pancreatitis is relatively common with an annual incidence of 10-20 million population.
- More than 80% of patients the disease is associated with alcohol or gallstones .
- Race : Hospitalization rates of patients with acute pancreatatis per 100,000 population are three time higher for blacks than for whites
- Sex : More commonly effects Males than Females
- Males - Often related to Alcohol
- Females - Biliary tract disease
- Age : The median age of onset for an alcohol related etiology is 39 years and 69 years for biliary tract disease

Autoprotection of the Pancreas :

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- Autoprotection of the Pancreas
- Autodigestion of the pancreas is prevented by the packaging of proteases in precursor form protease inhibitors – pancreatic secretory trypsin inhibitor (PSTI) serine protease inhibitor - kazal type 1 (SPINK1) These protease inhibitors are found in the acinar cell, the pancreatic secretions, and the α1 and α2 globulin fractions of plasma In addition, low calcium concentrations within the pancreas decrease trypsin activity Loss of any of these protective mechanisms leads to zymogen activation, autodigestion, and acute pancreatitis

General Considerations :

- The incidence of pancreatitis varies in different countries and depends on cause The estimated incidence in England is 5.4/100,000 per year
- in the United States it is 79.8/100,000 per year resulting in >200,000 new cases of acute pancreatitis annually

DEFINITION: :

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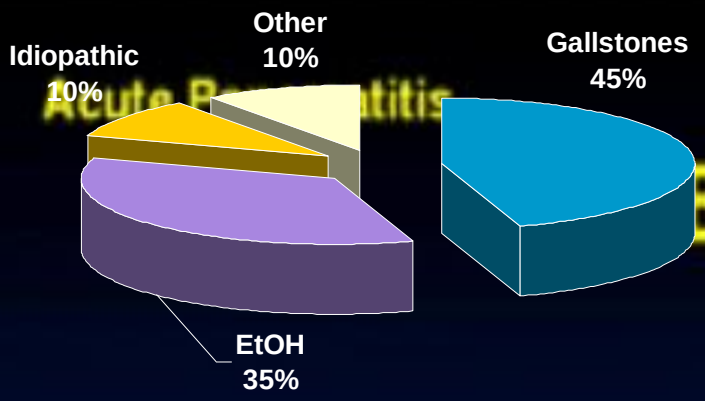
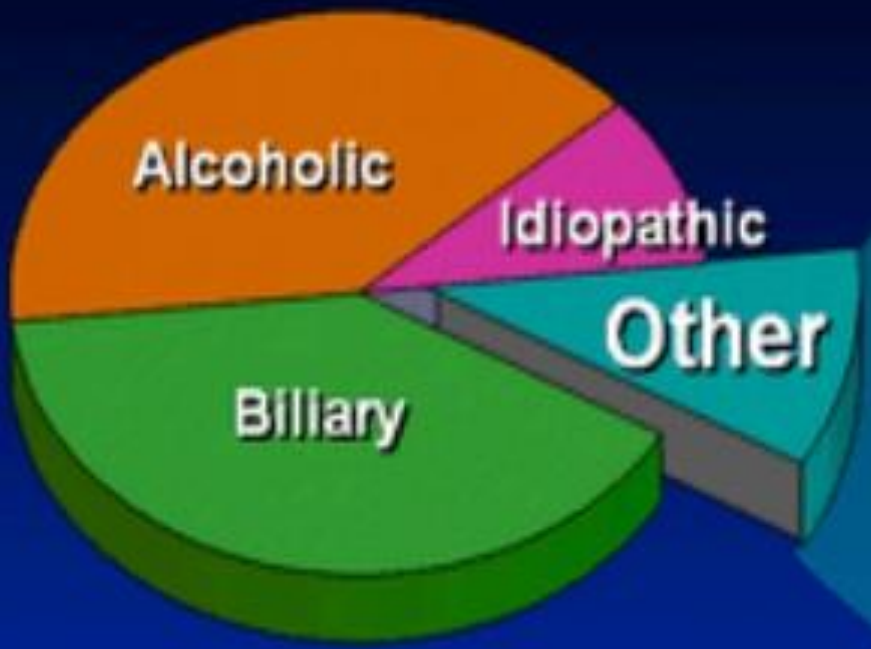
- DEFINITION: 1. “Acute pancreatitis is an acute inflammatory process of the pancreas with varying involvement of other regional tissues or remote organ systems” (BRADLEY)
- 2.Acute pancreatitis is an acute condition with a abdominal pain and is usully asociatted with raised raised pancreatic enzyme levels in the blood or urine as a result of pancreatic inflammation.(BAILEY & LOVE’S)

  3. An acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems Associated with raised pancreatic enzyme levels in blood and/or urine



Acute Pancreatitis: Etiologies



- Autoimmune
- Drug-induced
- Iatrogenic
- IBD-related
- Infectious
- Inherited
- Metabolic
- Neoplastic
- Structural
- Toxic
- Traumatic
- Vascular

Etiology

- Alcohol (30-40%)
  - Mechanism not fully understood
  - Not all alcoholics get pancreatitis (only about 15%)
  - This suggests a subset of the population predisposed to pancreatitis, with alcohol acting more as a co-precipitant

Etiology

- Gallstones (35%-60%)
  - Gallstone pancreatitis risk is highest among patients with small GS < 5mm and with microlithiasis
  - GS pancreatitis risk is also increased in women > 60 yrs
  - Drugs and Toxins (5%)
  - Azathioprine
- Cimetidine
- Estrogens
- Enalapril
- Erythromycin
- Furosemide
- Multiple HIV medications
- Scorpion Bites
- Sulfonamides
- Thiazides
- TMP/SMX

Trivia

- What is the name of the scorpion that causes pancreatitis?
  - Hint: you won't find it in the USA

- *Tityus Trinitatis*
- (Found in Central/
- South America and
- the Caribbean)

Etiology – Trauma

- Blunt Trauma
  - Automobile
  - Bicycle handlebar injuries
  - Abuse
- Iatrogenic – ERCP (1-7%)
  - Likely secondary to contrast but also very operator dependant
  - Risk is also increased with Sphincter of Oddi manometry

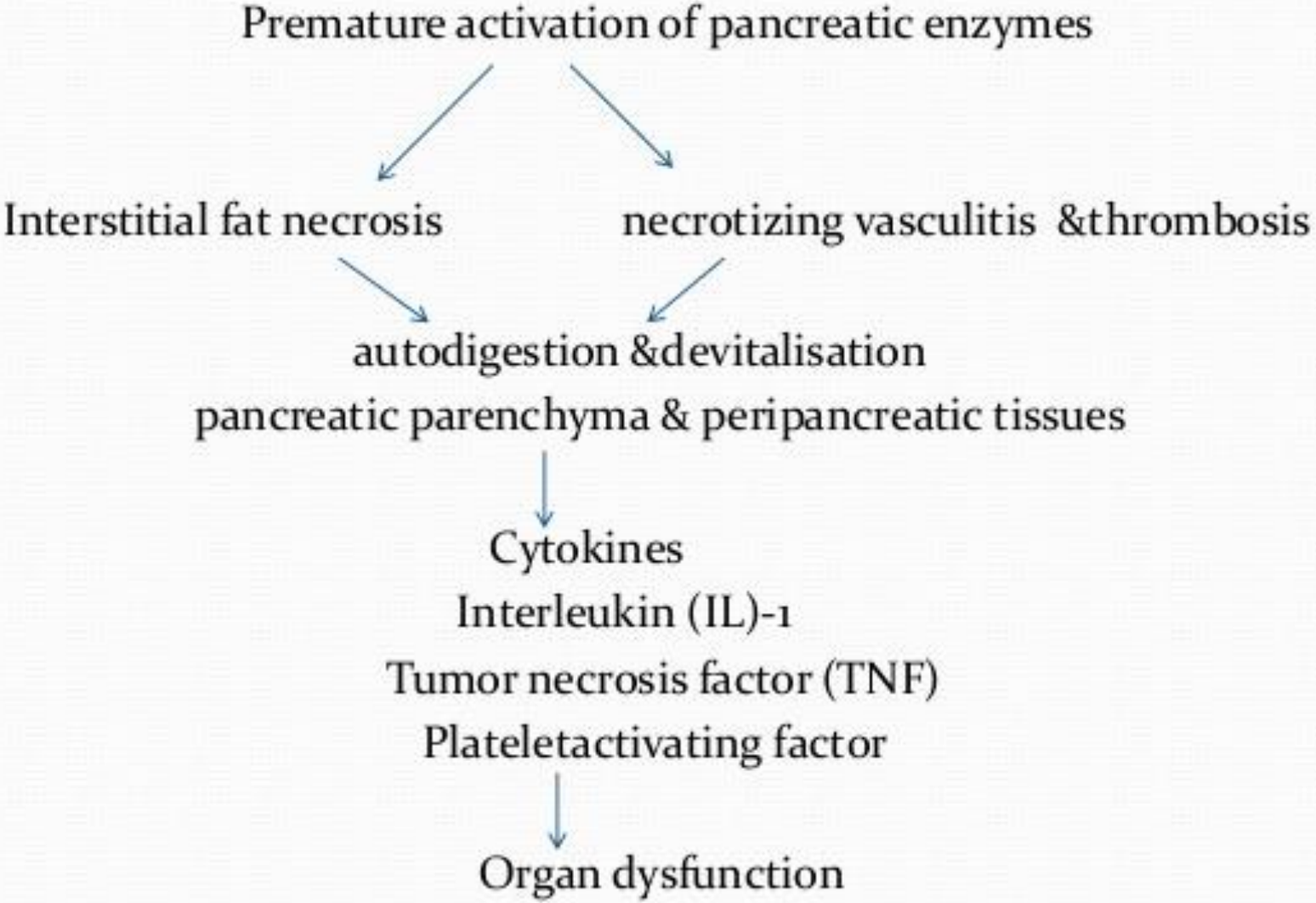
Etiology – Multi-System Disease

- Diabetic Ketoacidosis (10-15%)
- Hemochromatosis
- HUS
- Hypercalcemia
- Hyperparathyroidism
- Hypertriglyceridemia
- IBD
- Malnutrition
- Severe PUD
- Renal Failure
- SIRS
- SLE and other connective tissue dissorders
- Status-Post solid organ and BM transplant
- Vasculitis

Etiology – Idiopathic

- Experts suggest that idiopathic pancreatitis should account for no more than 5-10% of the total cases, yet the broadly quoted percentage in the literature at this time in the US is currently 20-25%.

Pancreatic Injury: Pathophysiology



- Trypsinogen

  Trypsinogen activation peptide (TAP) **I**
- Trypsin

- Inflammatory cascade **(IL6, IL-8, TNF-α)** **II**

  C - reactive protein **III**
- Pancreatic injury

- **Amylase, Lipase, Trypsinogen** **IV**

Pathogenesis :

- Autodigestion is one pathogenic theory, according to which pancreatitis results when proteolytic enzymes (e.g., trypsinogen, chymotrypsinogen, proelastase, and phospholipase A) are activated in the pancreas rather than in the intestinal lumen
- A number of factors (e.g., endotoxins, exotoxins, viral infections, ischemia, anoxia, and direct trauma) are believed to activate these proenzymes
- Activated proteolytic enzymes, especially trypsin, not only digest pancreatic and peripancreatic tissues but also can activate other enzymes, such as elastase and phospholipase

Pathogenesis :

-
- Evolves in three phases
- Initial phase – intrapancreatic digestive enzyme activation and acinar cell injury
- Zymogen activation mediated by lysosomal hydrolases such as cathepsin B (co localization hypothesis by van Acker GJ, Perides G, Steer ML)
- The second phase – activation, chemoattraction, and sequestration of neutrophils
- Neutrophil sequestration can activate trypsinogen
- Thus, intrapancreatic acinar cell activation of trypsinogen could be a two-step process, i.e., with a neutrophil-independent and a neutrophil-dependent phase

Pathogenesis :

- effects of activated proteolytic enzymes and cytokines, released by the inflamed pancreas, on distant organs Activated proteolytic enzymes, especially trypsin, not only digest pancreatic and peripancreatic tissues but also activate other enzymes such as elastase and phospholipase The active enzymes then digest cellular membranes and cause proteolysis, edema, interstitial hemorrhage, vascular damage, coagulation necrosis, fat necrosis, and parenchymal cell necrosis.

Pathogenesis :

- Cellular injury and death result in the liberation of bradykinin peptides, vasoactive substances, and histamine vasodilation, increased vascular permeability, and edema with profound effects on many organs, most notably the lung
- The systemic inflammatory response syndrome (SIRS) and acute respiratory distress syndrome (ARDS) as well as multiorgan failure may occur as result of this cascade of local as well as distant effects (lecithinase)

Pathophysiology and Clinical Phases of Acute Pancreatitis

PHASE	INITIAL	EARLY	MIDDLE	LATE
TIMING	Hours	1st week	2nd week	3rd-4th week
MAJOR EVENTS	Altered intra-acinar protein traffic Accumulation of trypsinogen in the interstitial space	Inappropriate activation of proteases Necrosis Microcirculatory disorders Macrophage activation Progression of necrosis		Gut and biliary bacteria Infection of necrosis
DEATHS	?	32%	12%	19% 37%
Causes M.O.F.	?	26%	0%	0 0
Causes Infection	?	0%	5%	12% 28%

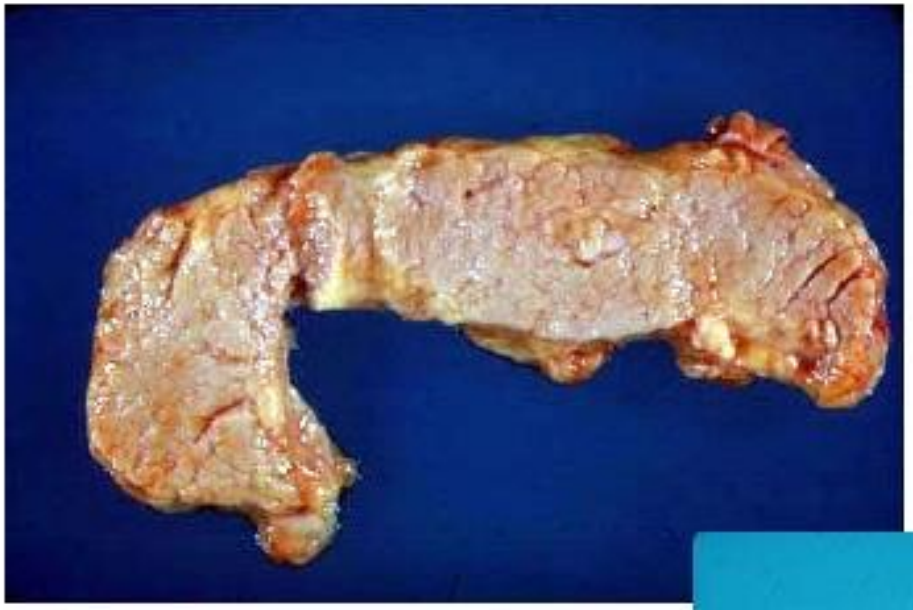
periods of acute pancreatitis:

1. period of hemodynamic disturbances (1-3 days)
2.functional failure of parenchymal organs (5-7 days)
3. postnecrotic complications (3-4 weeks)

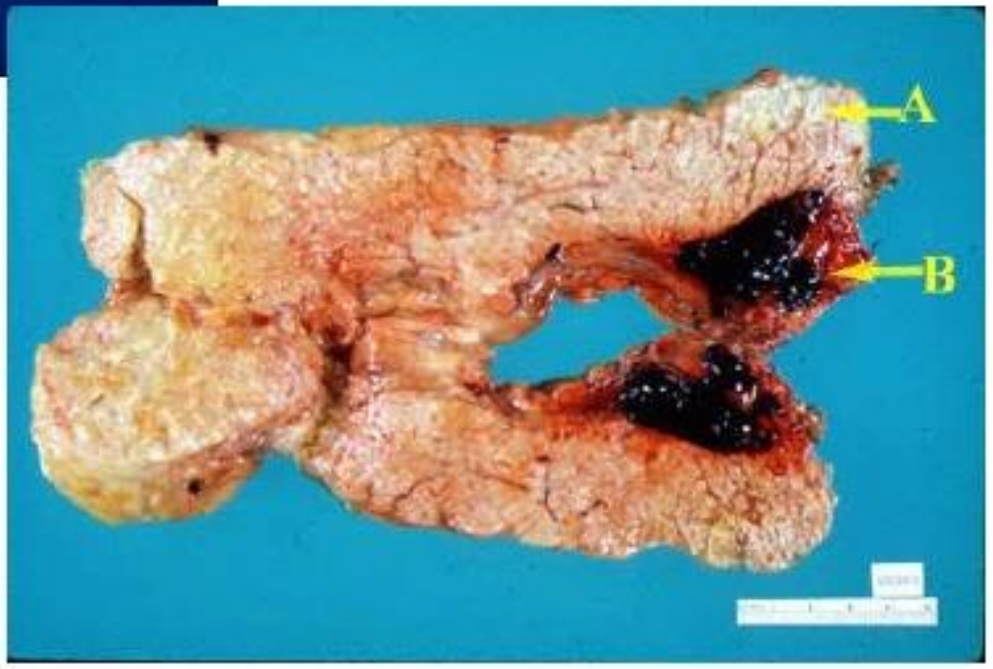
- Destruction of the blood vessels with subsequent interstitial hemorrhage

Acute Pancreatitis; Haemorrhage and necrosis

Normal pancreas

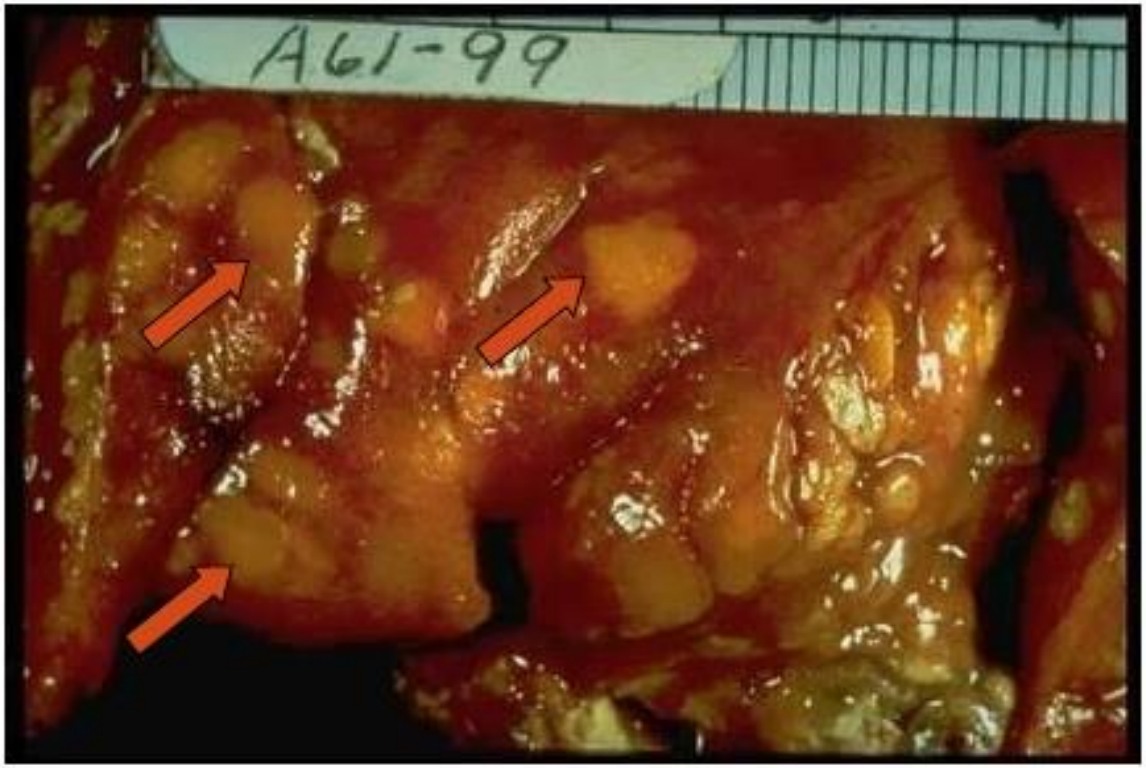


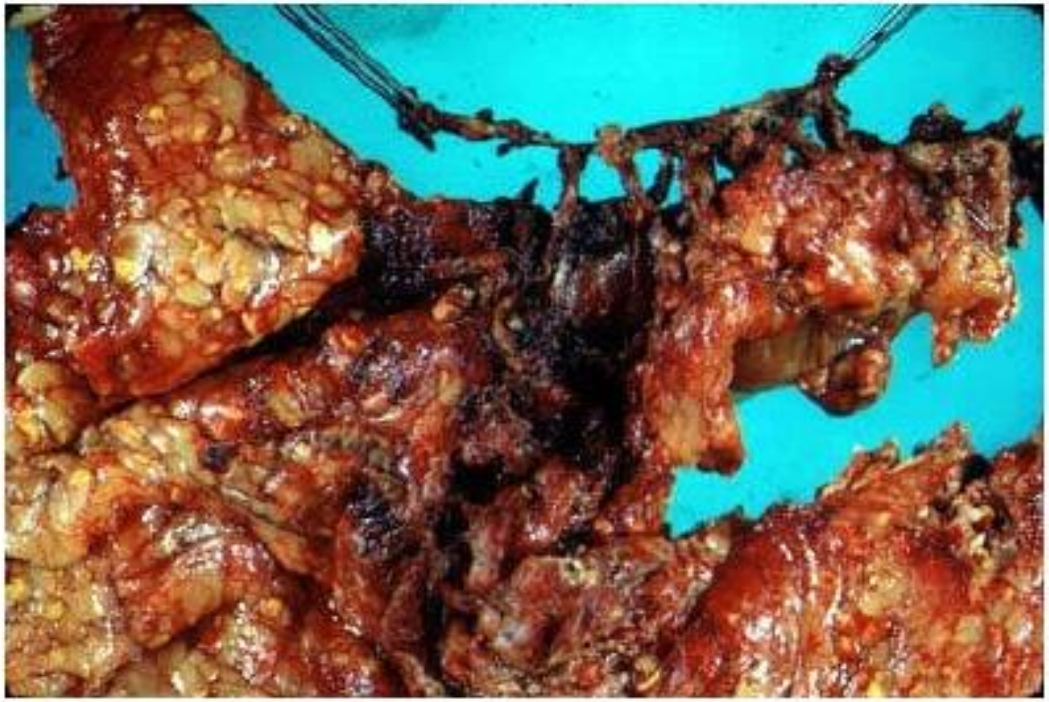
Acute pancreatitis



- Necrosis of regional fat by lipolytic enzymes

Saponification of fat





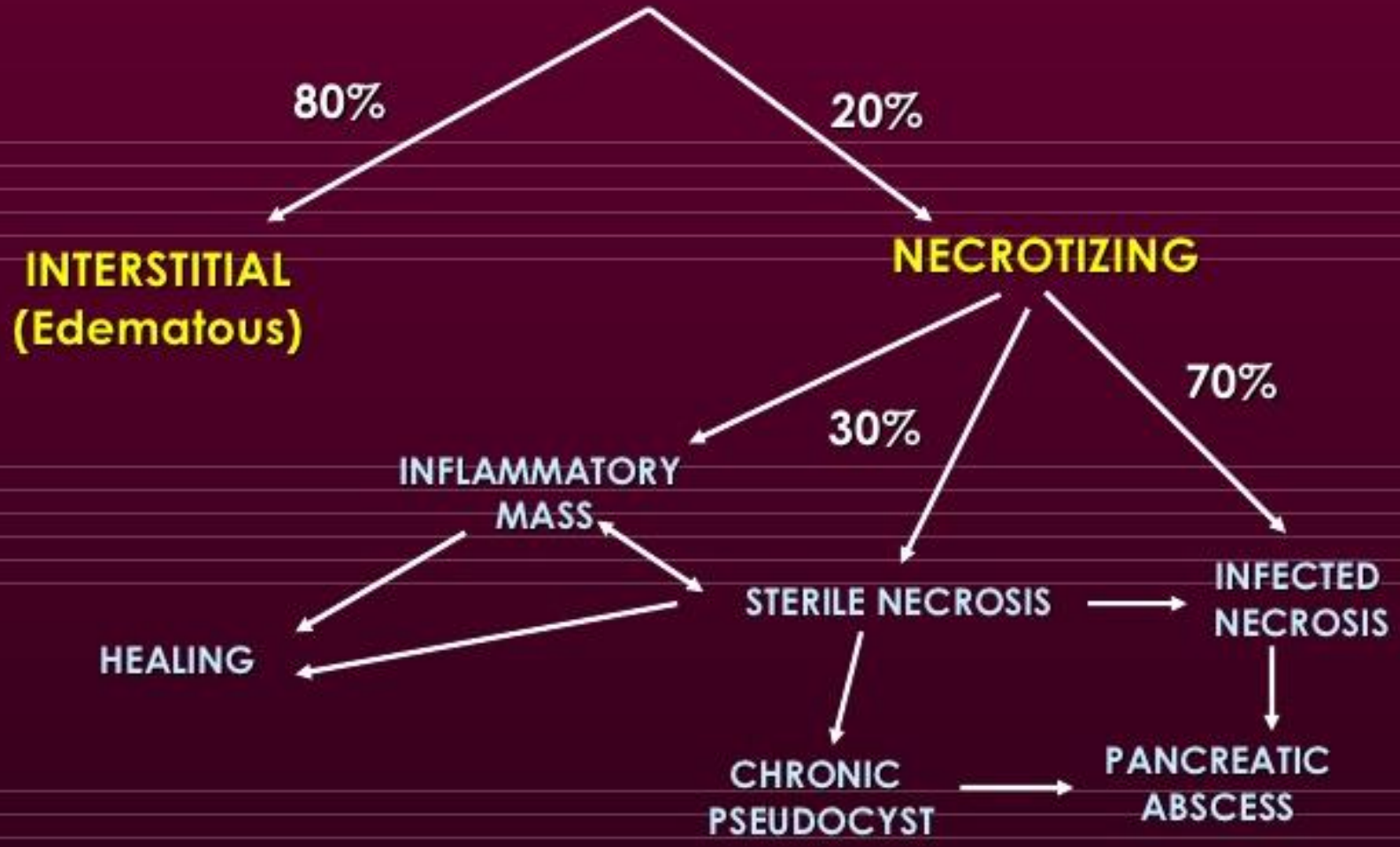
Acute Pancreatitis Necrosis of regional fat by lipolytic enzymes

A surgical specimen of the transverse colon and greater omentum shows extensive fat necrosis. Note bright yellow foci. The mesenteric fat has been completely digested away so as to reveal the isolated blood vessels.

CLASIFICTION ACUTE PANCREATITIS (Beger) Atlanta, 1992

- **oedematosis (interstitial) AP–** **71%**
- **necrotic AP –** **21%**
- Steril necrosis – 68%
- Infected necrosis – 32%
- **pancreatic abceses –** **3%**
- **Pseudopancreatic cyst –** **6%**

Acute Pancreatitis



CLASIFICATION OF THE ACUTE PANCREATITIS (V.C. Saveliev)

- **Anatomical -clinical forme**
- acute **oedema**
- Lipidic pancreonecrosis
- hemoragic pancreonecrosis
  - **By spreding**

  local, subtotal, total.
  - **Evoluţia:**
- Abortion
- Progresiv.
  - **By evolution**
- Pancreatic shok,
- Poliorganic failure
- Puss complications.

Classification of severity

Atlanta classification (1992)

- *Mild* : lack of organ failure and complications
- *Moderate* : transient organ failure and/or complications < 48hr
- *Severe* : persistent organ failure and complications

Reference : 2012 revision of atlanta classification of acute pancreatits

Organ failure and systemic complications

Evidence of organ failure

- Respiratory failure
  - PaO2 of less than 60 mm Hg
  - Ventilatory support.
- Cardiovascular system failure
  - Systolic BP of < 90 mm Hg
  - signs of peripheral hypoperfusion
  - need for vasopressor or inotropic agents
- Renal failure
  - serum creatinine level > 300 µmol/L
  - urine output < 500 mL/24 hr or < 180 mL/8 hr
  - need for hemo- or peritoneal dialysis.

Evidence of organ failure

- Central nervous system failure
  - Glasgow Coma Scale score greater than 6 in the absence of sedation
  - Sudden onset of confusion or psychosis.
- Hepatic failure
  - Serum bilirubin levels greater than 100 µmol/L
  - Alkaline phosphatase levels >3× the normal range.
- Hematologic system failure
  - Hematocrit level < 20%,
  - WBC < 2,000/mm3,
  - Platelet count of < 40,000/mm3.

ACUTE PANCREATITIS COMPLICATIONS

Toxic COMPLICATIONS

- Pancreatogen shok
- Peural efusion, „ pancreatogenic pneumonia ”, pancreatogenic lung,
- toxic pancreatogenic distrofia of the liver and kidney
- Erozive pancreatogenic gastritis
- coma.

POSTNECROTIC COMPLICATIONS

- degenerative (parapancreatic INFILTRATE, pseudocyst)
- supurative (pancreatic abcesis, abcesis of bursa omentalis, retroperitoneal abces-flegmon, subhepatic abcesis, subphrenicus abcesis, peritonitis

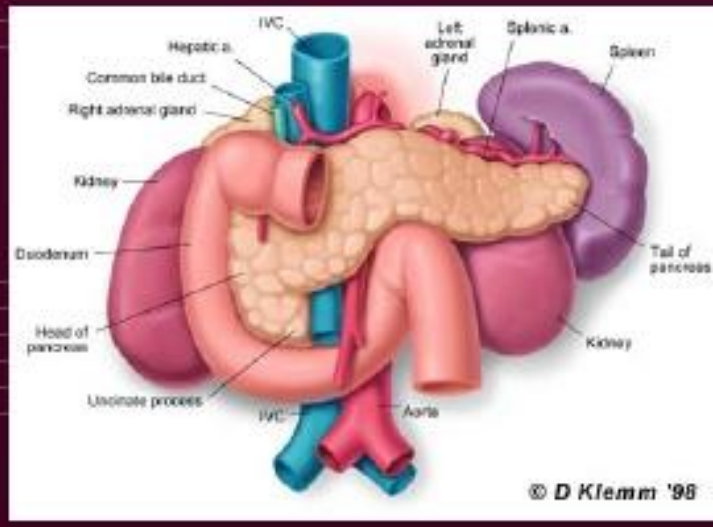
Visceral complications

- A fistula,
- erozive bleeding,
- ocluzive complication (compresie, trombosis).

Clinical Presentation

Anorexia
Weakness
Tachycardia
+/- Fever

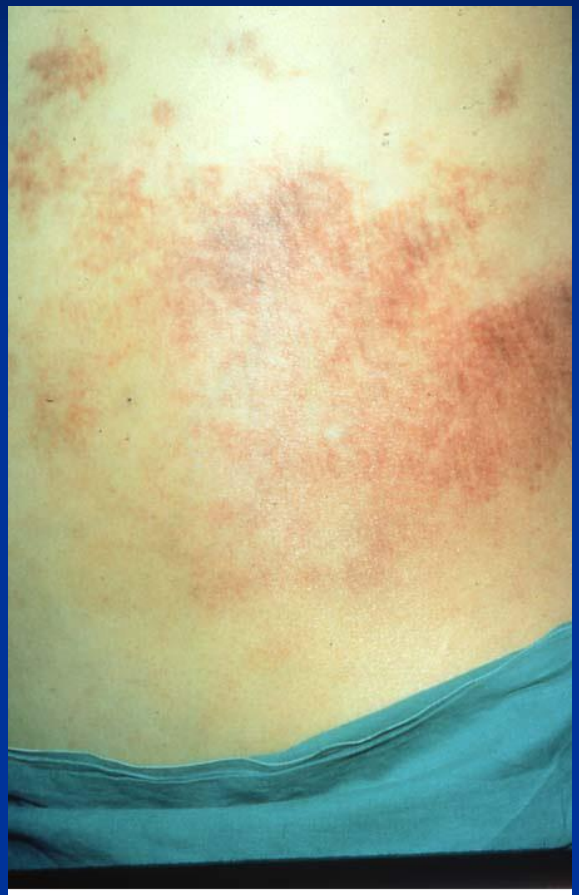
- Pain (95%)
  - Acute onset
    - Mid-abdominal or mid-epigastric
    - Radiates to the back (50%)
  - Peak intensity in 30 minutes
    - Lasts for several hours
- Nausea and vomiting (80%)
- Abdominal distension (75%)
- Abdominal guarding and tenderness (50%)
- Restlessness and agitation



Acute Pancreatitis - Clinical Presentation

- Less common
  - left pleural effusion
  - jaundice (more common in gallstone pancreatitis)
  - epigastric mass (pseudocyst)

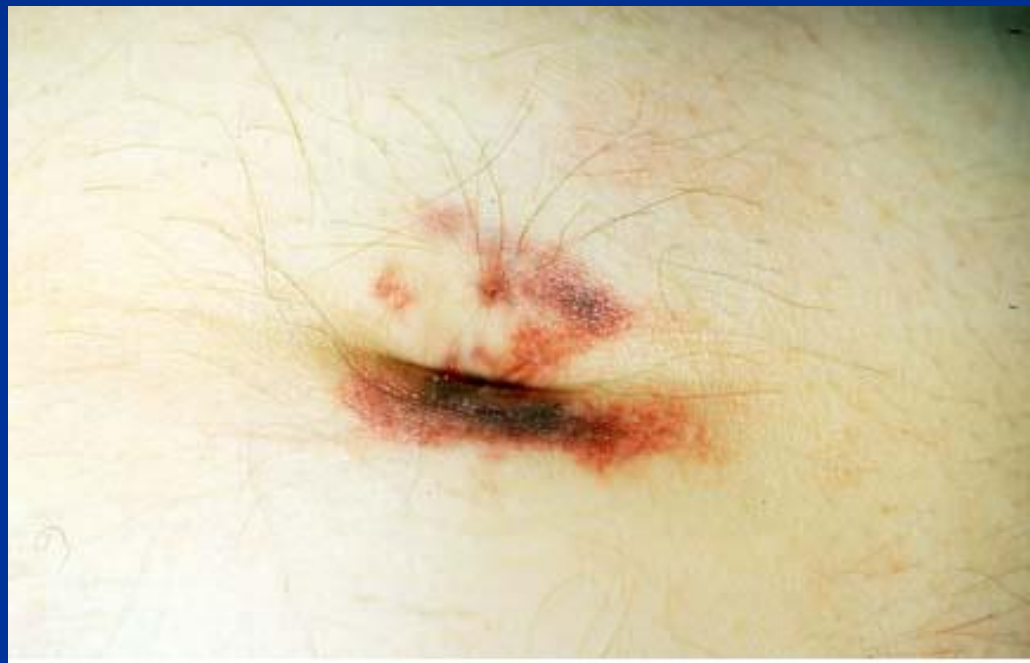
flank discoloration (Grey Turner sign)



Source: Lichtman MA, Shafer MS, Felgar RE, Wang N:
Lichtman's Atlas of Hematology: http://www.accessmedicine.com

- Grey Turner sign - flank discoloration due to retroperitoneal bleed in pt. with pancreatic necrosis (rare)

periumbilical discoloration (Cullen's sign)



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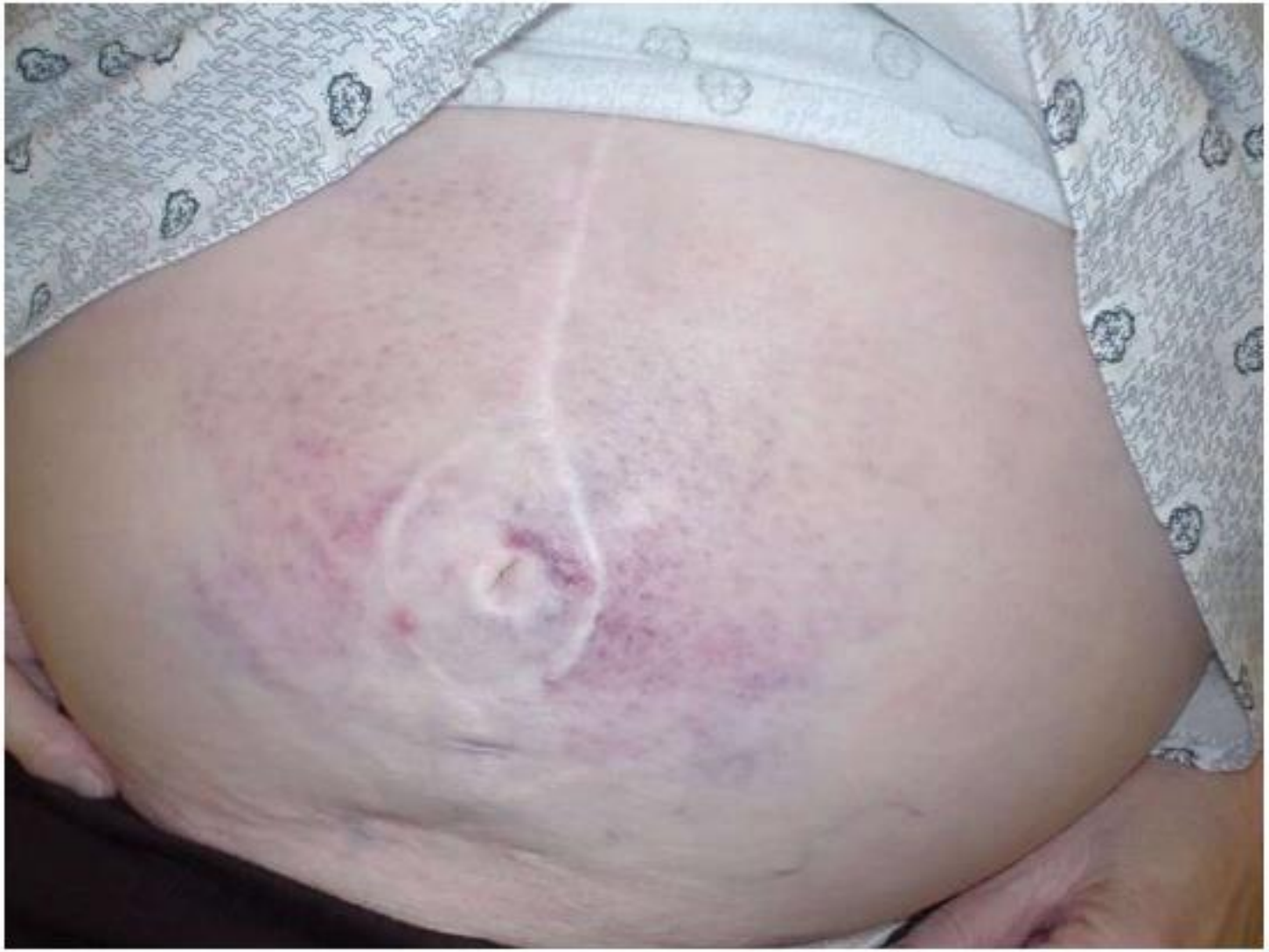
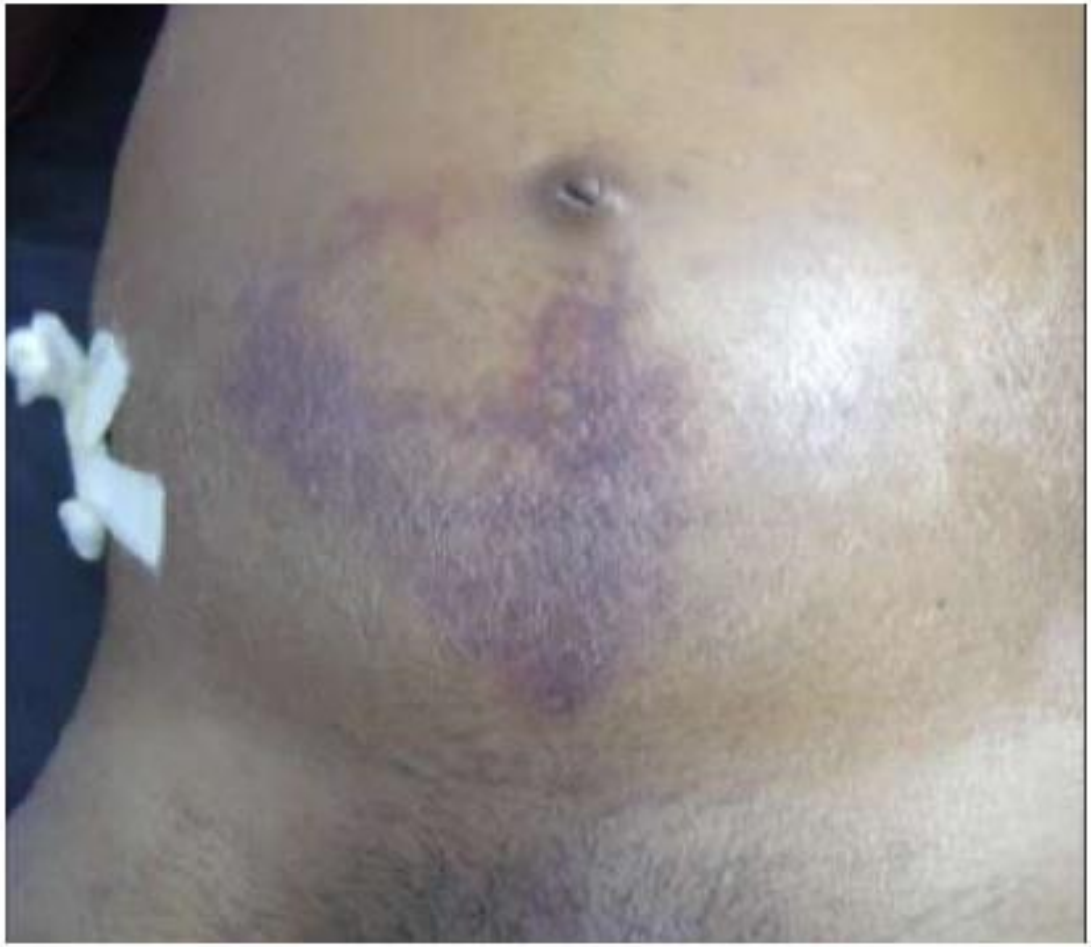
Cullen's sign - periumbilical discoloration (rare)

Cullen sign – discolouration around umbilicus



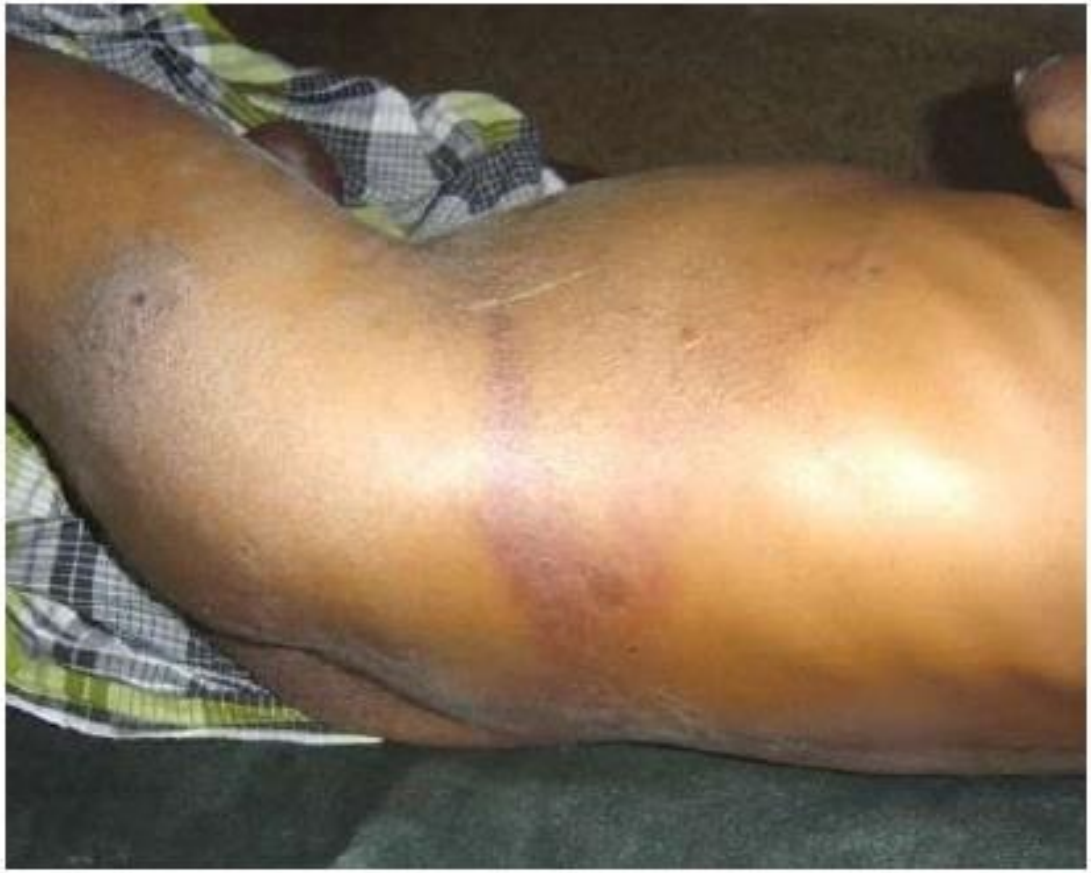
Cullen's sign – bluish peri-umbilical discoloration

Cullen sign

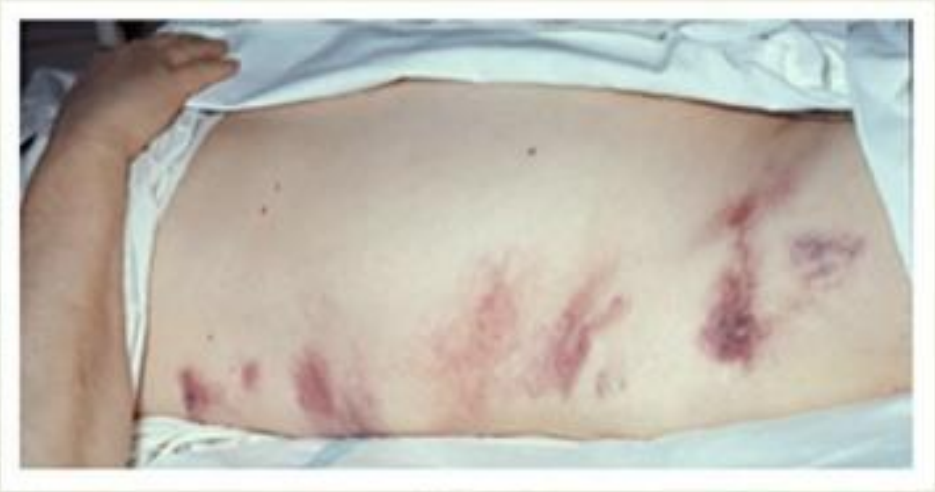


Cullen's sign - periumbilical discoloration (rare)

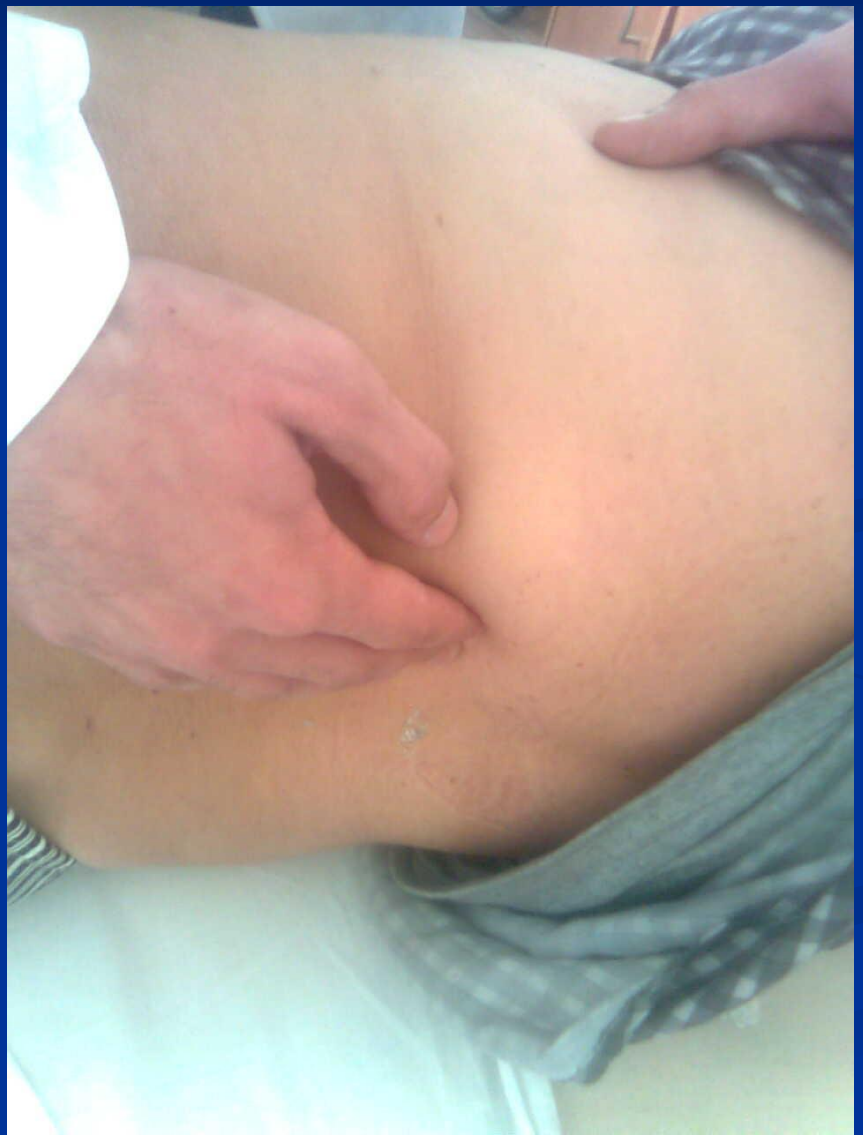
Grey-Turner sign- discolouration in the flanks



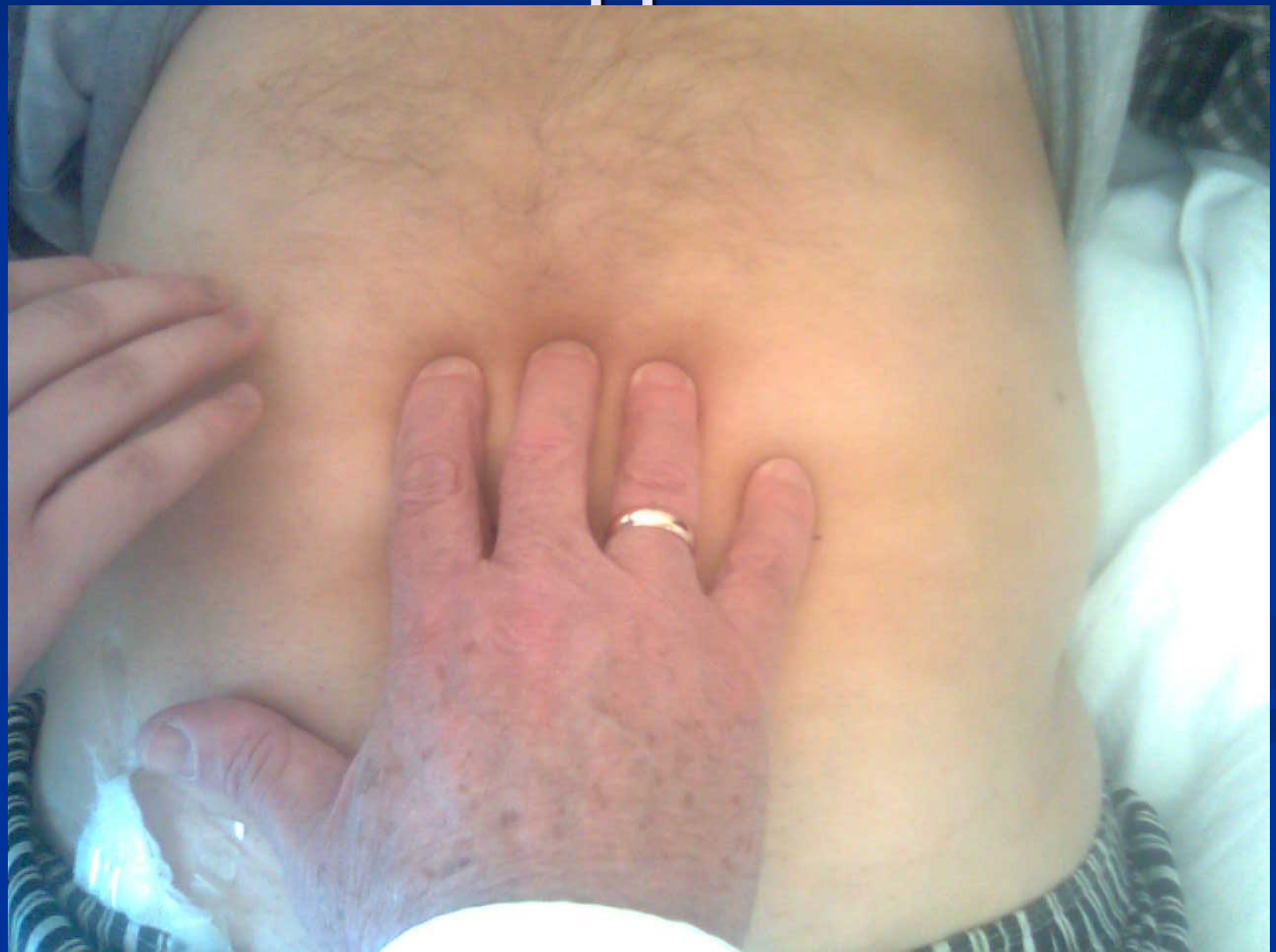
- *Grey Turner's Sign*
- Hemorrhagic discoloration of flank



Meio – Robson sign – the pain in the left costo-vertebral ungle.



Korte symptom - mild-to-moderate muscular rigidity and pain may be present in the upper abdomen



Acute Pancreatitis
Clinical Presentation

- 2º systemic effects – **worse prognosis***
  - Respiratory distress syndrome
  - Renal failure
  - Subcutaneous fat necrosis

Differential

- Not all inclusive, but may include:
  - Cholecystitis and Biliary Colic
  - Cholelithiasis
  - Cholangitis
  - Intestinal obstruction
  - Mesenteric Ischemia
  - MI (inferior)
  - Distal aortic dissection
  - PUD
  - Aneurysms, Abdominal
  - Gastroenteritis
  - Hepatitis

Normal level 23-85 U/L

Diagnosis – Amylase

- Amylase – levels start increasing from 2-12 hrs after the onset of the symptoms and peaks at 12-72 hrs and remain elevated for 4-5 days
- return to normal within one week.
- High specificity when using levels >3x normal
- Many false positives (see next slide)
- Most specific = pancreatic isoamylase (fractionated amylase)

Urine amylase

Female: 21-447 U/L
Maili: 16-491 U/L

- urinary levels may be more sensitive than serum levels.
- Urinary amylase levels usually remain elevated for several days after serum levels have returned to normal.

Diagnosis – Amylase Elevation

- Pancreatic Source
  - Biliary obstruction
  - Bowel obstruction
  - Perforated ulcer
  - Appendicitis
  - Mesenteric ischemia
  - Peritonitis
- SalivaryUnknown Source
  - Renal failure
  - Head trauma
  - Burns
  - Postoperative
- Unknown Source
  - Parotitis
  - DKA
  - Anorexia
  - Fallopian tube
  - Malignancies

Diagnosis - Lipase

- Lipase – increases within 4-8 hrs of symptoms and peak at about 24 Hrs, and decreased within 8-14 days.
- The preferred test for diagnosis
- Remains elevated for days
- Sensitivity 86-100% and Specificity 60-99%
- >3X normal S&S ~100%

- Liver enzyme elevation – especially GS pancreatitis
- Elevated ALT > 3x normal (in a non-alcoholic) has a positive predictive value of 95% for GS pancreatitis

- Hypocalcemia
- Hypoglycemia
- Leukocytosis

Conditions Associated with Hyperamylasemia and Hyperlipasemia

	Amylase	Lipase
Paroditis	yes	no
Tumors	yes	no
Biliary disease	yes	slight
Pancreatitis	yes	yes
Renal failure	yes	slight
Intestinal obstruction, ulceration, ischemia	yes	yes
Ectopic pregnancy	yes	no
Macroamylasemia	yes	no
Perforated viscus	yes	yes

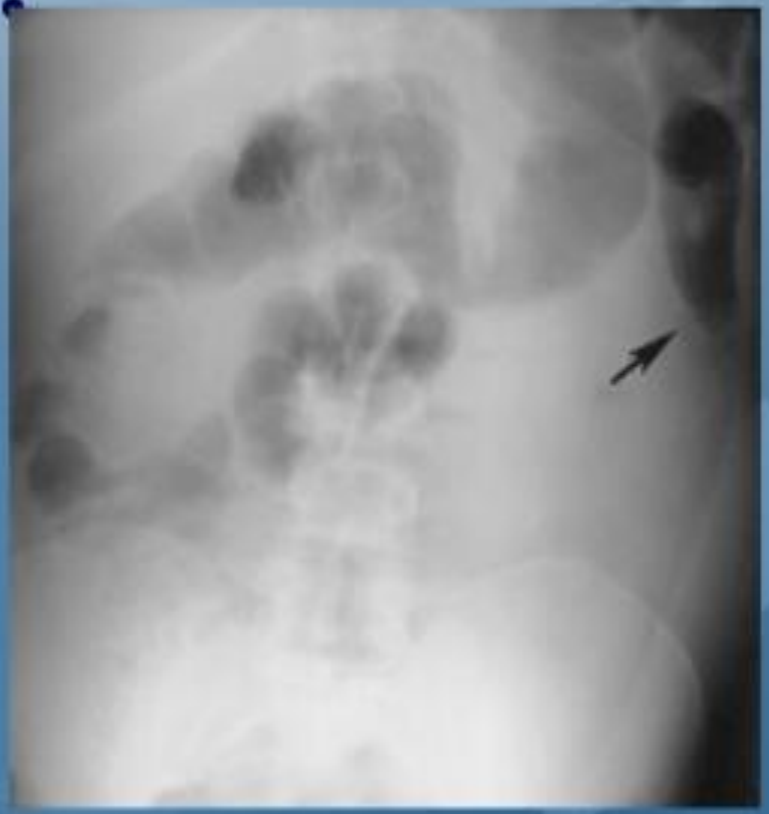
Plain Abdominal Radiograph

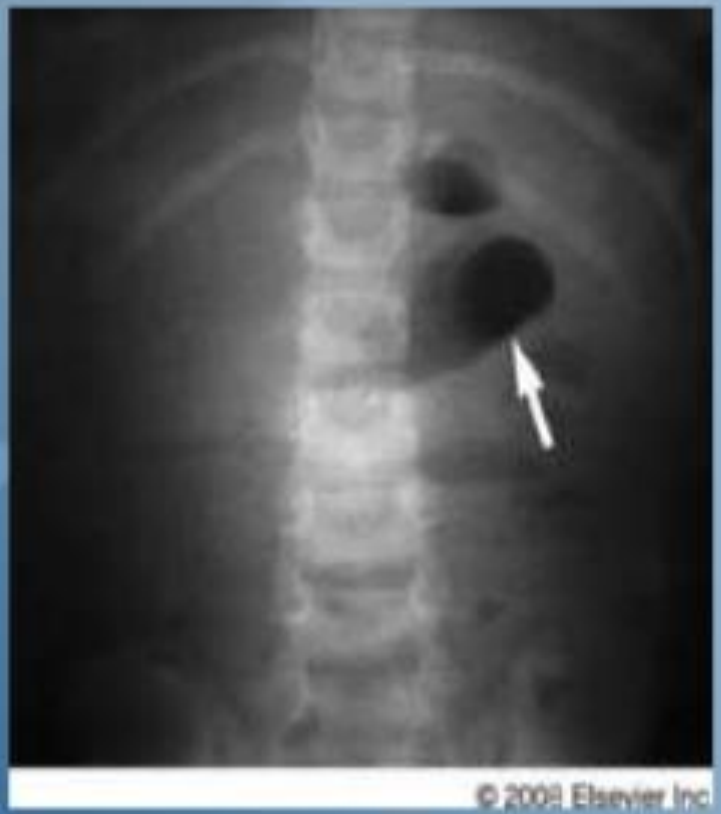
Acute Pancreatitis
X-ray (Imaging)

- Bowel ileus
- "Sentinel Loop"
- "Colon cut off sign"
- Loss of psoas shadow

- Helps exclude other causes of abdominal pain: bowel obstruction and perforation

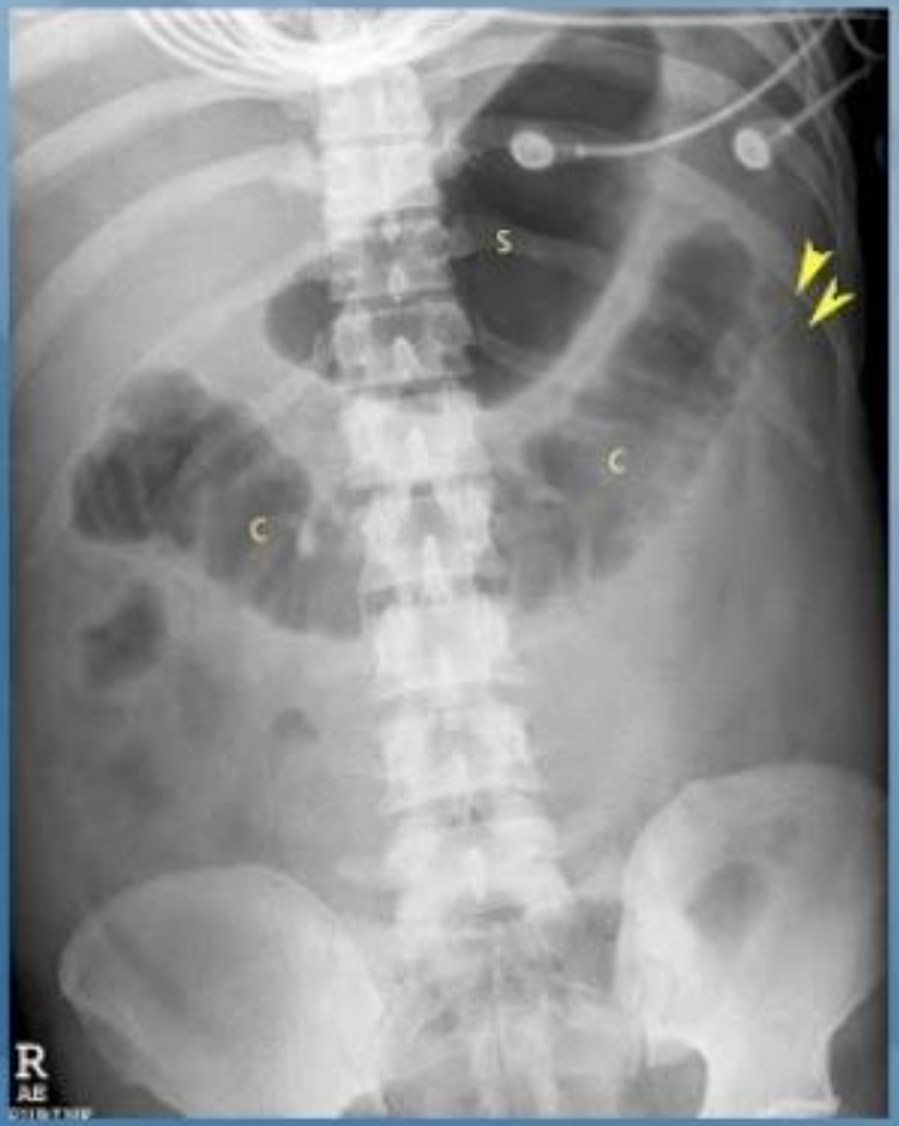
Sentinel Loop



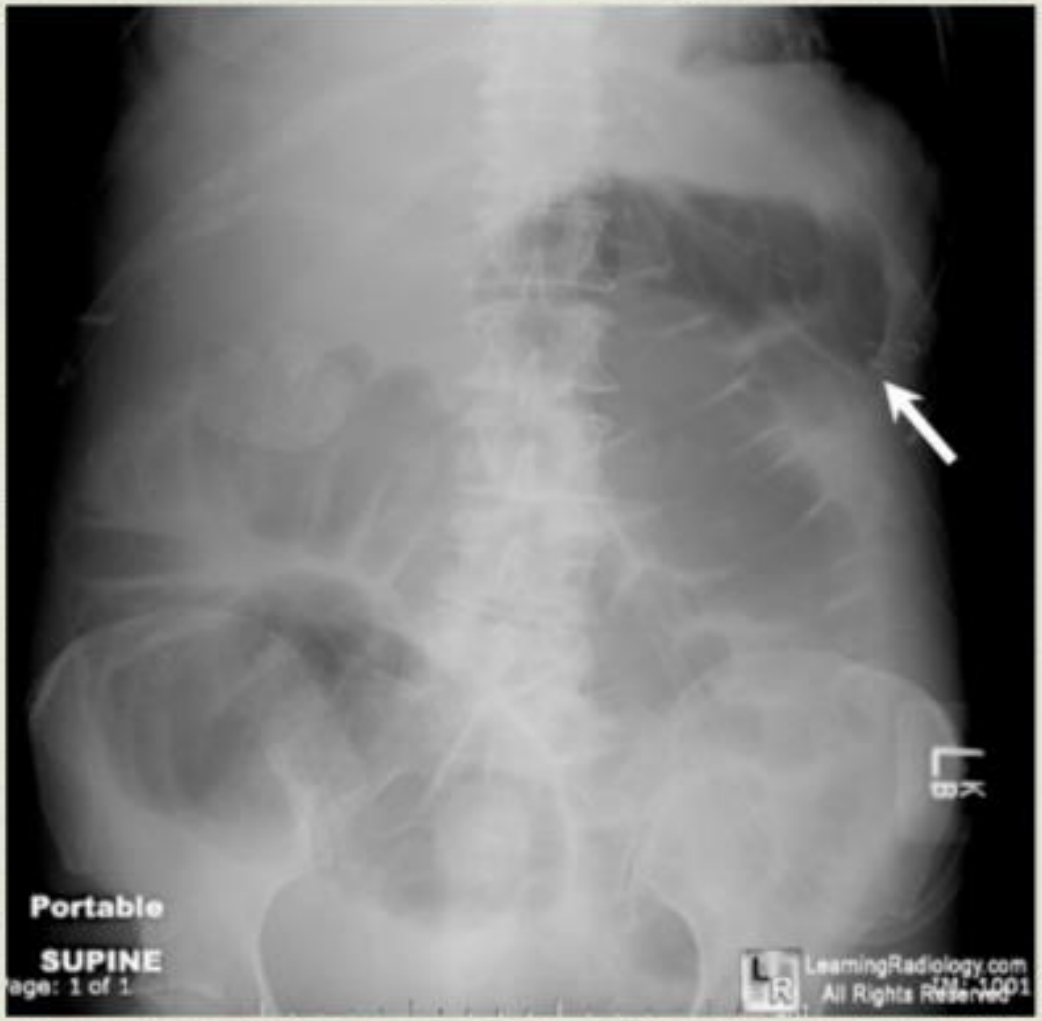


- localized ileus from nearby inflammation

Colon cutoff sign



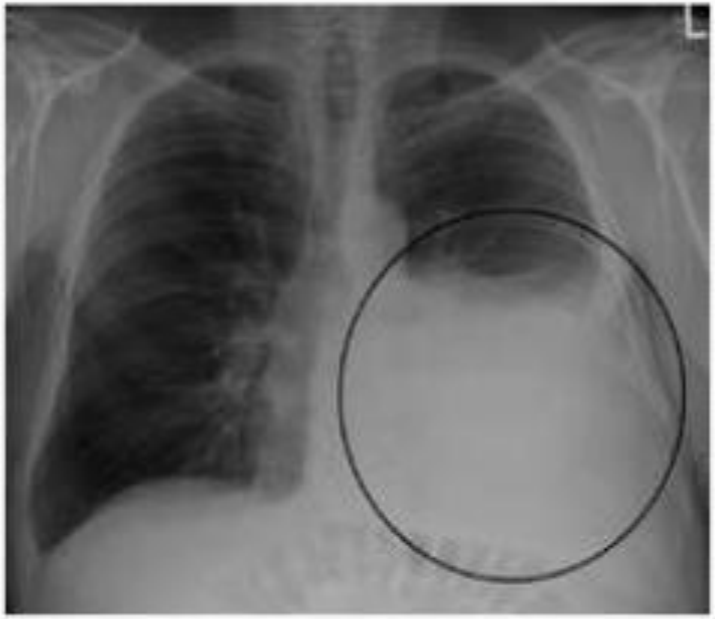
Colon cutoff sign



Radiographic signs

Chest

- Elevated diaphragm
- Pleural effusion: left sided.
- Left sided parenchymal changes



Ultrasound examination

Focal hypoechoic regions

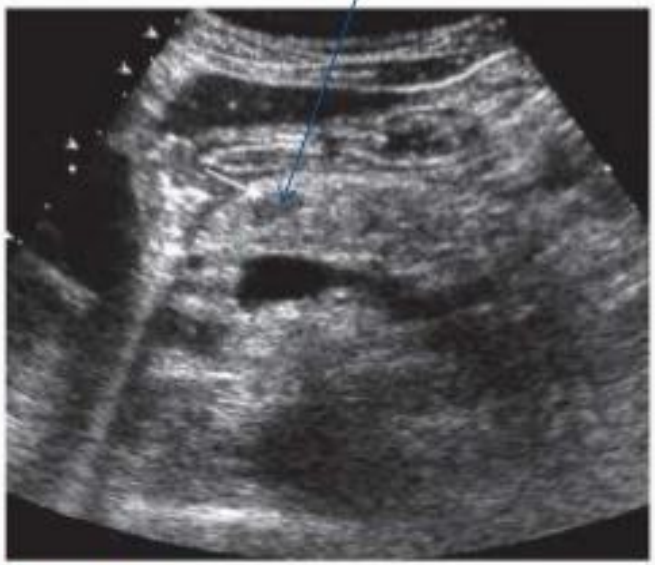


FIGURE 7-25. **Focal hypoechoic area, acute pancreatitis.** Transverse image shows heterogeneous pancreas with focal hypoechoic area *(arrow)*.

Pancreatic heterogeneity

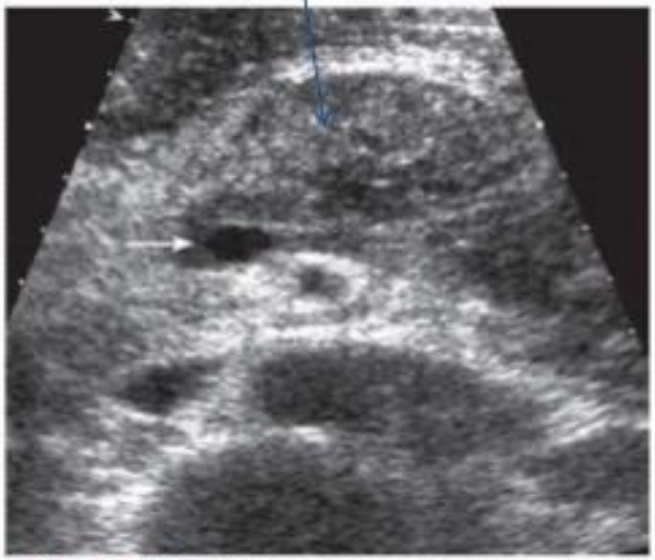


FIGURE 7-24. **Heterogeneous pancreas, acute pancreatitis.** Transverse image shows that heterogeneity can be a subtle, subjective finding. Arrow indicates the perivascular inflammation and splenic vein–superior mesenteric vein clot.

Enlargement of the pancreas

- Universal
- >22 mm (mean plus 3 standard deviations)

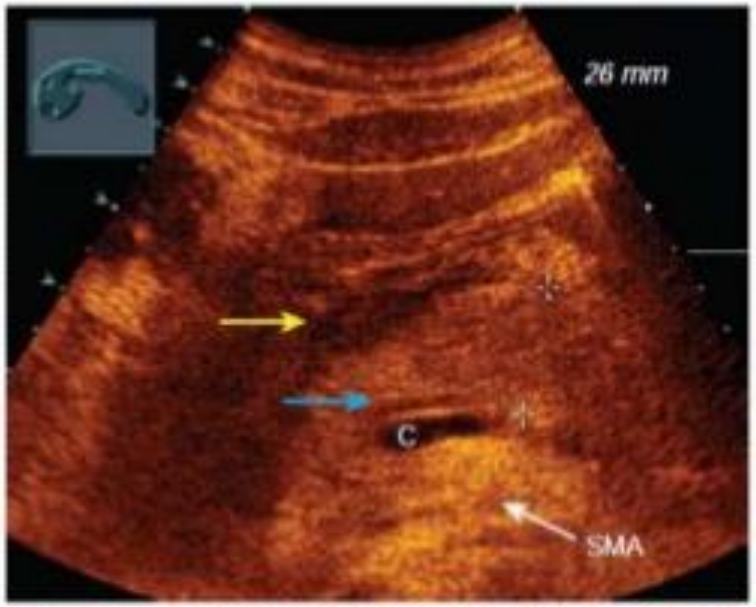
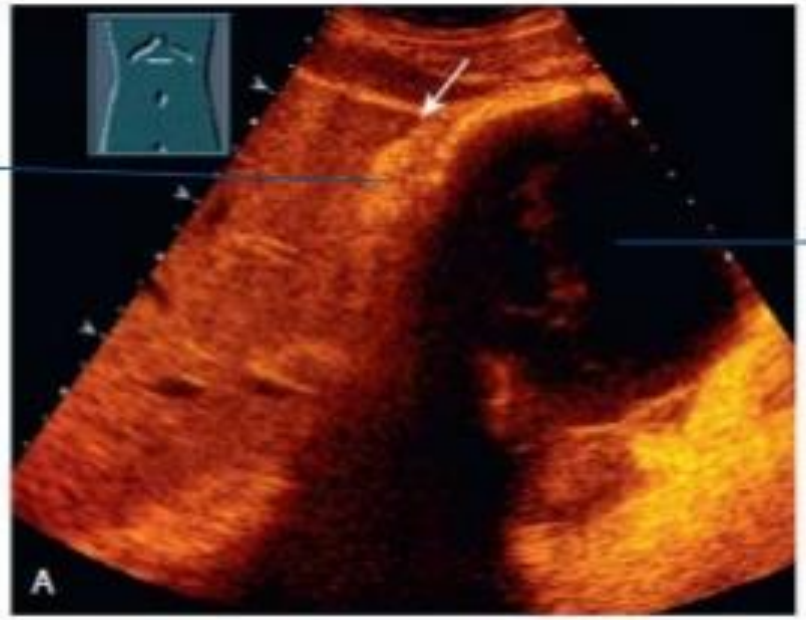


FIGURE 7-22. Enlarged pancreas, acute pancreatitis with inflammation. Transverse image of the pancreas shows 26-mm anteroposterior dimension at the level of the superior mesenteric artery *(SMA)*. Note the acute inflammation ventral to the pancreas *(yellow arrow)* and ventral to *(blue arrow)* the splenic vein–superior mesenteric vein confluence *(C)*.

acute fluid collections.

- Non encapsulated homogenous hypo echoic collections in the pancreas /retroperitoneum /abdomen
- Develop in 40% of AP half resolve spontaneously



Diagnosis – Imaging

- CT
  - Excellent pancreas imaging
  - Recommended in all patients with persisting organ failure, sepsis or deterioration in clinical status (6-10 days after admission)
  - Search for necrosis – will be present at least 4 days after onset of symptoms; if ordered too early it will underestimate severity
  - Follow-up months after presentation as clinically warranted for CT severity index of >3

When Do I Order A CT?

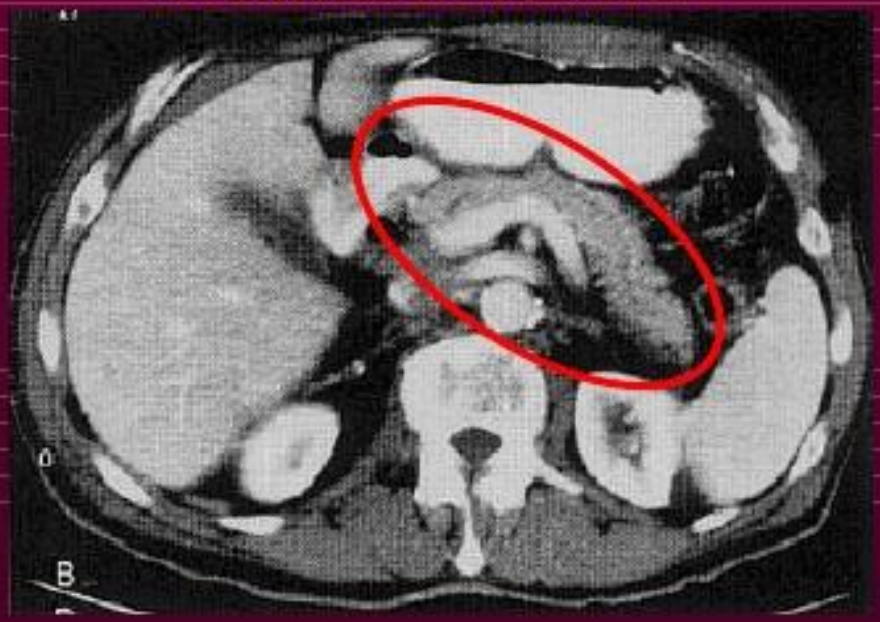
- **If the patient has…..**
  - Signs of severe acute pancreatitis
  - No signs of clinical improvement after several days
  - Diagnostic dilemma
  - Infection suspected
    - T > 101° F
    - Positive blood cultures
- **What kind of CT?**
  - Dynamic with rapid bolus IV contrast
- **What are you looking for?**
  - Necrosis: Lack of enhancement with contrast
  - Fluid Collections
  - Alternate diagnosis

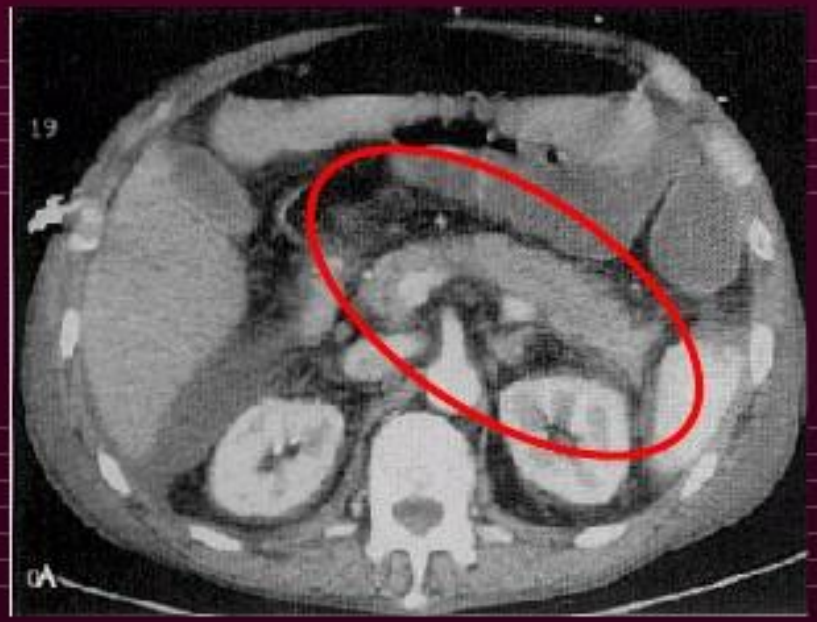
CT Findings

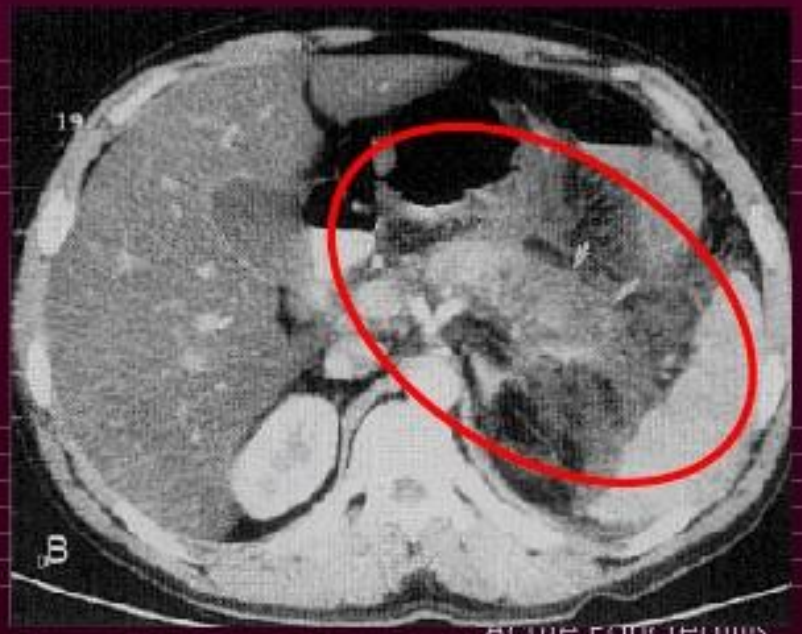
- **Pancreas**
  - Pancreatic enlargement
  - Decreased density due to edema
  - Intrapancreatic fluid collections
  - Blurring of gland margins due to inflammation
- **Peripancreatic**
  - Fluid collections and stranding densities
  - Thickening of retroperitoneal fat

*** It may take up to 72h for inflammatory changes to become apparent on CT ***

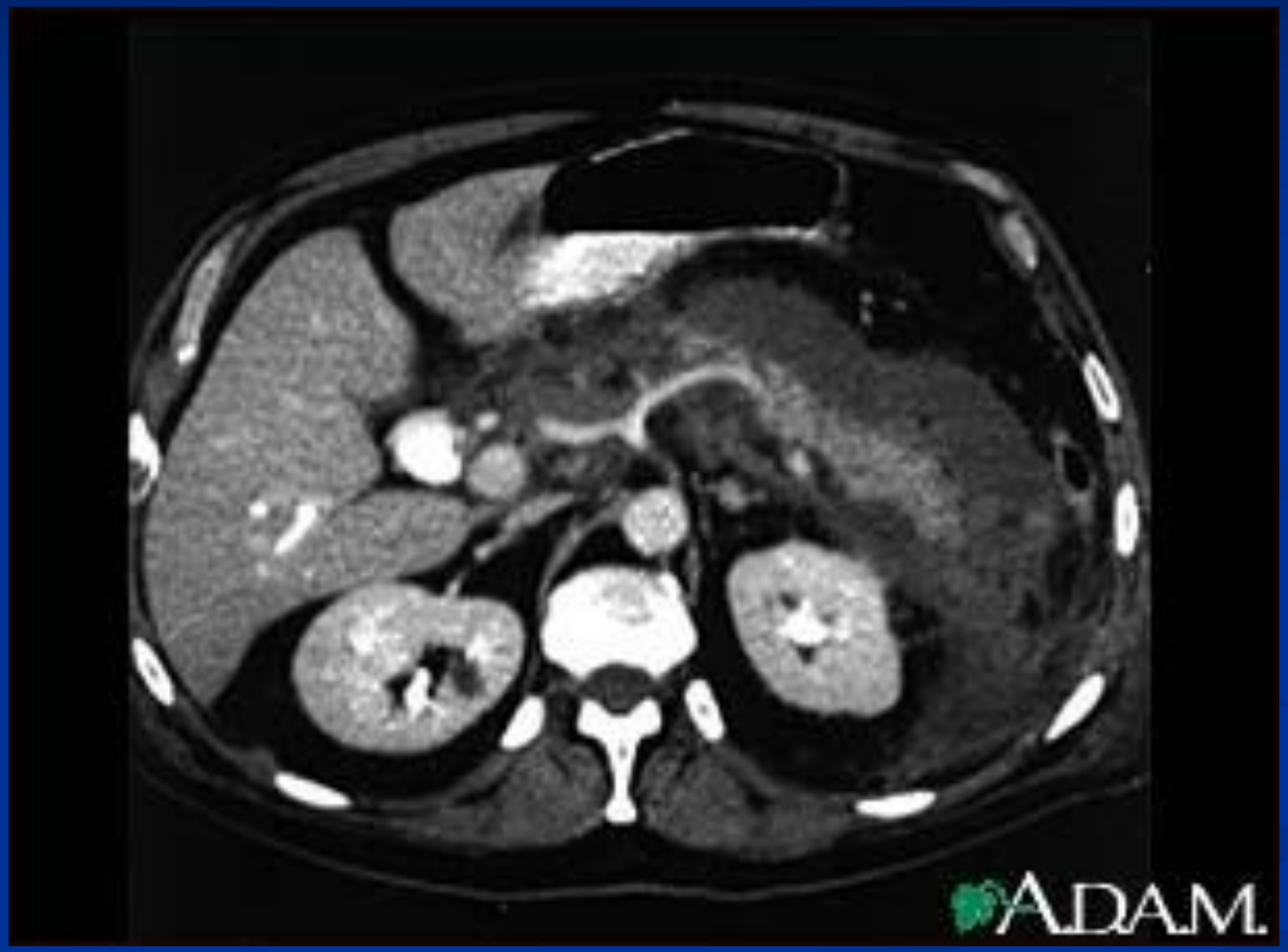
Normal Pancreas



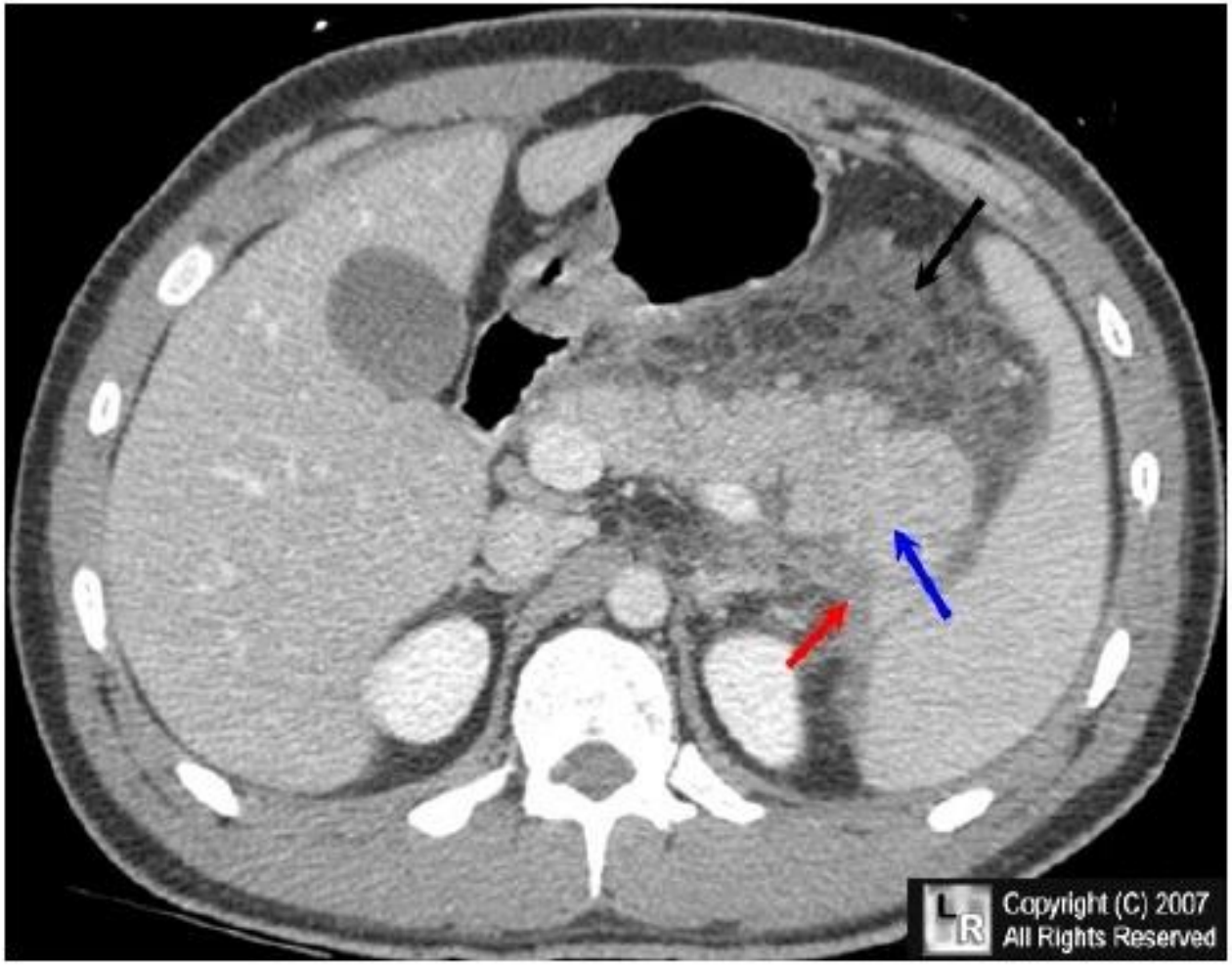




CT Scan of acute pancreatitis

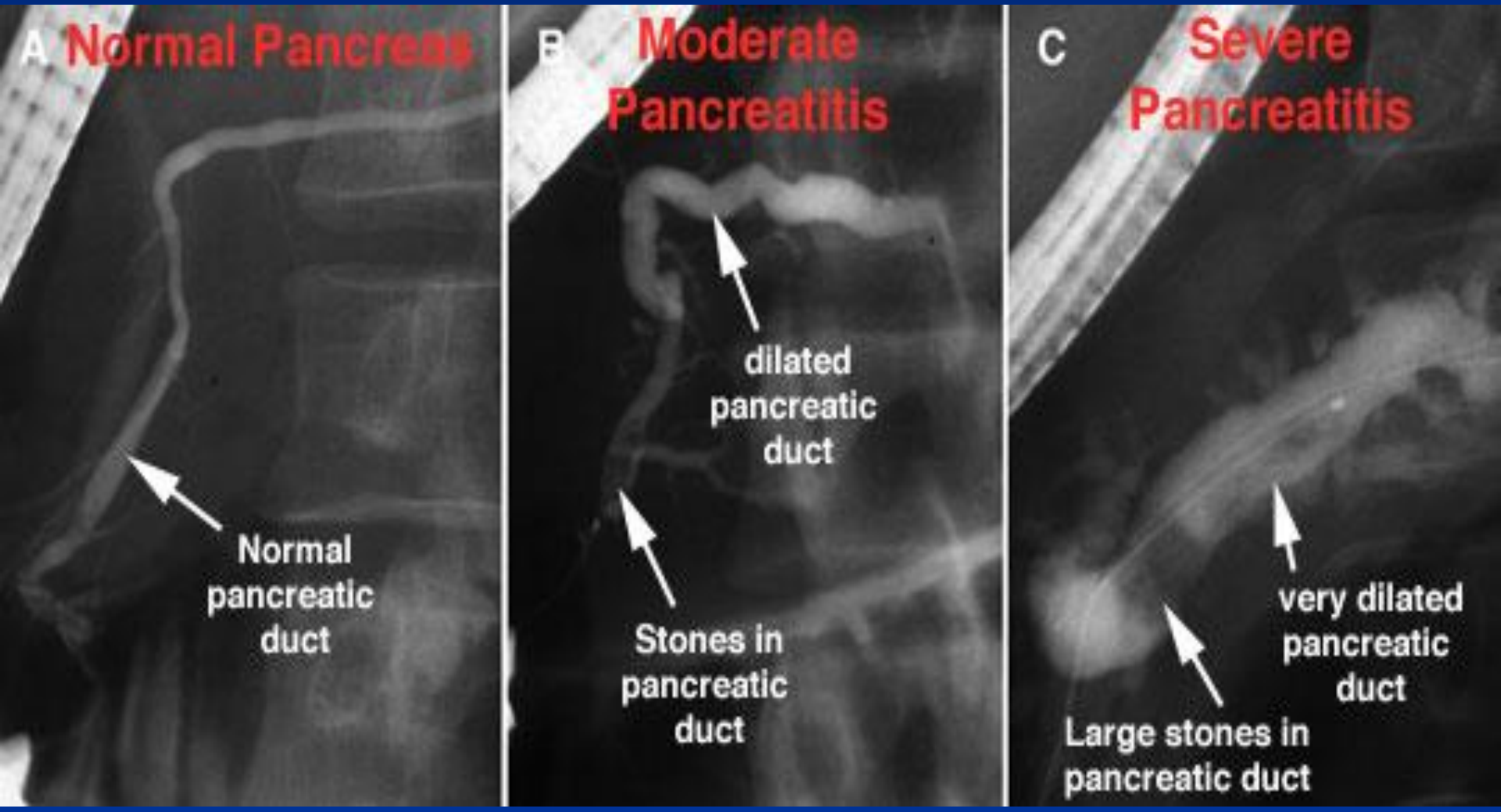


CT shows significant swelling and inflammation of the pancreas



Acute pancreatitis. The pancreas is enlarged (blue arrow) with indistinct and shaggy margins.
There is peripancreatic fluid (red arrow) and extensive peripancreatic infiltration of the surrounding fat (black arrow).

Gall stone pancreatitis by ERCP



Prognosis

- Many different scoring systems
  - Ranson (most popular & always taught in med-school)
    - No association found with score, and mortality or length of hospitalization
  - APACHE II
  - CT severity Index
    - Recent studies show this to be most predictive of adverse outcomes
      - CT score > 5 associated with 15x mortality rate
      - Problem is 1 CT study showing this was conducted 72 hours after admission (Ranson/Apache are 24 & 48 hours)
  - Imrie Score
- Atlanta Classification used to help compare various scores (clinical research trials)

Ranson Criteria

- Admission
  - Age > 55
  - WBC > 16,000
  - Glucose > 200
  - LDH > 350
  - AST > 250

During first 48 hours
- Hematocrit drop > 10%
- Serum calcium < 8
- Base deficit > 4.0
- Increase in BUN > 5
- Fluid sequestration > 6L
- Arterial PO2 < 60

5% mortality risk with <2 signs
15-20% mortality risk with 3-4 signs
40% mortality risk with 5-6 signs
99% mortality risk with >7 signs

Ransons Criteria

- Presence of 1-3 criteria represents mild pancreatitis,
- with minimal mortality rate.
- Ranson's score of 3-5 has 10-20% of mortality rate.
- Score higher than 5 has mortality rate of > 50%
- associated with more systemic complications

APACHE II score

(Acute Physiology And Chronic Health Evaluation)

Score 0 to 2 : 2% mortality Score 3 to 4 : 15% mortality

Score 5 to 6 : 40% mortality Score 7 to 8 : 100% mortality

- Hemorrhagic peritoneal fluid
- Obesity
- Indicators of organ failure
- Hypotension (SBP <90 mmHG) or tachycardia > 130 beat/min
- PO_2 <60 mmHg
- Oliguria (<50 mL/h) or increasing BUN and creatinine
- Serum calcium < 1.90 mmol/L (<8.0 mg/dL)
- serum albumin <33 g/L (<3.2.g/dL)>

Balthazar scoring

CT Severity Index

Balthazar Grade

Balthazar Grade	Appearance on CT	CT Grade Points
• Grade A	Normal CT	**0 points**
• Grade B	Focal or diffuse enlargement of the pancreas	**1 point**
• Grade C	Pancreatic gland abnormalities and peripancreatic inflammation	**2points**
• Grade D	Fluid collection in a single location	**3 points**
• Grade E	Two or more fluid collections and / or gas bubbles in or adjacent to pancreas **4points**	

Necrosis Score

Necrosis Percentage	Points
• No necrosis	**0 points**
• 0 to 30% necrosis	**2 points**
• 30 to 50% necrosis	**4 points**
• Over 50% necrosis	**6 points**

TOTAL SCORE = CT grade + Necrosis

0-1 points = 0% mortality
2-3 points = 3% mortality
4-6 points = 6% mortality
7-10 points = 17% mortality

The numerical CTSI (Computed Tomography Severity Index) has a maximum of **ten** points, it is the sum of the Balthazar grade points and pancreatic necrosis grade points

Mild (0-2 points), moderate (4-6 points), or severe (8-10 points).

Principles for managing patients with acute pancreatitis

- Fasting of the patient Nasogastric suction/NPO – pancreatic rest.
- Pain management using narcotic agents.
- Supportive care with close attention to volume status and electrolyte balance
- Adequate nutritional support

- Prophylactic broad-spectrum antibiotics for patients with predicted severe pancreatitis
- Early detection of complications
- Identification of patients who may benefit from ERCP (when severe pancreatitis is complicated by progressive jaundice or cholangitis)

Antibiotic therapy

- Early trials in 1970's did not show the benefit of antibiotics
- Antibiotics that did not penetrated the pancreatic tissue
- Prophylactic antibacterial treatment is **strongly recommended in severe pancreatitis**

Treatment :

- In most patients (85–90%) with acute pancreatitis, the disease is self-limited and subsides spontaneously, usually within 3–7 days after treatment is instituted Conventional measures include analgesics for pain 50 to 100 mg of meperidine (Demerol) parenterally every 3 hours

Indications for surgical intervention

- No universally valid answer

- Persistence of organ failure and/or systemic inflammatory signs after 72 h of maximal supporting intensive care therapy is an indication for operative treatment.

The timing of pancreatic debridement

- Controversial issue
- Demarcation of pancreatic necrosis (2-3 w) is a precondition for sufficient debridement
- Necrosectomy, performed later than three weeks after the onset of disease higher rate of successful debridement of pancreatic necrosis

Cholecystectomy??

- should be performed after recovery in all patient with gallstone pancreatitis

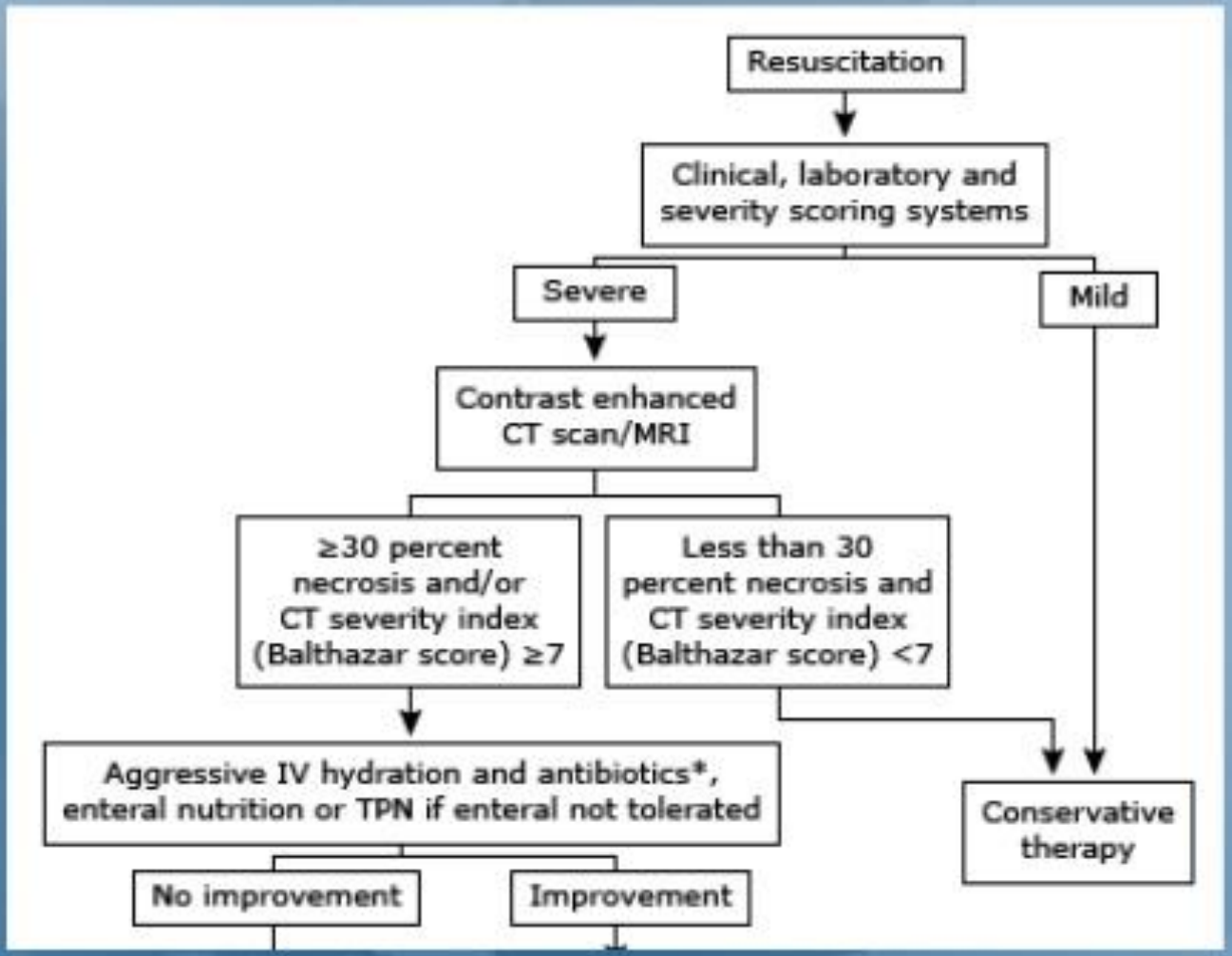
- Failure to perform a cholecystectomy is associated with a 25-30% risk of recurrent acute pancreatitis, cholecystitis, or cholangitis within 6-18 weeks

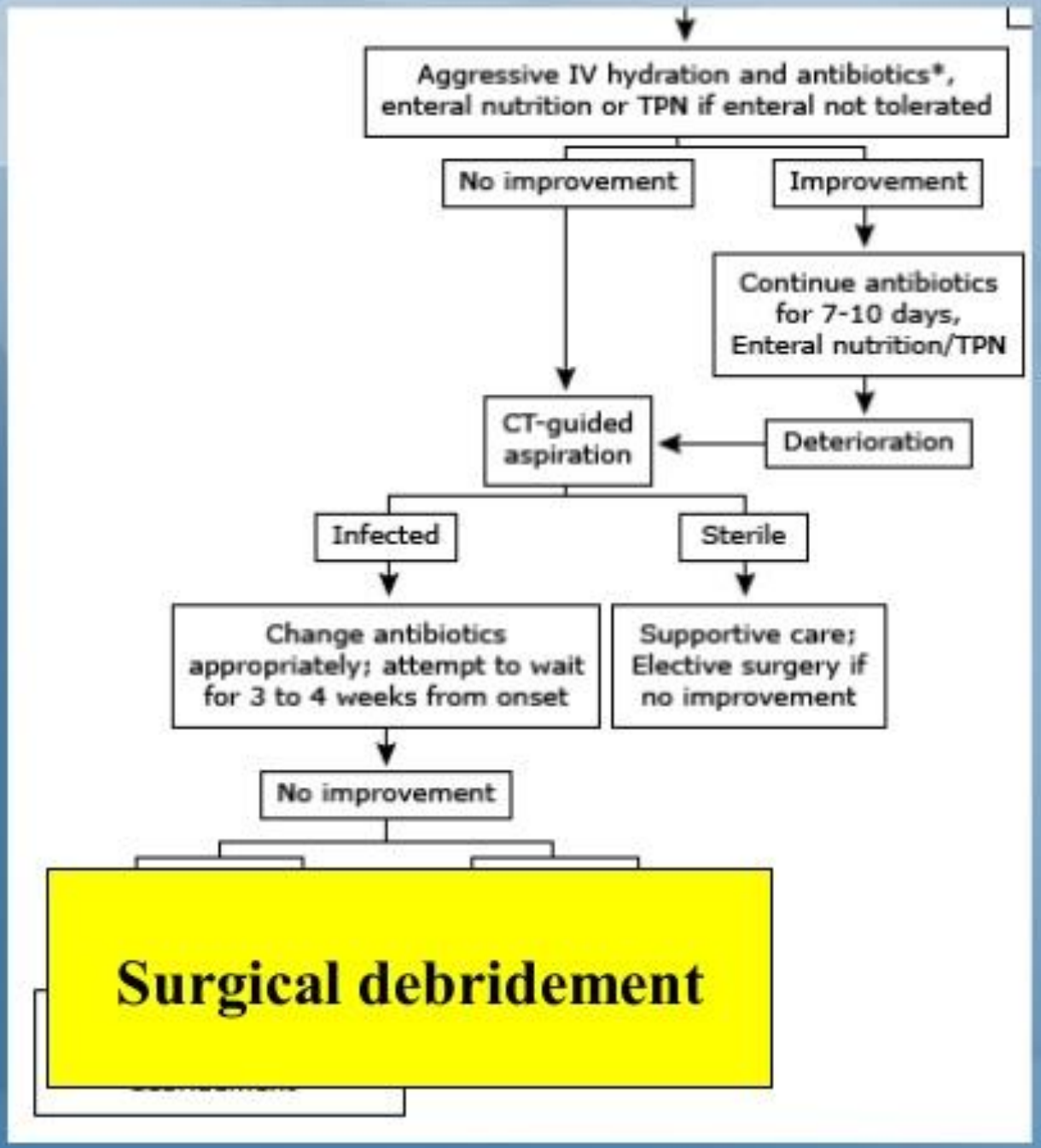
Cholecystectomy

- In mild pancreatitis case, an usually be performed safely within 7 days after recovery
- In severe pancreatitis case ,delaying for at least 3 wks may be reasonable

- If high suspicion of CBD stones, preoperative ERCP is the best test that therapeutic intervention will be required
- If low suspicion,intraoperative cholangiogram during cholecystectomy may be preferable to avoid the morbidity associated with ERCP

AGA Guideline




Aggressive IV hydration and antibiotics*, enteral nutrition or TPN if enteral not tolerated
No improvement
Improvement
Continue antibiotics for 7-10 days, Enteral nutrition/TPN
Deterioration
CT-guided aspiration
Infected
Sterile
Change antibiotics appropriately; attempt to wait for 3 to 4 weeks from onset
Supportive care; Elective surgery if no improvement
No improvement
Surgical debridement

Indication for pancreatic debridement

- Infected pancreatic necrosis
- Symptomatic sterile pancreatic necrosis
  - chronic low grade fever
  - Nausea
  - Lethargy
  - Inability to eat
  - * Fail medical treatment

Surgical approach

- **Open debridement with external drainage**
  – gold standard
- **Open debridement with internal drainage and cystgastrostomy**
  - only appropriate for patients with WON
- **Open packing**
  — Open packing with planned reoperation every 48-72 hrs until the necrosis is adequately removed
- **Laparoscopic debridement**

  -Video-assisted retroperitoneal debridement

  -Laparoscopically-assisted transperitoneal debridement

Surgical Intervention

- Mortality rates of up to 65 % have been described with early surgery in severe pancreatitis
- Prospective and randomized clinical trial comparing early (within 48 to 72 hr of symptoms) versus late (at least 12 days after onset) debridement in patients with severe pancreatitis, mortality rates were 56 %and 27 %
- Except patient with severe complications such as massive bleeding or bowel perforation, early surgery must be performed

Mild Acute Pancreatitis

Management

- Mild AP (Ranson score 2) = 85% patients
- Most cases will resolve in several days
- NPO, IV fluids, Analgesia
- Advance diet as tolerated
- Gallstone pancreatitis = cholecystectomy
- Failure to improve, status worsens
- .CT abdomen -

Severe ACUTE PANCREATITS

Management

- Severe AP (Ranson score 3) 15% patients
- ICU with close monitoring, strict VS charting
- NPO, NGT?, aggressive IV fluid replacement, analgesia
- Early ERCP + sphincterotomy if due to gallstone disease
- Pancreatic necrosis on CT = start IV antibiotics
- Maintain nutrition
- Enteral nutritional support
- NJ tube within 3-4 days

Complications

- **Early Complications :**
- Shock
- ARDS
- Renal failure
- DIC – depletion of coagulation factor
- Hypocalcemia
- Hypo/hyperglycemia

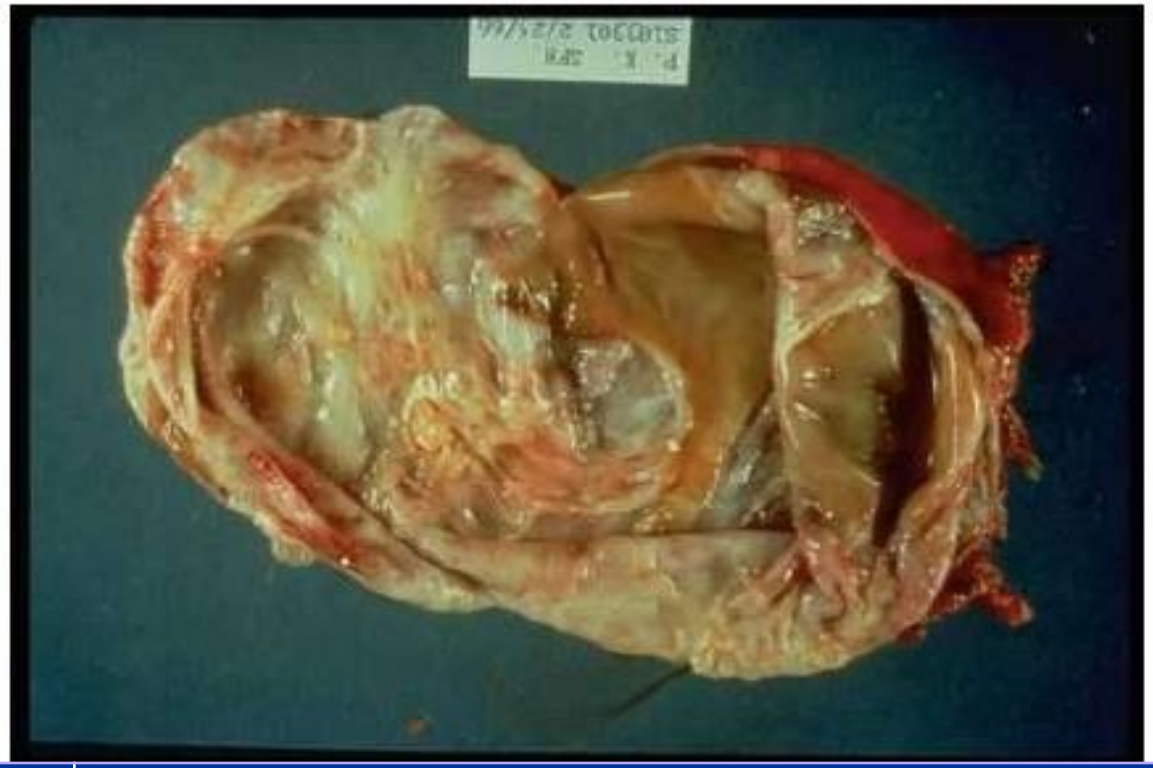
Late complications:

- Pseudocyst – (liquified debris with rim of pancreatic or other tissue (no epithelial lining) after 3 weeks of acute pancreatitis
  - Infection
  - Bleeding
  - Rupture
- Abscess – requires drainage (percutaneous or surgical).
- Pancreatic necrosis.
- Bleeding – elastase.
- Thrombosis of splenic, gastrodoudenal artery.

Complications

- Necrotizing pancreatitis
  - Significantly increases morbidity & mortality
  - Usually found on CT with IV contrast
- Pseudocysts
  - Suggested by persistent pain or continued high amylase levels (may be present for 4-6 wks afterward)
  - Cyst may become infected, rupture, hemorrhage or obstruct adjacent structures
    - Asymptomatic, non-enlarging pseudocysts can be watched and followed with imaging
    - Symptomatic, rapidly enlarging or complicated pseudocysts need to be decompressed

Pseudocyst of pancreas



Acute pseudocyst	Collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue, which arises as a result of acute pancreatitis, pancreatic trauma or chronic pancreatitis, occurring at least 4 weeks after onset of symptoms, is round or ovoid and most often sterile; when pus is present, lesion is termed a pancreatic abscess

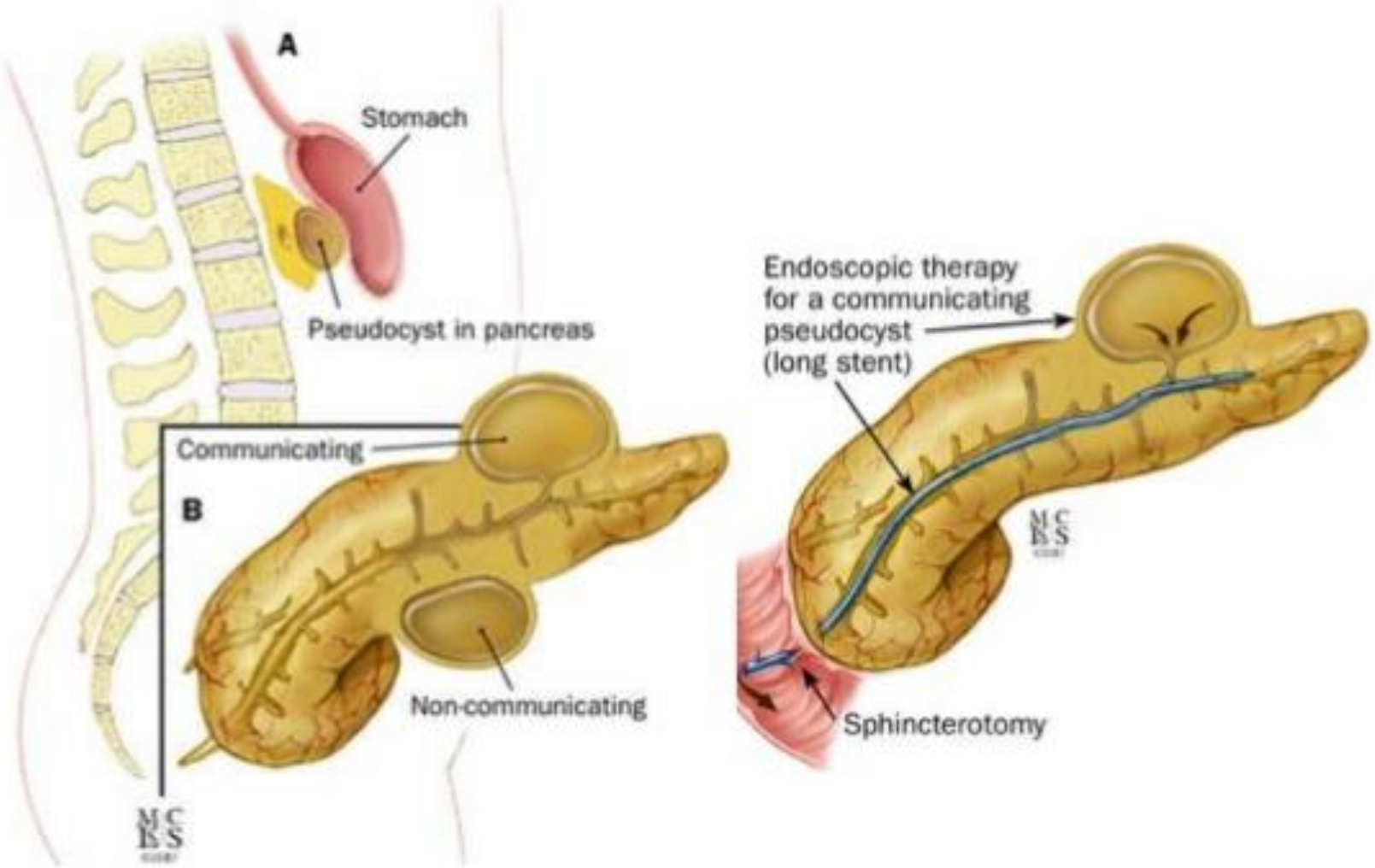
Complications of Pseudocyst

- Infection - 14%
- Rupture - 6.8%
- Hemorrhage - 6.5%
- Common bile duct obstruction - 6.3%
- GI obstruction - 2.6%

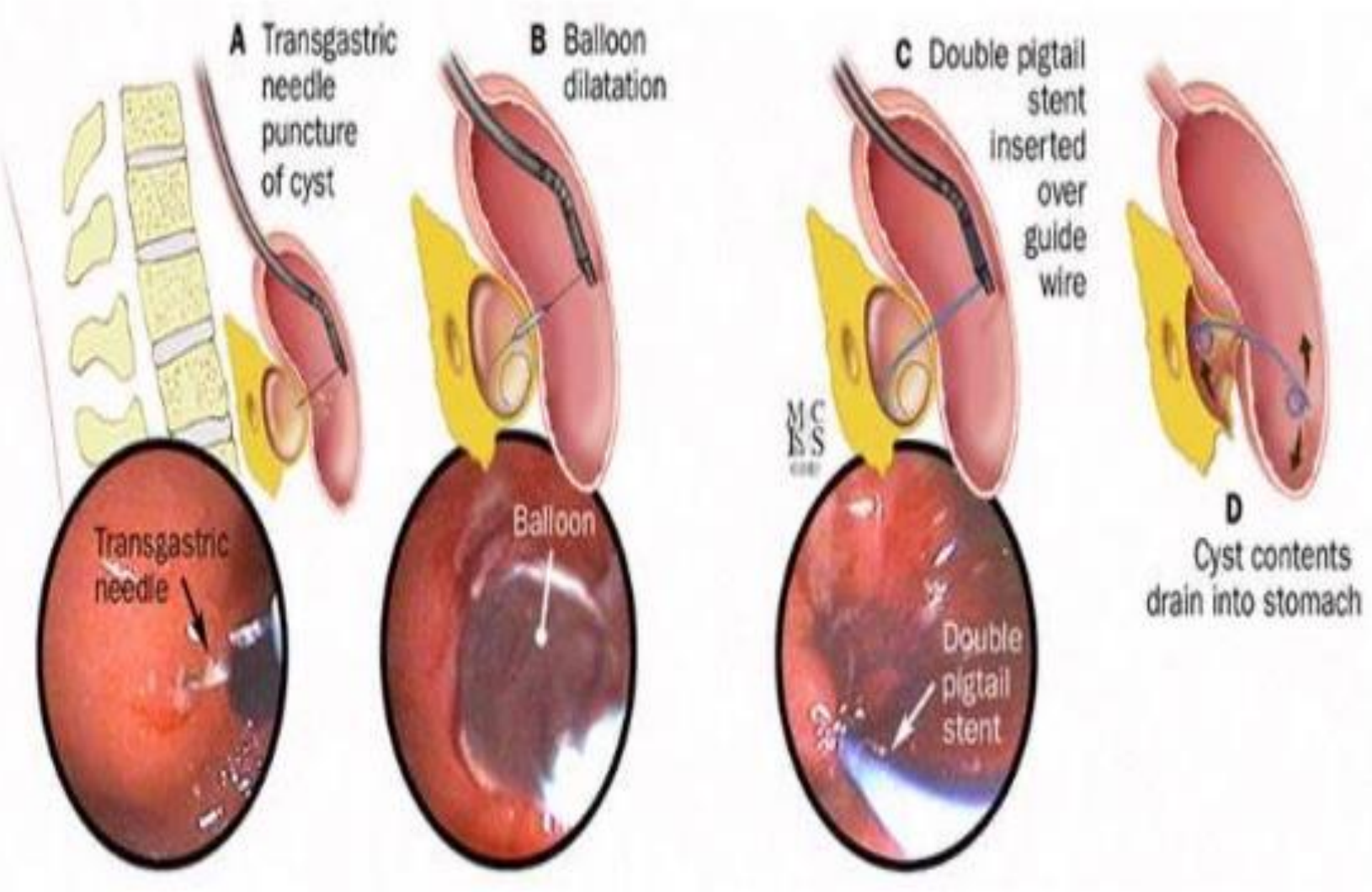
Pseudocyst Management

- Old thought
  - Pseudocysts > 5 cm that have been present > 6 weeks must be drained
- Current practice
  - Asymptomatic pseudocysts, regardless of size, do not require treatment

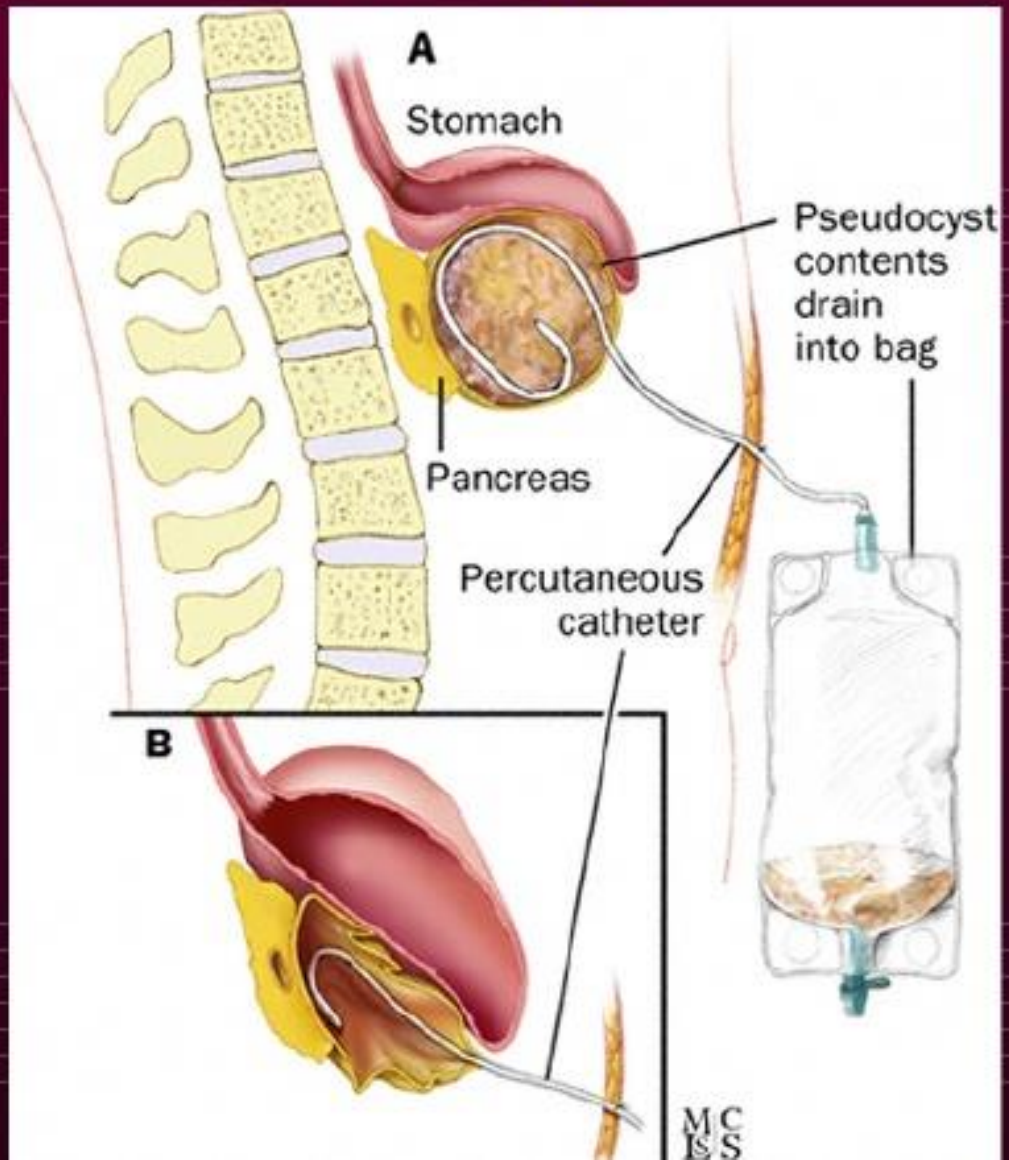
Communicating Non-communicating



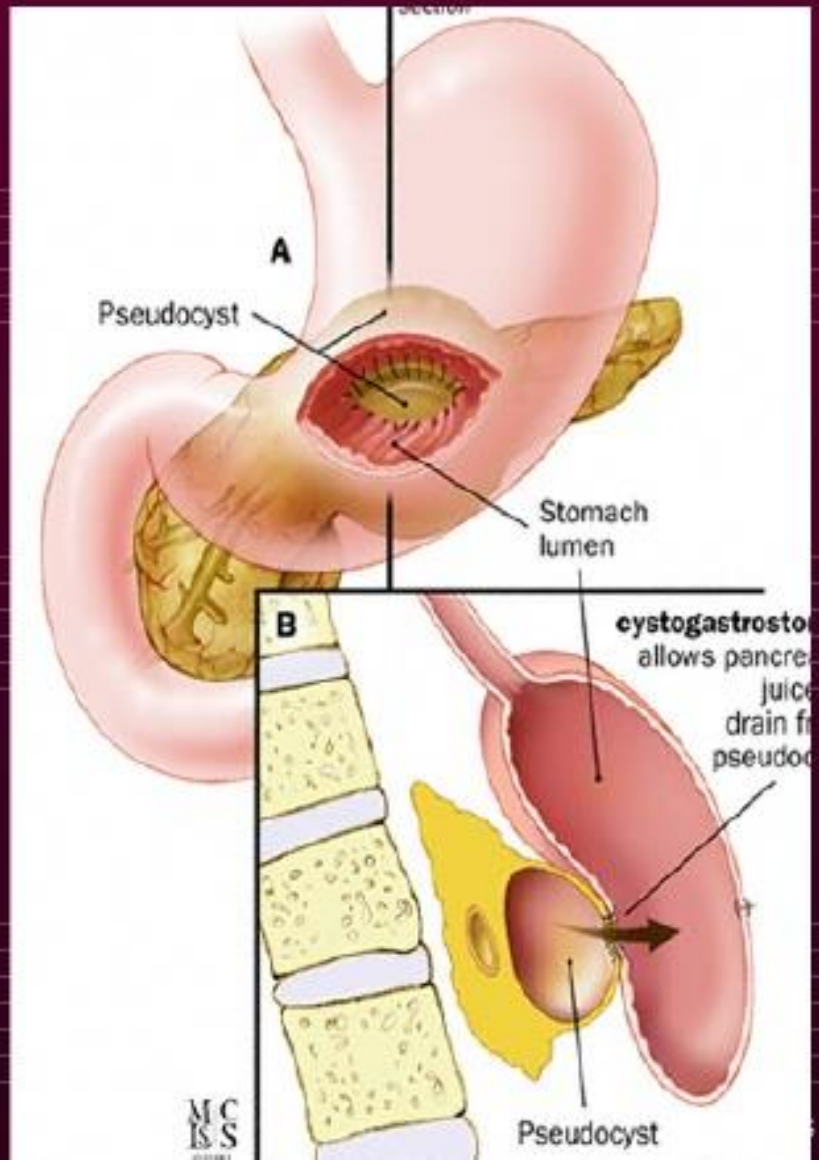
Endoscopic Pseudocyst Management



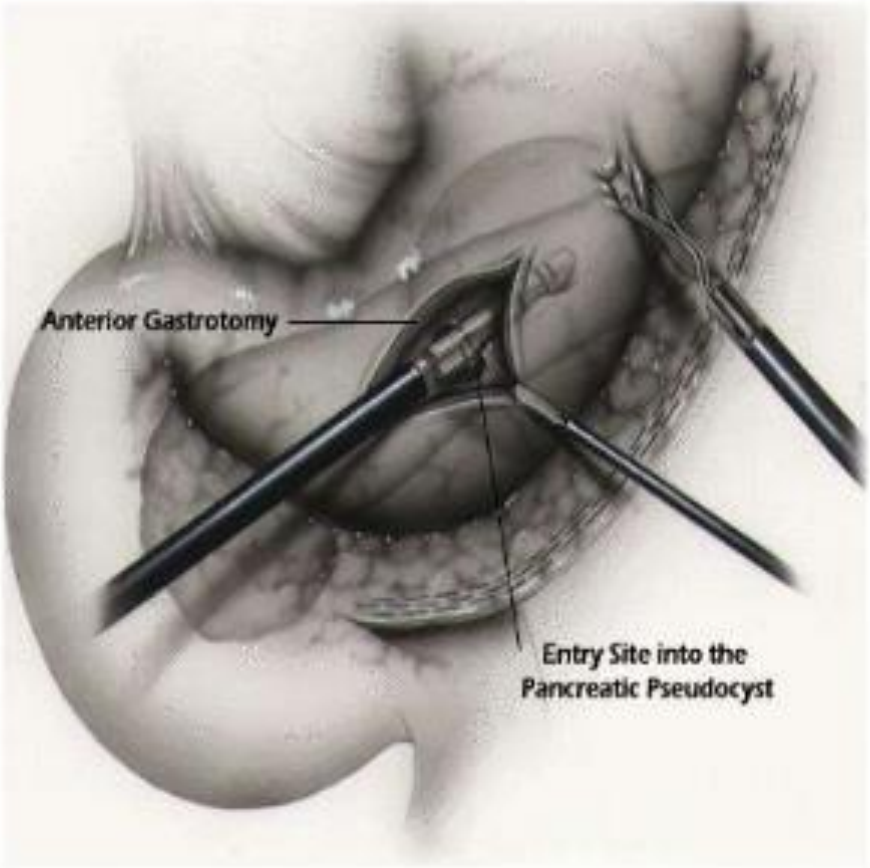
Percutaneous Pseudocyst Drainage

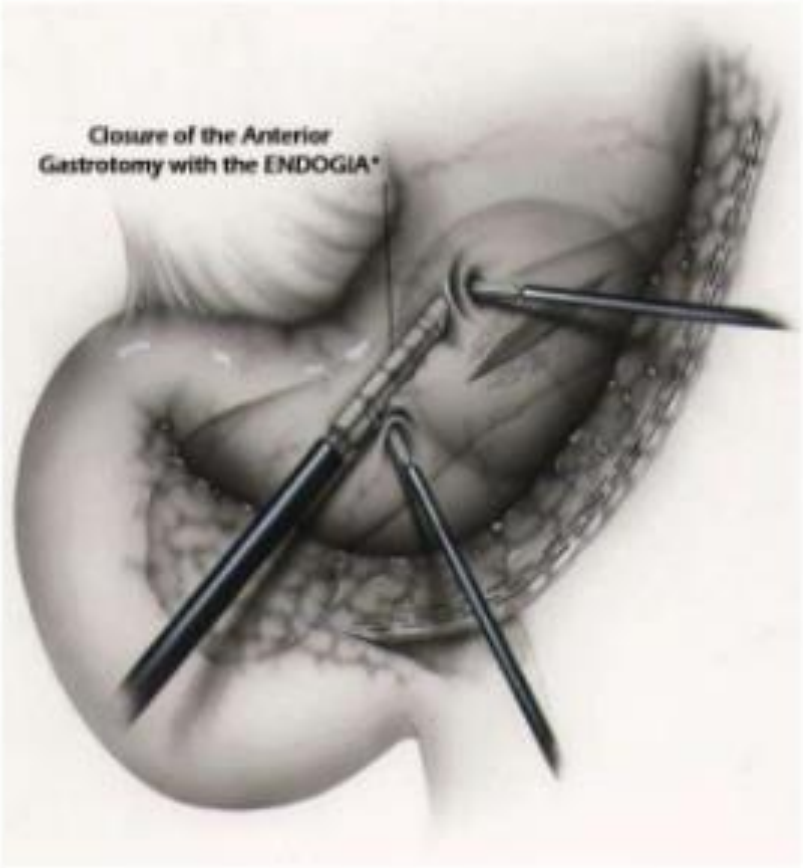


Open Cystgastrostomy



Laparoscopic Cyst Gastrostomy





Pancreatic abscess

Circumscribed, intra-abdominal collection of pus, usually in proximity to the pancreas, containing little or no pancreatic necrosis, which arises as a consequence of acute pancreatitis or pancreatic trauma

Often 4 weeks or more after onset

Pancreatic abscess and infected pancreatic necrosis differ in clinical expression and extent of associated necrosis

Complications continued #2

- Infection
  - Many areas for concern: abscess, pancreatic necrosis, infected pseudocyst, cholangitis, and aspiration pneumonia -> SEPSIS may occur
  - If concerned, obtain cultures and start broad-spectrum antimicrobials (appropriate for bowel flora)
  - In the absence of fever or other clinical evidence for infection, prophylactic antibiotics is not indicated
- Renal failure
  - Severe intravascular volume depletion or acute tubular necrosis may lead to ARF

Complications continued #3

- Pulmonary
  - Atelectasis, pleural effusion, pneumonia and ARDS can develop in severe cases
- Other
  - Metabolic disturbances
    - hypocalcemia, hypomagnesemia, hyperglycemia
  - GI bleeds
    - Stress gastritis
  - Fistula formation

Prognosis

- 85-90% mild, self-limited, recover within 2 weeks
  - Usually resolves in 3-7 days
- 10-15% severe requiring ICU admission
  - Mortality may approach 50% in severe cases
- Respiratory / Renal compromise are poor prognostic signs
- Infection is late complication

Famous people who have had pancreatitis

- Alexander the Great
- Ludwig von Beethoven
- Dizzie Gillespie
- Maximilian Schell
- Matthew Perry
- John Ashcroft







pavarotti
pavarotti
in hyde park
another tibor rudas production
DECCA
DIGITAL