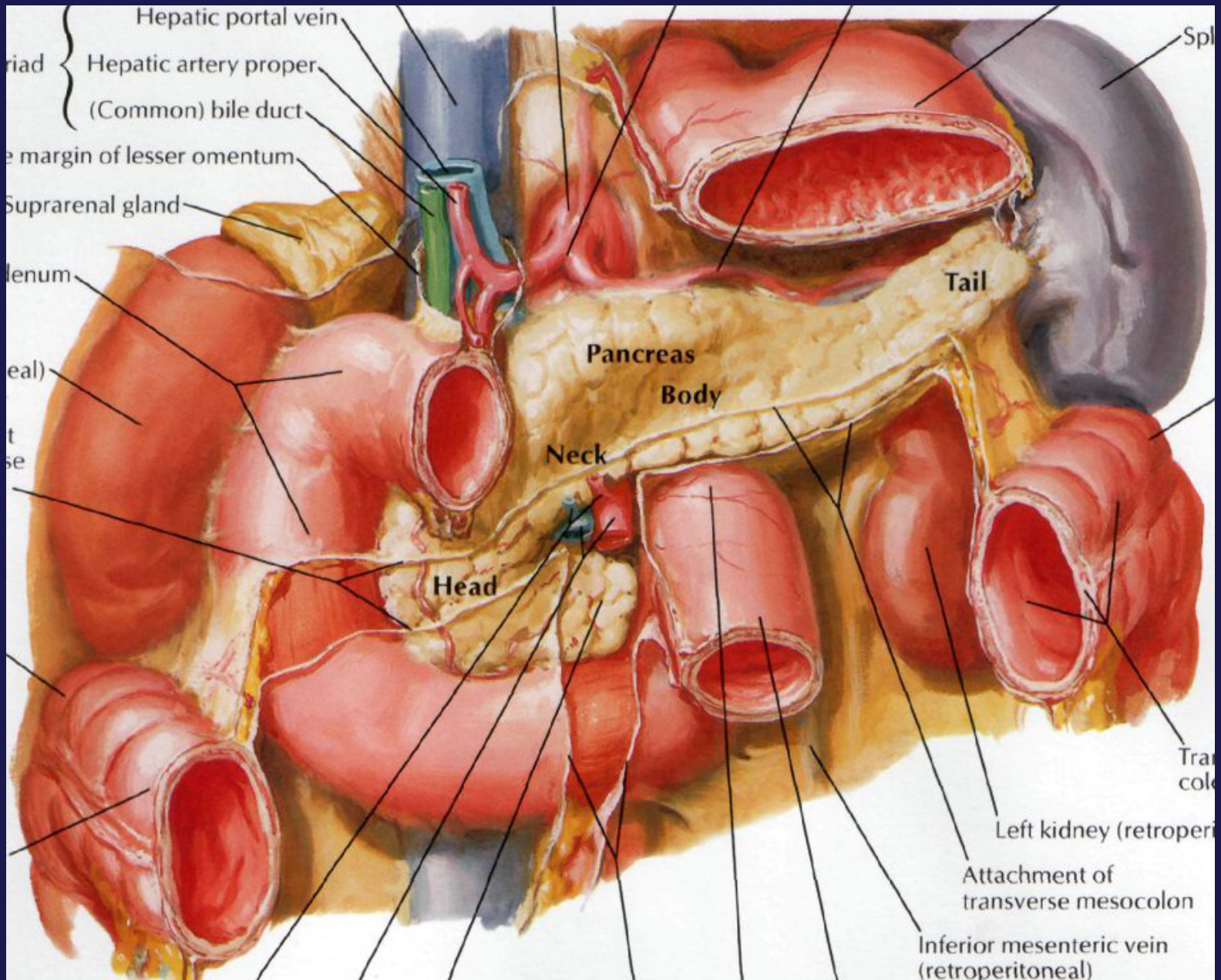
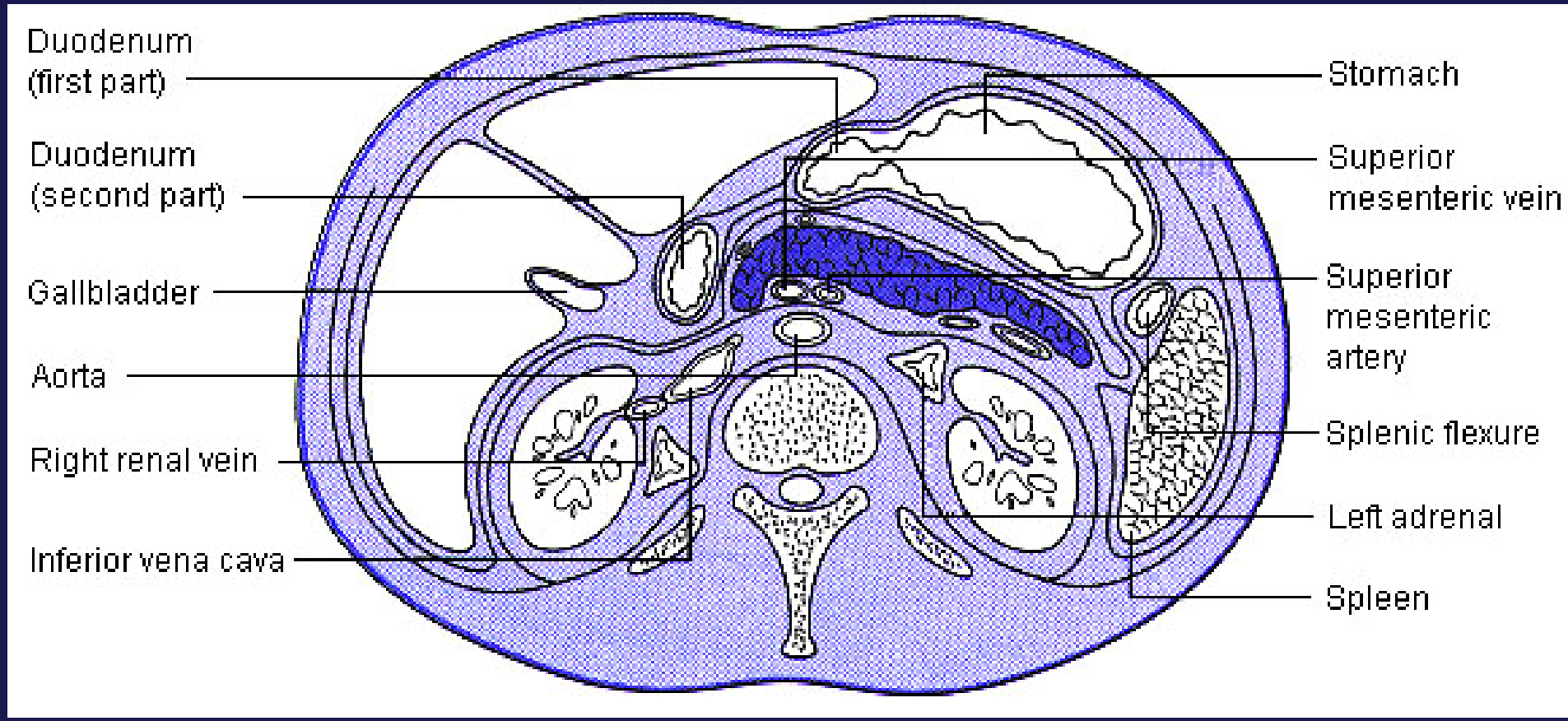
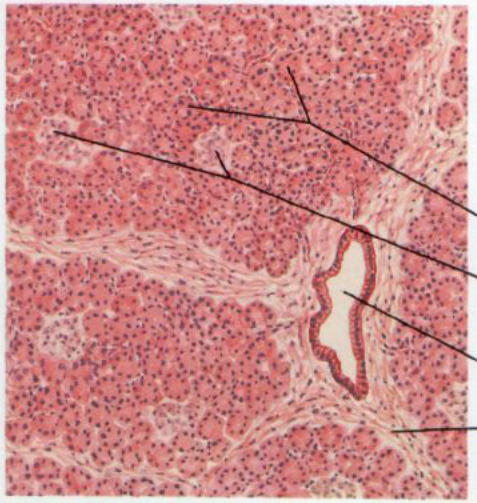
Acute Pancreatitis

Ghidirim Gh., Mishin I., Vozian M., Zastavnitsky Gh.


Hepatic portal vein
Hepatic artery proper
(Common) bile duct
margin of lesser omentum
Suprarenal gland
Tail
Pancreas
Body
Neck
Head
Left kidney (retroperi
Attachment of
transverse mesocolon
Inferior mesenteric vein
(retroperitoneal)


Duodenum
(first part)
Duodenum
(second part)
Gallbladder
Aorta
Right renal vein
Inferior vena cava
Stomach
Superior
mesenteric vein
Superior
mesenteric
artery
Splenic flexure
Left adrenal
Spleen



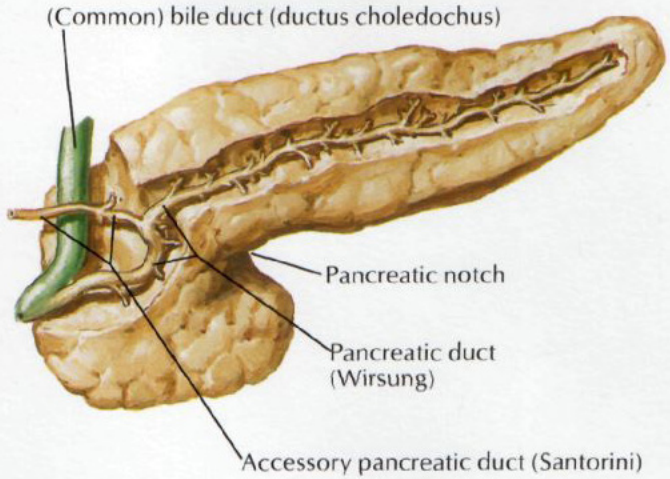
Low-power section of pancreas

Acini

Islets (Langerhans)

Interlobular duct

Interlobular septum

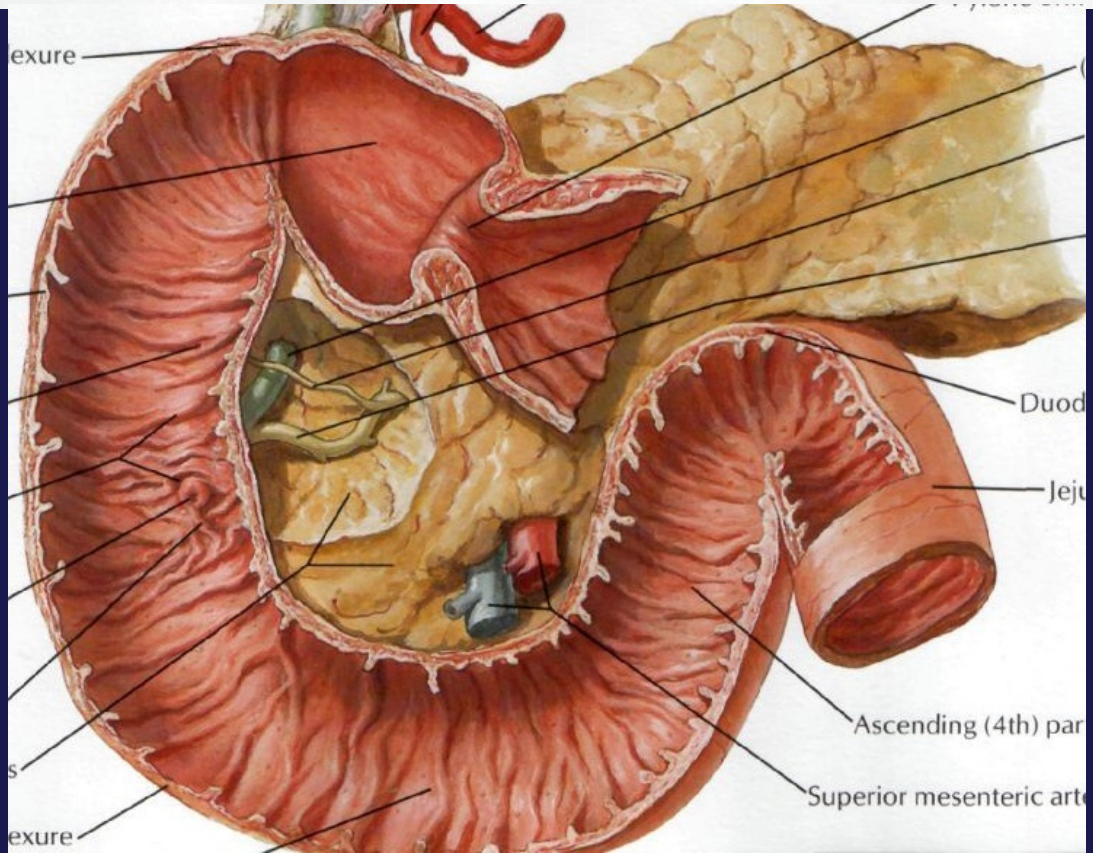


(Common) bile duct (ductus choledochus)

Pancreatic notch

Pancreatic duct (Wirsung)

Accessory pancreatic duct (Santorini)



exure

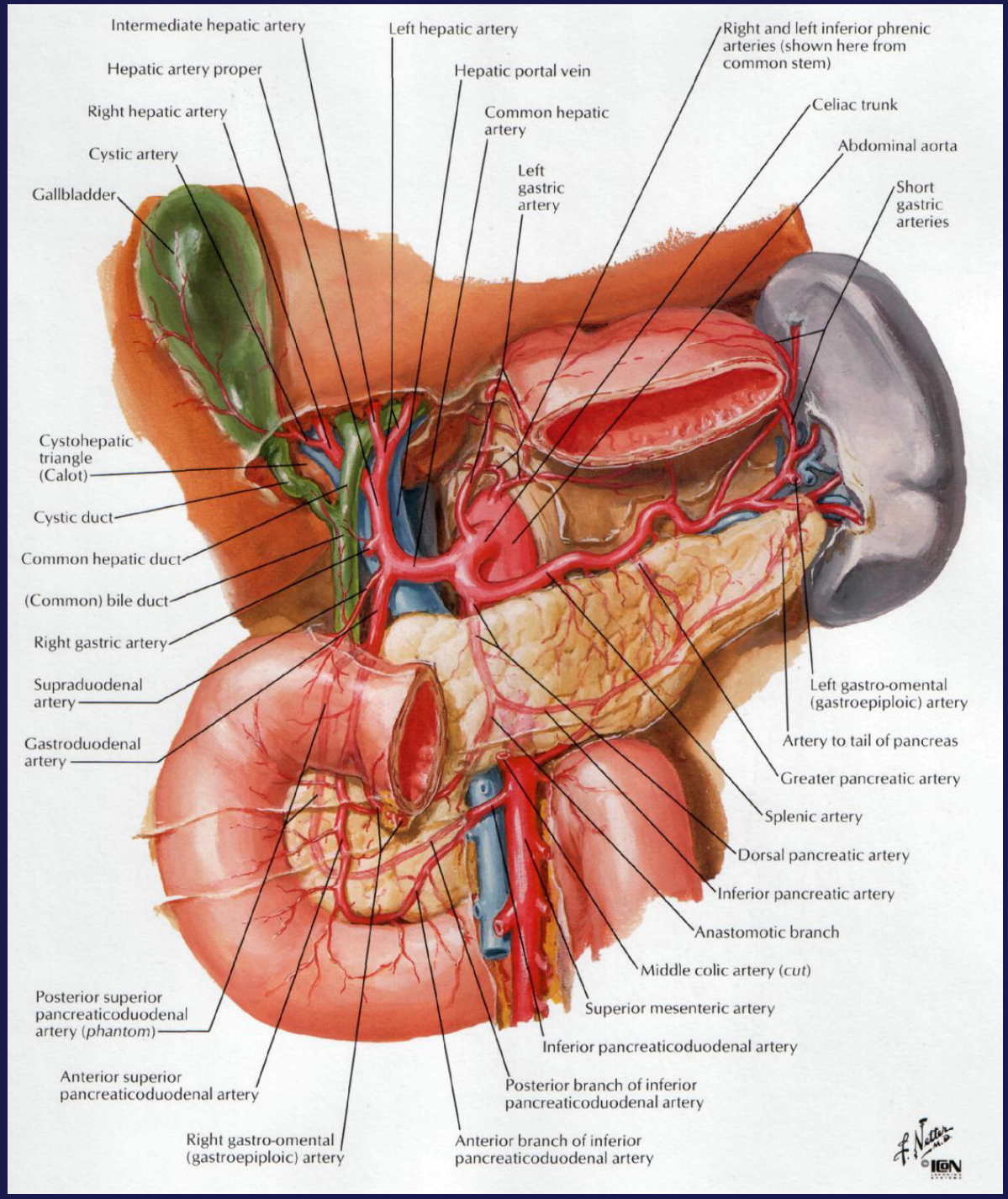
Duod

Jeju

Ascending (4th) par

Superior mesenteric art

exure

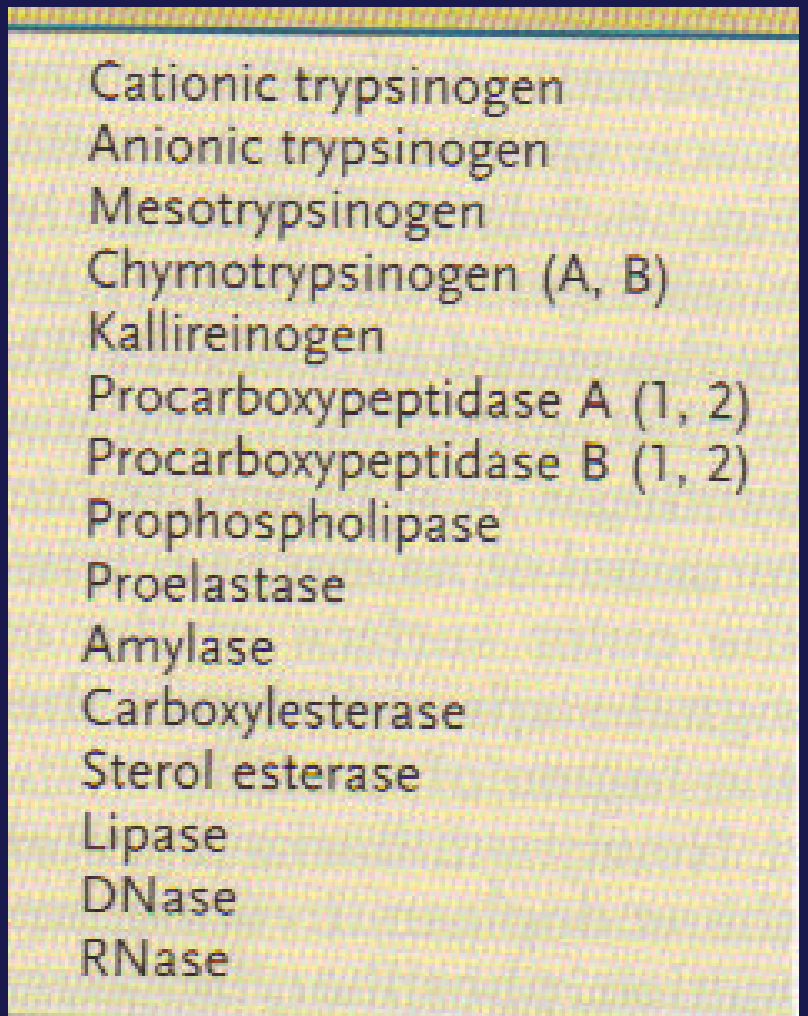

Intermediate hepatic artery
Left hepatic artery
Right and left inferior phrenic arteries (shown here from common stem)
Hepatic artery proper
Hepatic portal vein
Right hepatic artery
Common hepatic artery
Celiac trunk
Cystic artery
Abdominal aorta
Left gastric artery
Gallbladder
Short gastric arteries
Cystohepatic triangle (Calot)
Cystic duct
Common hepatic duct
(Common) bile duct
Right gastric artery
Supraduodenal artery
Gastroduodenal artery
Left gastro-omental (gastroepiploic) artery
Artery to tail of pancreas
Greater pancreatic artery
Splenic artery
Dorsal pancreatic artery
Inferior pancreatic artery
Anastomotic branch
Middle colic artery (cut)
Posterior superior pancreaticoduodenal artery (phantom)
Superior mesenteric artery
Inferior pancreaticoduodenal artery
Anterior superior pancreaticoduodenal artery
Posterior branch of inferior pancreaticoduodenal artery
Right gastro-omental (gastroepiploic) artery
Anterior branch of inferior pancreaticoduodenal artery
F. Netter M.D.
©IGN

PHYSIOLOGY

Pancreatic Acinar Cell Secretion Products

Proenzymes

Enzymes



PHYSIOLOGY

- Islets of Langerhan's
  - B cells (75%)
  - A cells (20%)
  - D cells
  - Pancreatic polypeptide cells

Definition

ACUTE PANCREATITIS IS A COMPLEX DISORDER OF THE EXOCRINE PANCREAS CHARACTERIZED BY ACUTE ACINAR CELL INJURY AND BOTH REGIONAL AND SYSTEMIC INFLAMMATORY RESPONSES

ACUTE PANCREATITIS IS A „NON-BACTERIAL INFLAMMATION, WHICH INITIATES BY PANCREATIC ENZYMES” (S.SCHWARTZ)

Definition

- Typical epigastric pain with
- Increased amylase or lipase (3x greater than normal) or
- Imaging c/w pancreatitis

Epidemiology

- 4.8-38 per 100,000 patients
- Cost of pancreatic diseases= $2.5 billion in 2000
- Acute pancreatitis ranks as the 2nd most common inpatient principal GI diagnosis

Classification

- Mild acute pancreatitis:
  - Minimal or no organ dysfunction
  - Uneventful recovery
- Severe pancreatitis:
  - Organ failure
  - Local complications (necrosis, abscess, pseudocyst)

CLASSIFICATION OF ACUTE PANCREATITIS (ATLANTA, GEORGIA, 1992)

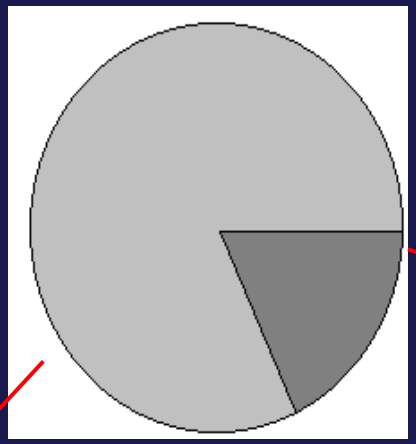
I. EDEMATOUS ACUTE PANCREATITIS

II. NECROTIZING ACUTE PANCREATITIS:

- sterile pancreatic necrosis
- infected pancreatic necrosis

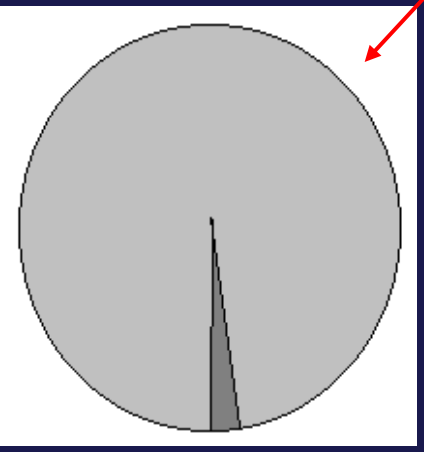
III. COMPLICATIONS OF ACUTE PANCREATITIS:

- parapancreatic infiltrate
- pancreatic abscess
- peritonitis
- retroperitoneal phlegmon
- pancreatic cysts (sterile and infected)
- digestive tract fistulas
- bleeding

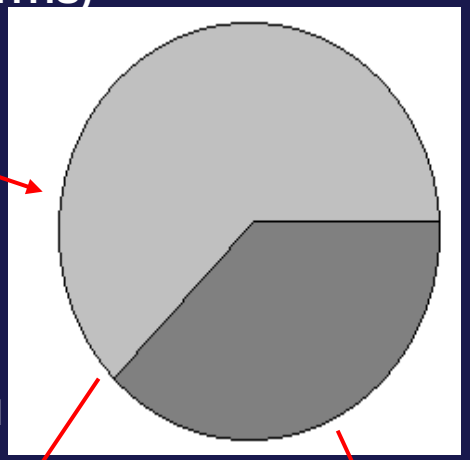


SEVERE FORM
15%
(NECROTISING
PANCREATITIS)

MILD FORM 85%
(EDEMATOUS
PANCREATITIS)

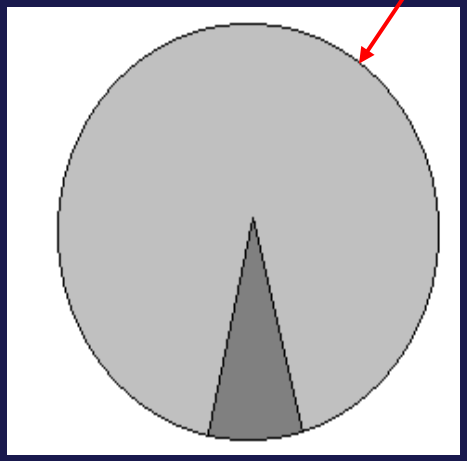


MORTALITY <1%

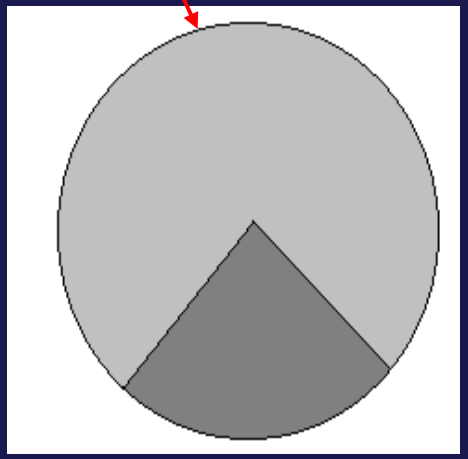


INFECTED
NECROSIS 40%

NO INFECTION
60%



MORTALITY 10%



MORTALITY 30%

Classification
V. Filin

1) **ACUTE EDEMATOUS (INTERSTITIAL) PANCREATITIS**
2) **ACUTE NECROTIZING PANCREATITIS**
3) **SUPPURATIVE-NECROTIZING PANCREATITIS, OR INFECTED NECROSIS OF THE PANCREAS**

FIRST PERIOD: HEMODYNAMICAL DISTURBANCES (1-3 DAYS)

SECOND PERIOD: MULTIORGAN SYSTEM FAILURE (5-7 DAYS)

THIRD PERIOD: LATE SUPPURATIVE COMPLICATIONS (3-4 WEEKS)

CLINICAL CLASSIFICATION OF ACUTE PANCREATITIS

Pancreatic Disease Group, 2005

Mild AP - the same clinical manifestations and biochemical changes as AP, without functional impairment or local complications, and responds well to supplementary fluid treatment

Severity scores are:
- Ranson <3 / APACHE II <8
- CT grade A, B or C

Severe AP - the same clinical manifestations and biochemical changes as AP, plus one of the following:
- the local complication of pancreatic necrosis
- pseudocyst or infected pancreatic tissue
- functional impairment of other organs

Severity scores are :
- Ranson ≥3 / APACHE II ≥8
- CT grade D or E

Classification

- **Working Group Classification – 2007**

❖**ACUTE PANCREATITIS**

- Interstitial edematous pancreatitis (IEP)
- Necrotizing pancreatitis (pancreatic necrosis and/or peripancreatic necrosis)
  - Sterile necrosis
  - Infected necrosis

Classification

- **FLUID COLLECTIONS DURING ACUTE PANCREATITIS**
- **(<4 weeks after onset of pancreatitis)**
- Acute peripancreatic fluid collection (APFC)
  - Sterile
  - Infected
- Post-necrotic pancreatic/peripancreatic fluid collection (PNPFC)
  - Sterile
  - Infected

Classification

- **(>4 weeks after onset of pancreatitis)**
- Pancreatic pseudocyst (usually has increased amylase/lipase activity)
  - Sterile
  - Infected
- Walled-off pancreatic necrosis (WOPN) (may or may not have increased amylase/lipase activity)
  - Sterile
  - Infected

CLINICAL TERMINOLOGY

The terms ‘acute edematous pancreatitis’ or ‘acute necrotizing pancreatitis’ are no longer in use unless there are positive pathologic findings. The terms ‘acute hemorrhagic necrotizing pancreatitis’ and ‘acute pancreatic cellulitis’ are obsolete.

Clinically, the diagnosis should include the etiology, grade of disease and complications e.g. AP (biliary in origin, severe degree, acute respiratory distress syndrome (ARDS)) or AP (biliary in origin, mild degree).

For clinical purposes only, one can use the Ranson’s score or CT grading; for clinical research, the APACHE score and CT grade must always be included.

Pancreatic Disease Group, 2005

Terminology

① Pancreatic necrosis:

- Focal or diffuse nonviable pancreatic parenchyma and usually peripancreatic fat necrosis. Can be infected or sterile.

② Acute fluid collection:

- Fluid in or near the pancreas that lacks a definite wall and that occurs early in the course of acute pancreatitis.

③ Pseudocyst:

- Fluid collection that persists for 4-6 weeks and becomes encapsulated by a wall of fibrous or granulation tissue

Terminology

④ Pancreatic abscess:

- Circumscribed intra-abdominal collection of pus after an episode of acute pancreatitis or pancreatic trauma
- It usually develops close to the pancreas and contains little pancreatic necrosis.

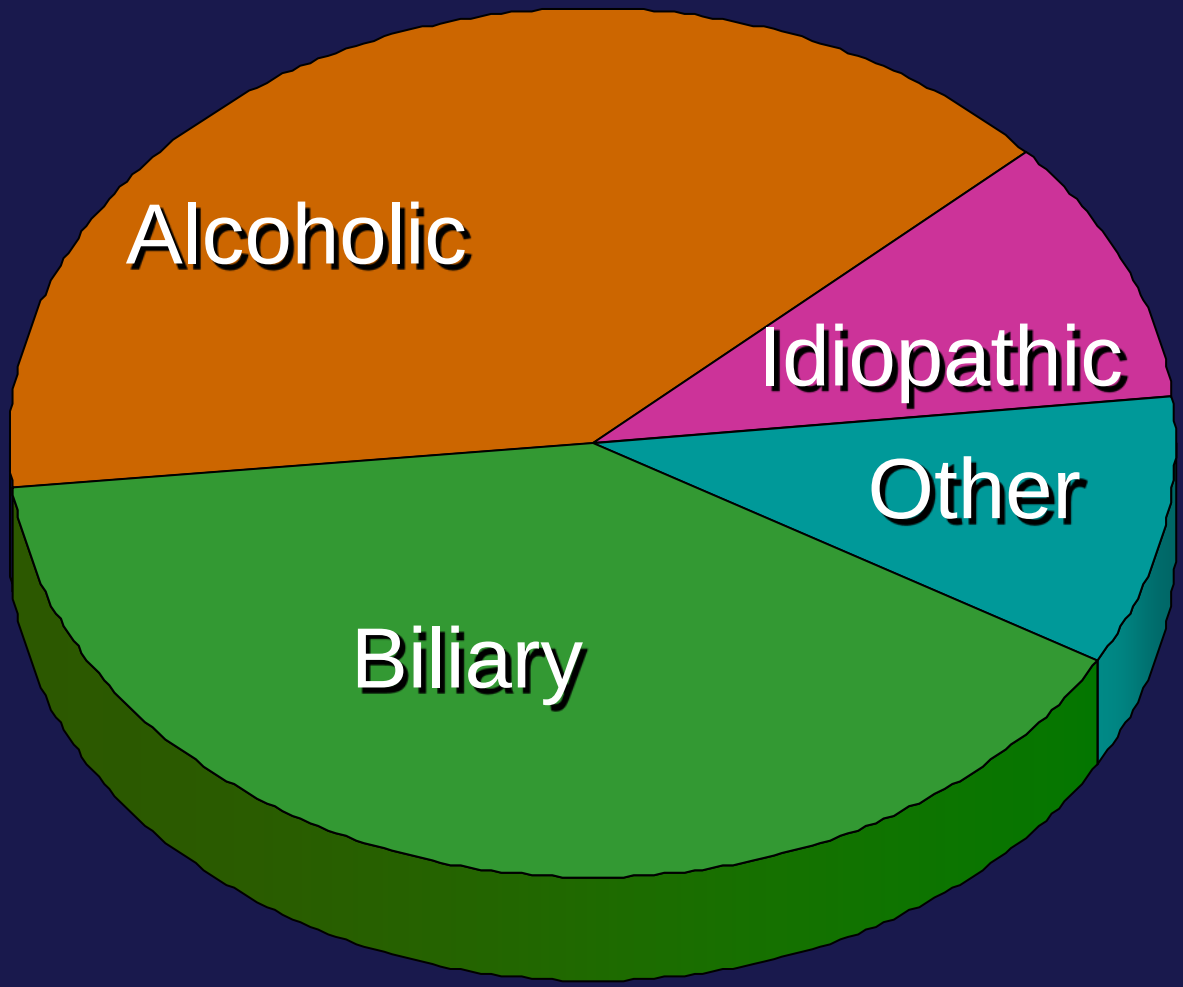
Natural History

- Mild:
  - 80% of attacks
  - Normal pancreatic morphology and function are the rule after recovery if there are no further attacks
- Severe:
  - 20% of attacks
  - Commonly accompanied by necrosis of the pancreas and/or organ failure
  - About 25-33% with severe pancreatitis die from their disease.
  - About 50% of deaths occur within the first 2 weeks, usually from multiorgan failure.

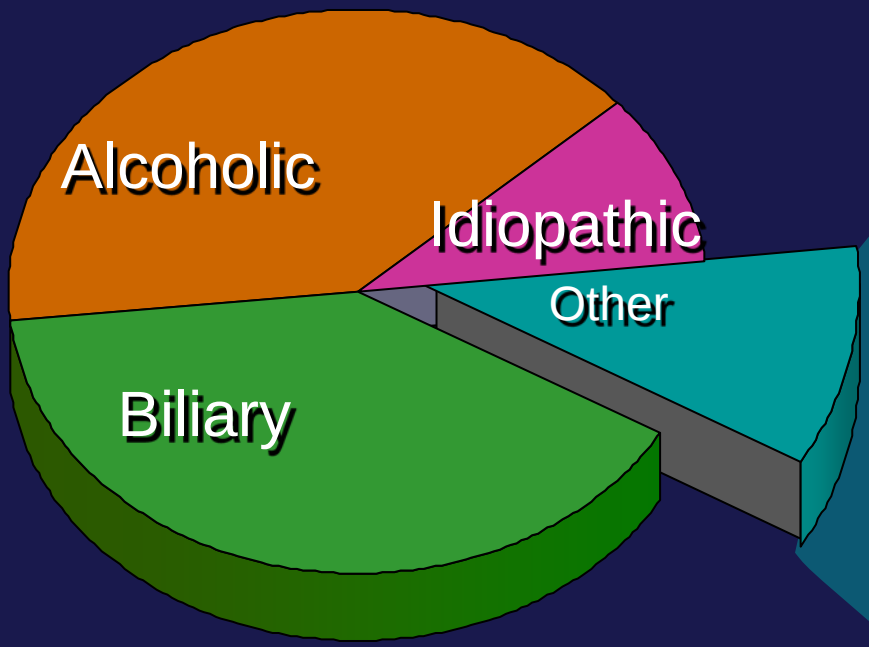
Natural History

- Patients who are older and have comorbid illnesses have a substantially higher rate of mortality.
- In those who survive the illness, severe pancreatic necrosis can scar the pancreas, resulting in a stricture of the main pancreatic duct with subsequent obstructive chronic pancreatitis and permanent diabetes and malabsorption.

Etiology



Etiology



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

Etiology

Class	Example	Mechanism
Viral	Coxsackie	Unclear
Parasitic	Ascaris	Obstructive
Fungal	Candida	Unclear
Bacterial	Salmonella	Toxin

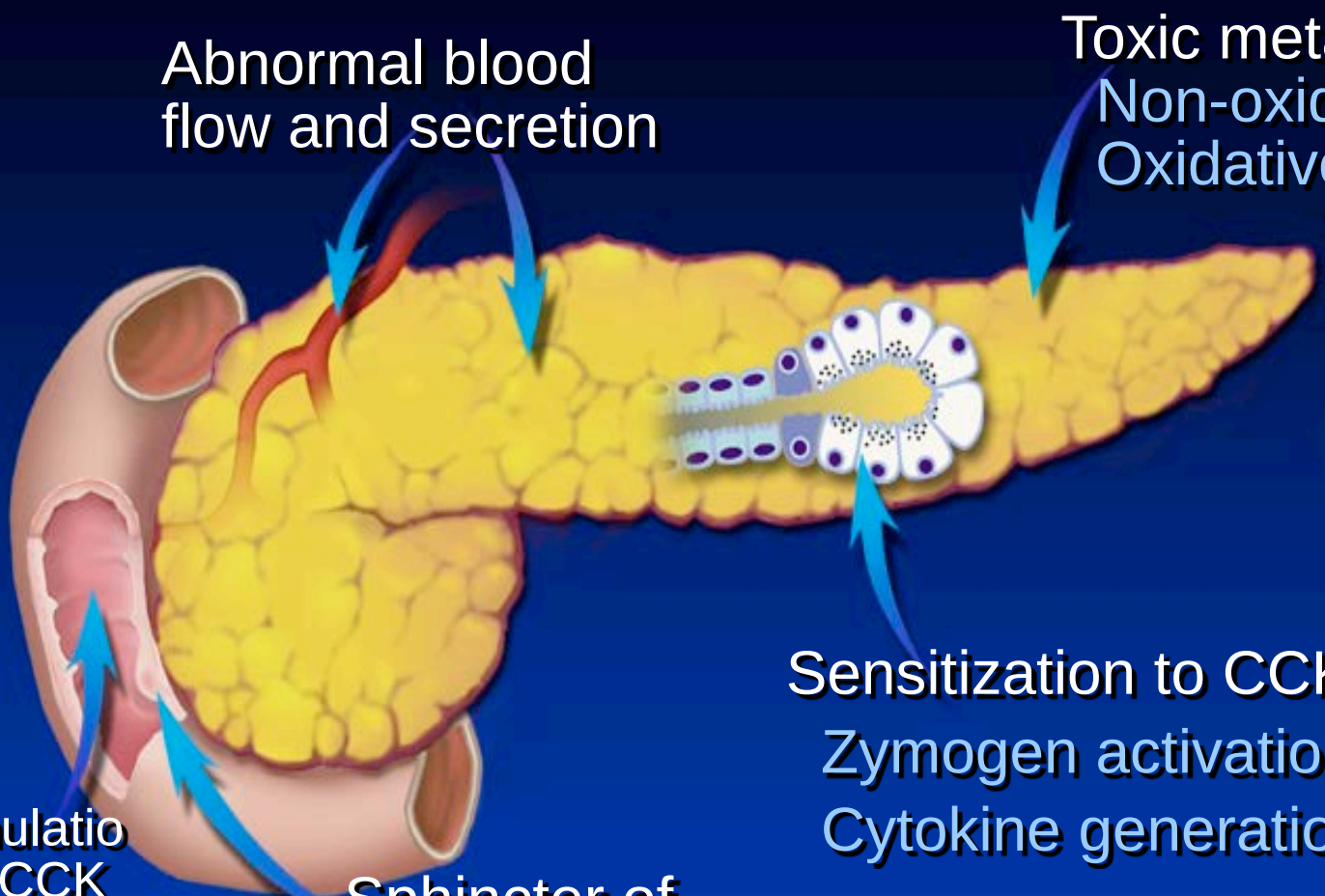
Inherited Causes

- Altered enzyme activity
  - Trypsinogen mutations
- Abnormal ion movement
  - Cystic fibrosis transmembrane regulator (CFTR) mutations
- Metabolic
  - Familial hypertriglyceridemia

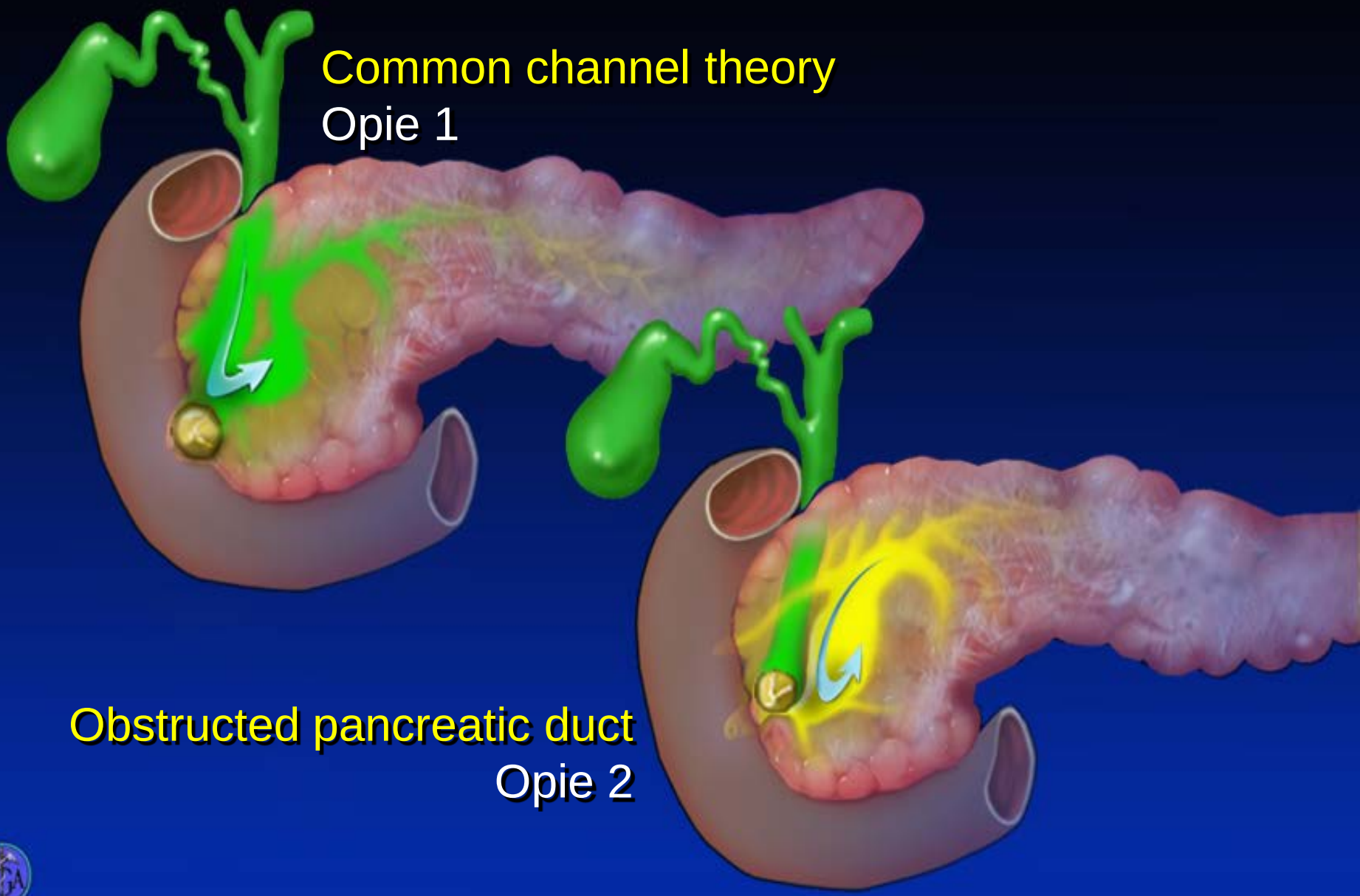
Drug Induced Pancreatitis Sorted by Incidence

Common	Uncommon	Rare
asparaginase	ACE inhibitors	carbamazepine
azathioprine	acetaminophen	corticosteroids
6-mercaptopurine	5-amino ASA	estrogens
didanosine (DDI)	furosemide	minocycline
pentamidine	sulfasalazine	nitrofurantoin
valproate	thiazides	tetracycline

Acute Alcohol Effects



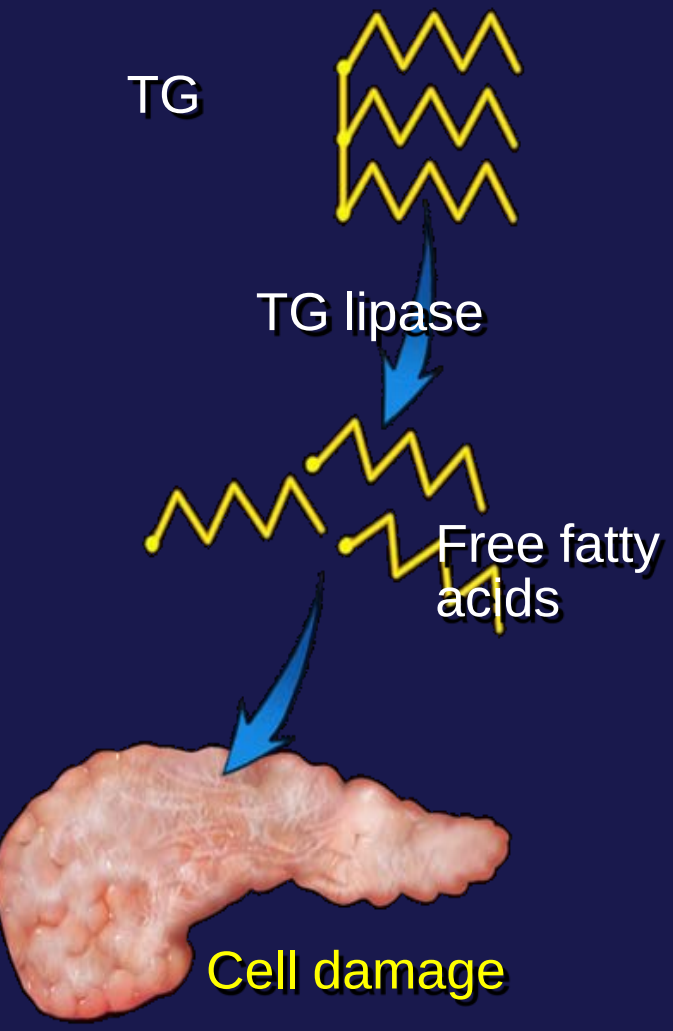
Gallstone Pancreatitis Mechanism



Hypertriglyceridemia

- Rare cause of acute pancreatitis
- Serum triglycerides usually >1000 mg/dL
- May cause chronic disease
- Can be drug-induced:

  Alcohol, estrogens, isotretinoin, HIV-protease inhibitors



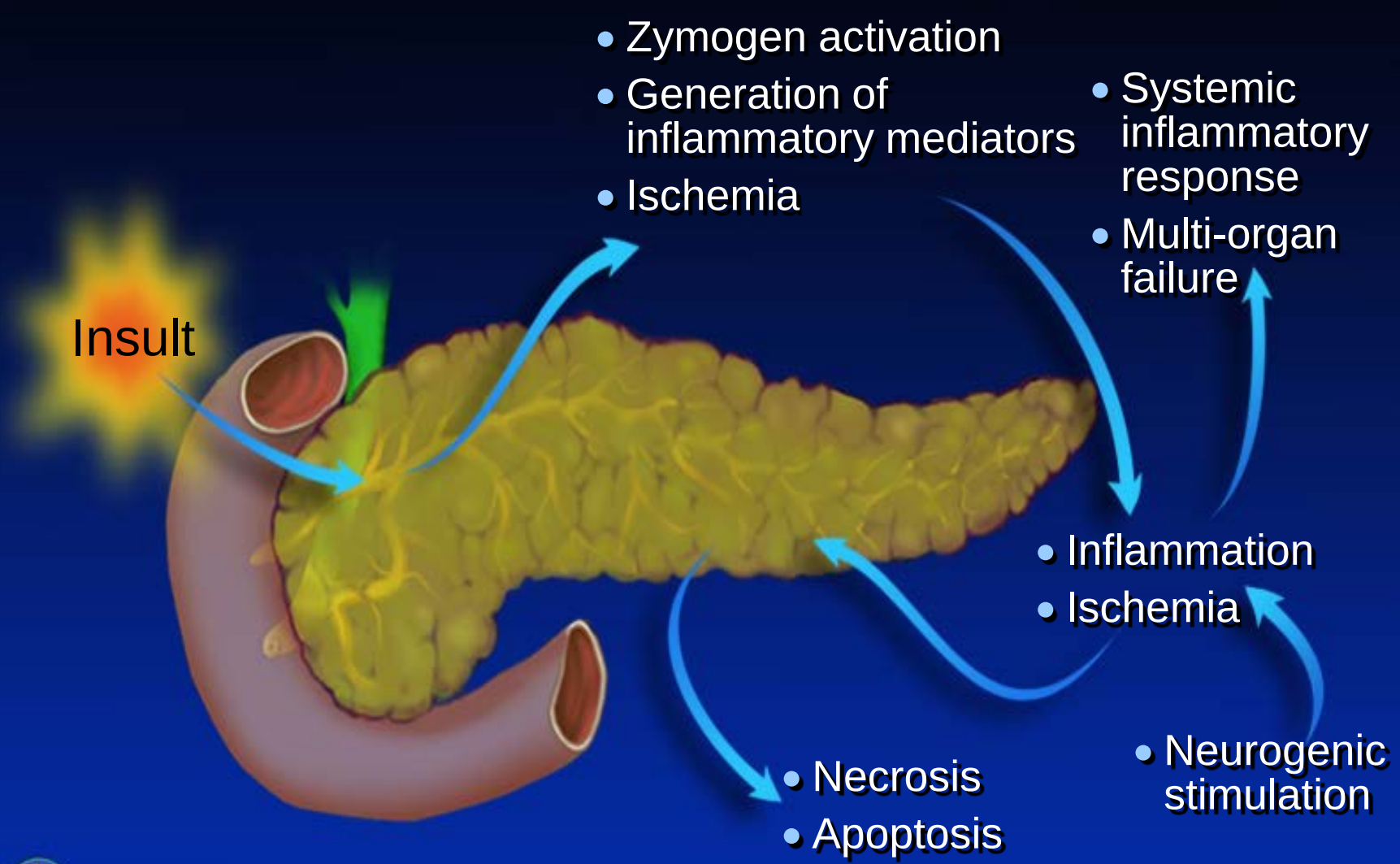
Pathogenesis

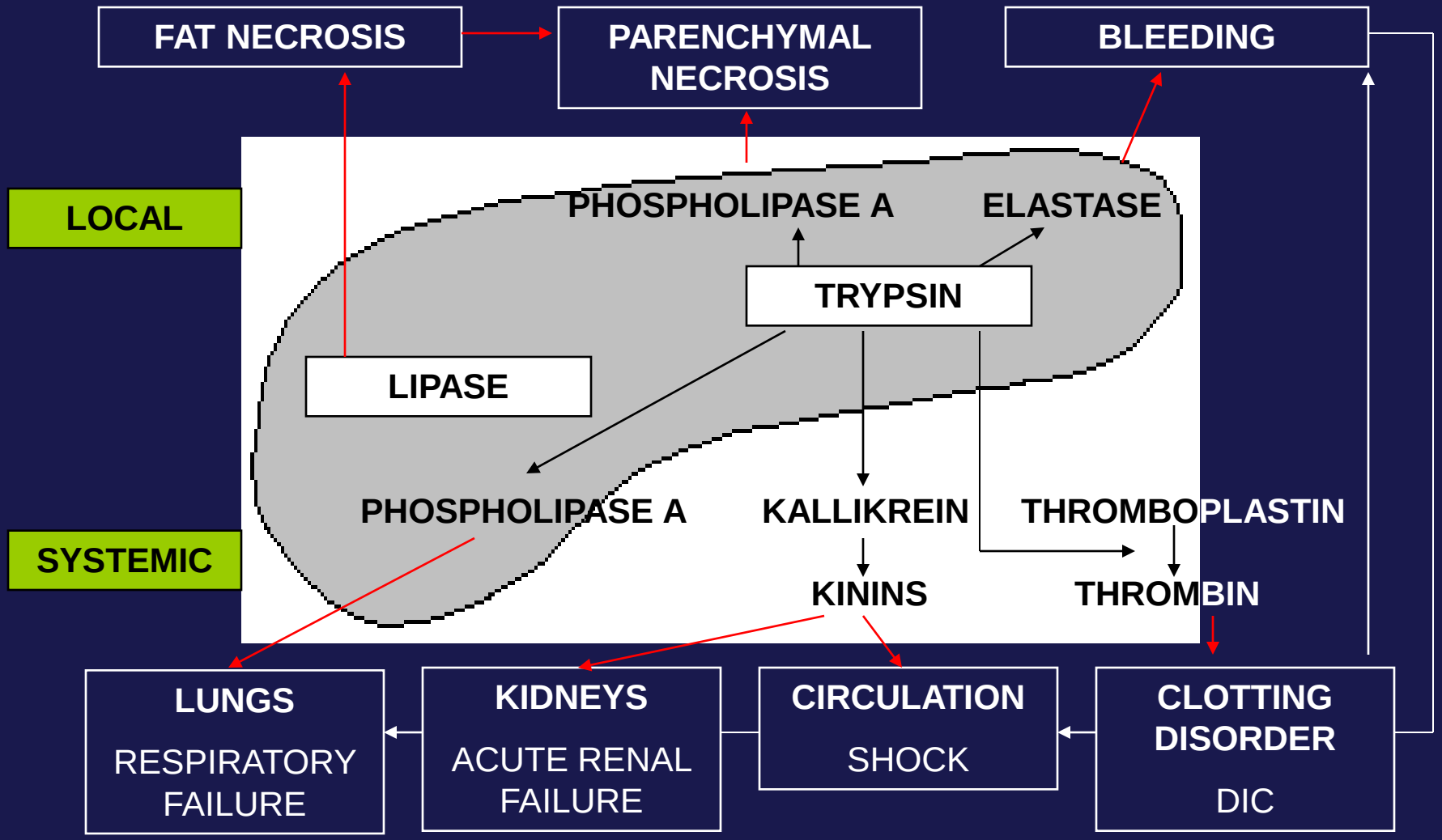
Pancreatitis evolves in 3 phases:

- First: Characterized by intrapancreatic digestive enzyme activation and acinar cell injury.
- Second: Involves the activation, chemoattraction, and sequestration of neutrophils in the pancreas resulting in an intrapancreatic inflammatory reaction of variable severity.
- Third: Due to the effects of activated proteolytic enzymes and mediators, released by the inflamed pancreas, on distant organs.

Pathogenesis

Mechanisms




FAT NECROSIS
PARENCHYMAL NECROSIS
BLEEDING
LOCAL
PHOSPHOLIPASE A
ELASTASE
TRYPSIN
LIPASE
SYSTEMIC
PHOSPHOLIPASE A
KALLIKREIN
THROMBOPLASTIN
KININS
THROMBIN
LUNGS
RESPIRATORY FAILURE
KIDNEYS
ACUTE RENAL FAILURE
CIRCULATION
SHOCK
CLOTTING DISORDER
DIC

Symptoms and Signs

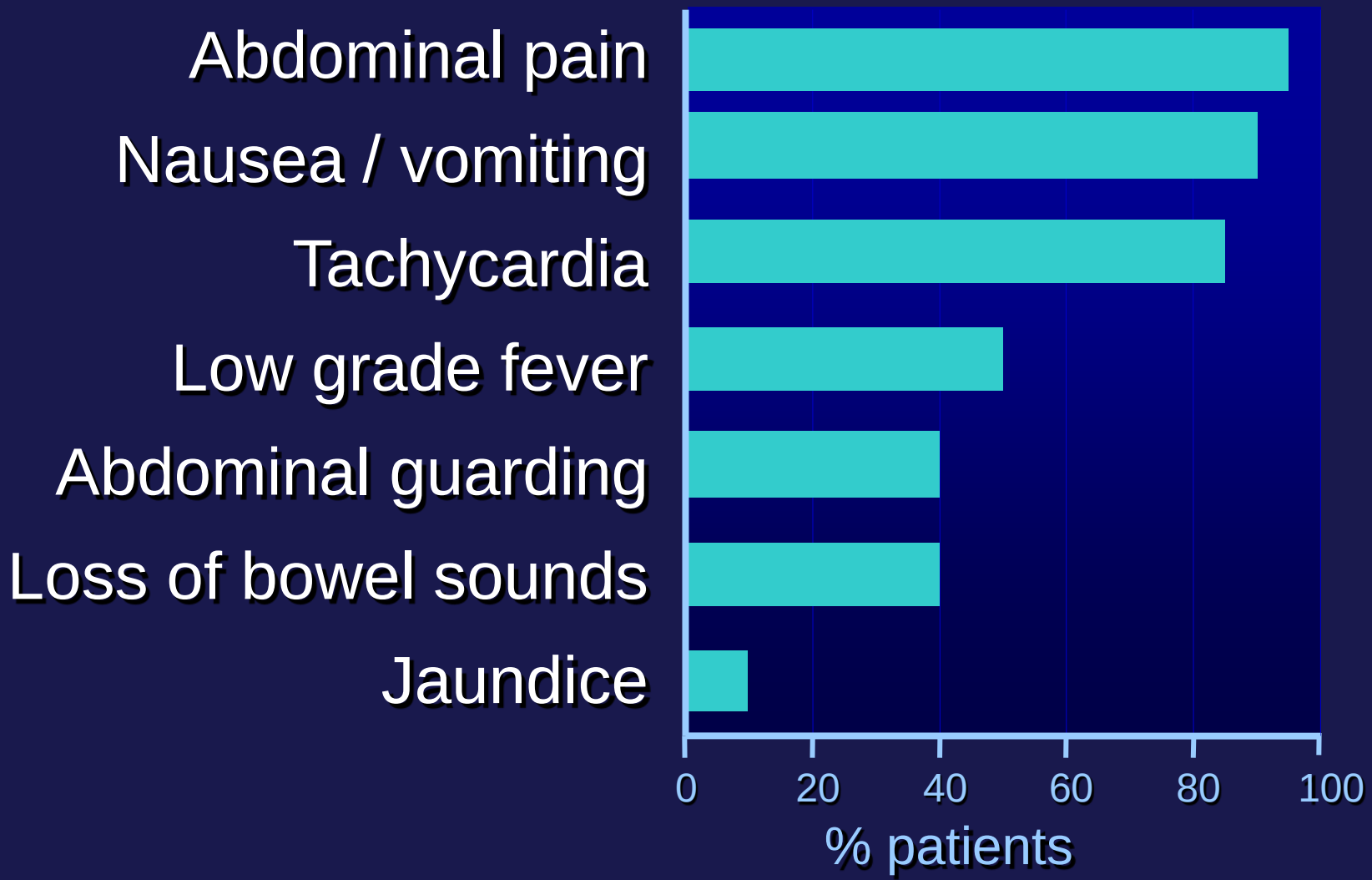
- Abdominal pain
- Nausea and vomiting
- Anorexia
- Fever
- Hypovolemia
- Ileus
- Abdominal tenderness
- Left pleural effusion
- Altered mental status
- Jaundice
- ARDS

- Mortality rate-1% for mild acute pancreatitis

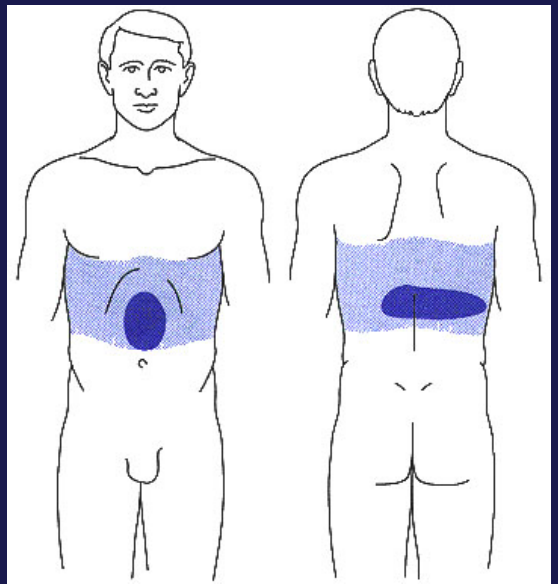
  75-90% for severe acute pancreatitis.

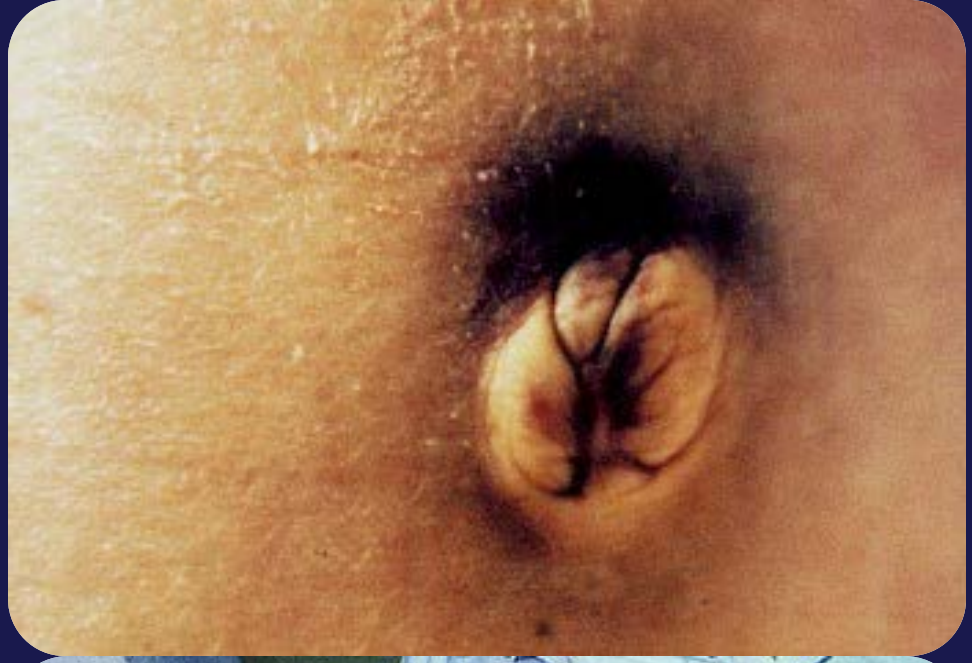
  Overall mortality rate of 15-20%

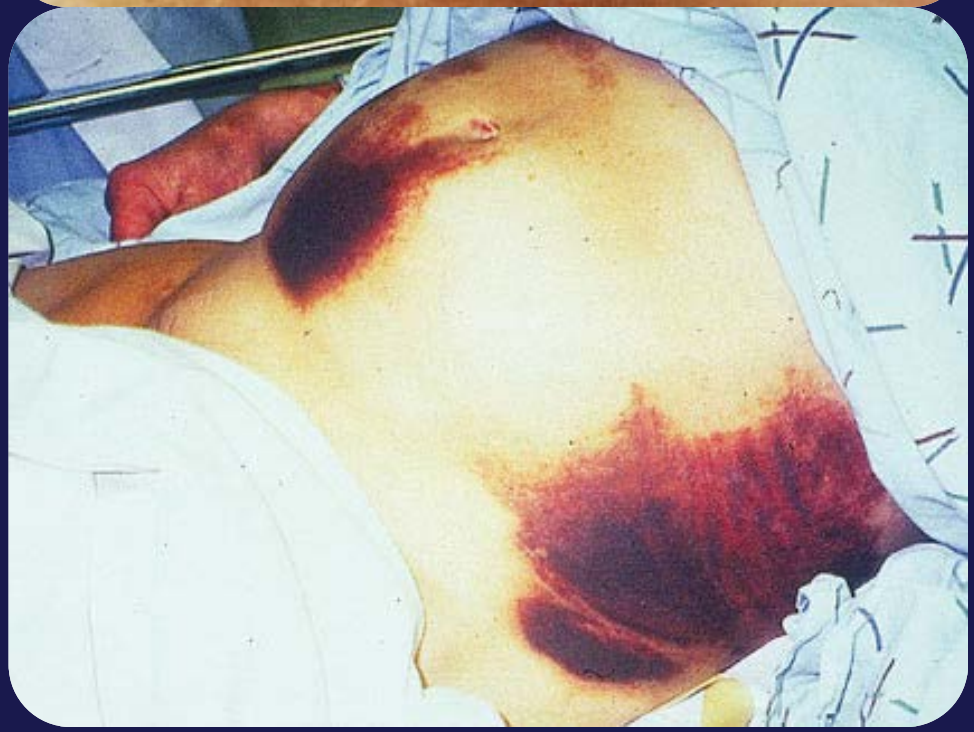
Presenting Features



- BEREZNIGOVSKI’S SIGN
- MAYO – ROBSON’S SIGN
- MONDOR’S SIGN
- GREY-TURNER’S SIGN
- CULLEN’S SIGN
- BONDE’S SIGN
- MANDEL-RAZDOLSCKI’S SIGN
- GOBIEF’S SIGN
- KERVEN’S SIGN
- KÖRTE’S SIGN
- VOSKRESENSKI’S SIGN







Lab Diagnosis

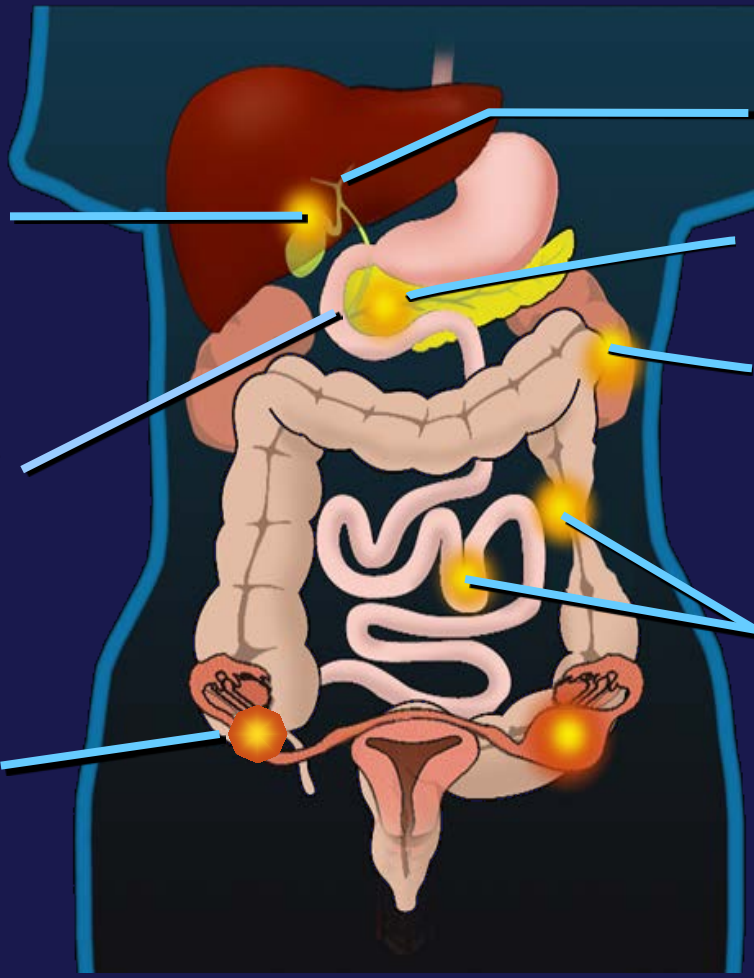
- Complete blood count
- Renal function tests
- Liver function
- Serum amylase - 3-4 times increase
  (Normal range <115IU)
- Serum lipase - lipase levels may have a slightly greater sensitivity, particularly when measured late (> 24 hours) after initial presentation
- Serum calcium
- Arterial blood gas

Lab Diagnosis

① Serum and Urine Amylase :

- Pancreas accounts for 40-45% of serum amylase, and the salivary glands account for the rest.
- The serum amylase value is usually increased on the first day of symptoms and remains elevated for 3 to 5 days in uncomplicated attacks.
- There is no definite correlation between the severity of pancreatitis and the degree of serum amylase elevation.
- Serum amylase value may be normal in hypertriglyceridemia associated pancreatitis

Elevated Serum Amylase



Cholangitis

Cholecystitis

Pancreatitis

Kidney stone

Peptic ulcer

Intestinal obstruction, ischemia or perforation

Ectopic pregnancy

Lab Diagnosis

② Serum Lipase:

- The sensitivity of measurements for the diagnosis is similar to that of serum amylase measurements (85-100%)
- Serum lipase is always elevated on the 1st day of illness and remains elevated longer than serum amylase content.
- Is Lipase more specific than amylase?

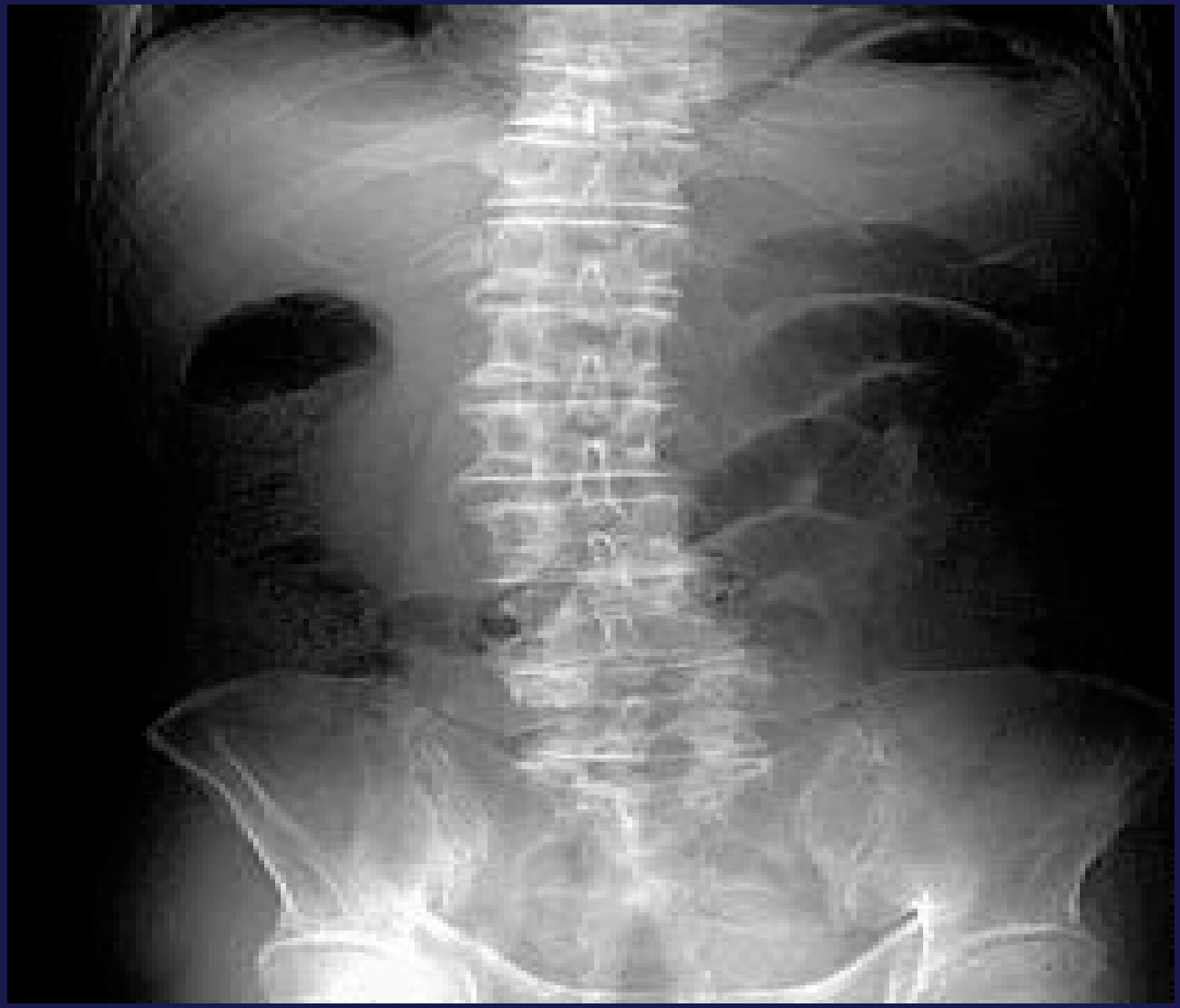
Conditions Associated with Hyperamylasemia and Hyperlipasemia

	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

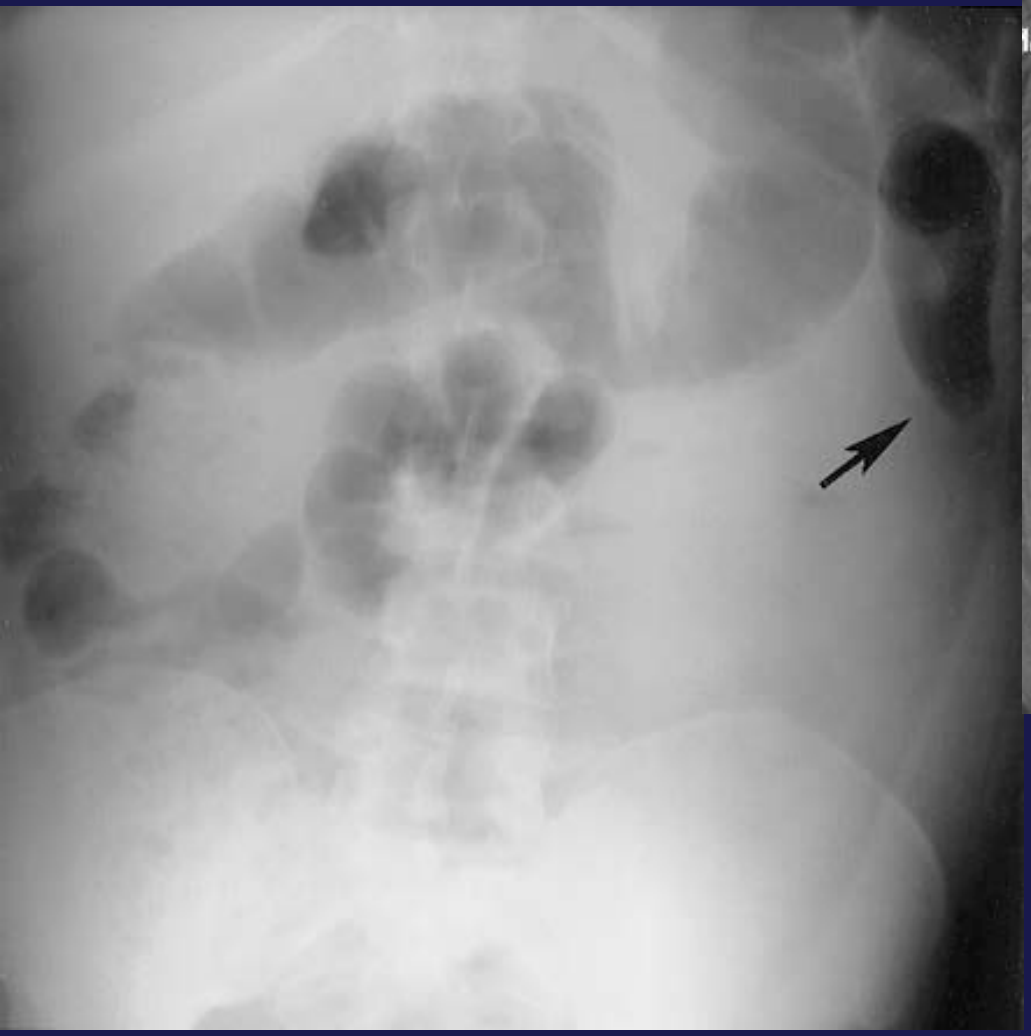
Radiologic Diagnosis

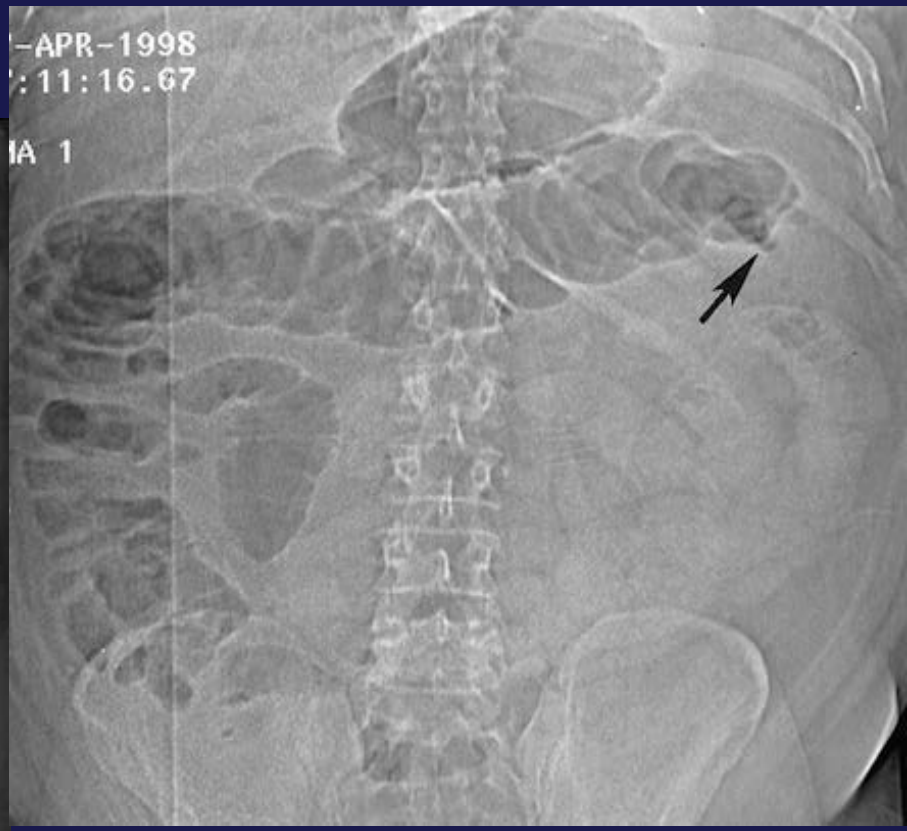
1. Abdominal and chest plain film:
   - Excludes other causes of acute abdominal pain-obstruction and perforation.
   - AXR - Frequently normal or may demonstrate ileus: multiple air fluid interphase; pancreatic calcifications, calcified gall stones, “sentinel loop” sign, “colon cut off” sign, “renal halo” sign
   - Chest radiograph can detect pulmonary complications:
     - Atelectasis
     - Pleural effusions (most commonly left-sided)
     - Infiltrates suggestive of adult respiratory distress syndrome

Sentinel Loop Sign



Colon Cut Off Sign



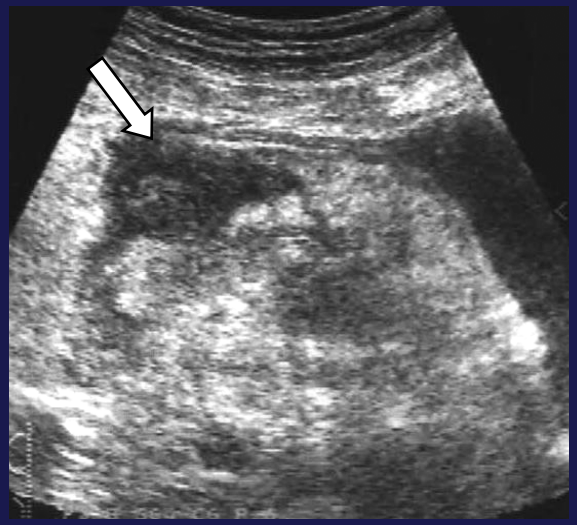


Radiologic Diagnosis

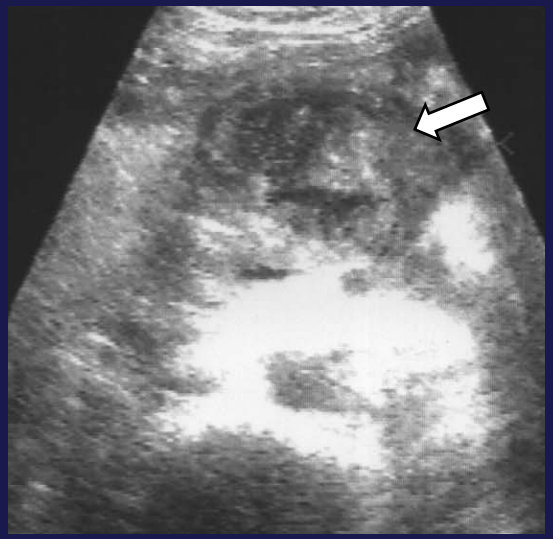
❷ Abdominal Ultrasound

- Used in 1st 24 hours to search for:
  - Gallstones
  - Dilatation of the common bile duct due to choledocholithiasis
  - Ascites
- Pancreas is usually diffusely enlarged and hypoechoic, interstitial edema, extra pancreatic fluid collections.
- Evidence of chronic pancreatitis (intraductal or parenchymal calcification) and dilation of the pancreatic duct, may also be seen.
- Not a good imaging modality to ascertain severity of pancreatitis.
- Limited value due to presence of intestinal gas

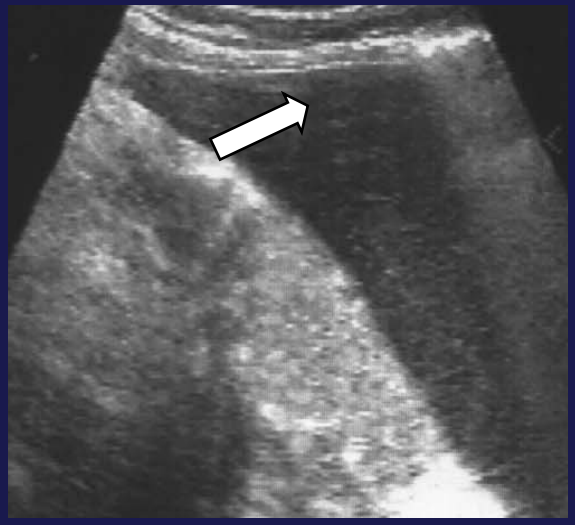
MAIN ECOGRAPHIC SIGNS



FREE FLUID IN THE LESSER SAC



LEFT SIDE RETROPERITONEAL PHLEGMON



FREE FLUID IN ABDOMINAL CAVITY

Radiologic Diagnosis

❸ EUS

- Not helpful in acute pancreatitis.
- More sensitive than either abdominal US or CT to detect common duct stones.
- May exclude a common duct stone in patients with severe pancreatitis and jaundice (serum bilirubin > 5 mg/dL).
- ERCP, in this situation, may worsen pancreatitis

Radiologic Diagnosis

❹ CECT:

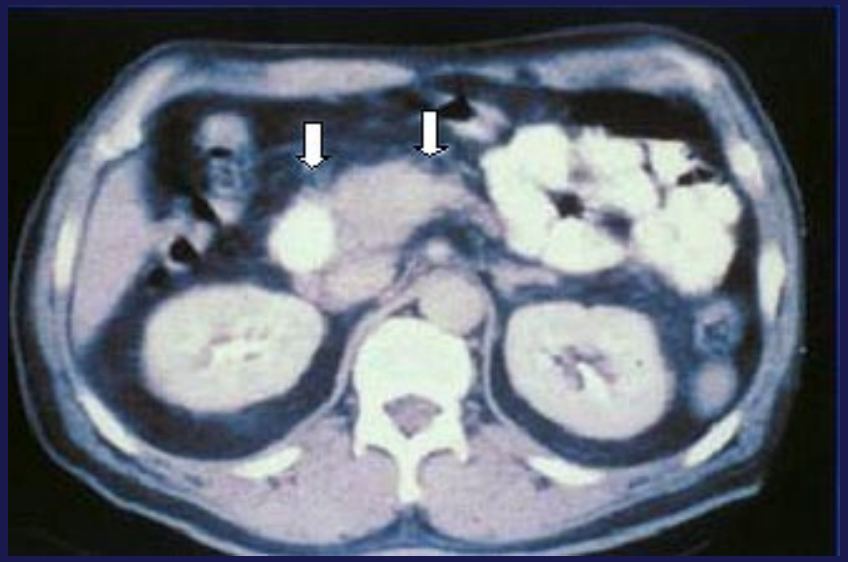
- The most important imaging modality for the diagnosis of acute pancreatitis and intra-abdominal complications.
- 3 main indications:
  - ① To exclude other serious intra-abdominal conditions: mesenteric infarction or a perforated ulcer
  - ② To stage the severity of acute pancreatitis
  - ③ To determine whether complications are present

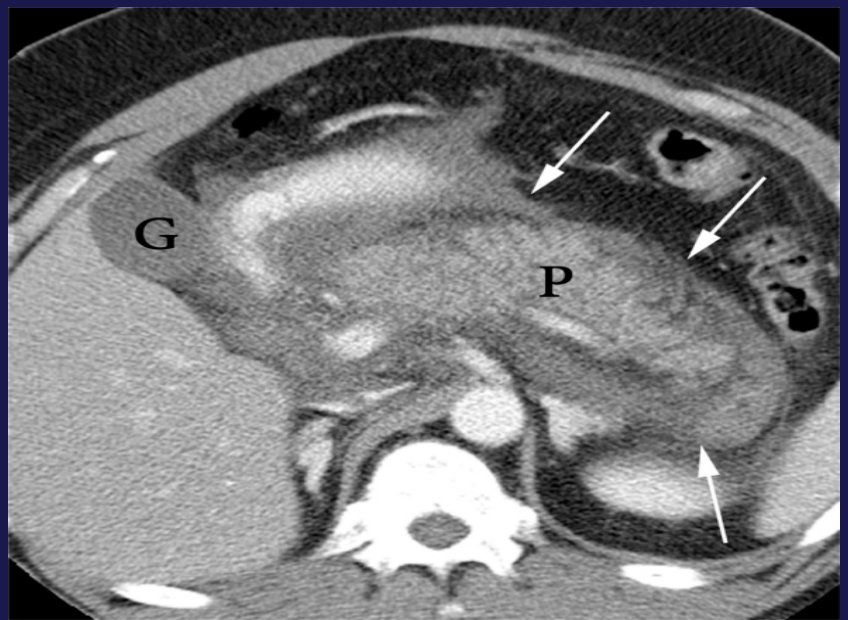
When to Take a CT

- When the diagnosis is in doubt
- Patients with persisting organ failure
- Signs of sepsis
- Deterioration in clinical status after admission
- CRP > 110 mg/lt
- Ranson score > 3, APACHE II score > 8

Radiologic Diagnosis

CT features in interstitial pancreatitis:





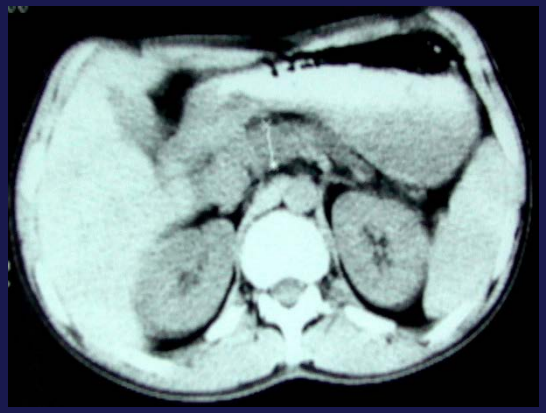
- Homogenous contrast enhancement
- Diffuse or segmental pancreatic enlargement
- Irregularity
- Heterogeneity
- Lobularity of the pancreas
- Obliteration of the peripancreatic fat planes

BALTHAZAR CT Grading

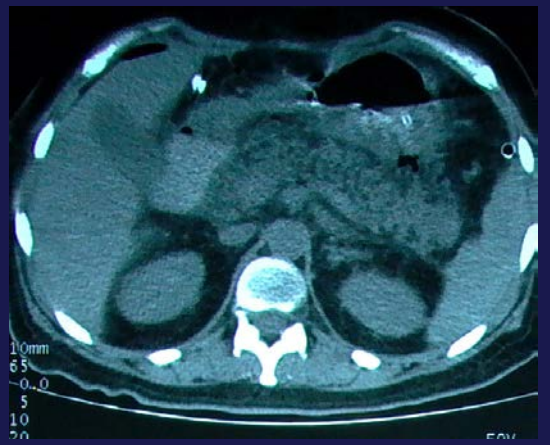
[A] Normal pancreas	0
[B] Edematous pancreatitis	1
[C] Any of the above + peripancreatic inflammation + < 30% pancreatic necrosis	2
[D] Any of the above + single extrapancreatic fluid collection + 30–50% pancreatic necrosis	3
[E] Any of the above+ extensive extrapancreatic fluid collection, pancreatic abscess + > 50% pancreatic necrosis	4

CT severity index = CT grade + necrosis score

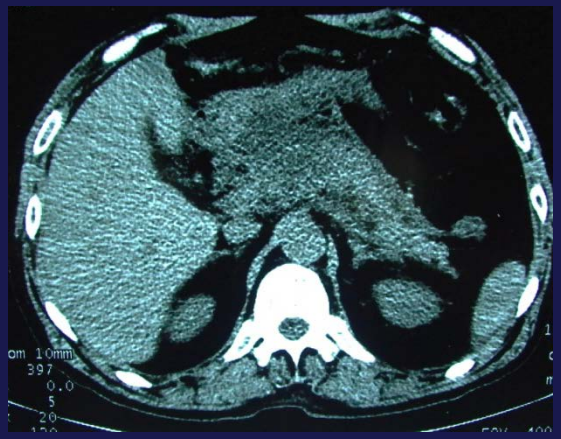
BALTHAZAR GRADING SYSTEM FOR ACUTE PANCREATITIS



A – NORMAL PANCREAS (0 POINTS)

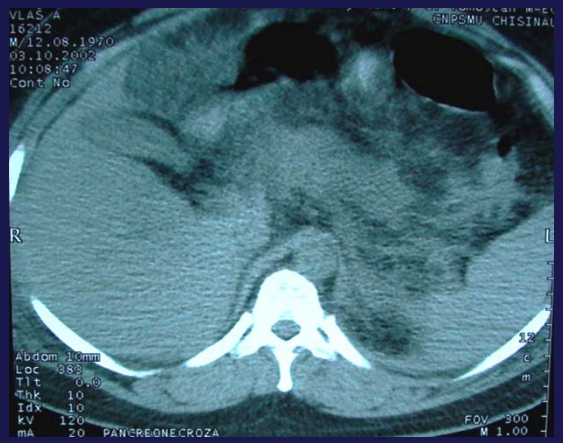


B – GLAND ENLARGEMENT, SMALL INTRAPANCREATIC FLUID COLLECTION (1 POINT)



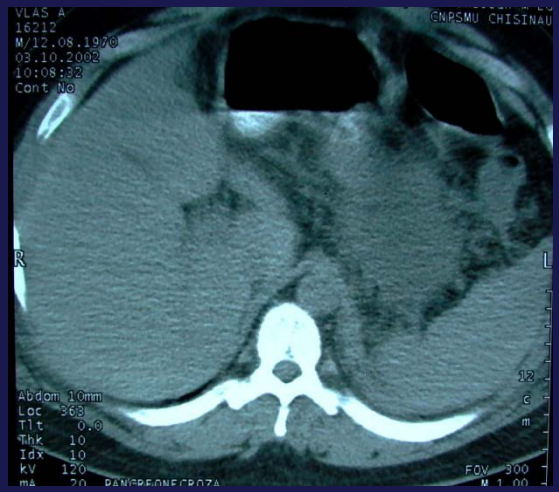
C – ANY OF THE ABOVE + PERIPANCREATIC INFLAMMATION (2 POINTS)

BALTHAZAR GRADING SYSTEM FOR ACUTE PANCREATITIS



D – ANY OF THE ABOVE + SINGLE EXTRAPANCREATIC FLUID COLLECTION (3 POINTS)

E – ANY OF THE ABOVE + EXTENSIVE EXTRAPANCREATIC FLUID COLLECTION, PANCREATIC ABSCESS (4 POINTS)



Normal pancreas (0 points)

Intrinsic pancreatic abnormalities with or without peripancreatic inflammatory changes (2 points)

Pancreatic/peripancreatic fluid collection or peripancreatic fat necrosis (4 points)

Pancreatic necrosis

- None (0 points)
- ⩽30% gland necrosis (2 points)
- >30% gland necrosis (4 points)

Extrapancreatic complications, i.e., pleural effusion, ascites, vascular or gastrointestinal involvement, parenchymal complications (2 points)

CTSI score and disease severity of acute pancreatitis

Score 0–3: mild acute pancreatitis

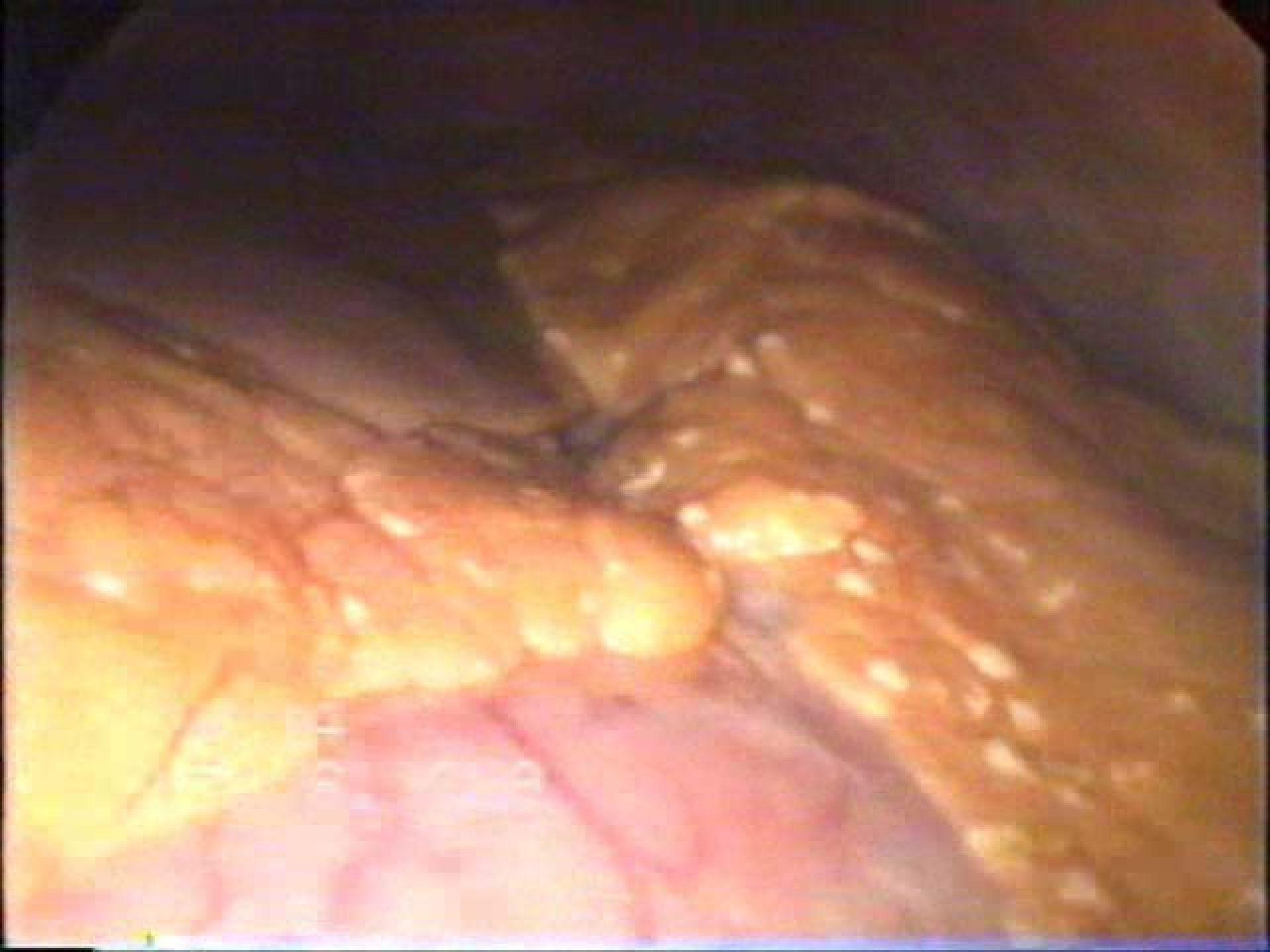
Score 4–6: moderate acute pancreatitis

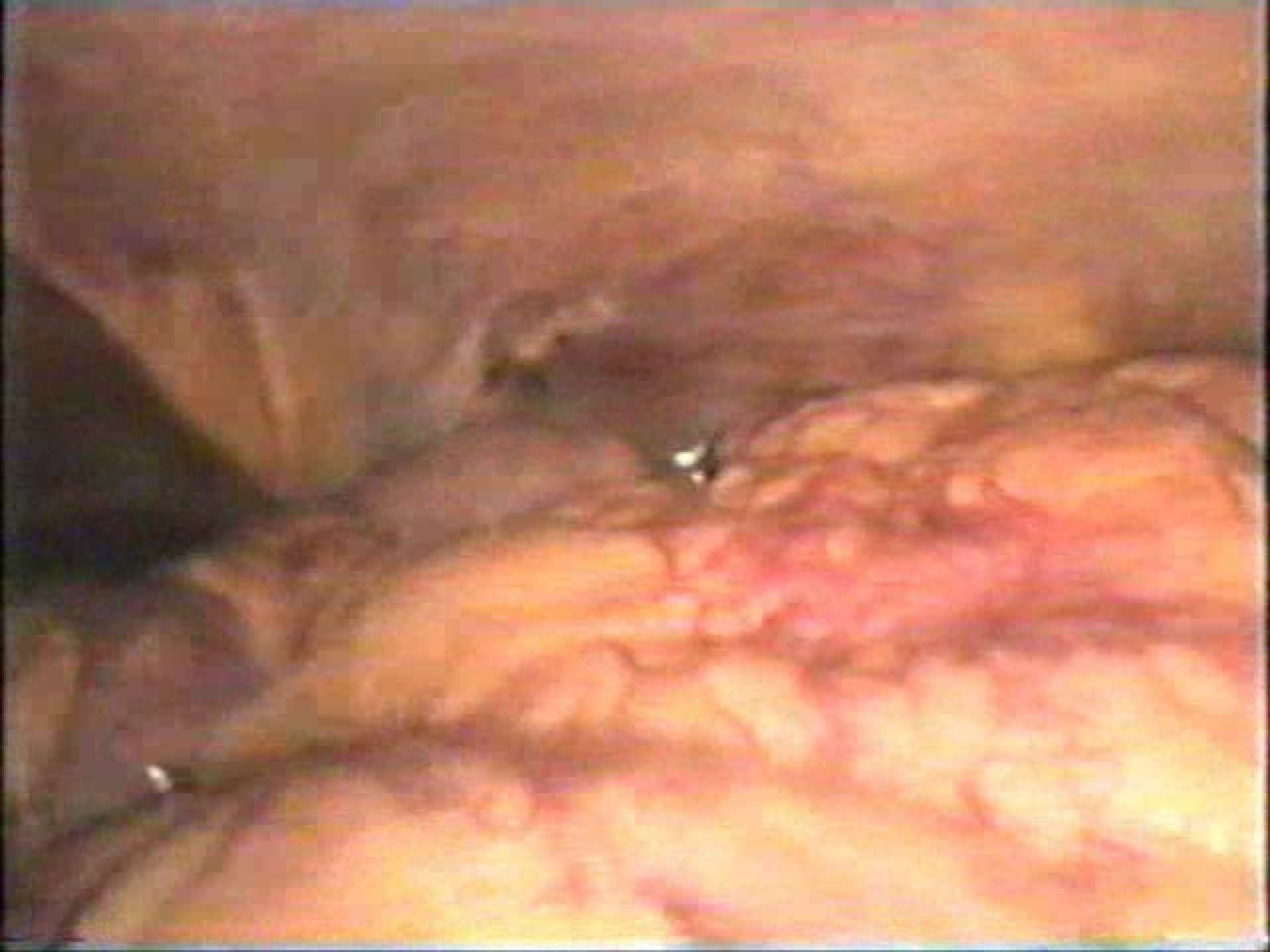
Score 7–10: severe acute pancreatitis

Radiologic Diagnosis

❺ MRI

- Provides information regarding the severity of pancreatitis similar to CT.
- As good as CT in detecting necrosis and fluid collections
- Gadolinium, unlike intravenous contrast agents used for CT, is safe to use in renal failure.
- Less accessible and more expensive than CT.





Predictors of Severity

Atlanta Criteria for Severe Acute Pancreatitis

Table 58-1 Atlanta Criteria for Severe Acute Pancreatitis[13]

Organ Failure
- a. Shock: systolic blood pressure <90 mm Hg
- b. Pulmonary insufficiency: PaO_2 ≤60 mm Hg
- c. Renal failure: serum creatinine >2 mg/dL
- d. Gastrointestinal bleeding: >500 mL/24 hr

Local Complications
- a. Necrosis
- b. Abscess
- c. Pseudocyst

Unfavorable Early Prognostic Signs
- a. Ranson's signs (see Table 58-2)
- b. APACHE-II points

Ranson's Criteria

At Admission

- Age in years >55years
- White blood cell count > 16000/mcL
- Blood glucose > 11 mmol/L (>200 mg/dL)
- Serum AST > 250 IU/L
- Serum LDH > 350 IU/L

After 48 Hours

- Haematocrit fall > 10%
- Increase in BUN by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration
- Hypocalcemia (serum calcium < 2.0 mmol/L (<8.0 mg/dL))
- Hypoxemia (P_{O2} < 60 mmHg)
- Base deficit > 4Meq/L
- Estimated fluid sequestration > 6L

- If the score >=3, severe pancreatitis likely
- If the score < 3, severe pancreatitis is unlikely

APACHE II Scores

- May be used daily
- Positive and negative predictive values similar to Ranson score at 48 hrs after admission.
- Point assignment:
  - 12 physiologic variables
  - Age
  - Chronic health status
- Scores on admission and within 48 hours help distinguish mild from severe pancreatitis and to predict death.
- 1st 48 hrs: Scores ≤9 may survive, scores ≥13 usually die

Apache II

A. Total Acute Physiology Score

- Temperature (°C)
- Mean arterial pressure (mmHg)
- Heart rate
- Respiratory rate
- Fi O2
- Serum HCO3-(mmol/L)
- Arterial pH
- Serum sodium (mmol/L)
- Serum potassium (mmol/L)
- Serum creatinine
- Hematocrit (%)
- WBC (x103/ mm3)

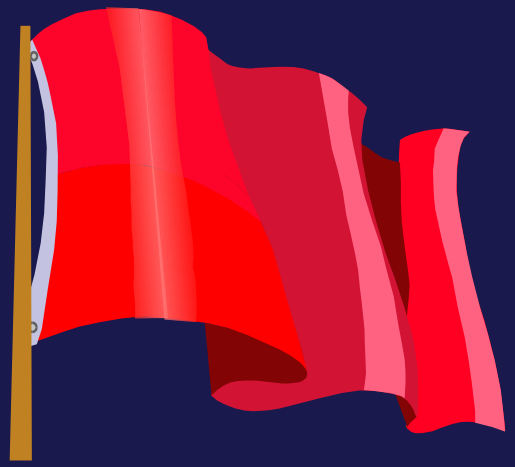
B. Age Points

C. Chronic Health Points

Score of 8 or more indicates severe acute pancreatitis

Early Indicators of Severity

- Tachycardia, hypotension
- Tachypnea, hypoxemia
- Hemoconcentration
- Oliguria
- Encephalopathy

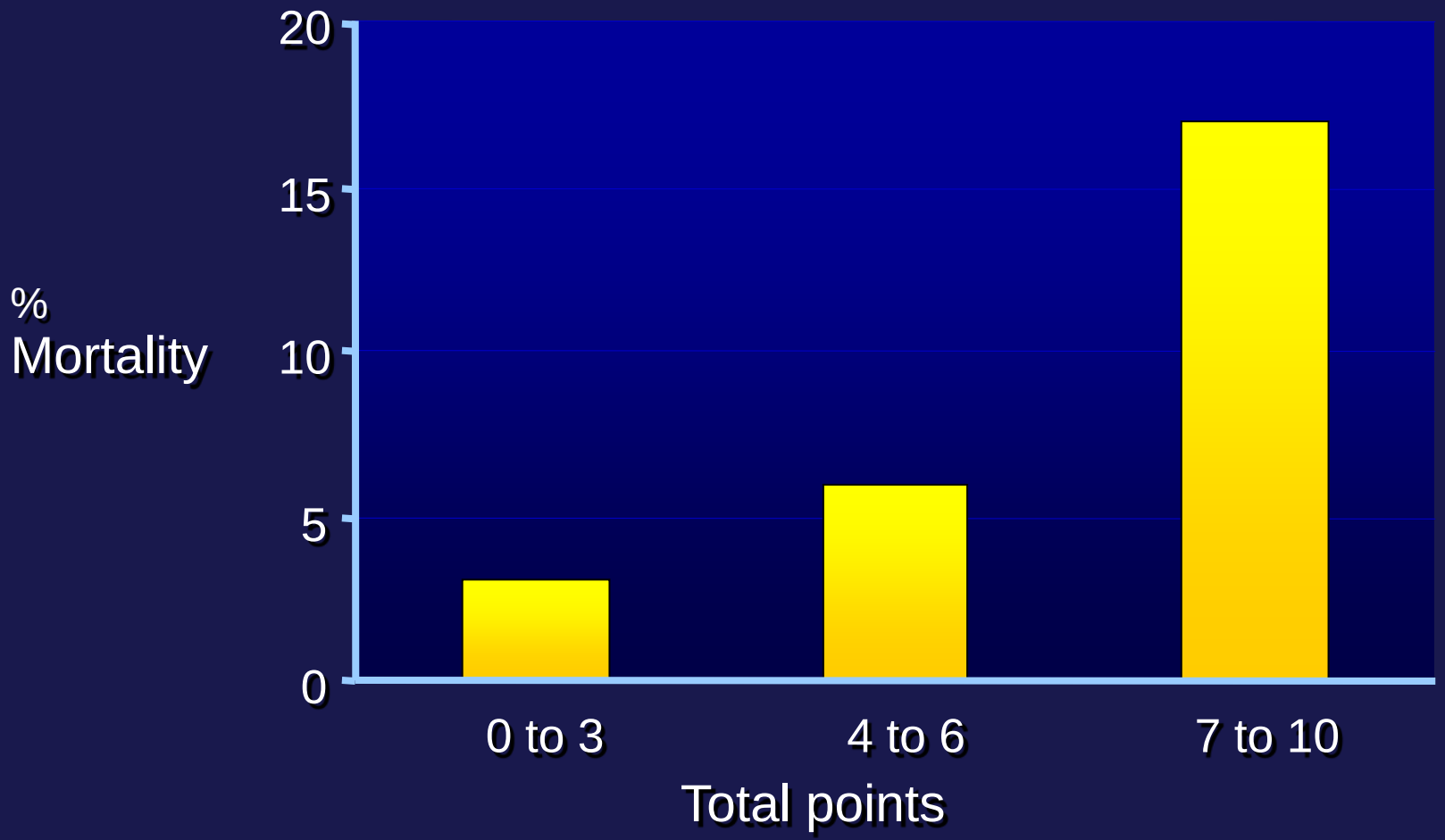


Hematocrit and Severity

Criteria	Incidence of Necrosis
Admission hematocrit >44% OR fails to fall over first 24 hours	50%
Neither present	4%

Brown J, et al., Pancreas 2000; 20:367

CT Findings and Mortality



Adapted from Balthazar, EJ Radiology 2002; 223:603

COMPLEXE CONSERVATIVE THERAPY

1. AGGRESSIVE INTENSIVE THERAPY IN ORDER TO CEASE PANCREATIC SHOCK BY HEMODYNAMIC RECOVERY, FLUID REPLACEMENT, CORRECTION OF ACID-BASE AND ELECTROLYTE IMBALANCE, SUPPLEMENTAL OXYGEN TO GUARANTEE OPTIMAL OXYGEN TRANSPORT

2. ELIMINATION OF ODDI SPASM AND MICROCIRCULATION DISTURBANCES

3. BLOCKING OF PANCREATIC SECRETORY FUNCTION

4. EXTRACORPORAL DETOXICATION

5. SEPTIC COMPLICATIONS PROPHYLAXIS AND TREATMENT

Treatment

Patient Management

- Initially, on confirmation of diagnosis admit in ICU
- General Management:
  - Fluid and electrolyte management by CVP line.
- Hourly urine out put
- Monitoring BP, pulse rate, O2 saturation , blood gas analysis to determine ventilatory support. Depending on cardiovascular changes arterial catheters.
- Strict asepsis
- Nursing

Treatment

Supportive care

- Aggressive fluid and electrolyte replacement
- Monitoring
  - Vital signs
  - Urine output
  - O_2 saturation
  - Pain
- Analgesia, anti-emetics

Other treatments

- Acid suppression
- Antibiotics
- NG tube
- Nutritional support
- Urgent ERCP

Naso-gastric Suction

Use for:

- Persistent vomiting
- Obstruction

Does not alter disease course

Fluid Resuscitation

- Maintaining adequate intravascular volume in severe disease may require 5-10 liters of fluid daily for the 1st few days.
- Experimentally: hemodilution to a hematocrit value of 30% with dextran 60 solution improved pancreatic microcirculation and oxygenation
- If hematocrit decreases to 25%, packed red blood cells should be infused, maintain hematocrit close to 30%.

Nutrition

- Mild acute pancreatitis
  - Allow oral fluids from day 1 until appetite returns
- Severe acute pancreatitis
  - Begin fluids as early as 72 hrs

Factors Supporting Early Enteral Nutrition

- Mucosal integrity is important in ASP
  - Glutamine, arginine, ω-3 fatty acids, nucleotides have important role in critically ill pts in maintenance of mucosal integrity and immune status

- Early parenteral nutrition harmful

Nutritional Support

Route of Alimentation

TPN

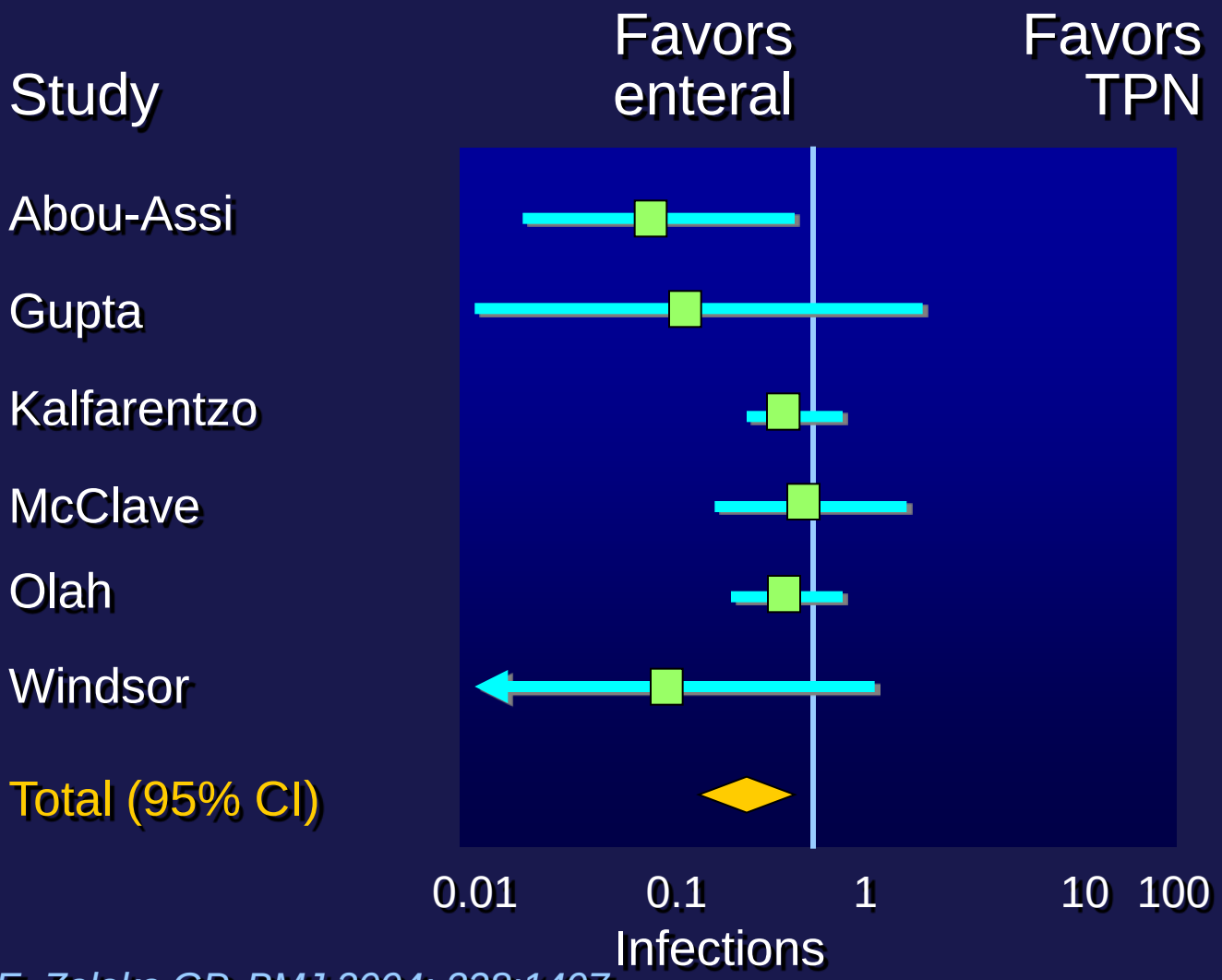
- Cost – high
- No pancreas stimulation
- Increased infections
- Electrolyte disturbances
- Detrimental to gut integrity

Enteral

- Cost – moderate
- May stimulate pancreas
- Reduced infections
- Electrolytes undisturbed
- May retain gut integrity

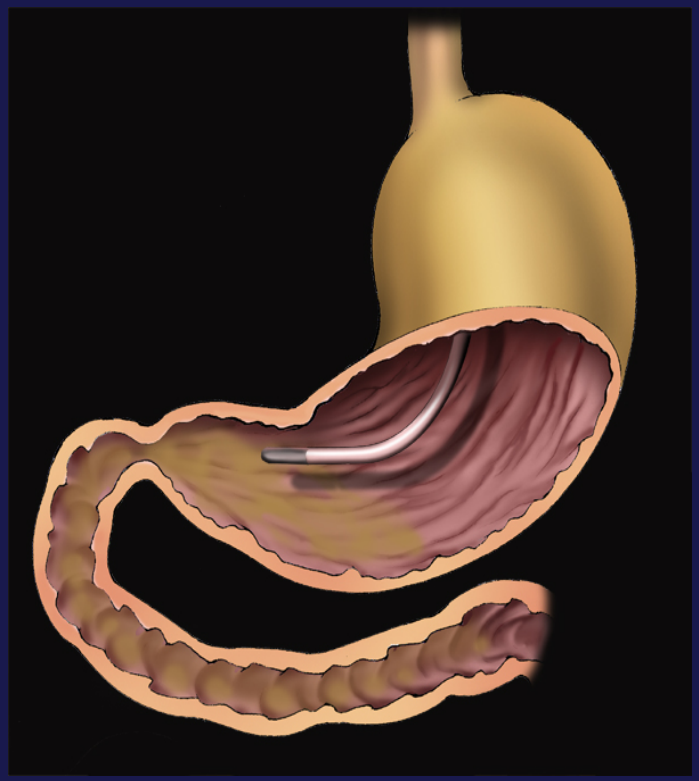
Acute Pancreatitis

Nutritional Support and Infection

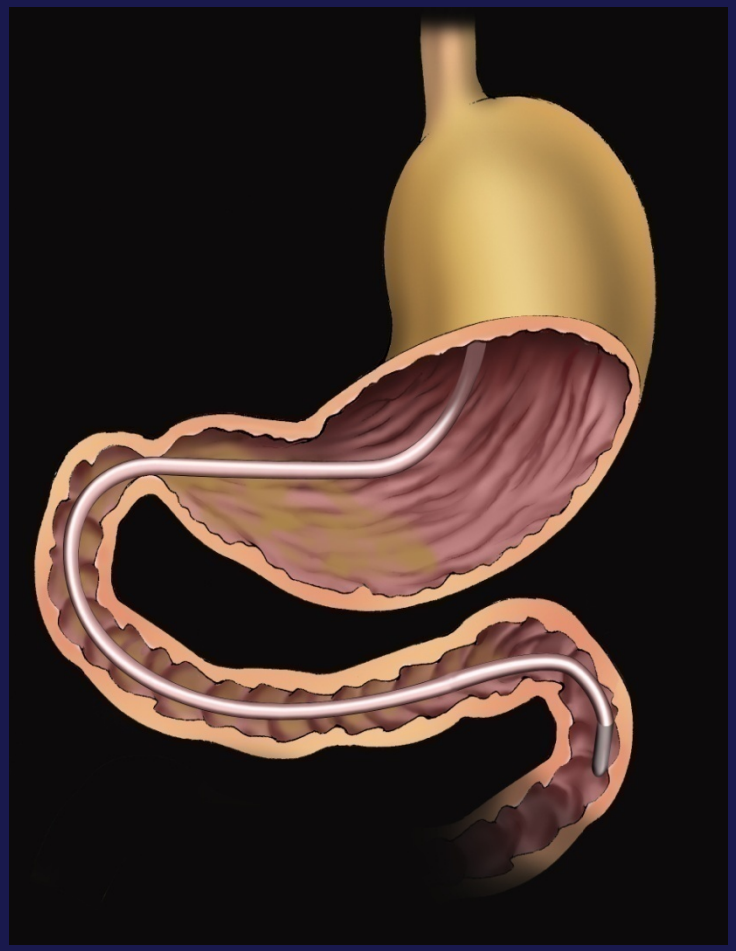


Marik PE, Zaloka GP. BMJ 2004; 328:1407

Enteral Feeding Sites



Nasogastric



Jejunal

Enteral Nutrition: Infusion Site

Gastric

- Easy placement
- Stable positioning
- Pancreatic stimulation probable
- Maybe tolerated in severe acute pancreatitis

Jejunal

- Difficult placement
- Frequent dislodgement
- Pancreatic stimulation unlikely
- Tolerated in severe acute pancreatitis

Outcomes may be similar

Eatok FC et al. AJG 2005; 432

Antibiotics

- Randomized trials: benefit for early initiation of broad-spectrum antibiotics- may prevent pancreatic infection
- Antibiotics with good pancreatic tissue penetration:
  - Imipenem (500 mg IV every 8 hours)
  - Cefuroxime (1.5 g IV every 8 hours)
  - Ciprofloxacin (400 mg IV every 12 hours)
- Potential drawbacks:
  - Development of resistant organisms
  - Fungal infections

Antibiotics

- Have been shown to decrease infection rates, but have not consistently demonstrated a mortality benefit.
- ACG guidelines: "in patients with necrotizing pancreatitis associated with organ failure, it is reasonable to initiate treatment with antibiotics with good spectrum of activity against aerobic and anaerobic bacteria."

Antibiotics

- Prophylactic antibiotics have shown no decrease in mortality in severe acute pancreatitis.

Conditions where antibiotics are justified are:

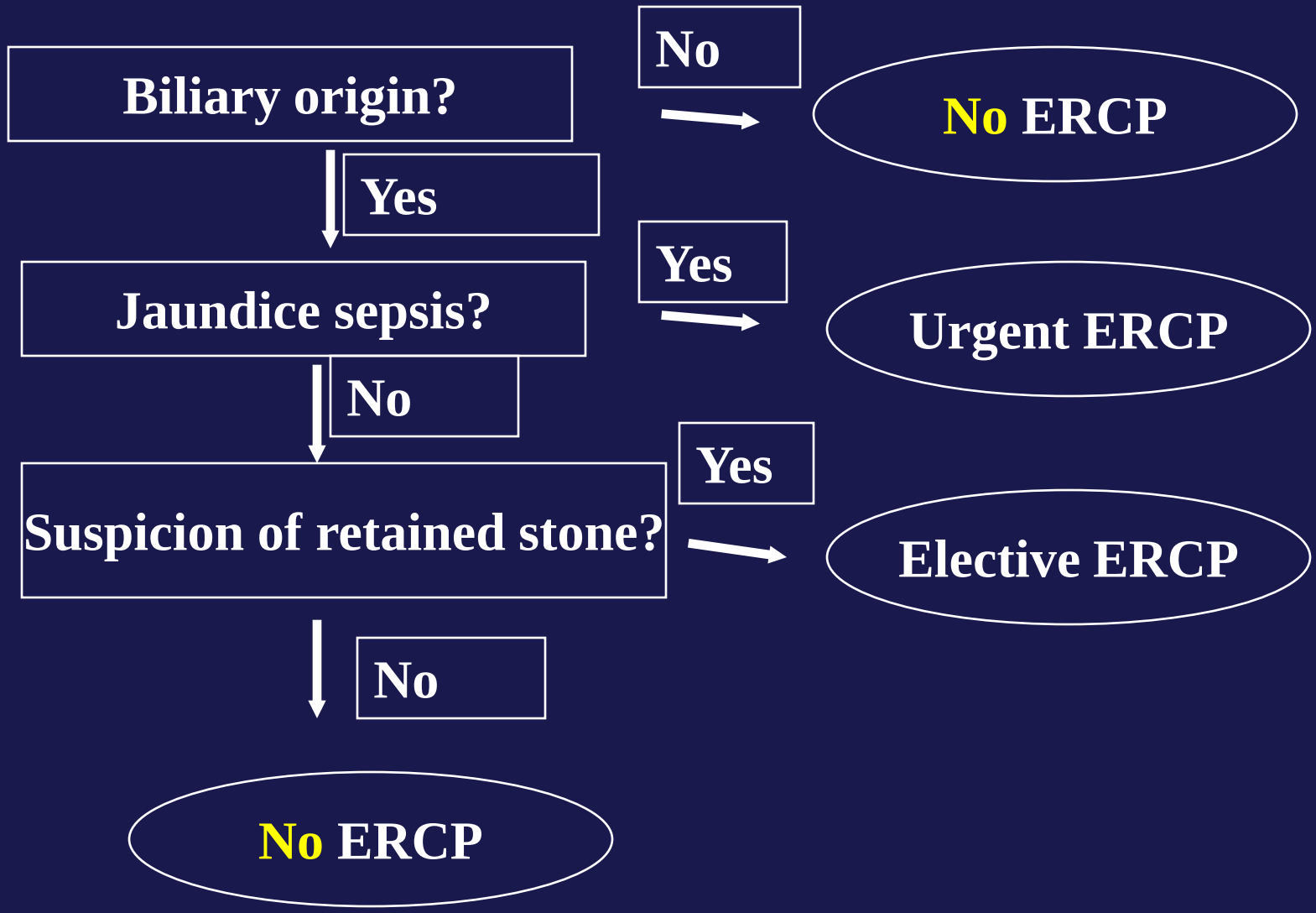
1. Gas in retroperitoneal space
2. Needle aspiration of necrotic material confirms infection
3. Presence of necrosis – 15 -> 50%
4. CRP of > 120 mg/L
5. Peripancreatic fluid collection
6. APACHE II score of > 6
7. Organ dysfunction

Endoscopy

- Urgent Removal of Gallstones in Gallstone Pancreatitis:

- Consensus that severe acute gallstone pancreatitis with ascending cholangitis (jaundice and fever) is an indication for urgent ERCP.
- In patients with acute biliary pancreatitis but without obstructive jaundice, early ERCP and papillotomy were not beneficial

Endoscopy


Biliary origin?
No
No ERCP
Yes
Jaundice sepsis?
Yes
Urgent ERCP
No
Suspicion of retained stone?
Yes
Elective ERCP
No
No ERCP

GALLSTONE PANCREATITIS
Timing of Cholecystectomy

- In mild pancreatitis, it is advisable to undertake laparoscopic cholecystectomy with intraoperative cholangiogram within 10 days.
- In severe cases, endoscopic cholangiopancreatography + endoscopic sphincterotomy(<48h) followed by LC once the inflammatory process is resolved.

Surgery for Acute Pancreatitis

Role of Surgery

- Surgery has no immediate role in patients with mild acute pancreatitis.
- The development of infected pancreatic necrosis is an indication for intervention, with surgery or an alternative technique
- Early surgery (within the first 14 days) should be avoided because it is associated with increased mortality.

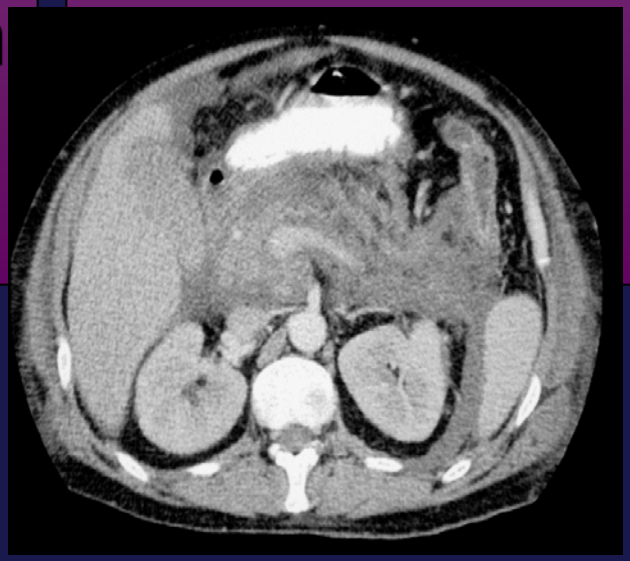
Treatment Strategies

Sterile

- Medical therapy
- Debridement for persistent organ failure?

Infected

- Antibiotics
- Debridement



Management of necrosis

- Sterile necrosis does not usually require therapy.
- The development of infected necrosis should be suspected in those patients with preexisting sterile pancreatic necrosis who have persistent or worsening symptoms or symptoms and signs of infection, typically after 7–10 days of illness.
- The finding of gas within the pancreas in CECT is highly suggestive, although not diagnostic, of infected necrosis
- Fine-needle aspiration guided by CT imaging should be performed and the sample should be cultured and Gram stained to document infection

Management of necrosis

- The standard approach to infected necrosis has been open surgical debridement.
- Increasing trend to delay surgery as long as possible, even in the face of a positive result on FNA, if the clinical situation allows.
- This delay has the advantage of allowing necrotic material to demarcate and begin to liquefy, making complete initial necrosectomy more likely, and reducing the need for repeated debridement.
- The delay-until liquefaction strategy also allows nonsurgical therapies

Indication for Surgical Management of Necrotizing Pancreatitis and Pancreatic Abscess

Clinical Criteria

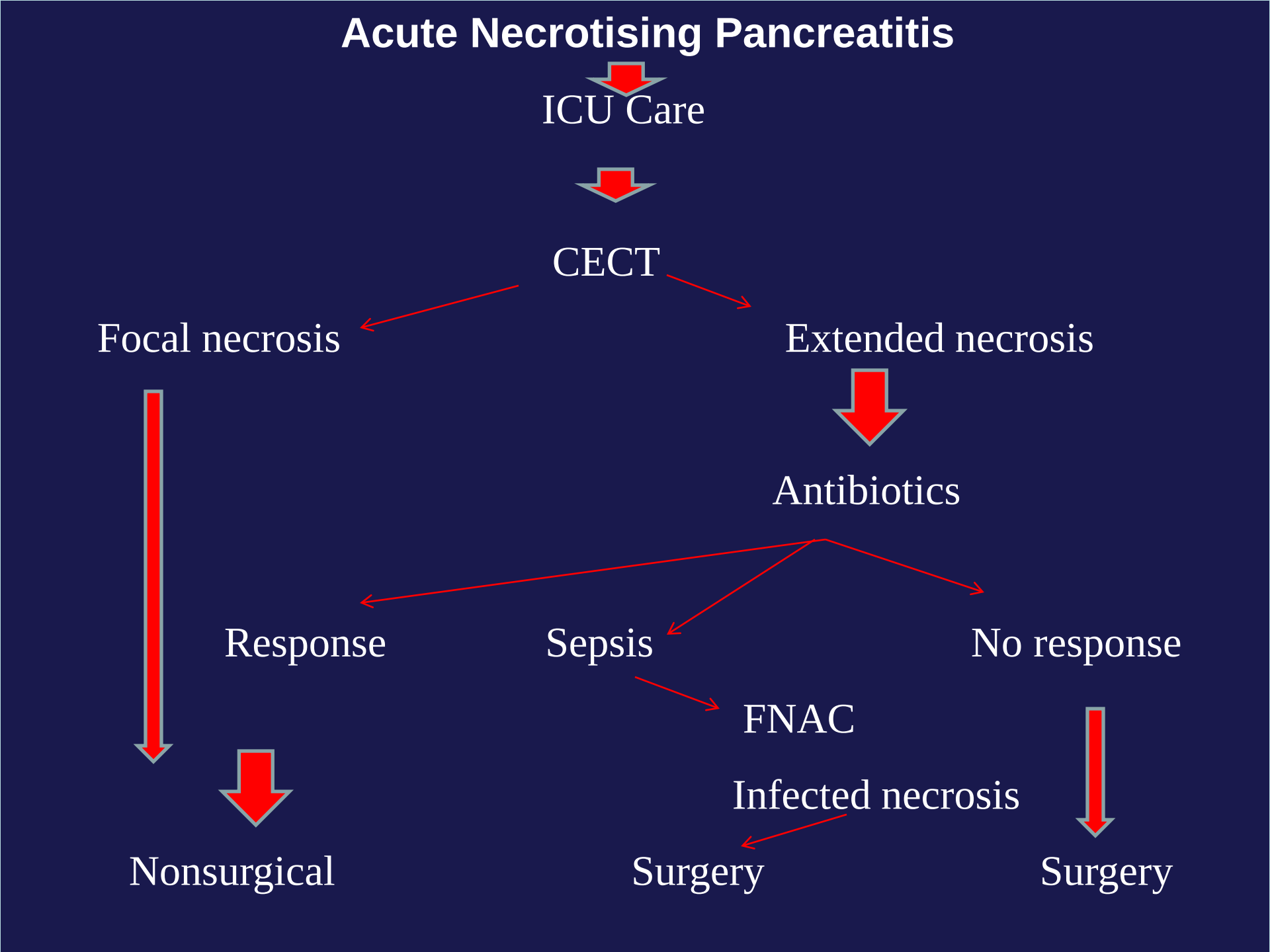
- Persistant sepsis
- No response to intensive care treatment (> 3 days),
- Persisting or increasing local or systemic complications

Surgery in necrotising pancreatitis

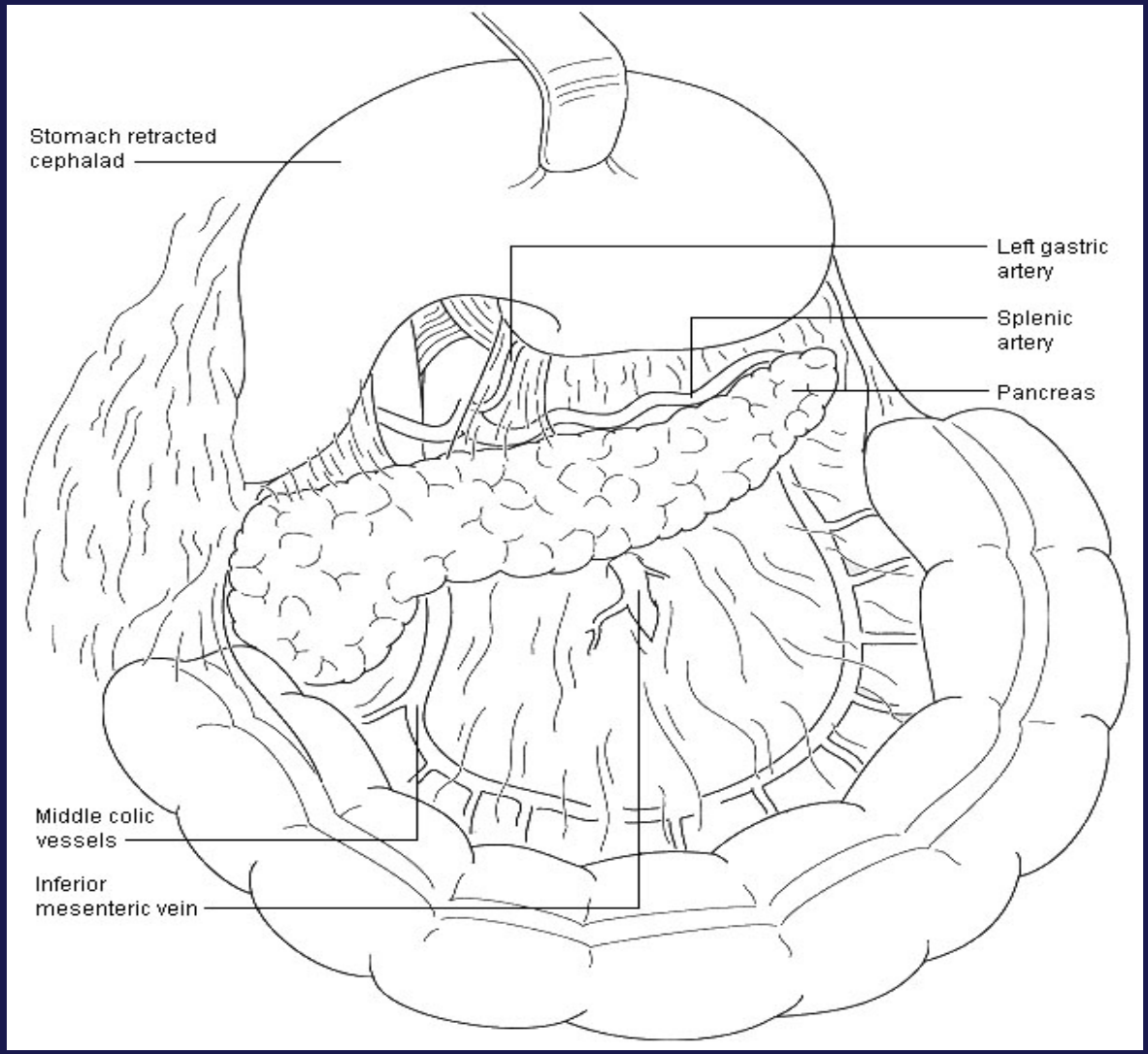
- Debridement with closure over drains
- Debridement with open packing
- Debridement with closure over irrigation drains and postoperative lavage.(Beger surgery)
- Minimally invasive surgery

Timing of Surgery

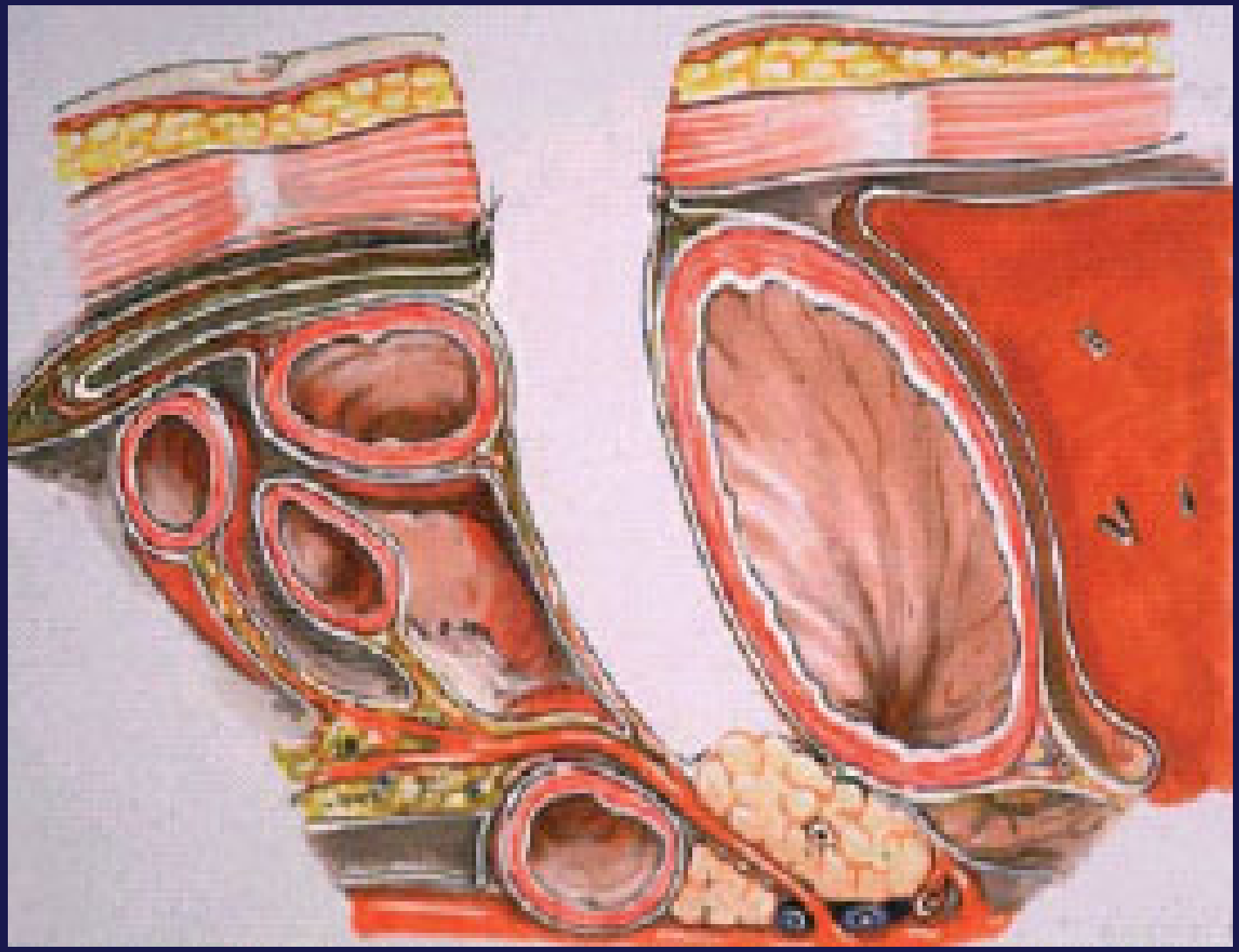
- Delaying surgery till the second to third week from the onset of disease is ideal
- Early intervention is unavoidable
  - In the presence of uncertain diagnosis.
  - Complications like hemorrhage associated with pancreatic necrosis


Acute Necrotising Pancreatitis
ICU Care
CECT
Focal necrosis
Extended necrosis
Antibiotics
Response
Sepsis
No response
FNAC
Infected necrosis
Nonsurgical
Surgery
Surgery

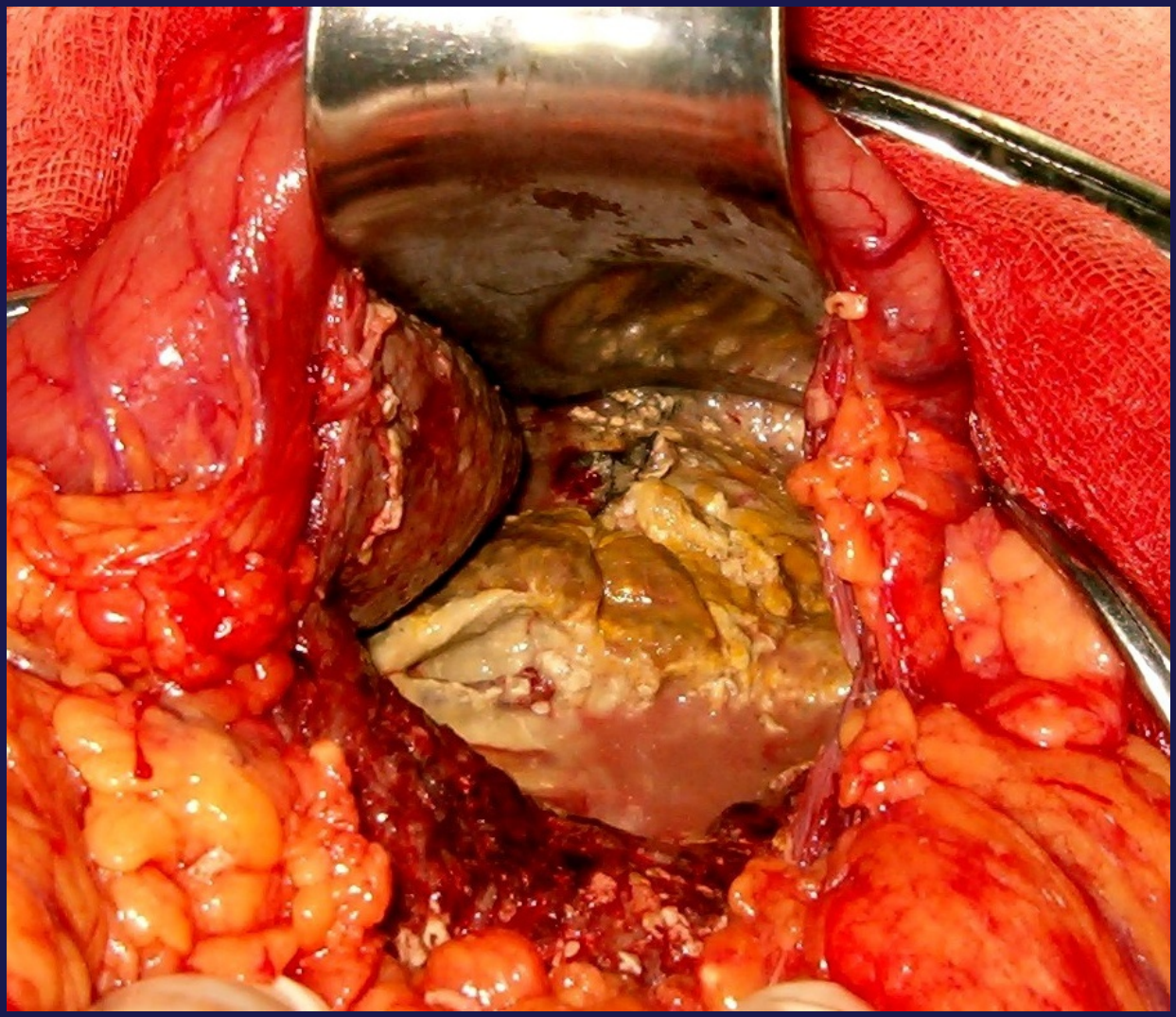
SURGICAL TREATMENT



SURGICAL TREATMENT

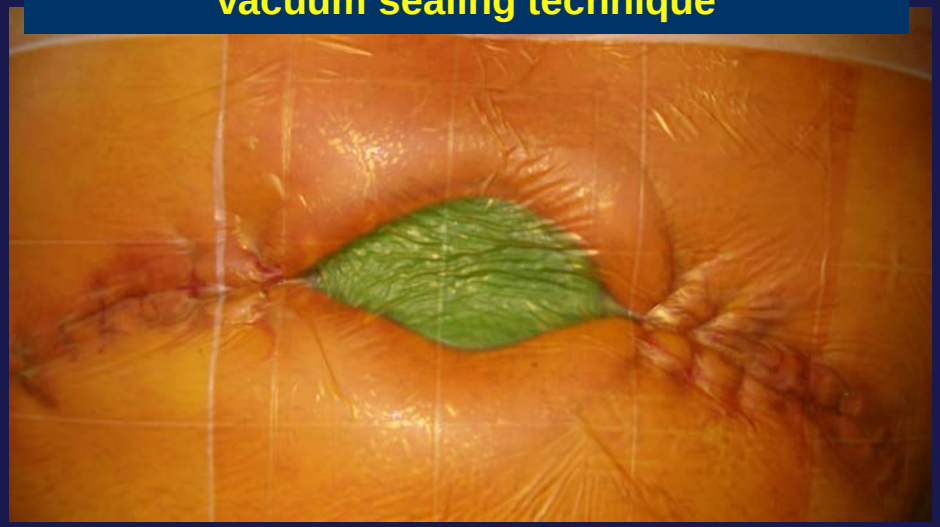


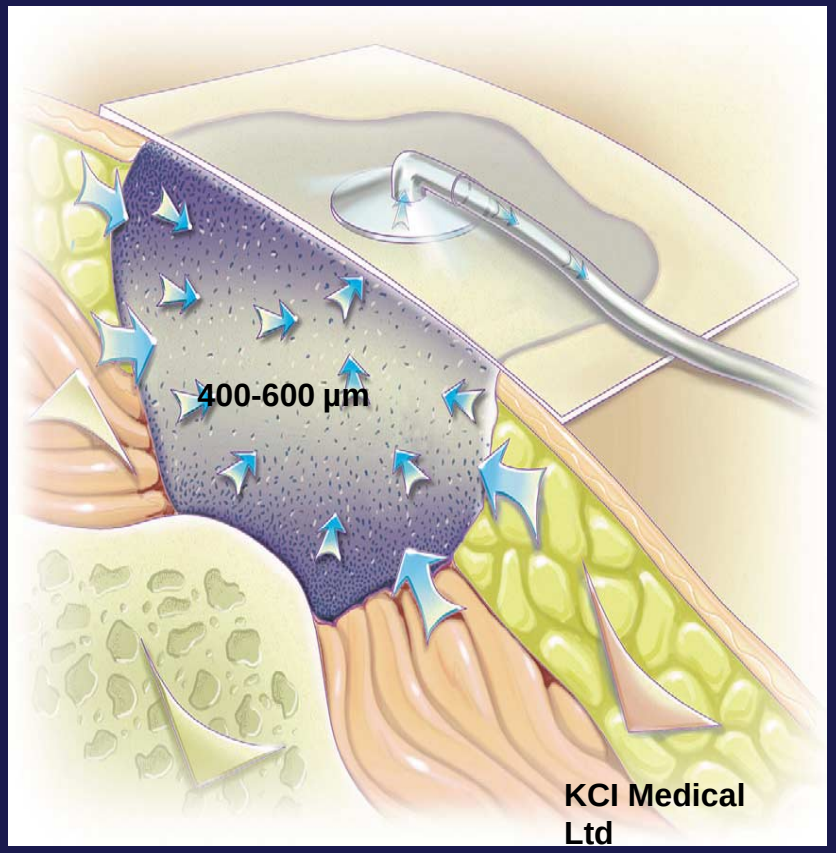
SURGICAL TREATMENT



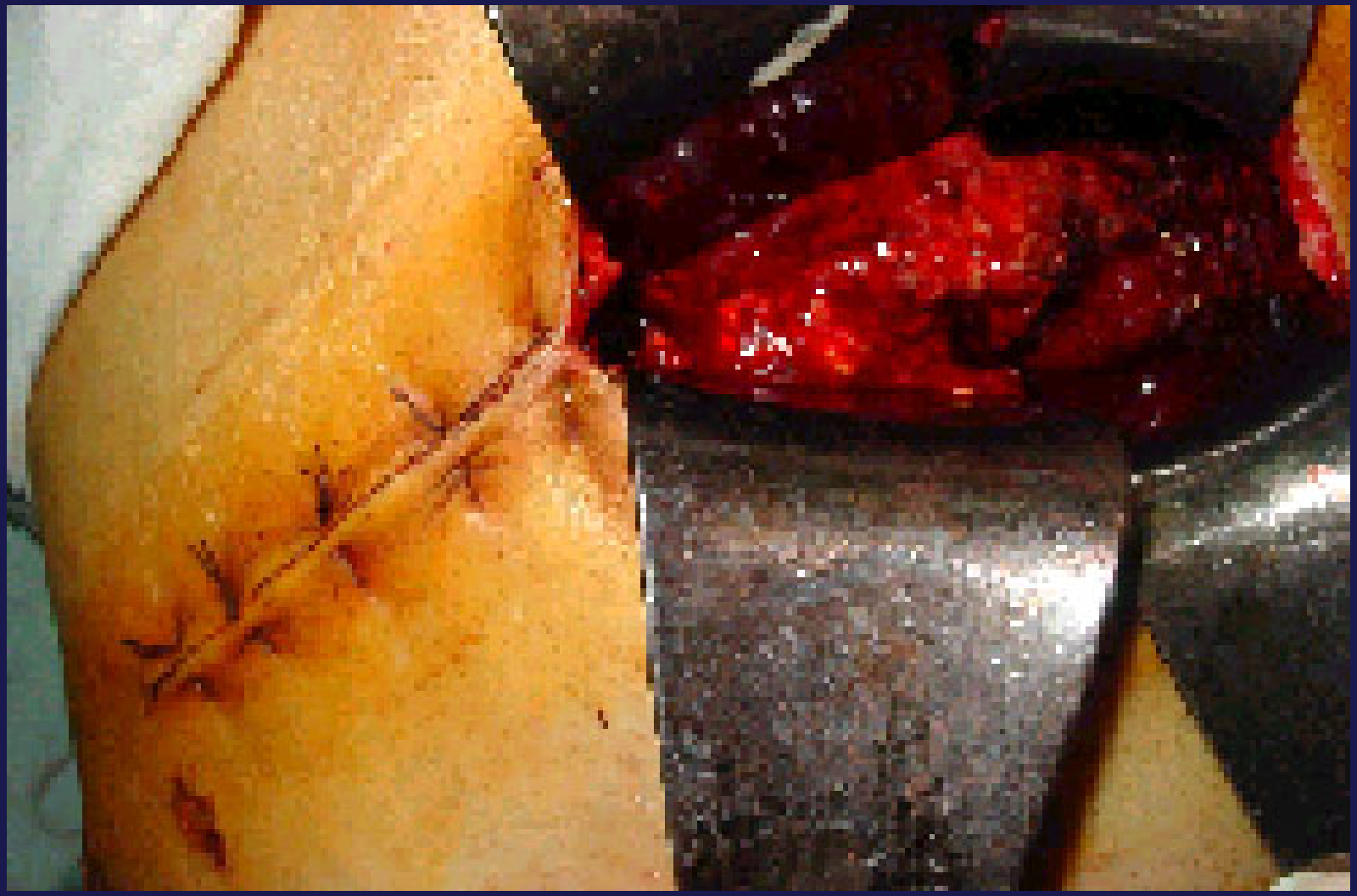
VIEW OF LESSER SAC THROUGH BURSOOMENTOSTOMY IN NECROTIZED PANCREATITIS (FIRST DEBRIDMENT)

Open-package of the lesser sac using vacuum sealing technique

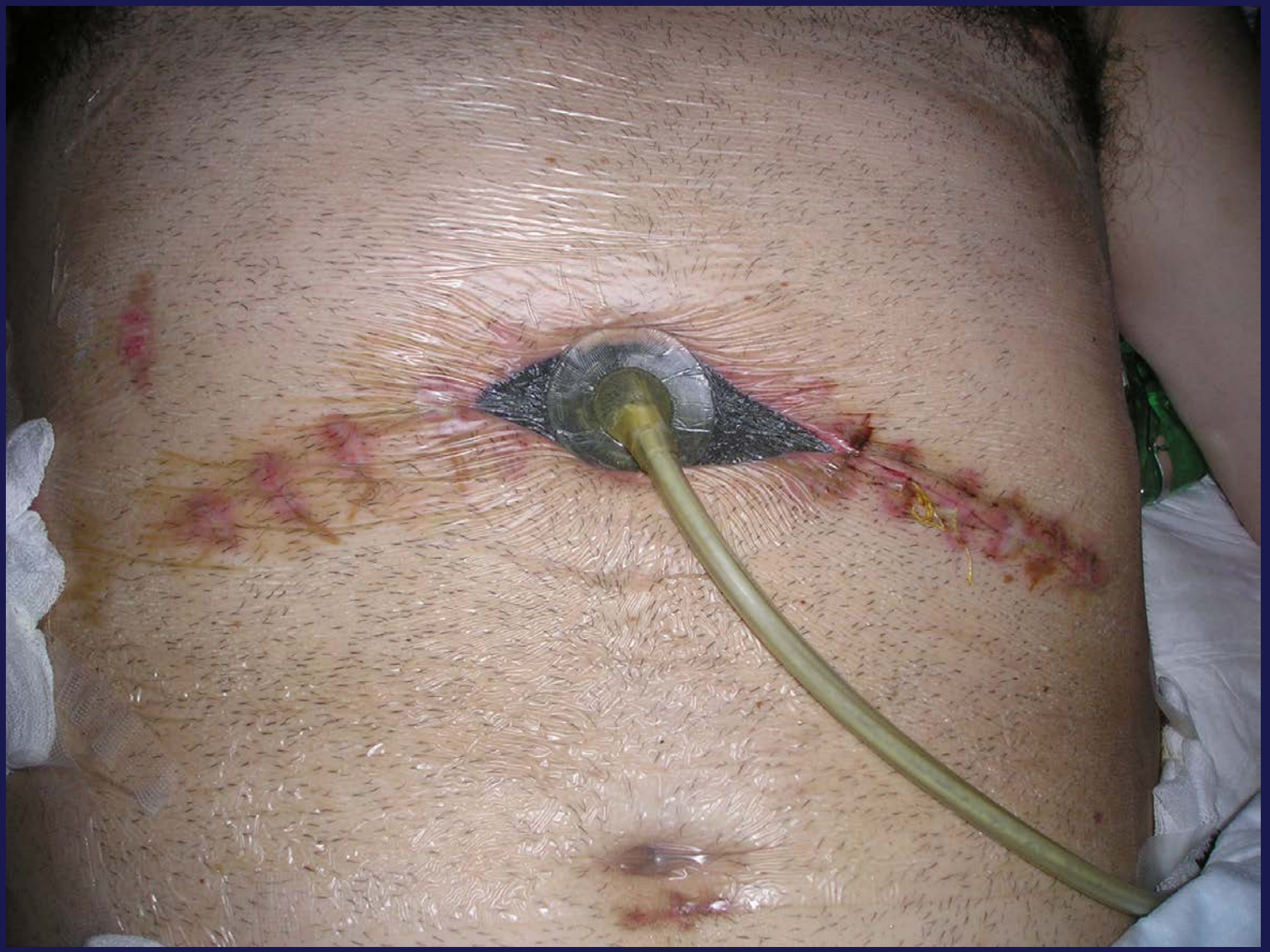




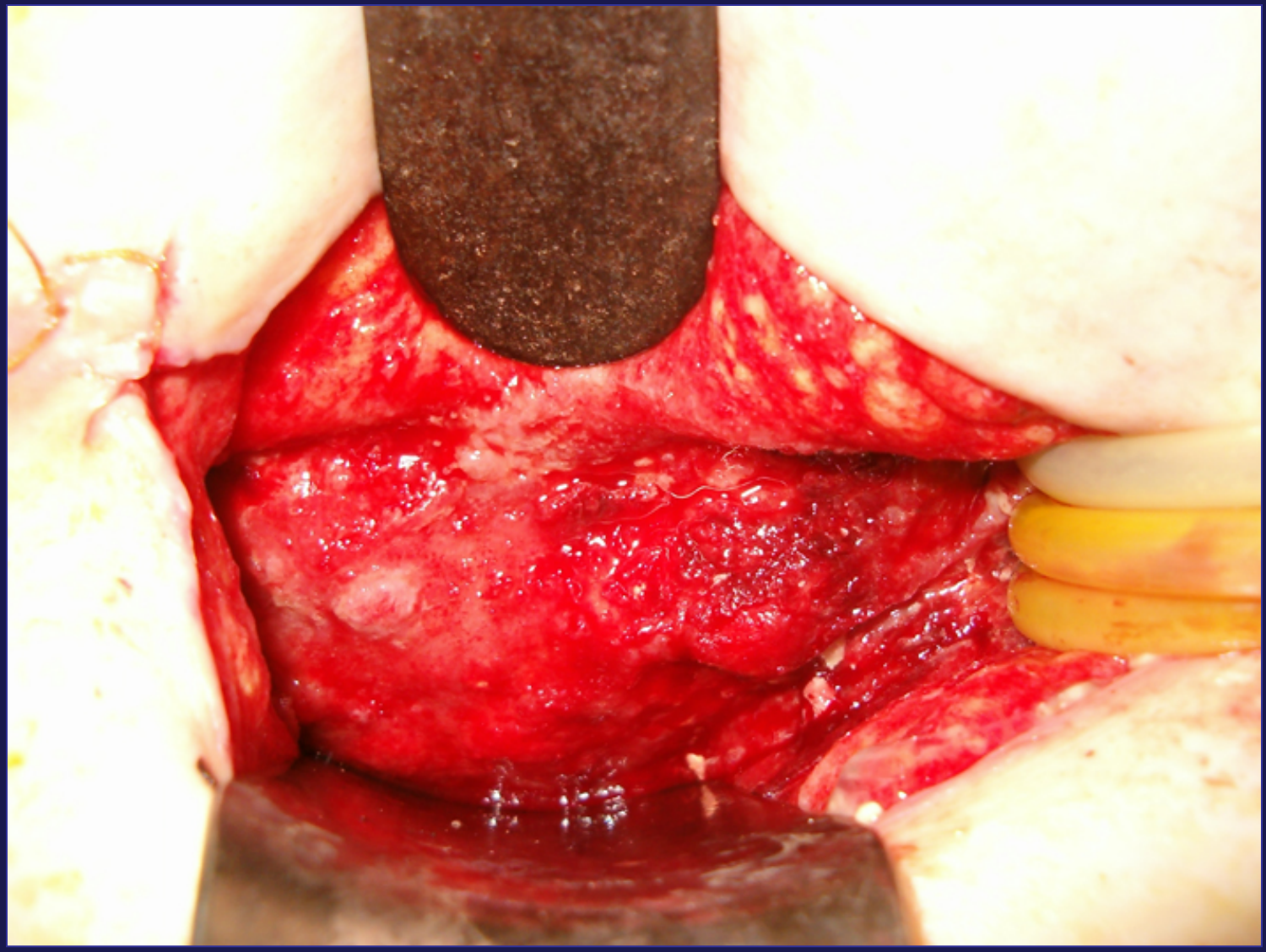
Dressing *in situ*



VIEW OF LESSER SAC THROUGH BURSOOMENTOSTOMY IN NECROTIZED PANCREATITIS (SECOND DEBRIDMENT)



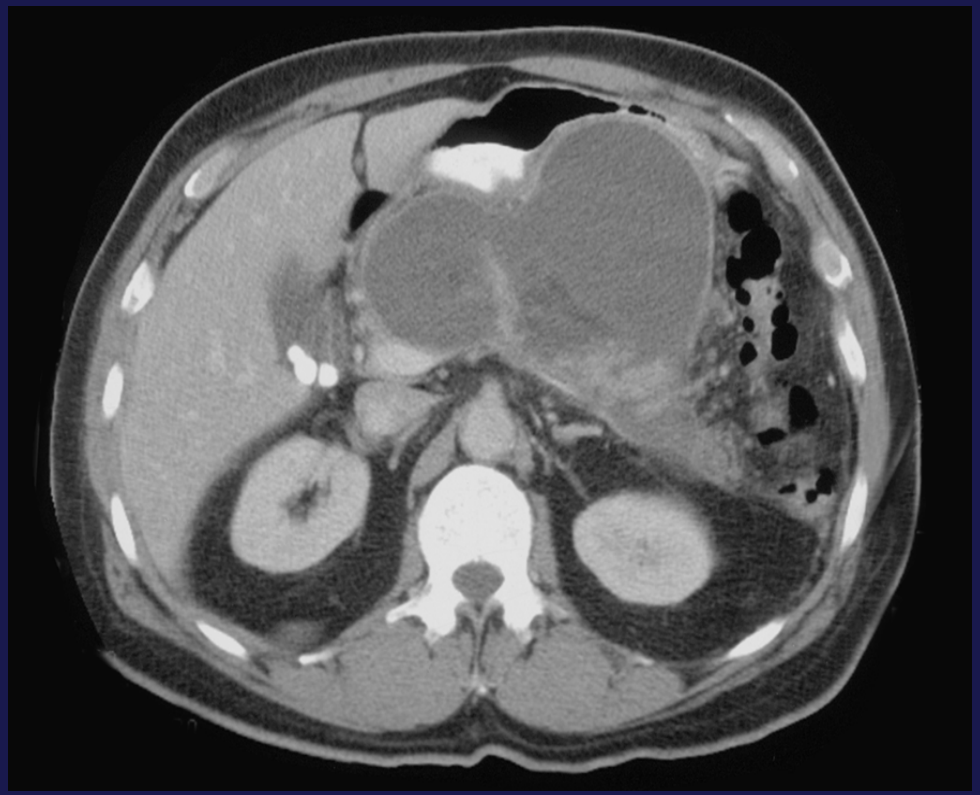
The original V.A.C. system *in situ*



VIEW OF LESSER SAC THROUGH BURSOOMENTOSTOMY IN NECROTIZED PANCREATITIS (BEFORE CLOSURE)

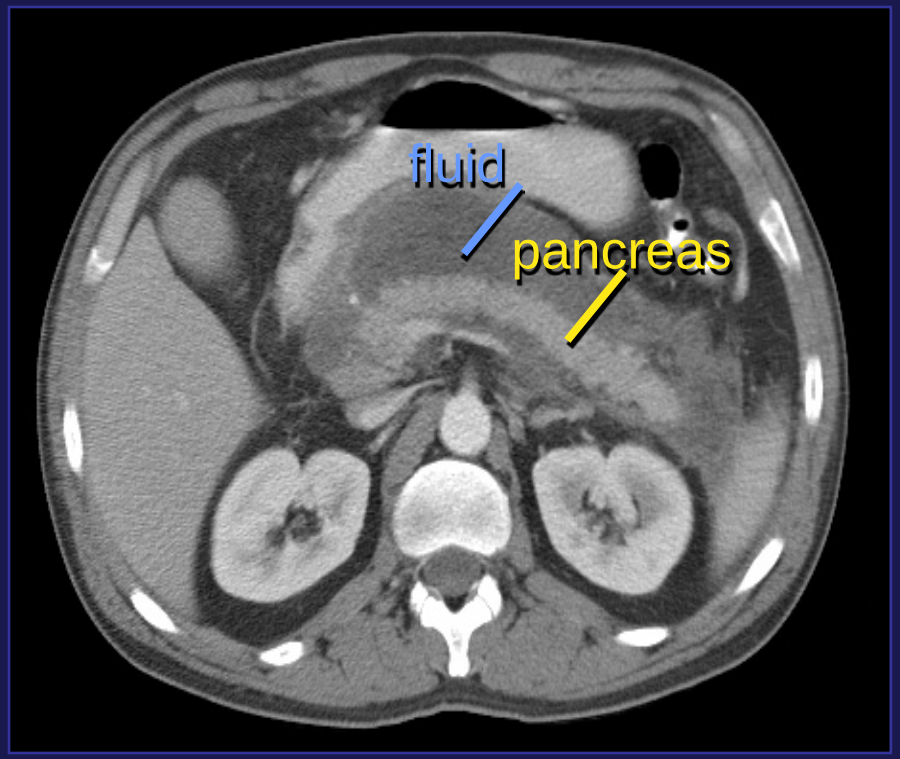
Fluid Collections in Acute Pancreatitis

- Acute collections
- Necrosis
- Pseudocysts
- Abscesses



- Acute Fluid Collections
  - Develops in 30-50% of patients with severe pancreatitis
  - Usually peripancreatic or intrapancreatic
  - Unlike psuedocyst lacks a wall
  - Either regresses or evolves into psuedocyst

Acute Fluid Collections



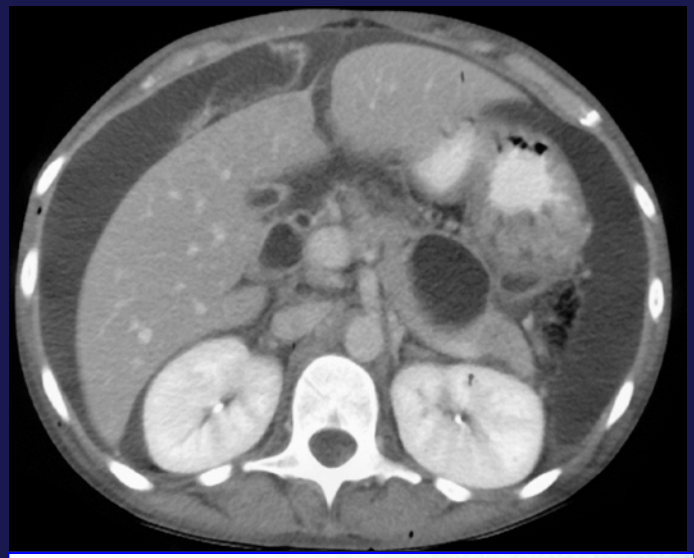
- Common
- May be complex
- Usually resolve spontaneously
- Drain if infected or symptomatic

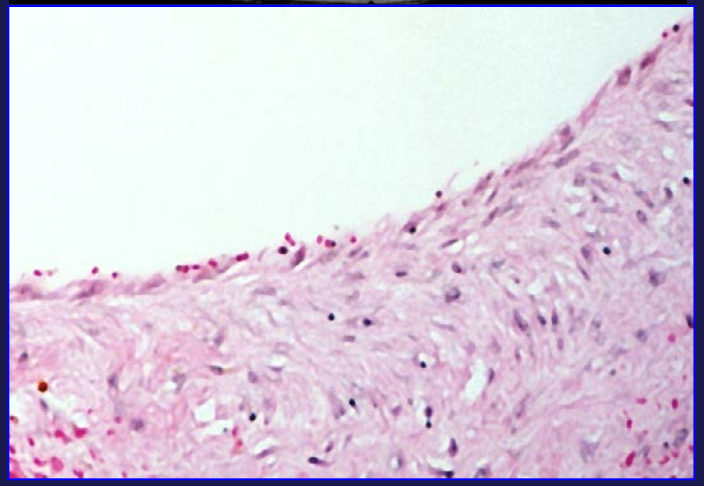
Pancreatic fluid collections and pseudocyst

- Acute fluid collections around the pancreas in the setting of acute pancreatitis require no therapy in the absence of infection or obstruction of a surrounding hollow viscus
- Approximately half of these fluid collections will resolve within 6 weeks, and up to 15% will persist as encapsulated pseudocysts
- Pseudocysts can be managed conservatively, particularly if they are small (6 cm) and asymptomatic

- **Psuedocyst of Pancreas**
- **Features**
  - Cyst wall lacks epithelial lining. It is predominantly formed by granulation tissue and fibrosis.
  - Most psuedocysts communicate with ductal system.
  - Persistently raised S.amylase levels
  - Most cases regress by themselves

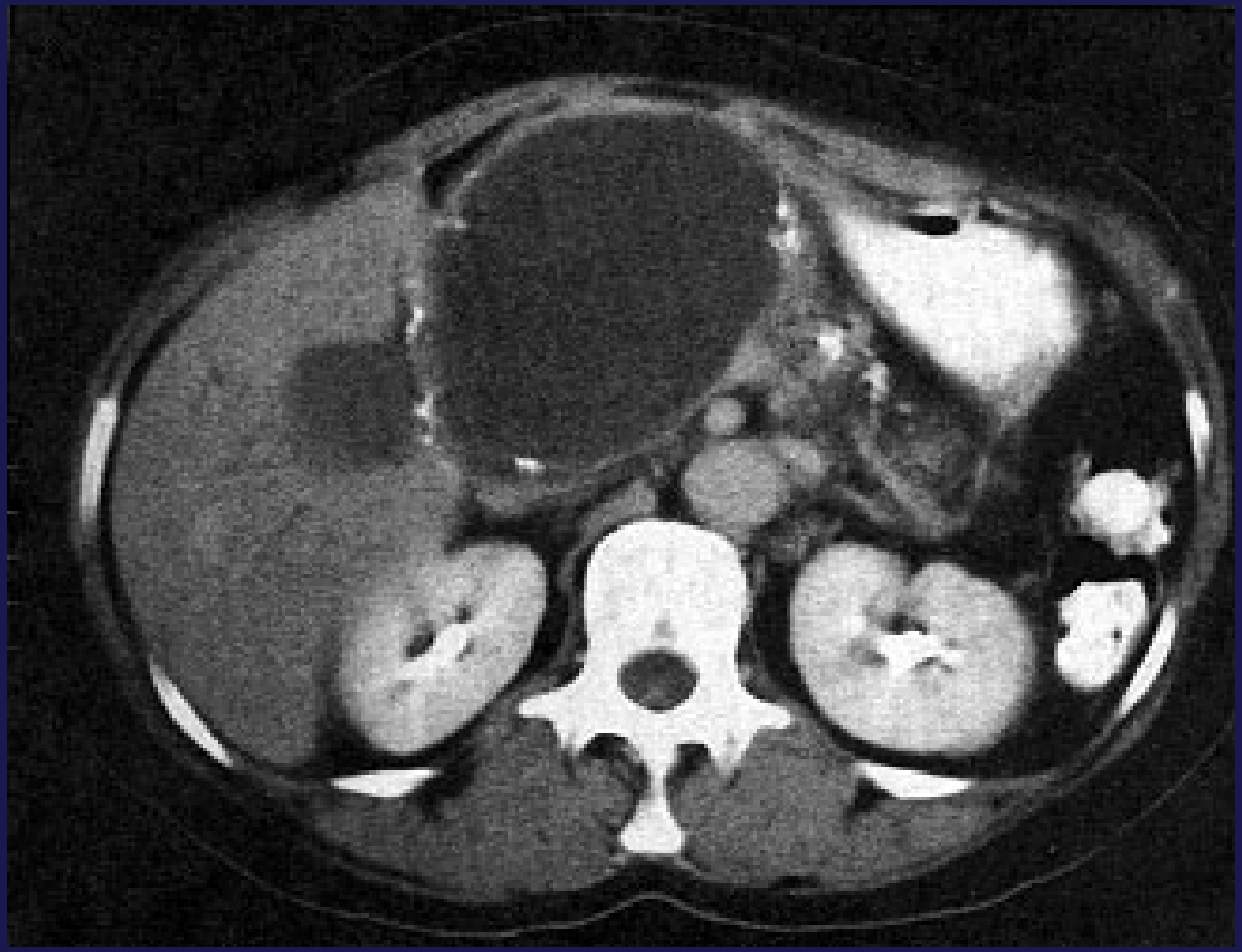
Pseudocysts





- Localized collections
- > 4 weeks after disease onset
- Ductal disruption or previous necrosis
- Not lined by epithelium

PANCREATIC PSEUDOCYST ON CT-SCAN



Pseudocysts

- Pseudocysts may produce symptoms (generally abdominal pain), obstruct surrounding organs (duodenum, stomach, or bile duct), become infected, rupture, or bleed
- Surgical, radiologic, and endoscopic options are available for the management of large or symptomatic or complicated pseudocysts.
- The choice of approach depends on location, size, pancreatic ductal anatomy, and, most importantly, local expertise

Intervention in Pseudocyst

- **Symptomatic**
- **Enlarging**
- **Size > 6 cm**
- **Duration more than 6 weeks**
- **Infected pseudocysts**
- **Complications due to pressure symptoms**
  - **GOO/obstructive jaundice**
- ***Haemosuccus pancreaticus***

Pseudocyst Management

Pseudocyst

Asymptomatic

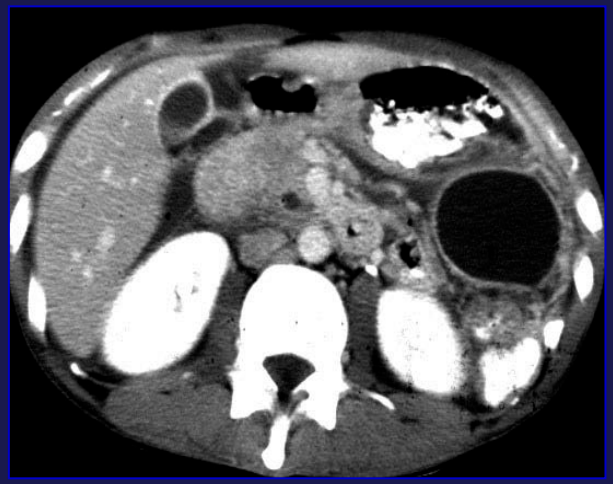
Symptomatic
Rapidly enlarging
Complication

Follow

Persists
6 - 12 months

Drain

* Observe

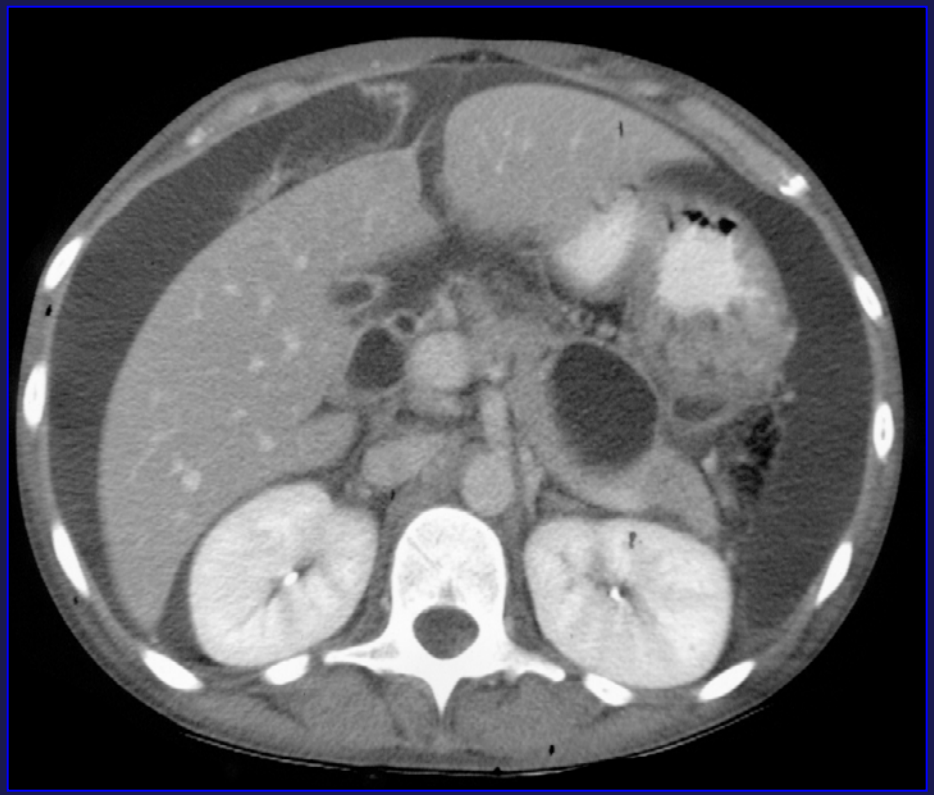


Drain

*Large cysts can be safely followed, but are more likely to require drainage

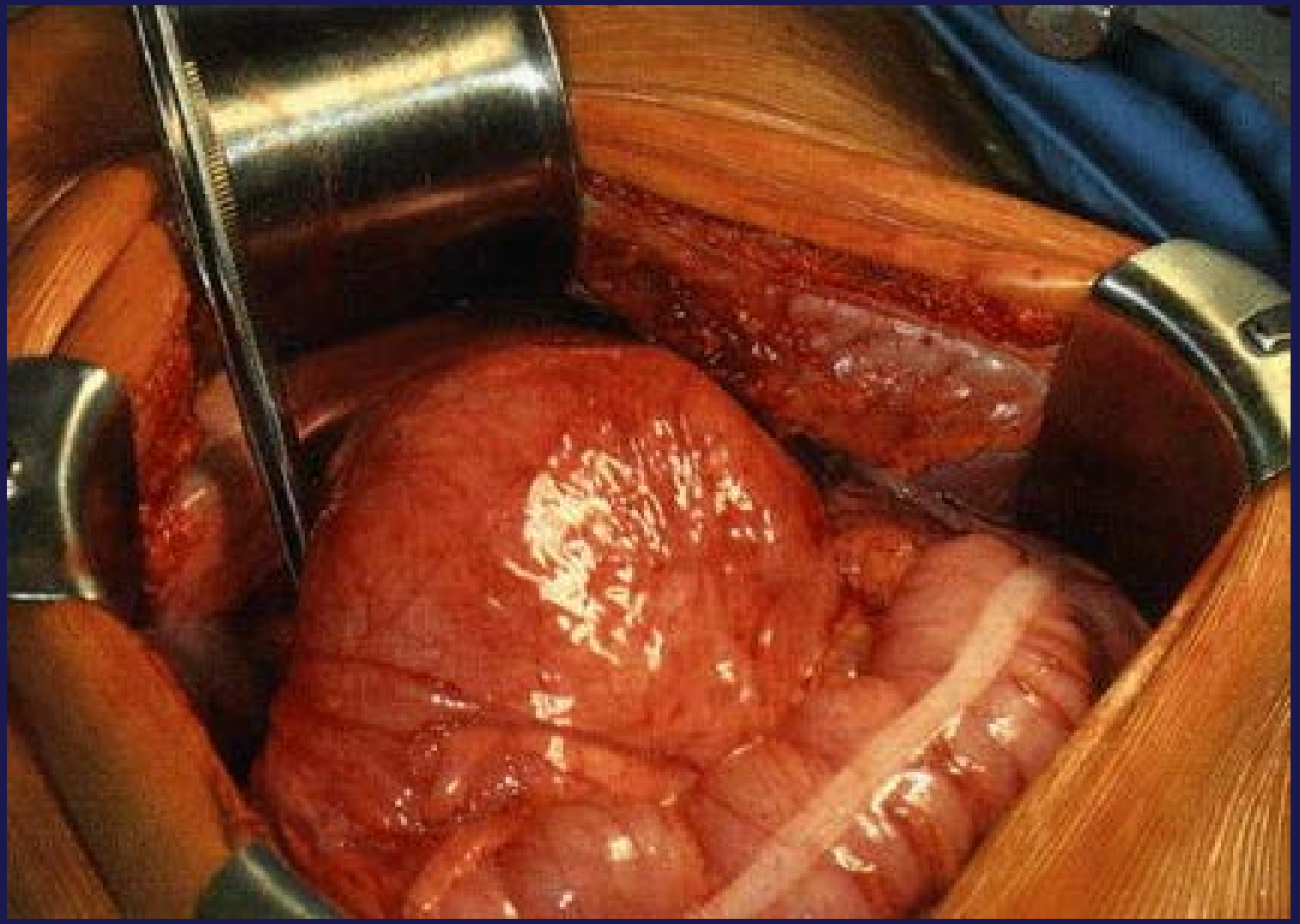
Pseudocyst Treatment

- Aspiration
- Internal drainage
- External drainage
- Trans-papillary drainage

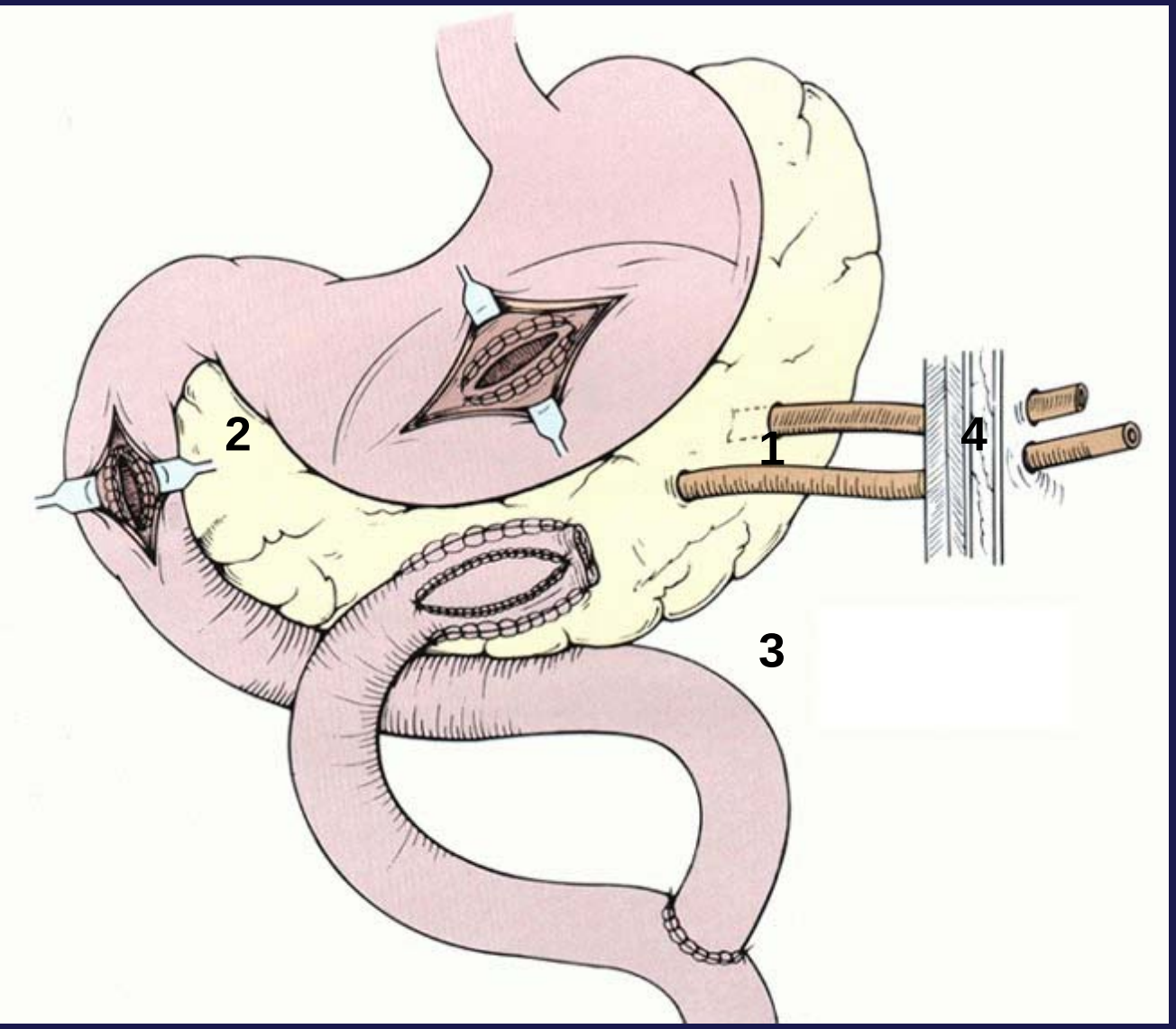


Pseudocyst Treatment

- **Percutaneous drainage - for poor risk patients only**
- **Open procedures**
  - **Distal pancreatectomy**
  - **Cystogastrostomy**
  - **Cystoduodenostomy**
  - **Roux – en – y cystojejunostomy**
  - **Whipple procedure**
- **Endoscopic procedures – stenting**
- **Laparoscopic procedures**



DRAINAGE PROCEDURES IN PSEUDOCYSTS



1. CYSTOGASTROSTOMY
2. CYSTODUODENOSTOMY
3. CYSTOJEJUNOSTOMY
4. EXTERNAL DRAINAGE

Pseudocyst Drainage

	Pros	Cons
Aspiration	Easily performed; minimally invasive	Often recur; risk fistula
Endoscopic Internal	Easily performed; moderately effective	Technical expertise; location of cyst
Surgical Internal	Most effective	Requires surgery
External stents	Easily performed; drain multiple cysts	Risk of fistulae
Trans-papillary	Moderately effective	Limited to lesions in pancreatic head

Summary

- S. lipase is more useful than amylase in diagnosing acute pancreatitis
- Prognosticate the patient
- Single and multiple prognostic factors can be used
- Identify acute mild and severe pancreatitis
- Timely resuscitation and invasive monitoring are standard
- No role for nasogastric tube

Summary

- Early enteral feeding
- Immune enhancing feeds has a role
- Prophylactic antibiotics for selected cases
- Early ERCP in severe biliary pancreatitis
- Surgery in selected cases of necrotising pancreatitis
- Delayed surgery is ideal unless indicated for early surgery

Summary

- For patients needing debridement, open surgical techniques remain the "gold standard" of management
- Debridement with open packing and lavage in early cases
- Debridement with closed drainage in elective cases
- Advances in minimally invasive technology hold promise as adjuncts to open procedures in the future

Thanks