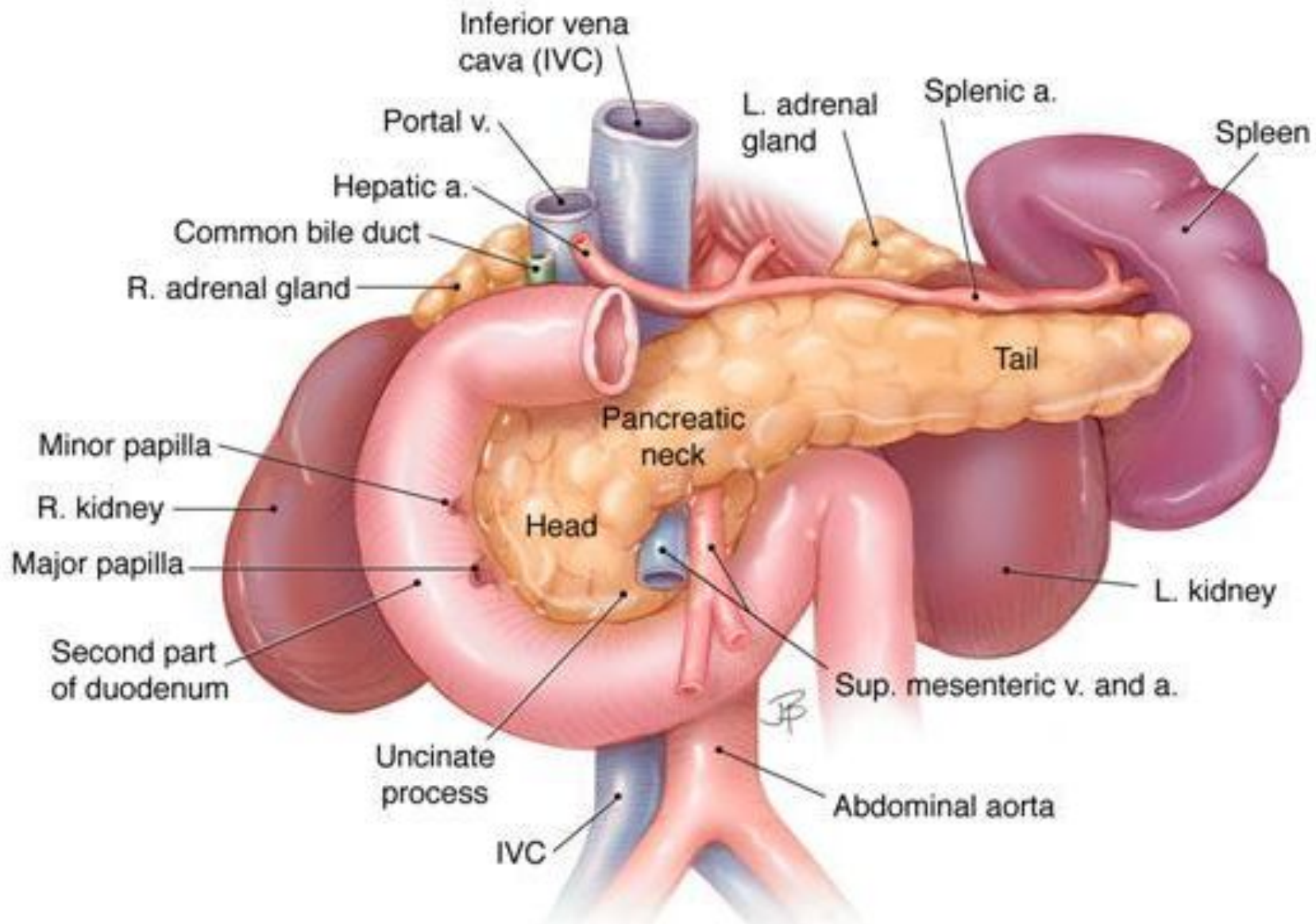
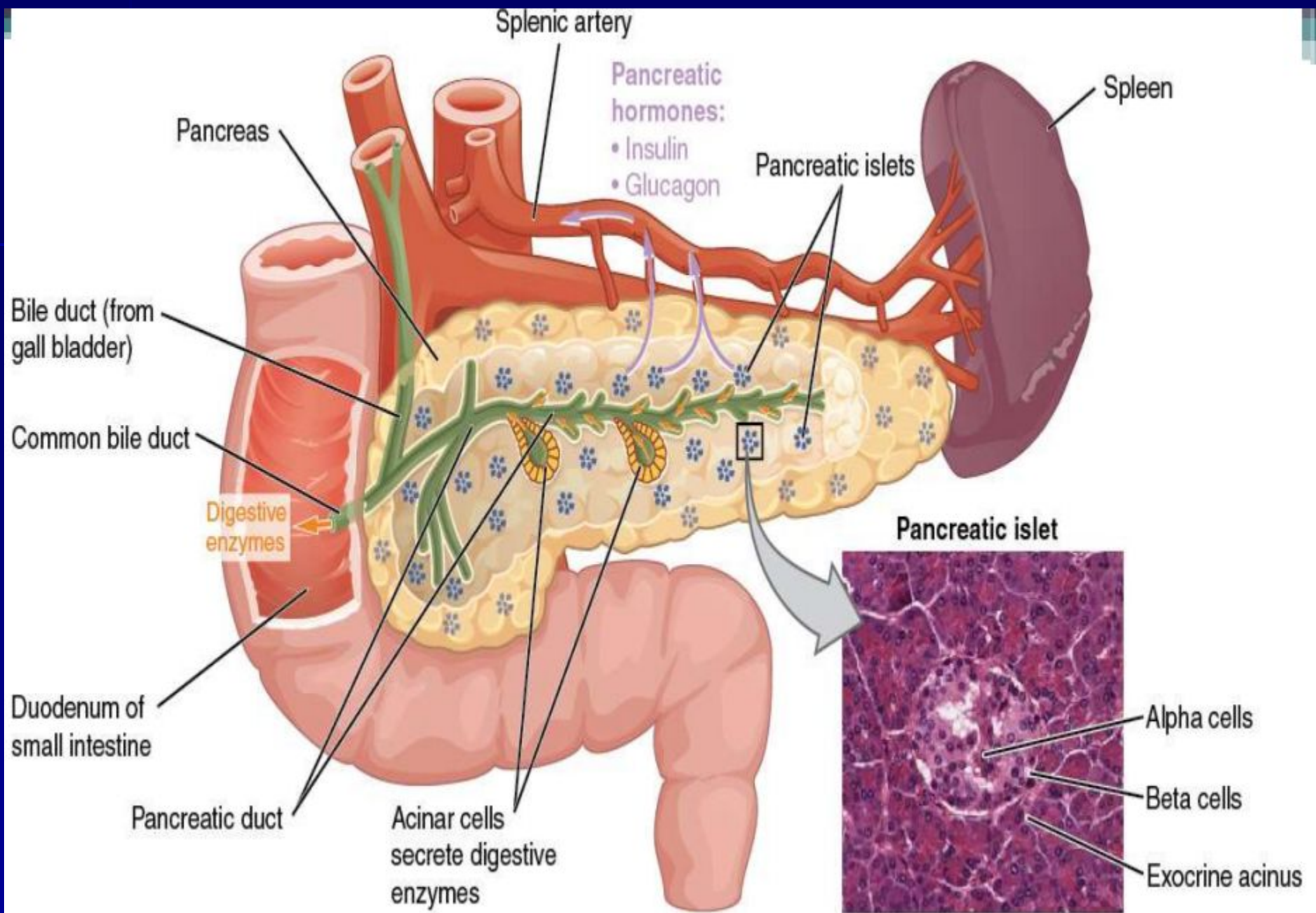
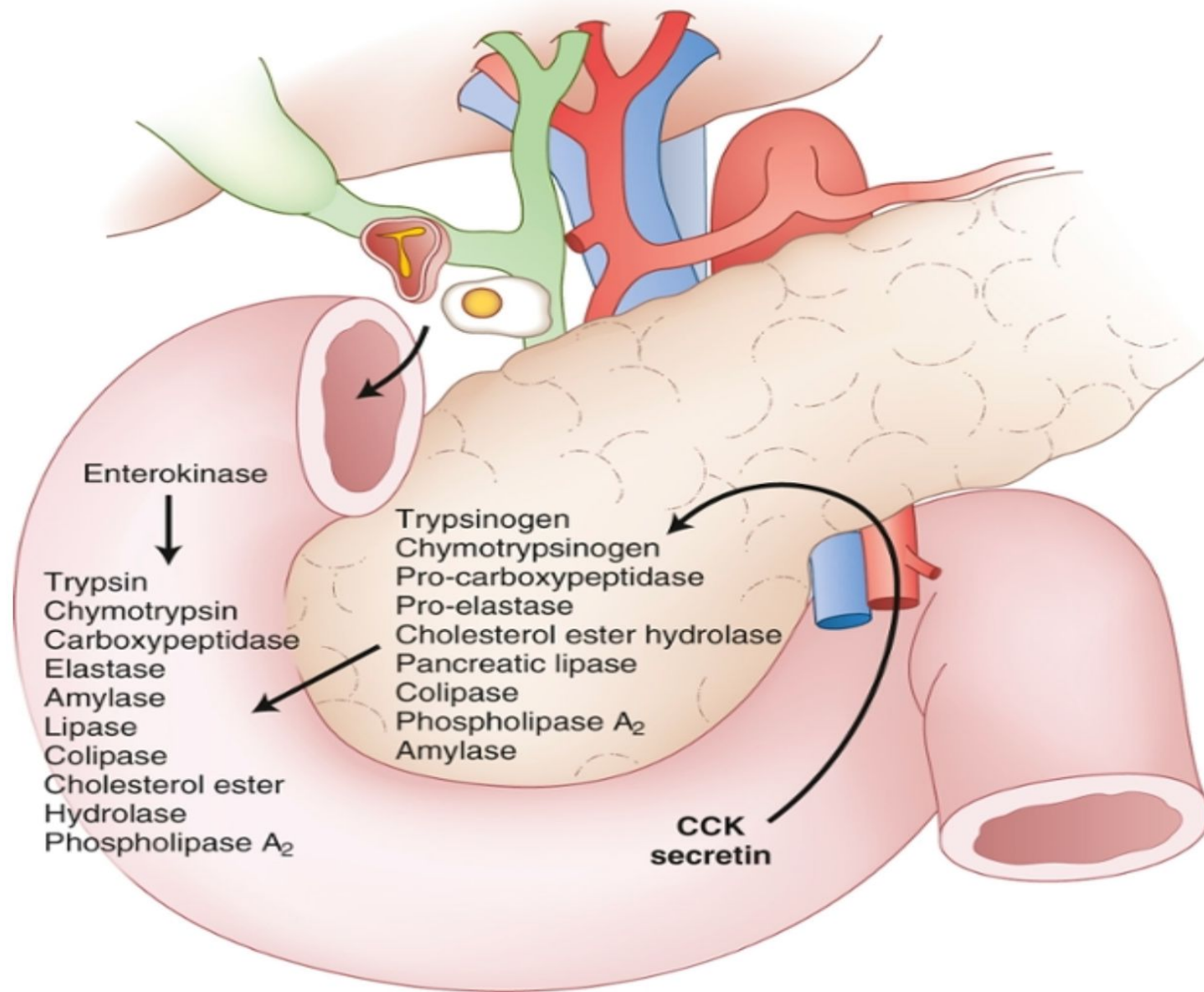


Acute Pancreatitis

E J Arteen
FRCSI







The Pancreas

Gallstone lodges in duct
blocking lumen and
aggravating
pancreas

Gallstone
in duct

Inflamed
pancreas

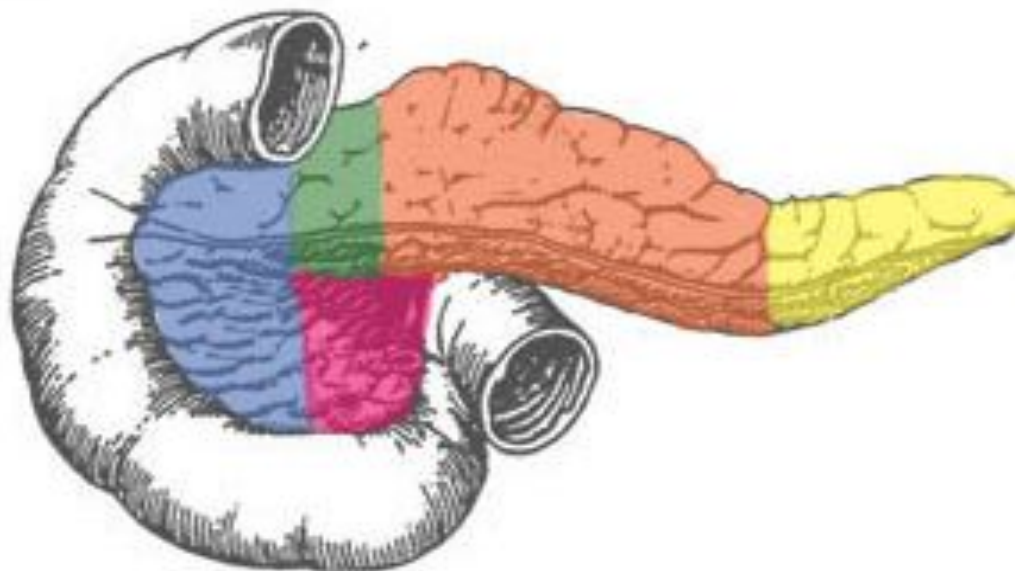
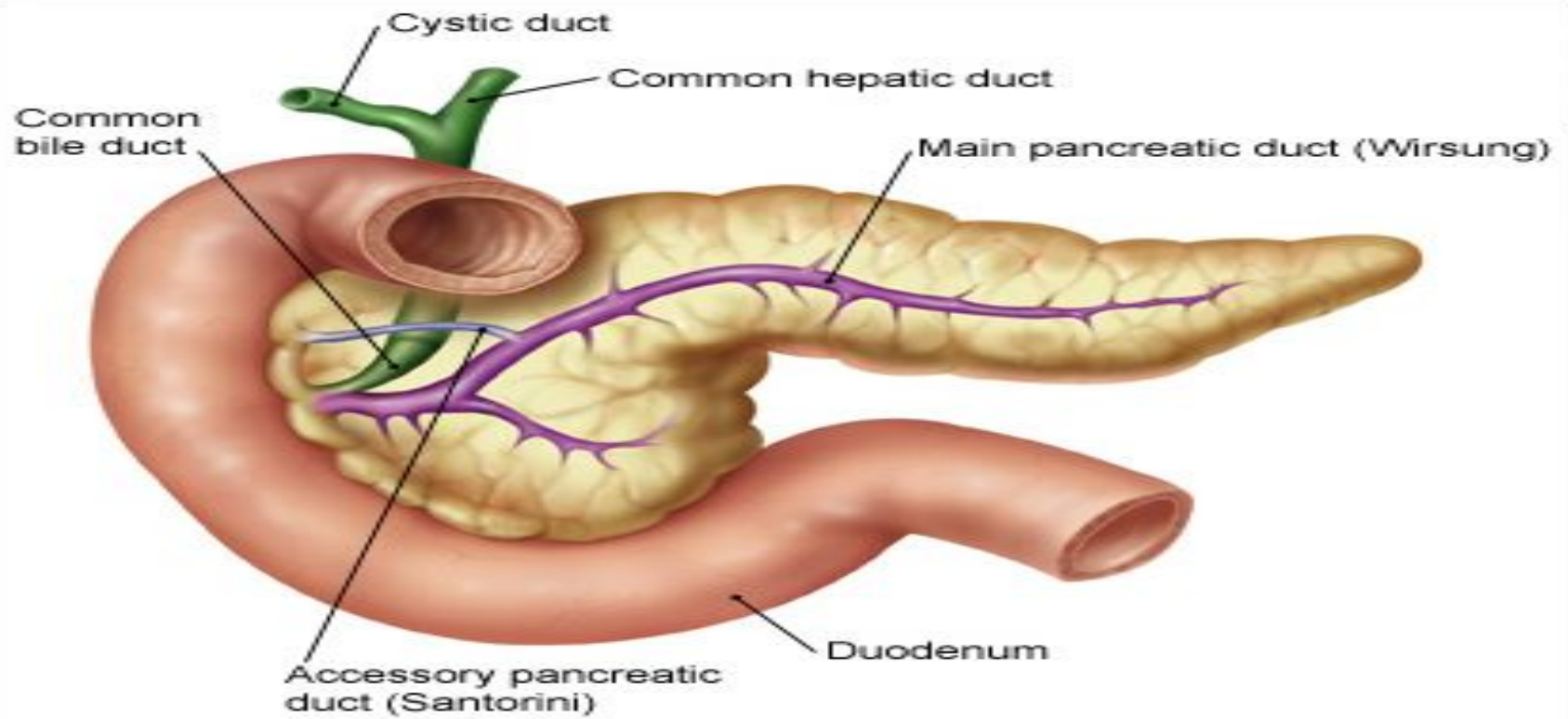
**Tail – shell for
spleen to rest on**

**Body –Forms
shell for stomach
to rest upon.**

Duodenum

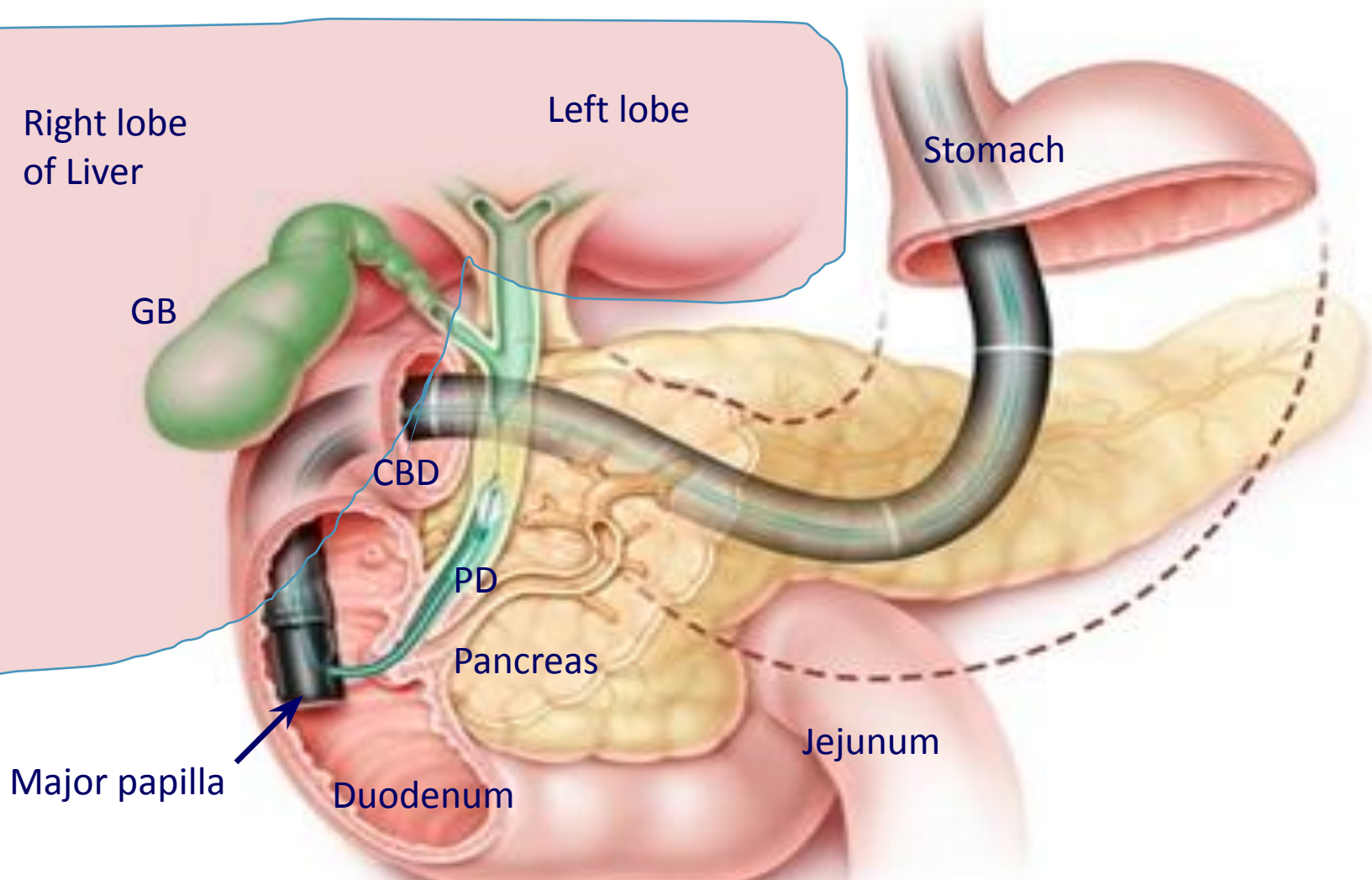
adam.com

**Head – joins Common
Duct at Ampulla of Vater**



- Uncinate process
- Head
- Neck
- Body
- Tail

Biliary anatomy



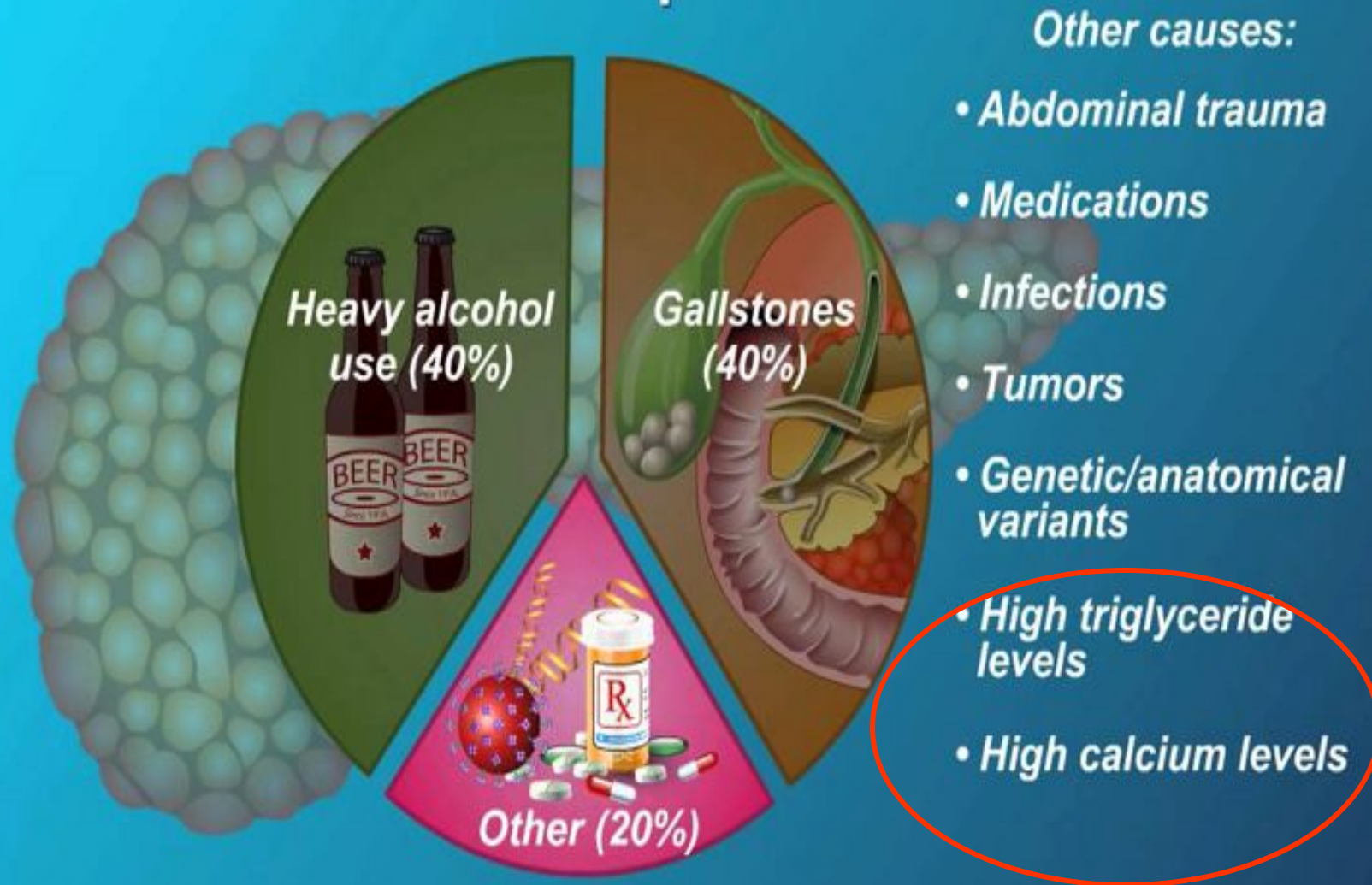
Acute Pancreatitis

- 350,000 hospitalizations annually in the United States.
- 3000 (1-2%) directly related deaths from complications.
- 10% to 15% of deaths occur almost exclusively as a result of Acute Necrotizing Pancreatitis .
- Rises to > 40% if Sterile necrosis becomes Superinfected .
- Increasing incidence (Detection ?Meds ? Iatrog?).
- Increases with increasing age ,onset before 15 yrs. unusual.

Etiology of Acute pancreatitis

- **Gallstones**
 - **Alcohol**
- 80-85 % of Cases.
- Endoscopic Retrograde Cholangio - Pancreatography (ERCP) . (overall 5% -10%)
 - Drugs
 - Trauma
 - Viral infection
 - Neoplasms
 - Anatomic variants
 - Metabolic problems
 - Hyper- Calcemia
 - Hyper-Triglyceridemia
- 10% of patients.

Causes of acute pancreatitis

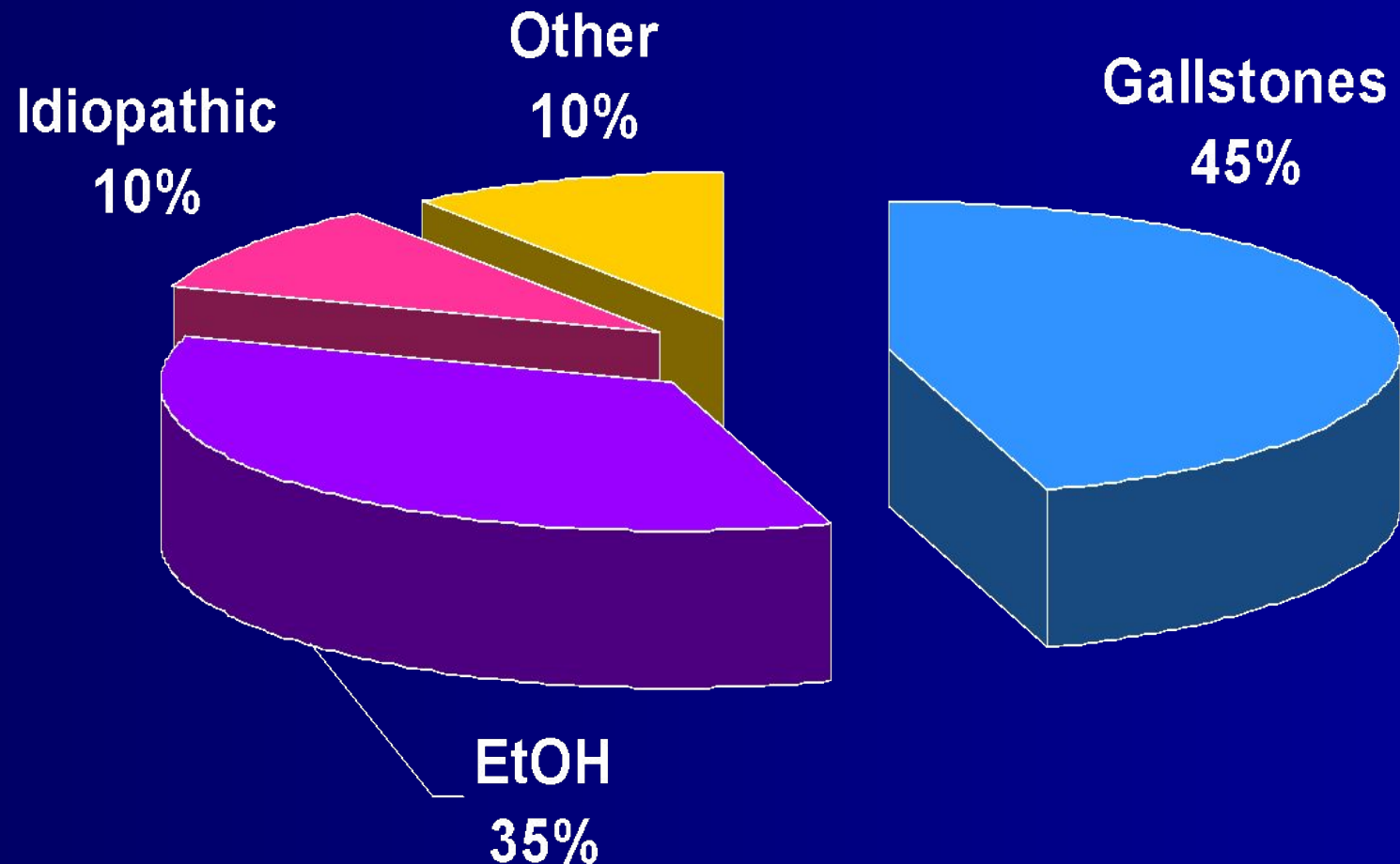


Etiology of Acute pancreatitis

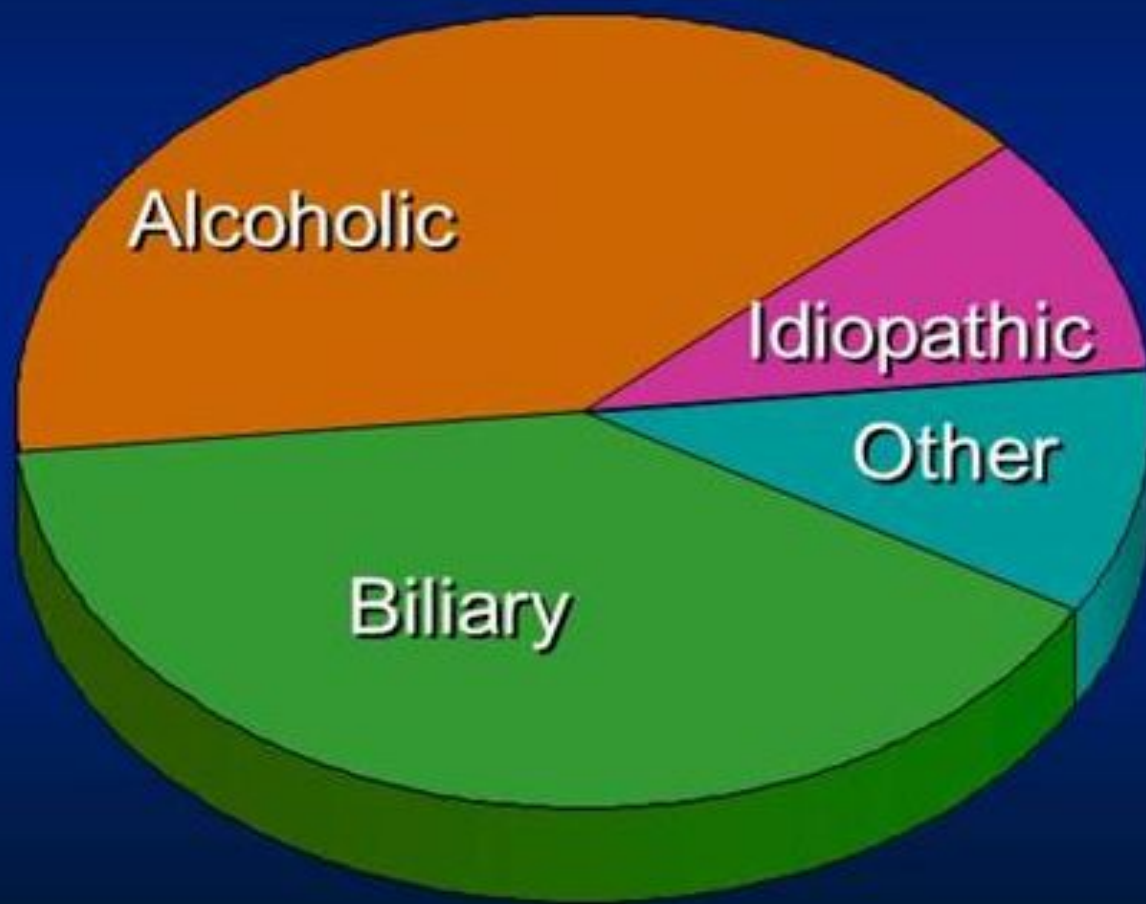
Rare causes of acute pancreatitis

- Annular pancreas
- Autoimmune Pancreatitis
- Hereditary Pancreatitis
- Familial adenomatous polyposis
- Pseudopapillary tumor of the pancreas

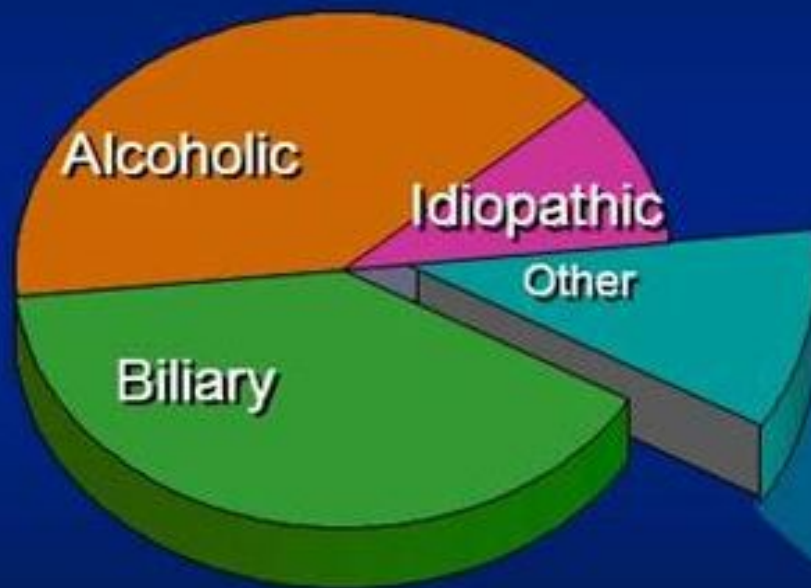
Acute Pancreatitis Etiology



Etiology



Etiology



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

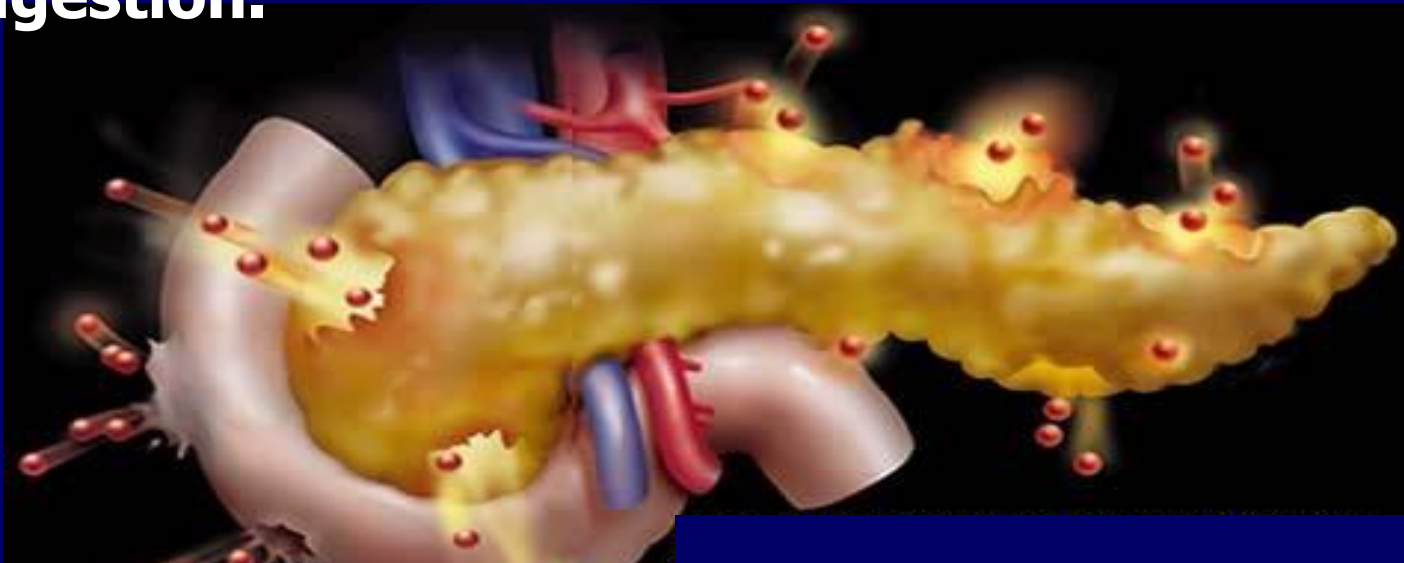
Prematurely activated digestive enzymes cause inflammation of the pancreas



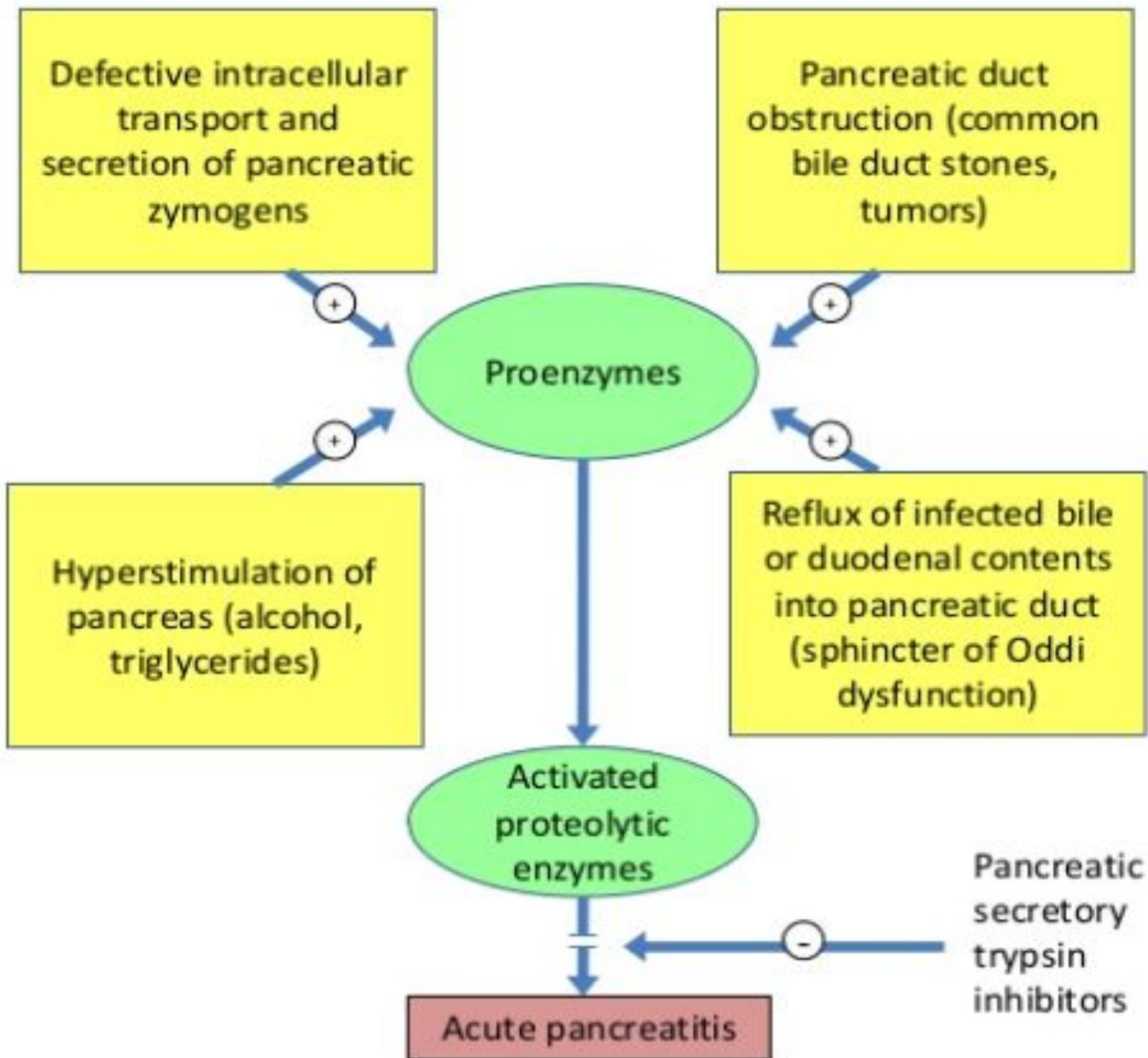
Acute Severe Pancreatitis

Pathophysiology

Injury or disruption pancreatic acini → pancreatic ducts disruption → Leakage of active pancreatic enzymes → Auto digestion.

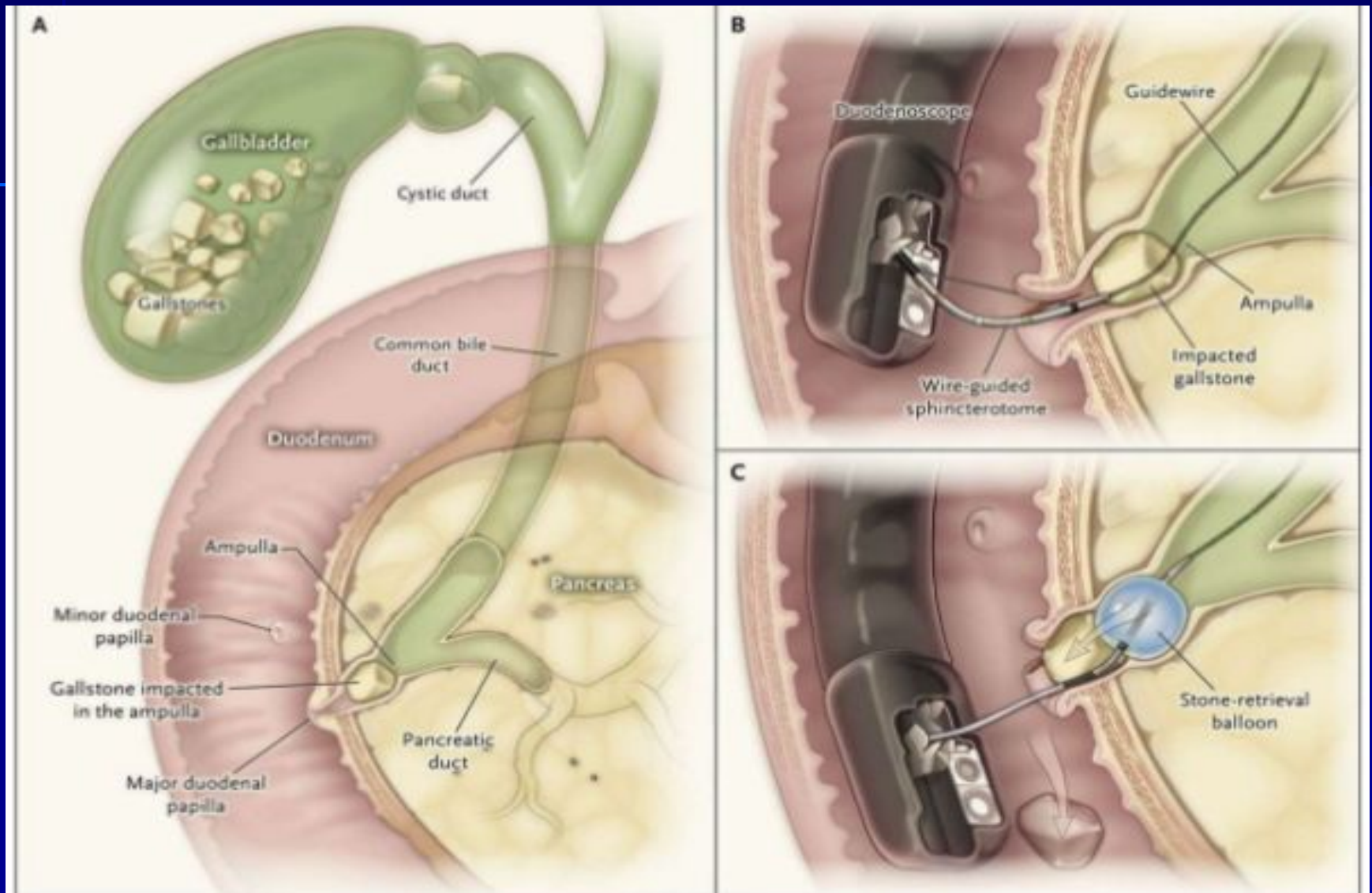


Breakdown of cell membranes → edema → vascular damage, hemorrhage, necrosis → inflammatory mediators → Shock, MODS, ...



Theory for AP

- **Obstructive Theory** : Obstruction of sphincter of oddi by stones increase back pressure & stasis in pancreatic duct.
- **Reflux Theory:** *Stone at ampulla of vater lead to reflux of bile and or duodenal contents which lead to AP.*

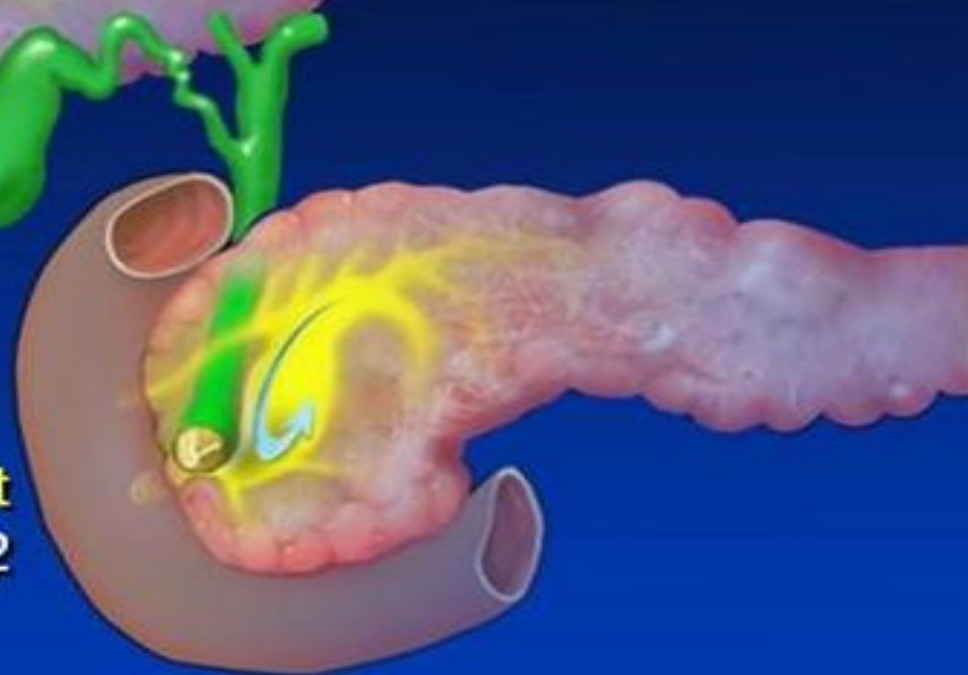


Gallstone Pancreatitis Mechanism

Common channel theory
Opie 1



Obstructed pancreatic duct
Opie 2



Clinical manifestations

- Abdominal pain (Constant – Severe) Radiating to back.
- Abdominal Exam is Not Related to Severity of Pain or Severity of Pancreatitis .
- 90% have Nausea and Vomiting that Does not Relieve pain .
- Septic Shock like ,Dehydration, Tachycardia ,Hypotension.
- Flank or para umbilical ecchymosis .
- Jaundice .
- Pleural effusion signs .

Pain, Oh the pain

- "Worse than Child-Birth" "Worse Than Being Shot".
- Starts Fast within 10 -20 min reaches peak
Third fastest pain onset in GI after perforation and SMA Thrombosis .
- Does not usually undulate (Not Colicky).
- Lasts Days (if no underlying chronic damage)
Longer than biliary colic which is hours.
- Radiate to Back in 50% .
- Sometimes diagnosed at autopsy (Painless)!!.

Diagnosis

- The Diagnosis of AP is most often established by the presence of Two of the Three following criteria:
 - Abdominal pain consistent with the disease.
 - Serum amylase and/or lipase greater than three times the upper limit of normal .
 - Characteristic findings from abdominal imaging.
- ◆ **NB:** Radiology is not usually Required for Diagnosis

Presenting Features



RESPIRATORY EXAMINATION

Clip slide

Left sided* Pleural effusion(10-20%) -
exudative



* Due to close approximation of body and tail of pancreas to the left sided
diaphragm



Cullen's sign



Grey Turner's sign

GREY TURNER¹ SIGN SIGN



CULLEN² SIGN



FOX³



1. Named after British surgeon George Grey Turner(1877-1951)
2. Named for Thomas Stephen Cullen (1869-1953), Canadian gynecologist who first described the sign in ruptured ectopic pregnancy in 1916
3. Named after George Henry Fox(1846-1937), American dermatologist

Assessment Physiological Variable

Diagnostic Tests

- Serum Amylase : (25-125 U/L)

↑ >200 U/L for 24-72 hours
starts to rise 2-6 hr after onset of pain

- Peaks @ 24 hours
- Return to normal @ 72 hr.

- Serum Lipase : (3-19 U/dL)

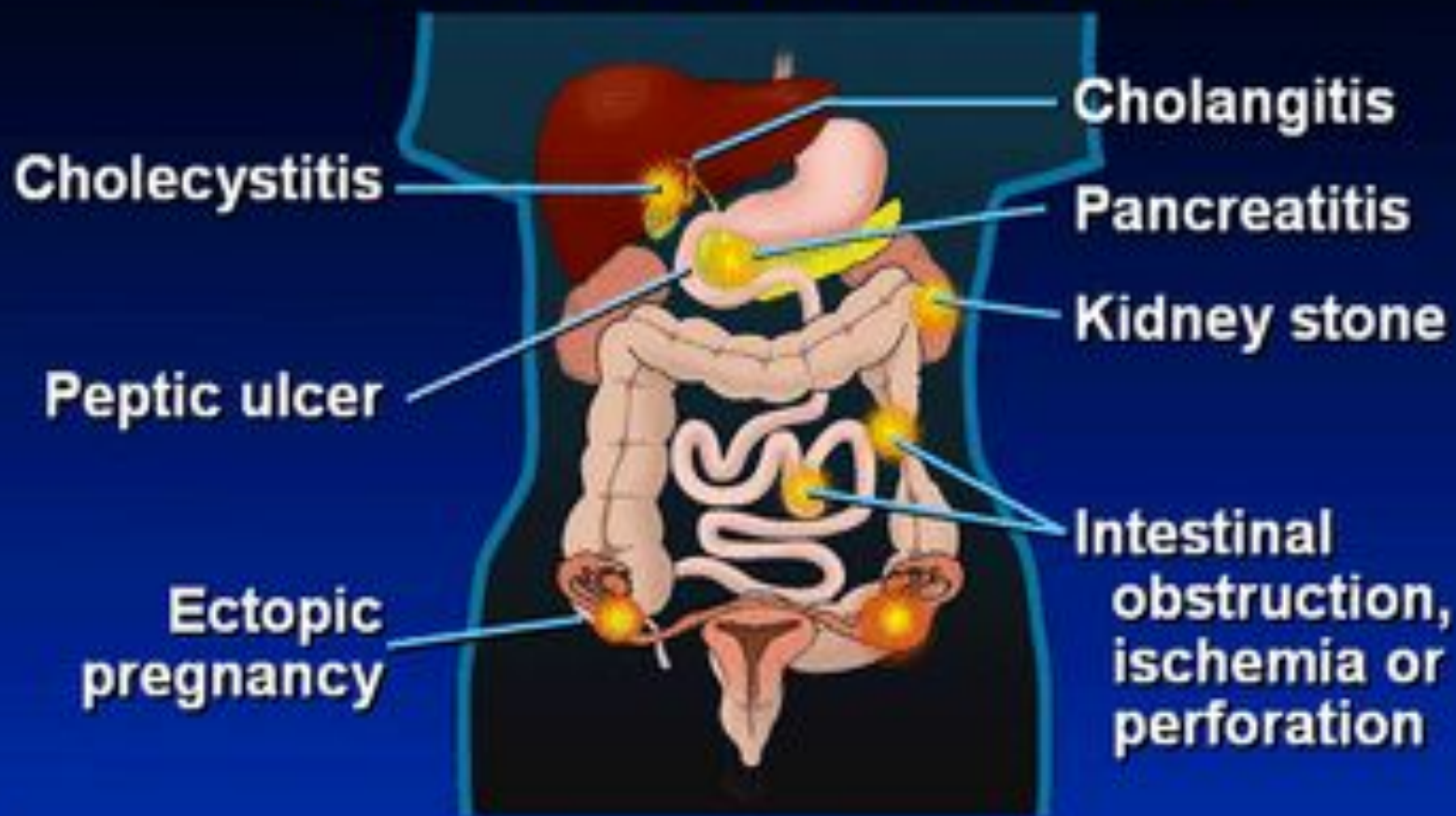
- Used with amylase; rises later than amylase (48 hours)
- Return to normal 5-7 days .

- CRP : Rise after 48-72 hours and correlates with severity of pancreatitis

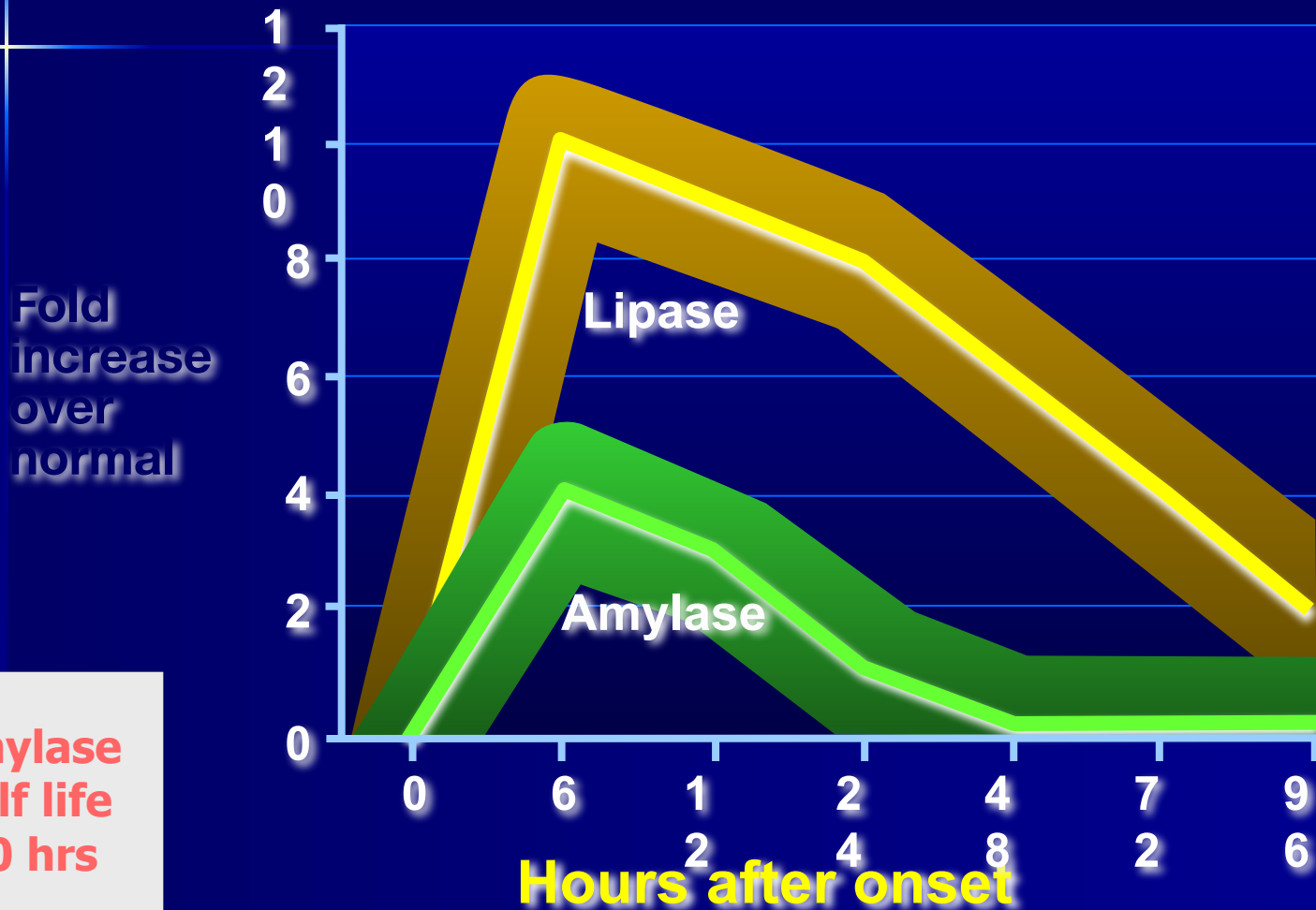
- ↑ WBC's
- ↑ glucose
- ↑ SGOT SGPT
- ↑ lipids
- ↓ calcium
- ↓ magnesium

Acute Pancreatitis

Elevated Serum Amylase



Acute Pancreatitis: Time course of enzyme elevations



Amylase
half life
10 hrs

HEMATOLOGICAL

Clip slide

• Pancreatic Enzymes' Assays

• Serum Amylase:

- ONSET: almost immediately
- PEAK: within several hours
 - 3-4 times upper limit of normal within 24 hrs (90%)
- RETURN to normal in (3-5 days)
- normal at time of admission in 20% cases
- Compared with lipase, returns more quickly to normal values.

Raised Amylase → may not AP

Normal Amylase → may be AP

• Serum Lipase:

- more sensitive/specific than amylase
- Remains elevated longer than amylase (12 days)
- Useful in late presentation and if the cause is High TG

SERUM INDICATOR OF HIGHEST
PROBABILITY OF DISEASE

Ranson's Criteria of Severity

Admission

- Age > 55 years
- WBC > 16,000 mm³
- Glucose > 200 mg/dl
- LDH > 350 IU/L
- AST > 120 IU/L

After 48 hrs

- Hct decrease > 10%
- BUN increase > 5 mg/dl
- Ca²⁺ < 8 mg/dl
- PaO₂ < 60 mm Hg
- Base deficit > 4 mEq/L
- Negative fluid balance > 6L

TABELA 1 - Pancreatite aguda grave estabelecida pelos critérios de Ranson

Ranson (alcoholic etiology or other)	Ranson (biliar etiology)
At admission Age > 55 years Leukocytes > 16 000/mm ³ LDH > 350 U/l AST > 250 U/l Glicemia > 200 mg/dl	At admission Age > 70 years Leukocytes > 18 000/mm ³ LDH > 250 U/l AST > 250 U/l Glicemia > 220 mg/dl
After 48 hours Reduction in hematocrit > 10% Increase in BUN > 5 mg/dl Calcium < 8 mg/dl PO ₂ < 60 mmHg Base excess > 4 mEq/l Fluid leakage > 6L	After 48 hours Reduction in hematocrit > 10% Increase in BUN > 2 mg/dl Calcium < 8 mg/dl PO ₂ < 60 mmHg Base excess > 5 mEq/l Fluid leakage > 4L

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
- PO₂ <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)>



APACHE II score = (acute physiology score)

1. Rectal temperature (°C)
2. Mean arterial pressure (mmHg)
3. Heart rate (bpm)
4. Respiratory rate (bpm)
5. Oxygen delivery (mL/min)
6. PO₂ mmHg)
7. Arterial pH
8. Serum sodium (mmol/L)
9. Serum potassium (mmol/L)
10. Serum creatinine (mg/dL)
11. Hematocrit (%)
12. White cell count (10³/mL)
13. History of severe organ insufficiency

Note : Do Not include
" Serum Calcium conc. "

APACHE II

- Acute **P**hysiology and **C**hronic **H**ealth **E**valuation
- as good as the Ranson or Glasgow at 24 and 48 hours of the admission
- APACHE II score > 8 = Severe acute pancreatitis
- Cumbersome to use if one does not use a pc or palm - where the formula is easily downloaded

Assessment of severity

BISAP score

Bedside index of severity in acute pancreatitis (BISAP) score

This calculator evaluates the following Clinical Criteria:

- ☐ BUN >25 mg/dL (8.9 mmol/L)
- ☐ Impairment of mental status with a Glasgow coma score <15
- ☐ SIRS (systemic inflammatory response syndrome)
- ☐ Age >60 years old
- ☐ Pleural effusion

Ranson's Criteria¹

(1 point each)

On Admission:

- WBC >16,000/ μ L
- Age >55 years
- Glucose >200 mg/dL
- AST >250 IU/L
- LDH >350 IU/L

Within 48 Hours of Admission:

- Hct decrease >10%
- BUN increase >5 mg/dL
- Serum calcium <8 mg/dL
- Arterial pO₂ <60 mmHg
- Base deficit >4 mEq/L
- Fluid needs >6 L

Ranson score < 3 → 0–3% mortality

Ranson score = 3–5 → 11–15% mortality

Ranson score ≥ 6 → 40% mortality

BISAP Score²

(1 point each)

B UN >25 mg/dL

I mpaired mental status

S IRS

A ge >60

P leural effusions

BISAP score < 2 → 0–0.5% mortality

BISAP score = 2 → 2% mortality

BISAP score ≥ 3 → 5–20% mortality

Classification of Acute pancreatitis

Mild

(Interstitial pancreatitis)

- Majority of cases
- Minimal organ failure
- Uneventful recovery
- Responds well to supportive therapy

Severe

(Necrotizing pancreatitis)

- Approximately 20% of patients
- Associated with
- Organ failure
- local complications
 - Necrosis
 - Infection
 - Pseud-ocyst formation
- Requires intensive monitoring and specific therapies and has a more guarded prognosis.

Acute Pancreatitis

**Two
phases**

Early

1st week

Late

After 1st week

Severity

Mild

No organ failure

Moderate

Organ failure
less than 48 h

Severe

Organ failure
longer than 48 h

**Two
types**

Oedematous

Complications

< 4 wk : acute peripancreatic collection
> 4 wk: pseudocyst

Necrotizing

< 4 wk : acute necrotic collection
> 4 wk : walled-off necrosis

Interstitial/Edematous Pancreatitis



Necrotizing Pancreatitis



- *There are 2 overlapping phases of AP, Early and Late.*
- *The early phase of AP takes place in the first 2 weeks after disease onset .*
- *The late phase can last weeks to months thereafter.*

The Role of Imaging in Acute Pancreatitis

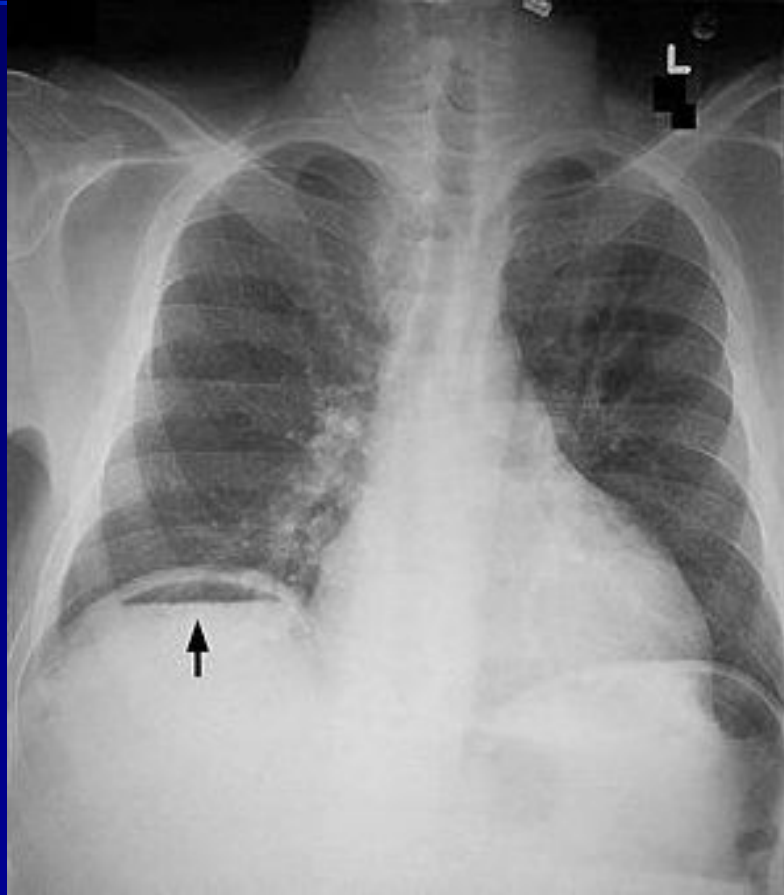
- **Cause of pancreatitis .**
- **Confirm the diagnosis .**
- **Identify necrosis .**
- **Determine the presence of complications**
 - **Fluid collections .**
 - **Vascular abnormalities**
- **Assessment of severity.**

Diagnostic Tests & Procedures

- **Abdominal and chest films**
- **CT scan MRI Scan**
- **Ultrasound**
- **Aspiration biopsy**
- **Peritoneal lavage**

- *Endoscopic Retrograde
Cholangio-pancreatography
(ERCP)*

PFAS



Abdominal X-ray

Acute Pancreatitis:

Findings are variable and have low sensitivity

- Normal or
- Localized ileus (“sentinel loop”) or
- “Colon cut-off sign” due to spasm of descending colon from inflammation



COLON CUT-OFF SIGN



SENTINEL LOOP SIGN



Ultrasound

Acute Pancreatitis

US Findings

Although transabdominal ultrasound is poorly reliable for imaging the pancreas itself, it is **the best initial radiographic test** for the evaluation of mild AP because:

1. it detects gallstones as a potential cause,
2. it rules out acute cholecystitis as a differential cause of pain and hyperamylasemia, and
3. it detects biliary dilatation suggestive of the need for early endoscopic retrograde cholangiopancreatography (ERCP).

The pancreas may appear completely normal in mild cases of acute pancreatitis.

CECT

- Multi-Detector CT.
 - Viability of Pancreatic parenchyma.
 - Amount of Peri-Pancreatic Inflammation.
 - Fluid collection.
 - Cause of pancreatitis.
- MRI , MRCP
- EUS

Multidetector-row computed tomography (MDCT)

- *The imaging study of choice for Acute pancreatitis.*
- *Faster image acquisition*
- *Improved resolution*
- *Can be converted into three-dimensional reconstructions*



Source: Curr Opin Gastroenterol © 2005 Lippincott Williams & Wilkins

CT severity index, by Balthazar in 1994

- **Focuses on the Presence and Degree of:**
 - Pancreatic inflammation (fluid collections).
 - Necrosis.
- **Successfully used to predict overall morbidity and mortality**
- **Limitations**
 - Does not correlate significantly with
 - Development of organ failure.
 - Extra pancreatic parenchymal complications.
 - Peri pancreatic vascular complications.
 - The inter-observer agreement is approximating 75%.

CT Severity Index

appearance		normal	enlarged	inflamed	1 fluid collection	2 or more collections
grade		A	B	C	D	E
score		0	1	2	3	4

necrosis	none	< 33%	33-50%	> 50%
score	0	2	4	6

score	morbidity	mortality
1-2	4%	0%
7-10	92%	17%

Balthazar et al. Radiology 1990.

Modified CT Severity Index by Koenraad in 2004

I- Pancreatic inflammation

Prognostic Indicator	Points
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4

Modified CT Severity Index by Koenraad in 2004

II- Pancreatic necrosis	
Prognostic Indicator	Points
None	0
$\leq 30\%$	2
$> 30\%$	4
Extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement)	2

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

Complications of acute pancreatitis

Complications	Causes and features
Shock and renal failure	Pancreatic failure is associated with leakage of fluid in the pancreatic bed also ileus with fluid filled loops of bowel leading to pre-renal azotemia and then acute tubular necrosis.
Hypoxia	ARDS due to micro thrombi in pulmonary vessels.
Hyperglycemia	Due to disruption of pancreatic islets.
Hypocalcemia	Sequestration of calcium in fat necrosis.
Hypoalbuminemia	Increased capillary permeability.

Complications of acute pancreatitis

Pancreatic complications	Causes and features
Necrosis	
Abscess	Rising fever, leukocytosis, localized tenderness and epigastric mass. It may be associated with left sided pleural effusion and enlarged spleen due to splenic vein thrombosis.
Pseudocyst	Encapsulated fluid collection with high enzyme content. Usually less than 6cm sized pseudocysts resolve spontaneously. They may become secondarily infected requiring drainage of abscess.
Ascites	Gradual increase in abdominal girth and persistent elevation of serum amylase in the absence of frank abdominal pain. It results from rupture of pancreatic duct or drainage of pseudocyst into the pancreatic cavity.

Principles for managing patients with acute pancreatitis

Assessing the Severity Remains the key Element in the Initial Assessment of Patients.

Principles for managing patients with acute pancreatitis

- Supportive care with close attention to volume status and electrolyte balance.
- Fasting of the patient .
- Pain management using narcotic agents.
- Predicting the severity of an attack and triaging of patients to intensive care units or a regular floor.

Initial Management

- Aggressive Hydration : defined as 250-500 ml per hour of isotonic crystalloid solution Ringer Lactate should be provided to all patients 12-24h.
- Analgesia : Pethidine , Diclofenac , Tramadol ,Paracetamol.
- Oxygen supplement .
- Antibiotics : should be given for an Extra-pancreatic infection.
- In mild AP, Oral Feedings can be started immediately if there is no nausea and vomiting, and abdominal pain has resolved .
- In patients with mild AP, found to have GBS in the gallbladder, a Cholecystectomy should be performed before discharge to prevent a recurrence of AP .

Principles for managing patients with acute pancreatitis (Cont'd)

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



THANK
YOU

21825

Revised Atlanta.....

- Acute pancreatitis identified two phases of the disease: **early and late.**
- Severity is classified as **mild, moderate or severe.**
 - Mild:
 - the most common form,
 - has no organ failure, local or systemic complications and
 - usually resolves in the first week.
 - Moderate:
 - Presence of transient organ failure, local complications or exacerbation of co-morbid disease.
 - Severe:
 - Persistent organ failure >48 h.
 - Local complications are peripancreatic fluid collections, pancreatic and peripancreatic necrosis (sterile or infected),

Characterization of ICU patients using a model based on the presence or absence of organ dysfunctions and/or infection

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

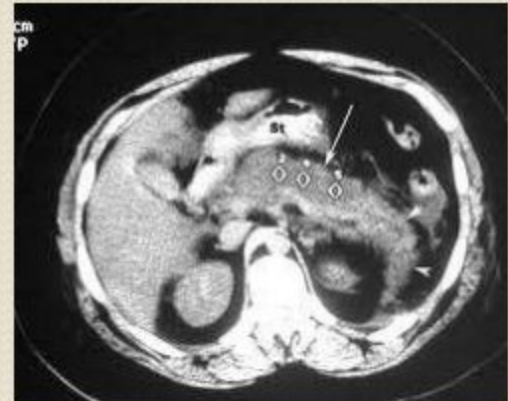
■ *Renal failure*

- Serum creatinine level > 300 µmol/L
- Urine output < 500 mL/24 hr or < 180 mL /8 hr
- need for hemo- or peritoneal dialysis.

Determinants of revised Atlanta classification

- **Local**

- Pancreatic or peripancreatic fluid collection
 - Sterile
 - Infected
- Necrosis
 - Sterile
 - Infected
- Pseudocyst and walled-off necrosis (sterile or infected).

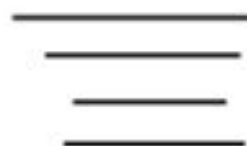


- **Organ failure**

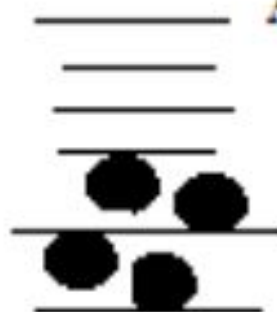
Objections to revised Atlanta classification

- Mere presence of fluid or necrosis do not determine the outcome
 - It is the infection that determines the outcome
 - A number of studies have demonstrated that infectious (peri)pancreatic complications (IPCs), rather than the presence of necrosis *per se*, are a key determinant of the high morbidity and mortality in patients with acute pancreatitis. (Büchler MW, Gloor B, Müller CA *et al.* Acute necrotizing pancreatitis: treatment strategy according to the status of infection. *Ann Surg* 2000;232:619–626. | [Article](#) | [PubMed](#) | [ISI](#) | [ChemPort](#) | Lytras D, Manes K, Triantopoulou C *et al.* Persistent early organ failure: defining the high-risk group of patients with severe acute pancreatitis? *Pancreas* 2008;36:249–254. | [Article](#) | [PubMed](#)), Le Mée J, Paye F, Sauvanet A *et al.* Incidence and reversibility of organ failure in the course of sterile or infected necrotizing pancreatitis. *Arch Surg* 2001;136:1386–1390. | [Article](#) | [PubMed](#)
-
- Mere organ failure does not determine the outcome
 - It is the rapidity, the severity, reversibility and number of organs affected that determines the outcome (5,6,7,8,9)

**Acute peripancreatic fluid
collection**



Acute necrotic collection



After 4 weeks

Pancreatic pseudocyst



**Walled off Necrotic
collection**



Acute peripancreatic fluid collection

 = infection



Acute necrotic collection

Infected



Pancreatic abscess



Walled off Necrotic collection

Infected



Organ failure:

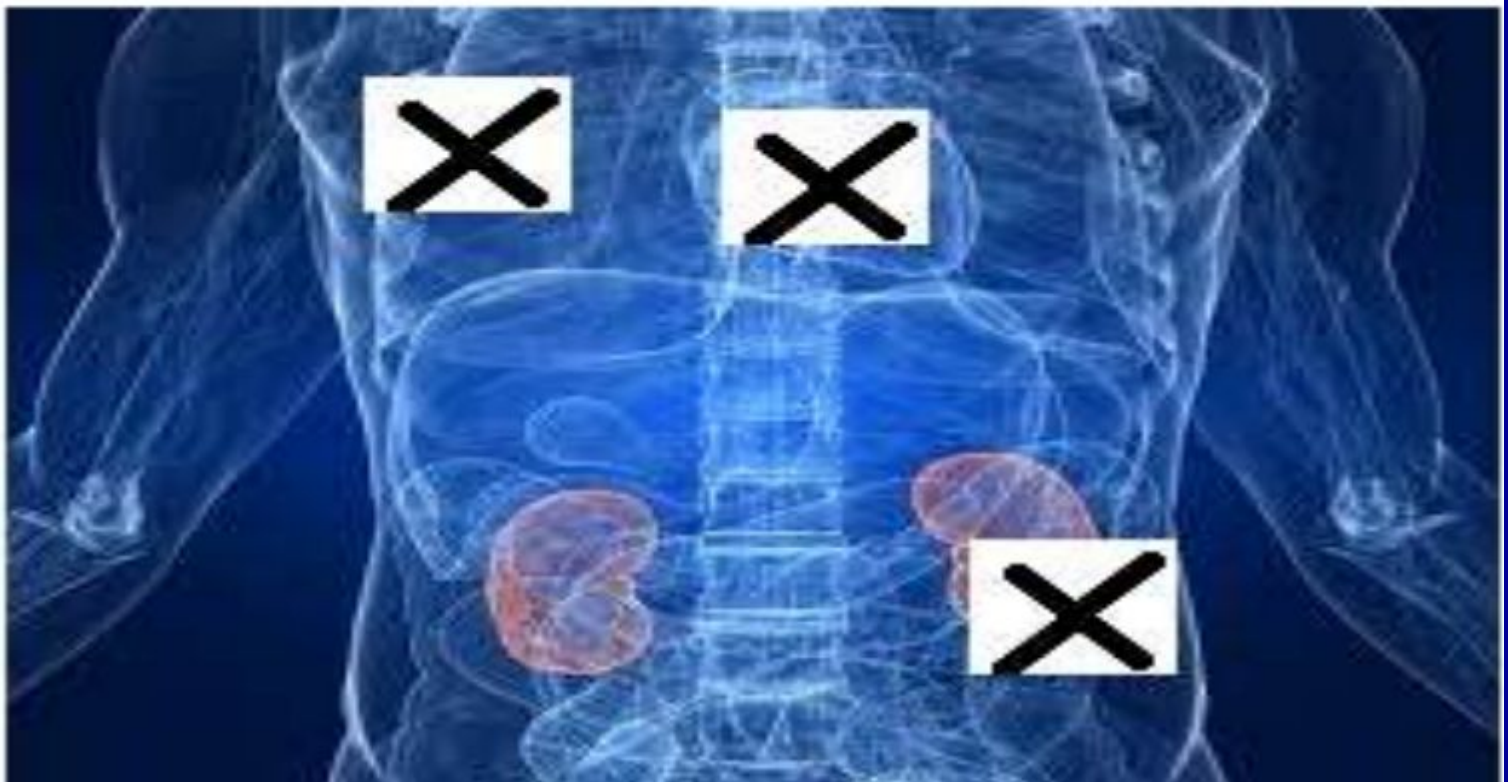
Onset after initial insult

Number of organs affected

Transient or persistent

Response to medical treatment

Duration of failure



Prevention of post-ERCP pancreatitis

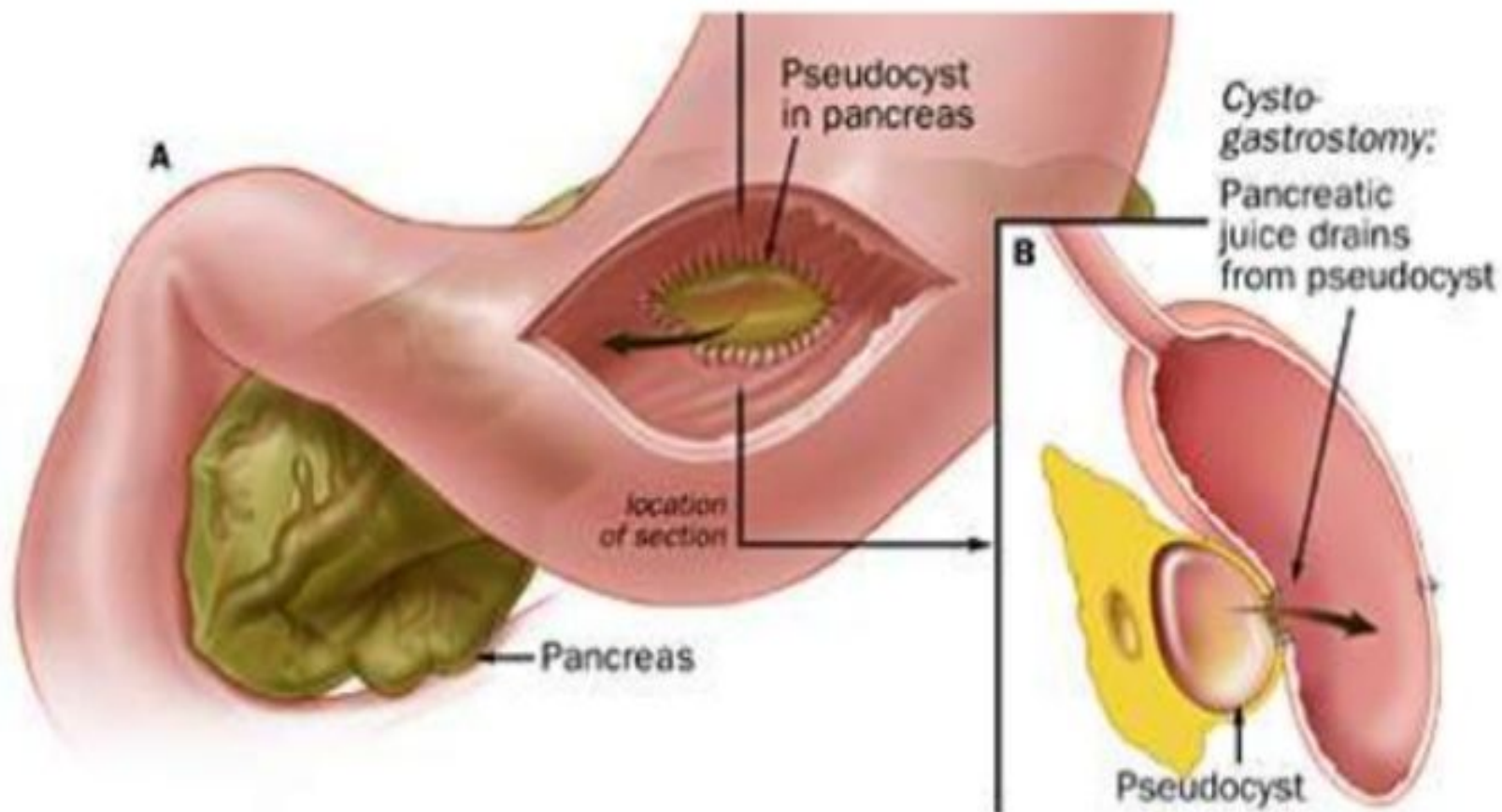
Diclofenac

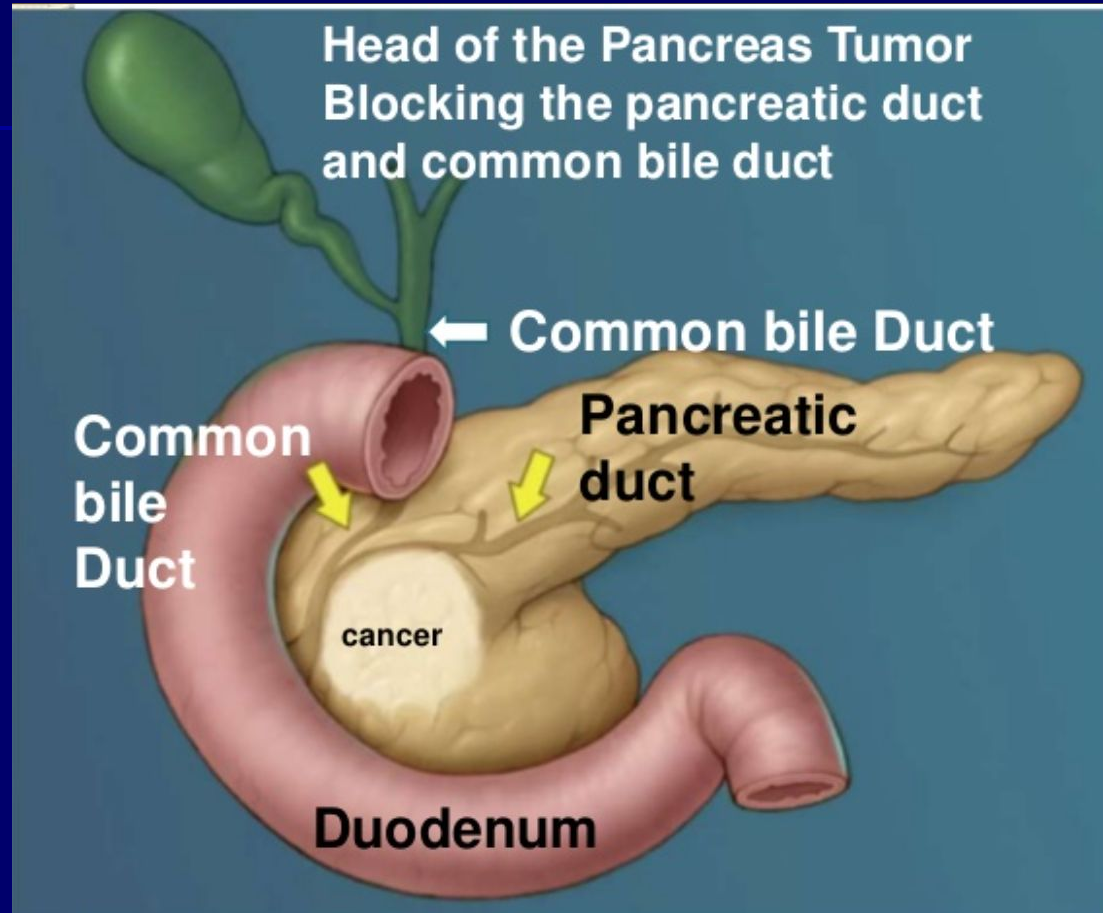
- Diclofenac is a potent inhibitor of phospholipase A2, which regulates inflammatory mediators, including prostaglandins, leukotrienes, and platelet activating factor.
- 100 mg rectal diclofenac given immediately after ERCP reduces the incidence of acute pancreatitis in patients at higher risk for post-ERCP pancreatitis

Prevention of post-ERCP pancreatitis

Nitroglycerine

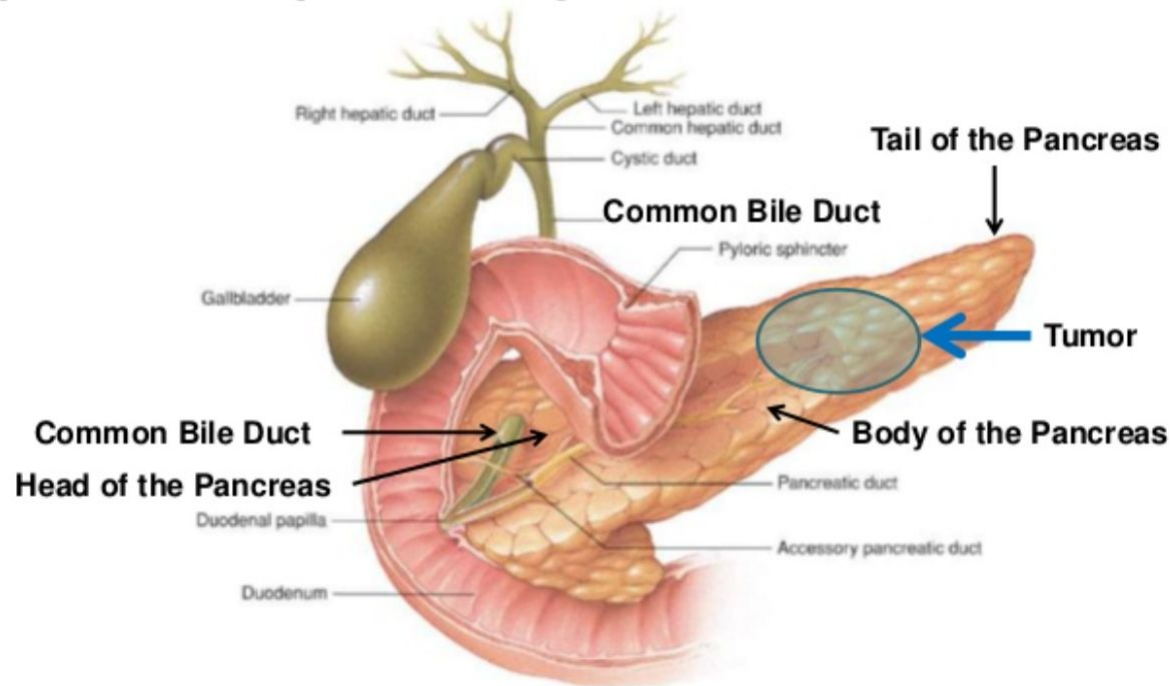
Transdermal glyceryl trinitrate patch placed a half hour before the procedure and continued for 24 hours led to a reduction in post-ERCP pancreatitis





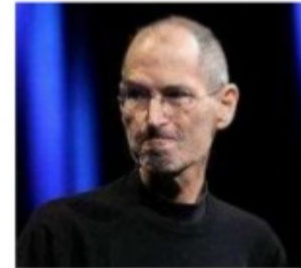
Tumors in the **body or tail** are more likely to present with pain or weight loss

Clip slide



Symptoms of Pancreas Cancer

- Asthenia (weakness) – 86 percent
- Weight loss – 85 percent
- Anorexia (no appetite)– 83 percent
- Abdominal pain – 79 percent
- Epigastric pain (stomach) – 71 percent
- Dark urine – 59 percent
- Jaundice – 56 percent
- Nausea – 51 percent
- Back pain – 49 percent
- Diarrhea- 44 percent
- Vomiting – 33 percent
- Steatorrhea (fatty stools)– 25 percent
- Thrombophlebitis – 3 percent



Signs of Pancreas Cancer

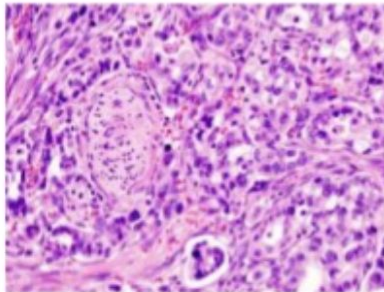
- Jaundice (yellow) – 55 percent
- Hepatomegaly (large liver) - 39 percent
- Right upper quadrant mass – 15 percent
- Cachexia (wasting) – 13 percent
- Courvoisier's sign (nontender but palpable distended gallbladder at the right costal margin) – 13 percent
- Epigastric mass (felt lump in stomach) – 9 percent
- Ascites (abdominal fluid) – 5 percent

Pathology

Clip slide

Ductal adenocarcinoma accounts for about 85% of all neoplasms. And more than 95% of all pancreatic cancers arise from the exocrine (digestive enzymes) elements.

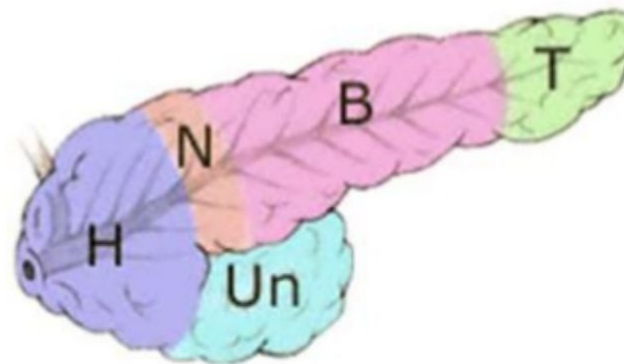
Cancers that arise from the endocrine cells (neuroendocrine, islet cells) account for 5% or less



Location of Pancreas Cancer

- 60 to 70 percent of exocrine pancreatic cancers are localized to the head
- 20 to 25 percent are in the body/tail and
- the remainder involve the whole organ

H = Head
N = Neck
B = Body
T = Tail
Un = Uncinate



CA 19-9

The reported sensitivity and specificity rates of CA 19-9 for pancreatic cancer range from 70 to 92, and 68 to 92 percent, respectively.

The rates of unresectable disease among all patients with a CA 19-9 level ≥ 130 units/mL versus < 130 units/mL were 26 and 11 percent, respectively. Among patients with tumors in the body/tail of the pancreas, more than one-third of those who had a CA 19-9 level ≥ 130 units/mL had unresectable disease.

Conditions associated with increased serum levels of the tumor marker CA 19-9

Malignant
Pancreatic exocrine and neuroendocrine cancers
Biliary cancer (gallbladder, cholangiocarcinoma, ampullary cancers)
Hepatocellular cancer
Gastric, ovarian, colorectal cancer (less often)
Lung, breast, uterine cancer (rare)
Benign
Acute cholangitis
Cirrhosis and other cholestatic diseases (including gallstones)

Elevated CA 19-9

Cancer

- Pancreas
- Biliary Cancer (gallbladder, cholangiocarcinoma, ampullary)
- Hepatocellular
- Gastric, ovarian, colorectal (less often)
- Lung, breast, uterus (rare)

Benign

- Acute cholangitis
- Cirrhosis and other cholestatic diseases (gall stones)

Ultrasound



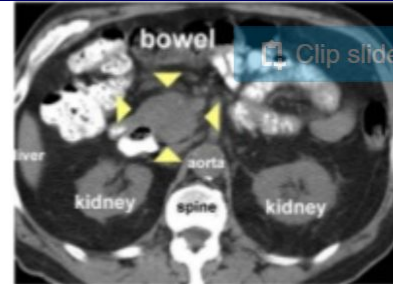
Clip slide

study of 900 patients who underwent ultrasound to work up painless jaundice, anorexia, or unexplained weight loss

The sensitivity for detection of all tumors in the pancreas was 89 percent Among the

779 patients who were followed over time and established not to have developed a pancreatic tumor, nine had false-positive US findings (specificity 99 percent).

CT



Sensitivity of CT for pancreatic cancer depends on technique and is highest (89 to 97 percent) with triple-phase, helical multidetector row CT

As expected, sensitivity is higher for larger tumors; in one study, the sensitivity was 100 percent for tumors >2 cm, but only 77 percent for tumors ≤ 2 cm in size

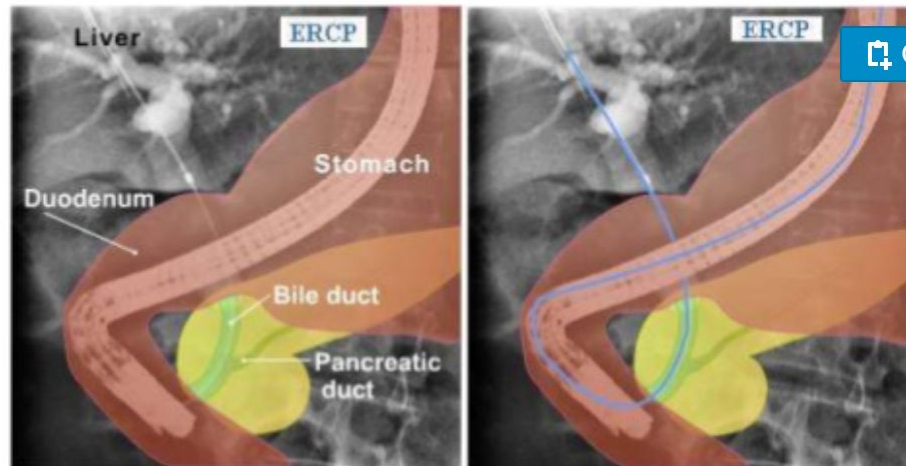
ERCP



Sensitivity of 92 percent and Specificity of 96 percent for diagnosing cancer of the pancreas by ERCP

ERCP provides an opportunity to collect tissue samples (forceps biopsy, brush cytology) for histologic diagnosis.

However, the sensitivity for detection of malignancy (approximately 50 to 60 percent) is lower than that of endoscopic ultrasound (EUS)-guided FNA (sensitivity 92 percent)



ERCP
Inject dye into
the duct system
and look for
compression

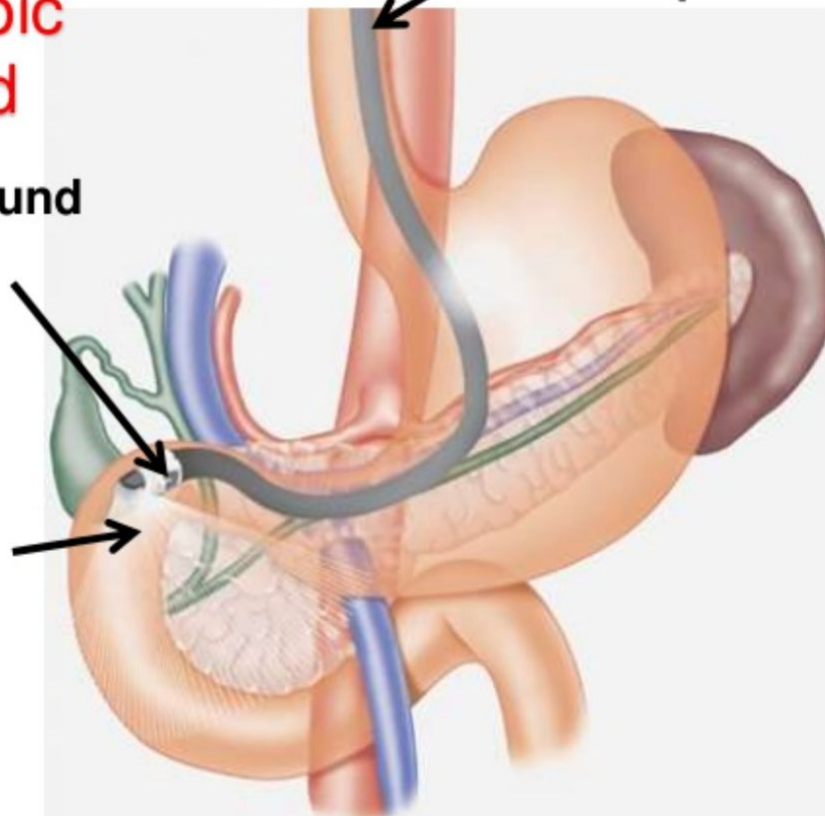


EUS (Endoscopic Ultrasound)

Ultrasound
Tip

Detailed
image of
this area

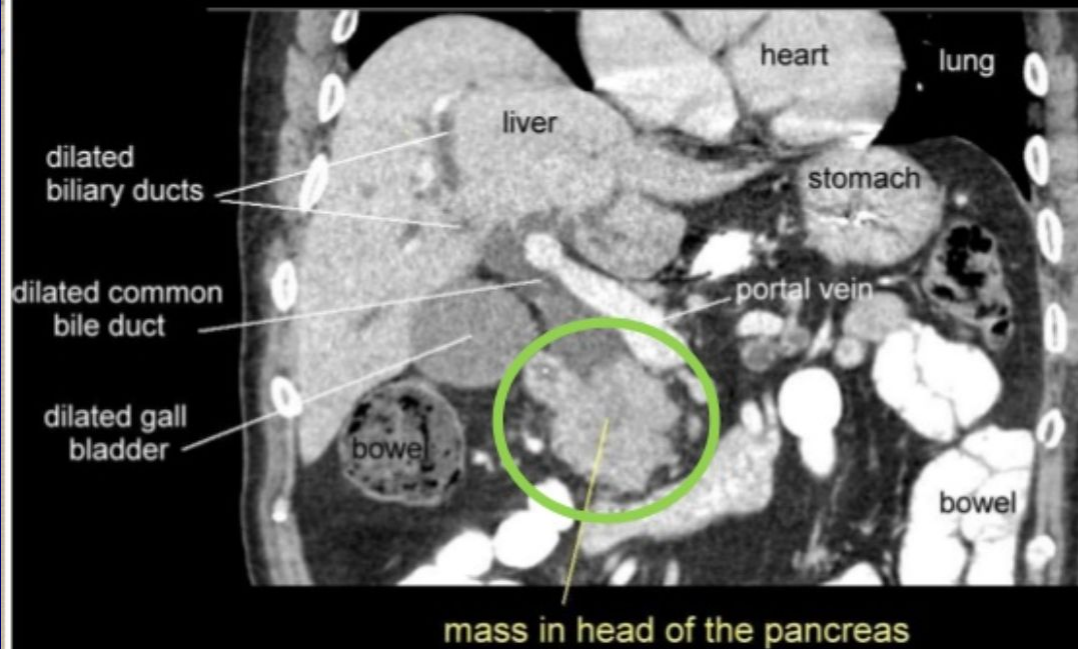
endoscope



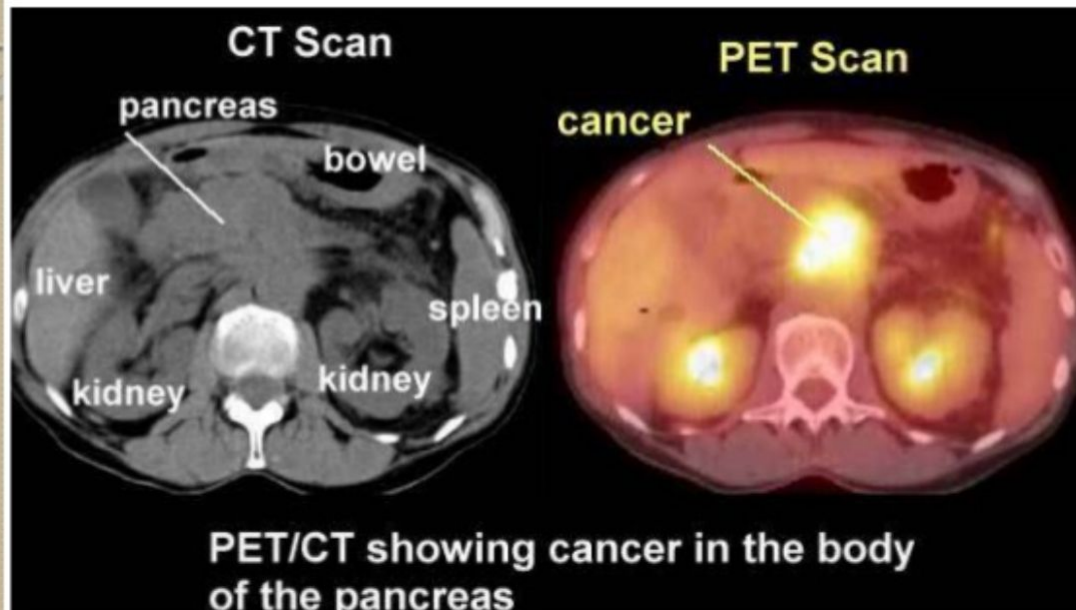
CT Scans

Clip slide

Pathology = 4cm adenocarcinoma in the head of the pancreas



CT or PET Scan



Survival

Surgery offers the only cure but only 10-20% are candidates for resection and even in this group the 5 year survival is only 20% and the median 13 to 20 months

Locally advanced the survival is 8 to 14 months

Up to 60% already have metastases and a median survival of only 4 to 6 months