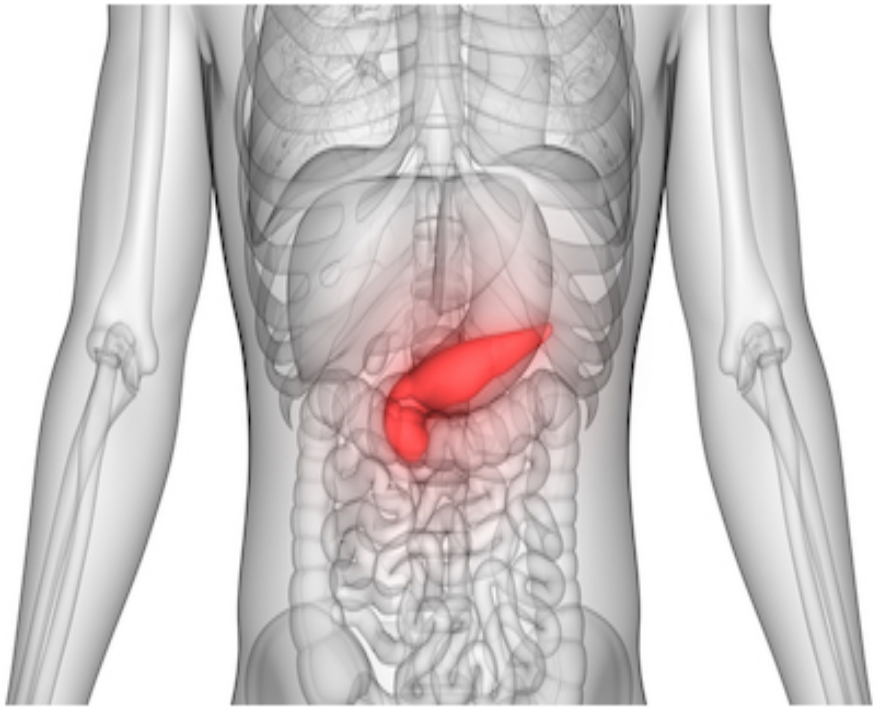


ANATOMY:

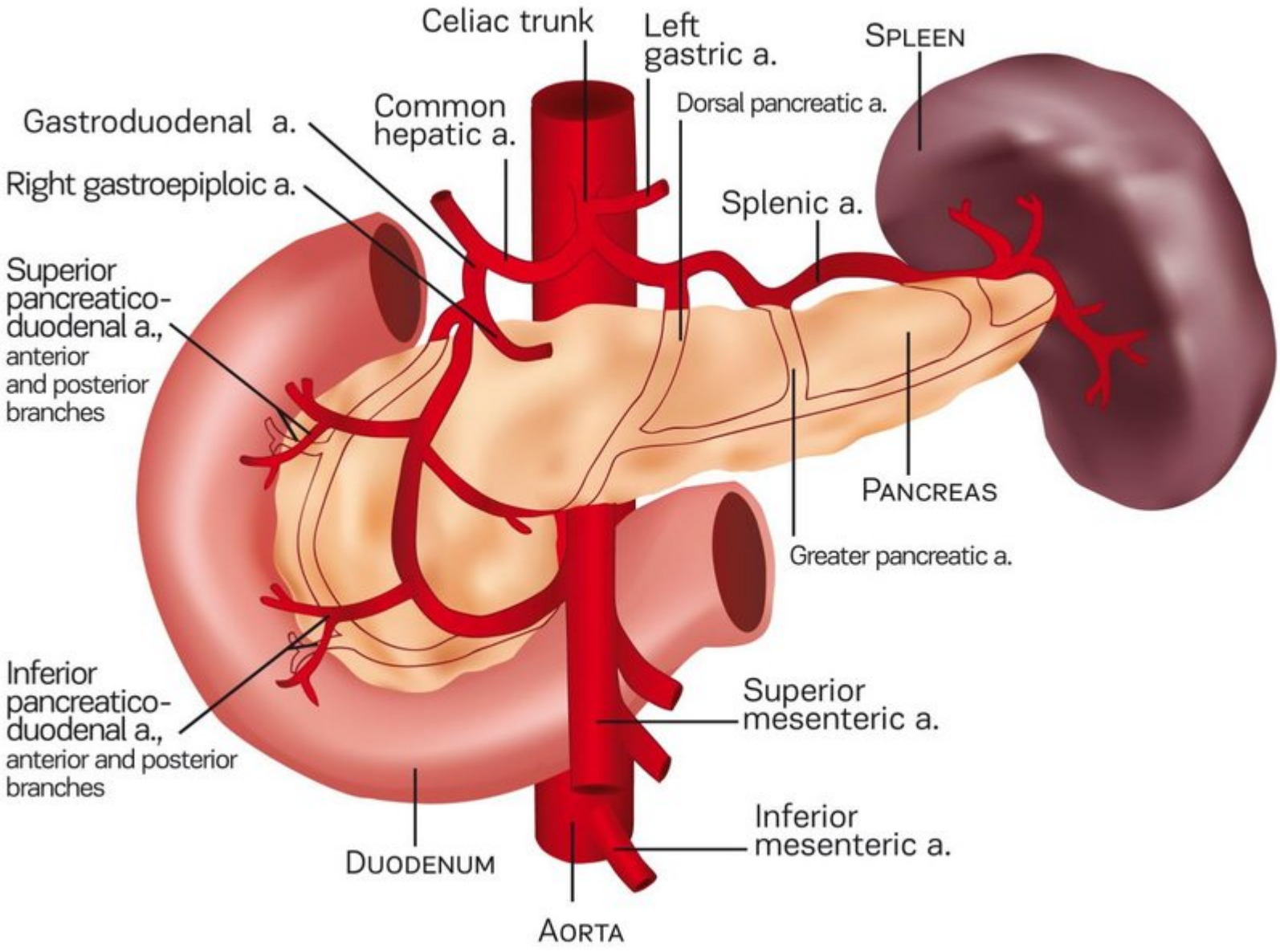
- Pancreas is an elongated retroperitoneal organ; 15–20 cm in length; lies against L1–L2 vertebra. It lies posterior to stomach, separated by lesser sac.

- The pancreas is situated in the retroperitoneum. It is divided into a head, which occupies 30% of the gland by mass, a neck,a body and tail, which together constitute 70%.

- The head lies within the curve of the duodenum, overlying the body of the second lumbar vertebra and the vena cava.

- The aorta and the superior mesenteric vessels lie behind the neck of the gland.

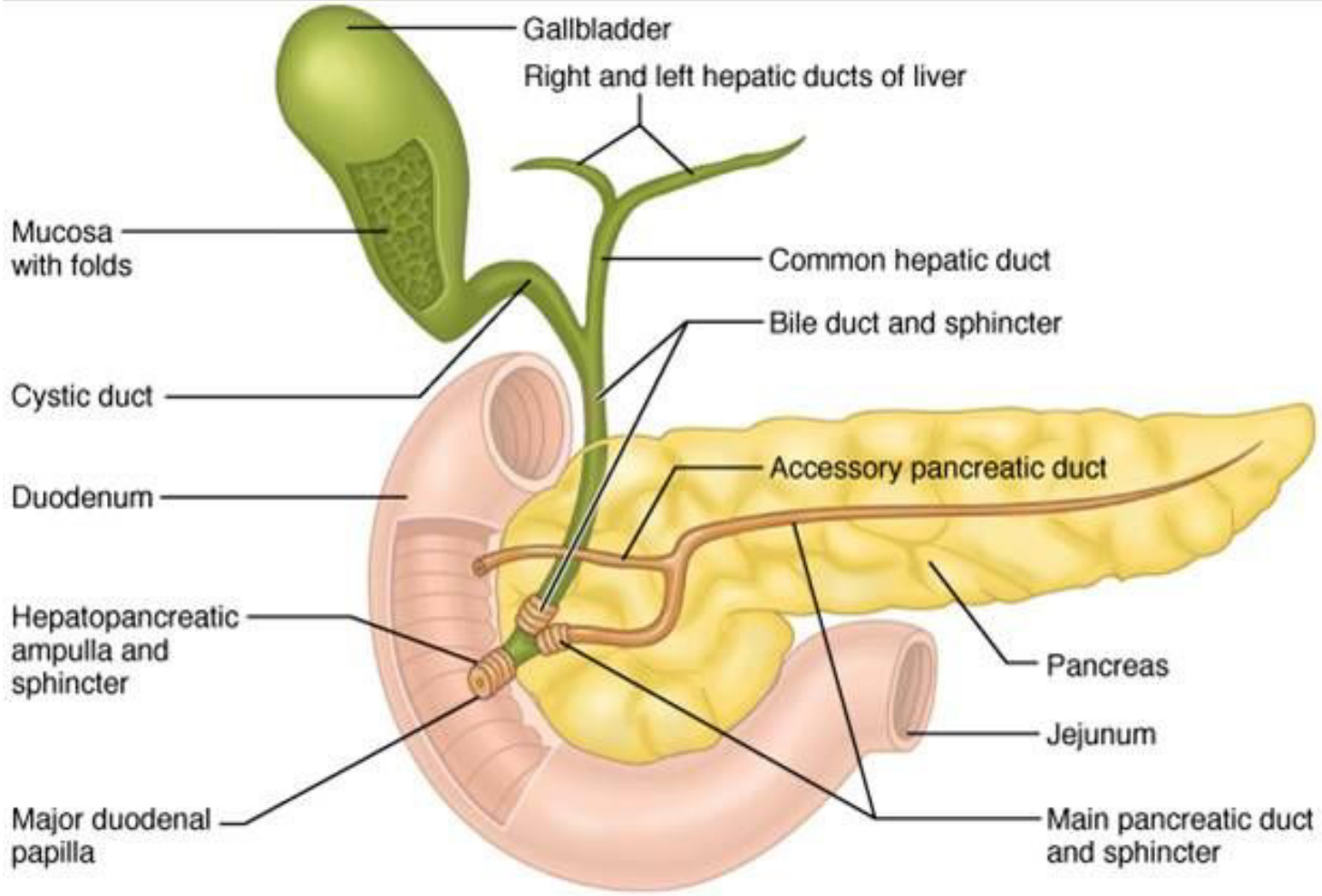
- Behind the neck of the pancreas, near its upper border, the superior mesenteric vein joins the splenic vein to form the portal vein.

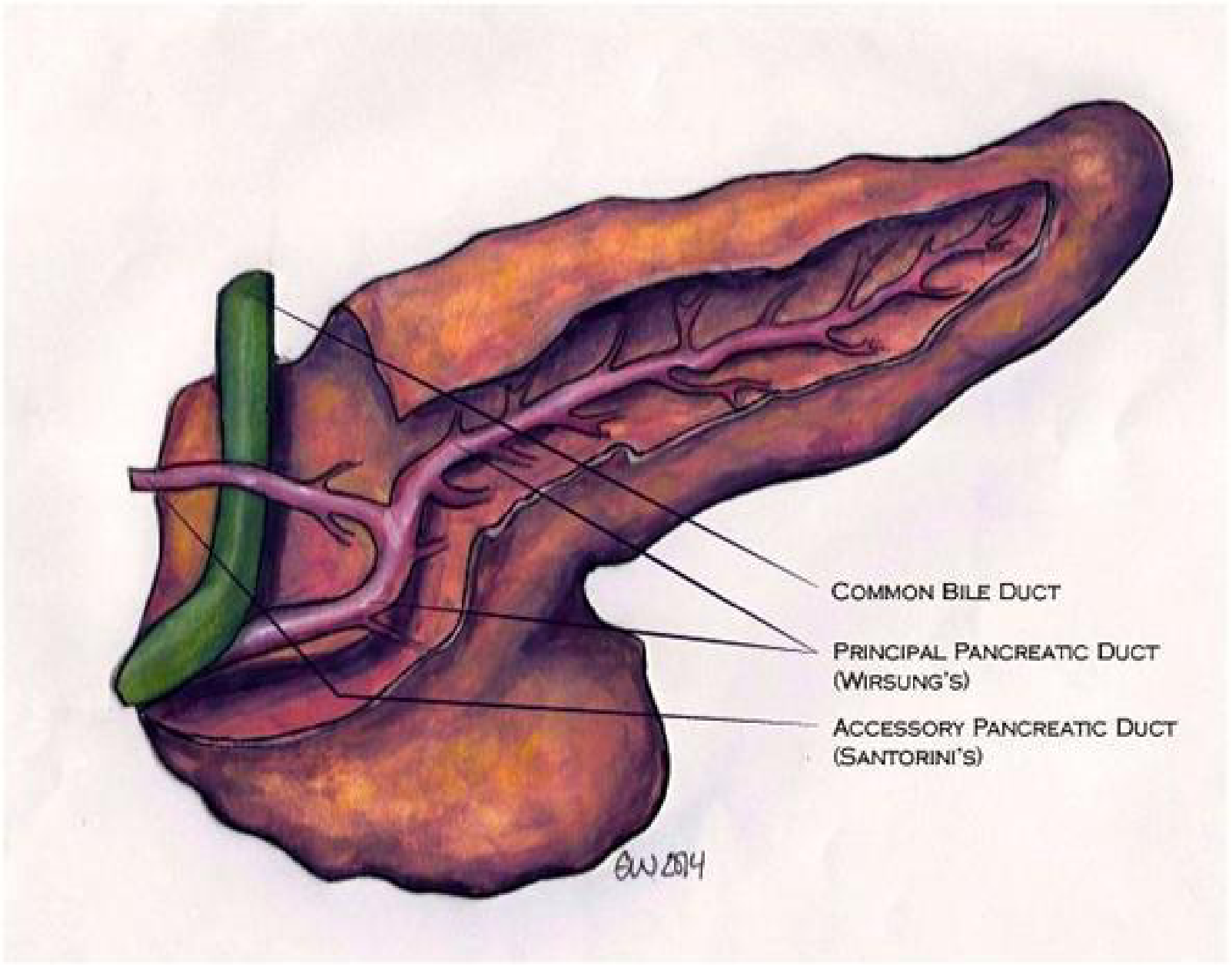

Celiac trunk
Left gastric a.
Spleen
Common hepatic a.
Dorsal pancreatic a.
Gastroduodenal a.
Right gastroepiploic a.
Splenic a.
Superior pancreatico-duodenal a., anterior and posterior branches
Pancreas
Greater pancreatic a.
Inferior pancreatico-duodenal a., anterior and posterior branches
Superior mesenteric a.
Inferior mesenteric a.
Duodenum
Aorta

- **Ducts of Pancreas:**

- 1. Main duct of pancreas (Duct of Wirsung): It begins in the tail of pancreas and runs on the posterior surface of the body and head of pancreas, receives numerous tributaries at right angle along its length (Herring bone pattern).
- It joins the bile duct in the wall of the second part of duodenum to form hepatopancreatic ampulla (of Vater) and opens on the summit of major duodenal papilla (8–10 cm from pylorus).

- 2. Accessory pancreatic duct (Duct of Santorini): It begins in the lower part of the head and opens into the duodenum at minor duodenal papilla (6–8 cm from the pylorus).


Gallbladder
Right and left hepatic ducts of liver
Mucosa with folds
Common hepatic duct
Bile duct and sphincter
Cystic duct
Accessory pancreatic duct
Duodenum
Hepatopancreatic ampulla and sphincter
Pancreas
Jejunum
Major duodenal papilla
Main pancreatic duct and sphincter


COMMON BILE DUCT
PRINCIPAL PANCREATIC DUCT
(WIRSUNG'S)
ACCESSORY PANCREATIC DUCT
(SANTORINI'S)

- The pancreas weighs approximately 80 g.

- Clusters of endocrine cells, known as islets of Langerhans, are distributed throughout the pancreas. Islets consist of different cell types:

- 75% are B cells (producing insulin);
- 20% are A cells (producing glucagon);
- and the remainder are D cells (producing somatostatin) and a small number of pancreatic polypeptide cells.

- **Blood Supply of Pancreas :**

- **Arterial:**

1. Pancreatic branches of splenic artery.
2. Superior pancreaticoduodenal artery.
3. Inferior pancreaticoduodenal artery.

Venous drainage is into portal vein.

Nerve Supply:

Parasympathetic supply is from vagus and sympathetic innervation is from splanchnic nerves.

- **Functions** :

- Exocrine part secretes pancreatic juice which helps in digestion of proteins, carbohydrates and fats.

- Endocrine part constitutes islets of pancreas which is distributed more numerous in tail of pancreas. β cells of islets secrete insulin. α cells secrete glucagon.

- Normal pancreatic juice is colourless bicarbonate rich fluid containing around 15 g of protein in total, 2.5 litres secretion/day. It alkalises duodenal content and helps digestion.

- Basal secretion of these enzymes is low at rest; rapidly increases by hormonal and neural stimulation. It is controlled by secretin (cAMP) and cholecystokinin (phospholipase C, calcium). Protein part of the juice is secreted by acinar cells. Duct cells secrete fluid and electrolytes. Pancreatic secretion is very low in resting phase.

- **SERUM AMYLASE:**

- Amylase is an enzyme that hydrolyses the starch, glycogen and polysaccharides into simple sugar. It is mainly secreted by pancreas and salivary glands as α amylase.

- One secreted by pancreas is called as amylase isoenzyme – P; one by salivary glands (parotid) is amylase isoenzyme – S. Normal value of serum amylase is 200–250 Somogyi units (40–140 IU/L).

- It increases more than 1000 units in acute pancreatitis; raising titer is more useful. But amylase estimation is not specific. P isoenzyme estimation is more relevant. It is estimated with urinary amylase and serum lipase to have a better sensitivity.

- Amylase level will be very high in peritoneal fluid in acute pancreatitis or pancreatic ascites, in fluid obtained from pseudocyst.

- Serum amylase is said to be elevated if level is more than three times of upper limit of the normal.

- **Raise in amylase level** is common in - Acute pancreatitis, pseudocyst of pancreas, pancreatic trauma, after ERCP.

- Amylase is also often **increased** in other conditions like – Salivary gland diseases like parotitis ,Mesenteric ischaemia , Ruptured aortic aneurysm ,Intestinal obstruction ,Ectopic gestation, salpingitis , Duodenal ulcer perforation , Ectopic amylase production in cancers of breast, lungs, ovary, multiple myeloma ,Renal failure .

- **Amylase may not raise in** - Chronic pancreatitis due to significant destruction and loss of acinar cells .

- 10% cases of severe pancreatitis with necrosis show normal serum amylase level. This is due to necrosis of most of the acinar cells retaining hardly any functioning cells at time.

- Rise in pancreatic amylase will be very high in comparison with nonpancreatic causes of rise where it is than 2–3 times the normal.

- Raised urinary amylase level persists for longer period than serum amylase level.

- Magnitude of amylase elevation is not related to severity of the pancreatitis. It increases once symptoms appear for 12–24 hours then decreases in 6 days.
- Persistent elevation suggests complications like pseudocyst, ascites and abscess formation.

- **SERUM LIPASE:**

- Lipase is secreted by pancreas, liver, intestines and other organs also. It hydrolyses triglycerides into fatty acids and glycerol.

- Normal value is 0–50 units/L but it depends on method and laboratory.

- Lipase is increased in acute and chronic pancreatitis, pseudocyst, cystic fibrosis, pancreatic cancer, bowel ischaemia, renal failure, liver diseases, alcoholism, after ERCP.

- Its half-life is 10 hours much more than amylase and so remains longer than amylase in the serum. It is mainly used in acute pancreatitis.

- **Investigation of the pancreas:**

- Serum enzyme levels
- Pancreatic function tests

Morphology

- Ultrasound scan
- Computed tomography
- Magnetic resonance imaging
- Endoscopic retrograde cholangiopancreatography
- Endoscopic ultrasound
- Plain radiography
  - -Chest
  - -Upper abdomen

- **MAGNETIC RESONANCE CHOLANGIOPANCREATOGRAPHY (MRCP):**

- It is a non-invasive diagnostic method using magnetic field and energy as resonance. T1-weighted images are used for pancreas. T2-weighted images are used for biliary tree. Both images are used to see invasion and adjacent structures.

- **Advantages:**
- It is non-invasive.
- It gives equal or better imaging than ERCP.
- Pancreatic divisum or annular pancreas are identified easily by MRCP.
- Ductal dilatation in chronic pancreatitis and visualization of duct beyond stricture is possible.
- Islet tumours are better visualised and diagnosed by MRCP.

- **Disadvantages:**
- Availability.
- It is only diagnostic. Not therapeutic (ERCP is both diagnostic as well as therapeutic). x Costly

MRCP image

PANCREATITIS

- Pancreatitis is inflammation of the pancreas acute, chronic or relapsing which may lead into complications.

- For clinical purposes, it is useful to divide pancreatitis into acute, which presents as an emergency, and chronic, which is a prolonged and frequently lifelong disorder resulting from the development of fibrosis within the pancreas.

- The underlying mechanism of injury in pancreatitis is thought to be premature activation of pancreatic enzymes within the pancreas, leading to a process of autodigestion.
- Anything that injures the acinar cell and impairs the secretion of zymogen granules, or damages the duct epithelium and thus delays enzymatic secretion, can trigger acute pancreatitis. Once cellular injury has been initiated, the inflammatory process can lead to pancreatic oedema, haemorrhage and, eventually, necrosis.

- **Classification :**

I. Marseilles' classification

1. Acute pancreatitis.
2. Acute relapsing pancreatitis.
3. Chronic relapsing pancreatitis.
4. Chronic pancreatitis.

Acute means acute inflammatory changes which are reversible.

Chronic means chronic continuous inflammatory changes which are morphologically irreversible.

II. **Trapnell's aetiological classification:**

- Biliary tract disease 50%—stones—most common cause
- Alcoholism 25%

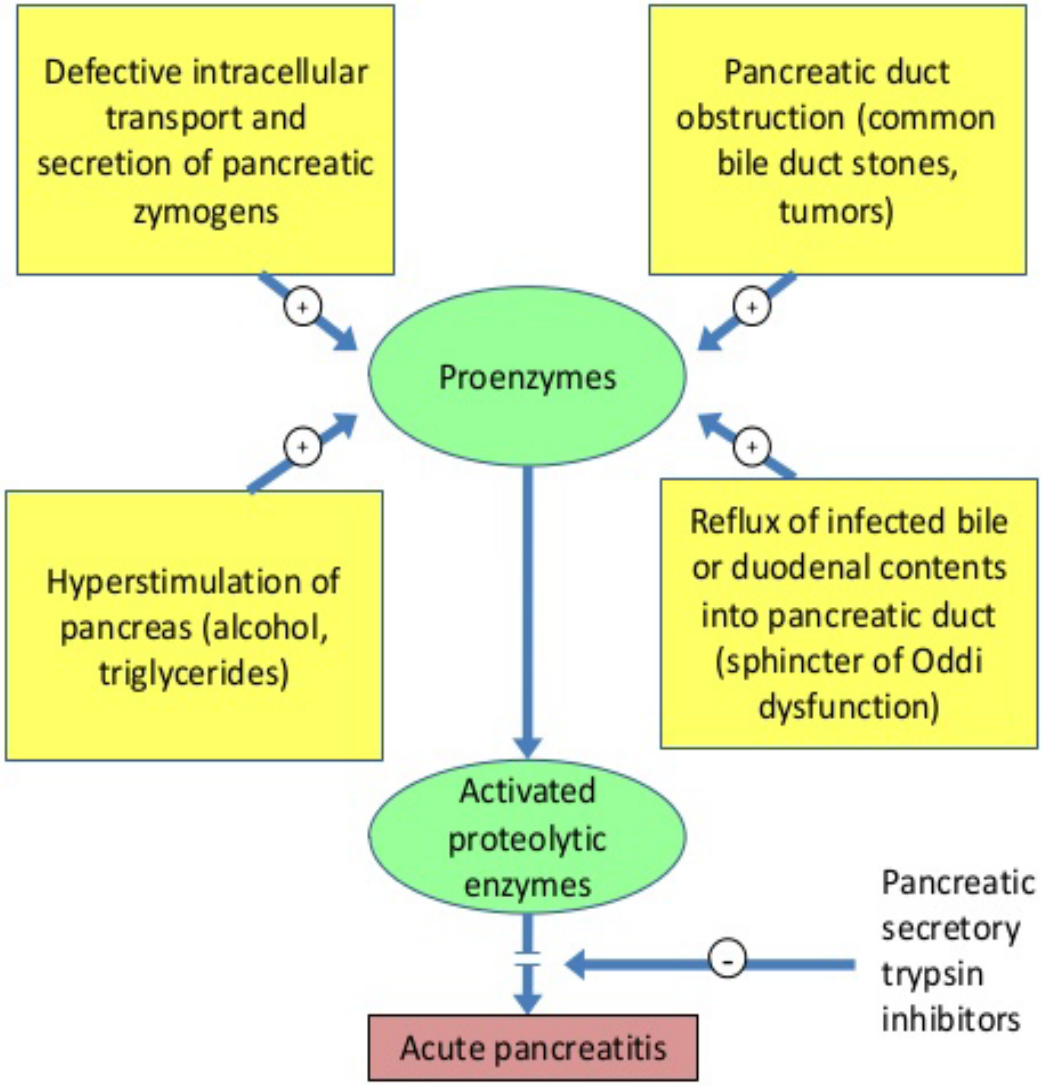
- Other causes
- Trauma After biliary, gastric, splenic surgery, ERCP
- Hyperparathyroidism ,Hypercalcaemia, hyperlipidaemia
- Diabetes
- Porphyria
- Drugs: Steroids, INH, diuretics (thiazides), septran, azathioprine, 5-amino salicylic acid, estrogens, tetracycline, valproic acid, petamidine – 5%
- Viral infections (mumps, coxsackie)
- Idiopathic
- Scorpion venom—Common in Trinidad
- Biliary ascariasis, Clonorchis sinensis Mycoplasma pneumoniae
- Infectious mononucleosis
- Pancreatic divisum

ACUTE PANCREATITIS

- Acute pancreatitis is an acute inflammation of the prior normal gland parenchyma which is usually reversible (but acute attack can occur in a pre-existing chronic pancreatitis) with raised pancreatic enzyme levels in blood and urine.

- Biliary tract disease is the commonest cause of acute pancreatitis.
- Early phase lasts for 2 weeks with oedematous pancreatitis or sterile necrosis which carry less mortality; death here occurs by multiorgan failure.

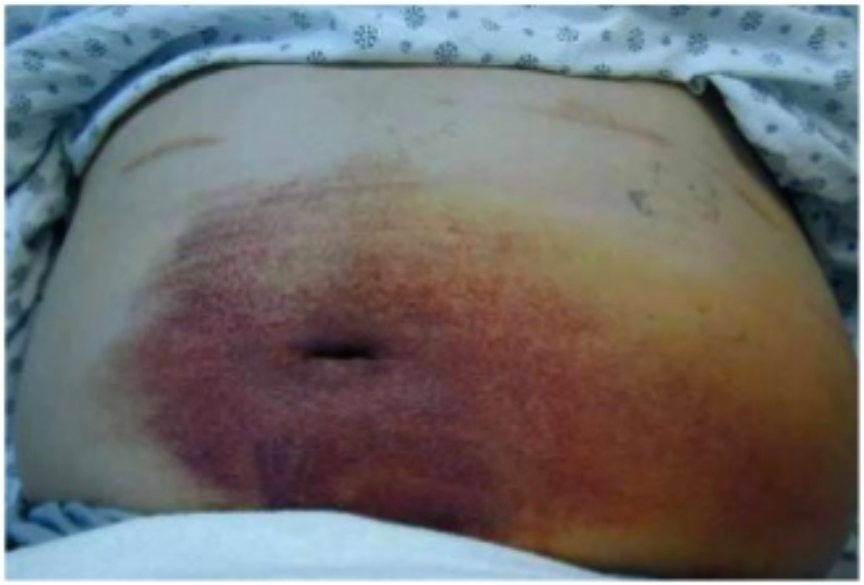
- In late phase after 2–3 weeks pancreatic abscess or infective necrosis can occur. Pancreatic abscess has got 40% mortality which needs drainage. Infective necrosis usually develops after 3 weeks and carries 100% mortality without surgical drainage (necrosectomy).


Defective intracellular transport and secretion of pancreatic zymogens
Pancreatic duct obstruction (common bile duct stones, tumors)
Proenzymes
Hyperstimulation of pancreas (alcohol, triglycerides)
Reflux of infected bile or duodenal contents into pancreatic duct (sphincter of Oddi dysfunction)
Activated proteolytic enzymes
Pancreatic secretory trypsin inhibitors
Acute pancreatitis
pathophysiology

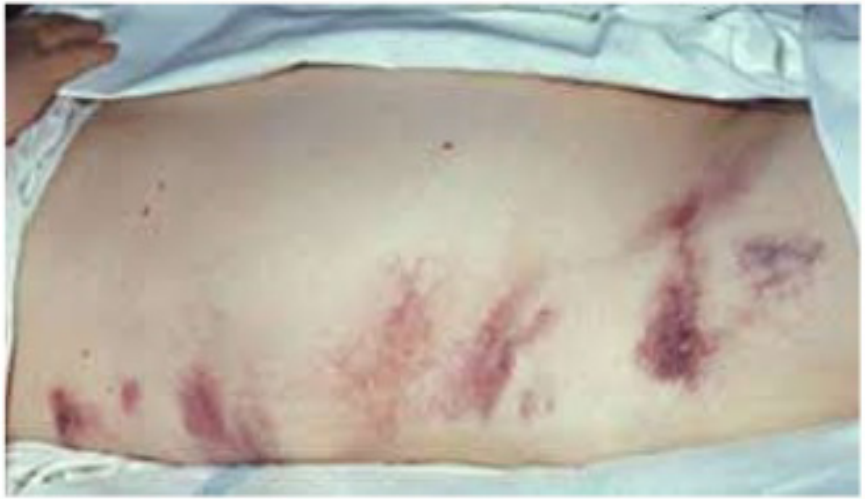
- Toxins released may lead to acute tubular necrosis and so acute renal failure.
- Left sided diaphragm gets elevated and left sided pleural effusion occurs.
- Lecithinase reduces the surfactant in the alveoli of lung, and infection leads to pulmonary insufficiency, ARDS and respiratory failure.
-
- Diffuse oozing in pancreatic bed occurs which utilizes platelets and causes disseminated intravascular coagulation (DIC).

- In severe cases, extensive necrosis with haemorrhage occurs causing acute haemorrhagic necrotising pancreatitis (Fulminant pancreatitis), which has got a high mortality.

- Here enzymes seep across the retroperitoneum causing haemorrhagic spots and ecchymosis in the **flanks (Grey-Turner's sign)**, or through falciform ligament causing discolouration around the umbilicus **(Cullen's sign),** umbilical black eye or below the inguinal ligament **(Fox sign).**



Cullen's sign



Grey Turner's sign

- **Organisms Involved :**

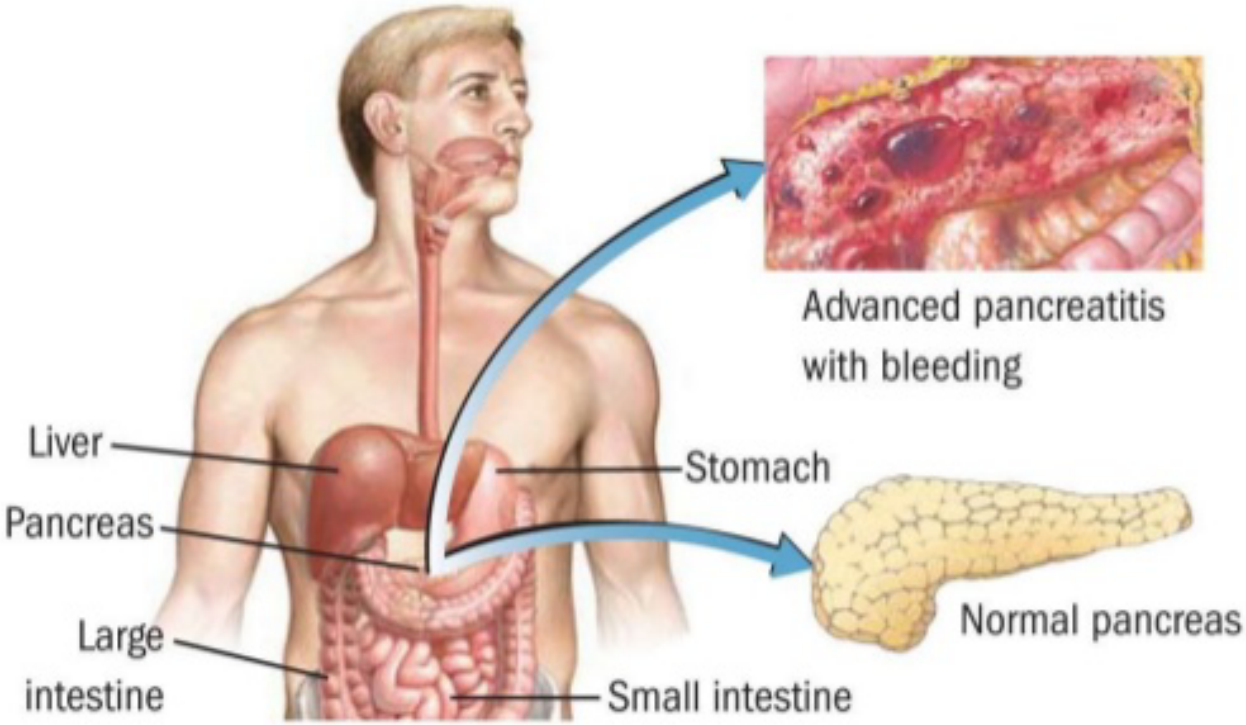
- Infection is commonly polymicrobial (60%). It may be from gallbladder, colon or small bowel via transmural migration or by haematogenous spread. Infection rate in one week is 24% and in 3 weeks it is 70%.

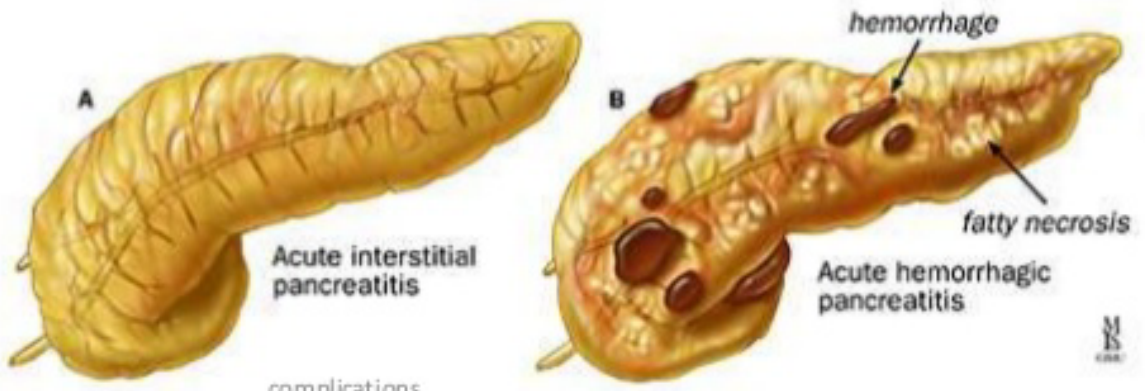
- E. coli (35%).
- Klebsiella (25%).
- Enterococcus (25%).
- Others—staphylococci, Pseudomonas, Proteus, Enterobacter, Anaerobes, Candida (10%).

- **Clinical Features:**
- Sudden onset of upper abdominal pain which is referred to back. Pain is severe, agonizing and refractory. Pain may be relieved or reduced by leaning forward.
- Vomiting and high fever, tachypnoea with cyanosis.
- Tenderness, rebound tenderness, guarding, rigidity and abdominal distension, severe illness.
- Often mild jaundice (due to cholangitis). Jaundice may also be due to bile duct disease/obstruction or cholestasis.
- Features of shock and dehydration.
- Oliguria, hypoxia and acidosis.
- Haematemesis/malaena due to duodenal necrosis, gastric erosions, decreased coagulability/DIC.
- Hiccough when present is refractory.
- Ascites may be present. Paralytic ileus is common.
- Pleural effusion (20%), pulmonary oedema, consolidation, features of rapid onset ARDS is often observed.
- Neurological derangements due to toxaemia, fat embolism, hypoxia, respiratory distress can occur. It may be mild psychosis to coma.

- **Differential diagnosis :**

- Perforated duodenal ulcer
- Cholecystitis
- Mesenteric ischaemia
- Ruptured aortic aneurysm
- Ectopic pregnancy
- Salpingitis
- Intestinal obstruction


Advanced pancreatitis
with bleeding
Liver
Pancreas
Large
intestine
Stomach
Small intestine
Normal pancreas


A
Acute interstitial
pancreatitis
B
hemorrhage
fatty necrosis
Acute hemorrhagic
pancreatitis

- **Investigations:**

- Serum amylase is very high (>1000 Somogyi units) or shows rising titre.
- Amylase creatinine clearance ratio is increased.. Normal value is 1–4%. More than 6% signifies acute pancreatitis.
- Serum lipase more specific than amylase. Serum lipase level after rise persists for longer period than amylase. Pancreas is the only source unlike amylase, hence more specific.
- Trypsinogen activation polypeptide (TAP) assay in serum and urine reveals the severity of the acute pancreatitis. CRP (>150 mg/L) is also useful. Phospholipase A2, LDH levels are also often assessed.
- Liver function tests: Serum bilirubin, albumin, prothrombin time, alkaline phosphatase.
- Blood urea, serum creatinine.
- Blood glucose (hyperglycaemia is seen).
- Serum calcium level (hypocalcaemia occurs)

- Total count, haematocrit, platelet count, coagulation profile.
- Peritoneal tap fluid shows high amylase and protein level (very useful method). Lipase level in ascitic fluid is also useful.

- Plain X-ray shows –

  - 'Sentinel loop' of dilated proximal small bowel.

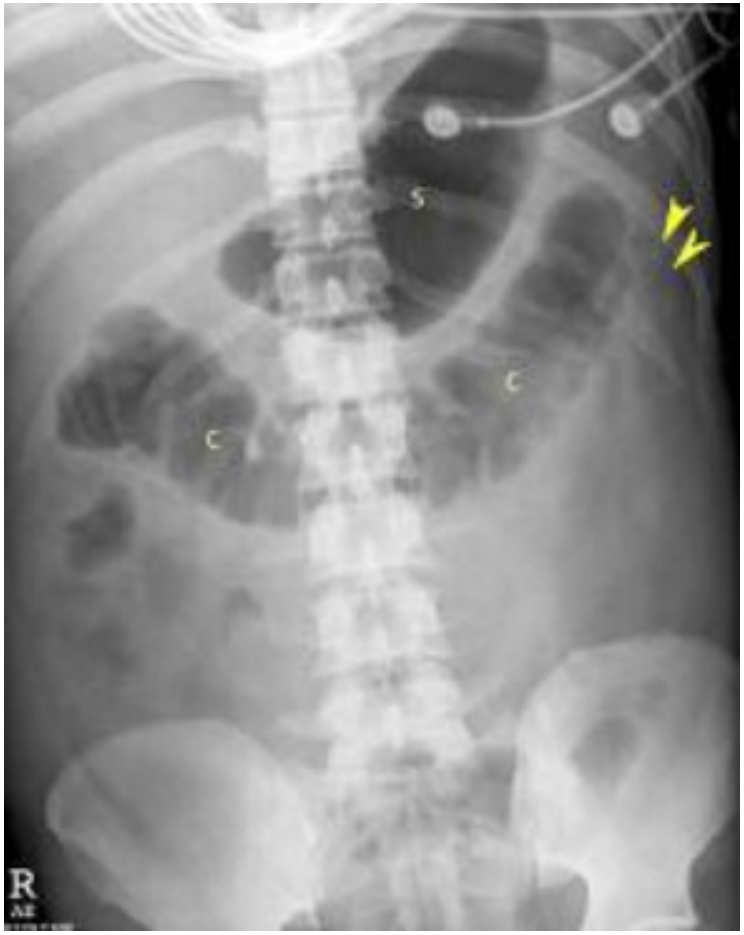
  -Distension of transverse colon with collapse of descending colon (colon cut off sign).

  -Air-fluid level in the duodenum.

  - Renal halo sign.

  -Obliteration of psoas shadow.

  - Localized ground glass appearance.



COLON CUTOFF SIGN IN ACUTE PANCREATITIS

- US abdomen.
- Spiral CT (CECT–contrast enhanced CT) is better—gold standard. It is done after 72 hours. To look for oedema, altered fat and fascial planes, fluid collections, necrosis (non-enhancement area > 30% or 3 cm), bowel distension, mesenteric oedema and haemorrhage.
- MRI, MRCP—should be done at a later date.
- ERCP is usually not done in acute phase.
- Chest X-ray for effusion and ARDS.
- EUS—to see necrosis, calcifications and to assess CBD.

- **Treatment of acute pancreatitis:**

- Conservative, 70–90%

- Surgical treatment when indicated, 10–30%

- Management of complications like acute pseudocyst, abscess, fistula, haemorrhage; systemic complications like ARDS, renal failure, MODS.

- **Conservative Treatment:**

- Rehydration is essential (250–400 ml/hour) as there is lot of fluid sequestration and 3rd space fluid loss. It is done by using ringer lactate, normal saline, dextrose saline, plasma and fresh blood transfusion/packed cells.

- Pain relief by pethidine and other analgesics. Morphine is not used as it causes spasm of sphincter of Oddi.

- In severe haemorrhagic episodes, fresh frozen plasma and platelet concentrate may be required in anticipation of DIC and haemorrhage.

- Nasogastric aspiration, urinary catheterisation to maintain and monitor urine output 50 ml hourly. Nasojejunal tube placement for feeding is very useful.

- Antibiotics like third generation cephalosporins, imipenem, meropenem, cefuroxime are commonly used to reduce the anticipated sepsis.

- CVP line is essential to monitor, for rapid fluid therapy and for Total Parenteral Nutrition (TPN) using carbohydrate, amino acids, vitamins, essential elements.

- Proper electrolyte management with monitoring is needed.

- It is always better to manage the patient in an intensive care set up so that when needed endotracheal intubation, ventilatory support, tracheostomy can be done as an emergency basis.

- Management of complications like acute lung injury, atelectasis, renal failure, GI bleeding, metabolic encephalopathy, electrolyte deficiency as and when needed by repeated observation and evaluation.

- **Surgery Indications:** (10% cases)

1. If condition of patient deteriorates in spite of good conservative treatment.
2. If there is pancreatic infected necrosis.
3. In severe necrotising pancreatitis as a trial to save the life of the patient which has got very high mortality.

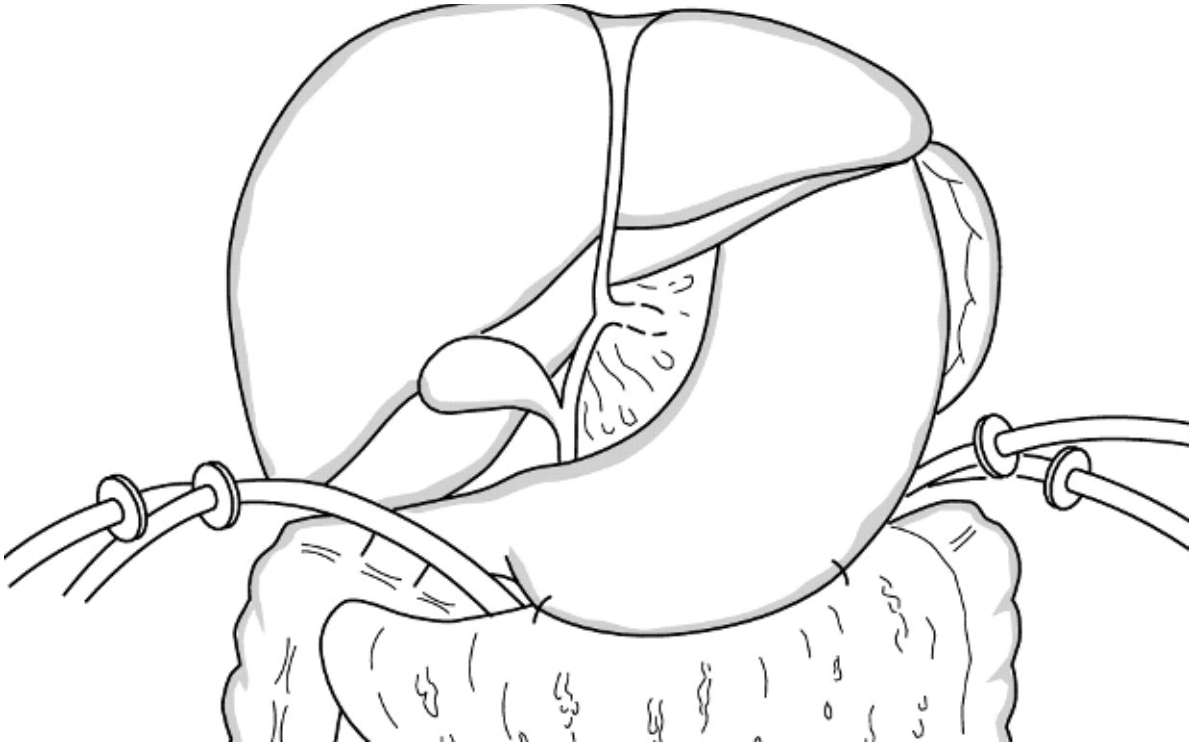
- Surgery removes intra- and extra-pancreatic necrotic materials, pancreatic fluid, and toxins. It permits preservation of viable pancreatic tissue. Open surgery is the gold standard for infected pancreatic necrosis.

- After opening the abdomen, all necrotic tissue, pus, infected fluid and toxins are removed. 10–12 litres of normal saline wash is given. Drainage tubes are placed liberally. Abdomen is closed in layers—**Conventional closed method** (necrosectomy, wide debridement, adequate drainage, cholecystectomy, closure). Re-laparotomy is done only on demand later.

- Laparotomy–necrosectomy–wide debridement–wash–wide packing. Wound is left open. Repeated wash and packings are done until healthy granulation develops – **open method.**

- Laparotomy–necrosectomy and closure with drain and re-laparotomy later—**semi-open method.**

- Continuous closed peritoneal lavage of the pancreatic bed and lesser sac is done with 10–12 litres of normal saline or hyperosmolar, potassium free dialysate fluid 2 litres/hour using multiple tubes to remove toxic material in the peritoneal cavity/retroperitoneal area until return fluid becomes clear—**Beger's lavage**. Procedure is done after initial surgical debridement.



COMPLICATIONS OF ACUTE PANCREATITIS :

- Shock—Hypovolemic and septic
- Respiratory failure and ARDS—Common in 7 days
- Septicaemia—Common after 7 days
- Hypocalcaemia
- Disseminated intravascular coagulation (DIC)
- Acute renal failure
- Pancreatic pleural effusion (left sided 20%)
- Pancreatic pseudoaneurysm
- Pancreatic ascites
- Colonic stricture
- Pseudocyst of pancreas
- Chronic pancreatitis
- Splenic vein thrombosis
- Abdominal compartment syndrome (ACS)
- Pancreatic endocrine (15%) and exocrine (20%) insufficiency as late sequelae can occur.