

# LIVER

The liver is the **largest organ** of the human body, weighs approximately 1500 g, and is located in the **upper right corner of the abdomen**.

The organ is closely associated with the small intestine, **processing the nutrient** enriched venous blood that leaves the digestive tract.

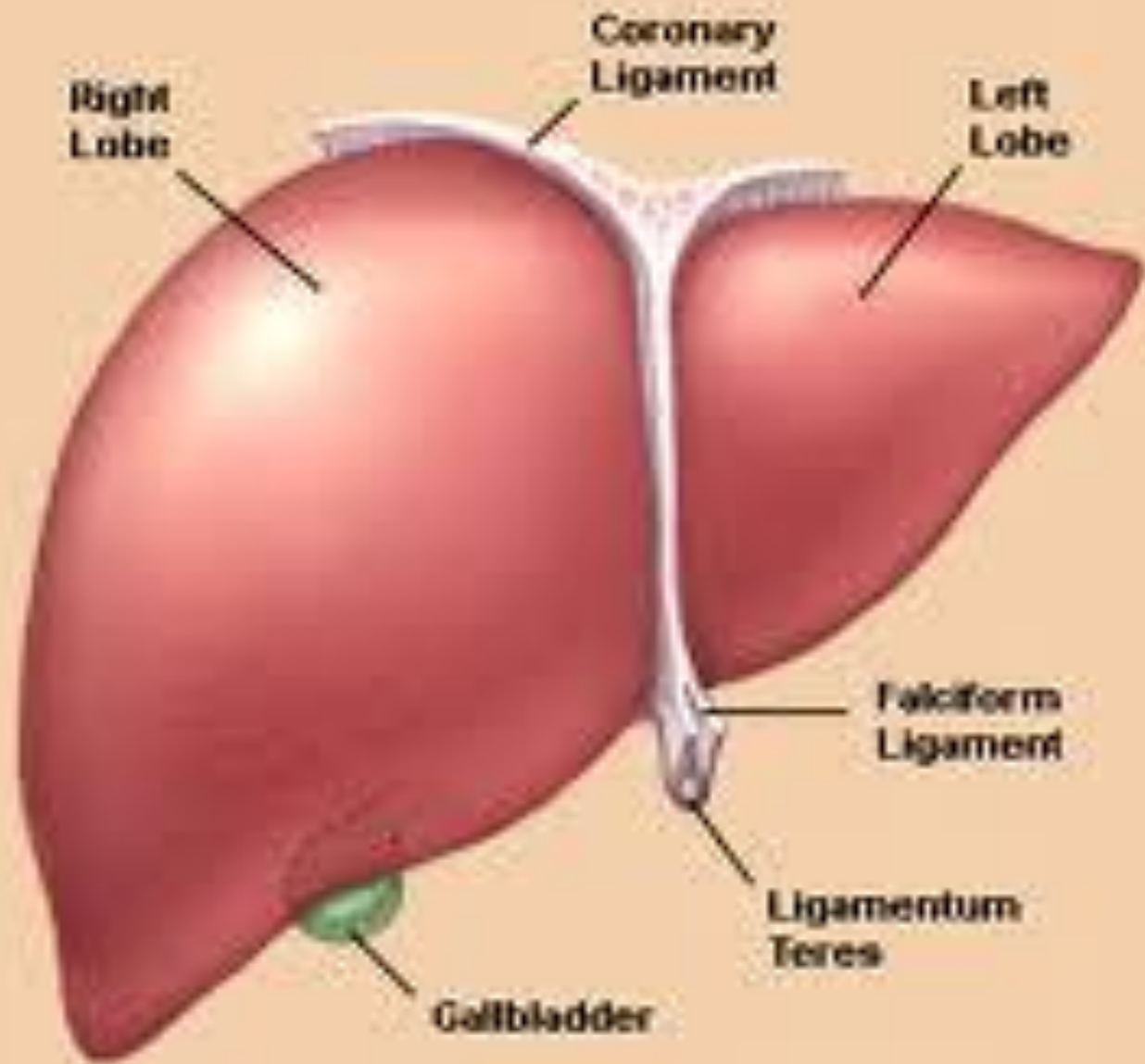
The liver performs over 500 metabolic functions, resulting in **synthesis of products** that are released into the blood stream (e.g. glucose derived from glycogenesis, plasma proteins, clotting factors and urea), or that are **excreted to the intestinal tract (bile)**.

# LIVER

From a surgical point of view, the liver is divided into **right and left lobes of almost equal size** by a **major fissure (Cantlie's line)** running from the gallbladder fossa in front to the IVC fossa behind. This division **is based on the right and left branches of the hepatic artery and the portal vein with tributaries of bile (hepatic) ducts following**. The middle hepatic vein (MHV) lies in Cantlie's line.

The left pedicle (left hepatic artery [LHA], left branch of the portal vein, and left hepatic duct has a longer extrahepatic course than the right.

# LIVER



The liver has **dual blood supply**:

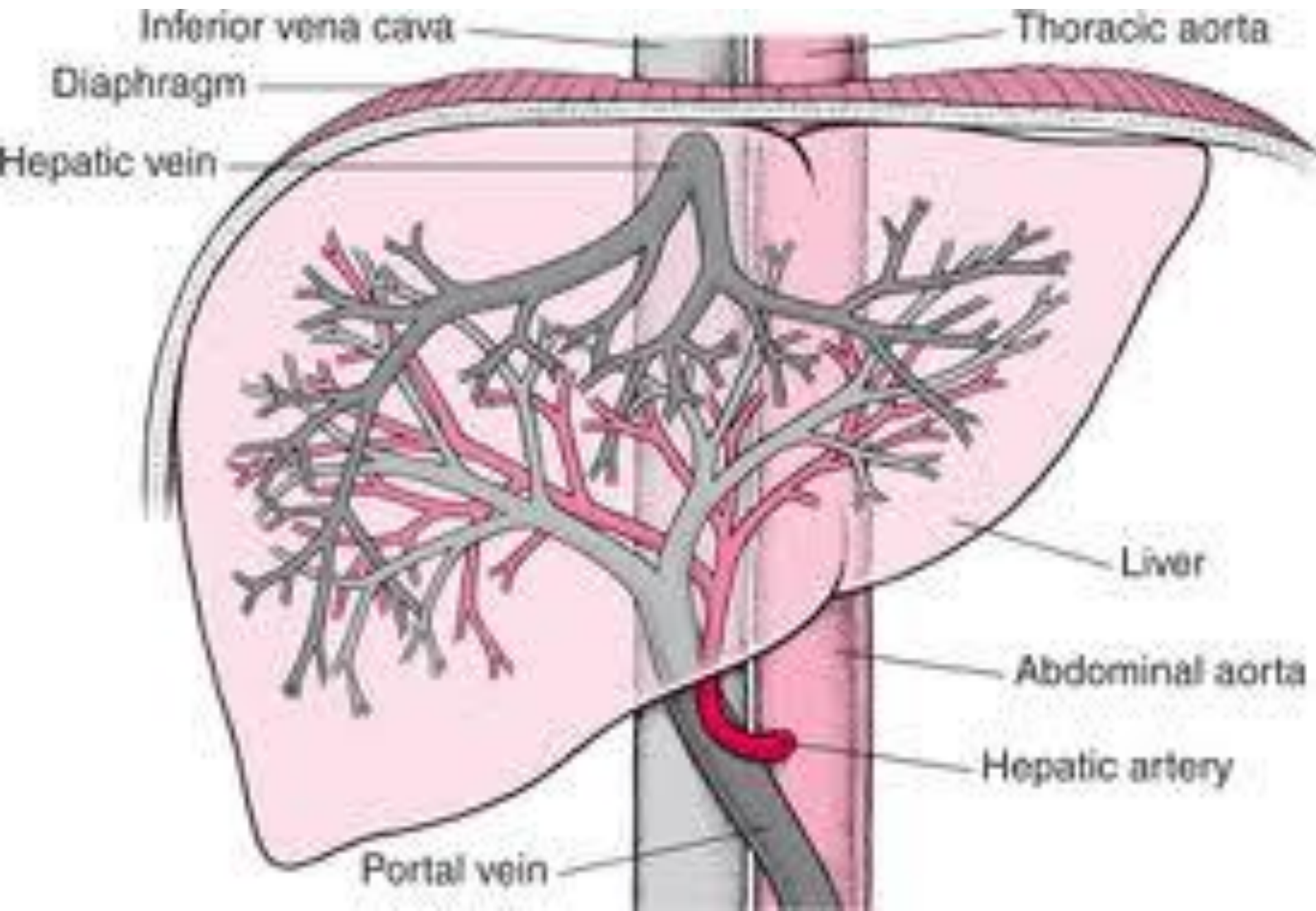
the **hepatic artery**, which branches from the celiac artery, and

the **portal vein**, which drains the intestine.

Blood from the liver passes through the **hepatic vein** and enters **the inferior vena cava**.



# LIVER



## Histologically,

the liver is divided into **lobules** with central veins draining each lobule.

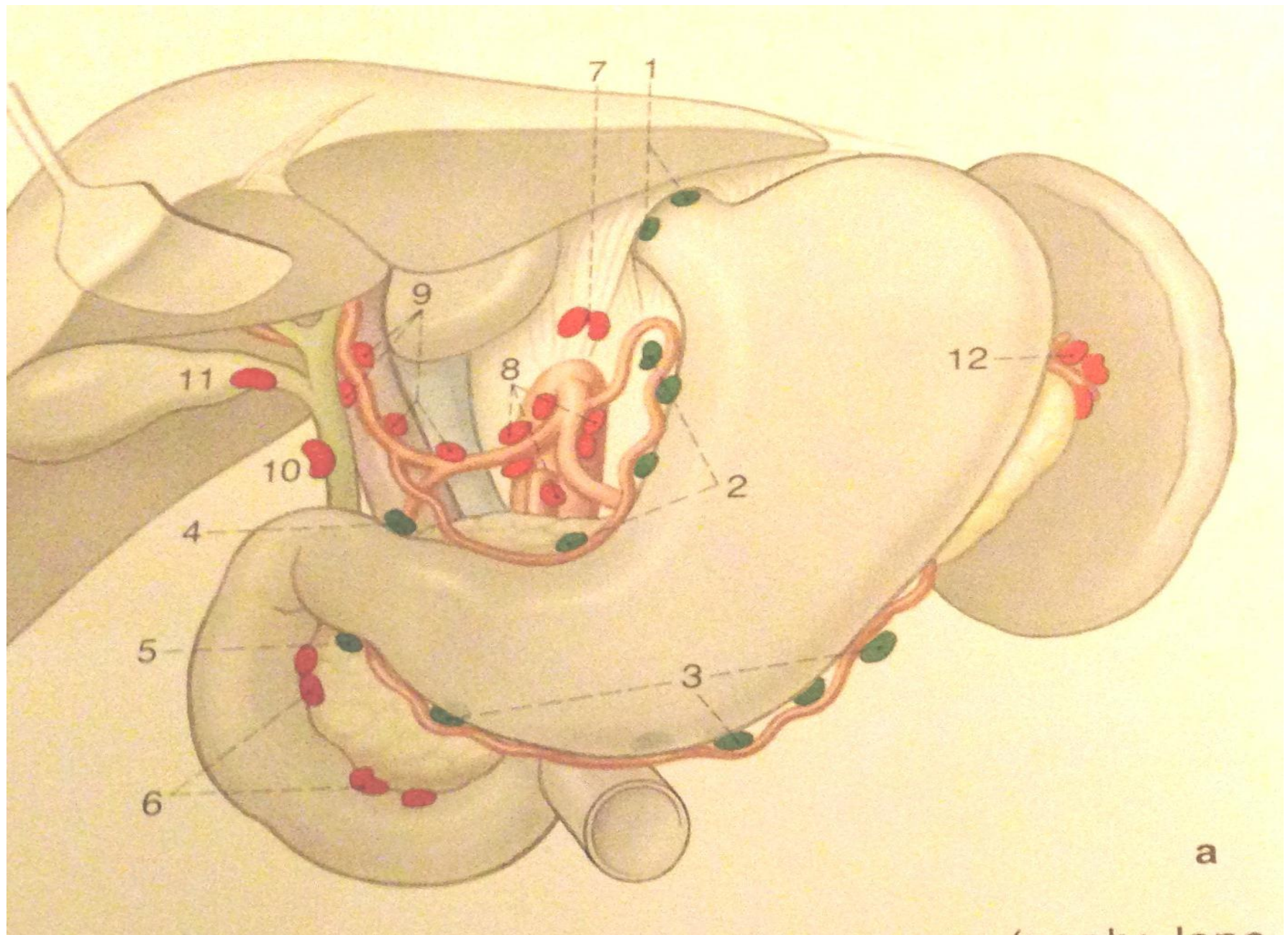
The portal spaces between the lobules contain:

- the intrahepatic **bile ducts** and
  - the blood supply, which consist of small branches of the **hepatic artery** and
  - portal vein**
- (**portal triads**)

## **Regional lymph nodes**

- **Hilar**
- **Hepatoduodenal ligament**
- **Caval lymph nodes**

**The most prominent are hepatic artery and portal vein lymph nodes**





# LIVER

Each lobe is divided into **2 sectors**.

The **right hepatic vein** (RHV) divides the right lobe into **anterior and posterior** sectors;

the **left hepatic vein** (LHV) divides the left lobe into **medial (quadrate) and lateral** sectors.

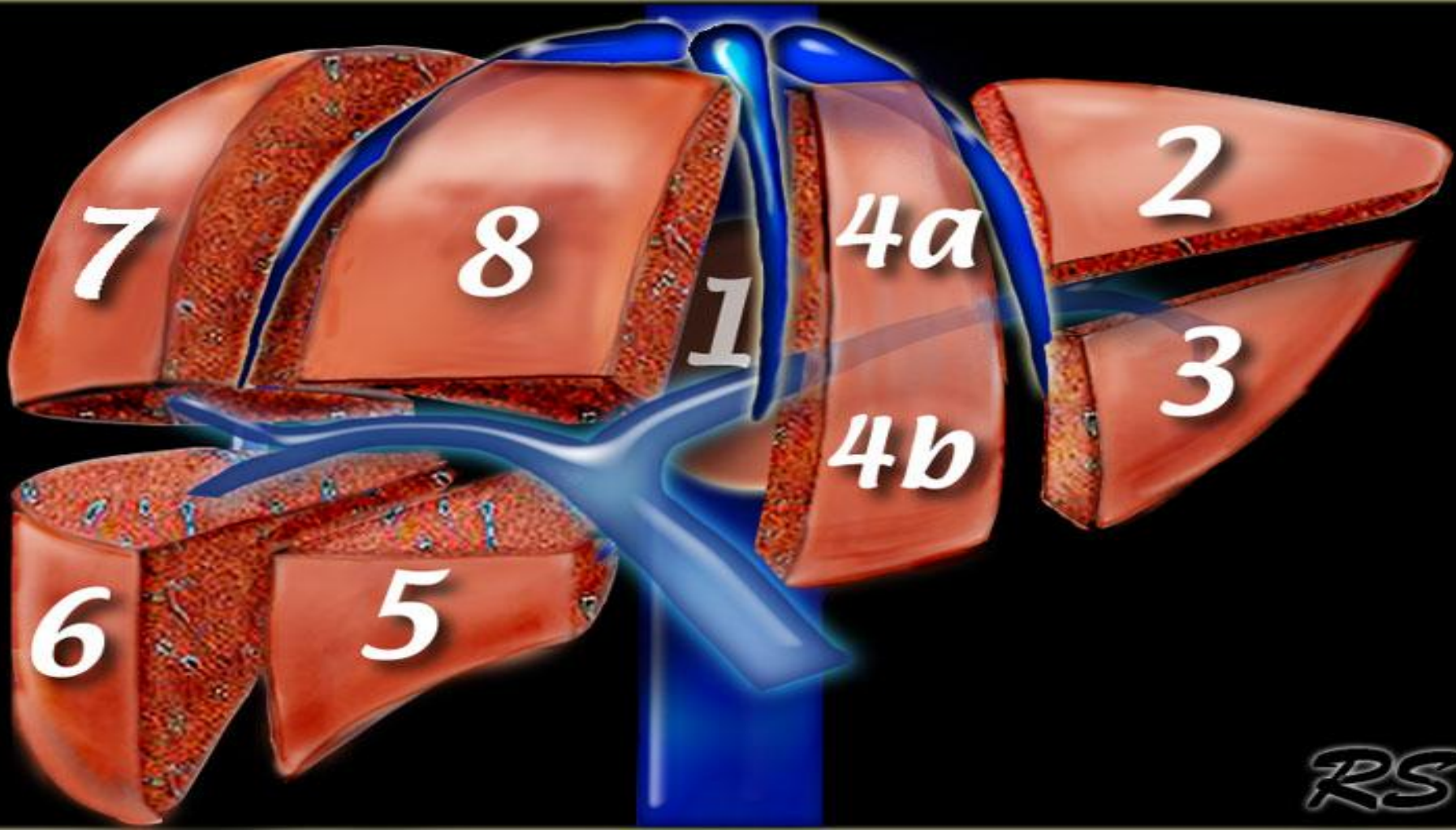
The posterior sector of the right lobe and the caudate lobe are not seen on a frontal view of the liver;

the anterior sector of the right lobe forms the right lateral border in this view.

The sectors are further divided into segments (after Couinaud);

# LIVER

**The Couinaud classification:** divides the liver into **eight functionally independent segments**. Each segment has **its own vascular inflow, outflow and biliary drainage**. In the centre of each segment there is a branch of the portal vein, hepatic artery and bile duct. In the periphery of each segment there is vascular outflow through the hepatic veins.



RS

# LIVER

## Segments numbering

There are eight liver segments. **Segment 4** is sometimes divided into segment 4a and 4b according to Bismuth. The numbering of the segments is in a **clockwise manner**.

**Segment 1** (caudate lobe) is located posteriorly. It is not visible on a frontal view. The caudate lobe is anatomically different from other lobes in that it often has direct connections to the IVC through hepatic veins, that are separate from the main hepatic veins. The caudate lobe may be supplied by both right and left branches of the portal vein.

**Right hepatic vein** divides the right lobe into anterior and posterior segments.

**Middle hepatic vein** divides the liver into right and left lobes (or right and left hemiliver). This plane runs from the inferior vena cava to the gallbladder fossa.

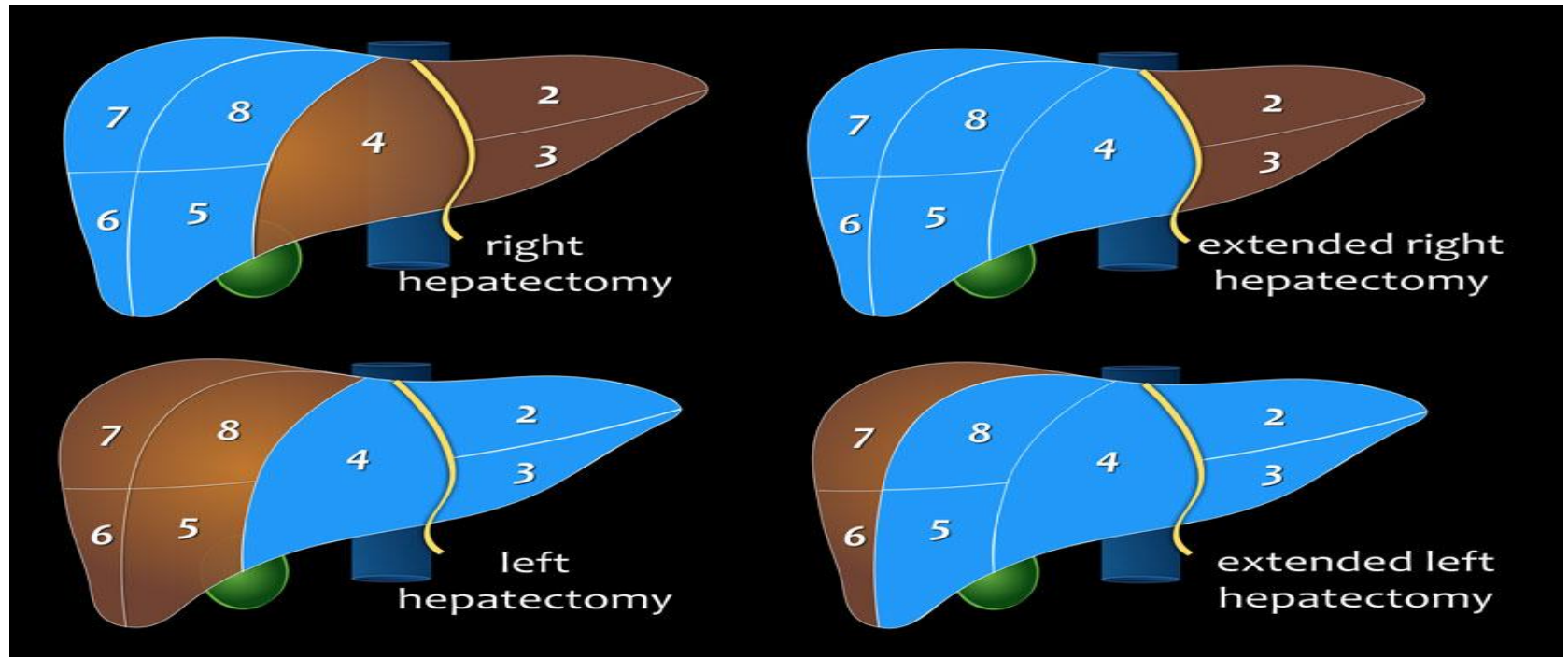
**Left hepatic vein** divides the left lobe into a medial and lateral part.

**Portal vein** divides the liver into upper and lower segments. The left and right portal veins branch superiorly and inferiorly to project into the center of each segment.

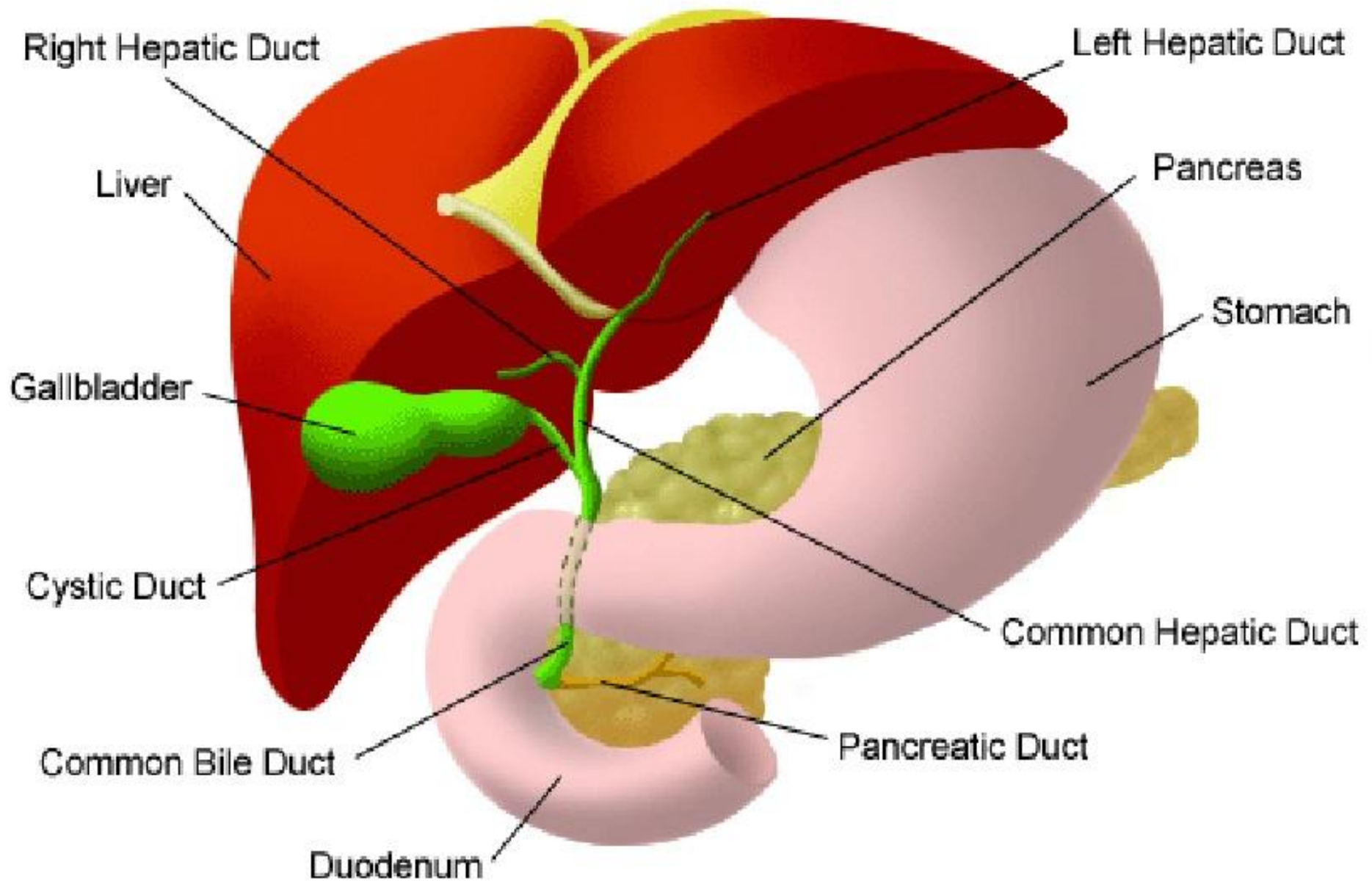


Because of this division into self-contained units, each segment **can be resected without damaging** those remaining. For the liver to remain viable, resections must proceed along the vessels that define the peripheries of these segments. This means, that resection-lines parallel the hepatic veins, the centrally located portal veins, bile ducts, and hepatic arteries are preserved.

### Typical resections along these planes



# Biliary System



# **LIVER DISEASES**

- CONGENITAL MALFORMATIONS (LIVER CYSTIC DISEASE)**
- INFLAMMATOR DISEASES**
- PARASITIC DISEASES**
- TUMORAL DISEASES**
- TRAUMATIC DISEASES**

# **Cystic disease of the biliary tree**

**This can take several forms:**

- (1) cysts in the main trunk (choledochal cysts),**
- (2) cysts (or lakes) in the small branches within the liver (Caroli's syndrome) or**
- (3) cysts in the liver separate from the biliary tree (polycystic liver disease).**

## **Caroli's syndrome**

**Caroli's syndrome (intrahepatic ductal ectasia) is a rare congenital disease. It is probably inherited. In this syndrome, the small branches of the biliary tree in the liver are abnormal. Small lakes alternate with narrowed segments of bile ducts, instead of the normal smooth contour.**

**These abnormalities may be present throughout the liver, or limited to only a small area.**

**If the bile duct becomes infected, the patient develops fever, abdominal pain and, rarely, jaundice.**

# Polycystic Liver Disease

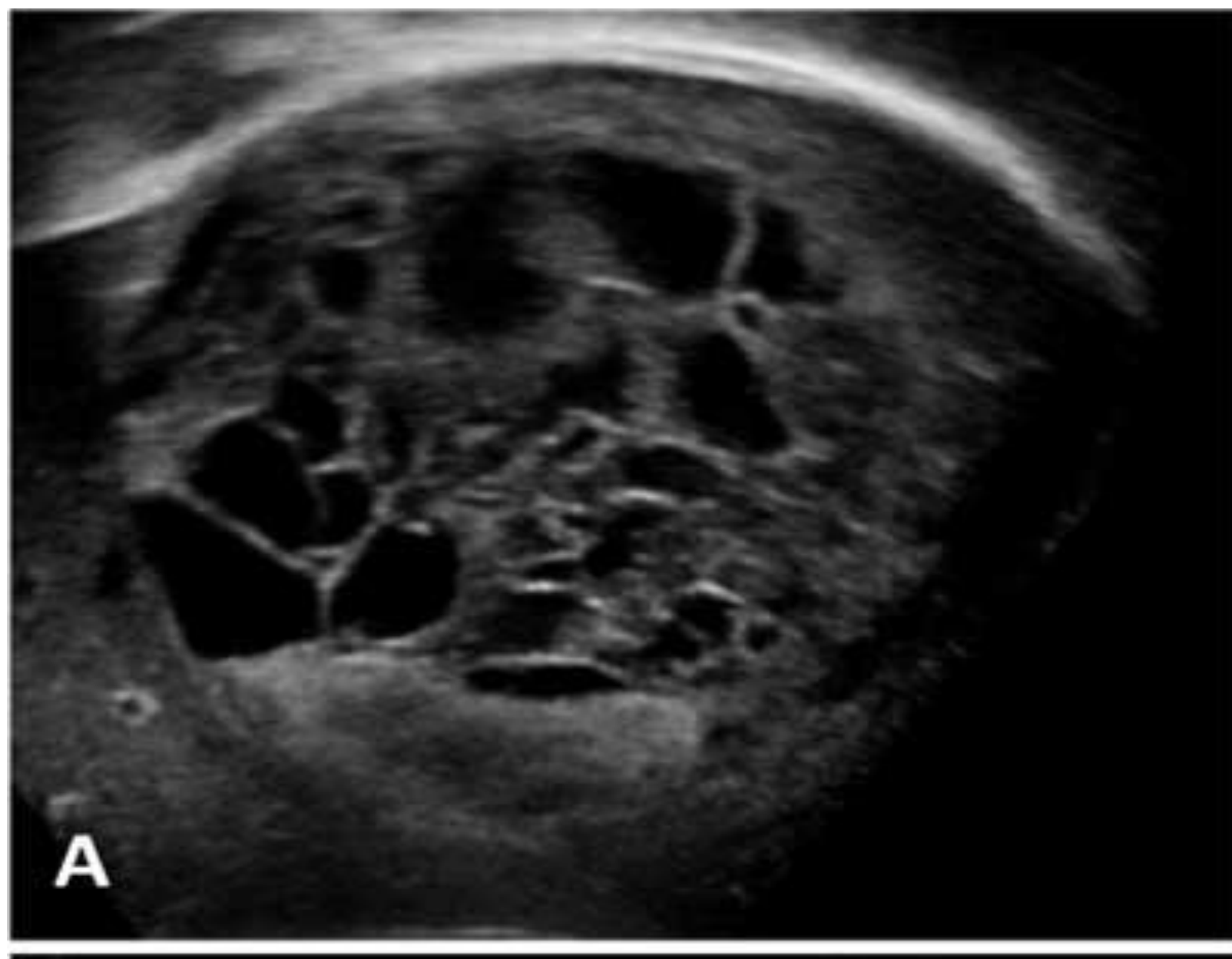
**In some patients, large lakes (cysts) separate from the biliary tree are formed in the liver.**

**In severe cases, the liver looks like a **sponge**.**

**These cysts may cause **pain**, but do not affect liver function.**

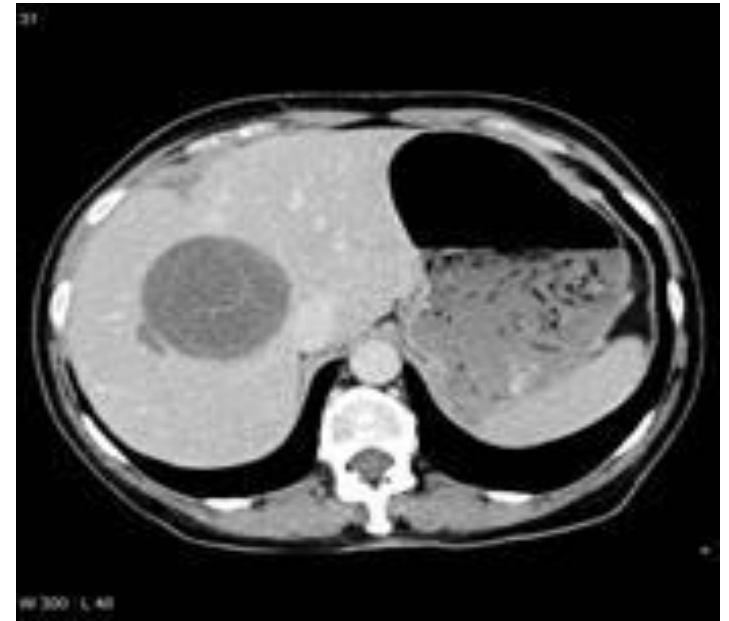
**In most patients, the kidneys are similarly affected with cysts, which may cause high blood pressure and kidney failure.**

**The tendency to form cysts is probably present at birth in these patients, but usually the cysts do not enlarge and give problems until adulthood.**



# Polycystic Liver Disease

**This condition may be detected using ultrasound or CAT scan.**



**Polycystic disease is inherited and once it has been detected in one member of a family, all the patient's relatives should be tested for it. There are two major categories of polycystic disease of the liver and kidney. In the more benign, the cysts are mostly in the liver and kidney function is near normal.**

# LIVER ABSCESS: Pyogenic abscess

## Pathogenesis:

the main etiological factor is bile duct infection with ascending cholangitis commonly due to **E.Coli** and anaerobic organisms.

Other sources of infection include an ascending pylephlebitis; it arises particularly with complicated diverticulitis.

Some hepatic abscesses of Staphylococcal and Streptococcal origin arise as a complication of generalized Septicemia.

Other arise by direct extension from suppurative cholangitis and under the diaphragm suppurations. Obviously trauma to the liver tissue and subsequent infection produces an abscess.

All types of abscesses are found more commonly in the right lobe.



# LIVER ABSCESS

## Pyogenic abscess

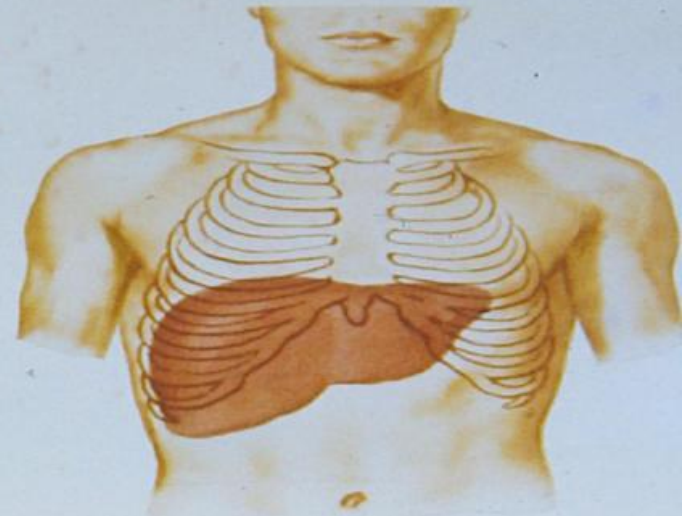
**Subjective findings:** chills, malaise, anorexia, nausea, vomiting, weight loss, and abdominal pain and discomfort.

**Objective findings:** fever, jaundice, and an enlarged tender liver.

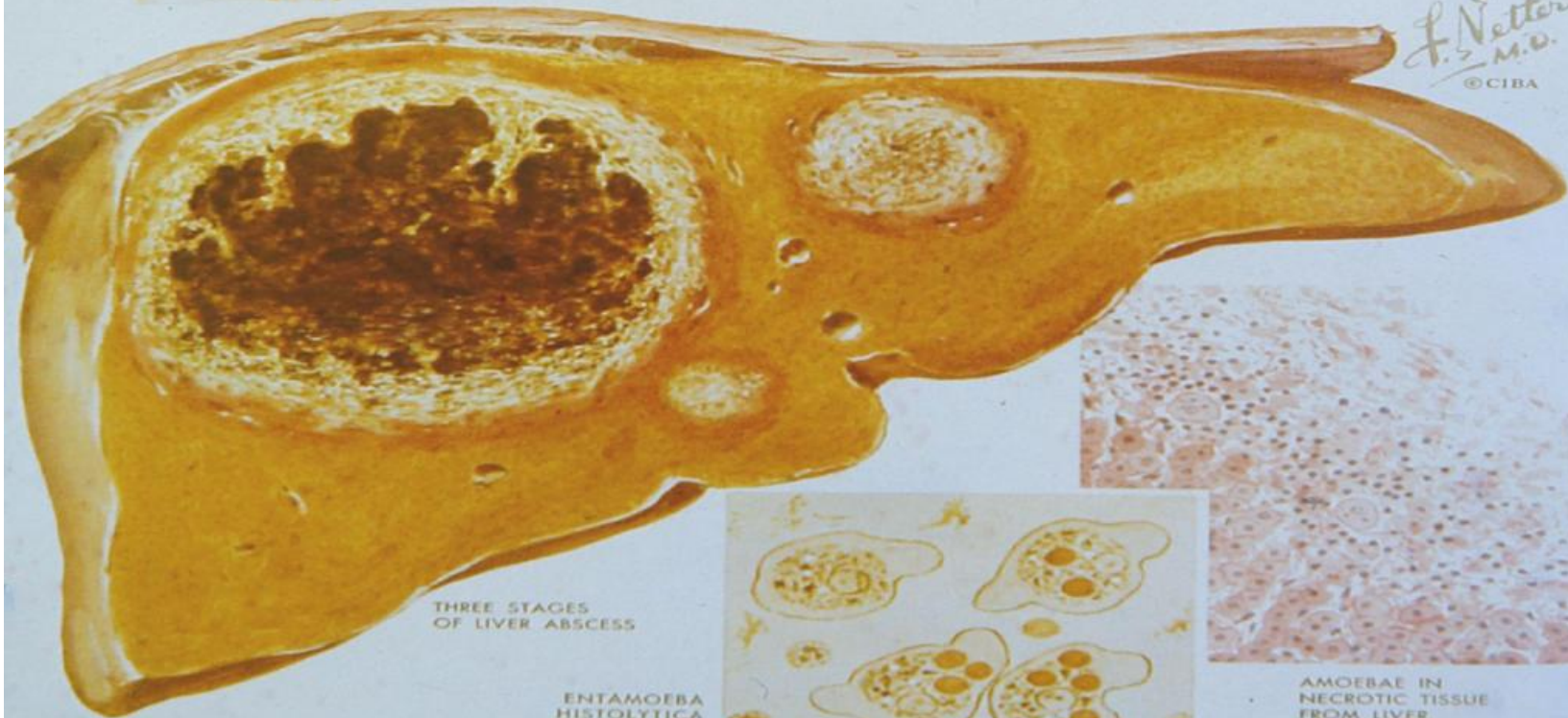
# LIVER



- PATHWAYS OF AMOEBIA
1. PORTAL OF ENTRY
  2. INTESTINAL FOCUS
  3. PORTAL VEIN ROUTE TO LIVER
  4. LIVER ABSCESS
  5. SUBHEPATIC ABSCESS
  6. SUBPHRENIC ABSCESS
  7. DIRECT EXTENSION TO LUNG ABSCESS
  8. VASCULAR ROUTE TO LUNG ABSCESS
  9. BRONCHIAL FISTULA
  10. BRAIN ABSCESS (VASCULAR ROUTE)



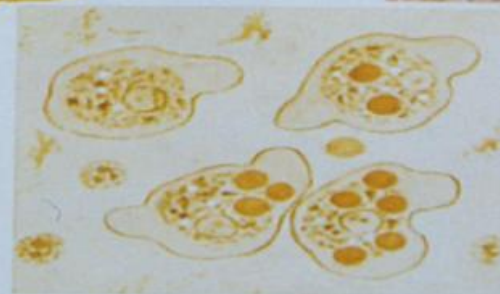
EARLY STAGE OF LIVER INVOLVEMENT:  
SWOLLEN, CONGESTED, TENDER



*F. Netter M.D.*  
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THREE STAGES  
OF LIVER ABSCESS

ENTAMOEBIA  
HISTOLYTICA  
IN STOOL  
(HANGING DROP  
PREPARATION)



AMOEBAE IN  
NECROTIC TISSUE  
FROM LIVER  
ABSCESS



# LIVER ABSCESS

## Pyogenic abscess

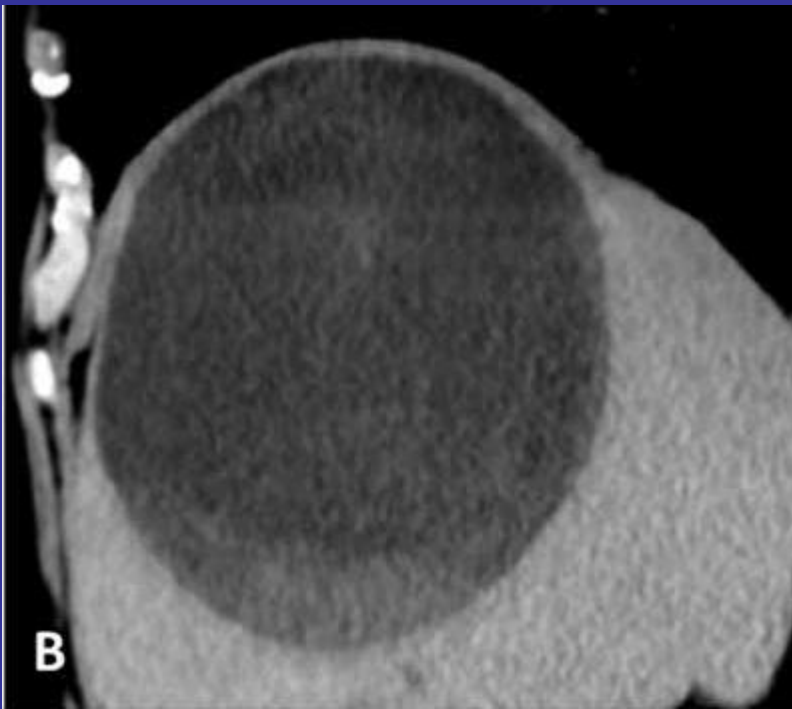
### Diagnostic plan:

- lab analysis (leukocytosis, anemia; alkaline phosphatase, SGOT, SGPT, serum bilirubin are elevated),
- Blood cultures
- chest roentgenogram: elevated diaphragm, pleural effusion on the right side or atelectasis,
- Abdominal roentgenogram: accumulation of gas in the area of the liver
- Liver isotope scans, ultrasound and CT scans may all help to localize a liver abscess.

**Management plan:** the treatment involves AB therapy, draining the abscess (surgically or percutaneously) and correcting any etiologic process.

# LIVER CYSTS

Liver cysts are usually not neoplastic.  
Nonparasitic cysts may be either  
**solitary or multiple.**



# LIVER CYSTS

## Treatment:

Small nonparasitic cysts require non treatment

Large benign cysts causing symptoms can be treating by:

- surgical excision or
- unroofing and external drainage, if excision is impossible because of proximity to the vascular or biliary ductal structures.

Cystadenomas, cystadenocarcinomas and cysts associated with other neoplasms should be excised.

# LIVER CYSTS

**Most common are due from infestation of Ecchinococcus.**

**Clinical manifestations** of hydatid cysts depend on the activity of the cysts, the localization, size, and stage of the cyst.

In asymptomatic patients without eosinophilia or positive Casoni skin tests the parasite may be dead.

**In active cysts: hepatomegaly, jaundice or pain, pruritus, and eosinophilia.**

Although liver hydatid cysts are usually asymptomatic, the most common symptoms are **pain and hepatomegaly**. **Fever and jaundice** may accompany complicated cysts.



# LIVER CYSTS

**Ultrasonography** is the primary diagnostic tool owing to its low cost, and high specificity and sensitivity.

**CT, MRI, and MRCP** may be used for better documentation and definition of the vascular/biliary anatomy.

Simple cysts and type 1 hydatid cysts of the liver are difficult to differentiate using radiologic modalities.

Serologic methods may be useful for the differential diagnoses in difficult cases

# Gharbi classification of hydatid cysts

Type I Pure fluid collection

Type II Fluid collection with a detached membrane

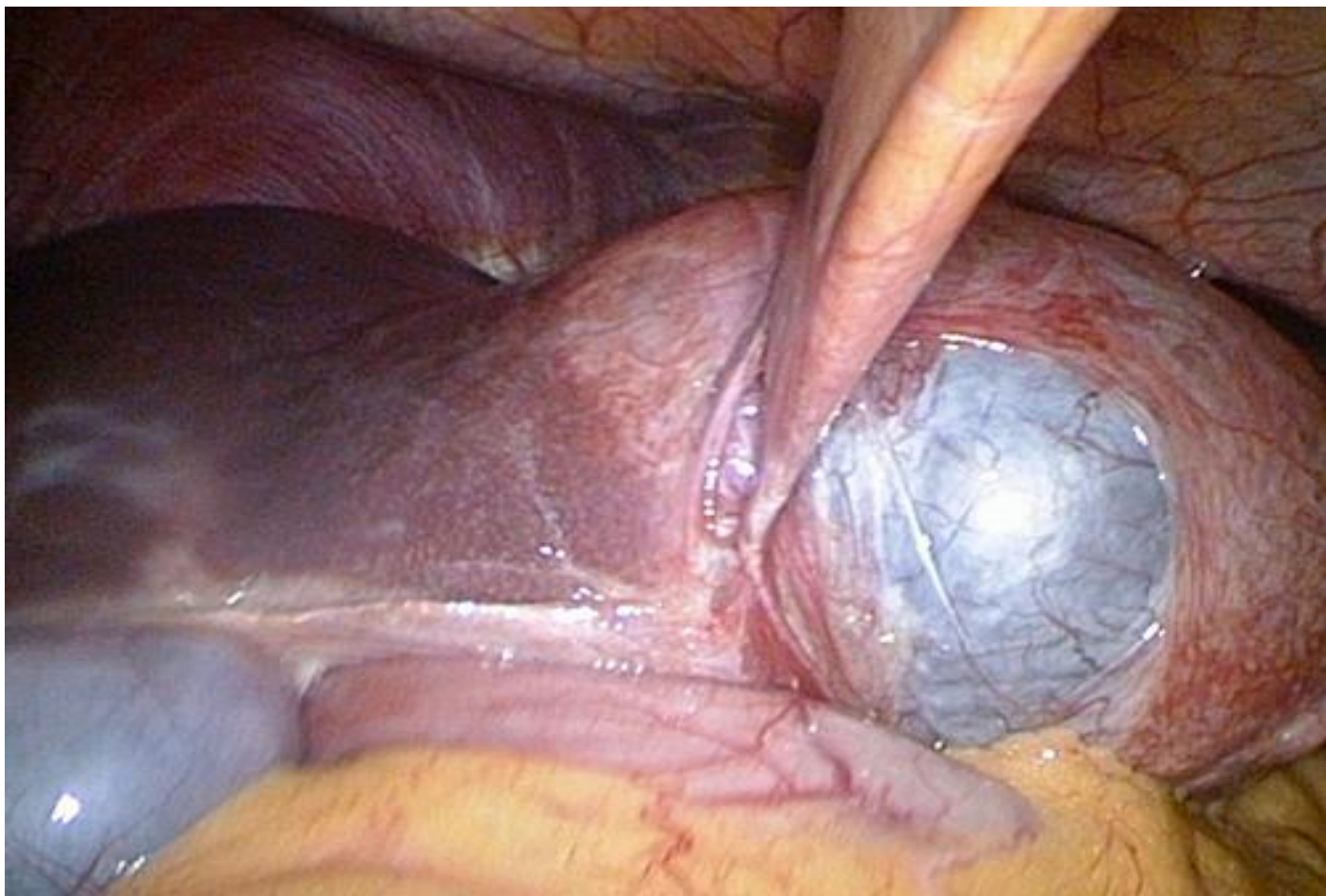
Type III Fluid collection with multiple septa and/or daughter cysts

Type IV Hyperechoic with high internal echoes

Type V Cyst with reflecting calcified thick wall



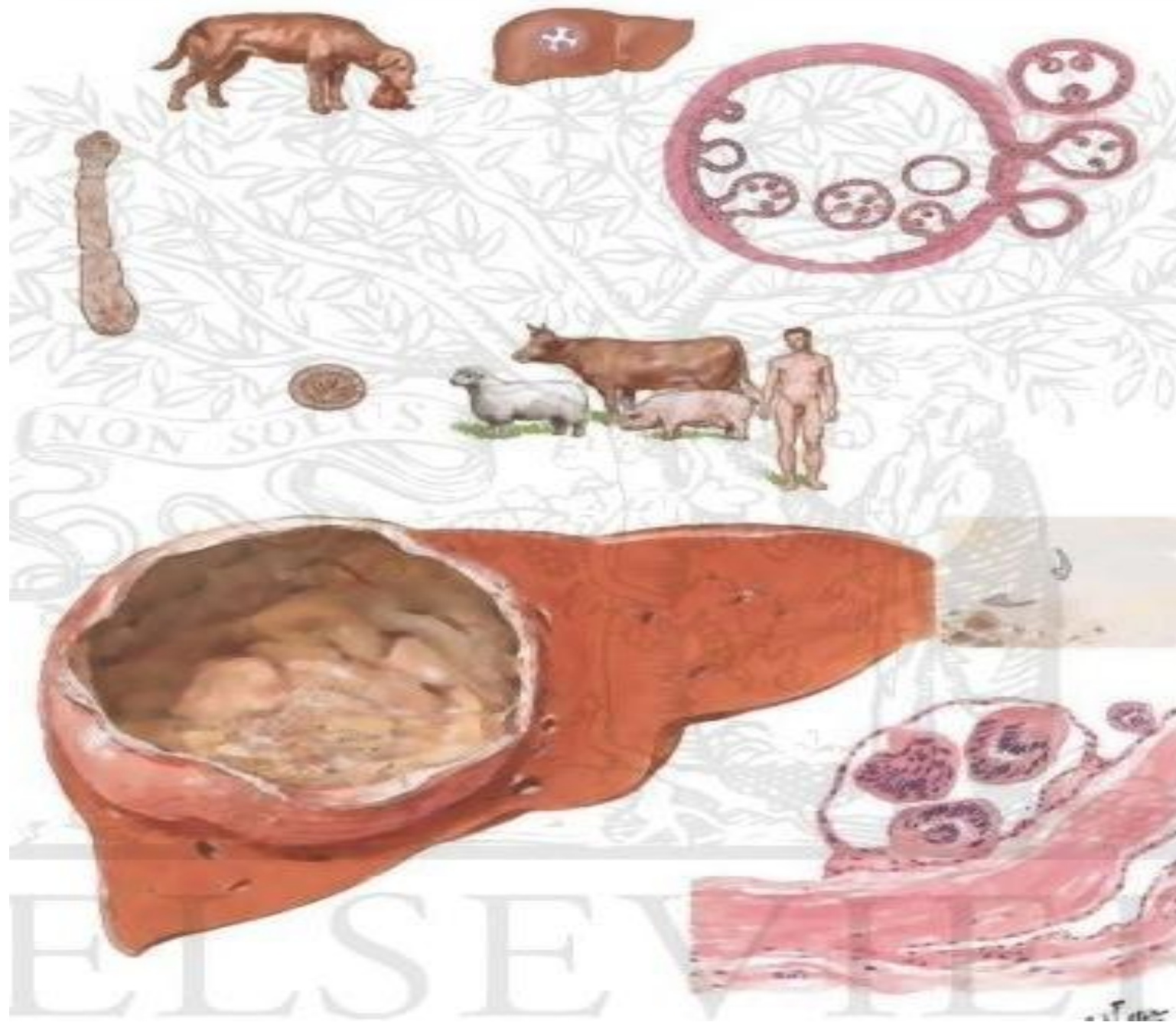
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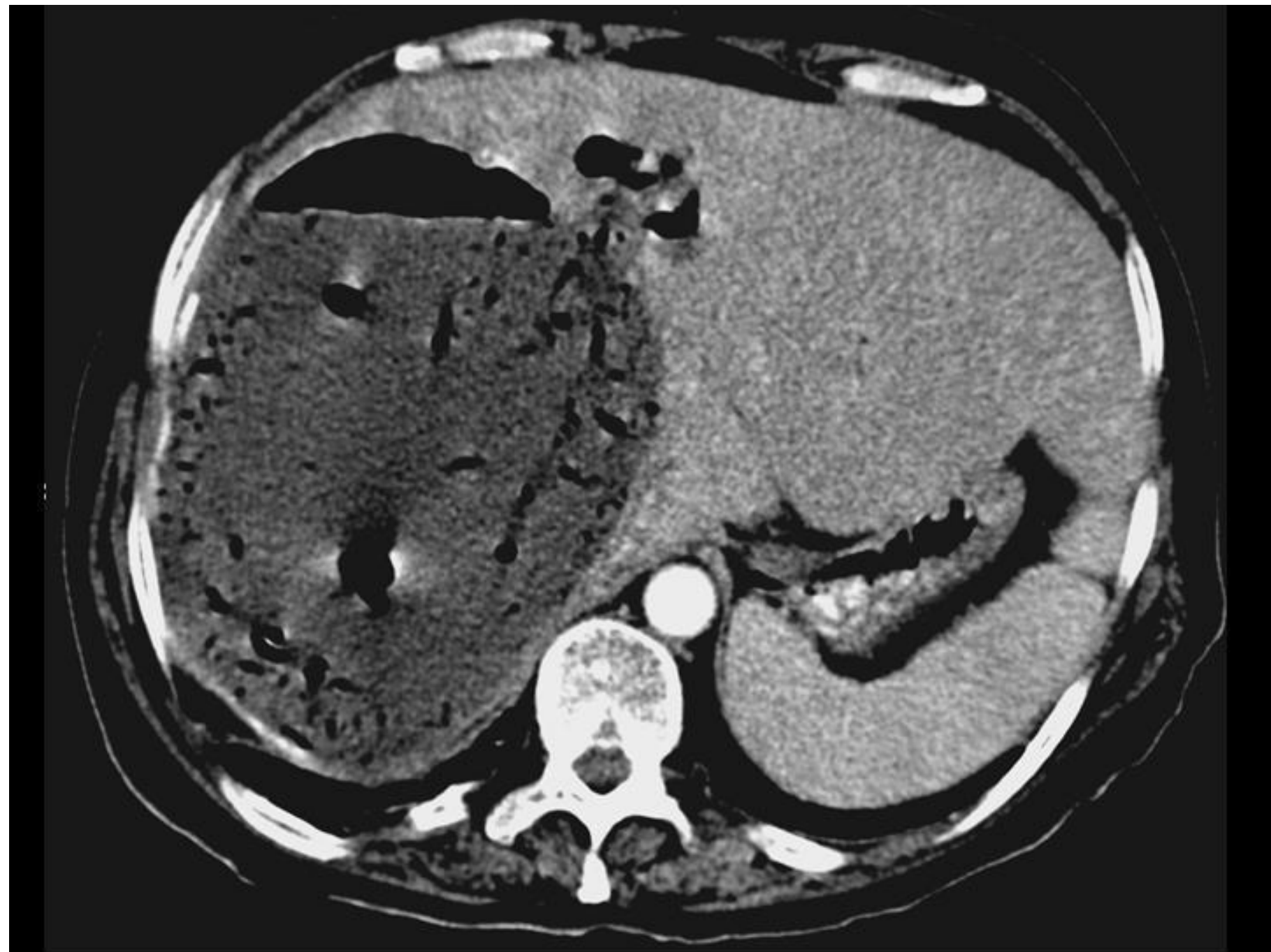












The surgical treatment technique for liver hydatid cyst (LHC) cannot be standardized, and the surgical technique should be tailored according to the **extent of the cyst and any adjunct complications of hydatid disease.**

Albendazole (10 mg/kg/day) for 15 days preoperatively.

A 33% hypertonic saline solution is used to deactivate the cyst content. The solution is left inside the cyst about 10 min to kill the scolices. If the cyst was localized to the liver periphery, the entire cyst could be excised; however, if the cysts were centrally located, they were drained through a hepatectomy. After evacuating the cystic contents, the cavity is explored for any possible communication with the bile duct. The offending bile duct is then sutured. The remaining cystic cavity is then drained or obliterated. Obliteration was achieved with omentoplasty or capitonnage. A drain is then placed into the subhepatic or subdiaphragmatic space.

Surgical treatment of hydatid cysts of the liver depends on communication of the cyst and the bile duct. If the cyst is localized peripherally, total cystectomy or hepatic resection is recommended because of the low rate of recurrence.

However, partial cystectomy and omentoplasty are the most frequently used operations for intraparenchymal hydatid cysts. Omentoplasty has been advocated for its absorptive capacity of residual fluid in the cystic cavity.

# Mass lesions of the liver

Hemangiomas, benign tumors, hepatocellular carcinoma or metastatic tumors to the liver.

**Benign solid tumors:** the most common are hepatic cell adenoma and focal nodular hyperplasia. The first occur in young women in assoc with oral contraceptive use. Other :hamartomas, lipomas, fibromas, leiomyomas, myxomas, and teratomas. Unless these tumors reach a large size, they are usually asymptomatic.

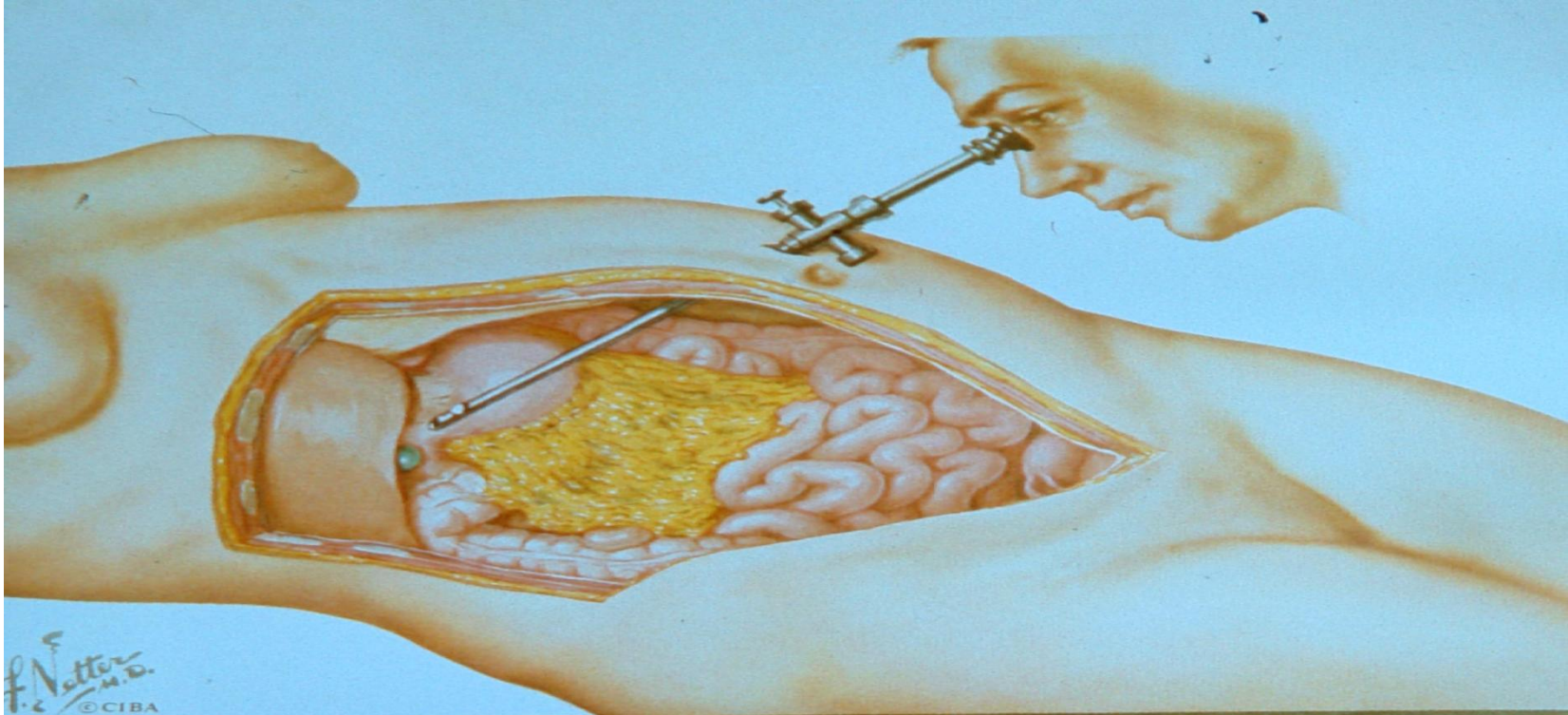
**Clinical findings:** because of complications (hemorrhage into the tumor- abdominal pain, or rupture and bleeding-shock hypovolemic)

**Diagnostic plan:** nuclear scans and ultrasound will demonstrate a mass,angiography and CT scans for ethiology. Fine needle aspiration biopsy may be done for tissue diagnosis.

**Management plan:**surgical treatment (resection) for adenomas is recommended because of high incidence of rupture or hemorrhage.



# L I V E R



NORMAL PERITONEUM,  
ROUND LIGAMENT



PERITONEAL  
CARCINOMATOSIS



PERITONEAL  
TUBERCULOSIS  
(SMALL INTESTINE)



NORMAL LIVER  
AND GALLBLADDER



METASTATIC  
CARCINOMA  
OF LIVER



CIRRHOSIS  
OF LIVER

# Hemangiomas:

h.cavernous is the most common benign tumor of the liver

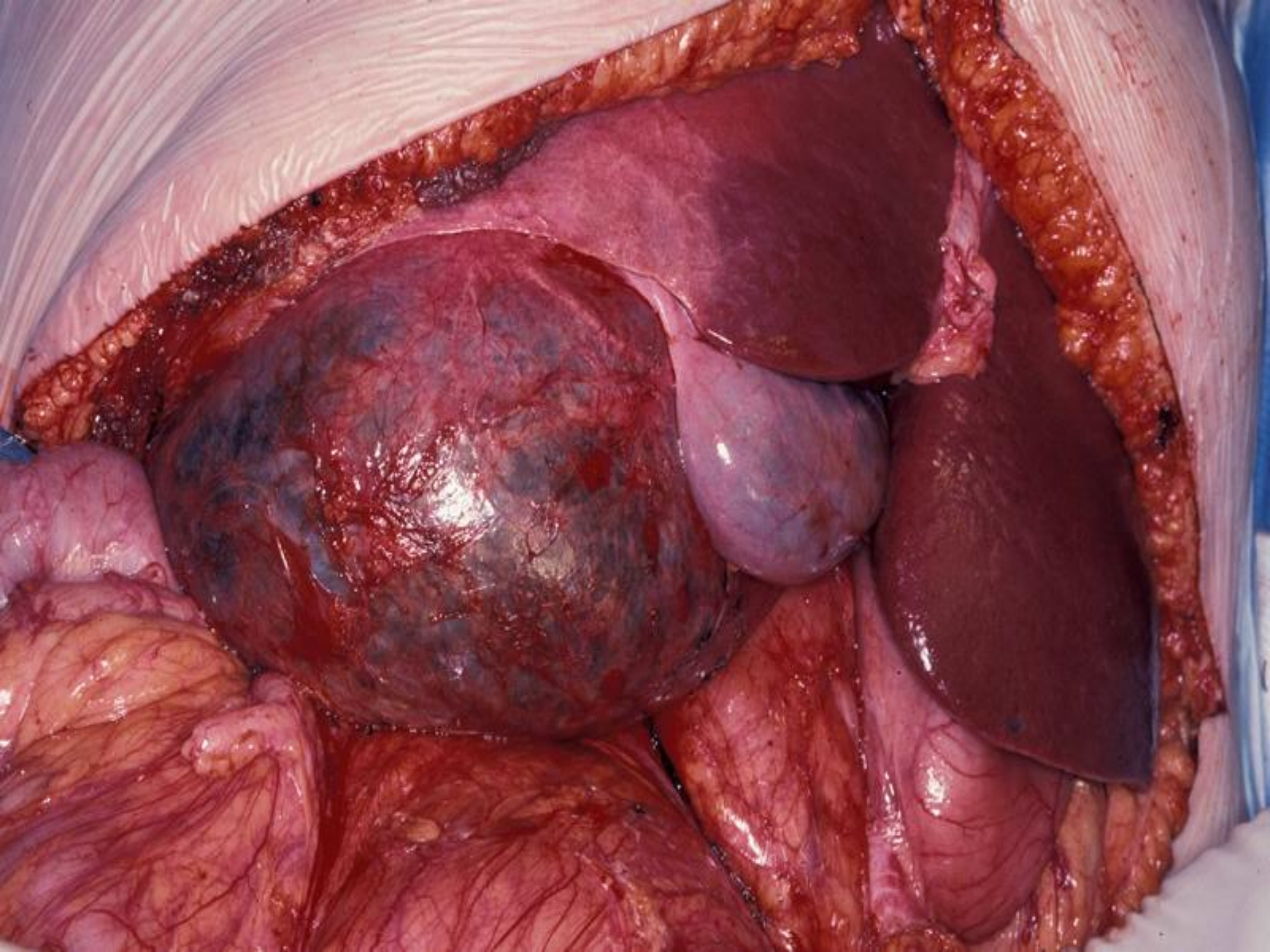
**Clinical findings:** large h. causes abdominal pain and palpable mass in the upper abdomen.

**Diagnostic plan:** nuclear flow scans and ultrasound tests for showing a mass effect in the liver; CT scans with contrast for dg.

Needle biopsy is contraindicated because of risk of uncontrollable bleeding.

**Management plan:** small asymptomatic lesions require no treatment except careful identification to avoid missing a neoplasm,  
if is clinically symptomatic require **surgical resection**.





# Hepatocellular carcinoma

## Causes and risk factors:

- **Hepatitis B infection**
- **Hepatitis C infection - cirrhosis**
- **Alcohol**
- **Aflatoxin B1**
- **Drugs, medications, and chemicals** (estrogens anabolic steroids) are associated with the development of hepatic adenomas
- **Hemochromatosis**
- **Diabetes and obesity**

# Hepatocellular carcinoma

**Clinical findings:** pain, abdominal mass, weight loss, lethargy, and shock (in rupture into the peritoneal cavity leading to hemoperitoneum) The presence of jaundice or ascites are late signs and carry a poor prognosis.

**Metastatic sites:** the main mode of dissemination of liver cc is via the portal vein and hepatic veins. Intrahepatic venous dissemination cannot be differentiated from satellitosis or multifocal tumors and is classified as multiple tumors.

Most common sites of extrahepatic dissemination are the lungs and bones

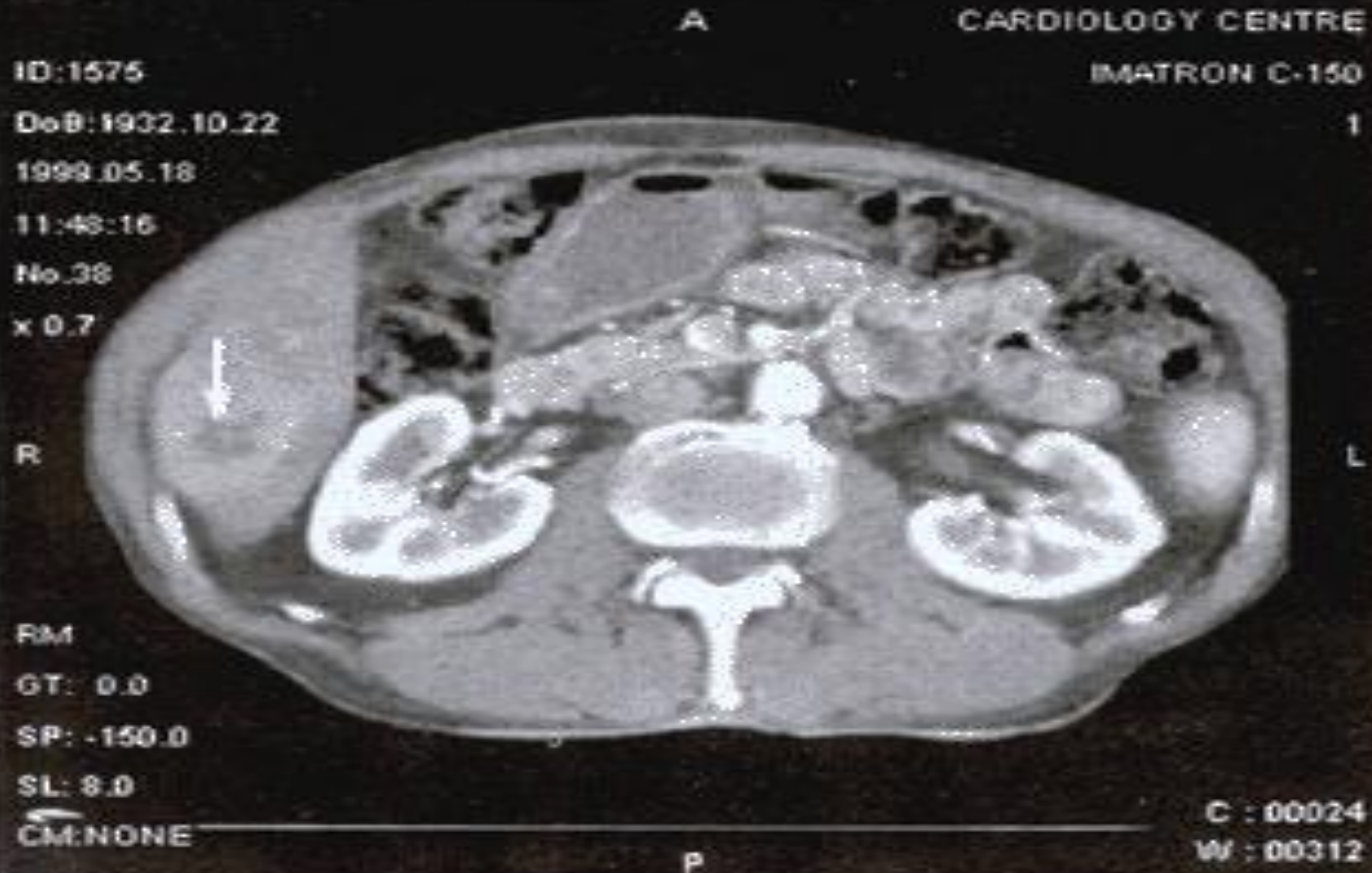
**Diagnostic plan:** serum alphafetoprotein, US, CT, MRI

**Management plan:**

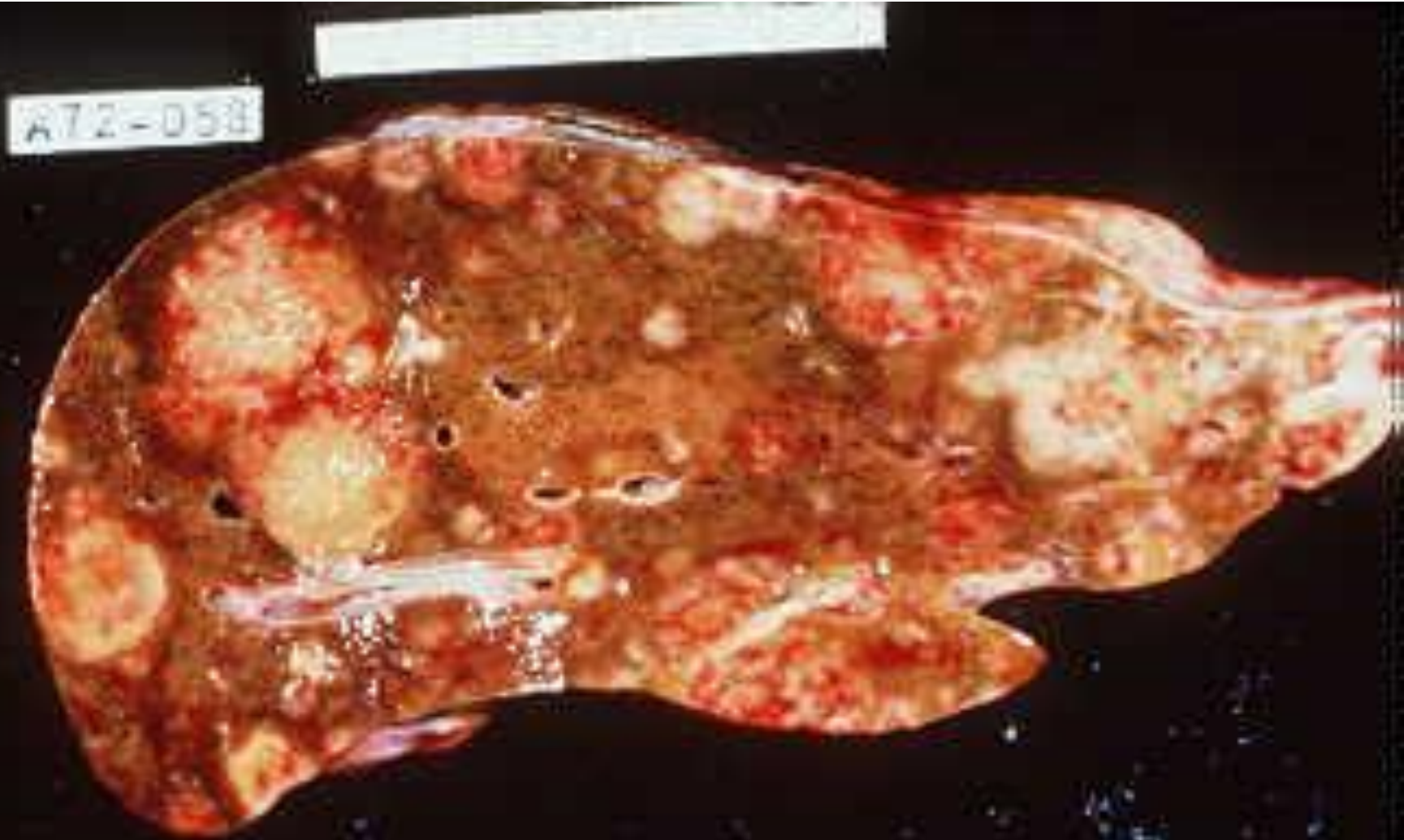
- tumors less than 3 cm should be resected;
- standard hepatic lobectomy or
- extended hepatic resections are performed.
- Chemotherapy



# liver carcinoma



# liver carcinoma





DIAPHRAGMATIC HIATUS FOR VENA CAVA

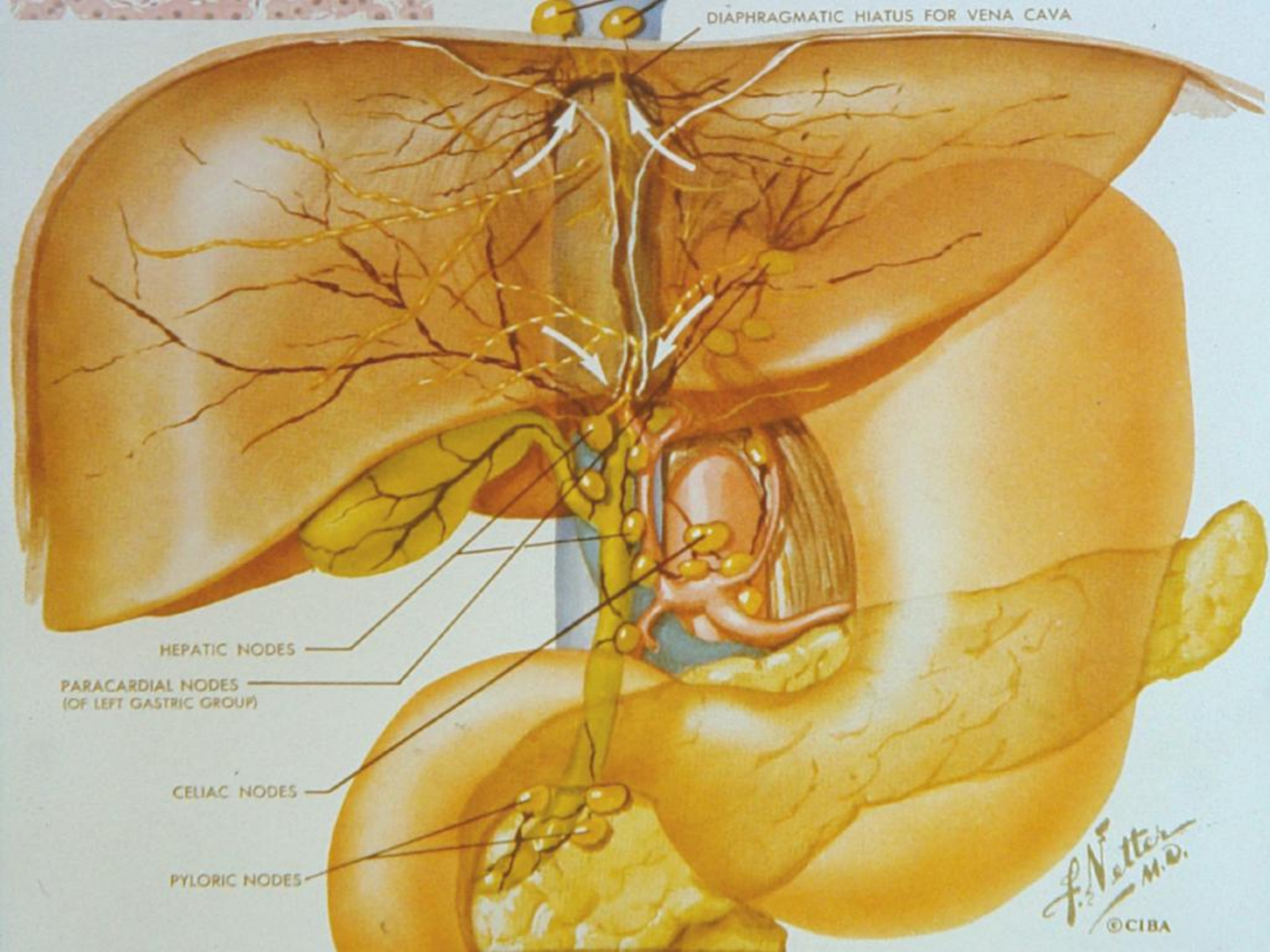
HEPATIC NODES

PARACARDIAL NODES  
(OF LEFT GASTRIC GROUP)

CELIAC NODES

PYLORIC NODES

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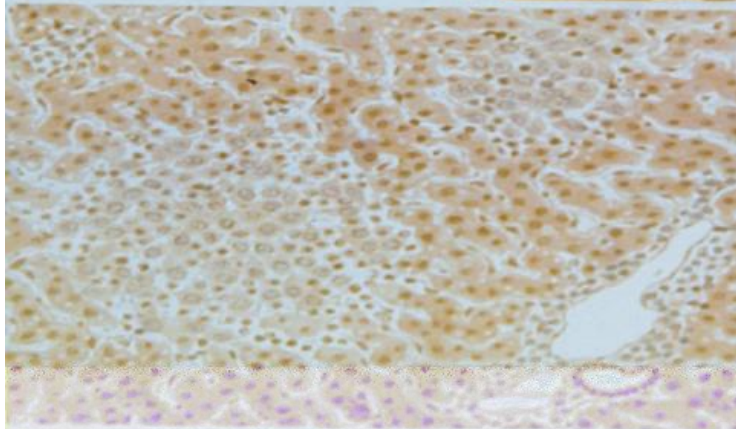


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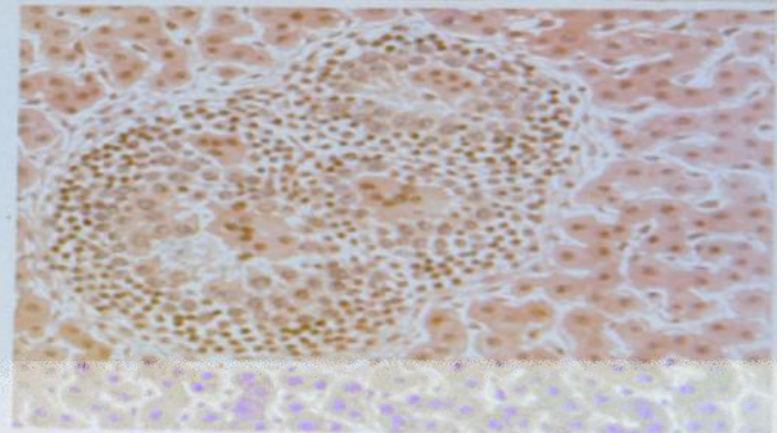


LARGE  
TUBERCULOMAS

MILIARY  
TUBERCULOSIS



ACUTE MILIARY GRANULOMAS  
(SOFT TUBERCLE — CHIEFLY HISTIOCYTES)



SUBACUTE CONGLOMERATE TUBERCLE  
(GIANT CELLS, CASEATION, HISTIOCYTES, SURROUNDED  
BY LYMPHOCYTES AND FIBROSIS)

**The treatment options** are dictated by **the stage** of liver cancer and the **overall condition of the patient**.

- The only proven cure for liver cancer is **liver transplantation for a solitary, small (<3cm) tumor**.
- **small tumor can be surgically removed** (partial hepatic resection).

(However, most patients with liver cancer also have cirrhosis of the liver and would not tolerate liver resection surgery).

- many patients who undergo hepatic resections will develop a recurrence of liver cancer in the liver within several years.
- **radiofrequency ablation**, of the tumor under ultrasound or CT scan guidance.
- **Thermal (heat) energy** is created by radio waves
- **Chemotherapy** drugs can be given directly into the blood vessel that feeds the liver and the tumors; a similar technique, using microscopic radioactive particles instead of chemotherapy injected into the blood vessels, is called radioembolization or selective internal radiation therapy (SIRT). This uses radioactive yttrium attached to glass microspheres and may be as effective as chemoembolization for small and multiple tumors.
- **Radiation therapy** uses high-dose energy like X-rays aimed at a small part of the body and can frequently destroy cancer cells. The liver, though, may be more sensitive to the radiation than the tumor is, so standard radiation is seldom used.

## Metastatic tumors

to the liver reflect advanced malignancy, the most common comes from the **colon**.

Metastatic disease from most noncolonic sites is not managed by resection

# PORTAL HYPERTENSION

**Cirrhosis** is the most common cause of this disorder.

Two important factors:

- **vascular resistance and**
- **blood flow** exist in the development of portal hypertension.

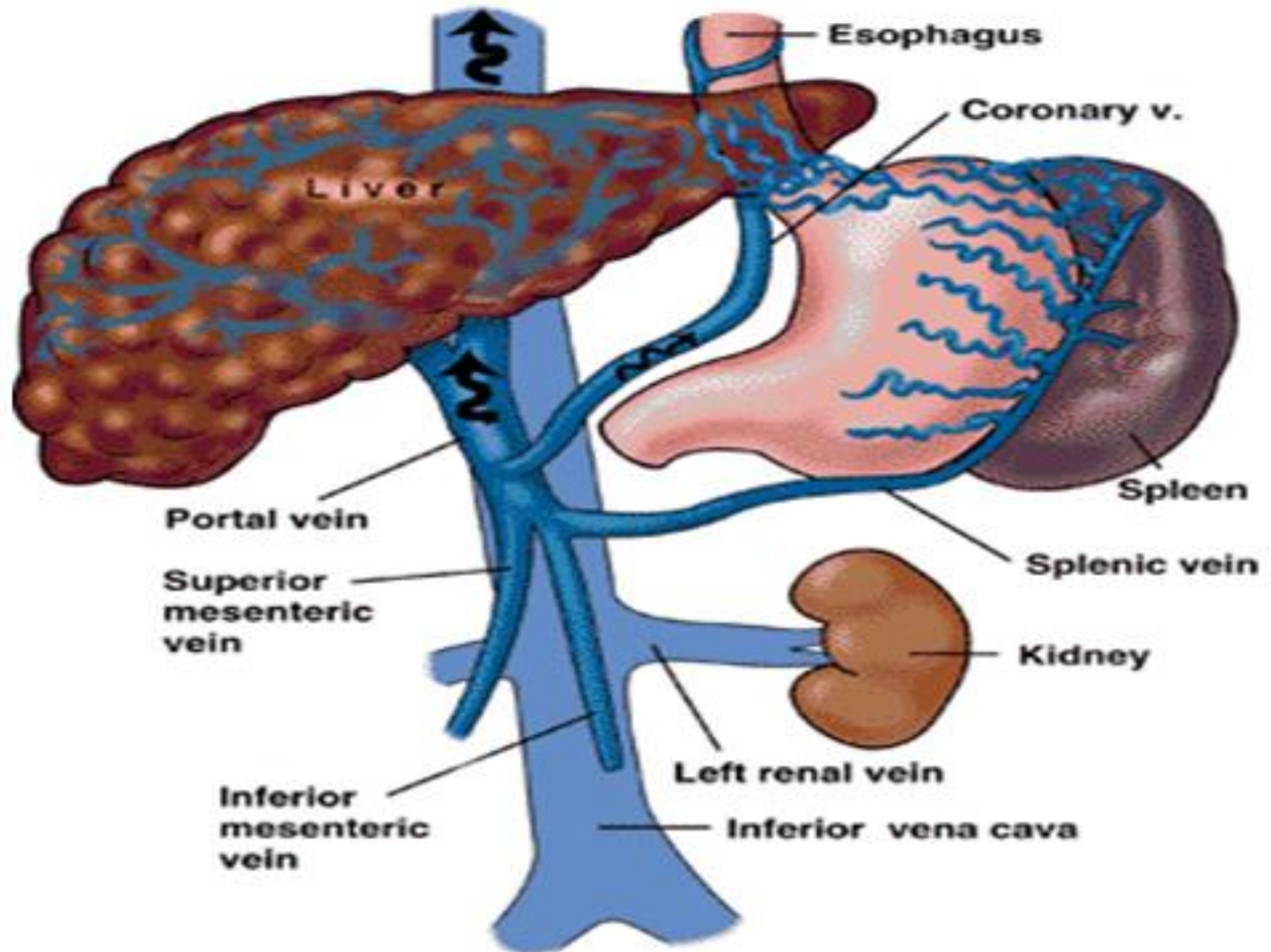
Normal portal pressure is generally considered to be between

**5 and 10 mm Hg.**

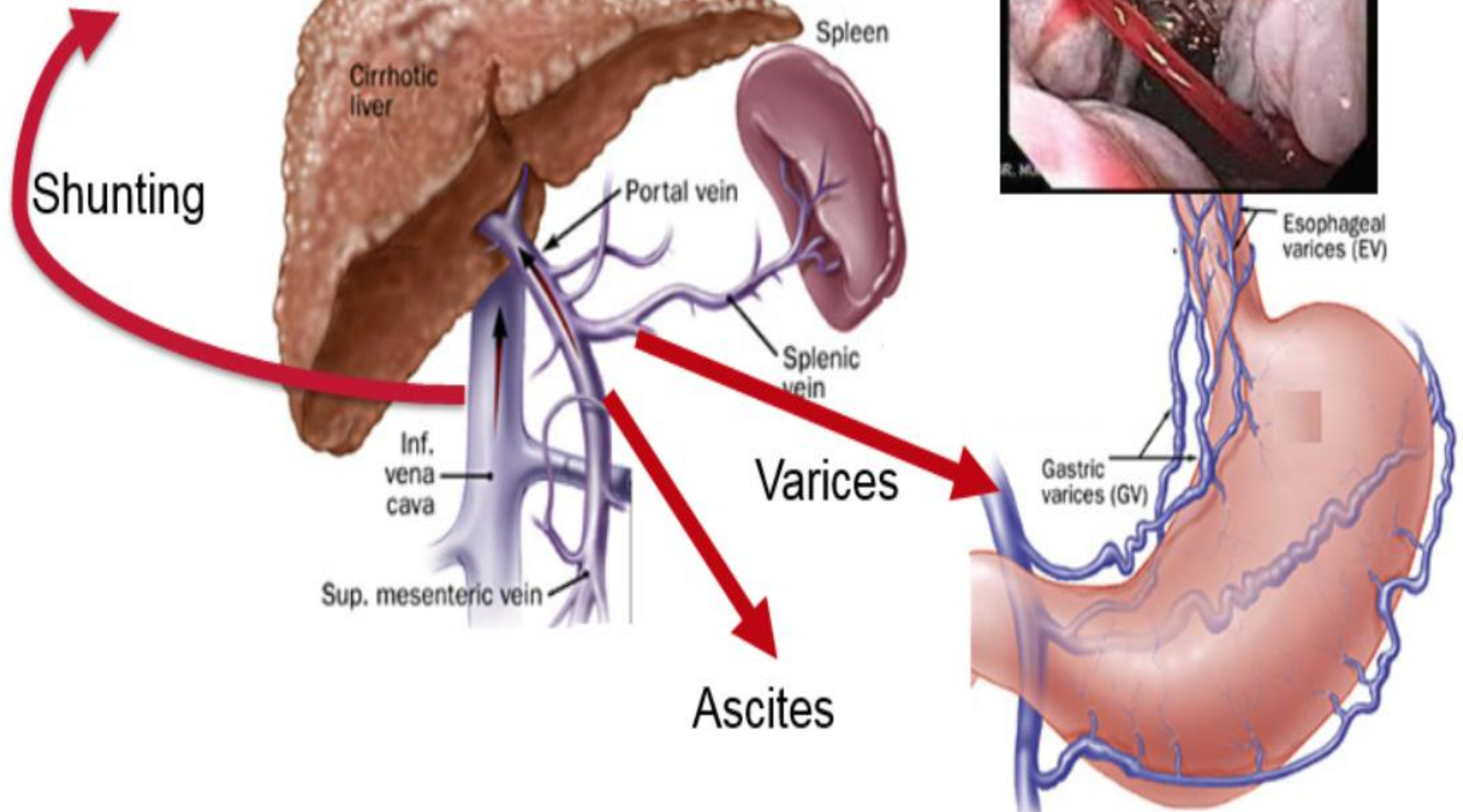
Once the portal pressure rises to 12 mm Hg or greater, complications can arise: varice, haemorrhoids and ascites. Indeed, **esophageal varices** are responsible for the main complication of portal hypertension, massive upper gastrointestinal (GI) hemorrhage.



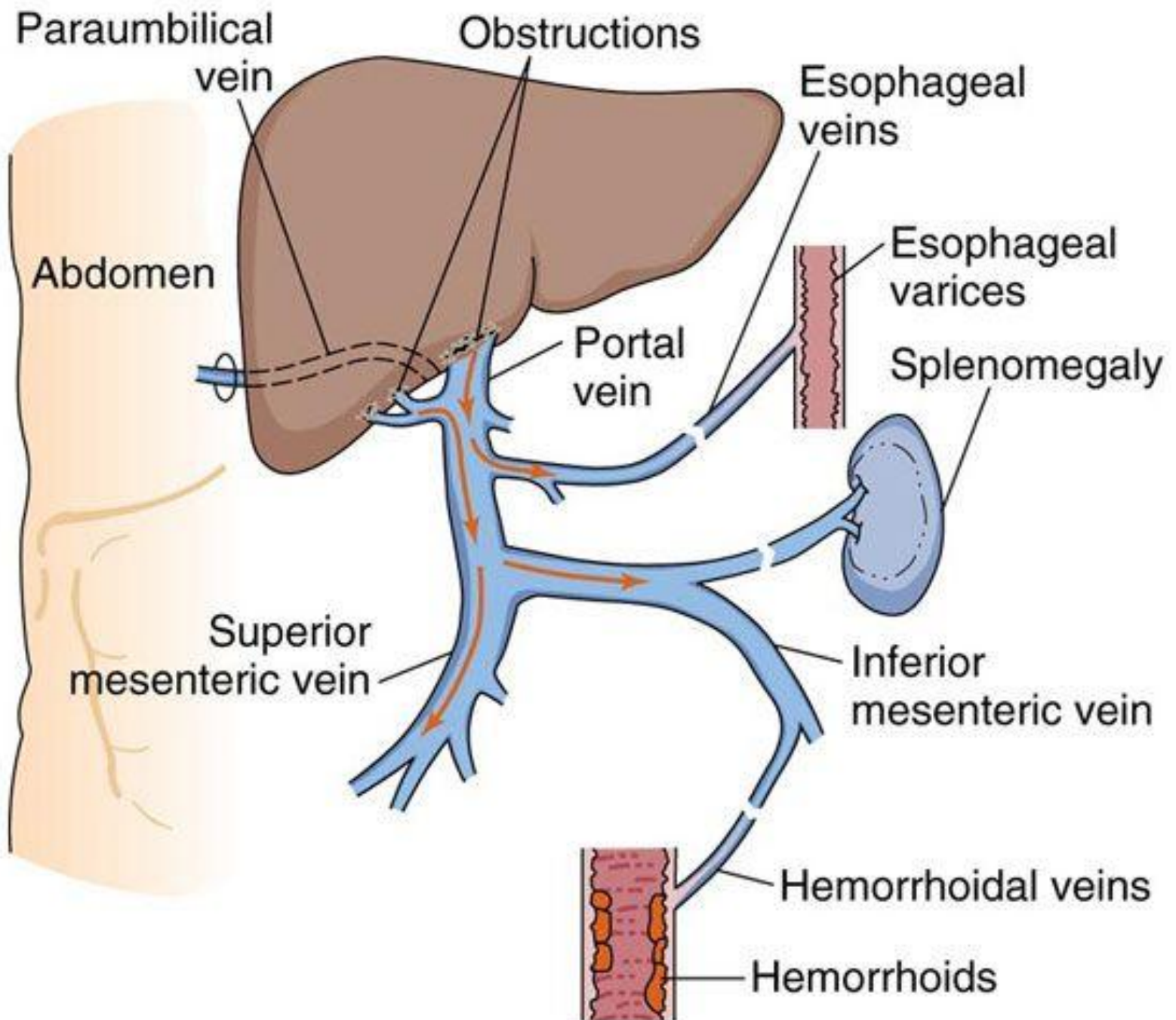
# Portal Hypertension

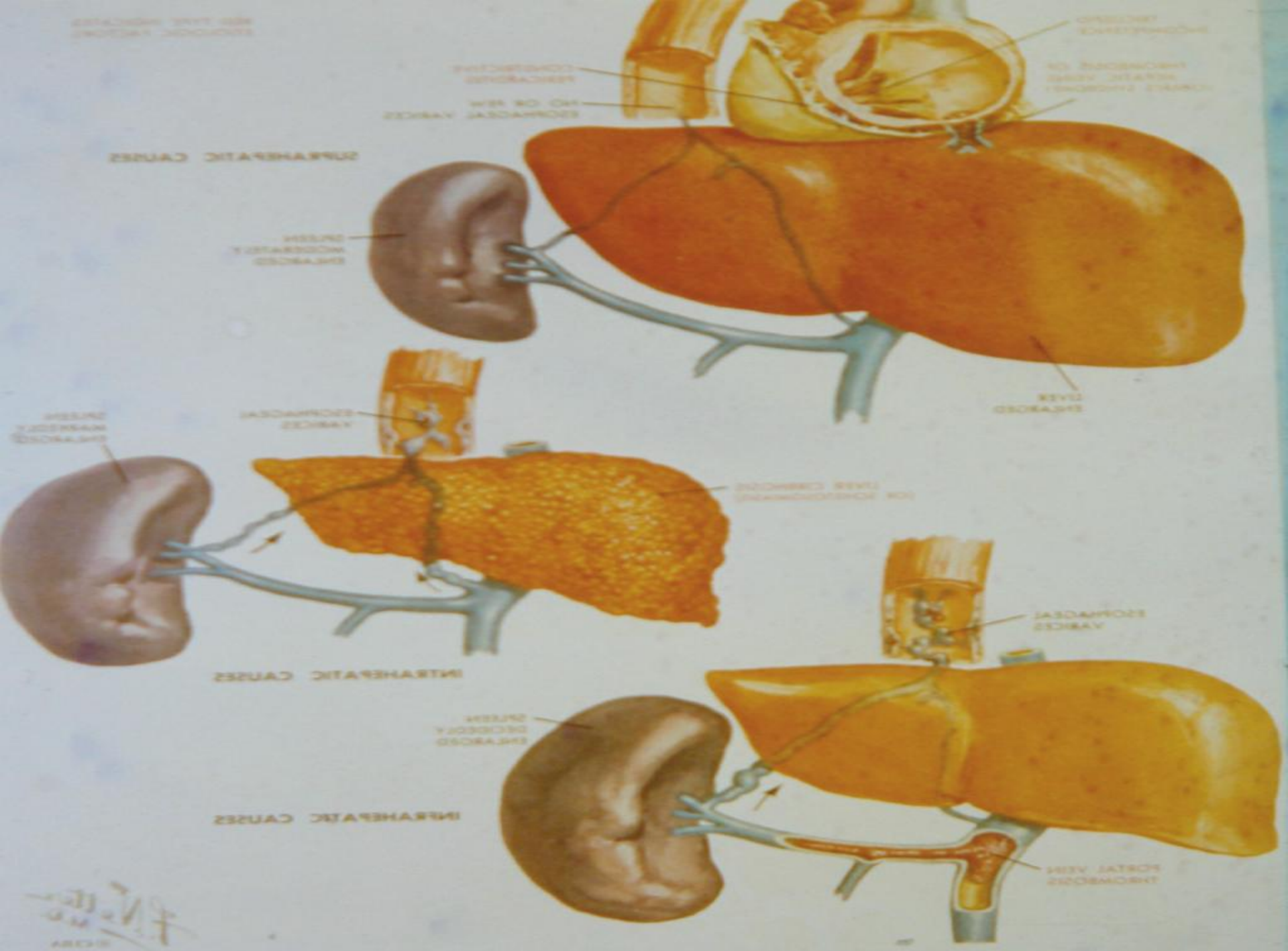


Encephalopathy  
↓ Liver Cell Function









## PORTAL HYPERTENSION

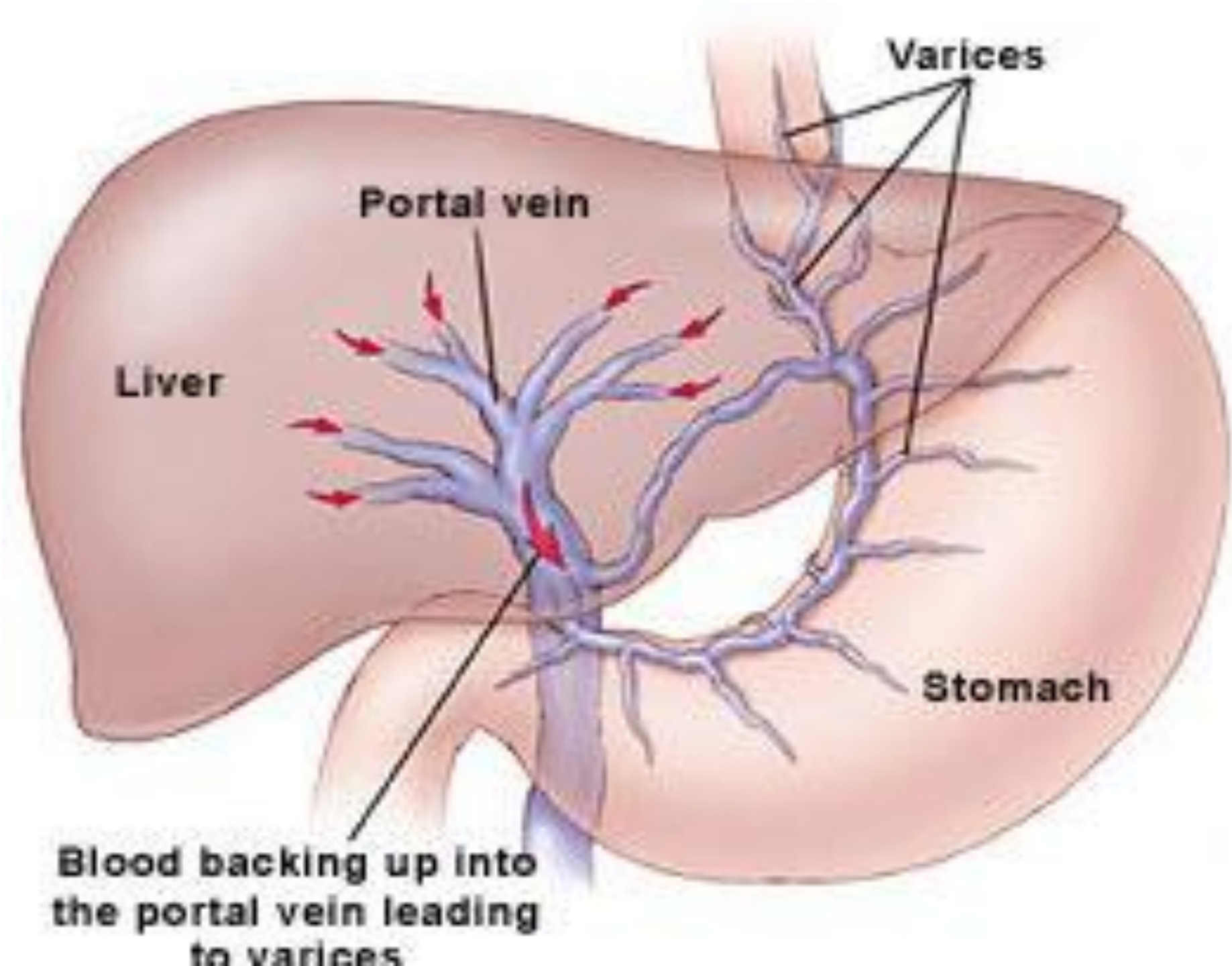
Obstruction of portal venous flow, whatever the etiology, results in a rise in portal venous pressure

The response to increased venous pressure is the development of **collateral circulation** that diverts the obstructed blood flow to the systemic veins.

These portosystemic collaterals form by the opening and dilatation of preexisting vascular channels connecting the portal venous system and the superior and inferior vena cava.

The most important **portosystemic anastomoses are the gastroesophageal collaterals**, which include esophageal varices. The gastroesophageal collaterals drain into the azygos vein.





## PORTAL HYPERTENSION

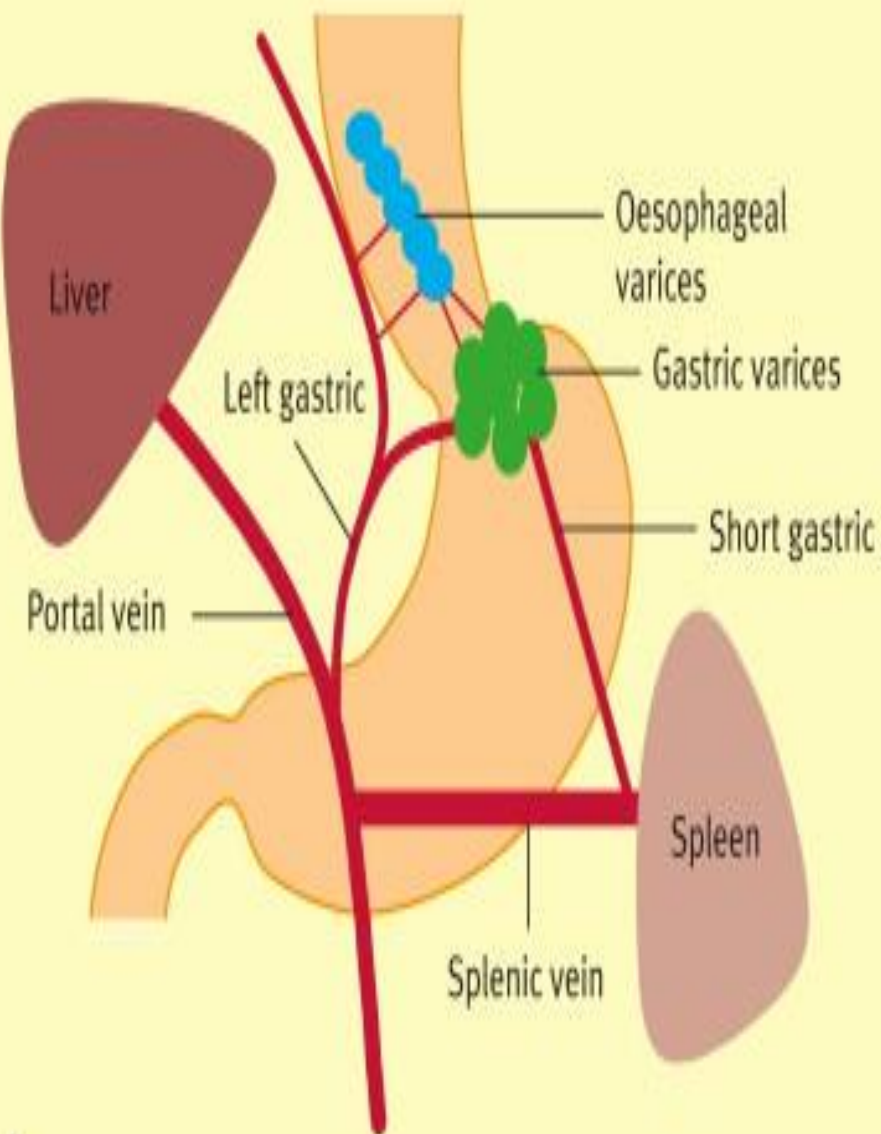
Patients with a known diagnosis of esophageal varices have a 30% chance of variceal bleeding within the first year after the diagnosis

The mortality rate of the bleeding episode depends on the severity of the underlying liver disease.

Patients who have had 1 episode of bleeding from esophageal varices have a 60-80% chance of rebleeding within 1 year after the initial episode; approximately one third of further bleeding episodes are fatal.

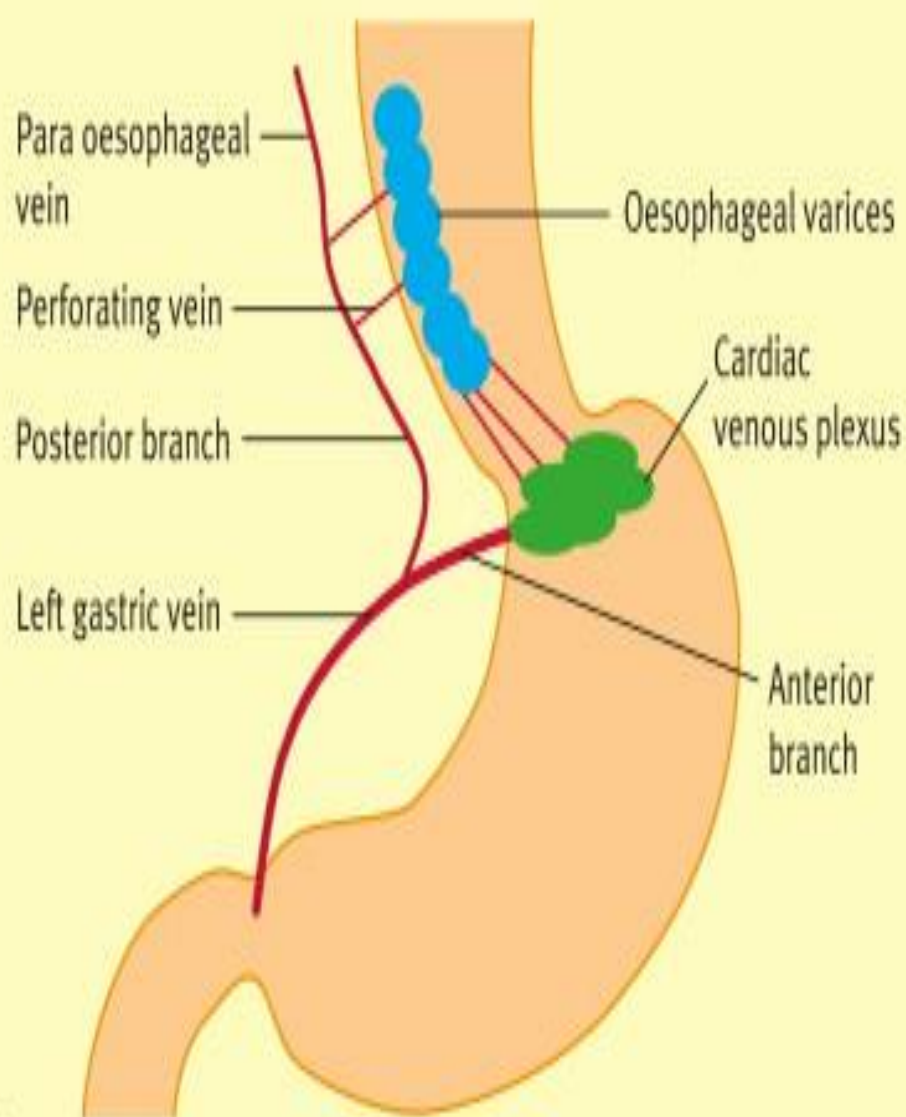
The risk of death is maximal during the first few days after the bleeding episode, this rate is higher when surgical intervention is needed<sup>1</sup>

## Portal-systemic collaterals in portal hypertension



a

## Portal-systemic collaterals and oesophageal varices



b

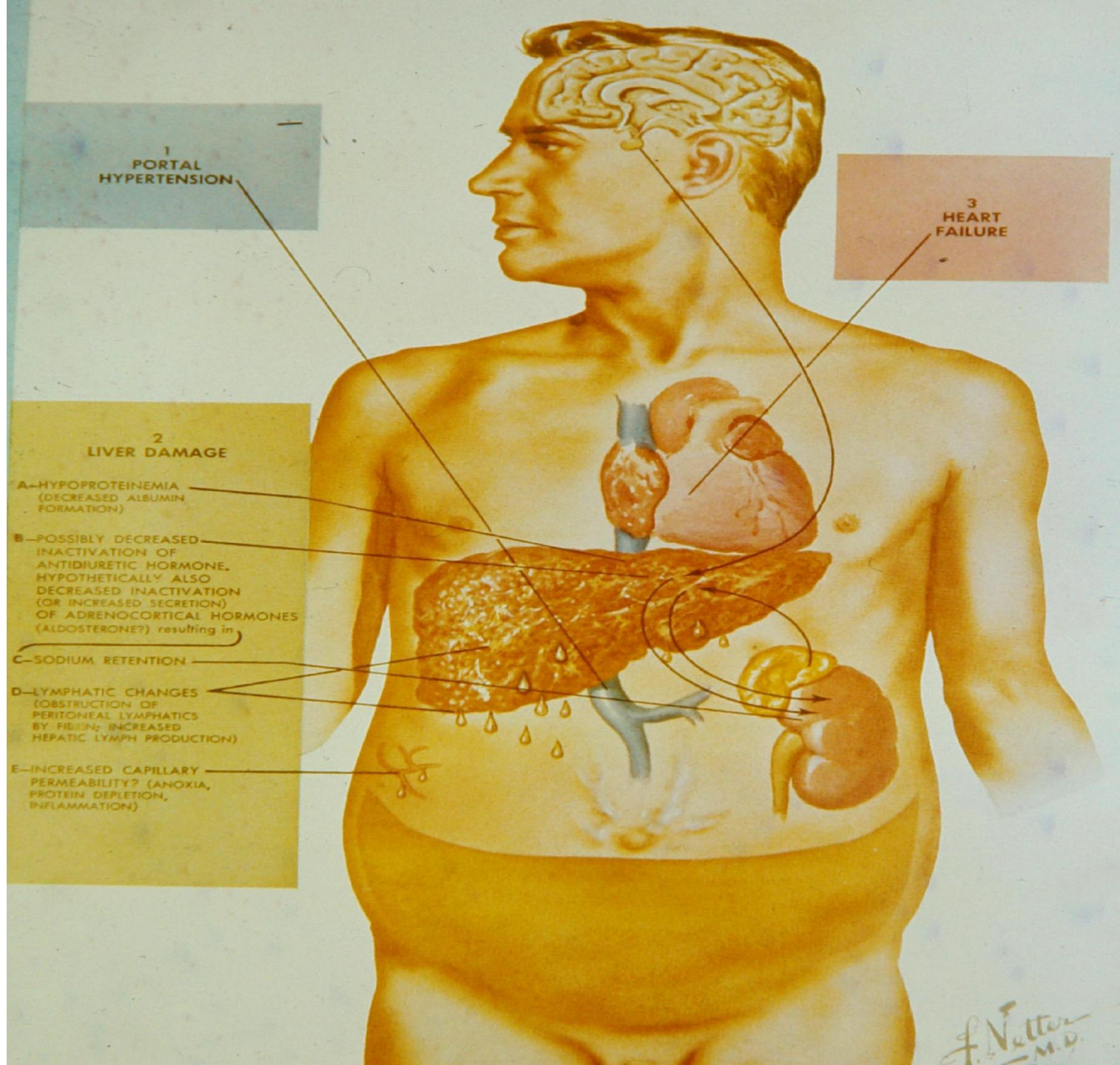
# PORTAL HYPERTENSION

Complications associated with portal hypertension and GI bleeding include the following:

- Hepatic encephalopathy
- Bronchial aspiration, aspiration pneumonia
- Renal failure
- Systemic infections, sepsis
- Spontaneous bacterial peritonitis
- Ascites
- Hepatorenal syndrome
- Bacteremia and/or endotoxemia
- Vascular collapse
- Cardiomyopathy
- Arrhythmias
- Hypotension
- Portal hypertensive gastropathy - This is a common complication of cirrhosis and portal hypertension, but not significant bleeding from this source.



# LIVER



**Surgery** has no role in primary prophylaxis. Its role in **acute variceal bleeding is exceedingly limited**, because therapy with endoscopic treatment controls bleeding in 90% of patients.

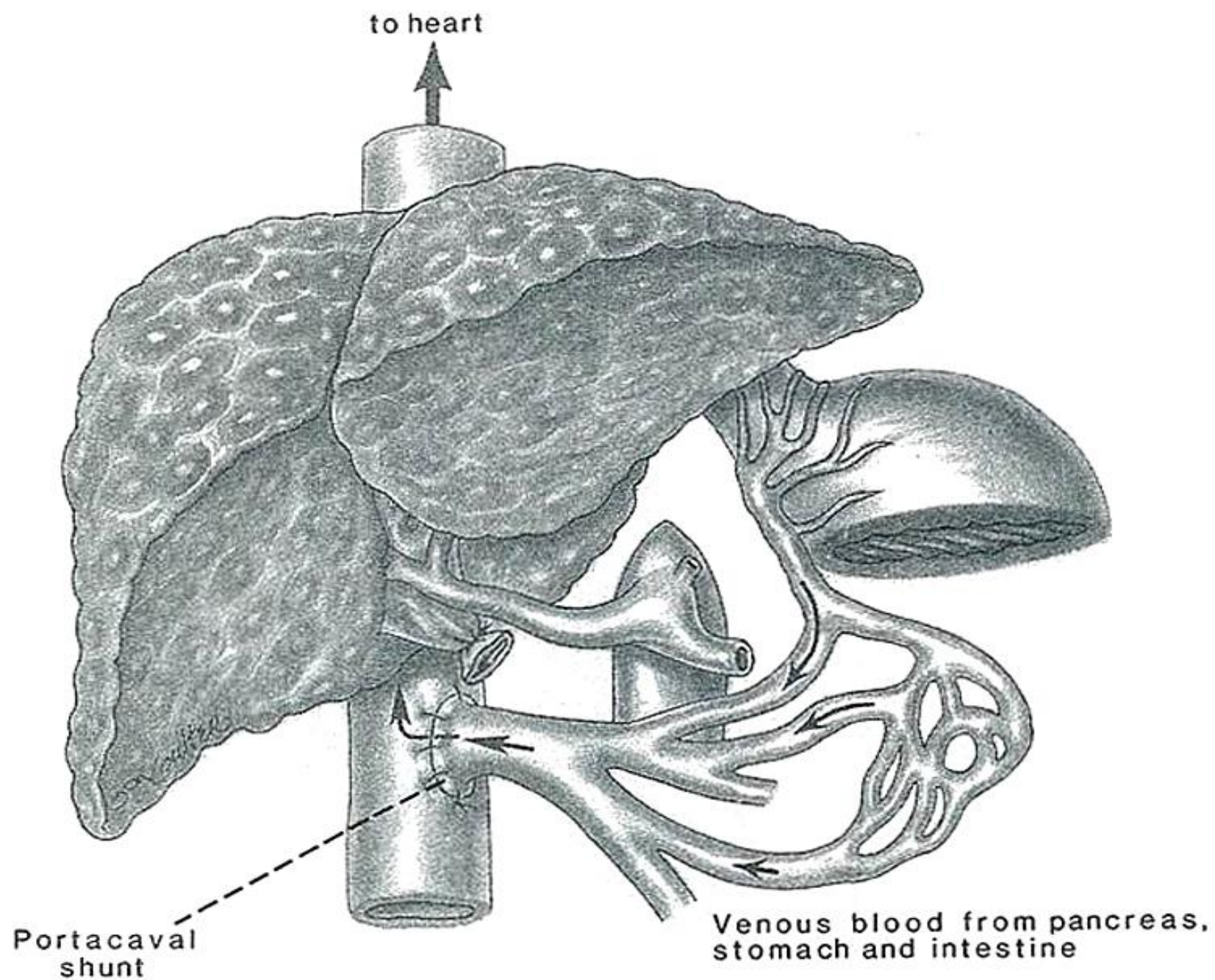
A **transjugular intrahepatic portosystemic shunt (TIPS)** is a viable option and is less invasive for patients whose bleeding is not controlled.

Consider **surgery for the prevention of rebleeding** when pharmacologic and/or endoscopic therapy have failed.

Surgical interventions include the following:

- Portosystemic shunt
- Devascularization procedures
- Orthotopic liver transplantation (OLT) - Treatment of choice in patients with advanced liver disease





**Surgical shunts** provide better control of rebleeding when compared to the combination therapy of beta-blocker and endoscopic variceal ligation (EVL). However, these shunts are associated with **higher incidence of hepatic encephalopathy** and should be reserved for Child class A patients with recurrent bleeding despite adequate combination therapy.

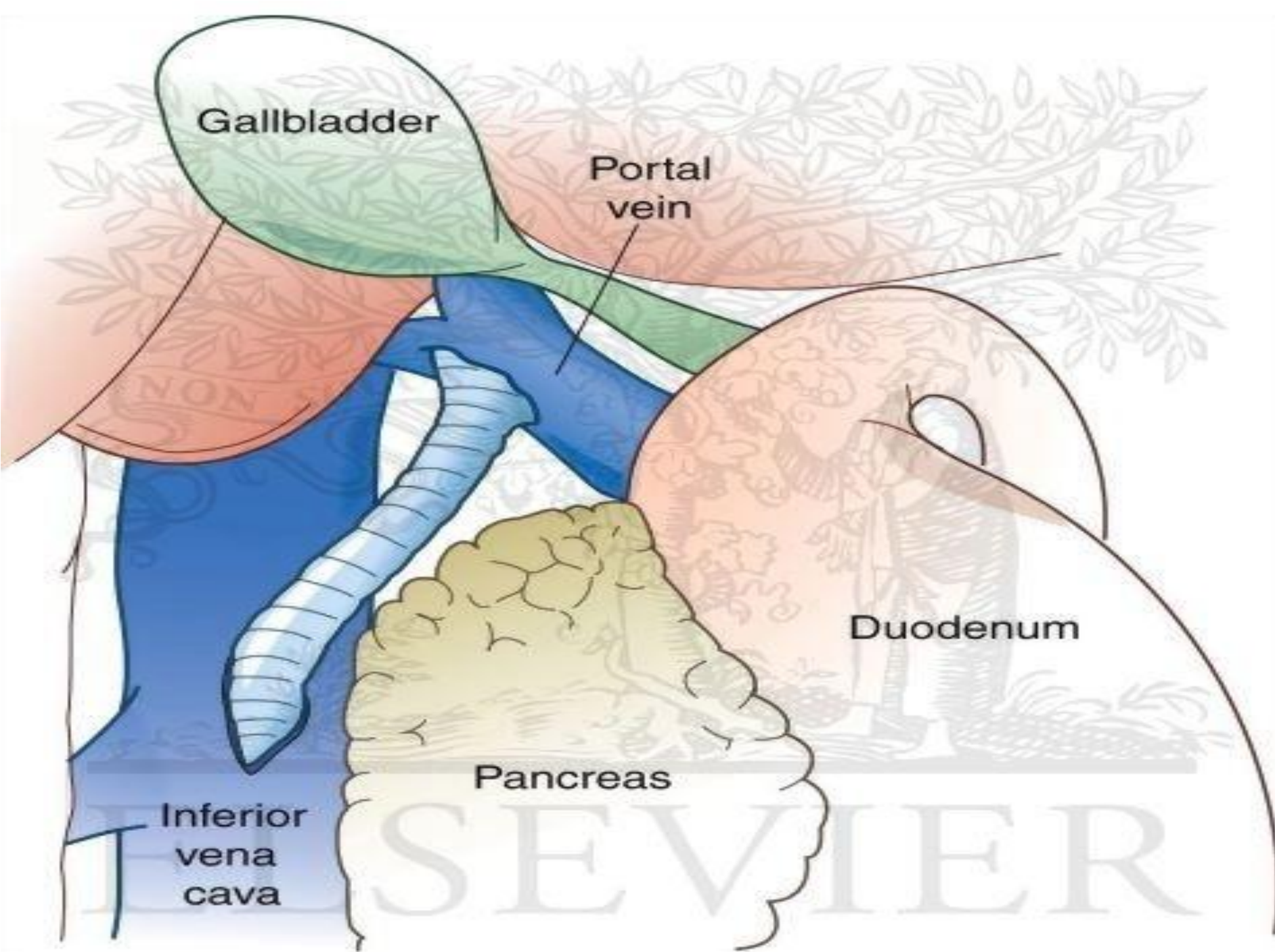
**Decompressive shunts** include:

- total portal systemic shunts,
- partial portal systemic shunts, and other
- selective shunts.

### **Partial portal systemic shunts**

Partial portal systemic shunts reduce the size of the anastomosis of a side-to-side shunt to 8 mm in diameter. Portal pressure is reduced to 12 mm Hg, and portal flow is maintained in 80% of patients.

The operative approach is similar to that for side-to-side portacaval shunts, except the interposition graft must be placed between the portal vein and the IVC



## Total portal systemic shunts

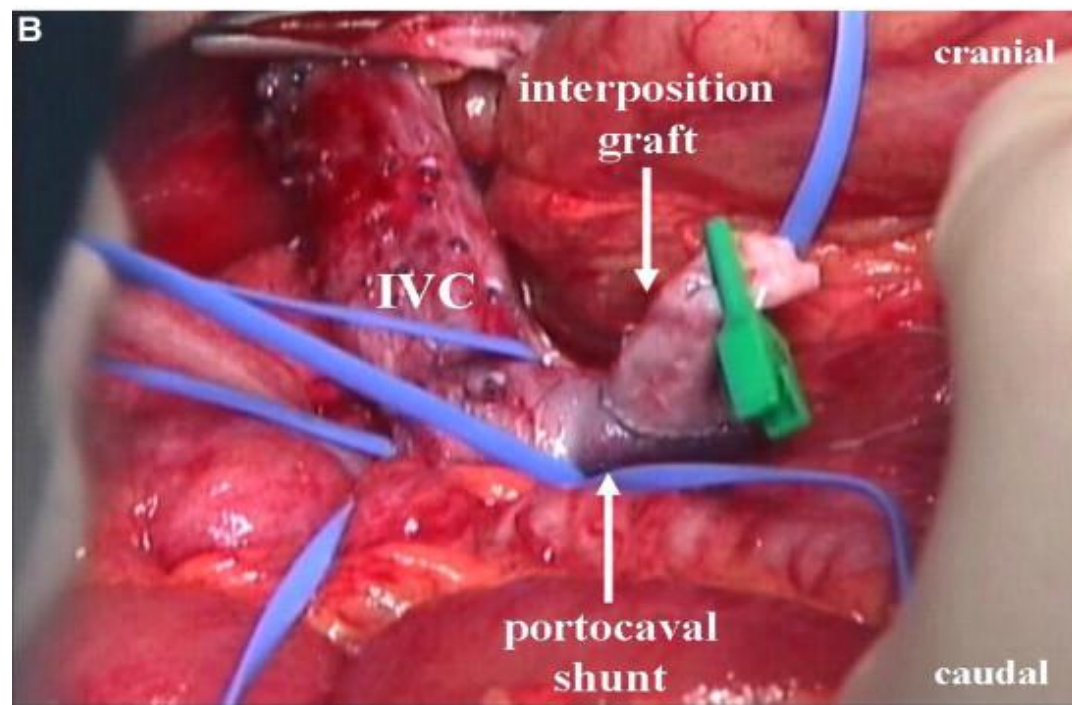
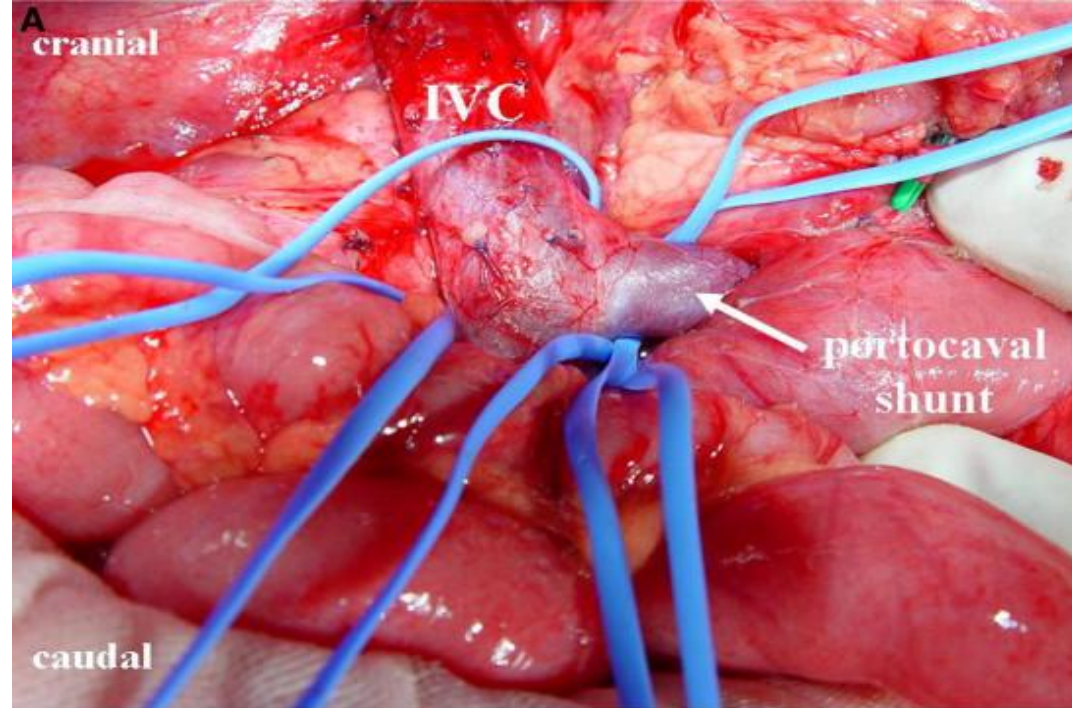
Total portal systemic shunts include any shunt larger than 10 mm in diameter between the **portal vein** (or one of its main tributaries) and the **inferior vena cava** (IVC) (or one of its tributaries).

For the side-to-side portacaval shunt, the portal vein and the infrahepatic IVC are mobilized after dissection and anastomosed. All portal flow is directed through the shunt, with the portal vein itself acting as an outflow from the obstructed hepatic sinusoids.

Excellent control of bleeding and ascites is achieved in more than 90% of patients. Encephalopathy (rate of 40-50%) and progressive liver failure are possible.

The procedure has relatively limited indications, which include massive variceal bleeding with ascites or acute Budd-Chiari syndrome without evidence of liver failure.

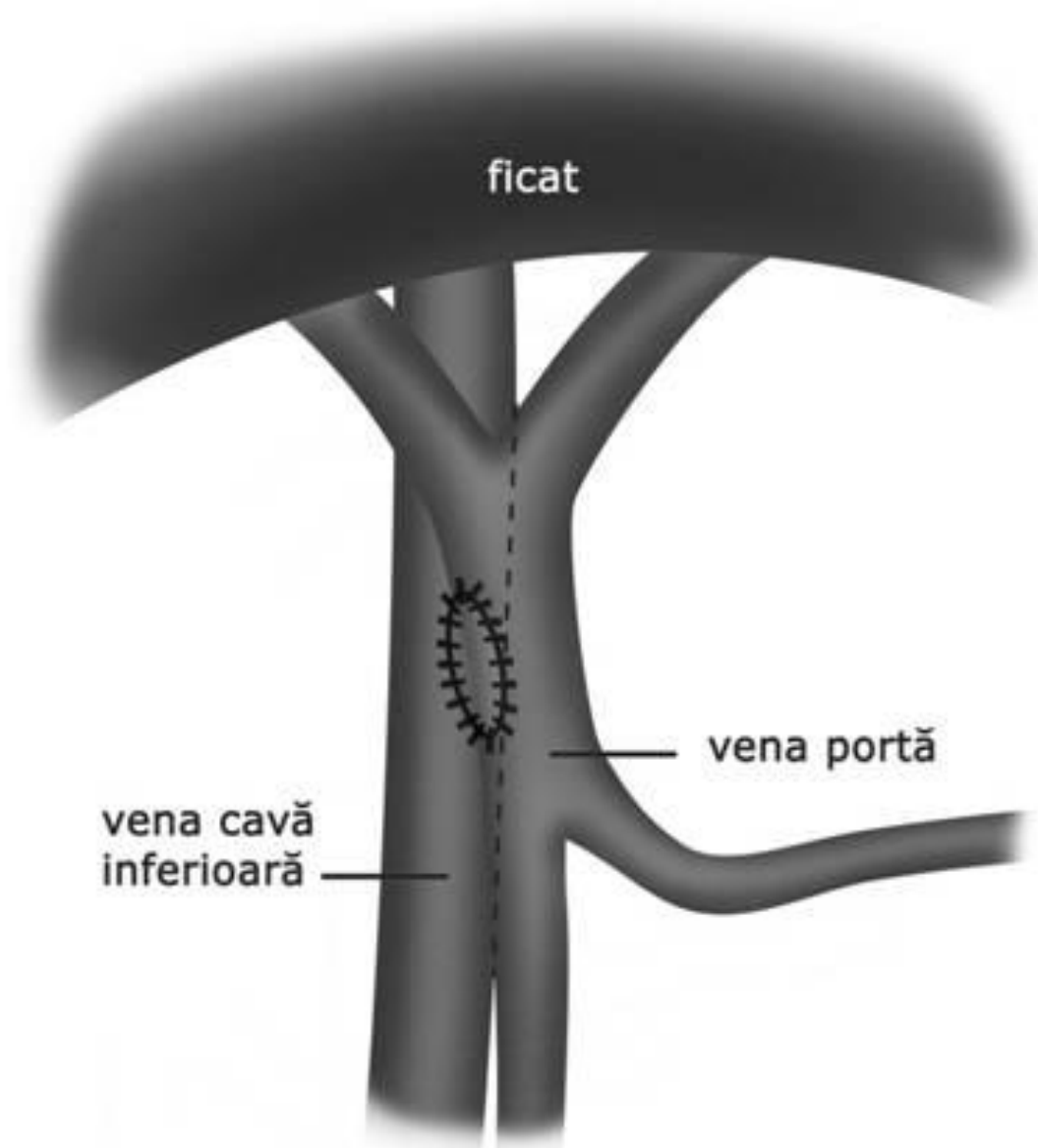




# Selective shunts

Selective shunts provide selective decompression of gastroesophageal varices to control bleeding while at the same time maintaining portal hypertension to maintain portal flow to the liver.

**distal splenorenal shunt**, which is the most commonly used decompressive operation for refractory variceal bleeding; it is used primarily in patients who present with refractory bleeding and continue to have good liver function. The distal splenorenal shunt decompresses the gastroesophageal varices through the short gastric veins, the spleen, and the splenic vein to the left renal vein. Portal hypertension is maintained in the splanchnic and portal venous system, and the shunt maintains portal flow to the liver. This type of shunt provides the best long-term maintenance of some portal flow and liver function, with a lower incidence of encephalopathy (10-15%) compared with total shunts. The operation produces ascites because the retroperitoneal lymphatics are diverted.



anastomoza porto-cavă latero-laterală

Devascularization is rarely performed but may have a role in patients with portal and splenic vein thrombosis who are not suitable candidates for shunt procedures and who continue to have variceal bleeding despite endoscopic and pharmacologic treatment.

Devascularization procedures consist of the transabdominal devascularization of the lower 5 cm of the esophagus and the upper two thirds of the stomach

The incidence of liver failure and encephalopathy is low following devascularization procedures, presumably because of better maintenance of portal flow.

## **Splenectomy**

The spleen is one of the major inflow paths to gastroesophageal varices. Splenectomy allows better access to the gastric fundus and the distal esophagus to complete the devascularization.

Portal vein thrombosis of as much as 20% is reported following splenectomy. Ascites is a frequent early postoperative complication because portal hypertension is maintained.

## **Gastroesophageal devascularization (Sugiura procedure)**

Gastroesophageal devascularization should devascularize the whole greater curve of the stomach from the pylorus to the esophagus and the upper two thirds of the lesser curve of the stomach. The esophagus should be devascularized for a minimum of 7 cm.

In patients who have undergone extensive and repeated sclerotherapy, the gastroesophageal junction is thickened and the ability to perform a satisfactory transection is limited.



**Liver transplantation** is the ultimate shunt, because:

- it relieves portal hypertension,
- prevents variceal rebleeding, and
- manages ascites and encephalopathy by restoring liver function.

It is the treatment modality that has significantly improved the outcome of patients with Child-Pugh class C disease and variceal bleeding

In most patients, it is impractical to use liver transplantation to treat portal hypertension, because these individuals can be managed successfully with lesser methods.

Therefore, the use of transplantation must be based on appropriate patient selections, as follows:

- Child class A disease, shunt surgery is recommended
- Child class B disease, shunt surgery or a transjugular intrahepatic portosystemic shunt (TIPS) is appropriate
- Child C class disease, TIPS or OLT is recommended
- Secondary prophylaxis is used to prevent rebleeding.

Variceal hemorrhage has a 2-year recurrence rate of approximately 80%.

# GALLBLADDER

(cholecyst, gall bladder, biliary vesicle )

In adults, the gallbladder measures approximately 8 centimetres in length and 4 centimetres in diameter when fully distended.

It is divided into three sections:

- fundus
- body
- neck.

The neck tapers and connects to the biliary tree via

the **cystic duct**,

which then joins the common hepatic duct to become

the **common bile duct**.

At the **neck** of the gallbladder is a mucosal fold called **Hartmann's pouch**, where gallstones commonly get stuck.

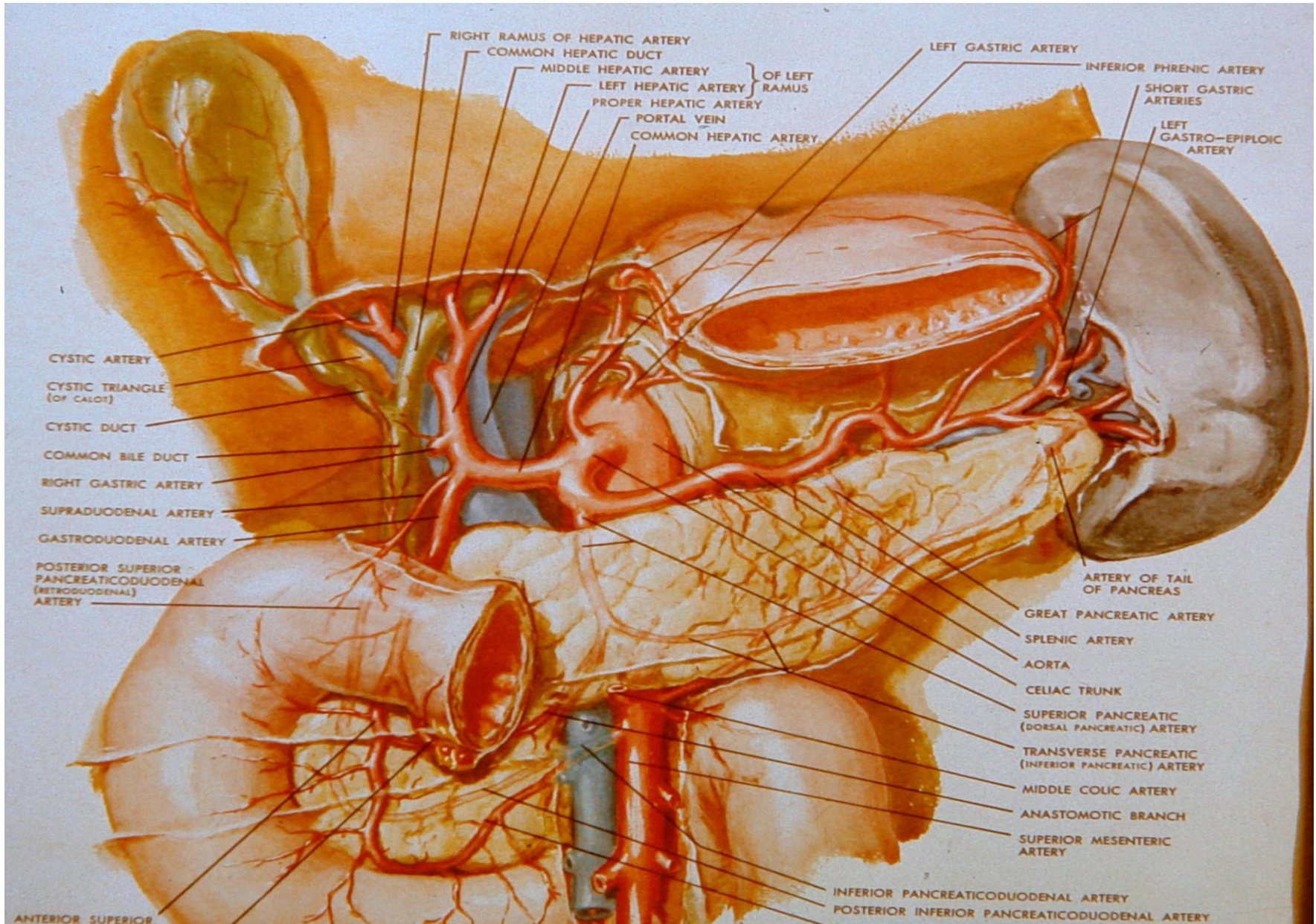
Abdominal projection of the gallbladder is located between the costal margin and the lateral margin of the rectus abdominis muscle.

When food containing fat enters the digestive tract, it stimulates the secretion of **cholecystikin** (CCK). In response to CCK, the adult human gallbladder, which stores about 50 millilitres of bile, **releases its contents into the duodenum**.

The bile, originally produced in the liver, emulsifies fats in partly digested food.

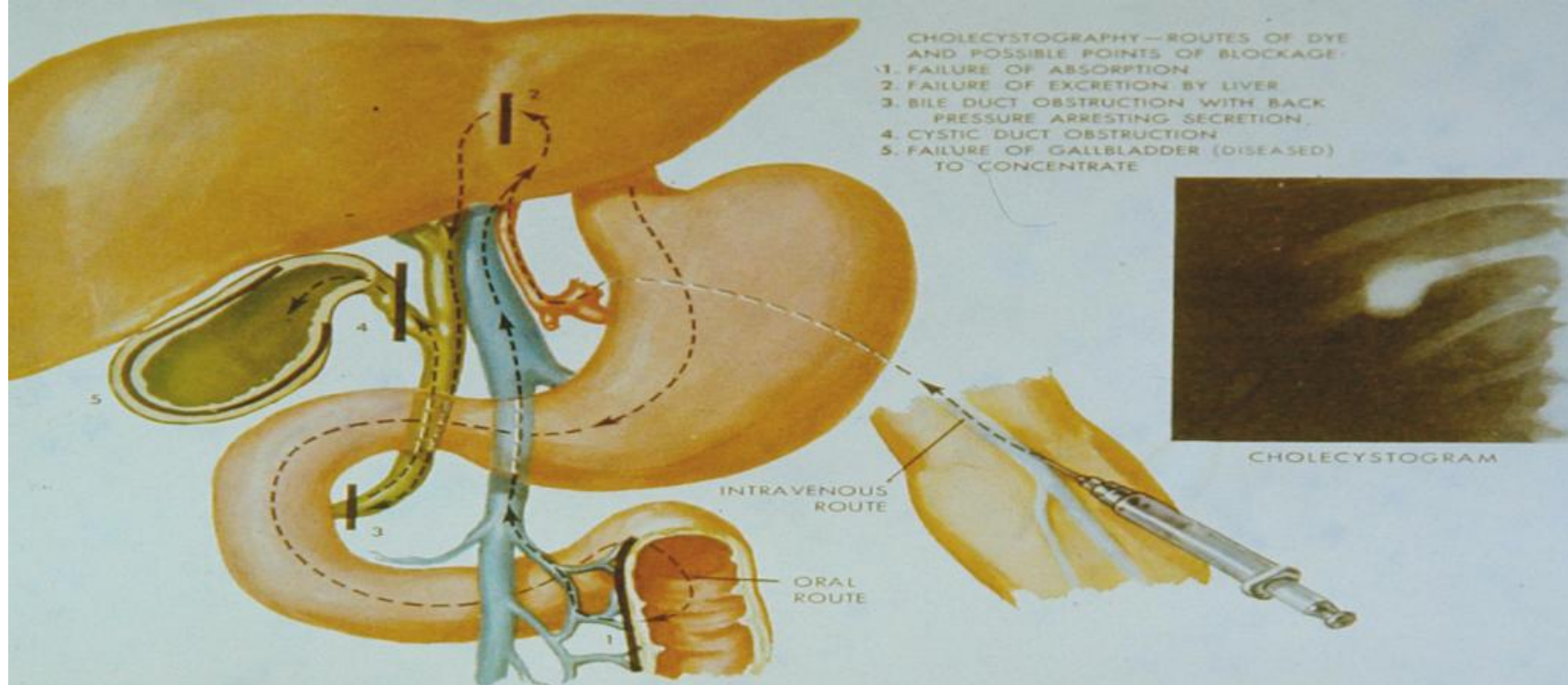
During storage in the gallbladder, **bile becomes more concentrated** which increases its potency and intensifies its effect on fats.

# GALLBLADDER

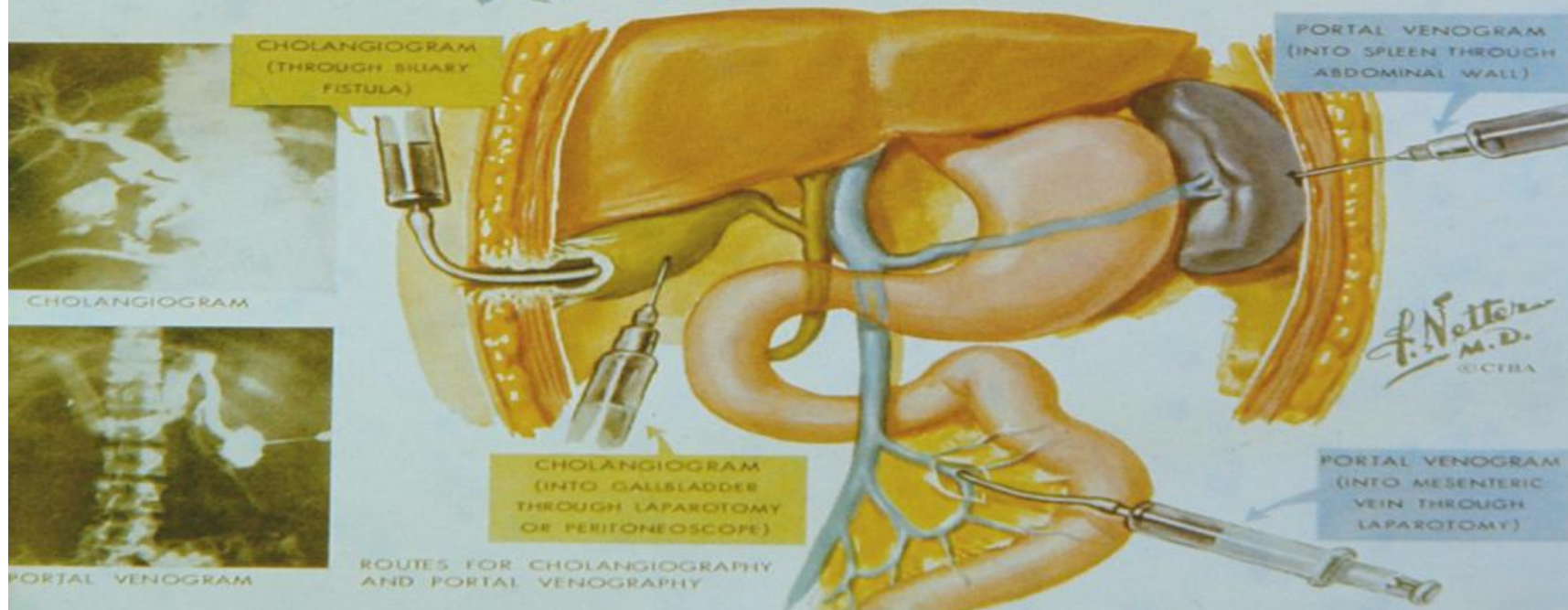




- CHOLECYSTOGRAPHY—ROUTES OF DYE AND POSSIBLE POINTS OF BLOCKAGE
1. FAILURE OF ABSORPTION
  2. FAILURE OF EXCRETION BY LIVER
  3. BILE DUCT OBSTRUCTION WITH BACK PRESSURE ARRESTING SECRETION
  4. CYSTIC DUCT OBSTRUCTION
  5. FAILURE OF GALLBLADDER (DISEASED) TO CONCENTRATE



CHOLECYSTOGRAM



CHOLANGIOGRAM

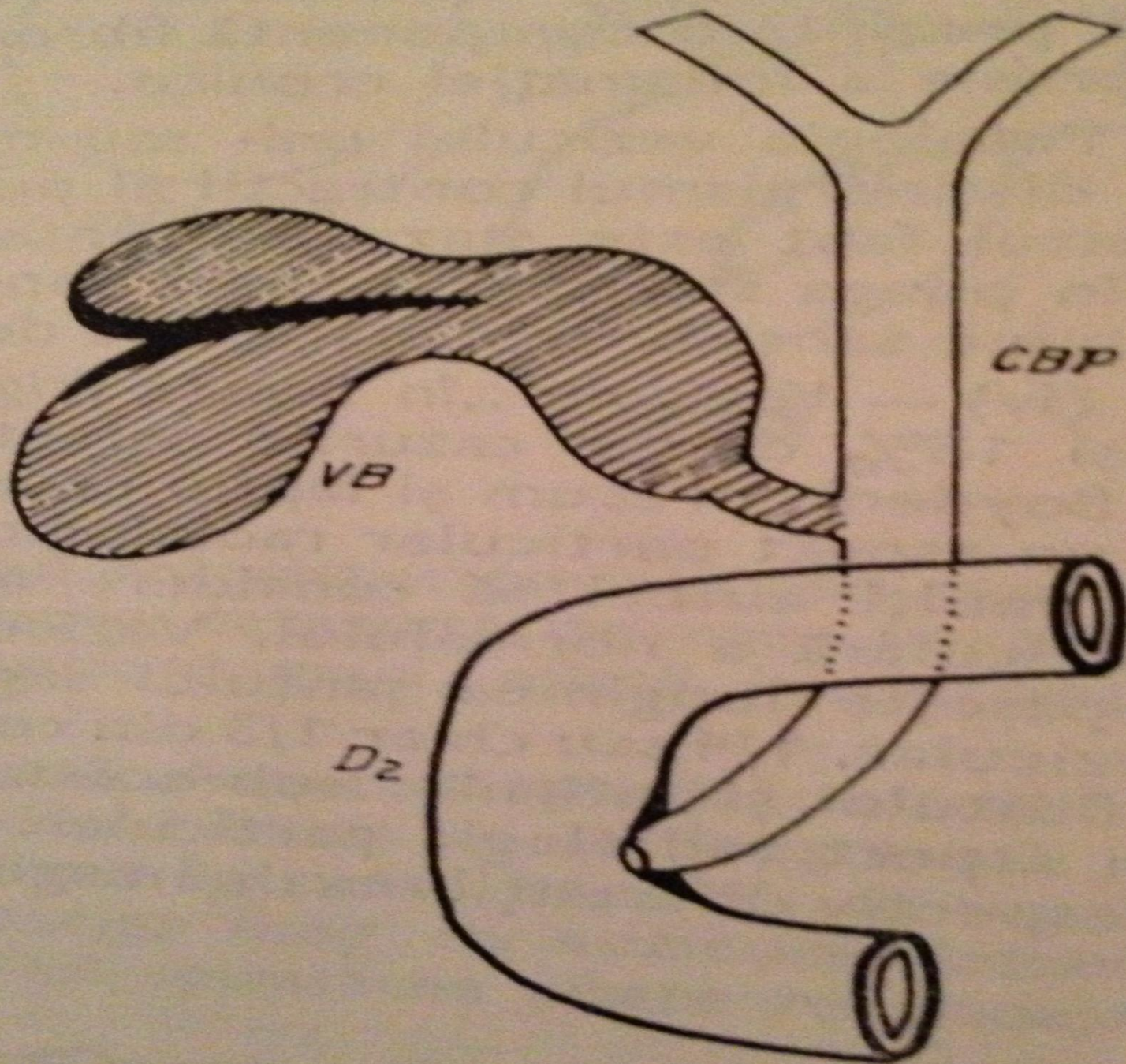


PORTAL VENOGAM

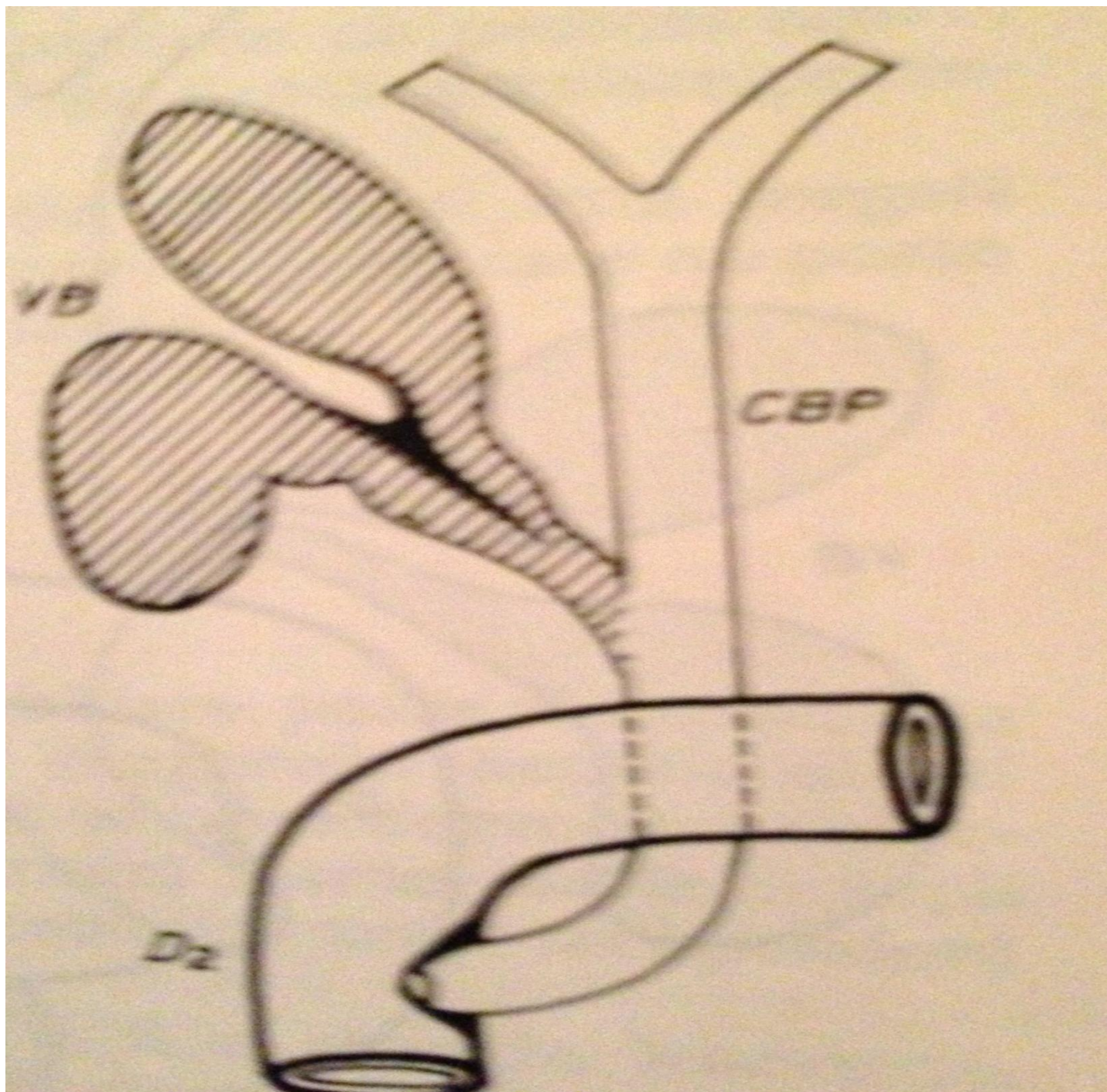
ROUTES FOR CHOLANGIOGRAPHY AND PORTAL VENOGRAPHY

*F. Netter M.D.*  
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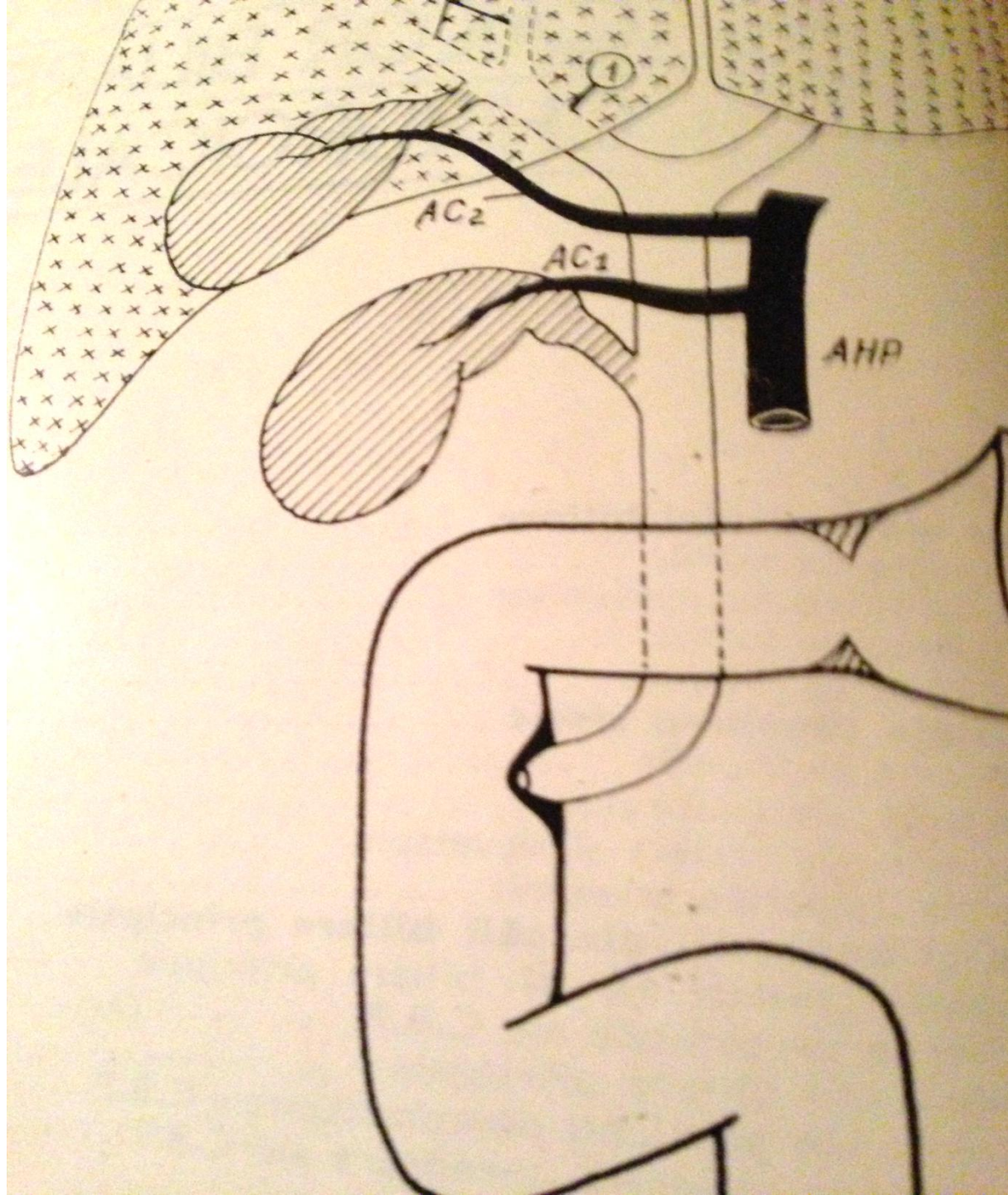




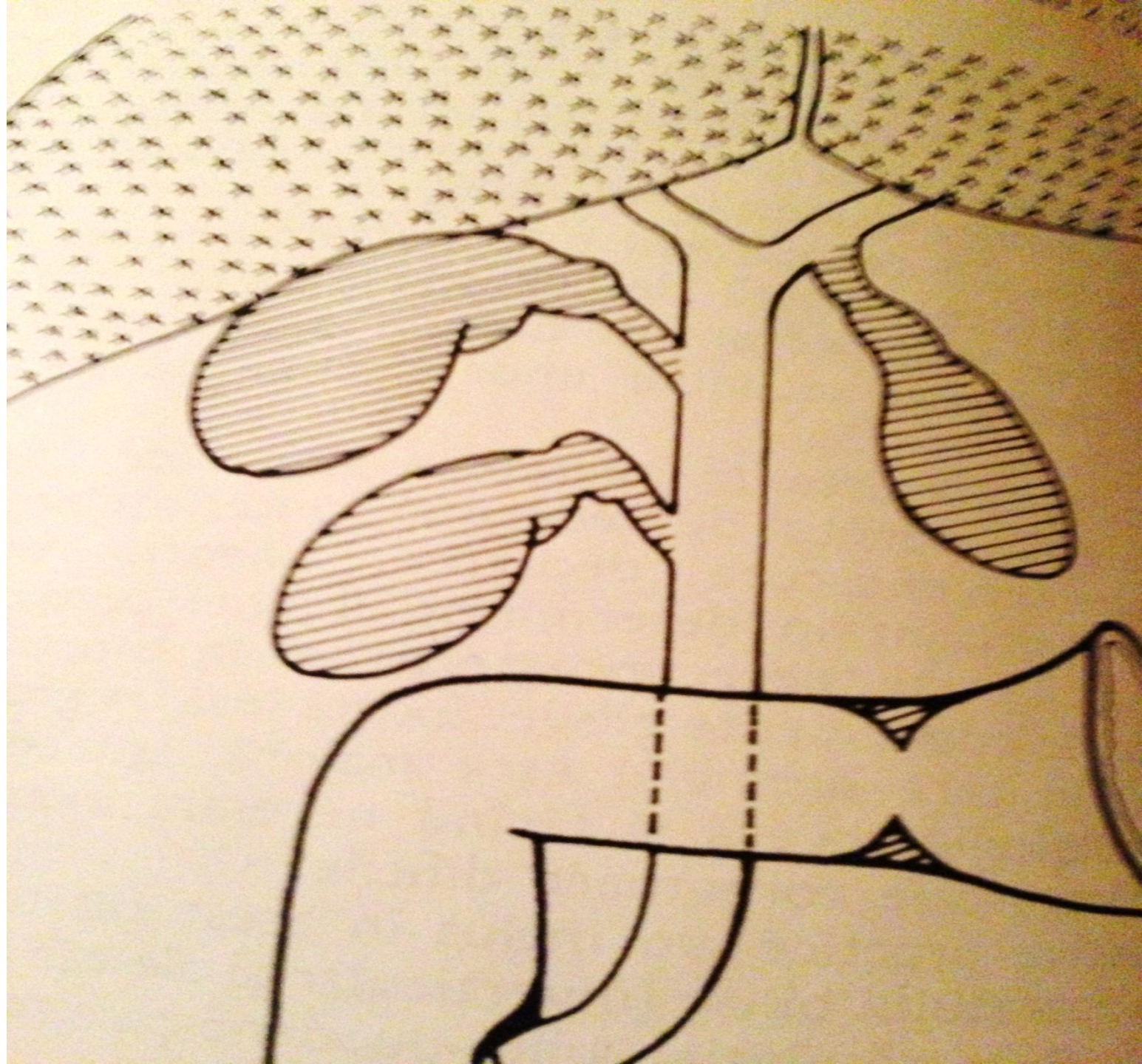














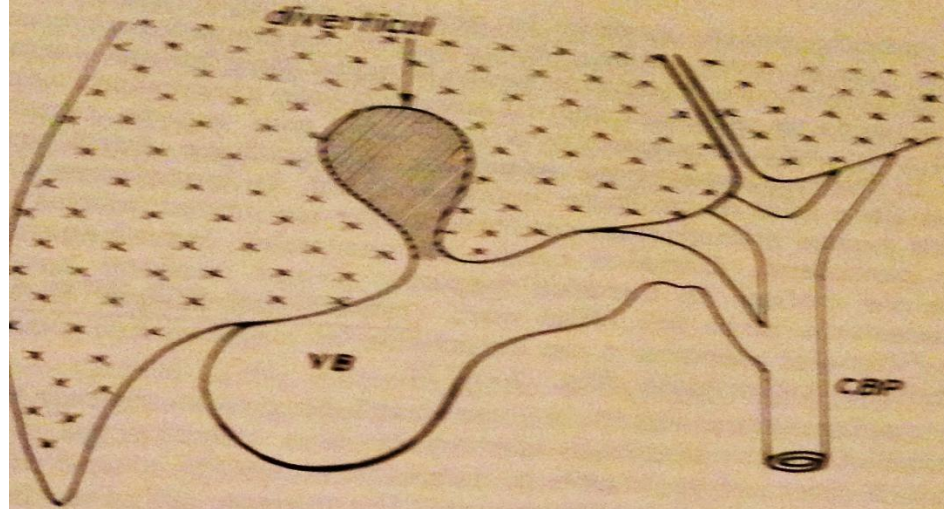
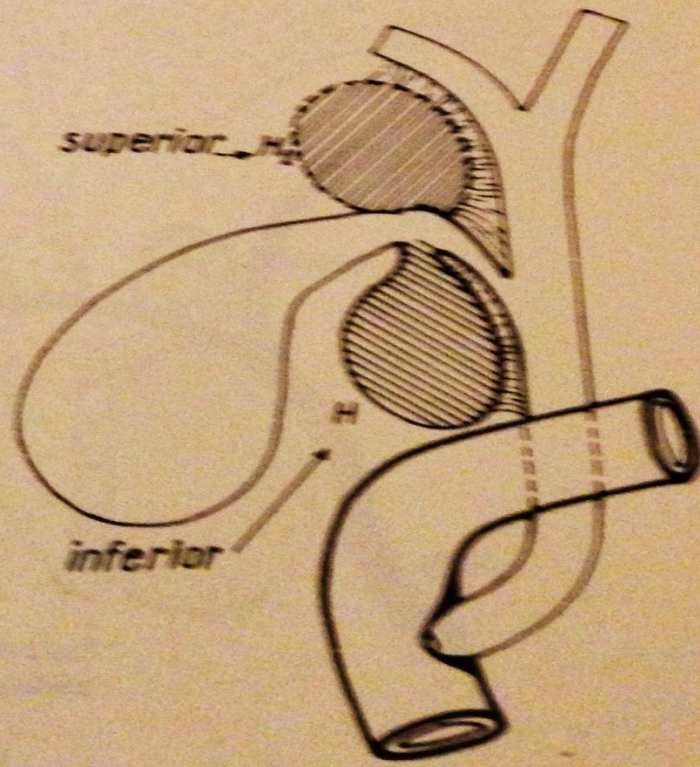


Fig. 1—11. — Diverticul al veziculei biliare dezvoltat superior, total intrahepatic.

2. — Pungă Hartmann  
tare inferioară (H) în  
u canalul coledoc;  
rtmann cu dezvoltare  
(H<sub>1</sub>) în relație cu  
atic comun și canalul  
epatic drept.





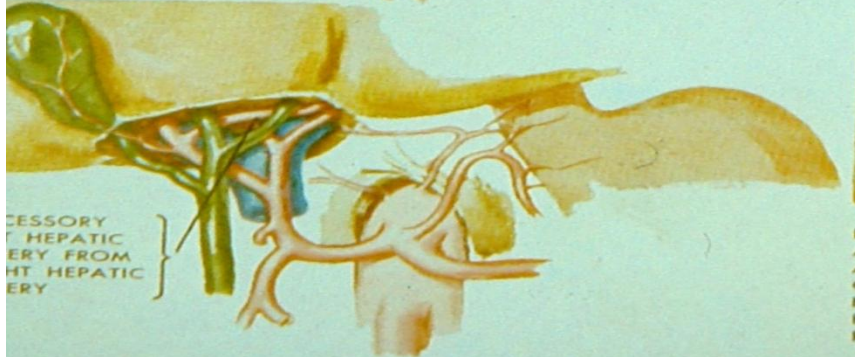
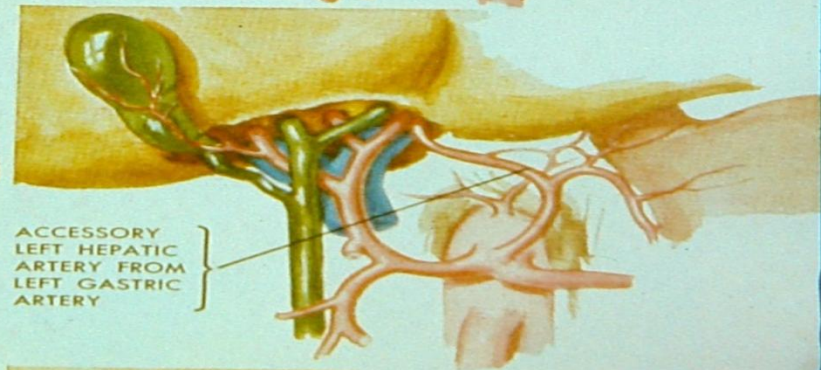
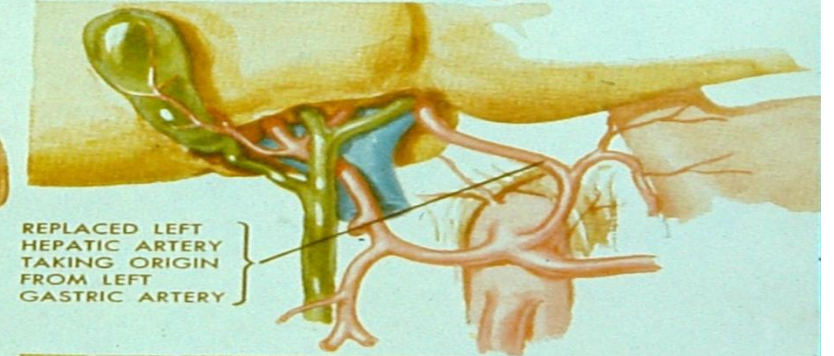
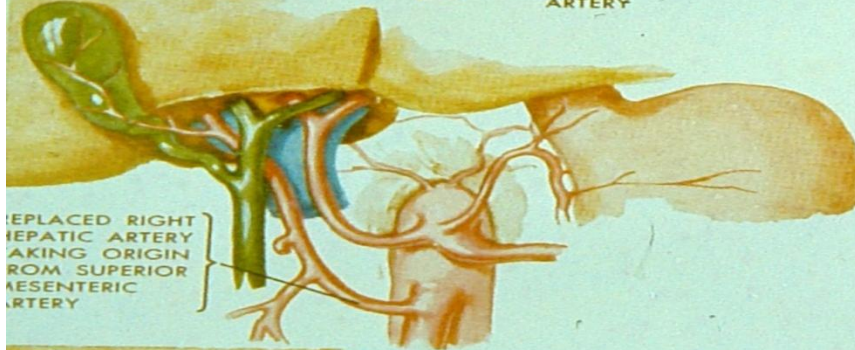
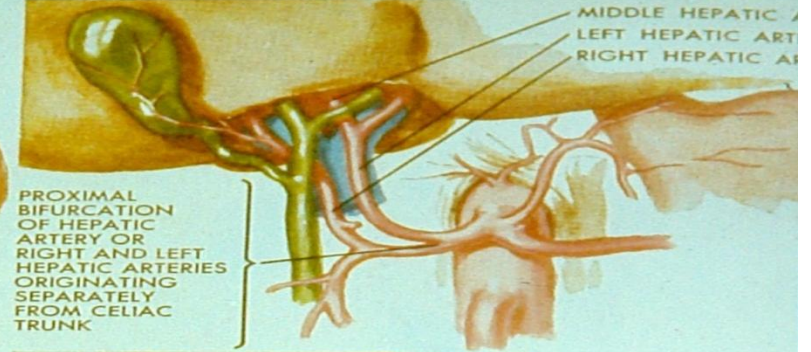
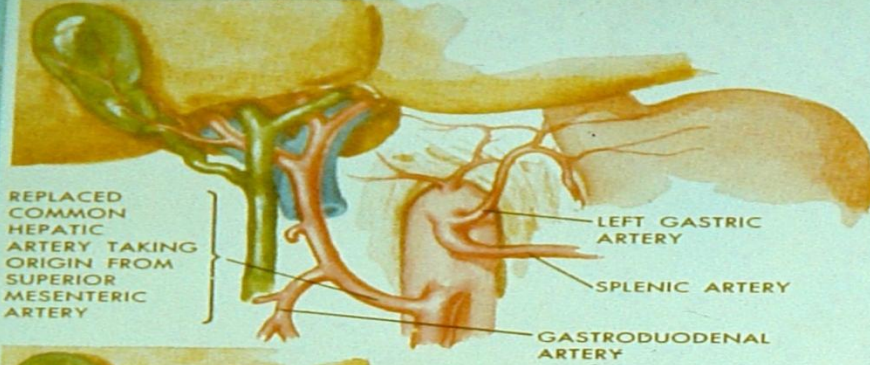
# GALLBLADDER

The **arterial blood supply** of the liver and biliary system is quite **variable**.

Most often the cystic artery arises from the right hepatic artery and is supplied by the celiac axis via the common hepatic artery.

An alternative origin of the right hepatic artery is the superior mesenteric artery.

The cystic duct varies in length from 2 to 4 cm and its site of entry can be either the common duct or occasionally the right hepatic branch.



*f. No.*

# Pathology

A **gallstone** is a crystalline concretion formed within the gallbladder by accretion of bile components.

These calculi are formed in the gallbladder but may distally pass into other parts of the biliary tract such as the cystic duct, common bile duct, or the ampulla of Vater. Rarely, in cases of severe inflammation, gallstones may erode through the gallbladder into adherent bowel potentially causing an obstruction termed **gallstone ileus**

Presence of gallstones in the gallbladder may lead to **acute cholecystitis**, an inflammatory condition characterized by retention of bile in the gallbladder and often secondary infection by intestinal bacteria, predominantly **Escherichia coli** and **Bacteriodes** species. Presence of gallstones in other parts of the biliary tract can cause **obstruction of the bile ducts**, which can lead to serious conditions such as ascending cholangitis or pancreatitis.



**Formation of gallstones** is a complex process that starts with **bile**, a fluid composed mostly of **water, bile salts, lecithin (a fat known as a phospholipid), and cholesterol.**

Most gallstones are formed from cholesterol. Cholesterol is not very soluble, so in order to remain suspended in fluid it must be transported within clusters of bile salts called **micelles.**

If there is an imbalance between these bile salts and cholesterol, then the bile fluid turns to **sludge.**

This thickened fluid consists of a **mucus gel containing cholesterol and calcium bilirubinate.**

If the imbalance worsens, cholesterol crystals form (a condition called *supersaturation*), which can eventually form **gallstones.**

The process of gallstone formation (cholelithiasis) is generally slow, and usually causes no pain or other symptoms.

The majority of gallstones are either the cholesterol or mixed type.

There are many events that may promote cholelithiasis:

- The liver secretes **too much cholesterol** into the bile.
- The **gallbladder may not be able to empty** normally, so bile becomes stagnant.
- The cells lining the gallbladder may not be able to efficiently **absorb cholesterol** and fat from bile.
- There are **high levels of bilirubin**.

Bilirubin is a substance normally formed by the breakdown of hemoglobin in the blood. It is removed from the body in bile.

Some experts believe bilirubin may play an important role in the formation of cholesterol gallstones.



## Pigment Stones.

Pigment stones are composed of calcium bilirubinate, or calcified bilirubin. Pigment stones can be **black** or **brown**.

Black stones form in the gallbladder are the more common type. They represent 20% of all gallstones. They are more likely to develop in people with hemolytic anemia (a relatively rare anemia in which red blood cells are destroyed) or cirrhosis (scarred liver). Brown pigment stones are more common in Asian populations. They contain more cholesterol and calcium than black pigment stones and are more likely to occur in the bile ducts. Infection plays a role in the development of these stones.

## Mixed stones.

Mixed stones are a mixture of cholesterol and pigment stones.

## Risk Factors:

People with **diabetes** are at higher risk for gallstones, and they also have a higher-than-average risk for other gallbladder problems.

Being **overweight** is a significant risk factor for gallstones.

## Identification of gallstones

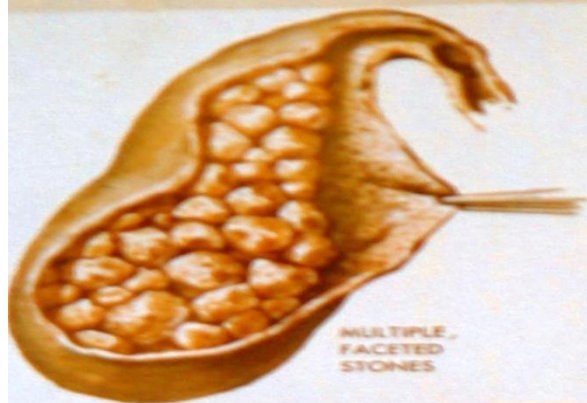
When no symptoms exist, gallstones may be identified during abdominal evaluation for a problem in another organ system.

Diagnosis may be made on

- abdominal roentgenograms,
- by ultrasound scans of the right upper quadrant,

Prophylactic cholecystectomy may be advised for young, healthy patients or for those with associated medical problems that would increase the risk should an acute episode of cholecystitis develop.

Elderly patients (65 years or older) or those with severe medical problems should not have a prophylactic cholecystectomy.



MULTIPLE,  
FACETED  
STONES



LARGE STONE  
AND NUMEROUS  
SMALL ONES:  
CHRONIC  
CHOLECYSTITIS



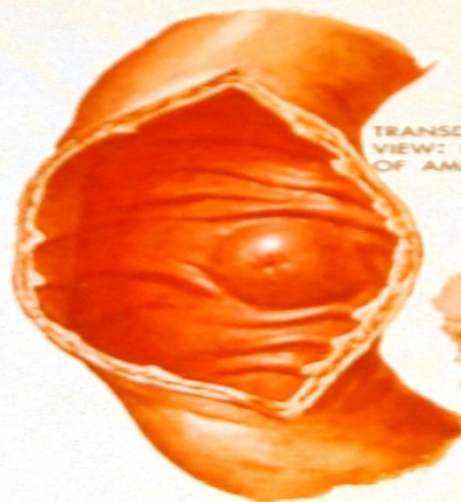
MARKEDLY THICKENED  
GALLBLADDER CONTRACTED  
ABOUT SOLITARY LARGE  
STONE



MULTIPLE,  
FACETED  
STONES IN  
COMMON  
BILE DUCT



SOLITARY  
STONE IN  
COMMON  
DUCT



TRANSDUODENAL  
VIEW: BULGING  
OF AMPULLA



AMPULLARY STONE



INTRAHEPATIC  
STONES

*F. Netter M.D.*  
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Biliary disease is a common problem in Western societies.

Approximately 15 % to 20 % of adults have gallstones; of persons age 75 and older, an estimated 35 % to 50 % have gallstones.

Most of operations are performed for inflammatory biliary disease :

- acute and chronic cholecystitis,
- cholangitis caused by stones in common bile duct
- primary inflammatory changes in the bile duct wall,
- obstructive jaundice from bile duct stones.

Congenital anomalies: biliary atresia and choledochal cysts account for small percentage of biliary problems.

Neoplasms of the biliary tree are not common but are occurring more frequently.

# GALLBLADDER



The 3 most common signs of biliary disease:

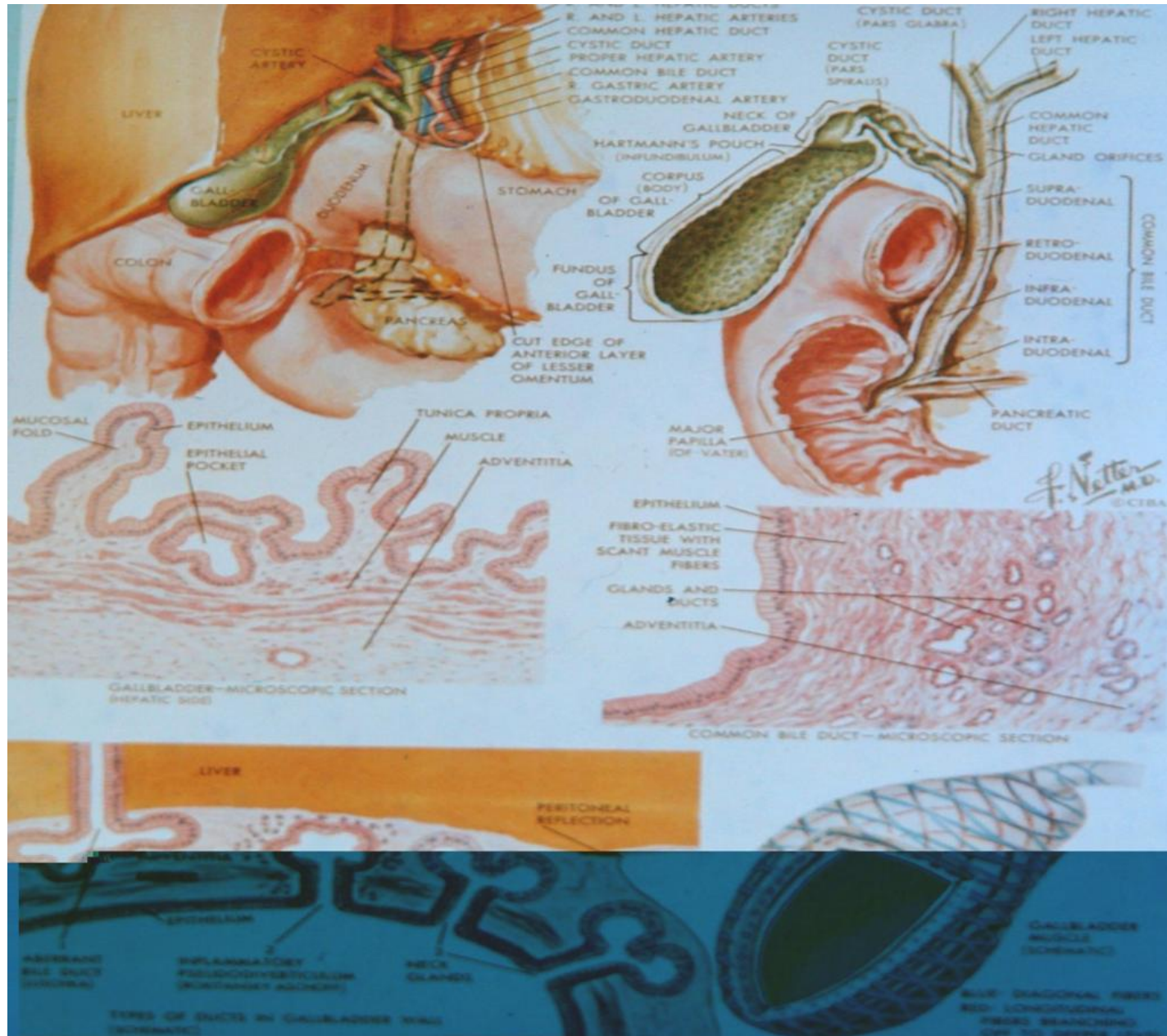
- **pain in the right upper abdomen** most often intermittent or **colicky** but also may be steady and radiate into the back,
- **fever** that is usually low grade but may be septic in nature, elevated in the morning, and
- **jaundice** caused by either an obstruction of bile flow or inflammatory changes in the liver, with diminished or altered metabolic function of bile excretion.

Other symptoms of biliary disease include:

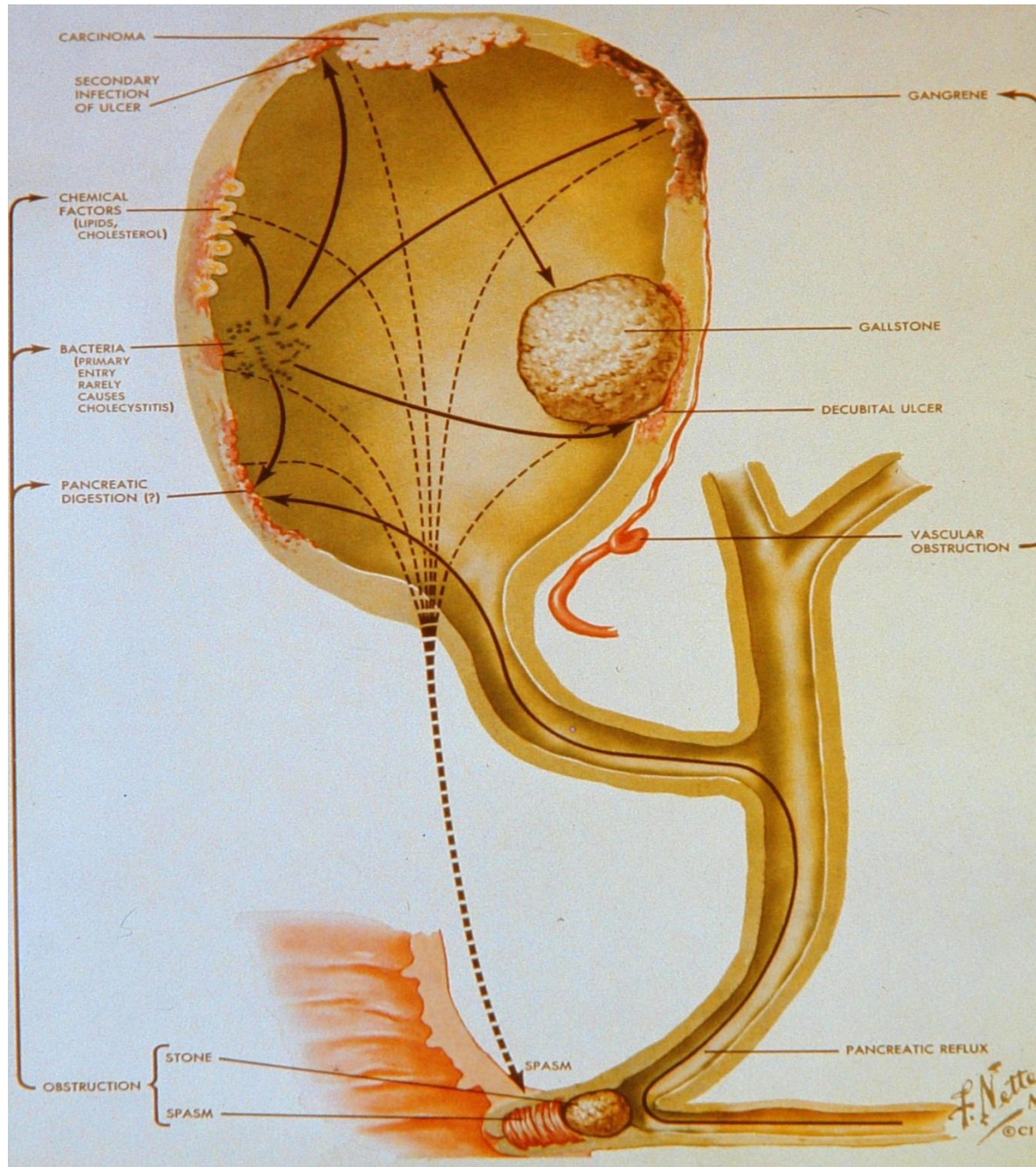
- nausea and vomiting,
- bloating,
- fatty food intolerance and
- weight loss.



# GALLBLADDER



# GALLBLADDER





# Acute cholecystitis

## Subjective findings:

- patients usually have severe epigastric or upper quadrant pain that radiates to right scapula
- nausea and vomiting.

The pain initially is colicky and then becomes steady and radiates to the shoulder or the back.

Vomiting is usually not severe and is normally preceded by the pain.

# Acute cholecystitis

**Objective findings:** the right upper abdomen is tender with localized peritoneal signs.

**Murphy's sign** is present if deep inspiration with cessation of breathing causes tenderness or pain as gallbladder descends against the examiner's hand.

A low-grade fever is often present.

A tender mass that represents the inflamed gallbladder may be palpable.

As the inflammatory process progresses, rigidity and guarding become more prominent. Perforation of the gallbladder results in generalized peritonitis or abscess.



# GALLBLADDER



# GALLBLADDER





# Acute cholecystitis

## Diagnostic plan.

- ☐ Leukocytosis is common
- ☐ In 20 % of patients the bilirubin level may be as great as 5 mg/dl.
- ☐ Values greater than 5 mg/dl imply common bile duct stones.
- ☐ Serum amylase should be measured (elevated serum amylase likely represents associated pancreatic inflammation).
- ☐ Ultrasonography of the gallbladder accurately identifies gallstones or other biliary pathologic conditions in greater than 90 % of patients.

# Acute cholecystitis

## Differential diagnosis:

- ☐ Acute pancreatitis
- ☐ Ulcer disease

## Complications

- ☐ Abscess
- ☐ Perforation
- ☐ Gallstone ileus
- ☐ Acute pancreatitis



# Acute cholecystitis

## Management plan.

**The initial** management of acute cholecystitis is non-operative. Intravenous fluid is administered to correct volume and electrolyte deficits.

No oral intake is permitted and nasogastric suction is added if the patient has vomited or has gastric ileus.

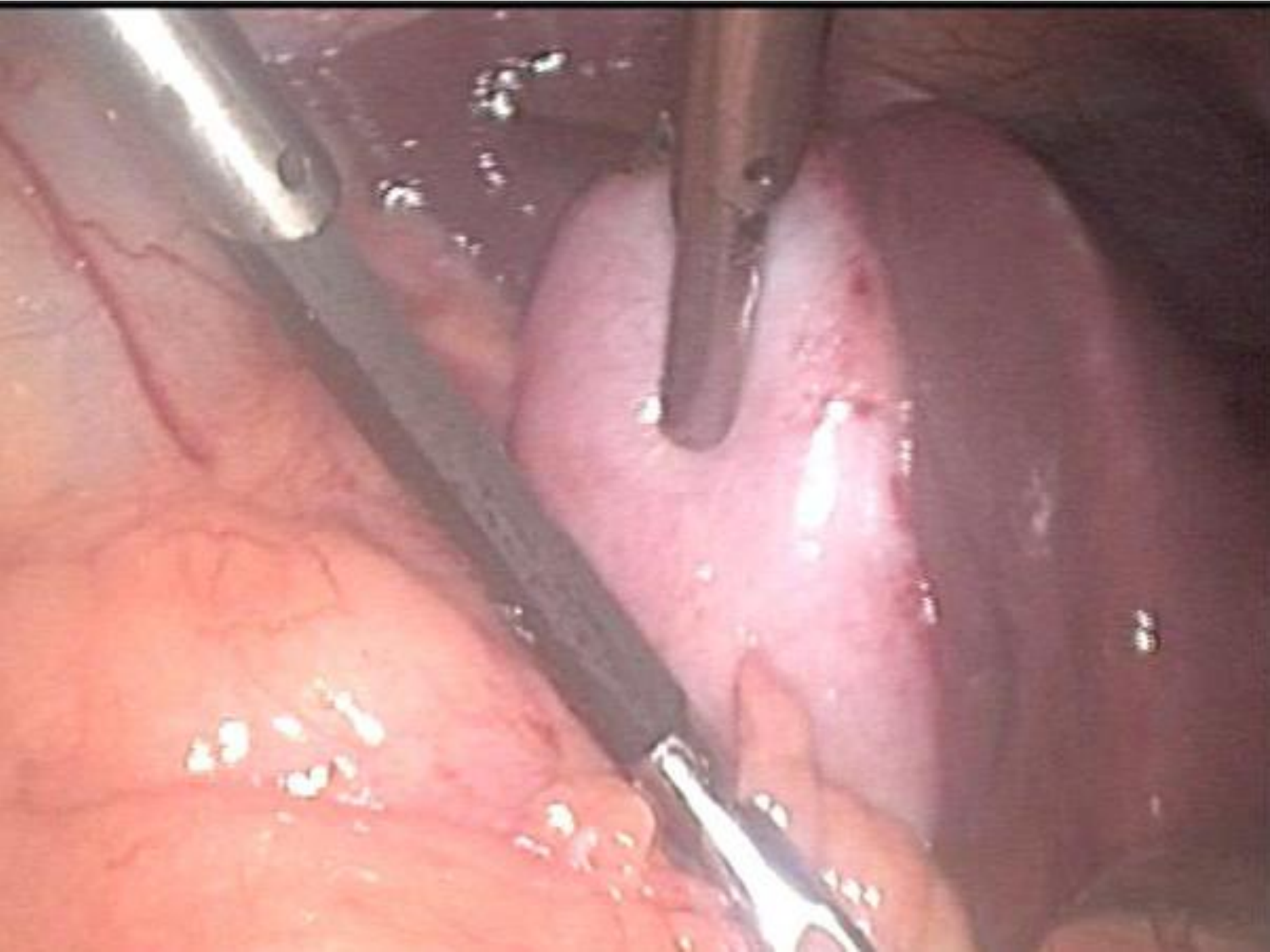
Analgesics and intravenous broad-spectrum antibiotics, such as second-generation cephalosporins are given.

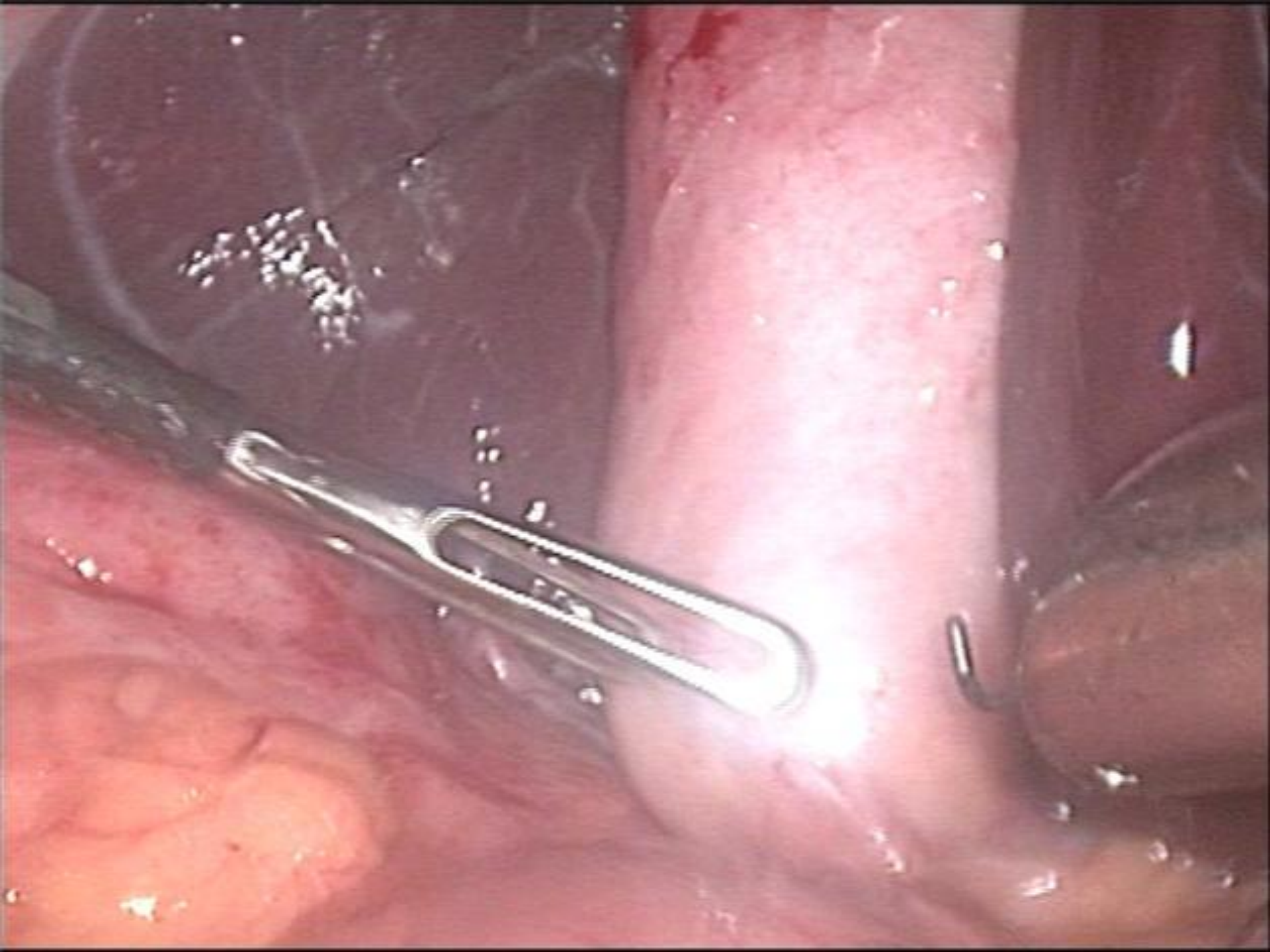
More than 90 % of patients will improve dramatically in 24 to 48 hours with this regimen.

**Urgent surgical intervention** is necessary for patients who develop **peritonitis** or who **deteriorate despite early medical therapy**.

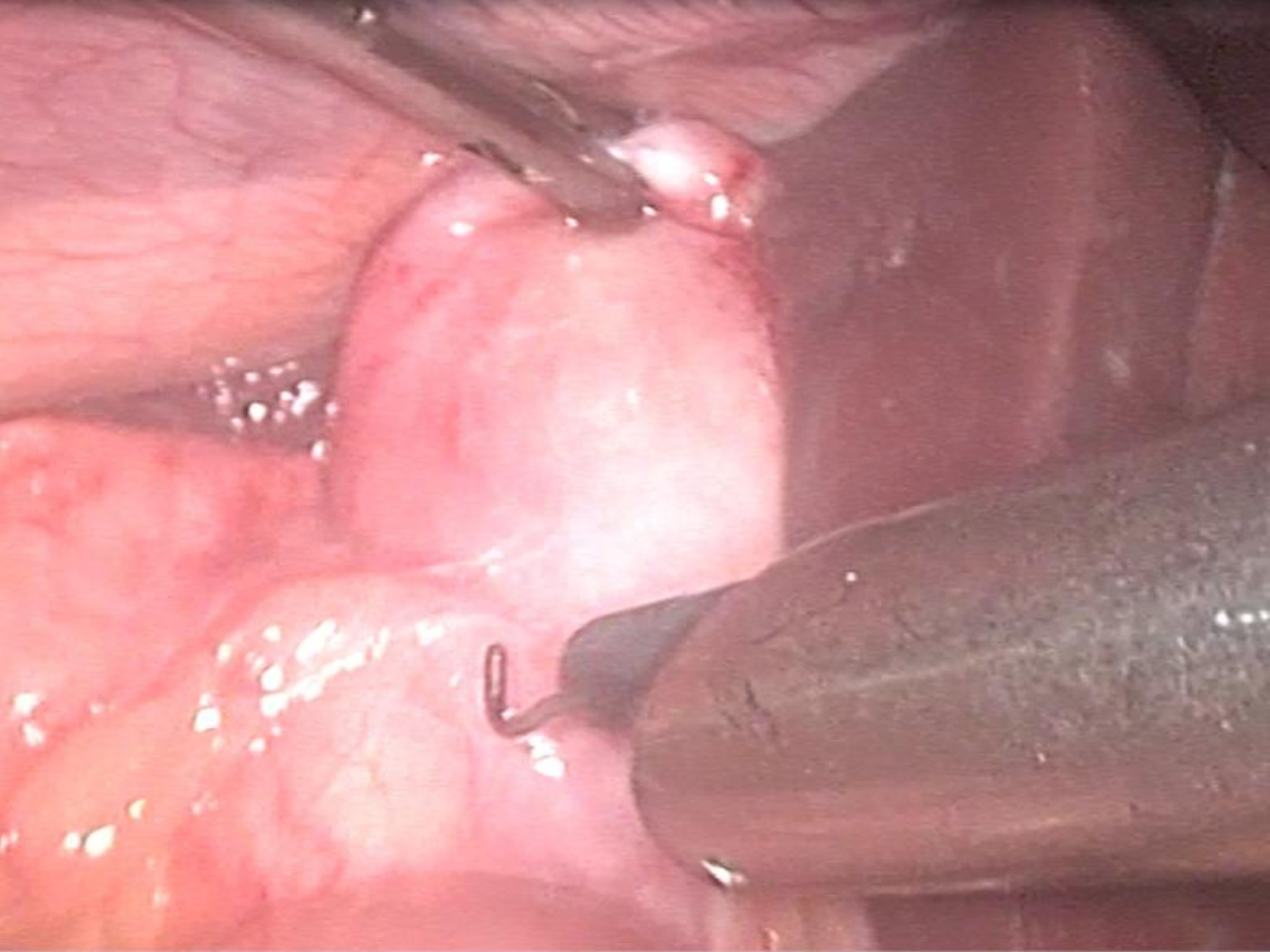
## Acute cholecystitis

- **Cholecystectomy**, which should be performed within 72 hours of admission (early cholecystectomy) is the definitive treatment for acute cholecystitis.
- **Intraoperative cholangiogram**
- **Cholecystostomy** if removal not possible





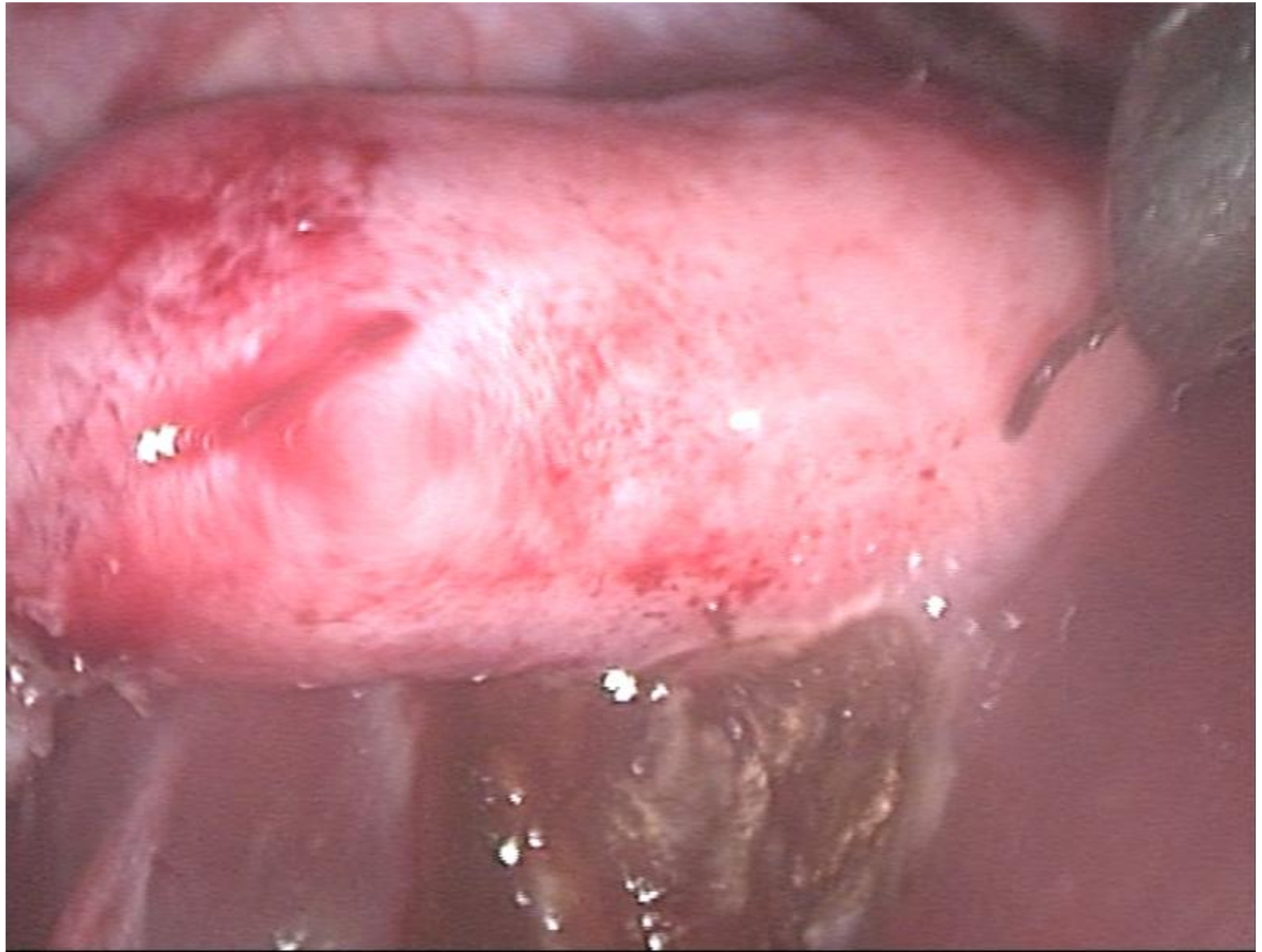












# Chronic cholecystitis

Chronic cholecystitis is the most common form of symptomatic inflammatory gallbladder disease.

## Subjective findings.

**Biliary colic** is the most specific symptom. Biliary colic **attacks**, located **in the epigastrium or right upper abdomen**, are typically sudden in onset, and may radiate to the shoulder or back. The pain may last for 30 to 60 minutes or occasionally-for several hours.

Many patients experience nausea with the colic episodes. Nonspecific but commonly associated complaints include belching, bloating, flatulence or a feeling of upper abdominal fullness.



# Chronic cholecystitis

## Subjective findings

- Epigastric pain radiates to right scapula, associated with fatty meals,
- nausea,
- belching,
- bloating,
- flatulence

## Objective findings.

Most patients with chronic cholecystitis have normal physical examination and laboratory test results.

Some patients may have mild tenderness in the upper abdomen or a palpable mass suggestive of a chronic hydrops of the gallbladder.

# **Chronic cholecystitis**

## **Diagnostic plan.**

- Ultrasonography confirms the presence of stones or a contracted gallbladder.
- Oral cholecystography may demonstrate gallstones and failure to opacify the gallbladder after 2 doses of telepaque tablets indicates with 95 % accuracy a diseased gallbladder.

## **Differential diagnosis:**

- Ulcer disease
- Gastritis
- Hiatus hernia
- Gastric neoplasm

# Chronic cholecystitis

## Management plan.

**Cholecystectomy** is the treatment of choice in most cases. Cholecystectomy is indicated to prevent complicated biliary problems such as cholangitis or abscess formation.

**Conservative measures** are reserved for patients who represent prohibitive surgical risks or who refuse surgery. A low-fat diet and antispasmodic therapy, such as dicyclomin hydrochloride (Bentyl) are standard recommendations.

## Complications

- Acute cholecystitis
- Common duct stones
- Jaundice
- Cholangitis
- Pancreatitis
- Gallstone ileus

# Chronic cholecystitis

The primary advantages of surgically removing of the gallbladder over nonsurgical treatment are that it can:

- ❑ eliminate gallstones,
- ❑ prevent
  - complications and
  - gallbladder cancer.



## *Open Procedures Versus Laparoscopy.*

**Until the early 1990s, open cholecystectomy (the removal of the gallbladder through a wide abdominal incision) was the standard treatment.**

**Now, laparoscopic cholecystectomy (commonly called lap choly), which uses small incisions, is the most commonly used surgical approach. First performed in 1987, lap choly is now used in most cholecystectomies everywhere.**

**Surgical Clinic 1 Febr. 1994**

# Chronic cholecystitis

**Laparoscopy** has largely replaced open cholecystectomy because it offers some significant **advantages**:

The patient can leave the hospital and resume normal activities earlier, compared to open surgery.

The incisions are small, and there is **less postoperative pain** and disability than with the open procedure.

There are **fewer complications**.

It is less expensive than open cholecystectomy over the long term.

# Chronic cholecystitis

The immediate treatment cost of laparoscopy may be higher than the open procedure, but the more rapid recovery and fewer complications translate into shorter hospital stays and fewer sick days, and therefore a greater reduction in overall costs.

Some experts believe, however, that the **open procedure** still has a number of **advantages** compared to laparoscopy:

It is faster to perform.

It poses less of a risk for bile duct injury compared with laparoscopy.

However, open surgery has more overall complications than laparoscopy, and bile-duct injury rates with laparoscopy are declining.

# Chronic cholecystitis

## Cholecystectomy

**Laparoscopic Cholecystectomy  
is the gold standard treatment.**

Classically cholecystectomy is performed through either a

- **right subcostal incision** or
- **an abdominal midline incision.**

If the dissection appears uncomplicated some surgeons will first dissect the cystic duct and the cystic artery duct and then dissect the gallbladder from the liver bed (**retrograde cholecystectomy**).

Probably safest, however is to perform all cholecystectomies by first dissecting the gallbladder and carrying the dissection down to the junction of the cystic duct and common bile duct, **anterograde cholecystectomy**.



# Chronic cholecystitis

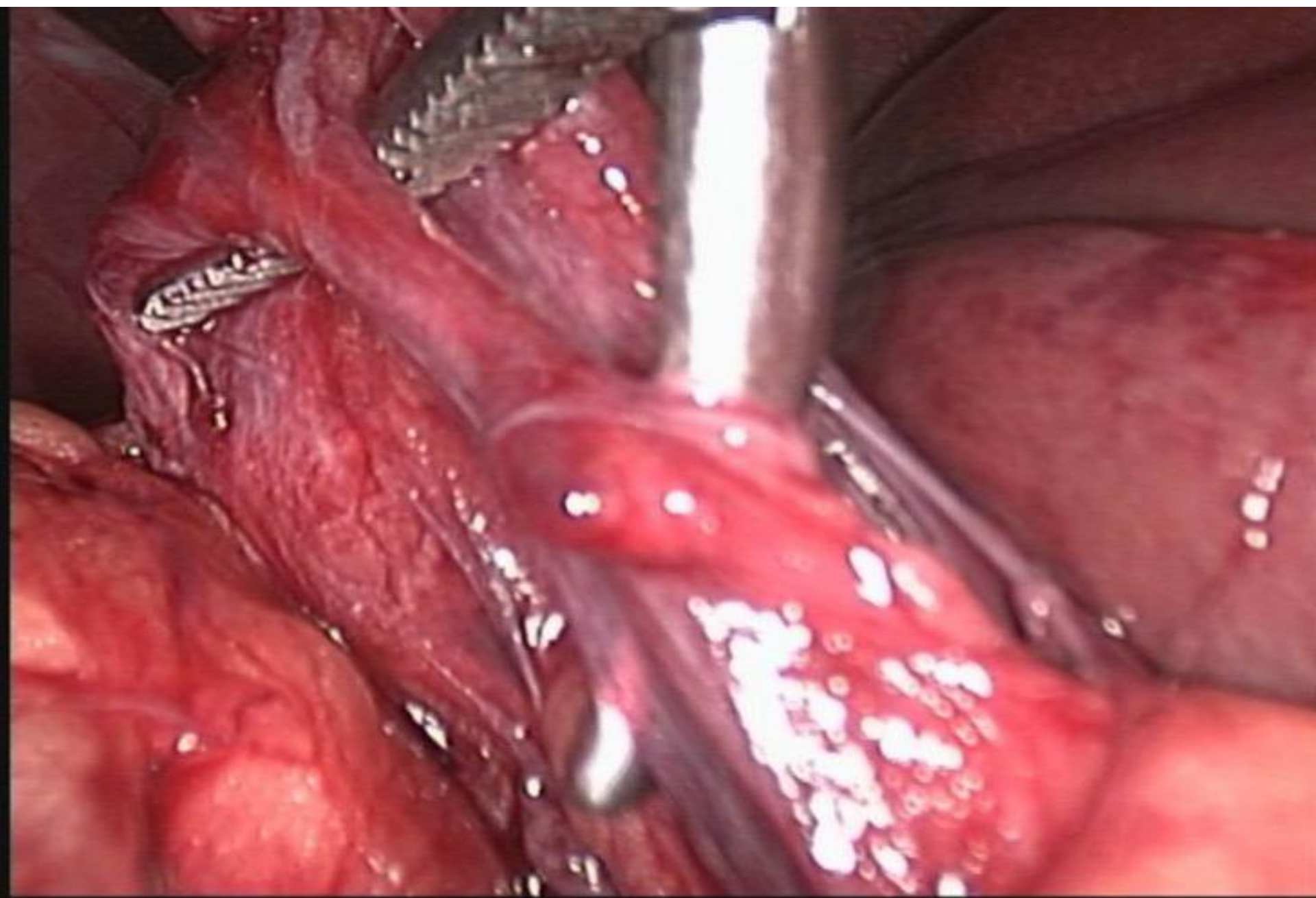
## Cholecystectomy

With traction of the gallbladder fundus, the cystic artery will be taut. The cystic artery should be isolated, clamped, divided and ligated with a silk ligature or clip. The cystic duct is dissected only to the point where the first ductal structure is identified. The stones in the duct are milked back into the gallbladder side of the duct for traction. The cystic duct is then opened or partially divided for an operative cholangiogram.

An **operative cholangiogram** should be a routine part of cholecystectomy. Closure of the liver bed is optional.

A **drain** is not routinely placed after cholecystectomy for chronic cholecystitis, however it is used after cholecystectomy for acute cholecystitis, after exploration of the common bile duct and occasionally if hemostasis is incomplete or if additional problems were found.

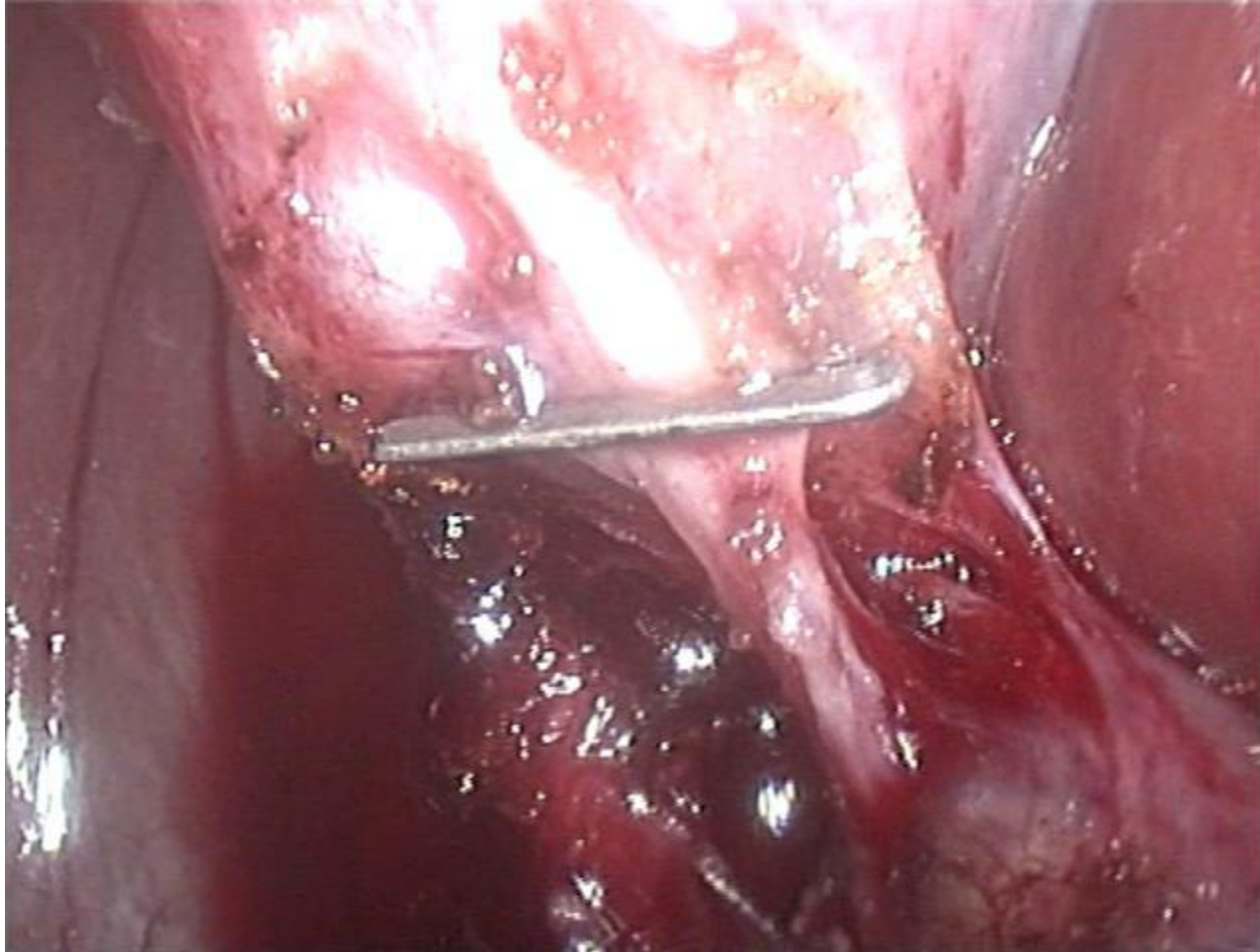
# GALLBLADDER



# GALLBLADDER



# GALLBLADDER





# GALLBLADDER



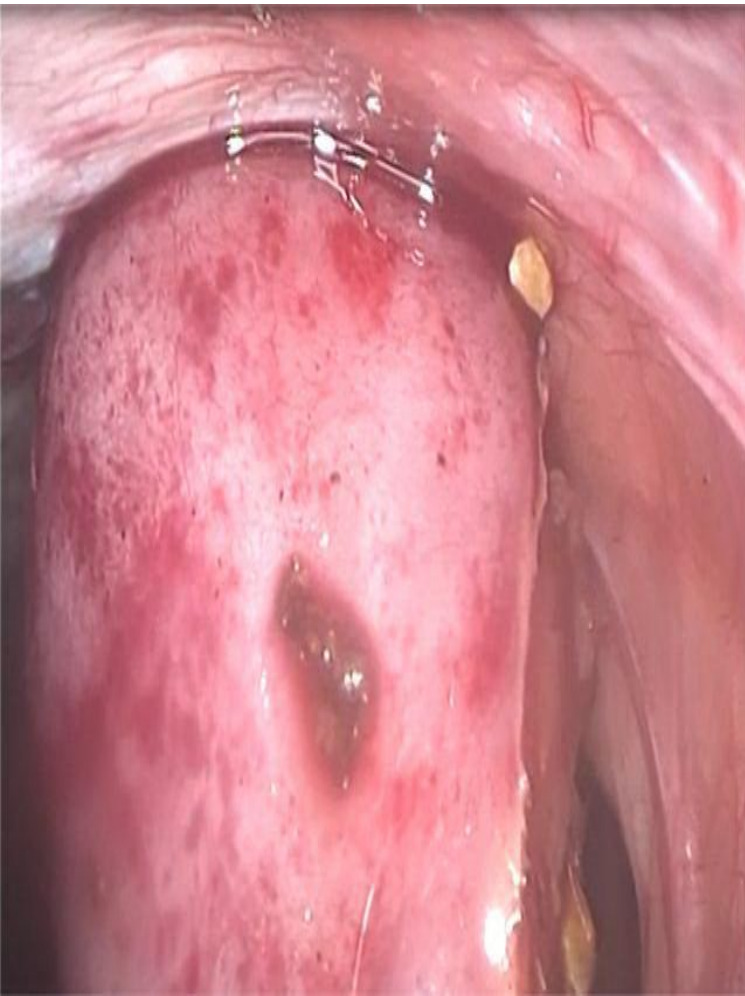
# GALLBLADDER





# GALLBLADDER

Biliar calculi lost into the abdominal cavity are usually fixed on the liver surface, triggering liver micro-abscesses.







## **Gallstone ileus**

**Gallstones ileus occurs when a gallstone erodes through the wall of the gallbladder into the gastrointestinal tract, usually the duodenum and causes obstruction of the small bowel.**

**Right upper abdominal pain or discomfort is frequently minimal.**

**Usually in elderly**

### **Subjective findings:**

- crampy abdominal pain,
- vomiting,
- abdominal distention and
- lack of bowel movements or gas passage.

## **Gallstone ileus**

### **Diagnostic plan.**

Laboratory studies frequently show leukocytosis.

Liver function studies may show elevated bilirubin or alkaline phosphatase level.

A significant diagnostic finding is **air in the biliary tree** from the biliary intestinal fistula.

### **Management plan.**

Removal of gallstone later repair fistula and cholecystectomy

Remove retained stones

**Cancers of the gallbladder** are staged according to their depth of penetration and extend of spread.

These cancers frequently spread to the liver ,which is involved in 70% of patients at the time of surgical evaluation.

Malignant tumors of the gallbladder can also directly invade other adjacent organs,

- particularly the common bile duct,
- the duodenum and
- the transverse colon.

Gallbladder cancers are insidious in their growth often metastasizing early before a diagnosis is made.

Tumors can also perforate the wall of the gallbladder, eventually causing intra-abdominal metastases, carcinomatosis and ascites.

More than 75% of the carcinomas of the gallbladder are associated with cholelithiasis.

Survival correlates with the stage of disease.

## **Clinical findings:**

- Symptoms of cholecystitis or jaundice,
- Mass in right upper abdomen

## **Differential diagnosis**

Cholecystitis

## **Diagnosis**

- Liver function tests
- Ultrasound
- CT

## **Treatment**

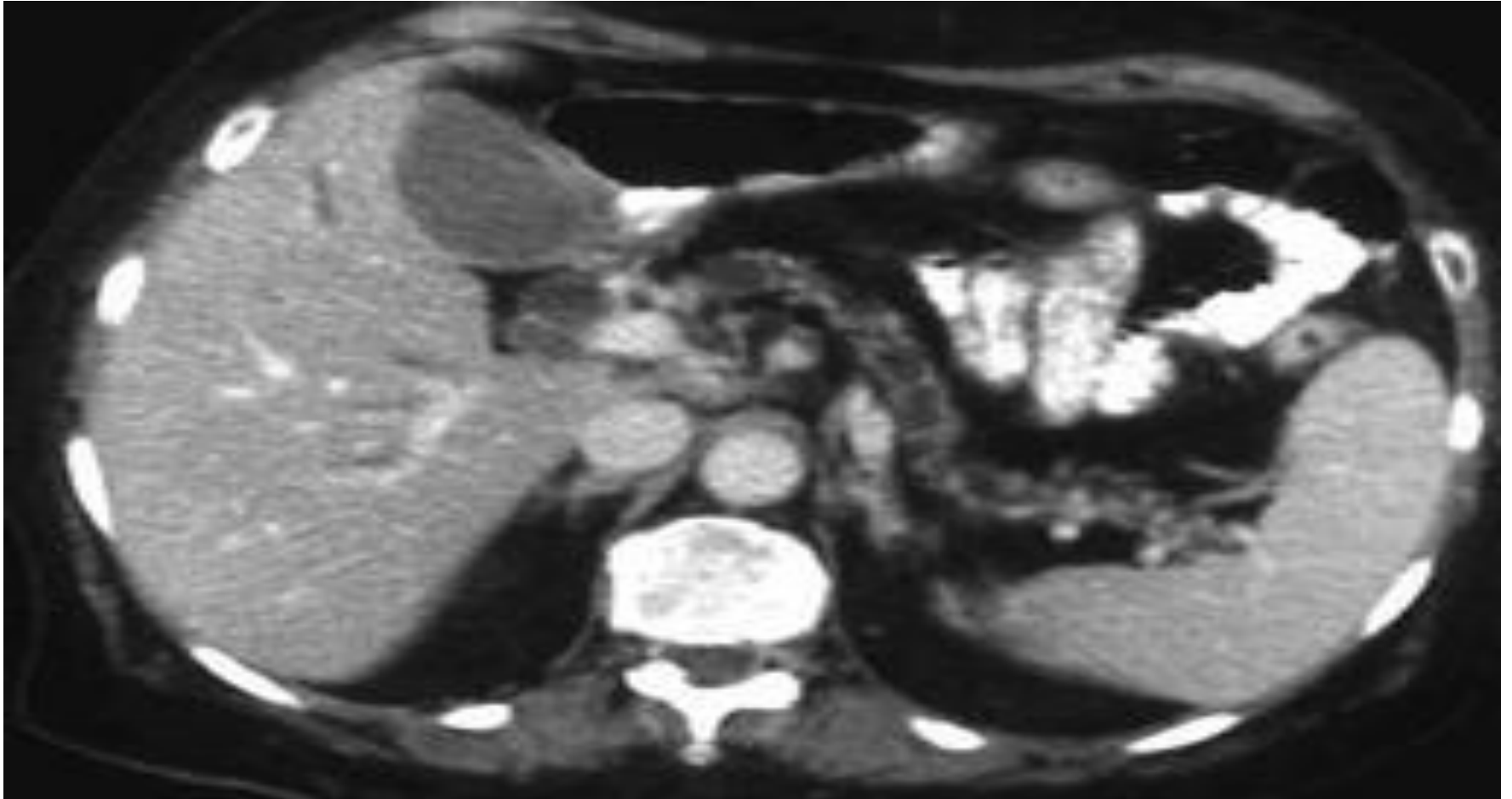
- Radical cholecystectomy
- Palliative drainage

Poor prognosis



# GALLBLADDER

**gallbladder carcinoma**



# **cystic disease of the biliary tree.**

## **Choledochal Cyst**

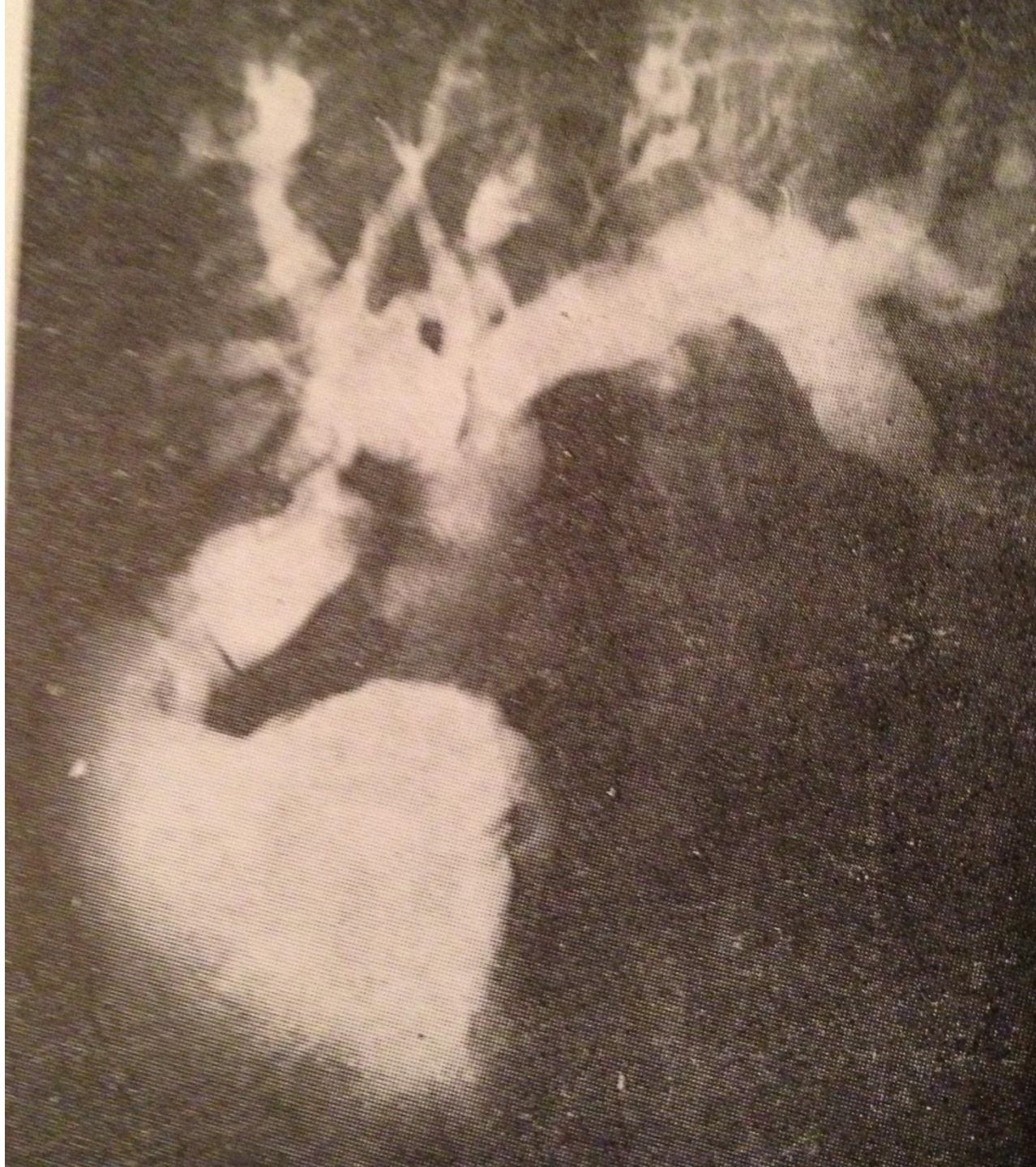
**In this condition, the main trunk (the common bile duct) of the biliary tree is structurally abnormal, probably congenital. The bile accumulates in the duct. It forms a sack or cyst which then presses on the bile duct and prevents bile from reaching the intestine. Bile backs up into the liver and the patient becomes jaundiced (yellow).**

**Occasionally this accumulation of bile becomes infected, causing abdominal pain and fever. In some patients the cyst can be felt by examining the abdomen. In most patients the diagnosis can be confirmed by ultrasound or by CT.**

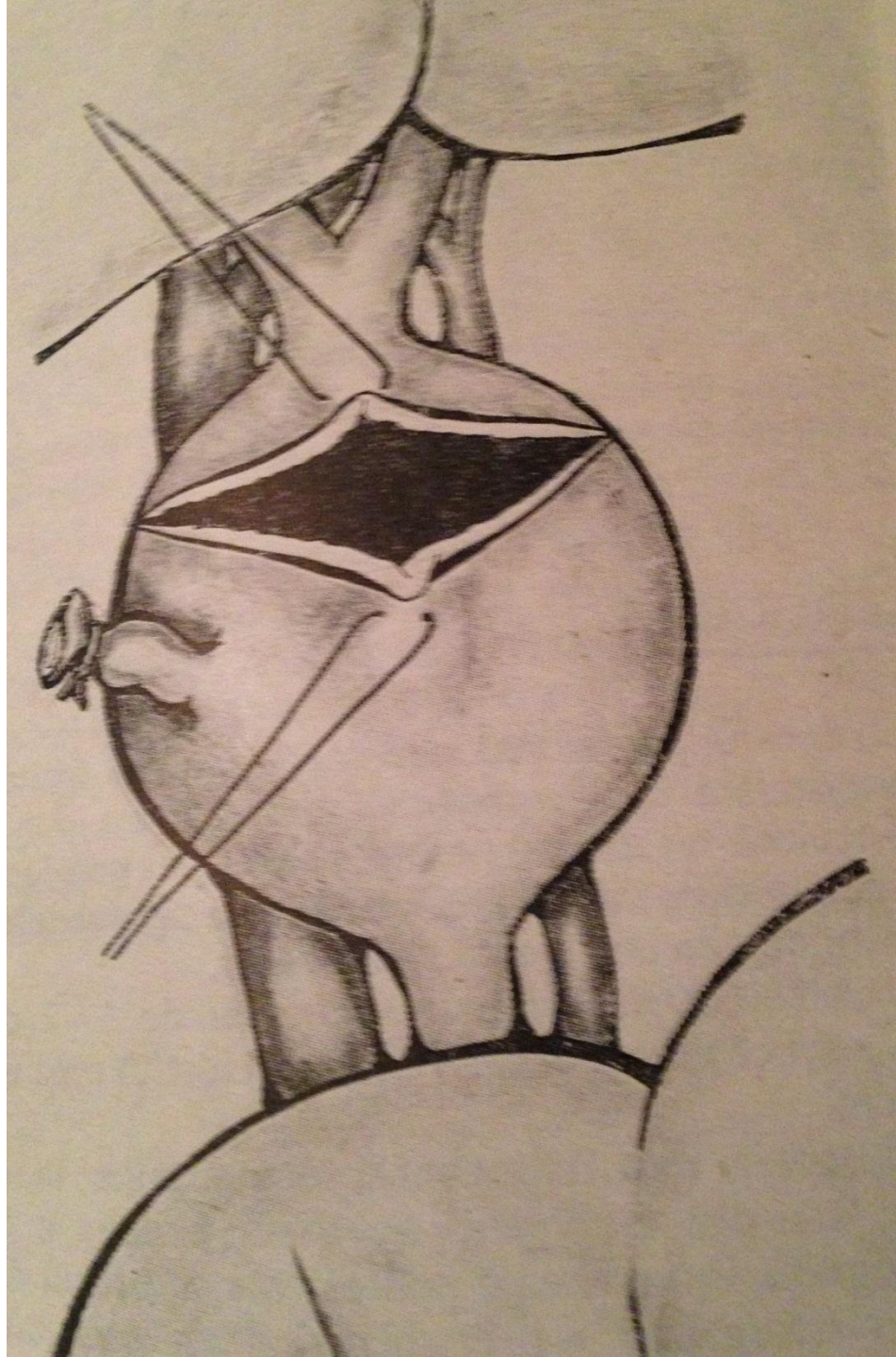
**Treatment is surgical. The abnormal bile duct is removed and a piece of intestine used to replace it. In most cases, surgery permanently corrects the disease. Rarely, infection in the newly formed biliary tree recurs. If the condition is not correctly diagnosed the blockage of bile may result in scarring in the liver (cirrhosis).**

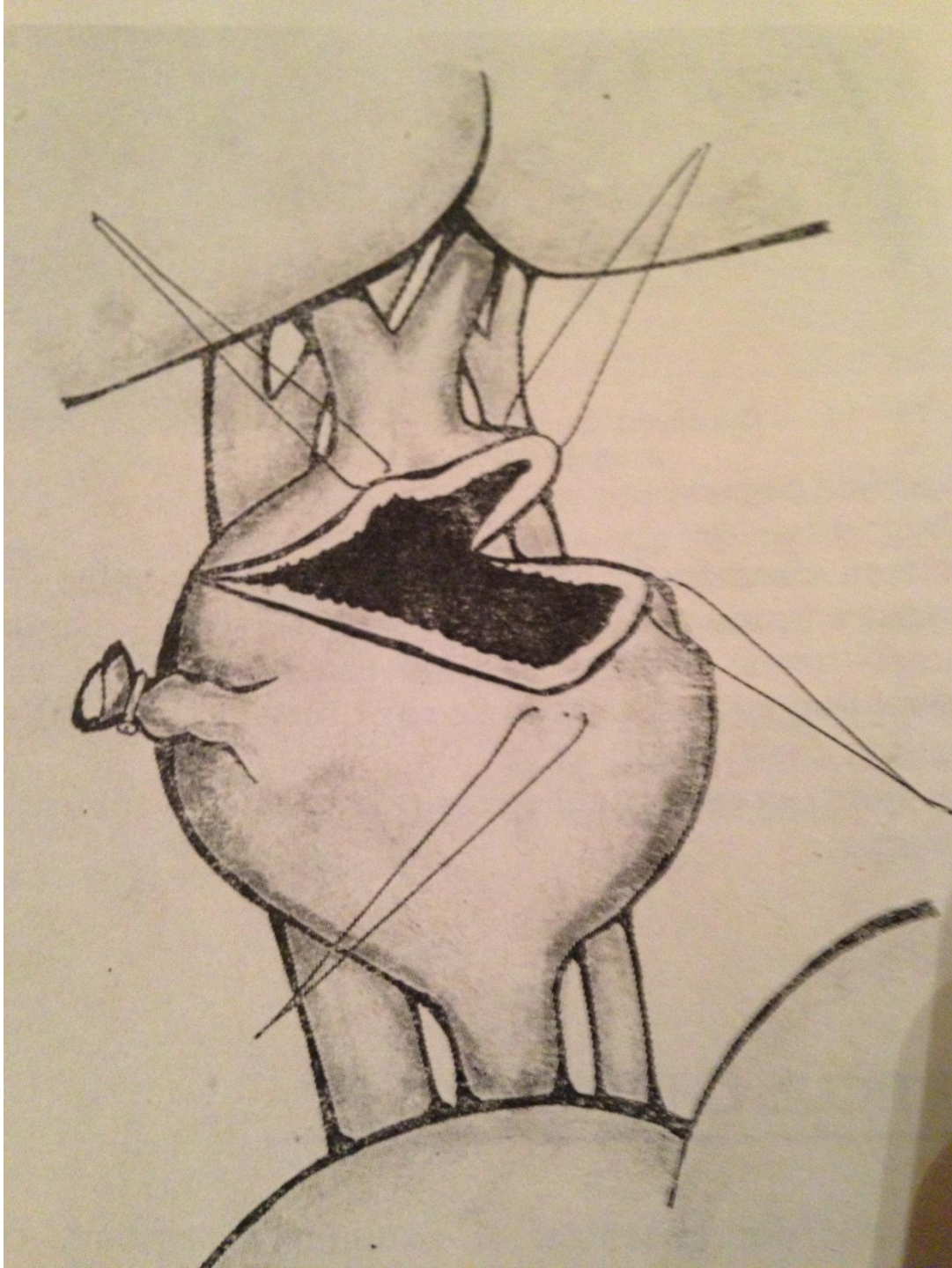




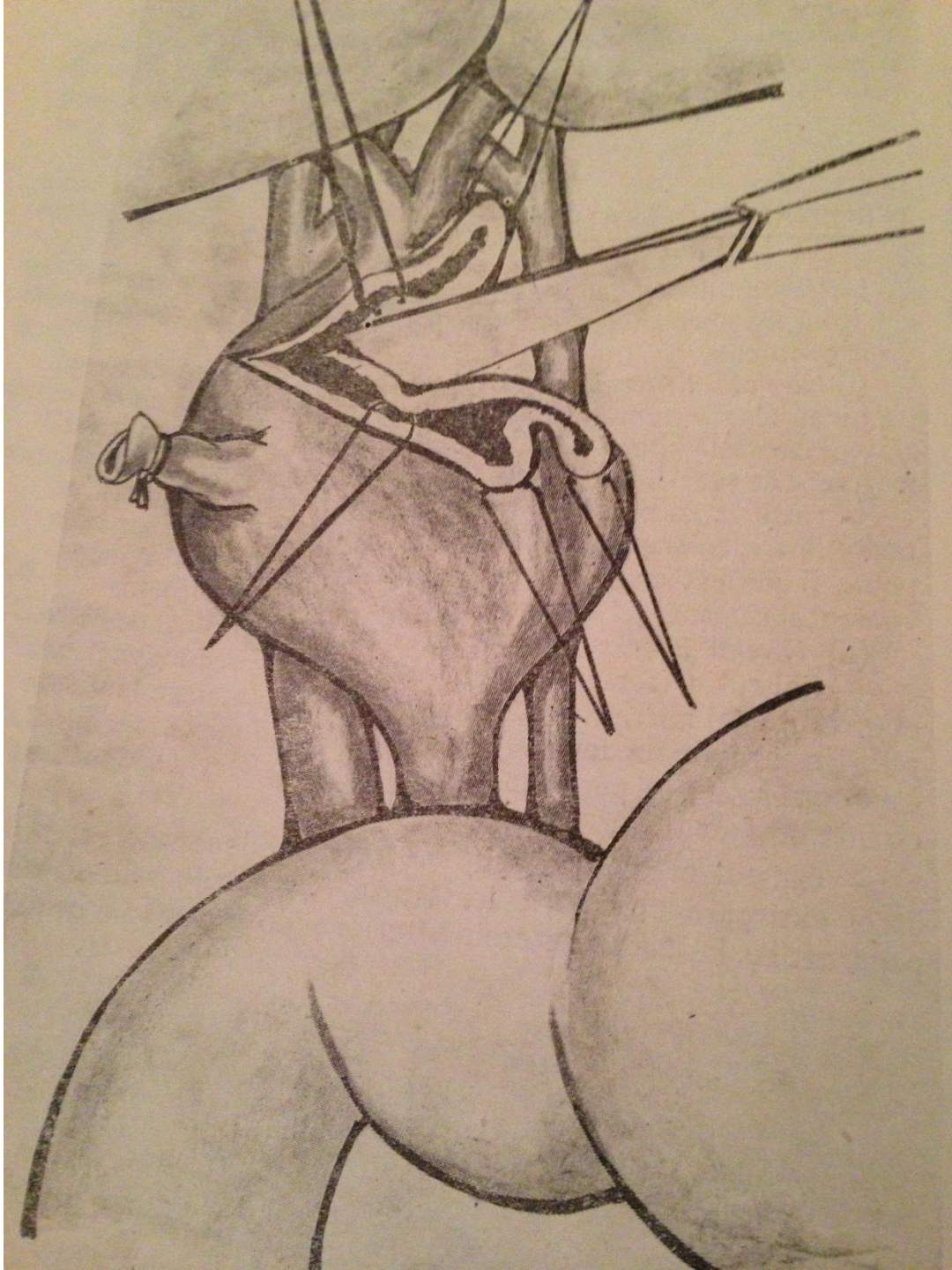


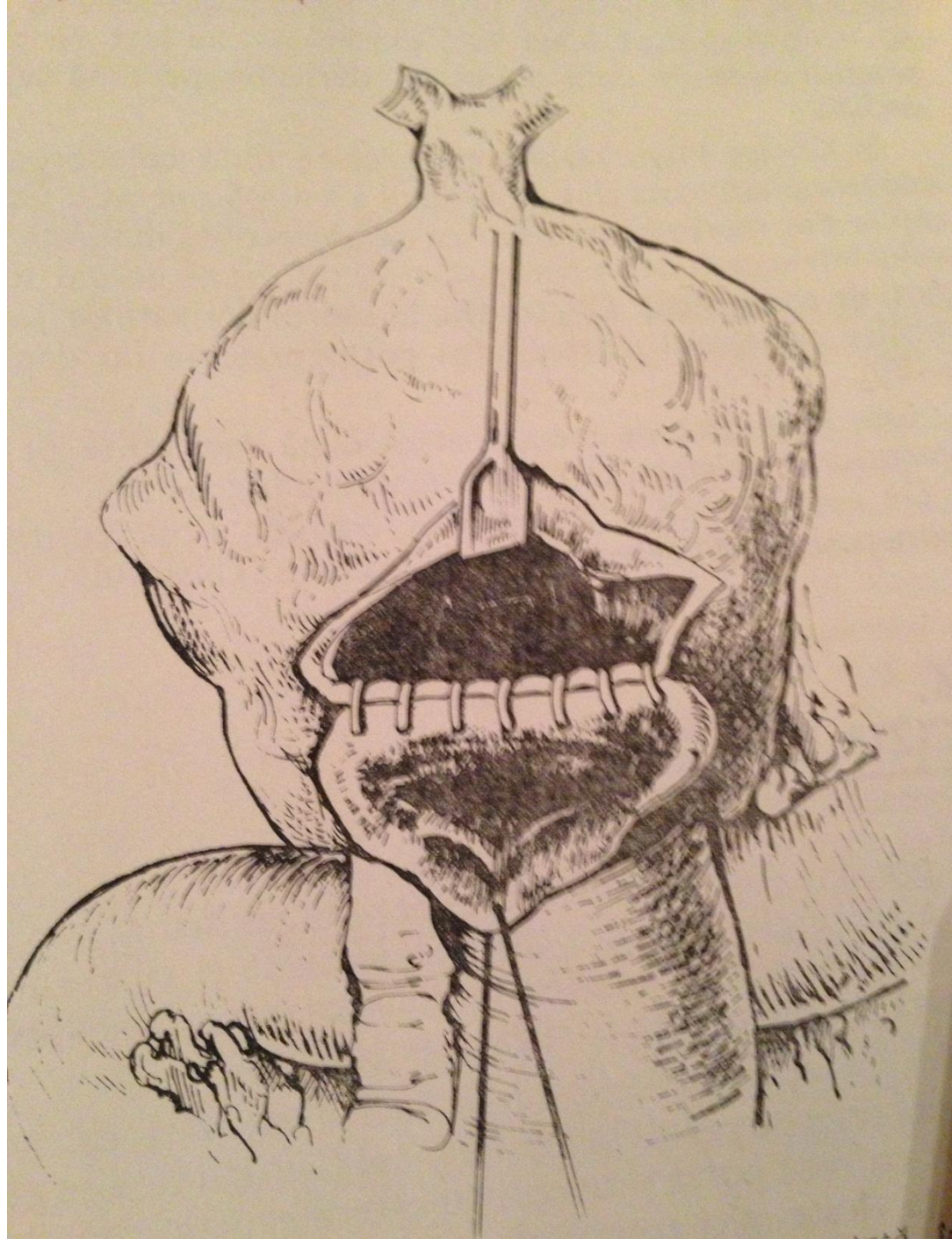






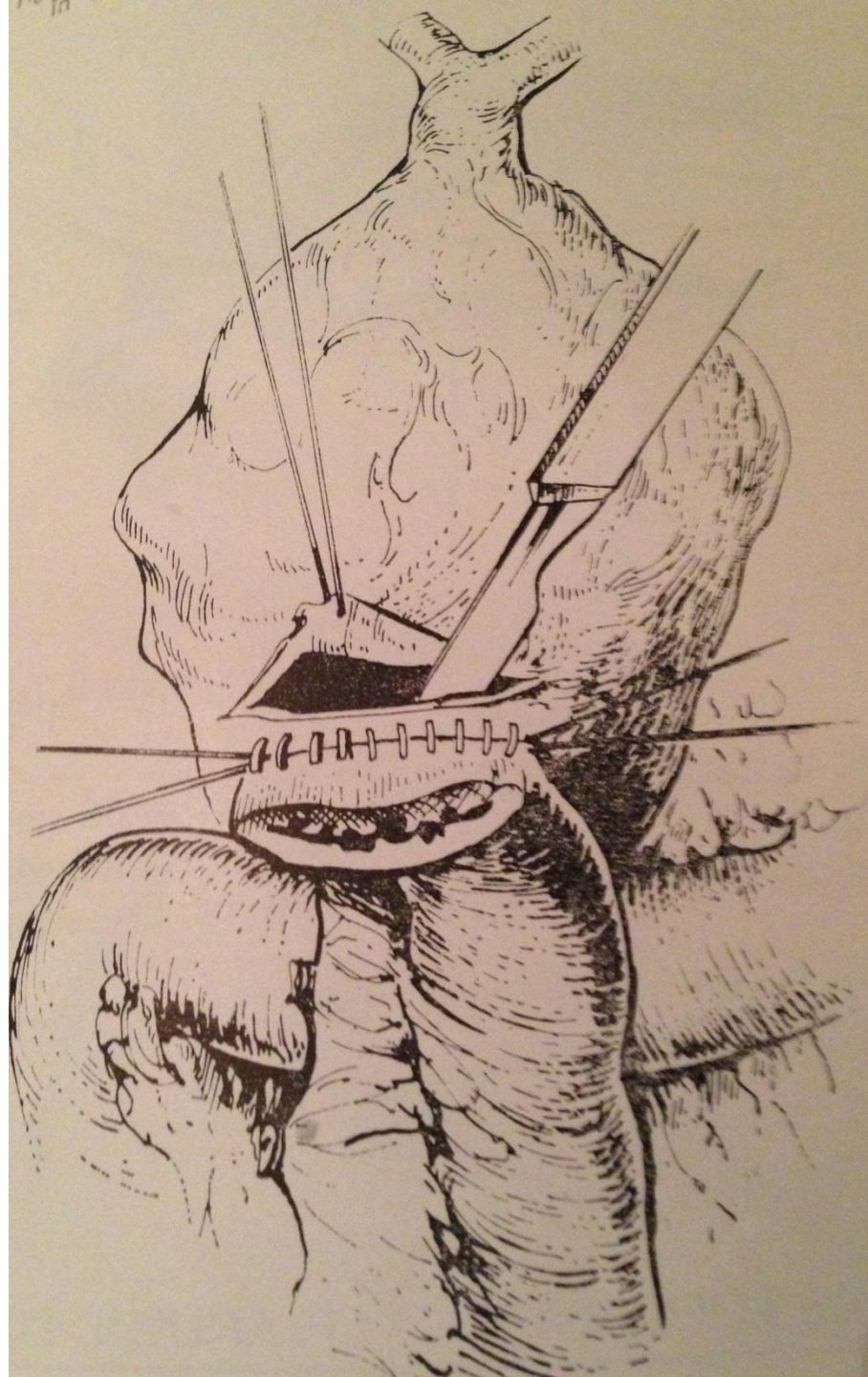


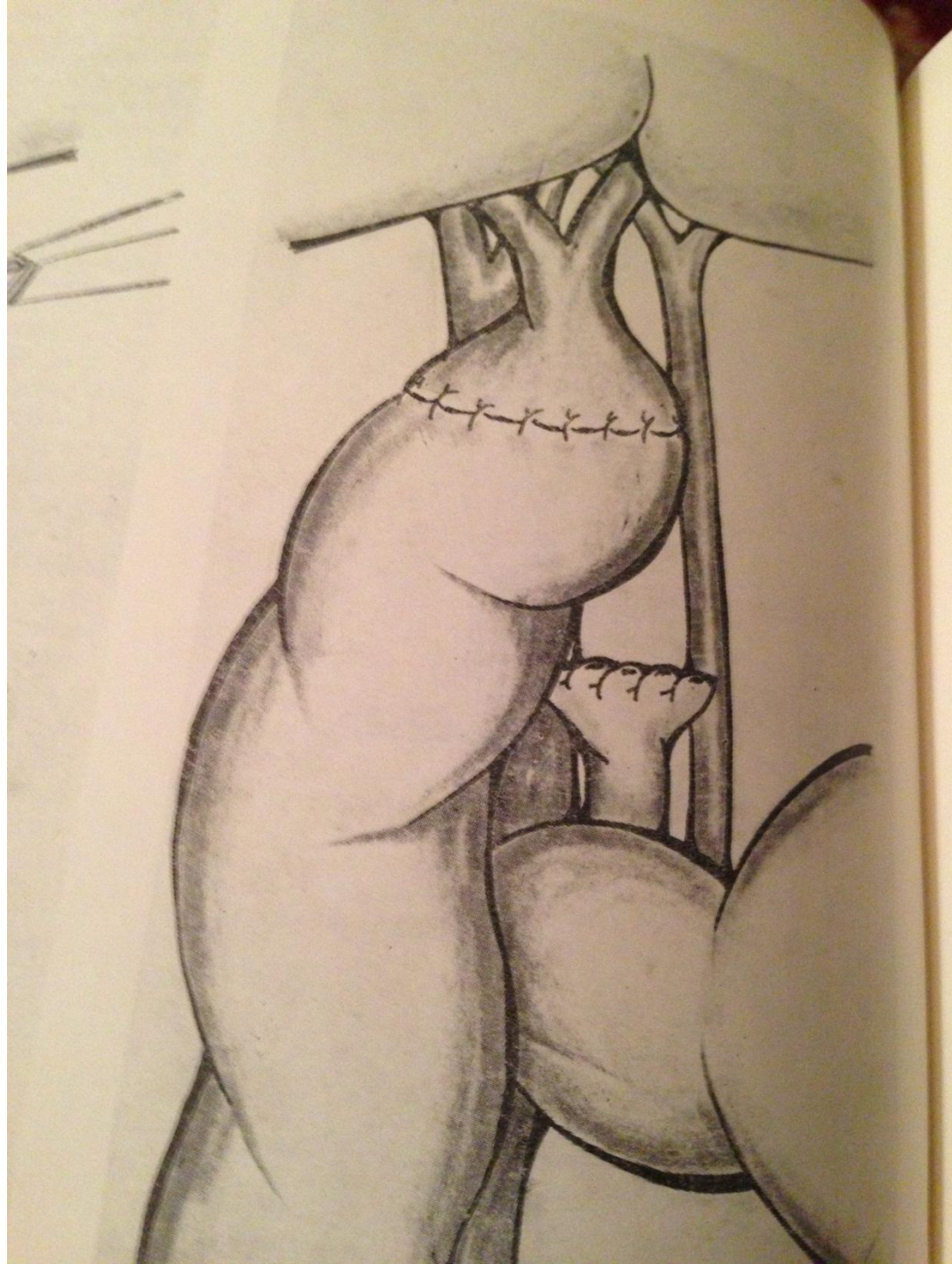




... de ansă montată









ectomy







# **Causes of obstructive jaundice:**

## **Benign:**

- Common bile duct stone
- Biliary stricture
- Choledochal cyst
- Primary sclerosing cholangitis
- Mirizzi's syndrome
- Stenosis of papilla
- Extra-hepatic biliary atresia

# Causes of obstructive jaundice

## Malignant

### 1) Primary

- Klatskin tumor
- Cholangiocarcinoma
- Carcinoma of the head of the pancreas
- Carcinoma of the duodenum
- Carcinoma of the ampulla of Vater
- Carcinoma of the gall bladder

### 2) Secondary- lymph nodes at the porta.

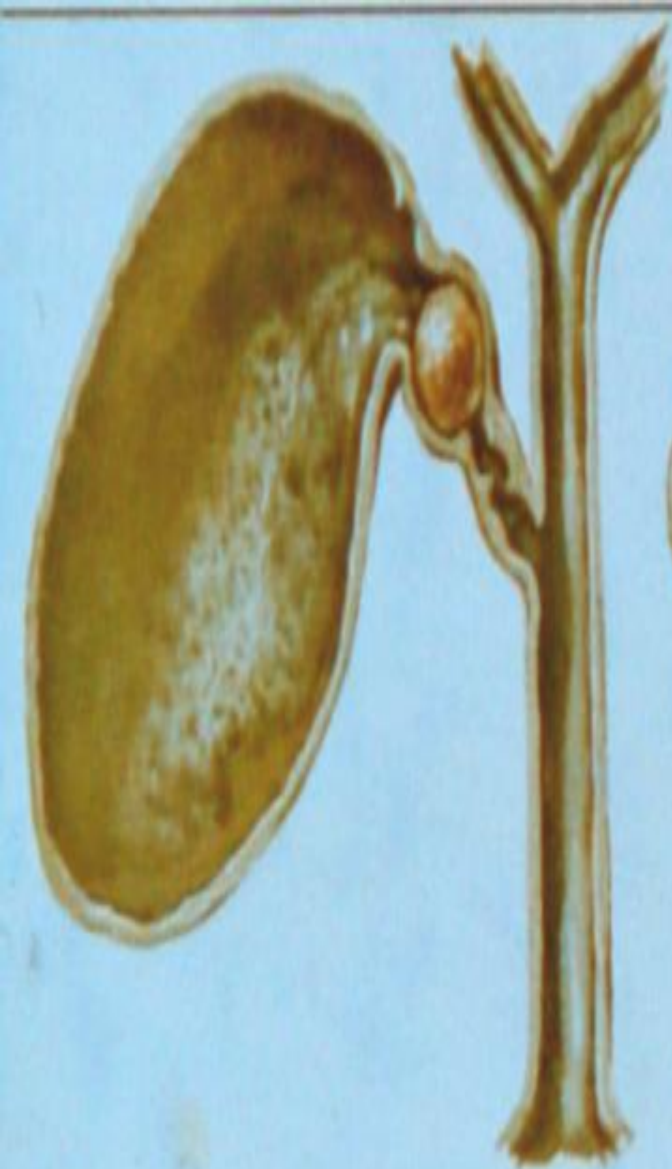
**Amongst these the commonest causes include CBD stones and peri-ampullary malignancies.**

**Obstructive jaundice** is a condition in which there is blockage of the flow of bile out of the liver. This results in an overflow of bile and its by-products into the blood, and bile excretion from the body is incomplete. Bilirubin is yellow, and this gives the characteristic yellow appearance of jaundice in the skin, eyes, and mucous membranes.

**Symptoms of obstructive jaundice:**

- yellow eyes and skin,
- abdominal pain,
- Fever
- Dark urine
- Pale stools
- Itching
- Anorexia
- nausea





CYSTIC DUCT OBSTRUCTION:  
HYDROPS



COMMON DUCT OBSTRUCTION:  
JAUNDICE



TERMINAL CYSTIC DUCT OBSTRUCTION:  
HYDROPS AND JAUNDICE



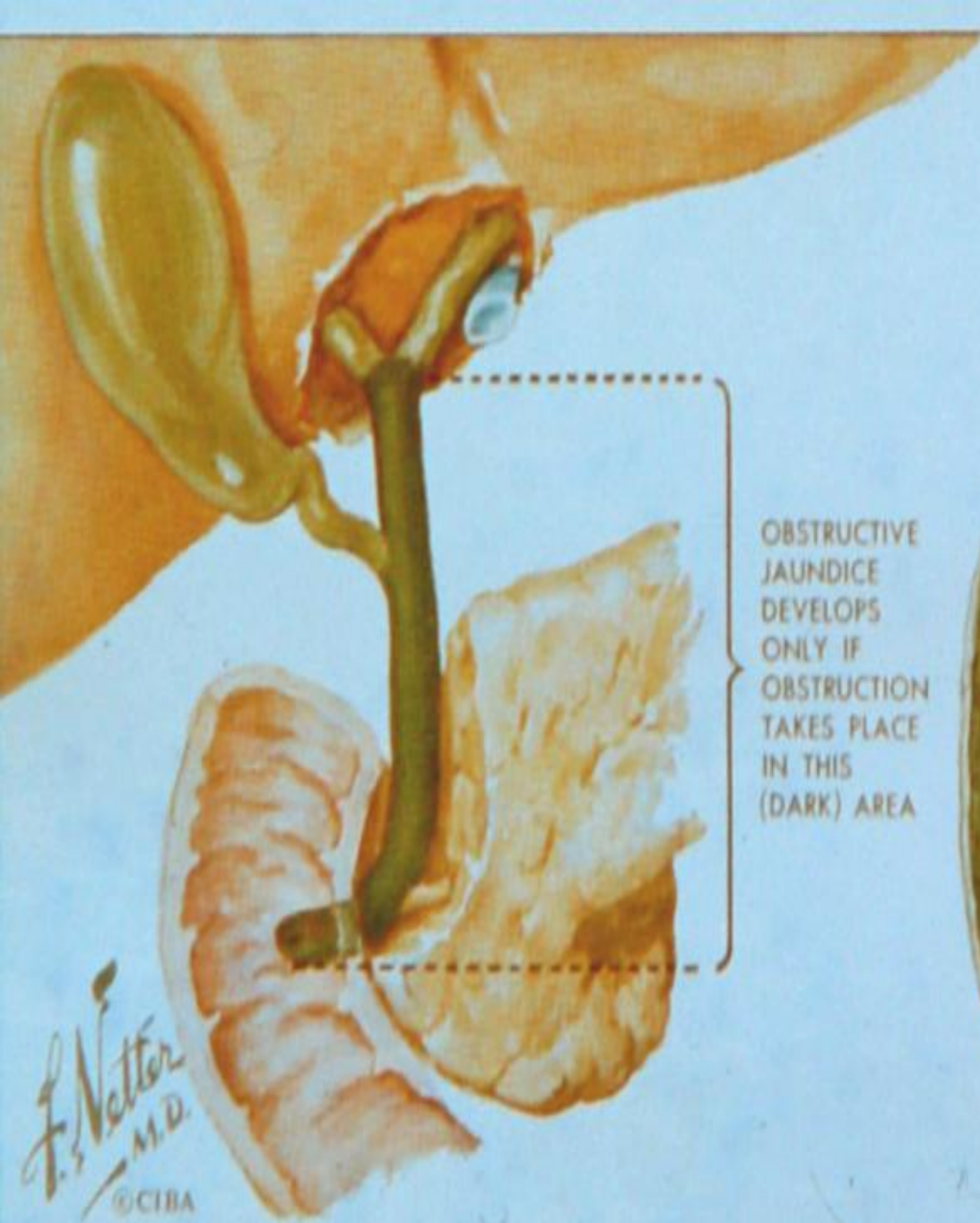
## **Clinical criterion's:**

- In calculus disease of biliary tract, intermittent or fluctuating jaundice can be seen.
- It is progressive and painless and is associated with weight loss and anorexia in malignant obstruction.
- In cases of infra-cystic obstruction due to malignant neoplasms, gall bladder may be palpable while in calculus diseases it is not palpable as it gets shrunken because of previous attacks of cholecystitis.

This is **Courvoisier's sign**.

## **Charcot triad:**

- constant severe right upper quadrant pain,
- obstructive jaundice,
- high swinging fevers



Most commonly, gallstones create the blockage.

Other causes of obstruction include:

- inflammation,
- tumors,
- trauma,
- pancreatic cancer.

The signs and symptoms of obstructive jaundice differ depending on the completeness of the blockage. Complete blockage may also occur, posing a risk of infection leading to liver and gallbladder damage.

Fortunately, in most cases, obstructive jaundice can be treated with:

- intravenous fluids,
- antibiotics, and
- **surgical removal of the obstruction.**



COMPLETE OBSTRUCTION

INCOMPLETE OBSTRUCTION



EXTRINSIC CANCER  
FIXING AND  
COMPRESSING DUCT

INTRINSIC  
CANCER

IMPACTED STONE  
WITH EDEMA

BALL-VALVE  
STONE

STRICTURE



## **Complications of obstructive jaundice include:**

- Ascending cholangitis
- Acute pancreatitis
- Cirrhosis (scarring of the liver)
- Coagulopathy (a blood clotting disorder)
- Liver failure
- Malabsorption syndrome (a disorder that interferes with the absorption of nutrients from food)
- Spread of cancer
- Spread of infection

# Diagnosis and investigations

## Basic tests

- Leucocytosis
- Creatinine,
- Bilirubin
- Alkaline phosphatase
- Serum amylase
- Clotting studies

## Advanced tests

- Ultrasound first line
- Magnetic resonance cholangiopancreatography (MRCP), noninvasive, best accuracy
- Endoscopic retrograde cholangiopancreatography( ERCP), risk: haemorrhage, acute pancreatitis, ascending infection, perforation-peritonitis,
- Percutaneous transhepatic cholangiography (PTC)-used for failure of ERCP, as therapeutic procedure, often in combination with ERCP, risk of: sepsis, tube movement, leakage around the tube, dehydration

**Treatment** options for obstructive jaundice **depend on the exact cause** of the jaundice and **on the severity of the disease:**

- Antibiotic therapy (if indicated for infection)
- Endoscopic retrograde cholangiopancreatography (ERCP), an imaging procedure that allows treatment of some bile duct problems, including removal of gallstones that are causing obstruction
- Intravenous fluids and pain medications
- Nutritional support
- **Surgery** or other procedures to repair anatomical defects or create alternative pathways for the flow of bile
- Transplantation of the liver (if all other methods are unsuccessful and all of the liver is damaged)
- Treatment for cancer, if present, which may include surgery, chemotherapy, or radiation therapy

## **Treatment for common bile duct stone:**

### **Emergency treatment, by**

- ERCP with extraction or stent insertion
- Occasionally PTC required

indicated in:

- ☐ unresolving gallstone pancreatitis,
- ☐ unresolving ascending cholangitis

### **Elective treatment: by:**

- ERCP or
- combined ERCP /PTC,
- CBD exploration at time of surgery,
- open CBD exploration requires a T-tube to be left in CBD

indicated for complications (pancreatitis, cholangitis, obstructive jaundice)  
for all patients with gallbladder stones due for cholecystectomy

Another possibilities:

- Common duct exploration
- Choledochoduodenostomy
- Basket extraction through T-tube site
- Endoscopic sphincteroplasty
- Choledochoscopy trough T-tube site

Obstructive jaundice which can be either due to intra-hepatic cholestasis or extra-hepatic biliary obstruction is amenable to surgical treatment



# Choledocholithotomy

finished with:

- A) **Simple closure**-It can only be attempted when one is sure about the patency of the distal duct system as evidenced by passing of dye into duodenum in a post procedure cholangiogram.
- B) **T-Tube drainage**-Most surgeons will still prefer **choledochorraphy to a T-Tube** in spite of a high incidence of cholangitis and biliary peritonitis. Hence, antibiotic prophylaxis is strongly recommended when T-Tube is in-situ.
- C) **Biliary enteric** procedures

indications for biliary-enteric anastomosis:

- Multiple primary duct stones with dilated ducts particularly in elderly
- Multiple stones in a dilated duct
- Irretrievable intrahepatic stones
- Proven papillary stenosis
- Impacted ampullary stone
- Terminal CBD stricture.

- 1) **CHOLEDOCHODUODENOSTOMY:** It is mainly indicated when CBD is more than 1.5 cm, patient is elderly and high risk. The supra-duodenal choledochoduodenostomy uses vertical CBD and horizontal duodenal incision.
- 2) **TRANSDUODENAL SPHINCTEROPLASTY:** It is indicated in a patient of impacted stone in ampulla or a very low CBD stricture (duct is dilated down to terminal few mms). Though an advantage to examine ampulla directly is got, disadvantages are too many. The complications include pancreatitis and duodenal leak.
- 3) **CHOLEDOCHOJEJUNOSTOMY:** This is done when CBD can be mobilized completely. A roux-en-y loop or a simple loop is used with an entero-enterostomy below. Its advantages are low incidence of cholangitis and reflux of indigestible intestinal contents into the biliary tree. The drawback is that there is increased incidence of post-operative peptic ulcer disease.

# **LAPAROSCOPIC APPROACH**

## **Laparoscopic cholecystectomy and trans-cystic CBD exploration.**

After cholecystectomy, cystic duct can be dilated laparoscopically and stones can be retrieved using baskets.

## **Laparoscopic choledochotomy**

It is an approach in patients with dilated CBD, calculi 1 cm or more in diameter, multiple calculi or those who require lithotripsy for impacted stones.



# BILIARY DUCTS

## ERCP with Endoscopic Sphincterotomy (ES)

*Reasons for performing the procedure:*

- Before gallbladder surgeries, when there is a strong suspicion that common bile duct stones are present.
- At the end of a cholecystectomy, if the surgeon detects stones in the common bile duct (only if there are experts in ERCP present, and equipment is available).
- For patients with gallstone cholangitis (serious infection in the common bile duct). In such cases urgent ERCP and antibiotics are required.

### **Technique:**

The patient is given a sedative and asked to lie on his or her left side.

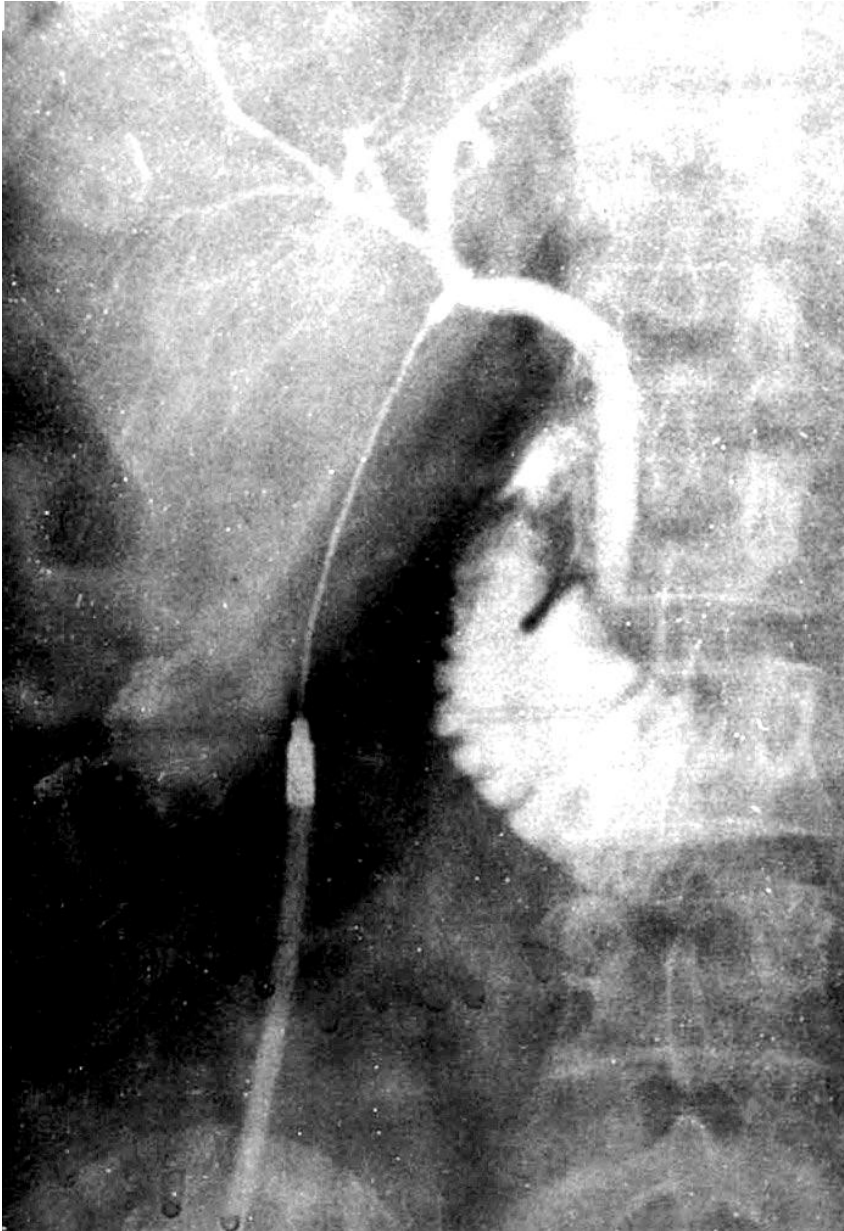
An endoscope (a tube containing fiber optics connected to a camera) is passed through the mouth and stomach and into the duodenum until it reaches the point where the common bile duct enters. This does not interfere with breathing, but the patient may have a sensation of bloating.

A thin catheter is then passed through the endoscope.

Contrast material is injected through the catheter into the opening of the duct for allows x-ray visualization of the biliary tree and any stones contained in the area.

Instruments may also be passed through the endoscope to remove any stones that are detected.

# BILIARY DUCTS



# BILIARY DUCTS

The next phase of the procedure is known as **endoscopic sphincterotomy** (ES). (It is also sometimes referred to as papillotomy, although this is a slightly different variation.)

ES widens the junction between the common bile duct and intestine (the ampulla of Vater) so that the stones can be extracted more easily.

With ES, a tiny incision is usually made in the opening of the common bile duct and through the muscles that enclose the lower common bile duct (the sphincter of Oddi).

**Malignant tumors** can develop anywhere along the extrahepatic bile ducts. Of these tumors 70-80% involve the confluence of the right and left hepatic ducts (hilar carcinomas) and about 20-30% arise more distally. Diffuse involvement of the ducts is rare, occurring in only about 2% of cases.

All malignant tumors of the extrahepatic bile ducts inevitably cause partial or complete ductal obstruction. Because the bile ducts have a small diameter, the signs and symptoms of obstruction usually occur while tumors are relatively small. Because of their invasion of major vascular structures and direct extension to the liver, hilar carcinomas are more difficult to resect than those that arise distally and are associated with a worse prognosis.

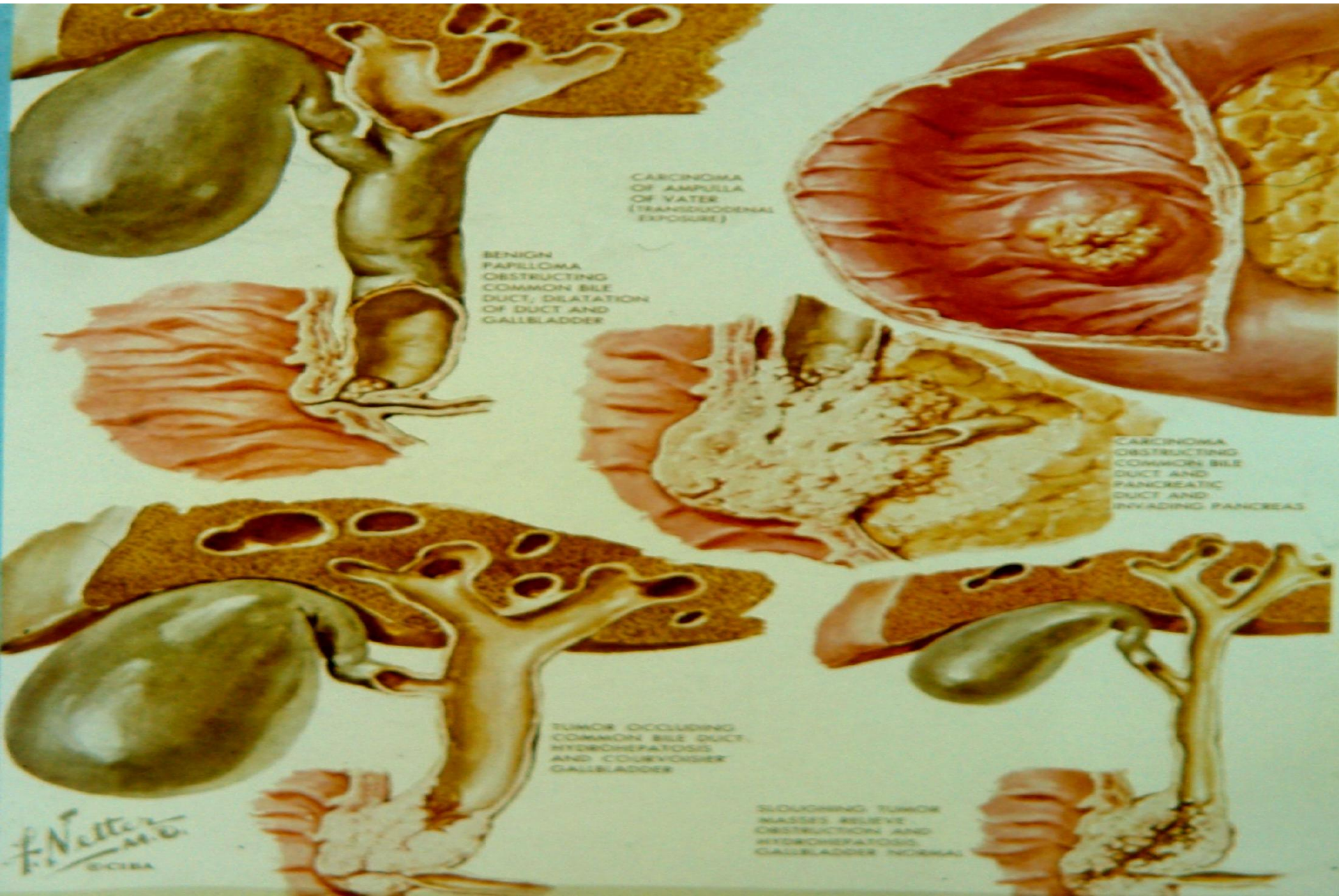
## **Regional lymph nodes**

Accurate tumor staging requires that all lymph nodes that are removed be analyzed. Optimal histologic examination of a regional lymphadenectomy specimen should include analysis of a minimum of **three lymph nodes**.

The regional lymph nodes are the same as those listed for gallbladder cancer and include the following: hilar, celiac, periduodenal, peripancreatic and superior mesenteric. The hilar nodes include the lymph nodes along the common bile duct, hepatic artery, portal vein and cystic duct.



# BILIARY DUCTS



# **Cholangiocarcinoma**

**Most bile duct tumors are **adenocarcinomas** and more than half involve the hepatic bifurcation (**Klatskin tumor**).**

**Despite a trend toward a more aggressive surgical approach to these tumors, the prognosis remain dismal.**

**Death generally results from biliary obstruction, liver failure or cholangitis, rather than from metastatic disease.**

## **Clinical findings:**

- Obstructive progressive jaundice
- Epigastric pain
- Significant weight loss
- Fatigue
- Cholangitis

## **Differential diagnosis**

- Common duct stones
- Stricture
- Sclerosing cholangitis
- Benign tumor
- Pancreatic cancer
- Metastatic cancer

## **Laboratory**

- Elevated bilirubin and alkaline phosphatase
- PTC
- ERCP

## **Treatment**

- Biliary enteric by-pass
- Operatively placed stents (U-tube)
- Whipple procedure for distal tumors

## **Hilar cholangiocarcinoma (Klatskin tumor)**

In most series, the overall resectability rate for hilar bile duct cancer averages between 40 and 60 %.The addition of major liver resection to surgery for hilar bile duct cancer did not show any survival advantage. Reconstruction is done by hepaticojejunostomy.

Palliative procedures include trans-tubal drainage using U tube and biliary enteric anastomosis.

PTC is usually necessary to delineate the duct system. Segment III is commonly used for bypass. The duct is approached by ligamentum teres approach, and is anastomosed side to side to a Roux-en-Y loop of jejunum.

## **Cholangiocarcinoma of the distal CBD**

This has been grouped under periampullary tumors and requires pancreatico-duodenectomy.



# Metastatic sites

Extrahepatic bile duct carcinomas can extend to the:

- liver,
- pancreas,
- ampulla of Vater,
- duodenum,
- great omentum,
- stomach or
- gallbladder.

Tumors arising in the right or left hepatic ducts usually extend proximally into the liver or distally to the common hepatic duct.

Neoplasms from the cystic duct invade the gallbladder, common bile duct or both.

Carcinomas that arise in the distal segment of the common bile duct can spread to the pancreas, duodenum, stomach, colon or omentum

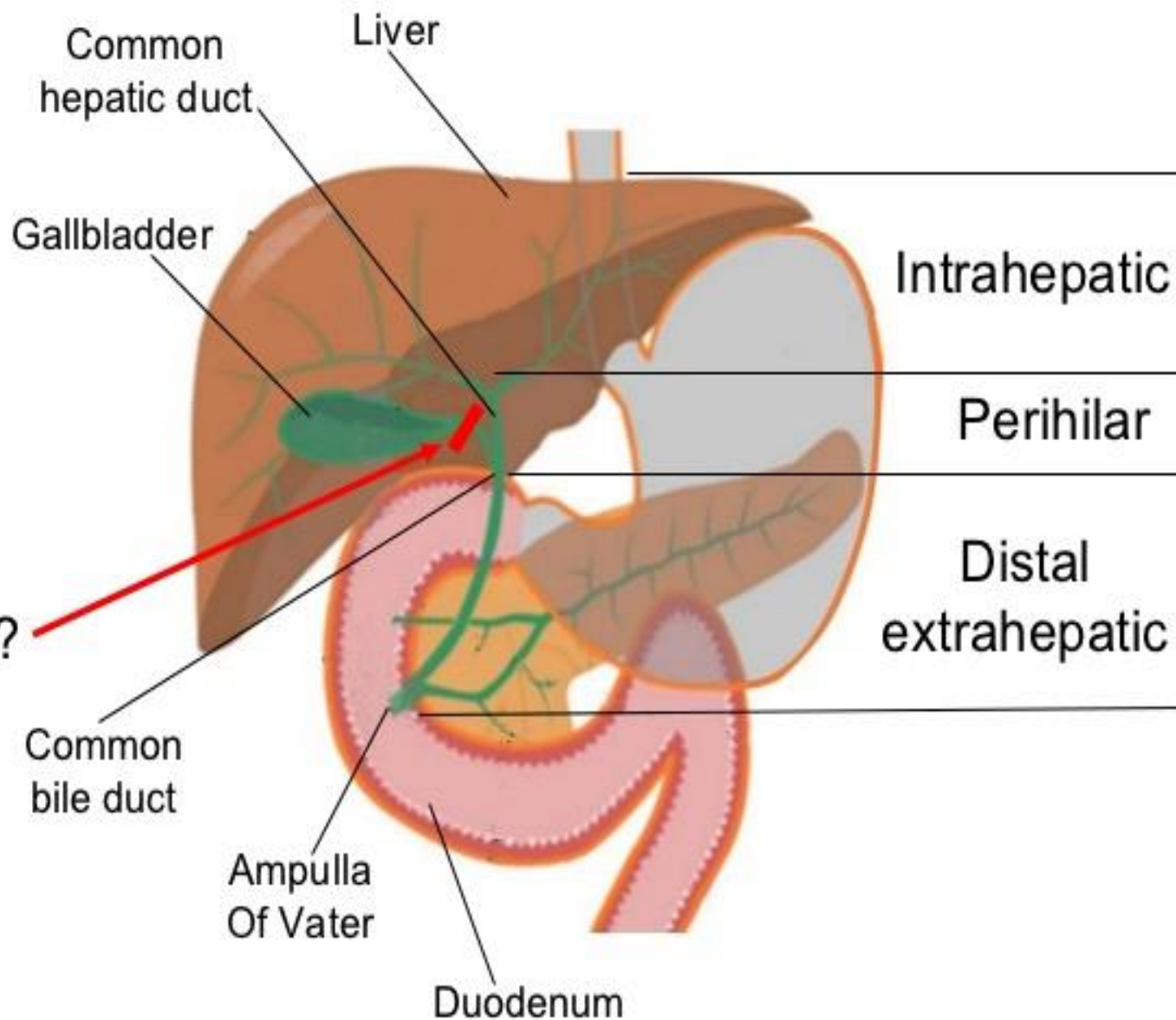
**Distal metastases** usually occur late in the course of the disease and are most often found in the **liver, lungs and peritoneum**.

# Ampullary tumors

The **ampulla of Vater** is a small dilated duct less than 1.5 cm long, formed in most individuals by the union of the terminal segments of the pancreatic and common bile ducts.

The ampulla opens into the duodenum, usually on the posterior-medial wall, through a small mucosal elevation, the duodenal papilla, which is also called the ampulla of Vater. Nearly all cancers that arise in this area are well-differentiated adenocarcinomas.

Does  
obstruction  
of the cystic  
duct  
or gallbladder  
cause jaundice?



# Ampullary tumors

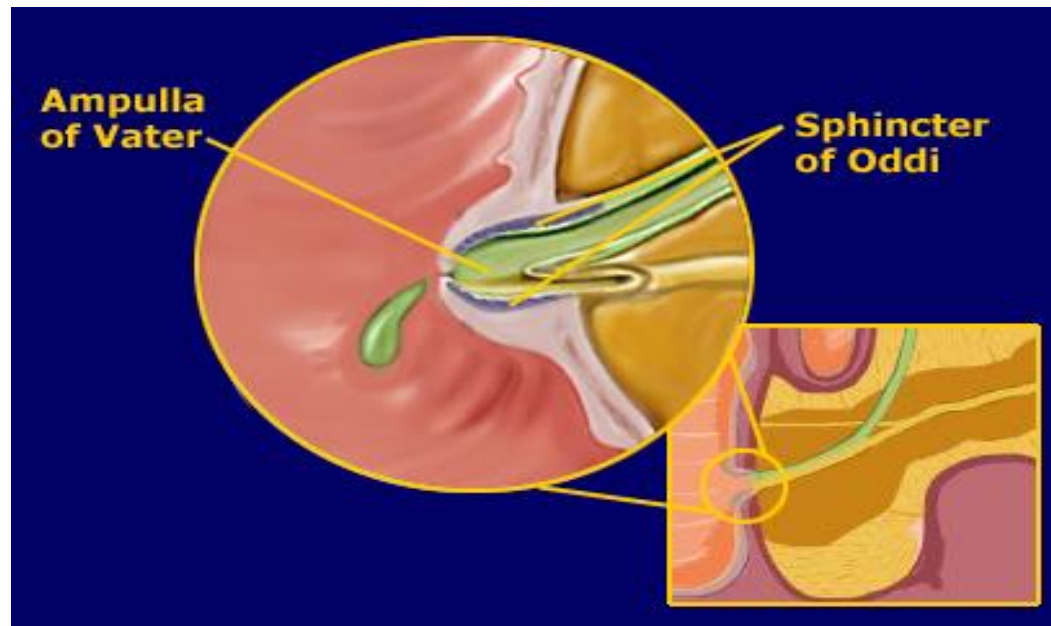
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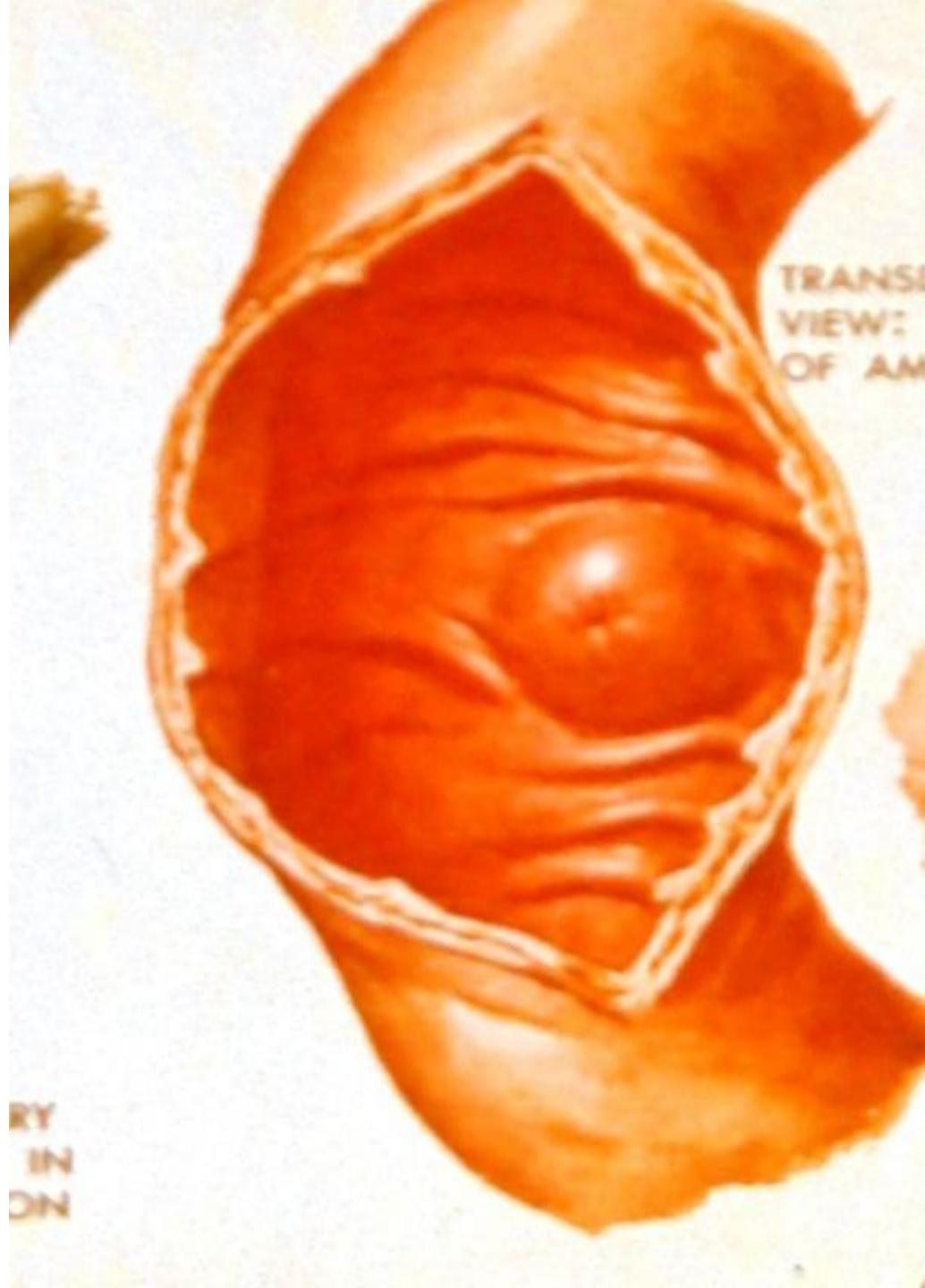
Clinically and pathologically, carcinomas of the ampulla may be difficult to differentiate from those arising in the head of the pancreas or in the distal segment of the common bile duct.

Tumors of the ampulla must be differentiated from those arising in the second part of the duodenum and invading the ampulla, which are staged as a small bowel tumors. Carcinomas of the ampulla and periampullary region are often associated with the familial

**adenomatous polypsis syndrome.**







TRANSI  
VIEW:  
OF AM

RY  
IN  
ON

# **Ampullary tumors**

## **Metastatic sites**

Tumors of the ampulla may infiltrate adjacent structures, such as the wall of the duodenum, the head of the pancreas, and extrahepatic bile ducts. Metastatic disease is most commonly found in the liver and peritoneum and is less commonly seen in the lungs and pleura.

# **Ampullary tumors**

## **Surgery**

**Local excision is suitable treatment for certain small tumors (less than 2 cm) arising from the ampulla of Vater or in the pancreatic or biliary duct within 2 cm of the ampulla, if there is suspicion of malignancy frozen section evaluation of the resected specimen is done and if it confirms malignancy pancreatico-duodenectomy should be done.**

**After local resection of ampullary tumors, follow up endoscopy is performed 6 to 12 months postoperatively to assess local recurrence. Patient who develop pancreatitis or jaundice months to years following local resection of ampullary tumors must be suspected of having local recurrence and should undergo repeat endoscopy with ERCP.**

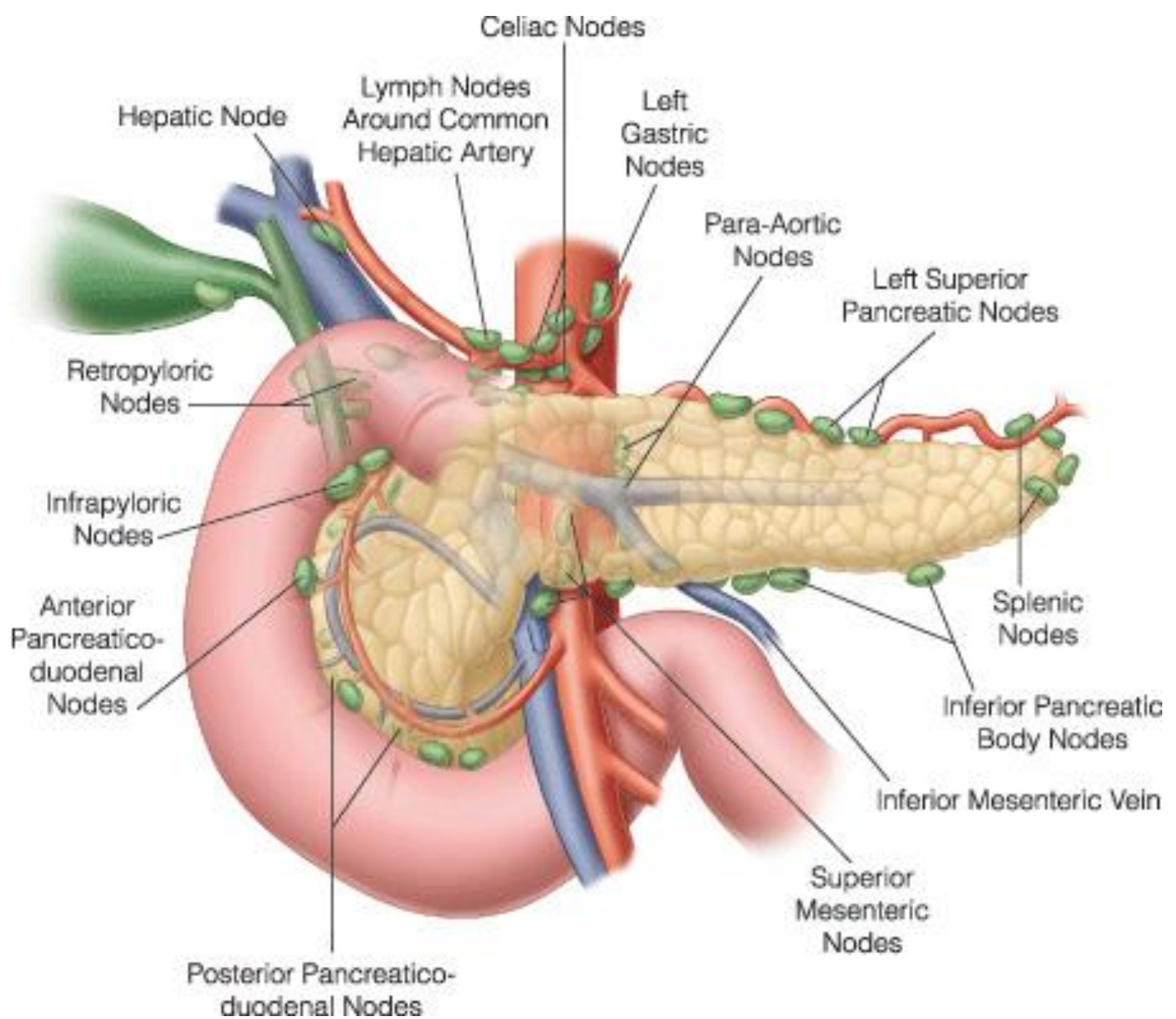


## **Regional lymph nodes**

A rich lymphatic network surrounds the pancreas and periampullary region and accurate tumor staging requires that all lymph nodes that are removed be analyzed.

Optimal histologic examination of a pancreaticoduodenectomy specimen should include analysis of a minimum of 10 lymph nodes.

The regional lymph nodes are peripancreatic lymph nodes, which also include the lymph nodes along the hepatic artery, celiac axis and pyloric regions.



# Nonpancreatic periampullary tumors

These are **carcinoma of the papilla Vater, primary bile duct carcinoma and primary duodenal carcinoma**. Characteristics include a much slower rate of growth and a tendency to remain localized.

Almost all patients have extrahepatic cholestasis, upper gastrointestinal bleeding and anemia occurring in these patients. The alcoholic nature of obstructive jaundice with upper gastrointestinal bleeding may lead to a silver grey color stool. Stools test positive for occult blood.

The diagnosis of ampullary or duodenal carcinoma may be made by endoscopy and imaging techniques.

Whipple's procedure is indicated as the curative resection, accompanied by cholecystectomy and truncal vagotomy as performed for pancreatic carcinoma.

In patients with metastases, palliative surgery should be considered.

## **BILIARY STRICTURE**

Variety of forms ranging from the stenosing process to complete surgical absence of most of the extrahepatic duct system.

A stricture becomes clinically evident when bile is prevented from entering the gastrointestinal tract from causes other than calculus, resulting in one or more of the symptoms of jaundice, cholangitis, external biliary drainage, or pain.

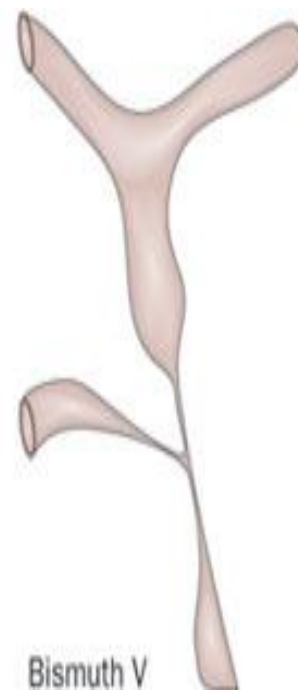
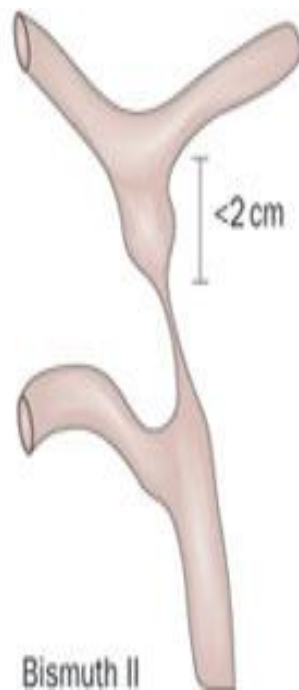
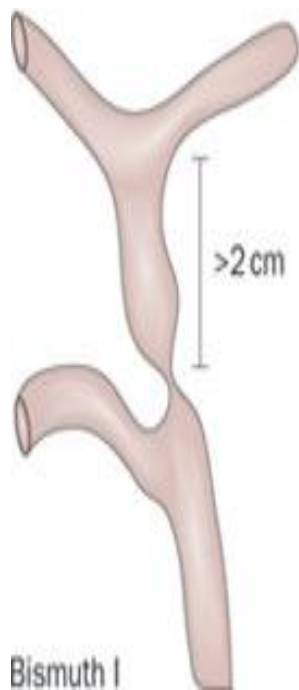
**Ethiology:** congenital, inflammatory, neoplastic, traumatic or surgical injury.

### **Treatment**

- External biliary fistula, partial external biliary fistula with T-tube,
- internal biliary fistula : choledocoduodenostomy, choledocojejunostomy.

Effective prophylaxis is surely far superior to the best reconstructive procedure.





Type I strictures, with a common duct stump longer than 2 cm, can be repaired without opening the left duct and without lowering the hilar plate.

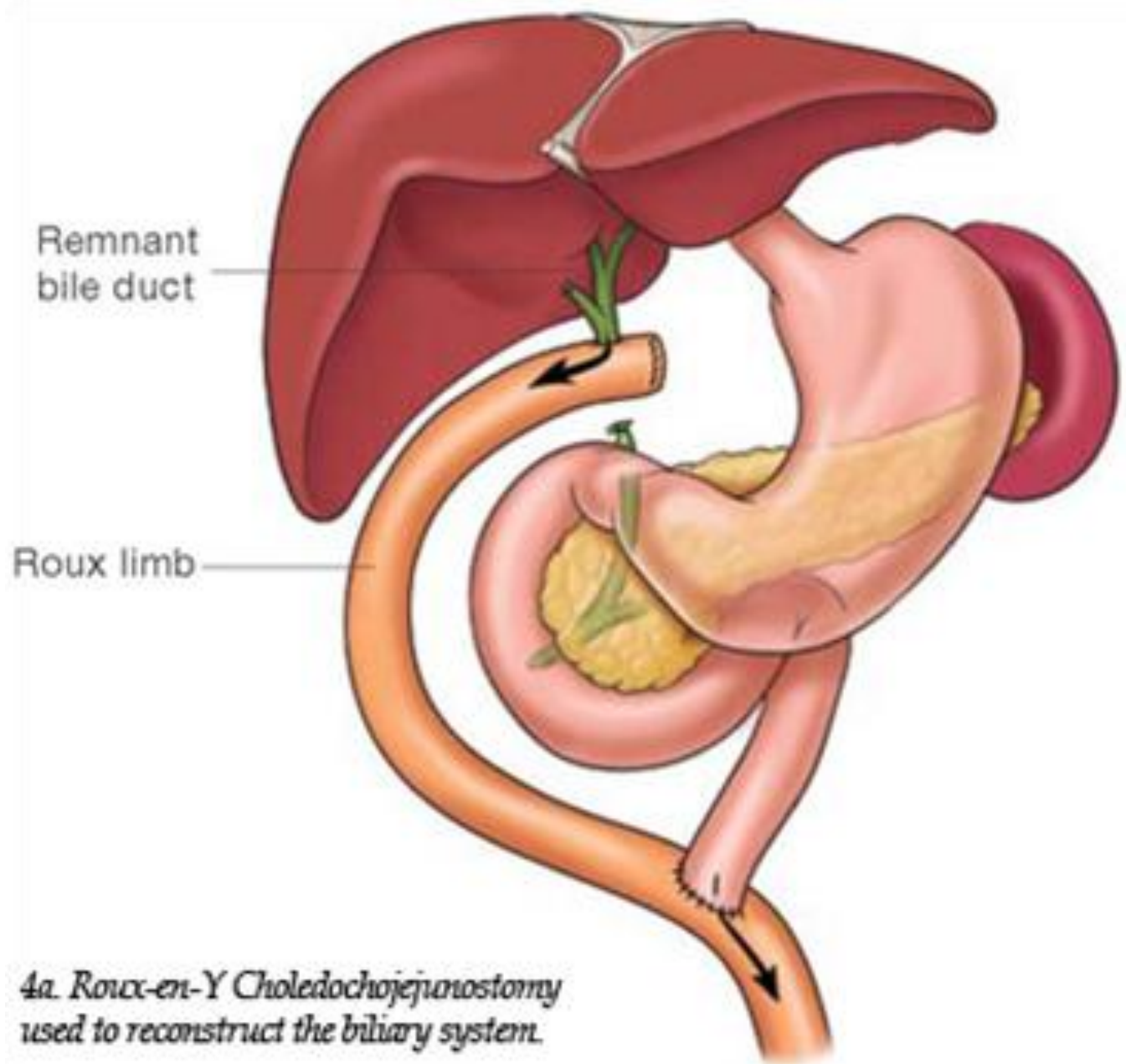
Type II strictures, with a stump shorter than 2 cm, require opening the left duct for a satisfactory anastomosis. Lowering the hilar plate is not always necessary but may improve the exposure.

Type III lesions, in which only the ceiling of the biliary confluence is intact, require lowering the hilar plate and anastomosis on the left ductal system. There is no need to open the right duct if the communication between the ducts is wide.

With type IV lesions the biliary confluence is interrupted and requires either reconstruction or two or more anastomoses.

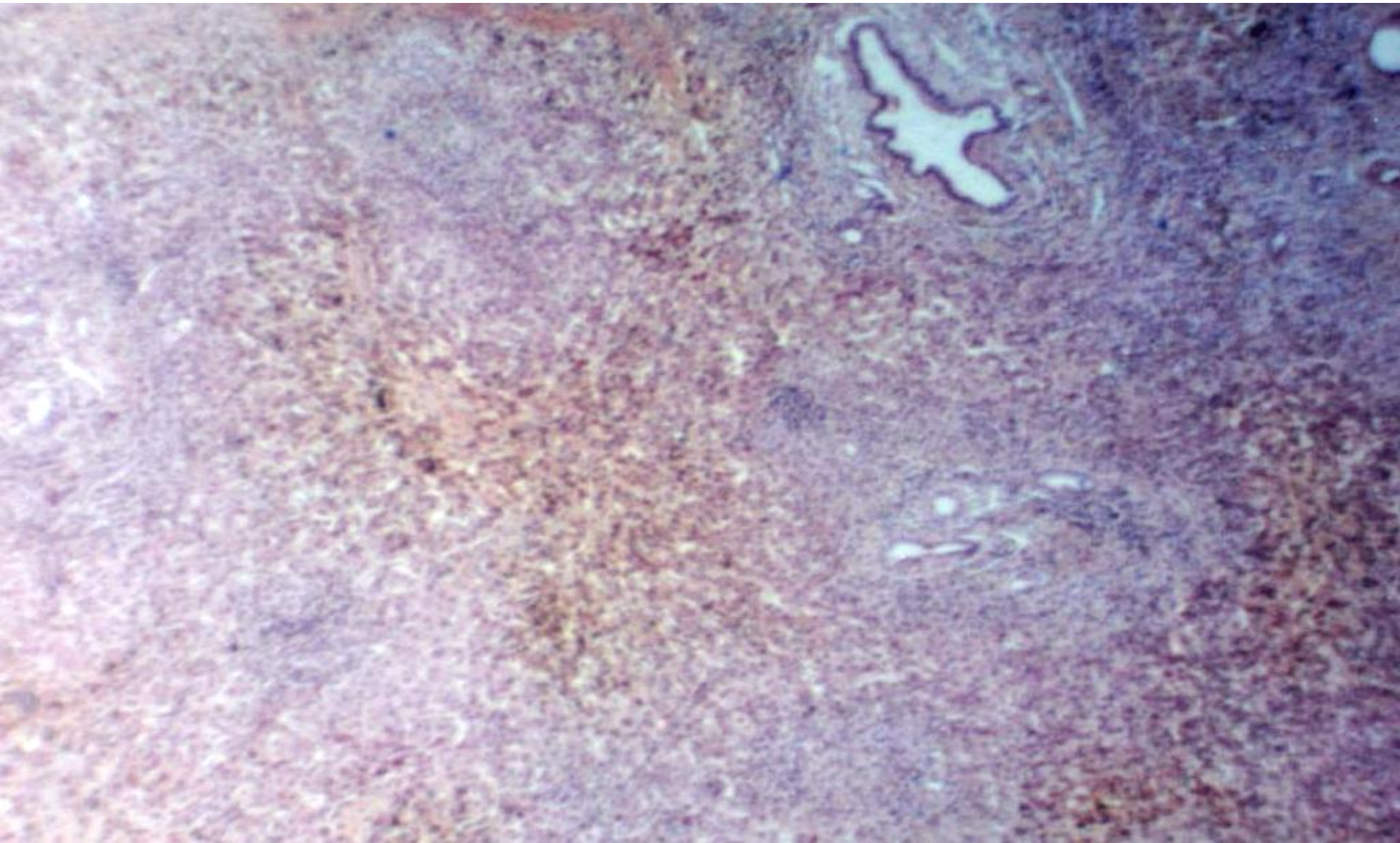
Type V lesions are strictures of the hepatic duct associated with a stricture on a separate right branch, and the branch must be included in the repair. Although this classification is intended for established strictures, it is commonly used to describe acute bile duct injuries.

The surgeon must be aware, however, that the established stricture is generally one level higher than the level of the injury at the original operation.



# BILIARY DUCTS

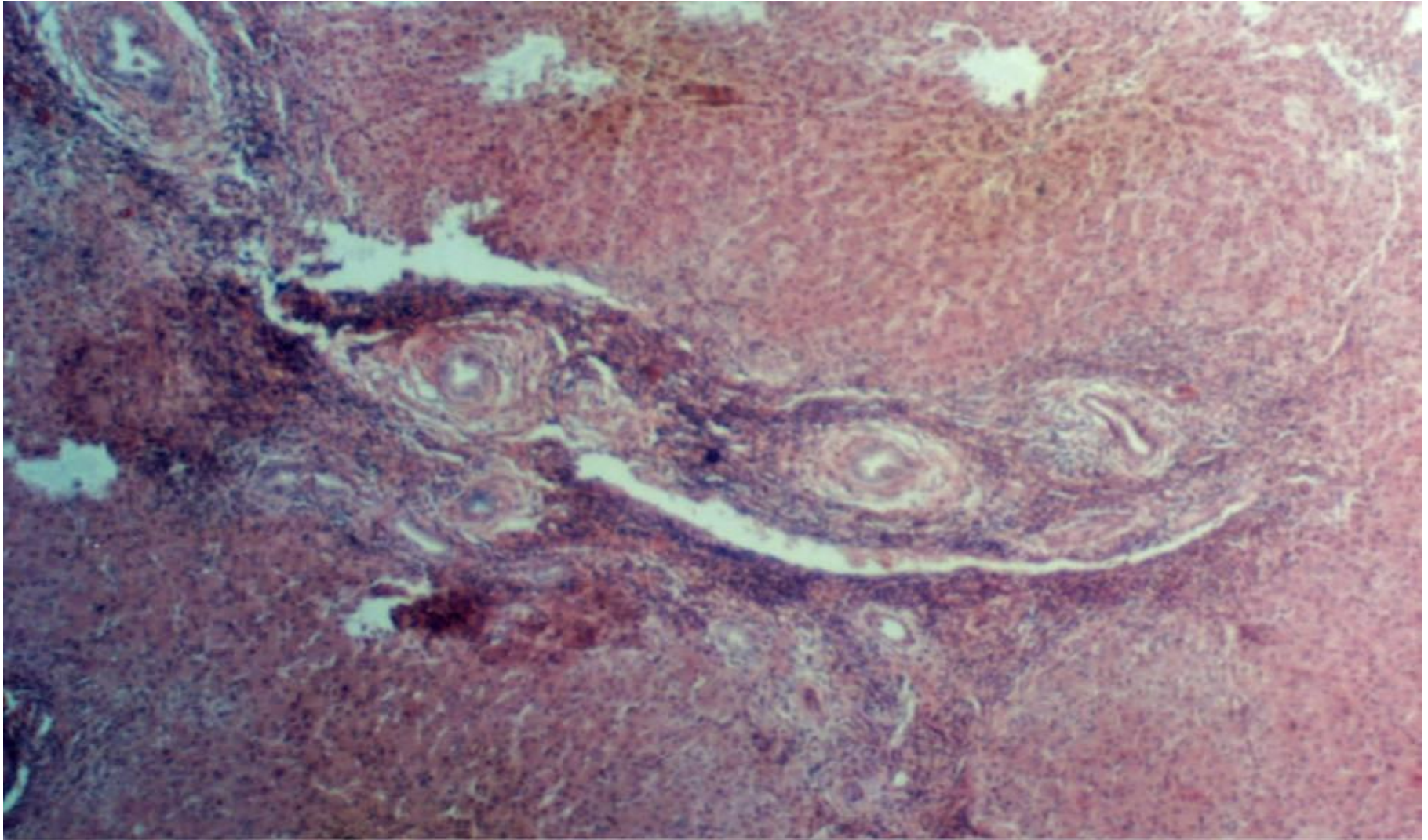
**intrahepatic cholestasis**





# BILIARY DUCTS

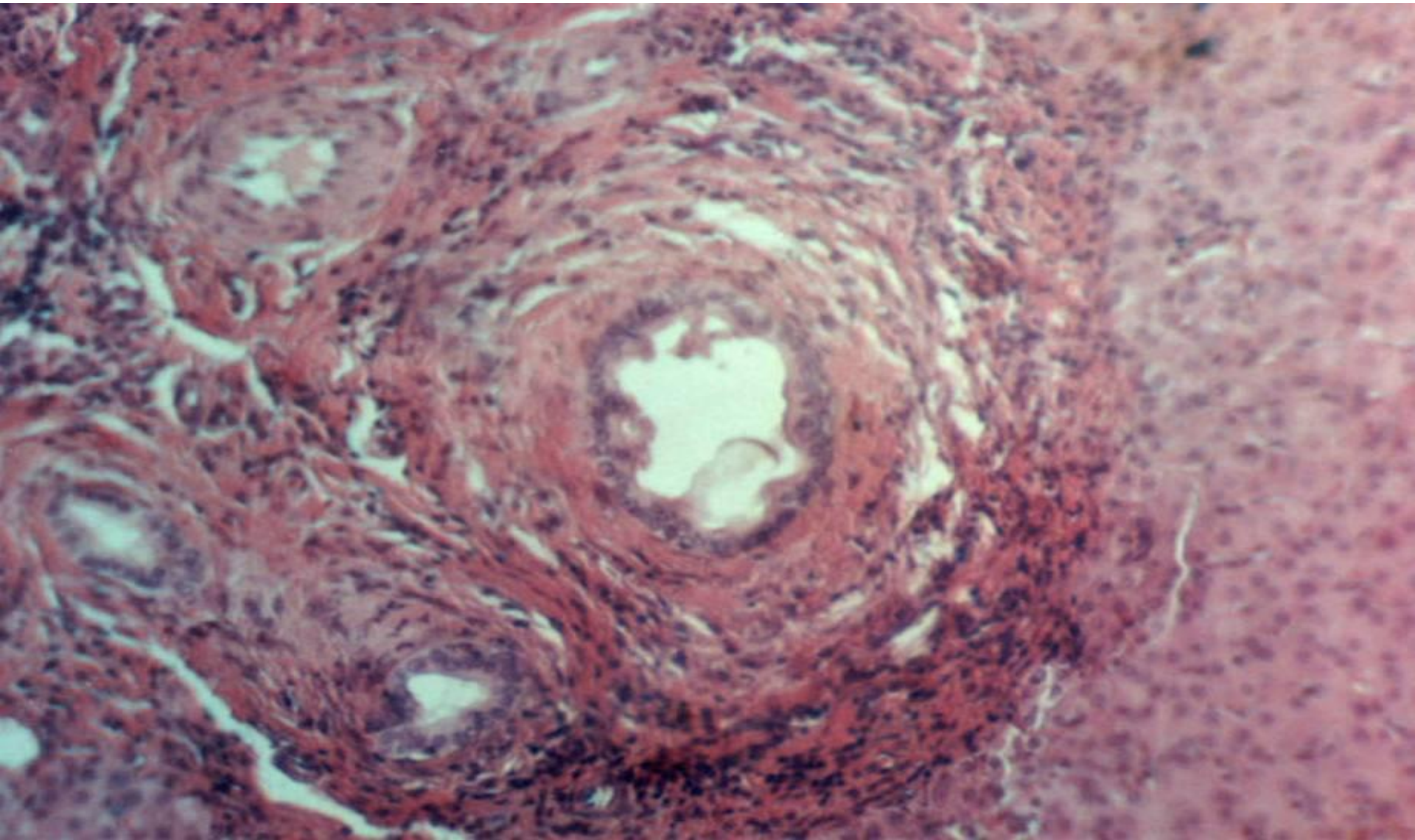
## Fibrous colangitis





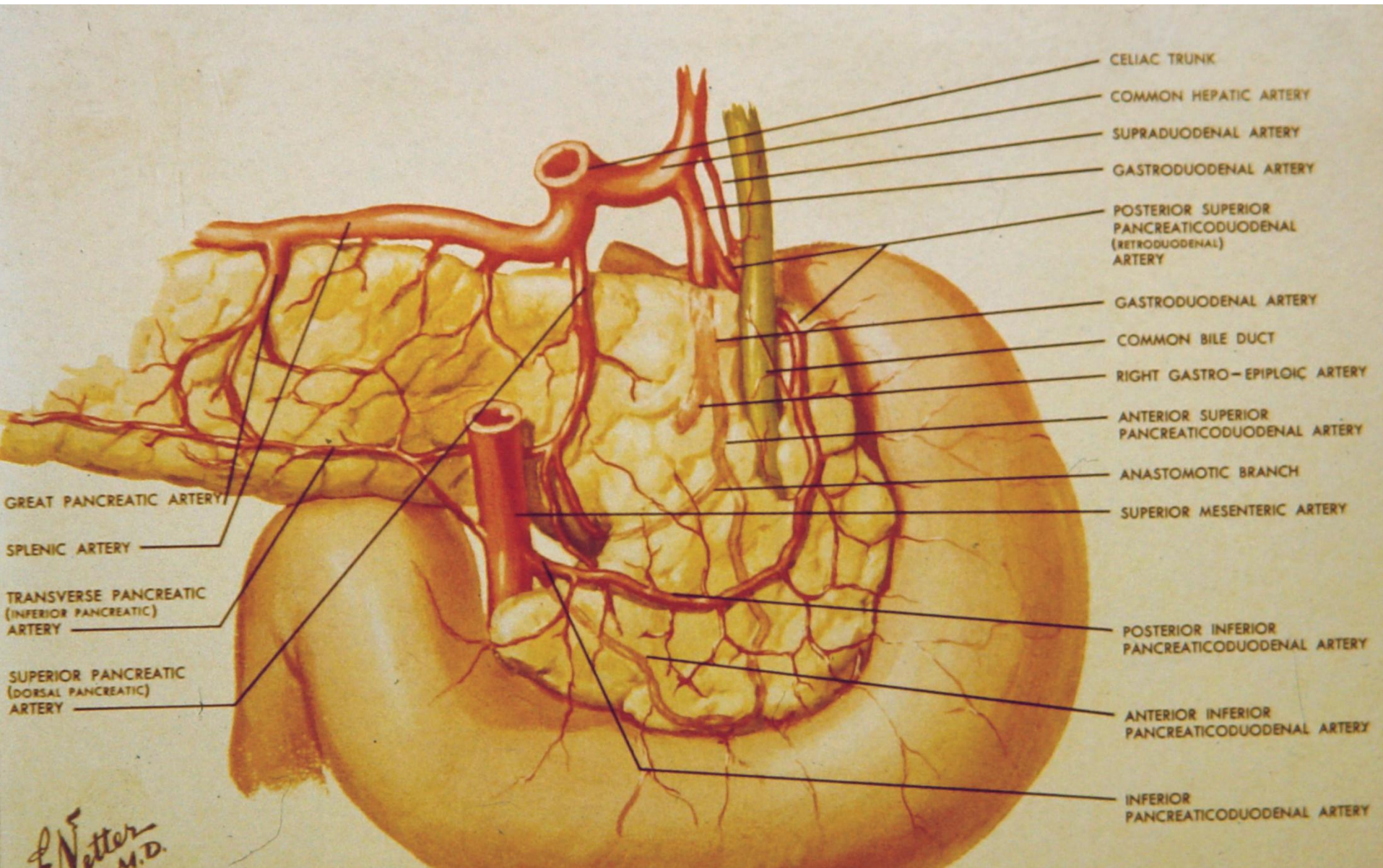
# BILIARY DUCTS

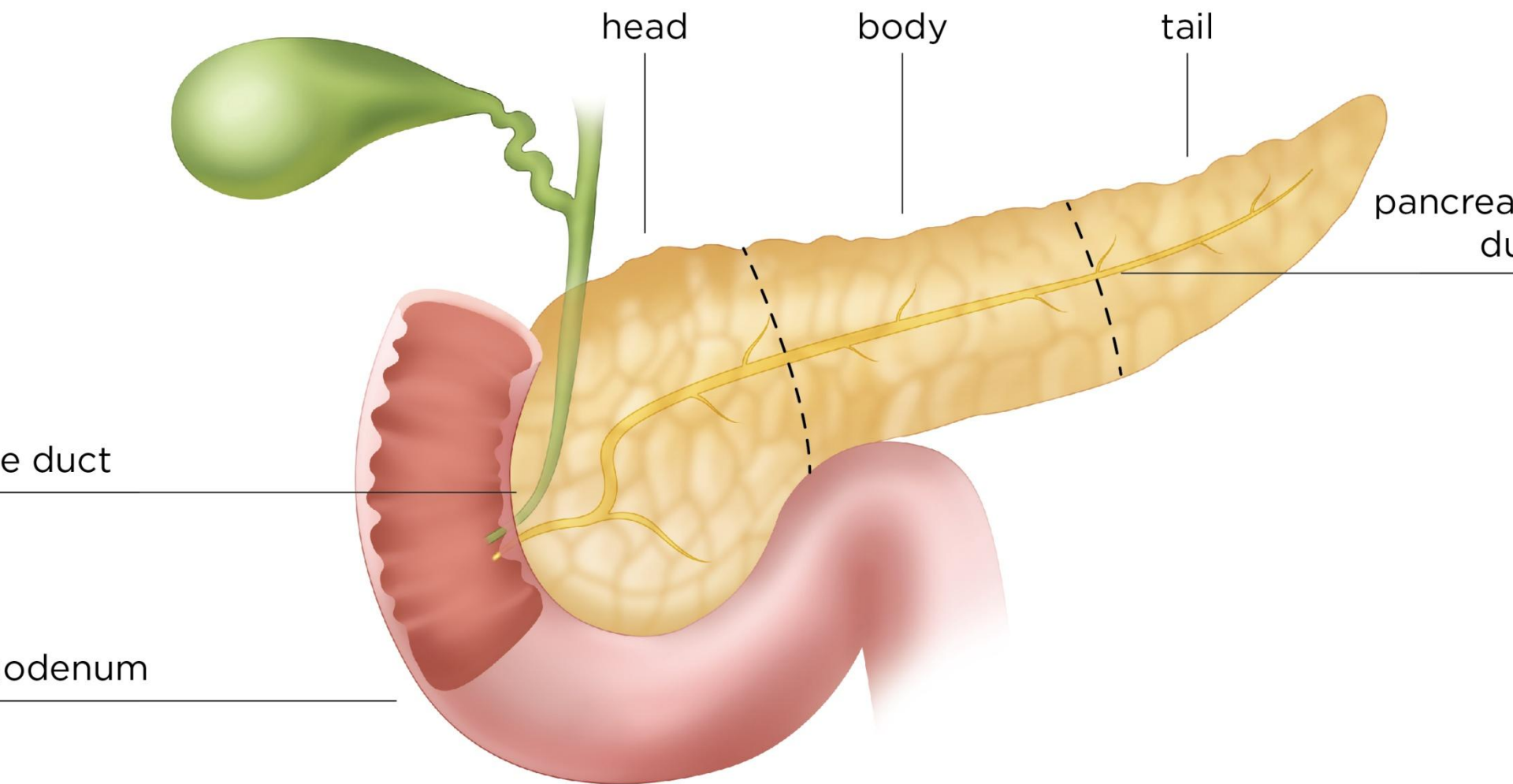
## Fibrous colangitis





# PANCREAS





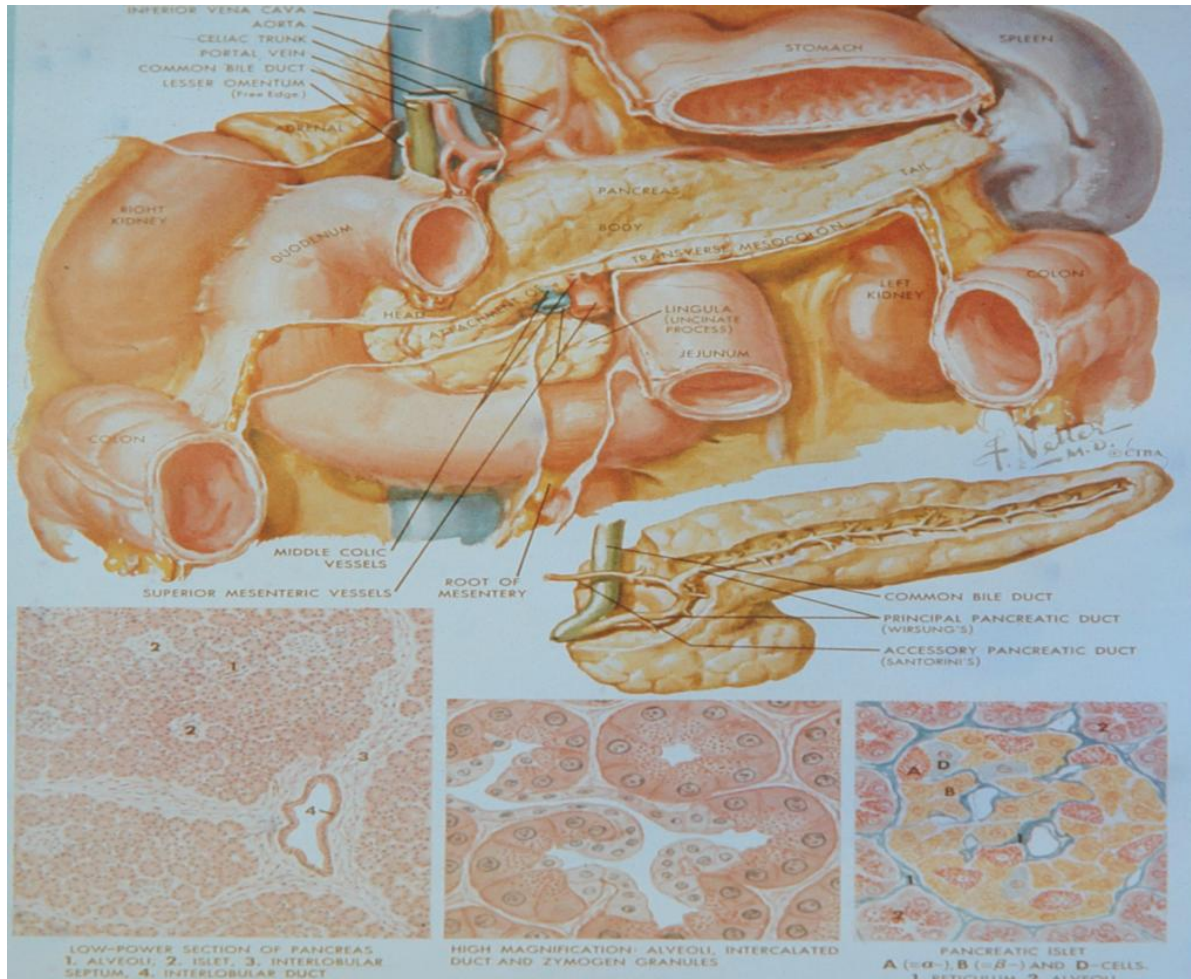


# PANCREAS

The pancreas is a retroperitoneal organ with intricate anatomic relationships to the

- duodenum,
- biliary tract,
- branches of the celiac axis and
- superior mesenteric artery,
- portal vein,
- spleen,
- stomach and
- colon.

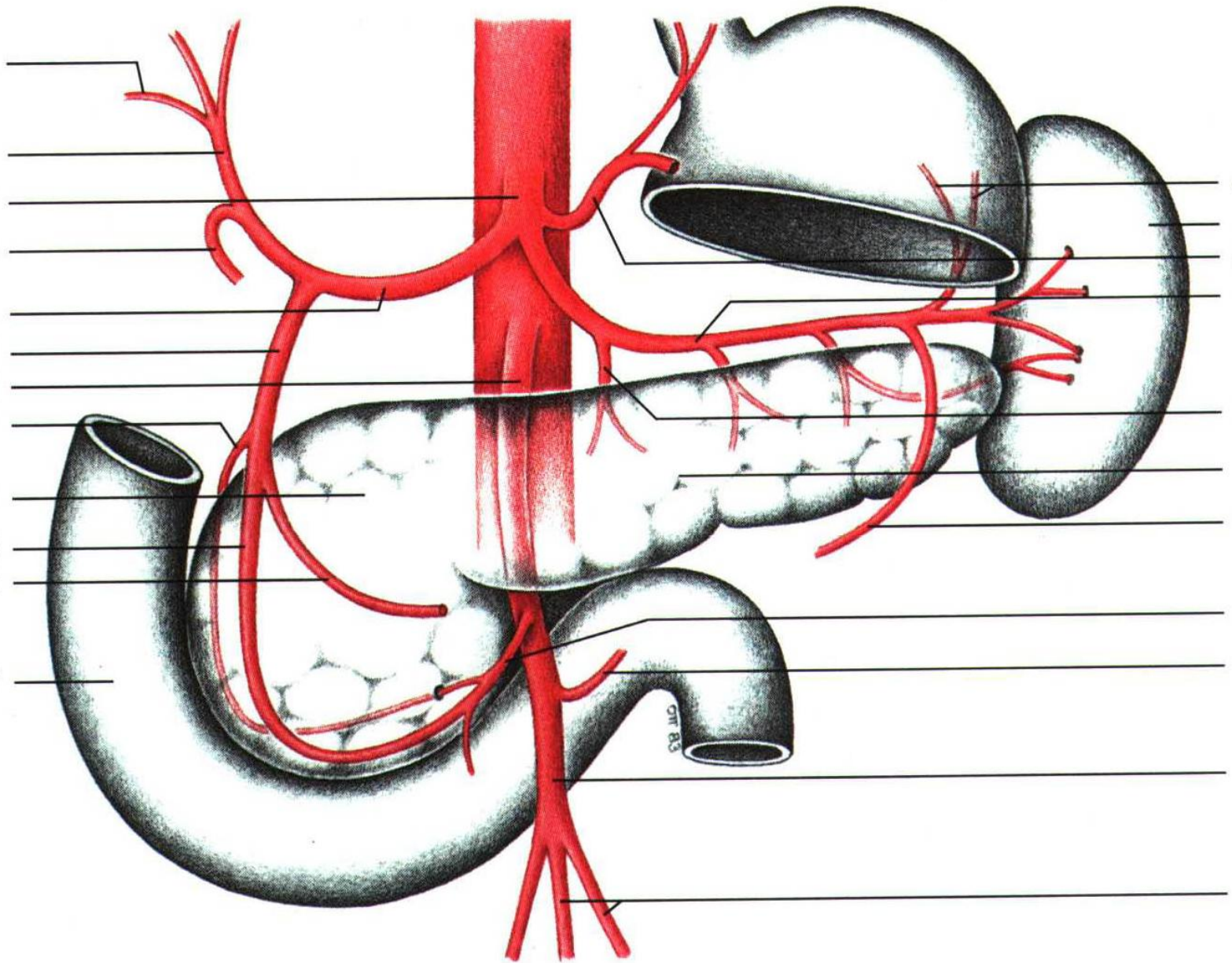
# PANCREAS



## Primary site

The pancreas is a long coarsely lobulated gland that lies transversely across the posterior abdomen and extends from the duodenum to the splenic hilum.

The organ is divided into a head with a small uncinate process, a neck, a body and a tail.



**Blood supply of upper abdominal organs** (branches of the celiac trunk and superior mesenteric artery). (Schematic drawing.)

## Regional lymph nodes

A rich lymphatic network surrounds the pancreas and accurate tumor staging requires that all lymph nodes are removed and analyzed.

Accordingly the pancreas has **two broad groups of regional nodes**:

- those that form a **ring around the organ** and
- those surrounding **the adjacent large vessels**:

- the abdominal aorta and its major branches including the celiac axis,
  - the superior mesenteric artery and
  - the renal arteries.

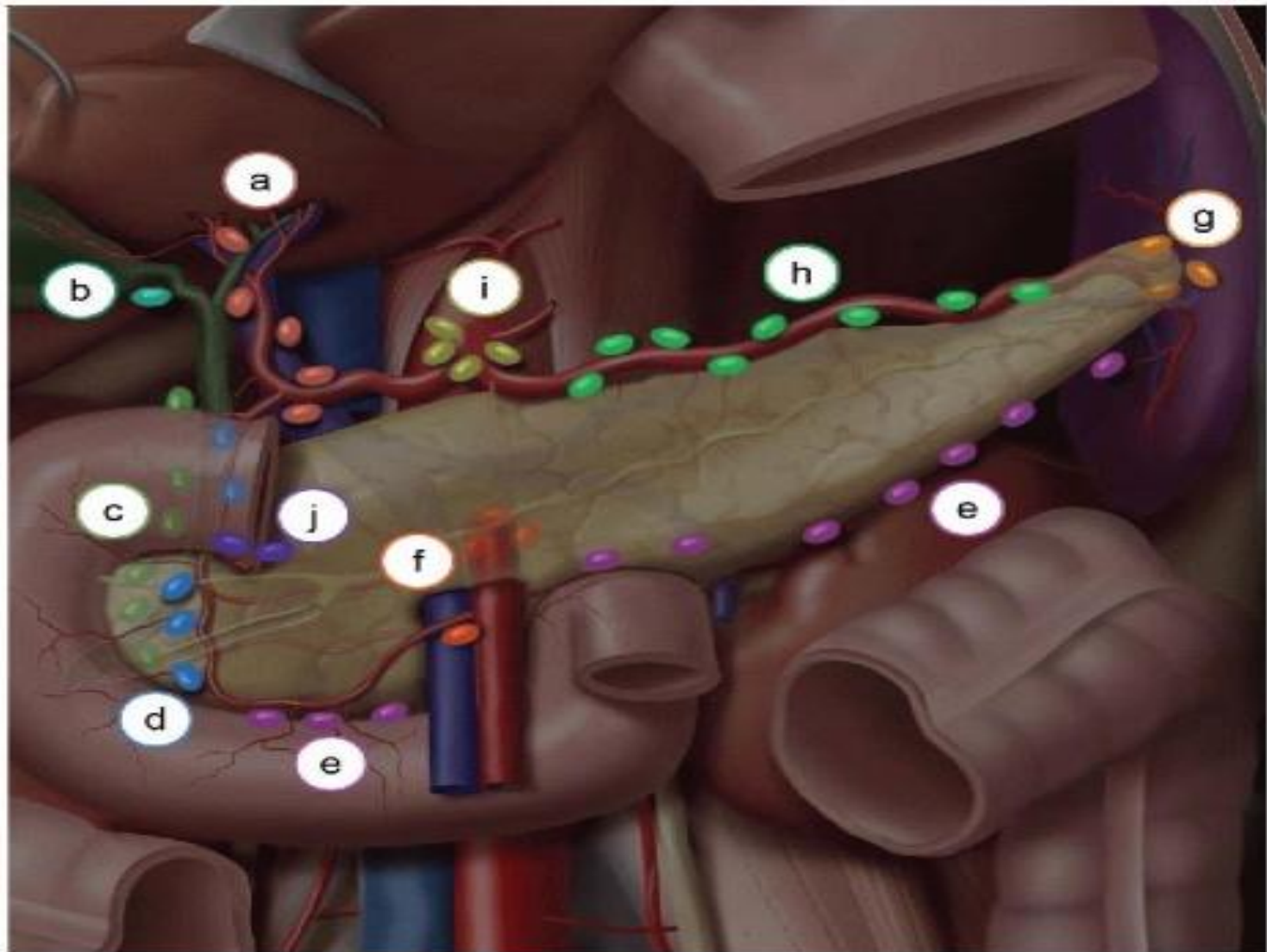


# Regional lymph nodes

The **specific nodal groups** include:

- ☐ the anterior pancreaticoduodenal nodes,
- ☐ the posterior pancreaticoduodenal nodes,
- ☐ the pyloric nodes,
- ☐ the gastroduodenal nodes,
- ☐ the hepatic nodes,
- ☐ the cystic nodes,
- ☐ the superior mesenteric nodes,
- ☐ the celiac nodes,
- ☐ the supra- and infrapancreatic nodes,
- ☐ the mesocolic nodes, the splenic nodes and
- ☐ gastrosplenic nodes.

## Peripancreatic Lymph Node Groups



The head of the pancreas is located on the right side of the abdomen, is the largest part of the organ, and is nestled in the duodenum.

The body of the pancreas extends across the spine and is in the midline of the abdomen.

The tail of the pancreas is located on the left side of the abdomen and is adjacent to the spleen.

The pancreas consists of two kinds of tissues:

**Exocrine** -- which make powerful **enzymes** to digest fats, proteins, and carbohydrates. The enzymes normally are produced and carried in an inactive form to the duodenum where activation occurs as needed. Exocrine tissues also make and secrete bicarbonates that work to neutralize stomach acids thereby allowing for the activation of the pancreatic enzymes. Without these enzymes, food is not digested and absorbed, leading to malnutrition, weight loss, and diarrhea.

**Endocrine** -- which produce the **hormones** insulin and glucagon and release them into the blood stream. These hormones regulate glucose transport into the body's cells and are crucial for maintaining normal glucose levels and energy production.

# PANCREAS

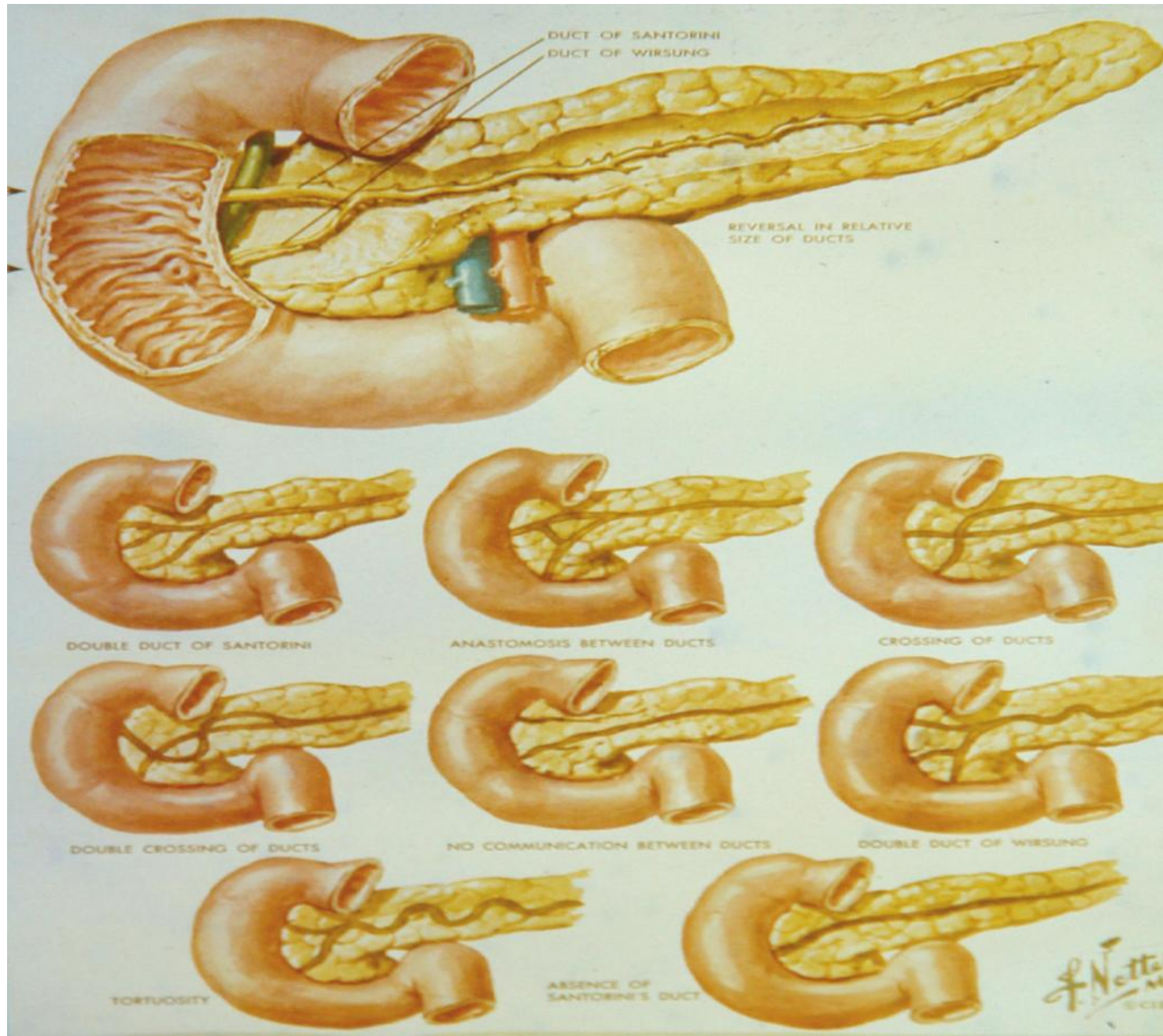
The pancreatic **duct** carry the  
**exocrine drainage**

from the pancreas to the duodenum.

Normally the main pancreatic duct begins in the tail of the pancreas and ends with the common biliary duct at **Vater~s papilla** (major duodenal papilla), approximately 10 cm from the pylorus.



# PANCREAS



# PANCREAS

## PANCREAS DIVISIUM

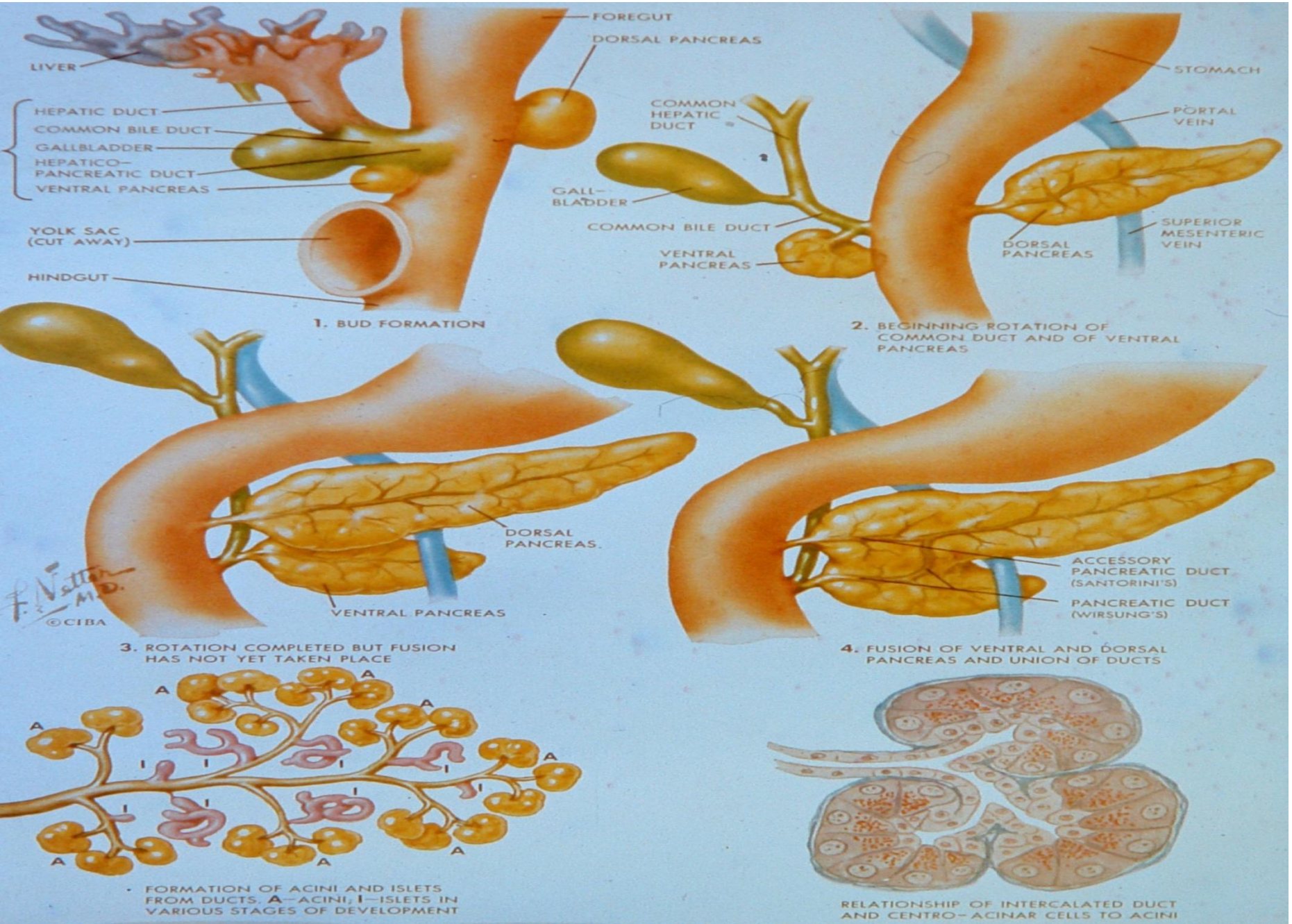
Pancreas divisum is a congenital anomaly of the pancreatic ducts in which the main pancreatic duct and the accessory pancreatic duct fail to fuse with the main pancreatic duct entering the duodenum proximal to Vater's papilla.

Most patients are asymptomatic and some patients will develop recurrent episodes of acute pancreatitis.

The surgical treatment is a sphincterectomy or sphincteroplasty of the minor duct orifice.



# PANCREAS



There are a variety of disorders of the pancreas including acute pancreatitis, chronic pancreatitis, hereditary pancreatitis, and pancreatic cancer.

The evaluation of pancreatic diseases can be difficult due to the inaccessibility of the pancreas.

There are multiple methods to evaluate the pancreas. Initial tests of the pancreas include:

- ❑ a physical examination, which is difficult since the pancreas is deep in the abdomen near the spine.
- ❑ Blood tests are often helpful in determining whether the pancreas is involved in a specific symptom but may be misleading.



The best radiographic tests to evaluate the structure of the pancreas include

- ❑ CAT (computed tomography) scan,
- ❑ endoscopic ultrasound, and
- ❑ MRI (magnetic resonance imaging).

Tests to evaluate the pancreatic ducts include

- ❑ ERCP (endoscopic retrograde cholangiopancreatography) and
- ❑ MRCP (magnetic resonance cholangiopancreatography).

There are also instances in which **surgical exploration** is the only way to confirm the macroscopic diagnosis of pancreatic disease.

The biopsy give the certain diagnosis.

## **Acute pancreatitis**

The causes of acute inflammation of the pancreas is a complex inflammatory process secondary to intraparenchymal activation of the digestive enzymes.

The degree of pancreatic inflammation and systemic toxicity is unpredictable at the beginning of an episode, therefore the mildness or severity may not be evident for 24 to 72 hours.

# Etiology

Biliary tract disease (e.g., cholelithiasis)

Alcohol

Drugs

Trauma

Hyperparathyroidism (elevated calcium)

Malnutrition (protein-calorie)

Hyperlipidemia

Pancreatic duct obstruction (e.g., pancreas divisum)

Duodenal obstruction

Infection

Ischemia

Hereditary

Scorpion venom

## **Etiological diagnosis**

- ultrasound scan
- CT scan
- biliary scan
- endoscopic retrograde cholangiopancreatography (ERCP)
- percutaneous transhepatic cholangiography (PTC)



# Acute pancreatitis mechanism

- proteolytic action of trypsin and chymotrypsin;
- elastase dissolving elastic fibers in vessel walls and cause bleeding
- release of bradykinin and kallikrein produce vasodilation and increased capillary permeability;
- turns histidine by decarboxylation produces histamine and vascular disorders;
- attacks lipase lipid components, initiate autodigestion of fat and produce fat necrosis;
- phospholipase A damage cell membranes and cause microbleeds and perivascular pancreatic necrosis;

Changes occurring in the chain is self-sustaining, cascading their character;

Amylase not interfere in the process of necrosis, but spreading in blood and urine has diagnostic value.

## Pancreatic secretion diffusion pathways:

- **canalicular** way through Wirsung
- leaking liquid in pancreatic juice, blood and necrotic debris in the peritoneal and retroperitoneal spaces;
- **intraperitoneal extravasation** of pancreatic secretion
- diffusion in **retroperitoneal space** of pancreatic secretion: kidney damage, the adrenal glands,
- In the **mediastinum** (pleural and pericardial exudates);
- path of **blood**, is done by portal vena

## **Pancreatic secretion diffusion pathways:**

lymphatic and about circuits described by Montaldo:  
a direct in his tank Pecquet,  
an indirect way pancreato -peritoneal (small  
circuit)  
peritoneal route (great track)

Biliary-way nodes;

# Regional and systemic **enzyme diffusion** causes **functional alterations** and **acute organ failure**

- electrolyte disorders hypotension (hypovolemia)
- shock pain (irritation of the solar plexus)
- irritation autonomic phenomena;
- shock by vasoactive substances action (kinine, histamine)
- ventilatory dysfunction
- clotting disorders, hypercoagulability (CID) or hypocoagulability.
- decreased ventricular function due to myocardial depressant factor (MDF) produced by the pancreas;
- **pancreatic encephalopathy**, the presence of pancreatic enzymes circulating in their blood and central nervous system, where it causes brain damage (edema, myelin degeneration, necrosis and hemorrhagic foci) consecutive neuropsychiatric disorders;
- renal failure, acute circulatory failure is due (hypovolemia), dehydration, electrolyte imbalance and the presence of emboli secondary to renal small vessels;
- liver with biliary obstruction mechanically (stones, papillitis or edema of the head of pancreas and direct toxic action of pancreatic enzymes in the liver reached via port system (cause direct suffering hepatocytes)
- hormone metabolic disorders (hyperglycemia and hiperglucagonemia).



# Mild acute pancreatitis

## Subjective findings.

The patient has **abdominal pain** that begins in the epigastrium and radiates across the upper abdomen into the back.

Because of the associated ileus, most patients have **nausea and vomiting** (either spontaneous or induced by food or fluid ingestion).

## Objective findings.

The patient may have **signs of dehydration** because of fluid extravasation in the retroperitoneum and peritoneal cavity and because of the associated gastrointestinal pooling caused by **ileus**.

**Postural hypotension** may be present. Most patients are afebrile with a sustained **tachycardia** of multifactorial origin including hypovolemia, pain, chemically mediated toxicity and possibly sepsis.

Findings on examination may range from **minimal epigastric tenderness to generalized rigidity and diffuse peritoneal irritation**. **Tachypnea** with reduced respiratory excursion occurs even in mild cases.

# acute pancreatitis

## Objective findings.

- Cyanotic spots in periumbilical region (**Cullen sign**)



# acute pancreatitis

## Objective findings.

- or on flanks **(Grey-Turner sign)**;



# acute pancreatitis

## Objective findings.

painful characteristic points:

**Mayo-Robson** sign: left angle of the  
ribs and vertebrae

**Mallet-Guy** sign: below the left grid

- ♣ abdominal wall percussion: high sonority
- ♣ auscultation: abdominal silentium (ileus).



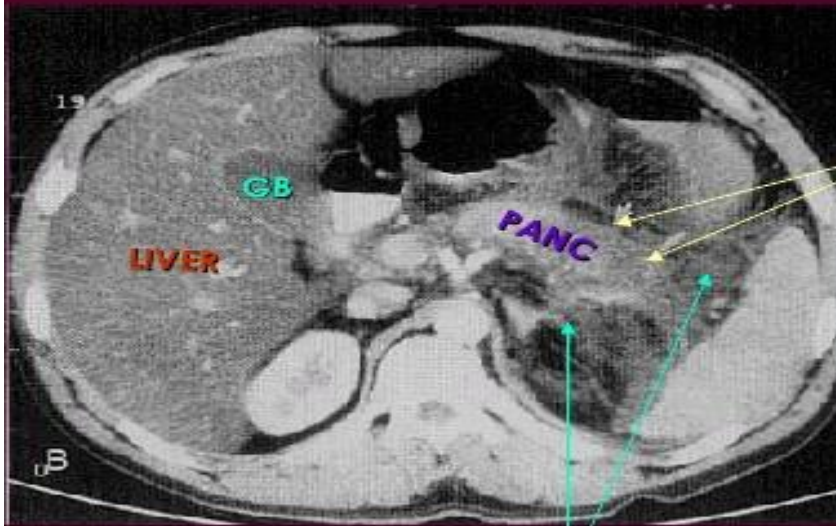
# **Acute pancreatitis**

## **Assessment.**

White blood cell count is usually elevated to 12.000 to 15.000/mm<sup>3</sup>

# Acute pancreatitis

## CT Findings Severe Pancreatitis



Unenhancing  
Necrosis

Peripancreatic edema  
and inflammation

# DIAGNOSTIC PLAN FOR ACUTE PANCREATITIS

## **Confirmation**

- Serum amylase,elevated lypase
- Amylase/creatinine clearance ratio

## **Determination of severity**

- hemoglobin, hematocrit, white blood count, platelets
- prothrombine time, partial thromboplastin time
- calcium, glucose, albumin
- arterial blood gases
- BUN,creatinine
- bilirubin, alkaline phosphatase, lactatedehydrogenase (LDH),
- serum glutamic-oxaloacetic transaminase (SGOT)

# CRITERIA OF SEVERITY OF ACUTE PANCREATITIS

Criteria initially present

- age over 55 years
- white blood cell count  $> 16.000/uL$
- blood glucose  $> 200\text{ mg/dL}$
- serum LDH  $>350\text{ IU/L}$
- SGOT(AST)  $>250\text{ IU/dL}$



# CRITERIA OF SEVERITY OF ACUTE PANCREATITIS

Criteria developing during first 48 hours

- hematocrit fall  $>10\%$
- BUN rise  $> 5\text{ mg/dL}$
- serum  $\text{Ca}^{2++}$   $< 8\text{mg/dL}$
- arterial  $\text{PO}_2 < 60\text{mm/Hg}$
- base deficit  $< 4\text{ mEq/L}$
- estimated fluid sequestration  $>6000\text{ mL}$

## **Morphological forms of acute pancreatitis**

- ☐ Edematous
- ☐ Hemorrhagic
- ☐ Necrotic

## **Clinical forms**

- ☐ Severe, with encephalopathy and death in 6 hours,
- ☐ Mimicking intestinal obstruction
- ☐ Mimicking biliary colicky
- ☐ Mimicking ulcer disease

## **Etiological forms**

- ♣ acute gallstone pancreatitis (50-60%);
- ♣ alcoholic acute pancreatitis with acute psychiatric disorders
- ♣ postoperative acute pancreatitis;
- ♣ traumatic acute pancreatitis;
- ♣ primitive acute pancreatitis.

# PANCREAS





## **Differential diagnosis**

is made with: conditions that simulate medical and surgical acute abdomen

- ♣ perforated gastroduodenal ulcer;
- ♣ intestinal occlusion;
- ♣ bowel infraction;
- ♣ acute cholecystitis,
- ♣ appendicitis and
- ♣ Acute diverticulitis;
- ♣ ruptured ectopic pregnancy;
- ♣ strangulated diaphragmatic hernia;
- ♣ dissecting aneurysm of the aorta.

# Differential diagnosis

- plain films of the abdomen (perforated viscus)
- CT scan
- angiography
- Laparotomy

Acute suppurative pancreatitis is usually a **complication of acute pancreatitis**, contamination by lymphatic or hematogenous, with germs. Pancreas can be turned into diffuse peripancreatic phlegmon.

# Objectives of treatment

- Management of Pain,
- Treatment of shock:
- Inhibition of pancreatic secretion
- high parenteral nutrition
- AB therapy:
- Treatment of respiratory failure:
- stress ulcer prophylaxis:

## **Surgery Management:**

- immediate emergency, in case of: uncertainty diagnostic peritoneal irritation signs;
- Emergency delayed (24 hours to 5 days) in acute pancreatitis refractory to
- medical treatment correctly or aggravated
- late response (after 2-3 weeks);
- Local complications:
  - abscess,
  - pseudocyst,
  - intraperitoneal bleeding;
  - etiological indications: cholelithiasis, Wirsung ductal stones



# CAUSES OF RECURRENT ACUTE PANCREATITIS (NONALCOHOLIC NONBILIARY)

- **Anatomic**
- **Metabolic**
- **Drug induced**
- **Infectious**
- **Vascular**
- **Idiopathic**

# **CAUSES OF RECURRENT ACUTE PANCREATITIS (NONALCOHOLIC NONBILIARY)**

## **1/Anatomic**

- ductal obstruction**
- pancreas divisum**
- congenital ductal strictures**
- carcinoma of pancreas ,Vater's ampulla**
- 'regional inflammation**
- peptic ulcer disease**
- postoperative pancreatitis**
- postgastrectomy states**

# CAUSES OF RECURRENT ACUTE PANCREATITIS (NONALCOHOLIC NONBILIARY)

## **2/Metabolic**

- »hyperlipidemia,
- »hypertriglyceridemia
- »Hyperparathyroidism

## **3/Drug induced**

- »diuretics
- »oral hypoglycemics
- »oral contraceptives
- »Immunosuppressives

# **CAUSES OF RECURRENT ACUTE PANCREATITIS (NONALCOHOLIC NONBILIARY)**

## **4/Infectious**

- viral (mumps, coxsackie virus)**
- bacterial (coliforms)**

## **5/Vascular**

- ischemic pancreatitis (major vascular occlusion)**
- systemic lupus erythematosus (SLE) and**
- other collagen vascular diseases**



# **INVESTIGATION OF RECURRENT ACUTE PANCREATITIS (NONALCOHOLIC, NONBILIARY)**

## **Clinical history**

- »familial diseases
- »drug history
- »viral exposure
- »previous gastrointestinal surgery

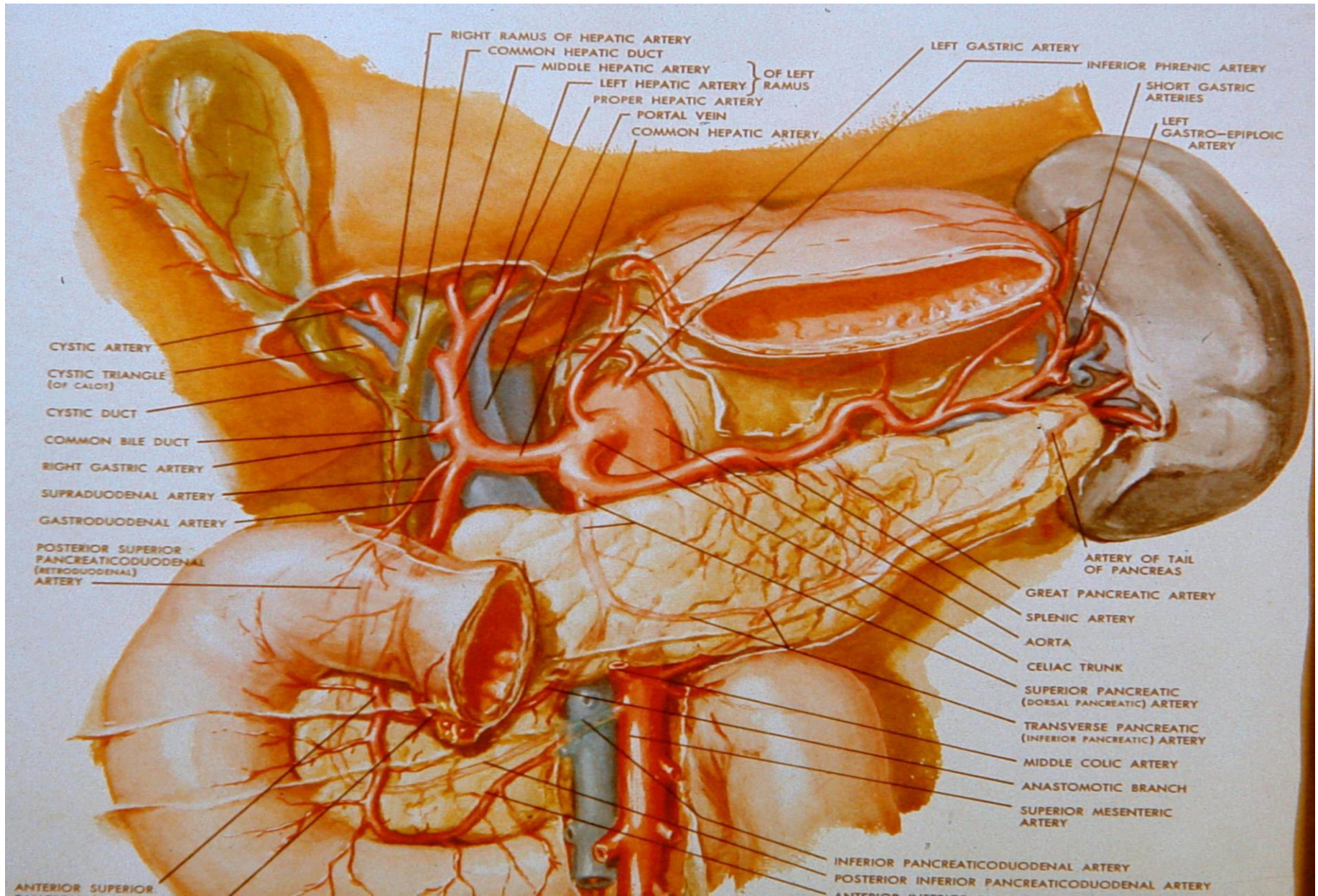
## **Laboratory investigation**

- »lipoprotein electrophoresis, triglyceride, cholesterol
- »calcium metabolism, parathormone
- »hematology/immunology-collagen vascular disease
- »viral studies

## **Radiology and endoscopy**

- »ultrasound scanning (CT scan)
- »upper gastrointestinal endoscopy
- »endoscopic retrograde pancreatography
- »selective angiography

# PANCREAS



# CHRONIC PANCREATITIS

## Subjective findings.

Repeated inflammation of the pancreas can lead to chronic pancreatitis, with **pain** aggravated by food intake and exocrine insufficiency.

**Malnutrition** and **weight loss** usually occur due to inadequate food ingestion. More than two thirds of the patients have history of alcoholism.

## Objective findings.

Tenderness is minimal or absent, voluntary guarding of the abdomen is common and nonspecific and a palpable mass should be considered a complication of pancreatitis or a neoplasm.

# Potential Causes of Pain in Chronic Pancreatitis

Pancreatic ductal hypertension

Inflammation of the intrapancreatic nerves

Loss of the protective perineural sheath in pancreatic nerves

Pancreatic ischemia

Pseudocysts

Pancreatic and peripancreatic infection

Biliary obstruction

Cholangitis

Duodenal obstruction



## **CHRONIC PANCREATITIS**

### **Diagnostic plan.**

Pancreatic calcification can be documented by:  
abdominal roentgenogram,  
abdominal ultrasound or  
CT scan.

The pancreatic duct changes in chronic pancreatitis are best identified by ERCP.

All patients suspected of having chronic pancreatitis should undergo examination for diabetes.

### **Management plan.**

Selected patients can receive surgical treatment for pain relief.  
A chronic alcoholic person is a contraindication to operative treatment for pain.

# **Indications for Surgery in Chronic Pancreatitis**

Pain

Common bile duct obstruction

Duodenal obstruction

Colon obstruction

Pseudocyst

Suspicion of pancreatic cancer

Splenic vein obstruction with left-sided portal hypertension

Portal vein obstruction with portal hypertension

## **CHRONIC PANCREATITIS**

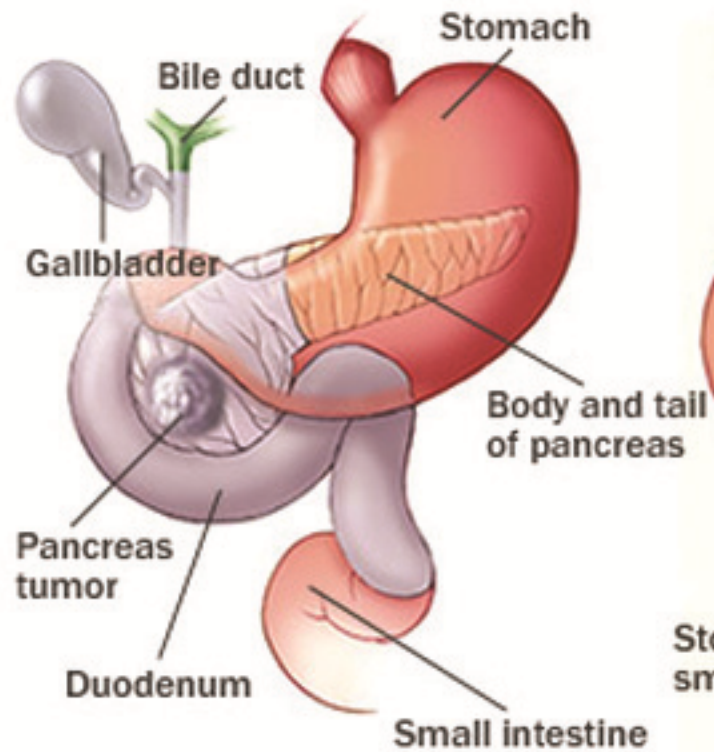
Operative procedures include:

- pancreatico-jejunostomy,

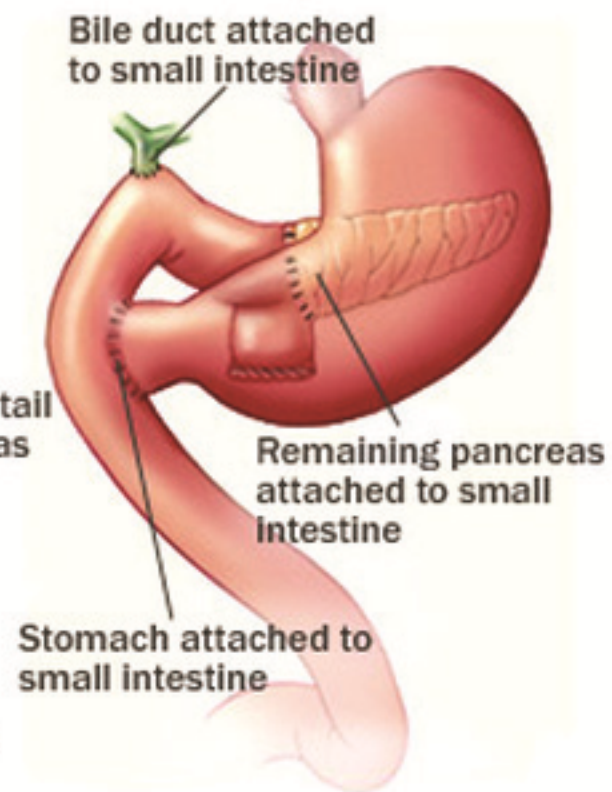
- pancreatic resection or

- intraoperative celiac ganglion block to interrupt afferent nerve impulses from the pancreas.

**Before surgery**

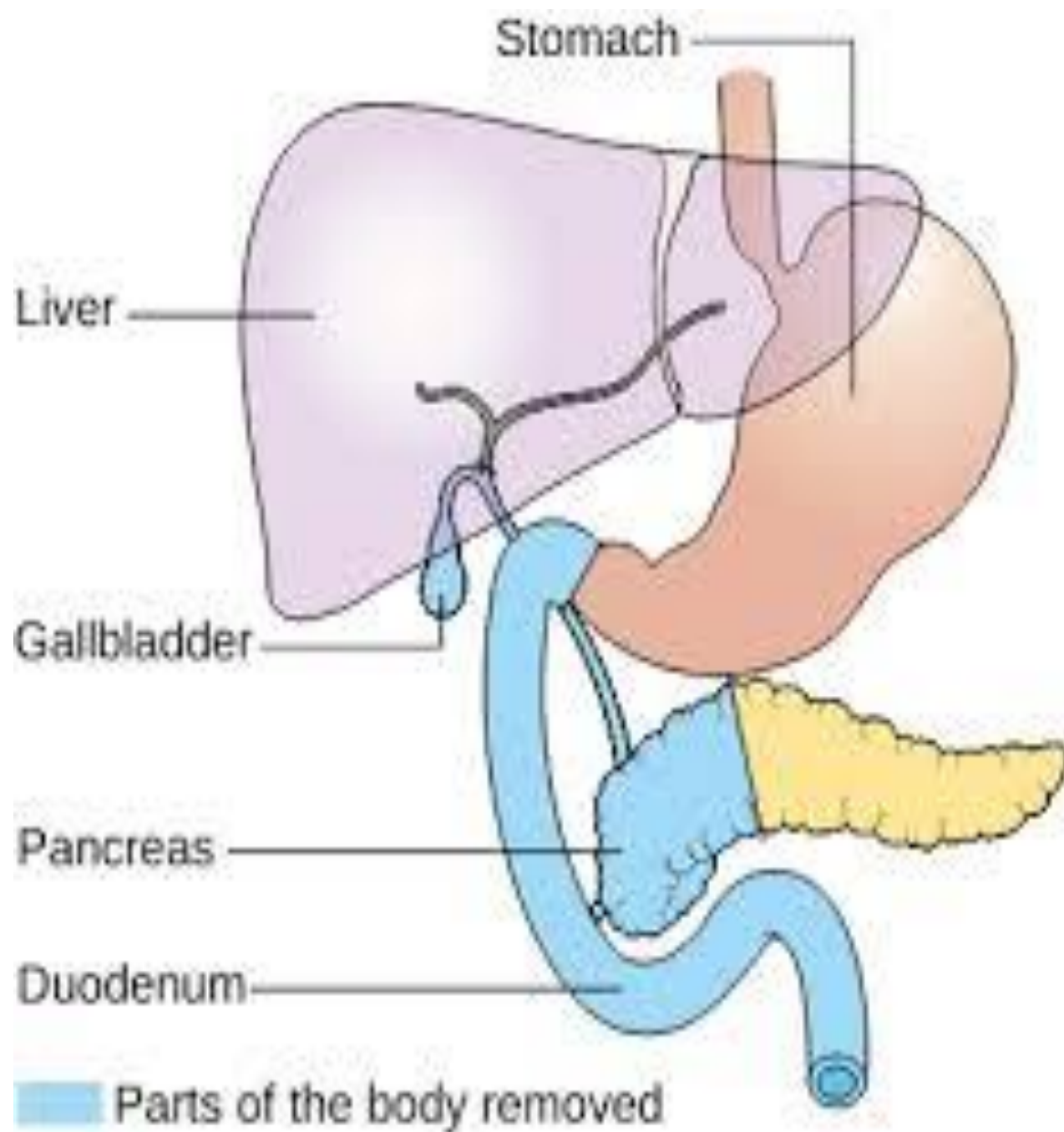


**After surgery**

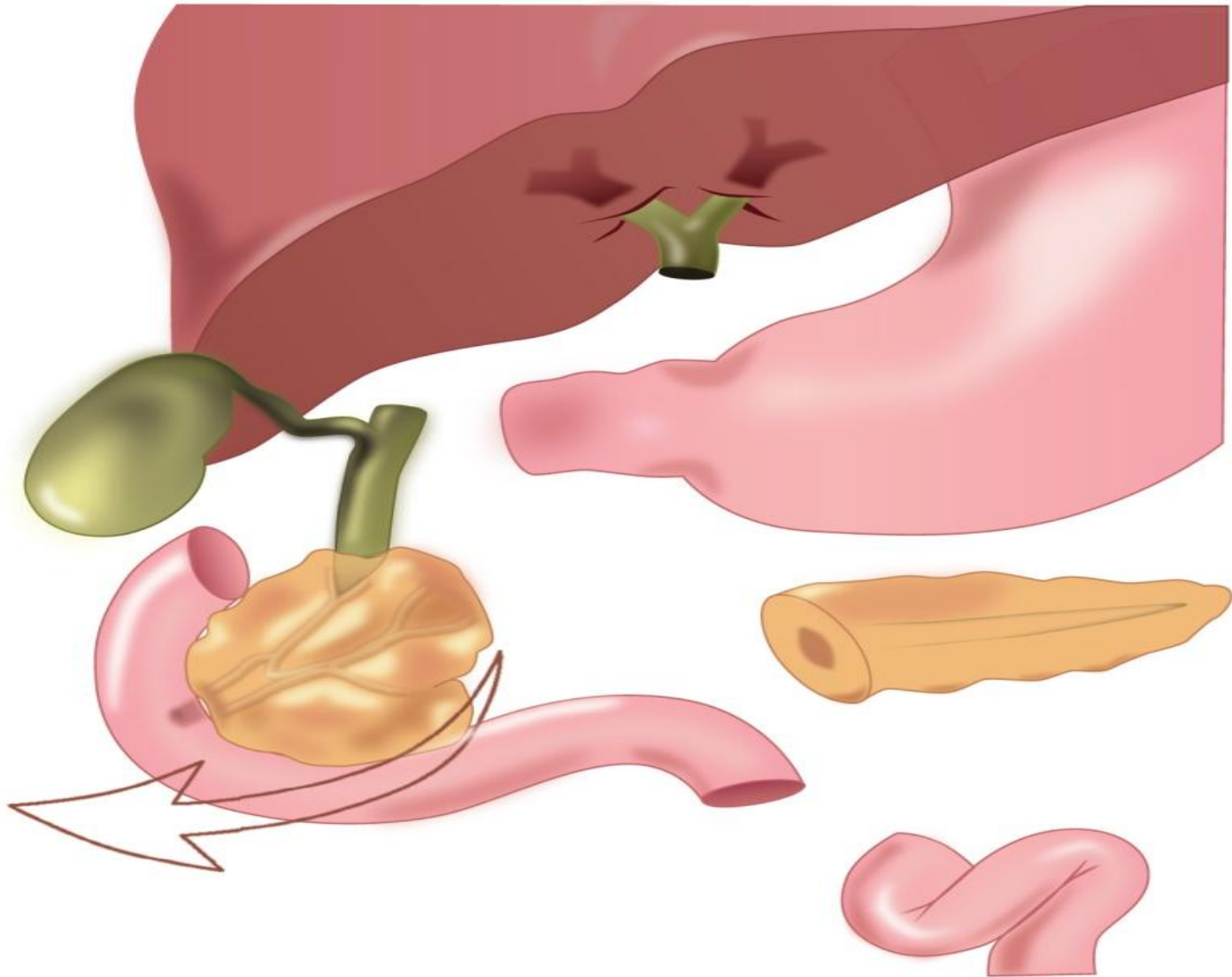


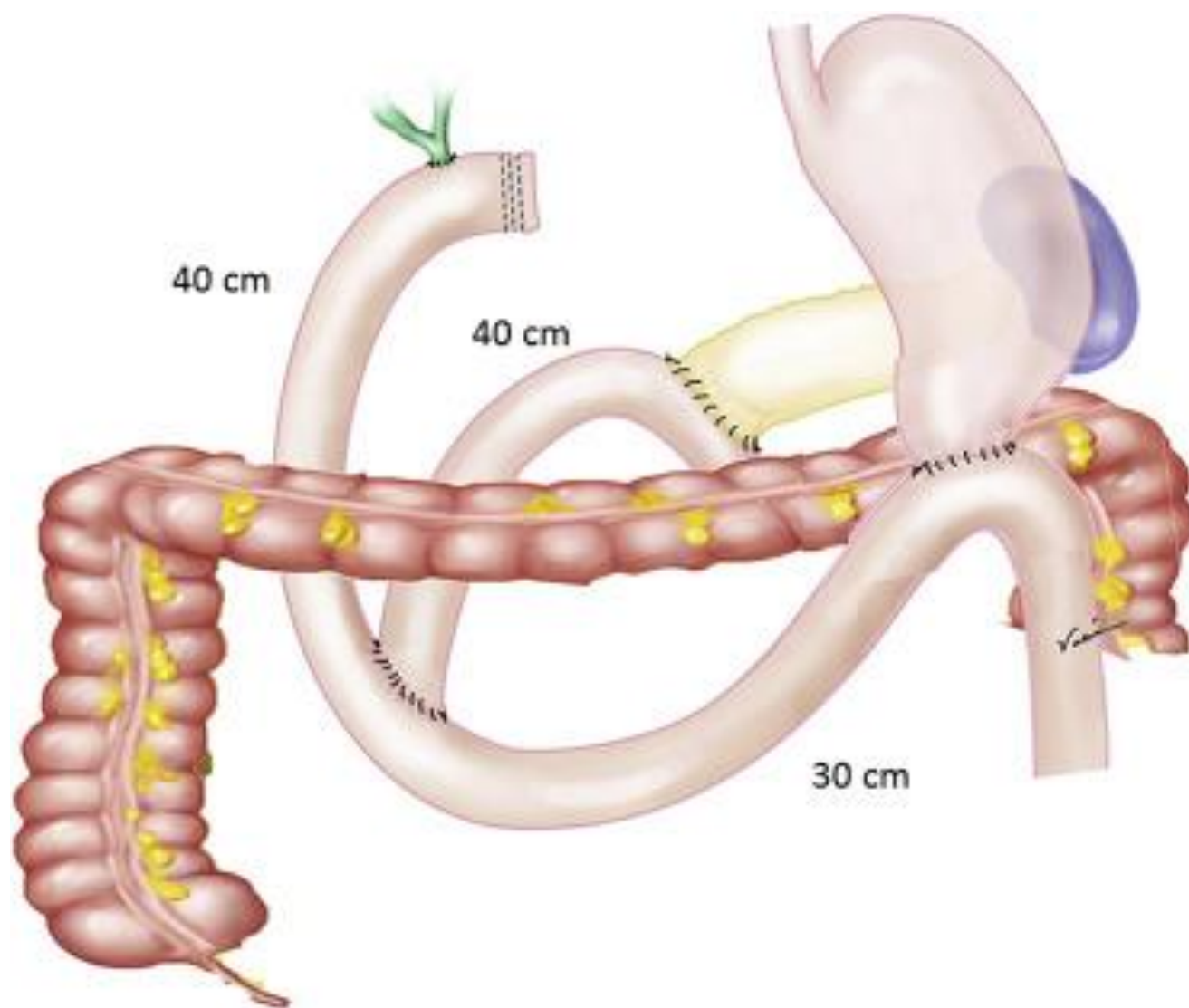


pancreatic resection

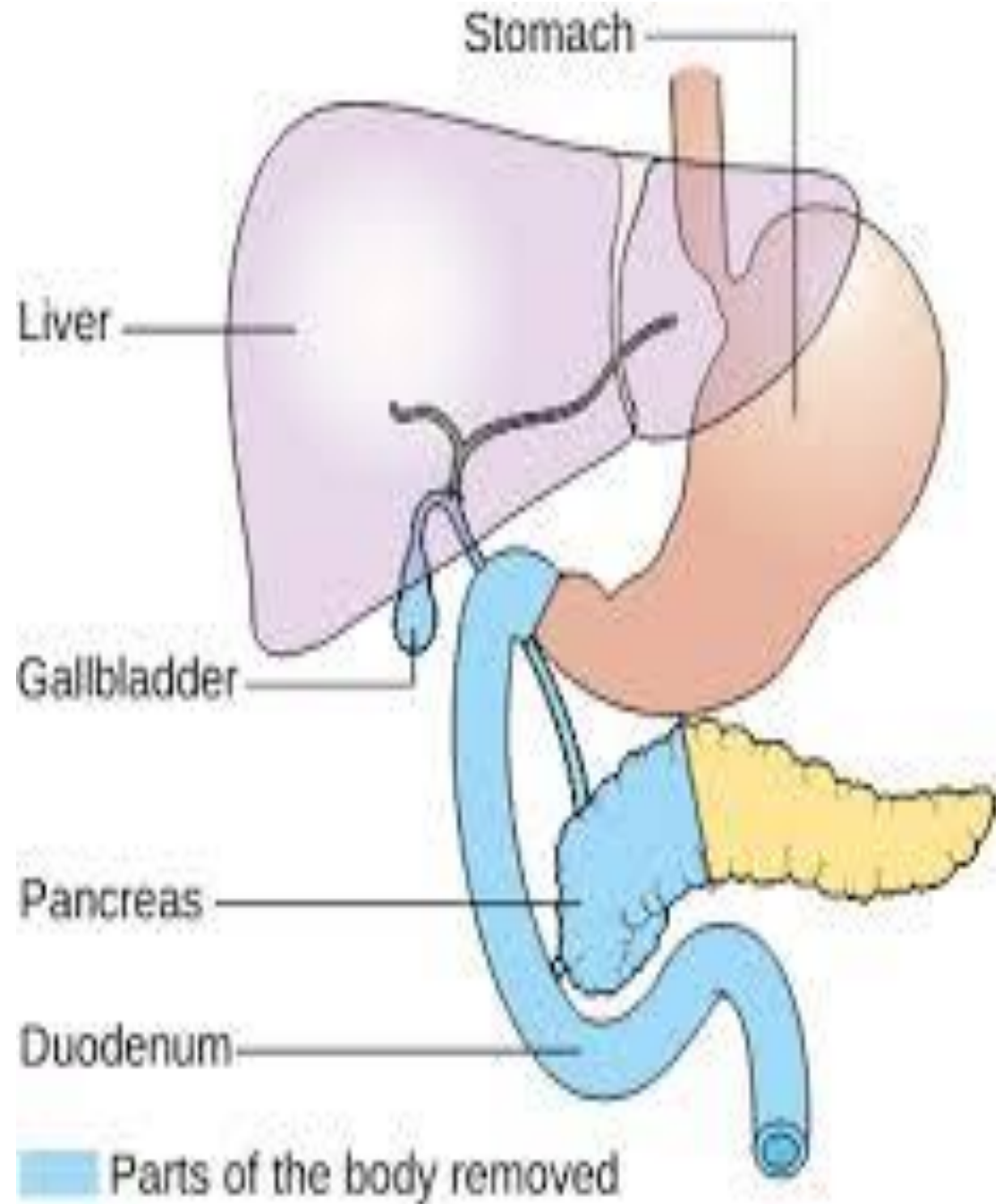
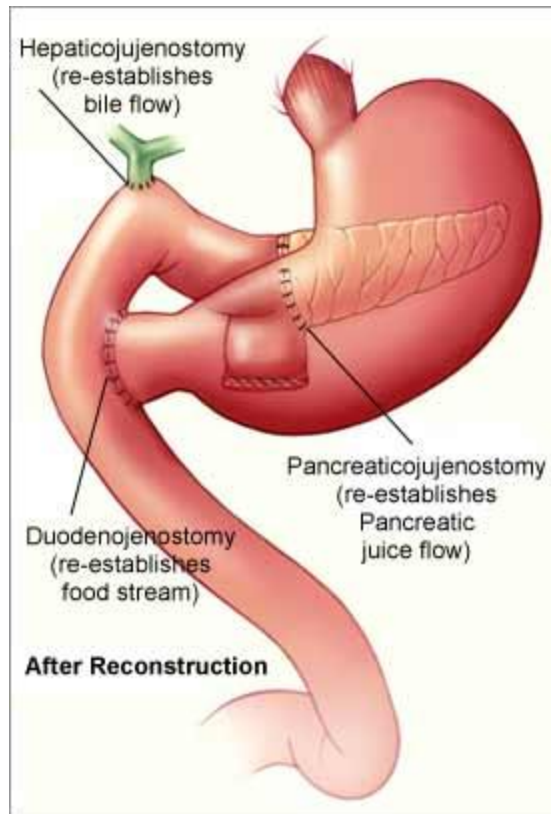


pancreatic resection



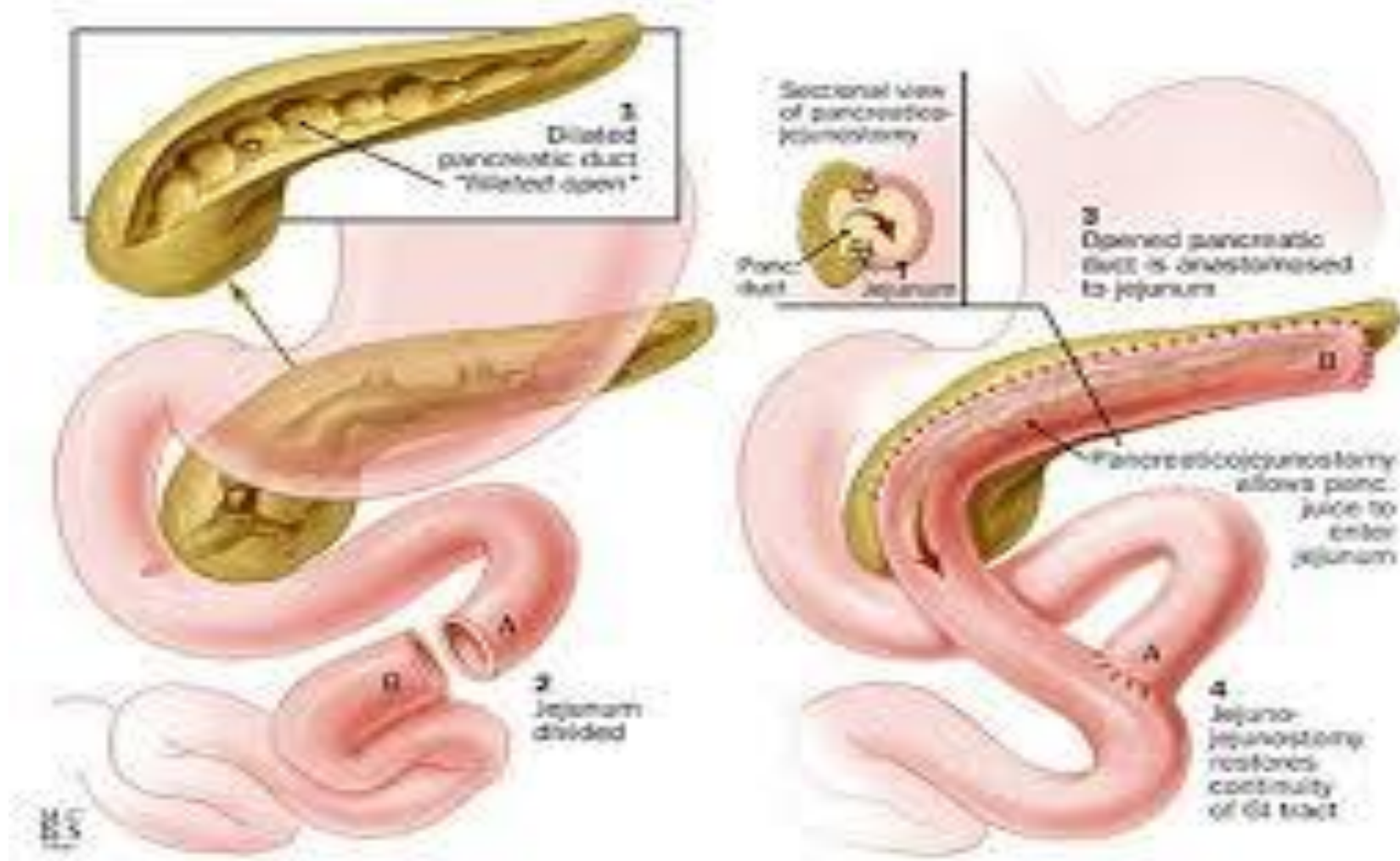


## pancreatic resection

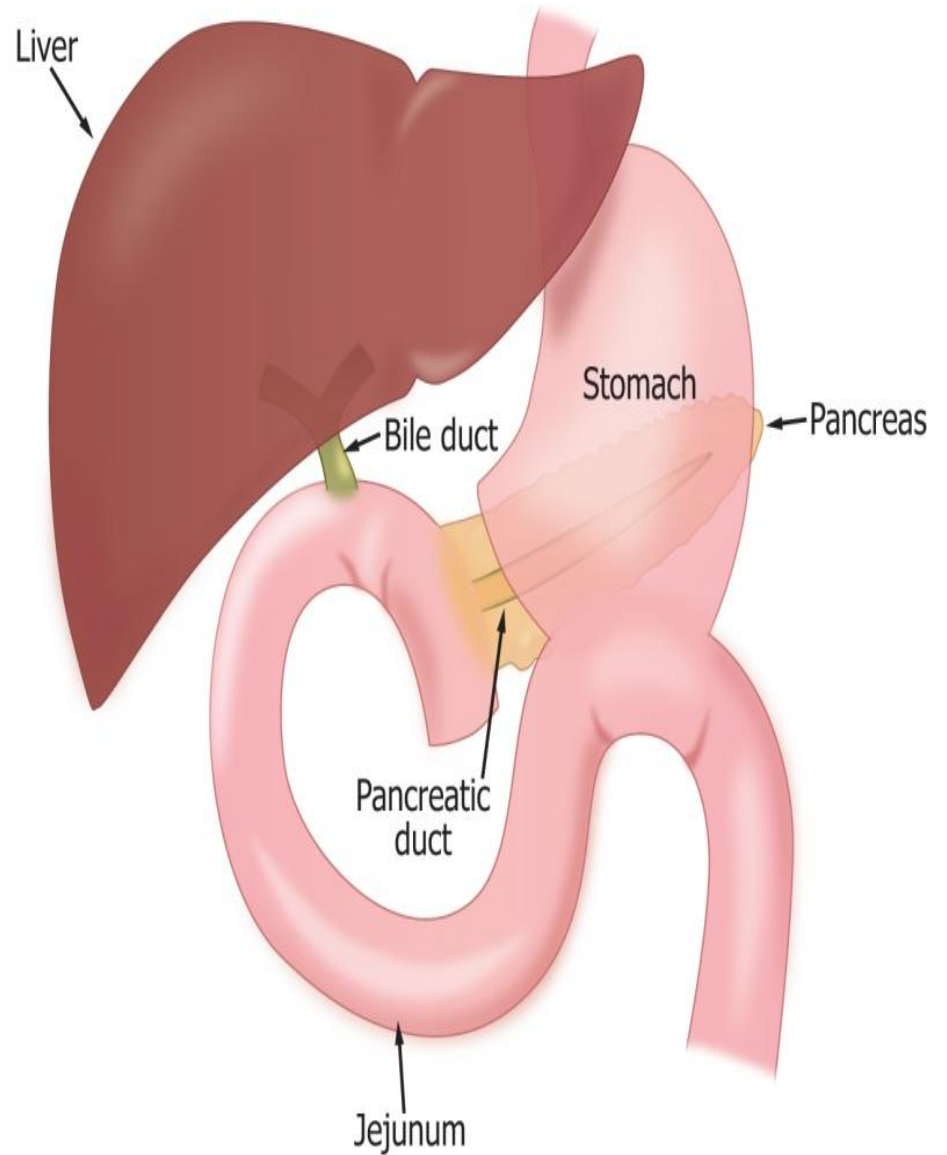
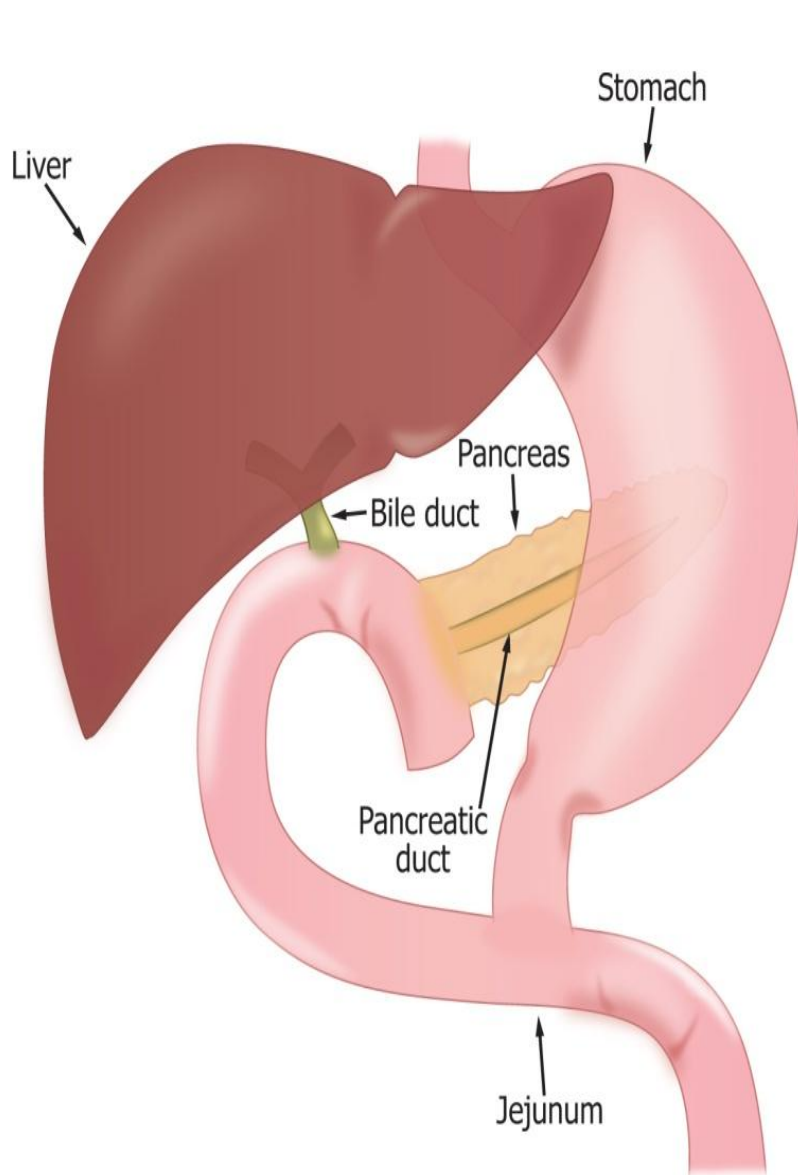




## pancreatic resection

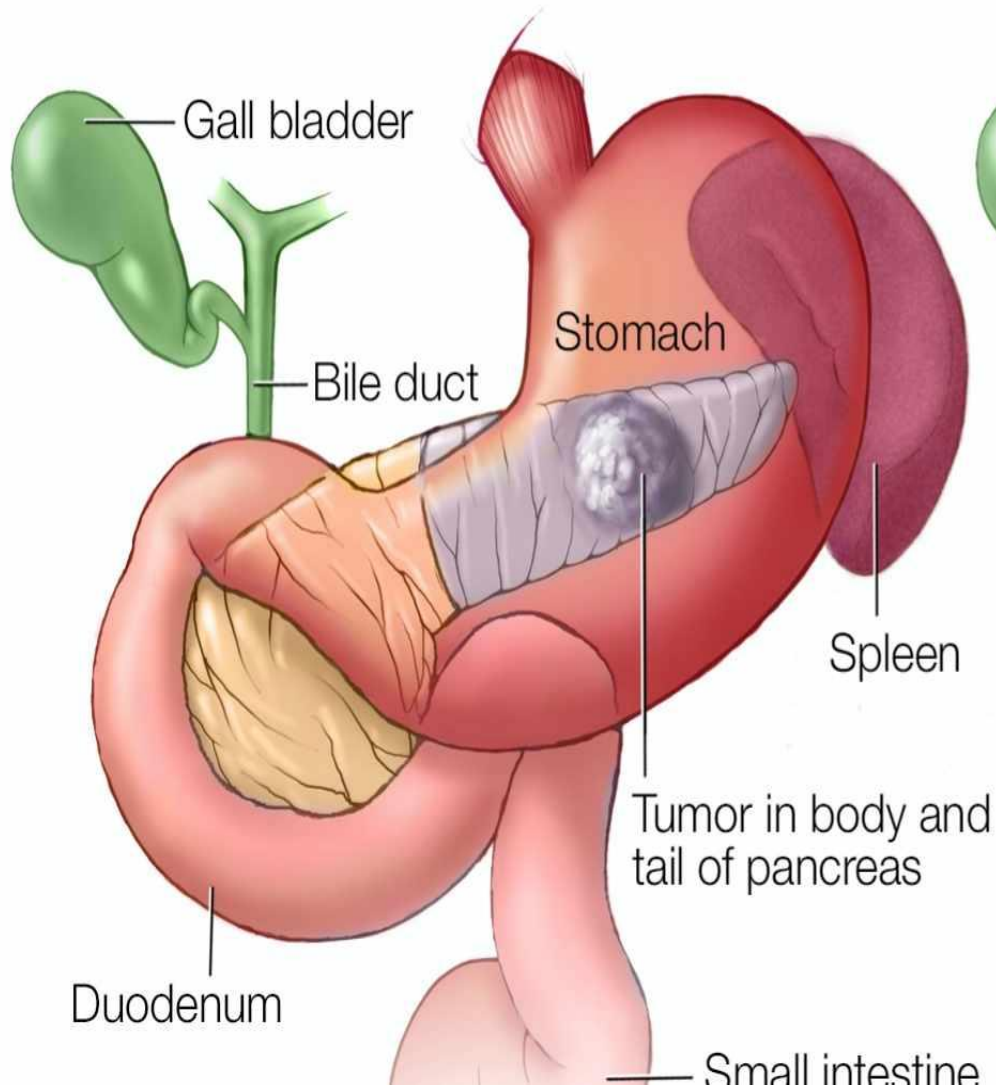


## Whipple procedure

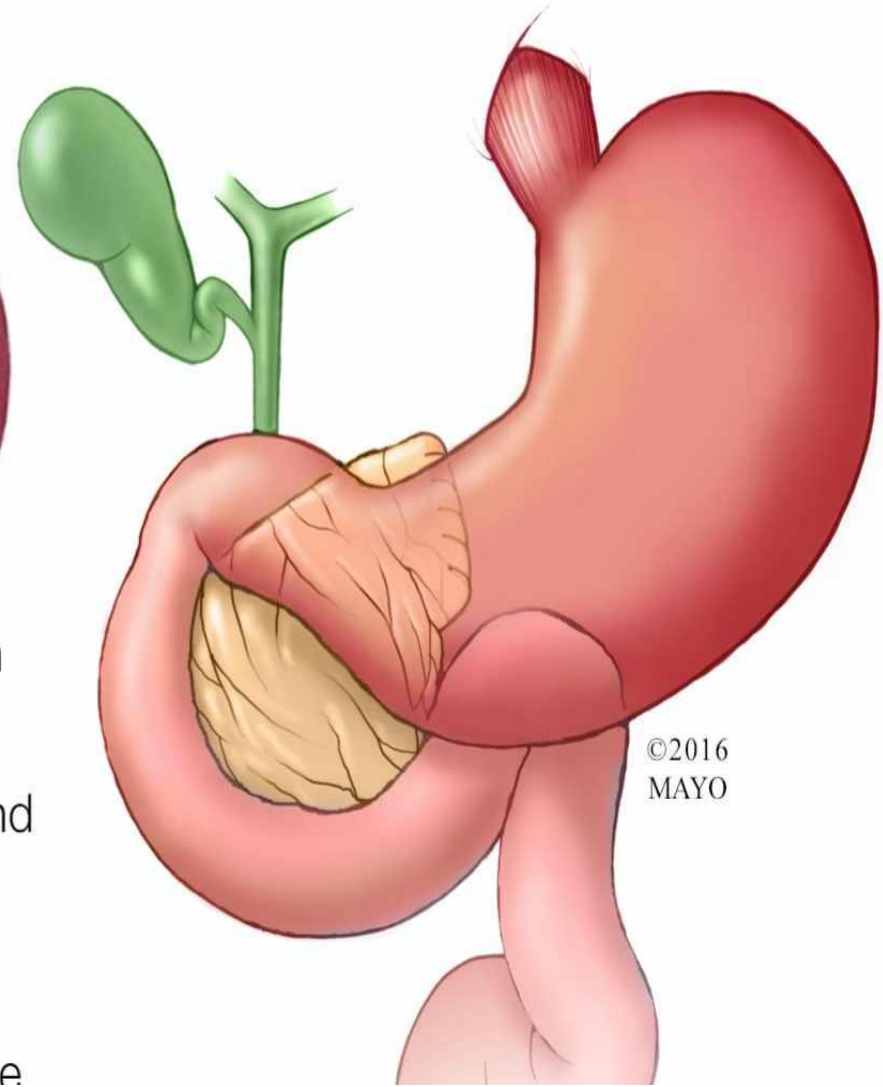


# Distal pancreatectomy

Before surgery



After surgery



# CYSTS

## Pseudocysts

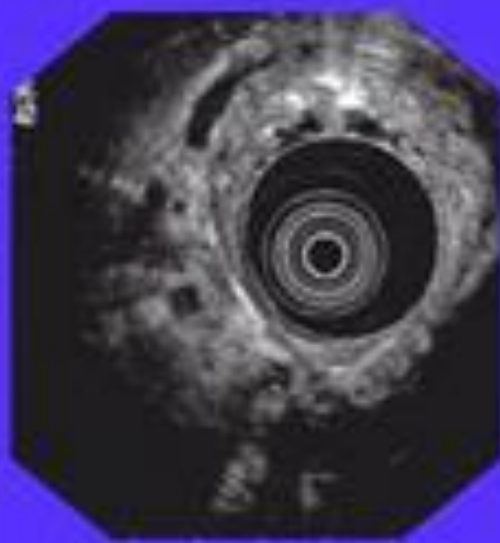
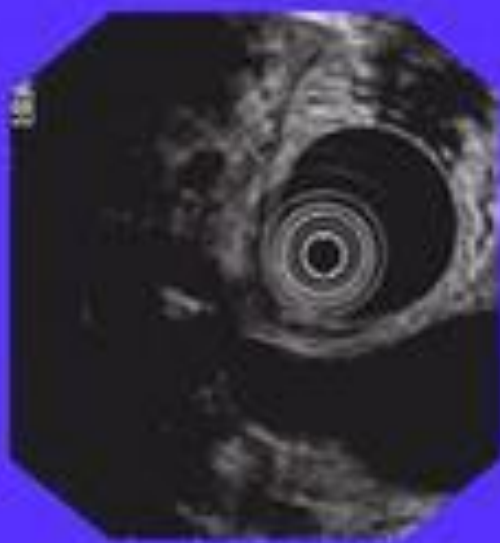
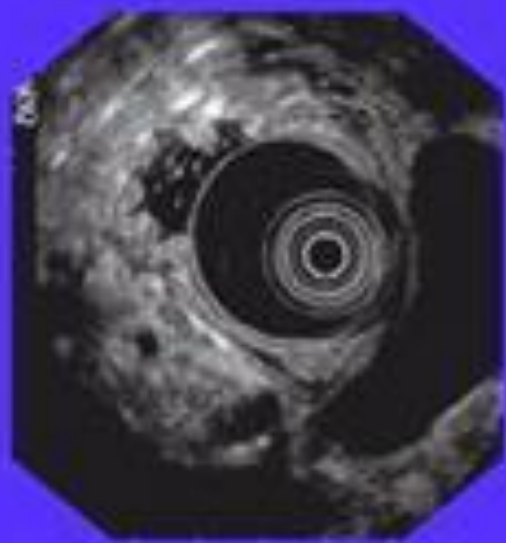
The fibrous wall of a **pseudocyst** surround a collection of pancreatic juice and necrotic or suppurative pancreatic tissue.

Location:

pancreas,  
transverse mesocolon or  
omentum,  
behind the pancreas,  
or mediastinum.



# Pancreatic Pseudocyst



# CYSTS

## Pseudocysts

### Clinical:

- persistent pain,
- Fever,
- Ileus,
- Nausea,
- Vomiting
- Anorexia
- Mass often change in size because partial drainage in the ductal system of the pancreas

Appearing 2-3 weeks after an attack of pancreatitis or trauma to the pancreas.

# **Characteristics of Cystic Neoplasms of the Pancreas versus Pseudocysts**

No history of pancreatitis

Internal septa and/or solid intracystic components seen on CT scan

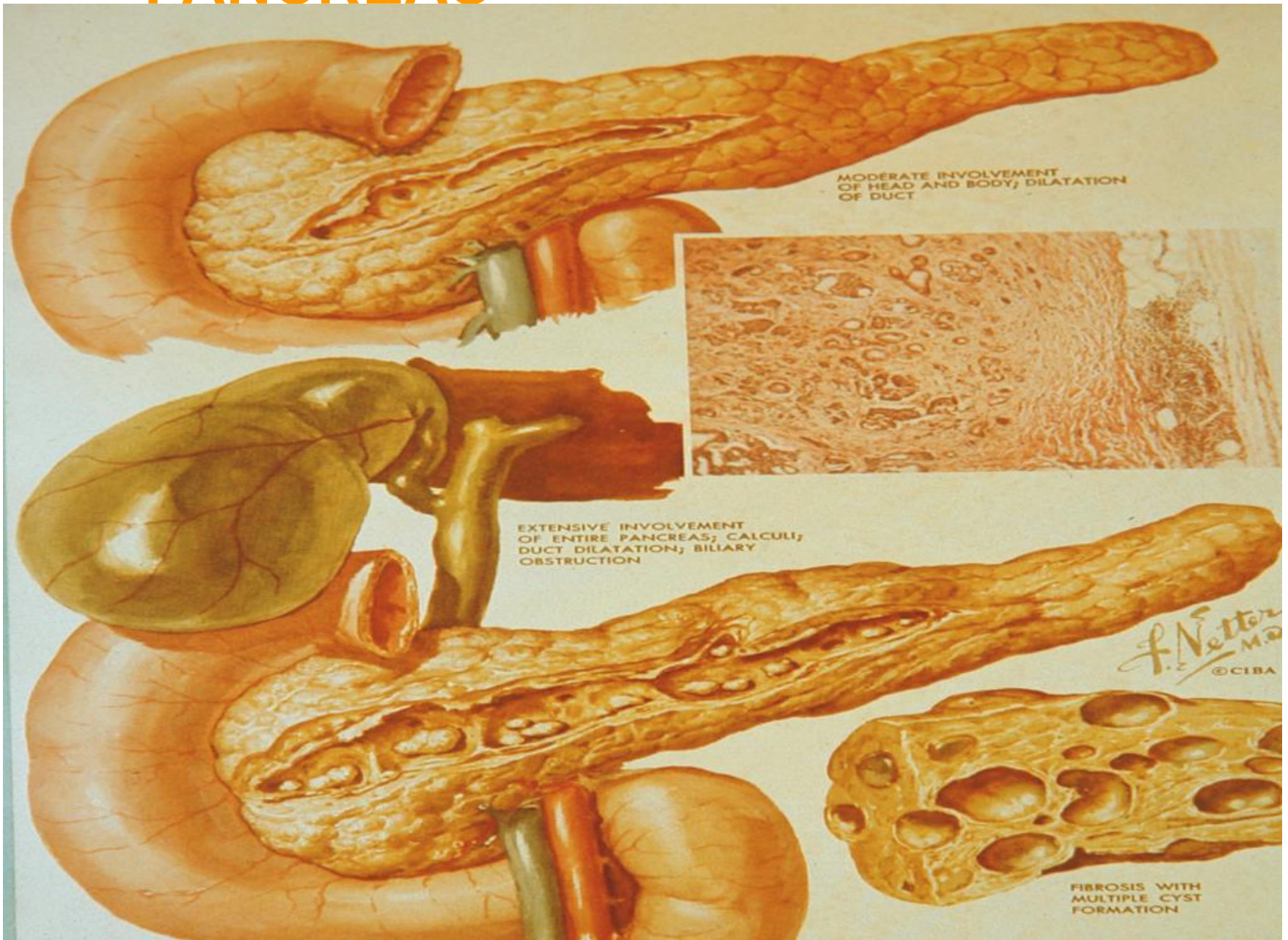
Calcification within the cyst or its wall

Recurrence or persistence of the cyst after treatment of any sort (i.e., surgical or nonsurgical)

If any of these features are present, suspicion that the cyst represents a neoplasm should be high.

Strong consideration should be given to surgical intervention, rather than nonoperative management (e.g., percutaneous drainage).

# PANCREAS





# Diagnosis

Radiologic examination barium study :extrinsic mass displacing the stomach superiorly anteriorly, the colon inferiorly, the duodenal loop may be widened. Pleural effusion and pulmonar manifestation.are common.

**Differential diagnosis:** Aortic aneurism

**Surgical treatment** is recommended for two reasons::

1. complications:secondary infections, severe hemorrhage, rupture into adjacent viscus or into free peritoneal cavity
  2. these lesions rarely resolve once a thick fibrous wall has developped
- If the mass is developped during an attack of acute pancreatitis, it should be observed for several weeks.

## Procedures:

- Extirpation, ideal method of therapy
- Simple external drainage
- Marsupialisation
- Internal drainage is the best treatment: **transgastric cystogastrostomy** when the lesion is aderent to the stomach or **transduodenal cystoduodenostomy** when it is located In the head of the pancreas

# CYSTS

## True pancreatic cysts

True cysts that involve the parenchyma or ducts of the pancreas are:

- far less common than pseudocysts,
- usually smaller than pseudocysts,
- more concentric and
- the contained fluid resembles normal pancreatic secretion.

True cysts of the pancreas include:

- retention cysts,
- neoplastic cysts and
- infectious cysts.

# CYSTS

## True pancreatic cysts

### Retention cysts

Retention cysts are also referred to as non continuous, distension or secretory cysts and are associated with chronic pancreatitis.

Their size depends on the atrophy associated with the underlying chronic pancreatitis.

Each cyst is lined by the duct epithelium, although with increasing size and age the epithelium may become flat and atrophic.

The cysts will be continuous with the ductal system in the early phases but lose ductal communication as their size increases.

The symptoms are identical to those of patients with chronic pancreatitis.

## CYSTS

### **True pancreatic cysts**

Ultrasound or CT scan usually demonstrates the cyst.

**Management** of retention cysts is related to management of the underlying chronic pancreatitis.

- Distal pancreatectomy, with splenectomy
- Simple resection in the head
- Pancreatico-duodenal resection when the common bile duct is involved



## Neoplastic cysts.

Cystadenoma and cystadenocarcinoma are the benign and malignant variants of an unusual primary neoplasm that involves the pancreas. These tumors are most often located in the head or tail of the pancreas and are not related to acute or chronic pancreatitis and tend to occur in the elderly.

Ultrasound or CT scanning are the usual **diagnostic procedures.**

Angiography shows vascular displacement without tumor vessels.

**Management** is by surgical resection.

# Neoplasms

The most common neoplasms involving the pancreas are primary malignant tumors arising from the duct epithelium.

**Adenocarcinoma** of the pancreatic head

**Subjective findings.** The most common early symptom is progressive jaundice, pain occurs in the epigastrium or back is typical of pancreatitis and may be caused by regional inflammation from tumor obstruction or direct neural involvement.

**Objective findings.** If the gallbladder does not appear diseased and fibrotic, it may be distended and palpable in more than one third of cases, indicating an obstruction distal to the cystic duct (Courvoisier sign).

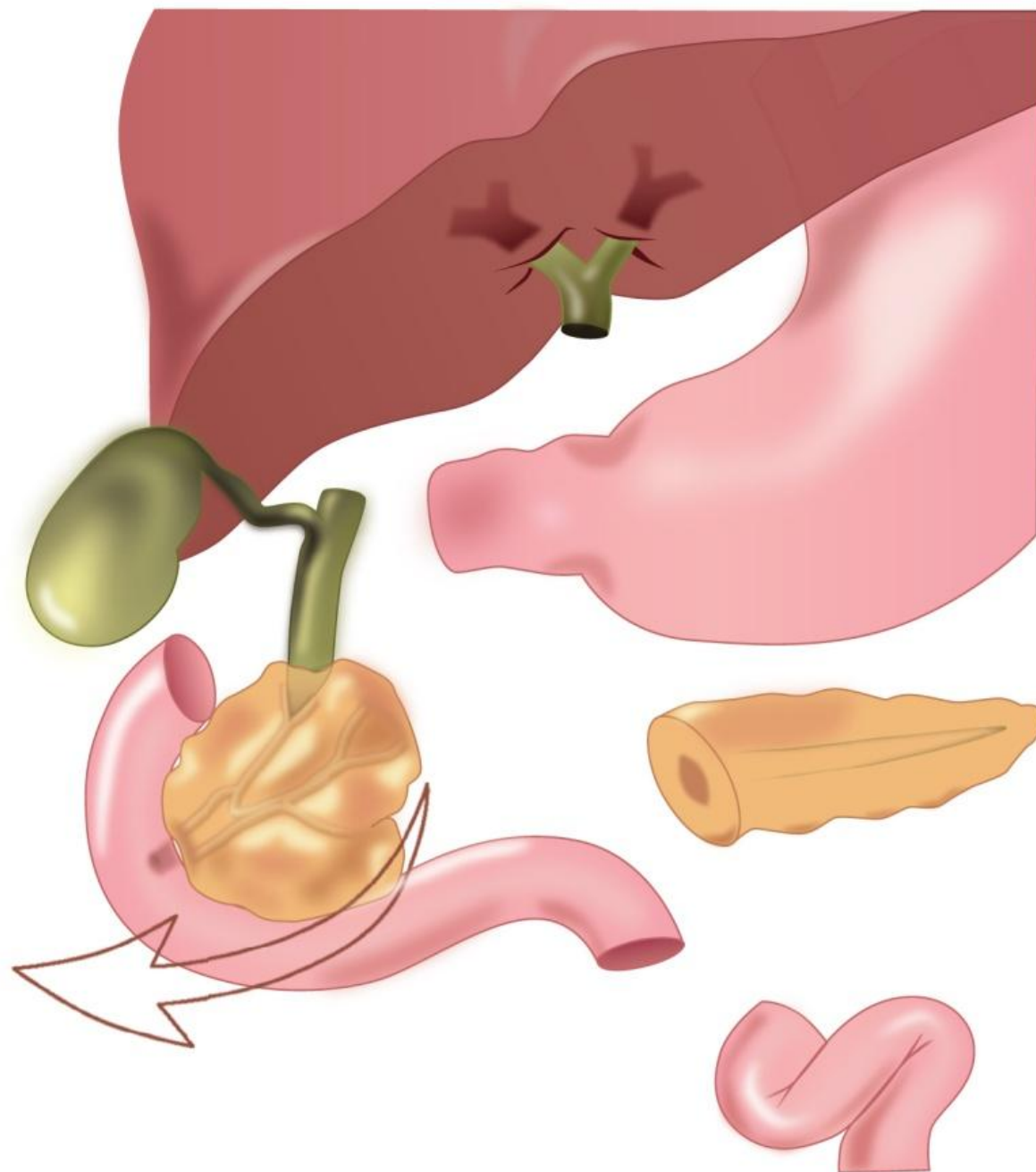
**Diagnostic plan.** The differential diagnosis of carcinoma includes any cause of extrahepatic cholestasis. Total and conjugated bilirubin levels are maximally elevated and the serum alkaline phosphatase level is disproportionately increased compared with the transaminase values. The best procedure to differentiate hepatocellular from extrahepatic cholestasis is abdominal ultrasound scanning, the technique is superior to a CT scan.

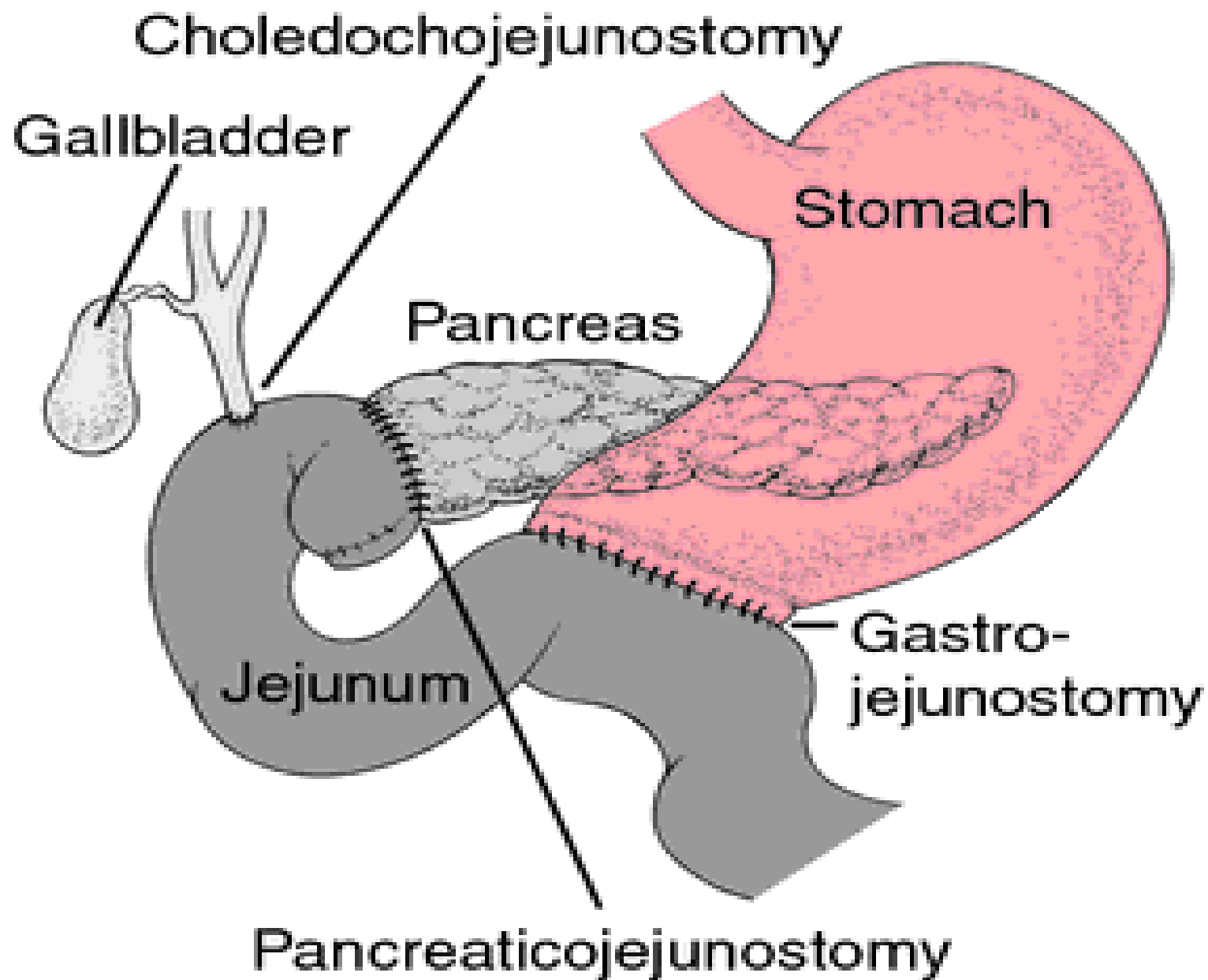
**Management plan.** Alternative management includes radical resection, palliative surgery and nonoperative biliary drainage procedures to relieve the jaundice. Complications after radical pancreatic resection include bile leak, postoperative bleeding and pancreatic fistula. The resective techniques currently include duodenectomy or with partial pancreatectomy (Whipple's procedure).

The Whipple Procedure, or pancreaticoduodenectomy, is the most commonly performed surgery to remove tumors in the pancreas.

In a standard Whipple procedure, the surgeon removes:

- the head of the pancreas,
- the gallbladder,
- part of the duodenum,
- a small portion of the stomach including pylorus,
- and the lymph nodes near the head of the pancreas.







**Pancreatic cancer** is the second most common tumor of the gastrointestinal tract and the fifth leading cause of cancer-related death in adults.

The disease is difficult to diagnose especially in the early stages. Most pancreatic cancers arise in the head of the pancreas, often causing bile duct obstruction that results in clinically evident jaundice.

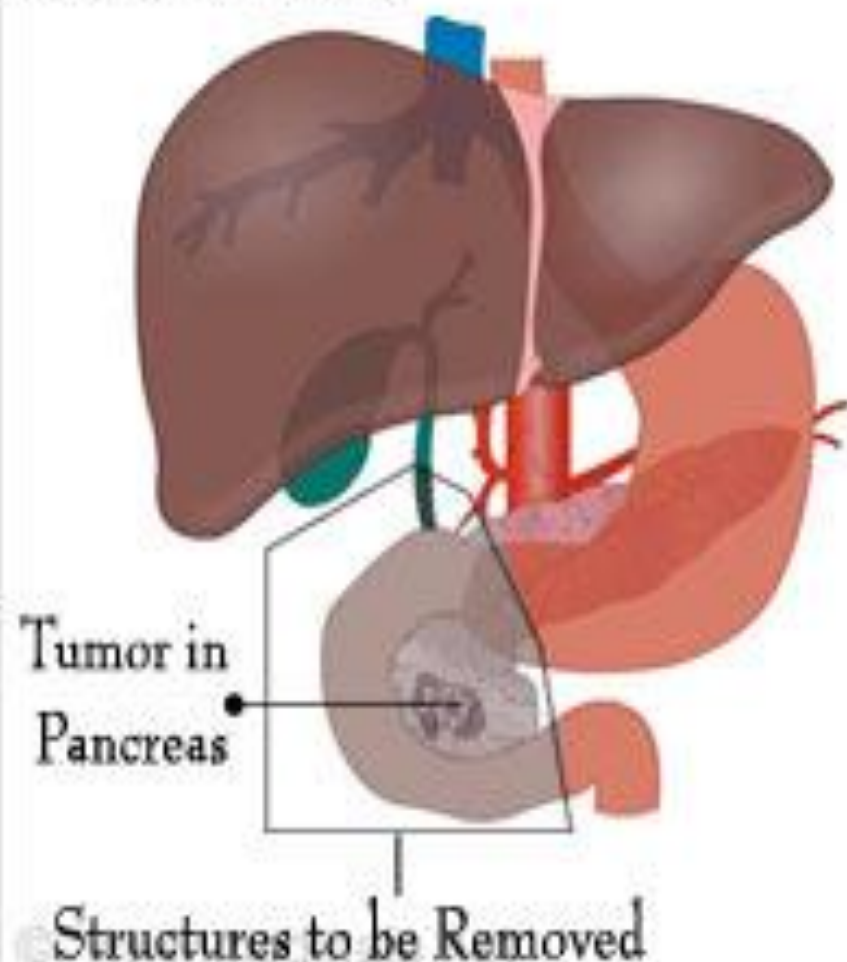
Cancers that arise in either the body or the tail of the pancreas are insidious in their development and often far advanced when first detected. Most pancreatic cancers are adenocarcinomas which usually originate from the pancreatic duct cells.

**Surgical resection** remains the only potentially curative approach, although multimodality therapy that includes innovative systemic agents and often radiation therapy is available.

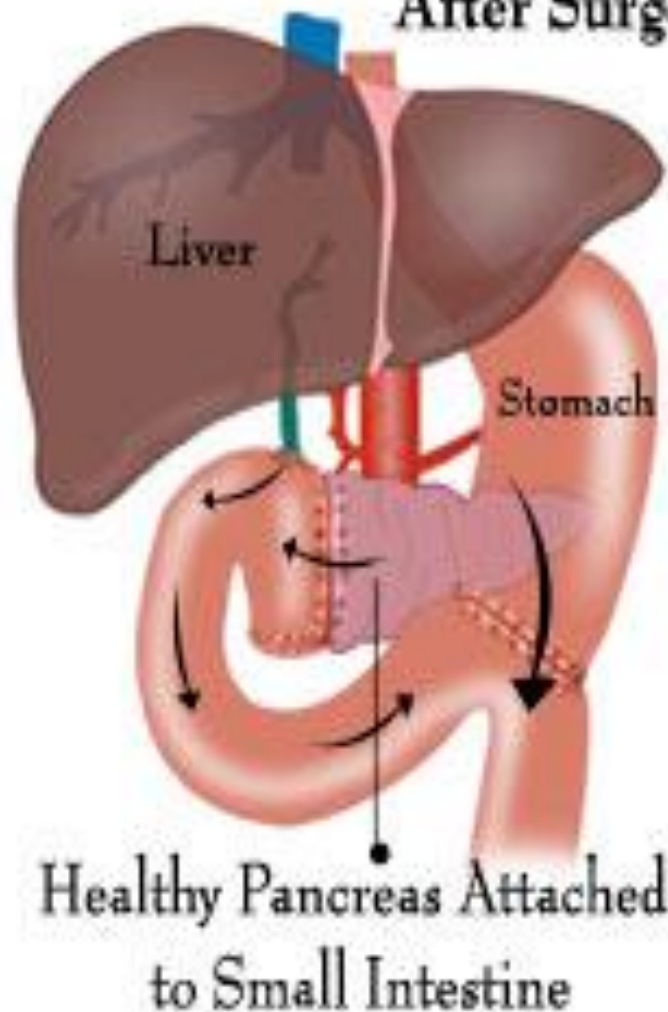
Staging of exocrine pancreas cancers depends on their size and extent of the primary tumor.

# Whipple's Surgical Procedure

Before Surgery

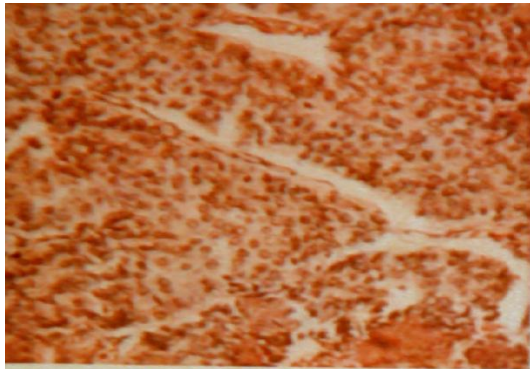


After Surgery

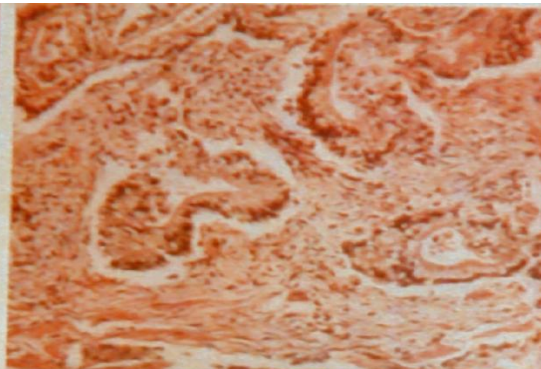




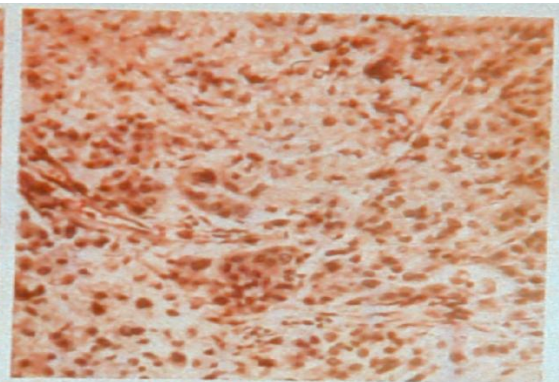
# PANCREAS



MEDULLARY CARCINOMA



CARCINOMA WITH DUCTLIKE STRUCTURES



ANAPLASTIC CARCINOMA

## METASTASES FROM PANCREAS

### MOST COMMON SITES:

1. REGIONAL NODES
2. LIVER
3. LUNG AND PLEURA
4. INTESTINE
5. PERITONEUM

### MODERATELY COMMON SITES:

6. ADRENAL
7. BONE
8. DIAPHRAGM
9. GALLBLADDER
10. KIDNEY

### OCCASIONAL SITES:

11. HEART
12. MEDIASTINUM
13. BLADDER
14. OVARY
15. SUPRA-CLAVICULAR NODES
16. MUSCLE OR SUBCUTANEOUS TISSUE

## METASTASES TO PANCREAS

### COMMON SOURCES:

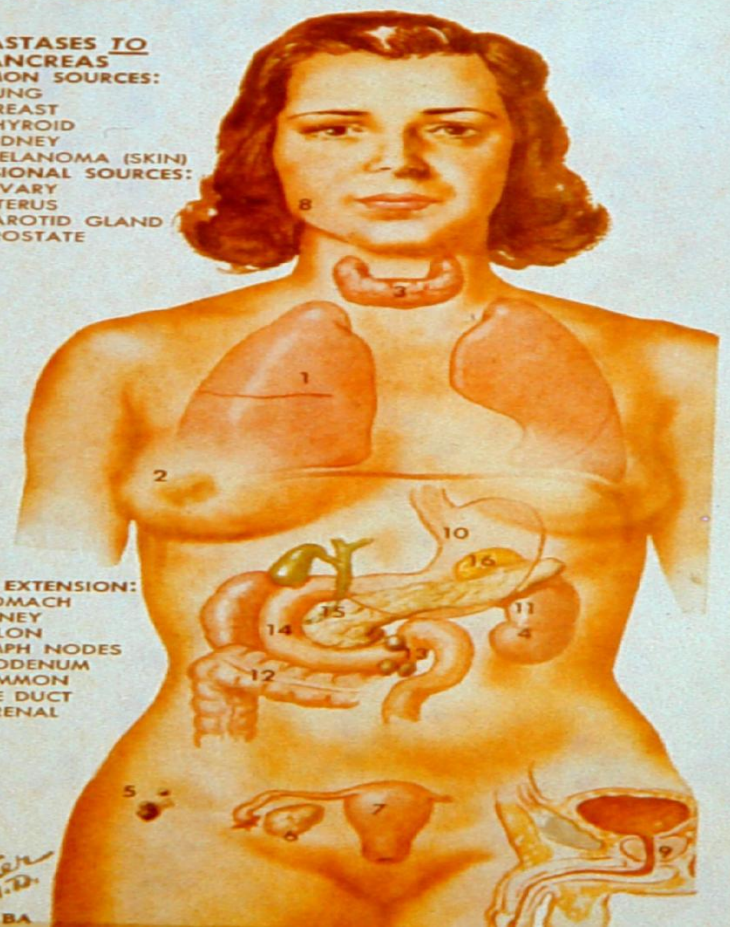
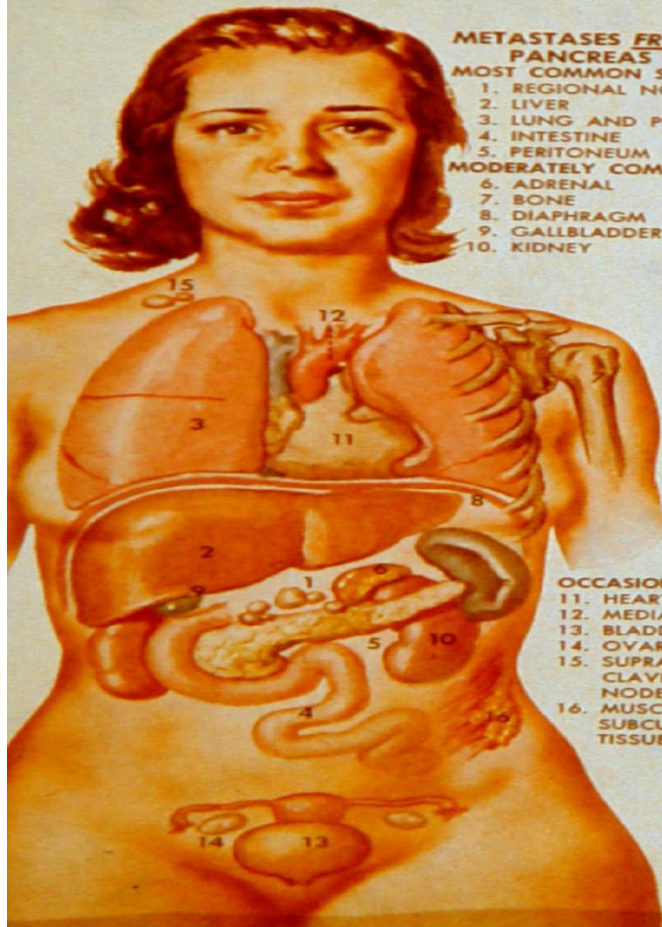
1. LUNG
2. BREAST
3. THYROID
4. KIDNEY
5. MELANOMA (SKIN)

### OCCASIONAL SOURCES:

6. OVARY
7. UTERUS
8. PAROTID GLAND
9. PROSTATE

### DIRECT EXTENSION:

10. STOMACH
11. KIDNEY
12. COLON
13. LYMPH NODES
14. DUODENUM
15. COMMON BILE DUCT
16. ADRENAL



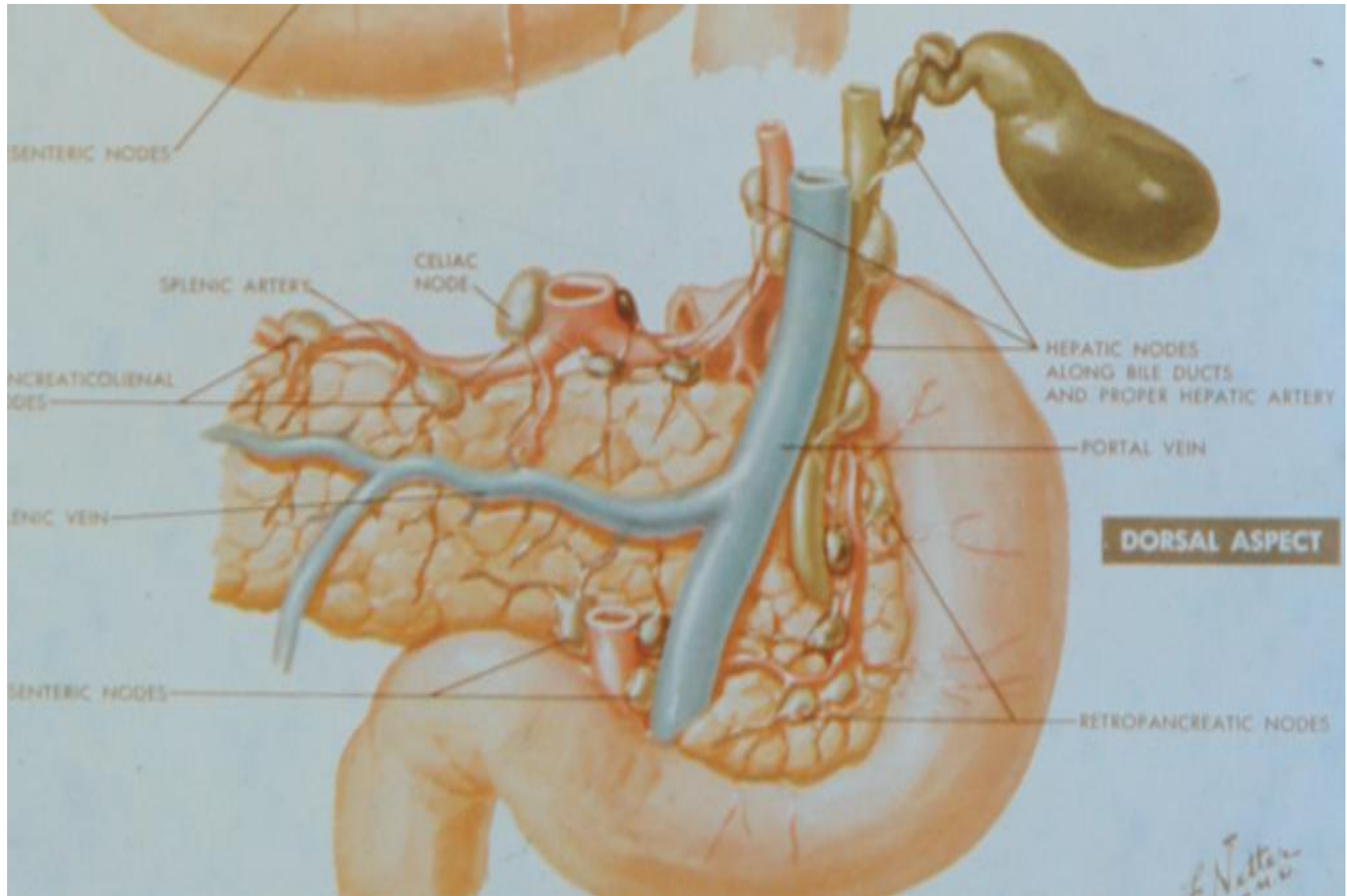
Pancreatic tumors may be treated surgically by subtotal resection of the pancreas together with adjacent organs.

Malignant neoplasms of the duodenum, terminal common duct, and ampulla of Vater can be removed in this manner, and patients suffering one or another of these serious tumors either cured or provided substantial palliation.

Occasionally gastrectomy or right colectomy is combined with pancreato-duodenectomy for cancers primary in the stomach or colon which have extended to the stomach, duodenum, colon, and pancreas in a localized malignant mass.



# PANCREAS





# PANCREAS

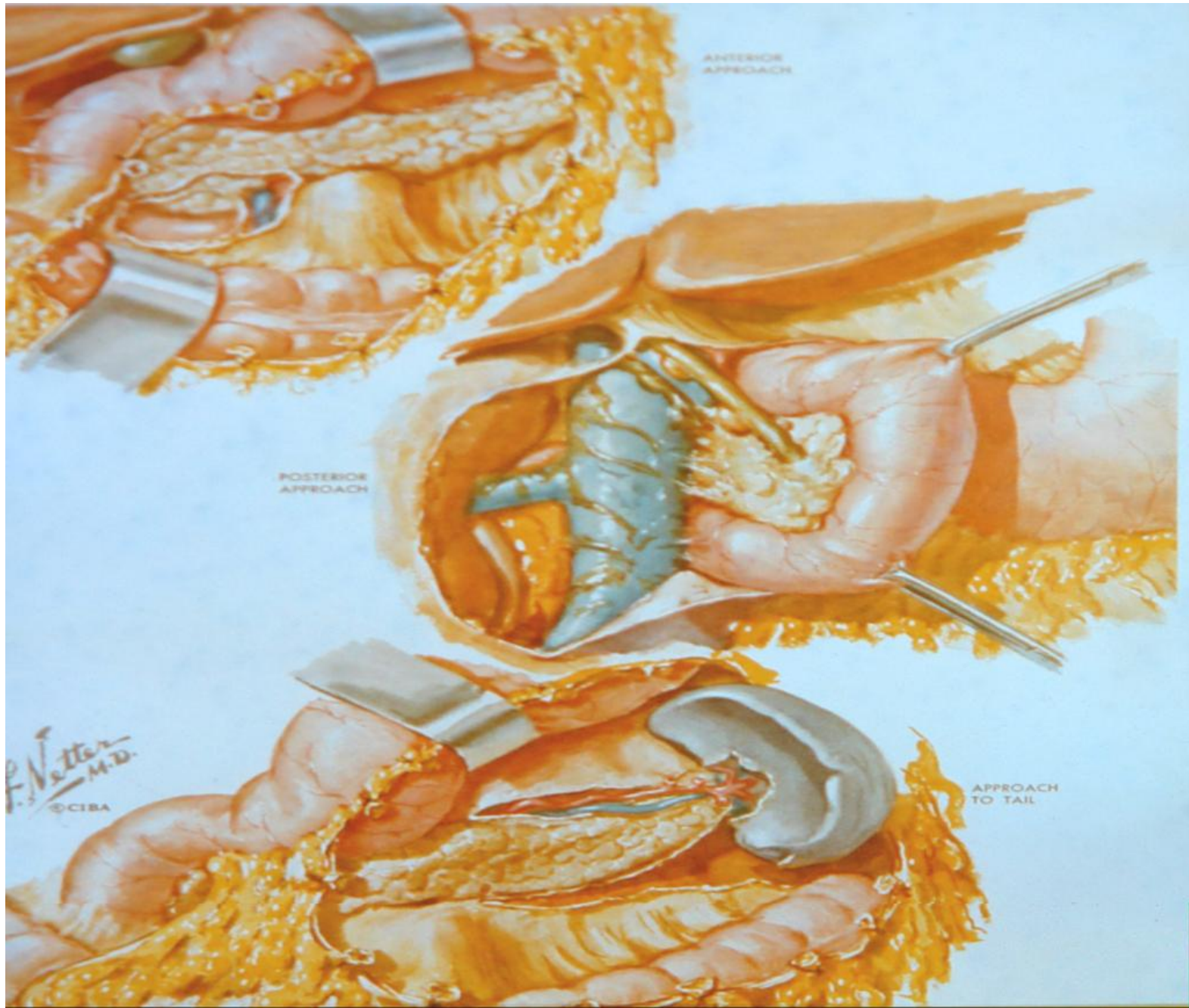
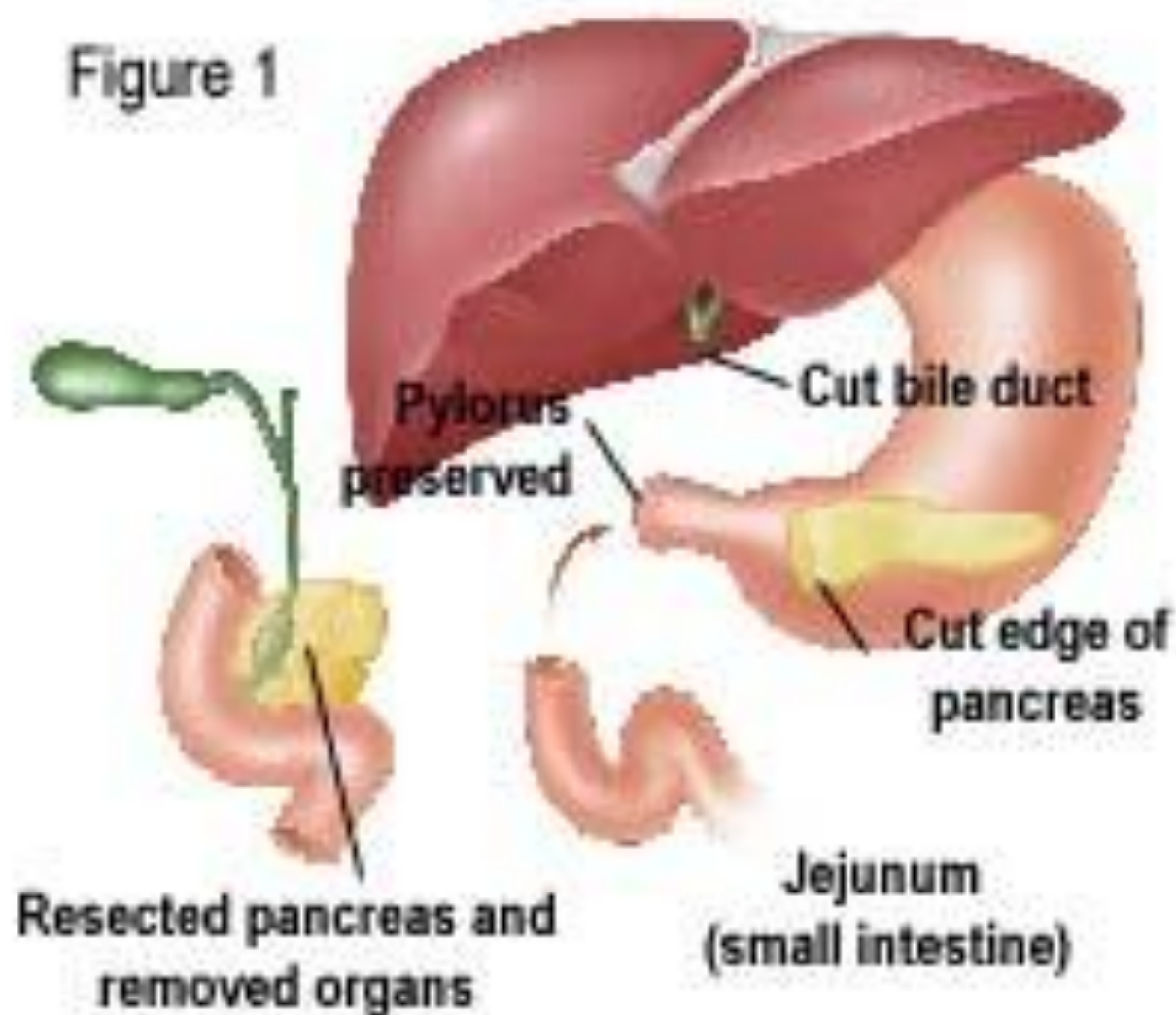
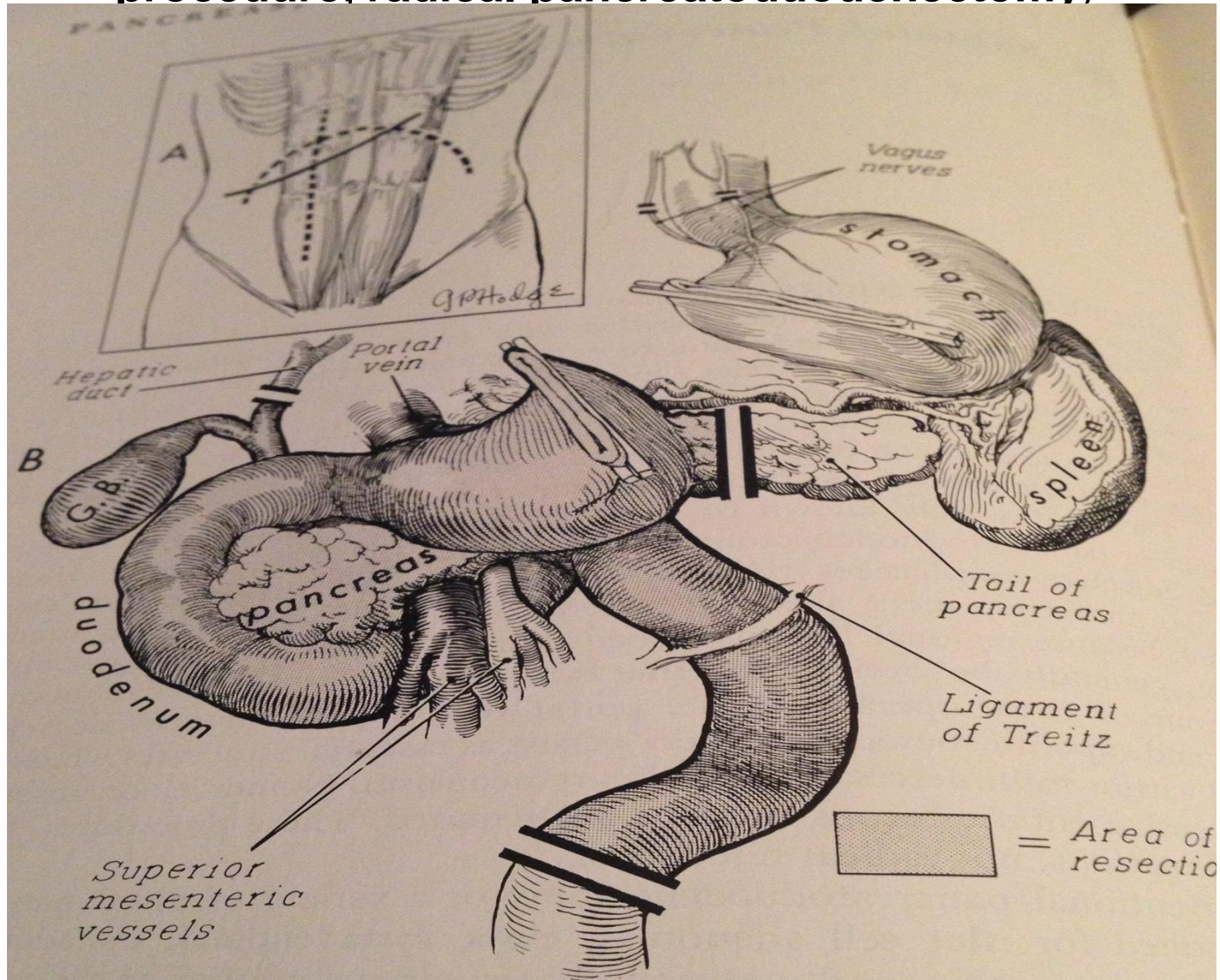


Figure 1



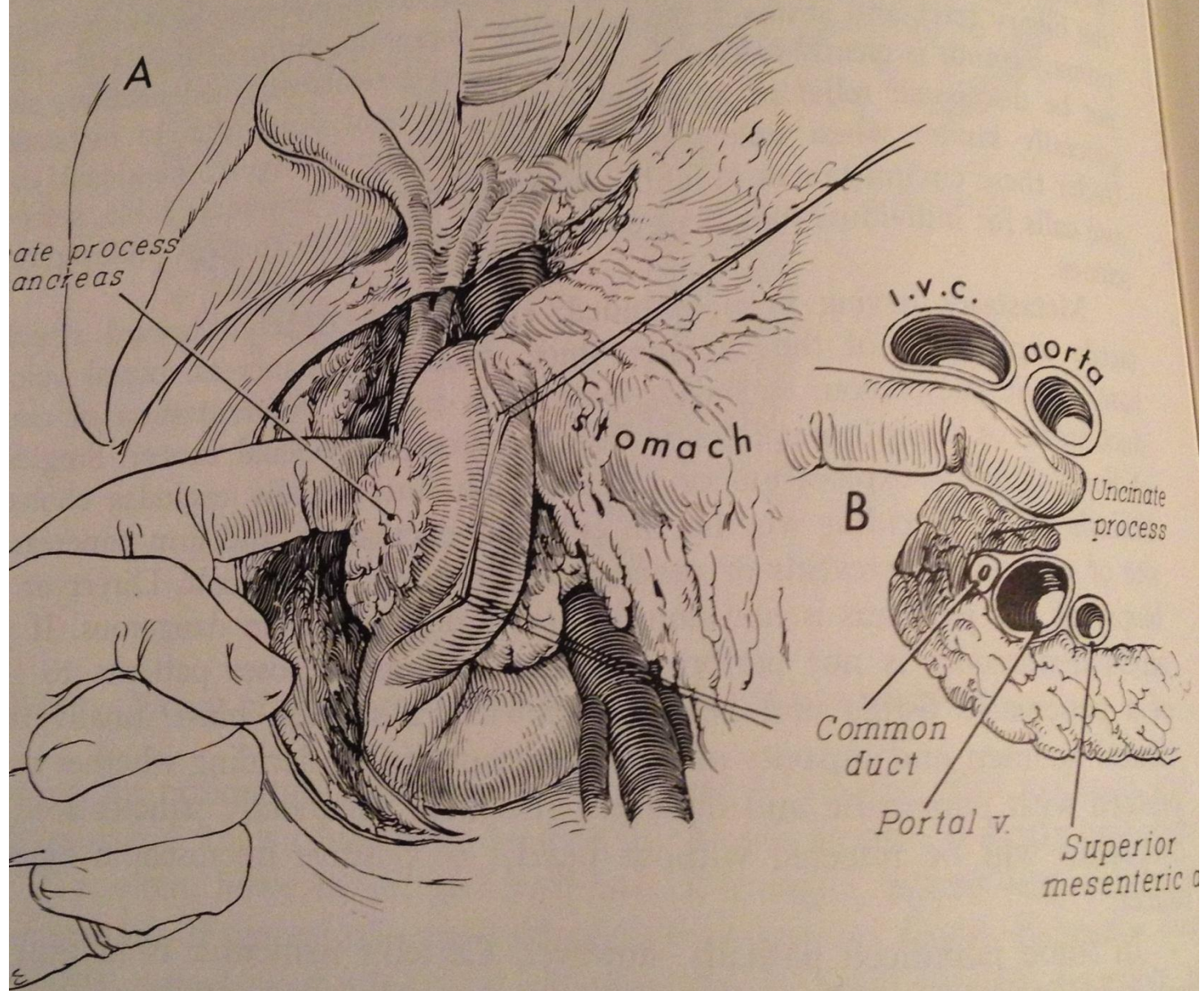


# Subtotal pancreatectomy for Pancreaticoduodenal cancer (Whipple procedure, radical pancreatoduodenectomy)



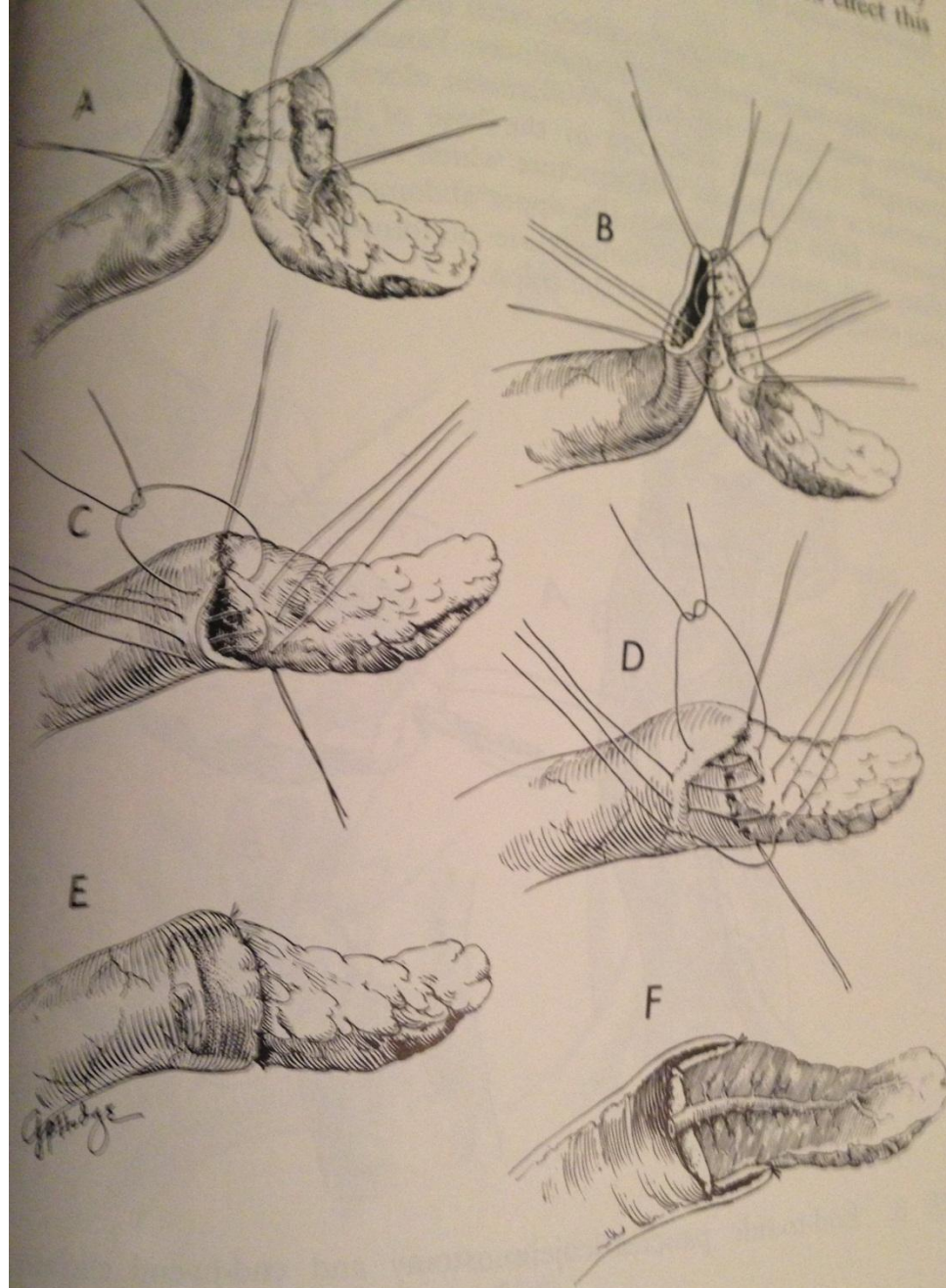
Extent of pancreatoduodenectomy (Whipple procedure).





E 2. Mobilization of the duodenum and head of the pancreas.





7. End-to-end pancreaticojejunostomy (invagination).



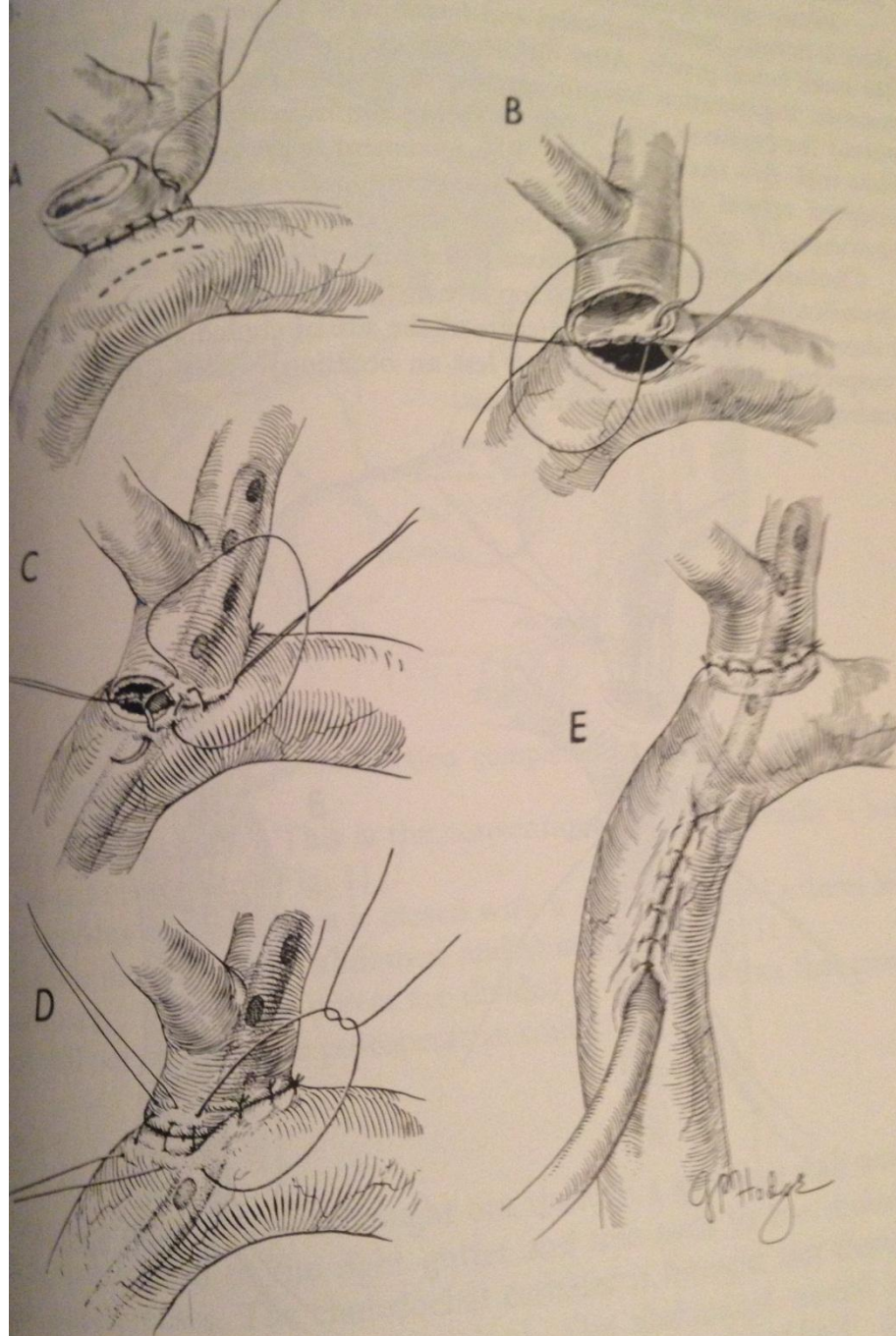
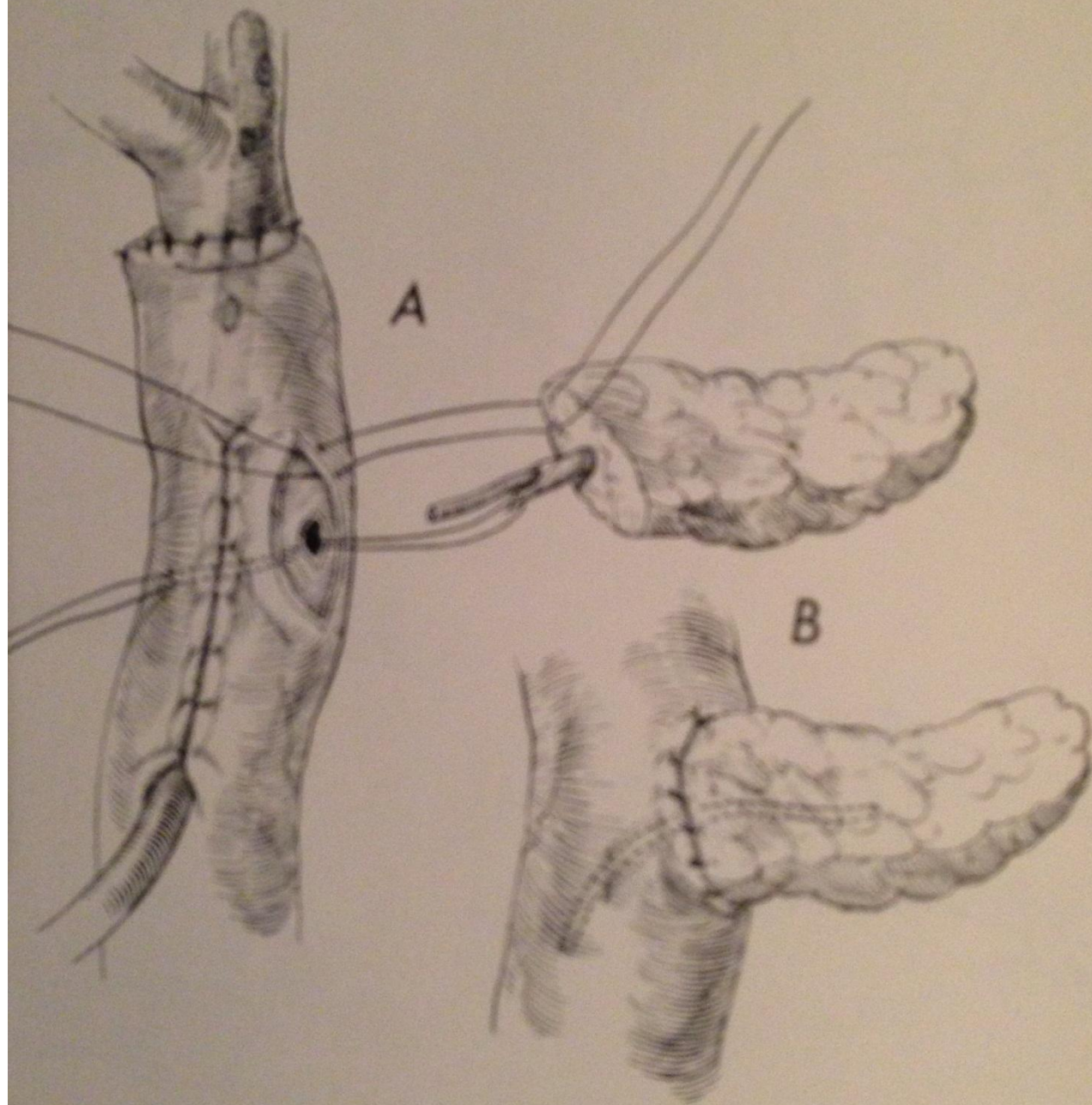


FIG. 9. End-to-side choledochojejunostomy.



End-to-side pancreaticojejunostomy and end-to-end choledochojejunostomy