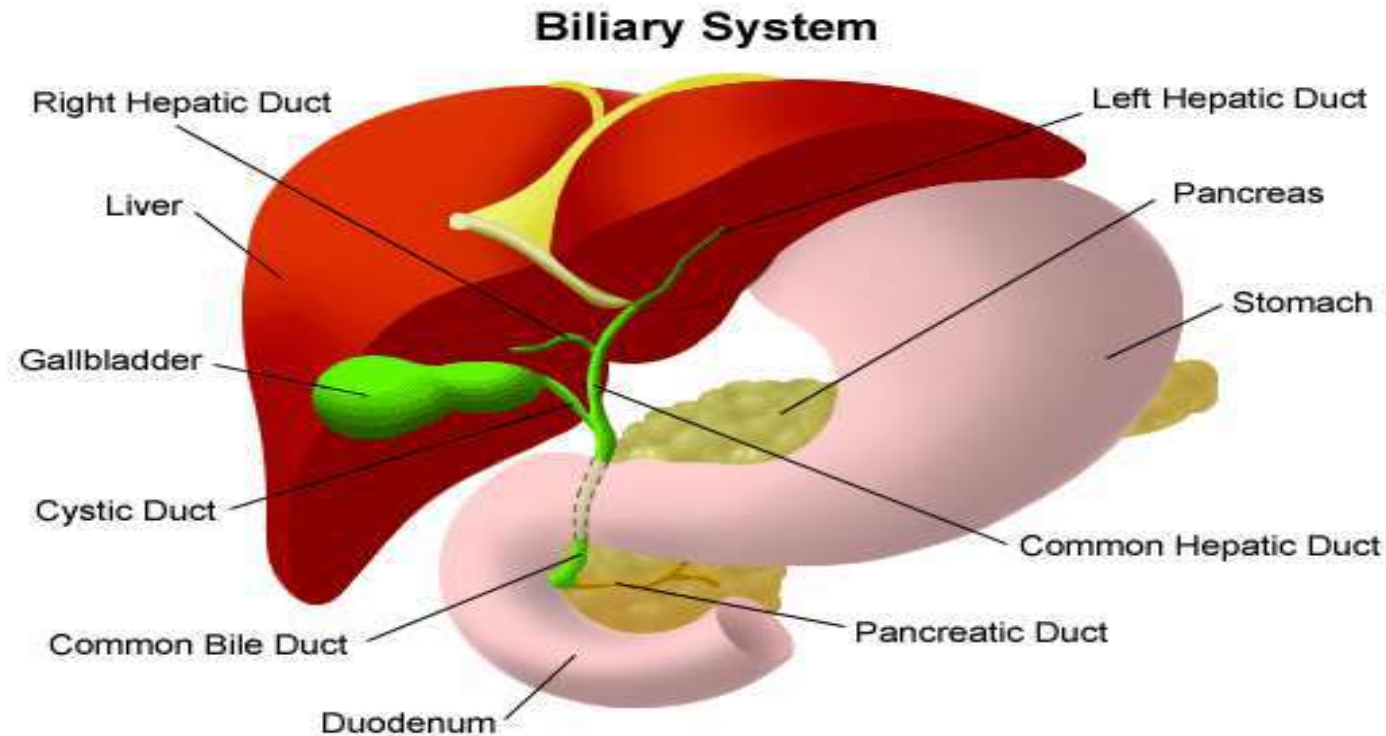


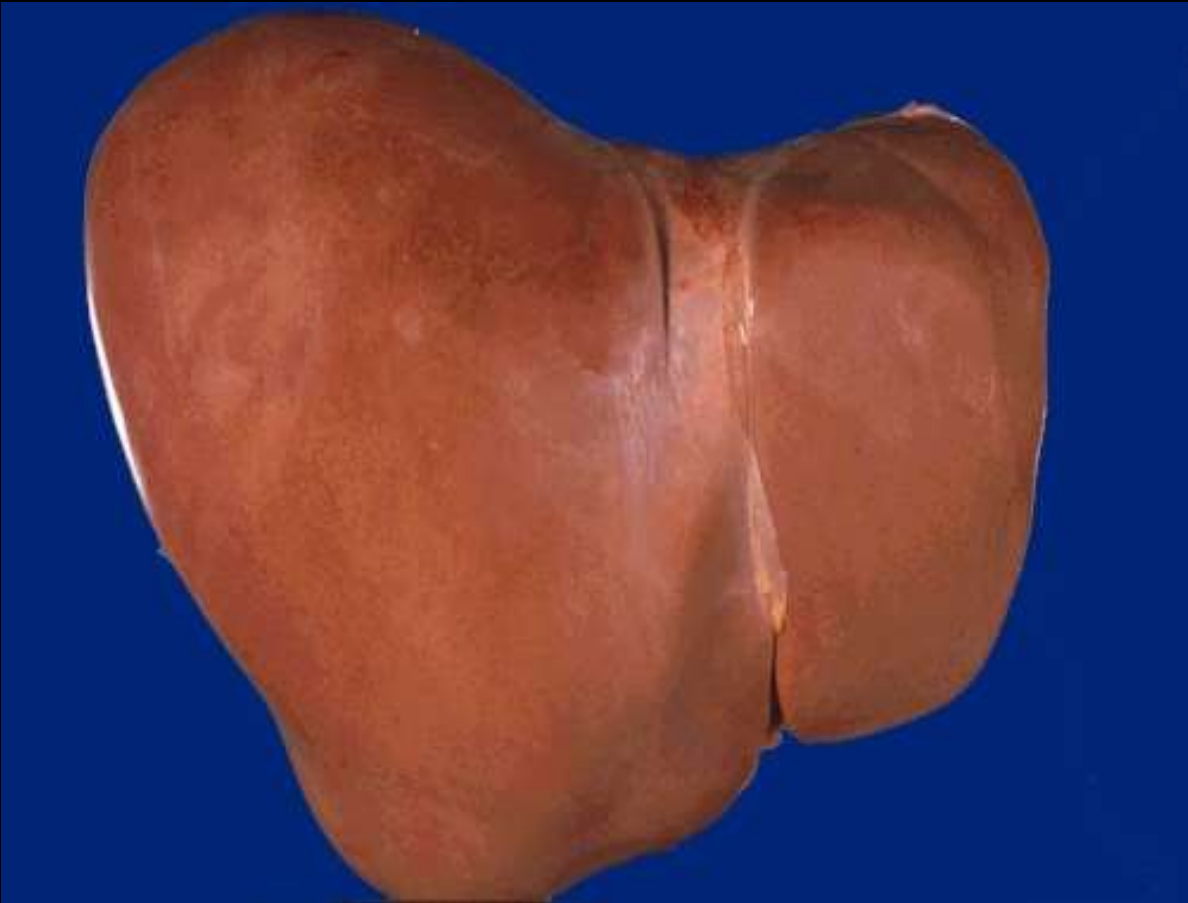
Hepatobiliary system

practical

Dr. Methaq Mueen

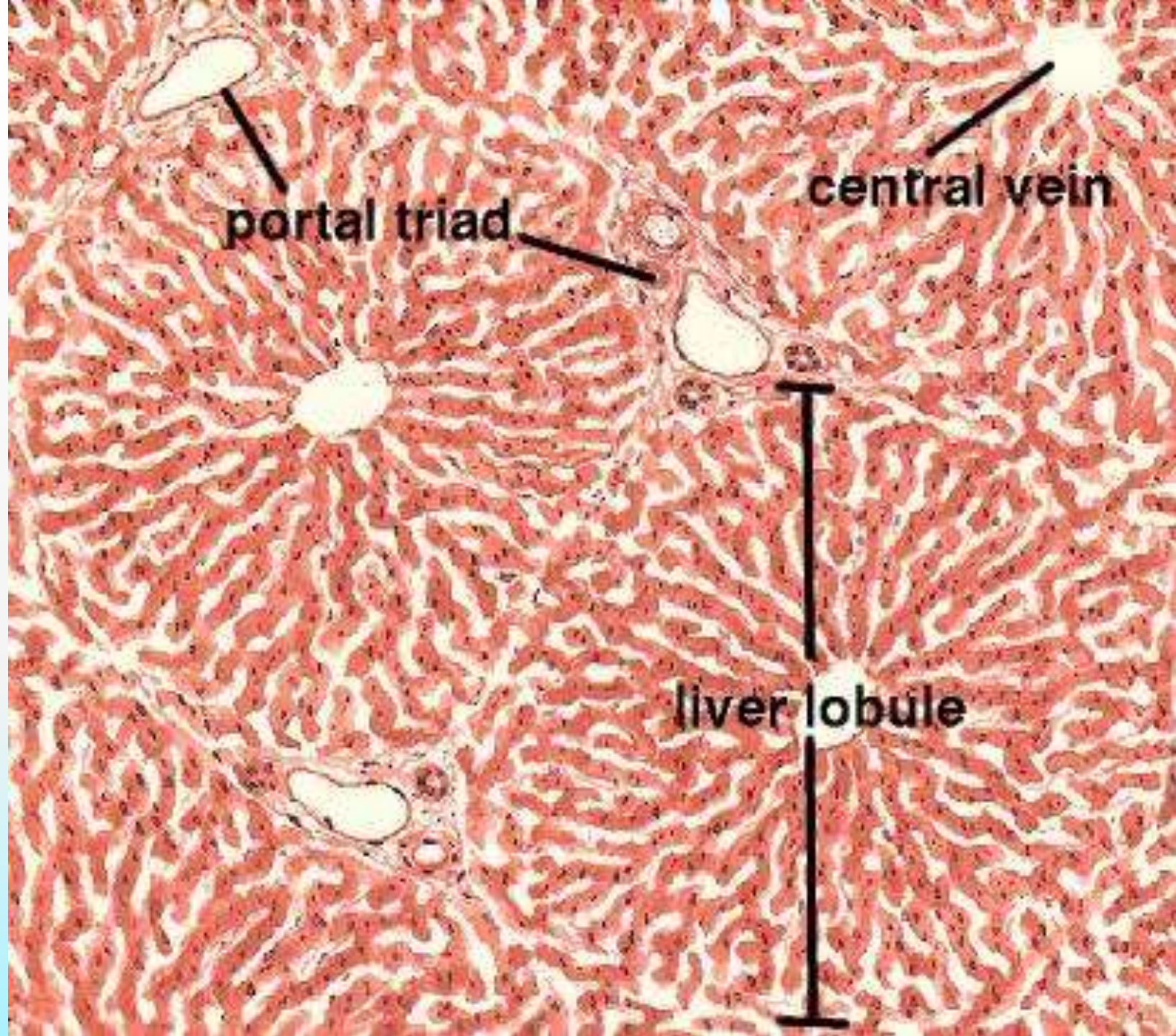


NORMAL ANATOMY



- External surface
- -red - brown
- - smooth
- - soft

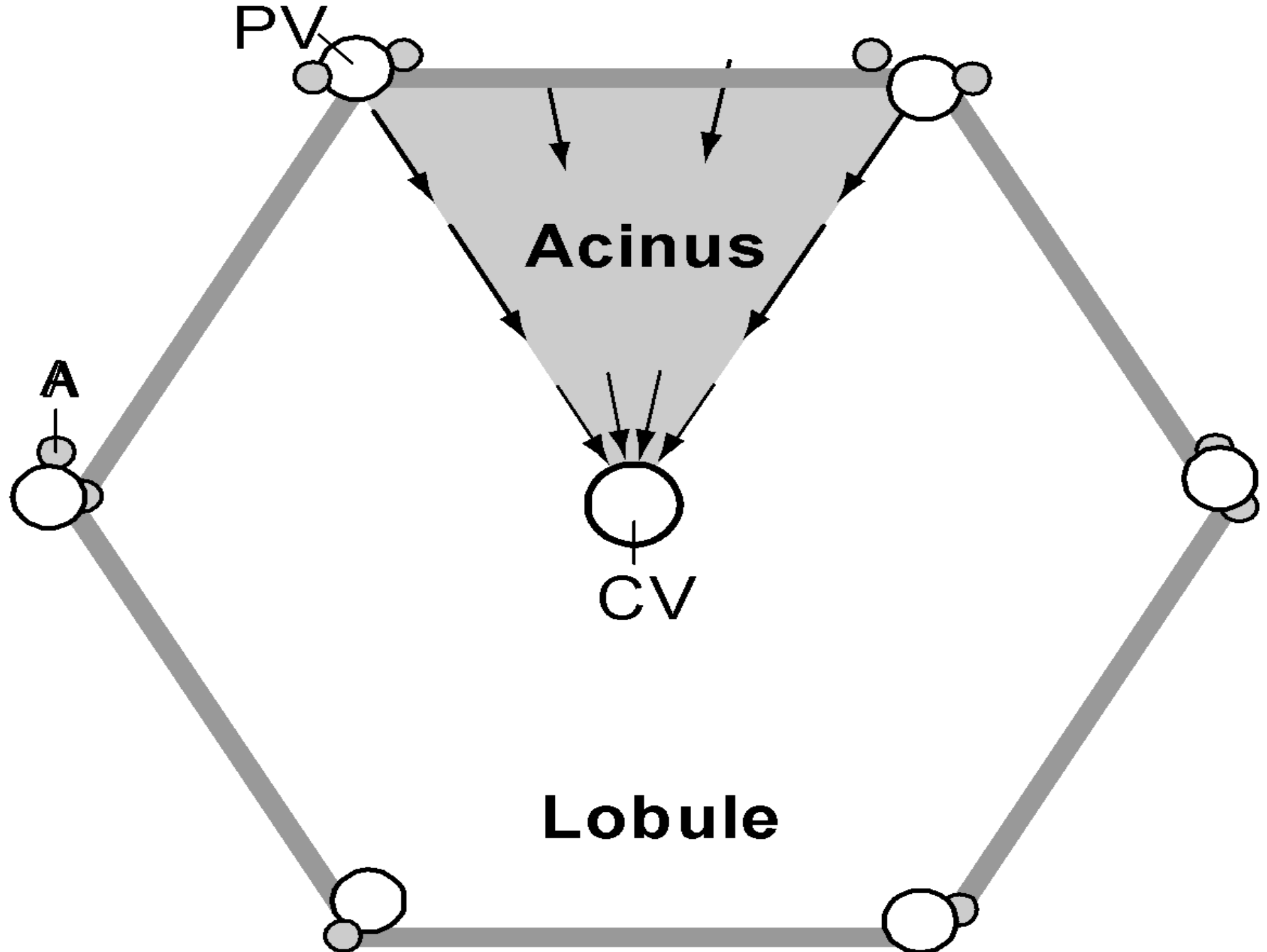
Normal liver: 1200 – 1600g

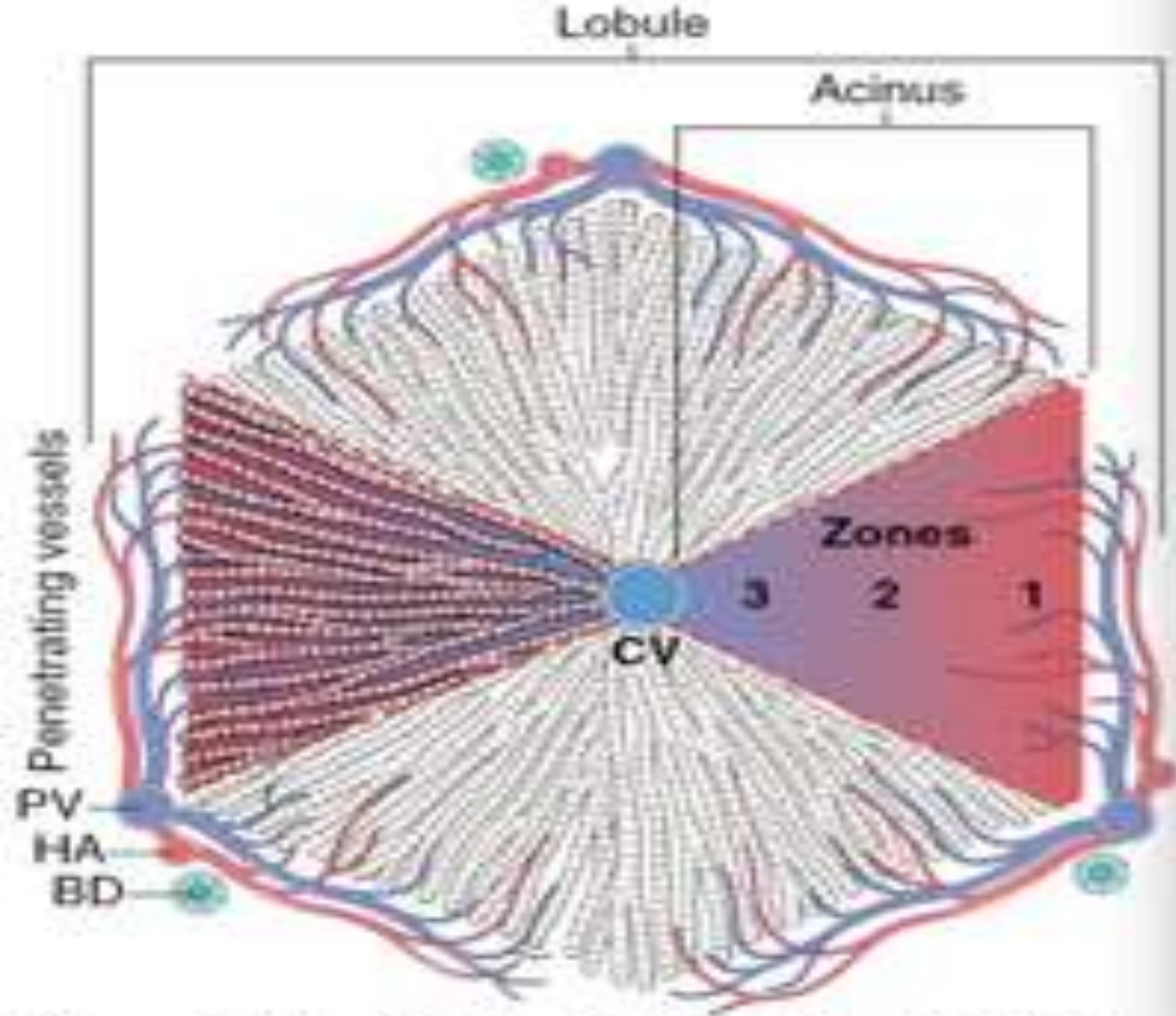


N

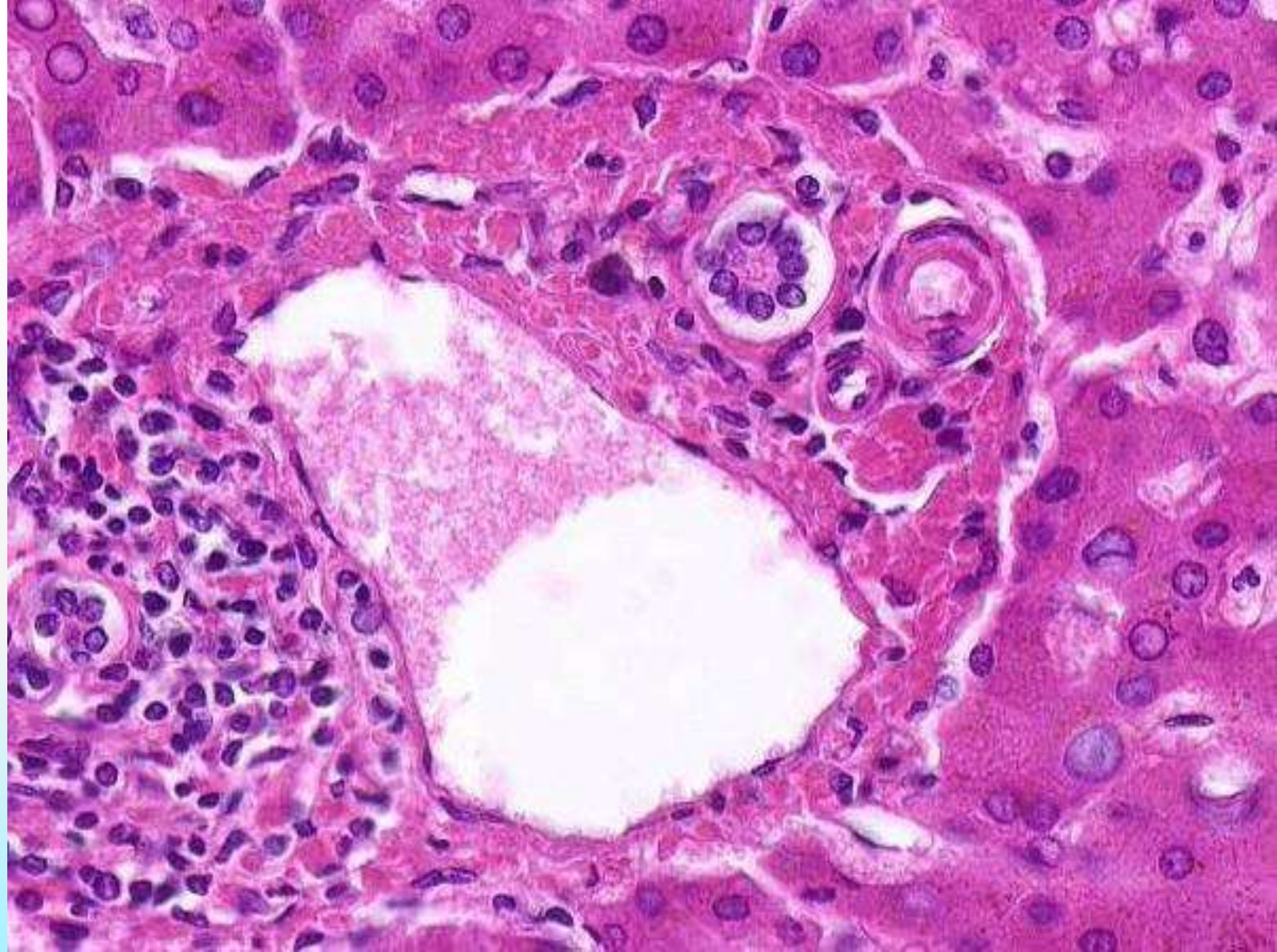
O

FIBROUS
TISSUE





PV = portal vein, HA = hepatic artery, BD = bile ductules



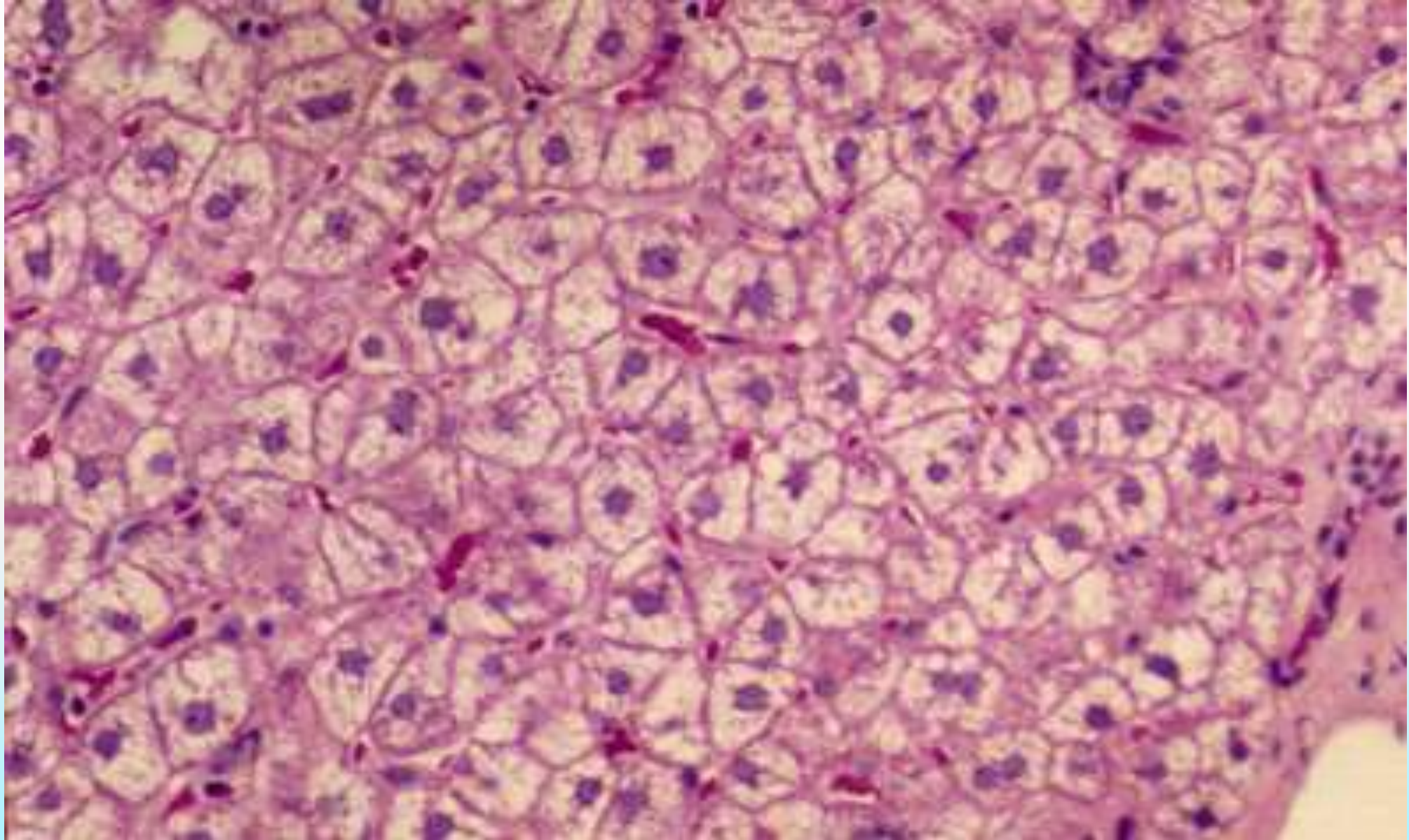


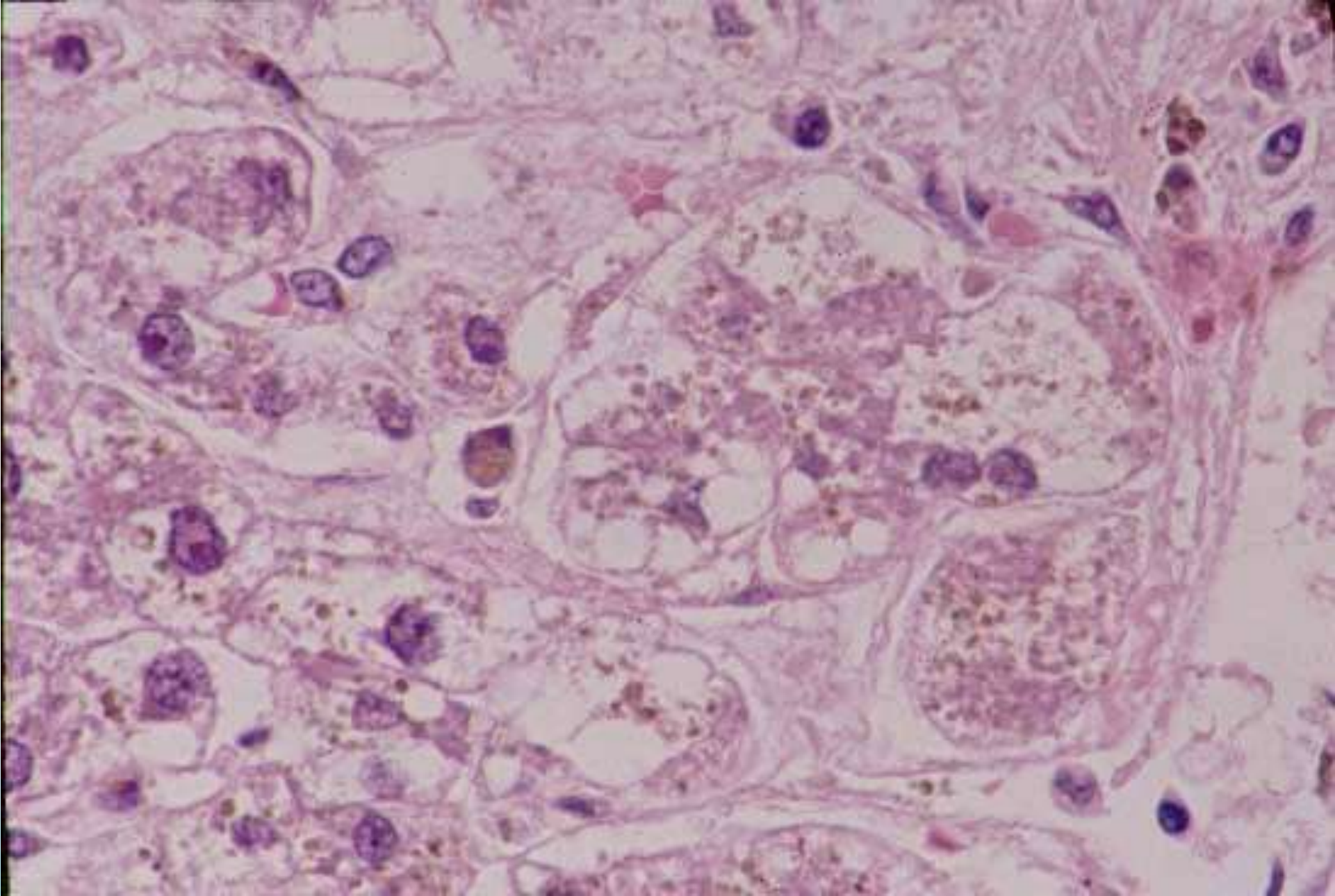
LIVER PATHOLOGY

General paranchymatous hepatic responses to liver injury

- 1- Inflammation (acute &chronic).**
- 2- Degeneration & intracellular accumulation.**
- 3- Necrosis and apoptosis.**
- 4- Regeneration.**
- 5- Fibrosis.**

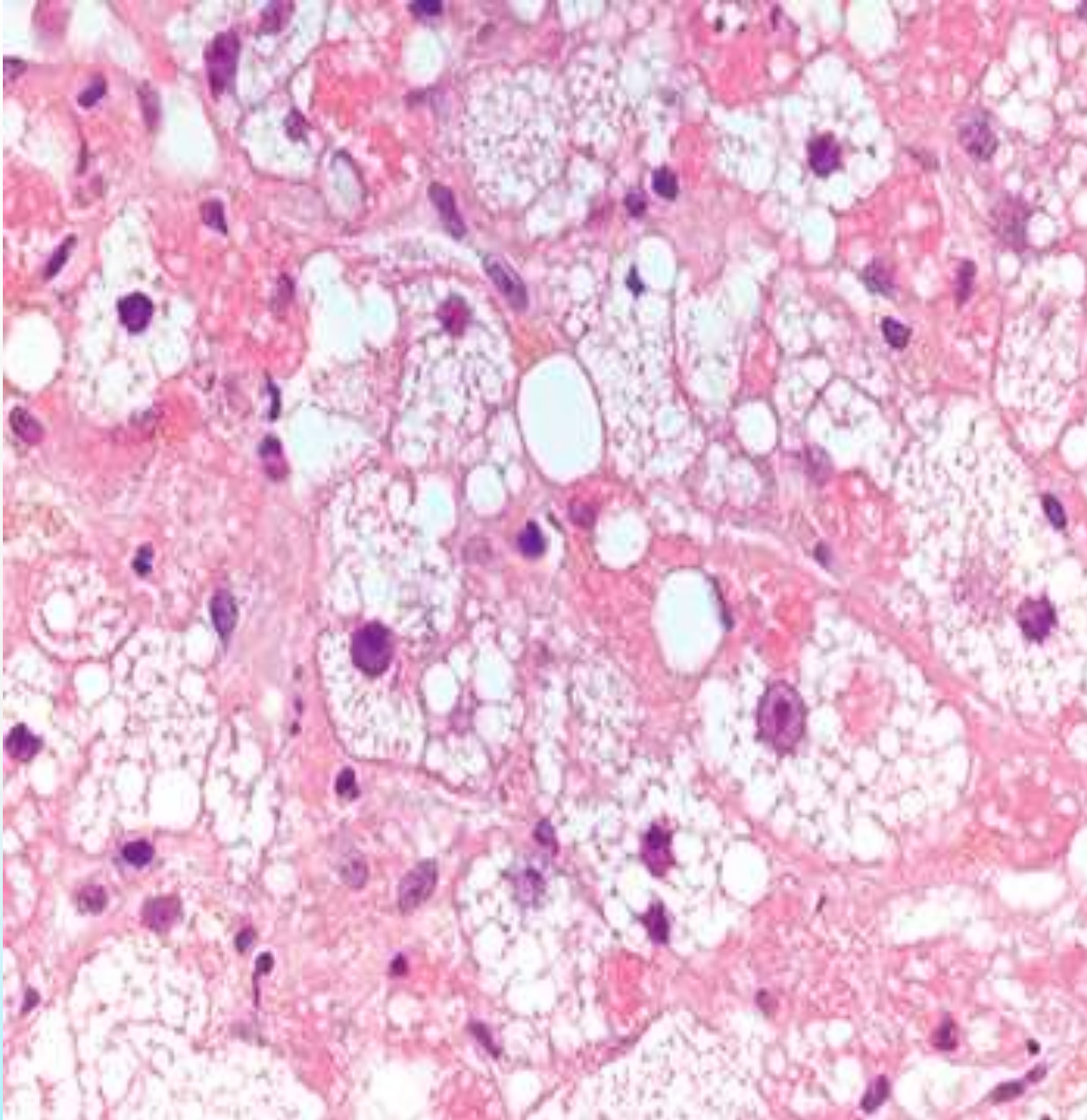
Ballooning degeneration



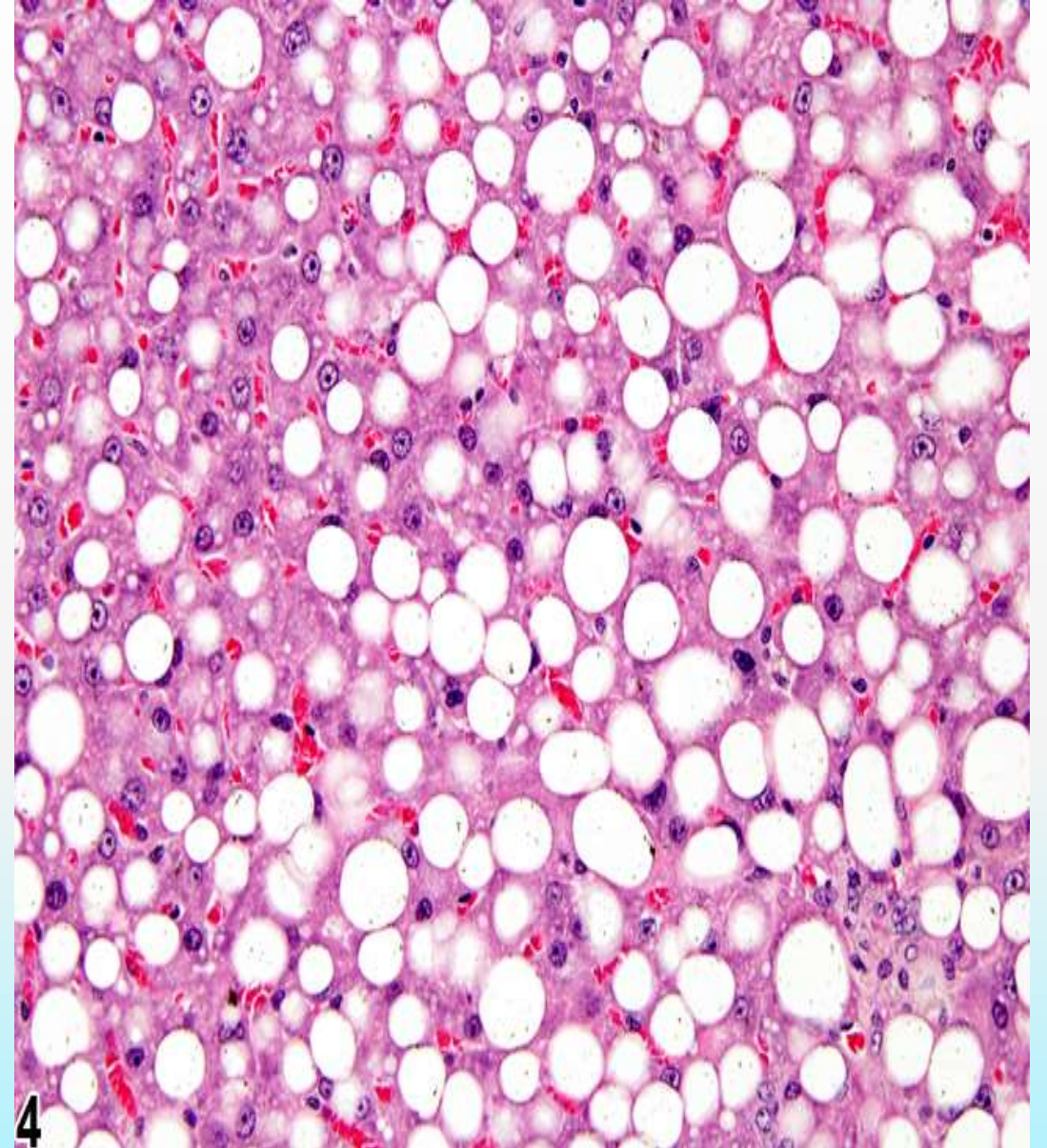


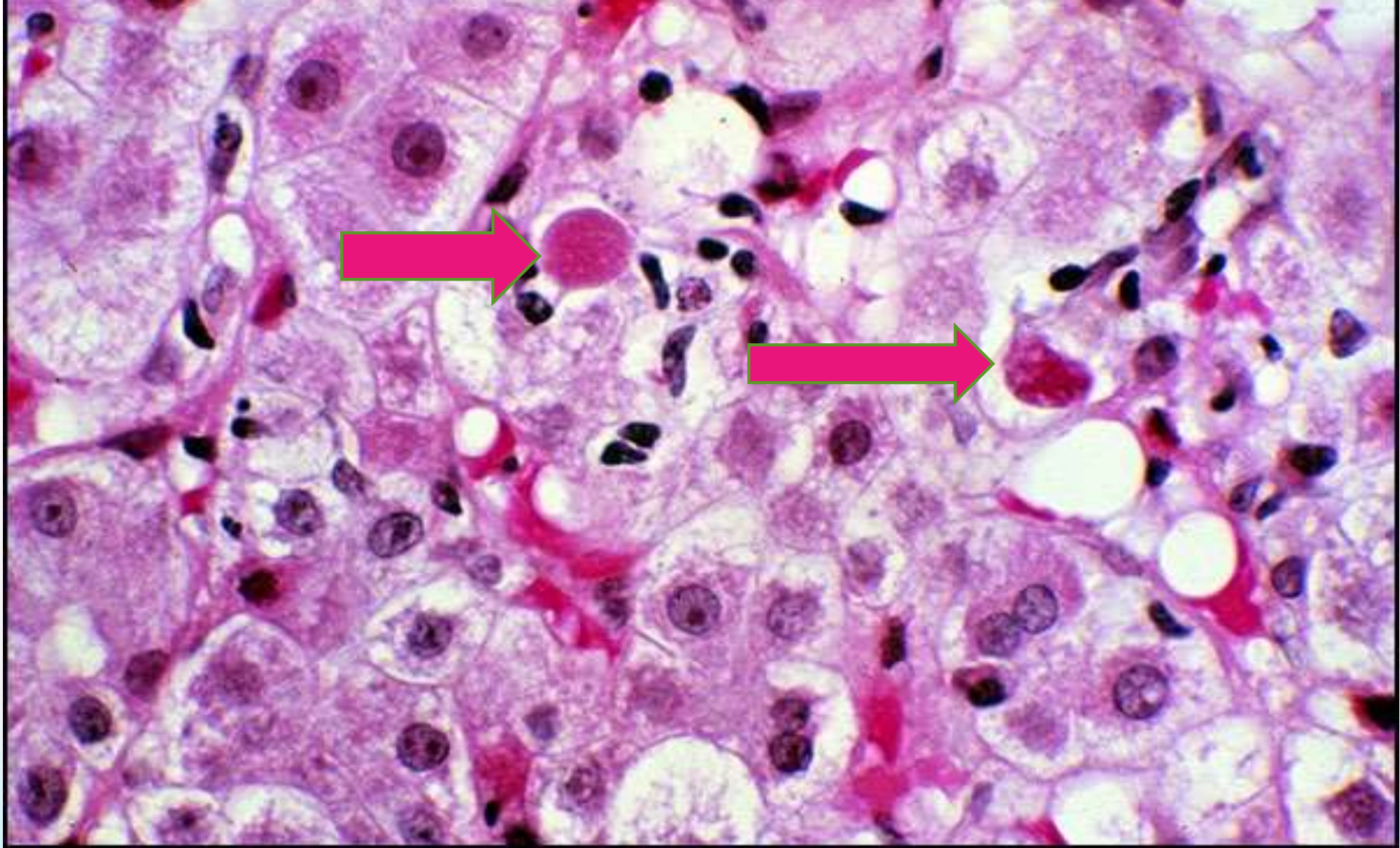
“FEATHERY” DEGENERATION

Microvesicular steatosis



Macrovesicular steatosis





Apoptosis COUNCILMAN body

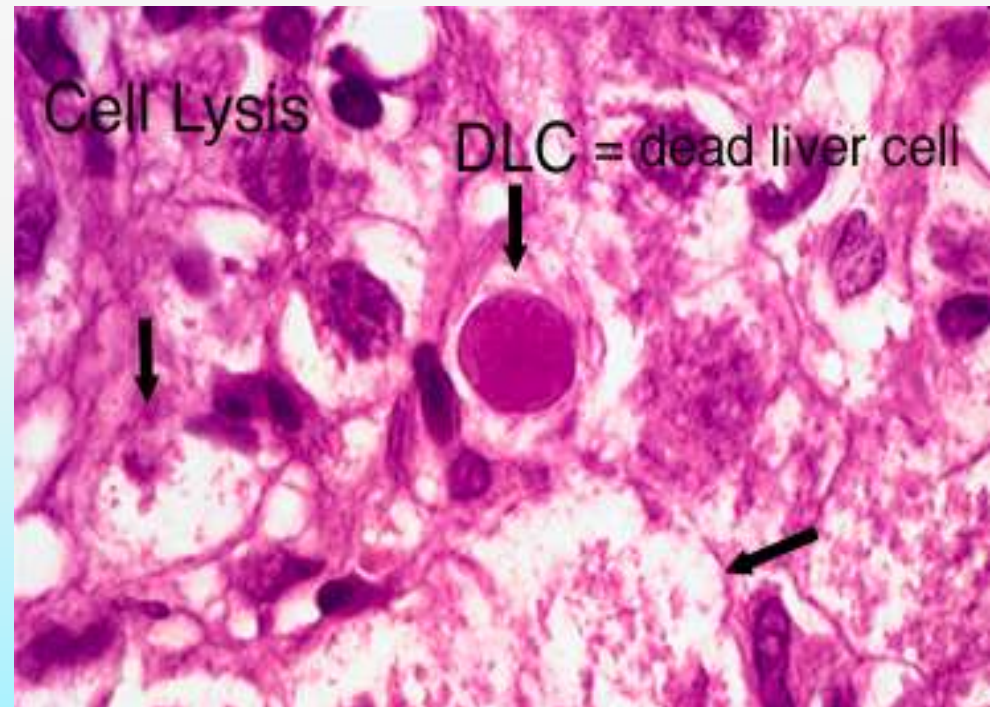
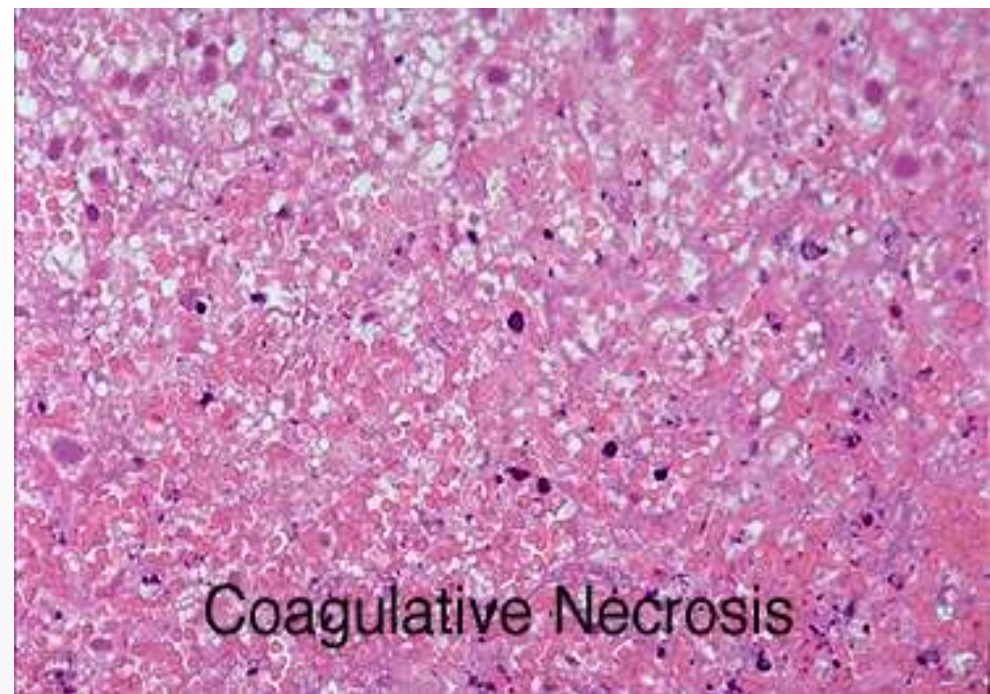
Histologic patterns of hepatic injury

NECROSIS

Coagulative necrosis: preserved
architecture loss of cellular details

Councilman bodies: dead
hepatocytes

Lytic necrosis: hepatocytes swell &
rupture



CLINICAL SYNDROMES OF VIRAL HEPATITIS

ACUTE HEPATITIS

Histology of acute hepatitis:

Ballooning degeneration

Cholestasis (bile plugs)

Steatosis (HCV)

Hepatocyte spotty necrosis (dead cells surrounded by macrophages)

apoptotic cells (Councilman bodies)

Kupffer cell hypertrophy & hyperplasia

Portal tracts infiltration by inflammatory cells with spill over to parenchyma (interface hepatitis)

Acute hepatitis ■

Lobular hepatitis is the predominate

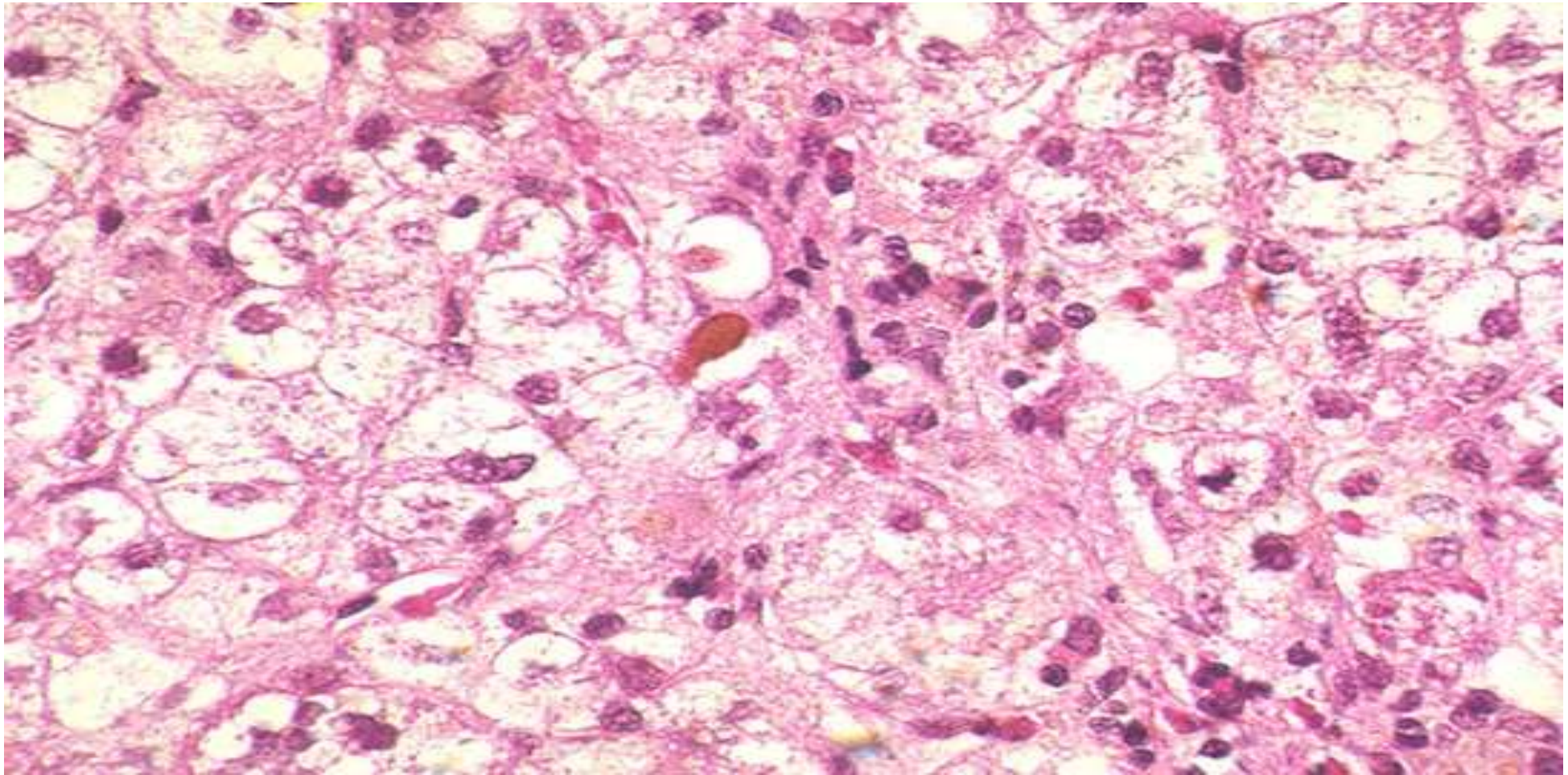
There is **lobular disarray** but no fibrosis

Chronic hepatitis

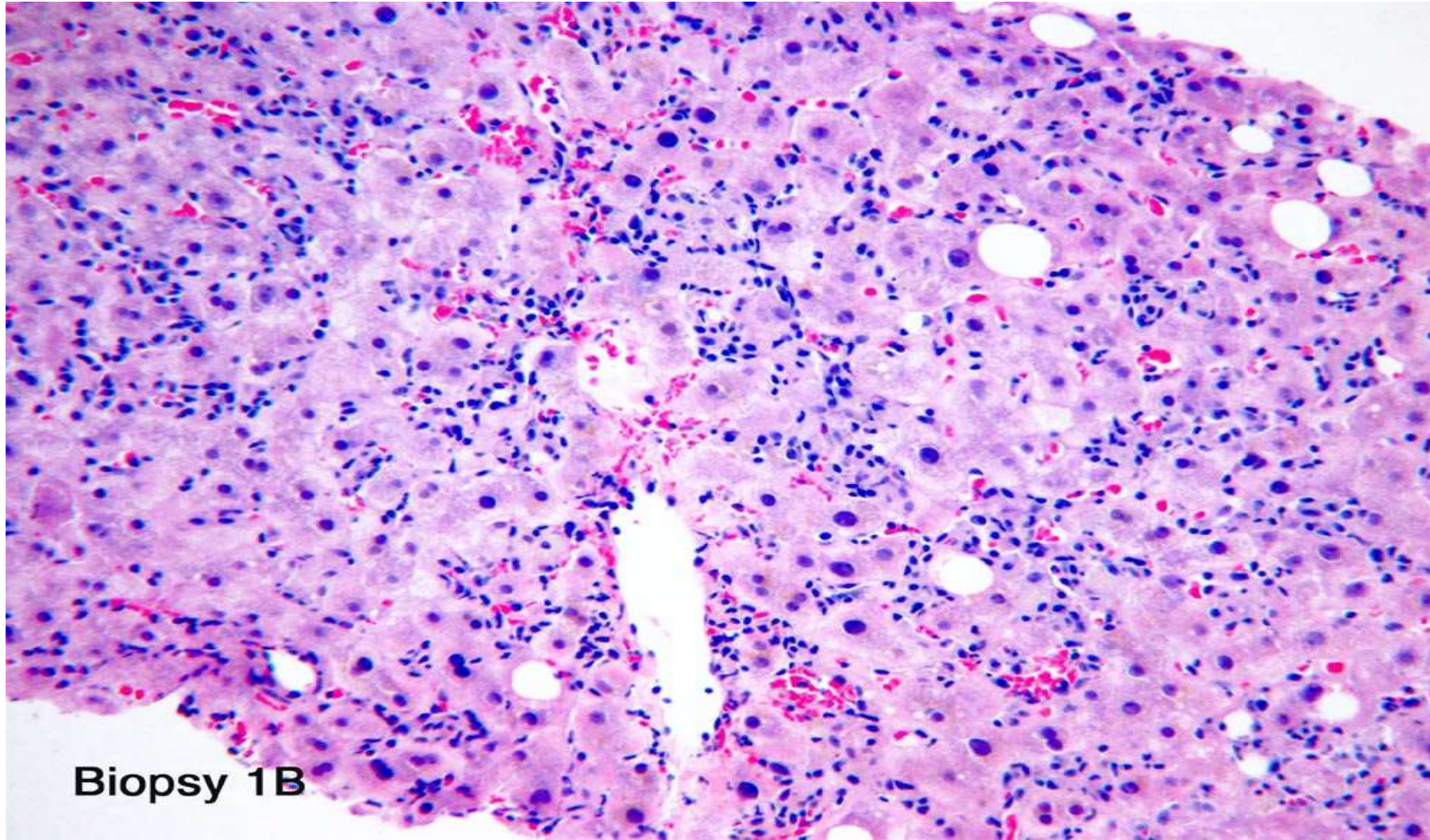
Portal inflammation is the Predominant(+/- interface hepatitis)

May proceed to fibrosis and then cirrhosis

"Lobular disarray": Loss of hepatocyte, inflammatory reaction, hepatocellular swelling distort the pattern of the liver plates somewhat confusing the lobular architecture.(H&E stain).

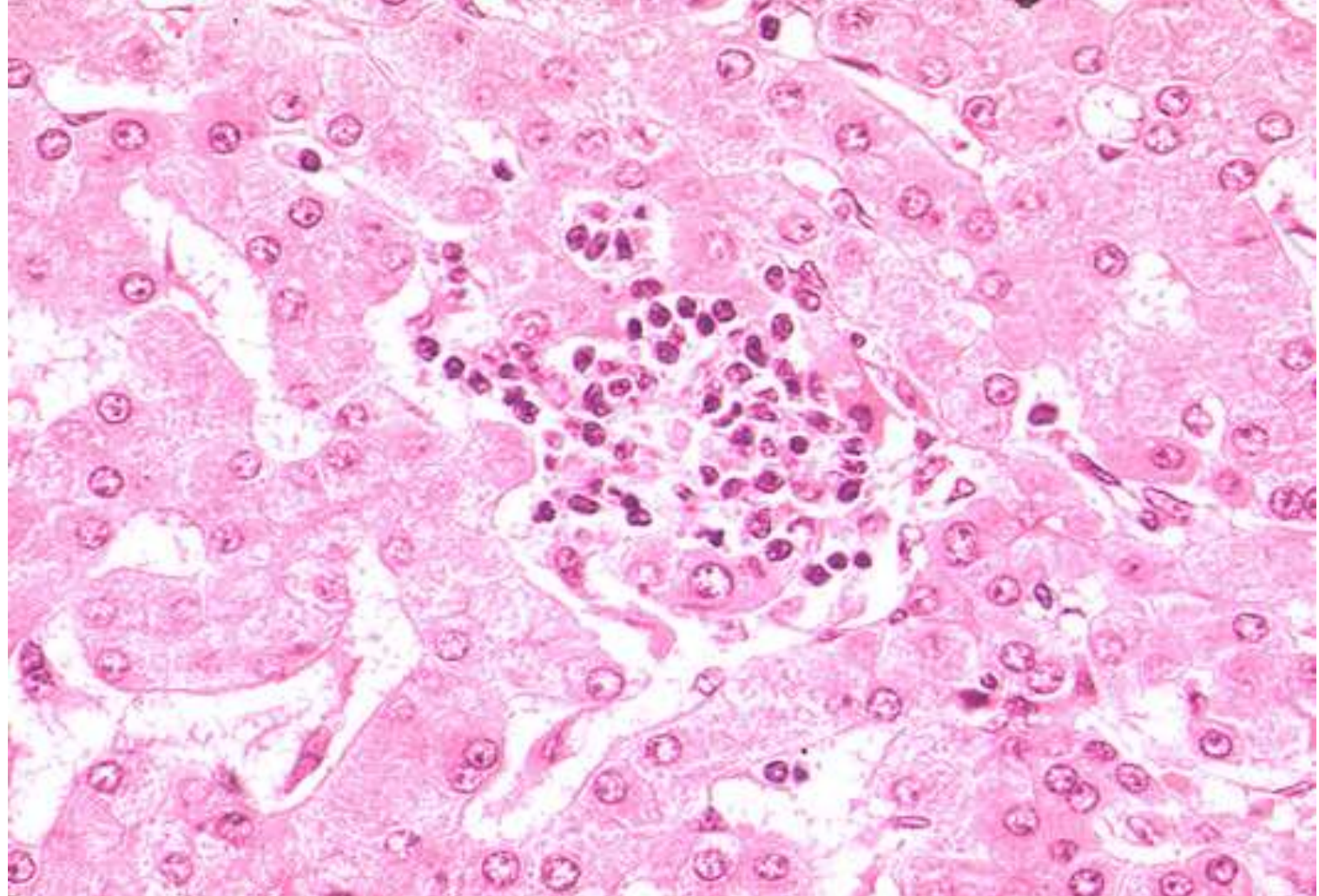


Marked lobular hepatitis with prominent sinusoidal lymphocytosis

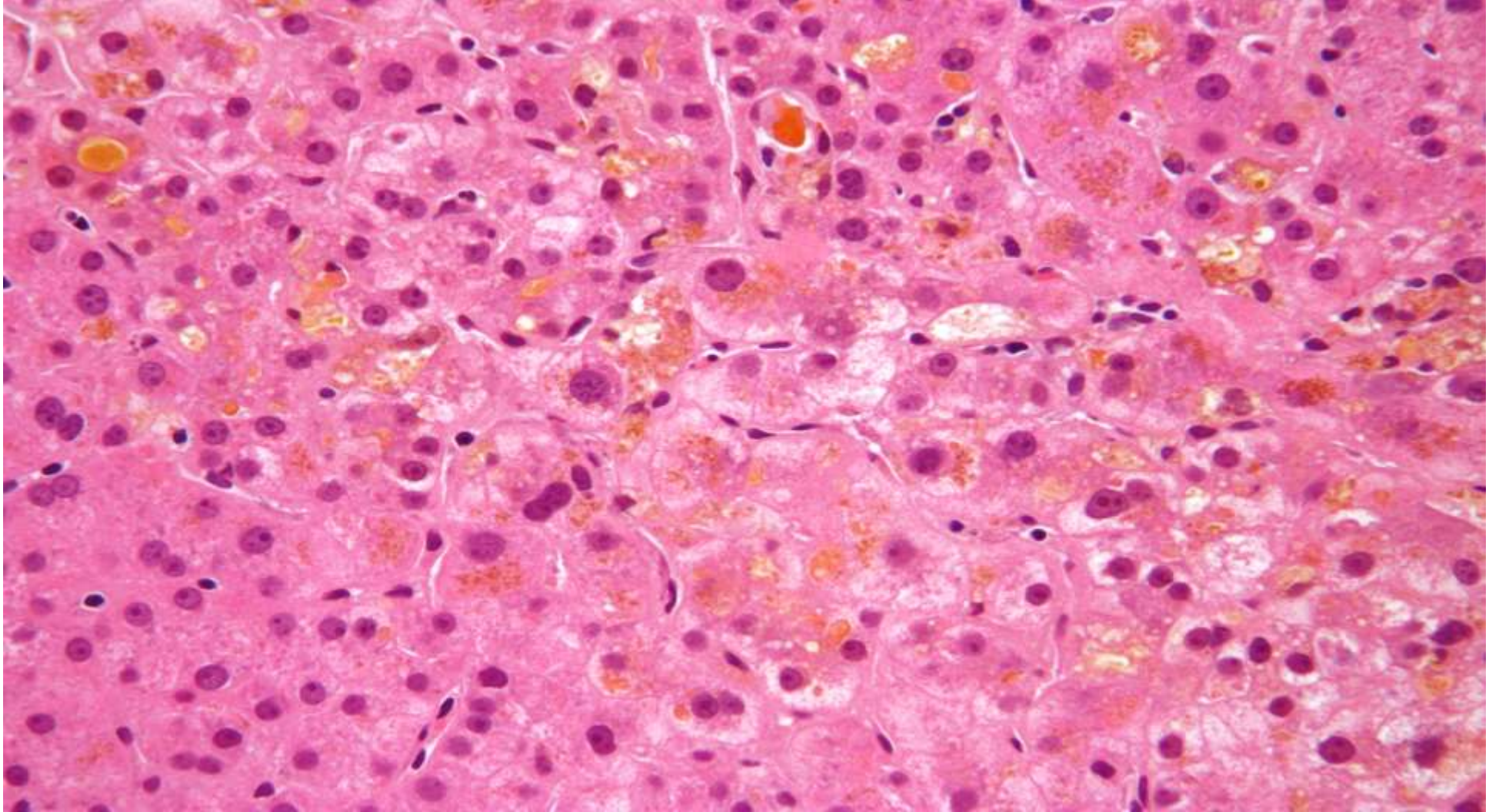


Biopsy 1B

Focal spotty necrosis: In the center of this slide there is a focus of cell dropout with inflammatory reaction



Cholestasis: deposition of bile in bile canaliculi
appears as brownish deposits



Morphology of chronic hepatitis

Most of morphologic changes in chronic hepatitis are **shared** with acute hepatitis.

But the following changes are only seen with chronic hepatitis.

a. inflammation is limited to the portal tracts & consist of **lymphocytes** , **macrophages**, rare neutrophils & eosinophils.

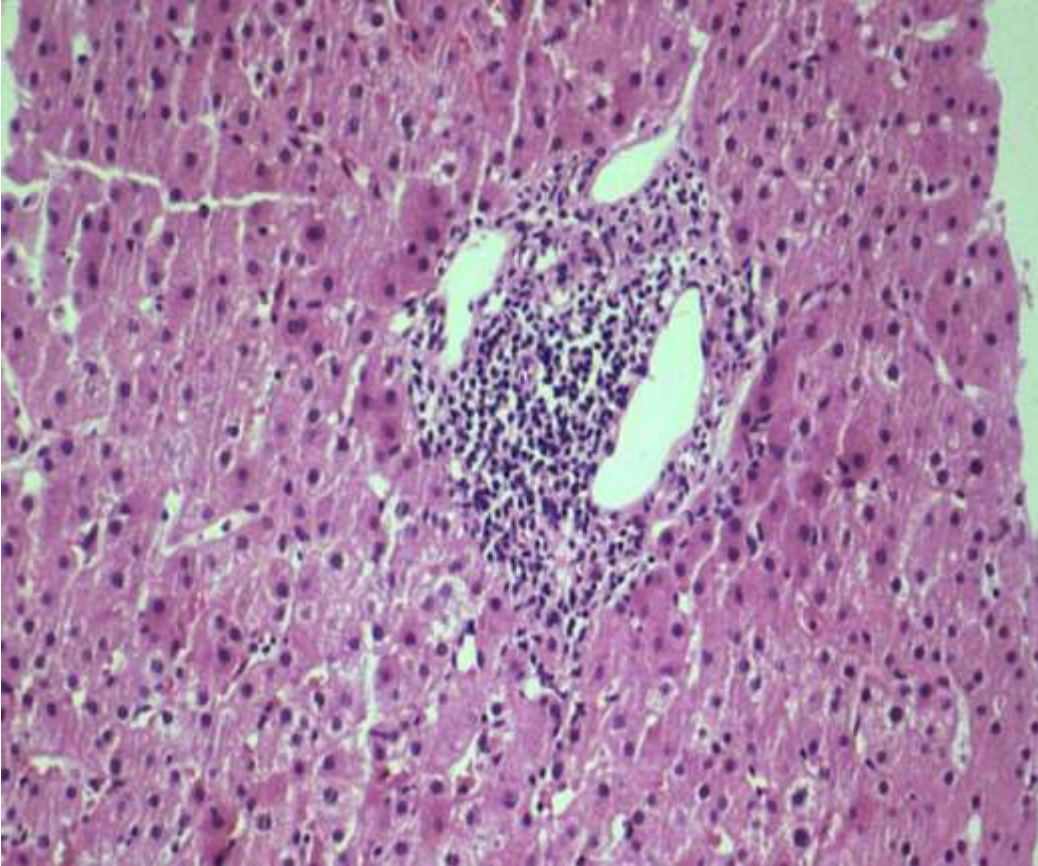
c. continuous periportal necrosis & bridging necrosis.....progressive liver damage

d. deposition of fibrous tissue (irreversible injury).

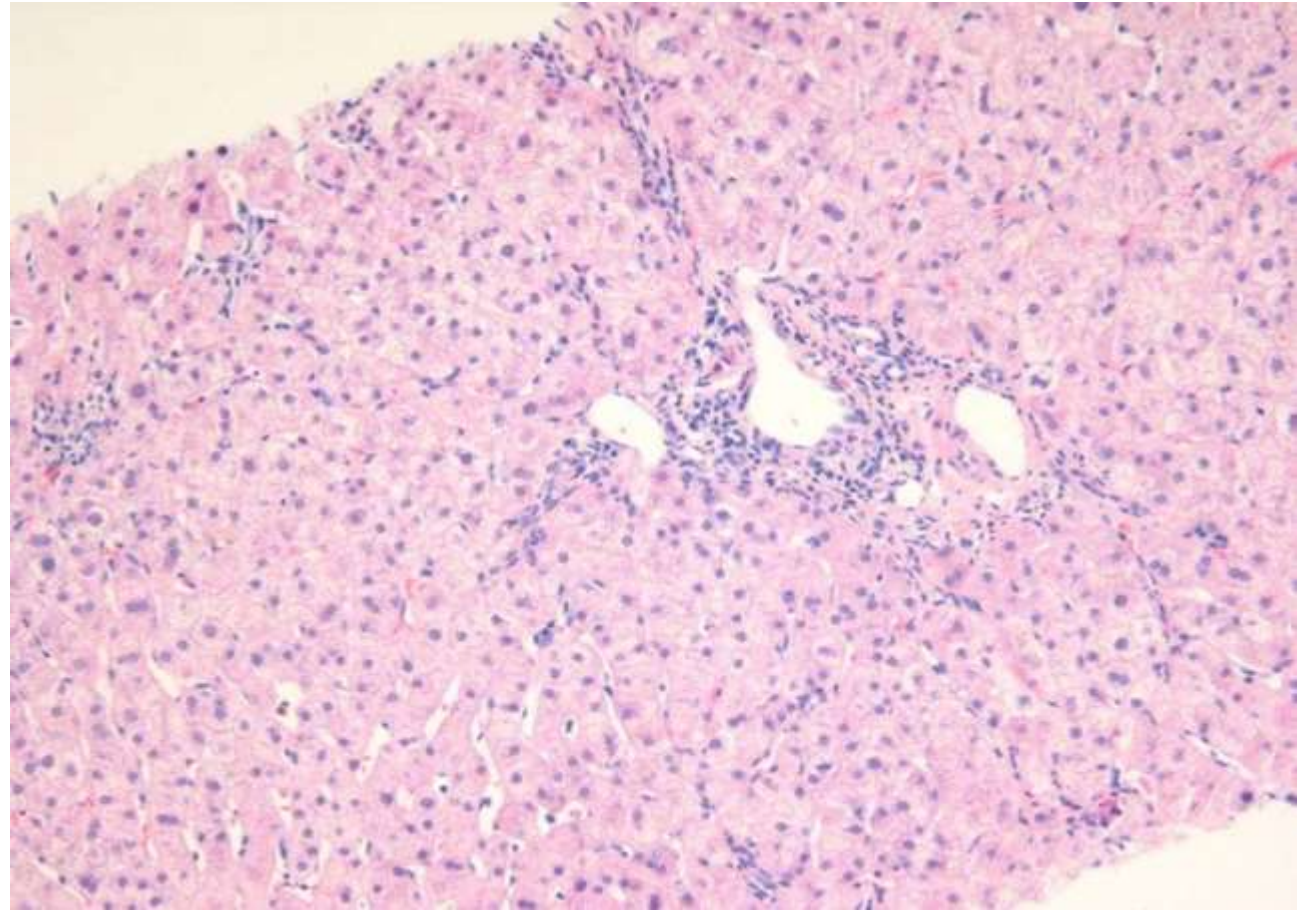
e. cirrhosis which is usually of macro nodular type.

Chronic hepatitis

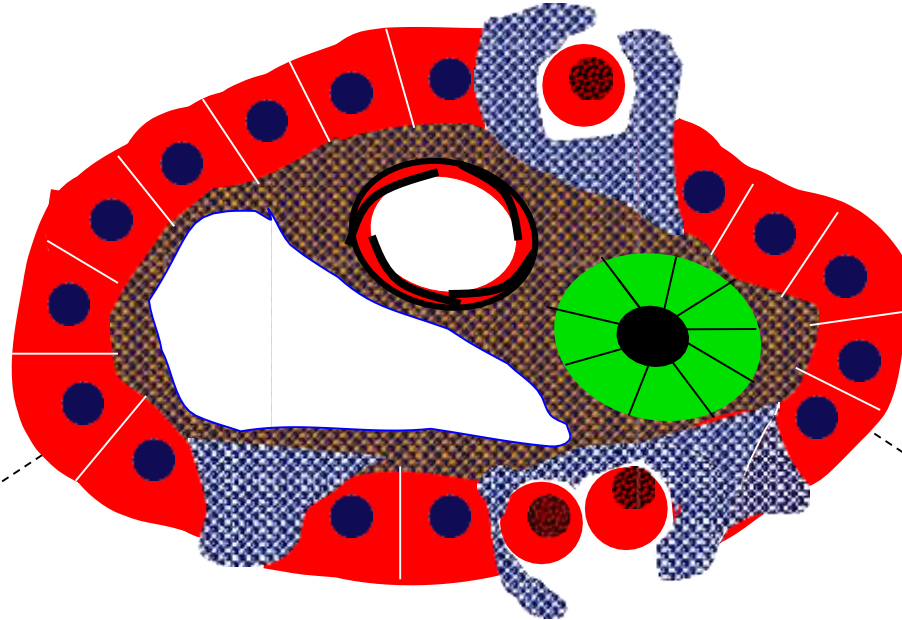
- Portal inflammation Without interface hepatitis



- Portal inflammation with interface hepatitis
- Some necroinflammatory foci in the lobule

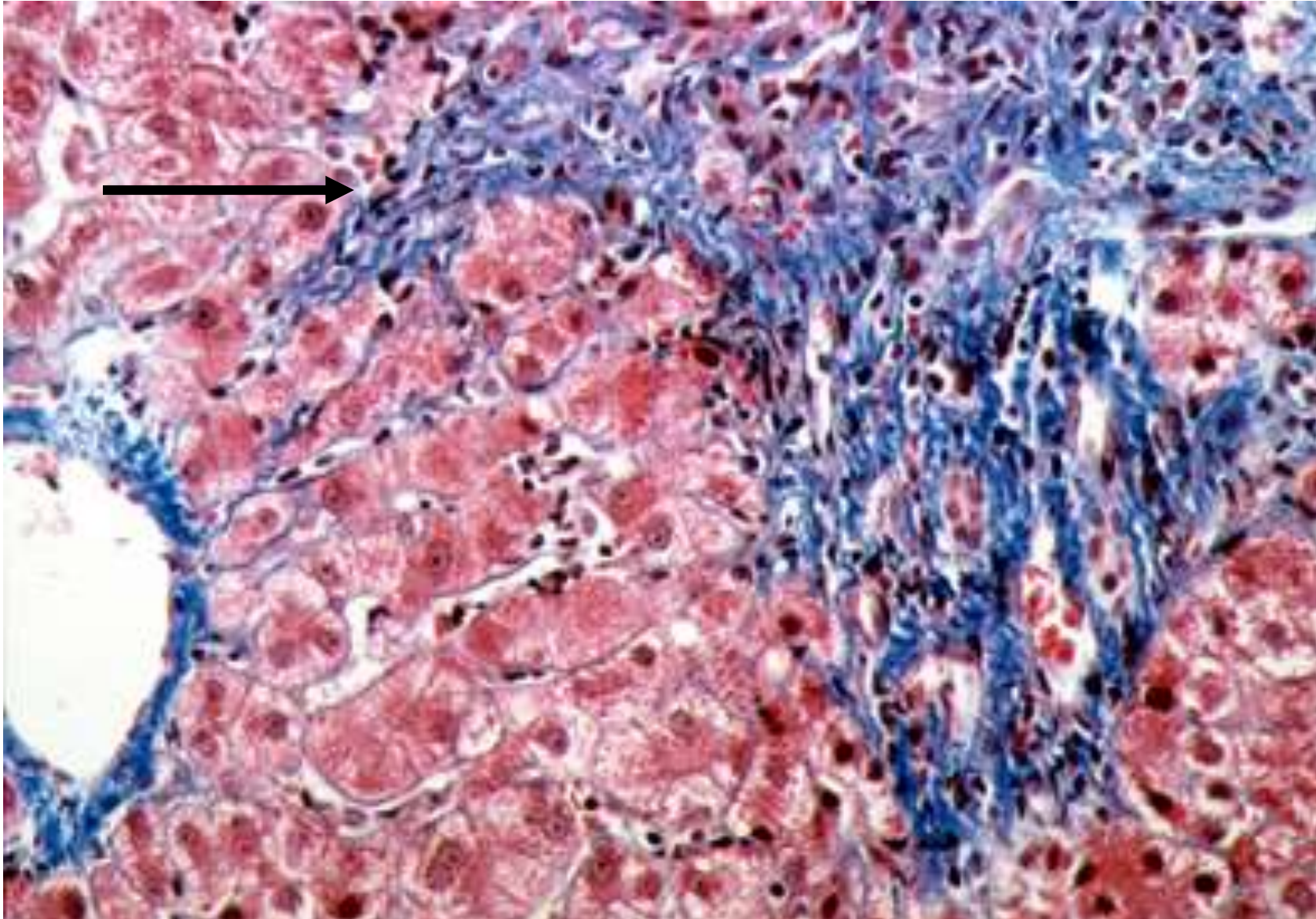


The disruption of the **“limiting plate”** is a classical concept in liver disease, especially chronic hepatitis. **The “limiting plate” is the fence of connective tissue which delineates a portal triad area from the surrounding hepatocytes. When this is breached, the disease process is regarded as more severe, and many say is the earliest step to cirrhosis**

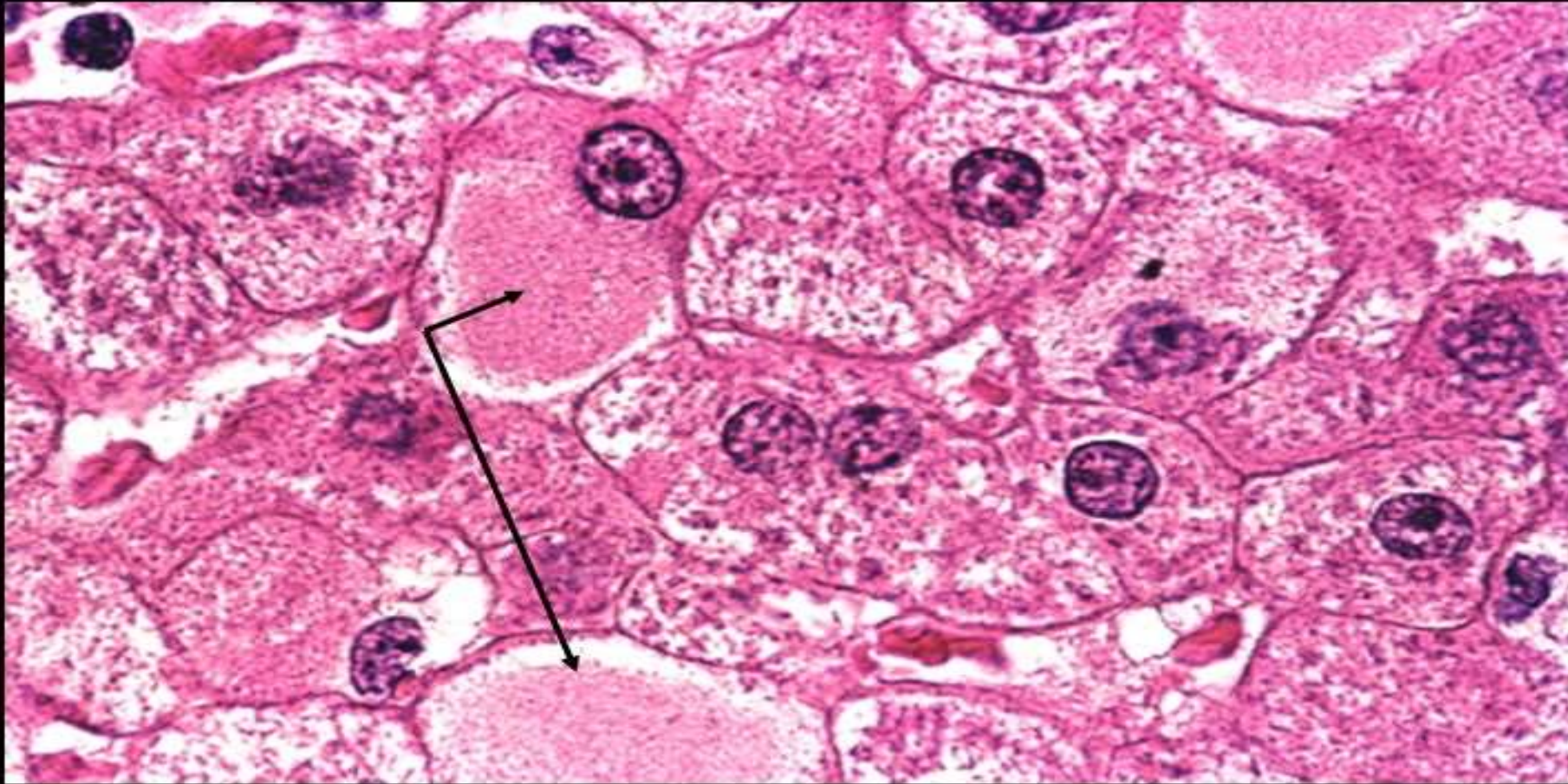


**“Piecemeal Necrosis”
or, better,
“Interface Hepatitis”**

Bridging Fibrosis



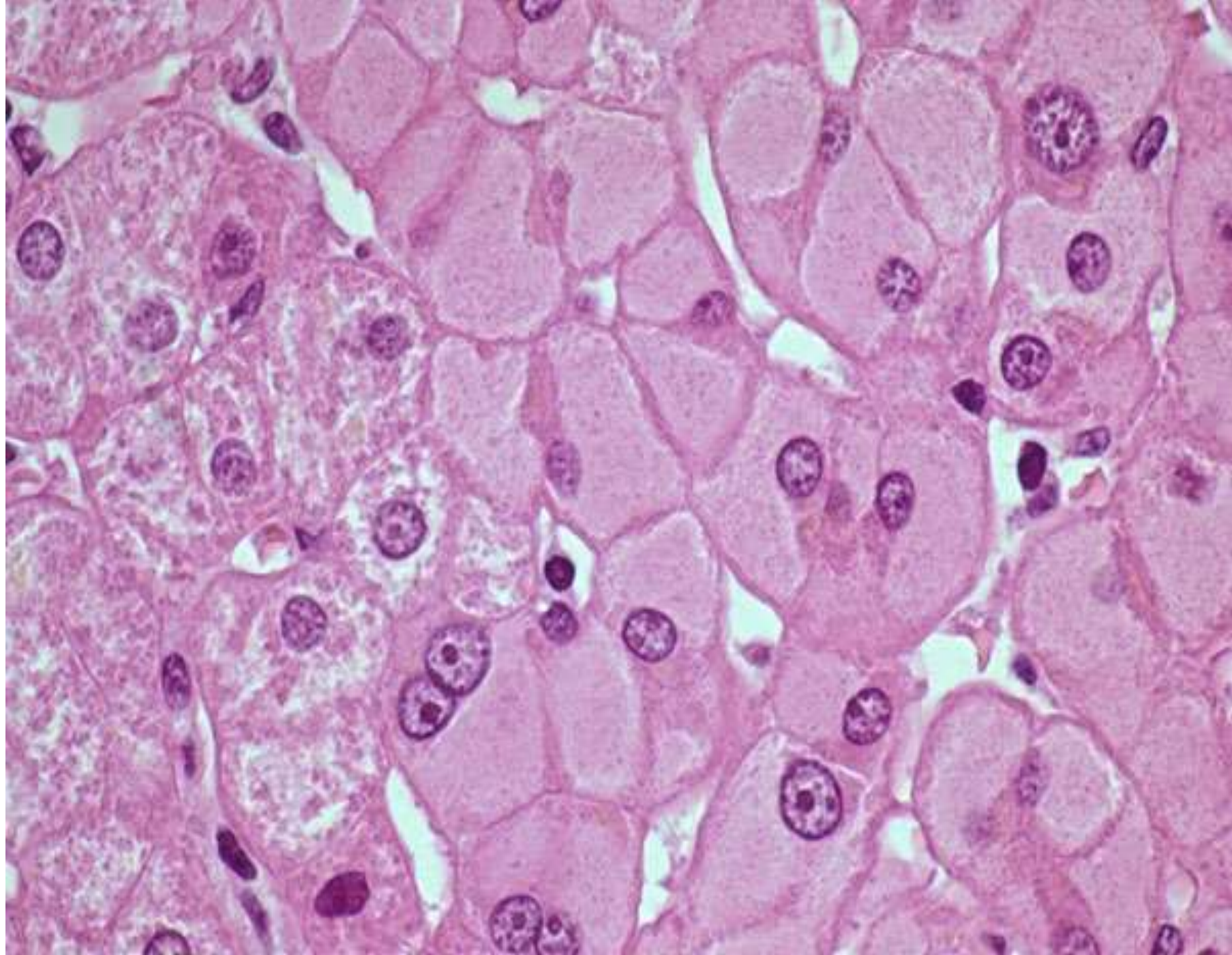
Chronic viral hepatitis B showing ground glass hepatocytes



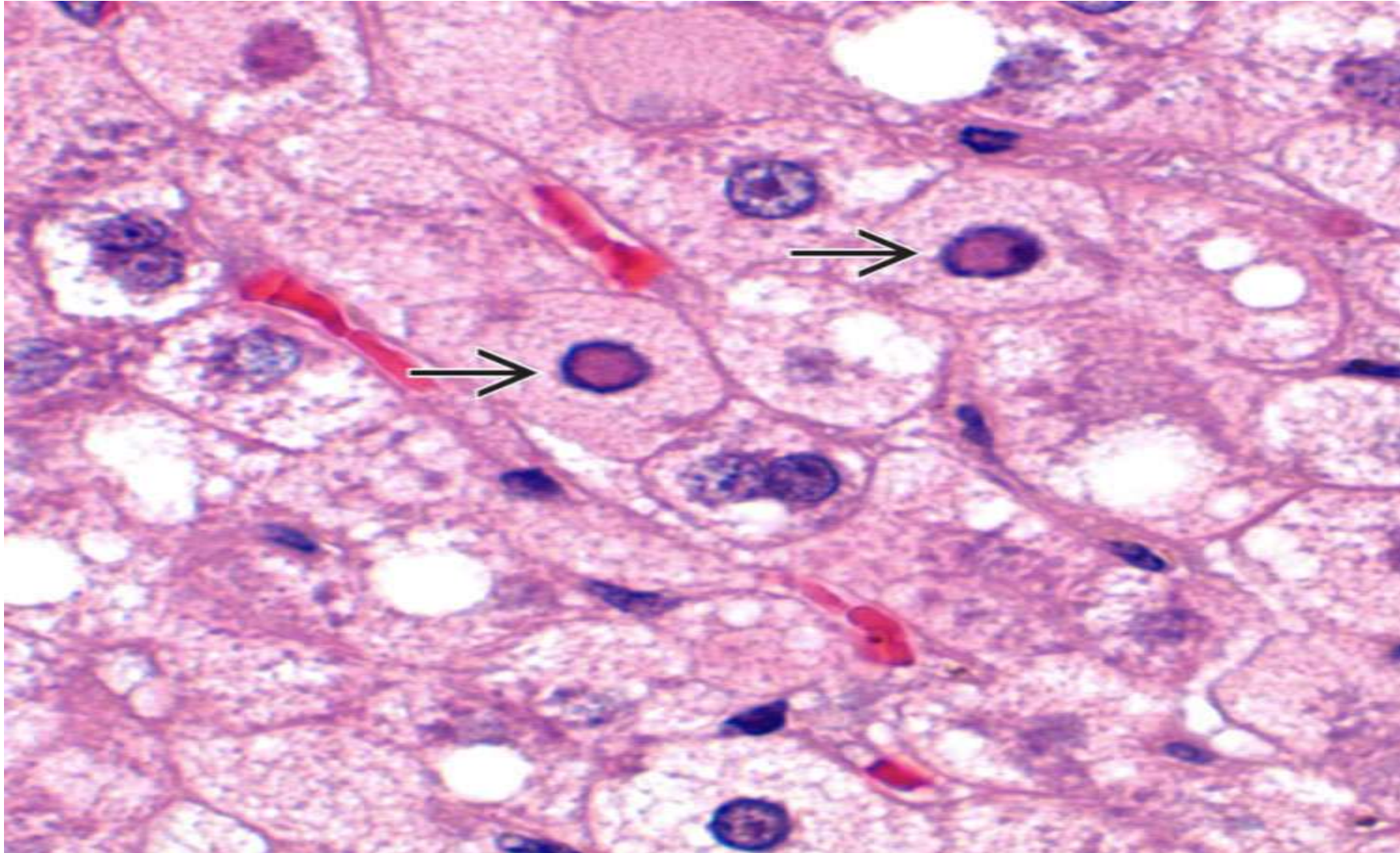
Ground glass hepatocytes, characterized by more pale, eosinophilic, and homogeneous cytoplasm than surrounding normal (more granular) hepatocytes. Note (artifactual) cleft between "ground glass" cytoplasm and hepatocellular cell membrane. The change corresponds to extensive endoplasmic reticulum hyperplasia and massive accumulation of HBsAg. (H&E)

CHRONIC HEPATITIS B. ground-glass hepatitis B inclusion





Sanded Nuclei Hepatitis B-infected hepatocytes may have pale pink, finely granular intranuclear inclusions (sanded nuclei image)





FATTY LIVER

ALCOHOLIC LIVER DISEASE

(fatty changes) **Steatosis**

**Lipid droplets
accumulate in
hepatocytes**

2 histologic types:

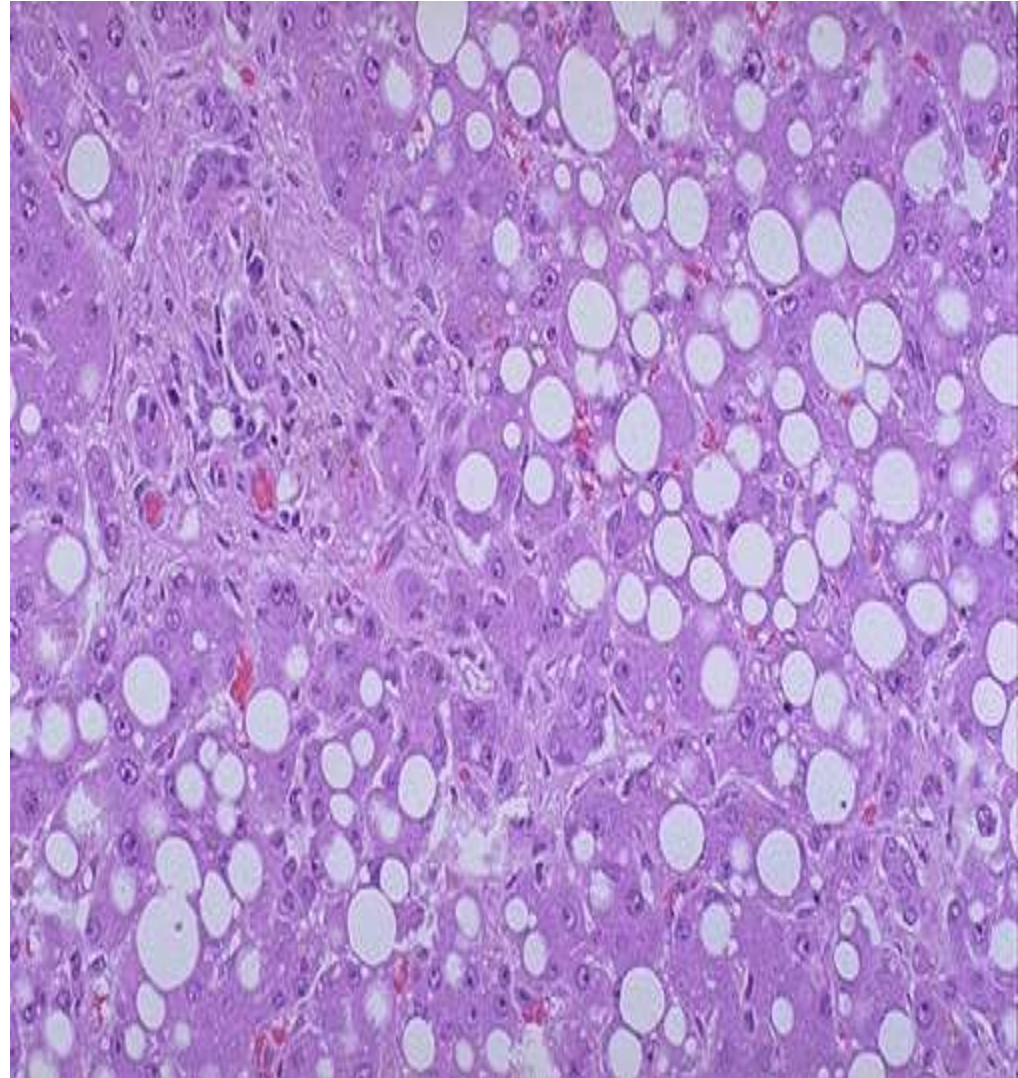
Microvesicular

Macrovesicular

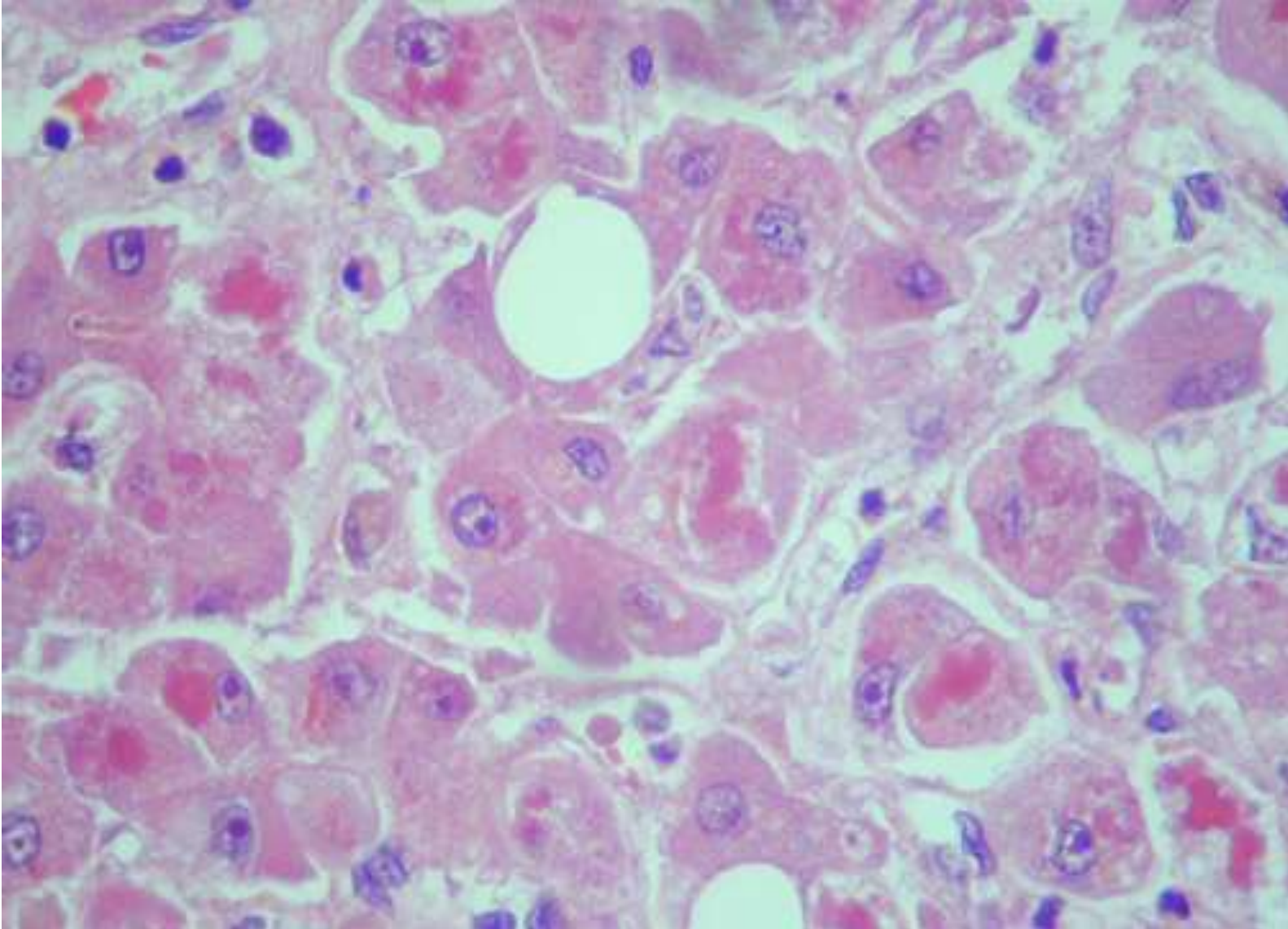
Initially centrilobular

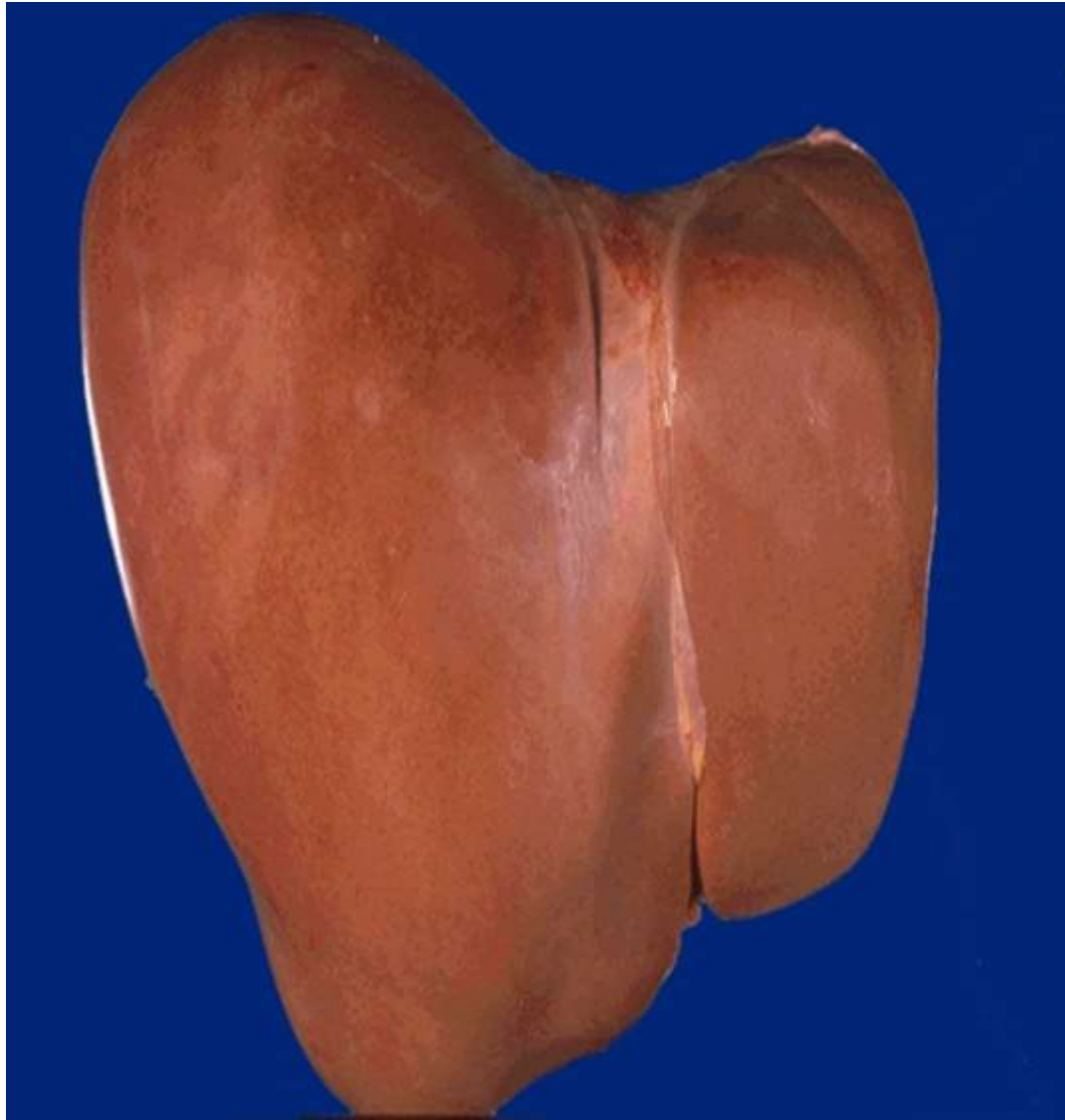
Later panlobular

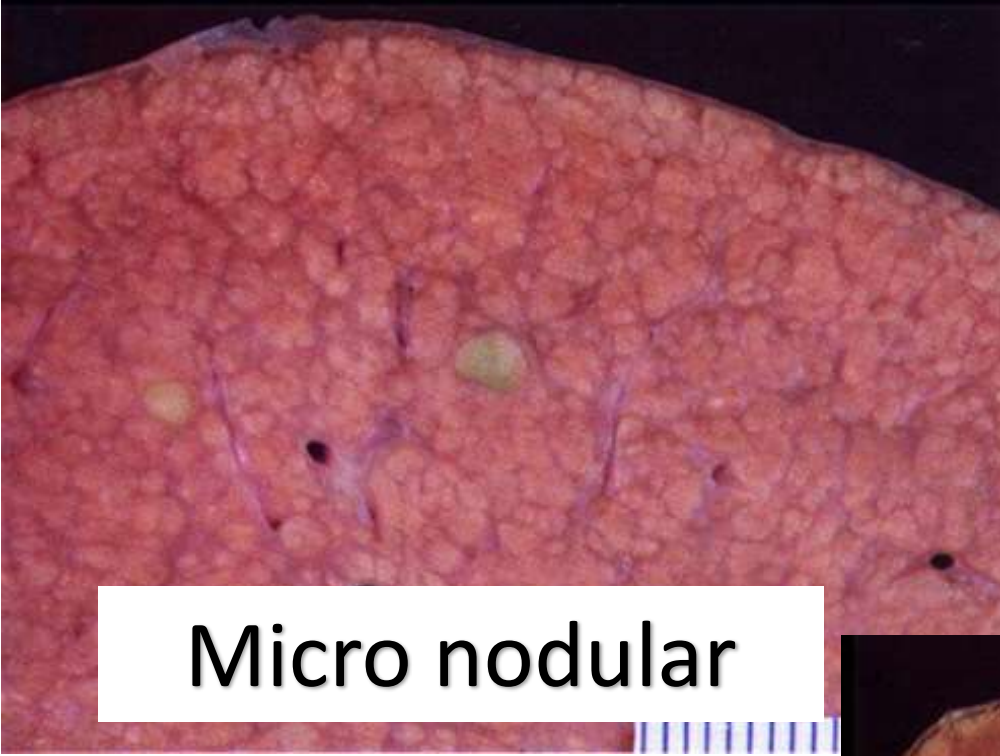
**Completely reversible if
there is abstention**



MALLORY'S bodies: eosinophilic cytoplasmic inclusions
(cytokeratin intermediate filaments)







Micro nodular

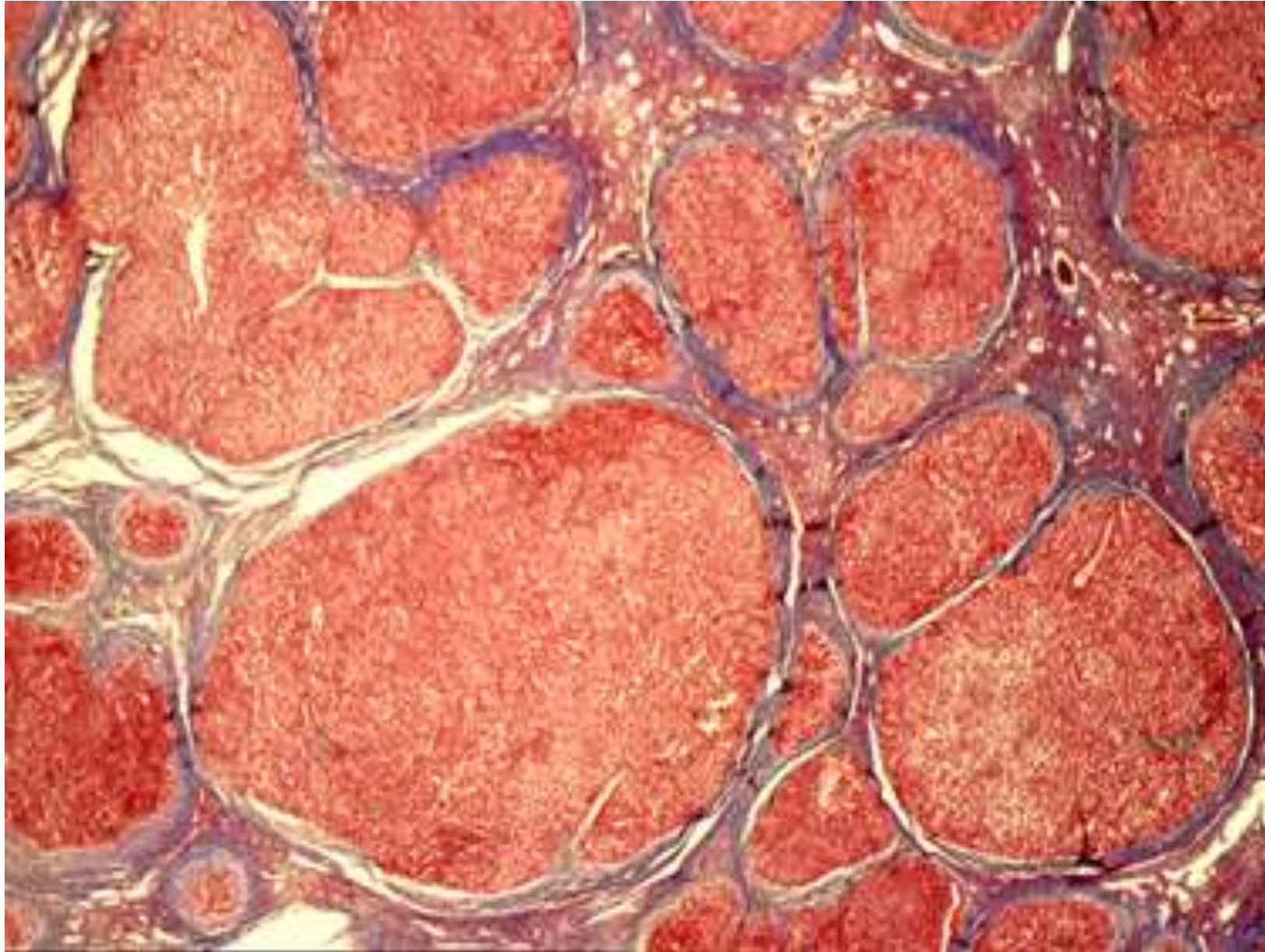


Macro nodular



CIRRHOSIS, TRICHROME STAIN

CIRRHOTIC LIVER--
regenerative nodules usually don't contain portal tracts



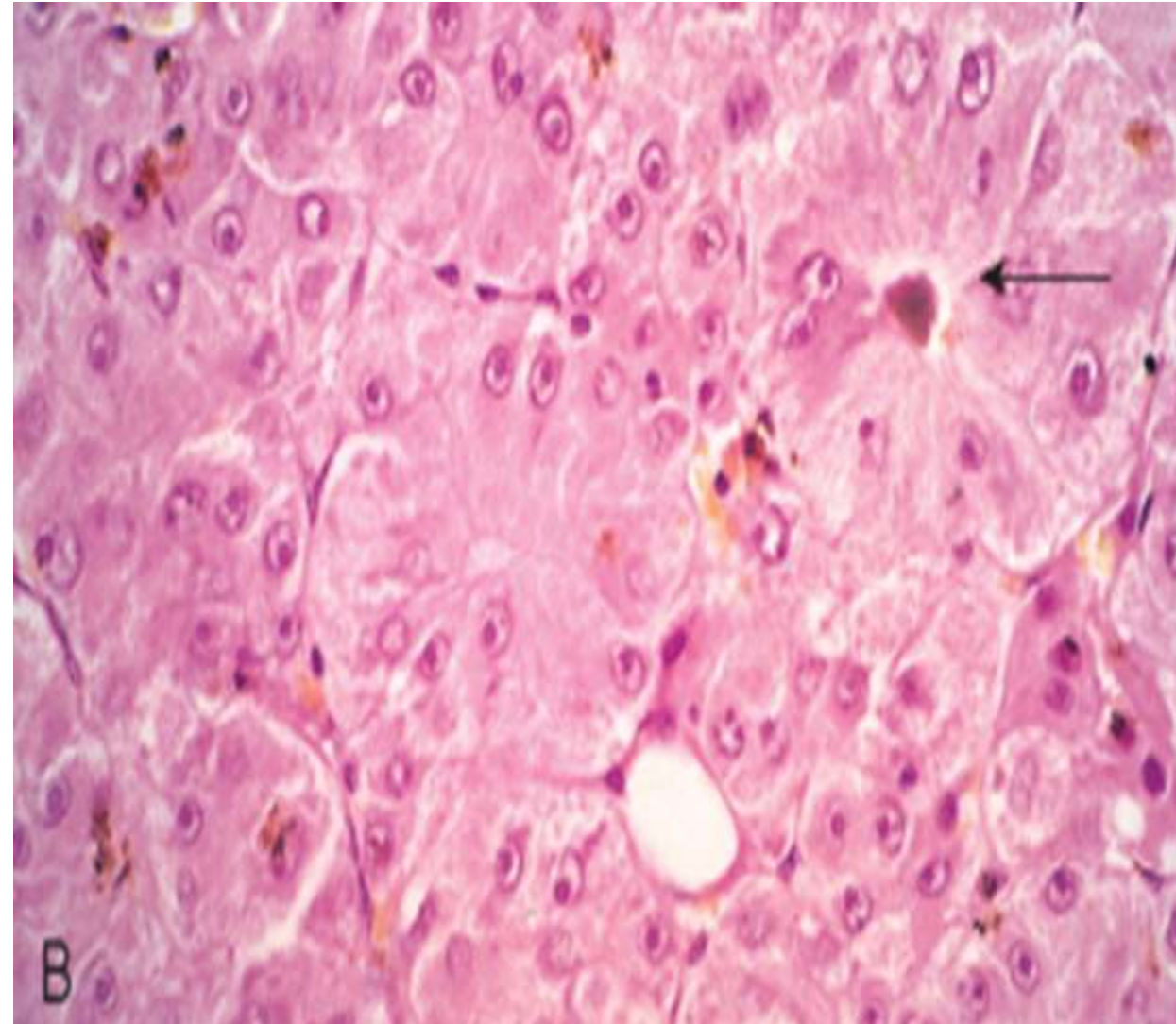
LIVER CELL ADENOMA



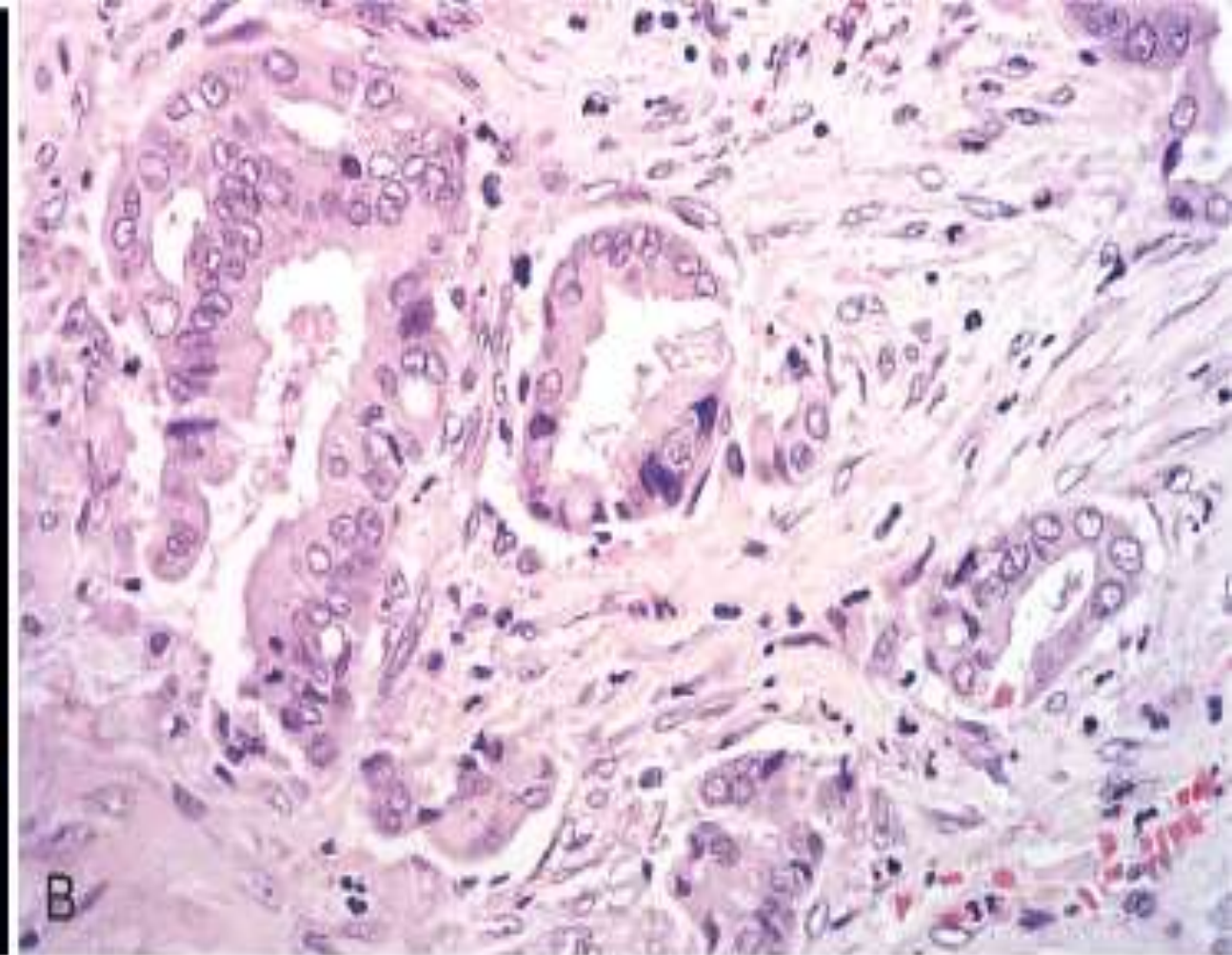
Hepatocellular ca gross and mic



© Elsevier 2005



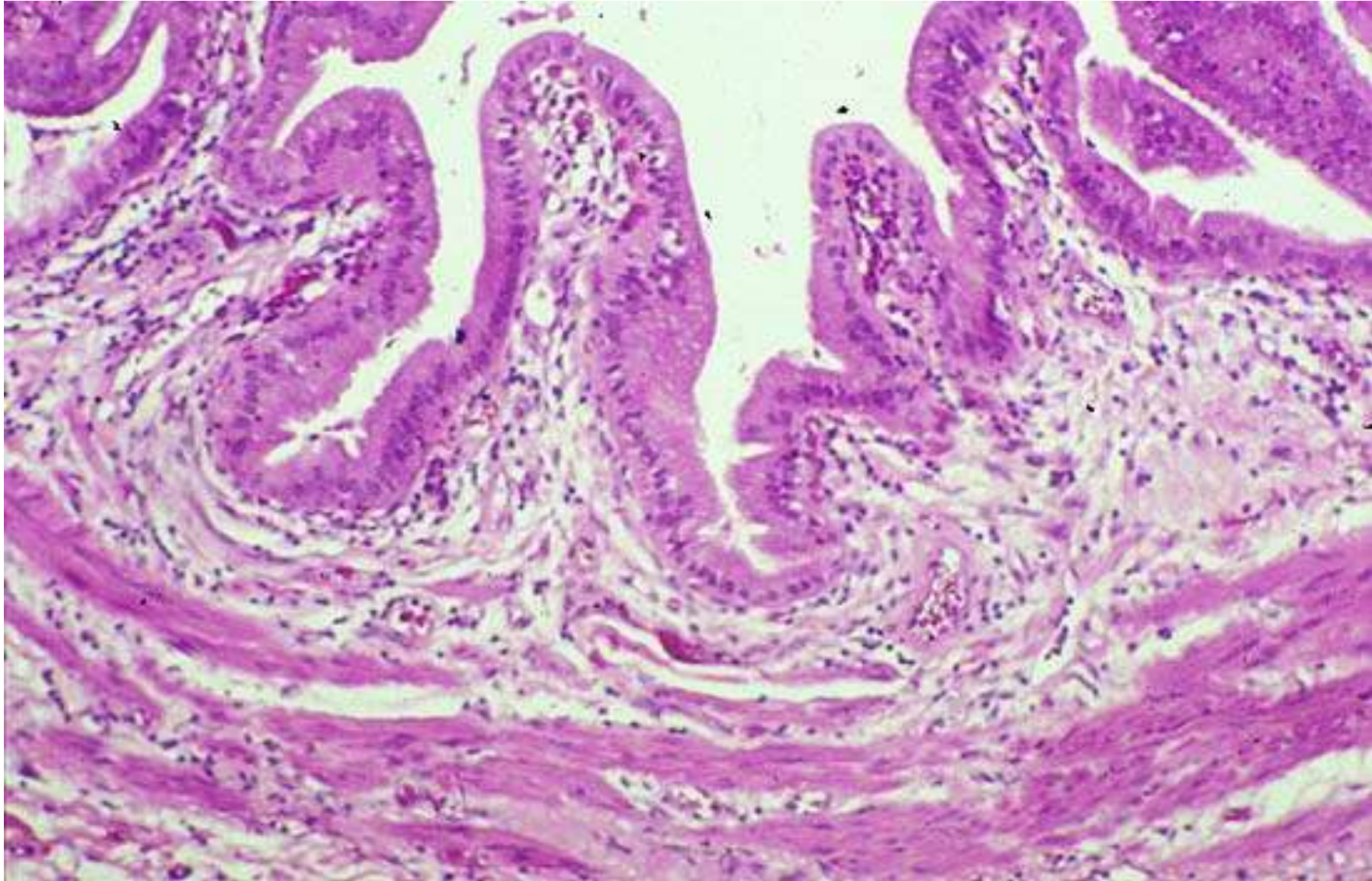
Cholangiocarcinoma gross and mic.



PATHOLOGY OF THE BILIARY TRACT

- **Disorders of the gallbladder**
 - **Cholelithiasis**
 - **Cholecystitis(acute and chronic)**
 - **Tumors**
- **Disorders of extrahepatic bile ducts**
 - **Choledocholithiasis**
 - **Ascending cholangitis**
 - **Tumors**

NORMAL GALLBLADDER



Cholesterol stones :

- Gross :
- **Pure** pale yellow, ovoid, firm, single to multiple with faceted surfacesm mostly radiolucent,
- **Mixed** :20%is radio opaque due to the presence of calcium carbonate content.



• Pigment stones

- **Black stone** (in **sterile gall bladder** bile)- small size, fragile to touch, numerous, 50-70% are radioopaque
- **Brown stone** (in **infected intrahepatic or extrahepatic ducts**)- single to a few, soft, greasy,.

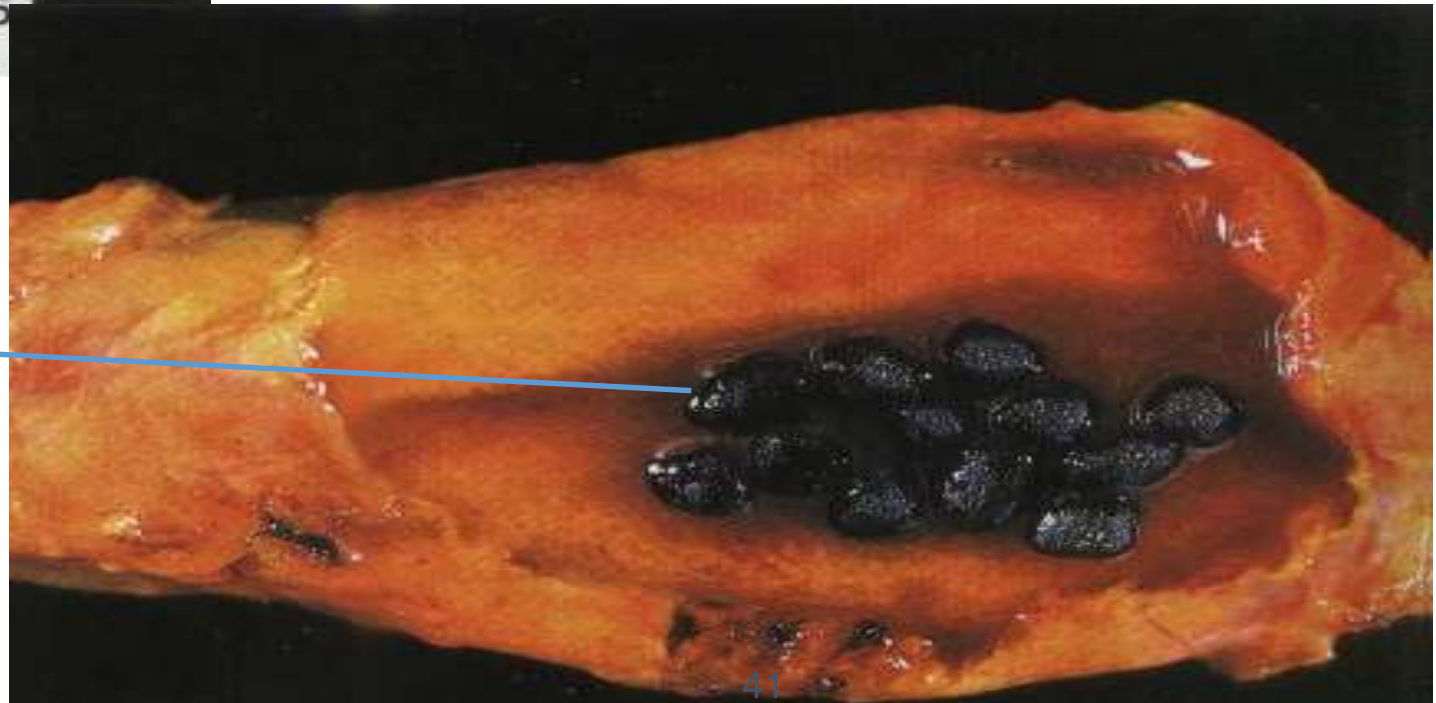


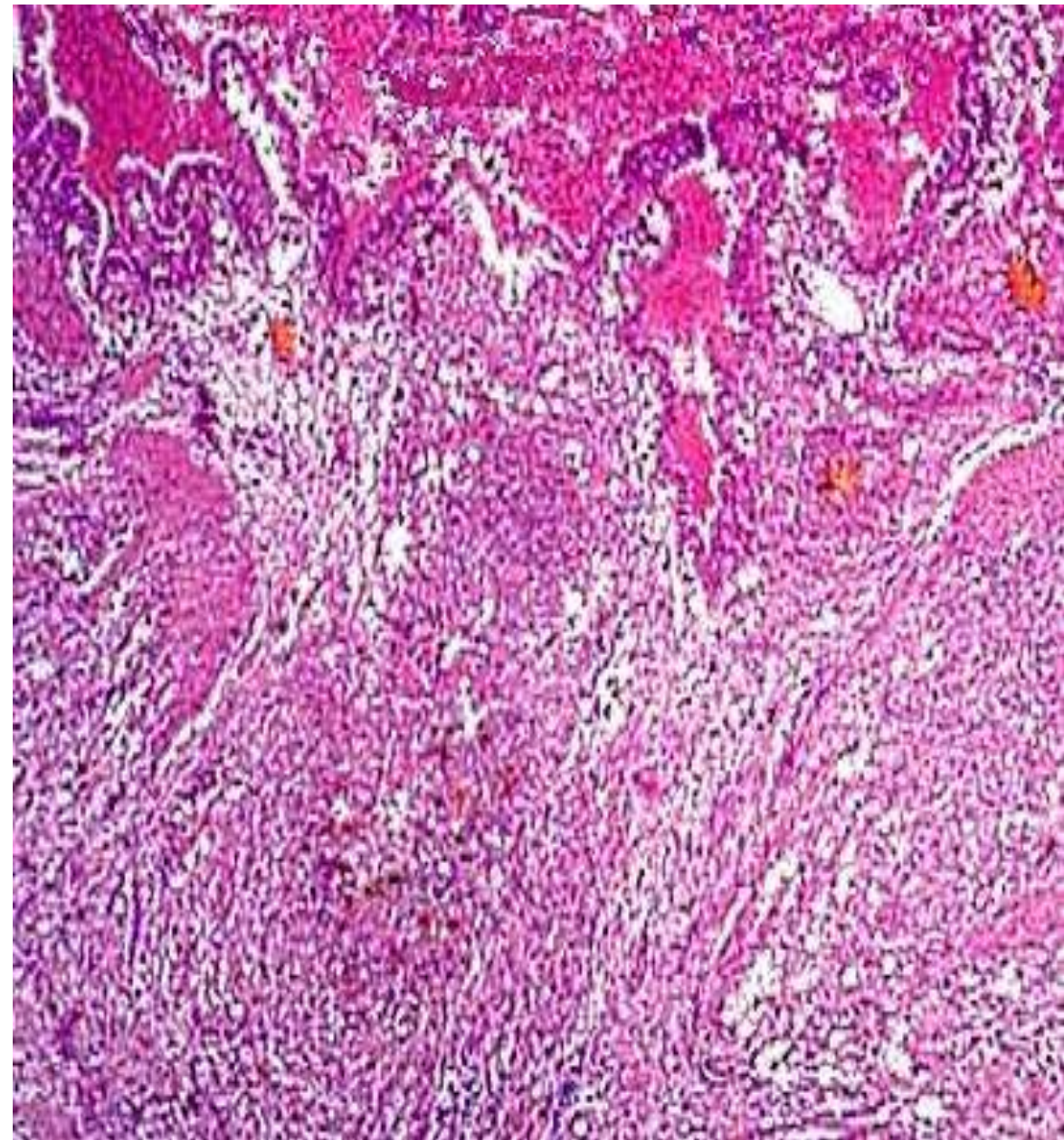


Cholesterol stones

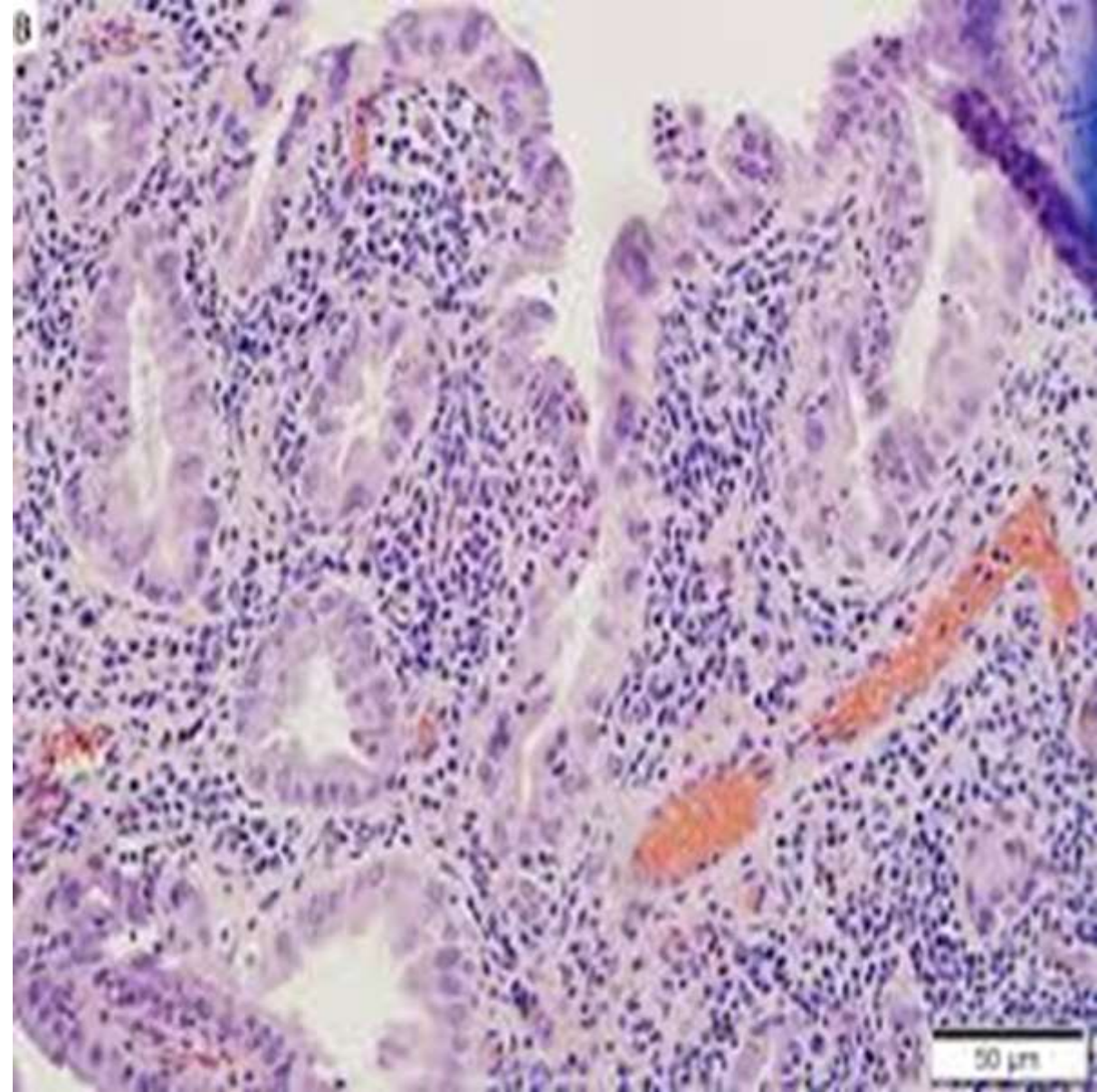
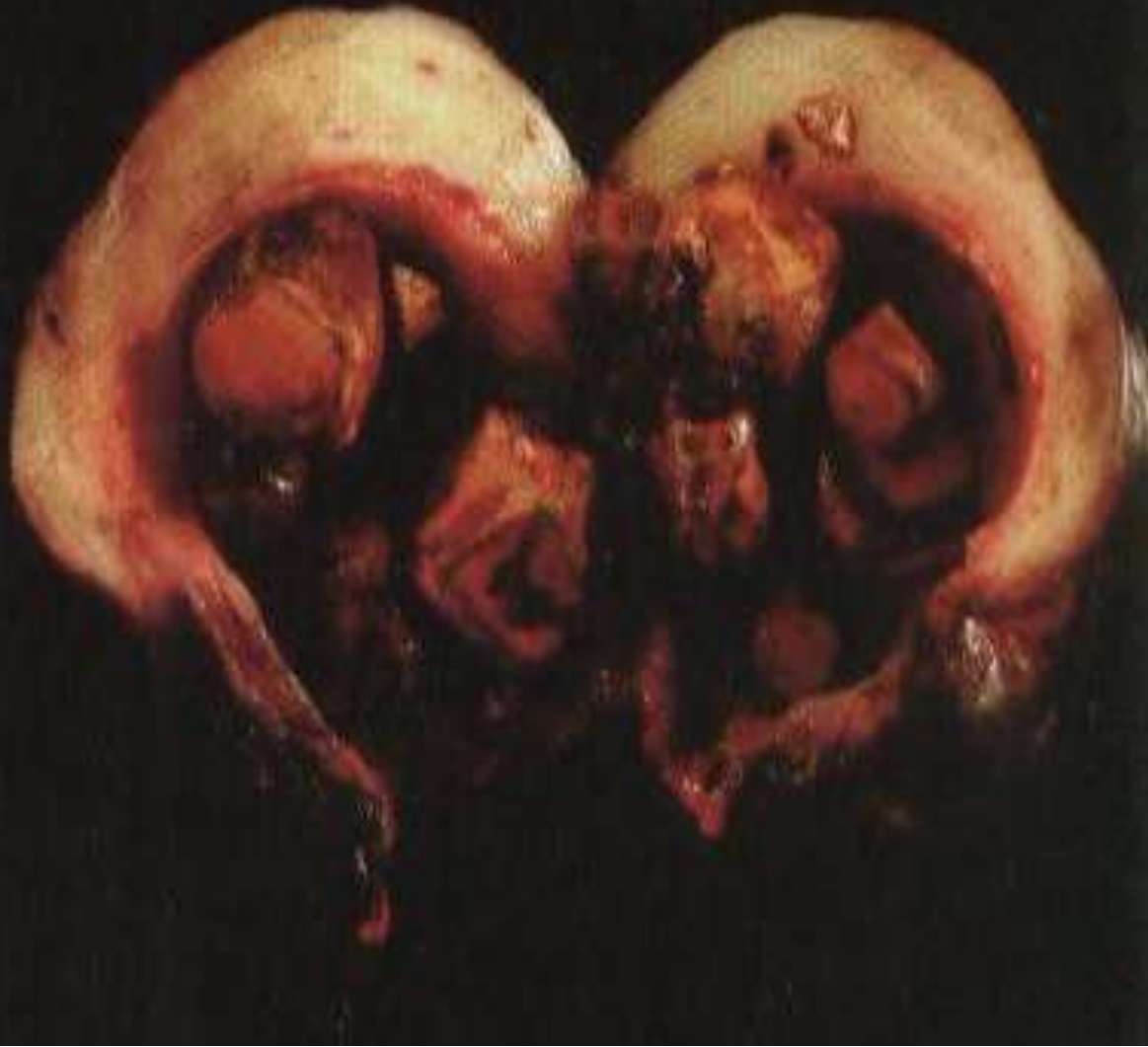


Pigmented gallstones

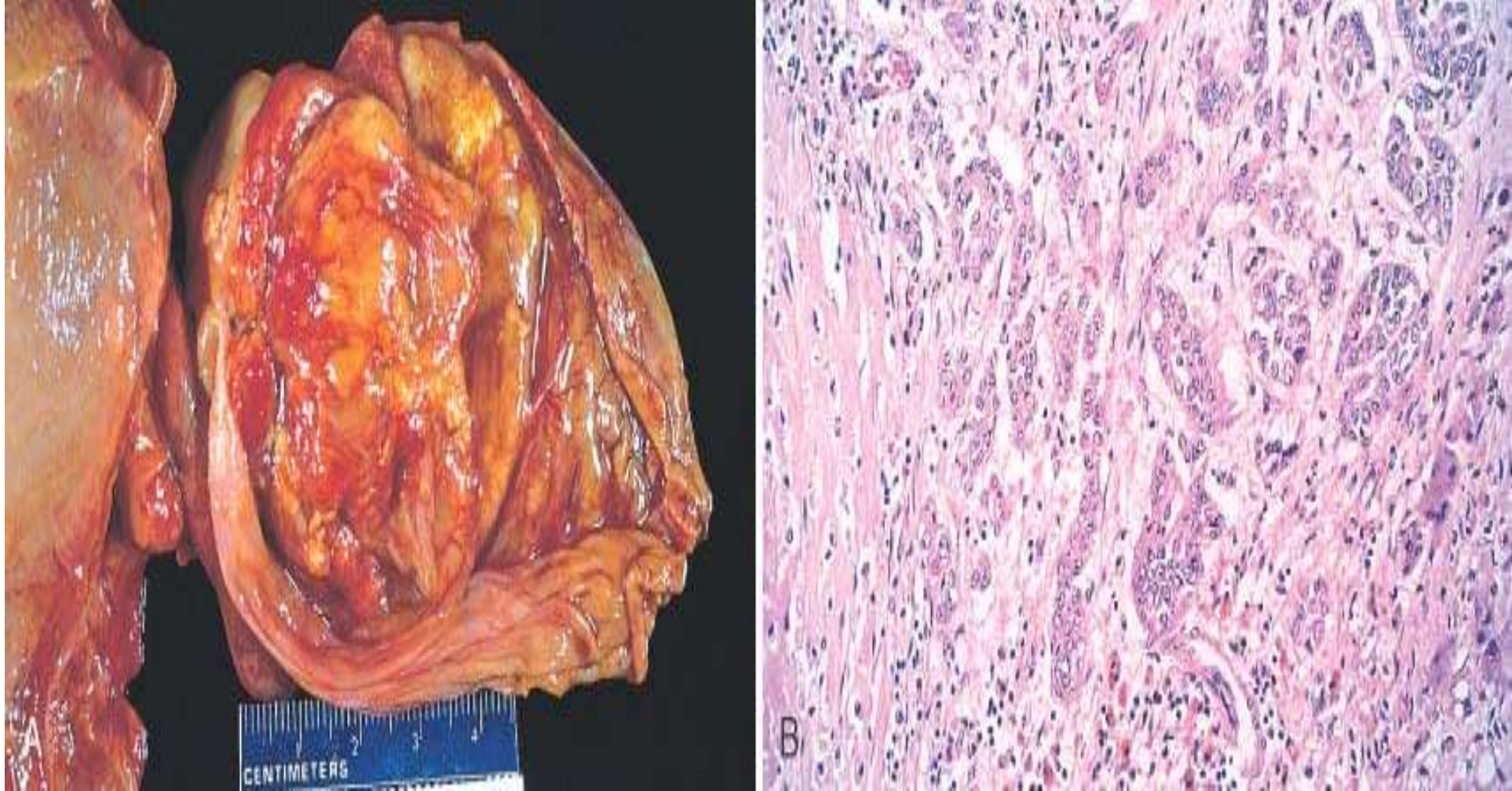




Acute cholecystitis:



Chronic cholecystitis:



**Adenocarcinoma of the
gallbladder**

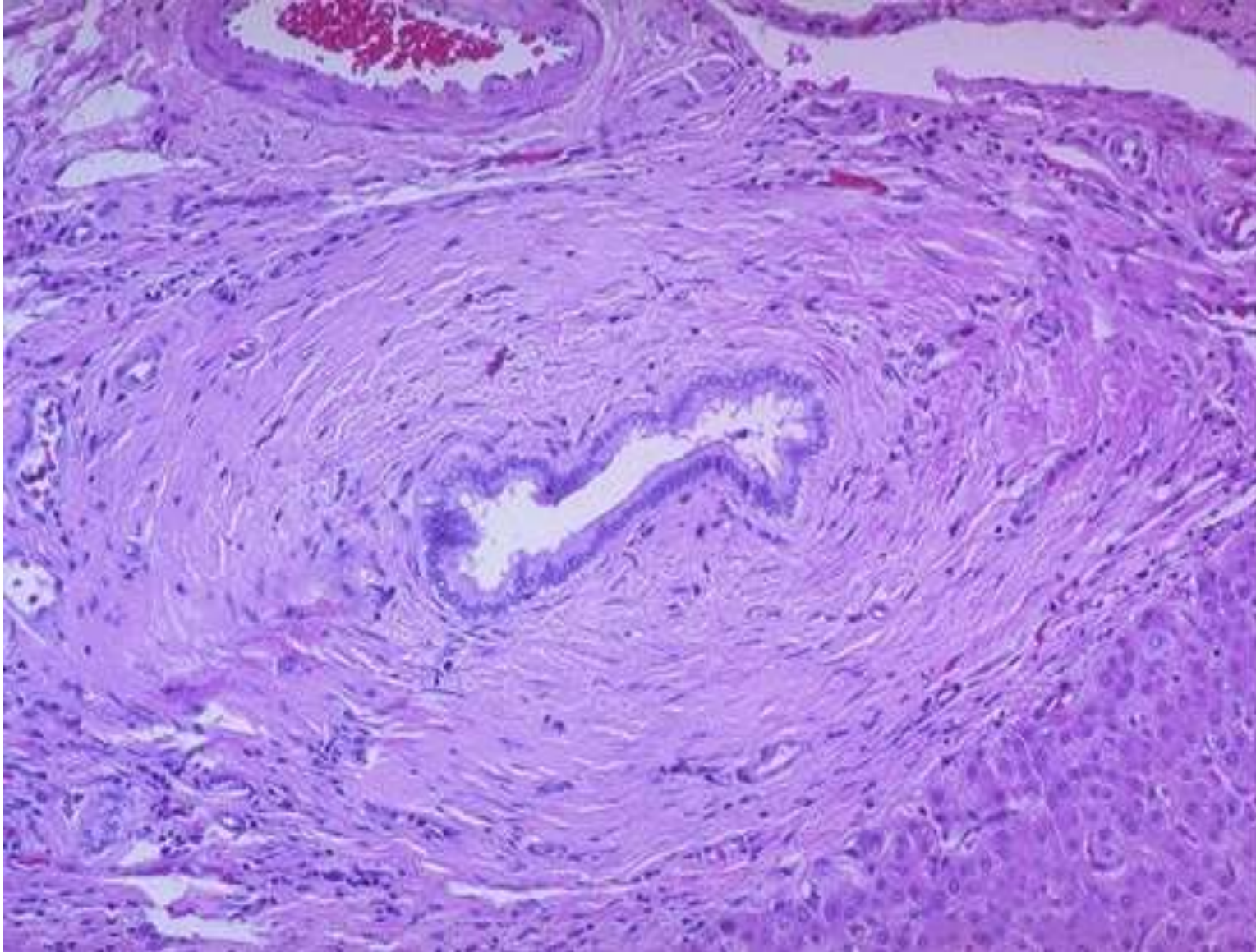
PBC bile duct damage



granulomas



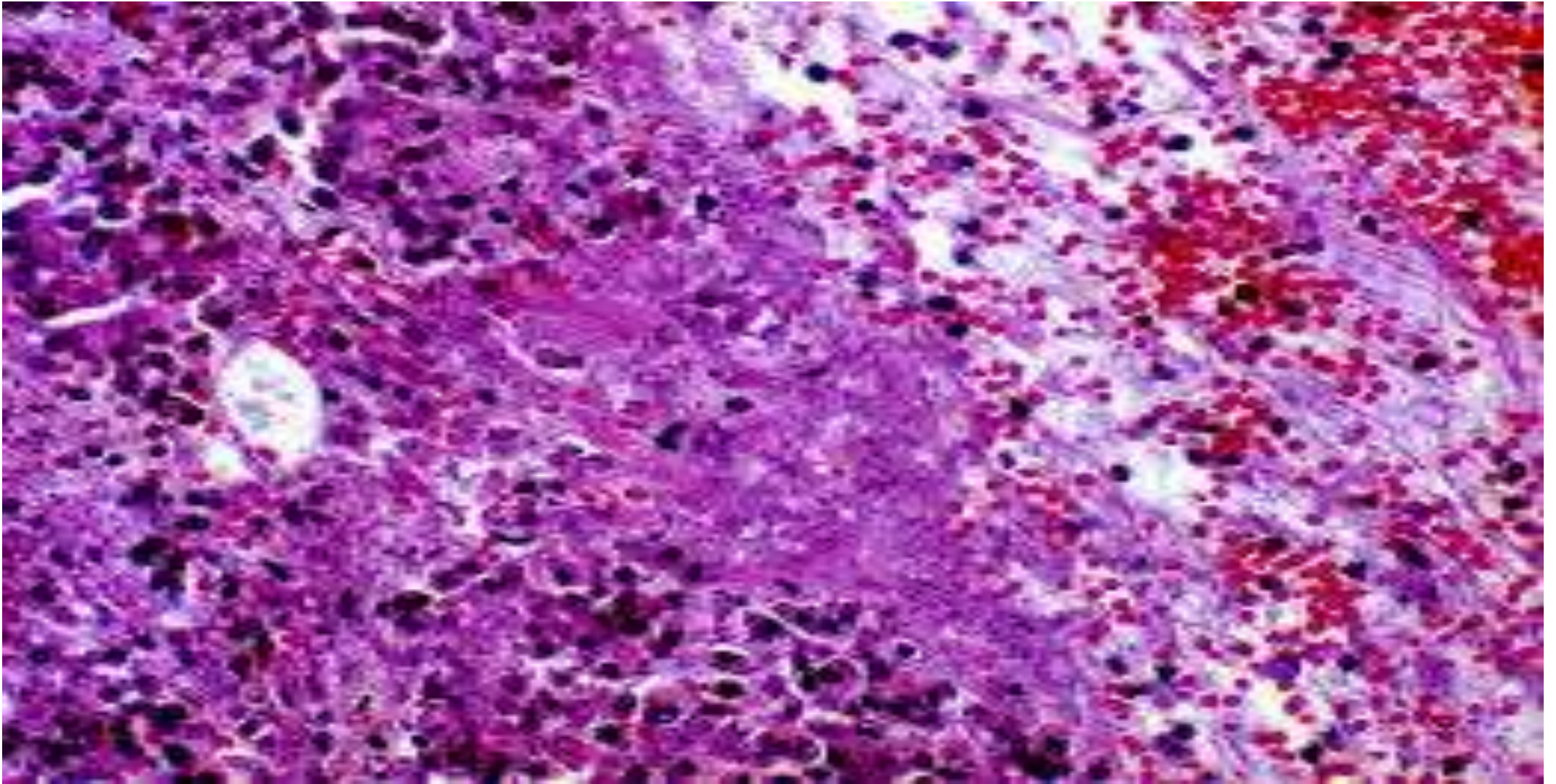
Primary sclerosing cholangitis

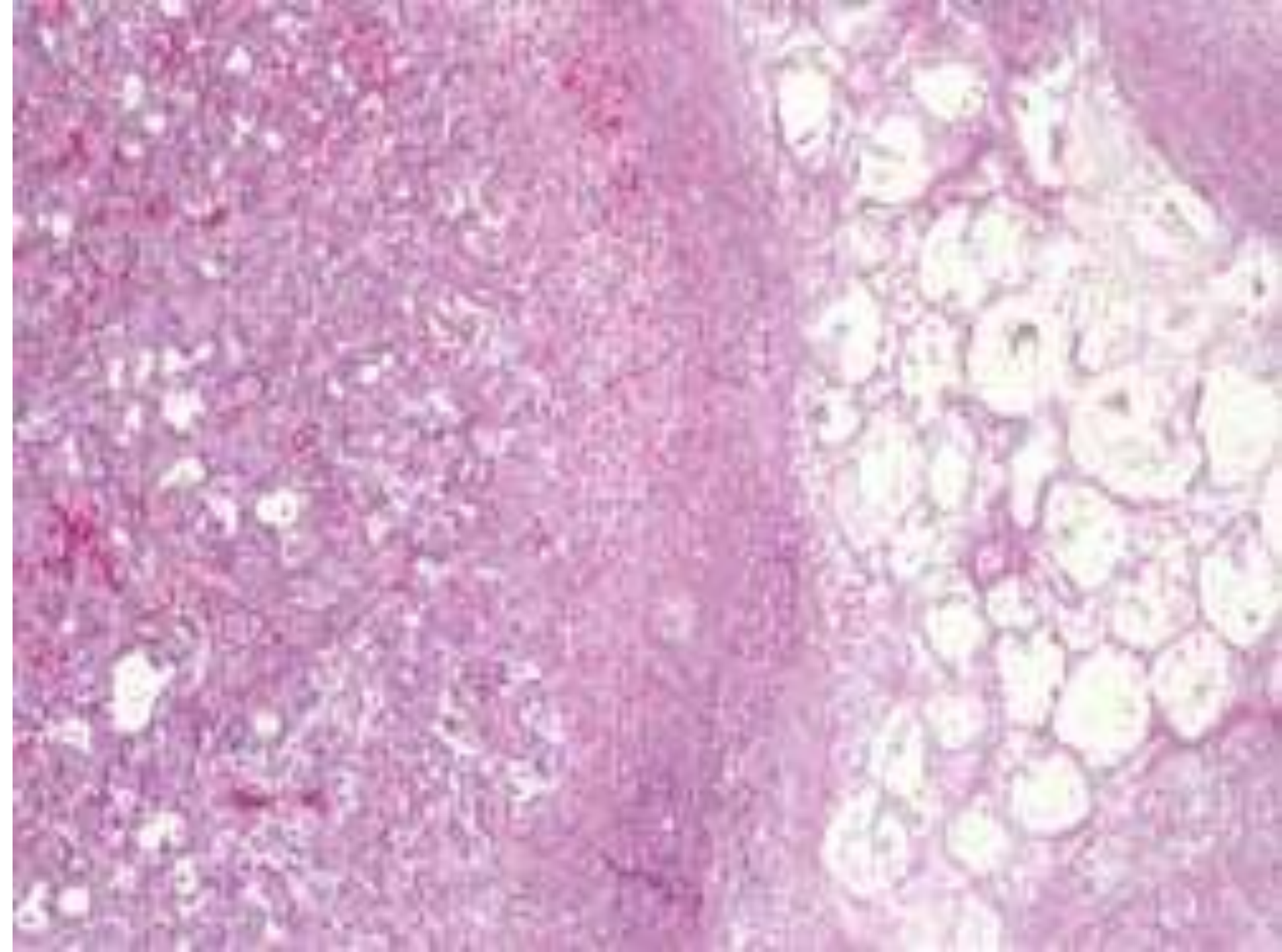


Acute hemorrhagic pancreatitis.



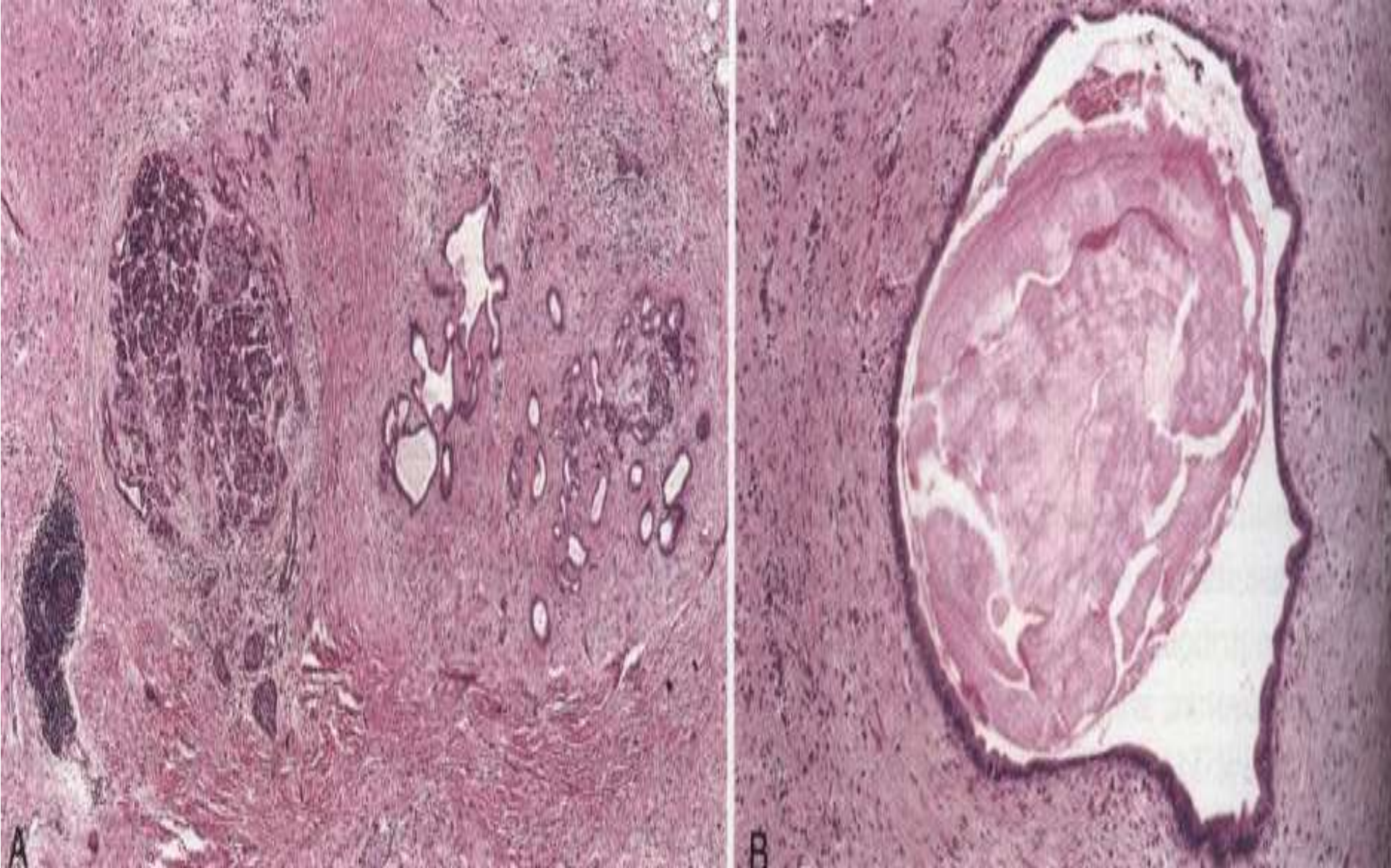
Acute hemorrhage with coagulation necrosis
of acinar tissue.





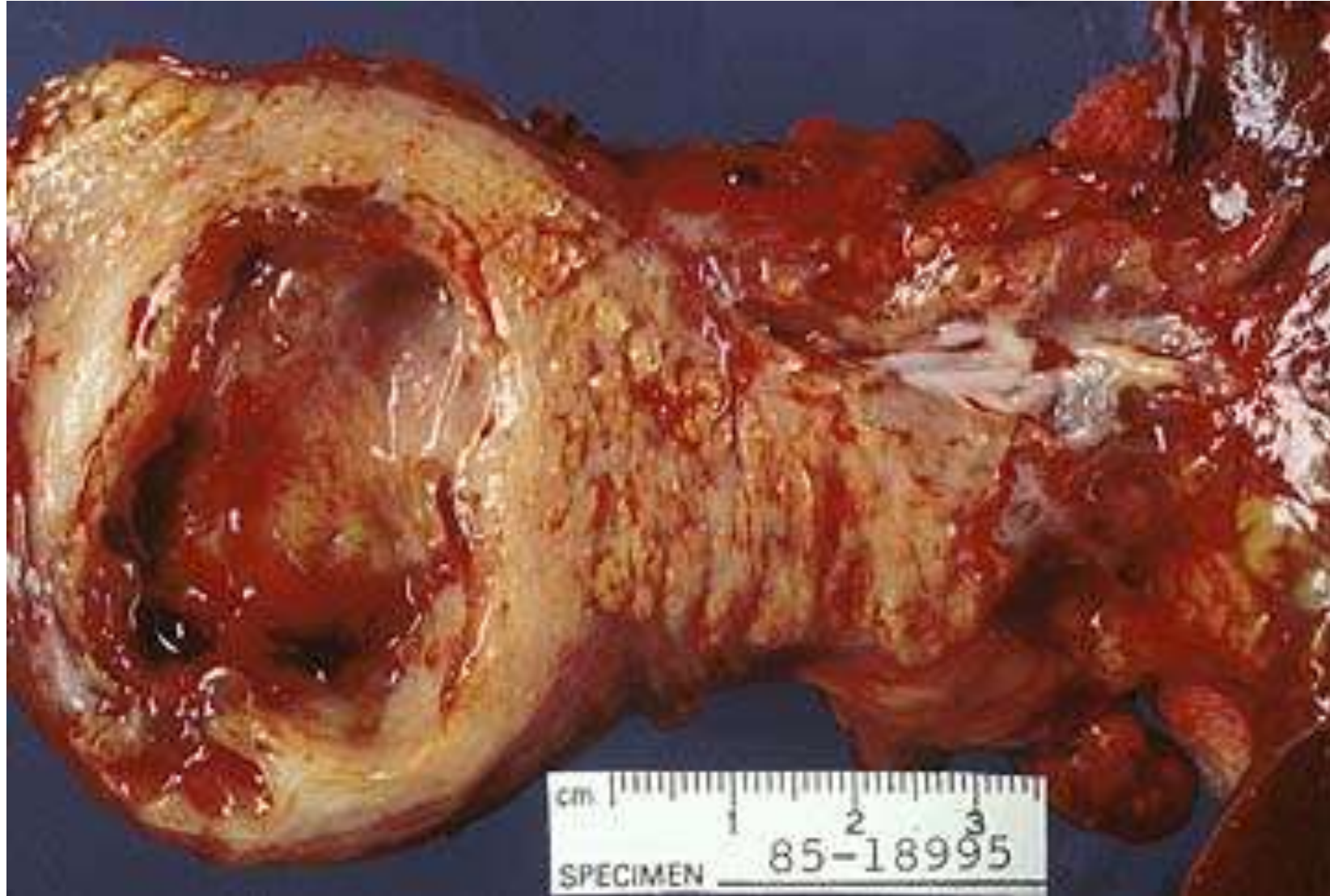
Enzymatic fat necrosis





Chronic pancreatitis

Pancreatic Pseudocyst



Adenocarcinoma replacing tail and body of pancreas. **Whitish firm mass**



Malignant glands in pancreatic adenocarcinoma.

