REKAP DAFTAR HADIR KULIAH PAKAR & KM BLOK 8
SEMESTER GASAL TAHUN AKADEMIK 2020/2021
PERIODE : 21 OKTOBER - 20 NOVEMBER 2020

NO	NAMA DOSEN	DEPARTEMEN	JLH JAM RENCANA	BLOK 8 & PKM OKTOBER					REALISASI KP
				9	12	16	17	19	
1	dr. Silphia Novelyn, M.Biomed.	Anatomi	4	-	-	-	4	-	4
2	dr. Moskwadina Gultom, M.Pd.Ked.	Anatomi	4	4	-	-	-	-	4
3	Dr. Dra. Trini Suryowati, MS	Biokimia Kedokteran	4	4	-	-	-	-	4
4	dr. Frisca R. Batubara, M.Biomed.	Biomedik Dasar	4	-	4	-	-	-	4
5	dr. Frisca Angreni	Anatomi	4	-	4	-	-	-	4
6	dr. Kurniyanto, SpPD	Ilmu Penyakit Dalam	4	-	4	-	-	-	4
7	dr. Fajar L. Gultom, SpPA	Pato. Anatomi	4	-	-	4	-	-	4
8	dr. Suryo Wijoyo, SpKF, MH.Kes.	IKF & Medikolegal	4	-	-	4	-	-	4
9	dr. Tiroy Sari Bumi Simanjuntak, SpPD	Ilmu Penyakit Dalam	4	-	-	4	-	-	4
10	dr. Jumaini Andriana Sihombing, M.Pd.Ked.	Anatomi	4	-	-	-	-	4	4
11	dr. Gregorius Septayudha, SpRad.	Radiologi	4	-	-	-	-	4	4
12	dr. Mildi Felicia, SpA	Ilmu Kesehatan Anak	4	-	-	-	-	4	4
13	Dra. Lusia Sri Sunarti, MS	Mikrobiologi	4	-	-	-	4	-	4
TOTAL			52						52
PERSENTASI KEHADIRAN KULIAH PAKAR BLOK 8 & KM			100%						

Jakarta, 23 November 2020

Mengetahui
Manager P2SK,

Dra. Lusia Sri Sunarti, MS

Koordinator Blok 8,

dr. Kurniyanto, SpPD

Universitas Kristen Indonesia
Fakultas Kedokteran

SURAT KEPUTUSAN
No. : 132/UKI.F5.D/HKP.3.5.6/2020
tentang

PENUGASAN TENAGA AKADEMIK DALAM MEMBERIKAN KULIAH PAKAR PIMPINAN FAKULTAS KEDOKTERAN UNIVERSITAS KRISTEN INDONESIA

MENIMBANG : Bahwa untuk kelancaran proses belajar mengajar dan meningkatkan mutu pendidikan di FKUKI diperlukan penugasan tenaga akademik FKUKI untuk memberikan Kuliah Pakar

MENGINGAT :
1. Peraturan Pemerintah No. 60 tahun 1999 tentang Pendidikan Tinggi
2. Surat Keputusan Dekan FKUKI No. 53/SK/FKUKI/11.2006 tanggal 21 November 2006 tentang Pemberlakuan Kurikulum Berbasis Kompetensi (KBK) di FKUKI
3. Surat Keputusan Rektor UKI No. 90/UKI.R/SK/SDM.8/2018 tentang pengangkatan Dekan Fakultas Kedokteran UKI
4. Surat keputusan pengangkatan sebagai tenaga akademik

MEMUTUSKAN

MENETAPKAN :
1. Penugasan dalam memberikan Kuliah Pakar :

Nama	dr. Fajar L. Gultom, SpPA
Departemen	Patologi Anatomi
Blok	8 (Gastrointestinal, Hepatobilier, dan Pankreas)
Judul Materi	Gambaran patologi anatomi kelainan saluran cerna
Semester	gasal 2020/2021
Kelas	A : 0,21 SKS
	B : 0,21 SKS
SKS	0,42 SKS

2. Apabila dikemudian hari ternyata terdapat kekeliruan dalam Surat Keputusan ini akan diperbaiki sebagaimana mestinya

Asli Surat Keputusan ini disampaikan kepada yang bersangkutan untuk diketahui

Ditetapkan di : Jakarta
Pada tanggal : 10 September 2020
Dekan,

Dr. dr. Robert Hotman Sirait, Sp.An.
NIP. UKI. 031 545

Tembusan:
1. Rektor UKI
2. Wakil Dekan Bidang Akademik FKUKI

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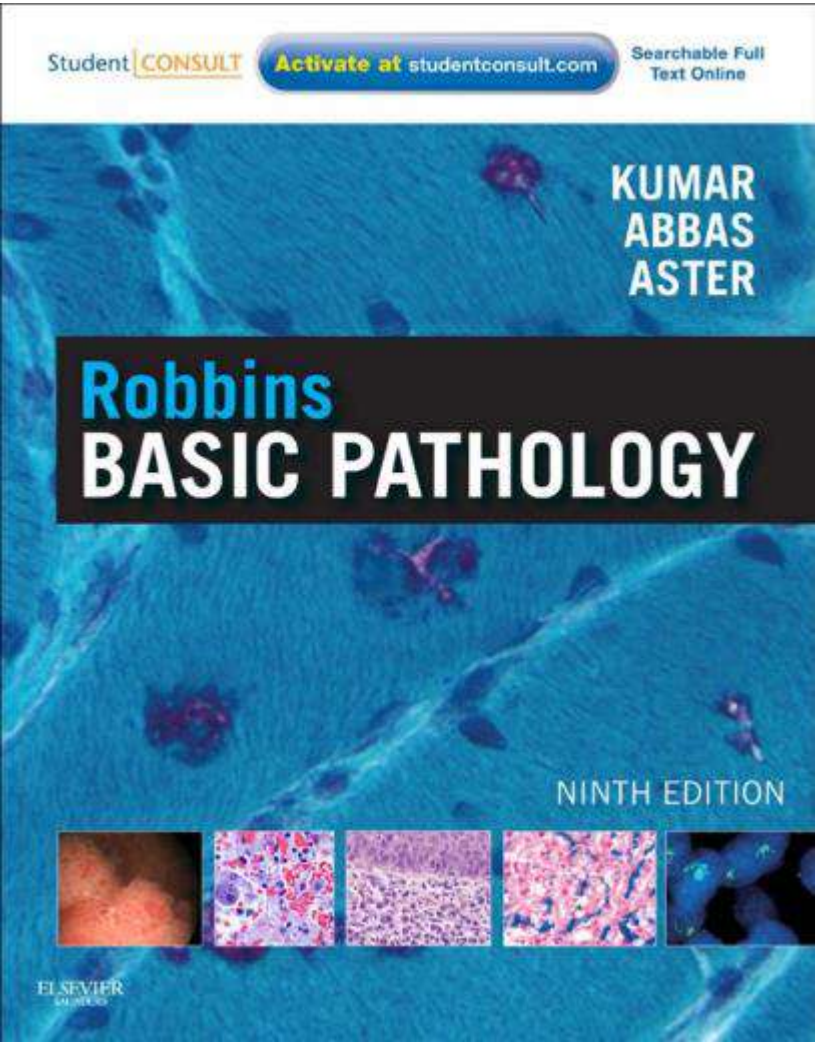
Liver, Gallbladder and Pancreas Pathology

Fajar L. Gultom
Departemen Patologi Anatomik
Fakultas Kedokteran
Universitas Kristen Indonesia
Desember 2020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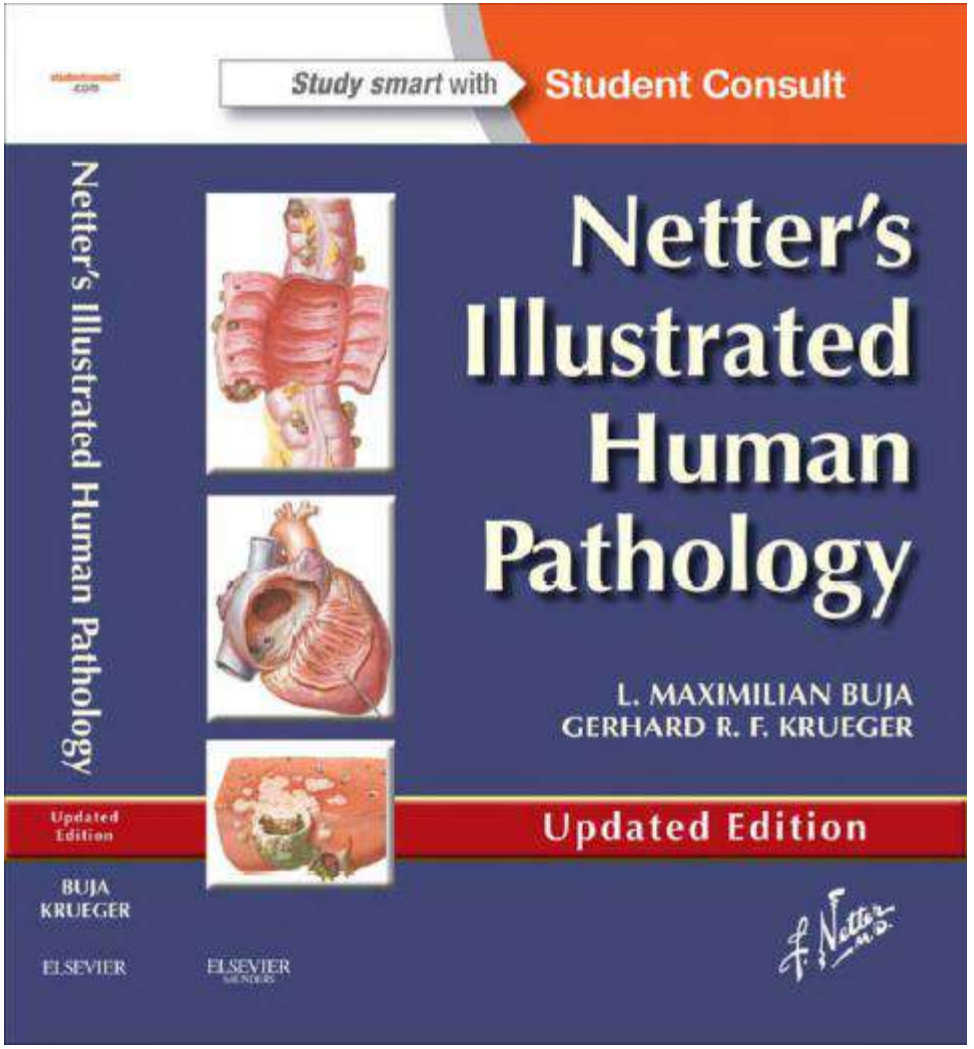
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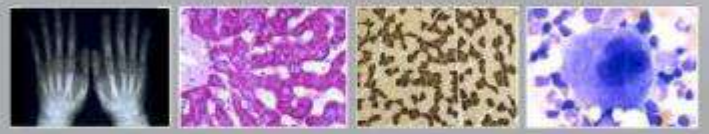
Robbins and Cotran ATLAS OF PATHOLOGY

THIRD EDITION



ELSEVIER

STANDAR KOMPETENSI
DOKTER INDONESIA

KONSIL KEDOKTERAN INDONESIA
Indonesian Medical Council
Jakarta 2012

Hepar		
49	Hepatitis A	4A
50	Hepatitis B	3A
51	Hepatitis C	2
52	Abses hepar amoeba	3A
53	Perlemakan hepar	3A
54	Sirosis hepatis	2
55	Gagal hepar	2
56	Neoplasma hepar	2

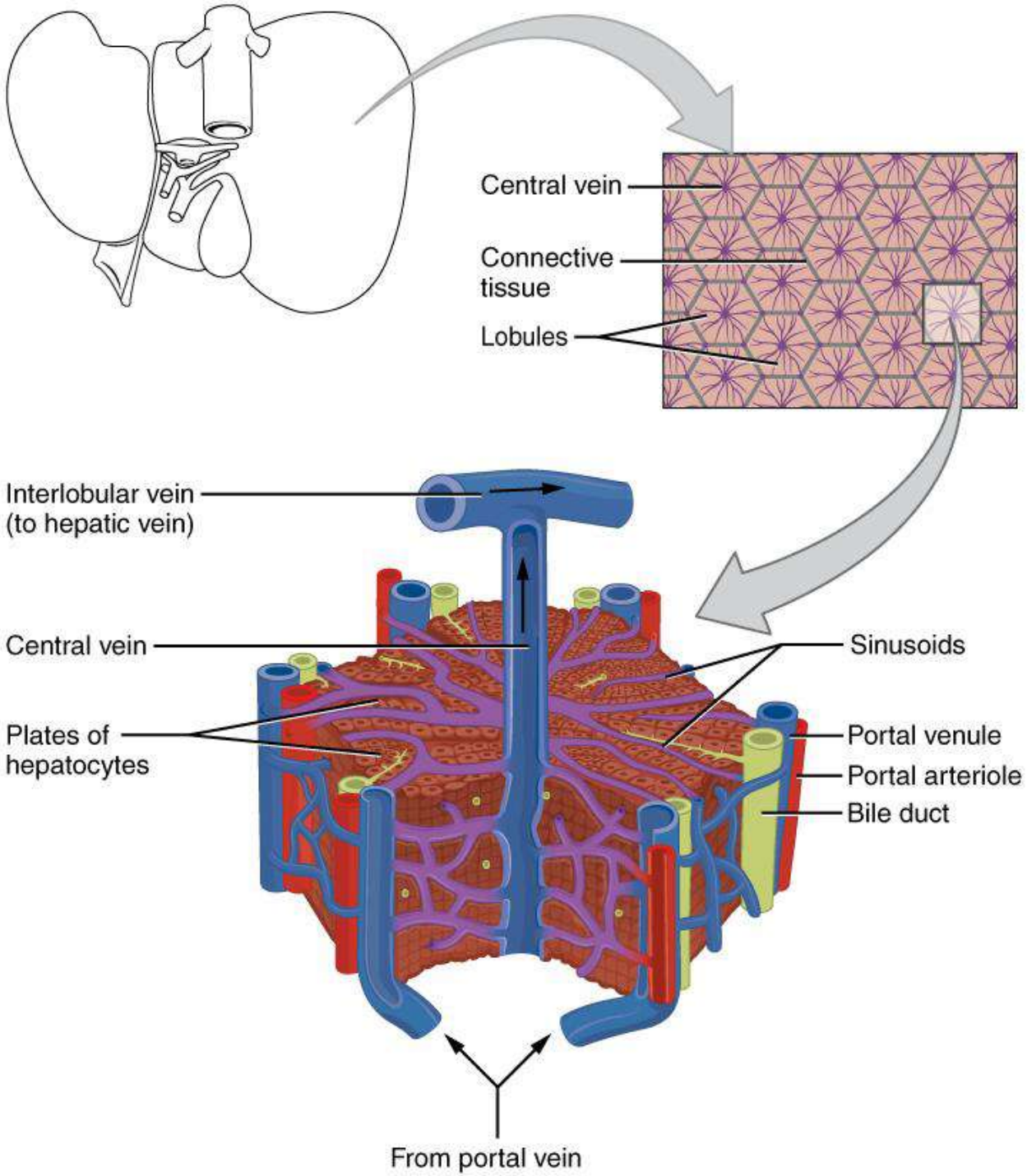
STANDAR KOMPETENSI
DOKTER INDONESIA

KONSIL KEDOKTERAN INDONESIA
Indonesian Medical Council
Jakarta 2012

Kandung Empedu, Saluran Empedu, dan Pankreas		
57	Kolesistitis	3B
58	Kole(doko)litiasis	2
59	Empiema dan hidrops kandung empedu	2
60	Atresia biliaris	2
61	Pankreatitis	2
62	Karsinoma pankreas	2

Liver

- Normal 1400 – 1600 gr.
- Dual blood supply: portal vein (60%), hepatic artery (40%).
- Hepatic microarchitecture → lobular model.
- Lobulus – 1-2 mm Ø.
- Hexagonal structures.


Central vein
Connective tissue
Lobules
Interlobular vein (to hepatic vein)
Central vein
Plates of hepatocytes
Sinusoids
Portal venule
Portal arteriole
Bile duct
From portal vein

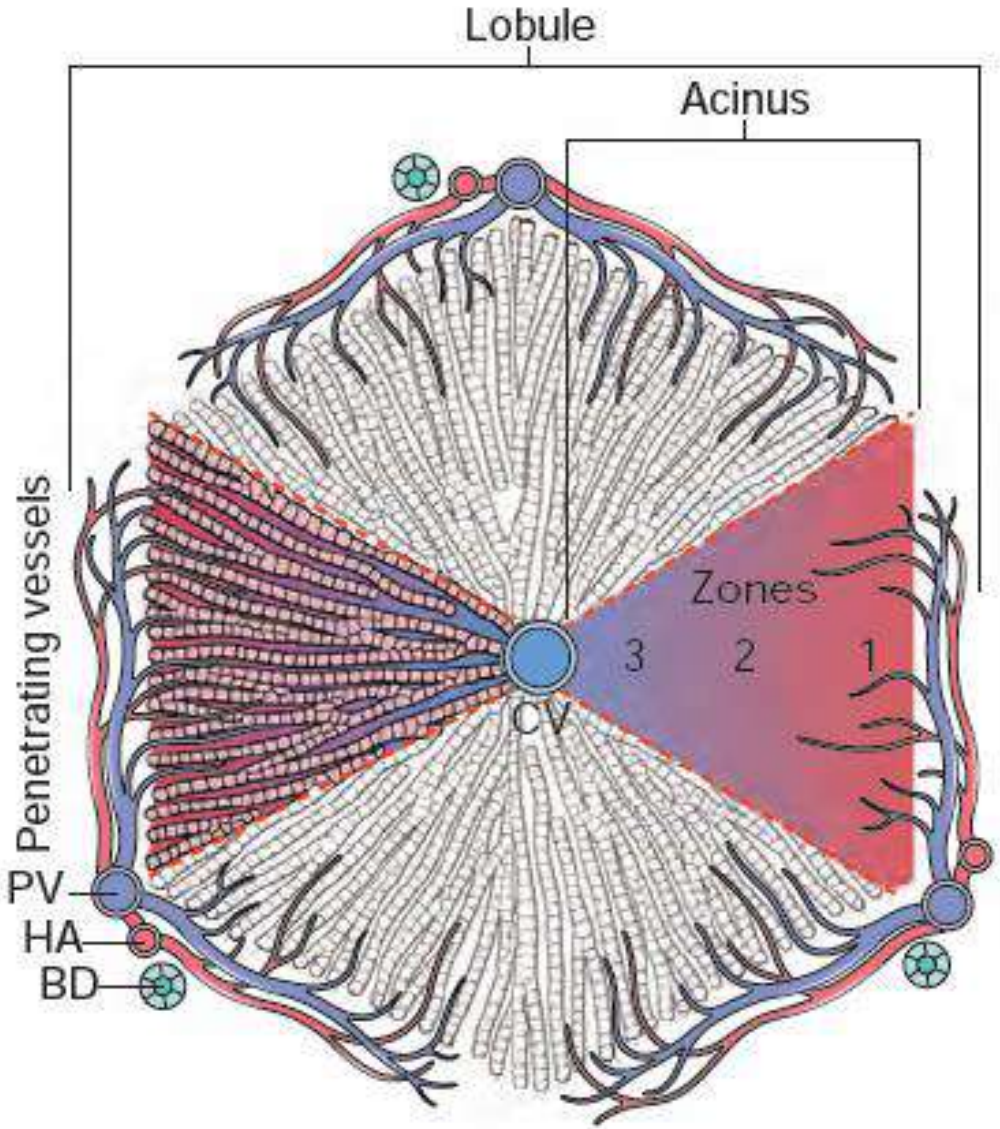
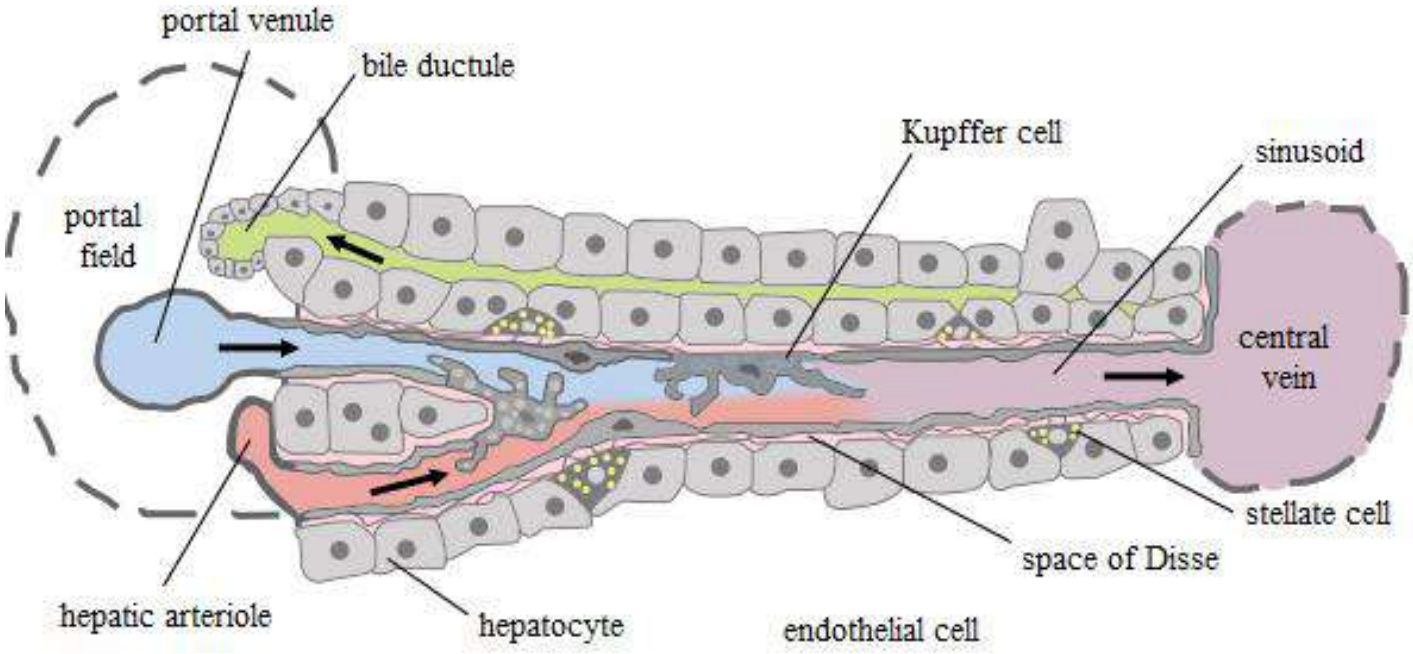


Figure 18-1 Models of liver anatomy. In the lobular model, the terminal hepatic vein (CV) is at the center of a "lobule," while the portal tracts (PV) are at the periphery. Pathologists often refer to the regions of the parenchyma as "periportal" and "centrilobular." In the acinar model, on the basis of blood flow, three zones can be defined, zone 1 being the closest to the blood supply and zone 3 being the farthest. BD, Bile duct; HA, hepatic artery.

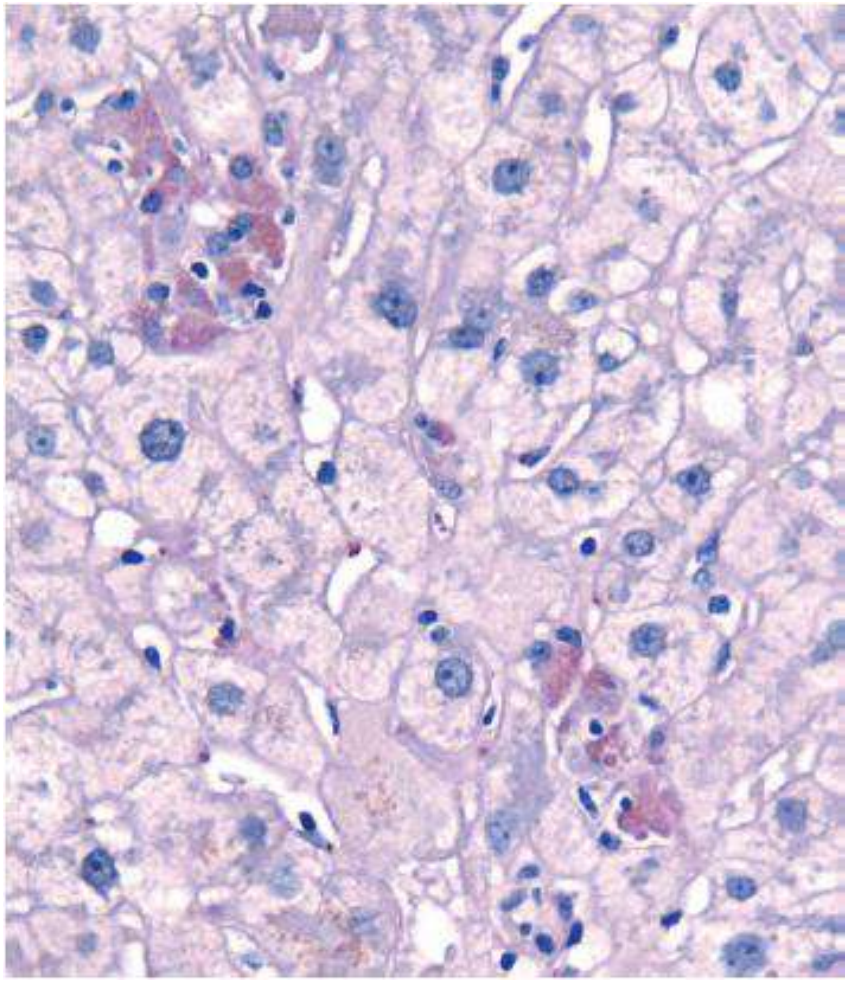
Robbins, Pathologic Basis of Disease 9th Ed, 2015

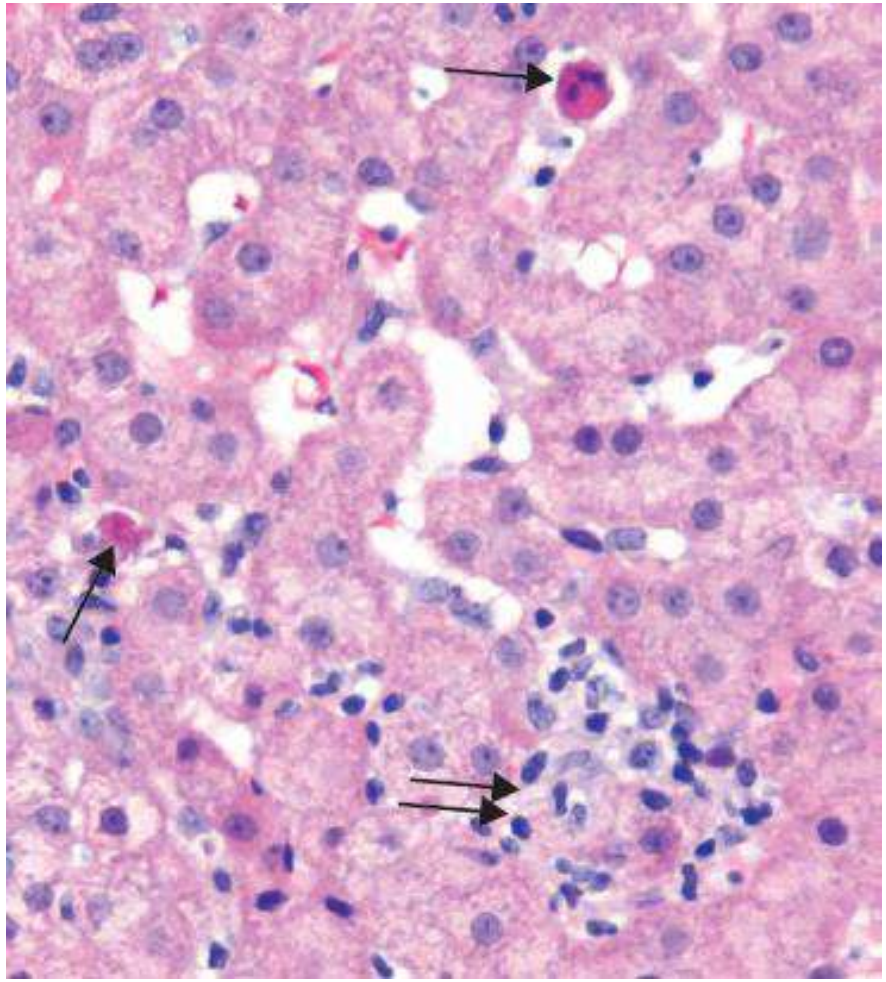


- Hepatocyte – anastomosing sheets/ plates
- Between plate – sinusoids – Kupffer cells
- Space of Disse: Hepatic Stellate Cell (HSC)

Mechanism of Injury and Repair

- **Potential reversible** changes: accumulation of fat (steatosis), bilirubin (cholestasis)
- **Irreversible**: necrosis or apoptosis
- Hepatocyte necrosis – **swells** – rupture – macrophages
- Hepatocyte apoptosis – programmed cell death – **shrinkage** – pyknosis – karyorrhexis – apoptotic bodies





Necrosis VS Apoptosis

Robbins, Pathologic Basis of Disease 9th Ed, 2015

Table 18-1 Laboratory Evaluation of Liver Disease

Test Category	Serum Measurement
Hepatocyte integrity	Cytosolic hepatocellular enzymes†
	Serum aspartate aminotransferase (AST)
	Serum alanine aminotransferase (ALT)
	Serum lactate dehydrogenase (LDH)
Biliary excretory function	Substances normally secreted in bile†
	Serum bilirubin
	Total: unconjugated plus conjugated
	Direct: conjugated only
	Urine bilirubin
	Serum bile acids
	Plasma membrane enzymes (from damage to bile canaliculus)†
	Serum alkaline phosphatase
	Serum γ-glutamyl transpeptidase (GGT)
Hepatocyte synthetic function	Proteins secreted into the blood
	Serum albumin‡
	Coagulation factors:
	Prothrombin (PT) and partial thromboplastin (PTT) times (fibrinogen, prothrombin,factors V, VII, IX,and X)
	Hepatocyte metabolism
	Serum ammonia†
	Aminopyrine breath test (hepatic demethylation)‡

†Increased in liver disease.
‡Decreased in liver disease.

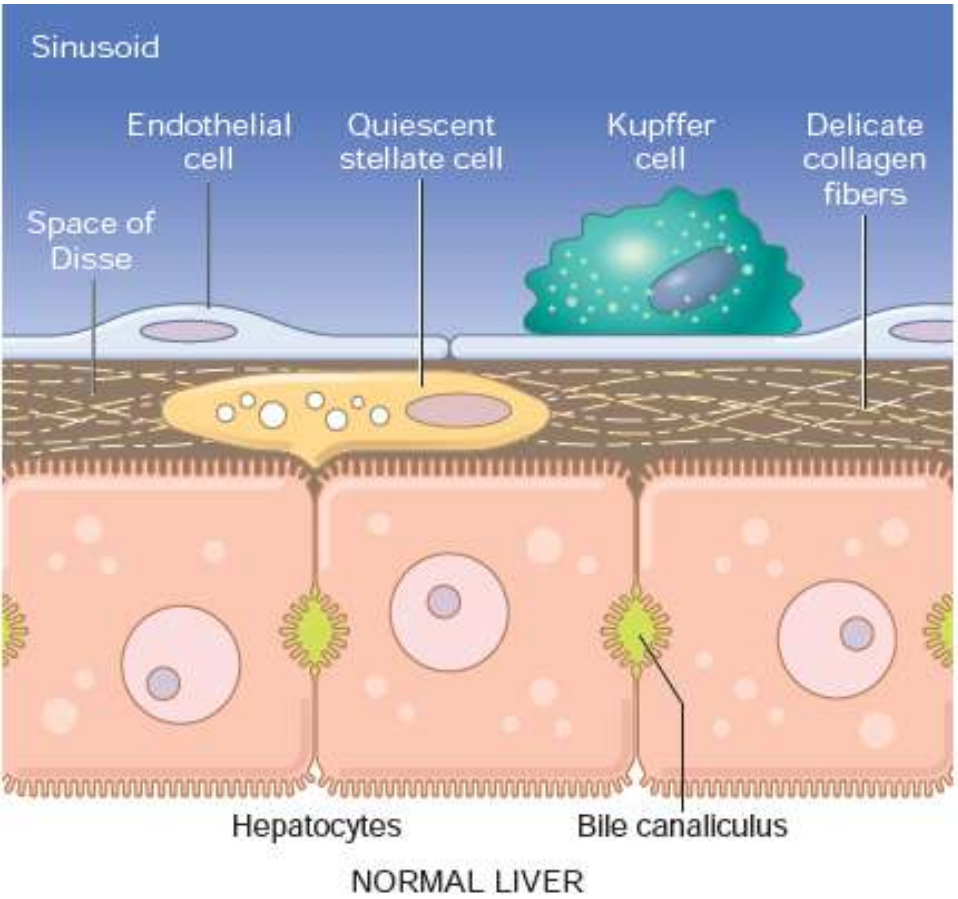
Robbins, Pathologic Basis of Disease 9th Ed, 2015

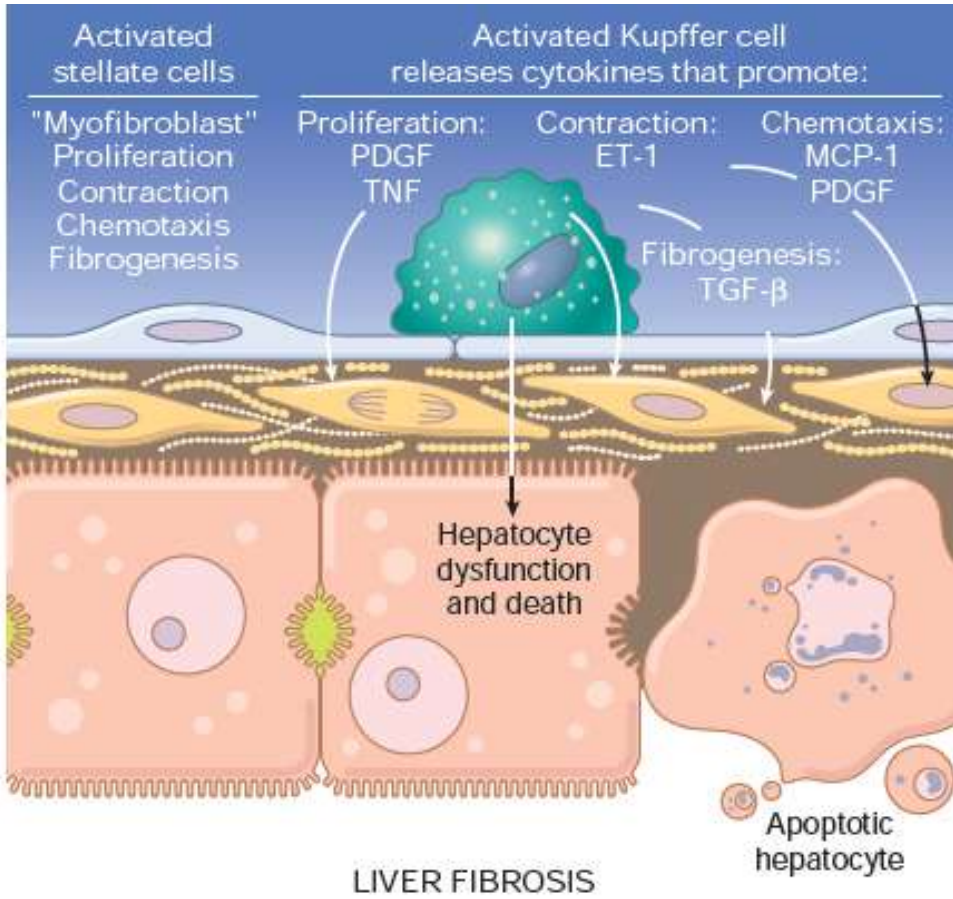
Injury n Repair

- Regeneration of lost hepatocytes – mitotic replication
- Hepatocytes – **stem cell-like** – ability to replicate in chronic injury
- Eventually → chronic disease – replicative senescence

Scar formation n Regression

- Principal cell – Hepatic Stellate Cell (HSC)
- Quiescent form – lipid storing (vit A) cell
- Acute n chronic injury – activated – highly fibrogenic myofibroblast
- Stimuli for activation: ROS (Reactive Oxygen Species), growth factors, cytokines (TNF, IL-1), TGF-β.





Robbins, Pathologic Basis of Disease 9th Ed, 2015

Liver disease

- Metabolic, toxic, microbial, circulatory and neoplastic.
- **Major:**

  Viral hepatitis

  Nonalcoholic fatty liver disease (NAFLD)
  Alcoholic liver disease

  Hepatocellular carcinoma (HCC)

Infectious Disorder

- Hepatitis – confusing word??
- Viral hepatitis – autoimun hepatitis
- EBV, Cytomegalovirus, Herpes simplex..
- **Hepatotropic virus A, B, C, D, E**
- Hep A n E – **A**cut**E**, **E**ndemic
- Hep B – **B**lood, **B**irthing, ***B****onking*
- Hep C – **C**hronic
- Hep D – **D**efective
- Biopsy – grading n staging → antiviral

Table 18-3 The Hepatitis Viruses

Virus	Hepatitis A	Hepatitis B	Hepatitis C	Hepatitis D	Hepatitis E
Type of virus	ssRNA	partially dsDNA	ssRNA	Circular defective ssRNA	ssRNA
Viral family	Hepatovirus; related to picornavirus	Hepadnavirus	Flaviviridae	Subviral particle in Deltaviridae family	Hepevirus
Route of transmission	Fecal-oral (contaminated food or water)	Parenteral, sexual contact, perinatal	Parenteral; intranasal cocaine use is a risk factor	Parenteral	Fecal-oral
Mean incubation period	2 to 6 weeks	2 to 26 weeks (mean 8 weeks)	4 to 26 weeks (mean 9 weeks)	Same as HBV	4 to 5 weeks
Frequency of chronic liver disease	Never	5%-10%	>80%	10% (co-infection); 90%-100% for superinfection	In immunocompromised hosts only
Diagnosis	Detection of serum IgM antibodies	Detection of HBsAg or antibody to HBcAg; PCR for HBV DNA	3rd-generation ELISA for antibody detection; PCR for HCV RNA	Detection of IgM and IgG antibodies; HDV RNA serum; HDAg in liver	Detection of serum IgM and IgG antibodies; PCR for HEV RNA

dsDNA, Double-stranded DNA; ELISA, enzyme-linked immunosorbent assay; HBcAg, hepatitis B core antigen; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; HCV, hepatitis C virus; HDAg, hepatitis D antigen; HDV, hepatitis D virus; HEV, hepatitis E virus; IV, intravenous; PCR, polymerase chain reaction; ssRNA, single stranded RNA.

From Washington K: Inflammatory and infectious diseases of the liver. In Iacobuzio-Donahue CA, Montgomery EA (eds): Gastrointestinal and Liver Pathology. Philadelphia, Churchill Livingstone; 2005.

Robbins, Pathologic Basis of Disease 9th Ed, 2015

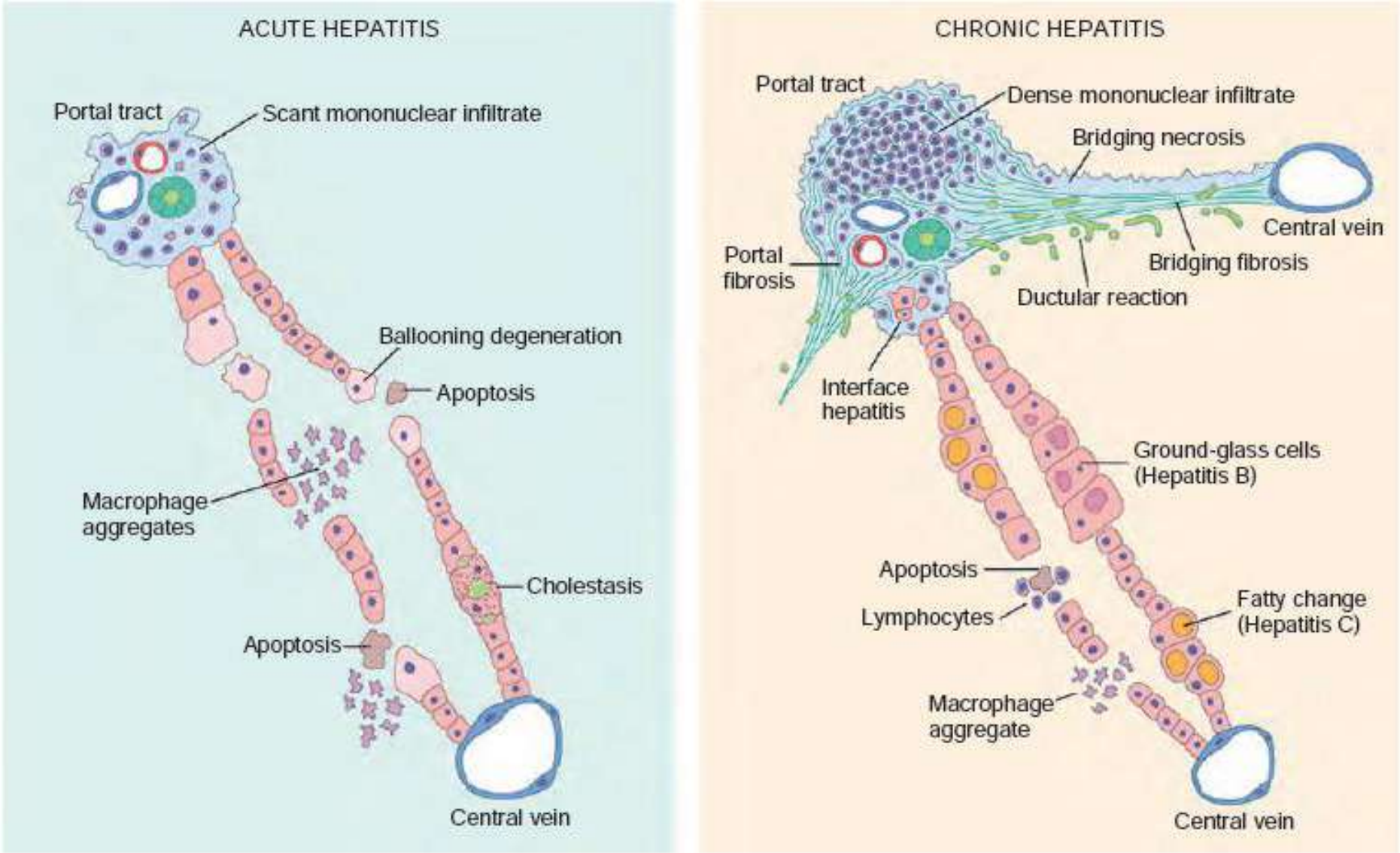


Figure 18-14 Diagrammatic representation of the morphologic features of acute and chronic hepatitis. Notice that there is very little portal mononuclear infiltration in acute hepatitis (or sometimes none at all), while in chronic hepatitis the portal infiltrates are dense and prominent—the defining change of chronic hepatitis. Bridging necrosis and fibrosis is shown only for chronic hepatitis, but bridging necrosis may also occur in more severe acute hepatitis. Ductular reactions in chronic hepatitis are minimal in early stages of scarring, but become extensive in late stage disease.

Robbins, Pathologic Basis of Disease 9th Ed, 2015

HIV and Chronic Viral Hepatitis

- Similar transmission – similar high risk patient
- US:

  10% HIV-infected co-infected HBV

  25% HIV-infected co-infected HCV
- Morbidity n mortality – successful anti-HIV th/

Alcoholic – Non Alcoholic Fatty Liver Disease (NAFLD)

- Alcohol – fatty liver – steatosis – steatohepatitis – cirrhosis
- Non alcoholic – metabolic syndrome

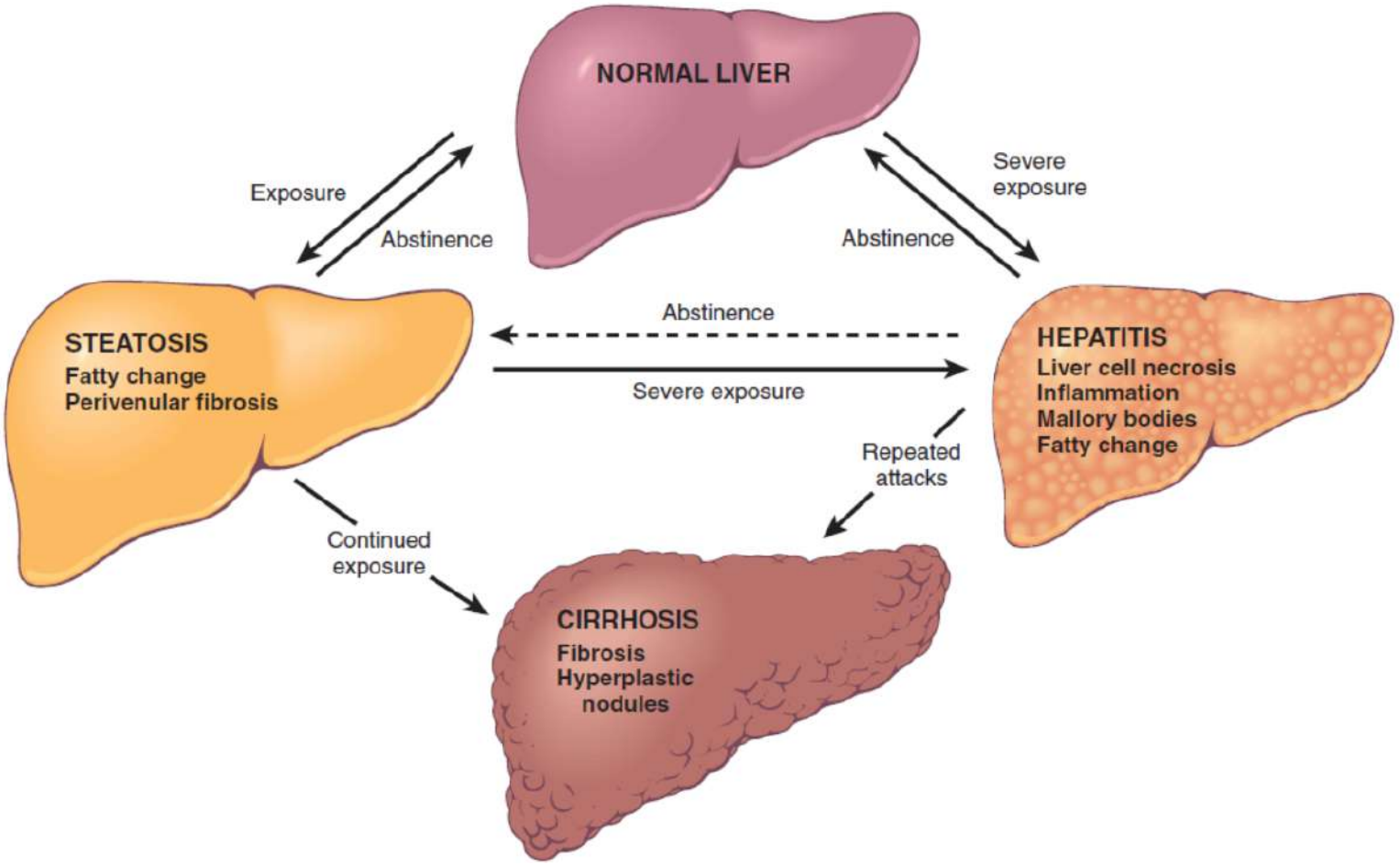
  insulin resistance, obesity, DM, HT, dyslipidemia

Effects of Alcohol

- Moderate amounts not injurious.
- **Excessive – serious physical and psychological damage.**

Despite all the attention given to illicit drugs such as cocaine and heroin, alcohol abuse is a far more widespread hazard and claims many more lives. Fifty percent of adults in the Western world drink alcohol, and about 5% to 10% have chronic alcoholism. It is estimated that there are more than 10 million chronic alcoholics in the United States and that alcohol consumption is responsible for more than 100,000 deaths annually. More than 50% of these deaths result from accidents caused by drunken driving and alcohol-related homicides and suicides, and about 15,000 annual deaths are a consequence of cirrhosis of the liver. Worldwide, alcohol accounts for approximately 1.8 million deaths per year (3.2% of all deaths).

Alcoholic Liver Disease



Robbins, Pathologic Basis of Disease 9th Ed, 2015

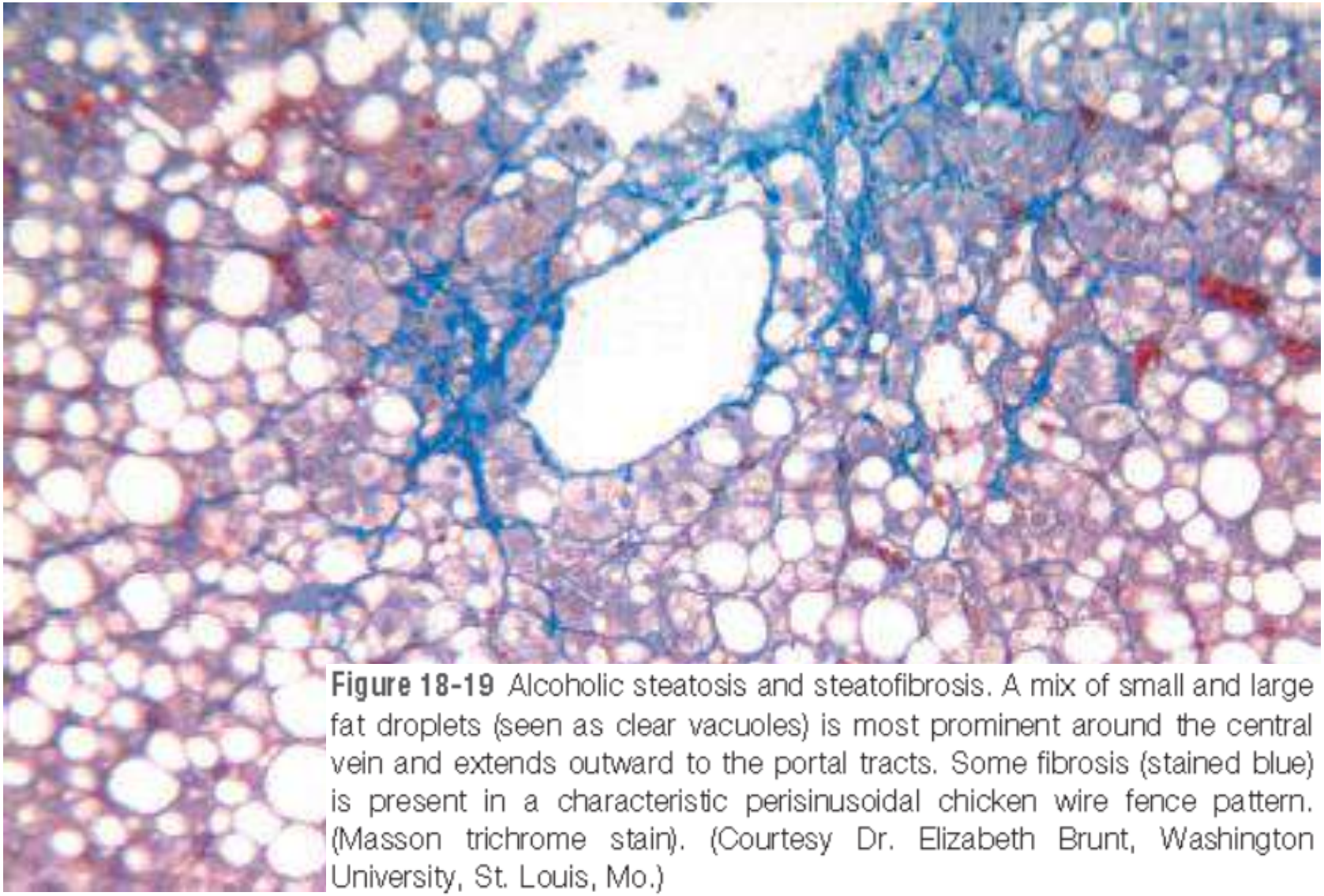


Figure 18-19 Alcoholic steatosis and steatofibrosis. A mix of small and large fat droplets (seen as clear vacuoles) is most prominent around the central vein and extends outward to the portal tracts. Some fibrosis (stained blue) is present in a characteristic perisinusoidal chicken wire fence pattern. (Masson trichrome stain). (Courtesy Dr. Elizabeth Brunt, Washington University, St. Louis, Mo.)

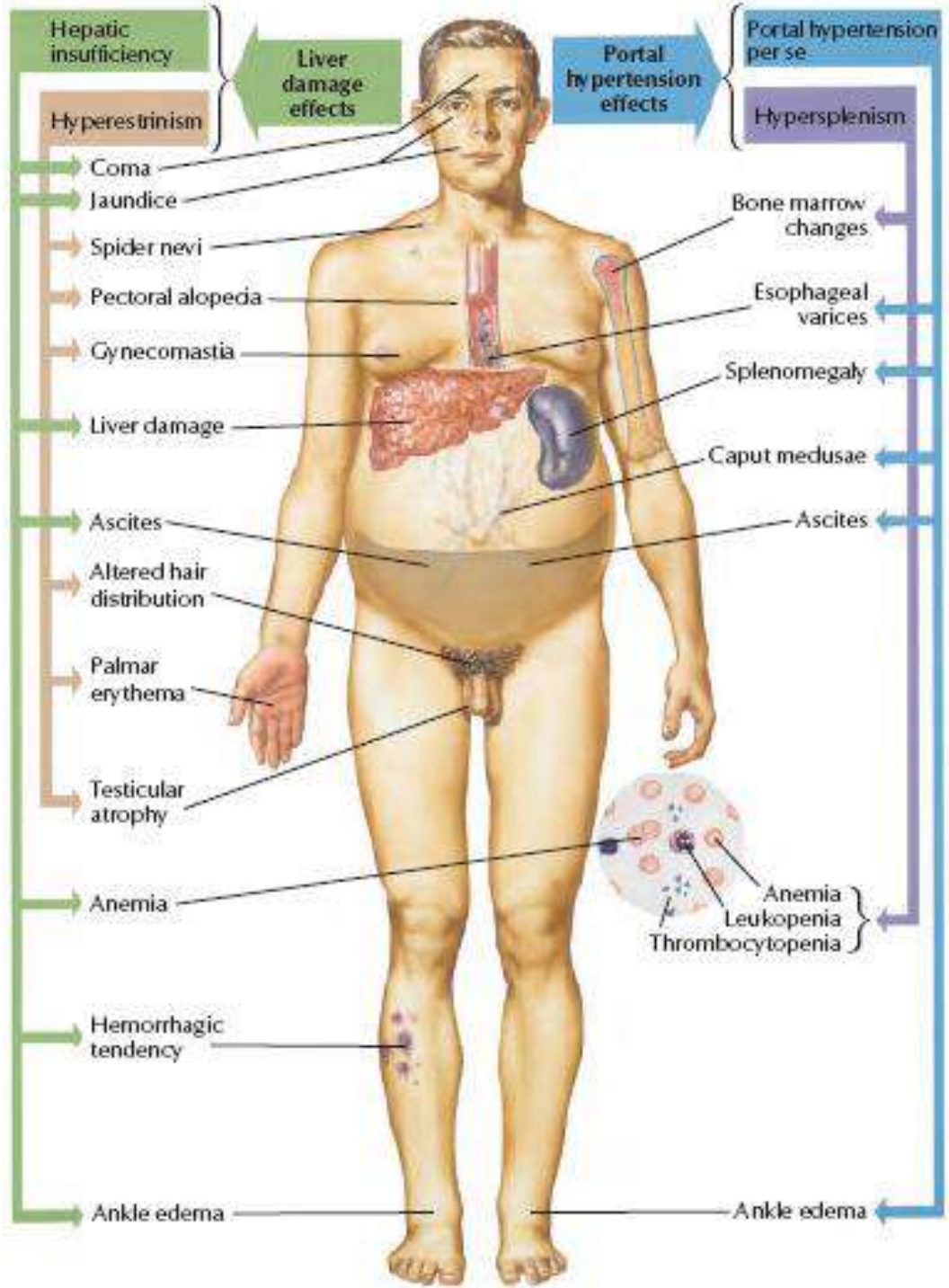
- Micro/ macrovesikular, fibrosis pericentral vein
- Fatty change → **completely reversible if abstinence**

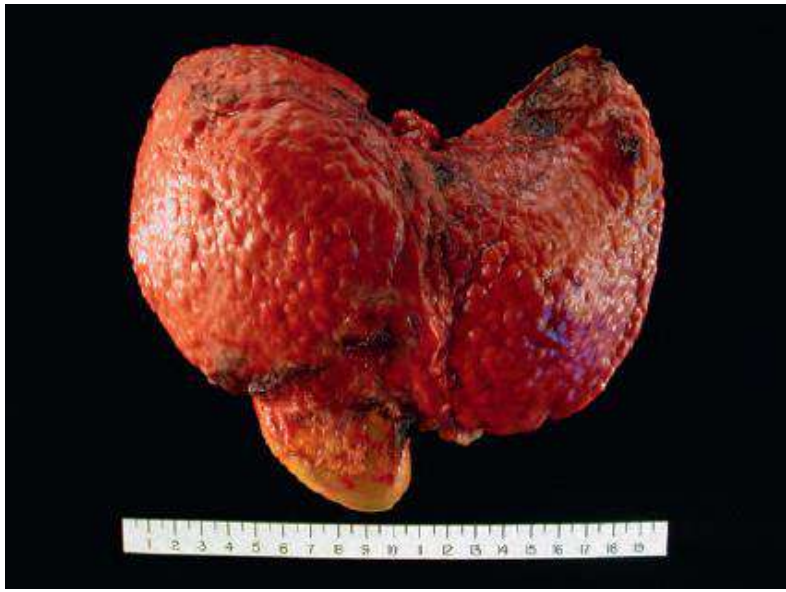
Robbins, Pathologic Basis of Disease 9th Ed, 2015

Chronic Liver Failure and Cirrhosis

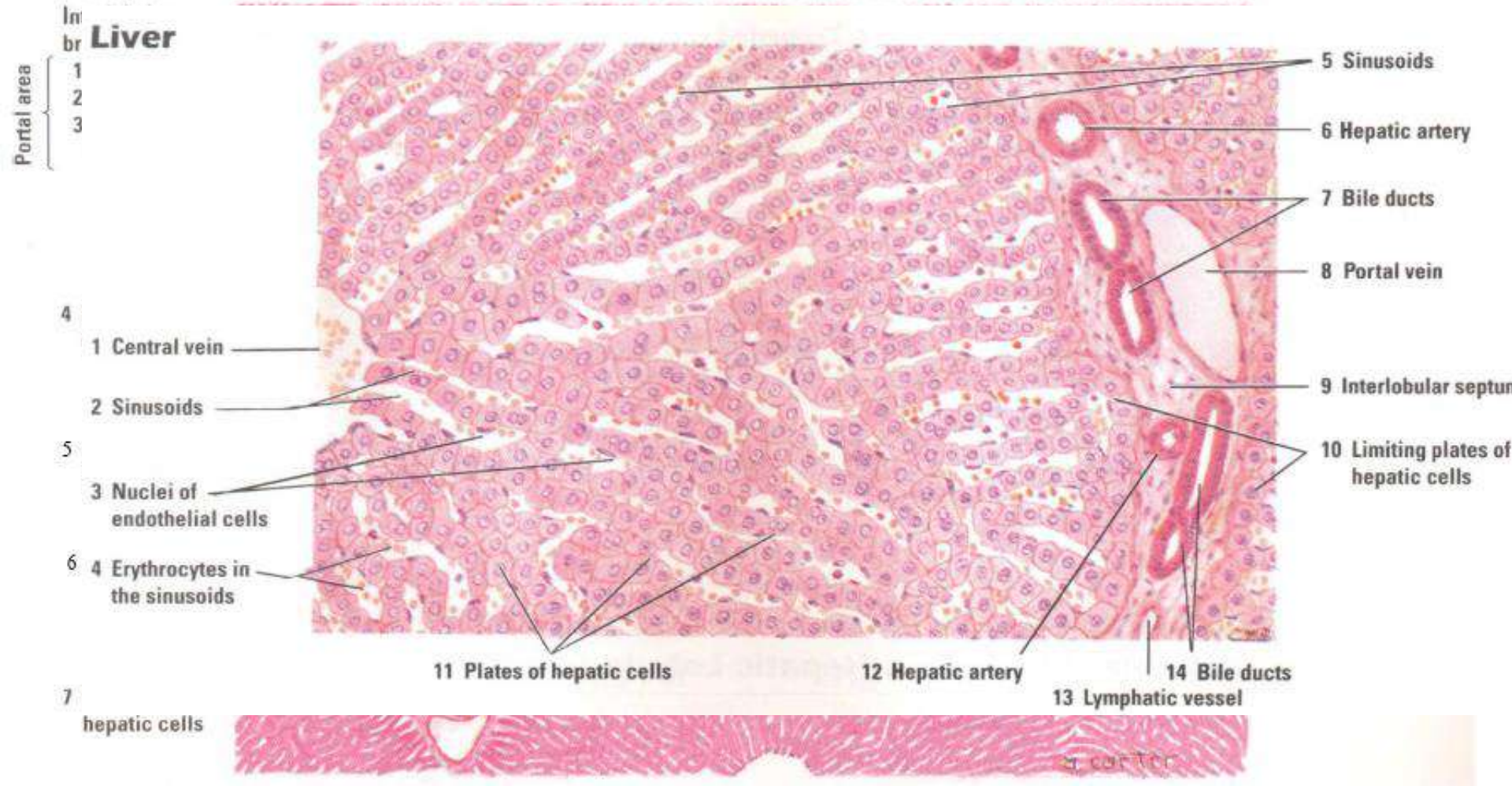
- Chronic Hep B, chronic Hep C, NAFLD, alcoholic liver disease
- Cirrhosis: **diffuse transformation** of the **entire liver** into **regenerative parenchymal nodules** surrounded by **fibrous bands** and vascular
- All cirrhosis → chronic liver failure ???

Stigmata Cirrhosis





Normal Histology



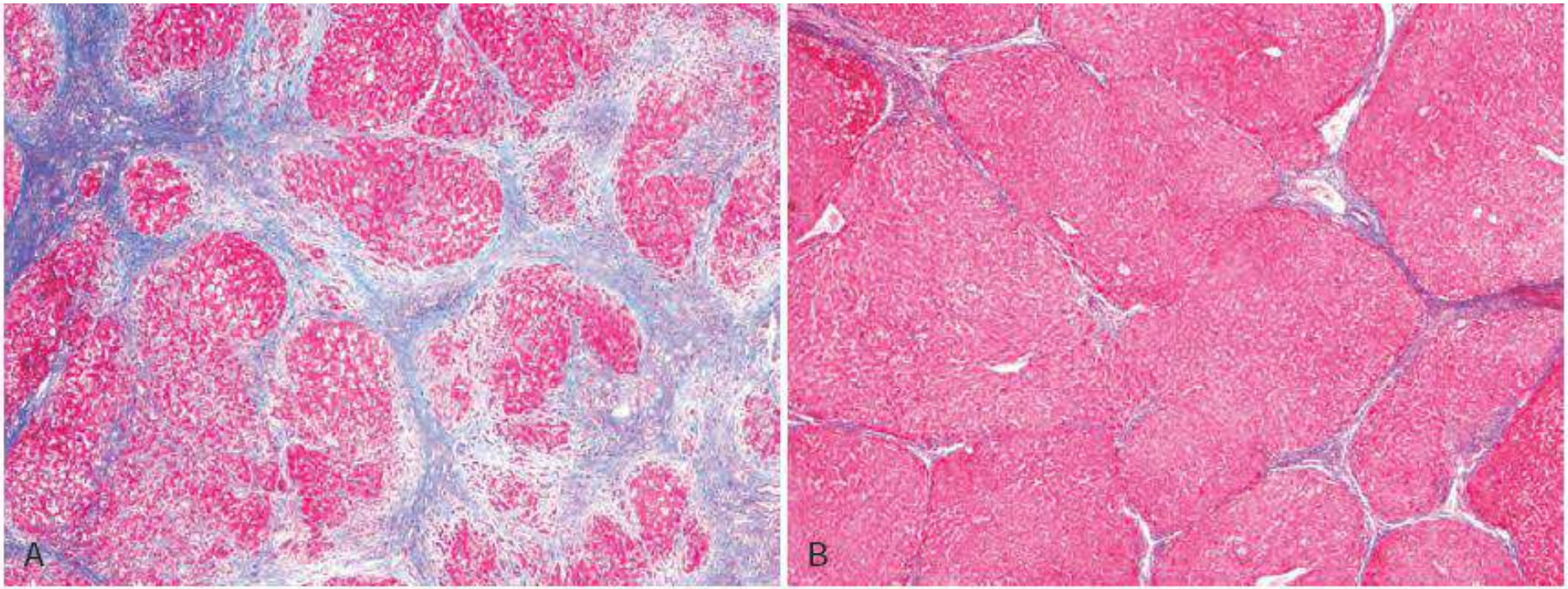


Figure 18-8 Alcoholic cirrhosis in an active drinker (A) and following long-term abstinence (B). **A,** Thick bands of collagen separate rounded cirrhotic nodules. **B,** After a year of abstinence, most scars are gone. (Masson trichrome stain) (Courtesy Drs. Hongfa Zhu and Isabel Fiel, Mount Sinai School of Medicine, New York.)

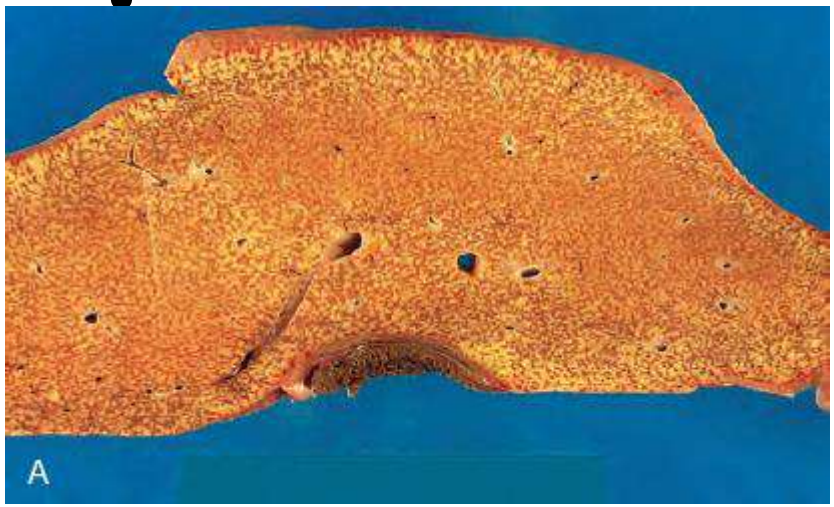
Robbins, Pathologic Basis of Disease 9th Ed, 2015

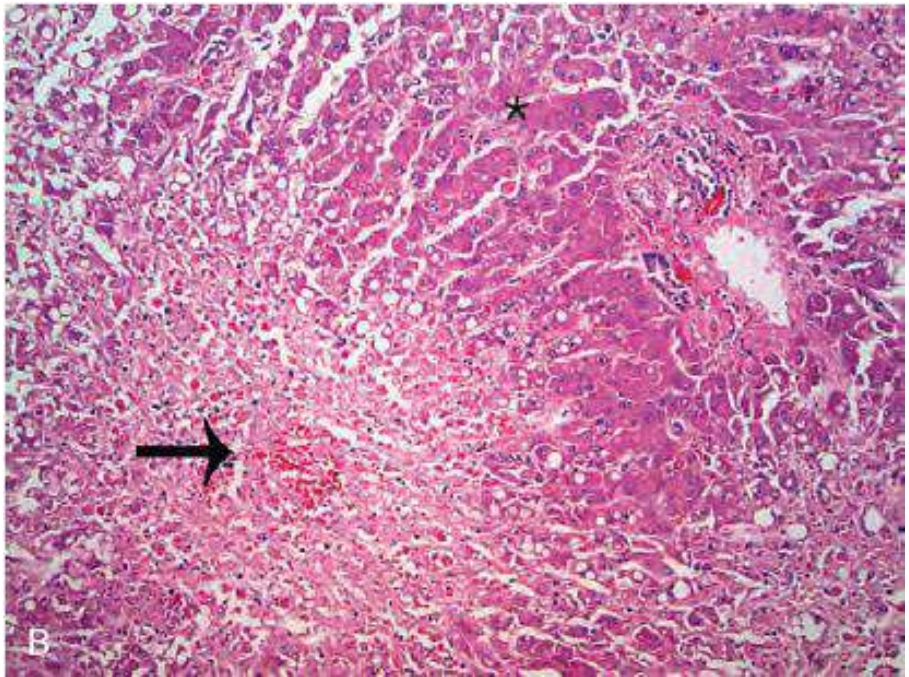
Liver Failure

- Most severe – sudden n massive hepatic destruction
- Acute liver failure – chronic liver failure – acute on chronic liver failure
- 80-90% loss of functional hepatic capacity
- Transplantation – best hope for survival
- Mortality 80%

Liver Failure

- Massive hepatic necrosis
- Acetaminophen (50% in US), autoimun hepatitis, acute Hep A n Hep B infection
- Encephalopathy, coagulopathy
-





Benign Tumors

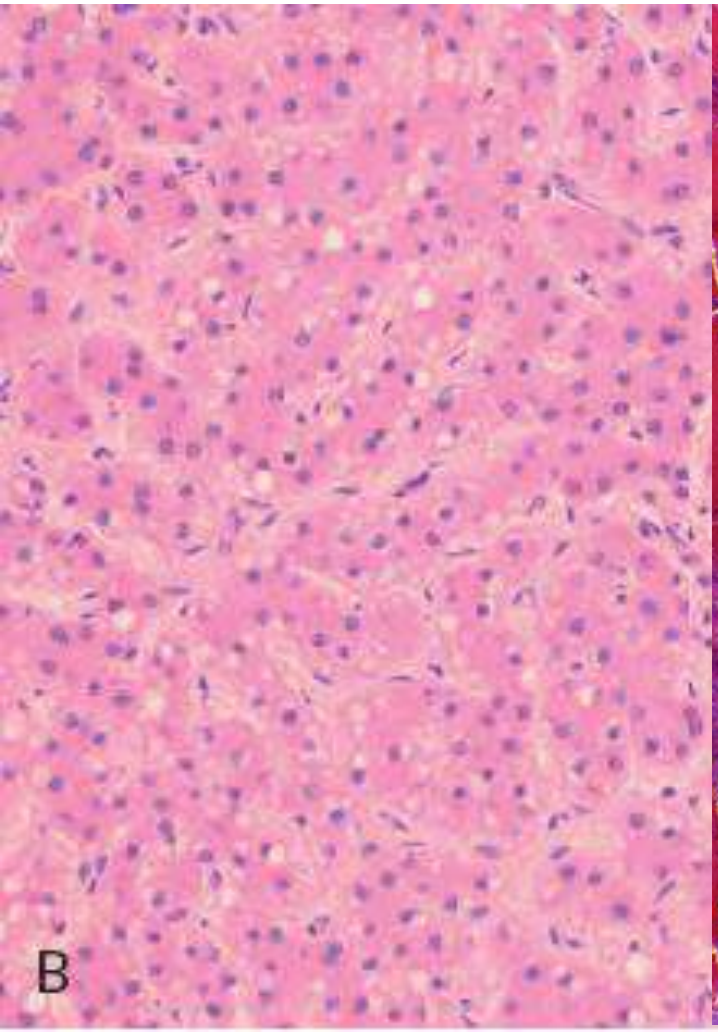
Hepatocellular adenoma

- 85% young women
- Exposure to estrogenic/ androgenic steroid
- Oral contraceptive

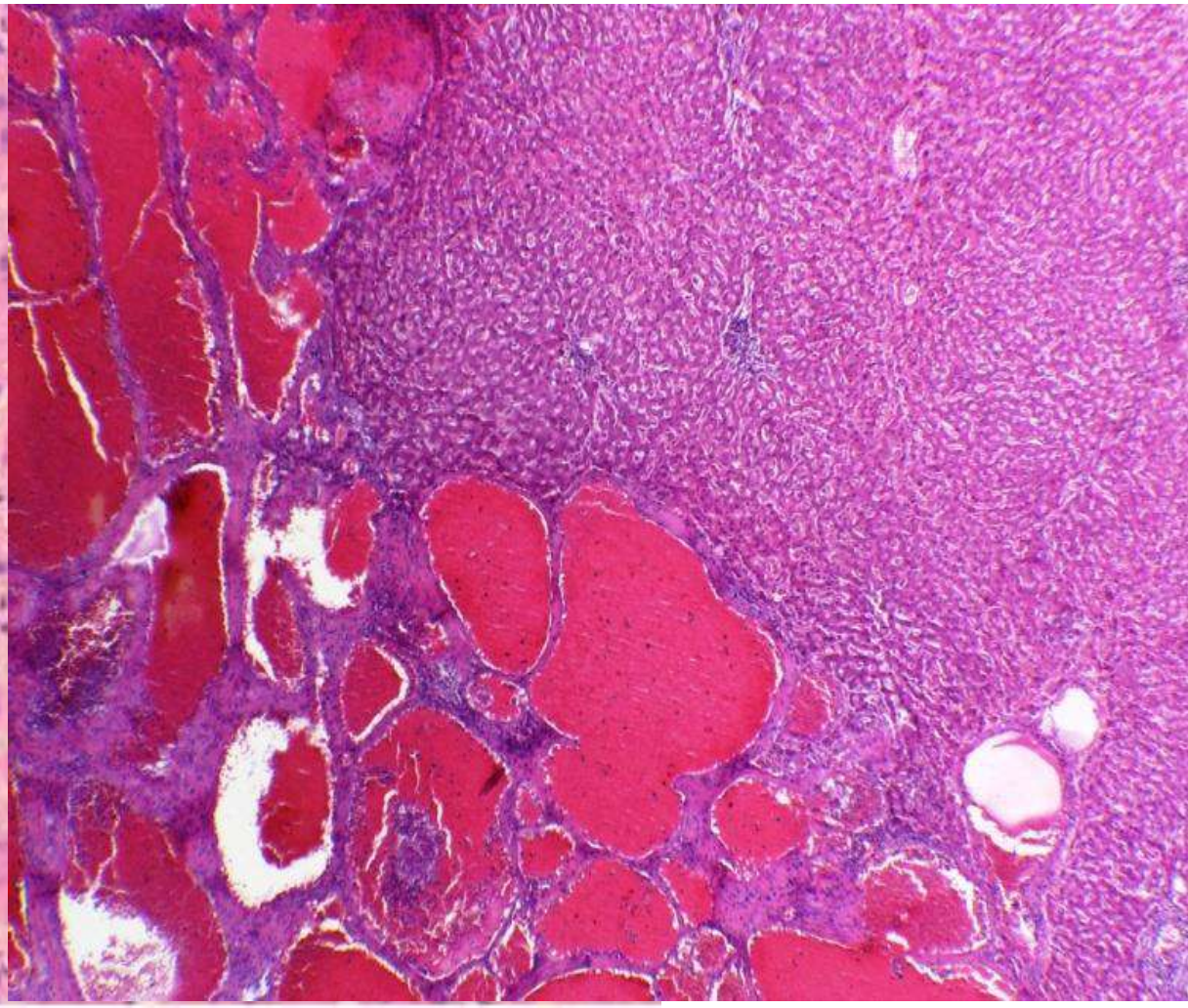
Cavernous hemangioma

- Most common benign tumor

Benign tumor



Hepatocellular Adenoma

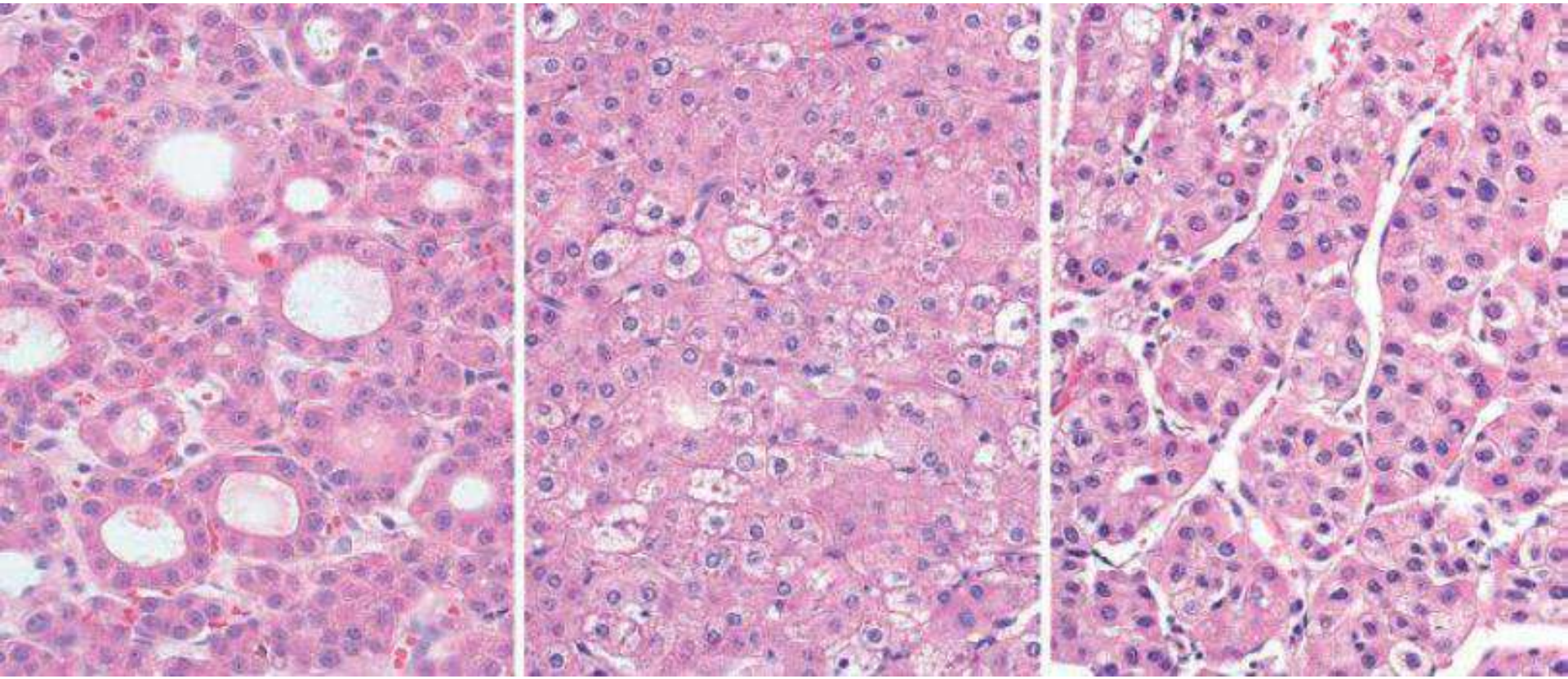


Hemangioma Kavernosum

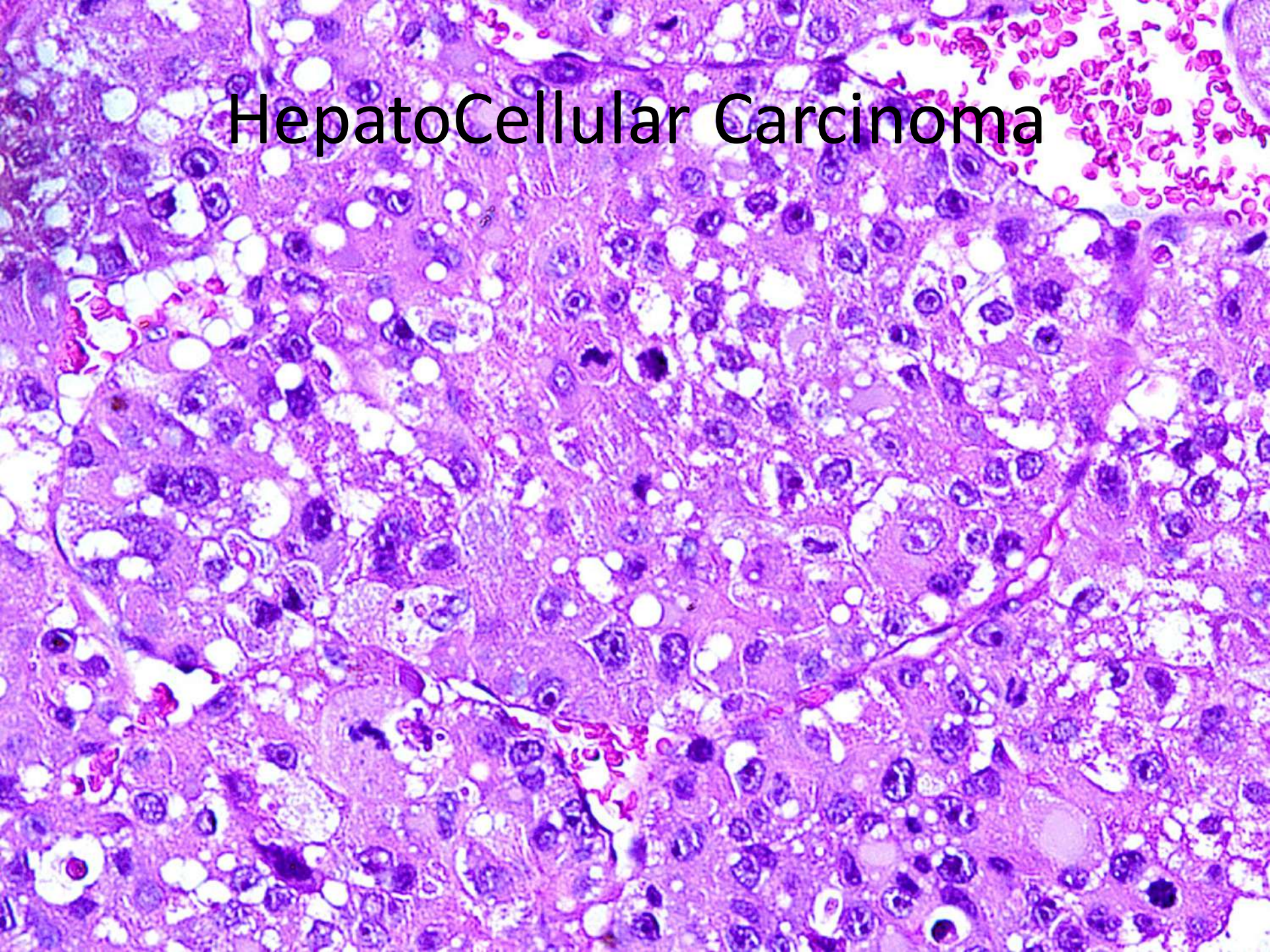
Liver Cancer

- 70-90% hepatocellular carcinoma (HCC), CholangioCa, metastatic from colon Ca, lung Ca, breast Ca
- Men >>> women (3:1 – 8:1)
- 85% in countries with chronic HBV infection
- Highest incidence: Asian countries
- Chronic liver disease: viral infections (HBV, HCV) and toxic injuries (aflatoxin, alcohol)

HepatoCellular Carcinoma

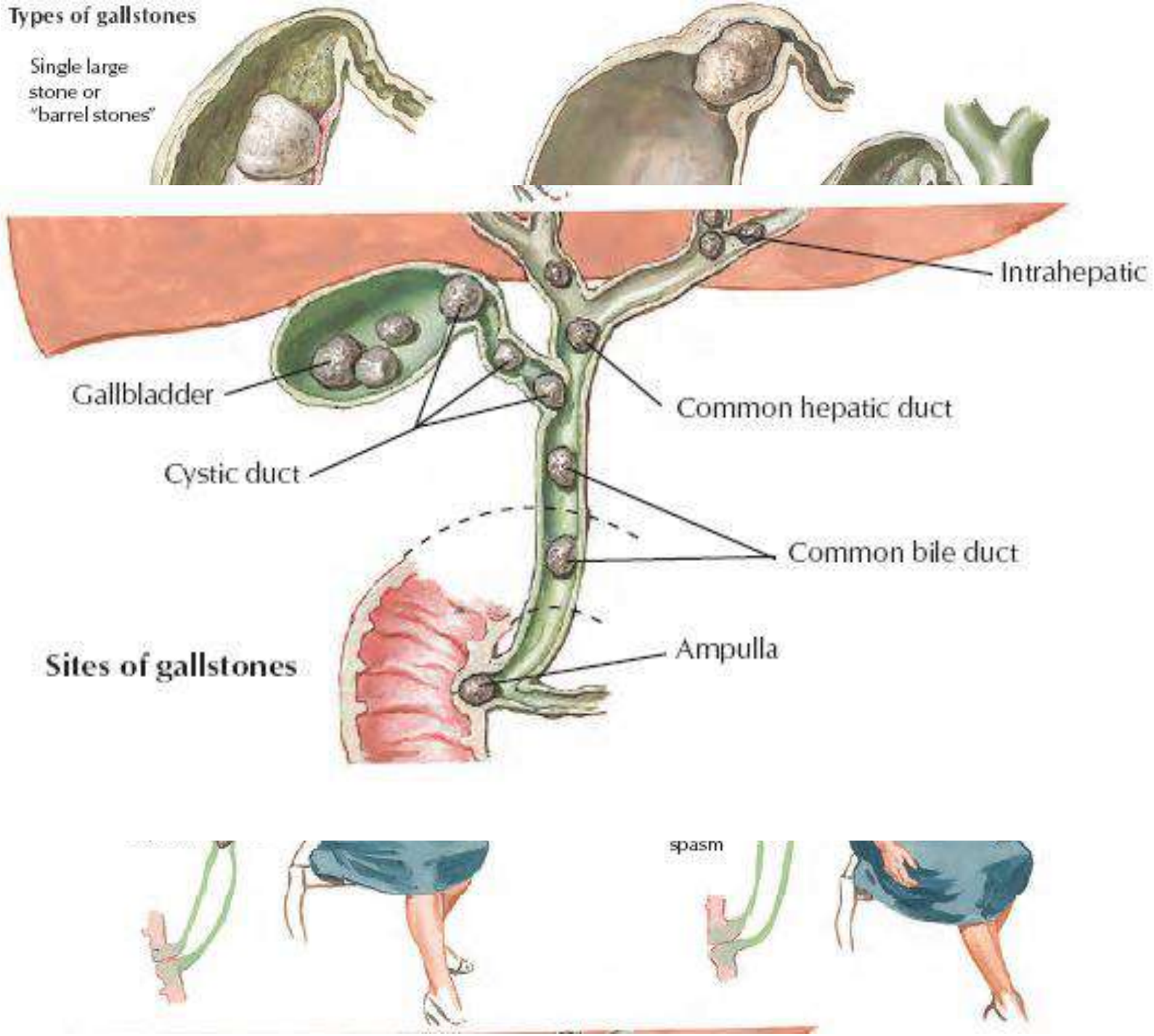


Pseudoasinar – Solid – Trabekular


HepatoCellular Carcinoma

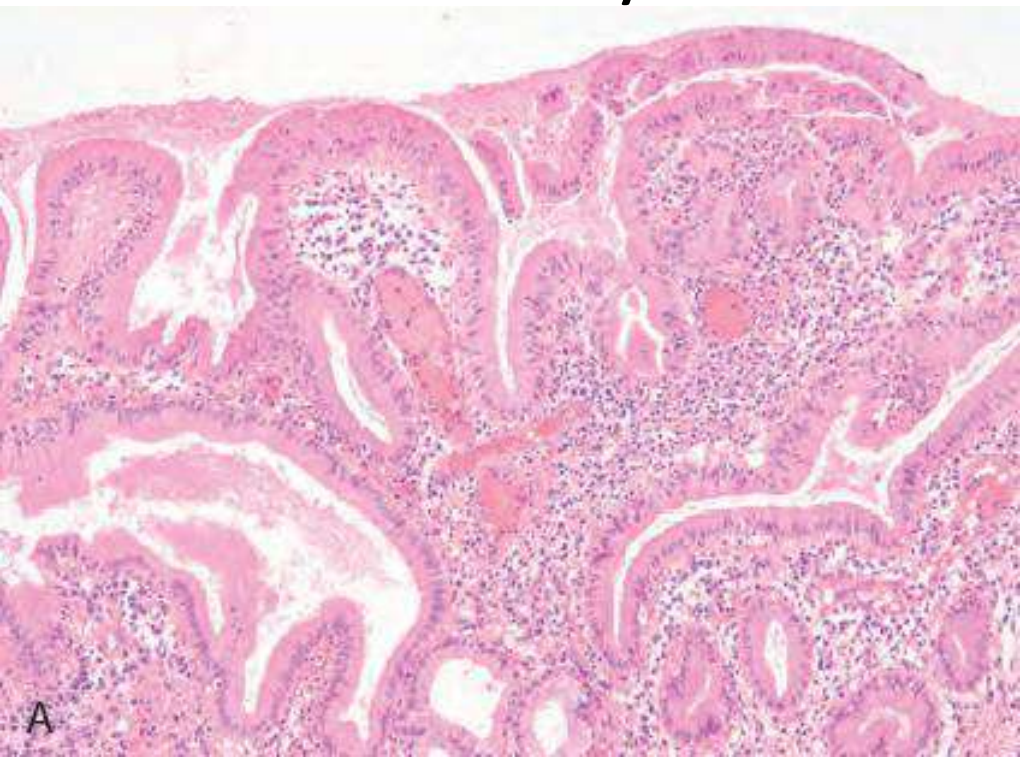
Gallbladder

- 1 Ltr bile excreted by liver per day
- Between meals, bile stored and concentrated
- Organ is not essential – indigestion/ malabsorption fat ≠ after cholecystectomy
- 95% → cholelithiasis (gallstones)
- 10-20% adult populations – developed countries
- 2 types: cholesterol stones, pigmen stones
- 4F – **F**emale, **F**orty, **F**atty, **F**ertile


Types of gallstones
Single large stone or "barrel stones"
Intrahepatic
Gallbladder
Common hepatic duct
Cystic duct
Common bile duct
Ampulla
Sites of gallstones
spasm

Cholecystitis

- Inflammation of gallbladder.
- Acute, chronic.
- Almost always association with gallstones.



Robbins, Pathologic Basis of Disease 9th Ed, 2015

Carcinoma of Gallbladder

- Risk factor: gender, ethnicity, gallstones.
- Adenocarcinoma – most common.
- Late diagnosis – unresectable – poor prognosis.

Pancreas

- Complex lobulated organ – exocrine n endocrine component.
- Exocrine: 80-85% - acinar cell – secreted enzymes for digestion (enzymes, proenzymes).
- Endocrine: islet of Langerhans.

Pancreatitis

- Acute – chronic
- Injurious – autodigestion by its own enzymes.
- Normal → protected by:
  - Inactive proenzymes
  - Activated by trypsin in small bowel
  - Acinar n ductal cells secrete trypsin inhibitor
- Protective mechanism **XXX** → pancreatitis

Table 19-1 Etiologic Factors in Acute Pancreatitis

Metabolic
Alcoholism
Hyperlipoproteinemia
Hypercalcemia
Drugs (e.g., azathioprine)
Genetic
Mutations in genes encoding trypsin, trypsin regulators, or proteins that regulate calcium metabolism
Mechanical
Gallstones
Trauma
Iatrogenic injury
Operative injury
Endoscopic procedures with dye injection
Vascular
Shock
Atheroembolism
Vasculitis
Infectious
Mumps

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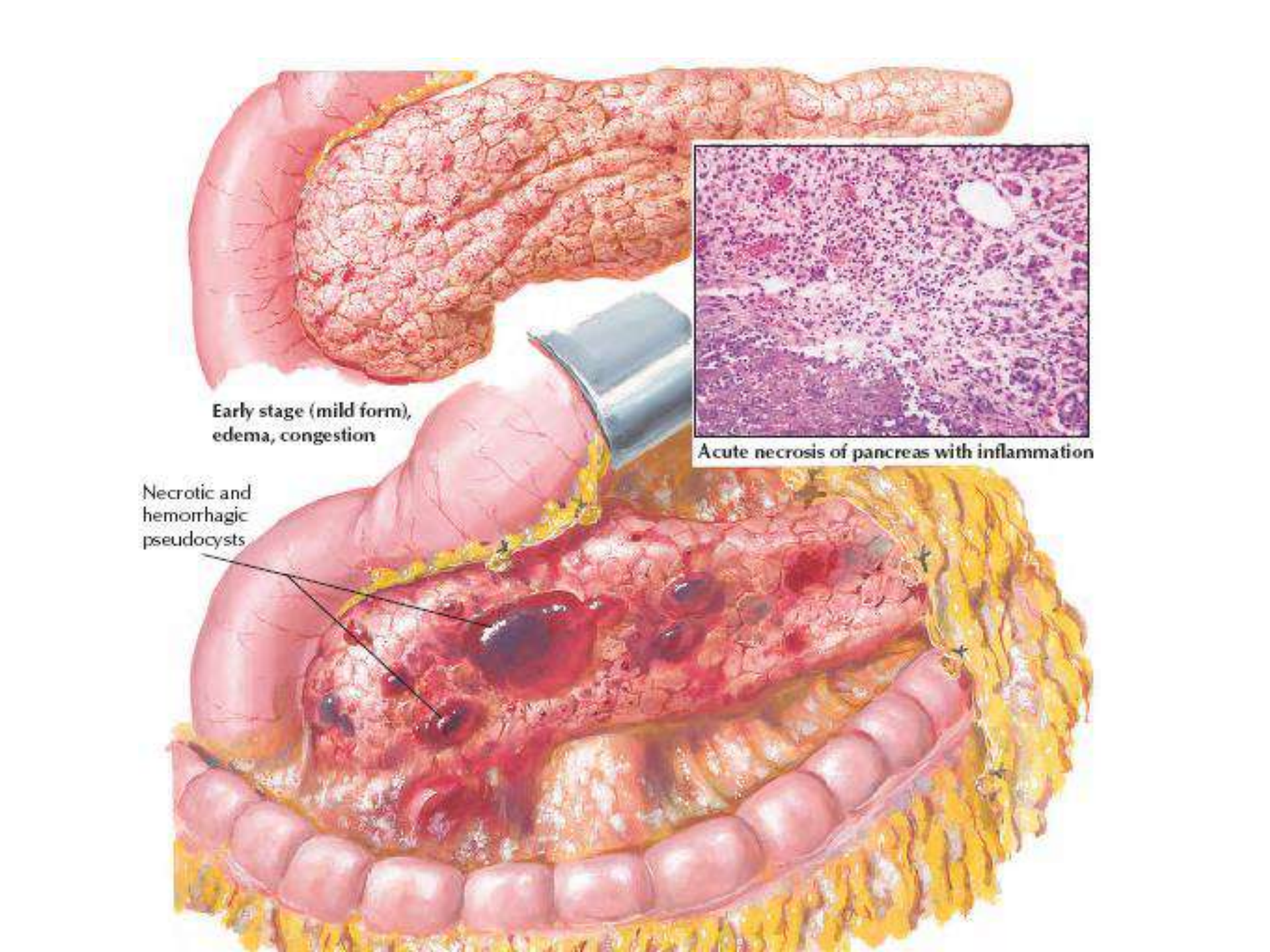
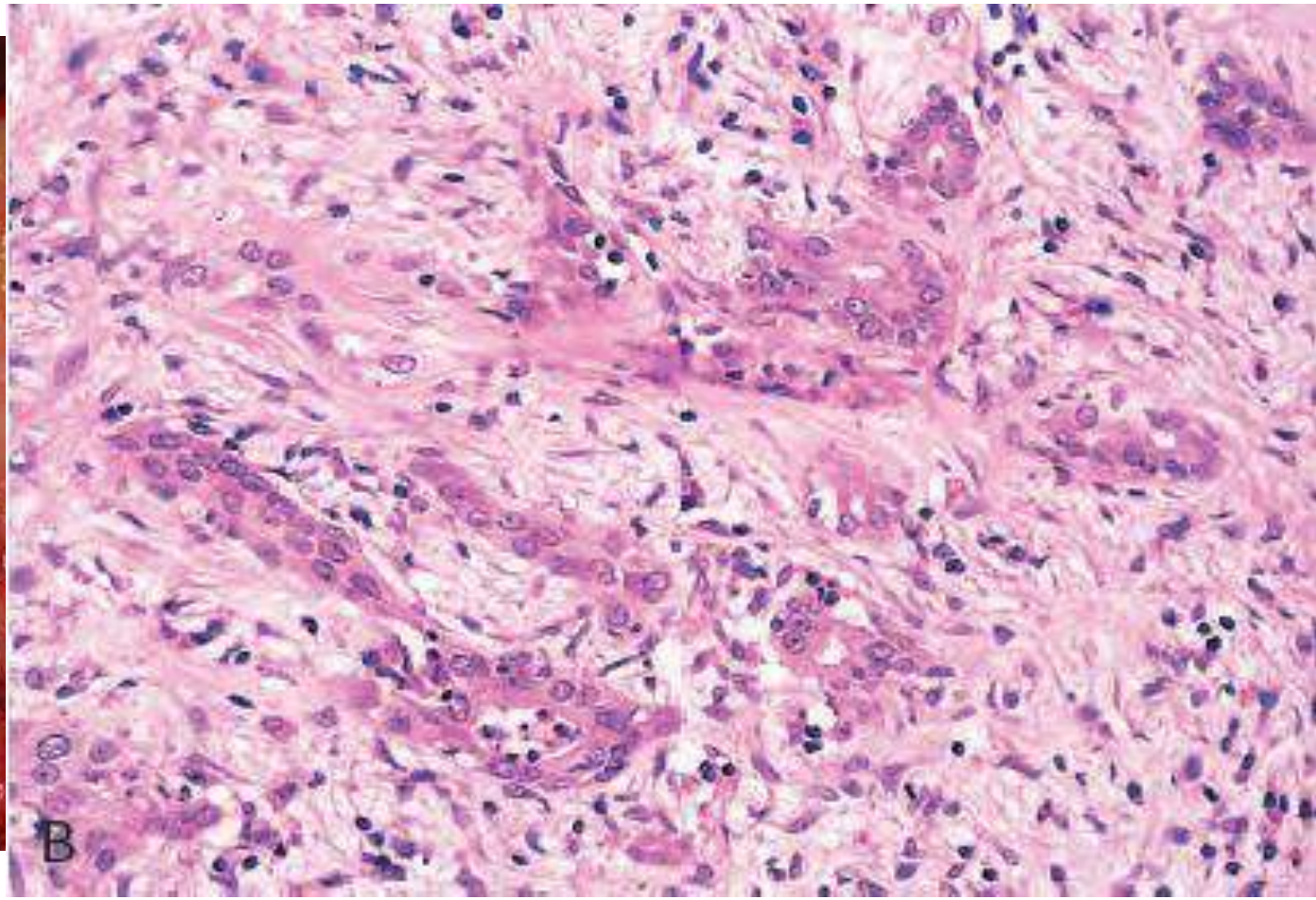

Early stage (mild form), edema, congestion
Acute necrosis of pancreas with inflammation
Necrotic and hemorrhagic pseudocysts

Neoplasm

- Cystic neoplasm: serous, mucinous → benign
- Pancreatic carcinoma
  - Highest mortality rates.
  - 5 year survival rate: <5%
  - Precursor lesion – PanIn (pancreatic intraepithelial neoplasia)

Carcinoma of The Pancreas



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