## I. ESOPHAGUS

### A. *Dysphagia*

1. Difficulty swallowing
    a. Oropharyngeal dysphagia
        "Transfer dysphagia". Difficulty *transferring* the food bolus into the upper esophagus.
    b. Esophageal dysphagia
        Difficulty moving the food bolus from the upper esophagus to the stomach.
2. Most common etiologies
    a. Oropharyngeal (Pre-esophageal)
        (1) Local structural lesions
            (a) Carcinoma
            (b) Zenker's diverticulum
            (c) **Plummer-Vinson syndrome**
                In females, iron deficiency anemia + esophageal webbing
        (2) **Neuromuscular diseases ≈80% of cases**
            (a) MS
            (b) Myasthenia gravis
            (c) Parkinson's disease
            (d) Stroke
        (3) Upper esophageal sphincter dysfunction
    b. Esophageal
        (1) Strictures
            (a) Peptic
            (b) Chemical
        (2) Carcinoma
        (3) Barrett's esophagus
            (a) Metaplastic columnar epithelium replaces normal squamous epithelium.
            (b) ↑ risk of malignant transformation
        (4) Schatski ring
        (5) Achalasia

### B. *Gastroesophageal Reflux Disease*

1. DEFINITION
    Reflux of gastric contents into the esophagus usually causing esophageal inflammation
2. DIAGNOSIS
    a. History and Physical
        (1) Odynophagia (Painful swallowing)
        (2) Pyrosis (Heartburn)

(3) Regurgitation
(4) Chest pain
(5) Dysphagia

b. Laboratory
(1) Barium swallow
(a) Although it can identify reflux, it is unreliable
(b) It can identify strictures
(2) Endoscopy
Direct observation of mucosa ± biopsies
(3) 24 hour esophageal luminal pH recording

3. TREATMENT
a. Nonpharmacologic
(1) Elevate the head of the bed
(2) Avoid late night meals or snacks
(3) ↓ cigarettes, caffeine and ETOH
(4) ↓ fats
(5) Weight loss

b. Pharmacologic
(1) Antacids
(2) $H_2$-receptor antagonists
(3) Metoclopramide
(4) Omeprazole
(5) Cisapride

c. Surgery
(1) Belsey repair
(2) Nissen fundoplication

C. *Esophageal Cancer*
1. DEFINITION
a. A primary malignancy of the esophagus
(1) **Squamous cell carcinoma.....≈95%**
(2) Adenocarcinoma.....≈5%
(a) Gastroesophageal junction
(b) Barrett's esophagus

b. Location
(1) Upper third.....≈10%
(2) Middle third.....≈40%
(3) Lower third.....≈50%

2. DIAGNOSIS
a. History and Physical
(1) **Dysphagia**

(2) Weight loss
(3) ± Pain

b. Laboratory
(1) Barium swallow → identification of esophageal lesion.
(2) Endoscopy + biopsy → diagnosis
(3) CT of the chest → staging

3. TREATMENT
a. Surgery
(1) Less than 10% 5-year survival.
(2) ↑ surgical mortality

b. Radiation
May be of some benefit in lesions of the upper third of the esophagus

c. Chemotherapy
Not useful

4. PROGNOSIS
Poor

## II. STOMACH

### A. *Peptic Ulcer Disease*

1. DEFINITION
a. A defect in the gastrointestinal mucosa which penetrates the muscularis mucosa.
b. Etiologies
(1) Genetic Predisposition
(2) ↑ acid secretion
(3) Direct injury to the mucosa
(a) Aspirin
(b) NSAID
(c) *Helicobacter pylori*
- Present in 90% of duodenal ulcers and 75% in gastric ulcers.
- Treatment ↓ recurrence of PUD by 75-90%

(4) ↓ bicarbonate
(a) Smoking
(b) Acidosis
(5) Stress? caffeine?
(6) Systemic disease
(a) Renal failure
(b) Chronic lung disease

c. **Duodenal ulcers are most common.**
d. Gastric ulcers are less common.

2. DIAGNOSIS
    a. History and Physical
        (1) Dyspepsia
            (a) Nausea
            (b) Vomiting
            (c) Anorexia
            (d) Epigastric fullness
            (e) Bloating
            (f) Pain
                ▸ Nocturnal
                ▸ Burning epigastric
            (g) Heartburn
            (h) Early satiety
            (i) Weight loss
    b. Laboratory
        (1) Upper GI
        (2) Endoscopy + biopsies of ulcer margins ± brush cytologies.
3. TREATMENT
    a. Nonpharmacologic
        (1) ↓ cigarettes
        (2) ↓ NSAID and aspirin
        (3) ↓ emotional stress
    b. Pharmacologic
        (1) Antacids
        (2) $H_2$-receptor antagonists
        (3) Sucralfate
        (4) Lansoprazole
        (5) Anxiolytics/antidepressants
        (6) Eradication of *Helicobacter pylori*
            (a) Tetracycline 500 mg or metronidazole 250 mg tid + bismuth tablets
            (b) Omeprazole + amoxicillin or clarithromycin
            (c) Ranitidine + metronidazole and amoxicillin
        (7) **Maintenance therapy to prevent recurrences.**
    c. Surgery
        (1) Subtotal gastrectomy
        (2) Truncal vagotomy and pyloroplasty
        (3) Truncal vagotomy and antrectomy
        (4) Proximal gastric vagotomy
4. COMPLICATIONS
    a. Hemorrhage

b. Perforation
c. Obstruction

B. *Cancer of the Stomach*

1. DEFINITION
   a. A primary malignancy of the stomach
      **(1) Adenocarcinomas**.....≈90%
      (2) Lymphomas, leiomyosarcomas.....≈10%
   b. Location
      (1) **Pylorus and antrum.....≈50%**
      (2) Lesser curvature.....≈20%
      (3) Body.....≈20%
      (4) Cardia.....≈ 7%
      (5) Greater curvature.....≈ 3%
2. DIAGNOSIS
   a. Early gastric carcinomas are frequently silent.
   b. History (as the disease progresses)
      (1) Anorexia
      (2) Pain
      (3) Weight loss
      (4) Vomiting
   c. Physical
      (1) There are no physical findings in early disease.
      (2) Physical findings in advanced disease are all related to metastases.
   d. Laboratory
      (1) Upper GI
         Identification of a stomach lesion which may or may not be suggestive of a malignancy.
      (2) Endoscopy + biopsy → diagnosis
3. TREATMENT
   a. < 33% of tumors are resectable for cure at presentation
   b. Surgery
      (1) Curative resection
      (2) Palliative resection
   c. Radiation therapy
      Gastric carcinoma is relatively radioresistant
   d. Chemotherapy
      Multiple agents have extended survival time.
4. PROGNOSIS
   a. Five-year survival
      For most patients is <5%.

III. **SMALL AND LARGE BOWEL**

A. Inflammatory Bowel Disease

1. *Ulcerative Colitis*

a. DEFINITION

(1) An inflammatory bowel disease characterized by ***superficial inflammation*** of the colonic mucosa.

(2) The inflammation begins at the rectum and moves proximally.

(3) The inflammation is diffuse and continuous

b. DIAGNOSIS

(1) History and Physical

(a) Rectal bleeding

(b) **Bloody diarrhea**

(c) Tenesmus

(d) Abdominal cramping

(e) Weight loss

(f) Extraintestinal disease can occur affecting the skin, eyes, joints, and liver

(2) Laboratory

(a) Barium enema

- Tubular *ahaustral* segment of colon ± edema ± ulcerations

(b) **Colonoscopy and biopsy**

- Rectal involvement
- Mucosal erythema, ulcerations, hemorrhage and exudate.
- The mucosa is friable
- Crypt abscesses

c. TREATMENT

(1) Diet

(2) Antidiarrheal and anticholinergics in mild/moderate disease only

(3) Oral anti-inflammatories

(a) Sulfasalazine

(b) 5-ASA preparations

- Olsalazine
- Mesalamine

(4) Topical anti-inflammatories

(a) Mesalamine enemas

(b) Steroid enemas

(5) Corticosteroids

Prednisone

(6) Immunosuppressive agents

(a) Azothioprine

(b) 6-mercaptopurine

(7) Surgery

Colectomy for intractable disease

d. COMPLICATIONS

(1) Perforation

(2) Toxic Megacolon

(3) Stricture

(4) ↑ **risk of colon cancer.**

2. *Crohn's Disease*

a. DEFINITION

(1) An inflammatory bowel disease characterized by *transmural inflammation.*

(2) The inflammation can occur from the mouth to the anus

Ileum and right colon are most common

(3) The inflammation is focal and asymmetric

(4) Noncaseating granulomas are present

(5) Extraintestinal disease can occur affecting the skin, eyes, joints, and liver

b. DIAGNOSIS

(1) History and Physical findings are dependent upon location and severity of inflammation.

(a) General

- Weight loss
- Fever and/or night sweats
- Fatigue

(b) Stomach

- Epigastric pain
- Nausea and vomiting

(c) Intestinal

- Abdominal pain
- Cramping

(d) Colon

- Abdominal pain
- Rectal bleeding
- Nonbloody diarrhea

(2) Laboratory

(a) Endoscopic findings

- Cobble stoning
- Linear ulcers
- Discontinuous involvement

(b) Barium enema

- Discontinuous involvement

- Classic "string sign"

c. TREATMENT
(1) Diet
(2) Antidiarrheal and anticholinergics in mild/moderate disease only
(3) Oral anti-inflammatories
(a) Sulfasalazine
(b) 5-ASA preparations
- Olsalazine
- Mesalamine

(4) Topical anti-inflammatories
(a) Mesalamine enemas
(b) Steroid enemas
(5) Metronidazole
(6) Corticosteroids
Prednisone
(7) Immunosuppressive agents
(a) Azothioprine
(b) 6-mercaptopurine
(8) Antibiotics (to treat microabscesses)
(a) Tetracycline
(b) Amoxicillin
(c) Ciprofloxacin
(d) Trimethoprim-sulfamethoxazole
(9) Surgery for intractable disease

d. COMPLICATIONS
(1) Fistulas
(2) Intestinal obstruction
(3) Strictures
(4) Abscess
(5) ↑ **risk of colon cancer.**

B. DIVERTICULAR DISEASE
1. *Diverticulosis*
a. DEFINITION
(1) The presence of diverticula in the colon
(a) Not true diverticula but herniations of mucosa and submucosa through the muscularis.
(b) Most are in the sigmoid colon
(2) The etiology is unknown

b. DIAGNOSIS
(1) History and Physical
(a) Usually asymptomatic
(b) LLQ pain
(c) Changes in bowel habit
(d) ± rectal bleeding
(2) Laboratory
(a) Barium enema
Identifies diverticula
c. TREATMENT
(1) ↑ fiber
(2) Antispasmodics

2. *Diverticulitis*
a. DEFINITION
(1) Microperforation of a diverticula secondary to inspissated feces
(2) The most common complication of diverticulosis.
(3) 90% of cases involve the sigmoid colon.
b. DIAGNOSIS
(1) History and Physical
(a) Fever
(b) LLQ pain
- ± guarding
- ± rebound

(c) Nausea and vomiting
(d) Change in bowel habit
(e) ↓ bowel sounds
(2) Laboratory
(a) ↑ WBC count
(b) Barium enema
(c) CT of the abdomen if there is a question of diagnosis
c. TREATMENT
(1) Mild/moderate disease
(a) NPO → Clear fluids → Soft diet → High fiber diet
(b) Pain medication
No opiates
(c) Antibiotics
- Amoxicillin
- Amoxicillin + metronidazole
- Tetracycline

(2) Severe disease
(a) Hospitalization
(b) IV fluids
(c) IV antibiotics
- Gentamycin + clindamycin
- Cefoxin
- Imipenem

(d) Surgery

d. COMPLICATIONS
(1) Hemorrhage
(2) Perforation
(3) Pericolic abscess
(4) Peritonitis
(5) Fistula
(6) Stricture

D. *Colonic Polyps*

1 General

a. Three types of polyps can arise in the colon
(1) Hyperplastic
(a) Small polyps usually <5 mm.
(b) Little to no malignant potential
(2) Inflammatory
(3) Adenomatous (Neoplastic)
(a) Tubular.....≈60%
(b) Tubulovillous.....≈20-30%
(c) Villous.....≈10%

b. The majority of polyps are *hyperplastic*

c. The majority of polyps are <1 cm.

d. Risk of malignant transformation
(1) ↑ with size
(a) <1 cm.....≈1%
(b) 1-2 cm.....≈10%
(c) >2 cm.....≈30-50%
(2) Polyp type
(a) Tubular.....-5%
(b) Tubulovillous.....≈5-20%
(c) Villous adenoma.....≈30-70%
(3) ↑ numbers of polyps ↑ the risk

e. Treatment
Removal of the polyps upon or shortly after diagnosis.

E. *Colonic Cancer*

1. DEFINITION
    a. A primary malignancy arising from the colonic mucosa.
    b. **98% are adenocarcinoma above the anal verge.**
    c. 80% are below the middle portion of the descending colon
2. DIAGNOSIS
    a. History and Physical
        (1) Left colon
            (a) **Rectal bleeding**
            (b) **Change in bowel habit**
            (c) Tenesmus
            (d) Pencil stools
            (e) Vague abdominal or back pain
        (2) Right colon
            (a) Guaiac + stools
            (b) RLQ pain
            (c) Weight loss
        (3) ≈33% of patients will present with metastasis
    b. Laboratory
        (1) CBC
            Iron deficiency anemia
        (2) + occult blood
        (3) ↑ CEA (not specific)
        (4) Liver scan
            If ↑ liver function studies or hepatomegaly
        (5) Bone scan
            If ↑ of alkaline phosphatase or bone pain
        (6) Barium Enema
            Can detect cancer >90% of cases
        (7) Colonoscopy with biopsy
            Has almost 100% rate of establishing diagnosis
        (8) CT scan
            Preoperatively to exclude synchronous lesions and metastatic disease
3. TREATMENT
    a. Surgery
        (1) Dependent upon location of tumor
            (a) Right- or left-sided lesions
                Hemicolectomy
            (b) Upper rectum, sigmoid
                Anterior resection with anastomosis to the rectal stump

(c) Distal lesions of the rectum
Abdominal-Perineal resection with permanent colostomy
(2) Palliative surgery for metastatic disease to control obstruction, bleeding or perforation
b. Radiation
(1) Pre- or postoperative radiation of rectal lesions to prevent local recurrence.
(2) Treatment of advanced disease to alleviate obstruction and bony pain
c. Chemotherapy
↑ survival for Duke's C with combination 5-fluorouracil + levamisole
4. PROGNOSIS
a. Duke's A.....>80%
b. Duke's B.....≈60-80%
c. Duke's C.....≈50%
d. Duke's D.....<25%
e. The ↑ in the penetration of the wall of the colon, the ↓ the prognosis.

## IV. GALLBLADDER

### A. *Acute Cholecystitis*

1. DEFINITION
a. An acute inflammation of the gallbladder
(1) Etiologies
(a) **95% calculous**
(b) 5% acalculous
2. DIAGNOSIS
a. History and Physical
(1) RUQ or epigastric pain
(2) Nausea and vomiting
(3) Anorexia
(4) Low-grade fever
(5) Murphy's sign
"Classic" physical finding in which there is inspiratory arrest during deep palpation of the right upper quadrant
b. Laboratory
(1) ↑ WBC count
(2) ± abnormal liver function studies
(3) **Abdominal ultrasound**
(a) Gallstones
(b) ↑ edema/thickness gallbladder wall
(c) Distention

3. TREATMENT
    a. Hospitalization
    b. NPO
    c. IV fluids
    d. Pain medication
    e. Antibiotics
        (1) Ampicillin
        (2) Cephalosporins
    f. Surgery following improvement
4. COMPLICATIONS
    a. Empyema
    b. Perforation
        (1) Pericholecystic abscess
        (2) Peritonitis
    c. Fistula

B. *Chronic Cholecystitis*

1. DEFINITION
    a. An inflammation of the gallbladder secondary to gallstones
    b. Characterized by *recurrent* attacks of RUQ pain secondary to *transient* obstruction of the cystic duct by gallstones.
2. DIAGNOSIS
    a. History and Physical
        (1) Postprandial RUQ or epigastric pain and tenderness
        (2) Nausea and vomiting
        (3) Belching
        (4) Bloating
        (5) Fatty food intolerance
    b. Laboratory
        (1) **Abdominal ultrasound**
            (a) + for gallstones
            (b) ↑ thickness wall and a contracted gallbladder
3. TREATMENT
    Surgery

C. *Cholelithiasis*

1. DEFINITION
    a. The formation of "stones" within the gallbladder
    b. Stone analysis
        (1) Cholesterol/mixed stones.....≈80%
        (2) Pigmented stones.....≈20%

c. Etiologies
(1) The four "F"
(a) Fat
(b) Forty
(c) Female
(d) Fertile
(2) Oral contraceptive use
(3) Pregnancy
(4) Diabetes
(5) Hyperlipidemia, type IV

2. DIAGNOSIS
a. History and Physical
(1) May be asymptomatic
(2) RUQ and/or epigastric pain following meals
(3) Nausea and vomiting
b. Laboratory
(1) **Abdominal ultrasound**
+ for gallstones

3. TREATMENT
Surgery

V. **LIVER**

A. Viral Hepatitis

1. *Hepatitis A*

a. DEFINITION
(1) An infection of the liver caused by a 27 nm RNA virus that attacks the hepatocytes in the liver.
(2) Transmission
(a) **Fecal-oral**
Water/food-borne contamination
(b) Sexual
(c) Blood
(3) Incubation
(a) 2-6 weeks
(b) Greatest infectivity the two weeks *before* clinical illness.
(4) **No chronic form or carrier state**
(5) Fulminant disease is rare

b. DIAGNOSIS
    (1) History and Physical
        (a) Mild disease
            Subclinical or flu-like symptoms
        (b) Moderate
            - Fatigue
            - Malaise
            - Fever
            - Anorexia
            - Alteration in taste and small
            - Nausea and vomiting
            - RUQ pain or discomfort
            - ± jaundice
            - Dark urine
    (2) Laboratory
        (a) Abnormal liver function studies
            - ↑ GGTP
            - ↑ AST
            - ↑ ALT
            - ± ↑ bilirubin
            - Moderate ↑ of alkaline phosphatase
        (b) Serologies
            - Anti-HAV IgM
                Current or recent infection or convalescence
            - Anti-HAV IgG
                Recovered or vaccination.

c. TREATMENT
    (1) Supportive care
    (2) Antiemetics
    (3) Monitor liver function
    (4) Alcohol should be avoided
    (5) **Corticosteroids have no place in the treatment of acute viral hepatitis**

d. PROPHYLAXIS
    (1) Treat contacts with immune serum globulin
    (2) Hepatitis A vaccine for *active immunization* for patients ≥ 2 years of age.

e. PROGNOSIS
    Excellent

2. *Hepatitis B*
    a. DEFINITION
        (1) An infection of the liver caused by a 42 nm DNA virus that attacks the hepatocytes in the liver.
        (2) Transmission
            (a) Parenteral contact
                - Needle stick/transfusion
                - Sexual contact
                - Perinatal transmission
            (b) **Hepatitis B is present in blood for a protracted period of time.**
        (3) Carrier state can occur
            (a) Persistent circulating viral particles
            (b) No histologic evidence of hepatic inflammation
            (c) Normal liver function studies
        (4) Chronic hepatitis can occur
        (5) Fulminant disease <5%
        (6) Incubation
            4 weeks to 6 months
        (7) Hepatitis B antigens
            (a) HBsAg
                - Surface antigen present before the onset of clinical illness and persists through early convalescence.
                - Persistence of circulating HBsAg may indicate progression to chronic hepatitis B.
            (b) HBcAg
                - Nucleocapsid core does not freely circulate
                - Found in hepatocytes when there is *active* viral replication
            (c) HBeAg
                - HBcAg *circulating form*
                - A marker of active viral replication and infectivity
                - Persistence of circulating HbeAg > 3-4 months indicates likely progression to chronic hepatitis B.
    b. DIAGNOSIS
        (1) History and Physical
            (a) Fatigue
            (b) ± fever
            (c) Nausea ± vomiting
            (d) Jaundice
            (e) Dark urine

(f) Immune complex disease
- ▸ Urticaria
- ▸ Arthritis
- ▸ Arthralgias
- ▸ Glomerulonephritis
- ▸ Vasculitis

(g) Fulminant hepatitis
- ▸ Liver failure
- ▸ Coagulopathy
- ▸ Encephalopathy

(2) Laboratory

(a) Abnormal liver function studies
- ▸ ↑ GGTP
- ▸ ↑ AST
- ▸ ↑ ALT
- ▸ ± ↑ bilirubin
- ▸ Moderate ↑ of alkaline phosphatase

(b) Serologies
- ▸ Anti-HBs
  - • Antibody to surface antigen
  - • Appears following hepatitis B infection or vaccination
  - • **Confers immunity and is protective**
- ▸ Anti-HBc
  - • Antibodies to HBV core antigen
    - ◦ Anti-HBc IgM
      - · + during acute hepatitis B infection
      - · ± during chronic hepatitis B infection
    - ◦ Anti-HBcTotal
      - · ↑ in acute infection
        - ↑ anti-HBc IgM
      - · ↑ in chronic infection
        - ↑ anti-HBc IgG
      - · ↑ in patients who have recovered
  - • Usually as infection resolves there is ↓ levels of anti-Hbc IgM and ↑ levels of anti-HBc IgG
- ▸ HbeAg
  - • Correlates to viral replication in the liver
  - • Appears early in acute infection and disappears in a few weeks

- Persistence > 3-4 months usually progression to chronic hepatitis B

(c) Liver biopsy

- ▸ Histologic diagnosis for chronic persistent or chronic active hepatitis
  - • **Chronic persistent hepatitis**
    - ◦ Mononuclear cell infiltrate limited to portal tracts
    - ◦ Usually nonprogressive
    - ◦ Cirrhosis is rare
  - • **Chronic active hepatitis**
    - ◦ Piecemeal necrosis
      - · Mononuclear cell infiltrate that extends past the portal tracts and into the peri-portal space
    - ◦ Progressive
    - ◦ Cirrhosis is more common
    - ◦ ↑ incidence of hepatocellular carcinoma

(3) Chronic hepatitis B

(a) Criteria for diagnosis

- ▸ Clinical evidence of HBV infection ≥ 6 months
- ▸ Abnormal liver function studies
- ▸ Documented histologic findings of ongoing hepatic infection

(b) Likelihood in developing chronic hepatitis B is ≈5-10%.

(c) Serologies

- ▸ + HbsAg
- ▸ ↑ levels of Anti-HBcTotal

(d) The prognosis for chronic hepatitis B is variable

c. TREATMENT

(1) Supportive care

(2) Antiemetics

(3) Monitor liver function

(4) Alcohol should be avoided

(5) **Corticosteroids have no place in the treatment of acute viral hepatitis**

(6) Interferon-alpha for chronic hepatitis B

d. PROPHYLAXIS

(1) Unvaccinated patients exposed to HBV

(a) Hepatitis B immune globulin

(b) HBV vaccine

(2) Previously vaccinated

Test for anti-HBs and if the titer is < 10 ml U/mL. treat as above

4. *Hepatitis C*

a. DEFINITION

(1) An infection of the liver caused by an RNA virus that attacks the hepatocytes in the liver.

(2) Transmission

(a) Parenteral contact

- **The major cause of post-transfusion hepatitis.**
- Needle sticks or injecting drug use

Injecting drug use accounts for ≈50% of the cases

(b) Sexual

(c) Perinatal

(3) Incubation

2 weeks-6 months

(4) Carrier state exists

(5) Chronic hepatitis C

(a) ≈50-60% of acute hepatitis C progresses to chronic hepatitis C

(b) ↑ risk of cirrhosis

(c) ↑ risk of hepatocellular carcinoma

(6) Fulminant disease in <5% of cases

b. DIAGNOSIS

(1) History and Physical

(a) Acute hepatitis C is clinically silent in over 90% of patients

(2) Laboratory

(a) Liver function studies

Fluctuating ↑ levels of GGTP, AST and ALT

(b) Serologies

- Anti-HCV
  - Second-generation enzyme radioimmunoassay
  - + weeks to months after exposure
  - Not protective
- Confirmation of Anti-HCV with RIBA-II

Recombinant immunoblot assay

(c) Liver biopsy in chronic disease

c. TREATMENT

(1) Supportive care

(2) Monitor liver function

(3) Interferon-alpha for chronic hepatitis C

(4) ? Immune serum globulin

5. *Hepatitis D*

a. DEFINITION

(1) An infection of the liver caused by an RNA virus or *delta agent* that attacks the hepatocytes in the liver.

(2) **HBV infection must be present for infection to occur.**

(a) Coinfection

Simultaneous infection with both hepatitis B and hepatitis D

(b) Superinfection

- Chronic hepatitis B infection when exposed to delta agent
- ↑ risk of cirrhosis
- ↑ risk of fulminant liver failure

(3) Transmission

(a) Blood

- In the United States and western Europe
  - Injecting drug use
  - Multiply transfused hemophiliacs

(b) Sexual and perinatal

South America, Middle East, Mediterranean basin

(4) Incubation

3 weeks-3 months

(5) Carrier state exists

(6) **Chronic hepatitis occurs in most patients**

(7) Fulminant disease occurs ≈5-20% of patients

b. DIAGNOSIS

(1) History and physical

See hepatitis B

(2) Laboratory

(a) Serologies

- Coinfection
  - Anti-HDV IgM in ↓ titers or transiently present
  - HDAg
- Superinfection
  - Anti-HDV in ↑ sustained titers
  - HDAg

c. TREATMENT

See hepatitis B

d. PROPHYLAXIS

None

6. *Hepatitis E*
    a. DEFINITION
        (1) An infection of the liver caused by an unclassified RNA virus that attacks the hepatocytes in the liver.
        (2) Transmission
            (a) Fecal-oral
            (b) Travelers to endemic areas are at risk.
            (c) No reported cases of hepatitis E acquired in the United States
        (3) Incubation
            15-60 days
        (4) Carrier state does not exist
        (5) Chronic hepatitis E does not occur
        (6) Fulminant disease 1-2% but 10-30% in pregnant women
    b. DIAGNOSIS
        (1) History and Physical
            Similar to hepatitis A
        (2) Laboratory
            (a) Serologies
                ▸ HEVAg and Anti-HEV assays available from the CDC
    c. TREATMENT
        See hepatitis A
    d. PROPHYLAXIS
        None

C. *Hepatic Carcinoma*
1. DEFINITION
    a. A primary malignancy of the hepatic parenchyma.
        Hepatocellular (Hepatoma) ≈70-90% of cases
    b. A primary malignancy of the hepatic bile ducts
        Cholangiocarcinoma ≈10-30% of cases.
    c. Predisposing conditions
        (1) **Cirrhosis**
        (2) Chronic hepatitis B
        (3) Chronic hepatitis C
2. DIAGNOSIS
    a. History and Physical
        (1) RUQ mass and tenderness
        (2) Abdominal pain
        (3) Weight loss
        (4) Nausea and vomiting
        (5) Ascites

b. Laboratory
(1) Abnormal liver function studies
(2) Alpha-fetoprotein
+ in ≈70% of cases but is not diagnostic
(3) Ultrasound
Excellent technique to screen for liver mass
(4) MRI or CT
Valuable to evaluate tumor and vascular invasion
(5) **Biopsy is the definitive test for diagnosis**

3. TREATMENT
a. Surgery
Only for solitary hepatoma
b. Radiation
Low-dose radiation to alleviate liver pain
c. Chemotherapy
Hepatic artery perfusion
≈50% have a response and median survival↑ to over a year
d. Liver transplantation
Appropriate for selected patients

4. PROGNOSIS
Median survival for all patients is ≈5 months

VI. **PANCREAS**

A. *Acute Pancreatitis*

1. DEFINITION
a. An inflammation of the pancreas. The inflammation caused by pancreatic auto-digestion, can run the gamut from mild disease to life-threatening illness.
b. Acute pancreatitis will usually resolve along with *normal* pancreatic function.
c. Etiologies
(1) **>80% secondary to gallstones or ETOH abuse**
(2) ≈10% are idiopathic
(3) Miscellaneous
(a) Trauma
(b) Hyperparathyroidism
(c) Hyperlipidemia
(d) Infection

2. DIAGNOSIS
a. History and Physical
(1) ↑ pain, "boring" or "stabbing" in the LUQ or midepigastrium.
(2) Nausea ± vomiting

(3) Dehydration
(4) Fever
(5) Tachypnea
(6) Tachycardia
(7) ↕ BP
(8) Abdominal distention
(9) ↓ bowel sounds
(10) ± guarding
(11) In severe disease
(a) Shock
(b) Hemorrhage (rare)
▸ Grey Turner sign
Large flank ecchymosis
▸ Cullen sign
Periumbilical ecchymosis

b. Laboratory
(1) ↑ serum amylase
(2) ↑ serum lipase
(3) Ultrasonography
May detect gallstones, ↑ edema or enlargement of pancreas
(4) CT of abdomen
Confirms the diagnosis

3. TREATMENT
a. Mild to moderate disease
(1) Hospitalization
(2) Correct dehydration
(3) Monitor calcium levels
(4) NPO
(5) Nasogastric tube to alleviate nausea and vomiting
(6) Pain control
(7) $H_2$ receptor antagonists
(8) Antibiotic therapy
(a) For severe necrotizing pancreatitis
(b) Adjunct during surgical intervention

b. Severe
(1) Admit ICU for treatment if any of the following present
(a) Shock
(b) Respiratory failure
(c) Renal failure

(d) Severe metabolic complications
- Hyperglycemia
- Hypercalcemia

4. COMPLICATIONS
   a. Intra-abdominal infections (pancreatic phlegmon)
      (1) Laparotomy with debridement/drainage
      (2) C&S of fluid/debris and appropriate antibiotic therapy.
   b. Hemorrhage (rare)
      Immediate laparotomy
   c. Pancreatic pseudocysts
      (1) Diagnosed by ultrasound cured by surgery
         (a) Complications
            - Rupture
            - Infection
            - Hemorrhage
   d. Pulmonary complications
      Atelectasis → pneumonia → ARDS
   e. Acute renal failure

B. *Chronic Pancreatitis*
   1. DEFINITION
      a. An inflammation of the pancreas caused by
         Recurrent bouts of acute pancreatitis yielding replacement of the exocrine and endocrine pancreas by scar tissue
      b. Chronic pancreatitis will result in the *loss* of normal pancreatic function.
      c. Common Etiologies
         (1) **ETOH abuse**
         (2) Idiopathic (>25%)
         (3) Trauma
         (4) Hypercalcemia
         (5) Cystic fibrosis
   2. DIAGNOSIS
      a. History and Physical
         (1) Abdominal pain
            (a) Usually epigastric with referred pain to the back 50% of the time
            (b) ↑ pain when eating
         (2) Weight loss
         (3) Steatorrhea
      b. Laboratory
         (1) ± mild ↑ amylase or lipase
         (2) ± mild ↑ liver function studies

(3) Abnormal bentiromide (Chymex) test
(4) Abnormal secretin stimulation test
(5) ↑ stool fats (quantitative)
(6) Plain films of the abdomen
Pancreatic calcifications a third to half the time.
(7) Abdominal ultrasound
Identify pseudocysts
(8) CT of abdomen
(a) Identify pseudocysts
(b) Identify dilation of main pancreatic duct
(c) Rule out cancer of the pancreas

3. TREATMENT
a. Pain control
b. Control malabsorption
c. Low fat diet
d. Stop ETOH
e. $H_2$-receptor antagonists
f. Fat-soluble vitamin supplementation
g. Correct the endocrine insufficiency

4. PROGNOSIS
a. Ranson's criteria
(1) On admission
(a) > age 55
(b) WBC > 16,000/mm$^3$
(c) Abnormal liver function studies
- LDH >350 IU/L
- SGOT >250 U/L

(d) Blood glucose >200 mg/dL
(2) Within 48 hours of admission
(a) ↑ BUN >5 mg/dL
(b) ↓ hematocrit by ≥10%
(c) ≥6 L volume replacement
(d) Base deficit >4 mEq/L
(e) Serum calcium <8 mg/dL
(f) $PaO_2$ <60 mm Hg
(3) Morbidity and mortality
(a) <3 criteria
1% mortality

(b) 3-7 criteria
- 50% require ICU admission
- 15% mortality

(c) >7 criteria
- 100% require ICU admission
- 50% mortality

C. *Pancreatic Cancer*

1. DEFINITION
   a. A primary malignancy of the exocrine pancreas.
   b. **≈90% are duct cell adenocarcinomas.**
      66% of the time these lesions are in the head of the pancreas
2. DIAGNOSIS
   a. History and Physical
      (1) **Signs and symptoms of illness occur late in the course of disease.**
         (a) Weight loss
         (b) Pain
         (c) Jaundice
         (d) Anorexia
         (e) Hepatomegaly (late)
         (f) Epigastric mass (late)
   b. Laboratory
      (1) Lipase, amylase, and liver function studies may be abnormal but cannot distinguish cancer from pancreatitis.
      (2) Abdominal CT scan
         Diagnosis ≈90% of the time
      (3) (ERCP) Endoscopic retrograde cholangiopancreatography
         Diagnosis ≈90-95% of the time
3. TREATMENT
   a. Surgery
      (1) ≈10% are resectable, of these 5-year survival is ≈10%.
      (2) Palliative surgery to relieve symptoms
   b. Chemotherapy ± radiation = partial response in ≈15%.
   c. Pain control
   d. Control malabsorption
4. PROGNOSIS
   5-year survival ≈1%.