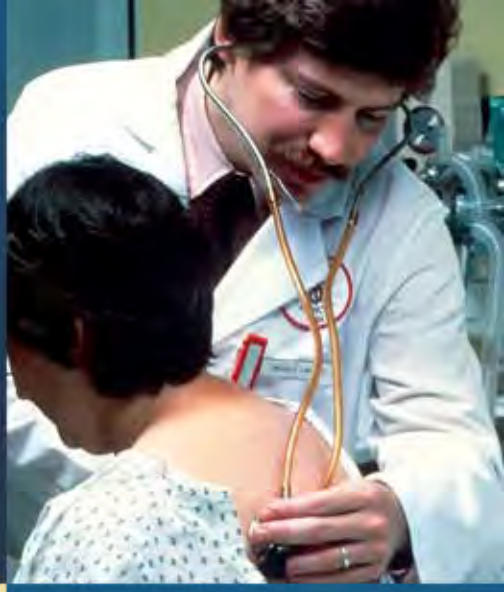
# Human Diseases



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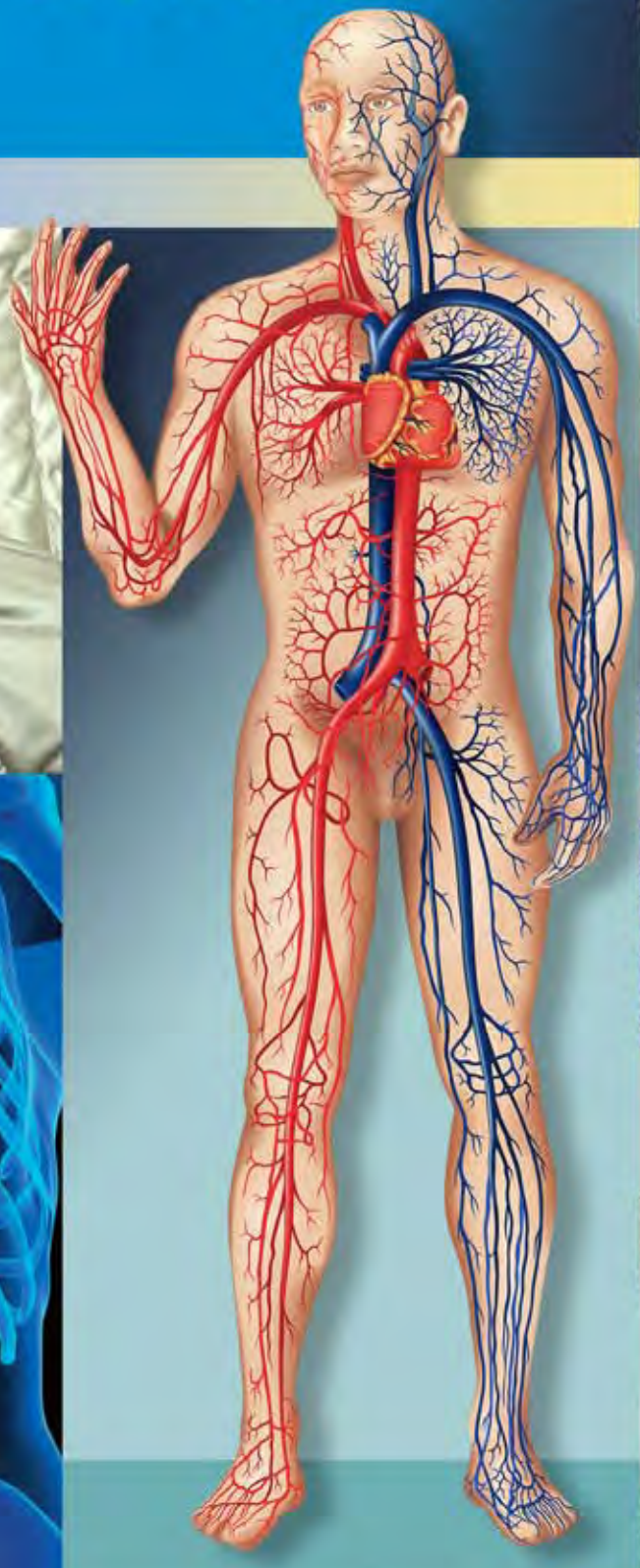

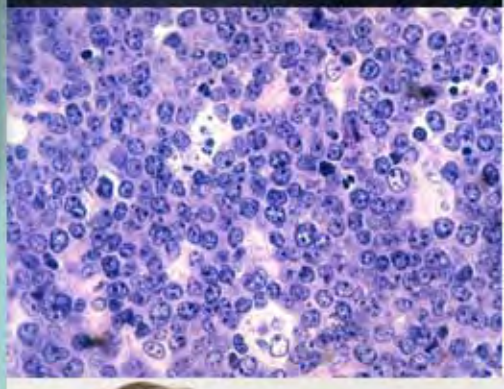

Health Professions Institute

# Human Diseases

## Third Edition

by

**John H. Dirckx, M.D.**

**Health Professions Institute**

**Modesto, California**

**2009**

# *Human Diseases*
## Third Edition

**by John H. Dirckx, M.D.**



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*For my*

*daughter Patricia*

*with love*

# Preface

*Human Diseases* is intended to provide students and practitioners of medical transcription with a grasp of basic information about the causes, symptoms, diagnosis, and treatment of common diseases. It should also prove useful to workers in the allied health professions, health information management, insurance, law, and other fields who need clear, concise data about these topics. Rare diseases and arcane, trivial, and controversial issues have been carefully avoided throughout.

Discussions of specific diseases are self-contained and can be profitably consulted in isolation from adjacent material. However, topics are presented in an orderly sequence (proceeding from the general to the particular and from the known to the unknown) so as to make *Human Diseases* useful as a textbook. The earlier chapters set forth basic principles, and the later ones discuss, one by one, the bodily systems and important disorders to which they are subject. Preliminary discussions in each of the later chapters review relevant anatomy and physiology and describe symptoms, signs, and diagnostic measures.

This book presupposes some familiarity with the basic concepts and terminology of human biology and healthcare. Most of the less common terms presented in the text are defined in parentheses when they first occur. A Glossary of concise definitions for these and many other terms can be found at the end of the book. Terms that cannot be found in the Glossary should be sought in the Index, and vice versa.

**About the Case Studies**. The exercises based on case studies (found on the accompanying CD-ROM on the inside back cover) have been designed to impart an element of reality and immediacy to this introduction to clinical medicine. The diagnostic process is frequently a series of fumbles and often enough the problem goes away (or the patient dies) before any clear diagnosis can be made. Efforts at treatment go awry, confuse the clinical picture, aggravate the condition under treatment. Social and psychological issues are ever-present to complicate diagnosis and therapy. The patient's personality, beliefs, and lifestyle may present insurmountable obstacles to a satisfactory outcome.

A puzzle is much harder to solve when pieces are missing, and some pieces are virtually always missing in clinical diagnosis. The history as presented to the physician may be incomplete or inaccurate, diagnostic maneuvers may yield equivocal or enigmatic results, and the physician may simply be unaware or ignorant of the information essential to correct diagnosis and appropriate treatment. To reproduce this ambience of uncertainty and ambiguity, the cases as presented may not accurately or fully represent the underlying reality, and some of the information needed to answer the questions is not in this book. And some of the questions simply have no answers.

*John H. Dirckx, M.D.*

# Contents


Page

Preface . . . . . v
Art Acknowledgments . . . . . viii
List of Figures . . . . . ix
About the Exercises . . . . . xi

Chapter
1 The Nature of Disease and the Diagnostic Process . . . . . 1
2 Genetic Disorders . . . . . 9
3 Infectious Diseases . . . . . 19
4 The Immune System . . . . . 29
5 Neoplasia . . . . . 39
6 Trauma and Poisoning . . . . . 47
7 Diseases of the Skin . . . . . 55
8 Diseases of the Cardiovascular System . . . . . 67
9 Diseases of the Ear, Nose, and Throat . . . . . 83
10 Diseases of the Respiratory System . . . . . 91
11 Diseases of the Digestive System . . . . . 99
12 The Excretory System, the Male Reproductive System, and Sexually Transmitted Diseases . . . . . 113
13 Diseases of the Female Reproductive System . . . . . 123
14 Pregnancy and Childbirth . . . . . 133
15 Disorders of Metabolism, Nutrition, and Endocrine Function . . . . . 143
16 Disorders of Blood Cells, Blood-Forming Tissues, and Blood Coagulation . . . . . 153
17 Musculoskeletal Disorders . . . . . 163
18 Diseases of the Eye . . . . . 171
19 Diseases of the Nervous System . . . . . 181
20 Mental Disorders . . . . . 197

Glossary . . . . . 208

Index . . . . . 219

The Author . . . . . 230

Exercises and Case Studies on CD-ROM, inside back cover

## Art Acknowledgments

The numerous medical images and illustrations throughout this textbook were obtained from a number of sources. Many images used are in the public domain from government institutes such as the National Cancer Institute, Centers for Disease Control, National Eye Institute, National Institutes of Health, National Institute of Mental Health, and Public Health Image Library (PHIL), some of which were made available at no charge through **www.Wikipedia.org**. Other images were licensed for publication use at very low fees from the popular on-line stock photos and image sites, **www.Fotolia.com** and **www.Dreamstime.com**.

# List of Figures


| Figure | | Page |
|---|---|---|
| 1 | Karyotype of Normal Chromosomes | 12 |
| 2 | Clubbing of Fingers | 13 |
| 3 | Cleft Lip and Palate | 16 |
| 4 | Down Syndrome | 16 |
| 5 | Oncogenes | 17 |
| 6 | Bacteria | 20 |
| 7 | Tick | 26 |
| 8 | Varicella (Chickenpox) | 27 |
| 9 | Herpes Zoster of the Chest | 28 |
| 10 | The Immune System | 30 |
| 11 | AIDS Life Cycle | 32 |
| 12 | Kaposi Sarcoma | 33 |
| 13 | Rheumatoid Arthritis | 35 |
| 14 | Metastasis Sites | 40 |
| 15 | How Cancer Spreads | 41 |
| 16 | Breast Cancer | 44 |
| 17 | Prostate and Nearby Organs | 45 |
| 18 | Bowel Resection for Colon Cancer | 46 |
| 19 | Microscopic Anatomy of the Skin | 56 |
| 20 | Oral Candidiasis (Thrush) | 60 |
| 21 | Herpes Simplex | 60 |
| 22 | Hemangioma (Strawberry Mark) | 62 |
| 23 | Urticaria (Hives) | 64 |
| 24 | Skin and Joint Changes in Psoriasis | 65 |
| 25 | Melanoma | 66 |
| 26 | Cardiovascular System | 68 |
| 27 | Heart and Vessels | 69 |
| 28 | Severe Stenosis of the Carotid Artery | 71 |
| 29 | Electrocardiogram Limb and Chest Leads | 72 |
| 30 | Holter Monitoring | 72 |
| 31 | Implanted Pacemaker | 76 |
| 32 | The Ear | 84 |
| 33 | Nose and Throat | 87 |
| 34 | CPAP Treatment for Obstructive Sleep Apnea | 90 |
| 35 | The Respiratory System | 92 |
| 36 | Gross Pathology of Centrilobular Emphysema Characteristic of Smoking | 97 |
| 37 | The Digestive System | 100 |

## List of Figures *(continued)*


| Figure | | Page |
|---|---|---|
| 38 | Colon and Rectum | 105 |
| 39 | Digital Rectal Exam | 106 |
| 40 | Inguinal Hernia | 107 |
| 41 | Liver and Nearby Organs | 108 |
| 42 | Pancreas | 111 |
| 43 | Pancreas and Nearby Organs | 111 |
| 44 | Male Urinary Tract | 114 |
| 45 | Female Urinary Tract | 114 |
| 46 | The Kidney | 116 |
| 47 | Male Reproductive System | 119 |
| 48 | Female Reproductive System, Lateral View | 124 |
| 49 | Female Reproductive System, Anterior View | 125 |
| 50 | Cells of Cervix | 131 |
| 51 | Breast and Lymph Nodes | 132 |
| 52 | Pregnancy | 137 |
| 53 | Clamping and Cutting of Newborn Umbilical Cord | 139 |
| 54 | Ectopic Pregnancy | 139 |
| 55 | Endocrine System | 144 |
| 56 | Thyroid Gland | 147 |
| 57 | Insulin Injection | 151 |
| 58 | Foot Examination of Patient with Diabetes | 152 |
| 59 | Blood Cells | 154 |
| 60 | Leukemia | 159 |
| 61 | Musculoskeletal System | 164 |
| 62 | Scoliosis | 166 |
| 63 | Bursitis of Elbow | 167 |
| 64 | Heberden Nodes | 170 |
| 65 | The Eye | 172 |
| 66 | Glaucoma Surgery | 177 |
| 67 | Retinal Detachment as Shown by Slit Lamp Examination | 179 |
| 68 | Brain and Spinal Cord | 182 |
| 69 | Major Parts of Brain | 183 |
| 70 | Meninges | 183 |

## About the Exercises

**To the Student**: Whether you are an independent study student or enrolled in a traditional classroom or distance-education program, you will find *Human Diseases* an interesting and engaging text. You will get the most from your study efforts if you first familiarize yourself with the entire book, reading all the introductory material and examining closely the Contents and the Index. Review the last section of Chapter 1 where the author describes how the material is presented and explains key terms. Before reading a chapter, review the Chapter Outline and Learning Objectives. Then look ahead to the "Questions for Study and Review" on the CD-ROM on the inside back cover. This "preview" sets the stage for what you are about to read and will improve your understanding and retention. Complete the exercises for each chapter as assigned by your teacher. When doing the "Case Study: You're the Doctor" sections, don't read ahead until you have completed all the questions for that segment. Don't look ahead to see what happens next! The "Suggestions for Additional Learning Activities" are for all students. Some require you to do research outside of this textbook while others may require the assistance of classmates, friends, or family members, especially for learning games. Even though these exercises may seem more like fun than actual work, they promote "whole brain learning" and will aid your mastery of the study of human diseases. Answers to objective questions appear on the accompanying CD-ROM as well. Some questions are more subjective and will not have a single right answer.

**To the Teacher**: This third edition of *Human Diseases* contains an expanded and multi-faceted selection of exercises to help your students master the material and build essential critical thinking skills that will help them excel in school and in the workplace. "Questions for Study and Review" on the CD-ROM can be assigned as self-graded homework and discussed in class or be completed in the classroom as a test of reading comprehension. In "Case Study: You're the Doctor," students are asked to render their opinions on both clinical and ethical dilemmas. Have students complete the first case study in the classroom, working together or in small groups, reviewing Dr. Dirckx's Preface before they begin. The case studies are presented in segments, each appearing in a shaded box followed by a series of questions. Encourage students to answer all the questions for each segment, without looking ahead to the next segment. In "Suggestions for Additional Learning Activities," you'll find ideas for creative classroom activities and homework assignments that will add interest and variety to your course. Some activities require students to go outside the text for more information. Others require interaction with others in learning groups. And virtually all of the individual Learning Activities can be adapted for the classroom by asking students to work together, compare their work with others, or present their findings to the class. The answers to objective questions are placed on the CD-ROM for easy access. An exhaustive Index is also included at the end of the textbook as an indispensable study aid.

*Georgia Green, CMT, AHDI-F*

# 11

# Diseases of the Digestive System

**Chapter Outline**


**ANATOMY AND PHYSIOLOGY OF THE DIGESTIVE SYSTEM**

**SYMPTOMS AND SIGNS OF DIGESTIVE DISORDERS**

**DIAGNOSTIC PROCEDURES IN DIGESTIVE DISORDERS**

**DISEASES OF THE ESOPHAGUS**
- Gastroesophageal Reflux Disease (GERD)

**DISEASES OF THE STOMACH AND INTESTINE**
- Peptic Ulcer Disease and Gastritis
- Gastroenteritis
- Appendicitis
- Irritable Bowel Syndrome (IBS)

**INFLAMMATORY BOWEL DISEASE**
- Crohn Disease (Regional Enteritis, Regional Ileitis)
- Ulcerative Colitis

**OTHER INTESTINAL DISORDERS**
- Diverticulosis and Diverticulitis
- Intestinal Obstruction
- Adynamic Ileus

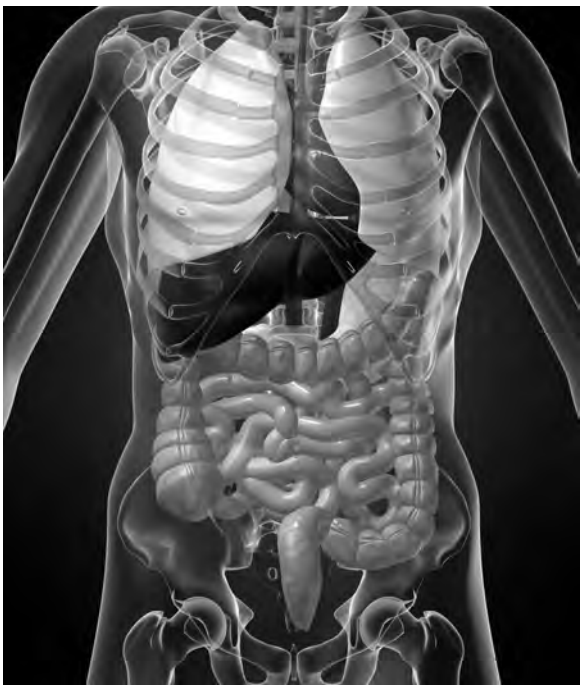
**DISORDERS OF THE RECTUM AND ANUS**
- Hemorrhoids
- Anal Fissure

**DISORDERS OF THE PERITONEUM**
- Acute Peritonitis


**LEARNING OBJECTIVES**

Upon completion of this chapter, you should be able to

- describe the basic anatomy and physiology of the digestive system;
- explain diagnostic procedures and treatments for digestive disorders and hepatobiliary disease;
- classify common diseases of the digestive system by their signs, symptoms, and treatment.



## ABDOMINAL HERNIA

## DISEASES OF THE LIVER

Hepatitis A
Hepatitis B
Hepatitis C
Hepatitis D (Delta Hepatitis)
Hepatitis Cirrhosis (Portal Cirrhosis, Laënnec Cirrhosis)

## DISEASES OF THE GALLBLADDER AND BILIARY TRACT

Cholelithiasis (Gallstones)
Acute Cholecystitis

## DISEASES OF THE PANCREAS

Acute Pancreatitis
Chronic Pancreatitis

## QUESTIONS FOR STUDY AND REVIEW

**See CD-ROM inside back cover.**

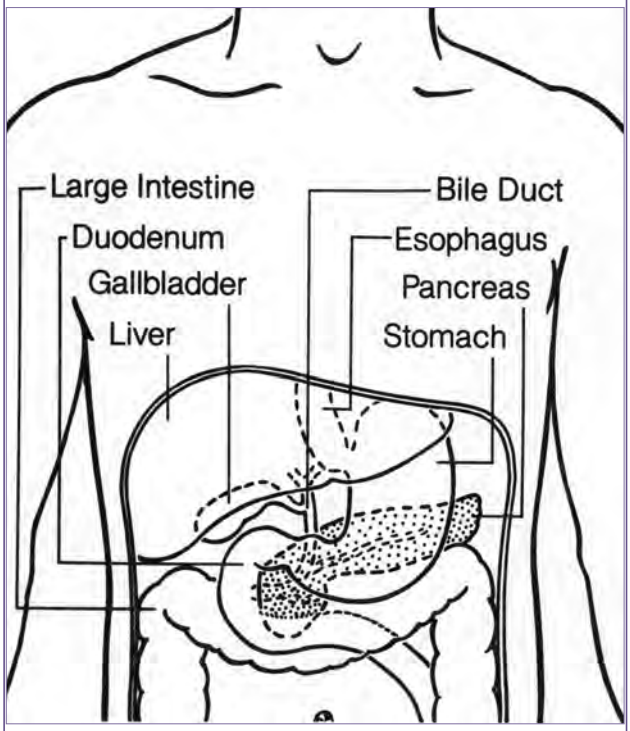
## DISEASES OF THE DIGESTIVE SYSTEM

## ANATOMY AND PHYSIOLOGY OF THE DIGESTIVE SYSTEM

The digestive system includes all those structures concerned with the ingestion of solids and liquids, their mechanical and chemical breakdown into usable nutrients, the absorption of these into the circulation, and the excretion of solid wastes. The alimentary canal is a coiled but unbranched tube extending from the lips to the anus and divided into mouth, oropharynx, esophagus, stomach, small intestine (duodenum, jejunum, ileum), and large intestine (colon, rectum) (Figure 37).

Numerous microscopic glandular structures occur in the walls of the digestive tract (gastric glands, intestinal glands), and in addition larger secretory organs (salivary glands, liver, pancreas) pour their products through ducts into parts of the tract. These secretions serve to liquefy and lubricate food and to break down

**Figure 37. The Digestive System**




National Cancer Institute

fats, proteins, and carbohydrates to fatty acids, amino acids, and simple sugars, respectively.

## SYMPTOMS AND SIGNS OF DIGESTIVE DISORDERS

**Dysphagia** (difficulty in swallowing), **odynophagia** (pain on swallowing).

**Anorexia** (loss of appetite), nausea, vomiting.

**Hematemesis** (vomiting of blood).

**Constipation** (firm, difficult stools), **obstipation** (total inability to pass stool).

**Diarrhea** (abnormal frequency, urgency, and looseness of stools), **lientery** (passage of undigested food in stools).

**Hematochezia** (passage of blood from the rectum), **melena** (black stools, often due to the presence of blood), pale or white stools (due to absence of bile flow into the intestine), **steatorrhea** (passage of bulky, greasy stools due to nonabsorption of dietary fat).

**Tenesmus** (painful, often ineffectual straining to defecate).

**Abdominal pain** (diffuse or localized; intermittent or constant; random or affected by body position, meals, certain foods, bowel movement, medicine). Sharp, crampy pains are often referred to as colicky. Burning pain in the epigastrium or chest due to digestive disorders may be called heartburn.

**Abdominal tenderness** (local or generalized), rebound tenderness (additional stab of pain when pressure on abdomen is released, often indicating peritoneal irritation), spasm of abdominal wall, palpable abdominal mass, enlargement of abdominal organs, abnormal bulge of abdominal wall.

**Bloating, belching, flatulence** (excessive intestinal gas). **Tympanites** (hollow percussion note due to distention of underlying digestive tract with gas).

**Borborygmi** (singular, borborygmus) (audible rumbling and gurgling sounds in the digestive tract).

**Hyperactive bowel sounds** on auscultation (due to diarrhea, mechanical obstruction), **diminished or absent bowel sounds** ("silent abdomen") due to intestinal spasm or ileus, often reflecting local or generalized peritonitis.

**Anal pain, itching, swelling, or bleeding.**

**Muscle wasting, pallor, fatigue.**

**Jaundice** (discoloration of skin and whites of eyes by excessive bile pigment).

## DIAGNOSTIC PROCEDURES IN DIGESTIVE DISORDERS

**History and physical examination**: Observation of abdomen for surgical or traumatic scars, swellings, discolorations, asymmetry; palpation of abdomen for masses, palpable organs, tenderness, spasm, hernia; percussion for hyperresonance (indicating excessive or displaced gas or air), flatness or dullness (indicating an abnormal mass, enlargement or consolidation of an organ, or impacted stool); auscultation for diminished, hyperactive, or high-pitched bowel sounds.

**External anal examination, digital rectal examination**.

**Examination of stool** for occult blood, fat, pathogens (bacteria, fungi, parasites), abnormal constituents.

**Absorption tests**, to assess the ability of the digestive tract to absorb certain nutrients or other materials, based on the determination of blood or stool levels of substances that have been ingested in measured amounts.

**Imaging studies**: Flat, upright, and (usually left) lateral decubitus films of the abdomen to assess distribution of gas and fluid in the stomach and bowel, identify free air in the peritoneal cavity, and detect other abnormalities (masses, calcifications, foreign bodies); fluoroscopic studies with swallowed or injected barium or other contrast medium (barium swallow, upper GI series, small bowel series, barium enema); percutaneous transhepatic cholangiography (PTC, injection of contrast material into biliary tract via catheter inserted through skin), endoscopic retrograde cholangiopancreatography (ERCP, endoscopically guided injection of contrast material into biliary and pancreatic ducts); CT, MRI, or ultrasound for specific indications (for example, to assess gallbladder, masses); hepatobiliary iminodiacetic acid (HIDA) scan with radioactive technetium.

**Endoscopy**: Esophagoscopy, gastroscopy, gastroduodenoscopy, anoscopy, sigmoidoscopy, colonoscopy; biopsy specimens, washings, cultures, and other materials can be obtained through endoscopes.

**Laparoscopy** (inspection of abdominal cavity through an endoscope inserted through an incision in the abdominal wall), exploratory laparotomy (inspection of the abdominal and pelvic cavities through an incision in the abdominal wall).

**Liver biopsy** (with biopsy needle inserted through skin of abdomen).

## DISEASES OF THE ESOPHAGUS

### Gastroesophageal Reflux Disease (GERD)

Backflow of gastric juice into the esophagus.

**Cause**: Structural or functional incompetence of the lower esophageal sphincter (LES), associated with disordered gastric motility and prolonged gastric emptying time. In a few cases, reflux of gastric juice may be facilitated by esophageal hiatus hernia (weakness or dilatation of the opening in the diaphragm where the esophagus passes through, with herniation of part or all of the stomach into the thorax; often asymptomatic). Reflux of acid gastric juice into the esophagus causes inflammation because the esophageal mucosa is not adapted to resist acid and digestive enzymes.

**History**: Recurrent epigastric and retrosternal distress, usually described as heartburn; belching, nausea, gagging, cough, hoarseness in varying proportions. There is a strong association with asthma, obesity, and diabetes mellitus. Symptoms are triggered or aggravated by recumbency (especially after a meal), vigorous exercise, smoking, overeating, caffeine, chocolate, alcohol, and certain drugs.

**Physical Examination**: Unremarkable.

**Diagnostic Tests**: Imaging studies confirm reflux of swallowed barium from the stomach and may identify ulceration or stricture. Twenty-four-hour monitoring of esophageal pH with a swallowed electrode confirms a sustained abnormal acid state in the esophagus. Endoscopy gives direct visual proof of inflammation and may identify a zone of Barrett esophagus (cellular change due to chronic inflammation).

**Course**: The underlying disorder of the LES and of gastric motility is irreversible. Severe reflux disease can lead to peptic ulceration of the esophagus, with eventual stricture due to scarring. Barrett esophagus, a metaplasia (transformation) of normal squamous esophageal epithelium into columnar epithelium, can progress to adenocarcinoma.

**Treatment**: Avoidance of smoking, alcohol, caffeine, and large meals. Over-the-counter antacids may suffice to control symptoms. Otherwise acid production may require suppression by $H_2$ antagonists (cimetidine, ranitidine, famotidine, nizatidine) or proton pump inhibitors (omeprazole, lansoprazole). Prokinetic drugs (bethanechol, metoclopramide) may improve sphincter function and gastric motility.

## DISEASES OF THE STOMACH AND INTESTINE

### Peptic Ulcer Disease and Gastritis

Inflammation and ulceration of the stomach, duodenum, or both by acid gastric juice.

**Cause**: Most commonly, infection of the gastric mucosa by *Helicobacter pylori*, a motile bacterium that survives in the acid environment of the stomach by secreting urease, an enzyme that converts urea to ammonia and bicarbonate, thus providing itself with a protective alkaline medium. *H. pylori* infection, which is spread from person to person by the fecal-oral route, results ultimately in a marked increase of acid production. Peptic ulceration can also result from regular use of prostaglandin-inhibiting drugs: adrenal corticosteroids and nonsteroidal anti-inflammatory agents such as ibuprofen and aspirin. In rare cases it is part of Zollinger-Ellison syndrome, in which a tumor of the pancreas produces excessive amounts of the hormone gastrin and thus causes hypersecretion of gastric acid. Severe stress, head injuries, and burns are sometimes complicated by peptic ulcer. Most peptic ulcers occur in the duodenum, but the stomach may be involved as well or instead.

**History**: Burning epigastric pain that comes on within an hour after meals and is relieved by taking antacids or food. Night pain is common. Tobacco, alcohol, caffeine, and certain foods aggravate symptoms, apparently by stimulating acid production. With complications: hematemesis, melena, early satiety (feeling that the stomach is full after only one or two mouthfuls of food), weight loss, severe abdominal pain, collapse.

**Physical Examination**: Unremarkable. Abdominal tenderness is variable and may be absent. With hemorrhage: pallor and tachycardia. With perforation: board-like rigidity of the abdomen due to chemical peritonitis.

**Diagnostic Tests**: Upper GI studies with barium contrast medium can show ulceration, scarring, obstruction, or perforation. Endoscopy visualizes ulcers, bleeding sites, and scarring, and is important to rule out carcinoma in gastric lesions. Infection by *H. pylori* can be confirmed by culture, biopsy, serologic testing, or breath-testing for evidence of urease activity on orally administered, radioactively tagged urea.

**Course**: Without treatment, peptic ulcer disease tends to persist, with remissions and exacerbations, for many years. The most serious complications are hemorrhage (the principal cause of ulcer mortality), obstruction due to scarring, perforation of the digestive

tract with release of gastric juice into the peritoneal cavity, and penetration into the retroperitoneal space.

**Treatment**: Smoking cessation, avoidance of alcohol and caffeine. Acidity may be adequately controlled by over-the-counter antacids, $H_2$ antagonists (cimetidine, ranitidine, famotidine, nizatidine) in over-the-counter or prescription strength, or proton pump inhibitors (omeprazole, lansoprazole). Proven *H. pylori* infection is treated with a course of therapy including bismuth subsalicylate and two antibiotics: tetracycline or amoxicillin, and metronidazole or clarithromycin.

## Gastroenteritis

Inflammation of the stomach and intestine, manifested by abdominal pain, vomiting, and diarrhea; usually acute, infectious, and self-limited.

**Causes**: Infection with viruses (adenovirus, echovirus, coxsackievirus, rotavirus), bacteria (*Escherichia coli* H157:O7 and other virulent strains, *Campylobacter*, *Yersinia*, *Salmonella*, *Shigella*, *Clostridium*), protozoa (*Entamoeba histolytica*, *Giardia lamblia*), fungi (*Candida albicans*). Most of these infections are acquired by the fecal-oral route. Some are much more likely to occur in immunocompromised persons. Outbreaks are usually due to contaminated food or water. "Food poisoning" is due to toxins produced by staphylococci, *Salmonella*, *Clostridium*, or other organisms. Gastroenteritis can also be a reaction to medicines, foods, poisonous plants, toxic chemicals.

**History**: Usually abrupt onset of abdominal distress or cramping, anorexia, nausea, vomiting, and diarrhea. Chills, fever, malaise. Hematemesis and bloody diarrhea are ominous signs. In severe or protracted disease, or in children or the elderly, dehydration and electrolyte depletion can lead to prostration, vascular collapse, and death.

**Physical Examination**: May be unremarkable. Abdominal tenderness, tympanites (hollow percussion note due to distention of bowel with gas), hyperactive bowel sounds. In severe disease, signs of dehydration and electrolyte depletion include dryness of mucous membranes, decreased skin turgor (loss of normal consistency and fullness), tachycardia, hypoactive deep tendon reflexes, and decreased urine output.

**Diagnostic Tests**: Stool examination for white blood cells and organisms, with culture for pathogenic bacteria. Blood studies may show hematologic abnormalities or fluid and electrolyte imbalance.

**Course**: Most cases of gastroenteritis, even those caused by bacteria such as *Salmonella*, *Campylobacter*, and *Yersinia*, resolve spontaneously without specific treatment. However, cholera (due to *Vibrio cholerae*; rare in the U.S.), bacillary dysentery (due to *Shigella* species), typhoid fever (due to *Salmonella typhi*), and pseudomembranous enterocolitis (due to toxin-producing *Clostridium difficile*, often following treatment with antibiotics that kill normal intestinal flora) are all severe and potentially fatal infections requiring prompt, aggressive antimicrobial treatment. Any case of gastroenteritis in small children or in elderly or debilitated persons can lead to dangerous electrolyte and water depletion and vascular collapse.

**Treatment**: Largely symptomatic and supportive. Over-the-counter products may suffice to control nausea, cramping, and diarrhea. Water and electrolytes may be replaced orally or intravenously as indicated. Antibiotic treatment is indicated only in certain specific infections. Trimethoprim-sulfamethoxazole or ciprofloxacin are effective in bacillary dysentery (shigellosis), typhoid fever, and cholera; pseudomembranous enterocolitis is treated with metronidazole or vancomycin.

## Appendicitis

Acute inflammation of the appendix.

**Cause**: Obstruction of the appendiceal lumen by a fecalith (stonelike mass of hardened feces), seed, or parasite, or by swelling due to infection or neoplasm. Obstruction is followed by inflammation, impairment of blood supply, necrosis, and rupture.

**History**: Gradual onset of generalized abdominal distress gradually becoming more severe and steady and localizing in the right lower quadrant. Anorexia, nausea, vomiting, fever, chills, constipation. Sudden spontaneous relief of pain suggests perforation.

**Physical Examination**: Slight fever and tachycardia, tenderness and rebound tenderness over McBurney point (about one-third of the distance from the right anterior superior iliac spine to the umbilicus), tenderness and rebound tenderness in the same area on rectal or pelvic examination. Diminished bowel sounds. After perforation, boardlike rigidity of the abdomen indicating peritonitis, signs of toxicity, vascular collapse. In infants, the elderly, and pregnant women the findings may be atypical or deceptively mild.

**Diagnostic Tests**: Moderate elevation of the white blood cell count, with left shift (increase of band or immature forms). Abdominal imaging (focused CT) may show a mass, ileus or other signs of peritonitis, or an opacity in the appendiceal lumen; barium injected by rectum fails to fill the appendix.

**Course**: Without treatment the condition has a mortality rate over 90%. Most cases progress to per-

foration within 12-36 hours, followed by generalized peritonitis, septicemia, and collapse.

**Treatment**: Surgical removal of the appendix (by open procedure or laparoscopy) is the only effective treatment. Perforation requires surgical repair, intravenous fluids, and antibiotics.

### Irritable Bowel Syndrome (IBS)

Intermittent or chronic abdominal distress and bowel dysfunction without any demonstrable organic lesion.

**Cause**: Unknown. A derangement of the normal interaction between the brain and the bowel is postulated. IBS is more likely to occur with emotional stress, dietary irregularities, and heavy intake of caffeine. Lactose intolerance and abuse of antacids or laxatives may be partly responsible. The disorder is more common in women and in persons under 65. As many as 50% of patients report a history of verbal or sexual abuse.

**History**: Intermittent lower abdominal pain, often relieved by having a bowel movement; alternating diarrhea and constipation; a sense of inadequate evacuation after bowel movement; excessive mucus in stools; flatulence.

**Physical Examination**: Essentially negative.

**Diagnostic Tests**: Stool examinations, barium enema, colonoscopy, and blood studies are all negative. X-ray does not confirm distention of bowel with gas.

**Course**: Symptoms tend to wax and wane for many years, with intervals of complete remission.

**Treatment**: Regular eating habits, avoidance of coffee and other triggering factors. Antispasmodics may be prescribed to reduce bowel motility and cramping.

## INFLAMMATORY BOWEL DISEASE

### Crohn Disease (Regional Enteritis, Regional Ileitis)

A chronic inflammatory disease of the bowel that can lead to intestinal obstruction, abscess and fistula formation, and systemic complications.

**Cause**: Unknown. The disease shows a familial pattern of incidence.

**History**: Recurrent crampy or steady abdominal pain, nausea, diarrhea, steatorrhea (excessive fat in stool), hematochezia (blood in stool), weakness, weight loss, and fever.

**Physical Examination**: Abdominal tenderness, signs of complications.

**Diagnostic Tests**: The white blood cell count and erythrocyte sedimentation rate are elevated. There may be mild anemia and reduction of serum levels of potassium, calcium, magnesium, and other substances because of excessive bowel losses. Barium enema shows regional narrowing of the lumen ("string sign") alternating with areas of normal caliber. Sigmoidoscopy and colonoscopy show local inflammation with skip areas (intervening zones of normal mucosa). On biopsy, all layers of the bowel are seen to be involved, not just the mucosa as in ulcerative colitis.

**Course**: Complications include intestinal obstruction, formation of abscesses and fistulas, perforation of the bowel.

**Treatment**: Low-fiber diet, drugs to reduce intestinal motility, specific anti-inflammatory drugs (azathioprine, sulfasalazine, olsalazine). Surgery may be necessary to deal with perforation or fistula formation. In severe disease, segmental resection of the bowel, or colectomy (removal of the colon) with ileostomy (formation of an artificial opening from the small bowel through the anterior abdominal wall), may be necessary.

### Ulcerative Colitis

A chronic inflammatory disease of the colon, chiefly the left colon, causing superficial ulceration.

**Cause**: Unknown.

**History**: Bloody diarrhea, abdominal cramps, tenesmus, anorexia, malaise, weakness, hemorrhoids or anal fissures. Bowel movements may occur more than 20 times a day, and may awaken the patient at night.

**Physical Examination**: Fever, abdominal tenderness, signs of complications.

**Diagnostic Tests**: The white blood cell count and erythrocyte sedimentation rate are elevated. Anemia may be present. Stool examination reveals mucus, blood, and pus, but no bacteria or parasites. Serum electrolytes and protein may be depleted. Sigmoidoscopy and colonoscopy show erythematous, friable mucosa with superficial ulceration and sometimes polyp formation. Biopsy shows chronic inflammation and microabscesses of the crypts of Lieberkühn.

**Course**: The course is intermittent, with spontaneous remissions and exacerbations. Physical and emotional stress and dietary irregularities may increase symptoms. Possible complications include colonic hemorrhage, perforation, toxic dilatation (extreme dilatation of the colon, compounded by effect of bacterial toxins),

polyp formation with progression to carcinoma, arthritis, spondylitis, iritis, oral ulcers.

**Treatment**: General supportive treatment and control of diet (high protein, low milk) are crucial to long-term control of the disease. Sulfasalazine, mesalamine, and corticosteroids suppress colonic inflammation and reduce symptoms. In severe disease, hospitalization with intravenous alimentation and fluid replacement and antibiotic treatment to combat sepsis may be necessary. In intractable disease, colectomy and ileostomy may be necessary.

## OTHER INTESTINAL DISORDERS

### Diverticulosis and Diverticulitis

A diverticulum (plural, diverticula) is a blister- or bubble-like outpouching of a hollow or tubular organ. Diverticulosis of the colon is the formation of one or more such outpouchings of the colon. Diverticulitis means inflammation and infection of colonic diverticula.

**Cause**: Unknown; more common in middle-aged and elderly.

**History**: Most patients with diverticulosis have no symptoms. The diverticula may be discovered incidentally on routine examination (barium enema, colonoscopy). A few patients may experience irregular bowel habits or abdominal pain. Diverticulitis can cause acute abdominal pain, nausea, vomiting, constipation, and sometimes fever or blood in the stools.

**Physical Examination**: There may be mild fever, abdominal tenderness, and even the sensation of a mass, most often in the region of the sigmoid colon (left lower quadrant of the abdomen).

**Diagnostic Tests**: The white blood cell count and sedimentation rate may be slightly elevated. The stool may be positive for occult blood. Barium enema, sigmoidoscopy, or colonoscopy may be performed to identify and localize the lesion, but are contraindicated in the presence of acute inflammation because of the danger of perforation of the bowel. X-ray studies may be used to identify free air in the peritoneal cavity due to perforation, and CT scan to detect abscess formation. Diagnostic evaluation needs to be particularly thorough to rule out malignancy.

**Course**: Diverticulitis may lead to hemorrhage, perforation of the bowel, obstruction due to fibrous scarring, fistula formation, or abscess formation.

**Treatment**: Patients with mild or no symptoms may require no treatment, but are often advised to follow a high-fiber diet. During the acute phase of diverticulitis, patients are kept at bed rest, with nothing by mouth, intravenous fluids and nutrition, and, if necessary, a nasogastric tube. Usually antibiotic treatment is used because of the risk of peritonitis and abscess formation. Metronidazole, ciprofloxacin, and trimethoprim-sulfamethoxazole are the drugs usually used. As many as one-third of patients with diverticulitis will need surgery to drain an abscess or to resect a segment of badly diseased colon.

Adenocarcinoma of the colon and rectum (anatomy, Figure 38) is discussed in Chapter 5.

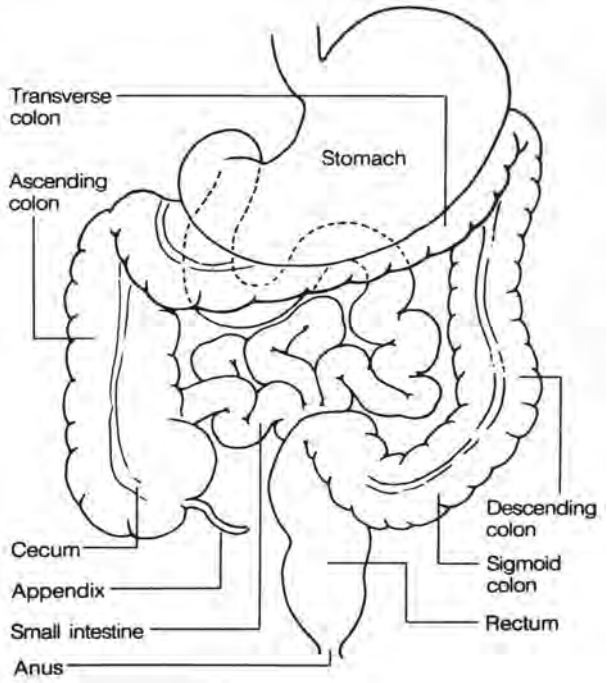
### Intestinal Obstruction

Blockage of the flow of digestive fluids through the small or large intestine.

**Causes**: Surgical adhesions, hernia, neoplasms, gallstones, volvulus (twisting of a loop of intestine), intussusception (passage of a segment of intestine into the segment distal to it), foreign body, fecal impaction. Obstruction due to causes outside the bowel (volvulus, hernia) are often complicated by strangulation (ischemia of the involved portion of bowel).

**History**: Crampy abdominal pain, nausea, vomiting, obstipation. Obstruction of the small intestine causes more severe and rapidly progressing symptoms than obstruction of the colon.

**Figure 38. Colon and Rectum**

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**Physical Examination**: Abdominal distention, borborygmi (gurgling sounds due to intestinal activity); increased bowel sounds, often high-pitched or in peristaltic rushes (urgent-sounding series of squeaking or gurgling sounds occurring with overactive peristaltic movements). A fullness or mass may be palpated at the site of obstruction. Tenderness, if strangulation has occurred. The rectum is empty of stool unless fecal impaction is the cause of obstruction.

**Diagnostic Tests**: The white blood cell count is elevated, particularly in the presence of strangulation. Blood chemistries may show electrolyte imbalance and dehydration due to vomiting and sequestration of fluid above the obstruction. Abdominal x-rays show dilated loops of bowel containing fluid levels, and may demonstrate the cause (volvulus, gallstone). Barium enema may be necessary to identify an obstruction in the colon.

**Treatment**: A nasogastric tube with suction to decompress the bowel proximal to the obstruction. Intravenous fluids to correct dehydration and electrolyte imbalance. Surgery is often necessary to relieve obstruction and to resect infarcted areas of bowel in cases of strangulation.

### Adynamic Ileus

Failure of normal flow of materials through the digestive tract because of atony or paralysis of the bowel.

**Causes**: Recent abdominal surgery, peritonitis, mesenteric ischemia or infarction, medicines (opiates, anticholinergics).

**History**: Nausea, vomiting, obstipation, abdominal distention. Pain mild or absent.

**Physical Examination**: Abdominal distention, little or no tenderness, bowel sounds diminished or absent.

**Diagnostic Tests**: X-ray of the abdomen shows distended loops of small intestine with fluid levels.

**Treatment**: Nasogastric tube and suction, intravenous fluids, correction of the underlying cause if possible.

## DISORDERS OF THE RECTUM AND ANUS

### Hemorrhoids

Dilated veins just above or just below the anus.

**Cause**: Unknown. Constipation with straining at stool, prolonged sitting, and local infection have been implicated.

**History**: Anorectal discomfort or pain, swelling or protrusion, and bleeding.

**Physical Examination**: Dilated veins externally or internally, as seen by external inspection or endoscopy. Sigmoidoscopy or colonoscopy and barium enema may be performed to rule out malignancy.

**Course**: Symptoms are typically mild and intermittent. Bleeding is occasionally significant. Thrombosis of a hemorrhoid results in acute pain and swelling, but the problem resolves spontaneously in a few weeks.

**Treatment**: High-fiber diet, stool softeners, hot sitz baths, soothing applications or suppositories. With severe pain or bleeding, surgery is indicated. Band ligation is used for internal hemorrhoids; external hemorrhoids are treated by excision or cryosurgery.

### Anal Fissure

A superficial longitudinal ulceration of the anal canal.

**Cause**: Probably trauma from a hard stool or hard, sharp material in stool. Chronic fissure may result from infection.

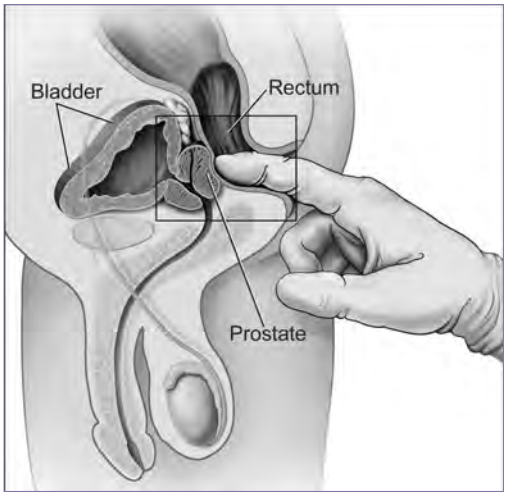
**History**: Anorectal pain and bleeding, chiefly with bowel movements.

**Physical Examination**: Fissure in anal canal. With chronic fissure a tag of anoderm (sentinel pile) may form below the fissure. Digital examination (Figure 39) demonstrates anal tenderness and spasm.

**Course**: Acute fissures heal spontaneously in a few days. Chronic fissure may persist for weeks or months.

**Treatment**: High-fiber diet, stool softeners, hot sitz baths, anesthetic or anti-inflammatory ointments or suppositories. Severe chronic fissure occasionally requires surgical excision.

**Figure 39. Digital Rectal Exam**

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## DISORDERS OF THE PERITONEUM

The peritoneum is a delicate serous membrane that lines the abdominal and pelvic cavities (parietal peritoneum) and also covers the stomach, small intestine, and colon (except for the distal part of the rectum), as well as the liver, spleen, uterus, ovaries, ureters, and dome of the bladder (visceral peritoneum). Structures such as the pancreas and kidneys that lie behind the peritoneal cavity are called retroperitoneal.

### Acute Peritonitis

Acute inflammation of the peritoneum.

**Causes**: Infection (penetrating abdominal wounds, surgery, peritoneal dialysis for renal failure, spread from digestive or urinary tract or from a systemic site); chemical irritation (leakage of gastric or intestinal contents, bile, or pancreatic secretions from an injured, diseased, or perforated structure); systemic disease, neoplasm.

**History**: Fairly abrupt onset of severe local or generalized abdominal pain, nausea, vomiting, fever.

**Physical Examination**: Elevated temperature and pulse. Boardlike rigidity of abdomen, tenderness, and rebound tenderness. Diminished or absent bowel sounds and abdominal distention due to ileus.

**Diagnostic Tests**: The white blood cell count is elevated. Blood studies may also show electrolyte imbalances due to peritoneal effusion, vomiting, and dehydration. Anemia may occur. Fluid obtained by abdominal paracentesis (entry of peritoneal cavity with a needle passed through the abdominal wall) may show amylase or lipase (indicating leak of intestinal contents or pancreatic juice), significant cellular abnormalities, or infecting microorganisms. Various types of imaging may be of use in confirming and identifying intra-abdominal catastrophe.

**Course**: Without treatment the outlook is poor. Septicemia and vascular collapse often occur within a few hours of onset. In some patients, peritonitis becomes localized, with abscess formation, particularly subphrenic (just below diaphragm) or pelvic. Peritonitis often results in eventual formation of fibrous adhesions that may produce intestinal obstruction.

**Treatment**: Hospitalization, nothing by mouth, gastrointestinal suction to decompress the bowel and draw off secretions, intravenous fluids, narcotics for pain, antibiotics for infection, surgery to repair underlying abnormality.

## ABDOMINAL HERNIA

A localized weakness in the musculoaponeurotic wall of the abdomen, with protrusion of abdominal contents. Abdominal hernias are classified according to position as:

Umbilical (at the navel): Often congenital, seldom requiring surgical repair because it resolves during infancy.

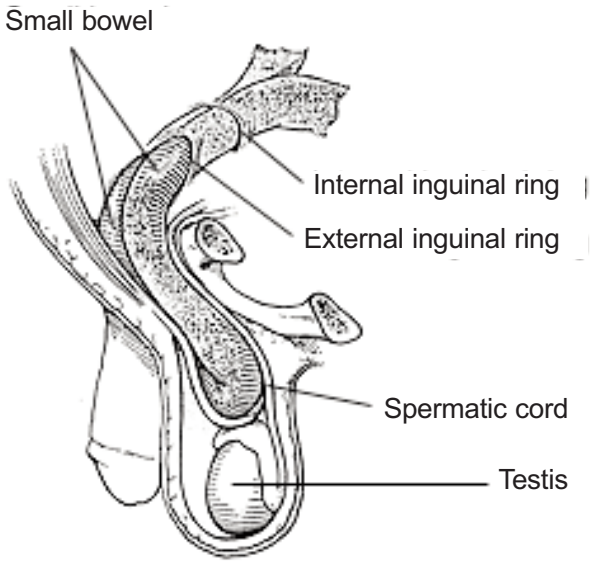
Inguinal (in the groin) (Figure 40):

- Direct inguinal: Due to thinning and stretching of the lower abdominal wall, often with aging.
- Indirect inguinal (usually congenital): Weakness and bulging in the inguinal canal, the passage through which, in the male fetus, the testicle descends from the abdominal cavity to the scrotum; a similar potential passage exists in women.

Femoral: Herniation into the femoral canal, through which the femoral artery and vein pass from the pelvis into the thigh.

**Cause**: Congenital weakness or malformation; thinning of the abdominal musculature by aging. Herniation may be precipitated or aggravated by vigorous or repeated straining of the abdominal wall (chronic constipation, urinary obstruction, heavy lifting, chronic cough).

**Figure 40. Inguinal Hernia**




National Institutes of Health, Wikipedia.org

**History**: A tender bulge in the abdominal wall that enlarges with straining. Intestinal obstruction may occur, with severe abdominal pain, nausea, vomiting, weakness, shock, and collapse.

**Physical Examination**: A fluctuant bulge in the abdominal wall that enlarges with straining and can be reduced with manipulation or recumbency unless incarceration has occurred. A defect in the abdominal wall at the site of the hernia can be palpated. Visible or palpable mass, tenderness. There may be evidence of strangulation or bowel obstruction.

**Diagnostic Tests**: Barium enema and other studies may be done to rule out obstructive disease of the bowel or urinary tract.

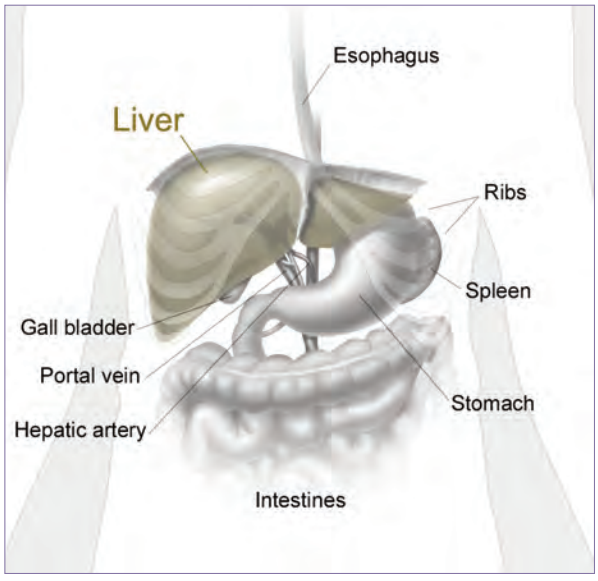
**Complications**: Strangulation (compromise of blood supply), incarceration (inability to reduce hernia), bowel obstruction.

**Treatment**: Surgical repair of the defect, sometimes with implantation of reinforcing mesh.

## DISEASES OF THE LIVER

The liver, the largest gland in the body, lies in the right upper quadrant of the abdomen just below the diaphragm and is largely covered by peritoneum (Figure 41). The portal vein carries nutrients and other substances from the digestive tract to the liver. The liver performs numerous vital functions and is intimately concerned with carbohydrate and nitrogen metabolism and with removal of certain waste products. Bile, the secretory product of the liver, passes through a duct into the duodenum. Bile contains bile salts, which help in the digestion of fats, and bilirubin, a breakdown product of hemoglobin. Bile does not flow steadily into the duodenum, but is stored in the gallbladder, a bulb or pouch connected by the cystic duct to the common bile duct. Ingestion of a fatty meal stimulates contraction of the gallbladder and increased flow of bile into the intestine.

**Figure 41. Liver and Nearby Organs**

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### Hepatitis A

**Cause**: Hepatitis A virus (HAV). Transmission is by the fecal-oral route. Contaminated food and water are important means of infection.

**History**: Anorexia, nausea, vomiting, malaise, upper respiratory or flulike symptoms, fever, joint pain, aversion to tobacco, abdominal discomfort, diarrhea or constipation. Infection may be asymptomatic in children.

**Physical Examination**: Fever, jaundice, enlargement and tenderness of the liver, splenomegaly, cervical lymphadenopathy.

**Diagnostic Tests**: The serum bilirubin is elevated, and liver function tests are abnormal. Atypical lymphocytes may appear in the blood. Anti-HAV (IgM) antibody appears early in the course of the disease and disappears after recovery. IgG antibody develops later and persists indefinitely, indicating past history of, and immunity to, the disease.

**Course**: Symptoms characteristically resolve within 2-3 weeks. The mortality is very low.

**Treatment**: Supportive and symptomatic.

### Hepatitis B

**Cause**: Hepatitis B virus. Transmission is by blood (shared needles, needlestick injury in healthcare workers) or sexual contact. Maternal transmission to neonates also occurs.

**History**: Fever, anorexia, nausea, vomiting, malaise, joint pain and swelling, rash, aversion to tobacco, abdominal pain, bowel irregularities.

**Physical Examination**: Fever, jaundice, enlargement and tenderness of liver. Splenomegaly, cervical lymphadenopathy.

**Diagnostic Tests**: The serum bilirubin is elevated, and liver function tests are abnormal. Atypical lymphocytes may appear in the blood. Hepatitis B surface antigen (HBsAg) appears early in the disease and indicates

presence of infection and infectivity of the patient. Antibody to surface antigen (AntiHBs) indicates recovery, immunity to future infection, and lack of infectivity. Presence of HBsAg after the acute phase suggests chronic infection.

**Course**: The incubation period may be 6-12 weeks or longer, and acute illness may persist for as long as 16 weeks. The mortality rate is somewhat higher than that of hepatitis A. Some patients become carriers of the disease, able to transmit infection months or years after recovery. In some, a chronic phase occurs. Chronic persistent hepatitis is mild and generally asymptomatic, while chronic active hepatitis leads to gradual deterioration of liver function, cirrhosis, and an appreciable risk of hepatocellular carcinoma.

**Treatment**: Chiefly supportive. Chronic hepatitis is treated with interferon alfa-2b and lamivudine.

### Hepatitis C

**Cause**: Hepatitis C virus (HCV). Transmission is by sharing of needles among drug-abusers (60% of all cases), contaminated blood or blood products, transplanted organs, sexual contact, and from mother to fetus.

**History**: Malaise, weakness, anorexia, fever, jaundice. Symptoms of acute infection are typically mild and occur in only one-third of patients. About 85% of patients eventually develop chronic disease, with unpredictable progression toward hepatic failure.

**Physical Examination**: May be unremarkable until signs of hepatic failure or cirrhosis appear.

**Diagnostic Tests**: Antibody to HCV is present in serum. Polymerase chain reaction (PCR) or other methods allow quantitative determination of viral DNA in serum. Liver function tests and liver biopsy to assess degree of hepatocellular damage.

**Course**: Progressive decline of hepatic function in chronic disease. Complications include hepatic cirrhosis (20%), hepatocellular carcinoma (5%), arthritis, glomerulonephritis, autoimmune syndromes.

**Treatment**: Interferon alfa, ribavirin. Liver transplantation.

### Hepatitis D (Delta Hepatitis)

Hepatitis due to a defective virus; it occurs only in persons already infected with hepatitis B, and is common among IV drug users. In itself a relatively mild illness, it may add to the severity of hepatitis B.

### Hepatic Cirrhosis (Portal Cirrhosis, Laënnec Cirrhosis)

A chronic disorder of the liver characterized by inflammation of secretory cells followed by nodular regeneration and fibrosis.

**Causes**: The principal cause is chronic alcohol abuse. About 20% of persons with hepatitis C eventually develop cirrhosis. Other toxic, metabolic, nutritional, and infectious factors may play a part in the genesis of this disorder. The cirrhotic liver contains various combinations of fatty change and fibrosis forming small and large nodules.

**History**: Usually gradual onset of anorexia, nausea, weakness, weight loss, abdominal swelling due to ascites (accumulation of fluid in the abdominal cavity), and often jaundice. Disturbance of sex steroid hormone metabolism causes impotence in men and amenorrhea in women.

**Physical Examination**: Fever, muscle wasting, pleural effusion, ascites, peripheral edema. The liver is usually enlarged and may be firm or even hard. The spleen may also be enlarged. Jaundice appears relatively late. Elevation of estrogen level causes gynecomastia in men, spider angiomas (spider nevi) on the face and upper trunk, and palmar erythema. The tongue may appear smooth, shiny, and inflamed. With advanced disease there may be coarse, flapping tremors (asterixis) and delirium due to hepatic failure, which may progress to hepatic coma.

**Diagnostic Tests**: Laboratory tests show elevation of bilirubin and enzymes such as transaminases, lactic dehydrogenase, and alkaline phosphatase, which rise in the presence of liver cell damage. Anemia may be present, and coagulation studies may yield abnormal results. Liver biopsy confirms presence of typical histologic changes. Imaging studies including radioactive liver scans provide further information. Esophagoscopy may show esophageal varices.

**Course**: Symptoms may wax and wane over a period of years, often in response to varying levels of alcohol consumption. Progressive hepatic failure often occurs. Fibrosis within the liver typically shuts off branches of the portal circulation and increases the pressure in the portal vein (portal hypertension). In consequence, other vessels (particularly the lower esophageal venous plexus) dilate and become varicose (bulging) or tortuous (coiled, twisted). Hemorrhage from bleeding esophageal varices is often life-threatening, particularly when hepatic disease causes a coagulation disorder. There is an increased incidence of hepatocellular carcinoma in persons with cirrhosis.

**Treatment**: Abstinence from alcohol, attention to nutrition, particularly carbohydrate, protein, vitamins. Rest, sodium restriction, and diuretics for edema and ascites. Severe ascites may require abdominal paracentesis (removal of peritoneal fluid with a needle passed through the abdominal wall). Patients with portal hypertension and bleeding esophageal varices may need a portacaval shunt (surgical procedure allowing portal vein blood to bypass the liver and empty directly into the inferior vena cava).

## DISEASES OF THE GALLBLADDER AND BILIARY TRACT

### Cholelithiasis (Gallstones)

The formation of gallstones is a common disorder, generally due to some disturbance in the flow of bile from the gallbladder or in the composition of bile. Gallstones are more common in women and in elderly persons. Risk factors include pregnancy, diabetes mellitus, high serum cholesterol, Crohn disease, and sickle cell anemia. In the latter condition, stones consist primarily of bilirubin from broken-down red blood cells. In the other conditions, gallstones are composed primarily of cholesterol.

Gallstones are often asymptomatic ("silent"), but about 90% of persons with cholecystitis (inflammation of the gallbladder) have preexisting cholelithiasis. Stones may be demonstrated on plain abdominal films, but ultrasound and imaging after injection of opaque medium are more sensitive and specific. Potential serious complications are blockage of the common bile duct by a stone with ensuing obstructive jaundice, blockage of the cystic duct with ensuing cholecystitis, and passage of a stone into the intestine with the potential for causing bowel obstruction (gallstone ileus). Treatment of symptomatic gallstones is surgical removal (along with the gallbladder), usually through a laparoscope. Stones can also be crushed and flushed out with instruments passed through an endoscope inserted through the mouth and threaded into the common bile duct. Oral bile salts (chenodeoxycholic acid, ursodeoxycholic acid) and extracorporeal shock-wave lithotripsy (ESWL) sometimes dissolve stones.

### Acute Cholecystitis

Acute inflammation of the gallbladder.

**Causes**: As mentioned above, most patients with cholecystitis have preexisting cholelithiasis. Impaction of a stone in the cystic duct leads to obstruction of the flow of bile from the gallbladder, with ischemia, acute inflammation, and sometimes abscess formation or perforation.

**History**: Fairly acute onset of severe epigastric and right upper quadrant pain, nausea, and vomiting.

**Physical Examination**: Fever and jaundice may be present. In the right upper quadrant of the abdomen, there are tenderness, rebound tenderness, and involuntary guarding (spasm of abdominal muscles on palpation). Bowel sounds are reduced or absent. Occasionally a mass can be felt below the liver edge, representing a distended gallbladder.

**Diagnostic Tests**: The white blood cell count, bilirubin, and levels of serum enzymes reflecting hepatic damage may all be elevated. Imaging studies (plain abdominal x-ray, ultrasound, scans with radiotagged media) may precisely identify the problem.

**Course**: Acute cholecystitis may resolve spontaneously. Often relapses occur, with gradual development of chronic cholecystitis. Inflammation may culminate in gangrene (tissue death due to compromise of blood supply) or perforation of the gallbladder, or may ascend into the liver via the biliary tract (ascending cholangitis).

**Treatment**: Chiefly supportive, with narcotics for pain, intravenous fluids, and close observation. Impending or actual perforation is treated by surgical (laparoscopic) decompression (drainage) of the gallbladder or, preferably, by removal of the gallbladder (cholecystectomy).

## DISORDERS OF THE PANCREAS

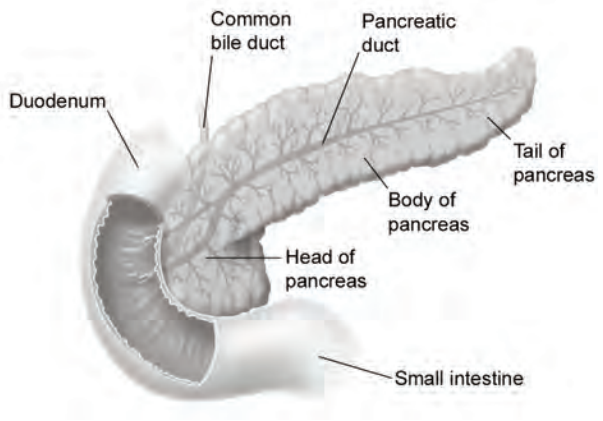
The pancreas is a flat retroperitoneal organ lying behind and below the stomach, with its right end (head) embraced by the C-shaped curve of the duodenum (Figure 42). It is composed of two types of glandular tissue: groups of cells that secrete enzymes for the digestion of carbohydrate, protein, and fat, which are poured through a duct into the duodenum near the orifice of the common bile duct; and other groups of cells that secrete hormones (insulin, glucagon, somatostatin) and release them directly into the bloodstream. The endocrine function of the pancreas is discussed in Chapter 15.

### Acute Pancreatitis

Acute inflammation of the pancreas (Figure 43).

**Causes**: Most cases occur in alcoholics or in persons with chronic biliary tract disease (cholelithiasis,

**Figure 42. Pancreas**

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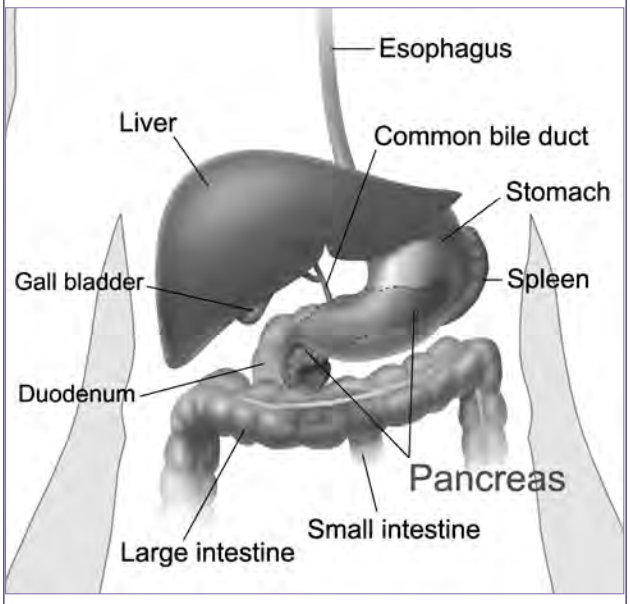
cholecystitis). In these instances, obstruction of the pancreatic duct by edema, or backflow of bile from the duodenum into the pancreatic duct, causes release of pancreatic enzymes into the substance of the gland, with resulting intense inflammation, necrosis, and often hemorrhage. Other causes are hypercalcemia (abnormally high level of calcium in the blood), hypertriglyceridemia (abnormally high level of triglycerides in the blood), abdominal trauma or surgery, certain medicines, and viral infection including mumps. An acute attack of pancreatitis is often precipitated by excessive alcohol consumption or by eating a large meal.

**Figure 43. Pancreas and Nearby Organs**

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**History**: Abrupt onset of severe, persisting epigastric pain, worse on lying flat, and radiating to the flanks and back. Nausea, vomiting, sweating, prostration, restlessness.

**Physical Examination**: Pallor, tachycardia, fever, epigastric tenderness, reduced or absent bowel sounds. Jaundice or hypotension may occur. In the presence of severe pancreatic hemorrhage, a bluish discoloration of the skin may appear over the left flank (Turner or Grey Turner sign). There may be evidence of ascites or a left pleural effusion.

**Diagnostic Tests**: Blood studies may show leukocytosis, hyperglycemia, anemia, and hypocalcemia (drop in serum calcium). Blood levels of pancreatic enzymes (amylase, lipase) are typically elevated. Imaging studies may show gallstones, a mass representing the swollen pancreas, left atelectasis (collapse of part of left lung caused by shallow breathing at site of pain), or left pleural effusion (inflammatory fluid in pleural cavity).

**Course**: Acute pancreatitis has a high mortality rate and, among survivors, a high recurrence rate. Possible outcomes include abscess formation, splenic vein thrombosis, ileus, shock, renal failure, adult respiratory distress syndrome, severe hypocalcemia with tetany, formation of pseudocysts (pockets of inflammatory fluid and debris between the pancreas and surrounding tissues), and progression to chronic disease.

**Treatment**: Hospitalization, narcotics for pain relief, nasogastric suction, intravenous fluids with attention to water balance, nutritional needs, and replacement of calcium. Surgery may be required to control hemorrhage, correct underlying disease, or drain pseudocysts.

## Chronic Pancreatitis

Chronic inflammation of the pancreas.

**Causes**: Essentially the same as for acute pancreatitis. With recurrent or chronic disease, fibrosis of the pancreas and its ducts leads to worsening disease, with loss of both endocrine and exocrine pancreatic function.

**History**: Recurrent bouts of left upper quadrant pain, anorexia, nausea, vomiting. Weight loss, flatulence, and steatorrhea (greasy stools) because of deficiency or absence of pancreatic enzymes.

**Physical Examination**: Epigastric and left upper quadrant tenderness, involuntary guarding, ileus.

**Diagnostic Tests**: Sugar may appear in the urine and blood sugar may be elevated as a result of diabetes mellitus due to destruction of pancreatic endocrine tissue. The serum levels of pancreatic amylase and lipase

may be elevated. The proportion of fat in the composition of the stools is increased because of deficiency of lipase in the intestine. Abdominal x-ray may show widening of the curve of the duodenum due to pancreatic edema. Retrograde injection of opaque medium into the pancreatic duct with a catheter placed endoscopically can clearly outline anatomic conditions (dilatation vs. narrowing of duct, stone formation).

**Course**: Without treatment, progressive deterioration of pancreatic function, with nutritional deficiencies, flatulence, steatorrhea, and diabetes mellitus. Pseudocyst formation.

**Treatment**: Avoidance of alcohol and fatty foods, oral supplementation of pancreatic enzymes. Surgery may be required to improve pancreatic drainage or remove pseudocysts.

## Chapter 11, Diseases of the Digestive System

## QUESTIONS FOR STUDY AND REVIEW

1. Name three gastrointestinal disorders that are due to infection:

   a.

   b.

   c.

2. Name three gastrointestinal disorders that may be precipitated or aggravated by alcohol abuse:

   a.

   b.

   c.

3. Name three gastrointestinal disorders that may be precipitated or aggravated by smoking:

   a.

   b.

   c.

4. Name three gastrointestinal disorders that can be complicated by life-threatening hemorrhage and result in intestinal obstruction:

   a.

   b.

   c.

5. Define or explain these terms:

   a. anorexia

   b. Crohn disease

c. dysphagia

d. flatulence

e. GERD

f. hematochezia

g. ileus

h. melena

i. pancreas

j. peritoneum

6. Name three kinds of gastroenteritis that must generally be treated with antibiotics:

   a.

   b.

   c.

7. Name three kinds of gastroenteritis that do not respond to antibiotics:

   a.

   b.

   c.

8. State three important differences between Crohn disease and ulcerative colitis:

   a.

   b.

   c.

9. State three important differences between hepatitis A and hepatitis B:

   a.

   b.

   c.

## CASE STUDY: YOU'RE THE DOCTOR

Ezra Toldt, a 57-year-old homeless white male, is brought by an ambulance crew to the hospital emergency department where you are on duty. He was found by custodial workers around 10 p.m. in a public park vomiting, confused, and "doubled over with pain." He has no known address and no known family. Past medical history and information about insurance coverage are unavailable.

1. What types of health problems does this background suggest?

Mr. Toldt is unkempt, unshaven, and unwashed. His clothing smells of urine and vomitus, and in addition there is a strong smell of liquor on his breath. He is moaning and holding his abdomen. He cries out in pain when rescue personnel transfer him to a gurney. He responds to some commands but not others. He answers questions incoherently or not at all.

2. To what extent do you expect his inability to give a clear history to impair your ability to diagnose and treat him appropriately? What reasons support your position?

Rectal temperature is 100.8, pulse 116, blood pressure 114/54. The skin is sallow, warm, damp, and of reduced turgor. Bruises and abrasions of various ages are noted over the chest and extremities. There is no evidence of head trauma. Examination of the eyes shows conjunctival injection, scleral icterus, and lateral nystagmus. The pupils are equal and sluggishly reactive. The pharyngeal mucosa appears dry and beefy red. The teeth are in poor repair. There are no cervical masses. The ribs are not tender. The heart is regular without murmurs, rubs, or third or fourth heart sound. The lungs are clear. The abdomen is slightly distended and shows no scars. Palpation of the abdomen reveals marked guarding, tenderness, and rebound tenderness in all four quadrants. A firm, smooth liver edge is palpated four

fingerbreadths below the right costal margin. The spleen is not palpable. No abdominal masses are felt. No bowel sounds are heard. Rectal examination is performed with difficulty because of the patient's pain and confusion. The prostate is symmetrically enlarged, slightly boggy, and apparently slightly tender. The rectal pouch contains soft dark stool that is positive for occult blood.

3. What diagnostic possibilities do these findings raise?

4. What further procedures will you use to narrow the diagnosis?

5. What forms of treatment are indicated immediately while you proceed with your evaluation?

## SUGGESTIONS FOR ADDITIONAL LEARNING ACTIVITIES

1. Write a history and physical examination report for a patient admitted to the hospital with a digestive disorder discussed in this chapter. Include a chief complaint, history of present illness, past medical history, family and social histories, review of systems, and complete physical examination. Describe the laboratory data ordered and obtained upon admission. Your differential diagnosis should include other possible conditions that need to be ruled out as the cause of the patient's problem. Draft a treatment plan. For an additional challenge, remove the diagnosis and treatment plan sections from your H&P and exchange reports with another student. Write a differential diagnosis and treatment plan for this new report and then compare your conclusions.

2. Make a 3-D (three-dimensional) model of the digestive system using modeling clay or any household objects (cardboard paper towel holders, small boxes, and so on). Assemble your model and label the individual structures, including explanations of the role of each structure in the function of the digestive system.

3. Medications are commonly prescribed for the treatment of gastrointestinal conditions. Compile a list of all the types of medications described in this chapter. Consult a drug reference book and locate several brand names for each type of drug mentioned. Write down the indications, usual dosage, and form for each drug.