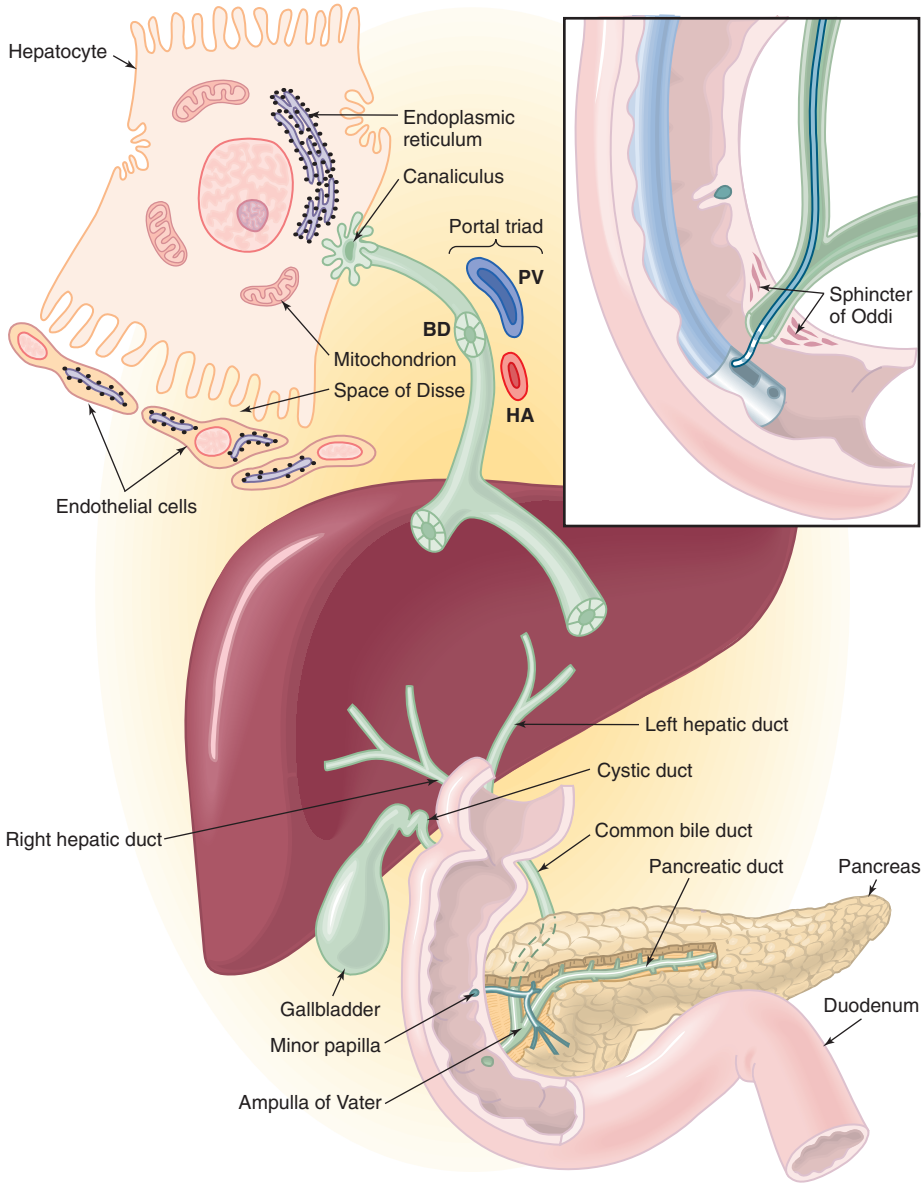

FIGURE 44-1 Normal anatomy and histology of the liver and biliary tract. Materials destined for metabolism or excretion by the liver (such as unconjugated bilirubin) enter the sinusoidal bed and cross the endothelial barrier and the space of Disse. Unconjugated bilirubin is taken up by the hepatocyte, conjugated with glucuronide to become water soluble, and excreted into bile across the canalicular membrane of the hepatocyte. The canaliculi empty into bile ductules (BD), which lead to the interlobular (small), septal (medium), and large intrahepatic bile ducts and finally to the main branches of the common bile duct. The portal areas, or portal triads, are composed mainly of portal vein (PV), hepatic artery (HA), and BD branches. During fasting, tonic contraction of the sphincter of Oddi, located in the region of the ampulla of Vater, diverts about one half of the bile through the cystic duct into the gallbladder, where it is stored and concentrated to be released later during meal times. Disease at any level of the biliary tree can lead to cholestasis and obstructive jaundice.

1. Biliary cholesterol saturation is increased by estrogens, multiparity, oral contraceptives, obesity, rapid weight loss, and terminal ileal disease, which decreases the bile acid pool.
2. Nucleation is enhanced by biliary parasites, recurrent bacterial infection of the biliary tract, and antibiotics such as ceftriaxone, which has a proclivity to concentrate and crystallize with calcium in the biliary tree. Total parenteral nutrition and blood transfusions also promote bile pigment accumulation and *gelfaction* of sludge.
3. Bile stasis is caused by gallbladder hypomotility (resulting from pregnancy, somatostatin, or fasting), bile duct strictures, choledochal cysts, biliary parasites, and total parenteral nutrition.

Clinical Manifestations of Gallstones

Gallstones develop at some point in 10% to 20% of Americans. Between 50% and 60% of these individuals remain asymptomatic, but about one third develop biliary colic or chronic cholecystitis, and 15% develop acute complications. The natural history of gallstone disease is outlined in Figure 44-2. Obstruction of the biliary tract at any level by stones or sludge is the underlying cause of the clinical manifestations of gallstone disease. Obstruction by gallstones can occur at the level of the