

Structure of the biliary tract

Gallbladder

- Pear shaped sac
- Holds up to 100 to 150 cc of bile (usually only 10 to 50 cc)
- Smooth muscle contracts to eject the bile
- Receives arterial blood from the cystic artery
- Venous blood drains via cystic veins
- Bile is only dispatched if fat enters the duodenum.
- GB receives bile from the liver via Right and Left Hepatic Ducts
- Bile travels from GB via cystic duct to common bile duct to small intestine

Bile

- provides digestion of fat through emulsification
- bile salts must be present or stones will develop
- **Sphincter of Oddi** is stimulated by a secretion that relaxes the sphincter allowing bile release

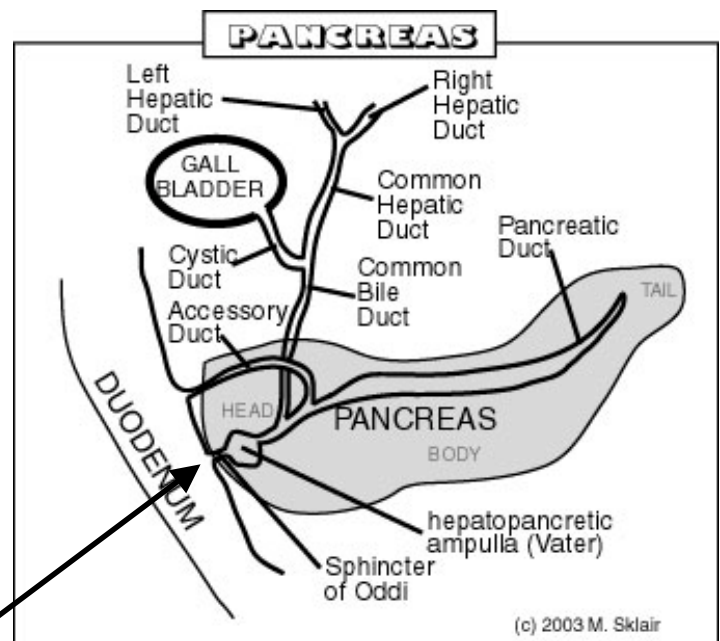
Structure of Pancreas

- large , many lobed
- posterior to stomach
- the aorta, inferior vena cava and hepatic artery are near the head of the pancreas
- endocrine=insulin/glucagon secretion-
1% of pancreatic cells do this work
- exocrine
 - digestive enzymes to duodenum
 - produced by acini cells-
99% of pancreatic cells do this work

Pancreatic duct joins the common bile duct as ampulla of Vater- opens into an area called duodenum papilla through Sphincter of Oddi

Even after cholecystectomy (removal of gallbladder) common bile duct can work to store bile in absence of GB.

Duodenal papilla help remove stones



Function of the biliary tract

Gallbladder

- stores and concentrated bile
- when GB is removed → ducts act as reservoirs
 - however can no longer excrete large quantities of bile after a fatty meal
- **cholesterol** must be present for bile salts
- Bile produced in liver consists of a very delicate balance:
 - Water
 - Bile salts
 - Cholesterol
 - Electrolytes
 - Phospholipid –Lecithin
 - Bilirubin (bile pigment) = yellow-olive green, brown

Function of Exocrine Pancreas

Excretion

- Produces 1200-3000 cc juice per day
 - ❑ Amylase – splits CHO's
 - ❑ Lipase – hydrolyzes fat
 - ❑ Trypsin – split proteins
 - Stored as trypsinogen
 - Trypsin inhibitor – prevents autodigestion
 - *Autodigestion is a common cause of pancreatitis*
 - Activated by enzymes secreted from the duodenum wall
- *Composition : water , some salts , HCO₃ , enzymes -> has alkaline base to act on HCl released from stomach*

Assessment of Biliary Disorders

History and Physical and Diagnostic Tests

- **Assessment : History**
 - Presenting signs and symptoms
 - *S/S may be similar to GERD, colycystitis , hepatitis*
 - If acute:
 - *When did it happen?*
 - *What were you doing?*
 - *Was your meal fatty?*
 - Often presents in the ED with pain or altered digestion
 - recent trauma
 - lifestyle pattern
 - *travel – “have you been out of the country lately” → could be an indicator of hepatitis*
 - history of illness (GB, colycystitis, blood transfusions)
 - meds, ETOH, tylenol, steroids)
 - diet
- **Chief Complaint**
 - ❑ Abd. Pain, anorexia
 - ❑ Fatty food intolerance
 - ❑ Melena./ clay- colored / steatorrhea (foamy, fatty) stools
 - *Bilirubin travels down GI tract and gives color to stools*
 - *With blockage of the common bile duct, clay-colored stools result*
 - ❑ GU : tea colored , dark urine
 - ❑ Derm: jaundiced skin, sclera (if dark-skinned patient: look at hard palate – should be pink if okay.
 - ❑ Edemateous , bruises

Gallbladder emptying on a regular basis keeps the organ healthy

- **Diagnostic Tests: Biliary**
 - ❑ Cholangiography – utilizes dye
 - ❑ ECRP - diagnostic and used for treatment (see below)
 - ❑ Angiography; Labs
 - ❑ Ultrasound – leads the pack as Preferred Test since it can be done without contrast dyes
 - ❑ CT Scan

Biliary Tract Disorders

Cholelithiasis

- **Etiology / Pathology**
 - Cause Unknown
 - *Related to conditions that upset the balance of cholesterol, bile salts and calcium IN the BILE -> leads to precipitation*
 - Precipitates into stones that are comprised of cholesterol, calcium, bile salts with bilirubin and protein
 - Often mixed in concentration : some cholesterol stones, some calcium stones
 - Stasis of bile – supersaturation with cholesterol → chemical changes in composition of bile
 - **Supersaturation:** *as it becomes concentrated, it should remain balanced in composition → if not → stones form!*

Precipitating Factors

- Pregnancy : hormonal factors delay emptying
- Immobility
- Inflammation: blocks bile ducts → stasis → supersaturation of cholesterol
- Obstruction of bile ducts (such as lesions)
- Parathyroidism: leads to hypercalcemia → stasis → stones

Gallstones

- **Types**
 - **Cholesterol** stones: smooth , white
 - **Pigment** stones: excess unconjugated bilirubin
 - Types of Pigment Stones
 - **Black:** *associated with cirrhosis and hemolysis*
 - **Earthy:** *calcium (associated with infections of biliary system)*
 - **Mixed** stones: combo of cholesterol and pigment stones

Cholelithiasis : Signs and Symptoms

- **Acute**
 - Severe excruciating spasms may be present or absent
 - Spasms may occur as stones move **3 to 6** hours after a **fatty meal**
 - Spasms may last up to an hour
 - **RUQ soreness may remain**
- **Chronic**
 - RUQ pain
 - Epigastric fullness
 - Colic – spasms of the duct
 - Nausea and vomiting
 - **Jaundice ONLY WITH OBSTRUCTION** (bilirubin doesn't enter intestine, blood levels rise)
 - Vitamin Deficiency (A,D,E,K)
 - K- normal blood clotting if obstruction is prolonged

Cholelithiasis : Diagnosis

- **Ultrasound**
 - Preferred procedure because dye is not required
 - If patient is jaundiced, they won't be able to excrete dye that is required with:
- **Cholangiography**
- **CT Scan**

Cholelithiasis : Treatment

- **Low Fat Diet**
- **Analgesics**
 - Avoid MORPHINE – Use Demerol
- **NG Tube / IV Fluids**
- **Oral Cholesterol Lowering Drugs**
 - **Side Effects :**
 - ❑ diarrhea
 - ❑ hepatotoxicity
- **UDCA**
 - ❑ Ursodeoxycholic acid – dissolves stones
 - ❑ No diarrhea
- **MTBE**
 - ❑ Infused into the gallbladder percutaneously or through a T-Tube
- **ERCP**
 - ❑ Used as a precursor to laparoscopic cholecystectomy
 - ❑ Reduces size of GB for removal through umbilicus
 - ❑ Sphincter of Oddi and papilla may have to be cut
 - ❑ Complication: damage to pancreas
- **Lithotripsy**
 - ❑ Performed in warm tub of water
- **Laser Pulse Technology**

If GB is not removed, stones are likely to return

Cholecystitis: Acute

- **Etiology/Pathology**
 - ❑ 90% gallstones
 - ❑ pus-filled obstructs bile flow out of GB
 - ❑ Vascular supply is compressed → gangrene
 - ❑ Autolysis can occur when excretions are activated → bacteria results
 - ❑ ACALCULUS – when no stones are present → cholecystitis is a result of severe trauma , or burns cause a lack of blood supply and subsequent electrolyte imbalance, torsion of veins , multiple blood transfusions, bile stasis and sludge formation.
- **Signs and Symptoms**
 - ❑ Similar to cholelithiasis
 - ❑ RUQ or epigastric pain
 - ❑ N/V
 - ❑ Signs of Inflammation

Cholecystitis: Surgical Treatment

- **Lap-Chole** (laparoscopic cholecystectomy)
 - ❑ Stones in common bile duct treated with ERCP then lap-chole performed
 - ❑ Performed through umbilicus – abdomen infused with CO2
 - ❑ Fiberoptic scope used to visualize, bile and stones are aspirated, GB is dissected and removed.

Cholecystitis: Surgical Treatment *(continued)*

- **Lap-Chole : Post -Op**
 - ❑ Few Complications r/t minor incisions
 - ❑ Low risk of pneumonia or atelectasis → short anesthesia
 - ❑ **24 hrs to 2 day** hospital stay
 - ❑ TCDB – *turn , cough and deep breath*
 - ❑ **Referred Pain in Right scapula** r/t CO2 administered during surgery
 - *treat with walking , heating pad and sitting up in bed*
 - ❑ teach for infection, loss of appetite and possible vomiting
 - *infection can occur r/t instrumentation of the area (watch for raised temp, N/V)*

- **Cholecystectomy**
 - ❑ Condition more extensive or acutely severe inflammation
 - ❑ **4 to 5 day** hospital stay - T-Tube remains in place for 7 to 14 days
 - ❑ Patient is NPO
 - ❑ Abdominal incision : T-Tube [drains to gravity] , NG Tube, Ivs
 - ❑ Analgesics
 - ❑ TCDB, Incentive Spirometer, Ambulate
 - ❑ Dressing Changes
 - ❑ Measure Drainage...
 - ❑ Be ALERT for **stark blood , excessive drainage s/p 24 hours**
 - ❑ High Tendebncy for Abdominal Distention and Vomiting
 - ❑ Peristalsis is SLOW to RETURN → listen for Bowel Sounds → Risk of Ileus

Pancreatic Disorders

Acute Pancreatitis

- Mild to severe
- Mild edema and inflammation of the pancreas
- Minimal organ dysfunction → return to normal in 6 months
- Mortality increases with age
 - Hypovolemic Shock (protein rich fluid forced into tissues and peritoneum)
 - ➔ *The fluid is moving into tissues NOT the intestines*
 - Sepsis
 - Fluid and Electrolyte disturbances
 - Autodigestion: Trypsin that is converted from trypsinogen WITHIN the bile ducts instead of in duodenum → tissue destruction occurs and fluids seep into peritoneum
 - Gallstones st Ampulla of Vater obstruct flow
 - **70% of cases are related to chronic alcoholism**
 - Bacteria / viral mumps * , blunt trauma, hyperlipidemia (“fatty pancreas”), hypercalcemia, use of thiazide diuretics, steroids, oral contraceptives
 - * CMV – cytomegalovirus – associated with HIV

Acute Pancreatitis- Signs and Symptoms

- Abdominal pain—sometimes excruciating
- Tenderness in mid epigastric area
- Acute Onset: 24 to 48 hours post meal or ETOH ingestion (*huge binge , not minor ETOH consumption*)
- Unrelieved by antacids
- Decreased peristalsis (r/t loss of blood flow to organs)
- Abdominal Distention, Abd, guarding
- Rigid or Board-like abdomen r/t peritonitis

Acute Pancreatitis- Signs and Symptoms (continued)

- Ecchymosis (bluish hue in flank or around umbilicus) – r/t pancreatic bleeding into tissue
- Acute RENAL FAILURE common
- **Respiratory Distress** and Hypoxia – r/t pulmonary infiltrates that move into lungs → hypovolemic shock
- Abnormal Blood Gases
- Hypocalcemia, hyperglycemia, DIC – *disseminated intravascular coagulation* (abnormal coagulation)

Turner's Sign
Bluish hue in **flank**

Cullen's Sign
Bluish hue around **umbilicus**

Individuals with 5 to 6 of these symptoms have a higher mortality rate

Acute Pancreatitis- Diagnosis

- Serum Amylase and Lipase in excess of **3 times normal** values
 - ⇒ **Amylase** (enzyme that hydrolyzes starch) up to 48 to 72 hours
 - ⇒ **Lipase** (enzyme that hydrolyzes fats) up to 14 days
- Urinary amylase (spills over from blood into urine)
- WBCs Elevated (15-16k) and Glucose **HIGH**
- Serum Calcium LOW
- Fatty Stools
- No ERCP (Pt is too sick, hypovolemic shock must be resolved first before looking for stones)
- **ULTRASOUND** and X-RAY of Abdomen and Chest →

Acute Pancreatitis- Treatment

- Relieve symptoms
 - Analgesics - DEMEROL (not morphine which can cause biliary spasms)
 - Anti-emetics
- **Digestive**
 - NPO → Total Parenteral Nutrition is a must
 - NG tube suction to reduce gastrin release from stomach – keep gastric contents from entering duodenum
 - H2 antagonists → help quiet down digestive systems
- **Hematologic**
 - IV's with NS and albumin to raise BP
 - Hemodynamic monitoring – catheter in heart
 - Antibiotics for infection
- **Respiratory**
 - O2 aggressive therapy
- **Biliary Drainage**
 - **Lavage**
 - To drain all debris to gravity – usually 3 or 4
 - Monitor heart pressures r/t hemovolemia
- **Surgery**
 - For necrotic pancreas

Acute Pancreatitis- Post Acute Care

<ul style="list-style-type: none"> ▪ change lifestyle ▪ no caffeine or ETOH 	<ul style="list-style-type: none"> ▪ eliminate offending medications 	<ul style="list-style-type: none"> ▪ low fat, low protein diet ▪ follow-up testing and care
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Chronic Pancreatitis

- progressive destruction
- cells replaced with fibrous tissue with repeated attacks
- pressure in the pancreas increases with obstruction of pancreatic and common bile ducts leading to duodenum
- continued ETOH use → hypersecretion of protein in pancreas → protein and calculi plugs form in pancreatic ducts
- ETOH also has direct toxic effect on cells

Chronic Pancreatitis – Signs and Symptoms

- attacks of upper abdominal pain and back pain
- vomiting
- Opioids don't always help...pain increasingly severe and more nagging...can create dependence
- Weight Loss and Anorexia
- Malabsorption and Steatorrhea
- Calcification of the pancreas

Chronic Pancreatitis – Diagnosis

- ERCP
- Tissue Analysis
- CT Scan
- US
- Labs

Chronic Pancreatitis – Treatment

- Attend to symptoms
- Surgical
 - If GB is diseased as well → remove
 - **Pancreaticjejunostomy** – joining the pancreatic duct to jejunum to allow for drainage of pancreatic secretions into the jejunum (pain relief, but often recurs with the disease progression)
 - **Whipple** – pancreaticoduodenectomy to relieve pain
 - **Autotransplantation** – for total pancreatectomy

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