GALLSTONE DISEASE
TOPICS

- Some physiology
- Biliary Colic
- Acute cholecystitis
- Chronic cholecystitis
- Cholecystectomy
- Cholangitis
- Obstructive jaundice
- Common bile duct exploration & ERCP
Bile Formation

- Continuous formation by the liver
- Bile flow directly to gallbladder with intact Sphincter of Oddi – bile concentration & storage
- Greatest absorptive power per unit area of any structure in the body – permits low pressure state in biliary tree
- Bile composition
  - Water, electrolytes, lipids (cholesterol & phospholipids from liver), bile salts (from cholesterol), proteins, bile pigments
- 500-1000mL produced per day
- Enterohepatic circulation – 80% of conjugated bile acids absorbed in terminal ileum
**Bile Secretion**

- 80% of secreted bile stored in gallbladder in fasting state
- Fasting phase: motilin stimulates
- Neurogenic, humoral, chemical stimuli
  - Vagal stimulation
  - Secretin from duodenum (released in response to partially digested fatty acids & proteins)
- Gallbladder secretes:
  - Glycoproteins – protection of mucosal
  - Hydrogen ions – lowers pH to increase Ca solubility
Bile Secretion cont...

- Neurohormonal control of contraction: Gastric distension; Vagal stimulation; Peptides
- CCK
  - Released when stimulated by acid, fat & amino acids into duodenum
  - from epithelial cells of upper GI tract directly causes:
    - gallbladder contraction
    - relaxation of distal CBD & sphincter of Oddi
STONE FORMATION

- Very simply – solute settling out of solution
- Main solutes in bile:
  - Bile salts, bilirubin, phospholipids, cholesterol
- Cholesterol stones (~80%)
  - Usually ~70% cholesterol by weight
- Pigment stones (~20%)
  - Black (~15-20%)
  - Brown (<5%)
**Cholesterol Stones**

- Most are radiolucent (hence decreased utility of X-rays in diagnosis)
- Solubility related to relative concentrations of cholesterol, bile salts & lecithin (main phospholipid)
- Formed from supersaturation of cholesterol in bile, mostly in gallbladder
- Supersaturation mostly due to cholesterol hypersecretion (vs low phospholipids or bile salts)
BLACK PIGMENT STONES

- Supersaturation of calcium bilirubinate, carbonate, and phosphate
- Small, brittle black stones, mostly in gallbladder
- Deconjugation of bilirubin normally at slow rate
- Unconjugated bilirubin much less soluble than conjugated
- If large amounts of conjugated bilirubin, have increased production of deconjugated bilirubin
- Hemolytic disorders (hereditary spherocytosis, sickle cell disease), cirrhosis
**Brown Pigment Stones**

- <1 cm in diameter, brownish-yellow, soft, often mushy
- Formed in gallbladder as well as bile ducts – due to stasis
- Stones mainly composed of precipitated calcium bilirubinate and bacteria
- Some bacteria (ex *E. coli*) secrete beta-glucuronidase – cleaves bilirubin glucuronide to produce insoluble unconjugated bilirubin
- Precipitates with calcium and bacteria – forms brown stones
- Asian populations: associated with stasis secondary to parasite infection
- Western population: primary bile duct stones in patients with
  - Biliary strictures
  - Other CBD stones that cause stasis and bacterial contamination
# Types of Stones

<table>
<thead>
<tr>
<th></th>
<th>Cholesterol</th>
<th>Brown Pigment</th>
<th>Black Pigment</th>
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<tbody>
<tr>
<td><strong>Prevalence</strong></td>
<td>80-90%</td>
<td>5-10%</td>
<td>&lt;5%</td>
</tr>
<tr>
<td><strong>Main composition</strong></td>
<td>50-90% cholesterol</td>
<td>~50% bilirubin</td>
<td>&gt;50% bilirubin</td>
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<tr>
<td><strong>Color</strong></td>
<td>Yellow-grey</td>
<td>Brown</td>
<td>Dark brown-black</td>
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<tr>
<td><strong>Etiology</strong></td>
<td>Cholesterol supersaturation</td>
<td>Increased deconjugation of bilirubin glucuronides</td>
<td>Increased biliary bilirubin load</td>
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<tr>
<td><strong>Risk Factors</strong></td>
<td>Increasing age, Female gender, Family history, Obesity</td>
<td>Biliary infections, Abnormal biliary anatomy</td>
<td>Liver cirrhosis, Ineffecient erythropoiesis, Hemolytic disorders</td>
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Dietary factors
Dyslipidemia
Hormonal factors
Life style factors
• Normal gallbladder
  - wall in thin, hypoechoic
  - may see “pseudothickening” in postprandial state
CASE 1

- 38 female presents to office with recurrent history of epigastric & RUQ pain, consistently post-prandial, although not after each meal; sometimes radiates to the back
- Associated with nausea & vomiting
- In the past went to walk-in clinic: blood tests have not shown any abnormalities
- Has occasionally taken Abx but is now tired of the pain and would like definitive treatment
**Biliary Colic**

- Definition – pain related to cystic duct obstruction - very variable: classically RUQ, aching, post-prandial pain lastly ~ 3 hours
- Approximately 3% of people with gallstones will become symptomatic per year
- Once symptomatic, tend to have recurring bouts
- Complicated gallstone disease occurs in ~3% of symptomatic patients per year
- I.e. Not common to experience complications from gallstone disease if not already symptomatic
Clinical Etiology of Biliary Colic

- Neurohormonal control of contraction: Gastric distension; Vagal stimulation; Peptides
- CCK – from epithelial cells of upper GI tract – directly causes gallbladder contraction + relaxation of sphincter of Oddi
- Contraction against obstruction
- Gallbladder ischemia from prolonged contraction
- Length classically coincides with length of time of gastric emptying – ie ~3 hours
DIAGNOSIS

- History
  - Classic history vs. not – large range of clinical presentations

- Physical
  - Usually normal between bouts
  - Epigastric and/or RUQ pain during bouts

- Labs
  - CBC, ALT, AST, ALP, GGT, Bilirubin
  - Labs usually normal – no infection; no obstruction

- Imaging
  - U/S, ERCP, EU/S, MRCP, CT, PTC
**Imaging Studies**

- Ultrasound
  - Shows stones in the gallbladder: sensitivity & specificity of >90%
  - (stones acoustically dense & show acoustic shadow)

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TREATMENT

- Symptomatic relief
  - Analgesia & antiemetics

- Definitive treatment
  - Laparoscopic cholecystectomy
  - Avoid large meals and fatty foods while waiting for surgery
  - Diabetic patients – more prone to serious complications
  - Pregnant patients – can safely undergo surgery if conservative management unsuccessful – open if 3rd trimester
  - ~90% have relief of symptoms post-op – more successful for patients with stones on U/S & typical symptoms
Case 2

- 48 male presents in ED with ongoing severe RUQ pain, radiating to the back
- Sharp RUQ pain going on for several hours, started after dinner out with his wife
- Wife documented fever of 38.7°C at home
- Arrives at ED – nausea, vomiting x 4, looks unwell
- Labs: WBC is elevated, mild ASL & AST elevation, normal bilirubin
**Acute Cholecystitis**

- **Definition** – Acute gallbladder obstruction (longer than that which leads to biliary colic) that leads to gallbladder distension, inflammation & edema
- **Usually inflammation resolves once obstruction resolves**
- **May however, lead to ischemia and necrosis of gallbladder wall & gallbladder perforation (3-15% of people with acute cholecystitis)**
- **Although obstruction is due is gallstones in ~95% of patients, could be acalculous, or due to tumour obstructing cystic duct**
- **Initial inflammatory process potentially mediated by lysolecithin, bile salts and platelet-activating factor**
- **Bacterial infection in about 50% of cases – difficult to predict who will become secondarily infected (E. coli, Klebsiella, Enterococcus, Enterobacter)**
- **Abscess/empyema forms within gallbladder – may see perforation with cholecystoenteric fistula formation**
**Diagnosis - History**

- Constant, dull epigastric and/or RUQ pain & tenderness; may radiate to back and between scapulae
- Febrile, anorexia, nausea, vomiting
- May see more subtle presentation in diabetic and elderly – also higher mortality in these populations
- Often history of self-resolving biliary colic with reports that this pain is more severe than “usual” colic
DIAGNOSIS – PHYSICAL & INVESTIGATIONS

- **General**
  - Appears unwell & uncomfortable
  - Reluctant to move – parietal peritoneum irritation
  - Vitals coincide with severity of illness: tachycardia, fever

- **Abdominal exam**
  - RUQ tenderness, +ve Murphy’s sign
  - Guarding
  - May have palpable gallbladder

- **Labs**
  - CBC (normal to moderate WBC – 12-15,000 cells/mm³) – higher WBC counts could mean more complicated disease (gangrenous cholecystitis, cholangitis)
  - ALT, AST – may see mild elevation in transaminases
  - ALP, GGT, Bilirubin – may be elevated such as with Mirizzi’s syndrome

- **Imaging**
  - U/S, ERCP, EU/S, MRCP, CT, PTC
IMAGING

- Ultrasound
  - >95% sensitivity & specificity: stones, inflammation, pericholecystic fluid
  - Thickened & edematous gallbladder wall; edema b/w gallbladder and liver
  - Sonographic Murphy’s sign

- HIDA Scan
  - May be useful in atypical cases – ex acalculous (80% sensitivity, 90% specificity)
  - Inject HIDA IV – cleared by Kupffer cells in liver & excreted into bile
  - Uptake in liver @ 10mins then biliary system @ 60mins
  - Acute cholecystitis: nonvisualized gallbladder with filling of CBD (sensitivity & specificity ~95% with clinical suspicion of acute cholecystitis)
TREATMENT

- **Resuscitation**
  - IV fluids
  - Analgesia – narcotics
  - Antibiotics – typically here see Cipro (gram-negatives) + Flagyl (anaerobes)

- **Cholecystostomy**
  - Goal to drain distended, inflamed gallbladder in those not suited for surgery/unable to tolerate surgery
  - Future cholecystectomy once recovered and still indicated

- **Laparoscopic cholecystectomy**
  - within 3 days (preferred over interval surgery after recovery with medical management)
SURGICAL ISSUES

General surgical issues
- Tolerance of pneumoperitoneum (cardiorespiratory disease)
- Body habitus
- Previous surgeries – adhesions, anatomy

Cholecystectomy-specific
- Previous history of cholecystitis – may make the surgery more difficult
- Predictors of converting to open: shrunken gallbladder, thickened gallbladder wall, pericholecystic fluid
- Patients at risk for CBD stones – may require pre-op or intra-op duct imaging
- Preoperative planning with imaging: ERCP, EUS, MRCP
SOME HISTORY

- First cholecystectomy – 1882 by Dr. Carl Langenbuch
- Laparoscopic cholecystectomy – 1987 by Dr. Phillipe Mouret
- Previous relative contraindications:
  - Acute cholecystitis, gangrene and empyema of the gallbladder, biliary-enteric fistulae, obesity, pregnancy, ventriculoperitoneal shunt, cirrhosis, and previous upper abdominal procedures
  - These are now identified as risk factors for potentially difficult laparoscopic cholecystectomy
Randomized trial of early *versus delayed laparoscopic cholecystectomy* for acute cholecystitis


- Previous studies had shown that early open cholecystectomy had shorter hospital stay (with same morbidity & mortality as delayed), but no such trial for laparoscopic technique in treatment of acute cholecystitis

- Randomized trail:
  - Early laparoscopic cholecystectomy – within 24 hours of randomization
  - Conservative management (fluids + Abx – amicillin, cefuroxmine, metronidazole) followed by elective laparoscopic cholecystectomy 6-8 weeks later
RESULTS

- 104 patients randomized:
  - 53 early
  - 51 delayed
- Same conversion rate
- No CBD injuries in either group
- Significantly longer operating time for early group
- Significantly shorter hospital stay in delayed group

- Now recommend early cholecystectomy
**Post Lap Chole**

- Difficult case – anatomy initially completely obscured
- Day-care surgery – patient discharged home with some mild-moderate abdominal pain
- Presents back to hospital 3 days post-op with increasing RUQ pain
- What do you do?
Hx – worsening pain since surgery; today low grade fever at home; nausea

Px - ?jaundice (difficult to tell); looks stable but unwell; RUQ tender

Labs – WBC 11; Hg 110; Bili 31; AST 120

Now what?
**Ultrasound**

- Biliary collection – how do you approach this?
- Drain/aspiration
  - If bilious – leave drain
  - If hematoma – no need to leave drain
  - If abscess – leave drain
- If bilious – go on to ERCP to assess to accessory duct vs CBD injury
Case 3

- 34 female, teacher
- Recurrent attacks of biliary colic
- Always subside within 24 hours
- Associated with nausea & vomiting
- Has been going on for months
- Has been to FP several times – normal LFTs, no episodes of jaundice
- Would like definitive treatment
**Chronic Cholecystitis**

- **Definition** – Ongoing inflammation with recurrent episodes of biliary colic
- About 2/3 of patients present in this way
- Various pathological changes to gallbladder – including scarring & non-functional gallbladder
- Histologically – subepithelial & subserosal fibrosis + inflammatory cell infiltration
DIAGNOSIS

- History
  - Critical in diagnosis

- Physical
  - Same is biliary colic: normal-appearing gallbladder and/or epigastric pain during attack

- Labs
  - CBC, LFTs

- Imaging
  - Ultrasound: contracted, thick-walled gallbladder
TREATMENT

- Symptomatic management
  - Analgesia & antiemetics
- Elective cholecystectomy
  - Same recommendations when waiting for surgery as biliary colic
Cholecystectomy

**Symptomatic**
- Stones
  * Biliary colic
  * Acute cholecystitis
- No stones
  * Sludge on 2 or more occasions on U/S

**Asymptomatic**
- Stones
  * >2cm diameter
  * Calcified gallbladder
- No stones
  * liver transplant
# Cholecystectomy

## Indications

| Urgent (w/i 24-72 hours) | - Acute cholecystitis  
|                          | - Emphysematous cholecystitis  
|                          | - Empyema of the gallbladder  
|                          | - Perforation of the gallbladder  
|                          | - Previous choledocholithiasis with endoscopic duct clearance  
| Elective                 | - Biliary dyskinesia  
|                          | - Chronic cholecystitis  
|                          | - Symptomatic cholelithiasis |

## Contraindications

- Coagulopathy
- End-stage liver disease
- Severe COPD or CHF (CO <20%)
Cholecystectomy - Complications

- General surgical complications
  - Bleeding
  - Infection – prophylactic Abx
  - General anesthetic

- Laparoscopic complications
  - Tolerance of pneumoperitoneum

- Cholecystectomy-specific complications
  - Conversion to open: approximately 5%
  - Injury to:
    - Vascular structures
    - Bowel
    - Bile duct
ACUTE ACALCULOUS CHOLECYSTITIS

- Accounts for 5-10% of acute cholecystitis presentations & 1-2% of cholecystectomy cases
- Presents in same was clinically as acute calculous cholecystitis
- However, has more fulminant course & more likely to proceed to:
  - Gangrene
  - Empyema
  - Perforation
- More frequently in:
  - Elderly
  - Critically ill patients after trauma, burns, long-term TPN, major operations such as abdominal aneurysm repair and cardiopulmonary bypass
- Unclear etiology – gallbladder stasis and ischemia have been implicated as causative factors
DIANGOSIS

- History – Same as calculous cholecystitis
- Physical – Fever; abdominal pain, but not necessarily always RUQ tender
- Labs – May see increased leukocytes, increased amylase
- Imaging – Ultrasound shows same gallbladder changes as in calculous cholecystitis, but absence of gallstones
TREATMENT

- Resuscitation
  - Fluids
  - Analgesia
  - Antibiotics

- Surgery
  - Not usually the first option because usually secondary to underlying severe medical illness

- Morbidity rate around 40% because patients often initially critically ill

- If unfit for surgery, percutaneous, US or CT-guided cholecystostomy
  - If diagnosis uncertain, percutaneous cholecystostomy can be both diagnostic and therapeutic - about 90% of patients improve
CASE 5

- 56 year old male
- 12 hour history of severe RUQ and epigastric pain, fever & chills
- Wife mentions that he looks more yellow today than normal
- On PMHx, mentions previous history of biliary colic
**CHOLANGITIS**

- **Definition** – Inflammation of the biliary tree, with ascending bacterial infection associated with partial or complete bile duct obstruction (ie need the obstruction as well as bacterial infection for cholangitis)

- One of major complications of gallstone disease (other is gallstone pancreatitis)

- Bile normally maintains sterility by constant movement through biliary tree (as well as immunoglobulins)

- Most common bacteria: *E. coli, Klebsiella pneumoniae, Streptococcus faecalis, and Bacteroides fragilis*

- Other causes of obstruction:
  - Benign & malignant strictures
  - Instrumention
  - Post-operative anastomotic strictures
DIAGNOSIS

- History
- Physical – may range from clinically stable to septic
- Labs – Elevated WBC, bilirubin, ALP & transaminases
- Imaging
  - Ultrasound, CT, MR – may all be useful in identifying cause of obstruction
  - ERCP (cholangiography): mandatory diagnostic and potentially therapeutic technique
- Reynolds’ Pentad: jaundice, fever/chills, abdominal pain, mental status changes, and hypotension
TREATMENT

- Resuscitation
  - IV antibiotics
  - Aggressive hydration
- Need to decide the severity of illness – as this changes management strategy
  - May require urgent decompression
- ERCP – diagnostic and therapeutic: level of obstruction, reason for obstruction, culture, biopsy, drainage, stent, dilation
- If ERCP not available, should do PTC
- If PTC not available, emergent OR with CBD decompression with T-tube
- Definitive operative therapy should be deferred until cholangitis has been treated, the patient stabilized, and the diagnosis confirmed
Ascending cholangitis

- Acute, but not toxic
  - IV antibiotic and IV hydration
    - Ultrasound
      - Stone disease
        - ERCP or LAP
        - Cholecystectomy with IOC
      - Ductal dilation, suspect mass
        - CT/MRCP
- Toxic
  - Resuscitation and stabilization
    - IV antibiotic and IV hydration
  - Urgent decompression
    - Proximal obstruction
      - PTC
      - Unsuccessful decompression
        - Operative decompression
      - Successful decompression
    - Distal obstruction
      - ERCP
      - Successful decompression
Symptomatic Disease

Low Risk (of CBD stones)
- No routine cholangiography

Moderate Risk –
- Elevated bilirubin
- Elevated ALP
- Pancreatitis
- Multiple gallstones

MRCP, EUS or intraoperative cholangiogram

High Risk –
- Clinical jaundice
- Cholangitis
- Visible choledocholithiasis
- Dilated CBD on US

Pre-operative ERCP with sphincterotomy if required
CHOLIDOCHOLITHIASIS

- U/S as first imaging modality – however is not sensitive in this setting
- Need to assess/investigate as in initial presentation of gallstone disease: LFTs, trans-abdominal U/S
- Endoscopic ultrasound and MR – highly effective in demonstrating CBD stones: need to determine which is more accessible locally – although other option is intra-operative cholangiography
- Prophylactic Abx should be given to patients with biliary obstruction or previous evidence of biliary sepsis
IMAGING

- Ultrasound
  - Dilated extrahepatic and intrahepatic ducts
  - Can be difficult to see stones in the CBD – small stones can get lodged at distal end
- CT Scan
  - Used more to delineate the surrounding anatomy, especially if suspect malignancy (gallbladder, pancreas, extrahepatic biliary system)
- PTC (percutaneous transhepatic cholangiography)
  - Most useful for bile duct strictures & tumours (ex. obstructing cholangiocarcinoma) – anatomy & therapeutic
- MRCP
  - Single non-invasive test to define the anatomy
- ERCP
  - Diagnostic & therapeutic – sphincterotomy, duct canulation and stent placement
- Endoscopic ultrasound
TREATMENT

- Resuscitative treatment
- ERCP
- Cholecystectomy

If find CBD stones intraoperatively, have choice:
- Convert to open
- Endoscopic CBD exploration
- Laparoscopic sphincterectomy
- Intraoperative ERCP
- Post-operative ERCP
Wait-and-see policy or laparoscopic cholecystectomy after endoscopic sphincterotomy for bile-duct stones: a randomised trial


Authors noted that after ERCP for choledocholithiasis, only 10% of patients with gallbladder stones develop symptoms – wanted to determine whether it was necessary for all patients post ERCP to have cholecystectomy
Lancet 2002 cont...

- Randomized trial (over 4 years) of 120 patients with ERCP and stone extraction and proven stones in gallbladder
  - Wait and see approach
  - Laparoscopic cholecystectomy
- Primary outcome: symptoms within 2-year follow-up
- Secondary outcomes: complications from cholecystectomy & QoL
RESULTS

- 47% of expectant management patients developed symptoms (vs 2%)
  - Majority of complications were pain and cholecystitis
  - Significantly more had to be converted to open procedure in “wait and see” group – although no increase in operative complications

- 37% needed cholecystectomy

- Best management is to provide cholecystectomy at initial presentation post ERCP
INTRAOPERATIVE CHOLANGIOGRAM

- Annals of Surgery 1999: Complication of cholecystectomy: Risks of the laparoscopic approach and Protective Effect of Intraoperative Cholangiogram
- Study done in a period where CBD injuries were still double what they were with the open procedure – whereas now back down to same rates
- Intraoperative cholangiogram recommended in moderate risk patients to assess for retained stones
INDICATIONS FOR INTRAOPERATIVE CHOLANGIOGRAM

- Elevated preoperative liver enzymes (AST, ALT, ALP, bilirubin)
- Unclear anatomy during laparoscopic dissection
- Suspicion of intraoperative injury to biliary tract
- Dilated common bile duct on preoperative imaging
- Gallstone pancreatitis without endoscopic clearance of common bile duct
- Jaundice
- Large common bile duct and small stones
- Unsuccessful preoperative ERCP for choledocholithiasis

(Sabiston)
Randomised trial of laparoscopic exploration of common bile duct versus postoperative endoscopic retrograde cholangiography for common bile duct stones

M Rhodes, L Sussman, L Cohen, M P Lewis

- 2-year period – 480 patients treated for symptomatic gallstones – 427 had adequate cholangiogram – 80 found to have CBD stones
- Randomized to (after intra-op cholangiogram)
  - LECBD:
    - Transcystic approach and choledochotomy
    - ERCP as need if LECBD failed
  - Post-op ERCP:
    - Within 48 hours of surgery
    - Repeat ERCP within 1 week
RESULTS

LECBD:
- 30 (75%) had duct cleared with initial LECBD
- 100% clearance with subsequent ERCPs
- 3 patients with post-op biliary leaks
- Significantly shorter hospital stay

ERCP:
- 30 (75%) had duct cleared with first ERCP
- 5 after 2\textsuperscript{nd} ERCP
- 2 after 3\textsuperscript{rd} ERCP
- 93% duct clearance (remaining 2 were treated with long-term biliary stents)

Morbidity rates similar in both groups (different reasons)

Overall conclusion that duct clearance rates are the same, with shorter hospital stay in LECBD
ERCP Indications – NIH Guidelines

- ERCP, MRCP and endoscopic US have comparable sensitivity and specificity in the diagnosis of choledocholithiasis.
- If low probability of choledocholithiasis: patients undergoing cholecystectomy do not need pre-op ERCP.
- Laparoscopic common bile duct exploration and postoperative ERCP are both safe and reliable in clearing CBD stones.
- ERCP + sphincterotomy and stone removal: valuable therapeutic in choledocholithiasis with jaundice, dilated CBD, acute pancreatitis, or cholangitis.
- Biliary cancer: ERCP as palliation of biliary obstruction when surgery is not elected.
- ERCP for tissue sampling in pancreatic or biliary cancer (not undergoing surgery) but not always diagnostic.
- ERCP is the best means to diagnose ampullary cancers.
- ERCP has no role in the diagnosis of acute pancreatitis except when biliary pancreatitis is suspected, where early intervention with ERCP reduces morbidity and mortality compared with delayed ERCP.
- ERCP with appropriate therapy is beneficial in selected patients who have either recurrent pancreatitis or pancreatic pseudocysts.
- Patients with type I sphincter of Oddi dysfunction respond to sphincterotomy.
- Patients with type II SOD should not undergo diagnostic ERCP alone. If SOD manometer pressures are >40 mmHg, ES is beneficial in some patients.
- Avoid unnecessary ERCP – ex. when low likelihood of biliary stone or stricture.
ERCP Indications – National Guideline Clearinghouse

- Jaundice thought to be the result of biliary obstruction
- Clinical and biochemical or imaging data suggestive of pancreatic or biliary tract disease
- Signs or symptoms suggesting pancreatic malignancy when direct imaging results are equivocal or normal
- Pancreatitis of unknown etiology
- Preoperative evaluation of chronic pancreatitis or pancreatic pseudocyst
- Sphincter of Oddi manometry
- Endoscopic sphincterotomy
  - Choledocholithiasis
  - Papillary stenosis or sphincter of Oddi dysfunction causing disability
  - Facilitate biliary stent placement or balloon dilatation
  - Sump syndrome
  - Choledochocoele
  - Ampullary carcinoma in poor surgical candidates
  - Access to pancreatic duct
- Stent placement across benign or malignant strictures, fistulae, postoperative bile leak, or large common bile duct stones
- Balloon dilatation of ductal strictures
- Nasobiliary drain placement
- Pseudocyst drainage in appropriate cases
- Tissue sampling from pancreatic or bile ducts
- Pancreatic therapeutics
ERCP Indications – American Society of Gastrointestinal Endoscopy

- ERCP is primarily a therapeutic procedure for the management of pancreaticobiliary disorders (C)
- Diagnostic ERCP should not be undertaken in the evaluation of pancreaticobiliary pain in the absence of objective findings on other imaging studies (B)
- Routine ERCP before laparoscopic cholecystectomy should not be performed (B)
- Endoscopic therapy of postoperative biliary leaks and strictures should be undertaken as first-line therapy (B)
- ERCP has an important role in patients with recurrent acute pancreatitis and can identify and, in some cases, treat the underlying cause (B)
- ERCP is effective in treating symptomatic strictures in chronic pancreatitis (B)
- ERCP is effective for the palliation of malignant biliary obstruction (B), for which self-expanding metallic stents have longer patency than plastic stents (A)
- ERCP can be used to diagnose and to treat symptomatic pancreatic-duct stones (B)
- Pancreatic-duct disruptions or leaks can be effectively treated via the placement of bridging or transpapillary pancreatic stents (B)
**Sphincter of Oddi**

- Regulates flow of bile (and pancreatic juice) into the duodenum
- Prevents the regurgitation of duodenal contents into the biliary tree
- Diverts bile into the gallbladder
- Basal resting pressure of about 13 mm Hg above the duodenal pressure – phasic contractions
- Relaxation occurs with a rise in CCK – increases bile into duodenum
SPHINCTER OF ODDI DYSFUNCTION

- Poorly defined syndrome
- Pain similar to biliary colic, normal liver function tests, episodes of acute pancreatitis
- Unclear pathogenesis; theories:
  - Gallstone migration causing fibrosis of the sphincter
  - Trauma
  - Pancreatitis
  - Congenital anomalies
- About 1% of patients undergoing cholecystectomy have sphincter of Oddi dysfunction
- Diagnostic clues:
  - Ultrasound: dilated common bile duct (>12 mm diameter) or increase in common bile duct diameter in response to CCK
  - ERCP: delayed emptying of contrast medium from CBD
  - Ampullary manometry: elevated basal sphincter pressure (>40 mm Hg)
- Treatment: sphincterotomy
Gallstone Ileus

- Passage of a stone through a spontaneous biliary-enteric fistula leading to a mechanical bowel obstruction
- Most fistulas between gallbladder and duodenum
- F>M
- Average age 70yrs
- 1% of bowel obstructions, up to 25% of bowel obstructions in elderly without history of surgery or hernias
- Usually occur after an episode of acute cholecystitis –
  - Gangrene and perforation of the gallbladder or
  - Pressure necrosis from an impacted gallstone.
PRESENTATION & DIAGNOSIS

- Signs & symptoms of intestinal obstruction: nausea, vomiting, abdominal pain
- ½ have history of gallbladder-related symptoms
- Tumbling Obstruction: pain may be episodic and recurrent as the impacted stone temporarily obstructs the bowel lumen and then dislodges and moves distally
- Abdominal films: evidence of intestinal obstruction with pneumobilia or a calcified stone distant from the gallbladder
- Most common site of obstruction is the terminal ileum because of its narrow lumen
ILEUS

PA barium X-ray: irregular collection of barium in the right upper quadrant (A, arrowheads), representing partial filling of the cystic duct.; jejunum and ileum are markedly dilated, with dilution of the barium in a pattern consistent with small bowel obstruction: abrupt termination of the barium column at the site of an oval intraluminal filling defect (A, arrow). A view of the barium column shows luminal obstruction by a smooth intraluminal mass (B, arrows) with faint calcification of the peripheral rim. (From Kaiser AM et al: Gallstone ileus. N Engl J Med 335:942, 1996.)
MANAGEMENT

- Removal of gallstone through a proximal enterotomy – the stone is “milked” proximally, and then removed from a healthy portion of bowel
- May be necessary to resect any portion of bowel with ischemia - prevent postimpaction wall necrosis and leak
- Recurrent obstruction in 10% of patients – need to evaluate for other gallstones
- Takedown of the biliary-enteric fistula and cholecystectomy – risk of recurrent cholecystitis and cholangitis
- If patient unstable or significant inflammation, can address fistula at a second laparotomy