# General Surgery

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- **Acute Cholecystitis**
- **Choledocholithiasis**
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- **Thyroid and Parathyroid**
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- **Skin Lesions**
- **Common Medications**

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General Surgery GS1
**Figure 1. Abdominal incisions**

**Layers from Superficial to Deep**

- Skin (epidermis, dermis, subcutaneous fat)
- Superficial fascia
  - Camper's fascia (fatty) → Dartos
  - Scarpa's fascia (membranous) → Colles' superficial perineal fascia
- Muscle (see Figure 3 and Figure 3)
  - External oblique → inguinal ligament → external spermatic fascia → fascia lata
  - Internal oblique → cremasteric muscle/fascia
  - Transversus abdominis → posterior inguinal wall
  - Transversalis fascia → internal spermatic fascia
  - Preperitoneal fat
  - Peritoneum → tunica vaginalis
- at midline
  - Rectus abdominus muscle: in rectus sheath, divided by line of ala
- Above arcuate line (semicircular line of Douglas), which is midway between symphysis pubis and umbilicus
  - Anterior rectus sheath = external oblique spongiosis and anterior leaf of internal oblique spongiosis
  - Posterior rectus sheath = posterior leaf of internal oblique spongiosis and transversus muscle spongiosis
- Below arcuate line
  - Anterior rectus sheath = spongiosis of external, internal oblique, transversus muscles
  - Posterior rectus sheath = transversalis fascia
- Arteries: superior epigastric (branch of internal thoracic), inferior epigastric (branch of external iliac); both arteries anastomose and lie behind the rectus muscle

**Figure 2. Continuity of the Abdominal Wall with Layers of the Scrotum and Spermatic Cord**

- Transversus abdominis muscle
- Internal oblique muscle
- External oblique aponeurosis
- Preperitoneal fat
- Inferior epigastric artery
- Inferior epigastric vein
- Transversalis fascia
- Deep inguinal ring
- Membranous layer of external oblique fascia (Scarpa's fascia)
- Fatty layer of superficial fascia (Camper's fascia)
- Superficial inguinal ring
- Testis
- Internal spermatic fascia
- Cremaster muscle
- External spermatic fascia
- Colles' superficial perineal fascia
- Dartos muscle
- Skin of scrotum
Figure 3. Midline Cross-Section of Abdominal Wall

Figure 4. Blood Supply to the GI Tract

Venous Flow
- end point is the portal vein

Figure 5. Venous Drainage of the GI Tract
# Differential Diagnoses of Common Presentations

## Acute Abdominal Pain

<table>
<thead>
<tr>
<th>RUQ</th>
<th>EPIGASTRIC</th>
<th>LRQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatobiliary</td>
<td>Cardiac</td>
<td>Pancreatic</td>
</tr>
<tr>
<td>- Bilary colic</td>
<td>- Aortic dissection/ruptured AAA</td>
<td>- Pancreatitis (acute vs. chronic)</td>
</tr>
<tr>
<td>- Choledolithiasis</td>
<td>- MI</td>
<td>- Pancreatic pseudocyst</td>
</tr>
<tr>
<td>- Choledangitis</td>
<td>- Pericarditis</td>
<td>- Pancreatic tumors</td>
</tr>
<tr>
<td>- GDU obstruction (stone, tumor)</td>
<td>- Gastrointestinal</td>
<td>Gastrointestinal</td>
</tr>
<tr>
<td>- Hepatitis</td>
<td>- Gastritis</td>
<td>Gastritis</td>
</tr>
<tr>
<td>- Biliary Cholangitis</td>
<td>- GERD/Esophagitis</td>
<td>- Peptic ulcer disease</td>
</tr>
<tr>
<td>- Acute cholecystitis</td>
<td>- Peptic ulcer disease</td>
<td>- Splenic injury/shear tear</td>
</tr>
<tr>
<td>- High amylase/mass</td>
<td>- Pancreatitis</td>
<td>- Splenic injury/shear tear</td>
</tr>
</tbody>
</table>

### Gastrointestinal

<table>
<thead>
<tr>
<th>RUQ</th>
<th>EPIGASTRIC</th>
<th>LRQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatitis</td>
<td>Early appendicitis, perforated appendicitis</td>
<td>Pancreatic</td>
</tr>
<tr>
<td>- Presentation of gastric, duodenal or pancreatic pathology</td>
<td>- Mesenteric ischemia</td>
<td>- Splenic injury/shear tear</td>
</tr>
<tr>
<td>- Hepatic failure pathology (CRF, subcapsular incisional hernia)</td>
<td>- Gastroenteritis/Colicis</td>
<td>- Splenic rupture</td>
</tr>
<tr>
<td>- Gastritis</td>
<td>- Constipation</td>
<td>- Splenic avulsion</td>
</tr>
<tr>
<td>- Nephrolithiasis</td>
<td>- Bezoar obstruction</td>
<td>- Carotid dissection (see RUQ and Epigastrix)</td>
</tr>
<tr>
<td>- Peptic ulcer</td>
<td>- Pancreatitis</td>
<td>- Carotid dissection (see RUQ)</td>
</tr>
<tr>
<td>- Renal: mass, ischemia, trauma</td>
<td>- Interstitial bowel disease</td>
<td>- LLQ</td>
</tr>
<tr>
<td>- Cecal/ileo</td>
<td>- Irritable bowel syndrome</td>
<td>- Gastrointestinal</td>
</tr>
<tr>
<td>- Peritonitis</td>
<td>- Ogilvie’s syndrome</td>
<td>- Diverticulitis</td>
</tr>
<tr>
<td>- Effusion/Empyema</td>
<td>- Colon cancer</td>
<td>- Diverticulitis</td>
</tr>
<tr>
<td>- GIH (causing hepatic congestion and retroperitoneal effusion)</td>
<td>- Liver failure</td>
<td>- Colorectal/Sigmoid/Rectal Cancer</td>
</tr>
<tr>
<td>- MI</td>
<td>- Peritonitis</td>
<td>- Fecal inspection</td>
</tr>
<tr>
<td>- Pericarditis</td>
<td>- Peritonitis</td>
<td>- Perforated duodenal ulcer, perforation; i.e. gastroduodenitis or cholecystitis</td>
</tr>
<tr>
<td>- Peptic ulcer</td>
<td>- Peritonitis</td>
<td>- Sigmoid diverticulitis</td>
</tr>
<tr>
<td>- Peoria</td>
<td>- Peritonitis</td>
<td>- Adhesions</td>
</tr>
<tr>
<td>- Miscellaneous</td>
<td>- Peritonitis</td>
<td>- Hypertension</td>
</tr>
<tr>
<td>- Appendicitis</td>
<td>- Peritonitis</td>
<td>- Toxic shock syndrome</td>
</tr>
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<td>- Appendicitis</td>
<td>- Peritonitis</td>
<td>- Diabetic ketoacidosis</td>
</tr>
<tr>
<td>- Appendicitis</td>
<td>- Peritonitis</td>
<td>- Addisonian crisis</td>
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<td>- Appendicitis</td>
<td>- Peritonitis</td>
<td>- Hyporexia</td>
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<tr>
<td>- Appendicitis</td>
<td>- Peritonitis</td>
<td>- Other</td>
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<tr>
<td>- Appendicitis</td>
<td>- Peritonitis</td>
<td>- Lead poisoning</td>
</tr>
<tr>
<td>- Peritonitis</td>
<td>- Peritonitis</td>
<td>- Tertiary syphilis</td>
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</tbody>
</table>

### SUPRAPUBIC

<table>
<thead>
<tr>
<th>RUQ</th>
<th>EPIGASTRIC</th>
<th>LRQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal (see RUQ/ LLQ)</td>
<td>Acute appendicitis</td>
<td>Acute appendicitis</td>
</tr>
<tr>
<td>- Appendicitis</td>
<td>- RUQ</td>
<td>- RUQ</td>
</tr>
<tr>
<td>- Cecal pseudoobstruction</td>
<td>- Ectopic pregnancy</td>
<td>- Ectopic pregnancy</td>
</tr>
<tr>
<td>- Thromboembolic disease</td>
<td>- Endometriosis</td>
<td>- Endometriosis</td>
</tr>
<tr>
<td>- Hemorrhagic shock</td>
<td>- Threatened/Imperfect abortion</td>
<td>- Threatened/Imperfect abortion</td>
</tr>
<tr>
<td>- Hemorrhagic shock</td>
<td>- Hemorrhagic shock</td>
<td>- Hemorrhagic shock</td>
</tr>
<tr>
<td>- Tubo-ovarian abscess</td>
<td>- Gynecological</td>
<td>- Gynecological</td>
</tr>
<tr>
<td>- Gynecological</td>
<td>- Torsion</td>
<td>- Torsion</td>
</tr>
<tr>
<td>- Torsion</td>
<td>- Gynecological</td>
<td>- Gynecological</td>
</tr>
<tr>
<td>- Gynecological</td>
<td>- Acute urinary retention</td>
<td>- Acute urinary retention</td>
</tr>
<tr>
<td>- Acute urinary retention</td>
<td>- Ectopic pregnancy</td>
<td>- Ectopic pregnancy</td>
</tr>
</tbody>
</table>

### Miscellaneous

- Suprapubic pain, nausea, vomiting, and gastrointestinal symptoms may also be present.

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**Note:** This list is not exhaustive and may require further investigation. Always consult a healthcare professional for a detailed diagnosis.

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**References:**
Abdominal Mass

Table 2. Differential Diagnosis of Abdominal Mass

<table>
<thead>
<tr>
<th>Right Upper Quadrant (RUQ)</th>
<th>Upper Midline</th>
<th>Left Upper Quadrant (LUQ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gallbladder – cholecystitis, cholangiocarcinoma, cholelithiasis</td>
<td>Pancreas – pancreatic adenocarcinoma, IPMT, other pancreatic cancer, pseudocyst</td>
<td>Spleen – splenomegaly, tumour, abscess, subcapsular splenic hemorhage, can also present as RUQ mass if extreme splenomegaly</td>
</tr>
<tr>
<td>Biliary tract – cholangiocarcinoma</td>
<td>Abdominal aorta – AAA (pulsatile)</td>
<td>Stomach – tumour</td>
</tr>
<tr>
<td>Liver – hepatocellular carcinoma, metastatic tumour, etc.</td>
<td>Gastric tumour (adenocarcinoma, gastrointestinal stromal tumour, carcinoid tumour), MALT lymphoma</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Right Lower Quadrant (RLQ)</th>
<th>Lower Midline</th>
<th>Left Lower Quadrant (LLQ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intestine – ileum, tumour (IOIC), mesenteric adenitis, appendicitis, appendiceal phlegmon or other abscess, typhilitis, intussusception, Crohn’s inflammation</td>
<td>Uterus – pregnancy, leiomyoma (fibroid), uterine cancer, pyometra, hematometria</td>
<td>Intestine – ileum, tumour, abscess (see RLQ)</td>
</tr>
<tr>
<td>GU – bladder dissection, tumour</td>
<td>Ovary – ectopic pregnancy, cyst (physiological vs pathological), tumour (serosa, mucosa, struma ovarii, germ cell, krukenberg)</td>
<td>Ovary – ectopic pregnancy, cyst, tumour (see RLQ)</td>
</tr>
<tr>
<td>Fallopian tube – ectopic pregnancy, tubo-ovarian abscess, hydrosalphinx, tumour</td>
<td>Fallopian tube – ectopic pregnancy, tubo-ovarian abscess, hydrosalphinx, tumour</td>
<td></td>
</tr>
</tbody>
</table>

GI Bleeding

- see Gastroenterology, G26-29

Indications for Surgery
- failure of medical management
- prolonged bleeding, significant blood loss (requiring >6 units of pRBCs in a short period of time), high rate of bleeding, hypotension
- bleeding that persists despite endoscopic and angiographic therapeutic maneuvers

Surgical Management of GI Bleeding
- upper GI bleeding:
  - bleeding from a source proximal to the ligament of Treitz
  - often presents with hematemesis and melena unless very brisk (then can present with BRBP, hypotension, tachycardia)
  - initial management with endoscopy, if fails, then consider surgery
- lower GI bleeding:
  - bleeding from a source distal to the ligament of Treitz
  - often presents with BRBP unless proximal to transverse colon
  - may occasionally present with melena
  - initial management with colonoscopy to detect and potentially stop source of bleeding
  - angiography, RBC scan to determine source as indicated
  - surgical intervention if no source found

Table 3. Differential Diagnosis of GI Bleeding

<table>
<thead>
<tr>
<th>Anatomical Source</th>
<th>Etiology</th>
<th>OTC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematological</td>
<td>Excess anticoagulation (coumadin, heparin, etc.)</td>
<td>Congenital bleeding disorders</td>
</tr>
<tr>
<td>Nose</td>
<td>Epistaxis</td>
<td></td>
</tr>
<tr>
<td>Esophagus</td>
<td>Esophageal varices</td>
<td>Auto-esophageal fistula (generally post endovascular stent repair)*</td>
</tr>
<tr>
<td></td>
<td>Mallory-Weiss tear</td>
<td>Esophageal cancer</td>
</tr>
<tr>
<td></td>
<td>Esophagitis</td>
<td></td>
</tr>
<tr>
<td>Stomach</td>
<td>Gastritis</td>
<td>Gastric ulcer</td>
</tr>
<tr>
<td></td>
<td>Gastric varices</td>
<td>Gastric cancer*</td>
</tr>
<tr>
<td></td>
<td>Delafay lesion</td>
<td></td>
</tr>
<tr>
<td>Duodenum</td>
<td>Duodenal ulcer</td>
<td>Duodenal cancer*</td>
</tr>
<tr>
<td></td>
<td>Peptic duodenal ulcer*</td>
<td></td>
</tr>
<tr>
<td>Jejunum</td>
<td>Tumours*</td>
<td></td>
</tr>
<tr>
<td>Ileum and Ileocecal Junction</td>
<td>Meckel’s diverticulum (rare surgical management)</td>
<td>Crohn’s disease*</td>
</tr>
<tr>
<td></td>
<td>Small bowel obstruction</td>
<td>Tuberculosis of ileocecal junction</td>
</tr>
</tbody>
</table>
Table 2. Differential Diagnosis of GI Bleeding (continued)

<table>
<thead>
<tr>
<th>Anatomical Source</th>
<th>Pathology</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large Intestine</td>
<td>Colorectal cancer*</td>
<td>Crohn’s disease (less frequently presents with bleeding)*</td>
</tr>
<tr>
<td></td>
<td>Necrotizing enterocolitis/obstructive ileus*</td>
<td>Pancreatitis (infarct, chemotherapy or radiation induced)</td>
</tr>
<tr>
<td></td>
<td>Ulcerative colitis* (unusual calibre if failure of medical management)</td>
<td>Bleeding post-partial gastrectomy</td>
</tr>
<tr>
<td></td>
<td>Angiodysplasia</td>
<td>Bleeding post-gastrectomy/index amputation</td>
</tr>
<tr>
<td>Sigmoid</td>
<td>Diverticulosis*</td>
<td>Polyposis (surgical management if not amenable to colonoscopy/polypectomy)</td>
</tr>
<tr>
<td></td>
<td>Sigmoid cancer*</td>
<td>Inflammatory bowel disease (IBD)</td>
</tr>
<tr>
<td>Rectum and Anal</td>
<td>Hemorrhoids</td>
<td>Polyposis (surgical management if not amenable to polypectomy)</td>
</tr>
<tr>
<td></td>
<td>Fissures</td>
<td>Crater or ulcerative colitis</td>
</tr>
<tr>
<td></td>
<td>Rectal cancer*</td>
<td>Crohn’s or ulcerative colitis</td>
</tr>
<tr>
<td></td>
<td>Anal varices</td>
<td>Solitary rectal ulcer syndrome</td>
</tr>
</tbody>
</table>

*Managed surgically in most cases

Jaundice

- see Gastroenterology C44

Differential Diagnosis

- Pre-hepatic
  - Pathology occurring prior to the liver
  - Hemolysis
  - Gilbert’s disease, Crigler-Najjar disease

- Hepatic
  - Pathology occurring at the level of the liver
  - Viral hepatitis
  - Alcoholic hepatitis, cirrhosis
  - Drug-induced hepatitis – acetaminophen, erythromycin, isoniazid, valproic acid, phenytoin, oral contraceptive pill
  - Dubin-Johnson syndrome

- Post-hepatic
  - Pathology is located after the conjugation of bilirubin in the liver
  - Cholelithiasis, choledocholithiasis, sderosing cholangitis, choledochal cyst
  - Benign biliary stricture
  - Carcinoma – bile duct, head of pancreas, ampulla of Vater, duodenum

Preoperative Preparations

Considerations

- Informed consent (see Ethical, Legal, and Organizational Aspects of Medicine, ELOAM8)
- Consults – anesthesia, medicine, cardiology as indicated
- NP0 after midnight, AAT (activity as tolerated), VSR (vital signs routine)
- IV – balanced crystalloid at maintenance rate (40:1 rule → roughly 100–125 cc/hr): normal saline or Ringer’s lactate; bolus to catch up on estimated losses including losses from bowel prep
- Patient’s regular medications including prednisone – consider pre-op stress dose if prednisone used in past year
- Prophylactic antibiotics (within 1 hour prior to incision): usually cefazolin (Ancef®)
  + metronidazole (Flagyl®)
- Bowel prep: cleans out bowel and decreases bacterial population
  - Oral cathartic (e.g. fleet Phospho-soda®) starting previous day
  - Used for left-sided or rectal resections (routine use is controversial and probably unnecessary)
- Consider DVT prophylaxis for all inpatient surgery (heparin)
- Hold ASA x 1 week prep
- Smoking cessation x 6 weeks prep can significantly decrease postop complications

Investigations

- Blood components: group and screen or cross and type depending on procedure
- CBC, electrolytes, BUN, creatinine
- INR/PTT, PTT with history of bleeding disorder
- ARBs if prediagnosed to respiratory insufficiency
- CXR (PA and lateral) if >50 years old or previously abnormal within past 6 months
- ECG if >50 years old or as indicated by history

Drains

- Nasogastric (NG) tube:
  - Indications: gastric decompression, analysis of gastric contents, irrigation/dilution of gastric contents; feeding (only if necessary due to risk of aspiration → nasojejunal tube preferable)
  - Contraindications: suspected basal skull fracture, obstruction of nasal passages due to trauma
- Foley catheter:
  - Indications: to accurately monitor urine output, decompression of bladder, relieve obstruction
  - Contraindications: suspected urethral injury, difficult insertion of catheter
Surgical Complications

Postoperative Fever

- Fever does not necessarily imply infection
- Timing of fever may help identify cause
- POD 0-2:
  - Atelectasis (most common cause of fever on POD 1)
  - Early wound infection (especially Clostridium, Group A Streptococcus – feel for crepitus and look for “dishwater” drainage)
  - Aspiration pneumonitis
  - Other: Addisonian crisis, thyroid storm, transfusion reaction
- POD 3:
  - Infections more likely
  - UTI, wound infection, IV site infection, septic thrombophlebitis
  - POD 5-6:
    - Leaks at bowel anastomosis (tachycardia, hypotension, oliguria, abdominal pain)
    - Intra-abdominal abscess (usually POD 5-10)
    - DVT/PE (can be anytime post-op, most commonly POD 7-10)
    - Drug fever (POD 6-10)
  - Other: cholecystitis, peri-rectal abscess, URTI, infected seroma/biloma/hematomas, parotitis, C. difficile colitis, endocarditis

Treatment
- Treat primary cause
- Antipyretics (e.g. acetaminophen)

Wound Complications

Wound Care
- Epithelialization of wound occurs 48 hours after closure
- Dressings applied in the operating room can be removed POD 2-4
- Leave uncovered if wound is dry
- Remove dressings if wet, signs of infection (fever, tachycardia, pain)
- Examination of the wound: Inspect, compress adjacent areas, swab drainage for C&S and Gram stain
- Skin sutures and staples can be removed POD 5:
  - Exceptions: incision cross closure (groin), closed under tension, in extremities (hand) or patient factors (elderly, corticosteroid use) removed POD 14, earlier if signs of infection
- Can bathe POD 2-3
- Negative pressure dressings consist of gel foam and suction, promote granulation
  - Ideal for large (grafted sites) or nonhealing wounds (irradiated skin, ulcer)

Drains
- Placed intra-operatively to prevent fluid accumulation (blood, pus, serum, bile, urine)
- Potential route of infection, bring out through separate incision (vs. operative wound) to decrease risk of wound infection
- Types of drains
  - Open (Penrose), higher risk of infection
  - Closed (Jackson-Pratt, Blake) connected to suction
  - Sump (DeVito) suction with airflow system to prevent obstruction
- Monitor drain outputs daily
- Drains should be removed once drainage is minimal (usually less than 30-50 cc/24 hr)

Wound Infection

Etiology
- S. aureus, E. coli, Enterococcus, Streptococcus spp., Clostridium spp.

Risk Factors
- Type of procedure:
  - Clean (elective, not emergent, not traumatic, no acute inflammation, resp/GI/biliary/GU tracts not entered): <1.5%
  - Clean-contaminated (elective entering of resp/GI/biliary/GU tracts): <3%
  - Contaminated (nonpurulent inflammation, gross spillage from GI, entry into biliary or GU tracts with infected bile/urine, penetrating trauma <4 hrs old): 5%
Surgical Complications

- dirty (purulent inflammation, pre-op perforation of resp/GI/biliary/GU tracts, penetrating trauma >4 hrs old): 33%
- increased risk with procedures >2 hrs long, use of drains
- patient characteristics:
  - age, DM, steroids, immunosuppression, obesity, burn, malnutrition, patient with other infections, traumatic wound, radiation, chemotherapy
- other factors:
  - prolonged preoperative hospitalization, reduced blood flow, break in sterile technique, multiple antibiotics, hematomas, sepsis, foreign bodies (drains, sutures, grafts)

Clinical Presentation
- typically fever: POD 3-6 (Streptococcus and Clostridium can present in 24 hrs)
- pain, blanchable wound erythema, induration, frank pus or purulonsanguinous discharge, warmth
- complications: fistula, sinus tracts, sepsis, abscess, suppressed wound healing, superinfection, spreading infection to myonecrosis or fascial necrosis (necrotizing fasciitis), wound dehiscence, eversion, hernia

Prophylaxis
- pre-op antibiotics for all surgeries [cefazolin (Ancef®)/metronidazole (Flagyl®)]
  - within 1 hour preincision; can re-dose with Ancef® after 4 hrs in the OR
  - post-op antibiotics for contaminated and dirty surgeries:
    - no evidence supporting more than 24 hrs of post-op antimicrobial prophylaxis for any case
    - generally no need for post-op antibiotics unless intra-abdominal infection
  - normothermia (maintain patient temperature >36°C during OR)
  - hyperoxygenation (consider FiO₂ >80 in OR)

Treatment
- re-open affected part of incision, culture wound, pack, heal by secondary intention
- antibiotics only if cellulitis or immunodeficiency
- debris necretect and non-viable tissue resectively

WOUND HEMORRHAGE/HEMATOMA
- secondary to inadequate surgical control of hemostasis

Risk Factors
- anticoagulant therapy, coagulopathies, thrombocytopenia, DIC, severe liver disease, myeloproliferative disorders, severe arterial hypertension, severe cough

Clinical Features
- pain, swelling, discolouration of wound edges, leakage
- rapidly expanding neck hematoma can compromise airway and is a surgical emergency

Treatment
- pressure dressing
  - if significant bleeding, may need to re-operate to find source

SEROMA
- fluid collection other than pus or blood
- secondary to transection of lymph vessels
- delays healing

Treatment
- pressure dressing ± needle drainage
  - if significant may need to re-operate

WOUND DEHISCENCE
- disruption of fascial layer, abdominal contents contained by skin only

Clinical Features
- typically POD #1-3, most common presenting sign is serosanguinous drainage from wound, ± evisceration (disruption of all abdominal layers and extrusion of abdominal contents – mortality of 15%)
- palpation of wound edge: should normally feel a "healing ridge" from abdominal wall closure (raised area of tissue under incision)
Risk Factors
- local: technical failure of closure, increased intra-abdominal pressure (e.g. COPD, ileus, bowel obstruction), hematoma, infection, poor blood supply, radiation
- systemic: smoking, malnutrition (hypalbuminemia, vitamin C), connective tissue diseases, immunosuppression (disease, steroids, chemotherapy), other (age, DM, sepsis, uraemia)

Treatment
- may consider conservative management
- operative closure, evisceraion is a surgical emergency

Urinary and Renal Complications

Urinary Retention
- may occur after any operation with general anesthesia or spinal anesthesia
- more likely in older males with history of benign prostatic hyperplasia (BPH), patients on anticholinergics

Clinical Presentation
- abdominal discomfort, palpable bladder, overflow incontinence

Treatment
- Foley catheter to rest bladder, then trial of void

Oliguria/Anuria (see also Nephrology, NP20)

Etiology
- pre-renal vs. renal vs. post-renal:
  - most common post-op cause is pre-renal + ischemic ATN
  - external fluid loss: hemorrhage, dehydration, diarrhea
  - internal fluid loss: third-spacing due to bowel obstruction, pancreatitis, post-op

Clinical Presentation
- urine output <0.5 cc/kg/hr, increasing Cr, increasing BUN

Treatment
- according to underlying cause; fluid deficit is treated with crystalloid, [normal saline (NS) or Ringer's lactate (RL)]

Postoperative Dyspnea
- see Respiratory Complications, GS9 and Cardiac Complications, GS11
- respiratory: atelectasis, pneumonia, pulmonary embolus (PE), acute respiratory distress syndrome (ARDS), asthma, pleural effusion
- cardiac: MI, arrhythmia, CHF
- pain

Respiratory Complications

Atelectasis
- comprises 90% of post-op pulmonary complications

Clinical Features
- low-grade fever on POD #1, tachycardia, crackles, decreased breath sounds, bronchial breathing, tachypnea

Risk Factors
- COPD, smoking, obesity, elderly persons
- upper abdominal/thoracic surgery, oversedation, significant post-op pain, poor inspiratory effort

Treatment
- pre-operative prophylaxis:
  - smoking cessation (most beneficial if >6 weeks pre-op)
- postoperative prophylaxis:
  - minimize use of respiratory depressant drugs
  - good pain control
  - incentive spirometry, deep breathing and coughing, chest physiotherapy, postural changes
  - early ambulation
PNEUMONIA/PNEUMONITIS
• may be secondary to aspiration of gastric contents during anesthetic induction or extubation, causing a chemical pneumonitis

Risk Factors
• aspiration: general anesthetic, decreased LOC, GERD, full stomach, bowel/gastric outlet obstruction + non-functioning NG tube, pregnancy, seizure disorder
• non-aspiration: atelectasis, immobility, pre-existing respiratory disease

Clinical Features
• productive cough, fever
• tachycardia, cyanosis, respiratory failure, decreased LOC
• CXR: pneumatic infiltrate

Treatment
• aspiration prophylaxis: pre-op NPO/NG tube, rapid sequence anesthetic induction
• immediate removal of debris and fluid from airway
• consider endotracheal intubation and flexible bronchoscopic aspiration
• IV antibiotics to cover oral nosocomial aerobes and anaerobes (e.g. cefotaxime, metronidazole)

PULMONARY EMBOLUS (see Respiratory, R16)

Clinical Features
• unilateral leg swelling and pain (DVT as a source of PE), sudden onset SOB, tachycardia, fever (POD #7-10)

Treatment
• IV heparin, long term warfarin (INR = 2-3) for 3 months
• Greenfield (IVC) filter if contraindications to anticoagulation
• prophylaxis: subcutaneous heparin (5000 units bid) or LMW heparin, compression stockings (TED stockings)

PULMONARY EDEMA

Etiology
• cardiogenic vs. non-cardiogenic
• circulatory overload: excess volume replacement, LV failure, shift of fluid from peripheral to pulmonary vascular bed, negative airway pressure, alveolar injury due to toxins (e.g. ARDS)
• more common with pre-existing cardiac disease
• negative pressure pulmonary edema due to inspiratory efforts against a closed glottis upon awakening from general anesthesia

Clinical Features
• SOB, crackles at lung bases, CXR abnormal

Treatment (LMNOP)
• Lasix
• Morphine (decreases symptoms of dyspnea, venodilator and afterload reduction)
• Nitrates (venodilator)
• Oxygen + non-invasive ventilation
• Position (sit patient up)

RESPIRATORY FAILURE

Clinical Features
• dyspnea, cyanosis, evidence of obstructive lung disease
• earliest manifestations: tachypnea and hypoxemia (RR >25, pO2 <60)
• pulmonary edema, unexplained decrease in SaO2

Treatment
• ABCs, O2, intubation
• bronchodilators, diuretics to treat CHF
• adequate blood pressure to maintain pulmonary perfusion
• if these measures fail to keep PaO2 >60, consider ARDS
Cardiac Complications

- Abnormal ECGs common in post-op period (compare to pre-op ECG)
- Common arrhythmias: supraventricular tachycardia (SVT), atrial fibrillation (secondary to fluid overload, PE, MI)

Myocardial Infarction (MI)

- See Cardiology and Cardiovascular Surgery, C25
- Surgery increases risk of MI
- Incidence:
  - 0.3% in previously asymptomatic men >50 years old
  - 46-fold increase in men >50 years old with previous MI

Risk Factors

- Pre-op hypertension, CHF
- Previous MI (highest risk ≤ 6 months, but risk never returns to baseline)
- Increased age
- Intra-operative hypotension
- Operations >3 hrs
- Anemia

Clinical Features

- Majority of cases on day of operation or POD #1-4
- Often silent without chest pain, may only present with new-onset CHF (dyspnea), arrhythmias, hypotension

Intra-abdominal Abscess

Definition

- Collection of pus walled-off from rest of peritoneal cavity by inflammatory adhesions and visceral

Etiology

- Usually polymicrobial: Gram-negative bacteria, anaerobes
- Consider Gram-positives if coexistent cellulitis

Risk Factors

- Emergent OR
- Post-op contaminated OR
- GI surgery with anastomoses
- Poor healing risk factors (DM, poor nutrition, etc.)
- May occur POD #3 after laparotomy when fluid re-distribution occurs

Clinical Features

- Persistent spiking fever, dull pain, weight loss
- Mass difficult to palpate
- Peritoneal signs if abscess perforation and secondary peritonitis
- Leukocytosis or leukopenia (immunocompromised, elderly)
- Co-existing effusion (pleural effusion with subphrenic abscess)
- Common sites: pelvis, Morrison's pouch (space between duodenum and liver), subphrenic, paracolic gutters, lesser sac, peri-appendiceal, post-surgical anastomosis, diverticular, psoas

Investigations

- CBC, blood cultures x 2
- CT x water-soluble contrast
- DRE (pelvic abscess)

Treatment

- IR percutaneous drainage
- Debridement of infected soft tissue around infection
- Antibiotics to cover aerobes and anaerobes (ampicillin/gentamicin/metronidazole or ciprofloxacin/metronidazole or clindamycin/gentamicin or cefotetan)

Paralytic Ileus

- See Bowel Obstruction, GS23

Delirium

- See Psychiatry, PS17 and Neurology, N10
Thoracic Surgery

Esophagus

Figure 6. Types of Hiatus Hernia – Sliding (left) and Paraesophageal (right)

SLIDING HIATUS HERNIA (Type I) (see Figure 6)
- herniation of both the stomach and the gastroesophageal (GE) junction into thorax
- 90% of esophageal hernias

Risk Factors
- age
- increased intra-abdominal pressure (e.g., obesity, pregnancy, coughing, heavy lifting, straining with constipation)
- smoking

Clinical Features
- majority are asymptomatic
- larger hernias frequently associated with GERD due to disruption of competence of GE junction and prevention of acid clearance once reflux has occurred

Complications
- most common complication is GERD
- other complications are rare and are related to reflux:
  - esophagitis (dysphagia, heartburn)
  - consequences of esophagitis (peptic stricture, Barrett’s esophagus, esophageal carcinoma)
  - extra-esophageal complications (aspiration pneumonia, asthma, cough, laryngitis)

Investigations
- barium swallow, endoscopy, or esophageal manometry (technique for measuring LES pressure) detect larger hernias
- 24-hour esophageal pH monitoring to quantify reflux
- gastroscopy with biopsy to document type and extent of tissue damage and rule out esophagitis, Barrett’s, and cancer
- CXR: globular shadow with air-fluid level visible over cardiac shadow

Treatment
- treat symptoms of GERD:
  - lifestyle modification:
    - stop smoking, weight loss, elevate head of bed, no meals < 3 hrs prior to sleeping, smaller and more frequent meals, avoid alcohol, coffee, mint and fat
  - medical:
    - antacid, H₂-antagonist, proton pump inhibitor, adjuvant prokinetic agent
  - surgical (<15%):
    - if severe complications or if refractory to medical management
    - Nissen fundoplication (usually laparoscopic)
      - fundus of stomach is wrapped around the lower esophagus and sutured in place
      - 90% success rate

Differential Diagnosis of Hiatus Hernia

GI Causes
- Cholelithiasis
- Diverticulitis
- Peptic ulcer
- Achalasia
- Peptic ulcer

Non-GI Causes
- MI
- Anemia
- Pericarditis
- GERD
- Gastritis
PARAESOPHAGEAL HIATUS HERNIA (Type II) (see Figure 6)
- herniation of all or part of the stomach through the esophageal hiatus into the thorax with an undischlocated GE junction
- least common esophageal hernia (<10%)

Clinical Features
- usually asymptomatic due to normal GE junction
- pressure sensation in lower chest, dysphagia

Complications
- hemorrhage, incarceration, strangulation, obstruction, gastric stasis ulcer

Treatment
- surgery to prevent severe complications:
  - reduce hernia and excise hernia sac, repair defect at hiatus, and Nissen fundoplication
  - may consider suturing stomach to anterior abdominal wall (gastroplasty)
  - in very elderly patients at high surgical risk consider PEG (percutaneous endoscopic gastrostomy)

MIXED HIATUS HERNIA (Type III)
- combination of Types I and II

TYPE IV HERNIA
- herniation of other abdominal organs into thorax: colon, spleen, small bowel

ESOPHAGEAL PERFORATION

Etiology
- iatrogenic (most common):
  - endoscopic, dilation, biopsy, intubation, operative, NG tube placement
- barotraumatic:
  - repeated, forceful vomiting (Bourlahe's syndrome)
  - trauma
- other: convulsions, defecation, labour (rare)
- ingestion injury:
  - foreign body, corrosive substance
  - carcinoma

Clinical Features
- neck or chest pain
- fever, tachycardia, hypotension, dyspnea, respiratory compromise
- subcutaneous emphysema, pneumothorax, hemothorax

Investigations
- CXR: pneumothorax, pneumomediastinum, pleural effusion, subdiaphragmatic air
- CT chest: widened mediastinum, pneumomediastinum
- contrast swallow (water-soluble then thin barium): contrast extravasation

Treatment
- supportive if rupture is contained:
  - NPO, vigorous fluid resuscitation, broad-spectrum antibiotics
  - surgical:
    - <24 hrs
      - primary closure of a healthy esophagus or resection of diseased esophagus
    - >24 hrs or non-viable wound edges
      - diversion and exclusion followed by delayed reconstruction (i.e. esophagostomy proximally, close esophagus distally, gastrostomy/jejunostomy for decompression/feeding)

Complications
- sepsis, abscess, fistula, empyema, mediastinitis, death
- post-op esophageal leak
- mortality 10-50% dependant on timing of diagnosis
ESOPHAGEAL CARCINOMA

Epidemiology
- male:female = 3:1
- onset 50-60 years of age
- upper (20-33%), middle (33%), lower (33-50%)
- squamous cell carcinoma (SCC) and adenocarcinoma occur with equal frequency, with adenocarcinoma becoming more common

Risk Factors
- geographic variation in incidence
- squamous cell carcinoma (SCC):
  - 4 S's: Smoking, Spirits (alcohol), Seeds (Betel nut), Scalding (hot liquids)
  - underlying esophageal disease such as strictures, diverticula, achalasia
- adenocarcinoma:
  - Barrett's esophagus (most important), smoking, obesity (increased reflux), GERD

Clinical Features
- frequently asymptomatic – late presentation
- progressive dysphagia (mechanical) – first solids then liquids
- odynophagia then constant pain
- constitutional symptoms
- regurgitation and aspiration (aspiration pneumonia)
- hematemesis, anemia
- tracheoesophageal or bronchoesophageal fistula
- direct, hematogenous or lymphatic spread:
  - trachea (coughing), recurrent laryngeal nerves (hoarseness, vocal paresthesia), aortic, liver, lung, bone, celiac and mediastinal nodes
- weight loss

Investigations
- barium swallow:
  - shows narrowing – suggestive but not diagnostic
- esophagoscopy:
  - biopsy/tissue diagnosis
  - determine extent and resectability of tumour
- CT chest/abdomen:
  - visualize local disease
  - staging workup (adrenal, liver, lung, bone metastases)
- endoscopic ultrasound (EUS):
  - visualize local disease
  - regional nodal involvement (most accurate way to stage the cancer)
- bronchoscopy:
  - rule out airway invasion in tumours of the upper and mid esophagus

Treatment
- if inoperable or unresectable (locally invasive disease or distant mets):
  - multimodal therapy:
    - concurrent external beam radiation and chemotherapy (cisplatin and 5-FU)
    - possibility of curative esophagectomy after chemoradiation if disease responds well
  - if unable to tolerate multimodal therapy or if highly advanced disease, consider palliative resection, brachytherapy, or endoscopic dilation/stenting/laser ablation for palliation
- if operable:
  - esophagectomy (transabdominal or transsternal approach) and lymphadenectomy
  - anastomosis in chest or neck
  - stomach most often used for reconstruction; may also use colon
  - neoadjuvant chemotherapy and radiation are controversial
  - adjuvant chemotherapy ± radiation usually recommended for post-op node-positive disease

Prognosis
- 5-8% operative death rate
- prognosis usually poor because presentation is usually at advanced stage

OTHER DISORDERS
- esophageal varices (see Gastroenterology, G27)
- Mallory Weiss tear (see Gastroenterology, G28)
Chest Wall

CONGENITAL ABNORMALITIES
- pectus excavatum, pectus carinatum, sternal fissures
- surgery for: cosmetic, psychosocial factors, respiratory or cardiovascular insufficiency

THORACIC OUTLET SYNDROME
- impingement of subclavian vessels and brachial plexus nerve trunk

Etiology
- congenital – cervical rib
- trauma
- degenerative – osteoporosis, arthritis

Clinical Features
- neurogenic – ulnar and median nerve motor and sensory function
- arterial – fatigue, weakness, coldness, ischemic pain, paresthesia
- venous – edema, venous dissection, collateral formation, cyanosis

Treatment
- conservative (50 to 90%)
  - physiotherapy, posture and behaviour modification
- surgical – if conservative treatment fails, removal of first or cervical rib (if applicable)

TUMOURS
- benign: fibrous dysplasia, eosinophilic granuloma, osteochondroma
- malignant: fibrosarcoma, chondrosarcoma, osteogenic sarcoma, Ewing's sarcoma, myeloma

Pleura, Lung, and Mediastinum

- see Respiratory, R21

TUBE THORACOSTOMY

Indications
- to drain abnormal large-volume air or fluid collections in the pleural space
  - hemothorax, chylothorax, empyema
  - pneumothorax, if:
    - large or progressive
    - patient is on mechanical ventilation
    - tension pneumothorax
- to facilitate pleurodesis:
  - i.e. obliteration of the pleural space by instilling talc or doxycycline to cause fibrosis and adherence of parietal and visceral pleura
  - indicated for recurrent pleural effusions (often malignant)
  - for long-term drainage of malignant effusions

Procedure
- tube size – varies according to indication; larger tube for more viscous drainage
- insertion site – typically 4th or 5th intercostal space in anterior axillary or mid-axillary line
- technique:
  - local anaesthetic
  - ~2 cm skin incision
  - Kelly clamp for blunt dissection to the pleural space, taking care to pass over the top of the rib to avoid neurovascular bundle
  - tube is inserted and sutured in place
  - tube is attached to a pleural drainage system (suction underwater seal, usually -20 mmH2O)
  - post-insertion CXR to ensure proper tube placement (posterior apex of lung)
- removal:
  - when drainage <100-200 cc/day, no air leak, and lung is fully expanded
  - consider clamping tube for 4-6 hrs then obtaining CXR to ensure lung remains expanded
  - brisk removal after patient expires and holds breath
Complications
- overall complications are rare (1-3%)
- malposition (most common complication), especially by inexperienced operators
  - tubes may dissect along the external chest wall, or may be placed below the diaphragm
- bleeding (anticoagulation is a relative contraindication)
- local infection, empyema
- perforation of lung parenchyma
- risk of re-expansion pulmonary edema when large volumes of air or fluid are drawn off quickly
  (>1.0 to 1.5 L)

Stomach and Duodenum

Peptic Ulcer Disease

GASTRIC ULCERS
- see Gastroenterology, G11, G27

Surgical Treatment
- increasingly rare due to H. pylori eradication and medical treatment

Indications for Surgery
- unresponsive to medical treatment (Intractability):
  - always operate if fails to heal completely, even if biopsy negative – could be primary gastric lymphoma or adenocarcinoma
  - dysplasia or carcinoma:
    - always biopsy ulcer for malignancy
  - hemorrhage – 3x greater risk of bleeding compared to duodenal ulcers
  - complications: obstruction, perforation, bleeding

Procedures
- distal gastrectomy with ulcer excision – Billroth I or Billroth II (see Figure 8)
- vagotomy and pyloroplasty only if acid hypersecretion – rare
- wedge resection if possible or biopsy with primary repair

DUODENAL ULCERS
- see Gastroenterology, G12
- most within 2 cm of pylorus (duodenal bulb)

Complications
- perforated ulcer (typically on anterior surface)
  - clinical features:
    - sudden onset of pain (possibly in RLQ due to track down right paracolic gutter)
    - acute abdomen – rigid, diffuse guarding
    - ileus
    - initial chemical peritonitis followed by bacterial peritonitis
  - investigations:
    - CXR – free air under diaphragm (70% of patients)
  - treatment:
    - oversew ulcer (plication) and omental (Graham) patch – most common treatment
- posterior penetration:
  - into pancreas – elevated amylase/lipase
  - constant mid-epigastric pain burrowing into back, unrelated to meals
- hemorrhage (typically on posterior surface):
  - gastro-duodenal artery involvement
  - treatment:
    - resuscitation initially with crystalloids; blood transfusion if necessary
    - diagnostic and/or therapeutic endoscopy (laser, cautery or injection); if recur, may have 2nd scope
    - surgery if severe or recurrent bleeding, hemodynamically unstable, or failure of endoscopy
      - oversowing of ulcer, pyloroplasty
  - gastric outlet obstruction:
    - etiology: ulcer can lead to edema, fibrosis of pyloric channel, neoplasm
    - clinical presentation:
      - nausea and vomiting (undigested food, non-bilious), dilated stomach, cramping abdominal pain
      - succussion splash (splashing noise heard when patient is shaken)
      - auscultate gas and fluid movement in obstructed organ
Gastric Carcinoma

Epidemiology
- male:female = 3:2
- incidence for adenocarcinoma ~ 10 per 100,000, incidence highest in Asia (Japan 80 times higher than in U.S.)
- most common age group = 50-59 years
- incidence has decreased by 2/3 in past 50 years

Risk Factors
- H. pylori, causing chronic atrophic gastritis
- hereditary nonpolyposis colorectal cancer (HNPPC)
- smoking, alcohol, smoked food, nitrosamines
- pernicious anemia associated with achlorhydria and chronic atrophic gastritis
- gastric adenocarcinoma polyps
- previous partial gastrectomy (>10 years post-gastrectomy)
- hypertrophic gastropathy
- blood type A

Clinical Features
- clinical suspicion:
  - ulcer fails to heal
  - lesion on greater curvature of stomach or cardia
- asymptomatic, insidious or late onset of symptoms:
  - postprandial abdominal fullness, vague epigastric pain
  - anorexia, weight loss
  - burning, nausea, vomiting, dyspepsia, dysphagia
  - hepatomegaly, epigastric mass (25%)
  - hematemesis, fecal occult blood, melena, iron-deficiency anemia
- signs of metastatic disease:
  - Virchow's node – left supraventricular node
  - Blumer's shelf – mass in pouch of Douglas
  - Krukenberg tumour – metastases to ovary
  - Sister Mary Joseph node – umbilical metastases
  - Iris sign – left axillary nodes
- metastasis:
  - liver, lung, brain

Investigations
- OGD and biopsy
- chest/abdo/pelvis CT
- CT for metastatic work-up (see Table 4)
Table 4. Staging of Gastric Carcinoma

<table>
<thead>
<tr>
<th>Stage</th>
<th>Criteria</th>
<th>Prognosis (5-year survival)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Muscosa and submucosa</td>
<td>70%</td>
</tr>
<tr>
<td>I</td>
<td>Extension to muscularis propia</td>
<td>30%</td>
</tr>
<tr>
<td>II</td>
<td>Extension to regional nodes</td>
<td>10%</td>
</tr>
<tr>
<td>III</td>
<td>Distant metastases or involvement of contiguous structures</td>
<td>0%</td>
</tr>
</tbody>
</table>

Treatment
- adenocarcinoma:
  - proximal lesions:
    - total gastrectomy and esophagojejunostomy – Roux-en-Y (see Figure 8)
  - distal lesions:
    - distal gastrectomy: wide margins, en bloc removal of omentum and lymph nodes
  - palliation:
    - gastric resection to decrease bleeding and relieve obstruction, enables the patient to eat
    - radiation therapy
    - studies are showing larger role for chemotherapy
- lymphoma:
  - chemotherapy ± radiation, surgery in limited cases (perforation, bleeding, obstruction)

Gastric Sarcoma

Gastrointestinal Stromal Tumour (GIST)
- most common mesenchymal neoplasm of GI tract
- derived from interstitial cells of Cajal (cells associated with Auerbach’s plexus that have autonomous pacemaker function co-ordinate peristalsis throughout the GI tract)
- 75-80% associated with tyrosine kinase (c-KIT) mutations
- most common in stomach (50%), and proximal small intestine (25%), but can occur anywhere along GI tract
- typically present with vague abdominal mass, feeling of abdominal fullness, or with secondary symptoms of bleeding and anemia
- often discovered incidentally on CT, laparotomy or endoscopy

Risk Factors
- Carney’s Triad: GISTs, paraganglioma, and pulmonary chondroma
- Type IA neurofibromatosis

Management
- surgical resection if >2 cm; follow with serial endoscopy if <2 cm then resect if growing or symptomatic
- pre-operative biopsy: controversial, but useful for indeterminate lesions:
  - not recommended if index of suspicion for GIST is high
  - percutaneous biopsy is NOT recommended due to high friability and risk of peritoneal spread
- localized GIST: surgical resection with preservation of intact pseudocapsule
- lymphadenectomy NOT recommended, as GISTs rarely metastasise to lymph nodes
- advanced disease: metastases to liver and/or peritoneal cavity:
  - chemotherapy with imatinib (tyrosine kinase inhibitor)
  - current research looking into role of imatinib as adjuvant or neoadjuvant therapy for localized GIST

Prognosis
- risk of metastatic potential depends on:
  - tumour size (worse if >10 cm)
  - mitotic activity (worse if >5 mitotic figures or 50/hpf)
  - degree of nuclear pleomorphism
  - location: with identical sites, extra-gastric location has a higher risk of progression than GISTs in the stomach
  - mets to liver, omentum, peritoneum; nodal mets rare

Bariatric Surgery
- weight reduction surgery for morbid obesity
- indications: BMI >40 or BMI >35 with related comorbidity (e.g. DM, CAD)
- requires multidisciplinary evaluation and follow-up

[Note: The note on gastric sarcoma, bariatric surgery, and the table on staging of gastric carcinoma are included for context and additional information, but the main focus is on the treatment of gastric carcinoma.]
Surgical Options
- malabsorptive/restrictive:
  - laparoscopic Roux-en-Y gastric bypass (most common)
  - staple off small gastric pouch (restrictive) with Roux-en-Y limb to pouch (malabsorptive)
  - with dumping syndrome physiology
  - most effective, higher complication rates
- restrictive:
  - laparoscopic adjustable gastric banding
  - silicone band around fundus creates pouch, adjustable through port under skin
  - laparoscopic vertical banded gastroplasty
  - vertical stapled small gastric pouch with placement of silastic ring band
- malabsorptive:
  - biliopancreatic diversion with duodenal switch
  - gastrectomy, enterenterostomy, duodenal division closure and duodenenterostomy

Complications
- perioperative mortality ~1% (anastomotic leak with peritoneal signs, PE)
- obstruction at enterenterostomy (see Complications of Gastric Surgery, below)
- staple line dehiscence
- dumping syndrome
- cholelithiasis due to rapid weight loss (20-30%)
- band abscess (if long-term)

Complications of Gastric Surgery
- most resolve within 1 year (see Figure 9)

Alkaline Reflux Gastritis (see Figure 9A)
- duodenal contents (biliary) reflux into stomach causing gastritis & esophagitis
- treatment:
  - medical: H2-blocker, metoclopramide, cholestyramine (bile acid sequestrant)
  - surgical: conversion of Billroth I or II to Roux-en-Y

Afferent Loop Syndrome (see Figure 9B)
- accumulation of bile and pancreatic secretions causes intermittent mechanical obstruction and distention of afferent limb
- clinical features:
  - early postprandial distention, RUQ pain, nausea, biliary vomiting, anemia
- treatment: surgery (conversion to Roux-en-Y increases afferent loop drainage)

Dumping Syndrome (see Figure 9C)
- early ~ 15 minutes post-prandial:
  - etiology:
    - hyperosmotic chyme released into small bowel (fluid accumulation and jejunal distention)
  - clinical features:
    - post-prandial symptoms
    - epigastric fullness or pain, emesis, nausea, diarrhea, palpitations, dizziness, tachycardia, diaphoresis
  - treatment:
    - small multiple low carbohydrate, low fat and high protein meals and avoidance of liquids with meals
    - last resort is interposition of antiperistaltic jejunal loop between stomach and small bowel to delay gastric emptying
  - late ~ 3 hours post-prandial:
    - etiology: large glucose load leads to large insulin release and hypoglycemia
    - treatment small snack 2 hours after meals

Blind-Loop Syndrome (see Figure 9D)
- bacterial overgrowth of colonic Gram-negative bacteria in afferent limb
- clinical features:
  - anemia/weakness, diarrhea, malnutrition, abdominal pain and hypocalcemia
  - treatment broad-spectrum antibiotics, surgery (conversion to Billroth I)

Postvagotomy Diarrhea (see Figure 9E)
- up to 25%
- bile salts in colon inhibit water resorption
- treatment: medical (cholestyramine), surgical (reversed interposition jejunal segment)
Small Intestine

Meckel’s Diverticulum

- remnant of the embryonic vitelline duct on antimesenteric border of ileum
- heterotopic – several types of mucosa including gastric, pancreatic, colonic
- most common true diverticulum of GI tract

Clinical Features
- 2% symptomatic
- GI bleed, small bowel obstruction (SBO), diverticulitis (mimics appendicitis)
- painless bleeding – ulceration caused by ectopic gastric mucosa
  - 50% of patients with this presentation are <2 years old

Investigations
- technetium-99 to identify the ectopic gastric mucosa (Meckel’s scan)

Complications
- fistula: umbilicus-ileum, umbilical sinus
- fibrous cord between umbilicus and ileum
- SBO due to volvulus, intussusception, perforation

Treatment
- Incidental finding – consider surgical resection
- symptomatic – fluid and electrolyte stabilization and surgical resection
- broad based – segmental resection to remove all mucosal types and ulcerated mucosa opposite the diverticulum (i.e. not simple diverticulectomy)

Tumours of Small Intestine

Risk Factors
- carcinogen exposure (red meat in diet)
- familial adenomatous polyposis (FAP), Peutz-Jegher syndrome, Gardner's syndrome
- Crohn’s disease, celiac disease
- Immunodeficiency, autoimmune disorders

Clinical Features
- usually asymptomatic until advanced
- intermittent obstruction, intussusception, occult bleeding, palpable abdominal mass, abdominal pain

Benign Tumours
- 10x more common than malignant
- usually asymptomatic until large
- most common sites: terminal ileum, proximal jejunum
- polyps:
  - adenomas
  - familial adenomatous polyposis (FAP) (see Familial Colorectal Cancer Syndromes, GS33)
  - hamartomatous
  - juvenile polyps
  - other: leiomyomas, lipomas, hemangiomas

Malignant Tumours
- usually asymptomatic until advanced stage
  - 25-30% associated with distant metastases at time of diagnosis
- adenocarcinoma:
  - most common primary tumour of small intestine
  - usually 50-70 years old, male predominance
  - usually in proximal small bowel, incidence decreases distally
  - risk factors: Crohn’s disease, FAP
  - early metastasis to lymph nodes – 80% metastatic at time of operation
  - investigations – CT abdomen, endoscopy
  - treatment – surgical resection ± chemotherapy
  - 5-year survival 25%
carcinoid:
- increased incidence 50-60 years old
- originate from enterochromaffin cell in crypt
- most commonly 60 cm from the ileocecal (IC) valve
  - appendix 45%, distal ileum 28%, rectum 17%
- often slow-growing
- classified by embryological origin (correlate with morphology, biological behaviour):
  - foregut – stomach, duodenum, pancreas
  - midgut – jejunum, ileum, appendix, ascending colon
  - hindgut – transverse, descending and sigmoid colon, rectum
- clinical features:
  - usually asymptomatic, incidental finding
  - obstruction, bleeding, crampy abdominal pain, intussusception
  - carcinoid syndrome (<10%):
    - hot flashes, hypotension, diarrhea, bronchoconstriction (wheezing), tricuspid/pulmonary valve insufficiency, right heart failure
    - requires liver involvement: lesion secretes serotonin, kynins and vasoactive peptides directly to systemic circulation (normally inactivated by liver)
      - EXCEPTION: carcinoid tumours arising in the bronchi can cause carcinoid syndrome without liver involvement because of access to systemic circulation
  - investigations:
    - most found incidentally at surgery for obstruction or appendectomy
    - elevated 5-HIAA (breakdown product of serotonin) in urine or increased 5-HT in blood
  - treatment:
    - tumour and metastases: surgical resection + chemotherapy
    - carcinoid syndrome: steroids, histamine, octreotide
  - prognosis:
    - metastatic risk 2% if size <1 cm, 90% if >2 cm
    - 5-year survival 70%, 20% with liver metastases
- lymphomas:
  - highest incidence at 70 years old, more common in males
  - usually non-Hodgkin's lymphoma
  - location:
    - usually distal ileum
    - proximal jejunum in patients with celiac disease
  - clinical features:
    - fatigue, weight loss, abdominal pain, fever, malabsorption
    - rarely – perforation, obstruction, bleeding, intussusception
  - treatment:
    - low grade: chemotherapy with cyclophosphamide
    - high grade: surgical resection, radiation
    - palliative: somatostatin, doxorubicin
  - 5-year survival 40%
- metastatic:
  - most common site of GI metastases in patients with metastatic melanoma
  - hemotogenous spread from breast, lung, kidney
  - direct extension from cervix, ovaries, colon
- gastrointestinal stromal tumours (GISTs):
  - see Gastric Sarcoma section, GS18

**Hernia**

**Definition**
- fascial defect → protrusion of a viscus into an area in which it is not normally contained

**Epidemiology**
- male/female = 9:1
- lifetime risk of developing a hernia: males 20-25%, females 2%
- 50% are indirect inguinal hernia, 25% are direct inguinal hernia, 5% are femoral
- most common surgical disease of males

**Risk Factors**
- activities which increase intra-abdominal pressure:
  - obesity, chronic cough, pregnancy, constipation, straining on urination or defecation, ascites, heavy lifting
  - congenital abnormality (e.g. patent processus vaginalis)
  - previous hernia repair
Clinical Features
- mass of variable size
- tenderness worse at end of day, relieved with supine position or with reduction
- abdominal fullness, vomiting, constipation
- transmits palpable impulse with coughing or straining

Investigations
- physical examination usually sufficient
- ultrasound ± CT?

Classification
- complete – hernia sac and contents protrude through defect
- incomplete – partial protrusion through the defect
- internal hernia – sac herniating into or involving intra-abdominal structure
- external hernia – sac protrudes completely through abdominal wall
- strangulated hernia – vascular supply of protruded viscus is compromised (ischemia)
  * requires emergency repair
- incarcerated hernia – irreducible hernia, not necessarily strangulated
- Richter’s hernia – only part of circumference of bowel (usually anti-mesenteric border) is incarcerated or strangulated so may not be obstructed
  * a strangulated Richter’s hernia may self-reduce and thus be overlooked, leaving a gangrenous segment at risk of perforation
- sliding hernia – part of wall of hernia formed by protruding viscus (usually cecum, sigmoid colon, bladder)

Anatomical Types
- groin (see Tables 5 and 6)
  * indirect and direct inguinal, femoral (see Figure 12)
  * pantaloon: combined direct and indirect hernias, peritoneum draped over inferior epigastric vessels
- epigastric: defect in linea alba above umbilicus
- incisional: ventral hernia at site of wound closure, may be secondary to wound infection
- other: Littre’s (involving Meckel’s), Amyand’s (containing ruptured appendix), lumbar, obturator, paraesophageal, umbilical, Spigelian (ventral hernia through linea semilunaris)

Complications
- incarceration: irreducible
- strangulation: irreducible with resulting ischemia:
  * small, new hernias more likely to strangulate
  * femoral >> indirect inguinal > direct inguinal
  * intense pain followed by tenderness
  * intestinal obstruction, gangrenous bowel, sepsis
  * surgical emergency
- DO NOT attempt to manually reduce hernia if septic or if contents of hernial sac gangrenous

Treatment
- surgical treatment (herniorrhaphy) is only to prevent strangulation and evisceration or for cosmetic or symptomatic reasons if asymptomatic can delay surgery
- repair may be done open or laparoscopic and may use mesh for tension-free closure
- most repairs are now done with a plug in the hernial defect and a patch over it or patch alone
- observation is acceptable for small asymptomatic inguinal hernias

Postoperative Complications
- recurrence (15-20%):
  * risk factors: recurrent hernia, age >50, smoking, BMI >25, poor pre-op functional status
  * (ASA ≥ 3 – see Anaesthesia, M4), associated medical conditions: type II DM, hyperlipidemia, immunosuppression, any comorbid conditions increasing intra-abdominal pressure
  * less common with mesh "tension-free" repair
- scrotal hematoma (3%):
  * painful scrotal swelling from compromised venous return of testes
  * deep bleeding – may enter retroperitoneal space and not be initially apparent
  * difficulty voiding
- nerve entrapment:
  * ilioinguinal
  * genital branch of genitofemoral (In spermatic cord)
- stenosis/occlusion of femoral vein:
  * acute leg swelling
  * ischemic colitis
### Groin Hernias

<table>
<thead>
<tr>
<th>Table 5. Groin Hernias</th>
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<tbody>
<tr>
<td><strong>Direct Inguinal</strong></td>
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<tr>
<td><strong>Epidemiology</strong></td>
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<tr>
<td><strong>Etiology</strong></td>
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<tr>
<td><strong>Anatomy</strong></td>
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<tr>
<td><strong>Treatment</strong></td>
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<tr>
<td><strong>Prognosis</strong></td>
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<table>
<thead>
<tr>
<th>Table 6. Superficial Inguinal Ring vs. Deep Inguinal Ring</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Superficial Inguinal Ring</strong></td>
</tr>
<tr>
<td>Opening in ext. abdominal aponaeurosis; palpable superior and lateral to pubic tubercle</td>
</tr>
<tr>
<td>Medial border: medial crus of ext. abdominal aponaeurosis</td>
</tr>
<tr>
<td>Lateral border: lateral crus of ext. oblique aponaeurosis</td>
</tr>
<tr>
<td>Roof: Intercrural fibres</td>
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</tbody>
</table>

### Bowel Obstruction

**Definition**
- partial or complete blockage of the bowel resulting in failure of intestinal contents to pass through lumen.

**Pathogenesis**
- disruption of the normal flow of intestinal contents → proximal dilation + distal decompression
- may take 12-24 hrs to decompress, therefore passage of feces and flatus may occur after the onset of obstruction
- bowel ischemia may occur if blood supply is strangled or bowel wall inflammation leads to venous congestion
- bowel wall edema and disruption of normal bowel absorptive function → increased intraluminal fluid → transudative fluid loss into peritoneal cavity, electrolyte disturbances

**Differential Diagnosis**
- small bowel obstruction (SBO), large bowel obstruction (LBO), pseudo-obstruction

**Clinical Features**
- must differentiate between obstruction and ileus, and characterize obstruction as acute vs. chronic, partial vs. complete (constipation vs. obstruction), small vs. large bowel, strangulating vs. non-strangulating, and with vs. without perforation

<table>
<thead>
<tr>
<th>Table 7. Bowel Obstruction vs. Paralytic Ileus</th>
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<tbody>
<tr>
<td><strong>SBO</strong></td>
</tr>
<tr>
<td>Nausea, Vomiting</td>
</tr>
<tr>
<td>Abdominal Pain</td>
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<tr>
<td>Abdominal Distention</td>
</tr>
<tr>
<td>Constipation</td>
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<tr>
<td>Other</td>
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<tr>
<td>Bowel Sounds</td>
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<tr>
<td>Abdominal Aperistals</td>
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<tr>
<td>AXR Findings</td>
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</table>
Complications of total obstruction
- strangulating obstruction (10% of bowel obstructions) = surgical emergency:
  - cramping pain turns to continuous ache; hematemesis, melena (if infarction)
  - fever, leukocytosis, tachycardia
  - peritoneal signs, early shock
  - see also Intestinal Ischemia, GS27
- other:
  - perforation: secondary to ischemia and luminal distention
  - sepsis
  - hypovolemia (due to third spacing)

Investigations
- radiological:
  - upright CXR or left lateral decubitus (LLD) to rule out air free, usually seen under the right hemidiaphragm
  - abdominal x-ray (3 views) to determine SBO vs. LBO vs. ileus (see Table 7)
    - if ischemic bowel look for: free air, pneumatosis, thickened bowel wall, air in portal vein, dilated small and large bowels, thickened or beak-like haustra (normally finger-like projections)
  - other:
    - CT provides information on level of obstruction, severity, cause
    - upper GI series/small bowel series for SBO (if no cause apparent, i.e. no hernias, no previous surgeries)
    - if suspect LBO, consider a rectal water-soluble (Gastrografin® for PO/PR; Hypaque® for IV) enema rather than barium enema (can thicken and cause complete obstruction)
    - may consider ultrasound or MRI in pregnant patients
- laboratory:
  - may be normal early in disease course
  - BUN, creatinine, hematocrit (hemococoncentration) to assess degree of dehydration
  - fluid, electrolyte abnormalities
  - anlyase elevated
  - metabolic acidosis due to frequent enemas
  - if strangulation: leukocytosis with left shift, lactic acidosis, elevated LDH (late signs)

Treatment
- stabilize vitals, fluid and electrolyte resuscitation (with normal saline/Ringer's first, then with added potassium after fluid deficits are corrected)
- NG tube to relieve vomiting, prevent aspiration and decompress small bowel by prevention of further distention by swallowed air
- Foley catheter to monitor in/out

Small Bowel Obstruction (SBO)

Etiology

<table>
<thead>
<tr>
<th>Common Causes of SBO</th>
<th>Intraluminal</th>
<th>Intramural</th>
<th>Extramural</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intussusception</td>
<td>Crohn's</td>
<td></td>
<td>Adhesions</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>Radiation stenosis</td>
<td></td>
<td>Incarcerated hernia</td>
</tr>
<tr>
<td>Ascites</td>
<td>Adenocarcinoma</td>
<td></td>
<td>Peritoneal carcinomatosis</td>
</tr>
</tbody>
</table>

Treatment
- consider whether complete or partial obstruction, ongoing or impending strangulation, location and cause:
- SBO with history of abdo/pelvic surgery → conservative management (likely to resolve) → surgery if no resolution in 48-72 hrs or complications
- complete SBO, strangulation → urgent surgery after stabilizing patient
- trial of medical management may be indicated in Crohn's, recurrent SBO, carcinomatosis
- special case: early postoperative SBO (within 30 days of abdominal surgery) – prolonged trial of conservative therapy is appropriate, surgery is reserved for complications such as strangulation

Prognosis
- mortality: non-strangulating <1%, strangulating 8% (25% if >36 hours), ischemic = up to 50%

Top 3 Causes of SBO (in order)
1. Adhesions
2. Blipe (hernias)
3. Cancer (neoplasms)
Large Bowel Obstruction (LBO)

Etiology

<table>
<thead>
<tr>
<th>Intraluminal</th>
<th>Intussusception</th>
<th>Volvulus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constipation</td>
<td>Adenocarcinoma</td>
<td>Diverticulae</td>
</tr>
<tr>
<td></td>
<td>Diverticulae</td>
<td>Ileoceleal intussuption</td>
</tr>
<tr>
<td></td>
<td>Radiation injury</td>
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</tbody>
</table>

Clinical Features (unique to LBO)
- open loop (10-20%) (safe):
  - incompetent ileocecal valve allows relief of colonic pressure as contents reflux into ileum, therefore clinical presentation similar to SBO
- closed loop (90-99%) (dangerous):
  - incompetent ileocecal valve, resulting in proximal and distal occlusions
  - massive colonic distention → increased pressure in cecum → bowel wall ischemia → necrosis → perforation

Treatment
- surgical correction of obstruction (usually requires resection + temporary diverting colostomy)
- volvulus requires sigmoidoscopic or endoscopic decompression followed by operative reduction if unsuccessful
  - if successful, consider sigmoid resection on same admission

Prognosis
- overall mortality: 10%
- caecal perforation + feculent peritonitis: 20% mortality

Pseudo-Obstruction

Definition
- condition with symptoms of intestinal blockage without any physical signs of blockage

Differential Diagnosis
- acute: toxic megacolon, trauma, postoperative, neurologic disease, retroperitoneal disease
- chronic: neurologic disease (enteric, central, peripheral nervous system), scleroderma

Toxic Megacolon

Pathogenesis
- extension of inflammation into smooth muscle layer causing paralysis
- damage to myenteric plexus and electrolyte abnormalities are not consistently found

Etiology
- inflammatory bowel disease (ulcerative colitis > Crohn’s Disease)
- infectious colitis: bacterial (C. difficile, Salmonella, Shigella, Campylobacter), viral (cytomegalovirus), parasitic (E. histolytica)
- volvulus, diverticulae, ischemic colitis, obstructing colon cancer are rare causes

Clinical Features
- infectious colitis usually present for >1 week before colonic dilatation
- diarrhea ± blood (but improvement of diarrhea may portend onset of megacolon)
- abdominal distention, tenderness, + local/general peritoneal signs (suggest perforation)
- triggers: hypokalemia, constipating agents (opioids, antidepressants, loperamide, anticholinergics), barium enema, colonoscopy

Diagnostic Criteria
- must have both colitis and systemic manifestations for diagnosis
- radiologic evidence of dilated colon
- three of: fever, HR >120, WBC >10.5, anemia
- one of: fluid and electrolyte disturbances, hypotension, altered LOC
Investigations
- CBC (leukocytosis with left shift, anemia from bloody diarrhea), electrolytes, elevated CRP, ESR
- metabolic alkalosis (volume contraction and hypokalemia) and hypoalbuminemia are late findings
- AXR: dilated colon >6 cm (right > transverse > left), loss of haustra
- CT: useful to assess underlying disease

Treatment
- NPO, NG tube, step constipating agents, correct fluid and electrolyte abnormalities, transfusion
- serial AXRs
- broad-spectrum antibiotics (reduce sepsis, anticipate perforation)
- aggressive treatment of underlying disease (eg. steroids in IBD, metronidazole for C. difficile)
- indications for surgery (50% improve on medical management):
  - worsening or persisting toxicity or dilation after 48-72 hrs
  - severe hemorrhage, perforation
- procedure: subtotal colectomy + end ileostomy with 2nd operation for re-anastomosis

Prognosis
- average 25-30% mortality

Paralytic ileus

Pathogenesis
- temporary paralysis of the myenteric plexus

Associations
- postoperative, intra-abdominal sepsis, medications (opiates, anesthetics, psychotropics), electrolyte disturbances (Na, K, Ca), C. difficile, inactivity

Treatment
- NG decompression, NPO, fluid resuscitation, correct causative abnormalities (e.g. sepsis, medications, electrolytes), consider TPN for prolonged ileus
- post-op: gastric and small bowel motility returns by 24-48 hrs, colonic motility by 3-5 d
- current interest in novel therapies such as gum chewing and pharmacologic therapy (opioid antagonists, neostigmine)

Ogilvie’s Syndrome

- acute pseudo-obstruction
- distention of colon without mechanical obstruction in distal colon
- arises in bedridden patients with serious extraintestinal illness or trauma
- exact mechanism unknown, likely autonomic motor dysregulation → possibly sympathetic deprivation to colon, unopposed parasympathetic tone, and interruption of sacral parasympathetic tone to distal bowel
- first presents with abdominal distention (>50%) & tenderness
- later symptoms mimic true obstruction

Associations
- most common: trauma, infection, cardiac (MI, CHF)
- disability (long term debilitation, chronic disease, bed-bound nursing home patients, paraplegia), drugs (narcotic use, laxative abuse, polypharmacy), other (recent orthopaedic or neurosurgery, post-partum, hypokalemia, retroperitoneal hematoma, diffuse carcinomatosis)

Investigations
- AXR: cecal distalation – if diameter ≥12 cm, increased risk of perforation

Treatment
- treat underlying cause
- NPO, NG tube
- decompression: rectal tube, colonoscopy, neostigmine (cholinergic drug), surgical decompression (ostomy/resection) uncommon
- surgery (extremely rare): if perforation, ischemia or failure of conservative management

Prognosis
- most resolve with conservative management
Intestinal Ischemia

Etiology
- acute:
  - arterial:
    - occlusive: thrombotic, embolic, extrinsic compression (e.g., strangulating hernia)
    - non-occlusive: mesenteric vasoconstriction 2nd to systemic hypoperfusion (preserves supply to vital organs)
  - trauma/dissection
  - venous thrombosis (prevents venous outflow): consider hypercoagulable state, deep vein thrombosis (DVT)
- chronic: usually due to atherosclerotic disease – look for CVD risk factors

Clinical Features
- acute: severe abdominal pain out of proportion to physical findings, vomiting, bloody diarrhea, bloating, minimal peritoneal signs early in course, hypotension, shock, sepsis
- chronic: postprandial pain, fear of eating, weight loss
- common sites: superior mesenteric artery (SMA) supplied territory, “watershed” areas of colon – splenic flexure, left colon, sigmoid colon

Investigations
- labs: leukocytosis (non-specific), lactic acidosis (late finding)
  - amylase, LDH, CK, ALP can be used to observe progress
- hypercoagulability workup if suspect venous thrombosis
- ABR: portal venous gas, intestinal pneumomatisis, free air if perforation
- contrast CT: thickened bowel wall, luminal dilatation, SMA or SMV thrombus, mesenteric/portal venous gas, pneumomatisis
- CT angiography is the gold standard for acute arterial ischemia

Treatment
- fluid resuscitation, NPO, prophylactic broad-spectrum antibiotics
- exploratory laparotomy
- angiogram, embolectomy/thrombectomy, bypass/graft, mesenteric endarterectomy, microsurgical therapy
- segmental resection of necrotic intestine:
  - assess extent of viability; if extent of bowel viability is uncertain, a second look laparotomy 12-24 hrs later is mandatory

Appendix

Appendicitis

Epidemiology
- 6% of population, M>F
- 80% between: 5-35 years of age

Pathogenesis
- luminal obstruction → bacterial overgrowth → inflammation/swelling → increased pressure
  → localized ischemia → gangrene/perforation → localized abscess (walled off by omentum) or peritonitis
- etiology:
  - children or young adults: hyperplasia of lymphoid follicles, initiated by infection
  - adult: fibrosis/stricture, fecolith, obstructing neoplasm
  - other: causes: parasites, foreign body

Clinical Features
- most reliable feature is progression of signs and symptoms
- low grade fever (38°C), rises if perforation
- abdominal pain then anorexia, nausea and vomiting
- classic pattern: pain initially periumbilical; constant, dull, poorly localized, then well localized pain over McBurney’s point
  - due to progression of disease from visceral irritation (causing referred pain from structures of the embryonic midgut, including the appendix) to irritation of parietal structures
  - McBurney’s sign

Figure 13. Appendix Anatomy

McBurney’s Sign
Tenderness 1/3 the distance from the ASIS to the umbilicus on the right side.
Laparoscopic vs. Open Appendectomy

Laparoscopic Surgery
- intra-abdominal abscesses 3 times more likely
- Mean length of hospital stay reduced by 2.7 d
- Safer regime in normotensive, void, small
- Costs outside hospital are reduced
- Reduced levels of pain on POD #1
Open Surgery
- Wound infections 1 times as likely
- Lower operation costs

Overview
Diagnostic laparoscopy led to a large reduction in the rate of negative appendectomies, and a reduction in surgery with unconfirmed diagnosis. This was especially pronounced in the women due to a trend defined for appendicitis.


Aspiration versus Fluids for Prevention of Postoperative Infections after Appendectomy

Cochrane Database of Systematic Reviews 2008, 3: Study: Meta-analysis of Randomized Controlled Trials (RCTs) and Controlled Clinical Trials (CCTs), on adolescents and children, in which any antibiotic regime was compared to placebo in patients undergoing appendectomy for suspected appendicitis.

Data Sources: Cochrane Central Register of Controlled Trials (2005 issue 3), PubMed (1966 to April 2003), EMBASE (1980 to April 2003), Cochrane Gastrointestinal Interventions Group Specialised Register (April 2004) and references from included studies.

Patients: Visible infection, 26 studies (n=2331), Postoperative intra-abdominal abscess, 14 studies (n=1013).

Conclusion: (1) No infection (decrease of pus from the wound) and (2) Postoperative intra-abdominal abscess (persistent pain after 10 days, fever, unspecified degree of abdominal pain, or fever after the operation). Results. Treatment with antibiotics decreased infection rates with an NNT-10 = 1 (95%CI0-3), while antibiotic therapy decreased infection rates with an NNT = 51 (95%CI 36-99).

Various prophylactic antibiotic antibiotic therapies are effective in preventing postoperative complications. Further studies are required to determine the chief regimen.

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Tumours of the Appendix

CARCINOID TUMOURS (most common type)
- see Tumours of Small Intestines: Carcinoid, GS21
ADENOCARCINOMA
- 50% present as acute appendicitis
- spreads rapidly to lymph nodes, ovaries, and peritoneal surfaces
- treatment: right hemicolectomy

OTHER
- malignant mucinous cystadenocarcinoma

Inflammatory Bowel Disease (IBD)

- see Gastroenterology, G19

Principles of Surgical Management
- can alleviate symptoms, address complications, improve quality of life
- conserve bowel – respect as little as possible to avoid short gut syndrome
- perioperative management:
  - optimize medical status: may require TPN (especially if >7 days NPO) and bowel rest
  - hold immunosuppressive therapy pre-op, provide pre-op stress dose of corticosteroid if patient had recent steroid therapy
  - deep vein thrombosis (DVT) prophylaxis: heparin (IBD patients at increased risk of thromboembolic events)
Crohn's Disease

Treatment
- surgery is NOT curative, but over lifetime ~70% of Crohn's patients will have surgery
- indications for surgical management:
  - failure of medical management
  - stricture (due to stricture/inflammation); indication in 50% of surgical cases
  - abscess, fistula (enterocutaneous, vesicular, vaginal, cutaneous abscess), quality of life, perforation, hemorrhage, chronic disability, failure to thrive (children), perianal disease
- surgical procedures:
  - resection and anastomosis/stoma if active or subacute inflammation, perforation, fistula
  - resection margin only has to be free of gross disease (microscopic disease irrelevant to prognosis)
  - stricuroplasty - widens lumen in chronically scarred bowel - relieves obstruction without resecting bowel (contra-indicated in acute inflammation)

Complications of Treatment
- short gut syndrome (diarrhea, steatorrhea, malnutrition)
- fistulas
- gallstones (if terminal ileum resected, decreased bile salt resorption → increased cholesterol precipitation)
- kidney stones (loss of calcium in diarrhea → increased oxalate absorption and hyperoxaluria → stones)

Prognosis
- recurrence rate at 10 years: ileocaecal (25-50%), small bowel (30%), colonic (40-50%)
- re-operation at 5 years: primary resection (20%), bypass (50%), stricturoplasty (10% at 1 year)
- 80-85% of patients who need surgery lead normal lives
- mortality: 15% at 30 years

Ulcerative Colitis

Treatment
- indications for surgical management:
  - failure of medical management (including inability to taper steroids)
  - complications: hemorrhage, obstruction, perforation, toxic megacolon (emergency), failure to thrive (children)
  - reduce cancer risk (1-3% risk per year after 10 years of disease)
- surgical procedures:
  - proctocolectomy and ileal pouch-anal anastomosis (IPAA) ± rectal mucosectomy (operation of choice)
  - proctocolectomy with permanent end ileostomy (if not a candidate for ileoanal procedures)
  - colectomy and IPAA ± rectal mucosectomy
  - in emergency: total colectomy and ileostomy with Hartmann closure of the rectum, rectal preservation

Complications of Treatment
- early: bowel obstruction, transient urinary dysfunction, dehydration (high stools output), anastomotic leak
- late: stricture, anal fistula/abscess, pouchitis, poor anorectal function, reduced fertility

Prognosis
- mortality: 5% over 10 years
- total proctocolectomy will completely eliminate risk of cancer
- perforation of the colon is the leading cause of death in ulcerative colitis
Diverticular Disease

Definitions
- diverticulum – abnormal sac or pouch protruding from the wall of a hollow organ
- diverticulosis – presence of multiple false diverticuli
- diverticulitis – inflammation of diverticuli
- right sided (true) diverticuli – contains all layers (congenital) (see Figure 14)
- left sided (false) diverticuli = contains only mucosal and submucosal layers (acquired)

![Figure 14. Diverticular Disease – Cross-Sections of True and False Diverticuli](image)

Diverticulosis

Epidemiology
- 25-50% of general population, M:F
- increased incidence in 5th to 7th decades of life
- 95% involve sigmoid colon (site of highest pressure)
- higher incidence in Western countries, related to low fibre diet

Pathogenesis
- risk factors:
  - low-fibre diet (increases gut transit time and intraluminal pressure)
  - muscle wall weakness from aging and illness (e.g., Ehlers-Danlos, Marfan’s)
  - possible genetic component
  - high intraluminal pressures cause outpouching to occur at area of greatest weakness; most commonly at the site of penetrating vessels at antimesenteric tenia, therefore increased risk of hemorrhage

Clinical Features
- uncomplicated diverticulosis: asymptomatic (70-80%)
- episodic LLQ abdominal pain, bloating, flatulence, constipation, diarrhea
- absence of fever/leukocytosis
- no physical exam findings or poorly localized LLQ tenderness
- complications:
  - diverticulitis (15-20%)
  - bleeding (5-15%): PAINLESS rectal bleeding, 2/3 of massive lower GI bleeds

Treatment
- uncomplicated diverticulosis: high fibre, education
- diverticular bleed:
  - initially work up and treat as any lower GI bleed
  - if hemorrhage does not stop, resect involved region

Diverticulitis (“left sided appendicitis”)

Definition
- Infection or perforation of a diverticulum

Pathogenesis
- erosion of the wall by increased intraluminal pressure (or inspissated food particles)  → microperforation/macropereforation  → inflammation and focal necrosis
- usually mild inflammation with perforation walled off by pericolic fat
- sigmoid colon most often involved
Clinical Features
- Severity ranges from mild inflammation to feculent peritonitis
- LLQ pain/tenderness, present for several days before admission
- Altering constipation and diarrhea, urinary symptoms (dysuria if inflammation adjacent to bladder)
- Palpable mass if phlegmon or abscess, nausea, vomiting
- Low-grade fever, mild leukocytosis
- Occult or gross blood in stool less common
- Generalized tenderness suggests macroperforation and peritonitis
- Complications:
  - Abscess - on physical exam may find palpable abdominal mass
  - Fistula - colocelestial (most common), coloenteric, colovesical, colocolonic
  - Obstruction - due to scarring from repeated inflammation
- Macroperforation → peritonitis (feculent vs. purulent)
  * Recurrent attacks RARELY lead to peritonitis

Investigations
- AXR, upright CXR:
  - Localised diverticulitis (ileus, thickened wall, SBO, partial colonic obstruction)
  - Free air may be seen in 30% with perforation and generalized peritonitis
- CT scan (optimal method of investigation):
  - 97% sensitive, very useful for assessment of severity and prognosis
  - Very helpful in localizing an abscess
- Hypeaque (water soluble) enema - SAFE (under low pressure):
  - Saw-tooth pattern (colonic spasm)
  - May show site of perforation, abscess cavities or sinus tracts, fistulas
- Barium enema: contraindicated during an acute attack:
  - Risk of chemical peritonitis (because of perforation)
- Sigmoidoscopy/colonoscopy:
  - Not during an acute attack, only done on an elective basis
  - Take biopsies to rule out other diagnoses (polyps, malignancy)

Treatment
- Sdnh, NPO, fluid resuscitation, NG + suction, IV antibiotics covering B. fragilis (e.g. ciprofloxacin, metronidazole)
- Indications for surgery:
  - Unstable patient with peritonitis
  - Hinchey stage 2-4 (see Table 10)
  - After 1 attack if (a) immunosuppressed, (b) abscess needing percutaneous drainage
  - Consider after 2 or more attacks, recent trend is toward conservative management of recurrent mild/moderate attacks
  - Complications: generalized peritonitis, free air, abscess, fistula, obstruction, hemorrhage, inability to rule out colon cancer on endoscopy, or failure of medical management
- Surgical procedures:
  - Hartmann procedure: resection + colostomy and rectal stump → colostomy reversal in 3-6 months (see Figure 15)
  - Resection + primary anastomosis (2 - pre-op bowel prep or on-table lavage); controversial
    - Anastomosis of inflamed tissues = increased risk of anastomotic leakage

Prognosis
- 13-30% recurrence after 1st attack, 30-50% after 2nd attack

Table 10. Hinchey Staging and Treatment for Diverticulitis

<table>
<thead>
<tr>
<th>Hinchey Stage</th>
<th>Description</th>
<th>Acute treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pylephlegm / small perioid abscess</td>
<td>Medical</td>
</tr>
<tr>
<td>2</td>
<td>Large abscess / fistula</td>
<td>Abscess drainage, resection or primary anastomosis</td>
</tr>
<tr>
<td>3</td>
<td>Perforated peritonitis (ruptured abscess)</td>
<td>Hartmann procedure, sometimes primary anastomosis</td>
</tr>
<tr>
<td>4</td>
<td>Feculent peritonitis</td>
<td>Hartmann procedure</td>
</tr>
</tbody>
</table>
Colorectal Neoplasms

Colorectal Screening Guidelines

- Men and Women
- Symptomatic
- Diagnostic Work-up

Asymptomatic regardless of age but positive family history

- HNPCC or FAP
- Genetic counseling and special screening
  - HNPCC
    - Colonoscopy every 1-2 years
    - Begin at age 20 or 10 years younger than the earliest age in the family, whichever comes first
  - FAP
    - Sigmoidoscopy annually
    - Begin at age 10-12
  - APC
    - Colonoscopy annually
    - Begin at age 16-18

- One first-degree relative with cancer or adenomatosus polyp at age < 50 or two or more second-degree relatives with polyp or colon cancer at any age
  - Colonoscopy every 5 years
  - Begin at age 40 or 10 years younger than the earliest age of polyp or cancer in the family, whichever comes first

- One first-degree relative with cancer or adenomatosus polyp affected at age > 60 or two or more second-degree relatives with polyps or cancer
  - Average risk screening
  - Begin at age 50

- One second-degree relative or third-degree relative affected
  - Polyps found at colonoscopy
  - 1-2 tubular adenomas < 1 cm: colonoscopy in 5 years
  - ≥2 adenomas: colonoscopy in 3 years
  - Incrimate examination, numerous polyps, advanced adenoma, malignant or large sessile adenoma: colonoscopy after a short interval based on clinical judgement

Figure 16. Approach to Higher Risk Screening

AFC (hereditary adenomatous polyposis); FAP (familial adenomatous polyposis); HNPCC (hereditary nonpolyposis colorectal cancer); First degree relatives: parents, siblings, children; second degree relatives: grandparents, aunts, uncles, 3rd degree relatives: great grandparents or cousins.


Colorectal Polyps

Definition
- Polyp: small mucosal outgrowth into the lumen of the colon or rectum
- Sessile (flat) or pedunculated (on a stalk) (see Figure 17)

Epidemiology
- 30% of population have polyps by age 50, 40% by age 60, 50% by age 70

<table>
<thead>
<tr>
<th>Table 11. Characteristics of Tubular vs. Villous Polyps</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tubular</strong></td>
</tr>
<tr>
<td>Incidence</td>
</tr>
<tr>
<td>Size</td>
</tr>
<tr>
<td>Attachment</td>
</tr>
<tr>
<td>Malignant Potential</td>
</tr>
<tr>
<td>Distribution</td>
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</table>

Clinical Features
- 50% in the rectosigmoid region, 50% are multiple
- Usually asymptomatic, but may have rectal bleeding, change in bowel habit, mucus per rectum
- Usually detected during routine endoscopy or familial/high risk screening

Pathology
- Non-neoplastic:
  - Hyperplastic – most common non-neoplastic polyp
  - Pseudopoly pard – inflammatory, associated with IBD, no malignant potential

Figure 17. Sessile and Pedunculated Polyps
• neoplastic:
  - hemirrhagia: juvenile polyps (large bowel), Peutz-Jeghers syndrome (small bowel)
  - malignant risk due to associated adenomas (large bowel)
  - low malignant potential → most spontaneously regress or autolocate
  - adenomas – premalignant, often carcinoma in situ:
    - some may contain invasive carcinoma ("malignant polyp" – 3-9%); invasion into muscularis
    - malignant potential: villous > tubulovillous > tubular (see Table 11)

Investigations
• flexible sigmoidoscopy can reach 60% of polyps in men and 35% of polyps in women; if polyps detected, proceed to colonoscopy for examination of entire bowel and biopsy
• colonoscopy still the gold standard

Treatment
• indications: symptoms, malignancy or risk of malignancy (i.e., adenomatous polyps)
• endoscopic removal of entire growth
• surgical resection for those invading into muscularis (high risk of malignancy) and those too large to remove endoscopically
• follow-up endoscopy 1 year later, then every 3-5 years

Familial Colon Cancer Syndromes

FAMILIAL ADENOMATOUS POLYPOSIS (FAP)

Pathogenesis
• autosomal dominant (AD) inheritance, mutation in adenomatous polyposis coli (APC) gene on chromosome 5q

Clinical Features
• hundreds to thousands of colorectal adenomas usually by age 20 (by 40’s in attenuated FAP)
• extracolonic manifestations:
  - carcinoma of duodenum, bile duct, pancreas, stomach, thyroid, adrenal, small bowel
  - congenital hypertrophy of retinal pigment epithelium presents early in life in 2/3 of patients
• virtually 100% lifetime risk of colon cancer (because of number of polyps)
• variants:
  - Gardner's syndrome: FAP + extraintestinal lesions (sebaceous cysts, osteomas, desmoid tumours)
  - Turcot's syndrome: FAP + CNS tumours (glioblastoma multiforme)

Investigations
• genetic testing (80-95% sensitive, 99-100% specific) (see sidebar)
• if no polyps found: annual flexible sigmoidoscopy from puberty to age 50, then routine screening
• if polyps found: annual colonoscopy and consider surgery (see Figure 16)

Treatment
• surgery indicated by age 17-20
• total proctocolectomy with ileostomy OR total colectomy with ileorectal anastomosis
• doxorubicin-based chemotherapy
• NSAIDS for intra-abdominal desmoid

HEREDITARY NON-POLYPOSISS COLORECTAL CANCER (HNPCC)

Pathogenesis
• AD inheritance, mutation in a DNA mismatch repair gene resulting in genomic instability and subsequent mutations

Clinical Features
• early age of onset, right > left colon, synchronous and metachronous lesions
• mean age of cancer presentation is 44 years, lifetime risk 70-80% (M>F)
• Lynch syndrome II: hereditary site-specific colon cancer
• Lynch syndrome II: cancer family syndrome – high rates of extracolonic tumours
  (endometrial, ovarian, hepatobiliary, small bowel)

Diagnosis
• diagnosis is clinical – based on Amsterdam Criteria:
  - at least 3 relatives with colorectal cancer or HNPCC related CA
  - 2 or more generations involved, and 1 must be 1st degree relative of the other 2
  - 1 case must be diagnosed before 50 years old
• FAP is excluded

Referral Criteria for Genetic Screening for APC
• To confirm the diagnosis of FAP
  (in patients with ≥100 colorectal adenomas)
• To provide pre-symptomatic testing
  for individuals at risk for FAP (1st degree relatives who are ≥10 years old)
• To confirm the diagnosis of attenuated
  FAP (in patients with ≥20 colorectal adenomas)
Investigations
- genetic testing (90% sensitive) - colonoscopy mandatory even if negative
- refer for genetic screening individuals who fulfill EITHER the Amsterdam Criteria (as above) OR the revised Bethesda Criteria (see sidebar)
- colonoscopy (starting age 20) annually
- surveillance for extracolonic lesions

Treatment
- total colectomy and ileorectal anastomosis with yearly proctoscopy

Colorectal Carcinoma (CRC)

Epidemiology
- 3rd most common cancer (after lung, prostate/breast), 2nd most common cause of cancer death

Risk Factors
- most patients have no specific risk factors
- FAP, HNPCC, family history of CRC
- adenomatous polyps (especially if >1 cm, villous, multiple)
- age >50 (dominant risk factor in sporadic cases), mean age is 70
- IBD (especially UC risk is 1.2x; if UC >10 yrs)
- previous colorectal cancer (also gonadal or breast)
- diet (increased fat, red meat, decreased fibre) and smoking
- diabetes mellitus (insulin is a growth factor for colon mucosal cells) and acromegaly

Screening Tools
- digital rectal exam (DRE): most common exam, but not recommended as a screening tool
- fecal occult blood test (FOBT):
  - proper test requires 3 samples of stool collected at 3 different times
  - recommended annually by the World Health Organization (WHO)
  - results in 16-33% reduction in mortality in RCTs
- Minnesota Colon Cancer Study: RCT showed that annual FOBT can decrease mortality rate by 1/3 in patients 50-80 years old
- sigmoidoscopy:
  - can identify 30-60% of lesions
  - sigmoidoscopy + FOBT misses 24% of colonic neoplasms
- colonoscopy:
  - can remove or biopsy lesions during procedure
  - can identify proximal lesions missed by sigmoidoscopy
  - used as follow-up to other tests if lesions found
  - disadvantages: expensive, not always available, poor compliance, requires sedation, risk of perforation (0.2%)  
  - virtual colonoscopy (CT colonography): 91% sensitive, 17% false positive rate
  - air contrast barium enema (ACBE): 50% sensitive for large (>1 cm) adenomas, 39% for polyps

Pathogenesis
- adenoma-carcinoma sequence: rarely arise de novo

Clinical Features (see Table 12)
- often asymptomatic
- hematochezia/melena, abdominal pain, change in bowel habits
- others weakness, anaemia, weight loss, palpable mass, obstruction
- 3-5% have synchronous lesions

spread:
- direct extension, lymphatic, hematogenous (liver most common, lung, rarely bone and brain)
- peritoneal seeding: every, Blumer's shelf (pelvic cul-de-sac)
- intraluminal

Table 12. Clinical Presentation of CRC

<table>
<thead>
<tr>
<th></th>
<th>Right Colon</th>
<th>Left Colon</th>
<th>Rectum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>22%</td>
<td>35%</td>
<td>33%</td>
</tr>
<tr>
<td>Pathology</td>
<td>Exophytic lesions with occult bleeding</td>
<td>Annular, invasive lesions</td>
<td>Ulcerating</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Weight loss, weakness, rarely obstruction</td>
<td>Constipation + overflow (alternating bowel pattern), abdominal pain, decreased stool caliber, rectal bleeding</td>
<td>Obstruction, tenesmus, rectal bleeding</td>
</tr>
<tr>
<td>Signs</td>
<td>Fe-deficiency anemia, RLO mass (10%)</td>
<td>BRBPR, LEO</td>
<td>Palpable mass on DRE, BRBPR</td>
</tr>
</tbody>
</table>
Investigations
- colonoscopy (best), look for synchronous lesions; alternative: air contrast barium enema (*apple core* lesion) + sigmoidoscopy
- if a patient is POBT +ve, has microcytic anemia or has a change in bowel habits, do colonoscopy
- metastatic workup: CXR, abdominal CT/ultrasound
- bone scan, CT head only if lesions suspected
- labs: CBC, urinalysis, liver function tests, CEA (before surgery for baseline)
- staging (see Table 13 and sidebar)
- rectal cancer: pelvic MRI or endorectal ultrasound to determine T and N stage

<table>
<thead>
<tr>
<th>Table 12. TNM Classification System for Staging of Colorectal Carcinoma</th>
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<tbody>
<tr>
<td><strong>Primary Tumour (T)</strong></td>
</tr>
<tr>
<td>T0 Less than 2 cm</td>
</tr>
<tr>
<td>Tis Carcinoma in situ</td>
</tr>
<tr>
<td>T1 Invasion into submucosa</td>
</tr>
<tr>
<td>T2 Invasion into muscular propria</td>
</tr>
<tr>
<td>T3 Invasion through muscular and into serosa</td>
</tr>
<tr>
<td>T4 Invasion into adjacent structures or organs</td>
</tr>
</tbody>
</table>

Treatment
- surgery (indicated in potentially curable or symptomatic cases – not usually in stage IV)
  - curative: wide resection of lesion (5 cm margins) with nodes and mesentery
  - palliative: if distant spread, then local control for hemorrhage or obstruction
  - 80% of recurrences occur within 2 years of resection
  - improved survival if metastasis consists of solitary hepatic mass that is resected
  - colectomy:
    + most patients get primary anastomosis [e.g. hemicolectomy, low anterior resection (LAR)] (see Figure 18)
    + if cancer is low in rectum, patient may require an abdominoperineal resection (APR) with a permanent end colostomy, especially if lesion involves the sphincter complex
    + complications: anastomotic leak or stricture, recurrent disease, pelvic abscess, enterocutaneous fistula
- radiotherapy and chemotherapy:
  - chemotherapy (5-FU based regimen) for patients with node-positive disease
  - radiation: for patients with node-positive or transmural rectal cancer (pre-op or post-op), not effective as 1st treatment of colon cancer
  - adjuvant therapy: chemotherapy (colorectal) and radiation (rectum)
  - palliative chemotherapy/radiation therapy for improvement in symptoms and survival
  - neoadjuvant chemotherapy for T3 or T4 rectal cancer

Case Finding for Colorectal Cancer (symptomatic or history of UC, polyps, or CRC)
- surveillance (when polyps are found): colonoscopy within 3 years after initial finding
- patients with past CRC: colonoscopy every 3-5 years, or more frequently
- IBD: some recommend colonoscopy every 1-2 years after 8 years of disease (especially UC)

Follow-Up
- intensive follow-up improves overall survival in low risk patients
- currently there are no data suggesting optimal follow-up
- combination of periodic CT chest/abdomen/pelvis, CEA and colonoscopy is recommended
- carcinoembryonic antigen (CEA): to monitor for initial response to treatment, and to assess for recurrence q3 months (not a screening test)
Other Conditions of the Large Intestine

Angiodysplasia

Definition
vascular anomaly: focal submucosal venous dilatation and tortuosity

Clinical Features
- most frequently in right colon of patients >60 years old
- bleeding typically intermittent (melena, anemia, guaiac positive stools)

Investigations
- endoscopy (cherry red spots, branching pattern from central vessel)
- angiography (slow filling/early emptying mesenteric vein, vascular tuft)
- RBC technetium-99 scan
- barium enema is contraindicated (obscures other x-rays, i.e. angiogram)

Treatment
- none if asymptomatic
- cautery, right hemicolectomy, embolization, vasopressin infusion, sclerotherapy, band ligation, laser, octreotide, and rarely segmental resection if other treatments fail

Volvulus

Definition
- rotation of segment of bowel about its mesenteric axis
  - sigmoid (70%), cecum (30%)

Risk Factors
- age (50% of patients >70 yrs; stretching/elongation of bowel with age is a predisposing factor)
- high fibre diet (can cause elongated/redundant colon), chronic constipation, laxative abuse, pregnancy, bedridden, institutionalized (less frequent evacuation of bowels)
- congenitally hypermobile cecum

Clinical Features
- symptoms due to bowel obstruction (GS13) or intestinal ischemia (GS27)

Investigations
- AXR: “omegas”, “bent inner tube”, “coffee-bean” signs (see sidebar)
- barium/gastrografin enema: “ace of spades” (or “bird’s beak”) appearance due to funnel-like luminal tapering of lower segment towards volvulus
- sigmoidoscopy or colonoscopy as appropriate
- CT

Treatment
- initial supportive management with fluid, electrolyte resuscitation
  - cecum:
    - nonsurgical
      - may attempt colonoscopic detorsion and decompression
    - surgical
      - right colectomy + ileocecal anastomosis
  - sigmoid:
    - nonsurgical
      - decompression by flexible sigmoidoscopy and insertion of rectal tube past obstruction
    - subsequent elective surgery recommended (50-70% recurrence)
    - surgical: Hartmann procedure (U’urgent)
      - indications: strangulation, perforation or unsuccessful endoscopic decompression
Fistula

Definition
- Abnormal communication between two epithelialized surfaces (e.g., enterocutaneous, colovesical, aortovesical, enterointestinal)

Etiology
- Foreign object erosion (e.g., gallstone, graft)
- Infection, IBD (especially Crohn's), diverticular disease
- Iatrogenic/surgery (e.g., postoperative anastomotic leak)
- Congenital, trauma
- Neoplastic

Investigations
- Contrast radiography (fistulogram)
- Sonogram
- CT scan
- Measure amount of drainage from fistula

Treatment
- Fluid resuscitation, manage electrolytes
- Bowel rest - NPO
- Drain any abscesses/control sepsis
- Nutrition - elemental/low residue, TPN
- Decrease secretions - octreotide/somatostatin/omnipaque
- Skin care (for enterocutaneous fistula)
- Surgical intervention - dependent upon etiology (for non-closing fistulas); uncertainty of diagnosis

Ostomies

Definition
- An opening of the GI tract onto the surface of the abdomen wall
- Types (see Figure 19): colostomy vs. ileostomy, temporary vs. permanent, continent vs. incontinent, end vs. loop, ileoileostomy
  - End (Brooke) ileostomy: for incontinent, continuous drainage in patients requiring total colectomy
  - Koch ileostomy: for continent, manual drainage – rarely used

Complications (10%)
- Obstruction: herniation, stenosis (skin and abdominal wall), adhesive bands, valvulas
- Peri-ileostomy abscess and fistula
- Skin irritation
- Prolapse or retraction
- Diarrhea (excessive output)

![Figure 19. Ostomies](image-url)
Anorectum

Hemorrhoids

Etiology
- vascular and connective tissue complexes form a plexus of dilated veins (cushion)
  - internal: superior hemorrhoidal veins, above dentate line, portal circulation
  - external: inferior hemorrhoidal veins, below dentate line, systemic circulation

Risk Factors
- Increased intra-abdominal pressure: chronic constipation, pregnancy, obesity, portal hypertension, heavy lifting

Clinical Features and Treatment
- Internal hemorrhoids (see Figure 20):
  - engorged vascular cushions usually at 3, 7, 11 o'clock positions (patient in lithotomy position)
  - painless rectal bleeding, anemia, prolapse, mucus discharge, pruritus, burning pain, rectal fullness:
    - 1st degree: bleed but do not prolapse through the anus
      - treatment: high fibre/bulk diet, sitz baths, steroid cream, peroxine (Anusol*), rubber band ligation, sclerotherapy, photocoagulation
    - 2nd degree: prolapse with straining, spontaneous reduction
      - treatment: rubber band ligation, photocoagulation
    - 3rd degree: prolapse requiring manual reduction
      - treatment: same as 2nd degree, but may require closed hemorrhoidectomy
    - 4th degree: permanently prolapsed, cannot be manually reduced
      - treatment: closed hemorrhoidectomy
  - external hemorrhoids (see Figure 20):
    - dilated veins usually mildly symptomatic
    - pain after bowel movements, associated with poor hygiene
    - medical treatment: dietary fibre, stool softeners, steroid cream (short course), peroxine (Anusol*), avoid prolonged straining
    - thrombosed hemorrhoids are very painful:
      - resolve within 2 weeks, may leave excess skin = perineal skin tag
      - treatment: consider surgical decompression within first 48 hours of thrombosis, otherwise medical treatment

Anal Fissures

Definition
- tear of anal canal below dentate line (very sensitive squamous epithelium)
  - 90% posterior midline, 10% anterior midline
  - if off midline: consider IBD, STIs, TB, leukemia or anal carcinoma
  - repetitive injury cycle after first tear:
    - sphincter spasm occurs preventing edges from healing and leads to further tearing
    - ischemia may ensue and contribute to chronicity

Etiology
- large, hard stools and irritant diarrheal stools
- tightening of anal canal secondary to nervousness/pain
- others: habitual use of cathartics, childbirth

Clinical Features
- acute fissure:
  - very painful bright red bleeding especially after bowel movement
  - treatment: is conservative: stool softeners, sitz baths
- chronic fissure:
  - trial: fissure, sentinel skin tags, hypertrophied papillae
  - treatment:
    - stool softeners, bulking agents, sitz baths
    - topical nitroglycerin or nitidipine – increases local blood flow, promoting healing and relieves sphincter spasm
  - surgery (most effective) – lateral internal sphincterotomy; objective is to relieve sphincter spasm → increases blood flow and promotes healing; but 5% chance of fecal incontinence therefore not commonly done
  - alternative treatment:
    - botulinum toxin – inhibits release of acetylcholine (ACh), reducing sphincter spasm
Anorectal Abscess

Definition
- Infection in one or more of the anal spaces (see Figure 21)
- Usually bacterial infection of blocked anal gland at the dentate line
  - *E. coli*, *Proteus*, *Streptococci*, *Staphylococci*, *Bacteroides*, anaerobes

![Diagram of normal and inflamed anal spaces]

Figure 21. Different Types of Perianal Abscesses

Clinical Features
- Throbbing pain that may worsen with straining and ambulation
- Abscess can spread vertically downward (perianal), vertically upward (supralevator) or horizontally (ischiorectal)
- Tender perianal/rectal mass on exam

Treatment
- Incision and drainage
  - CURATIVE in 50% of cases
  - 50% develop anorectal fistulas
  - May require antibiotics if diabetic, heart murmur or cellulitis

Fistula-In-Ano

Definition
- Connection between two epithelialized surfaces, one must be the rectum or anus
- An inflammatory tract with internal or at dentate line, external or on skin

Etiology
- See Fistula, GS37
- Same perianal process as anal abscess therefore usually associated with abscess
- Other causes: post-op, trauma, anal fissure, malignancy, radiation proctitis

Clinical Features
- Intermittent or constant purulent discharge from perianal opening
- Pain
- Palpable cord-like tract

Treatment
- Identification:
  - Internal opening:
    - Goodall’s rule (see Figure 22):
      - A fistula with an external opening anterior to the transverse anal line will have its internal opening at relatively the same position (e.g., external opening at 2 o'clock = internal opening at 2 o'clock) whereas all external openings posterior to the line will tend to have their internal openings in the midline.
  - Fistulous tract:
    - Probing or fistulography under anesthesia

![Diagram of Goodall’s Rule]

Figure 22. Goodall’s Rule
- surgery:
  - fistulotomy: unroof tract from external to internal opening, allow drainage
  - low lying fistula (does not involve external sphincter) → primary fistulotomy
  - high lying fistula (involves external sphincter) → staged fistulotomy with Seton suture placed through tract:
    - promotes drainage
    - promotes fibrosis and decreases incidence of incontinence
    - delineates anatomy
    - usually done to spare muscle cutting

Postoperative
- sitz baths, irrigation and packing to ensure healing proceeds from inside to outside

Complications
- recurrence
- rarely fecal incontinence

Pilonidal Disease

Definition
- acute abscess or chronic draining sinus in sacrococcygeal area

Epidemiology
- occurs most frequently in young men age 15-40 yrs

Etiology
- obstruction of the hair follicles in this area → formation of cysts, sinuses or abscesses

Clinical Features
- asymptomatic until acutely infected, then pain/tenderness, purulent discharge

Treatment
- acute abscess:
  - incision and drainage
  - wound packed open
  - 40% develop chronic pilonidal sinuses
- chronic disease:
  - pilonidal cystotomy
  - excision of sinus tract and cyst: + marsupialization (cyst edge sewn to surrounding tissue to leave sinus tract open)

Rectal Prolapse

Definition
- protrusion of full thickness of rectum through anus

Epidemiology
- extremes of ages – <5 years old and >5th decade
- 85% women

Etiology
- lengthened attachment of rectum secondary to constant straining
  - 3 types:
    I – false/mucosal: redundant rectal mucosa, radial furrows
    II – incomplete: rectal intussusception without sliding hernia
    III – true-complete (most common) (see Figure 23):
      - protrusion of entire rectal wall through anal orifice with herniation of pelvic peritoneum/cal-5e-sac
      - circular furrows

Risk Factors
- gynecological surgery
- chronic neurologic/psychiatric disorders affecting motility

Clinical Features
- extrusion of mass with increased intra-abdominal pressure:
  - straining, coughing, laughing, Valsalva
- difficulty in bowel regulation:
  - tenesmus, constipation, fecal incontinence
- permanently extruded rectum with excoriating, ulceration and constant soiling
- may be associated with urinary incontinence or uterine prolapse
**Treatment**
- Types I and II (false/mucosal/incipient):
  - conservative—gently reposition of prolapsed area, especially in children
  - hemorrhoidectomy with excision of redundant mucosa, mostly in adults
- Type III (true/completed):
  - conservative—reduce if possible
  - surgery—abdominal, perineal, transsacral approaches

**Anal Neoplasms**

**ANAL CANAL**

Squamous Cell Carcinoma (SCC) of Anal Canal (above dentate line)
- most common tumour of anal canal (75%)
- anus prone to human papilloma virus (HPV) infection, therefore at risk for anal squamous intraepithelial lesions (ASIL)
- high grade squamous intraepithelial lesion (HSIL) and low grade squamous intraepithelial lesion (LSIL) terminology used
- clinical features: anal pain, bleeding, mass, ulceration
- treatment: chemotheraphy + radiation + surgery
- prognosis: 80% 5-year survival

Malignant Melanoma (MM) of Anal Canal
- 3rd most common site for primary MM after skin, eyes
- aggressive, distant metastases common at time of diagnosis
- treatment: early radical surgery
- prognosis: <5% 5-year survival

**ANAL MARGIN**
- clinical features and treatment as for skin tumours elsewhere
- squamous and basal cell carcinoma, Bowen's disease (SCC in situ) and Paget's disease

**Liver**

![Liver Anatomy Diagram](image)

**Liver Cysts**

**SIMPLE CYSTS**
- most common type of liver cyst, may have multiple simple cysts
- clinical features: usually asymptomatic, if large may present with pain or mass
- treatment: generally not required for simple cysts unless very large
- complications: intracystic hemorrhage (may be confused with complex cysts)

**POLYCYSTIC LIVER DISEASE**
- progressive condition where cysts replace much of the liver
- 50% associated with polycystic kidney disease
- treatment: if symptomatic treat by partial liver resection or by creating drainage for cysts
CHOLEDODCHAL CYSTS
- congenital malformations of pancreaticobiliary tree
- 4 types, most extreme form called Caroli's disease (multiple cystic dilations in intrahepatic ducts)
- clinical features: recurrent abdominal pain, intermittent jaundice, RUQ mass
- diagnosis: U/S, transhepatic cholangiography, LFTs
- treatment:
  - high risk of malignancy, current treatment is complete excision of cysts
  - extent of resection depends on type of cyst
  - liver transplant indicated if cyst involves intrahepatic bile ducts (Caroli's disease)
- complications of chronic choledochal cyst: biliary cirrhosis, portal hypertension, cholangiocarcinoma

HYDATID LIVER CYSTS (CYSTIC ECHINOCOCOSIS)
- etiology:
  - infection with parasite Echinococcus granulosus
  - endemic to Southern Europe, Middle East, Australasia, South America
  - associated with exposure to dogs, sheep and cattle
- clinical features:
  - asymptomatic mass (most often) or chronic pain, hepatomegaly
  - rupture can cause biliary colic, jaundice or anaphylactic reaction
- investigations:
  - detection of anti-Echinococcus Ab (IgG) using ELISA or RIA
  - U/S, CT: presence of mass, often calcified
  - DO NOT perform needle biopsy as can cause seeding of abdominal cavity or anaphylaxis
- treatment:
  - medical: albendazole (anti-helminthic) – cure up to 30%
  - surgical (risk of spillage into abdomen):
    - conservative – open embolisation ± omentoplasty
    - radical – partial hepatectomy or total pericystectomy

CYSTADENOMA (PREMALIGNANT)/CYSTADENOCARICINOMA
- clinical features:
  - appear as complex cysts on imaging: internal septae, papillary projections, irregular lining
  - all complex, multiloculated cysts (except echinococcal) should be excised because of malignancy risk

Liver Abscesses

Etiology
- types:
  - pyogenic (bacterial): most often Gram-negatives – E. coli, Klebsiella, Proteus
  - parasitic (amoebic): Entamoeba histolytica
  - fungal
- sources: direct spread from biliary tract infection, portal spread from GI infection, systemic infection (e.g. endocarditis)

Clinical Features
- fever, malaise, chills, anorexia, weight loss, abdominal pain, nausea
- RUQ tenderness, hepatomegaly, jaundice, dullness to percussion

Investigations
- leucocytosis, anemia, elevated liver enzymes, hemaggutination titres for Entamoeba antibodies
- U/S, CXR (right basilar atelectasis/effusion), CT

Treatment
- treat underlying cause
- surgical or percutaneous drainage and IV antibiotics

Prognosis
- overall mortality 15% – higher rate if delay in diagnosis, multiple abscesses, malnutrition

Neoplasms

BENIGN LIVER NEOPLASMS

Hemangioma (cavernous)
- pathogenesis: most common benign hepatic tumour; results from malformation of angioblastic fetal tissue
- risk factors: E1M = 6c, steroid therapy, estrogen (exogenous, pregnancy)
- clinical features:
  - usually small and asymptomatic, larger tumours may produce pain or compress nearby structures
  - shock if ruptured (very rare)
investigations:
- contrast CT (well-demarcated hypodense mass with peripheral enhancement and delayed venous emptying), US (homogeneous hyperechoic mass), arteriography (rarely used; "cotton woof" appearance), RBC scan
- biopsy may result in hemorrhage

Treatment:
- usually none unless tumour bleeds or is symptomatic, then excision by lobectomy or enucleation

Adenoma
- definition: benign glandular epithelial tumour
- risk factors: female, age 30-50, estrogen (OC/ pregnancy)
- clinical features: asymptomatic, 25% present with RUQ pain or mass
- investigations: CT (well-demarcated masses, often heterogeneous, isoechoic on non-contrast CT, peripheral enhancement/isoechoic/hypodense on contrast CT), US (variable appearance; usually hyperechoic), biopsy
- treatment:
  - stop anabolic steroids or OCP
  - excise, especially if large (>5 cm), due to risk of malignancy and spontaneous rupture/hemorrhage

Focal Nodular Hyperplasia
- pathogenesis thought to be due to local ischemia and tissue regeneration
- risk factors: female, middle age
- clinical features: asymptomatic, rarely grows or bleeds, no malignant potential
- investigations: central stellate scar on CT scan, technetium-99 scan is helpful
- treatment: may be difficult to distinguish from adenoma (malignant potential) → often resected

MALIGNANT LIVER NEOPLASMS

Primary
- usually hepatocellular carcinoma (HCC)/hepatozelloma
- others include angiosarcoma, hepatoblastoma, hemangioblastoma
- epidemiology: uncommon in North America, but represents 20-25% of all carcinomas in Asia and Africa
- risk factors:
  - chronic liver inflammation: chronic hepatitis B (inherently oncogenic) and C, cirrhosis (especially macronodular), hemochromatosis, ß-anti-trypsin
  - meds: OCPs (3x increased risk), steroids
  - smoking, alcohol
  - chemical carcinogens (asbestos, vinyl chloride – associated with angiosarcoma)
- clinical features:
  - RUQ discomfort, right shoulder pain
  - jaundice, weakness, weight loss, ± fever
  - hepatomegaly, bruit, rub
  - ascites with blood (sudden intra-abdominal hemorrhage)
  - peripancreatic syndromes – e.g., Cushing's syndrome, hyperglycemia
  - metastases: lung, bone, brain, peritoneal seeding
- investigations:
  - elevated ALP, bilirubin, and ß-fetoprotein (80% of patients)
  - US (poorly-defined margins with internal echoes), triphasic CT (enhancement on arterial phase and washout on portal venous phase), MRI, CT or MRI angiography
  - biopsy
- treatment:
  - cirrhosis is a relative contraindication to tumour resection due to decreased hepatic reserve
  - surgical: resection (10% of patients have resectable tumours)
  - liver transplant (if cirrhosis plus solitary nodule < 5 cm, or less than 3 nodules each <3 cm (Milan criteria); generally not with extrahepatic disease or vascular invasion)
  - non-surgical: radiofrequency ablation, percutaneous ethanol injection, transcatheter arterial chemosembolization (TACE), chemotherapy (limited efficacy)
  - prognosis:
    - 70% have metastases to nodes and lung
    - survival without treatment: 3 months
    - 5 year survival: all patients – 5% patients undergoing complete resection – 11-40%

Secondary
- most common hepatic malignancy
- etiology:
  - GI (most common), lung, breast, pancreas, ovary, uterus, kidney, gallbladder, prostate
- treatment:
  - hepatic resection if control of primary is possible, no extrahepatic or extrapulmonary metastases and if possibility of "curative" resection
  - possible chemotherapy
- prognosis: 30-40% 5-year survival with a "curative" resection; prognosis same if metastases are multifocal compared to confined to one lobe
Liver Transplantation

<table>
<thead>
<tr>
<th>Parenchymal Disease</th>
<th>Cholangiologic Disease</th>
<th>Inborn Errors</th>
<th>Tumors</th>
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<tr>
<td>Chronic hepatitis B or C*</td>
<td>Biliary atresia**</td>
<td>α1-antitrypsin deficiency</td>
<td>Hepatoma</td>
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<tr>
<td>Alcoholic cirrhosis</td>
<td>Primary biliary cirrhosis</td>
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<tr>
<td>Acute liver failure</td>
<td>Sclerosing cholangitis</td>
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<td>Budd–Chiari syndrome</td>
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<td>Congenital hepatic fibrosis</td>
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<tr>
<td>Cystic fibrosis (CF)</td>
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</tbody>
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*leading cause in adults; **leading cause in children

Clinical Indications
- Early referral for transplant should be considered for all patients with progressive liver disease not responding to medical therapy, especially decompensated cirrhosis, unresectable primary liver cancers and fulminant hepatic failure
- End-stage liver disease with life expectancy <1 year and if no other therapy is appropriate
- Progressive jaundice, refractory ascites, spontaneous hepatic encephalopathy, recurrent sepsis, fulminant hepatic failure
- Recurrent variceal hemorrhage, coagulopathy, severe fatigue

Criteria for Transplantation
- Model for End-Stage Liver Disease (MELD): considers probability of death within 3 months if patient does not receive transplant; based on creatinine, bilirubin, INR
- Child–T ate-C Doyle Score: patient must have ≥7 points (Class B)

Contraindications
- Sepsis, HIV positive status
- Active alcohol/substance abuse
- Extravascular metastasis
- Advanced cardiopulmonary disease

Post-op Complications
- Primary non-function (graft failure) – urgent re-transplantation is indicated
- Acute and chronic rejection, ischemia-reperfusion injury
- Vascular – hepatic artery or portal vein thrombosis, IVC obstruction
- Biliary complications – fever, increasing bilirubin and ALP
- Recurrence of hepatitis B – prophylactic medical therapy is usually effective in preventing recurrence in graft; hepatitis C anti-recurrence therapy is less effective but recurrence can be controlled medically

Prognosis
- Patient survival at 1 year – 85%
- Graft survival at 1 year – 60–70%, at 5 years – 40–50%

Biliary Tract

Cholelithiasis

Definition
- The formation of gallstones (see Figure 25)

Pathogenesis
- Imbalance of cholesterol and its solubilizing agents (bile salts and lecithin)
- Excessive hepatic cholesterol secretion → bile salts and lecithin are "overloaded" → supersaturated cholesterol can precipitate and form gallstones
- North America: cholesterol stones (80%), pigment stones (20%)

Risk Factors
- Cholesterol stones:
  - Obesity, age <50
  - Estrogens: female, multiparity, OCs
  - Ethnicity: First Nations heritage > Caucasian > Black
  - Terminal ileal resection or disease (e.g., Crohn’s disease)
  - Impaired gallbladder emptying: starvation, TPN, DM type I
  - Rapid weight loss: rapid cholesterol mobilization and biliary stasis
- Pigment stones (contain calcium bilirubinate):
  - Cholecystitis
- Chronic biliary disease
- Biliary stasis (strictures, dilation, biliary infection)
Figure 26. Gallstones

Clinical Features
- asymptomatic (80%):
  - most do NOT require treatment
  - consider cholecystectomy if: porcelain (calcified) gallbladder (25% risk of malignancy), sickle cell disease, pediatric patient, having bariatric surgery, diabetes, immunosuppression
- biliary colic (10-25%)
- cholecystitis
- cholangitis
- gallstone pancreatitis (see Acute Pancreatitis, GS49)
- gallstone ileus

Investigations
- U/S – diagnostic procedure of choice:
  - Image for signs of inflammation, obstruction, localization of stones
  - ERCP (endoscopic retrograde cholangiopancreatography):
    - visualization of upper GI tract, ampullary region, biliary and pancreatic ducts
    - method for treatment of CBD stones in periampullary region
    - complications: traumatic pancreatitis (1-2%), pancreatic or biliary sepsis
  - MRC (magnetic resonance cholangiopancreatography):
    - same information gained as ERCP but non-invasive
    - cannot be used for therapeutic purposes
  - PTC (percutaneous transhepatic cholangiography):
    - injection of contrast via needle passed through hepatic parenchyma
    - useful for proximal bile duct lesions or when ERCP fails or not available
    - requires prophylactic antibiotics
    - contraindications: coagulopathy, ascites, perihepatic sepsis, disease of right lower lung or pleura
    - complications: bile peritonitis, chylothorax, pneumothorax, sepsis, hemobilia
  - HIDA scan (hepato-biliary iminodiacetate scan):
    - used less commonly
    - radiolabeled technetium-99 injected into a vein is excreted in high concentrations into bile, allowing visualization of the biliary tree
    - does not visualize stones, diagnosis by seeing occluded cystic duct or CBD

Biliary Colic

Pathogenesis
- gallstone transiently impacted in cystic duct, no infection

Clinical Features
- steady pain in epigastrium or RUQ for minutes to hours, crescendo-decrescendo pattern
- frequently occurs at night or after fatty meal
- may radiate to right shoulder or scapula
- patients often restless
- no peritoneal findings, no systemic signs
Acute Cholecystitis

Pathogenesis
- Inflammation of gallbladder resulting from sustained gallstone impaction in cystic duct or Hartmann's pouch
- No cholelithiasis in 5-16% (see Acalculous Cholecystitis, GS47)

Clinical Features
- Often have history of biliary colic
- Severe constant (hours to days) epigastric or RUQ pain, anorexia, nausea, vomiting, low grade fever (>38.5°C)
- Focal peritoneal findings: Murphy's sign, palpable, tender gallbladder (in 39%)
- Bb's sign: right subcostal pain

Investigation
- Bloodwork: elevated WBC and left shift, mildly elevated bilirubin, AST, ALT, ALP
- U/S:
  - 98% sensitive, consider HIDA scan if U/S negative
  - Features on U/S (5 signs):
    - Distended gallbladder
    - Pericholecystic fluid
    - Stone in cystic duct
    - Thickened gallbladder wall (>3 mm)
    - Sonographic Murphy's sign: maximum tenderness on inspiration when probe over gallbladder

Complications
- Gallbladder mucocelle (hydrops) – long term cystic duct obstruction results in mucous accumulation in gallbladder (clear fluid)
- Gangrene, perforation – result in abscess formation or peritonitis
- Empyema of gallbladder – supplicative cholecystitis, pus in gallbladder + sick patient
- Cholecystoenteric fistula, from repeated attacks of cholecystitis, can lead to gallstone ileus
- Emphysematous cholecystitis – bacterial gas present in gallbladder, wall or pericholecystic space (risk in diabetic patient)
- Mirizzi's syndrome – extra-luminal compression of CBD/CHD due to large stone in cystic duct

Treatment
- Admit, hydrate, NPO, NG tube (if persistent vomiting from associated ileus), analgesics once diagnosis is made
- Antibiotics:
  - E. coli, K. pneumoniae, Enterococcus and Clostridium account for >80% of infections
  - Amoxicillin + gentamicin OR Cipro + Flagyl®
- Cholecystectomy:
  - Early (within 72 hrs) vs. delayed (after 6 weeks)
  - Reduced mortality and morbidity
  - Early cholecystectomy preferred: shorter hospitalization and recovery time
  - Emergent OR indicated if high risk, e.g., emphysematous, diabetic patient
- Laparoscopic is standard of care (convert to open for complications or difficult case)
- Laparoscopic: reduced risk of wound infections, shorter hospital stay, reduced post-op pain, increased risk of bile duct injury
- Intra-operative cholangiography (IOC):
  - Indications: clarity bile duct anatomy, obstructive jaundice, history of biliary pancreatitis, small stones in gallbladder with wide cystic duct (>15 mm), single faceted stone in gallbladder, bilirubin >137 µmol/L
- Percutaneous cholecystostomy tube: critically ill or if general anesthetic contraindicated
Acalculous Cholecystitis

Definition
- acute or chronic cholecystitis in the absence of stones

Pathogenesis
- typically due to gallbladder stasis → sludge forms in gallbladder

Risk Factors
- DM, immunosuppression, ICU admission, trauma patient, TPN, sepsis

Clinical Features
- see Acute Cholecystitis, GS46
- occurs in 20% of cases of acute cholecystitis

Investigations
- U/S: shows sludge in gallbladder, other U/S features of cholecystitis (see Acute Cholecystitis, GS46)
- CT or HIDA scan

Treatment
- cholecystectomy
- if patient unstable → cholecystostomy

Choledocholithiasis

Definition
- stones in common bile duct (CBD)

Clinical Features
- 50% asymptomatic
- often have history of biliary colic
- tenderness in RUQ or epigastrium
- acholic stool, dark urine, fluctuating jaundice
- primary vs. secondary stones:
  - primary: formed in bile duct, indicates bile duct pathology (e.g. benign biliary stricture, sclerosing cholangitis, choledochal cyst)
  - secondary: formed in gallbladder (85% of cases in U.S.)

Investigations
- CBC: usually normal; leucocytosis suggests cholangitis
- LFTs: increased bilirubin, ALP
- amylase/lipase: to rule out gallstone pancreatitis
- U/S: intra/extra-hepatic duct dilatation
- ERCP, PTC
- MRCP (90% sensitive, almost 100% specific, not therapeutic)

Complications
- cholangitis, pancreatitis, biliary stricture and biliary cirrhosis

Treatment
- if no evidence of cholangitis treat with ERCP for CBD stone extraction possibly followed by elective cholecystectomy in 25% of patients

Acute Cholangitis

Pathogenesis
- obstruction of CBD leading to biliary stasis, bacterial overgrowth, suppuration and biliary sepsis

Etiology
- choledocholithiasis (60%), stricture, neoplasm (pancreatic or biliary), extrinsic compression (pancreatic pseudocyst or pancreatitis), instrumentation of bile ducts (PTC, ERCP), biliary stent
- organisms: E. coli, Klebsiella, Pseudomonas, Enterobacter, H. fragilis, Proteus

Clinical Features
- Charcot's triad – fever, RUQ pain, jaundice
- Reynolds' pentad – fever, RUQ pain, jaundice, shock, confusion
- may have nausea, vomiting, abdominal distension, ileus, acholic stools, tea-coloured urine

Investigations
- CBC: elevated WBC + left shift
- may have positive blood cultures
- LFTs: obstructive picture (elevated ALP and conjugated bilirubin, mild increase in AST, ALT)
- amylase/lipase: rule out pancreatitis
- U/S: intra/extra-hepatic duct dilatation
**Treatment**
- initial: NPO, fluid and electrolyte resuscitation, ± NG tube, IV antibiotics
- decompression:
  - ERCP + sphincterotomy – diagnostic and therapeutic
  - PTC with catheter drainage – if ERCP not available or unsuccessful
- laparotomy with CBD exploration and T-tube placement if above fails
- all patients should also have a cholecystectomy, unless contraindicated

**Prognosis**
- suppurative cholangitis – mortality rate 50%

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**Gallstone Ileus**

**Pathogenesis**
- repeated inflammation causing a cholecystoenteric fistula (usually duodenal) → large gallstone enters the gut and impacts at or near the ileocecal valve, causing a true bowel obstruction (note: ileus is a misnomer in this context)

**Clinical Features**
- crampy abdominal pain, nausea, vomiting (see Bowel Obstruction, GS23)

**Investigations**
- AXR: dilated small intestine, air fluid levels, may reveal radiopaque gallstone, air in biliary tree (40%)
- CT: biliary tract air, obstruction, gallstone in intestine

**Treatment**
- fluid resuscitation, NG decompression
- surgery: enterotomy and removal of stone, inspect small and large bowel for additional proximal stones
- fistula usually closes spontaneously
- elective cholecystectomy after recovery if patient experiences gallbladder symptoms

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**Carcinoma of the Gallbladder**

**Risk Factors**
- chronic symptomatic gallstones (70% of cases), old age, female, gallbladder polyps, porcelain gallbladder

**Clinical Features**
- majority are adenocarcinoma
- may be incidental finding on elective cholecystectomy (~1% of elective cholecystectomies)
- many patients are asymptomatic until late
- local: vague RUQ pain, ± palpable RUQ mass
- systemic: jaundice (50%) due to invasion of CBD or compression of CBD by pericholedochal nodes, weight loss, malaise, anorexia
- early local extension to liver, may extend to stomach, duodenum
- early metastasis common to liver, lung, bone

**Investigations**
- U/S: mural thickening, calcification, loss of interface between gallbladder and liver, fixed mass
- endoscopic U/S (EUS): good for distinguishing carcinomas from other diagnoses such as polyps, good for staging, allows sampling of bile for cytology
- abdominal CT: polypoid mass, mural thickening, liver invasion, nodal involvement, distant mets

**Treatment**
- if carcinoma of the gallbladder is suspected preoperatively, an open cholecystectomy should be done to avoid tumour seeding of trocar sites
- confined to mucosa (rare) – cholecystectomy
- beyond mucosa – cholecystectomy, en bloc wedge resection of 3-5 cm underlying liver, dissection of hepatoduodenal lymph nodes

**Prognosis**
- poor 5-year survival (10%) as gallbladder carcinoma is often detected late
Cholangiocarcinoma

Definition
• malignancy of extra and intrahepatic bile ducts

Risk Factors
• age 50-70, gallstones, ulcerative colitis, primary sclerosing cholangitis, choledochal cyst, 
  *Cholangitis sinensis* infection (liver fuse)

Clinical Features
• majority are adenocarcinoma
• gradual signs of biliary obstruction: jaundice, pruritis, dark urine, pale stool
• anorexia, weight loss, RUQ pain, Courvoisier’s sign (if CBD obstructed), hepatomegaly
• early metastases are uncommon, but commonly tumour grows into portal vein or hepatic artery
• Kistler tumour – cholangiocarcinoma located at bifurcation of common hepatic duct

Investigations
• LFTs show obstructive picture
• U/S, CT: bile ducts usually dilated, but not necessarily
• ERCP or PTC: to determine resectability, for biopsies
• CXR, bone scan: for metastatic workup

Treatment
• generally palliative
• if resectable: biliary drainage and wide excision margin
  • upper third lesions: duct resection + Roux-en-Y hepaticojejunostomy, ± liver resection
  • middle third lesions (uncommon): duct resection + Roux-en-Y hepaticojejunostomy
  • lower third lesions: Whipple procedure
• unresectable lesions: stent or choledochojejunostomy (surgical bypass)

Prognosis
• radiotherapy useful for additional palliation, chemotherapy may be helpful
• the more proximal to the liver, the worse the prognosis
• overall 5-year survival – 15%

Pancreas

Acute Pancreatitis

• see *Gastroenterology*, G48

Gallstone Pancreatitis

Pathogenesis
• obstruction of pancreatic duct by large or small gallstones and biliary sludge
• backup of pancreatic enzymes can cause autodigestion of the pancreas

Clinical Features (pancreatitis of any etiology)
• pain (epigastric pain radiating to back), nausea, vomiting, ileus, peritoneal signs, jaundice, fever
• “Ingle’s signs: pain worse when supine, better when sitting forward
• rarely may have coexistent cholangitis or pancreatic necrosis
• Ranson’s criteria for determining prognosis of acute pancreatitis (see below)

Investigations
• high amylase (higher than alcoholic pancreatitis), lipase, high liver enzymes, leukocytosis
• U/S may show multiple stones (may have passed spontaneously), edematous pancreas
• CXR, AXR, CT (if severe to evaluate for complications)

Treatment
• supportive
• NPO, hydration, analgesia and antibiotics for severe cases of necrotizing pancreatitis or signs of 
  sepsis
• stone often passes spontaneously (~90%); usually no surgical management in uncomplicated 
  acute pancreatitis
• cholecystectomy during same admission after acute attack has subsided (25-60% recurrence if 
  no surgery)

Ranson’s Criteria

A. At admission
1. Age > 55 yrs
2. WBC > 16 x 10^9/L
3. Glucose > 11 mmol/L
4. LDH > 250 IU/L
5. AST > 250 IU/L

B. During initial 48 hours
1. Hct drop > 10%
2. WBC rise > 1.8 mmol/L
3. Arterial PO2 < 60 mmHg
4. Base deficit > 4 mmol/L
5. Cakiur < 2 mmol/L
6. Fluid sequestration > 6 L

C. Interpretation
  • ≥2 – difficult course
  • ≥3 – high mortality
Chronic Pancreatitis

Surgical Treatment

- Indications for surgery:
  - failure of medical treatment
  - debilitating abdominal pain
  - pseudocyst complications: persistence, hemorrhage, infection, rupture
  - CBD obstruction (e.g. strictures), duodenal obstruction
  - pancreatic fistula, virulent hemorrhage secondary to splenic vein obstruction
  - rule out pancreatic cancer
  - anatomical abnormality causing recurrent pancreatitis
  - pre-op CT and/or ERCP are mandatory to delineate anatomy
  - surgical options:
    - drainage procedures – only effective if ductal system is dilated
    - endoscopic duct decompression
    - Puestow procedure (longitudinal pancreateojejunostomy) – improves pain in 80% of patients
    - pancreatostomy – best option in absence of dilated duct
    - proximal disease – Whipple procedure (pancreatecododenectomy); pain relief in 80%
    - distal disease – distal pancreatectomy + Roux-en-Y pancreateojunostomy
    - total pancreatectomy – refractory disease
    - nerve ablation:
      - celiac plexus block – lasting benefit in 30% patients, much less invasive
      - pseudocyst (most resolve spontaneously with pancreatic rest):
        - cyst wall must be mature (4-6 weeks)
        - internal drainage (preferred): Roux-en-Y cyst-jejunostomy or cyst-gastrostomy
        - external drainage may require second operation to treat pancreatic fistula
        - consider biopsy of cyst wall to rule out cystadenocarcinoma

Pancreatic Cancer

Epidemiology

- fourth most common cause of cancer-related mortality in both men and women in Canada in 2007 (Canadian Cancer Society)
- male:female = 1.7:1, average age: 60-70

Risk Factors

- increased age
- smoking – 2-5x increased risk, most clearly established risk factor
- high fat/low fibre diets, heavy alcohol use
- DM, chronic pancreatitis
- chemicals: beta-sitosterolamine, benzidine
- African descent
**Clinical Features**
- head of the pancreas (70%):
  - weight loss, obstructive jaundice, vague constant mid-epigastric pain (often worse at night, may radiate to back)
  - painless jaundice (occurs more often with peri-ampullary), Courvoisier's sign (see sidebar GS49)
  - palpable tumour mass → generally incurable
- body or tail of pancreas (30%):
  - tends to present later and usually inoperable
  - weight loss, vague mid-epigastric pain
  - <10% jaundiced
  - sudden onset diabetes

**Investigations**
- serum chemistry non-specific: elevated ALP and bilirubin >300 µmol/L
- US/contrast CT (also evaluates metastasis and resectability), ERCP

**Pathology**
- ductal adenocarcinoma – most common type (75-80%); exocrine pancreas
- intraductal papillary mucinous neoplasm (IPMN)
- other: mucinous cystic neoplasm (MCN), acinar cell carcinoma, islet-cell (insulinoma, gastrinoma, VIPoma, glucagonoma, somatostatinoma)

**Treatment**
- resectable (20% of pancreatic cancer)
  - no involvement of liver, peritonum or vasculature (hepatic artery, SMA, SMV, portal vein, IVC, aorta), no distant metastasis
  - Whipple procedure (pancreateoduodenectomy) for cure – 5% mortality (Figure 26)
  - distal pancreatectomy ± splenectomy, lymphadenectomy if carcinoma of midbody and tail of pancreas
- non-resectable (palliative → relieve pain, obstruction)
  - most body/tail tumours are not resectable (due to late presentation)
  - relieve biliary/duodenal obstruction with endoscopic stenting or double bypass procedure (choledochenterostomy + gastroenterostomy)
  - chemotherapy (gemcitabine), radiotherapy – only slightly increase survival

**Prognosis**
- most important prognostic indicators are lymph node status, size >3 cm, perineural invasion (invasion of tumour into microscopic nerves of pancreas)
- overall 5 year survival is 1%
- average survival – 6 months if unresected, 12-18 months with curative resection

---

**Figure 26. Schematic of Whipple Resection, Showing the Resected Components**
Spleen

Splenectomy

Indications
- splenic trauma (most common reason for splenectomy), hereditary spherocytosis, primary hypersplenism, chronic immune thrombocytopenia purpura (ITP), splenic vein thrombosis causing esophageal varices, splenic abscess, thrombotic thrombocytopenia purpura (ITP), non-Hodgkin’s lymphoma, primary splenic tumour (rare)
- does not benefit all thrombocytopenic states (e.g. infection, most malignancies involving the bone marrow, drugs/toxins)
- probability of cure of ITP by splenectomy is 60-70%, may be predicted by response to IV Ig

Complications
- short-term:
  - stasis of left lower lung, bleeding, infection
  - injury to surrounding structures (e.g. gastric wall, tail of pancreas)
  - post-op thrombocytosis, leukocytosis
  - subphrenic abscess
- long-term:
  - post-splenectomy sepsis (encapsulated organisms): 4% of splenectomized patients
  - 50% mortality
  - pre-op prophylaxis with vaccinations (pneumococcal, H. influenzae and meningococcus)
  - liberal use of penicillin especially in children <6 years old

Breast

Levels of Axillary Lymph Nodes
- Level I: lateral to pectoralis minor
- Level II: deep to pectoralis minor
- Level III: medial to pectoralis minor
(Higher level = worse prognosis)

Figure 27. Anatomy of the Breast
Benign Breast Lesions

NON-PROLIFERATIVE LESIONS
- Ska fibrocystic change, chronic cystic mastitis, mammary dysplasia
- Benign breast condition characterized by fibrous and cystic changes in the breast
- No increased risk of breast cancer
- Age 30 to menopause (and after if HRT used)
- Clinical features:
  - Breast pain, focal areas of nodularity or cysts often in the upper outer quadrant, frequently bilateral, mobile, varies with menstrual cycle, nipple discharge (straw-like, brown or green)
- Treatment:
  - Evaluation of breast mass and reassurance
  - If >40 years old: mammography every 3 years
  - No strong evidence for avoidance of xanthine-containing products (coffee, tea, chocolate, cola)
  - Analgesia (ibuprofen, ASA)
  - For severe symptoms: OCP, danazol, bromocriptine

PROLIFERATIVE LESIONS – No Atypia

Fibroadenoma
- Most common benign breast tumour in women under age 30
- Risk of subsequent breast cancer is increased only if fibroadenoma is complex, there is adjacent stypia or a strong family history of breast cancer
- Clinical features:
  - Nodules: smooth, rubbery, discrete, well-circumscribed, non-tender, mobile, hormone dependent
  - Unlike cysts, needle aspiration yields no fluid
- Investigations:
  - Core or excisional biopsy required
  - Ultrasound and FNA alone cannot differentiate fibroadenoma from phyllodes tumour
- Treatment:
  - Generally conservative: serial observation
  - Consider excision if size 2-3 cm and rapidly growing on serial ultrasound, if symptomatic or patient preference

Intraductal Papilloma
- Solitary intraductal benign polyp
- Present as nipple discharge (most common cause of spontaneous, unilateral bloody nipple discharge), breast mass, nodule on US
- Can harbour areas of stypia or DCIS
- Treatment: excision of involved duct to ensure no stypia

Ductal Hyperplasia Without Atypia
- Increased number of cells within the ductal space
- Cells retain benign cytology
- No treatment required
- Slightly increased cancer risk if moderate or florid hyperplasia

PROLIFERATIVE LESIONS – With Atypia

Atypical Hyperplasias
- Can involve ducts (ductal hyperplasia with stypia) or lobules (lobular hyperplasia with atypia)
- Cells lose apical basal orientation
- Increased risk of breast cancer
- Diagnosis: core or excisional biopsy
- Treatment: complete resection, risk modification (avoid exogenous hormones), close follow-up

OTHER LESIONS

Fat Necrosis
- Uncommon, result of trauma (may be minor, positive history in only 50%), after breast surgery (i.e. reduction)
- Firm, ill-defined mass with skin or nipple retraction, ± tenderness
- Regress spontaneously, but complete imaging ± biopsy to rule out carcinoma
Mammary Duct Ectasia
- obstruction of a subareolar duct leading to duct dilation, inflammation, and fibrosis
- may present with nipple discharge, bluish mass under nipple, local pain
- risk of secondary infection (abscess, mastitis)
- resolves spontaneously

Montgomery Tubercles
- Montgomery tubercles aka Mongagni tubercles are popular projections at the edge of the areola
- obstruction of these glands can lead to inflammation or cystic collections (cyst of Montgomery aka retroareolar cyst)
- if signs of secondary infection, start treatment for mastitis
- resolves spontaneously in weeks to years

Abscess
- lactational (see Obstetrics OB30) vs. periductal/subareolar
- unilateral localized pain, tenderness, erythema, subareolar mass, nipple discharge, nipple inversion
- rule out inflammatory carcinoma, as indicated
- treatment: initially broad-spectrum antibiotics and I&D, if persistent total duct excision (definitive)
- if mass does not resolve: fine needle aspiration (FNA) to exclude cancer, U/S to assess for presence of abscess

Breast Cancer

Epidemiology
- 2nd leading cause of cancer mortality in women (1st is lung cancer)
- 1/9 women in Canada will be diagnosed with breast cancer in their lifetime
- 1/27 women in Canada will die from breast cancer

Risk Factors
- gender (99% female)
- age (80% >40 years old)
- most important risk factors are prior history of breast cancer and/or prior breast biopsy (regardless of pathology)
- 1st degree relative with breast cancer (greater risk if relative was premenopausal)
- increased risk with high breast density, nulliparity, first pregnancy >30 years old, menarche <12 years old, menopause >55 years old
- decreased risk with lactation, early menopause, early childbirth
- radiation exposure (e.g. Mantle radiation for Hodgkin’s disease)
- >5 years HRT

Investigations
- mammography
  - indications:
    - screening (see Table 15):
      - every 1-2 years for women age 50-69
      - positive family history in 1st degree relative: every 1-2 years starting 10 years before the youngest age of presentation
    - diagnosis: investigation of patient complaints (discharge, pain, lump)
    - follow up after breast cancer surgery
  - findings indicative of malignancy:
    - mass that is poorly defined, spiculated border
    - microcalcifications
    - architectural distortion
    - interval mammographic changes
    - normal mammogram does not rule out suspicion of cancer based on clinical findings
  - other radiographic studies:
    - ultrasound – differentiate between cystic and solid
    - MRI – high sensitivity, low specificity
    - galactogram/ductogram (for nipple discharge) – identifies lesions in ducts
    - metastatic workup as indicated (usually after surgery or if clinical suspicion of metastatic disease) – bone scan, abdomen U/S, CXR, head CT

Any palpable dominant breast mass requires further investigation.

Diagnostic mammography is indicated in all patients, even in women <50 years old.
Table 15. Screening for Breast Cancer in Women of Average Risk

<table>
<thead>
<tr>
<th>Test/Maneuver</th>
<th>Effectiveness</th>
<th>Level of Evidence</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mammography, with or without clinical examination*, women aged 40-48 years</td>
<td>Controversial; routine mammography with or without clinical examination, has not conclusively been shown to reduce breast cancer mortality or overall mortality (7 RCTs, meta-analysis)</td>
<td>RCTs (i)</td>
<td>Current evidence does not support the recommendation that screening mammography be included or excluded from the periodic health examination of women aged 40-48 with average risk of breast cancer (Grade C)</td>
</tr>
<tr>
<td>Mammography, with or without clinical examination*, women aged 55-69 years</td>
<td>Statistically significant reduction in breast cancer mortality (RR 0.76) though overall mortality not affected (7 RCTs, meta-analysis)</td>
<td>RCTs (i)</td>
<td>Based on breast cancer-specific mortality, the Canadian Task Force on Preventive Health Care concluded there was good evidence for screening women aged 55-69 by mammography (and clinical breast exam). (Grade A) The breast surveillance evidence does not provide conclusive direction regarding annual versus biennial screening.</td>
</tr>
<tr>
<td>Teaching of Breast Self Examination (BSE) to women aged 40-69 years</td>
<td>Evidence of no benefit in terms of survival from breast cancer (RCTs (i))</td>
<td>Non-RCTs (i-1)</td>
<td>Fair evidence of no benefit and good evidence of harm, therefore fair evidence not to recommend routine teaching of BSE from the periodic health examination (Grade D)</td>
</tr>
</tbody>
</table>

* The utility of adding clinical breast examination (CBE) to mammography is unclear. Some of the 7 RCTs that evaluated CBE and mammography in combination and some separately, do not take the contribution of mammography and clinical breast exam into account, both maneuvers are recommended by the Canadian Task Force on Preventive Health Care.

Diagnostic Procedures
- needle aspiration: for palpable cystic lesions; send fluid for cytology if blood or cyst does not completely resolve
- U/S or mammography guided core needle biopsy (most common)
- fine needle aspiration (FNA): for palpable solid masses; need experienced practitioner for adequate sampling
- excisional biopsy: only performed as second choice to core needle biopsy; should not be done for diagnosis if possible

Genetic Screening
- consider testing for BRCA1/2 if:
  - patient diagnosed with breast AND ovarian cancer
  - strong family history of breast/ovarian cancer (e.g. Ashkenazi Jewish)
  - family history of male breast cancer
  - young patient (<35 years old)

Staging (see Table 16)
- clinical:
  - tumour size by palpation, mammogram
  - nodal involvement by palpation
  - metastasis by physical exam, CXR and abdo U/S (or CT cistern/abdo/pelvis), bone scan
  - (usually done pre-op if node-positive disease)
- pathological:
  - tumour size
  - grade: modified Bloom and Richardson score (1 to III) - histologic, nuclear and mitotic grade
  - number of axillary nodes positive for malignancy out of total nodes removed, extranodal extension, sentinel node positive/negative
  - estrogen receptor (ER) + progesterone receptor (PR) testing
  - HER2Neu receptor testing
- margins: negative, <1 mm, positive
- lymphovascular invasion (LVI)
- extensive in situ component (EIC): DCIS in surrounding tissue
- involvement of dermal lymphatics (Inflammatory) - automatically Stage IIIb

Table 16. Staging of Breast Cancer (American Joint Committee on Cancer)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Tumour Size</th>
<th>Nodes (regional) (clinical)</th>
<th>Metastasis</th>
<th>Survival (5-Year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>&lt;2 cm</td>
<td>None</td>
<td>None</td>
<td>99%</td>
</tr>
<tr>
<td>II A</td>
<td>&lt;2 cm</td>
<td>Mobile intraductal</td>
<td>None</td>
<td>85%</td>
</tr>
<tr>
<td>II B</td>
<td>2.5 cm or &gt;3 cm</td>
<td>Mobile or multicellular</td>
<td>None</td>
<td>70%</td>
</tr>
<tr>
<td>II A</td>
<td>Any</td>
<td>Fixed intraductal or intramyxillary</td>
<td>None</td>
<td>52%</td>
</tr>
<tr>
<td>II B</td>
<td>Any</td>
<td>Intraductal or intramyxillary</td>
<td>None</td>
<td>40%</td>
</tr>
<tr>
<td>II C</td>
<td>Any</td>
<td>Intraductal or intramyxillary</td>
<td>None</td>
<td>33%</td>
</tr>
<tr>
<td>III</td>
<td>Any</td>
<td>Distant</td>
<td>Distant</td>
<td>12%</td>
</tr>
</tbody>
</table>
Pathology
- non-invasive (cannot penetrate basement membrane):
  - ductal carcinoma in situ (DCIS):
    - proliferation of malignant ductal epithelial cells completely contained within breast ducts, often multifocal
    - 80% non-palpable, detected by screening mammogram
    - risk of invasive ductal carcinoma in same breast up to 35% in 10 years
  - treatment:
    - lumpectomy with wide excision margins + radiation (5-10% risk invasive cancer)
    - mastectomy if large area of disease, high grade or multifocal (risk of invasive cancer reduced to 1%)
    - possibly tamoxifen as an adjuvant treatment
    - 99% 5-year survival
- lobular carcinoma in situ (LCIS):
  - neoplastic cells completely contained within breast lobule
  - no palpable mass, no mammographic findings, usually incidental finding on breast biopsy for another indication
  - treatment:
    - clinical follow-up
    - chemoprevention (tamoxifen)
    - surgery (uncommon)
  - not a precursor lesion, but considered a risk factor for breast cancer development
- invasive:
  - invasive ductal carcinoma (most common 80%):
    - originates from ductal epithelium and infiltrates supporting stroma
    - characteristics hard, scirrhus, infiltrating tentacles, gritty on cross-section
  - invasive lobular carcinoma (8-15%):
    - originates from lobular epithelium
    - 28% bilateral (i.e. more often than infiltrating ductal carcinoma)
    - does not form microcalcifications, harder to detect mammographically (may benefit from MRI)
  - Paget's disease (1-3%):
    - ductal carcinoma that invades nipple with scaling, eczematoid lesion
  - inflammatory carcinoma (1-4%):
    - ductal carcinoma that invades dermal lymphatics
    - most aggressive form of breast cancer
    - clinical features: erythema, skin edema, warm, swollen and tender breast & lump
    - peau d'orange indicates advanced disease (IIIb-IV)
  - male breast cancer (<1%):
    - most commonly invasive ductal carcinoma
    - often diagnosed at later stages
    - stage-for-stage similar prognosis to breast cancer in females
    - consider genetic testing
  - sarcomas: rare
    - most commonly phyllodes tumour, a variant of fibroadenoma with potential for malignancy
  - lymphomas: rare
  - other: papillary, medullary, macnous, tubular cancers
    - generally better prognosis

Treatment

<table>
<thead>
<tr>
<th>Stage</th>
<th>Primary Treatment Options</th>
<th>Adjuvant Systemic Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (in situ)</td>
<td>BCS + radiotherapy</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>BCS alone if margins &gt;1 cm and low nuclear grade</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mastectomy* + SLNB</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>BCS + axillary node dissection + chemotherapy</td>
<td>May not be needed; discuss data/benefits of chemotherapy and tamoxifen</td>
</tr>
<tr>
<td></td>
<td>Mastectomy* + axillary node dissection/SLNB</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>BCS + axillary node dissection + chemotherapy</td>
<td>Chemotherapy for premenopausal women or postmenopausal and estrogen receptor (ER) negative, follow by tamoxifen if ER positive</td>
</tr>
<tr>
<td></td>
<td>Mastectomy* + axillary node dissection/SLNB</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Likely mastectomy + axillary node dissection + adjuvant therapy</td>
<td>Neoadjuvant therapy may be considered i.e. preparative chemotherapy and/or hormone therapy. Adjuvant radiation and chemotherapy may also be appropriate (i.e. postop)</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Likely mastectomy + axillary node dissection + radiotherapy</td>
<td>Neoadjuvant therapy</td>
</tr>
<tr>
<td>I</td>
<td>Surgery as appropriate for local control</td>
<td>Primary treatment is systemic therapy i.e. chemotherapy and/or hormone therapy</td>
</tr>
</tbody>
</table>

BCS = breast conserving surgery; SLNB = sentinel lymph node biopsy
* if reason to select mastectomy, choice between BCS + radiotherapy and mastectomy can be made according to patient's preference since choice of local treatment does not significantly affect survival if local control is achieved
Primary Surgical Treatment
- breast-conserving surgery (BCS) – lumpectomy with wide local excision
  - for treatment of stage I and II disease
  - must be combined with radiation for survival equivalent to mastectomy
- contraindications:
  - high risk of local recurrence – extensive malignant-type calcifications on mammogram, multifocal primary tumours, or failure to obtain tumour-free margins after re-excision
  - contraindications to radiation therapy (pregnancy, previous radiation, collagen vascular disease)
  - large tumour size relative to breast
- mastectomy
  - Radical mastectomy (rarely done anymore) – removes all breast tissue, skin, pectoralis muscle, axillary nodes
  - Modified radical mastectomy (MRM) – removes all breast tissue, skin, and axillary nodes
  - Simple mastectomy – removes all breast tissue and skin
- see Plastic Surgery, PL31 for breast reconstruction
- axillary lymph node dissection (ALND)
  - performed if SLNB is positive or nodes are clinically concerning
  - risk of arm lymphedema (10-15%), decreased arm sensation, shoulder pain
- sentinel lymph node biopsy (SLNB)
  - technetium-99m blue dye injected at tumour site prior to surgery to identify sentinel node(s)
  - intraoperative frozen section
  - proceed with ALND if positive
  - 5% false negative rate

Adjuvant/Neoadjuvant
- radiation
  - indications:
    - decrease risk of local recurrence; almost always used after BCS, sometimes after mastectomy (ax > 4 nodes positive or tumour > 5 cm)
    - inoperable locally advanced cancer
  - axillary nodal radiation may be added if nodal involvement
- hormonal
  - indications:
    - ER positive plus node-positive or high-risk node-negative
    - palliation for metastasis
  - tamoxifen if premenopausal or aromatase inhibitors (e.g. anastrozole)
  - ovarian ablation (e.g. goserelin/GnRH agonist, oophorectomy), progestins (e.g. megestrol acetate), androgens (e.g. fluoxymesterone) are other options
- chemotherapy
  - indications:
    - ER negative plus node-positive or high-risk node-negative
    - ER positive and young age
    - stage I disease at high risk of recurrence (high grade, lymphovascular invasion)
    - palliation for metastatic disease

Post-Treatment Follow-up
- visits q3-6 months x 2 years and annually thereafter (frequency is controversial)
- annual mammography; no other imaging unless clinically indicated
- psychosocial support and counselling

Local/Regional Recurrence
- recurrence in treated breast or ipsilateral axilla
  - 1% per year up to maximum of 15% risk of developing contralateral malignancy
  - 5x increased risk of developing metastases

Metastasis
- bone > lungs > pleura > liver > brain
- treatment is palliative: hormone therapy, chemotherapy, radiation
Surgical Endocrinology

Thyroid and Parathyroid

- see Endocrinology, E28 and Otolaryngology, OT32, OT34

Adrenal Gland

- see Endocrinology, E35
- functional anatomy:
  - cortex: glomerulosa (mineralocorticoids), fasciculata (glucocorticoids), reticularis (see steroids)
  - medulla: catecholamines (epinephrine, norepinephrine)
- types: functional (e.g., Cushing's syndrome, Conn's syndrome) or non-functional

Incidentaloma

- adrenal mass discovered by investigation of unrelated symptoms

Epidemiology

- benign adenoma (38%) > metastases to adrenal (22%) >> cysts, carcinoma, pheochromocytoma, neuroblastoma
- metastases to adrenal gland from: lung > breast, colon, lymphoma, melanoma, kidney
- peak incidence of carcinoma: females ages 50-60, risk decreases with increasing age and male gender

Investigations

- MRI, CT: size >6 cm is best predictor of primary adrenal carcinoma (92% are >6 cm)
- functional studies:
  - pheochromocytoma: 24 hour urine epinephrine, norepinephrine, metanephrine, normetanephrine, VMA (vanillylmandelic acid)
  - Cushing's: 24 hour urine cortisol or 1 mg overnight dexamethasone suppression test
  - aldosteronoma: electrolytes, aldosterone: renin level, saline suppression test if appropriate
  - adrenal androgens: 17-OH progesterone, DHEAS
  - FNA biopsy: if suspect metastases to adrenal (must exclude pheochromocytoma first)
  - indicated if history of cancer or patient is smoker
  - iodochlorhydroxyquin scintigraphy: may distinguish benign vs. malignant disease

Treatment

- functional tumour: resect
- non-functional tumour:
  - >6 cm: resect
  - 3-6 cm: MRI (T2 density, shape, margins), more likely to resect in females and if <60 years old
  - <3 cm: follow with repeat CT in 12-18 months

Skin Lesions

- see Dermatology, D6; Emergency, ER17; Plastic Surgery, PL14

Common Medications

Antiemetics

- dimenhydrinate (Gravol®) 25-50 mg PO/IV/IM q4-6h prn
- prochlorperazine (Stemetil®) 5-10 mg PO/IV/IM bid-tid prn
- metoclopramide (Maxeran®) 10 mg IV/IM q1-3h prn, 10-15 mg PO qid (30 min before meals and qhs)
- ondansetron (Zofran®)
- granisetron (Kytril®) 1 mg PO bid (for nausea from chemotherapy/radiation)
**Analgesics**
- acetaminophen & codeine (Tylenol® #3/plain) 1-2 tabs q4-6h PO/PR prn
- morphine 2.5-10 mg IM/SC q 4-6h prn + 1-2 mg IV q1h prn for breakthrough
- ketorolac (Toradol®)
- Percocet® (acetaminophen/oxycodeone, 325/5 mg) 1-2 tabs PO q4-6h prn

**DVT Prophylaxis**
- heparin 5000 units SC bid, if cancer patient then heparin 5000 units SC ttd
- dalteparin (Fragmin®) 5000 units SC daily
- enoxaparin (Lovenox®) 40 mg SC daily

**Antidiarrheals**
- loperamide (Imodium®) 4 mg PO initially, then 2 mg PO after each loose stool up to 16 mg/d
- diphenoxylate + atropine (Lomotil®) 2 tabs/16 ml PO qid

**Laxatives**
- sennosides (Sephos®) 1-2 tabs qhs
- docusate sodium (Colace®) 100 mg PO bid
- glycerine supposit 1 tab PR prn
- lactulose 15-30 ml PO qid prn
- milk of magnesia (MOM) 30-60 ml PO qid prn
- bisacodyl (Dulcolax®) 10-15 mg PO prn

**Sedatives**
- zopiclone (Imovane®) 5-7.5 mg PO qhs prn
- lorazepam (Ativan®) 0.5-2 mg PO/SL qhs prn

**Antibiotics**
- cefazolin (Ancef®) 1 g IV/IM on call to OR or q8h – GP except Enterococcus, GN only E. coli, Klebsiella and Pneumonia
- cefalexin (Keflex®) 250-500 mg PO qid – Listeria, GP except Enterococcus, GN only E. coli, Klebsiella and Pneumonia
- ceftriaxone 1-2 g IM/IV q24h – broad coverage including Pseudomonas
- ampicillin 1-2 g IV q6-8h – Listeria, GP (Enterococcus) except Streptococcus and E. coli, oral anaerobes except Bacteroide
- gentamicin 3-5 mg/kg/day IM/IV divided q8h; monitor creatinine, gentamicin levels – GN including Pseudomonas
- ciprofloxacin 400 mg IV q12h, 500 mg PO bid – GN including Pseudomonas
- metronidazole (Flagyl®) 500 mg PO/IV bid. (500 mg PO tid for C. difficile) – anaerobes
- clindamycin 600-900 mg IV q8h, 150-400 mg PO qid – GP except Enterococcus, anaerobes

**Over-the-Counter Medications**
- Pepto-Bismol® (bismuth subsalicylate) 2 tabs or 30 ml PO q30min-1hr up to 8 doses/day
  * side effects: black stools, risk of Reye’s syndrome in children
- Alka-Seltzer® (ASA + citrate + bicarbonate) 2 tabs in 4 oz water PO q4h prn, max 8 tabs
- Maalox® (aluminum hydroxide + magnesium hydroxide) 10-20 ml or 1-4 tabs PO prn
- Tums® (calcium carbonate) 1-3 g PO q2h prn
- Rolaid® (calcium carbonate and magnesium hydroxide) 2-4 tabs PO q1h prn, max 12 tabs/day