CARDIOLOGY

Aortic Aneurysm

-Occurs when blood collects between the aortic vessel layers, with true aneurysms involving all 3 layers (intima, media, adventitia)
-Most commonly occurs below the kidney

Risk Factors
-Smoking
-HTN
-Hyperlipidemia
-Male
-Atherosclerosis
-FH

Differential
-Pseudoaneurysm: a collection of blood and connective tissue located outside of the vessel wall

Prognosis
-Can spontaneously rupture
-Post-op complications: MI, reduced blood flow to LEs from emboli, AKI, mesenteric or spinal cord ischemia, device migration or endoleak with graft placement

Abdominal Aortic Aneurysm

-Normally AA is 2 cm, becomes aneurysmal when > 3 cm
-Caused by atherosclerosis and inflammation
-Categorized based on morphology
-Usually infrarenal in location

Screening
-USPSTF recommends US screen in all men 65-75 who have ever smoked
-May also want to screen women with cardio risk factors and anyone > 50 with a FH

Signs & Symptoms
-Usually asymptomatic and discovered incidentally on abdominal exam
-Abdominal or back pain
-May have signs of limb ischemia

Workup
-Abdominal US for diagnosis
-Abdominal CT for further characterization and measurement

Management
-Surgical repair indicated when > 5 cm; may be endovascular (stent) or open graft repair; endovascular has lower short-term mortality/morbidity but open repairs have better long-term outcome
-Watchful reimaging and risk reduction if < 5 cm: smoking cessation
-Consider elective nonrepair and cessation of surveillance imaging if life expectancy is < 2 years

Thoracic Aortic Aneurysm

-Further classified as ascending, descending, or arch
-Ascending thoracic AA usually due to elastin degradation, which can be normal aging or accelerated by HTN, connective tissue disorder, RA, or bicuspid aortic valve
-Descending thoracic aneurysm is caused by atherosclerosis
-Arch aneurysm seen in trauma or deceleration injuries

Signs & Symptoms
-Aortic insufficiency symptoms from dilation of valve
-CHF
-Compression of SVC by enlarging aorta → SVC syndrome
-Tracheal deviation
-Cough
-Hemoptysis
-Dysphagia
-Hoarseness
-Steady, deep, severe substernal, back, or neck pain

Workup
-CXR for widened mediastinum, enlarged aortic knob, tracheal displacement
-MRI or CTA are test of choice for characterization and dx
-Echo

Management
-BP control, β-blockers preferred
-Re-image with CT or MRI every 6 mos
-Surgical management is risky and complicated = rarely done, need to weigh risk of rupture
-Surgical repair indicated for thoracic AA ≥ 6 cm, rapid expansion of aneurysm, or symptomatic aneurysm

Prognosis
-Less likely to spontaneously rupture than AAA

Varicose Veins

-Usually occur in the saphenous veins

Causes
-Incompetent valves from damage or venous dilation
-AV fistula
-Congenital venous malformations

Signs & Symptoms
-Dull or aching pain in legs that is worse after standing
-Pruritus
-May have h/o DVT
-Brownish thinning of the skin above the ankles

Differential
-Claudication
-Superficial thrombophlebitis
-Arthritis
-Peripheral neuropathy

Management
-Compression stockings
-Leg elevation
-Venous ablation
-Sclerotherapy
-Great saphenous vein stripping

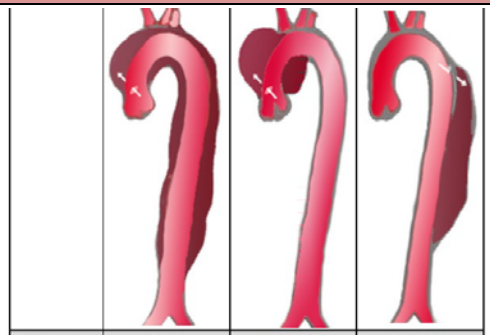
Prognosis
-Complication of thrombophlebitis

Aortic Dissection

-Occurs when tear in the inner wall of the aorta causes blood to flow between the wall layers → creation of false lumen
-Acute or chronic
-Usually in ascending aorta
-Typically in men 60-70
-Debakey and Stanford classifications

Risk Factors
-Connective tissue disorders
-Bicuspid aortic valve
-Coarctation of the aorta
-HTN

Causes
-Usually a result of HTN
-Increased risk in pregnancy, connective tissue disease, bicuspid aortic valve, aortic coarctation



Percentage	60%	10–15%	25–30%
Type	DeBakey I	DeBakey II	DeBakey III
	Stanford A (Proximal)		Stanford B (Distal)

Signs & Symptoms
-Sudden onset of "ripping" retrosternal and back pain
-HTN
-Hypovolemia
-Syncope
-Shock
-Pulse discrepancies
-Cardiac tamponade
-May have focal neuro deficits or CVA due to poor perfusion of the brain

Workup
-EKG may show infarct pattern or LVH
-CXR will show widened mediastinum, L sided pleural effusion
-Bedside TEE test of choice, CT if unavailable

Management
-Achieve hypotension and bradycardia with β-blocker and nitroprusside
-Surgical repair for Stanford type A
-Stanford type B admitted to ICU for medical management: morphine for pain control, β-blockers and nitroprusside

Prognosis
-Greater than 20% intra-op mortality
-50% mortality within 10 years of all hospital survivors

Deep Venous Thrombosis

Signs & Symptoms
-Palpable cord
-Calf pain
-Ipsilateral edema, warmth, tenderness, erythema

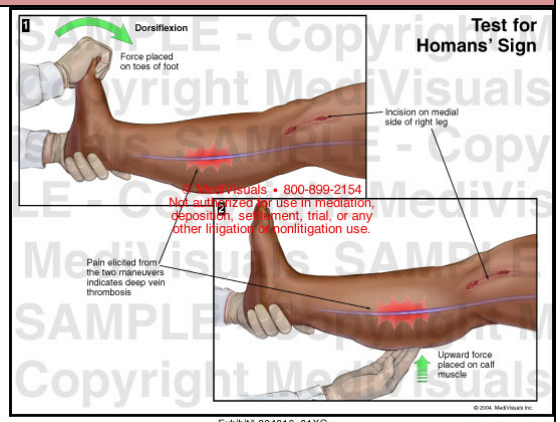
Workup
-Homan's is only + 50% of the time
-Determine probability with Well's criteria → < 2 indicates unlikely, > 6 highly likely
-Further investigation using D-dimer
-US for at least moderate Well's score

Management
-Immediate anticoagulation with heparin, LMWH, or fondaparinux
-Lytics or thrombectomy for select cases
-3 months of anticoagulation for initial distal DVT or consider IVC filter if not a good candidate

Table 5. Wells et al Clinical Model For Predicting Pretest Probability For DVT[24]

Clinical Characteristic	Score
Active cancer (patient receiving treatment for cancer within previous the 6 months or currently receiving palliative treatment)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recent bedridden for greater than 3 days or major surgery within the previous 12 weeks requiring general or regional anesthesia	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling at least 3 cm larger than that on the asymptomatic leg (measured 10 cm below tibial tuberosity)	1
Pitting edema confined to the symptomatic leg	1
Collateral superficial veins (nonvaricose)	1
Previously documented deep-vein thrombosis	1
Alternative diagnosis at least as likely as deep-vein thrombosis	-2

A total score of two or higher indicates that the probability of deep-vein thrombosis is likely; a total score of less than two indicates that the probability of deep-vein thrombosis is unlikely. In patients with symptoms in both legs, use the more symptomatic leg.



Coronary Artery Disease

-Risk of developing CAD for 40 year olds in the US is 49% for men and 32% for women
-Risk factors: age, males, FH, sedentary lifestyle, tobacco, HTN, DM, ↑ lipids

Classification
-Class I = no limitations or symptoms with normal activity
-Class II = slight limitations and normal activity results in symptoms
-Class III = marked limitation and minimal activity results in symptoms
-Class IV = symptoms persist with minimal activity and rest

Screening
-Consider stress test in asymptomatic pts with multiple risk factors

Signs & Symptoms
-Angina
-SOB
-Sudden cardiac death is the first symptom in 15%

Chest pain differential
-Atherosclerosis
-Vasospasm from cocaine or stimulants
-Prinzmetal's angina: women under 50
-Coronary artery or aortic dissection
-Congenital abnormality
-Aortic stenosis
-HCM
-Coronary thrombus or embolus
-Non-cardiac: costochondritis (reproducible on palpation), intercostal shingles, cervical or thoracic spine disease (reproducible with specific movements of the head or neck, causes paresthesias), PUD, GERD, cholecystitis, PE, pneumonia, pneumothorax (dyspnea)

Workup
-PE findings: S4, arterial bruits, abnormal funduscopic exam, corneal arcus, xanthelasma, tendinous xanthoma, CHF, murmurs
-EKG
-Refer for stress test if pt has low to intermediate probability of CAD
-Refer for cardiac cath if pt has high probability of CAD

Management of new disease or worsening symptoms
-Referral to cardiology
-ER via ambulance if EKG shows new ischemic changes: ST depression or elevation, inverted T waves or there is hemodynamic instability

Management of stable disease
-LDL goal <100 or <70
-β-blocker (proven mortality benefit), CCB, statin, clopidogrel, nitrates PRN
-New drug ranolazine for refractory chest pain
-PCP visits every 6 months: annual CBC to check for anemia, annual lipids, FBG
-Cardiologist every 1-2 years
-Consider early revascularization for significant narrowing of LAD, left main CAD, LVEF < 30%, or large area of myocardium at risk

PULMONOLOGY

Acute Respiratory Distress Syndrome (ARDS)

-Noncardiogenic pulmonary edema caused by capillary leaking from infection or inflammation → parenchymal inflammation and edema → impaired gas exchange and systemic release of inflammatory mediators → further inflammation, hypoxemia, and frequently multiple organ failure

Acute lung injury (ALI) = a less severe form of ARDS

Inciting Events
-Pneumonia of any kind
-Chemical inhalation
-Chest trauma
-Sepsis
-Pancreatitis
-Connective tissue disease: lupus
-Vasculitis
-Hypersensitivity rxn to blood transfusion
-Burns

Stages
-Stage I: clear CXR, infiltration of PMNs begins
-Stage II: develops over 1-2 days with patchiness on CXR with edema and type I alveolar cell damage
-Stage III: develops over 2-10 days with diffuse infiltration on CXR, exudates, proliferation of type II alveolar cells functioning as repair cells
-Stage IV: develops > 10 days with diffuse infiltration on CXR, involvement of lymphocytes → pulmonary fibrosis

Workup
Diagnostic criteria: known clinical insult < 1 week ago, bilateral opacities consistent with pulmonary edema on CXR or CT, not explained by cardiac failure or fluid overload, hypoxemia present on minimal ventilator settings ($PaO_2/FiO_2 < 300$)

Management
-Treat underlying cause!
-Usually mechanical ventilation is needed, using low tidal volumes and PEEP
-Use of steroids is controversial
-Supportive care: sedatives, nutritional support, BG control, DVT prophylaxis, GI prophylaxis, neuromuscular blockade for vent dyssynchrony to ↓O_2 demand, keep fluid balance even to negative

Prognosis
-Overall mortality 40-60%

Lung Cancer (Bronchogenic Carcinoma)

-85% of cases occur among smokers
-Other contributing causes include radon gas, asbestos, and environmental pollutants
-2 major groups (small cell and non-small cell) account for 95% of lung cancers
-Other lung cancers are rarer and include primary pulmonary lymphoma, carcinoid tumors, bronchoalveolar cancers, and mesotheliomas
-Overall survival rate of 14%

Signs & symptoms
-Lung cancers are more like to cause paraneoplastic syndromes such as hypercalcemia, SIADH, ectopic ACTH secretion, Lambert-Eaton myasthenic syndrome, and hypercoagulable states
-Nonspecific cough or dyspnea
-Chest pain
-Hemoptysis
-Anorexia, weight loss, fevers, night sweats
-Hoarseness due to compression of the recurrent laryngeal nerve
-Facial or UE swelling from SVC syndrome
-Bone, brain, liver, or adrenal symptoms from mets
-Axillary or supraclavicular adenopathy
-Digital clubbing

Differential
-TB
-Fungal infection
-Mets to the lung
-Sarcoidosis

Workup
-Begin with CXR
-F/u masses with CT
-Sputum cytology
-Bronchoscopy
-Transthoracic needle biopsy
-Node sampling via transbronchial biopsy, mediastinoscopy, or mediastinotomy

Management
-Assess feasibility of surgical resection and overall patient health/quality of life issues
-Radiation for advanced disease or nonsurgical candidates
-Combination chemotherapy for candidates
-Monitoring for recurrence

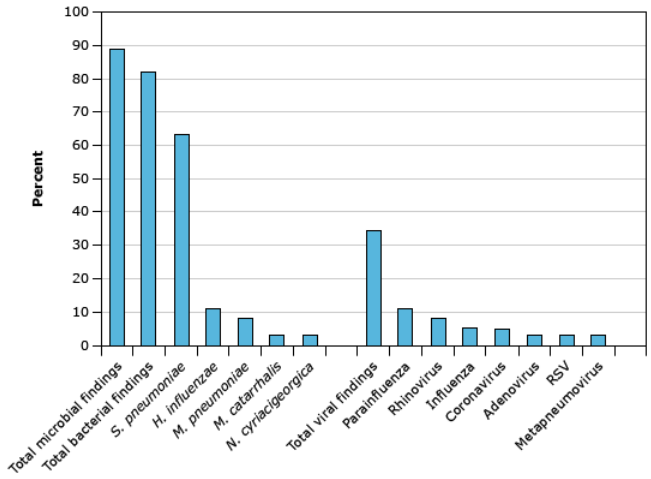
2004 WHO classification of invasive malignant epithelial lung tumors

Squamous cell carcinoma
Variants: papillary, clear cell, small cell, basaloid
Small cell carcinoma
Variant: combined small cell carcinoma
Adenocarcinoma
Adenocarcinoma, mixed subtype
Acinar adenocarcinoma
Papillary adenocarcinoma
Bronchioloalveolar carcinoma
Variants: nonmucinous, mucinous, mixed nonmucinous and mucinous or indeterminate
Solid adenocarcinoma with mucin production
Variants: fetal adenocarcinoma, mucinous ("colloid") carcinoma, mucinous cystadenocarcinoma, signet ring adenocarcinoma, clear cell adenocarcinoma
Large cell carcinoma
Variants: large cell neuroendocrine carcinoma, combined large cell neuroendocrine carcinoma, basaloid carcinoma, lymphoepithelioma-like carcinoma, clear cell carcinoma, large cell carcinoma with rhaboid phenotype
Adenosquamous carcinoma
Sarcomatoid carcinoma
Variants: pleomorphic carcinoma, spindle cell carcinoma, giant cell carcinoma, carcinosarcoma, pulmonary blastoma
Carcinoid tumor
Variants: typical carcinoid, atypical carcinoid
Salivary gland tumors
Variants: mucoepidermoid carcinoma, adenoid cystic carcinoma, epithelial-myoepithelial carcinoma

Non-Small Cell Carcinoma	Small Cell Carcinoma	Bronchial Carcinoid Tumor	Squamous Cell Carcinoma	Adenocarcinoma
-Arises as discrete masses within the lung parenchyma that can spread to regional lymph nodes and then metastasize to distant sites -Squamous, adeno, and large cell carcinomas -Staged by TNM -Limited response to chemo -Surgical resection of limited tumors can be curative	-Metastasize rapidly to regional lymph nodes and distant sites -Classified as limited or extensive disease -Very responsive to chemo -Remission is common but so is recurrence → overall survival of 5%	-Previously known as bronchial adenoma -Rare group of pulmonary neoplasms characterized by neuroendocrine differentiation and relatively indolent clinical course -Can also arise in the thymus, GI tract, and ovary -Surgical resection is treatment of choice	-Associated with slow growth and late metastasis	-Peripheral -Rapid growth with mets -Associated with lung scarring, not smoking

Pneumonia

Microbial etiology of community-acquired pneumonia in patients who underwent comprehensive testing



Prevention with pneumococcal vaccination	
23 valent (Pneumovax)	-Adults over 65 -Persons aged 19-64 years with chronic cardiovascular disease (including CHF and cardiomyopathy), chronic pulmonary disease (including asthma and COPD), DM, alcoholism, chronic liver disease (including cirrhosis), CSF leak, cochlear implant, cigarette smoking - Persons aged 19-64 years who are residents of nursing homes or long-term care facilities -Singe revaccination recommended if adult was < 65 and it was more than 5 years ago when they got it, and in immunocompromised 5 years after initial dose
13 valent	-Adults who are immunocompromised (should get 23 valent also, but not at same time) -Routine for all kids under 5 -Kids 6-18 who have sickle cell disease, HIV or other immunocompromising conditions, cochlear implant, or CSF leak
7 valent	-No longer being used

Signs & symptoms
-Rigors, sweats, fever or subnormal temp, cough ± sputum, dyspnea, pleuritic chest pain, fatigue, myalgias, abdominal pain, anorexia, headache, AMS
-Pleural effusion: pulmonary consolidation, crackles, dullness to percussion, ↓ breath sounds

Outpatient

Workup
-CXR: may lag behind PE findings!
-Urine test for *Legionella*
-CBC, BMP

CXR Findings
-Can't tell explicitly viral vs pneumonia by patterns (old myth!)
-Lobar pneumonia: suggests *Strep pneumo, H flu, Legionella*
-Patchy infiltrates in multiple lung areas (bronchopneumonia): suggests *Staph aureus*, gram negs, atypicals, viruses
-Fine dense granular infiltrates (interstitial pneumonia): suggests influenza, CMV, PCP
-Lung abscess: suggests anaerobes
-Nodular lesions suggests fungal

Management
-CAP → macrolide
-Underlying comorbidity (higher risk = need to cover resistant *Strep pneumo*, enterics, *Moraxella*, anaerobes) → antipneumococcal FQ like levo, or macrolide + β-lactam (cefpodoxime, cefuroxime, amox HD, ceftriaxone)

Disposition
-Use PORT score or CURB-65 to estimate risk (QX Calculate app) and determine outpatient vs inpatient
-ER if RR > 30, HR > 125, SBP < 90, comorbidities

Prognosis
-Fever clears after 2-4 days of treatment
-CXR clears after 30 days (up to 6 mos if elderly)

Inpatient

HAP = pneumonia appearing > 48 hours after admission, or PNA in a recently hospitalized pt

HCAP = PNA in non-hospitalized pt that has had extensive healthcare contact (group home, SNF, IV therapy, HD, etc)

VAP = ventilator-associated pneumonia

Additional Workup
-ICU or EtOH or pleural effusion→ blood culture, sputum culture, *Legionella* & pneumococcal antigen testing

Non-HCAP Management
-Non ICU → initial therapy with anti-pneumococcal β-lactam (ceftriaxone, ertapenem, or ampicillin-sulbactam) + macrolide (cover atypicals), or monotherapy with a FQ
-ICU patients → initial therapy same as non-ICU, add vanco if suspecting MRSA, add anti-pseudomonal drug for COPD or frequent steroid or AB users (β-lactam + FQ)
-Clinical improvement should occur within 72 hours
-Switch from IV to orals with clinical improvement
-F/u CXR for patients over 50 at 7-12 weeks

Empiric HAP/HCAP/VAP Management
-Need to cover MRSA: vanco or linezolid
-Need to cover *Pseudomonas* and other gram negs: Zosyn, cefepime, ceftazidime, aztreonam (only for severe PCN allergy b/c it's not as effective)
-Need additional coverage for gram negs and atypicals: cipro, levo, gentamycin, tobramycin, or carbapenem

ENDOCRINOLOGY

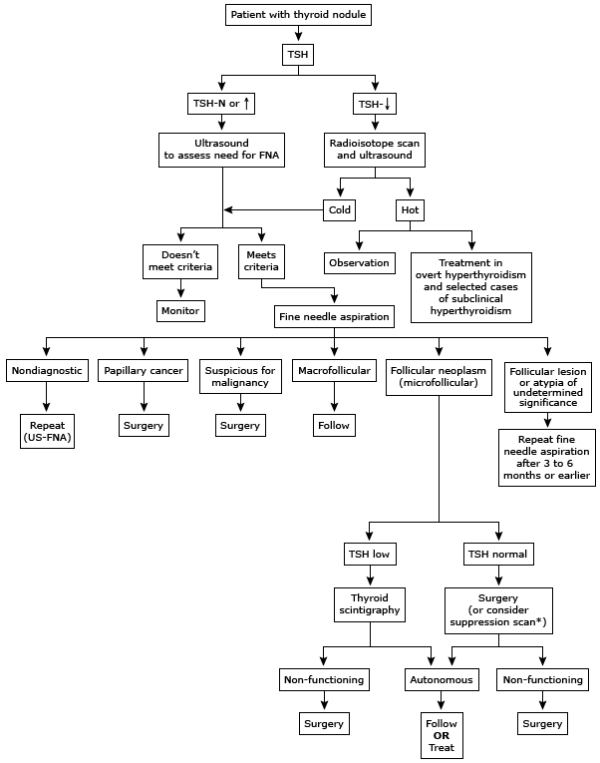
Thyroid Neoplasms and Thyroid Nodules

-More common in women
-Not associated with hypo or hyperthyroidism
-Characteristics suggesting malignancy: age < 20 or > 70, solid or complex, cold nodules, single nodule, nodule that grows with TH replacement, hoarseness or obstruction symptoms, hx of neck or head radiation

Workup
-Check TSH for all patients
-Can check autoantibodies
-Refer for neck US to assess for size and shape
-Low TSH → radionuclide scan to check hot/coldness of nodule
-Refer for FNA if US results show risk of malignancy
-Refer for resection if FNA cytology is suspicious

Management
-Follow low-risk nodules every 6 months with palpation and US
-Benign nodules may disappear over time
-Surgical removal if concern for malignancy

Prognosis
-10% of palpable nodules will be malignant
-Surgical complications: recurrent laryngeal nerve damage, parathyroid damage



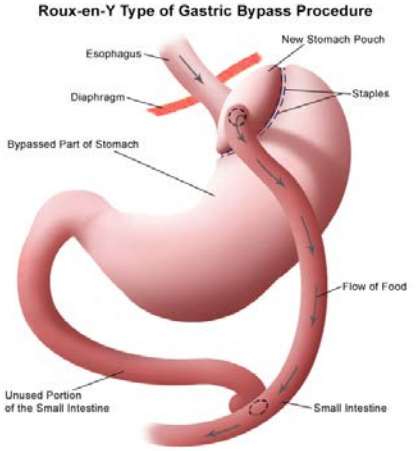
Benign Thyroid Neoplasms	Malignant Thyroid Neoplasms	
Follicular cell adenoma	**Papillary adenocarcinoma** -Most common type of thyroid cancer **Follicular adenocarcinoma** -Diagnosis usually occurs during evaluation of a cold thyroid nodule -Treatment is through radioactive iodine ablation with hormone replacement to suppress TSH **Hurthle cell thyroid cancer**	**Medullary Adenocarcinoma** -Arises from C-cells of the thyroid -Age > 40 -Associated with MEN type 2 -Regional lymph node involvement with mets to the lung, bone, and liver -Evaluate serum calcitonin, CEA, Ca, and plasma fractionated metanephrines -Very deadly

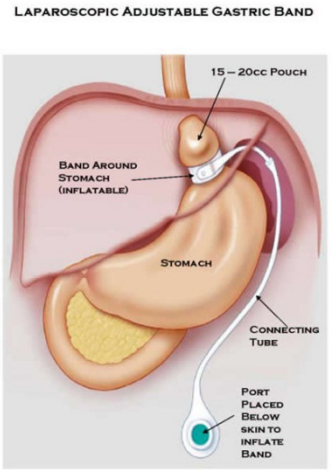
Obesity

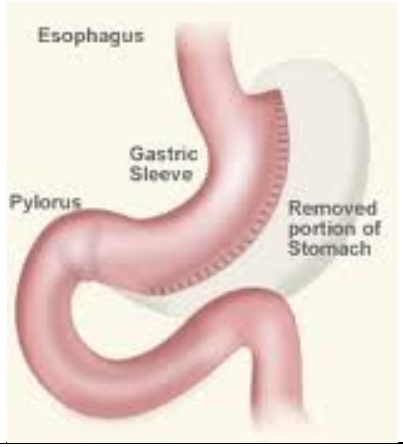
-Overweight = BMI 25-29.9
-Obesity = BMI > 30 → greater risk of DM, stroke, CAD, early death

Pharmacologic options
-Catecholaminergics (phentermine, diethylpropion, mazindol): short-term use only
-Orlistat: inhibits lipase

Bariatric surgery
-NIH recommends limiting to patients with BMI > 40, or > 35 if obesity complications are present
-Results in significant reduction in deaths from obesity
-Options: adjustable "lap band", sleeve gastrectomy, Roux-en-Y bypass







EENT

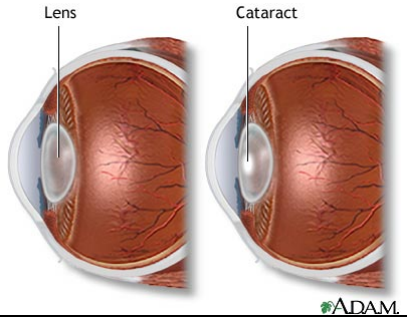
Cataracts

-Opacification of the lens

Etiologies
-Age
-Steroids
-Diabetes
-Electrocution
-Congenital anomaly
-Trauma

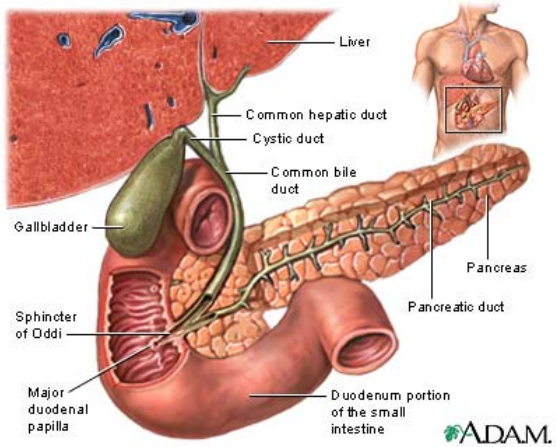
Signs & symptoms
-Gradual loss of vision
-Blurred or smoky vision
-Glares
-Decreased vision in bright light or at night

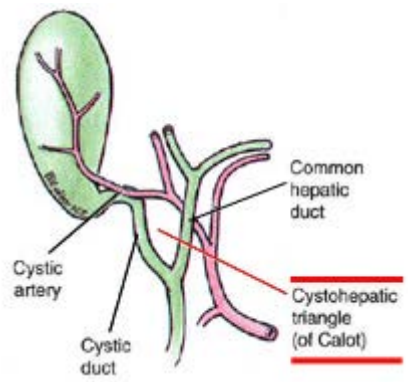
Management
-Surgical removal when it interferes with ADLs with replacement with an artificial lens

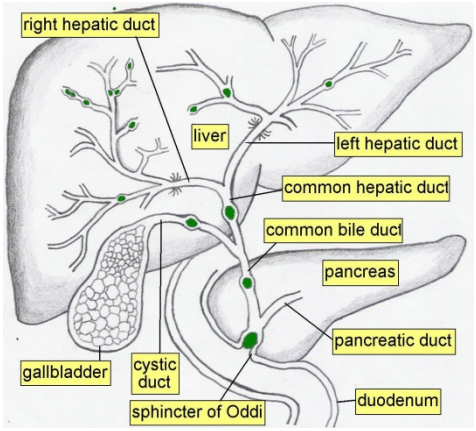


GASTROENTEROLOGY

Biliary Disease







Biliary Disease	Info	Risk Factors	Signs & Symptoms	Differential	Workup	Management	Complications
Cholelithiasis	-Most stones are cholesterol, fewer are pigment stones	-Obesity -Rapid weight loss -DM -High carb diet -Crohn's disease -High TG -Pregnancy -Hemolytic anemia -Cirrhosis -Estrogens -TPN -Native American	-Frequently asymptomatic and discovered incidentally -Biliary colic: infrequent episodes of steady severe pain in epigastrium or RUQ with radiation to right scapula (Boas' sign) -May be ppt by large or fatty meal	-Perforated peptic ulcer -Acute pancreatitis -Appendicitis with high-lying appendix -Perforated colonic carcinoma -Liver abscess -Hepatitis -Pneumonia with pleurisy	-RUQ US	-Lap chole is surgical treatment of choice -Nonsurgical candidates: dissolution therapy, shockwave lithotripsy, percutaneous stone removal or drain placement	-Bile peritonitis -Subhepatic abscess -Gallstone pancreatitis -Pancreatitis s/p endoscopic sphincterotomy
Cholecystitis	-Usually due to stone lodged in cystic duct (but can be acalculous) → secondary bacterial infection (usually *Klebsiella* or *E. coli*)		-Severe pain and tenderness in right hypochondrium or epigastrium -N/v -Fever -Murphy's sign, guarding, rebound tenderness -Can be jaundiced -Referred shoulder pain not commonly seen -Dark urine or light stools with choledocholithiasis		-CBC shows leukocytosis -↑ serum bili, AST/ALT, ALP Amylase may be ↑ -Cholecystitis: US is initial test of choice -HIDA if suspecting acalculous cholecystitis -Choledocholithiasis: endoscopic US or MRCP are tests of choice -Cholangitis: ERCP or abdominal US shows common bile duct dilation		
Choledocholithiasis	-Stone has traveled to common bile duct					-Endoscopic sphincterotomy to allow stone passage through sphincter of Oddi	
Ascending Cholangitis	-When choledocholithiasis progresses to infection -Consider in any pt with h/o biliary disease who presents with recurrent symptoms -Mortality of 50%		-Charcot's triad: fever, RUQ pain, jaundice -Reynold's pentad: Charcot's + confusion and hypotension			-Zosyn or ceftriaxone + metronidazole -Biliary drainage via ERCP	

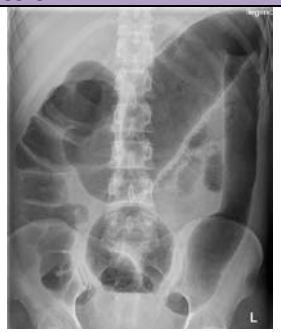
Toxic Megacolon

-A potentially lethal complication of colitis that is characterized by total or segmental nonobstructive colonic dilation + systemic toxicity

Etiologies
-IBD
-Infectious colitis
-Ischemic colitis
-Volvulus
-Diverticulitis
-Obstructive colon cancer

Signs & symptoms
-Severe bloody diarrhea

Workup
-Abdominal plain film showing R colon dilation > 6 cm, dilation of transverse colon, absence of normal colonic haustral markings, and air-filled crevices between large pseudopolypoid projections extending into the gut lumen



Management
-Fluid resuscitation
-Correction of abnormal labs
-IV vanco and metronidazole
-Complete bowel rest
-Bowel decompression with NGT
-Surgical consult for subtotal colectomy with end-ileostomy for pts not improving on medical management

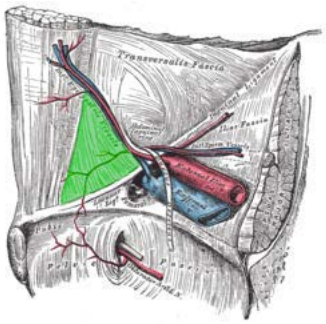
Hernias

Inguinal Hernias

-Risk factors: h/o or FH of hernia, older age, chronic cough, chronic constipation, strenuous exercise, abdominal wall injury, h/o AAA, smoking, ascites
-Differential: hydrocele, inguinal adenitis, varicocele, ectopic testis, lipoma, hematoma, sebaceous cyst, hidradenitis, psoas abscess, lymphoma, metastatic neoplasm, epididymitis, testicular torsion, femoral hernia, femoral adenitis, femoral aneurysm
-Workup: groin US if uncertain of mass etiology

Direct Inguinal Hernia

-When intestine plows through weak abdominal tissue in area of Hesselbach's triangle (bordered by inguinal ligament, inferior epigastric vessels, and rectus abdominis)



Causes
-Increased intra-abdominal pressure
-Weakening of tissue due to age or smoking

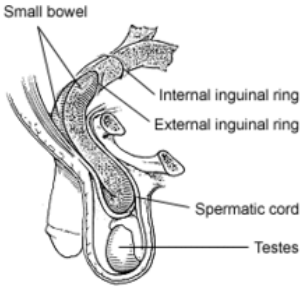
Signs and Symptoms
-Bulge in area of Hesselbach's triangle
-Only mild, intermittent pain or discomfort unless incarcerated or strangulated
-Signs of sepsis in an incarcerated hernia

Management
-If only mild symptoms or asymptomatic → consider watchful waiting
-Attempt manual reduction of incarcerated hernias
-If symptomatic → surgical hernia repair, usually laparoscopic if bilateral or recurrent

Prognosis
-High post-op recurrence

Indirect Inguinal Hernia

-Most common type of hernia
-Occurs when intestine slips through an abnormally open inguinal canal (patent processus vaginalis)



-Variation is a *pantaloon hernia* which is a combined direct and indirect inguinal hernia where both hernias straddle each side of the inferior epigastric vessels

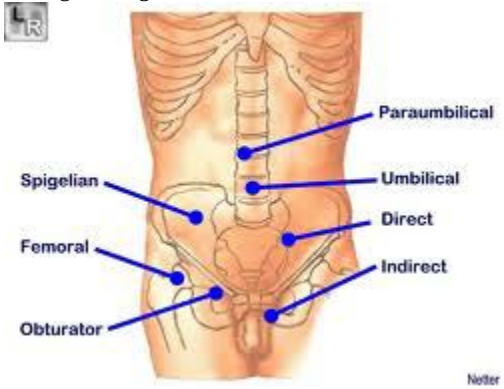
Signs and Symptoms
-Bulge in scrotum due to herniation through inguinal canal

Management
-Higher risk of strangulation so surgical repair is indicated

Prognosis
-Risk of postoperative pain syndrome from damage to ilioinguinal nerve

Femoral Hernia

-Occurs through the femoral canal, which is just below the inguinal ligament



-Bordered by femoral vein laterally, lacunar ligament medially, and Cooper's ligament below
-More common in females

Signs and Symptoms
-Commonly presents emergently as an incarceration or strangulation

Management
-Surgical repair

Umbilical Hernia

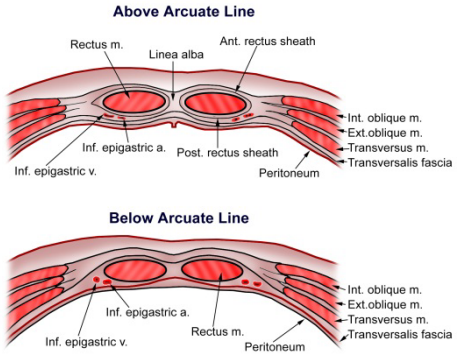
-Technically a type of ventral hernia since it is an abdominal wall defect
-Caused by open umbilical ring, which usually closes in all kids by 5 years but may be slower to close in black children
-May interfere with feeding if it contains bowel
-Rarely become incarcerated or strangulated in kids

Management
-Referral for surgical repair indicated when hernia is incarcerated, extremely large, or symptomatic

Ventral Hernias

-Caused by defects in the abdominal wall
-Diastasis recti is an abdominal wall defect but is not a true hernia and does not require repair

Types
-Incisional: occurs through site of previous surgical incision
-Epigastric: occur between umbilicus and xiphoid process
-Spigelian: hernia through Spigelian fascia



Signs and Symptoms
-Spigelian hernias may not be detected on physical exam but pts present with mid or lower abdominal pain and swelling lateral to rectus muscle

Workup
-CT to visualize Spigelian hernia

Management
-Most incisional hernias should be repaired (mesh is preferred) due to risk of incarceration unless very small or large, or upper abdominal and asymptomatic
-Epigastric hernias have low risk for incarceration and only need repair if symptomatic
-Surgical repair of Spigelian hernias due to high risk of strangulation

Other Hernias

Internal Hernia	Obturator Hernia	Littre Hernia	Richter Hernia	Richter Hernia
-Occurs after abdominal surgeries when the bowel gets trapped as a result of new anatomic relationships	-Occurs when small bowel herniates into the obturator canal	-Any groin hernia that contains Meckel's diverticulum	-Occurs when a knuckle of bowel protrudes into a hernia defect, but only a portion of the circumference is involved and the bowel lumen remains patent	-Any hernia that contains intra-abdominal organs

Esophageal Neoplasms

Benign

Leiomyoma
-Tumor of smooth muscle
-Surgical removal if symptomatic

Adenoma
-Tumor arising from glandular tissue
-Usually found in areas of Barrett's esophagus

Esophageal Papilloma
-Associated with transformation to SCC

Malignant

Usually occurs in males 50-70

Types
-Squamous cell carcinoma: occurs in the upper 2/3 of the esophagus, risk factors are alcohol use, tobacco, achalasia, caustic esophageal injury, head and neck cancers, Plummer-Vinson syndrome, black ethnicity, male
-Adenocarcinoma: occurs in the lower 1/3 of the esophagus, risks are Barrett's esophagus, white ethnicity, males
-Lymphoma: very rare in esophagus
→ Recent trend towards adenocarcinoma

Signs & symptoms
-Progressive solid food dysphagia
-Weight loss
-Usually is late stage by time patient is symptomatic

Workup
-CXR showing mediastinal widening, lung or bony mets
-Barium swallow showing many infiltrative or ulcerative lesions and strictures
-Chest CT
-Endoscopic US for staging

Management
-Surgical resection with gastric pull-up or colonic interposition
-Palliative radiation
-Chemo
-Palliative stenting

Gastric Neoplasms

Benign	Malignant	
Gastric Polyps -Many can undergo malignant transformation so these should be biopsied **Others** -Lipoma -Fibroma -Glomus tumor -Hemangioma	**Leiomyosarcoma** -Arises from smooth muscle -Rare -Requires local resection, rarely metastasizes **Lymphoma** -Either MALT or diffuse large B cell lymphoma **Sarcoma** -GIST	**Adenocarcinoma** -High incidence in Korea, Japan, and China -Usually occurs after age 60 -Risk factors: pickled foods, salted foods, smoked foods, *H. pylori*, atrophic gastritis, polyps, radiation -Signs & symptoms: early disease is asymptomatic; indigestion, nausea, early satiety, anorexia, weight loss, palpable stomach, hepatomegaly, pallor, Virchow's nodes (L sided supraclavicular) or Sister Mary Joseph nodes (umbilical); advanced: pleural effusions, SBO, bleeding -Workup: EGD preferred, endoscopic US to assess tumor depth, barium swallow, CT of pelvis, chest, and abdomen to look for mets -Management: depends on stage; resection (Billroth or roux-en-Y), chemo, radiation, adjuvants if needed -Prognosis: difficult to cure, most will die of recurrent disease even after resection

Pancreatic Neoplasms

Benign	Malignant
-Usually asymptomatic and found incidentally -Eval further using MRI, endoscopic US with FNA **Serous Cystadenoma** -Most common benign pancreatic lesion -Low malignancy potential -Resection not recommended **Mucinous Cystadenoma** -Moderate malignancy potential **Intraductal Papillary Mucinous Neoplasm** -High malignancy potential if located within main duct **Solid Pseudopapillary Neoplasm** -Low to moderate malignancy potential -Should be resected	-Resection via Whipple procedure **Ductal Adenocarcinoma** -Includes signet ring cell carcinoma, adenosquamous carcinoma, undifferentiated carcinoma, and mucinous non-cystic carcinoma -Accounts for > 95% of pancreatic malignancies -Typically occurs in ages 70-80 -Most commonly in the head of the pancreas -Risk factors: tobacco use, chronic pancreatitis, exposure to dye chemicals, DM2 in nonobese person arising after age 50, history of partial gastrectomy or cholecystectomy, genetic factors including BRCA2 -Signs & symptoms: pain, steatorrhea, weight loss, jaundice, Courvoisier's sign (palpable gallbladder due to compression of bile duct), Trosseau's sign (hypercoagulable state created by the malignancy → migratory thrombophlebitis throughout body) -Workup: ALP, bili, initial imaging with RUQ US and ERCP, CT for "double duct sign" (dilation of the common bile and main pancreatic ducts), endoscopic US if other imaging is not convincing, confirmation with histologic diagnosis -Management: serial CA-19-9 to follow trend; surgical resection + radiation (if there is no invasion, lymphatic involvement, or mets), locally advanced disease → radiation only; chemo, pain control, and palliative stents for metastatic disease -Prognosis: half of all pancreatic cancers are metastatic by the time of diagnosis, with a life expectancy of 3-6 months; resectable disease survival is < 1.5 years; locally advanced disease survival is 6-10 months **Others** -Acinar cell carcinoma -Pancreatoblastoma

Small Bowel Neoplasms

-Malignant tumors very rare when compared to incidence in large bowel	**Signs and Symptoms** -Crampy, intermittent abdominal pain	**Risk Factors for Malignancy** -Familial cancer syndromes: HNPCC, Peutz-Jeghers, FAP

-Usually located in the ileum	-Weight loss, nausea, vomiting -GI bleed -Intestinal obstruction -Usually asymptomatic if benign	-Chronic inflammation: IBS, celiac disease -Intake of alcohol, refined sugar, red meat, or salt-cured or smoked foods -Smoking? -Obesity?

Benign	Malignant	
Adenoma -Villous adenomas can transform to malignancy -Duodenal adenomas associated with increased risk for colon cancer -Tubular adenomas most common in the duodenum **Leiomyoma** -Arise from intestinal submucosa **Lipoma** -Arise from submucosal or subserosal adipose **Other benign small bowel tumors:** desmoid tumor, hemangioma, fibroma	**Adenocarcinoma** -Arises from glandular tissue -Occurs in duodenum **Carcinoid Tumors** -A neuroendocrine cell tumor arising from the enterochromaffin cells of the gut -Most common type of small bowel malignancy -Usually occurs in the ileum -With mets can have carcinoid syndrome: watery diarrhea, flushing, sweating, wheezing, dyspnea, abdominal pain, hypotension	**Lymphoma** -Almost always non-Hodgkin -Includes MALT lymphoma, diffuse large B-cell lymphoma, mantle cell lymphoma, and Burkitt lymphoma **Sarcoma** -Tumor of the mesenchymal cells -Most common type is GI stromal tumor (GIST): may be considered benign but all have the potential for malignant transformation; should be resected if > 2 cm; consider imatinib (Gleevec) as neoadjuvant prior to resection if large; rarely metastasizes

Colorectal Neoplasms

Benign	Malignant: Adenocarcinoma	
Non-Neoplastic Polyps -Hyperplastic polyps = not pre-malignant → more frequent screening not needed -Hamartomatous polyps -Inflammatory polyps -Lymphoid polyps **Neoplastic Epithelial Polyps** -These are pre-malignant → need screens more frequently for monitoring -Tubular adenoma -Tubulovillous adenoma -Villous adenoma **Leiomyoma** -Tumor of smooth muscle -Can occur in colon or rectum **Others** -Lipoma -Neuroma -Hemangioma -Lymphangioma	-Accounts for 95% of primary colon cancers -30% will be in the rectum, 25% on the right colon -Risk factors: age, FH (up to 30% have a genetic component), DM2, metabolic syndrome, ethnicity, IBD, high red meat or processed meat consumption, inactivity, obesity, smoking, heavy alcohol use -Prevention: diet with plant foods, healthy BMI, limited red meats, physical activity **Associated Familial Syndromes** -FAP: also incurs risk of thyroid, pancreas, duodenal, and gastric cancers -HNPCC: associated with endometrial, ovarian, gastric, urinary tract, renal cell, biliary, and gallbladder cancers -Most occur after age 50 **Screening** -Begin assessing risk at age 20 -Begin screening at 40-45 for AA patients, at 50 for all other patients of average risk; continue until life expectancy is estimated to be less than 10 years or 85 years at the latest -Begin screening those with FH at least 10 years before the age at which the youngest affected family member was diagnosed -Colonoscopy every 10 years -CT colonography or flexible sigmoidoscopy every 5 years -FOBT annually for patients in whom imaging or visualization is not possible	**Signs and symptoms** -Rectal bleeding -Iron deficiency anemia -Fatigue and weight loss -Obstruction -Change in stool quantity or caliber -Abdominal mass or pain -Weakness -Mets to the liver and lung **Workup** -Colonoscopy for biopsy -Abdominal/pelvis CT for staging (“apple core” lesions) -CXR for mets -Labs: CBC, CMP, baseline CEA for f/u -PET **Management** -Early stage tumors may be removed endoscopically -Hemicolectomy with lymph node dissection -Local treatment of mets -Chemo to eradicate micromets -Radiation not typically used due to its high toxicity in the gut

Liver Neoplasms

-Most pts are asymptomatic
-Diagnostic approach depends on risk factors of patient and size of lesion
-Workup: ↑AFP indicates malignancy

Benign

Hemangioma
-The most common benign liver tumor
-Small, asymptomatic
-Finding is incidental

Hepatic adenoma
-Associated with long-term estrogen use
-Can rupture and bleed, so it should be resected

Focal nodular hyperplasia
-May be a response to a congenital malformation
-Should be resected

Others
-Hamartoma
-Cysts: simple, infectious, polycystic liver, biliary cystadenoma, Von Meyenburg complex

Malignant

-Risk factors: EtOH, autoimmune hepatitis, viral hepatitis, alpha-1 antitrypsin deficiency, Wilson's disease

Hepatocellular carcinoma
-Usually occurs with chronic liver disease or cirrhosis
-High risk pts should be screened every 6 months via US
-Heightened suspicion for malignancy in previously compensated cirrhosis pts who develop decompensation
-Lab findings will be nonspecific, but baseline AFP will rise
-Diagnostic imaging shows multiphasic tumor
-Treat by resection or radiofrequency ablation, palliative embolization, or liver transplant

Metastatic Disease
-The most common malignant hepatic neoplasms in the Western world

Biliary Cancer

-90% are fatal
-Pts are usually asymptomatic
-Endoscopic US is imaging of choice for workup

Risk Factors
-Gallbladder polyps
-Gallstone disease
-Congenital biliary cysts
-Anomalous pancreaticobiliary junction
-Chronic infection
-Porcelain gallbladder

Cholangiocarcinoma
-Arises in bile ducts
-Risk factors: primary sclerosing cholangitis, choledochal cysts, *Clonorchis sinensis* infection
-S/s: Courvoisier's sign (palpable nontender gallbladder + jaundice)

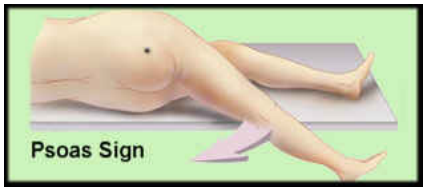
Gallbladder Adenocarcinoma
-Accounts for 80% of gallbladder malignancies
-S/s: RUQ pain, weight loss, anorexia, nausea, obstructive jaundice, ascites
-Management: surgical resection

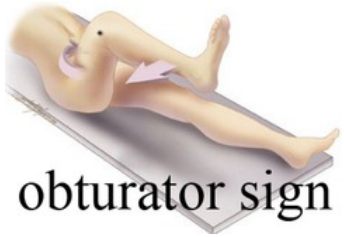
Other Malignancies
-SCC
-Neuroendocrine tumors
-Lymphoma
-Sarcoma

Appendicitis

-Usually caused by a fecalith

Signs & Symptoms
-Dull periumbilical pain that progresses to focal sharp pain with RLQ radiation
-Anorexia, n/v
-Low grade fever
-McBurney's point tenderness
-Obturator sign
-Psoas sign
-Rovsing's sign





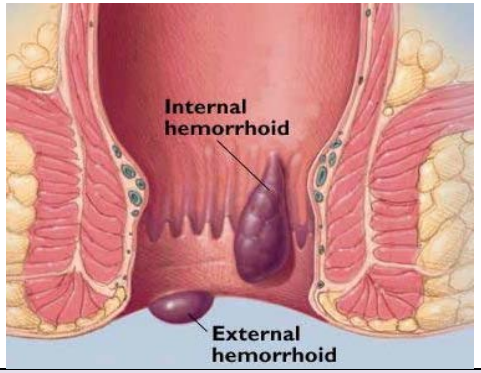
Workup
-CBC may show leukocytosis (but can be late finding)
-Abdominal CT preferred in adults and nonpregnant women
-US preferred in peds and pregnancy
-If probability for appendicitis is high, can go straight to surg consult in many cases

Management
-Surgical consult
-Presence of abscess or rupture will require cipro or Zosyn treatment first followed by appendectomy in 3-4 weeks in kids or colonoscopy in adults (colon ca can manifest as appendiceal abscess)

Hemorrhoids

-Engorgement of the venous plexuses of the rectum, anus, or with; with protrusion of the mucosa, anal margin, or both
-Classified as internal or external based on position in relation to dentate line

Causes
-Constipation or straining
-Portal HTN
-Pregnancy



Internal Hemorrhoids

Classification

- Grade I hemorrhoids are visualized on anoscopy and may bulge into the lumen but do not extend below the dentate line.
- Grade II hemorrhoids prolapse out of the anal canal with defecation or with straining but reduce spontaneously.
- Grade III hemorrhoids prolapse out of the anal canal with defecation or straining, and require the patient to reduce them into their normal position.
- Grade IV hemorrhoids are irreducible and may strangulate.

Signs & symptoms
-Painless bleeding after defecation
-Visible during anoscopy
-Not palpable or painful on DRE

Management
-1% hydrocortisone
-Refer to GI for rubber band ligation if prolapsed (bulging out of anus)

External Hemorrhoids

Signs & symptoms
-Rarely bleed but are extremely painful, especially if thrombosed (exquisitely tender blueish perianal nodule)
-Itching
-Visible externally on perianal exam

Management
-Sitz bath
-1% hydrocortisone
-Stool softeners
-May need to remove thrombosed clot
-Surgical referral if refractory to medical management

Inflammatory Bowel Disease

-Both are autoimmune
-Incidence highest in 15-40 year olds and > 60 year olds
-Tend to run in families
-Extraintestinal manifestations possible: eye (uveitis, episcleritis), skin (erythema nodosum, pyoderma gangrenosum), liver, joints
-Diagnosis relies on a combination of endoscopy, histology, radiography, labs, and clinical data
-Not everyone needs continued treatment or any treatment at all; treat the affected area
-Response to any given treatment is only 30-70%
-Use steroids sparingly to induce remission
-During flare, check WBCs, H/H, f/u with endoscopy referral if not improving

Crohn's Disease

-Can affect any portion of GI tract from lips to the anus and has transmural involvement, however most common site is ileum
-Disease skips areas → skip lesions
-Bouts of flares and periods of remission

Signs & symptoms
-Aggravated by smoking
-Fistulas and abscesses
-Perianal disease
-Obstructions
-Prolonged diarrhea and abdominal pain
-Fatigue
-Weight loss

Differential
-Ulcerative colitis
-IBS
-Appendicitis
-*Yersinia enterocolitica* enteritis
-Mesenteric adenitis
-Intestinal lymphoma
-Segmental colitis: ischemia, TB, amebiasis, *Chlamydia*
-Diverticulitis with abscess
-NSAID-induced colitis
-Perianal fistula: lymphogranuloma venereum, cancer, rectal TB

Workup
-Labs are not specific or reliable
-Initial imaging is upper GI series with small bowel follow-through
-Colonoscopy shows cobblestoning with varying degrees of mucosal ulceration

Management
-Steroids for flares
-Gentle wiping, sitz baths, perianal pads for perianal disease
-Low-roughage diet only for obstructive symptoms
-Mesalamine trials show that it is not effective for Crohn's
-Antibiotics during flares have shown little to no efficacy
-Steroid courses PRN: budesonide has fewer side effects
-Immunomodulating agents for pts unresponsive to steroids or requiring chronic steroids (refer to rheumatology): azathioprine, mercaptopurine, methotrexate
-Annual colonoscopy recommended with > 8 year disease history

Complications
-Small bowel strictures
-Fistulae to bowel, bladder, vagina, or skin
-High oxalate from malabsorption of ingested fat (binds Ca) → kidney stones, gallstones
-Often require surgical management

Ulcerative Colitis

-Disease begins in the rectum and is limited to the colon with superficial penetration of the mucosal wall
-Bouts of flares and periods of remission

Signs & symptoms
-Proctitis
-Tenesmus
-Lower abdominal or pelvic cramping
-Bloody diarrhea
-Mucus or pus per rectum
-Fever

Differential
-Infectious colitis: *Salmonella*, *Shigella*, *Campylobacter*, amebiasis, *C. diff*, enteroinvasive EC, CMV
-Ischemic colitis
-Crohn's disease
-Diverticular disease
-Colon cancer
-Antibiotic-associated diarrhea or pseudomembranous colitis
-Infectious proctitis: gonorrhea, *Chlamydia*, HSV, syphilis
-Radiation colitis or proctitis

Workup
-Labs: ↓ serum albumin, CBC for anemia, ↑ ESR
-Negative stool cultures
-Sigmoidoscopy with biopsies showing crypt abscesses, chronic colitis for dx
-Barium enema may show "stovepipe" colon due to loss of haustral folds

Management
-Distal colitis → DOC is topical mesalamine, hydrocortisone suppositories PRN, second-line therapy is oral sulfasalazine
-Mild-mod colitis (above sigmoid colon) → oral 5-ASAs, add hydrocortisone foam or enema if needed, refer for immunomodulating agents if no response
-Severe flare → send to ED for hospitalization
-Screening colonoscopies ever 1-2 years for patients with > 8 year history of disease

Complications
-Toxic megacolon
-Extension of colonic disease
-Perforation
-Strictures

Bowel Obstruction

-Obstruction can be mechanical (intrinsic: post-op) or functional (paralytic: electrolyte abnormality, DM)

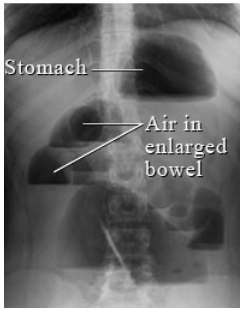
Types
-Simple obstruction = blood supply intact
-Strangulated obstruction = compromised blood supply
-Closed loop
-Obstruction can be complete, partial, or intermittent

Causes of Large Bowel Obstruction
-#1 is neoplasms
-Diverticular disease
-Volvulus: usually sigmoid or cecal
-Adhesions

Causes of Small Bowel Obstruction
-#1 cause is adhesions from previous surgeries
-Hernias
-Neoplasm
-Strictures
-Intussusception
-Meckel's diverticulum
-Volvulus
-Intramural hematoma

Signs & Symptoms
-Crampy, generalized abdominal pain
-No signs of peritonitis
-Abdominal distension with diffuse midabdominal tenderness to palpation
-Suspect ischemia with localized TTP
-Nausea
-Vomiting, may have coffee-ground emesis or feculent material
-Reduced urine output
-Inability to pass gas
-However, pts may still be passing gas and having flatus up to 12-24 hours after onset of obstruction, since the colon requires this much time to empty distal to the obstruction

Differential
-Paralytic ileus: occurs post-op or after peritonitis (will see dilated small bowel in presence of dilated colon on KUB)
-Intestinal pseudo-obstruction: recurrent abdominal distension in the setting of no mechanical obstruction
-Gastric outlet obstruction
-Intestinal malrotation



Workup
-BMP shows electrolyte derangements from fluid shifts
-Check lactate if concerned for bowel strangulation or ischemia
-Initial imaging with KUB shows distended loops of small bowel, air-fluid levels, free air under diaphragm if perforated, "swirl sign" where bowel has twisted on its mesentery, and "bird's beak" or "corkscrew" if volvulus is also present
-Can f/u KUB with CT for further localization
-SBO in absence of prior abdominal surgery should trigger malignancy workup

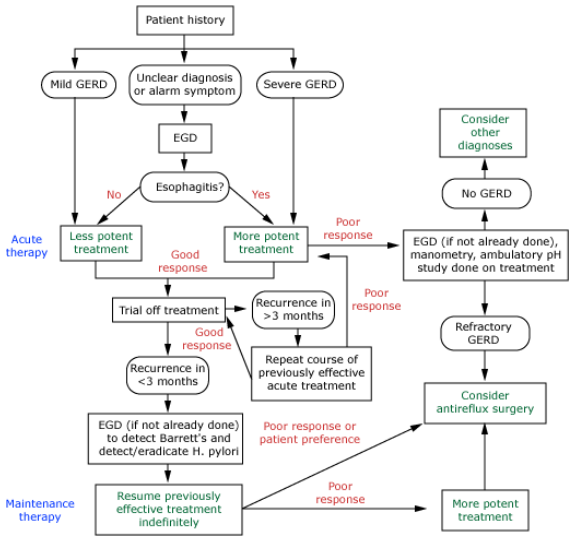
Management
-IVF
-Antibiotics
-NPO with NGT decompression
-Volvulus: rectal tube for decompression followed by surgical repair to prevent recurrence
-Ischemia or perforation: immediate surgical intervention

GERD

-Common cause is impaired LES function
-Can be associated with hiatal hernia
-Severity of symptoms does not correlate with tissue damage

Signs & Symptoms
-Heartburn 30-60 minutes after meals and/or on reclining
-Regurgitation of gastric contents
-Hoarseness
-Loss of dental enamel
-Relief with antacids
-Alarm symptoms: dysphagia, odynophagia, weight loss, iron deficiency anemia, symptom onset after age 50, symptoms persistent despite PPI therapy → refer for urgent workup and upper endoscopy
-Asthma

Differential
-PUD
-Gastritis
-Non-ulcer dyspepsia
-Cholelithiasis
-Angina pectoris
-Infectious esophagitis: *Candida*, CMV, HSV
-Pill esophagitis
-Esophageal motility disorder
-Radiation esophagitis
-Gastrinoma
-Delayed gastric emptying



Management
-Mild, intermittent symptoms → lifestyle modifications like elimination of triggers, weight loss, avoid lying down after meals, elevate HOB; PRN antacids
-Oral H2 agonists have a 30 minute delay
-Troublesome frequent symptoms → PPI for 4-8 weeks, then consider chronic PPI use if symptoms relapse or persist
-Known GERD complications → long-term PPI therapy
-If unresponsive to PPI → verify drug compliance, refer for endoscopy
-Antireflux surgery such as Nissen fundoplication is last resort

Complications
-Barrett esophagus occurs in 10% of patients with chronic reflux → screening EGD recommended for adults 50 or older with 5-10 year history of GERD, and EGD q 3 years for patients with known Barrett
-Peptic strictures → progressive solid food dysphagia
-Esophagitis
-Asthma, laryngitis, or chronic cough

Anal Fissure

-A tear or erosion in the epithelium of the anal canal
-Acute or chronic

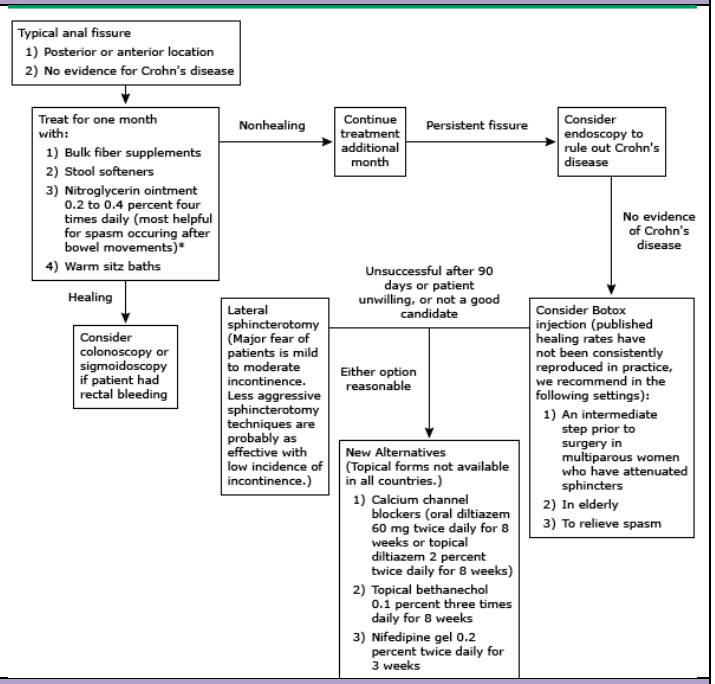
Causes
-Usually due to large or hard-to-pass stool
-Infectious: TB, syphilis, HIV, occult abscess
-Carcinoma
-Granulomatous disease
-IBD
-Prolonged diarrhea
-Anal sex
-Childbirth

Prevention
-Avoid constipation with high fiber and fluid intake
-Wiping with moist cloth

Signs and Symptoms
-Tearing pain with BMs, although less painful if chronic
-Small amount of bright red blood on toilet paper
-Usual location is posterior midline
-Perianal pruritus or skin irritation
-Acute fissures appear like a paper cut
-Chronic fissures usually have raised edges with external skin tags and hypertrophied pillae

Differential
-Perianal ulcer: IBD, TB, STDs
-Anorectal fistula: differentiate from fissure by tract formation

Management
-Stool softeners
-Sitz baths
-1% hydrocortisone cream
-2% nitroglycerine cream: ↑ blood flow and reduces pressure on internal anal sphincter
-Surgical consult if not improving in 6 weeks; possible need for internal sphincterotomy



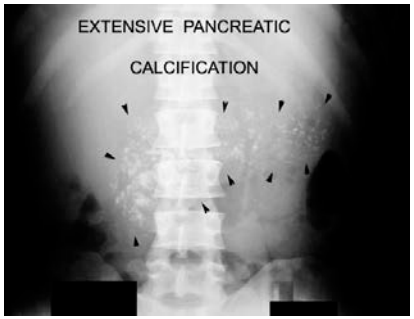
Chronic Pancreatitis

-Chronic inflammation leads to irreversible fibrosis of the pancreas

Etiologies
-Chronic alcohol use
-Chronic pancreatic duct obstruction
-Malnutrition
-Autoimmune
-Hereditary
-Idiopathic

Signs & symptoms
-Recurrent episodes of epigastric and LUQ pain
-Steatorrhea
-Fat soluble vitamin deficiency
-Diabetes

Workup
-Amylase and lipase won't be elevated because the pancreas is burned out by now
-Secretin stimulation test to see if the pancreas still works
-Abdominal x-ray showing pancreatic calcifications
-CT showing calcifications and atrophy
-ERCP showing "chain of lakes" or areas of dilation and stenosis along the pancreatic duct



Management
-Abstinence from alcohol
-Pancreatic enzyme replacement + PPI + low fat diet
-Insulin
-Surgical options for refractory cases: decompression, resection, or denervation procedures

Acute Pancreatitis

-Occurs with inappropriate activation of trypsin within the pancreas → enzymatic damage to the pancreas

Etiologies
-Gallstones are the most common cause
-Alcohol use
-Other obstructions: pancreatic or ampullary tumors, sphincter of Oddi dysfunction, pancreatic malformation
-Meds: diuretics, azathioprine, 6-mercaptopurine, sulfa drugs, ACEIs, HIV meds
-Infections: mumps, rubella, Coxsackie, echovirus, EBV, HIV
-Metabolic: ↑TG, hyperCa
-Toxins: methanol, ethanol, scorpion sting in Trinidad
-Vascular: vasculitis, ischemia
-Abdominal trauma
-Post-ERCP
-Inherited causes

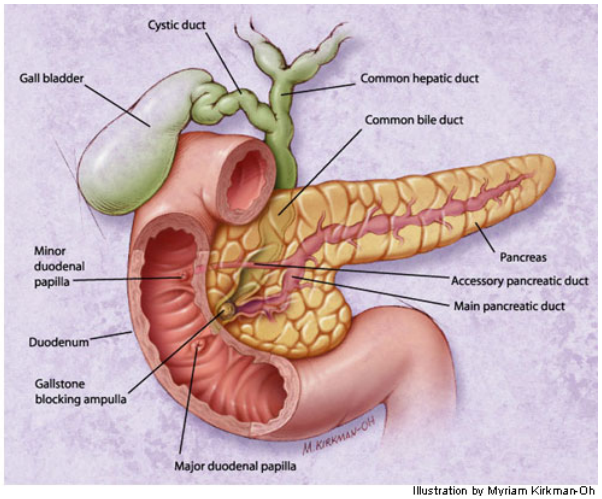
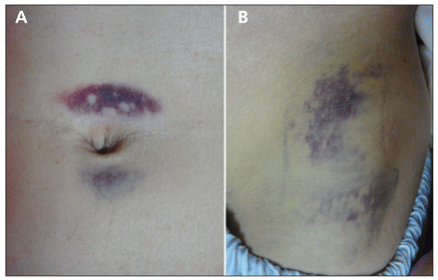


Illustration by Myriam Kirkman-Oh



Signs & symptoms
-Range of severity from mild illness to severe multiorgan failure
-Constant epigastric pain radiation to the back
-Nausea and vomiting
-Tachycardia secondary to hypovolemia from leaky vessels and 3rd spacing
-Fever
-Sepsis
-Icterus or jaundice if there is biliary obstruction
-Abdominal tenderness with rigidity and guarding
-Acute interstitial pancreatitis: mild, with pancreatic edema

-Acute necrotizing pancreatitis: severe, with necrosis of parenchyma and vessels → Gray-Turner's sign and Cullen's sign

Differential
-Acute cholecystitis or cholangitis
-Penetrating duodenal ulcer
-Ischemic colitis
-SBO
-AAA
-Nephrolithiasis
-Pancreatic pseudocyst

Workup
-↑ Amylase: not specific, can be ↑ in appendicitis, cholecystitis, perf, ectopic pregnancy, or renal failure; elevated for 24 hours
-↑ Lipase: more specific for pancreatitis, but can be elevated in renal failure; stays elevated for 3 days
→ Elevated amylase or lipase alone without clinical signs are NOT pancreatitis!
→ Amylase/lipase #s DON'T correlate to severity of disease!
-Bili will be elevated if there is an obstruction blocking it from leaving the liver
-Elevated BUN and hct with vol depletion
-US showing large, hypoechoic pancreas
-CT showing pancreatic enlargement and peripancreatic edema (imaging of choice for pancreatitis)
-MRCP or ERCP

Management
-If mild → NPO with IFV, correction of electrolytes, pain control; resolves in 3-7 days
-Severe → ICU monitoring, early NGT with tube feeds
-Acute necrotizing pancreatitis → imipenem
-Gallstone pancreatitis → sphincterotomy if suspecting risk of cholangitis, otherwise plan for lap chole after recovery

Prognosis
-Complications: inflammatory cascade can cause ARDS, sepsis, or renal failure; pancreatic necrosis or abscess, pancreatic pseudocyst

GENITOURINARY

Prostate Cancer

-Usually adenocarcinoma
-The most commonly diagnosed male cancer and 2nd leading cause of male cancer deaths
-Risk factors: age, black, high fat diet, FH, obesity
-No association with smoking, sexual activity, prior infections, or BPH

Screening
-USPSTF grade I for men up to age 75 and grade D after 75
-If patient elects, DRE and PSA should be done every 2 years
-PSA will be elevated in cancer, inflammation, or BPH, and will naturally rise as men age

Signs & symptoms
-Asymptomatic early in disease
-Later disease: obstructive urinary symptoms, hematuria, hematospermia
-Bone pain with mets

Workup
-Prostate biopsy guided by transurethral US, with scoring by Gleason system
-MRI
-PET if suspected mets
-CXR, LFTs for mets

Management
-Treatment based on life expectancy, general health, tumor characteristics
-Treatment is controversial for localize disease
-Radical prostatectomy
-Radiation
-Hormone therapy for advanced or metastatic disease

Bladder Neoplasms

-More common in men than women
-Risk factors: smoking, exposure to dyes and solvents

Signs & symptoms
-Painless hematuria
-Urinary frequency or urgency
-May be asymptomatic

Workup
-Cystoscopy is initial test of choice
-Repeat urine cytologies (low sensitivity)
-CT to assess local extent of disease
-Staging based on biopsy results and imaging

Management
-Neoadjuvant chemo
-If superficial, resection (usually total cystectomy with urinary diversion) ± intravesicular chemo
-If advanced, combo chemo ± radiation

Prognosis
-Early disease has > 80% survival

Benign Bladder Neoplasms

Low-Grade Intraurothelial Neoplasia

Urothelial Papilloma
-Can have malignant potential

Inverted Papilloma
-Can have malignant potential

Malignant Bladder Neoplasms

Carcinoma In Situ

Squamous Cell Carcinoma
-More common in areas of the world with schistosomal infections
-Aggressive

Adenocarcinoma
-Aggressive

Small Cell Carcinoma
-Neuroendocrine in origin
-Aggressive clinical course with poor prognosis

Metastatic Disease
-Commonly from the colon or rectum, prostate, or cervix

Invasive Urothelial Cell Carcinoma
-AKA transitional cell carcinoma
-Most common form of bladder cancer in US

Testicular Cancer

-Risk factors: cryptorchidism, abnormalities in spermatogenesis, FH
-Most commonly germ cell tumor, but cana lso be stromal tumor

Screening
-USPSTF grade D in asymptomatic adolescents and adult males

Signs & symptoms
-Firm, painless mass arising from the testis
-Scrotal pain
-Affected area is usually unilateral
-Signs of mets: cough, GI, back pain, neuro signs, supraclavicular lymphadenopathy

Investigation
-Scrotal US: distinguishes benign vs malignant and intra vs extratesticular
-Excisional biopsy
-β-hCG levels: will be elevated in some carcinomas and seminomas
-AFP: elevation excludes diagnosis of seminoma
-Chest, abdomen, and pelvis CT

Management
-Inguinal orchiectomy with f/u of tumor markers
-May need chemo

Prognosis
-High survival rate if caught early

Vulvar Neoplasms

-Vulvar intraepithelial neoplasia (VIN) is a premalignant lesion that is difficult to distinguish or may exist in association with invasive squamous cell carcinoma, lichen sclerosus, or lichen planus
-Malignant lesions include squamous cell carcinoma (90% of vulvar cancers), melanoma, and basal cell carcinoma

Risk Factors
-HPV infection
-Immunosuppression
-Cigarette smoking
-Lichen sclerosus (can transform to SCC)

Differential
-Flesh-colored lesion: sebaceous gland, vestibular papillae, skin tag, cyst, wart, molluscum contagiosum
-White lesion: lichen sclerosus, lichen simplex chronicus, vitiligo
-Brown, red, or black lesion: could be anything, need to biopsy

Signs & Symptoms
-Vulvar pruritus and pain
-Visible or palpable abnormality, may be in multiple locations
-Dysuria

Workup
-Any lesion not previously known on the vulva warrants biopsy via physical exam or colposcopy

Management
-Wide local excision of VIN if high risk based on lesion characteristics and pt age
-Laser ablation or topical therapy with imiquimod for VIN lesions that would cause significant vulvar mutilation if excised
-Excision of malignant lesions with inguinofemoral lymph node evaluation ± chemo or radiation

Prognosis
-VIN recurs in 30% of women and 4-8% will go on to develop locally invasive vulvar cancer

Renal Neoplasms

-"Small renal mass" is often detected incidentally and defined as a contrast-enhancing mass < 4 cm; most are renal cell carcinomas

Signs & Symptoms
-Most are asymptomatic
-Hematuria
-Paraneoplastic syndrome
-Abdominal or flank mass
-Abdominal pain

Differential
-Polycystic kidney disease

Workup
-Imaging can't reliably differentiate a benign tumor from RCC
-Dedicated renal CT or MRI for incidental lesions
-Surgical resection for masses 1-4 cm
-Percutaneous biopsy for low malignancy suspicion or for nonsurgical candidates

Management
-Active surveillance if < 1 cm

Prognosis
-Neither tumor size at diagnosis nor growth rate are accurate predictor of malignancy status

Benign Renal Neoplasms

Simple Renal Cyst

Renal Oncocytoma

Cystic Nephroma

Angiomyolipoma

Metanephric Adenoma

Renal Medullary Fibroma

Malignant Renal Neoplasms

Renal Cell Carcinoma
-Accounts for 80% of renal cancers
-More common in men than women
-Risk factors: smoking, obesity, HTN, polycystic kidney disease, occupational exposures, prolonged NSAID use, chronic hep C, sickle cell disease
-Signs & symptoms: hematuria, flank pain or abdominal mass, cough, bone pain with mets, paraneoplastic syndromes
-Nephrectomy needed

Prognosis
-Good for cancers confined to renal capsule
-50-60% for tumors extending beyond capsule
-0-15% for node positive tumors

Ovarian Neoplasms

-Vary from annoying and benign to invasive and malignant
-Functional ovarian cysts (corpus luteum cyst or follicular cysts) are NOT considered to be neoplasms because they are a result of a normal physiologic process
-Ovarian neoplasms are derived from neoplastic growth of ovarian cell layers

Benign Ovarian Neoplasms
-Mucinous cystadenoma
-Serious cystadenoma
-Endometrioma (chocolate cyst)
-Fibroma
-Brenner tumor
-Thecoma
-Sertoli-Leydig cell tumors
-Dermoid cyst (teratoma): can contain hair, teeth, sebaceous glands, and thyroid cells producing TH
-Uterine leiomyoma

Malignant Ovarian Neoplasms
-Adenocarcinoma
-Granulosa cell tumor
-Dysgerminoma
-Clear cell carcinoma
-Endometrioid carcinoma

Risk Factors
-Nulliparity
-Fertility treatments
-FH of breast or ovarian cancer

Protective Factors
-Prolonged OCP use
-Pregnancy
-Tubal ligation or hysterectomy

Signs & Symptoms
-Thyrotoxicosis with dermoid tumor
-Torsioned ovary or cyst → signs of acute abdomen
-Malignancy symptoms are nonspecific like pelvic pain and bloating

Workup
-Transvaginal US: signs indicative of malignancy include large amounts of free fluid in the abdominal cavity, solid ovarian enlargement or mixed cystic and solid enlargement, thick-walled or complex ovarian cysts
-Serum CA-125: will also be elevated in infection, endometriosis, ovulation, and trauma
-Staging and grading of malignancies

Management
-Malignancy: local excision vs total hysterectomy and bilateral SO vs partial bowel resection depending on stage of cancer, usually followed by radiation ± chemo
-Benign neoplasms will persist unless excised, which is usually done to prevent ovarian torsion
-Simple cysts in a postmenopausal woman may be followed by serial US and CA-125s

Breast Cancer

-Usually arises from ducts or lobules
-Most commonly diagnosed female cancer
-Only 5-10% are due to genetic mutations

Risk Factors
-Obesity or inactivity
-Use of hormone therapy
-Nulliparity
-First birth after age 30
->1 alcoholic drink per day
-Not breastfeeding
-Increasing age
-White
-Hx of chest irradiation
-Hx of atypical hyperplasia on previous biopsy
-FH of breast cancer or inherited mutations

Prevention
-Women with high risk can consider chemoprevention with tamoxifen or raloxifene

Screening
-Mammography is USPSTF grade C for women 40-49, grade B for women 50-74 every 2 years
-Clinical breast exam
-Breast self-exam is USPSTF grade D
-Dedicated breast MRI for high risk populations

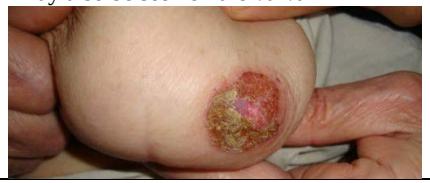
Signs & Symptoms
-Most commonly found on the upper outer quadrants
-Early: single, painless firm mass with ill-defined margins or possibly no palpable mass but an abnormality is detected on mammogram
-Later: skin or nipple retractions, axillary adenopathy, breast enlargement, erythema, peau d'orange, edema, pain, fixation of mass to chest wall
-Very late: ulceration, supraclavicular adenopathy, arm edema, mets to bone, liver, lung, brain, or adrenal glands

Differential
-Fibrocystic disease
-Fibroadenoma
-Intraductal papilloma
-Lipoma
-Abscess
-Fat necrosis
-Phyllodes tumor

Workup
-Biopsy of suspicious lesion
-Pathology and genomic marker assay

Management
-TNM classification
-Tumor marker profiling
-Surgical management: lumpectomy, sentinel node biopsy, or mastectomy
-Chemo is typically 3-6 months and is initiated for visceral mets, failed endocrine therapy, or ER-/PR- tumors
-Endocrine therapy with tamoxifen (premenopausal)or aromatase inhibitors (postmenopausal)
-Radiation therapy as an adjuvant

Prognosis
-Surgical complications: long thoracic nerve injury, lymphedema

Breast Cancer Type	Info	S/S	Management
Ductal Carcinoma in Situ	-Some consider this to be a pre-malignant lesion -Arises from ductal hyperplasia and fills ductal lumen -Very early malignancy without basement membrane penetration -Less than 30% recurrence rate following lumpectomy	-Typically asymptomatic and discovered on screening mammogram as calcifications -Usually not palpable on PE	-Lumpectomy followed by radiation is most common -Tamoxifen or aromatase inhibitor therapy for 5 years if receptor+ tumor
Invasive Ductal Carcinoma	-The most common breast cancer -Worst and most invasive	-Pt is typically postmenopausal -Mammogram detects spiculated margins -Firm, fibrous, rock-hard mass with sharp margins and small, glandular, duct-like cells -Likes to metastasize	-Chemo with Herceptin and Tykerb for HER2+ tumors
Lobular Carcinoma in Situ	-Some consider this to be a pre-malignant lesion -Contains signet ring cells -Will progress to invasive lobular carcinoma in 10%	-Usually not palpable and hard to detect on mammo -Often bilateral	
Invasive Lobular Carcinoma	-2nd most common breast cancer	-Orderly row of cells in stroma that are fluid and mobile -Often bilateral	-Assessment with US preferred over mammography
Medullary Carcinoma	-Fleshy, cellular, lymphocytic infiltrate -Good prognosis although it is a rare subtype of invasive ductal carcinoma	-Mammogram detects linear crystallization pattern	
Comedocarcinoma	-Subtype of DCIS -Ductal caseating necrosis		
Paget's Disease of the Breast	-Subtype of ductal carcinoma	-Presents as eczematous lesions on the nipple -May also be seen on the vulva 	

Endometrial Neoplasms

-Endometrial neoplasia involves proliferation of the endometrial glands that can progress to or coexist with endometrial carcinoma
-Endometrial carcinoma is the most common GYN cancer in the US and is usually adenocarcinoma

Risk Factors
-Age > 50
-Uopposed estrogen use
-PCOS
-DM
-Obesity
-Nulliparity
-Late menopause
-Tamoxifen use
-HNPCC

Signs & Symptoms
-Abnormal uterine bleeding
-Postmenopausal bleeding
-Abnormal pap cytology

Differential for Postmenopausal Bleeding
-Atrophy (59%)
-Endometrial polyps
-Endometrial cancer
-Endometrial hyperplasia
-Hormonal effects
-Cervical cancer

Workup
-Endometrial biopsy can be done in clinic and is 99.6% sensitive in premenopausal women and 91% in postmenopausal women
-Transvaginal US to assess endometrial stripe: thin stripe < 4-5 mm associated with low risk of cancer while stripe > 5 mm warrants biopsy

Management
-Benign pathology on biopsy watched, no action warranted unless bleeding persists
-Endometrial hyperplasia on pathology without atypia is treated with progesterone cream, ovulation induction, or IUD to induce massive menses and endometrial sloughing
-Atypical endometrial hyperplasia needs D&C or hysterectomy + BSO

Breast Abscess

-Can be lactational or nonlactational
-Etiologist is usually *Staph aureus*, with MRSA becoming increasingly more common

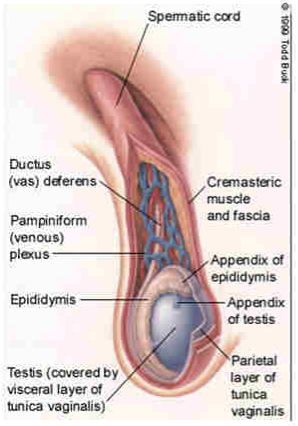
Risk Factors
-Obesity
-Smoking
-Black

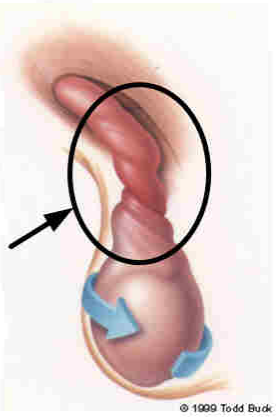
Workup
-Wound culture

Management
-Needle aspiration if overlying skin is intact
-I&D for compromised overlying skin or failed needle aspiration
-Antibiotics: dicloxacillin, cephalexin, or clindamycin
-Bactrim, clindamycin, or linezolid if suspecting MRSA
-Continue breastfeeding

Testicular Torsion

-Twisting of the spermatic cord within a testicle, cutting off blood supply
-A result of inadequate fixation to the tunica vaginalis
-Can be spontaneous or post trauma
-More common in neonates or postpubertal males





Signs & Symptoms
-Scrotal pain and swelling
-N/v
-Abdominal pain
-May wake child up in the middle of the night
-Tender epididymis, elevated testis, and scrotal discoloration
-Absent cremasteric reflex

Management
-Color Doppler US

Management
-Surgical emergency, must be treated within 4-6 hours with irreversible damage and possible infertility after 12 hours
-Manual detorsion if surgery unavailable

Prostate Cancer

-Usually adenocarcinoma
-The most commonly diagnosed male cancer and 2nd leading cause of male cancer deaths
-Risk factors: age, black, high fat diet, FH, obesity
-No association with smoking, sexual activity, prior infections, or BPH

Screening
-USPSTF grade I for men up to age 75 and grade D after 75
-If patient elects, DRE and PSA should be done every 2 years
-PSA will be elevated in cancer, inflammation, or BPH, and will naturally rise as men age

Signs & symptoms
-Asymptomatic early in disease
-Later disease: obstructive urinary symptoms, hematuria, hematospermia

Workup
-Prostate biopsy guided by transurethral US, with scoring by Gleason system
-MRI
-PET if suspected mets

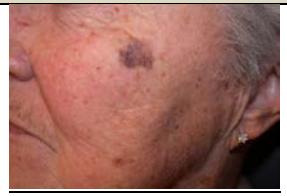
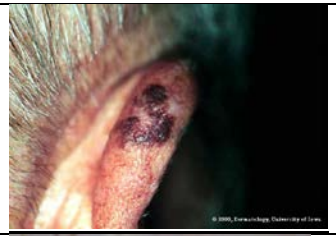
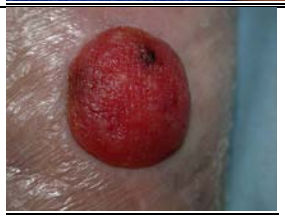
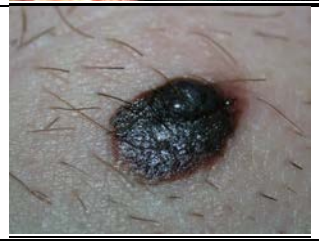
Management
-Treatment based on life expectancy, general health, tumor characteristics
-Treatment is controversial for localize disease
-Radical prostatectomy
-Radiation
-Hormone therapy for advanced or metastatic disease

DERMATOLOGY

Melanoma

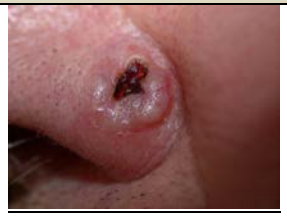
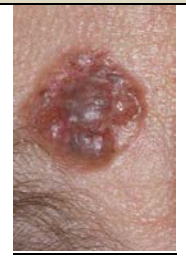
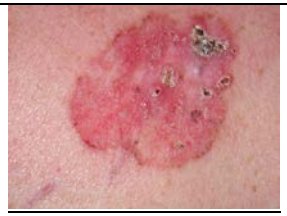
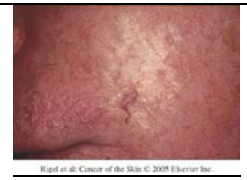
-Flat, raised, nodular, or ulcerated
-Variable color
-Consider in any new mole or a mole changing shape, size, or color

Workup
-Lymph node palpation
Punch or incisional biopsy

Type of Melanoma	Info	Picture	Type of Melanoma	Info	Picture
Lentigo maligna (melanoma in situ)	Melanoma restricted to epidermis.		**Acral lentiginous melanoma**	Primarily on hands, feet, nails. Most common type of melanoma in blacks and Asians. Common in males.	
Superficial spreading melanoma	Most common type of melanoma. Asymmetric, flat lesions > 6 mm. Vary in color. Lateral spread.		**Amelanotic melanoma**	Innocent-appearing pink to red colored papules that enlarge to plaques and nodules. Scary.	
Nodular melanoma	Rapid growth vertically from and through skin. Most common on extremities.				

Basal Cell Carcinoma

-Most common skin cancer, and most common human cancer
-Slow growing, locally destructive
-No mets
-Risk factors: sun, sunburns < age 14, arsenic ingestion, radiation
-More common in males
-Usually after age 40
-Several subtypes with different treatments

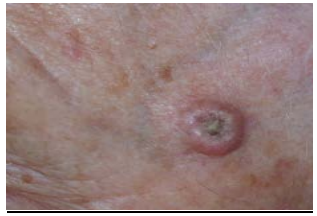
Type of BCC	Info	Investigation & Treatment	Picture	Type of BCC	Info	Investigation & Treatment	Picture
Nodular BCC	Most common BCC. Pearly white or pink dome shaped papule with overlying telangiectasias → ulceration, raised borders, bleeding, scaling.	ED&C, excision, Mohs.		**Pigmented BCC**	Resembles melanoma.	ED&C, excision, Mohs.	
Superficial BCC	Least aggressive BCC. Erythematous scaly plaques or papules +/- rolled borders. Can look like psoriasis, eczema, others.	ED&C, excision.		**Morpheaform BCC**	Least common variant. White to yellow patch with poorly-defined borders.	Mohs needed.	

Squamous Cell Carcinoma

-AKA Bowen's disease if SCC in situ
-Potentially invasive malignancy of keratinocytes in the skin or mucous membranes
-Most caused by UV radiation but other risks are chemicals, tobacco, infection, burns, HPV
-Erythroplasia of Queyrat is SCC of the penis

Presentation
-Flesh, pink, yellow, or red indurated papules plaques, or nodules with scale
-Can have ulcerations and erosions

Workup & Management
-Palpate regional lymph nodes for mets.
-ED&C, excision, Mohs.
-Bowen's: 5-FU, cryo, ED&C, excision, Mohs.



Pilonidal Disease

-An acquired condition likely related to mechanical forces on the skin overlying the natal pilonidal cleft → cavity formation containing hair, debris, and granulation tissue

Signs & Symptoms
-May have asymptomatic cyst
-Tenderness, erythema, and abscess formation just above the gluteal cleft

Management
-Acute abscess needs I&D with debridement of all visible hair
-Refer for surgical excision of recurrent pilonidal disease
-Primary closure associated with faster healing but delayed closure associated with lower likelihood of recurrence
-Antibiotics only for cellulitis

Hidradenitis Suppurativa

-Chronic inflammatory skin disorder characterized by pustules, inflammatory nodules, and sinus tract development, usually in intertriginous areas

Prevention
-Avoiding skin trauma
-Careful skin hygiene
-Smoking cessation
-Reducing carb intake
-Weight reduction

Signs & Symptoms
-Affected areas may be in the axillae, genitofemoral region, gluteal folds, or perianal areas
-Small, painful subcutaneous nodules can be palpated
-Pruritus
-Erythema
-Burning pain
-Local hyperhidrosis
-Sinus tract formation
-Hyperpigmentation, scars, and pitting of the skin

Management
-Hot packs for mild cases
-Topical or systemic antibiotics: clindamycin or doxycycline
-Retinoids
-Accutane
-Surgical removal, I&D, or skin grafting for severe cases
-I&D will not alter course of disease and should be reserved for pt comfort in times of tight skin abscess formation

HEMATOLOGY

Disseminated Intravascular Coagulation

-Can be acute or chronic

Etiologies
-Usually occurs when shock causes widespread activation of the clotting cascade
-Sepsis
-Trauma and tissue destruction
-Malignancy
-Obstetric complications: placental abruption, HELLP syndrome, hemorrhage, septic abortion

Signs & Symptoms
-Bleeding diathesis: petechiae, ecchymosis, oozing from wounds and IVs
-Thromboembolism
-Renal dysfunction: AKI
-Hepatic dysfunction: jaundice
-Respiratory dysfunction: hemoptysis, dyspnea
-Shock
-CNS involvement: coma, delirium, TIAs

Workup
-Peripheral smear shows microangiopathic hemolytic anemia
-Low platelets and clotting factors (may be near normal in chronic DIC)

Management
-Treat underlying disease
-Hemodynamic support
-Most coagulopathies are short-lived but some pts with severe bleeding may need platelets or FFP transfusions

INFECTIOUS DISEASE

Postoperative Fever

Etiologies
-Not always infectious!
-Wind: atelectasis, pneumonia
-Wound infection: usually occur several days to 1 week after operation
-Water: UTI
-Walking: DVT or thrombophlebitis
-Wonder drugs: medication-induced fever (heparin or abx)
-Women: postpartum fever, endometritis
-Blood transfusion

Prevention
-Avoid atelectasis: early ambulation, incentive spirometry
-Avoid pneumonia: use humidified O2
-DVT prophylaxis
-Judicious use of Foley catheters with d/c ASAP
-Clear instructions for home care of wound sites

Workup
-Fever in patient < 2 days out from surgery who is otherwise doing well is usually self-limiting and does not require workup
-CBC
-CXR: may lag behind PE findings
-Consider LE US for DVT
-Blood cultures?

Management
-Change out infected or thrombosed lines
-Wound infection: open infected area, start antibiotics
-D/c unnecessary meds, NGT, catheters
-Treat fever with acetaminophen
-Broad spectrum antibiotics only for hemodynamically unstable pts while source is being identified

Surgical Site Infection

-SSI is defined as infection related to the operative procedure occurring at or near the surgical incision within 30 days of an operative procedure or within 1 year of an implant
-Occur in 2-5% of patients undergoing surgery
-Nonteaching hospitals have lower rates than teaching hospitals
-Most common source is direct inoculation of pt's endogenous flora at the time of surgery
-Incidence of resistant pathogens cultured from SSIs is increasing: MRSA, MRSE, VRE
-Incidence of fungi cultured from SSIs is increasing: Candida albicans

Risk Factors
-Obesity
-Smoking
-DM
-Systemic corticosteroids
-Immunosuppression
-Malnutrition
-Preoperative nasal carriage with Staph aureus
-Presence of remote focus of infection
-Long duration of preoperative hospitalization
-Preoperative severity of pt illness
→ Can predict pt risk of SSI with National Healthcare Safety Network risk index

Prevention
-Preop showering with antimicrobial soaps
-Preop prepping of operative site with antiseptics (chlorhexidine superior to iodine)
-Washing and gloving of surgeon's hands (alcohol rubs may be as effective as traditional soap scrubbing)
-Use of sterile drapes
-Use of gowns and masks by OR personnel
-Good surgical technique: gentle traction, effective hemostasis, removal of devitalized tissue, obliteration of dead space, irrigation with saline, use of fine nonabsorbable monofilament suture, judicious use of closed suction drains, wound closure without tension
-Antibiotic prophylaxis: should be administered within 60 min of first incision; may need to be repeated more than once depending on length of surgery
-Hair removal: may increase risk of surgical site infection, must use clippers or depilatories if removing hair vs razor
-Tight glucose control in diabetic pts
-Perioperative warming (bear huggers) to prevent hypothermia, warmed IVF, hats and booties
-Minimally invasive and laparoscopic procedures associated with ↓ risk of SSI

Components of Optimal Wound Healing
-Well-vascularized wound bed
-Wound free of devitalized tissue
-Wound clear of infection
-Moist wound

Management of SSI
-Opening, exploration, draining, irrigation of wound
-Sharp surgical debridement of devitalized tissue
-Wound can be closed or allowed to heal by secondary intention once granulation tissue is apparent

Surgical Antimicrobial Prophylaxis

Nature of Operation	Common Pathogens	Recommended Antimicrobials	Alternative Antimicrobials	Nature of Operation	Common Pathogens	Recommended Antimicrobials	Alternative Antimicrobials
Cardiac	-Staph aureus -Staph epidermidis	-Cefazolin	-Cefuroxime -Vancomycin	Surgical abortion Hysterosalpingogram Chromotubation		-Doxycycline	
Esophageal or gastroduodenal	-Enteric gram neg rods -Gram pos cocci	-Cefazolin only for high risk individuals		Incisions through oral or pharyngeal mucosa	-Anaerobes -Enteric gram neg rods -Staph aureus	-(Clindamycin or cefazolin) + metronidazole	-Zosyn
Biliary tract	-Enteric gram neg rods -Enterococci -Clostridia	-Cefazolin only for high risk individuals		Neurosurgery Orthopedic surgery	-Staph aureus -Staph epidermidis	-Cefazolin	-Vancomycin
Colorectal	-Enteric gram neg rods -Anaerobes -Enterococci	-Preop neomycin + erythromycin colon prep -Cefoxitin	-Preop metronidazole colon prep -Cefotetan or (cefazolin + metronidazole) or Zosyn	Ophthalmic surgery	-Staph epidermidis -Staph aureus -Streptococci -Enteric gram neg rods -Pseudomonas	-Gentamicin drops	-Tobramycin drops -Ciprofloxacin, or gatifloxacin, or levofloxacin, or moxifloxacin, or ofloxacin (all drops) -Neomycin-gramicidin-polymyxin B drops -Cefazolin subconjunctival injection

Appendectomy (non-perforated)	-Enteric gram neg rods -Anaerobes -Enterococci	-Cefoxitin + metronidazole	-Metronidazole + (cefotetan or cefazolin)	Noncardiac thoracic surgery	-Staph aureus -Staph epidermidis -Streptococci -Enteric gram neg rods	-Cefazolin	-Zosyn -Vancomycin
Cystoscopy	-Enteric gram neg rods -Enterococci	-Ciprofloxacin only for high risk individuals (oral)	-Septra	Vascular: arterial surgery involving a prosthesis, the abdominal aorta, or a groin incision	-Staph aureus -Staph epidermidis -Enteric gram neg rods	-Cefazolin	-Vancomycin
Open or laparoscopic GU surgery	-Enteric gram neg rods -Enterococci	-Cefazolin (IV)		Vascular: LE amputation for ischemia	-Staph aureus -Staph epidermidis -Enteric gram neg rods -Clostridia	-Cefazolin	-Vancomycin
Hysterectomy Urogyn procedures C-section		-Cefazolin	-Cefoxitin -Cefotetan -Zosyn -(Clindamycin or vancomycin) + (gentamicin or aztreonam or FQ)	Laparoscopic GYN procedure except hysterectomy Hysteroscopy IUD insertion Endometrial biopsy		-None	