ACUTE WOUND HEALING

General Considerations: Wounds- injury (trauma) or intentional (surgery)

- Always consider Tetanus vaccination status
  - Give tetanus toxoid always unless already given 3 doses in lifetime or at least one given in the last 5 years
  - Give tetanus immune globulin only if in a tetanus-prone wound and < 3 doses given in lifetime (or unknown)
- Only important way to decrease bacterial count in a wound is debridement, in addition to dressing changes q4hr.
- Apply no tension on skin (causes necrosis)
- Close along Langer’s lines if possible
- Absorbable suture (gut, vicryl, PDS) is more inflammatory than non-absorbable (silk, ethibond, prolene, nylon, steel)
- Tensile strength: very little until 3 weeks, level off at around 7 weeks. At 4 weeks is 85% of its maximum strength.

Steps of wound healing:

1. Substrate/Inflammatory/Lag/Exudate phase - 0 to 7 days in primary intention, indefinitely for secondary/tertiary until epithelialization occurs
   - Hemostasis: Activated platelets, coagulation factors --> hemostatic plug. - 1 to 2 hours
     - Impaired in chronic liver disease and uremia
   - Inflammation: PDGF, TGF-B and fibronectin are chemoattractive. - 0 to 2 days
     - PMNs, macrophages, complement C5A, kallikrein
       - PMNs are the most prominent cell type during this period
       - Macrophages are the most important cell type, they release metalloproteinases for remodeling/debridement of necrotic tissue as well as growth factors
     - Impaired by steroids, immunosuppressants, congenital or acquired immunodeficiency
     - MOST IMPORTANT stage to prevent wound infection: macrophages can handle small #s of bacteria. If there are large #s, or neutropenia, clinical wound infection occurs.
     - Systemic Abx don't work during this phase b/c they don't penetrate granulation tissue. Use topical Abx.

2. Proliferative phase: Epithelialization, collagen synthesis, angiogenesis (VEGF-mediated) - 4 days to 3 weeks
   - Epithelialization must occur as the first step for the rest of the phase to continue.
   - Net production of collagen during this phase
     - Alpha helix, every 3rd AA is glycine, has unique AAS OH-proline, OH-lysine
   - Primary cell type: fibroblast
   - Impaired by Vitamin C def, iron def, or Dz of collagen synthesis (osteogenesis imperfecta, Ehlers-Danlos, etc)

3. Maturation: Remodeling, contraction - 3 weeks to 1 year
   - Cross linking of collagen
   - “flattening” of the scar, takes 9 months in an adult
   - No net production of collagen during this period
Factors affecting wound healing

- Aging
- Nutritional status
  - Need anabolic positive protein balance for optimal healing
  - Vitamin A deficiency: delayed wound healing, epithelial keratinization, anemia
  - Vitamin C deficiency: delayed wound healing, wound dehiscence. Is a cofactor in matrix metalloproteinases which cleave collagen fibrils, helping it mature to full tensile strength.
  - Vitamin K deficiency: essential for carboxylation of glutamate, synthesis of X, IX, VII, II coagulation factors
- Chronic disease: ex. Diabetes
- Oxygen delivery: Peripheral vascular disease, smoking (↓ oxygen carrying capacity)
- Chronic edema: CHF
- Medications: ex. steroids

Wound Closure

- **Primary Intention**: edges of wound are approximated by either sutures, skin grafts, etc
  - Immediate coverage with epithelial elements. Epithelialization occurs in 12-24 hours
  - Use absorbable suture for deep closure and nonabsorbable for skin closure (less irritating)
  - Contraindicated with contaminated wound (human bite, farm injury), severe crush injury
- **Secondary Intention**: spontaneous wound closure. Granulation at base first, margins contract, epithelialization last
  - Contracts at 1mm/day For wounds to large to close.
- **Tertiary intention**: Granulation occurs, then wound edges approximated a few days later.
  - *Should only be performed in wounds with bacterial counts of less than 10^5, therefore NON-infected wounds.*

Wound contamination status

<table>
<thead>
<tr>
<th>Type</th>
<th>Characteristics</th>
<th>Chance of infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clean</td>
<td>&lt;12 hours old, minimal bacteria. No GI, GU, respiratory tract.</td>
<td>1-4%</td>
</tr>
<tr>
<td>Clean-Contaminated</td>
<td>Penetration into GI, GU, respiratory, but prepped</td>
<td>5-15%</td>
</tr>
<tr>
<td>Contaminated</td>
<td>Gross contamination, &gt; 10^5 organizz. Fresh traumatic wound.</td>
<td>16-25%</td>
</tr>
<tr>
<td>Infected</td>
<td>Prior infection in region of wound. Ex. perforated viscus.</td>
<td>50%</td>
</tr>
</tbody>
</table>

**Types of wounds**: Avulsion, Crush: need debridement    Abrasion: suture    Puncture: no closure necessary

**Chronic Wounds**

- Diabetic foot ulcer
  - Foot architecture changes due to loss of intrinsic muscles of the foot (Charcot foot)
  - Loss of sensation and protective reflexes
- Pressure ulcer
- Arterial insufficiency ulcer
- Venous stasis
  - Usually in anteromedial leg NOT foot
  - Tx: compression stockings
- Open wounds (stalled inflammatory phase)

Wound Care

- Debridement: surgical, hydrotherapy (to separate eschar), enzymatic (Elase, Travase, Biozyme-C)
- Negative pressure devices
- Recombinant growth factors: still in clinical trials
- Hyperbaric oxygen: controversial in care of chronic wounds
- Topical agents: Hydrogen peroxide (not bactericidal, but cleanses w/bubbling), Neosporin, sulfadiazene, nitrofurazon
- Dressings
  - Conventional gauze: helps in gentle debridement. Other dressings better once granulation bed present
  - Semipermeable: minimize moisture loss
  - Impermeable hydrocolloids: Anaerobic, may improve reepithelialization rate
10% of the population have gallstones. May be asymptomatic. **Risk factors:** Female, fat, fertile, forty, flatulent.

10% of people with cholecystitis are acalculus: ICU patients, due to bile stasis. **Tx:** Perc. decompression and interval cholecystectomy.

**Indications for cholecystectomy:** Symptomatic, calcified gallbladder, stones > 3cm (with or without symptoms)

<table>
<thead>
<tr>
<th>Cholelithiasis</th>
<th>Acute cholecystitis</th>
<th>Acute ascending cholangitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sx</strong></td>
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</tr>
<tr>
<td>- Biliary colic: <strong>colicky RUQ pain,</strong> radiates to shoulder (Boas sign), worse w/fatty foods</td>
<td>- Cholelithiasis, but worse</td>
<td>- Cholecystitis, but worse</td>
</tr>
<tr>
<td>- Physical exam often unremarkable</td>
<td>- Nausea/vomiting</td>
<td>- High fever (104 F), chills</td>
</tr>
<tr>
<td>- Mild leukocytosis (12-15k)</td>
<td>- <strong>Constant</strong> pain, not colicky</td>
<td>- Severe leukocytosis (22,000)</td>
</tr>
<tr>
<td>- Mild jaundice (2-3mg/dl)</td>
<td>- Mild fever (101 F)</td>
<td>- LFTs abnormal: ↑ bili, ↑ alk phos</td>
</tr>
<tr>
<td>o In 20%. From inflammatory cholestasis, not obstruction</td>
<td>- Mild leukocytosis (12,000)</td>
<td>- Charcot’s triad: RUQ pain, fever, jaundice (seen in 50-70%)</td>
</tr>
<tr>
<td></td>
<td>- Hypoactive bowel sounds</td>
<td>- Reynold’s pentad: + sepsis &amp; MS Δ</td>
</tr>
<tr>
<td></td>
<td>o <strong>Except alk phos</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Muscle guarding</td>
<td>• 60% due to choledocholithiasis</td>
</tr>
<tr>
<td></td>
<td>• Murphy’s sign pathognomonic</td>
<td></td>
</tr>
</tbody>
</table>

| Dx | 1. Ultrasound (95% sensitive) | 1. Ultrasound |
| 1. Ultrasound (Pericholecystic fluid, thickened gallbladder) | 2. HIDA scan (4 hours) | 3. CT if suspect complication (abscess, perforation, pancreatitis) |

| Tx | 1. NPO, IVF, (NGT if N/V) | 1. IV antibiotics |
| 2. IV antibiotics (2nd gen cephalosporins) | 2. Decompress biliary tree (percutaneous) |
| 3. IV analgesia (**Meperidine, not morphine**—contracts sphincter of Oddi) | 3. ERCP after acute phase: treats underlying cause - obstruction |

| Cx. | 33% cholecystitis in 2 years | |
| Hepatic a ligation: ischemia, stricture | 1. Gangrenous cholecystitis | 1. Sepsis |
| | Perforation (very bad) | 2. Hepatic abscess (very bad) |
| | Emphysematous cholecystitis | |
| | Gallstone ileus | |

**Other differentials**
- RUQ pain and jaundice, but afebrile: Suspect **Choledocolithiasis**
  - **Diagnosis:** Some combination of: ERCP, cholangiogram (mandatory if pancreatitis), lap. cholecystectomy
  - **Treatment**
    - Stones < 3mm: observe
    - Biliary stricture (if previous cholecystectomy): Choledochojunaljunostomy
- Ultrasound: gallbladder distention with **internal echoes** + gallstones: **Empyema of the gallbladder**
  - **Treatment:** IV antibiotics, percutaneous cholecystostomy, cholecystectomy
- Ultrasound: No gallbladder stones, but dilated ducts with air: **Suppurative cholangitis**
  - **Treatment:** IV antibiotics, ERCP for decompression
- **Other therapeutic Measures:**
  - Deoxycholic acids—help dissolve some bile salts
  - Lithotripsy
  - Antibiotics if gangrenous-use 3rd gen cephalosporins
  - Pain control: meperidine (morphine contracts sphincter of Oddi)
NUTRITION

Digestion:
- Fat: 100g/day
- Protein: 100g/day
- Carbohydrates: 400g/day

Reserves:
- 160,000 calories stored in liver as glycogen, elsewhere as muscle, fat.

Nitrogen balance: \( (g) = \frac{(Protein \text{ intake} \ (g)}{6.25}) - (\text{Urine Nitrogen +4}) \)

Caloric requirement: (Basal energy expenditure [BEE]):
- Males = 25 kcal/kg/day
- Females = 22 kcal/kg/day

Protein requirement:
- 24 hr urine nitrogen is the most accurate reflection of individual protein requirements
  - 0.8-1.0 g/kg. More in burns, less in refeeding after starvation and hepatic failure
  - Nondepleted: 1g/kg/day, calories 120% of BEE
  - Depleted: 2-2.5g/kg/day, calories 150-200% of BEE
  - Need 1 mEq K+/kg/day, 1.5 mEq Cl- /kg/day, 2 mEq Na+/kg/day

Starvation:
- 0-48 hours: Protein and fat are the only major sources of calories. Nitrogen loss: 15g/day
  - Primary sources: skeletal muscle to feed the brain, RBCs, and WBCs
  - Hepatic glycogen depleted in 36 hours. Lactate/pyruvate exchanged through Cori Cycle from glycolysis.
  - “Protein sparing effect” - Give surgical patient carbs, spares AAs released from muscle (insulin-mediated)
- 48+ hours: nitrogen loss decreases to 4g/day
  - Occurs due to switch of brain from glucose to ketoacids (produced from fat)
    - Switch mediated by insulin and alanine

Nutritional assessment:
- Preoperative
  - Anthropometry (triceps skin fold thickness, arm circumference)
  - Lab tests: Albumin less than 3.2. Prealbumin (thyroid binding globulin) is more sensitive b/c lower half life.
  - Energy skin antigens
  - Patient unable to eat for 10 days or more
  - Severe trauma, burn patient
  - Major GI surgery
- Postoperative: studies are inconclusive whether this helps or not!

Indications for nutritional support
- Enteral nutrition: Used when gut is working, but oral may be difficult.... AMS, ventilator, upper GI disorder
  - Not sufficient in increased metabolic states (anorexia, sepsis, trauma, burns)
  - Decreased risk of infection compared to TPN
  - Glutamine (enterocytes), Arginine, Leucine, Isoleucine, Valine (colonocytes) important
  - Contraindicated when patient cannot protect airway
- Total Parenteral Nutrition (TPN)
  - Indications
    - When patients can’t tolerate adequate GI intake within 5-7 days
    - Newborns with catastrophic GI abnormalities or FTT
    - Short bowel syndrome and/or vascular compromise of bowel
    - GI obstruction, ileus
    - Acute renal failure since catabolism would overload kidneys
  - Complications
    - Infection
    - Hyperosmolar, nonketotic hyperglycemia.
      - Treatment: replace volume and electrolytes, add insulin drip
    - Hepatic steatosis
    - CO2 retention aggravating respiratory insufficiency
  - Contraindications: Cardiovascular instability, Less than 8 cm of normal bowel

Signs of nutritional deficiency
- Fatty acids: Dry, scaly dermatitis
- Trace metals: Eczematoid rash in intertriginous areas, changes in taste, microcytic anemia, glucose intolerance
- Vitamin A: hair loss, night blindness
- Vitamin C: cheilosis
- Vitamin K: Ecchymosis and bleeding
- Vitamin B1 (thiamin): Ataxia, parasthesia, muscle weakness, beriberi
## ACUTE ABDOMEN

### Right Upper Quadrant
- Biliary colic/Acute cholecystitis
- Peptic ulcer disease (PUD)
- Gastritis
- Cholangitis
- Hepatitis
- Pneumonia
- Pleurisy

### Epigastrium (u/s, empiric PPI... EGD)
- GERD
- Pancreatitis (acute/chronic)
- Peptic ulcer disease (PUD)
- Angina, myocardial infarction
- Gastroenteritis
- Perforated vissus
- Esophagitis/gastritis
- Abdominal aortic aneurysm (AAA)
- Early acute appendicitis
- Abdominal splenic rupture (Kehr’s sign)
- Gastritis
- Peptic ulcer disease (PUD)

### Indications for urgent operation:
- Involuntary guarding, rigidity
- Increasing severity/distention
- High fever, hypotension
- Equivocal findings w/sepsis, bleeding
- Pneumoperitoneum, distension, space-occupying lesion, mesenteric occlus.

### Left Upper Quadrant
- Acute pancreatitis
- Perforated viscus
- Myocardial infarction
- Splenic rupture
- GERD
- Gastritis
- Peptic ulcer disease (PUD)

### Right Lower Quadrant
- Acute appendicitis
- Inflammatory bowel disease
- Meckel’s diverticulum
- Acute cholecystitis
- Pyelonephritis/Nephrolithiasis
- Diverticulitis
- Ovarian torsion, cyst
- Ruptured ectopic pregnancy, PID
- Intussusception
- Colon Cancer

### Workup:
- CBC, electrolytes, LFTs, amylase & lipase, UA, urine microscopic exam, urine culture
- All female patients need a pelvic exam and a pregnancy test
- CXR and AXR to look for free air - highly suggestive of a perforated viscus. Most common cause: PUD
- Abdominal CT scan and/or RUQ ultrasound

### Expectant management: NPO, NG tube if N/V, IVF, foley. Exploratory laparotomy: unstable patients when surgery can help

## APPENDICITIS

### Pathophysiology:
- Hyperplasia of lymphoid tissue (60%), fecalith (35%), foreign body, parasite, carcinoid tumor (rare - 5%)
  - Obstruction leads to bacterial overgrowth, inflammation, distention can compromise blood supply (appendicular artery is a branch of the ileocolic artery which is a branch of the SMA).
- Complications: necrosis, infarct, perforation, abscess, peritonitis

### Symptoms:
- Abdominal pain, mid-epigastrium, moves to umbilicus, then to RLQ, anorexia is always present. Nausea, vomiting

### Signs:
- Tenderness in RLQ (most intense at McBurney’s point), rebound tenderness, guarding, diminished bowel sounds, low grade fever (if high, think perforation)
- Rovsing’s sign (push on LLQ, referred pain to RLQ), Psoas sign, Obturator sign, if palpate a mass on rectal, think appendiceal abscess. Preggo women may have RUQ pain due to the location of the appendix in the gravid uterus.
- Atypical appendicitis: N/V without peritoneal signs, may mimic obstruction

### Diagnosis:
- Peak incidence: teens to mid-20s, and elderly
- Acute appendicitis is a clinical diagnosis. Leukocytosis (mild), CT scan (sensitive 98-100%), ultrasound (90%)
- Rectal exam (retrocecal appendix), Pelvic exam (r/o gynecological cause)

### Treatment:
- Appendectomy (laparoscopic) 20% false positive. Risk is ok in pregnancy (10-15% increased risk of premature labor)
- With cecum involvement: Right hemi-colectomy
- Carcinoid tumor of the appendix
  - <2cm and not involving cecum: appendectomy alone sufficient (probably +baseline 5-HIAA, octeotride scan)
  - >2cm or involving cecum: Right hemi-colectomy, baseline 5-HIAA, octeotride scan for mets.

### Complications:
- PERFORATION (20% of all cases): High fever, tachycardia, marked leukocytosis, peritoneal signs, toxic appearance.

### Differential diagnosis (if negative on laparoscopic appendectomy, search for other cause of symptoms):
- Gastroenteritis, Mesenteric adenitis (Yersinia enterolytica), Meckel’s, Intussusception, typhoid fever, primary peritonitis, terminal enteritis, diverticulitis, carcinoid tumor (baseline 5-HIAA, CT, octeotride scan)
- Gynecological: Ectopic pregnancy, PID, Ovarian torsion/tumor/cyst, UTI/pyelo, ureteral stone

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- Gynecological: Ectopic pregnancy, PID, Ovarian torsion/tumor/cyst, UTI/pyelo, ureteral stone
DIVERTICULITIS - “LLQ appendicitis”, 95% of time: sigmoid colon

Pathophysiology:
- Limited infection of one or more diverticula, including extension into adjacent tissue.
- Fecalith obstruction --> microperforation.

Symptoms: LLQ pain (subacute onset), alteration in bowel habits (constipation OR diarrhea)
  - Lower GI bleeding rare (common in diverticulosis, not -itis)
  - Fever, LLQ pain, leukocytosis (“LLQ appendicitis”)

Diagnosis/Workup
- AXR, CT with oral and IV contrast.
- Barium enema and colonoscopy are CONTRAINDICATED during acute phase (2-3 weeks) due to perforation risk, unless signs of obstructive or fistula symptoms.

Treatment: Directed at specific complication.
- Acute: medical treatment 85% of the time.
  - Admit, IVF, IV non-opioid analgesics, NPO, IV abx(aminoglycoside, coverage for B. fragilis) x 5-7 days.
- Surgery: Resection

Indications for surgery: Perforation, obstruction, intractability (recurrence), bleeding, fistula
- All patients with diverticulitis must undergo a full colonoscopy 4-6 weeks after the attack to r/o malignancy
- Procedure: sigmoid colectomy, temporary colostomy

Complications
- Fistula: skin, vagina, bladder (most common complication - 4%. Pneumaturia, recurrent UTI)
- Perforation, abscess, fistula, obstruction

DIVERTICOLOSIS
- 70% of elderly patients may have asymptomatic diverticulosis. Increases from 50s-80s.
- 80% an asymptomatic finding on barium enema.
- 95% occur in sigmoid colon. Usually occurs where branches of marginal artery penetrate the wall of the colon.
- Etiology: higher than normal intraluminal pressures as would occur with low fiber diet.
- Symptoms: 40% of lower GI bleeds. Most common cause of lower GI bleeds in < 60 years old.
  - 25% of bleeds are massive (requires 4+ units of blood in 24 hours to maintain hemodynamics)
  - Usually asymptomatic. Recurrent abdominal pain in LLQ, change in bowel habits. Bleeding.
  - Fever and leukocytosis MUST be absent.
- Treatment: Diet of increased fiber, psyllium.
GENERAL:

PERIPHERAL VASCULAR DISEASE

Pathophysiology:

- Patients usually have underlying CAD (with CHF, Hx of MI, etc) or other chronic problems (IDDM, lung disease, smoking, hyperlipidemia, HTN, homocystinemia)
- Change in arterial pressure is usually small until stenosis >50% (Poiseuille's law)
- Most common cause of death is IC patients is cerebral or cardiac events

Types

- Femoropopliteal: Below inguinal ligament: --> Hunter’s (adductor) canal
  - Most common site overall. Typically occurs at the adductor hiatus
- Tibial occlusive disease: distal to popliteal artery trifercation
  - Most common in: diabetes, ESRD, advanced age
- Aortoiliac: Leriche Syndrome: Impotence, no femoral pulse, claudication, buttock muscle wasting
  - Generally a progressive disease that will fail medical management. PTA works best here.
- Superficial Femoral: Calf claudication

Symptoms

- Intermittent claudication: cramping leg pain with exercise that is reproducible, improved by rest (vs. neurogenic)
- Rest pain: distal metatarsals, prominent at night, awakens from sleep

Signs

- Diminished/absent pulses, muscular atrophy, decreased hair growth, thick toenails, ↓ skin temp
- Ischemic ulceration (usually on toes): local necrosis, tissue infarct/gangrene (advanced)
- Buerger’s sign: rubor when dependent, pale when elevated (advanced disease)

Diagnosis

- Doppler: Triphasic waveform (healthy). Systole, backflow as elastic arteries recoil, forward in diastole
- Ankle to brachial (ABI) index: normal 1.0, claudication <0.7, rest pain <0.4
- Pulse volume recording (assess segmental limb perfusion)
- Arteriography (contrast, X-ray): gold standard. Not performed to diagnose, but instead as preoperative study.

Treatment

- Medical management
  - Stop smoking - 50% improve!!! Graduated exercise program, foot care, atherosclerotic risk factor reduction, avoid extremes of temperature (esp. Toledo)
  - Medicine: aspirin, Pentoxifylline (aka Trental, lowers blood viscosity)
- Surgery: bypass graft (aortobifemoral, axillofemoral). Angioplasty

Surgery indications: Failed medical Tx, pain interferes with lifestyle/occupation, impending/actual tissue loss, rest pain.

Prognosis: 70% remain stable or improve, 20% progress and require revascularization, 10% require amputation

- The more distal and more severe the disease, the more likely the graft is to fail.
ABDOMINAL WALL HERNIAS

Pathophysiology:
- Occur in areas where striated muscle does not protect
- The smaller it is, more likely to strangulate
- Inguinal canal boundaries:
  - Anterior: External oblique aponeurosis
  - Posterior: Transverse abdominal muscle aponeurosis and transversalis fascia

Symptoms:
- Pain (increases with hard work/straining/worse at end of day, relieved at night/lying down), non-specific discomfort
- Dull, dragging pain referred to testes. “Bulge in groin”
- Asymptomatic

Signs: Bulge palpated or visible upon standing and coughing.

Diagnosis: Clinical diagnosis. Radiology only used in special circumstances (obesity, clinical exam not reliable)

Types:
- **Direct inguinal**: enters canal via weak posterior wall, does not pass thru internal ring
  - Lies posterior to spermatic cord, occurs almost exclusively in males, older, smokers
  - Never enters the scrotum, can damage the ilioinguinal nerve
  - Protrude medial to the inferior epigastric vessels, not assoc. with processus vaginalis.
  - Herniate thru Hesselbach’s Triangle:
    - Inferior border: Inguinal ligament
    - Medial border: Rectus abdominus
    - Lateral border: Inferior epigastric vessels (lateral umbilical fold)
- **Indirect Inguinal**: Need patent process vaginalis (congenital lesion) AND stress (20% of cadavers have some patency)
  - Most common in both sexes
  - Communicating hydrocele is same process but no bowel yet
  - Noncommunicating: diagnosed by unchanged location by position or pressure
  - New onset without identifiable stress should prompt workup: COPD, micturation straining, defacation straining
  - More common on right side in first decade of life (right testis descends later)
  - Children often bilateral
- **Femoral**: Very susceptible to incarceration b/c of inguinal ligament, lacunar ligament, Cooper’s (superior public lig)

Treatment: All hernias need surgical repair. (Even though evidence says only 0.1% strangulate.)
- Attempt to manually reduce when patient is in Trendelenburg: 60-70% successful
- Surgical: tension-free repair of defect using fascia, aponeurosis, or mesh
- Herniotomy (young, healthy, muscles): sac is freed, ligated, reduced
- Herniorrhaphy and plasty: herniotomy with repair of posterior wall and inguinal ring.
- Mesh: 50-75% decrease in rate of recurrence
- Non-surgical: (moribund patients) “hernia truss” to keep hernia reduced, limited in use
- Children: high ligation only is required

Differential diagnosis:
- Abdominal wall mass
- Desmoids (locally aggressive fibrous proliferation)
- Neoplasm, adenopathy
- Rectus sheath hematoma
- Hydrocele (transilluminate, hernias are not translucent but hydroceles are)

Other Hernias:
- **Umbilical Hernia**: Children: ignore, adult <1.5cm: suture, adult >1.5cm: mesh
- **Incisional (Ventral) Hernia**: Risk factors: deep wound infection (most common), obesity, steroid use
- Spigelian (through semilunar line @ junction of semicircular line of Douglas),
- Richter (wall only, no bowel obstruction sx)
- Lumbar: Grynfelt (superior lumbar), Petit (inferior lumbar)
- Hernia sac includes viscera: Littre (includes Meckel’s diverticulum in hernia), Armand (includes appendix)
- Obturator, Hesselback (like femoral but lateral to femoral vessels), epigastric, diastasis recti
GASTROINTESTINAL BLEEDING: 75% are upper GI. Divider of upper/lower is Ligament of Treitz

Presentation
- Hematemesis: upper GI endoscopy
- Hematochezia: 1) r/o hemorrhoids, 2) colonoscopy, 3) upper GI endoscopy
- Melena: 1) upper GI endoscopy 2) colonoscopy (DDx: bismuth, iron, spinach, charcoal, licorice)
- Fecal occult blood test (FOBT): colonoscopy

Determination of source: NG tube placement with lavage
- Bile + blood indicates upper GI source
- Bile, no blood indicates NOT upper GI source
- Clear fluid, w/no bile is equivocal

Etiologies
Upper GI bleed (75% of GI bleeds are upper)
- PUD (70%)- duodenal (25%), ulcer (20%), gastritis (25%). 80% stop spontaneously, ask about NSAID & anticoagulant use
- Reflux, varices, Mallory-Weiss, hemobilia, aortoenteric fistula (Hx of AAA repair)
- Dieulafoy’s vascular malformation: submucosal dilated arterial lesions
- Neoplasm: bleeding is not rapid, usually not emergent.

Lower GI bleed (25%)
- Diverticulosis (40%) - most common in < 60 years old
  - Asymptomatic or vague LLQ pain (20%)
  - Diagnosis: barium enema
  - Treatment: High fiber diet, psyllium
  - Complications: Painless bleed, diverticulitis
- Diverticulitis (rarely presents as bleed)
  - Fever, LLQ pain, leukocytosis (“LLQ appendicitis”)
  - Diagnosis: CT scan of abdomen and pelvis. NOT enema or colonoscopy during acute phase. Perform in 4-6 weeks.
  - Treatment: IV antibiotics, NPO. Recurrent episode: resection
  - Complications: Bowel obstruction, abscess, fistula
- Angiodysplasia (40%) - most common in > 60 years old
- Colorectal cancer – usually left sided cancers have gross blood, right sided have occult blood
- Polyp
  - Premalignant: FAP, villous adenoma, adenomatous polyp
  - Benign: Juvenile, Peutz-Jeghers, inflammatory, hyperplastic
- Acute mesenteric ischemia
  - Symptoms: acute, severe onset of pain that is disproportionate to benign physical findings
  - Diagnosis: mesenteric angiography, AXR, Barium enema showing “thumb print” sign
  - Treatment: IVF, papaverine injection into SMA (if arterial), heparin (if venous thrombosis, hx of DVT)
- Chronic mesenteric ischemia
  - Symptoms: postprandial pain (because of increased splanchnic demand for blood)
  - Diagnosis: angiography
  - Treatment: surgical revascularization

Rectal bleed
- Internal hemorrhoids: bleed, don’t hurt. Treatment: rubber band ligation
- External hemorrhoids: hurt, don’t bleed. Treatment: surgery

Management: Resuscitation (IV fluids, transfusion)
- CBC, PT/PTT/INR, LFTs, Bun/Cr ratio (elevated with upper GI bleed if no concurrent renal issues)
- Upper: EGD with sclerotherapy, gastrografin swallow study if you suspect perforation
  - 80% stop bleeding spontaneously (e.g. Mallory-Weiss), only need supportive therapy.
- Lower: r/o hemorrhoids.
  - Slow bleed (<0.5ml/hr): radionuclide (99mTc) scan.
  - Fast bleed (2+ ml/hr): emergent angiogram.

Indications for surgery:
1. Hemodynamically unstable (no response to IVF), transfusion, endoscopic intervention, or correction of coagulopathies
2. Severe initial bleed or recurrence of bleed with endoscopic treatment
3. Continued bleeding for more than 24 hours
4. Visible vessel at base of ulcer (30-50% rebleed)
5. Ongoing transfusion requirement (4-6+ units of blood required, unstable, blood type indeterminate/Jehovah’s witness)
REFLUX ESOPHAGITIS

Pathophysiology:
- Causes:
  - Incompetent LES: Structurally defective sphincter (ex. hiatal hernia)
    - Type I: sliding
    - Type II: paraesophageal. Tx: surgery, risk of strangulation - acidosis, hypotension, gastric volvulus
    - Type III: Mixed
    - Type IV: Other visceral besides stomach herniate
  - Insufficient clearance of acid
  - Increased intragastric pressure: distension with air, food, delayed gastric emptying, ↑ abd. pressure (COPD)
- Prolonged exposure of esophagus to low pH from gastric contents (acid, pepsin, and duodenal contents such as biliary and pancreatic secretions) leads to irritation of the esophageal mucosa (and respiratory epithelium).
- Prevalence increase with age.

Signs/Symptoms:
- Retrosternal pain after eating (heartburn)
- Exacerbated by lying down, may mimic cardiac chest pain, regurgitation
- Waterbrash (reflex salivary hypersecretion), cough, early satiety
- Atypical: N/V, post-prandial fullness, choking, chronic cough, wheezing, hoarseness
- Laryngitis (if severe), aspiration pneumonitis/recurrent pneumonia, idiopathic pulmonary fibrosis, asthma.
- If GERD is associated with dysphagia, think peptic stricture/mobility disorder/cancer
- Long term: dental erosion, gingivitis

Diagnosis:
- Rule out cardiac and pulmonary disease (H&P, ECG, cardiac enzymes)
- Diagnostic tests are not necessary for typical, uncomplicated, initial cases of GERD due to empiric treatment.
- Endoscopy with biopsy
- 24 hour ambulatory pH monitoring of the esophagus = gold standard (useful for determining severity)
- Barium swallow study to look for anatomical cause (achalasia, hernia, stricture) or complications from GERD
- Esophageal manometry = to determine competence of LES, only if mobility disorder suspected

Treatment: Goal: prevent progression to Barrett’s, cancer, pulmonary problems (recurrent aspiration)
1. Lifestyle modification, medication to reduce acid:
   - Elevate head of bed, eat small frequent meals, don’t sleep for 3-4 hours after eating
   - Antacids
   - Education to avoid EtOH, coffee, chocolate, peppermint (aggravate), nicotine (decreases LES tone),
2. Add H2 blocker
3. Switch to PPI
4. Metoclopromide: to promote gastric emptying

Medical therapy trial 6-12 weeks before further diagnostics. 85% resolve.
5. Combination therapy (phase 2+4, phase 3+4, or increase 2, 3, dose)
6. Surgery

Indications for surgery: Failure of medical therapy (Nissen) OR dysplasia of esophageal mucosa (resection)
- Preoperative studies: EGD, Manometry, endoscopy, barium swallow
  - Manometry to demonstrate normal peristalsis for postop feeding and abnormal LES tone.
    - If normal LES tone, do 24 hour pH monitoring to be sure gastric reflux is present

Complications/Considerations:
- Patients who initially present with symptomatic GERD for 5+ years should have endoscopy with biopsy first. If patient has documented Barrett’s metaplasia without dysplastic changes, surveillance endoscopy every 3 years.
- If patient suffers from recurrent aspiration pneumonia with reflux esophagitis, bronchoscopy with aspirate cytology (which will show lipid-laden macrophages) is indicated.
TRAUMA & SHOCK

Four phases: Primary survey, resuscitation, secondary survey, definitive care

Background: Deaths are trimodal: Immediate (major vessels, brain), Early (head), Late (sepsis, MODS)

1) PRIMARY SURVEY: ABCDEF

Airway (and c-spine)
- Assess airway patency, jaw thrust to initially open airway (protects c-spine) clear foreign bodies, insert temp airway
- Intubate if: Use orotracheal (unconscious), nasotracheal (conscious, no basilar skull f.), cricothyroidotomy (facial/neck)
  - Airway compromise: supraglottic (stridor, snoring, gurgling), laryngeal (dysphonia, hoarseness), hematoma
  - Inadequate respiratory effort (Apnea, agitation = hypoxia, obtundation =hypercarbia)
  - Inability to protect the airway (GCS < 8, aspiration risk)
  - Severely compromised respiratory mechanics (Retractions, sucking chest wound)
- Assume C-spine injury if: Perform: In-line immobilization of C-spine: Cervical collar AND backboard.
  - Head injury Clear C-Spine by: Palpating neck, assessing neuro, No EtOH, CT
  - Altered level of consciousness
  - Multisystem blunt trauma

Breathing
- Inspect, auscultate, and palpate chest (Is trachea midline? Neck veins distended?  Ventilating both lungs?)
- Ensure adequate ventilation and immediate identify and treat: pneumothorax, flail chest, etc.

Circulation/Control of hemorrhage
- Control obvious hemorrhage.  Use direct pressure, no tourniquet.   Don’t use vasoconstrictors.
- Assess tissue perfusion: pulse (rhythm, rate, quality), skin color, level of consciousness.
- Place two large bore (16g or bigger) IVs.   Resuscitation step starts here simultaneously.

Disability (neurologic)
- Glasgow Coma Scale or AVPU (alert, responsive to verbal, responsive to pain, unresponsive)
  - EYE opening:  Spontaneous (4), verbal (3), painful (2), None (1)
  - VERBAL: Orient x3 (5), disoriented (4), inappropriate (3), incomprehensible (2), None (1), intubated (T)
  - MOTOR: Verbal (6), localize pain (5), withdraw pain (4), flexor (decort) (3), extensor (decereb) (2), none
  - Prognosis, prediction of mortality. 3-4: 97%, 5-6: 65%, 7-8: 28%

Exposure/Environment: Undress patient, give warm blankets to prevent hypothermia

2) RESUSCITATION  Performed simultaneously with the primary survey (translation: nursing staff) > 1500ml = transfusion

  - Class I:  15% (750ml) HR < 100  Urine 30ml/hr+  Crystalloid only
  - Class II:  15-30% (750-1500ml) HR 100+ Urine 20-30 ml/hr  Crystalloid only
  - Class III: 30-40% (1500-2000ml) HR 120+ Urine 5-15 ml/hr  Crystalloid AND blood
  - Class IV  40%+ (2000ml+) HR 140+ Urine negligible Crystalloid AND blood
- Gastric intubation (use OGT if think that the cribriform plate is fractured, don’t want to suck out brains with a NGT)
- Vitals, pulse-ox, ECG monitored during this phase

3) SECONDARY SURVEY
- Detailed head-to-toe assessment
- AMPLE History (Allergies, Meds, PMH/Pregnant, Last meal, Events of injury)
- ECG, pulse oximetry, baseline labs, NG or OG if basilar skull f. (prevents gastric dilation - vasovagal hypotension)
- Portable x-ray (AP chest, AP pelvis, lateral c-spine)
- FAST, diagnostic peritoneal lavage (DLP)(may miss retroperitoneal hemorrhage), any other radiography

4) DEFINITIVE CARE

Thoracic trauma
  - Tension pneumothorax: needle in 2nd intercostal space, chest tube in 5th intercostal space
  - Open pneumothorax: cover with vaseline gauze.  Tape on 3 sides.
  - Massive hemothorax (1500ml): insert chest tube with cell saver
  - Flail chest: respiratory problems due to lung contusion (V/Q mismatch), splinting.  Intubate/spinal epidural, PEEP
  - Cardiac tamponade: pericardiocentesis, exploratory thoracotomy to find bleeding source (can be myocardium)
  - Blunt cardiac injury: ECG monitoring with antiarrhythmics and inotropic support ready
  - Aortic rupture: often @ L subclavian artery.  PA CXR (wide mediastinum), helical CT, angiogram, TEE.  Open repair.
  - Traumatic diaphragmatic rupture: 90% is left (liver on right).  Bowel sounds in chest, NGT.
  - Tracheal laceration: crepitus, hemoptysis, pneumomediastinum.  Need intubation or operative repair.
  - Nonlethal injuries: Simple pneumothorax (observe, chest t), simple hemothorax (<1500), rib fracture (pain control)
• Abdominal trauma: anything below the nipple in males or scapula.
  o Diagnosis
    ▪ FAST: RUQ (Morison’s pouch), LUQ (Splenoportal recess), pelvis, pericardium --> expl. laparotomy
    ▪ DPL: 10ml gross blood, 100k RBCs, 500wbc's, bacteria, bile, amylase > serum. (98% sensitive) --> ex. lap.
    ▪ Deteriorating vitals, falling hematocrit, worsening abdomen --> exploratory laparotomy.
    ▪ Abdominal CT: use in hemodynamically stable patients
  o Treatment
    ▪ Zone 1 (central retroperitoneum): Explore because likely to be aorta or pancreas.
    ▪ Zone 2 (lateral retroperitoneum): Observe if stable, even if kidney is injured.
    ▪ Zone 3 (pelvis): Externally stabilize. Arteriography if this fails to stop bleeding. Exploration difficult.
    ▪ Splenic rupture: low-grade (expectant), moderate (contrast, embolization), instability (ex. lap)
    ▪ Liver laceration: all but liver avulsion managed expectantly unless unstable (Pringle: port triad clamp)
    ▪ Hollow viscous injury: Abdominal CT with free air --> repair or resection
    ▪ Pancreatic: resection with temporary duodenal diverticulum
    ▪ Gunshot: Exploratory laparotomy regardless of hemodynamic status.
  • Head trauma: most common cause of trauma death. 60% of MVA deaths.
  o SCALP (Skin, subCut fat, Aponeurosis, Loose areolar connective tissue, Periostium)
  o CBF = CPP/CVR. CPP=MAP-ICP. Normal ICP<10. ICP > 20 requires treatment. Brain death when CPP < 50 mmHg
    ▪ Cushing reflex: Brain tries to protect CPP by increasing BP. Triad: Hypertension, bradycardia, resp. dep.
    ▪ CVR inversely related to PCO2. Linear in 20-80mmHg.
    ▪ Goal: keep ICP <20mm Hg and CPP 60-70 mmHg
  o Diagnosis
    ▪ Periorbital bruise(raccoon eyes), perimastoid (Battle’s sign), hemotympanum, CSF (ring sign on paper)
    ▪ CT without contrast and cervical radiographs
    ▪ ICP monitoring: ventricular ("gold standard"), subarachnoid bolt, fiberoptic,
  o Types of injuries
    ▪ Concussion: brief loss of neurologic functon (59% of head injuries)
    ▪ Diffuse axonal injury: Gray-white junction. 90% coma, CT scan shows no lesion. Nonsurgical mgt.
    ▪ Contusion: coup-contrecoup. Focal deficits, confusion. (15% of head injuries)
    ▪ Subdural: Clinically evident when serious underlying brain injury has occurred. (30% of head injury)
    ▪ Subarachnoid: Headache, stiff neck. Not surgical because no mass lesion.
    ▪ Intracerebral: Severe. Associated with diffuse axonal injury, gunshot wounds.
  o Nonsurgical treatment
    ▪ Pressors, head elevation
    ▪ Hyperventilation to PCO2 of 26 to 30 mmHg (only if signs of impending brain herniation)
    ▪ Osmotic diuresis (Mannitol): 0.25 – 1g / kg with a goal of 300 - 310 mOsm
    ▪ Hypertonic saline
    ▪ Sedation: reduces metabolic demand ICP down (Barbiturates)
  • Neck injury: Any injury penetrating the platysma carries risk of underlying severe injury
    ▪ Indications for surgery: vascular, airway, esophagus injury, penetrating injury in zone II
    ▪ Zone I (below cricoid cartilage): high mortality because occult. Angiogram, especially if in subclavian area.
    ▪ Zone II (cricoid to angle of mandible): usually obvious hematoma, easy surgery. Exploratory surgery.
    ▪ Zone III (above mandible): hard to do surgery on. Angiogram
  • Spinal shock: flaccidity and areflexia instead of expected hyperreflexia. Give steroids within 8 hours.
  • Trauma in pregnant women
    ▪ Resuscitation is most important variable. Once shock develops, fetus survival is only 20%.
    ▪ Place in left decubitus to increase venous return.
    ▪ Greatest risk of radiation-induced anomalies is during the first 16 weeks of gestation. <10 rad okay. CXR ok.
  • Compartment syndrome
    ▪ Treatment: fasciotomy. Assess renal function since myoglobinuria (alkalinize, give fluids)
    ▪ Decreased sensation: pain first (unmyelinated C-fibers), touch, proprioception.
    ▪ Pain on passive movement. Decreased pulse is unreliable because it is a late finding.
    ▪ Compartmental pressure > 35-40 mmHg confirm diagnosis
SHOCK: Inadequate end-organ perfusion
Hemorrhagic Shock: Hypovolemia due to bleeding. Stop bleeding, restore volume. DO NOT use pressors.

- Large bore needles to infuse LR, blood: Infuse faster, small would hemolyze blood products
1. Infuse 2 L of Lactated Ringer and check for response in vitals.
2. No response: Bolus 1-2 L again.
3. No response: Infuse blood and/or more crystalloid.
- Field adjuncts: PASG/MAST pants, inflate below the abdomen and increase SVR and L ventricle afterload, increasing BP.

Cardiogenic shock: myocardial dysfunction due to cardiac tamponade, contusion, air embolus, MI (rare)
- Cardiac tamponade
  - Beck's triad: JVD, hypotension, muffled/distant heart sounds
  - Pulsus paradoxus (decrease of BP >10 with inspiration)
  - Kussmaul's sign (an increase in CVP with inspiration, usually as jugular venous distention (JVD))
- Tension pneumothorax: produces shock by impeding venous return.

Neurogenic shock: injury to descending sympathetic pathway (caused by high thoracic and cervical spine injuries)
- UNIQUE: hypotension without tachycardia (no sympath. response)
- Unlike hemorrhagic shock, can use vasoconstrictors to achieve hemodynamic stability.

Septic shock: not really assoc. with trauma; resembles hypovolemic or normovolemic shock.

Systemic Inflammatory Response Syndrome
- SIRS 2+ of the following:
  - temp >38 or <36
  - HR >90bpm
  - RR >20 or PCO2 <32 or mechanical ventilation
  - WBCs >12,000 or <4,000 or >10% bands
- Sepsis SIRS + identifiable source of infection
- Severe sepsis Sepsis + organ dysfunction
- Septic shock Sepsis + CV collapse requiring vasopressors

Indications for Swan-Ganz (CVP, pulmonary artery - PCWP)
- Diagnosis of shock states, pulmonary edema
- Assessing hemodynamic response to resuscitation
- Example: post-operative normal vitals but oliguria
  - PEEP can cause decrease in CO, oliguria
  - CVP high: cardiogenic shock
  - CVP low & PCWP high (>20 mmHg): pulmonary edema

Normal values measured by Swan-Ganz
- CVP/Right atrium: 0-6*
- Pulmonary artery: 20-30/6-12* PCWP/Left atrium: 6-12*
- Left ventricle: 100-140/5-14 Arteries: 100-140/60-80

Burns
- Criteria for referral to burn center:
  - Full thickness >5% BSA or partial thickness >20% BSA
  - Age <5 or >50. Face, hands feet, genitalia, perineum
  - Inhalational injury, Circumferential, Chemical or electrical
- Epidermal burn (1st degree)
  - Red from capillary dilation. Blanches with pressure. No blister. Limited physiologic effects
  - Treatment: PO H2O, analgesia
- Partial-thickness burn (2nd degree)
  - Into dermis. Superficial: eschar, deep: leathery coagulation necrosis (white)
  - Treatment: Conservative for 10-14 days to see if skin regenerates
- Full-thickness burn (3rd degree)
  - Painless. Skin proteins contract when burned, creating tourniquet-like contraction
  - Treatment: require skin grafting
- Fluid Replacement (Parkland Formula): 4 x %TBSA x kg of Lactated Ringer's.
  - Give 1/2 the requirement over 8 hours, 1/2 the requirement over following 16 hours. Maintenance: D5W
  - Colloid (if the patient fails to respond to crystalloid after 24 h): 0.5ml plasma x %BSA over 8 hours
- Topical antibiotics. Don’t use systemic because they don’t penetrate eschar and select for resistant bacteria.
  - Silver nitrate: Broad spectrum. Painless - limited eschar penetration.
  - Mafenide acetate: Broad spectrum except yeast. Painful - good eschar penetration. Acidosis, hypersensitivity
  - Silver sulfadiazine: Gram + and yeast only. Painless - limited eschar penetration. Neutropenia/thrombocytopenia.
ONCOLOGY: General rule: any lymph node >1cm, 4+ week, w/out infection should be biopsied.

BREAST CANCER

Workup by presentation:

Palpable mass
- <30 years old: ultrasound – cystic or solid.
  - Mammogram useless due to dense breast tissue
- >30 years old: mammogram
  - Characterizes lesion as cystic or solid
  - Examines for synchronous lesion in contralateral
- Biopsy if solid (stereotactic, needle guided)
  - Infiltrating ductal carcinoma: see staging table
  - DCIS
    - Multicentric: simple mastectomy
    - Solitary: Lumpectomy + nodes
  - LCIS: mammogram every 6 months

Bloody discharge
- Mammogram, galactograph, resection of papilloma

18 year old rubbery, movable mass
- Fibroadenoma (98%). Observe for a few menstrual cycles

35 year old, 10 year cyclic tenderness: Fibrocystic change
- Aspirate cyst, send bloody fluid for cytology
- Biopsy if cyst does not reduce or if it recurs

Cracks, abscess, fluctuant, red hot mass, fever, leukocytosis
- ONLY “allowed” in lactating women, otherwise do full workup for possible inflammatory cancer

Risk factors
- Age, family history (first degree relatives), previous breast cancers, benign breast disease, palpable masses, estrogen replacement therapy, age of menarche, age of menopause, nulliparity.

Diagnostic Studies:
- Mammography (2 views each breast): used for screening and diagnostics. Purpose is to detect non-palpable cancer.
  - Most common finding: microcalcifications and round/smooth dominant masses (cystic lesion or fibroadenoma)
  - Suspicious finding: spiculated lesion.
  - 5-10% false negative rate so if suspicious, continue workup regardless of radiography
- Ultrasound: Most useful in young women (<35) with dense breast tissue. Can distinguish between a mass and a cyst
  - Can be used for guided biopsy. Like mammography, doesn’t replace clinical suspicion.
- FNA: 20% false negative rate. Best for cysts. It only tells you cytologic diagnosis, not histologic.
- Core needle biopsy (CNB): risk of hematoma, pneumothorax, and also has a 20% false negative rate
- Excisional biopsy: definitive histologic diagnosis. Can be diagnostic AND therapeutic. (surgery under anesthesia)
- Needle localization breast biopsy: good for non-palpable lesions detected on mammogram(surgery under anesthesia)

Screening
- Self exam: monthly, 5 days post period
- Physician breast exam: 20-30 (q 2-3 years), 40+ (annual)
- Mammogram: 35-45(baseline), 40-50(q 1-2 years), 50+ (annual)

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THYROID “24 year old woman with asymptomatic 3cm neck ‘bump’ she noticed 1 week ago. She is otherwise well.”

Risk Factors
- Radiation, family history of MEN, solitary nodule (vs. multinodular), hoarseness or voice changes
- Cyst which is large (>4cm) or recurs - 15% risk of malignancy.
- Women, aged 25-65.

Diagnosis
- Fine needle aspiration (FNA) for cytology
  - Radiolabeled iodine uptake scan if equivocal
- Family history of Men-2A: serum calcium (PTH hyperplasia), calcitonin (medullary), epinephrine metabolites (pheo.)
- TSH, ultrasound: Not necessary for solitary nodule, but may be useful if multinodular, or hyper/hypothyroid symptoms

Cytology results:
- Papillary: Psammoma bodies - spreads via lymphatics
- Follicular (more aggressive than papillary): spreads hematogenously
- Medullary: 20% associated with MEN. Diagnosis: Amyloid deposits on FNA, serum calcitonin levels
- Anaplastic: worst prognosis
- Lymphocytic infiltrate: lymphoma or chronic thyroiditis. Differentiate with flow cytometry.
- Adenoma: Hurtle cells on FNA

Treatment:
- Papillary: Smaller lesions (<1cm) and no history of radiation: lobectomy. 100% 10-year survival in low-risk.
- Follicular: microinvasive and smaller than 4cm: lobectomy
- Other papillary, follicular, medullary, anaplastic (if resectable): Total thyroidectomy
- Anaplastic (unresectable): chemotherapy and radiation
- Lymphoma: radiation
- Adenoma: Hurtle cells are somewhat equivocal, so lobectomy is still necessary.
- Grave’s disease: subtotal thyroidectomy

Postoperative
- Thyroid suppression with thyroid hormone
- Possibly \( {^{131}}I \) ablation in Papillary and Follicular, NOT Medullary since it is of parafollicular C-cell origin.

Complications of thyroidectomy
- Parathyroid resection: Low calcium, high phosphate manifested as perioral tingling, parasthesia, MS change
- Recurrent laryngeal nerve damage (0.5-5% risk): Hoarseness. If bilateral, may require tracheostomy.
COLORECTAL CANCER

• **Screening:**
  ○ Fecal occult blood testing annually after 50 years of age. 20% positive predictive value
  ○ Sigmoidoscopy at 50 and at 5 year intervals OR colonoscopy at 50 and at 10 year intervals

• **Presentation:**
  ○ Most common cause of bowel obstruction in an individual without prior abdominal surgeries (generally right sided cancers have worse prognosis due to late presentation (later obstruction vs. left-sided tumors))
  ○ Heme-positive stool (generally right-sided cancers)
  ○ Unexplained anemia in a male OR FEMALE over 40-50
  ○ History of inflammatory bowel disease (especially UC)
  ○ Family history of colon cancer, FAP, HNPCC, Peutz-Jeghers

• **Diagnosis:** Colonoscopy & biopsy
  ○ 50% of colon cancer occurs in the rectum, 20% sigmoid, 15% ascending

• **Treatment**
  ○ Hemicolecotomy + node dissection (50% involvement)
    ■ Stage I + II (any T, no nodes, no mets): Resection only
    ■ Stage III + IV (nodes or mets): Neoadjuvant and postoperative
  ○ Baseline CEA (to help detect recurrences)
  ○ Chest X-ray (to look for metastasis)
  ○ Liver function tests to look for metastasis - liver is the most common site - 20% at diagnosis. 0% 5-year surv.

• **Prognosis:** *most important variable is lymph node involvement*
  ○ 20% recur. 90% of recurrences in 3 years.
  ○ T1 (A): limited to mucosa/submucosa
  ○ T2 (B1): invades into, not beyond muscularis
  ○ T3 (B2): full thickness
  ○ T4: invades other organs
  ○ Positive nodes (C)
  ○ Distant mets (D)
  ○ Stage 5-year survival

• **Postoperative:** Colonoscopy or barium enema every 6 months. Bimonthly CEA level (70% predictive). PET, CXR, LFTs.
  ○ Recurrence: Liver metastasis
    ■ Resectable: 33% 5-year survival. Get 1 cm margin.
    ■ Unresectable: alcohol, cryotherapy, RF ablation, chemoembolization
  ○ Anastomotic leak: persistent ileus, mechanical obstruction from adhesions, Richter’s hernia
    ■ CT, small bowel series, Gastrografin enema

RECTAL SQUAMOUS CANCER

Diagnosis: transrectal ultrasound (wall invasion), CT (invasion of surrounding structures - prostate, bladder, ureter)

Staging: same as with adenocarcinoma

Treatment: <5cm from anal verge: can spread via inguinal lymphatics, margins include sphincter mechanism

Small tumor: small resection, larger tumor: NIGRO neoadjuvant therapy
LUNG CANCER

Presentation:
- Smoker (85%) with a NEW “coin” lesion discovered on chest x ray or a rapidly enlarging one (a few months)
  - If enlarges “too fast” (e.g. a few hours), likely is an infectious process (ex. abscess)
- Pleural effusion without CHF
- Hemoptysis

Diagnosis
1. Sputum cytology
2. CT (Chest AND abdomen for mets)
3. Biopsy (percutaneous if lesion is peripheral, broncoscopy-guided if lesion is central)

Staging:
- Stage I: limited to lung (70% cure rate)
- Stage II: hilar nodes only. Mediastinal would make it IIIIB (40-50% 5 year survival)
- Stage III: beyond lung
- Stage IV: distant mets

Treatment
- Non-small cell lung carcinoma: Resection, radiation, no chemo: 14% 5 year survival
  - Preop assessment: FEV1 (800ml minimal to survive), V/Q scan to see how valuable that lung is
- Small cell lung carcinoma: Non operable. Chemo, radiation if localized: 2 years max to live

MELANOMA

General Principles
- Above the lip: Basal cell (doesn’t metastasize, responds well to local excision). Do Moh’s excision, chemo, radiation.
- Below the lip: Squamous cell (metastasizes, but still pretty good prognosis). Get 1 cm margins.
  - Excise palpable lymph nodes only. Chemo, radiation.

Presentation: ABCDs: Asymmetry, Border, Color (black color & multiple colors are worse), Diameter (> 0.6cm is suspicious)

Risk factors
- Fair skin, sun exposure, family history, genodermatoses (Xeroderma pigmentosum), advanced age, increased number of moles (50% of melanomas arise from existing moles), dysplastic nevi, giant congenital nevi

Types: All have the same prognosis when controlled for thickness
- Superficial spreading (75%)
- Nodular: worst prognosis, grows vertically early
- Lentigo maligna: best prognosis, typically on head & neck from a Hutchinson’s freckle (slow growing)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clark level</th>
<th>Breslow thickness</th>
<th>TNM</th>
<th>Treatment</th>
<th>5-year survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>I: epidermis</td>
<td></td>
<td>T0N0M0</td>
<td>0.5-1 cm margin = cure</td>
<td>&gt;95%</td>
</tr>
<tr>
<td>I</td>
<td>II: papillary dermis</td>
<td>≤ 0.75 mm</td>
<td>T1N0M0</td>
<td>Resect 1 cm margin</td>
<td>89%</td>
</tr>
<tr>
<td>I B</td>
<td>III: breadth of papillary dermis</td>
<td>0.75-1.5 mm</td>
<td>T2N0M0</td>
<td>75%</td>
<td></td>
</tr>
<tr>
<td>II A</td>
<td>IV: into reticular dermis</td>
<td>1.51-4 mm</td>
<td>T3N0M0</td>
<td>Resect 2 cm margin</td>
<td>58%</td>
</tr>
<tr>
<td>II B</td>
<td>V: subcutaneous fat</td>
<td>≥ 4 mm</td>
<td>T4N0M0</td>
<td>Resect 2-3 cm margin</td>
<td>25%</td>
</tr>
<tr>
<td>III</td>
<td></td>
<td></td>
<td>T anyN1M0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td></td>
<td></td>
<td>T anyN anyM1</td>
<td>Interferon IFN-a2b</td>
<td></td>
</tr>
</tbody>
</table>

- Ulceration: Bumps stage up one level. 1/3 reduction in survival regardless of thickness.
- Sentinel node biopsy if palpable nodes. Dissect remainder of nodes if sentinel node is positive.
- Get CXR, AXR looking for mets. CBC, LFTs.
PREOPERATIVE ASSESSMENT and POSTOPERATIVE FEVER

Prognostic Factors

Cardiac
- CHF -> JVD
- MI within 7 months
- Arrhythmia
- Age > 70
- Emergent

Pulmonary
- FEV1

Hepatic
- Bilirubin
- Albumin
- PT
- Encephalopathy

Nutritional
- Recent weight loss
- Albumin <3
- Skin anergy

Postoperative fever
- Wind (Atelectasis) POD 1
- Water (UTI) POD 3
- Walking (DVT) POD 5
- Wound POD 7
- Wonder where (Abscess) POD 10-15
- Wonder drug POD 7+

SOURCES:
- Essentials of General Surgery, 4th edition, 2006, Peter E. Lawrence, Lippincott Williams & Wilkins
- NMS Surgery Casebook, 2003, Bruce E. Jarrell, Lippincott Williams & Wilkins
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- Surgical Recall, 5th edition, 2009, Lorne H. Blackbourne, Lippincot Williams & Wilkins