Acknowledgement
Much of this material was assembled and made publicly available by the Class of 2005, with further input from Pocket Medicine, Surgical Recall, and the NMS Surgery Casebook. Each review was meant to be a stand-alone study guide for the surgery oral exam. They are in the framework of thought process that is part of a typical patient encounter, from development of a differential diagnosis to information gathering and treatment planning.

A Note on Abbreviations
Abbreviations were essential to shorten the material, but many are not standard. For example, components of the history of present illness may be written as: “HPI: O/C/Qu/S/F/D/L/R/AA Fx/ASx,” which stands for onset, context, quality, severity, frequency, duration, location, and radiation of symptoms, as well as aggravating and alleviating factors, and associated symptoms. “Fx” may also be used to denote failure. Cancer is usually abbreviated “CA.”

General Format for Addressing Oral Exam Questions
Approach every patient the same way!

1. repeat case history and chief complaint

2. if the patient appears acutely ill, always first assess ABC’s and resuscitate as necessary
   • assess ABC’s = note all 5 vital signs, UOP & 24° I/O’s, cardiopulmonary exam, brief neuro exam (level of alertness, etc.), and any other available data (CVP, PCWP, labs, etc.)
     a. Airway: stabilize spine and assess airway availability/threats
     b. Breathing: O2/bag-mask/intubate if necessary
     c. Circulation: stop bleeders, start IV’s, Foley, monitors (O2, EKG), defib, etc. as necessary
        • automatic for any major bleed: 2 IV’s with LR and/or blood, Foley, monitors, CBC/T&C/coags

3. consider differential dx

4. don’t get sidetracked short of taking a full H&P (CC, HPI, PMH/PSH/Ob-Gyn [pregnancies, menses, sexual hx], Meds/All, FH/SH, Health Maintenance Activities, ROS)
   • basic 14-system ROS (systematic, head-to-toe) – be appropriately selective, but in most cases, it’s helpful to at least touch on Gen, HEENT, GI, and GU
     Gen: fever, chills (infxn); night sweats, fatigue, anorexia, wt loss (CA); pain
     Psych: depression
     Neuro: headaches, LOC, weakness, numbness/tingling
     Skin: skin chg’s
     HEENT: palpable lymph nodes, vertigo, dysphagia, hearing or vision chg’s
     Breasts: chg’s
     Pulm: SOB, cough, hoarseness, hemoptysis
     CV: chest pain, extremity edema, palpitations
     GI: anorexia, dysphagia, regurg/heartburn, N/V, abd pain/cramping, last BM, chg’s in BM’s – diarrhea/constipation/blood/tenesmus/timing
     GU: dysuria, incr freq/urgency, slow stream, hematuria
     Repro: ED, irregular menses, vaginal bleeding
     Heme: abnormal bruising
     Endo: polyuria, polydipsia, polyphagia, excessively warm/cold
     Musc: joint pain, weakness

5. complete exam: start with vital signs & gen appearance, then head to toe (Neuro, HEENT, etc.)

6. order appropriate labs, non-invasive studies, invasive studies, and confirmatory tests to make your working diagnosis – beware making too much out of nonspecific results

7. with malignancy, always figure out how you’re going to stage the dz and work-up for metastases (e.g., to find bone mets – H&P, bone scan; liver mets – LFT’s, CT; lung mets – CXR; brain mets – H&P, CT)

8. treatment – know the options, criteria for choosing among them, likely outcomes, and possible complic’s
   • take resuscitative measures first
   • identify and fix causes if possible, treat effects, prevent complications, and prevent recurrence
**Topic 1: Breast Mass**

**Differential Dx: assume cancer if > 35 y/o**
- benign disease: mastitis (Strep/Staph), fibrocystic dz (lumpy breasts with premenstrual tenderness, worse with caffeine & low risk of CA), fat necrosis
- benign neoplasms: fibroadenoma (freely movable, nontender, rubbery mass), phylloides tumor (large, smooth malignant mass with calcifications), intraductal papilloma (bloody discharge)
- malig neoplasms: CIS – lobular/ductal, infiltrating lobular/ductal, Paget’s dz, inflam CA (lymphatic invasion), sarcomas

**History:** *start with basic pt data – age, menopause status*

- Mass: number & location, duration, chg in size, pain, chg’s with menses/breastfeeding, discharge – unilateral vs. bilateral, clear vs. bloody
- 4X Risk Fx’s: age (most imp); 1st deg FH of premenop BrCA; prev biopsy dx of atypical hyperplasia, LCIS/DCIS, or CA
- 2X Risk Fx’s: obesity, menarche < 12, first preg > 35/nullip, menopause > 55, h/o ovarian/endometrial CA, HRT, rads exp

**Complete Hx:** PMH / PSH / Ob-Gyn / FH (other CA, known genetic problems) / SH / HMA (exams/mammo) / ROS

**Physical Exam:** complete, including Vital Signs, Gen Appearance, etc., with focus on the breasts and axillae
- inspect for skin chg’s and asymmetry (4Sposition)
- palpate & describe mass, examine for nipple discharge, and palpate axillary nodes

**Further Workup:** (underlined is the definitive w/u for a highly suspicious mass, or in setting of high risk)
- if the patient is < 30, you can observe for resolution over 1-2 menstrual cycles & do an U/S if the mass persists – most will be cysts or fibroadenomas; if neither, do an FNA for cytology
- if the patient is > 30, do an U/S with FNA
  - if it’s a simple cyst with non-bloody fluid that resolved with aspiration, simply reassure pt & continue routine screening
  - if it’s a solid mass or persists after aspiration, do bilateral mammography & tissue biopsy; do an excisional biopsy if the core/fine needle biopsy is undiagnostic
  - **calcifications, border irregularity are concerning on mammogram**
    - galactography is another possibility when nipple discharge is present (retrograde contrast injxn)

**Stageing (TNM):** I (sm tumor, no nodes/mets), II (lg tumor, few nodes), III (many nodes), IV (mets)
- note: send fresh excisional biopsy specimens to test for ER/PR status & Her-2/neu overexpression

**Pre-operative Workup (for mets)**
- CBC, LFT’s
- bilateral mammography, CXR for lung mets, bone scan if symptomatic

**Treatment:** no fixed protocols; surgery (appropriate even for benign lesions like fibroadenomas, IDP’s, etc.), XRT, chemo, & hormonal tx

**Principles**
- tumor < 1 cm & no nodes: no adjuvant therapy necessary
- tumor > 1 cm: post-op adjuvant therapy required, typically XRT if nodes (−) & tamoxifen if ER (+)
- tumor < 4 cm: pt can choose between MRM & lumpectomy with SNB/ALND + XRT
- + nodes, Her-2/neu (+): post-op chemo required, regardless of tumor size, with ? benefit from Herceptin for Her-2/neu OE

**Specific Recommendations**
- LCIS: close surveillance ± chemoprevention; consider prophylactic b/l mastectomy
- DCIS: mastectomy or lumpectomy + XRT
- inflammatory breast CA: pre-op Adriamycin, modified radical mastectomy, followed by XRT
- Stage II: surgery + XRT, adjuvant chemo for tumor > 1 cm with (+) LN or ER/PR (−)
- Stage III: pre-op Adriamycin, tumor removal + axillary dissection, post-op XRT & chemo (anthracycline, 5-FU, taxanes)

**Surgical options:** RM (only necessary if you find extensive tumor invasion of pectoral muscles), MRM (no muscle removal, but everything else), SM (no nodes, done for prophylaxis), lumpectomy with SNB/ALND
- * spare long thoracic n., thoracodorsal n., pectoralis bundle, and intercostobrachial n.

**Screening:** women 20-39 – monthly self-exam, clinical exam q 3 yrs; > 40: monthly self-exam, yearly clinical exam & mammo (or every other yr, 40-49)

**Prevention:** SERM’s (Tamox decrease risk of contralat breast CA in adjuvant setting, not clearly beneficial for prev, ? Raloxifene); BRCA1/2 carriers – surveillance, prophylactic mastec & salpingooopherectomies, limited data supporting Tamox

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**Topic 2: Changing Skin Lesion (mole)**
as with any topic on CA, prevention comes into play – avoid sun exposure, skin self-exams, etc.

Differential Dx (CIINTS)
- noncancerous skin lesions: actinic keratosis, seborrheic keratosis, and nevi
- cancerous lesions: basal cell CA (75%), squamous cell CA (20%), melanoma (4%)
- types of melanoma: superficial spreading (75%), lentigo maligna, acral lentiginous, nodular, amelanotic

History
- HPI: onset, changes, associated S/Sx: pruritis, pain, bleeding
- risk factors for malignancy … white patients with blonde/red hair, freckling, etc.
  - SCC: sun exposure, arsenic, actinic keratosis (head, neck, and hands), immunosupp
  - BCC: sun exposure, fair skin, chronic dermatitis (head, neck, and hands)
  - Melanoma: history of blistering sunburns
- PMH: melanoma/skin CA, xeroderma pigmentosum (risk fx for SCC), dermatitis (BCC)
- ROS: systemic symptoms of malignancy, especially headaches or neurologic symptoms, palpable LN’s, SOB, abdominal pain, jaundice, GI bleeding, bone pain

Physical Exam (IP)
- Vital Signs& General Appearance
- Skin: note pale skin, head to toe assessment, ABCD’s of melanoma (D = diameter > 6 mm and dark lesions), ulcerations & irregular texture … most common site in men = back (1/3), women = legs (1/3)
- HEENT: ophthy exam, palpate lymph nodes for mets
- Rectal: examine for anal melanoma

If ABCD’s & RF’s point to melanoma: excisional biopsy (or incisional bx for lg lesions) – never do a shave biopsy!
- superficial spreading: most common type (75%), occurs in sun-exposed & nonexposed areas
- acral lentiginous; common in African-Amer’s; typically on palms & soles
- lentigo maligna: best prognosis; typically on head & neck, arising from a Hutchinson’s freckle
- nodular melanoma: worst prognosis, with a tendency to grow vertically
- amelanotic melanoma: from melanocytes but with lack of pigment

Melanoma staging systems: TNM, Clark’s, Breslow’s, and the AJCC
- T 0-4, N 0-3, M 0-1
  - Clark’s: I-V (I: epidermis, II: into papillary dermis, III: through papillary dermis but not into reticular dermis, IV: into reticular dermis, V: into SQ fat); progressively incr recurrence rates
  - Breslow’s depth: the actual depth; < 0.76 mm has a 90% cure rate with excision, > 4.0 mm has an 80% risk of recurrence/mets within 5 years
  - the AJCC uses 1.5 cm depth as cutoff between Stage I & II, positive nodes for III, and mets for IV

Classifying BCC: nodular (flesh-colored), superficial (scaly red plaque), morpheaform (hypopigmented), pigmented

Labs & Non-invasive Studies: LFT’s, CXR; (bone scan, CT scan, and MRI reserved for symptoms)

Treatment: adequate margin depends on depth of the cancer
- SCC: small lesions (< 1 cm) should be excised with a 0.5 cm margin, large lesions with a 1-2 cm margin
- BCC: excision with 1 cm margins (Mohs)
- Melanoma: melanoma in situ: 0.5 cm margin, 1 mm thick: 1 cm margin, > 1 mm thick: 2-3 cm margin – excision should go down to the fascia overlying the muscle (deep fascia)
  - Treatment for digital melanoma: amputation
  - Treatment for intestinal mets: surgical resection to prevent bleeding/obstruction (melanoma is the most common malignancy to metastasize to the bowel)
  - Anal melanoma: APR or wide excision (most common Sx: bleeding)
- Sentinel node biopsy if >1.0 mm (highly pref’d to LND because of high morbidity associated therewith)
- Adjuvant therapy: interferon-alfa 2b in patients with thick T4 tumors and nodal mets, plus other experimental therapies (vaccinations against melanoma-specific Ag’s, IL-2, chemotherapy, Ab’s, etc.)

Melanoma Prognosis: depth (Breslow’s), node status/mets, ulceration, site, pt age

**Topic 3: Neck Mass**

Resuscitation: typically unnecessary, but review pt’s ABC’s, especially for respiratory compromise
Differential Diagnosis (CIINTS): 80% benign in kids, 80% malignant in adults > 40 y/o
Infants: congenital – branchial cleft cysts (lateral), thyroglossal duct cysts (midline, elevating with tongue protrusion), dermoid cysts (midline submental), hemangioma, cystic hygroma
Adolescents: inflam/infxs – cervical adenitis, cat-scratch dz, mononucleosis, mumps (parotid); congenital
Adults: malignancy – squamous cell CA, 1° or mets; benign neoplasms – lipoma, salivary gland, salivary gland tumor

History:
- first note the patient’s age (thyroid nodules in younger pts are more concerning) & gender (males: > likelihood of CA)
  - CC (cardinal Sx): dysphagia, odynophagia, hoarseness, stridor (upper airway obstrxn), globus, speech disorder, referred ear pain (CN V, IX, or X)
  - HPI (focus on the mass): # & lxn, detailed desc, onset & prog of size/Sx (congenital [years] vs. malig [months] vs. inflam [days]); associated S/Sx, including pain, Sx of thyroid/parathyroid dysfxn or infxn
  - PMH: recent URI, radiation exposure (papillary thyroid CA), neck trauma, DM, HIV, other CA, Gardner’s syndrome
  - Complete Hx: PSH / FH (other CA’s – MEN?) / SH: smoking, EtOH, cats, sig other sick / HMA
  - ROS: F/C, fatigue, wt/app chg, depr & MS chg’s, palp’s, abd pain, kidney stones, bone pain, excessively warm or cold

Physical Exam (IPPA)
- Vital signs: tachycardia may suggest hyperthyroidism
- General appearance
- Neuro: CN palsies
- HEENT (in any case of dysphagia, dyspnea, etc.): complete exam, noting lxn of masses, solitary/dom vs. multiple, tenderness, elev with tongue protrusion, mobility vs. fixation, lymphadenopathy & mobility/consistency of LN’s; thyroid size, masses, and tenderness; tracheal mobility/deviation; mouth & oropharynx exam; check for exophthalmos
- Abd: HSM (mono in teenager)
- Musc/Extremities: pretibial myxedema, bone pain (mets)

Labs:
- CBC, Monospot, TFT’s (TSH, T3, T4, thyroid-stimulating Ab’s, antithyroglobulin & microsomal Ab’s), viral titers (EBV), PTH/Ca, calcitonin (medullary thyroid CA)

Non-invasive Studies
- U/S: will reveal the size & cystic (congenital) vs. solid; must be done prior to FNA if the mass if pulsatile
- CT, MRI helpful for deep, suspicious masses; CXR for mets

Invasive Studies (do not do an open excisional biopsy, because of adverse effect on survival if malignant)
- Evaluation of thyroid mass: TSH, FNA, RAIU – see below
- Scope & guided bx: for non-thyroid mass – laryngoscopy (speech Sx), bronchoscopy (resp Sx), esophagoscopy (GI Sx)

Evaluation of Thyroid Nodule (25% of cold nodules are malignant)
- #1: FNA or TSH – if TSH is normal, do FNA; if TSH is low, follow with RAIU (scintigraphy) – if RAIU is hot, you have a benign toxic adenoma (surgically resect) or Graves; if RAIU is cold (non-functioning) or non-specific, do FNA
- #2: FNA results:
  - (a) benign 70% – observe or provide suppressive Tx (PTU, etc.), (b) indeterm 15% – options: suppressive Tx with observation, RAIU, or surgery, (c) suspicious 10% – options: RAIU or surgery, (d) malig 5% – surgery

Diffuse Thyroid Enlargement
- Graves disease: most common cause of hyperthyroidism, diffuse goiter, exophthalmos, pretibial myxedema
  - Dx: incr T3/T4, anti-TSH receptor Ab’s, destr TSH, global RAI uptake
  - Tx for hyperthyroidism: medical blockade (PTU, methimazole: 50% chance of recurrence after 1 yr; iodine; propranolol – symptomatic relief), radioiodide ablation (most popular therapy, complicated by hypothyroidism in > 75% of pts), surgical resection (b/l subtotal thyroidectomy)
- Toxic multinodular goiter (Plummer’s syndrome): multiple nodules with one or more hyperfunctioning
  - Dx: nodule can be localized with a radioactive iodide scan
  - Tx: surgically remove hyperfunctioning nodule(s) with lobectomy or near-total thyroidectomy
    - Note: thyroid storms are treated with medical blockade (PTU, iodine, propranolol), hydration, cooling, and Dexamethasone
- Acute thyroiditis (etiologies: *Strep/Staph* 2/2 thyroglossal fistula; painful, swollen thyroid with fever)
  - Tx: antibiotics, drainage of abscess, needle aspiration for Ctx; most pts need surgical repair of the fistula
- Subacute thyroiditis (DeQuervain’s): caused by viral infxn following URI; Dx: elevated ESR
- Chronic thyroiditis
  - Hashimoto’s – most common cause of hypothyroidism in U.S.; firm, rubbery gland, 95% women; elevated antithyroglobulin & microsomal Ab’s
  - Reidel’s – benign inflammatory enlargement of thyroid with fibrosis (pres: painless, large thyroid)

Thyroid CA
- Papillary: most common, assoc with neck irradiation & Gardner’s syndrome, psammoma bodies, good prognosis – positive cervical nodes do NOT affect prognosis, either!
- Follicular: rubbery, encapsulated, mets to bone are most common, FNA will be indeterminate – proceed with surgery
Medullary: associated with MEN II & III, so look for pheo & hyperparathyroidism, too; elev calcitonin, esp when stimulated by pentagastrin; 95% cure rate when found by MEN screening, but < 20% cure rate when found by palpable mass
Hürthle cell: originate from follicular cells, FNA also indeterminate – proceed with surgery
Anaplastic/undifferentiated

Parathyroid CA: marked by hypercalcemia (>13, may cause a crisis), elevated PTH, a palpable parathyroid gland, neck pain, recurrent laryngeal n. paralysis
• labs: HCG (tumor marker), PTH/CA
• Tx - manage hyperCa with normal saline 4-6 L/day (200/hr), Lasix, bisphos, calcitonin, and glucocort’s
- surgical resection of PTh mass with ipsilateral thyroid lobectomy & LN resection

Laryngeal CA: assoc with tobacco, EtOH; most SCC; supraglottic, glottic, and subglottic

Salivary gland tumors: mobility vs. fixed on exam is key to determining benign vs. malignant; 80% of minor salivary gland tumors are malignant (usually mucoepidermoid carcinoma), 80% of parotid tumors are benign (usually pleomorphic adenomas)
• Dx: FNA (never do an excisional biopsy of a parotid mass)
• Tx: adequate surgical resection, sparing CN VII if possible; neck dissecn for node (+) & post-op XRT if high-grade

Mets: represent 80% of non-thyroid neoplastic neck masses
** initial objective: locate the primary tumor, primarily by noting the location of enlarged LN’s
  ○ LAN high in neck, posterior triangle: nasopharyngeal CA
  ○ Jugulodigastric LAN: tonsils, tongue, supraglottic larynx
  ○ Supraclavicular LAN: consider entire GI tract, lungs, breast, GU tract, and thyroid

Cancer Staging
• thyroid: CXR, radioisotope scan
• others (typically II-IV): CXR, CT scan, LFT’s

Treatment
Conservative Management
- in likely infxs etiology (F/C, elev WBC, tenderness), give a 2-week trial of Abx; if mass persists, do an FNA

Surgical Indications: congenital or neoplastic processes …
Radical neck dissection: removal of nodes from clavicle to mandible, SCM, submandibular gland, tail of parotid, LI, digastric, stylohyoid & omohyoid mm., fascia within the anterior & posterior triangles, CN XI, and cervical plexus sensory nerves
• Indications: clinically positive lymph nodes, fixed but resectable cervical mass
• Contraindications: distant mets, fixation to unremovable structure, low neck masses

Modified neck dissection, Type I (preserving CN XI), II (preserves CN XI & LI) or III (preserves CN XI, LI, and SCM)
• Advantages: decreased morbidity, adequate for many N0 lesions
• Disadvantages: ? increased mortality from local recurrence

Thyroid surgery options: total thyroidectomy (for CA > 2 cm) ± LN dissection, near-total thyroidectomy, lobectomy/isthmectomy

Post-op Management of Thyroid CA
• following surgery for thyroid CA, provide Synthroid to suppress TSH
• post-op radioiodide scan can locate residual tumor & distant mets that can be treated with ablative doses in the case of papillary and follicular CA

Topic 4: Abdominal Pain

Resuscitation: assess pt’s ABC’s & need for IVF, Foley, and NG tube; expect to aggressively resuscitate pts with pancreatitis, high-grade SBO, and GI bleed

Differential Diagnosis (CIINTS): appendicitis, cholecystitis, pancreatitis, diverticulitis, ulcers, bowel or ureteral obstrnx/perf, ischemic bowel, Crohn’s, UC, PID, perimenstrual pain, ovarian torsion, ruptured aneurysm
• NOTE: bowel ischemia occurs 2/2 (1) atherosclerosis (chronic postprandial abd pain), (2) low flow (identify the cause and fix medically), or (3) emboli (SMA, a-fib)
• REMEMBER: MI, pneumonia, DKA, hepatitis, and musculoskeletal pain may also manifest as abd pain
History: pt’s age & gender are your first tipoffs to Dx; also differentiate acute vs. chronic (NOTE: young women’s abd pain is almost always appendicitis or gynecologic)

- HPI: LOCATION, onset/context, QUAL, progression, duration, prior h/o similar pain, aggrav/reliev fx (meals, pos’n)
- PMH: AAA, CAD, CVA, hyperlipidemia, CHF, HTN, GI disease, hernias, CA, sickle cell, STD’s, IBD
- PSH: abdominal surgery, including operative complications – major RxFx for SBO
- Ob/Gyn: LMP, parity (FFF > 40 y/o at higher risk for cholecystitis), contraception, menstrual prob’s
- Meds/All: Coumadin, NSAID’s
- FH: CF, CA, heart disease, AAA
- SH: smoking, EtOH, drugs, trauma
- HMA: colonoscopies, FOBT, etc.
- ROS: Gen – F/C, chg in wt/app, including anorexia; GI – last BM, chg in BM’s, D/C, blood, N/V – bilious vs. non-bilious/bloody; GU – urinary chg’s, vag d/c

Physical Exam: complete physical, focusing on vital signs, gen appearance, abd, rectal, and pelvic

- Vital signs
- General appearance: position in bed, motion?, obvious pain
- Abdomen: inspect (Cullen’s/Gray Turner’s signs), auscultate (bowel sounds?), percuss (distension), palpate (rebound/guarding – voluntary/involuntary, masses, McBurney’s point tenderness, psoas/obturator signs, Rovsing’s sign)
- Rectal: masses, blood
- Pelvic exam: cervical motion tenderness, bleeding

Labs: CBC, complete chemistries with LFT’s, beta-HCG, U/A, amylase/lipase, hepatitis screen, lactate – if a dx of pancreatitis is made, make sure you have all the labs necessary for Ranson’s criteria (LDH, ABG; G-200 A-250 L-350 A-55 W-16, C-<8 H-10% O-<60 B-<-4 B-<5 S-<6); T&S if bleeding

Non-invasive Studies: upright CXR, upright AXR, supine AXR, U/S (abdominal & pelvic), CT
  - U/S findings suggestive of cholecystitis = wall > 3 mm, pericholecystic fluid, distended GB, (+) stones

Invasive Studies: DPL, angiography

Key Points
- appendicitis: periumbilical pain S> RLQ, anorexia/nausea, if perf’d S> peritoneal signs, specific PE findings
- pancreatitis: try to ID etiology – EtOH, gallstones, trauma, Ca, hyperlip; epigast pain -> back; ≥ 7 RC 99% mortality, 5-6 40%, 3-4 15%, ≤ 2 5%; CT scan is appropriate
- acute cholecystitis: RUQ pain with fatty meals; order HIDA scan to confirm diagnosis (IV radionucleotide)
- diverticulitis: LLQ steady/cramping pain; if + fever or WBC, get a CT scan & do not scope; if not, go ahead

Treatment
Conservative Management
- appendicitis: IVF, pre-op & post-op Abx, x 7 days if perf’d (Cefoxitin)
- pancreatitis: NPO, IVF to maintain hemodynamics, with TPN and possibly Imipenem if severe necrotizing pancreatitis; in some cases, may need to recommend lifestyle chg’s
- acute cholecystitis: IV Abx (Amp/Gent); if very ill, observe for 24-48 hrs to stabilize
- diverticulitis: NPO, NG tube, IVF, IV Abx, no morphine – use meperidine for pain; if abscess, use CT-guided drainage
- if bowel ischemia occurs 2/2 low flow, start a glucagons drip to paralyze the muscularis and increase flow to the villi

Surgical Indications
- acute appendicitis or cholecystitis (go to the OR ASAP if the patient isn’t extremely ill & no IBD)
- free air/peritoneal signs (rebound, guarding, distension); if bowel perf’d, leave wound open
- refractoriness to medical management (e.g., diverticulitis - sigmoidectomy)
- no indication for OR for diverticulosis/-itis unless perf’d, refractory, or there is an abscess

**Topic 5: Bilious Vomiting of the Newborn (Intestinal Obstruction)**

* bilious vomiting indicates pathology beyond the ampulla of Vater, 1 of 4 signs of congenital intestinal obstruction, the others being polyhydramnios, abdominal distension, and failure to pass meconium … BV is a surgical emergency

Resuscitation: assess ABC’s, Foley, IVF to maintain 2 cc/kg/hr UOP, NGT, prophylactic Abx (watch for dehydration, sepsis)

Diff Dx: intestinal malrot +/- volvulus (until proven otherwise), SBO/atresia – duodenal/jejunal, intussusception, Hirschsprung’s, meconium ileus, meconium plug, necrotizing enterocolitis, small left colon syndrome

History
- CC: is the vomit bilious (yellow-grn)? (causes of non-BV include EA, TEF, GERD, pyloric stenosis, & gastroenteritis)
HPI: onset, context (infant’s health), frequency, progression, vomitus characteristics – amt, color, blood, AA Fx (knee/chest posture -> intussusception), associated Sx/pain

PMH: other medical problems (VACTERL: Trisomy 21 -> atresia), previous vomiting, prematurity/high stress birth (NEC, meconium plug), failure to pass meconium in first 24 hrs (Hirschsprung’s), polyhydramnios & other pregnancy complications, maternal diabetes (small left colon syndrome), recent viral illness (intussusception)

PSH, Meds/All

FH: Down’s (atresia), CF (meconium ileus), Hirschsprung’s

ROS: Gen – F/C, wt/app chg; GI – constipation -> Hirschsprung’s/meconium plug/ileus, diarrhea, meleina/hematochezia -> NEC, red currant jelly stools -> intussusception, feeding habits/stooling habits

Physical Exam (IPPA): complete physical exam

Vital Signs

General Appearance

Abdominal: distension, tenderness, BS, masses (RUQ mass/sausage = Dance’s sign -> intussusception)

Rectal: r/o anal atresia, currant jelly stool -> intussusception, empty ampulla

Labs: CBC, Basic, LFT’s, amylase/lipase, lactate, U/A, chloride sweat test

Studies

(1) plain AXR

if complete obstruction is demonstrated (no distal air), no further imaging studies are needed

“double bubble” sign = evidence to go to the OR (indicates malrotation, annular pancreas, or duodenal atresia)

“soap bubble” sign of a foamy, meconium-filled bowel in the RLQ -> meconium ileus

(2) upper GI + SBFT: if AXR suggests partial/proximal obstruction (some distal air), get an upper GI study with SBFT

(3) BE: if a distal obstruction appears likely (lots of distended bowel loops), get a contrast enema

(4) a rectal biopsy is necessary for definitive diagnosis of Hirschsprung’s

Special Notes

• intestinal malrotation: sudden onset bilious vomiting in infants < 1 y/o; abd distension with pain; SBFT shows D-J jxn to the right of midline, beak-shaped termination; barium enema shows cecum in RUQ

• duodenal obstruction/ atresia: bilious vomiting at birth, VACTERL, double bubble on AXR

• SBO/jejunileoal atresia: h/o polyhydramnios is common, failure to pass meconium

• intussusception: h/o viral illness is common, knees to chest with abd pain, currant jelly stools, Dance’s sign; BE is definitive

• Hirschsprung’s: < 2 y/o, chronic constipation, small diameter stools, AXR shows a lot of stool in proximal colon; may be complicated by severe enterocolitis, dehydration, peritonitis, and sepsis

• meconium ileus: associated with CF, soap bubble sign on AXR

• meconium plug: associated with prematurity, visualize the plug with BE

• necrotizing enterocolitis: assoc with prematurity/high stress birth (hypoxia, sepsis), bloody stool, sepsis (lethargy, febrile, acidotic, + blood cultures; AXR shows bowel loop dilation, pneumatosis intestinalis, portal venous air, free air if perf’d

Treatment

* Surgical Indications: peritoneal signs/free air/+ DPL suggest intestinal perf (likely NEC); may try a peritoneal drain for 48 hrs

• intestinal malrotation: NGT, IVF, IV Abx, to OR for Ladd’s procedure (CCW volvulus redxn, remove dead bowel, split Ladd’s bands, do appendectomy)

• intestinal obstruction/atresia: NG decompression, IVF, IV Abx (Amp/Gent), to OR for primary anastomosis

• intussusception: 85% reduce with the hydrostatic pressure of an air/barium enema; surgery if it doesn’t reduce

• Hirschsprung’s: resection of involved colon, colostomy with later endorectal pullthrough vs. primary anastomosis; if complicated by enterocolitis/peritonitis, tx with IVF, Abx, and colonic irrigation

• meconium ileus: gastrografin enema is successful in 60% of cases, replace pancreatic enzymes & give IVF; if enema fails, perform an enterotomy

• meconium plug: contrast enema is both diagnostic and therapeutic

• NEC: most recover with medical mgmt (NPO, IVF, NGT, Abx, bicarb to correct acidosis, TPN, ± vent); surgery if perf’d, full thickness necrosis, or clinical instability

• small left colon syndrome: contrast enema

* remember that kids with duodenal atresia should be screened for VACTERL deformities

**Topic 6: Shock (= inadequate tissue perfusion)**

Differential Diagnoses/Causes of Shock: each has distinctive features, exam/lab findings, etc.

• hypovolemic: consider hemorrhage, third-spacing, vomiting/diarrhea, inadequate intake, burns, & pancreatitis; also be aware of corticosteroid withdrawal – refractory to fluids/pressors

• septic: hypotension refractory to fluids, caused by infxn (fever/WBC ct); gram (–) more acute than gram (+); may be complicated by DIC (hemolysis 2/2 vessel dmg), ARDS, DKA/HONK, and MOSF

• cardiogenic: intrinsic (MI, CHF, valve fx, arrhythmia) vs. extrinsic (tension PTX, tamponade, increased abd pressure)
neurogenic: hypotension with BRADYCARDIA! 2/2 spinal cord injury or spinal anesthesia

anaphylactic: Type I hypersensitivity reaction, with elevated IgE, marked by wheezing, urticaria, vomiting, abd cramps

Assess ABC’s by noting all 5 vitals (BP, HR, etc.), UOP/I&O’s, mental status and cardiopulm exam – an abnormal exam (rales, JVD, edema, etc.) may indicate cardiac failure/fluid overload & need for urgent intubation

- signs of shock = tachycardia (earliest sign), hypotension – first orthostatic with hypovolemia, poor UOP, tachypnea, MS chg’s (late signs), poor periph perfusion with CFT > 3 sec, cold & clammy/diaphoretic skin

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Resuscitate – fluid boluses (500cc–1L) are always the first step in treating hypotension, with use of pressors as indicated (see below) and intubation if fluid overload results in respiratory distress and/or hypoxia (GOAL: mean BP 65 mmHg)

1. Airway/Breathing/Spine immobilization: place on O2 by nasal canula, bag/mask, or intubate if unstable
   - establishing airway: chin lift, jaw thrust with oral/nasal airway placement, ET intubation, or cricothyroidotomy
   - Glasgow Coma Scale (3-15) – motor 1-6, verbal 1-5, eyes 1-4 (intubate if < 9)

2. Circ: IV access, Foley (after a normal rectal exam in cases of trauma) & run fluids while realizing that you may need pressors, inotropes, and more for non-hypovolemic shock … minimal UOP for adult trauma pt: 50 ml/hr
   - Swan-Ganz catheter/central line in unstable patient or with indeterminable fluid status
   - urgent studies & labs if unstable & hypovolemic shock unlikely
     - EKG, CXR, enzymes, ABG, CBC, Basic, T&C
   - 3 types of fluids: crystalloid, colloid, and blood products – “3 for 1” rule guides crystalloid after blood loss; give colloid if alb < 2, PRBC’s if Hct < 25%, FFP if PTT > 35 or PT > 15, & platelets if count < 50,000

History: #1 question: What happened to the pt recently? Surgery – what kind/complic’s/I&O? Bee sting, transfusion?

#2 question: cardinal symptoms of non-hypovolemic shock: CP radiating to jaw, fevers/chills, LE paralysis or paresthesias, or wheezing/itching/ cramps

- CC/HPI: symptomatic? detail onset, acuity/chronicity, etc.
- PMH: recent infxns, CAD, CHF, HTN, adrenal insufficiency, immunosuppression (DM, post-TXP), bleeding disorders
- PSH: surgery, including I/O & complications; recent central lines
- Meds: corticosteroids, anti-hypertensives, coumadin/heparin (FFP/Vit K/protamine antitoxides), anti-hypertensives (beta-blockers blunt the tachycardic response to shock)
- Allergies: bee stings, foods, penicillin, contrast dyes, blood transfusions
- FH: CAD, bleeding disorders
- SH: smoking (CAD risk factor), EtOH, IVDU
- ROS (head to toe): F/C, CP, diaphoresis, SOB, cough, wheezing, N/V/cramps, D/C, urinary pain/freq/urge, paralysis

Physical Exam: Vital Signs: shock = BP < 90; General Appearance/Mental Status: Neuro – motor/sensory; Skin; HEENT – mucous membranes, JVD; Cardiopulm – R&R, murmurs, JVD, edema, rales, wheezes (anaphylaxis); Abd – pulsatile mass, tenderness; Rectal – sphincter tone; Extr – edema, periph perfusion, neuro

Labs: CBC, Basic, ABG, T&C, coags, cardiac enzymes, blood cultures (sepsis), fibrinogen/D-dimers (DIC is a potential complication of septic shock), lactate (a marker for perfusion status)

Non-invasive Studies: CXR, EKG, echocardiogram (murmur/EKG chg’s), consider CT scan in septic shock of unknown etiology (possible abscess)

Invasive Studies: cardiac cath as indicated

Classifying Hemorrhage
Class I – 750 cc, <15%: anxiety, normal VS
Class II – 750-1500 cc, 15-30%: tachy, tachypnic
Class III – 1500-2000 cc, 30-40%; decr SBP, low UOP, confused
Class IV – >2000 cc, >40%; tachy > 140, tachypnic > 35, lethargic, no UOP

Treatment – serially assess effectiveness of treatment using UOP, BP, HR, mental status, CFT & extremity perfusion

Hypovolemia: stop bleeding, resuscitate with LR 3:1 & PRBC’s if Hct <30; if still unstable after 2L crystalloid and 4-6 U PRBC, go to OR; do not use vasopressors for hypovolemic shock

Sepsis: try to identify the source of infxn (UTI, pneumonia, abscess); give IVF, chg all lines, empiric Abx
   (Vanco/Gent/Flagyl), drain abscesses, use pressors/inotropes if BP doesn’t respond to fluids (phenylephrine, dopamine, norepi), watch for complications (multi-organ failure, DIC)

Cardiogenic: identify the cause & address ASAP (cath lab, pericardiocentesis, emergent surgery), Swan-Ganz catheter, inotropes, pressors, IABP/VAD for MI; give diuretics for CHF

Neurogenic: IVF, phenylephrine

Anaphylaxis: epinephrine + albuterol, Benadryl, hydrocortisone, pressors

**Topic 7: GI Bleeding**

* with any bleeder, the primary treatment is always resuscitation; secondary concern involves localizing the etiology of the bleeding, which could be occurring anywhere from mouth to anus in the GI tract
* upper = above ligament of Treitz, lower = below ligament of Treitz
* 85% stop bleeding spontaneously with < 5% mortality; 25% rebleed with 30% mortality, & 20% require surgery

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Differential Dx (CIINTS)

- **UGIB**: PUD 50%, gastritis 15% (NSAID’s, EtOH, stress), Mallory-Weiss tear 10% (2/2 retching), varices 10% (2/2 cirrhosis), erosive esophagitis 10% (in either immunocompetent or compromised hosts), AVM’s/Dieulafoy’s (vascular erosion)/aorto-enteric fistula/vasculitis 5%
- **LGIB**: diverticulosis 30%, AVM’s 25%, cancer, colitis (infxn, ischemia, XRT, IBD: UC >> CD), hemorrhoids; Meckel’s diverticulum is the most common cause in children < 2 y/o (50% of all incidents)

**Resuscitation (see above on Shock)**: ABC’s: assess VS, UOP & I/O’s, pt’s AMPLE hx, cardiopulm exam, mental status, etc.; protect airway (especially if massive hematemesis), provide O2, establish large-bore 16G IV access x 2 with Foley, NGT, volume resuscitate by the “3 for 1” rule if possible, otherwise 2L wide-open if unstable; transfuse as nec (careful about using H&H) – get T&C 4u, use O-neg if pt exsanguinating; correct coagulopathies & d/c ASA or other anticoag’s (FFP, platelets)

- **Labs**: CBC, coag’s, complete metabolic profile (including LFT’s), FOBT, amylase/lipase, T&C
  - BUN/Cr ratio > 36 in UGIB 2/2 GI resorption of blood and/or prerenal azotemia

- NGT with subsequent lavage:
  - for gastric decompression (therapeutic) & to initiate diagnostic w/u – remember, it’s not 100% sensitive to UGIB’s
  - presence of non-bloody bile in lavage excludes active UGIB
  - coffee-grounds (old blood) vs. bright red blood (BRB) dictates urgency of endoscopy; both get IV PPI’s & Octreotide

**History**: delineate the nature of the pt’s Sx, and elicit risk fx for possible dx’s

- **CC/Presentation**: melena (usually UGIB, but not always active), hematochezia (LGIB in 89% of cases), hematemesis (almost always UGIB), positive FOBT (source may be anywhere in GI tract)
- **HPI**: GIB onset, prior bleeding, acute vs. chronic, context (retching, inebriation), duration/frequency, AAF (meals? all ulcers hurt for hours after a meal, but initially, food generally makes duodenal ulcers feel better, gastric ulcers worse), ASx (either before, during, or after onset, with description: abd pain – PUD/divertic [note that a perf’d ulcer may result in lower quadrant pain], vomiting – MWT)
  - UGIB – painful: PUD, gastritis/esophagitis, tumor; painless – varices, AVM’s
  - LGIB – painful: colitis, hemorrhoids, anal fissure, ischemia; painless – divertic, AVM’s, tumor, hemorrhoids
- **PMH**: diverticulosis, PUD, CAD/CHF, burns, EtOHism – can cause GIB by several etiologies, GERD (“heartburn”), hepatitis, cirrhosis (rule of two-thirds), CA, IBD, hemorrhoids, HIV/DM (or other causes of immunodef)
- **PSH & Ob/Gyn**: aortic grafts (fistulas), polypectomy (perf)
- **Meds/All**: NSAID’s & ASA (#1 cause of UGIB today), anticoag’s (will need FFP/Vit K to reverse warfarin, protamine for hep)
- **FH**: CA, PUD, IBD
- **SH**: smoking, EtOH, IVDU, exposure to radiation, heightened stress
- **HMA**: previous studies of the GI tract, either upper or lower
- **ROS**: F/C, wt/app chg, GI, urinary …
  - UGIB > LGIB: N/V, hematemesis/coffee-grounds, epigastric pain, syncope, melena
  - LGIB > UGIB: diarrhea, tenesmus (ineffective straining), BRBPR or maroon stools

**Physical Exam** – early signs of shock = tachycardia, diaphoresis, late signs = confusion, lethargy

- vital signs
- general appearance: mental status chg’s may = shock, hepatic enceph, intoxication, or hypoglycemia
- skin / stigmata of cirrhosis: jaundice, spider angiomas, gynecomastia, testic atrophy, palmar erythema, caput medusae
- abd exam: note pain out of proportion to exam, localizable tenderness or peritoneal signs, HSM, ascites, masses
- rectal exam: appearance of stool, inspect for hemorrhoids or anal fissures, blood/hemoccult

**Labs**: see above, consider ordering additional labs – serial CBC’s and coag’s

Non-invasive studies: CXR to r/o free air or mediastinal chg’s, AXR for similar purposes, CT if suspicious

**Invasive studies** *(principle: rule out UGIB before attempting to localize presumed LGIB, endoscopy is pref’d)*

- **NG lavage** is #1 for all GIB
  - Coffee-grounds (“old” UGIB): IV PPI, Octreotide, endoscopy next available (AM if nec.)
  - Bright red blood (“current” UGIB): IV PPI, Octreotide, urgent endoscopy
- Endoscopy should be performed if NG lavage suggests UGIB ... potentially Rx via banding, coag, epi, etc. – note that of pts with known varices, 50% are bleeding from another source
- Angiography can also be therapeutic via vasopressin injxn or embolization

Treatment of UGIB – c/s GI as necessary
- Varices: octreotide-somatostatin (85% success!), beta-blockers ± nitrates, endoscopic band ligation pref’d over sclerotherapy; other options: epi/EtOH/thrombin injxn; Sengstaken-Blakemore if bleeding severe (inflate only the gastric balloon – maintain for up to 48-72 hrs); embolization vs. TIPS if endoscopy fails
- PUD (gastric or duodenal; pref is the worst complication) – note that all pts with PUD do not get an endoscopy, only those who bleed or have S/Sx of malignancy; also note that hemorrhage AND perf may indicate two “kissing” ulcers
  - High dose PPI + Amp/Metro/Clarithro (H. pylori)
  - ? octreotide
  - Endoscopic therapy – injxn, thermal, laser – always biopsy
  - Arteriography with infusion of vasopressin or embolization
  - Surgery if perf’d or pharmy, endoscopic, and arteriographic Rx fails -> vagotomy + pyloroplasty or ligation of gastroduodenal a. for duodenal ulcers
- Risk of rebleeding is based on ulcer appearance – active bleeding 90%, visible vessel 50%, adherent clot 30%, oozing without visible vessel < 30%, clean base (minimal)
- Esophagitis/gastritis: PPI, H2-antagonists
- Mallory-Weiss (75% in the stomach): usually stops spontaneously or with water lavage; endoscopic cautery or embolization if active bleeding; do not use the S-B balloon for M-W tears
- Boerhaave’s Syndrome: postemetic esophageal rupture, resulting in PTX, crepitus, CP – surgical repair

Diagnostic studies in LGIB (no indic for barium enema) … selecting a test depends on the rapidity of the bleed:
- If low clinical suspicion for LGI etiology, do an EGD first to r/o UGIB
- If bleeding spontaneously stops -> sigmoid-/colonoscopy (70% success rate, potentially therapeutic)
- Stable but continued bleeding -> colonoscopy after rapid lavage & purge OR bleeding scan (99-Tc tagged RBC’s): detects bleeding rates ≥ 0.1 cc/min
- Unstable -> arteriography (detects bleeding ≥ 0.5 cc/min) – potentially therapeutic (intraarterial vasopressin infusion or embolization
- Exploratory laparotomy – see indications below

* If upper and lower endoscopy fails to localize bleed, it might be in the small intestine: capsule endoscopy vs. push enteroscopy vs. enterolysis vs. bleeding scan vs. angiography vs. Meckel’s 99-Tc scan

Treatment of LGIB – c/s GI as necessary
- Divertic dz: bleeding usually stops spontaneously, but endoscopic Rx (epi injxn) vs. arterial vasopressin or embolization vs. surgery are the other options
- Bleeding polyps/vascular ectasias: laser, electrocoagulation, local epinephrine injxn
- Angiodysplasia: arterial vasopressin, endoscopic therapy (epi), surgery, hormonal Rx (??)
- Exploratory laparotomy with segmental colectomy – see indications below

Surgical indications in either UGIB or LGIB
- > 6 u PRBC transfusions (in 24 hrs)
- > 3 u PRBC’s to stabilize pt’s hemodynamics
- significant rebleed, especially if the site is known – if it’s not, you might end up doing a total colectomy with ileorectostomy (primary Anastomosis)
- cancer, obstruction, perforation – can Graham patch with omentum, ischemia

Further notes on surgical therapies
- Vagotomy options: truncal with pyloroplasty/antrectomy/gastrojejunostomy, selective (preserves vagal fibers to the pylorus)
- While shunting to alleviate varices decreases portal pressure, it increases encephalopathy

**Topic 8: Oliguria/Anuria**
- urine output is the single most significant and valuable tool in assessing volume status
- anuria = UOP < 100 cc/24 hr; oliguria = UOP 100-400 cc/24 hr or < 0.5 cc/kg/hr in an adult or < 1 cc/kg/hr in a child < 10 kg (would like to see UOP 30-70 cc/hr in ICU pts)
- ARF usually presents as oliguria or rise in serum Cr (2X over baseline = 50% decr in GFR)

Diff Dx/Overview: postrenal, prerenal, or renal causes; resuscitate – keep it simple: fluids first, unless overloaded (resuscitation may be diagnostic)
- always consider postop hemorrhage as the #1 cause of deq UOP or hemodynamic instability
1. **check for** (rule-out) ESRD/dialysis by examining pt’s history
2. **examine for** urinary obstrx by irrigating Foley (*pt MUST have a Foley*)
3. prerenal causes are most common post-op (50-90%): hypovolemia (bleeding or 3rd-spacing), poor CO, renal vasoconstrxn (ACEI’s, NSAID’s), lg vessel dz (RAS), hypotension – get U/S to check renal blood flow, kidney size & symmetry
4. if no obstrx, pt doesn’t respond to fluids & no obvious cause of prerenal failure, think of renal parenchymal dz – (a) ATN (ischemia; nephrotoxins – drugs like AG’s, Mgb/Hgb, etc.; contrast), (b) AIN (allergy – NSAID’s, beta-lactams, sulfas; infxn – pyelonephritis; infiltrative – sarcoidosis, lymphoma), (c) renovascular small vessel dz (HUS/TTP, DIC, preeclampsia, HTNsive crisis), and (d) glomerulonephritis

**Resuscitation:** assess the pt’s ABC’s by noting all 5 vitals, UOP/I&O’s, mental status, resp distress, complaints, etc., and resuscitate as necessary; if the pt doesn’t have a Foley and/or IV access, establish it
- **place or replace the Foley/flush with 30cc; then bolus 500cc LR absent signs of fluid overload/CHF**
- address hypoxia/respiratory distress with O2/intubation
- if the pt is also febrile, consider the 6 W’s (wind – atelectasis: encourage C/DB/IS; water – UTI; etc.)

**History**
- HPI: is the pt symptomatic? if so, inquire further in a ROS (F/C, dizzy/HA, CP/SOB, N/V/D/C, urinary)
- PMH: kidney dz/dialysis, BPH, neurogenic bladder, recent procedures involving *contrast exposure*, hemolysis, liver disease (hepatorenal syndrome – “looks” like prerenal fx)
- PSH: abd surgery, review op report, noting any significant hypotension and I/O’s
- Meds: analgesics, including spinals/epidurals; antibiotics; diuretics

**PE (IPPA):** vitals, gen appearance, HEENT, cardiopulm – rubs/rales, abd – ascites/compt syndrome, rectal – BPH, extr/vasc – assess for hyper/eu/hypovolemia (JVP, mucus membranes, rales, hypoxia) as well as for hemorrhage

**Labs:** no need to order labs immediately
- if pt doesn’t respond to one 500cc bolus, get a CBC, coags, and T&C
- if pt’s mental status is altered or the pt doesn’t respond to fluids, check a basic & U/A
- 5 labs to assess prerenal vs. renal etiologies: BUN/Cr ratio >/< 20, FeNa >/< 1, spec grav >/< 1.02, Una < 20/> 40, UOsm >500/< 350
- assessing true renal fx (ATN, AIN, GN, or RV): urine sed, CBC, LDH/haptoglobin, CPK, etc.

**Non-invasive Studies:** bladder U/S (PVR), prostate U/S (BPH), renal vessel U/S to ensure good blood flow, EKG

**Invasive Studies:** if a pt appears fluid overloaded in setting of oliguria, a Swan is beneficial
- bolus 500cc LR absence signs of fluid overload/CHF & another 500cc with CBC if pt doesn’t respond – serial exams for fluid overload (rales, hypoxia, JVD) – improving renal perf should alleviate prerenal fx
- if pt responds to bolus, incr IVF (D5 1/2 NS, 20 mEq KCl) to 2-3X nl maint [4/2/1 ml/hr based on kg’s]
- if the pt’s Hct < 8 (nl adult) or < 10 (elderly, compromised), give 2u PRBC’s & monitor CBC
- if pt doesn’t respond or is fluid overloaded, move to ICU, consider central line/Swan, A-line, etc. & r/o MI
- try albumin if the patient doesn’t respond after several boluses
- if you suspect true renal fx, try a Lasix challenge (100-150 mg) – a polyuric response is a good prognostic sign: in this case, continue the Lasix with intermittent hemodialysis; if oliguria continues, the pt will likely need chronic dialysis
- d/c nephrotoxic medications (Vanco, Gent, Cyclosporine, etc.), check serial levels, & diurese/dialyze with incr fluids to clear toxins
- provide extra nutrition to patients with true renal fx – low Na, low K, low protein
- consider dialysis for any pt who develops AEIOU reasons for dialysis (academia, hyper-K, fluid overload, uremia – pericarditis, encephalopathy, bleeding) … it’s preferable to dialyze before uremia (symptoms) ensues, while they’re still just azotemic (incr BUN) – numerous options for dialysis exist (hemo, peritoneal, CVVH, CAVH, etc.)

**Surgical Indic’s:** unstable VS, unstable Hct, signs of hemorrhage – return to OR for expl, given likely hemorrhage

**Topic 9: Burns**

**Overview:** stop the burning process, resuscitate while conducting primary and secondary surveys, initiate further treatment, and manage the burns while monitoring for potential complications (scarring/infection)

**Resuscitation: primary survey**
- stop the burning process
- **airway/breathing:** look for indications of smoke inhalation – facial burns, singed nasal hairs, etc. – and inquire into whether the burn occurred in a closed space
  - low threshold for intubation before oropharyngeal swelling occludes the airway, or trach
  - administer 100% O2 until carboxyhemoglobin can be ruled out
- circulation & fluid resuscitation
  - crystalloid via large-bore peripheral IV’s using Parkland Formula (TBSA% x wt x 2-4 for shallow-deep burns), giving half in first 8 hrs from time of injury and remainder over the next 16 hrs – that’s ~ 400 cc/hr for first 8 hrs in a 70 kg pt with 20% 3rd burns
  - monitor volume status: BP, HR, peripheral perfusion, mental status & UOP (Foley a must!)
  - Foley minimal UOP > 0.5 cc/kg/hr; consider Swan/cataline line if burns are >30%, patient is elderly, unstable, or suffered severe inhalation injury
- assess disability, make environment warm & expose/examine entire body surface
- other stabilization issues to include in primary survey
  - labs: CBC, Chem7 (follow Na closely), coags, ABG (pulse ox inaccurate with Chg), carboxyhemoglobin, urine Mgb, T&C • carboxyhemoglobin > 60%: 50% mortality; treat with 100% O2 ± hyperbaric O2 and time • myoglobinuria warrants aggressive diuresis with mannitol and alkalization with IV bicarb • tetanus toxoid/dig is mandatory in all pts except those immunized within 12 mos b/c of high susceptibility of devitalized tissue to tetanus infection • NGT to decompress stomach if > 20% TBSA burned, given likelihood of paralytic ileus and aspiration pneumonia • Watch out for acidosis, dysrhythmia, and coagulopathies, especially if pt. is cold (EKG)

Resuscitation: secondary survey
- assess what type of burn, how deep, and how extensive it is
  - type: electrical, acid/alkali (more serious), fire, sun, etc.
  - depth: 1st – no blisters (painful, red, dry); 2nd – blisters, openly weeping; 3rd – “full thickness” including dermis, painless, swollen, dry, mottled white/charred areas like “dry leathers”; 4th – burn injury into bone/muscle
  - TBSA: rule of 9’s for amt of surface area affected by second/third degree burns (modified slightly for kids, whose heads are disproportionately larger); palms ~ 1%
- history: mech of injury; closed space exposure (elevates risk of smoke inhalation); AMPLE hx – extrication time, delay seeking treatment, fluids received, previous illnesses, associated trauma
- physical exam: especially examine for signs of smoke inhalation – smoke/soot in sputum/mouth/nose, nasal/facial hair burns, confusion with low O2 sat; fluorescein for 3rd
- studies: radiographs, depending on mech of injury and need to document central line placement

Further Treatment/Referral
- enteral nutrition: hypercatabolism/protein wasting are severe after major burns, so start enteral nutrition in the first 24 hours and advance as tolerated
- fluids: D5W IV beginning 24 hrs post-burn, with 5% albumin at 0.5 cc/kg/%) TBSA over the next 6 hours (after the capillaries “re-seal”); glucose is CI’d for first 24 hrs b/c of stress response
- prophylaxis: H2 blocker to prevent development of exudant’s ulcer (high cortisol)
- transfer to burn center for 2nd burns > 20% TBSA, 3rd burns > 5% TBSA, or any burns involving face/hands/feet/perineum/inhalation injury/trauam/electricity

Burn Management (basic principles: cleansing, debridement, topical antibiotic ointment, sterile dressing)
- first degree: keep clean with Neosporin, pain meds
- second degree: remove blisters, Silvadene – no lyte imbalances/little eschar penetration/may cause neutropenia (need CBC’s)/misses Pseudomonas/best for small burns, pain meds; most 2nd burns don’t require skin grafting (other antibiotic cream options: sulfamylon – broad spectrum except Staph/penetrates eschar so good for 3rd burns already contaminated/causes pain, metabolic acidosis & allergic rxn’s, polysporin – best for facial burns/narrow spectrum)
- third degree: early excision of eschar within 1st week post-burn (tourniquets/epi/thrombin decr bleeding), and split-thickness skin grafting; watch for compartment syndrome (> 30 mmHg), especially when involvement is circumferential (particularly chest/abd – measure press with Foley/esophagoscope), and perform escharotomy (full-thickness longitudinal incision down to the fat) if necessary

Burn Complications: scarring & infections
  1. first PT minimizes complications of burn scar contract (immobilization of ext, disfig)
  2. monitor for infxn (fever, WBC, eschar discoloration, deepening wound) – most common bugs are Staph, Pseudomonas, Strep, and Candida – tx with IV Abx if Ctx positive (> 10^9 bacteria/g of tissue); operative debridement removes the eschar, which is the nidus for bacterial growth, and improves outcomes (scalpel/electrocautery debridement to fascia/fat)
  3. monitor for pneumonia (Staph aureus, Pseudomonas), UTI’s, and central line infections and tx with IV Abx – chg lines every 3/4 days, signs/sx of a central line infxn include unexplained hyperglycemia, fever, MS chg, hypotension/tachycardia, pus, and erythema at the site; if no obvious signs of infxn, chg the line over a wire and culture the tip (if > 15 CFU, remove the line and place at a different site)

Topic 10: Cool, Pulseless Foot
- THROMBOSIS vs. EMBOLI (80% of ALI, usually AFib, mural thrombus post-MI, valve veg, or aneurysm)

Resusc: assess the pt’s stability by asking for resp distress, all 5 VS, UOP and managing ABC with O2/intub, IV access, Foley, and placing the pt on monitors (incl EKG)

Diff Dx: thromboembolic dz, compartment syndrome, trauma, aortic dissection, arterial or venous spasm

History – if this is 2/2 emboli, you want to identify the source of the emboli, then Rx appropriately
• HPI: time/context/severity of onset are crucial, as are the presence of the 6P’s – neurovascular compromise (3 of each: poikilothermia, pulseless, pale, paresthesias, paralysis, pain); distinguish rest pain vs. claudication, progressive vs. alleviated, prior hx of these Sx – tx?; always ask about OTHER Sx
  ○ assess pt activity at the time of onset, as well as the precise level of NV sx
  ○ associated swelling? think about compartment syndrome more strongly
• PMH: recent injury/surgery to the foot or leg, CAD, HTN, DM, high chol, aneurysms, PVOD, known arrhythmias, prosthetic valve, rheumatic heart disease, Marfan’s/ED, GIB (CI to heparin)
• PSH, Meds/All (Coumadin, ASA, BCP’s, HRT), FH (vascular disease, arrhythmias), SH (smoking, EtOH, IVDU), HMA (EKG, previous AB I’s or vessel scans)
• ROS: F/C; headache, visual Sx; SOB, CP; N/V/D/C, abd pain; urinary chg’s, erectile dysfxn

Physical Exam (IP)
• Vital signs – all 5
• General appearance
• Cardiac exam: note irregular rhythm/bruits/murmurs/rubs
• Abdominal exam: listen for bruit, palpate for AAA
• LE Vasc/Neuro Exam – comparison to the other extr will provide clues as to thrombotic vs. embolic origin
  ○ inspect for wounds (possible infxn)/burns/constricting eschars, S/Sx of PVOD (hair loss, ulcers, thick nails, thin/discolored skin, gangrene – wet/dry?), blue toes (peripheral emboli)
  ○ document the 6P’s & assess limb viability by doing a neuro exam (sensation/motor) and checking pulses THROUGHOUT THE BODY & from femoral on down/color to determine level of occlusion (use Doppler if nec) – iliac (mid-thigh), femoral (mid-calf), popliteal (mid-ankle)

Non-inv Studies: Doppler U/S to det lxn/severity of occl, EKG ± echo (MI, AF, clot, valve veg), ? CT AAA scan

Invasive Studies: arteriogram is used preoperatively for mapping purposes – also, it’s ALWAYS done in the case of dislocation of knees/elbows, with or without symptoms

Treatment
Immediate Mgmt:
• IV heparin bolus followed by infusion, except if CI’s exist (GIB, new neuro deficit, head injury, active bleeding) (NOTE: OR/angio are NOT CI’s to heparin)
• aspirin
• fluid resuscitation & correction of metabolic acidosis
• Dextran, Mannitol (oncotic load decr thrombus propagation)
• Maintain extremity in a dependent position & keep pt warm, with analgesia prn

Surgical Indications: no improvement on medical therapy within 1-3 hrs after onset

Embolism (CFA, usually) – surgical embolectomy via cutdown and Fogarty balloon (tPA is optional as adjuvant)
• transition to Coumadin if the source of the emboli is unknown or persists (e.g., a-fib)
• consider aortic endarterectomy if that happens to be the source of the emboli

Thrombosis (SFA, usually) – if limb-threatening (i.e., acute onset with ominous exam findings), thrombectomy; otherwise bypass vs. thrombolytic therapy (pref’d for poor operative candidates) vs. amputation

Compartment s.: > 35 mmHg; may occur 2/2 arterial or crush injury, fracture, or reperfusion – decr sensation, pain exacerbated by passive DF, weakness of involved mm., swelling but pulse usu. still present – Tx: fasciectomy after measuring the compartment pressure

** Monitor post-reperfusion complications: rhabdomyolysis/renal failure, hyperkalemia (from dmg’d cells), lactic acidosis, MI, & compartment s., the first 3 of which can be minimized with adequate hydration

** Topic 11: Deep Venous Thrombosis (DVT)
• pts don’t need admission for tx of uncomplicated DVT’s: Lovenox bridge to Coumadin is OK
• think in terms of Virchow’s triad – find sources for hypercoag, stasis, or endothelial dmg
• consider measures of thromboprophylaxis: early ambulation, SCD’s/compression hose, SQ heparin

Resuscitation: assess the patient’s vital signs, UOP, and CP exam; resuscitate as necessary

Diff Dx of LE edema/leg pain: DVT, compartment syndrome, chronic venous insufficiency, cellulitis, CHF, trauma, lymphangitis, renal failure
Hx (complete Hx, but note Well’s criteria for DVT – major criteria are in bold type, minor criteria in italics)
- HPI
- PMH/PSH (Virchow’s triad): prior DVT, cancer, coagulopathy (Factor V Leiden is most common), recent immob/paralysis, hospitalization in past 6 mos., pregnancy (mechanical compression), trauma in past 2 mos.
- Meds/All: birth control pills, HRT, Coumadin, ASA
- FH: DVT in ≥ 2 1st relatives, PE
- SH: smoking, EtOH, IVDU, occupational hx requiring prolonged standing/immobilization
- ROS: F/C; SOB, pleuritic CP, cough, hemoptysis

Phys Exam
- vital signs
- general appearance
- cardiopulmonary exam: tachypnea, pleural friction rub, tachycardia, loud P2, S3, JVD
- extremities: neurovascular status, calf edema 3 cm larger circ than ASx side, pitting edema of thigh AND calf, erythema, warmth, localized tenderness along veins, palpable cord, dilated superficial veins, Homan’s sign on DF, color chg’s (white – phlegmasia alba dolens, cyanotic – phlegmasia cerulea dolens)

Well’s criteria for PE: S/Sx of PE (3), S/Sx of DVT (3), HR > 100 (1.5), recent immob/surg (1.5), prior DVT/PE or FH (1.5), hemoptysis (1), malig (1)

Using Well’s Criteria
DVT: high prob = ≥ 3 major + no alternative dx or ≥ 2 major + ≥ 2 minor + no alternative dx (85%)
PE: high prob = > 6 points, low prob = 0-2 points (so if a pt has a DVT, get a helical CT, compression U/S)
- low prob: D-dimers
- intermed OR high prob: helical CT & compr U/S (if neg but suspicion remains, do a V/Q scan or angiography)

Labs & Non-Invasive Studies
- suspected DVT: coags, duplex U/S
- PE: coags, ABG (decr pO2 AND pCO2 from hypervent, incr Aa gradient), EKG (STD, TWI, RAD), CXR (Westermark’s)

Invasive Studies if prelim studies negative but high suspicion: venography (DVT), angiography (PE gold std.)

Treatment
- anticoag: IV unfractionated heparin to PTT 50-70 OR Lovenox (outpatient) followed by Coumadin (INR 2.3) when therapeutic or on Day 1 of Lovenox (expect sx of DVT to resolve over 3-6 mos.)
- tPA: be aggressive in cases of extensive DVT or PE causing hemodynamic compromise (low reserve)
- thrombectomy may be beneficial in cases of limb-threatening ischemia or PE
- Greenfield filter: if anticoag is CI’d (GIB, recent stroke, cerebral AVM, hemophilia) or refractory

Long-term Anticoag: 1st event – 3-6 mos warfarin, if idiopathic – 5 yrs warfarin; 2nd event or active cause – 12 mos to lifelong; after withdrawal of anticoagulation, pts should have a D-dimer and an U/S done

Complications: post-thrombotic syndrome (CVI) with venous HTN, pain, swelling, color chg’s, and ulcs – Rx: stockings

Further Workup: thrombophilia workup if recurrent/idiopathic DVT/PE; malignancy workup (12% of pts with idiopathic DVT/PE; this workup starts with a complete H&P for symptoms and signs of malignancy)

Topic 12: TIA’s
- TIA: sudden neuro def, resolving within 24 hrs (usu. 1 hr), suggests impending CVA

Resuscitation: assess whether the pt is experiencing any symptoms currently, ABC’s as necessary

Differential Dx (CIINTS / VINDICATE)
- TIA’s: low flow (cardiac arrest, large vessel stenosis) vs. emboli (A-fib, LV thrombus) vs. small vessel dz (HTN)
- hypoglycemia, hypoxia (cardiopulmonary dz), hyponatremia, seizures, migraine auras, syncope, encephalopathy, brain tumors, trauma, vasculitis, amaurosis fugax, CVA
History: utilize to elicit risk factors for CVD
- HPI: onset – acuity, time of day (wake up?); context; freq/duration; progression vs. stability; AA fx – head positions; ASx – amaurosis fugax (curtain), mentation chg’s, vision chg’s, weakness/paralysis or N/T (sensorimotor), speech disturbances, loss of consciousness, dizziness, dysphagia
- PMH: HTN, hyperlip, DM, MI, valve disease, arrhythmias; neurologic disease (prior TIA’s/RIND’s/CVA’s)
- PSH: carotid surgery, CABG, others
- Meds/All: Coumadin, ASA, HRT, BCP’s
- FH: strokes/TIA’s, heart disease
- SH: smoking, EtOH, IVDU
- HMA: cholesterol, BP
- ROS: Gen – F/C (infxn); Neuro – see above; CV – SOB, CP; GI – abd pain

Physical Exam: Vital Signs / Gen Appearance / Complete Neuro Exam (MS, CN, motor, reflex, sensory) – may need to repeat serially / Cardiovascular Exam / Ophthy Exam (Hollenhorst plaques @ branch points, indicating cholesterol emboli)

Labs: CBC, Chem 7 (r/o metabolic causes), coags, LFT’s, tox screen & U/A

Non-invasive Studies:
- EKG: look for A-Fib or recent MI
- CT/MRI is indicated in all patients with suspected TIA
- Carotid duplex ± transcranial Doppler
- TTE before TEE, and TEE only if the TTE is negative (TTE will identify most valve dz)

Invasive Studies: consider angiography only when Dx is uncertain by non-invasive methods and proof of Dx is essential for proper stroke prevention

Treatment: to prevent a stroke (risk: 1/3 if h/o TIA; 1/3 ASx, 1/3 more TIA’s) …
- antiplatelet therapy (ASA, clopidogrel, or ASA + dipyridamole), and …
- anticoagulation (heparin, transition to warfarin), only if the source is known/presumed to be cardioembolic or large vessel atherosclerosis

Indications for CEA ± patch angioplasty (for b/l dz, take care of the dominant hemisphere, first)
- symptomatic and stenosis >70% – proven benefit, 2-yr ARR of stroke 35 to 17% vs. medical mgmt. (NASCET) … all pts go on aspirin after a CEA
- symptomatic with stenosis >30% is acceptable but no mortality benefit
- asymptomatic but with stenosis > 60% is an indication for surgery if LE > 5 yrs & surgical risk < 3% (ACAS)

Complications: CVA, MI, hematoma, infxn, hemorrhage, hypo-/hypertension, X/XII n. injury

NOTE: treatment for a CVA includes thrombolysis if onset is within 3 hrs and ASA, but anticoag only if the source is known/presumed to be cardioembolic

Topic 13: Weight Loss (typically colon or pancreatic cancer for oral exams)
* seek to elicit additional Sx with history-taking, as well as risk fx’s that point you in the direction of the cause

Resuscitation: utilize whatever information you have to assess ABC’s

Differential Dx: cancer, polyps alone, achalasia, gastric ulcer, chronic mesent ischemia, IBD (Crohn’s/UC), TB/HIV, pheochrom, hyperthyroidism, DM, Hodgkin’s lymphoma, soft tissue sarcoma, carcinoid syndrome
- brief notes on Tx – CMI: bypass/endarterectomy, IBD: surgery if complications arise, pheo: adrenalectomy, hyperthyroidism: medical PTU vs. RAIA/surgical mgmt

History: Most Impt Questions: #1 Voluntary or Invol? #2 Incr or Decr Appetite? #3 % Chg from Baseline #4 Lifetime Variability
CC: what symptoms is the pt having?
HPI: how much/soon, meals, lifestyle chg’s, depression
PMH: previous CA, Crohn’s/UC, polyps (villous are worst); DM; MI/angina (risk fx for mesent isch)
PSH
Meds/All
FH (up to 25% of CRC’s have FH): CA’s, IBD, polyps (FAP/Gardner’s/P-J/HNPCC), heart dz, DM, vascular dz
SH: smoking, EtOH, occupational exposures, radiation exposure, travel, diet (low fiber, high fat?)
HMA: annual rectal exams & FOBT (at age 40), colonoscopy/flex sig/BE (at age 50 and 3-10 yrs thereafter, unless FH)
  ○ However, Guiue is only 10% specific to colorectal CA
ROS …
  ○ Gen: F/C, fatigue/appetite chg
  ○ Pulm/Cardiac: SOB, CP
  ○ GI: appetite/anorexia, dysphagia, N/V, regurg, early satiety, D/C, chg’s in BM’s – clues R vs. L vs. rectal (CRC is #1 cause of obstrxn in adults), bloody emesis/stool, tenesmus, abdominal pain, jaundice/pruritis
  ○ GU: chg’s in urination
  ○ Endoc: heat intolerance, palpitations, restlessness, polydipsia, polyuria, polyphagia

Physical Exam: “complete physical exam” (IPPA) focusing on …
  ● Vital signs
  ● General appearance
  ● HEENT: proptosis, lymph nodes, neck bruits, thyroid nodules/enlargement
  ● Cardiopulm
  ● Abd: tenderness (including RUQ – liver mets), masses, distension (r/o obstrxn), ascites, umbilical lymphadenopathy
  ● Rectal (always with GI-type Sx):
    ○ occult blood, masses (10% palpable), gross blood, Blummer’s shelf
  ● Extr

Labs: CBC (Fe-defic microcytic anemia is common, especially with R-sided tumors), Chem10, LFT’s to stage CRC, TSH/free T4 pm, 24-hr urine catechol’s & byproducts (VMA/HMA) pm, U/A pm, albumin to check nutrition, amylase/lipase, lactate pm, Ctx if septic – Strep bovis bacteremia goes along with CRC, as does Clostridium septicum sepsis
  - after making a diagnosis of CA, you’ll need to run thru this order of business again & draw more labs (e.g., CA 19-9/CEA for pancreatic CA, CEA for colorectal CA, etc.)

Non-invasive Studies: CXR/abd plainfilms, endorectal U/S (best test to assess depth of invasion & LN status), CT (chest/abdomen, head if Sx)

Invasive Studies: upper GI/BE, flex sig before colonoscopy, ERCP – biopsy where appropriate, and conduct mets workup if (+)

Staging: CA spreads directly, lymphatically, hematogenously, and by “seeding”
  • see painless jaundice (next page) for staging of pancreatic CA

  • Duke’s staging for CRC (basics): A – ≤ submucosa, B – beyond submucosa with (–) LN’s, C – (+) LN’s, D – dist mets
  ● CT is inaccurate for assessing the depth of invasion and malignant lymph nodes
  ● Intraoperative staging is essential for evaluating extracolonic spread

Treatment
Resuscitation: take into account what you now know about the patient’s ABC’s

Stage I: surgery alone ± pre-op XRT to shrink tumor (resect with 5 cm distal, 7 cm proximal margins – APR vs. LAR for rectosigmoid CA, colectomy taking the involved vasculature/lymph nodes for colon CA – SMA: ileocolic, right colic, middle colic; IMA: left colic, sigmoid, superior hemorrhoidal; internal iliac: middle/inferior hemorrhoidal)
  • bowel prep: Golytely or Fleets enema, PO antibiotics (cefotetan) – allows primary anastomosis to be made
Stage II: surgery (no established role for chemo)
Stage III: surgery + 5-FU/Leucovorin or Levamisole (50% survival)
Stage IV: chemotherapy (5-FU/Leucovorin/Irinotecan) ± palliative surgery (5% survival); may resect solitary liver mets
  • Followup: CEA levels q 3 mos. to follow response to therapy and detect recurrence; physical exam, stool guiac

Topic 14: Painless Jaundice * hyperbilirubinemia > 2.5 with yellowing of the skin

Diff Dx (CHINTS) of Jaundice – painless jaundice in bold lettering
  ● prehepatic: hemolysis (sickle-cell, G6PDD …), blood transfusion
  ● hepatic: hepatitis, cirrhosis, drug toxicity, sepsis, congenital conjugation defects
  ● posthepatic: gallstones, cholangitis, biliary duct injury, pancreatitis, sclerosing cholangitis, tumors (pancr, cholangio, gallbladder adeno, mets), pancreatic pseudocyst

Resuscitation: assess the patient’s ABC’s using available information (VS, UOP, etc.)
be aware that jaundice, fever, and RUQ pain is Charcot’s triad of cholangitis & requires immediate attention before the patient progresses to mental status chg’s & septic shock (Reynold’s pentad)

History – cover all your bases (full 10-part history); don’t be presumptuous, but focus appropriately
- HPI: onset, duration, location, and progression of jaundice; dark urine, light stools, pruritis
- PMH: hepatitis (etiolo?y?), cirrhosis, PBC/SC, UC, gallstones, acute/chronic pancreatitis, DM (including recent-onset), transf
- PSH: cholecystectomy, abdominal surgery
- Meds/All: Tylenol, thorotrast contrast dye
- FH: CA, autoimmune hepatitis, PBC/SC, hereditary hemolytic states
- SH: smoking, EtOH, IVCT, sexual history
- HMA: screening FOBT, other tests
- ROS (think head-to-toe): Gen – F/C, fatigue/wt loss/app chg; GI – abd pain (radiating to back?), N/V, diarrhea, wt loss: Extr – migratory pain (thrombophlebitis)

Physical Exam (IPPA): vital signs, gen appearance (obesity), skin (spider telang, jaundice); HEENT (sublingual, scleral, cutaneous jaundice); chest (gynecomastia); abdomen (ascites, caput meduseae, HSM, Courvoisier’s sign (palpable/nontender GB), distension, tenderness); rectal; genitals (testicular atrophy); extremities (palmar erythema, muscle wasting)

Labs: CBC, Chem 10, LFT’s with bili & alk phos, coags, U/A
- hemolysis: unconj >> conj bili; decr haptoglobin, Hct; incr LDH, retic’s; smear: frag RBC’s
- hepatitis: serologies (Ag’s, Ab’s)
- drug toxicity: tox screens for acetaminophen …
- tumors: CA 19-9, CEA (both apply to pancreatic CA)

Non-invasive Studies: U/S #1, pancreatic protocol CT with contrast if suspicious of pancreatic CA, CXR for mets
- double duct sign: pancreatic CA

Invasive Studies: ERCP/PTC with biopsy/brushings for cytology

Staging CA’s causing Painless Obstructive Jaundice – assess depth of invasion/extent of spread
- pancreatic CA (endoscopic U/S is the most accurate local staging modality): I-III; I = no extrapancreatic disease, resectable; II = locally advanced into the vasculature (venous occlusion, celiac axis/SMA) but no distant mets; III = mets typically to liver, peritoneum, or lung
- cholangiocarcinoma (diagnosis is suggested by a bile duct stricture on ERCP); gallbladder CA

Treatment (medical vs. surgical causes of jaundice!)
Pancreatic CA – depends on lxn of the tumor & resectability, which may need to be assessed laparoscopically (inoperable if vasc encasement, peritoneal/distant mets) … nodal status is the most important prognostic factor
- head of pancreas (typically painless): Whipple (removal of GB, CBD, majority of duodenum, 15 cm jejunum, head of pancreas, ± antrectomy with truncal vagotomy – choledochojejunostomy, panc-jej, gastro-jej)
- body or tail (typically with abd pain radiating to the back): distal resection
- palliative treatment: stent placement via PTC/ERCP or biliary bypass for biliary obstrxn, gastrojejunostomy for gastric outlet obstruction, pancreatic enzyme replacement for malabsorption, pain relief
- pre- and/or post-op adjuvant therapy: chemotherapy (5-FU or gemcitabine) and XRT

Cholangiocarcinoma – resection with Roux-en-Y hepaticojejunostomy for proximal tumors, Whipple for distal

Gallbladder CA – think about it if the gallbladder is calcified (“porcelain”); cholecystectomy may be sufficient, or wedge resection of overlying liver may be necessary with LN dissection (1/100 people with gallstones get CA) – this needs to be done OPEN to avoid seeding the trocar sites

Topic 15: Lung CA
- 70% of solitary pulmonary nodules are benign (mostly granulomas) and 30% malignant; what you worry about are those with associated signs and symptoms; lung CA – leading CA killer
- look for symptoms in each of 3 categories: local effects, systemic effects, and 5 major types of PN syndromes
- of the non-SCLC’s, there are adeno, large cell, and squamous cell (the S’s grow centrally, adeno/large cell peripherally)

Resuscitation: evaluate ABC’s/vital signs for resuscitation needs

Diff Dx (CHN): benign (granulomas, hamartomas) vs. malig (75% bronchogenic CA, 20% mets, sarcoma, lymphoma – assoc with Sjögren’s)
Note the pt’s age as one of the important risk factors

CC: 10% asymptomatic, chg in cough, hemoptysis, dyspnea, hoarseness, dysphagia, etc.

HP: O/C/Qu/S/D/L/R/acute vs. chronic/stable vs. progressive/AA fx/ASx

PMH/PSH: prior CA, XRT, prior TB infection, rheumatoid arthritis

Meds/All:

FH: CA

SH: smoking (although adenoma is the most common in non-smokers – in fact, bronchoalveolar adenomaCA may arise from a virus, jazziekte), drinking, drugs, asbestos, radon, XRT exposure

HMA: prior CXR’s/imaging for comparison

ROS: F/C, fatigue/weakness/wt loss/app chg; Pulm: cough, hemoptysis, dyspnea, wheezing; headaches, facial swelling (SVC syndrome); Pancoast’s – Horner’s, brachial plexus involvement, bone pain (PTH)

Physical Exam (IPPA)

- Vital signs, Ht & Wt with BMI
- Gen appearance: cachectic?
- Skin: acanthosis nigricans (a paraneoplastic syndrome)
- Neuro: weakness (Eaton-Lambert: small cell, Pancoast’s)
- HEENT: swelling (SVC syndrome), lymphadenopathy
- Cardiopulmonary: IPPA
- Abd
- Ext/Musc: clubbing (non-Ssmall cell), periosteal proliferation (HPO: adenoma), atrophy (Pancoast’s), bone pain (PTH)

Labs: TB test, CBC, Chem7, Ca/Mg/PO4, coags – be aware of tipsoffs to paraneoplastic syndromes (ACTH, PTH, ADH, hypercoag: adenoma, DIC, marantic endocarditis)

- small cell: hyponatremia (SIADH), Cushing’s (ACTH), Eaton-Lambert
- squamous cell: PTHrP
- carcinoid tumors secrete serotonin and produce bronchospasm, flushing, diarrhea, and right-sided heart failure

Non-Invasive Studies

- no proven benefit to screening CXR or sputum cytology but they can be used
  - If you have a new SPN on CXR, seek previous X-rays; if unchanged over 2 years, just follow …
  - CXR may show pleural effusion
  - solitary pulm nodule --> CT scan; > 2.3 cm, ill-defined borders, spiculated edges, age > 60, + smoking are suggestive of CA;
  - hamartomas have a “popcorn” pattern; granulomas “laminated”; chest CT must include liver & adrenals

Invasive Studies: biopsy/brushings: transbronchial, transthoracic (CT-guided) (tissue is the issue), VATS (for pleural/periph lesions)

Staging (CRUCIAL TO ANY CANCER DIAGNOSIS)

- TNM staging system for NSCLC (T3+ = extrapulm invasion, N0-3); limited (confined to one radiation port) vs. extensive for SCLC
- utilize mediastinoscopy, VATS, or thoracentesis for intrathoracic staging, PET scan or integrated PET-CT for extrathoracic staging, both of which are more sensitive than CT alone (mets: brain, bone, liver, adrenal, skin)
  - direct extension
  - small cell has the greatest predilection for spread to lymph nodes: intrapulmonary, then bronchopulmonary (hilar, then intralobar)
  - hematogenous spread is also common
  - a brain MRI/bone scan is indicated for all SCLC patients and for symptomatic NSCLC pts
- once CA has been confirmed, get LFT’s

Treatment (Contraindications to surgery: STOP IT)

Resuscitation: take into account what you now know about the patient’s ABC’s

Non-cancerous Lesion

- for low-risk patients, serial CT’s are appropriate (q 3 mos x 4, then q 6 x 2)
- intermed to high-risk lesions warrant VATS rexn, with lobectomy if the lesion is found to be malignant – PFT’s showing FEV1 > 2 are required for pneumonectomy, > 1.4 for lobectomy

Non-Small Cell Lung CA (survival ranges <1% - > 60% 5-year for Stage I)

- Stage I & II: surgical resection, ? benefit from chemo
- Stage III: optimal combo of chemo/XRT/surgery unknown
- Stage IV: chemo (carboplatin + paclitaxel) prolongs survival; may consider surgical rexn/XRT for palliation in the case of brain mets

- note surgical options: pneumonectomy, lobectomy, wedge resection, segmentectomy, or bronchoplasty for dz near the carina

SCLC

- chemo platinum plus etoposide, with thoracic radiation
- thoracic radiation improves survival in limited stage disease

**Topic 16: Esophageal CA**

* SCC most common in upper 2/3’s; AC most common nearer the GE jxn; most common in the 6th decade

CC: dysphagia of solids, weight loss, chest pain, back pain, hoarseness, symptoms of metastases
**Resuscitation:** assess vital signs & need for resuscitation; be attuned to potential nutritional needs

**Differential Diagnoses of Dysphagia & the 3 Most Important Questions**
- dysphagia of solids (mechanical problem) or solids/liquids (motility)?
  - mechanical: esophageal ring, peptic stricture, esophageal CA
  - motility: spasm, scleroderma, achalasia
- intermittent or progressively worse? the first in each group is intermittent/chronic
- chronic GERD or not? the second in each group manifests chronic GERD
  * other causes of dysphagia: GERD, mediastinal tumors (leiomyoma, lymphoma, mets), diabetic gastroparesis

**History**
- FOCUS ON RISK Fx’s & Sx’s (local, systemic, mets) to RULE-IN CA & R/O DIFFERENTIALS
- HPI: O/acute vs. chronic/stable vs. progressive/S/AA fx/ASx – pain, vomiting
- PMH: GERD, prior CA, DM, premalig lesions – achalasia, hiatal hernia, radiation, Plummer-Vinson
- PSH: previous gastric surgery
- Meds/All
- FH: DM, CA
- SH: smoking, EtOH; ingestion of hot foods/bev’s, lye exposure – soap prodxn; ADL’s
- HMA: esophagoscopy? (Barrett’s? metaplasia)
- ROS: F/C, fatigue/wt loss/app chg, SOB/cough/hoarseness, CP, abd pain/N/V ± blood/D/C, chg’s in urination, back pain, bone pain

**Physical Exam (IPPA):** goal is to identify signs of presence of tumor/mets, or differentials
- Vital signs, Ht & Wt with BMI
- Gen appearance: cachectic?
- HEENT: palpable masses, nodules, or lymph nodes
- Cardiopulm / Abd / Extremities

**Labs:** CBC, Chem7, LFT’s, coags

**Non-invasive Studies:** CXR

**Invasive Studies:** UGI barium swallow, EGD with biopsy, TEU/S ± bronchoscopy to show depth of invasion/LN involvement, CT of chest/abd, bone scan if Sx

**Staging (MUST ASK FOR THE TUMOR’S STAGE):** I – invades thru mucosa/LP up to submucosa, II – invades thru muc propria up to adventitia, III – invades adventitia or regional nodes, IV – distant mets

**Treatment**

**Conservative Management**
- palliation (relief of dysphagia) – XRT, stents vs. traction/pulsion tubes
- chemo: cisplatin, 5-FU

**Surgical Options**
1. Internal bypass of unresectable tumor (e.g., colon interposition)
2. Transhiatal esophagectomy with intrathoracic esophagogastric anastomosis
   - disadv: thoracoab incision -> resp complications (atelectasis, splinting), intrathoracic anast leak -> mediastinitis (50% fatal), reflux esophagitis, potential suture-line recurrence, longer hosp stay
3. Transhiatal esophagectomy (THE) without thoracotomy & cervical esophagogastric anastomosis – may combine with chemo/XRT; need to sacrifice only the short gastric aa.
   - advantages: no thoracotomy, cervical anastomosis avoids risk of mediastinitis, <20% post-op reflux, maximum vertical margins (no survival benefit, though)
4. Radical esophagectomy with en bloc node dissection: no better survival, much larger operation
5. Colonic interposition is another alternative, but a larger and more complex operation

**Prognosis:** in one study, 97% were candidates for THE – 23% 5-yr survival (80% for Stage I, 33% for Stage II)

**Topic 17: GERD**
* reflux of gastric contents/acid into the lower esophagus -> Barrett’s -> AC in 10%

**Causes:** (1) decr LES tone, (2) decr esophageal motility, (3) gastric outlet obstrxn, (4) hiatal hernia

**CC:** heartburn/pyrosis (subternal burning pain), regurgitation, dysphagia (2/2 scarring), cough, asthma, pneumonia, hoarseness, melena/anemia (2/2 esophagitis)
Differential Dx

- CAD (must rule-out), aortic dissec, PE, PTX, PUD, & all other causes of chest/epigastric pain
- other causes of dysphagia: esophageal CA, spasm, achalasia, and many others

History

- FOCUS ON NATURE OF SYMPTOMS, PRECIPITANTS, & RULE OUT DIFFERENTIALS
- HPI: stable vs. progr/AA fx (large meals, supine position, fatty foods, caffeine, EtOH, cig’s, CCB’s vs. exertion)
- PMH: GERD, CAD, PUD, blood clots
- PSH
- Meds/All: any alleviating medications?
- FH: CAD, DM, GERD/esophageal CA
- SH: smoking, EtOH, ingestion of hot foods (higher risk of esophageal CA)
- HMA: esophagoscopy? (Barrett’s?)
- ROS: F/C, fatigue/wt loss/app chg, SOB/cough/wheezing/hoarseness, abd pain/N/V/D/C

Physical Exam (IPPA): goal is to exclude CA and cardiopulmonary disease

- Vital signs, Ht & Wt with BMI
- Gen appearance
- HEENT: palpable masses or nodules
- Cardiopulm
- Abd, Extremities

Labs: unnecessary unless suspicion for CAD

Non-invasive Studies

- diagnosis is often based on Hx and a successful 7-day trial of PPI bid (Omeprazole 40/20)
- EKG

Invasive Studies

- barium swallow should be done in all pts with dysphagia – provides “road map” for EGD but will fail to show reflux in up to 50% of patients
- EGD ± biopsy shows esophagitis, ulcer, Barrett’s (columnar metaplasia), or stricture (15/40 cm)
- pH probe in lower esophagus if diagnosis is uncertain
- manometry is another diagnostic possibility to assess esophageal function/spasm

Treatment

Resuscitation: take into account what you now know about the patient’s ABC’s

Conservative Management

- medical: PPI’s, H2B’s, antacids, metoclopramide (a prokinetic agent)
- mechanical: weight loss, small meals, elevate HOB, avoid late meals and other precipitants
- “dietary”: avoid tobacco, EtOH, fatty food, chocolate, peppermint
- follow people with significant reflux with regular EGD’s with biopsies

Surgical Indications

1. failure of conservative therapy to alleviate symptoms (medical, mechanical, dietary)
2. severe esophageal injury, e.g., ulcers, strictures, hemorrhage, Barrett’s
3. respiratory problems 2/2 aspiration of reflux documented by 24-hr pH probe (likely RLL)
4. young age at onset

Major Surgical Options

1. Nissen fundoplication ± esophageal lengthening with Collis gastroplasty: open or laparoscopic, 360° wrap of cardia around lower 4-6 cm of the esophagus, so the GE sphincter passes thru a short tunnel of stomach – 90% effective
   * complications: gas-bloat syndrome (inability to burp/vomit), stricture, dysphagia, injury to other organs
2. Hill repair: GE jxn is sutured to the median arcuate lig to reposition it correctly; hiatus may also be reduced somewhat
3. Partial fundoplication (Belsey or Toupet): avoids gas-bloat syndrome or dysphagia
4. Esophageal resection if GERD persists or Barrett’s/hard stricture is present

**Topic 18: Achalasia**

* failure of LES to relax during swallowing, with loss of esophageal peristalsis 2/2 infection – Chagas’ disease or neurologic disease (ganglionic degeneration, vagus n. injury, etc.)
* food stasis (retention esophagitis) leads to Barrett’s and increased risk of adenocarcinoma
* other potential complications: megaesophagus
CC: dysphagia of both liquids/solids but especially liquids, progressively worse, often with regurg

**Differential Diagnoses & the 3 Most Important Questions**
- dysphagia of solids (mechanical problem) or solids/liquids (motility)?
  - mechanical: esophageal ring, peptic stricture, esophageal CA
  - motility: spasm, scleroderma, achalasia
- intermittent or progressively worse? the first in each group is intermittent/chronic
- chronic GERD or not? the second in each group manifests chronic GERD
  ** other intrathoracic masses may also cause dysphagia

**History**
- first ask the 3 most important questions listed above
- HPI: O / acute vs. chronic / stable vs. progressive / AA fx / ASx – pain, vomiting
- PMH: scleroderma, GERD or other esophageal diseases, CA
- PSH / Meds / All / FH: esophageal disease
- SH: travel history to South America (Chagas)
- ROS: F/C, wt loss/anorexia/fatigue, GI: N/V/D/C

**Physical Exam (IPPA)**
- Vital signs
- Gen appearance
- HEENT exam will be highest yield, palpating for masses, swallowing capability
- Cardiopulmonary, Abd, Extremity exams – simply ask, “are there any abnormalities?”

**Labs:** unnecessary

**Non-invasive Studies**
- CXR

**Invasive Studies**
- **UGI series/barium swallow (#1 test):** dilated esophageal body with bird’s beak tapering distally
- **manometry (not always necessary):** increased LES pressure and failure to relax during swallowing (vs. spasm, in which the LES RELAXES with swallowing)
- **esophagoscopy (a necessary follow-up test),** given risk of esophageal carcinoma 2/2 Barrett’s from food stasis & retention esophagitis

**Treatment**
Resuscitation: take into account what you now know about the patient’s ABC’s

**Conservative Management**
- balloon dilation of the LES – 65% effective

**Surgical Indications:** refractory to conservative mgmt, development of Barrett’s

**Surgical Management**
- esophagomyotomy (incising lower esophagus and LES) ± subsequent 270° fundoplication to prevent reflux vs. medical treatment of reflux– 90% effective
- transhiatal esophagectomy if Barrett’s is present