New

- **Retroperitoneal bleeds!!!**
- Increased r/o GIB w/ SSRI use
- **If exsanguinating then GS/IR consult**
- Controversial but some recommend provocative testing w/ heparin to induce bleeding and help w/ dx!!!
- If +Guaiac
    - +Anemia or UGI Sx then EGD/Colon
    - –Anemia and No UGI Sx then just Colon
- Jehova's Witness: determine if other blood products can be given (plts, albumin, IVIG, coag factors, etc), have pt sign a legal document indicating that they do not want blood products and that they understand the consequences including death, resuscitate w/ crystalloids and pressors, give oxygen, correct coags, stop AP/AC/NSAIDs, give VitK, Folate, VitB12, IV Iron, consider prohemostatics (tranexamic acid, desmopressin, etc), limit phlebotomy for labs)
- HIV: normal common (PUD is the most common cause of UGIB), Cancer (KS and Lymphoma) and Infection (CMV Colitis and Bartonella w/ Bacillary Angiomatosis)

Iron Deficient Anemia (IDA)	
Mechanism	• Infants: human breast milk diet is low in iron and growth • Adolescents: growth • Menstruating Females: chronic menstrual loss • Pathology Adults: chronic GI loss vs decreased iron absorption (increased gastric pH, celiac, post-gastrectomy, HP) • Pregnant Women: growth
Unique S/S	• Plummer-Vinson Syndrome (refer) • Pica (ice, starch, clay)

Diagnosis	• PBS: Micro/normocytic, hypochromic, anisocytosis, reticulocytosis
	• BM Bx: low iron stain (most sensitive and earliest sign, gold standard)
	• Iron Studies: iron exists free in plasma, bound in plasma to transferrin, and bound in tissue to ferritin, ↓ Fe (unlike ferritin when it is low it can mean many things from IDA to AOCD/APR but when it is high it means iron overload), ↓ Ferritin (most important, when low it always means IDA and when high it means AOCD, iron overload, APR, NB even when there is inflammation going on a ferritin of >100 r/o IDA) ↑ TIBC (tells you how much unbound transferrin there is, low in AOCD) ↓ %Sat (tells you how much bound transferrin there is)
	• Soluble Transferrin Receptor Assay (high in IDA vs low in AOCD, most sensitive in that it changes before your classic iron studies)
	• ↑ Platelets (>600,000)
	• Mentzer Index MCV/RBC
	  ○ >13 Iron Deficiency (anemia more impressive than the microcytosis)
	  ○ <13 Hemoglobinopathies (microcytosis more impressive than the anemia)

Treatment	• PO Ferrous Sulfate/Gluconate (each has the same efficacy)
	  - BID dosing, take with OJ or vitC b/c you need an acidic environment for best absorption, hence no PPIs
	  - Increases Absorption: lower pH, VitC, Citrate, AA, Carbs
	  - Decreases Absorption: higher pH (hence PPIs), PO4, oxalate, divalent cations
	  - SEs: constipation therefore give with laxative (non-compliance is big)
	  - 325mg = 65mg of elemental iron
	  - Niferex (iron polysaccharide) has less GI SEs and more elemental iron per mg than ferrous
	• IM/IV Iron Sucrose (Venofer)
	  - Qwk dosing
	  - Iron Dextran has been found to cause anaphylaxis therefore no longer used **except in TPN**
	  - Indications: pts who have poor absorption, pts who require so much iron that oral replacement is not sufficient, pts who cannot tolerate PO (NB iron is absorbed in proximal duodenum and requiring stomach acid)
	• NB takes 6wks to correct anemia and 6mos to correct iron stores

Descriptions

- 1st: **Occult** (GIB that is unknown to pt in that there is no visible blood but there is evidence of GIB like IDA and/or FOBT) vs **Overt**
- 2nd: **Obscure** (GIB that persists/recurs without obvious etiology after initial endoscopy/imaging, 5% of GIB) vs **Non-Obscure**
- 3rd: **Upper** (5x) vs **Lower** (1x)

Approach

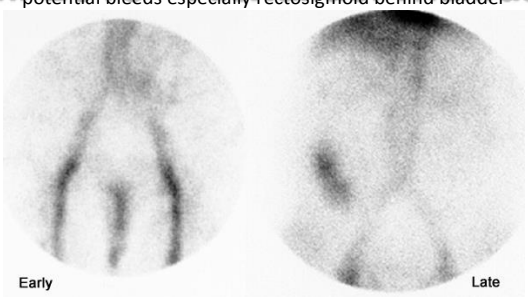
- **1st Stabilize/Risk Stratify/Labs/Brief H&P (use GIB order set)**
    - Blatchford or Rockall Score
    - Variceal Bleed automatically make it a severe GIB
    - Stabilize Hemodynamically (IV Access w/ two large bore peripheral IVs and a central line and start fluids, intubate if ongoing bleeding and r/o aspiration b/c of AMS, T&C many units and begin transfusion NB Hct can be falsely normal)
    - Labs (CBC, LFTs, Coags, Hemostasis Profile, Iron Studies, HP Serology, Serial Hcts Q6hrs)

- Brief H&P
- NGT Lavage
- Keep NPO

- **2nd Assess U vs L GIB**
  - UGIB
    - Hx: N/V, upper ab pain
    - Labs: high BUN/Cr
    - Stool
      - Acute/Severe: Hematochezia aka BRBPR (need >1000mL, mixed in) if brisk bleed and Hematemesis, fresh blood in NGT (Aljebreen in 2004 GIE reported that 15% of UGIB had no blood return on NGT aspirate)
      - Chronic/Mild: Melena (only need >100mL, black, tarry, sticky stools w/ a characteristic odor from digested blood, NB iron makes stool green/black and pepto makes stool black but no change in odor) w/ chronic IDA and Coffee-Ground Emesis w/ +Guaiac in NGT
  - LGIB
    - Hx: D/C, lower ab pain
    - Labs: nl Bun/Cr
    - Stool
      - Acute/Severe: Maroon (R Colon) / Hematochezia (L Colon / Rectum)
      - Chronic/Mild: (same as above)
  - **Fecal Occult Blood Test (FOBT)**
    - Normal fecal blood loss (0.5-1.5mL/d) w/ most FOBT tests becoming + when >2.0mL/d
    - Hgb is cleaved into heme and globin by gastric pepsin or pancreatic protease
    - Types
      - **Guaiac, Hemoccult-II, Hemoccult Sensa** apply stool to colorless guaiac impregnated paper w/ hydrogen peroxide, heme has pseudoperoxidase activity, so if blood is present then heme with hydrogen peroxide turns the colorless guaiac into blue quinine, rehydration of stool increases sensitivity
        - False Negative
          - more upper GIB b/c heme and its psuedoperoxidase are degraded as it moves down the GI tract
          - sampling error due to intermittent bleeding
          - very mild bleed (<2mL/d))
        - False Positive
          - diet rich in peroxidase: red meat and vegetables (radishes, turnips, beets, horseradish, mushrooms, broccoli, apples, bananas, cantaloupe)
          - meds: iron, aspirin???
          - nose bleed or hemorrhoids
      - **Fecal IHC Test (FIT):** uses Ab to Hgb, much higher sensitivity (>0.3mL/d) and specificity (myoglobin in red meat and oxidizing agents in vegetables do not give a + test) but a little more expensive, send out not at bedside
      - **Heme Porphyrin Test (HemoQuant):** uses spectrometry to measure heme related porphyrin, much higher sensitivity (?, UGIB are still positive) and specificity (oxidizing agents in vegetables do not give a + test) but a little more expensive, send out not at bedside

- **3rd Meds**
  - Protonix 80mg IV x1 then 8mg/hr IV gtt (severe, at admission) then 40mg IV BID (mild, once stabilized b/f EGD) then PO BID (after EGD) then PO QD
    - why? gastric acid impairs clot formation (pH<6), promotes platelet disaggregation (pH<6) and activates pepsin (pH<4), therefore you want to raise pH to ideally >6.8, IV vs PO likely doesn't matter just remember short t1/2 hence if give PO then give very frequently, data is only on omeprazole/pantoprazole, IV PPI can rarely cause an adverse event called "non-arteric anterior ischemic optic neuropathy" in which there is sudden loss of visual acuity)
    - NB interestingly studies indicate that AA and H2B are not effective at all b/c they are not able to bring pH >6.8 for an extended period of time b/c of tachyphylaxis
  - Octreotide
    - comes in a solution of 5mcg/mL, 100mcg IV x1 if actively bleeding then 50mcg/hr IV gtt then 25mcg/hr IV gtt then stop
    - when you are banding it is sometimes good to give a 100mcg bolus before you start to decrease pressure
    - NB SOME SAY DO IT FOR NON-VARICEAL BLEEDS b/c inhibits acid and decreases gastroduodenal blood flow but the data says NO!!!
  - Abx for every cirrhotic w/ GIB
  - Correct Hct/Plt/Coags: PRBC, Plt, **VitK 10mg IV x1 & 3U FFP**
  - Prep: erythromycin 3mg/kg IV x1 over 30min, 60min b/f EGD (promotes gastric emptying of blood clots) AND PEG 1L PO Q30min until done for colonoscopy
  - Before Discharge

        - Oral PPI
        - Carafate
        - misoprostol (esp if pt has to take a strong NSAID as for Rheumatoid Arthritis but higher r/o diarrhea)
        - change to COX-2 if cause is 2/2 NSAIDs
        - eradicate HP
        - need for iron
- **4th Diagnosis/Treatment**
    - Endoscopy (the question is when or even if to do endoscopy, if trivial bleed consider elective out-pt endoscopy following empiric Tx, some do a second look 24hrs later to investigate rebleeds, long term f/u endoscopy is important to f/u healing and malignancy, always look for lesions during insertion and not withdrawal b/c lesions can look like trauma from scope, never Bx vascular lesions, naloxone can enhance colonic vasculature)
        - **EGD**
            - always r/o UGIB first b/c 10% of pts you think have a LGIB have an UGIB
            - if you have a large clot then (1) turn the pt around to move clot around to visualize stomach, (2) use a large channel scope and hook a "US Endoscopy Biovac Direct Suction Device and Velocity Irrigation System" to the therapeutic channel and (a) suck directly thru there instead of going thru control head of scope which decreases suction force and (b) shoot a high power water jet, (3) place a rubber-Ewald (not as good) or plastic-Edlitch (much better) tube (large bore tube that has holes on side to suck out large clots, perform warm (to break down clot) or cold (to constrict blood vessels but doesn't actually work) tap water lavage, don't use actual suction b/c it will suck up mucosa rather hang to gravity)
            - 2nd look ONLY if clinically signs suggest recurrent bleeding or if hemostasis during first EGD was questionable
        - **FlexSig/Colonoscopy** (if EGD negative then explore LGIB w/ PR enema then FlexSig or w/ PO PEG purge via NGT w/ HOBE and give Erythromycin/Reglan (however in some pts with lots of blood, the blood is a cathartic itself), then Colonoscopy, often done non-urgently b/c of poor visibility w/o proper prep, potential for complications and possible adverse effects of purging colon in setting of active GIB, a study comparing urgent vs elective colonoscopy showed no improved clinical outcomes w/ the urgent approach)
        - **Capsule/BE/SE**
    - Radiology (these tests are mainly done for LGIB only as there is lots of background activity in upper GI tract b/c unbound Tc is naturally excreted by stomach)
        - **99m-Tc Tagged RBC Scintigraphy Scan**
            - Good: cheap, less invasive, can be repeated, risk of contrast reaction, sensitive down to slow bleeds 0.2cc/min
            - Bad: not accurate, no therapeutic option
            - Mainly used as a screening/localization test for mesenteric angiography and/or surgery rather than a true definitive test
            - Often pts are rescanned at 6,12,24hrs to make sure there is no bleed
            - A bleeding site should show increased activity that then travels antegrade (but also sometimes retrograde b/c blood is a GI motility stimulant) over time conforming to shape of bowel and if it doesn't then it probably is not a bleed but an aneurysm or angiodysplasia
            - b/c 99m-Tc is cleared renally the kidney/ureters/bladder while also light up sometimes obscuring potential bleeds especially rectosigmoid behind bladder



(GIB in ascending colon)

        - **Mesenteric Angiography**
            - Good: very accurate, therapeutic capabilities
            - Bad: expensive, more invasive, cannot be repeated, sensitive down to fast bleeds 1.0cc/min, r/o contrast reaction, vessel damage, bowel ischemia from arterial thrombosis, etc
            - Only do if pt has active bleeding b/c often LGIB stop spontaneously
            - Go thru L femoral artery and selectively cannulate the Celiac/SMA/IMA
            - Studies comparing colonoscopy to angiography indicate that colonoscopy is much safer and has a higher diagnostic yield
            - Tx: a catheter is fed selectively to the bleeding site and vasopressin is continuously infused (not done anymore b/c not very effective and lots of SEs), selective embolization with steerable

microcatheters using coils and various chemicals (r/o bowel ischemia, coil migration, contrast nephropathy)
        - **99m-Tc Pertechnetate Scan aka Meckel's Scan (refer)**
    - Surgery
        - Needed for recurrent or continuing GIB of known (cancer, Meckel's, etc) or unknown (blind subtotal colectomy based on radiographic findings but source cannot be found on endoscopy but this has high M&M and is only done as last resort)
        - **Exploratory Laparotomy w/ IntraOperative Endoscopy** (oral/anal/enterotomy) esp if aortoenteric fistula, large AVMs, ulcers on lesser curvature of stomach or posterior duodenal wall (considered a last ditch effort)
- NB 98% of rebleeds occur 3-4d after initial episode w/ RFs including: acute bleed, >60yo, bloody NGT aspirate, >5U PRBCs, varices, cancer, arterial spurting, visible vessel, adherent clot that does not wash off on endoscopy

UGIB (proximal to Ligament of Trietz – LOT, 75%, NB 30% have multiple causes while 10% have no identifiable cause, rule out swallowed oro/nasopharyngeal blood)

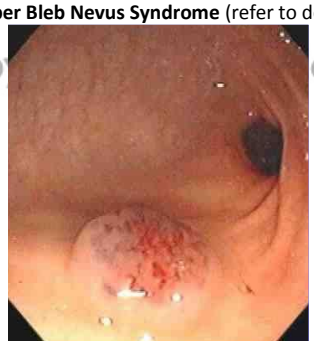
- **PUD** (40%) (refer to stomach)
- **Not Identified** (25%)
- **Esophagitis/Gastritis/Duodenitis** (20%)
    - often deemed the cause of UGIB when nothing else is present but it is unlikely that mucosal inflammation actually causes significant bleeding
- **Varices** (15%)
- **Mallory Weiss Tears** (10%)
- Other (5%)
    - **AVM** (refer to derm)
    - **Large Hiatal Hernia w/ Cameron's Erosion**
        - First noted by Cameron and Higgins in 1986
        - Linear ulcerations in the neck of LARGE hiatal hernias
        - Along lesser curvature at level of diaphragmatic hiatus
        - Often missed on EGD b/c of location and b/c they are often lost b/t gastric rugae
        - Due to wear and tear from diaphragmatic movement
        - Can be multiple
        - Seen in 5% of hiatal hernias, more likely the larger the hernia
        - Often incidental finding as they rarely bleed but they can chronically ooze
        - If refractory to PPI then anti-reflux surgery
    - **Dieulafoy Lesion (DL)**
        - Historical Perspective
            - First described as an "aneurysms miliares" by Theodore Gallard in 1884 and then later characterized by the French surgeon, George Dieulafoy, in 1897 and coined the term "exulceratio simplex" b/c he believed that these lesions represented the initial stage of a gastric ulcer
        - Definition
            - arteriole that retains the caliber of its feeding vessel as it approaches mucosa hence much larger than usual (1-3mm, 10x normal size) eventually eroding thru the mucosa
            - pathogenesis unknown but likely congenital though very few pediatric cases have been reported
            - the trigger for bleeding is unclear and likely a combination of physical stress, peptic digestion, and mucosal damage from ingestants (NSAIDs, EtOH, etc)
            - there is some evidence that these vessels are abnormally fixed and undergo physical stress during peristalsis
        - Pathology
            - Large, tortuous, thick walled (subintimal fibrosis w/ loss of elastic fibers) arteriole that courses thru the muscularis mucosa
            - Importantly there is no evidence of aneurismal dilation, atherosclerosis or vasculitis though pt's with Takayasu and Behcet's vasculitis have been known to present w/ a Dieulafoy like lesion
            - These abnormal arterioles are also accompanied by abnormal veins and one study suggests that ¾ of DL bleeds are 2/2 concomitant arterial and venous bleeds
        - Location
            - 1° Stomach (79%)
                - Proximal Stomach (65%)
                    - most are 6cm distal from GEJ along lesser curvature b/c this part of the stomach is not supplied by a submucosal plexus but instead derives its blood supply from tributaries from of the L and R gastric artery
                - Body Stomach (23%)
                - Distal Stomach (4%)
                - Anastomosis (8%)
            - 2° SI (15%: D-14%, J/I-1%)
            - 3° LI (5%)
            - 4° Esophagus (1%)

- 5° Extra-GI eg. Bronchus (rare)

- Epidemiology
  - most studies of UGIB at tertiary care centers indicate that DLs represent 1-5% of all UGIB
  - ~60yo but there have been case reports from every age group
  - 2M:1F
  - pt's often have multiple co-morbidities (vasculopathy, metabolic syndrome, et al) and med use (NSAIDs, antiplatelets, anticoagulants, et al) typical for that age group but whether these are contributing factors is unclear
- S/S
  - no prior GI Sx
  - p/w acute massive UGIB that is often intermittent and recurrent
- Dx
  - (1) visible active arterial spurting / micropulsatile streaming at a small mucosal defect (<3mm) w/ normal surrounding mucosa, (2) protruding vessel at a small mucosal defect (<3mm) w/ normal surrounding mucosa, (3) adherent clot with a narrow attachment point at a small mucosal defect (<3mm) w/ normal surrounding mucosa
  - when overt bleeding or stigmata of recent bleeding is not present these lesions are often missed and multiple endoscopies are performed
  - good preparation w/ adequate removal of blood clot and careful inspection of proximal stomach is imperative
  - Doppler EUS can often show the large abnormal vessel coursing submucosally for several centimeters until it emerges
- Prognosis
  - Before modern flexible endoscopy mortality from a DL bleed was high (~80%) as the only available therapy was surgical
  - Presently mortality is still high around 10% however exsanguination is only responsible for death in a fraction of pts, in most other cases pts often succumb to their comorbidities
  - Rebleeding occurs uniformly if unTx
- Tx
  - Stabilize Patient + PPI gtt
  - Endoscopic Therapy
    - higher diagnostic/therapeutic yield the sooner endoscopy is performed
    - combination therapy is advocated w/ initial epi injection for hemostasis followed by thermal coagulation (BICAP, APC) or mechanical occlusion (hemoclipping, banding)
      - NB best mode of therapy is still not established as there remains to be a prospective, randomized, controlled trial that demonstrates the superiority of one modality over another
    - BICAP: typically used to ablate deep high flow lesions (eg. ??)
    - APC: typically used to ablate superficial low flow lesions (eg. GAVE, AEs, Radiation Enteritis, et al) but recent studies indicate that APC can sufficiently coagulate DL given the fact that they are superficial and exceeding small
    - Hemoclips: given that this is a single vascular lesion the use of hemoclips seems to be a logical Tx modality, the largest series published by Chung et al demonstrated a 100% hemostasis in the 24 patients they had prospectively studied.
    - EBL: bands which classically have used to treat varices have been used with some success to treat DL, the largest series published by Nikolaides et al demonstrated a 96% hemostasis in the 23 patients they had prospectively studied, however there is a risk of perforation as parts of the stomach are thin and recurrent bleeding after ulcer formation and band loss
    - Doppler EUS can be used to confirm ablation by documenting the absence of blood flow
    - Consider tattooing b/c they often rebleed
  - Initial hemostasis is effective in 90% of cases but rebleeding after endoscopy is common occurring in 6-40% of cases
    - b/c of the rebleeding rate some advocate that tattooing the site could help in future endoscopic/surgical Tx
    - when endoscopic therapy has failed selective arterial embolization by IR or wedge resection by GS should be considered

- **Watermelon Stomach (WS) or Gastric Antral Vascular Ectasia (GAVE)**
  - RFs: old female with connective tissue disorders (esp scleroderma), gastric autoimmune disorders (esp pernicious anemia, autoimmune chronic gastritis w/ achlorhydria), other organ disorders (esp cirrhosis) and after BM Tx, very different from portal hypertensive gastropathy which is seen in fundus, pathogenesis otherwise unclear but believed to be related to abnormal gastric peristalsis w/ prolapse of loose antral mucosa, delayed gastric emptying, overproduction of various factors (gastrin, PGE2, serotonin, VIP), etc
  - Endoscopy: longitudinal antral folds covered by tortuous red ecstatic vessels radiating out from pylorus (can be misdiagnosed as gastritis) but if more generalized then it is called "Diffuse Gastric Vascular Ectasia"

        - Bx: superficial fibromuscular hyperplasia of gastric antral mucosa w/ capillary ectasia and microvascular thrombosis in the lamina propria (but rarely Bx b/c will bleed like crazy)
        - S/S: chronic and/or acute GIB
        - Tx: endoscopic contact (heater probe) or non-contact (APC) thermal ablation only if Plt>45k and INR<1.4 and if fails then antrectomy, there is some evidence that estrogens work (35mcg estradiol and 1mg norethindrone PO QD), liver transplantation is effective but NOT TIPS suggesting that it is not from portal HTN but from liver dz itself
    - **Portal Hypertensive Gastropathy (PHG) (refer)**
    - **Aortoenteric Fistula**
        - Primary (no prior surgery, graft, etc just 2/2 underlying atherosclerosis, very rare, only 250 reported cases) vs Secondary (post vascular surgery, vascular graft, infection esp TB/Syphilis, tumor invasion, radiotherapy, foreign body, etc)
        - Location depends on where secondary process occurs (eg. thoracic aorta, abdominal aorta, etc) but generally 55% 3rd part of duodenum 28% esophagus, 15% SB/colon, 2% stomach
        - Men >60yo, can occur anywhere from 21d-15yrs after secondary insult
        - Mechanism: theoretically with each systolic pulsation there is gradual erosion to adjacent small bowel resulting in eventual erosion into bowel (this is increased when the graft has a chronic infection w/ S. aureus or E. coli)
        - p/w first "herald" or "mild" bleed from bowel erosion which is self-limited b/c of hypotension/thrombosis/spasm but variably after then there is a "major" bleed from aortic fistula itself
        - Triad: GIB + AB pain + Pulsatile Ab Mass
        - high mortality
        - Dx: 1st: CT (air in aorta, etc) 2nd: Endoscopy has low yield (often missed but if something is seen it is variable from subtle erosion with thrombus to visible fistula w/ graft)
        - Tx: abx (b/c of the potential bacteremia that occur from organisms passing from GI tract thru fistula into blood but b/c blood heads other direction into GI tract infection is uncommon), surgical endovascular graft or IR stenting
    - **Hemobilia**
        - Hemorrhage into biliary tree 2/2 communication b/t vessels and biliary tree 2/2 trauma, iatrogenic procedure (liver biopsy, cholangiogram, cholecystectomy, TIPS, stenting), cholecystitis/cholelithiasis, hepatic artery aneurysms, liver abscess, cancer, etc
        - Triad of biliary colic + obstructive jaundice + UGIB
        - Tx: mesenteric angiographic coiling
    - **Hemosuccus Pancreaticus**
        - DDx: chronic pancreatitis, psuedocysts, pancreatic tumors, trauma from therapeutic ERCP, etc which leads to erosion of pancreatic duct into peripancreatic blood vessels (1° Splenic 2° GDA)
        - Tx: mesenteric angiographic coiling
    - **Malignancy**
        - significant bleeding suggests malignant vs benign, most common is gastric adenocarcinoma
        - Tx: injection and snare electrocoagulation if small vs surgery if large
    - **Blue Rubber Bleb Nevus Syndrome** (refer to derm)



    -
    - **Hereditary Hemorrhagic Telengectasia aka Osler-Weber Rendu Disease** (refer to derm)
    - **Kaposi Sarcoma**
    - **Vasculitis**
    - **Amyloidosis**
    - **Malignant Atrophic Papulosis aka Kohlmeier-Degos's Disease**
    - **Psuedoxanthoma Elasticum**
    - **CTDs esp Scleroderma**
    - **Duodenal Diverticulosis**
    - **Boerhaave's Syndrome**
    - **Intussception**
    - **Volvulus**

LGIB (distal to Ligament of Trietz – LOT, 25% but under-reported, increases w/ age, if massive likely a surgical emergency, NB 10% have a more proximal source that is just so brisk that it manifests as a LGIB)

- 1st: rule out **Anorectal Disease**
    - There is emerging evidence that internal hemorrhoids can cause a severe hematochezia as thus is now considered the 2nd most common cause NOT AEs!!!
- 2nd: likely **Colonic Pathology**
    - **Non-Painful (often pts have non bleeding diverticulae and angioectasias so you really don't know the cause)**
        - **Diverticular Bleed** (30%)
            - usually a DOE in which pt has diverticula but no direct evidence of bleed but you ruled out everything else so you just assume that the diverticula was the cause of LGIB
            - bleeding is rarely seen on colonoscopy and angiography
            - abrupt painless hematochezia 2/2 thin artery than ruptures (a new concept is that NSAIDs are a RF for bleeding therefore have pts have pts avoid NSAIDs if they have a h/o a diverticular bleed)
            - though diverticula more common in L colon, bleeding diverticula more common in R colon
            - 75% usually stop spontaneously however 30% have massive bleed
            - 30/50% have a 2nd/3rd episode
            - Tx
                - Endoscopic (epi injection/electrocautery at margin never go blindly into dome but if wide mouthed you can Tx, some have also tried hemoclipping the mouth closed, NB often diverticular bleed expectedly stop bleeding on their own, NB dangerous b/c R colon has a thin wall, NB difficult to find the index tic b/c of blood and the multiple number of tics)
                - IR (IA vasopression or embolization)
                - Surgery (partial colectomy) esp for recurrent cases
        - **Angiectasia (AE) aka Angiodysplasia (AD)** (10%)
            - Mechanism: repeated/intermittent obstruction of mucosal/submucosal small vessels (venules/capillaries/arterioles) from contracting bowel throughout life leading to tortuous dilatation (aka ectatic) w/ creation of AV communications
            - Location: ascending colon but can be found anywhere, usually multiple, <10mm
            - RFs: 1° ESRD, 2° Cirrhosis, Scleroderma, Radiation, CVD, von Willebrand
            - Epidemiology: increases w/ age
            - DDx: radiation injury, spider telengiectasias, HHT
            - Dx: endoscopy but also angiography (densely opacified, slow emptying, dilatated, tortuous vein)
                - NB if you find them incidentally on endoscopy don't Tx b/c their r/o bleeding is low (unless they have a problem w/ bleeding, anemia, etc)
            - S/S: low-grade/recurrent/chronic lower GI bleeding although 15% result in massive hemorrhage
                - NB In 1958 Dr. Heyde described a relationship b/t aortic stenosis and AEs = William's/Heyde's Syndrome (refer), this relationship is controversial, it has been recommended that AVR be done for difficult to control AE bleeding!!!
            - Tx: if non-bleeding then don't do anything but if bleeding know that 90% of bleeds stop spontaneously but if you find one during colonoscopy then do endoscopic Tx, if that fails then do angiography w/ microcoil embolization or vasopression instillation, if that fails then consider hormone therapy w/ E+P (eg. Ortho-Novum) mech is unknown but believed to be 2/2 procoagulant effects, other (thalidomide, octreotide, Aminocaproic Acid, Tranexamic Acid, Danazol, etc) and if that fails then R hemicolectomy
                - NB often disappear w/ low BP from dehydration, shock, anesthesia
        - **Neoplasm/Polyps** (10%) usually very mild
    - **Painful**
        - **Colitis** (20%) esp ischemic but also infectious, radiation, IBD, eosinophilic, et al
    - **Other**
        - **Congenital AVM** (refer to derm)
        - **Hemangioma** (refer to derm)
        - **Klippel-Trenaunay Parkes Weber Syndrome** (refer to derm)
        - **Colonic Varices**
        - **Endometriosis**
        - **Postpolypectomy** (refer to imaging)
        - **Intussception**
        - **Volvulus**
        - **Dieulafoy**
        - **Long Distance Runner LGIB**
        - **Portal Hypertensive Colopathy** (refer above)
- 3rd: assess **SI Pathology**
    - **Crohn's**
    - **Cancer**
    - **AE/ADs**

    - **AVMs**
    - **Meckel's Diverticulum**
    - **Radiation**
    - **Neoplasm**
- 4th: Psych
    - **Ingestion of Blood**