Essential Learning
GI Bleeding

- **What is the differential diagnosis for vomiting blood?**
    - Common causes include: peptic ulcer disease, erosive gastritis, esophagitis, variceal bleeding and Mallory Weiss tear.
    - Less common causes include: epistaxis, aortoenteric fistula after AAA repair, Boerhaave syndrome, carcinoma, vascular anomalies, and caustic ingestion.
    - History is important in differentiating between these etiologies.
        - Important aspects include history of GERD, ulcers, anticoagulant use, NSAID use, alcohol use, recurrent vomiting, AAA repair, and ingestions. In patients with known history of cirrhosis, variceal bleeding should be on top of the differential as this is a potentially life-threatening presentation of GI bleeding.
        - Another important aspect of the history is asking about the color of vomit and stool. Upper GI bleeding classically presents with hematemesis and melena. Vomiting bright red blood indicates ongoing acute hemorrhage whereas coffee ground emesis suggests subacute upper GI bleed. Melena or dark stools point to digested upper GI bleeding sources.

- **How can you differentiate between upper GI bleed and lower GI bleed?**
    - GI bleeding is divided into upper and lower based on the location of the source of bleeding. Upper GI bleeding is classified as originating proximal to the ligament of Treitz. In lower GI bleeding, the source is below the ligament of Treitz.
    - Upper GI bleeding usually presents with coffee ground emesis (subacute bleeding), hematemesis (acute bleeding), and melena.
    - Lower GI bleeding usually presents with maroon colored stool or hematochezia. However, brisk upper GI bleeding can also present with hematochezia.
    - The source of bleeding can often be differentiated by a thorough history and physical exam including rectal exam and hemoccult stool testing. Ultimately, direct visualization with EGD or colonoscopy is often necessary to identify the source.
    - BE AWARE! Common mimickers of dark stool include iron supplements and pepto bismol which will be hemoccult negative. Common mimickers of GI bleeding are red foods such as gatorade and beets. Be sure to ask about consumption.

- **What is the pathophysiology of esophageal varices?**
    - Esophageal varices are swollen veins in the esophagus due to portal hypertension. The veins in the stomach and esophagus empty into the portal vein which is within the liver. In patients with cirrhosis, there is significant scarring within the liver. Scarring impedes blood flow through the liver resulting in back pressure within the portal system.

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Increased portal vein pressure is referred to as portal hypertension. As pressure builds up within the portal system, the esophageal and gastric veins which empty into the portal system also have a build up of pressure. Veins are thin walled and, under high pressure, can swell.

- Varices can remain stable, increase, or decrease based on liver disease progression or improvement. They are not dangerous until they rupture or leak.

- **What are the critical steps of ED management and why?**
  - Critical steps in ED management start with the ABCs and include securing a patient's airway if not maintaining their own, establishing adequate IV access for resuscitation with IVF, pRBC and PLT (< 50), and early involvement of GI specialist for definitive management.
  - Airway protection: patients will often require airway protection if significant hematemesis is present and/or they are unable to maintain their own airway. Additionally, intubation is required if placing a Blakemore or Minnesota tube.
  - IV access: Due to the high pressure within the portal system causing varices to rupture or leak, varices have the propensity to bleed briskly which subsequently requires resuscitation in large volumes. That is why establishing large bore IV access with two large bore IVs (14G or 16G preferred) or a central line is critical.
  - Volume resuscitation: Variceal bleeding often results in hemodynamic instability due to large volume loss. Patients can lose up to 20% of blood volume before showing a drop in hemoglobin. It is important to support hemodynamics with IVF while awaiting blood, then transfuse with pRBC to restore blood volume. If PLT < 50, platelet transfusion is indicated to help with coagulopathies associated with liver dysfunction. While volume resuscitation is an important and critical aspect to management of variceal bleeding, it is equally important to be aware that over-resuscitation can lead to increased pressure in the portal system and thus, worsen the problem. There must be a careful balance!
  - Consultation: Early involvement of GI specialists is critical for definitive management. Exsanguinating patients should also have surgical consultation
  - Disposition: Patients should be admitted to the ICU.

- **What are temporizing and preventative therapies that can be initiated in the ED while awaiting definitive GI management of variceal bleeding?**
  - Definitive management by GI is often delayed if patients are very unstable. In the emergency department, temporary measures and preventative therapies can and should be initiated beyond the critical steps previously discussed.
    - Pharmacologic therapies such as octreotide can be utilized to reduce portal pressure. Octreotide is a somatostatin analog that reduces portal hypertension, azygos vein blood flow, and wall tension of varices. ED providers should give octreotide early in the management of esophageal varices.
    - Because cirrhotic patients are susceptible to infection and bacteremia most often from spontaneous bacterial peritonitis (SBP), empiric treatment with a

third generation cephalosporin (ceftriaxone 1g IV) or fluoroquinolone (ciprofloxacin 400 mg IV) has been shown to improve patient outcomes.
        - Acid suppression with a PPI (bolus and infusion or twice daily dosing) is also recommended for upper GI bleeding but this is more relevant if bleeding is related to acid production rather than varices.
        - Consider treating coagulopathies if the patient has life-threatening bleeding and is on anticoagulant medications. PCC, vitamin K, FFP, cryoprecipitate, and tranexamic acid are considerations.
        - Consider DDAVP if the patient has GI bleeding and is uremic or taking antiplatelet medications.
    - If life-threatening variceal bleeding is not controlled with the aforementioned resuscitation efforts, providers can place a Blakemore or Minnesota tube to tamponade bleeding as a temporizing measure prior to EGD. The following link provides instruction and videos on tube placement: https://emcrit.org/emcrit/blakemore-tube-placement/
        - This is a temporizing measure (max 24 hours) that has potentially fatal complications including aspiration, migration, and esophageal perforation.

- **What are the indications for blood transfusion and coagulopathy reversal in patients with an UGIB?**
    - Overtransfusion with IVF or blood can increase the risk of rebleeding; titrate transfusions to clinical status as noted below.
    - For unstable patients with massive hemorrhage or persistent hypotension, aggressive transfusion, including massive transfusion protocols, may be indicated.
    - For “stable” patients, most guidelines recommend transfusing when Hb < 7 for UGIB with or without portal hypertension in the absence of hypotension, shock, or preexisting cardiovascular disease.
    - There is no consensus for when to transfuse actively bleeding patients, but transfusion should be considered in markedly hypotensive patients even before the Hb reaches 7.
    - There is little evidence to guide correction of coagulopathies and thrombocytopenia. PT/INR is an unreliable predictor of coagulation status in patients with cirrhosis (can consider TEG). No randomized controlled trials have evaluated vitamin K for UGIB in patients with liver disease. That being said, it is reasonable to reverse coagulopathy if INR is elevated or platelet count is < 50,000.
        - Consider treating coagulopathies if the patient has life-threatening bleeding and is on anticoagulant medications. PCC, vitamin K, FFP, cryoprecipitate, and tranexamic acid are considerations.

- **How are esophageal varices definitively treated?**
    - Definitive management begins with early visualization with endoscopy by a gastroenterologist. Endoscopic therapies include variceal banding or sclerotherapy. Banding involves placing a rubber band around the varices causing them to become necrotic. Sclerotherapy involves injecting varices with sclerosing agents resulting in thrombosis and tissue necrosis. Banding is the preferred method but for deeper and

more hard to visualize varices, sclerotherapy is often utilized. If these methods do not work, a patient can undergo a more invasive procedure known as a TIPS (transjugular intrahepatic portosystemic shunt) procedure. This involves an interventional radiologist creating a shunt between the high pressure portal system and the hepatic vein, thereby shunting the blood away from the liver and reducing pressure. Ultimately, patients may require liver transplantation.

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- Image References
    - Chest X-ray from “JetEM”: http://jetem.org/ettcxr/