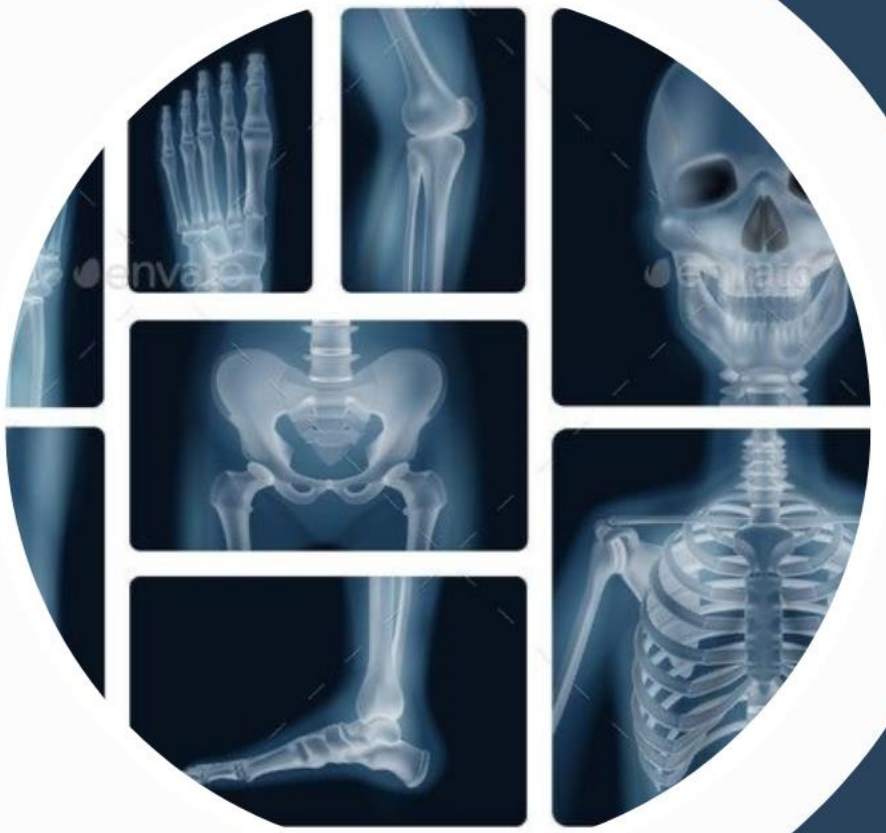
GI

PBL

LEC no. 2



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Non-Variceal
Upper Gastro-Intestinal Bleeding

Variceal GI bleeding is bleeding related to liver failure and portal hypertension and its complications, not our topic in this lecture

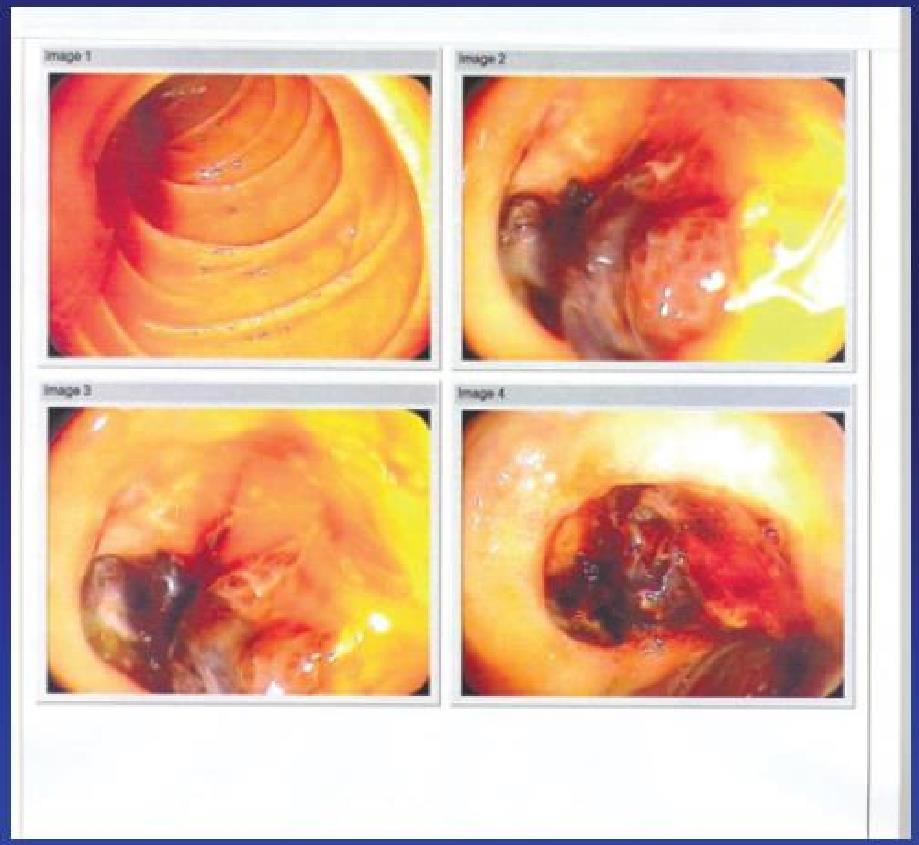
Non-variceal GI bleeding is not related to Liver diseases, which is our topic

Acute Upper GI Bleeding: A Lethal Disease

Outcomes include

- DEATH
- CARDIAC ARREST
- MI
- CVA
- INJURY (e.g. Fx, head)
- SEIZURES
- SURGERY or ANGIOGRAPHY
- RISK FOR FUTURE BLEEDING

ASA-associated DU eroding into GD artery



The outcomes of GI bleeding vary depending on the severity of the injury and the amount of blood lost, they could be mild like artery rupture from excessive vomiting (Mallory weiss disease), or severe with a lot of blood lost like in big peptic ulcers

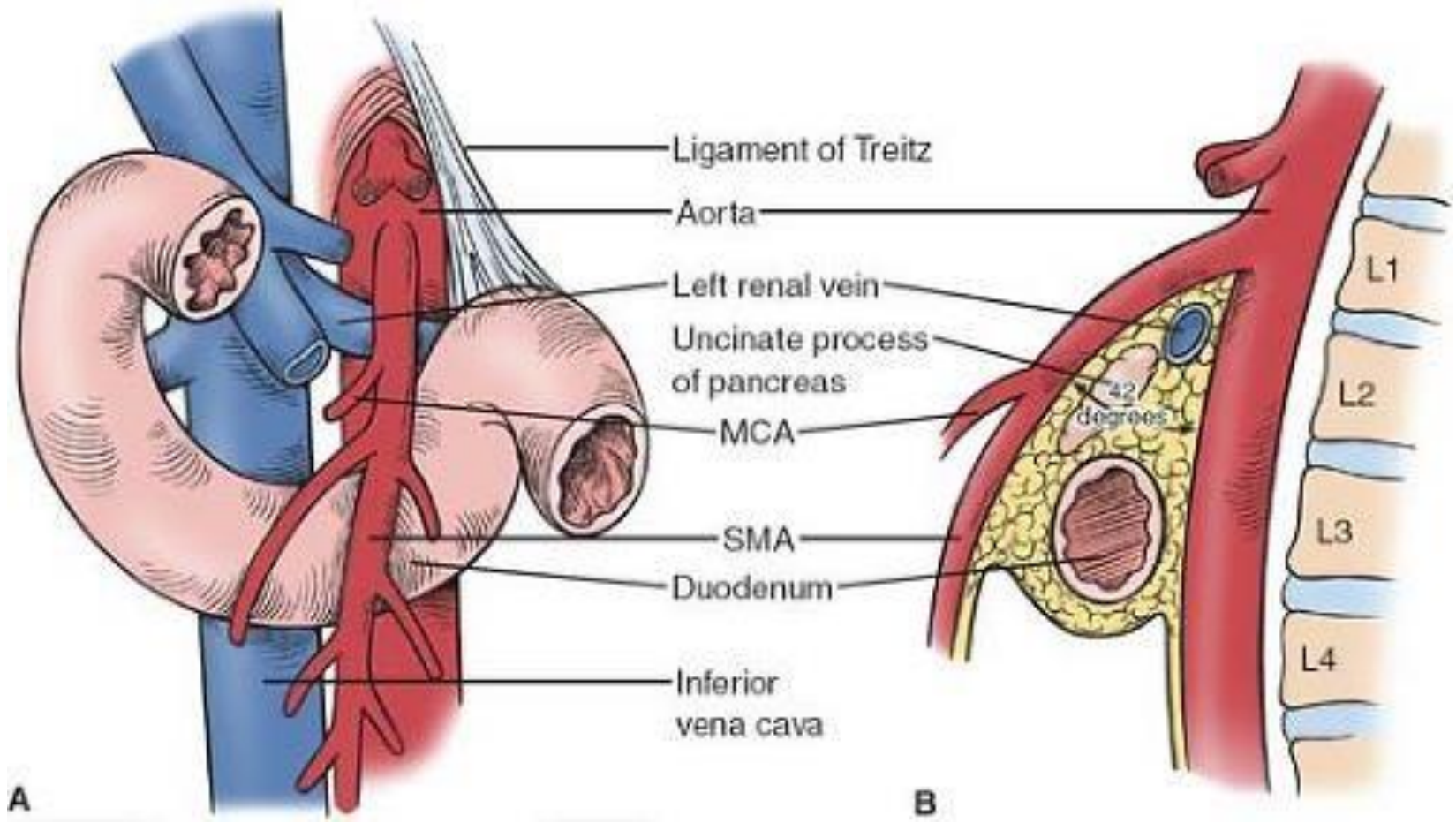
(Upper GI Bleeding) UGIB

The only difference between upper and lower GI bleeding is anatomical

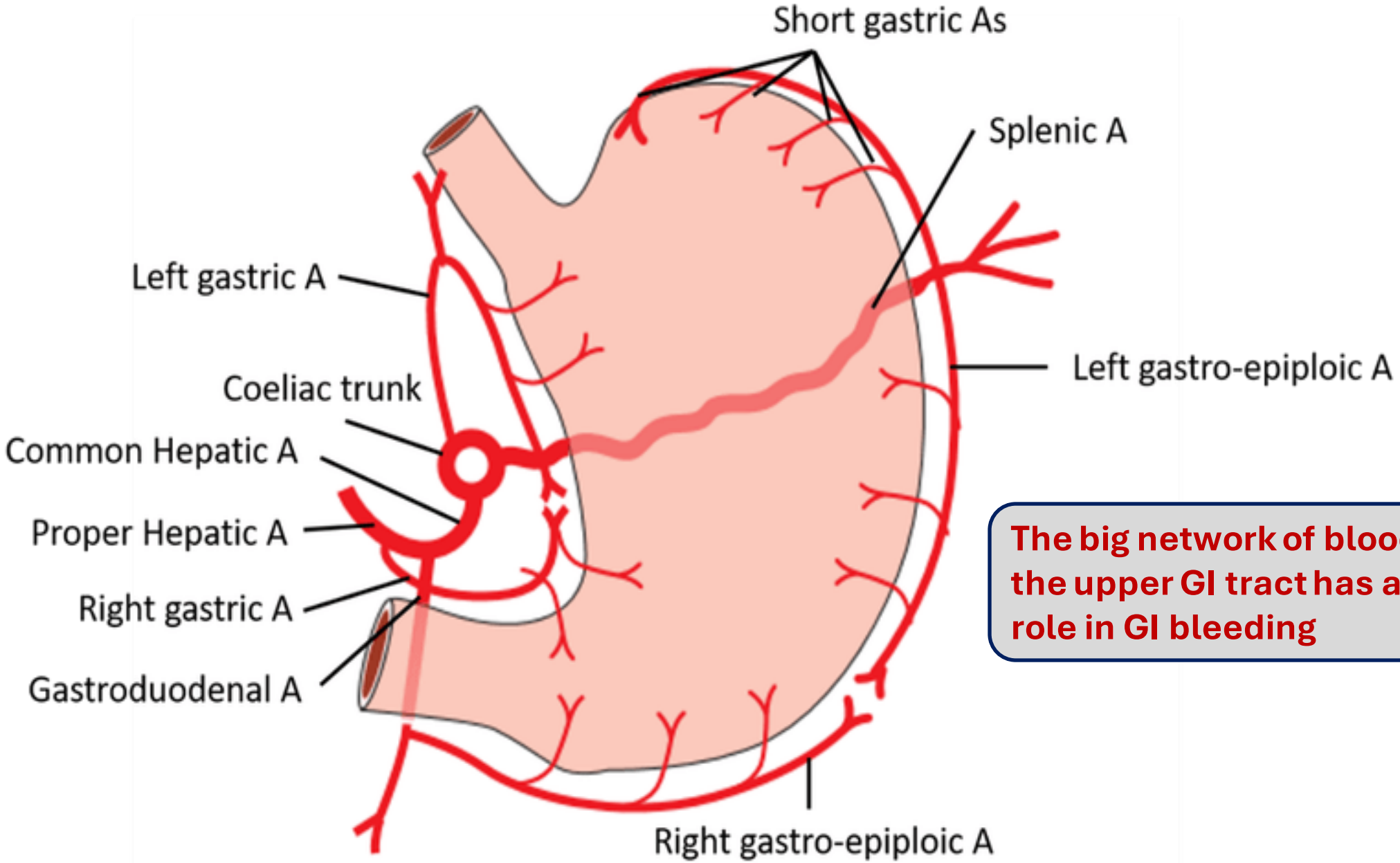
Is bleeding proximal to:

“Ampulla of Vater or (precisely) Ligament of Treitz”

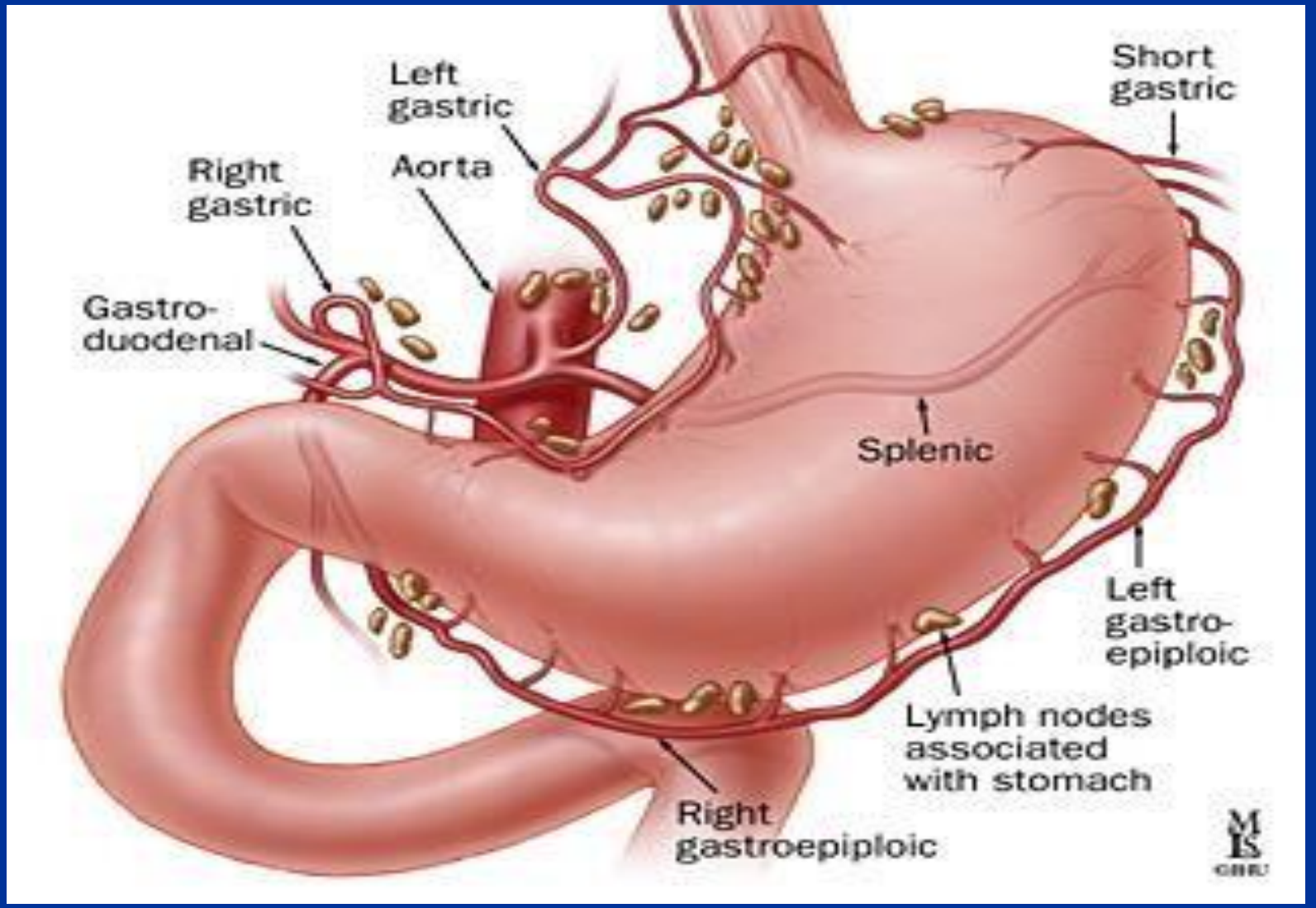
(50% of all GI Bleeding)



Ligament of Treitz is the dividing point, before ligament of Treitz is upper GI and after it is lower GI.



The big network of blood vessels to the upper GI tract has and important role in GI bleeding


Left
gastric
Short
gastric
Right
gastric
Aorta
Gastro-
duodenal
Splenic
Left
gastro-
epiploic
Lymph nodes
associated
with stomach
Right
gastroepiploic

UPPER GI BLEEDING

Signs and Symptoms

- Hematemesis
- Melena
- Dizziness
- Abd. Pain and symptoms of Peptic ulcer disease
- Hx of NSAID's use
- Pallor
- Hypotension
- Orthostasis
- Jaundice and other stigmatas of chronic liver diseases

Different combinations of symptoms and signs could indicate different etiologies of GI bleeding.

Hematemesis: Vomiting blood, either fresh blood or dark denatured blood coffee like, different from hematopoiesis: (coughing blood) which comes from respiratory system

Melena : very dark sticky blood coming from the anus after being exposed to GI tract juices, looks like tar (الزفتة).

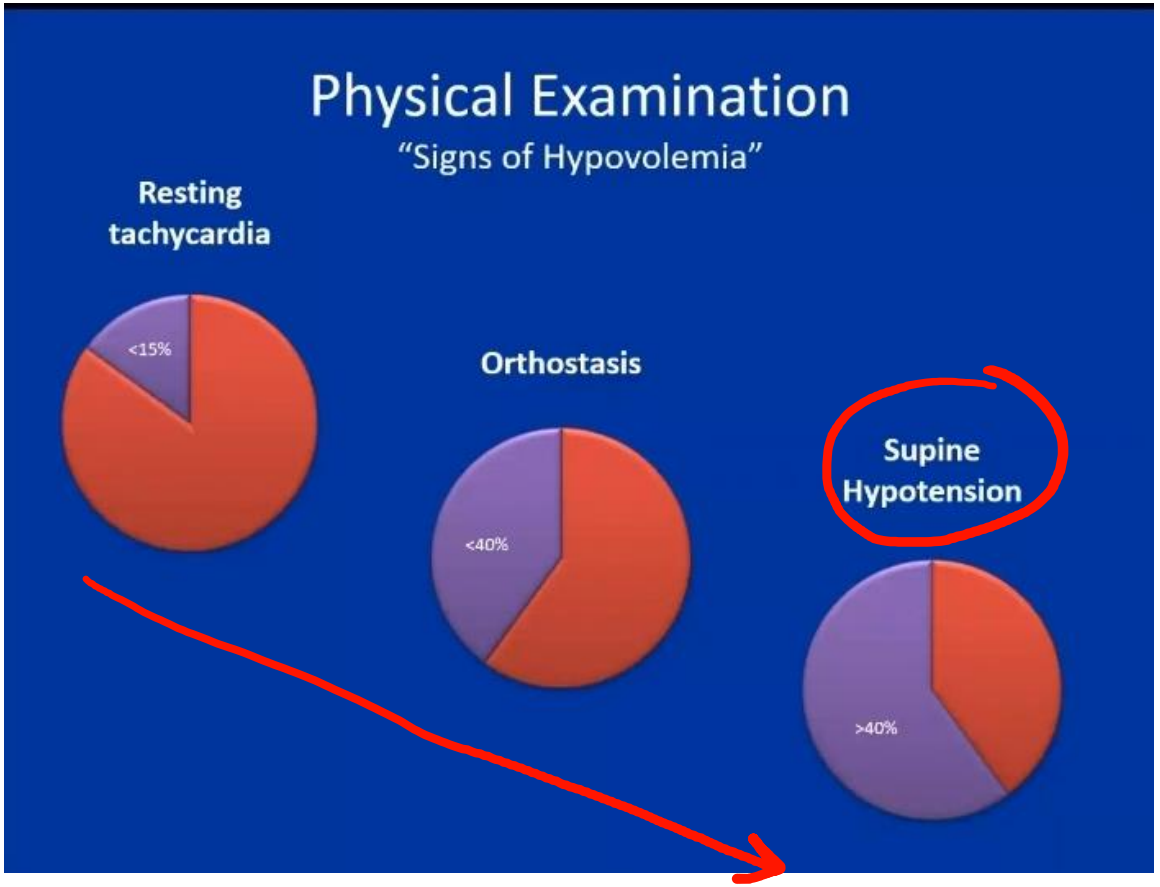
Dizziness : caused by the drop of blood volume.

Pallor (شحوب) : also because the drop in blood volume and being compensated by the body with increase in plasma.wich dilute blood

Orthostasis : the change of the posture of the patient changes the blood pressure noticeably.

Dysphagia : difficulty swallowing.

Odynophagia : pain when swallowing.



- worse
- more blood loss

- The most important thing to pay attention to in a GI bleeding patient are **signs of hypovolemia**.

1. Resting tachycardia (high heart rate), it's a mechanism made by the body to make the blood reach all organs despite having low blood volume, it's an important sign for hypovolemia.
2. Orthostasis
3. Supine hypotension, normally, making the patient go supine makes the blood pressure increase, but having hypotension despite being in supine position is a serious sign of hypovolemia.

HCO_3 & Mucus

(1)The mucosal barrier is a thick, alkaline, unstirred, aqueous layer of dissolved **bicarbonate** & **mucus**, which neutralizes the effects of gastric juice H+.

(2)(Gastric surface mucus cells) & (duodenal enterocytes & goblet cells) with a **lipid bilayer membranes** forming a barrier to H+ & **tight junctions** between cells

Two Simple mechanisms that prevent the acidity of the stomache to cause ulcers and GI bleeding are (1) HCO3 that neutralize the acidity on the inner surface of GI. And (2) the mucus lining the inner surface of GI tract.
3) Blood flow important

Blood Flow

Sub-mucosal blood flow **drains H+ away from the mucosa** & buffers H+ with plasma HCO_3 & proteins.

pH gradient changes from the gastric lumen pH of 2.0, the mucosal cell surface of pH 7.0, the mucosal cell interior pH of 7.0, and the circulating blood pH of 7.4.

Blood flow is the most important factor.
Because constant blood flow decreases the acidity of the stomach.

Blood Flow

- If HCO3 secretion ↓: when proteolysis of mucus is ↑ (as in **inflammation**),

- or when mucosal blood flow is ↓ (as with using **NSAIDs**), intracellular acidosis occurs, leading to cell necrosis.

PGs

Prostaglandins protect gastro-duodenal mucosa by **secretion of mucus**, (**PG-E2**) ⊕ **bicarbonate secretion** & **maintenance of blood flow** during periods of potential injury. Mucosal peptides and growth factors, including trefoil-family peptides and transforming growth factor alpha, also participate to ensure normal epithelial function by regulating responses to injury.

PGs

NSAIDs, which block the synthesis of prostaglandins, predispose to mucosal injury and peptic ulceration.

That's why NSAIDS cause hyperacidity therefore peptic ulcers.

Summary:.
NSAIDS BLOCK PGS synthesis leading to
→ DECREASE BLOOD FLOW
→ DECREASE MUCUS and HCO3 SECRETION
INCREASING the susceptibility of ULCERS →UGIB

The major causes for ULCERS
1.NSAIDS
2.H. PYLORI –treated by eradication

H. Pylori

Pan-Gastritis (Body + Corpus)
"Early life infection"

- Multifocal/Pan-gastric gastritis →

  Antral & Corpus Atrophy (**parietal cell loss**) + intestinal metaplasia →

  ↓HCL outputs → ↑risk for **Gastric Ulcer** & **Adeno-CaRcinoma**

- Depletion of antral somatostatin ⊖ effect→

  ↑gastrin levels.

h.Pylori could cause atrophy of the body of the stomach therefore decreased HCL, this will not cause peptic ulcer, but will cause other problems, like increased risk of adeno carcinoma.

Antral Gastritis Only (↑ %)
"Late life infection"

- Antral-predominant active chronic gastritis →

  Antral Atrophy & ↓ **antral D cells** → (Corpus- sparing) →

  ↑HCL output → ↑risk for **Doudenal Ulcer** + duodenal gastric metaplasia → HP colonize duodenum

- Depletion of antral somatostatin ⊖ effect→

  ↑gastrin levels.

Or h.pylori could cause increased HCL. Which the body of stomach can tolerate but the antrum and duodenum can not, which will cause duodenal ulcer.

UPPER GI BLEEDING

Peptic Ulcer Disease