



GI Bleeding

Surgery 2

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Gastrointestinal Bleeding

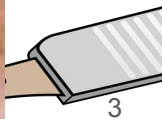
Any kind of the hemorrhage from oral cavity to anus.

- **Upper GI bleeding** (proximal to the Treitz ligament)
- **Lower GI bleeding** (distal to the Treitz ligament)

MODE OF PRESENTATION

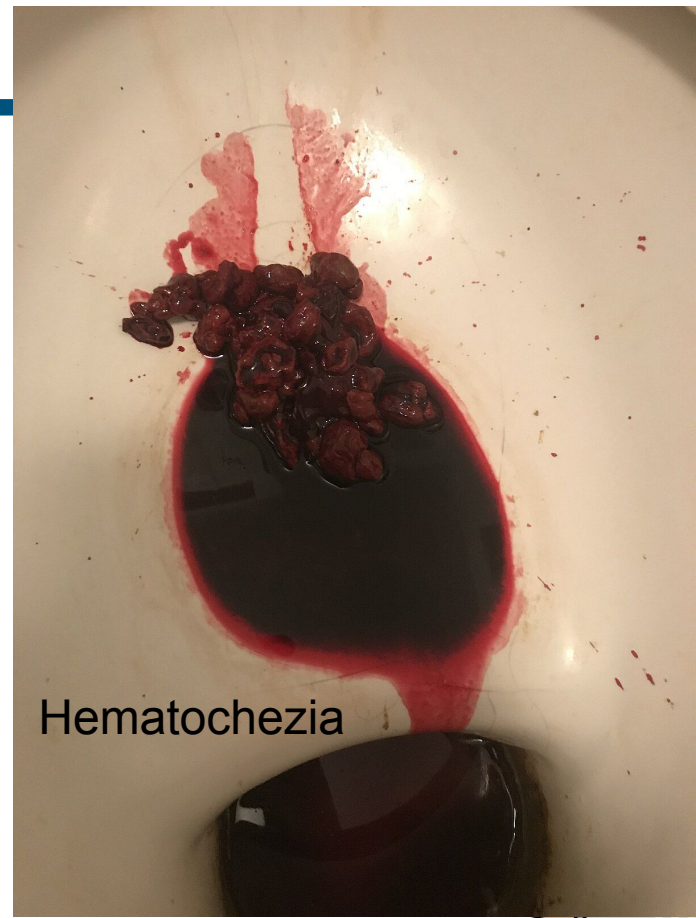
Presentation	Description	Indication
<u>Hematemesis (50%)</u>	Vomiting blood	Bright red = significant bleeding. Coffee ground = no active bleeding
<u>Melena (80%)</u>	Passage of foul smelling black stool	Presence in GIT for more than 14 h
<u>Hematochezia(20%)</u>	Bright blood per rectum	
<u>Occult bleeding</u>	No evidence of visible blood loss but +ve microscopic blood	
<u>Symptoms of blood loss or iron deficiency anemia</u>	Symptoms of anemia	Chronic bleeding

Hematemesis

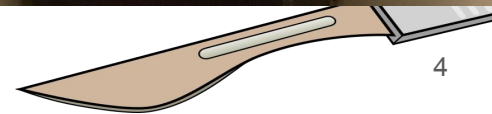




Melena

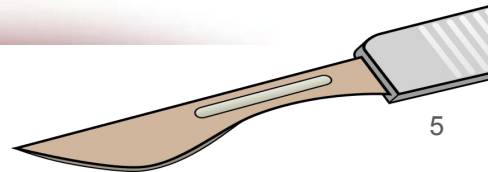


Hematochezia



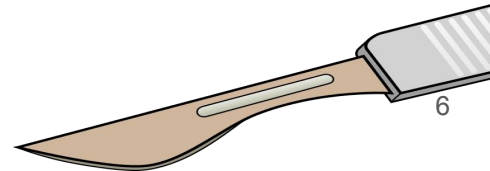
Vital signs and postural changes in heart rate and blood pressure are insensitive and nonspecific.

- Tachycardia
- Tachypnea
- Hypotension
- skin signs (e.g., color, warmth, and moisture to assess for shock, or presence of lesions such as telangiectasia, bruises, or petechiae to assess for vascular diseases or hypocoagulable states)



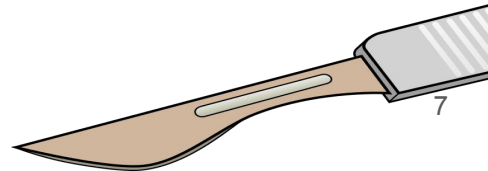
Blood Loss

- Class I hemorrhage (loss of 0-15%)
 - Only minimal tachycardia is seen. A delay in capillary refill of longer than 3 seconds corresponds to a volume loss of approximately 10%.
- Class II hemorrhage (loss of 15-30%)
 - Tachycardia (rate >100 beats per minute), tachypnea, ↓pulse pressure, cool clammy skin, delayed capillary refill, and slight anxiety. Catecholamine → ↑**diastolic BP**.
- Class III hemorrhage (loss of 30-40%)
 - Marked tachypnea and tachycardia, **decreased systolic BP**, oliguria, and significant changes in mental status, such as confusion or agitation. In patients without other injuries or fluid losses, 30-40% is the smallest amount of blood loss that consistently causes a decrease in systolic BP. Most of these patients require blood transfusions, but the decision to administer blood should be based on the initial response to fluids.
- Class IV hemorrhage (loss of >40%)
 - Marked tachycardia, ↓↓systolic BP, ↓↓pulse pressure (or immeasurable diastolic pressure), ↓↓↓urinary output, depressed mental status (or loss of consciousness), and cold and pale skin. This amount of hemorrhage is immediately life threatening.



Hypovolemic shock

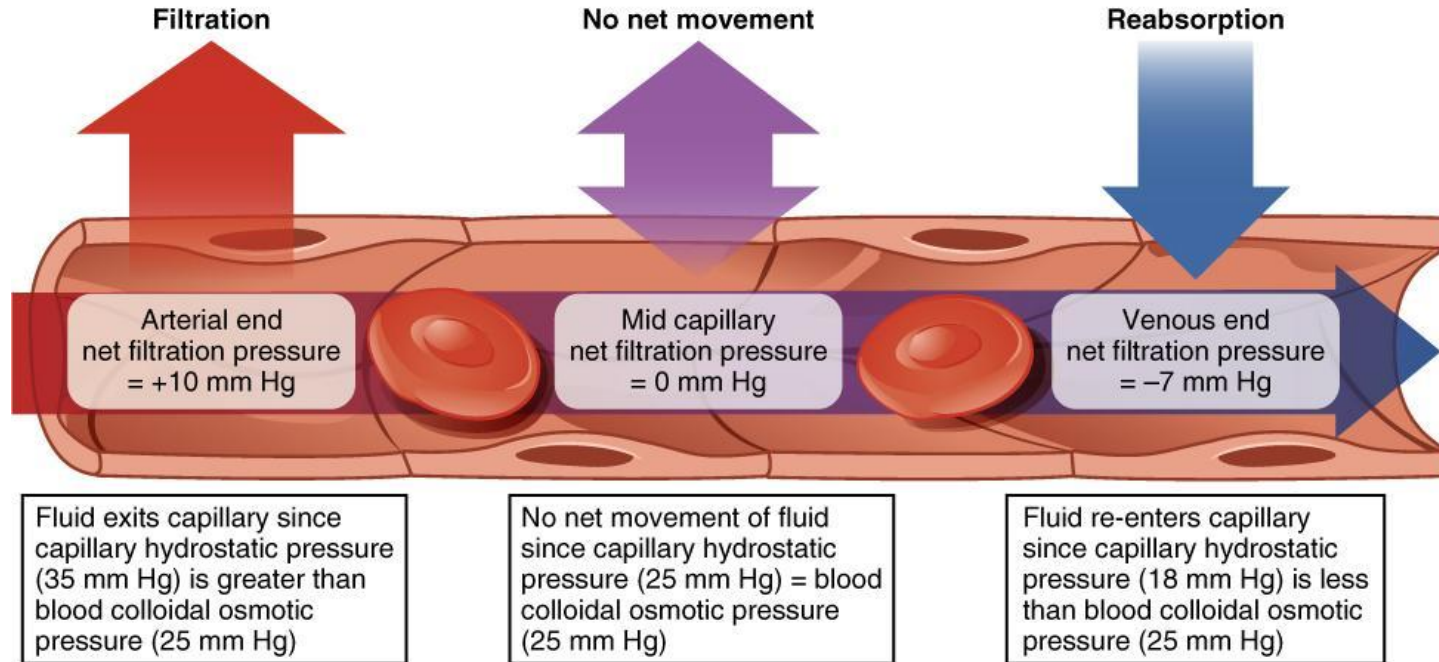
- The hematologic system - Activating the coagulation cascade
- The cardiovascular system
 - Increased release of norepinephrine
 - Baroreceptors → ↓baseline vagal tone
 - ↑heart rate, ↑myocardial contractility, and constricting peripheral blood vessels.
 - Redistributing blood to the brain, heart, and kidneys and away from skin, muscle, and GI tract.
- The renal system
 - Renin converts angiotensinogen to angiotensin I, which subsequently is converted to angiotensin II by the lungs and liver.
 - Vasoconstriction
 - Stimulation of aldosterone secretion by the adrenal cortex.
 - Aldosterone ↑sodium reabsorption and subsequent water conservation.
- The neuroendocrine system
 - Antidiuretic hormone - increases reabsorption of water and salt



What is the shock?



Capillary Exchange



Pathophysiology of hypovolemic shock

	Aerobic	Anaerobic
Reactants	Glucose and oxygen	Glucose
Combustion	Complete	Incomplete
Energy Yield	High (36 ATP)	Low (2 ATP)
Byproducts	CO₂ and H₂O	Lactic acid
<i>Location</i>	Cytoplasm and mitochondria	Cytoplasm
Stages	Glycolysis Fermentation Krebs cycle Electron transport chain	Glycolysis Fermentation

Decreased ATP



Loss of Na/K Pump



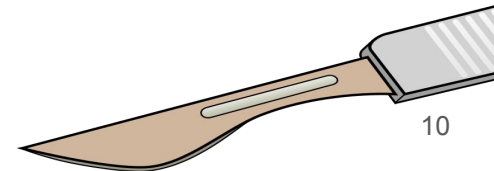
Cellular Swelling
Loss of function



Lysosomes rupture
Auto-digestion



Cell death



Stages of Shock

1. Initial Stage

Hypoperfusion

→ hypoxia → anaerobic respiration → Krebs cycle is slowed → lactic acidosis.

2. Compensatory Stage

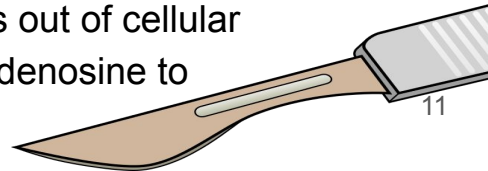
- Sympathetic Nervous System
 - Vasoconstriction, ↑HR, ↑contractility, ↓UOP
- Renin-Angiotensin-Aldosterone
 - Response to ↓BP/ ↓blood volume with ↑Na⁺
- Tissue Injury → cytokines
 - Vasodilation, vasoconstriction, ↑capillary permeability

3. Progressive Stage - Compensatory mechanisms begin to fail.

↑Metabolic acidosis, ↑precapillary sphincters relax
→ to leakage of fluid and protein into the surrounding tissues → ↑Hemoconcentration, ↑viscosity increase → blockage of the microcirculation → vital organs get compromised. Bowel ischemia → bacterial translocation → endotoxic shock.

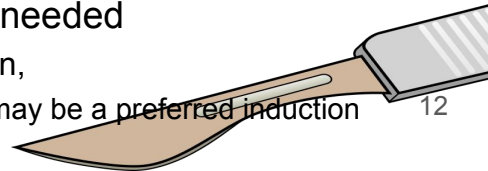
4. Refractory Stage - vital organs have failed

Brain damage and cell death
ATP has been degraded into adenosine
Adenosine easily perfuses out of cellular membranes, there is no adenosine to phosphorylate into ATP



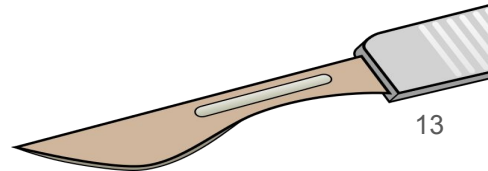
Treatment of hypovolemic shock

- Two large bore IVs (18 gauge or larger)
 - Intraosseous Access if IV Access is unavailable
- Fluid Replacement
 - Crystalloid (LR or NS)
 - Packed Red Blood Cell transfusion as indicated (replace blood loss with blood)
- Fluid Responsiveness Markers
 - See targets of adequate perfusion
 - Improved mentation
 - Improved Capillary Refill
 - Mean arterial pressure (MAP): >65 mmHg (>50 mmHg may be preferred in Trauma)
 - Urine output: >0.5 ml/kg/h
 - IVC Ultrasound for Volume Status - IVC collapses >50% on inspiration
 - Central Venous Pressure (CVP): 8-12 mmHg
 - Central Venous Oxygen Saturation
 - Assess Stroke Volume, cardiac output or surrogates before and after fluid challenge
 - Administer 250-500 cc IV crystalloid challenge (over 10 minutes)
 - Expect increased Stroke Volume >10-15% after fluid challenge
 - Consider Vasopressors if poor response after the first liter (2L in Septic Shock)
 - Passive Leg Raise Maneuver - Provides a transient autologous fluid bolus of 300-400 cc
- Vasopressors (refractory severe Hypotension)
 - Norepinephrine
 - Vasopressin
 - Epinephrine
 - Dopamine, dobutamine
- Respiratory support as needed
 - Noninvasive Ventilation,
 - Intubation - Ketamine may be a preferred induction agent in Hypotension



Some constants

- Hemoglobin concentration $<7\text{g/dL}$ (or $<8\text{dL}$ + ischemic heart disease) and/or hematocrit $<25\%$ is a trigger (indication) for blood therapy.
- After transfusion of each unit of the RBC hematocrit can be expected to increase by $\sim 3\%$ and hemoglobin level by $\sim 1\text{ mg/dL}$.
- Target urine output: $>0.5\text{ ml/kg/h}$
- Target central Venous Pressure (CVP): 8-12 mmHg



Upper GI bleeding

Upper GI Bleeding

1. Peptic ulcer disease

Most Common

Peptic ulcer	35–66%
Gastroduodenal erosions	8–15%
Esophagitis	8–15%
Varices (esophageal and gastric)	5–10%
Mallory–Weiss tear	5%

Less Common

Epistaxis, hemoptysis
Aortoenteric fistula
Boerhaave syndrome
Carcinoma
Vascular anomalies
Caustic ingestion
Anastomotic ulcer

Esophageal causes

- Esophageal varices
- Esophagitis
- Esophageal cancer
- Esophageal ulcers
- Mallory-Weiss tear

Gastric causes

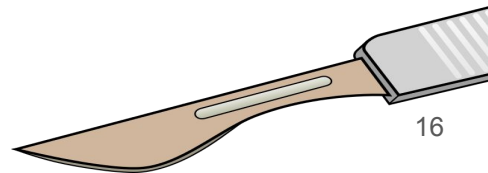
- Gastric ulcer
- Gastric cancer
- Gastritis
- Gastric varices
- Dieulafoy's lesions
- Gastric Antral Vascular Ectasia
- Portal Hypertensive Gastropathy

Duodenal causes

- Duodenal ulcer
- Vascular malformation including aorto-enteric fistulae
- Hematobilia, or bleeding from the biliary tree
- Hemosuccus pancreaticus, or bleeding from the pancreatic duct
- Severe superior mesenteric artery syndrome

Uncommon causes of the upper GI bleeding

- **Swallowed blood (nosebleed);**
- Esophageal, gastric, or intestinal neoplasm;
- Anticoagulant and fibrinolytic therapy;
- Hypertrophic gastropathy (Ménétrier's disease);
- Aortic aneurysm;
- Aortoenteric fistula (from aortic graft);
- Arteriovenous malformation;
- Telangiectases (Osler-Rendu-Weber syndrome);
- Dieulafoy lesion (ectatic submucosal vessel);
- Vasculitis;
- Connective tissue disease (pseudoxanthoma elasticum, Ehlers-Danlos syndrome);
- Blood dyscrasias;
- Neurofibroma;
- Amyloidosis;
- Hemobilia (biliary origin).



Classic Symptoms of Upper GI Bleeding

- **Hematemesis**

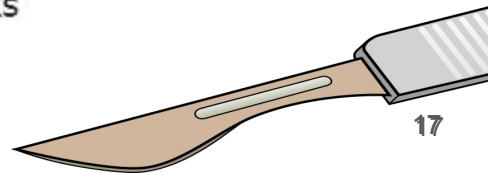
- Indicates bleeding above the ligament of Trietz
- Bright red or maroon indicates active, profuse bleeding
- "Coffee-ground" emesis is blood mixed with gastrin, causing it to turn brown

- **Hematochezia**

- Usually indicates lower GI bleeding
- Blood is a cathartic and moves rapidly through the system
- Can indicate upper GI bleeding if bleeding is massive

- **Melena**

- Blood in the stools that turns them black
- Indicates upper GI bleeding in 90% of cases
- May take several days to clear stools
- Stools will remain positive for occult blood for 1 to 2 weeks



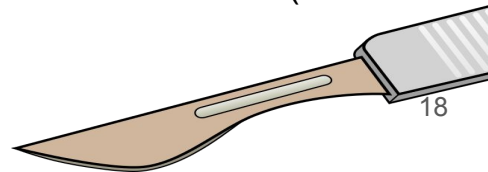
BLEEDING PEPTIC ULCER

- Melena and/or hematemesis
- Resting tachycardia (HR >100 bpm)
- hypotension (SBP <100 mmHg), or orthostatic changes (HR ↑20 bpm or a SBP ↓20 mmHg on standing).
- Hypovolemic shock
- Pain is uncommon



Approach to the bleeding patient:

1. Assess hemodynamic status
2. Blood tests:
 - a. CBC ⇒ Hb, Hc, Pt
 - b. electrolytes (including BUN and creatinine)
 - c. Coagulation panel
 - d. ABO and Rh blood type, cross-match.
3. NPO
4. Initiate resuscitation
 - a. Large bore peripheral or central venous access
 - b. Crystalloids and blood products, if indicated
 - c. oxygen.
5. Nasogastric aspiration and lavage with cold water
6. PPI – IV bolus + continuous infusion. (No H₂ blockers)
7. **Early endoscopy**



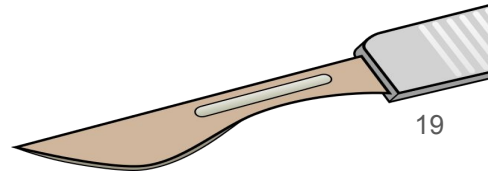
Localization of the Bleeding Site

1. MAIN METHOD

- a. Esophagogastroduodenoscopy

2. ALTERNATIVE METHODS:

- a. CT angiography→requires bleeding rate 0.3mL/min
- b. Radioisotope bleeding scan - 99mTc-labeled RBC→requires bleeding rate >0.05 mL/min
- c. Arteriography: requires bleeding rate >0.5 mL/min→defines site of bleeding or abnormal vasculature.





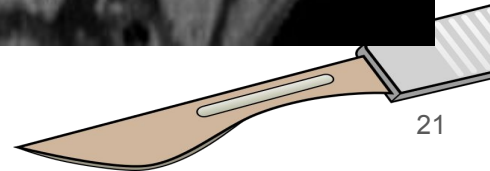
CT angiography: Bleeding from the first portion of the duodenum

Figure 2 Male, 65-year-old patient presenting with hematemesis. CT angiography in the arterial phase (A) demonstrating active contrast medium extravasation (arrow) in the first portion of the duodenum, which enhances in the portal phase (B).



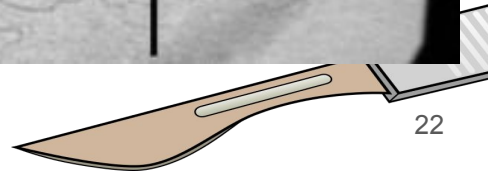
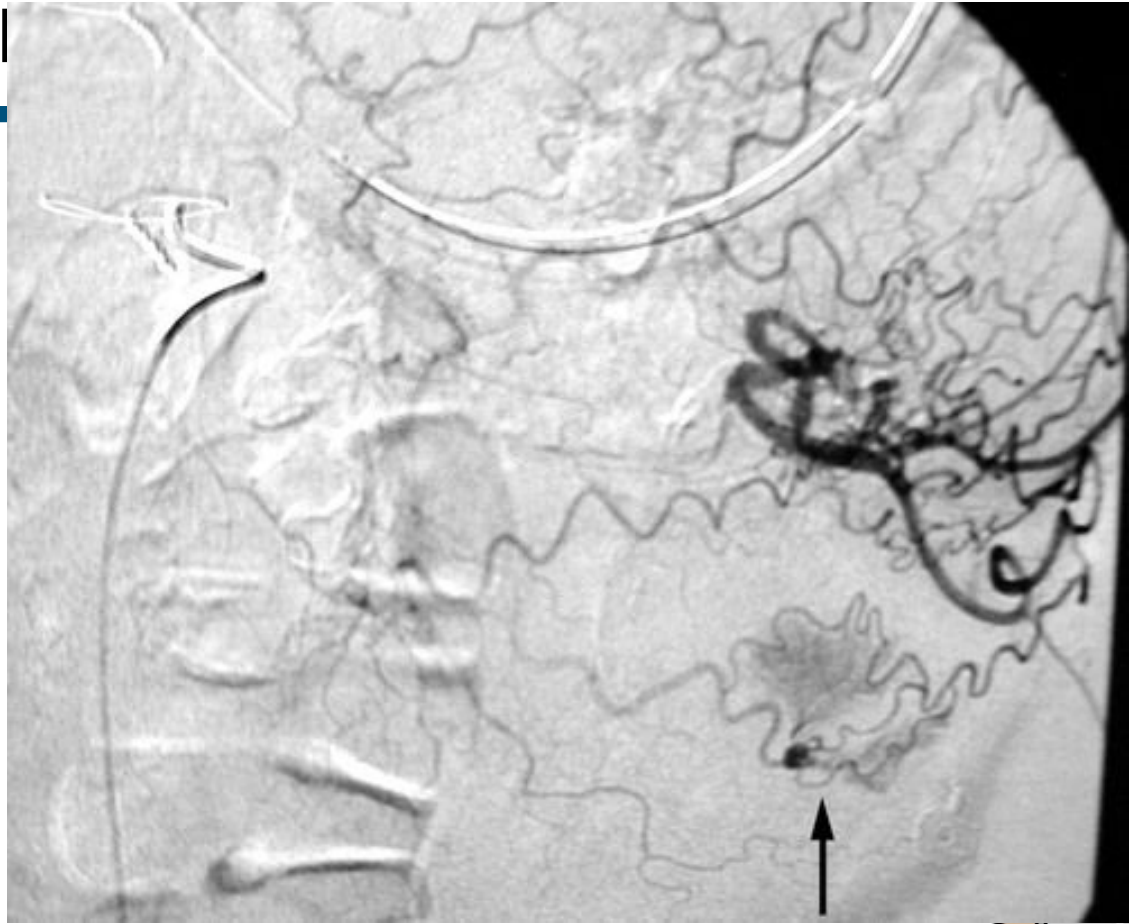
CTA

Coronal CT angiogram through the gastric body and antrum shows a large amount of clotted blood within the distended stomach (*) and pooling of extravasated contrast material (arrow).



Angiograph

Angiogram obtained after injection of the left gastric artery shows a bleeding gastric branch (arrow).

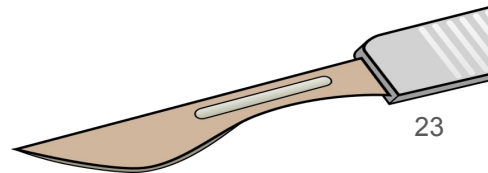


Assessment for Upper GI Bleeding

- Past medical history
 - History of GI bleeds
 - 60% of GI bleeds rebleed at same site
 - Epigastric pain, dyspepsia
 - Underlying medical conditions
 - History of alcohol use
 - Vomiting, retching or coughing
- Physical exam
 - Change in CV status
 - Signs/symptoms of shock or hypovolemia
 - Mental status changes
 - Board-like rigid abdomen - peritonitis

Laboratory Studies to Confirm Upper GI Bleeding

- Hemoglobin and hematocrit levels (drop)
- WBCs and glucose (elevated)
- Hypokalemia
- Hypernatremia
- BUN (elevated)
- PT/PTT (elevated if liver dysfunction)



BLEEDING PEPTIC ULCER

FORREST - classification of upper gastrointestinal hemorrhage

Acute hemorrhage

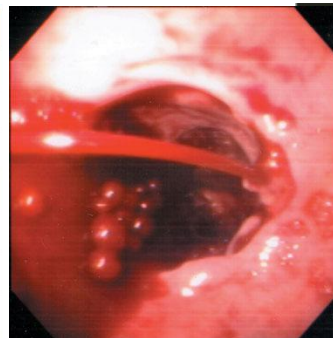
Forrest IA	Active spurting hemorrhage
Forrest IB	Oozing hemorrhage

Signs of recent hemorrhage

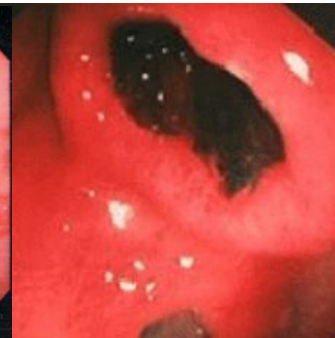
Forrest IIA	Non-bleeding visible vessel
Forrest IIB	Adherent clot
Forrest IIC	Hematin on ulcer base

Lesions without active bleeding

Forrest III	Clean-base ulcers
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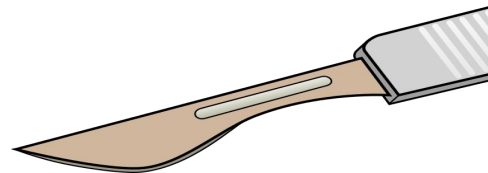


Spurt blood (grade IA)



Adherent clot (grade IIB)

- **High-risk** — active bleeding or non bleeding visible vessel (Forrest grade IA, IB, or IIA)
- **Medium-risk** — adherent clot (Forrest grade IIB)
- **Low-risk** — flat, pigmented spot or clean base (Forrest grade IIC or III)



BLEEDING PEPTIC ULCER

1. Nonsurgical treatment: $\frac{3}{4}$

of the patients stop bleeding if given acid suppression and NPO

2. Endoscopic therapy

- i. Cauterization
- ii. Epinephrine injection
- iii. Clip application
- iv. Laser
- b. Planned second-look endoscopy in 24 hrs
- c. In case of failure→Repeat endoscopy

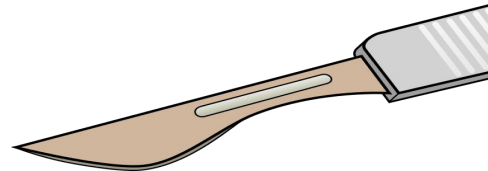
3. Transcatheter embolization

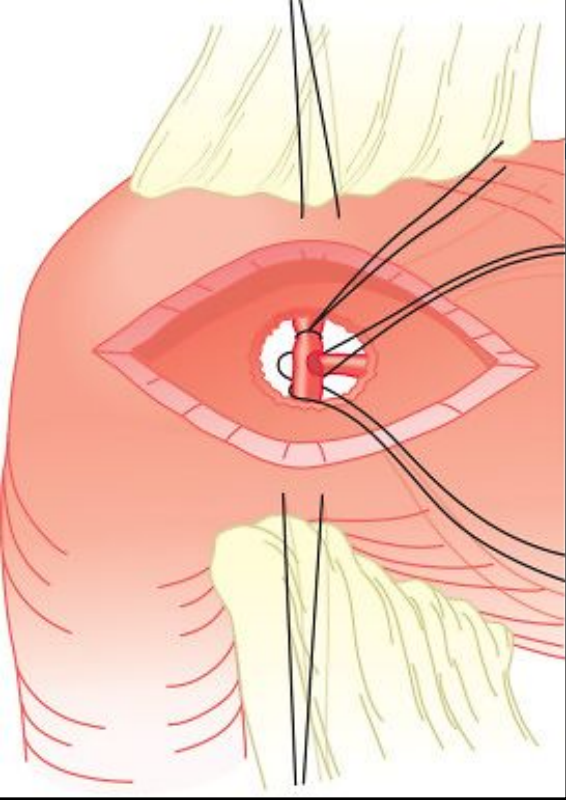
- a. in patients in whom endoscopic therapy has failed, HIGH-RISK SURGICAL CANDIDATES.
- b. Success rate – 52-94%.

4. Surgery

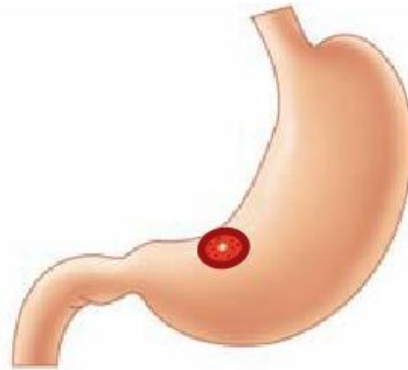
- a. Gastrotomy/Duodenotomy (opening stomach or duodenum) and bleeding ulcer oversewing
- b. Partial gastrectomy (Bleeding ulcer must be removed)

25

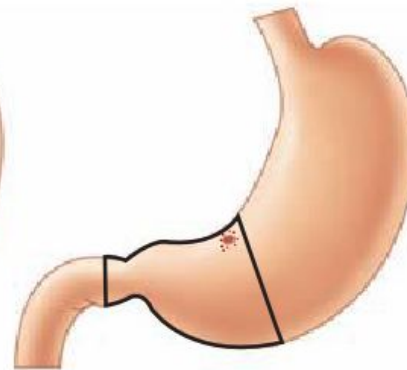




Duodenotomy
and bleeding
ulcer oversewing



Ulcer excision

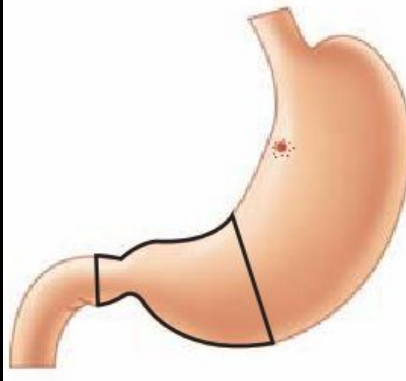


Antrectomy

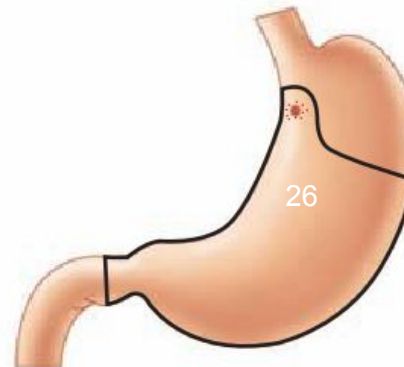


Pauchet procedure

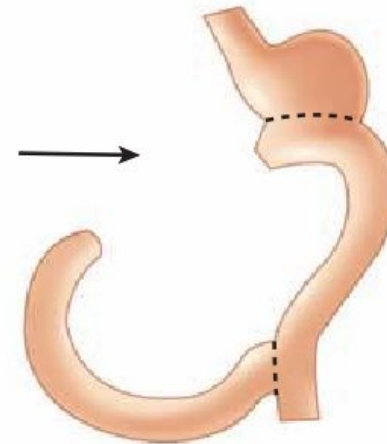
Distal partial gastrectomy
(Bleeding ulcer must be removed)



Kelling-Madlener
procedure



Subtotal gastrectomy
Roux-en-Y esophagojejunostomy

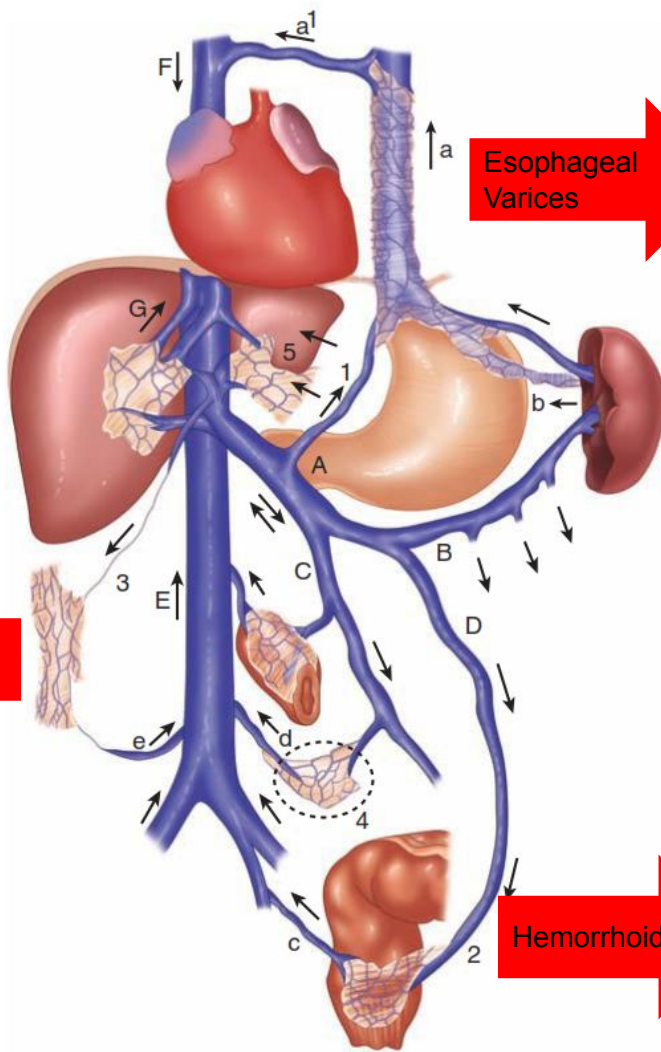


Csendes procedure

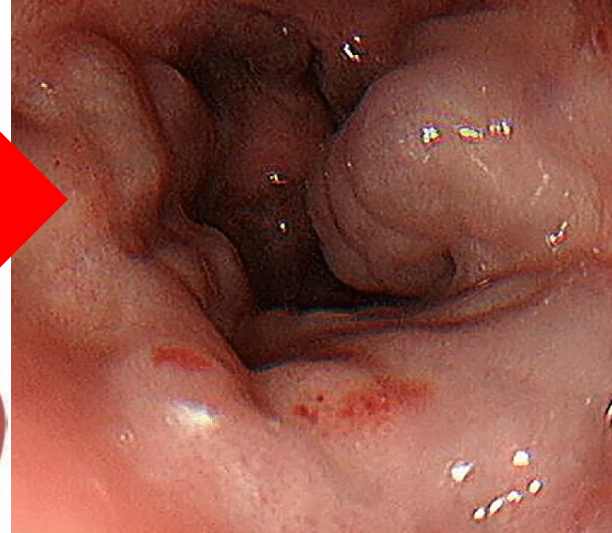
Variceal Hemorrhage



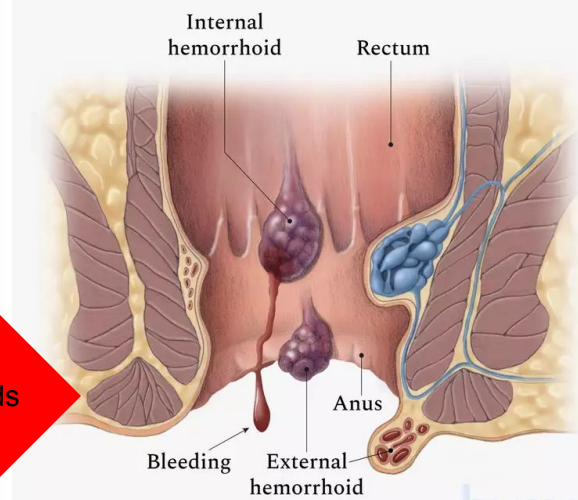
Caput Medusae



Esophageal Varices

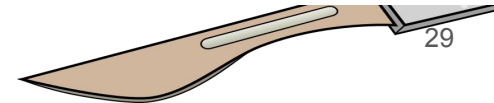
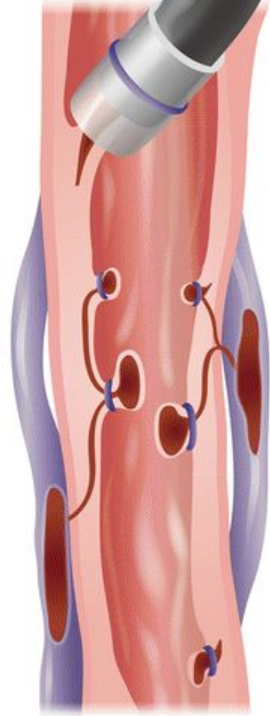
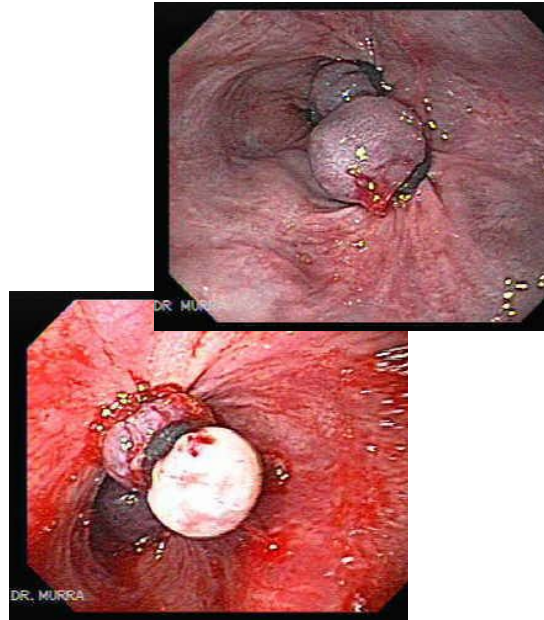


Hemorrhoids

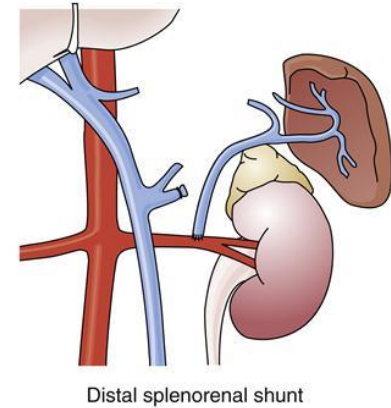
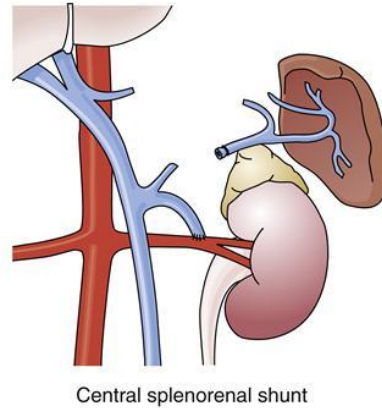
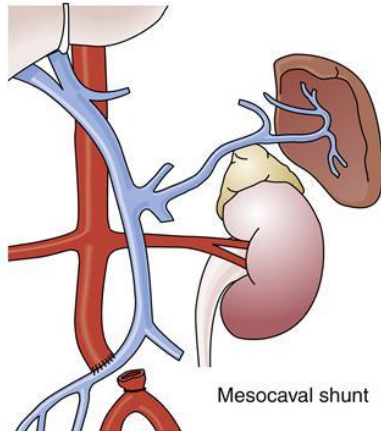
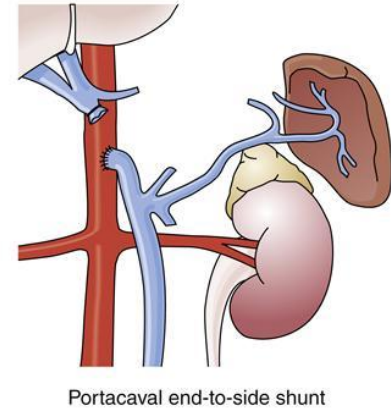
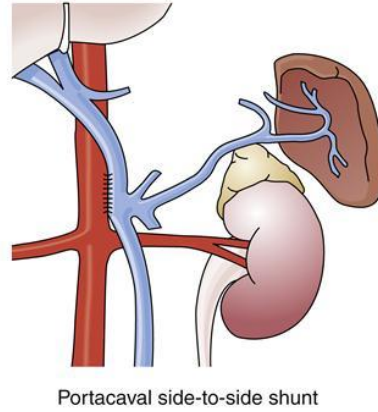
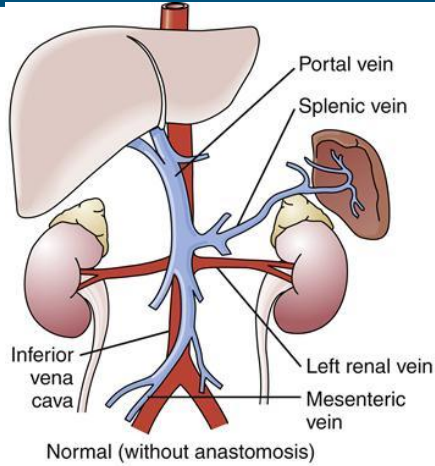


Prevention of Variceal Bleeding

- Nonselective β -blockers
- Endoscopic surveillance with variceal band ligation



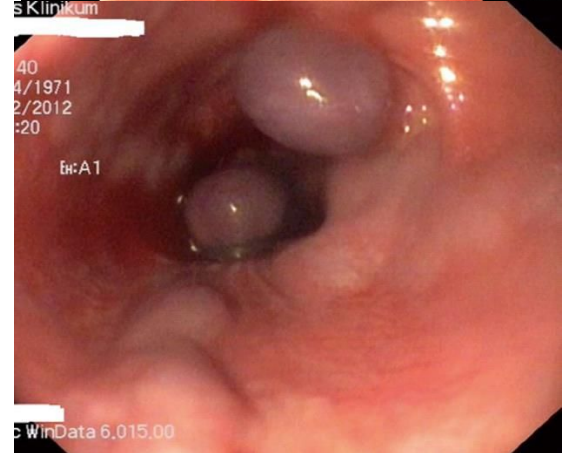
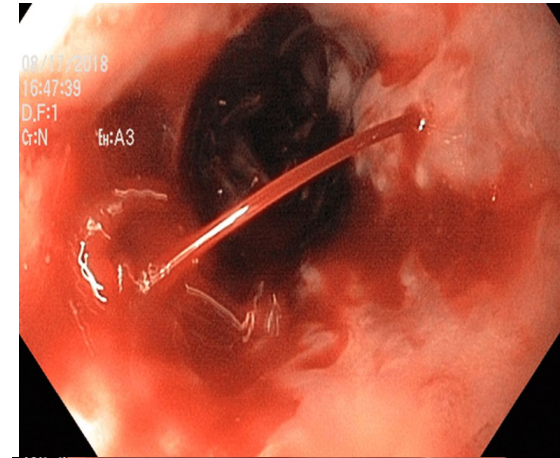
Prevention of Variceal Bleeding

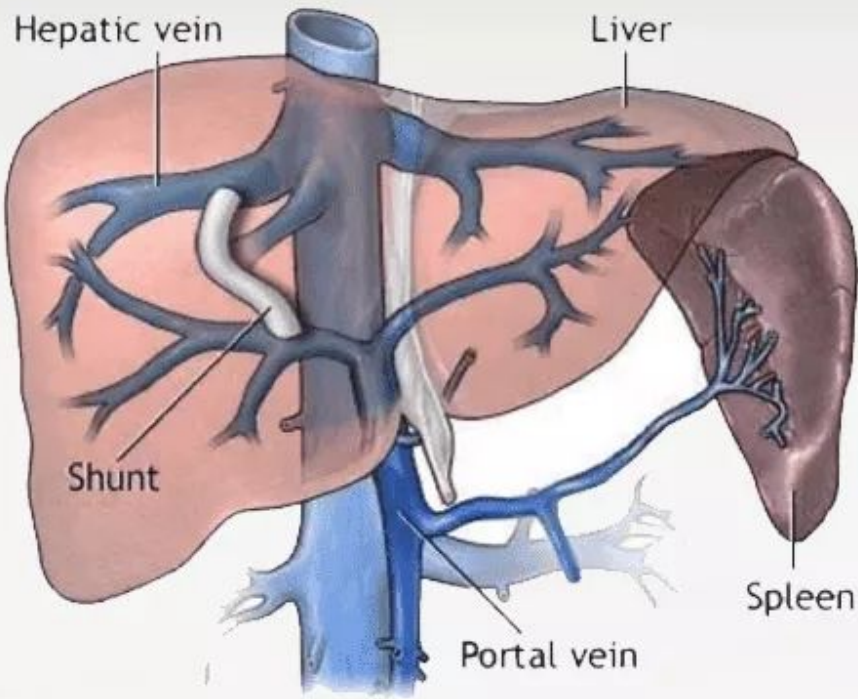


Treatment of Variceal Bleeding

Management of Acute Variceal Hemorrhage

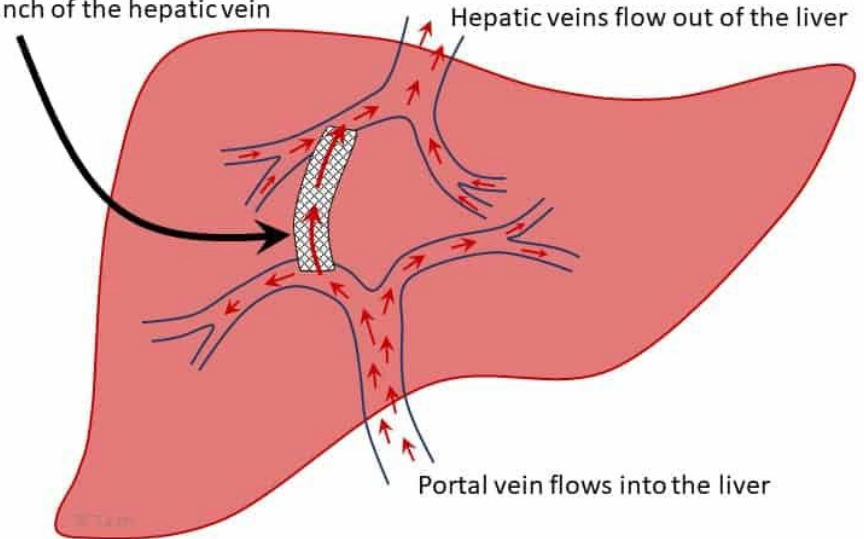
1. Should be admitted to an ICU, resuscitation, CBC, coagulation panel, ABO and rhesus typing
2. **Decrease portal pressure:**
 - Octreotide, a somatostatin analog
 - Vasopressin
3. **ENDOSCOPY: Band ligation of varices**
4. **TIPS - Transjugular Intrahepatic Portosystemic Shunt**
 - Intra-abdominal bleeding or hemobilia
 - Hepatic encephalopathy
 - High rate of thrombosis
5. Balloon tamponade using a **Sengstaken-Blakemore tube** (should not exceed 36 hours to avoid tissue necrosis)
6. **Sugiura Procedure**



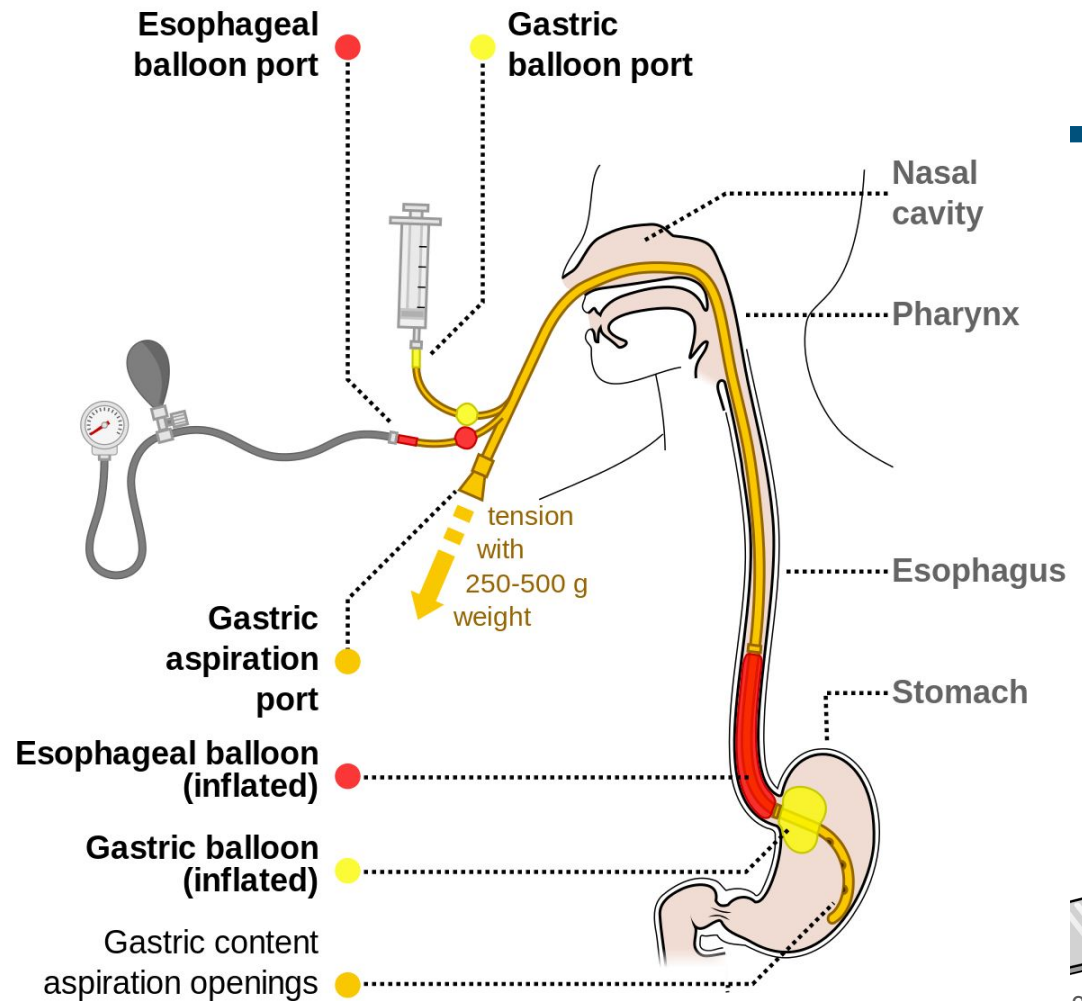


TIPS

Connects a branch of the portal vein to
a branch of the hepatic vein



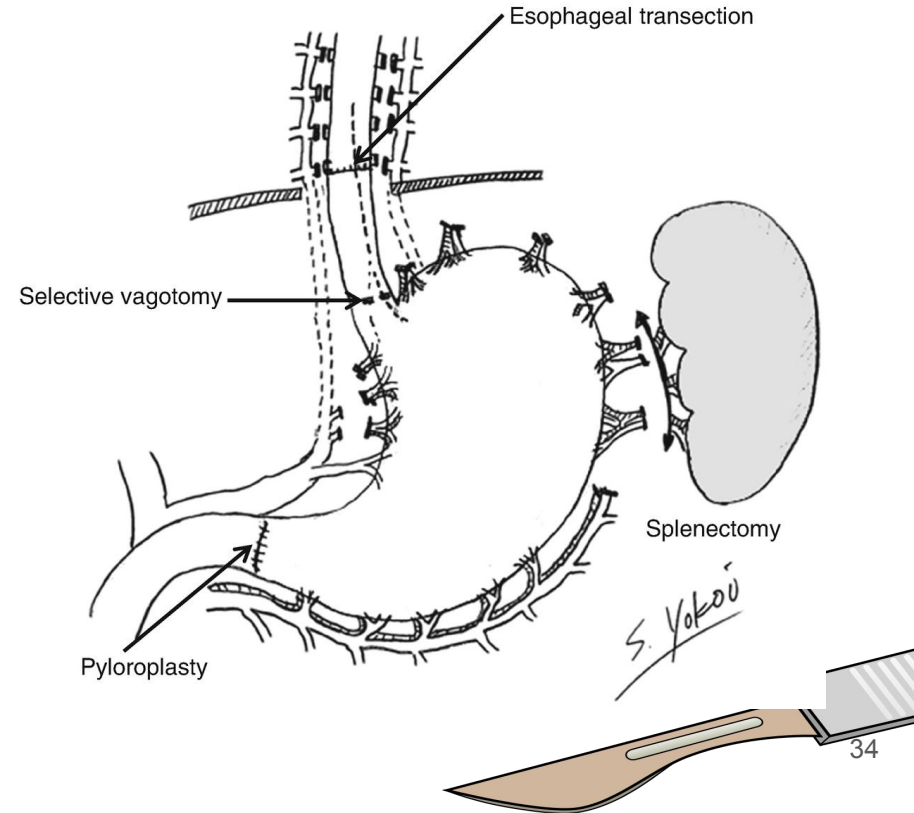
Sengstaken-Blakemore tube



Treatment of Variceal Bleeding

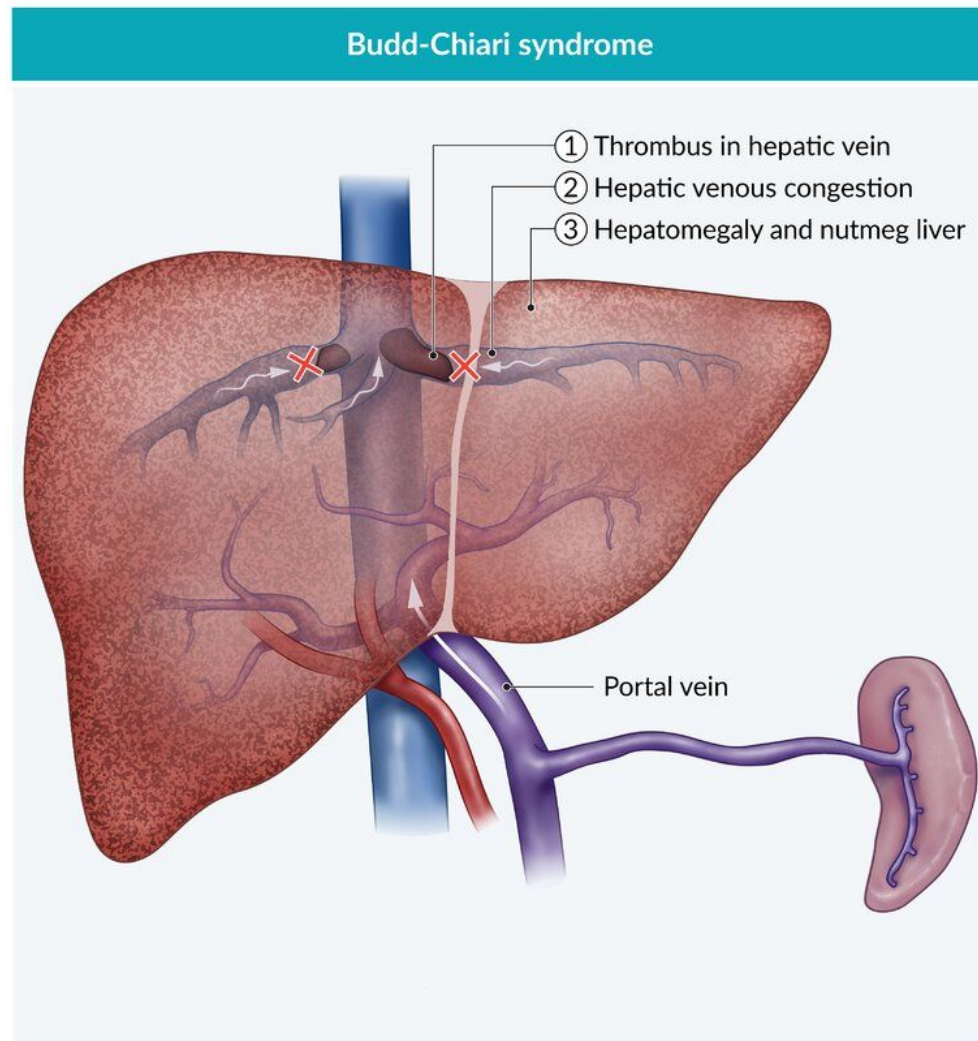
- The Sugiura procedure

- Devascularization of the stomach and distal esophagus
- Transection of the esophagus
- Splenectomy
- Truncal vagotomy
- Pyloroplasty.



Budd-Chiari syndrome

- Budd-Chiari syndrome (BCS) is an uncommon congestive hepatopathy characterized by the obstruction of hepatic venous outflow.
- Obstruction of two or more of the major hepatic veins.
- Hepatomegaly, liver congestion, and right upper quadrant pain, chronic portal hypertension and ascites.
- Caudate lobe hypertrophy occurs in approximately 50% of cases
- Thrombolytic therapy
- Surgical shunting



Budd Chiari Syndrome

In all cases

Medical therapy

- Lifelong anticoagulation
- Treat procoagulant condition as necessary
- Hepatocellular carcinoma surveillance

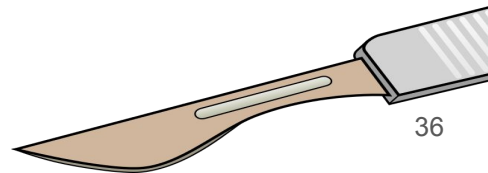
Fulminant presentation
Diffuse HV obstruction
Rotterdam class III

Transjugular intrahepatic portosystemic stent-shunt*

Symptomatic portal hypertension

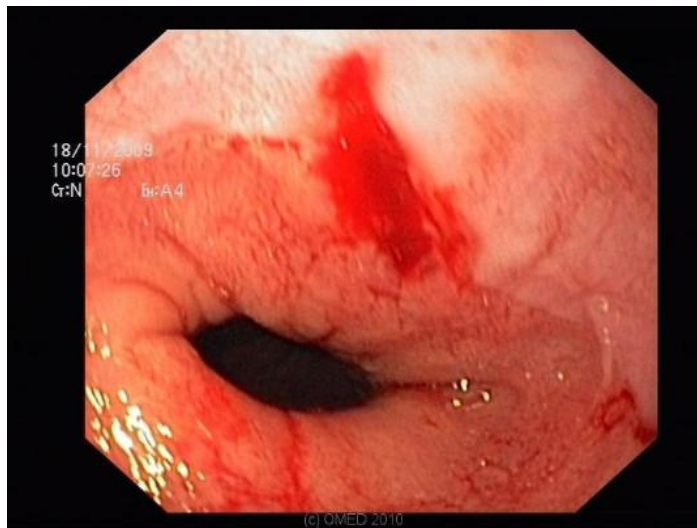
Hepatic vein (HV) interventions if feasible with dilatation and stenting

Failure of HV interventions

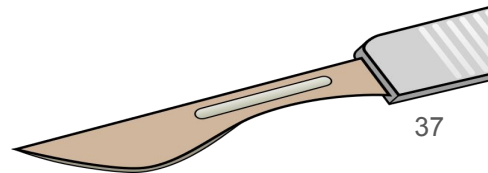


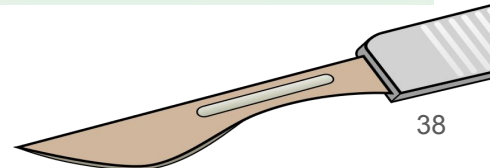
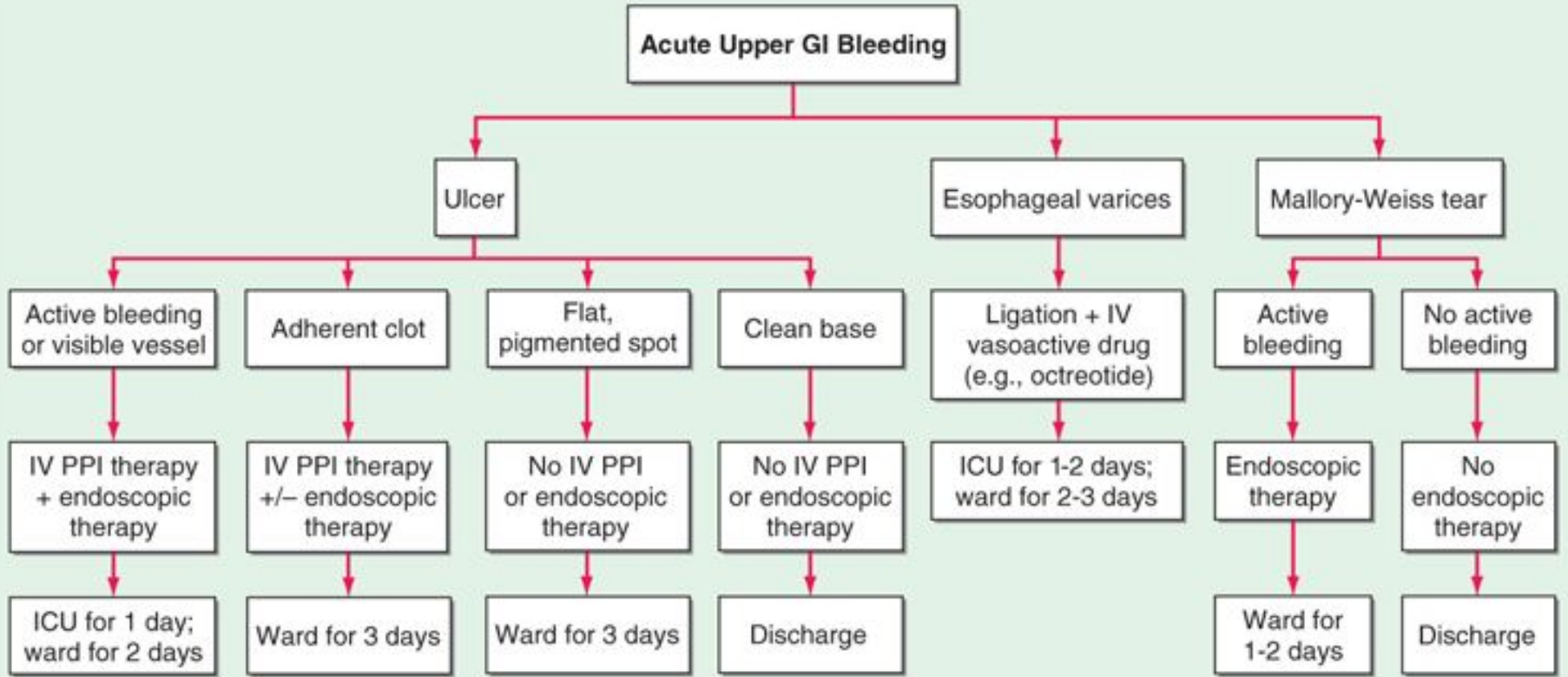
MALLORY-WEISS SYNDROME

- GI bleeding from mucosal tears at the GEJ
- Arterial bleeding, which may be massive
- Upper endoscopy



- Sengstaken-Blakemore tube will not stop the bleeding, as the pressure in the balloon is not sufficient to overcome arterial pressure.
- Endoscopic injection of epinephrine
- Bleeding often stops spontaneously.
- Laparotomy, gastrotomy with oversewing of the linear tear





Lower GI bleeding

Causes of the lower GI bleeding

	Causes	Frequency
Colorectal (80%)	Diverticular disease	5.2-42%
	Angiodysplasia	1.2-4%
	Neoplasm	2.9.19%
	Inflammatory bowel disease	
	Ischemic colitis	7-18%
	Infectious colitis	2.6%
	Radiation proctitis	9-13%
	Anorectal disease (haemorrhoids, fissurae)	20%
	Post-polypectomy / post-anastomotic bleeding	0-12.8%
Small bowel source (10%)	AV malformations	
	Meckel diverticulum	
	IBD	
	Neoplasia	
UGI source (10%)	Vasculitis	
	Ulcer	
	Neoplasm	

Table 1. Causes of Acute Lower Gastrointestinal Bleeding in Adults.*

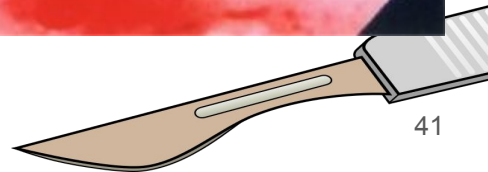
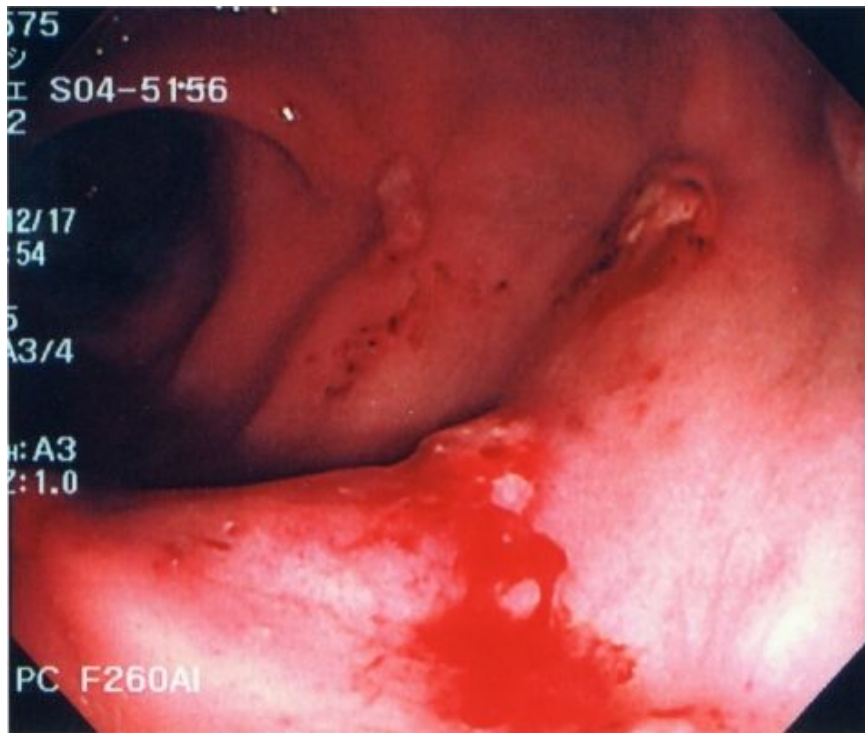
Cause	Percentage of Cases
Diverticulosis	30–65
Ischemic colitis	5–20
Hemorrhoids	5–20
Colorectal polyps or neoplasms	2–15
Angioectasias	5–10
Postpolypectomy bleeding	2–7
Inflammatory bowel disease	3–5
Infectious colitis	2–5
Stercoral ulceration	0–5
Colorectal varices	0–3
Radiation proctopathy	0–2
NSAID-induced colopathy	0–2
Dieulafoy's lesion	Rare

* NSAID denotes nonsteroidal antiinflammatory drug. Adapted from Strate and Naumann.¹⁷

Treatment

Approach to the bleeding patient:

1. Assess hemodynamic status
2. Blood tests:
 - a. CBC ⇨ Hb, Hc, Pt
 - b. electrolytes (including BUN and creatinine)
 - c. Coagulation panel
 - d. ABO and Rh blood type, cross-match.
3. NPO
4. Initiate resuscitation
 - a. Large bore peripheral or central venous access
 - b. Crystalloids and blood products, if indicated
 - c. oxygen.
5. Nasogastric aspiration and lavage with cold water
6. **Early endoscopy**



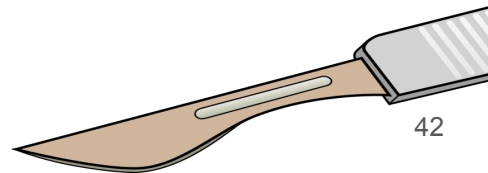
Localization of the Bleeding Site

1. MAIN METHOD

a. Colonoscopy

2. If colonoscopy fails = BLEEDING OF OBSCURE ORIGIN:

- a. In about 10% of patients presenting with lower gastrointestinal bleeding (LGIB), the source of bleeding is from the upper gastrointestinal tract ⇨ Esophagogastroduodenoscopy!
- b. CT angiography → requires bleeding rate 0.3mL/min
- c. Radioisotope bleeding scan - 99mTc- labeled RBC → requires bleeding rate >0.05 mL/min
- d. Arteriography: requires bleeding rate >0.5 mL/min → defines site of bleeding or abnormal vasculature.
- e. Often small-bowel source.
 - i. Consider small-bowel enteroclysis x-ray (careful barium radiography via peroral intubation of small bowel),
 - ii. Meckel's scan (Technetium-99m pertechnetate scintigraphy)
 - iii. Enteroscopy (small-bowel endoscopy)
 - iv. Exploratory laparotomy with intraoperative enteroscopy.



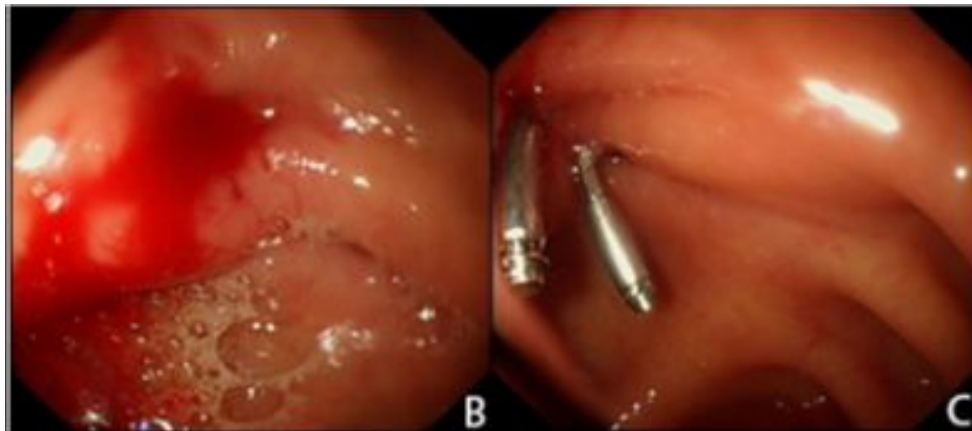
Therapeutic Colonoscopy.

Therapeutic Colonoscopy (main treatment):

- In cases of diverticular bleeding:
 - Bipolar probe coagulation
 - Epinephrine injection
 - Sclerosing agent injection
 - Metallic clips.
- Radiation-induced bleeding:
 - Topical application of formalin
 - Nd:YAG (neodymium-doped yttrium aluminium garnet) laser therapy
 - Argon plasma coagulation.
- Bleeding due to polyps ⇒ polypectomy.

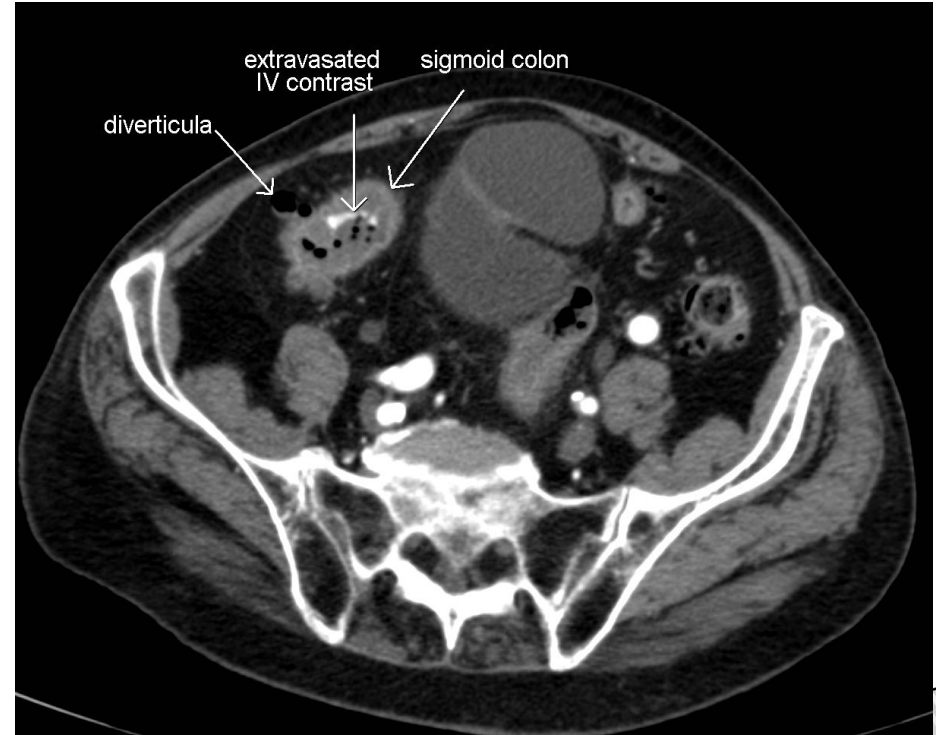
If endoscopic therapy fails:

1. Vasoconstrictive Therapy – Intra-arterial **vasopressin infusions**.
2. Superselective transarterial Embolization.
3. Emergent Surgery.



CT Angiogram (CTA)

- First line radiological modality to assess actively bleeding,
- Hemodynamically stable patients.
- A triphasic scan
- Oral contrast is not recommended as it increases anaesthetic risk and may obscure extravasated contrast
- Requires bleeding rate $>0.3\text{mL/min}$
- Sensitivity of 85.2%
- Specificity of 92.1%





[a] Diverticular hemorrhage on arterial-phase coronal image

[b] Reformatted image from same CT scan shows vascular anatomy leading to lesion (arrowhead). This could facilitate precisely targeted invasive angiography.



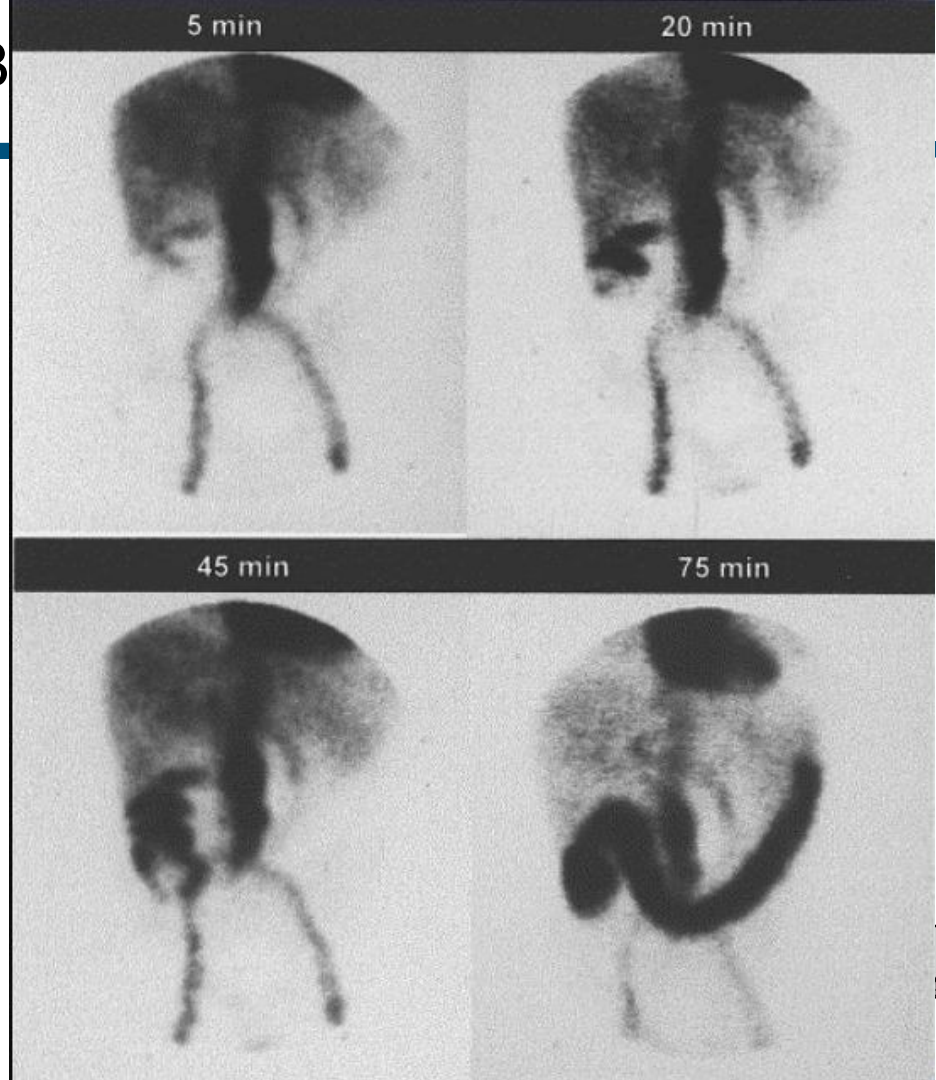
CT angiogram obtained immediately after a demonstrates pooling of contrast material within the terminal ileum (arrow).



Catheter angiogram shows active bleeding from an ileocolic branch of the SMA and pooling of contrast material within the terminal ileum (arrow)

^{99m}Tc -labeled RB

Bleeding from the hepatic flexure



Meckel's scan

^{99m}Tc -pertechnetate Meckel's scan (anterior view) in patient with gastrointestinal bleeding.



Surgery

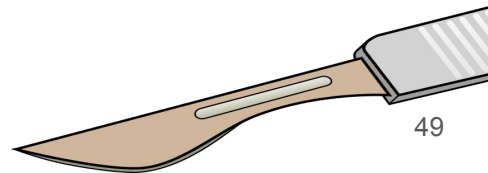
Indications:

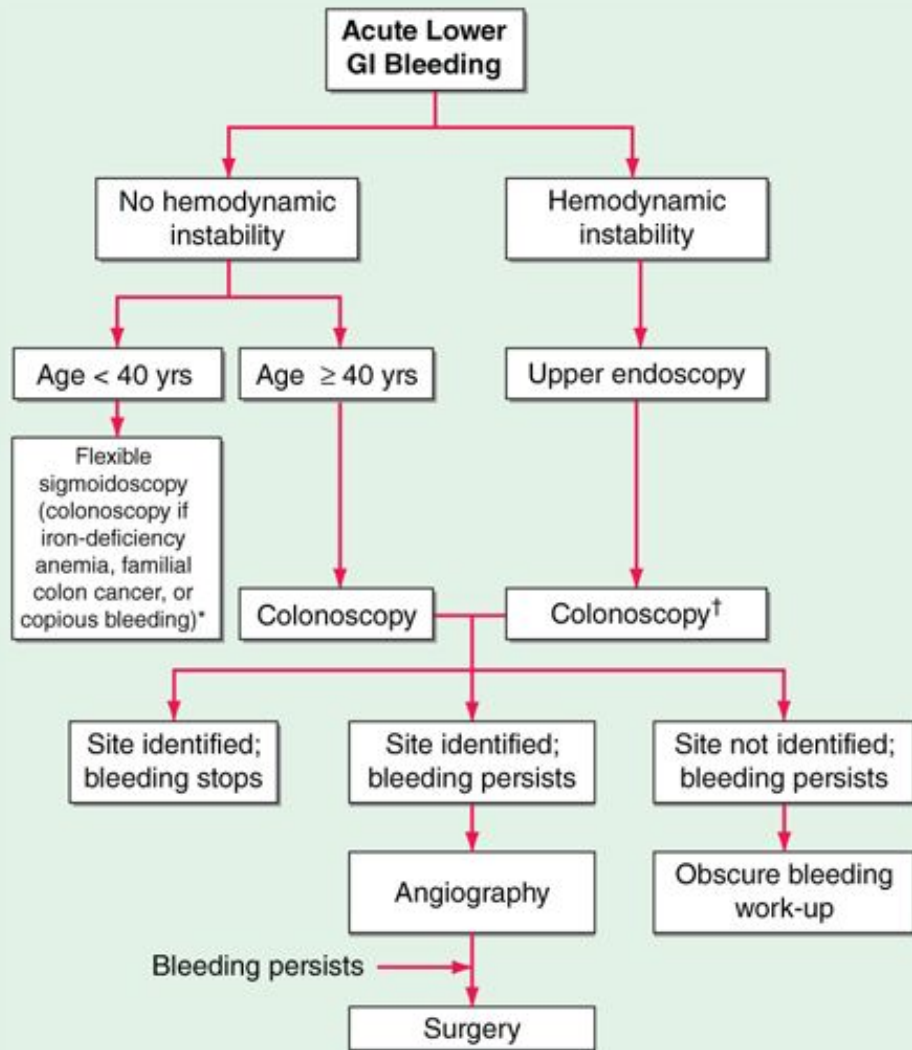
- Persistent hemodynamic instability with active bleeding
- Persistent, recurrent bleeding
- Transfusion of more than 4 units packed red blood cells in a 24-hour period, with active or recurrent bleeding
- Patients diagnosed with colonic tumors

Precise localization of the bleeding point → **Segmental bowel resection**

Active bleeding from an unknown source → **Subtotal colectomy**

Blind segmental resection should not be performed because of a prohibitively high rate of rebleeding up to 75%, morbidity up to 83%, and mortality up to 60%.





Cardiac Arrest

