UPPER AND LOWER GASTROINTESTINAL BLEEDING

Prof. G. Zuliani

Gastrointestinal Bleeding

- **Hematemesis:** vomiting of bright red blood
  - usually represents bleeding proximal to the ligament of Treitz
- **Hematochezia:** bright red blood per rectum; indicates a lower GI source of bleeding
- NOTE: blood has a laxative effect; so with upper massive bleeding the stool may be bright red

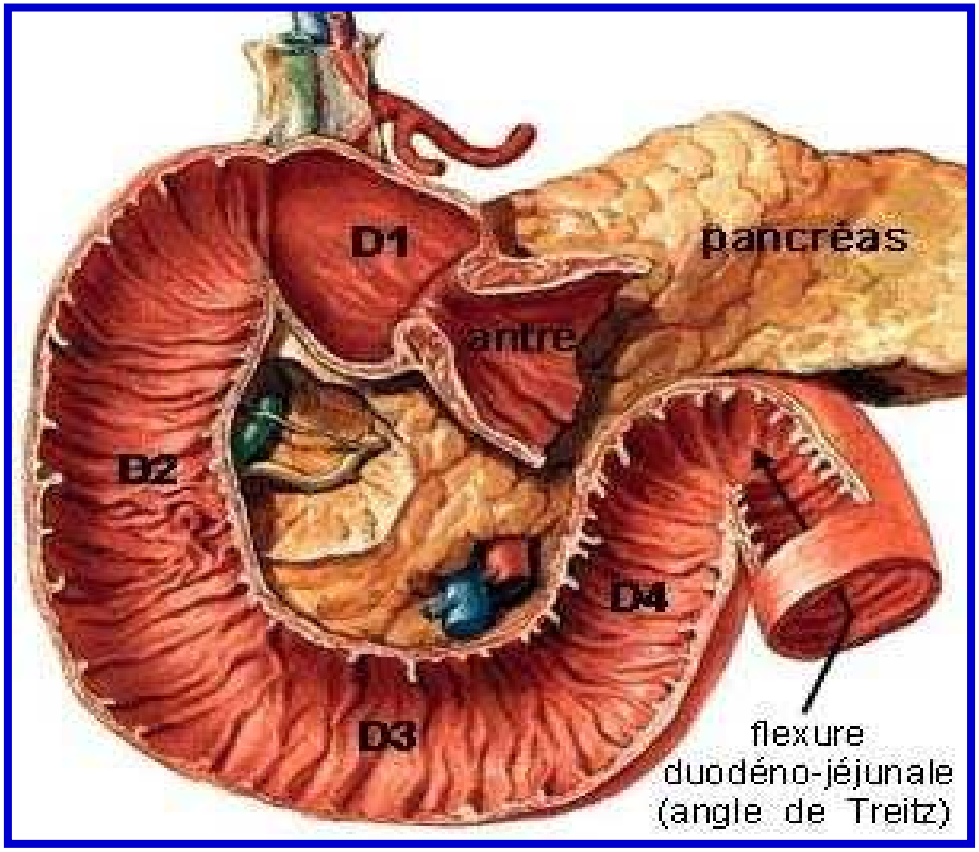
Incidence of GIB

- Upper GI bleed: about 100/100.000
  - Above the ligament of Treitz
- Lower GI Bleed: about 20/100.000
  - Below the ligament of Treitz
- Both are more common in ***Males*** and in the ***Elderly.***

1. UPPER GASTROINTESTINAL BLEEDING

Hematemesis

- GI bleeding derived from any source proximal to the Ligament of Treitz



About 1/1000 in USA experienced upper GI bleeding

Men : women = 2:1
Mortality rate = 10%!

DEFINITIONS

Upper and lower gastrointestinal bleeding

Upper gastrointestinal bleeding (or haemorrhage) is that originating proximal to the ligament of Treitz; in practice from the oesophagus, stomach and duodenum. Lower gastrointestinal bleeding is that originating from the small bowel and colon. This guideline focuses upon upper GI and colonic bleeding since acute small bowel bleeding is uncommon.

Haematemesis *(and coffee-ground vomitus)*

Haematemesis is vomiting of blood from the upper gastrointestinal tract or occasionally after swallowing blood from a source in the nasopharynx. Bright red haematemesis usually implies active haemorrhage from the oesophagus, stomach or duodenum. This can lead to circulatory collapse and constitutes a major medical emergency. Patients presenting with haematemesis have a higher mortality than those presenting with melaena alone.[2]

Coffee-ground vomitus refers to the vomiting of black material which is assumed to be blood. Its presence implies that bleeding has ceased or has been relatively modest.

Melaena

Melaena is the passage of black tarry stools usually due to acute upper gastrointestinal bleeding but occasionally from bleeding within the small bowel or right side of the colon.

Causes of Upper GIB

- **Common**
- Peptic Ulcer
- Erosive Esophagitis
- Gastritis (H. pylori)
- Mallory-Weiss syndrome
- Nasopharyngeal bleeding

- **Less Common**
- Esophageal varices
- Foreign body
- Bleeding disorders
- Vascular malformation
- Tube trauma

Table 6: Major causes of upper gastrointestinal bleeding

Cause of bleeding	**Relative frequency** *(% of those in whom any abnormality was identified at endoscopy)*
Peptic ulcer	44
Oesophagitis	28
Gastritis/erosions	26
Erosive duodenitis	15
Varices	13
Portal hypertensive gastropathy	7
Malignancy	5
Mallory Weiss tear	5
Vascular malformation	3

NB. In approximately 20% of patients presenting with apparent acute upper gastrointestinal bleeding endoscopy does not reveal a cause.

Causes of Upper GI Bleed

1) **Peptic ulcer disease**: is the most common cause
   A) ***duodenal ulcer***
      - will re-bleed in 10% of cases within 24-48h
   B) ***gastric ulcer***
      - more likely to re-bleed
   C) ***stomal ulcer*** <5%

Causes of Upper GI Bleed

2) **Erosive gastritis, esophagitis, duodenitis**

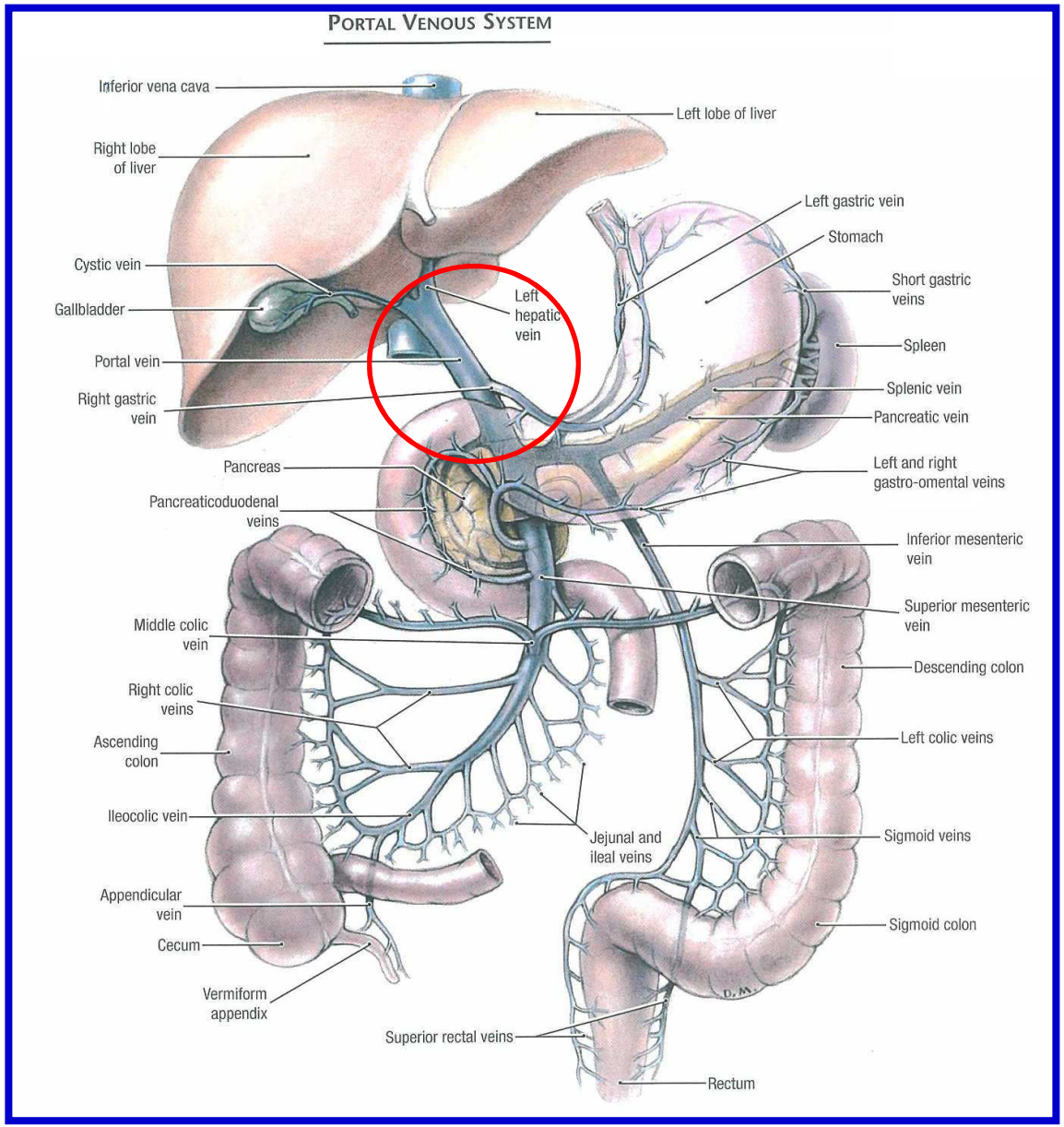
- some causes are: *Alcohol, ASA, NSAIDs*

3) **Esophageal and gastric varices**

- causes by *portal hypertension*

4) **Mallory-Weiss syndrome**: longitudinal mucosal tear in the cardio-esophageal region caused by repeated retching

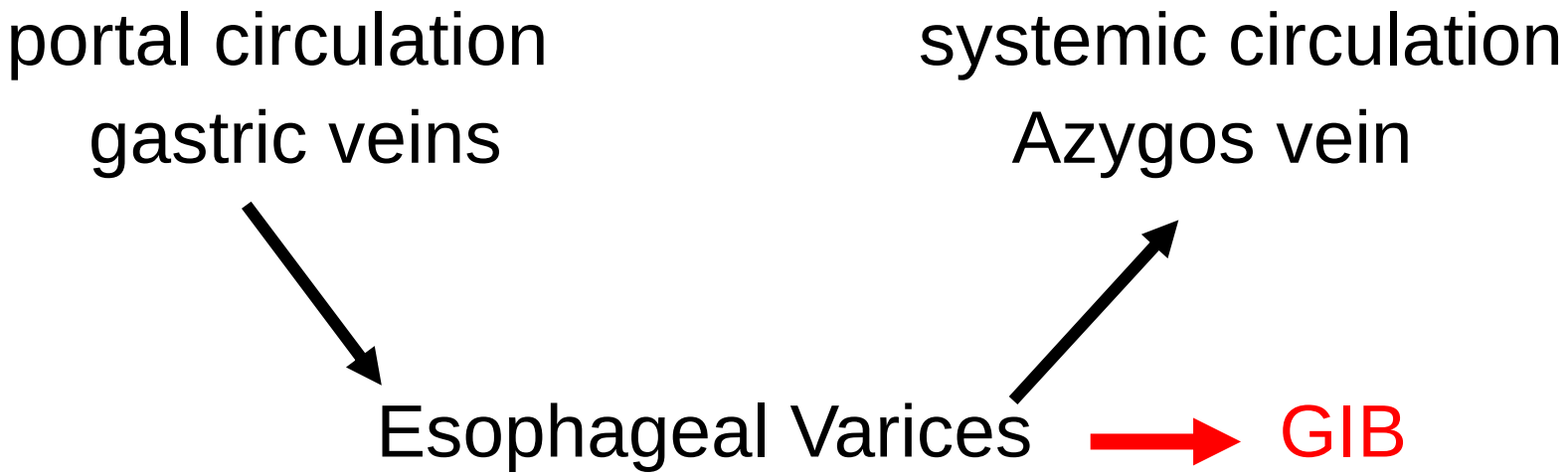
Portal Hypertension



Portal Hypertension

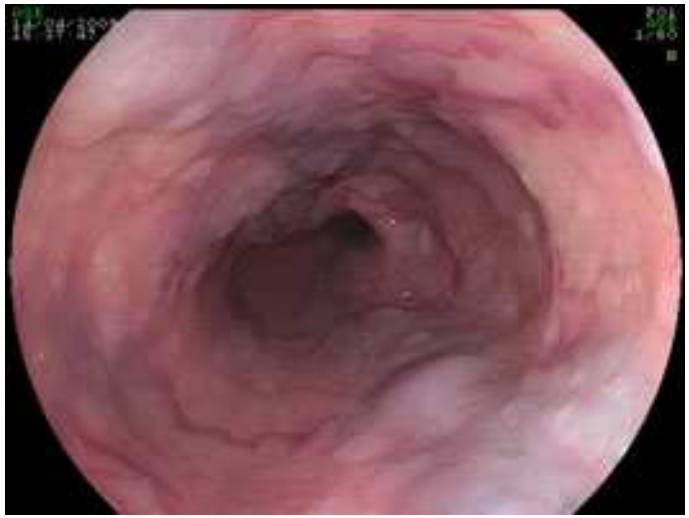
- Defined as pressure difference between the portal vein and the hepatic veins >= 5 mmHg

Portacaval Anastomosis



Esophageal Varices Clinical Presentation

- Melena: 70-80%
- Hematemesis: 40-50%
- Presyncope - 43%
- Epigastric pain - 41%
- Heartburn - 21%
- Hematochezia: 15-20%
- Dyspepsia - 18%
- Syncope - 14.4%
- Weight loss - 12%
- Diffuse abdominal pain - 10%
- Dysphagia - 5%
- Jaundice - 5.2%



Esophageal Varices Clinical Presentation

CHILDS-PUGH grading of chronic liver disease

	Score		
Clinical/laboratory findings	**1**	**2**	**3**
Encephalopathy	None	Mild (grade 1-2)	Severe (grade 3-4)
Ascites	None	Mild/Slight	Moderate/Large
Bilirubin (micromol/l)	< 34	34-51	> 51
Albumin (g/l)	≥ 35	28-35	< 28
Prothrombin time prolongation (secs)	< 4	4-6	> 6
or international normalised ratio (INR)	< 1.3	1.3 – 1.5	> 1.5

Chronic liver disease is classified into Child-Pugh class A to C, employing the total score from the above table.

Total Points	Child-Pugh class
5-6	A
7-9	B
10-15	C

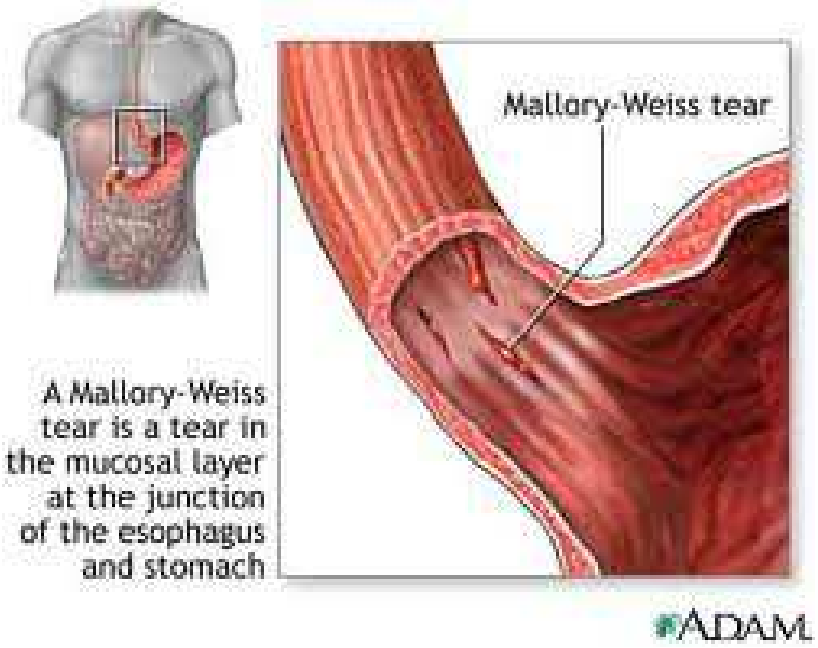
Prevention of Esophageal Varices rebleeding

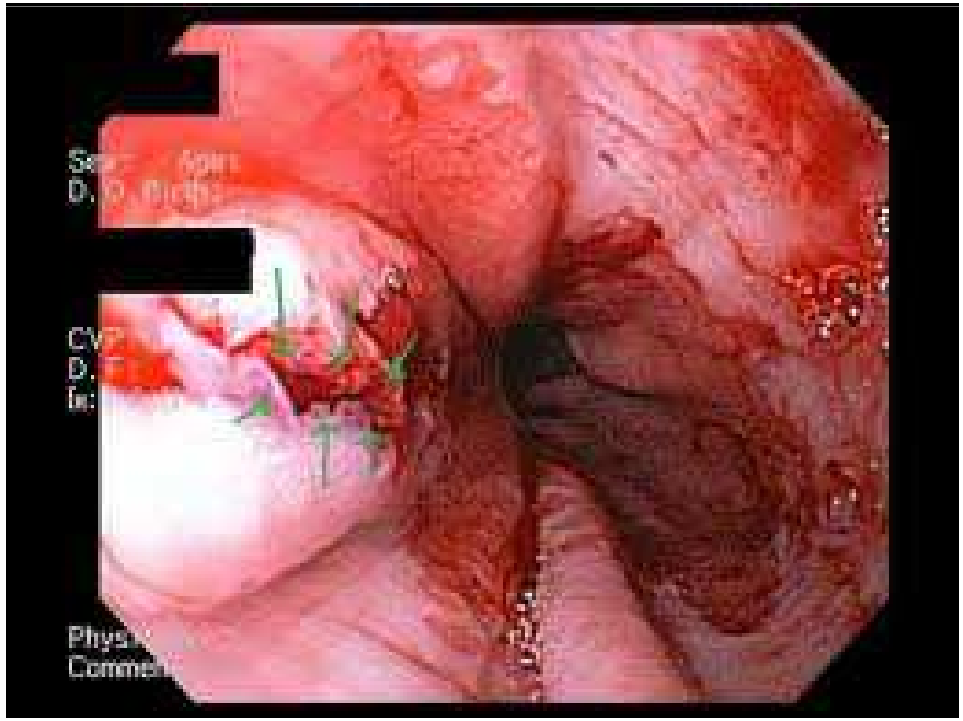
A	Variceal band ligation combined with a beta blocker is recommended as secondary prevention for oesophageal variceal haemorrhage.
A	In patients unsuitable for variceal band ligation combination of non-selective beta blocker and nitrate is recommended as secondary prevention for oesophageal variceal haemorrhage.

Mallory-Weiss syndrome

- **Cause:** the sudden increase of intragastric pressure caused by repeated retching
- **Alcohol intoxication**
- **Pathology:** Rupture of the mucosa in the cardia
- **Treatment:** Conservative treatment usually sufficient, no need of operation

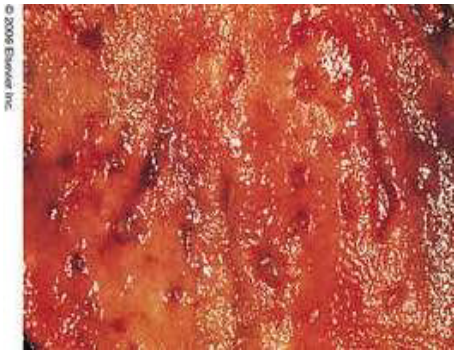
Mallory-Weiss syndrome





Causes of Upper GI Bleed

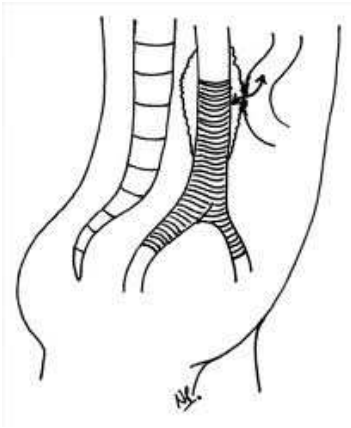
5) Stress ulcers



6) Arteriovenous malformation

7) Malignancy (stomach, esophagus)

8) Aorto-enteric fistula



Pathophysiology of GI Bleeding

1. Mucosal lesions:

   Acid-peptic disease, drug-induced (ASA, NSAIDs), H. pylori, inflammatory bowel diseases (IBD)

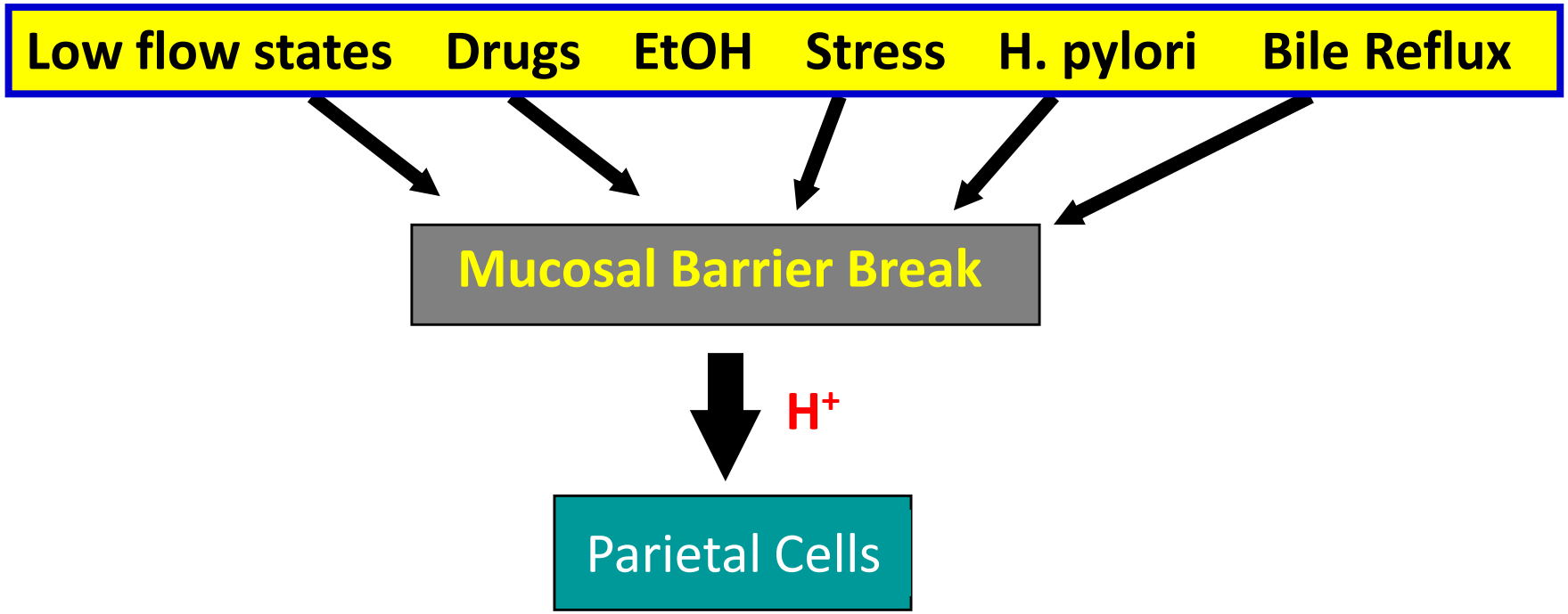
2. Portal hypertension:

   Esophageal varices, hypertensive gastropathy

3. Coagulopathy: Hemophilia, hepatic coagulopathy, CHF with hepatic congestion, CID

4. Vascular lesions: hemangiomas

Causes and Effects of H^+ Ions
Back-diffusion



Release of histamine + Vasodilatation

Increased HCl and Pepsin Secretion

Diagnosis

- **1° Questions to ask in history:**

- **Is it hematemesis, coffee-ground emesis, melena, or hematochezia ?**
- ***History of ASA, NSAID's, steroids use/abuse; OTC drugs***
- ***Vomiting and retching***
- ***Alcohol abuse***
- Weight loss or changes in bowel habits
- History aortic graft

- *History of **Iron** or **Bismuth** which can simulate melena and **Beets** which can simulate hematochezia/melena*

Diagnosis

Physical examination:

- Vital signs may show hypotension and tachycardia
- Cool, clammy skin: ***shock***
- Spider angiomata, palmar erythema, jaundice, and gynecomastia: ***liver disease***
- Petechiae and purpura: ***coagulopathy***
- Rule out causes that can mimic upper GI bleeds
- Proper abdominal exam and rectal exam

Diagnosis

Laboratory:

- **CBC**
- Electrolytes
- BUN / Creatinine ratio: BUN will be elevated in upper GI bleeds
- **Coagulation studies**
- Liver function studies
- Type and cross-match (RBC transfusion)

Where is it from?

GI TRACT	RESPIRATORY TRACT
Dark red or brown	Bright red
In clumps	Foamy, runny & bubbly
Mixed with food	Mixed with mucous
Acidic pH	Alkaline pH
Stomachache, abdominal discomfort	Chest pain
Nausea, retching before/after episode	Persistent cough

Peptic Ulcer Disease
Diagnostic Evaluation

- History (medications, family history)
- Physical exam (include Hem occult - RSO)
- CBC, type & screen for GI bleeding
- PT, PTT
- **EGDS**
- ***H. pylori* search - antibody**
- Fasting gastrin level

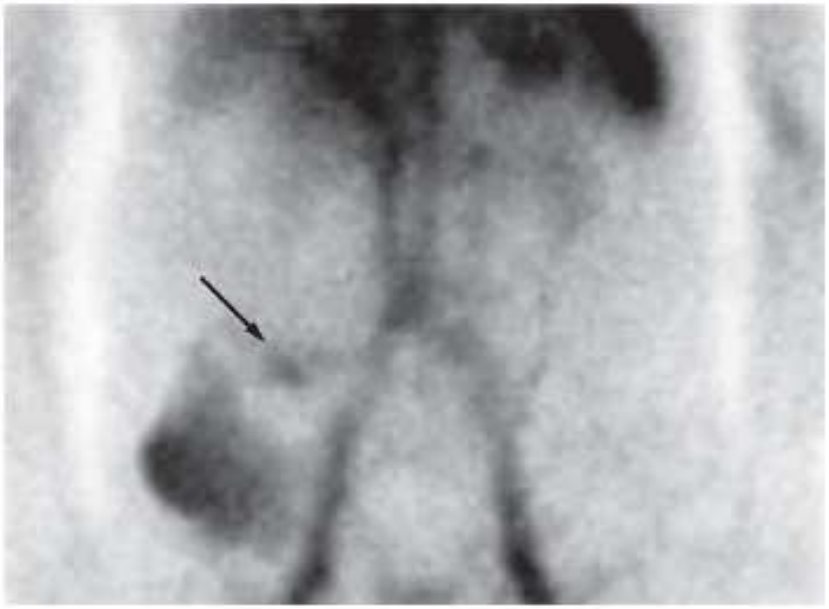
Indications for EGDS

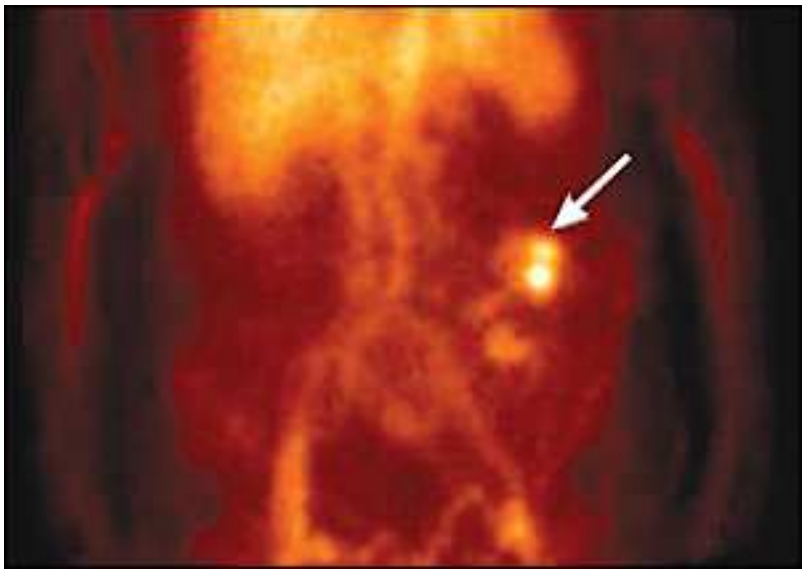
- Hematemesis, Melena
- Severe abdominal pain, weight loss
- Unexplained anemia
- Symptoms persisting despite trial of anti-secretory therapy
- Evaluation of abnormal UGI series
- Evaluation of status of *H. pylori*

^{99m}Tc- Labeled Red Cell Scan

- ^{99m}Tc-sulfur colloid is added to a sample of the patient's red cells and re-infused IV
- patient is scanned with gamma camera
- Half-life is short (2.5 min) so that after 10 minutes only 10% is left in the circulation
- ^{99m}Tc accumulates at the bleeding site and lights up on scan - *can detect 0.1 ml/min*

^{99m}Tc- Labeled Red Cell Scan





Treatment of GI Bleeding

- ABC - protect airway with hematemesis in an obtunded patient
- IV access: two lines (0.9% NS in one line; RBCs not compatible with dextrose)
- Transfuse for: Hgb <=7 – better 8 if active bleeding is present
- NG lavage with normal water

- PPI: IV 2 mg/kg loading dose, then 1 mg/kg/day IV

Drug Efficacy in Healing Ulcers

Drug	Regimen	Ulcers Healed 4 weeks	8 weeks
H2RA			
• Cimetidine	40 mg/k/d	**80%**	**90%**
• Ranitidine	4-8 mg/k/d		
• Famotidine	1-2 mg/k/d		
PPIs			
• Omeprazole	0.7-3 mg/k/d	**85%**	**95%**
• Lansoprazole	0.7-4 mg/k/d		
Sucralfate	40-80 mg/k/d	75%	86%

GI Bleeding - Treatment

Iced saline? NO.

- With cooling, bleeding time increases up to 3 x control, clotting time increases up to 60%, and PT can increase up to 2 x control, and can cause hypothermia
- NG tube is useful to monitor bleeding, much less in active treatment
- Therapeutic endoscopy (sclerotherapy) useful in variceal hemorrhage

GI Bleeding - Octreotide

- Somatostatin analog: **Octreotide** has a longer half-life than somatostatin
- ***OCT decreases splanchnic blood flow and gastrointestinal secretion***
- Make a 1 μg/ml drip - begin drip at a rate of 0.1 μg/kg/min - increase to 0.5 μg/kg/min until bleeding stops, then wean rate
- Possible side effects: nausea, gas, hyperglycemia, gallstones, elevated liver enzymes

ATLS Classification of Shock

Table 5: Classification of hypovolaemic shock by blood loss in adults

	Class I	Class II	Class III	Class IV
Blood loss, volume (ml)	<750	750-1500	1500-2000	>2000
Blood loss (% of circulating blood)	0-15	15-30	30-40	>40
Systolic blood pressure	No change	Normal	Reduced	Very reduced
Diastolic blood pressure	No change	Raised	Reduced	Very reduced/ unrecordable
Pulse (beats per minute)	Slight tachycardia	100-120	120 (thready)	>120 (very thready)
Respiratory rate	Normal	Normal	Raised (>20/min)	Raised (>20/min)
Mental state	Alert, thirsty	Anxious or aggressive	Anxious, aggressive or drowsy	Drowsy, confused or unconscious

Adapted from Baskett, PJF. ABC of major trauma. Management of Hypovolaemic Shock. BMJ 1990; 300: 1453-1457.

NHS Quality Improvement Scotland

GI Bleeding - Shock

- Class 1: no anemia, no active bleeding on lavage, may be followed up as an out or inpatient
- Class 2: mild anemia, active bleeding may be monitored on wards
- Class 3 or 4: admit to ICU, central line, arterial line, IV fluids boluses, transfusion as needed

Rockall score: outcome of GIB

Score					
Variable	**0**	**1**	**2**	**3**	
Age	<60 years	60-79 years	≥80 years		**Initial score criteria**
Shock	'no shock', SBP* ≥100 mm Hg, pulse <100 beats per minute	'tachycardia', SBP≥100 mm Hg, pulse ≥ 100 beats per minute	'hypotension', SBP <100 mm Hg,		
Comorbidity	no major comorbidity		cardiac failure, ischaemic heart disease, any major comorbidity	renal failure, liver failure, disseminated malignancy	
Diagnosis	Mallory-Weiss tear, no lesion identified and no SRH	all other diagnoses	malignancy of upper GI tract		**Additional criteria for full score**
Major stigmata of recent haemorrhage (SRH)	none, or dark spot only		blood in upper GI tract, adherent clot, visible or spurting vessel		

*SBP - systolic blood pressure *SRH - Stigmata of recent haemorrhage
Maximum additive score prior to diagnosis = 7
Maximum additive score after diagnosis = 11.

Rockall numerical risk scoring system

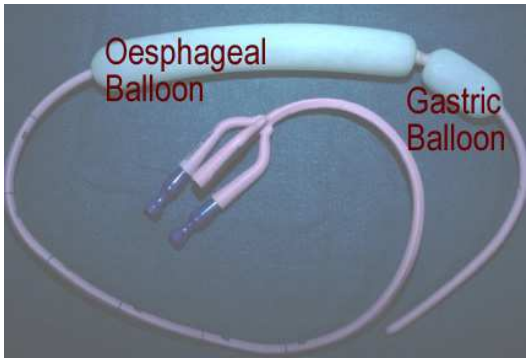
NHS Quality Improvement Scotland

Oesophageal Bleeds

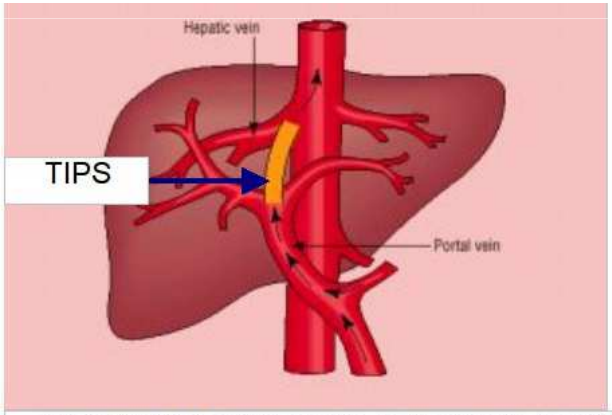
- Reflux: small volumes, bright red
- CA: scanty debris, rusty, other symptoms
- Varices: sudden onset, painless, large volumes, darker, sings of portal HT
- Mallory-Weiss: bright red, history of vomiting

Esophageal Varices treatment

- **Balloon tamponade:**
  - Sengstaken-Blakemore
  - Linton



- **Sclerotherapy (EGDS)**



Transjugular intrahepatic portosystemic shunt

- Oesophageal trans-section
- Variceal ligation or banding
- **TIPS** (transjugular intrahepatic portosystemic shunt)

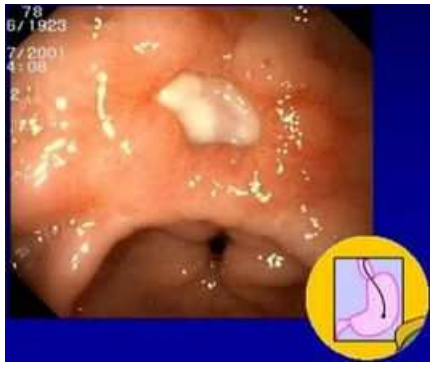
Stomach Bleeds

- Gastritis: small volumes, bright, follows NSAIDS/alcohol/stress
- Ulcer: larger size, painless, herald smaller bleeds; coffee grounds
- CA: uncommon, small bleeds, suggestive history
- Congenital lesions: spontaneous in young, otherwise well, moderate bleeds

Duodenal Bleeds

- Ulcer: history, usually melena present, risk factors, coffee grounds

- Aorto-enteric fistula: rare (except in post-op AAA patients), often fatal (!)



Peptic Ulcer Epidemiology

- 10% of USA population >17 years of age have peptic ulcer disease at some time
- White Americans have a 10% prevalence of H. pylori by age 35 and 50-80% by age 75.
- Black Americans have a 45% prevalence of H. pylori by age 25.

PU Pathophysiology

- Prostaglandins produce mucous and bicarbonate ions which protect the tissue in the stomach by being destroyed with hydrochloric acid and pepsin.
- ***Imbalance between the protective mucosa and acid/pepsin.***
- Peptic ulcer which is a defect beyond muscularis mucosa will develop if there is an imbalance.
- Stress ulcers do not extent through the muscularis mucosa.

PU Causes 1

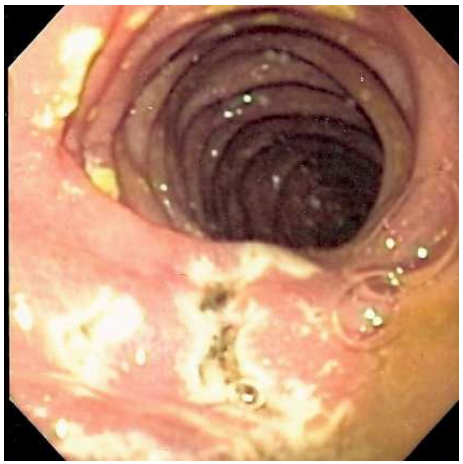
- **H. pylori**: a spiral, urease producing flagellated bacterium which lives between the mucus gel and mucosa
- Its production of ***urease, cytotoxins, proteases*** and other compounds disturb the gel and increase tissue exposure to acid and pepsin
- H. pylori is seen in >90% of patients with duodenal ulcers and > 80% of gastric ulcers
- Note: only 10-20% of patients who are infected with H. pylori will develop ulcers

PU Causes 2

- **NSAID's:** inhibit prostaglandins which in turn increases tissue exposure to acid and pepsin
- **Cigarette smoking:** inhibits bicarbonate ion production and increases gastric emptying
- **Zollinger-Ellison syndrome**

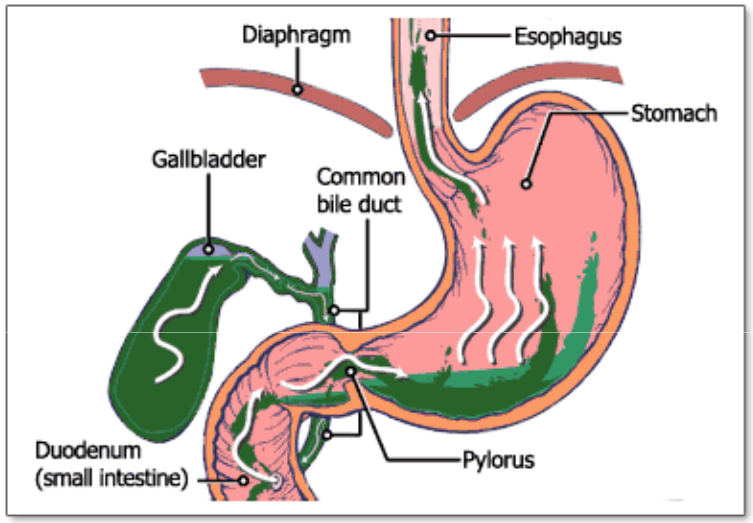
Zollinger–Ellison syndrome

- Is a triad of ***gastric acid hypersecretion + severe peptic ulceration + non-beta cell islet tumor of pancreas (gastrinoma).***
- In this syndrome increased levels of gastrin are produced, causing the stomach to produce excess hydrochloric acid.
- The cause is a tumor (gastrinoma) located in the duodenum or pancreas.
- Gastrin causes an excessive production of acid which lead to peptic ulcers in almost 95% of patients with **GI bleeding**



PU Causes 3

- Bile salts reflux
- Emotional stress
- Prolonged use of corticosteriods
- Caffeinated beverages
- Type O blood

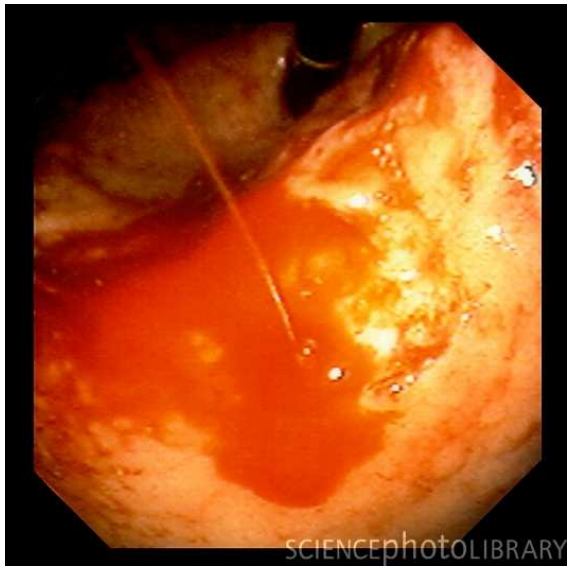


PU Clinical Features

- **Epigastric pain:** (gnawing, aching or burning) is the main complaint
- *Gastric ulcers usually develop pain shortly after eating*
- *Duodenal ulcers usually develop pain 2-3 hours after eating and awaken patients at night. Pain can be relieved by food*
- Physical exam of uncomplicated PUD, there may be a finding of **epigastric tenderness**

Complications of PU

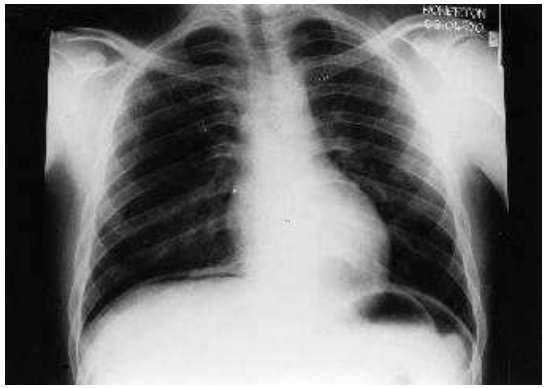
- **Bleeding** is the most common complication of PU and the most common cause of upper GI bleeding !



Complications of PU

Perforation

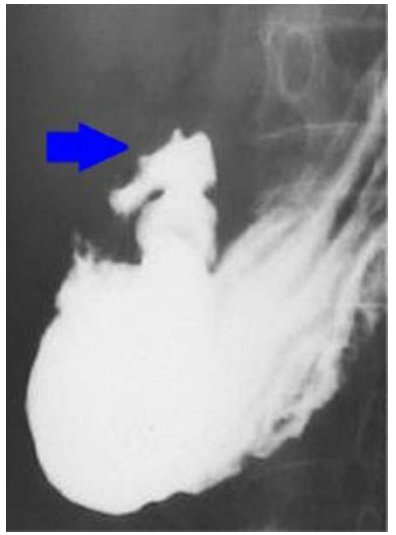
- Initially a chemical peritonitis develops which then progresses to a bacterial peritonitis
- **Anterior perforation** - patients will have sudden abdominal pain with guarding and rebound. 60-70% will demonstrate free air of x-rays
- **Posterior perforation** - patients will develop ***back pain*** with no free air on x-ray and may mimic pancreatitis but lipase will be normal or only slightly elevated
- **No free air on x-rays cannot rule perforation for shure**



Complications of PU

Gastric outlet obstruction

- Scaring from healed ulcers or edema from active ulcer with development of obstruction
- ***Obstruction will cause gastric dilation, vomiting, dehydration*** with metabolic alkalosis
- Patients will develop upper abdominal pain with vomiting, early satiety, weight loss, succussion splash (borborigmi)
- Abdominal x-ray will show dilated stomach shadow with large air-fluid level



PROGNOSTIC FACTORS FOR UPPER GASTROINTESTINAL BLEEDING

The following factors are associated with a poor outcome, defined in terms of severity of bleed, uncontrolled bleeding, rebleeding, need for intervention and mortality. These factors should be taken into account when determining the need for admission or suitability for discharge.

- **Age** - mortality due to UGIB increases with age across all age groups. Odds ratio (OR) for mortality is from 1.8 to 3 for age >60 years (compared to patients aged 45-59 years), and from 4.5 to 12 for age >75 years (compared to patients ≤75 years).[2,4,6]
- **Comorbidity** - the absence of significant comorbidity is associated with mortality as low as 4%.[2,4,6,7] Even one comorbidity almost doubles mortality (OR 1.8) and the presence of cardiac failure (OR 1.8) or malignancy (OR 3.8) significantly worsens prognosis.
- **Liver disease** - cirrhosis is associated with a doubling of mortality and much higher risk of interventions such as endoscopic haemostasis or transfusion.[8] The overall mortality of patients presenting with varices is 14%.[1]
- **Inpatients** have approximately a threefold increased risk of death compared to patients newly admitted with GI bleeding. This is due to the presence of comorbidities in established inpatients rather than increased severity of bleeding.[4,5]
- **Initial shock** (hypotension and tachycardia) is associated with increased mortality (OR 3.8)
- **Continued bleeding** after admission is associated with high risk of intervention (OR 1.8)[7] and up to a 50-fold increased mortality.[6]
- **Haematemesis** - the presence of initial haematemesis doubles mortality.[2,7]
- **Haematochezia** - the presence of haematochezia doubles rebleeding, mortality and surgery rates.[9]
- **Elevated blood urea** is associated with a need for intervention.[10]

NHS Quality Improvement Scotland

What else after UGB ?

A	**Patients with peptic ulcer bleeding should be tested for Helicobacter pylori** *(with biopsy methods or urea breath test)* **and a one week course of eradication therapy prescribed for those who test positive. A further three weeks ulcer healing treatment should be given.**
☑	Medicines known to increase the risk of upper gastrointestinal complications should, where possible, be given in monotherapy and at the lowest effective dose to minimise the risk of upper gastrointestinal complications.
A	**Patients with healed bleeding ulcers who test negative for Helicobacter pylori require concomitant proton pump inhibitor therapy at the usual daily dose if NSAIDs, aspirin or COX-2 inhibitors are indicated.**
A	▪ **Aspirin and NSAIDs should be discontinued when patients present with peptic ulcer bleeding.** ▪ **Once ulcer healing and eradication of Helicobacter pylori are confirmed, aspirin and NSAIDs should only be prescribed if there is a clear indication.**
D	**Selective serotonin reuptake inhibitors should be used with caution in patients who have an increased risk of gastrointestinal bleeding, especially in patients taking NSAIDs or aspirin. A non-SSRI antidepressant may be an appropriate choice in such patients.**
D	**Oral anticoagulants or corticosteroids should be used with caution in patients at risk from gastrointestinal bleeding, especially in those taking aspirin or NSAIDs.**

Drugs Licences

Annex 2

Drug licensing status

All drugs recommended in this guideline are licensed for the indication included in the recommendation with the following exceptions:

Section	Drug
5.3.2	Proton pump inhibitors are not licensed for the reduction in rate of rebleeding in patients with bleeding peptic ulcers.
6.2.1	Somatostatin and vapreotide are not licensed for use in the management of variceal bleeding.
6.2.2	Somatostatin and octreotide are not licensed for use in the management of variceal bleeding.
7.2.1	With the exception of propranolol, beta blockers are not licensed for secondary prevention of oesophageal variceal haemorrhage.

NHS Quality Improvement Scotland

2. LOWER GASTROINTESTINAL BLEEDING

Lower GI Bleeding

- ***Blood streaks*** on the stool: indicates *anal outlet bleeding*
- ***Blood mixed with stool:*** indicates *bleeding source higher than the rectum*
- ***Blood with mucus:*** indicates an *infectious or inflammatory disease of the colon*
- ***Currant jelly-like*** material: might indicate vascular congestion and hyperemia (intussusception or midgut volvulus)

Lower GI Bleeding

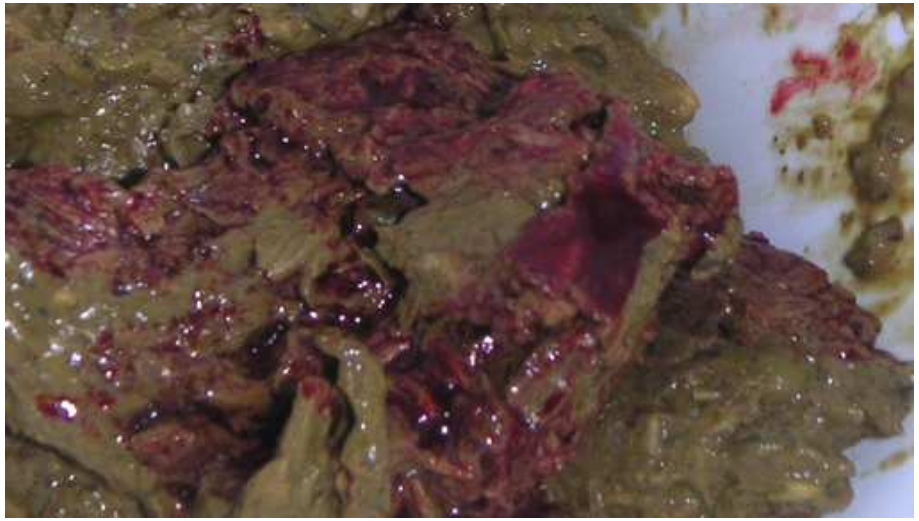
- ***Haematochezia:*** indicate voluminous bleeding with or in the stools (may also occur from a brisk upper gastrointestinal bleed)
- ***Rectorrhagia:*** rectal bleeding ***is not*** associated with defecation.
- ***Melena:*** passage of ***black, sticky (tarry) stools*** suggests upper GI tract bleeding (but can be as distal as the right colon - slow stool progression)

  Hematemesis suggests a large bleed with possible recurrence while melena alone indicates less voluminous bleeding

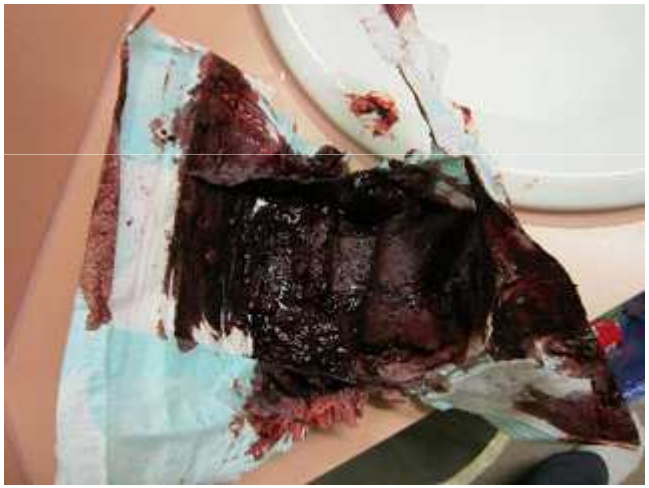
Lower GI Bleeding

Hematochezia

Hematochezia is the passage of fresh or altered blood per rectum usually due to colonic bleeding. Occasionally profuse upper gastrointestinal or small bowel bleeding can be responsible.



Haematochezia



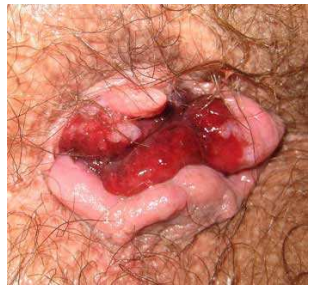
Melena

Causes of Lower GI Bleeding

Table 9: Major causes of colonic bleeding

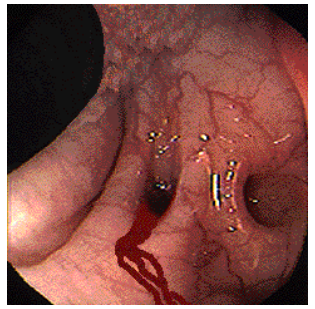
Major causes of colonic bleeding
diverticular disease
vascular malformations (angiodysplasia)
ischaemic colitis
haemorrrhoids
inflammatory bowel disease (eg ulcerative proctitis, Crohn's disease)
neoplasia (carcinoma or polyps)
radiation enteropathy

Causes of Lower GI Bleeding



1) **Hemorrhoids**: the most common cause

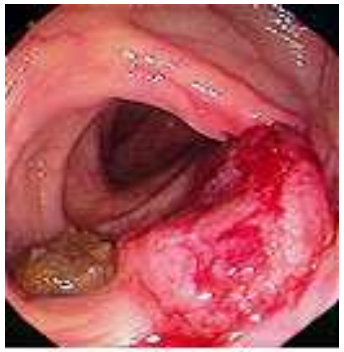
2) **Diverticulosis**: common, painless, and can be a massive bleeding
   - Caused from an erosion into a penetrating artery from the diverticulum

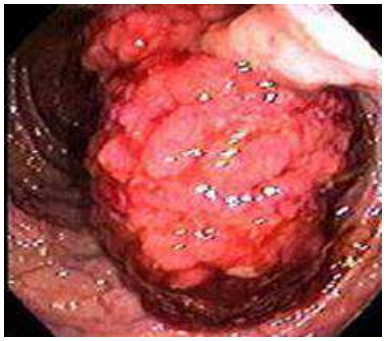


3) **Arteriovenous malformations**

Causes of Lower GI Bleeding

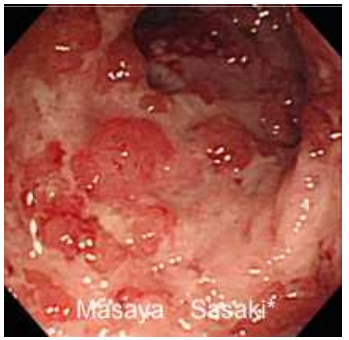
4) **CA or polyps**





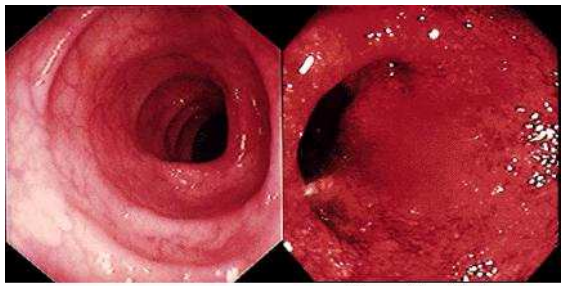
Cancer

5) Inflammatory bowel diseases



6) **Infectious gastroenteritis**

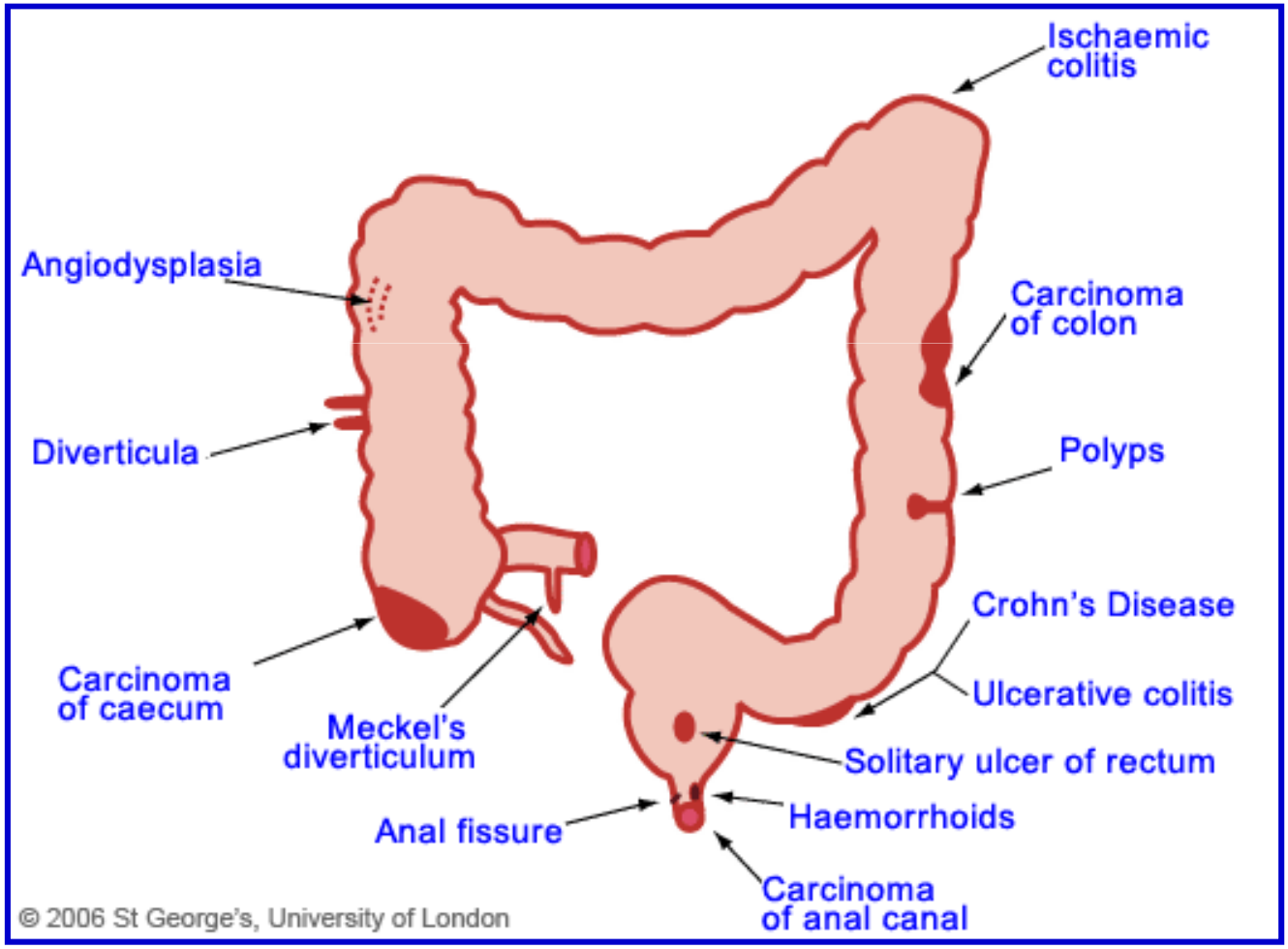
7) **Meckel diverticulum**



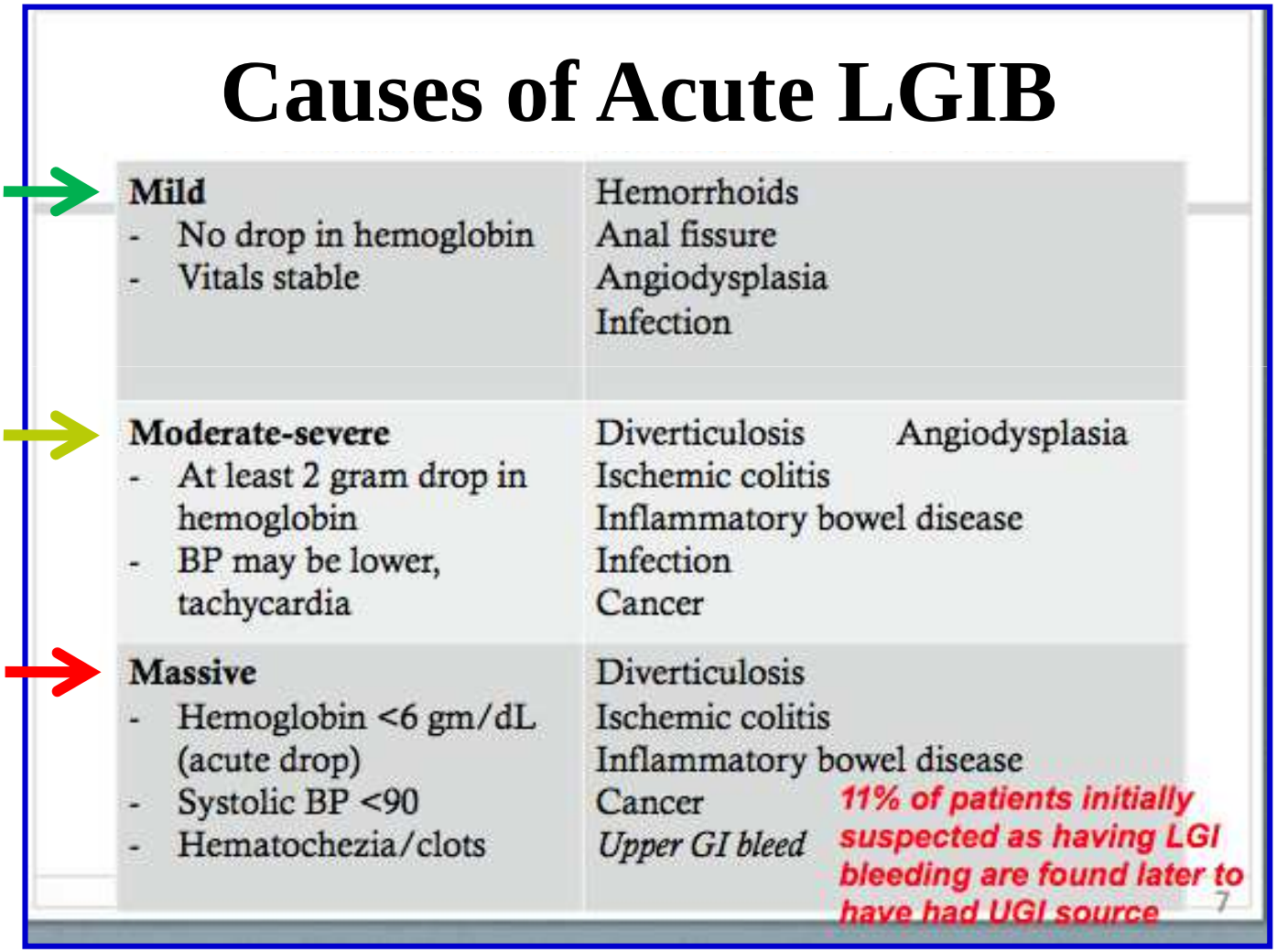
Healthy Colon

Ulcerative Colon

Lower GI Bleeding



Causes of Lower GI Bleeding



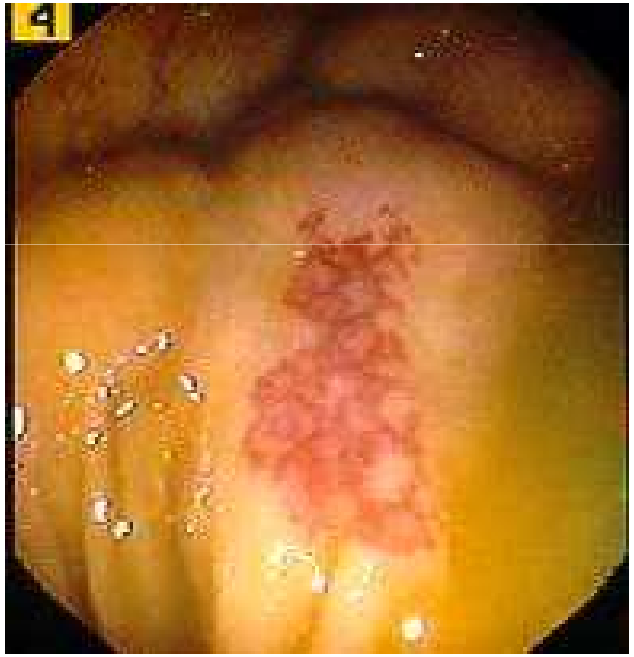
Lower GI bleeding Diagnosis

Diagnostic:

- **Abdominal series**: not beneficial unless specific indications
- **Angiography**: ***can be diagnostic and therapeutic but requires a brisk bleed at 0.5-2ml/min***
- **Bleeding scans** **(scintigrafia)**: can only be diagnostic but are more sensitive then angiography and require a bleeding rate of only 1ml/min
- **Colonoscopy**: is diagnostic and therapeutic and more accurate than bleeding scans and angiography

Angiodysplasia of the Colon

• ***Angiodysplasia is the most common vascular lesion of the gastrointestinal tract***. It may be asymptomatic as it may cause GI bleeding
• The vessel walls are thin, with little or no smooth muscle, and the vessels are ectasic and thin
• After diverticulosis, it is the second leading cause of lower intestinal bleeding in patients older than 60 years
• Angiodysplasia may account for approximately 6% of cases of lower GI bleeding.



PROGNOSTIC FACTORS FOR LOWER GASTROINTESTINAL BLEEDING

Acute lower gastrointestinal bleeding

There is limited evidence available on the initial assessment of patients with acute lower gastrointestinal bleeding (LGIB). One general review of management[15] and one guideline were identified.[16] Other evidence comes from case series and epidemiology, and from expert opinion. Two uncontrolled case series analyse early predictors of severity, one prospective[17] and one retrospective.[18] The available evidence identifies the following factors associated with uncontrolled bleeding and/or death.

- **Age** - acute lower GI bleeding occurs most often in the elderly. The precise relationship between age and mortality is statistically less well defined than for UGIB.[15,18,19]
- **Acute haemodynamic disturbance** (OR 3 to 4.3) and gross rectal bleeding on initial examination (OR 2.3 to 3) are important predictors of subsequent severe bleeding.[17,18]
- **Comorbidity** - the presence of two comorbid conditions doubles the chance of a severe bleed (OR 1.9).[18]
- **Specific drugs** – patients taking aspirin or NSAIDs are at increased risk of severe lower GI bleeding (OR 1.8 to 2.7).[18,20]
- **Inpatients** who are hospitalised for another condition and who subsequently bleed after admission have a mortality rate of 23% compared with 3.6% in those admitted to hospital because of rectal bleeding ($p < 0.001$).[19]

The patient's history is important for accurate assessment of risk and can give important clues to the diagnosis and need for admission. For example, a history of previous LGIB from a known diagnosis of diverticular disease (the commonest cause of LGIB accounting for 23-48% of cases) predicts a further episode with a 10% chance of recurrence at one year and 25% at four years. Diverticular bleeds resolve spontaneously in 75% of cases.[19]

NHS Quality Improvement Scotland