

Types of patients:

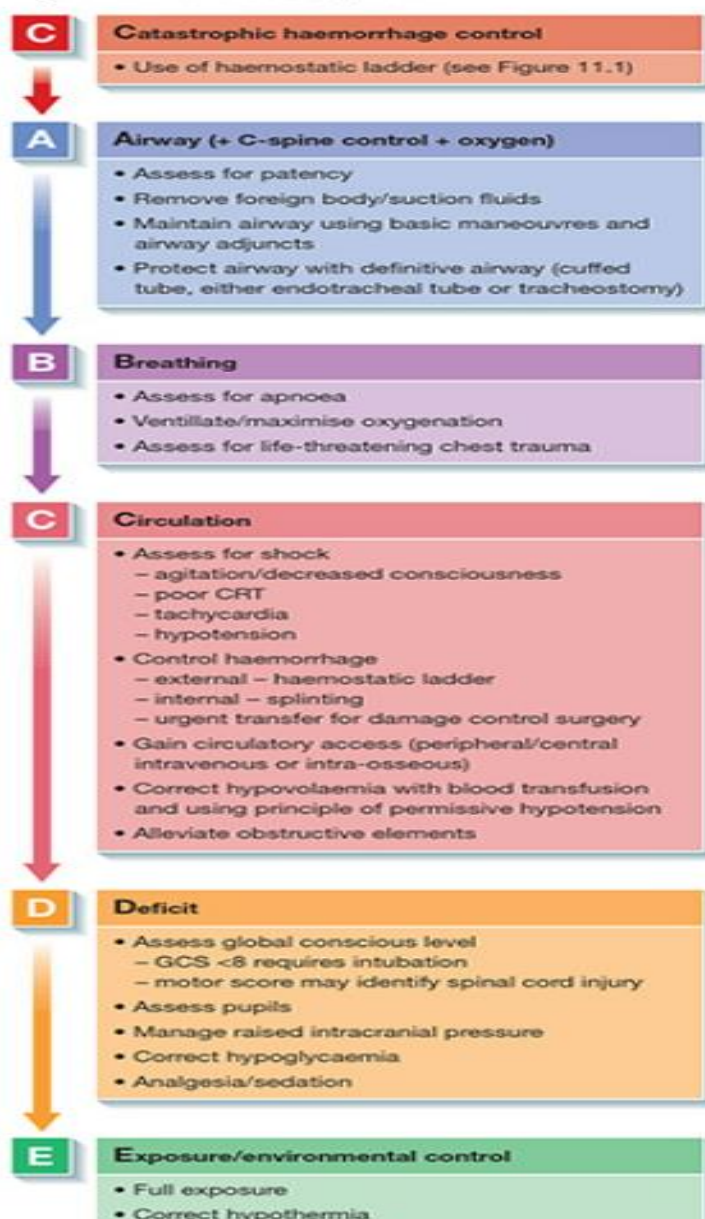
1. acutely ill patients require a rapid, targeted evaluation ('airway, breathing, circulation, disability, exposure [ABCDE] assessment') for life-threatening disorders.
2. patients who are stable, including those initially assessed by the ABCDE approach, should have a full history and clinical examination, alongside basic tests relevant to the specific presentation.

ABCDE Assessment (Primary Survey):

prompt identification of life-threatening pathology with immediate management of any abnormalities detected

used for patients who appear unwell or are unresponsive, or exhibit evidence of acute physiological derangement on basic observations (HR, RR, BP, arterial oxygen saturation [SpO₂], temperature)

Figure 10.1 C-ABCDE approach



Behaviour	Response	Score
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	No response	1
Best response: verbal	Fully oriented	5
	Confused speech	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best response: motor	Obeys commands	6
	Localises to painful stimulus	5
	Withdraws from painful stimulus	4
	Flexes to pain (decorticate)	3
	Extends to pain (decerebrate)	2
	No response	1
Severity of TBI	Minor brain injury	13–15
	Moderate brain injury	9–12
	Severe brain injury	3–8

 Clinical tool The ABCDE assessment	
What to look for	How to respond
A Airway	
A1 Ask 'How are you feeling?' If patient can speak normally, airway is patent – move straight to B.	If signs of obstruction: • Get help! • Try simple airway manoeuvres: head tilt/chin lift; jaw thrust (Fig. 2.1) • Remove foreign bodies/secretions from pharynx under direct vision • Insert Guedel or nasopharyngeal airway If obstruction persists: • Get expert assistance immediately • Consider laryngeal mask airway or tracheal intubation • In the context of anaphylaxis (see below), if throat or tongue swelling, give IM adrenaline (0.5 mg)
B. Breathing	
B1 Give high concentration O₂ initially if hypoxaemic	
B2 Assess rate, depth and symmetry of breathing noting: • Poor respiratory effort: ↓↓RR, feeble, shallow Breaths • High respiratory effort: RR >20/min, use of Accessory muscles, visibly tiring • Asymmetrical chest expansion	If respiratory effort inadequate: • Get help! • Manually ventilate via bag-valve-mask • Consider a trial of naloxone, if any suspicion of opiate toxicity
B3 Check for tracheal deviation	If severe respiratory distress and signs of tension pneumothorax (p. 264), perform immediate needle aspiration.
B4 Percuss and auscultate chest, noting: • Dullness – suggesting pleural effusion, collapse, consolidation • ↓breath sounds – suggesting collapse pneumothorax, pleural effusion • Wheeze – suggesting bronchospasm • Crackles – suggesting pulmonary oedema, fibrosis consolidation • Bronchial breathing suggesting consolidation	If widespread wheeze, check for signs of anaphylaxis (see below) If present, manage as described; otherwise, give nebulized bronchodilator.
B5 Record SpO₂	If chronic type 2 respiratory failure or severe chronic obstructive pulmonary disease (COPD), titrate fraction of inspired oxygen (FIO ₂) to patient's baseline SpO ₂ (if known) or 90–92%. In all other critically ill patients, continue high FIO ₂ .

Clinical tool—cont'd The ABCDE assessment	
What to look for	How to respond
C. Circulation	
C1 Check colour & temperature of hands • Cold, clammy, blue, mottled? • Pink and warm?	In patients with evidence of shock, e.g. ↑CRT, cold peripheries, thready pulse, ↑HR, ↓BP: Secure IV access (large bore if possible) If ventricular tachycardia (VT), attempt defibrillation with a synchronized DC shock (ask anaesthetist to sedate if conscious). If bradycardia: • Give atropine 0.5–3 mg • if no response or HR <40/min, get expert help and consider IV adrenaline or transcutaneous pacing If tongue/throat swelling, severe respiratory distress, widespread wheeze and/or a new rash, assume anaphylaxis: • Stop any potential trigger • Give 0.5 mg IM adrenaline (anterolateral aspect of middle 1/3 of the thigh) • Give fast IV fluids • Get immediate anaesthetic help Otherwise, give fluid challenge unless evidence of pulmonary oedema.
C2 Measure capillary refill time (CRT) Press firmly over fingertip for 5 sec, release pressure then record time taken for skin to return to normal colour • ≤2 sec is normal • ≥2 sec suggests ↓peripheral perfusion	
C3 Palpate radial and carotid pulse: • Tachycardia: >100 bpm • Bradycardia: <60 bpm (or inappropriately slow for context) • Thready, weak – suggesting ↓cardiac output, e.g. hypovolaemia • Bounding – suggesting hyperdynamic circulation, e.g. early sepsis	
C4 Measure blood pressure	
C5 Assess height of JVP at 45°	
C6 Auscultate the heart for: • Murmurs • 3rd heart sound/gallop rhythm	
C7 Attach ECG monitor and review rhythm: • Regular broad complex tachycardia – likely ventricular tachycardia (VT) (p. 234) • Regular narrow complex tachycardia, e.g. sinus tachycardia, supraventricular tachycardia (SVT), atrial flutter (p. 232) • Irregular tachycardia – likely atrial fibrillation (p. 232) • Bradycardia ≤40 bpm, e.g. 2nd or 3rd degree atrioventricular (AV) block (p. 234)	
C8 Perform a 12-lead ECG if chest pain or arrhythmia	
D. Disability	
D1 Check capillary blood glucose (CBG)	If CBG <3 mmol/L: • Send blood for formal lab glucose measurement • Give immediate IV dextrose If ↓GCS • Perform an ABG if any suspicion of hypercapnia, e.g. chronic lung disease, depressed ventilation • Give 0.8–2 mg IV naloxone if ↓pupil size or no obvious cause • Assess response after 1 min and consider further doses if partial response
D2 Record Glasgow Coma Scale (p. 73)	



Clinical tool—cont'd The ABCDE assessment

What to look for	How to respond
D3 Take a '3D' history Description of symptoms Drugs and allergies Disorders/Disability prior to this illness D4 Examine pupils with a pen torch: • Bilateral pinpoint – suggests opioid intoxication or pontine lesion • Bilateral dilated – suggests cocaine/amphetamine or tricyclic antidepressant intoxication or atropine • Unilateral fixed, dilated suggests ↑intracranial pressure or 3rd nerve palsy	
E. Exposure	
E1 Record body temperature E2 Fully expose the body (preserve dignity), looking for: • Bleeding or injuries • Rashes • Jaundice • Medic alert bracelet E3 Examine the abdomen for distension, tenderness, guarding, rigidity	If temperature <34°C, confirm core temperature with a low-reading thermometer and start rewarming measures. Repeat the ABCDE assessment if a significant abnormality was noted at any stage. Refer to the relevant chapter for further assessment of specific presentations, e.g. dyspnoea, shock, chest pain, ↓GCS, abdominal pain, headache.

History taking & Physical Exam of Abdomen

GIT SYMPTOMS:

1. **painful mouth**: the most common cause is recurrent aphthous stomatitis (painful, shallow)

mostly **idiopathic** but can be associated with underlying systemic diseases:

celiac disease

IBD

Behcet disease: multisystemic disease. 100% of patients will have recurrent oral ulcers (the m.c. feature of Behcet dz. Recurrent: means 3 times or more per year)

SLE

acute HIV infection

neutropenia

micronutrients deficiencies (Fe, folate B9, B12, Vit. C)

drugs eg. Methotrexate

2. **Heart Burn / acid reflux**: the most likely dx is GERD

Differentiate heartburn from cardiac chest pain by its burning quality, upward radiation, association with acid reflux and its occurrence on lying flat or bending forward.

3. **Dysphagia & Odynophagia**:

Dysphagia always needs investigation to exclude malignancy!

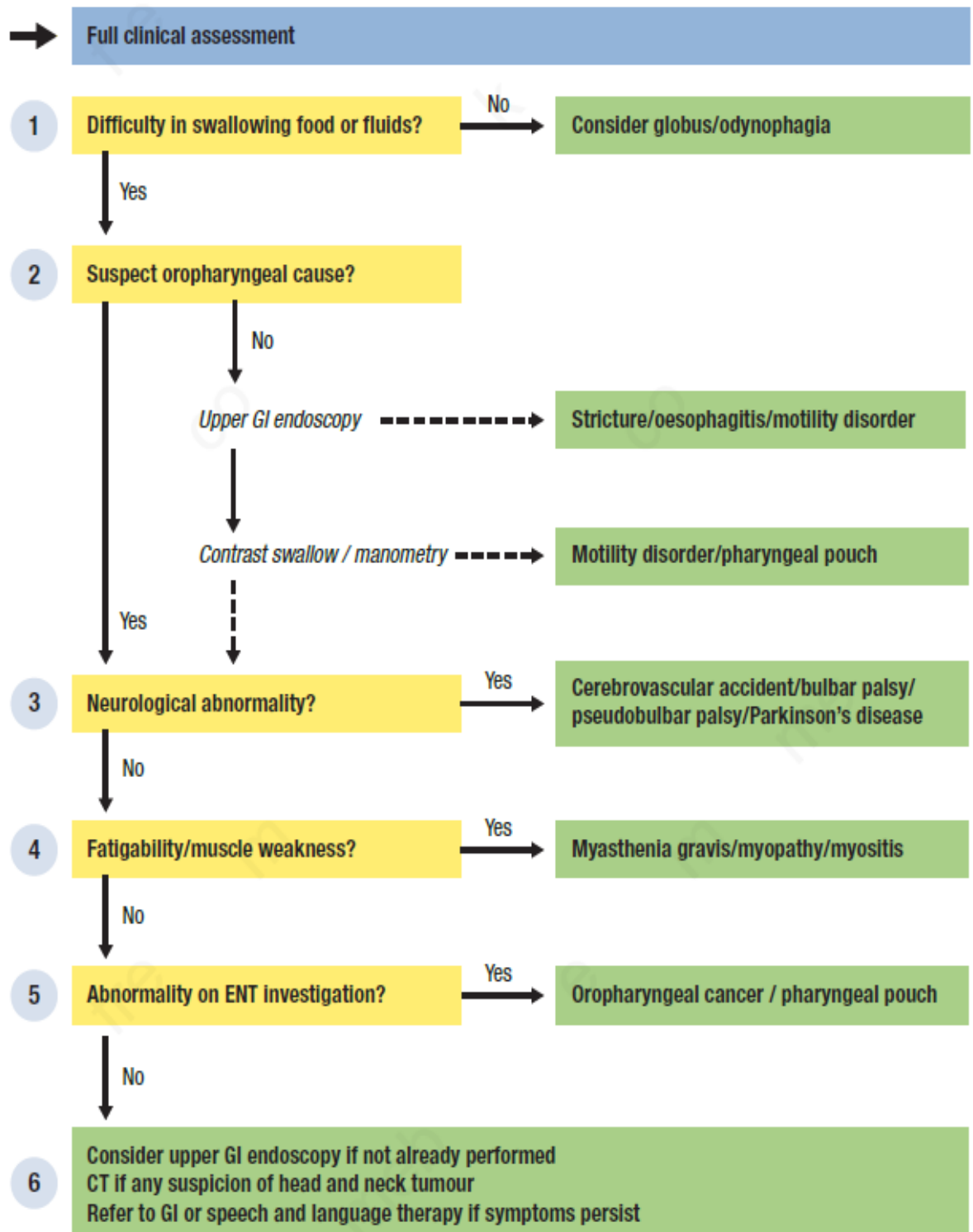
Key questions to ask?

- Is the Dysphagia for both solid & liquid from the start (motility) or solid then liquid (structural)?
- Difficulty to initiate the swallowing movement / cough on swallowing? (bulbar)
- Painful swallowing (odynophagia)? Think of sore throat, esophagitis, or esophageal spasm
- Is the dysphagia intermittent (spasm) or constant & getting worse (malignant stricture)?
- Neck Bulging or gurgle on drinking? (Pharyngeal pouch)

Dysphagia		
Oropharyngeal dysphagia	Esophageal dysphagia	
DDx:	Structural	Motility disorders
Bulbar palsy: Lower motor neuron palsies of cranial nerves IX–XII result in weakness of the tongue and muscles of chewing /swallowing. The tongue is flaccid with fasciculation, Pseudobulbar palsy: Bilateral upper motor neuron lesions of cranial nerves IX XII produce a small, contracted, slowly moving tongue & pharyngeal muscles with a <u>brisk jaw jerk</u> Myasthenia gravis: Fatigability of oropharyngeal muscles causes increasing swallowing difficulty after the first few mouthfuls. Pharyngeal pouch posterior herniation of the pharyngeal mucosa between the thyropharyngeus and cricopharyngeus muscle, and is usually found in elderly associated with regurgitation of undigested food, halitosis, the feeling of a lump in the neck	Structural causes usually cause dysphagia for solids Malignant stricture: esophageal cancer typically present with progressive, painless dysphagia to solid foods esophageal cancer associations: male, GERD, smoking, alcohol, Barret's, Achalasia Benign stricture: May be caused by: GERD Esophageal web: seen in iron deficiency, typically posterior to cricoid Schatzki rings (benign idiopathic strictures of the lower esophagus) caustic substances ingestion radiotherapy eosinophilic esophagitis	motility disorders may cause dysphagia solids and liquids Achalasia loss of peristalsis in the distal esophagus and LES fails to relax due to degeneration of <u>myenteric plexus</u>. Dysphagia is also associated with <u>food regurgitation & wt loss</u>. Dx: Barium swallow (bird peak) Chagas disease Caused by Trypanosoma Cruzi. The major symp. Are Cardiomyopathy & GI Scleroderma Diffuse esophageal spasm Barium swallow: corkscrew appearance

Table 11.1 Comparison of signs in bulbar and pseudobulbar palsy

	Bulbar	Pseudobulbar
Speech	Nasal tone; difficulty forming consonants (especially 'R'); may become slurred	'Donald duck' voice
Tongue	Weak, wasted with fasciculation	Small, stiff
Jaw jerk	Normal or absent	Brisk
Gag reflex	Absent	Present
Emotions	Normal	Labile (e.g. uncontrollable laughing/crying)



4. Nausea & Vomiting:

Nausea is the unpleasant sensation of being about to vomit.
Vomiting is the forceful expulsion of gastric contents often regurgitation there is appearance of gastric contents without any effort.

Key Questions to ask:

1. **Content of Vomit:** recognizable food = gastric, coffee ground = UGIB
Greenish = I.O. below sphincter of Oddi (Biliary)
Feculent = bowel obstruction or Bacterial overgrowth
2. **Timing of vomiting:**
Morning ≈ pregnancy or high ICP (vomiting without Nausea)
1 hr post food ≈ gastric stasis/gastroparesis (DM)
vomiting that relieves pain ≈ peptic ulcer
preceded by loud gurgling ≈ GI obstruction
3. **Associated symptoms:**
Fever, jaundice, headache, chest pain, Abdominal pain, diarrhea
4. **Duration:** if ≤ 10 days: acute (infectious cause?), >10 chronic

Causes

1. Alimentary causes:

1. **GI infection:** Gastroenteritis (GE) is the MCC of acute N&V and usually associated with diarrhea, fever, Abdominal cramps
GE is most commonly due to viral causes: norovirus, rotavirus, adenovirus
Bacterial causes of acute GE (vomiting): *Bacillus cereus* and *Staph. aureus*.
2. **GI obstruction:**
4 cardinal symptoms: vomiting, Abd pain, constipation, distension
The characteristics of the vomitus may suggest the location of obstruction:
food – gastric outlet obstruction
bilious material – small bowel obstruction
feculent material – distal obstruction
3. **Other GI causes:**
Peptic ulcer disease: N&V typically associated with epigastric discomfort/dyspepsia
Gastroparesis: vomiting after food ingestion due to delayed gastric emptying; suspect this in patients with poorly controlled diabetes/multiple diabetes related complications, scleroderma or previous gastric surgery.

2. Extra-alimentary causes

8.9 Non-alimentary causes of vomiting	
Neurological	
• Raised intracranial pressure, e.g. meningitis, brain tumour • Labyrinthitis and Ménière's disease	• Migraine • Vasovagal syncope, shock, fear and severe pain, e.g. renal colic, myocardial infarction
Drugs	
• Alcohol, opioids, theophyllines, digoxin, cytotoxic agents, antidepressants	• Consider any drug
Metabolic/endocrine	
• Pregnancy • Diabetic ketoacidosis • Renal failure	• Liver failure • Hypercalcaemia • Addison's disease
Psychological	
• Anorexia nervosa	• Bulimia

Box 25.2 Clinical / biochemical features often present in patients with adrenal insufficiency

Symptoms (often insidious)

- Lethargy, fatigue
- Anorexia, weight loss
- Nausea and vomiting
- Postural hypotension

Physical signs

- Pigmentation (skin, mucous membranes)
- Vitiligo
- Postural hypotension

Blood

- Hyponatraemia
- Hyperkalaemia
- Hypoglycaemia (fasting or spontaneous)
- Normal-anion gap metabolic acidosis (mild)

Other

- History of other autoimmune disorders

CNS causes of N&V

Raised intracranial pressure may produce vomiting, often without nausea. When chronic, e.g., space occupying lesion, vomiting classically occurs shortly after waking and is associated with headache (worse on lying, bending or straining) and papilledema.

In acute causes, e.g., intracerebral hemorrhage, there may be sudden onset headache initially followed by a progressive decline in GCS. Nausea, with or without vomiting, occurs during episodes of migraine in ~80% of patients and is often severe; however, headache +/- aura is usually

the dominant symptom.

N&V accompanied by vertigo suggests vestibular or brainstem pathology. Vomiting may also occur in CNS infection, e.g., meningitis but usually not as the main presenting feature.

Box 30.1 Clinical features of shock

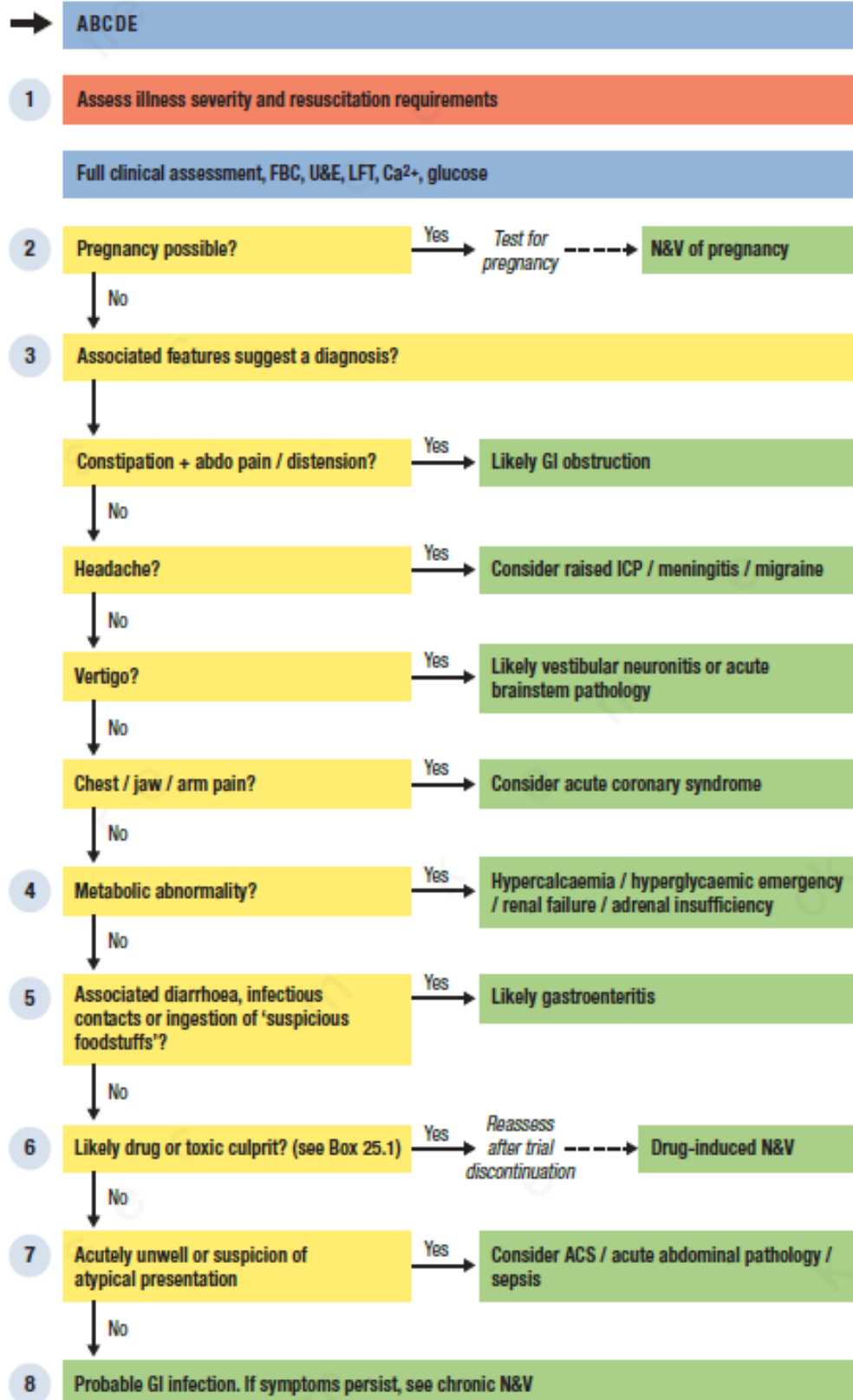
Haemodynamic

- Systolic BP <100 mmHg or a drop of >40 mmHg from baseline
- Pulse >100/min

Tissue hypoperfusion

- Skin: capillary refill time >2 sec or cold/pale/mottled extremities
- Renal: oliguria (<0.5 mL/kg/hr) or ↑serum creatinine (acute)
- CNS: GCS <15, confusion or agitation
- Global: ↑lactate (in the absence of hypoxaemia)

≥2 of the above indicate shock



Cont. of GI symptoms:

5.dyspepsia/indigestion

Discomfort/epigastric pain centered in the upper abdomen.

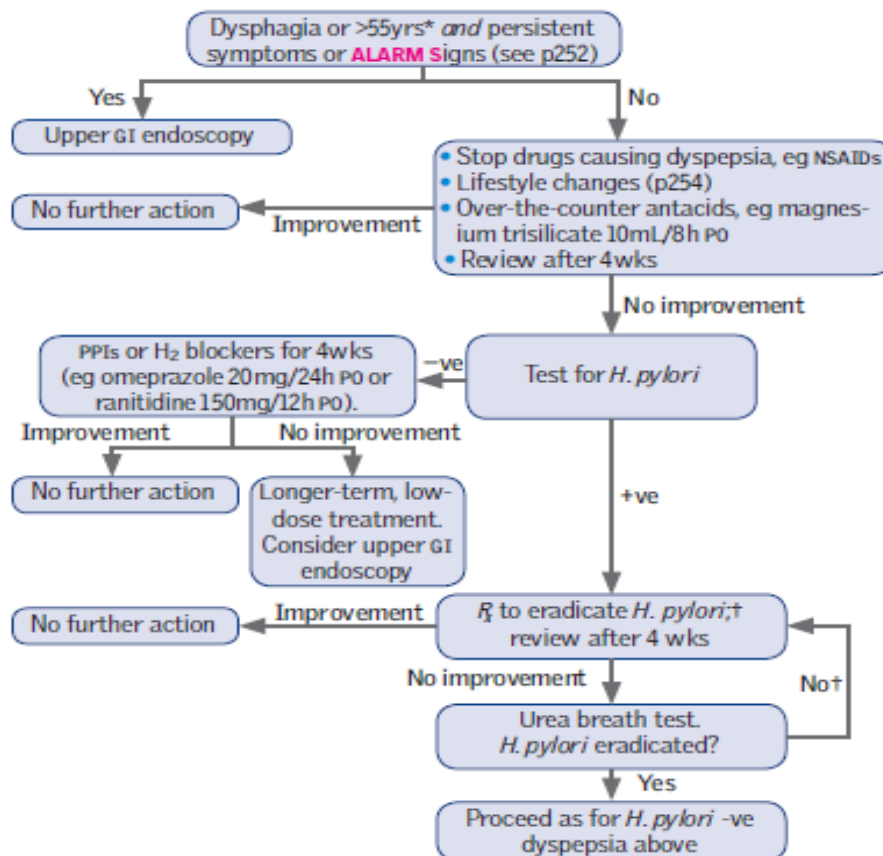
Majority of cases it's functional dyspepsia (no identifiable cause) but it's a dx of exclusion so you have to rule out other causes.

Alarm symptoms: Anemia (iron deficiency); loss of weight; anorexia; recent onset/progressive symptoms; melaena/hematemesis; swallowing difficulty.

Dyspepsia that is worse with empty stomach and eased by eating is the classical symptom of PUD

Differential diagnosis of dyspepsia

- | | | |
|-----------------------|--------------------------|--------------------|
| • Non-ulcer dyspepsia | • Duodenal/gastric ulcer | • Duodenitis |
| • Oesophagitis/GORD | • Gastric malignancy | • Gastritis (p257) |



As per NICE guidance, patients over the age of 55 with recent onset, unexplained, and persistent dyspepsia (over 4–6 weeks) should be referred urgently for endoscopy to exclude cancer.

If there're any alarm symptoms, proceed to do upper endoscopy regardless of age

Lifestyle: Weight loss; smoking cessation; small, regular meals; reduce hot drinks, alcohol, citrus fruits, tomatoes, onions, fizzy drinks, spicy foods, caffeine, chocolate; avoid eating < 3h before bed. Raise the bed head.

Table 6.2 Indications for upper GI endoscopy

Diagnostic indications	Therapeutic indications
Haematemesis/melaena	Treatment of bleeding lesions
Dysphagia	Variceal banding and sclerotherapy
Dyspepsia (≥ 55 yrs old + alarm symptoms or treatment refractory, p252)	Argon plasma coagulation for suspected vascular abnormality
Duodenal biopsy (?coeliac)	Stent insertion, laser therapy
Persistent vomiting	Stricture dilatation, polyp resection
Iron deficiency (cancer)	

if the patient is on aspirin, clopidogrel, warfarin, or DOACS these need stopping only if therapeutic procedure.

Duodenal biopsy: The gold standard test for coeliac disease. also useful in unusual causes of malabsorption, e.g., giardiasis, lymphoma, Whipple's disease.

Peptic Ulcer Disease (PUD):

A peptic ulcer is a defect in the gastric or duodenal mucosa that extends through the muscularis mucosa into the deeper layers of the wall. Peptic ulcers may present with dyspeptic or other gastrointestinal symptoms (abdominal pain), or may be initially asymptomatic (70%) and then present with complications such as hemorrhage or perforation. Acute upper gastrointestinal hemorrhage is the most common complication of PUD. Ulcer perforation should be suspected in patients who suddenly develop severe, diffuse abdominal pain. A classic triad of sudden onset of abdominal pain, tachycardia, and abdominal rigidity is the hallmark of peptic ulcer perforation.

Duodenal ulcer (DU) 4-fold commoner than GU.

Major risk factors: *H. pylori* (90%); drugs (NSAIDs; steroids; SSRI), association with blood group O; smoking.

Symptoms: Asymptomatic or epigastric pain (relieved by antacids)

Signs: Epigastric tenderness.

Diagnosis: Upper GI endoscopy. Test for *H. pylori*. Measure gastrin concentrations when off PPIs if Zollinger–Ellison syndrome is suspected.

DDx: Non-ulcer dyspepsia; duodenal Crohn's; TB; lymphoma; pancreatic cancer

Follow-up: None; if good response to Rx (e.g., PPI).

Gastric ulcers (GU) Occur mainly in the elderly, on the lesser curve of the stomach.

Ulcers elsewhere are more often malignant.

Risk factors: *H. pylori* (~80%); smoking; NSAIDs; reflux of duodenal contents; delayed gastric emptying; **stress ulcer**, e.g., neurosurgery/head trauma or burns (Cushing's or Curling's ulcers).

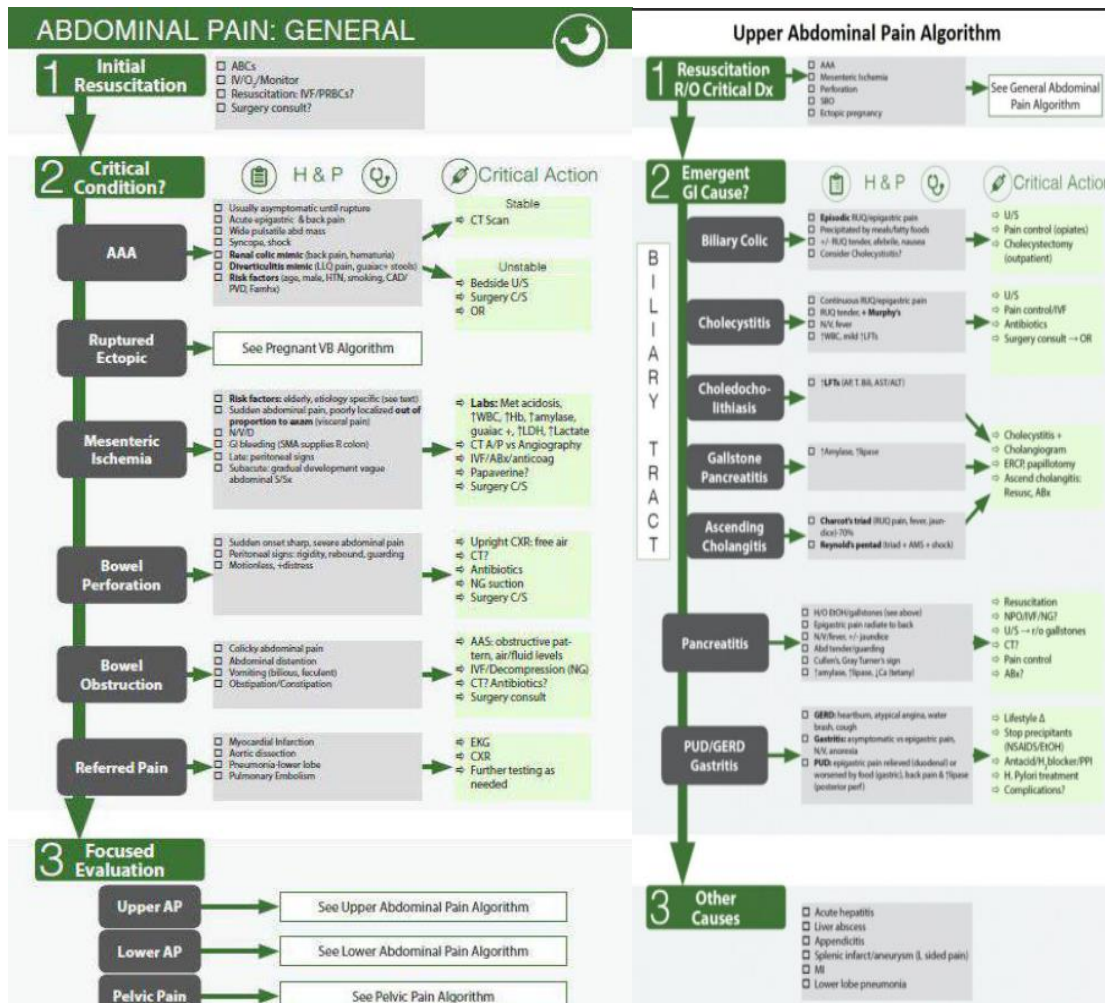
Symptoms: Asymptomatic or epigastric pain (relieved by antacids)

Tests: Upper GI endoscopy to exclude malignancy; multiple biopsies from ulcer rim and base (histology, *H. pylori*). Repeat endoscopy after 6–8 weeks to confirm healing and exclude malignancy.

6. Abdominal Pain

First, Resuscitate & Rule out life threatening/urgent conditions.

Then, you can analyze the pain for non-urgent causes (SOCRATES)



Site: Visceral abdominal pain is poorly localized in the midline (conducted via sympathetic splanchnic nerves)
 Somatic pain from the parietal peritoneum and abdominal is localized to the area of inflammation.
 (Conducted via intercostal (spinal) nerves)
 foregut (stomach, pancreas, liver and biliary system) is localized above the umbilicus.
 Central abdominal pain arises from midgut structures (small bowel and appendix)
 Lower abdominal pain arises from hindgut structures (colon)

Onset, Coarse, Duration

sudden onset of severe abdominal pain, rapidly progressing to become generalized and constant, suggests a hollow viscus perforation (eg perforated PUD), a ruptured abdominal aortic, aneurysm or mesenteric infarction.

Character:

burning or gnawing, as is typical of gastroesophageal reflux and peptic ulcer disease, or colicky, as in the cramping pain of gastroenteritis or intestinal obstruction.

Radiation:

Pain radiating from the right hypochondrium to the shoulder or interscapular region may reflect diaphragmatic irritation in acute cholecystitis

Pain radiating from the loin to the groin and genitalia is typical of renal colic.

Central upper abdominal pain radiating through to the back, partially relieved by sitting forward, is common in pancreatitis.

Central abdominal pain, which later shifts into the right iliac fossa, occurs in acute appendicitis.

The combination of severe back and abdominal pain may indicate a ruptured or dissecting AAA.

Associated symptoms

Other gastrointestinal symptoms – We ask about associated nausea, vomiting, diarrhea, constipation, hematochezia, melena, and changes in stool (eg, change in caliber).

For patients with right upper quadrant pain or concern for liver disease, we also ask about jaundice and changes in the color of urine and stool. The bowel habit is an important part of the history for chronic abdominal pain. Irritable bowel syndrome (IBS) often presents with swings between diarrhea and constipation, a pattern that is much less likely with organic disease.

●**Genitourinary symptoms** – Patients with symptoms such as dysuria, frequency, and hematuria are more likely to have a genitourinary cause for their abdominal pain. Exclude Genitourinary causes in Females

●**Constitutional symptoms** – Symptoms such as fevers, chills, fatigue, weight loss, and anorexia would be concerning for infection, malignancy, or systemic illnesses (eg, inflammatory bowel disease [IBD]).

●**Cardiopulmonary symptoms** – Symptoms such as cough, shortness of breath, orthopnea, and exertional dyspnea suggest a pulmonary or cardiac etiology. Orthostatic hypotension may indicate early shock or be associated with adrenal insufficiency.

Timing (course/duration)

During the first hour or two after perforation, a ‘silent interval’ may occur when abdominal pain resolves transiently. The initial chemical peritonitis may subside before bacterial peritonitis becomes established. Change in the pattern of symptoms suggests either that the initial diagnosis was wrong, or that complications have developed. In acute small-bowel obstruction, a change from typical intestinal colic to persistent pain with abdominal tenderness suggests intestinal ischemia, e.g., strangulated hernia, an indication for urgent surgical intervention.

Exacerbating and relieving factors

Pain exacerbated by movement or coughing suggests inflammation (peritonitis?). Patients tend to lie still in order not to exacerbate the pain.

Patients with colic typically move around or draw their knees up towards the chest during painful spasms. Biliary and renal ‘colic’ are misnamed, as the pain is rarely colicky; pain rapidly increases to a peak intensity and persists over several hours before gradually resolving. Biliary or renal colic is usually promptly relieved by parenteral analgesia.

Causes of epigastric abdominal pain

Epigastric	Clinical features	Comments
Acute myocardial infarction	May be associated with shortness of breath and exertional symptoms.	Consider particularly in patients with risk factors for coronary artery disease.
Acute pancreatitis	Acute-onset, persistent upper abdominal pain radiating to the back.	
Chronic pancreatitis	Epigastric pain radiating to the back.	Associated with pancreatic insufficiency.
Peptic ulcer disease	Epigastric pain or discomfort is the most prominent symptom.	Occasionally, discomfort localizes to one side.
Gastroesophageal reflux disease	Associated with heartburn, regurgitation, and dysphagia.	
Gastritis/gastropathy	Abdominal discomfort/pain, heartburn, nausea, vomiting, and hematemesis.	Variety of etiologies including alcohol and nonsteroidal antiinflammatory drugs (NSAIDs).
Functional dyspepsia	The presence of one or more of the following: postprandial fullness, early satiety, epigastric pain, or burning.	Patients have no evidence of structural disease.
Gastroparesis	Nausea, vomiting, abdominal pain, early satiety, postprandial fullness, and bloating.	Most causes are idiopathic, diabetic, or postsurgical.

Causes of right upper quadrant (RUQ) abdominal pain

RUQ	Clinical features	Comments
Biliary		
Biliary colic	Intense, dull discomfort located in the RUQ or epigastrium. Associated with nausea, vomiting, and diaphoresis. Generally lasts at least 30 minutes, plateauing within one hour. Benign abdominal examination.	Patients are generally well-appearing.
Acute cholecystitis	Prolonged (>4 to 6 hours) RUQ or epigastric pain, fever. Patients will have abdominal guarding and Murphy's sign.	
Acute cholangitis	Fever, jaundice, RUQ pain.	May have atypical presentation in older adults or immunosuppressed patients.
Sphincter of Oddi dysfunction	RUQ pain similar to other biliary pain.	Biliary type pain without other apparent causes.
Hepatic		
Acute hepatitis	RUQ pain with fatigue, malaise, nausea, vomiting, and anorexia. Patients may also have jaundice, dark urine, and light-colored stools.	Variety of etiologies include hepatitis A, alcohol, and drug-induced.
Perihepatitis (Fitz-Hugh-Curtis syndrome)	RUQ pain with a pleuritic component, pain is sometimes referred to the right shoulder.	Aminotransferases are usually normal or only slightly elevated.
Liver abscess	Fever and abdominal pain are the most common symptoms.	Risk factors include diabetes, underlying hepatobiliary or pancreatic disease, or liver transplant.
Budd-Chiari syndrome	Symptoms include fever, abdominal pain, abdominal distention (from ascites), lower extremity edema, jaundice, gastrointestinal bleeding, and/or hepatic encephalopathy.	Variety of causes.
Portal vein thrombosis	Symptoms include abdominal pain, dyspepsia, or gastrointestinal bleeding.	Clinical manifestations depend on extent of obstruction and speed of development. Most commonly associated with cirrhosis.

Causes of left upper quadrant (LUQ) abdominal pain

LUQ	Clinical features	Comments
Splenomegaly	Pain or discomfort in LUQ, left shoulder pain, and/or early satiety.	Multiple etiologies.
Splenic infarct	Severe LUQ pain.	Atypical presentations common. Associated with a variety of underlying conditions (eg, hypercoagulable state, atrial fibrillation, and splenomegaly).
Splenic abscess	Associated with fever and LUQ tenderness.	Uncommon. May also be associated with splenic infarction.
Splenic rupture	May complain of LUQ, left chest wall, or left shoulder pain that is worse with inspiration.	Most often associated with trauma.

Causes of lower abdominal pain

Lower abdomen	Localization	Clinical features	Comments
Appendicitis	Generally right lower quadrant	Periumbilical pain initially that radiates to the right lower quadrant. Associated with anorexia, nausea, and vomiting.	Occasional patients present with epigastric or generalized abdominal pain.
Diverticulitis	Generally left lower quadrant; right lower quadrant more common in Asian patients	Pain usually constant and present for several days prior to presentation. May have associated nausea and vomiting.	Clinical presentation depends on severity of underlying inflammatory process and whether or not complications are present.
Nephrolithiasis	Either	Pain most common symptom, varies from mild to severe. Generally flank pain, but may have back or abdominal pain.	Cause symptoms as stone passes from renal pelvis to ureter.
Pyelonephritis	Either	Associated with dysuria, frequency, urgency, hematuria, fever, chills, flank pain, and costovertebral angle tenderness.	
Acute urinary retention	Suprapubic	Present with lower abdominal pain and discomfort; inability to urinate.	
Cystitis	Suprapubic	Associated with dysuria, frequency, urgency, and hematuria.	
Infectious colitis	Either	Diarrhea as the predominant symptom, but may also have associated abdominal pain, which may be severe.	Patients with <i>Clostridioides difficile</i> infection can present with an acute abdomen and peritoneal signs in the setting of perforation and fulminant colitis.

7. Abdominal distension:

8.11 Causes of abdominal distension	
Factor	Consider
Fat	Obesity
Flatus	Pseudo-obstruction, obstruction
Faeces	Subacute obstruction, constipation
Fluid	Ascites, tumours (especially ovarian), distended bladder
Fetus	Check date of the last menstrual period
Functional	Bloating, often associated with irritable bowel syndrome

8.12 Causes of ascites	
Diagnosis	Comment
Common	
Hepatic cirrhosis with portal hypertension	Transudate
Intra-abdominal malignancy with peritoneal spread	Exudate, cytology may be positive
Uncommon	
Hepatic vein occlusion (Budd–Chiari syndrome)	Transudate in acute phase
Constrictive pericarditis and other right heart failure	Check jugular venous pressure and listen for pericarditic rub
Hypoproteinaemia (nephrotic syndrome, protein-losing enteropathy)	Transudate
Tuberculosis peritonitis	Low glucose content
Pancreatitis	Very high amylase content

Ascites: Fluid collection in the peritoneal cavity

Uncomplicated ascites

Ascites that is not infected and which is not associated with the development of the hepatorenal syndrome. Ascites can be graded as follows:

- Grade 1 (mild). Ascites is only detectable by ultrasound examination.
- Grade 2 (moderate). Ascites causing moderate symmetrical distension of the abdomen.
- Grade 3 (large). Ascites causing marked abdominal distension.

Refractory ascites

Ascites that cannot be mobilized or early recurrence of which (that is, after therapeutic paracentesis) cannot be satisfactorily prevented by medical therapy. This includes two different subgroups.

- Diuretic resistant ascites—ascites that is refractory to dietary sodium restriction and intensive diuretic treatment (spironolactone 400 mg/day and furosemide 160 mg/day for at least one week, and a salt restricted diet of less than 90 mmol/day (5.2 g of salt)/day).
- Diuretic intractable ascites—ascites that is refractory to therapy due to the development of diuretic induced complications that preclude the use of an effective diuretic dosage.

To determine the cause of Ascites, you need to analyze the ascitic fluid for:

1. **Cell count with differential**

corrected neutrophil count $\geq 250/\text{mm}^3$ PMN cells= SBP, you have to do C&S and start Antibiotic.

The WBC and neutrophil counts need to be corrected in patients with bloody samples (traumatic). One WBC should be subtracted from the WBC count for every 750 red blood cells to yield the "corrected white blood cell count," and one neutrophil should be subtracted from the absolute neutrophil count for every 250 red blood cells to yield the "corrected neutrophil count"

2. **Total protein**

Ascitic fluid can be classified as an exudate if the total protein concentration is ≥ 2.5 or 3 g/dL and a transudate if it is below this cutoff.

3. **Albumin (SAAG)**

The serum-to-ascites albumin gradient identifies the presence of portal HTN or not

Table 1-15: Causes of Ascites and Associated Findings		
Albumin and Protein in Ascites		
Causes	SAAG	Ascites T. protein
Cirrhosis, liver failure, Budd-Chiari syndrome, myxedema, and SBP	> 1.1	< 2.5
Right heart failure	> 1.1	> 2.5
TB peritonitis, bacterial/fungal peritonitis, nephrotic syndrome, pancreatitis, and peritoneal carcinomatosis	< 1.1	> 2.5

SAAG = Albumin in serum – Albumin in Ascitic fluid

If > 1.1 then the ascites is due to portal HTN

If < 1.1 meaning there's high level of Albumin in the ascitic fluid and no portal HTN

8. GI Bleeding

UGIB: may present Hematemesis (vomiting of fresh blood or degraded brown coffee ground) or Melena (black, tarry, offensive stool)

Oral iron and Bismuth can also cause black stool.

8.19 Causes of upper gastrointestinal bleeding	
• Gastric or duodenal ulcer • Mallory–Weiss oesophageal tear • Oesophagitis, gastritis, duodenitis	• Oesophagogastric varices • Oesophageal or gastric cancer • Vascular malformation

Peptic ulcer is the most common cause of upper GI bleeding (50%) and an important cause of massive hemorrhage.

LGIB: Hematochezia: passage of Fresh rectal bleeding and indicates a disorder in the anal canal, rectum or colon. **However**, 15% of cases result from massive upper GI bleeding.

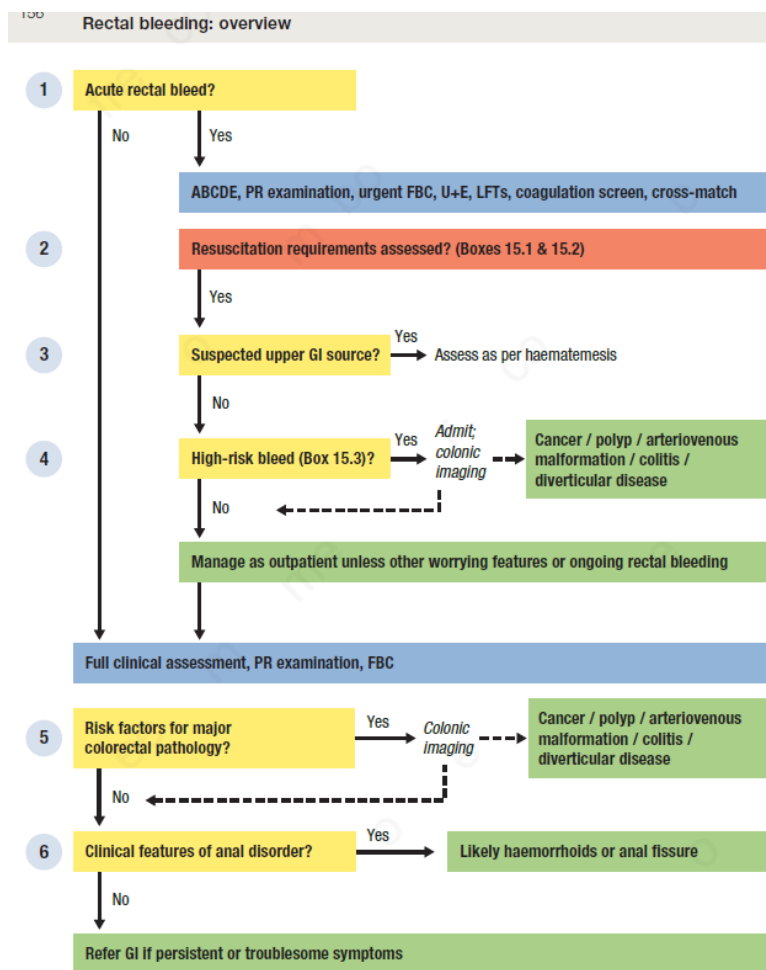
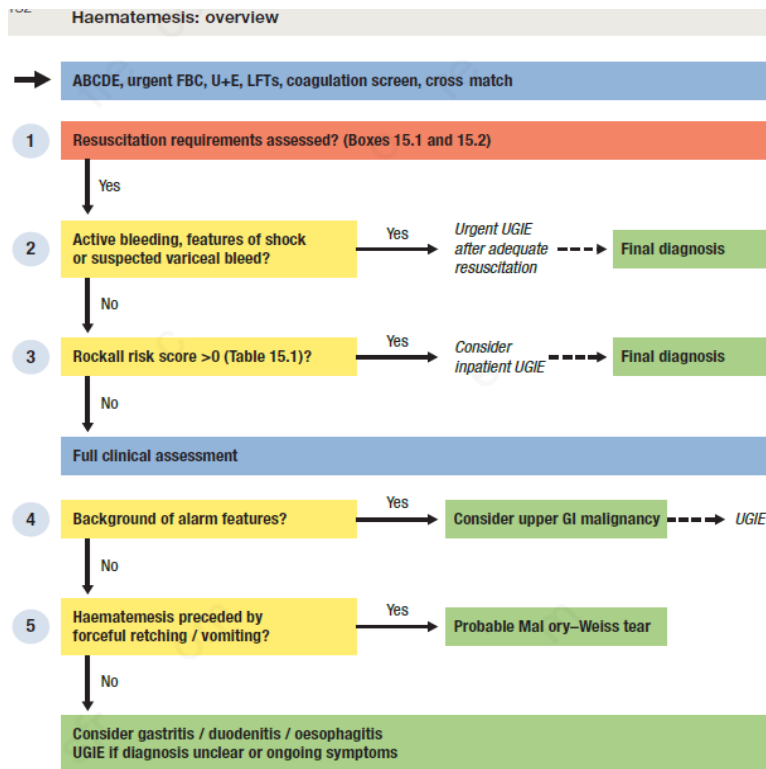
8.21 Causes of rectal bleeding	
• Haemorrhoids • Anal fissure • Colorectal polyps • Colorectal cancer • Inflammatory bowel disease	• Ischaemic colitis • Complicated diverticular disease • Vascular malformation

Anorectal bleeding (hemorrhoids, anal fissure, perianal Crohn's disease and anal cancer) typically presents with intermittent episodes of minor, fresh bright red bleeding during or after defecation, which is not mixed with the stools.

Colon bleeding: stool mixed with blood

Diverticular disease is the most common cause of severe acute rectal bleeding and typically occurs in older patients.

Angiodysplasia (arteriovenous malformation) an important cause of severe lower GI bleeding



Box 15.1 Resuscitation in acute haemorrhage

First steps

- A+B before C: recheck airway frequently, especially if ↓GCS/vomiting. Give high concentration O₂
- Get help: one pair of hands is not enough. If necessary call the medical emergency or cardiac arrest team

Secure IV access and monitoring

- 2 large bore cannulae in antecubital veins or equivalent
- Central venous cannulation, e.g. femoral line if peripheral access difficult
- Get expert help immediately if you cannot obtain adequate access
- Urgent FBC, U+E, LFTs, coagulation screen and cross-match (Inform blood bank)
- Measure pulse, BP, RR, GCS and reassess peripheral perfusion every 10–15 min
- Insert urinary catheter; monitor hourly urine output

Resuscitate and reassess

Early shock features (see Box 15.2) or any ongoing blood loss:

- 0.5–1 L stat of IV crystalloid or colloid
- 2 units of red cells if Hb <8 g/dL, ongoing bleeding or >2 L IV crystalloid or colloid given

Reassess

- If advanced shock features or major blood loss on reassessment, resuscitate as per the adjacent column.
- Otherwise, continue with treatment and discuss with haematologist if any of the following:
 - known coagulopathy/anticoagulant therapy
 - INR >1.4
 - platelets <100 × 10⁹/L
 - fibrinogen <1 g/L
 - ≥4 L IV fluid in any form

Advanced shock features (see Box 15.2) or ongoing major blood loss:

- 1–2 L stat of IV crystalloid
- use red cells as soon as available
- consider O negative blood if significant delay in cross-matching (use group specific blood as soon as possible)

Reassess

- If ongoing advanced shock features or major blood loss, activate the major haemorrhage protocol: administer red cell concentrate (RCC) and fresh frozen plasma (FFP) and consider platelets and cryoprecipitate.
- Seek specialist input, e.g. ICU, GI, surgical, as appropriate and discuss haemostatic support, with haematologist.

Box 15.2 Clinical features of hypovolaemic shock

Early

- HR >100 bpm
- Capillary refill time (CRT) >2s
- Narrow pulse pressure
- ↓Postural BP
- Pale, sweaty, anxious, thirsty

Advanced

- Systolic BP <100 mmHg (or ↓40 mmHg from baseline)
- HR >120 bpm
- RR >20 breaths/min
- Cold, mottled peripheries
- Confused, lethargic, ↓GCS

Rockall score in UGIB: predicts risk of rebleeding and death pre-endoscopic score of 0 predicts an extremely low risk of death (0.2%) and rebleeding (0.2%), but predicted mortality rises to 2.4% for a score of 1 and 5.6% for a score of 2. Admit patients with a pre-endoscopy Rockall score >0 for further assessment and observation

Table 15.1 Rockall risk score

Variable	Score			
	0	1	2	
Age	<60 years	60–79 years	≥80 years	Pre endoscopy: Initial score criteria
Shock	'No shock', systolic BP ≥100 mmHg, pulse <100 bpm	'Tachycardia', systolic BP ≥100 mmHg, pulse ≥100 bpm	'Hypotension', systolic BP <100 mmHg	
Comorbidity	No major comorbidity		Cardiac failure, ischaemic heart disease, any major comorbidity	Post-endoscopy: Additional criteria for full score
			Renal failure, liver failure, disseminated malignancy	
Diagnosis	Mallory–Weiss tear, no lesion identified and no stigmata of recent haemorrhage	All other diagnoses	Upper GI tract cancer	
Major stigmata of recent haemorrhage	None, or dark spot only		Blood in upper GI tract, adherent clot, visible or spurting vessel	

9. altered bowel habit

Normal bowel habit ranges from 3 stools/day to once every 3 days

Diarrhea: frequent passage of loose stool

Bloody diarrhea may be due to inflammatory bowel disease, colonic ischemia or infective gastroenteritis.

8.13 Causes of diarrhoea	
Acute	
• Infective gastroenteritis, e.g. <i>Clostridium difficile</i>	• Drugs (especially antibiotics)
Chronic (>4 weeks)	
• Irritable bowel syndrome • Inflammatory bowel disease • Parasitic infestations, e.g. <i>Giardia lamblia</i>, amoebiasis, <i>Cryptosporidium</i> spp. • Colorectal cancer • Autonomic neuropathy (especially diabetic) • Laxative abuse and other drug therapies	• Hyperthyroidism • Constipation and faecal impaction (overflow) • Small-bowel or right colonic resection • Malabsorption, e.g. lactose deficiency, coeliac disease

High volume Diarrhea (>1 L/day): stool water content is increased (the principal site of water absorption being the colon) and may be:

- secretory, due to intestinal inflammation, e.g., infection, or IBD. Fasting doesn't stop secretory diarrhea
- osmotic, due to malabsorption, adverse drug effects, or motility disorders. Osmotic diarrhea stops when the patient fasts.

Low-volume diarrhea is associated with the irritable bowel syndrome. IBS doesn't wake the pt from sleep.

Questions to ask?

Acute or chronic? If acute (<2wks) suspect gastroenteritis—any risk factors: Travel? Diet change? Contact with D&V? Any fever/pain. Chronic diarrhea alternating with constipation suggests irritable bowel S.

Weight loss, nocturnal diarrhea, or anemia mandate close follow-up (coeliac/UC/Crohn's?)

Bloody diarrhea: *Campylobacter*, *Shigella*/*Salmonella*, *E. coli*, amoebiasis, UC, Crohn's, colorectal cancer, colonic polyps, pseudomembranous colitis (*C. difficile* after use of broad spectrum antibiotics), ischemic colitis.

Mucus: Occurs in IBS, colorectal cancer, and polyps.

Frank pus: Suggests IBD, diverticulitis, or a fistula/abscess.

Explosive: cholera; *Giardia*; *Yersinia*; *Rotavirus*.

Steatorrhea: Characterized by gas, offensive smell, and floating, hard-to-flush stools—consider pancreatic insufficiency or biliary obstruction.

Constipation

the passage of ≤ 2 bowel motions/wk often passed with difficulty, straining, or pain, and a sense of incomplete evacuation.

8.17 Causes of constipation	
• Lack of fibre in diet • Irritable bowel syndrome • Intestinal obstruction (cancer) • Drugs (opioids, iron)	• Metabolic/endocrine (hypothyroidism, hypercalcaemia) • Immobility (stroke, Parkinson's disease)

10. Jaundice:

Jaundice refers to yellowing of skin, sclerae, and mucosae from high plasma bilirubin. Jaundice is classified by the site of the problem (prehepatic, hepatocellular, or cholestatic/obstructive) or by the type of circulating bilirubin (conjugated or unconjugated).

8.22 Common causes of jaundice	
Increased bilirubin production	
• Haemolysis (unconjugated hyperbilirubinaemia)	
Impaired bilirubin excretion	
• Congenital • Gilbert's syndrome (unconjugated) • Hepatocellular • Viral hepatitis • Cirrhosis • Drugs • Autoimmune hepatitis	• Intrahepatic cholestasis • Drugs • Primary biliary cirrhosis • Extrahepatic cholestasis • Gallstones • Cancer: pancreas, cholangiocarcinoma

Box 19.1 Causes of haemolysis	Box 19.2 Causes of acute liver failure
Hereditary • Erythrocyte membrane defects • Hereditary spherocytosis • Hereditary elliptocytosis • Erythrocyte enzyme defects • Glucose-6-phosphate dehydrogenase deficiency • Haemoglobinopathies • Thalassaemia • Sickle cell	Drugs/toxins • Paracetamol overdose¹ • Anti-tuberculous drugs • Ecstasy • Halothane • <i>Amanita phalloides</i> • Carbon tetrachloride
Acquired • Immune • Autoimmune haemolytic anaemia • Alloimmune: haemolytic transfusion reaction, haemolytic disease of the newborn • Fragmentation syndromes • Microangiopathic haemolytic anaemia, e.g. haemolytic uraemic syndrome/thrombotic thrombocytopenic purpura • Mechanical heart valves • Drugs, e.g. dapsone • Infections, e.g. malaria • Paroxysmal nocturnal haemoglobinuria	Infection • Acute viral hepatitis (A, B, E)¹ • Cytomegalovirus (CMV), Epstein–Barr virus (EBV)
	Vascular • Shock/ischaemic hepatitis • Budd–Chiari syndrome
	Other • Wilson's disease • Autoimmune hepatitis • Acute fatty liver of pregnancy • Extensive malignant infiltration

¹Denotes common cause.

11. Weight Loss:

Clinically important weight loss is generally defined as loss of more than 5 percent of usual body weight over 6 to 12 months.