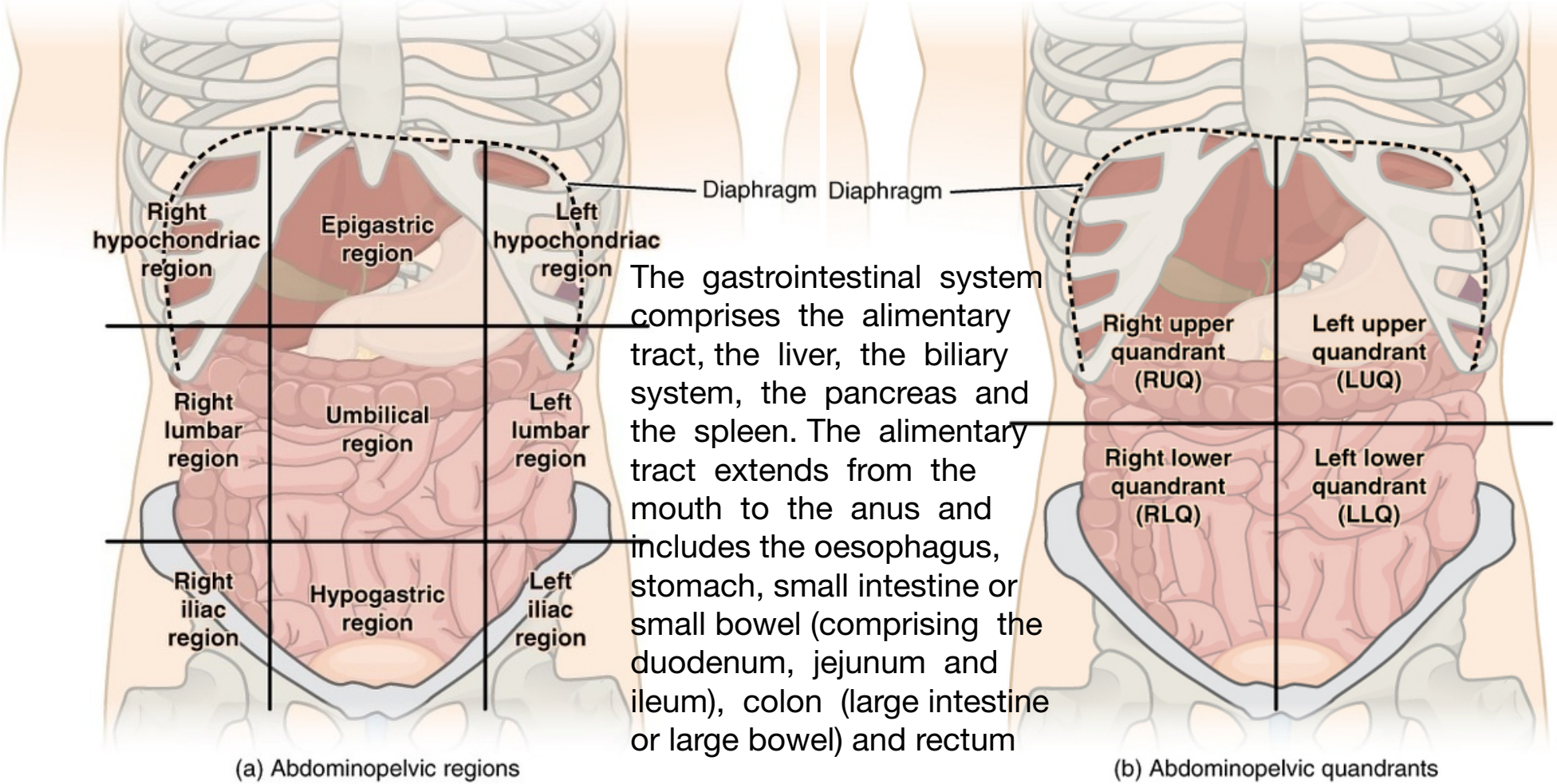
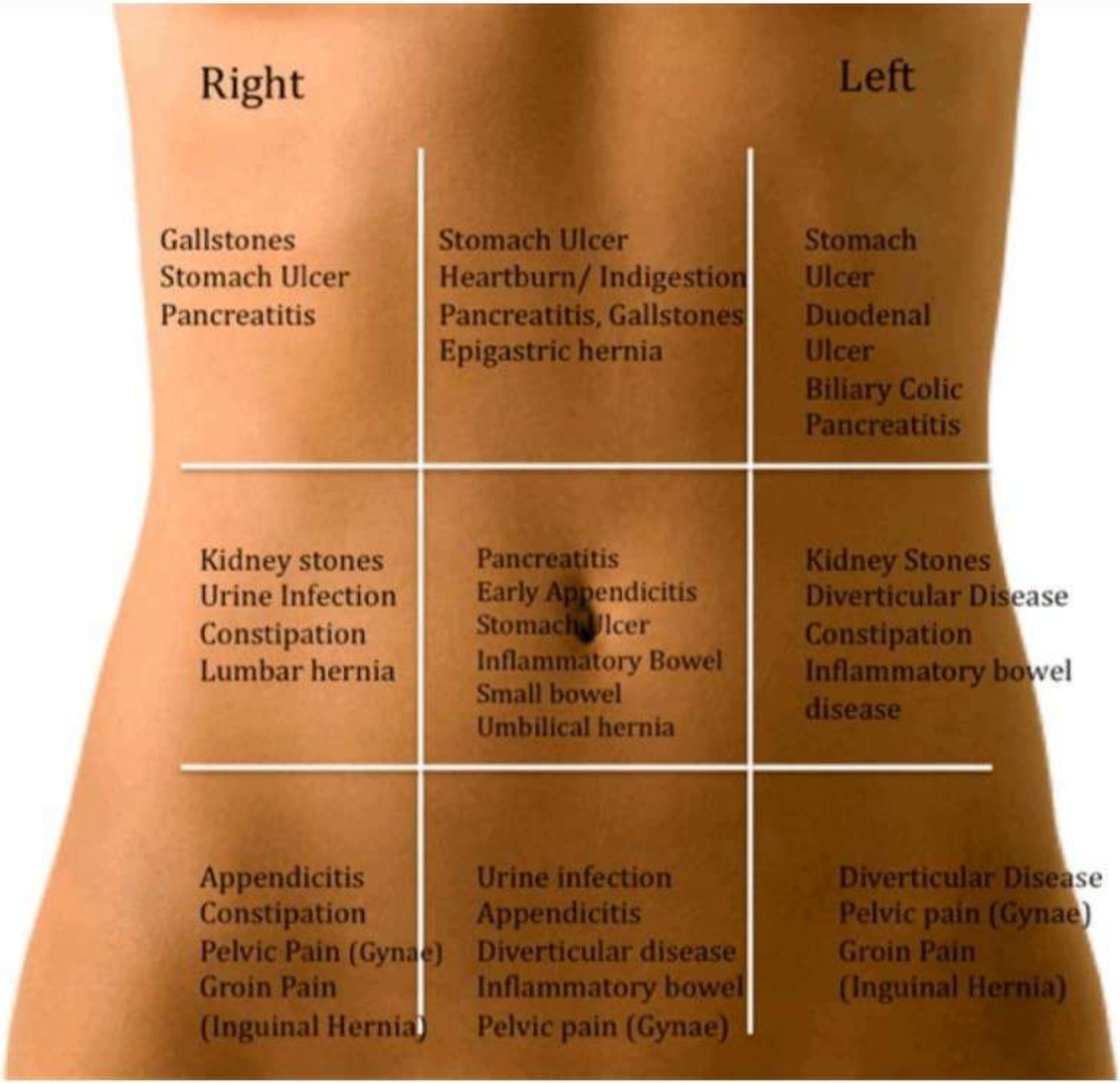
Gastrointestinal System History Taking

Surface Anatomy



The gastrointestinal system comprises the alimentary tract, the liver, the biliary system, the pancreas and the spleen. The alimentary tract extends from the mouth to the anus and includes the oesophagus, stomach, small intestine or small bowel (comprising the duodenum, jejunum and ileum), colon (large intestine or large bowel) and rectum


Right
Left
Gallstones
Stomach Ulcer
Pancreatitis
Stomach Ulcer
Heartburn/ Indigestion
Pancreatitis, Gallstones
Epigastric hernia
Stomach
Ulcer
Duodenal
Ulcer
Biliary Colic
Pancreatitis
Kidney stones
Urine Infection
Constipation
Lumbar hernia
Pancreatitis
Early Appendicitis
Stomach Ulcer
Inflammatory Bowel
Small bowel
Umbilical hernia
Kidney Stones
Diverticular Disease
Constipation
Inflammatory bowel
disease
Appendicitis
Constipation
Pelvic Pain (Gynae)
Groin Pain
(Inguinal Hernia)
Urine infection
Appendicitis
Diverticular disease
Inflammatory bowel
Pelvic pain (Gynae)
Diverticular Disease
Pelvic pain (Gynae)
Groin Pain
(Inguinal Hernia)

Surface Anatomy, cont.

Nine abdominal regions

Horizontal planes

Lines drawn between easily palpable bony points. The horizontal planes are also of importance as they provide useful landmarks on cross-sectional imaging. The two horizontal lines are:

Transpyloric at the level of L1 vertebra

Transtubercular plane: a line uniting the two tubercles of the iliac crests, upper border of L5 vertebra and the confluence of the common iliac veins (i.e. IVC origin) lie on this plane

Vertical planes

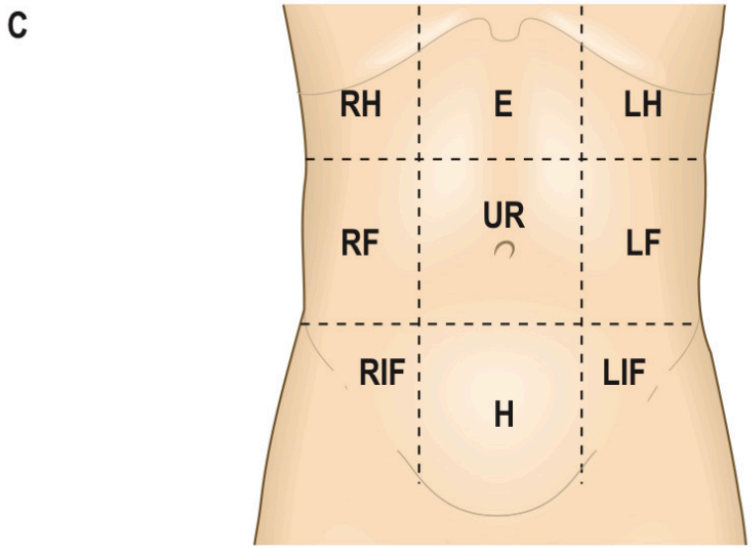
Lines from midclavicular point to the midinguinal point.

Four Quadrants

Horizontal plane: line drawn through the umbilicus.

Vertical plane: midline of the body, overlying the linea alba from the xiphoid to the pubic symphysis.

Surface Anatomy, cont.



1 Oesophagus
2 Stomach
3 Pyloric antrum
4 Duodenum
5 Duodenojejunal flexure
6 Terminal ileum
7 Caecum
8 Appendix (in pelvic position)
9 Ascending colon
10 Transverse colon
11 Descending colon
12 Sigmoid colon

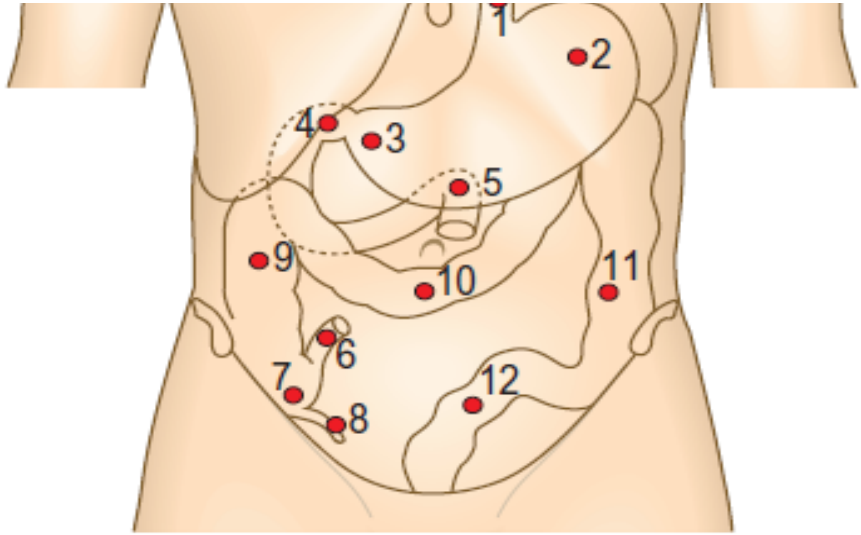
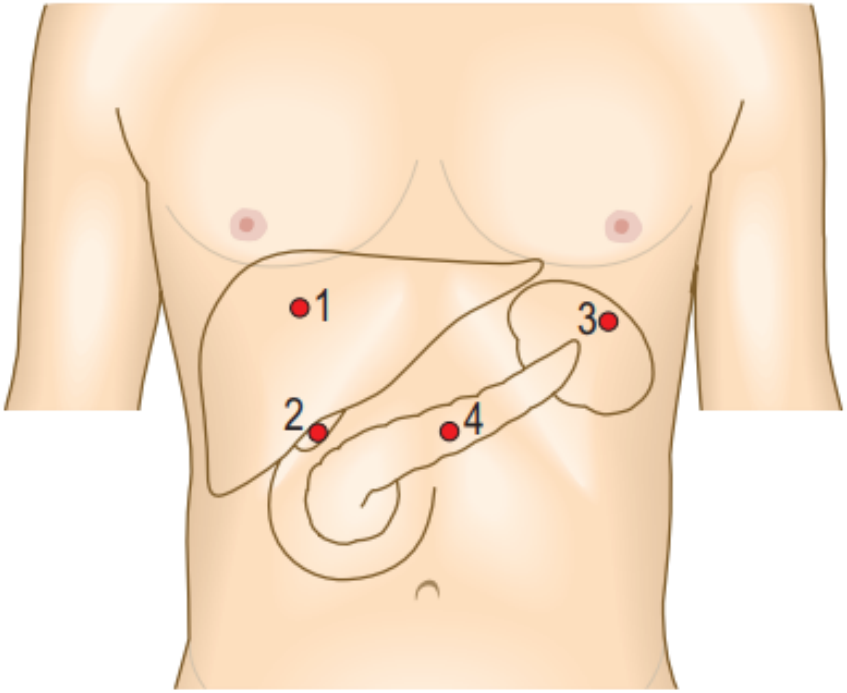


Fig. 6.1 Surface anatomy. A Abdominal surface markings of non-alimentary tract viscera. B Surface markings of the alimentary tract. C Regions of the abdomen. *E,* epigastrium; *H,* hypogastrium or suprapubic region; *LF,* left flank or lumbar region; *LH,* left hypochondrium; *LIF,* left iliac fossa; *RF,* right flank or lumbar region; *RH,* right hypochondrium; *RIF,* right iliac fossa; *UR,* umbilical region.

Surface Anatomy, cont.



1 Liver
2 Gallbladder
3 Spleen
4 Pancreas

6.1 Surface markings of the main non-alimentary tract abdominal organs

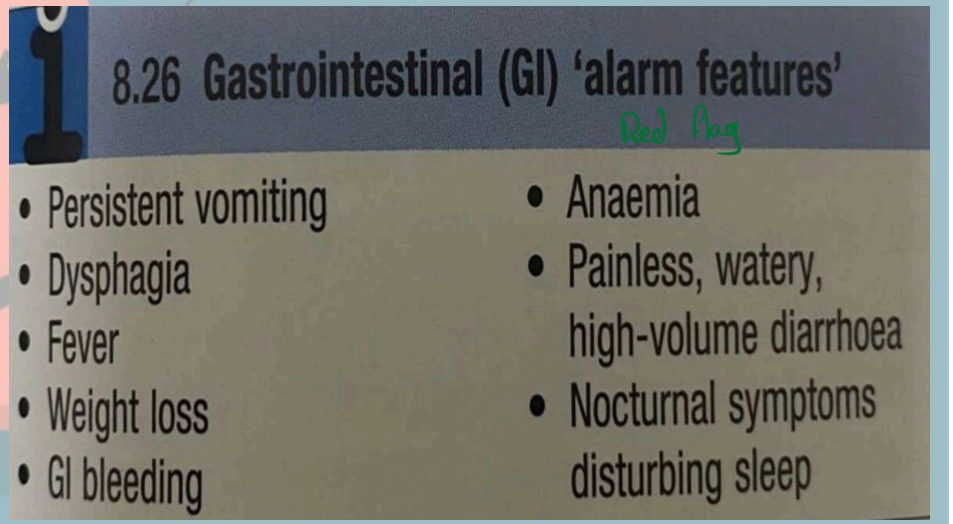
Structure	Position
Liver	Upper border: fifth right intercostal space on full expiration Lower border: at the costal margin in the mid-clavicular line on full inspiration
Spleen	Underlies left ribs 9–11, posterior to the mid-axillary line
Gallbladder	At the intersection of the Right midclavicular line and the costal margin, i.e. tip of the ninth costal cartilage
Pancreas	Neck of the pancreas lies at the level of L1; head lies below and right; tail lies above and left
Kidneys	Upper pole lies deep to the 12th rib posteriorly, 7 cm from the midline; the right is 2–3 cm lower than the left

Q

Gastrointestinal system presenting symptoms

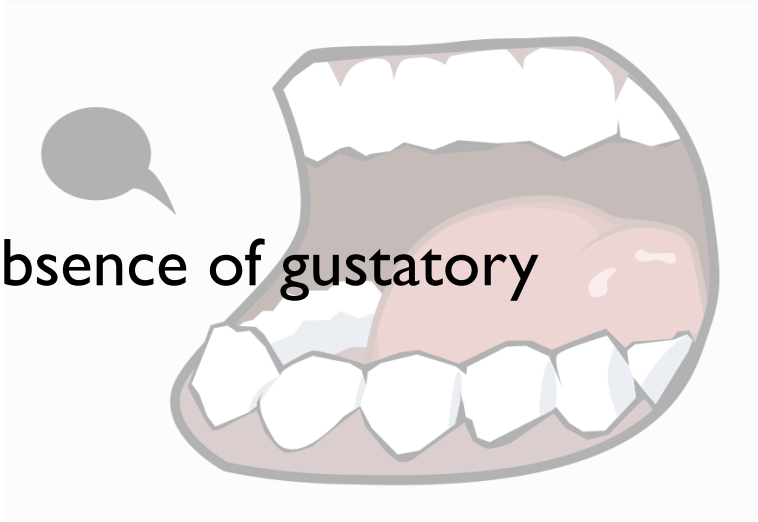
Gastrointestinal symptoms are common and are often caused by functional dyspepsia and irritable bowel syndrome. Symptoms suggesting a serious alternative or coexistent diagnosis include persistent vomiting, dysphagia, gastrointestinal bleeding, weight loss, painless, watery, high-volume diarrhoea, nocturnal symptoms, fever and anaemia. The risk of serious disease increases with age

Always explore the patient's ideas, concerns and expectations about the symptoms



Mouth Symptoms

- Halitosis: Bad breath; gingival, dental or pharyngeal infection
- Xerostomia: Subjective sensation of dryness in the mouth.
  - Diabetes, dehydration, salivary gland dysfunction , sjogren
- Dysgeusia: altered sense of taste
  - Intra-oral infection, zinc deficiency
- Cacogeusia: A sensation of bad taste in absence of gustatory stimuli
  - Amyloidosis

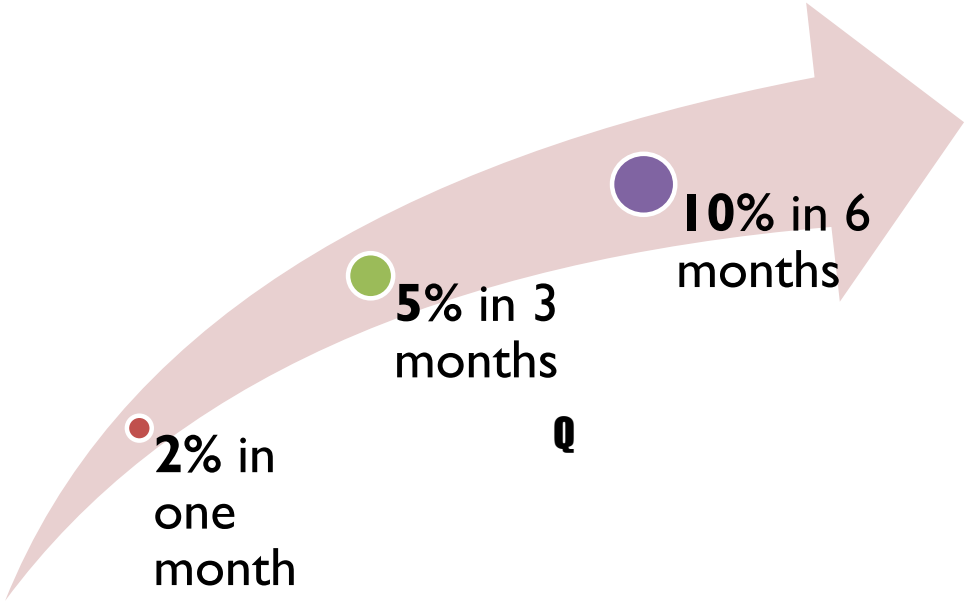


Anorexia And Weight Loss

- Anorexia: loss of appetite and/or lack of interest in food.
  "Are you abstaining from food intake to avoid abdominal pain or because you have no appetite?"
- Weight loss: 3 parts if documented: How much was lost, duration, intentional or not?
- If not documented, has their clothes become loose fitting?

Weight loss

- Reduced intake vs. increased energy expenditure
- GI diseases that cause weight loss: chronic inflammatory diseases and malabsorptive disorders (e.g. IBD, celiac disease), malignancies. Liver disease



Weight loss, cont.

Energy requirements average 2500 kcal/day for males

- A net calorie deficit of 1000 kcal/day produces a weight loss of approximately 1 kg/week .

- Greater weight loss during the initial stages of energy restriction arises from salt and water loss and depletion of hepatic glycogen stores, and not from fat loss.

- Rapid weight loss over days suggests loss of body fluid as a result of vomiting, diarrhea or diuretic therapy.

(1 L of water = 1 kg)

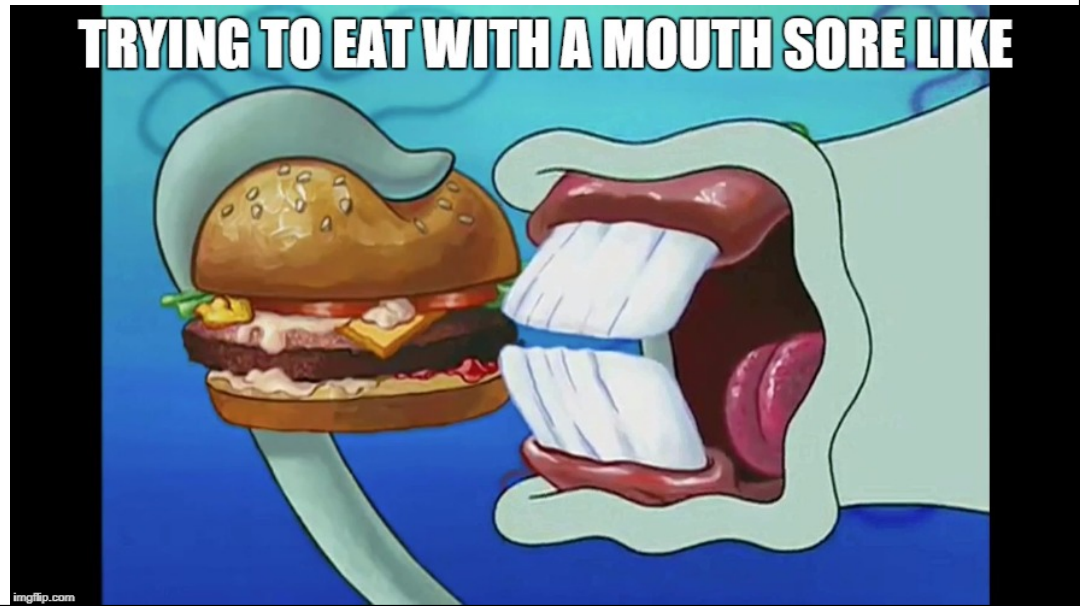
3) pain

Painful Mouth

Causes of sore lips, tongue or buccal mucosa

- Deficiencies; Iron, Folate, Vitamin B12, Vitamin C deficiency
- Aphthous ulcer; idiopathic, maybe asociated with Crohn's or Behcet disease.
- Dermatological (lichen planus)
- Chemotherapy (Oral mucositis)
- Infective stomatitis

- inammatory bowel disease and coeliac disease,
associated with mouth ulcers.



Painful Mouth, cont.

Angular Stomatitis

Aphthous Ulcer

Heartburn And Reflux

- Heartburn is a hot, burning, retrosternal discomfort that radiates upwards.
- When heartburn is the principal symptom, GERD is the most likely diagnosis

character of pain: burning
precipitating factors: lying at or bending forward

- Associated symptoms:
  - Reflux is a sour taste in the mouth from regurgitating gastric acid.
  - Waterbrash is the sudden appearance of fluid in the mouth due to reflex salivation as a result of GERD or, rarely, peptic ulcer disease.
  - atypical (extraesophageal) symptoms, such as; coughing and/or wheezing, hoarseness, sore throat, otitis media

عسر الهضم Dyspepsia

- ***Pain or discomfort*** centered in the upper abdomen.
- Indigestion: vague, ill-defined symptoms

Ask about:

- site of pain
- character of pain
- exacerbating and relieving factors, such as food and antacid
- associated symptoms, such as nausea, belching, bloating and premature satiety.

Reflux-like dyspepsia

- (heartburn-predominant dyspepsia)

Ulcer-like dyspepsia*

- (epigastric pain relieved by food or antacids)

Dysmotility-like dyspepsia (gastroparesis, DM)

- (nausea, belching, bloating and premature satiety).

Patients below the age of 55 without alarm symptoms and with a negative Helicobacter pylori test can be positively diag- nosed as having functional dyspepsia thus avoiding unnecessary investigations but if symptoms persist then further investigations should be considered. However, in patients over the age of 55 organic pathology should always be excluded by upper gastro- intestinal (GI) endoscopy.

Dyspepsia

Peptic ulcer disease

	Gastric ulcer	Duodenal ulcer
PAIN	Can be Greater with meals—weight loss	Decreases with meals—weight gain
H PYLORI INFECTION	~ 70%	~ 90%
MECHANISM	↓ mucosal protection against gastric acid	↓ mucosal protection or ↑ gastric acid secretion
OTHER CAUSES	NSAIDs	Zollinger-Ellison syndrome
RISK OF CARCINOMA	↑	Generally benign
OTHER	Biopsy margins to rule out malignancy	Hypertrophy of Brunner glands

Often there is no structural cause and the dyspepsia is functional. There is considerable overlap, however, and it is impossible to diagnose functional dyspepsia on history alone without investigation. Dyspepsia that is worse with an empty stomach and eased by eating is typical of peptic ulceration. The patient may indicate a single localised point in the epigastrium (pointing sign), and complain of nausea and abdominal fullness that is worse after fatty or spicy meals. 'Fat intolerance' is common with all causes of dyspepsia, including gallbladder disease.

Odynophagia

- Pain upon swallowing.
- It can be present with or without dysphagia
- Often precipitated by drinking hot liquids.
- It indicates active oesophageal ulceration or oesophagitis from GERD or oesophageal candidiasis (inhaled corticosteroid use or in immunocompromised).

It implies intact mucosal sensation, making oesophageal cancer unlikely.

Dysphagia

- Dysphagia is difficulty swallowing.
- Patients complain from food or drinks getting stuck when they swallow
- Oral vs esophageal
- Not Early satiety: premature fullness
- Not Globus: feeling of lump that does not interfere with swallowing and is not related to eating

Dysphagia, cont.

- Site: Where they feel the food sticks; unreliable
- Onset: recent or longstanding
- Character: Liquids vs. Solids.
- Timing: Progressive vs. intermittent.
- Associated symptoms (odynophagia, regurgitation of food or fluids
- wt loss, heartburn)

Neurological dysphagia resulting from bulbar or pseudobulbar palsy is worse for liquids than solids and may be accompanied by choking, spluttering and fluid regurgitating from the nose.

Neuromuscular dysphagia, or oesophageal dysmotility, presents in middle age, is worse for solids and may be helped by liquids and sitting upright.

Achalasia, when the lower oesophageal sphincter fails to relax normally, leads to progressive oesophageal dilatation above the sphincter.

Overflow of secretions and food into the respiratory tract may then occur, especially at night when the patient lies down, causing aspiration pneumonia.

Oesophageal dysmotility can cause oesophageal spasm and central chest pain, which may be confused with cardiac pain.

A pharyngeal pouch may cause food to stick or be regurgitated, and may lead to recurrent chest infections due to chronic silent aspiration.

'Mechanical' dysphagia is often due to oesophageal stricture but can be caused by external compression.

With weight loss, a shorthistory and no reflux symptoms, suspect oesophageal cancer.

Longstanding dysphagia without weight loss but accompanied by heartburn is more likely to be due to benign peptic stricture. Record the site at which the patient feels the food sticking; this is not a reliable guide to the site of oesophageal obstruction, however.

Eosinophilic oesophagitis is the most common cause of food bolus obstruction and should be considered in younger patients with dysphagia; it is associated with atopy and food allergy.
If dysphagia is experienced high in the neck, consider tumours of the pharynx or larynx or extrinsic compression from a mass lesion such as a thyroid goitre.

Dysphagia, cont.

Neurological; bulbar or pseudobulbar palsy

- liquids > solids , choking , spluttering , regurge from nose

Neuromuscular; dysmotility

- worse for solids , improves with liquid and setting upright. Can cause central chest pain, aspiration pneumonia

Pharyngeal pouch:

- Halitosis , recurrent chest infections (aspiration)

Mechanical; stricutre or external compression

- benign vs malignant , ask about associated symptoms

8.7 Causes of dysphagia

Oral

- Tonsillitis, glandular fever, pharyngitis, peritonsillar abscess
- Painful mouth ulcers

Neurological

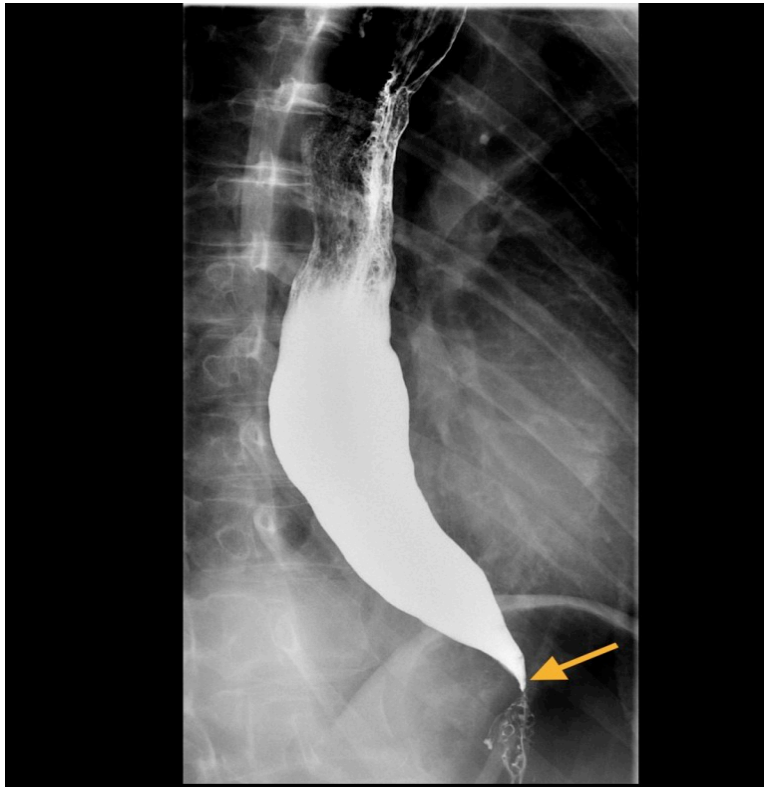
- Bulbar or pseudobulbar palsy
- Cerebrovascular accident

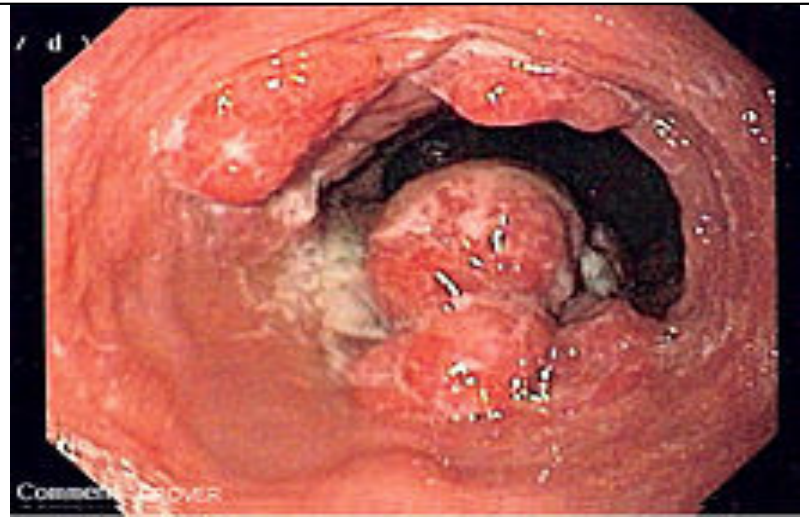
Neuromuscular

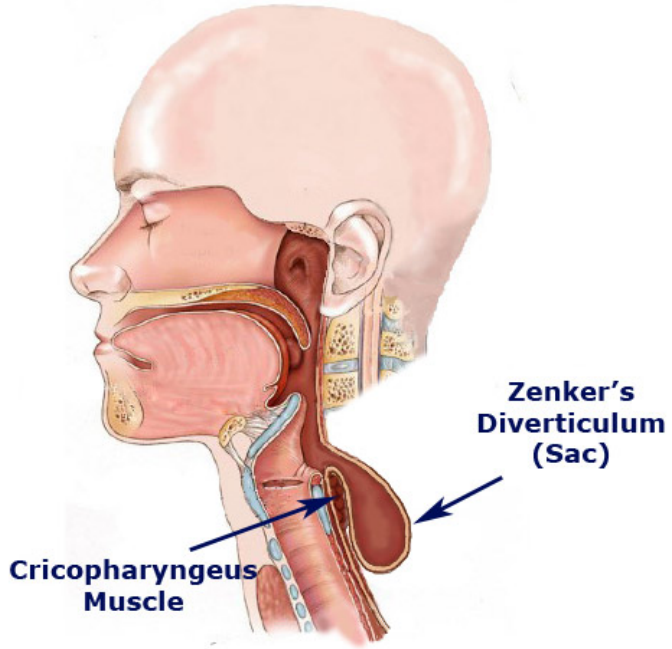
- Achalasia
- Pharyngeal pouch
- Myasthenia gravis
- Oesophageal dysmotility

Mechanical

- Oesophageal cancer
- Peptic oesophagitis
- Other benign strictures, e.g. after prolonged nasogastric intubation
- Extrinsic compression, e.g. lung cancer
- Systemic sclerosis






Zenker's
Diverticulum
(Sac)
Cricopharyngeus
Muscle

Abdominal Pain

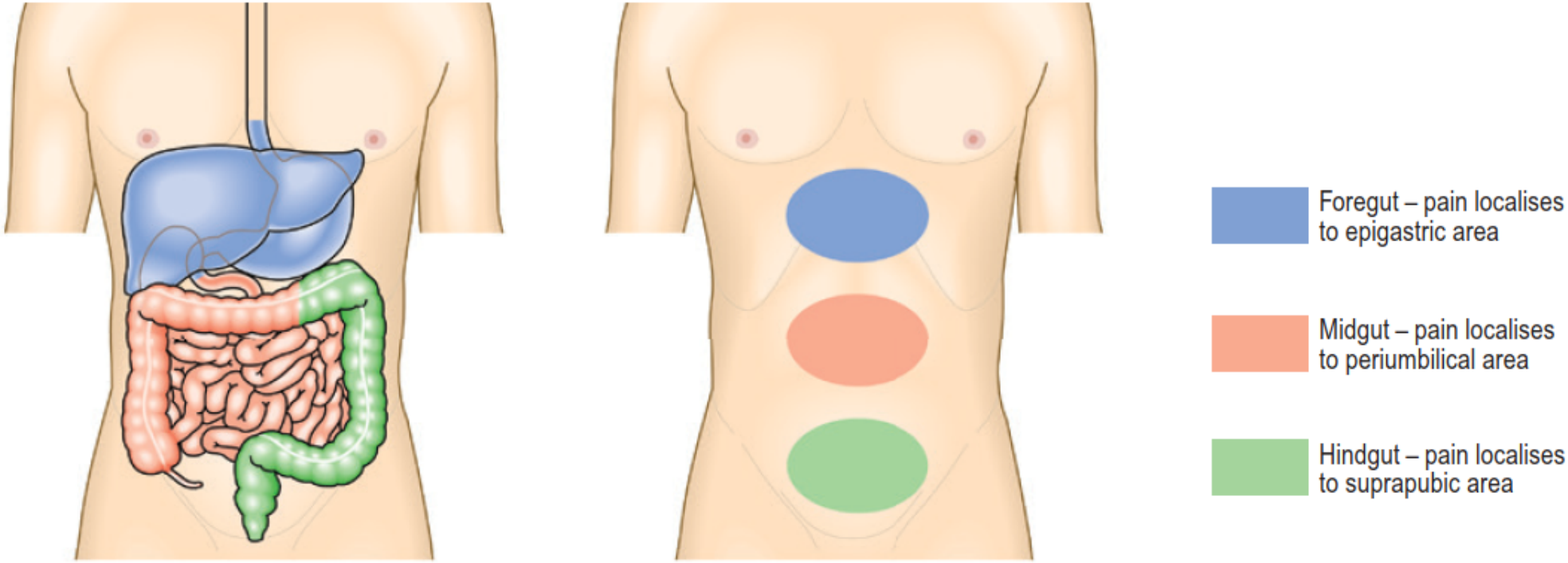


Fig. 6.4 Abdominal pain. Perception of visceral pain is localised to the epigastric, umbilical or suprapubic region, according to the embryological origin of the affected organ.

Site

Visceral Vs. Somatic Pain

Visceral abdominal pain

- Arises from *visceral peritoneum*, distension of hollow organs, mesenteric traction or excessive smooth-muscle contraction
- It is deep and poorly localized in the midline.
- It is conducted via sympathetic splanchnic nerves.

Somatic pain

- Arises from the *parietal peritoneum* and abdominal wall.
- It is lateralised and localised to the area of inflammation, and conducted via *intercostal* (spinal) nerves.
- Examples: cholecystitis , appendicitis, diverticulitis..

Abdominal pain, Cont.

- Pain from paired structures, such as renal colic, is felt on and radiates to the affected side.
- Ureteric colic, although lateralized and severe pain, is considered visceral pain.
- Other paired structures: Testes, ovaries, ovarian tubes

Pain from an unpaired structure, such as the pancreas, is midline and radiates through to the back. Pain from paired structures, such as renal colic, is felt on and radiates to the affected side . Torsion of the testis may present with abdominal pain . In females, consider gynaecological causes like ruptured ovarian cyst, pelvic inammatory disease, endometriosis or ectopic pregnancy.

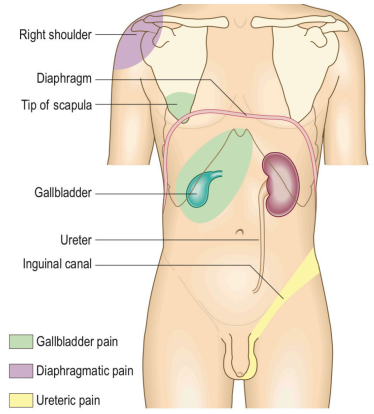
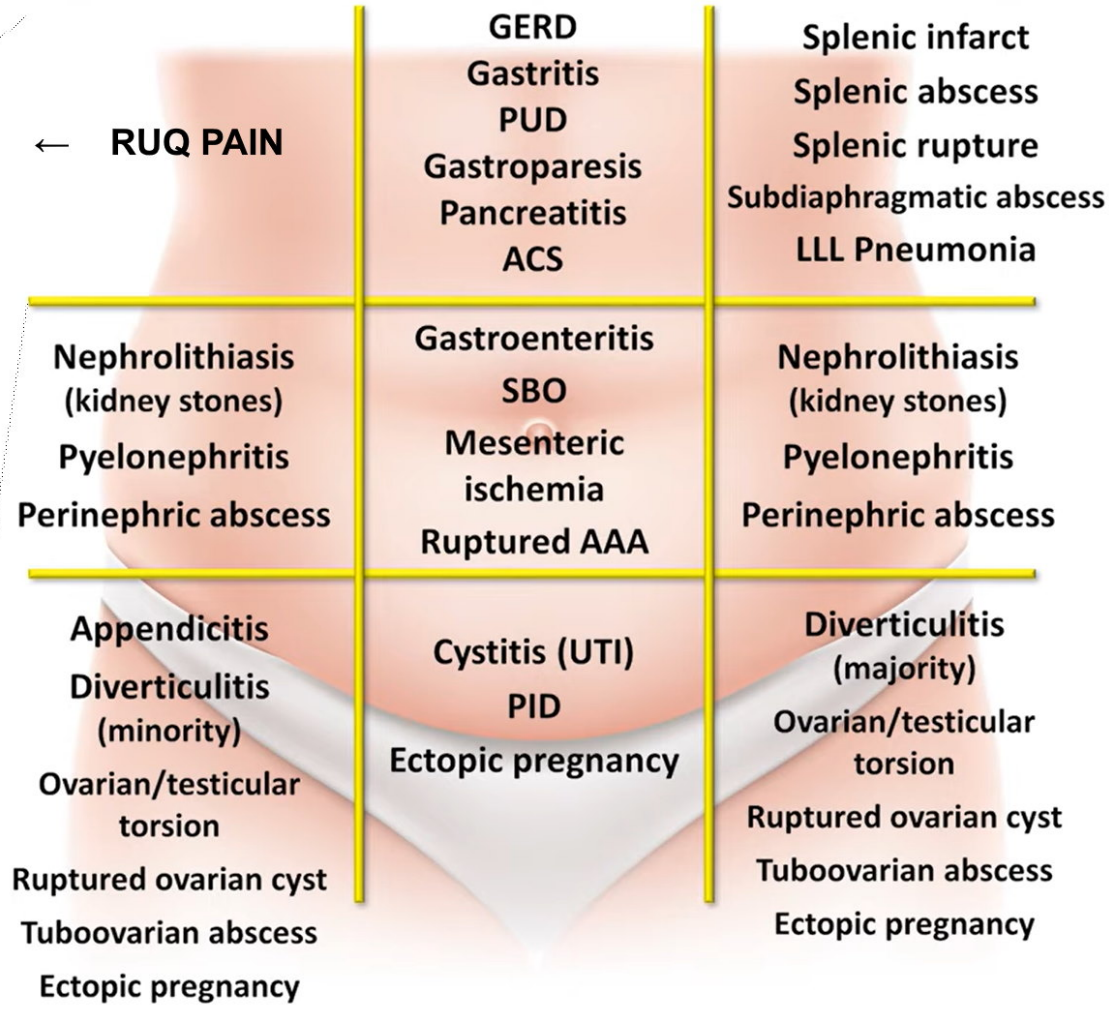


Fig. 6.5 Characteristic radiation of pain from the gallbladder, diaphragm and ureters.

Abdominal Pain - Differential Diagnosis

Right Upper Quadrant Pain

Liver	Biliary	Other
<u>Infectious</u> ▪ Acute viral hepatitis ▪ Liver abscess <u>Non-infectious</u> ▪ Acute non-infectious hepatitis • Alcohol • Medications ▪ Hepatic congestion (e.g. heart failure) ▪ Budd-Chiari syndrome	<u>Infectious</u> ▪ Acute cholecystitis ▪ Acute cholangitis (a.k.a. "ascending cholangitis") <u>Non-infectious</u> ▪ Gallstones (a.k.a. "biliary colic") ▪ Choledocholithiasis	RLL Pneumonia Subdiaphragmatic abscess



Onset

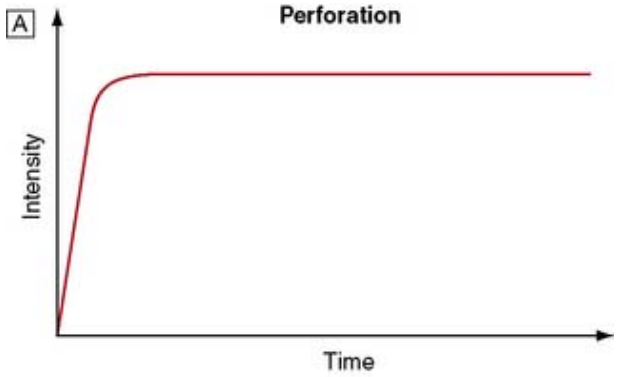
- The sudden onset of severe abdominal pain, rapidly progressing to become generalized and constant, suggests a hollow viscus perforation, a ruptured abdominal aortic aneurysm or mesenteric infarction.

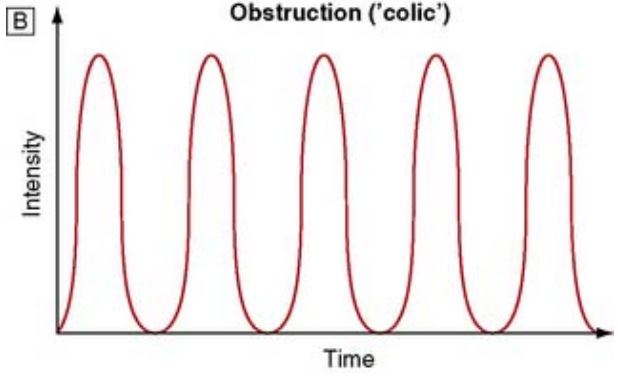
- Cecal or sigmoid volvulus (Torsion) occur with sudden abdominal pain associated with intestinal obstruction

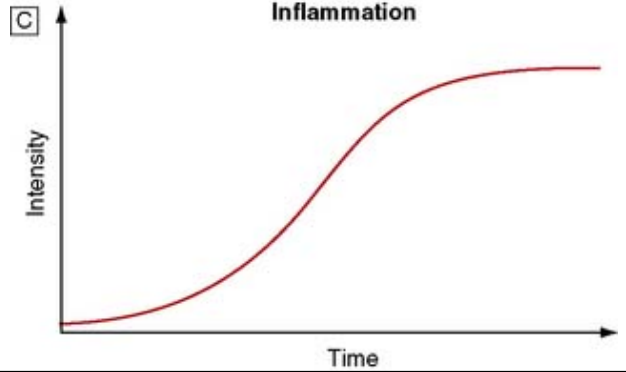
Character

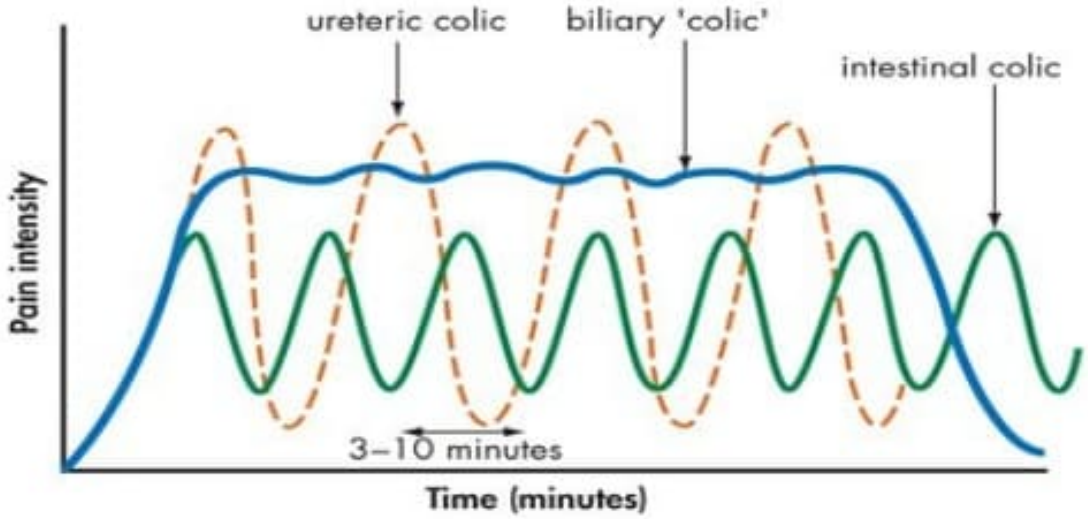
- Colicky pain lasts for a short time (seconds or minutes) eases off and then returns.
- It arises from hollow structures such as small and large bowel obstruction. or the uterus during labour.
- "Hyperperistalsis against an obstructed hollow viscus"
- Dull constant vague and poorly localized pain is suggestive of inflammation, e.g.. salpingitis, appendicitis or diverticulitis.

Character, cont.









Source: John Murtagh, Jill Rosenblatt: *John Murtagh's General Practice*, 6e: www.murtagh.mhmedical.com

Character, cont.

Renal
+

- Biliary colic is a misnomer, as the pain is rarely colicky, pain rapidly increases to a peak and persists over period of time before gradually resolving.
- Unlike elsewhere in the intestinal tract, the gallbladder does not have a muscularis mucosae, and the muscular fibres are not arranged in distinct layers. The interspersed muscle fibres lie in longitudinal, oblique and transverse directions, and are not arranged in separate layers.

Radiation

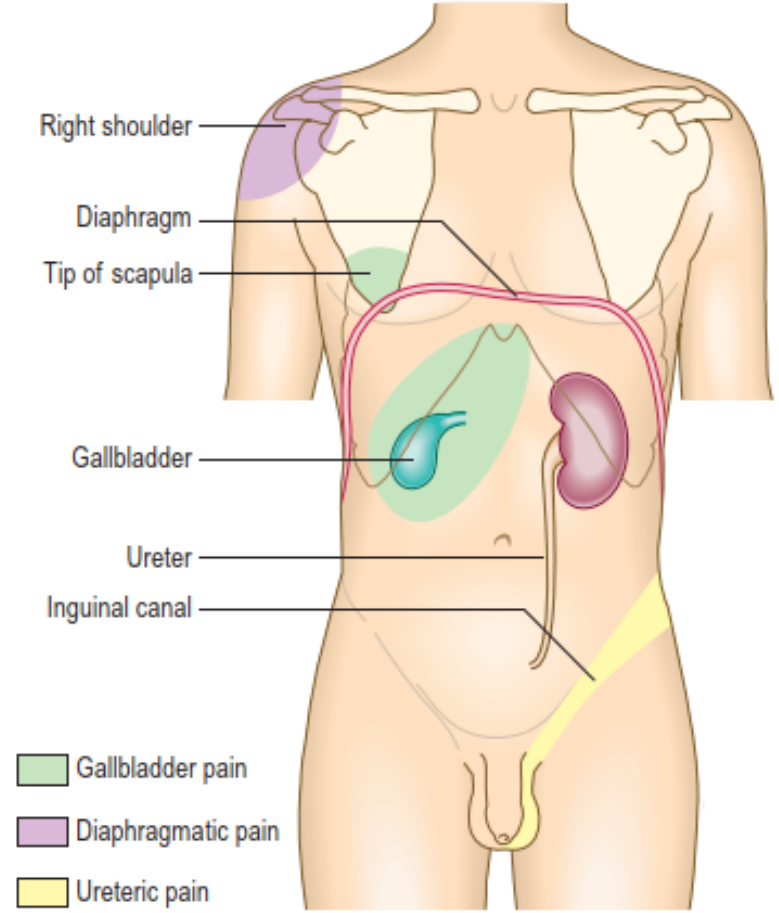


Fig. 6.5 Characteristic radiation of pain from the gallbladder, diaphragm and ureters.

Pain arising from foregut structures (stomach, pancreas, liver and biliary system) is localised above the umbilicus.

Central abdominal pain arises from midgut structures, such as the small bowel and appendix.

Lower abdominal pain arises from hind gut structures,such as the colon.

Inflammation may cause localised pain: for example, left iliac fossa pain due to diverticular disease of the sigmoid colon.

Pain radiating from the right hypochondrium to the shoulder or interscapular region may reect diaphragmatic irritation, as 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, suggests pancreatitis. Central abdominal pain that 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 abdominal aortic aneurysm.

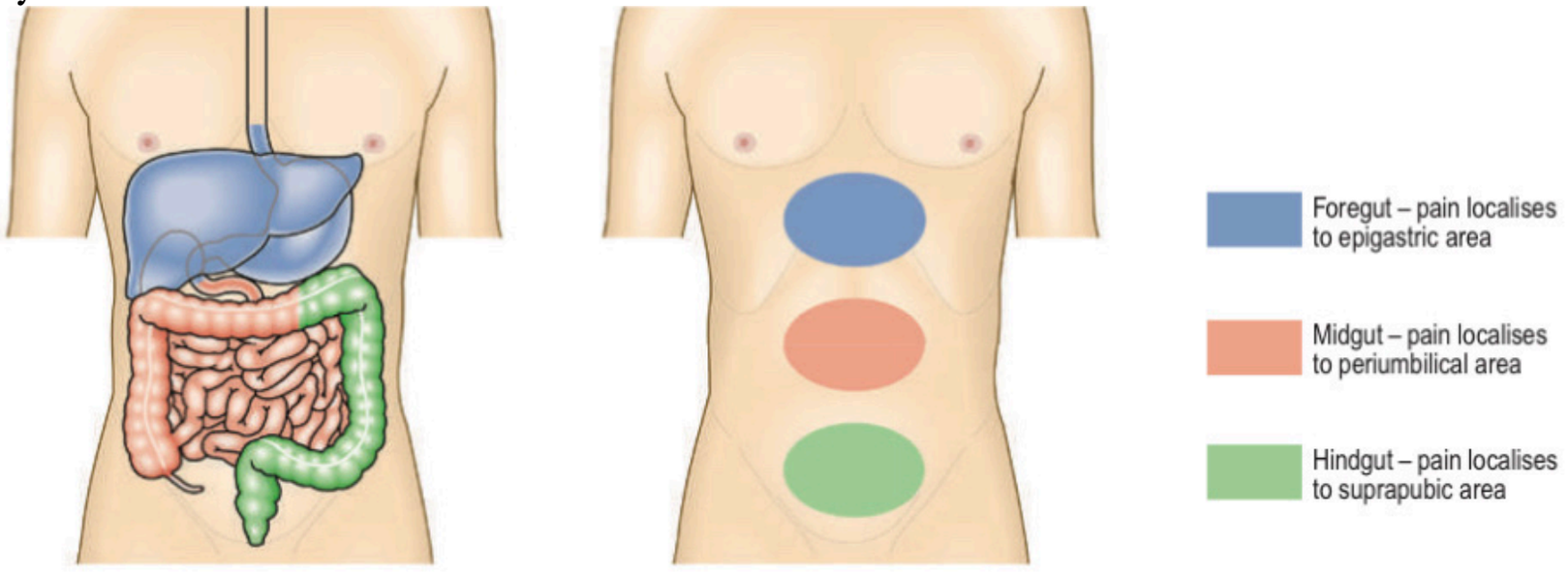


Fig. 6.4 Abdominal pain. Perception of visceral pain is localised to the epigastric, umbilical or suprapubic region, according to the embryological origin of the affected organ.

Associated symptoms

- Non-specific symptoms : Reflex anorexia, nausea and vomiting are common but may be absent even in advanced intra-abdominal disease.
- Altered bowel habits : IBS, CRC, diverticular disease
- Tachycardia , hypotension – sepsis or bleeding from a peptic ulcer, a ruptured aortic aneurysm or an ectopic pregnancy.
- Breathlessness and Palpitations – non alimentary causes

Timing

- Frequency and duration
- *Silent interval:* 1-2 hours after perforation; relief of obstruction
- Change of pattern: either wrong diagnosis or complications happened
- Abdominal pain persisting for hours or days suggests an inflammatory disorder

(appendicitis,cholecystitis,diverticulitis)

Timing, Cont.

- Acute appendicitis pain course: starts periumbilical (visceral, lumen obstruction) then migrates to the right iliac fossa in about 6-12 hours (somatic, inflammation)
- If it perforates, a possible silent interval (1-2 hours) if chemical peritonitis subsides, then bacterial peritonitis and generalized peritonitis may develop
- Occasionally, appendiceal mass develops; abscess or phlegmon.

Timing, Cont.

- Intestinal obstruction: Intestinal colic (visceral pain)
- If pain becomes more constant, systemic symptoms develop and the patient develops tenderness, that suggests intestinal ischemia and possible perforation

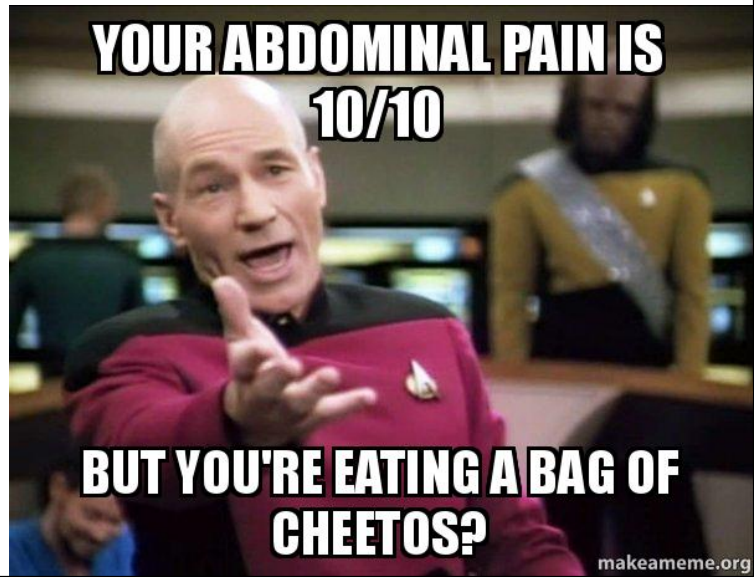
as in strangulated hernia, and is an indication for urgent surgical intervention.
Abdominal pain persisting for hours or days suggests an inammatory disorder, such as acute appendicitis, cholecystitis or diverticulitis.

Exacerbating and relieving factors

- Pain due to ***inflammation*** is exacerbated by movement or coughing.
- 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.

Severity

- Excruciating pain, poorly relieved by opioid analgesia, suggests an ischemic vascular event, e.g. bowel infarction or ruptured abdominal aortic aneurysm.
- Severe pain rapidly eased by potent analgesia is more typical of acute pancreatitis or peritonitis secondary to a ruptured viscus.



6.2 Diagnosing abdominal pain

	Disorder			
	Peptic ulcer	**Biliary pain**	**Acute pancreatitis**	**Renal colic**
Site	Epigastrium	Epigastrium/right hypochondrium	Epigastrium/left hypochondrium	Loin
Onset	Gradual	Rapidly increasing	Sudden	Rapidly increasing
Character	Gnawing	Constant	Constant	Colicky
Radiation	Into back	Below right scapula	Into back	Into genitalia and inner thigh
Associated symptoms	Non-specific	Non-specific	Non-specific	Non-specific
Timing				
Frequency/periodicity	Remission for weeks/months	Attacks can be enumerated	Attacks can be enumerated	Usually a discrete episode
Special times	Nocturnal and especially when hungry	Unpredictable	After heavy drinking	Following periods of dehydration
Duration	½–2 hours	< 6 hours	>24 hours	4–24 hours
Exacerbating factors	Stress, spicy foods, alcohol, non-steroidal anti-inflammatory drugs	Eating – unable to eat during bouts Fatty food	Alcohol Eating – unable to eat during bouts	–
Relieving factors	Food, antacids, vomiting	–	Sitting upright Leaning forward	
Severity	Mild to moderate	Severe	Severe	Severe

8.5 Diagnosing abdominal pain

	Disorder			
	Peptic ulcer	**Biliary colic**	**Acute pancreatitis**	**Renal colic**
Site	Epigastrium	Epigastrium/right hypochondrium	Epigastrium/left hypochondrium	Loin
Onset	Gradual	Rapidly increasing	Sudden	Rapidly increasing
Character	Gnawing	Constant	Constant	Constant
Radiation	Into back	Below right scapula	Into back	Into genitalia and inner thigh
Timing				
Frequency/periodicity	Remission for weeks/ months	Able to enumerate attacks	Able to enumerate attacks	Usually a discrete episode
Special times	Nocturnal and especially when hungry	Unpredictable	After heavy drinking	Following periods of dehydration
Duration	½–2 hours	4–24 hours	>24 hours	4–24 hours
Exacerbating factors	Stress, spicy foods, alcohol, non-steroidal anti-inflammatory drugs (NSAIDs)	Unable to eat during bouts	Alcohol Unable to eat during bouts	
Relieving factors	Food, antacids, vomiting		Eased by sitting upright	
Severity	Mild to moderate	Severe	Severe	Severe

Non Gastrointestinal causes

6.3 Non-alimentary causes of abdominal pain

Disorder	Clinical features
Myocardial infarction	Epigastric pain without tenderness *Angor animi* (feeling of impending death) Hypotension Cardiac arrhythmias
Dissecting aortic aneurysm	Tearing interscapular pain *Angor animi* Hypotension Asymmetry of femoral pulses
Acute vertebral collapse	Lateralised pain restricting movement Tenderness overlying involved vertebra
Cord compression	Pain on percussion of thoracic spine Hyperaesthesia at affected dermatome with sensory loss below Spinal cord signs
Pleurisy	Lateralised pain on coughing Chest signs, e.g. pleural rub
Herpes zoster	Hyperaesthesia in dermatomal distribution Vesicular eruption
Diabetic ketoacidosis	Cramp-like pain Vomiting Air hunger Tachycardia Ketotic breath
Salpingitis or tubal pregnancy	Suprapubic and iliac fossa pain, localised tenderness Nausea, vomiting Fever
Torsion of testis/ovary	Lower abdominal pain Nausea, vomiting Localised tenderness

Acute abdomen

The acute abdomen

The majority of general surgical emergencies are patients with sudden severe abdominal pain (an ‘acute abdomen’). Patients may be so occupied by recent and severe symptoms that they forget important details of the history unless asked directly. Seek additional information from family or friends if severe pain, shock or altered consciousness makes it difficult to obtain a history from the patient. Note any relevant past history, such as acute perforation in a patient with known diverticular disease. Causes range from self-limiting to severe life-threatening diseases (Box 6.4). Evaluate patients rapidly, and then resuscitate critically ill patients immediately before undertaking further assessment and surgical intervention. Parenteral opioid analgesia to alleviate severe abdominal pain will help, not hinder, clinical assessment. In patients with undiagnosed acute abdominal pain, reassess their clinical state regularly, undertake urgent investigations and consider surgical intervention before administering repeat analgesia.

6

Nausea And Vomiting

the sensation of feeling sick. Vomiting is the expulsion Abdominal distension of gastric contents via the mouth. .

- Relation to meals and timing during the day
- Amount
- Associated symptoms: Dyspepsia, abdominal pain. Does vomiting relieve them? Weight loss.
- The presence of hypotension and tachycardia is an indication of severe dehydration
- Bile-stained, blood-stained or faeculant?
- Medications that may cause vomiting:

Nausea And Vomiting, cont.

Bile Colour

Nausea And Vomiting, cont.

- Both could be associated with pallor, sweating and hyperventilation.
- Dyspepsia: causes nausea without vomiting
- Peptic ulcers?
- Gastric outlet obstruction: causes projectile vomiting of large volumes of gastric content without significant pain that is not bile-stained (green).
- Obstruction distal to the pylorus (intestinal obstruction): bile-stained vomitus or faeculent vomitus.

Vomiting is common in gastroenteritis, cholecystitis, pancreatitis and hepatitis.
It is typically preceded by nausea but in raised intracranial pressure may occur without warning. Severe pain may precipitate vomiting, as in renal or biliary colic or myocardial infarction.
Anorexia nervosa and bulimia are eating disorders characterised by undisclosed, self-induced vomiting. In bulimia, weight is maintained or increased, unlike in anorexia nervosa, where profound weight loss is common.

- In intestinal obstruction; the more distal the level of intestinal obstruction, the more marked the accompanying abdominal distension and colic.
- In peritonitis, the vomitus is usually small in volume but persistent.

Nausea and vomiting, particularly with abdominal pain or discomfort, suggest upper gastrointestinal disorders.

Peptic ulcers seldom cause painless vomiting unless they are complicated by pyloric stenosis, which causes projectile vomiting of large volumes of gastric content that is not bile-stained.

Faeculent vomiting of small bowel contents (not faeces) is a late feature of distal small bowel or colonic obstruction.

Non-Gastrointestinal Causes

- Drugs: alcohol, opioids, theophyllines, digoxin, cytotoxic agents or antidepressants
- Pregnancy
- DKA
- Renal or Liver failure
- Hypercalcemia
- Addsion's disease
- Raised intracranial pressure (no preceding nausea, early morning)
- Vestibular disorder
- Self-induced: eating disorders

Wind and flatulence

- Includes bleching, excessive or offensive flatus, borborygmi, abdominal distension
- *Belching* is due to air swallowing (aerophagy) and has no medical significance.
- It may indicate anxiety, but sometimes occurs in an attempt to relieve abdominal pain or discomfort, and accompanies GERD.

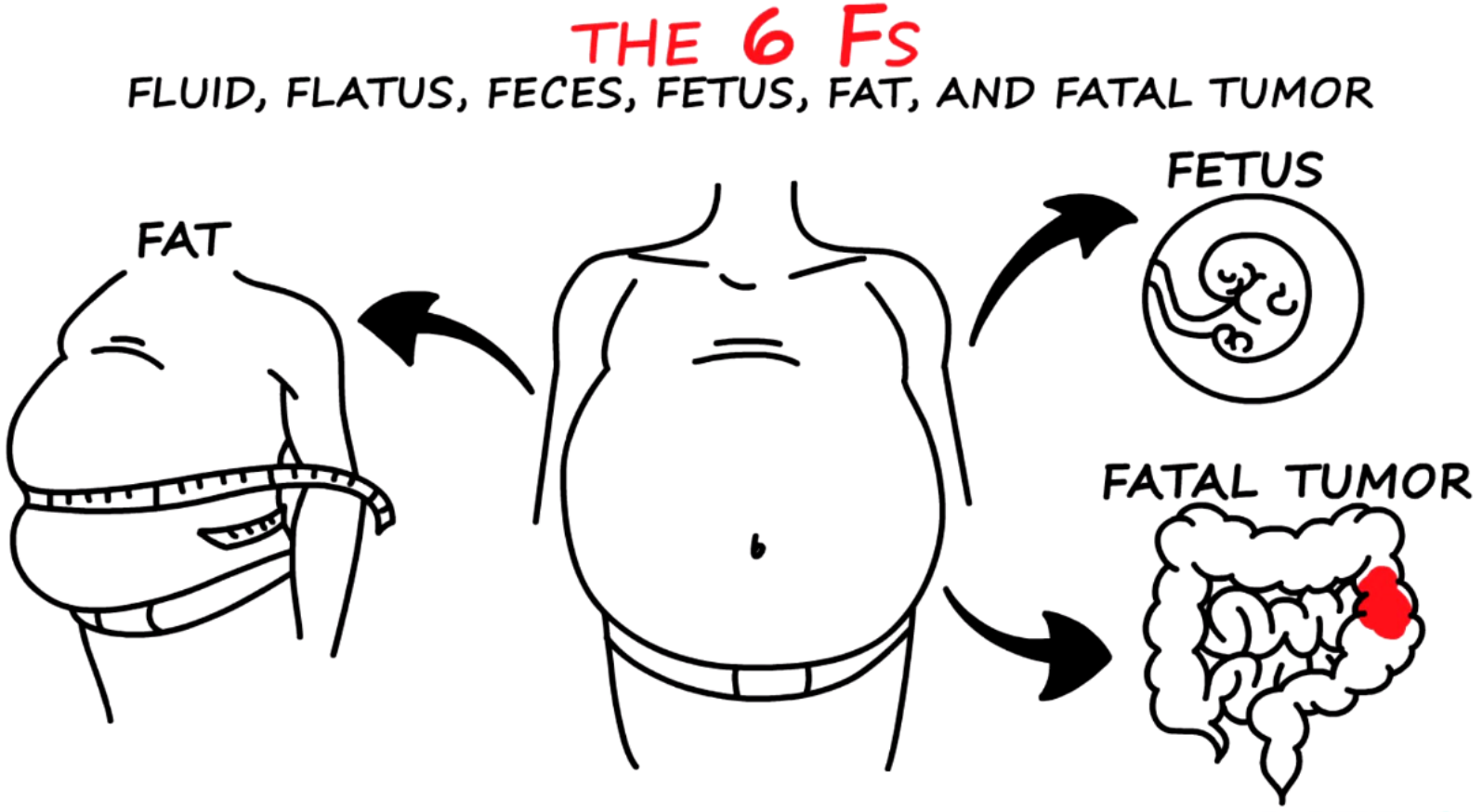
Wind and flatulence, cont.

- Flatus: Mixed gases from swallowed air and bacterial fermentation of carbs in colon
- Normally 200–2000 ml of flatus is passed each day.
- Excessive flatus occurs in lactase deficiency and intestinal malabsorption.

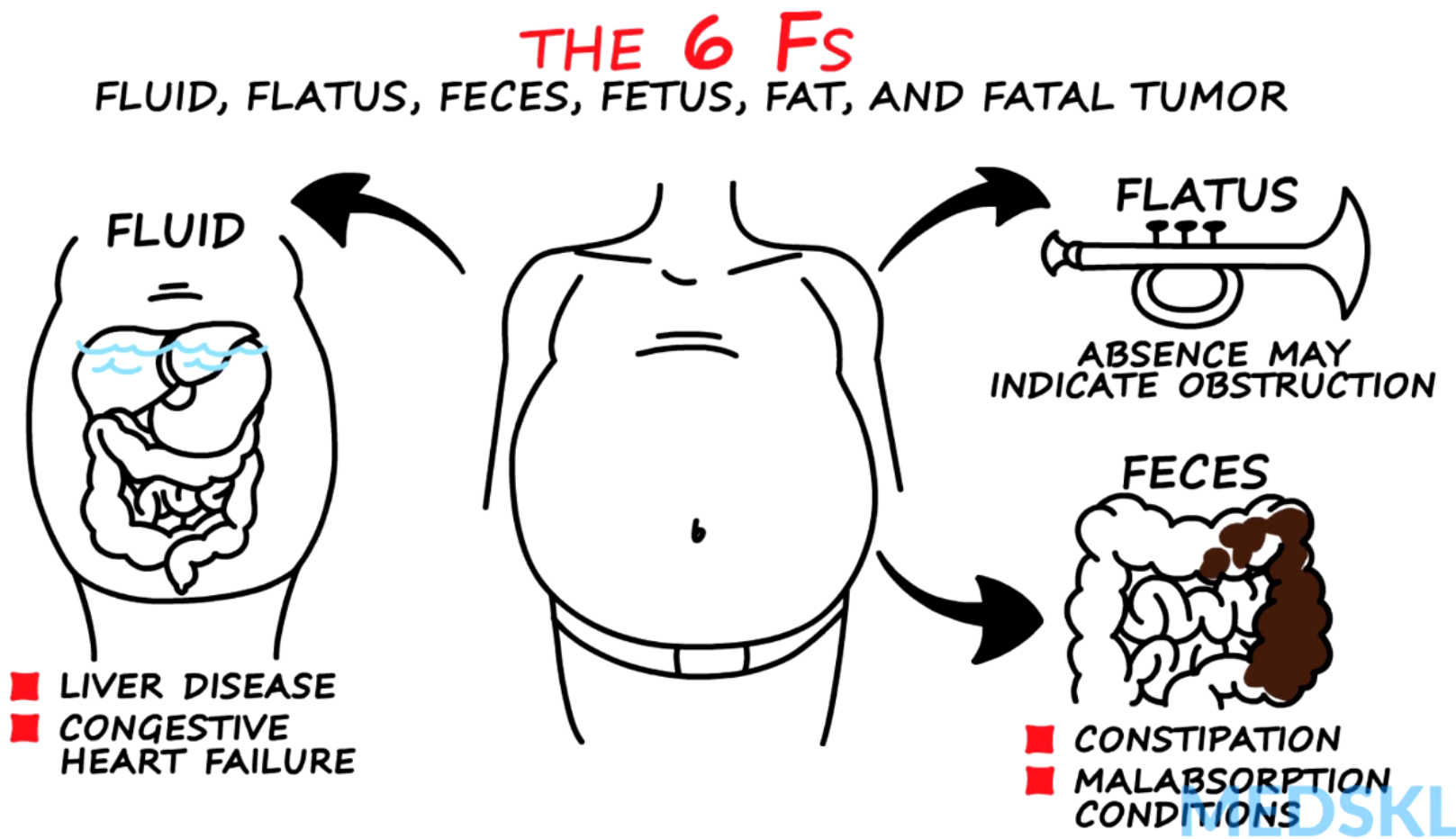
Borborygmi

- *Audible bowel sounds* from movement of fluid and gas along the bowel.
- Loud borborygmi, particularly if associated with colicky discomfort, suggest small-bowel obstruction or dysmotility.

Abdominal distention



Abdominal distention



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 develops during the day and resolves overnight

SAAG: Serum-Ascites Albumin Gradient

- SAAG = serum albumin – ascites albumin.
- Obtained with diagnostic paracentesis and ascitic fluid analysis.
- A high gradient (SAAG >1.1 g/dL) indicates portal hypertension and suggests a nonperitoneal cause of ascites., transudate
- ↑ pressure in portal vein → ↑ hydrostatic pressure in the hepatic vessels → pushing of fluid out from the intravascular space to the peritoneal cavity

SAAG: Serum-Ascites Albumin Gradient

- A low gradient (SAAG < 1.1 g/dL) indicates nonportal hypertension and suggests a peritoneal cause of ascites.
- ↓ intravascular osmotic gradient → secondary influx of water from the intravascular space to the peritoneal cavity, exudate (exudative ascitic fluid is high in protein, like eggs.)

SAAG: Serum-Ascites Albumin Gradient

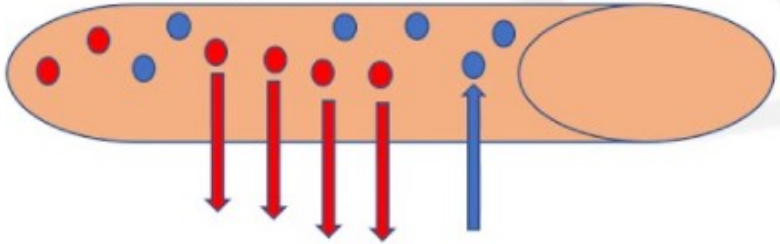
SAAG

HIGH SAAG (> 1·1 g/dl)

Increased portal pressure

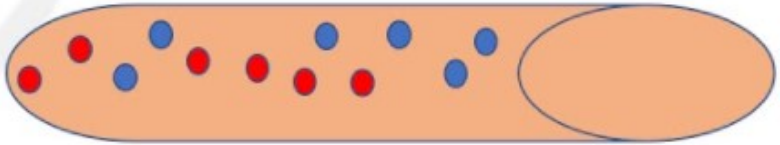
(either liver related or non-liver related)

Increased pressure forces fluid/water out from the vessels into the peritoneal cavity but leaves albumin within the blood vessels leading to HIGH SAAG



LOW SAAG (< 1·1 g/dl)

Portal pressure remains normal



Serum-ascites albumin gradient (SAAG)

	SAAG (g/dL)	
	≥ 1.1	< 1.1
Total protein (g/dL)		
< 2.5	Cirrhosis	Nephrotic syndrome
	Acute liver failure	
≥ 2.5	CHF	Peritoneal carcinomatosis
	Constrictive pericarditis	TB peritonitis
	Budd-Chiari syndrome	Pancreatic ascites
	Veno-occlusive disease	Chylous ascites

Altered Bowel Habit

Normal frequency ranges from three bowel movements daily to once every 3 days.

- Diarrhea: bowel movements more than 3 times daily or frequent passage of loose stool
- Clarify : frequency vs. consistency
- Change from their usual habits?
- Steatorrhea, related to fat malabsoption
  - Excretion of more than 7 g of fat per day is diagnostic of malabsorption
  - Stool is greasy, pale, bulky, floating, difficult to flush

Diarrhea, Cont.

- Onset : Acute, Chronic, intermittent
- Stool: frequency, volume, color, consistency (watery, unformed, semisolid)
- Content (red blood, mucus, pus)
- Associated features:
  - urgency, fecal incontinence, tenesmus, abdominal pain, vomiting, sleep disturbance.
- Recent travel
- Medications (antibiotics, laxatives, PPI)

Diarrhea, Cont.

- High-volume diarrhea (>1 liter per day) occurs when stool water content is increased
- Low-volume diarrhea is associated with the irritable bowel syndrome.
- Overflow diarrhea: Fecal impaction secretions and particulate/fluid stool matter seeps around the impacted fecal material.

Diarrhea, Cont.

- Secretory: due to intestinal inflammation, e.g. infection, or inflammatory bowel disease.

- Osmotic: due to malabsorption, adverse drug effects or motility disorders, related to food intake.

If the patient fasts, osmotic diarrhoea stops but secretory diarrhoea persists.

Causes Of high volume diarrhea

- Infective gastroenteritis – most common , norovirus/ salmonella/ c.diff , if > 4 weeks → chronic (giardiasis , amebiasis) cryptosporidiosis
- IBD, colonic ischemia, infective gastroenteritis → bloody
- Colon cancer → especially right sided
- Thyrotoxicosis → secretory diarrhoea or steatorrhoea and weight loss.
- Celiac disease, chronic pancreatitis, cystic fibrosis → Pancreatic insufficiency steatorrhea

Change in the bowel habit towards diarrhoea can be a manifestation of colon cancer, in particular cancer of the right side of the colon and in patients over 50 years

Causes Of Low Volume Diarrhea

- Irritable bowel syndrome → pain , dyspepsia , bloating
- Rome IV criteria is used for diagnosis of IBS

non-alimentary symptoms

presence of abdominal pain and its relationship to defecation, and the frequency and consistency of stools

Constipation

- Infrequent passage of hard stool
- Onset: lifelong, recent
- Stool frequency: How frequent, time spent straining
- Shape of stool → Bristol classification
- Associated symptoms: abdominal pain, anal pain, rectal bleeding
- Drugs

Terminology

- Obstipation: Absolute constipation with no gas or bowel movements, suggesting complete intestinal obstruction and is usually associated with pain, vomiting and distension.
- Tenesmus: feeling of incomplete evacuation, suggests rectal disease such as inflammation or cancer (the sensation of needing to defecate although the rectum is empty)
- Anismus : difficulty to empty the rectum despite straining due to paradoxical contraction of puborectalis muscle

Faecal impaction can occasionally present as overflow diarrhoea.

Causes

- Lack of dietary fibre
- Impaired motility
- Mechanical obstruction
- Colorectal cancer
- Impaired rectal sensation or anorectal dysfunction
- IBS
- Drugs (iron, PPI, opioids)
- Immobility (Parkinson's disease, stroke)
- Metabolic\endocrine (hypercalcemia, hypothyroidism)

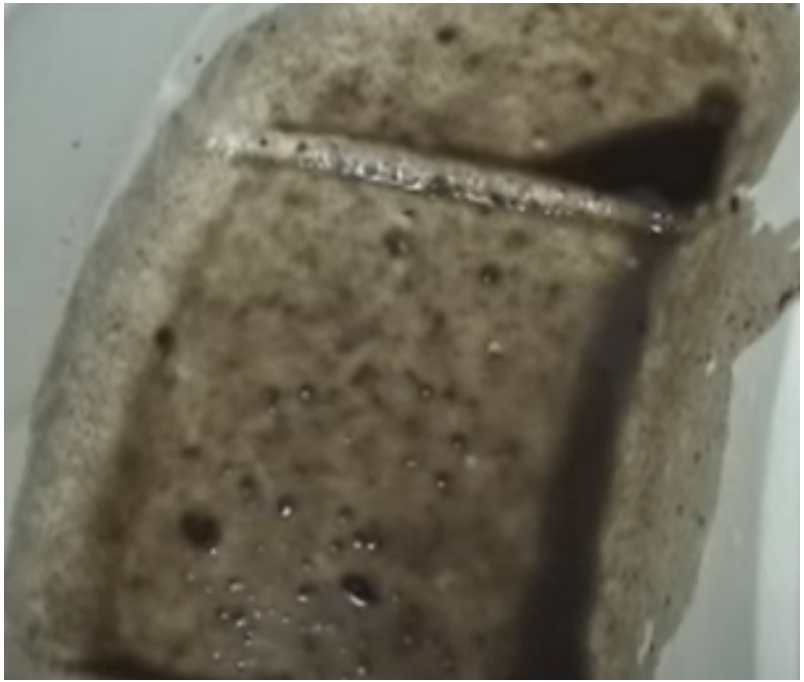
10

Bleeding

- Haematemesis: vomiting blood
- Ask about:
  - Color: fresh and red, or dark brown in colour and resembles coffee grounds
    - Coffee ground emesis usually occurs with slower bleeding and coagulation of blood after exposure to gastric acid.
  - Amount
  - Onset , was it preceded by intense retching?
  - Previous history: dypepsia, peptic ulcer, GI bleeding, liver disease
  - Drugs and Alcohol intake: NSAIDs, glucocorticosteroids

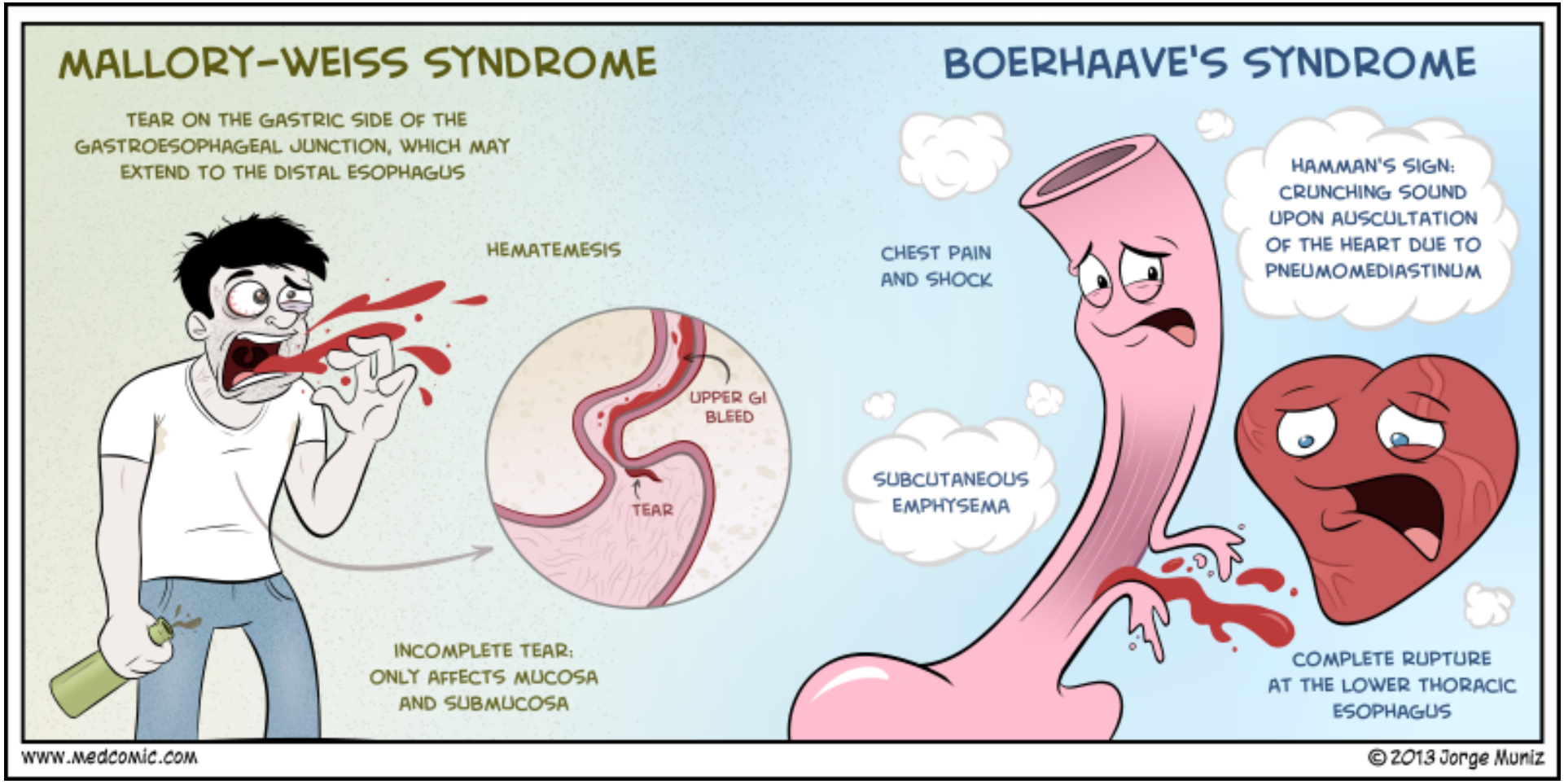
Haematemesis, cont.

If the source of bleeding is above the gastro-oesophageal sphincter, as with oesophageal varices, fresh blood may well up in the mouth, as well as being actively vomited. With a lower oesophageal mucosal tear due to the trauma of forceful retching (Mallory–Weiss syndrome), fresh blood appears only after the patient has vomited forcefully several times.



Coffee Ground Vomitus

Haematemesis



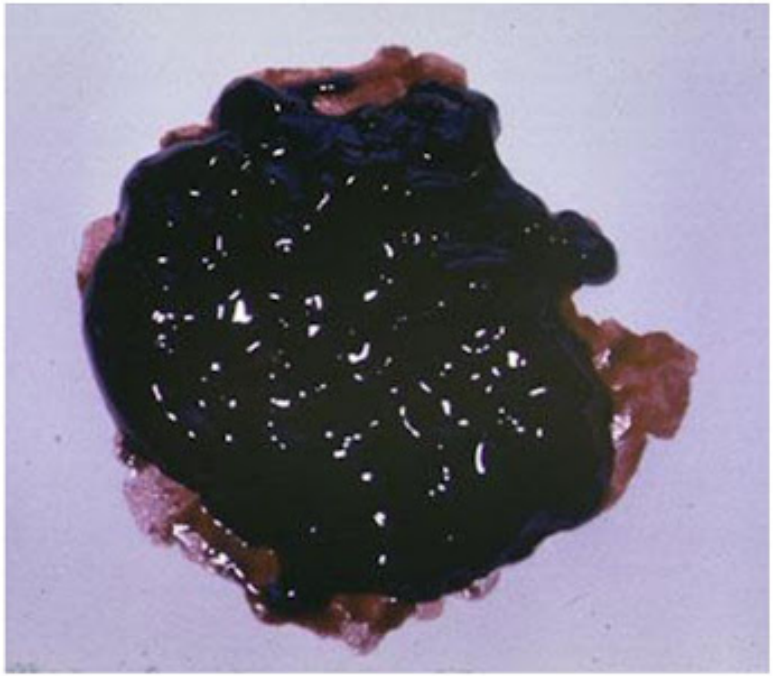
Bleeding, Cont.

- Melaena the passage of tarry, shiny black stools with a characteristic odor and results from upper gastrointestinal bleeding.

- Distinguish this from the matt black stools associated with oral iron or bismuth therapy.

Peptic ulceration (gastric or duodenal) is the most common cause of upper gastrointestinal bleeding and can manifest with melaena, haematemesis or both. Excessive alcohol ingestion may cause haematemesis from erosive gastritis, Mallory–Weiss tear or bleeding oesophagogastric varices in cirrhotic patients. Oesophageal or gastric cancer and gastric angioectasias (Dieulafoy lesion) are rare causes of upper gastrointestinal bleeding.A profound upper gastrointestinal bleed may lead to the passage of purple stool or, rarely, fresh blood.

Melaena



Melena
The specimen consists of a black tarry stool passed per anus.
Note the mahogany color at the edge of the specimen (filter paper).

Melaena: > 50 ml\day

Hemoccult: >20 ml\day

Bleeding, Cont.

- Fresh rectal bleeding (heamatocazia): indicates a disorder in the anal canal, rectum or colon.
- Blood may be mixed with stool, coat the surface of otherwise normal stool, or be seen on the toilet paper or in the pan.
- During severe upper gastrointestinal bleeding, blood may pass through the intestine unaltered, causing fresh rectal bleeding.

Causes of rectal bleeding

- Haemorrhoids — painless, bright red bleeding at the end of defecation
- Anal fissure — blood on the toilet paper or in the pan
- Colorectal polyps
- Colorectal cancer
- Inflammatory bowel disease
- Ischaemic colitis
- Complicated diverticular disease
- Vascular malformation

Jaundice

- Jaundice is a yellowish discoloration of the skin, sclerae and mucous membranes due to hyperbilirubinaemia.
- Most clinicians will recognize jaundice when bilirubin levels exceed 3 mg\dl 50 µmol /L

Jaundice

- Ask about:
  - Associated symptoms: abdominal pain, fever, weight loss, itching
  - Colour of stools(normal or pale) and urine (normal or dark)
  - Alcohol intake
  - Travel history and immunisations
  - Use of illicit or intravenous drugs
  - Sexual history
  - Previous blood transfusions
  - Recently prescribed drugs.

Jaundice related symptoms

- Appetite and weight change
- Abdominal pain, altered bowel habit
- Gastrointestinal bleeding
- Pruritus, dark urine, rigors
- Drug and alcohol history
- Past medical history (pancreatitis, biliary surgery)
- Previous jaundice or hepatitis
- Blood transfusions (hepatitis B or C)
- Family history, e.g. congenital spherocytosis, haemochromatosis
- Sexual and contact history (hepatitis B or C)
- Travel history and immunisations (hepatitis A)
- Skin tattooing (hepatitis B or C)

Jaundice, Cont.

- Stercobilin is the substance that gives the stool its normal color. Obstructive jaundice causes pale stool.
- Urobilin is the substance that gives the urine its yellow color.
- Urine bilirubin is normally absent.
- Conjugated hyperbilirubinemia causes the urine to become dark.

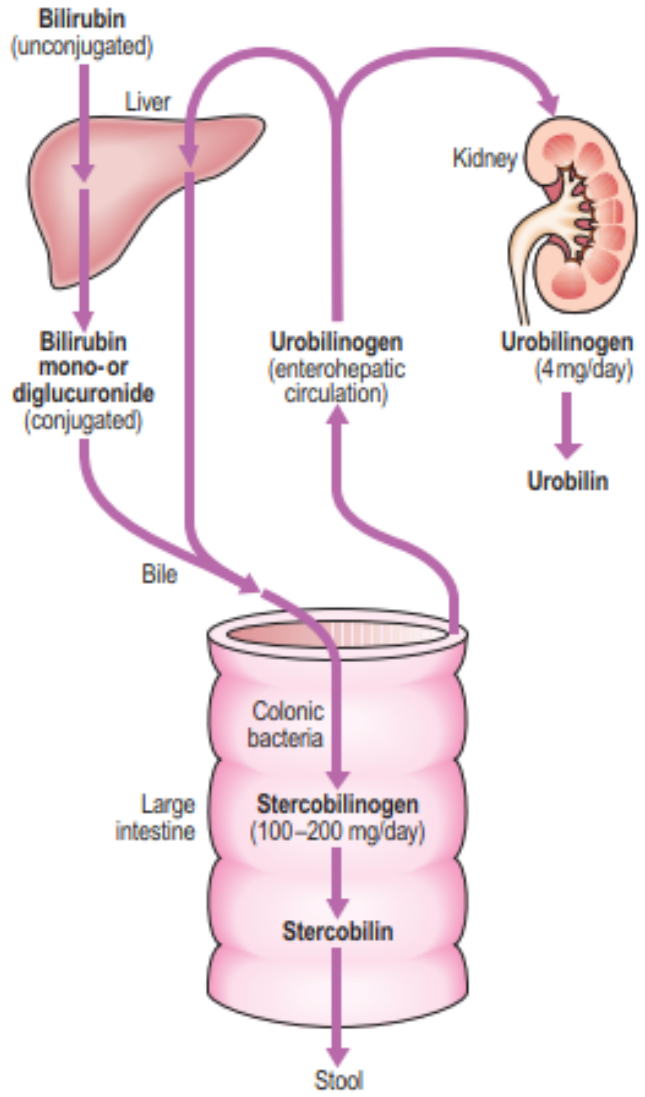
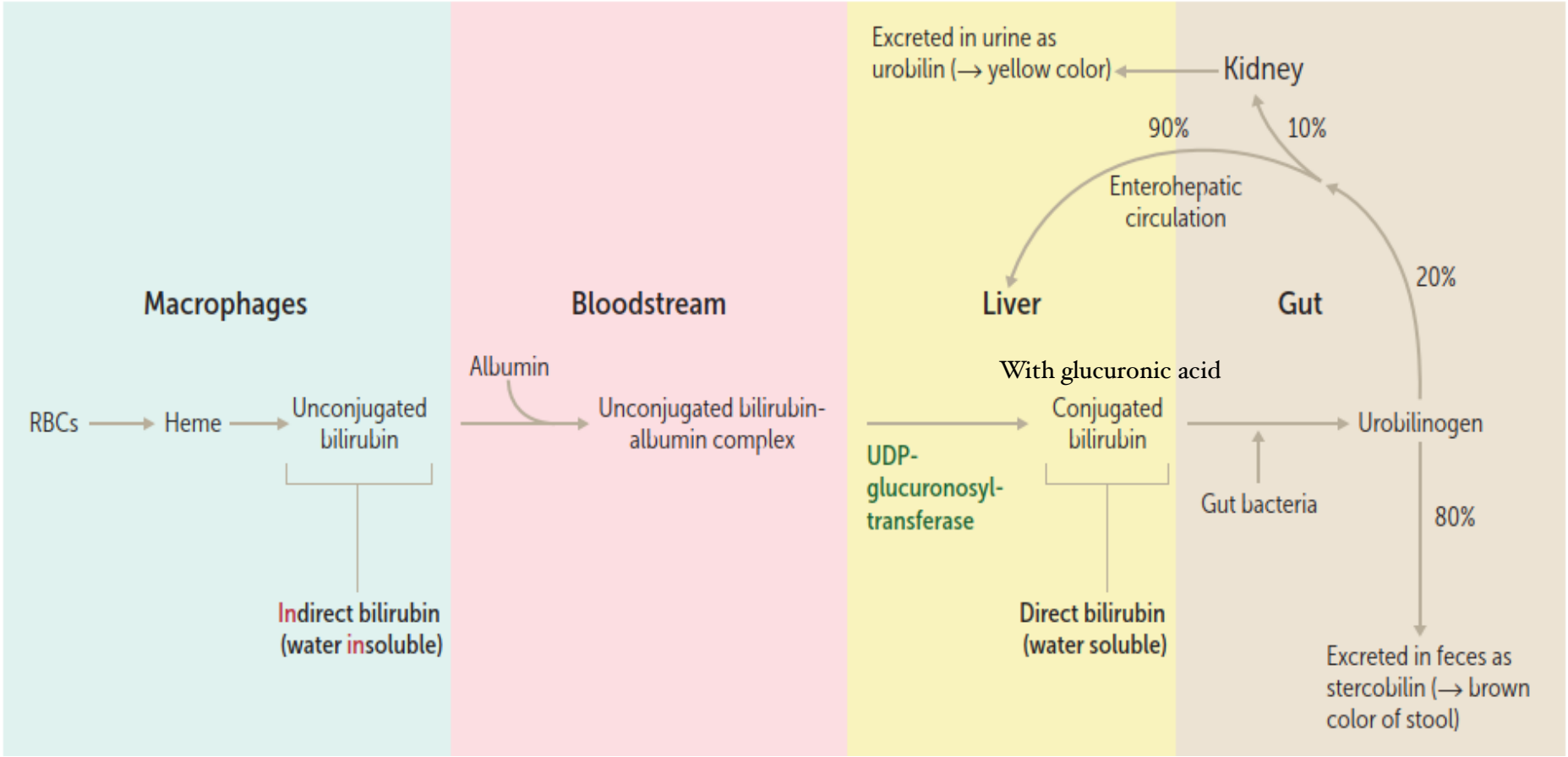


Fig. 8.9 Pathway of bilirubin excretion.

Jaundice, Cont.



Jaundice

Unconjugated bilirubin is insoluble and binds to plasma albumin; it is therefore not filtered by the renal glomeruli. In jaundice from unconjugated hyperbilirubinaemia, the urine is a normal colour (acholuric jaundice; Box 6.7).

Bilirubin is conjugated to form bilirubin diglucuronide in the liver and excreted in bile, producing its characteristic green colour. In conjugated hyperbilirubinaemia, the urine is dark brown due to the presence of bilirubin diglucuronide. In the colon, conjugated bilirubin is metabolised by bacteria to stercobilinogen and stercobilin, which contribute to the brown colour of stool. Stercobilinogen is absorbed from the bowel and excreted in the urine as urobilinogen, a colourless, water-soluble compound.

Prehepatic jaundice

In haemolytic disorders, anaemic pallor combined with jaundice may produce a pale lemon complexion. The stools and urine are normal in colour. Gilbert's syndrome is common and causes unconjugated hyperbilirubinaemia. Serum liver enzyme concentrations are normal and jaundice is mild (plasma bilirubin <100 μmol/L (5.85 mg/dL)) but increases during prolonged fasting or intercurrent febrile illness.

Hepatic jaundice

Hepatocellular disease causes hyperbilirubinaemia that is both unconjugated and conjugated. Conjugated bilirubin leads to dark brown urine. The stools are normal in colour.

Posthepatic/cholestatic jaundice

In biliary obstruction, conjugated bilirubin in the bile does not reach the intestine, so the stools are pale. Obstructive jaundice may be accompanied by pruritus (generalised itch) due to skin deposition of bile salts. Obstructive jaundice with abdominal pain is usually due to gallstones; if fever or rigors also occur (Charcot's triad), ascending cholangitis is likely. Painless obstructive jaundice suggests malignant biliary obstruction, as in cholangiocarcinoma or cancer of the head of the pancreas. Obstructive jaundice can be due to intrahepatic as well as extrahepatic cholestasis, as in primary biliary cirrhosis, certain hepatotoxic drug reactions (Box 6.8) and profound hepatocellular injury.

Quick Review

- Heme->biliverdin->bilirubin->to liver->conjugation to bile (aka **conjugated bilirubin**)->secreted into duodenum->broken down in large intestine by bacteria->**urobilinogen**->stercobilinogen OR resorbed into bloodstream and oxidized to **urobilin** which gives urine its color.

  In obstructive jaundice, you don't have secretion of conjugated bilirubin (bile) into the GI tract, so the bacteria in the large intestine does not have any bilirubin to breakdown into sterobilin which is why you get pale stool!
  So why do you still get dark urine? Note: you also don't get any urobilin either. BUT the buildup of conjugated bilirubin leaks into the urine. Bilirubin is also pigmented! bili-rubin (rubin for red, which is why you get dark urine).

Jaundice, Cont.

6.7 Urine and stool analysis in jaundice

	Urine			Stools
	Colour	Bilirubin	Urobilinogen	Colour
Unconjugated	Normal	–	++++	Normal
Hepatocellular	Dark	++	++	Normal
Obstructive	Dark	++++	–	Pale

Jaundice, Cont.

6.6 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

Jaundice, Cont.

- Prehepatic jaundice: extravascular hemolysis, decreased bilirubin uptake (Gilbert disease)
- Hepatic jaundice: Hepatocellular disease
- Posthepatocellular/ cholestatic jaundice: intrahepatic vs extrahepatic.
  - Extra: Gallstones (abdominal pain), cholangiocarcinoma (painless)
  - Intra: PBC, PSC, alcohol

Direct VS. Indirect hyperbilirubenemia

Cojugated bilirubin/ total bilirubin

- Indirect : <20 % of conjugated(D) bilirubin
- Mixed : 20-50% of conjugated(D) bilirubin
- Direct : >50% of conjugated(D) bilirubin

Groin swellings and lumps

- Hernias
- Lymph node
- Skin and subcutaneous Lumps
- Saphena varix
- Hydroceele
- Undescended testis
- Femoral aneurysm
- Psoas abscess

Ask about:

- associated pain
- precipitating/exacerbating factors, such as straining due to chronic constipation, chronic cough, heavy manual labour and relationship with micturition
- timing: when the symptoms are worse.

Hernias are common causes of groin lumps and frequently present with dull, dragging discomfort (rather than acute pain), which is often exacerbated by straining and after long periods of standing or activity. Patients can often manually reduce the hernia by applying gentle pressure over the swelling or by lying flat. Other causes of groin swellings include lymph nodes, skin and subcutaneous lumps and, less commonly, saphena varix (a varicosity of the long saphenous vein), hydrocoele of the spermatic cord, undescended testis, femoral aneurysm and psoas abscess.

Past Medical History

- History of a similar problem may suggest the diagnosis: for example, bleeding peptic ulcer or inflammatory bowel disease.

•Atrial fibrillation; acute mesentric ischemia (embolic)

- Primary biliary cirrhosis and autoimmune hepatitis are associated with thyroid disease.

- (NAFLD) is associated with diabetes and obesity.

Drug History

6.8 Examples of drug-induced gastrointestinal conditions

Symptom	Drug
Weight gain	Oral glucocorticoids
Dyspepsia and gastrointestinal bleeding	Aspirin Non-steroidal anti-inflammatory drugs
Nausea	Many drugs, including selective serotonin reuptake inhibitor antidepressants
Diarrhoea (pseudomembranous colitis)	Antibiotics Proton pump inhibitors
Constipation	Opioids
Jaundice: hepatitis	Paracetamol (overdose) Pyrazinamide Rifampicin Isoniazid
Jaundice: cholestatic	Flucloxacillin Chlorpromazine Co-amoxiclav
Liver fibrosis	Methotrexate

Family history

- Inflammatory bowel disease is more common in patients with a family history of either Crohn's disease or ulcerative colitis.

- Colorectal cancer in a first-degree relative increases the risk of colorectal cancer and polyps.

- Peptic ulcer disease is familial but this may be due to environmental factors, e.g. transmission of Helicobacter pylori infection.

Family History, Cont.

- Gilbert's syndrome is an autosomal dominant condition.
- Haemochromatosis and Wilson's disease are autosomal recessive disorders.
- Autoimmune diseases, particularly thyroid disease, are common in relatives of those with primary biliary cirrhosis and autoimmune hepatitis.
- A family history of diabetes is frequently seen in the context of NAFLD

Social history

- Dietary history and food intolerance (specific types)
- alcohol consumption
- Smoking increaes risk of malignancies, Crohn's disease and peptic ulcer, while it appears to decrease the severity of Ulcerative Colitis
- Stress, exacerbates irritable bowel syndrome and dyspepsia
- Foreign travel: liver diseases, diarrhea

Risk factors for liver disease

- IV drug abuse
- Tattoos
- Foreign travel
- Blood transfusion
- Homosexuality
- Multiple sexual partners
- History of hepatitis B or C