Gastritis and gastropathy  
Reflux oesophagitis  
Oesophageal varices  
Gastric varices  
Stomach Ca (rare)  
Iatrogenic (2% of patients undergoing PCI)  
(Haemorrhagic viral infections in developing world)  

Drugs e.g. NSAIDs (not corticosteroids) and alcohol make all of these more likely  
Anticoagulants do not cause acute GI haemorrhage but make bleeding that does occur more severe  

**Rockall risk assessment score**  
Initial scoring determines urgency of endoscopy taking into account…  
  - Age  
  - Co-morbidities  
    - Most significant: chronic kidney disease, *liver failure* or disseminated malignancy  
    - Also: cardiac disease or any other major disease  
  - Presence of classical features of shock  

Post-endoscopy gives indication of risk of rebleeding and death  
  - Endoscopic diagnosis  
    - Mallory-Weiss tear ranks similarly to no lesion  
    - Malignancy gives the greatest number of points  
  - Endoscopic stigmata of recent bleeding  
    - None or dark spots is reassuring  
    - Blood in the upper GI tract, adherent clot or spurting vessel is worrying  

NB: Blatchford score is similar but omits endoscopic findings  

**Emergency management if shocked**  
1. Protect airway  
2. High-flow oxygen by facemask  
3. 2x large-bore cannulae  
4. Bloods: FBC, U+E, LFT, glucose, clotting screen  
5. Cross-match 6 units  
6. IV fluids (crystalloid up to 1L)  
7. Blood if still shocked after 1L or Hb < 70g/L (group-specific or O negative)  
   - If suspected variceal bleeding, keep Hb < 100g/L  
8. Correct clotting abnormalities with vitamin K, FFP, prothrombin complex concentrate or platelets  
9. Monitoring  
   - Urinary catheter  
   - Central venous pressure line  
10. Stop NSAIDs, aspirin, clopidogrel and warfarin (discuss urgently with cardiology)  
11. Keep patient NBM  
12. Empirical IV PPI  
13. Antibiotics for patients with cirrhosis
• Bacterial overgrowth of anaerobes, which is often secondary to something else causing reduced motility, autonomic neuropathy, dilatation, strictures, diverticulae etc (commonest cause of watery diarrhoea and/or steatorrhoea in elderly)
• ? Tropical sprue

Non-infective
• IBD
• Radiation proctitis or colitis (acute or chronic)
• Beheet’s disease
• Diverticular disease
• Ischaemic colitis
• GI lymphoma
• Colon Ca
• Malabsorption e.g. coeliac
• Gut resection
• Bile acid malabsorption
  o Ileal resection esp. ileocaecal valve
  o Ileal disease inc. active or inactive Crohn’s
  o Primary bile acid diarrhoea
  o Postinfective gastroenteritis
  o Rapid small bowel transit
  o Post-cholecystectomy
• Drugs
  o Laxatives
  o Metformin
  o Anticancer drugs
  o Statins
  o PPIs
  o Antacids containing magnesium
  o Orlistat (reduce lipase production)
• Faecal impaction with overflow
• IBS

Endocrine
• Zollinger-Ellison syndrome
• Somatostatinoma
• Glucagonoma
• Carcinoid syndrome
• Thyrotoxicosis
• Medullary carcinoma of thyroid
• Diabetic autonomic neuropathy

Factitious
• Purgative abuse
• Dilutional diarrhoea

Specific presentations
Single episode: probably dietary indiscretion or anxiety

Large volume watery stools: always due to organic cause, watery diarrhoea suggests small bowel
Bloody diarrhoea: implies inflammatory colonic and/or rectal disease

Sudden onset diarrhoea with crampy abdominal pain, vomiting and fever lasting 2-5 days: most often infective, take stool cultures to diagnose, viral tends to last 24-48h, viral and bacterial do not last more than 2 weeks

Pale offensive stools that float associated with anorexia and weight loss: steatorrhoea (see specific section)

Investigations
Acute diarrhoea
• No investigation required unless it lasts > 1 week
• Stool culture and examination for ova, cysts and parasite and C. diff toxin assay
• Sigmoidoscopy with rectal biopsy

Chronic diarrhoea
• Always needs investigation
• May culture in the first instance
• If blood, must exclude colonic lesions
  o #1 Rectal biopsy
  o #2 Colonoscopy with biopsies
• Without blood
  o #1 Rectal biopsy
  o #2A If rectal biopsy is abnormal, progress to colonoscopy
  o #2B Normal rectal biopsy, imaging of small and large bowels +/- colonoscopy as well as blood tests
  o #2C If rectal biopsy shows melanosis coli, think purgatives

If steatorrhoea, exclude small bowel and pancreatic lesions (see specific section for flow chart)

Management
Acute diarrhoea
• Monitor fluid balance
  o May require oral fluid and electrolyte replacement
• Avoid using anti diarrhoeal drugs in infective diarrhoea though may be necessary for short-term relief
  o Codeine phosphate
  o Loperamide
• Antibiotics may be needed to clear some infective causes

Chronic diarrhoea
• Depends on cause

Clostridium difficile
Features
• Anything from 2 days to months post-antibiotics
• Usually elderly hospitalised patients
• Mild disease: mild diarrhoea
• Severe disease: profuse, watery, haemorrhagic colitis with abdominal pain
• Can cause inflamed and ulcerated colonic mucosa (colitis) and may be covered by a pseudomembrane

Complications
• Toxic megacolon

Diagnosis
• A or B toxin can be detected in stool by ELISA or PCR

Management
• Metronidazole (mild or moderate disease)
• Oral vancomycin (severe or relapsing disease)

Common causative antibiotics
• Broad-spectrum penicillins
• Broad-spectrum cephalosporins e.g. ceftriaxone
• Fluoroquinolones e.g. ciprofloxacin
• Clindamycin
• Macrolides e.g. clarithromycin, erythromycin

Rectal Bleeding
General
Massive bleeding from the lower GI tract is rare

Differentials
Haemorrhoids (most common – small, recurrent bleeds)
Anal fissure (common)
Diverticular disease
Ischaemic colitis
Carcinoma (colon, caecum – often occult)
Polyps (small, frequent bleeds)
Non-ischaemic colitis e.g. UC, Crohn’s or infective (usually alongside diarrhoea)
Solitary ulcer of rectum
Meckel’s diverticulum
Angiodysplasia (occult)
Healed acute proctitis (telangiectases)

Initial management if unwell patient
Most start and stop spontaneously
If bleeding continues and haemodynamically unstable, management is similar to upper GI bleeding
Surgery is rarely required

Features in the history that suggest colorectal cancer
Absence of anal symptoms (2 week wait referral)
Change in bowel habit looser
Anorexia, Malabsorption and Weight Loss

Differential diagnosis is mostly of small bowel disease

Coeliac
DM

Tropical sprue
Parasites e.g. Giardia (can look very like tropical sprue), Cryptosporidiosis (often in context of HIV)
Bacterial overgrowth
Whipple’s disease (many systemic symptoms)

Chronic radiation enteropathy

Ileal resection (more significant than jejunal as ileum is where bile salts and B12 are absorbed)
Ileal disease e.g. Crohn’s
Massive intestinal resection leaving <1m of small bowel (short-bowel syndrome)

Lymphoma that has infiltrated small bowel mucosa

Anorexia
May also be due to…
Systemic disease e.g. cancer (late)
Psychiatric

Steatorrhoea
= Malabsorption of fat
Pale, bulky, offensive, float, hard to flush and leave fatty film on the water

Specific causes of steatorrhoea…

- Lipase insufficiency
  - Pancreatic disease
  - Drugs e.g. orlistat
- Bile salt insufficiency either lack of production or not present in gut
  - Liver disease
  - Cholestatic jaundice
  - Bacterial overgrowth causes steatorrhoea due to deconjugation of bile salts, also B12 deficiency
  - Ileal resection or disease can cause problems with bile salt reabsorption
  - Drugs e.g. cholestyramine
- Malabsorption
• Low calcium, high ALP of osteomalacia

Faecal elastase to exclude pancreatic disease

OGD and biopsy
• Even if antibodies positive
• Gold standard
• Subtotal villous atrophy (coeliac disease is commonest cause)
• Following gluten challenge

Colonoscopy
• In some groups need to do this to exclude malignancy and symptoms may look similar

DEXA scan
• Osteoporosis can occur even on gluten free diet

Assessing for complications
• Small bowel follow through
• Capsule endoscopy
• MRI
• Laparoscopy with full thickness small bowel biopsy

Treatment
Gluten-free diet for life
• Clinical improvement within days or weeks
• Morphological improvement may take months
• Reduces risk of malignant complications
• ‘Non-responsive coeliac disease’ is often due to non-compliance or concomitant problem
• ‘Refractory coeliac disease’ is truly diet-resistant and has a poor prognosis

Replacement minerals and vitamins may be needed initially to replace body stores
Pneumococcal vaccine every 5 years (splenic atrophy)

Inflammatory Bowel Disease

General
Classification
• Crohn’s
• UC
• Colitis of undetermined type and etiology (CUTE) or indeterminate colitis
• Microscopic colitis (no macroscopic evidence of inflammation)
  o Lymphocytic
  o Collagenous

Risk factors
Shared
• Jewish, caucasian
For severe colonic disease in either Crohn’s or UC as lower risk of perforation
May also use technetium-labelled white cell scan
OGD
Crohn’s: if relevant symptoms
Video capsule enteroscopy for small bowel involvement (after normal radiology)

Small bowel radiology is needed for all patients with suspected Crohn’s (not much used in UC)
AXR: essential in severe attacks to exclude dilatation
USS may show thickened small bowel or distended loops as well as free-fluid
MRI enteroclysis (can show fistula and strictures as well as extent of inflammation)
(Small bowel barium follow-through detects gross anatomical defects e.g. strictures)
CT with oral contrast, can also show extraintestinal features e.g. abscesses

Perianal MRI or endoanal USS

If TB possible, microscopy and culture of any available tissue and may require therapeutic trial of anti-TB therapy

<table>
<thead>
<tr>
<th>Bowel changes</th>
<th>Crohn’s disease</th>
<th>Ulcerative colitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Macroscopic</strong></td>
<td>Bowel thickened and often narrowed</td>
<td></td>
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<tr>
<td>Cobblestoning</td>
<td>Aphtoid ulcers</td>
<td></td>
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<tr>
<td>Fistulae and abscesses</td>
<td>Strictures</td>
<td></td>
</tr>
<tr>
<td><strong>Inflammation</strong></td>
<td>Deep (transmural)</td>
<td></td>
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<tr>
<td>Patchy (skip lesions)</td>
<td>Lymphoid hyperplasia</td>
<td></td>
</tr>
<tr>
<td><strong>Granulomas</strong></td>
<td>++ Non-caseating epithelioid cell aggregates with Langhan’s giant cells</td>
<td></td>
</tr>
<tr>
<td><strong>Goblet cells</strong></td>
<td>Present</td>
<td></td>
</tr>
<tr>
<td><strong>Crypt abscesses</strong></td>
<td>+</td>
<td></td>
</tr>
<tr>
<td><strong>Fulminant disease</strong></td>
<td>Mucosa lost leaving only islands of oedematous mucosa</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Toxic dilatation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mucosa can return to normal with healing though leaves scarring</td>
<td></td>
</tr>
</tbody>
</table>

Management of Crohn’s
Medical
Induction of remission
Oral or IV glucocorticosteroids
Acute small bowel (mesenteric) ischaemia
- Often embolus from AF
- Sudden onset pain and vomiting
- Distention
- Tenderness
- Absent bowel sounds
- Very ill patient

Ischaemic colitis
- Often at the splenic flexure
- Thumbprinting on AXR

**Perforated viscus**
**Recognition**
- Peritonitis
- Shock

**Investigation**
- CXR: air under diaphragm
- AXR: Rigler’s sign (aka double wall sign), air on both sides of intestine wall
- Amylase – pancreatitis can look very similar

**Emergency management**
1. IV fluids (0.9% saline)
2. Crossmatch 2 units OR group and save
3. Blood cultures
4. Antibiotics (cefuroxime and metronidazole IV)
5. Analgesia
6. Imaging if appropriate (CXR, AXR, ECG)
7. Requires laparotomy

**Enterocutaneous fistula**
**Management**
- SNAPP system
- Sepsis (by far the most important thing – imaging, cultures, antibiotics etc)
- Nutrition (preferably enteral but may need TPN)
- (Anatomy)
- Protection of skin
- Planned surgery many months later

**Diverticular Disease**
**Definitions**
Diverticula = pouches of mucosa extruding through muscular wall through weakened areas near blood vessels, a common finding in the colon, most frequently in the sigmoid, incidence increases with age, related to low fibre diet

Diverticulosis = presence of diverticula
• Thalassaemia
• Storage disease e.g. Gaucher’s

Mild
• Other myeloproliferative disorders
• Haemolytic anaemia
• (Megaloblastic anaemia)
• Infection inc. EBV, hepatitis, endocarditis
• Vasculitis e.g. SLE, RA
• Infiltration e.g. amyloidosis, sarcoidosis

Groin Lumps
Differential diagnosis
Hernias: inguinal, femoral
Lymph nodes
Arterial: femoral artery aneurysm
Infection: psoas abscess (lateral to femoral artery, back pain, limp, swinging pyrexia)
Undescended testis (empty ipsilateral scrotum, different from retracted which can be manipulated back into scrotum)
Sebaceous cyst
Lipoma
Venous: saphena varix (cough impulse)
Neuroma of femoral nerve
Tumour

Anatomy
Boundaries of femoral canal
• Anterosuperior = inguinal ligament
• Posterior = pectineal ligament (anterior to superior pubic ramus)
• Medial = lacunar ligament
• Lateral = femoral vein

Boundaries of inguinal canal (MALT)
• Superior (roof) = 2 muscles
  o Internal oblique
  o Transverse abdominus
• Anterior = 2 aponeuroses
  o External oblique
  o Internal oblique
• Inferior (floor) = 2 ligaments
  o Inguinal
  o Lacunar
• Posterior = 2 Ts
  o Transversalis fascia
  o Conjoint Tendon

Inguinal canal = ASIS to pubic symphysis
• Rebound tenderness possible
• (If haemorrhagic, can get shock)

Investigation
• USS

Emergency management if haemodynamically unstable premenopausal patient or any postmenopausal patient
1. Fluid resuscitation
2. Broad spectrum antibiotics
3. Surgical exploration