Management:

| Steroids:                                                                 | • Most potent medical treatment.  
|                                                                           | • Oral prednisolone or IV hydrocortisone  
|                                                                           | • Long-term risks: adrenal suppression, osteopenia.  
| 5-Aminosalicylic compounds:                                               | • E.g. sulfasalazine, mesalazine  
|                                                                           | • Maintenance treatment of colonic Crohn’s  
| Azathioprine, 6-mercaptopurine or methotrexate:                           | • Effective maintenance treatments  
|                                                                           | • Used in those with frequent or severe relapses  
| Biologicals – antibodies to TNF-alpha (Infliximab):                       | • For steroid-resistant disease or perianal fistulae  
| Surgery:                                                                 | • Conservative – patchy and recurrent nature of Crohn’s means it’s not a great option  
|                                                                           | • Reserved for structural disease e.g. strictures, that is not responding to medical therapy  
|                                                                           | • Or for complications e.g. abscesses, fistulae  

Nutrition:

| • High metabolic demand from acute inflammation and the small intestinal dysfunction makes this challenging.  
| • Liquid diets very effective  
| • ‘Elemental diet’ where nitrogen source is in form of amino acids  
| • ‘Polymeric diet’ providing short peptides  

Recognition and management of acute colitis:

<table>
<thead>
<tr>
<th>Table 1 Truelove and Witts' severity index</th>
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</thead>
<tbody>
<tr>
<td>Mild</td>
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<tr>
<td>------</td>
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<tr>
<td>Bowel movements (no. per day)</td>
</tr>
<tr>
<td>Fewer than 4</td>
</tr>
<tr>
<td>Blood in stools</td>
</tr>
<tr>
<td>No more than small filaments of blood</td>
</tr>
<tr>
<td>Pyrexia (temperature greater than 37.8°C) *</td>
</tr>
<tr>
<td>Pulse rate greater than 90 bpm *</td>
</tr>
<tr>
<td>Anaemia *</td>
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<tr>
<td>Erythrocyte sedimentation rate (mm/hour) *</td>
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**Gallstones**

*Biliary colic, cholecystitis, ascending cholangitis, gall-stone ileus.*

Progressive increase in incidence of gallstones with age, unusual to get them before the 3rd decade. The prevalence is 2-3x higher in women than in men.

Gallstones are more common in Scandinavia, South America and Native North Americans. They are less common in Asian and African groups.

**Types of gallstones:**

* Most are one of two types:
  * Cholesterol stones
  * Bile pigment stones – Ca bilirubinate or polymer-like complexes with Ca, copper and some cholesterol.
* **Cholesterol gallstones**
  * Accounts for 80% in the Western world
  * The stones form due to cholesterol crystallization from gall bladder bile, this depends on:
    * Cholesterol supersaturation of bile
      * Due to excess cholesterol secretion into bile which may be due to increased HMG-CoA reductase activity.
      * Leptin has been shown to increase cholesterol secretion into bile – elevated levels of leptin during rapid weight loss may account for the increased incidence of stones.
      * Can also get supersaturation due to decreased bile salt content, this may occur as a consequence of bile salt loss e.g. terminal ileal resection or ileal involvement with Crohn’s disease
    * Crystallization-promoting factors within the bile
      * Need a balance between cholesterol crystallizing and solubilizing factors.
      * A high cholesterol diet increases biliary cholesterol secretion and decreases bile salt synthesis.
  * Motility of the gall bladder
* **Bile pigment stones**
  * Black pigment stones:
    * Calcium bilirubinate + network of mucin glycoprotein – complex with salts such as calcium carbonate and/or calcium phosphate
    * Have glass-like cross sectional surfaces and a mottled appearance.
    * Seen in 40-60% of patients with conditions that involve chronic excess bilirubin production e.g. haemolytic conditions, sickle cell disease and hereditary spherocytosis.
    * Pigment stones have also been linked to bacterial colonization of the biliary tree.
  * Brown pigment stones:
    * Muddy hue, cross section shows alternating brown and tan layers.
    * Ca salts + fatty acids + calcium bilirubinate
    * Almost always found in presence of bile stasis and/or biliary infection
    * Common cause of recurrent bile duct stones following cholecystectomy.

**Clinical presentation:**

* The majority are asymptomatic – detected incidentally. Over 10-15y period around 20% of these will cause symptoms.
* **Biliary or gallstone colic:**
  * Pain associated with temporary obstruction of the cystic or common bile duct by a stone migrating from the gall bladder
  * Pain is severe, but constant, and has a crescendo characteristic.
  * Often related to over indulgence with food, particularly that of high fat content.
  * Most common time is therefore mid-evening, lasting until early hours of the morning.
  * Location – epigastrium, RUQ, may radiate over the right shoulder and right subscapular region.
  * N+V may accompany more severe attacks.
  * If the pain is more protracted, associated with fevers and rigors, then it suggests secondary complications e.g. cholecystitis, cholangitis, or gallstone-related pancreatitis.
  * Differential diagnosis
    * IBS (spasm of the hepatic flexure)
    * Carcinoma of the right side of the colon
    * Atypical peptic ulcer disease
    * Renal colic
    * Pancreatitis
* **Acute cholecystitis:**
  * The initial event is due to obstruction to the gall bladder emptying – 95% of the time it is due to a gall bladder stone.
Leads to increase of gall bladder glandular secretion – progressive distension – can compromise the vascular supply to the gall bladder.

Also get inflammatory response due to the retained bile in the gall bladder.

Infection can then occur after the vascular and inflammatory events.

Initial features are similar to biliary colic. But will then progress to severe localized RUQ abdominal pain, due to parietal peritoneal involvement in the inflammatory process.

- Tenderness, guarding, rigidity

Differential diagnosis
- Acute pancreatitis
- Perforated peptic ulcer
- Intrahepatic abscess
- Basal pneumonia

Investigations:

- WCC and CRP
  - Normal if biliary colic and stone is in the neck of the gall bladder or cystic duct
  - Moderately raised in acute cholecystitis

- Serum bilirubin, ALP, ALT
  - Elevated when there is bile duct obstruction
  - May be marginally elevated in cholecystitis alone, even in the absence of duct obstruction

- Abdominal USS
  - Look for:
    - Gallstones in the gall bladder
    - Focal tenderness over the underlying gall bladder
    - Thickening of the gall bladder wall

- Biliary scintigraphy using technetium derivatives of iminodiacetate
  - Taken up by the hepatocytes and excreted into bile
  - Delineate the extrahepatic biliary tree

Management of Gall Bladder Stones:

Cholecystectomy

- Treatment of choice for nearly all symptomatic patients
  - If patient presents with gallstone-related complications, it should be carried out during that admission
  - If the patient has pain alone, an attack procedure can be planned

- Acute cholecystitis:
  - Initial management: NBM, fluids, analgesia, IV abx
  - Laparoscopic approach usually offered and done a few days, when the symptoms have settled.

- Laparoscopic approach:
  - Cost benefits
  - Day-care basis
  - Short period of ileus
  - CIs: extensive previous upper abdominal surgery, ongoing bile duct obstruction, portal HTN.
  - Complications: biliary leak from the cystic duct or gall bladder bed, injury to the bile duct itself (0.5%), overall mortality of 0.2%.

- Post-cholecystectomy syndrome:
  - RUQ pain, biliary type, occurring a few months after the operation.
  - Patients often report that the pain is identical to that for which the original operation was carried out.
  - Related to functional large bowel disease and colonic spasm at the hepatic flexure.
  - In a small proportion of patients the pain is related to a retained stone in the common bile duct
  - In an even smaller proportion of patients, hypertension of the sphincter of Oddi is implicated – can treat with endoscopic sphincterotomy.

Stone dissolution and shock-wave lithotripsy

- Pure or near-pure cholesterol stones can be solubilized by increasing the bile salt content of bile.
  - Use oral chenodeoxycholic acid and ursodeoxycholic acid

- Can use cholesterol-lowering agents e.g. statins, ezetimibe
- Extracorporeal shock wave lithotripsy – shockwave directed radiologically or by USS.

Complications of Gallstones:

1. Acute cholecystitis
2. Acute/ascending cholangitis
3. Gallstone-related pancreatitis
4. Biliary enteric fistula – where a gallstone has eroded through the wall of the gall bladder into the intestine, passage of a gallstone through into the small bowel can give rise to an ileus or a true obstruction.
Chronic Liver Disease

- When liver pathology follows an indolent (causing little or no pain) course it can present with features of chronic liver disease.
- Compensated = patient is relatively well
- De-compensated = substantial Sx and signs

Pathophysiology and clinical features:

Normal liver – insult or injury – inflammation – fibrosis – cirrhosis – liver failure or liver cancer

- Long-term, low-grade liver damage results in this progressive fibrosis
- This leads to reduced liver mass and portal HTN

Clinical features are a manifestation of these two things:

Other symptoms and signs include:

- Jaundice
- Parotid enlargement
- Hypotension
- Clubbing
- Dupuytren’s contractures
- Metabolic flap
- Small liver
- Splenomegaly
- Oedema
- Oestrogenic signs:
  - Palmar erythema, spider naevia, testicular atrophy

investigations:

- Identify the cause of the underlying liver disease
- Identify the trigger for decompensation
- Bloods:
  - Low Hb - haemorrhage, hypersplenism
  - Prolonged PT - synthetic failure, DIC
- Autoimmune profile/immunoglobulins
- Iron studies (haemochromatosis)
- Copper studies (Wilson’s disease)
- Viral serology (HBV, HCV)
- Alpha1-antitrypsin levels

Management:

- Identify and treat the cause of the clinical decompensation
- Acute complications:
  - Variceal bleeding
    - Blood products
    - Control the bleeding:
      - Urgent endoscopy and band ligation of the varices
      - Acute percutaneous portosystemic shunting – transjugular intrahepatic portosystemic stent shunt (TIPSS)
      - Vasoconstrictors (terlipressin)
    - If overwhelming haemorrhage
      - Balloon tamponade (Sengstaken-Blakemore tube)
    - Longer term:
      - Injection/banding varices
      - Beta-blockers to reduce risk
  - Acites – spironolactone, salt restriction

Problems due to reduced liver cell mass:

- Encephalopathy (metabolic flap/asterixis)
- Loss of lean body mass – most evident on shoulders, oedema and ascites often means that extent of loss is underestimated
- Coagulopathy

Problems caused by portal HTN:

- Varices: at sites of port-systemic communication e.g. lower oesophagus, piles, caput medusa. These can bleed torrentially and provoke decompensation.
- Ascites: due to Na retention, high portal pressure and low albumin. Have to also exclude spontaneous bacterial peritonitis (usually pneumococcus).
Cancer of the Oesophagus

- Post-cricoid carcinoma usually occurs in women, associated with Plummer-Vinson syndrome.
- Other oesophageal growths more commonly occur in elderly men.
- Distal tumours = most common
- Mid-third tumours = less common
- Proximal upper oesophageal tumours = least common
- 12/100,000 incidence
- Overall prognosis: 3-year survival <10%

Risk Factors:
1. Tobacco
2. Alcohol
3. SCC linked to: achalasia, coeliac disease
4. Adenocarcinoma linked to: Barrett’s oesophagus
(metaplastic change at the gastro-oesophageal junction)

Pathology:
- Macroscopic:
  - Nodule – ulcer – papilliferous mass or annular constriction
- Microscopic:
  - Adenocarcinomas (majority)
  - Squamous cell carcinomas

Spread:
- Local
  - Mediastinum = trachea, aorta, pleura and lungs
- Lymphatics
  - Paraoesophageal, tracheobronchial, supraventricular, subdiaphragmatic nodes
- Haematogenous: Liver, lungs

Clinical Features:
- Local symptoms: dysphagia, hoarseness, bovine cough
  - Short history of dysphagia in an elderly male is invariably due to carcinoma of the oesophagus or the upper end of the stomach.
  - Dysphagia progresses from solid to liquids
  - Hoarseness suggests invasion of the left recurrent laryngeal nerve by an upper oesophageal tumour
- Secondary deposits: enlarged neck nodes, may have jaundice and/or hepatomegaly
- General manifestations of malignant disease: weight loss, anorexia, anaemia.

Investigations:
- Oesophagoscopy - biopsy
- CT of the thorax and abdomen – primary, local invasion, secondary spread to liver and lymph nodes
- Endoscopic USS – assess tumour depth, detect local and lymphatic spread, facilitates FNA of lymph nodes, facilitates pre-op staging.
- PET – may be used with CT for staging and looking for metastatic disease
- Laparoscopy – exclude peritoneal metastases

Treatment:
- Curative?
  - Chemotherapy, then resection. Close the defect by mobilizing the stomach up to anastomose.
- Palliative?
  - Intubation with a stent if inoperable
  - Endoscopic laser therapy, radiotherapy (useful for SCCs), chemotherapy.
  - If inoperable: average life expectancy is around 3 months.

Plummer-Vinson Syndrome
- Dysphagia
- Iron-deficiency anaemia
- Middle-aged or elderly women
- Associated with hyperkeratinisation of the oesophagus
- Formation of a web in the upper part of the oesophagus
- Pre-malignant = associated with development of carcinoma in the cricopharyngeal region

Barrett’s oesophagus is an adenocarcinoma:
- Normal oesophagus: stratified squamous epithelium, long-standing reflux can lead to metaplasia to intestinal type columnar epithelium.
- Continuing inflammation can lead to dysplasia and malignant change – adenocarcinomas
- Male smokers, long Hx (>10y) of Barrett’s metaplasia, frequent symptoms of reflux (>3/week)
- Patients with metaplasia should undergo regular endoscopic surveillance, with biopsies to look for dysplasia
- Severe dysplasia (carcinoma in situ) is indication for endoscopic treatment or resection.

Benign:
Leiomyoma

Primary malignancy:
Carcinoma – squamous or adenocarcinoma
Leiomyosarcoma

Secondary malignancy:
Direction invasion from lung or stomach

Other oesophageal growths more commonly occur in elderly men.

Gastric metaplasia
Normal oesophagus is stratified squamous epithelium, to metaplasia to intestinal type columnar epithelium.
Complications of oesophagectomy:

- Procedure-specific
  - Anastomotic leak
  - Ischaemia
  - Stricture
  - Recurrent laryngeal nerve injury
  - Chylothorax – chyle leak into the thorax

- Functional disorders
  - Dysphagia
  - Delayed gastric emptying
  - Reflux
    - Dumping syndrome – secondary to vagotomy = weakness, abdo discomfort, abnormally rapid bowel evacuation after meals.
  - Diaphragmatic hernias

- Pulmonary complications
  - Pneumonia
  - Bronchospasm
  - ARDS
  - PE
  - Acute exacerbation of COPD

- Cardiac complications
  - AF
  - MI

Staging:

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<td>M1</td>
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