POCKET CONSULTANT

Gastroenterology

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Preface to the third edition

The field of gastroenterology continues its rapid pace of growth in the understanding of disease processes and the discovery of new diagnostic and treatment strategies. The success of the first and second editions of *Pocket Consultant in Gastroenterology* published in 1991 and 1998, in concert with significant advances in gastroenterology and hepatology over the past 5–6 years, has led to this third edition. The book's enduring appeal is a tribute to the original co-authors, Dr George Misiewicz and Rodney Taylor. Dr. Simon Travis is the leading force in this very useful book, and he has enlisted the fresh perspective of new authors, including Drs. Jane Collier, Tariq Ahmad and Hillary Steinhart. This third edition very effectively captures advances in new knowledge regarding the pathophysiology of gastrointestinal and hepatobiliary diseases and the management of patients with these disorders that has developed since the second edition. The authors continue to use the user-friendly format of short, focused paragraphs, and bulleted lists, supplemented by tables and figures where appropriate. The authors have struck the balance of remaining concise, yet being thorough in their discussions of all major disease entities. The whole story is provided in an economical format that makes this handbook so useful to busy clinicians seeking information in the fast pace of contemporary practice. This remarkable little book should be of value to primary care physicians, trainees in internal medicine and gastroenterology, as well as busy gastroenterology consultants, by providing a rapid and efficient way of refreshing themselves on specific topics. The font and layout of this book makes it particularly easy to seek out specific information. This third edition of *Pocket Consultant in Gastroenterology* is a goldmine of current information distilled into a terse and didactic style, making it indispensable in the clinic and office of the generalist and specialist gastroenterologist.

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Professor of Medicine
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President of the American Gastroenterology
Association 2003–4

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Simon Travis
Tariq Ahmad
Jane Collier
Hillary Steinhart

1 Alimentary Emergencies

1.1 Swallowed foreign body

Toddlers, the mentally disturbed and the elderly most commonly swallow foreign bodies. If no history is available, look for excessive salivation, regurgitation, choking or distress. Objects impact in the pharynx, lower end of the oesophagus, or pylorus. Pain or fever suggests perforation. Once through the pylorus, spontaneous passage is the rule, but perforation can occur in the ileocaecal region.

All cases

- Look in the mouth
- If the object is impacted in the fauces, call the ENT surgeon
- X-ray the chest and abdomen, but failure to visualise an object does not exclude its presence
- Look for surgical emphysema, mediastinal and subdiaphragmatic gas on X-ray
- Barium or Gastrografin examination is not indicated and may hinder endoscopy
- Address the underlying issues to avoid repeated ingestion, especially in prisoners or the mentally disturbed who may have ingested foreign bodies for individual gain

Bones, pins, glass and batteries

- Chest pain suggests perforation, and a small haematemesis may herald perforation of a major vessel. In either case, contact the thoracic surgeon urgently
- Sharp objects should be removed by an experienced endoscopist, unless they have passed the duodenum. A plastic sleeve over the endoscope helps prevent trauma during withdrawal. If possible, the endoscopist should practice snaring or grasping a similar object before starting the procedure in order to determine the best means of retrieval
- After endoscopic removal, further chest pain may indicate delayed perforation
- Batteries, especially small alkaline batteries ingested by toddlers, should be retrieved immediately if in the oesophagus. Corrosive perforation or heavy metal intoxication has been reported. Once in the small intestine, safe passage is the rule

Coins, beads and blunt objects

- Almost always pass spontaneously unless more than 5 cm long or 3 cm in diameter
- Reassure the patient or parents and advise them to check stools for 3 days
- Repeat abdominal X-ray after 36 h if there is doubt about progress. Documented arrest by X-rays for 72 h is an indication for surgical exploration

Body-packing (ingested packets of drugs)

- Smuggled packets of drugs may be swallowed, or secreted per rectum
- Intact packets can cause intestinal obstruction and if packets do not pass within 72 h, surgical removal is advisable

- Endoscopic removal is contraindicated because of the risk of rupturing the bags
- Packets may burst spontaneously and cause life-threatening overdose
- Heroin overdose causes constricted pupils, bradypnoea or coma. Hypoglycaemia or non-cardiogenic pulmonary oedema may occur later. Give intravenous naloxone 0.8 mg rapidly, to a maximum of 2.4 mg if necessary
- Cocaine causes dilated pupils, tachycardia and agitation. Convulsions, metabolic acidosis or coma may occur. Sedate with intravenous midazolam 5–10 mg and give oral propranolol 40 mg three times daily for a few days
- Severe overdose of any narcotic is an indication for ventilation and surgical removal of the packets, to stop drug absorption
- The doctor's immediate duty is the treatment of the patient if body-packing is discovered. Once treatment has been initiated, the appropriate authorities should be notified according to local legal regulations
- Questioning of the patient must wait until the patient is fit, and be sanctioned by the most responsible physician

1.2 Complete oesophageal obstruction

Bolus obstruction causes sudden, complete dysphagia for solids and liquids, with inability to swallow saliva. Food impacted against a benign or malignant stricture is the usual cause. Occasionally the presentation is delayed for a few days in the mentally handicapped or severely debilitated. The obstruction must be relieved urgently.

Clinical features

Ask about and look for:

- Duration of symptoms preceding obstruction
- Predisposing disease (stricture, carcinoma, Schatzki's ring)
- Triggering factors (steak, toast, fibrous foods, tablets)
- Dehydration
- Weight loss (suggests malignant obstruction)
- Supraclavicular nodes (from a carcinoma of the cardia)
- Complications (aspiration pneumonia, perforation)

Investigations

The endoscopist should be contacted as a priority.

- Full blood count—anaemia suggests carcinoma
- Serum electrolytes—high urea indicates dehydration
- Chest X-ray—look for a mediastinal fluid level (obstruction), absent gastric air bubble (obstruction) or right lower lobe consolidation (aspiration)
- Urgent endoscopy—must be performed by an experienced endoscopist
- A barium swallow risks aspiration and is inappropriate, unless the diagnosis is in doubt. This is *not* the same as in dysphagia without obstruction (Section 2.1, p. 51)

Management

- Intravenous fluids
- Endoscopic removal of the obstructing bolus

- Endoscopic dilatation can be done immediately after disimpaction
- Carbonated drinks occasionally disimpact fibrous debris, but endoscopy is needed when a food bolus is stuck for a few hours
- Fine-bore nasogastric feeding, or nutritional supplements are needed (Section 12.2, p. 378) if dilatation is delayed after removal of the bolus. Endoscopic placement of the tube is awkward, but indicated if it cannot be inserted in the normal way (p. 413)
- Intravenous metronidazole 500 mg and cefuroxime 750 mg three times daily for 5 days, if aspiration pneumonia is present

Prevention

Simple measures decrease the risk of acute obstruction in patients with oesophageal strictures or prosthetic oesophageal tubes (pp. 59 and 65):

- Avoid fibrous food (apples, oranges), steak and toast
- Wear dentures if edentulous
- Chew all solids well
- Carbonated drinks with meals
- Avoid oral potassium supplements, salicylates and large tablets>
- A proton-pump inhibitor (PPI; e.g. omeprazole 20–40 mg, lansoprazole 30 mg or rabeprazole 20 mg daily) delays or prevents restricturing in most patients and heals associated oesophagitis. PPIs should be continued indefinitely

1.3 Oesophageal rupture

Sudden chest pain after forceful vomiting is the cardinal symptom when the distal posterior oesophageal wall tears longitudinally in spontaneous perforation (Boerhaave's syndrome). Traumatic perforation after instrumentation or chest injury is more common than spontaneous rupture.

Differential diagnosis

Perforation presents with chest pain, respiratory distress, painful swallowing or subcutaneous emphysema. Early diagnosis is crucial to survival. Failure to consider the possibility is the commonest reason for misdiagnosis.

- Myocardial infarction (ECG, cardiac enzymes)
- Dissecting aneurysm (pulses, chest X-ray, urgent CT scan)
- Perforated peptic ulcer (rigid, silent abdomen, erect chest X-ray)
- Acute pancreatitis (amylase more than fourfold elevated)
- Spontaneous pneumothorax (chest X-ray in expiration)

Investigations

Confirm the diagnosis and site of perforation.

- Chest X-ray—look for mediastinal or subdiaphragmatic gas, or a hydro/pneumothorax (Fig. 1.1)
- Gastrografin swallow—in spontaneous rupture, tears are usually large and leak contrast; after instrumental rupture, tears are often small and do not leak contrast. Upper oesophageal perforations tend to leak into the mediastinum; mid-oesophageal perfo-

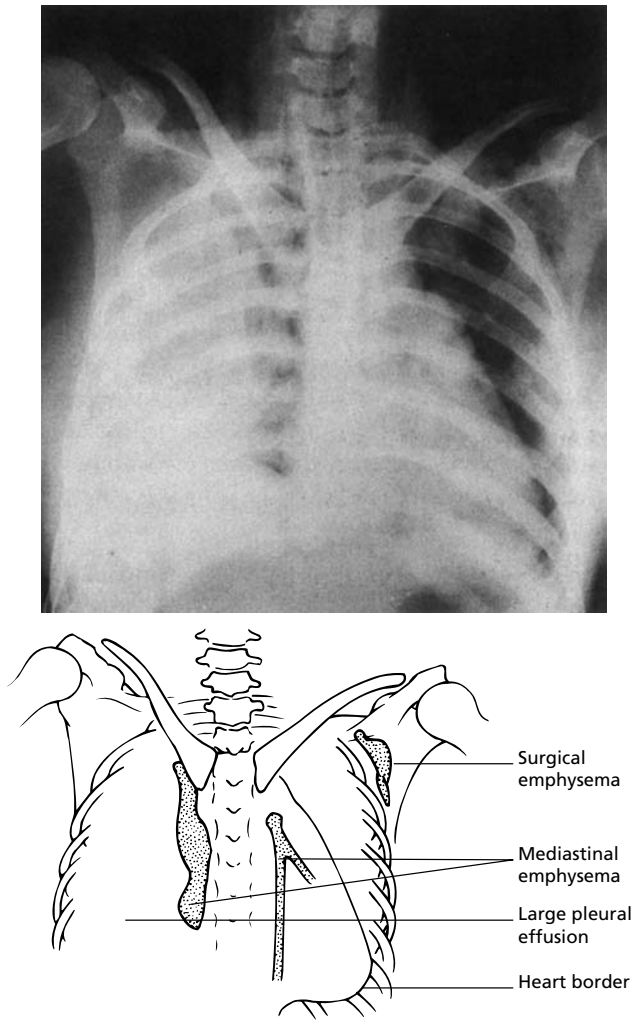

Fig. 1.1 Chest X-ray in oesophageal rupture showing consequences of oesophageal perforation. There is a large right pleural effusion, mediastinal emphysema and gross surgical emphysema in the neck and upper chest wall.

rations into the mediastinum and right pleura; and distal oesophageal perforations into the mediastinum, left pleural cavity or abdomen

Management

Oesophageal perforation is a potentially lethal condition. Conservative management is confined to highly specific situations. If the perforation has involved the pleural cavity, or has been contaminated by saliva, gastric contents or food, then surgery is mandatory.

Resuscitation

- Intravenous fluids

- Analgesia—intravenous morphine 2.5–5 mg every hour until pain is relieved, then every 4 h
- Involve thoracic surgical colleagues at an early stage

Spontaneous rupture
- Nil by mouth
- Surgical repair and drainage is almost always needed and should occur within 24 h
- Antibiotics (intravenous metronidazole 500 mg and cefuroxime 750 mg three times daily for 5 days)
- Enteral nutrition through a jejunostomy fashioned at the time of surgery is best

Instrumental rupture
- Small tears (with minor symptoms and no leakage of contrast) may be managed conservatively in conjunction with the surgeons. Large tears that leak contrast are managed as for spontaneous rupture
- Nil by mouth
- Nasogastric aspiration for 3 days
- Intravenous fluids
- Antibiotics as above
- Indications for surgery are a persistent fever, or pneumothorax after 48 h
- When perforation complicates palliative treatment of a malignant stricture, patients who are unfit for surgery may be managed by endoscopic insertion of a cuffed oesophageal tube

1.4 Caustic oesophageal injury

Ingestion of caustic cleaning fluids can cause progressive and devastating injury to the oesophagus and stomach. Most occur as accidents in children under 5 years. Symptoms and the appearance of the pharynx do not correlate with the extent of oesophageal or gastric injury.

Clinical features
- There may be no symptoms initially, but diagnosis is not difficult if an accurate history is obtained
- Identify the specific fluid ingested
- Hoarseness and stridor indicate pharyngeal or laryngeal injury
- Painful dysphagia or haematemesis indicate oesophageal oedema and ulceration. This may occur rapidly or be delayed for several hours
- Respiratory distress and shock occur when mediastinitis develops due to oesophageal necrosis
- Acute symptoms may resolve, to be followed by dysphagia after several weeks or months as scar tissue causes an oesophageal stricture

Investigations
- Chest X-ray—look for mediastinal air or oedema
- Direct laryngoscopy and endoscopy—to record the extent of injury. This should be performed under general anaesthetic, since sedation is unsatisfactory in children and

does not allow adequate views of the larynx or hypopharynx. Circumferential burns lead to strictures

Management

Resuscitation
- Nil by mouth
- Establish the airway by intubation or tracheostomy if respiratory symptoms or stridor are present
- Intravenous fluids
- Do *not* give neutralising agents

Minor, non-circumferential burns
- Allow liquids and then a light diet if tolerated
- Arrange psychiatric assessment if ingestion was a suicide attempt

Deep ulcers or circumferential burns
- Remain nil by mouth for 48 h
- Monitor for chest pain or fever, indicating delayed perforation or mediastinitis. If these occur, give intravenous antibiotics, liaise with thoracic surgical colleagues and start nasojejunal feeding
- Allow liquids and a light diet if asymptomatic after 48 h
- Omeprazole 40 mg daily, lansoprazole 30 mg daily, rabeprazole 20 mg daily or equivalent PPI for 4 weeks may reduce the rate of late stricturing, but steroids have no effect

Late complications
- Oesophagogastric strictures occur in about 25%, but almost invariably in those with deep ulcers or circumferential burns
- Review patient after 4 weeks and at 3, 6 and 12 months
- Arrange a barium swallow if dysphagia occurs, before oesophageal dilatation
- Repeated dilatation is frequently necessary. Shortening of the oesophagus may promote gastro-oesophageal reflux, so omeprazole 40 mg daily, lansoprazole 30 mg daily or equivalent PPI is reasonable to reduce restricturing
- Attention to nutrition is vital
- Surgery is indicated if patients cannot tolerate repeated dilatation
- The risk of carcinoma of the oesophagus is substantially increased

1.5 Acute bleeding: upper gastrointestinal tract

The aims of management in upper gastrointestinal bleeding are to stabilise the patient, stop active bleeding and prevent recurrent bleeding. There are about 90 admissions per 100 000 adults annually in the UK, with an overall mortality of 14% unless patients are admitted to a specialised bleeding unit, where a mortality of about 5% can be expected. Most bleeds from peptic ulcers stop spontaneously and about 25% can be identified who have no risk of rebleeding and can be rapidly discharged. The task is to distinguish the 20% who rebleed in hospital and who may need surgical intervention. A standard

clinical approach is recommended for every patient, so that patients at highest risk of rebleeding and death are identified early.

Clinical approach

- Assess severity
- Resuscitate
- Establish the site of bleeding
- Liaise with the surgical and intensive care teams on call
- Medical intervention
- Early surgery when appropriate

Assessment

The aim is to identify patients at high risk of rebleeding and death, by clinical and endoscopic examination. The Rockall score is an independently validated risk assessment score that is simple to apply and recommended. All patients with haematemesis or melaena must be treated actively until a stable baseline has been established. There is no room for complacency.

Rockall score

- The Rockall score is applied in two stages. First, there is a clinical score to be performed upon arrivals that estimates mortality:

Criterion		Score
• *Age*	< 60 years	0
	60–79 years	1
	> 80 years	2
• *Shock*	None	0
	Pulse & sBP > 100	1
	sPB < 100	2
• *Comorbidity*	None	0
	Cardiac/any major	2
	Renal/liver/malig.	3
• *Total initial score*		(max = 7)

Initial mortality risk score (pre-endoscopy)
0 = 0.2%
1 = 2.4%
2 = 5.6%
3 = 11.0%
4 = 24.6%
5 = 39.6%
6 = 48.9%
7 = 50.0%

- Then, after endoscopy the mortality score is updated to produce a final score:

Criterion	Score
• *Endoscopic diagnosis*	
No lesion, or M-W tear	0
All other diagnoses	1
Malignancy of upper GI tract	2
• *Stigmata of recent haemorrhage*	
None/haematin	0
Clot, visible vessel, blood in stomach	2
• *Final score after endoscopy*	(max = 11)

Final mortality score (after endoscopy)
0 = 0.0%
1 = 0.0%
2 = 0.2%
3 = 2.9%
4 = 5.3%
5 = 10.8%
6 = 17.3%
7 = 27.0%
8 = 41.1%

Document the following
In addition to a record of the assessment of the patient:
- Preceding symptoms (dyspepsia, vomiting, weight loss)
- Drug and alcohol ingestion
- Presence or absence of melaena on rectal examination
- Signs of chronic liver disease (Table 5.2, p. 137)

Causes
See Table 1.1.

Investigations and management

Resuscitation on arrival
- Ensure a patent airway
- Insert one 14- or two 18-gauge intravenous cannula
- If pulse > 100 b.p.m., give 500–1000 mL colloid (such as Haemaccel, Gelofusin, Pentaspan or pentastarch) over 30–60 min and repeat if necessary whilst waiting for blood
- Transfuse blood until haemodynamically stable in the first few hours, because initial haemoglobin is a poor indicator of the severity of the bleed. Subsequently transfuse up to haemoglobin of 10 g/dL
- Synthetic colloid or crystalloid will cause haemodilution: 1000 mL decreases the pre-transfusion haemoglobin by about 10%
- Reserve group O rhesus negative blood for dire emergencies (such as continuing massive bleeding and systolic BP < 80 mmHg despite 1000 mL intravenous colloid), when the risk from hypotension exceeds that from uncrossmatched blood
- Insert a urinary catheter in patients who need a central venous line (p. 11), to monitor urine output for information regarding fluid balance
- Ensure that the patient remains nil by mouth until endoscopy
- Do not insert a nasogastric tube, because this increases the risk of haemorrhage from gastric and oesophageal lesions
- Admission to a designated specialised unit for gastrointestinal bleeding reduces mortality to 5% or less. If this is not available, consider admission of patients with a predicted mortality > 10% (initial Rockall score ≥ 3) to a critical care unit and contact surgical colleagues as soon as the patient is resuscitated

Table 1.1 Differential diagnosis of haematemesis or melaena

Common	Less common (<5%)	Rare (1%)
Duodenal ulcer (35%)	Duodenitis	Hereditary telangiectasia
Gastric ulcer (20%)	Oesophageal varices	Aortoenteric fistula
Gastric erosion (6%)	Oesophagitis	Haemostatic defect
Mallory–Weiss tear (6%)	Tumours	Pseudoxanthoma elasticum
No lesion found (20%)		Haemobilia
		Pancreatitis
		Angiodysplasia
		Portal hypertensive gastropathy

Initial investigations

- Full blood count, crossmatch, coagulation studies and electrolytes
- Crossmatch 4 units of blood for patients with > 10% mortality risk (Rockall ≥ 3), but group and save alone for lower risk patients. Note that this is a practical application of the Rockall score, but it has not been validated for this purpose
- Haemodynamic status is a better guide to transfusion requirements than measured haemoglobin
- Arterial gases in those with cardiorespiratory disease
- ECG in high-risk patients
- Chest X-ray in high-risk patients (abdominal films rarely help)

Indications for a central venous line

- Signs of major haemorrhage (pulse > 100 b.p.m., systolic BP < 100 mmHg). Reasons for not inserting a central line in patients with a high (> 10%) predicted mortality (p. 9) must be carefully considered
- Rebleed during the same admission
- Inadequate peripheral venous access
- If a central venous line is needed, monitoring in a critical care area is advisable

Establish site of bleeding

- Arrange endoscopy after resuscitation, ideally within 12–24 h. Mucosal lesions and stigmata for rebleeding are otherwise missed. Ensure that the presence or absence of stigmata (p. 11) is recorded
- Indications for emergency endoscopy are continued bleeding, a rebleed in hospital, or if the patient is being considered for surgery
- Profuse haemorrhage may obscure the bleeding site. Gastric lavage to remove clots rarely alters management and can be hazardous. Repeat endoscopy after a further 12 h resuscitation is recommended. Immediate surgery should be a joint decision between surgeons and physicians
- Table 1.1 (p. 10) shows the differential diagnosis
- Interpret the endoscopy report intelligently: it should identify stigmata of recent haemorrhage (predicts risk of rebleeding), state whether there has been intervention (e.g. sclerotherapy of ulcers) and describe the position of ulcers (posterior duodenal ulcers overly a branch of the gastroduodenal artery that can rebleed vigorously)
- Stigmata of recent haemorrhage in the base of an ulcer and risk of rebleeding are:

Stigma	Risk of rebleeding
None	< 1%
Haematin (black spots)	5%
Adherent clot	30%
Visible vessel	50%
Bleeding vessel	80%

- Endoscopic intervention (below) halves, but does not abolish, the risk of rebleeding

Monitoring and discharge

- Pulse, BP, central venous pressure and urine output hourly, until stable
- Re-examine after 4 h
- Coagulation studies if > 4 units transfused
- Daily full blood count, urea and electrolytes for patients being transfused and for 2 days after
- Keep 2 units in the blood bank for 48 h after bleeding has stopped
- Patients who do not have endoscopic stigmata of high rebleeding risk (p. 11) and who have not had endoscopic intervention can safely start eating and drinking immediately after endoscopy and be discharged at any time thereafter, as long as there is adequate support at home
- Patients who have had endoscopic intervention (p. 11) should be kept in hospital for 72 h after bleeding has stopped

Medical intervention

These measures are not an alternative to surgery if an operation is indicated (p. 15), but may help stop bleeding or reduce the risk of rebleeding. Endoscopic intervention is indicated for patients with a peptic ulcer and active bleeding or non-bleeding visible vessel.

- Endoscopic intervention: all techniques halve the risk of rebleeding, but depend on local expertise and may not be available. Injection of adrenaline (up to 10 mL 1 : 10 000) around peptic ulcers, thermocoagulation and laser photocoagulation all have similar efficacy. Sclerosant (ethanolamine) is best avoided as necrosis and perforation have been reported. A combination of adrenaline (1 : 10 000) and thrombin (1000 U/mL) may be more efficacious at preventing rebleeding
- Intravenous omeprazole or pantoprazole (20–80 mg in 250 mL saline infused over 1 h, then 8 mg/h for 72 h) is only indicated after endoscopic intervention for bleeding peptic ulcers. It halves the risk of rebleeding and surgery and reduces mortality by one-third
- Ranitidine, oral omeprazole or other acid-suppressing drugs have **no** place in the initial treatment of bleeding. They should be reserved for treatment once a peptic ulcer has been diagnosed
- Tranexamic acid (1 g intravenously, three times daily for 72 h) has been shown on meta-analysis to reduce rebleeding and mortality. In the absence of a previous thromboembolic event, it is a reasonable adjunct in the treatment of high-risk patients until further trials are available. It also reduces the risk of recurrent bleeding from angiodysplasia
- Determination of *Helicobacter pylori* status should be done at the time of emergency endoscopy, using a biopsy urease (*Campylobacter*-like organism, CLO) test in patients with a bleeding ulcer (p. 87). Eradication therapy for *H. pylori*-positive patients (table 3.2, p. 89) is indicated as soon as oral feeding is restarted
- Terlipressin (Glypressin) 2 mg bolus, then 2 mg every 4 h is indicated for varices (below); Glypressin may not be available in all countries
- Other drugs (octreotide, vasopressin) do not have a proven role in the management of acute non-variceal gastrointestinal bleeding. A combination of ethinyloestradiol (50 μg) and norethisterone (1 mg/day) may decrease episodes of recurrent acute bleeding from angiodysplasia (such as hereditary telangiectasia)

Rebleeding

Rebleeding greatly increases mortality. Patients at high risk of rebleeding (based on endoscopic stigmata, p. 11) need to be recognised and the surgeons told of their admission. Patients are best admitted to a critical care area, where signs of rebleeding should be detected early. Signs of rebleeding are:

- Rise in pulse rate (a sensitive and early sign)
- Fall in central venous pressure
- Decrease in hourly urine output
- Haematemesis or continued melaena
- Looking at the patient (pallor, pulse, postural pressure drop and poor peripheral circulation) is as important as looking at the charts

Indications for surgery

Contact surgical colleagues at the outset, before an operation is necessary rather than when it is inevitable. Delay increases mortality. When the following criteria are met, surgery may be appropriate. Any decision not to operate should only be taken after discussion with the consulting surgeon.

- Age > 60 years and
  - > 4 units transfused in 24 h, or
  - one rebleed in hospital, or
  - continued bleeding, or
  - spurting vessel at endoscopy
- Age < 60 years and
  - > 8 units transfused in 24 h, or
  - one rebleed in hospital, or
  - continued bleeding, or
  - spurting vessel at endoscopy

The differential diagnosis of upper gastrointestinal bleeding is shown in Table 1.1 (p. 10). Individual topics are discussed below.

Oesophageal varices

Cirrhosis is the commonest cause of portal hypertension (p. 152) and oesophageal varices in the UK and North America. Whilst oesophageal varices can be found at endoscopy in almost 50% of patients with cirrhosis, less than one-third of these will bleed from their varices. Mortality during an acute bleed depends on the severity of liver disease on admission. Mortality according to Child's grade A is 10%, grade B is 25% and grade C is 50% (Table 5.5, p. 144), but 60–80% of all patients who bleed from varices will be dead within 4 years.

Assessment

- Bleeding from oesophageal varices is a complex clinical emergency for which control of bleeding is only one aspect
- Attention to infection, control of ascites (p. 147), encephalopathy (p. 144), alcohol withdrawal (Fig. 5.8, p. 177) and nutrition (p. 371) are vital for a successful outcome

Acute bleeding

- Resuscitate and monitor (p. 11). Colloids (synthetic, albumin or blood) are indicated and saline can be used in the acute situation. Avoid 5% dextrose if hyponatraemic

- 30% with known varices have another source of haemorrhage
- During active bleeding, correct disordered coagulation to international normalized ratio (INR) < 1.5 or prothrombin time < 22 s with fresh frozen plasma (FFP). However, FFP is contraindicated in the absence of bleeding, because this increases intravascular volume and variceal pressure, which may precipitate haemorrhage. Platelet transfusion may be necessary for thrombocytopenia (platelet count < 60×10^9/L). Discuss with the haematologists
- Arrange urgent endoscopy for banding or sclerotherapy by an experienced endoscopist
- Control of the airway, with endotracheal intubation if necessary, is extremely important when carrying out emergency endoscopy in a patient with an active variceal bleeding
- Give oral lactulose, starting at 90 mL/day or phosphate enemas if nil by mouth to prevent/treat hepatic encephalopathy (p. 145), intravenous vitamins (p. 393) and benzodiazepine, as necessary, for alcohol withdrawal (p. 177).

If endoscopic therapy is not available, or massive bleeding continues:

- Insert a Sengstaken tube until sclerotherapy/banding can be performed or repeated after 12 h
- Start splanchnic vasoconstrictors such as Glypressin (2 mg bolus, then 2 mg every 4 h, for up to 96 h). Glypressin rarely can be associated with ischaemic complications
- For recurrent bleeding after two attempts at endoscopic therapy the alternatives are transjugular intrahepatic portosystemic shunt (TIPSS) or oesophageal transection (see below)
- Where a stent is inserted between the hepatic and portal vein under radiological control, TIPSS can only be performed by experienced interventional radiologists, but is probably the procedure of choice where bleeding is not controlled by other means (Section 5.4, p. 153). Control of bleeding is excellent (approaching 100%), but 1 month mortality is still high (30–40%) owing to liver failure. Late complications (blocked stent, encephalopathy) are common
- Oesophageal transection has a mortality of 50% and should only be considered for patients without other organ failure, who were admitted in Child's group A or B (Table 5.5, p. 144). Splenectomy and proximal gastric devascularisation are needed to prevent subsequent bleeding from gastric varices
- Bleeding gastric varices are one cause of failed endoscopic haemostasis. TIPSS should be considered (Section 5.4, p 153), but preliminary reports suggest that endoscopic injection of bovine thrombin (2–10 mL of 1000 U/mL) or histoacryl (mixed with 1 : 1 lipiodal, 1–2 mL injected) controls active bleeding, although varices are not eradicated. Histoacryl has the potential to glue up the endoscope and should only be used by experienced endoscopists

Balloon tamponade

- Indicated for uncontrolled variceal bleeding, or recurrent haemorrhage despite sclerotherapy or banding
- To be inserted by experienced operators only
- Sedation, or a general anaesthetic to insert an endotracheal tube and secure the airway, may be necessary
- Insert a cooled, lubricated Sengstaken or Minnesota tube beyond 45 cm. The tube can usually be stiffened by inserting a well-lubricated pair of paediatric endoscopic biopsy forceps down the central lumen

- Inflate the gastric balloon with 300 mL tap water containing 50 mL of any intravenous X-ray contrast medium or 300 mL of air. Ensure gastric balloon channel is double-clamped
- Tie a 250 mL bag of saline to the tube to provide traction at the gastro-oesophageal junction, but be very careful to protect the mouth to prevent pressure necrosis
- Aspirate gastric and oesophageal ports (if present) hourly, and connect to a bag for continuous drainage
- It is rarely necessary to inflate the oesophageal balloon. If necessary because of persistent bleeding, inflate the oesophageal balloon to 30 mmHg with air, measured by manometer. Deflate for 5 min every hour.
- X-ray to check position (Fig. 1.2)
- Active bleeding is arrested in 90%. Continued bleeding usually means that the tube is misplaced or that there are gastric varices
- Deflate the oesophageal balloon after 6 h and the gastric balloon after 12–24 h to allow further endoscopic sclerotherapy or banding
- Complications include tracheal intubation, oesophageal rupture from inflating the gastric balloon in the oesophagus, or mucosal necrosis from leaving the balloon inflated for too long

Prevention of rebleeding from varices

- The highest risk of rebleeding is in the first 6 weeks
- Most gastroenterologists repeat endoscopic therapy at 1–2-week intervals until varices are obliterated
- Varices recur after obliteration in 40%, usually within 1 year
- Propranolol 40 mg three times daily decreases the risk of bleeding, can be used following initial endoscopic therapy and in this situation is as effective as repeated endoscopic therapy.
- Portal hypertensive gastropathy may cause bleeding after variceal obliteration. The gastric mucosa (usually fundal) has a characteristic 'snakeskin' appearance at endoscopy. Propranolol is the treatment of choice (p. 153)
- Surgical or percutaneous TIPSS are indicated for recurrent bleeding, especially in non-cirrhotic portal hypertension (p. 153)

Mallory–Weiss tears

A mucosal tear at the oesophagogastric junction due to forceful vomiting results in haematemesis. The typical features are:

- Initial vomitus does not contain blood
- Vomiting has often been provoked by alcohol
- 90% settle with conservative treatment
- Acid-suppressing drugs are unnecessary
- Continued bleeding can be controlled by endoscopic injection or thermocoagulation. Surgery is very rarely needed
- Bleeding after forceful vomiting may also be caused by prolapse of the gastric mucosa resulting in a focal area of haemorrhagic gastropathy ('hernia gastropathy') opposite the gastro-oesophageal junction

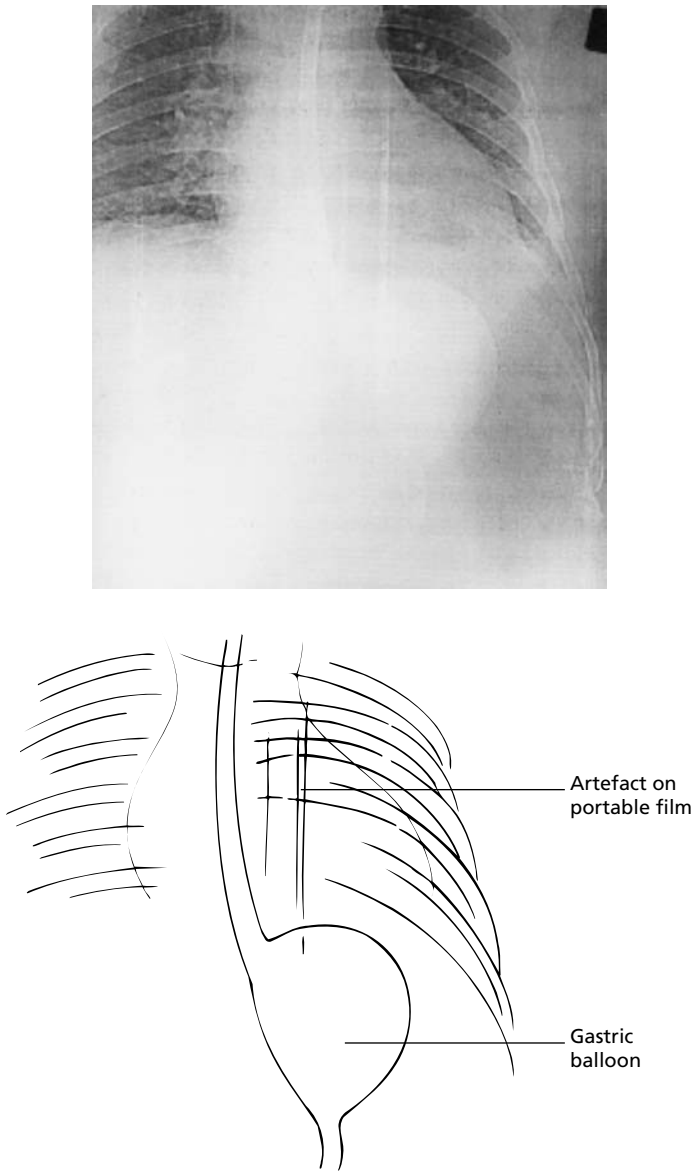

Fig. 1.2 X-ray showing Minnesota tube in correct position. Gastric balloon has been inflated with 300 mL water mixed with 50 mL contrast medium.

Acute gastric erosions and haemorrhagic gastropathy

Erosions are diagnosed endoscopically, but may be obscured by oozing from haemorrhagic gastropathy (gastritis is a misleading term that should be reserved for a histological diagnosis). Major haemorrhage from superficial gastric injury is unusual and the cause is often readily apparent.

Causes
- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Alcohol
- Stress (trauma, major surgery or patients in intensive care)

Specific treatment
- A PPI (any) is usually given for 1–4 weeks, depending on the cause
- Persistent bleeding is treated with intravenous tranexamic acid 1 g three times daily, in addition to a PPI (e.g. omeprazole or pantoprazole), which can be given intravenously
- Total gastrectomy is the last resort for continued bleeding after all medical treatment has been vigorously applied for 24–48 h, and should only be performed by an experienced surgeon
- It should be noted that despite the fact that antacids, ranitidine or sucralfate may prevent stress erosions in intensive care patients, there is almost no evidence that they reduce clinically significant bleeding or mortality

Gastric ulcer (Section 3.5, p. 92)
- Consider provoking causes (such as NSAIDs)
- Appropriate endoscopic haemostasis (p. 12) reduces rebleeding and mortality
- Give a PPI (lansoprazole 30 mg, omeprazole 20 mg, pantoprazole 40 mg, rabeprazole 20 mg daily) for 4 weeks once bleeding has stopped, together with *H. pylori* eradication therapy if endoscopic biopsies confirm infection
- For patients with NSAID-associated ulcers who cannot stop NSAIDs (p. 109), concomitant PPI therapy (lansoprazole or omeprazole) has replaced misoprostol in healing ulcers and preventing recurrence
- Arrange a repeat endoscopy after 8–12 weeks, to biopsy and take brushings for cytology from the ulcer site
- If surgery is needed for continued bleeding, a Billroth 1 gastrectomy is usually performed. Undersewing with a vagotomy and pyloroplasty is a simpler operation, but the ulcer cannot be examined histologically to exclude cancer. Wedge resection removes the ulcer and has the lowest morbidity in high-risk elderly patients, but long-term acid suppression is then necessary because it does not prevent recurrent ulceration

Duodenal ulcer (Section 3.9, p. 102)
- Combine ulcer healing with eradication of *H. pylori*
- The optimum treatment is triple, or quadruple eradication therapy (Table 3.2, p. 89)
- Repeat endoscopy is unnecessary, except in special circumstances (e.g. patients needing warfarin)
- Always confirm that eradication of *H. pylori* has been successful after an ulcer has bled, preferably by an isotope breath test (p. 86)
- Successful eradication of *H. pylori* significantly reduces the risk of rebleeding and the risk of bleeding from another ulcer to an extent similar to acid suppression. It is also cheaper in the long term and provides a cure
- Risk of repeat haemorrhage without eradication or maintenance therapy is 20% over 5–10 years, but is higher if associated with NSAIDs
- NSAID-associated ulcers heal with PPIs even if NSAIDs have to be continued, but are not directly associated with *H. pylori* (p. 110)

- Maintenance acid suppression (lansoprazole 15 mg, omeprazole 10 mg daily) is only indicated for patients at high risk of dying from the complications of recurrent ulceration (p. 110) when eradication therapy has been unsuccessful, or when NSAIDs have to be continued

'No source of bleeding found'

This is common (up to 20% acute bleeding) and can produce difficult management problems. Possible causes are:

- Lesion missed on endoscopy
- Mucosal lesion healed before patient endoscoped:
  - erosions
  - Mallory–Weiss tear
  - Dieulafoy's lesion (bleeding vessel with no surrounding ulceration, usually high on the greater curve)
- Bleeding from third part of the duodenum, or beyond:
  - jejunum (ulcerative jejunitis)
  - Meckel's diverticulum
  - colon
- Other:
  - nose bleed
  - rare causes of bleeding (Table 1.1, p. 10)

Management

The management of gastrointestinal bleeding from obscure and occult sources is discussed in more detail on p. 8 and Section 9.7 (p. 329; Fig. 9.9, p. 330).

- Reassess the patient—no further action is necessary for low-risk patients (p. 9)
- Repeat endoscopy in patients with a predicted mortality > 10% (p. 9)
- Investigate rare causes of bleeding (recheck coagulation, discuss small bowel radiology, endoscopic retrograde cholangiopancreatography (ERCP), ^{99}Tc sulphur colloid red cell scan (p. 20) or ^{99}Tc pertechnate scan with radiologists)
- Selective angiography during active bleeding (which must be at a rate of 1 U/4 h) is indicated after two negative endoscopies, preferably in a specialist unit
- Small bowel enteroscopy, including video capsule endoscopy (Section 9.7, p. 329) may be available in specialist units, but referral is necessary, and other procedures (mesenteric angiography, small bowel enema) will normally be repeated in the specialist unit
- Laparotomy, careful examination of the whole bowel with a bright light (e.g. sigmoidoscopy light source) and peroperative endoscopy is the ultimate procedure for recurrent episodes of active bleeding from obscure origin, but may still not identify the source and it is usually wise to refer to a specialist unit if this is contemplated

Aortoenteric fistula

Consider this rare diagnosis in *every* patient with an aortic graft and gastrointestinal bleeding. Exsanguination at the first bleed is uncommon. Small 'herald' bleeds occur for up to 2 weeks. Urgent abdominal CT scan, the diagnostic procedure of choice, may show haematoma around the graft. Endoscopy, if performed, should be to the fourth part of the duodenum, but surgery should not be delayed if hypotension has occurred. Aggressive surgery is needed as soon as the diagnosis is made, preferably in a specialist unit.

Acute bleeding: lower gastrointestinal tract

Bleeding from the colon is recognised by the passage of fresh red or reddish-brown altered blood per rectum. It is usually readily differentiated from upper gastrointestinal bleeding, because it has neither the smell, nor the tarry-black appearance of melaena. Upper tract bleeding rapid enough to cause red rectal bleeding is uncommon, and invariably associated with haemodynamic disturbance.

Clinical approach

- Resuscitate
- First episode, or recurrent (obscure) bleeding? See p. 329
- Establish the site of bleeding
- Specific treatment

Causes

See Table 1.2.

Investigations—first episode

Severe bleeding

- Full blood count, coagulation studies, crossmatch and check electrolytes
- Urgent colonoscopy after full bowel preparation (within 24–48 h) is safe and effective, although some prefer flexible sigmoidoscopy after phosphate enema preparation
- Gastroscopy—particularly in those with haemodynamic disturbance, to exclude brisk upper gastrointestinal haemorrhage
- Mesenteric angiography—if bleeding continues in excess of 1 U/4 h (Fig. 1.3; see Fig. 9.9, p. 330)

Slight/moderate bleeding

- Blood tests as above
- Colonoscopy once the bleeding has stopped

Investigations—obscure (recurrent) bleeding

Bleeding of obscure origin is defined as recurrent bouts of acute or chronic bleeding for which no source has been found after initial upper and lower gastrointestinal endoscopy. The topic is complex and further addressed in Section 9.7 (p. 329) and Fig. 9.9 (p. 330). Consider:

- Repeat colonoscopy—by an experienced operator, since angiodysplasia can be missed

Table 1.2 Causes of rectal bleeding

Common	Less common	Rare
Perianal conditions haemorrhoids fissures, prolapse Colorectal polyps Colorectal carcinoma Ulcerative colitis	Ischaemic colitis Crohn's disease Diverticular disease	Angiodysplasia Anorectal varices Small intestinal diverticula lymphoma Solitary rectal ulcer Vasculitis

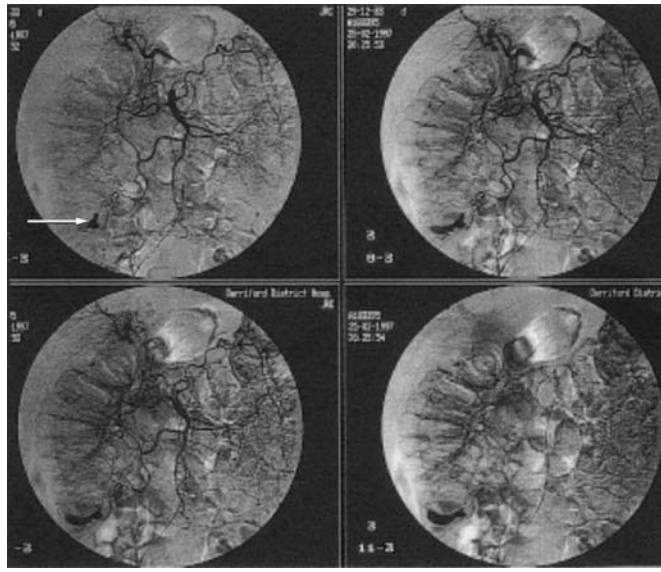

Fig. 1.3 Serial films from mesenteric angiography in a 63-year-old man with continuous bleeding (8 units transfusion in 24 h) and a normal upper gastrointestinal endoscopy. The bleeding vessel in the caecum (→) could not be embolised and laparotomy was necessary.

- Small bowel enema—better than a follow-through for identifying mucosal lesions (p. 423)
- Video capsule endoscopy (p. 329)
- ^{99}Tc sulphur colloid red scan—care must be taken not to overinterpret the scans, which may indicate the wrong area because blood accumulates in the colonic lumen. Early phase scans are the most helpful in localising the site of bleeding
- ^{99}Tc pertechnate Meckel's scan—at an early stage in young patients, but sensitivity is < 80%
- Angiography—during a subsequent episode of brisk bleeding (1 U/4 h)
- Self-induced rectal trauma is a rare cause of recurrent bleeding

Management

Severe bleeding stops spontaneously in 80% of cases after adequate blood replacement. Treatment of the cause (Table 1.2, p. 19) is then needed. In the remainder, bleeding is continuous or recurs, sometimes frequently, over many months. Identifying the site of persistent or recurrent bleeding is one of the most difficult problems in acute gastroenterology. Once the site is identified, surgical resection is indicated. If the site cannot be found, treatment depends on the pattern of bleeding.

Continuous bleeding

Referral to a specialist unit is advisable if bleeding persists after replacement of 6 units of blood and the source cannot be found. Surgery is an alternative and depends on local expertise. The options are:

- Laparotomy with peroperative colonoscopy after lavage through an appendicostomy. The physician is well advised to attend the laparotomy, but the difficulty of performing colonoscopy in these circumstances should not be underestimated

- Segmental resection if the site can be identified
- 'Blind' hemicolectomy can rarely be justified. If the site cannot be found and surgery is essential to stop bleeding, a subtotal colectomy should be performed

Intermittent bleeding
Referral to a specialist gastroenterology unit is advisable if the site of bleeding cannot be identified after three episodes of bleeding.

Rectal bleeding in children

The differential diagnosis is:
- Intussusception—commonest at 6–12 months
- Meckel's diverticulum
- Ulcerative colitis
- Foreign body
- Juvenile polyps—usually in the descending colon
- Intestinal haemangiomas
- Child (sexual) abuse

1.6 Acute abdominal pain

Clinical diagnostic accuracy is about 50%. Metabolic and extraintestinal causes (Table 1.3) should be considered if the diagnosis is in doubt.

Causes

The type of pain, relieving factors and progress are so variable that they rarely discriminate between diseases causing acute pain (Table 1.3; Table 1.4).

Table 1.3 Causes of acute abdominal pain

Common	Less common	Rare
Appendicitis	Cholangitis	Necrosis
Biliary colic	Mesenteric infarction	hepatoma
Cholecystitis	Pyelonephritis	fibroid
Diverticulitis	Torsion	Splenic infarction
Intestinal obstruction	ovarian cyst	Pneumonia
Perforated viscus	testicle	Myocardial infarction
Pancreatitis	omentum	Diabetic ketoacidosis
Peritonitis	Rupture	Porphyria
Salpingitis	ovarian cyst	Addisonian crisis
Mesenteric adenitis	ectopic pregnancy	Lead poisoning
Renal colic	aortic aneurysm	Tabes dorsalis
'Non-specific'	Prolapsed disc	Inflammatory aneurysm
	Abscesses	Volvulus
	Exacerbation of peptic ulcer	sigmoid
	Ileitis	caecum
	Crohn's	gastric
	Yersinia spp.	Herpes zoster

Table 1.4 Patterns of acute abdominal pain

	Appendicitis	Cholecystitis	Perforated viscus	Renal colic	Pancreatitis	Diverticulitis	Salpingitis	Intestinal obstruction
Site	C/RLQ	RUQ	UQs	R/L loins	UQs	LQs	LQs	Symmetrical
Duration	12–48 h	Days	< 12 h	< 12 h	< 48 h	Days	> 24 h	< 48 h
Severity	Moderate	Severe	Severe	Severe	Severe	Moderate	Moderate	Severe
Radiation	Nil	Shoulder, back	Nil	Groin	Nil	Nil	Groin, thigh	Nil
Aggravating factors	Movement cough	Inspiration	Movement cough	Nil	Movement	Movement cough	Nil	Eating

C/RLQ, central or right lower quadrant.
RUQ, right upper quadrant.
UQs, upper quadrants.
R/L, right or left.
LQs, lower quadrants.

Discriminating questions
- Site
- Duration
- Severity
- Radiation
- Aggravating factors

Also ask about
- Vomiting (if it precedes pain, a surgical cause is less likely)
- Time last ate or drank
- Bowel disturbance
- Urinary frequency
- Date of last menstrual period
- Previous abdominal surgery

Specifically examine for
- Distension
- Visible peristalsis
- Rebound tenderness, guarding or rigidity
- Pulsatile mass and peripheral pulses
- Hernial orifices
- Rectal *and pelvic* tenderness or masses
- Bowel sounds
- Epigastric bruit (normally audible in about 10% of thin patients)
- Fever (> 39°C with rigors suggests pyelonephritis, cholangitis or pneumonia)

Investigations

Every patient with acute abdominal pain should have on admission:
- Full blood count—leucocytosis may be absent in the elderly
- Electrolytes and creatinine
- Amylase—but many causes of slight elevation other than acute pancreatitis (p. 28)
- Group and save serum
- Urine examination—including *pregnancy* test if doubtful
- Erect chest X-ray—look for basal atelectasis and gas under diaphragm
- Supine abdominal X-ray—look for biliary and renal calculi, dilated bowel (> 2.5 cm small intestine, ≥ 6.0 cm colon), air in the biliary tree (p. 422)
- ECG
- Blood cultures—if febrile
- Abdominal ultrasound—the most discriminating investigation to identify appendicitis, cholecystitis, renal, or pelvic inflammatory disease
- Abdominal CT scan is indicated when ultrasound is technically difficult (often due to bowel gas or obesity) or does not identify a specific cause for acute abdominal pain. It is the initial imaging procedure of choice for suspected pancreatitis

Management—general principles

- Analgesia—do not withhold opiates for severe pain 'pending a surgical opinion', if the diagnosis is clear

- Perforation, peritonitis, or obstruction needs emergency surgery
- Observation overnight often clarifies a difficult diagnosis
- Nil by mouth until a decision about surgery has been made
- Specific management of common causes of abdominal pain are discussed below

Appendicitis

See Section 7.4 (p. 236).

Biliary colic

Distinguishing features

- Biliary colic typically causes a few minutes of right upper quadrant pain, with intervals of 1 h, and subsides after several hours. The pain may be exclusively high epigastric in location. Recurrent colic is a feature of chronic cholecystitis
- Fever, leucocytosis or pain lasting more than 12 h is likely to be due to acute cholecystitis
- Murphy's sign (tenderness in the right upper quadrant on inspiration) is positive in acute cholecystitis
- Flatulence, distension, fat intolerance and nausea are frequent but non-specific, and occur in other common conditions, especially irritable bowel syndrome (IBS) or non-ulcer dyspepsia
- Daily pain is unlikely to be due to biliary colic, even if gallstones are present

Management

- Ultrasonography will detect gallstones, although difficult in the obese and those with a fibrosed gall bladder. Repeat ultrasound after a fatty meal is a test of gall bladder function, and is abnormal (no contraction) in acute or chronic cholecystitis
- Other laboratory investigations are usually unhelpful, but a transient (<48 h) elevation in aspartate transaminase (AST) may occur in uncomplicated biliary colic
- Cholecystectomy is appropriate if symptoms are typical (Table 1.4, p. 22). Non-surgical options are discussed on p. 193

Acute cholecystitis

Distinguishing features

- Fever and persistent pain distinguish acute cholecystitis from biliary colic or chronic cholecystitis
- Impaction of a gallstone in the cystic duct causes > 90%
- Typical pain (Table 1.4, p. 22) occurs in < 50%
- Pain may be provoked by a fatty meal and builds up to a peak over 60 min, unlike the short spasms of biliary colic
- Fever develops after 12 h due to bacterial invasion and pain then becomes continuous
- Murphy's sign is sensitive, but not specific
- Calcified calculi (15%) and very rarely gas within biliary tree due to gas-forming organisms or a spontaneous choledochoduodenal fistula may be visible on plain abdominal X-ray

Complications

See Chapter 6 (p. 189).

- Recurrence (50%)
- Cholangitis due to associated common duct stones (10%)
- Mucocoele, empyema or gangrene of the gall bladder (1%)
- Biliary peritonitis (0.5%, with a mortality of 50%)
- Mirizzi's syndrome (obstructive jaundice due to external pressure on the common bile duct from inflammation around a stone impacted in the cystic duct)

Conservative management
- Confirm the diagnosis by ultrasound. Tenderness under the ultrasound probe in the presence of gallstones and a thickened gall bladder wall is effectively diagnostic. Isotope—hepatobiliary iminodiacetic acid (HIDA)—scans are also accurate, but not universally available
- Analgesia (intramuscular pethidine/meperidine 100 mg and hyoscine 20 mg, but not morphine, which can increase the pain)
- Intravenous fluids
- Nasogastric suction may be helpful, to alleviate vomiting if present
- Antibiotics (intravenous amoxycillin 500 mg three times daily and gentamicin 5 mg/kg once daily)
- Cholecystectomy at the earliest opportunity (see below)

Surgical management
- Optimum treatment is surgery on the same admission, on the next available list. Morbidity, total hospital stay, costs and mortality from complications of acute cholecystitis are lower compared to delayed cholecystectomy. Early surgery is especially appropriate in elderly patients or diabetics, because septic complications are more common
- Laparoscopic cholecystectomy is safe during acute cholecystitis in experienced hands. Cholecystostomy or percutaneous drainage of an empyema may be more appropriate in very sick elderly patients, but is rarely necessary
- The longer the interval between cholecystitis and surgery, the greater the risk of a recurrent attack: concern about an increased surgical complication rate 7–14 days after an acute attack is probably unfounded
- Other indications for surgery include signs of peritonitis and uncertainty about the diagnosis (when perforation or retrocaecal appendicitis cannot be excluded)

Cholangitis
See Section 6.4 (p. 200).

Diverticulitis
See Section 9.4 (p. 312).

Perforated viscus
The commonest cause is a perforated duodenal ulcer, followed by appendicitis, sigmoid diverticulum or carcinoma, Crohn's disease and gastric ulcers. Beware of a perforated peptic ulcer in patients already or recently in hospital for another reason.

Distinguishing features
- Sudden onset of severe, unremitting pain

- Temporary improvement 3–6 h later can trap the unwary
- Pain and peritonism may be absent in the elderly or those on steroids
- Abdomen fails to move with respiration
- Bowel sounds are usually absent
- Gas under the diaphragm on an erect chest X-ray is usual (70%), but not universal
- Lateral decubitus films for the very sick will also show free gas, but can be difficult to interpret
- Spontaneous sealing of the perforation occurs rarely

Surgical management
- Emergency surgery after vigorous intravenous resuscitation is almost invariably indicated
- Oversewing, omental patch and peritoneal lavage are customary for gastroduodenal perforation
- Hemicolectomy is indicated for right-sided colonic perforation, but distal perforation is probably best managed by resection, colostomy and rectal closure (Hartmann's procedure)
- Late complication of subphrenic abscess is best detected by ultrasound or CT scan, but an abscess may cause an immobile diaphragm, which can be readily detected by X-ray screening

Conservative management
- Indicated for the few patients in whom the risks are too high, or who refuse surgery
- Give intravenous fluids, antibiotics and nasogastric suction

Some surgeons advocate starting intravenous fluids, antibiotics and suction for 4–6 h and operating on those who do not improve, since this may have a lower mortality than emergency surgery for all patients. Whilst preoperative resuscitation is always advisable, this conservative surgical approach is not widespread.

Peritonitis

Fever, guarding, rebound tenderness and rigidity may be minimal in the elderly, the very young, patients on steroids and the immunocompromised. Bowel sounds are absent.

Causes
- Perforated viscus
- Local:
  - appendicitis
  - cholecystitis
  - diverticulitis
  - pancreatitis
  - salpingitis
- Spontaneous bacterial peritonitis (Section 5.2, p. 145)
- Continuous ambulatory peritoneal dialysis (CAPD)
- Rare:
  - tuberculous
  - sclerosing
  - granulomatous
  - periodic (familial Mediterranean fever)

Management

- Intravenous resuscitation
- Intravenous antibiotics (cefuroxime 750 mg and metronidazole 500 mg three times daily), after blood cultures
- Laparotomy
- Spontaneous bacterial peritonitis is usually due to *Escherichia coli* or *Streptococcus pneumoniae* in cirrhotic patients with ascites (p. 146). It does not display clinical features of pain, rebound and absent bowel sounds, but presents with encephalopathy or decompensation of stable chronic liver disease. Ascitic fluid should be sent for immediate Gram stain and absolute neutrophil count. Oral or intravenous ciprofloxacin 500 mg twice daily (400 mg twice daily for the intravenous form) should be started if the neutrophil count is > 250/mL, pending the result of culture. Long-term prophylaxis with ciprofloxacin is then appropriate
- CAPD peritonitis is usually caused by Gram-positive skin flora. Cloudy effluent, abdominal pain and tenderness are usual. Patients are often afebrile. Send fluid for Gram stain and absolute neutrophil count: a neutrophil count > 100/mL or the presence of organisms is diagnostic. Renal unit antibiotic policies differ, but intraperitoneal vancomycin 15 mg/L dialysate and gentamicin 4 mg/L are appropriate. Always consider silent intestinal perforation if bacteria other than skin flora are isolated. Laparotomy without further delay by an experienced surgeon is then indicated
- Tuberculous peritonitis is usually diagnosed at laparotomy, but can be suspected by a high ascitic adenosine deaminase level, although this is not widely available. Standard antituberculous chemotherapy for 9 months is advised (p. 357)

Acute pancreatitis

Acute pancreatitis affects around 200 000 people annually in the US, either as isolated or recurrent attacks. Recurrent attacks are distinguished from chronic pancreatitis by the absence of permanent impairment of exocrine or endocrine function. Initial symptoms are a poor indicator of prognosis. Complications (affecting 30%, p. 115) should be sought because early recognition improves prognosis and recovery is potentially complete. 10–15% of patients develop systemic inflammatory response syndrome (SIRS), leading to a fulminant course with pancreatic necrosis and multi-organ failure. Isolated and recurrent attacks are distinguished from chronic pancreatitis by the absence of permanent impairment of exocrine or endocrine function, but there is a spectrum of disease. Overall mortality ranges from 2–10% but this rate rises to 25% in the presence of infected pancreatic necrosis.

Distinguishing features

Abdominal pain with a serum amylase > fourfold the upper limit of normal is usually diagnostic, but late presentation (> 12 h) of a perforated duodenal ulcer, or ectopic pregnancy, may cause a similar rise in amylase

- The severity, rather than the nature, of the symptoms (pain and vomiting) characterises pancreatitis
- Diabetic coma is occasionally caused by acute pancreatitis

Predisposing factors

- Small gall stones—30–50%, more common in women, causing transient impaction at the ampulla

- No predisposing cause is found in about 15%. Many may be due to undetected microlithiasis
- Alcohol—10–40%, more common in men and in recurrent or chronic pancreatitis
- Trauma—about 5%, postoperative, post-ERCP or after blunt trauma
- Other causes of acute pancreatitis are rare:
  - drugs (azathioprine, mercaptopurine, sulphonamides, sodium valproate, antiretroviral agents)
  - pancreatic duct obstruction by benign stenosis, dyskinesia, pancreatic or ampullary carcinoma
  - pancreas divisum (congenital absence of pancreatic fusion)
  - end-stage renal failure
  - organ transplantation (drug-, viral- or lipid-related)
  - hypercalcaemia (acute or chronic pancreatitis)
  - hypertriglyceridaemia (> 10 mmol/L)
  - hypothermia
  - viral (mumps, coxsackie B4), pregnancy, hypothermia and arteritis cause isolated cases

Other abdominal causes of a moderately (< fourfold) raised serum amylase are:

- Acute-on-chronic pancreatitis in an alcoholic
- Perforated peptic ulcer (posterior perforation provokes pancreatitis)
- Ectopic pregnancy (amylase-secreting cells in the fallopian tube)
- Intestinal ischaemia, or infarction
- Aortic dissection
- Renal failure
- After any ERCP
- Consistent clinical features, a predisposing cause and an associated abnormality (such as hypocalcaemia or hypoxia) help discriminate acute pancreatitis from other causes of a moderately raised serum amylase

Complications of acute pancreatitis

- Local:
  - inflammatory mass (phlegmon)
  - pseudocyst (fluid collection; persistently raised amylase, p. 115)
  - pancreatic duct disruption (causing pseudocyst or ascites)
  - abscess (swinging pyrexia 1 week after attack)
  - jaundice (pancreatic oedema, or stones, can occlude the common bile duct)
- Paralytic ileus—exacerbates fluid and electrolyte imbalance
- Hypovolaemic shock—due to vomiting, hypoalbuminaemia, ascites or retroperitoneal haemorrhage
- Grey Turner's (flank) and Cullen's (periumbilical) signs are caused by tracking of blood-stained fluid
- Hypoxia—(PaO_2 < 8 kPa, or 60 mmHg) a prognostic factor and clinically underdiagnosed
- Hypocalcaemia—(< 2.0 mmol/L, corrected by adding 0.02 mmol/L for every g/L that the serum albumin < 40 g/L). Tetany is rare
- Acute renal failure—due to hypovolaemia, or disseminated intravascular coagulation (rare)
- Effusions—ascitic and pleural exudates with a high amylase