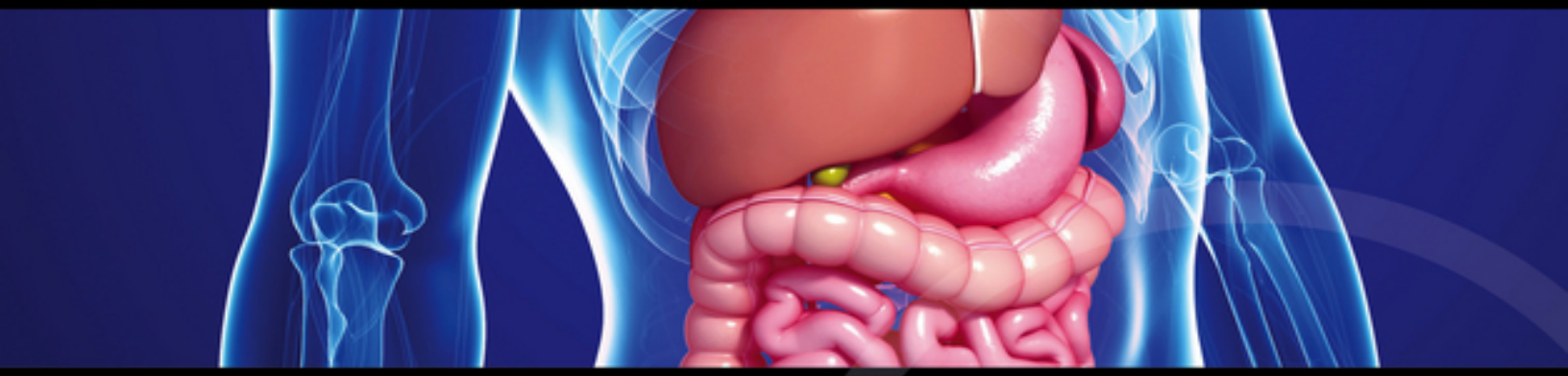
# GASTROENTEROLOGY AND HEPATOLOGY

## Lecture Notes



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2nd Edition

with website

WILEY Blackwell

# Preface to the second edition

> *He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all.*
>
> William Osler 1849–1919

> *Let the young know they will never find a more interesting, more instructive book than the patient himself.*
>
> Giorgio Baglivi 1668–1707

With the first edition of *Lecture notes in Gastroenterology and Hepatology* we strove to create a book that read just as we teach, incorporating the important and pertinent parts of anatomy, physiology and pathology into the structure of the lesson. In this way the building blocks of clinical understanding can illuminate rather than distract, or worse yet bore, the student or aspiring gastroenterologist. With this edition we have attempted to augment and clarify this concept by using a very uniform structure. Each section, where it is at all appropriate, is divided into subsections on the epidemiology, causes, clinical features, investigation, treatment and prognosis of the condition being considered. We hope this will help with understanding the material and integrating it into practice, as well as improve the textbook as a reference source or revision aid. Icons that alert the reader to those aspects of a disease that we believe are especially important, whether it be from a basic science, clinical or emerging topic perspective, have been added to focus the reader further.

This textbook is intended as a source of information and advice for all who are starting out in the important work of assisting people with disturbances of gastroenterological

and hepatological function, from the most junior of medical students to those preparing for specialist exams. To this end we have added ‘key point’ summaries to each chapter, as an aid to revision and understanding. We have also added an extensive ‘best answer’ multi-choice question section, in the style of the MRCP and FRACP examinations. These questions remain very clinically focused and draw heavily on our own clinical experiences. We believe that those early in their training will find them just as illuminating as those further along will find them challenging. Additionally, we have added further line diagrams and clinical images, with the aim of illustrating the important concepts without cluttering the book.

We firmly believe that our patients are the people who teach us the most. However, guidance from our colleagues and sources such as this book help light the path that each of us must walk to become the best clinician we can. We hope this book guides you in the same way that writing it has us.

*Stephen Inns and Anton Emmanuel*

# Preface to the first edition

> *Science is the father of knowledge, but opinion breeds ignorance*.
>
> Hippocrates 460–357 BCE

> *Specialised knowledge will do a man no harm if he also has common sense; but if he lacks this he can only be more dangerous to his patients.*
>
> Oliver Wendell Holmes 1809–94

The content of any textbook has, by definition, got to be factual. There are two potential consequences of this. The first, and most important, is that medical fact is based on science, and we have based this book on the anatomical, physiological and pathological basis of gastrointestinal practice. The second potential consequence of a factual focus is that the text can become rather dry and list like. To limit this we have tried to present the information from a clinical perspective – as the patients present in outpatients or casualty.

Gastroenterology is well suited to such an approach. It is a fundamentally practical speciality, with a strong emphasis on history, examination and endoscopy. The importance of integrating clinical assessment with investigation – both anatomical and physiological – is emphasised by the curiously limited range of symptoms despite the complexity of the gastrointestinal tract. The gut contains about three-quarters of the body’s immune cells; it produces a wider range of hormones than any single endocrine organ; it has almost as many nerves as the spinal cord; it regulates the daily absorption of microgram quantities of vitamins

simultaneously with macronutrients in 100 million times that amount.

We have tried to combine a didactic approach to facts alongside recurrently occurring themes to aid memory. For example, we have referred to the principles of embryology of the gut to give a common-sense reminder of how abdominal pain is referred and how the blood supply can be understood; approached lists of investigations by breaking them down to tests which establish the condition, the cause or the complications; approached aetiological lists by breaking down into predisposing, precipitating and perpetuating ones. We have eschewed ‘introductory chapters’ on anatomy, physiology and biochemistry as these are frequently skipped by readers who are often studying gastroenterology alongside some other subject. Rather, we have included preclinical material in the practical context of relevant disease areas (fluid absorption physiology in the section on diarrhoea, haemoglobin biochemistry in that on jaundice, etc.). Ultimately, we hope the reader uses this book as a source of material to help understand a fascinating speciality, pass exams in it, but above all be able to get as much as possible out of each patient seen with a gastrointestinal complaint.

*Anton Emmanuel and Stephen Inns*

# About the companion website

*Gastroenterology and Hepatology Lecture Notes* is accompanied by a companion website, featuring 16 in-depth case studies:

**www.lecturenoteseries.com/gastroenterology**

# Part I
# Clinical Basics

# 1
# Approach to the patient with abdominal pain

In gastroenterological practice, patients commonly present complaining of abdominal pain. The clinician's role is to undertake a full history and examination, in order to discern the most likely diagnosis and to plan safe and cost-effective investigation. This chapter describes an approach to this process. The underlying diagnoses and pathological mechanisms encountered in chronic pain are often quite different from those seen in acute pain, and for this reason each is considered in turn here.

## Chronic abdominal pain

### Anatomy and physiology of abdominal pain

Pain within the abdomen can be produced in two main ways: irritation of the parietal peritoneum or disturbance of the function and/or structure of the viscera (Box 1.1). The latter is mediated by autonomic innervation to the organs, which respond primarily to distension and muscular contraction. The resulting pain is dull and vague. In contrast, chemical, infectious or other irritation of the parietal peritoneum results in a more localised, usually sharp or burning pain. The location of the pain correlates more closely with the location of the pathology and may give important clues as to the diagnosis. However, once peritonitis develops, the pain becomes generalised and the abdomen typically becomes rigid (guarding).

Referred pain occurs due to the convergence of visceral afferent and somatic afferent neurons in the spinal cord.

Examples include right scapula pain related to gallbladder pain and left shoulder region pain from a ruptured spleen or pancreatitis.

### Box 1.1 Character of visceral versus somatic pain

*Visceral*

- Originates from internal organs and visceral peritoneum
- Results from stretching, inflammation or ischaemia
- Described as dull, crampy, burning or gnawing
- Poorly localised

*Somatic*

- Originates from the abdominal wall or parietal peritoneum
- Sharper and more localised

## Clinical features

### History taking

Initially the approach to the patient should use *open-ended* questions aimed at eliciting a full description of the pain and its associated features. Useful questions or enquiries include:

- ‘Can you describe your pain for me in more detail?’
- ‘Please tell me everything you can about the pain you have and anything you think might be associated with

it.’

- ‘Please tell me more about the pain you experience and how it affects you.’

Only following a full description of the pain by the patient should the history taker ask closed questions designed to complete the picture.

In taking the history it is essential to elucidate the presence of warning or ‘alarm’ features (Box 1.2). These are indicators that increase the likelihood that an organic condition underlies the pain. The alarm features guide further investigation.

### Box 1.2 Alarm features precluding a diagnosis of irritable bowel syndrome (IBS)

*History*

- Weight loss
- Older age
- Nocturnal wakening
- Family history of cancer or IBD

*Examination*

- Abnormal examination
- Fever

*Investigations*

- Positive faecal occult blood
- Anaemia
- Leucocytosis
- Elevated ESR or CRP
- Abnormal biochemistry

Historical features that it is important to elicit include those in the following sections.

## Onset

- **Gradual or sudden?** Pain of acute onset may result from an acute vascular event, obstruction of a viscus or infection. Pain resulting from chronic inflammatory

processes and functional causes is more likely to be gradual in onset.

## Frequency and duration

- **Colicky pain (which progresses and remits in a crescendo–decrescendo pattern)?** Usually related to a viscus (e.g. intestinal, renal and biliary colic), whereas constant intermittent pain may relate to solid organs (Box 1.3).
- **How long has the pain been a problem?** Pain that has been present for weeks is unlikely to have an acutely threatening illness underlying it and very longstanding pain is unlikely to be related to malignant pathology.

## Box 1.3 Characteristic causes of different patterns of abdominal pain

*Chronic intermittent pain*

- Mechanical:
  - Intermittent intestinal obstruction (hernia, intussusception, adhesions, volvulus)
  - Gallstones
  - Ampullary stenosis
- Inflammatory:
  - Inflammatory bowel disease
  - Endometriosis/endometritis
  - Acute relapsing pancreatitis
  - Familial Mediterranean fever
- Neurological and metabolic:
  - Porphyria
  - Abdominal epilepsy
  - Diabetic radiculopathy
  - Nerve root compression or entrapment
  - Uraemia
- Miscellaneous:
  - Irritable bowel syndrome
  - Non-ulcer dyspepsia
  - Chronic mesenteric ischaemia

*Chronic constant pain*

- Malignancy (primary or metastatic)
- Abscess
- Chronic pancreatitis
- Psychiatric (depression, somatoform disorder)
- Functional abdominal pain

## Location: Radiation or referral (Figure 1.1 right)

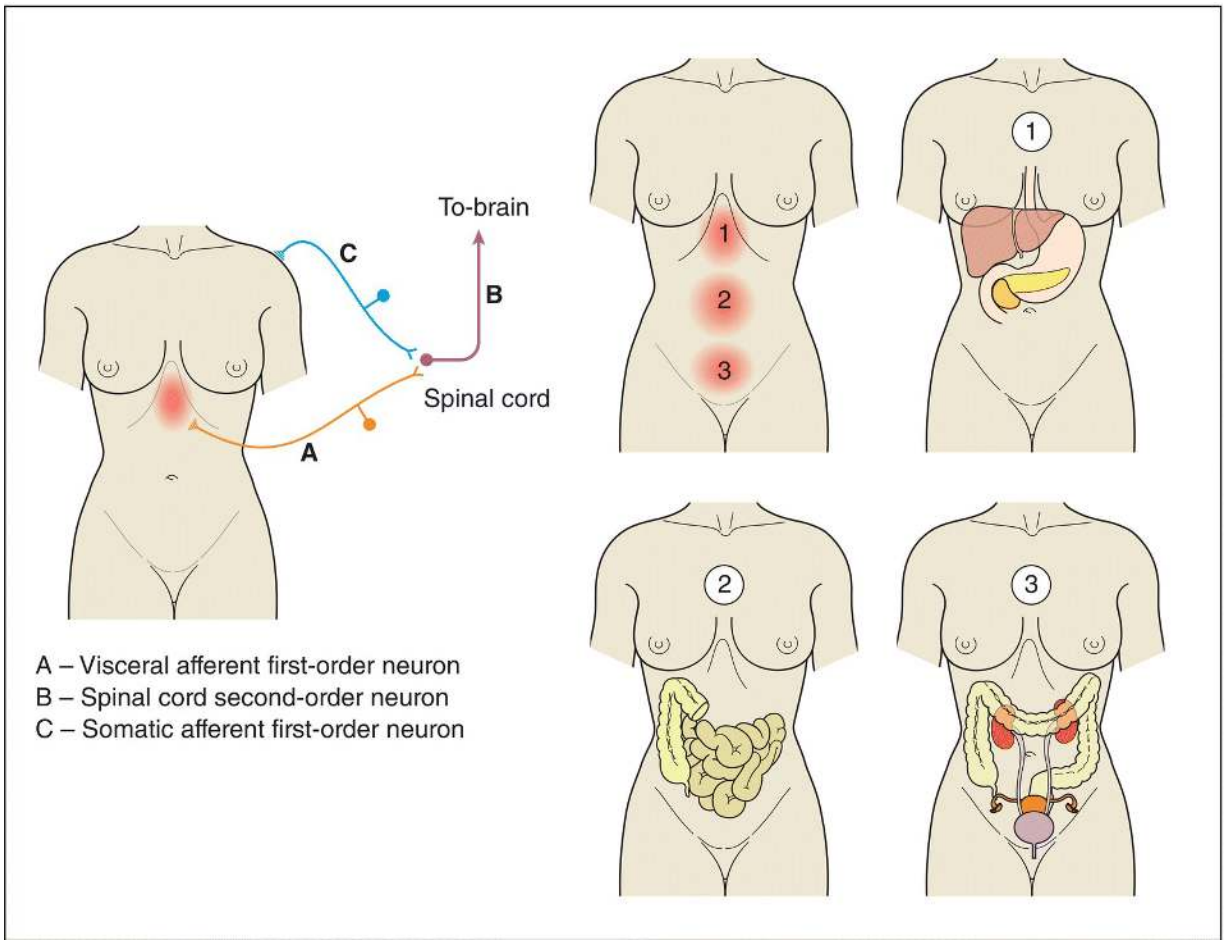


**Figure 1.1** *Left*: Mechanism of referred pain. *Right*: Location of pain in relation to organic pathology.

Source: Frederick H. Millham, in Feldman M, Friedman L, Brandt L (eds) (2010) *Sleisenger and Fordtran's Gastrointestinal and Liver Disease*, 9th edn, Philadelphia, PA: Saunders, Figure 10.1. Reproduced with permission of Elsevier.

- **Poorly localised?** Usually related to a viscus (e.g. intestinal, renal and biliary colic).
- **Located to epigastrium?** Disorders related to the liver, pancreas, stomach and proximal small bowel (from the embryological foregut).
- **Located centrally?** Disorders related to the small intestine and proximal colon (from the embryological midgut).
- **Located to suprapubic area?** Disorders related to the colon, renal tract and female reproductive organs (from the embryological hindgut).

**Radiation of pain** may be useful in localising the origin of the pain. For example, renal colic commonly radiates from the flank to the groin and pancreatic pain through to the back.

**Referred pain** (Figure 1.1 left) occurs as a result of visceral afferent neurons converging with somatic afferent neurons in the spinal cord and sharing second-order neurons. The brain then interprets the transmitted pain signal to be somatic in nature and localises it to the origin of the somatic afferent, distant from the visceral source.

## Character and nature

- **Dull, crampy, burning or gnawing?** Visceral pain: related to internal organs and the visceral peritoneum.
- **Sharp, pricking?** Somatic pain: originates from the abdominal wall or parietal peritoneum (Box 1.1).

One process can cause both features, the classic example being appendicitis, which starts with a poorly localised

central abdominal aching visceral pain; as the appendix becomes more inflamed and irritates the parietal peritoneum, it progresses to sharp somatic-type pain localised to the right lower quadrant.

## Exacerbating and relieving features

Patients should be asked if there are any factors that ‘bring the pain on or make it worse’ and conversely ‘make the pain better’. Specifically:

- **Any dietary features, including particular foods or the timing of meals?** Patients with chronic abdominal pain frequently attempt dietary manipulation to treat the pain. Pain consistently developing soon after a meal, particularly when associated with upper abdominal bloating and nausea or vomiting, may indicate gastric or small intestinal pathology or sensitivity.
- **Relief of low abdominal pain by the passage of flatus or stool?** This indicates rectal pathology or increased rectal sensitivity.
- **The effect of different forms of analgesia or antispasmodic when used may give clues as to the aetiology of the pain.** Simple analgesics such as paracetamol may be more effective in treating musculoskeletal or solid organ pain, whereas antispasmodics such as hyoscine butylbromide (Buscopan) or mebeverine may be more beneficial in treating pain related to hollow organs.
- **Pain associated with twisting or bending?** More likely related to the abdominal wall than intra-abdominal structures.
- **Pain severity** may be affected by stress in functional disorders, but increasing evidence shows that psychological stress also plays a role in the mediation of

organic disease, such as inflammatory bowel disease (IBD).

### Any associated symptoms?

The presence of associated symptoms may be instrumental in localising the origin of the pain.

- **Relationship to bowel habit: frequency, consistency, urgency, blood, mucus and any association of changes in the bowel habit with the pain are important.** Fluctuation in the pain associated with changes in bowel habit is indicative of a colonic process and is typical of irritable bowel syndrome (IBS).
- **Vomiting or upper abdominal distension?** Suggestive of small bowel obstruction or ileus.
- **Haematuria?** Indicates renal colic.
- **Palpable lump in the area of tenderness?** Suggests an inflammatory mass related to transmural inflammation of a viscus, but may simply be related to colonic loading of faeces.

## Examination technique

The physical examination begins with a careful **general inspection**.

- **Does the patient look unwell?** Obvious weight loss or cachexia is an indicator of malabsorption or undernourishment.
- **Is the patient comfortable? If in acute pain, are they adopting a position to ease the pain?** The patient lying stock still in bed with obvious severe pain may well have peritonitis, whereas a patient moving about the bed, unable to get comfortable, is more likely to have visceral pain such as obstruction of a viscus.

- **Observation of the skin** may demonstrate jaundice, pallor associated with anaemia, erythema ab igne (reticular erythematous hyperpigmentation caused by repeated skin exposure to moderate heat used to relieve pain) or specific extraintestinal manifestations of disease (Table 1.1). Leg swelling may be an indicator of decreased blood albumin related to liver disease or malnutrition.
- **Observe the abdomen** for visible abdominal distension (caused by either ascites or distension of viscus by gas or fluid).
- **Vital signs, including the temperature**, should be noted.
- **Examination of the hands** may reveal clues to intra-abdominal disease. Clubbing may be related to chronic liver disease, IBD or other extra-abdominal disease with intra-abdominal consequences. Pale palmar creases may be associated with anaemia. Palmar erythema, asterixis, Dupytren's contractures and spider naevi on the arms may be seen in chronic liver disease.
- **Inspection of the face** may reveal conjunctival pallor in anaemia, scleral yellowing in jaundice, or periorbital corneal arcus indicating hypercholesterolaemia and an increased risk of vascular disease or pancreatitis.
- **Careful cardiac and respiratory examinations** may reveal abnormalities associated with intra-abdominal disease. For example, peripheral vascular disease may indicate that a patient is at risk for intestinal ischaemia; congestive heart failure is associated with congestion of the liver, the production of ascites and gut oedema; and pain from cardiac ischaemia or pleuritis in lower-lobe pneumonia may refer to the abdomen.

- **Examination of the gastrointestinal (GI) system per se begins with careful inspection of the mouth with the aid of a torch and tongue depressor**. The presence of numerous or large mouth ulcers or marked swelling of the lips may be associated with IBD. Angular stomatitis occurs in iron deficiency. Glossitis may develop in association with vitamin $B_{12}$ deficiency caused by malabsorption.
- **Examination of the thyroid is followed by examination of the neck and axilla** for lymphadenopathy.
- **Careful inspection of the abdomen is repeated and the abdominal examination is completed as described in Part IV, taking great care to avoid causing undue additional discomfort**. The examiner must be careful to ask first whether there are any tender spots in the abdomen before laying on a hand. Special care should be taken, starting with very light palpation, asking the patient to advise the examiner of any discomfort felt and by watching the patient's expression at all times. Only if light palpation is tolerated in an area of the abdomen should deep palpation be undertaken in that area. A useful additional sign to elicit when areas of localised tenderness are found is Carnett's sign. While the examiner palpates over the area of tenderness, the patient is asked to raise their head from the bed against the resistance provided by the examiner's free hand on their forehead. If the palpation tenderness continues or intensifies during this manoeuvre, it is likely to be related to the abdominal wall rather than to intra-abdominal structures. 

**Table 1.1** Extraintestinal manifestations of hepatogastrointestinal diseases.

| Disease | Dermatological | Musculoskeletal |
|---|---|---|
| Inflammatory bowel disease: | | |
| • Crohn's disease | Erythema nodosum, pyoderma gangrenosum | Axial arthritis more common |
| • Ulcerative colitis | Erythema nodosum, pyoderma gangrenosum | Axial and peripheral arthritis similar in frequency |
| Enteric infections (Shigella, Salmonella, Yersinia, Campylobacter) | Keratoderma blennorrhagica | Reactive arthritis |
| Malabsorption syndromes: | | |
| • Coeliac sprue | Dermatitis herpetiformis | Polyarthralgia |
| Viral hepatitis: | | |
| • Hepatitis B | Jaundice (hepatitis), livedo reticularis, skin ulcers (vasculitis) | Prodrome that includes arthralgias; mononeuritis multiplex |
| • Hepatitis C | Jaundice (hepatitis), palpable purpura | Can develop positive rheumatoid |

| Disease | Dermatological | Musculoskeletal |
| --- | --- | --- |
| | palpable purpura | rheumatoid factor |
| Henoch−Schönlein purpura | Palpable purpura over buttocks and lower extremities | Arthralgias |

## Approach to differential diagnosis of pain and directed investigation

Following a careful history and examination, the clinician should be able to develop an idea of which organ(s) is/are likely to be involved and what the likely pathogenesis might be considering the demographics of the patient and the nature of the pain. It is important to list the most likely diagnoses based on these factors first. The differential can then be expanded by the application of a surgical sieve (as described in Part IV) to add the less likely possibilities.

Most patients should have a minimal blood panel to rule out warning features and to make any obvious diagnoses. These would include full blood count (FBC); urea, creatinine and electrolytes; liver function tests (LFTs); and coeliac antibodies, especially if there is any alteration of bowel habit. Further testing should be directed at each of the most likely diagnoses in the list of differential diagnoses. The clinician should attempt to choose the range of investigations that will most cost-effectively examine for the greatest number of likely diagnoses with the greatest sensitivity and specificity (see Clinical example 1.1).

## CLINICAL EXAMPLE 1.1

**HISTORY** Ms AP is a 37-year-old woman who describes 1 year of intermittent right lower quadrant abdominal pain. She is Caucasian, her body mass index is 19 $kg/m^2$ and she smokes 20 cigarettes/day. The pain first came on following an illness associated with vomiting and diarrhoea. She saw her GP and was given antibiotics, but stool culture revealed no pathogens. The diarrhoea settled spontaneously and she currently opens her bowels three times a day to soft-to-loose stool with no blood or mucus. The pain is aching and intermittent, but seems to be worse during periods of life stress. It often occurs about half an hour after meals and is associated with abdominal bloating and on occasion nausea, but no vomiting. It lasts 30 minutes to some hours at a time. There is no position in which she can get comfortable and she describes herself as 'writhing around' with the pain. She has reduced the size of her meals and avoids excess fibre, which seems to help. No specific foods contribute to the symptoms. Opening her bowels does not relieve the pain. She has trialled no medications. She has lost 5 kg in weight in the last year. The pain does not wake her at night and there is no nocturnal diarrhoea. There has been no change in the menstrual cycle and no association of the pain with menses. There has been no haematuria and she has never passed stones with the urine. She is on no regular medication. There is no significant family history.

**EXAMINATION** Observation reveals a thin woman with no hand or face signs of gastrointestinal disease; in particular, no pallor, skin lesions, angular stomatitis, mouth ulceration or tongue swelling. The abdomen is not distended. There is localised tenderness in the right

lower quadrant. No mass is palpable. Carnett's sign is negative (the tenderness disappears when the patient lifts her head from the bed). There is no organomegaly. Bowel sounds are normal.

**SYNTHESIS (SEE TABLE 1.2)** In considering the differential diagnosis and investigation plan, one must first consider which organ(s) might be involved, then what the possible pathologies in those organs might be, before considering the investigations that are useful for each possible pathology in each organ system. This will allow a tailored approach to directed investigation that is cost-effective and limits the potential harm to the patient.

**Likely organ involved** In considering the differential diagnosis, one must first consider which organ(s) might be involved. The central and aching nature of the pain, as well as the fact that it causes the patient to writhe around, suggest that it is originating in a hollow organ, perhaps the small bowel or proximal colon. The localised tenderness further localises the pain to the distal small bowel or proximal colon. The onset was associated with a probable gastroenteritis and the bowel habit is mildly disturbed, also suggesting an intestinal cause. The lack of association with menses and the absence of other urinary symptoms make conditions of the reproductive system and renal tract less likely.

**Likely pathology** The most likely diagnoses in this setting are inflammatory bowel disease and functional GI disease (IBS). Most patients with gastrointestinal symptoms require serological testing for coeliac disease, as it is very common and its symptoms commonly mimic other diseases. Use of a surgical sieve applied to the distal small bowel and proximal colon expands the list to include infection, neoplasia (including benign neoplasia

resulting in intermittent intussusceptions) and, although unlikely in a young woman, intestinal ischaemia. Less likely causes in other organ systems include biliary colic, ovarian pain and renal colic.

**Investigation plan** Initial investigation reveals a microcytic anaemia but no abnormality of the renal and liver tests and negative coeliac antibodies. Stool culture and examination for ova, cysts and parasites are negative. Urine dipstick shows no blood. Warning features in the form of weight loss and anaemia prompt further investigation. The investigation of choice to rule out inflammatory disease in the terminal ileum and colon is ileocolonoscopy and biopsy. The standard investigation for the remaining small bowel is computed tomography (CT) or magnetic resonance imaging (MRI) enterography. This will also effectively investigate for biliary disease, ovarian disease and renal disease. More expensive and invasive investigations designed to examine for the less likely diagnoses are not utilised in the first instance (see Chapter 6).

At colonoscopy the caecum and terminal ileum are seen to be inflamed and ulcerated. Biopsies show chronic inflammation, ulceration and granuloma formation, suggestive of Crohn’s disease. CT shows no disease of the ovaries, kidneys or biliary tree, but does suggest thickening and inflammation of the terminal ileum and caecum. There is no significant lymphadenopathy. A diagnosis of probable Crohn’s disease is made and the patient treated accordingly.

**Table 1.2** Approach to differential diagnosis and directed investigation for Ms AP.

| Likely organ involved | Likely pathology | Investigation choices | Investigation plan |
|---|---|---|---|
| Small bowel and colon | Inflammatory bowel disease | Ileocolonoscopy<br>CT/MRI enterography<br>US small bowel<br>Capsule endoscopy | Stool test<br>Ileocolonoscopy<br>CT (or MRI) enterography |
| | Irritable bowel syndrome | Suggestive symptom complex in the absence of other diagnoses | |
| | Infection | Stool culture and examination for *C. difficile*, ova, cysts and parasites<br>Specific parasitic serology if peripheral eosinophilia | |
| | Neoplasia | Ileocolonoscopy and enterography (CT/MRI) or capsule endoscopy | |
| | Ischaemia | Angiography | |

| Likely organ involved | Likely pathology | Investigation choices | Investigation plan |
|---|---|---|---|
| Biliary system | Biliary stones, neoplasia | Ultrasound abdomen<br>MRCP<br>Endoscopic ultrasound<br>ERCP | |
| Ovary | Ovarian cyst, torted ovary | Ultrasound pelvis<br>CT pelvis | |
| Renal | Renal stones | Ultrasound abdomen<br>CT urogram | |

US, ultrasound; MRI, magnetic resonance imaging; MRCP, magnetic resonance cholangiopancreatography; ERCP, endoscopic retrograde cholangiopancreatography; CT, computed tomography scan.

# Acute abdominal pain

The patient presenting with acute abdominal pain is a particular challenge to the clinician. Pain production within the abdomen is such that a wide range of diagnoses can present in an identical manner. However, a thorough history and examination still constitute the cornerstone of assessment. It is essential to have an understanding of the mechanisms of pain generation. Equally, it is important to recognise the alarm symptoms and initial investigative findings that help to determine which patients may have a serious underlying disease process, who therefore warrant more expeditious evaluation and treatment.

## Clinical features

### History taking

The assessment of the patient with abdominal pain proceeds in the same way whatever the severity of the pain; however, in the acute setting, assessment and management may need to proceed simultaneously and almost invariably involve consultation with a surgeon. Much debate has centred on the pros and cons of opiate analgesia in patients with severe abdominal pain, as this may affect assessment. The current consensus is that while judicious use of opiate analgesia may affect the examination findings, it does not adversely affect the outcome for the patient and is preferable to leaving a patient in severe pain.

The history (Table 1.3) gives vital clues as to the diagnosis and should include questions regarding the location (Figure 1.2), character, onset and severity of the pain, any radiation or referral, any past history of similar pain, and any associated symptoms.

**Table 1.3** Historical features in acute abdominal pain examination.

| **Where is the pain?** | **See Figure 1.2** |
|---|---|
| **Character of the pain?** | Acute waves of sharp constricting pain that ‘take the breath away’ (renal or biliary colic)<br>Waves of dull pain with vomiting (intestinal obstruction)<br>Colicky pain that becomes steady (appendicitis, strangulating intestinal obstruction, mesenteric ischaemia)<br>Sharp, constant pain, worsened by movement (peritonitis)<br>Tearing pain (dissecting aneurysm)<br>Dull ache (appendicitis, diverticulitis, pyelonephritis) |
| **Past similar pain?** | ‘Yes’ suggests recurrent problems such as ulcer disease, gallstone colic, diverticulitis or mittelschmerz |
| **Onset?** | Sudden: ‘like a thunderclap’ (perforated ulcer, renal stone, ruptured ectopic pregnancy, torsion of ovary or testis, some ruptured aneurysms)<br>Less sudden: most other causes |
| **Severity of the pain?** | Severe pain (perforated viscus, kidney stone, peritonitis, pancreatitis)<br>Pain out of proportion to physical findings (mesenteric ischaemia) |

| | |
|---|---|
| **Where is the pain?** | **See Figure 1.2** |
| **Radiation/referral?** | Right scapula (gallbladder pain)<br>Left shoulder region (ruptured spleen, pancreatitis)<br>Pubis or vagina (renal pain)<br>Back (ruptured aortic aneurysm) |
| **Relieving factors?** | Antacids (peptic ulcer disease)<br>Lying as quietly as possible (peritonitis) |
| **Associated symptoms?** | Vomiting precedes pain and is followed by diarrhoea (gastroenteritis)<br>Delayed vomiting, absent bowel movement and flatus (acute intestinal obstruction; the delay increases with a lower site of obstruction)<br>Severe vomiting precedes intense epigastric, left chest or shoulder pain (emetic perforation of the intra-abdominal oesophagus) |

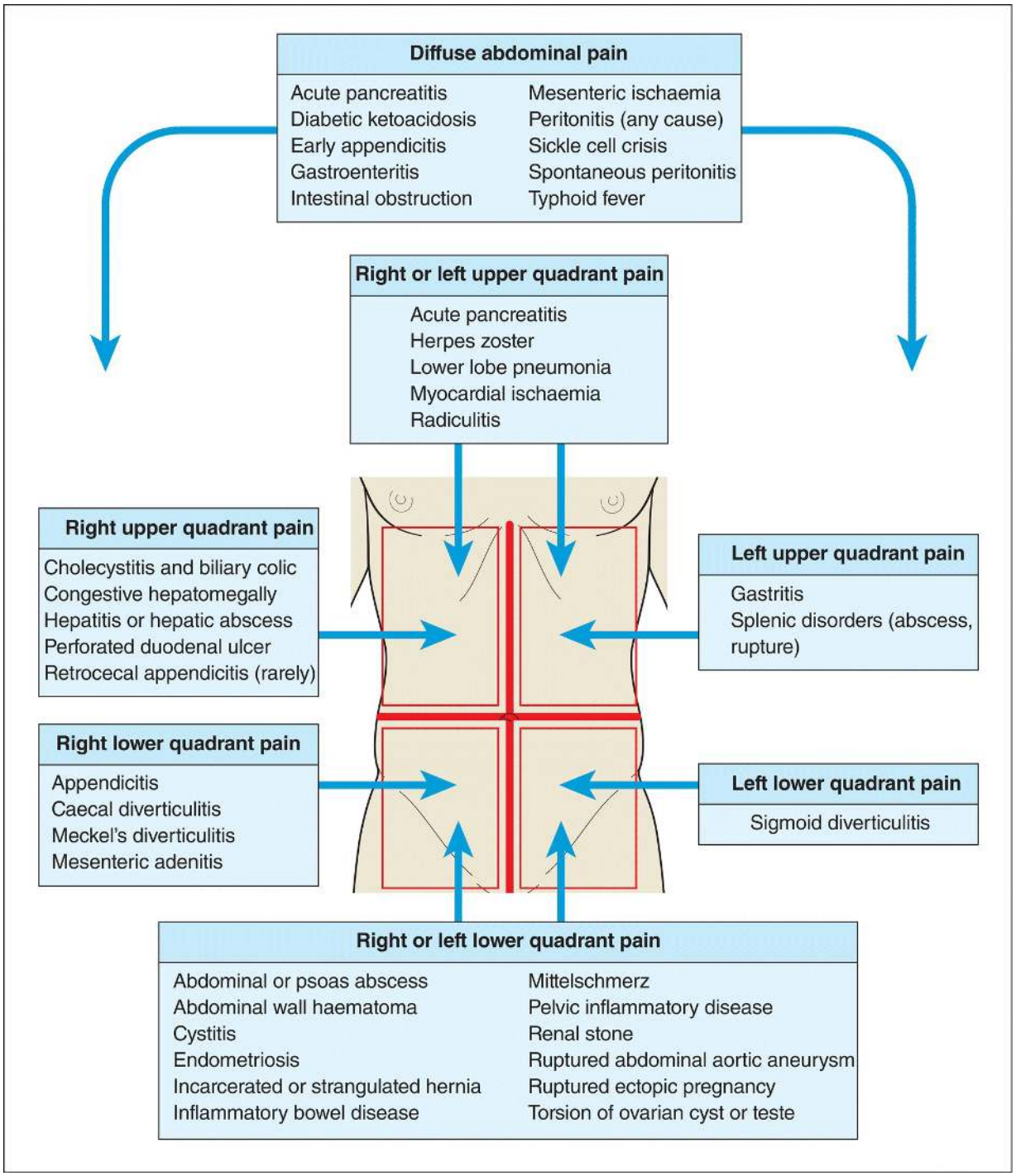


**Figure 1.2** Likely pathologies according to location of acute pain.

Source: Frederick H. Millham, in Feldman M, Friedman L, Brandt L (eds) (2010) *Sleisenger and Fordtran's Gastrointestinal and Liver Disease*, 9th edn, Philadelphia, PA: Saunders, Figure 10.3. Reproduced with permission of Elsevier.