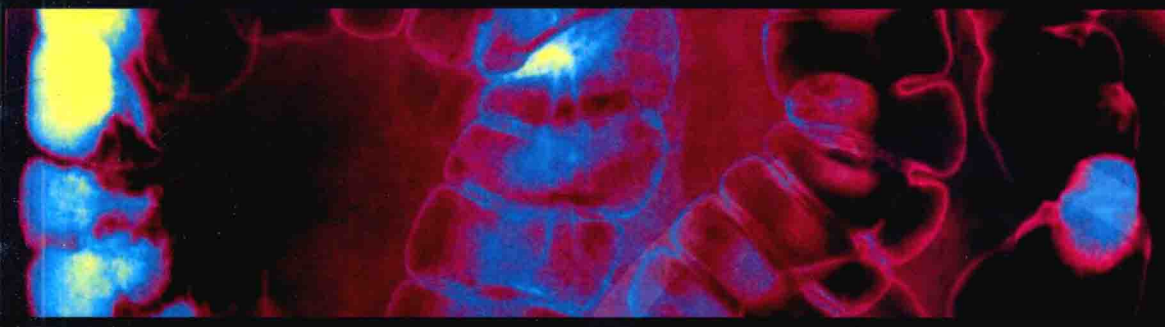
GASTROENTEROLOGY AND HEPATOLOGY

Lecture Notes



Anton Emmanuel
Stephen Inns

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Lecture Notes:
Gastroenterology and Hepatology

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Lecture Notes: **Gastroenterology and Hepatology**

Anton Emmanuel

BSc MD FRCP
Senior Lecturer in Neurogastroenterology
and Consultant Gastroenterologist
University College Hospital
London, UK

Stephen Inns

MBChB FRACP
Clinical Lecturer in Gastroenterology
Otago School of Medicine, Wellington
and Consultant Gastroenterologist
Hutt Valley Hospital
Wellington, New Zealand

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Preface

Science is the father of knowledge, but opinion breeds ignorance.

(Hippocrates 460–357 BC)

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 1809–1894)

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 upon 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
(a.emmanuel@ucl.ac.uk)
Stephen Inns
(stephen.inns@huttvalleydhb.org.nz)

Contents

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.

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 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 1.1). These are indicators that increase the likelihood that an organic condition underlies the pain. The alarm features guide further investigation.

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

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 are more likely to be of gradual 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 1.2).
- **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 pain of very longstanding duration is unlikely to be related to malignant pathology.

Location: radiation or referral (Figure 1.1)

- **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).

Box 1.1 Alarm features precluding a diagnosis of irritible 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

Box 1.2 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

- **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 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 (Figure 1.1).

One process can cause both features, the classical 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.

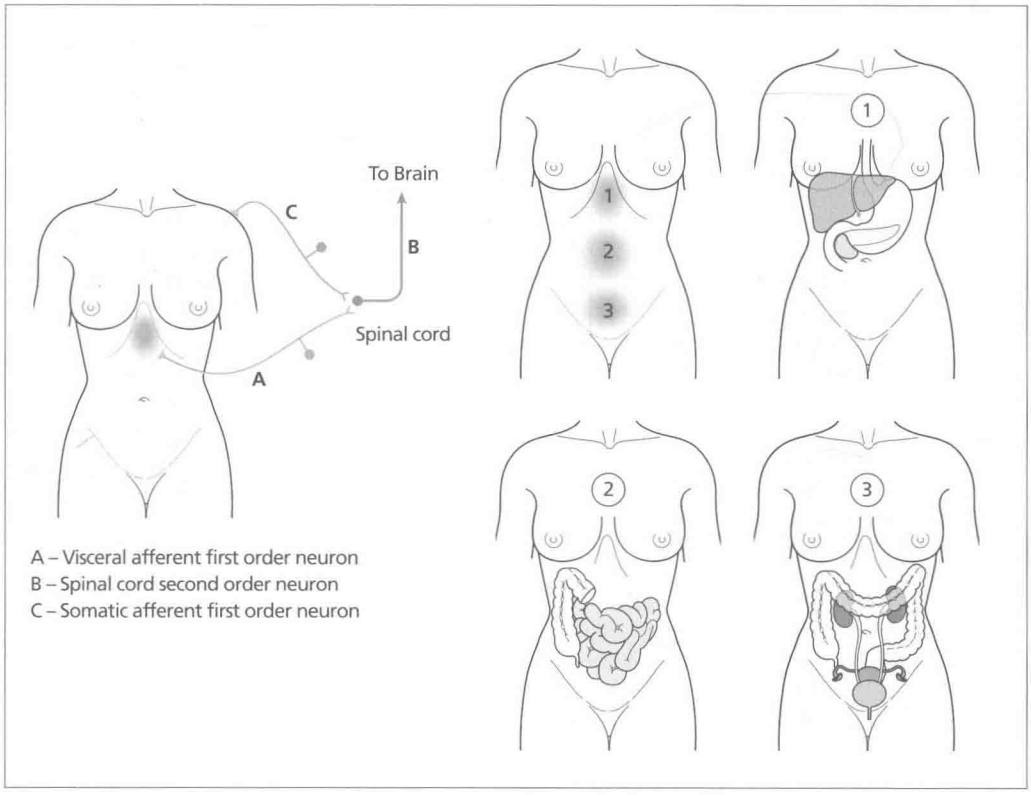

Figure 1.1 Location of pain in relation to organic pathology. A, Visceral afferent first-order neuron; B, spinal cord second-order neuron; C, somatic afferent first-order neuron.

- **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 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 is 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 yellow in jaundice, periorbital arcus senilis 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 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 GI system *per se* begins with careful inspection of the mouth with the aid of**

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 factor
Henoch–Schönlein purpura	Palpable purpura over buttocks and lower extremities	Arthralgias

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 first ask if 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. Whilst 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.

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 1.3). 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).

Box 1.3 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

Referred pain occurs due to 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 from a ruptured spleen or pancreatitis.

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) 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 FBC; urea, creatinine and electrolytes; 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).

CLINICAL EXAMPLE

Clinical Picture 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 is 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 mucous. 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 min to some hours at a time. There is no position in which she can get comfortable with the pain 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.

Observation reveals a thin woman with no hand or face signs of gastrointesintal 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, 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.

The most likely diagnoses in this setting are IBS and functional GI disease. 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, unlikely in a young woman, intestinal ischaemia. Less likely causes in other organ systems include biliary colic, ovarian pain and renal colic.

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 CT (or 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.

Likely organ involved	Likely pathology	Investigation choices	Investigation plan
Small bowel and colon	Inflammatory bowel disease	Ileocolonoscopy CT enterography US small bowel MRI	
	Irritable bowel syndrome	Suggestive symptom complex in the absence of other diagnoses	
	Infection	Stool culture and examination for C. difficile, ova, cysts and parasites Specific parasitic serology if peripheral eosinophilia	Stool test Ileocolonoscopy CT (or MRI) enterography
	Neoplasia	Ileocolonoscopy and small bowel follow-through	
	Ischaemia	Angiography	
Biliary system	Biliary stones, neoplasia	Ultrasound abdomen MRCP ERCP	
Ovary	Ovarian cyst, torted ovary	Ultrasound pelvis CT pelvis	
Renal	Renal stones	Ultrasound abdomen 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 presents 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 provides 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, and therefore warrant more expeditious evaluation and treatment.

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. 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.

Careful exclusion of past or chronic health problems that may have progressed to, or be associated with, the current condition is important. A patient with chronic dyspepsia may now be

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 'takes the breath away' (renal or biliary colic) Waves of dull pain with vomiting (intestinal obstruction) Colicky pain that becomes steady (appendicitis, strangulating intestinal obstruction, mesenteric ischaemia) Sharp, constant pain, worsened by movement (peritonitis) Tearing pain (dissecting aneurysm) 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) Less sudden: most other causes
Severity of the pain?	Severe pain (perforated viscus, kidney stone, peritonitis, pancreatitis) Pain out of proportion to physical findings (mesenteric ischaemia)
Radiation/referral?	Right scapula (gallbladder pain) Left shoulder region (ruptured spleen, pancreatitis) Pubis or vagina (renal pain) Back (ruptured aortic aneurysm)
Relieving factors?	Antacids (peptic ulcer disease) Lying as quietly as possible (peritonitis)
Associated symptoms?	Vomiting precedes pain and is followed by diarrhoea (gastroenteritis) Delayed vomiting, absent bowel movement and flatus (acute intestinal obstruction; the delay increases with a lower site of obstruction) Severe vomiting precedes intense epigastric, left chest or shoulder pain (emetic perforation of the intra-abdominal oesophagus)

presenting with perforation of a duodenal ulcer. The patient with severe peripheral vascular disease, or who has had recent vascular intervention, might have acute mesenteric ischaemia. A binge drinker with past episodes of alcohol-related pain is at risk for acute pancreatitis, as is the patient with known cholelithiasis. Patients with past multiple abdominal surgeries are at risk for intestinal obstruction.

Questioning regarding current and past prescribed, illicit and complementary medicine use is necessary. The patient using NSAIDs is at risk of peptic ulceration; use of anticoagulants increases the risk of haemorrhagic conditions; prednisone or immunosuppressants may blunt the inflammatory response to perforation or peritonitis resulting in less pain than expected.

Examination

Initial assessment is aimed at determining the seriousness of the illness. A happy, comfortable-appearing patient rarely has a serious problem, unlike one who is anxious, pale, sweaty or in obvious pain. Vital signs, state of consciousness and other signs of peripheral perfusion must be evaluated.

- **Examination of the non-abdominal organ systems** is aimed at determining any evidence for an extra-abdominal cause for the pain:
  - Abdominal wall tenderness and swelling with rectus muscle haematoma . Extremely tender, sometimes red and swollen scrotum with testicular torsion;
  - Resolving (sometimes completely resolved) rash in post-herpetic pain;
  - Ketones on the breath in diabetic ketoacidosis;
  - Pulmonary findings in pneumonia and pleuritis.
- **Examination of the abdomen** focuses on the detection of peritonitis, any intra-abdominal masses or organomegaly, and localisation of the underlying pathology:
  - Distension of the abdomen may be associated with intestinal obstruction;

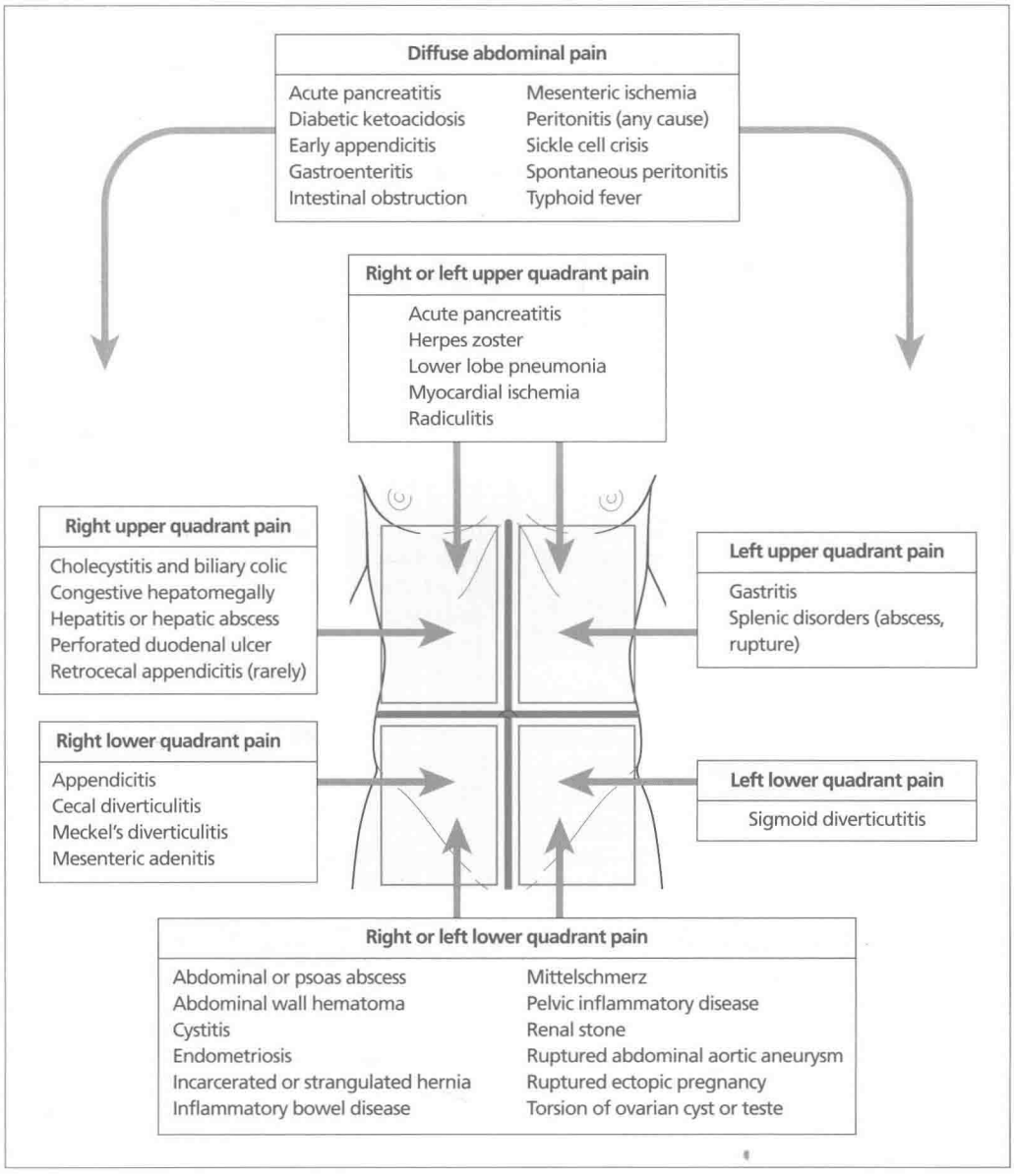

Figure 1.2 Likely pathologies according to location of acute pain.

- Bruising at the flanks (Grey Turner's sign) and periumbilically (Cullen's sign) is occasionally seen in acute haemorrhagic pancreatitis;
- Absent bowel sounds is indicative of ileus and in the presence of severe pain suggests peritonitis;
- High-pitched or over-active bowel sounds might indicate intestinal obstruction.

- **Palpation** should start with very light examination well away from the area of greatest pain. Guarding, rigidity and rebound indicate peritoneal irritation. Guarding is a slow and sustained involuntary contraction of the abdominal muscles, rather than the flinching that is observed with sensitive or anxious patients. Careful exclusion of hernias at the inguinal canals and over surgical scars,

as well as pelvic and rectal examination, is essential.

Investigations

Most patients will have a FBC, urea, creatinine and electrolytes, and dipstick urinalysis performed, although the results from these tests are neither sensitive nor specific. Serum lipase, however, is useful in detecting acute pancreatitis. It is essential that erect chest and abdomen, and supine abdominal X-rays are performed when there is the possibility of intestinal perforation or obstruction. If the patient cannot sit up, the left lateral position may be used.

Modern imaging can detect the underlying pathology in acute abdominal pain with high sensitivity and specificity. While ultrasound examination has the benefits of portability and avoidance of radiation exposure, it is most useful in detecting disease of the gallbladder, and gynaecological and obstetric conditions. CT has emerged as the dominant imaging tool for evaluation of the patient with severe acute abdomen. This has come about with the frequent advent of easy access to helical CT within or adjacent to the emergency department. The proper execution and interpretation of CT in this setting has been shown to reduce the need for exploratory laparotomy and hence morbidity, mortality and medical expense.

2

Approach to the patient with liver disease

Patients with liver disease can present with a wide range of complaints, and the clinician must remain alert at all times to the possibility of hepatic involvement in disease. Increasingly commonly, asymptomatic patients will present because of liver test abnormalities discovered incidentally. Once the presence of hepatic dysfunction has been established, the not always straightforward task of defining the underlying pathology is critical to planning appropriate management.

History taking

Liver disease can present in a variety of ways:

- **Non-specific symptoms** include fatigue, anorexia, nausea and, occasionally, vomiting;
- **Loose, fatty stools (steatorrhoea)** can occur if cholestasis interrupts bile flow to the small intestine;
- **Fever (due to liver pyrogens)** may be the first feature in viral or alcoholic hepatitis;
- **Jaundice** becomes visible when the serum bilirubin reaches 34–43 μmol/l (2–2.5 mg/dl). While jaundice may be related to hepatic dysfunction, equally it can be a result of bilirubin overproduction. Mild jaundice without dark urine suggests unconjugated hyperbilirubinaemia (most often caused by haemolysis or Gilbert's syndrome).

The historical features that it is important to elicit include the following.

Onset and duration

- **Did the symptoms come on gradually or suddenly? How long have the symptoms been a problem?** Symptoms of acute onset may result from an acute vascular event, toxic cause, obstruction of the biliary system or acute infection. Symptoms resulting from chronic inflammatory processes are more likely to be of gradual onset. The development of dark urine (bilirubinuria) due to increased serum bilirubin, from hepatocellular or cholestatic causes, often precedes the onset of visible jaundice.
- **Identify precipitating events** related to the onset of the symptoms; direct questions often need to be asked regarding exposure to common causes (Box 2.1), in particular:
  - Any association with pain that might relate to biliary obstruction?
  - Any use of medicines – prescribed, complementary or illicit?
  - Any trauma or major stress including surgery?
  - Any association with starvation (important in Gilbert's syndrome; see Chapter 20)?
  - Any history of marked weight loss or gain?
  - Any association with vascular events or hypotension?
  - Any possible infectious contact or exposure?

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