# PART 11 | Abdominal

CHAPTER 65

# The peritoneum, mesentery, greater omentum and retroperitoneal space

## Learning objectives

To understand:

- The development and anatomy of the mesentery and peritoneum
- Surgical conditions of the peritoneum, mesentery, greater omentum and retroperitoneal space

## DEVELOPMENT OF THE MESENTERY AND PERITONEUM

Early in development, the human abdomen comprises a wall and enclosed space (coelom). A partition (mesentery) subdivides the cavity into left- and right-sided spaces (***Figure 65.1***). The mesentery comprises a cell body (mesodermal mesentery) lined on either side by a sheet of mesothelial cells (***Figure 65.2***). At first the cell body is continuous with the posterior abdominal wall, and the mesothelial covering of the mesentery is continuous with that lining the inner surface of the posterior abdominal wall. Soon after this arrangement has developed, the cell body of the mesentery separates from the posterior abdominal wall but remains in contact with it (***65.1***). The surface mesothelium of the mesentery remains in continuity with that of the posterior abdominal wall (***Figure 65.2***).

The mesentery remains continuous throughout development, following birth and into adult life (***65.2***). Early during development, the mid-region of the mesentery forms a fold that subdivides the mesentery into upper (pre-fold), mid- (fold) and lower (post-fold) regions (***Figure 65.1***). The upper region develops as a sack overlapping the mid-region. As it develops, the spleen, stomach and liver emerge.

The mid-region fold has left and right sides (on either side of the midline). The mid-region nearest the posterior abdominal wall is termed the central zone (***Figure 65.1***). The remainder of the fold is termed the peripheral zone. Soon after the mid-region fold first emerges, the sides of the periphery (but not of the central zone) switch position relative to the superior mesenteric artery (SMA). After the switch, the original right side of the mid-region commences on the right side centrally but continues peripherally on the left of the SMA. The original left side commences peripherally on the right side of the vessel then returns centrally on the left of the SMA (***Figure 65.1***). Failure of switch formation occurs in malrotation (see ***Rotational disorders***).

During development of the upper region of the mesentery, the region of the mesodermal mesentery that is nearest the posterior abdominal wall adheres to the wall (***65.1***). This process progresses from the midline laterally to the left. Adhesion of both displaces the mesothelial junction between the mesentery and the adjacent posterior abdominal wall (***Figure 65.2***). At completion of development the posterior wall of the upper region (the dorsal mesogastrium) is fully adherent to the abdominal wall of the left upper quadrant (LUQ). The spleen is located at the lateral part of the upper region sac, in the LUQ. The original mesothelial junction remains as the peritoneal reflection linking the surface lining the mesenteric domain (in this case the spleen) with the surface lining of the adjacent non-mesenteric domain (in this case the LUQ).

During development of the mid-region fold, the region of the mesodermal mesentery that is nearest the posterior abdominal wall (i.e. the central zone) progressively adheres to the posterior abdominal wall. With continued adhesion, the periphery of the mid-region fold also adheres (***65.2 and 65.3***). Adhesion occurs inferolaterally to the right, and from the central to peripheral zones. The mesothelial junction between the mid-region mesentery and abdominal wall is displaced in tandem, inferolaterally to the right. At completion of development the mid-region fold is adherent to the posterior abdominal wall at the right mesocolon. The original mesothelial junction is at the periphery of the fold and persists as a peritoneal reflection bridging the surface lining of the mesenteric and non-mesenteric domains (***Figure 65.2***) (***65.3***). Incomplete adhesion of the mid-region fold is associated with increased mobility of the ileocaecal region and volvulus (see ***Volvulus of the intestine and adjoining mesentery***).

During development of the lower region of the mesentery, the part nearest the posterior abdominal wall progressively adheres to the wall (***65.1***). This proceeds from the midline laterally to the left. The overlying mesothelial junction is displaced in tandem (***Figure 65.2***). At completion of development, the lower region has fully adhered at the left mesocolon, medial mesosigmoidal and mesorectal levels. The lateral part of the mesosigmoid remains mobile. The mesothelial junction remains as a peritoneal reflection bridging the surface lining of the mesenteric and adjacent non-mesenteric domain

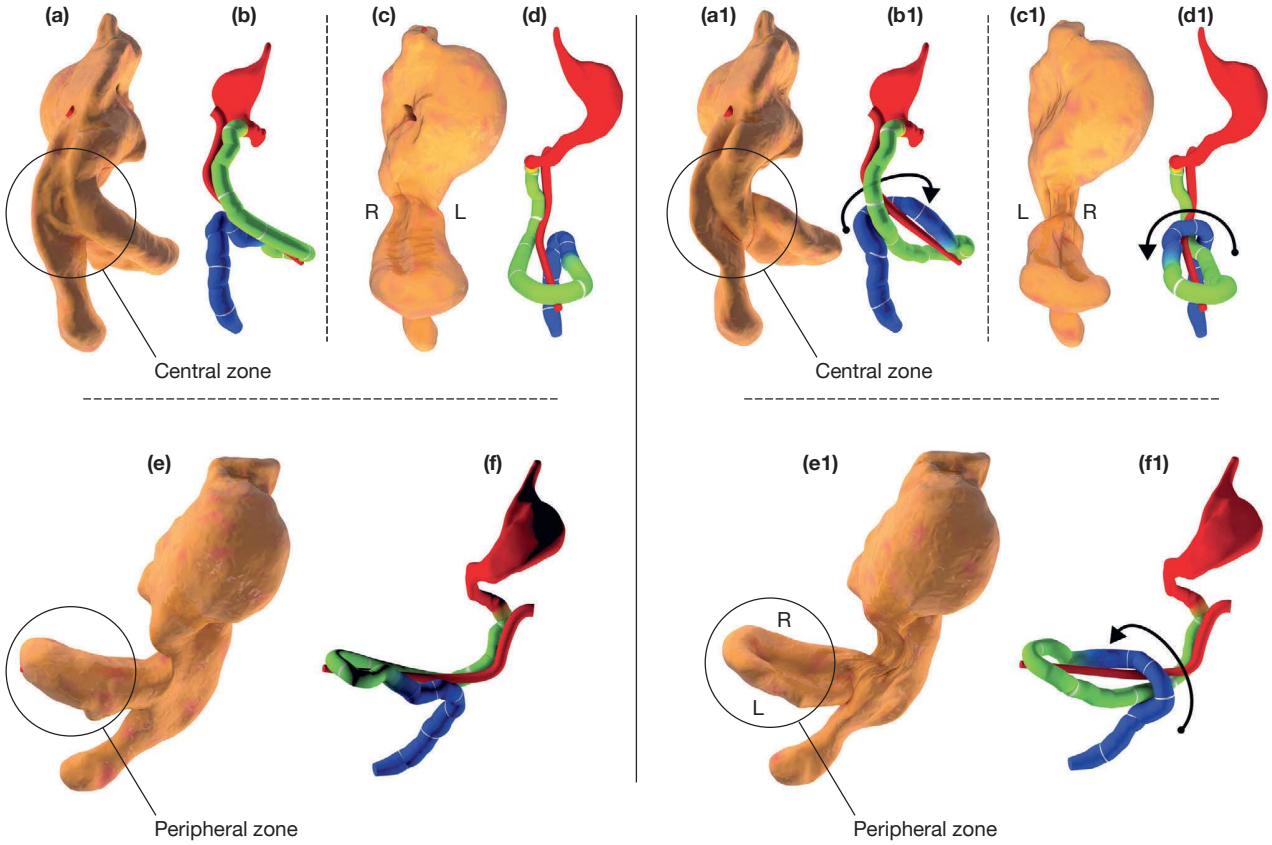


**Figure 65.1** Switching of the mid-region of the mesentery during development. **(a–f)** Digital reconstructions of the developing mesentery prior to switching of the sides of the mid-region fold. **(a, c, e)** Three-dimensional reconstructions of the mesentery before switching. The intestine develops within the mesentery. **(b, d, f)** Three-dimensional reconstructions of the intestine. The mesentery has been removed to expose the developing intestine. **(a1, c1, e1)** Digital reconstructions of the developing mesentery after switching of the sides of the mid-region fold. The mesentery is coloured yellow. **(b1, d1, f1)** Three-dimensional reconstructions of the intestine after the mid-region switch. The mesentery has been removed to expose the developing intestine. Yellow, the mesentery; red, upper region of intestine; green, developing mid-region; blue, lower region of the intestine. The superior mesenteric artery (blind-ending red tube) has been included for reference.

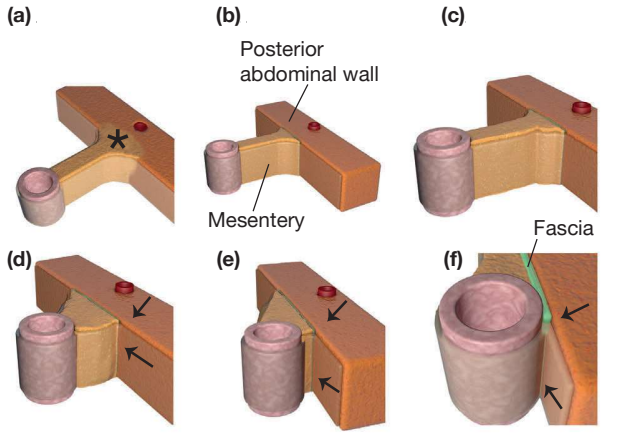


**Figure 65.2 (a-f)** Adhesion and displacement. Development of the junction between the mesentery (*) and the posterior abdominal wall. The peritoneal reflection (thick arrows) is displaced towards the periphery during adhesion of the mesentery to the abdominal wall.

(▶ *65.3*). Incomplete adhesion (and hence anchorage) of the lower region is associated with sigmoid volvulus (see ***Volvulus of the intestine and adjoining mesentery***).

## ANATOMY OF THE MESENTERY AND PERITONEUM

All abdominal digestive organs develop *in* or *on* the mesentery and then remain directly connected to it (***Figure 65.3***). In the adult setting, these collectively comprise a discrete anatomical unit, the mesenteric domain (***Figure 65.4***). All genitourinary organs develop on and remain on the musculoskeletal mainframe of the abdomen. In the adult, these are collectively termed the non-mesenteric domain. Thus, the adult abdomen comprises two discrete anatomical compartments: the mesenteric and non-mesenteric domains (***Figure 65.5***) (▶ *65.2*).

When development is complete, the mesentery and conjoined digestive organs (intestine, pancreas, spleen and liver) have taken shape and adherence to the posterior abdominal wall is nearly complete. The dorsal mesogastrium, mesoduodenum, right and left mesocolon and mesorectum are

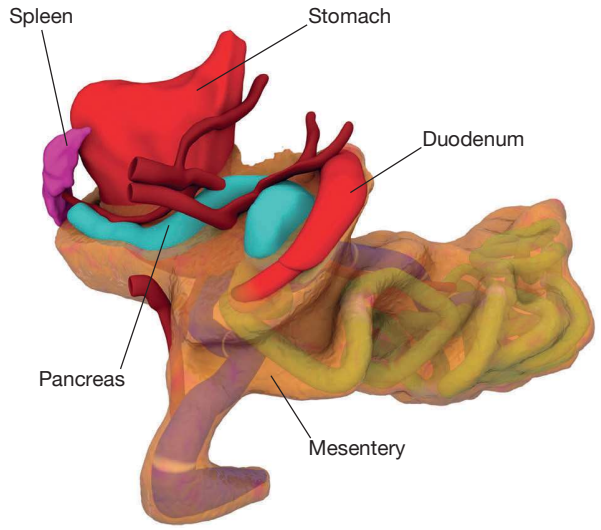


**Figure 65.3** The right lateral aspect of the mesentery during development. The mesentery has been sectioned to expose the developing stomach (red), pancreas (light blue) and major blood vessels.

anchored to the subjacent abdominal wall (or pelvic sidewall) (***Figure 65.4***). The small intestinal region of mesentery, transverse mesocolon and lateral mesosigmoidal mesentery are not adherent and thus are mobile.

Adhesion of the mesentery to the abdominal wall anchors the mesenteric to the non-mesenteric domain, maintained by peritoneal reflection at the periphery of the mesenteric domain (***65.1 and 65.3***). At the periphery, the peritoneal reflection bridges the surface lining of the mesenteric and non-mesenteric domains (***Figure 65.6***). The peritoneum thus comprises visceral peritoneum (corresponding to the surface lining of the mesenteric domain), parietal peritoneum (corresponding to the surface lining of the non-mesenteric domain) and the reflection joining both. Peritonitis refers to inflammation of any region (see ***Peritonitis***).

The surface contours generated by the organisation of the domains and the peritoneum explains the sacs, recesses, fossae and pouches in which abnormal fluid collections arise in the abdomen (***65.3***). On completion of development, the free (non-adherent) surface of each organ of the mesenteric domain is peritonealised. The opposing adherent surface is not peritonealised.

In the male, the peritoneal cavity is normally closed. In the female, the peritoneal cavity is open to the environment at the fimbrial entrance to the fallopian tubes. In both sexes (but more frequently in the male) a peritoneal tube (processus vaginalis) can persist at the deep inguinal ring and predispose to inguinal hernia formation.

The interface between adherent regions of the mesenteric and non-mesenteric domains is termed the retroperitoneal space (***65.4***). The retroperitoneal space normally contains connective tissue fascia. The space (and fascia) continues into the thorax superiorly and into the pelvis inferiorly. The retroperitoneum is deep to the retroperitoneal space. It includes the kidneys, ureters, gonadal vessels, lumbosacral plexus and the musculoskeletal frame of the posterior abdominal wall.

The arterial inflow to the mesenteric domain is limited to the coeliac trunk and superior and inferior mesenteric arteries. The venous drainage of the mesenteric domain occurs via the hepatic veins at the junction of these and the inferior vena cava. In between the arterial inflow and venous drainage, the vasculature of the abdominal digestive organs is entirely intramesenteric and aligned with the mesenteric regional anatomy (***65.5***). The limited routes of arterial inflow and venous drainage have significant implications when these are affected by pathology (discussed in ***Vascular abnormalities of the mesentery***).

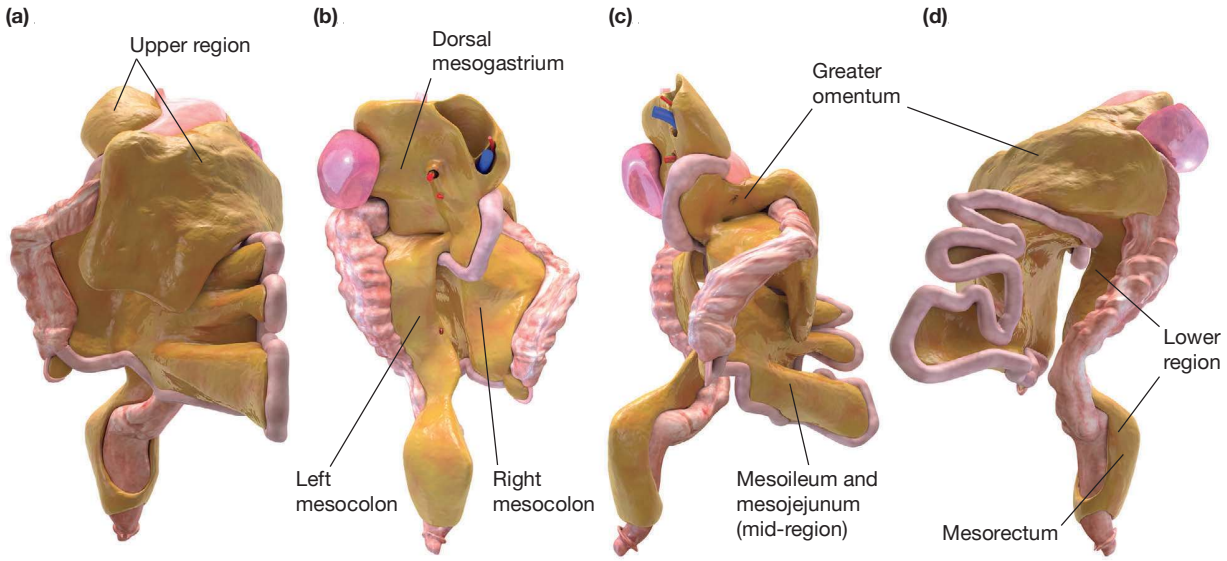


**Figure 65.4** The mesentery. **(a–d)** The adult mesentery (and mesenteric domain) as seen from anterior **(a)**, posterior **(b)**, right posterolateral **(c)** and left anterolateral **(d)** perspectives.

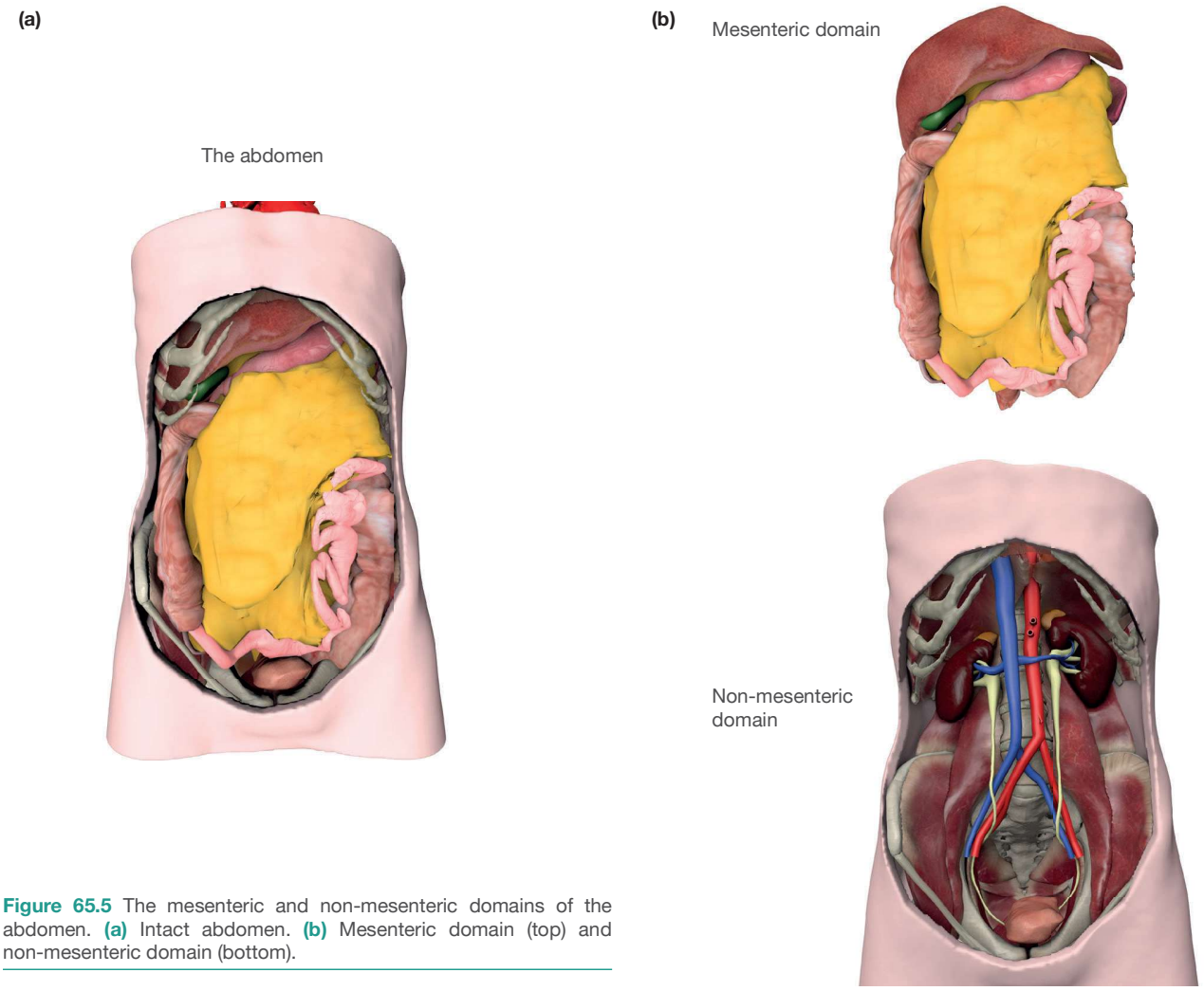


**Figure 65.5** The mesenteric and non-mesenteric domains of the abdomen. **(a)** Intact abdomen. **(b)** Mesenteric domain (top) and non-mesenteric domain (bottom).

A knowledge of the anatomical relationships between the mesenteric and non-mesenteric domains provides the student of abdominal surgery with a roadmap by which to perform safe and optimal abdominal surgery. The above description is termed the mesenteric model of abdominal anatomy. It is a model that matches observations during development with clinical observations *in vivo* and with radiological depictions of the abdomen. It provides the anatomical context on which surgical diseases of the abdomen and pelvis arise. Given this, it is rapidly substituting the peritoneal model in reference anatomical texts.

## THE PERITONEUM

The peritoneal cavity is the largest cavity in the body, the surface area of its lining membrane (2 $m^2$ in an adult) being nearly equal to that of the skin (*65.3*). The peritoneal membrane is composed of flattened polyhedral cells (mesothelium), one layer thick, resting on a thin layer of fibroelastic tissue. Beneath the peritoneum, supported by a small amount of areolar tissue, lies a network of lymphatic vessels and a rich plexus of capillary blood vessels from which all absorption and exudation must occur. In health, only a few millilitres of peritoneal fluid are found in the peritoneal cavity. The fluid is pale yellow, somewhat viscid and contains lymphocytes and other leukocytes; it lubricates the viscera, allowing easy movement and peristalsis. The parietal portion is richly innervated and, when irritated, causes severe pain that is accurately localised to the affected area. The visceral peritoneum, in contrast, is poorly innervated and irritation causes pain that is usually poorly localised to the midline.

### Summary box 65.1

#### Functions of the peritoneum

**In health**

- Visceral lubrication
- Fluid and particulate absorption

**In disease**

- Pain perception (mainly parietal)
- Inflammatory and immune responses
- Fibrinolytic activity

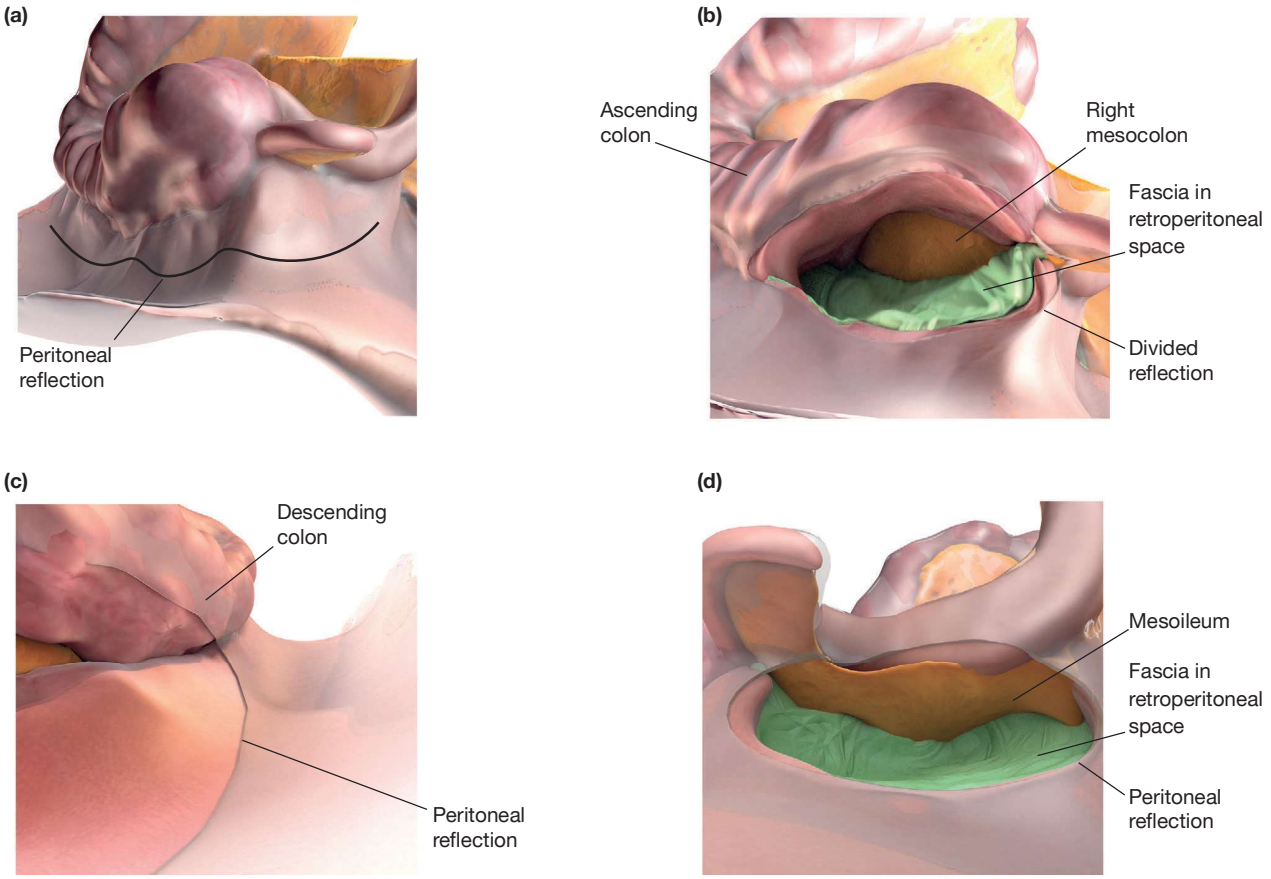


**Figure 65.6** The reflection at the periphery of the mesenteric domain, i.e. the junction between the mesenteric and non-mesenteric domains. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles.* Boca Raton: CRC Press, 2017.)

The peritoneum has the capacity to absorb large volumes of fluid; however, the peritoneum can also produce large volumes of fluid (ascites) and an inflammatory exudate when injured (seen in peritonitis). During expiration, intra-abdominal pressure is reduced and peritoneal fluid, aided by capillary attraction, travels in an upward direction towards the diaphragm. Particulate matter and bacteria are absorbed within a few minutes into the lymphatic network through a number of 'pores' in the diaphragmatic peritoneum. The circulation of peritoneal fluids may be responsible for the occurrence of abscesses anatomically remote from primary disease. The two sites most prone to collection are the pelvis and subdiaphragmatic areas, reflecting the effects of gravity while standing and lying, respectively.

## PERITONITIS

Peritonitis is inflammation of the peritoneum and can be categorised as localised or diffuse, acute or chronic or according to the primary underlying pathology. In the clinical setting, the most useful categorisation of peritonitis is based on whether it is localised or diffuse.

### Summary box 65.2

#### Causes of peritoneal inflammation

- Bacterial, gastrointestinal and non-gastrointestinal
- Chemical, e.g. bile, barium
- Allergic, e.g. starch peritonitis
- Traumatic, e.g. operative handling
- Ischaemia, e.g. strangulated bowel, vascular occlusion
- Miscellaneous, e.g. familial Mediterranean fever

### Summary box 65.3

#### Paths to peritoneal infection

- Gastrointestinal perforation, e.g. perforated ulcer, appendix, diverticulum
- Transmural translocation (no perforation), e.g. pancreatitis, ischaemic bowel, primary bacterial peritonitis
- Exogenous contamination, e.g. drains, open surgery, trauma, peritoneal dialysis
- Female genital tract infection, e.g. pelvic inflammatory disease
- Haematogenous spread (rare), e.g. septicaemia

## Localised peritonitis

This is where a localised area of the peritoneum has become inflamed. If the parietal peritoneum is involved, the patient complains of pain (somatic pain) in the area affected. Vital signs may be normal, but tachycardia and pyrexia are common. The characteristic signs are involuntary guarding (reflex abdominal wall contraction to reduce further peritoneal irritation) and rebound tenderness (worsening of pain on lifting the examining hand off the abdominal wall). Collectively these signs and symptoms are termed *peritonism* and the patient is described as *peritonitic* (see ***Chapter 63***).

If inflammation arises under the diaphragm, shoulder tip ('phrenic') pain may be felt. This is referred pain to the C5 dermatome. In cases of pelvic peritonitis, e.g. from an inflamed appendix or salpingitis, abdominal signs may be limited; deep-seated tenderness may be detected by digital rectal or vaginal examination. Signs may be limited in obese patients or in patients on immunosuppressive medications.

The aim is to diagnose the underlying cause and guide treatment. Diagnosis of the underlying condition is made through a combination of history and physical examination, supplemented by laboratory and radiological investigations. Laboratory biomarkers will support a diagnosis of acute inflammation, but are rarely diagnostically specific. The investigation of choice is computed tomography (CT) scanning. Modalities such as ultrasound can be used but lack specificity except in the case of tubo-ovarian pathology (see ***Chapter 87***). Laparoscopy may be required if the above investigations are inconclusive.

The aims of treatment are to remove the underlying cause and to lavage or dilute residual contamination. At surgery the inflamed peritoneum appears reddened, thickened and has a velvety texture. Plaques of yellow/white fibrin may be apparent, causing loops of intestine (and mesentery) to adhere to themselves and to the parietes. There is a reactionary, serous exudate (rich in leukocytes and plasma proteins) that gradually becomes turbid in appearance. The fluid may transform to frank pus if not evacuated.

## Diffuse (generalised) peritonitis

This normally signifies the occurrence of a life-threatening pathology. It means that regions (not just focal areas) of the parietes (parietal peritoneum) are inflamed. It normally arises as a result of pressure-related perforation of a viscus (e.g. in the setting of an obstructed colon), when large volumes of blood abruptly enter the peritoneal cavity (ruptured aortic aneurysm) or when substantial volumes pour incessantly (albeit not under pressure) into the peritoneal cavity (e.g. perforated duodenal ulcer or anastomotic leak).

The patient may describe acute or gradual onset abdominal pain of considerable intensity. The pain may be localised at first and then become diffuse. The patient is gravely ill looking (Hippocratic facies) and usually lies as still as possible to minimise fluid movement within the peritoneal cavity. The entirety of the abdominal musculature undergoes a reflex contraction and feels board-like on palpation ('board-like' rigidity). In a thin patient, contraction of the rectus abdominis muscles may be reflected in a scaphoid appearance of the abdomen (see ***Chapter 63***). A generalised ileus occurs and the abdomen may become distended.

Vital signs are usually deranged. In advanced cases the patient is hypotensive, tachycardic and pyrexial. At first the patient may seem confused, drowsy and disoriented. If the underlying pathology is not corrected the patient will lose consciousness. Signs may be limited in obese patients or in patients on immunosuppressive medications.

Investigation and treatment must be undertaken expediently as the time available to salvage may be limited. Investigations aim to identify the underlying cause and to guide treatment. An erect chest radiograph can be useful in identifying subdiaphragmatic gas (***Figure 65.7***). If a patient is particularly unwell and a CT is not available, then a lateral decubitus radiograph serves the same purpose as an erect radiograph (provided the patient has been appropriately positioned for long enough for the gas to rise within the peritoneal cavity).

### Summary box 65.4

#### Clinical features of peritonitis

- Abdominal pain, worse on movement, coughing and deep respiration
- Constitutional upset: anorexia, malaise, fever, lassitude
- Gastrointestinal upset: nausea +/– vomiting
- Pyrexia (may be absent)
- Raised pulse rate
- Tenderness +/– guarding/rigidity/rebound of abdominal wall
- Pain/tenderness on rectal/vaginal examination (pelvic peritonitis)
- Absent or reduced bowel sounds
- 'Septic shock' (systemic inflammatory response syndrome [SIRS] and multiorgan dysfunction syndrome [MODS]) in later stages

### Summary box 65.5

#### Management of peritonitis

**General care of patient**

- Correction of fluid and electrolyte imbalance
- Insertion of nasogastric drainage tube and urinary catheter
- Broad-spectrum antibiotic therapy
- Analgesia
- Vital system support

**Surgical treatment of cause when appropriate**

- 'Source control' by removal or exclusion of the cause
- Peritoneal lavage +/– drainage

**Hippocrates of Kos**, Greek physician and surgeon, and by common consent 'the father of medicine', was born on the island of Kos, off Turkey, about 460 BCE and probably died in 375 BCE.

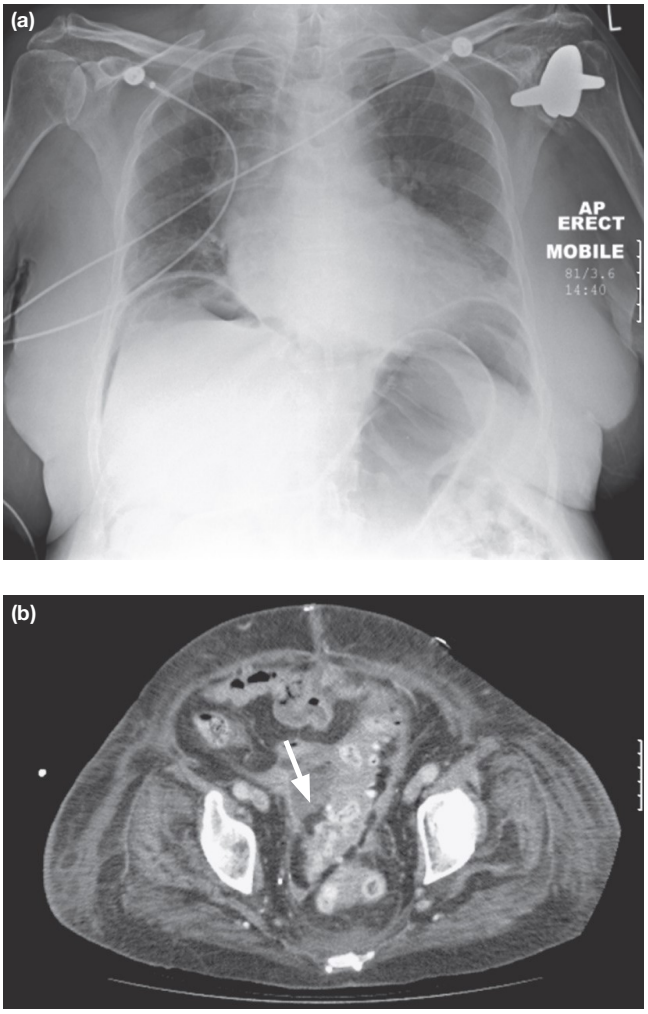


**Figure 65.7** Intraperitoneal perforation. **(a)** Erect chest radiograph demonstrating air under the diaphragm on the right side. **(b)** Axial computed tomography image showing a segment of sigmoid diverticulosis with localised perforation (arrow).

## Acute bacterial peritonitis

Acute bacterial peritonitis most commonly arises from perforation of a viscus of the alimentary tract. Other routes of infection can include the female genital tract and exogenous contamination. Less common forms involve a primary 'spontaneous' peritonitis due to streptococcal, pneumococcal or *Haemophilus* infection.

## Non-gastrointestinal causes of acute bacterial peritonitis

Pelvic infection via the Fallopian tubes is responsible for a high proportion of 'non-gastrointestinal' infections. The most common offending organisms are *Chlamydia* spp. and gonococci. These organisms lead to a thinning of cervical mucus and allow bacteria from the vagina to pass into the uterus and oviducts, causing infection and inflammation. A variant of transperitoneal spread of such organisms is perihepatitis, which can cause scar tissue to form on Glisson's capsule, a thin layer of connective tissue surrounding the liver (Fitz-Hugh–Curtis syndrome). Fungal peritonitis is rare but may complicate severely ill patients.

## Biliary peritonitis

Biliary peritonitis is mostly seen after cholecystectomy and arises from slippage of a clip off the cystic duct, drainage of bile from an accessory cystic duct or perforation of the common bile or hepatic duct (see ***Chapter 71***). It can also arise after hepatectomy or duodenal surgery, although this is unusual if a drain has been placed at the time of surgery.

Investigation follows the principles and steps described in ***Peritonitis***. The natural course of biliary peritonism varies depending on the volume of contamination. In severe contamination the patient will be extremely unwell and urgent intervention is required.

Localised collections can be treated by percutaneous insertion of a drain followed by endoscopic retrograde pancreatography (ERCP) to identify the source of bile leak. ERCP enables placement of a stent across the source of the leak. Diffuse or high-volume contamination, or the presence of multiple separate locules, normally mandates surgical exploration with the aim being lavage and drainage.

## Spontaneous bacterial peritonitis

Spontaneous bacterial peritonitis (SBP; sometimes called primary bacterial peritonitis) is an acute bacterial infection of ascitic fluid. There is often a history of cirrhosis and ascites. The clinical picture is highly variable as the patient may be asymptomatic. The course can be prolonged.

The diagnosis is made by paracentesis and should be considered in cirrhotic patients and those with ascites even when there is a low index of suspicion. The diagnosis is confirmed by finding an increased neutrophil count of 250/mm$^3$ in aspirated ascitic fluid. Culture of ascites is negative in as many as 60% of patients with clinical manifestations of SBP. When culture is positive the most common pathogens include Gram-negative bacteria, usually *Escherichia coli*, and Gram-positive cocci (mainly streptococci and enterococci).

Empirical treatment of SBP must be initiated immediately after diagnosis and before the results of culture have been received. Although the choice of antibiotic may vary, a third-generation cephalosporin, e.g. cefotaxime, is a reasonable first-line treatment that avoids the renal toxicity of aminoglycosides. Alternatives are amoxicillin/clavulanic acid and quinolones such as ciprofloxacin.

**Francis Glisson**, 1597–1677, Regius Professor of Medicine, Cambridge, UK.
**Fitz-Hugh–Curtis syndrome**: named after the two physicians, **Thomas Fitz-Hugh, Jr** 1894–1963, physician, University of Pennsylvania, Philadelphia, PA, USA, and **Arthur Hale Curtis** 1881–1955, gynecologist, Chicago, IL, USA, who first reported this condition in 1934 and 1930, respectively.

## Primary pneumococcal peritonitis

The incidence of pneumococcal peritonitis has declined greatly and the condition is now rare. It may complicate nephrotic syndrome or cirrhosis in children; however, otherwise healthy children may also be affected. In girls, the route of infection may be via the vagina and Fallopian tubes, while a blood-borne route secondary to respiratory tract or middle-ear disease is also possible.

The clinical onset is usually sudden, with pain usually localised to the lower half of the abdomen. The temperature is raised to 39°C or more and there is usually frequent vomiting. After 24–48 hours, profuse diarrhoea is characteristic. There is usually increased frequency of micturition. The last two symptoms are caused by severe pelvic peritonitis. On examination, peritonism is usually diffuse but less prominent than in cases of a perforated viscus, leading to peritonitis.

An underlying pathology must always be excluded before primary peritonitis can be diagnosed with certainty. Causative organisms include *Haemophilus* spp., group A streptococci and a few Gram-negative bacteria. Idiopathic streptococcal and staphylococcal peritonitis can also occur in adults.

After starting antibiotic therapy and correcting dehydration and electrolyte imbalance, early surgery is required unless spontaneous infection of pre-existing ascites is strongly suspected, in which case a diagnostic peritoneal tap is useful. Laparotomy or laparoscopy may be used. Assuming that no other cause for the peritonitis is discovered, some of the exudate is aspirated and sent to the laboratory for microscopy, culture and sensitivity tests. Thorough peritoneal lavage is carried out and the incision closed. Antibiotics and fluid replacement therapy are continued and recovery is usual.

## Tuberculous peritonitis

Intra-abdominal tuberculosis (TB) is common in resource-poor countries; however, the incidence is rising in resource-rich countries as a consequence of migration and immunosuppression. *Mycobacterium avium intracellulare* is becoming increasingly prevalent with the widespread increase in human immunodeficiency virus (HIV) co-infection. The abdomen is involved in approximately 11% of patients with extrapulmonary TB and includes intraperitoneal, gastrointestinal tract and solid organ disease forms. TB peritonitis requires specific mention because it is often diagnosed late, resulting in undue patient morbidity and mortality.

TB can spread to the peritoneum through the gastrointestinal tract (typically the ileocaecal region) via mesenteric lymph nodes or directly from the blood, usually from the 'miliary' (***Figure 65.8a***) but occasionally from the 'cavitating' form of pulmonary TB, lymph and the Fallopian tubes; 50–80% of patients with abdominal TB can be expected to have peritoneal involvement.

The most common form of TB peritonitis is the wet, ascitic-type disease (90%), which is characterised by generalised or loculated ascites. Multiple tubercle deposits are present on both layers of the peritoneum. In the less common form fibrotic fixed loops of bowel and omentum are matted together and may present with subacute intestinal obstruction. Ascites is not present in the dry, plastic type. Presentation is often insidious with abdominal pain, weight loss and abdominal distension. Distinction from diffuse peritoneal metastases is difficult and may require biopsy.

Diagnosis is via abdominal ultrasonography or CT to detect ascites and lymphadenopathy with/without diffuse thickening of the peritoneum, mesentery and/or omentum (***Figure 65.8b,c***). Ascitic fluid is typically a straw-coloured exudate (protein >25–30 g/L) with white cells >500/mL and lymphocytes >40%. Unfortunately, diagnostic smears for acid-fast bacilli are often not diagnostic and culture may take up to 4–8 weeks. Laparoscopy and peritoneal biopsy may thus be helpful to couple typical appearances with histology. The value of new laboratory investigations such as the Xpert® MTB/RIF assay and the interferon-gamma release assay in diagnosing extrapulmonary TB remains to be determined; however, measurement of adenosine deaminase activity in ascitic fluid has a high sensitivity and specificity in diagnosing peritoneal TB.

TB management is principally supportive (nutrition and hydration) and medical (systemic anti-TB therapy, noting that multidrug resistance may be higher for abdominal than for pulmonary TB), although surgery may be required for specific complications such as intestinal obstruction.

### Summary box 65.6

**Tuberculous peritonitis**

- Acute (may be clinically indistinguishable from acute bacterial peritonitis) and chronic forms
- Abdominal pain, sweats, malaise and weight loss are frequent
- Ascites common, may be loculated
- Caseating peritoneal nodules are common – distinguish from metastatic carcinoma and fat necrosis of pancreatitis
- Intestinal obstruction may respond to anti-TB treatment without surgery

## Familial Mediterranean fever

Familial Mediterranean fever (FMF; synonym familial paroxysmal polyserositis) is an autosomal recessive inherited autoinflammatory syndrome characterised by episodic diffuse abdominal pain and tenderness, mild pyrexia and joint pain. Symptoms are usually mild and resolve within 24–72 hours. Rarely pericardial or meningeal inflammation may occur. Amyloidosis is a long-term complication.

FMF is associated with mutations in the *MEFV* (Mediterranean fever) gene most frequently found in Arab, Armenian and Jewish populations. *MEFV* encodes the protein pyrin, which is expressed in neutrophils and is thought to regulate interleukin-1B (a proinflammatory cytokine) release.

Symptoms often present in childhood and may be misdiagnosed as appendicitis. Treatment of an acute episode is symptomatic. Colchicine can be used to reduce the frequency and severity of attacks and to prevent development of amyloidosis.

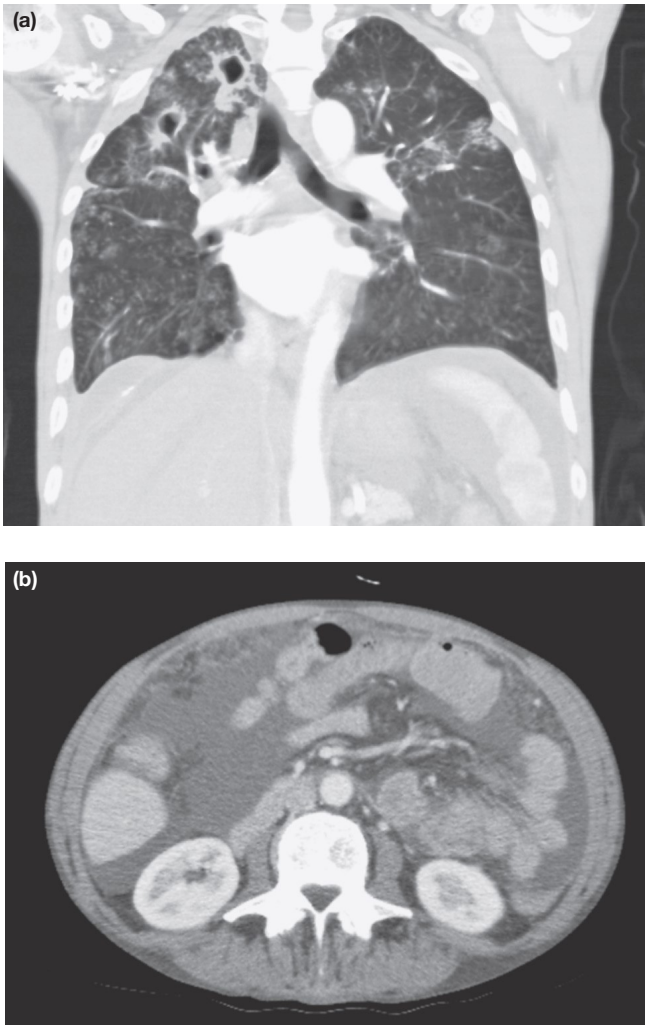


**Figure 65.8 (a)** Plain chest radiograph from a 55-year-old man showing miliary tuberculosis (TB); **(b, c)** representative computed tomography images from the same patient showing gross ascites, nodular stranding in the omentum and mesentery, as well as nodular enhancement of the peritoneum – TB peritonitis (courtesy of Dr S Burke, Homerton University Foundation Trust, London, UK).

# TUMOURS OF THE PERITONEUM

## Primary peritoneal malignancy

Primary tumours of the peritoneum are rare. They arise in the mesothelium of the peritoneum. Mesothelioma of the peritoneum is less frequent than in the pleural cavity but is equally lethal. Asbestos is a recognised cause. It has a predilection for the pelvic peritoneum. Cytoreductive surgery with heated intraperitoneal chemotherapy (HIPEC) or systemic cisplatin-based chemotherapy are the mainstays of treatment.

## Secondary peritoneal malignancy

### *Peritoneal carcinomatosis*

Peritoneal carcinomatosis is common and refers to malignant nodules on the surface of the peritoneum. It normally arises in conjunction with ovarian malignancy or malignancy in an organ of the mesenteric domain. It can be localised or diffuse. Any peritoneal surface can be involved. Sometimes the omentum is diffusely involved, forming a mass termed an omental cake.

The symptoms and signs are mainly related to the primary pathology. If the tumour burden is considerable, a mass may be palpable and the accompanying ascites substantial. Radiological cross-sectional imaging with CT or magnetic resonance imaging (MRI) is usually diagnostic; however, histological or cytological confirmation is essential to distinguish it from peritoneal TB. The visceral origin of peritoneal carcinomatosis is important as this can guide chemotherapy and cytoreductive or extirpative surgery.

The visceral origin of peritoneal carcinomatosis is important because if curative resection of the primary tumour is deemed feasible and the peritoneal disease considered resectable, resection with peritonectomy and HIPEC should be considered. HIPEC is a highly concentrated, heated (41–42°C) chemotherapy delivered directly into the abdomen for 90 minutes after cytoreductive surgery. HIPEC is particularly valuable in treatment of pseudomyxoma peritonei and has become the standard of care in carefully selected patients assessed in specialist centres (Sugerbaker) (see ***Chapter 76***).

**Paul H Sugerbaker**, b. 1941, Director of Surgical Oncology, Washington Cancer Institute, Washington, DC, USA.

In the majority of patients with peritoneal carcinomatosis treatment is palliative. Subacute intestinal obstruction may require intestinal bypass or a defunctioning stoma (see *Chapter 78*). Malignant ascites may be drained externally or via a peritoneovenous shunt (LeVeen).

# PERITONEAL INCLUSION CYSTS

## Introduction

These are benign cysts lined by peritoneal mesothelium. They can arise at any location of the peritoneum, in continuity with the surface of either parietal or visceral peritoneum. They can gradually expand and rarely rupture. Most are asymptomatic and are identified incidentally on cross-sectional imaging for other indications. There is a loose association with ovarian malignancy and thus MRI evaluation of the ovaries is advisable in the premenopausal context. If symptomatic an inclusion cyst may be drained under imaging control, deroofed or excised. Recurrent rates are high.

## Abdominal fluid collections

Abdominal collections are subdivided into intraperitoneal and retroperitoneal collections. Retroperitoneal collections are further subdivided into those limited to the retroperitoneal space (e.g. in pancreatitis) and collections arising in relation to retroperitoneal organs such the kidney. The latter are collections of the retroperitoneum. Collections of the retroperitoneal space and retroperitoneum are considered in the final section of the chapter.

## Intraperitoneal collections

### *Ascites*

Peritoneal fluid is constantly secreted and absorbed. Accumulation of peritoneal fluid, termed ascites, occurs when there is excess production or reduced absorption. Production of large volumes of a protein-rich fluid occurs in peritonitis and carcinomatosis peritonei. Reduced absorption occurs when capillary pressure is increased as a result of generalised water retention, cardiac failure, constrictive pericarditis or vena cava obstruction. Capillary pressure is also raised selectively in the portal venous system in the Budd–Chiari syndrome, hepatic cirrhosis or extrahepatic portal venous obstruction. Plasma colloid osmotic pressure may be lowered in patients with reduced nutritional intake, diminished intestinal absorption, abnormal protein losses or defective protein synthesis, such as occurs in cirrhosis. Peritoneal lymphatic drainage may be impaired, resulting in the accumulation of protein-rich fluid.

Hepatic cirrhosis is the most common cause of ascites due to portal venous hypertension secondary to fibrosis of the intrahepatic venous bed. In the Budd–Chiari syndrome (see *Chapter 69*), thrombosis of hepatic veins leads to obstruction of venous outflow from the liver and hence from the mesenteric domain in general. Alternative routes of venous drainage may open up. One such route involves the vestigial umbilical vein at the base of the falciform ligament. Venous drainage via this route may reach the systemic venous drainage at the umbilicus. This is termed a portosystemic shunt and has a characteristic clinical appearance (involving veins) at the umbilicus (caput medusae).

Congestive heart failure increases pressure in the vena cava and resistance to the venous outflow from the liver. In this setting, ascitic fluid is light yellow and has a low specific gravity and low protein concentration (<25 g/L). In constrictive pericarditis there is a diminished capacity of the right heart. This leads to simultaneous peritoneal and pleural effusions due to engorgement of the venae cavae (Pick's disease).

Ascites occurring in peritoneal metastases is due to excessive exudation of fluid and lymphatic blockage. The fluid is dark yellow and frequently blood stained. The specific gravity and protein content (>25 g/L) are high. Rarely, ascites and pleural effusion are associated with solid fibromas of the ovary (Meigs' syndrome). These effusions disappear when the tumour is excised.

**Summary box 65.7**

**Causes of ascites**

**Transudates (protein <25 g/L)**

- Low plasma protein concentrations
  - Malnutrition
  - Nephrotic syndrome
  - Protein-losing enteropathy
- High central venous pressure
  - Congestive cardiac failure
- Portal hypertension
  - Portal vein thrombosis
  - Cirrhosis

**Exudates (protein >25 g/L)**

- Peritoneal malignancy
- Tuberculous peritonitis
- Budd–Chiari syndrome (hepatic vein occlusion or thrombosis)
- Pancreatic ascites
- Chylous ascites
- Meigs' syndrome

Ascites normally becomes clinically recognisable when greater than 1.5 litres of fluid is apparent (although greater volumes may be required in obese patients). The abdomen is distended evenly with fullness of the flanks, which are dull to percussion. Usually, shifting dullness is present but, when there is a very large accumulation of fluid, this sign is absent. In such cases, flicking the abdominal wall produces a characteristic

**Harry H LeVeen**, 1915–1997, Professor of Surgery, University of South Carolina, Columbia, SC, USA.
**George Budd**, 1808–1882, Professor of Medicine, King's College Hospital, London, UK.
**Hans Chiari**, 1851–1916, Professor of Pathological Anatomy, Strasbourg, Germany (Strasbourg was returned to France in 1918 at the end of the First World War).
**Caput Medusa (head of Medusa)**, in Greek mythology depicted as having venomous snakes instead of hair.
**Friedel Pick**, 1867–1926, physician, Prague, the former Czechoslovakia, described this disease in 1896.
**Joe Vincent Meigs**, 1892–1963, Professor of Gynecology, Harvard University Medical School, Boston, MA, USA.

fluid thrill on the other side of the abdomen. This is not a reliable clinical sign. In women, ascites must be differentiated from an enormous ovarian cyst.

### Investigations

The aims are identification of ascites and determination of the underlying cause. Liver function tests (LFTs), cardiac function, ultrasonography and/or CT scanning (***Figure 65.9***) may help diagnose aetiology, e.g. carcinomatosis or liver disease.

Ascitic aspiration or tap under imaging guidance helps minimise the risk of visceral injury. It can be both diagnostic and therapeutic. After the bladder has been emptied, puncture of the peritoneum is carried out under local anaesthetic using a moderately sized trocar and cannula. A peritoneal drain may be inserted at the time.

In cases where the effusion is caused by cardiac failure, fluid must be evacuated slowly. Fluid is sent for microscopy/cytology, culture, including mycobacteria (see ***Tuberculus peritonitis*** above), and analysis of protein content and amylase. Unless other measures are taken the fluid soon accumulates and repeated tappings remove valuable protein.

### Management

Management aims to address any reversible primary pathology (following which the ascites resolves) or symptom-based management of the ascites itself. If portal venous pressure is raised, it may be possible to lower it by treatment of the primary condition or by transjugular intrahepatic portosystemic shunt or transjugular intrahepatic portosystemic stent shunting (commonly abbreviated as TIPS or TIPSS).

Dietary sodium restriction to 200 mg/day may be helpful, but diuretics are usually required (combination of spironolactone and furosemide). For patients failing to respond to such measures, therapeutic needle paracentesis can be performed. Serial large volume paracentesis (4–6 L/day and up to 8 litres in one session) can be performed safely with colloid replacement and can be performed in patients with cirrhosis and deranged clotting. Guidelines recommend albumin replacement after paracentesis to reduce complications. It may also be possible to leave an indwelling external drain for smaller volume home paracentesis.

## Chylous ascites

In some patients, the ascitic fluid appears milky because of an excess of chylomicrons (triglycerides). Most of these cases are associated with malignancy (usually lymphoma). Other causes include cirrhosis, TB, filariasis, nephrotic syndrome, abdominal trauma (including surgery), constrictive pericarditis, sarcoidosis and congenital lymphatic abnormality. The prognosis is poor unless the underlying condition can be cured. In addition to other measures used to treat ascites, patients should be placed on a fat-free diet with medium-chain triglyceride supplements.

# INTRAPERITONEAL ABSCESS FORMATION

An intraperitoneal abscess is a collection of pus in the peritoneal cavity (***Figure 65.10***). It normally arises secondary to another pathology. Inflammation of any viscus, if unresolved, will lead to hypersecretion of peritoneal fluid. The nature of the fluid progresses to frank pus unless adsorbed or drained. Hence, abscess formation commonly accompanies inflammation of an abdominal viscus and is usually labelled either according to location (subphrenic, intrapelvic) or with reference to nearby organs (periappendiceal, paracolic, subhepatic).

Intraperitoneal abscess formation is associated with a spectrum of symptoms and signs. At one end, patients may be asymptomatic or may feel somewhat unwell, anorectic, fatigued, with failure to maintain or gain weight. At the opposite end, patients may have significant abdominal symptoms and signs (nausea, vomiting, abdominal pain, diarrhoea) and be extremely unwell. A swinging pyrexia is strongly suggestive of intraperitoneal abscess formation.

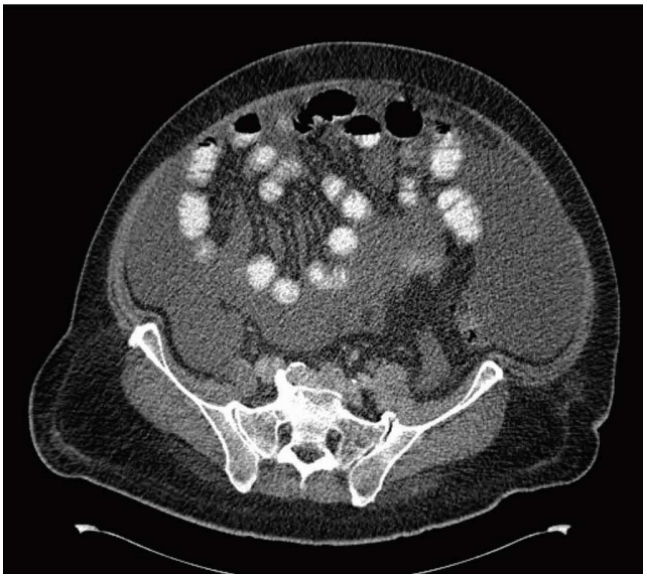

**Figure 65.9** Computed tomography axial scan of the abdomen showing gross ascites.

### Summary box 65.8

#### Clinical features of an abdominal/pelvic abscess

**Symptoms**

- Malaise, lethargy – failure to recover from surgery as expected
- Anorexia and weight loss
- Sweats +/– rigors
- Abdominal/pelvic pain
- Symptoms from local irritation, e.g. shoulder tip/hiccoughs (subphrenic), diarrhoea and mucus (pelvic), nausea and vomiting (any upper abdominal)

**Signs**

- Increased temperature and pulse +/– swinging pyrexia
- Localised abdominal tenderness +/– mass (including on pelvic examination)

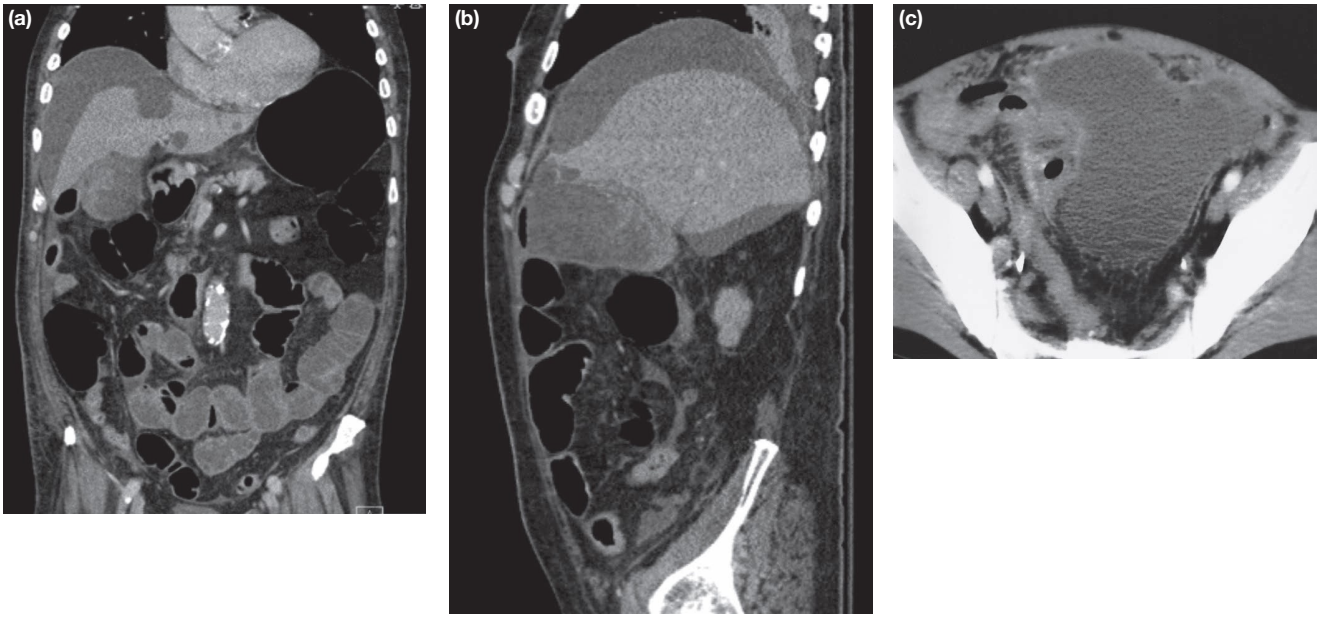


**Figure 65.10** Intraperitoneal abscesses. **(a)** and **(b)** Subphrenic and subhepatic abscesses seen on computed tomography (CT) scanning. **(c)** Pelvic abscess seen on CT scanning.

## Investigation

The modern diagnosis of an abscess is radiological using CT (***Figure 65.10***). CT imaging can also guide treatment by drain placement or aspiration. Ultrasound is a useful (though non-specific) modality for use in select populations (e.g. paediatric or pregnant patients). Serial imaging is used to monitor treatment efficacy or disease progression. Radiolabelled white cell scanning may occasionally prove helpful if an abscess is suspected but has not been identified by the above means.

## Treatment

Abscesses less than 5 cm in diameter normally resolve with intravenous antibiotic treatment. As antibiotics take effect, the magnitude of the swinging pyrexia can decrease with each successive spike in temperature. Serial monitoring of C-reactive protein levels is useful to non-invasively monitor response to treatment.

Abscesses greater than 5 cm require either percutaneous aspiration/drainage or surgical intervention. If percutaneous radiological approaches fail, then operative washout is indicated. This can be conducted laparoscopically (laparoscopic lavage) or via an open approach. The technical challenges involved are such that this should only be undertaken by an experienced surgeon. The bowel may be matted and difficult to separate in order to access the abscess. All regions of the peritoneal cavity should be accessed, with a view to drainage of any residual collections. The entirety of the small intestine and adjoining mesentery should be exposed to ensure that there are no residual interloop abscesses. If a phlegmon is apparent, then only in the setting of life-threatening circumstances should the components of this be separated.

## Special considerations: Prevention of abscess formation after appendicitis

During appendectomy, it is important to aspirate the pelvic, paracolic and subhepatic spaces prior to closure of the abdominal wall. This simple measure can reduce the incidence of postoperative abscess, which is common after appendectomy, occurring in up to 30% of patients following appendectomy for a perforated appendix. It can lead to frustration for patients if not forewarned of the possibility.

## Special considerations: Abscess formation following intestinal surgery and anastomosis

The development of an abscess following intestinal resection and anastomosis signifies infection of a haematoma or an anastomotic leak. Locules of gas or free contrast (Gastrografin) on CT support anastomotic leak. Pelvic abscess formation is not uncommon following excision of the rectum and formation of a pelvic anastomosis.

## Special considerations: Subphrenic abscess

This refers to the presence of pus immediately beneath the diaphragm. Patients may complain of shoulder tip pain. The diaphragm also develops at the same level as the C5 dermatome. If the parietal peritoneum under the diaphragm is irritated, pain is referred to the shoulder tip. This also explains why patients frequently complain of shoulder tip pain following laparoscopic or robotic surgery. In the era preceding that of cross-sectional imaging via CT, the adage ‘pus somewhere, pus nowhere, pus under the diaphragm’ was useful.

## Special considerations: Pelvic abscess

The pelvis is the most common site of abscess formation because the vermiform appendix is often pelvic in position and the Fallopian tubes are also frequent sites of infection. A pelvic abscess can also occur as a sequel to diffuse peritonitis and is common after anastomotic leakage following colorectal surgery.

### Clinical features

The most characteristic symptoms are pelvic pain, diarrhoea and passage of mucus in the stools. The patient may complain of lower back pain or a pressure sensation in the pelvis. This symptom can be quite severe in intensity. The abscess may discharge into the anal canal as the pelvic collection points through an anastomotic leak (the point of least resistance). Rectal or vaginal examination can be extremely uncomfortable for the patient.

### Investigation and management

If any uncertainty exists, the presence of pus should be confirmed by ultrasonography or CT scanning. Pelvic abscesses can be drained transanally or transgluteally. The past vogue for transintestinal drainage is no longer practised because of the high incidence of complications such as fistulae. Laparotomy may sometimes be indicated.

# PERITONEAL (MESOTHELIAL) SAC AND HERNIA FORMATION

The processus vaginalis refers to a peritoneal tube that advances into the inguinal ligament in tandem with migration of the testes. The lumen of the processus vaginalis is in continuity with the peritoneal cavity. Given this, it provides a conduit for herniation of abdominal contents. Even a residual indentation at the ostium of the processus vaginalis represents a mechanical defect at which repeated episodes of raised intraperitoneal pressure can lead to gradual extension of the parietal peritoneum into the inguinal canal.

The mesothelial sac is a near constant feature of incisional and parastomal hernias. In these instances, the peritoneum gradually advances over subcutaneous fat, or the serosal surface of the intestine, bringing that region of anatomy directly in continuity with the peritoneal cavity. Not surprisingly, incisional or parastomal hernias gradually increase in size with time. In addition, they are frequently complicated by parastomal herniation of intestinal contents and intestinal compromise.

# THE MESENTERY

## GENERAL CONSIDERATIONS

The arterial supply and venous and lymphatic drainage of each digestive organ are located in the mesentery. Thus diseases of individual organs can have significant effects on the adjoining mesentery and its components. The pancreas and the effects of pancreatitis are a good example. The pancreas is positioned on the mesentery (▶ *65.5*). It arches over the SMA and superior mesenteric vein (both of which are intramesenteric). The neck of the pancreas is anterior to the region of the mesentery that contains the portal vein. The body and tail of the pancreas are in the dorsal mesogastrium (i.e. the posterior wall of the upper region sack) and the tip of the tail of the pancreas is located at the hilum of the spleen (see ***Development of the mesentery and peritoneum***). Given these anatomical relations, acute inflammation of the pancreas can affect any of these structures. Complications of acute pancreatitis thus include thrombosis in the portal and splenic vein, gastric outlet obstruction and arterial haemorrhage (see ***Chapter 72***).

The mesentery is remarkably well preserved in most diseases. Although rarely encountered in clinical practice, mesenteric necrosis is mostly seen in advanced necrotising pancreatitis. Primary defects (i.e. non-surgical causes) of the mesentery are rare. These are always accompanied by failure of normal development of the adjoining organ. For example, intestinal atresia arises when a section of the adjoining mesentery fails to develop.

The mesentery comprises adipose, connective tissue, neurological, lymphatic and vascular components. Abnormalities can arise in any of these and lead to either solid (tumour deposits, lymphatic metastases) or cystic lesions. The supportive capacity of the mesentery is reflected in the finding of splenunculi, heterotopic pancreas, ossification, teratomas and even ectopic pregnancies in different regions of the mesentery.

## MESENTERIC HAEMATOMA

A mesenteric haematoma can follow abdominal compression in trauma (e.g. seat-belt syndrome) or during abdominal surgery, when the mesentery must be manipulated. The mesenteric stroma is mainly adipose and thus easily damaged. It bleeds readily if disrupted. A haematoma may form and quickly enlarge to compress mesenteric veins. Mesenteric haematoma can sometimes occur during surgery for Crohn's disease.

## MESENTERIC ADENITIS

This is inflammation of the lymph nodes of the mesentery (***Figure 65.11***). It mostly occurs in the ileocaecal region because of the volume of lymphatic tissue. It is often the site of viral or infective lymphadenopathy (*Yersinia* spp., *Campylobacter* spp., *Mycobacterium tuberculosis*) and may follow an upper respiratory tract infection with either a viral or bacterial pathogen. Mesenteric adenitis caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is not uncommon. Swelling of ileocaecal lymph nodes results in capsular stretch and somatic pain in the right iliac fossa. The patient is frequently pyretic (often the temperature is markedly elevated) and may have enlarged cervical lymph nodes.

During childhood, acute, non-specific mesenteric adenitis is a common condition. The typical history is one of short attacks of central abdominal pain lasting from 10 to 30 minutes, commonly associated with vomiting. The patient seldom looks ill. In more than half of the cases the temperature is

elevated. Abdominal tenderness is poorly localised and, when present, shifting tenderness is a valuable sign for differentiating the condition from appendicitis. The neck, axillae and groins should be palpated for enlarged lymph nodes.

### Investigation

There is often a leukocytosis of 10 000–12 000/μL (10–12 × $10^9$/L) or more on the first day of the attack, but this falls on the second day. Ultrasonography may be helpful in differentiating this from appendicitis. Sometimes a CT or exploratory laparoscopy is required.

### Treatment

This is normally supportive. Viral mesenteric adenitis normally resolves spontaneously but can recur. The symptoms in bacterial mesenteric adenitis include cramping pain, vomiting and diarrhoea. They can be severe and require hospitalisation.

## MESENTERIC ADENITIS AND THE MESENTERY IN CROHN'S DISEASE

Ileocaecal mesenteric adenitis occurs in ileocolic Crohn's disease and the mesentery is thickened, shortened and oedematous with a tendency to bleed readily when handled. The vascular pedicles within the mesentery may not be apparent, thus great care is needed when dividing the mesentery as normal techniques may not be suitable.

In Crohn's disease, the mesentery can extend over adjoining intestine as 'fat wrapping' or 'creeping fat' (***Figure 65.12***). These appear to be specific to Crohn's disease. The mesentery changes from normal to abnormal at the mesenteric transition zone. Conventional surgery for Crohn's disease involves amputation of the intestine at its intersection with adjoining mesentery. The mesentery is thus retained. Increasingly, surgeons are removing the mesentery adjoining the diseased intestine (see ***Chapter 75***).

## TUBERCULOSIS OF THE MESENTERIC LYMPH NODES

Tuberculous mesenteric lymphadenitis is considerably less common than acute non-specific lymphadenitis. Tubercle bacilli, usually, but not necessarily, bovine, are ingested and enter the mesenteric lymph nodes by way of Peyer's patches. Sometimes only one lymph node is infected; usually there are several, and occasionally massive involvement occurs. The presentation may be with abdominal pain (a rare differential for appendicitis) or with general constitutional symptoms (pyrexia, weight loss, etc.). Calcified lymph nodes may be demonstrated on a plain radiograph of the abdomen, where they must be distinguished from other calcified lesions, e.g. renal or ureteric stones.

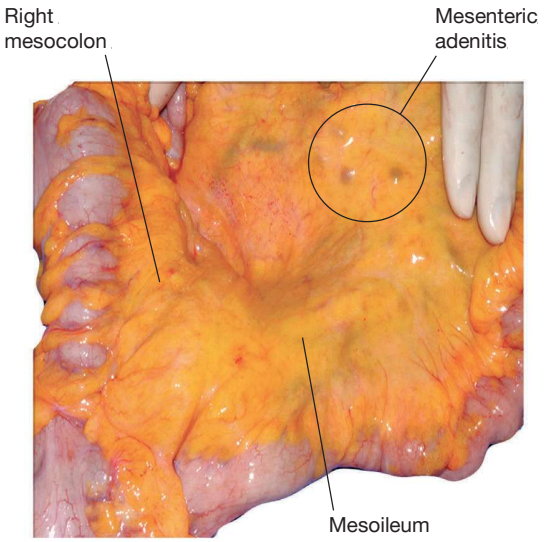


**Figure 65.11** Mesenteric adenitis. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles*. Boca Raton: CRC Press, 2017: 69–84.)

## VASCULAR ABNORMALITIES OF THE MESENTERY

### Acute mesenteric ischaemia

The arterial inflow to the mesenteric domain is limited to three major vessels: the coeliac trunk and the superior and inferior mesenteric arteries (***65.5***). Additional arterial inflow in the pelvis comes via the middle rectal arteries. The limited number of arterial inputs to the mesenteric domain mean that narrowing or occlusion at the origin of any one vessel can have significant clinical effects. Acute mesenteric ischaemia mostly follows embolisation to the origin of either the coeliac or superior mesenteric arterial trunk. Unless quickly reversed, it can lead to ischaemia and necrosis of most of the intestine. At first, the severity of abdominal pain does not match clinical findings on examination. If ischaemia and necrosis occur, the patient develops peritonism as a result of irritation of the parietal peritoneum by the necrotic intestine.

The inferior mesenteric artery (IMA) is usually divided at open repair of an abdominal aortic aneurysm; however, anastomoses between peripheral branches of the SMA and IMA, referred to as the marginal artery of Drummond, usually prevent critical ischaemia of the sigmoid and descending colon. If ischaemia is limited to the mucosa, the patient may experience cramping suprapubic pain and diarrhoea that normally settles. If ischaemia is transmural the colon may become necrotic and require resection.

**Johann Conrad Peyer**, 1653–1712, Professor of Logic, Rhetoric and Medicine, Schaffhausen, Switzerland, described the lymph follicles in the intestine in 1677.
**Sir David Drummond**, 1852–1932, born Dublin, Ireland, pathologist and physician at the Royal Victoria Infirmary, Newcastle (1878–1920), President of the British Medical Association (1921–1922) and vice chancellor of the University of Durham (1920–1922).

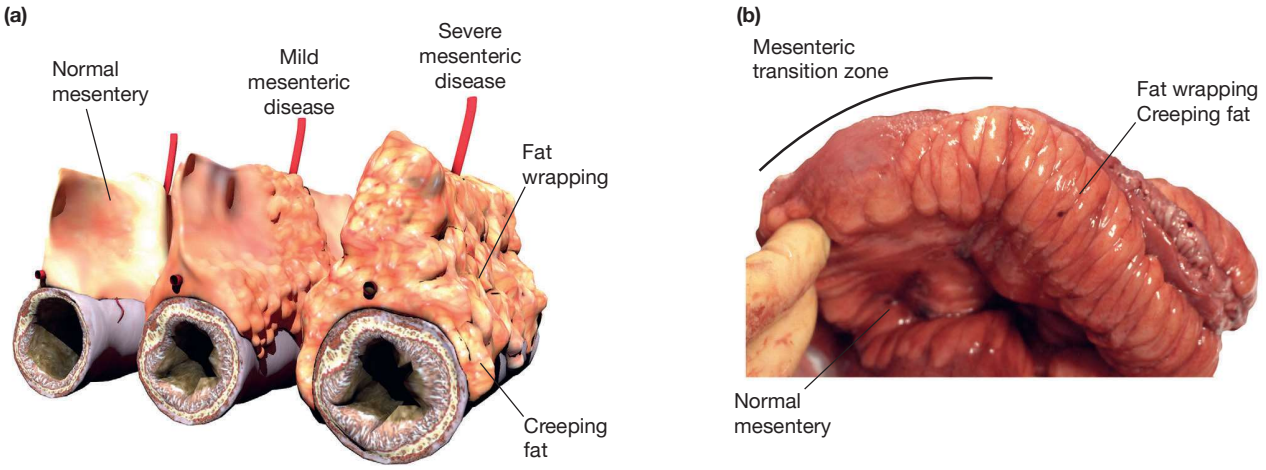


**Figure 65.12** The mesentery in Crohn's disease. **(a)** Levels of mesenteric disease manifestations. **(b)** The mesenteric transition zone and creeping fat. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles*. Boca Raton: CRC Press, 2017: 85–108.)

## Chronic mesenteric ischaemia

Chronic mesenteric ischaemia is due to atherosclerotic narrowing at the origin of any of the three arterial trunks. Patients describe postprandial abdominal pain that can be severe, resulting in a fear of eating with progressive weight loss. The diagnosis requires CT angiography. Radiological stent placement may be successful, but surgical endarterectomy of bypass repair may be required.

### *Venous ischaemia*

The venous drainage of the mesenteric domain is limited to the junction between the hepatic veins and the inferior vena cava. Narrowing or blockage of the lumen at this junction occurs in Budd–Chiari syndrome. It has major clinical implications. Most venous drainage of the mesenteric domain returns to the liver via the portal vein. Portal venous thrombosis impedes venous drainage of all abdominal digestive organs unless an alternative drainage route opens (i.e. a portosystemic shunt) or is created (see ***Chapter 69***) (***65.5***).

# ROTATIONAL DISORDERS

## Malrotation

Malrotation refers to a failure of formation of the mid-region switch (described in ***Development of the mesentery and peritoneum***) (***Figure 65.13***) and is the most common abdominal surgical emergency in the neonatal period. Early during development, the right and left side of the mid-region fold of the mesentery are aligned from the central to peripheral zones. Later, the sides switch position at the periphery but not at the central zone. Adjoining intestine similarly changes position to take up the normal conformation. In malrotation, the switch does not occur, and the sides of the mid-region fold remain aligned. This explains why the duodenum, jejunum and ileum are aligned in the right flank of the abdomen. Malrotation in itself is not pathogenic. However, the small intestine and adjoining mesentery are abnormally mobile and can undergo torsion around the superior mesentery artery, which can be life-threatening.

### *History*

The neonate with a volvulus due to malrotation is patently distressed, vomiting and has a distended abdomen.

### *Investigation*

Urgent CT is mandated and clarifies the position of the duodenojejunal junction. Normally the duodenojejunal flexure is positioned at or to the left of the midline. In volvulus due to malrotation, the duodenojejunal junction is on the right of the midline (i.e. the mid-region switch has not occurred).

### *Treatment*

The mainstay of treatment is Ladd's procedure, in which the volvulus is first reversed and then the intestine is secured to the posterior abdominal wall. Although this does not correct the underlying mesenteric abnormality, it does reduce the mobility of the intestine and mesentery and reduces the likelihood of further volvulus. It is possible however to address the underlying mesenteric abnormality by recapitulating the mid-region switch. This returns the intestine and mesentery to a normal conformation. They can then be fixed to the posterior abdominal wall.

## Volvulus of the intestine and adjoining mesentery

Volvulus can only be understood if considered in mesenteric terms. Where a section of intestine is curved, the adjoining mesentery is buckle-shaped or folded. If the intestine progresses from a curve to a coil, the adjoining mesentery acquires a

**William Edwards Ladd**, 1880–1967, Surgeon in Chief, Boston Children's Hospital, Boston, MA, USA.

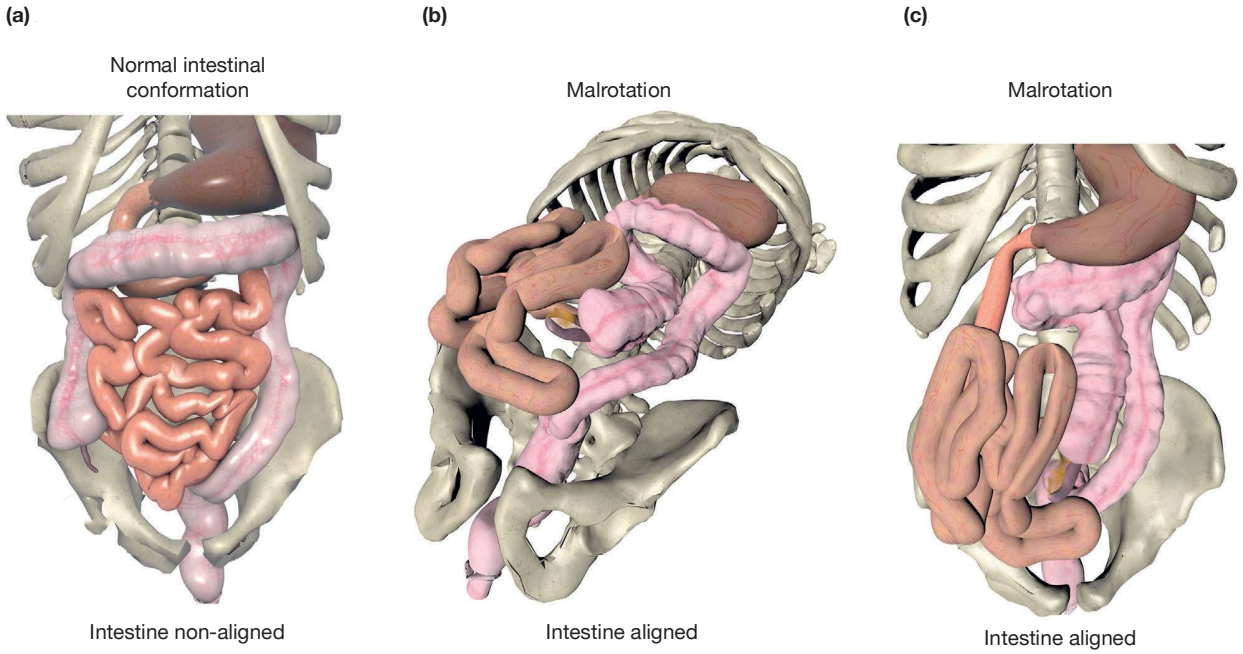


**Figure 65.13** Malrotation. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles*. Boca Raton: CRC Press, 2017: 85–108.)

spiral shape. The resultant coil/spiral complex of the intestine and mesentery is termed a volvulus. Its formation is normally prevented by adhesion of the mesentery to the posterior abdominal wall during development. Wherever mesenteric adhesion is inadequate, there is a risk of volvulus formation. Sigmoid volvulus is the commonest type, followed by ileocaecal. However, the anatomy of the mesentery is such that volvulus can theoretically arise at any level from the oesophagogastric to anorectal junction.

### History

In ileocaecal volvulus the patient often describes a longstanding history of intermittent, colicky abdominal pain associated with distension and vomiting. In between episodes (when the volvulus has detorted) the patient is entirely asymptomatic and repeated CT and endoscopic examinations are normal. The clinical picture is similar to that of irritable bowel syndrome.

Sigmoid volvulus occurs mainly in the elderly as the intestine and mesentery continue to lengthen throughout life.

### Investigation

A plain film of the abdomen is frequently diagnostic. CT is recommended when planning operative intervention.

### Treatment

Sigmoid volvulus can be quickly reversed with endoscopic decompression, but recurrence is common. Most patients will ultimately require surgery to resect the intestine and adjoining mesentery. These patients often have multiple comorbidities and present challenging anaesthetic and ethical dilemmas (see ***Chapter 77***).

## Mesenteric stretch during colonoscopy

Development of pain doing colonoscopy is frequently attributed to distension of the colon and stretch of the colon wall. In reality, substantial volumes of gas would be required to insufflate the colon to the degree at which the colon stretches. Pain during colonoscopy is common and arises mainly because of stretch of the adjoining mesentery. Looping of the intestine is a frequent event during colonoscopy. The loop corresponds to a coil, which means that the mesentery adjoining the loop must form a spiral. It thus resembles the anatomical arrangement seen in volvulus. Attempts to advance the endoscope beyond a coil will place both the intestine and mesentery under stretch. Stretch of the mesentery leads to severe central colicky abdominal pain, often accompanied by bradycardia. An understanding of the anatomical basis of loop formation enables the endoscopist to take appropriate preventative or corrective measures.

## Mesenteric sclerosis and panniculitis

### Mesenteric sclerosis

This is also termed sclerosing encapsulating peritonitis or abdominal cocoon syndrome (***Figure 65.14***). It occurs mostly in patients on long-term peritoneal dialysis. It is a disease of the mesothelial component of visceral peritoneum (i.e. peritoneum overlying organs of the mesenteric domain). The mesothelium undergoes hypertrophy and the peritoneum becomes thickened. Underlying organs become encapsulated by a peritoneal 'cocoon'. Mesenteric sclerosis may follow intraperitoneal sepsis, when fibrin plaques accumulate along the

intestine and mesentery leading to a reactionary mesothelial hypertrophy.

#### History and investigation

The clinical picture is highly variable, as is the natural history of the condition among individuals. Mesenteric sclerosis can lead to obstruction of the intestine. The diagnosis is normally made based on CT and intraoperative appearances and post-operative surgical histology.

#### Treatment

Treatment is supportive and surgery is reserved for emergency cases only as surgical intervention may lead to further peritoneal sclerosis.

### *Mesenteric panniculitis*

This is inflammation of the mesodermal mesentery (i.e. the mesenteric stroma). It is always present in Crohn's disease (see ***Mesenteric adenitis and the mesentery in Crohn's disease***). Often it is an incidental finding on cross-sectional imaging of the abdomen by CT ('misty mesentery'; ***Figure 65.14***). Although there are concerns over malignant potential, this is not supported by the general literature. It can arise secondary to inflammation in any digestive organ. It is associated with connective tissue disorders (including Weber–Christian disease). As with mesenteric sclerosis, treatment is normally medical and surgery is rarely required. Serial CT scanning is indicated to ensure resolution.

### *Sclerosing mesenteritis*

Sclerotic (mesothelial) and inflammatory (mesodermal) abnormalities of the mesentery may coexist. Although this can arise *de novo*, it is normally a secondary manifestation of another pathology (i.e. immunoglobulin G4 [IgG4] disease, which is a systemic fibroinflammatory disease).

## Adhesions

### *Pathology*

Adhesions are best classified with reference to their appearance. They are subdivided into peritoneal, areolar and dense adhesions.

#### Peritoneal (sclerotic) adhesions

These are mesothelial adhesions between two mesothelial surfaces (***Figure 65.15a***). They reflect mesothelial proliferation, resemble peritoneum and are generally soft and non-vascular. They are similar in appearance (though not in consistency) to the peritoneal cocoon that occurs in mesenteric sclerosis. These can be band-like. They may occur following laparoscopic surgery, where they form a band linking a viscus to the inner surface of a port site. Band adhesions can lead to focal abdominal pain, internal herniation (through the window created with surrounding related structures) or intestinal torsion around the band.

#### Areolar adhesions

These flimsy connective tissue adhesions are identical to the connective tissue that fills the retroperitoneal space between the mesentery and posterior abdominal wall (***Figure 65.15b***). Given this appearance they have been called 'angel hairs'. They are generated in a process similar to that involved when the mesentery adheres to the posterior abdominal wall.

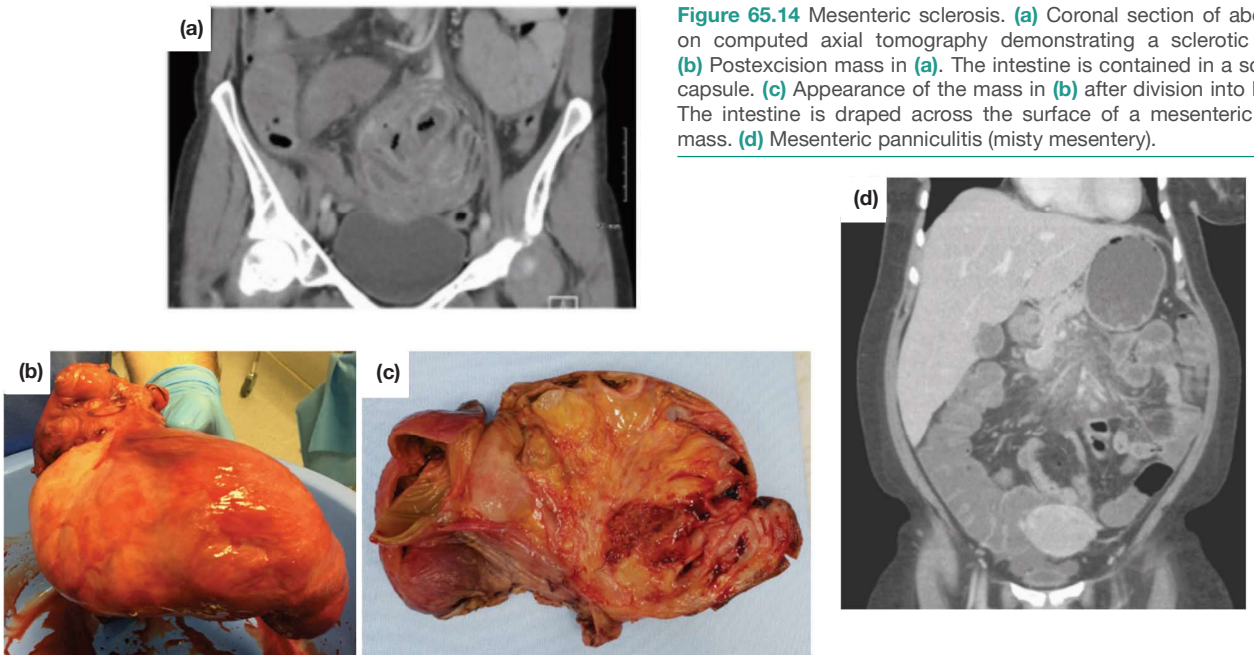


**Figure 65.14** Mesenteric sclerosis. **(a)** Coronal section of abdomen on computed axial tomography demonstrating a sclerotic mass. **(b)** Postexcision mass in **(a)**. The intestine is contained in a sclerotic capsule. **(c)** Appearance of the mass in **(b)** after division into halves. The intestine is draped across the surface of a mesenteric tissue mass. **(d)** Mesenteric panniculitis (misty mesentery).

**Frederick Parkes Weber**, 1863–1962, physician, Mount Vernon Hospital, London, UK.
**Henry Asbury Christian**, 1876–1951, pathologist, Boston, MA, USA.

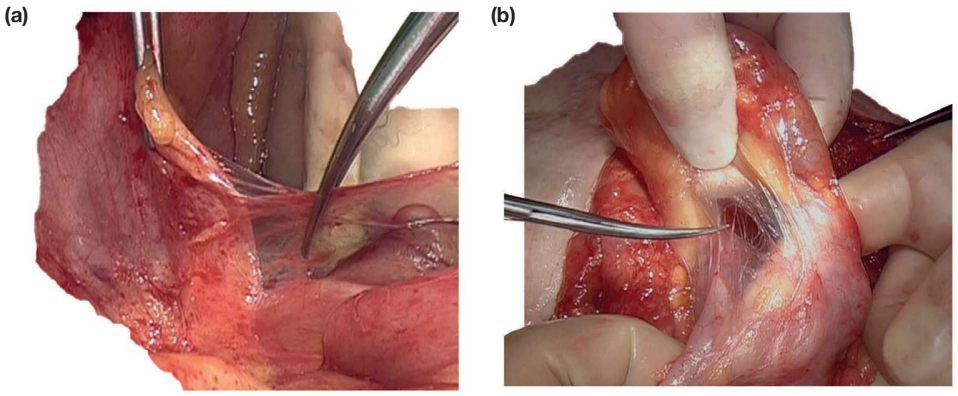


**Figure 65.15** Adhesions. **(a)** Peritoneal (sclerotic) adhesions. **(b)** Areolar adhesions. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles*. Boca Raton: CRC Press, 2017: 333–42.)

The capacity to adhere is shared by all organs of the mesenteric domain. This explains the anatomical relations between the liver, colon, spleen and mesentery on one side and the abdominal wall on the other. The capacity is retained to varying degrees among individuals and explains adhesion formation in the adult setting. Adhesion occurs following abdominal surgery when the intestine and mesentery adhere to the inside surface of the anterior abdominal wall. The resultant anatomical arrangement is similar to that observed during development, except the anterior abdominal wall is involved.

Postoperative adhesion formation occurs to varying degrees. At one end of the spectrum, it may be entirely absent. At the other end, mesenteric, intestinal and other components of the mesenteric domain may adhere over broad areas to the anterior abdominal wall. This normally commences in the midline and extends laterally towards the flanks, displacing the overlying peritoneum. The conformation of the peritoneal cavity changes markedly. In the most extremes cases, the peritoneal cavity may be obliterated or limited to small pockets at the flanks. This generates considerable technical challenges during reoperative surgery.

### Dense adhesions

These differ markedly in appearance from peritoneal or areolar adhesions. They can bridge the abdominal wall and intestine, or intestine and mesentery, and lead to fusion of the bridged structures. Surgical division can be challenging. Sometimes it is not possible to separate conjoined organs without disrupting the integrity of one of them. Such dense adhesions arise mostly following severe intraperitoneal contamination (e.g. after perforation) at sites of gross fibrin deposition and are highly variable in terms of vascularity.

## *Complications of adhesions*

The most common adhesion-related problem is small bowel obstruction (SBO). Adhesions are the most frequent cause of SBO in resource-rich countries and are responsible for 60–70% of SBOs (see ***Chapter 74***). Adhesions are also implicated as a major cause of secondary infertility (see ***Chapter 87***). The relationship of adhesions to chronic abdominal and pelvic pain is contentious. Unguided division of adhesions has not been shown to reduce chronic abdominal pain although conscious pain mapping (laparoscopy under local anaesthesia) to direct lysis may improve success rates.

A substantial industry has developed around the prevention of adhesions. To date, no agent or mechanism has been identified that reliably reduces adhesion formation.

# Mesenteric cysts

Cysts may occur in any region of the mesentery (***Figure 65.16***). They are most often observed in mesentery adjoining the small intestine (60%) or the colon (40%) and can be classified as follows:

- chylolymphatic;
- enterogenous;
- traumatic;
- hydatid.

## *Chylolymphatic cysts*

Although mesenteric cysts are rare, this is the most common variety, probably arising in congenitally misplaced lymphatic tissue that has no efferent communication with the lymphatic system (most frequently in the mesentery of the ileum). The thin wall of the cyst, which is composed of connective tissue

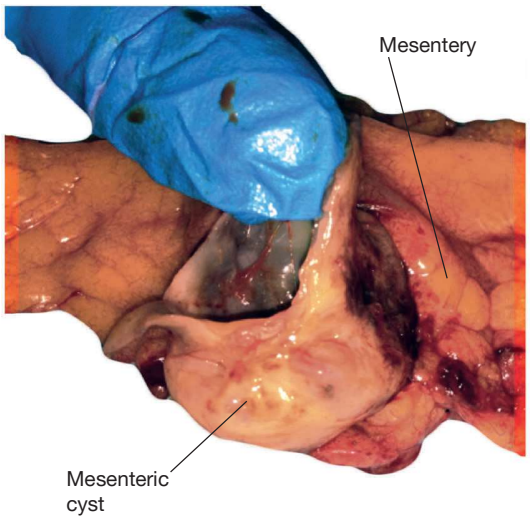


**Figure 65.16** Mesenteric cyst. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles.* Boca Raton: CRC Press, 2017: 85–108.)

lined by flat endothelium, is filled with clear lymph or, less frequently, with chyle, varying in consistency from watered milk to cream. Occasionally, the cyst attains a large size. More often unilocular than multilocular, a chylolymphatic cyst is almost invariably solitary, although there is an extremely rare variety in which myriads of cysts are found in various regions of the mesentery. A chylolymphatic cyst has a blood supply that is independent from that of the adjacent intestine and, thus, enucleation is possible without the need for resection of the gut.

### *Enterogenous cysts*

These are believed to be derived either from a diverticulum of the mesenteric border of the intestine that has become sequestrated from the intestinal canal during embryonic life or from a duplication of the intestine (see ***Chapter 18***). An enterogenous cyst has a thicker wall than a chylolymphatic cyst and is lined by mucous membrane, which is sometimes ciliated. The content is mucinous and either colourless or yellowish brown as a result of past haemorrhage. The muscle in the wall of an enteric duplication cyst and the bowel with which it is in contact have a common blood supply; consequently, removal of the cyst always entails resection of the related portion of intestine.

## Tumours of the mesentery

Primary tumours of the mesentery include carcinoid, lymphoma, sarcoma and desmoid tumours. The mesentery is affected in local lymphatic spread of carcinoma arising from abdominal viscera (***Figure 65.17***). If indicated, a benign tumour of the mesentery may be excised with resection of the adjacent intestine. A malignant tumour of the mesentery requires biopsy confirmation and specific, usually non-surgical, treatment, e.g. chemotherapy for lymphoma.

### *Diffuse fibromatosis*

Fibromatosis is rare, characterised by an abnormal proliferation of myofibroblasts. Although non-metastasising, and said to be benign, it can nevertheless prove widely invasive, compressing and infiltrating surrounding tissues such as the bowel and mesentery with complications thereof. There is an association with familial adenomatous polyposis (FAP).

**Summary box 65.9**

**Mesenteric cysts: clinical features**

- Cysts occur most commonly in adults with a mean age of 45 years
- Twice as common in women as in men
- Rare: incidence around 1 per 140 000
- Approximately a third of cases occur in children younger than 15 years
- The mean age of children affected is 5 years
- The most common presentation is of a painless fluctuant abdominal swelling near the umbilicus
- Other presentations are with recurrent attacks of abdominal pain with or without vomiting (pain resulting from recurring temporary impaction of a food bolus in a segment of bowel narrowed by the cyst or possibly from torsion of the mesentery) and acute abdominal catastrophe due to:
  - torsion of that portion of the mesentery containing the cyst
  - rupture of the cyst, often as a result of a comparatively trivial accident
  - haemorrhage into the cyst
  - infection

**Summary box 65.10**

**Mesenteric tumours**

**Benign**

- Lipoma
- Fibroma
- Fibromyxoma
- Desmoid

**Malignant**

- Lymphoma
- Secondary carcinoma
- Neuroendocrine tumours
- Lymphatic metastases
- Tumour deposits (lymphovascular and perineural)
- Peritoneal carcinomatosis

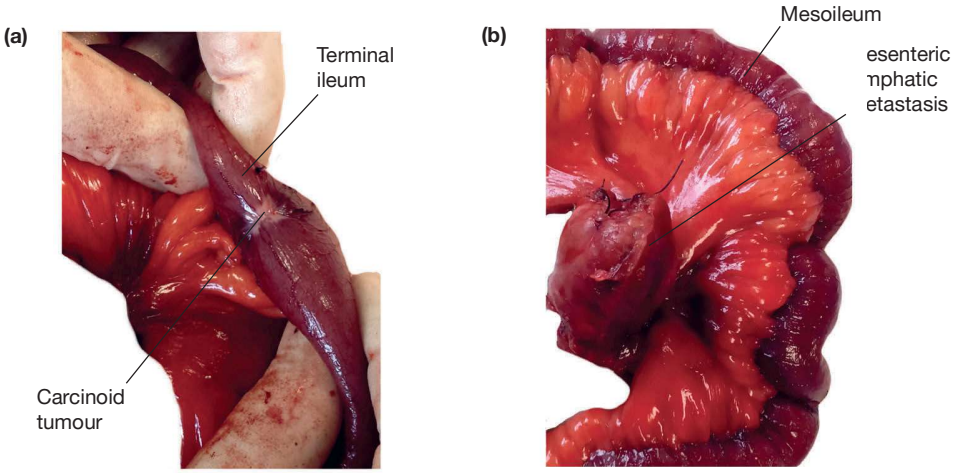


**Figure 65.17** Terminal ileal carcinoid **(a)** and lymphatic metastasis **(b)**. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles*. Boca Raton: CRC Press, 2017: 85–108.)

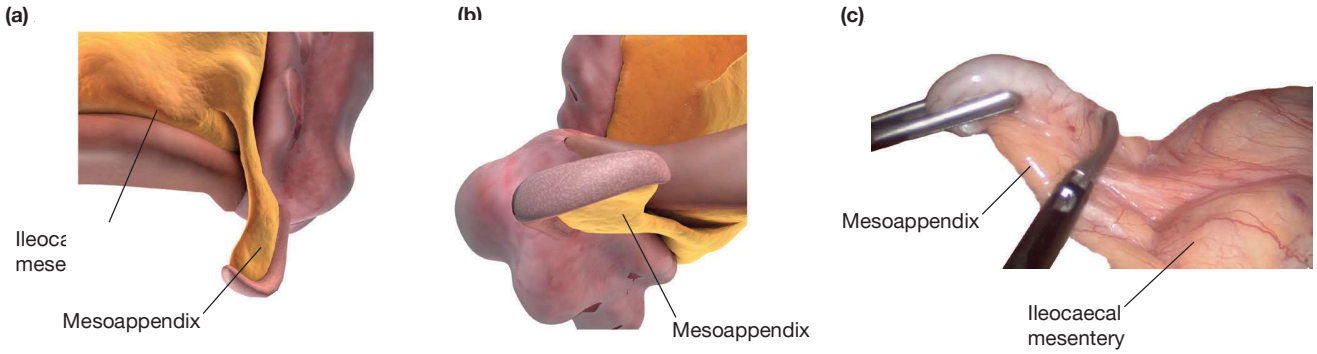


**Figure 65.18** The mesoappendix. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles*. Boca Raton: CRC Press, 2017: 11–40.)

## THE GREATER OMENTUM

The **greater omentum** corresponds to the anterior wall of the upper region of the mesentery. Rutherford Morison called the greater omentum 'the abdominal policeman'. The greater omentum attempts, often successfully, to limit intraperitoneal infective and other noxious processes. For instance, an acutely inflamed appendix is often found wrapped in omentum, and this saves many patients from developing diffuse peritonitis. The omentum often plugs the neck of a hernial sac and prevents a coil of intestine from entering and becoming strangulated. The omentum can also be a cause of obstruction (acting as a large adhesion). The omentum is usually involved in tuberculous peritonitis and carcinomatosis of the peritoneum.

The **mesoappendix** arises from the posterior aspect of the ileocaecal region of the mesentery (***Figure 65.18***). As a result, it often occupies a retrocaecal position, as does the adjoining appendix. The position of the mesoappendix is thus a determinant of the symptoms and signs associated with acute appendicitis (see ***Chapter 76***).

A **Meckel's diverticulum** (see ***Chapter 74***) resembles the appendix in that it comprises an intestinal diverticulum at the periphery of a flange of mesentery. The latter arises from a nearby small intestinal region of mesentery.

The bloodless fold of Treves, a flange of mesentery at the antimesenteric side of the ileum just proximal to the ileocaecal valve, is a mesenteric remnant that arises after differentiation of the intestine at that level.

## THE RETROPERITONEAL SPACE AND RETROPERITONEUM

The non-mesenteric domain is posterior to the mesenteric domain. The space between both is termed the retroperitoneal space (***Figure 65.19***) (***65.4***). It is a conceptual space as it contains areolar connective tissue. Regions of the connective tissue were separately named Toldt's, Waldeyer's, Denonvilliers', Gerota's and Fredet's fascia, as if they are separate entities. These are merely different zones of the same connective tissue layer that is interposed between the mesenteric domain in front and the non-mesenteric domain behind.

The space continues into the thorax and thereafter into the neck. This explains why, on occasion, a patient with an intestinal perforation during colonoscopy develops surgical emphysema and crepitus at the neck level. In these cases, perforation occurs into the retroperitoneal space (***Figure 65.20***). Gas tracks along the space into the thorax and thereafter into the neck, where it accesses subcutaneous tissue to generate surgical emphysema and crepitus. The volume of gas insufflated can be considerable given that the peritoneal cavity will not have been entered and the endoscopist may not recognise the perforation. The space may be obliterated following radiation treatment, in Crohn's disease or in longstanding diverticular inflammation. This presents considerable challenges for the surgeon who needs access to the plane whenever conducting visceral surgery.

**James Rutherford Morison**, 1853–1939, Professor of Surgery, University of Durham, Durham, UK.
**Sir Fredrick Treves**, 1853–1923, surgeon, the London Hospital, London, UK, renowned for operating on King Edward VII for appendicitis, resulting in the postponement of the coronation and his care for Joseph Merrick, the 'elephant man'.
**Carl Toldt**, 1840–1920, Professor of Anatomy in Prague, the former Czechoslovakia, and later Vienna, Austria.
**Wilhelm von Waldeyer-Hartz**, 1836–1921, Professor of Anatomy, Berlin, Germany.
**Charles-Pierre Denonvilliers**, 1808–1872, Professor of Surgery and Anatomy, Paris, France.
**Dimitrie Gerota**, 1867–1939, Professor of Surgical Anatomy, Bucharest, Romania.
**Pierre Fredet**, 1870–1946, surgeon, Paris, France.

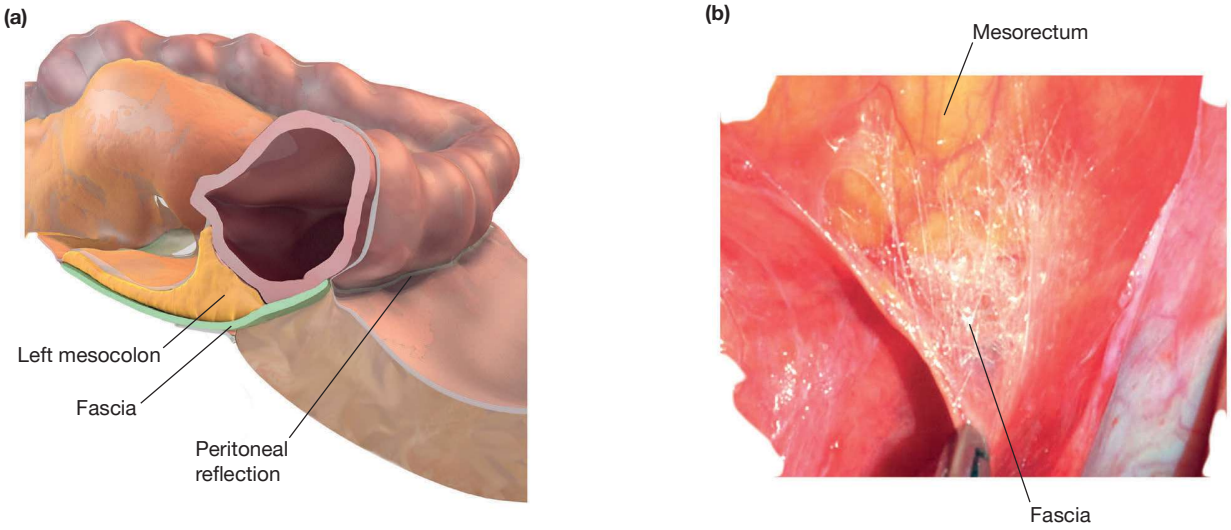


**Figure 65.19** The retroperitoneal space. **(a)** Digital image of the fascia (green) located in the retroperitoneal space. **(b)** Intraoperative appearance of fascia in the retroperitoneal space. (Reproduced with permission from Coffey JC, Lavery I, Sehgal R (eds). *Mesenteric principles of gastrointestinal surgery: basic and applied principles*. Boca Raton: CRC Press, 2017: 11–40 and 57–68.)

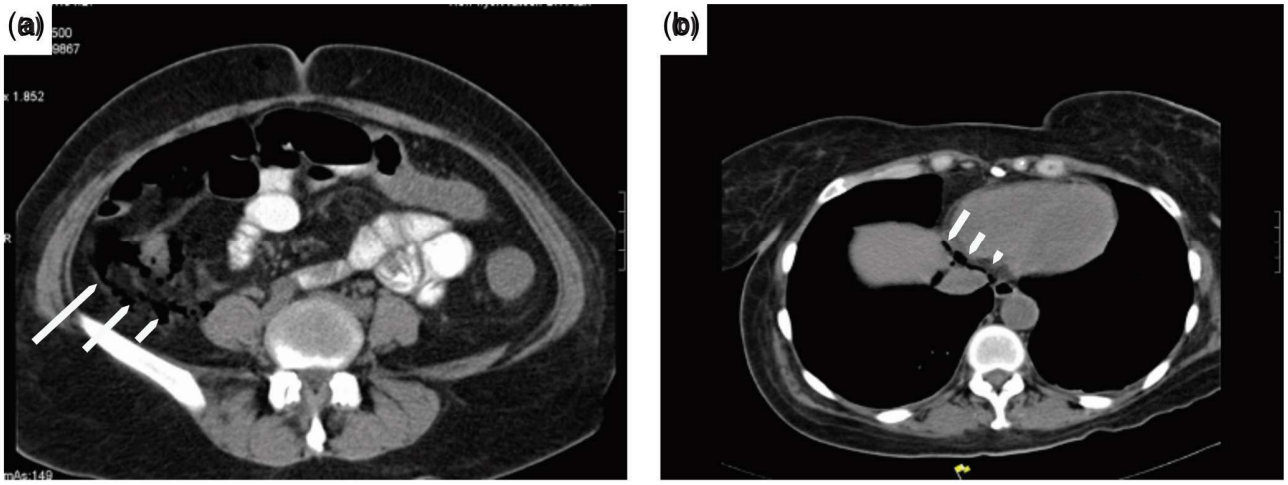


**Figure 65.20** Perforation into the retroperitoneal space. Axial computed tomography section of the abdomen demonstrating gas (arrows) in the retroperitoneal space **(a)** and mediastinum **(b)**.

## RETROPERITONEAL SPACE COLLECTIONS

These are fluid collections in the retroperitoneal space and these differ from intraperitoneal collections because of their location (***Figure 65.21***). They are a common finding in moderate to severe acute pancreatitis. Fluid accumulates as a result of pancreatic inflammation, dissecting the left mesocolon off the underlying fascia and posterior abdominal wall. With continued expansion retroperitoneal space collections track subperitoneally around the flanks. A rapidly expanding retroperitoneal collection, such as occurs with a ruptured aortic aneurysm, may rupture intraperitoneally.

## THE RETROPERITONEUM

The retroperitoneum is the region of the non-mesenteric domain deep to the retroperitoneal space. It contains the kidneys, adrenal glands, major vessels, ureters and gonadal vessels and is surrounded by adipose tissue.

Swellings in the retroperitoneum include abscess, haematoma, cysts and malignancy from retroperitoneal organs (kidney, ureter, adrenal). The term retroperitoneal tumour refers to primary tumours arising in connective tissues in this region.

### Retroperitoneal fibrosis

This is a relatively rare diagnosis characterised by development of a flat grey/white plaque of tissue that usually develops in

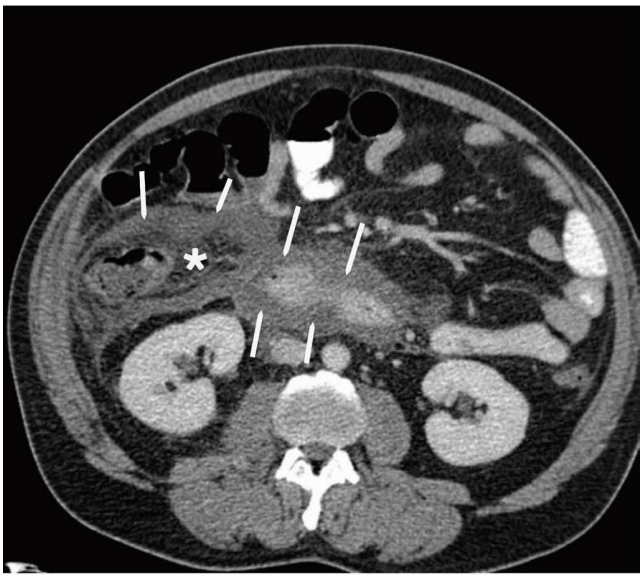

Figure 65.21 Computed tomography scan demonstrating retroperitoneal space collection in acute pancreatitis. The collection (arrows) has dissected the right mesocolon (asterisk) and duodenum, off the underlying retroperitoneum. Fluid is apparent in the retroperitoneal space.

the low lumbar region and later spreads laterally and upwards to encase the common iliac vessels, ureters and aorta. Histological appearances vary from active inflammation with a high cellular content interspersed with bundles of collagen through to one of relative acellularity and mature fibrosis/calcification. Its aetiology is obscure in most cases (idiopathic; synonym Ormond's disease), being allied to other fibromatoses (others being Dupuytren's contracture and Peyronie's disease).

**Summary box 65.11**

**Causes of retroperitoneal fibrosis**

**Benign**

- Idiopathic (Ormond's disease)
- Chronic inflammation
- Extravasation of urine
- Retroperitoneal irritation by leakage of blood or intestinal content
- Aortic aneurysm (inflammatory type)
- Trauma
- Drugs (chemotherapeutic agents and previously methysergide)

**Malignant**

- Lymphoma
- Carcinoid tumours
- Secondary deposits (especially from carcinoma of stomach, colon, breast and prostate)

## Retroperitoneal (psoas) abscess

The psoas abscess is an abscess of the retroperitoneum (*Figure 6.22*). At the start of the twentieth century, psoas abscess was mainly caused by TB of the spine (Pott's disease). With the decline of *M. tuberculosis* as a major pathogen in resource-rich countries, a psoas abscess was mostly found secondary to direct spread of infection from the inflamed digestive or urinary tract with or without perforation. In more recent years it is most commonly seen in advanced Crohn's disease. Rarely, it arises due to haematogenous spread from an occult source in immunocompromised patients and in association with intravenous drug misuse.

### History

Clinical presentation is with back pain, lassitude and fever. A swelling may point to the groin as it tracks distally along the iliopsoas muscle, under the inguinal ligament. Pain may be elicited by passive extension of the hip or a fixed flexion of the hip evident on inspection.

### Investigation and treatment

Radiological investigation is by CT scanning and treatment is usually by percutaneous CT-guided drainage and appropriate antibiotic therapy. Surgical intervention is required if these are unsuccessful.

## Retroperitoneal lipoma

The patient may seek advice on account of a swelling or because of indefinite abdominal pain. The swelling sometimes reaches an immense size. Diagnosis is usually by CT scan. A retroperitoneal lipoma sometimes undergoes myxomatous degeneration, a complication that does not occur in a lipoma

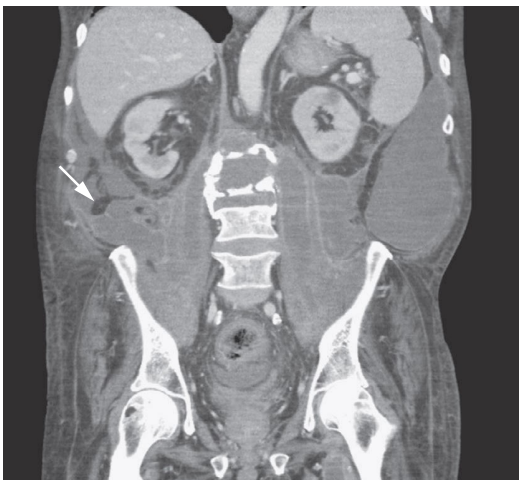

Figure 65.22 Representative sagittal computed tomography reconstruction of a right-sided psoas abscess (arrow) (courtesy of Dr K Patel, Homerton University Foundation Trust, London, UK).

**John Kelso Ormond**, 1886–1978, urologist, Ann Arbor, MI, USA.
**Baron Guillaume Dupuytren**, 1777–1835, Surgeon in Chief, Hôtel Dieu, Paris, France.
**François Gigot de la Peyronie**, 1678–1747, surgeon to King Louis XIV of France.
**Percival Pott**, 1714–1788, surgeon, St Bartholomew's Hospital, London, UK.

in any other part of the body. A lesion that rapidly increases in size is often malignant (liposarcoma).

## Retroperitoneal sarcoma

Retroperitoneal sarcomas are rare tumours accounting for only 1–2% of all solid malignancies (10–20% of all sarcomas are retroperitoneal). The peak incidence is in the fifth decade of life, although they can occur at almost any age. The most frequently encountered cell types are:

- liposarcoma;
- leiomyosarcoma;
- malignant fibrous histiocytoma.

### *History and examination*

Patients with sarcomas present late because these tumours arise in the large potential spaces of the retroperitoneum and can grow to a considerable size without producing symptoms. Moreover, when symptoms do occur, they are non-specific, such as abdominal pain and fullness, and are easily dismissed as being caused by other less serious processes. Retroperitoneal sarcomas are therefore often very large at the time of presentation.

### *Investigation*

Detailed multiplanar imaging (CT and MRI) with reconstructions is required not only for tumour detection, staging and surgical planning but also for guiding percutaneous or surgical biopsy of these tumours.

### *Treatment*

The definitive treatment of primary retroperitoneal sarcomas is surgical resection. Chemotherapy and radiotherapy without surgical debulking have rarely been beneficial, when used alone or in combination. A multidisciplinary treatment approach with imaging review will be required when assessing operability (based on adjacency or involvement of vital structures) and approach. Up to 75% of retroperitoneal sarcoma resections involve resection of at least one adjoining intra-abdominal visceral organ (commonly large or small bowel or kidney). The most common types of vascular involvement precluding resection are involvement of the proximal superior mesenteric vessels or involvement of bilateral renal vessels.

### *Prognosis*

Survival rates are in general poor, even after complete resection, being of the order of 35–50% (excluding low-grade liposarcomas, which may frequently be cured by resection).

## ACKNOWLEDGEMENTS

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