Ascites

Introduction

Ascites is a common finding on clinical examination during a general medical take, and regularly features in postgraduate examinations. Approximately 75% of patients presenting with ascites have underlying liver cirrhosis, in whom the condition is associated with a 50% increase in morbidity and mortality (Kim et al, 2002). As there is an increasing frequency of both alcoholic and non-alcoholic liver disease in the UK, the burden of cirrhosis will continue to rise (Roberts et al, 2005). This makes ascites a likely case to encounter in station 1 of the PACES examination.

Pathophysiology

Ascites occurs as a result of accumulation of peritoneal fluid within the abdominal cavity. There are two main processes thought to contribute to this: portal hypertension, and sodium and water retention. Other factors include inflammatory, infective or malignant pathology, and protein wasting.

Portal hypertension causes increased hydrostatic pressure within hepatic sinusoids. This in turn increases pressure within the splanchnic circulation, causing fluid to extravasate and accumulate in the peritoneal cavity. Portal hypertension arises as a result of changes within the structure of the liver resulting from fibrosis by increased collagen deposition, combined with increased blood flow through the splanchnic circulation. Patients with a hepatic venous portal gradient of less than 12 mmHg rarely develop ascites (Llach et al, 1988).

Dr Selina J Chavda *is ST3 National Institute of Health Research Academic Clinical Fellow Haemato-oncology in the Department of Haematology, Royal Marsden Hospitals NHS Foundation Trust, Sutton, Surrey SM2 5PT and* **Dr Stuart L Bloom** *is Consultant Gastroenterologist in the Department of Gastroenterology, University College London Hospital, London*

Correspondence to Dr SJ Chavda (selinachavda@doctors.org.uk)

The wedged hepatic venous pressure is used to measure the pressures within the hepatic sinusoids and is calculated by placing a catheter into the hepatic vein to ensure it is occluded. The pressure of the proximal static blood is then measured. This pressure is equivalent to the hepatic sinusoid pressures and allows calculation of the hepatic venous pressure gradient, a useful clinical indicator. The hepatic venous pressure gradient is a measurement of the gradient between the wedged hepatic venous pressure and the free hepatic venous pressures. It is therefore an estimate of the pressure gradient between the portal vein and inferior vena cava and is useful in chronic liver disease as a means of monitoring treatment response and likelihood of disease progression (Kumar et al, 2008).

Patients with liver cirrhosis also tend to develop systemic vasodilatation, because the fibrotic liver produces vasodilatory compounds, including nitric oxide and prostacyclin (Ginès et al, 1997). The resultant decrease in systemic vascular resistance in turn drives increased sympathetic activity and activation of the renin–angiotensin system, ultimately leading to reduced urinary sodium excretion via aldosterone. Sodium reabsorption is also enhanced in both the proximal and distal renal tubules, which concomitantly increases water reabsorption from the kidneys, further contributing to fluid retention (Moore and Van Thiel, 2013).

Additional mechanisms can also promote the development of ascites. Reduced plasma oncotic pressure secondary to poor protein synthesis (seen in malnutrition and end-stage chronic liver disease) or protein wasting (seen in nephrotic syndrome and protein-losing enteropathies) alters the balance of Starling forces and favours the leakage of fluid out of the vascular compartment (Schrier, 1988; Umar and DiBaise, 2010). Finally infectious, autoimmune or neoplastic pathologies can cause local secretion of inflammatory exudate into the peritoneal space, faster than intravascular re-absorption can occur (Williams and Simel, 1992).

Examining for ascites

Symptoms of ankle oedema, weight gain or a change in abdominal girth should be elicited. Ascites is unlikely when patients report no increase in abdominal girth, and very unlikely in male patients who report no history of recent ankle swelling (Williams and Simel, 1992).

Inspection

The patient should be exposed from xiphisternum to the groins, and be positioned supine. Owing to gravity, fluid tends to collect within the flanks and causes symmetrical distension of the abdomen (or 'bulging flanks'; *Figure 1*). In gross ascites, the umbilicus can evert as the abdomen distends to accommodate the excess peritoneal fluid, and divarication of the rectus muscle may be seen when the patient tenses the anterior abdominal wall. Look carefully for scars or dressings from previous ascitic taps or paracentesis. Serial measurements of abdominal girth can be useful in the outpatient setting. Inspect for signs of chronic liver disease or portal hypertension, which may provide clues as to the underlying aetiology (*Table 1*).

Palpation

The liver and spleen should be palpated carefully to determine whether the patient has significant portal hypertension causing splenomegaly. Most cases will result from intrinsic hepatic disease,

Figure 1. A patient with bulging flanks.

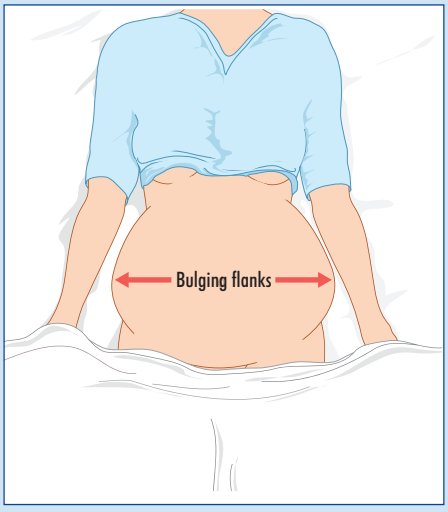


Table 1. Signs of chronic liver disease

General inspection	Cachexia, bruising
Hands	Leuconychia, Terry's nails (white nails proximally, distal third of nails reddened by telangiectasia), Dupuytren's contracture, clubbing, palmar erythema
Face	Parotid enlargement, fetor hepaticus, xanthelasma
Torso	Spider naevi, caput medusae, gynaecomastia, abnormal body hair distribution, testicular atrophy

often associated with a small cirrhotic liver (Brown et al, 2011). However, hepatomegaly may result from malignant infiltration (craggy and irregular) or post-hepatic portal hypertension as a result of Budd–Chiari syndrome, inferior vena cava thrombosis, right heart failure or constrictive pericarditis (smoothly enlarged and often tender; pulsatile in severe tricuspid regurgitation).

Percussion

To confirm that bulging flanks are caused by ascites and not obesity, percuss for flank dullness. Start in the midline at the umbilicus and percuss towards the flanks. The examiner's hands should be placed vertically on the patient's abdomen (*Figure 2*). In ascites, the gas-filled bowel loops float above the intra-peritoneal fluid, resulting in tympanic percussion at the umbilicus and dullness laterally where the fluid level is found (*Figure 3*). This has a sensitivity of 84% and specificity of 59% for detecting ascites (Simel et al, 1988; Williams and Simel, 1992).

Shifting dullness can be demonstrated by performing percussion as above, then asking the patient to roll towards the examiner. He/she should remain in this position for a few seconds at which point the manoeuvre is repeated. If ascites is present, the area of dullness shifts towards the dependent side, while the area of tympanic percussion shifts towards the top (*Figure 4*; Williams and Simel, 1992). Shifting dullness has a sensitivity of 77% and specificity of 72% in detecting ascites (Williams and Simel, 1992; Runyon, 2009). It is important therefore to note that shifting dullness may miss some cases of ascites (particularly where the volume of fluid is small), and that it is not pathognomonic (it can arise in the absence of intra-abdominal fluid).

A fluid thrill or wave can be elicited in tense ascites. The test is performed by asking the patient or an assistant to place the ulnar border of his/her hands on the patient's abdomen in the midline, to block transmission of a wave through subcutaneous fat. The examiner should tap one flank sharply and firmly, and feel for an impulse with his/her fingertips on the opposite flank. Although this sign lacks sensitivity at 62%, if present it is highly specific at 90% (Williams and Simel, 1992).

Auscultation

Auscultation should be performed as routine in the general abdominal examination. A technique of auscultatory percussion for ascites has been described (Guarino, 1986). In this test, the patient is asked to stand or sit after voiding, allowing free fluid to collect within the pelvis and the viscera to float above. Finger-flicking percussion is then performed from the xiphisternum down to the midline to the pelvis, while the examiner listens with the diaphragm of the stethoscope placed over the pubis (*Figure 5*). The percussion note should change from a dull to a loud note at the level of increased pelvic density. In patients without ascites, this should normally occur at approximately 4.5 cm above the pelvic crest. Patients with ascites will have a level higher than this as a result of the excess fluid, and the technique is said to be able to detect as little as 140 ml of ascites. The patient then lies supine, and the fluid level disappears as ascites redistributes into the flanks (McLean, 1987; Williams and Simel, 1992). Nonetheless, this technique has not entered the routine examination as it was found to have low sensitivities and specificities in larger studies (Simel et al, 1988).

The 'puddle sign' has been frequently described in the literature. The patient is asked to lie prone on the examination couch for 5 minutes, then squat on his/her knees and elbows. The examiner places his/her stethoscope on the most dependent part of the abdomen and flicks the lateral aspect of the flank with his/her finger. This is repeated while the examiner

Figure 2. How to percuss the abdomen.

Figure 3. Areas of the abdomen which should be tympanic and which should be dull.

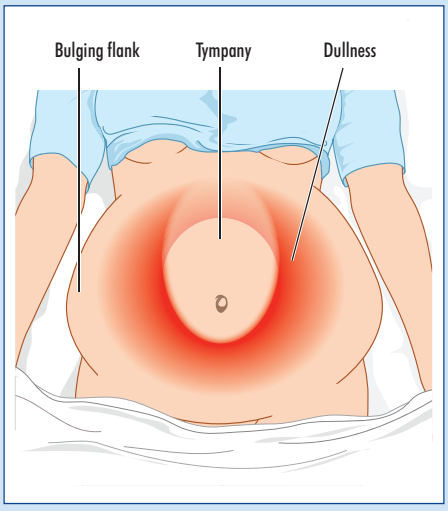
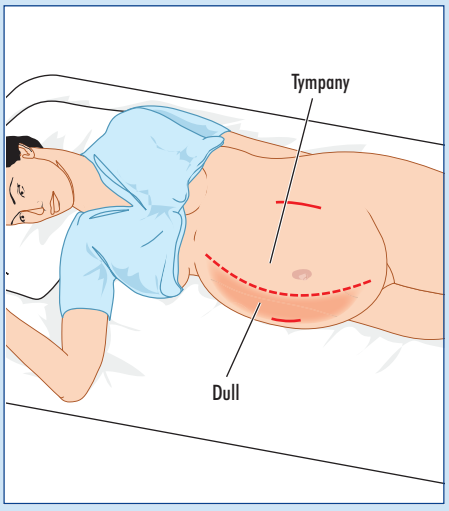


Figure 4. Shifting dullness.

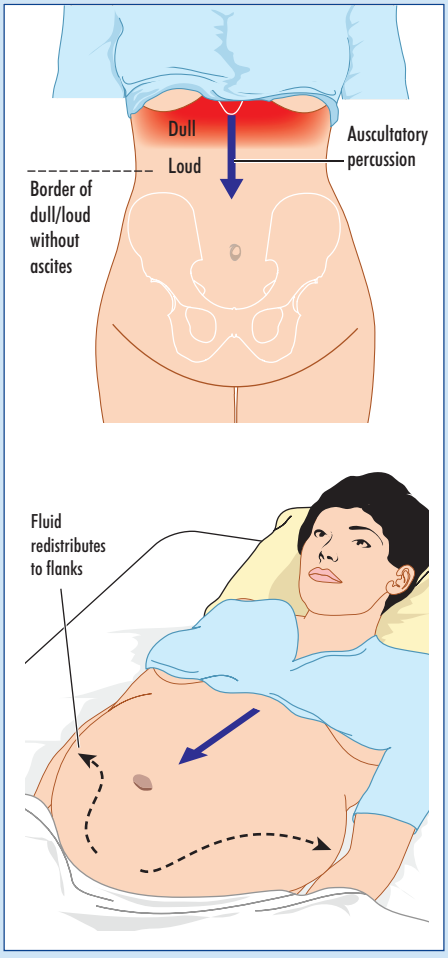


moves his/her stethoscope across the abdomen, away from his/her own body. The sound caused by flicking the flank should become louder at the farther edge of the 'puddle' of ascites, but should not change in intensity when the patient sits. This test has also fallen out of favour as it is difficult to perform properly, uncomfortable for the patient and lacks sensitivity, ranging from 43–55% (Williams and Simel, 1992).

Reliability of physical signs

Table 2 summarizes the reliability of the classical clinical signs used to detect ascites (after Williams and Simel, 1992). In general, there is high inter-observer agreement for these signs, of approximately 75% when all clinicians are tested and up to 95% when examination is restricted to senior physicians (Espinoza et al, 1987).

Figure 5. Auscultatory percussion. a. Patient with ascites standing resulting in a transition point between air and fluid noted on auscultation. b. When patient lies flat, fluid redistributes, obliterating level.



All have limits to their sensitivity and specificity, partially dependent on the volume of ascites present, and therefore the signs should be used in conjunction. The most useful clinical features for excluding the presence of ascites are a combination of absence of ankle swelling or increased abdominal girth, alongside inability to demonstrate bulging flanks, flank dullness or shifting dullness. The most effective features to allow a positive diagnosis are the presence of a fluid wave, alongside shifting dullness and peripheral oedema.

Grading ascites

Ascites is graded in terms of its volume (*Table 3*). There is some variability in the published literature as to the precise volumes of fluid that cause each grade of ascites. Ascites that is not infected nor associated with hepatorenal syndrome is 'uncomplicated'. Refractory ascites is defined as that recurring rapidly after therapeutic paracentesis despite appropriate diuretic therapy (Saich and Chapman, 2006).

Causes of ascites

The causes of ascites, and clues to the aetiology on physical examination, are detailed in *Table 4*. The three most common causes are chronic liver disease (75%), liver meta-stases (10%) and right-sided heart failure (3%). Causes of ascites can also be divided into those causing low protein transudates or high protein exudates. Although historically a cut-off of 25 g/dl of ascitic protein has been used, the serum ascites–albumin gradient is more accurate (*Table 5*). Most causes of ascites generate transudates.

Ascites is also used in several clinical hepatology scoring systems to assess severity of disease and predict mortality. A commonly used clinical scoring system in hepatology is the Child–Pugh scoring system. This system helps to assess prognosis of chronic liver disease patients and ultimately the need for liver transplantation. Ascites is one of the key components of this scoring system (*Table 6*) (Saich and Chapman, 2006). Scores are calculated and patients divided into groups as shown:

Score	Class	One-year survival
5–6	A	100%
7–9	B	81%
10–15	C	45%

Investigation and management

The investigation and management of ascites is detailed briefly below. For further details of management of cirrhosis, please see Bashyam et al (2015) in this issue. Laboratory investigations should include routine blood tests, and analysis of ascitic fluid for white cell count and differential (for evidence of infection), albumin and total protein, Gram stain and culture (including for acid/alcohol-fast bacilli), cytology, lactate dehydrogenase and amylase. A neutrophil count of >250 cells/mm^3 is diagnostic for spontaneous bacterial peritonitis, and clinicians should instigate prompt antibiotic therapy.

Table 2. Pooled results of physical examination studies

Physical sign	Sensitivity	Specificity	Positive likelihood ratio	Negative likelihood ratio
Bulging flanks	81% (69–93%)	59% (50–68%)	2.0 (1.5–2.6)	0.3 (0.2–0.6)
Flank dullness	84% (68–100%)	59% (47–71%)	2.0 (1.5–2.9)	0.3 (0.1–0.7)
Shifting dullness	77% (64–90%)	72% (63–81%)	2.7 (1.9–3.9)	0.3 (0.2–0.6)
Fluid wave	62% (47–77%)	90% (84–96%)	6.0 (3.3–11.1)	0.4 (0.3–0.6)

after Williams and Simel (1992). 95% confidence intervals are shown

Table 3. Grading of ascites

Grade of ascites	Severity	Clinical features
1	Mild	Only detectable using ultrasound
2	Moderate	Abdomen moderately distended, detectable by flank bulging and shifting dullness
3	Large	Abdomen grossly distended and tense, fluid thrill or wave usually present

Table 4. Causes of ascites

Causes of ascites		Signs to look for on examination
Transudative	Cirrhotic liver disease	See *Table 1*. Also reduced liver span, splenomegaly, signs of portal hypertension
	Right-sided heart failure	Raised jugular venous pressure, hepatomegaly (pulsatile in severe tricuspid regurgitation), peripheral oedema, third heart sound
	Nephrotic syndrome	Peripheral ± peri-orbital oedema, anasarca, leuconychia, proteinuria
	Protein-losing enteropathy	Weight loss, leuconychia, peripheral oedema
	Chylous ascites	Milky fluid seen on ascitic tap, with raised triglyceride count. Search for a history or evidence of trauma, and consider lymphoma (lymphadenopathy, splenomegaly)
	Budd–Chiari syndrome*	Tender hepatomegaly, rarely jaundice (Takagi et al, 2002)
	Constrictive pericarditis*	Kussmaul's sign, right heart failure, hepatosplenomegaly
	Meig's syndrome*	Right pleural effusion in a female patient
Exudative	Malignancy†	Cachexia, radiotherapy tattoos, adenopathy, masses, irregular hepatomegaly, palpable peritoneal deposits
	Tuberculosis	Lymphadenopathy, apical fibrosis, weight loss
	Pancreatitis	Tachycardia, fever, Grey–Turner's sign (flank bruising), Cullen's sign (peri-umbilical bruising)
	Spontaneous bacterial peritonitis	Fever and abdominal pain, sudden deterioration. Neutrophil count on ascitic tap >250 cells/mm^3 diagnostic
	Traumatic ascites	History or evidence of trauma, abdominal wall ecchymoses or other injuries, haemodynamic compromise
	Serositis* Inflammatory pathologies (e.g. systemic lupus erythematosus)	Past medical and family history, malar rash, erysipeloid reaction on legs, scarring alopecia, oral aphthous ulcers, renal involvement, arthropathy
	Familial Mediterranean fever	
	Hypothyroidism*	'Peaches and cream' complexion, dry skin, cold intolerance, bradycardia, goitre, slow-relaxing reflexes

*indicates a rare cause. † = gastric, ovarian, colonic, breast or pancreatic metastatic adenocarcinomas, hepatocellular cancer, peritoneal mesothelioma.

The serum ascites–albumin gradient should be calculated, as it guides both diagnosis (*Table 5*) and management (diuretic therapy, and human albumin solution during paracentesis, are indicated for transudative ascites but are generally ineffective in exudative ascites; Runyon, 2009).

Table 5. Causes of ascites by serum ascites–albumin gradient

Causes of ascites giving a serum ascites–albumin gradient >1.1	Ascites associated with portal hypertension (97% accuracy)
	Cardiac failure
	Budd–Chiari syndrome
Causes of ascites giving a serum ascites–albumin gradient <1.1	Ascites in patients with normal portal pressures
	Tuberculosis
	Malignancy
	Nephrotic syndrome
	Protein-losing enteropathy
	Pancreatic ascites
	Hypothyroidism

From Moore and Aithal (2006)

An ultrasound of the abdomen should be performed to confirm and quantify ascites, assess for neoplastic liver lesions and portal hypertension, and exclude hepatic or portal vein thromboses by Doppler analysis. Basic ultrasound scanning can be performed by a clinician to diagnose ascites. Studies have shown that acute medical trainees were able to diagnose ascites with a high level of accuracy (100% of participants were able to diagnose ascites, negative predictive value 93%) (Ismaeel et al, 2010). Additional cross-sectional imaging (by computed tomography or magnetic resonance cholangiopancreatography) may subsequently be required (Saich and Chapman, 2006).

The patient should be resuscitated if he/she is haemodynamically unstable, and spontaneous bacterial peritonitis treated promptly with broad-spectrum antibiotics as per local guidelines (Takagi et al, 2002). Any reversible underlying cause of ascites should be addressed.

Patients should be advised to have a 'no added salt' diet (approximately 90 mmol sodium/day), and be referred to a dietician for education and advice (Moore and Aithal, 2006). Diuretics are the mainstay of treatment, particularly loop diuretics (furosemide and bumetanide) and aldosterone antagonists (spironolactone and amiloride).

Table 6. Child–Pugh scoring system

	1 point	2 points	3 points
Total bilirubin (μmol/litre; mg/dl)	<34	34–50	>50
Serum albumin (g/litre)	>35	28–35	<28
International normalized ratio	<1.7	1.71–2.30	> 2.30
Ascites	None	Mild	Moderate to severe
Hepatic encephalopathy	None	Grade I–II (or suppressed with medication)	Grade III–IV (or refractory)

Grade 3 or diuretic-resistant ascites is treated with therapeutic paracentesis. There should be concurrent administration of human albumin solution for patients in whom the process is transudative (Alves, 2009). This helps to prevent paracentesis-induced circulatory dysfunction and hepatorenal syndrome (Saich and Chapman, 2006), particularly if more than 5 litres of fluid is removed (Moore and Van Thiel, 2013). In particular, if an ascitic drain is inserted for symptomatic relief, it should be removed after 6 hours to reduce the risk of spontaneous bacterial peritonitis (Wong et al, 2008).

In patients in whom ascites is frequently recurrent and increasingly difficult to manage, a transjugular intrahepatic portosystemic shunt can be considered, and ultimately liver transplantation. Transjugular intrahepatic portosystemic shunts are inserted by an interventional radiologist under fluoroscopic guidance, by creating an artificial shunt between the portal and hepatic veins. The aim of the procedure is to reduce the raised portal venous pressures, therefore decreasing the risk of variceal bleeding and reducing ascites burden over time. Common complications include transient hepatic encephalopathy caused by raised ammonia levels as a result of reduced first pass metabolism of nitrates from the gut, and less commonly hepatic ischaemia (Moore and Van Thiel, 2013).

Conclusions

Ascites is a common clinical finding, and frequently features in postgraduate exams. The burden of ascites is likely to increase as a result of the number of patients with both alcohol-related cirrhosis and non-alcoholic fatty liver disease. The physical examination should focus on demonstrating the presence of ascites, then trying to determine its underlying aetiology. Further investigations can then be implemented to ascertain the cause. Management involves salt restriction, diuretic therapy, paracentesis, and transjugular intrahepatic portosystemic shunts or liver transplantation in advanced cases. **BJHM**

Conflict of interest: none.

Alves A (2011) Current indications for the use of albumin in the treatment of cirrhosis. *Ann Hepatol* **10**(Suppl 1): S15–20 (doi: 10.1016/j.ejim.2011.06.013)
Bashyam M, Lepore M, Harbord M (2015) Management of cirrhotic ascites. *Br J Hosp Med (Lond)* **76**(2): C28–C32 (doi: 10.12968/hmed.2015.76.Sup2.C28)
Brown NF, Marks DJ, Smith PJ, Bloom SL (2011) Splenomegaly. *Br J Hosp Med (Lond)* **72**(11): M166–9 (doi: 10.12968/hmed.2011.72.Sup11.M166)
Espinoza P, Ducot B, Pelletier G et al (1987) Inter-observer agreement in the physical diagnosis of alcoholic liver disease. *Dig Dis Sci* **32**: 244–7 (doi: 10.1007/BF01297048)
Ginès P, Fernández-Esparrach G, Arroyo V, Rodés J(1997) Pathogenesis of ascites in cirrhosis. *Semin Liver Dis* **17**: 175–89
Guarino JR (1986) Auscultatory percussion to detect ascites. *N Engl J Med* **315**: 1555–6 (doi: 10.1056/NEJM198612113152424)
Ismaeel SM, Day NJ, Earnshaw D, Lorains JW (2010) Training requirements for point of care ultrasound in acute medicine. *Acute Med* **9**(2): 87–90
Kim WR, Brown RS Jr, Terrault NA, El-Serag H (2002) Burden of liver disease in the United States: summary of a workshop. *Hepatology* **36**: 227–42 (doi: 10.1053/jhep.2002.34734/)
Kumar A, Sharma P, Sarin SK (2008) Hepatic venous pressure gradient measurement: time to learn! *Indian J Gastroenterol* **27**(2): 74–80
Llach J, Ginès P, Arroyo V et al (1988) Prognostic value of arterial pressure, endogenous vasoconstrictive systems, and renal function in cirrhotic patients admitted to the hospital for the treatment of ascites. *Gastroenterology* **94**: 482–7
McLean ACJ (1987) Diagnosis of ascites by auscultatory percussion and hand-held ultrasound unit. *Lancet* **ii**: 1526–7 (doi: 10.1016/S0140-6736(87)92662-6)
Moore C, Van Thiel DH (2013) Cirrhotic ascites review: Pathophysiology, diagnosis, and management. *World J Hepatol* **5**(5): 251–63 (doi: 10.4254/wjh.v5.i5.251)
Moore KP, Aithal GP (2006) Guidelines on the management of ascites in cirrhosis. *Gut* **55**: 1–12 (doi: 10.1136/gut.2006.099580)
Roberts SE, Goldacre MJ, Yeates D (2005) Trends in mortality after hospital admission for liver cirrhosis in an English population from 1968 to 1999. *Gut* **54**: 1615–21 (doi: 10.1136/gut.2004.058636)
Runyon B (2009) Management of adult patients with ascites due to cirrhosis: an update. *Hepatology* **49**: 2087–98 (doi: 10.1002/hep.22853)
Saich R, Chapman R (2006) What's new in... Liver disorders. *Medicine* **34**: 1–4
Schrier RW (1988) Pathogenesis of sodium and water retention in high-output and low-output cardiac failure, nephrotic syndrome, cirrhosis, and pregnancy. *N Engl J Med* **319**(16): 1065–72 (doi: 10.1056/NEJM198810273191705)
Simel DL, Halvorsen RA, Feussner JR (1988) Quantitating bedside diagnosis: clinical evaluation of ascites. *J Gen Intern Med* **260**: 652–4 (doi: 10.1007/BF02595917)
Takagi H, Otsuka T, Mori M et al (2002) Early diagnosis and radical surgical treatment of Budd-Chiari syndrome. *Hepatogastroenterology* **49**(48): 1676–8
Umar SB, DiBaise JK (2010) Protein-losing enteropathy: case illustrations and clinical review. *Am J Gastroenterol* **105**(1): 43–9 (doi: 10.1038/ajg.2009.561)
Williams JW, Simel D (1992) Does this patient have ascites? How to divine fluid in the abdomen. *JAMA* **267**: 2645–8 (doi: 10.1001/jama.1992.03480190087038)
Wong CL, Holroyd-Leduc J, Thorpe KE, Straus SE (2008) Does this patient have bacterial peritonitis or portal hypertension? How do I perform paracentesis and analyze the results? *JAMA* **299**: 1166–78 (doi: 10.1001/jama.299.10.1166)

KEY POINTS

- The commonest causes of ascites are liver cirrhosis (75%), malignancy and heart failure.
- Ascites is unlikely to be present if there is no history of increased abdominal girth, or (in men) ankle swelling.
- All physical signs of ascites have limitations and none is pathognomonic.
- The absence of bulging flanks, flank dullness and shifting dullness effectively excludes the diagnosis of all but grade 1 ascites.
- The presence of shifting dullness with a fluid thrill establishes the diagnosis.
- Auscultatory percussion and the 'puddle sign' have been described as means of detecting small volume ascites clinically, but are complex to perform and lack sensitivity.
- A diagnostic tap should be sent for albumin, cell count, Gram stain and culture, and cytology.
- Management involves treating underlying cause, a 'no-added salt diet', diuretics, paracentesis and, in a subset of patients, transjugular intrahepatic portosystemic shunt or liver transplantation.