may be signs of an abdominal abscess: these patients may have a high swinging fever, localised tenderness, a palpable mass and evidence of bowel obstruction (pain, vomiting and constipation with dehydration, abdominal distension and tenderness, and an empty rectum). Anal disease is common, including skin tags, fissures, fistulae and abscesses. Colonic involvement produces the same signs as ulcerative colitis.

• **Signs of complications:** these are similar to those of ulcerative colitis with the following exceptions: (i) *liver disease*—primary sclerosing cholangitis is less common; (ii) *osteomalacia* and *osteoporosis*, which may occur in patients with extensive terminal ileal involvement, results in bone tenderness and fracture; (iii) *signs of malabsorption;* (iv) *finger clubbing* is more common; (v) *signs of gastrointestinal malignancy* (small bowel or colonic carcinoma) are uncommon but the incidence is increased; (vi) the incidence of *gallstones and renal stones* is increased; (vii) *renal disease* due to pyelonephritis, hydronephrosis or very rarely secondary amyloidosis may occur.

The abdominal X-ray: a systematic approach

Interpretation of the plain radiograph requires knowledge of basic anatomy and pathological processes.

The soft-tissue density of the abdominal organs is similar to that of water. Therefore they are usually not visible unless outlined by fat or adjacent gas. For example, fluid-filled bowel is not visible, but the bowel walls are outlined by the contained gas.

Because of this intrinsic lack of contrast in the abdomen, radio-opaque contrast media are introduced to show up various organs. Barium meals, barium enemas, intravenous urograms and arteriograms are contrast studies.

Radiography

As with the chest X-ray, the name and date should be checked. The left and right sides should be easily distinguished by the stomach gas on the left and the triangular bulky soft tissue of the liver seen in the right hypochondrium.

Review of an abdominal X-ray

• **Boundaries:** diaphragm, psoas muscles, the extraperitoneal fat (‘flank lines’).

• **Bones:** lower ribs and costal cartilages, lumbar spine, pelvis.

• **Hollow viscera gas:** check gas outlining the stomach, small bowel and large bowel.

• **Solid organs:** size of liver, spleen and kidneys.

• **Pelvic organs:** bladder size.

• **Vascular:** aortic calcification.

• **Abnormalities:** renal or biliary calculi, dilated bowel, free peritoneal gas (Figure 6.38).

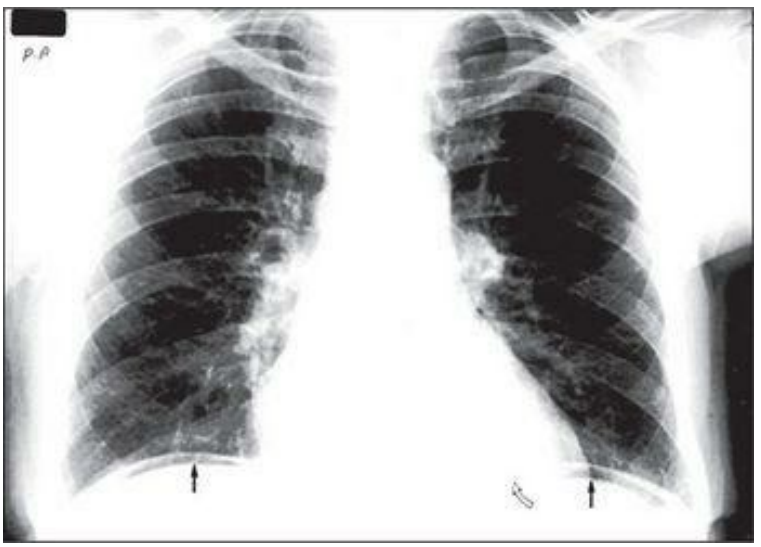


Figure 6.38 Free peritoneal gas
The erect chest X-ray is superior to an erect abdominal film for the demonstration of free gas. On the erect chest X-ray, free peritoneal gas is seen below the hemidiaphragm (black arrows). The free gas on the left must be distinguished from gas in the gastric fundus (open arrow). This free gas (black arrow) on the left is crescentic in shape because it outlines the spleen and lies at the apex of the hemidiaphragm. It indicates a perforation of a hollow abdominal viscus unless there has been recent surgery or penetrating trauma.

Bowel gas pattern

Supine films are taken in most conditions to show the distribution of the bowel gas. In patients with an acute abdomen, a horizontal beam film, usually an erect view, is also taken to show air–fluid levels.

With obstruction, there is an accumulation of fluid and gas proximally.

In inflammatory or ischaemic colitis, the swollen bowel mucosa will be outlined by gas ('thumb-printing').

Bowel dilatation

When an ileus (Figure 6.39) or obstruction (Figures 6.40 and 6.41) is present, it is possible to distinguish small- from large-bowel dilatation. The *large-bowel loops* are peripheral, few in number, have diameters greater than 5 cm, contain faeces, and have haustral margins that do not extend across the bowel lumen. In contrast, the *small-bowel loops* are central, multiple, between 3 and 5 cm in diameter, and do not contain faeces. Valvulae conniventes which extend completely across the bowel lumen are seen in the jejunal loops.

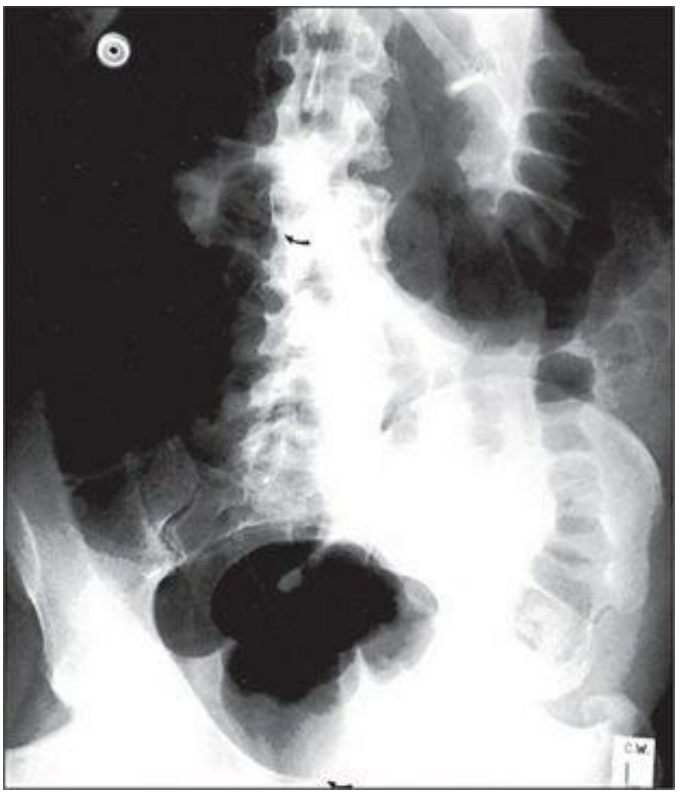


Figure 6.39 Generalised ileus.
The large bowel is filled with gas and is dilated, except in the descending colon. Dilated small bowel is also seen in the right hypochondrium (arrow). As gas is seen around to the rectum (arrow), mechanical obstruction is excluded.

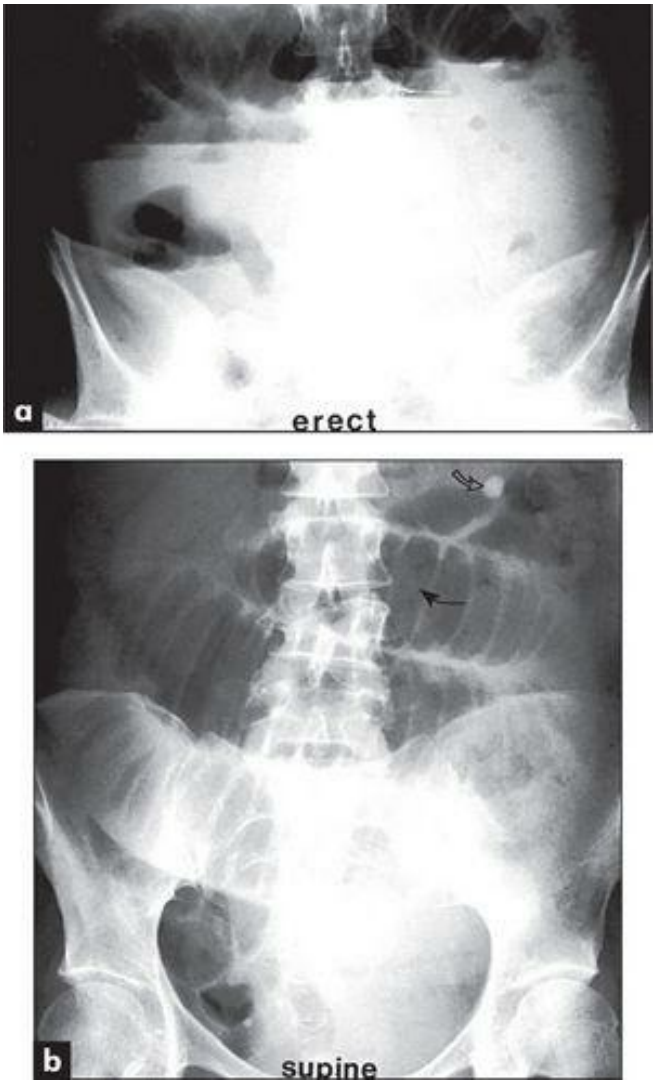
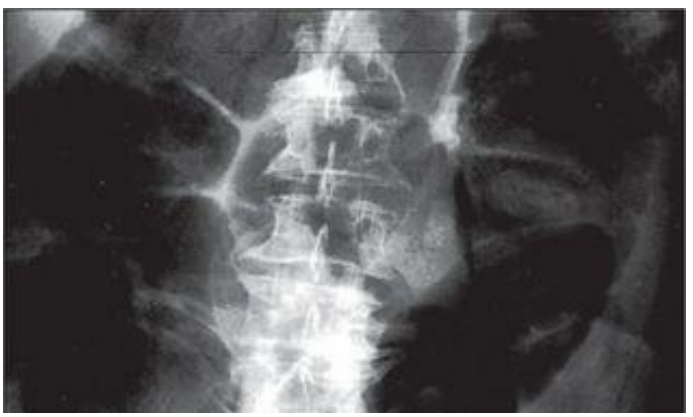


Figure 6.40 Small bowel obstruction
There is gross dilatation of the small bowel. It is recognised as small bowel from its central position and its transverse mucosal bands—the valvulae conniventes (black arrow). Air–fluid levels are seen on the erect view (a). The supine view (b) gives a better view of the distribution of the dilated loops. From the number and position of the displayed dilated loops, the obstruction would be at the level of the mid-small bowel. The round radio-opaque shadow in the left hypochondrium is a tablet (open arrow).



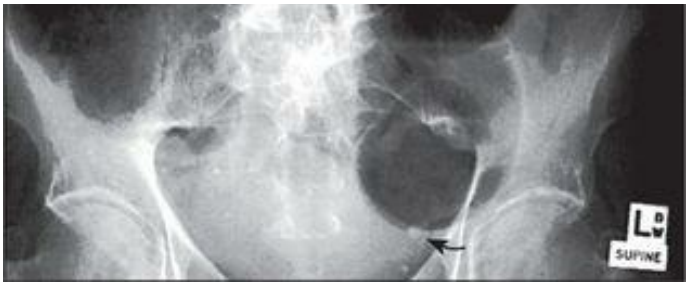


Figure 6.41 Large bowel obstruction
The large bowel is markedly distended around to the sigmoid colon, where it abruptly stops (arrow). The common causes of obstruction are carcinoma or diverticular stricture. The increased peristalsis occurring at the onset of obstruction can remove the gas and faeces distal to the obstruction. Therefore no gas is seen in this patient.

With gastric dilatation, the stomach may be massively enlarged and distended with air (Figure 6.42).

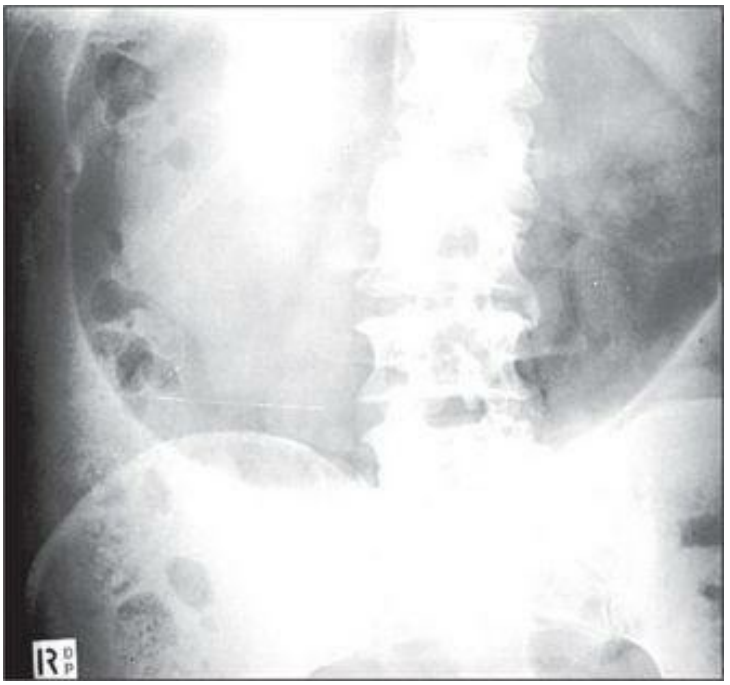


Figure 6.42 Gastric dilatation
The stomach is massively enlarged and distended with air. When this occurs acutely, prompt nasogastric aspiration is necessary. Mechanical obstruction due to a pyloric ulcer or carcinoma needs exclusion. Atonic dilatation is usually a postoperative complication, but may occur with diabetic coma, trauma, pancreatitis or hypokalaemia.

Calcification

Calcification shows up well against the grey, soft-tissue densities.

About 90% of renal stones are calcified (Figure 7.4, page 203), whereas only 10% of gallstones are calcified. To identify radiolucent gallstones, an ultrasound examination is the test of choice.

Calcification may be seen in the pancreas in chronic pancreatitis (Figure 6.43).

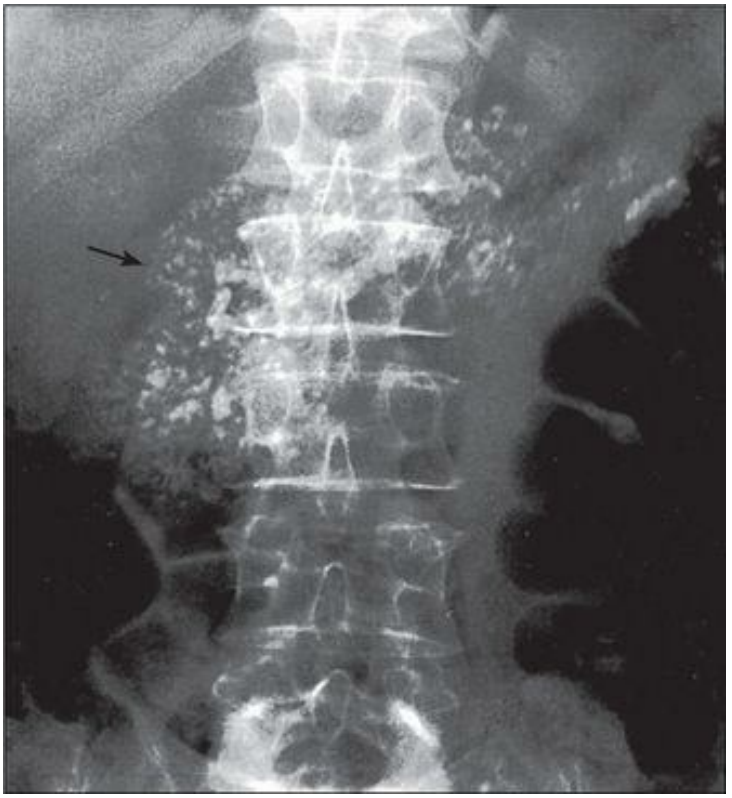


Figure 6.43 Pancreatic calcification
Stippled calcification is seen in the region of the pancreas (arrow), indicating chronic calcific pancreatitis. The most likely cause is alcohol excess.

Costal cartilage calcification is commonly seen in elderly patients, projected over the hypochondrial regions.

Calcification in the walls of an abdominal aortic aneurysm may be seen on a lateral abdominal film. Splenic and renal artery aneurysms are also often calcified.

Vascular calcification is often seen in the elderly.

Ascites

With accumulation of peritoneal fluid within the peritoneal cavity, the film looks generally grey and lacks detail. On the supine film the bowel loops float towards the middle of the abdomen.

Summary

The gastrointestinal examination: a suggested method (Figure 6.44)

As with the other systems this examination will usually be targeted. However it cannot be performed properly, even in a busy clinic, unless the patient lies down and removes sufficient clothing—if necessary in stages and with a chaperone.

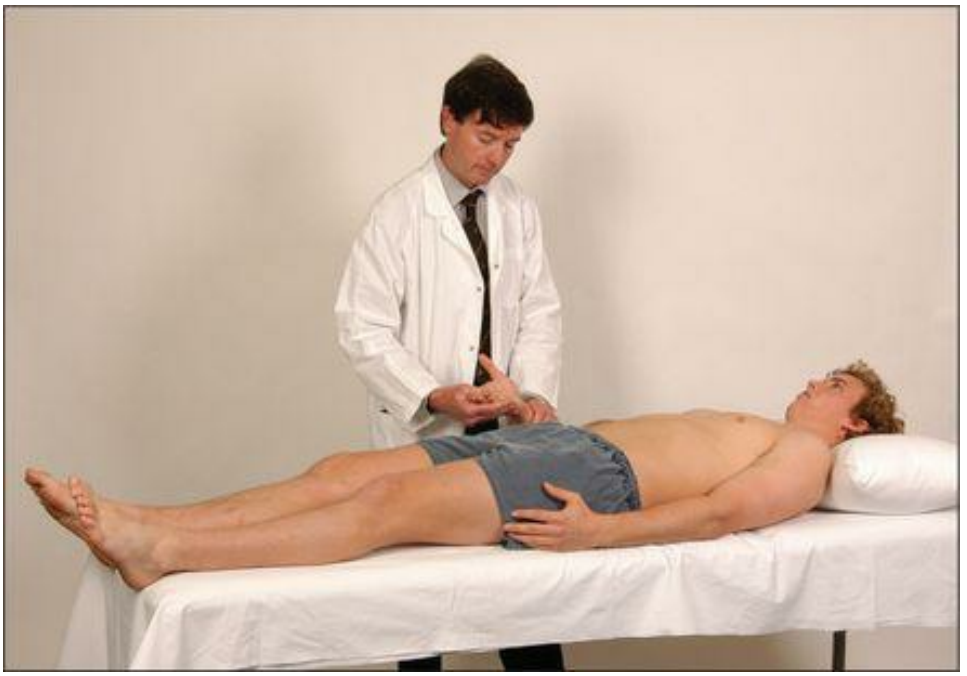


Figure 6.44 Gastrointestinal system
Lying flat (1 pillow)

1. **General inspection**
   Jaundice (liver disease)
   Pigmentation (haemochromatosis, Whipple's disease)
   Xanthomata (chronic cholestasis)
   Mental state (encephalopathy)

2. **Hands**

3. **Nails**
   - Clubbing
   - Leuconychia

   Palmar erythema
   Dupuytren's contractures (alcohol)

Arthropathy
Hepatic flap

4. **Arms**
   Spider naevi
   Bruising
   Wasting
   Scratch marks (chronic cholestasis)

5. **Face**
   Eyes
   - Sclerae: jaundice, anaemia, iritis
   - Cornea: Kayser-Fleischer rings (Wilson's disease)

   Parotids (alcohol)
   Mouth
   - Breath: fetor hepaticus
   - Lips: stomatitis, leucoplakia, ulceration, localised pigmentation (Peutz-Jeghers syndrome), telangiectasia (hereditary haemorrhagic telangiectasia)
   - Gums: gingivitis, bleeding, hypertrophy, pigmentation, Monilia
   - Tongue: atrophic glossitis, leucoplakia, ulceration

6. **Cervical/axillary lymph nodes**

7. **Chest**
   Gynaecomastia
   Spider naevi
   Body hair

8. Abdomen
   Inspect
   - Scars
   - Distension
   - Prominent veins—determine direction of flow (caput Medusae; inferior vena cava obstruction)
   - Striae
   - Bruising
   - Pigmentation
   - Localised masses
   - Visible peristalsis

   Palpate
   - Superficial palpation—tenderness, rigidity, outline of any mass
   - Deep palpation—organomegaly (liver, spleen, kidney), abnormal masses

abnormal masses
Roll onto right side (spleen)
Percuss
- Liver, spleen
- Ascites—shifting dullness

Auscultate
- Bowel sounds
- Bruits, hums, rubs

9. **Groin**
   Testes
   Lymph nodes
   Hernial orifices (standing up)

10. **Legs**
   Bruising
   Oedema
   Neurological signs (alcohol)

11. Other
   Rectal examination—inspect (fistulae, tags, blood, mucus), palpate (masses)
   Urine analysis (bile)
   Cardiovascular system (cardiomyopathy, cardiac failure, constrictive pericarditis)
   Temperature chart (infection)

Position the patient correctly with one pillow for the head and complete exposure of the abdomen. Look briefly at the **general appearance** and inspect particularly for signs of chronic liver disease.

Examine the **hands**. Ask the patient to extend his or her arms and hands and look for the hepatic flap. Look also at the nails for clubbing and for white nails, and note any palmar erythema or Dupuytren's contractures. The arthropathy of haemochromatosis may also be present. Look now at the **arms** for bruising, scratch marks and spider naevi.

Then go to the **face**. Note any scleral abnormality (jaundice, anaemia or iritis). Look at the corneas for Kayser-Fleischer rings. Feel for parotid enlargement; then inspect the mouth with a torch and spatula for angular stomatitis, ulceration, telangiectasiae and atrophic glossitis. Smell the breath for fetor hepaticus. Now look at the chest for spider naevi and in men for gynaecomastia and loss of body hair.

Inspect the **abdomen** from the side, squatting to the patient's level. Large masses may be visible. Ask the patient to take slow deep breaths and

Large masses may be visible. Ask the patient to take slow deep breaths and look especially for the hepatic, splenic and gallbladder outlines. Now stand up and look for scars, distension, prominent veins, striae, hernia, bruising and pigmentation.

Palpate lightly in each region for masses, having asked first if any area is particularly tender. This will avoid causing the patient pain and may also provide a clue to a site of possible pathology. Next palpate each region more deeply; then feel specifically for hepatomegaly and splenomegaly. If there is hepatomegaly, confirm this with percussion and estimate the span. If no spleen is felt, percuss over the left costal margin in the left anterior axillary line during complete expiration (dullness suggests splenomegaly). Always **roll** the patient onto the right side and palpate again if the spleen is not felt initially. Attempt now to feel the kidneys bimanually. Remember the important distinguishing features of a spleen as opposed to a kidney.

Percuss routinely for ascites. If the abdomen is resonant right out to the flanks, do not roll the patient over. Otherwise test for shifting dullness. This is performed by percussing away from your side of the bed until you reach a dull note. Then **roll** the patient towards you and, after waiting a minute or so, begin percussing again for resonance.

By **auscultation** note the presence of bowel sounds. Next auscultate briefly over the liver, spleen and renal areas, listening for bruits, hums and rubs.

Examine the **groin** next. Palpate for inguinal lymphadenopathy. Examine for hernias by asking the patient to stand and then cough. The testes must always be palpated. Now look at the legs for oedema and bruising. Neurological examination of the **legs** may be indicated if there are signs of chronic liver disease.

If the **liver** is enlarged or cirrhosis is suspected ask the patient to sit up to 45 degrees and estimate the jugular venous pressure. This will avoid missing constrictive pericarditis or chronic cardiac failure as a cause of liver disease, or haemochromatosis, which can cause a dilated cardiomyopathy. While the patient is sitting up, palpate in the supraclavicular fossae for **lymph nodes** and feel at the back for **sacral oedema**. If ascites is present, it is necessary to examine the chest for a pleural effusion. If malignant disease is suspected, examine all lymph node groups, the breasts and the lungs.

A **rectal** examination should always be considered and specimens of the patient's vomitus or faeces should be inspected, if available. Perform a **urinalysis** (for bilirubin and urobilinogen, and glucose) and check the temperature.

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1. Heidrix TR. Art and science of history taking in the patient with difficulty swallowing. *Dysphagia*. 1993;8:69-73. A very good review of the key historical features that must be obtained when a patient presents with trouble swallowing

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[a] Robert Milton Zollinger (b. 1903), American surgeon, and Edwin H Ellison (b. 1918), American physician. This syndrome is characterised by gastric acid hypersecretion, peptic ulceration and in 40% of cases diarrhoea, due to a gastrinoma (gastrin-secreting tumour). It was described in 1955.

[b] Harold Hirschsprung (1830–1916), physician, Queen Louise Hospital for Children, Copenhagen, described this disease in 1888. It had previously been described by Caleb Parry, English physician, in 1825.

[c] John Peutz (1886–1957), physician at St John's Hospital, The Hague, Holland, fi rst described this condition in 1921. Harold Jeghers (b. 1904), professor of medicine, Boston City Hospital, USA, described it in 1949.

[d] Henri Rendu (1844–1902), French physician. Frederick Weber (1863–1962), English physician. The condition was described in 1907.

[e] These changes were fi rst described by Terry in 1954 in association with cirrhosis. They are also found in patients with cardiac failure and become more common in normal people with age. In a patient under the age of 50, their presence indicates cirrhosis, heart failure or diabetes, with a likelihood ratio of 5.3.

[f] Baron Guillaume Dupuytren (1777–1835), Surgeon-in-Chief at the Hotel-Dieu in Paris. A cold, rude, ambitious and arrogant man, he was called'the Napoleon of surgery'. He saw 10,000 private patients a year.

[g] From Greek a, 'not' and sterixis, 'fi xed position'.

[h] Primary biliary cirrhosis (PBC) is an uncommon chronic non-suppurative destructive cholangitis of unknown aetiology; 90% of affected patients are female.

[i] Spider naevi were fi rst described in 1867 by Erasmus Wilson, an English physician

[j] Campbell de Morgan (1811–76), London surgeon. He was one of the 300 original Fellows of the Royal College of Surgeons. He described his spots in 1872 and believed them to be a sign of cancer (which they are

not).

[k] Pierre Bitot (1822–88) described this in 1863.

[l] Bernhard Kayser (1869–1954), German ophthalmologist, described these rings in 1902. Bruno Fleischer (1848–1904), German ophthalmologist, described them in 1903.

[m] Jean Descemet (1732–1810), professor of surgery and anatomy, Paris. He described the membrane in 1785.

[n] Samuel Alexander Wilson (1878–1937), London neurologist at Queen Square. His colleagues there included Gowers and Hughlings Jackson. He described his disease in 1912 in his MD thesis. He also described the glabellar tap sign in Parkinson's disease, which is sometimes called Wilson's sign. He did not, however, describe the Kayser-Fleischer rings.

[o] Burrill Bernard Crohn (1884–1983), American gastroenterologist at Mount Sinai Hospital, New York, described this disease in 1932. It had previously been described by Morgagni (1682–1771) in 1769.

[p] Charles Émile Troisier (1844–1919), professor of pathology in Paris, described this sign in 1886.

[q] Franz von Leydig (1821–1908), Bonn anatomist and zoologist, Germany.

[r] Sister Joseph of St Mary's Hospital, Rochester, Minnesota, described this sign to Dr William Mayo (1861–1939) of the Mayo Clinic.

[s] Thomas S Cullen (1869–1953), professor of gynaecology at Johns Hopkins University, Baltimore, originally described this sign as an indication of a ruptured ectopic pregnancy.

[t] George Grey-Turner (1877–1951), surgeon, Royal Victoria Infi rmary, Newcastle-on-Tyne, England.

[u] Bernhard Riedel (1846–1916), German surgeon, described this in 1888.

[v] John Murphy (1857–1916), American surgeon, professor of surgery at Rush Medical College, Chicago, described this in 1912.

[w] Ludwig Courvoisier (1843–1918), professor of surgery, Switzerland, described this principle in 1890. He was a keen natural historian and wrote 21 papers on entomology.

[x] AH Curtis described hepatic adhesions associated with pelvic

AH Curtis described hepatic adhesions associated with pelvic inflammatory disease in 1930, while T Fitz-Hugh described right upper abdominal acute gonococcal peritonitis in 1934. However, this syndrome was actually first described by C Stajano in 1920.

[y] Jean Cruveilhier (1791–1874), professor of pathological anatomy, Paris, who had been Dupuytren's registrar, and Paul von Baumgarten (1848–1928), German pathologist.

[z] Franz Hesselbach (1759–1816), professor of surgery, Würzburg, described this triangle bounded by the inguinal ligament, the inferior epigastric artery and the rectus abdominis.

[aa] George Blumer (1858–1940), professor of medicine at Yale, in 1909 described cancer in the pouch of Douglas forming a shelf-like structure.

[bb] It can be useful to perform the rectal examination with the patient supine and the head of the bed elevated; this allows the intra-abdominal contents to descend and a bimanual examination (using the opposite hand to compress the lower abdomen) is possible.

[cc] The middle one of three transverse folds of mucous membrane in the rectum, described in 1830 by John Houston (1802–45), Irish surgeon.

[dd] Charles McBurney (1845-1913), New York surgeon described his sign to the New York Surgical Society in 1889.

[ee] Thorkild Rovsing (1862–1937), professor of surgery, Copenhagen.

[ff] Henry Plummer (1874–1936), physician at the Mayo Clinic, described the syndrome in 1912; Porter Vinson (1890–1959), physician, Medical College Virginia, described the syndrome in 1919.

[gg] Donald Paterson (1863–1939), Cardiff otolaryngologist, and Adam Brown-Kelly (1865–1941), Glasgow otolaryngologist, described this syndrome in 1919.

[hh] Friedrich Albert Zenker (1825–1898), Munich pathologist.

[ii] George Hoyt Whipple (1878–1976), Baltimore pathologist, described this rare disease characterised by diarrhoea, arthralgia, central nervous system signs and pigmentation. He shared the 1934 Nobel Prize for work on liver treatment in anaemia and coined the word *thalassaemia*.

Chapter 7

The genitourinary system

> You know my method. It is founded upon the observation of trifles.
> *Sherlock Holmes, created by Sir Arthur Conan Doyle (1859–1930)*

Despite their very different functions the male and female genital and urinary symptoms are intimately associated anatomically and usually assessed together.

The genitourinary history

Presenting symptoms (Table 7.1)

These may include a change in the appearance of the urine, abnormalities of micturition, suprapubic or flank pain or the systemic symptoms of renal failure. Some patients have no symptoms but are found to be hypertensive or to have abnormalities on routine urinalysis or serum biochemistry. Others may feel unwell but not have localising symptoms (*Questions box 7.1*). The major renal syndromes are set out in Table 7.2.

Table 7.1 Genitourinary history

Major symptoms
Change in appearance of urine, e.g. haematuria Change in urine volume or stream • Polyuria • Nocturia • Anuria • Decrease in stream size • Hesitancy • Dribbling • Urine retention

- Strangury
- Pis-en-deux—double-voiding (incomplete bladder emptying)
- Incontinence of urine

Renal colic

Dysuria (painful micturition)

Frequency, urgency

Fever, loin pain

Urethral discharge

Symptoms suggestive of chronic renal failure (uraemia)
- Oliguria, nocturia, polyuria
- Anorexia, a metallic taste, vomiting, fatigue, hiccup, insomnia
- Itch, bruising, oedema

Menses
- Age of onset
- Regularity
- Last period (date)
- Dysmenorrhoea, menorrhagia

Erectile dysfunction

Loss of libido

Infertility

Pregnancies: number and any complications

Urethral or vaginal discharge

Genital rash

Genital rash

Questions box 7.1

Questions to ask the patient with renal failure or suspected renal disease

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. How did your kidney problems begin? Have you had tiredness, the need to pass urine at night (nocturia) or loss of appetite?

2. Was the kidney trouble thought to be brought on by any medications you were taking (e.g. non-steroidal anti-inflammatory drugs, ACE inhibitors/angiotensin receptor blockers, or contrast used for an X-ray procedure)?

3. Were you told there was inflammation of the kidneys (glomerulonephritis) or protein in the urine?

4. Have you had kidney infections recently or as a child?

5. Have you had kidney stones or urinary obstruction?

6. Have you passed blood in the urine?—Urinary tract malignancy

7. Have you had a biopsy of your kidney? Do you know the result?

8. Have you had diabetes or high blood pressure?

9. Have you had cardiovascular disease or peripheral vascular disease?

10. Have you had kidney surgery or removal of a kidney, or have you been told you have only one functioning kidney?

11. Is there a history in the family of enlarged kidneys and high blood pressure?—Polycystic kidneys

12. Have you had problems with rashes or arthritis?— Systemic lupus erythematosus, scleroderma

13. Have you had problems with swelling or shortness of breath?—Fluid retention

14. Have you been told how bad your kidney function is and whether you may need dialysis one day?

15. Are you taking medications to help the kidney function?

16. What tablets and medications (including over-the-counter products, herbal remedies, etc.) are you taking?

Table 7.2 The major renal syndromes

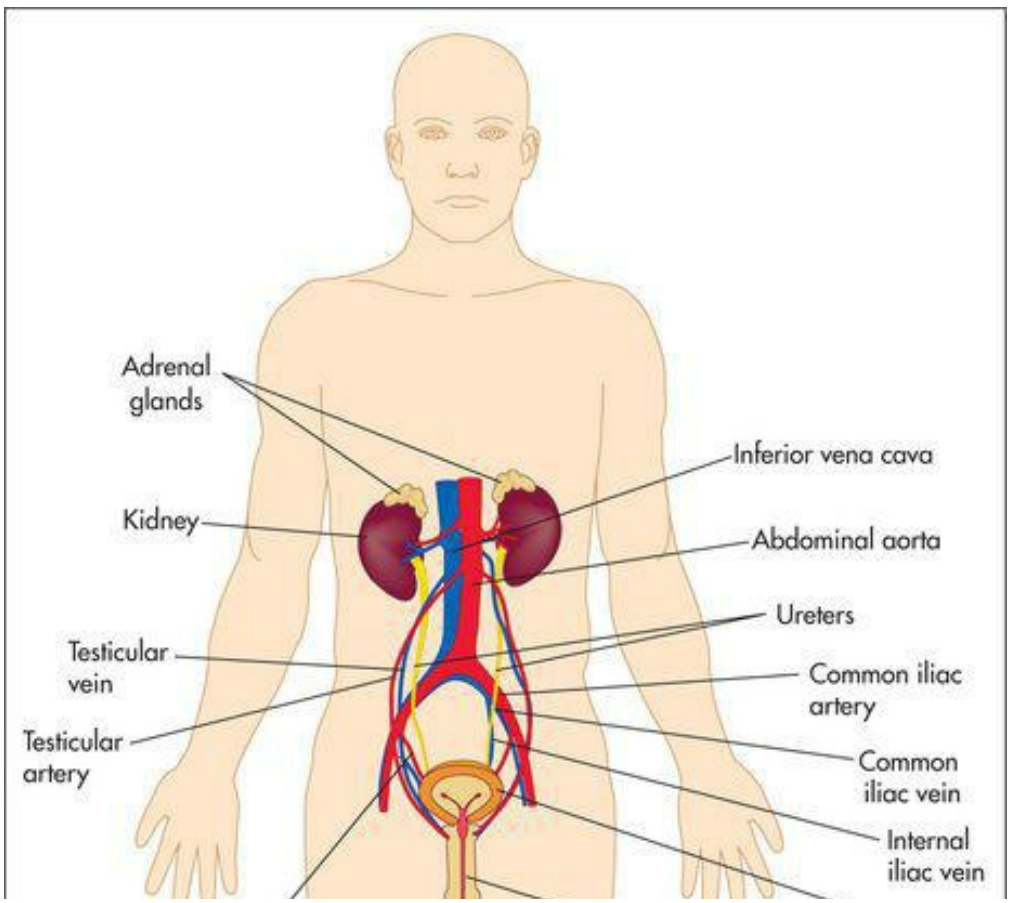
Name	Definition	Example
Nephrotic	Massive proteinuria	Minimal change disease
Nephritic	Haematuria, renal failure	Post-streptococcal glomerulonephritis
Tubulointerstitial nephropathy	Renal failure, mild proteinuria	Analgesic nephropathy
Acute renal failure*	Sudden fall in function, rise in	Acute tubular necrosis

failure	creatinine	
Rapidly progressive renal failure	Fall in renal function, over weeks	Malignant hypertension or 'crescentic' glomerulonephritis
Asymptomatic urinary abnormality	Isolated haematuria, or mild proteinuria	Immunoglobulin A nephropathy

* Newly defined as acute kidney injury, AKI; Levin A, Warnock D, Mehta R, Kellum J, Shah S, Melitoris B, Ronco C. Improving outcome for AKI. *Am J Kidney Dis* 2007; 50(1):1–4.

Examination anatomy

Figure 7.1 shows an outline of the anatomy of the urinary tract. Figure 7.2 shows the arterial supply of the kidneys as demonstrated on a CT renal angiogram and Figure 7.3 shows the outline of the renal collecting system. Problems with function can arise in any part, from the arterial blood supply of the kidneys, the renal parenchyma, the ureters and bladder (including their innervation), to the urethra.



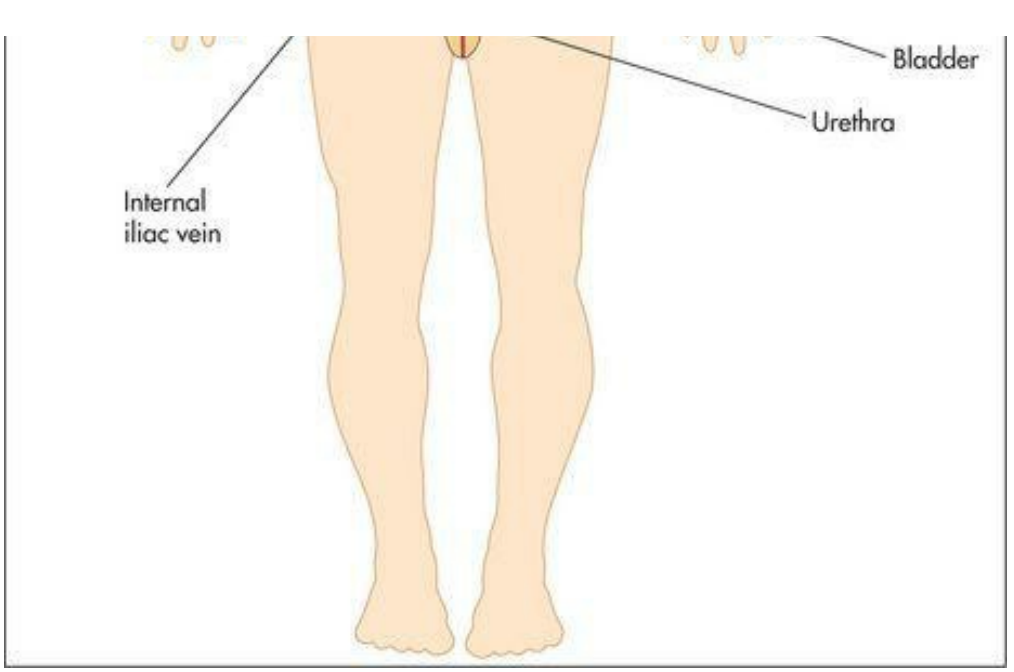


Figure 7.1 The anatomy of the kidneys and urinary tract

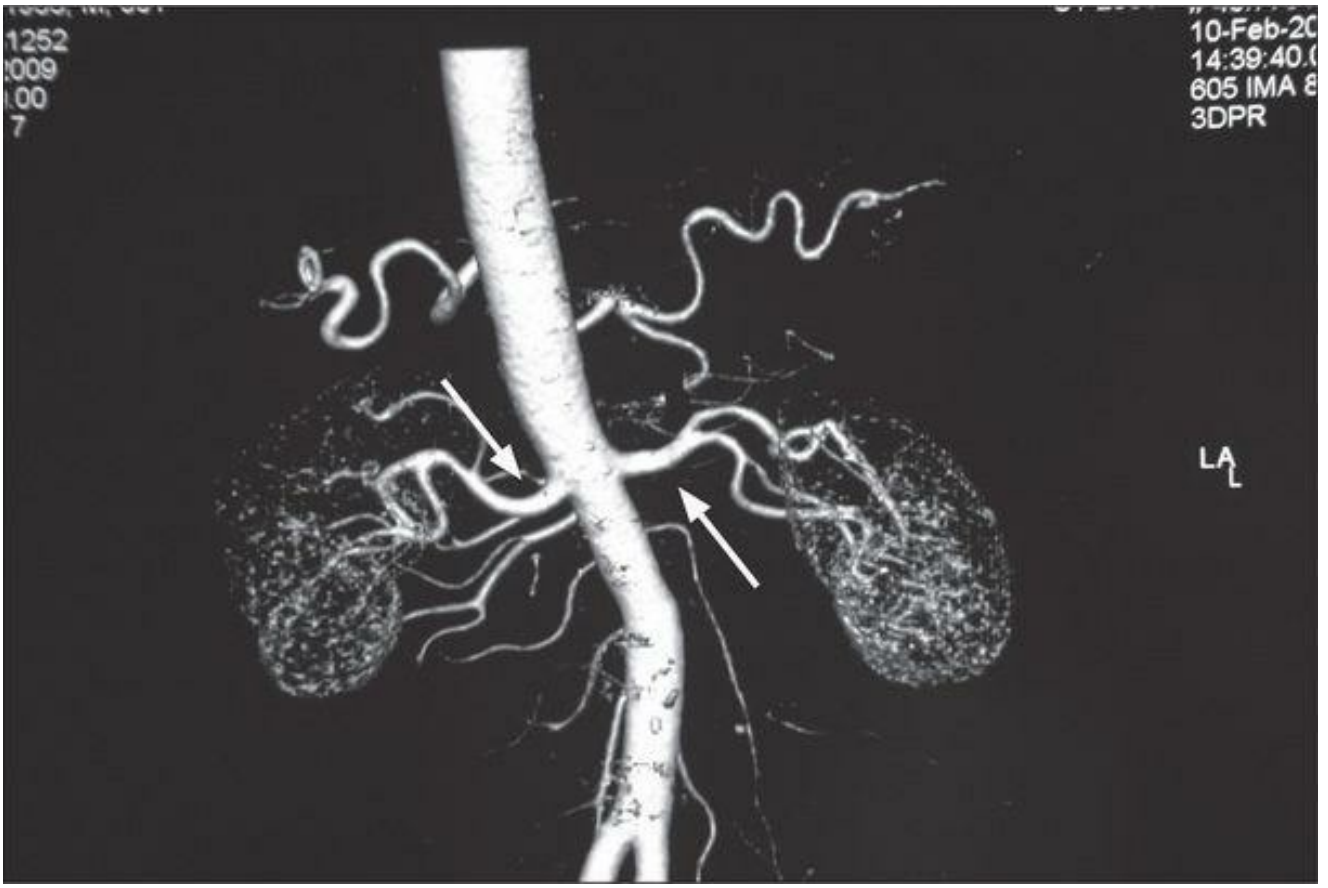


Figure 7.2 CT angiogram showing the origins and course of the renal arteries (large arrows) from the abdominal aorta; the left and right inferior phrenic arteries are visible arising superiorly

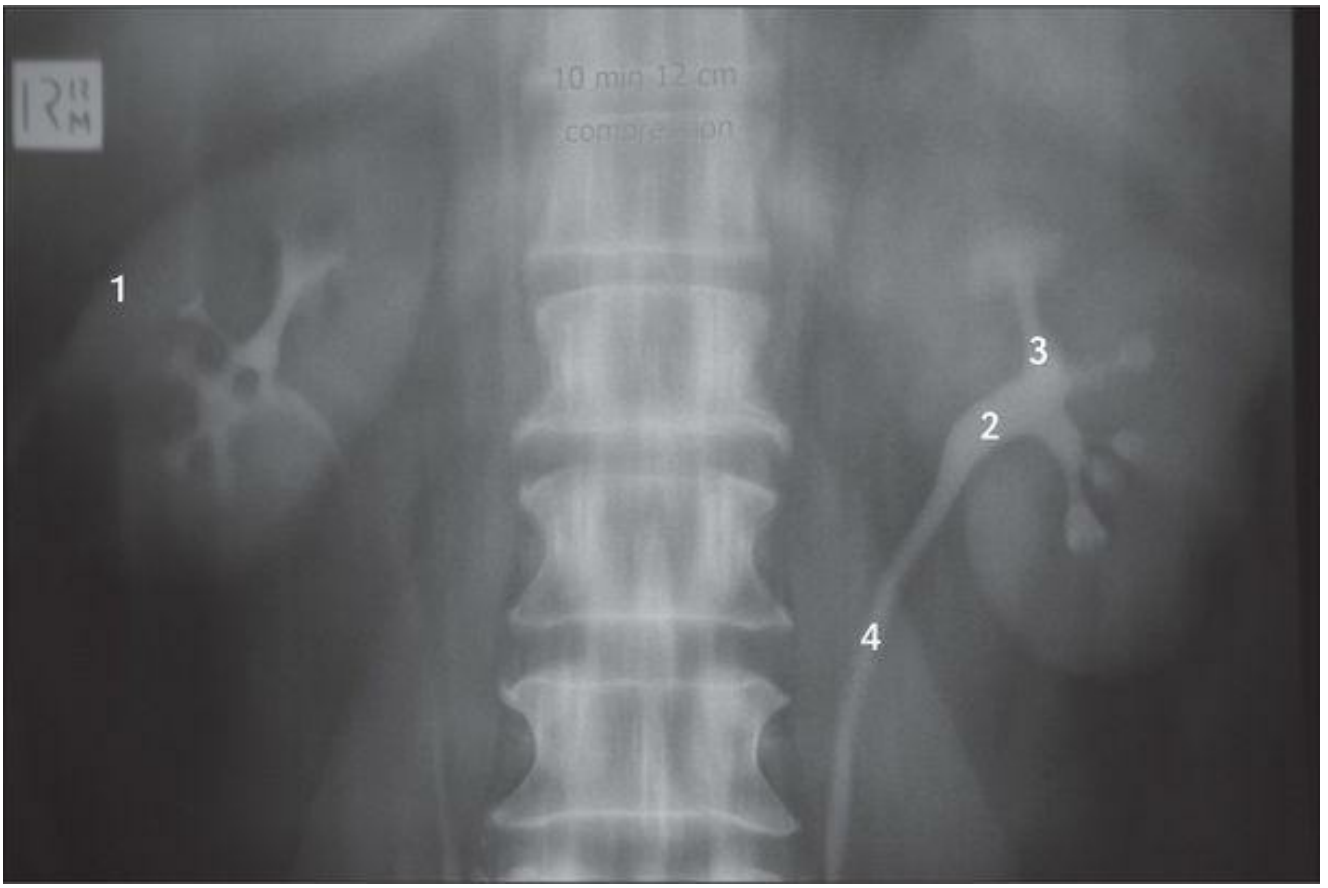


Figure 7.3 Outline of the renal collecting system
This intravenous pyelogram shows the outline of the kidneys (1), the renal pelves (2) and the calyces (3) and ureters (4).

Basic male and female reproductive anatomy is shown in Figures 7.9 (page 216) and 7.13 (page 218).

Change in appearance of the urine

Some patients present with discoloured urine. A red discoloration suggests haematuria (blood in the urine).[1] Urethral inflammation or trauma, or prostatic disease, can cause haematuria at the beginning of micturition which then clears, or haematuria only at the end of micturition (Table 7.3). Patients with porphyria can have urine that changes colour on standing. Consumption of certain drugs (e.g. rifampicin) or of large amounts of beetroot and, rarely, haemoglobinuria (due to destruction of red blood cells and release of free haemoglobin) can cause red discoloration of the urine (page 212). Patients with severe muscle trauma may have myoglobinuria as a result of muscle breakdown. This can also cause red discoloration. Foamy, tea-coloured or brown urine may be a presenting sign of nephrosis or kidney failure. It is worth noting that the colour of the urine is not a reliable guide to its

worth noting that the colour of the urine is not a reliable guide to its concentration.

Table 7.3 Haematuria

1 Favours urinary tract infection Dysuria Fever (prostatitis, pyelonephritis) Suprapubic pain (cystitis) Moderate flank or back pain (pyelonephritis)
2 Favours renal calculi Severe loin pain
3 Favours source not glomerular Clots in urine
4 Favours blood not in urine Menstruation
5 Favours immunoglobulin A nephropathy Multiple episodes over months
6 Favours trauma Recent indwelling urinary catheter or procedure Recent back or abdominal injury

7 Favours bleeding disorder
Use of anticoagulant drugs

Urinary tract infection (UTI)

This condition includes both upper urinary tract (renal) infection and lower UTI (mostly the bladder—cystitis). Possibly as many as 50% of lower UTIs also involve the kidneys. Renal infection may be difficult to distinguish clinically from lower UTIs but is a more serious condition and more likely to involve systemic complications such as septicaemia.

Urinary tract infection is much more common in women than in men, but there are a number of risk factors for the disease (Table 7.4). It can be strongly suspected on the basis of the patient's symptoms.[2] These include: dysuria (pain or stinging during urination), frequency (need to pass small amounts of urine frequently), haematuria, and loin (more suggestive of upper UTI) or back pain. Physical examination may reveal fevers, rigors, lower abdominal discomfort and loin pain when the renal angle is balloted posteriorly. The latter findings are more suggestive of complicated UTI or pyelonephritis. The presence of a vaginal discharge is against the diagnosis. Elderly patients with a urinary tract infection often present with confusion and few other symptoms or signs. A UTI in a male or frequent, relapsing or recurrent UTI in a female suggests an anatomical abnormality and requires urological evaluation.

Table 7.4 Risk factors for urinary tract infection (UTI)

Female sex
Coitus
Pregnancy
Diabetes
Indwelling urinary catheter

Previous UTI
Lower urinary tract symptoms of obstruction

Urinary obstruction

Urinary obstruction is a common symptom in elderly men and is most often due to prostatism (now called lower urinary tract symptoms—LUTS) or bladder outflow obstruction. The patient may have noticed hesitancy (difficulty starting micturition—urination), followed by a decrease in the size of the stream of urine and terminal dribbling of urine. Strangury (recurrently, a small volume of bloody urine is passed with a painful desire to urinate each time) and pis-en-deux/double-voiding (the desire to urinate despite having just done so) may occur.[3] When obstruction is complete, overflow incontinence of urine can occur. Obstruction is associated with an increased risk of urinary infection.

Renal calculi can cause ureteric obstruction (Figure 7.4). The presenting symptom here, however, is usually severe colicky or constant loin or lower quadrant pain which may radiate down towards the symphysis pubis or perineum or testis (renal colic). Urinary obstruction can be a cause of acute renal failure (kidney injury) (Table 7.5).

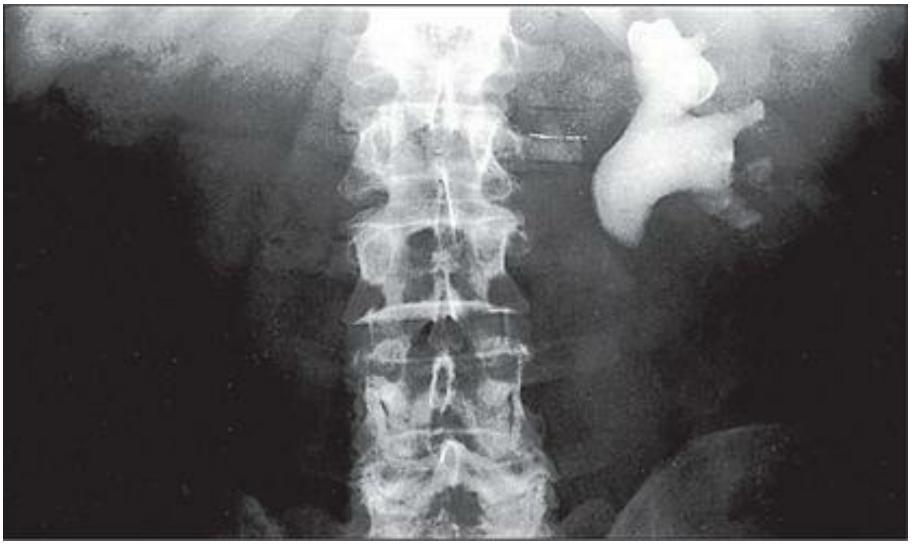


Figure 7.4 Renal calculus
Phleboliths (calcifications related to blood vessels) are rounded opacities seen in the pelvis below the level of the ischial spines, whereas ureteric calculi lie above this level, in the line of the ureters. The large staghorn calculus shown here is occupying the calyces of the left renal pelvis. This type of calculus is almost always radio-opaque. An abdominal ultrasound examination (IVPs are almost never performed in this context today) is necessary to check whether there is an obstruction at the pelviureteric junction. In general, 90% of renal calculi

whether there is an obstruction at the pelviureteric junction. In general, 90% of renal calculi are radio-opaque and visible on plain X-ray films. A significant proportion of patients presenting with renal colic due to calcium calculi have hyperparathyroidism.

Table 7.5 Causes of acute renal failure (acute kidney injury, AKI*)

a. Onset over days
This is defined as a rapid deterioration in renal function severe enough to cause accumulation of waste products, especially nitrogenous wastes, in the body. Usually the urine flow rate is less than 20 mL/hour or 400 mL/day, but occasionally it is normal or increased (high-output renal failure).
Prerenal
Fluid loss: blood (haemorrhage), plasma or water and electrolytes (diarrhoea and vomiting, fluid volume depletion)

Hypotension: myocardial infarction, septicaemic shock, drugs
Renovascular disease: embolus, dissection or atheroma
Increased renal vascular resistance: hepatorenal syndrome
Renal
Acute-on-chronic renal failure (precipitated by infection, fluid volume depletion, obstruction or nephrotoxic drugs)—see Table 7.7
Acute renal disease: • e.g. primary or secondary glomerulonephritis, connective tissue diseases
Acute tubular necrosis secondary to: • ischaemia (hypovolaemia) • toxins and drugs (such as aminoglycoside, antibiotics, radiocontrast material, heavy metals) • rhabdomyolysis, haemoglobinuria
Tubulointerstitial disease: • e.g. drugs (such as proton pump inhibitors, sulfonamides, cyclosporin A), urate or calcium deposits, phosphate, oxalate, crystal nephropathy
Vascular disease: • e.g. vasculitis, scleroderma
Myeloma
Acute pyelonephritis (rare)
Postrenal (complete urinary tract obstruction)
Urethral obstruction: • e.g. calculus or blood clot, sloughed papillae, trauma, phimosis or paraphimosis

of paraphimosis
At the bladder neck: • e.g. calculus or blood clot, prostatic hypertrophy or cancer
Bilateral ureteric obstruction: • intraureteric, e.g. blood clot, pyogenic debris, calculi • extra-ureteric, e.g. retroperitoneal fibrosis (due to radiation, methysergide or idiopathic), retroperitoneal/pelvic tumour or surgery, uterine prolapse
b. Causes of rapidly progressive renal failure (onset over weeks to months)
Urinary tract obstruction
Rapidly progressive glomerulonephritis
Bilateral renal artery stenosis (may be precipitated by angiotensin-converting enzyme inhibitor or angiotensin receptor blocker use)
Multiple myeloma
Scleroderma renal crisis
Malignant hypertension
Haemolytic uraemic syndrome

Note: Anuria may be due to urinary obstruction, bilateral renal artery occlusion, rapidly progressive (crescentic) glomerulonephritis, renal cortical necrosis or a renal stone in a solitary kidney.

* Levin A, Warnock D, Mehta R, Kellum J, Shah S, Melitoris B, Ronco C. Improving outcome for AKI. *Am J Kidney Dis* 2007; 50(1):1–4.

Urinary incontinence

This is the inability to hold urine in the bladder voluntarily. It is not a consequence of normal ageing alone. The problem can occur transiently with urinary tract infections, delirium, excess urine output (e.g. from the use of diuretics), immobility (because patients are unable to reach the toilet), urethritis or vaginitis, or stool impaction.

Causes of established urinary incontinence include: (i) *stress*

incontinence (instantaneous leakage after the stress of coughing or after a sudden rise in intra-abdominal pressure of any cause)—this problem is more common in women due to vaginal deliveries or an atrophic vaginal wall postmenopause causing a hypermobile urethra; (ii) urge incontinence (*overactivity of the detrusor muscle*) which is characterised by an intense urge to urinate and then leakage of urine in the absence of cough or other stressors—this occurs in men and women; (iii) *detrusor underactivity*—this is rare and is characterised by urinary frequency, nocturia and the frequent leaking of small amounts of urine from neurological disease; (iv) overflow incontinence (*urethral obstruction*)—this occurs typically in men with disease of the prostate, and is characterised by dribbling incontinence after incomplete urination; and (v) a *vesico/urethral fistula*—a complication of obstructed labour.

Chronic renal failure (chronic kidney disease)

The clinical features of chronic renal failure can be deduced in part by considering the normal functions of the kidneys.

1. Failure of excretory function leads to accumulation of numerous 'uraemic' toxins, hence the widely used term 'uraemia'. This frequently leads to malaise, lethargy, anorexia, malnutrition and hiccups.

2. Urinary concentrating ability may be lost early, leading to the risk of dehydration; nocturia can be an early symptom.

3. Various factors such as the failure to excrete sodium may lead to hypertension.

4. Damage to the renal tubules may lead to sodium loss and hypotension.

5. Excretion of potassium depends in part on urine volume. Hyperkalaemia usually becomes a problem when a patient is oliguric (passes less than 400 mL urine/day) and may occur when taking potassium-sparing diuretics or agents that promote potassium retention (ACE inhibitors, angiotensin receptor blockers, non-steroidal anti-inflammatory drugs).

6. Failure of acid excretion leads to metabolic acidosis.

7. Disordered mineral and bone metabolism (abnormal levels of calcium, phosphorus, parathyroid hormone [PTH] and vitamin D) may lead to abnormalities in bone and vascular or soft-tissue calcification.[4]

8. Failure to secrete erythropoietin leads to normochromic normocytic anaemia.

9. Alterations in the metabolism of those medications which are excreted by the kidneys.

Adequacy of renal function is defined by the glomerular filtration rate (GFR). This is the volume of blood filtered by the kidneys per unit of time. The normal range is 90–120 mL/min. The GFR is estimated by calculating the clearance of creatinine (a normal breakdown product of muscle) from the blood. The serum creatinine and urea levels also provide a measure of accumulation of uraemic toxins and therefore of renal function. Most laboratories now provide an estimated GFR (eGFR) measurement calculated from the serum creatinine and the patient's age and sex.

A new definition and classification of chronic kidney disease (CKD) has been introduced. CKD is defined as kidney damage or GFR <60 mL/min/1.73 m^2 for 3 months or more, irrespective of cause.[5] Further kidney disease has been divided into 6 groups according to GFR (Table 7.6). These allow planning of investigations and treatment that might slow progression of the disease.

Table 7.6 Classification of chronic kidney disease by glomerular filtration rate (GFR)

Stage	Description	GFR (mL/min/1.73 m^3)
–	Increased risk for chronic kidney disease (e.g. diabetes, hypertension)	>90
1	Kidney damage but normal GFR	>90
2	Kidney damage and mild GFR reduction	60–89
3	Moderate reduction in GFR	30–59
4	Severe reduction in GFR	15–29
5	Kidney failure	<15

A uraemic patient may present with anuria (defined as failure to pass more than 50 mL urine daily), oliguria (less than 400 mL urine daily), nocturia (the need to get up during the night to pass urine) or polyuria (the passing of abnormally large volumes of urine) (page 297). Nocturia may be an indication of failure of the kidneys to concentrate urine normally, and polyuria may indicate complete inability to concentrate the urine.

The more general symptoms of renal failure include anorexia, vomiting, fatigue, hiccups and insomnia. Pruritus (a general itchiness of the skin), easy bruising and oedema due to fluid retention may also be present. Other symptoms indicating complications include bone pain, fractures because of renal bone disease, and the symptoms of hypercalcaemia (including anorexia, nausea, vomiting, constipation, increased urination, mental confusion) because of tertiary (or primary) hyperparathyroidism.[a] Patients may also present with the features of pericarditis, hypertension, cardiac failure, ischaemic heart disease, neuropathy or peptic ulceration.

Find out whether the patient is undergoing dialysis and whether this is haemodialysis or peritoneal dialysis. There are a number of important questions that must be asked of dialysis patients (*Questions box 7.2*).

Questions box 7.2

Questions to ask the dialysis patient

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem

problem.

1. What fluid restriction have you been recommended?

2. Have phosphate-binding drugs been prescribed? When do you take these relative to meals?

3. Do you use haemodialysis or peritoneal dialysis? Do you do this at home? How many times a week?

4. Have you had abdominal pain or fever recently?—Peritonitis related to peritoneal dialysis

5. Have there been any problems with haemodialysis, such as low blood pressure, or with the fistula used for haemodialysis? Have there been any problems with peritonitis with peritoneal dialysis?

6. How much weight do you gain between each haemodialysis?

7. Do you still pass any urine? If so, how much?

8. Are you on a renal transplant list or have previously had a transplant?

9. Do you follow recommended dietary restrictions?

10. What other medications do you take?

11. Have you had heart or blood vessel problems?

12. Have you had overactive parathyroid glands or parathyroid surgery?

Ask about any complications that have occurred, including recurrent peritonitis with peritoneal dialysis or problems with vascular access for haemodialysis.

A common form of treatment for renal failure is renal transplantation. A patient may know how well the graft is functioning, and what the most recent renal function tests have shown. Find out whether the patient knows of rejection episodes, how these were treated, and if there has been more than one renal transplant. It is necessary to ascertain if there have been any problems with recurrent infection, urine leaks or side-effects of treatment. Long-term problems with immunosuppression may have occurred, including the development of cancers, chronic nephrotoxicity (e.g. from cyclosporin or tacrolimus), obesity and hypertension from steroids, or recurrent infections. The patient should be aware of the need to avoid skin exposure to the sun and women should know that they need regular Papanicolaou[b] (Pap) smears for cancer surveillance.

Menstrual and sexual history

A menstrual history should always be obtained. The menarche or date of the first period is important (page 296). The regularity of the periods over the preceding months or years and the date of the last period are both relevant. The patient may complain of dysmenorrhoea (painful menstruation) or menorrhagia (an abnormally heavy period or series of periods).

Vaginal discharge can occur in patients with infections of the genital tract. Sometimes the type of discharge is an indication of the type of infection present. The history of the number of pregnancies and births is relevant: gravidity refers to the number of times a woman has conceived, while parity refers to the number of babies delivered (live births or stillbirths). One should also ask about any complications that occurred during pregnancy (e.g. hypertension).

The sexual history is also relevant.[6] Ask about contraceptive methods and the possibility of pregnancy.[7] Ask men about erectile dysfunction (impotence). Erectile dysfunction is defined as inability to achieve or maintain a satisfactory erection, for more than 3 months. Most causes are organic (neurogenic [e.g. diabetes] or vascular, or drug related [e.g. beta-blockers, thiazide diuretics]), with a slow onset and loss of morning erections in older men.

Treatment

A detailed drug history must be taken. Note all the drugs, including steroids and immunosuppressants, and their dosages. In patients with decreased renal function, the dosages of many drugs that are cleared by the kidneys must be adjusted. The patient with chronic renal failure should be well informed about the need for protein, phosphate, potassium, fluid or salt restriction. Patients with urinary tract infections may have had a number of courses of antibiotics. Treatment of hypertension should be documented. Certain drugs should be used with caution. For example, non-steroidal anti inflammatory drugs can worsen renal function or cause CKD.

Past history

Find out whether there have been previous or recurrent urinary tract infections or renal calculi. There may have been operations to remove urinary tract stones, or pelvic surgery may have been performed because of

urinary incontinence in women or prostatism in men. The patient may know about the previous detection of proteinuria or microscopic haematuria at a routine examination. Glomerulonephritis will usually have been diagnosed by renal biopsy, a procedure that is often a memorable event. History of other urological disorders and results for prior urological evaluations are important. Histories of diabetes mellitus or gout are relevant, as these diseases may lead to renal complications. It is most important to find out about hypertension, because this may not only cause renal impairment but is also a common complication of renal disease. Similarly, a history of acute kidney failure episodes, history of cancer treated with chemotherapy or radiotherapy, severe allergic reactions, and exposures to nephrotoxic substances are all relevant. A history of childhood enuresis (bedwetting) beyond the age of three years may be relevant: it can be associated with vesicoureteric reflux and subsequent renal scarring.

Ask about previous myocardial infarction, congestive heart failure or valvular heart disease and about liver disease, especially hepatitis and about other systemic infections. Renovascular disease is more likely if there is a history of vascular disease elsewhere, such as myocardial ischaemia or cerebrovascular disease. In elderly patients, specific questions relating to ingestion of Bex or Vincent's powders may suggest a diagnosis of analgesic nephropathy. This is particularly important as these patients require surveillance for urothelial malignancy in addition to managing their renal impairment.

Social history

Occupational and travel exposure to toxins or infections, tobacco exposure, and excess consumption of alcohol are important.

Patients with chronic renal failure may have many social problems. There may be a need for access to equipment at home for dialysis. Whom does the patient contact if there is a problem with home dialysis? One must ask detailed questions to find out how the patient and his or her family is coping with the chronic illness and its complications. Has the patient been able to work? Find out how well-informed the patient is about the transplant, if this has been the treatment. Also find out what sort of support the patient has obtained from relatives and friends.

Family history

Some forms of renal disease are inherited. Polycystic kidney disease, for example, is an autosomal-dominant condition. Ask about diabetes and

hypertension in the family. A family history of deafness and renal impairment suggests Alport's[c] syndrome, a hereditary form of nephritis. A family history of kidney disease of any type is a risk factor for development of CKD.

The genitourinary examination

A set examination of the genitourinary system is not routinely performed. However, if renal disease is suspected or known to be present then certain signs must be sought. These are mostly the signs of chronic renal failure (uraemia) and its causes (Table 7.7). On the other hand, examination of the male genitalia or female pelvis is part of the routine general examination.

Table 7.7 Causes of chronic renal failure (chronic kidney disease, CKD[*])

This is defined as a severe reduction in nephron mass over a variable period of time resulting in uraemia.[†]
1 Glomerulonephritis
2 Diabetes mellitus
3 Systemic vascular disease

4 Analgesic nephropathy
5 Reflux nephropathy
6 Hypertensive nephrosclerosis
7 Polycystic kidney disease
8 Obstructive nephropathy
9 Amyloidosis
10 Renovascular disease
11 Atheroembolic disease
12 Hypercalcaemia, hyperuricaemia, hyperoxaluria
13 Autoimmune diseases
14 Haematological diseases
15 Toxic nephropathies
16 Granulomatous diseases
17 Chronic tubulointerstitial nephritis
Clinical features suggesting that renal failure is chronic rather than acute
Small kidney size (except with polycystic kidneys, diabetes, amyloidosis and myeloma)
Renal bone disease
Anaemia (with normal red blood cell indices)
Peripheral neuropathy

* Levey A, Eckarrdt K, Tsukanoto Y, Levin A, Coresh J, Rossert J, Zeeuw W, Hostetter T, Lamiere N, Eknoyan G. Definition and classification of CKD: A position statement of KDIGO. *Kidney Int* 2005; 67:2089–2100.

† Note that this list is not all-inclusive.

General appearance

The general inspection remains crucial. Look for *hyperventilation*, which may indicate an underlying metabolic acidosis. *Hiccupping* may be present and can be an ominous sign of advanced uraemia. There may be the ammoniacal fish breath (‘*uraemic fetor*’) of kidney failure. This musty smell is not easy to describe but once detected is easily remembered. Patients with

is not easy to describe but once detected is easily remembered. Patients with chronic renal failure commonly have a sallow complexion (a dirty brown appearance or '*uraemic tinge*'). This may be due to impaired excretion of urinary pigments (urochromes) combined with anaemia. The skin colour may be from slate grey to bronze, due to iron deposition in dialysis patients who have received multiple blood transfusions, but these signs are becoming less frequent with the use of exogenous erythropoietin. In terminal renal failure, patients become drowsy and finally sink into a coma due to nitrogen or toxin retention. Twitching due to myoclonic jerks, and tetany and epileptic seizures due to neuromuscular irritability or a low serum calcium level, occur late in renal failure. Over-vigorous correction of acidosis (e.g. with bicarbonate infusions) may also precipitate seizures and coma. There may be typical skin nodules related to calcium phosphate deposition.

It is essential to assess the state of fluid balance in all patients with renal disease. Severe fluid-volume depletion can be a cause of acute renal failure and can cause precipitous decompensation in patients with chronic renal failure. Conversely, volume excess can result from intravenous infusions of fluid used in an attempt to correct acute renal failure, resulting in pulmonary oedema. Patients should be weighed regularly as an objective measure of their fluid status.

The distinctive ketone-like smell of a UTI may be apparent. There may be evidence of urinary incontinence on the patient's clothing.

The hands

The *nails* should be inspected: look for leuconychia; Muehrcke's nails[d] refer to paired white transverse lines near the end of the nails; these occur in hypoalbuminaemia (e.g. nephrotic syndrome).[8] A single transverse white band (Mees' lines,[e] Figure 7.5) may occur in arsenic poisoning, as well as in renal failure. Half-and-half nails (distal nail brown or red, proximal nail pink or white) are also seen in chronic renal failure. Non-pigmented indented transverse bands can occur with any cause of a catabolic state (Beau's lines[f]).

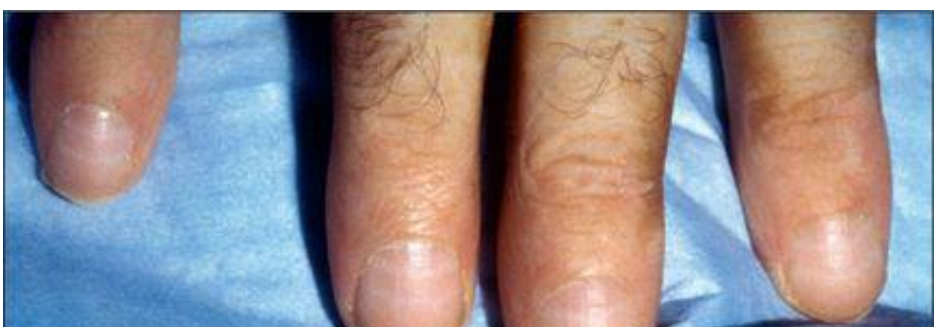


Figure 7.5 Mees' lines

From McDonald FS, ed., Mayo Clinic images in internal medicine, with permission. ©Mayo Clinic Scientific Press and CRC Press.

Anaemia is common and causes palmar crease pallor. There are a number of causes of anaemia in patients with chronic renal failure, including poor nutrition (especially folate deficiency), blood loss, erythropoietin deficiency, haemolysis, bone marrow depression and the chronic disease state.

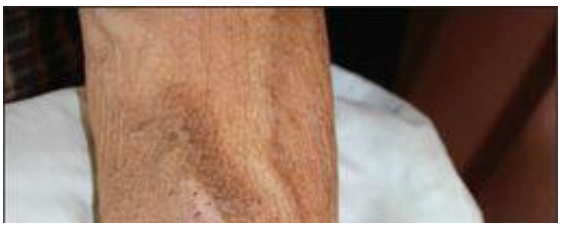
Asterixis may be present in terminal chronic renal failure.

Inspect the wrist and forearms for scars and palpate for surgically created arteriovenous fistulae or shunts, used for haemodialysis access. There is a longitudinal swelling and a palpable continuous thrill present over the fistula. There may be scars from previous thrombosed shunts or carpal tunnel syndrome surgery present on either side. Look for signs of the carpal tunnel syndrome.

The arms

*Bruisi*ng occurs because of nitrogen retention which causes impaired prothrombin consumption, a defect in platelet factor III, and abnormal platelet aggregation in chronic renal failure. *Skin pigmentation* is common, reflecting a failure to excrete urinary pigments. *Scratch marks and excoriations*, due to uraemic pruritus, often associated with hyperphosphataemia, may be present. This occurs commonly and can be extremely debilitating. *Uraemic frost* is a fine white powder present on the skin where very high concentrations of urea have precipitated out of the sweat in terminal chronic renal failure; it is very rare. Evidence of *vasculitis*, which can cause renal disease, should also be sought.

An arterio-venous fistula may be visible and palpable in the forearm. A working fistula has a characteristic buzzing feel. This is used for vascular access for dialysis (Figure 7.6).



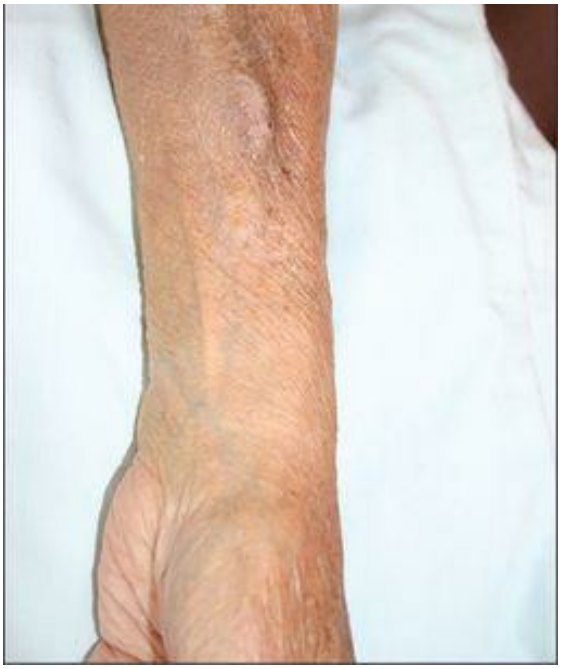


Figure 7.6 Arterio-venous fistula in the forearm of a haemodialysis patient

Look for signs of *peripheral neuropathy* in the limbs. Sensory impairment is more marked than motor impairment initially. Myopathy and bone tenderness can also occur.

The face

The eyes are important; look for signs of *anaemia* and, rarely, *jaundice* (retention of nitrogenous wastes can cause haemolysis). *Band keratopathy* is a calcium deposition beneath the corneal epithelium in line with the interpalpebral fissure—it is due to secondary or tertiary hyperparathyroidism, or excessive replacement of calcium in patients with chronic renal failure.

The mouth should always be examined. A uraemic *fetor* may be present. This is an ammoniacal, musty odour due to breakdown of urea to ammonia in the saliva. Mucosal *ulcers* can occur as there is a decrease in saliva flow, and patients with chronic renal failure are prone to infection (e.g. thrush), due to decreased acute inflammatory responses as a result of nitrogen retention. Transplant patients treated with calcineurin inhibitors (cyclosporin and tacrolimus) frequently develop *gingival hyperplasia* (thickening of the gums).

The presence of a *rash* or skin tethering may indicate an underlying connective tissue disease such as systemic lupus erythematosus or systemic sclerosis.

The presence of hearing aids may be consistent with Alport's syndrome (hereditary nephritis often with sensorineural hearing loss and eye disease of the retina or cornea).

the retina or cornea).

The neck

Carefully check the *jugular venous pressure* to help assess the intravascular volume status. Auscultate for *carotid artery bruits*; these provide a clue that there may be generalised atherosclerotic disease (which can cause renal artery stenosis or complicate chronic renal failure). Look for signs of previous *jugular vein puncture* due to previous vascular access insertion ('vascath') for haemodialysis. Surgical scars from previous *parathyroidectomy* performed for management of tertiary hyperparathyroidism may be present.

The chest

Examine the heart and lungs. In chronic renal failure there may be *congestive cardiac failure* due to fluid retention, and *hypertension* as a result of sodium and water retention or excess vasoconstrictor activity or both. Signs of *pulmonary oedema* may also be present due to uraemic lung disease (a type of non-cardiogenic pulmonary oedema associated with typical 'bat's wing' pattern on chest X-ray; see Figure 4.61, page 99), volume overload or uraemic cardiomyopathy.

Pericarditis, which can be fibrinous or haemorrhagic in chronic renal failure, is secondary to retained metabolic toxins and can cause a pericardial effusion; there may be a pericardial rub or signs of cardiac tamponade. Lung infection is also common due to the immunosuppression present from the chronic renal failure itself or as a result of treatment.

The abdominal examination

Abdominal examination is performed as described on page 194. However, particular attention must be paid to the following.

Inspection

The presence of a Tenckhoff catheter (peritoneal dialysis catheter) should be noted. It is important to look for nephrectomy *scars* (see Figure 6.18, page 165). These are often more posterior than one might expect. It may be necessary to roll the patient over and look in the region of the loins. Renal transplant scars are usually found in the right or left iliac fossae. A

transplanted kidney may be visible as a bulge under the scar, as it is placed in a relatively superficial plane. Peritoneal dialysis results in small scars from catheter placement in the peritoneal cavity; these are situated on the lower abdomen, at or near the midline.

The abdomen may be distended because of large polycystic kidneys or *ascites* (as a result of the nephrotic syndrome, or peritoneal dialysis fluid).

Inspect the scrotum for masses and genital oedema.

Palpation

Particular care is required here so that renal masses (Table 7.8) are not missed. Remember that an enlarged kidney usually bulges forwards, while perinephric abscesses or collections tend to bulge backwards. Transplanted kidneys in the right or left iliac fossa may be palpable as well. In polycystic kidney disease, hepatomegaly from hepatic cysts may be found (Table 7.9). Feel for the presence of an enlarged bladder. Also palpate for an abdominal aortic aneurysm. In the patient with abdominal pain, renal colic should be suspected if there is renal tenderness (positive LR 3.6) or loin tenderness (positive LR 27.7).[9]

Table 7.8 Renal masses

1 Unilateral palpable kidney
Renal cell carcinoma
Hydronephrosis or pyonephrosis
Xanthogranulomatous pyelonephritis
Polycystic kidneys (with asymmetrical enlargement)
Normal right kidney or solitary kidney
Acute renal vein thrombosis (unilateral)
Acute pyelonephritis

Renal abscess Compensatory hypertrophy of single functioning kidney
2 Bilateral palpable kidneys Polycystic kidneys Hydronephrosis or pyonephrosis bilaterally Renal cell carcinoma bilaterally Diabetic nephropathy (early) Nephrotic syndrome (Table 7.12) Infiltrative disease, e.g. amyloid, lymphoma Acromegaly Bilateral renal vein thrombosis

Table 7.9 Adult polycystic kidney disease

If you find polycystic kidneys, remember these very important points.
1 Take the blood pressure (75% have hypertension).
2 Examine the urine for haematuria (due to haemorrhage into a cyst) and proteinuria (usually less than 2 g/day).
3 Look for evidence of anaemia (due to chronic renal failure) or polycythaemia (due to high erythropoietin levels). Note that the

haemoglobin level is higher than expected for the degree of renal failure.
4 Note the presence of hepatomegaly or splenomegaly (due to cysts). These may cause confusion when one is examining the abdomen.
5 Tenderness on palpation may indicate an infected cyst.
Note: Subarachnoid haemorrhage occurs in 3% of patients with polycystic kidney disease due to rupture of an associated intracranial aneurysm. As polycystic kidney disease is an autosomal-dominant condition, all family members should also be assessed.

Balloting

From the French word meaning *to shake about*, balloting is an examination technique for palpating the kidney by attempting to flick it forward. One hand is placed under the renal angle and the examiner's fingers flick upwards while the other hand, placed anteriorly in the right or left upper quadrant, waits to feel the kidney move upwards and then float down again (Figure 7.7).

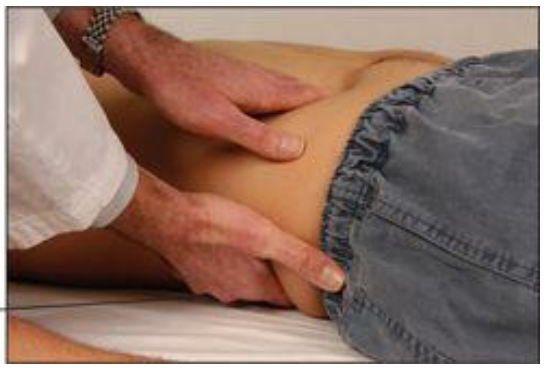


Figure 7.7 Balloting the kidneys

Percussion

This is necessary to confirm the presence of ascites by examining for shifting dullness. Also percuss for an enlarged bladder. Obesity and ascites make direct percussion of the bladder difficult. This is an opportunity to attempt

auscultatory percussion.[10] The diaphragm of the stethoscope is placed just above the border of the symphysis pubis and direct percussion of the abdominal wall performed, starting at the subcostal margin in the middle line. There is a sudden increase in loudness when the upper border of the bladder is reached. It is even possible to estimate the volume of urine in the bladder by this method. An upper border less than 2 cm from the stethoscope suggests a fairly empty bladder, while an upper border more than 8 cm higher corresponds to a urine volume of between 750 mL and a litre.

Auscultation

The important sign here is the presence of a *renal bruit*. Renal bruits are best heard above the umbilicus, about 2 cm to the left or right of the midline. Listen with the diaphragm of the stethoscope over both these areas. Next ask the patient to sit up, and listen in both flanks. The presence of a systolic and diastolic bruit is important. A diastolic component makes the bruit more likely to be haemodynamically significant. Its presence suggests renal artery stenosis due to fibromuscular dysplasia or atherosclerosis. Approximately 50% of patients with renal artery stenosis will have a bruit. In a patient with hypertension that is difficult to control, the presence of a systolic/diastolic abdominal bruit has a positive LR for renal artery stenosis of over 40.[9] On the other hand, if only a soft systolic bruit is audible, at least half these patients do not have any significant renal artery stenosis. In such cases the aorta or splenic artery may be the source of the sound. The absence of hypertension makes the diagnosis of renal artery stenosis less likely. The occurrence of unexplained pulmonary oedema of sudden onset (‘flash’ pulmonary oedema) in a patient with renal impairment and hypertension makes a diagnosis of renal artery stenosis more likely.

Rectal and pelvic examination

Here the presence of prostatomegaly[11,12] in men and a frozen pelvis from cervical cancer in women is important, as this may be a cause of urinary tract obstruction and secondary renal failure.

The back

Strike the vertebral column gently with the base of the fist to elicit bony tenderness. This may be due to renal osteodystrophy from osteomalacia, secondary hyperparathyroidism or multiple myeloma. Back pain in the

context of renal failure should always raise the possibility of an underlying paraproteinaemia.

Gentle use of the clenched fist to strike the patient in the renal angle is known as Murphy's kidney punch (Figure 7.8) and is designed to elicit renal tenderness in patients with renal infection. Similar information may be gained from more gentle balloting of the renal angle when the patient lies supine. Look also for sacral oedema in a patient confined to bed, particularly if the nephrotic syndrome or congestive cardiac failure is suspected. The presence of ulcerations of the toes suggests atheroembolic disease.

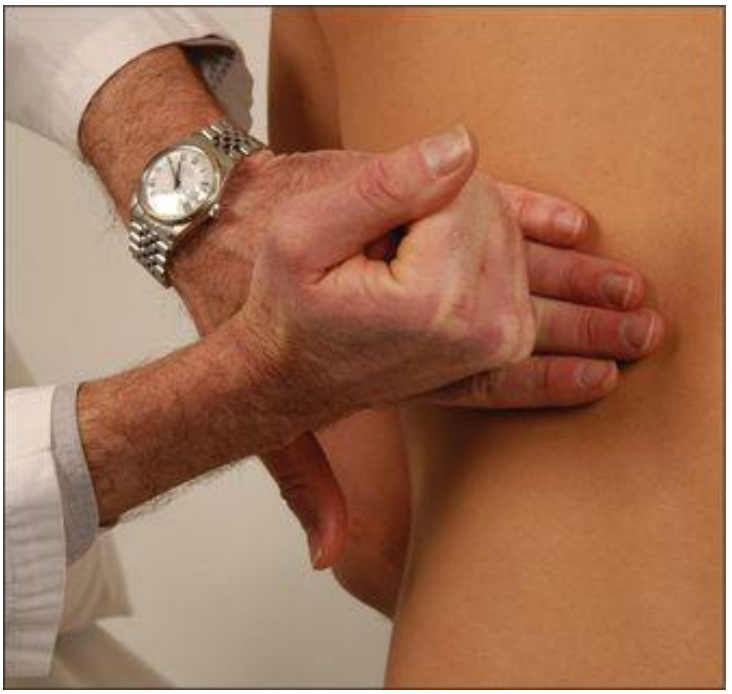


Figure 7.8 Murphy's kidney punch (not too hard)

The legs

The important signs here are oedema, purpura (page 226), livedo reticularis (a red-blue reticular pattern from vasculitis or atheroembolic disease), pigmentation, scratch marks and signs of peripheral vascular disease. Examination for peripheral neuropathy and myopathy is indicated, as in the arms. Gouty tophi or the presence of gouty arthropathy may very occasionally provide an explanation for the patient's renal failure (although secondary uric acid retention is common with chronic renal failure, it rarely causes clinical gout).

The blood pressure

It is of the utmost importance to take the blood pressure in every patient with renal disease. This is because hypertension can be the cause of renal disease or one of its complications. Test for postural hypotension, as hypovolaemia may precipitate acute renal failure.

The fundi

Examination of the fundi is important. Look especially for Keith-Wegerer hypertensive changes and diabetic changes. Diabetes can be a cause of chronic renal failure.

The urine

> The ghosts of dead patients that haunt us do not ask why we did not employ the latest fad of clinical investigation; they ask why did you not test my urine?
> *Sir Robert Hutchison (1871–1960)*

This valuable fluid must not be discarded in any patient in whom a renal, diabetic, gastrointestinal or other major system disease is suspected.

Colour

Look at the colour of the urine (Table 7.10).

Table 7.10 Some causes of urine colour changes

Colour	Underlying causes
Very pale or colourless	Dilute urine (e.g. overhydration, recent excessive beer consumption, diabetes insipidus, post-obstructive diuresis)
Yellow-orange	Concentrated urine (e.g. dehydration)
	Bilirubin
	Tetracycline, anthracene, sulfasalazine, riboflavin, rifampin
Brown	Bilirubin
	Nitrofurantoin, phenothiazines; chloroquine, senna, rhubarb (yellow to brown or red)
Pink	Beetroot consumption
	Phenindione, phenolphthalein (laxatives), uric acid crystalluria (massive)
Red	Haematuria, haemoglobinuria, myoglobinuria (may also be pink, brown or black)
	Porphyrins, rifampicin, phenazopyridine, phenytoin, beetroot
Green	Methylene blue, triamterene
Black	Severe haemoglobinuria
	Methyldopa, metronidazole, unipenem
	Melanoma, ochronosis; porphryins, alkaptonuria (red

	to black on standing)
White/milky	Chlyuria

Transparency

Phosphate or urate deposits can occur normally and produce white (phosphate) or pink (urate) cloudiness.

Fainter cloudiness may be due to bacteria. Pus, chyle or blood can cause a more turbid appearance.

Smell

A mild ammoniacal smell is normal. A urinary tract infection causes a fishy smell, and antibiotics can sometimes be smelt in the urine, as can asparagus.

Specific gravity

A urinometer, which is a weighted float with a scale, is used to measure specific gravity. The depth to which the float sinks in the urine indicates the specific gravity, which is read off the scale on the side. The specific gravity can also be estimated by dipstick methods.

Water has a specific gravity of 1, and the presence of solutes (especially heavy solutes such as glucose or an iodine contrast medium) in urine increases the specific gravity. The normal range is 1.002 to 1.025. A consistently low specific gravity suggests chronic renal failure (as there is failure of the kidneys to concentrate the urine) or diabetes insipidus (where there is a deficiency of antidiuretic hormone resulting in passage of a large volume of dilute urine). A high specific gravity suggests fluid volume depletion, or diabetes mellitus with the presence of large amounts of glucose in the urine.

There is a rough correlation between the specific gravity of the urine and its osmolarity. For example, a specific gravity of 1.002 corresponds to an osmolarity of 100 mOsm/kg, while a specific gravity of 1.030 corresponds to 1200 mOsm/kg.

Chemical analysis

A chemical reagent colour strip allows simultaneous multiple analyses of pH, protein, glucose, ketones, blood, nitrite, specific gravity, presence of leucocytes, bile and urobilinogen. The strip is dipped in the urine and colour changes are measured after a set period. The colours are compared with a chart provided. It should be noted that specific gravity by dipstick is pH dependent and insensitive to nonionised molecules, and therefore correlates poorly with urine osmolality.

pH

Normal urine is acid, except after meals when for a short time it becomes alkaline (the alkaline tide). Measuring the pH of urine is helpful in a number of critical circumstances. Sometimes the urine has to be made alkaline for therapeutic purposes, such as treating myoglobinuria or recurrent urinary calculi due to uric acid or cystine. Distal renal tubular acidosis should be suspected if the early morning urine is consistently alkaline and cannot be acidified. Urinary tract infections with ureasplitting organisms, such as *Proteus mirabilis*, can also cause an alkaline urine which, in turn, favours renal struvite stone formation.

Protein

The colours are compared with a chart provided. The strip tests give only a semi-quantitative measure of urinary protein (+ to ++++) and, if positive, must be confirmed by other tests. It is very important to note that the dipstick is sensitive to albumin but not to other proteins. A reading of + of proteinuria may be normal, as up to 150 mg of protein a day is lost in the urine. Causes of abnormal amounts of protein in the urine are listed in Tables 7.11 and 7.12. Chemical dipsticks do not detect the presence of Bence-Jones proteinuria[g] (immunoglobulin light chains).

Table 7.11 Causes of proteinuria

Persistent proteinuria
1. Renal disease
Almost any renal disease may cause a trace of proteinuria. Moderate or large amounts tend to occur with glomerular disease (Table 7.12).
2. No renal disease (functional)
Exercise
Fever
Hypertension (severe)
Congestive cardiac failure
Burns
Blood transfusion
Postoperative
Acute alcohol abuse
Orthostatic proteinuria
Proteinuria that occurs when a patient is standing but not when recumbent is called orthostatic proteinuria. In the absence of abnormalities of the urine sediment, diabetes mellitus, hypertension or reduced renal function, this entity probably has a benign prognosis.

Table 7.12 Nephrotic syndrome