the testicle to twist around upon its cord. This anatomical variation is almost invariably present on the other testicle, so after surgically correcting the torted testis, orchidopexy should always be done on the other side also.

When a testicle torts, time is of the essence. One study suggests that the testicular salvage rate is 90% if detorsion occurs less than 6 hours after the onset of symptoms, but this drops to 50% after 12 hours and <10% after 24 hours.[3]

Torsion of a testicular appendage

Torsion of one of the testicular appendages (most commonly the hydatid of Morgagni) accounts for 60% of cases of acute scrotal pain in children[1] (torsion of the testis 30%, and epididymo-orchitis, idiopathic and other causes comprise the remaining 10%). Vestigial remnants to the testis or the epididymis are present in 90% of the male population.[1] Torsion of a testicular appendage has a similar presentation to that of torsion of the testis but is less severe (see FIG. 103.1).

Torsion of a testicular appendage can be diagnosed by the appearance of a dark blue nodule at the upper pole of the testis called the ‘blue dot sign’ (provided that it is not masked by an associated hydrocele).[3] Surgical exploration may be needed to distinguish this from torsion of the testis.

Torsion of the testis versus epididymo-orchitis

With torsion of the testicle there is pain of sudden onset, described as a severe, aching, sickening pain in the groin that may be accompanied by nausea and vomiting. In epididymo-orchitis the attack usually begins with malaise and fever and is often associated with a urinary infection. The testicle soon becomes swollen and acutely tender; however, elevation and support of the scrotum usually relieves pain in this condition (Prehn sign), whereas it tends to increase pain with a torsion. A comparison of the clinical presentations is given in TABLE 103.2 .

Table 103.2 Clinical presentations of torsion of testis and acute epididymo-orchitis

	Torsion of testis	Epididymo-orchitis
Typical age	Early teens, average range 5–15 years	Young adults Elderly
Onset	Usually sudden but can be gradual	Gradual
Severity of pain	Very severe	Moderate

Associated symptoms	Vomiting Groin pain Possibly abdominal pain	Fever ± Dysuria
Examination of scrotum	Very tender and red Testis high and transverse Scrotal oedema Possibly an acute hydrocele	Swollen, tender and red; can be tender on rectal examination Possibly an acute hydrocele
Effect of gentle scrotal elevation	No change to pain or worse pain	Relief of pain
Investigations	Technetium-99m scan (if available, time permits and diagnosis doubtful)	Leucocytosis Possibly pyobacteria of urine

Radiology as a diagnostic aid

Doppler ultrasound is useful in distinguishing a cystic scrotal lump (such as a hydrocele) from a solid tumour. Its use to distinguish between a torsion and epididymo-orchitis is controversial as it cannot reliably detect changes that are diagnostic of early torsion. Since the investigation can involve unnecessary delay in treatment it is generally not recommended. A technetium-99m scan can differentiate between the two conditions: in torsion the testis is avascular, while it is hyperaemic in epididymo-orchitis. Again, the risk is the delay.

At surgery the testicle is untwisted and if viable an orchidopexy is performed. A gangrenous testicle is removed (see FIG. 103.2).

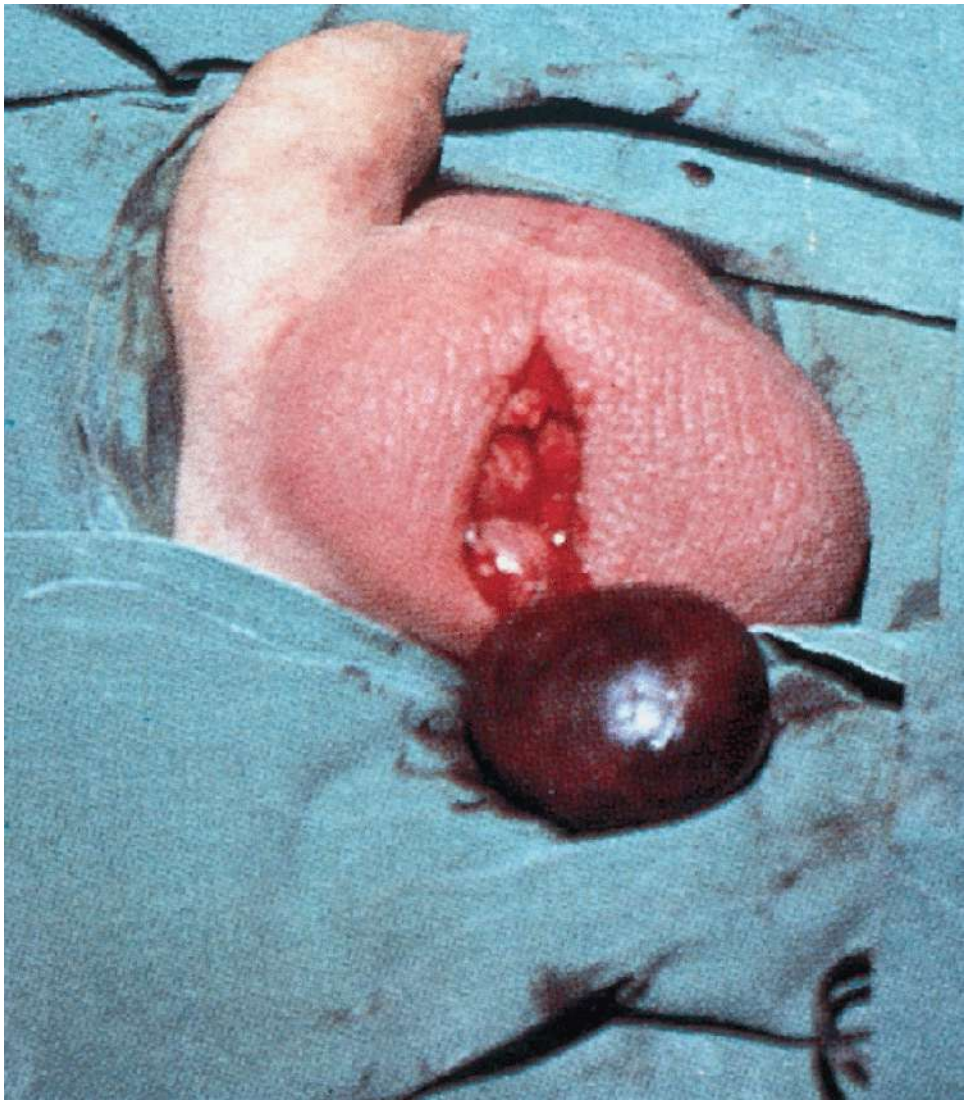

FIGURE 103.2 Torsion of the testis resulting in gangrene 12 hours from onset of pain. The testis was excised and the other normal testis ‘anchored’.

Scrotal pain at various ages

Acute epididymo-orchitis

Now that vaccination has rendered mumps so rare, acute epididymo-orchitis is usually caused by sexually transmitted pathogens in sexually active young males, most commonly *Chlamydia trachomatis* but also *Mycoplasma genitalium* and *Gonococcus* and by urinary tract pathogens in older males. In older men, it usually follows urinary tract obstruction and infection or instrumentation of the lower genitourinary tract.

Investigations

Blood cell count:	leucocytosis
Urine microscopy and culture:	pyuria, bacteria and possibly *Escherichia coli* a sterile culture suggests the possibility of a chlamydial or gonorrhoeal infection[4]
NAAT/PCR:	*Chlamydia, Gonococcus, Mycoplasm genitalium*
Swabs of discharge:	*Gonococcus* (in particular)
Ultrasound:	can differentiate a swollen epididymis from a testicular tumour

Treatment

- Bed rest
- Elevation and support of the scrotum
- Analgesics
- Antibiotics[4]

Sexually active men should be treated empirically for chlamydial or gonorrhoeal infection:

use ceftriaxone 500 mg (in 2 mL of 1% lignocaine) IM (or 500 mg IV) as a single dose

plus

azithromycin 1 g (o) as a single dose

plus either

another dose of 1 g azithromycin a week later, or doxycycline 100 mg bd for 14 days

Associated with urinary infection:

trimethoprim 300 mg (o) daily (child 4 mg/kg) for 14 days

or

cephalexin 500 mg (child 12.5 mg/kg) (o) 12 hourly for 14 days

or

amoxicillin/clavulanate 500/125 mg (child 12.5/3.1 mg/kg) (o) 12 hourly for 14 days

or (if resistance to above suspected or proven) norfloxacin 400 mg (child 10 mg/kg up to 400 mg) (o) 12 hourly for 14 days

If severe infection, administer parenteral gentamicin + ampicillin followed by norflaxin.[5]

Orchitis

Acute orchitis is invariably due to mumps and occurs during late adolescence. Mumps orchitis has become relatively rare with vaccination. It is usually unilateral (see FIG. 103.3) but may be bilateral.

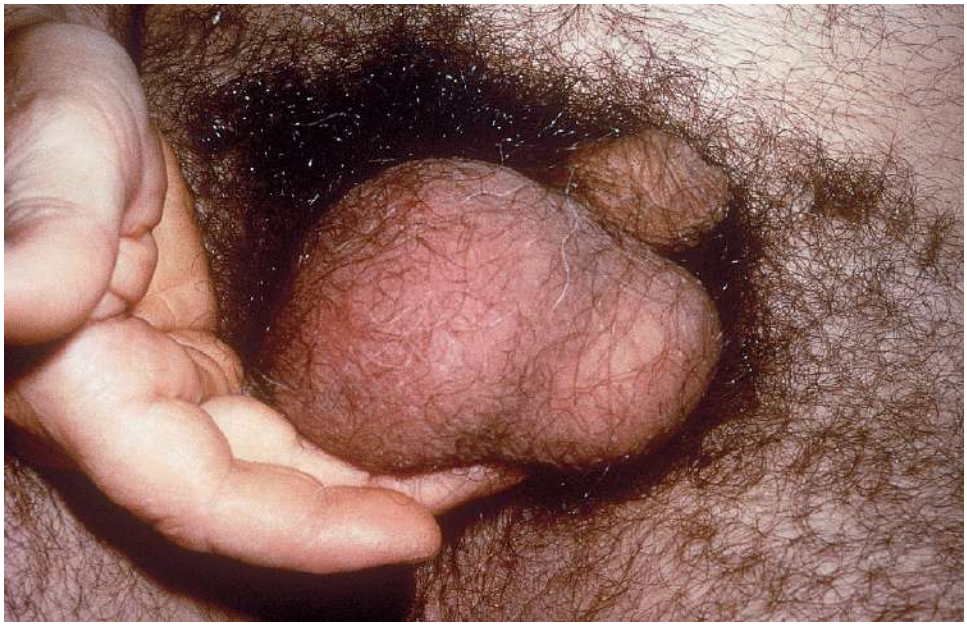

FIGURE 103.3 Mumps orchitis with a swollen, tender testicle

Testicular neoplasm

Testicular tumours can occur at all ages but are more common in young men aged 20–30 years (teratoma) and 25–40 years (seminoma). Sometimes they can mimic an acute inflammatory swelling and present with acute pain. See CHAPTER 104 .

Strangulated inguinoscrotal hernia

It is possible that a supposed testicular torsion may be found to be a strangulated inguinoscrotal hernia, usually an indirect inguinal hernia extending into the scrotum. It can be detected by careful palpation of the base (neck) of the scrotum, feeling for a cough impulse and being unable to ‘get above’ the swelling.

Trauma and haematoceles

A diffuse haematoma into the scrotum that causes no significant problems can follow surgery to the inguinal area, a blow to this area or a fracture of the pelvis. These conditions cause extravasation of blood distally. However, a haematocele of the tunica vaginalis can be either acute or an ‘old clotted haematocele’ following injury, such as a blow to the testis, or the drainage of a hydrocele. Sometimes it can arise spontaneously. All types of haematoceles require surgical exploration to exclude testicular rupture or a tumour.

Trauma to the scrotum may produce urethral injury and extravasation of urine into the scrotum. This problem requires urgent surgery.

Problems of scrotal skin

Sebaceous cysts are common and may be infected/inflamed and require excision or drainage. Less commonly, idiopathic scrotal oedema can present, usually in boys aged 5–10 years. With this condition, scrotal swelling and mild redness and tenderness begin gradually and spread, often across the midline and also possibly beyond the scrotum. Palpation reveals normal, non-tender testes but torsion needs to be excluded in some instances of the swollen red scrotum. Idiopathic scrotal oedema is believed to be allergic in origin, either localised (e.g. insect bite) or globalised as part of urticaria. It sometimes results from exposure to cold water. There may be a tender enlarged draining inguinal lymph node near the external ring.[1] Treatment includes scrotal support, analgesics and antihistamines.

Referred pain

Pain can be referred to the scrotal region from ureteric colic and quite commonly from disorders of the thoracolumbar spine, notably a disc disruption at the T12–L1 level involving the L1 nerve root. The pain therefore may be referred or radicular. In elderly men, referred pain can arise (uncommonly) from a ruptured abdominal aortic aneurysm or acute aortic dissection.

When to refer

- Any suspicion of torsion of the testis
- Sudden onset of acute scrotal pain at any age
- A history of recurrent transient testicular pain in a young man
- Presence of a tender testicular lump
- Presence of a haematocele surrounding the testis

Note: Referral should be urgent, given the critical importance of time in testicular survival.

Practice tips

- Acute scrotal pain in infancy and adolescence should be regarded as torsion of the testis until proved otherwise.
- A history of recurrent transient pain (with or without swelling of the testis) in a young person means recurrent torsion. Referral is essential.
- A pitfall is the phenomenon of ‘testis redux’ in which the descended testis undergoes torsion, is pulled into the superficial inguinal pouch by the cremasteric reflex and then becomes fixed by oedema. Always check both testes are present in the scrotum in boys and young men with inguinal pain.
- The development of an acute hydrocele should be regarded with suspicion.
- Beware of the strangulated inguinoscrotal hernia presenting as a testicular torsion.
- Consider dissecting aneurysm in an older person presenting with testicular pain.

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3 Davenport M. ABC of general surgery in children. Acute problems of the scrotum. BMJ, 1996; 312: 435–7.

4 Genital and sexually transmitted infections [published 2019]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2019. www.tg.org.au, accessed March 2021.

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104 Inguinoscrotal lumps

He has a rupture, he has sprung a leake.

BEN JONSON (1573–1637), *THE STAPLE OF NEWS*

Lumps in the groin are common to both sexes but males are likely to have a greater variety of swellings in this area and several may be associated with scrotal lumps.

Lumps in the groin

The commonest swellings encountered in the groin or inguinal area are enlarged lymph nodes and hernias. The diagnosis of a hernia is usually straightforward but it must be differentiated from other swellings, including Malgaigne bulgings—these are not true hernias but diffuse swellings in both inguinal regions seen in people with poor lower abdominal musculature.[1] TABLE 104.1 lists the differential diagnoses of groin lumps.

Table 104.1 Differential diagnoses of a groin mass

Hernia—femoral, inguinal
Malgaigne bulgings
Lipoma
Undescended testis (incomplete)
Spermatic cord swelling—encysted hydrocele, lipoma
Lymph node—localised, generalised
Haematoma (post femoral artery puncture)
Neoplasm—lipoma, others
Psoas abscess
Spermatocele
Vascular anomalies:

- saphenous varix
- femoral aneurysm

Hernias

The commonest types of hernias in the groin are inguinal, femoral and a combination of the two. Rare hernias in the region are obturator, Spigelian (low abdominal), preperitoneal inguinal and prevascular femoral. The basic parts of a hernia are shown in FIGURE 104.1 and important anatomical landmarks in FIGURE 104.2 . An indirect inguinal hernia is a hernia emerging through the deep inguinal ring, originating lateral to the inferior epigastric vessels, following the path of the spermatic cord, and which can traverse the whole length of the inguinal canal (see FIG. 104.3). It may also pass through the superficial inguinal ring into the scrotum—an inguinoscrotal hernia.

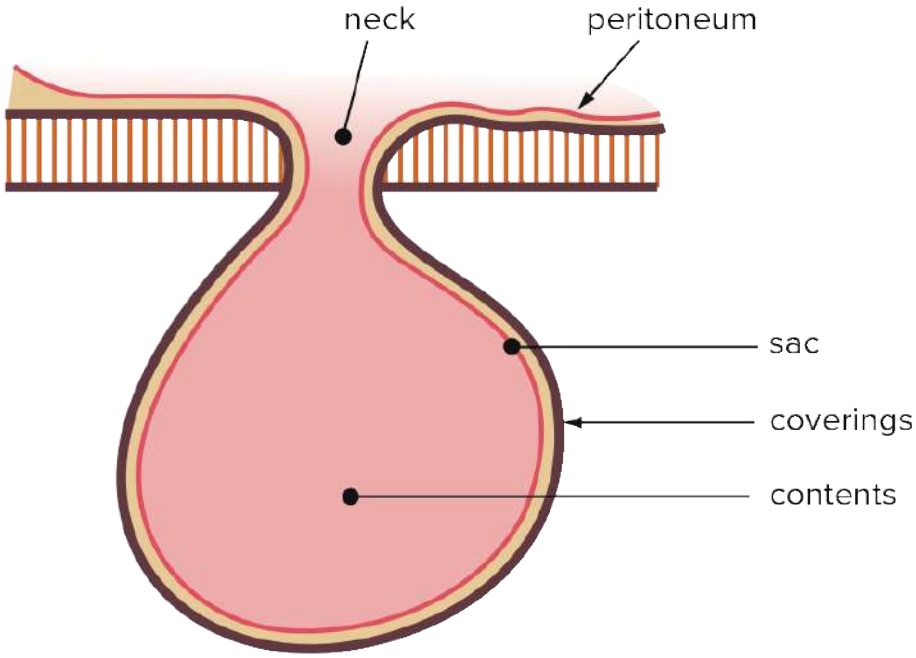

FIGURE 104.1 Basic components of a hernia

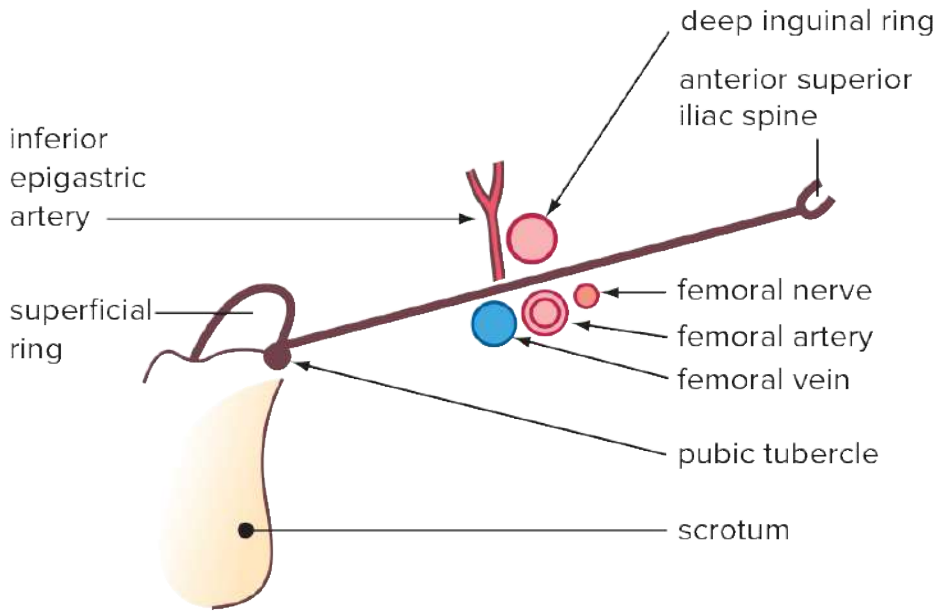

FIGURE 104.2 Key landmarks in the left inguinal region: the deep inguinal ring lies above the mid-inguinal point (between the ASIS and the pubic tubercle); the femoral artery lies below this point

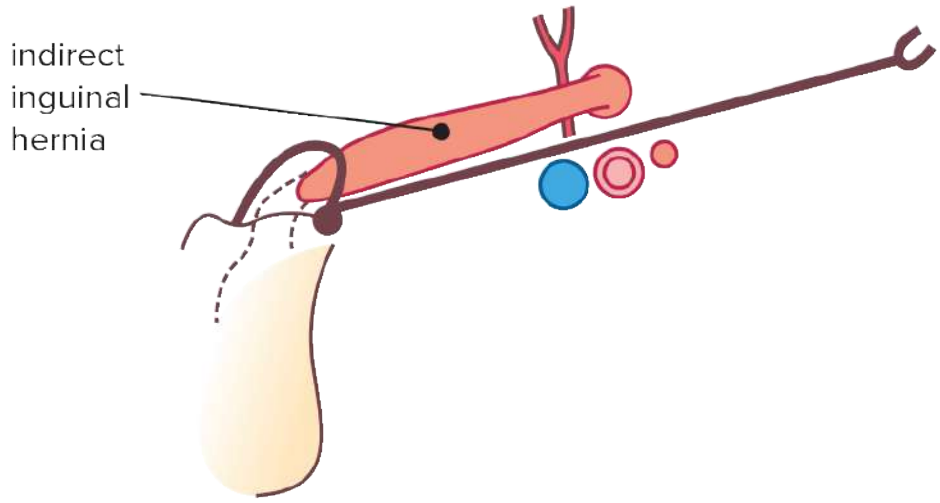

FIGURE 104.3 Left indirect inguinal hernia: it emerges lateral to the inferior epigastric artery and passes into the scrotum medial to the pubic tubercle

Because of their narrow neck and oblique path in the inguinal canal, such hernias are often irreducible and can occasionally lead to strangulation of entrapped bowel.

A direct inguinal hernia originates medial to the inferior epigastric vessels and protrudes through the posterior wall of the inguinal canal, and is therefore separate from the spermatic cord (see

FIG. 104.4). It is almost always seen in men and rarely descends into the scrotum. Due to a wider neck, strangulation and obstruction are most unusual. It must be emphasised that the distinction between a direct and an indirect inguinal hernia can be very difficult (and of no clinical benefit)[1] and the two may occur together.

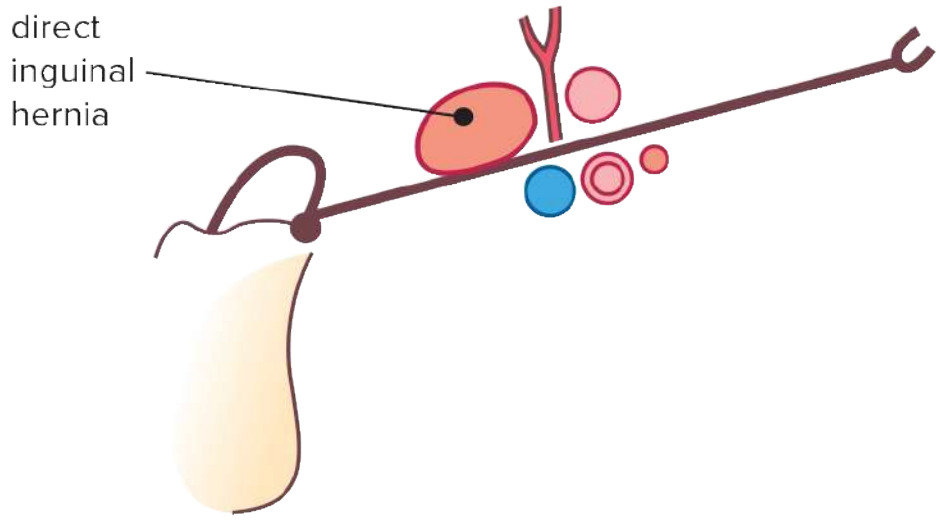

FIGURE 104.4 Left direct inguinal hernia: it emerges medial to the inferior epigastric artery and bulges forward

A femoral hernia herniates through the femoral ring (also known as the femoral canal), which is the medial component of the femoral sheath. The hernia tends to bulge forward and then upwards as it becomes larger. The neck is lateral to the pubic tubercle (see FIG. 104.5).

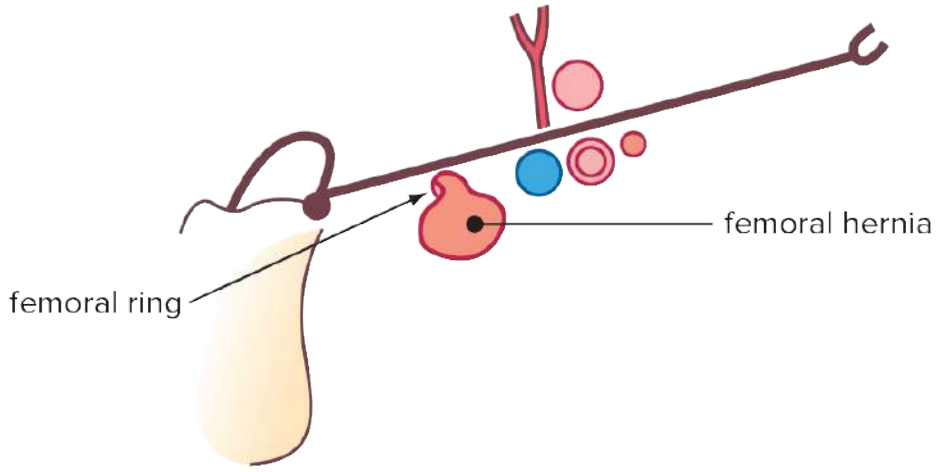

FIGURE 104.5 Left femoral hernia: its neck is lateral to the pubic tubercle and it lies below the inguinal ligament

Femoral hernias are often small, usually occur in females and may be unnoticed by the patient. They are particularly liable to produce bowel obstruction or strangulation.

Guidelines

Acquired hernia

- Always examine the scrotum and both sides
- Frequently bilateral
- Result from muscular weakness
- Commonest—direct inguinal
- Predisposing factors:

  older age

  obesity

  pregnancy
- Precipitating factors (related to above factors):

  increased intra-abdominal pressure:

  difficulty of micturition

  straining at stool (constipation)

  chronic cough (e.g. bronchitis)

  straining or lifting heavy objects

  nerve damage to muscle wall (e.g. post appendicectomy)
- Complications:

  intestinal obstruction (see TABLE 104.2)

  incarceration

  strangulation

  sliding

Table 104.2 Symptoms and signs of hernial

obstruction
Colicky abdominal pain
Nausea and vomiting
Constipation and failure to pass flatus
Abdominal distension
High-pitched tinkling bowel sounds
Local tenderness and swelling of the hernia
No expansile cough impulse

Clinical features

The main symptoms and signs:

- lump
- discomfort or pain:
    - a dragging pain
    - worse after standing or walking
    - referred to testicle (indirect inguinal)
- testicular pain—referred or with compression of the spermatic cord
- expansile impulse on coughing

Note:

- A femoral hernia is easily missed in people with obesity.
- Larger femoral hernias are often irreducible.
- Always attempt reduction in the recumbent position (direct hernias usually reduce easily).

Treatment

Surgery

All symptomatic hernias require repair. Those patients who are not symptomatic (a third of patients with hernias)[1] and who are medically fit should also be offered surgery. This is not so much because of the risk of strangulation (which is rare) but because most patients usually

become symptomatic eventually and a watch-and-wait approach delays rather than prevents surgery.[2] Obstructed and strangulated hernias require urgent surgery. The risk of strangulation is greatest with femoral hernias, occasional with indirect inguinal hernias and very rare with direct inguinal hernias.

While generally a very safe procedure, inguinal hernia surgery complications include bruising, numbness, haematoma, infection (1–7%), recurrence and chronic pain (defined as persisting for >3 months after surgery). Chronic pain is the most serious long-term complication of hernia repair and may persist for several years. It may be severe in up to 3% of patients[1] and can require referral to pain clinics or further surgery. Laparoscopic repair is now a popular surgical option with less severe postoperative pain.[3] An increase in infertility risk and testicular atrophy on the side of the surgery has been noted but is not common.

Most patients return to work after hernia repairs in around 7 days, or 14 days for those doing strenuous work.[1]

Conservative

Asymptomatic inguinal hernias in patients with associated medical conditions, and who pose a significant operative risk, such as the frail elderly patient, can be treated with a watch-and-wait approach. Trusses have been used since ancient times to help patients with hernias, but can be difficult for a patient to manage and cannot be recommended as a definitive form of treatment.[4]

Scrotal lumps

The scrotum contains the testes and distal parts of the spermatic cords, covered by layers of fascia and the dartos muscle. The testes are invested with tunica vaginalis derived from the peritoneal cavity during their descent.[1]

Disorders of the scrotum may be acute or chronic and bilateral or unilateral. Lumps may be cystic, solid or otherwise, such as a varicocele, oedema or hernia. Solid lumps include a testicular tumour, epididymo-orchitis, and torsion of the testes. Cystic lumps include hydroceles, epididymal cysts and spermatoceles. A comparison of scrotal lumps appears in FIGURE 104.6 and TABLE 104.3 . Lumps in the scrotum usually develop from deeper structures, particularly the testes and their coverings, rather than scrotal skin.

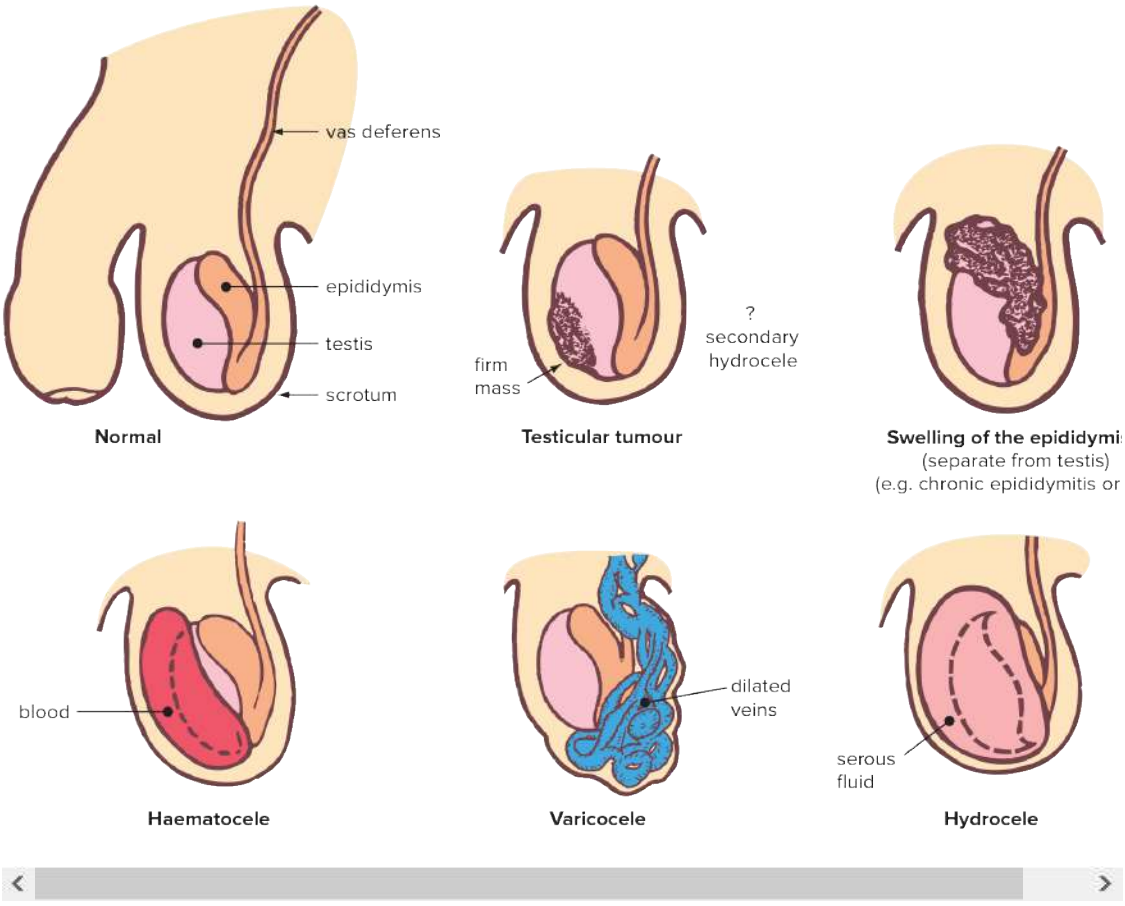

FIGURE 104.6 Basic comparison of scrotal lumps

Table 104.3 Features of scrotal lumps

	Possible clinical setting	Position	Palpation	Transillumination
Hydrocele	Any age Primary or secondary: • tumour • infection • torsion	Confined to scrotum Anterior: surrounds testis except posteriorly	Smooth, pear-shaped Lax or tense Testis impalpable, non-tender	Yes
Cyst of epididymis	Asymptomatic or dragging	Behind and above	Smooth and tense	Yes

Epididymal cysts and spermatoceles clinically similar	sensation	testis	Multilocular swelling Testis easily palpable Appears separate from testis	
Chronic epididymo-orchitis	Non-specific Tuberculosis *Chlamydia* (Occasional associated small hydrocele)	Behind and above testis	Firm swelling Hard and craggy Normal testis	No
Varicocele	Dragging discomfort	Usually left-sided Along line of spermatic cord Above testis	Soft, like bunch of worms or grapes Collapses when patient supine and testis elevated Testis often smaller	No
Cancer	Young men 20–40 Painless lump Loss of testicular sensation	In body of testis Usually felt anteriorly May be hydrocele	Enlarged firm testis Feels heavy if large Normal epididymis (palpable)	No

The cardinal sign of a true scrotal mass is that it is possible to palpate it from above (i.e. get above the lump) (see FIG. 104.7).

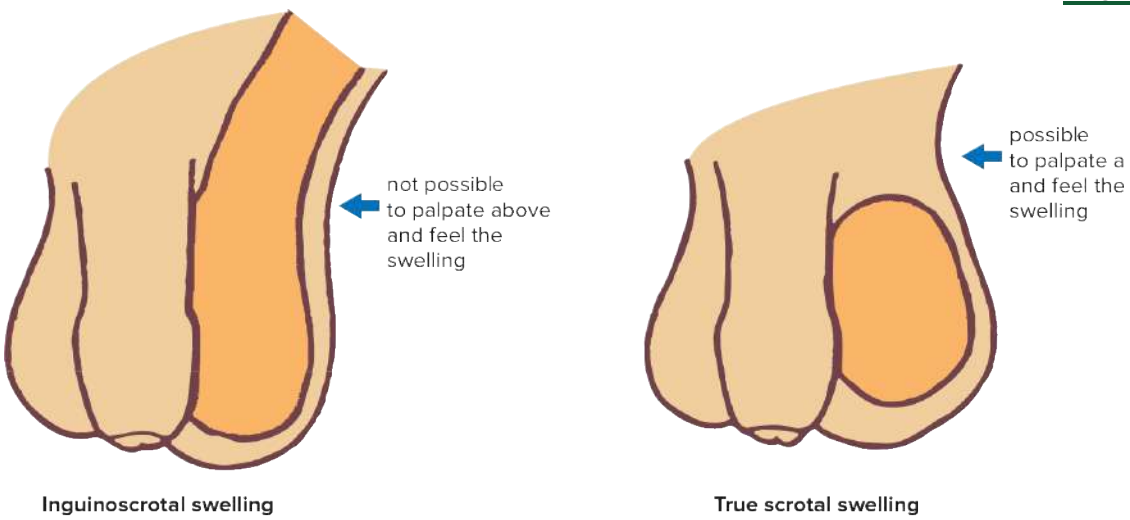

FIGURE 104.7 Difference between a true scrotal swelling and an inguinoscrotal swelling

The patient usually presents with pain or a lump.

A painless testicular lump is cancer until proven otherwise, but a testicular lump doesn't necessarily mean cancer.

Examination of the scrotum

The scrotum should be examined with the patient supine and then standing. The left testis usually hangs lower than the right. On inspection note any sebaceous cysts in the scrotal skin (common); scabies if there are very pruritic nodules; and scrotal oedema, which causes taut pitting skin. Careful palpation will elicit the relevant structures in the scrotum. Gently palpate each testis and epididymis between the thumb and the first two fingers. The spermatic cord is palpable as it enters the scrotum after passing through the superficial ring, and the testis and epididymis are readily palpable.

After palpation, test for translucency of any swelling in a darkened room by shining the beam of a strong torch from behind the scrotum through the swelling. Transilluminable swellings that light up with a red glow (referred to as a 'Chinese lantern sign') include hydroceles and cysts of the epididymis. Swellings that contain blood or other tissue, such as testicular tumours and most hernias, do not transilluminate.

Unilateral scrotal swelling

It is important to determine whether the lump is inguinoscrotal or scrotal. It is scrotal if it is possible to get above the lump. If it is not possible to get above the lump then it is a large inguinal hernia or a combined hernia and hydrocele (see FIG. 104.7). This palpation should be coordinated with the cough impulse. The next feature to determine is whether the testis and/or epididymis can be palpated or whether they are obscured by a swelling.[5]

Small testes

Normal testicular size in children and adolescents is 4–14 mL or 15–35 mL in adults (this can be assessed with an orchidometer).[6,7] Length measurement is 3.5–7.5 cm in adults (average 5 cm). Small, firm testes less than 10 mL are a feature of Klinefelter syndrome. Small soft testes indicate atrophy, which may follow mumps orchitis, oestrogen therapy, androgen deficiency or anti-androgen therapy, hypopituitarism, cirrhosis and other related conditions.

Hydrocele

A hydrocele is a collection of clear amber fluid in the tunica vaginalis and can be primary or secondary. If a hydrocele develops it is important to rule out intrascrotal disease, such as a tumour or infection. Ultrasound examination of the scrotum is helpful in assessing the state of the testis in the presence of a hydrocele. Hydroceles may be symptomless or cause dragging discomfort in the scrotum and groin.

Hydrocele in the neonate[5,8]

Hydroceles present at birth are communicating (failed closure of the processus vaginalis) or (less commonly) non-communicating, where the tunica vaginalis contains fluid, which can sometimes be loculated or cystic. Transillumination will prove that it is cystic but if the diagnosis is in doubt perform an ultrasound. Hydroceles may vary in size from day to day and can appear quite large. They do not extend proximal to the external inguinal ring so it is possible to palpate above them. There is no impulse on crying or straining. Of the 5% of those born with a hydrocele, most will resolve spontaneously within 12 months. If the hydrocele is very large or persists beyond 12 months, surgical intervention should be considered. Surgery is the most effective long-term treatment.

Treatment of a primary hydrocele in adults

If underlying pathology has been excluded, hydroceles can be managed conservatively with reassurance and scrotal support.[9] If the hydrocele is large and uncomfortable, simple aspiration with or without the injection of sclerosing agents can be attempted, but the fluid usually reaccumulates and there is a risk of bleeding or infection with repeated procedures. However, aspiration can prevent fluid accumulation and after two or three times can often cure the problem. This sclerotherapy may be complicated by pain and inflammatory reaction to the sclerosant. Surgery is generally considered a second-line procedure after observation with or without aspiration. However, surgery is the most effective long-term treatment.

Practice tip

Consider testicular cancer in a young man presenting with a hydrocele. Perform an ultrasound examination.

Encysted hydrocele of the cord

This is a localised fluid-filled segment of the processus vaginalis within the spermatic cord. It is palpable as a cystic lump in the upper scrotum above the testis. It characteristically moves down with traction on the testis. Treatment is not usually needed.

Epididymal cysts

These are common, often multiple and are usually found in middle-aged/elderly men. The majority of epididymal cysts are about the size of a pea and contain a clear colourless fluid. If the cysts communicate with the vasa efferentia, a spermatocele filled with whitish fluid containing spermatozoa may form.

Extratesticular, fluctuant, cystic swellings which transilluminate and are readily palpable separate from the body of the testis are epididymal cysts and do not usually need further investigation.[10]

Epididymal cysts may be asymptomatic or they may cause discomfort and cosmetic embarrassment and if so can be excised. Fertility may be impaired in patients undergoing bilateral cyst excision.

Aspiration and injection of sclerosant agents can also be used for epididymal cysts.

Varicoceles

A varicocele is a varicosity of the veins of the pampiniform plexus (see FIG. 104.6). It is seen in 8–10% of normal males and occurs on the left side in 98% of affected patients, due to a mechanical problem in drainage of the left kidney vein. A relationship with infertility has been observed but its nature is controversial, as is whether repairing varicoceles in subfertile men improves fertility chances.[9]

Most varicoceles are asymptomatic and incidental findings. They can cause a dragging discomfort in the scrotum. Investigation is usually not necessary but an ultrasound is useful where the diagnosis is doubtful or a neoplasm is suspected. Treatment is indicated if it is symptomatic or for infertility. Firm-fitting underpants may relieve discomfort. Surgical treatment is by venous ligation, above the deep inguinal ring. Ligation is indicated if there is any reduction in the size of the left testis.

Haematoceles

These can be either acute, resulting from trauma such as a fall astride, sports injury or tapping of a hydrocele, or chronic, where there is no obvious history of injury. Haematoceles are anterior to the testis and not transilluminable. Surgical drainage is required with acute injury where there is a possibility of testicular rupture (associated urethral injury has to be considered); and a tumour has to be excluded with the chronic type. Pressure atrophy of the testis can occur with injury and is much more common if early drainage is not instigated.

Sperm granulomas

- Firm tender lumps
- Post-vasectomy—at cut end of vas

Treatment

- Leave to resolve.
- Consider NSAIDs.
- Refer for excision if symptomatic and enlarging.

Fordyce spots

These are harmless ectopic sebaceous glands 1–3 mm in diameter that appear as small raised lumps or spots of various colours (mainly red) on the shaft of the penis (Tyson glands) or scrotum. They can be mistaken for genital warts. They are best left, but if unacceptable they can be treated with electrodissection or pulsed dye lasers or a micro-punch technique.

Testicular tumours[11,12]

A mass that is part of the testis, and solid, is likely to be a cancer. Malignant testicular tumours account for about 1–1.5% of malignant tumours in men. They mainly affect fit young men and represent the commonest cancer in men aged 15–40 years in Australia (see TABLE 104.4). Some 90–95% of testicular tumours arise from the germ cells, and for practical purposes are classified into:

- seminomas 40%
- non-seminoma germ cell tumours (NSGCT) 60%

Table 104.4 Testicular tumours

Tumour	Incidence (%)	Peak incidence (years)
Seminoma	40	25–40
Teratoma	32	20–35
Mixed seminoma/teratoma	14	20–40
Lymphoma	7	60+
Other tumours (e.g. interstitial—Leydig, gonadoblastoma)	uncommon	variable

Clinical features

- Young men aged 15–40 years (NSGCTs peak in the third decade of life, seminomas peak in the fourth decade)
- Most common presentation is a painless lump in the body of one testis (only 1–2% present with bilateral tumours)
- Up to a quarter of men have pain at presentation
- Loss of testicular sensation
- Associated presentations (may mask tumour):

    hydrocele

    varicocele

    epididymo-orchitis

    swollen testis with trivial injury

    gynaecomastia (teratoma)

    back and flank pain

Risk factors

Those at high risk include those men with:

- cryptorchidism/orchidopexy (testicular dysgenesis syndrome)
- previous testicular cancer
- family history of testicular cancer (brother/father)

- Klinefelter syndrome
- infertility

Golden rules

- All solid scrotal lumps are malignant until proved otherwise and must be surgically explored.
- Beware of hydroceles in young adults.
- Tumours can mimic acute epididymo-orchitis—the so-called ‘inflammatory’ or ‘flash fire’ presentation.

If a man has a testicular lump he should visit his GP and have ultrasound screening and baseline tumour markers.

Metastases

Testicular tumours spread by direct infiltration via the lymphatics, including retroperitoneal lymph nodes, and the bloodstream. Metastases typically occur in the para-aortic nodes and so may not be detected by abdominal palpation. They are best detected by a CT scan of the abdomen and chest. Metastases also occur in the neck, brain, liver, chest and bones.

Investigations

Investigations to aid diagnosis include:

- ultrasound of the testis should always be done as it can detect and diagnose with considerable precision underlying testicular lumps plus any invasion of the tunica
- tumour markers: α-fetoprotein and β-hCG—indicates teratomas
- lactic dehydrogenase may be elevated

Investigations for staging include:

- chest X-ray
- CT scanning of abdomen, pelvis and chest for node involvement (any spread is usually direct to the para-aortic nodes)
- lactate dehydrogenase—monitors secondary spread and indicates tumour mass

Note: Avoid scrotal needling biopsy because of the potential risk of tumour implantation in the scrotal wall. Avoid scrotal incisions for surgery.

Treatment

The initial treatment is orchidectomy through an inguinal incision with inguinal division of the spermatic cord. Further treatment then depends on the type and staging of the tumour. Early seminomas are given radiation to the ipsilateral lymph nodes or a single dose of chemotherapy. Early NSGCTs undergo active surveillance (tumour markers, CXR, CT scans) but no further treatment. More advanced disease may have further chemotherapy or radiotherapy. The results for NSGCTs in general are not as satisfactory as for seminoma, though prognosis is good for both, with current 5-year relative survival rates of 99% for localised disease (majority of presentations), 96% for regional disease and 74% for distant disease.[13] The advent of chemotherapy since the 1960s has revolutionised survival rates for most testicular cancers from almost certain death to almost certain survival. A comparison of the testicular tumours is summarised in TABLE 104.5 .

Table 104.5 Comparison of the common testicular cancers

	Seminoma	Non-seminoma (NSGCT)
Typical age	30–40 years	20–30 years
Incidence	40%	60%
Growth rate	Slow	Rapid
Nature	Solid	Mixed—solid + cystic
Stage at presentation	90%—stage 1	60%—stage 1
Tumour markers:		
α-FP	Never	Common
β-hCG	Occasional	Common
Treatment	Inguinal orchidectomy + radiotherapy	Stage 1: orchidectomy Relapse: chemotherapy
Sensitive to chemotherapy	+ + +	+ + +
Sensitive to radiotherapy	+ + +	±

Surgery should not affect the remaining testis but production of motile and functional sperm may

be reduced. However, sperm production can be temporarily or permanently reduced following radiotherapy and chemotherapy. Pretreatment sperm storage may be discussed with the patient, and the psychological implications of testicular cancer need to be carefully monitored. A testicular prosthesis is an option. The GP may also play a role in coordinating appropriate active surveillance investigations.

Screening and testicular self-examination

Studies of testicular cancer have shown the benefits of early detection. However, studies to date indicate that there is insufficient evidence to screen routinely for testicular cancer in asymptomatic patients. It is recommended for those at high risk. This screening includes colour Doppler ultrasound and tumour markers. Evidence also indicates that, to date, there is little evidence to show that those performing testicular self-examination are more likely to detect early stage tumours or have better survival than those who do not.[14]

Undescended testes (cryptorchidism)[15]

An undescended testis is one that fails to reach the bottom of the scrotum by 3 months of age despite manual manipulation. It has stopped in the normal path of descent and can occupy the intra-abdominal, inguinal canal, emergent (just outside the external ring), high scrotal and mid-scrotal positions (see FIG. 104.8). The cause of maldescent is most probably mechanical.

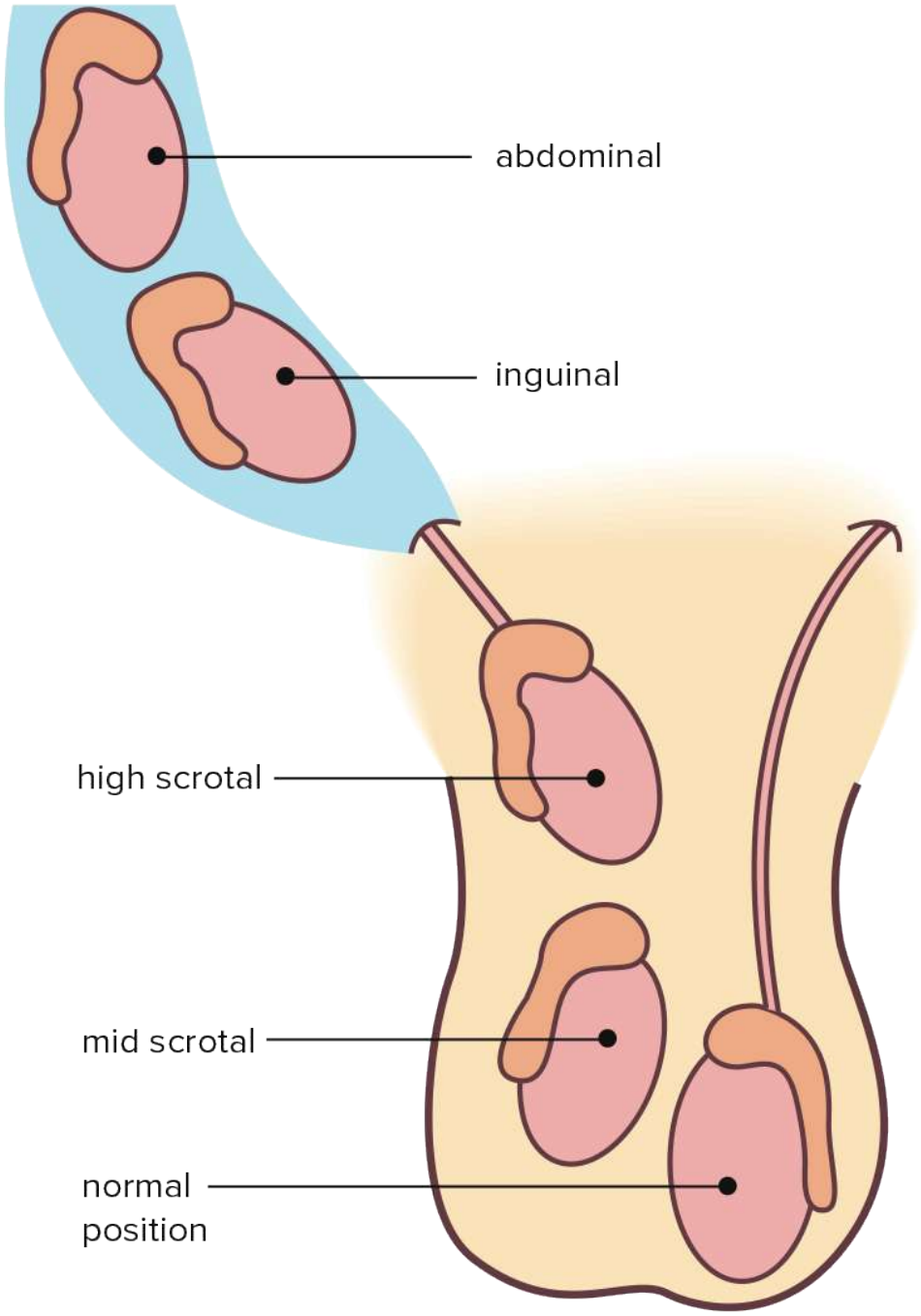

FIGURE 104.8 Undescended testis: arrested in the line of descent

After the indirect inguinal hernia, it is the most common problem in paediatric surgery. The incidence at birth is 3–4%, but this falls to 1% in the first 3 months of life as testicular descent can continue, though further descent after 3 months is rare. More than two-thirds of undescended testes are located in the superficial inguinal pouch; that is, they are palpable in the groin.

The testis is usually normal at birth but may become secondarily dysplastic if left outside the scrotum.

Retractile testis

A retractile testis is one that can be manipulated into the scrotum irrespective of the position in which it is first located. It is a common condition and requires no further investigation or treatment. The testis can be present in the scrotum under circumstances such as a warm bath but retracted out of the scrotum when cold. Cremasteric contraction is absent in the first few months after birth and is maximal between 2 and 8 years.

Ectopic testis

An ectopic testis is one that has left the normal path of descent and cannot be manipulated into the scrotum. It can be found in the perineum, upper thigh (femoral), base of the penis (prepubic), anterior abdominal wall or in the superficial inguinal pouch (see FIG. 104.9). True ectopic testes form only about 2.5% of all undescended testes.

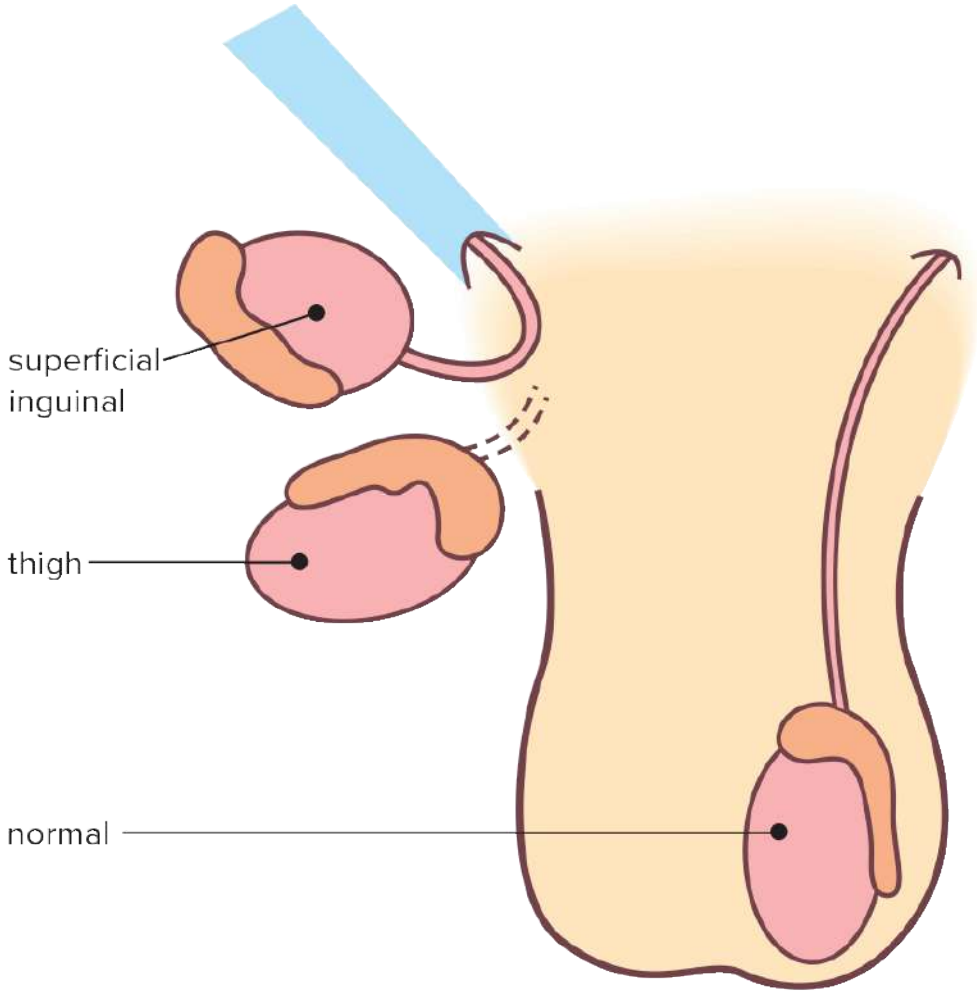

FIGURE 104.9 Undescended testis: ectopic

Ascending testis

An ‘ascending’ testis is one that was in the scrotum in infancy but subsequently moved back to the groin because the spermatic cord failed to elongate at the same rate of body growth.

Examination[12]

The examination of the testes should take place in a warm room and relaxed environment. Begin by placing one finger on each side of the neck of the scrotum to prevent a retractile testis from being retracted when palpation is commenced with the other hand. The scrotum is then carefully palpated for a testis. If impalpable, the fingertips of one hand are placed just medial to the anterior superior iliac spine and moved firmly towards the pubic tubercle where the other hand waits to entrap the testis should it appear. The diagnosis then depends on carefully determining the range of movement.

Practice tip

If the testis is not palpable at birth, review in 3 months. Refer for specialist evaluation if it still cannot be palpated.

The problem of non-descent

- Testicular dysgenesis and dysplasia
- Susceptible to direct trauma (if in inguinal region)
- Risk of malignant change (seminoma) is 5–10 times greater than normal

Optimal time for surgery

The optimal time for orchidopexy is 6–12 months of age. It is considered to be satisfactory as long as the testis is in the scrotum by 2 years. The production of spermatozoa is adversely affected in undescended testes from the age of 2 years onwards. Exploration for the uncommon impalpable testis is worthwhile: 50% salvage rate, while in the other 50% either there is no testis or an abnormal and potentially neoplastic testis is removed.

The advantages of early orchidopexy are summarised in TABLE 104.6 .

Table 104.6 Advantages of early orchidopexy (1 year)

Provides optimal chance of fertility
Corrects indirect inguinal hernias (coexists in 90%)
Reduces risk of trauma
Reduces risk of torsion
Reduces psychological consequences
Probably lessens the risk of malignancy (seminoma)

Hormone injections

Injections of chorionic gonadotrophic hormones are generally not recommended. They are ineffective except for cases of borderline retractile testes.

Vasectomy

Caution required

Take extra precautions with communication and fully informed consent if:

- Not in a long-term relationship
- Young <35 years old
- No children
- Emotional crisis/current depression
- Spouse/partner not involved in the decision

Postoperative

- Avoid strenuous activity and sex for 4–7 days.
- Use alternative contraception until negative semen analysis.
- First semen analysis at 3 months.
- Inform not immediately infertile; need to ejaculate 20 times.

Complications[16]

- Postoperative bleeding and haematoma 4–22%
- Wound infection <1.5%
- Ongoing pain—around 10%, usually mild but can be severe
- Formation of sperm antibodies ~50%. May be a problem if subsequently requests vasectomy reversal
- Failure rate 1–2:1000 (can be from tubes not fully tied off, growing back together or a third vas deferens existing)
- Psychological and psychosexual problems

Other facts

- No difference in ejaculate appearance (sperm makes up 1% ejaculate volume) or sex drive

- No known association with prostate and testicular cancers

Practice tips

Inguinoscrotal lumps

General rules for optimal times for surgical repair in children:

- inguinal hernia—ASAP
- umbilical hernia—aged 4 years (most resolve)
- femoral hernia—ASAP
- undescended testes—6 to 18 months
- hydrocele—after 12 months if still large; most resolve
- varicocele—leave and review

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Hernia, inguinal
- Scrotal lumps
- Testicle: undescended
- Testicular cancer
- Testicular self-examination

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105 Disorders of the penis

Ironically there is no organ about which more misinformation has been perpetuated than the penis.

WILLIAM MASTERS & VIRGINIA JOHNSON, *HUMAN SEXUAL RESPONSE* (1970)

The most common penile disorders are those of psychosexual dysfunction and STIs, but there are many other problems and these are most often related to the foreskin.

Disorders affecting the foreskin and glans

Phimosis

Phimosis is tightness of the foreskin (prepuce), preventing its free retraction over the glans penis. The foreskin is normally attached to the glans and is non-retractile in most newborns; it may remain so until puberty.[1,2] The proportion of boys with retractile foreskins is: 40% at 1 year, 90% at 4 years and 99% at 15 years.[3] Forceful retraction of the penis should be avoided until spontaneous separation occurs.[1] Once the foreskin easily retracts, the boy should learn to do this as part of normal washing, ensuring he rinses off any soap and pushes the foreskin back over the glans afterwards.[4]

'True' phimosis is caused by forceful retraction, infection or balanitis xerotica obliterans (see later in this chapter).[1,2] Indicators of true phimosis (rather than simple non-retractile foreskin) are:[3]

- foreskin not retractile by the time of established puberty
- previously retractile foreskin becomes non-retractile
- obvious ring of scar tissue at foreskin opening
- inability to visualise the urethral meatus when foreskin opening is lifted away from the glans
- ballooning during and after micturition, with pinhole foreskin opening and a squirting urinary stream (though mild ballooning during micturition is common and normal)

Treatment

Inflammatory phimosis can be treated by local corticosteroid cream (e.g. 0.05% betamethasone valerate cream qid for 2–4 weeks)[3] applied generously to the tight, shiny part of the foreskin where the inner skin meets the outer skin. If the tip of the foreskin is inflamed, gently retracting it will cause the inflamed opening to ‘cauliflower’ up so the cream can be applied. A stronger steroid cream can be tried if this fails.

True scarring that fails to respond to steroid creams may require circumcision, though this is uncommon.[1,2] Some patients with true phimosis may have problems once they start to have intercourse and will require circumcision.

Paraphimosis

Paraphimosis occurs when a tight foreskin is forcibly retracted over the glans, gets stuck in the sulcus and cannot be pulled forward again. The glans and the foreskin distal to the tight area become swollen and painful (see FIG. 105.1) and this demands urgent resolution.[1] This problem occurs in boys aged 8–12 and the elderly, especially if a mild degree of phimosis is already present. Typically it occurs when the penis is erect or after catheterisation.

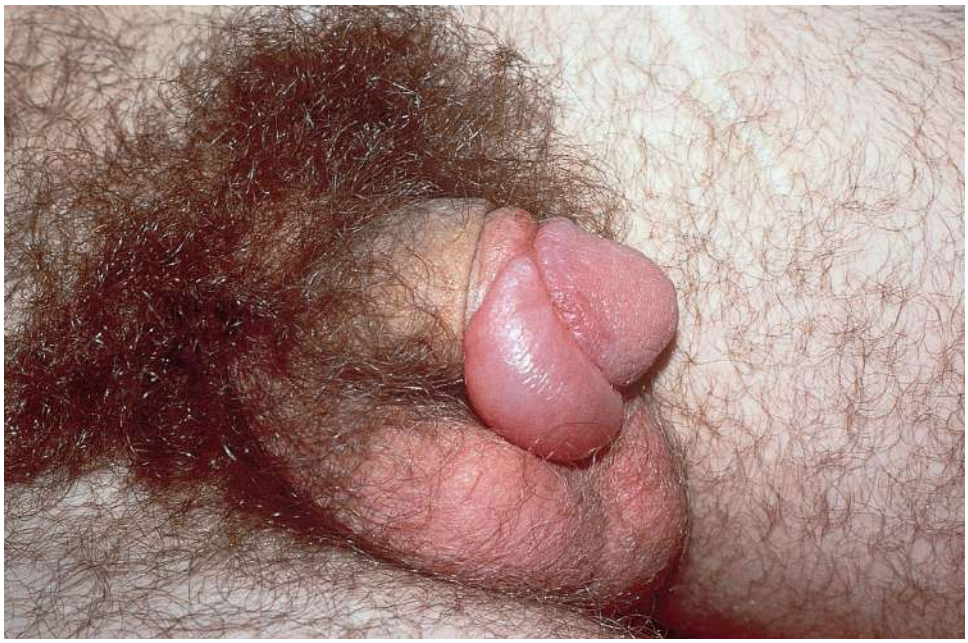


FIGURE 105.1 Paraphimosis showing retracted, swollen, oedematous foreskin

Management

Paraphimosis can usually be corrected without surgery, and urgent manual reduction should be

attempted first.[3] It is usually performed without anaesthesia but a penile block (remembering never to use adrenaline), systemic analgesia ± sedation or a generous application of 2% lignocaine gel or Emla cream may be appropriate depending on the geographic location and the clinical circumstances. Local anaesthetic infiltration before manual reduction should be avoided as it increases the swelling.

Note: Do not apply ice.

Method 1: Squeeze and manual reduction A

The glans penis and oedematous tissue distal to the constricting ring of foreskin are gently squeezed for several minutes to reduce the oedema. Using a lubricating jelly, manual reduction can then be performed by trying to advance the prepuce over the glans with the index fingers while gently compressing the glans with both thumbs (see FIG. 105.2).

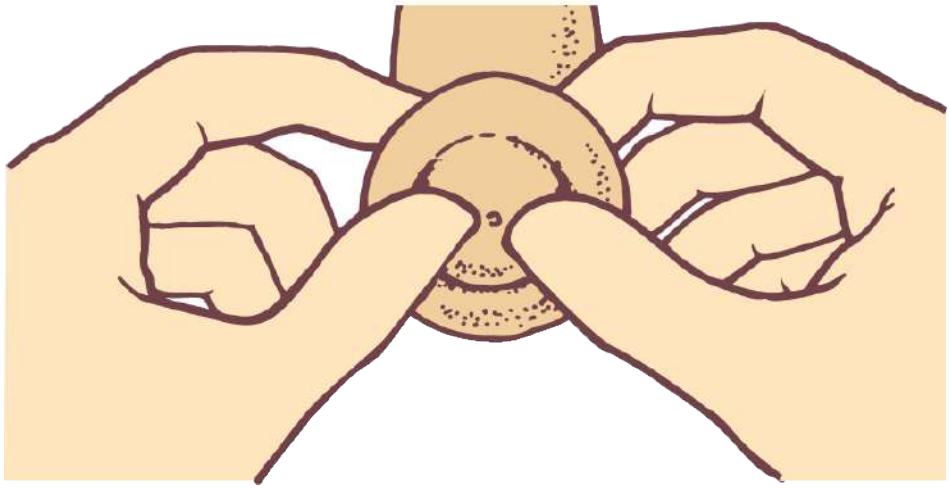


FIGURE 105.2 Acute paraphimosis: method of manual reduction

Method 2: Squeeze and manual reduction B

- Take hold of the oedematous part of the glans in the fist of one gloved hand and squeeze firmly. A gauze swab or cool towelette will help to achieve a firm grip (see FIG. 105.3).

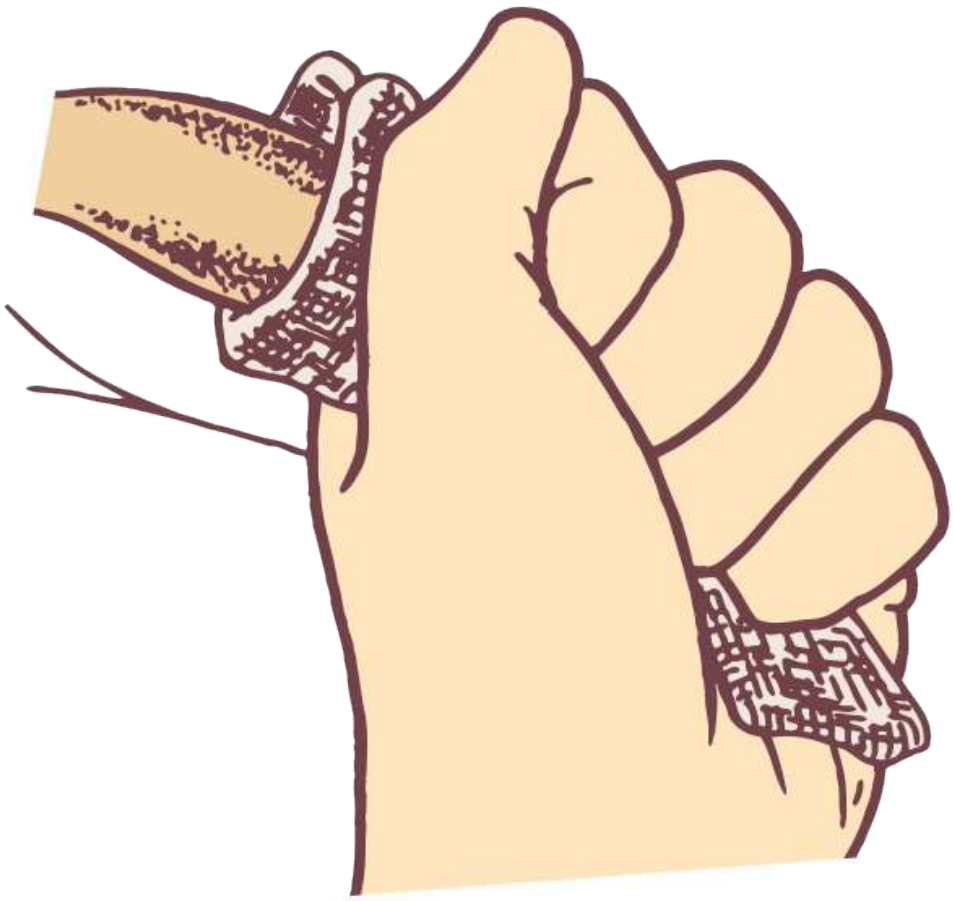


FIGURE 105.3 Acute paraphimosis: squeezing with a swab method

- Exert continuous pressure until the oedema passes under the constricting collar to the shaft of the penis.
- The foreskin can then usually be pulled over the glans.

Note: If these simpler methods are successful, educate the patient about proper foreskin management as this may prevent further episodes.

Method 3: Dorsal slit

Immediate referral is necessary if manual reduction methods fail. As an emergency, a dorsal slit incision can be made in the constricting collar of skin under local or general anaesthetic (see FIG. 105.4). The incision allows the foreskin to be advanced and reduces the swelling. Circumcision can be considered some days later when the inflammation has settled and any phimosis assessed, but a single episode of paraphimosis is not an absolute indication for circumcision.[3]

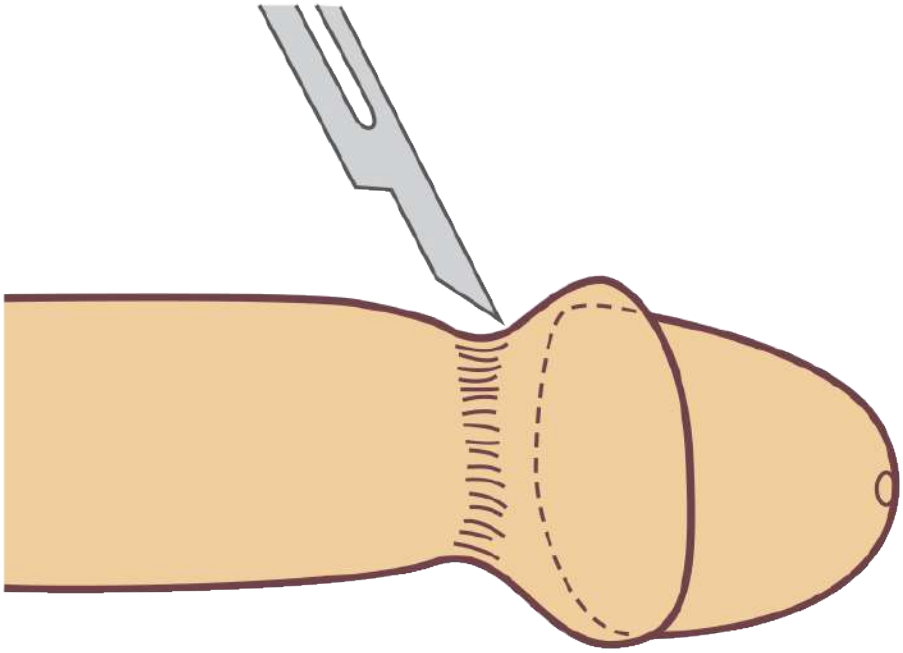


FIGURE 105.4 Acute paraphimosis: dorsal slit incision in the constricting collar of skin

Method 4: The Dundee puncture method[5]

Under a ring block local anaesthetic or general anaesthetic, make approximately 20 puncture holes in the oedematous prepuce with a 26 gauge needle (outer diameter 0.45 mm). Then, using firm but gentle pressure, express fluid from the foreskin until it is decompressed. Consider subsequent circumcision.

Balanitis

Balanitis is inflammation of the glans; inflammation of the foreskin is called posthitis and the combination is balanoposthitis, though balanitis is the most commonly used term. Urine trapped under the foreskin converts into ammonia compounds, and an ammoniacal dermatitis (similar to that of nappy rash) causes inflammation. Other causes of inflammation include irritation by soaps and other environmental substances, and trauma such as sexual activity. This inflamed area can become infected with commensal bacteria or fungi leading to infection.[1] Balanitis is common, affecting 6% of uncircumcised and 3% of circumcised males.[3]

Balanitis is actually a collection of disparate conditions with similar clinical presentation and varying aetiologies affecting a particular anatomical site.[6,7] These include:

- infections—*Candida* (the most common cause), *Streptococcus pyogenes* (uncommon but very

severe), anaerobes, staphylococci, *Gardnerella*, herpes simplex

- inflammatory dermatoses—lichen sclerosis, lichen planus, psoriasis, Zoon balanitis, eczema (including irritant, allergic and seborrhoeic)
- premalignant—Bowen disease, Bowenoid papulosis, erythroplasia of Queyrat

Men presenting with balanitis should be considered for predisposing conditions such as diabetes or HIV.

Treatment

Treatment will be dependent on the cause, which will in turn be determined by the clinical picture, patient risk factors and potential culture results. Cultures can be useful, but may also pick up commensals unrelated to the aetiology so cannot always be relied upon.

- Mild cases felt to be caused by an inflammatory dermatosis may be treated with gentle saline bathing, a barrier cream or hydrocortisone 1% cream to the tip of the penis, and careful washing behind the foreskin.
- If there is a purulent discharge, take swabs for culture.
- Candidal balanitis is suggested by blotchy erythema with small papules that may be eroded, or dry, dull, red areas with a glazed appearance, and associated soreness ± itch.

If yeasts present:

- topical hydrocortisone + an imidazole (e.g. miconazole or clotrimazole) cream

If trichomonads present:

- metronidazole or tinidazole (oral treatment)

If bacterial infection is suspected:

- topical antibiotic ointments under the foreskin using a narrow, longish nozzle (e.g. chloramphenicol)
- severe cases caused by bacterial infection may have purulent discharge with spreading cellulitis down the penile shaft and require oral or intravenous antibiotics

Small tubes of ointment designed for ear application (e.g. Otocomb Otic) combine a corticosteroid, antibacterial and antifungal, and may be useful for balanitis.

Balanitis xerotica obliterans (BXO)

Thickening of the foreskin with skin pallor suggests balanitis xerotica obliterans. There is white, thickened scarring, giving an ‘icing sugar’ appearance to the glans. It results in progressive

phimosis, typically in late childhood, usually 10–12 years. It may respond to corticosteroid cream if it is mild, but circumcision is usually indicated.

Frenuloplasty

A congenitally tight frenulum may lead to a tear during intercourse. Repeated bleeding occurs. Division of the frenulum and suturing in the opposite direction is preferable to circumcision.

The ‘buried’ penis

In the ‘buried’ penis syndrome (also known as the ‘concealed’ or ‘inconspicuous’ penis) the penis is not adequately exposed and looks small. There is failure of skin fixation at the base of the penis with possible excessive prepubic fat pad at the base. It is commonly seen in small children, especially chubby boys, and can cause parental concern. Manually pressing down the fat pad at the base of the penis back towards the pubic bone will allow a more reliable assessment of penile length (and often reassure the parents). This situation usually improves spontaneously over time. In adolescents and adults, a buried penis is usually associated with obesity and rarely can require surgical intervention for cosmetic or functional reasons.[8]

Foreskin hygiene

The normal foreskin in infants and children does not need special care and should be retracted for cleaning only after spontaneous separation of the foreskin has occurred. After this, males can practise proper hygiene by gently retracting the foreskin and washing the area as often as washing behind the ears.

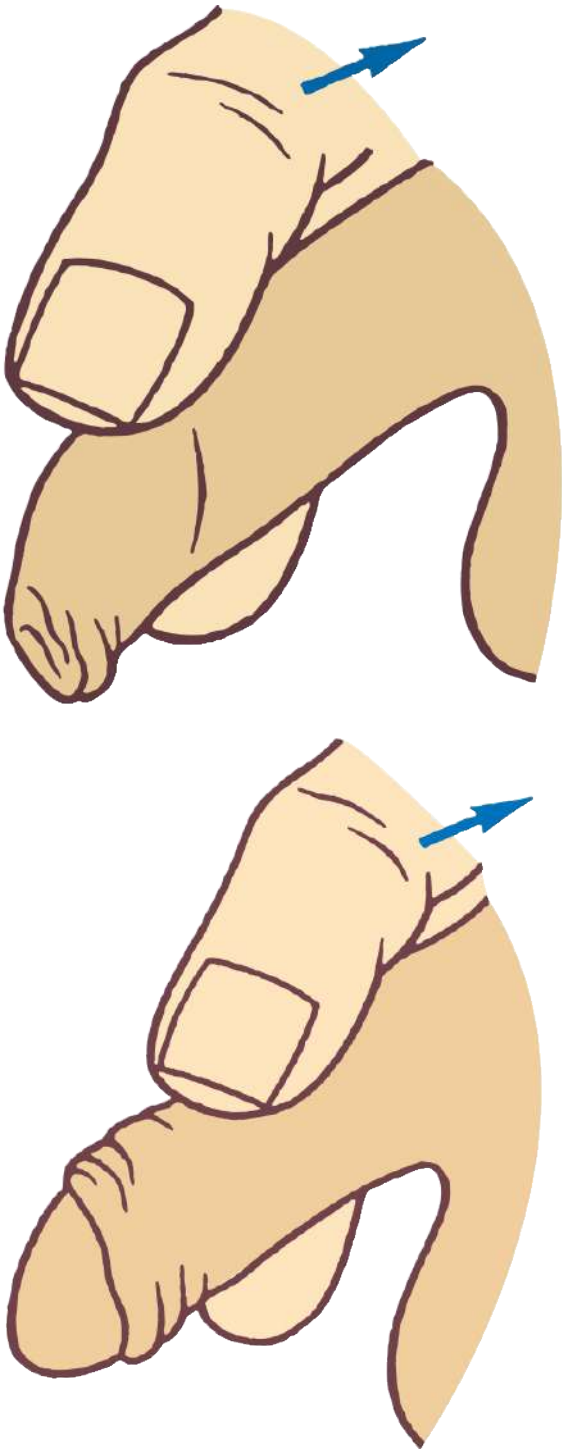
Basic rules

- The foreskin should be retracted only by its owner (or a health professional)
- The foreskin should not be retracted forcibly
- Once retraction has spontaneously occurred, encourage daily retraction and gentle cleansing in the bath or shower

Instructions to patients

- During a shower or bath slide the foreskin back towards your body (see FIG. 105.5).
- Wash the end of the penis and foreskin with soap and water and then rinse off.
- After washing the area, dry the end of your penis and foreskin and then replace the foreskin.
- If the foreskin has a tendency to become irritated and smelly, slide the foreskin back

sufficiently to allow free urination.



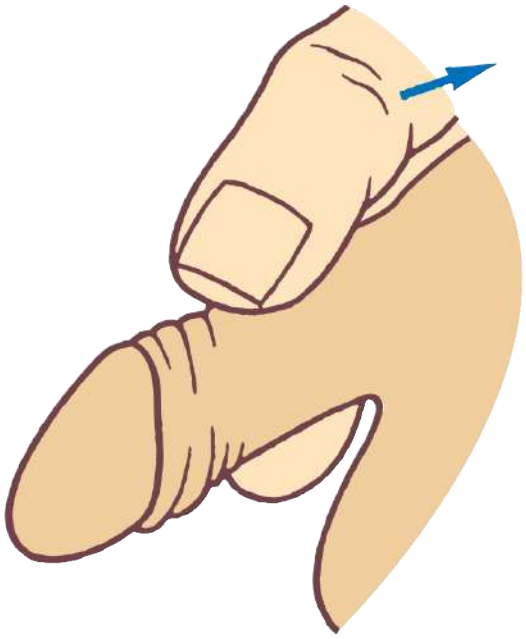


FIGURE 105.5 Foreskin hygiene: sliding foreskin back for washing

Smegma pearls

Smegma is made up of shed skin cells from the inner aspect of the foreskin and sebaceous gland excretion. Smegma cysts/deposits (yellow–white lumps beneath the foreskin) can be visible or palpable through the thin foreskin of boys whose foreskins have not retracted. They are sometimes called ‘smegma pearls’, and are normal and need no treatment. They can discharge a white ooze from under the foreskin. They can also be mistaken for sebaceous cysts and may cause concern for the boy or his parents, who can be reassured all is well.[1]

Circumcision

Circumcision is now performed in 10–20% of Australian males after peaking at 85% in the 1950s.[7] Circumcisions attracting a Medicare rebate were performed on 11% of infants in 2013.[9] Potential medical indications for circumcision include true phimosis not resolving with steroid creams, paraphimosis, recurrent balanitis, BXO and boys at high risk of UTIs. However, improved personal hygiene and better management of some of these conditions has led to a reduced medical need for circumcision. Apart from these situations and circumcision for religious reasons, circumcision for social reasons is generally discouraged. A policy statement from the Paediatric and Child Health Division of the RACP recommends against routine infant circumcision.[10]

Circumcision is generally a safe procedure but there are risks of minor complications and some rare but serious complications. In newborns, because of their small blood volume, any bleeding is of major concern and blood loss over 25 mL can be life-threatening.[1] A bleeding circumcision

site can be the presentation of a coagulopathy disorder.[3] Also, because of a newborn's relatively poor immunity, septicaemia from coliform infection on the wound site is a risk. If circumcision is done, it is recommended that it be done after 6 months of age and ideally in an operating theatre under general anaesthetic and with careful surgical technique.[1]

Arguments for routine circumcision include a reduction in risk of UTIs (and their complications) in infancy, reduction of sexually transmitted infections (including HIV) and a possible reduction in the risk of penile cancer in men and cervical cancer in their female partners later in life.

Circumcision reduces the risk of UTIs by about 10-fold.[11] However, because of the low baseline risk, 111 circumcisions would be required to avoid one UTI in boys without abnormal urinary tracts.[10] The reduction in STIs including HIV has been shown to be a benefit in males in high-risk populations (such as men in Africa), but this benefit has not been consistently evident in studies in lower-risk populations such as Australia and New Zealand. While some studies have demonstrated circumcision provides a protective benefit against HPV transmission and subsequent cervical cancer risk, the HPV vaccination has dramatically reduced the baseline risk of these conditions. Cancer of the penis is so rare that the overall benefit offered by circumcision would be very small, and is overshadowed by recent improved penile hygiene in men and management of phimosis. In summary, the RACP guidelines conclude the benefits do not warrant the risks in routine circumstances, but that it is reasonable for parents to weigh up the risks and benefits (after being fully informed), and to make the decision themselves with parental choice being respected.

Complications of circumcision:

- bleeding
- infection (local/septicaemia)
- ulceration of glans/meatus
- meatal stenosis
- penile deformity

Absolute contraindications:

- hypospadias and other congenital abnormalities (the foreskin may be a vital source of skin for subsequent repair)
- chordee (painful dorsolateral curvature of the penis during erection, which interferes with sexual intercourse)
- 'buried' penis
- sick, 'unstable' infants
- family history of bleeding not ruled out in the infant

- inadequate experience of proceduralist

Disorders affecting the urethral meatus

Meatal stenosis

Meatal stenosis or stricture may be congenital or acquired. It may be acquired in the circumcised child due to abrasion and ulceration of the tip of the glans. The incidence can be reduced by the application of moisturising cream for 2 weeks on the glans after circumcision.[1] Uncommon causes are direct trauma during circumcision and irritation from ammoniacal dermatitis. Meatal ulceration predisposes to meatal stenosis. It usually presents as pain during micturition or as slight bleeding on the nappy or underpants. Significant stenosis requires surgical correction by meatotomy.

Catheter trauma is the usual cause in adults.

Hypospadias

Hypospadias is a condition where the urethra opens on the underside or ventral aspect of the penis. It occurs in 1 in 350 males. Hypospadias is classified based on the location of the proximally displaced urethral orifice:[2]

- distal–anterior (located on the glans or distal shaft of the penis and the most common type)
- intermediate–middle (penile)
- proximal–posterior (penoscrotal, scrotal, perineal)

There is often associated chordee (a ventral bending of the penis associated with erection) due to a ‘bowstring’ effect from the lack of tissue on the ventral surface, and also a high incidence of cryptorchidism (10%) and open processes vaginalis or inguinal hernia (9–15%).[2]

Hypospadias may cause the stream of urine to be deflected downwards or splash or drip back along the penile shaft. Unless it is very distal, surgical repair is usually advised, using the available foreskin, and is usually done between 6 and 18 months.[2] Chordee may be corrected at the same time to allow eventual successful sexual intercourse. These boys should not undergo routine circumcision.

Other disorders of the penis

Penile warts

Penile warts are usually multiple fleshy, papillomatous outgrowths, commonly found around the coronal sulcus, the adjacent prepuce and the meatus (see FIG. 105.6). They are caused by

human papillomavirus and usually transmitted sexually. Look for warts within the meatus by allowing gentle dilation of the distal urethra with mosquito forceps.

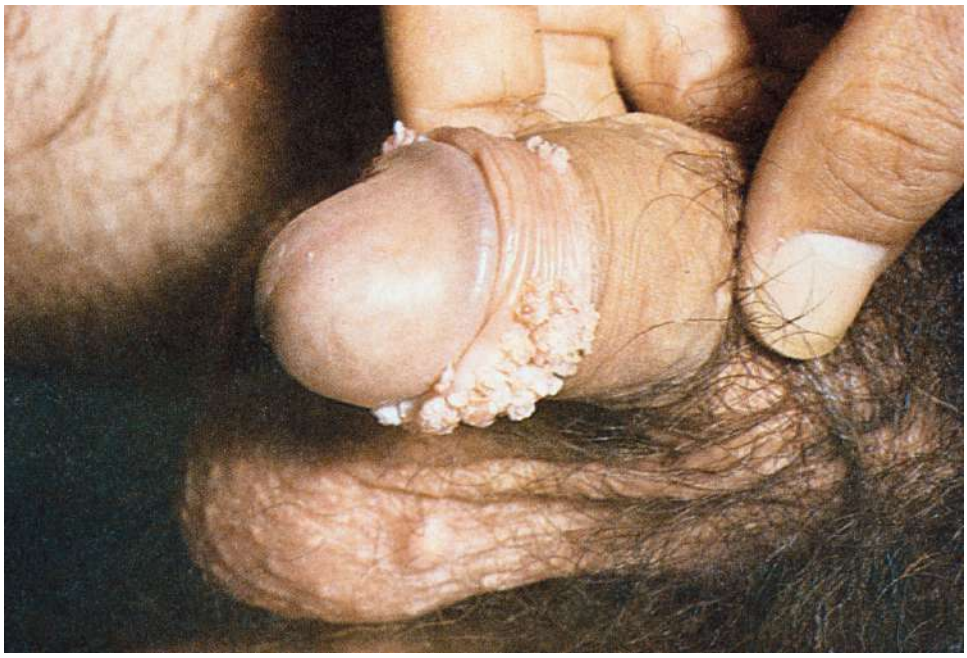


FIGURE 105.6 Penile (genital) warts caused by the human papillomavirus

Treatment is aimed at reducing any pain, bleeding, itch or embarrassment. Treatment options include:[12]

- no treatment
- self-applied treatments at home
- treatment at the surgery or a clinic

The following treatments[13] can be used with or without periodic cryotherapy:

- imiquimod 5% cream topically applied to each lesion, 3 times per week at bedtime (wash off after 6–10 hours) until the warts disappear (usually 8–16 weeks)
- podophyllotoxin 0.15% cream or 0.5% paint topically applied to each wart, twice daily for 3 days followed by a 4-day break, then repeat weekly for 4–6 cycles until the warts disappear

Pearly penile papules

These are very small, regular round lumps (actually angiofibromas) that appear on the corona of the glans of the penis (see FIG. 105.7). They are common and are often first noticed by

adolescent males who should be reassured that they are normal variants. They are not premalignant and require no treatment.[14]

Fordyce spots of the penis: refer to CHAPTER 104 .

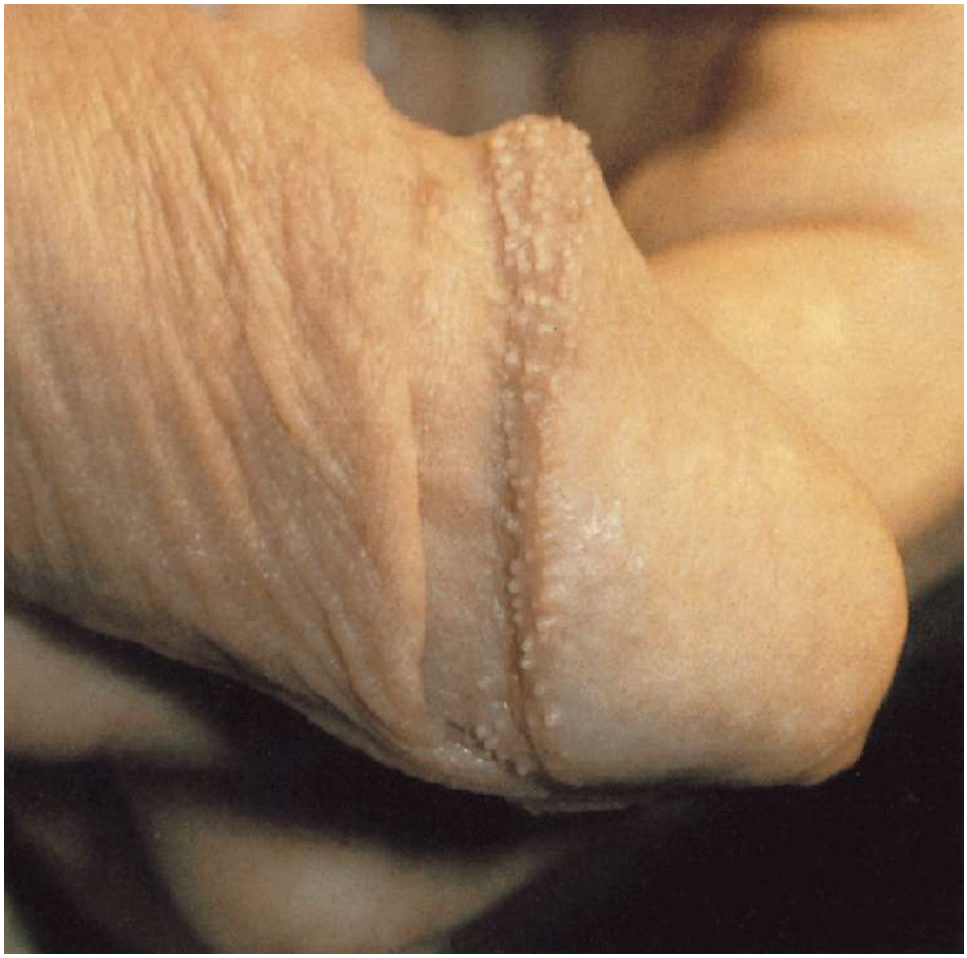


FIGURE 105.7 Pearly penile papules

Penile ulcers

A common cause of penile ulcers is trauma, related to sexual activity, to the frenulum if it is congenitally tight. Such traumatic ulcers may be slow to heal and the frenulum may need surgical division. The ulcers may resemble a venereal ulcer (e.g. syphilitic chancre or herpes simplex). Another important (although rare) cause is cancer of the penis. Various causes are listed in TABLE 105.1 .

Table 105.1 Causes of penile lesions

Non-ulcerative
Balanitis: • *Candida albicans* • diabetes mellitus • poor hygiene
Skin disease: • psoriasis • lichen planus
Fordyce spots
Venereal warts
Ulcerative
Trauma (tender)
Cancer (non-tender)
Herpes simplex (tender)
Syphilis (non-tender)
Chancroid (tender)
Granuloma inguinale (donovanosis)

Cancer of the penis

Cancer of the penis is rare, occurring in fewer than 1 in 100 000 of the male population,[15] though it can be much more common in the developing world. Some 95% of penile cancers are squamous cell carcinomas. Phimosis, smoking, multiple sexual partners and poor hygiene are the main risk factors for penile cancer.[16] There is an association with being uncircumcised and HPV infection, particularly types 16, 18 and 31.[15]

Cancer usually starts as a nodular warty growth (or ulcer) on the glans penis or in the coronal sulcus.[16] Initially it may resemble a venereal wart. Fungal balanoposthitis can be misdiagnosed as cancer. The presenting symptom may be a bloodstained or foul-smelling discharge as the lesion can be hidden by the foreskin. It is usually seen in elderly patients with poor hygiene. Associated lymphadenopathy, which is present in 50% of patients on presentation, may be infective or neoplastic. Metastases to distal sites are uncommon. Refer for management, which includes radiotherapy—usually effective.

Priapism[17]

Priapism is a persistent penile erection that continues hours beyond, or is unrelated to, sexual stimulation. It may follow injected erectile dysfunction treatments. Typically, the corpora

cavernosa are engorged but the corpus spongiosum and glans remain flaccid. It is a medical emergency, as it often leads to fibrosis of the cavernosal tissue and subsequent erectile dysfunction or impotence.

Subtypes of priapism include:

- Ischaemic (veno-occlusive or low flow)—this results from a failure of the detumescence mechanism (relaxation of the outflow of blood from the penis). The corpus cavernosa are rigid and tender to palpation, and the patient is in discomfort. It requires emergency treatment, and there can be subsequent penile oedema, ecchymoses and partial erections.

- Non-ischaemic (arterial or high flow)—this is from an unregulated cavernous arterial inflow. The penis is neither fully rigid nor painful. Trauma is often the cause. It does not require emergency treatment.

- Stuttering—this occurs from intermittent ischaemic priapism, and is manifested by repeated painful erections with intervening periods of detumescence. It may require emergency treatment if the events are prolonged, severe or frequent. Urologist consultation is advised.

The assessment focuses on distinguishing ischaemic from non-ischaemic. Apart from the clinical features mentioned above, cavernosal blood gases can confirm ischaemic priapism.

Duplex ultrasound can also be used, with ischaemic priapism showing little or no flow in the cavernosal arteries, and non-ischaemic showing normal to high blood flow. It can also demonstrate anatomical abnormalities such as a cavernous artery fistula in the perineum from trauma. A full blood count and film can also be part of the assessment, looking for abnormalities such as sickle-cell disease, leukaemia, acute infections or platelet abnormalities. Drug screening can also be performed if suspected.

Emergency treatment for ischaemic priapism can include intracavernous injection of sympathomimetic agents such as phenylephrine as well as aspiration via a 19 or 21 gauge needle into the corpus cavernosum. If this fails, a surgical (cavernoglanular) shunt can be considered.

Peyronie disease[18]

Peyronie disease is a fibrotic process, sometimes associated with Dupuytren contracture, which affects the shaft of the penis and results in discomfort and deformity on erection. It may result from trauma while the penis is erect. It usually affects men between 45 and 60. Typically, the patient presents with painful ‘crooked’ erections. There is abnormal curvature of the erect penis. The penile deformity may prevent satisfactory vaginal penetration. On examination, a non-tender hard plaque may be palpable in the shaft of the penis at the point where the penis curves. Mild cases require reassurance. The problem may increase, remain static or spontaneously lessen over 1–2 years. If there is ongoing discomfort or sexual dysfunction that distresses the patient, surgical treatment by penile plication (a sutured tuck on the opposite side to straighten the deformity) may be warranted. In more severe deformities, incision and grafting of the scar or implants can be considered. Steroid injections are not recommended. Oral vitamin E has been used for treatment with unclear outcomes apart from one positive study.[19]

Chordee

Chordee is ventral or rotational curvature of the penis where the penile head usually curves upwards or downwards. It is a congenital abnormality usually caused by a ventral deficiency in the foreskin. It is usually detected soon after birth to about 18 months of age. It is often associated with hypospadias. The deformity is most apparent on erection. Early referral to a paediatric surgeon is advisable.

‘Fractured’ penis

A ‘fractured’ penis describes sudden rupture of the penile erectile tissue during intercourse, usually with a woman in the superior position, resulting in a snapping sensation with severe pain. The management is urgent urological consultation for possible surgical repair. The disruption can affect the corpus spongiosum (has a better prognosis) or the corpora cavernosa, which may be treated by drainage of the blood clot, in which case permanent erectile dysfunction is a possible complication.

Foreskin injury

Injuries to the penis are not uncommon and one is the entrapped foreskin in the trouser zipper, when attempts to free the zipper aggravate the problem. In the office, cut the zipper from the trousers (see FIG. 105.8) and under local anaesthetic (no adrenaline), crush the zipper with pliers to open the teeth of the zipper and free the foreskin. Another method is to use a scalpel to cut the zipper immediately below the metal tag.

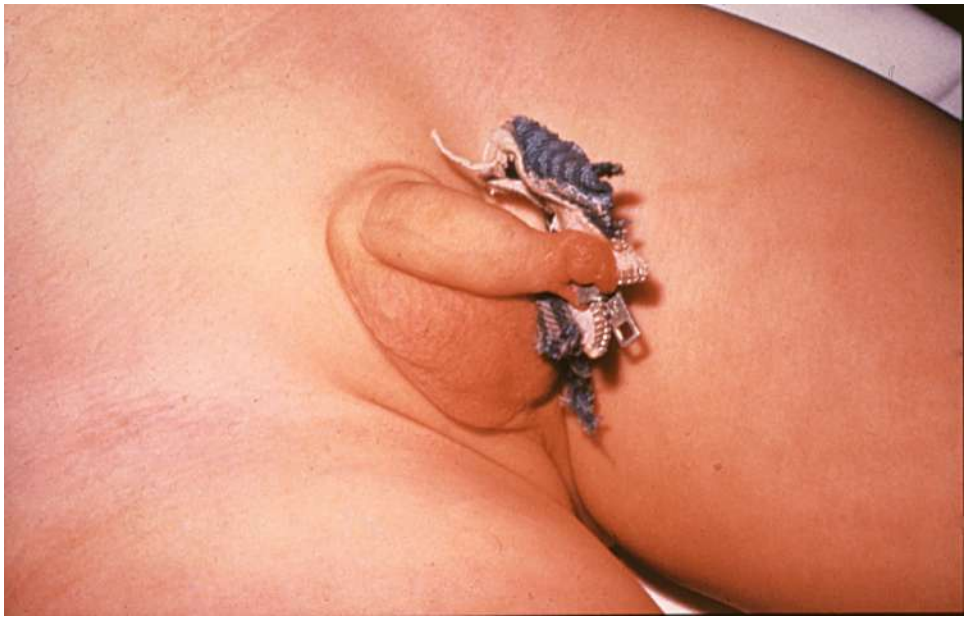


FIGURE 105.8 Entrapped foreskin in trouser zipper

Photo courtesy Bryan Walpole

Erectile dysfunction

Refer to CHAPTER 108 .

Haematospermia

Haematospermia, which is blood in the semen, presents as a somewhat alarming symptom. It is sometimes encountered in young adults and middle-aged men. The initial step is to determine that the blood is actually in the semen and not arising from warts inside the urethral meatus or from the partner.

It usually occurs as an isolated event but can be secondary to urethral warts or prostatitis, or can occur with prostatomegaly or prostatic tumour (especially in elderly patients). If a micro-urine shows no accompanying haematuria, and prostate-specific antigen and blood pressure are normal, reassurance and a 6-week review are appropriate as spontaneous cessation of haematospermia is the rule.

Red flags for haematospermia

- Symptoms lasting longer than 4 weeks
- Palpable lesion in the prostate or along the epididymis
- Recent travel to schistosomiasis-prevalent region

Patient education resource

Hand-out sheet from *Murtagh's Patient Education* 8th edition:

- Circumcision

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106 Disorders of the prostate

Prostate cancer is like golf. You need to play it as it lies. Because the disease is variable, each treatment solution requires a unique strategy.

DR CHARLES 'SNUFFY' MYERS, MEDICAL ONCOLOGIST AND PROSTATE CANCER SURVIVOR

The main function of the prostate gland is to aid in the nutrition of sperm and keep the sperm active. It does not produce any hormones so there is usually no alteration in sexual drive following prostatectomy.

Prostatitis

Prostatitis embraces a group of conditions with voiding discomfort and pain in the prostate referred to the perineum, low back, urethra and testes. It typically affects men aged 25–50 years. Prostatitis usually occurs in the absence of identifiable bacterial growth, when it is termed non-bacterial prostatitis. The prostate may develop acute or chronic bacterial infection. Acute bacterial prostatitis, while uncommon, can be life-threatening if left untreated.[1]

Bacterial prostatitis is usually caused by urinary pathogens—*Escherichia coli* (commonest), *Enterococcus, Proteus, Klebsiella, Pseudomonas* or *Staphylococcus.* Rarely, chronic infections have been shown to be associated with *Chlamydia trachomatis.*[2]

Prostatodynia means the presence of symptoms typical of prostatitis but without objective evidence of inflammation or infection (see TABLE 106.1).

Table 106.1 Classification of prostatitis syndromes

	Prostatic pain	Prostatic rectal examination	Positive urine or prostate secretion culture	Positive prostatic secretion or urine white cells
Acute bacterial	Yes	Very	Yes	Yes

prostatitis		tender, swollen		
Chronic bacterial prostatitis	Often	Normal or indurated	Occasionally	Low counts
Chronic prostatitis/chronic pelvic pain syndrome	Often	Normal	No	Nil or occasional

It is preferable to use the term ‘prostatitis syndromes’ to embrace the three terms used in TABLE 106.1 .

Clinical features of acute bacterial prostatitis

Symptoms

- Fever, sweating, rigors
- Pain in perineum (mainly), back and suprapubic area
- Urinary frequency, urgency and dysuria
- Variable degrees of bladder outlet obstruction (BOO)
- ± Haematuria

Signs

- Fever
- Rectal examination: prostate exquisitely tender, swollen, firm, warm, indurated

DxT dysuria + fever + perineal pain → acute prostatitis

Complications

- Abscess
- Recurrence
- Epididymo-orchitis

- Acute retention
- Bacteraemia/septicaemia

Chronic bacterial prostatitis

Chronic bacterial prostatitis is diagnosed by a history of mild irritative voiding with perineal, scrotal and suprapubic pain. Ejaculatory pain can occur. The gland may be normal on clinical examination or tender and boggy. It should be suspected in men with recurrent UTI (see TABLE 106.2).

Table 106.2 Features of chronic bacterial prostatitis

Difficult to treat
Relapsing infection
Perineal pain
Some leucocytes in expressed prostatic secretions

Investigations

- Urine specimens and expressed prostatic secretions (EPS) obtained after prostatic massage can show excess white cells.
- Culture of the urine or ejaculate may be negative or give low counts.
- Prostatic stones (demonstrated by plain X-ray or transrectal ultrasound) may prevent successful treatment.
- Prostate-specific antigen (PSA): elevation occurs with inflammation and may cause confusion with cancer.

Treatment

Acute bacterial prostatitis[1]

For milder infection, oral treatment for 14 days with trimethoprim or cephalexin is suitable; if culture demonstrates resistance use ciprofloxacin or norfloxacin.[2] For more severe infections (as for pyelonephritis):

amoxicillin (or ampicillin) 2 g IV 6 hourly

plus

gentamicin 4–6 mg/kg/day as a single daily dose until there is substantial improvement, when therapy may be changed to an appropriate oral agent, based on the sensitivity of the pathogen(s) isolated, for the remainder of 10–21 days, depending on clinical response.[2,3]

Urinary retention or abscess formation almost always requires endoscopic deroofing for drainage.

Chronic bacterial prostatitis

Treatment of chronic bacterial prostatitis is made difficult by uncertainty in differentiating it from non-bacterial prostatitis as cultures may grow low counts of what may be normal flora. Some 90–95% of cases of chronic prostatitis are not due to infection.[2] Avoid overtreatment with antibiotics and review regularly. Reassurance is important and it is worth suggesting frequent ejaculation and hot baths. Antibiotics should be used in patients who are culture positive with pus cells, based on culture susceptibility results. Regimes used include:[2]

ciprofloxacin 500 mg (o) 12 hourly for 4 weeks

or

norfloxacin 400 mg (o) 12 hourly for 4 weeks

or

trimethoprim 300 mg (o) daily for 4 weeks

Chronic prostatitis/chronic pelvic pain sydrome[3]

This is the commonest and least-understood form. It is often recurrent and each episode can last several months. Recurrent antibiotic therapy is inappropriate.[2] Perform a thorough genitourinary tract investigation. The symptoms may reflect retrograde passage of urine into prostatic tissue with urate crystallisation. Management is targeted at symptom relief and can include NSAIDs, massage therapy and 5-alpha-reductase inhibitors (if BPH is present).[4] Alpha blockers, which have previously been recommended, are now considered ineffective based on a recent large randomised trial.[5] Emphasise good voiding habits. Avoid straining at the end of micturition. Encourage normal sexual activity and use stress management. Psychological counselling or a pain clinic referral may be appropriate in refractory cases.

Prostate pain syndrome is a symptomatic diagnosis, and a useful assessment tool to monitor the severity, progression and response to treatment is the International Prostate Symptom Score (IPSS).[6]

Practice tip

Consider bladder cancer if persistent dysuria.

Lower urinary tract symptoms (LUTS)

These symptoms can be grouped as voiding symptoms (obstructive) or storage symptoms (irritative).[7] Irritative symptoms may be caused by a bladder problem only. Obstructive symptoms are usually caused by the prostate (which can also cause irritative symptoms). The old term ‘prostatism’ is ill defined and best dropped.

Voiding (obstructive) symptoms

- Hesitancy
- Weak stream
- Postmicturition dribble or irregular stream
- Incomplete emptying/urinary retention
- Straining

Storage (irritative) symptoms

- Urgency
- Urge incontinence
- Frequency
- Nocturia
- Suprapubic pain

Bladder outlet obstruction (BOO)

Symptoms of BOO are present in most men after the age of 60 years. The commonest cause is benign prostatic hyperplasia (BPH). BPH is a histological diagnosis that, strictly, should not be used for symptoms. Only a minority of patients require surgery for relief of obstructive symptoms. Bladder outlet obstruction can also be caused by bladder neck obstruction and urethral sphincter spasm (see FIG. 106.1).

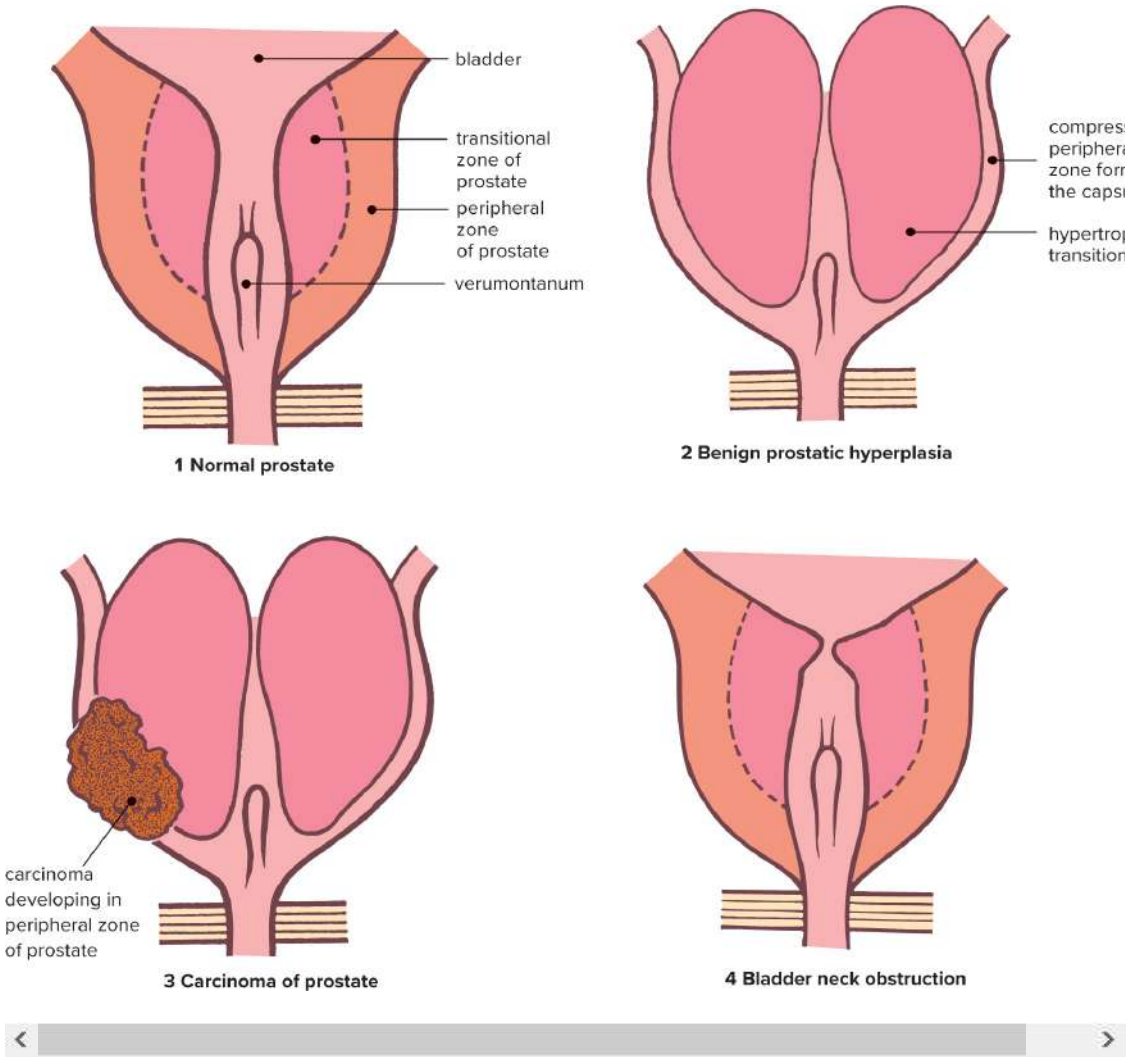

FIGURE 106.1 Diagrammatic comparison of bladder outlet obstruction

DxT poor urine flow + straining to void + frequency → BOO

Clinical features of benign prostatic obstruction

- Hesitancy
- Frequency of micturition
- Urgency

- Nocturia
- Slow interrupted flow
- Terminal dribbling
- Acute retention
- Retention with overflow incontinence (less common)
- Haematuria from ruptured submucosal prostatic veins can occur
- Rectal examination usually detects an enlarged prostate

Note: Small prostate glands can also cause BOO.

The medical history should ideally include an International Prostate Symptom Score (IPSS).[8, 9] The physical examination should include an abdominal examination, a digital rectal examination (DRE) and a genital examination.

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Red flags for LUTS[5]

- Haematuria
- Incontinence, especially at night
- Urinary retention
- Recurrent urinary infection
- Bladder calculus
- Renal impairment
- Hydronephrosis

Refer to a urologist.

Investigations

These include:

- midstream urine: microscopy, culture and sensitivity
- kidney function (urea, electrolytes and creatinine)

- PSA
- urinary ultrasound including a bladder residual volume
- voiding diary (frequency and volume measured for 2–3 days, and should be done in men with daytime frequency or who awaken two or more times a night to void)[7]
- urodynamic study
- urethrocystoscopy

When initial assessment demonstrates the patient having non-bothersome LUTS with or without non-suspicious prostate enlargement, and the patient does not want treatment, it is reasonable not to investigate further.[7] The patient can be reassured and reviewed as necessary. If there are bothersome LUTS, causes such as prostatic obstruction, overactive bladder or polyuria/nocturnal polyuria should be considered. Polyuria is defined as >3 L/24 hours, and nocturnal polyuria as being greater than a third of the 24-hour urine output.

If the initial assessment does reveal these or other concerns, such as an abnormal DRE/PSA, haematuria, recurrent UTI or palpable bladder, the patient should be referred to a urologist before treatment is advised.[7] Further investigations that may be organised by the urologist can include urine flow studies, cystoscopy, possibly an MRI and prostatic biopsy. A flow rate below 15 mL/s suggests obstruction and below 10 mL/s significant obstruction (see FIG. 106.2).

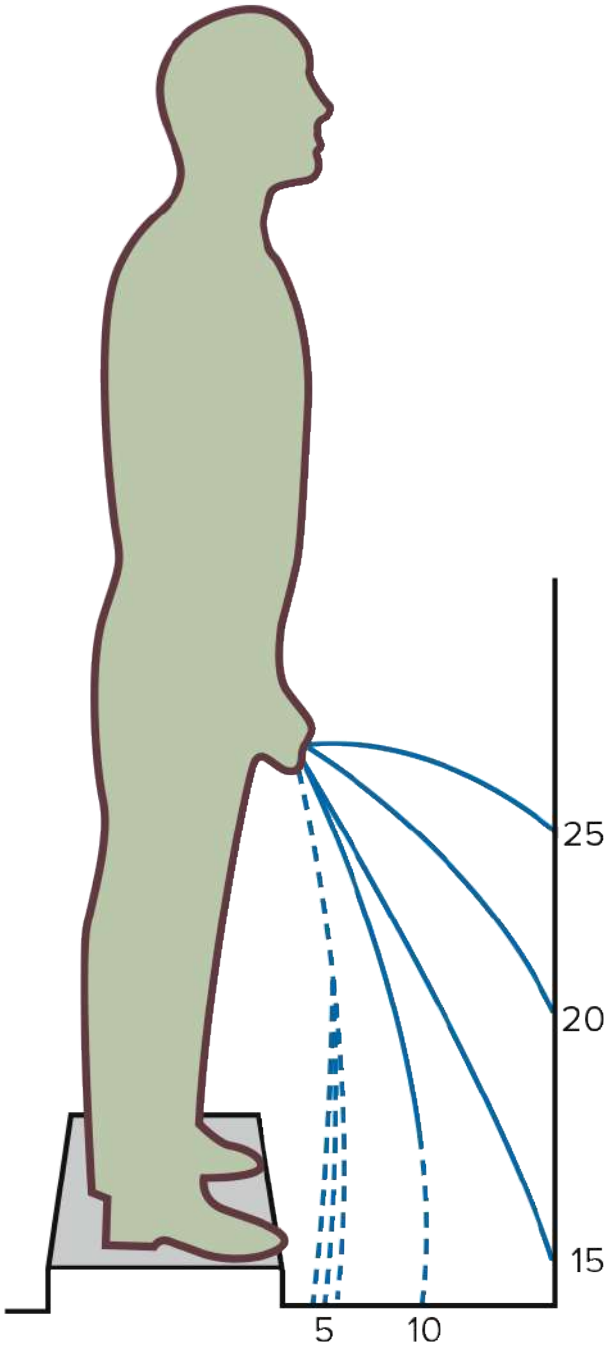

FIGURE 106.2 A visual scale of urinary flow. The numbers signify the flow rate in millilitres per second. When assessing a patient with voiding dysfunction, ask him to indicate which stream is closest to his own.

Complications of prostatic obstruction

- Retention
- Urinary infection
- Bladder calculus formation
- Uraemia

Advice for patients with mild symptoms[10]

- Avoid certain drugs, especially OTC cough and cold preparations.
- Avoid or reduce caffeine and alcohol.
- Avoid highly seasoned or irritative foods.
- Avoid fluids before bedtime.
- Keep active/increase activity.

Treatment

Treatment options beyond basic management and watchful waiting (with yearly review) include medical management and surgical options.

Medical treatment

BOO patients with significant symptoms may be helped with alpha-adrenergic blocking drugs such as alfuzosin, tamsulosin, terazosin and prazosin to inhibit contraction of the smooth muscle in the prostate and bladder neck. Typical doses are tamsulosin 0.4 mg daily and prazosin 0.5 mg bd, or 1 mg nocte after commencing with 0.5 mg nocte. Prazosin can be increased to a maximum of 2 mg bd. Symptoms are not improved by increasing beyond this dose. Side effects can include dizziness, and complications can occur in cataract surgery in men who have used alpha blockers, possibly due to an effect on the smooth muscle that dilates the iris (called the intra-operative floppy iris syndrome),[11] so caution needs to be used here.

The 5-alpha-reductase inhibitors (5-ARIs, e.g. finasteride, dutasteride) reduce prostatic volume by reducing testosterone conversion, and can be used in men with large prostates in association with LUTS. They are not as effective in reducing symptoms as the alpha blockers and can have sexually related side effects such as decreased libido and ejaculatory dysfunction. Urine flow improves by 3 months, plateauing at 6 months, but not to the same degree as with surgery. Two large recent studies support the combined use of alpha blockers and 5-ARIs in the medical management of BOO with large prostates.[12,13] Anticholinergic medication can also be used for symptoms caused by an overactive bladder.

Surgical management

The gold standard surgical treatment for obstruction remains transurethral resection of the prostate (TURP)[7] (see FIG. 106.3). Surgery should be considered for men with moderate to severe symptoms who are keen on intervention and have had the risks and benefits of the procedure fully explained (including failure of relieving symptoms).

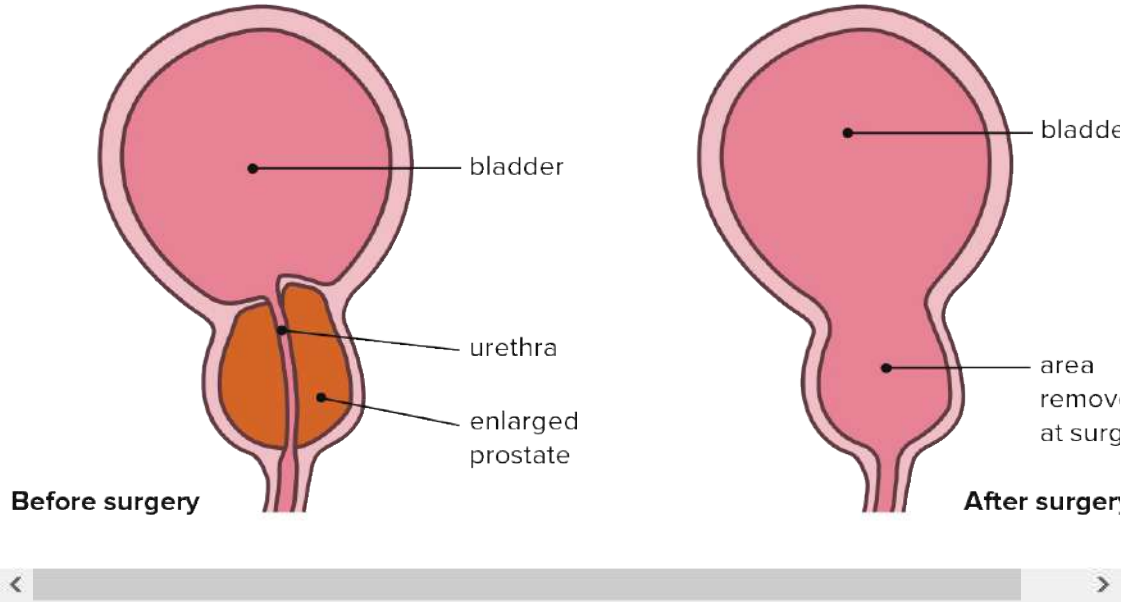

FIGURE 106.3 The process of prostatectomy

Many new surgical techniques are appearing as potential alternatives to TURP.[7] These include minimally invasive procedures that can be done as an outpatient such as transurethral needle ablation (TUNA) or transurethral incision of prostate (TUIP). These may have less risk of complications, but are not as effective at relieving symptoms as TURP and may lack sufficient durability of effect. They are sometimes considered as an option for men with anaesthetic risk or with smaller prostates. Other new surgical techniques such as Holmium laser ablation and laparoscopic and robotic prostatectomy show promise but lack robust long-term data to date. They are also dependent on operator experience and local availability.

Risks involved with TURP include urinary incontinence (settles in all but 1%), urethral strictures (4%) and decline in sexual function.[14,15] Retrograde ejaculation (into the bladder) is the norm, but is not painful or harmful as long as fertility is not an issue.

Absolute indications for prostatectomy include deterioration in kidney function, the development of upper tract dilatation, retention (following drainage and assessment) and bladder stones. Eighty per cent of patients have surgery for bothersome symptoms.

Postoperative guidelines for the patient

- There may be urgency even to the point of incontinence for a few days.
- Bleeding can occur intermittently for 3 weeks, so increase fluid intake.

- Avoid intercourse for 3 weeks.
- Orgasms continue but there is usually no emission with ejaculation. The semen is ejaculated back into the bladder.
- If obstructive problems recur early there may be a stricture.

Note:

- Persisting postoperative frequency bothers about 15% of patients
- 10–15% reveal unsuspected cancer

Drugs causing LUTS

Certain drugs can cause LUTS due to the effect of the drug on the bladder. It is very important for the family doctor to enquire about these drugs when evaluating such a patient. The problem is mainly an adverse effect of drugs with anticholinergic activity.

Anticholinergics

- Atropine and hyoscine compounds, for example:
  - isopropamide
  - mazindol
  - phenothiazines
  - dicyclomine
  - propantheline
  - other belladonna alkaloids
- Antidepressants:
  - especially tricyclic compounds
- Antiparkinson agents, for example:
  - amantadine
  - benzhexol
  - benztropine

biperiden

orphenadrine

procyclidine

Beta-adrenoceptor agonists

- Ephedrine
- Salbutamol
- Terbutaline
- OTC preparations (mainly for coughs and colds—e.g. sympathomimetics including ephedrine)

Prostate cancer

Prostate cancer is the commonest malignancy in men and the third commonest cause of death from malignant disease in Australia.[16] It is very much age dependent, with the risk increasing with age. Prostate cancer is rare before the age of 50 years, but by the age of 80 years, 80% of men have histologic carcinoma within the gland (but most are dormant—men 'die with' the cancer rather than 'die of' it).[17] The lifetime risk of being diagnosed by the age of 85 years is 1 in 5, but the risk of dying of prostate cancer by this age is 1 in 25.[16] Those men with a new diagnosis have a 92% chance of surviving for at least 5 years compared with the general population.[16] While this may sound reassuring, a younger man with a new diagnosis will have a higher risk of dying prematurely from prostate cancer as the cancer has a longer time available to progress.[18]

A family history increases risk, particularly with a brother or father diagnosed with prostate cancer before the age of 65 years.[19] There is also increased risk for men with a first-degree relative with familial breast cancer (*BRCA1* or *BRCA2*).[19] Prostate cancer may be asymptomatic, even when it has extended beyond the prostate. It usually commences in the peripheral part of the gland. There are significant racial differences in the frequency that tend to change with migration, indicating that prostate cancer reflects environmental influences (and possibly dietary fat).[20]

Clinical features

Unsuspected cancers are often detected by the tumour marker PSA (a glycoprotein) or histologically after TURP. Clinical prostate cancer presents typically with rapidly progressive symptoms of lower urinary tract obstruction or of metastatic spread, especially to bone (pelvis and vertebrae).[21] Symptoms include BOO, acute retention, back or other bony pain, haematuria and uraemia, tiredness, weight loss and perineal pain.

Digital rectal examination (DRE) may reveal a nodule. Locally advanced cancer typically reveals

a hard, nodular and irregular gland. The tumour may be large enough to obliterate the median sulcus. The borders may lack definition. On the other hand, with cancer, the prostate may feel normal.

Signs of abnormal prostate (DRE):

- hard lump
- asymmetry
- induration
- loss of median sulcus

Investigations

Blood analysis

- PSA:

  can be elevated without cancer (e.g. BPH, exercise, infection, instrumentation, recent ejaculation)

  if measured, should be accompanied by a DRE[22]

  is prostate specific, not prostate cancer specific

PSA guidelines (ng/mL)

Individual laboratory reference values may vary from the figures stated:

<4: normal (but in 15–25% of cancers)

4–10: intermediate

>10: strongly suggestive of cancer

>20: suggests metastatic spread

Other PSA parameters:[23]

- Age-specific PSA reference range:

  40–49 years: 0.25–2.5 ng/mL

50–59 years: 0.25–3.5 ng/mL

60–69 years: 0.3–5.5 ng/mL

70–79 years: 0.3–7.5 ng/mL

- Free/total PSA ratio: BPH tends to have more variable PSA concentrations due to intermittent physical disturbance of the gland and subclinical prostatitis. This leads to ‘showers’ of inactive (non-protease) free PSA. Prostate cancer, however, constantly increases bound active PSA so the F/T PSA ratio tends to be lower (below 8%) in prostate cancer. This ratio has been suggested as being useful in men with moderately raised PSA levels (4–10 ng/mL).
- Above age-related median PSA. This has been postulated as a technique for selecting out those men who should have their PSA monitored more closely.
- The rate of PSA changes. Otherwise known as PSA kinetics, these include the ‘PSA velocity’ (the rate of change in PSA levels) and ‘PSA doubling time’ (the time taken for the PSA to double). A high velocity or a short doubling time may suggest a urology review is indicated.

The role of all of these parameters remains controversial, as does whether we should be screening for prostate cancer in low-risk men at all.

Screening

The RACGP *Guidelines for Preventive Activities in General Practice*[22] state that screening for prostate cancer is not recommended unless:

1. the man specifically asks for it *and*
2. he is fully counselled on the pros and cons

Because population-wide screening is not encouraged, GPs are not advised that they should raise the issue of PSA screening while doing a health check or routine blood test.

Similar recommendations have been made by other authorities.[24] The latest NHMRC-approved guidelines are:[25]

- men should be offered the opportunity to consider and discuss the benefits and harms of PSA testing before making the decision whether or not to be tested
- the harms of PSA testing may outweigh the benefits, particularly for men aged ≥70
- men at average risk of prostate cancer who decide to undergo regular testing should be offered PSA testing every 2 years from age 50–69, and offered further investigation if the total PSA is greater than 3.0 mg/mL
- men with a family history of prostate cancer who decide to be tested should be offered PSA testing every 2 years from age 40/45–69, with the starting age depending on the strength of

their family history

- digital rectal examination (DRE) is not recommended in asymptomatic men as a routine addition to PSA testing in the primary care setting but remains an important part of assessment in the higher-risk specialist setting.

Fully counselling a man can be challenging. A useful guide[26] on the outcomes of screening 1000 men aged 60 (with no significant family history and screened annually for 10 years) would result in the following:

- 1–2 men will avoid death from prostate cancer before the age of 85
- 2 men will avoid metastatic prostate cancer before the age of 85
- 85 men who do not have prostate cancer will have a biopsy
- 28 men will experience a side effect from the biopsy they consider a moderate/major problem
- 28 men will be diagnosed with prostate cancer, many of whom would have remained asymptomatic
- 25 men will undergo cancer treatment, many of whom would have remained asymptomatic
- 7–10 men will develop persistent impotence and/or urinary incontinence, and some will develop persistent bowel problems due to the treatments

Core biopsy

A urologist will consider biopsy guided by transrectal ultrasound or a transperineal approach (under general anaesthesia) if the DRE is positive or if the PSA is elevated, depending on the man's individual risk profile, clinical findings and PSA levels. A biopsy is the only certain measure of diagnosis.

Grading and staging

A good way of explaining to patients about grading and staging is to say 'the grade is how aggressive the cancer is, and the stage is how much there is and how far it has spread'.

From the biopsy, the pathologist determines the grading using the Gleason score (see TABLE 106.3 and FIG. 106.4). The score is based on the addition of the grading of the 2 most common types of cells found in the samples, graded from 1 (well differentiated) to 5 (most dysplastic). Therefore the lowest score possible is 2 and the highest 10.

Table 106.3 Gleason score and risk of cancer

Gleason score	Threat from cancer
2–4	Low
5–6	Moderate
7	Intermediate
8–10	High

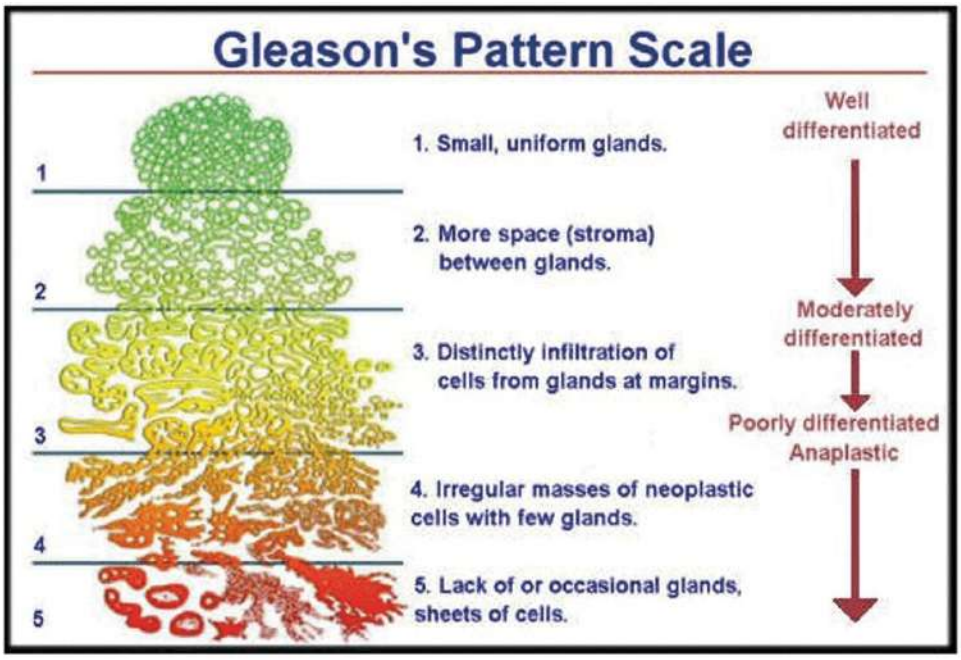

FIGURE 106.4 Gleason pattern (grading) scale of prostate cancer[27]

Source: Reproduced with permission from Gleason DF. Histologic grading and clinical staging of prostatic carcinoma. In Tannenbaum M, Urologic Pathology: The Prostate. Philadelphia, PA: Lea and Febiger, 1977: 171–97

Staging[27,28]

Clinical staging is based on five sources of information:

- the extent of the primary tumour (T category)
- whether the cancer has spread to nearby lymph nodes (N category)
- the absence or presence of distant metastasis (M category)
- the PSA level at the time of diagnosis
- the Gleason score, based on the prostate biopsy (or surgery)

Local prostate gland disease (T1–T2) is based on the DRE and transrectal ultrasound (TRUS) with T3 to T4 indicating spread beyond the capsule. Stages T1 and T2 have a good prognosis, and some T3 tumours can be cured. Whole-body radionucleide bone scanning, CT, MRI scanning and surgical pathology results also assist staging. Axial skeleton metastases are involved in 85% of patients who die of prostate cancer.[29]

After the TNM status is determined, this information is combined with the PSA and Gleason scores in what is termed *stage grouping*, from I (the least advanced) to IV (the most advanced). This will help determine treatment options and prognosis. This information can also be put into nomograms (see www.nomograms.org) by either the doctor or patient to clarify the patient's status and options.

Treatment[30,31,32]

Deciding on treatment options for prostate cancer needs to take into account not only the grade and stage, but also the age and general health/life expectancy of the patient, available treatments and, importantly, patient preferences. Psychosocial support (both at the time of diagnosis and subsequently) and appropriate patient education will help make these patient preferences more clear.

Options for localised prostate cancer include radical prostatectomy, radiotherapy and no active treatment—termed 'active surveillance', monitoring for changes such as a rising PSA or deteriorating biopsy results. Most of the evidence points to there being little difference (when the reduction in cancer risk is offset against the potential negative consequences of the interventions) in long-term outcomes between the three different approaches. This, however, needs to be put into the context of the individual patient's situation, his clinical presentation and his preferences, so active interventions may well be warranted.

Radical prostatectomy is most likely to benefit the man with a relatively long life expectancy (>10 years), no significant surgical risk factors, a low volume stage and a low PSA and who, after being informed of the risks and benefits, prefers surgery. Approaches include radical perineal, laparoscopic and robot-assisted prostatectomy. Major complication issues include incontinence and impotence, and the risk of these will vary depending on the surgical approach and the patient's age and health.

Radiotherapy is also likely to benefit those with a long life expectancy and low-volume low PSA, who have a moderately (or worse) differentiated tumour and who, after being informed of the risks and benefits, prefer external beam or interstitial radiotherapy (brachytherapy). The main adverse reactions from external radiotherapy are faecal urgency and diarrhoea together with urine frequency. Impotence is common for up to 2 years after external beam radiotherapy.

Active surveillance is often used for those with a low life expectancy, or those who prefer to avoid the possible complications of surgery or radiotherapy. PSA monitoring is done for surveillance, or for follow-up after treatment with curative intent.

For locally advanced or metastatic disease, androgen deprivation is the cornerstone of treatment,

the options being:

bilateral orchidectomy

or

daily anti-androgenic tablets, for example:

cyproterone acetate (Androcur)

flutamide (Eulexin)

bicalutamide (Cosudex)

abiraterone (Zytiga)

or

luteinising hormone releasing hormone (LHRH) agonists: depot injections of LHRH analogues, for example:

goserelin (Zoladex)

leuprorelin acetate (Lucrin, Eligard)

Treatment combinations for low volume metastatic prostate cancer may prolong life, for example:

- orchidectomy *plus* flutamide
- LHRH agonists *plus* flutamide or bicalutamide—LHRH agonists cause an initial surge of testosterone so a preliminary anti-androgenic agent is advised to prevent a flare in the cancer.

As well as androgen deprivation, radiotherapy to bony metastases or local disease and other adjuvant therapy options can be considered.

Complementary medicine

Among the disorders most widely promoted as benefiting from natural remedies are those of the prostate, though none has enough evidence to warrant its recommendation.[6] Herbal remedies that have been widely used include saw palmetto (*Serenoa repens*), stinging nettle (*Urtica dioica*), African prune (*Pygeum africanum*, syn. *Prunus africana*), willowherb (*Epilobium*) and cernilton.

Saw palmetto has been used widely, especially in Germany, with initial studies suggesting efficacy in LUTS consistent with BPH. However, a recent Cochrane review concludes it is not more effective than placebo.[33]

BPH has been treated with isoflavone phytoestrogens. A weak oestrogen agonist effect in males

may antagonise the growth-promoting effects of androgens on the prostate. Epidemiological data indicate that in countries such as Japan where isoflavone diets are prevalent, prostatic enlargement occurs less with ageing. The most widely used source of phytoestrogens is soy protein. It is also present in lentils, chickpeas, some variations of beans and alfalfa sprouts.

A variety of nutrients may play a role in the development and progression of prostate cancer. There is evidence from some epidemiological studies that selenium, leucopenes (from tomato and tomato products), vitamin D, vitamin E, calcium and green tea in the diet protect against cancer of the prostate, though their true role remains largely unclear.[34]

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Part 9 Sexual health

107 The subfertile couple

You notice that the tabetic has the power of holding water for an indefinite period. He is also impotent—in fact, two excellent properties to possess for a quiet day on the river.

DR DUNLOP, TEACHING AT CHARING CROSS HOSPITAL, 1913

Infertility is defined as the absence of conception after a period of 12 months of unprotected sexual intercourse.[1] The inability to conceive can be a very distressing and emotional problem for a couple, who need considerable care, empathy and relatively rapid investigation of their problem. In assessing a couple with the problem of subfertility, it is appropriate to involve both partners in the consultation.

Human fertility depends on a complex series of events, including:[2]

- production of gametes (sperm and oocytes) capable of fertilisation
- release of the oocyte into the fallopian tube after ovulation
- the right number of sperm have to be placed in the right place at the right time
- implantation and development of the embryo in the hormonally primed uterine mucosa
- maintenance of the growing fetus in the uterus until pregnancy is full term

As a general rule, the major factors limiting fertility are approximately one-third female, one-third male and one-third combined male and female. In addition, there are couples who fulfil the three primary fertility factors (egg, sperm and tubes) but do not conceive. This is known as unexplained (idiopathic) subfertility and accounts for 10–20% of subfertile couples.

Key facts and checkpoints

- Infertility affects about 10–20% (1 in 7) of couples.[2]
- Fertility in women and men declines naturally with age.
- After the age of 32, female fertility decreases by 1.5% per year.
- Increasing female age is a common cause of decreased fertility.
- The main identifiable causes of male infertility are failure of spermatogenesis, failure of sperm delivery, impaired sperm quality and sperm autoimmunity.[2]
- Female factors include ovulation dysfunction, tubal disease, uterine disorders and peritoneal pathology.[3]
- Polycystic ovarian syndrome (PCOS) is the most common cause of ovulatory dysfunction.[4]
- In up to 20% of couples, no apparent cause is identified (idiopathic).[2]
- Assisted reproductive technology (ART) helps the majority of subfertile couples to achieve pregnancy, although success rates reduce with increasing maternal age.[5]

Physiological factors[6]

Male fertility

Fertility in the male requires:

- normal hypothalamic function producing gonadotrophin-releasing hormone (GnRH)
- normal pituitary function producing the gonadotrophin hormones—follicle stimulating hormone (FSH) and luteinising hormone (LH)
- normal seminiferous tubule and Leydig cell function
- normal sperm transport and delivery

Facts about sperm viability:

- the maximum number of viable sperm is found in the ejaculate after a 48-hour abstinence
- after entering receptive cervical mucus, sperm are capable of fertilising an egg for up to 5 to 6 days

- sperm survive for less than 30 minutes in the vagina due to an acidic environment

Female fertility

Fertility in the female requires:

- normal function of the ovulatory cycle, which requires:

  normal hypothalamic-pituitary function producing the hormones GnRH, FSH and LH

  normal ovarian function with follicular response to FSH and LH (see FIG. 107.1)

  appropriate prolactin levels (which are normally low); excessive prolactin secretion (hyperprolactinaemia) causes anovulation

- normal tubal transport and access of the ovum to incoming sperm
- receptive cervical mucus
- normal uterus to permit implantation of the fertilised ovum

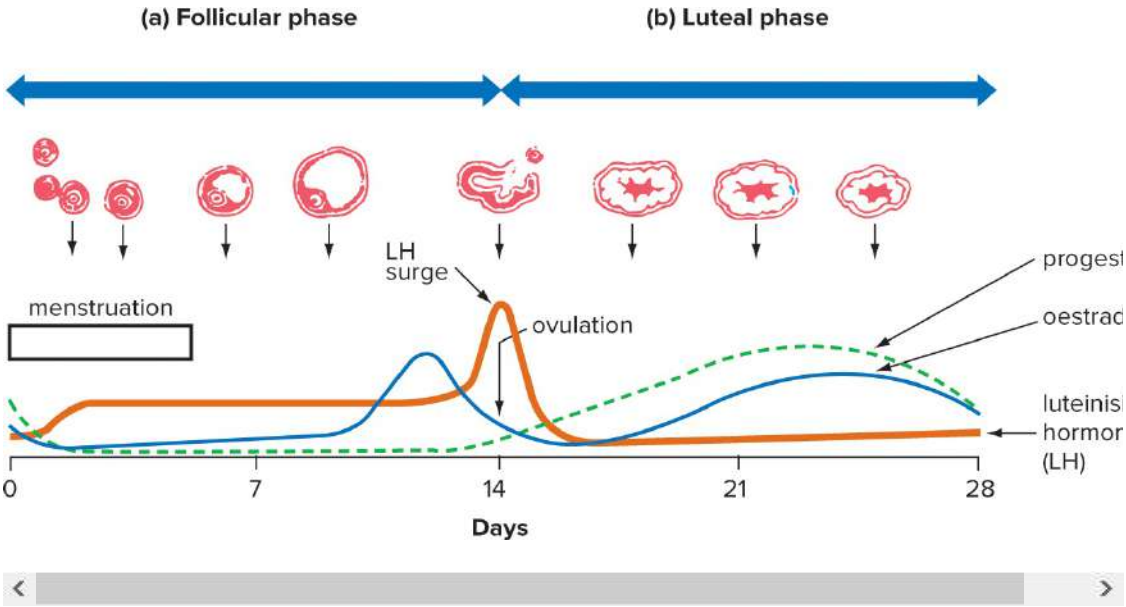

FIGURE 107.1 The normal ovulatory cycle: ovulation occurs at 14 days, 36 hours after the LH surge starts

Probabilities of pregnancy

About 50% of couples, having unprotected intercourse at least twice a week, will probably achieve pregnancy in 6 months, 85% in 1 year and 95% in 2 years.[6]

Causes of infertility

Significant causes of infertility are summarised in TABLE 107.1 and illustrated in FIGURE 107.2 .

Table 107.1 Causes of subfertility[3]

Female factors

Ovulation dysfunction:

- hypothalamic/pituitary disorders
  - suppression of the hypothalamic-pituitary axis from stress, chronic illness, extreme exercise, malnutrition or eating disorders
  - hyperprolactinaemia
  - hypothalamic or pituitary tumours
- PCOS
- ovarian failure
  - premature ovarian failure
  - secondary to chemotherapy or radiotherapy, ovarian surgery or autoimmune disease
- other endocrine disorders
  - thyroid dysfunction
  - hyperandrogenism

Tubal disease:

- PID
- previous ectopic pregnancy
- previous tubal ligation
- tubal obstruction

Uterine abnormalities:

- fibroids
- endometrial polyps
- congenital (e.g. septate uterus, bicornuate uterus)
- acquired (e.g. Asherman syndrome)

Peritoneal pathology:

- endometriosis
- adhesions
- previous peritonitis
- inflammatory bowel disease

Endometrial functional abnormalities:

- coagulation disorders or immunopathology, e.g. SLE, antiphospholipid syndrome

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Male factors[7]

Pre-testicular:

- hypogonadotropic disorder (e.g. Kallman syndrome)
- hyperprolactinaemia

Testicular:

- congenital cryptorchidism (maldescent)
- inflammation (e.g. mumps orchitis, infection, trauma, torsion)
- antispermatogenic agents:
    - chemotherapy
    - drugs
    - radiation
    - heat
- Klinefelter syndrome (46 XXY)
- antisperm antibodies

Post-testicular:

- sexual dysfunction
- retrograde ejaculation
- obstruction:
    - vasectomy
    - congenital absence of the vas deferens, consider cystic fibrosis
    - ejaculatory duct obstruction or seminal vesicle dysfunction

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Couple factors

Joint subfertility

Psychosexual dysfunction

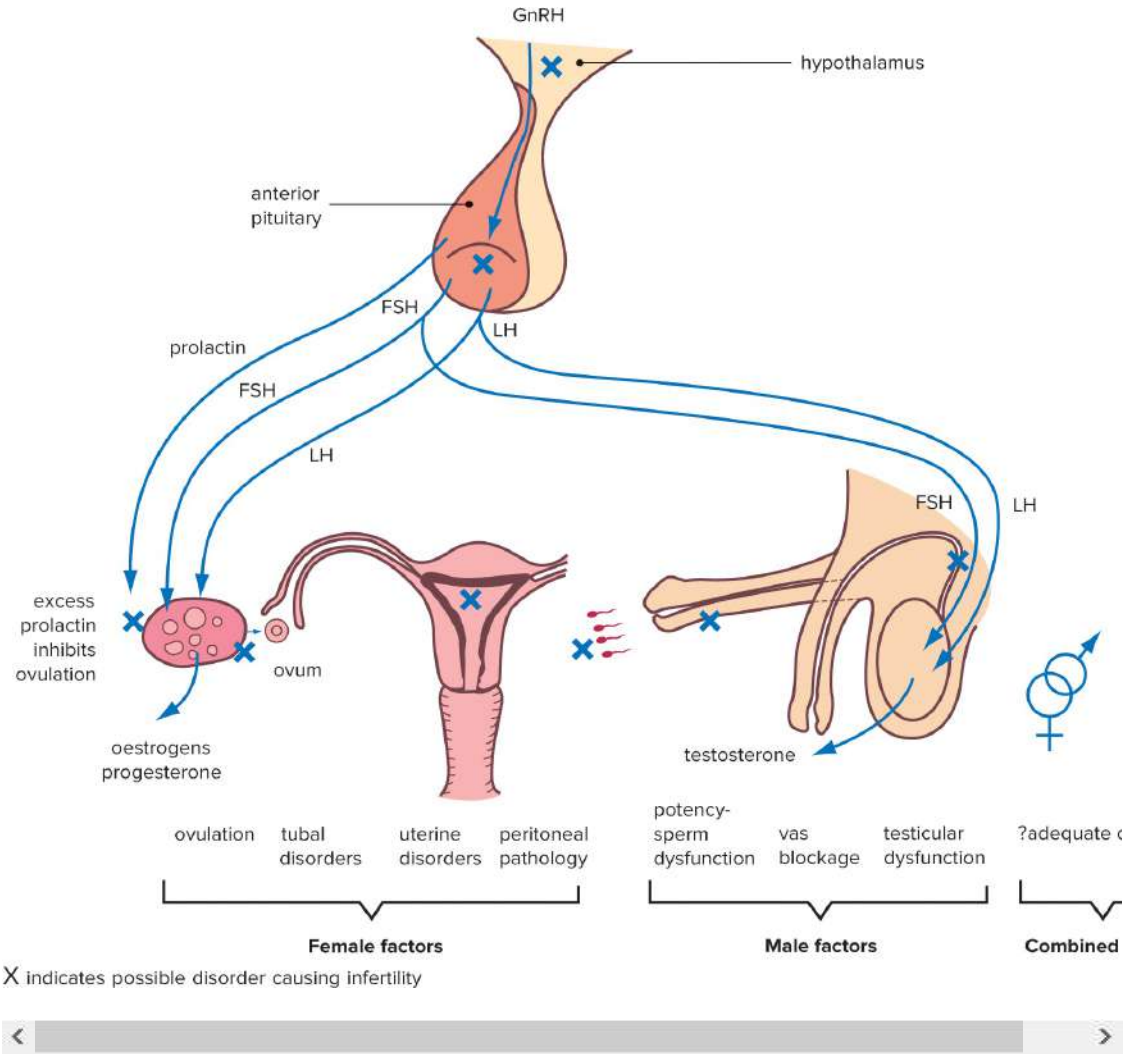

FIGURE 107.2 The major factors involved in subfertility

Source: Adapted from Kumar and Clarke[8]

A diagnostic approach

History

Ask each partner for their age and take a general medical history, including family history, medication and drug use. Enquire about previous pregnancies in current or past relationships and outcomes.

The man

- Sexual function (problems with erections, ejaculation)

- Age of puberty
- Previous testicular problems/injury (e.g. orchitis, trauma, undescended testes, torsion)
- Past history (PH) of STIs
- PH of mumps
- PH of urethral problems
- Genitourinary surgery (e.g. hernia, vasectomy reversal)
- Occupational history (exposure to heat, pesticides, herbicides)
- Medications and drug use:
    - alcohol
    - smoking
    - marijuana
    - anabolic steroids
    - chemotherapy
    - radiotherapy
    - aminoglycoside antibiotics
    - sulfasalazine
    - cimetidine/ranitidine
    - colchicine
    - spironolactone
    - antihypertensive agents
    - narcotics
    - phenytoin
    - nitrofurantoin

The woman

- Onset of menarche

- Menstrual history
- Symptoms of ovulation (cervical mucus changes, mittelschmerz)
- Symptoms of endometriosis (dysmenorrhoea, pelvic pain, dyspareunia)
- PH of STIs and pelvic infection
- Previous contraception use/IUD use
- PH of intra-abdominal surgery (e.g. appendicitis, ovarian cyst)
- PH of genitourinary surgery
- Medications and drug use:

    alcohol

    smoking, especially >20/day

    past contraception, especially depot provera

    anabolic steroids

Combined history

- Time trying to conceive
- Frequency and timing of intercourse
- Attitudes to pregnancy and subfertility
- Expectations for the future

Examination

A general assessment of body habitus, BMI, general health and secondary sexual characteristics should be noted in both man and woman.

The man

- Secondary sexual characteristics; note any gynaecomastia
- Genitalia

    size and consistency of the testes—can compare to an orchidometer: normal range 15–35 mL (average 18 mL); small in Klinefelter syndrome (approx. 7–8 mL)

palpate epididymis and vas (present and non-tender is normal)

evidence of varicocele

PR: check prostate

note penis and location of urethra (always retract the foreskin for examination)

The woman

- Secondary sexual characteristics
- Thyroid status
- Note skin for acne, hirsutism
- Vaginal and pelvic examination:

  assess uterus and ovaries (normal—present, mobile and non-tender)

  the adnexae (any masses)

Investigations

These are usually performed after referral but the family doctor should organise initial investigations to assess where to refer (e.g. andrologist, endocrinologist, gynaecologist or fertility specialist).

Initial investigations

Male—semen analysis[7]

Collection should be made directly into a sterile container 2–3 days after sexual abstinence. If collected at home, semen should be kept at body temperature during transport to the laboratory and examined within 1 hour of collection. A repeat test in 1–3 months is indicated if the first is abnormal.[7]

- Normal values (based on WHO criteria):

  volume ≥1.5 mL (average 2–6 mL)

  pH ≥7.2

  concentration ≥15 million sperm/mL

  total sperm count ≥39 million

progressive motility ≥32%

vitality ≥58%

normal forms ≥4%

leucocytes <1.0 × 10^6/ml

Female—ovulation status[3]

In most cases, regular menstrual cycles between 26 and 34 days, with appropriate variation in cervical mucus (thin at mid-cycle and thicker in the luteal phase) suggest ovulation is occurring. If ovulatory dysfunction is suspected, the following investigations are appropriate:

- serum LH, FSH and oestradiol (help identify primary hypothalamic-pituitary failure or dysfunction and primary ovarian disease; see TABLE 107.2)
- midluteal phase progesterone (day 21 of 28-day cycle, or 7 days before next expected period); >30 nmol/L can be used to confirm ovulation
- androgens (free testosterone, SHBG, free androgen index (FAI), 17-OH progesterone)
- thyroid function tests (TSH <2.5 is ideal prior to conception)
- serum prolactin
- transvaginal high-resolution ultrasound (day 5–9 of cycle) for 'antral follicle count'—allows assessment of ovarian reserve, ovarian pathology or uterine structural abnormalities.
- anti-Müllerian hormone (AMH)—a predictor of ovarian function and reserve (is age-dependent, can be influenced by chronic disease and hormonal contraception, may require specialist input for interpretation)

Table 107.2 WHO classification of ovulatory dysfunction[9]

Group 1	Hypothalamic-pituitary failure	Low FSH, LH (e.g. anorexia nervosa)
Group 2	Hypothalamic- pituitary dysfunction	Normal FSH (e.g. PCOS)
Group 3	Ovarian failure	High FSH (e.g. ovarian failure)

Further investigations[3]

Male

If azoospermia or severe oligospermia (usually under specialist guidance):

- serum FSH level (if 2.5 times normal, indicates irreversible testicular failure)
- LH
- testosterone
- prolactin
- antisperm antibodies (in serum, semen or directly bound to sperm)
- sperm function tests
- genetic testing:
    - karyotype
    - Y chromosome microdeletions
    - cystic fibrosis gene mutation
- ultrasound of the scrotum ± urogenital tract
- post-ejaculatory urine analysis (for retrograde ejaculation)

Female[10]

- Routine preconception screening
- Thyroid antibodies (TPO antibody-positive females have a higher incidence of subfertility)
- Chlamydia (first-pass urine NAAT) if indicated
- Offer genetic carrier testing for fragile X gene mutation, cystic fibrosis and spinal muscular atrophy

Specialised investigations:

- hysterosalpingo-contrast-ultrasonography (Hy-Co-Sy)
- hysteroscopy/laparoscopy
- tubal dye studies

Management principles

- Both partners should be involved in management decisions since fertility is a couple's issue.
- All couples should be encouraged to optimise their general health through lifestyle changes such as weight optimisation, exercise, smoking cessation and minimised alcohol intake.
- Infertility can cause considerable emotional stress, including the taking or placing of blame by a partner and subsequent feelings of guilt. Sensitive and empathetic support is essential. This may include couples counselling.

Fertility awareness

Couples may require the following education on timing of intercourse:

- it is ideal to have intercourse every 2 days in the week before the expected time of ovulation[3]
- ova can survive up to 24 hours and sperm up to 5 days within the female genital tract
- ovulation generally occurs 14 days before menstruation
- ovulation may be identified with cervical mucus changes, when mucus becomes more slippery or like egg white
- the chance of fertilisation is best on the day of ovulation or during the time of the slippery sensation
- 2 days after ovulation, basal temperature rises slightly, around ¼–½ a degree
- recording temperature every morning for a few months can show if ovulation is regular and help predict the day of ovulation
- many women find fertility tracking websites or apps a useful aid
- urine-based ovulation detection kits are available but can be difficult to interpret
- for women with irregular periods, ovulation tracking may be of value through fertility laboratories in the treatment setting

'Fertility awareness practitioners' are available for couples interested in pursuing natural fertility methodology as an alternative to assisted reproductive technology.

Polycystic ovarian syndrome[4]

PCOS is a common condition, present in 12–18% of women of reproductive age.[11] It should be noted that it is not the same as polycystic ovaries, which occur in approximately 25% of

asymptomatic, normal women.[12]

General features

Patients may present with the following:

- ovulatory dysfunction—irregular menstrual cycle, subfertility, oligomenorrhoea, anovulation
- androgen excess—acne, hirsutism and male-pattern balding
- metabolic features—upper truncal obesity, impaired glucose tolerance, dyslipidaemia, diabetes
- psychological symptoms—anxiety, depression, eating disorders

Aetiology[2]

Polycystic ovarian syndrome is typically associated with insulin resistance. Insulin resistance and hyperinsulinaemia drive ovarian androgen production and suppress sex hormone–binding globulin (SHBG), leading to greater bioavailability of androgens. There is a strong hereditary basis and it is more common in Aboriginal and Torres Strait Islander and South-East Asian women. The onset can be triggered by environmental factors, particularly weight gain.

Diagnosis/investigations[13]

A diagnosis of PCOS can be made if a woman presents with:

- irregular periods

and

- hyperandrogenism—clinical *or* biochemical:

    clinical—hirsutism, acne, male-pattern balding

    biochemical—elevated free testosterone or FAI

Irregular periods are considered normal in the first year post menarche. Irregular periods are defined as:

- >90 days >1 year post menarche
- <21 or >45 days in the first 1–3 years post menarche
- <21 or >35 days >3 years post menarche
- primary amenorrhoea by age 15 or > 3 years post breast development

If only irregular cycles *or* hyperandrogenism are present, a pelvic ultrasound confirming the

presence of polycystic ovary morphology (at least 10 follicles in each ovary) may also confirm the diagnosis.

Note: Pelvic ultrasound is not recommended <20 years due to the high incidence of polycystic ovary morphology in adolescents.

Differential diagnoses include:

- thyroid dysfunction
- hyperprolactinaemia
- premature ovarian insufficiency
- CAH, Cushing syndrome, adrenal tumours
- hypogonadotrophic hypogonadism

Suggested screening for all women with PCOS

- Smoking history
- Blood pressure
- Fasting blood glucose levels
- Oral glucose tolerance test (every 2 years in women with additional risk factors)
- Llipid profiles

Management strategies

First-line

- Lifestyle modification—weight reduction and exercise (even 5% weight loss may restore normal ovarian function)
- Consider PCOS support group or psychological support

For women with subfertility

- Screening and treat if necessary:

    impaired glucose intolerance and diabetes mellitus

    hyperlipidaemia

    hypertension

- Primary treatment of insulin resistance:

  metformin

- Ovulation induction—clomiphene/letrozole/gonadotrophins
- Laparoscopic ovarian surgery/drilling (second-line after medical treatment)
- Assisted reproductive technology (ART)

Counselling the subfertile couple[14]

The counselling of subfertile couples has to be adapted to the level reached by the couple along the infertility pathway. The needs of each couple may be very different depending on their emotional nature, their lifestyle and their moral, religious and ethical beliefs. However, their suffering can run very deep and deserves attention, time and opportunities for free expression of feelings and concerns.

The medical counselling model developed by Colagiuri and Craig[14] (see FIG. 4.1 in CHAPTER 4) is very useful as it empowers patients to make their own decisions through facilitation as opposed to the directive and advisory medical model.

The couple are provided initially with accurate and appropriate information. Anxiety is alleviated by reassurance and by dispelling myths.

The facilitation process enables the couple to ventilate any feelings of guilt, anxiety, fear, anger and sexuality. The style of questioning should aim to explore the influence that the problem has had on the couple and then the influence they have over it. These processes then lead to decision making by the couple about further management strategies.

A graph of emotional responses to the infertility (see FIG. 107.3) can be used to help the couple explore their current and past emotional responses to their problem. Apart from helping them realise that their problem is not unique, it provides opportunities for ventilation of important feelings that can act as a basis for counselling.

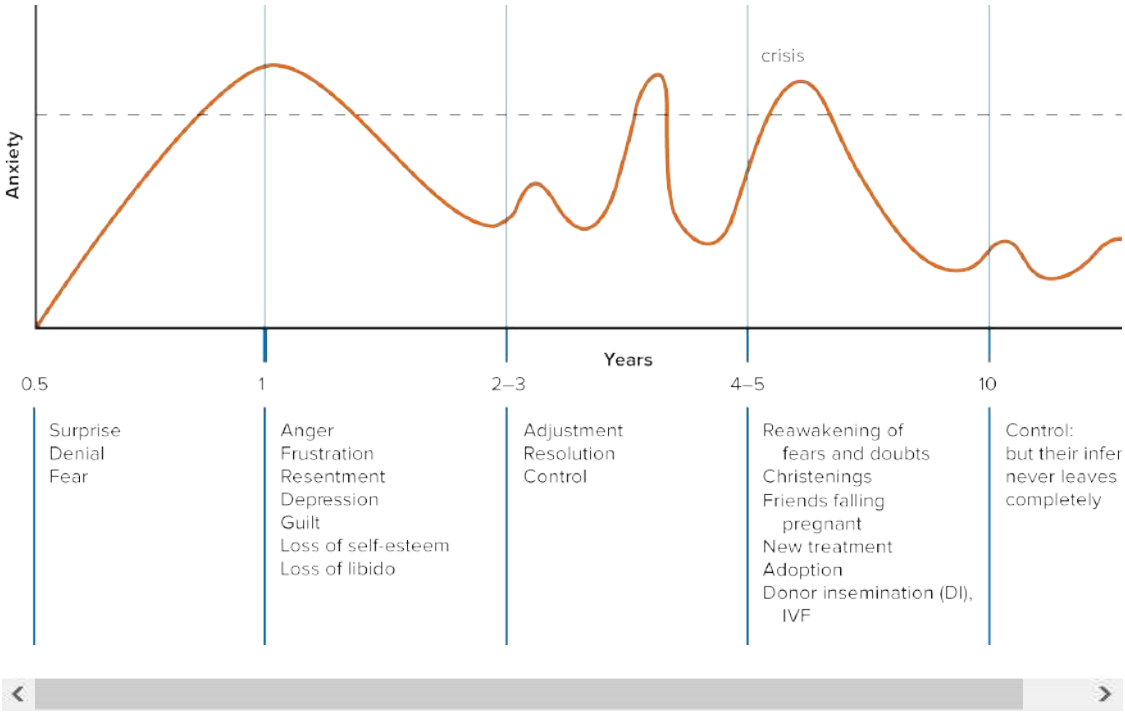

FIGURE 107.3 Emotional responses to infertility

Source: Reproduced with permission from Craig[14]

Treatment[2]

Treatment options for female subfertility:

- Ovulation induction

  metformin (for women with PCOS, use alone or with letrozole or clomiphene)

  letrozole

  clomiphene citrate

  gonadotrophins

    FSH

    LH

    human chorionic gonadotrophin (hCG)

  cabergoline or bromocriptine for hyperprolactinaemia

- Endometriosis

surgical ablation/excision of deposits and division of adhesions

- Surgical repair of tubal obstruction, removal of uterine septum, myomectomy and hysteroscopic resection of intra-uterine adhesions are other surgical treatment options.

Treatments for male subfertility:[7]

- Gonadotrophin therapy for men with hypogonadotrophic hypogonadism
- Microsurgical vasovasostomy (vasectomy reversal)
- Surgical correction of varicocele with oligozoospermia (role unclear)
- Sympathomimetic agents (e.g. pseudoephedrine) may correct retrograde ejaculation

Assisted reproductive technology (ART)

Assisted reproductive technologies (ARTs) such as in vitro fertilisation (IVF) have revolutionised the treatment of infertility. One in 25 Australian babies is now born via IVF and it is estimated that more than 6 million children worldwide have been conceived using these technologies over the last three decades.[15]

The most recent data from Australia and New Zealand estimated the cumulative live birth rate of ART for all ages was 32.7% for the first complete cycle, rising to 54.3–77.2% by the eighth cycle. Success rates were significantly lower in women aged over 40, with an estimated cumulative live birth rate of 10.7% for the first complete cycle, rising to 21.0–37.9% by the eighth cycle. IVF is rarely recommended to women aged over 44 years.[5]

Treatment options include:[12]

- IUI (intra-uterine insemination with the partner’s sperm)
- IVF (in vitro insemination)

  involves the retrieval of ova, which are mixed with sperm and incubated for 5 days—a resultant embryo is then transferred into the uterus via the cervix

  embryos can be frozen and stored cryogenically; frozen embryos can be thawed and transferred to the uterus

- ICSI (intra-cytoplasmic sperm injection)

  a single sperm is injected into an egg for fertilisation outside the body and then replaced into the uterus

- Pre-implantation genetic testing (PGT)

testing can be for aneuploidy or single-gene disorders

The role of the general practitioner

Since poor outcomes in subfertile couples are associated with delay in identification and treatment, early investigation instituted by the GP is important to expedite possible specialist involvement.

Referral is recommended after planned conception for:[3]

- 1 year if the woman is ≤35 years
- 6 months if the woman is >35 years

Consider early referral for women with amenorrhoea or oligomenorrhoea, or for couples with a known reason for infertility.

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108 Sexual health

[Drink,] sir, provokes the desire, but it takes away the performance.

WILLIAM SHAKESPEARE (1564–1616), *MACBETH*, ACT 2, SCENE 1

Sexual health is the ability to embrace and enjoy our sexuality throughout our lives. Our sexuality is a fundamental human experience that encompasses sex, gender identities and roles, sexual orientation, eroticism, pleasure, intimacy and reproduction.[1]

Family doctors are often asked to provide advice and help for sexual concerns and are continually challenged to detect such problems presenting in some other guise. Since we deal with so much illness and prescribe so many drugs, we must be aware of the possible implications of their various effects on sexual health.

Sexual dysfunction[2]

Sexual dysfunction occurs when an individual is unable to enjoy their sexuality as they would wish.

Several studies have demonstrated that sexual concerns and problems are common, with a prevalence ranging from 20–40%. Sexual difficulties are summarised in TABLE 108.1 . There is also evidence that health professionals do not discuss sexual concerns in consultations as often as patients would like.

Table 108.1 Sexual difficulties and dysfunction[2]

Female problems:

- lack of libido: 4 in 10 women
- orgasmic difficulties: 1 in 3 women
- painful intercourse (dyspareunia, vaginismus): 1 in 10 women
- concerns about labia

Male problems:

- lack of libido: 1 in 4 men
- erectile dysfunction: 1 in 5 men
- premature/delayed ejaculation: 1 in 4 men
- anorgasmia
- concerns about penis

Other:

- sexual desire discrepancy
- substance/medication-induced sexual dysfunction
- compulsive sexual behaviour

Sexual dysfunction can be the bodily expression of many sorts of distress and requires consideration of biological, psychological, sociocultural and relationship factors. The unique place of general practice and the family doctor provides ideal opportunities to address the sexual concerns of patients as the family doctor often has considerable insight into the family dynamics and first-hand perspective of the individuals involved.

Presentation of sexual concerns

Although some patients may present directly with a complaint of sexual dysfunction, many will be less direct and use some other pretext or complaint as a 'ticket of entry' for their sexual concerns (see TABLE 108.2). Despite a seemingly terse approach the issue must be recognised and treated with considerable importance. This may mean scheduling an appropriate time to discuss the concerns.

Table 108.2 How sexual issues may present in family practice[3]

Minor non-sexual complaint—'entry ticket'
Specific sexual concern
Marital or relationship problem
Non-sexual problem (as perceived by the patient)
Sexual enquiry as part of illness management
Sexual enquiry as part of total health check-up
Infertility
Menopausal symptoms

The effect of illness on sexual function

Doctors seldom enquire about the impact of an illness on the sexual function of patients and their partners (see TABLE 108.3). It is most appropriate to enquire about these issues with patients, e.g. the postmyocardial infarction patient, the postprostatectomy patient, the patient taking antihypertensives or other drugs (see TABLE 108.4), and the post-mastectomy or post-hysterectomy patient. Diabetes deserves special attention as more than 60% of diabetic men have reported erectile difficulties.[4]

Table 108.3 Medical conditions affecting sexual performance

Cardiovascular:

- previous myocardial infarction
- angina pectoris
- peripheral vascular disease
- hypertension and its treatment

Respiratory:

- asthma
- COPD

Endocrine:

- diabetes mellitus
- thyroid dysfunction
- Cushing syndrome
- hypogonadism

Neurological:

- multiple sclerosis
- neuropathy
- spinal cord lesions
- Parkinson disease

Musculoskeletal:

- arthritis

Psychogenic:

- depression
- anxiety
- PTSD

Kidney:

- kidney failure

Urological:

- prostatectomy
- prostatitis
- phimosis
- Peyronie disorder
- priapism
- Klinefelter syndrome

Hepatobiliary:

- cirrhosis

Gynaecological:

- endometriosis
- vaginal repair/birth trauma
- pelvic prolapse
- hysterectomy
- vestibulodynia

Chronic pain

Cancer

Colorectal

- anal pain
- colitis

Table 108.4 Drugs affecting sexual arousal and function

Alcohol

Anticholinergics

Anti-androgens

Antidepressants, e.g. SSRIs, SNRIs

Anti-epileptics

Antihypertensives

Antipsychotics

Aromatase inhibitors

Benzodiazepines

Cytotoxic drugs

Hormone contraception, e.g. combined oral contraceptive

Tamoxifen

Marijuana

Opioids

Taking a sexual history

It is important to create a safe environment for patients to talk about their sexual behaviour and sexuality. Consider explaining why you are asking for this information and request permission to proceed. Avoid being too formal or too familiar but aim to display a wise, matter-of-fact, empathic, commonsense rapport. CHAPTER 109 covers the sexual history required when assessing for sexually transmitted infections (STIs).

Be aware that many patients will have an undisclosed history of sexual abuse.

Probing questions for a suspected sexual problem

- How are things going in your sex life?
- Do you have any difficulties with your relationship?
- Do you have any pain or discomfort during intercourse?
- Have you had any difficulties with erections (men)?
- It's common for people in your situation to experience sexual difficulties. Is that happening to you?

Sexual history

When a patient wishes to discuss a sexual symptom, consider the following:

- phases of the sexual response (interest, arousal, erectile function, orgasm)
- range of sexual practices including masturbation
- situations in which the symptoms occur
- associated distress with symptoms

- pain during sex

Further questions that may be helpful:

- What sex education did you have as a child at home or at school?
- What were your parents' attitudes to sex?
- Have you experienced unwanted sexual experiences?
- Are you experiencing stress in any area of your life—relationship, work or home-life?
- Are you able to talk to your partner about sex?
- Do you have concerns about your sexual orientation or gender identity?

A general medical history should be taken including:

- menstrual history
- past STIs
- chronic health issues
- mental health issues
- medication use, including contraception
- surgical history
- recreational drug and alcohol use
- smoking

Examination

The routine medical examination should include the basics such as urinalysis, BP measurement, genital examination and neurological examination where indicated.

Consider a male genital exam to check for Peyronie disease, small testicle size (may indicate hypogonadism or Kleinfelter syndrome) or retractable foreskin.

Consider a female genital examination in women with dyspareunia or vaginismus.

Investigations

No particular routine tests are recommended. Tests for male erectile dysfunction (impotence) are outlined later in this chapter. Tests that may help exclude significant causes of low libido are

those for diabetes, liver dysfunction, thyroid dysfunction and endocrine dysfunction. Endocrine dysfunction tests include prolactin, free testosterone, FSH, LH and oestradiol estimations. Other investigations may include pelvic ultrasonography, colposcopy or laparoscopy.

Basic sexual counselling

The family doctor can learn to be an effective sex counsellor. Sex counselling can be emotionally demanding and, while good interviewing skills, interest, support and basic advice are important, additional skills are needed to be an effective counsellor.

Principles for basic sexual counselling:

- give the patient permission to talk openly about sexual matters
- allow for a relaxed exchange of information
- dispel sexual myths and correct misunderstandings
- explore the patient's anxiety and any impact on their relationship
- de-emphasise the modern-day obsession with performance and orgasm and emphasise the value of alternative forms of sexual expression (e.g. caressing, kissing, and manual and oral stimulation)
- reassure the patient that he or she is normal (where appropriate)

Inappropriate doctor behaviour is presented in TABLE 108.5 .

Table 108.5 Sexual counselling: inappropriate doctor behaviour

Overfamiliarity
Being too formal
Being too talkative
Blunt questioning
Being judgmental
Making assumptions about the other's sexuality
Imposing one's own beliefs and standards
Dogmatism
Tackling problems beyond one's experience

Sexual problems can be grossly underestimated. Human beings generally have a basic craving