

- mainly on trunk, buttocks and thighs, especially axillae and groin
- itchy
- small pustules surrounded by circular red–purple halo
- follows immersion in a hot spa bath or tub

Treatment is based on the sensitivity of the cultured organisms (e.g. ciprofloxacin). Many cases resolve spontaneously in 1–2 weeks.

## Folliculitis of trunk from spa baths

‘Hot tub folliculitis’ is caused by *P. aeruginosa* (usually) in poorly chlorinated water maintained at temperatures 37–40°C.

Treatment is with ciprofloxacin 500 mg (o) bd for 7 days.

## Pseudofolliculitis<sup>8</sup>

Sterile folliculitis is usually due to maceration, resulting from obesity, heavy sweating, contact with occlusive substances (e.g. oils), shaving and waxing.

Folliculitis barbae is a chronic inflammatory disorder caused by a foreign-body reaction to the hair shaft. It is not an infection but a complication of hair removal techniques. It is common in men with curly hair and in the groin area. It tends to recur.

## Management

- Prior to shaving, wash the skin with warm water and a soap substitute.
- Change shaving habits: avoid close shaving; shave less often; shave in direction of hair growth; shave on relaxed skin (i.e. do not pull the skin taut) and use good-quality blades.
- Consider laser hair reduction if appropriate.
- Permanently stopping hair removal techniques is the best treatment.
- If persistent, use benzoyl peroxide 5% gel or solution topically, twice daily.
- If no response after 6 weeks, add clindamycin–benzoyl peroxide + clindamycin 5% + 1% gel topically, once daily.

## ¶ Deep folliculitis

Deep forms are usually very tender and painful. Examples are styes, boils (furuncles) and

carbuncles.

## Boil (furuncle)

This is an *S. aureus* infection of a hair follicle and may occur in any hair-bearing site. The painful red nodule enlarges, becomes fluctuant and develops a necrotic centre, which discharges a core of thick yellow pus tinged with blood (with associated relief of pain).

### Treatment (adults)

- (According to swabs) di(flu)cloxacillin 500 mg or cephalixin 500 mg (orally, 6 hourly for 5–7 days) or clindamycin 450 mg (8 hourly for 5 days)

## Boils—recurrent

- Obtain swabs
- 3% hexachlorophene body wash daily
- Mupirocin to the lesions and nares
- Antibiotics (as above)—according to swabs

## Carbuncle

This is a cluster of small abscesses involving a group of adjoining hair follicles and discharging pus from several points. Common sites are the back of the neck, the shoulders, buttocks or over the hips. Treatment is as for a boil.

## Stye of eye

- Apply heat with direct steam from a thermos onto the closed eye or by a hot compress (helps spontaneous discharge).
- Perform lash epilation to allow drainage (incise with a size 11 blade if epilation doesn't work).
- Only use topical antibiotic ointment (e.g. chloramphenicol) if infection is spreading locally, and systemic antibiotics if distal spread noted by pre-auricular adenitis.

### Practice tips

- Be vigilant for the deadly meningococcal septicaemia, which may present as an erythematous rash initially prior to the development of purpura.
- Prescribed drugs are a common cause of rash, especially toxic erythema, e.g.

antibiotics (especially penicillins), thiazides, anti-epileptics, allopurinol and NSAIDs.

## Patient education resources

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

- Herpes simplex (cold sores)
- Herpes zoster (shingles)
- Kawasaki syndrome
- Measles
- Pityriasis rosea
- Rubella
- Varicella (chicken pox)

## References

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- 1 Hunter JAA, Savin JA, Dahl MV. *Clinical Dermatology* (3rd edn). Oxford: Blackwell Scientific Publications, 2002: 64.
- 2 Murtagh J. *Patient Education* (7th edn). Sydney: McGraw-Hill, 2016.
- 3 Thomas RM. Drug eruptions. Med Int, 1988; 49: 2038–42.
- 4 Cutaneous drug reactions [published 2015]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2015. [www.tg.org.au](http://www.tg.org.au), accessed April 2021.
- 5 Shingles [published 2019]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2019. [www.tg.org.au](http://www.tg.org.au), accessed April 2021.
- 6 Pain associated with shingles (herpes zoster) [published 2020]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2020. [www.tg.org.au](http://www.tg.org.au), accessed April 2021.
- 7 Dwyer DE, Cunningham AL. Herpes simplex and varicella zoster virus infection. Med J Aust, 2002; 177: 267–72.
- 8 Infected skin [published 2015]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2015. [www.tg.org.au](http://www.tg.org.au), accessed April 2021.

## 115 Skin ulcers

*An ulcer, which occurring at any of the vital parts of the body secretes a copious quantity of pus and blood, and refuses to be healed, even after a course of proper and persistent medical treatment, is sure to have a fatal determination.*

SUSHRUTA-SAMHITA (5TH CENTURY BCE)

An ulcer is a localised break in the epidermal tissue of the surface of the skin or mucous membrane as a result of trauma, pressure or infection; chronic wounds are associated with an underlying pathology. This applies particularly to leg ulcers. Ulcers are commonly found on the legs and feet, on areas exposed to the sun and over bony prominences on the sacrum and heels. They may be clean, sloughy or necrotic.

The national morbidity survey (UK) showed that 2–3 per 1000 patients per annum consulted their GP with ‘chronic ulcers of the skin’.

### Key facts and checkpoints<sup>1,2</sup>

- The great majority of leg ulcers (approximately 80%) are vascular in origin due to arterial insufficiency or venous hypertension, or to a combination of the two, i.e. mixed ulcers.
- Approximately 20% of leg ulcers are atypical and their cause will vary from autoimmune disease, e.g. systemic lupus erythematosus (SLE), to inflammatory disease, e.g. Pyoderma gangrenosum.
- If clinical findings don't provide the diagnosis, ordering the ankle-brachial index (ABI) is essential if pulses are not palpable to exclude arterial disease. Duplex Doppler ultrasound is the key investigation for both venous and arterial disease.
- Most ulcers are multifactorial:
  1. venous + obesity + immobility (resulting in venous stasis) + poor compliance or
  2. venous + arterial + trauma + infection

- Identify and treat any intrinsic or extrinsic factors impairing wound healing.
- High-stretch compression bandages are better than short-stretch compression bandages, which are mainly used in lymphoedema, and multilayer is best.
- Elastic bandages are better than non-elastic bandages.
- Venous surgery can improve outcomes—newer methods use non-surgical foam sclerosant injections and lasers.
- A moist environment delivered by modern dressings provides a physiological environment for wound healing.
- Adequate wound debridement is essential to remove necrotic material and slough, and enable healing to commence and progress.
- It is important to consider biopsy as hypergranulation that does not respond quickly to topical treatment with hypertonic saline, silver nitrate and compression bandaging may be a squamous cell carcinoma (SCC).
- Diabetic ulcers may be either neuropathic, which are mostly caused by pressure damage due to sensory neuropathy, or ischaemic due to loss of peripheral arterial circulation. They may also be a combination, i.e. neuroischaemic.
- Autoimmune diseases are the underlying cause of vasculitic wounds. They result from damage caused by circulating antibodies often seen in RA, lupus and scleroderma. They are very painful and difficult to heal without the use of immunosuppressants.
- Accurate diagnosis of the ulcer is vitally important for management decisions.
- Bacterial swabs are unhelpful because all chronic ulcers will become colonised with both Gram-positive and Gram-negative bacteria.
- If infection is suspected, clean the wound surface with a non-bacterial cleanser and then perform a biopsy for microscopy.

## The clinical approach

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It is useful to keep in mind the various causes of ulcers (see TABLE 115.1). The commonest causes or types are venous and ischaemic ulcers of the leg, pressure ulcers (decubitus) and trauma. It is important not to misdiagnose malignant ulcers, including ‘Marjolin ulcer’, which is a squamous cell carcinoma (SCC) developing in unstable chronic scars or ulcers (e.g. burns, venous ulcers, tropical ulcers) of long-standing duration. Amelanotic melanoma is a specific trap.

**Table 115.1** Types and causes of skin ulcers

Traumatic

Decubitus (related to trauma—pressure injury)

Vascular:

- Venous
  - varicose veins
  - post thrombophlebitis
- Arterial insufficiency
- Skin infarction (thrombolytic ulcer)
- Vasculitis
  - rheumatoid arthritis, SLE, scleroderma

Infective:

- Tropical ulcer
- Tuberculosis
- *Mycobacterium ulcerans* (Buruli ulcer)
- Post cellulitis
- Chronic infected sinus

Malignant:

- Squamous cell carcinoma
- Marjolin ulcer (SCC) in long-standing ulcer
- Basal cell carcinoma (rodent ulcer)
- Malignant melanoma
- Ulcerating metastases

Neurotrophic:

- Peripheral neuropathy (e.g. diabetes)
- Peripheral nerve injuries (e.g. leprosy)

Haematological:

- Polycythaemia
- Spherocytosis
- Sickle-cell anaemia

Miscellaneous:

- Artefactual
- Medications, e.g. nicorandil, nicotine

- Pyoderma gangrenosum inflammatory ulcer
  - Insect and spider bites
  - Martorell ulcer caused by hypertension
- 

## History

A careful history helps determine the cause of the ulceration. Relevant history includes previous deep venous thrombosis or pulmonary embolism, diabetes, rheumatoid arthritis, inflammatory bowel disease, chronic skin ulcers and arterial insufficiency, including a history of intermittent claudication and ischaemic rest pain.

A drug history is important, considering especially beta blockers and ergotamine, which can compromise the arterial circulation, corticosteroids and NSAIDs, which affect healing, nifedipine, which tends to aggravate ankle oedema, and hydroxyurea, which can cause ulcers.

## Examination<sup>3</sup>

Any ulcer should be assessed for the following characteristics:

- site
- shape
- size
- depth
- edge—consider consistency
- floor
- base
- discharge
- surrounding skin:
  - colour (?signs of inflammation)
  - sensitivity
- mobility in relation to underlying tissue
- regional lymph nodes

## Site of ulcer

Venous ulcers typically occur on the medial side of the leg in relation to incompetent perforating veins in the traditional gaiter area (see FIG. 115.1 ).



**FIGURE 115.1** Area typically affected by varicose eczema and ulceration (the 'gaiter' area)

Ischaemic ulcers tend to occur on the lateral side and anterior part of the leg and the foot.

Trophic ulcers, which are associated with neuropathy, occur on parts subject to repeated pressure and trauma, such as the ball of the foot or the pulps of the fingers.

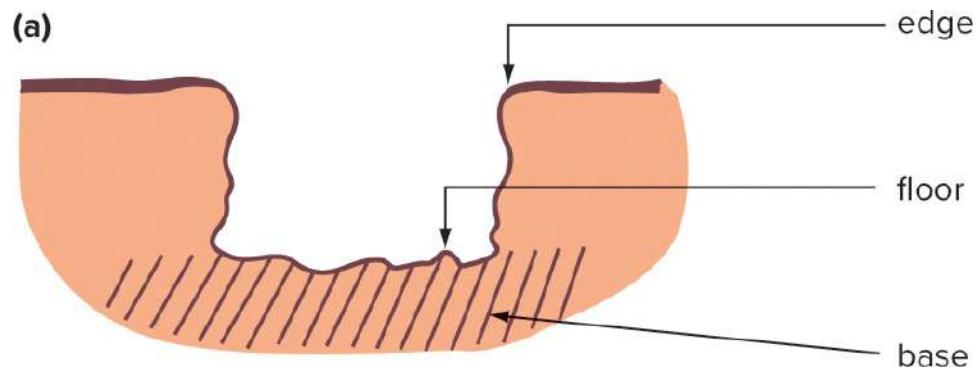
## Postsolar keratoses

Solar-induced ulcers, such as SCCs and BCCs, occur on such parts exposed to the sun. It should be noted if the ulcer is related to old scars, including burns and chronic ulcers.

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## Size, shape and edge

The classic appearances of various ulcers are presented in FIGURE 115.2 . These are general guidelines only. Infective ulcers due to *Mycobacterium* species and pressure injury tend to have an undermined edge while a trophic ulcer is punched out and typically round in surface shape. A raised firm ulcer edge may indicate malignancy.



(b)	Edge	Example
	punched out	trophic ulcer arterial ulcer
	undermined	pressure injury, e.g. bed sore
	everted	squamous cell carcinoma
	rolled	basal cell carcinoma

**FIGURE 115.2 (a) Parts of an ulcer, (b) types of ulcer**

Source: Davis et al. *Symptom Analysis and Physical Diagnosis* (2nd edn), page 309. Reproduced with permission from Pergamon Press.

## Floor of the ulcer

The floor or base of the ulcer provides useful clinical information. A dry or extended base or necrotic eschar in the floor implies ischaemia. Venous ulcers, on the other hand, are often superficial and tend to have fibrinous exudate and ooze, sometimes purulent fluid.

Colour guide:

- black—necrosis, ischaemia
- yellow—slough
- red—granulation
- pink—epithelium
- green—infection

## Investigations

The following should be considered, according to the clinical findings:<sup>4</sup>

- full blood count
- ESR/CRP
- random blood sugar/HbA1c (known diabetes)
- kidney function tests
- rheumatoid factor tests
- duplex Doppler ultrasound
- swab for specific organisms
- biopsy, especially if SCC suspected (be careful of biopsy if melanoma: amelanotic melanomas are a trap); also accurately identify an infective organism

## Lower limb ulceration

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The most common causes of lower limb ulceration are venous disease, arterial disease, mixed venous and arterial, and diabetes.

Differentiating between leg ulcers (85%) and foot ulcers (15%) is very important since they present two very different problems.<sup>3</sup> According to the survey by Stacey, venous disease is present in two-thirds of leg ulcers, while arterial disease occurs in 28% (see TABLE 115.2). Ulceration on the foot frequently has an arterial aetiology (72%), with many of these patients also having diabetes, whereas venous disease is present in only 6%.<sup>5</sup>

**Table 115.2** Causes of chronic ulceration of the leg and foot<sup>5</sup>

	%
<b>The leg</b>	
Venous disease	52
Mixed venous and arterial disease	15
Arterial disease	13
Others	20
<b>The foot</b>	
Arterial disease	72
Mixed venous and arterial disease	2
Venous disease	4
Others (includes pressure)	22

The differential characteristics are presented in [TABLE 115.3](#).

**Table 115.3** Comparison of typical features of venous and arterial ulceration of the leg

	Venous	Arterial
<b>Site</b>	Around ankle and lower third of leg (gaiter area)  Just above medial and lateral malleoli	Distal to ankle  Over pressure points on toes, side of foot, metatarsal heads
<b>Pain</b>	Nil to mild	Usually moderate to severe
<b>Oedema</b>	Pitting oedema usually present	Usually absent
<b>Ulcer features</b>	'Ragged' edge  Often superficial  Ooze + + +	'Punched out'  Often deep, involving deep fascia  Dry
<b>Associated limb features</b>	Varicosities  Leg warm, red, oedematous  Varicose dermatitis  Haemosiderin deposits  Atrophie blanche	'Cold' extremities  Ischaemic changes  Diminished or absent peripheral pulses  Thin, shiny, dry skin  Poor perfusion
<b>History</b>	Limb oedema	Peripheral vascular disease—

Past DVT	claudication, rest pain
Failed graft	Diabetes
	Smoker

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<b>ABI</b>	>0.9	<0.5–0.7
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Source: Based on a table prepared by Dr Denise Findlay; reproduced with permission

A general examination, including the leg, is very important. This includes examining the venous drainage ([CHAPTER 55](#) ), the arterial pulses and the sensation of the leg, and checking for the presence of diabetes.

Appropriate investigations (if required) include:

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- full blood count
- blood sugar
- duplex Doppler ultrasound for arterial circulation

## To swab or not to swab

A routine ulcer swab is not considered to be of significant value. If specific organisms such as *Mycobacterium ulcerans* are suspected, then PCR testing or cultures are necessary. Biopsy is considered to be accurate.

## Measurement of ankle/brachial pressure index

To plan management of a leg ulcer, it is ideal to determine the blood flow with hand-held Doppler ultrasound. Measure ankle and brachial systolic pressures and then determine the ABI, which is the ankle pressure divided by the brachial pressure. Typical levels are:<sup>4</sup>

Normal range	0.91–1.3
Borderline/acceptable	0.9–1.0
Moderate PAD	0.5–0.8
Severe PAD	<0.5
Normal	>0.9 (venous ulcer)
Ischaemic	<0.5 (arterial ulcer)
Claudicant	0.5–0.9 (mixed arterial–venous ulcer, significant ischaemia if <0.8)

The ability to determine the cause of the ulceration and thus treat accordingly, especially with pressure dressings, has been a major advance in management. An ABI <0.8 warrants caution in applying any compression; <0.4 demands urgent referral.

*General rule:* Low compression 0.7–0.8; no compression <0.7.

## Arterial (ischaemic) ulcers

Ischaemic ulcers are generally localised to the most peripheral areas below the ankle joint (see FIG. 115.3 ), such as the tips of the toes and the point of the heel, or to pressure points such as the heels, malleoli or head of the first metatarsal.



**FIGURE 115.3** Ischaemic 'arterial' ulcers in an elderly woman with a long history of intermittent claudication and a recent history of nocturnal ischaemic rest pain in the feet. The ulcers healed after reconstruction for arterial obstruction.

*Photo courtesy Terry Devine*

### Clinical features

- Painful
- Punched out
- Minimal granulation tissue

Management is directed towards reperfusion.

## Venous ulceration

Venous ulceration (synonyms: ‘varicose’, ‘stasis’ and ‘gravitational’ ulcers) accounts for the majority of leg ulcers. Chronic venous insufficiency is one of the most common medical problems in the elderly, with an estimated incidence of 5.9%.<sup>6</sup>

The problem is invariably secondary to lack of movement leading to venous stasis or deep venous thrombophlebitis. The subsequent chronic venous hypertension produces trophic changes such as hyperpigmentation (see FIG. 115.4 ), fibrotic thickening, induration and oedema. The end point of this process is ulceration, which affects 3% of those with varicose veins and 30% of those with trophic changes.<sup>7</sup>



**FIGURE 115.4** Venous ulceration in an elderly patient with postphlebitic varicose ‘eczema’. Venous pigmentation, dermatitis, atrophy and calcification of the subcutaneous tissues are present.

*Photo courtesy Terry Devine*

### Clinical features<sup>8</sup>

- Occur in same area as venous eczema
- Shallow (but can reach periosteum)
- More common medial than lateral
- Sometimes circumferential
- Granulating floor sometimes with surrounding inflammation—may be cellulitis
- Notoriously slow in healing without adequate compression

- Generally not tender but can be painful
- Associated pain is usually relieved by raising the leg

On examination, superficial varicosities are usually but not invariably present. Pitting oedema may be present early but with time fibrosis and firm induration develop. Other clinical features include dermatitis (eczema), punctate capillary proliferation, haemosiderin, hyperpigmentation and ‘atrophie blanche’ (porcelain white scar with rim of telangiectasia).<sup>8</sup>

## Management (venous leg ulcers)

A major advance in the management of venous ulcers has been the finding that wounds heal better in an occluded or semi-occluded state.<sup>9</sup> A moist environment also aids healing. The dressing will control the wound environment. The main treatment is graduated compression at 20–40 mmHg. See [TABLE 115.4](#).

**Table 115.4** Wound management principles

The good	The bad
Hydration	Dryness
Washing with water or saline	Excess antiseptics
Insulation protection	Exposure to air
	Scabs, crust, slough
<b>Dressings</b>	
Compression (venous)	Dry dressings
Hydrogel	Gauze packing
Foams	Tulles
Minimal changes	Oedema/lymphoedema

## Principles of optimal management

- Explanation about the cause, and promotion of patient compliance
- Promoting clean granulation tissue to permit healing (see [TABLE 115.4](#))
- Meticulous cleansing and dressing (avoid soaps and sensitising preparations)
- Prevention and control of infection—antibiotics indicated only if cellulitis (cephalexin or erythromycin): if infection is confirmed, NOT where inflammation is present (as in most venous ulcers)

- Firm elastic compression bandage—an elastic compression bandage applied with 30% stretch from base of toes to just below the knee; the degree of compression depends on the blood flow and is proportional to it
- Bed rest with elevation (if severe, 45–60 minutes twice a day and at night): ensure legs are elevated higher than the heart
- Encourage early ambulation and exercise
- Appropriate modification of lifestyle including weight reduction, smoking cessation (NB)
- Good nutrition includes a healthy balanced diet with ample protein and complex carbohydrates
- Be aware of drugs that can adversely affect healing (see TABLE 115.5 )

**Table 115.5** Drugs that can hamper healing<sup>10</sup>

Nicotine/smoking

Corticosteroids

Cytotoxic agents

Aspirin/NSAIDs

Antibiotics

Beta blockers

Hydrourea

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*Note:* Firm compression is the single most important factor in the healing of venous ulcers.<sup>2,7</sup> Options include elastic stockings, elastic bandages, zinc paste bandages (Unna's boots) and legging orthoses.<sup>11</sup>

### Cleansing/debridement agents

There are many cleansing agents, including N saline, surfactants, e.g. QV wash and warm tap water.

As a rule, avoid antiseptics, which destroy cells, although cadexomer iodine, which is a slow-release form of iodine, is non-toxic to tissues, reduces bacterial load and clears odorous slough. However, if using Povidone-iodine solution, wash off after 5 minutes. Iodosorb is a low-dose iodine dressing appropriate for infected, contaminated wounds, particularly a diabetic wound. A good combination is normal saline cleansing followed by IntraSite Gel for debridement, covered with a foam dressing. Strong salt dressings (e.g. Mesalt or Curasalt) are very good for cleaning contaminated, infected wounds and hypergranulation, but need changing daily and to be covered

by a very absorbent overlying dressing.

Hydrogels such as IntraSite Gel, which have been found to be effective at debridement (including black necrotic areas), have generally replaced enzyme dressings.

### Lymphoedema<sup>12</sup>

This is not pitting oedema but a reduction in the function of the lymph vessels to drain extracellular fluid.

Primary lymphoedema is caused either by malformation of the lymph vessels or by damage from bacteria, fungi, parasites, insects, chemicals, radiotherapy or surgery. Secondary lymphoedema is caused by primary malignancy or intralymphatic propagation of a tumour. Lymphoedema results in major changes to the skin, such as thickening of the skin, build-up of scale and keratin and worsening hyperkeratosis, producing a warty appearance.

Wounds are common in lymphoedema. They are managed by treating the wound environment with appropriate dressing, but the main treatment is significant compression with either specially developed garments or inelastic bandages. Difficult cases should be referred to a specialist lymphoedema clinic.

A simple clinical test for determining a lymphoedema component in a swelling of the lower leg is the Stemmer sign test. This test is performed by lifting and pinching the skin at the dorsum of the second toe on each foot. A positive Stemmer sign results when the skin cannot be lifted and pinched together. The test is negative for lymphoedema when the skin can be pinched and lifted. A positive Stemmer sign *always indicates lymphoedema, but a negative sign does not always rule out lymphoedema.*

### Wound dressing<sup>9,13</sup>

There are six main types of modern wound dressings: films, hydrogels, hydrocolloids, alginates, foams and hydroactives (foam-like)—all expensive. Films, hydrogels and hydrocolloids increase the wound moisture, whereas alginates and foams absorb exudate. The more traditional dressings such as tulle gras, non-adherent pad dressings and saline soaks have little use. A lightweight cohesive bandage or a lightweight tubular bandage can be used to hold non-adherent dressings in place.

General rules:<sup>10</sup>

- allow 2–3 cm of dressing greater than the wound
- place one-third above and two-thirds below the wound
- remove when ‘strike-through’ occurs
- remove with care in older patients

- remove under the shower if necessary
- when in doubt, DO NOT HARM: use foam and gel combinations

## Medicated occlusive bandages

There are several suitable occlusive paste bandages for ambulant patients, which ideally should be left on for 7–14 days. These contain zinc oxide. Examples include Gelocast and Zipzoc (which are zinc paste bandages with no preservatives). A new dressing containing sucrose octasulfate (e.g. Urgostart) has been shown to be superior in the management of both diabetic and venous ulcers.<sup>14</sup>

Patch testing for an allergic response should be performed for a few days beforehand.

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## Bandages

Bandages have two main uses:

- retention: to keep a dressing in place
- compression: to assist venous return

High-stretch compression bandages are best.

Method of application:

- spiral application from toes with figure 8 around ankles, then spiral to knee
- 50% overlap
- constant tension provides graduated compression (Laplace's Law)

## Pitfalls and other factors to be considered

- Treat the primary cause by surgery or other means (e.g. varicose veins, vascular insufficiency).
- If oedema, elevate legs and prescribe diuretics. An ulcer will not heal in the presence of significant ankle oedema.
- Clarify the cause of oedema, which may be due to medication, e.g. calcium-channel blockers.
- Do not use crepe bandages or anti-embolic stockings as they do not provide appropriate compression
- Be careful of allergy to local applications (e.g. zinc).

- Be careful of irritation from local applications (e.g. antibiotics). Antibiotic-impregnated dressings are not generally recommended.
- Avoid heavy packing of the wound.
- Consider grafting (pinch skin or split thickness).
- Consider oxpentifylline (Trental 400) for chronic occlusive arterial disease and venous disease.<sup>8</sup>

## Post-healing and prevention of ulcers

- Encourage preventive measures, such as regular walking, good nutrition, no smoking, elevation of leg when resting, great care to avoid trauma.
- Apply emollients for varicose eczema.
- Wear a compression-grade elastic stocking (e.g. Jobst, Venosan or Sigvaris) or velcro wrap for varicose ulcers.

A recommended treatment routine for a leg ulcer is presented in TABLE 115.6. It is desirable (for the outpatient) to leave the dressings and bandages in place for 1 week, perhaps 2 weeks, depending on the state of the dressing.

**Table 115.6** A recommended leg ulcer treatment method

Clean with normal saline

- If slough, apply IntraSite Gel and cover with a foam dressing
- Dressing: non-adherent, e.g. Melolin
- Occlusive paste bandage—e.g. zinc oxide (7–14 days), plus compression bandage  
or  
Compression bandage (e.g. Tensopress) to just below knee
- Consider tubular elastic stocking cover (3 layers is an alternate method of compression)

Principles of management for chronic ulcers are summarised in TABLE 115.7.

**Table 115.7** Principles of management of chronic ulcers<sup>15</sup>

Ulcer type	Major management principles
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Venous	Control venous insufficiency: <ul style="list-style-type: none"> <li>• compression bandage</li> <li>• improve calf muscle pump action (ambulation, exercises)</li> <li>• vertical leg drainage</li> </ul>
Arterial	Vascular assessment for surgical intervention
Mixed venous/arterial	Vascular assessment for surgical intervention
Pressure	Eliminate or reduce pressure

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## Decubitus ulcer (pressure injury)

Pressure injuries tend to occur in immobile patients, especially those who are unconscious, paralysed or debilitated. The cause is skin ischaemia from sustained pressure over a bony area, particularly the heels, sacrum, hips and buttocks. Poor general health, including anaemia, is a predisposing factor.

The classification of pressure injuries is based on the level of tissue damage and depths of the wound:

- Stage 1: non-blanching erythema
- Stage 2: partial thickness (superficial) ulceration
- Stage 3: full thickness ulceration
- Stage 4: deep full thickness—skin loss with extensive tissue loss

Unstageable: deep tissue injury.

### Clinical features

- Preliminary area of fixed erythema at pressure site
- Relatively sudden onset of necrosis and ulceration
- Ulcer undermined at edges
- Possible rapid extension of ulcers
- Necrotic slough in base

### Prevention

- Good nursing care including turning patient every 2 hours
- Regular skin examinations by the nursing and medical staff
- Special care of pressure areas, including gentle handling
- Special beds, mattresses (e.g. air-filled ripple) and sheepskin to relieve pressure areas
- Good nutrition and hygiene
- Control of urinary and faecal incontinence
- Avoid the donut cushion
- Avoid soaps

## **Treatment<sup>13</sup>**

The most important principle is early intervention, including relief of pressure, friction and shear. Use above prevention measures, plus:

- clean base with warm water or saline solution and a surfactant (applied gently via a syringe)
- general guidelines for dressings:
  - deep ulcers—alginates (e.g. Algisite M, Kaltostat)
  - shallow ulcers—hydrocolloids (e.g. DuoDERM, CGF)
  - dry or necrotic ulcers—hydrogels (e.g. IntraSite, Purilon gel)
  - heavy exudative ulcers—foams (e.g. Lyofoam Max, Allevyn, Cutinova Hydro)
- give vitamin C, 500 mg bd
- give antibiotics for spreading cellulitis (otherwise of little use)
- for non-healing large pressure wounds, apply negative pressure therapy
- review patient's nutritional status as it impacts on both formation of pressure injury and slow healing
- healing is usually satisfactory but, if not, surgical intervention with debridement of necrotic tissue and skin grafting may be necessary; this is very effective if the patient can cope

## **Undressing wounds**

Removal of dressings from ulcerated wounds is very important. The contact layer should be removed slowly to prevent detachment of fragile epithelial surface cells and trauma to healthy

granulation tissue.<sup>16</sup>

### Role of honey

Honey has been advocated for centuries for healing ulcers. A particular type, Medihoney, is marketed. It provides moist healing and has antibacterial properties. Care has to be taken with over-moisturisation and maceration. Its role is still somewhat controversial, as is the application of sugar, cromoglycate powder, maggots and hyperbaric oxygen.

## ⌚ Trophic ulcers

Trophic ulcers are due to neuropathy causing loss of sensation (invariably diabetic) and usually follow an injury of which the patient was unaware (see FIG. 115.5).



**FIGURE 115.5** Neuropathic ulcer under the third metatarsal of the right foot in a patient with diabetes

A feature is a deep, punched-out lesion (similar to ischaemic ulcers) over pressure points. A common site is the ball of the foot under the first metatarsal head, but the heel or a bunion may also be affected.

The ulcers may extend to the bone and into joints. They are prone to secondary infection.

Treatment is based on controlling the diabetes and clearing infection with appropriate antibiotics, but referral for surgical management is usually essential.

## ***Mycobacterium ulcerans* (Buruli ulcer)**

Buruli ulcer (also known as a Bairnsdale or Daintree ulcer) is caused by *Mycobacterium ulcerans* and usually begins as a painless papule or nodule that forms a necrotic ulcer over weeks to months. An undermined edge is a classic feature. Diagnosis is by PCR (may need to be repeated) and biopsy.

It occurs in specific geographic locations, namely coastal Victoria, Far North Queensland and the tropical regions of Central and West Africa.

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In 2021, there was an epidemic in coastal Victoria, where possums were believed to be a reservoir. All age groups may be affected, including children.

Early referral to an infectious diseases unit is recommended, with 8 to 12 weeks of oral antibiotics (usually rifampicin and clarithromycin) required, along with specialist wound care. Surgical debridement of the ulcer may also be necessary.

## **Dermatitis artefacta and neurotic excoriations**

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These self-inflicted ulcerated or erosive skin lesions have a psychological basis.

### **Dermatitis artefacta**

In this condition, patients deny self-trauma and may have deep-seated psychological problems, or they may be malingering or manipulative for secondary gain.

### **Neurotic excoriations**

These lesions, which are usually identical to the artefactual lesions, are caused by patients who admit to scratching, picking or digging at their skin (FIG. 115.6). It occurs at times of stress and treatment is seldom successful. Treatment consists of counselling with CBT, a trial of antidepressants and topical antipruritics such as:

coal tar solution (liquor picis carbonis) and menthol in sorbolene cream

or

menthol (0.5%) or phenol (1.0%) in aqueous cream



**FIGURE 115.6** Neurotic excoriations (neurodermatitis) seen on three of four extremities. The fourth extremity is a prosthetic leg.<sup>17</sup>

### Practice tips

#### Principles of treatment:<sup>10</sup>

- Occluded and moist wounds heal faster.
- Maintain moist wound environment.
- Control exudate and debris (remove excess—leave enough to allow cellular regeneration).

- Maintain/improve circulation.
- Insulate and protect.

## Patient education resources

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

- Pressure sores (bed sores)
- Leg ulcers

## Resources

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Grey JE, Enoch S, Harding KG. ABC of wound healing: wound assessment. BMJ, 4 February 2006; 332(7536): 285–8.

Australian Wound Management Association. Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury. Osborne Park, WA: Cambridge Publishing, 2012.

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## References

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- 1 Kelly R. Leg ulcers and wound healing. In: *Dermatology Conference Notes*. Melbourne: Combined Alfred Hospital/Skin and Cancer Foundation, 2002: 29.
- 2 Sussman G. Ulcer dressings and management. Aust Fam Physician, 2014; 43(9): 588–92.
- 3 Davis A, Bolin T, Ham J. *Symptom Analysis and Physical Diagnosis* (2nd edn). Sydney: Pergamon Press, 1990: 380–9.
- 4 Ulcer and Wound Management [updated 2019]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2019. [www.tg.org.au](http://www.tg.org.au)
- 5 Australian Wound Management Association and the New Zealand Wound Care Society. *Australian and New Zealand Clinical Practice Guideline for Prevention and Management of Venous Leg Ulcers*. Osborne Park, WA: Cambridge Publishing, 2011.
- 6 Beauregard S, Gilchrest BA. A survey of skin problems and skin care regimens in the elderly. Arch Dermatol, 1987; 123: 1638–43.
- 7 Fry J, Berry HE. *Surgical Problems in Clinical Practice*. London: Edward Arnold, 1987: 115–17.

- 8** Weller C, Evans S. Venous leg ulcer management in general practice—practice nurses and evidence based guidelines. *Aust Fam Physician*, 2012; 41(5): 331–7.
- 9** Fitzpatrick JE. Stasis ulcers: update on a common geriatric problem. *Modern Medicine Australia*, 1990; June: 81–8.
- 10** Sussman G. An introduction to chronic wounds and their management. *Proceedings Monash University Update Course*. Melbourne: Monash University, 2009: 1–28.
- 11** Vernick SH, Shapiro D, Shaw FD. Legging orthosis for venous and lymphatic insufficiency. *Arch Phys Med Rehabil*, 1987; 68: 459–61.
- 12** Rockson SG. Update on the biology and treatment of lymphedema. *Curr Treat Options Cardiovasc Med*, April 2012; 14(2): 184–92.
- 13** Sussman G, Weller C. Wound dressing products update. *J Pharm Prac Res*, 2006; 36(4) 318–24.
- 14** Edmonds M et al. Sucrose octasulfate dressing versus control dressing in patients with neuroischaemic diabetic foot ulcers (Explorer): an international, multicentre, double-blind, randomised controlled trial. *The Lancet Diabetes & Endocrinology*, March 2018; 6(3): 186–96.
- 15** Findlay D. Wound management and healing in general practice. *Annual Update Course notes*. Melbourne: Monash University, 1996; 13–16.
- 16** Rowland J. Pressure ulcers: a literature review and a treatment scheme. *Aust Fam Physician*, 1993; 22: 1819–27.
- 17** Usatine RP, Saldan-Arragui MA. Excoriations and ulcers on the arms and legs. *J Fam Pract*, 2004; 53(9): 713–16.

# 116 Common lumps and bumps

*It will never get well if you pick it.*

AMERICAN PROVERB

Lumps and bumps are very common presentations and the skin a very common site for neoplastic lesions. Most of these lesions only invade locally, with some important exceptions, notably malignant melanoma.

Pigmented skin tumours thus demand very careful consideration, although only a very few are neoplastic. The optimum time to deal with the problem and cure any skin cancer is at its first presentation. The family doctor thus has an important responsibility to screen these tumours and is faced with two basic decisions: the diagnosis and whether to treat or refer.

Most skin lumps are benign and can be left in situ, but the family doctor should be able to remove most of these lumps if appropriate and submit them for histological verification. The main treatment options available in family practice are: biopsy, cryotherapy, curette and cauterity, excision or intralesional injections of corticosteroid.<sup>1</sup> A list of common and important lumps is presented in TABLE 116.1 .

**Table 116.1** Important lumps and their tissue of origin<sup>2</sup>

## Skin and mucous membranes

Fibroepithelial polyp (skin tag)

Epidermoid (sebaceous) cyst

Implantation cyst

Sebaceous hyperplasia

Mucocele

Hypertrophic scar and keloid

Warts and papillomas

Pox virus lumps:

- molluscum contagiosum
- orf
- milker's nodules

Seborrhoeic keratoses

Granuloma annulare

Dermatofibroma

Solar keratosis/actinic keratosis

Keratoacanthoma

Malignant tumours:

- basal cell carcinoma
- squamous cell carcinoma
- Bowen disease
- malignant melanoma
- Kaposi sarcoma
- secondary tumour

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### **Subcutaneous and deeper structures**

Lipoma

Neurofibroma

Lymph node (see [CHAPTER 50](#) )

Pseudoaneurysm

Musculoskeletal:

- ganglion
  - bursae
- 

## **A diagnostic approach to the lump**

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As with any examination, the routine of look, feel, move, measure, auscultate and transilluminate should be followed.

The lump or lumps can be described thus:

- number
- site

- shape—regular or irregular
- size (in metric units)
- position
- consistency (very soft, soft, firm, rubbery or hard)
- solid or cystic
- mobility
- surface or contour
- special features:

attachments (superficial/deep)

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exact anatomical site

relation to anatomical structures

relation to overlying skin

colour

temperature (of skin over lump)

tenderness

pulsation (transmitted or direct)

impulse

reducibility

percussion

fluctuation (?contains fluid)

bruit

transilluminability

special signs: slipping sign, emptying sign of cavernous haemangioma

spread: local, lymphatic, haematogenous

regional lymph nodes

?malignancy (is it primary or secondary?)

## Relation of the lump to anatomical structures<sup>2</sup>

The question ‘In what tissue layer is the lump situated?’ needs to be addressed.

- Is it in the skin? The lump moves when the skin is moved (e.g. epidermoid cyst).
- Is it in subcutaneous tissue? The skin can be moved over the lump. The slipping sign: if the edge of the lump is pushed, the swelling slips from beneath the finger (e.g. lipoma).
- Is it in muscle? The lump is movable when the muscle is relaxed but on contraction of the muscle this movement becomes limited.
- Is it arising from a tendon or joint? Movement of these structures may cause a change in the mobility or shape of the tumour.
- Is it in bone? The lump is immobile and best outlined with the muscles relaxed.

## Lumps of the skin and mucous membranes

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### Fibroepithelial polyps

Synonyms: skin tags, acrochordon, benign squamous papilloma, soft fibroma.

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#### Clinical features

- Benign skin overgrowth
- Pedunculated soft fibroma
- Increased incidence with age, obesity, diabetes
- Commonest on neck, axillae, trunk, groins
- No malignant potential
- Can be irritating or unsightly to patient

#### Management

- Can leave or remove
- To remove:

snip off with scissors or bone forceps (see FIG. 116.1 )

or

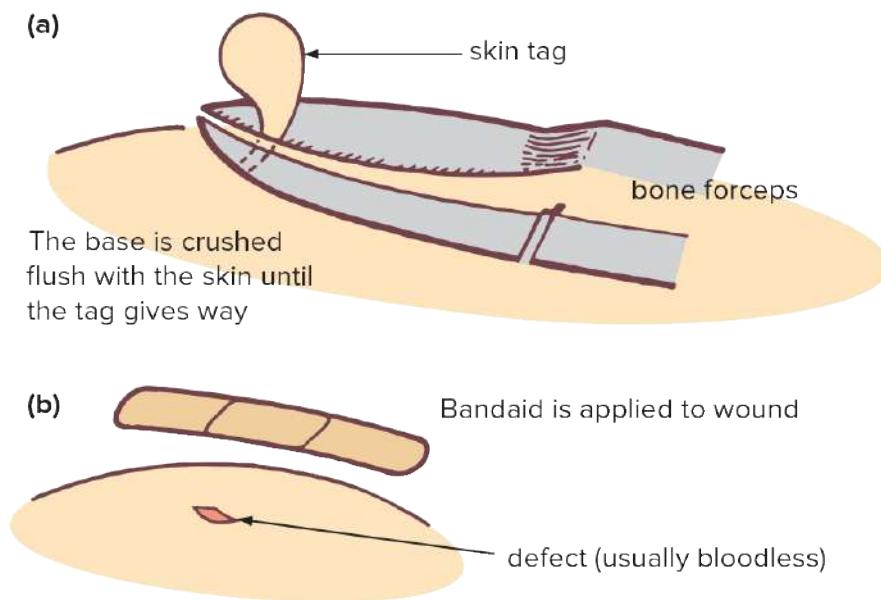
tie base with fine cotton or suture material

or

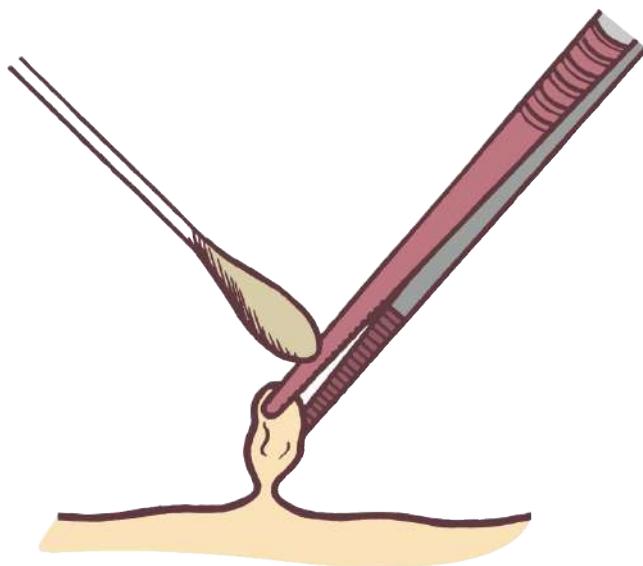
diathermy or electrocautery of base

or

apply liquid nitrogen (see FIG. 116.2 )



**FIGURE 116.1** Removal of skin tag using bone forceps



**FIGURE 116.2** Removal of skin tag by liquid nitrogen: a cotton bud soaked in liquid nitrogen is applied to the forceps, which grasp the tag firmly

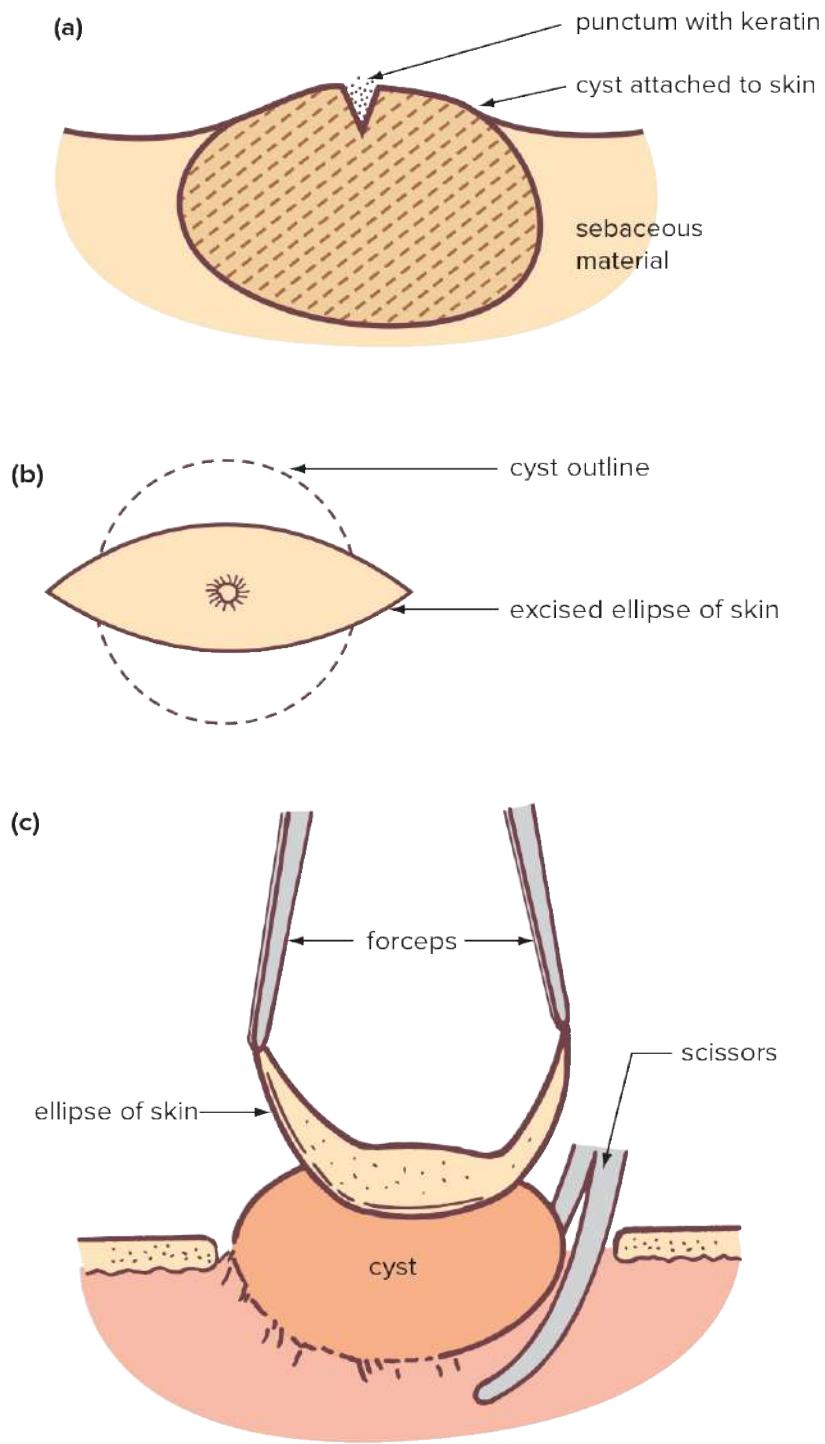
These methods do not require local anaesthetic.

## ⌚ Epidermoid (sebaceous) and pilar cysts

Synonyms: trichilemmal cyst, keratinous cyst, wen

### Clinical features

- Firm to soft regular lump (usually round)
- Fixed to skin but not to other structures (see FIG. 116.3A )
- Move with the skin
- Found in hair-bearing skin mainly on scalp—then face, neck, trunk, scrotum
- Contains keratinous material
- Usually fluctuant
- May be a central punctum containing keratin (pilar cysts on the scalp typically lack punctum)
- Tendency to painful inflammatory reaction with rupture of the cyst wall



**FIGURE 116.3** (a) Configuration of a sebaceous cyst; (b), (c) standard dissection of a large sebaceous cyst

## Management

If before puberty—think of polyposis coli or basal cell naevus syndrome. Can leave if small and not bothersome.

### Surgical removal methods

There are several methods of removing epidermoid cysts after infiltrating local anaesthetic over and around the cyst. These include:

- *Method 1: Incision into cyst* Most appropriate for cysts not previously incised or infected. Make an incision into the cyst to bisect it, squeeze the contents out with a gauze swab and then avulse the lining of the cyst with a pair of artery forceps or remove with a small curette.
- *Method 2: Incision over cyst and blunt dissection* Most appropriate for scalp pilar cysts. Make a careful skin incision over the cyst, taking care not to puncture its wall. Free the skin carefully from the cyst by blunt dissection. When it is free from adherent subcutaneous tissue, digital pressure will cause the cyst to ‘pop out’.
- *Method 3: Standard dissection* Incise a small ellipse of skin to include the central punctum over the cyst (see FIG. 116.3B ). The objective is to avoid rupture of the cyst. Inserting curved scissors (e.g. McIndoe scissors), free the cyst by gently opening and closing the blades (see FIG. 116.3C ). Bleeding is not usually a problem. Send the cyst for histopathology.

*Note:* Failure to remove the entire cyst wall, punctum and contents is likely to result in cyst recurrence and inflammation.

### Treatment of inflamed cysts

Incise the cyst to drain purulent material. When the inflammation has resolved completely, the cyst should be removed by method 3.

## § Implantation cyst

Synonym: implantation dermoid.

### Clinical features

- Small cystic swelling
- May be tender
- Usually follows puncture wounds
- Especially on finger pulp (e.g. hairdressers, sewers)
- Contains mucus

### Management

- Incision removal (similar to epidermoid cyst but there is no punctum to consider)

## **Mucocele**

A mucous retention cyst.

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### **Clinical features**

- A benign tumour
- Cyst containing mucus
- Appears spontaneously, most resolve spontaneously
- Common on lips and buccal mucosa
- Smooth and round
- Yellow or blue colour

### **Management**

- Those that do not resolve spontaneously may be treated with incision removal

## **Hyperplastic scarring**

### **Hypertrophic scar**

A hypertrophic scar is simply a lumpy scar caused by a nodular accumulation of thickened collagen fibres. It does not extend beyond the margins of the wound and regresses within a year but sometimes can be permanent.

### **Keloid**

A keloid is a special type of hyperplastic scar that extends beyond the margins of the wound.

### **Clinical features**

- Firm, raised, red-purple, skin overgrowth
- Common on ear lobes, chin, neck, shoulder, upper trunk
- Hereditary predisposition (e.g. dark-skinned person)
- Follows trauma, even minor (e.g. ear piercing)

- May be burning or itchy and tender

## Management of scarring

- Prevention (avoid procedures in keloid-prone individuals). If unavoidable, minimise wound tension.
- Compression and silicone dressings.
- Intradermal injection of corticosteroids in early stages (2–3 months) or intralesional cytotoxics (e.g. fluorouracil) or radiotherapy of surgical wounds within 24–48 hours of operation.<sup>3</sup>
- Consider re-excision of hypertrophic scarring with caution.

## ⌚ Warts and papillomas

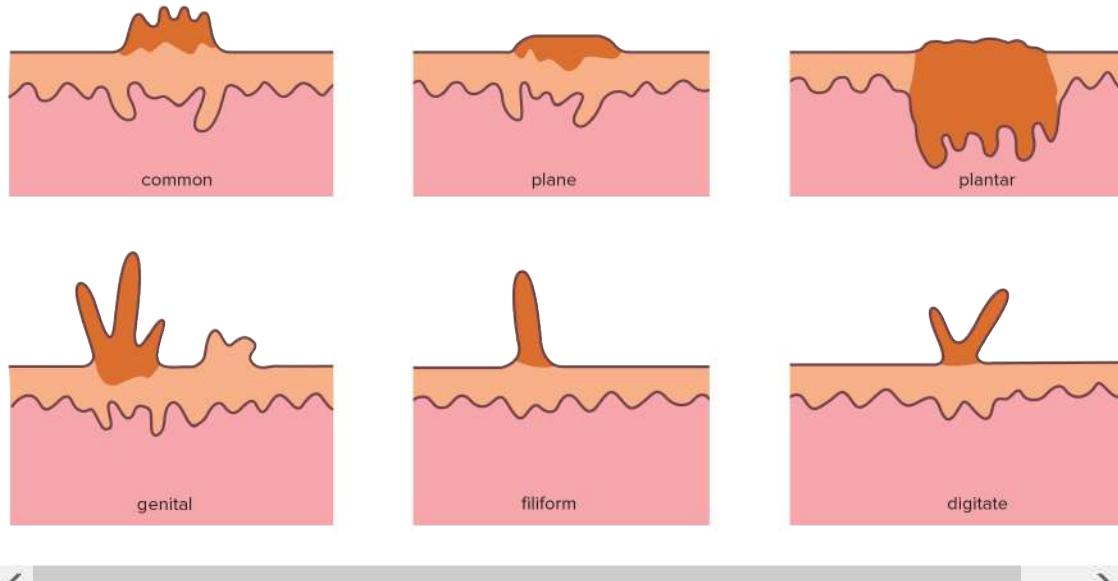
Warts are skin tumours caused by the human papillomavirus (HPV). The virus invades the skin, usually through a small abrasion, causing abnormal skin growth. Warts are transmitted by direct or fomite contact and may be autoinoculated from one area to another.<sup>4</sup>

### Clinical features

- Average incubation period—4 months
- Increased incidence in children and adolescents
- Peak incidence around adolescence
- Occurs in all ethnicities at all ages
- About 25% resolve spontaneously in 6 months<sup>4</sup> and 70% in 2 years
- Present as various types

### Types of warts

These include common warts, plane warts, filiform warts (fine elongated growths, usually on the face and neck), digitate warts (finger-like projections, usually on scalp), genital and plantar warts (see FIG. 116.4 ).



**FIGURE 116.4** Configuration of various types of warts

### Common warts

These are skin-coloured tumours with a rough surface, found mainly on the fingers, elbows and knees.

### Plane warts

These are skin-coloured, small and flat, occurring in linear clusters along scratch lines (see FIG. 116.5 ). They mainly occur on the face and limbs. They are difficult to treat because they contain very few virus particles. They are prone to Koebner phenomenon, which is seeding when a scratch passes through a plane wart.



**FIGURE 116.5** Plane warts on the dorsum of the hand

### Treatment options

Topical applications:<sup>4</sup>

- salicylic acid—for example: 5–20% in flexible collodion (apply daily or bd) or 16–17% salicylic acid + 16–17% lactic acid
- formaldehyde 2–4% alone or in combination
- podophyllotoxin 0.5% for anogenital warts, 0.5% paint for external keratinised skin, 0.15% cream for the perianal area, introital area and under the foreskin
- cytotoxic agents (e.g. 5-fluorouracil: very good for resistant warts such as plane warts and periungual warts)
- the immunomodulator, imiquimod 5%

### Cryotherapy

Carbon dioxide ( $-56.5^{\circ}\text{C}$ ) or liquid nitrogen ( $-195.8^{\circ}\text{C}$ ) destroys the host cell and stimulates an immune reaction.

*Note:*

- excessive keratin must be pared before freezing
- the results are often disappointing

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## Curettage

A most common treatment; some plantar warts can be removed under LA with a sharp spoon curette. The problem is a tendency to scar, so avoid over a pressure area such as the sole of the foot.

## Electrodesiccation

A high-frequency spark under LA is useful for small, filiform or digitate warts. A combination of curettage and electrodesiccation is suitable for large and persistent warts. Practitioners should ensure protection from inhalation of viral particles in smoke plume.

## Vitamin A and the retinoids

- Topical retinoic acid (e.g. tretinoin 0.1% cream—Retin-A) is effective on plane warts
- Systemic oral retinoid, acitretin (Neotigason) for extensive recalcitrant warts (with care)

## Medication

- Consider cimetidine (although studies show poor efficacy)

## Specific wart treatment

The method chosen depends on the type of wart, its site and the patient's age.

- *Plantar warts*: refer to [CHAPTER 57](#)
- *Genital warts*: podophyllotoxin 0.5% paint or imiquimod 5% (best for penile warts, see [CHAPTER 109](#))
- *Filiform and digitate warts*: liquid nitrogen or electrodessication
- *Plane warts*: liquid nitrogen; salicylic acid 20% co; consider 5-fluorouracil cream or tretinoin 0.05% cream (Retin-A)
- *Common warts*: a recommended method:
  - Soak the wart/s in warm soapy water.
  - Rub back the wart surface with a pumice stone.
  - Apply keratolytic paint (only to the wart; protect the surrounding skin with Vaseline). The

paints: formalin 5%, salicylic acid 12%, acetone 25%, collodion to 100%.<sup>5</sup>

Do this daily or every second day.

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Carefully remove dead skin between applications.

*or (preferable applications)*

(adult) 16% salicylic acid, 16% lactic acid in collodion paint (Dermatec, Duofilm), apply once daily until wart cleared

(children) 8% salicylic acid, 8% lactic acid in collodion

Combined method: salicylic acid 70% paste in linseed oil. Leave 1 week then pare and freeze (cryotherapy).

- *Periungual warts (fingernails):* consider 5-fluorouracil or liquid nitrogen with care. Always use a paint rather than ointment or paste on fingers.

Specialised therapies include intralesional bleomycin, immunotherapy (e.g. topical diphencyprone—DPCP), cantharidin, carbon dioxide laser and pulsed dye laser.

## ¶ Pox virus lumps

Skin tumours can be caused by pox viruses, some of which result from handling infected sheep, cows and monkeys and other animals such as deer. Hence they are usually found in sheep shearers, farmers and zookeepers.

## Molluscum contagiosum

This common pox virus infection can be spread readily by direct contact, including sexual contact (see CHAPTER 109 ). The incubation period is 2–26 weeks.

### Clinical features

- Common in school-aged children
- Single or multiple (more common)
- Shiny, round, pink-white papule (see FIG. 116.6 )



**FIGURE 116.6** Molluscum contagiosum with the round, pink, pearly appearance and central punctum

- Hemispherical up to 5 mm
- Central punctum gives umbilical look
- Can be spread by scratching

### Management

They are difficult to treat. Avoid using the bath—they spread to other body parts and those sharing the bath. Showering is preferable. In immune competent children, approximately half of cases clear in 12 months and two-thirds by 18 months. There is a strong case for simply reassuring the family and waiting for spontaneous resolution. Coexisting eczema should be treated to avoid scratching and spread.

### Treatment options

- Liquid nitrogen with care (a few seconds following topical anaesthetic), then dry dressings for 2 weeks
- Pricking the lesion with a pointed stick soaked in 1% or 2.5% phenol
- Application of 15% podophyllotoxin
- Application of 30% trichloroacetic acid
- Application of imiquimod 0.1% cream tds for 6 weeks
- Destruction by curettage, electrocautery or diathermy

- Ether soap and friction method
- Lifting open the tip with a sterile needle inserted from the side (parallel to the skin) and applying 10% povidone-iodine (Betadine) solution (parents can be shown this method and continue to use it at home for multiple tumours)
- If more localised, covering with a piece of Micropore or Leukosilk tape—change every day after showering (may take a few months). This method also prevents spread
- For large areas, aluminium acetate (Burow's solution 1:30) applied bd can be effective

*Note:* The extract of the *Cantharis beetle* (cantharidin) (prepared as Cantharone) if available is reportedly very effective.

Avoid treatments that may cause scarring, especially as the condition is usually self-resolving.

## Orf

Orf is due to a pox virus and presents as a single papule or group of papules on the hands of sheep-handlers after handling lambs with contagious pustular dermatitis. The papules change into pustular-like nodules or bullae with a violaceous erythematous margin. It clears up spontaneously in about 3–4 weeks without scarring and usually no treatment is necessary. Intralesional steroids<sup>6</sup> and topical imiquimod<sup>7</sup> application can hasten resolution.

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### Practice tip for orf

Rapid resolution (days) can be obtained by an intralesional injection of triamcinolone diluted 50:50 in normal saline.<sup>6</sup>

## Milker's nodules (pseudo cowpox)

In humans 2–5 papules appear on the hands about 1 week after handling cows' udders or calves' mouths. It is caused by a parapoxvirus. The papules enlarge to become tender, grey nodules with a necrotic centre and surrounding inflammation (see FIG. 116.7). The patient can be reassured that the nodules are a self-limiting infection and spontaneous remission will occur in 5–6 weeks without residual scarring. One infection gives lifelong immunity.



**FIGURE 116.7** Milker's nodule in a person who milks cows showing the grey nodule with the necrotic centre

### Practice tip for milker's nodule

Rapid resolution (days) can be obtained by an intralesional injection of triamcinolone diluted 50:50 in normal saline.<sup>6</sup>

## ⌚ Seborrhoeic keratoses

Synonyms: seborrhoeic warts, senile warts, senile keratoses (avoid these terms).

### Clinical features

- Very common
- There are a variety of subtypes
- Increasing number and pigmentation with age >40 years
- Sits on skin, appears in some like a ‘sultana’ pressed into the skin (i.e. well-defined border)
- Has a ‘pitted’ surface (see FIG. 116.8 )



**FIGURE 116.8** Seborrhoeic keratosis in a 70-year-old man. The large pigmented warty mass appears to sit on top of the skin.

*Photo courtesy Robin Marks*

- May be solitary but usually multiple
- Common on face and trunk, but occurs anywhere
- Usually asymptomatic
- Usually causes patients some alarm (confused with melanoma)

## Management

- Usually nil apart from reassurance
- Does not undergo malignant change
- Can be removed for cosmetic reasons
- Light cautery to small facial lesions or ablative laser therapy
- Freezing with liquid nitrogen (especially if thin) decolours the tumour
- 10% (or stronger) phenol solution applied carefully—repeat in 3 weeks
- Apply trichloroacetic acid to surface: instil gently by multiple pricks with a fine-gauge needle, twice weekly for 2 weeks
- May drop off spontaneously

- If diagnosis uncertain, remove for histopathology

## ⌚ Stucco keratoses

This subtype of seborrhoeic keratoses comprises multiple, non-pigmented (often white), small, friable keratoses over the lower legs. They can be treated with a topical keratolytic such as 3–5% salicylic acid in sorbolene.

## ⌚ Granuloma annulare

Granuloma annulare is a common condition where grouped papules are arranged in an annular fashion.

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### Clinical features

- Most common among children and young adults
- Firm papules grouped in a ‘string of pearls’ pattern (see FIG. 116.9 )



**FIGURE 116.9** Granuloma annulare: this annular plaque was longstanding on this finger. After taking a small biopsy, 20 mg of methylprednisolone acetate was injected into the tumour.

- Dermal nodules
- May be associated with minor trauma
- Possible association with diabetes, hypercholesterolaemia

- Usually on dorsum or sides of fingers (knuckle area), backs of hands, the tops of feet, elbows and knees
- Usually self-limiting, but may recur or persist for years

## Management<sup>8</sup>

- Consider checking blood sugar, lipids
- Give reassurance (about half subside by 2 years)
- Cosmetic reasons:
  - first-line: potent topical corticosteroids ± occlusion, apply bd for minimum of 4–6 weeks
  - if ineffective: intradermal injection into the extending outer margin of triamcinolone 10% or similar corticosteroid (dilute equal volume with N saline); can repeat at 6-weekly intervals if effective

## § Pyogenic granuloma

Synonyms: granuloma, granuloma telangiectaticum, acquired haemangioma.

A pyogenic granuloma is a 5–10 mm soft vascular lesion (without pus) due to a proliferation of capillary vessels. It is considered to be an abnormal reaction to minor trauma (see FIG. 116.10 ).



**FIGURE 116.10** Pyogenic granuloma showing bright red, friable tumour on face. It followed a puncture from a spiky plant in the garden.

*Photo courtesy Robin Marks*

## Clinical features

- Common in children and young adults, pregnant women
- Usually on hands and face
- Bright red ‘raspberry’-like lesion
- Raised, sometimes pedunculated
- Friable—bleeds easily

Beware of misdiagnosing pyogenic granuloma for a nodular melanoma.

## Management

It must be distinguished from amelanotic melanoma or anaplastic SCC. Shave biopsy or curettage with electrocautery of base. The specimen must be sent for histological examination.

## Dermatofibroma

Synonyms: sclerosing haemangioma; histiocytoma.

This is a common pigmented nodule arising in the dermis due to a proliferation of fibroblasts, believed to develop as an abnormal response to minor trauma including insect bites. The nodule gives a characteristic button-like feel and dimpling when laterally compressed (pinched) from the side with the fingers.

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## Clinical features

- Usually multiple
- Firm, well-circumscribed nodules
- Oval, 0.5–1 cm in diameter
- Freely mobile over deeper structures
- Slightly raised in relation to skin
- Mainly on limbs, especially legs
- May itch
- Mainly in women
- Variable colour, pink or brown, tan or grey or violaceous

- Characteristic ‘dimple’ sign on pinching margins

## Treatment

- Reassurance
- Simple excision if requested

## Sebaceous hyperplasia

Sebaceous hyperplasia presents as single or multiple papules on the face, especially in older people. The papules are small, yellow–pink, slightly umbilicated and are found in a similar distribution to basal cell carcinomas (BCCs), for which they may be mistaken. There is no need for surgical excision. Cryotherapy or fine wire diathermy achieves good results.

## Skin cancer

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In Australia, over one million general practice consultations are due to skin cancer. The three main skin cancers are the non-melanocytic skin cancers—basal cell carcinoma (BCC) and squamous cell carcinoma (SCC)—and melanoma. The approximate relative incidence is BCC 80%, SCC 15–20% and melanoma less than 5%.<sup>9</sup> Two out of every three Australians will be diagnosed with a skin cancer before the age of 70.<sup>10</sup> About 70% of skin cancer deaths are due to melanoma and the rest mainly due to SCC.<sup>9</sup>

## Solar keratoses<sup>11</sup>

Solar keratoses (actinic keratoses or sun spots) are reddened, adherent, scaly hyperkeratotic thickenings occurring on light-exposed areas. They represent intra-epidermal keratinocytic dysplasia with a potential for malignant change, especially on the ears.

### Clinical features

- Sun-exposed fair skin
- Mainly on face, ears, scalp (if balding), forearms, dorsum of hands (especially) (see FIG. 116.11 )
- Vary in size from 2–20 mm in diameter
- Dry, rough, adherent scale
- Usually asymptomatic
- Discomfort on rubbing with towel
- Scale can separate to leave oozing surface

- A small proportion undergo malignant change



**FIGURE 116.11** Solar keratoses showing the reddened, scaly thickenings on sun-exposed areas. Biopsy of one of the lesions proved SCC.

### Management<sup>11</sup>

- Can disappear spontaneously
- Strict sun protection may prevent and improve actinic change
- Liquid nitrogen
- Topical field treatment for patients with multiple solar keratoses or who cannot tolerate repeated cryotherapy:
  - 5-fluorouracil 5% cream twice daily for 2 weeks on face or 3–4 weeks on arms and legs  
*or*  
imiquimod 5% cream, once daily 3 times a week for 3-4 weeks (for one to three cycles with 4-week spells between cycles)  
*or*  
ingenol mebutate 0.015% gel topically on face or scalp, once daily for 3 consecutive days  
*or*  
ingenol mebutate 0.05% gel topically on trunk or limbs, once daily for 2 consecutive days  
*or*  
diclofenac 3% gel twice daily for 90 days
- Surgical excision for suspicious and ulcerating lesions
- Other treatments include ablative laser, chemical peels and photodynamic therapy

- Tenderness on lateral pressure requires a biopsy to exclude SCC
- Biopsy if doubtful

*Note:* Topical field treatments cause severe inflammation that can last up to several weeks; warn the patient and show them the expected erythema using patient information hand-outs.

Fluorouracil is most commonly used due to its lower cost but causes inflammation for several weeks. Ingenol mebutate produces dramatic erythema and vesiculation within 24–48 hours, but the skin heals in approximately 10–14 days. Diclofenac is the best tolerated but maintained compliance is an issue.

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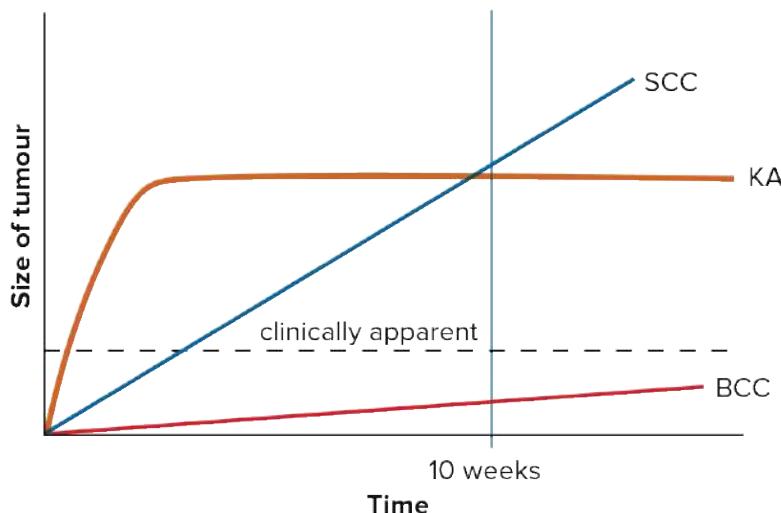
## Terminology

**Solar keratoses** ‘sun spots’

**Solar lentigines** ‘age spots’ or ‘liver spots’

## ¶ Keratoacanthoma

Keratoacanthomas (KA), which are rapidly evolving tumours of keratinocytes, occur singly on light-exposed areas. They are now considered a low-risk variant of SCC.<sup>11</sup> The major problem is they can be clinically and histopathologically indistinguishable from SCCs. The relative growth rates of three types of skin tumours are shown in FIGURE 116.12 .



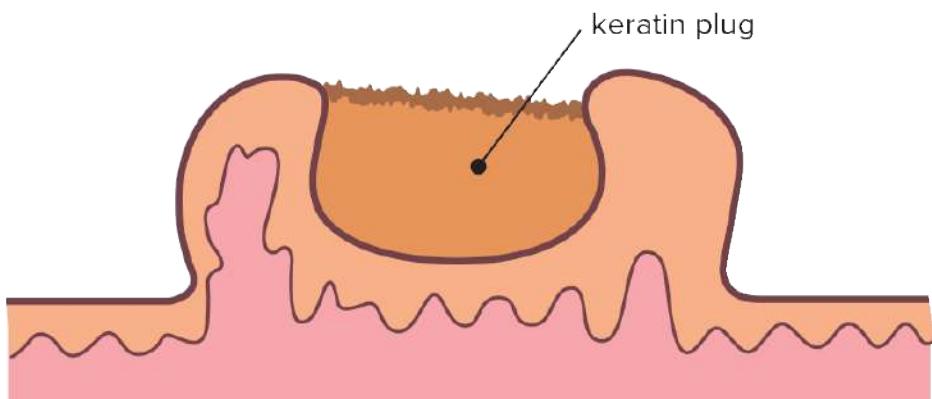
**FIGURE 116.12** Relative growth rates of three types of skin tumours: keratoacanthoma, squamous cell carcinoma and basal cell carcinoma

## Clinical features

- Rapidly growing lesion on sun-exposed skin
- May be precipitated by trauma
- Raised crater with central keratin plug (see FIGS 116.13 and 116.14 )
- Typically 5–15 mm, may grow to 2 cm or more
- Can be painful or asymptomatic
- Arises over a few weeks, remains static, then spontaneously disappears after about 4–6 months; can leave a large atrophic scar



**FIGURE 116.13** Keratoacanthoma: this tumour, with its central plug, appeared suddenly on the face of a 63-year-old man. It may be confused with squamous cell carcinoma. Surgical excision is appropriate treatment.



**FIGURE 116.14** A typical keratoacanthoma

## Management

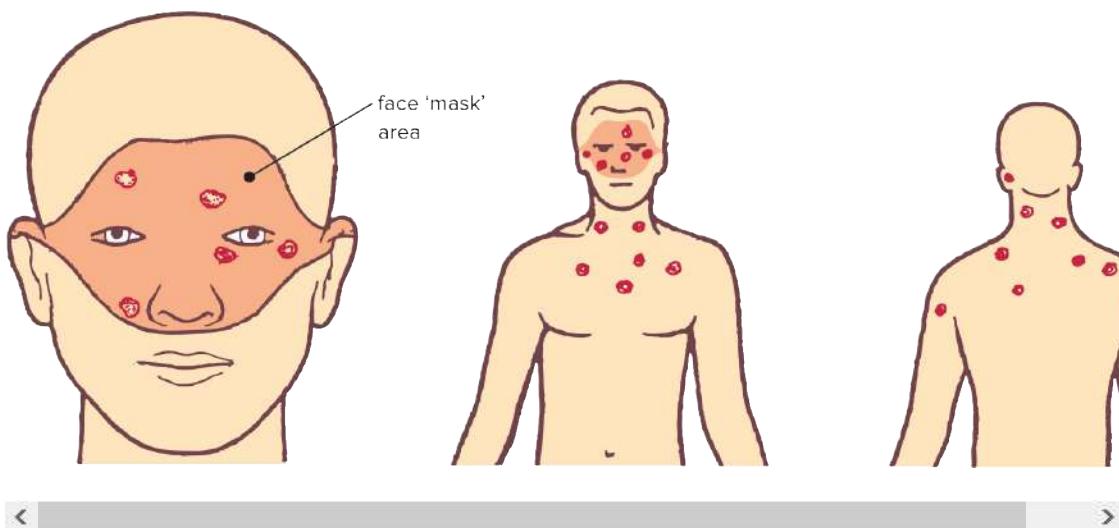
- Remove by excision—perform biopsy
- If clinically certain—curettage/diathermy
- Treat as SCC (by excision) if on lip/ear

The recommended treatment is surgical excision with 2–4 mm margins. Histological examination with clear margins should be obtained.

## § Basal cell carcinoma<sup>12</sup>

### Clinical features

- Most common skin cancer (70–80% of non-melanoma skin cancers)
- Age: usually >35 years
- More frequent in males
- Mostly on sun-exposed areas: face (mainly), neck, upper trunk, limbs (10%) (see FIG. 116.15 )



**FIGURE 116.15** Typical areas in which basal cell carcinomas occur

- May ulcerate easily = ‘rodent ulcer’
- Slow-growing over years

- Has various forms: nodular, pigmented, ulcerated, etc.
- Stretching the skin demarcates the lesion, highlights pearliness and distinct margin
- Metastases very rare (<0.1%, usually if untreated or multi-recurrent)
- Local spread is a problem (see FIG. 116.16 )
- Can spread deeply, especially infiltrative tumours of the nose, eye or ear

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## Red flag pointer for BCC



**FIGURE 116.16** Grossly neglected basal cell carcinoma on back

*Photo courtesy Robin Marks*

## Clinical types

1. Cystic nodular—translucent or pale grey
2. Ulcerated—nodular BCC that has necrosed centrally
3. Pigmented—usually spotted, may be all black
4. Superficial—erythematous scaly patch, may be misdiagnosed as eczema or psoriasis
5. Morphoeic (fibrotic)—scar-like, poorly defined margin

5. Common: pearly edge, telangiectasia, ulcerated (see FIG. 116.17 )

7. Basosquamous—mixed BCC and SCC (more aggressive)



**FIGURE 116.17** Basal cell carcinoma showing a pearly nodular appearance with telangiectatic vessels

### Management<sup>12</sup>

- Simple elliptical excision (3–4 mm margin) is best.
- If not excision, do biopsy before other treatment.
- Radiotherapy is an option, especially in frail people.
- Mohs micrographic surgery—a form of surgical treatment suitable for large aggressive or recurrent tumours or those in a site where maximal normal tissue needs to be preserved.
- Imiquimod 5% daily 5 times a week for 6 weeks, for biopsy-proven low-risk superficial BCC on the body.
- Photodynamic therapy—response rate is about 80% for superficial BCCs, less for nodular.
- Cryotherapy is suitable for well-defined, histologically confirmed, superficial tumours at sites away from head and neck. Use judiciously and infrequently.

## § Squamous cell carcinoma<sup>11</sup>

SCC is the second most common type of skin cancer. It is found on sun-exposed areas, especially in fair-skinned people. It tends to arise in premalignant areas such as solar keratoses, burns, chronic ulcers, leukoplakia and Bowen disease, or it can arise de novo. Keratoacanthoma is

considered a variant.

*Note:* Although BCC and SCC are related to cumulative sun exposure, they are not always found in sun-exposed areas.

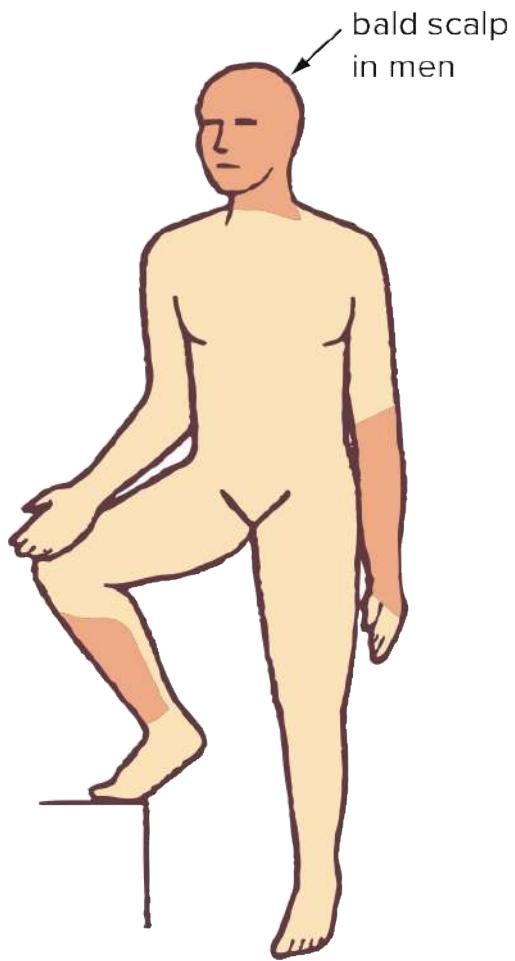
## Clinical features

- Usually >50 years
- Initially firm thickening of skin, especially in solar keratosis
- Surrounding erythema
- The hard nodules may ulcerate (see FIG. 116.18 )



**FIGURE 116.18** Squamous cell carcinoma. This recurrent, non-healing lesion on the index finger of a 58-year-old man had raised hard edges and was fixed to tendon and bone. Treatment was by surgical amputation of the finger.

- Occurs on the hands and forearms and the head and neck (see FIG. 116.19 )



**FIGURE 116.19** Common sites of squamous cell carcinoma

- Ulcers have a characteristic everted edge
- Metastasis rate of 3–5%, higher risk if on the head and neck, large, deep or poorly differentiated and for immunosuppressed patients
- SCCs of ear, lip, oral cavity, tongue and genitalia are serious and need special management

### Management

- Early excision of tumours <1 cm with a 3–5 mm margin, to deep fat level.
- Referral for specialised surgery and/or radiotherapy if large, in difficult site or lymphadenopathy.
- SCCs of the ear and lip, which have considerably more malignant potential, can be excised by wedge excision.

- There is no alternative to surgery if the SCC is over cartilage—central nose or helix.

*Note:* Surgery is the treatment of choice for most tumours; cryotherapy, imiquimod and curettage are not.

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Radiotherapy is an optional treatment in a biopsy-proven tumour where surgery is not feasible or will cause unacceptable morbidity.

## **Bowen disease<sup>11</sup>**

Intra-epidermal carcinoma (Bowen disease) is SCC in situ of the skin. It begins as a slowly enlarging, sharply demarcated, thickened, red plaque, especially on the lower legs of females. It may resemble solar keratosis, dermatitis or a patch of psoriasis. It remains virtually unchanged for months or years. It may become very crusty, ulcerate or bleed. It has a potential for malignant change since it is a full-thickness SCC in situ.

### **Management**

- Biopsy first for diagnosis
- Surgical excision if small (highest cure rate)
- Skin grafting may be required
- Cryotherapy suitable for small lesions
- Curettage with electrodessication an option for small lesions on the body
- Topical therapy with fluorouracil 5% cream twice daily for 6 weeks

*Note:* Biopsy a single patch of suspected psoriasis or dermatitis not responding to topical steroids.

## **Lumps on ears**

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Lumps on ears, especially on the helix, demand close attention. SCCs that arise here have a 15% metastasis rate and demand early wedge resection.

Causes of ear lumps include:

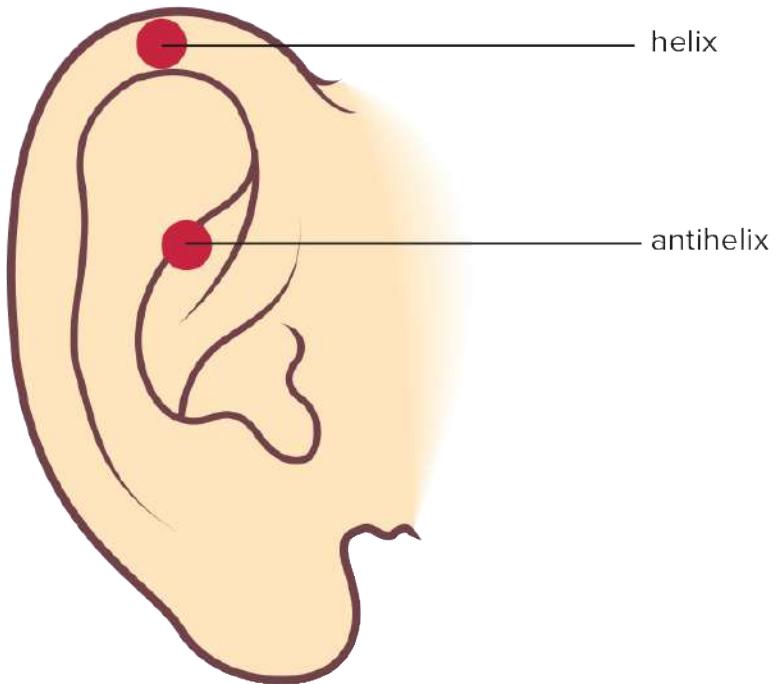
- solar keratosis
- BCC
- SCC
- keratoacanthoma

- gouty tophi
- chondrodermatitis nodularis helicis

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## ⌚ Chondrodermatitis nodularis helicis<sup>13</sup>

This lump, which is not a neoplasm, presents as a tender nodule on the most prominent part of the helix or antihelix of the ear (see FIGS 116.20 and 116.21). It is seen more often in men while it is found more often on the antihelix in women, thought to be due to points of local pressure and exposure. It appears to be caused by pressure between the head and the pillow at night. Histologically, a thickened epidermis overlies inflamed cartilage. It resembles a small corn, is tender and affects sleep if that side of the head lies on the pillow. It can be treated initially by cryotherapy or an intralesional injection of triamcinolone. Patients should aim to sleep on the opposite side and avoid pressure. If other methods fail, surgical excision under local anaesthetic is an effective treatment.



**FIGURE 116.20** Typical sites of chondrodermatitis nodularis helicis



**FIGURE 116.21** Chondrodermatitis nodularis helicis on the right ear in a 44-year-old man

### ⌚ Malignant melanoma

These are usually enlarging pigmented lesions with an irregular, notched border. Refer to [CHAPTER 117](#) on pigmented skin lesions.

### ⌚ Secondary tumour

These complex tumours may metastasise from the lung, melanoma or bowel and may arise in surgical scars (e.g. for breast cancer).

### ⌚ Kaposi sarcoma<sup>14</sup>

Kaposi sarcoma is a tumour of vascular and lymphatic endothelium that is related to human herpes virus type 8. There are three types:

- ‘classic’ or ‘sporadic’ form of primary tumour seen mostly in elderly males of Mediterranean

or Ashkanazi Jewish descent

- ‘endemic’ form seen in males from Central Africa
- immunosuppressed-related form commonly associated with AIDS. Widespread lesions affect skin, bowel, oral cavity and lungs

Kaposi sarcoma presents as brownish-purple papules on the skin and mucosa (any organ).

Treatment is with radiotherapy, immunotherapy or chemotherapy.

## Lumps of subcutaneous and deeper structure

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### ⌚ Lipoma

Lipomas are common benign tumours of mature fat cells situated in subcutaneous tissue.

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### Clinical features

- Soft and may be fluctuant
- Well defined; lobulated (see FIG. 116.22 )



**FIGURE 116.22** Lipoma: this 66-year-old woman had a longstanding soft, fluctuant, rubbery lump of 18 years. It was surgically removed for cosmetic reasons

- Soft to rubbery consistency

- May be one or many
- Painless
- Most common on limbs (especially arms) and trunk
- Can occur at any site

## Management

- Reassurance about benign nature
- Surgical excision for cosmetic reasons or to relieve discomfort from pressure

### Surgical excision

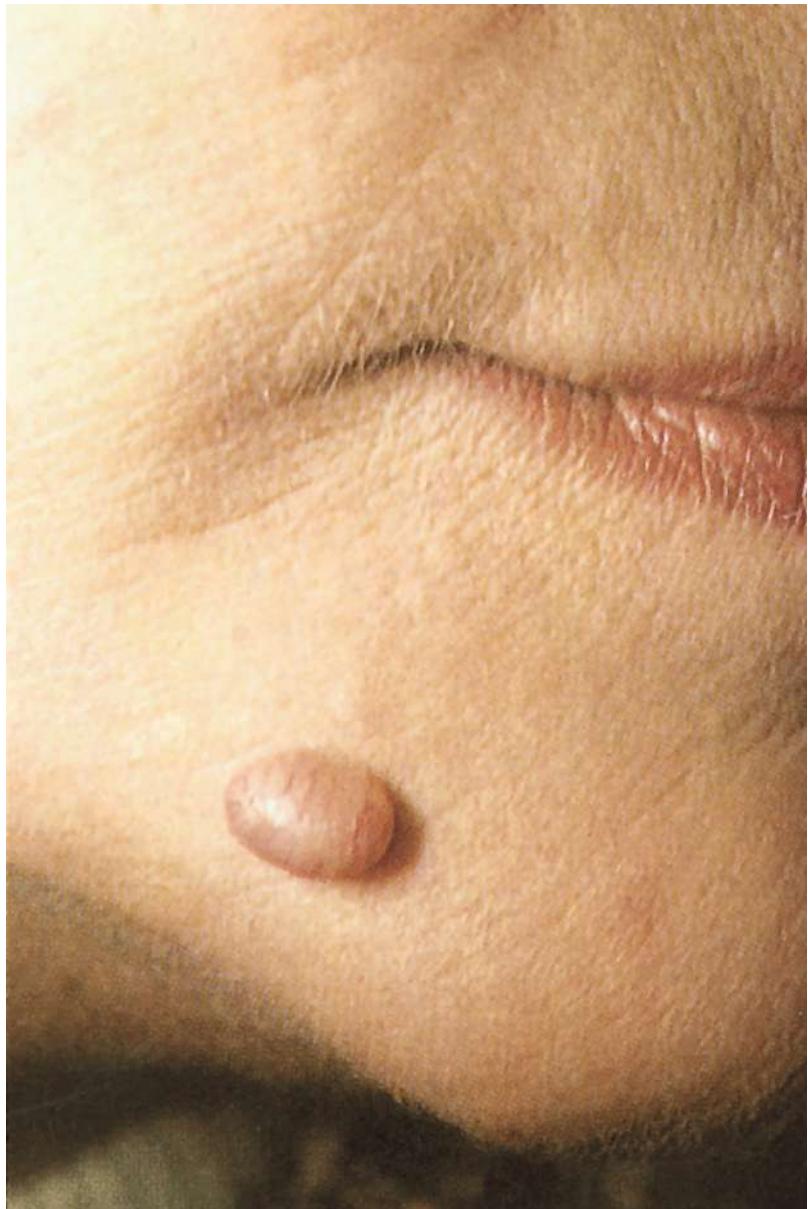
Many lipomas can be enucleated using a gloved finger, but there are a few traps: some are deeper than anticipated, and some are adjacent to important structures such as large nerves and blood vessels. Others are tethered by fibrous bands, and can recur. Recurrence is also possible if excision is incomplete. Attention to haemostasis and layered closure including the subcutaneous tissues is important to prevent haematoma, infection and scar dimpling.

*Caution:* Lipomas on back (don't shell out easily). If >5 cm consider referral.

*Note:* Ultrasound is good at assessing depth of lipoma. CT scan or MRI will help diagnose and define lipomas that are large or deep.

## Neurofibroma

These benign tumours are firm (sometimes soft), painless, subcutaneous lumps, most frequently found on the trunk, head and proximal limbs (see FIG. 116.23 ). Neurofibromas may be invaginated with direct digital pressure, exhibiting ‘the buttonhole sign’. When many lesions are present exclude neurofibromatosis. No treatment is required for solitary lesions, but they may be surgically excised when problematic.



**FIGURE 116.23** Neurofibroma. This mobile firm subcutaneous lump was tender to firm pressure.

## ⌚ Bursae

Bursae are cystic sacs between the skin and an underlying bony prominence or sacs of gelatinous fluid that separate and aid gliding of adjacent tendons and ligaments.

## ⌚ Pseudoaneurysm<sup>15</sup>

A pseudoaneurysm is a sac-like dilatation of the arterial wall (but not all three layers of the wall).

It presents as an expanding subcutaneous nodule located close to a superficial artery. It can form after blunt or penetrating trauma that injures the vessel, leading to a haemorrhage into the vessel wall. Investigate with ultrasound and manage with caution. Refer to a vascular surgeon for surgical management.

## Ganglion

Ganglia are firm cystic lumps associated with joints or tendon sheaths.

### Clinical features

- Deep subcutaneous lumps
- Around joints or tendon sheaths (see FIG. 116.24 )
- Mostly around wrists, fingers, dorsum of feet
- Immobile, fixed to deep tissues
- Translucent
- Contain viscid gelatinous fluid
- Associated with arthritis and synovitis
- May disappear spontaneously
- Recurrences common



**FIGURE 116.24** Ganglion of wrist: firm, immobile and translucent. It was eventually treated by aspiration of gelatinous fluid followed by infusion of

40 mg methylprednisolone acetate.

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## Management

- Can be left—wait and see
- Do not ‘bang with a Bible’
- Needle aspiration and steroid injection

*or*

surgical excision (can be difficult)

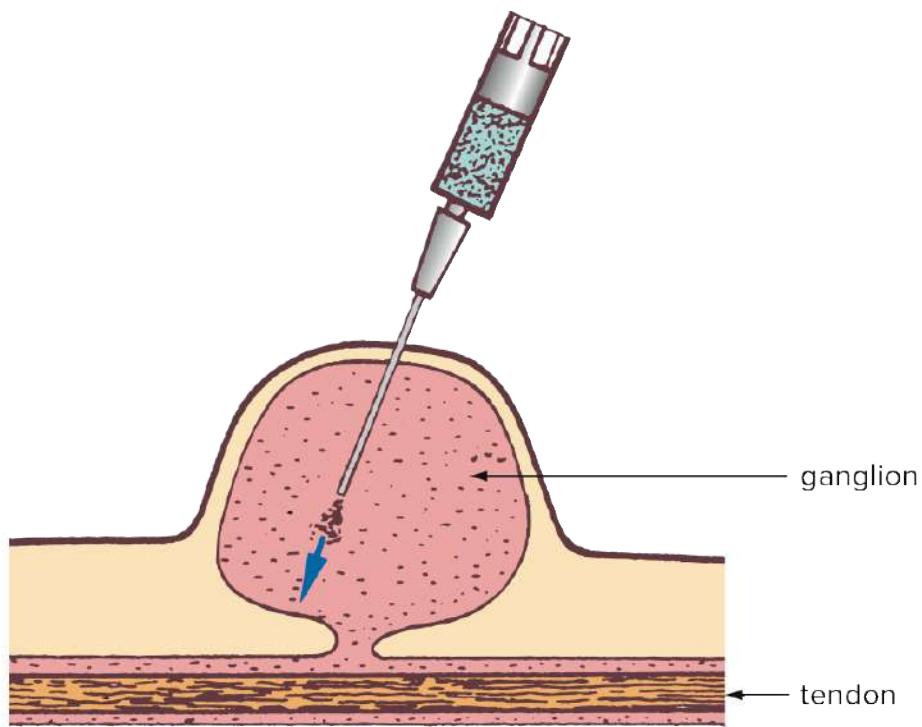
- Suture compression technique: a larger gauge catgut suture is inserted through the middle of the ganglion and firmly tied over it. Side pressure may express the contents through the needle holes. Remove the knot 12 days later.

## Injection treatment of ganglia

Ganglia have a high recurrence rate after treatment, with a relapse rate of 30% after surgery. A simple, relatively painless and more effective method is to use intralesional injections of long-acting corticosteroid, such as methylprednisolone acetate.<sup>16</sup>

### Method

1. Insert a 21 gauge needle attached to a 2 mL or 5 mL syringe into the cavity of the ganglion.
2. Aspirate some (not all) of its jelly-like contents, mainly to ensure the needle is in situ.
3. Keeping the needle exactly in place, swap the syringe for an insulin syringe containing up to 0.5 mL of steroid.
4. Inject 0.25–0.5 mL (see FIG. 116.25 ).
5. Rapidly withdraw the needle, pinch the overlying skin for several seconds and then apply a light dressing.
6. Review in 7 days and, if still present, repeat the injection using 0.25 mL of steroid.



**FIGURE 116.25** Injection treatment of ganglion

Up to six injections can be given over a period of time, but 70% of ganglia will disperse with only one or two injections.<sup>13</sup> Be aware of possible subsequent hypopigmentation of overlying skin, especially in people with darker skin.

## Some preferred therapeutic options

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### Liquid nitrogen therapy

Ideally, liquid nitrogen is stored in a special large container and decanted when required into a small thermos flask or spray device. The common methods are.

- 1. use of cotton wool pledge
- 2. high pressure spray.

Method A: this method of application to superficial skin tumours (see TABLE 116.2) is via a ball of cotton wool rolled rather loosely on the tip of a wooden applicator stick. The ball of cotton wool should be slightly smaller than the lesion, to prevent freezing of the surrounding skin.

<b>Table 116.2</b>	Superficial skin tumours suitable for
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## cryotherapy

Warts (plane, periungual, plantar, anogenital)

Skin tags

Seborrhoeic keratoses

Molluscum contagiosum

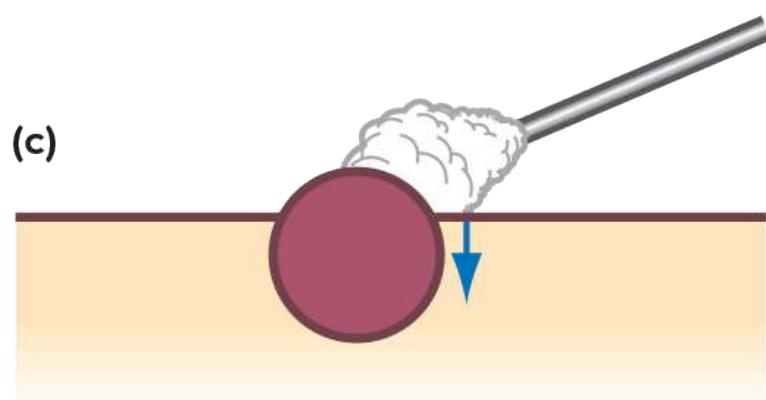
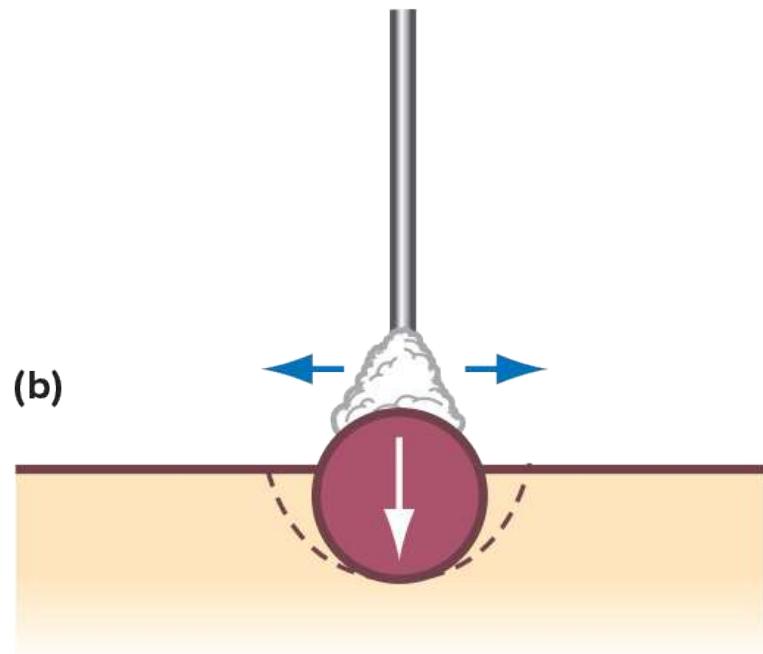
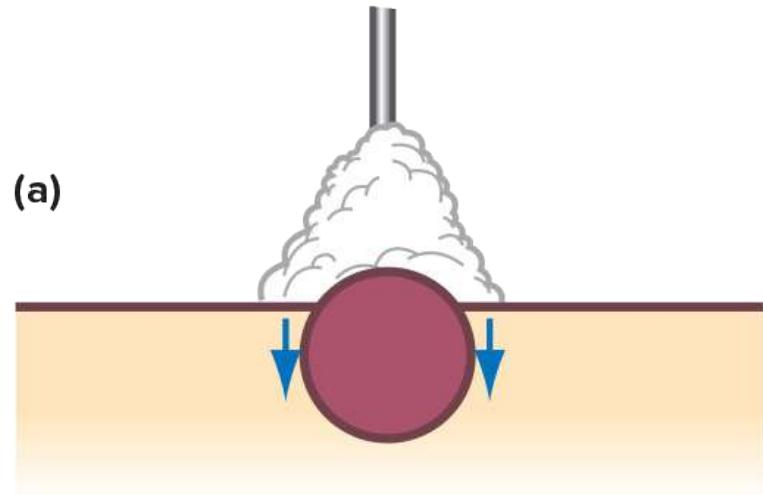
Sebaceous hyperplasia

Solar keratoses

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### **Method A (basic steps)**

1. Inform the patient what to expect.
2. Pare excess keratin with a scalpel.
3. Use a cotton wool applicator slightly smaller (not larger—see FIG. 116.26A ) than the lesion.
4. Place applicator vertically (see FIG. 116.26B and FIG. 116.26C ) on tumour surface.
5. Apply with firm pressure: do not dab.
6. Freeze until a 2 mm white halo appears around the lesion.



**FIGURE 116.26** Application of liquid nitrogen with cotton wool applicator: **(a)** applicator too large, **(b)** correct size and approach of applicator, **(c)** correct size but wrong position of applicator

### Method B: cryotherapy spray method

Spraying liquid nitrogen under high pressure (the timed spot freeze open-spray method) is the most effective method of cryotherapy. It produces sufficient intense cold to treat deeper lesions.

Explain likely reaction to patient, such as the appearance of blisters (possibly blood blisters). The optimal time for retreatment of warts is in 2–3 weeks (not longer than 3 weeks).

## Biopsies

There are various methods for taking biopsies from skin lesions. These include scraping, shaving and punch biopsies, all of which are useful but not as effective or safe as excisional biopsies.

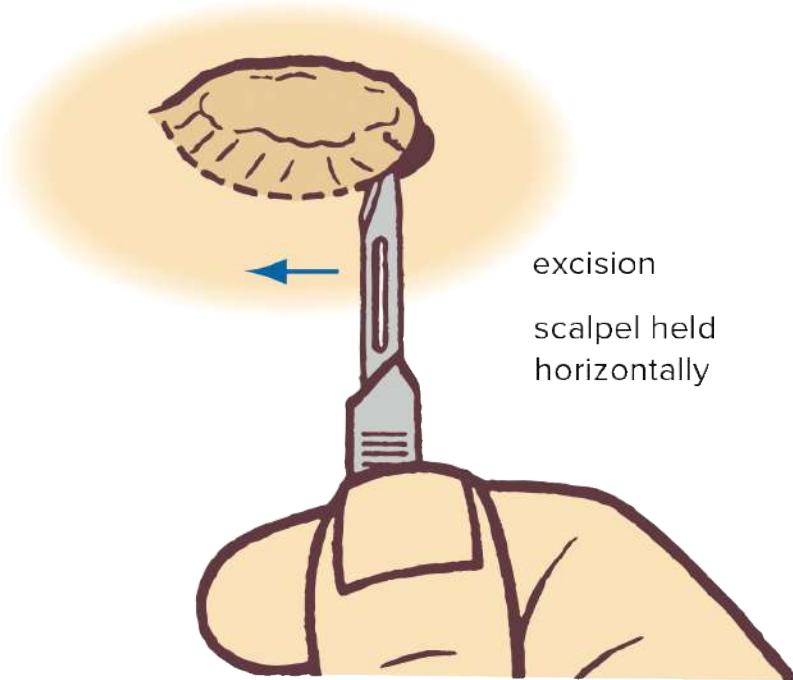
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### Shave biopsies

This simple technique is generally used for the tissue diagnosis of premalignant lesions and some malignant tumours, but not melanoma.

#### Method

1. Infiltrate with LA.
2. Holding a number 10 or 15 scalpel blade horizontally, shave off the tumour just into the upper dermis (see FIG. 116.27 ).
3. Diathermy may be required for haemostasis.

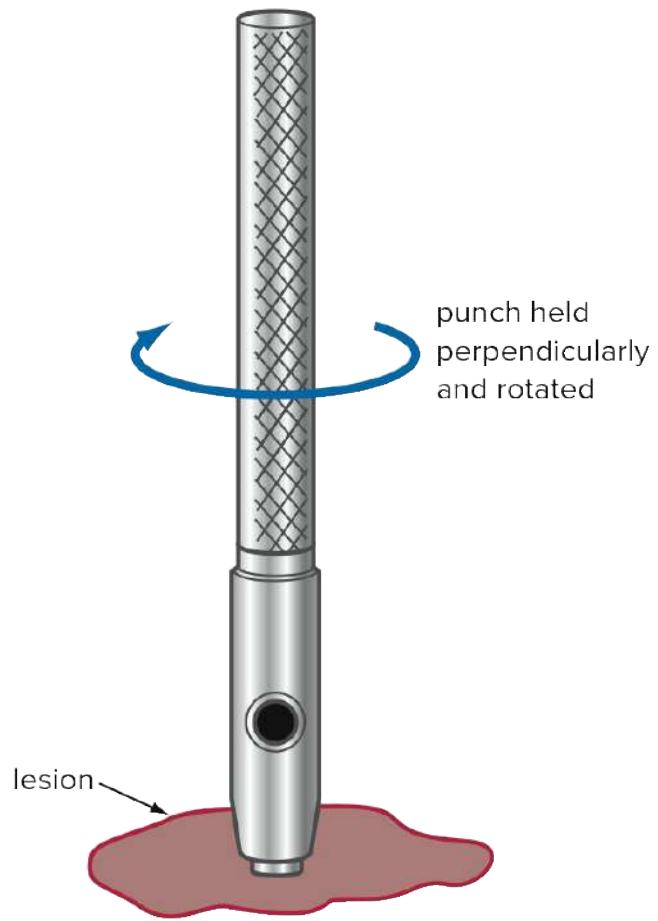


**FIGURE 116.27** Shave biopsy

The biopsy site usually heals with minimal scarring.

### Punch biopsy

This biopsy (see FIG. 116.28 ) has considerable use in general practice where full-thickness skin specimens are required for histological diagnosis. (Good quality disposable biopsy punches are available from Dermatech Laboratories.)



**FIGURE 116.28** Punch biopsy

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## Steroid injections into skin lesions

Suitable lesions for steroid injections are:

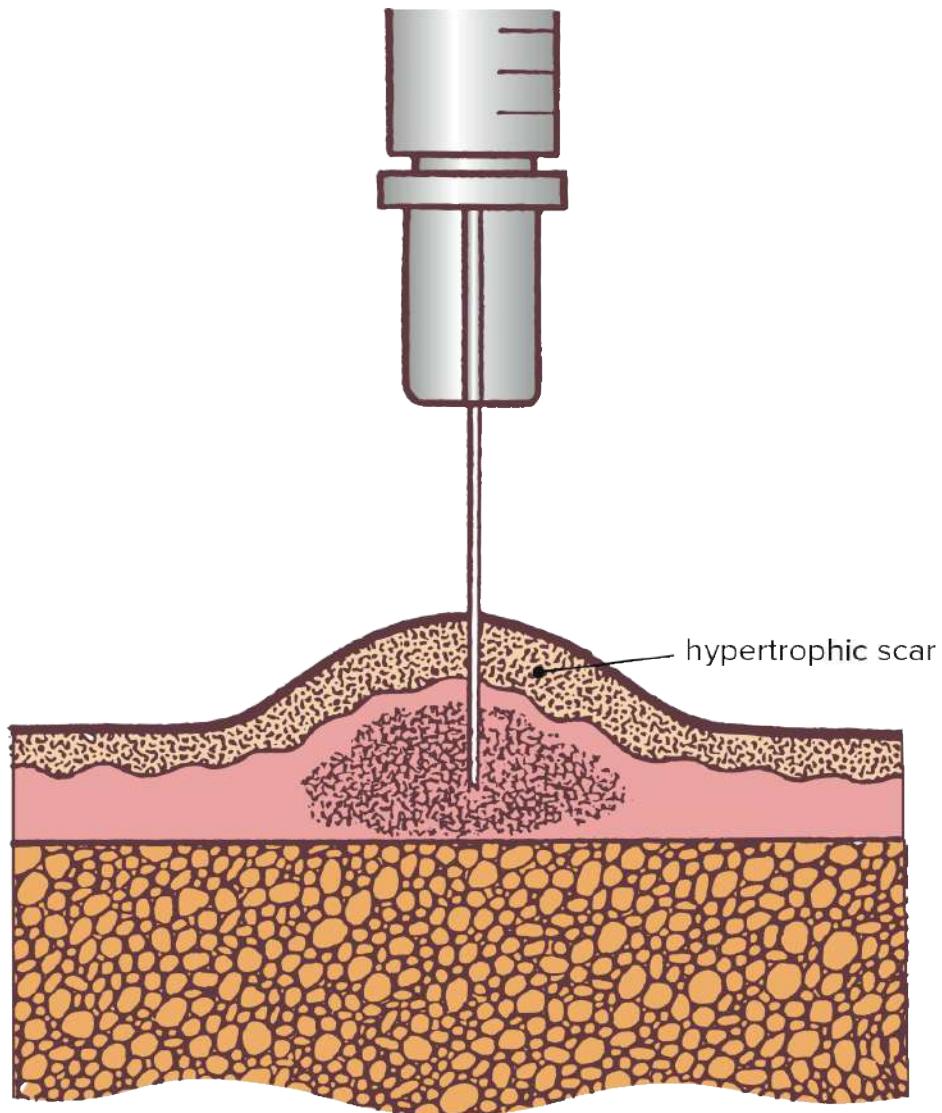
- plaque psoriasis
- granuloma annulare
- hypertrophic scars
- keloid scars (early development)
- alopecia areata
- chondrodermatitis nodularis helicis

- lichen simplex chronicus
- necrobiosis lipoidica
- hypertrophic lichen planus
- orf and milker's nodules

Triamcinolone is the appropriate long-acting corticosteroid (10 mg/mL). It may be diluted in equal quantities with saline.

## Method

1. The steroid should be injected into the lesion (not below it).
2. Insert a 25 or (preferably) 27 gauge needle, firmly locked to a small insulin-type 1 mL syringe, into the lesion at the level of the middle of the dermis (see FIG. 116.29 ).
3. High pressure is required with some lesions (e.g. keloid).
4. Inject sufficient steroid to make the lesion blanch.
5. Several sites will be needed for larger lesions, so preceding LA may be required in some instances. Avoid infiltration of steroid in larger lesions: use multiple injections.

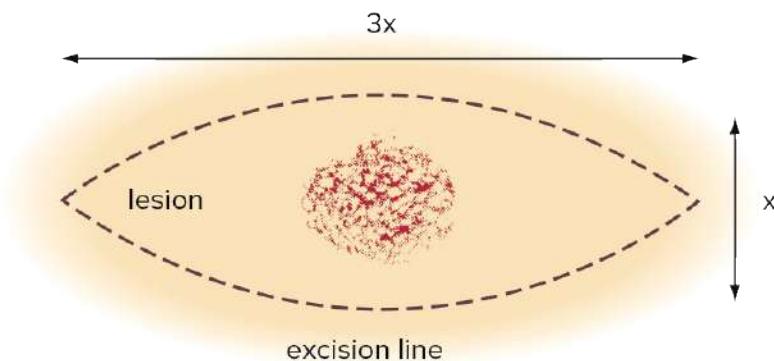


**FIGURE 116.29** Injection of corticosteroid into mid-dermis

## Elliptical excisions

Small lesions are best excised as an ellipse. Generally, the long axis of the ellipse should be along the skin tension lines identified by natural wrinkles.

The intended ellipse should be drawn on the skin (see FIG. 116.30 ). The placement will depend on such factors as the size and shape of the lesion, the margin required (usually 3 mm) and the skin tension lines.



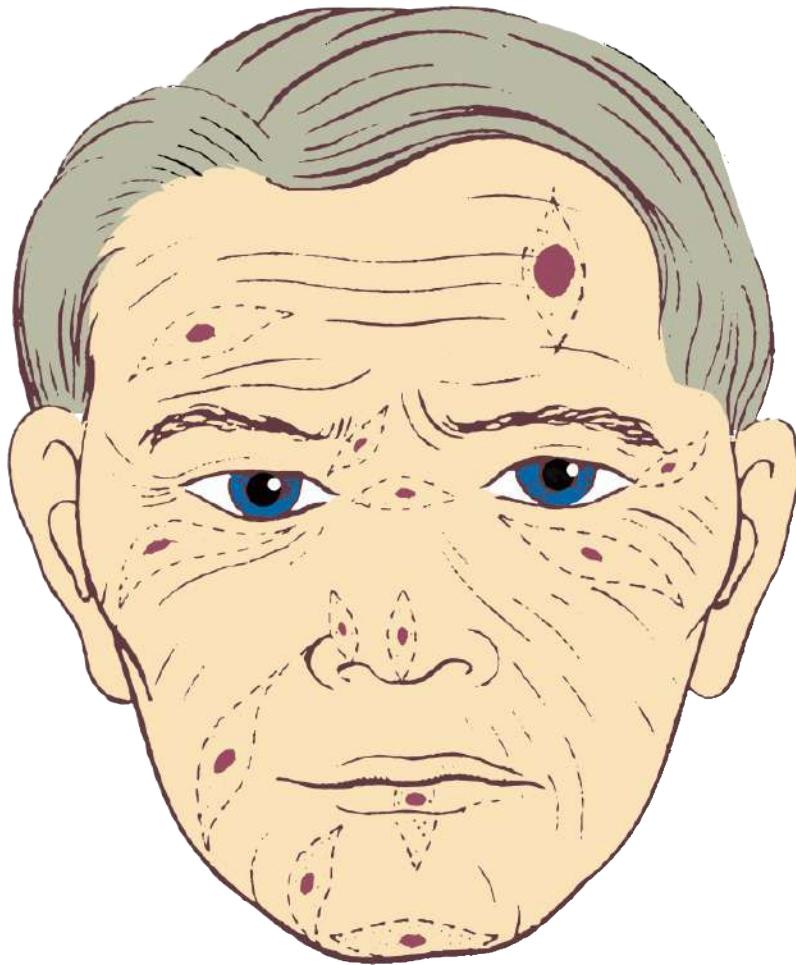
**FIGURE 116.30** Ellipse excision

### General points<sup>13</sup>

- The length of the ellipse should be three times the width.
- This length should be increased (say, to four times) in areas with little subcutaneous tissue (dorsum of hand) and high skin tension (upper back).
- A good rule is to obtain an angle at each end of the excision of 30° or less.
- These rules should achieve closure without ‘dog ears’.

### Excisions on the face

It is important to select optimal sites for elliptical excisions of tumours of the face. As a rule it is best for incisions to follow wrinkle lines and the direction of hair follicles in the beard area. Therefore, follow the natural wrinkles in the glabella area, the ‘crow’s feet’ around the eye and the nasolabial folds (see FIG. 116.31 ). To determine non-obvious wrinkles, gently compress the relaxed skin in different directions to demonstrate the lines.



**FIGURE 116.31** Recommended lines for excisions on face

Source: Adapted from JS Brown, *Minor Surgery: A Text and Atlas*. London: Chapman & Hall, 1986

For tumours under 1 cm on the forehead, make horizontal incisions. Vertical incisions may be used for large tumours of the forehead. Ensure that you keep your incisions in the temporal area quite superficial, as the frontal branch of the facial nerve is easily cut.

## When to refer<sup>13</sup>

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Referral should be considered for:

- uncertainty of diagnosis
- suspicion of melanoma
- tumours larger than 1 cm

- recurrent tumours, despite treatment
- incompletely excised tumours, especially with poor healing
- doubts about appropriate treatment
- recommended treatment beyond skills of practitioner
- frequent multiple tumours (e.g. organ transplant patients)
- SCC on the lip or ear
- infiltrating or scar-like morphoeic BCC, particularly those on the nose or around the nasal labial fold
- cosmetic concerns, such as lesions in the upper chest and upper arms where keloid scarring is a potential problem

## Key points

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- Uncomplicated small tumours are best removed by an elliptical excision with a 3 mm margin for BCC and a 4 mm margin for SCC.
- Caution should be used in the management of tumours on the face, including the ears and lips.

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## Patient education resources

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

- Fatty tumour (lipoma)
- Molluscum contagiosum
- Seborrhoeic keratoses
- Skin cancer
- Warts

## Resource

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Hayes M. *Skin Cancer, Melanoma and Mimics*. Brisbane: Skin Cancer Books, 2013.

## References

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- 1 Paver R. The surgical management of cutaneous tumours in general practice. Modern Medicine Australia, 1991; March: 43–51.
- 2 Davis A, Bolin T, Ham J. *Symptom Analysis and Physical Diagnosis* (2nd edn). Sydney: Pergamon Press, 1990: 302–6.
- 3 Huang Q, Veness M, Richards S. The role of adjuvant radiotherapy in recurrent keloids. Australas J Dermatol, 2004; 45: 162–6.
- 4 Berger P. Warts: how to treat them successfully. Modern Medicine Australia, 1990; August: 28–32.
- 5 de Launey WE, Land WA. *Principles and Practice of Dermatology* (2nd edn). Sydney: Butterworths, 1984: 280–1.
- 6 Reddy J. Intralesional injection for orf: a practice tip. Aust Fam Physician, 1993; 22: 65.
- 7 Erbağci Z, Erbağci İ, Tuncel A. Rapid improvement of human orf (*ecthyma contagiosum*) with topical imiquimod cream: report of four complicated cases. Journal of dermatological treatment, 2005; 16(5–6): 353–6.
- 8 Miscellaneous skin conditions [published 2015]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2015. [www.tg.org.au](http://www.tg.org.au), accessed April 2021.
- 9 Marks R. Skin cancer. In: *MIMS Disease Index* (2nd edn). Sydney: IMS Publishing, 1996: 469–72.
- 10 Staples MP et al. Non-melanoma skin cancer in Australia: the 2002 national survey and trends since 1985. Med J Aust, 2 January 2006; 184(1): 6–10.
- 11 Solar damage and skin cancer [published 2015]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2015. [www.tg.org.au](http://www.tg.org.au), accessed April 2021.
- 12 Telfer NR, Colver GB, Bowers PW. Guidelines for the management of basal cell carcinoma. BJ Dermatology, 2008; 159: 35.
- 13 Sinclair R. Skin cancer and benign lesions. Australian Doctor, 7 Sept 2012: 25–32.
- 14 Wolff K, Johnson RA. *Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology* (5th edn). New York: McGraw-Hill, 2006: 536–8.
- 15 Murrell DF. Pseudoaneurysm. Medical Observer, 11 July 2008: 38.
- 16 La Villa G. Methylprednisolone acetate in local therapy of ganglion. Clinical Therapeutics, 1986; 47: 455–7.

# 117 Pigmented skin lesions

*The skin calls for the faculty of close observation and attention to detail.*

LOUIS A DUHRING (1845–1913), VALEDICTORY ADDRESS, UNIVERSITY OF PENNSYLVANIA MEDICAL SCHOOL

The management of pigmented skin lesions is a constant concern for all practitioners and requires careful evaluation based on the natural history of these lesions and the increasing incidence of malignant melanoma in particular.

Most pigmented lesions are benign and include simple moles or melanocytic naevi, seborrhoeic keratoses, freckles and lentigines. Reassurance is all that is necessary in the management of these problems.

However, one-third of all melanomas arise in pre-existing naevi, many of which are dysplastic, and it is the recognition and removal of such naevi that is so important in the prevention of melanoma.<sup>1</sup>

Malignant melanoma is doubling in incidence each decade, which is an alarming statistic considering the public education programs about the hazards of sun exposure. Of equal interest is the fact that the cure rate for melanoma is also increasing, reflecting earlier diagnosis and treatment. The most important factor in management is early detection. It is most appropriate for GPs to acquire skills in dermoscopy, which significantly improves diagnostic accuracy for melanoma.<sup>2</sup>

A classification of pigmented skin lesions is given in TABLE 117.1 .

**Table 117.1** Classification of pigmented skin lesions

## Non-melanocytic

Pigmented basal cell carcinoma

Seborrhoeic keratoses

Talon noir (black heel)

## Tinea nigra

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### Melanocytic

Non-melanoma

Freckles

Lentigines

Naevi:

1. congenital
2. acquired:

junctional → compound → intradermal  
halo  
blue  
Spitz  
dysplastic

Melanoma:

1. Lentigo maligna (Hutchinson melanotic freckle)
  2. Superficial spreading melanoma
  3. Nodular melanoma
  4. Acral lentiginous melanoma
- 

## Key facts and checkpoints

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- The incidence of melanoma is greatest in white Caucasians and increases with proximity to the equator.
- The early diagnosis and treatment of melanoma profoundly affects the prognosis.
- Melanoma is extremely rare before puberty.
- Currently the greatest rate of increase is in men >55 years.
- Most people have 5–10 melanocytic naevi on average.
- Multiple dysplastic naevi carry a higher risk of malignant change, which may occur in young adults. Such patients require regular observation (with photography).

## ⌚ Talon noir ('black heel')

Talon noir is a black spotted appearance on the heel and is common in sportspeople. A similar lesion (probably smaller) is often found on the other heel.

'Black heel' is formed by small petechiae caused by the trauma of the sharp turns required in sport: shearing stress on the skin of the heel produces superficial bleeding. The diagnosis can be confirmed by gentle paring of the callus to reveal the multiple small petechial spots in the epidermis; these are then pared away. If there is doubt about the diagnosis (malignant melanoma is the main differential diagnosis), the lesion should be excised.

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## **Tinea nigra**

Tinea nigra is characterised by solitary black macular lesions on the palm or sole. The simple technique of taking skin scrapings to reveal fungal elements will allow easy differentiation from malignant melanoma.

## **Freckles**

Freckles are small, brown flat macules (usually <0.5 cm), coloured by excessive epidermal melanin without any increase in the number of naevus cells (melanocytes). They occur mainly on light-coloured skin and tend to darken in summer and fade in winter. Cosmetic improvement can be achieved through the use of sunscreens.

## **Melasma (chloasma)**

Melasma (see [CHAPTER 8](#), [FIGURE 8.3](#)) is facial pigmentation commonly seen in people with darker skin and women in a high oestrogen phase, such as during pregnancy and oestrogen therapy (e.g. OCP). These people must seek sun protection and withdraw the therapy, if possible. A first-line topical depigmentation agent is hydroquinone 2% cream applied twice daily for 2–4 months.

## **Lentigines**

Lentigines are small, rounded, brown to black macular areas ranging from 1 mm to 1 cm or more across. They are very common and may appear in childhood as a few scattered lesions, often on areas not exposed to the sun. In the elderly, lentigines often develop on sun-damaged skin, usually on the backs of the hands (so-called 'liver spots') and on the face.

Unlike freckles, they have increased numbers of melanocytes.

## **Management**

- Treatment is usually unnecessary. Liquid nitrogen or excision can be used for cosmetically unacceptable lesions. Sunscreens are needed to prevent further darkening of existing lesions.

- Apply tretinoin 0.05% cream daily at night, if necessary.
- Laser for severe cases.

## **Congenital melanocytic naevi**

These moles are present at birth and are sometimes large. Infants with giant naevi should be referred to an expert for management advice.

### **Clinical features**

- Variable colour—brown to black
- Sometimes hairy and protruding
- Increased risk of malignant change (especially in larger ones)

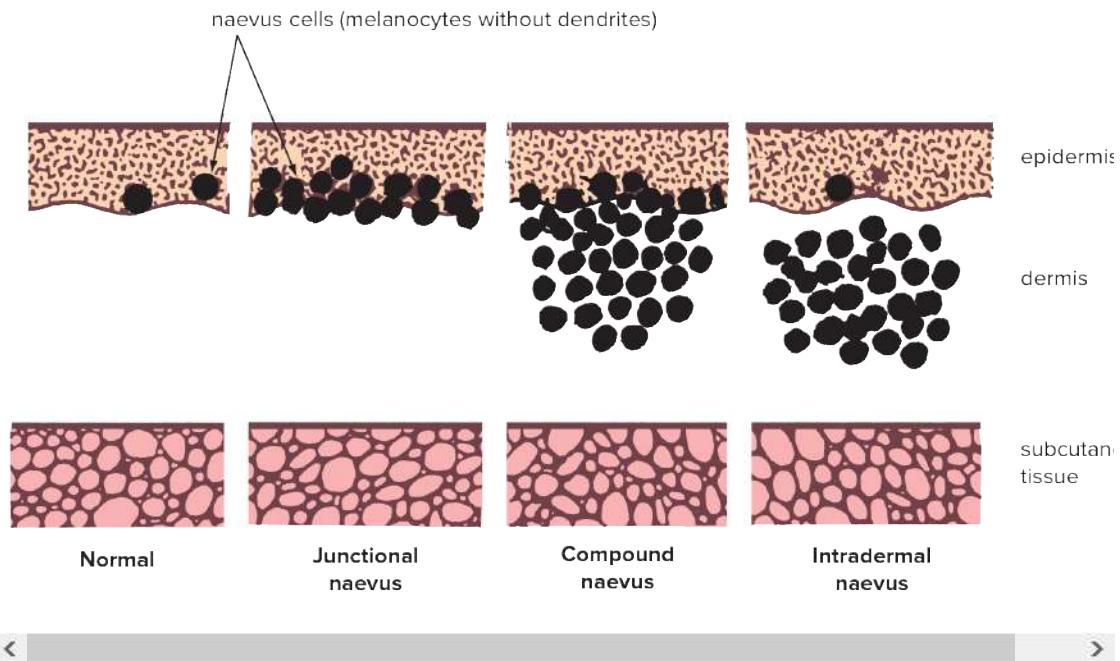
## **Becker naevus**

Becker naevus is a faint, brown, diffuse pigmented area with a component of coarse hairs and is usually found on the shoulder and upper trunk. It occurs mainly in boys around puberty. It is a late-onset epidermal naevus or birthmark, it is benign and reassurance is appropriate.

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## **Common acquired naevi**

These are the common moles for which an opinion is so often sought. The moles are localised, benign proliferations of naevus cells. There may be a sharp increase in numbers during pregnancy. New lesions appear less frequently after the age of 20 years. The types are junctional, compound and intradermal. Naevi in children are usually the junctional type with proliferating naevus cells clumped at the dermo-epidermal junction. With time the naevus cells ‘move’ into the dermis. A compound naevus has both junctional and dermal elements. With maturation all the naevus cells move into the dermis. Refer to [FIGURE 117.1](#).



**FIGURE 117.1** Comparison of melanocyte (naevus cell) distribution in various common acquired naevi

## Clinical features

### Junctional

- Usually <5 mm
- Circular-shaped macules
- May be slightly elevated
- Colour usually brown to black
- May be ‘fuzzy’ border

Most naevi of the palms, soles and genitals are junctional but there is no evidence to support the traditional view that naevi in these sites have more malignant potential.<sup>2</sup>

### Compound

- Dome-shaped, slightly raised pigmented nodules
- Up to 1 cm in diameter
- Colour varies from light to dark brown/black, but lighter than junctional naevi (see FIG. 117.2 )

- Most are smooth but surface can be rough or verrucoid
- Larger ones may be hairy, especially after puberty
- Become ‘flesh’-coloured in time



**FIGURE 117.2** Compound naevus

*Source:* Usatine 2019

### Intradermal

- Look like compound but less pigmented
- Often skin-coloured
- May evolve to pink or brown senile nodules or to soft, pedunculated tags

### Malignant potential of common acquired melanocytic naevi

- *Junctional:* have significant potential to undergo malignant change (as long as junctional activity is present)
- *Compound:* very rarely undergo malignant change
- *Intradermal:* these are totally benign lesions

### Management

- Provide appropriate reassurance.

- Observe.
- If lesion is changing or there is uncertainty, perform surgical excision (2 mm margin) for histopathology.

## **Halo naevus**

A halo naevus consists of a depigmented halo around a central melanocytic naevus (see FIG. 117.3). It is the result of an autoimmune reaction. The central naevus gradually involutes. It tends to occur around puberty. Multiple halo naevi are often seen on the trunk of adolescents.



**FIGURE 117.3** Halo naevus in a child. The central lesion is usually a benign pigmented naevus.

*Photo courtesy Robin Marks*

*Caution:* A halo can occur around a melanoma.

## **Management**

Measure the lesion. Reassure and do nothing, as it usually disappears over the next few years; if doubtful at all, remove and obtain histological diagnosis.

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## **Blue naevus**

A blue naevus presents as a solitary, slate-grey-blue dermal lesion. Blue naevi usually present in childhood and adolescence on the lower back and buttocks and the limbs, especially dorsa of the hands and feet. Malignant change is rare. They are often excised for cosmetic reasons.

## ⌚ Spitz naevus (benign juvenile melanoma)

Spitz naevi are also called benign juvenile melanomas or spindle cell naevi.

### Clinical features

- Solitary pigmented or erythematous nodules
- Often appear in children, usually 4–8 years
- Develop over 1–3 months
- Well-circumscribed, dome-shaped lesions

### Management

Surgical excision is treatment of choice (because of rapid growth and best ‘reassurance’ policy).

## ⌚ Dysplastic melanocytic naevi

These are large, irregular moles that appear predominantly on the trunk in young adults (see FIG. 117.4 ). They can be familial or sporadic and are markers of an increased risk of melanoma, rather than necessarily being premalignant lesions. Even so, melanoma may arise within these lesions more frequently than would be expected by random chance.<sup>3</sup>



**FIGURE 117.4** Dysplastic melanocytic naevi. These lesions may have ill-defined borders and irregular pigmentation.

*Photo courtesy Robin Marks*

They are considered to be intermediate between benign naevi and melanoma.

## Clinical features

- Age: adolescence onwards
- Large >5 mm (variable size)
- Most common on trunk
- Irregular and ill-defined border
- Irregular pigmentation
- Background redness
- Variable colours—brown, tan, black, pink, red
- Variation of colours within the naevus
- Most are stable and do not progress to melanoma

## Dysplastic naevus syndrome

The presence of multiple, large, irregular pigmented naevi, mainly on the trunk, presents a difficult management problem, especially if there is a family history of melanoma. The lifetime risk of melanoma may approach 100% for such patients.

## Management

Use a follow-up program (similar to excised early melanoma) of 6-monthly review for 2 years (3 monthly if family history of melanoma) and yearly thereafter, provided no lesions become malignant during the first 2 years. During this time the patient and family should become well versed in surveillance. Apart from measurement, good professional-quality photographs of areas of the body including total body photography or specific lesions of concern may also be helpful.

Any suspicious lesions should be excised for histological examination.

## Advice to patients

To decrease your chances of getting a melanoma, you should protect yourself from the sun. These rules should be followed.

- Try to avoid direct sunlight when the sun is at its strongest (from 10 am to 3 pm).
- Always wear a broad-brimmed hat and long-sleeved shirt in the sun.
- Use an SPF 50 or more sunscreen on exposed skin and renew the sunscreen regularly.

- Sunbaking might give you a good tan but it is also going to increase your chances of getting a melanoma, so you should avoid it.

## Melanoma

Melanomas are malignant tumours derived from melanocytes, with the skin being the most common site.

The early diagnosis of melanoma is vital to outcome. Thickness of a melanoma when it is removed is the major factor determining prognosis: it is vital to detect melanomas when they are in the thin stage and look like an unusual freckle.

In Australia, only about 30% of melanomas develop in pre-existing melanocytic naevi [Page 1312](#) (moles).<sup>2,3</sup> The majority arise in apparently normal skin. Initially the tumour tends to spread laterally in many cases and it should be removed at this stage when it is easily cured. An irregular border or margin is suggestive of the tumour.

### Risk factors

- History of previous melanoma (10-fold)
- More than 5 atypical/dysplastic naevi (sixfold)
- Presence of many moles (50 or more)
- Family history (one or more members, especially first-degree relatives)
- History of many blistering sunburns as a child or adolescent
- Sun-sensitive skin/fair complexion
- Patient age and sex: increasing age and male
- Tanning (including solarium) treatments
- History of multiple non-melanoma skin cancers
- Marked solar skin damage
- Immunodeficiency

### Skin surveillance for high-risk patients<sup>4</sup>

- Educate patients how to perform skin self-examination and recognise suspicious lesions
- Emphasise sun-smart behaviour
- Full skin examination is recommended every 6 to 12 months by the patient's preferred health professional
- Use of dermoscopy is advised, which has been shown to significantly reduce the ratio of

- benign to malignant lesions excised
- Consider the use of total body photography

## Clinical features

- Typical age range 30–50 years (average 40 years)
- Can occur anywhere on the body—more common:  
lower limbs in women, upper back in men
- Often asymptomatic
- Can bleed or itch

## Warning signs

The sign of major importance is a recent change in a ‘freckle’ or mole:

- change in size—at edge or thickening
- change in shape
- change in colour—brown, blue, black, red, violet, white, including combinations
- change in surface
- change in the border
- bleeding or ulceration
- other symptoms (e.g. itching)
- development of satellite nodules
- lymph node involvement

### Red flag pointers for melanoma<sup>5</sup>

- New or changing lesion (see preceding change list)
- Rapidly growing nodule of any colour
- Non-healing lump or ulcer
- The ‘ugly duckling’ syndrome: a prominent pigment lesion that stands out from any other

- A lesion that concerns the patient
- Dermoscopic changes on follow-up or poor dermoscopic–clinical correlation, e.g. asymmetric pigmentation, radial streaming

## Types

### Lentigo maligna (LM) and lentigo maligna melanoma (LMM)<sup>3</sup>

Lentigo maligna (LM or Hutchinson melanotic freckle) is an *in situ* melanoma that presents as a slow-growing, irregular brown/black macule on areas exposed to light (usually the face), predominantly in older patients (see FIG. 117.5). They have all the variations in size, shape and colour of superficial melanomas. Dermoscopic features include an annular-granular pattern, asymmetric follicular pigmented openings and rhomboidal structures.<sup>5</sup> Lentigo maligna should be excised.

If left untreated, LM can develop into invasive melanoma called lentigo maligna melanoma (LMM). LMM is reported to develop in 3–10% cases of LM.<sup>6</sup> Prognosis is similar to that for other invasive melanomas.



**FIGURE 117.5** Lentigo maligna in a 72-year-old man. Excision is recommended as it is a form of intra-epidermal melanoma.

*Photo courtesy Robin Marks*

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### Superficial spreading melanoma

Like lentigo maligna, the initial growth is in a lateral or radial intra-epidermal manner, rather

than in an invasive downward or vertical manner (see FIG. 117.6 ). It exhibits a striking colour variation. Through a dermatoscope there is typically a range of colours with architectural disorder, radial streaming, branched streaks, pseudopods, peripheral black dots and blue-white structures.<sup>5</sup> It accounts for 70% of melanomas. It can be detected early by biopsy—shave is preferred to punch (less sampling error). Excisional biopsy is preferable.



**FIGURE 117.6** Superficial spreading melanoma with an irregular border which has altered and variable colours. Requires excision.

*Photo courtesy Robin Marks*

### Nodular melanoma

Nodular melanomas account for 20% of melanomas, are aggressive and invasive, and have no radial growth phase. They are typically found on the trunk and limbs of young to middle-aged individuals (see FIG. 117.7 ). It may have a ‘blueberry’-like nodule. Prognosis is determined by thickness at the time of excision. Dermatoscopy is usually less useful.



**FIGURE 117.7** Nodular melanoma on the back. It has no radial growth phase and because it grows vertically can be readily misdiagnosed. The ABCDE rule often does not apply but this lesion shows variable colours and an irregular border.

*Photo courtesy Robin Marks*

### The early nodular melanoma problem<sup>5,7</sup>

Nodular melanoma can present a diagnostic dilemma since the ABCDE rule (see later in this chapter) often does not apply. Early melanomas tend to be symmetrical, non-pigmented, even in colour, of small diameter and to grow vertically.

They are often mistaken for a haemangioma or a pyogenic granuloma. If one is suspicious, refer early to a specialist/specialist clinic.

### Acral lentiginous melanoma

These typically occur on palms, soles and distal phalanges (see FIG. 117.8). They have a poorer prognosis than other types. They occur mainly in people with dark skin.



**FIGURE 117.8** Acral lentiginous melanoma. A 30-year-old man presented with a 'mole' on his toe that had become 'lumpier'. This type of melanoma, which occurs on the distal areas of the limbs, begins as a spreading pigmented macule before developing into a nodule surrounded by a pigmented halo (as shown).

*Photo courtesy Robin Marks*

### Desmoplastic melanoma<sup>5</sup>

This is a rare and aggressive subtype of melanoma. They are often subtle clinically and sometimes scar-like. Most are non-pigmented.

### Variations

Amelanotic melanomas are flesh-coloured papules that increase in size or change shape. These lesions can be extremely difficult to diagnose and the poor prognosis associated with them is due to late diagnosis rather than an increased malignancy.

The features and associations of melanoma subtypes are presented in TABLE 117.2 .

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**Table 117.2** Features and associations of melanoma subtypes

Melanoma subtype	Frequency %	Radial growth phase	Location	Average age	Occupation profile
Superficial	70	+	Trunk (back), limbs	Middle-	Indoor

spreading			limbs (legs)	aged	worker
Nodular	20	-	Trunk, limbs	Middle-aged	Indoor worker
Lentigo maligna melanoma	7.5	+	Head, neck	Elderly	Outdoor worker
Acral lentiginous	2.5	+	Palms, soles mucosae	Not known	Not known

Courtesy of J Kelly<sup>7,8</sup>

## Prognosis

The prognosis of melanoma mainly depends on the depth of the tumour in mm at the time of excision (Breslow thickness). The chance of cure is greater than 90% if a melanoma is removed when it is less than 0.75 mm thick.<sup>3</sup> If the lesion has invaded to a thickness of 4 mm or more, the likelihood of a cure is reduced to less than 30%.<sup>3</sup>

Sentinel lymph node (SLN) biopsy should be considered for all patients with melanoma greater than 1 mm in thickness and for patients with melanoma greater than 0.75 mm with other high-risk factors.

Other determinants of prognosis include:<sup>3</sup>

- site (worse on head and neck, trunk)
- sex (worse for men)
- age (worse >50 years)
- amelanotic melanoma
- ulceration

## Differential diagnosis

There are several common skin lesions that may be mistaken for melanoma.<sup>1</sup> They are:

- haemangioma (thrombosed)
- dermatofibroma (sclerosing haemangioma)
- pigmented seborrhoeic keratosis
- pigmented BCC

- junctional and compound naevi
- blue naevi
- dysplastic naevi
- lentigines

## Facilitating early diagnosis of melanoma

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An adequate light source without shadows is essential. Refer to [CHAPTER 111](#) for use of the ‘Maggylamp’ and the dermatoscope, which is a very important and useful adjunct to diagnosis.

### Clinical examination of the skin<sup>1</sup>

It is important to examine the entire skin and not just the lesion presented by the patient. Comparison of pigmented skin lesions is very helpful in differentiating between benign and malignant. One satisfactory routine is:

- Starting at the head, examine the hairline, backs of ears, neck, back and backs of the arms. Pull down the underwear to expose the buttocks; examine the backs of the legs.
- With the patient facing you, examine the anterior hairline, the front of the ears, the forehead, cheeks and neck, moving downwards to the anterior chest. Move bra straps as required to achieve complete coverage. Then examine the abdomen, pulling down the underwear to examine as far as the pubic hairs.
- Then examine the anterior surfaces of the legs. The ‘Maggylamp’ is very useful for this examination.

After scanning the entire skin surface and comparing and contrasting naevi, specific lesions may be examined with the dermatoscope. Compare suspicious lesions with similar lesions elsewhere on the patient’s skin.

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### Applying the ABCDE system<sup>1</sup>

#### **A = Asymmetry**

Melanoma is almost always asymmetrical. Most non-melanoma lesions are symmetrical, oval or round.

#### **B = Border**

The border of the melanoma is usually well defined, especially in the more malignant, compared with the dysplastic naevus, which is almost always indistinct with a fading-out ‘shoulder’ effect.

The border of the melanoma is irregular while most benign lesions have a regular edge.

**C = Colour**

The classic blue–black colour is helpful but the *variety* of colours present in most melanomas is the most helpful. Magnification usually visualises greys, whites, violets, reds, oranges and shades of brown interspersed in the darker blue–black pigmentation. Early melanomas developing in dysplastic naevi tend not to have this deep pigmentation.

**D = Diameter**

The majority of melanomas when first seen are at least 7 mm in diameter, especially if arising from a pre-existing naevus. However, it is possible to diagnose small nodular melanomas <5 mm.

**E = Evolution and/or Elevation**

A melanocytic naevus is usually stable, whereas a melanoma evolves/changes over time. Elevation indicates invasion and is a sign of more advanced disease and a flat lesion may become raised.

**Practice tip**

Not all melanomas are black and black may not be present in some melanomas.

## Diagnosis by exclusion

In the diagnostic process consider the lesions outlined in the differential diagnosis and check out the various characteristics. Haemangiomas may have an emptying sign when pressed with a finger. Pigmented BCCs can be difficult if they are fully pigmented but this is uncommon. The characteristic pearly-grey look and the telangiectasia are usually still visible on magnification with the ‘Maggylamp’. The most useful feature of dysplastic naevi is that they are usually multiple and lesions for comparison can generally be found elsewhere. Dysplastic naevi also have greater breadth and height and often a darker nodule in the centre—the ‘target’ sign. A small but significant number of melanomas are undiagnosable.

## Pitfalls in diagnosis of melanoma

- Nodular melanoma
- Small melanoma
- Amelanotic melanoma
- Regressing melanoma

- Rapidly growing melanoma

## Management points for naevi and melanomas

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- Do not inject local anaesthetic directly into the lesion.
- Incisional biopsy of a melanoma or suspicious mole is best avoided.
- Accurate clinical diagnosis, with the definitive treatment performed in one stage, is optimal, rather than biopsy with follow-up surgery.

### Management tips<sup>1</sup>

- If a lesion is clinically suspicious for malignancy, ideally completely excise with a margin of 2 mm.<sup>4</sup> A partial biopsy may not represent the lesion as a whole.<sup>5</sup>
- If melanoma is diagnosed or strongly suspected, referral to a consultant is necessary.
- If a malignant lesion is identified and re-excision is required, it may be appropriate to be performed by a specialist team. This is especially the case for melanoma.
- Re-excision should ideally be performed within 4 weeks.<sup>5</sup>
- Beware of the pigmented BCC—it is easily missed but it usually has a shiny surface.
- Do not freeze a pigmented lesion—take a biopsy.

### Guidelines for excision margins<sup>5,9</sup> (Breslow thickness)

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- Suspicious lesion—margin 2 mm
- Melanoma *in situ*—margin 5–10 mm
- Melanoma <1 mm thick—margin 1 cm
- Melanoma 1–4 mm thick, minimum margin 1 cm and maximum 2 cm
- Melanoma >4 mm thick, margin 2 cm (minimum)

### Follow-up

Patients with a melanoma are at risk of further melanomas and non-melanoma skin cancers, and need lifelong surveillance. Follow-up may be shared between specialists and GPs.

A rough guide for surveillance following melanoma:<sup>5</sup>

- 3-monthly skin and lymph node checks for 2 years
- followed by 6-monthly checks for a further 2 years
- yearly checks thereafter (from 4 years)

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### Practice tips<sup>4</sup>

- Excision biopsy with 2 mm margin is appropriate whenever possible.
- Discuss unexpected pathology results with the reporting pathologist.
- For melanoma, re-excision is then required to achieve wider margins based on the Breslow thickness.
- Specialist referral is appropriate if melanoma is diagnosed or suspected.<sup>5</sup>

## References

- 1 McCarthy W. The management of melanoma. *Aust Fam Physician*, 1993; 22: 1177–86.
- 2 Roberts H, Haskett M, Kelly J. Melanoma: clinical features and early diagnostic techniques. *Medicine Today*, May 2006; 7(5): 39–47.
- 3 Marks R. Skin cancer. In: *MIMS Disease Index* (2nd edn). Sydney: IMS Publishing, 1996: 469–72.
- 4 Cancer Council Australia Melanoma Guidelines Working Party. *Clinical practice guidelines for the diagnosis and management of melanoma*. Sydney: Cancer Council Australia. Available from: <https://wiki.cancer.org.au/australia/Guidelines:Melanoma>, accessed April 2021.
- 5 Solar damage and skin cancer [published 2015]. In: *Therapeutic Guidelines* [digital]. Melbourne: Therapeutic Guidelines Limited; 2015. [www.tg.org.au](http://www.tg.org.au), accessed April 2021.
- 6 Lentigo maligna and lentigo maligna melanoma. DermNet NZ, 2011 [online]. Available from: <https://dermnetnz.org/topics/lentigo-maligna-and-lentigo-maligna-melanoma/>, accessed April 2021.
- 7 Kelly J. Nodular melanoma—no longer as simple as ABC. *Aust Fam Physician*, 2003; 32(9): 702–9.

- 8** Kelly J. Malignant melanomas—how many have you missed? *Med J Aust*, 1996; 164: 431–6.
- 9** Sladden MJ et al. Updated evidence-based clinical practice guidelines for the diagnosis and management of melanoma: definitive excision margins for primary cutaneous melanoma. *Med J Aust*, 2018; 208(3): 132–42.

# 118 Hair disorders

*Look it's only death. It's not like losing your hair.*

HAROLD BRODKEY UPON LEARNING HE HAD AIDS, *NEW YORKER MAGAZINE* 1994

The hair of our scalp is referred to as our crowning glory and the threat of hair loss in both sexes provokes extraordinary anxiety bordering on grief in some people. It behoves us as medical practitioners to treat the patient presenting with 'I'm losing my hair' with appropriate support and understanding. Likewise, the problem of hirsutism provokes similar anxiety and concerns about body image. Interestingly, women present with hair loss more than men.

## Key facts and checkpoints

- There are two types of hair: terminal hair, which is coarse and well pigmented, and vellus hair, which is fine, soft and relatively unpigmented.
- Alopecia is a generic term for hair loss.
- Hair loss (alopecia) generates considerable anxiety and the fear of total hair loss should be addressed with the patient and a realistic prognosis given.
- Androgenic alopecia is the most common cause of human hair loss, affecting 50% of men by age 40 and up to 50% of women by age 60.<sup>1</sup> Other common causes are alopecia areata, seborrhoeic dermatitis and tinea capitis (see TABLE 118.1 ).
- In acute telogen effluvium, the traumatic event has preceded the hair loss by about 2 months (peak loss at 4 months). White bulbs are diagnostic.
- Although severe stress could precipitate alopecia areata, day-to-day stressors are not considered to be a trigger. Stress seems to be a consequence of alopecia rather than the cause of it.<sup>2</sup>
- Hair loss can be patchy or diffuse where it involves the entire scalp.

- Patchy loss—alopecia areata and trichotillomania.
- Generalised loss—telogen effluvium, systemic disease, drugs (see TABLE 118.2 ).
- Alopecia areata has a poor prognosis if it begins in childhood, if there are several patches and there is loss of eyebrows or eyelashes.
- Scarring alopecia can be an indicator of lupus erythematosus or lichen planus.

**Table 118.1** Hair loss: diagnostic strategy model

### Probability diagnosis

Androgenetic alopecia (male pattern baldness)  
 Alopecia areata (diffuse type)  
 Telogen effluvium (incl. postpartum)  
 Anagen effluvium (esp. cytotoxic therapy)  
 Lichen  
 Scars (e.g. trauma, burns)

### Serious disorders not to be missed

Infection:

- tinea capitis/kerion
- bacterial folliculitis
- secondary syphilis
- post-febrile state
- cutaneous leishmaniasis

Cancer:

- treatment for cancer

Other:

- systemic disease (e.g. lupus)
- scarring alopecia (e.g. lichen planopilaris)

### Pitfalls (often missed)

Rarities:

- heavy metal poisoning

Nutritional:

- severe dieting
- malnutrition

- zinc/iron deficiency

### Seven masquerades checklist

Drugs (cytotoxics, anticoagulants, anti-epileptics, amphetamines, anti-thyroid agents, various hormones, cessation OCP)

Thyroid/other endocrine (hypothyroidism)

### Is the patient trying to tell me something?

Emotional stress → telogen effluvium; trichotillomania

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**Table 118.2** Causes of diffuse hair loss

Androgenetic alopecia

Telogen effluvium

Postpartum telogen effluvium

Alopecia areata (diffuse type)

Drugs—cytotoxics and others

Hypothyroidism

Nutritional:

- iron deficiency
- severe dieting
- zinc deficiency
- malnutrition

Post-febrile state

Anagen effluvium

## The normal hair growth cycle

An understanding of the process of normal hair growth is necessary to comprehend and evaluate hair disorders. Each follicle progresses quite independently through regular cycles of growth and shedding (see FIG. 118.1 ).