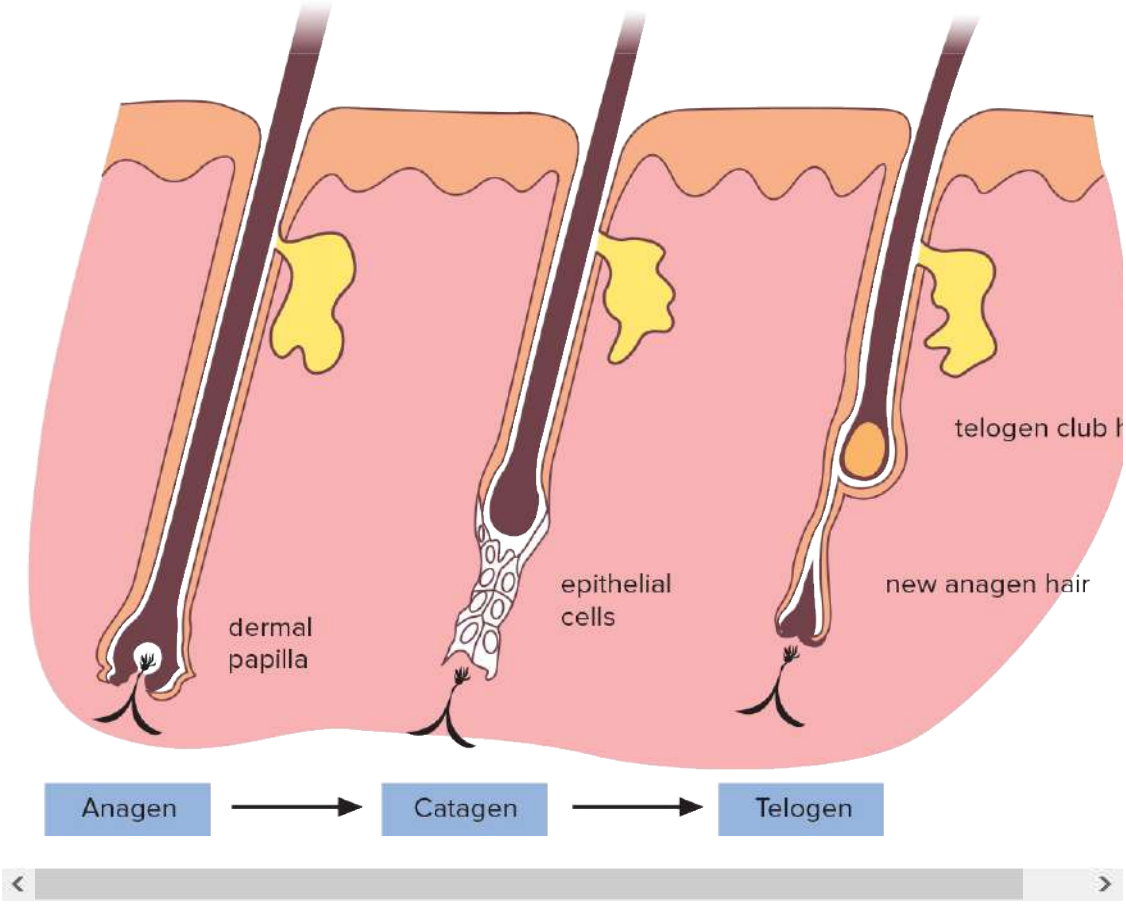

FIGURE 118.1 The normal hair cycle

The three phases of follicular activity are:[3]

1. Anagen phase—the active growth phase of hair production.
    - The dermal papilla stimulates division of epithelial cells that produce the hair shaft.
    - The hair shaft grows 1 cm per month.
    - It lasts for about 2.5–5 years on the scalp (average 1000 days).
    - It lasts 1–2 months on eyebrows and eyelashes and 6–9 months in the axilla and pubis.
    - It varies between individuals.
2. Catagen phase—a short transition phase from active growth to inactivity (the involutional stage).
    - The base of the hair becomes club-shaped.
    - It lasts for only about 2 weeks.

3. Telogen phase—the resting (dormant) phase of the cycle at the end of which the club hair with its non-pigmented bulb is shed.
    - This lasts 2–4 months (average 100 days).
    - The percentage of hairs in telogen varies from site to site—10% in scalp to 60–80% in pubic hair.[1]
    - The hair is anchored in the follicle but does not grow longer.
    - The follicle then re-enters anagen.

Thus every 3–5 years every hair on the scalp is shed and replaced.

Some facts on hair numbers[3]

- Hair growth is asynchronous (i.e. continuous production and shedding).
- Humans produce 1 km of hair a month.
- About 50–100 hairs are shed daily without a reduction in density.
- The scalp contains, on average, 100 000 hair follicles.
- The hair follicle is subject to melanocytic activity.
- At least 25% of hair must be shed before a noticeable loss of density occurs.
- Hair loss counts consistently above 100 per day indicate excessive hair loss.
- Significant hair loss tends to block the shower drain or be visible all over the pillow.

Key history

- Onset, duration, quantity and rate of loss
- Localised or generalised loss
- Associated symptoms (e.g. pruritus, scaling, pustules)
- Systems review including fever, acute illness, surgery, stressors
- Symptoms of hyperandrogenism in women (e.g. hirsutism, acne)
- Endocrine features
- Past history including skin disorders, cancer, thyroid disorders

- Family history of hair loss

Key examination

- General review with emphasis on endocrine system and examination of scalp
- Look for exclamation-mark hair, ‘white bulb’ hair, state of bald patch (clean, scaly, scarred or inflamed) and the unusual pattern of trichotillomania

Key investigations

Consider:

- FBE
- TSH
- pituitary hormones (FSH/LH/prolactin)
- iron studies
- zinc
- syphilis serology
- hair pull test
- trichogram
- scalp biopsy
- skin scrapings and hair sample for fungal microscopy and culture

Office procedures

The following procedures can be useful in determining diagnosis and prognosis.

The hair pull test[3]

In this simple method, 50–100 hairs are grasped between thumb and forefinger and gently pulled proximal to distal. This is repeated 6–8 times and should yield a total of 2–5 telogen hairs, which can be analysed. More than eight hairs per pull is abnormal.

Trichogram

Twenty to 50 hairs are extracted by a short, sharp pull with artery forceps. The anagen-to-telogen ratio is calculated. The ratio is decreased in telogen effluvium because of the increased numbers

of hairs in telogen.

Scalp biopsy

This differentiates between scarring and non-scarring alopecia and also between alopecia areata and trichotillomania. Taking two separate 4 mm punch biopsies is ideal.

Light microscopy of hair shafts

Light and/or electron microscopy is indicated if a hair shaft defect is suspected. Skin scrapings and hair samples are taken for fungal microscopy and culture.

Alopecia areata, alopecia totalis and alopecia universalis (patterned alopecia)

Alopecia areata is a disorder of the hair follicle causing a sudden onset of localised or diffuse hair loss. It is thought to be an autoimmune disorder that has a genetic susceptibility (20% have a positive family history). Men and women are affected equally, with the disorder most commonly occurring in the first two decades of life.[4] The hairs are affected during the growth phase, resulting in cessation of anagen.

DxT patch of complete hair loss + clean scalp + exclamation-mark hairs → alopecia areata

Clinical features

- Complete hair loss (small patch or diffuse)
- Pigmented hairs often lost first
- Clean normal scalp
- No or minimal inflammation
- Exclamation-mark hairs, especially around the periphery (see FIG. 118.2)

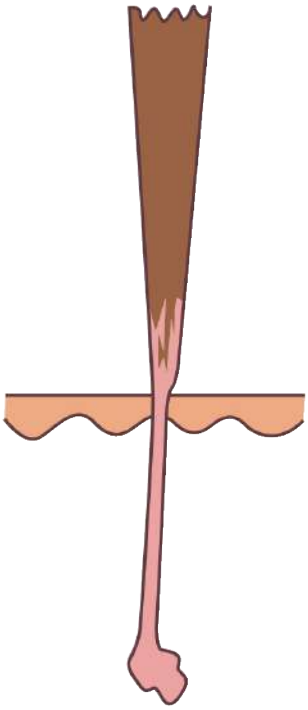

FIGURE 118.2 Exclamation-mark hair (a feature of alopecia areata)

Associations

- Loss of facial (beard in men) or body hair
- Nail changes—dystrophy or pitting

Alopecia areata[2]

An unpredictable pattern of remission and relapse is characteristic. Patches may:

- regrow hair spontaneously (up to 50% within 12 months if a single patch)
- stay the same for many months
- enlarge and coalesce with other patches

The prognosis is poorer with more widespread involvement and younger age of onset (<5 years of age).

Localised patches

Treatment options include:

- topical potent corticosteroid (especially in children), e.g. betamethasone dipropionate 0.05% cream or lotion, once or twice daily for 3–4 months
- intralesional steroids—triamcinolone 10 mg/mL or betamethasone acetate/phosphate 5.7 mg/mL bd (caution needed on face—risk of cutaneous atrophy). Multiple injections required
- topical minoxidil 5% bd (for 4 or more months) only when hair growing

Extensive area (>50% loss)

Treatment options include:

- counselling
- refer to a consultant for expert care
- alopecia areata support group
- use of cosmetic aids (e.g. eyebrow tattoos, topical camouflage spray/powder, wigs, hair pieces, scarves) and camouflage
- topical steroids not recommended—ineffective
- topical immunotherapy or systemic immunosuppresion, including Janus kinase agents, under dermatologist care may help

Alopecia totalis

- This is where the alopecia extends over the total scalp.
- There is, at best, a 50% chance of hair recovery in a fit adult but only a very small chance in childhood.

Alopecia universalis

- This is where the alopecia involves the eyebrows and eyelashes as well. Recovery is rare.

Scarring alopecia[3]

In this condition, the hair follicles are damaged and if the follicular openings cannot be seen with a magnifying lens, regrowth of hair cannot be expected. Thus, the process is irreversible and a scalp biopsy is essential to determine the diagnosis. Apart from obvious causes such as trauma, severe burns, a carbuncle and scalp ringworm with kerion, the causes of scarring alopecia are as follows.

- Lichen planopilaris—this is a variant of lichen planus that produces small follicular papules in the scalp that tend to heal with scarring and destruction of hair follicles; treatment, which is difficult, includes corticosteroids and antimalarials[3]
- Discoid lupus erythematosus—this gives a similar picture to the former and tends to be treated in a similar manner
- Folliculitis decalvans—a chronic folliculitis of the scalp, probably as a response to staphylococci on the scalp; treated with long-term tetracyclines
- Pseudopelade—a slowly progressive, non-inflammatory, scarring condition causing patchy areas of hair loss without any obvious preceding skin disease

Telogen effluvium[5]

Telogen effluvium, which is increased shedding of hairs in the telogen phase, is one of the most common causes of diffuse hair loss and can be triggered by a variety of stressors. It might be acute or chronic hair loss.

It is worth noting that follicular matrix cells have a high metabolic rate second only to haematological tissue, and stress can result in shunting into premature telogen with cessation of anagen.[3]

An obligatory delay occurs between the 'insult' or precipitating event and the onset of hair shedding because the hair follicle cycles through catagen and telogen—approximately 2–3 months when the club hairs with white bulbs of telogen are shed.

Greater than 25% of hair must be lost before there is a perceptible thinning and in this disorder up to 50% loss is common.

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DxT stressful event + 2–3 month gap to diffuse hair loss + 'white bulbs' → telogen effluvium

Patients usually complain of large clumps of hairs with white bulbs coming out with gentle tugging on combing or shampooing (this can exceed 150 hairs per day compared with the normal average of 50–100 hairs).

Stress precipitants of acute telogen effluvium

- Any severe stress
- Childbirth (common)
- High fever

- Weight loss, especially crash dieting
- Trauma—surgical or accidental
- Oral contraceptive pill (OCP) cessation
- Perimenopause
- Malnutrition
- Haemorrhage

Course of telogen effluvium

If uncomplicated, spontaneous recovery can be expected in 6–9 months so reassurance with explanation is usually all that is required. If it persists longer than 6 months, consider the chronic idiopathic form or an unmasked androgenetic alopecia. However, if there is concern about non-recovery and the stress factors are corrected, topical minoxidil 5% lotion (2% if too irritant) twice daily for a minimum of 4 months is an option.[2] Referral to a specialist is advised for relapsing episodes or incomplete recovery.

Chronic telogen effluvium[6]

This occurs usually in perimenopausal and postmenopausal women. It may be primary and idiopathic or secondary to hypothyroidism, hyperthyroidism, malnutrition or cancer. The feature is episodes of dramatic hair shedding that recover but recur weeks to months later and last up to several days. It does not result in obvious balding—it is self-limiting and does not usually need treatment.

Anagen effluvium

This is hair loss during the anagen phase and is typically seen in association with cancer chemotherapy and radiotherapy to the scalp, which results in immediate metabolic arrest. Hair loss is diffuse, involving the whole scalp. Anagen hair shafts are identified by their long and pigmented hair bulb. The follicle may remain in anagen, leading to a quick recovery, or move into telogen, thus delaying growth by about 3 months.

Drug-induced alopecia

Drugs are a very important cause of alopecia (see TABLE 118.3). They may cause telogen effluvium, anagen effluvium or accelerate androgenetic alopecia.

Table 118.3 Drug-induced causes of alopecia generally from prolonged use[1,2]

Cytotoxic agents/thallium
Amphetamines
Anticoagulants: heparin, warfarin
Anti-epileptics: phenytoin, sodium valproate, carbamazepine
Anti-inflammatories: indomethacin, gold, penicillamine, salicylate
Antigout agents: allopurinol, colchicine
Antihelminthic agents: albendazole/mebendazole
Antiparkinson: levodopa, bromocriptine
Anti-thyroid agents/thyroxine/iodine/carbimazole
Cardiovascular agents: • amiodarone • statins, clofibrate • selected ACE inhibitors • selected beta blockers
Cimetidine
Gentamicin
Hormones: OCP, androgens, danazol
Interferon
Lithium
Vitamin A derivatives/retinoids: isotretinoin

Drugs tend to cause telogen effluvium but cancer chemotherapy, radiation to the scalp, thallium/mercury/arsenic and colchicine in high dosage cause anagen effluvium. Acceleration of androgenetic alopecia is caused by hormone therapy, namely the OCP, danazol, testosterone and anabolic steroids.

Androgenetic alopecia (male pattern baldness)

This is the most common form of alopecia, which is age related and is genetically determined in addition to being androgen dependent. The key androgen is dihydrotestosterone, which is produced from testosterone by 5-alpha reductase. The typical pattern of hair loss is recession of the frontotemporal hair with progression to the crown, with some men losing hair quickly and others more slowly. In others the pattern is unpredictable. The typical male pattern is shown in FIGURE 118.3 .

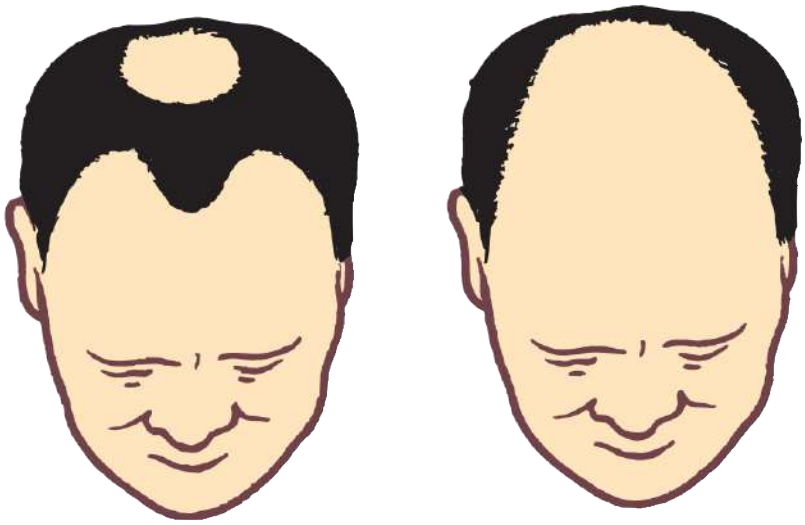

FIGURE 118.3 Typical male pattern baldness

Alopecia affects 30% of men by age 30 and 50% by age 50.

Androgenetic alopecia in women

- In women, the pattern of hair loss is different from men.
- Diffuse thinning occurs, usually on the top of the head (the crown). The front hairline usually remains but in some women this can recede with bitemporal loss (see FIG. 118.4).
- Although hair loss can appear in men and women as early as the 20s, it may not appear before the age of 50 in women.
- Some women notice a short period of considerable hair loss but this may be followed by a long stable period of no loss. Total loss of hair rarely occurs in women.
- It may be unmasked after an episode of diffuse hair loss such as after childbirth or an acute illness.
- Hair loss may be accelerated by conditions associated with androgen excess (e.g. polycystic ovarian syndrome); however, fewer than 5% of women with androgenic alopecia will have elevated serum androgens.[2]

FIGURE 118.4 Typical female pattern baldness

Treatment (men)

It is appropriate to counsel men to accept their balding as part of a natural ageing process. This includes cutting the hair short to make it look better cosmetically. If baldness is not acceptable, some options include wearing a toupee, a wig or other hair substitute or having a hair transplant operation. However, with hair transplantation, the surrounding untreated hair will continue to be lost with time.

Medical treatments are generally expensive and improvements are only sustained for the duration of treatment, so it is important to explore the patient's expectations when discussing treatment options.

Available medications[2]

- Minoxidil 5% foam or lotion, 1 mL applied once or twice daily to the scalp for at least 6–12 months. The results vary from good to no change. Reassure the patient that it is normal for hair shedding to increase in the first 4–6 weeks of minoxidil treatment.
- Finasteride 1 mg tablet taken daily for a minimum of 2 years. There is a risk of sexual dysfunction (e.g. erectile dysfunction, reduced libido) and gynaecomastia (rare), which tend to resolve with time or cessation. Rare reports of persistent sexual dysfunction after stopping finasteride have been published. Mental health problems have also been noted.
- For severe cases, use topical minoxidil and oral finasteride as combination therapy.

Treatment (women)

Physical treatments/hair styling

This includes the use of wigs, hair transplantation and camouflage. Wigs can be worn on the whole head or on the bald spot, or fibres can be interwoven into the remaining hairs.

Camouflage can be used either by having the existing hair bleached by a skilled hairdresser or by colouring the scalp the same colour as the hair. Mascara can be lightly brushed into the roots of the hair at receding hairlines or along gaps.

Medications[1]

minoxidil 5% topical application bd for at least 6–12 months (to assess efficacy) although one large trial has approved its effectiveness generally in women[7]

or

spironolactone 50 to 100 mg daily, increasing to 200 mg daily if no apparent benefit after 6 months; monitor blood pressure, kidney function and liver biochemistry

Spironolactone is contraindicated in pregnancy and side effects include postural hypotension, polyuria and irregular menses. Concurrent use with the combined oral contraceptive pill is appropriate to control menses and for contraception in women of reproductive age.

As for men, these drugs tend to prevent further loss and, if effective, need long-term use.

Trichotillomania (hair pulling)

This is patchy hair loss caused by deliberate plucking or twisting of hair shafts. It is reasonably common in young children, where it may be of little significance, simply being a ‘habit’. In older children and adults, it may be an obsessive–compulsive disorder and psychologist or psychiatrist referral is indicated.[3]

Clinical features

- Incomplete patchy alopecia
- Hairs of different length
- Hairs broken and twisted
- Strange pattern of loss
- Tends to occur on side of dominant hand
- Eyelashes or eyebrows may be involved

Hair disorders in children

Loose anagen (growing hair) syndrome[8]

This is a disorder of the hair follicle characterised by the ability to pluck anagen hairs painlessly

from the scalp by gentle pulling. It presents as very thin, wispy new hair growth. It is an autosomal dominant trait.

Clinical features

- Thin wispy hair with tatty ends
- More common in girls; can affect boys
- Onset in early childhood—usually <5 years
- Large clumps easily pulled out at play
- Light microscopy of hair shafts aids diagnosis

DxT fair females under 5 + thin wispy hair + easy loss with pulling → loose anagen syndrome

Outcome

- Spontaneous improvement with age
- Usually normal hair by teens

Treatment

- Reassurance and explanation
- Gentle hair care

Traction alopecia

Traction alopecia is thinning of the hair seen in female children and young women due to very tight hairstyles, as with ponytails, excessive hair rolling and hair braiding in particular. The bald areas that show short broken hairs and sometimes scarring are found in areas of maximal tug. The most common type is ‘marginal alopecia’ found in the forehead and sides at the hair edges (see FIG. 118.5). Patients should be advised to cease the procedure. Rollers that cause traction can be replaced by those that heat.

FIGURE 118.5 Traction alopecia in an adult from pulling the hair up in a tight bun

Trichotillomania[8]

This occurs in children typically between 4 and 10 years, usually as a nocturnal habit; parents may be unaware of the hair pulling. The affected areas are usually on the anterior and temporal areas of the head (see FIG. 118.6). The areas are never completely bald. A characteristic feature is an irregular-shaped area of incomplete patchy alopecia containing hairs of different length. The variable length is due to the fact that some hairs will not break with pulling while others will break at varying distances from the scalp surface. There may be associated follicular pustules. However, scrapings should be taken to exclude a particular type of tinea capitis (black dot ringworm) caused by *Trichophyton tonsurans.* The management is similar to thumb sucking or nail biting with a low-key approach. It does not imply a significant psychological problem.

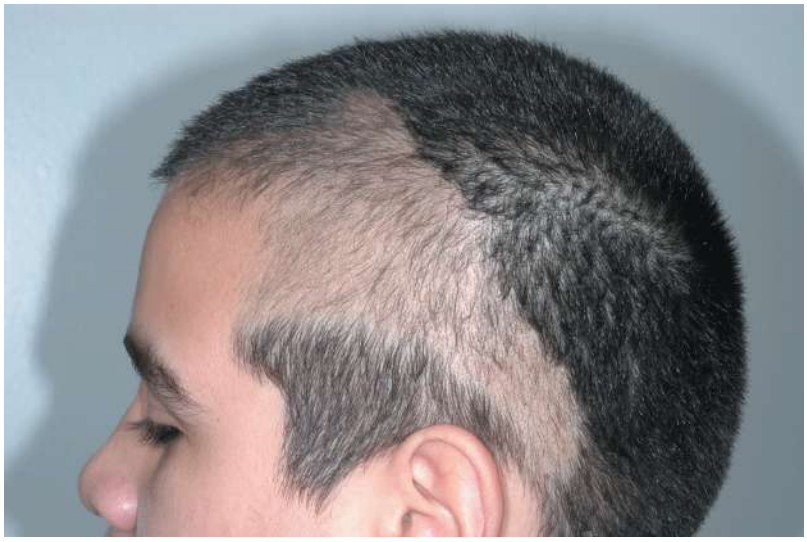

FIGURE 118.6 Trichotillomania in an 11-year-old boy. Note the incomplete patchy hair loss and unusual pattern.

Localised alopecia areata

This can also occur in children, presenting as a circumscribed area with a normal ‘clean’ scalp surface. A pathognomic sign is the presence of ‘exclamation-mark’ hairs at the margins. Most childhood cases resolve spontaneously but it can progress to total hair loss or recurrent alopecia can occur. Regrowth decades later is a possibility. Treatment with potent topical steroids for 12 weeks or so may help.

Tinea capitis

This is a dermatophyte infection that produces an area of incomplete, ‘unclean’ alopecia with various degrees of scaling and inflammation of the scalp surface. A boggy swelling (kerion) can develop in severe cases. Important dermatophyte carriers include pet dogs, cars and rodents. Wood’s light examination will be positive in only 50% of cases. Confirm diagnosis with scalp scrapings for microscopy and culture.

Hirsutism

Hirsutism is growth in the female of excess, coarse, terminal pigmented hair in androgen-dependent sites, namely in a male sexual pattern (e.g. upper lip, beard area and back; see FIG. 118.7).

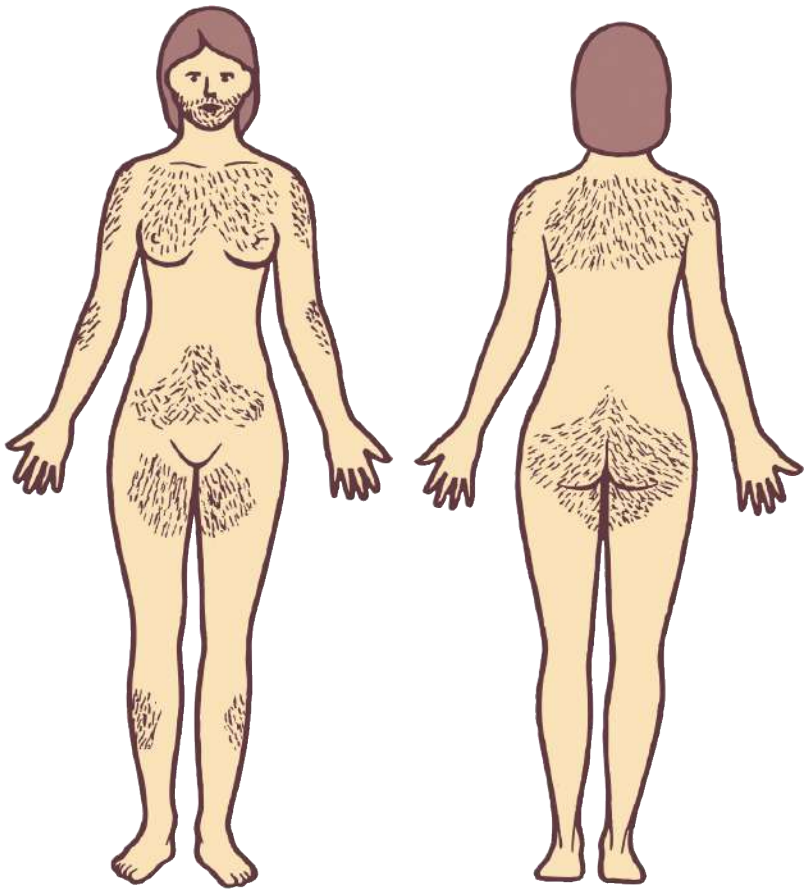

FIGURE 118.7 Areas prone to hirsutism

Most cases are due to idiopathic hirsutism, which may follow ethnic or familial factors, or to polycystic ovarian syndrome. Other causes include adrenal hyperplasia, virilising adrenal tumours, Cushing syndrome, virilising ovarian tumours and certain medications.

The diagnostic strategy model is presented in TABLE 118.4 and followed by the key features.

Table 118.4 Hirsutism in women: diagnostic strategy model

Probability diagnosis
Constitutional (physiological or familial) Polycystic ovarian syndrome (PCOS)
Serious disorders not to be missed
Cancer/tumour: • virilising ovarian tumour • adrenal tumours (cancer and adenoma)

- ectopic (paraneoplastic) hormone production (e.g. lung cancer, carcinoid)

Pitfalls (often missed)

Postmenopausal

Rarities:

- porphyria cutanea tarda
- congenital adrenal hyperplasia
- gonadal dysgenesis

Seven masquerades checklist

Drugs (many incl. phenytoin, danazol, minoxidil, anabolic steroids, cyclosporin, corticosteroids, phenothiazines, interferon α, penicillamine)

Thyroid/other endocrine (prolactinaemia, Cushing syndrome, acromegaly, hypothyroidism)

Key history

History of age and rate of onset, extent and activity of the hair. Menstrual history, family history and past medical history including endocrine disorders and drugs, especially those listed above.

Key examination

- General inspection including distribution and character of the hair growth, endocrine abnormalities (e.g. Cushing syndrome), skin, abdomen and breasts
- Note other features of virilisation, specifically androgenic alopecia, acne, deepening of the voice and clitoromegaly (if indicated)

Key investigations

- Consider pituitary hormones (e.g. FSH, LH, ACTH, TSH, prolactin)
- Androgens (e.g. serum testosterone, SHBG, DHEAS, 17-OH progesterone)
- Pelvic ultrasound (polycystic ovaries)
- Urinary porphyrins
- Imaging of pituitary and adrenal regions

Routine investigation is not required unless hirsutism is severe, is of sudden onset or there are other features of virilisation.

Hirsutism of reasonably sudden onset is an important marker for serious underlying pathology. It is important to exclude adrenal or ovarian pathology.

For patients with severe hirsutism and regular menses, serum testosterone, DHEAS and early morning 17-hydroxyprogesterone levels should be measured. If menses are irregular, measure follicle stimulating hormone, luteinising hormone and serum prolactin.

Appropriate investigations include pelvic ultrasound to diagnose polycystic ovaries and serum testosterone and dehydroepiandrostenedione (DHEA), which indicates an adrenal cause of androgen excess if elevated.

Red flags

- Virilisation
- Abrupt onset of hirsutism
- Pelvic or abdominal mass

Principles of management

- Exclude underlying adrenal or ovarian pathology.
- Support the patient to achieve an acceptable body image through appropriate advice and referral.
- Recommend appropriate cosmetic measures, which include bleaching, waxing, depilatory creams or shaving.
- Do not pluck hairs, especially around the lips and chin—plucking stimulates hair growth but shaving appears to have no effect.
- Laser may also help but seems to be most suitable for dark hair on a light skin and provides temporary reduction, lasting up to 9 months.

Pharmaceutical options[9]

These include either suppression of ovarian and/or adrenal androgen production (oral contraception or dexamethasone) or inhibition of androgen action at the hair follicles (spironolactone and cyproterone acetate).

If the woman is of reproductive age and has no immediate wish for fertility, use:

combined oral contraceptive pill (COCP): ethinyloestradiol + cyproterone or drospirenone

Cyproterone and drospirenone have anti-androgenic properties. Other COCPs may also be useful because they increase SHBG production and reduce ovarian androgen production.

Anti-androgenic drugs:[1]

spironolactone 100–200 mg (o) daily (takes 6–12 months to respond)

For women who are premenopausal or perimenopausal, use:

cyproterone acetate 10–100 mg (o) daily for 10 days of each menstrual cycle plus a COCP

Cyproterone used alone usually leads to hypo-oestrogenism. Therefore, it should be combined with oestrogen unless this is contraindicated. Alternative oestrogen combination regimens are required for postmenopausal women.

Hypertrichosis

Hypertrichosis is the increased growth of fine vellus or downy hair over the body that may either be generalised (usually) or localised. It is non-androgen dependent hair and does not respond to anti-androgen therapy. The cause is generally unknown—it may be primary or constitutional where it is usually apparent prior to puberty and where it is evenly distributed over the back and limbs. Prepubertal hypertrichosis is commonly familial but a positive family history is not always determined.

An important cause of secondary hypertrichosis is drugs. Drugs that are implicated include phenytoin, minoxidil, cyclosporin, interferon alpha and corticosteroids, both systemic and topical.

It is also associated with an underlying neoplasia, such as stomach cancer, when it is termed hypertrichosis lanuginosa acquisita or ‘malignant down’.

Other causes include anorexia nervosa, the menopause, systemic illness, starvation and a few rare syndromes (e.g. Cornelia de Lange syndrome). Treat the underlying cause, otherwise treatment is only necessary if it concerns the patient. Physical hair removal techniques are appropriate for hair that is non-androgen dependent.

Scaly scalp disorders

Dandruff

Dandruff (pityriasis capitis) is mainly a physiological process, the result of normal desquamation of scale from the scalp. It is most prevalent in adolescence and worse around the age of 20.

If it is persistent with heavy scaling, seborrhoeic dermatitis and scalp psoriasis, which is distinguished by palpable plaques, are likely causes.

Dandruff and seborrhoeic dermatitis are related conditions on a continuum of severity. They are caused mainly by the fungal genus *Malassezia*, such as *M. globosa*, which produces lipases that break down sebum. They both have a common pathogenesis and are chronic recurring conditions presenting with scaling and a varying degree of itch. More importantly, they all respond to similar treatments.

Treatment

Shampoos:

- zinc pyrithione (e.g. Dan-Gard, Head and Shoulders)

  or

- selenium sulfide (e.g. Selsun)

Method: massage into scalp, leave for 5 minutes, rinse thoroughly—twice weekly.

Treatment of persistent or severe dandruff:

- coal tar plus salicylic acid compound (Sebitar) shampoo

  or

- T Gel/Ionil T plus shampoo

Method: as above, followed by Sebi Rinse or ketoconazole (Nizoral) shampoo. If persistent, especially itching, and Nizoral shampoo is ineffective, use a corticosteroid (e.g. betamethasone scalp lotion).

Dry hair

Advice to patients

- Don’t shampoo every day.
- Use a mild shampoo (labelled for ‘dry or damaged hair’).
- Use a conditioner.
- Have hair cut regularly to remove split or frayed ends.
- Avoid heat (e.g. electric curlers, hair dryers).
- Wear head protection in hot winds.
- Wear a rubber cap when swimming.

Oily hair

Advice to patients

- Shampoo daily with a ‘shampoo for oily hair’.
- Massage the scalp during the shampoo process.
- Leave the shampoo on for at least 5 minutes.
- Avoid hair conditioners.
- Avoid overbrushing.
- Attend to lifestyle factors: relaxation and balanced diet are important.

Patient education resources

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

- Dandruff
- Hair loss in women
- Hirsutism
- Male pattern baldness
- Trichotillomania

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119 Nail disorders

A physician should wear white garments, put on a pair of shoes, carry a stick and an umbrella in his hand, and walk about with a mild and benignant look as a friend of all created beings. He should be cleanly in his habits and well shaved, and should not allow his nails to grow.

SUSHRUTA-SAMHITA (5TH CENTURY BCE)

Making a diagnosis of abnormal nails for a concerned patient can be quite simple for a few obvious conditions. However, in many cases the diagnosis can be elusive when we are not familiar with classic patterns that are seen so infrequently. The diagnostic process can be facilitated by learning the basic anatomy and function of the nail, as well as characteristic patterns, which are presented with the aid of diagrams in this chapter. There are, in fact, only a limited number of ways in which injury, infection and inflammation can present in a nail.[1]

The main nail problems encountered in general practice are trauma, onychomycosis, infection, ingrowing toenails, paronychia and psoriasis. Fungal nail infection and psoriasis are the commonest causes of nail dystrophy. Damage to the nail from trauma or disease results in nail dystrophy. The problem of nail changes due to onychotillomania, be it from excessive nail biting, picking or cleaning, should be suspected from the history and examination.

The examination should include a general inspection of the skin including in the webbing of the toes, looking for evidence of a skin disorder such as psoriasis, atopic eczema, alopecia areata, lichen planus and tinea.

Key facts and checkpoints

- The growth rate of nails varies between individuals: fingernails average 0.5–1.2 mm per week while toenails grow approximately half that rate.[2]
- An avulsed or totally dystrophic nail will take up to 9 months to regrow.
- It takes approximately 6 weeks to grow a new cuticle.
- Beau lines associated with a severe acute illness take about 3 months to appear.

- Do not confuse chronic paronychia with onychomycosis. The former affects the nail folds, the latter mainly affects the distal nail.
- Nail clippings and subungual scrapings for culture and microscopy may be the only way to differentiate between nail dystrophy and onychomycosis.
- Not all white crumbly nails are caused by a fungus.
- Suspect melanoma in any subungual pigmented lesion. Do not confuse with subungual haematomas, which 'grow out' with the nail. They usually present as a longitudinal pigmented streak. Beware of amelanotic melanoma, which may mimic chronic paronychia or a pyogenic granuloma.[2,3] Any suspicion necessitates early referral.
- Various dermatoses and connective tissue disorders can affect the nails—psoriasis, lichen planus, lupus erythematosus, scleroderma, bullous pemphigoid, Darier disease (keratosis follicularis).[4]
- Clubbing of the fingers is basically an abnormality of the fingertips rather than nails—look for evidence of major pulmonary or cardiac disease.
- Significantly bitten or traumatised nails may be a symptom of a major anxiety disorder—explore psychogenic issues.

A summary of causes of abnormal nails is presented in TABLE 119.1 .

Table 119.1 Abnormal nails: diagnostic strategy model (modified)

Probability diagnosis
Fungal infection: onychomycosis
Onycholysis • trauma to nail bed • trauma from biting • trauma from habit picking
Onychogryphosis
Paronychia
Psoriasis
Serious disorders not to be missed
Melanoma
Iron deficiency: koilonychia

Liver disease: leuconychia
Endocarditis: splinter haemorrhages
Chronic kidney failure: white bands, half-and-half nail
Glomus tumour
Bowen disorder/SCC
Pitfalls (often missed)
Atopic dermatitis
Lichen planus
Pyogenic granuloma (usually with ingrowing toenails)[3]
Drug effects (e.g. tetracycline)
Pseudomonas infection
Connective tissue disorders (e.g. SLE)
Arsenic (Mees stripes)

Key history

Enquire about a history of severe stress or illness and possible onychotillomania, be it excessive nail biting, picking or cleaning. Ask whether the hands are frequently in wet work (e.g. dishwashing, soaps and detergents) or dirt.

Key examination

Careful examination of nails: look for associated skin disease (e.g. psoriasis, atopic dermatitis, alopecia areata, tinea pedis, lichen planus).

Key investigations

- Nail clippings and subungual scrapings for culture and microscopy
- FBE and ESR
- Consider LFTs

Anatomy and function of the nail

The basic nail unit (apparatus) consists of the nail matrix, the proximal and lateral nail folds, the cuticle and the nail plate (see FIG. 119.1). The hard keratin of the nail plate is formed in the nail matrix. The matrix, which contains germinal epithelium, lies in an invagination of the epidermis (the nail fold) and is protected by a waterproof seal formed by the cuticle. The matrix runs from the proximal end of the nail fold to the cuticle.

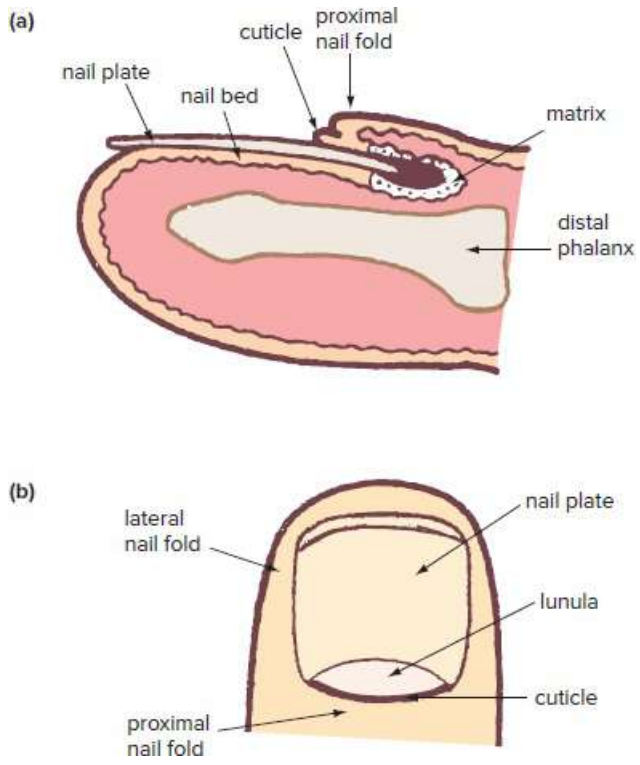

FIGURE 119.1 Normal anatomy of the nail. Diagram of the nail apparatus: **(a)** lateral section view, **(b)** dorsal surface view

The nail bed closely approximates the distal phalanx. The nail has an important functional and cosmetic role. It enhances fine touch and motor skills for picking up fine objects, such as a pin from a flat surface, not possible without fingernails.

Nail disorders and their causes[1]

Lifting of the nail plate (onycholysis)

See FIGURE 119.2 .

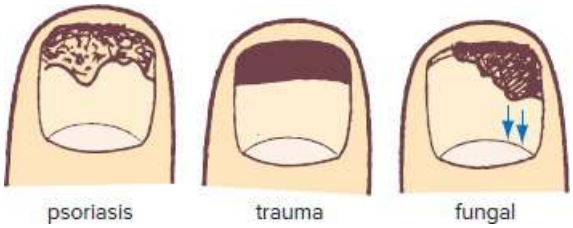

FIGURE 119.2 Lifting of the nail plate (onycholysis)

Causes

- Trauma
- Factitious (self-induced)
- Tinea
- Psoriasis
- Photosensitivity, usually tetracyclines
- Others (e.g. warts, lichen planus)
- Hyperthyroidism
- Nail destruction:

    squamous cell carcinoma

    melanoma

    lichen planus

Thickening of the nail plate

Causes

- Developmental
- Trauma
- Tinea
- Psoriasis (see FIG. 119.3)
- Onychogryphosis
- Age-related changes

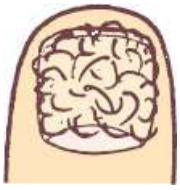

FIGURE 119.3 Psoriasis

Thinning of the nail plate

Causes

- Trauma—wear and tear (repeated water immersion)
- Artificial fingernails—application and removal
- Lichen planus
- Peripheral vascular disease (impaired peripheral circulation)
- Twenty-nail dystrophy, usually children
- Brittle nails

Pitting of the nail plate

See FIGURE 119.4 .

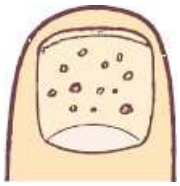

FIGURE 119.4 Pitting of the nail plate

Causes

- Psoriasis
- Alopecia areata
- Atopic dermatitis

Longitudinal marks in the nail plate

See FIGURE 119.5 .

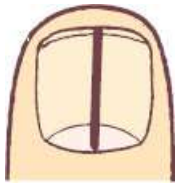

FIGURE 119.5 Longitudinal marks in the nail plate

Causes (grooved)

- Myxoid cyst
- Angiofibroma
- Ageing

Causes (central single stripe)

- Darier disease (red and white bands)
- Hereditary/congenital
- Mechanical trauma

Horizontal grooves in nail plate

Causes

- Beau lines (from acute illness) (see FIG. 119.6)
- Habit tic (picking cuticle of thumb nail) (see FIG. 119.7)
- Atopic dermatitis
- Raynaud phenomenon

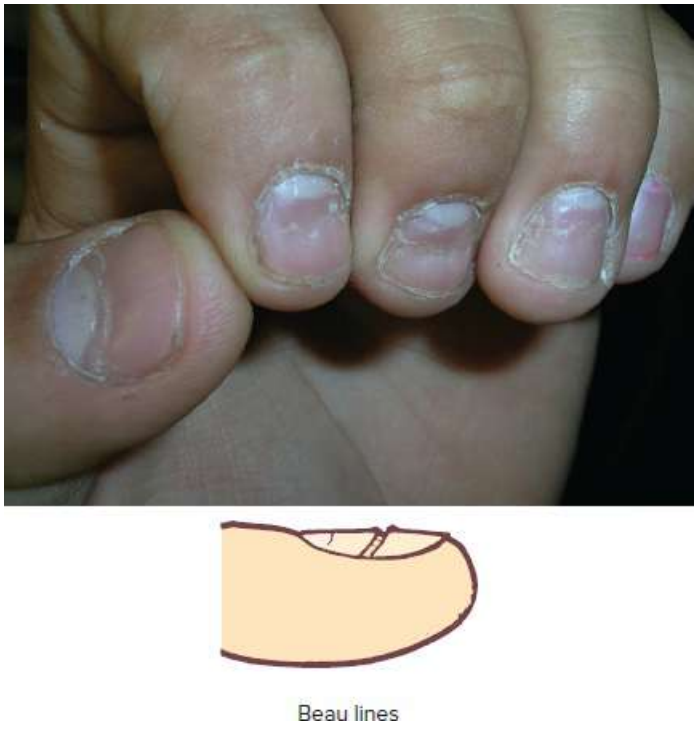

FIGURE 119.6 Beau lines in nails following an episode of acute cholecystitis a few months earlier

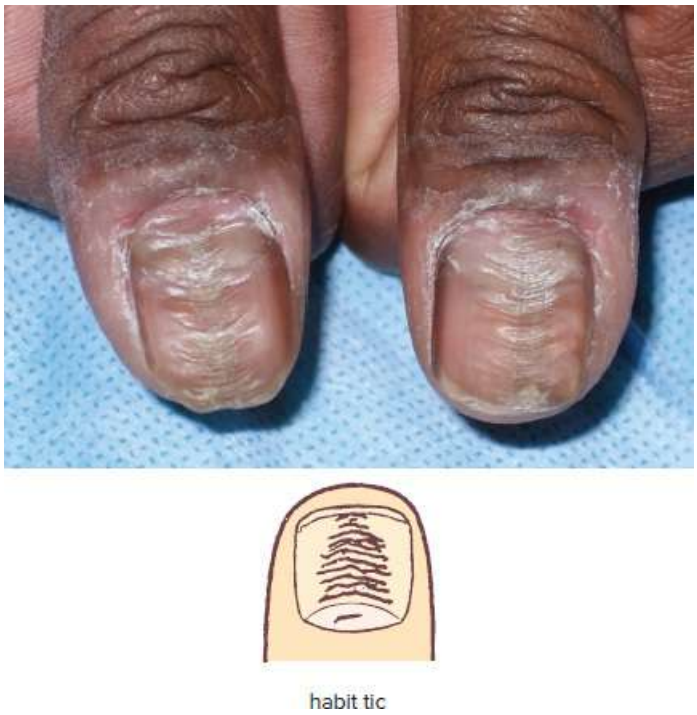

FIGURE 119.7 Habit tic: horizontal grooves on thumb nails

Horizontal single white lines or band

Causes

- Chemotherapy
- Arsenic poisoning
- Kidney failure

Lamellar splitting (onychoschizia)

See FIGURE 119.8 .

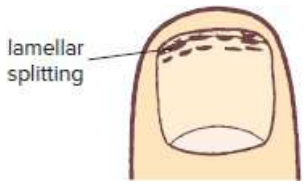

FIGURE 119.8 Lamellar splitting

Causes

- Trauma—repeated wetting and drying (e.g. housework)
- Age-related

Abnormal curvature

Causes

Spoon nails: koilonychia

See FIGURE 119.9 .

- Idiopathic (most cases, esp. children)
- Hereditary
- Trauma
- Iron deficiency

- Haemochromatosis

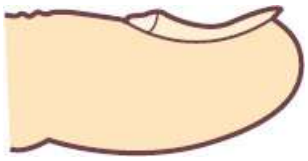

FIGURE 119.9 Spoon nails (koilonychia)

Overcurvature

- Hereditary pincer nail

Clubbing

See FIGURE 119.10 .

- Hereditary/congenital
- Lung disease, most common acquired cause (e.g. cancer, pulmonary fibrosis, bronchiectasis)
- Heart disease (e.g. congenital cyanotic, SBE)
- Liver disease (e.g. cirrhosis)
- Gastrointestinal disorders (e.g. Crohn disease)

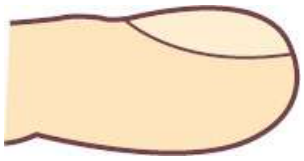

FIGURE 119.10 Clubbing

Splinter haemorrhages

See FIGURE 119.11 .

FIGURE 119.11 Splinter haemorrhages

Causes

- Minor trauma (e.g. manual workers)
- Psoriasis
- Bacterial endocarditis

Discolouration of the nail plate

- White:
  - striate leuconychia (usually trauma)
  - liver cirrhosis
  - hypoalbuminaemia (total whiteout)
- Red:
  - splinter haemorrhages
  - kidney failure
  - polycythaemia
  - CO poisoning
  - glomus tumour
  - onychopapilloma
- Brown:
  - fungal infection
  - cigarette staining
  - drugs—gold, chlorpromazine, cytotoxic drugs
  - Addison disease
  - psoriasis (‘oil stain’ patches)
  - melanoma

- Black:
    - haematoma
    - melanoma
    - racial pigmentation (usually multiple nails)
    - naevus
    - minocycline (usually multiple nails)
    - cytotoxic drugs (transverse bands in multiple nails)
- Green:
    - *Pseudomonas* infection
    - *Aspergillus* infection
- Blue:
    - antimalarials
    - argyria
- Yellow:
    - yellow nail dystrophy (?respiratory illness)
    - fungal infections
    - psoriasis
    - slow growth
    - tetracyclines
- Half (pale proximal) and half (brown or red distal) (i.e. half-and-half nails):
    - kidney failure (chronic)
    - liver cirrhosis
    - trauma (biting, splits, splinters)

Swelling of the proximal nail folds (paronychia)

- Trauma (e.g. biting)
- Manicure (retracting the cuticles)
- *Candida* infection (chronic)
- Staphylococcal infection (acute)
- Herpetic whitlow (recurrent)

Swelling of the lateral nail folds

- Ingrowing toenails
- Overcurvature of the nail plate
- Medications (retinoids, antiretrovirals, targeted cancer therapies)

Tumours of the nail fold

Benign:

- myxoid (mucus) cyst
- warts
- periungual fibroma

Malignant:

- squamous cell carcinoma
- melanoma

Destruction of the nail apparatus

- Trauma
- Lichen planus
- Melanoma
- Bowen disease
- Squamous cell carcinoma

Onycholysis[1]

Onycholysis refers to the separation of the nail plate from the underlying nail bed and is a sign rather than a disease. This separation creates a subungual space with an air interface that gathers unwanted debris, such as dirt and keratin. It is usually seen in fingernails but it can develop in toenails from rubbing against shoes. Adverse local reactions to agents such as formaldehyde and resins in polishes or nail glues can distort nails.

Self-induced trauma is a common cause from obsessive manipulation, including meticulous cleaning and frequent manicuring.

The band of discolouration at the end of the separated nail is usually in a straight line compared with other causes such as psoriasis and tinea. Tinea may be distinguished from other causes by white or yellow streaks or ‘spears’ travelling proximally in the nail.

Greenish discolouration indicates invasion by *Pseudomonas pyocyanea* or *Aspergillus.*

Management[5]

First exclude psoriasis, tinea (check toe webbing) and trauma (check history).

Fingernails

- Keep nails as short as possible
- Avoid insertion of sharp objects under nails for cleaning out debris
- Apply tape (Micropore or similar) over free edge for months, until healed
- Avoid unnecessary soaps and detergents—wear cotton-lined gloves for housework, gardening, etc.
- Keep hands out of water
- Use a mild soap and shampoo
- First-line treatment for mild infection is white vinegar soaks, diluted 1:1 with water—10 minutes two times daily for 3–4 weeks.[6]

Toenails

- Exclude fungal infection (clinical tinea pedis)—culture
- Improve footwear to avoid any rubbing

Pharmaceutical treatment

- Daily application of an imidazole (e.g. clotrimazole) or terbinafine
- Potent topical corticosteroids in lotion form may be useful

- For *Pseudomonas* infection soak the nails in vinegar or Milton's solution and/or gentamicin sulfate cream
- If *Candida albicans* confirmed, best to use oral treatment, e.g. fluconazole (150–300 mg (o) once weekly) or itraconazole[6]

Refer difficult and unresponsive cases to a dermatologist.

Psoriasis of nails

Psoriasis can have many manifestations, such as pitting, onycholysis, discolouration, splinter haemorrhages, distal subungual hyperkeratosis (which can resemble warts) and severe total nail dystrophy (often with arthropathy). Psoriasis can closely mimic onychomycosis, which should be excluded by fungal culture and histology before commencing presumed tinea therapy. The two conditions may also coexist.

There is no effective topical therapy for psoriasis of the nails but a trial of a potent corticosteroid lotion may help selected cases. Intralesional steroid injections, which are painful and require multiple treatments, can help. Caution should be taken to avoid nail trauma, which may result in Koebnerisation. Successful treatment of the skin does not help the nails.

Onychomycosis (fungal nail infection)

Key facts and checkpoints

- Affects 3–5% of population and 40% over 60 years.[1]
- Classified as distal (most common), proximal or superficial.
- Toenails affected more commonly than fingernails.
- The most common form is distal lateral subungual caused by *Trichophyton mentagrophytes* var. *interdigitale* (typical of toe web space tinea and responds well to terbinafine) or by *Trichophyton rubrum* (common on sole of foot and more resistant).
- Superficial white onychomycosis is also common, and is usually confined to the toenails with small superficial white plaques with distinct edges and caused by *Trichophyton mentagrophytes* var. *interdigitale.*
- Proximal onychomycosis is associated with immunocompromise such as HIV.
- Total dystrophic onychomycosis—whole nail affected, thickened, opaque and yellow brown (caused by *Trichophyton* sp.).

- *Candida albicans* and other fungi are not a common factor.
- Diagnosis—always confirm by culture and microscopy of the distal nail plate clippings and scrapings from the underside of the nail. Positive in 60–80% of cases.

Treatment[5,7]

In many cases it is not a major clinical problem and no treatment is a reasonable plan. Regular nail clipping is important. The antifungal treatment of choice for all types of toenail tinea is:

terbinafine 250 mg (o) daily (adult dose) for 12 weeks (cures 70–80% of cases); 6 weeks for fingernails

An alternative is itraconazole 200 mg (o) bd for the first week of each month for 2 months for fingernails; 3–4 months for toenails.

Tips

No improvement apart from the proximal part of the nail will be noticed after months because it takes 12 months or more for the toenail to grow.

Mark the base of the dystrophic nail with a scalpel blade or black ink to assess progress.

Topical treatment for superficial or distal nail involvement[8]

amorolfine 5% (Loceryl) nail lacquer 1–2 times weekly after filing (fingernails: 6 months; toenails: 9–12 months)

or

miconazole tincture daily until resolution

or

ciclopirox lacquer daily until resolution

or

arthrospira maxima lotion bd

A systematic review found poor evidence for the effectiveness of topical therapy for onychomycosis.[9]

Consider twice-daily applications of tea-tree oil indefinitely for tinea pedis and tinea unguium.

Treatment may be less effective in patients with extensive involvement, peripheral vascular disease, diabetes and immunocompromise.

Paronychia

Acute paronychia

This is a painful condition that is mainly due to bacterial infection after minor trauma, especially *Staphylococcus aureus*.

Management

Uncomplicated with localised pus:

- simple elevation of nail fold or puncture the fold to drain pus
- advice on hygiene
- oral antibiotics not usually necessary
- if not responding to drainage or if more extensive, use (di)flucloxacillin
- exclude diabetes
- if recurrent, consider HSV (herpetic whitlow)

Complicated with subungual extension:

- small vertical incision alongside the nail

  or

- removal of nail in part or totally
- exclude diabetes

Herpetic whitlow

A very painful type of paronychia due to herpes simplex virus of the posterior aspect of the nail fold. Blistering is a key feature and the patient may have a cold sore. Treat with oral antiviral therapy as for a primary episode of oral mucocutaneous herpes simplex virus (see CHAPTER 114).

Chronic paronychia

Clinical features

- Painless

- A form of traumatic nail dystrophy
- Loss of cuticle fundamental to diagnosis

Causes

- Excessive manipulation of cuticles (e.g. by manicurists)
- Occupational (e.g. chefs, housewives, nurses, fishmongers)
- Frequent contact with water, detergents and chemicals
- Hand dermatitis
- Habit tic—picking the nail fold

Note:

- Secondary infection with *Candida* common but not basic cause.
- The damaged cuticle permits access of water and grit to the nail matrix and causes inflammation of the proximal nail fold.

Management[4,5]

- Culture organisms
- Exclude diabetes
- Basic nail care advice:

    keep hands dry (avoid wet work if possible)

    wear cotton-lined gloves when washing dishes (for max. of 15 minutes)

    minimise contact with water, soap, detergents, lipid solvents and other irritants

    never pick, push back or manicure cuticles

    never insert anything beneath cuticle for cleaning

    wear cotton gloves in garden

    use a mild soap and shampoo

Medications[5]

For *Candida* (if cultured):

tincture miconazole 2% bd for 7 days

or

clotrimazole topical preparations

For *Staphylococcus* (if cultured):

Bactroban topical ointment

Topical medications to nail folds (especially if persistent exudate):

4% thymol in alcohol (SVR) qid

or

10% sulfacetamide in alcohol

- Vaseline (to waterproof) can be applied frequently (5–10 times daily) when it is dry and without exudate.
- Very potent topical corticosteroids are helpful.
- Refer unresponsive cases.

Atopic dermatitis

- Avoid irritants: special soaps, wear gloves for dishwashing
- Good nail hygiene
- Apply potent topical steroids to proximal nail fold

Lichen planus

This commonly presents in the nail with atrophy of one or more nails. The nail plate is predisposed to breaking and splitting as it thins. If advanced, a pterygium arising from the proximal nail fold grows as the nail matrix is destroyed and eventually total, permanent loss of the nail may occur.

Biopsy of the nail matrix is recommended before treatment.

Intralesional steroids into the proximal nail fold are the treatment of choice if seen prior to destruction of the nail unit. Otherwise a trial of prednisolone 25 mg (o) daily for 4 weeks, reducing the dose gradually over 1–2 weeks, may induce temporary remission. Dermatologist referral is advised.

Twenty-nail dystrophy

This is an uncommon disorder seen in preschool or pre-adolescent children with thinning and roughening of all or almost all 20 nails. It may be idiopathic or associated with alopecia areata (most common), psoriasis, eczema or lichen planus. The condition tends to be self-limiting and most cases resolve over 2–3 years.[4]

It is reported to respond well to the very potent topical steroid, clobetasol.

Brittle nails

The nails break easily, usually at their distal end. Brittle nails are age related and are usually caused by local physical factors, such as repeated water immersion, and exposure to chemicals, such as detergents, alkalis and nail polish removers and also hypothyroidism and digital ischaemia. Systemic causes such as deficiency of iron and vitamins are not considered to be a common factor.

Calcium does not contribute to the hardness of nails and calcium deficiency does not cause brittle nails.[4] No cosmetic applications appear to be helpful.

Management[4]

- Avoid excessive hydration and trauma
- Wear rubber gloves with cotton liners for wet work
- Massage Vaseline or nail creams (e.g. Eulactol or NeoStrata) into the nail several times daily
- Nail polishes and hardeners (preferably without formalin) may give a good cosmetic result

Nail apparatus melanoma

Clinical features[5]

- Rare but high fatality
- Responsible for 0.7–3.5% of all melanomas
- All age groups but especially in 5th–7th decades
- Affects all ethnic groups in all climates
- Presents as a longitudinal pigmented streak >3mm in the nail (see FIG. 119.12)
- Amelanotic in a quarter of cases

- Hutchinson sign (pigmentation of the proximal nail fold) may be present
- Usually delayed diagnosis
- 5 year survival 51%
- Early recognition and referral may result in a cure

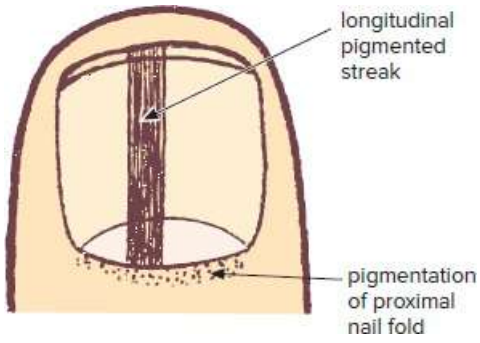

FIGURE 119.12 Longitudinal pigmented streak: sign of nail apparatus melanoma

Management

- All cases require a biopsy of the nail matrix for diagnosis
- If confirmed, treatment is based on Breslow thickness and level of invasion
- Level 1 or in situ—removal of whole nail apparatus
- Invasive melanoma—amputation of distal phalanx

Glomus tumours

Glomus tumours are those mysterious little tumours that occur beneath the nail plate and can give paroxysms of sharp pain.

They are small purplish lesions that are exquisitely tender to touch and activate with changes in temperature. They may not be visible but may be revealed when the nail bed is blanched with gentle pressure. MRI may be needed for diagnosis. Treatment is surgical.

Onychogryphosis

Onychogryphosis, or irregular thickening and overgrowth of the nail, is commonly seen in the big toenails of the elderly and appears to be related to pressure from footwear (see FIG. 119.13). It is really a permanent condition. Simple removal of the nail by avulsion is followed by recurrence some months later. Softening and burring of the nail gives only

temporary relief, although burring sometimes provides a good result.

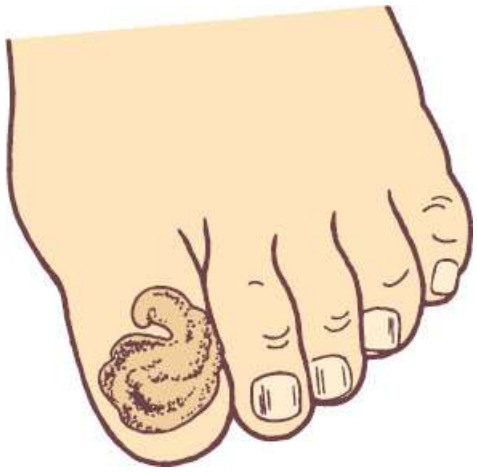

FIGURE 119.13 Onychogryphosis

The powder from burring can be used as culture for fungal organisms. Permanent cure requires ablation of the nail bed after removal of nail.

Two methods of nail ablation are:

1. total surgical excision
2. cauterisation with phenol (with care)

Ingrowing toenails

Refer to section in CHAPTER 57 .

Periungual warts

These are similar to other warts and fortunately rarely spread under the nail to the nail bed, presenting a challenge for safe yet effective treatment.

Cryotherapy must be used with caution to avoid damage to the nail matrix—otherwise it is usually safe but often ineffective. Consider keratolytic wart paints under occlusion. Avoid nail biting as this may lead to Koebnerisation.

Subungual haematoma[10]

The small, localised haematoma

There are several methods of decompressing a small localised haematoma under the fingernail or toenail that causes considerable pain. The objective is to release the blood by drilling a hole in the overlying nail with a hot wire or a drill/needle.

Treatment

Method 1: the sterile needle

Simply drill a hole by twisting a standard disposable hypodermic needle (21 or 23 gauge) into the selected site. Some practitioners prefer drilling two holes to facilitate the release of blood.

Method 2: the hot paper clip

Take a standard, large paper clip and straighten it. Heat one end (until it is red hot) in the flame of a spirit lamp. Immediately transfer the hot wire to the nail, and press the point lightly on the nail at the centre of the haematoma. After a small puff of smoke, an acrid odour and a spurt of blood, the patient will experience immediate relief (see FIG. 119.14).

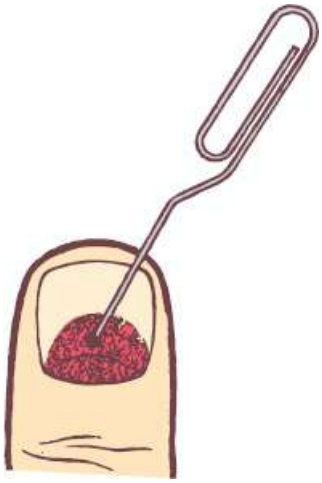

FIGURE 119.14 Treatment of subungual haematoma. The point of a heated end of a paper clip is pressed lightly on the nail at the centre of the haematoma.

Method 3: electrocautery

This is the best method. Simply apply the hot wire of the electrocautery unit to the selected site (see FIG. 119.15). It is very important to keep the wire hot at all times and to be prepared to withdraw it quickly, as soon as the nail is pierced. It should be painless.

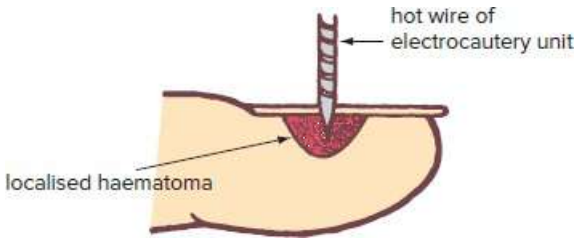

FIGURE 119.15 Electrocautery to subungual haematoma

Important precautions

- Reassure patients that the process will not cause pain; they may be alarmed by the preparations.
- The hot point must quickly penetrate, so go no deeper than the nail. The blood under the nail insulates the underlying tissues from the heat and, therefore, from pain.
- The procedure is effective for a recent traumatic haematoma under tension. Do not attempt this procedure on an old, dried haematoma as it will be painful and ineffective.
- Advise the patient to clean the nail with spirit or an antiseptic and cover with an adhesive strip to prevent contamination and infection.
- Advise the patient that the nail will eventually separate and a normal nail will appear in 6–9 months.

The large haematoma

Where blood occupies the total nail area, a relatively large laceration is present in the nail bed.

Treatment

To permit a good, long-term functional and cosmetic result it is imperative to remove the nail and repair the laceration (see FIG. 119.16).

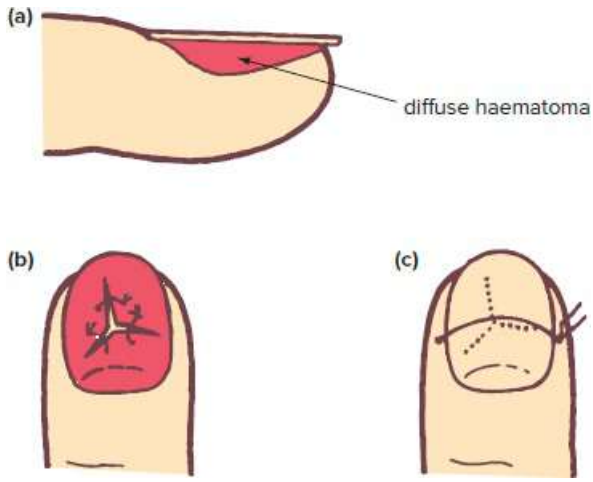

FIGURE 119.16 Treating the diffuse haematoma: **(a)** the diffuse haematoma, **(b)** sutures to laceration, **(c)** fingernail as splint

Method

- Apply digital nerve block to the digit.
- Remove the nail.
- Repair the laceration with 4/0 plain catgut.
- Replace the fingernail, which acts as a splint, and hold this in place with a suture for 10 days.

Myxoid pseudocyst

There are two types of digital myxoid pseudocysts (also known as mucous cysts) appearing in relation to the distal phalanx and nail in either fingers or toes (more common) (see FIG. 119.17). One type occurs in relation to, and often connecting with, the distal interphalangeal joint and the other occurs at the site of the proximal nail fold. The latter (more common) is translucent and fluctuant, and contains thick clear gelatinous fluid, which is easily expressed after puncture of the cyst with a sterile needle. Osteoarthritis of the DIP is associated with leakage of myxoid fluid into the surrounding tissue to form the cyst.

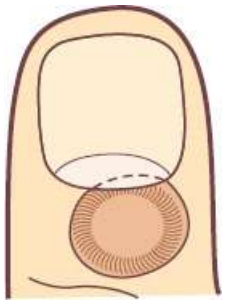

FIGURE 119.17 Myxoid pseudocyst: typical position of the cyst

Some pseudocysts resolve spontaneously. If persistent and symptomatic attempt:[10]

repeated aspiration (aseptically) at 4–6 weekly intervals

or

cryosurgery

or

puncture, compression, then infiltration intralesionally with triamcinolone acetonide (or similar steroid)

They tend to persist and recur; if so, refer to a hand surgeon for total excision of the proximal nail fold and/or ligation of the communicating stalk to the DIP or (preferably) microscopic dissection to the capsule defect and capsule repair.

Care should be taken to avoid septic arthritis due to communication with the distal interphalangeal joint.

Patient education resource

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

- Nail disorders

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Part 11 Accident and emergency medicine

120 Emergency care

When Elisha arrived, he went alone into the room and saw the boy lying dead on the bed. He closed the door and prayed to the Lord. Then he lay down on the boy, placing his mouth, eyes and hands on the boy's mouth, eyes and hands. As he lay stretched out over the boy, the boy's body started to get warm—the boy sneezed seven times and then opened his eyes.

II KINGS 4: 32–5 (A MIRACLE OR SUCCESSFUL ARTIFICIAL RESUSCITATION?)

Definition of the emergency

Emergency: 'An event demanding immediate medical attention'.

The GP must be available and organised to cope with the medically defined emergency when it comes. Emergency care outside the hospital represents one of the most interesting and rewarding areas of medical practice. City doctors will have to modify their degree of availability, equipment and skills according to the availability of paramedical emergency services, while others, especially remote doctors, will need total expertise and comprehensive equipment to provide optimal circumstances to save lives.

The immediate approach to a specific emergency differs from normal, less urgent medical practice. The usual method of history and examination is replaced with a technique of rapid assessment and immediate management. In fact, the primary diagnosis is sometimes possible on the information available over the telephone, or during the first few seconds of surveying the patient.

An obvious yet important concept is that of 'time criticality', which implies that certain patients are at high risk of a critical outcome of deterioration if there is significant delay in appropriate management. A classic example in general practice would be acute coronary syndromes.

Refer also to childhood emergencies (see CHAPTER 89).

Key facts and checkpoints

- The most common emergency calls in a survey of a typical rural general practice[1] were accidents and violence (51%), abdominal pain (10%), dyspnoea (7%), chest pain (6%), syncope/blackout (5%), other acute pain (5%).
- The prevalence of emergency calls was 2.6 per 1000 population per week.
- The most common specific conditions in this study[1] were lacerations (19%), fractures (11%), injuries from transport accidents (11%), asthma (4%), ischaemic heart pain (3.5%), appendicitis (3%).
- The most common causes of sudden death were myocardial infarction (67%), accidents (10%), cerebrovascular accidents (7%), pulmonary embolism (6%), suicide (4%).
- The main vital emergency procedures were cardiopulmonary resuscitation, intubation and ventilation, intravenous access (including venous cutdown), intravenous (or rectal) dextrose and arrest of haemorrhage.

Principles of management

The important principles of management of the emergency call can be summarised as follows:

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1. The practitioner must be aware of life-threatening conditions.
2. The practitioner should be prepared mentally and physically.

   PLAN, EQUIP and PRACTISE.
3. Chest pain/collapse/myocardial infarction (collectively) represents *the* premium emergency call.
4. Beware of children with respiratory distress and traumatic injuries.
5. The most saveable patients are those with blood loss. Hence IV fluids for intravascular volume expansion are essential.
6. The necessary basic skills to cope with most emergencies involve DRSABCD—danger, response, send for help, airway, breathing, circulation, defibrillator.
7. Have the equipment and the skills to handle body fluids potentially contaminated with blood-borne viruses.

3. Seventy per cent of cardiac arrests occur in the home, so the availability of a portable defibrillator is important.

Vital basic skills

1. Rapid intravenous access
2. Cardiopulmonary resuscitation, including upper airway relief, ventilation (intubation if practical), treatment of cardiac arrhythmias and defibrillation
3. Cricothyroidotomy
4. Arrest of haemorrhage
5. Knowledge of usage of common emergency drugs

Alarming symptoms and signs

- Unconsciousness
- Convulsions
- Chest pain in an adult, especially associated with pallor and sweating
- Pallor and sweating in any patient with pain, collapse or injury
- Collapse, especially at toilet
- Significant haemorrhage
- Increasing breathlessness, including asthma
- The agitated person threatening homicide or suicide (beware ‘danger’; take a police officer for company)

Don’t forget the value of oxygen

Ideally, the doctor who attends emergency calls should carry an oxygen delivery unit or at least rely on the simultaneous arrival of an ambulance with resuscitation equipment. Remember, oxygen is a treatment for hypoxia, not for breathlessness; a pulse oximeter is very useful. Most cases require a high flow rate of 8–10 L/minute.

Medical emergencies typically requiring high-flow oxygen:[2]

- acute pulmonary oedema
- acute anaphylaxis

- cardiopulmonary arrest
- collapse
- status epilepticus
- shock, sepsis
- major haemorrhage, major trauma

Medical emergencies where oxygen should be used with caution, unless oximetry confirms hypoxia:[2]

- underlying COPD—however, in the above medical emergency scenarios, start with high flow during urgent phase, but reduce after reassessment
- myocardial infarction—high-flow oxygen may increase infarct size
- stroke
- obstetric emergencies—may harm the fetus if the mother is not hypoxaemic

Twelve golden rules

Here are 12 important rules for the diagnostic approach to the emergency call.

1. In the unconscious person, always consider the possibilities of hypoglycaemia and opioid overdosage.
2. Consider intra-abdominal bleeding first and foremost in someone with abdominal pain who collapses at the toilet.
3. Acute chest pain represents myocardial infarction until proven otherwise.
4. Exclude meningitis and septicaemia in a child with a rapid onset of drowsiness and pallor.
5. Consider a ruptured intra-abdominal viscus in any person, especially a child, with persistent post-traumatic abdominal pain.
6. Consider acute anaphylaxis in those with a past history of allergies.
7. Consider substance use and organic causes for those with acute psychosis or bizarre behaviour.
8. Consider ectopic pregnancy in any woman of child-bearing age presenting with acute abdominal pain.
9. If a person is found cyanosed, always consider upper-airway obstruction first.

). Beware of the asthmatic who is cyanosed with a ‘silent chest’ and tachycardia.

. Consider ventricular fibrillation or other arrhythmia in an adult with sudden collapse or dizziness.

. Consider subarachnoid haemorrhage in anyone with a sudden onset of severe headache.

Important medical emergencies in adults

This section includes summarised protocols for management of emergencies. Further detail is available on websites such as the medical blog Life in the Fast Lane (see: https://lifeinthefastlane.com/).

Acute anaphylaxis and anaphylactic reactions

Fatalities from anaphylaxis have markedly increased in Australia in the past two decades, and hospital admissions for food allergies in children have increased more than fivefold.[3] Common causes of anaphylaxis are: bee stings, wasp stings, other bites (e.g. imported red fire ants, jack jumper ants), parenteral antibiotics (especially penicillin), food reactions (e.g. seafood, peanuts). See FIGURE 120.1 .

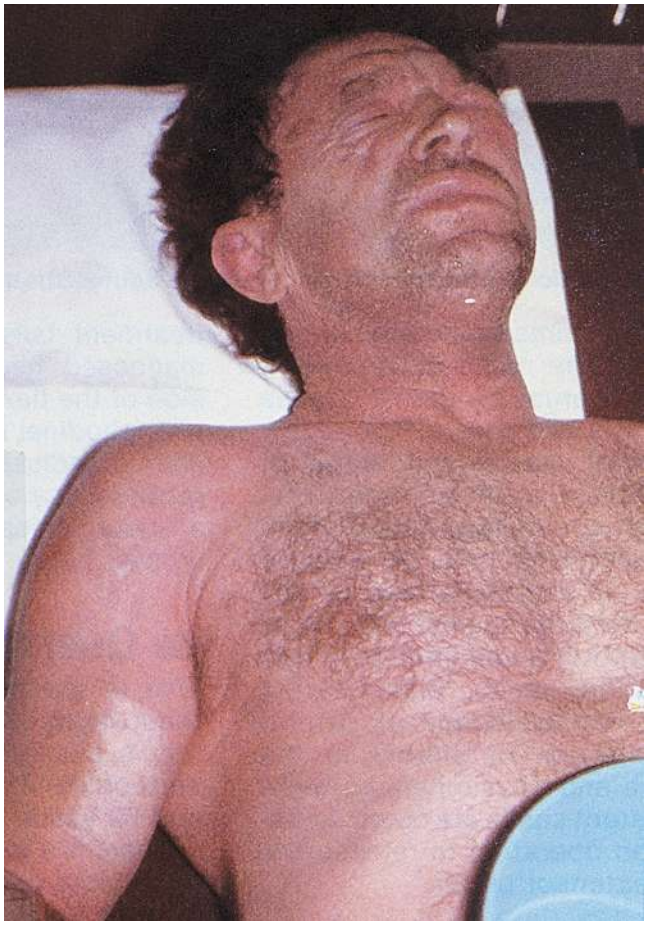

FIGURE 120.1 Acute anaphylaxis with respiratory difficulty caused by a European wasp sting

Other causes include: allergic extracts, blood products, antivenom, radiological contact materials, anaesthetic agents, NSAIDs.

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The onset of anaphylaxis from exposure is usually rapid—typically 10–20 minutes. The early administration of adrenaline saves lives. The diagnosis is basically a clinical one.[4]

Symptoms

- Skin: pruritus—generalised, palate, hands, feet, urticaria, angio-oedema
- Respiratory: wheeze, stridor
- Nausea and vomiting, abdominal pain, diarrhoea
- Hypotension: syncope, collapse
- Palpitations

Note: The early danger symptom is itching of the palms of hands and soles of the feet.

Differential diagnosis: syncope

Refer to the adult anaphylaxis kit in CHAPTER 72 .

First-line treatment (adults)[4,5]

- Call for help
- Remove cause (e.g. bee sting) if possible. Lay the patient down
- Oxygen 8 L/min (by face mask)
- Adrenaline 0.5 mg (0.5 mL of 1:1000, because mg = mL of 1:1000 adrenaline) IM best given in mid antero-lateral thigh
- Set up IV access
- If no rapid improvement:

  repeat IM adrenaline every 3–5 minutes

  set up adrenaline infusion: 1 mg adrenaline to 1000 mL N saline (i.e. 1 mL = 1 mcg adrenaline) bolus of 50 mcg (= 50 mL) can be given as required (best with ECG monitor)
- Set up additional IV line (preferably two ‘wide bore’ lines) and infuse crystalloid solution (e.g. N saline 1–2 L) with bolus (20 mL/kg) over 1–2 min
- Salbutamol aerosol inhalation (or nebulisation if severe), especially if wheeze/stridor
- Consider promethazine (25 mg) IV and hydrocortisone (250 mg) IV
- Admit to hospital (observe at least 6 hours)
- Discharge on promethazine 25 mg tds + prednisolone 50 mg/day for 3 days. Provide an adrenaline autoinjector

Angio-oedema and acute urticaria

Acute urticaria and angio-oedema are essentially anaphylaxis limited to the skin, subcutaneous tissues and other specific organs. They can occur together.

Treatment

- Uncomplicated cutaneous swelling—antihistamines

  e.g. diphenhydramine or promethazine 50 mg (o)

or

25 mg IM if more severe or prednisolone 25–50 mg (o) as a single dose

- Upper respiratory involvement

  adrenaline 0.3 mg SC or IM or 5 mg nebulised

Acute cardiogenic pulmonary oedema

Keep in mind the mnemonic LMNOP (lasix, morphine, nitrates, O_2, CPAP).

- Keep the patient propped up in bed with legs over side
- Oxygen by mask or intranasally (8 L/min)
- Glyceryl trinitrate (GTN spray or tablet) 300–600 mcg sublingual; can use IV nitrates (if BP >100 mmHg)
- Insert IV line (large bore cannula)
- Frusemide 40 mg IV, increasing to 80 mg IV as necessary (or twice the patient's normal oral dose)
- Morphine 1–2.5 mg IV—but caution: reserve for chest pain and anxiety
- CPAP (continuous positive airway pressure)—for unresponsive cases or BiPAP (if available) —in ambulance, ED or CCU

Give amiodarone 3–5 mg/kg loading dose IV infusion if rapid atrial fibrillation.

Note: Keep in mind underlying cause, e.g.:

- myocardial infarction (?silent)
- arrhythmia
- cardiomyopathy
- anaemia

Severe asthma[5]

Severe asthma is a life-threatening condition that is resistant to standard treatment. It requires intensive medication because of marked obstruction to the air passages, due to severe smooth muscle spasm and inflammation, producing mucosal oedema and mucous impaction. Beware the 'silent chest'. (Refer to CHAPTER 73 .)

Initial treatment (summary)

- Oxygen 8 L/min by mask to maintain SpO_2 >95%
- Salbutamol 12 puffs (adult) in a spacer (4 × 4 × 4 rule)

  or

- Continuous nebulised 0.5% salbutamol by face mask, using 8 L/min oxygen for nebulisation

  Insert IV line

- hydrocortisone 250 mg IV or IM

  If poor response:

- add ipratropium 500 g into the salbutamol nebuliser
- magnesium sulphate 2 g (25–100 mg/kg) IV over 20 minutes
- adrenaline 0.5 mg 1:1000 SC or IM or 1:10 000 IV

  If exhausted, moribund and not responding:

- intubation with intermittent positive pressure ventilation (IPPV)
- hydration with IV fluids

Opioid respiratory depression[5]

- Attend to airway (e.g. pocket mask and bag)
- Naloxone 0.4 mg IV boluses titrated to clinical effect

Beware of recurrence of respiratory depression or neurogenic pulmonary oedema from excess naloxone.

Refer to CHAPTER 64 .

Severe hypoglycaemia (confirm by blood glucose around 3 mmol/L or less)

glucagon 1 mL (=1 mg, one ampoule) IM or SC or IV (0.5 mL in child <35 kg)

then oral glucose if able to swallow—can repeat glucagon

or

50% dextrose 20 mL IV through securely positioned cannula, large vein (if IV line difficult, administer rectally by pressing the nozzle of a large syringe into the anus and injecting slowly)

Myocardial infarction/unstable angina

Refer to CHAPTER 30 .

First-line management

- Insert IV line
- Glyceryl trinitrate (GTN spray or tablet) 300 mcg (½ tab) SL, unless BP <90 mmHg or pulse <50/min
- Aspirin 300 mg oral (½ or 1 tab)
- Morphine 1 mg per minute IV until pain relief (up to 15 mg)—usually 2.5–10 mg (lower dose if elderly/frail)
- ECG (set up by an assistant)
- CPAP or BiPAP (if available)
- Arrange ambulance and hospitalisation

Hyperventilation

Calm the patient with a firm but reassuring voice. Remove them from anxiety-provoking stimuli such as panicking bystanders. Consider the possibility of an alternative diagnosis causing hypoxia.

Status epilepticus and serial seizures

Status epilepticus = repeated convulsions (usually >5 minutes) without regaining consciousness after initial tonic–clonic seizure. Significant mortality at >30 minutes.

Serial seizures = repeated convulsions after regaining consciousness.

Management of status epilepticus[6,7]

Focal status

- A high index of suspicion is needed to diagnose
- Oral medication usually adequate

- Avoid overtreatment

Generalised status

Absence attacks (petit mal)

- Hospitalisation
- IV diazepam

Note: These can cause long-term damage.

Tonic–clonic (dangerous!)

Give a benzodiazepine followed by an anti-epileptic agent. Lie patient on side. Ensure adequate oxygenation: attend to airway (e.g. Guedel tube); give oxygen 8 L/min plus:

midazolam 5–10 mg (child: 0.15–0.2 mg/kg up to 10 mg) IM or IV, over 2–5 minutes

or

midazolam 5–10 mg (child: 0.2–0.3 mg/kg up to 10 mg) buccally or intranasally. One drop = 0.3 mg. Repeat once 15 minutes later if seizure continues

or

diazepam 10–20 mg (child: 0.1–0.25 mg/kg up to 20 mg) IV, over 2–5 minutes (max. 5 mg/min). Repeat once 15 minutes later if seizure continues (beware of respiratory depression)

or

clonazepam 1 mg (child 0.25–0.5 mg) IV over 2–5 minutes. Repeat once 15 minutes later if necessary

followed immediately (for all of above) by an anti-epileptic:

phenytoin 15–20 mg/kg IV in a glucose-free solution over 30 minutes (child 25 mg/min) careful monitoring of ECG and BP

or

sodium valproate 10 mg/kg (child: 15–30 mg/kg up to 800 mg by slow IV injection

If uncontrolled, consider intubation with expert assistance for continuing intensive care

Note: Midazolam is suitable for all types of seizures and can be given IM, buccally or intranasally.

Diazepam can be given rectally. In an adult, 10 mg is diluted in 5 mL of isotonic saline and introduced via the nozzle of the syringe into the rectum. The dose in children is 0.5 mg/kg.

Intranasal midazolam (0.2–0.3 mg/kg/dose) up to maximum of 10 mg may be given in children.

DOs and DON'Ts for the onlookers of a seizure

- Don't move the person to another location (unless necessary for safety).
- Don't force anything into the person's mouth.
- Don't try to stop the fit.
- Do roll the person on to their side with the head turned to one side and chin up.
- Do call for medical help if the seizure lasts longer than 10 minutes or starts again.
- Do remove false teeth and help clear the airway once the fit is over.

Recurrent prolonged or repetitive status epilepticus

Those with recurrent prolonged convulsive seizures or serial seizures can be given the following medication options (following appropriate training of carers):

midazolam 10 mg buccally

or

diazepam 10–20 mg rectally (may be available from hospital pharmacies)

Electric shock

Facts

- Direct current (DC) from welding machines or lightning produces more electrolyte tissue damage and burns than does AC (domestic supply).
- Injuries occur at sites distant from entry or exit.
- Severe muscle contractions can cause bone fracture or posterior dislocation of the shoulder.
- Household shocks tend to cause cardiac arrest (ventricular fibrillation), and myocardial damage is common.
- Ischaemic necrosis of a limb or digit is possible.
- Apparently minor initial injuries may be very misleading (see FIG. 120.2).

- Neurological deficits and psychoneurotic sequelae are common in survivors.

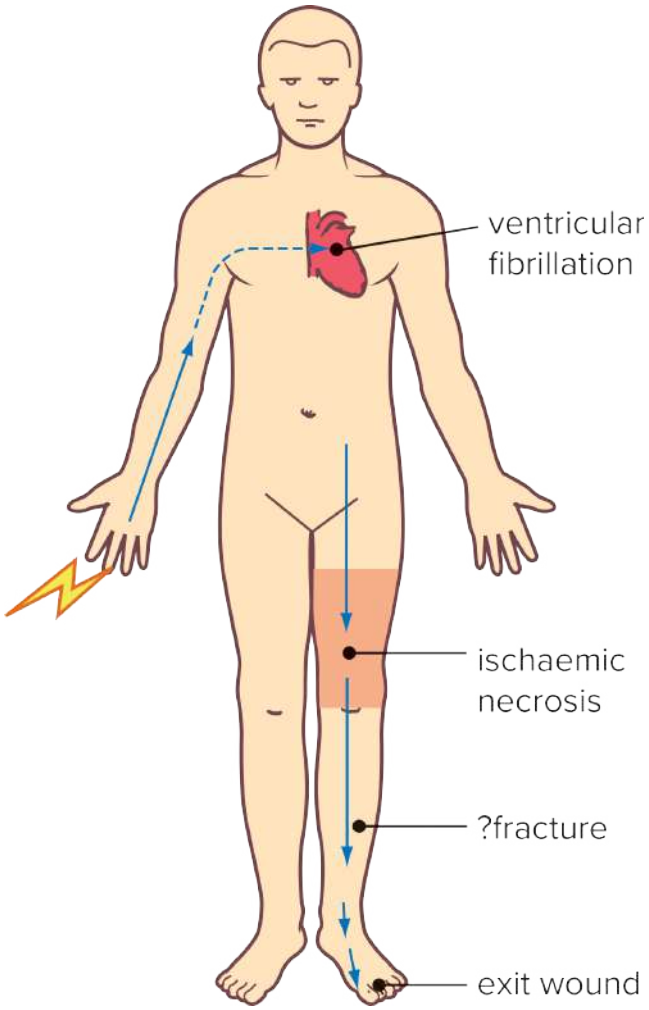


FIGURE 120.2 Effect of electric shock passing through the body

Principles of management

- Attend to DRSABC (see FIG. 120.7). 'Danger' must always come first.
- Make the site safe: switch off the electricity. Use dry wool to insulate rescuers.
- 'Treat even the clinically dead'.
- Give a precordial thump in a witnessed arrest.
- Consider a cervical collar (?cervical fracture).

Bystander CPR and prolonged respiratory support for electric shocks and lightning strike can be lifesaving.

- Provide basic cardiopulmonary resuscitation, including defibrillation (as required).
- Carefully examine the whole body, especially the limbs.
- Consider:

  X-ray of limbs or spine as appropriate

  checking for myoglobinuria and kidney failure

  tetanus and clostridial prophylaxis
- Get expert help—intensive care unit, burns unit.

Lightning strike

Prevention (during an electrical storm)

- Don’t shelter under trees or tall objects (‘splash effect’—see FIG. 120.3).[8]
- Stay indoors—shelter in a building or closed car.
- Avoid using phone.
- Avoid holding metal objects (e.g. golf clubs).
- Keep as low to ground as possible (e.g. curl up in a ditch).
- Avoid being in a group.

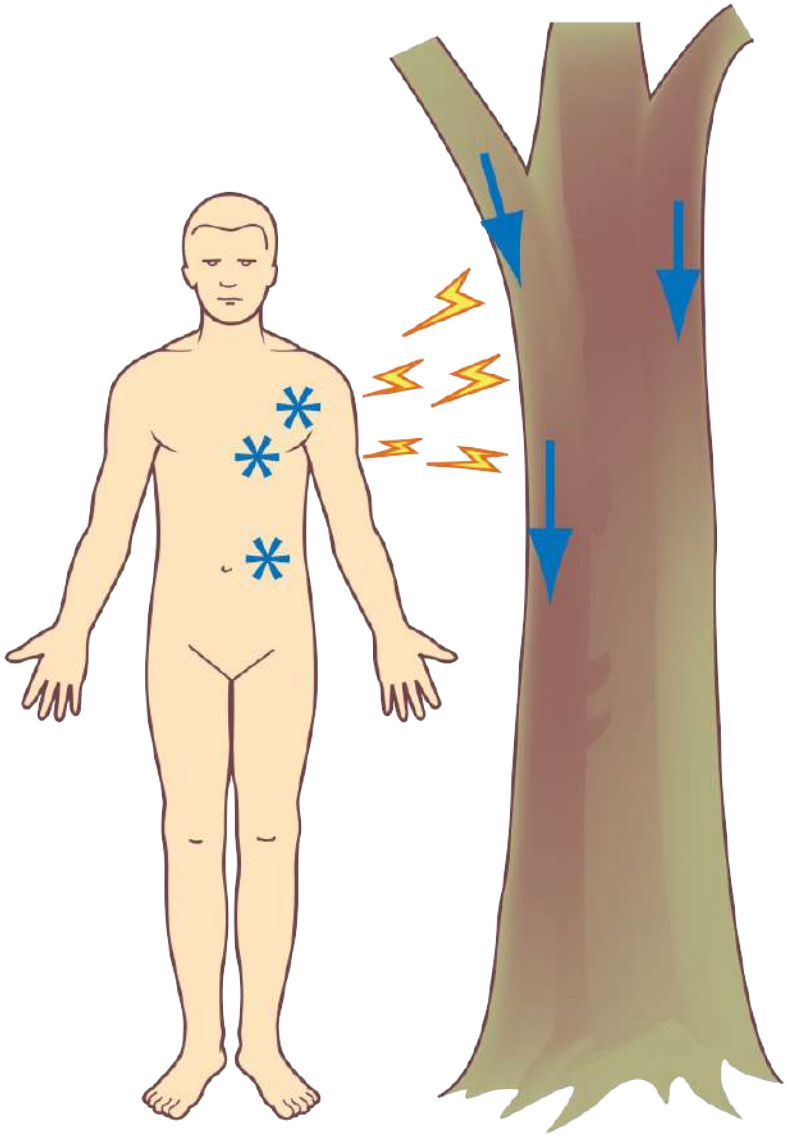


FIGURE 120.3 ‘Splash effect’, where current jumps from a path of higher resistance, such as a tree

Clinical effects

- Burn injury: the ‘flashover’ phenomenon—clothing disintegrates
- Blast injury: ruptured spleen, subdural, ruptured eardrum
- Electrical injury as for household shock (uncommon)

Petrol and solvent sniffing

Substances commonly sniffed include petrol, glues, spirit-based paints, paint spray cans and other aerosols. Refer to drug and alcohol services wherever possible.

The three main acute problems are:

1. shaking and fitting: give midazolam IV or IM or diazepam IV (as for convulsions)
2. agitation/aggressive behaviour, self-harm: try to calm patient in a well-lit room; sedate with diazepam; give haloperidol for hallucinations or delusions
3. general debilitation: this may include acute infections (e.g. chest infection), anaemia, malnutrition

Near-drowning[5]

Near-drowning is survival after asphyxia from submersion in a liquid medium. Hypoxia is the final common pathway in drowning or near-drowning, whether by water aspiration (wet drowning) or by glottic closure (dry drowning).

The rules to remember are that victims can respond to resuscitation after considerable immersion time (up to 30 minutes) and that mouth-to-mouth resuscitation should always be attempted, even if pulseless or with fixed, dilated pupils. The usual routine of basic life support and CPR applies. All symptomatic patients should receive high-flow oxygen and ideally CPAP or BiPAP. Those requiring intubation should receive positive end expiratory pressure (PEEP) in an in-patient facility.

Artificial surfactant given via an endotracheal tube is supported by some evidence in children, but not (yet) in adults.[9]

There is no significant difference in outcome and management between salt-water and fresh-water drowning. Hypothermia should be attended to with warming, such as a hot-air blanket if available and warm fluids.

Epistaxis

Refer to CHAPTER 48 .

Migraine

Refer to CHAPTER 45 .

Bites and stings

Bites and stings from animals, spiders and insects are common in Australia and the US but fatal bites are uncommon.

Snake bites

Snake bites are more common and severe in those handling snakes and in those trying to kill the snake, particularly while inebriated. Snakes are more aggressive when mating or sloughing their skin (about four times a year). They strike for one-third of their length at 3.5 metres/second. Over 70% of bites are on the legs. Sea snakes are not a major problem in Australian waters.

First aid

1. Keep the person as still as possible.
2. Do not wash, cut, apply ice, manipulate the wound or use a tourniquet.
3. Immediately bandage the bite site firmly (same tightness as for sprained ankle). A 15 cm elasticated or crepe bandage is ideal: it should extend above the bite site for 15 cm (e.g. if bitten around the ankle, the bandage should cover the leg up to the knee). Adequate pressure prevents venom moving through the lymphatic system into the bloodstream.
4. Splint the limb to immobilise it: a firm stick or slab of wood would be ideal.
5. Transport to a medical facility for definitive treatment. Do not give alcoholic beverages or stimulants.

Note: A venom detection kit is used to examine a swab of the bitten area or a fresh urine specimen, to identify the snake species involved.

The bandage can be cautiously removed when the patient is safely under medical observation. Observe for symptoms and signs of envenomation.

Antivenom is polyvalent or species-specific for most venomous Australian snakes.

Envenomation

Not everyone bitten becomes envenomated and the antivenom should not be given unless there is evidence of this. Envenomation is more likely when the snake has a clear bite, such as in snake handlers, barefooted people or those who have placed their hands in burrows.

Apart from non-specific systemic effects (as listed) there may be major organ effects according to the type of snake and its toxin. These include coagulopathy, neurotoxicity, myolysis and kidney damage.

Important early symptoms of snake bite envenomation include:

- nausea and vomiting (a reliable early symptom)
- abdominal pain
- excessive perspiration

- severe headache
- dizziness, blurred vision
- difficulty speaking or swallowing
- coagulation defects (e.g. haematuria)
- tender lymphadenopathy

The greatest danger is respiratory obstruction and failure or unexpected catastrophic bleeding (e.g. intracerebral haemorrhage).

Refer to FIGURE 120.4 for detailed effects.

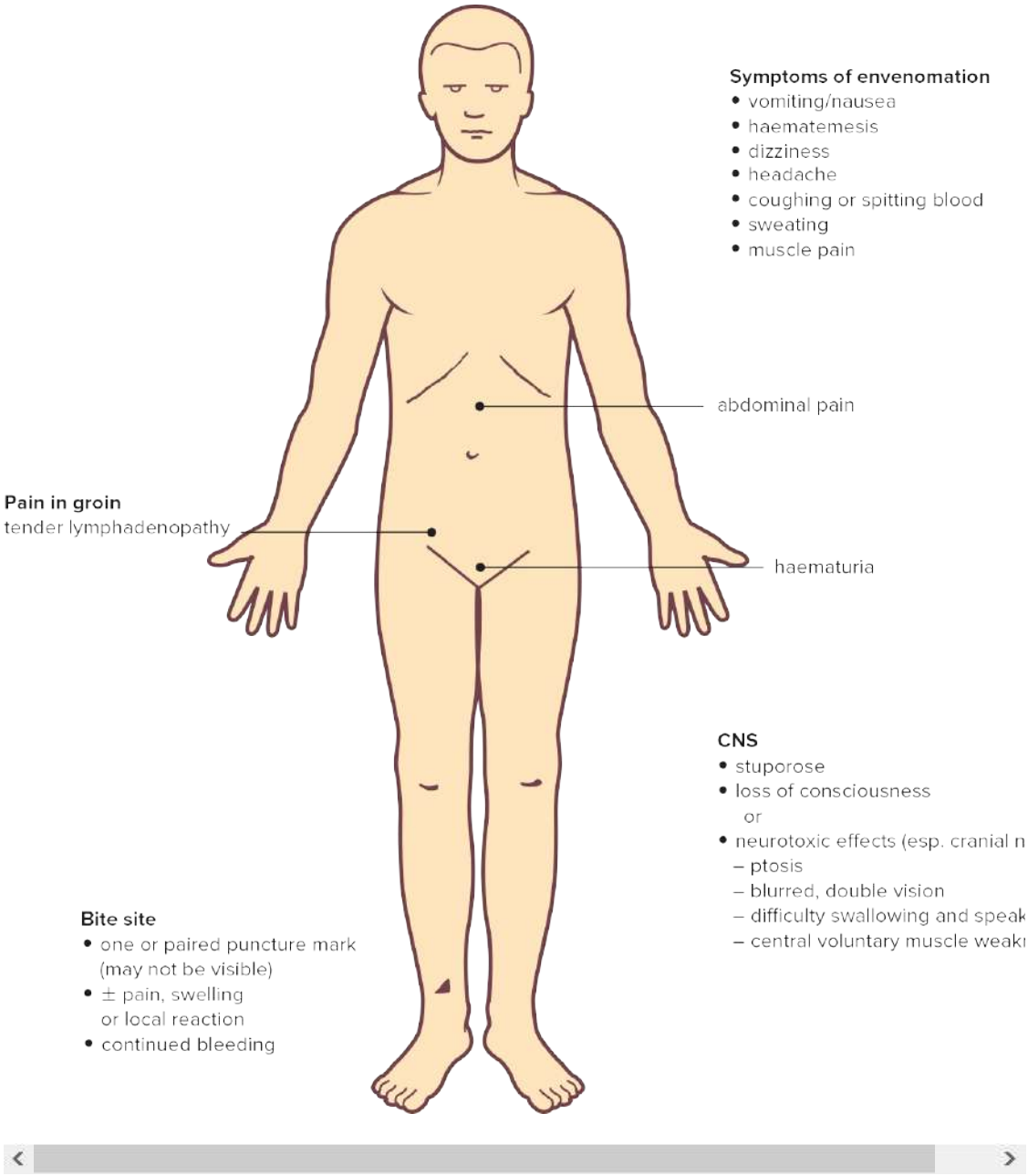


FIGURE 120.4 How to recognise snake bite envenomation

Investigations and observation

- Careful observations (e.g. vital signs, conscious state)
- Test all urine for blood and protein
- FBE, UEC, LFT
- Watch for coagulopathy (e.g. spitting and coughing blood, bleeding from wounds/IV site,

haematuria)

- Serial whole blood clotting time (a plain glass tube): normal <5–8 min, >15 min significant
- Coagulation screen—APTT and PT or INR
- Venom detection kit: wound site (best) or urine (less reliable, delayed positive)

Treatment of envenomation

- Reassure patient at all times
- Set up a slow IV infusion of N saline
- Do not give antivenom unless clinical signs of envenomation or biochemical signs (e.g. positive urine, or abnormal clotting profile)
- If antivenom is indicated, one vial is usually sufficient
- Dilute the specific antivenom (1 in 10 in N saline) and infuse slowly over 30 minutes via the tubing of the saline solution
- Have adrenaline, antihistamines, oxygen and steroids on standby
- Monitor vital signs
- Provide basic life support as necessary

Note: The use of prophylactic adrenaline is controversial and some authorities reserve it for a reaction to the antivenom. It is best avoided with brown snake envenomation and with coagulopathy.

Spider bites[5]

The toxin of most species of spider causes only localised pain, redness and swelling, but the toxin of some, notably the deadly Sydney funnel-web spider (*Atrax robustus*), can be rapidly fatal.

Rule: a bite from a large black spider should be managed initially as for a funnel-web spider, particularly within a 160 km radius of Sydney.

First aid

- Sydney funnel-web: as for snake bites
- Other spiders: apply an ice pack, do not bandage

Treatment

Sydney funnel-web

Signs of envenomation (in order):

- muscle fasciculation—limb → tongue/lip
- marked salivation or lacrimation
- piloerection
- dyspnoea
- neurological symptoms (e.g. disorientation, coma)

Treatment of envenomation

- specific antivenom (usually 4–8 vials)
- admit to a hospital critical care area
- resuscitation and other supportive measures

Other spider bites

The toxins of most species of spiders cause only localised symptoms, but the venom of a select few, namely the red-back spider of Australia (*Latrodectus hasseltii*) and the related black widow spider of the US (*Latrodectus mactans*), can cause envenomation. This is rarely fatal but is more serious in the young, the frail and the elderly. The bite wounds are prone to infection.

Treatment of envenomation; ice, analgesia.

- Antivenom exists, but rarely needed. Given IM or IV.

Bee stings

First aid

1. Scrape the sting off sideways with a fingernail or knife blade. Do not squeeze it with the fingertips.
2. Apply 20% aluminium sulphate solution (Stingose) or methylated spirits.
3. Apply ice to the site.
4. Rest and elevate the limb that has been stung.

If anaphylaxis, treat as outlined earlier.

Preventive measures (if hypersensitive)

- Avoid bees (and wasps) if possible.
- Immunotherapy to honey bee (or wasp) venom. There is no cross-allergy between the honey bee, the ‘yellow jacket’ (*Vespula*) wasp and the paper wasp. For the bee, use pure venom antigen. However, *Vespula* requires specific hyposensitisation.
- Immunotherapy should be offered to those:

  with a history of asthma who have had a single severe reaction to a bee sting

  who have had a minimum of three stings with serial crescendo reactions

  occupationally exposed who manifest severe reactions

  with elevated venom-specific IgE (RAST) antibodies, or positive venom prick tests

Centipede and scorpion bites

The main symptom is pain, which can be very severe and prolonged. Scorpions found in Australasia usually cause only minor systemic symptoms.

First aid[10]

1. Apply local heat (e.g. hot water with ammonia—household bleach).
2. Clean site.
3. Local anaesthetic (e.g. 1–2 mL of 1% lignocaine) infiltrated around the site.
4. Consider opioids—morphine or fentanyl—for pain.
5. Check tetanus immunisation status.

Box jellyfish or sea wasp (*Chironex fleckeri*)[5]

This is the most dangerous jellyfish in Australian waters and has been responsible for at least 80 extremely painful and sudden deaths.[11] Death can occur in minutes due to cardiopulmonary failure, especially in children. The jellyfish has up to 180 metres of stinging tentacles. The sting gives a ‘frosted ladder’ appearance. The jellyfish is limited to tropical waters north of the Tropic of Capricorn (Exmouth in west to Gladstone in east) and is found in coastal waters during the summer.

Prevention

- Avoid swimming, paddling and wading in ‘jellyfish alert’ areas in unsafe months.
- Otherwise, use a ‘stinger suit’.

Treatment[4]

- Remove the victim from the water to prevent drowning.
- Immediately remove tentacles with tweezers, stick or gloved/clothed hand.
- Check respiration and pulse.
- Start immediate cardiopulmonary resuscitation if necessary.
- Pour vinegar liberally (up to 2 L) over sting site and surrounding area.
- Use a cold pack for small stings and ice massage for large areas.
- Gain IV access; give oxygen and up to 5 mL inotropes if necessary.
- Give box jellyfish antivenom by IV injection for major stings (may need at least 6 ampoules if patient remains in cardiac arrest).
- Provide pain relief if required (ice, lignocaine and analgesics—fentanyl or morphine).
- The use of IV magnesium is supported only by anecdotal evidence.[12]

Note: A delayed reaction can occur—the stings can cause pain after weeks (oral steroids are used).

Do not use pressure immobilisation bandaging for *Chironex* stings.

Irukandji syndrome[5]

This is caused by *Carukia barnesi*, a tiny box jellyfish that can penetrate safety nets for *Chironex*, and possibly other species of box jellyfish. Initially a mild sting with a delayed severe syndrome (usually after 30 minutes):

- severe generalised back, abdominal and muscle pain and muscle cramps
- chest pain, sweating and anxiety
- anxiety, restlessness, ‘impending doom’ feeling
- headache, nausea, vomiting
- tachycardia, hypertension

There is no specific first aid or antivenom but resuscitation measures may be needed as death can occur from pulmonary oedema or cardiac arrest. Measures include morphine or fentanyl 5 mg IV every 5 minutes as required, blood pressure control (e.g. phentolamine, CPAP and oxygen for pulmonary oedema) and possibly IV magnesium (minimal evidence for this).[12]

Common jellyfish stings[5]

These include bluebottle (*Physalia* species) and others that cause intense local pain (up to an hour or more) and linear skin redness. Systemic effects are uncommon.

Treatment

- Wash the sting site with sea water.
- Remove any tentacles with gloved hands or water.
- Immerse affected site in hot water at 45°C for 20 minutes. This is quite hot, so check tolerance to temperature on other limb.
- Vinegar is not helpful.

Stinging fish[5]

The sharp spines of the stinging fish have venom glands that can produce severe pain if they spike or even graze the skin. The best known of these is the stonefish. Others include bullrout, catfish, sea-urchins and crown of thorns (may need an X-ray). The toxin is usually heat-sensitive. Stingrays cause a gash wound with possible superinfection.

Envenomation

- Intense pain
- Localised swelling
- Bluish discolouration

Treatment

- Clean the wound—consider exploration.
- Bathe or immerse the affected part in very warm to hot (45°C, not scalding) water—this may give instant relief.[5]
- Give simple analgesics.
- If pain persists, give a local injection/infiltration of lignocaine 1% or even a regional block. If still persisting, consider morphine IV or fentanyl IV.

- A specific stonefish antivenom is available. Best given IV by slow infusion in CCU.

Mollusc bite (blue-ringed octopus, cone shell)[5]

Mollusc venoms usually cause numbness or paraesthesia but can be rapidly fatal if prolonged muscular weakness leads to respiratory paralysis.

Treatment

- Apply compression bandage to bite site (usually hand/arm).
- Immobilise the limb.
- Arrange transport (preferably by ambulance) to a medical facility.
- Observe for respiratory paralysis—ensure adequate DRABC.

Other bites and stings

Bites from the sandfly (biting midge) have anecdotally been said to be prevented by oral thiamine. No published evidence supports this theory.

For these, and bites from ants, wasps and mosquitoes:

First aid

1. Wash the site with large quantities of cool water.
2. Apply vinegar (liberal amount) or aluminium sulphate 20% solution (Stingose) to the wound for about 30 seconds (scant evidence).
3. Apply ice for several minutes.
4. Use soothing anti-itch cream or 5% lignocaine cream/ointment if very painful.

Medication is not usually necessary unless an acute allergic reaction develops.

The embedded tick[5]

Some species of ticks can be very dangerous to human beings, especially to children. In Australia, tick paralysis is confined to the eastern seaboard. Be careful in children 1–5 years—ticks are usually found in the scalp behind the ears. As it is impossible to distinguish between dangerous and non-dangerous ticks, early removal is mandatory. The tick should be totally removed—do not leave the mouthparts behind. Do not attempt to grab the tick by its body and tug. This is rarely successful in dislodging the tick, and may inject more toxin into the host.

As an office procedure, many practitioners grasp the tick’s head as close to the skin as possible

with fine forceps or tweezers, and pull the tick out sideways with a sharp rotatory action. This is acceptable, but not as effective as the methods described here.

First aid outdoor removal method

- Loop a strong thin thread (as a half-hitch lasso) around the tick's head as close to the skin as possible, and pull sharply with a twisting motion. Suitable materials include strong silk sutures or dental floss.
- A pyrethrin-based spray is often used.

Office procedure

- Infiltrate a small amount of local anaesthetic into the skin around the site of embedment.
- With a number 11 or 15 scalpel blade, make the necessary very small excision, including the mouthparts of the tick, to ensure total removal (see FIG. 120.5).

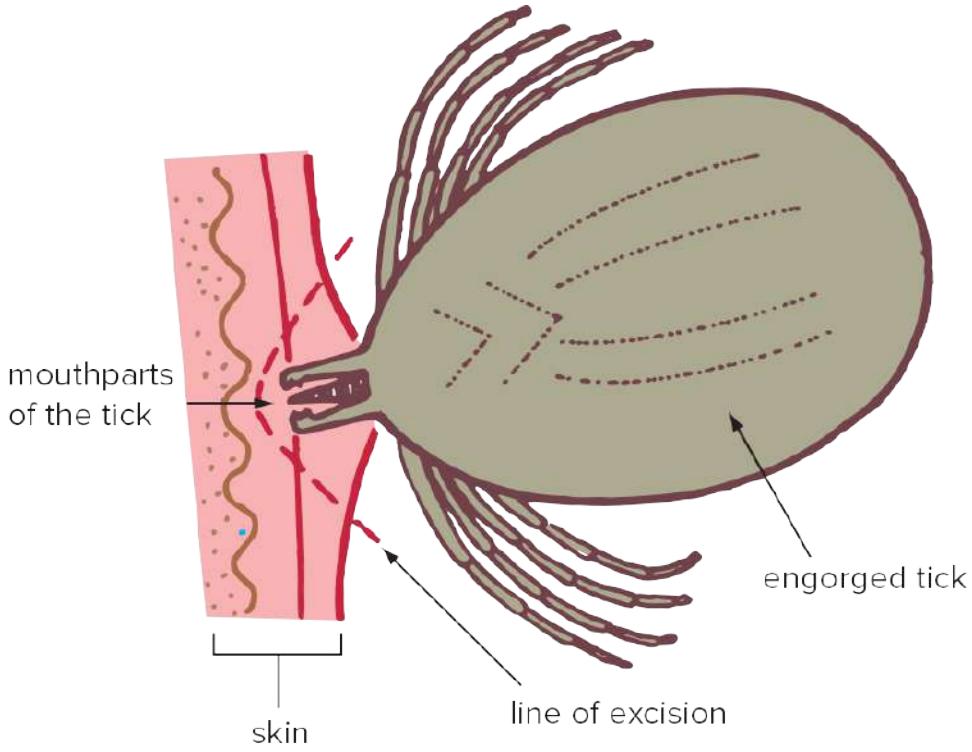


FIGURE 120.5 Removing the embedded tick

- If the tick body tears away, the skin containing the mouthparts is easily excised using a 2 or 3 mm punch biopsy blade.
- The small defect can usually be closed with a bandaid (or Steri-Strips), rather than a suture.

- Careful observation is needed after removal.

Human bites and animal bites

These bites can cause problems of suppurative infection and management in general. They are outlined in CHAPTER 123 .

Vital emergency skills

Cardiopulmonary resuscitation

Cardiopulmonary arrest (CPA)

It is essential that all doctors are familiar with the protocol for instituting basic life support in such an eventuality. People with cardiac conditions visit our offices daily and the potential for sudden collapse, including a cardiac arrest, is ever present. About 75% of arrests are due to ventricular fibrillation and more than 75% of victims have severe coronary artery disease.[13] After 3 minutes of CPA (unconsciousness, no pulse, no respiration) there is an increasing risk of permanent cerebral dysfunction.

Important causes of sudden death are outlined in TABLE 120.1 .[14]

Table 120.1 Causes of sudden death

Cardiac arrhythmias:
- ventricular fibrillation (75%)
- ventricular tachycardia
- torsade de pointes VT (?drugs)
- sick sinus syndrome
- severe bradycardia

Sudden pump failure:
- acute myocardial infarction
- cardiomyopathy

Cardiovascular rupture:
- myocardial rupture
- dissecting aneurysm of aorta
- subarachnoid haemorrhage

Acute circulatory obstruction:
- pulmonary embolism

Others:

- aortic stenosis
- pulmonary hypertension
- mitral valve prolapse
- electrolyte abnormalities
- glue sniffing

The ABC basic life support for cardiac arrest should be followed, but DRSABCD is best. The S (send for help) should include sending for a defibrillator, because the final D (defibrillation) is urgent—the outcome appears to be directly related to the speed of defibrillation. The 'chain of survival' principle for a victim of out-of-hospital cardiac arrest is presented in FIGURE 120.6 .

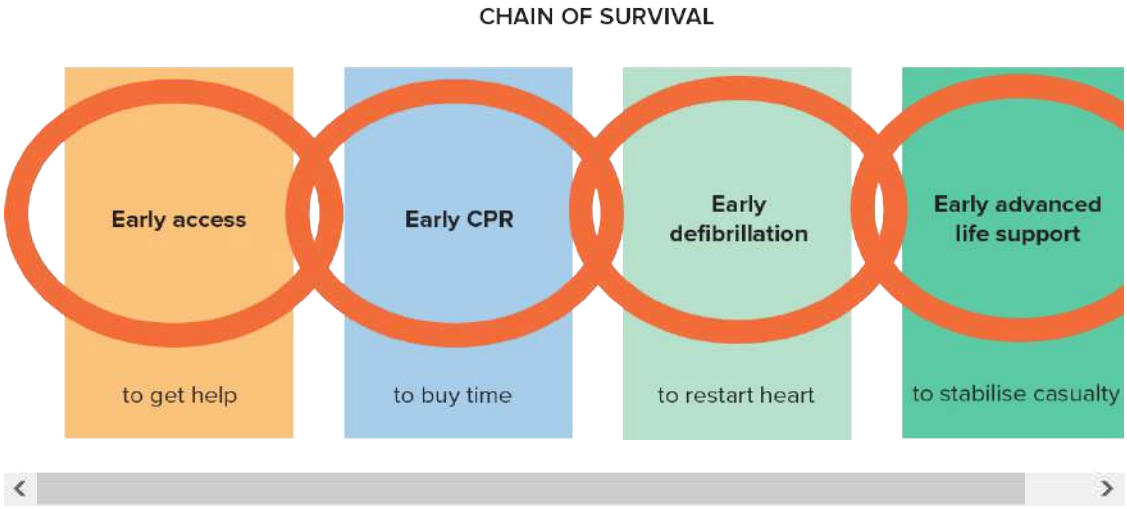


FIGURE 120.6 The chain of survival for a victim of out-of-hospital cardiac arrest

Basic life support[15]

FIGURE 120.7 represents a logical DRSABCD plan for the adult patient who collapses or is found apparently unconscious.

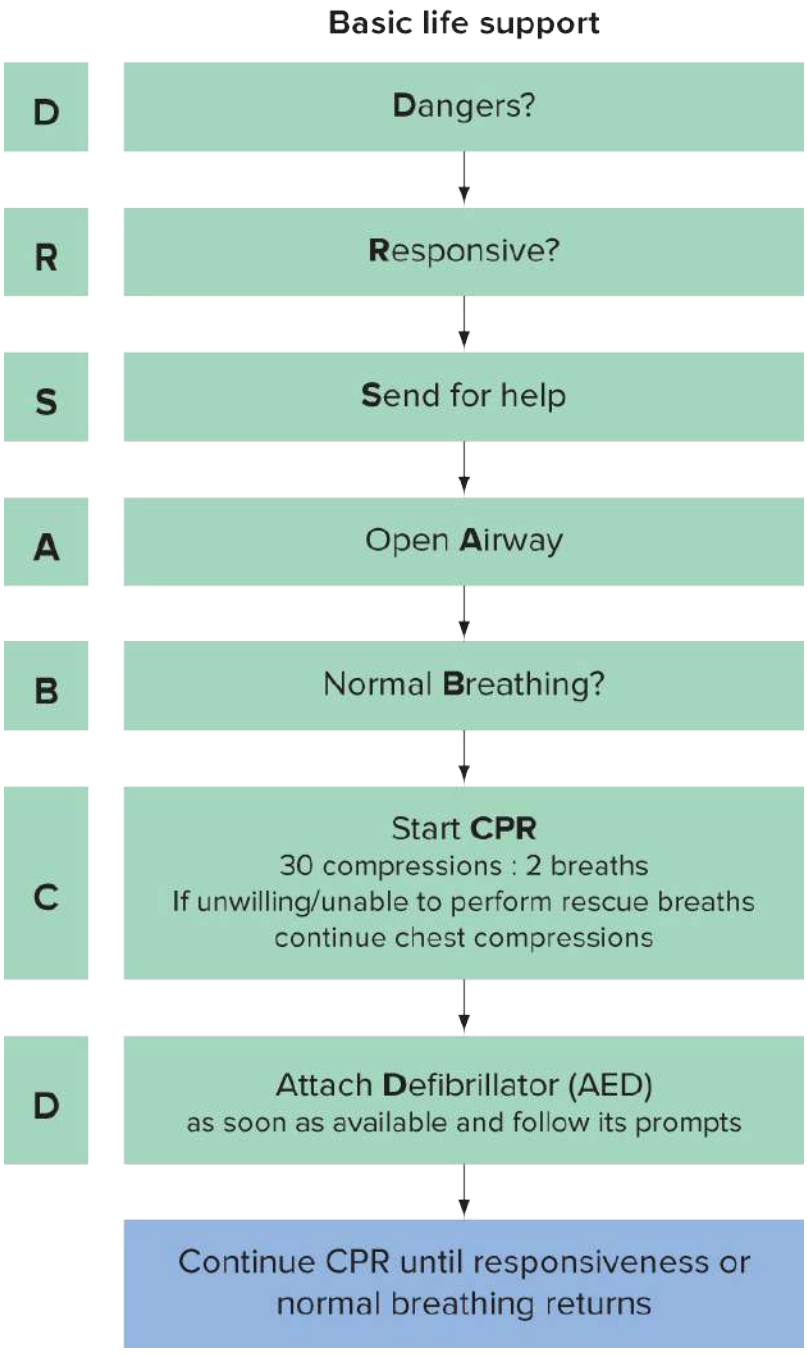


FIGURE 120.7 Basic life support algorithm

Source: Adapted from Australian Resuscitation Council guidelines https://resus.org.au/guidelines/

1. Shake and shout at the patient.
2. Check breathing. There is a high incidence of abnormal gasping (agonal gasps) after cardiac arrest, which still requires CPR.

3. If breathing, check pulse (feel carotid adjacent to thyroid cartilage). If unresponsive and not breathing, do not delay CPR to check for a pulse.

4. Call for help (if unresponsive). Ring 000 (112 also works on mobile services—the international standard number).

5. Finger sweep oropharynx (clear it).

6. Place victim on back on firm surface.

7. Thump precordium (if arrest witnessed).

8. Tilt head back (cervical caution if traumatic/unknown cause).

9. Lift chin (use airway if available).

0. Commence basic life support:

    rescue breaths (RBs)—2 strong breaths

    external chest compressions (no pause)—30 compressions

    continue alternating 30 compressions (at rate of 100 beats/min) with 2 strong breaths

Attach automated external defibrillator (if necessary).

Note: The ratio of 30:2 with one or two rescuers is favoured. Some authorities recommend continuous compression only. This is most appropriate for the untrained resuscitator.

The basic schedule for cardiopulmonary resuscitation is presented in TABLE 120.2 .

Table 120.2 Basic schedule for CPR

	Infant <1 year	Child	Adult
Compression (rate per min)	100–120	100–120	100–120
Depth of compression (cm)	4	5	5–6
Position of compression	Centre sternum	Centre sternum	4 cm above xiphisternum
Method	2 fingers	1 hand	2 hands
Ventilation (rate per minute)	20	16	6–10

Head tilt	Nil	Mid	Full

Note: Check the International Liaison Committee on Resuscitation (ILCOR) guidelines www.ilcor.org.

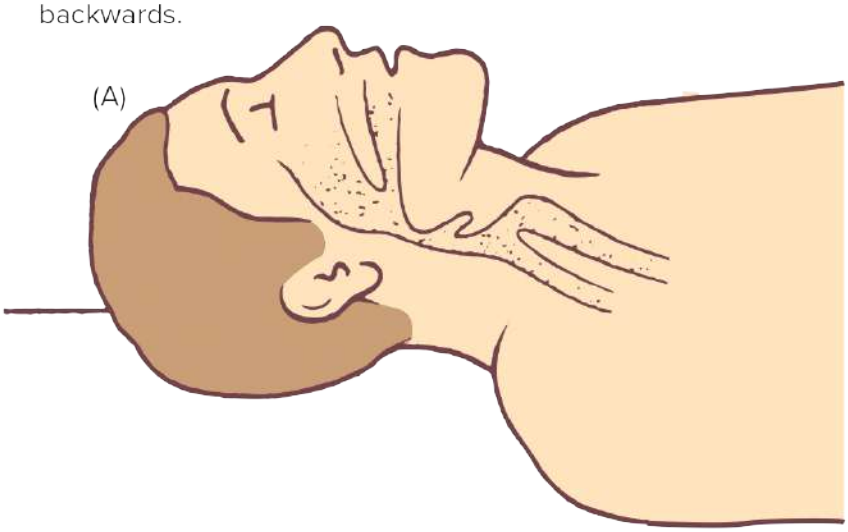
Method of rescue breathing/ventilation

With the victim's head in the 'sniffing the morning air' position (head tilted back and chin pulled forward) the rescuer takes a deep breath and seals his or her lips around the mouth (nose is occasional alternative) of the victim. Pinch the victim's nose if using mouth-to-mouth resuscitation. Give two full breaths and check for chest expansion (see FIG. 120.8 and FIG. 120.9). If the chest does not move easily, the lip-seal has failed or obstruction is present. If available, a sucker should be used to clear the oropharynx. Firmly fitting dentures should be left in place as they make artificial respiration easier. A Resuscitube or Laerdal pocket mask (which should be in the doctor's bag) is ideal for expired air resuscitation (EAR); it saves mouth-to-mouth contact and probably improves the efficacy of ventilations.

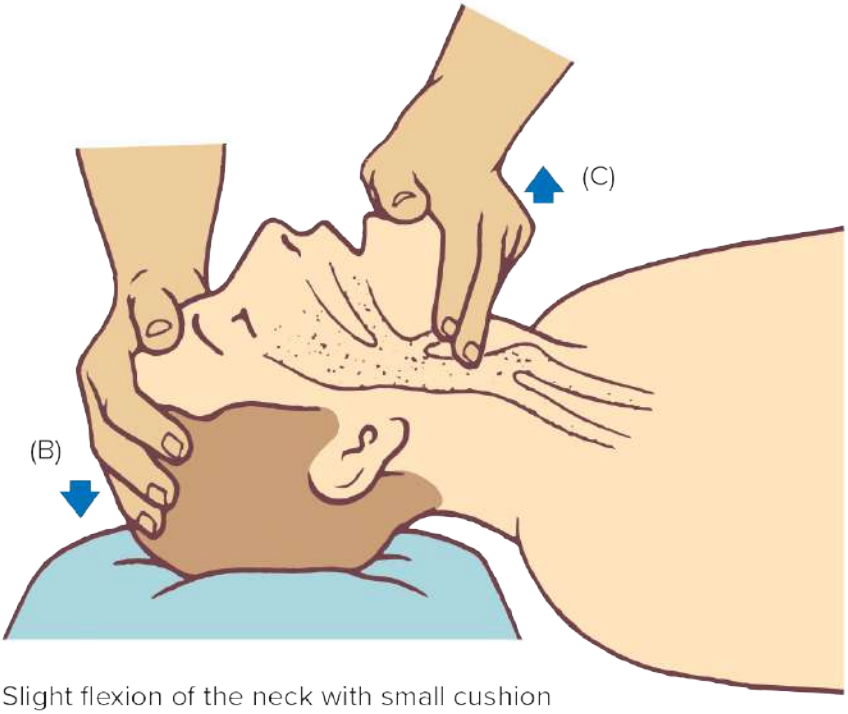
For optimal airway patency:

1 Clear foreign matter from mouth—use finger sweep from airway: blow between shoulder blades; consider a Heimlich manoeuvre

2 Lay patient supine on flat, firm surface (A). Note how the soft tissue of the pharynx can obstruct the airway by falling backwards.



3 In order to overcome this, apply a head tilt (B) plus a chin lift (C) or jaw thrust manoeuvre.
(*Note:* Avoid excessive movement of the neck if spinal injury is suspected, but clearing the airway has first priority.)



Slight flexion of the neck with small cushion

FIGURE 120.8 Basic life support: A = Airway

Rescue breathing:

1. Give two full breaths.
2. Observe rise of chest, not of abdomen.
3. Look, listen and feel for exhalation.
4. Check the carotid pulse.
5. If no pulse, commence full cardiopulmonary resuscitation.

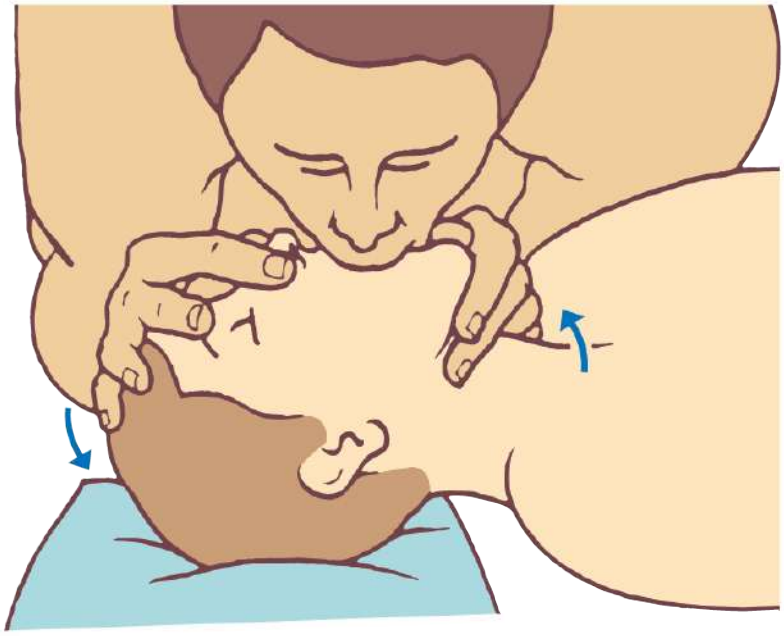


FIGURE 120.9 Basic life support: B = Breathing

External chest compression

Evidence supports the need to maintain cardiac compression following a cardiac arrest. This should be commenced on any person who is not responsive and not breathing normally.

Compressions are safely performed by finding the xiphisternal notch then placing the broad heel of one hand over the lower half of the sternum (in adults) with the heel of the second hand placed over the first with the fingers interlocked. Remember to keep the arms and elbows straight as the sternum is rhythmically depressed for 5–6 cm or one-third of chest length. Try to keep to this position as ‘wandering’ causes fractured ribs or worse. The fingers must be kept off the chest.

Compressions should be smooth, regular and uninterrupted. The compressions should ideally produce an impulse in the femoral pulse. Another person can check for carotid or femoral pulsation during CPR, and check pupil size.

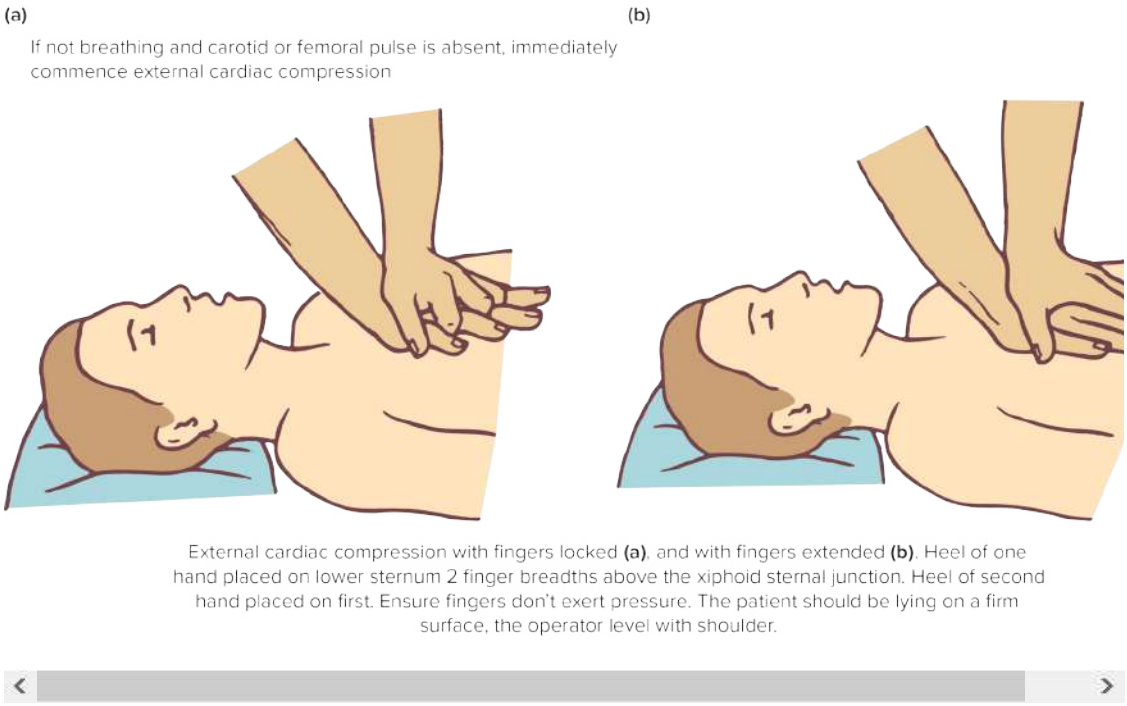


FIGURE 120.10 Basic life support: C = Circulation

Maintenance of CPR

Consider ceasing CPR if there is no improvement in 30 minutes. Exceptions where prolonged CPR can be successful are cold water drowning, electrical injuries, marine envenomation, snake bite and certain poisonings (e.g. cyanide and organophosphate).

Advanced cardiac life support

Advanced life support depends on the availability of skilled personnel and appropriate equipment.

Optimal initial support involves:

- airway management
- endotracheal intubation (otherwise bag and oxygen)
- ECG monitoring
- intravenous access (large peripheral or central vein)

Optimal initial therapy involves:

- defibrillation

- oxygen
- cardioactive drugs, especially adrenaline

If an ECG recording is unavailable the best course of action is:

- defibrillation
- if unsuccessful, adrenaline IV; repeat every 3 minutes

Consider antiarrhythmic agents—amiodarone, lignocaine, magnesium.

Advanced life support: defibrillation[15,16]

- Minimise interruptions to chest compressions.
- Give a single shock instead of stacked shocks (single shock strategy) for ventricular fibrillation/pulseless ventricular tachycardia.
- Where the arrest is witnessed by a health care professional and a manual defibrillator is available, then up to three shocks may be given (stacked shock strategy) at the first defibrillation attempt.
- Biphasic defibrillation is usually set at 360 joules and monophasic at 200 joules, unless machine is programmed differently.
- After each defibrillation attempt, give 2 minutes of CPR before checking rhythm and pulse.

For defibrillation, two paddles or pads should be placed correctly on the chest wall, using one of two positions:

- one to right of upper sternum (under right clavicle) and the other over the apex of the heart (see FIG. 120.11)
- one over anterior wall of chest and the other under tip of left scapula

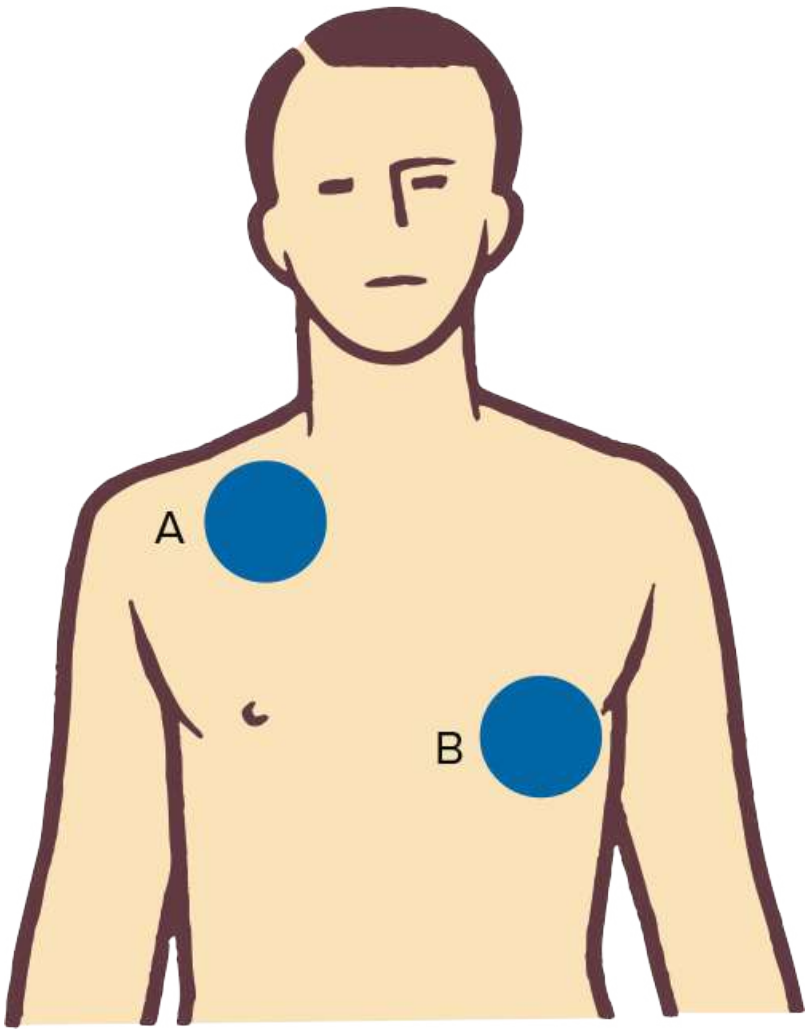


FIGURE 120.11 Standard position of two paddles for defibrillation: A under right clavicle, B under left breast

Hairs on the chest should be shaved (if doesn't delay) to accommodate the paddles.

Urgent intravenous access

It is preferable to aim for transcutaneous cannulation of veins initially so a peripheral line should be introduced into a vein in the cubital fossa. Several lines may be required with massive blood loss.

Alternative routes:

- central venous cannulation: most doctors should be able to cannulate the external jugular vein with a standard cannula, aiming towards the toes
- peripheral venous cutdown

- intraosseous infusion (see CHAPTER 89)

Disaster medicine

The emergence of COVID-19 in 2020 highlighted the importance of preparing for and managing disasters. Although traditionally most resources are concentrated on hospitals and specialised medical or public health teams, in practice much of the work falls to general practitioners.

The frequency of disasters and the number of people affected by them are increasing. This is due to a number of factors, including global movement of populations, human habitation in more disaster-prone areas and the effects of anthropogenic transformation of the earth's surface—including disease vectors and climate change.

Disaster management is a crucial, rapidly expanding discipline in the world of general practice. 'The World Medical Association recommends that disaster medicine training be included in the curricula of university and postgraduate courses in medicine'.[17]

Disaster situations 'from the medical standpoint … are characterised by an acute and unforeseen *imbalance* between the capacity and *resources* of the medical profession and the *needs* of survivors who are injured, or whose health is threatened, over a given period of time'.[18]

Disaster management differs relative to the time of disaster impact, with four phases to be considered: prevention, preparedness (disaster impact), response and recovery (PPRR). Management also varies depending on the hazard that caused the disaster: for example, pandemic, bushfire, flood, heatwave, drought, nuclear accident or terrorism.

Key facts and checkpoints

- Preparedness:

  Evidence shows that preparedness helps in recovery and GPs should have a disaster plan and practise their plan.[19]

- Response:

  It is valuable for GPs to maintain an up-to-date vulnerable patients list.

  Maintenance of business-as-usual is the most crucial GP response. This may have to adapt during disasters: e.g. using alternative premises, telehealth.

- Recovery:

  May take days, weeks, months, years or decades.

  Most people recover with limited to no assistance, and the temptation to insist

upon psychological therapy for all should be resisted.

- Self-care:

  GPs could be considered a vulnerable group during a disaster in their community. Self care must be a priority.

A systematic approach

It is useful to consider a GP's role according to the four phases of disaster, PPRR. In order to do this a general practice would benefit from an emergency response plan or disaster plan.[20]

Response

During the acute event, emergency response systems, including fire, ambulance and police services, work with GPs to provide the most benefit, and to minimise the risk of potential harm to themselves, the patients or other responders. It is important that GPs understand these systems and avoid duplications and gaps in patient care.

In an isolated area, the GP may be the only respondent for a considerable time. In this situation triage and improvisation are important.

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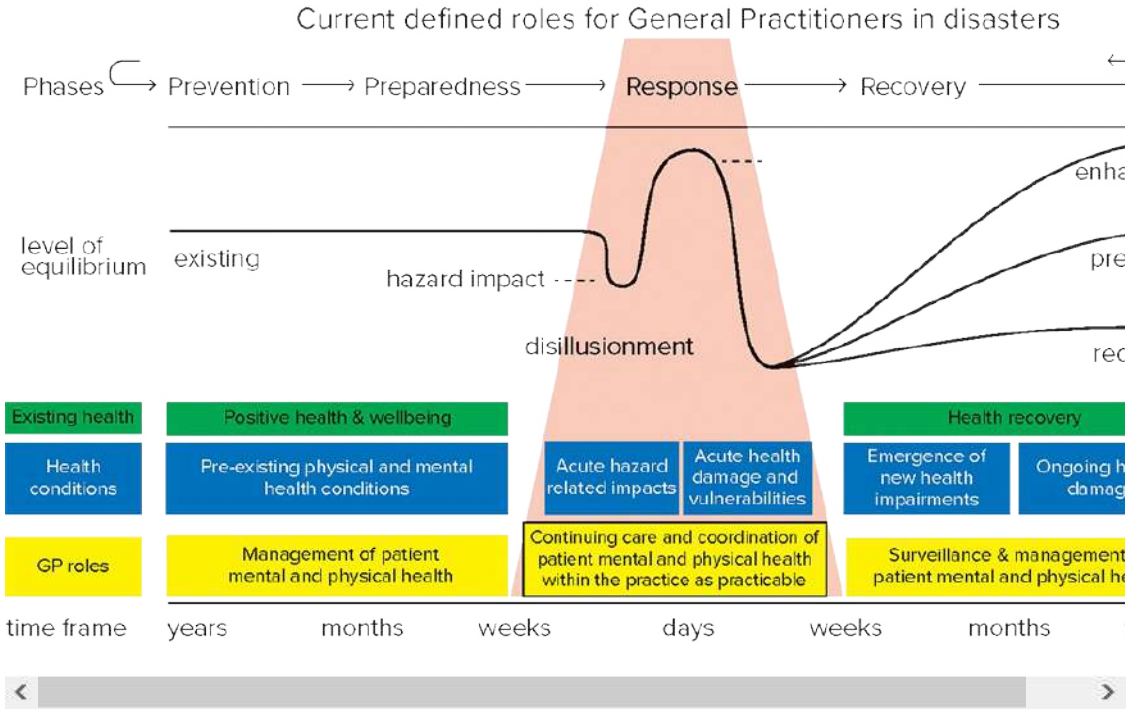

FIGURE 120.12 Phases of disaster: a timeline of disaster over time shows GP roles across the phases of disaster and prevention/preparedness/response/recovery (PPRR)

Source: Reproduced with permission from Burns P, Douglas K, & Hu W. Primary care in disasters: opportunity to address a hidden burden of health care. Med J Aust 2019; (7): 210.

Recovery

In the first weeks

There is distress, denial and disbelief, lasting days to weeks. Most people will recover. Some will require minimal support; a few will require greater support.

In order to reduce distress due to a traumatic experience, psychological first aid (PFA) aims to improve short-term and long-term functioning. It is evidence-informed and endorsed by the World Health Organization (WHO) and the Australian Red Cross.[21] Such first aid opens the door for some to go on to more significant counselling, but this more formal therapy should not be 'thrust upon' everyone by well-meaning authorities.

Psychological first aid (PFA)

PFA requires no psycho-education. It involves:

- ensuring individuals are physically safe, with shelter and basic human needs met
- comforting and quietly listening to what individuals want to say. It is *not* asking individuals to talk about what happened, or telling them it is good for them to talk about it
- connecting individuals with loved ones or friends
- orientating individuals to goals to improve the sense of efficacy—small, practical, achievable tasks assist the return to normal routine[21]

GPs who are already part of the community have a significant head start when it comes to offering PFA. PFA is not *critical incident stress debriefing*, which has not been shown to be useful, and may even be harmful to some individuals.

In the following weeks, months and years

As mentioned above, most patients and families will recover unassisted. Physical and mental health effects of a disaster may coexist in the aftermath.[21] A key element is surveillance for deterioration of chronic conditions, emerging mental and physical health conditions,[22] and increased substance use. The economic fallout from large-scale disasters such as COVID-19 and bushfires may have substantial longer-term impact via the 'social determinants of health', such as employment, educational attendance and societal factors affecting mental health.

Supporting children is best done by supporting their parents[23] and by assisting them to return to a normal routine.[24] Recurrent viewing of the event on media is not recommended.

Disasters divide lives in two; for some the effects may endure for decades, or span generations,

depending on the characteristics of the individuals, the families and the disaster.[25]

The doctor’s bag and other emergency equipment

GPs who perform home visits and nursing home visits require the traditional doctor’s bag that includes the basic tools of trade: drugs (including those for emergency use), stationery and various miscellaneous items. Country doctors will by necessity use their bag for more emergency home and roadside calls. These recommended contents are simply a guide for cross-checking.

Essential requirements for the bag

- Sturdiness
- Lockable (e.g. combination lock)
- Ready interior access
- Uncluttered
- Disposable single-use items
- Light, portable equipment
- Regular checks to ensure non-expired drugs
- Storage in a cool place (not boot of car)

Traditionalists may prefer the Gladstone bag, whose style confers a sense of time-honoured assurance. Pragmatists may opt for a fishing tackle box: far from elegant, but the sight of those organised compartments emerging during a crisis can calm the nerves.

Paperwork and reference material

The laptop computer replaces most of the traditional stationery items such as letterhead, certificates, referral forms and Medicare slips. However, pen and paper remains essential, and a record book is required for dispensing S8 medications.

Phone apps for emergencies are plentiful, although drug dosage and emergency regimens are ideally pre-loaded onto the phone or laptop rather than relying on variable internet speeds and mobile phone services at the emergency location. This also applies to local street maps (Google map regions can be saved permanently) and emergency phone numbers.

Equipment

- Sphygmomanometer (aneroid)
- Stethoscope
- Pulse oximeter
- Diagnostic set (auriscope + ophthalmoscope)
- Tongue depressors
- Tourniquet
- Small needle disposal bottle
- Scissors
- Syringes 2, 5, 10 mL
- Needles 19, 21, 23, 25 gauge
- Butterfly needles
- IV cannulae 16, 18, 20 gauge
- Alcohol swabs
- Micropore tape
- Thermometer
- Spacer (asthma drugs)
- Artery forceps
- Urine testing sticks
- Urine specimen containers
- Skin swabs, throat swabs
- LED torch
- Patellar hammer
- Oral airway (e.g. Revivatube, Resuscitube—FIG. 120.13, Guedel)
- Laerdal pocket mask (FIG. 120.14)
- Scalpel

- Examination gloves

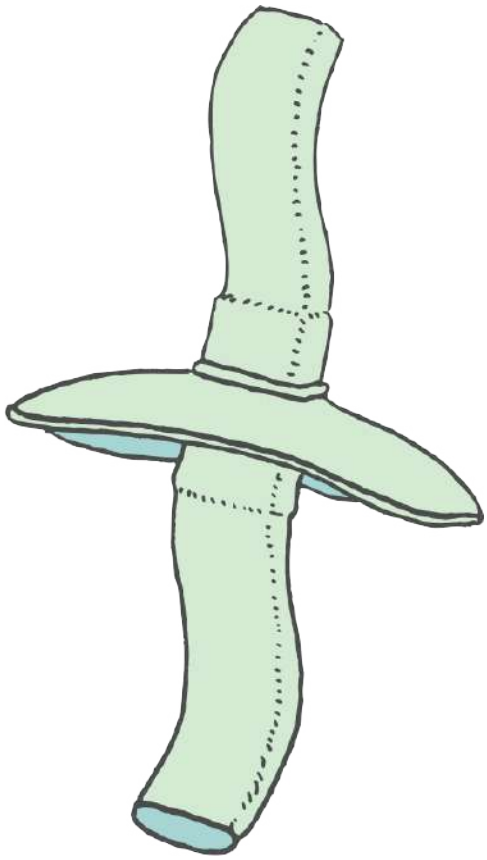

FIGURE 120.13 The two-way Resuscitube

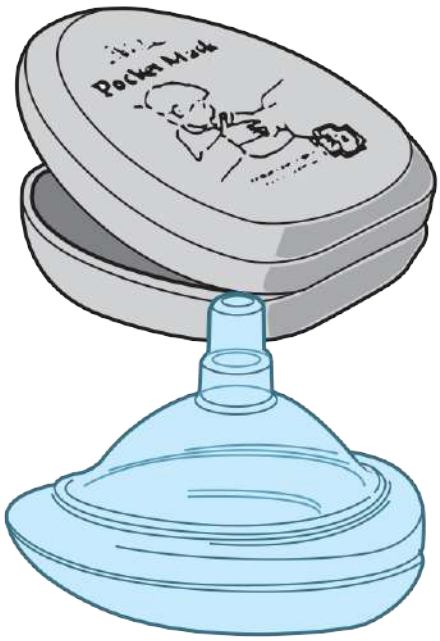

FIGURE 120.14 The Laerdal pocket mask

Drugs

Drugs (oral)

- Samples of commonly used:
    - analgesics
    - antibiotics
    - antidiarrhoeal agents
    - anti-emetics
    - antihistamines
    - sedatives
- Soluble aspirin (for myocardial infarction)
- A triptan (for migraine)

Drugs (sprays)

- Glyceryl trinitrate spray (tablets deteriorate)
- Salbutamol MDI

Drugs (topical)

- Anaesthetic eyedrops
- Combination ointment (e.g. Otocomb otic—antibacterial/antifungal/steroid)

Drugs (injectable)

Refer to TABLE 120.3.

Table 120.3 Drugs available on PBS (except those marked #) www.pbs.gov.au/browse/doctorsbag[26,27]

Drug	Presentation	Indications
Adrenaline*	1 mg/mL (1:1000)	Hypersensitivity reactions and anaphylactic shock, asthma†, ventricular asystole, croup, ventricular fibrillation (to assist CPR)
Atropine sulphate	0.6 mg/1 mL	Bradycardia (after myocardial infarction), 2nd or 3rd degree heart block, ureteric colic†, organophosphate poisoning
Benztropine*	Cogentin 2 mg/2 mL	Acute dystonic reactions
Benzylpenicillin*	3 g with 10 mL water	Meningococcaemia, pneumonia (adults)
Ceftriaxone#	2 g powder with 10 mL solvent	Meningococcaemia, septicaemia
Dexamethasone	4 mg/1 mL	Severe asthma (esp. elderly), moderate to severe croup, anaphylaxis, acute Addisonian crisis
Frusemide*	Lasix 20 mg/2 mL	Left ventricular failure, acute pulmonary oedema
Glucagon*	1 mg + 1 mL solvent	Hypoglycaemia (insulin or oral therapy)

Glucose[#]	50% (500 mg/mL in 50 mL)	Hypoglycaemia
Glyceryl trinitrate*	Spray 400 mcg/dose	Acute coronary syndrome
Haloperidol*	5 mg/1 mL	Psychiatric emergencies such as severe agitation, psychoses; migraine
Hydrocortisone sodium succinate*	100 or 250 mg/2 mL	Anaphylaxis, severe asthma, Addisonian crisis, thyrotoxic crisis, acute allergies
Methoxyflurane	3 mL pack with inhaler	Analgesia for acute pain
Midazolam	5 mg/1 mL	Status epilepticus and other convulsions such as eclampsia, sedation in acute anxiety and severe tension headache, psychiatric emergencies including acute alcohol withdrawal
Metoclopramide* or	Maxolon 10 mg/2 mL	Severe vomiting (e.g. Ménière syndrome, gastritis), acute labyrinthitis, migraine
Prochlorperazine	Stemetil 12.5 mg/mL	
Morphine sulphate*	15 mg/1 mL or 30 mg/1 mL	Acute pulmonary oedema, relief of severe pain (e.g. cardiac pain, colic)
Naloxone (more than one ampoule)*	Narcan 0.4 mg/mL	Opiate respiratory depression
Promethazine*	Phenergan 50 mg/2 mL	Acute allergic conditions (see FIG. 120.15), anti-emetic[†]
Salbutamol*	MD inhaler and/or nebuliser solution	Bronchial asthma, other bronchospasm
Tramadol	100 mg/2 mL	Moderate to severe pain

**Essential drugs*

†May be useful as an alternative drug

#Drugs not supplied as PBS doctor's bag items

All drugs listed are injectable except salbutamol inhaler

Note: The author recommends the 'Minijet' syringe packs for ideal emergency use. The range includes naloxone 5 mL, aminophylline, atropine, adrenaline, dextrose, lignocaine, isoprenaline, sodium bicarbonate

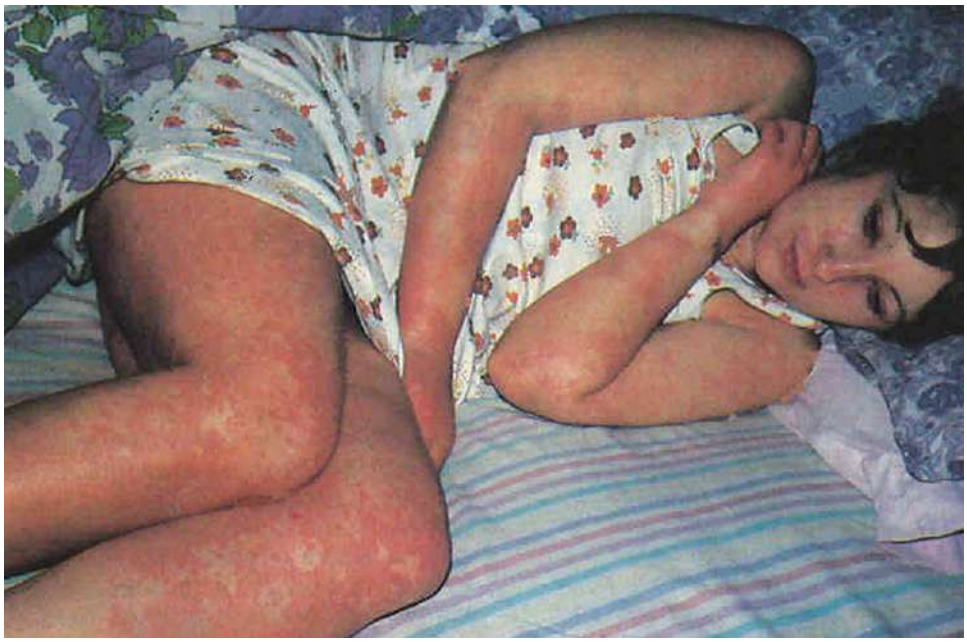

FIGURE 120.15 Home visit to a 25-year-old woman with acute urticaria caused by an upper respiratory viral infection. She was given 25 mg promethazine IM for severe pruritus and vomiting.

A study of 512 Queensland GPs revealed the range of emergency equipment and essential drugs carried.[28] The most common emergencies were acute asthma, psychiatric emergencies, seizures, hypoglycaemia, anaphylaxis, impaired consciousness, shock, poisoning and overdose. The drugs used most were adrenaline, benztropine, diazepam, glucagon, haloperidol, hydrocortisone, naloxone and salbutamol (inhaler).[29]

Drugs (suppositories)

- Indomethacin

The country doctor's bag

Rural doctors, especially in isolated areas, usually carry additional equipment in their motor

vehicles when called to the scene of an accident or other emergency. The equipment will vary according to geographic factors, the ambulance service and the special interests and enthusiasm of the practitioner.

Storage of drugs

The main issues are safe storage of opioid drugs, avoidance of overheating (keep <25°C), accessibility in emergencies and careful recording of Schedule 8 drugs in a register. Diphtheria/tetanus vaccines should be stored in a refrigerator.

Medical emergencies in general practice—a brief guide

Note: Using basic equipment and doctor's bag (mainly adult dosages)

Don't forget:

- **Secure IV line (may need rapid bolus N saline)**
- **Oxygen (where recommended)**
- **Measure vital signs**

Acute cardiogenic pulmonary oedema (ventricular failure)	Frusemide 40–80 mg IV (or twice usual dose) Glyceryl trinitrate spray 1 dose Consider (especially if chest pain)—morphine 2.5–5 mg IV
Acute anaphylaxis	Adrenaline 0.3–0.5 mg (1:1000) IM, repeat every 3 minutes as necessary or set up adrenaline infusion If no rapid improvement: • salbutamol inhalation • IV fluids • ?hydrocortisone/glucagon
Angio-oedema and acute	Promethazine 25 mg IM

urticaria	
Asthma	Salbutamol 6 (<6 years)–12 (adults) puffs by spacer Hydrocortisone 200 mg IV If severe: • adrenaline 0.3–0.5 mg 1:1000 IM or SC or infusion
Croup (severe)	Dexamethasone 0.15 mg/kg IM or prednisolone 1 mg/kg (o)
Epilepsy (seizure)	Midazolam 0.2 mg/kg IM
Opiate respiratory depression	Naloxone HCl 0.4 (or 0.2) mg IV + 0.4 mg IM
Acute coronary syndrome	Aspirin 300 mg soluble tab Glyceryl trinitrate spray or tablets (max. 3) If pain, morphine sulphate 2.5–5 mg IV + metoclopramide
Hypoglycaemia	Glucagon 1 mg/mL SC, IM or IV, then sweet drink or 20–30 mL 50% glucose IV
Migraine (severe)	Prochlorperazine 12.5 mg IV *or* Metoclopramide 10 mg IV ± dihydroergotamine IV or IM *or* Haloperidol 5 mg IM or IV
Cluster headache	100% oxygen 6 L/min for 15 minutes Metoclopramide 10 mg IV
Movement disorders (from antipsychotic medication)	Benztropine 1–2 mg IV or IM
Meningococcaemia	Benzylpenicillin 60 mg/kg IV
Ureteric colic	Morphine 10–15 mg IM or IV ± metoclopramide Indomethacin suppository
Vertigo (acute)	Prochlorperazine 12.5–25 mg IM or promethazine 25 mg IM
Vomiting	Prochlorperazine 12.5 mg IM or IV or metoclopramide 10 mg

Practice tips

- Check your doctor's bag every month for drugs that may be expired, damaged or in short supply (practice nurse can do this).[26]
- Replace any used drugs or materials the day after use.
- Always have your bag handy but don't carry it in the car in hot weather. It is best to be able to grab it from a safe, accessible spot when you leave for an emergency.
- Security is an issue. Drugs of addiction (tramadol and morphine) may be kept separate and then taken from their secure place when their use is anticipated (e.g. myocardial infarction, severe biliary or kidney colic). Tramadol appears to be a satisfactory alternative but beware of the serotonin syndrome.
- Keep a spare kit in your surgery if you or your assistants or locums perform a lot of emergency work.
- Familiarise yourself with the layout of your bag so that using it in urgent circumstances is efficient.
- Use a large intravenous cannula wherever possible if rapid infusion is required.

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