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121 Stroke and transient ischaemic attacks

A mild attack of apoplexy may be called death's retaining fee.

GILLES MÉNAGE (1613–1692)

Glossary of terms

Stroke A focal neurological deficit lasting longer than 24 hours caused by intracerebral haemorrhage or infarction.

Stroke in evolution An enlarging neurological deficit, presumably due to infarction, which increases over 24–48 hours.

Transient cerebral ischaemic attack (TIA) A transient episode of neurological dysfunction caused by focal brain, spinal cord or retinal ischaemia without infarction.[1]

Key facts and checkpoints

- Stroke is the second most common (10–12%) cause of death in the Western world. Most are ischaemic (thrombotic or embolic), but 15–20% are haemorrhagic.
- A stroke or TIA must be considered a medical emergency.
- One in 10 patients with a TIA is likely to have a stroke shortly afterwards—usually within 2 weeks and most within 48 hours. The risk is greatest if older than 60, symptoms last more than 10 minutes and there is weakness or a speech impediment with the TIA.[2]

- Clinical assessment (including neurological examination) investigations and treatment should be commenced quickly.
- The best approach to stroke management is aggressive attention to primary and secondary prevention.
- The main risk factors for stroke are atrial fibrillation, hypertension, smoking, age and diabetes.
- Cardiac disease can be a source of emboli.
- Most patients with a stroke or TIA require urgent imaging to find the cause and guide treatment.
- Ideally, patients should be referred to a stroke unit ASAP—within 3 hours.
- Order a CT or MRI scan on all patients with suspected TIAs and strokes (if not referring to a stroke unit): if normal, repeat within 7 days (CT scans unreliable after 7 days). Such imaging is required to differentiate between ischaemia and haemorrhage.
- Consider the possibility of a cryptogenic stroke,[1] especially from a patent foramen ovale (PFO) (in 20–25% of population and responsible for 50% of cryptogenic strokes) in relatively young people presenting with a stroke: this leads to paradoxical emboli (from veins to the brain). PFOs may be detected by echocardiography and sealed with a percutaneous closure device, although this is not routine.
- Consider the possibility of endocarditis if there is a heart murmur.
- Keep in mind atherosclerotic disease of the aortic arch as a source of cerebral embolism.
- The place of carotid endarterectomy for asymptomatic carotid stenosis remains controversial. It should be seriously considered if the stenosis is severe, the risk of surgery is low (3% risk of major stroke), the team has proven expertise and the patient is medically fit with a good life expectancy.[1]
- Carotid artery stenting for the treatment and prevention of stroke is an evolving procedure.

Modifiable risk factors for cerebrovascular disease[2]

Major: hypertension, smoking, cardiovascular disease, atrial fibrillation (especially valvular),

diabetes.

Others: cardiac failure, dyslipidaemia, obesity, alcohol excess, oral contraception, migraine, stress.

Control of risk factors is the key approach to management. Control of hypertension, including systolic hypertension in the elderly, and smoking cessation are vital factors for reduction of the incidence of stroke. A meta-analysis of 14 randomised trials showed that a reduction of blood pressure of 5–6 mmHg is associated with about 40% reduction in stroke incidence.[1]

Stroke

Facts[1]

- Stroke is the third most common cause of death in Australia.[3]
- About one-third of people who have a stroke will die within 1 month.
- About 50% of ischaemic strokes are preceded by TIAs.
- Symptomatic carotid artery stenosis is the major risk factor for stroke.
- Thromboembolism from vascular disease outside the brain causes 70% of strokes and 90% of TIAs.
- Such sources are atheromatous plaques within the carotid or vertebral systems or cardiac causes (e.g. postmyocardial infarction).
- Echocardiography is an important investigation with TIAs since LV dysfunction and the size of the left atrium are the strongest independent predictors of thromboembolism.

Pathophysiological groups of cerebral infarction

The three main groups are:

- single penetrator or small vessel disease (lacunar syndrome)—probably due to in situ small vessel disease
- cardio–emboli (heart to artery embolism)
- large vessel artery-to-artery embolic infarcts (see TABLE 121.1)

Table 121.1 Types and incidence of stroke[2]

Stroke subtype	Frequency (%)
Haemorrhagic stroke:	10
• primary intracerebral	5
• subarachnoid haemorrhage (SAH)	
Ischaemic stroke:	30
• large vessel (artery-to-artery embolism)	20
• cardio–embolic	15
• small vessels (lacunar infarcts)	15
• uncertain type	5
• rare (e.g. venous infarction)	

Differential diagnosis (‘stroke mimics’)

- Syncope
- Seizure (and subsequent Todd paresis)
- Migraine
- Cerebral tumour and other space-occupying lesions
- Hypoglycaemia
- Hyponatraemia
- Delirium
- Head injury
- MELAS syndrome
- Medically unexplained (e.g. somatisation)

Diagnostic guidelines

- Sudden stroke is typical of embolism.
- The clinical picture depends on the vessel involved.
- In young people <50 years consider patent foramen ovale.
- With cerebral haemorrhage, the stroke evolves steadily, often over hours: the putamen (50%) is the commonest site.

- Lacunar CVAs:

    small, deep infarcts

    pure motor hemiplegia most common effect

    lack of cortical signs

    the neurological deficit may progress over 24–36 hours

    outcome usually good

- Investigate all, including SAH, with CT scans or MRI (may need a lumbar puncture for SAH diagnosis). MRI is preferred if available.
- Carotid duplex Doppler ultrasound scan can accurately determine atherosclerotic narrowing of the extracranial carotid circulation.

Pitfalls

- Mistaking visual or sensory migraine equivalents in young adults for TIAs
- Mistaking a CVA for labyrinthitis (rare over 50 years)
- Failure to perform carotid duplex Doppler ultrasound or CT scan before starting aspirin for TIA or small stroke (because of missing small haemorrhage, unsuspected tumour or a subdural)
- Diagnosing small stroke as a lacuna (may be a stroke in evolution)

Management

The development of an acute stroke or TIA is a medical emergency and admission to a stroke unit (if available) as soon as possible is advised. Care in a stroke unit gives the greatest chance of independent survival.[4]

Immediate

- Stabilise ventilation—consider intubation and oxygen.
- Exclude head trauma.
- Obtain urgent non-contrast CT (thin slice—2 mm) or MRI scan (MRI more sensitive and specific, but urgency is the important factor).[5]
- Treat any seizures.
- Treat any hypoglycaemia.

General

- Investigate, including carotid duplex Doppler ultrasound (for carotid territory symptoms).
- Treat hypertension (systolic >140) vigorously—it carries a six times increased risk of stroke.
- Give IV fluid, electrolyte and nutritional support (nil orally until swallowing has been assessed).
- Good nursing care is the cornerstone of management.
- Physiotherapy and speech therapy.
- Vigorous rehabilitation.
- Clot extraction for intracerebral haemorrhage: consider urgent (<6 hrs) surgical evacuation for haematomas of the posterior fossa (cerebellum) and cerebral white matter.[1] Shunt insertion may be needed. No medical therapy is of proven value for haemorrhagic CVA.
- SAH: requires urgent referral (vasospasm and rebleeding are the main causes of morbidity and mortality):

  nimodipine ± surgery

- Ischaemic stroke: give aspirin 150–300 mg (o) daily within 48 hours if CT scan/MRI has excluded cerebral haemorrhage or other blood-thinning agents are not used. Early intervention within 4.5 hours (earlier the better) of onset with tissue plasminogen activator (tPA). Recent trials indicate improved outcomes with tPA (but not with streptokinase). However, there appears to be a 5–7% risk of intracerebral haemorrhage[1] and slight increase in incidence of death in the first few days.

The usual thrombolytic tPA therapy is alteplase 0.9 mg/kg up to 90 mg IV over 1 hour, with 10% of the dose given as an initial bolus[1] or tenecteplase 0.25 mg/kg, max. 25 mg. Withhold aspirin for 24 hours.

Note: Avoid steroids, mannitol, haemodilution and anticoagulation in acute stroke.[6]

Transient ischaemic attacks

Clinical features

- Sudden onset and short duration (<60 minutes)
- Complete clinical recovery in less than 24 hours (usually <2 hours)
- Around 10% will have a stroke within a week following a TIA,[7] but this risk is substantially

less for those receiving urgent care in an emergency department

- Consciousness usually preserved
- Ninety per cent usually in anterior circulation
- Carotid TIAs—unilateral features
- Vertebrobasilar TIAs—often have bilateral or crossed features

A comparison of the main clinical features of carotid (anterior circulation) ischaemia and vertebrobasilar (posterior circulation) ischaemia is presented in FIGURE 121.1 . The carotid circulation accounts for 80% of TIAs.

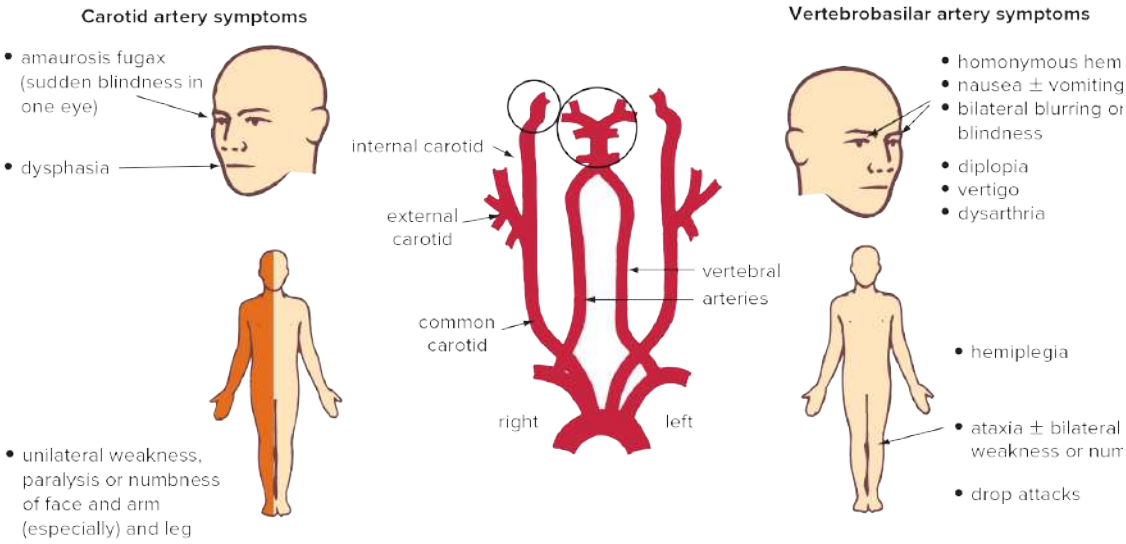

FIGURE 121.1 Cerebral arterial circulation with some important clinical features of carotid and vertebrobasilar ischaemia

Source: Reproduced with permission from C Kenna and J Murtagh, *Back Pain and Spinal Manipulation*, Sydney: Butterworth, 1989

Differential diagnoses of TIAs are presented in TABLE 121.2 .

Table 121.2 Differential diagnosis of TIAs

Classic migraine (with aura)
Unusual migraine variants:

- hemiplegic
- ophthalmoplegic
- retinal

Focal epileptic seizures:

- complex partial
- simple partial

Multiple sclerosis

Transient global amnesia

Syncope

Intracranial structural lesions:

- arteriovenous malformation
- tumour

Vestibular disorders:

- acute labyrinthitis

Benign paroxysmal positional vertigo

Ménière syndrome

Hypoglycaemia

Adverse drug reactions

Toxic reactions

Peripheral nerve lesions:

- carpal tunnel syndrome
- Bell palsy

Psychological conditions:

- anxiety/hyperventilation
- panic attacks
- somatisation

Some ischaemic syndromes

- Transient monocular blindness (amaurosis fugax)
- Transient hemisphere attacks
- The ‘locked-in’ syndrome
- Vertebrobasilar:

  bilateral motor loss

  crossed sensory and motor loss

  diplopia

  bilateral blurring or blindness

Amaurosis fugax

This is the sudden transient loss of vision in one eye (like a ‘curtain or shade’ coming from above or below) due to the passage of an embolus through the retinal vessels from ipsilateral carotid vessel disease. It is a feature of a TIA in the carotid artery circulation and is often the first clinical evidence of carotid stenosis.[8] About 20% of all TIAs present as amaurosis fugax.[9] Amaurosis fugax may forewarn of the development of hemiparesis or blindness and should be considered a matter for urgent attention and rectification. Give aspirin immediately. Carotid endarterectomy may be required for high-grade stenosis.

Differential diagnosis of transient amaurosis fugax

- Thromboembolic phenomena
- Vasospasm
- Temporal arteritis
- Blood—sickle cell, polycythaemia
- Retinal—haemorrhage, venous thrombosis, detachment
- Optic neuritis
- Vitreous floaters

Transient hemisphere attack (usually middle cerebral artery)

- Affects motor or sensory or both
- Usually face and arm (more than leg)
- Dysphasia common

The ‘locked-in’ syndrome

In this syndrome, which may be transient or persistent, patients remain conscious and aware of their dilemma but are unable to speak or move the limbs, particularly the arms. It may be possible to communicate with patients using eye responses to commands. The cause is invariably a lesion in the brain stem.

Significance of TIAs

- Five years after a TIA, 22–51% (average 1 in 3) patients (without treatment) will have a stroke.[8] This figure may be higher for those with ipsilateral high-grade (70% or more) carotid stenosis.

- The highest risk is in the first 6 months.
- A carotid artery TIA has more serious prognostic significance. Such patients are at high risk of developing a stroke that is potentially preventable.
- Referral for investigation is appropriate.
- All patients should have a carotid duplex Doppler ultrasound and CT scan or MRI at presentation.
- Cardiac status should be addressed because of an association with myocardial infarction.

ABCD 2 stroke risk tool[9]

This screening tool is useful as a predictor for risk of stroke in the first 7 days of a TIA.

A = **A**ge ≥60 years (1 point)

B = **B**P ≥140 systolic or ≥90 diastolic (1 point)

C = **C**linical features: any unilateral limb weakness (2 points), speech impairment without weakness (1 point)

D1 = **D**uration: ≥60 minutes (2 points), 10–59 minutes (1 point), <10 minutes (0 points)

D2 = **D**iabetes: 1 point

Maximum 7 points

Scores: 0–3 = 1% risk of stroke at 2 days; 4–5 = 4% risk of stroke at 2 days; 6–7 = 8% risk of stroke at 2 days.

Variants on this tool include ABCD 3.

Investigations[10]

- A CT scan, 12-lead ECG and carotid imaging are essential investigations in the first hours of a suspected TIA
- Blood tests, echocardiography, MRI and Holter or bedside cardiac monitoring are likely to improve diagnosis

- Carotid duplex Doppler ultrasound for carotid territory symptoms
- Transoesophageal echocardiography

Management

- All high-risk patients admitted to a stroke unit or specialist TIA clinic: urgent attention for atrial fibrillation and carotid artery stenosis
- Aim to minimise the risk of a major stroke
- Determine cause and correct it (if possible)
- Early neurorehabilitation
- Advise cessation of smoking and treat hypertension (if applicable)
- Antiplatelet therapy (especially for carotid ischaemia):

  aspirin 100–300 mg (o) daily (gives 30% protection from stroke or death after TIA)[11]

  or

  clopidogrel 75 mg (o) daily (or clopidogrel + aspirin)

  or

  dipyridamole CR + aspirin 200 mg/25 mg (o) bd (shows better outcomes than aspirin alone)[12]

  or

  ticlopidine (only if others unsuitable)
- Recent studies have shown that the benefit of aspirin plus clopidogrel is greatest in the first 3 weeks after a high-risk TIA or minor ischaemic stroke, after which time the bleeding risk of dual therapy starts to outweigh the diminishing ongoing benefit. Therefore Australian guidelines recommend commencing dual therapy within 24 hours and ceasing at three weeks.[5]
- Antiplatelet therapy reduces the relative risk of stroke, myocardial infarction or vascular death by 22% (95% confidence interval 14–30%) in people with an ischaemic stroke or TIA due to arterial disease, compared with no treatment.[2]
- Anticoagulation therapy: warfarin:

  for vertebrobasilar ischaemia (with increasing frequency of TIAs)

  for failed antiplatelet therapy

  use direct oral anticoagulants (DOACs) for non-valvular atrial fibrillation >65 years of age

- Carotid endarterectomy has been proven to have a place in the management of carotid artery stenosis; the decision depends on the expertise of the unit. There is no evidence that surgery is appropriate for asymptomatic patients, nor for symptomatic patients with a stenosis less than 50%. Symptomatic patients benefit from surgery particularly for stenoses greater than 70%.[5,13] If the stenosis is >90%, refer immediately. Greater than 75% stenosis is associated with a 2% per annum rate of ipsilateral ischaemic stroke.[1]
- Percutaneous transluminal angioplasty (stenting). Carotid artery stenting for the treatment and prevention of stroke should be restricted to those who are unsuitable for carotid endarterectomy.[5]

Guiding rules for carotid artery stenosis

- 70–99%—intervention
- 50–69%—'grey area': refer
- <50%—observe

Atrial fibrillation[1,14]

- The main source of cardio–embolic infarction
- Increased with risk factors—hypertension, previous embolism and recent CHF (previous 3 months)
- With non-valvular AF, annual risk of CVA is 2.5% (no risk factors) to 17.6% (2+ risk factors)
- Risk of stroke about 6 times that of other people
- Intermittent AF can also be a risk

Management

- People with AF aged under 65 with no other risk factors do not require anticoagulant therapy.[15]
- Valvular disease: warfarin—target INR 2–3
- Non-valvular AF: calculate the risk–benefit ratio for anticoagulant therapy.
- Do not offer aspirin monotherapy solely for stroke prevention to people with AF.[15]

Selecting antithrombotic therapy in non-valvular AF is assisted by the $CHADS_2$ index,

a validated tool (see TABLE 121.3). Many guidelines use a variation, the CHA_2DS_2-VASc score.

Table 121.3 $CHADS_2$ criteria and stroke risk[14] (for carotid and vertebral arteries)

$CHADS_2$ criteria	Points	Stroke risk	Recommended therapy
Previous stroke or TIA	2	High (2–6)	Warfarin (INR 2–3) or DOACs
Age ≥75 years	1	Moderate (1)	Warfarin or DOACs, but bleeding risk may outweigh benefit
Hypertension	1	Low (0)	Nil
Diabetes mellitus	1		
Heart failure	1		

Cerebral venous thrombosis[2]

This rare cause of stroke may present as an acute or chronic cerebrovascular disorder. It should be suspected particularly in women in the postpartum period presenting with severe headache or focal neurological defects. Diagnosis is by MRI. In the acute phase, treatment is by anticoagulation with heparin followed by warfarin for approximately 6 months.

Indications for carotid duplex Doppler ultrasound

- Bruit in neck
- TIAs if diagnosis uncertain
- Crescendo TIAs (two or more in 1 week and longer lasting)
- Internal carotid artery symptoms
- Hemispheric stroke
- Prior to major vascular surgery (e.g. CABG)

Stroke in children

Stroke in children is relatively uncommon. Causes include paradoxical embolus (e.g. PFO), cerebral vasculopathy (e.g. vasculitis), arterial dissection and metabolic disease. The most common clinical presentation of stroke in childhood is acute hemiparesis due to involvement of the carotid distribution.[1] Investigation includes immediate brain MRI or CT scan, transthoracic echocardiography, thrombophilia factors (e.g. V Leiden) and urine homocysteine. Consider also sickle-cell disease. Initial treatment within 48 hours and after imaging is aspirin 2–5 mg/kg up to 300 mg orally daily.

When to refer

- Refer symptomatic cerebral ischaemia immediately via ED to a stroke unit
- Suspicion of SAH
- Carotid artery stenosis on carotid duplex Doppler ultrasound scan
- Cerebellar haemorrhage on CT scan
- Stroke in a person <50 years of age (consider PFO and other, less common causes)

Key checkpoints

Proven strategies

Three proven strategies to improve outcome of acute stroke (level I evidence):

- management in a stroke unit
- giving IV tPA within 3 hours of ischaemic stroke
- giving antiplatelet agents ASAP and within 48 hours of ischaemic stroke

First aid for stroke

Is it a stroke?

Think **FAST**

↓

Face (ask person to smile)

Arms (raise both arms)

Speech (speak a simple sentence)

Time (within 3 hours)

then, if yes

Refer to stroke unit ASAP

Don’t give aspirin

Patient education resource

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

- Stroke

Resources

Stroke Foundation (Australia). Clinical guidelines for stroke management [internet]: https://informme.org.au/Guidelines/Clinical-Guidelines-for-Stroke-Management

European Society of Cardiology Guidelines: www.escardio.org

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122 Thrombosis and thromboembolism

Defil'd with ropy gore and clots of blood … Dismal and cold, where not a beam of light invades the winter.

OVID, METAMORPHOSES, 8 AD

A thrombus is defined as a clot formed in the circulation from the constituents of the blood, whereas emboli are fragments of thrombus that break off and block vessels downstream. Almost half of adult deaths in Western countries are due to thrombosis of coronary or cerebral arteries, or to pulmonary embolism.[1] A thrombus is the result of a cascade of events involving platelets, RBCs, coagulation factors and the vessel wall.

Conditions predisposing to thrombosis:

- thrombophilia
- thrombocytosis (platelets)
- polycythaemia rubra vera

Thrombophilia[2]

Thrombophilia refers to a disorder of haemostasis in the form of a primary coagulopathy leading to a tendency to thrombosis. This should be considered in those with major unprecipitated venous thromboembolism with or without a strong family history of venous thrombosis. Several causes can be tested, both inherited and acquired:

- inherited:
  - factor V Leiden gene mutation (activated protein C resistance)
  - prothrombin gene mutation
  - protein C deficiency
  - protein S deficiency
  -
  -

antithrombin deficiency

- acquired:

  antiphospholipid antibodies (anticardiolipin or anti-ß$_2$ GPI)

  elevated homocysteine level

  lupus anticoagulant

The above factors can all be measured in the laboratory with specific genetic, coagulation or antibody-based tests. Other acquired causes (regarded as risk factors) include smoking (the big one), malignancy, oral contraceptives, HRT, immobilisation, obesity, pregnancy and major surgery. Most DVTs do not require investigation for an underlying cause, but consideration should be given to screening patients with an unprovoked DVT. Refer to a haematologist if thrombophilia is proven or suspected. Of particular interest is factor V Leiden, which occurs in about 4% of Caucasians; the relative risk of venous thrombosis is increased three to seven times in heterozygotes and 80 times in homozygotes.[2]

Indications for investigation

- Recurrent or unusual thrombosis
- Arterial thrombosis <30 years
- Skin necrosis, especially on warfarin
- Recurrent fetal loss
- Familial thromboembolism

Venous thromboembolism

A feature of venous thrombosis is that it develops in normal vessels, with key factors being stasis and increased coagulability, including thrombophilia. The classic example is deep venous thrombosis of the leg veins. Another poorly recognised thrombosis is axillosubclavian venous thrombosis, which is associated with pulmonary embolism in 30% of cases.[3]

Other sites of deep vein thrombosis are the pelvic/ovarian veins, which should be suspected in a person at risk with pelvic pain and swollen upper thighs, mesenteric venous thrombosis and cerebral sinus thrombosis, which are usually due to thrombophilia.

Deep venous thrombosis

Past history is very important.

Risk factors

- Family history
- Thrombophilia
- History of previous thromboembolism
- Drugs (e.g. OCP, tamoxifen, HRT)
- Malignancy (watch idiopathic DVT)
- Increasing age; age >40
- Varicose veins
- Significant illness, esp. heart failure and cancer
- Other chronic illness
- Recent surgery
- Major/orthopaedic surgery
- Immobility
- Long flights
- Pregnancy/puerperium
- Obesity
- Dehydration

There is an association of up to 20% with pulmonary emboli, of which 30% may be fatal. DVT may be asymptomatic, but usually causes tenderness in the calf. DVT may present with painless unilateral leg swelling. Because of the potentially serious consequences from untreated thromboembolism, it is essential to objectively confirm or exclude clinically suspected disease.

Examination

May be low-grade fever.

Examine both legs. Look for:

- swelling of calf and thighs
- asymmetry

- erythema
- superficial veins

Feel for:

- warmth
- tenderness (gently squeeze calves)
- pitting oedema

Don’t test Homan sign (pain on sharp dorsiflexion of foot) as it may dislodge a thrombus.

Refer to Wells criteria (e.g. https://www.nice.org.uk/guidance/ng158 or www.mdcalc.com/wells-criteria-for-dvt/).

Investigations

- Duplex ultrasound: accurate for above-knee thrombosis; improving for distal calf
- Should be repeated in 1 week if initial test normal
- Contrast venography: reserved if ultrasound doubtful

Note:

- MRI has roughly similar sensitivity and specificity as ultrasound for DVT.[4]
- The plasma D-dimer can help to ‘rule out’ thrombosis. Where the clinical probability of venous thrombosis is low, a normal D-dimer effectively excludes the diagnosis. However, where clinical probability is high, appropriate imaging with Doppler ultrasound or a lung scan should be performed. A raised D-dimer is non-specific and is little help in confirming the diagnosis.

Treatment[3,5]

Provide education and counselling. Admit to hospital (usually 5–7 days)—can treat as an outpatient. Drugs used in the treatment of thromboembolism are summarised in general format in TABLE 122.1 .

Table 122.1 Drugs employed in thrombotic disorders

Antiplatelet

Aspirin

Clopidogrel
Dipyridamole
Prasugrel
Ticagrelor
Ticlopidine
Glycoprotein IIb/IIIa inhibitors (e.g. abciximab)

Anticoagulants

Heparin:

- unfractionated/standard
- low molecular weight
    - dalteparin
    - enoxaparin
    - danaparoid

Vitamin K antagonists:

- phenindione
- warfarin

Clotting factor inhibitors:

- direct thrombin inhibitors[6]
    - bivalirudin (IV use)
    - dabigatran (oral)
    - lepirudin (IV use)
- factor Xa inhibitors
    - apixaban (oral)
    - fondaparinux (SC use)
    - rivaroxaban (oral)

Thrombolytics

Alteplase
Reteplase
Streptokinase
Tenecteplase
Urokinase

- Collect blood for APTT, international normalised ratio (INR) and platelet count
- Check renal and liver function
- The choice of two regimens depends on relative contraindications: either warfarin with heparin cover or NOAC without heparin cover (exception is dabigatran, which requires

heparin)[3]

- *Method 1:*

  LMW heparin, e.g. enoxaparin or dalteparin

  or

  unfractionated heparin (UFH) 330 U/kg, SC loading dose then 250 U/kg bd SC (or monitor with APTT)

  or

  UFH 5000 U bolus IV then infusion in N saline (12 500 U over 12 hours)

  monitor with APTT after 4–8 hours then for 5–7 days

  or

  fondaparinux SC, according to weight, e.g. 50–100 kg: 7.5 mg SC daily

  Oral anticoagulant (warfarin) for 3 months (or 6 months if unprovoked);

  commence on day 1 or 2, usually 5 mg nocte for 2 nights and then according to INR monitoring (max. 30 mg in 3 days)

  Method 2:

  apixaban 10 mg (o) bd for 7 days, then 5 mg bd

  or

  rivaroxaban 15 mg (o) bd for 21 days, then 20 mg daily

- Do not give aspirin
- Mobilisation within the limits of pain, tenderness and swelling
- Class II graded compression stocking to affected leg in proximal DVT associated with significant swelling. The stocking may be above or below the knee, depending on the extent of the swelling
- Duration of anticoagulation therapy ranges from 6 weeks (distal DVT with major provoking factor resolved) to 3 months (most common duration: proximal DVT or PE with major provoking factor resolved, and also most unprovoked thromboses) to extended/indefinite (for high risk—seek advice)[3]

Long term: complete resolution 50–80% at 6 months and almost 100% by 12 months.

Prevention

Surgery

- Early ambulation
- LMW heparin or new anticoagulants for orthopaedic (for 14–35 days) or other high-risk surgery (for 1 week or until fully mobile) (see TABLE 122.1)
- Graded pressure elastic stockings
- Physiotherapy
- Pneumatic compression (especially in high-risk patients where heparin is contraindicated)
- Electrical calf muscle stimulation during surgery

Prolonged travel/immobilisation

- Keep hydrated—ample water.
- Avoid or restrict alcohol and coffee.
- Exercises—3–4 minutes per hour (e.g. walking, calf contraction—e.g. foot pumps, see FIG. 122.1 , ankle circles, knee lifts).
- Injections—LMWH just prior to flying and on arrival for those at high risk. Use a prophylactic dose (e.g. enoxaparin 40 mg or dalteparin 5000 U—both SC injections twice daily).

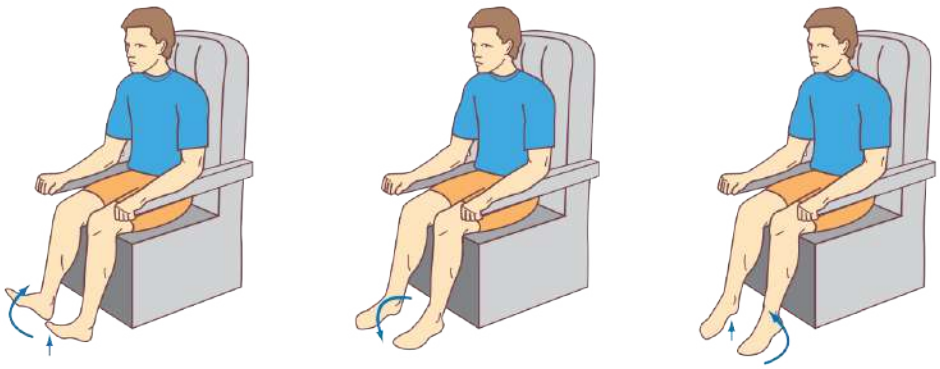

FIGURE 122.1 Foot pump exercises to prevent DVT during prolonged air travel

1 Start with the feet flat and both heels on the floor, lift the feet (toes up) as high as you can.
2 Return the feet to the flat position, pressing them firmly on the floor.
3 Lift the heels high, keeping the balls of the feet on the floor.

Continue this up-and-down movement for at least 30 seconds. Repeat often.

Pulmonary embolism[7]

Refer to CHAPTER 30 for more detail on the clinical features and management of pulmonary embolism. CT pulmonary angiography is very specific and appears to be as sensitive as V/Q scanning for embolism, and is currently the preferred first-line investigation. The basis of treatment is LMW heparin and warfarin, as for DVT.

Arterial thromboembolism

The common serious manifestations of this are myocardial infarction, stroke, occlusion of the arterial system of the lower limbs and 'eye' embolism (e.g. central retinal artery thrombosis). Emboli originate from the left side of the heart, the carotid arteries or the iliac arteries. Atrial fibrillation is an important predisposing factor.

Preventing systemic embolism in atrial fibrillation[7]

Atrial fibrillation (AF) accounts for about 15% of ischaemic strokes caused by systemic embolism of cardiac thrombi. This risk gradually increases with age. Use of warfarin or DOAC reduces the annual incidence of stroke during AF from 4.5% to 1.4%—a risk reduction of almost 70%. The decision to use it or an antiplatelet agent is a difficult one and should be made in consultation with a cardiologist. Although aspirin reduces stroke rate by about 20% in patients with AF when compared with no treatment, it has half the effectiveness of warfarin/DOACs and is less able to prevent severe strokes. As a general rule, all patients with AF should start on warfarin or DOAC unless <65 years old or have a major contraindication to its use. It is not indicated in patients with lone AF who are less than 60 years of age with no risk factors. If using warfarin, start with a low dose (e.g. 2–4 mg) and maintain an INR of 2–3 with regular checks. Anticoagulant therapy is also required to prevent embolism after cardioversion.

Drug treatment of thrombotic disorders

The direct oral anticoagulants (DOACs)[3,6]

The term DOAC is interchangeable with NOAC—the 'N' originally meant 'novel' but now stands for 'non-vitamin-K'. These alternatives to warfarin are currently indicated for thromboprophylaxis in non-valvular atrial fibrillation, and for treatment and secondary prevention of DVT and PE, particularly following hip and knee replacement surgery. Apixaban, rivaroxaban and dabigatran need less monitoring than warfarin, but at the time of writing only dabigatran has an effective antidote (idarucizumab). Intracranial bleeding is less likely with DOACs than with warfarin. Caution for use is required in renal impairment and they are contraindicated in patients with renal failure. This requires measurement of creatinine clearance (not eGFR, which does not allow for weight).

Warfarin

Warfarin is an oral agent used in the treatment and prevention of venous thromboembolism, unlike the antiplatelet drugs which have little or no benefit and are recommended in arterial disease. Before prescribing warfarin, the risk of bleeding should be evaluated and discussed with each individual. Indications for warfarin treatment are outlined in TABLE 122.2 .

Table 122.2 Warfarin anticoagulation

Indications
Prosthetic cardiac valves
Deep venous thrombosis, pulmonary thromboembolism
Atrial fibrillation (selected cases)
Postoperatively in lower limb orthopaedic surgery (low dose)
Postcoronary bypass surgery (selected cases)
Thrombosis in antiphospholipid antibody syndrome
Contraindications
Active bleeding
History of intracranial haemorrhage
Uncontrolled hypertension
Liver disease with impaired synthetic function—based on INR
Pregnancy

Actions[5]

- Antagonises vitamin K.
- Depresses factors VII, IX and X (half-life of 30–40 hours) and prothrombin.
- Achieves full anticoagulation effect after 5–7 days.
- Prothrombin time (INR ratio) of 2–3 times normal control indicates therapeutic effect.
- The INR is a good indicator of effectiveness and risk of bleeding.
- Duration of effect is 4–5 days after cessation.
- It can cause thrombocytopenia.
- Antidote is vitamin K ± plasma or prothrombin complex concentrate.

Initiation of warfarin treatment[8,9]

An estimate of the patient's final steady dose is made. The patient is commenced on this dose and the INR monitored daily and the dose altered accordingly.

- Measure INR first to establish baseline.
- Generally warfarin is commenced on same day or day after heparin is commenced.
- Heparin can be ceased when INR >2 for 2 consecutive days.
- Typical loading dose is 5–10 mg (o) daily (usually 5 mg) for 2 days (avoid dose >30 mg over 3 days without INR).
- Adjust the dosage according to an INR table (*Therapeutic Guidelines*: www.tg.org.au) from the third day. Some pathology laboratories offer patients this personalised adjustment service.
- Establish the INR in the therapeutic range, usually 2–3 (average 2.5).
- Maintenance dose is usually reached by day 5.
- The INR reflects the warfarin dose given 48 hours earlier.
- Warfarin is best taken in the evening and INR measured in the morning.

Note:

- Warfarin should be discontinued at 3 months following a first episode of a non-extensive venous thromboembolic event that occurred in the setting of a major, transient risk factor.[10]
- Watch for potential drug interactions.

Recommended target INR values are given in the box.

Recommended INR target values

Prevention of DVT	2.0–3.0
Treatment of DVT or PE	2.0–3.0
Preventing systemic embolism:	2.0–3.0

- atrial fibrillation
- post-myocardial infarction
- tissue heart valve
- valvular heart disease

Mechanical prosthetic heart valve	2.5–3.5
Prevent recurrence of MI	2.0–3.0
Antiphospholipid antibody syndrome thrombosis	2.0–3.0

Overdosage of warfarin

Signs of warfarin overdosage include:

- unexpected bleeding after minor trauma
- epistaxis
- spontaneous bruising
- unusually heavy menstrual bleeding
- gastrointestinal bleeding

Management of overdosage[11]

1. Urgent measurement of INR is required.
2. If the only evidence of overdosage is a small increase of the INR above the therapeutic range, cessation of warfarin for 1 to 2 days followed by a continuation at a lower dose is appropriate.
3. If the INR is markedly elevated (>5.0) consider giving oral vitamin K, e.g. 10 mg tablet.
4. If bleeding is minor, transient action as in point 2 is still appropriate.
5. If bleeding is persistent, or severe, or involves closed body cavities (such as pericardium, intracranial, fascial compartment), urgent admission to hospital is essential. The anticoagulation may need to be reversed by administering oral or parenteral vitamin K. Infusion of fresh frozen plasma and/or prothrombin complex concentrate (best option) may also be necessary.

Drug interactions

There are so many potential interactions between warfarin and other drugs that the following general principles should be applied:

1. Maintain the simplest possible drug regimens. Avoid polypharmacy.
2. Aspirin is contraindicated while the patient is on warfarin because of the combined antiplatelet and anticoagulation effects. The risk of gastrointestinal bleeding is also increased. Other NSAIDs should also be avoided (see TABLE 122.3).

3. If the patient's drug regimen must be altered during warfarin therapy, then the INR should be followed closely until stable.

Table 122.3 Some important drug interactions with warfarin

Effects on warfarin activity	Drug
↑ Increased	Allopurinol Amiodarone Anabolic steroids Antibiotics (broad spectrum) Antifungals Aspirin—salicylates (high doses) Chloral hydrate Cimetidine Clofibrate Gemfibrozil Metronidazole Miconazole NSAIDs, including COX-2 inhibitors Paracetamol (large doses) Phenytoin Proton-pump inhibitors Quinidine/quinine Ranitidine SSRIs Sulfonamides Tamoxifen Thyroxine Herbal medicines: • dong quai • papaya • St John's wort
↓ Decreased	Antacids Antihistamines Barbiturates Anti-epileptics (e.g. carbamazepine) Cholestyramine (reduced absorption)

	Griseofulvin Haloperidol Oestrogen/oral contraceptives Rifampicin Vitamin C
Increased or decreased	Alcohol Chloral hydrate Diuretics Ranitidine

Advice to the patient

- Keep to a consistent diet.
- Do not take aspirin or liquid paraffin.
- Always mention that you take warfarin to any doctor, dentist or chemist you are consulting.
- Remember to take tablets strictly as directed and have your blood tests.
- Report signs of bleeding, such as black motions, blood in urine, easy bruising, unusual nose bleeds, heavy periods, ‘purple toes’.

Note: Give the patient the information sheet about risks.

Bleeding while on heparin

- Recheck APTT
- Cease or reduce heparin
- Admit patient—alert laboratory and haematologist

Antidotes:

- protamine sulphate will reverse (use with caution)
- fresh frozen plasma
- clotting factors (as guided by consultant)

Practice tips for warfarin[12]

- Consider avoiding use if patient compliance is likely to be poor.
- The INR result reflects the warfarin dose administered 48–72 hours earlier.
- Advise and encourage patient to keep a record in an 'anticoagulant diary' of drug dosage and INR results.
- An unacceptable INR is >5.0.
- Discontinue therapy if skin necrosis or 'purple toes syndrome' occurs.

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123 Common skin wounds and foreign bodies

The variety of foreign bodies which have found their way into the rectum is hardly less remarkable than the ingenuity displayed in their removal.
A turnip has been delivered PR by the use of obstetric forceps.
A stick firmly impacted has been withdrawn by inserting a gimlet into its lower end.
A tumbler, mouth downwards, has several times been extracted by filling the interior with a wet plaster of Paris bandage, leaving the end of the bandage extruding, and allowing the plaster to set.

BAILEY AND LOVE, *SHORT PRACTICE OF SURGERY*, 1943

Injuries to the skin, including simple lacerations, abrasions, contusions and foreign bodies, are among the commonest problems encountered in general practice. To manage these cosmetically important injuries well is one of the really basic and enjoyable skills of our profession.

Key facts and checkpoints

- Keep a well-prepared treatment room with good, sterile instruments and dressings, and an assistant who facilitates their management.
- With lacerations, check carefully for nerve damage, tendon damage and arterial damage.
- In wounds caused by glass, beware of slivers—explore carefully and X-ray (or high-resolution ultrasound) if in doubt.
- Beware of electrical or thermal wounds because marked tissue necrosis can be hidden by slightly injured skin.
- Beware of roller injuries such as car wheels.
- Beware of pressure gun injuries such as oil and paint. The consequences can be disastrous.

- Beware gravel rash wounds, where retained fragments of dirt and metal can leave a ‘dirty’, tattoo-like effect in the healed wound.
- Avoid suturing the tongue, and animal and human bites, unless absolutely necessary.
- Keep diagrams or photographs of wounds in your medical records.
- Have a management plan for medical needle-stick injuries.

Contusions and haematomas

A contusion (bruise or ecchymosis) is the consequence of injury causing bleeding in subcutaneous or deeper tissue while leaving the skin basically intact. It might take weeks to resolve, especially if extensive.

A haematoma is a large collection of extravasated blood that produces an obvious and tender swelling or deformity. The blood usually clots and becomes firm, warm and red; later (about 10 days) it begins to liquefy and becomes fluctuant.

Principles of management

- Explanation and reassurance
- **RICE** (for larger bruises/haematomas) for 48 hours

  R = **R**est

  I = **I**ce (for 20 minutes every 2 waking hours)

  C = **C**ompression (firm elastic bandage)

  E = **E**levation (if a limb)
- Analgesics: paracetamol
- Avoid needle aspiration (some exceptions)
- Avoid massage
- Local heat may be applied after 72 hours
- Consider possibility of bleeding disorder if bleeding is out of proportion to the injury

Problematic haematomas

Some haematomas in certain locations can cause deformity and other problems.

Haematoma of nasal septum[1]

Refer to CHAPTER 48 .

Haematoma of the pinna[1]

When trauma to the pinna causes a haematoma between the epidermis and the cartilage, a permanent deformity known as ‘cauliflower ear’ may result. The haematoma, if left, becomes organised and the normal contour of the ear is lost.

The aim is to evacuate the haematoma as soon as practical and then to prevent it reforming. One can achieve a fair degree of success, even on haematomas that have been present for several days.

Method

Under aseptic conditions insert a 25 gauge needle into the haematoma at its lowest point and aspirate the extravasated blood (see FIG. 123.1A). Apply a padded clamp to the haematoma site and leave on for 30–40 minutes (see FIG. 123.1B). Generally, daily aspiration and clamping are sufficient to eradicate the haematoma completely.

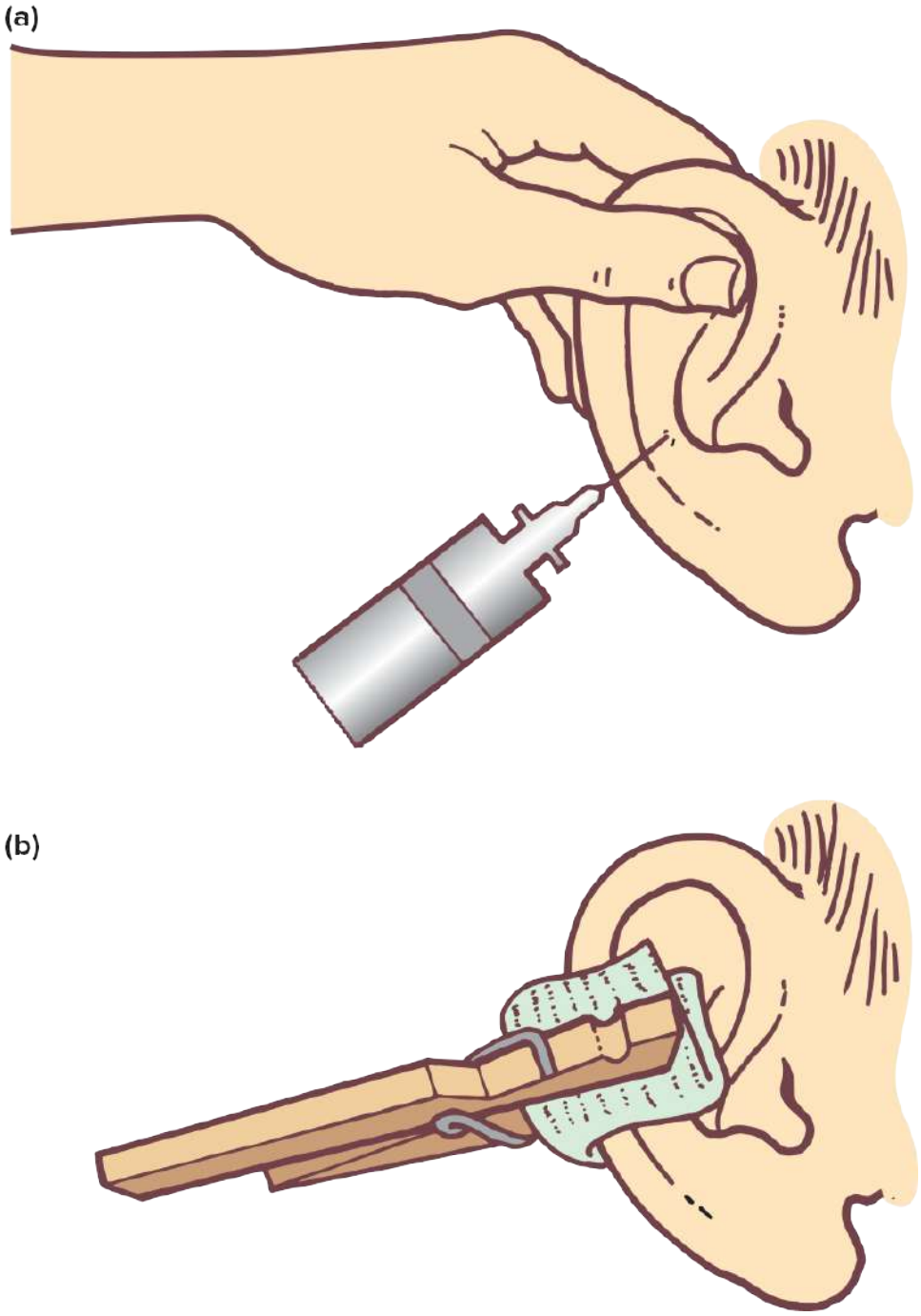

FIGURE 123.1 Treatment of haematoma of the pinna

Subungual haematomas

These important haematomas are discussed in CHAPTER 119 .

Abrasions

Abrasions vary considerably in degree and potential contamination. They are common with bicycle, motorcycle and skateboard accidents. Special care is needed over joints such as the knee or elbow.

Rules of management

- Clean meticulously; remove all ground-in dirt, metal, clothing and other material.
- Scrub out dirt with sterile normal saline under anaesthesia (local infiltration or general anaesthesia for deep wounds).
- Treat the injury similarly to a burn.
- When clean, apply a protective dressing (some wounds may be left open).
- Use paraffin gauze and non-adhesive absorbent pads such as Melolin.
- Ensure adequate follow-up.
- Immobilise a joint that may be affected by a deep wound.

Lacerations

Lacerations vary enormously in complexity and repairability. Very complex lacerations and those involving nerves or other structures should be referred to an expert.

Principles of repair

- Good approximation of wound edges minimises scar formation and healing time.
- Pay special attention to debridement.
- Avoid deep layers of suture material in a contaminated wound—consider drainage.
- Inspect all wounds carefully for damage to major structures such as nerves and tendons and for foreign material:

  shattered glass wounds require careful inspection and perhaps plain X-ray or ultrasound

  high-energy wounds (e.g. motor mowers) are prone to having metallic foreign bodies and associated fractures
- Consider X-rays of wounds to look for foreign objects or fractures (compound fractures).

- Trim jagged or crushed wound edges, especially on the face.
- Close wounds such that opposite layers line up.
- Avoid leaving dead space.
- Do not suture an ‘old’ wound (greater than 8 hours) if it is contaminated. If after four days it remains uninfected, you may excise a thin section off each edge to obtain a new healing surface and suture.
- Take care in poor healing areas, such as backs, necks, calves and knees, and in areas prone to hypertrophic scarring, such as over the sternum, chest or shoulder.
- Use atraumatic tissue-handling techniques, with minimal handling of wound edges.
- Everted edges heal better than inverted edges.
- A suture is too tight when it blanches the skin between the thread—loosen or replace it.
- Avoid tension on the wound, especially in fingers, lower leg, foot or palm.
- A finer scar and better result is obtained by using a large number of fine sutures rather than fewer thick sutures more widely spread.
- Avoid haematoma.
- Apply a firm pressure dressing when appropriate, especially with swollen skin flaps.
- Consider appropriate immobilisation for wounds. Many wound failures could have been avoided by immobilisation via a volar slab (hand) or a back slab (leg).

Practical aspects

Suture material

See TABLE 123.1 .

Table 123.1 Selection of suture material (guidelines)

Skin	Nylon 6/0	Face
	Nylon 3/0	Back, scalp
	Nylon 5/0	Elsewhere
Deeper tissue (dead space)	Catgut 4/0	Face
	Dexon/Vicryl 3/0 or 4/0	Elsewhere

Subcuticular	Monofilament absorbable, e.g. Monocryl 4/0
Small vessel ties	Plain catgut 4/0
Large vessel ties	Dexon/Vicryl

- Monofilament nylon sutures are generally preferred for skin repair.
- Use the smallest calibre compatible with required strains.
- The synthetic, absorbable polyglycolic acid or polyglactin sutures (Dexon, Vicryl) are stronger than catgut of the same gauge, but are not as suitable as catgut on the face or subcuticularly.

Instruments

Examples of good-quality instruments:

- locking needle holder (e.g. Crile–Wood 12 cm)
- skin hooks
- iris scissors
- toothed forceps

Holding the needle

The needle should be held about two-thirds of the way back from the tip; it can distort if held right near the back end (see FIG. 123.2). Tougher tissues may require grasping the needle at its centre.

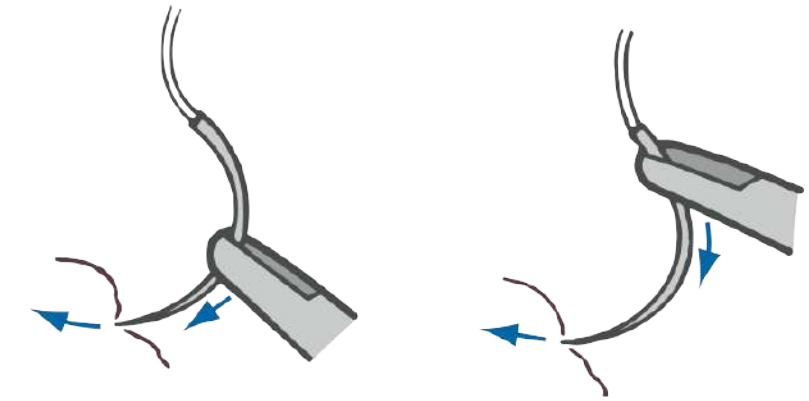

FIGURE 123.2 Holding the needle

Dead space

Dead space should be eliminated to reduce tension on skin sutures. Use buried, absorbable sutures to approximate underlying tissue. This is done by starting suture insertion from the fat to pick up the fat/dermis interface so as to bury the knot (see FIG. 123.3).

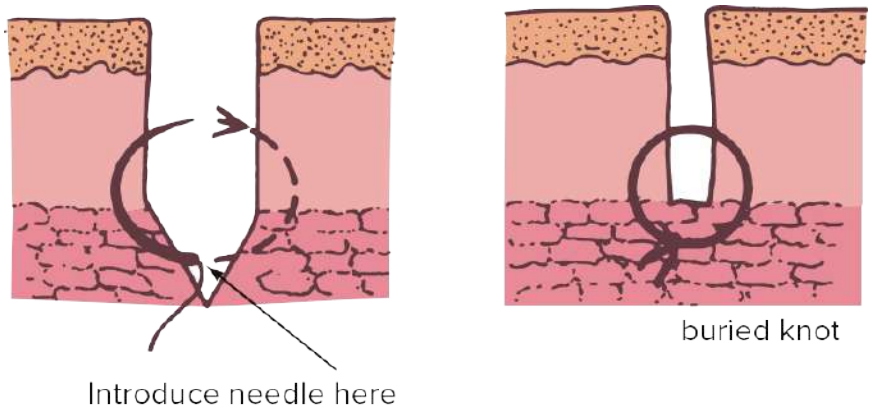

FIGURE 123.3 Eliminating dead space

Everted wounds

Eversion is achieved by making the ‘bite’ in the dermis wider than the bite in the epidermis (skin surface) and making the suture deeper than it is wide. Shown are:

- simple suture (see FIG. 123.4A)
- vertical mattress suture (see FIG. 123.4B)

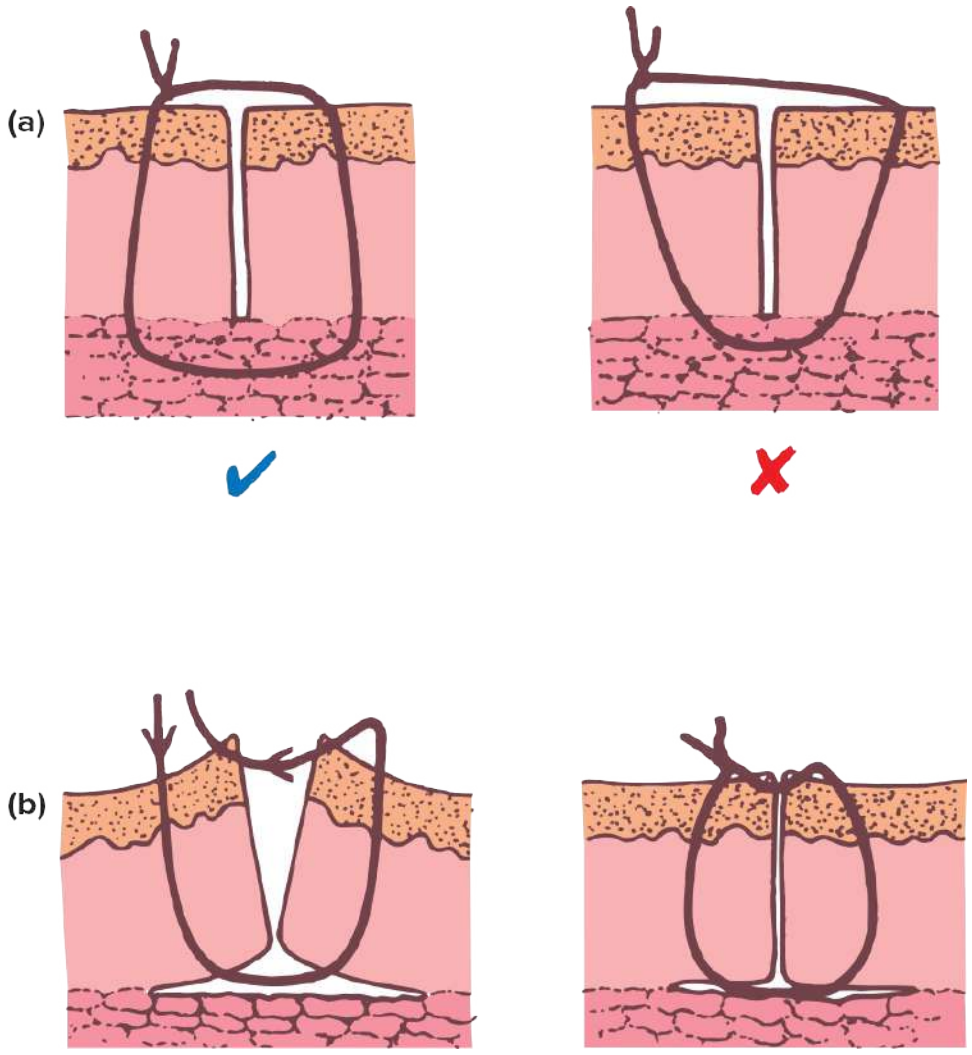

FIGURE 123.4 Everted wounds: correct and incorrect methods of making a **(a)** simple suture, **(b)** vertical mattress suture

The mattress suture is the ideal way to evert a wound.

Number of sutures

One should aim to use a minimum number of sutures to achieve closure without gaps but sufficient sutures to avoid tension. Place the sutures as close to the wound edge as is reasonably possible.

Practice tips

- Have the patient lie down for suturing and the parents of children sit down.

- Avoid using antibiotic sprays and powders in simple wounds—resistant organisms can develop.
- Consider tetanus and gas gangrene prophylaxis in contaminated and deep necrotic wounds.
- Give a tetanus booster if the patient has not had one within 5 years for dirty wounds or within 10 years for clean wounds.
- Give tetanus immunoglobulin if patient is not immunised and the wound is grossly contaminated.
- Never send head-wound patients home before thoroughly washing their hair and carefully examining for other lacerations.
- A laceration in the cheek, mandible or lower eyelid may damage the facial nerve, parotid duct or lacrimal duct respectively.
- When a patient falls onto glass it takes bone to halt its cutting path. Assume all structures between skin and bone are severed.

Special techniques for various wounds

The three-point suture

In wounds with a triangular flap component, it is often difficult to place the apex of the flap accurately. The three-point suture is the best way to achieve this while minimising the chance of strangulation necrosis at the tip of the flap.

Method

1. Pass the needle through the skin of the non-flap side of the wound.
2. Then pass it through the subcuticular layer of the flap tip at exactly the same level as the reception side.
3. Finally, pass the needle back through the reception side so that it emerges well back from the V flap (see FIG. 123.5).

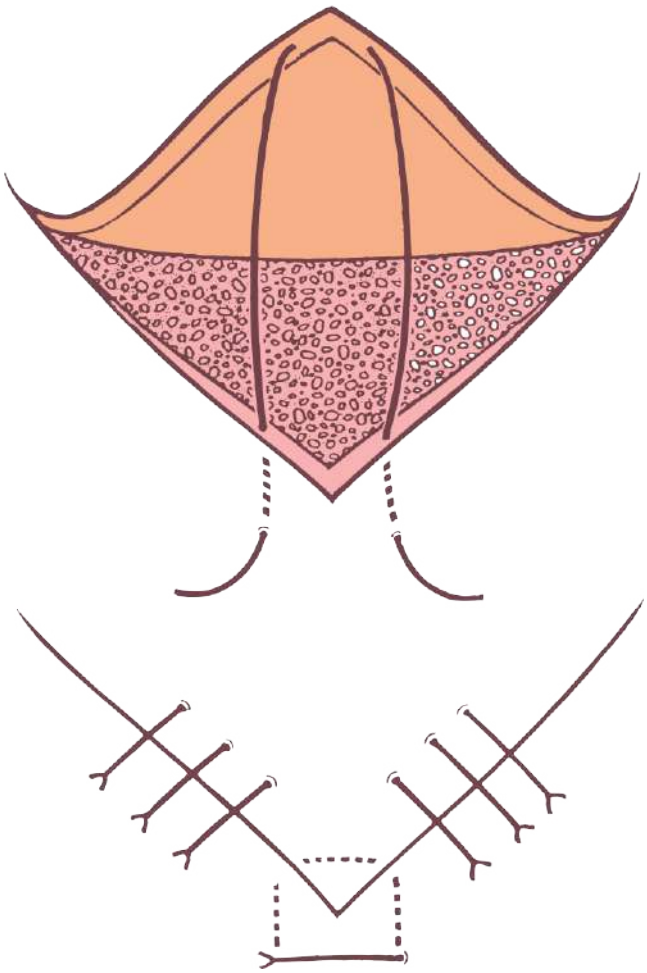

FIGURE 123.5 The three-point suture

Triangular flap wounds on the lower leg

Triangular flap wounds below the knee are a common injury and are often treated incorrectly. Similar wounds in the upper limb heal rapidly when sutured properly, but lower limb injury will not usually heal at first intention unless the apex of the flap is given special attention.

Proximally based flap

A fall through a gap in floorboards will produce a proximally based flap; a heavy object (such as the tailboard of a trailer) striking the shin will result in a distally based flap.

Often the apex of the flap is crushed and poorly vascularised; it will not survive to heal after suture.

Treatment methods (under infiltration with LA)

1. Preferred method: to attempt to salvage the distal flap, scrape away the subcutaneous tissue on the flap and use it as a full-thickness graft.

2. An alternative is to excise the apex of the flap, loosely suture the remaining flap and place a small split-thickness graft on the raw area (see FIG. 123.6).

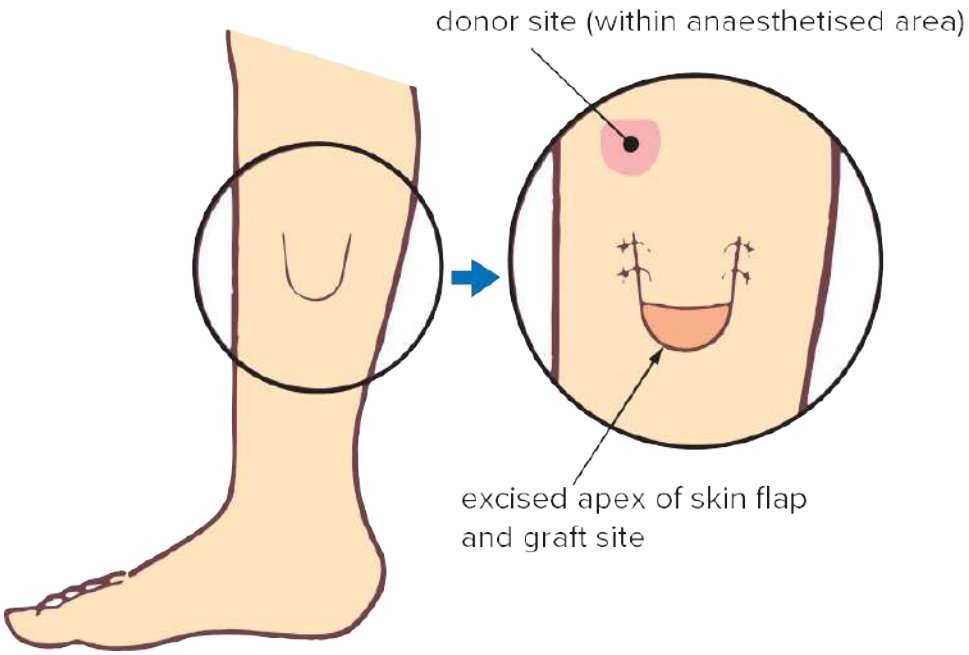

FIGURE 123.6 Triangular flap wound repair: proximally based flap

For both methods apply a suitable dressing and strap firmly with a crepe bandage. The patient should rest with the leg elevated for 3 days.

Distally based flap

See FIGURE 123.7 . This flap, which is quite avascular, has a poorer prognosis. The same methods as for the proximally based flap can be used. Trimming the flap and using it as a full thickness graft has a good chance of repair in a younger person but a poor chance in elderly people.

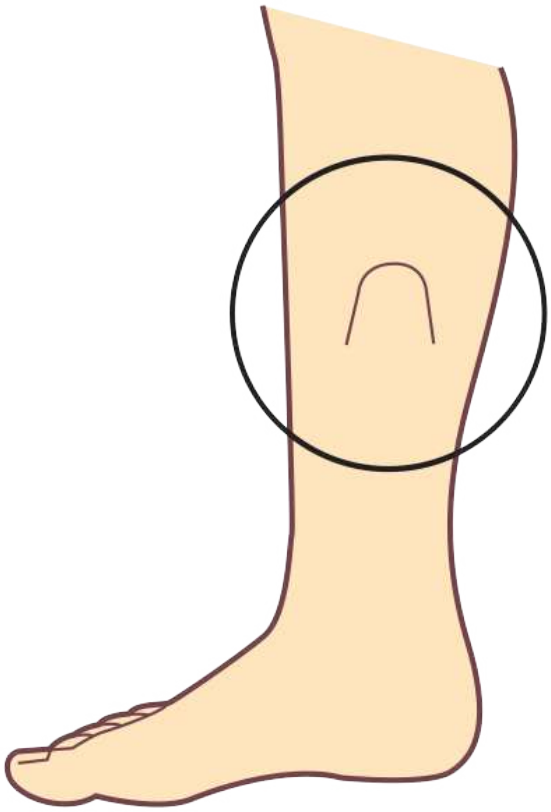

FIGURE 123.7 Triangular flap wound repair: distally based flap

Repair of cut lip

While small lacerations of the buccal mucosa of the lip can be left safely, more extensive cuts require careful repair. Local anaesthetic infiltration may be adequate, although a mental nerve block is ideal for larger lacerations of the lower lip.

For wounds that cross the vermilion border, meticulous alignment is essential. It may be advisable to pre-mark the vermilion border with gentian violet or a marker pen. It is desirable to have an assistant.

Method

1. Close the deeper muscular layer of the wound using 4/0 CCG. The first suture should carefully appose the mucosal area of the lip, followed by one or two sutures in the remaining layer.
2. Next, insert a 6/0 monofilament nylon suture to bring both ends of the vermilion border together. The slightest step is unacceptable (see FIG. 123.8). This is the key to the procedure.
3. Close the inner buccal mucosa with interrupted 4/0 plain catgut sutures.

4. The outer skin of the lip (above and below the vermilion border) is closed with interrupted nylon sutures.

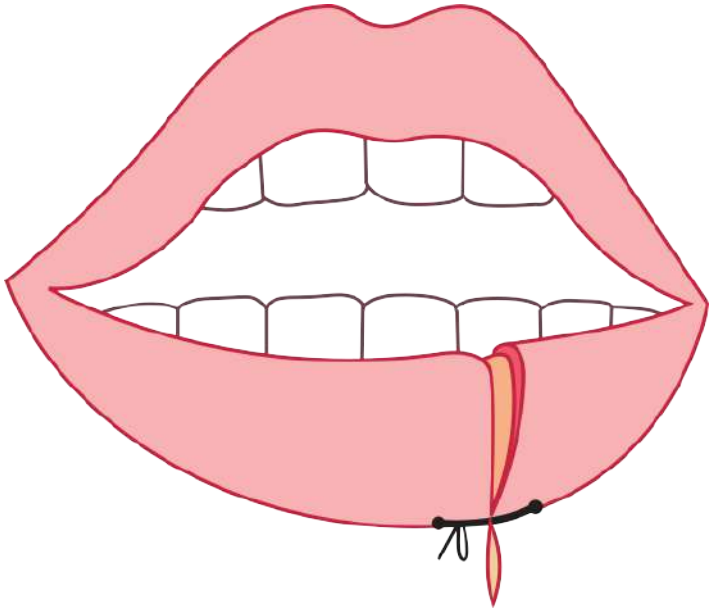

FIGURE 123.8 The lacerated lip: ensuring meticulous suture of the vermilion border

Post-repair

1. Apply a moisturising lotion (or petroleum jelly) along the lines of the wound.
2. Remove nylon sutures in 3–4 days (in a young person) or 5–6 days (in an older person).

Repair of lacerated eyelid

General points

- Preserve as much tissue as possible.
- Do not shave the eyebrow.
- Do not invert hair-bearing skin into the wound.
- Ensure precise alignment of wound margins.
- Tie suture knots away from the eyeball. If necessary, leave suture ends long and tape the strands away from the eye.

Method

1. Place an intermarginal suture behind the eyelashes if the margin is involved.
2. Repair conjunctiva and tarsus with 6/0 catgut.
3. Then repair skin and muscle (orbicularis oculi) with 6/0 nylon (see FIG. 123.9).

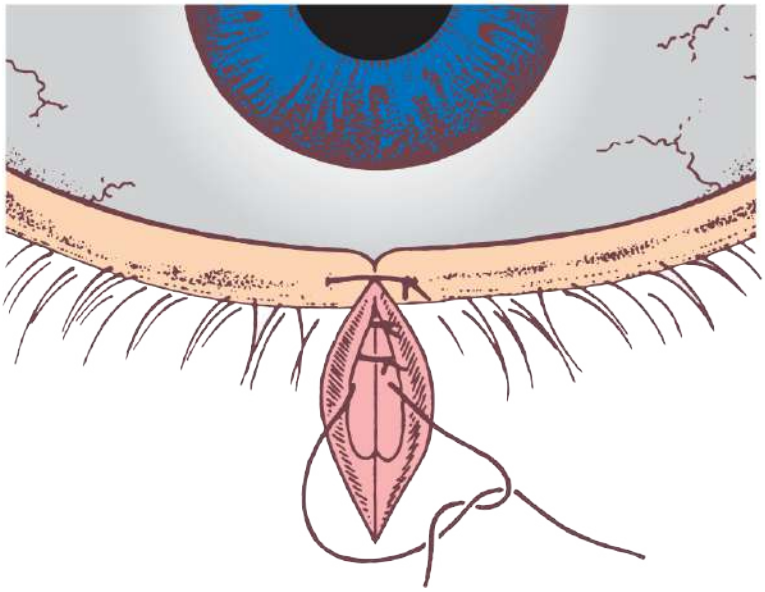

FIGURE 123.9 The lacerated eyelid

Repair of tongue wound

Wherever possible, it is best to avoid repairs to tongue wounds because these heal rapidly. However, large flap wounds to the tongue on the dorsum or the lateral border may require suturing. The best method is to use buried catgut sutures.

Method

1. Get patient to suck ice for a few minutes, then infiltrate with 1% lignocaine and leave for 5–10 minutes.
2. Use 4/0 or 3/0 catgut sutures to suture the flap to its bed, and bury the sutures (see FIG. 123.10).

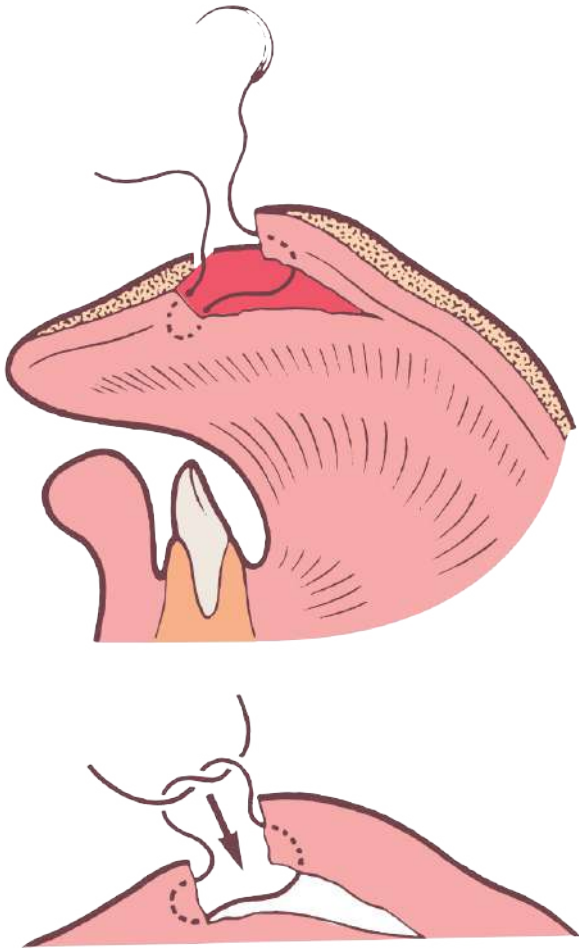

FIGURE 123.10 Repair of tongue wound

It should not be necessary to use surface sutures. If it is, 4/0 silk sutures will suffice.

Instruct the patient to rinse the mouth regularly with salt water until healing is satisfactory.

The amputated finger

In this emergency situation, instruct the patient to place the severed finger directly into a fluid-tight sterile container, such as a plastic bag or sterile specimen jar. Then place this ‘unit’ in a bag containing iced water with crushed ice.

Note: Never place the amputated finger directly in ice or in fluid such as saline. Fluid makes the tissue soggy, rendering microsurgical repair difficult.

Care of the finger stump

Apply a simple, sterile, loose, non-sticky dressing and keep the hand elevated.

Bite wounds

Human bites and clenched fist injuries

Human bites and clenched fist injuries can present a serious problem of infection. Beta-lactamase-producing anaerobic organisms in the oral cavity (e.g. Vincent's) can penetrate the damaged tissue and form a deep-seated infection. *Streptococcus* species, staphylococcal organisms and *Eikenella corrodens* are common pathogens. Complications of the infected wounds include cellulitis, wound abscess and lymphangitis. A Cochrane review of antibiotic prophylaxis concluded that it reduces the risk of infection.[2]

Principles of treatment

- Clean and debride the wound carefully (e.g. aqueous antiseptic solution or hydrogen peroxide).
- Give prophylactic penicillin if a severe or deep bite.
- Avoid suturing if possible.
- Tetanus toxoid (although minimum risk).
- Consider rare possibility of HIV and hepatitis B or C infections.
- For high-risk wounds, give procaine penicillin 1.5 g IM statim and/or amoxicillin/clavulanate 875/125 mg bd for 5 days.[3]
- If established infection in a deep wound, take a swab and give metronidazole 400 mg (o) bd for 14 days plus either cefotaxime 1 g IV 8 hourly or ceftriaxone 1 g IV daily for 14 days.

Dog bites

Non-rabid

Dog bites typically have poor healing and carry a risk of infection with anaerobic organisms, including tetanus, staphylococci and streptococci. Puncture and crush wounds are more prone to infection than laceration. Up to 25% of dog bite wounds become infected, with the first signs appearing in about 24 hours.[4]

Principles of treatment (see FIG. 123.11):

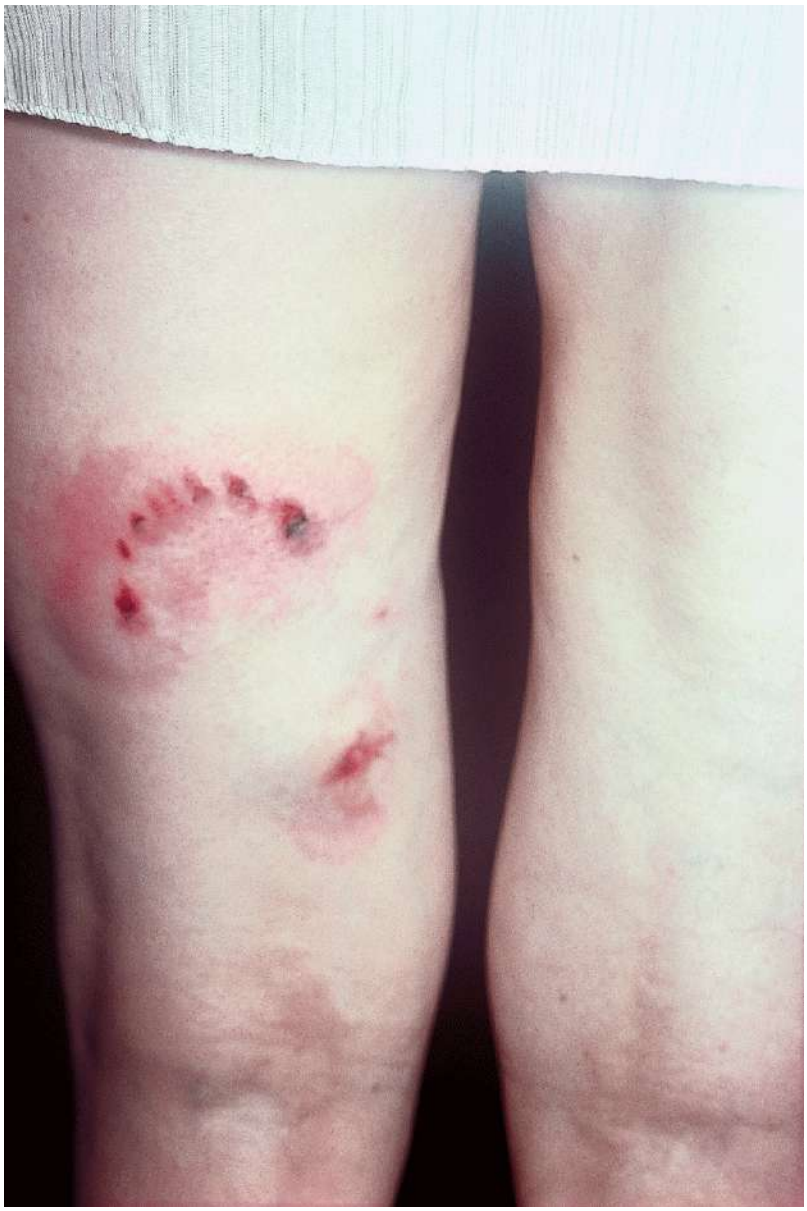

FIGURE 123.11 Dog bite treated by sterile dressing and anti-tetanus vaccination

- Clean and debride the wound with aqueous antiseptic, allowing it to soak for 10–20 minutes.
- Aim for open healing—avoid suturing if possible (except in ‘privileged’ sites with an excellent blood supply such as the face and scalp).
- Apply non-adherent, absorbent dressings (paraffin gauze and Melolin) to absorb the discharge from the wound.
- Tetanus prophylaxis: immunoglobulin or tetanus toxoid.

- A 2008 Cochrane review found no evidence supporting prophylactic antibiotics for dog or cat bites.[2] Small wounds presenting within 8 hours that do not involve joints or tendons do not require antibiotics.
- However, for a severe or deep bite, give prophylactic penicillin: 1.5 million units procaine penicillin IM statim, then orally for 5–10 days. An alternative is amoxicillin/clavulanate for 5–7 days. Use this antibiotic for 7–10 days for an established infection (depending on swab).[4]
- Inform the patient that slow healing and scarring are likely.

Rabid or possibly rabid dog (or other animal)

Not currently applicable in Australia (see CHAPTER 129).

- Wash the site immediately with detergent or saline (preferable) or hydrogen peroxide or soap (if no other option).
- Do not suture.
- If rabid:

  human rabies immune globulin (passive)

  antirabies vaccine (active)
- Uncertain: capture and observe animal, consider vaccination.

Cat bites

Cat bites have the greatest potential for suppurative infection, with *Pasteurella multocida* being the most common organism. The same principles apply as for the management of human or dog bites. For deep or delayed wounds, use amoxicillin + clavulanate for prophylaxis for 5 days. For infection, swab the wound but start with metronidazole + doxycycline or ciprofloxacin.[3] It is important to clean a deep and penetrating wound. Another problem is cat-scratch disease, presumably caused by a Gram-negative bacterium, *Bartonella henselae.*

Clinical features of cat-scratch disease

- An infected ulcer or papule pustule at bite site (30–50% of cases) after 3 days or so[5]
- 1–3 weeks later: fever, headache, malaise, regional lymphadenopathy (may suppurate)
- Intradermal skin test positive
- Benign, self-limiting course usually not requiring antibiotic treatment

- Sometimes severe symptoms for weeks, especially in immunocompromised
- For lymphadenopathy unresolved after 1 month, or significant morbidity, treat with erythromycin or roxithromycin for 10 days[3]

Water-related wound infections[3]

These complex infections may require expert advice.

Coral cuts

Wounds from coral cuts are at risk of serious infection with *Vibrio* organisms (marine pathogens) or *Streptococcus pyogenes.* Such wounds require cleaning with antiseptics, debridement, dressing and antibiotic cover with doxycycline 100 mg bd or cephalexin 500 mg bd for 7 days.

Fish tank/swimming pool granuloma

Due to *Mycobacterium marinum*, which causes a localised papular or nodular skin lesion, usually in people who clean aquaria or swimming pools. Diagnosis is by biopsy and culture (acid-fast bacilli).

Treatment: single lesion excision (may suffice). May need antibiotic therapy, e.g. clarithromycin (o) bd for 3–4 months plus rifampicin for severe or unresponsive infection. Seek expert advice.

Aeromonas species wound infections

From fresh or brackish water or mud exposure to open wounds. Treat with ciprofloxacin for 14 days.[3]

Shewanella putrefaciens

From salt or brackish water, particularly legs with vascular compromise. Causes severe cellulitis with necrosis, even sepsis. Treat with ciprofloxacin or meropenem/imipenem.

Scalp lacerations in children

If lacerations are small but gaping use the child's hair for the suture, provided it is long enough.

Method

Make a twisted bunch of the child's own hair on each side of the wound. Tie a reef knot and then an extra holding knot to minimise slipping. Ask an assistant to drop compound benzoin tincture solution (Friar's Balsam) on the hair knot. Leave the hair 'suture' long and get the parent to cut the knot in 5 days.

Forehead and other lacerations in children

Despite the temptation, avoid using reinforced paper adhesive strips (Steri-Strips) for children with open wounds. They will merely close the dermis and cause a thin, stretched scar. They can be used only for very superficial epidermal wounds, or in conjunction with sutures.

Adhesive glue for wound adhesion

A tissue adhesive glue can be used successfully to close superficial smooth and clean skin wounds, particularly in children. Commercial preparations such as Histoacryl, Dermabond and Epiglu (active ingredient enbucrilate) are available. SuperGlue also serves the purpose although sterility and toxicity have to be considered. The glue should be used only for superficial, dry, clean and fresh wounds. No gaps are permissible with this method; glue in the wound will block healing. Avoid glues if possible.

Wound anaesthesia in children

Consider topical anaesthetic preparations for wound repair in children. They include lignocaine and prilocaine mixture (Emla cream) and adrenaline and cocaine (AC) liquid. Use the latter with caution.

Some practitioners use an ice block to freeze the lacerated site, with or without a little lignocaine dripped into the wound. The child is then asked to hold the ice in their hand as a distraction while a suture is rapidly inserted.

Removal of skin sutures

Suture marks are related to the time of retention of the suture, its tension and position. The objective is to remove the sutures as early as possible, as soon as their purpose is achieved. The timing of removal is based on common sense and individual cases. Silk and other polyfilament sutures are more reactive and should be removed early. After suture removal, it is advisable to support the wound with Micropore skin tape/Steri-Strips for 1–2 weeks, especially in areas of skin tension.

Method

1. Use good light and have the patient lying comfortably.
2. Use fine, sharp scissors that cut to the point, or a disposable suture cutter blade, and a pair of fine, non-toothed dissecting forceps that grip firmly.
3. Cut the suture close to the skin below the knot with scissors or blade (see FIG. 123.12A).
4. Gently pull the suture out towards the side on which it was divided—that is, always towards

the wound (see FIG. 123.12B).

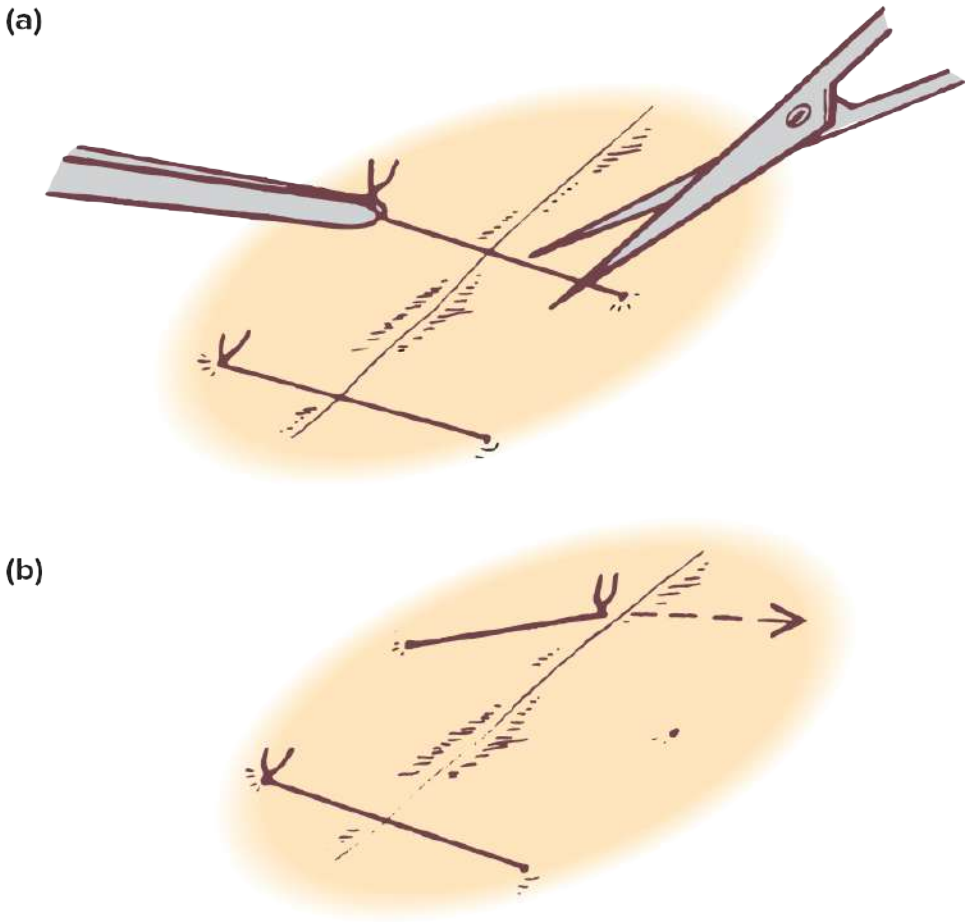

FIGURE 123.12 Removal of skin sutures: **(a)** cutting the suture, **(b)** removal by pulling towards wound

Blood clot obscuring a suture can be removed after soaking for a minute or two in lubricant gel. If necessary, the bevel edge of a 21 gauge needle can 'saw' through a buried suture.

When to remove non-absorbable sutures

For removal of sutures after non-complicated wound closure in adults, see TABLE 123.2 .

Table 123.2 Time after insertion for removal of sutures

Area	Days later
Scalp	6

Face	3 (or alternate at 2, rest 3–4)
Ear	5
Neck (anterior)	4 (or alternate at 3, rest 4)
Chest	8
Arm (including hand and fingers)	8–10
Abdomen	8–10 (tension 12–14)
Back	12
Inguinal and scrotal	7
Perineum	2
Legs	10
Knees and calf	12
Foot (including toes)	10–12

Note: Decisions need to be individualised according to the nature of the wound and health of the patient and healing. In general, take sutures out as soon as possible. One way of achieving this is to remove alternate sutures a day or two earlier and remove the rest at the usual time. Steri-Strips can then be used to maintain closure and healing.

Additional aspects

In children, tend to remove 1–2 days earlier. Allow additional time for backs and legs, especially the calf. Nylon sutures can be left in longer because they are less reactive. Alternate sutures may be removed earlier (e.g. face in women).

Burns[5,6]

Management depends on extent and depth (burns are classified as superficial or deep—otherwise first, second or third degree).

First degree burns are superficial and involve only the epidermis, causing pain, redness and swelling. A scald, which is a burn caused by moist heat, is an example. Healing proceeds quickly.

Second degree or partial skin thickness burns cause the epidermis to blister and become necrotic with subsequent serous ooze.

In *third degree* or full thickness burns, there is deep necrosis and perhaps anaesthesia from destroyed nerve endings. If extensive (>9% of body surface area) and deep, there is a possibility of hypovolaemia and shock.

A major burn, which is a medical emergency, is an injury of more than 20% of the total body surface for an adult and 10% for a child.

First aid

The immediate treatment of burns, especially for smaller areas, is immersion in cool to cold, running water such as tap water, for a minimum of 20 minutes. Do not disturb charred adherent clothing but remove wet clothing.

Chemical burns should be liberally irrigated with water. Apply 1 in 10 diluted vinegar to alkali burns and sodium bicarbonate solution for acid burns.

Refer the following burns to hospital:

- >9% surface area, especially in a child (see 'rule of nines', CHAPTER 111); the patient's hand size equals approximately 1%[7]
- >5% in an infant
- all deep burns
- burns of difficult or vital areas (e.g. face, hands, perineum/genitalia, feet)
- burns with potential problems (e.g. electrical, chemical, circumferential)
- suspicion of inhalational injury

Always give adequate pain relief. During transport, continue cooling by using a fine-mist water spray.

Treatment

1. *Very superficial—intact skin.* Can be left with application of a mild antiseptic only (e.g. aqueous chlorhexidine). Review if blistering.
2. *Superficial—blistered skin.* Apply a dressing to promote epithelialisation (e.g. hydrocolloid sheets, hydrogel sheets) covered by an absorbent dressing *or* (best option) a retention adhesive material (e.g. Fixomull, Mefix, Hypafix) with daily or twice daily cleaning of the serous ooze and reapplication of outer stretch bandage. Fixomull can be left in place for up to 2 weeks.

Guidelines to patient for retention dressings

- First 24 hours: keep dry. Pat dry any ooze coming through the dressing with a clean tissue.

- From day 2: wash over dressing twice daily. Use gentle soap and water, rinse then pat dry. Do not soak. Rinse only. Do not remove the dressing as it may cause pain and damage to the wound. If the wound becomes red, hot or swollen, or if pain increases, return to the clinic.
- From day 7: return to the clinic for removal of the dressing. Two hours prior to coming into the clinic, soak the dressing with olive oil then cover with cling wrap (e.g. GLAD Wrap).

Note: Dressing must be soaked off with oil (e.g. olive, baby, citrus or peanut). Debride 'popped blisters'. Only pop blisters that interfere with dermal circulation.

3. *Deep burns.* If considerable ooze, apply the following in order:

- SoloSite gel, Solugel or similar
- non-adherent neutral dressing (e.g. Melolin)
- layer of absorbent gauze or cotton wool (larger burns)

Change every 2–4 days with analgesic cover. Surgical treatment, including skin grafting, may be necessary.

The Alfred Hospital publishes excellent burns guidelines at www.vicburns.org.au.

Exposure (open method)

- Keep open without dressings (good for face, perineum or single surface burns)
- Renew coating of antiseptic cream every 24 hours

Dressings (closed method)

- Suitable for circumferential wounds
- Cover area with non-adherent tulle (e.g. paraffin gauze)
- Dress with an absorbent, bulky layer of gauze and wool
- Use a plaster splint if necessary

Burns to hands

For superficial blistered burns to the hand or similar 'complex' shaped parts of the body apply strips of the retention stretch adhesive dressings as described above. They conform well to digits. Apply an outer bandage. At 7 days, soak the dressings in oil for 2 hours prior to coming in to the

clinic.

Foreign bodies

Penetrating gun injuries

Injuries to the body from various types of guns present decision dilemmas for the treating doctor. The following information represents guidelines, including special sources of danger to tissues from various foreign materials discharged by guns.

Gunshot wounds

Airgun

The rule is to remove subcutaneous slugs but to leave deeper slugs unless they lie within and around vital structures (e.g. the wrist). A special common problem is that of slugs in the orbit. These often do little damage and the ophthalmologist may decide to leave them alone.

0.22 rifle (the pea rifle)

The same principles of management apply but the bullet must be localised precisely by X-ray. Of particular interest are abdominal wounds, which should be observed carefully, as visceral perforations can occur with minimal initial symptoms and signs.

0.410 shotgun

The pellets from this shotgun are usually dangerous only when penetrating from a close range. Again, the rule is not to remove deep-lying pellets—perhaps only those superficial pellets that can be palpated.

Pressure gun injuries

Injection of grease, oil, paint and similar substances from pressure guns (see FIG. 123.13) can cause very serious injuries, requiring decompression and removal of the substances.

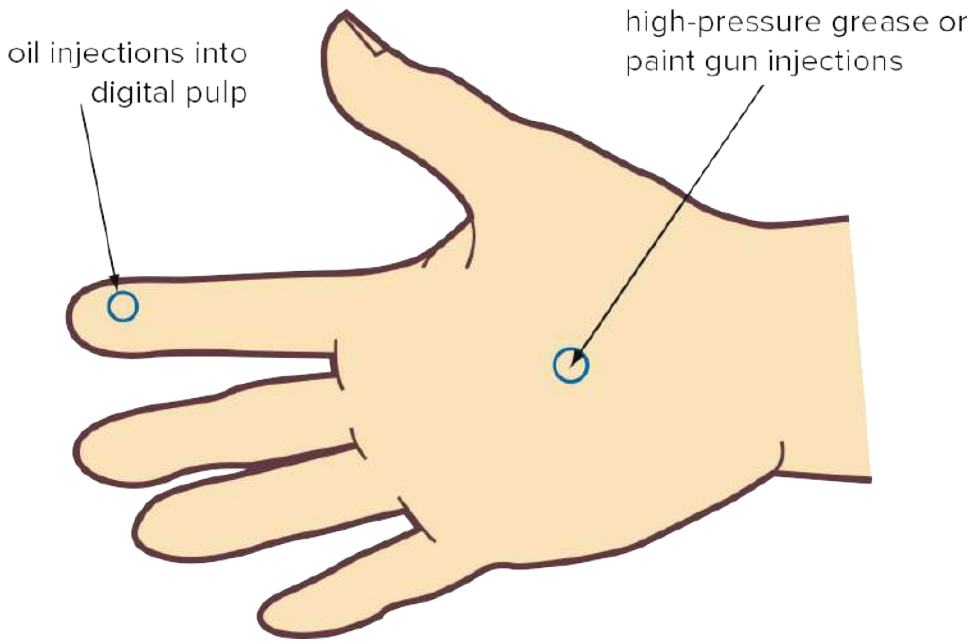

FIGURE 123.13 Dangerous accidental injections into the hand

Grease gun and paint gun

High-pressure injection of paint or grease into the hand requires urgent surgery if amputation is to be avoided. There is a deceptively minor wound to show for this injury, and after a while the hand feels comfortable. However, ischaemia,[1] chemical irritation and infection can follow, with gangrene of the digits, resulting in, at best, a claw hand due to sclerosis. Treatment is by immediate decompression and meticulous removal of all foreign material and necrotic tissue.

Oil injection

Accidental injection of an inoculum in an oily vehicle into the hand also creates a serious problem with local tissue necrosis. Such injections are common on poultry farms during fowl-pest injections. If injected into the digital pulp, this may necessitate amputation.

Splinters under the skin

The splinter under the skin is a common and difficult procedural problem. Instead of using forceps or making a wider excision, use a disposable hypodermic needle to ‘spear’ the splinter (see FIG. 123.14) and then use it as a lever to ease the splinter out through the skin.

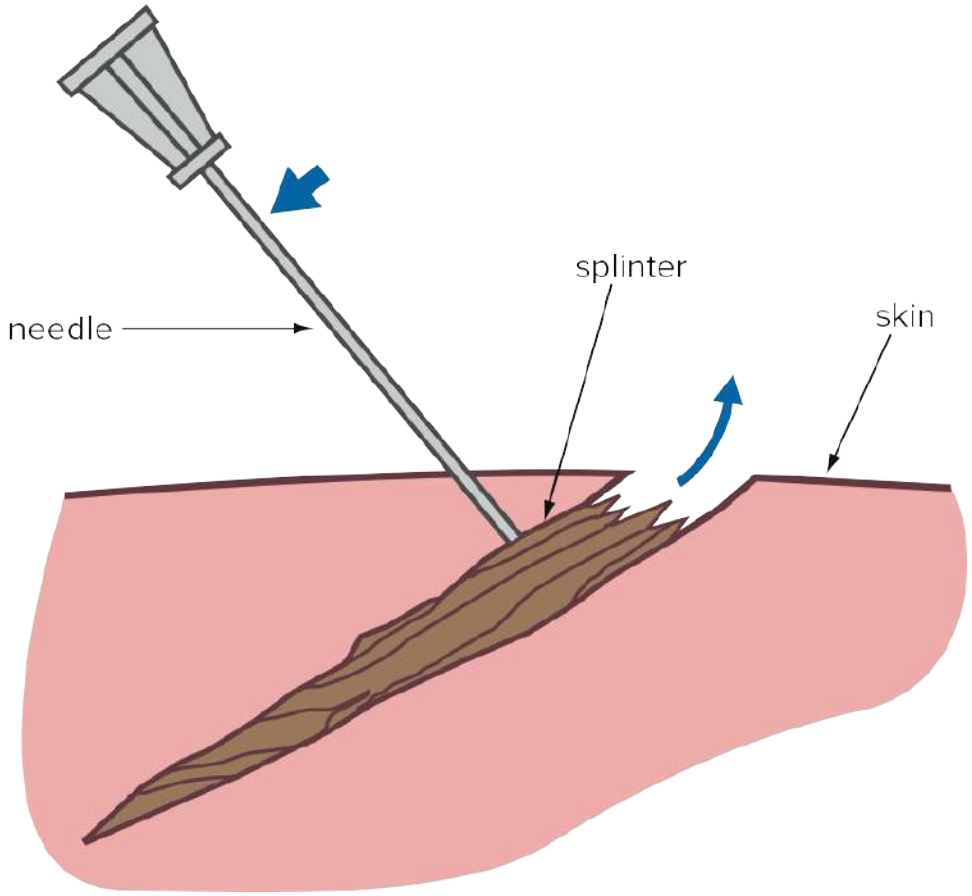

FIGURE 123.14 Removal of splinters in the skin

A ‘buried’ wooden foreign body can be detected by ultrasound.

Embedded fish hooks

Two methods of removing fish hooks are presented here, both requiring removal in the reverse direction, against the barb. Method 2 is recommended as first-line management.

Method 1

1. Inject 1–2 mL of LA around the fish hook.
2. Grasp the shank of the hook with strong artery forceps.
3. Slide a D11 scalpel blade in along the hook, sharp edge away from the hook, to cut the tissue and free the barb (see FIG. 123.15).
4. Withdraw the hook with the forceps.

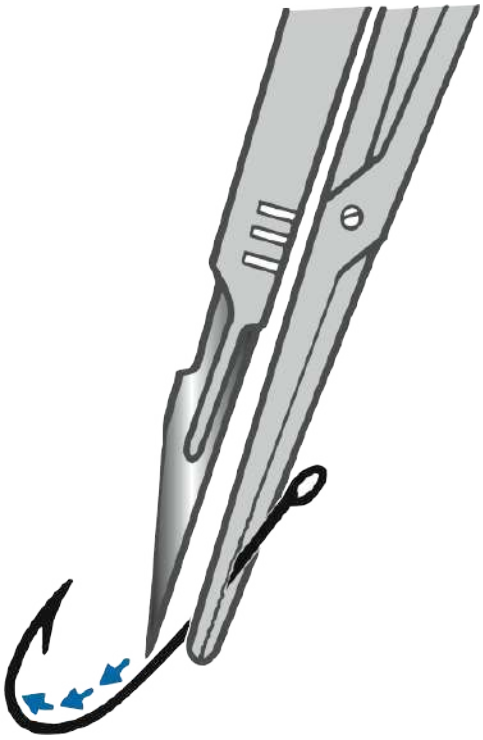

FIGURE 123.15 Removal of fish hook by cutting a path in the skin

Method 2

This method, used by some brave fishermen, relies on a loop of cord or fishing line to forcibly disengage and extract the hook intact. It requires no anaesthesia and no instruments—only nerves of steel, especially for the first attempt.

1. Take a piece of string about 10–12 cm long and make a loop. One end slips around the hook, the other hooking around one finger of the operator.
2. Depress the shank with the other hand in the direction that tends to disengage the barb.
3. At this point give a very swift, sharp tug along the cord.
4. The hook flies out painlessly in the direction of the tug (see FIG. 123.16). Wear safety goggles!

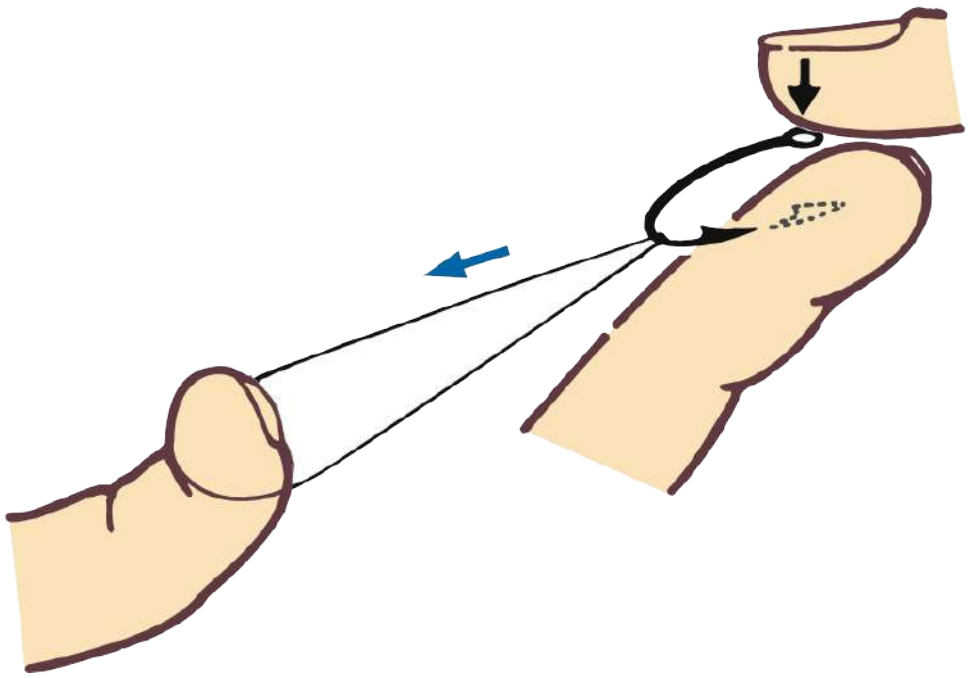

FIGURE 123.16 Fisherman's method of removing a fish hook intact

Note: You must be bold, decisive, confident and quick—half-hearted attempts do not work.

For difficult cases, some local anaesthetic infiltration may be appropriate. Instead of a short loop of cord, a long piece of fishing line double looped around the hook and tugged by the hand, or flicked with a thin ruler in the loop, will work.

Needle-stick and sharps injuries

Accidental skin puncture by contaminated 'sharps', including needles (with blood or bloodstained body fluids), is of great concern to all health care workers. Another problem that occurs occasionally is the deliberate inoculation of people such as police by angry, sociopathic individuals. A needle-stick accident is the commonest incident with the potential to transmit infections such as HIV and hepatitis B, C or D. The part of the venipuncture that is most likely to cause the accident is the recapping or resheathing of the needle. This practice should be discouraged.

Infections transmitted by needle-stick accidents are summarised in TABLE 123.3 . The risk from a contaminated patient is greatest with hepatitis (10–30%), while the risk of seroconversion or clinical infection after a needle-stick injury with HIV-positive blood is very low (probably about 1 in 300).[8] In children injured by needle stick incidentally in the community (e.g. at the beach or park), there are no published reports of transmission of a blood-borne virus such as HIV or hepatitis B or C.[9] The risk of tetanus, especially for outdoor injuries, is significant and should be addressed.

Table 123.3 Infections transmitted by needle-stick accidents

Viruses
HIV
Hepatitis B, C, D
Herpes simplex
Herpes varicella zoster
Bacteria
Streptococcus
Staphylococcus
Syphilis
Tetanus
Tuberculosis
Other
Malaria

Prevention

- Avoid physically struggling with overdose victims or high-risk patients for lavage or venipuncture.
- Do not recap needles.
- Dispose of needles immediately and directly into a leak-proof, puncture-proof sharps container.
- Avoid contact with blood.
- Wear protective gloves (does not prevent sharps injury).

Postexposure prophylaxis

Postexposure prophylaxis (PEP) must be considered for high-risk exposure such as hollow blood-containing needles, deep injury and high viral load or late-stage HIV infection of the source.[3] Although PEP is not justified for most sharps injuries, do seek expert advice.

Management[10]

- Wash affected site with soap and water without scrubbing. Also wash or irrigate any areas of skin, eye/conjunctiva or other mucous membrane exposed to blood or body fluids.
- Do not suck or squeeze wound.
- Encourage bleeding.
- Reassure the patient that the risk of viral infection is very low.
- Obtain information about and blood from the sharps victim and the source person (source of body fluid). A known carrier of hepatitis B surface antigen or an HIV-positive source person will facilitate early decision making.

Note: It takes 3 months to seroconvert with HIV so the patient may be infected but negative on initial tests.

Consider the exposed person's wishes after discussing the risks/benefits of treatment including adverse effects.

Known hepatitis B carrier source person

- If injured person is immune—no further action
- If non-vaccinated and non-immune:

  give hyperimmune hepatitis B gammaglobulin within 48 hours

  commence course of hepatitis B vaccination within 24 hours

Known hepatitis C carrier source person[10]

- The recipient needs to have follow-up HCV antibody tests at 1 and 6 weeks and ALT levels at 4–6 months.
- There is no effective immunoprophylaxis available. Consider early therapy should seroconversion occur.

Known HIV-positive source person[10]

Refer to consultant about relative merits of drug prophylaxis (ART) and serological monitoring. A case control study by the US Centers for Disease Control and Prevention indicated that giving zidovudine following a needle-stick injury decreases the rate of HIV seroconversion by 79%.[11]

HIV postexposure prophylaxis consists of 2–3 anti-retroviral medications administered for 28 days. Seek advice urgently, because prophylaxis is ideally commenced within 2 hours of exposure.

Unknown risk source person

Take source person's blood (if consent is given) and sharps victim's blood for hepatitis B (HBsAg and anti-HBs) and hepatitis C and HIV status tests. Commence hepatitis B vaccination if not vaccinated.

Note: Informed consent for testing and disclosure of test results for involved person should be obtained.

Tetanus prophylaxis

Tetanus is a very serious disease but completely preventable by active immunisation. Immunisation should be universal, as per the childhood and adult immunisation program. However, all patients with wounds should be assessed for their tetanus status and managed on their merits. For severe wounds, the possibility of gas gangrene should also be considered.

Tetanus-prone wounds:

- compound fractures
- penetrating injuries
- foreign bodies
- extensive crushing
- delayed debridement
- severe burns
- pyogenic infection

For the primary immunisation of adults, tetanus toxoid (available combined with diphtheria and, often, pertussis) is given as two doses 6 weeks apart, with a third dose 6 months later. Booster doses of tetanus toxoid are given as an adolescent and at age 50. Give a booster dose at the time of major injury unless it is less than 5 years since the previous dose.[12]

Passive immunisation

Passive immunisation, in the form of tetanus immunoglobulin 250 units by IM injection, is reserved for non-immunised individuals or those of uncertain immunity wherever the wound is contaminated or has devitalised tissue. It should be given as soon as possible, on the day of injury. Wounds at risk include those contaminated with dirt, faeces/manure, soil, saliva or other foreign material; puncture wounds; and wounds from missiles, crushes and burns.

The guide is outlined in TABLE 123.4 .

Table 123.4 Guide to tetanus prophylaxis in wound management[3]

Time since vaccination	Type of wound	Tetanus toxoid	Tetanus immunoglobulin
History of 3 or more doses of tetanus toxoid			
<5 years	All wounds	No	No
5 to 10 years	Clean minor wounds	No	No
	All other wounds	Yes	No
>10 years	All wounds	Yes	No
Uncertain vaccination history or less than 3 doses of tetanus toxoid			
	Clean minor wounds	Yes	No
	All other wounds	Yes	Yes

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124 Common fractures and dislocations

The broken bone, once set together, is stronger than ever.

JOHN LYLY (1554–1606)

Common fractures and dislocations usually apply to the limbs, the shoulder girdle and the pelvic girdle and their management requires an early diagnosis to ensure optimum treatment and to prevent complications. Early diagnosis depends on the physician being vigilant and on having knowledge of the less common conditions so that a careful search for the diagnosis can be made.

The diagnosis is dependent on a good history followed by a careful examination, good-quality X-rays appropriate to the injury (e.g. stress view) and, if necessary, special investigations. The family doctor should develop the habit of looking at X-rays, as back-up to the radiologist's report, to help avoid missed diagnoses.

The usual rule is: if in doubt, X-ray. However, in response to over-ordering of X-rays in many circumstances where a fracture is very unlikely, handy algorithms have been developed, such as the Ottawa Ankle and Knee rules and the Canadian C-spine Rule.[1]

There are many pitfalls involved in managing fractures and dislocations. Many injuries, such as fractures of the arm and hand, may seem trivial but can lead to long-term disability. This chapter presents guidelines to help avoid these pitfalls.

Key facts and checkpoints

- The classic signs of fracture are:
  - pain
  - tenderness
  - loss of function
  - deformity
  -
  -

swelling/bruising

crepitus

- A fracture usually causes deformity but may cause nothing more than local tenderness over the bone (e.g. scaphoid fracture, impacted fractured neck of femur).
- X-ray examination of the upper limb should include views of joints proximal and distal to the site of the injury, and X-rays in both AP and lateral planes.
- If an X-ray is reported as normal but a fracture is strongly suspected, one option is to splint the affected limb for about 10 days and then repeat the X-ray.
- As a rule, displaced fractures must be reduced whereby bone ends are placed in proper alignment and then immobilised until union occurs.
- Fractures prone to loss of position should be monitored radiologically, particularly in the first 1–2 weeks following reduction.
- Bone union is assessed clinically by reduced pain at the fracture site and reduced fracture mobility. It is assessed radiologically by X-ray features such as trabecular continuity across the fracture site and bridging callus.
- Non-union is caused by such factors as inadequate immobilisation, excessive distraction, loss of healing callus, infection or avascular necrosis.
- Stiffness of joints is a common problem with immobilisation in plaster casts and slings, so the joints must be moved as early as possible. Early use is possible if the fracture is stable. Frozen shoulders are a particular complication in the elderly with an upper limb fracture.
- A dislocation is a complete disruption of one bone relative to another at a joint.
- A subluxation is a partial displacement such that the joint surfaces are still in partial contact.
- A sprain is a partial disruption of a ligament or capsule of a joint.
- Always consider associated soft-tissue injuries such as neuropraxia to adjacent nerves, vascular injuries and muscle compartment syndromes.
- The key strategy of most reduction manoeuvres is traction, especially for dislocations. This may be supplemented with translation or leverage.
- A stress fracture is an incomplete fracture resulting from repeated small episodes of trauma, which individually would be insufficient to damage the bone. Stress fractures, especially in the foot, are most likely to result from sport, ballet,

gymnastics and aerobics. These overuse injuries were initially described as ‘march’ fractures in military recruits with rapidly increased exercise levels.[2]

- Typical stress fractures (with their usual cause) include:

  navicular (sprinting sports, football)

  metatarsal neck (running, walking, basketball, jumping)

  base of fifth metatarsal (dancing)

  femur—neck or shaft (distance running)

  ulna (weight-lifting)

  distal radial and ulnar epiphyses (gymnastics)

  talus (running)

  proximal tibia (running, football)

  lumbar spine

  medial tibia (running, football)

  distal phalanges (guitar playing)

  cervical spinous process (gardening)

  lumbar vertebrae—pars interarticularis (fast bowling)

  spiral humerus (throwing sports)

  rib—1st (weight-lifting)

  rib—8th (tennis)

Easily missed fractures: red flags

- Supracondylar fracture in children
- Elbow fractures in children, especially lateral humeral condyle
- Trampoline injuries in children
- Scaphoid fracture

- Scapholunate dislocation
- Skull fractures, especially temporal
- Talar dome fractures
- All intra-articular fractures
- Avascular heads of humerus and femur

Testing for fractures[3]

This method describes the simple principle of applying axial compression for the clinical diagnosis of fractures of bones of the forearm and hand, but also applies to other limb bones.

Many fractures are obvious when applying the classic methods of diagnosis but it is sometimes more difficult if there is associated soft-tissue injury from a blow or if there is only a minor fracture such as a greenstick fracture of the distal radius.

A soft-tissue injury of the forearm will, like a fracture, show pain, tenderness, swelling and possibly loss of function. It will not, however, be painful if the bone is compressed axially—that is, in its long axis. Therefore, asking about pain while compressing the bone from end to end (avoiding any direct pressure on the traumatised area) is a useful way to highlight a fracture.

Walking is another method of applying axial compression; pain makes walking very difficult in the presence of a fracture in the weight-bearing axis or pelvis.

Method

1. Grasp the affected area both distally and proximally with your hands.
2. Compress along the long axis of the bones by pushing in both directions, so that the forces focus on the affected area (fracture site, see FIG. 124.1A). Alternatively, compression can be applied from the distal end with stabilising counterpressure applied proximally (see FIG. 124.1B).
3. The patient will accurately localise the pain at the fracture site.

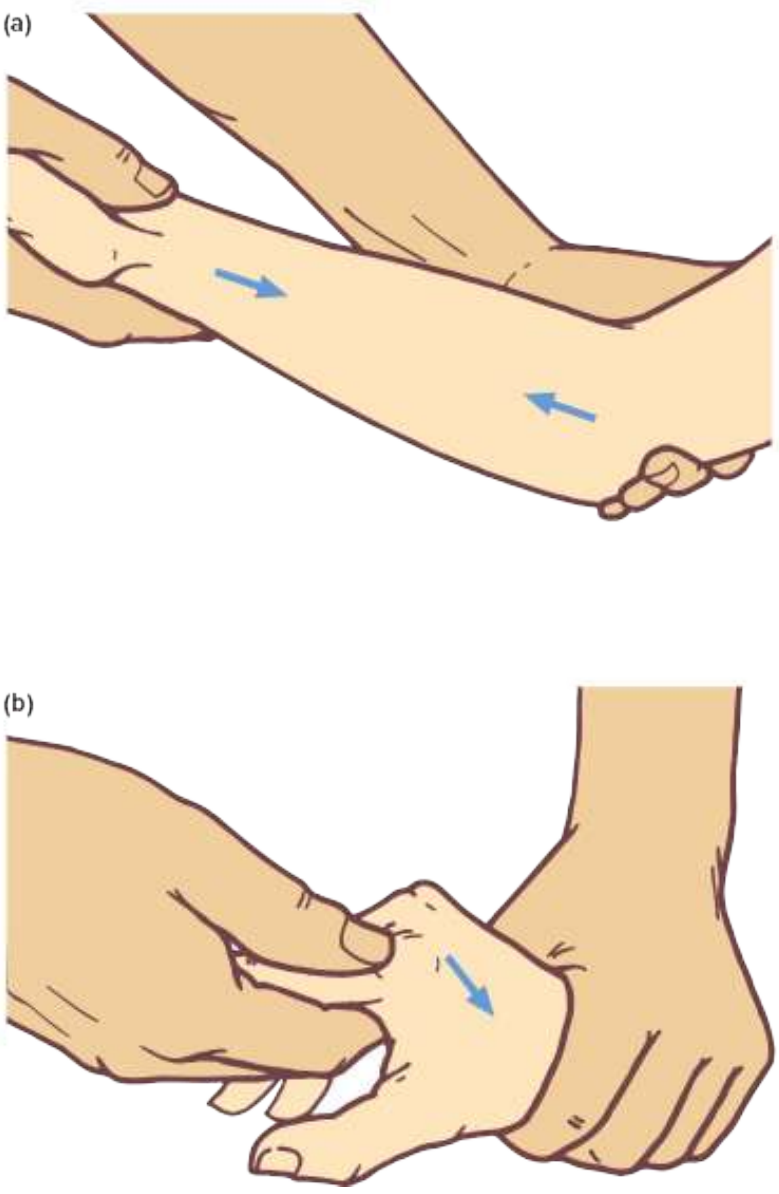


FIGURE 124.1 Testing for fractures: **(a)** axial compression to detect a fracture of the radius or ulna bones, **(b)** axial compression to detect a fracture of the metacarpal

Treatment of head, spine and chest fractures

Injuries of the skull and face

Skull fractures

Closed fractures without any neurological symptoms do not require active intervention. Depressed fractures may require elevation of the depressed fragment. Compound fractures of the vault require careful evaluation and referral. Special care is required over the midline as manipulation (usually by elevation) of any depressed fragment can tear the sagittal sinus, causing profuse and fatal bleeding. Beware of the associated extradural or subdural haematoma (see CHAPTER 64).

Base of skull fractures

These fractures are difficult to diagnose on radiography but their presence is indicated by bleeding from the nose, throat or ears, or by ‘raccoon eyes’. CSF may be observed escaping, especially through the nose, if the dura is also torn.

Treatment of basal fractures is based on looking for and treating intracranial infection, and avoidance of excessive interference with the nose or ear; avoid packing and nasogastric tubes. There is no role for antibiotic prophylaxis as this does not reduce the risk of meningitis.[4]

Malar fracture

A fractured zygomaticomaxillary complex (malar) is a common body contact sports injury or injury resulting from a fight. See FIGURE 124.2 .

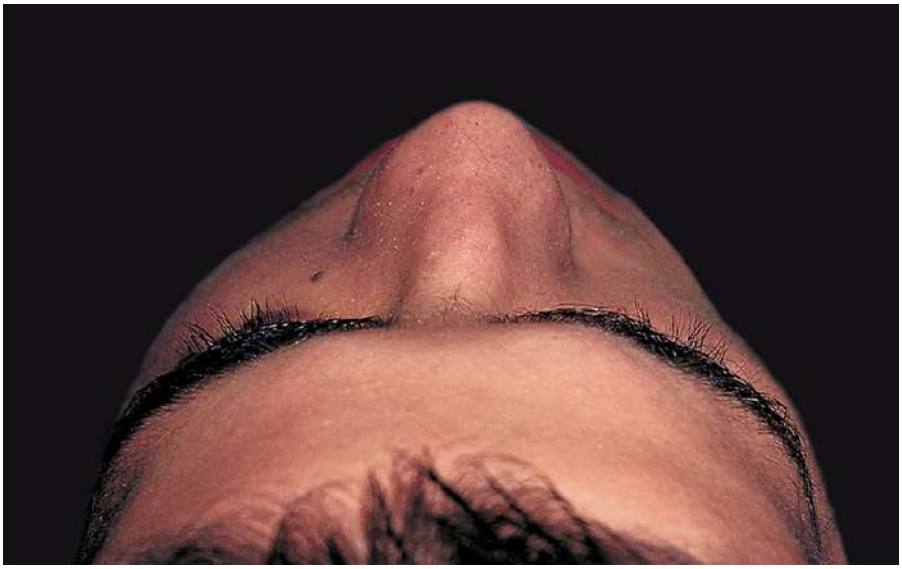


FIGURE 124.2 Fractured malar (left) showing circumocular haematoma and depression of the infraorbital margin

Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ, eds. *The Atlas of Emergency Medicine* (4th ed). New York: McGraw-Hill, 2016. Photo contributor: Edward S. Amrhein, DDS

Clinical features

- Swelling of cheek
- Circumocular haematoma
- Subconjunctival haemorrhage
- Palpable step in infraorbital margin
- Flat malar eminence when viewed from above
- Paraesthesia due to infraorbital nerve injury
- Loss of function (i.e. difficulty opening mouth)

Management

- Head injury assessment
- Exclude ‘blow-out’ fracture of the orbit
- Exclude ocular trauma:
    - remove contact lenses if worn
    - check visual acuity
    - check for diplopia
    - check for hyphaema
    - check for retinal haemorrhage
- Persuade patient not to blow nose (can cause surgical emphysema)
- If fracture displaced, refer for reduction under general anaesthesia

Reduction methods

- Elevation by temporal or intraoral approach—healing can be expected in 3–4 weeks
- Some require interosseous wiring or plating or pinning

Fracture of mandible

A fracture of the mandible follows a blow to the jaw. The patient may have swelling (which can vary from virtually none to severe), pain, deformity, inability to chew, malalignment of the jaw and teeth and drooling of saliva. Intraoral examination is important as submucosal ecchymosis in the floor of the mouth is a pathognomonic sign.

A simple office test for a suspected fractured mandible is to ask patients to bite on a wooden tongue depressor (or similar firm object). Ask them to maintain the bite as you twist the spatula. If they have a fracture they cannot hang onto the spatula because of the pain.[5]

X-rays:

- AP views and lateral obliques
- an orthopantomogram provides a global view

First aid management

- Check the patient's bite and airway
- Remove any free-floating tooth fragments and retain them
- Replace any avulsed or subluxed teeth in their sockets

Note: Never discard teeth.

- First aid immobilisation with a four-tailed bandage, which can be made by splitting a bandage from both ends, leaving an intact central area to support the chin (see FIG. 124.3)

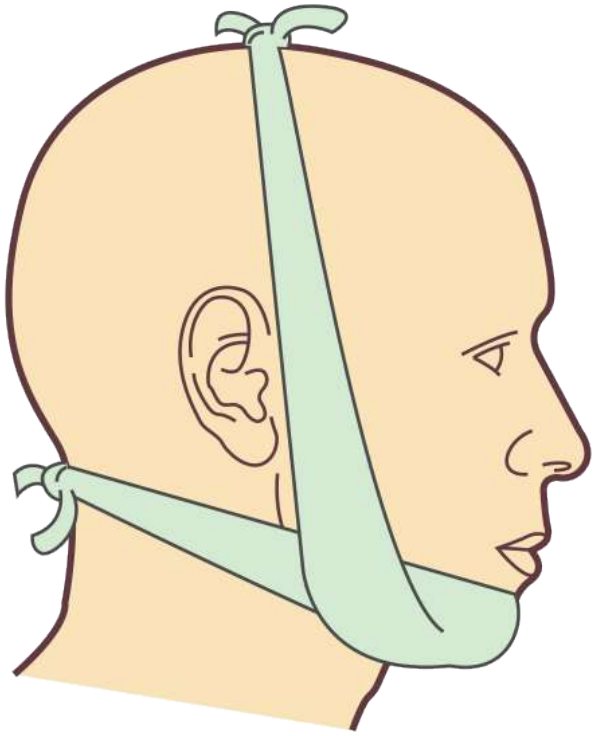


FIGURE 124.3 Immobilisation of a fractured mandible in a four-tailed bandage

Treatment

Refer for possible internal fixation.

A fracture of the body of the mandible will usually heal in 6–12 weeks (depending on the nature of the fracture and fitness of the patient).

Dislocated jaw

The patient may present with unilateral or bilateral dislocation. The jaw will be ‘locked’ and the patient unable to articulate or close the mouth. It is very distressing.

Method of reduction

- Get the patient to sit upright with the head against the wall. Wear protective gloves, if available.
- Wrap a handkerchief or cloth around both thumbs and place the thumbs over the lower molar teeth, with the fingers firmly grasping the mandible on the outside.
- Firmly thrusting with the thumbs, push downwards towards the floor (see FIG. 124.4).

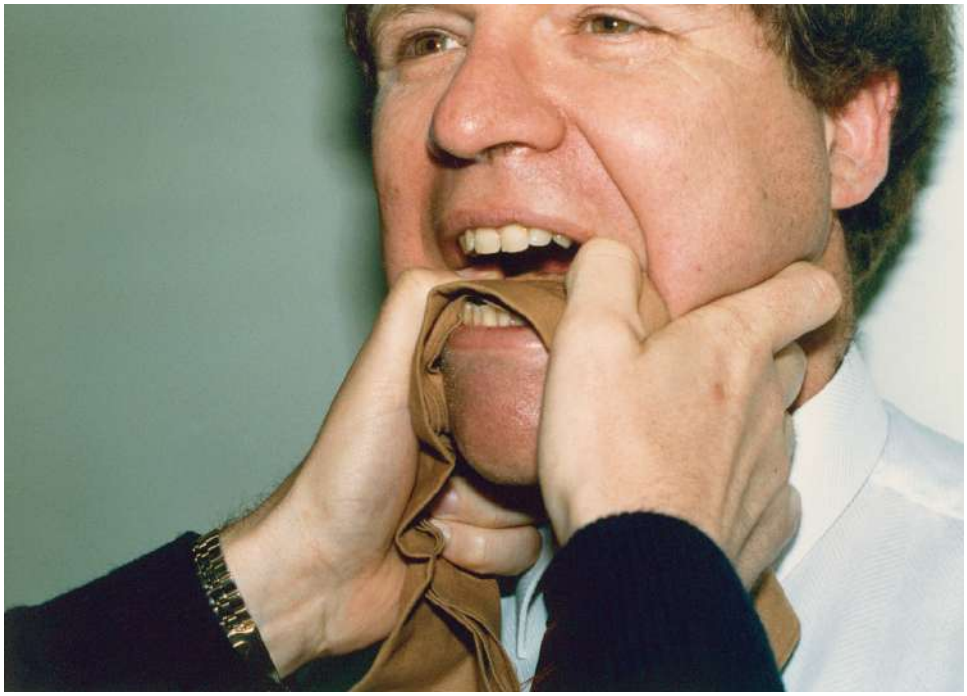


FIGURE 124.4 Method of reduction of a dislocated jaw by downward traction on the mandible. Wear gloves if available.

This action invariably reduces the dislocation, but the reduction can be reinforced by the fingers

rotating the mandible upwards as the thumbs thrust downwards.

Injuries of the spine

Cervical fractures, especially of the atlas (C1), axis (C2) and odontoid process, require early referral with the neck immobilised in a cervical collar, in a supine position. A hard collar is preferred, but a soft collar with sandbags on either side of the head to prevent movement will suffice.

Thoracolumbar fractures

Fractures or fracture dislocations of the thoracic and lumbar vertebrae, without neurological deficit, are classified as either stable or unstable. Due to the forces involved, these fractures often coexist with other injuries.

Stable fractures

- Compression fractures of vertebral body with <50% loss of vertical height
- Minor fractures
- Laminar fractures

Treatment: a custom-fitted back brace is worn, usually for 6–12 weeks, with physiotherapy involvement to restore function.

Special problems:

- retroperitoneal haematoma
- paralytic ileus
- associated kidney rupture with L1 fractures
- underlying vertebral body pathology in the elderly (e.g. myeloma or metastases)

Unstable fractures

Burst fractures and shearing fractures are usually unstable. They are often associated with partial or complete paraplegia and require urgent referral.

Fractures of sacrum and coccyx

No treatment apart from symptomatic treatment is required. Manual reduction per rectum can be attempted for significant forward displacement of the coccyx. Advise the use of a rubber ring or

special cushion (such as a Sorbo cushion) when sitting. For persistent coccydynia consider corticosteroid injection or excision.

Injuries of the thoracic cage

Fractured rib

Clinical features

- Pain over the fracture site, especially with deep inspiration and coughing
- Localised tenderness and swelling
- Pain in the site upon whole-chest compression (rib springing)
- X-ray confirms diagnosis and excludes underlying lung damage (e.g. pneumothorax). There is a high incidence of false-negative fractures on X-ray, so caution is necessary.
- Suspect splenic, hepatic and kidney trauma with lower rib fractures

Treatment

A simple rib fracture can be extremely painful. The first treatment strategy is to prescribe analgesics, such as paracetamol, and encourage breathing within the limits of pain. An intercostal nerve block is effective in the acute situation, but is very temporary. An elastic rib belt with Velcro fastening has often been used for single or double rib fractures, although the evidence for effectiveness is scanty (see FIG. 124.5).

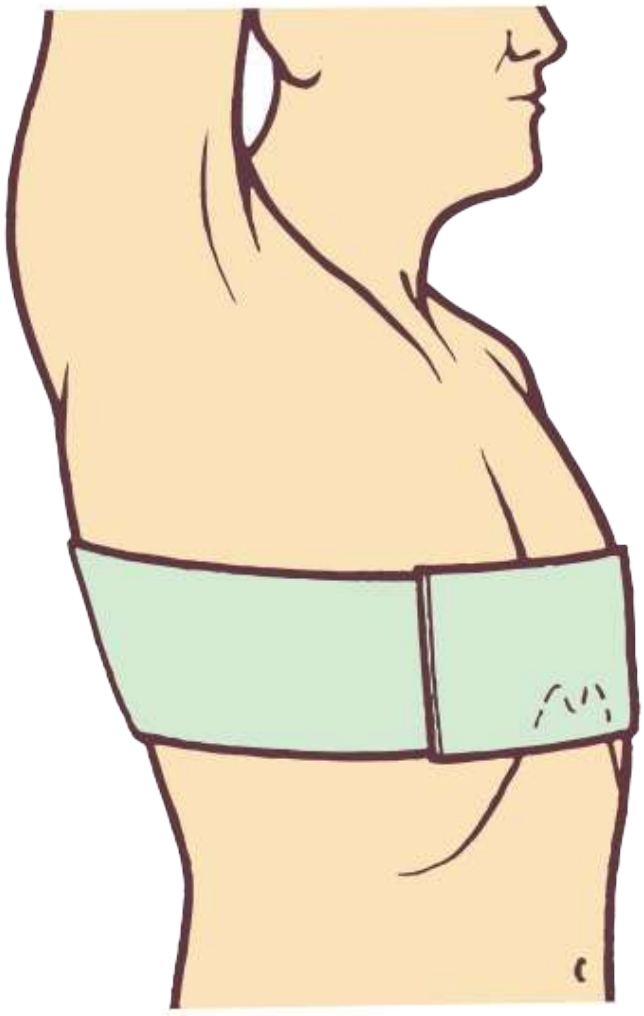


FIGURE 124.5 The universal rib belt

Healing time

Healing may take 3–6 weeks; local discomfort may persist much longer.

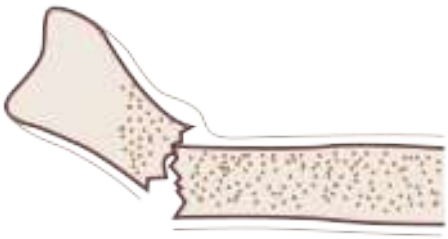
Fractures of the sternum

These are treated symptomatically with analgesics but careful evaluation of thoracic injuries, including cardiac tamponade or myocardial contusion, is essential. A significantly depressed fracture should be referred. An ECG is advisable.

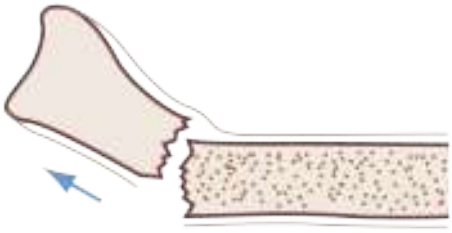
Treatment of limb fractures

To properly reduce any displaced fracture, the following steps must be taken (see FIG. 124.6A).[6]

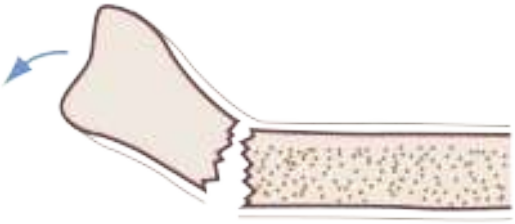
(a) fracture (impacted)



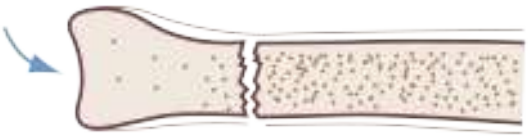
Step 1. Disimpaction



Step 2. Establish length



Step 3. Establish alignment



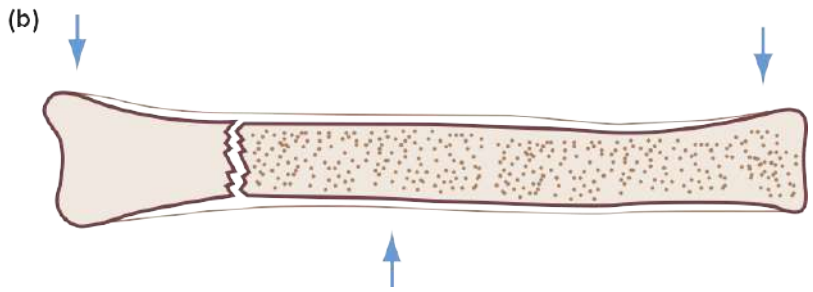


FIGURE 124.6 **(a)** Principles of reduction of fractured bones, **(b)** principles of moulding to maintain reduction: the arrows indicate the three-point pressure areas required to maintain reduction

1. Disimpact the fragments, usually by increasing the deformity.
2. Re-establish the correct length of the bone.
3. Re-establish the correct alignment by proper reduction of the fracture.
4. Stabilise the bone in an acceptable position for as long as it takes to heal.

The above steps will only be achieved with adequate anaesthesia, analgesia and relaxation. Maintenance of the reduction depends upon the moulding, which utilises the intact periosteal bridge to hold the fracture fragments in a reduced position. FIGURE 124.6B illustrates the principle of moulding to maintain reduction.[6]

Orthopaedic problems that cause difficulties in diagnosis and management are outlined in TABLE 124.1 .

Table 124.1 Important orthopaedic problems that cause difficulties in diagnosis and management[7]

Shoulders
Posterior dislocation of the shoulder
Recurrent subluxations
Unstable surgical neck fractures of humerus
The avascular humeral head
Elbow
Supracondylar fractures with forearm ischaemia
Fracture of the lateral humeral condyle in children
Fractured neck of radius in children

The Monteggia fracture with dislocation of radial head
Wrist
Scaphoid fractures
Scapholunate dislocation
The unstable Colles fracture
Fingers
Phalangeal fractures
Intra-articular fractures
Penetrating injuries of the MCP joint
Gamekeeper’s thumb (MCP joint)
The hip
Developmental dysplasia of the hip
Septic arthritis
Slipped capital femoral epiphysis
Subcapital fractures
Stress fractures of the femoral neck in athletes
Impacted subcapital femoral neck fracture in the elderly
Foot and ankle
Talar dome lesions
Stress fractures of the navicular
Intra-articular fractures

Fractures of the clavicle

This fracture typically follows a history of a fall onto the outstretched hand or elbow, although it may also occur with a direct blow to the clavicle or the point of the shoulder. The patient has pain aggravated by shoulder movement and usually supports the arm at the elbow and clasped to the chest. The most common fracture site is at the junction of the outer and middle thirds, or in the middle third. Consider the possibility of neurovascular injury.

Treatment

- St John’s elevated sling to support arm—for 3 weeks
- Figure-of-eight bandage (used mainly for severe discomfort)
- Early active exercises to elbow, wrist and fingers

- Active shoulder movements as early as possible

Special problem

Type II fracture at the lateral end of the clavicle that is displaced: this fracture, which usually occurs in elderly patients following low energy injuries,[8] is often subject to delayed or non-union. The line of fracture passes through the conoid and trapezoid ligaments. Consider referral for open reduction.

Healing time

Healing time is 4–8 weeks.

The appropriate use of slings for fracture-dislocations is presented in TABLE 124.2 .

Table 124.2 Appropriate use of slings for fracture–dislocations

Collar and cuff	Fractured shaft of humerus
Broad arm sling (hand horizontal)	Fractured forearm Fractured scapula
St John's high sling (hand points at opposite shoulder)	Fractured clavicle Fractured neck of humerus Subluxed acromioclavicular joint Dislocated acromioclavicular joint Subluxed sternoclavicular joint

Fractures of the scapula

Fractures of the scapula may include:

- body of scapula: due to a crushing force, considerable blood loss, may be rib fractures
- neck of scapula (may involve joint)
- acromion process (due to a blow or fall on the shoulder)
- coracoid process (due to a blow or fall on the shoulder)

Treatment

- Broad-based triangular sling for comfort

- Early active exercises for shoulder, elbow and fingers as soon as tolerable
- A large glenoid fragment usually requires surgical reduction because of potential glenohumeral joint instability

Healing time

Healing takes several weeks to months.

Dislocations of the shoulder and clavicle

Acromioclavicular joint dislocation/subluxation

A fall on the shoulder, elbow or outstretched arm can cause varying degrees of separation of the acromioclavicular (AC) joint, causing the lateral end of the clavicle to be displaced upwards (see FIG. 124.7).

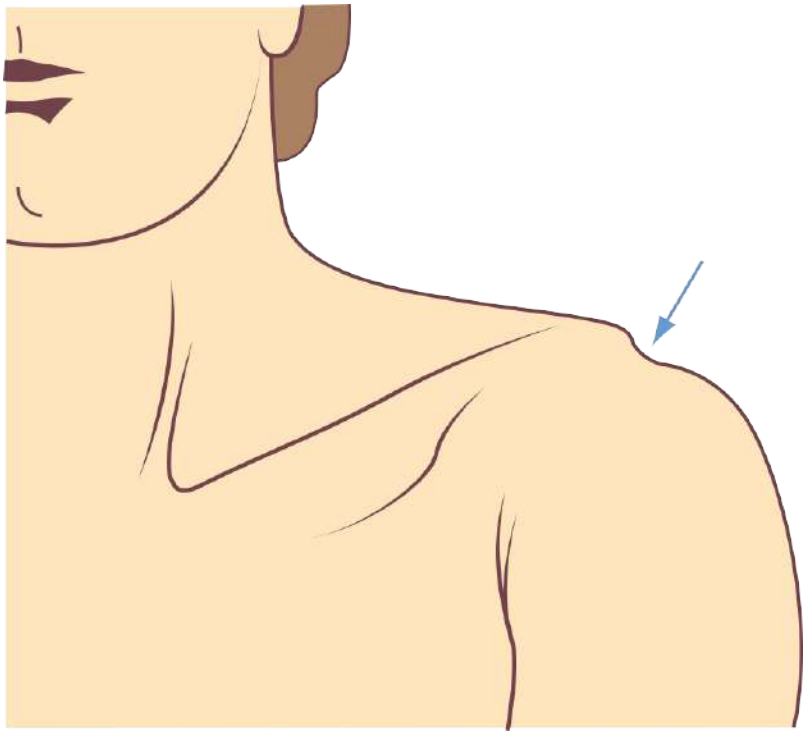


FIGURE 124.7 Subluxation of the acromioclavicular joint: typical appearance

- Grades I, II: partial separation, involving tearing of the AC capsule and ligaments
- Grade III: complete tearing, also affecting the coracoclavicular ligaments

Treatment

- Analgesics
- St John’s high sling (suitable for all injuries)
- Mobilisation exercises as soon as possible
- For Grade III, a compression bandage (or long straps of adhesive low-stretch strapping) with padding at pressure points—elbows, clavicle and coracoid. The clavicle should be manipulated into its correct position and the forearm elevated: applying pressure from above (clavicle) and below (elbow) to achieve compression, apply a bandage over the outer end of the clavicle and round the elbow joint, which is flexed to 90°. The bandage or strapping is worn for 2–3 weeks.[8] Many patients are unable to tolerate this method of treatment. Skin irritation or blisters are common. This occurs particularly with adhesive strapping and the deformity commonly requires correction after the removal of the bandage or strapping. The same effect may be achieved with an orthotic device known as a Kenny-Howard sling or brace.
- The issue of internal fixation versus conservative treatment for a complete dislocation is controversial in that the bulk of patients treated conservatively have minimal residual symptoms. However, a significant minority have residual symptoms in the form of AC joint pain and traction effects on the brachial plexus due to loss of scapular suspension. The patients most likely to have these symptoms are those with high grades of separation, involvement of the dominant shoulder and participation in employment or sports that place heavy physical demands on the shoulder girdle. If there is disruption of the suspensory ligaments of the clavicle, surgical reduction and stabilisation is the preferred option.[8] If in doubt, refer within the first few weeks of injury for consideration of the pros and cons of conservative versus surgical treatment.

Sternoclavicular joint dislocation/subluxation

This uncommon injury is caused by a fall or very heavy impact on the shoulder, causing the medial end of the clavicle to move forwards or anterior (making it prominent) or backwards. Plain X-rays are difficult to interpret and a CT scan is the ideal diagnostic method.

Special problem

A special problem is backward (inward) displacement of the clavicular end with danger to major blood vessels and the trachea. This is one of the few potentially life-threatening orthopaedic injuries. Urgent referral for reduction is essential, especially if stridor or venous obstruction is present. A first aid measure is to place a sandbag between the shoulders with the patient supine and to extend the abducted arm on the affected side.[9] Closed reduction can usually be achieved under anaesthesia. The reduction is nearly always stable.

Treatment

Forward subluxation or dislocation, unlike posterior dislocation, is nearly always unstable and resists attempts at maintaining closed reduction. Despite the persistence of a medial clavicular swelling, most patients need a sling for only 1–2 weeks and the bulk of their pain settles over the following months. Surgery is generally indicated only for an unusually painful and chronic anterior sternoclavicular dislocation.

Dislocation of the shoulder

Dislocations of the shoulder joint can be caused by an impact on the arm by falling directly on the outer aspect of the shoulder, or by a direct violent impact, or by a forceful wrenching of the arm outwards and backwards.

Types of dislocation

- Anterior (forward and downward)—95% of dislocations
- Posterior (backward)—diagnosis often overlooked
- Recurrent anterior dislocation (recurrent posterior dislocation extremely rare)

Anterior dislocation of the shoulder

Management

AP and lateral X-rays should be undertaken to check the position and exclude an associated fracture. The arm should be assessed for the presence of neurological injury before reduction. Reduction can be achieved under general anaesthesia (easier and more comfortable) or with intravenous morphine ± diazepam/midazolam. A variety of methods can be used for anterior dislocation; two are described below. Satisfactory analgesia and patient relaxation are vital to the success of any of the methods.

Kocher method

(See FIG. 124.8 .)

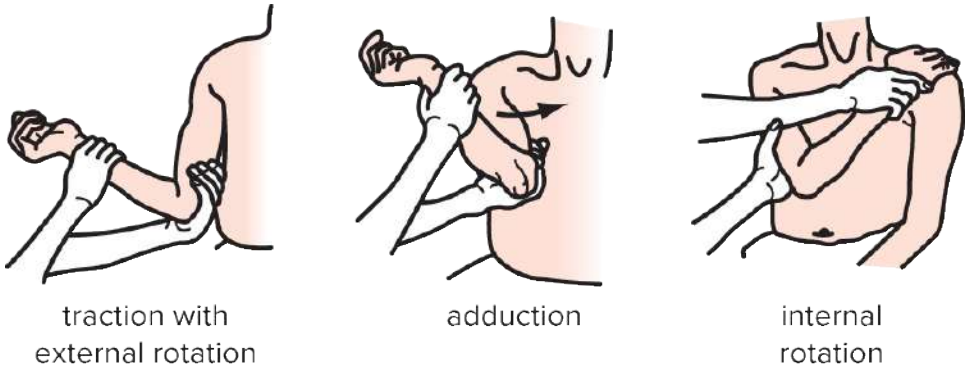


FIGURE 124.8 Kocher method for a dislocated shoulder

- Elbow flexed to 90° and held close to the body
- Slowly rotate arm laterally (externally)
- Adduct humerus across the body by carrying point of elbow while simultaneously applying longitudinal traction along the line of the humerus
- Rotate arm medially (internally)

Hippocratic method

Apply traction to the outstretched arm by a hold on the hand with countertraction from stockinged foot in the medial wall of the axilla. This levers the head of the humerus back. It is a good method if there is an associated avulsion fracture of the greater tuberosity.

Postreduction

- Reduction is complete if the hand can rest comfortably on the opposite shoulder.
- Confirm reduction by X-ray in two planes and again assess for unsuspected fractures (e.g. glenoid rim or greater tuberosity fractures).
- Keep the arm in a sling for 2 weeks.
- Apply a swathe bandage to the chest wall.
- After immobilisation, begin pendulum and circumduction exercises.
- Combined abduction and lateral rotation should be avoided for 3 weeks.

Posterior dislocation of the shoulder

This is the most commonly misdiagnosed major joint dislocation.[7] Posterior dislocation most often follows an epileptic seizure or electrical shock. The postictal patient with a painful shoulder has a posterior dislocation of the shoulder until proven otherwise. Less often this injury is caused by a fall onto the outstretched hand with the arm internally rotated or by a direct blow to the front of the shoulder. If any doubt persists about the diagnosis, a CT scan is appropriate.

The shoulder contour may look normal, but the major clinical sign is painful restriction of external rotation, which is usually completely blocked. Beware of the problem of pain in the shoulder after a convulsion. An ‘axillary shoot through’ X-ray view should be routinely ordered following shoulder trauma.

Reduction of posterior dislocation

Using appropriate analgesia or anaesthesia, apply traction to the shoulder in 90° of abduction (with the elbow at right angles) and laterally (externally) rotate the limb. Referral for reduction is advisable.

Recurrent anterior dislocation

Acute anterior shoulder dislocation may tear or stretch the anterior capsular ligaments from their bony origin. This may predispose to recurrent anterior dislocation or subluxation. Recurrent posterior instability is rare.

A simple procedure for reducing recurrent anterior dislocation is as follows.

- Get the patient to sit comfortably on a chair with legs crossed.
- The patient then interlocks hands and elevates the upper knee so that the hands grip the knee.
- The knee is gradually lowered until its full weight is taken by the hands. At the same time the patient has to concentrate on relaxing the muscles of the shoulder girdle. This method usually effects reduction without the use of force.

Recurrent dislocation often requires definitive surgery, depending on the frequency of dislocations and the degree of apprehension between episodes.

The Bankart lesion

Teenagers and young adults who sustain a traumatic dislocation of the shoulders tend to have the Bankart lesion, which is avulsion of the anteroinferior capsulolabral complex, leading to a high rate of recurrent dislocation. This should be considered for an arthroscopic anterior stabilisation.

Pitfalls

- Nerve injury, especially axillary (circumflex) nerve
- A fractured neck of the humerus, especially in the elderly, may mimic a dislocation
- Associated fractures (greater tuberosity, head of radius, glenoid) may require internal fixation
- Great difficulty with some reductions (this is often related to inadequate analgesia; the use of excessive force may result in fracture)
- Failing to X-ray all suspected dislocations before and after reduction; failing to obtain an axillary view to show posterior displacement or fractures of the humerus or glenoid

Fractures of the humerus

Fractured greater tuberosity of humerus

Treat with a combination of immediate mobilisation and rest in a sling unless grossly displaced, when surgical reduction is advisable. Shoulder stiffness can be a disabling problem, so early movement is encouraged, with review in 7 days. This fracture should be monitored by X-ray within 2 weeks after injury. Undetected displacement may lead to mechanical impingement against the acromion. This fracture may also be an indication of the patient having had a transient glenohumeral dislocation.

Fractured surgical neck of humerus

This usually occurs in the elderly due to a fall onto the outstretched hand. The fragments may be impacted. The greater tuberosity may also be fractured. Watch out for associated dislocation. In adolescents, fracture–separation of the upper humeral epiphysis occurs.

Treatment (no displacement or impaction)

- Triangular sling
- When pain subsides (10–14 days), encourage pendulum exercises in the sling
- Aim for full activity within 8–12 weeks post-injury

Displaced fractures may require internal fixation. Severely comminuted fractures may predispose to post-traumatic osteoarthritis or humeral head avascular necrosis. Consider referral with a view to prosthetic hemiarthroplasty.

Healing

Union usually occurs in 4 weeks and consolidation at 6 weeks.

Pitfalls with fractures of the surgical neck

Minimally displaced fractures of the surgical neck of the humerus are usually managed conservatively, but overzealous early mobilisation can lead to non-union.[9] If there is a communication of this fracture with joint fluid, movement washes away the fracture haematoma and leads to the development of true pseudoarthrosis. Judicious early immobilisation will avert this complication.

The cardinal fracture management rule is: ‘First ensure that stability of the fracture is sufficient to allow healing before prescribing rehabilitation exercises or early use of the extremity’.[7] However, prolonged immobility can cause its own problems. Hand and elbow exercises should commence early, and gentle shoulder ‘pot stirring’ at around 2–3 weeks.

The management of various humeral fractures is summarised in FIGURE 124.9 .

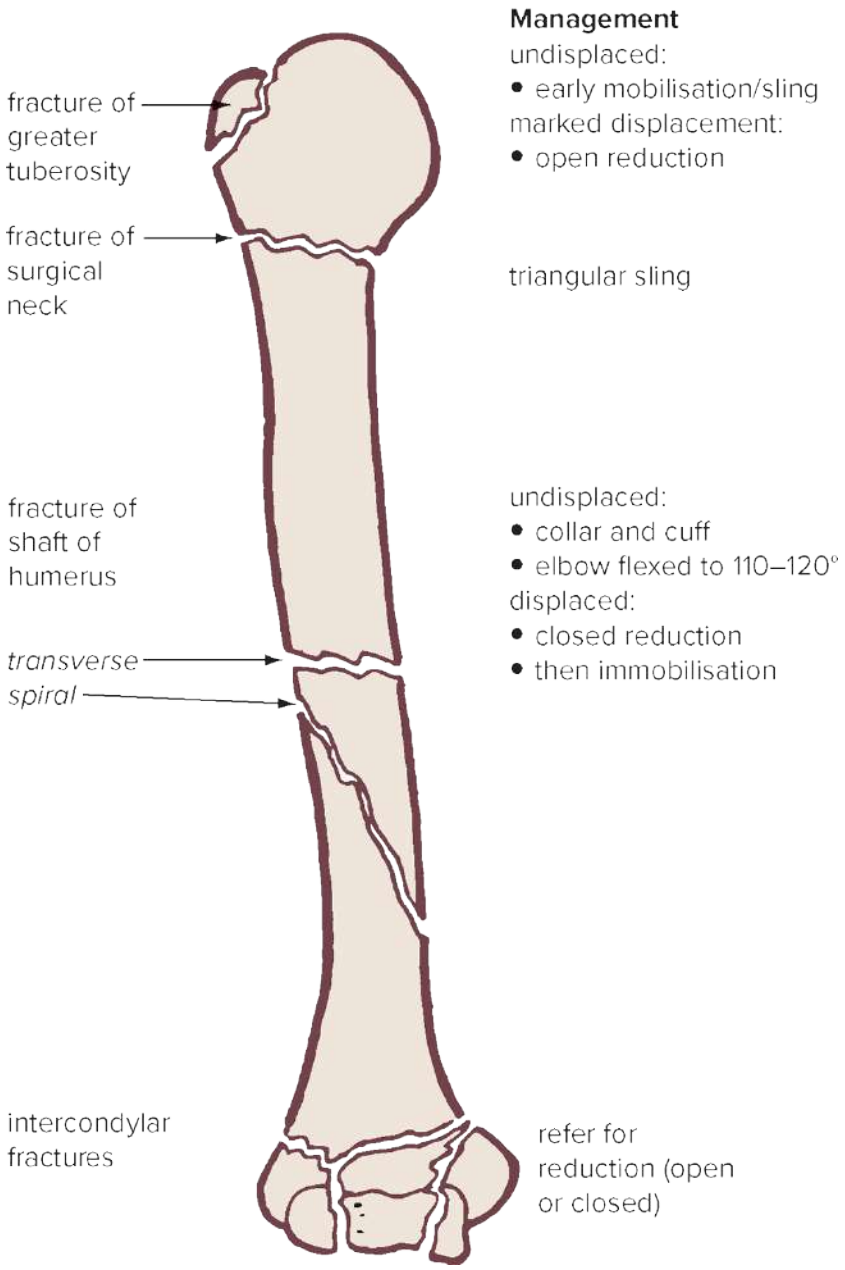


FIGURE 124.9 Various fractures of the humerus in adults

Fracture of shaft of humerus

Humeral shaft fractures may be:

- spiral—due to a fall on the hand
- transverse or slightly oblique—fall on elbow with arm abducted

- comminuted—heavy blow

Caution: watch for radial nerve palsy.

Treatment

- Perfect bony opposition is not necessary; some overriding is acceptable but distraction of the fragments is not.
- Undisplaced fracture: collar and cuff with elbow flexed to 110–120°.
- Significantly displaced humeral shaft fractures may require manipulation under anaesthetic. However, the vast majority of shaft fractures realign to a satisfactory extent under gravitational effects in a sling once muscle spasm and oedema have subsided. A U-shaped hanging cast or slab enhances the gravitational effect and assists splintage.

Intercondylar fractures in adults

Intercondylar fractures, which may be T-shaped or Y-shaped, are usually caused by a fall on the point of the elbow, which drives the olecranon process upwards, splitting the condyles apart. Fractures involving the joint can cause long-term problems of post-traumatic osteoarthritis and joint stiffness. Referral for reduction (closed or open) is appropriate.

Injuries of the elbow and forearm

Fractures and avulsion injuries around the elbow joint in children

Potentially severe deforming injuries include:

- supracondylar fractures
- fracture of the lateral humeral condyle
- fracture of medial humeral epicondyle (see FIG. 124.10)
- fracture of neck of radius

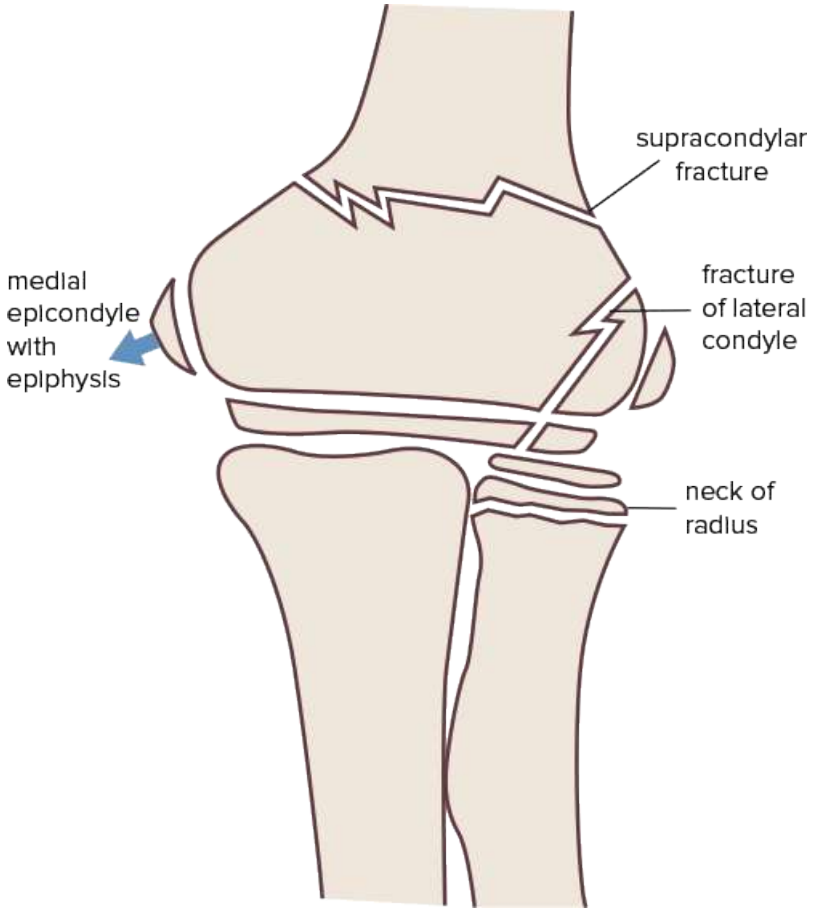


FIGURE 124.10 Fractures and avulsion injuries around the elbow joint in children

Fractures around the elbow in children require referral to consultants experienced in radiology and fracture management.

Supracondylar fractures with forearm ischaemia

Supracondylar fractures represent about half of all elbow fractures in children and most are extension fractures following falls onto the outstretched arm.

Pressure of the displaced bony fragments causes impingement on the brachial artery, which can lead to impending forearm flexor compartment ischaemia and muscle death. Severe forearm pain is the most significant and important sign of ischaemia. Neuropraxia of the median, radial or ulnar nerves is common. These injuries almost invariably recover.

This diagnosis must always be assumed in displaced supracondylar fractures in children. Thus, it is the GP's responsibility to ensure treatment is expedited. The brachial and radial pulses should be assessed carefully.

The fracture is reduced by hyperflexion of the elbow during traction (after lateral displacement has been corrected), and then immobilisation in collar and cuff and stockinet vest. The fully flexed elbow with the usually intact posterior periosteal hinge provides fracture stability. Plaster casting is unnecessary and some would suggest contraindicated because of the significant risk of ischaemic contracture. Circulatory status requires monitoring in the first 24 hours following injury. The collar and cuff should be used for 6 weeks. The invariably stiff elbow quickly resolves without a need for formal therapy.

Fracture of the lateral humeral condyle

Fractures of the lateral humeral condyle also result from a fall onto the outstretched arm in children (see FIG. 124.10). The fracture line passes vertically or obliquely through the lateral condyle and thus crosses the distal humeral growth plate. It occurs in an age group prior to the appearance of the epiphysis of the lateral epicondyle. Pain and swelling over the lateral elbow, but without the gross deformity of a supracondylar fracture of the humerus, could make one suspect this injury. The fracture is commonly overlooked on X-ray. Comparison views of the opposite elbow are particularly helpful in diagnosing this injury.

Recognition of the fracture and early open reduction and internal fixation with wires are vital to reduce the risk of premature plate closure. Such growth plate disturbance may result in a progressive valgus deformity of the elbow and the late development of an ulnar nerve palsy.

The Salter–Harris classification of epiphyseal injuries (FIG. 124.11), which is widely used, has therapeutic and prognostic implications. Most of these injuries heal well but some have the potential for growth arrest or asymmetrical bone growth, leading to deformity and permanent disability, especially type V. Types I and II (the most common) have an excellent prognosis but type III and IV require precise reduction.[10]

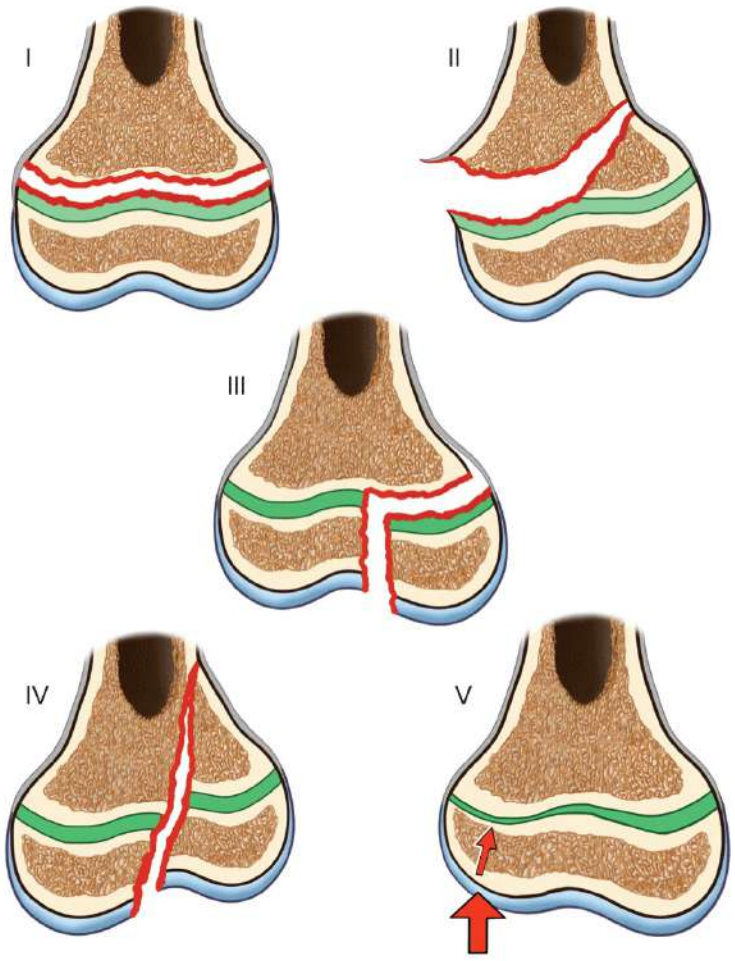


FIGURE 124.11 Salter–Harris classification of growth plate fractures. V represents compression of the growth plate (green in the figure) as shown by the region arrowed.

Fracture of the medial humeral epicondyle

This problem occurs typically in adolescents following a fall onto the outstretched hand. The medial epicondyle may be avulsed by massive flexor pronator muscle contraction together with abduction stresses on the forearm. Avulsion of the epicondyle occurs in the young patient before the epiphysis is united. If displaced, this fracture is best treated by open reduction and internal fixation. Untreated injuries commonly result in non-union, elbow pain and restricted elbow extension.

Fractured neck of radius

This fracture is caused by a child falling on to the outstretched hand. The fracture line is

transverse and is situated immediately distal to the epiphysis.

The degree of tilt is critical. Up to 15° of tilt is acceptable but, beyond that, reduction (preferably closed) will be necessary. The head of the radius must never be excised in children.

Dislocated elbow

A dislocated elbow (note: distinct from a *pulled elbow*) is caused by a fall onto the outstretched hand, forcing the forearm backwards to result in posterior and lateral displacement (see FIGS 124.12 and 124.13). The peripheral pulses and sensation in the hand must be assessed carefully. It may result in vascular injury to the brachial artery or injury to the median and ulnar nerves. Check the function of the ulnar nerve before and after reduction.

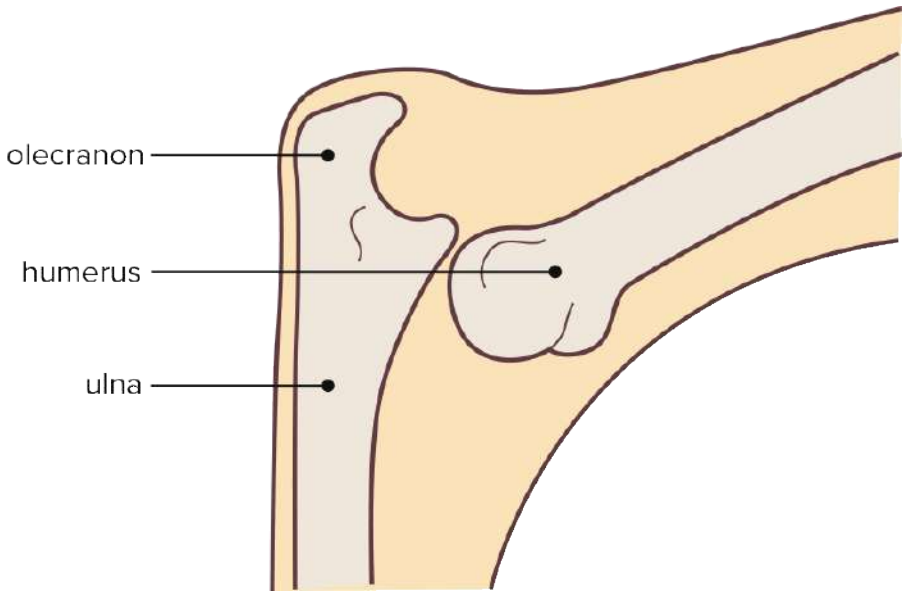


FIGURE 124.12 Dislocated elbow: uncomplicated posterior dislocation

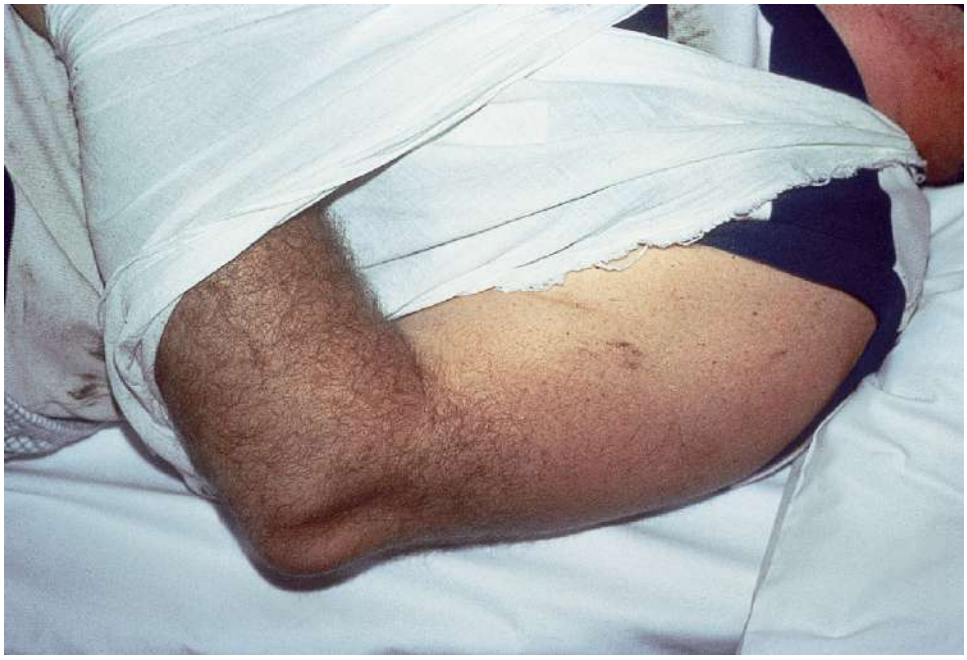


FIGURE 124.13 Dislocated elbow showing posterior displacement of the ulna and radius, with the typical deformity

Treatment

Attempt reduction with patient fully relaxed under anaesthesia. It is important to apply traction to the flexed elbow while allowing it to extend (to approximately 20–30° of flexion) to enable correction of the lateral displacement and then the posterior displacement.

Follow-up

Encourage early mobilisation with gentle exercises in between resting the elbow for 2–3 weeks in a collar and cuff with the elbow flexed above 90°, avoiding passive movements. A plaster cast should not be used because of the risk of ischaemic necrosis of muscle. This will minimise the possibility of myositis ossificans. Recurrent dislocation of the elbow is uncommon.

A simple method of reduction

This method reduces an uncomplicated posterior dislocation of the elbow without the need for anaesthesia or an assistant. The manipulation must be gentle and without sudden movement.

Method

1. The patient lies prone on a stretcher or couch, with the forearm dangling towards the floor.
2. Grasp the wrist and slowly apply traction in the direction of the long axis of the forearm (see FIG. 124.14).

3. When the muscles feel relaxed (this might take several minutes), use the thumb and index finger of the other hand to grasp the olecranon and guide it to a reduced position, correcting any lateral shift.

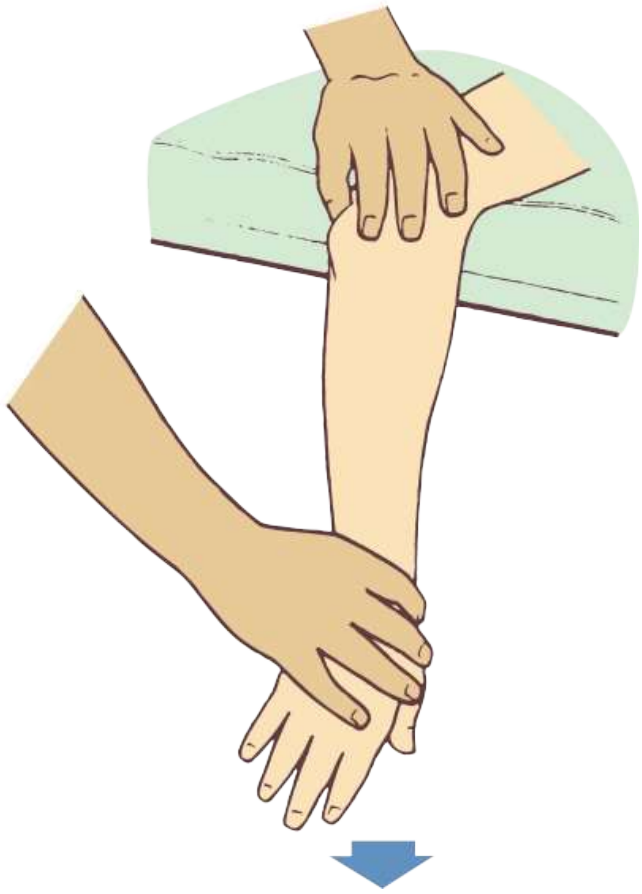


FIGURE 124.14 Dislocated elbow: method of reduction by traction on the dependent arm

Pitfalls

- Incomplete reduction: ulna articulates with capitellum and not the trochlea
- Injury to ulnar nerve (spontaneous recovery usually occurs after 6–8 weeks)
- Associated fractures (e.g. coronoid process), which may cause instability

‘Pulled’ elbow

See CHAPTER 53 .

Fractured head of radius (adults)

If the fracture is very slight and undisplaced, treat conservatively with the elbow at right angles in a collar and cuff until the pain subsides sufficiently to allow flexion/extension and pronation/supination exercises.

Elbow stiffness is a major problem even after apparently trivial radial head fractures. Early mobilisation is vital. Excision of the radial head should be considered for highly comminuted fractures that limit the ability to mobilise the elbow early or predispose to post-traumatic osteoarthritis. Associated distal radio-ulnar joint or wrist injuries are often overlooked.

Fractured olecranon

- Comminuted fracture (with little displacement): sling for 3 weeks and active movements
- Transverse (gap) fracture: open reduction with screw or wire

Monteggia fracture–dislocation of the radial head

Fractures of the proximal third of the ulna with dislocation of the radial head (Monteggia fracture–dislocation) (see FIG. 124.15) have a history of mismanagement during treatment. The radial head dislocation is easily overlooked.

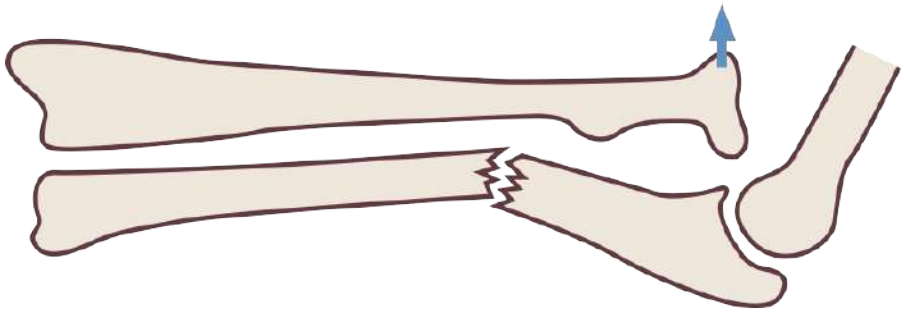


FIGURE 124.15 Monteggia fracture: dislocation of the radial head; it is important not to miss a dislocated head of radius with a fracture of the proximal third of the ulna

Redislocation or subluxation of the radial head is common.

Since surgical intervention is advisable, referral of displaced forearm fractures for early surgery is recommended. Surgical plating of the ulnar shaft maintains the radial head in a reduced position. Follow-up X-rays are mandatory to ensure that there has not been a late redislocation of the radial head.

Fracture–dislocation in the lower forearm (Galeazzi

injury)

This injury is usually caused by a fall on to the hand and is a combination of a fractured radius (at the junction of its middle and distal thirds) and subluxation of the distal radio-ulnar joint. The patient should be referred, as open reduction is often required.

Fractures of the radius and ulna shafts

General features

In adults, it is more common to break both forearm bones. Displaced fractures of both bones require perfect reduction, which can generally only be achieved by surgical reduction and plating. Less-than-satisfactory reduction interferes with normal pronation and supination. A fracture of one bone alone is uncommon and usually caused by a direct blow. For a fracture of one bone alone, look for evidence of an associated dislocation of the other forearm bone. In children, greenstick fractures are common. Fractured radial shafts tend to slip and ulnar fractures heal slowly. Dislocation of the head of the radius or inferior radio-ulnar joint can be missed if X-rays do not include the elbows and wrist joints. The rule is to X-ray the joints above and below any injury.

Reduction

- A greenstick fracture is readily straightened by firm pressure.
- A complete fracture (spiral or transverse) is reduced by traction and rotation.
- A slight overlap and angulation is permissible in children, but perfect reduction is essential in adults.
- A plaster cast should include both the elbow and the wrist joints.

Healing time: (adults) spiral fracture—6 weeks; transverse fracture—12 weeks.

Injuries of the wrist

Colles fracture of lower end of radius

Colles fractures rival clavicular in being the most common of all fractures. A Colles is a supination fracture of the distal 3 cm of the radius, caused by a fall onto the outstretched hand.

Clinical features

- Usually an elderly woman

- Osteoporosis is common
- Fall on dorsiflexed hand
- Fracture features:

  impaction

  posterior displacement and angulation

  lateral displacement and angulation

  supination

  dinner fork deformity (see FIG. 124.16)

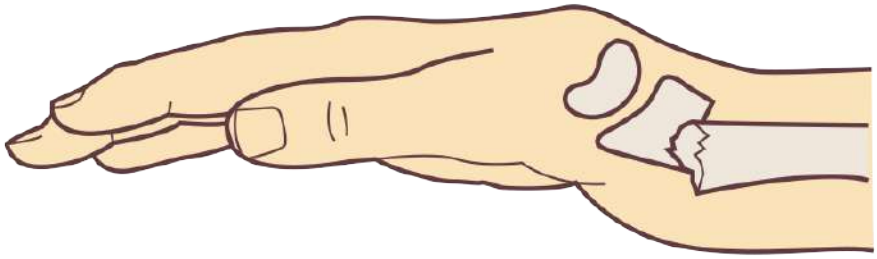


FIGURE 124.16 Dinner fork deformity of Colles fracture: a fracture of the distal head of the radius showing impaction and posterior displacement and angulation

Treatment

- If minimal displacement—below-elbow plaster for 4 weeks, then a crepe bandage
- If displaced: meticulous reduction under anaesthesia:

  set in flexion 10°, ulnar deviation 10° and pronation (see FIG. 124.17)

  below-elbow plaster 4–6 weeks (6 weeks maximum time)

  unstable fractures may require an above-elbow cast initially with the forearm in pronation

  check X-ray at 10–14 days; position may be lost as swelling subsides and plaster becomes loose