Hip dislocations and acetabular fractures

Acetabular fracture

(See Fig. 9.51.) Often accompanies traumatic hip dislocation following violent injury. Posterior rim fractures are the most common. Complications include massive haemorrhage, sciatic nerve damage, myositis ossificans, and secondary OA. Resuscitate, give analgesia, and deal with priorities first. CT demonstrates the exact injury better than X-ray. Refer to orthopaedics.

Central dislocation of the hip

A serious acetabular fracture, which involves the head of the femur being driven through the acetabular floor following a fall or force directed along the length of the femur (eg car dashboard).

Traumatic posterior dislocation of the hip

(See Fig. 9.52.) Implies major trauma, often with other critical injuries (eg dashboard knee injury in a car crash) or fractured posterior acetabulum. The limb is shortened and internally rotated, with the hip flexed and adducted. This appearance may be absent if there is also a femoral shaft fracture. Check for sciatic nerve damage—examine foot dorsiflexion and below-knee sensation. Complications: sciatic nerve injury, avascular necrosis of the femoral head (risk ↑ the longer the hip is dislocated), and secondary OA.

Resuscitate, give analgesia, and address ABC priorities. Refer for reduction under GA. In unconscious or anaesthetized patients, reduce in the ED.

Reduction technique for posterior dislocation ('Allis technique')

- It is easiest and safest to reduce dislocation if the anaesthetized patient is placed on the floor. If this is not possible, stand on the trolley. An assistant presses down on the patient's anterior superior iliac spines to hold down the pelvis.
- Flex the hip and knee, both to 90°, and correct adduction and internal rotation deformities.
- Grip the patient's lower leg between your knees, and grasp the patient's knee with both hands.
- Lean back and lever the knee up, pulling the patient's hip upward. A 'clunk' confirms successful reduction. X-ray to confirm reduction.

Anterior dislocation of the hip

Less common. The leg is held abducted and externally rotated. Complications include damage to the femoral nerve, artery, and vein. Give analgesia and refer for reduction under GA.

Dislocated hip prosthesis

Relatively common (affects ~3% of total hip replacements). It can follow minor (or even no) trauma—sometimes, crossing legs or flexing the hip to 90° can be enough. The patient presents in severe pain, unable to move the hip. Confirm posterior dislocation of the hip prosthesis by X-ray (see Fig. 9.53). Give IV opioid, and refer to orthopaedics for MUA (and assessment of prosthesis stability) under GA. Depending on protocols, expertise, and resources (especially for recovery), it may be possible to do this in the ED.

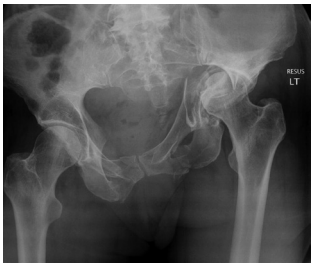

Fig. 9.51 Major pelvic trauma involving fractures through all four pubic rami, the acetabulum, and the left sacroiliac joint.

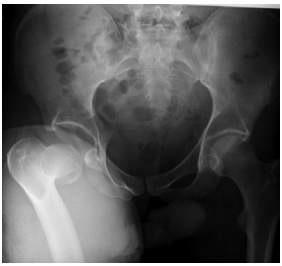

Fig. 9.52 Posterior hip dislocation.

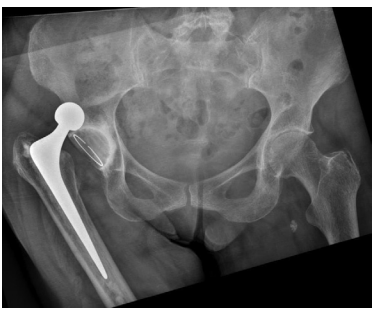

Fig. 9.53 Dislocated hip prosthesis.

Sacral and coccygeal fractures

Fractures of the sacrum

Usually follows violent direct trauma such as falls. Damage to sacral nerve roots may occur. Check carefully for saddle anaesthesia, ↓ anal tone, lower limb weakness, or bladder dysfunction. Refer to the orthopaedic team.

Fracture of the coccyx

Follows a fall onto the bottom. Do not X-ray routinely—the diagnosis is clinical. Perform a PR examination, and check for local coccygeal tenderness, palpable fractures, or rectal damage. Complications are unusual, but refer patients with rectal tears to the general surgeon and refer to the orthopaedic team if the coccyx is grossly displaced, as it may require manipulation under LA or even excision. Treat the rest (the majority) symptomatically (eg suggest a ring cushion and provide analgesia).

Hip fractures

Intracapsular fractures of the neck of femur

Can follow relatively minor trauma. Risk ↑ in the elderly, because of osteoporosis, osteomalacia, and ↑ rate of falls. These fractures can disrupt the blood supply to the femoral head, causing avascular necrosis.

Fractures around the hip in younger patients imply high-energy injury—the incidence of non-union or avascular necrosis may be as high as 20%.

Diagnosis

Usually follows a fall onto the hip or bottom. Pain may radiate down towards the knee. The affected leg may be shortened and externally rotated. Check for hypothermia and dehydration (the patient may have been lying for hours). Look for tenderness over the hip, particularly on rotation. Suspect hip fracture in an elderly person who:

- Exhibits a sudden inability to weight-bear. There may be no history of injury, particularly in the presence of confusion or dementia.
- Is unable to weight-bear and has pain in the knee (the hip may not be painful).
- Has 'gone off her feet'.

X-rays

Look closely for disrupted trabeculae/cortices and abnormal pelvic contours (Shenton's lines). Fractures of the femoral neck are not always visible on initial X-rays. Repeat X-rays, CT, or MRI may be required if symptoms continue. Intracapsular femoral neck fractures are graded according to the Garden classification (see Table 9.4 and Fig. 9.54).

Table 9.4 Garden classification of intracapsular hip fractures

Garden I	Trabeculae angulated, but inferior cortex intact. No significant displacement
Garden II	Trabeculae in line, but a fracture line visible from superior to inferior cortex. No significant displacement
Garden III	Obvious complete fracture line, with slight displacement and/or rotation of the femoral head
Garden IV	Gross, often complete, displacement of the femoral head

Treatment

- Obtain IV access and send blood for U&E, glucose, FBC, and cross-match.
- Start IVI if indicated (eg dehydration or shock).
- Give IV analgesia plus an antiemetic. Provide all analgesia IV in small increments every few minutes until pain is controlled.
- Perform a fascia iliaca compartment block (see ➔ Fascia iliaca compartment block, p. 312).
- Obtain an ECG to look for arrhythmias/MI, and consider the need for CXR.
- Arrange other investigations as indicated by the history/examination.
- Admit to the orthopaedic ward.

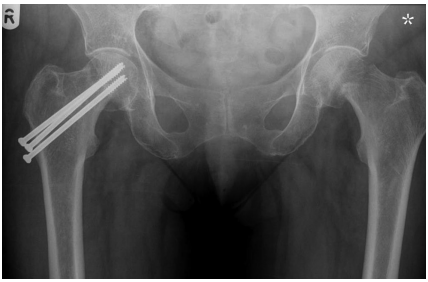

Fig. 9.54 Garden IV left subcapital hip fracture with previous fixation to the right side.

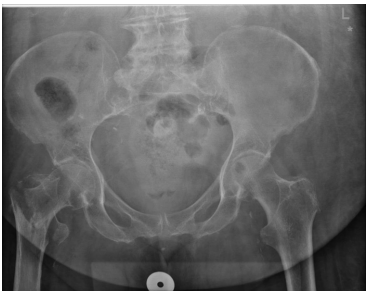

Fig. 9.55 Comminuted intertrochanteric right hip fracture.

Intertrochanteric fracture

These affect the base of the femoral neck and the intertrochanteric region (see Fig. 9.55). Initial management is identical to that for neck of femur fractures outlined previously.

Isolated trochanteric avulsion fracture

Sudden force may avulse insertions of the gluteus medius (greater trochanter) or iliopsoas (lesser trochanter). Give analgesia; assess mobility with crutches, and refer for follow-up for gradual mobilization and symptomatic treatment.

Hip pain after injury, but no fracture seen

Elderly patients who report hip pain and struggle to walk after a fall, and yet have no fracture of the hip or pubic rami on X-ray, may need assessment by an occupational therapist or physiotherapist before deciding if they can be safely discharged home with analgesia and appropriate walking aid. A small, but significant, proportion of such patients will turn out to have a hip fracture. In patients with significant pain and difficulty weight-bearing, consider requesting a CT (or MRI) scan at the time of initial presentation to show up a hip fracture not identified on plain X-rays.

For patients who have normal X-rays, are able to mobilize satisfactorily, and are being discharged, advise them to return for review and further imaging (CT or MRI) if the pain continues for >1 week—ideally, provide an advice leaflet to explain and reinforce this.

Shaft of femur fractures

Enormous force is required to break an undiseased adult femoral shaft. Fractures are frequently associated with multisystem trauma. Treatment of immediately life-threatening injuries takes priority. Transverse, spiral, or segmental shaft fractures usually result from falls, crushing injuries, or high-speed road traffic collisions. There is often associated dislocation of the hip or other serious injury to the lower limb ± major trauma affecting the head, chest, abdomen, and pelvis.

Complications

Closed fractures of the femoral shaft, even without obvious vascular injury, may be associated with marked blood loss. Up to 1.5L of blood may be lost without visible thigh swelling. Rarely, gross blood loss may occur from compound femoral fractures. Later complications include fat embolism/ARDS. The incidence of complications is ↓ by early splintage and early definitive treatment (usually closed intramedullary nailing).

Diagnosis

The diagnosis is usually clear on examination, with deformity, shortening, external rotation, and abduction at the hip on the affected side. The fracture may be felt or even heard on movement of the lower limb. Carefully check for associated pelvic, knee, or distal limb injuries or for the presence of associated wounds. Document sensation and pulses in the limb, and recheck frequently.

Treatment

Before X-rays, resuscitate, exclude life-threatening injuries, replace IV fluids, give adequate analgesia, and splint fractures as follows:

- Assess ABC, establish priorities, and resuscitate as for patients presenting with major trauma (see ➲ Major trauma: treatment principles, p. 330).
- Commence fluid replacement via two large-bore IV cannulae—start with 1000mL of 0.9% saline.
- Obtain blood for cross-matching, plus U&E, FBC, and coagulation screen.
- Administer IV analgesia—give small increments of opioid (with an antiemetic) until pain is controlled.
- Give IV tranexamic acid (see ➲ Major trauma: treatment principles, p. 330).
- Strongly consider femoral nerve block (see ➲ Femoral nerve block, p. 313) or fascia iliaca block (see ➲ Fascia iliaca compartment block, p. 312). As this starts to take effect (~5–10min), prepare splintage and immobilize in a traction splint (eg Kendrick).
- Arrange imaging of the femur—very often a trauma pan-CT scan of the head, neck, chest, abdomen, and pelvis can be appropriately extended down to involve the femoral shafts.

Note: there was a time when the August and February junior doctor job changeover in the UK took a terrible toll on sleep-deprived doctors who fell asleep whilst travelling long distances between hospitals. The doctor who sustained the fractures shown in Fig. 9.56 as part of multiple injuries was successfully resuscitated in the middle of the night by a senior ED doctor—both later joined forces to write the first edition of the *Oxford Handbook of Emergency Medicine*.

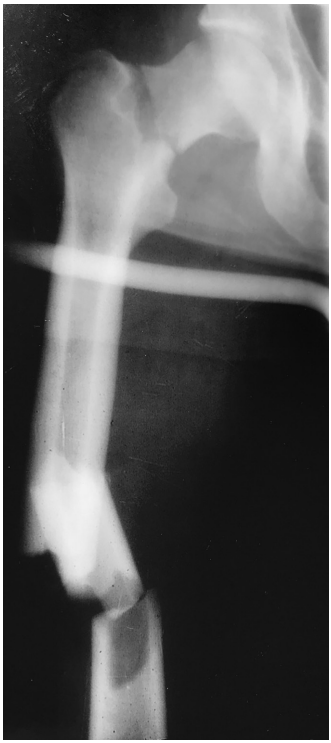

Fig. 9.56 Comminuted right femoral shaft fracture with a 'butterfly fragment' plus a Garden II intracapsular hip fracture in a 26y old doctor involved in a high-speed road traffic collision with a lorry.

Subtrochanteric fractures

These involve the most proximal part of the femoral shaft, at or just distal to the trochanters. They typically involve high-energy trauma in younger patients and are often associated with other serious injuries. They can also occur as isolated injuries following relatively minor trauma in those with osteoporosis or metastatic disease. Treat as for femoral shaft fractures.

Supracondylar femoral fractures

Fractures of the distal third of the femur usually follow violent direct force. They are frequently comminuted and often intra-articular with associated damage to the knee joint. In adults, the distal femoral fragment tends to rotate due to pulling from the gastrocnemius. Treat as for femoral shaft fractures, but note that femoral nerve block may not be as effective.

Approach to knee injuries

History

Many knee injuries result from sports, particularly football and rugby. Carefully elicit the exact mechanism of injury, as it provides clues to the diagnosis. Valgus or varus stresses can damage the medial and lateral collateral ligaments, respectively. Flexed, twisting knee injuries are frequently associated with meniscal injuries. The anterior cruciate ligament (isolated or associated with medial collateral and/or medial meniscal injuries) may tear during forced flexion or hyperextension. Posterior cruciate injuries may follow falls or dashboard impact where the tibia is forced backwards violently (often associated with medial or lateral ligament injuries).

Rapid-onset tense swelling in a knee is usually an *acute haemarthrosis*. Swelling developing more gradually over several days is more likely to represent a reactive effusion. Ask about previous knee problems: swelling, clicking, locking, or giving way (the last two suggest underlying meniscal pathology). Document any previous knee surgery or the presence of other joint problems. In a hot, swollen, painful, and stiff knee without a history of significant trauma, consider and exclude septic arthritis.

Examination

Examine both legs, with the patient lying supine. If there is much discomfort, consider giving oral analgesia and re-examine in 10–15min. Reassure him/her that you will not suddenly pull or move the leg without warning.

- *Observe* how the patient mobilizes (or not) to enter the examination cubicle.
- *Look for* bruising, swelling, redness, abrasions, or other wounds.
- *Feel* for warmth, crepitation, or the presence of a knee effusion.
- *Check straight leg raise*: the ability to do this against resistance virtually excludes quadriceps and patellar tendon rupture or transverse patellar fractures. If unable (possibly due to pain), ask the patient to kick forwards whilst sitting, with the affected leg dangling free.
- *Assess tone and bulk* of the quadriceps muscle, and compare sides.
- *Assess knee movement*: gentle encouragement or supporting the limb may be required, but do not use any force.
- *Assess the cruciate ligaments*: this may not be easy or possible soon after some acute knee injuries. If the patient is struggling to achieve 90° flexion, assess with slight flexion (~10°) (Lachman test).
- *Assess the collateral ligaments*: with the leg straight, gently apply a valgus stress to the knee joint (ie move the lower leg laterally), examining for laxity or pain in the medial collateral ligament. Next apply a varus stress (ie move the lower leg medially), examining for laxity or pain in the lateral collateral ligament complex. Repeat the procedure with the knee in ~20° flexion, as this will relax the cruciate ligaments. Compare both sides.

X-rays for knee injuries

X-rays are the mainstay of initial imaging—CT and MRI may be indicated after specialist consultation. Obtain X-rays if there is suspected fracture or other significant injury. Use the Ottawa knee rules to assist the decision (in those aged between 18 and 55y) as to whether or not to X-ray.

X-rays are only required if any of the following are present:

- There is isolated bony tenderness of the patella.
- There is bony tenderness over the fibula head.
- The patient cannot flex the knee to 90°.
- The patient could not weight-bear (take at least four steps), both immediately after the injury and at the time of examination.

Adopt a lower threshold for obtaining X-rays in those aged <18 or >55y, patients intoxicated with alcohol, those suffering from bone disease (eg RA, documented osteoporosis), and those who re-attend the ED with the same injury (having not been X-rayed initially).

Patella fracture

This may follow a direct blow or fall onto the patella or sudden violent knee flexion or contraction of the quadriceps muscle. Look for pain, swelling, crepitus, and difficulty extending the knee. Displaced transverse fractures result in an inability to straight leg raise (this is also a feature of rupture of the quadriceps tendon or patellar tendon—see ➲ Soft tissue knee injuries, p. 492). There may be associated haemarthrosis.

X-rays may be difficult to interpret, as the patella overlies the distal femur on the AP view and can obscure subtle fractures (see Fig. 9.57). Take care not to mistake a bipartite patella for a fracture (the accessory bone is typically in the upper, lateral part of the patella).

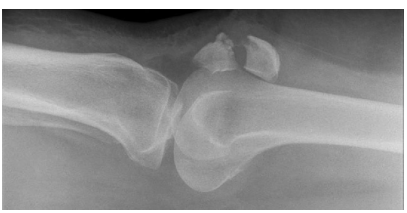

Fig. 9.57 Lateral X-ray showing a transverse fracture of the patella with separation of the fragments.

Treatment

- Treat vertical fractures with analgesia; immobilize in a non-weight-bearing above-knee POP backslab; supply crutches, and arrange orthopaedic follow-up.
- Transverse fractures tend to displace due to the pull of the quadriceps. Treat with analgesia and immobilization in a POP backslab, and refer to the orthopaedic team for probable ORIF (occasionally, the orthopaedic team may treat an undisplaced transverse fracture conservatively).

Dislocations of the patella and knee

Dislocation of the patella

The patella typically dislocates laterally. This often follows medial stress to the knee—the dislocation may reduce spontaneously. There may be a history of recurrent dislocation. The patient has a painful knee, held in flexion, with obvious lateral displacement of the patella. X-rays are not generally required prior to reduction of the dislocation. Reduction can usually be achieved using Entonox®—IV analgesia is seldom required. Stand on the lateral side of the affected limb and hold the affected knee gently. Using a thumb, lever the patella medially in one smooth, firm movement, whilst gently extending the knee at the same time. Successful reduction is obvious and should rapidly relieve symptoms. Once reduced, obtain X-rays, immobilize in a canvas ('cricket pad') back-splint or backslab cast POP, provide analgesia, and arrange orthopaedic follow-up. An MRI scan at follow-up may help to identify the extent of damage to the medial patellofemoral ligament—a knee specialist will decide about possible surgical repair.

Spontaneous reduction/patella subluxation The patient who has experienced spontaneous reduction and/or subluxation prior to arrival at the hospital will typically have maximal tenderness over the medial aspect of the upper patella, reflecting damage to the attachment of the vastus medialis. There may be 'apprehension' when gentle lateral pressure is applied to the patella. If clinical features are dramatic, rest in a splint (occasionally, cylinder POP may be needed); otherwise refer for physiotherapy and orthopaedic follow-up.

Dislocation of the knee

Although rare, this injury indicates severe disruption of the ligamentous structures and soft tissues of the knee. Look carefully for associated injuries (eg femur or lower limb), and document distal pulses and sensation—the popliteal artery or nerve are often injured. Reduction requires adequate (IV opioid) analgesia and usually GA or sedation with full precautions. Reduce by simple traction on the limb, correcting the deformity. Check distal pulses and sensation after reduction; immobilize in a long leg POP backslab, and arrange orthopaedic admission. Check the circulation repeatedly, since popliteal artery damage may not become apparent for some hours—angiography is usually required. Compartment syndrome is another recognized complication.

Tibial plateau fracture

Falls onto an extended leg can cause compression fractures of the proximal tibia. Valgus stresses crush or fracture the lateral tibial plateau. These injuries are commonly seen in pedestrians injured following impact with car bumpers. Varus injuries result in crushing or fracture of the medial tibial plateau and are usually associated with rupture of the opposite collateral ligaments. Examine for tenderness over the medial or lateral margins of the proximal tibia. Look for swelling, haemarthrosis, or ligamentous instability (also try to assess the cruciate ligaments—see ➲ Approach to knee injuries, pp. 488–9). Look carefully on X-rays for breaks in the articular surfaces of the proximal tibia, avulsions from the ligamentous attachments, or loss of height from the medial and lateral tibial plateaux, but beware this may be subtle (see Fig. 9.58). Adopt a low threshold to request a CT scan to clarify the nature and extent of the injury.

Treat with immobilization in a long leg POP backslab, following adequate analgesia, and refer to orthopaedic staff. Fractures of the tibial plateau often require elevation ± ORIF with bone grafting. Admit all patients with acute haemarthrosis. Treat small, isolated avulsions without haemarthrosis with immobilization, crutches, and analgesia, and arrange orthopaedic follow-up.

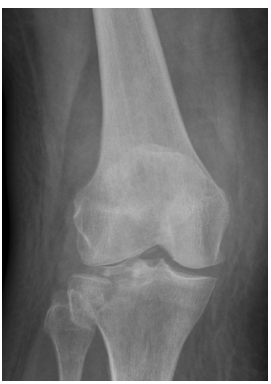

Fig. 9.58 AP knee X-ray showing a displaced lateral tibial plateau fracture, with a large associated joint effusion.

Postero-lateral corner injuries

The postero-lateral corner of the knee comprises a group of ligaments and muscles/tendons that add to the stability of the joint. Postero-lateral corner injuries often occur in association with other significant knee trauma (eg dislocations, rupture of anterior or posterior cruciate ligaments), but isolated injuries can occur. Suspect this injury when significant symptoms follow the application of varus force to the anteromedial aspect of the extended knee. Chronic instability can result. X-rays may be normal or show subtle avulsions or widening of the lateral joint space. Urgent MRI and orthopaedic referral will enable prompt treatment.

Soft tissue knee injuries

Acute haemarthrosis

Rapid-onset swelling following a knee injury, often warm, tense, and painful. Common causes include cruciate ligament rupture, tibial avulsion, and tibial plateau or other fractures. An acute haemarthrosis indicates serious injury. Refer for orthopaedic appraisal following splintage, analgesia, and appropriate X-rays. Aspiration of a haemarthrosis (advocated by some experts to provide analgesia) requires a strict aseptic technique.

Cruciate ligament rupture

Pain and swelling can make it hard to elicit classical physical signs. An audible 'pop' at the time of injury is highly suggestive of anterior cruciate ligament rupture.

Anterior cruciate ligament tears often occur in association with tears of the medial collateral ligament and/or medial meniscus. Examine for the presence of haemarthrosis, abnormal ↑ anterior glide of the tibia ('+ve anterior drawer test'), and injuries to the medial collateral ligament or other structures. Look carefully at X-rays for avulsion of the anterior tibial spine (anterior cruciate insertion). Give analgesia, and refer to the orthopaedic surgeon.

In *posterior cruciate ligament tears*, the tibia may appear to sag back when the knee is flexed, so the tibia can be pulled into a more normal position, causing a 'false +ve' anterior drawer test. X-rays may reveal the relevant posterior tibial spine to be avulsed. Provide analgesia and refer.

Collateral ligament injuries

Tenderness over the medial or lateral collateral ligament, with pain at this site on stress testing, indicates collateral ligament injury. Most injuries are isolated and have no associated haemarthrosis and no abnormality on X-ray. The degree of laxity on stress testing will help to guide treatment:

- Local tenderness with no laxity (or very slight laxity) implies a grade I injury. Treat with analgesia and physiotherapy (± crutches), with an expectation of full recovery in 2–4 weeks.
- Local tenderness with minor/moderate laxity, but with a definite end-point, implies a grade II injury. Provide analgesia, crutches, and instruction on quadriceps exercises, and refer for orthopaedic follow-up.
- Major laxity (ie the joint opening up >1cm) with no end-point implies complete rupture. Consider a POP cylinder (or splint), and provide crutches, analgesia, quadriceps exercises, and orthopaedic follow-up.

Ruptured quadriceps

Complete rupture of the distal quadriceps insertion can result from a direct injury or from sudden, violent contraction of the quadriceps muscle. Examination reveals complete inability to straight leg raise—never assume this is just due to pain. There may be a palpable defect in the muscle insertion. Refer to the orthopaedic surgeon for repair.

Ruptured patellar tendon

Examine for complete inability to straight leg raise and a high-riding patella, a palpable defect in the patellar tendon. There is frequently an associated avulsion of the tibial tuberosity. Refer to orthopaedics for repair.

Other knee problems

Acutely locked knee

A springy block to full extension (which varies from just a few degrees to much more) in the knee indicates an underlying meniscal injury or other loose body in the knee joint. Obtain knee X-rays (including a tunnel view), which may show a loose body. Do not attempt to unlock the knee by manipulation, as this is usually painful and futile. Give analgesia, and refer for arthroscopy.

Prepatellar and infrapatellar bursitis

This results from inflammation of the fluid-filled bursa in front of or just below the patella, respectively, typically from unaccustomed kneeling. Treat with rest (which may involve the use of crutches), a short course of NSAID, and avoidance of the causative activity. Persistent symptoms may necessitate elective excision of the bursa. Infective bursitis may occur (↑ T° and cellulitis are clues to this)—aspirate fluid for culture and sensitivity, and start antibiotics (eg flucloxacillin).

Other causes of knee pain

Patients present not infrequently with knee pain of variable duration and with no history of trauma.

In adults, causes include Baker's cyst, OA (especially in the elderly), and acute arthritic conditions, including septic arthritis (rare, but important). Also rare, but worthy of consideration, is osteosarcoma, which typically affects teenagers or young adults, producing pain and swelling.

In children, causes include sepsis (including both septic arthritis and osteomyelitis—see ➔ The limping child, pp. 726–7), Osgood–Schlatter's disease, osteochondritis dissecans, Johansson–Larsen's disease (see all in ➔ Osteochondritis, pp. 730–1), chondromalacia patellae, referred pain from the hip, and malignancy (eg leukaemic deposits).

Tibial and fibular shaft fractures

Adult tibial fractures are usually a result of direct blows or falls onto the tibial shaft. Spiral fractures of the tibia or fibula follow violent twisting injuries, usually from sports (eg soccer, rugby, skiing). Displaced fractures typically involve both the tibia and the fibula. A large portion of the tibia has relatively little soft tissue covering—compound injuries are common. Displaced tibial shaft fractures may be complicated by injury to the popliteal artery and compartment syndromes (see ➲ Crush syndrome, pp. 406–7). Fractures of the proximal fibula may be associated with injury to the common peroneal nerve. Check (repeatedly) for distal pulses and sensation.

Diagnosis Is usually easy. Look for deformity, localized swelling, or tenderness. Regard all wounds near the fracture site as potential compound injuries.

X-rays Ensure X-rays show the whole length of the tibia and fibula. Examine closely for the presence of other injuries (eg around the knee or ankle).

Stress fractures can occur and may not be visible on initial X-rays. Refer if there are persisting symptoms suggestive of stress fracture.

Tibial shaft fractures

Treat undisplaced transverse tibial shaft fractures with analgesia and long leg POP backslab. Spiral and oblique fractures also need immobilization but are potentially unstable, so refer to the orthopaedic team for admission. Immobilize displaced fractures in a long leg POP backslab, following IV analgesia, and refer (to consider MUA or closed intramedullary nailing). Badly comminuted or segmental fractures may require ORIF. Contact orthopaedics immediately if suspected vascular injury, sensory deficit, or gross swelling.

Treat compound fractures initially as described in ➲ Open (compound) fractures, p. 349, and refer to the orthopaedic surgeon for urgent wound toilet, debridement, and fixation (see http://www.boa.ac.uk).

Fibular shaft fractures

These can occur in combination with a tibial fracture, as a result of a direct blow or from twisting injuries. The common peroneal nerve may be damaged in proximal fibular injuries. Examine specifically for weakness of ankle dorsiflexion and ↓ sensation of the lateral aspect of the forefoot.

Treat undisplaced proximal or fibular shaft fractures with analgesia and elevation. Consider support with a light bandage. If unable to weight-bear, use a below-knee POP for comfort, with crutches until weight-bearing is possible. Arrange follow-up in all cases. Refer displaced or comminuted fractures.

Stress fractures of the fibula are relatively common, typically affecting the fibular neck of military recruits and athletes following vigorous training. Treat symptomatically with rest and analgesia.

Maisonneuve fracture

(See ➲ Eponymous fractures, pp. 514–18.)

Transmitted forces may fracture the proximal fibula following an ankle injury. This usually involves fracture of the medial malleolus and fracture of the proximal fibula or fibular shaft, and implies damage to the distal tibiofibular syndesmosis. Examine the proximal fibula in all ankle injuries, and X-ray if locally tender.

Pretibial lacerations

Common in the elderly following relatively minor trauma. Most pretibial lacerations can be satisfactorily treated in the ED with adhesive strips ('Steri-Strips™'). Clean and irrigate to remove clots, and close using Steri-Strips™ under appropriate anaesthesia. Aim to leave gaps of ~0.5cm between the Steri-Strips™. Apply a non-adherent dressing and light compression bandage. Instruct the patient to elevate the limb whenever possible (see Fig. 9.59). Arrange follow-up (ED or GP) for 5 days' time for wound inspection and dressing change (but leave the underlying Steri-Strips™ until the wound is healed). Consider admission for patients with poor social support.

Note: suturing pretibial wounds is not usually recommended as the pretibial skin is friable and undue tension compromises wound healing.

Complications are likely in patients with large, distally based, and poorly viable skin flaps and in patients on steroids or anticoagulants (check clotting control). Refer to plastic surgeons large lacerations where skin edges cannot be opposed or where complications are likely.

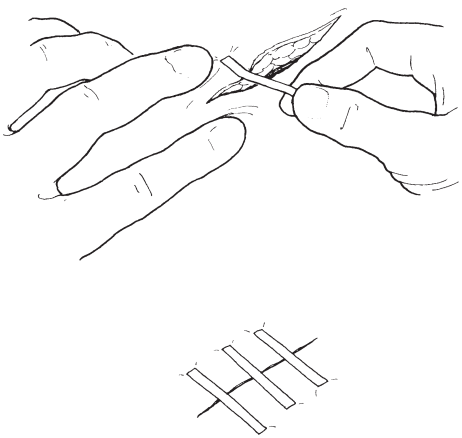

Fig. 9.59 Application of Steristrips™.

Calf and Achilles tendon injuries

Calf muscle tears

Acute tears of the gastrocnemius muscle often occur during sports. They can also occur simply from stepping from a bus or kerb or from a sudden jump. Sharp or burning pain in the calf is followed by stiffness or pain on weight-bearing. Examine for localized tenderness and/or swelling over the calf muscle bellies. The medial head of the gastrocnemius is more commonly injured.

Carefully check the Achilles tendon for signs of rupture (see ➲ Achilles tendon rupture, pp. 496–7). Differential diagnosis includes DVT (see ➲ Deep vein thrombosis, pp. 122–3) or rupture of a Baker's cyst.

Treat with analgesia, NSAID, and initial ice application. Raising the heel with a pad may also help. Advise elevation of the leg and progressive weight-bearing, as guided by symptoms. Use of crutches may be required if symptoms are severe (in this case, arrange follow-up and early physiotherapy).

Calf muscle bruising

Direct blunt calf trauma can result in haematoma formation and considerable swelling. Be alert to the possibility of compartment syndrome, particularly where there is a significant mechanism of injury (eg see ➲ Crush syndrome, pp. 406–7).

Achilles tendon rupture

Achilles tendon rupture can occur without prior symptoms during sudden forceful contraction of the calf. Usually this occurs during sports (notoriously badminton). It also occurs in other situations (eg running for a bus or missing a step and landing heavily). Patients on ciprofloxacin or oral steroids or those with a history of steroid injection of the Achilles tendon area are at ↑ risk. The patient often describes a sudden sharp pain ('snap') behind the ankle. Patients may mistakenly initially believe they have sustained a blow to the back of the ankle. Examination may reveal swelling, pain, bruising, and often a (diagnostic) palpable defect (gap) in the tendon ~5cm above the calcaneal insertion. Plantar flexion against resistance will be weaker than on the uninjured side, but do not rely on this when making a diagnosis.

Beware plantar flexion (even standing on tiptoes) may still be possible due to action of the tibialis posterior and peroneal and toe flexor muscles.

Calf squeeze test (Simmonds/Thompson's test) Kneel the patient on a chair, facing the back, with the feet hanging free over the edge. Alternatively, position the patient to lie prone on a trolley, with the ankles over the end. Gently squeeze mid-calf, and look for normal plantar flexion of the ankle (see Fig. 9.60). To avoid confusion, do not describe the result as +ve or –ve—just state 'calf squeeze test normal' or 'abnormal'.

Treatment Remains controversial, so follow local policy. Options are:

- *Conservative management*: many ruptures are managed with crutches, analgesia, and immobilization for 6 weeks in a long leg (equinus) plaster, with the ankle in plantar flexion and the knee flexed to ~45°. This is followed by careful rehabilitation under the care of the orthopaedic team and physiotherapist.
- *Primary surgical repair*: often employed in young patients and athletes. Refer to the orthopaedic team to consider this.

Note: sometimes a 'partial' Achilles tendon rupture is suspected. In this instance, the safest initial treatment is immobilization in a non-weight-bearing below-knee POP (BKPOP) with ankle flexion, crutches, and orthopaedic follow-up. USS can help to determine the state of the tendon.

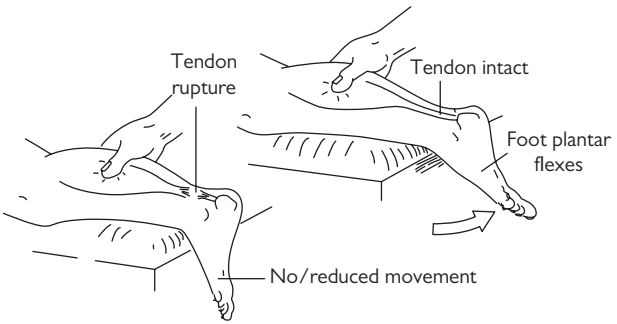

Fig. 9.60 Calf squeeze test to check the integrity of the Achilles tendon.

Achilles tendonitis/paratendonitis/tendinopathy

This frequently follows unaccustomed activity or overuse (eg dancing, jumping, running, or even walking) and may be associated with familial hypercholesterolaemia. There is usually a history of ↑ pain, aggravated by ankle movements. Examine for localized pain, swelling, and palpable crepitus over the Achilles tendon (the most common site is ~5cm from its insertion). The calf squeeze (Simmonds) test is normal. Check lipid profile.

Treat with analgesia, NSAID, and a brief period of rest (eg 1–2 days) before gradually returning to normal activities, as guided by symptoms. Occasionally, 1–2 weeks in a below-knee walking POP (BKWPOP) may be useful. A heel pad inserted into footwear may help. Athletes may benefit from removal of heel tabs from training shoes if implicated. Avoid local steroid injection, which may ↑ the risk of tendon rupture by impeding healing or by allowing premature resumption of activity.

Calf/leg pain with no history of trauma

A variety of conditions may be implicated, including:

- *Shin splints*: a variety of pathophysiological processes have been suggested, including tibial periostitis. This condition is characterized by pain over the anterior distal tibial shaft after running on hard surfaces. Advise rest and NSAID.
- *Stress fractures*: can affect the tibia (as well as the fibula—see ➲ Tibial and fibular shaft fractures, p. 494). Treat with analgesia and POP, with orthopaedic follow-up.
- *Bursitis*: inflammation of the bursae around the insertion of the Achilles tendon—responds to conservative measures.
- *DVT* (see ➲ Deep vein thrombosis, pp. 122–3).
- *Cellulitis* (see ➲ Infected wounds and cellulitis, p. 419).
- *Ischaemia* (see ➲ Acute limb ischaemia, p. 538).
- *Ruptured Baker's cyst*.

Approach to ankle injuries

Ankle injuries are amongst the most common problems presenting to the ED. Adopt a logical, consistent approach to identify which patients are likely to have a fracture and to avoid unnecessary X-rays in patients with uncomplicated sprains.

History

Establish the exact mechanism of injury. Most are inversion injuries (where the sole of the foot turns to face medially as the ankle is plantar flexed) causing damage to structures around the lateral malleolus (most notably, the anterior talofibular ligament). Eversion injuries occur less commonly and damage the structures around the medial malleolus. Hyper-dorsiflexion and plantar flexion injuries occur less frequently.

The following are relevant in the initial assessment of ankle injuries:

- A fracture is more likely in patients who are unable to weight-bear immediately following the injury.
- A 'crack' or 'snap' may be heard and is not indicative by itself of a fracture.
- Ice, analgesia, and elevation may influence the appearance of an ankle injury.

Examination

Examine from the knee down for tenderness over the:

- Proximal fibula.
- Lateral malleolus and ligaments.
- Medial malleolus and ligaments.
- Navicular.
- Calcaneum.
- Achilles tendon.
- Base of the fifth MT.

Is an X-ray required?[1]

Follow the Ottawa ankle rules (see Fig. 9.61) for adults and X-ray the ankles if patients:

- Were unable to weight-bear for four steps both immediately after the injury and at the time of examination.
- Have tenderness over the posterior surface of the distal 6cm (or tip) of the lateral or medial malleolus.

Note that tenderness over the navicular, calcaneum, base of the fifth MT, or proximal fibula require specific X-rays to exclude fractures.

Adopt a lower threshold for X-ray in the very young, the elderly, and in patients who are difficult to assess (eg intoxicated).

[1] Source: data from Stiell IG (1993) Decision rules for the use of radiography in acute ankle injuries. Refinement and prospective validation. *J Am Med Ass* 269: 1127–32.

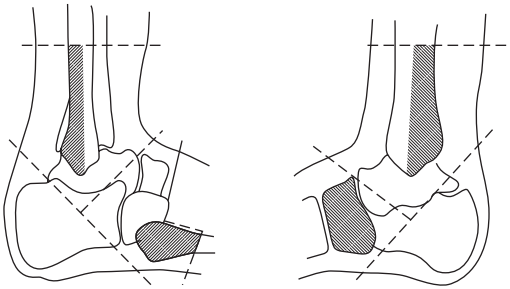

Fig. 9.61 The Ottawa ankle rules. Guidelines for X-ray in a simple ankle injury. Bony tenderness over the points indicated requires an X-ray. X-ray is also required if the patient is unable to weight-bear immediately after the injury or to walk four steps in the ED. X-ray the ankle for malleolar tenderness and the foot for metatarsal/tarsal tenderness. If the patient is not X-rayed, then they are given instructions to return after 5 days if they have trouble weight-bearing.

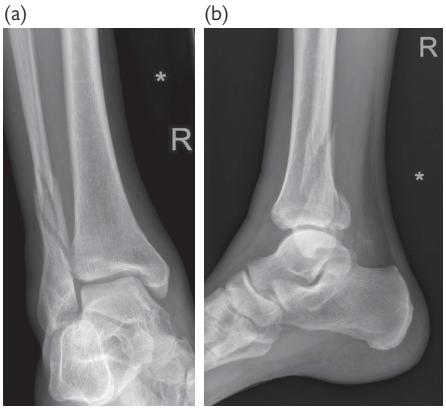

Fig. 9.62 AP and lateral ankle X-rays showing a (Weber C) fracture of the distal fibula plus a fracture of the posterior distal tibia, but no obvious talar shift.

Table 9.5 Weber classification of lateral malleolus fractures

Weber A	Fracture below syndesmosis, usually stable
Weber B	Fracture at the level of syndesmosis, may be stable or unstable
Weber C	Fracture above syndesmosis, usually unstable

Ankle fractures and dislocations

Ankle fractures

(See Table 9.5 and Figs. 9.62, 9.63, and 9.64.) Fractures around the ankle most commonly involve the malleoli—medial, lateral, and what is commonly referred to as the 'posterior malleolus' (the posterior part of the distal tibia). The ankle mortice joint allows very little rotation or angulation at the ankle joint, so forced twisting or angulation of the ankle joint causes fractures associated with ligamentous injuries and, in severe cases, disruption of the distal tibiofibular syndesmosis.

Treatment This depends upon a combination of clinical findings and X-ray appearances. Look carefully for talar shift.

- *Small avulsion fractures* essentially reflect ligament/joint capsule damage. Treat with rest, elevation, analgesia, and early mobilization, as for sprains.
- *Larger avulsion fractures* may require initial immobilization in a boot ± crutches and orthopaedic follow-up.
- *Undisplaced, isolated medial or lateral malleolar fractures* are usually stable and do well with conservative measures. Provide analgesia and crutches, and immobilize in a well-padded BKPOP cast. Advise limb elevation, and arrange orthopaedic follow-up. Note that an isolated 'high' lateral malleolus fracture may only be apparent on the lateral X-ray and may be associated with deltoid (medial) ligament injury with instability—some require ORIF.
- *Displaced fractures of the medial or lateral malleolus* require ORIF. Give analgesia and, as appropriate, IV sedation to allow reduction of talar shift. Immobilize the limb in a BKPOP slab, and refer to the orthopaedic team.
- *Bimalleolar or trimalleolar fractures* are unstable. Having attempted to reduce any significant talar shift (with appropriate sedation), place in a BKPOP, obtain fresh X-rays, and refer to the orthopaedic team.

Ankle dislocation

Dislocation of the ankle is a true emergency. Treat promptly on diagnosis. Examination shows gross deformity of the ankle, severe stretching of the skin (resulting in fracture blisters, skin necrosis, or even converting the injury to a compound fracture), and often deficits in peripheral pulses or sensation. The ankle can dislocate in the absence of associated fractures, but this is uncommon.

Treatment Prompt closed reduction and immobilization in POP usually have to precede X-ray (unless available immediately). 'Prompt treatment' does not justify reduction without considering analgesia or sedation.

- Give Penthrox®, IV analgesia/sedation, as needed, with full precautions.
- Warn that there may be a brief ↑ in discomfort as the ankle reduces.
- With the knee flexed and supported, gently grasp the heel with one hand and support the patient's calf with the other.
- Pull smoothly on the heel—it may be necessary to slightly exaggerate the deformity in order to obtain reduction. Success is indicated by return of normal ankle contours, relief of skin tension, and often dramatic relief of pain.
- Once reduced, re-check pulses and sensation; immobilize in a POP slab, and arrange check X-rays.
- Refer the patient to the orthopaedic team immediately.

Ankle X-rays

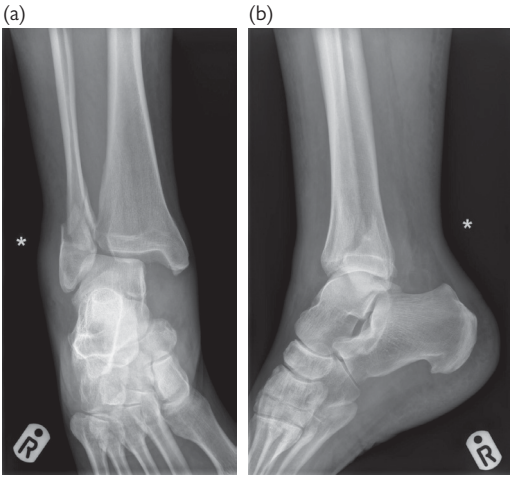

Fig. 9.63 AP and lateral views of Weber B right lateral malleolus fracture with significant talar shift requiring MUA apparent on AP view.

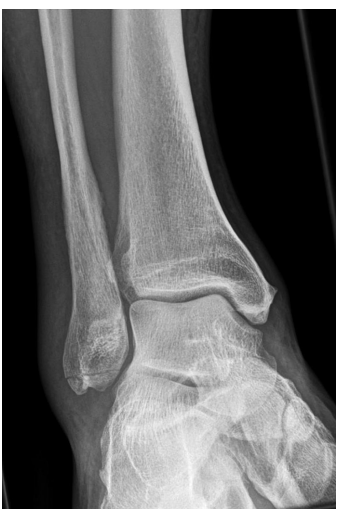

Fig. 9.64 AP of undisplaced Weber A fracture in a 73y old patient.

Ankle sprains

Clinical assessment and imaging after ankle injury are outlined in ➲ Approach to ankle injuries, p. 498. The structures most frequently injured in inversion injuries are the lateral joint capsule and the anterior talofibular ligament. ↑ injury causes additional damage to the calcaneofibular ligament and posterior talofibular ligament.

Treatment

Historically, treatment of sprained ankles has been based upon 'RICE' (rest, ice, compression, elevation), but the scientific basis for all the elements of this is distinctly lacking!

Advise initial rest, elevating the ankle above hip level, and to consider applying ice intermittently during the first 2 days for periods of 10–15min. Begin to weight-bear as soon as symptoms allow, but elevate at all other times. An elastic support from toes to knee is traditional, but of no proven value (and may be harmful by ↑ pain without speeding recovery). If used, ensure that it is not worn in bed. Advise the patient to gently exercise the ankle in all directions and to use simple analgesia regularly until symptoms improve. Most patients with minor sprains can expect full recovery in ~4 weeks. It may be possible to resume sports gradually within 2 weeks, depending on progress.

The inability to weight-bear implies more severe injury. Provide crutches to those completely unable to weight-bear despite analgesia, with advice to elevate the ankle. Arrange review at 2–4 days—if still unable to weight-bear, consider 10 days' immobilization in a boot or below-knee cast, with subsequent outpatient follow-up. Other approaches include use of adhesive strapping or preformed ankle braces. These may be useful in selected cases. Patients can usually expect good functional recovery and should not regard the ankle as 'weak'. Long-term problems (eg weakness/instability whilst walking over rough ground) are often related to ↓ ankle proprioception following immobilization, so aim to mobilize as soon as possible.

Long-term complications

Do not regard ankle sprains simply as trivial injuries—patients may suffer long-term morbidity (which often causes them to return to the ED):

- *Instability* often manifests itself by recurrent ankle sprains. Refer to physiotherapy (to include isometric exercises).
- *Peroneal tendon subluxation* reflects a torn peroneal retinaculum, allowing the peroneal tendons to slip anteriorly. The clinical presentation includes clicking and a sensation of something slipping. Movement of the foot/ankle (especially eversion) reproduces the subluxation. Refer for orthopaedic follow-up—surgery is an option.
- *Peroneal nerve injury* is relatively common, but not frequently sought for. Neurapraxia results from stretching of branches of the peroneal nerve at the time of injury, with subsequent ↓ sensation over part of the dorsum of the foot and ↓ proprioception at the ankle joint (reflecting injury to the articular branches).

Venous thromboembolism prevention

Patients who are not fully weight-bearing and are immobilized in a cast or boot may be at risk of developing a DVT, so consider the need for providing prophylaxis in the form of LMWH. Most departments employ a venous thromboembolism (VTE) risk assessment tool to guide staff. An example scoring system is shown in Table 9.6 (adapted from Derriford Hospital).

Table 9.6 An example VTE risk assessment score for adults not fully weight-bearing in cast or boot

Risk factor	Score
Overweight (BMI >30kg/m^2)	2
Achilles tendon rupture or repair	3
Previous DVT or PE	3
Pregnant or within 6 weeks of delivery	3
Complex surgery of lower leg or pelvic surgery in past 6 weeks	3
Active cancer	3
History of DVT or PE in first-degree relative	2
Unable to walk before the injury	2
Age over 60y	1
Abdominal surgery within the last 6 weeks	1
Gross varicose veins	1
Taking oral contraceptive pill or hormone replacement therapy	1
Inflammatory bowel disease (Crohn's or ulcerative colitis)	1
TOTAL SCORE generated by adding individual scores	

Recommendation using the above scoring system

- Score 0–2 = no prophylaxis.
- Score 3 or more = LMWH daily until cast/boot removed and fully weight-bearing (unless contraindications—see ➲ Contraindications to LMWH, p. 503).

Typical VTE prophylaxis regimes

- If estimated glomerular filtration rate (eGFR) is >30mL/min—dalteparin 5000U SC od.
- If eGFR is <30mL/min—enoxaparin 20mg SC od.

Contraindications to LMWH

- Concurrent use of oral anticoagulation (eg warfarin, rivaroxaban).
- Acquired or inherited bleeding disorder (eg haemophilia).
- Thrombocytopenia (platelet count <75 × 10^9).
- Uncontrolled hypertension (>230/120mmHg).
- Active bleeding from any source.

Foot fractures and dislocations

Crushing or other violent injuries to the foot can result in significant long-term disability. Delayed or inadequate treatment results in high rates of post-traumatic OA. Compartment syndromes (see ➲ Crush syndrome, pp. 406–7) or vascular injuries may occur. Amputations or severe mangling injuries of the foot are rarely suitable for reconstruction/re-implantation due to poor long-term functional results.

Talar injuries

Falls onto the feet or violent dorsiflexion of the ankle (eg against car pedals in a crash) can result in fractures to the anterior body or articular dome of the talus. Displaced fractures and dislocations frequently result in avascular necrosis.

Treat with analgesia and immobilization in a backslab POP, and refer promptly for orthopaedic treatment (may require MUA and/or ORIF). Dislocations of the talus require prompt reduction under GA.

Upper/midfoot dislocations

These injuries follow violent twisting, inverting, or everting injuries of the foot. *Peritalar/subtalar dislocations* involve the articulation between the talus and the calcaneum. Give adequate analgesia, and refer to orthopaedics for prompt reduction under GA. *Mid-tarsal dislocations* involve the mid-tarsal joint (comprising the calcaneum and talus posteriorly and the navicular and cuboid anteriorly) and are treated similarly. *Isolated dislocation* of the talus is rare and requires prompt reduction under GA.

Calcaneal fracture

Calcaneal fractures most often follow a fall from height directly onto the heels. Always exclude associated injuries of the cervical and lumbar spine, pelvis, hips, or knees. Examine for swelling, bruising, and tenderness over the calcaneum, particularly over the sides. Examine both calcanei for comparison, remembering that fractures are commonly bilateral. Examine the Achilles tendon for injury (see ➲ Calf and Achilles tendon injuries, pp. 496–7). Request specific calcaneal X-rays and scrutinize carefully breaks in the cortices, trabeculae, or subtle signs of compression (reduction in Bohler's angle—see Fig. 9.65). Refer all fractures to orthopaedic staff. The majority will require admission for elevation, analgesia, and, in selected cases, ORIF following CT scanning.

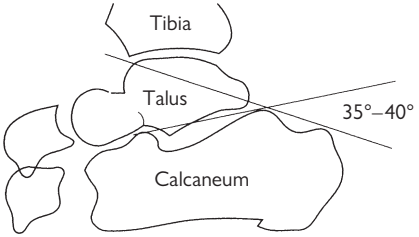

Fig. 9.65 Bohler's angle (normally 35–45°).

Clinically suspected calcaneal fracture, but X-rays normal

Sometimes, clinical suspicion of a calcaneal fracture is not confirmed by initial X-rays. Either arrange CT or treat clinically with analgesia, rest, elevation, and crutches, and arrange review at 7–10 days when consideration can be given to further imaging if symptoms persist.

Metatarsal fractures and dislocations

Multiple MT fractures may follow heavy objects falling onto the feet or, more commonly, after being run over by a vehicle tyre or wheel. In all such cases, consider the possibility of tarso-metatarsal (Lisfranc) dislocation (see Fig. 9.66). This can be easily missed on standard foot X-rays, which do not usually include a true lateral view—look to check that the medial side of the second MT is correctly aligned with the medial side of the middle cuneiform. Check for the dorsalis pedis pulse. Support in a backslab POP, and refer if there are multiple, displaced, or dislocated MT fractures.

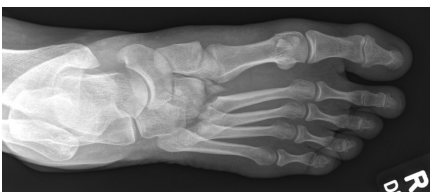

Fig. 9.66 X-ray of Lisfranc fracture–dislocation, with significant widening between the first and second MTs and fractures at the base of the second MT.

Isolated avulsion fractures of the fifth MT base

Inversion may avulse the base of the fifth MT by the peroneus brevis. Always examine this area in ankle injuries, and request foot X-rays if tender. Do not mistake accessory bones or the epiphysis (which runs parallel, not transverse, to the fifth MT base). Give analgesia, elevation, and support in a padded crepe bandage or, temporarily, in a boot if symptoms are severe. Discharge with advice or fracture clinic follow-up, according to local policy.

Jones fracture (of the fifth MT)

This is a transverse fracture of the fifth MT just distal to the inter-MT joint. It is a significant fracture, as it is prone to non-union. Treat with analgesia, crutches, BKPOP or boot, and orthopaedic follow-up.

Stress fractures of MTs

These typically follow prolonged or unusual exercise ('march fracture') but often occur without an obvious cause. The most common site is the second MT shaft. Examine for swelling over the forefoot (there may be none) and localized tenderness over the MT shaft. X-rays are usually normal initially—callus or periosteal reaction is seen at ~2–3 weeks.

Treat symptomatically with analgesia, elevation, rest, and modified daily activity, as required. Suggest a padded insole. Firm shoes or boots may be more comfortable than flexible trainers. Expect full recovery in 6–8 weeks. If unable to weight-bear, consider a brief period in a BKPOP or boot.

Toe injuries

Most toe injuries do not require X-ray.
The treatment of isolated closed fractures of the toe phalanges without clinical deformity or other complicating factors is not altered by X-rays.
X-ray the following:
- Obvious deformity, gross swelling, or suspected dislocation.
- Suspected compound injuries.
- If any tenderness over the MT head or metatarsophalangeal joint (MTPJ).
- Suspected FB.

Toe fractures

Treat uncomplicated phalangeal fractures with simple analgesia, elevation, and support with padded buddy strapping. Advise the patient to resume normal activities as soon as possible, but explain that some discomfort may be present for up to 4–6 weeks. Hospital follow-up is not normally required. Manipulate displaced fractures under LA digital block (as described for fingers in ➲ Digital nerve block, pp. 304–5). Angulated toe phalangeal fractures can be difficult to manipulate—a useful trick is to use a pen (or needle holder) placed between the toes as a fulcrum. Once satisfactorily reduced, buddy strap and confirm the position with X-rays.

Dislocated toes

Untreated, toe dislocations may cause troublesome, persistent symptoms. Reduce promptly under LA digital block and splint by buddy strapping. Always confirm reduction by X-ray, and discharge with analgesia and advice on elevation and gradual mobilization.

Compound toe injuries

Careful wound toilet, debridement, and repair are essential to ensure rapid healing and avoid infective complications. Ensure that there is adequate tetanus prophylaxis. Always clean wounds thoroughly under adequate anaesthesia (usually LA digital block); provide antibiotics and analgesia. Advise the patient to elevate the injured foot, and arrange follow-up according to local practice. More severe injuries will require exploration and repair under GA. Refer these cases to the orthopaedic team.

Mangled or amputated toes

Functional results of attempted re-implantation of amputated toes or repair of badly mangled toes are often poor. Provide analgesia, and refer to the orthopaedic surgeon for wound management and amputation of unsalvageable toes.

Soft tissue foot problems

Puncture wounds to the foot

- *'Simple' puncture wounds*: see ➲ Puncture wounds, p. 413.
- *Weever fish injuries*: see ➲ Specific bites and stings, pp. 422–3.

FBs embedded in the foot

Searching for small FBs in the sole of the foot has been likened to searching for a needle in a haystack. Follow the principles set out in ➲ Further assessment of skin wounds, p. 412. Nerve blocks (see ➲ Nerve blocks at the ankle, pp. 314–15) can be useful to allow exploration of foot wounds.

Morton's metatarsalgia

A burning discomfort radiating to the toes may result from an interdigital nerve neuroma at the level of the MT heads. The nerve between the second and third MT heads is frequently affected. There is localized tenderness, which is also reproduced on compression of the MT heads together. Advise simple analgesia and GP follow-up to consider referral to a foot surgeon.

Plantar fasciitis

Plantar fasciitis can occur spontaneously or as a chronic overuse injury. Inflammation develops in the plantar fascia, typically at its calcaneal insertion. This results in gradually ↑ burning pain in the sole of the foot and heel, which is worse on weight-bearing. Examine for localized tenderness over the calcaneal insertion of the plantar fascia and heel pad. X-ray may reveal a calcaneal spur, but this is not a useful diagnostic feature.

Advise NSAID, rest, and elevation for 1–2 days, with GP follow-up. A padded shoe insole or sorbothane heel pad may help. Severe, persistent cases are occasionally treated with local steroid injection or even surgical division of the plantar fascia.

Osteochondritis dissecans

Osteochondritis of an MT head (usually the second—Freiberg's disease) causes gradual-onset pain on weight-bearing. The cause is often unclear, but it may follow minor injury. Examination may reveal local tenderness, but little else. X-ray for evidence of flattening, widening, or fragmentation of the MT head or narrowing of the MTPJ. (See ➲ Osteochondritis, pp. 730–1.)

Treat initially with simple analgesia. Refer persistent cases to orthopaedics to consider excision of the MT head or osteotomy.

Ingrowing toenails

Refer back to the GP for elective treatment, unless there is evidence of infection. In this case, consider oral antibiotics (eg flucloxacillin) or, if there is acute paronychia, incision and drainage under LA. It is not usually appropriate to excise a wedge of nail under LA in the ED.

Atraumatic low back pain

Low back pain is the most common cause of lost work days in the UK. The initial ED approach is to identify any patients who may have immediately life-threatening problems (eg leaking aortic aneurysm) and to sort the rest into:

- *Simple ('mechanical') back pain*: no investigations or referral required.
- *Nerve root pain*: referral and investigation needed if symptoms persistent or progressive.
- *Possible serious spinal pathology*: referral and investigation required.
- *Suspected cord compression*: immediate neurosurgical/orthopaedic referral mandatory.

Psychogenic back pain is not an ED diagnosis. If in doubt, refer.

History

- *General*: note the onset and duration of symptoms, character, position and radiation of pain, and exacerbating or relieving factors. Precipitants include injuries, falls, heavy lifting, or unaccustomed activity.
- *Past history*: detail any previous back problems or surgery and other medical conditions (eg RA, OA, osteoporosis).
- *Drug history*: is the patient using analgesia (and has it helped?)? Ask about corticosteroids and contraindications to NSAIDs.
- *Social history*: ask about home circumstances, work, and stress.
- *Systemic enquiry*: weakness, altered sensation, weight loss, anorexia, fever, rigors, cough, sputum, haemoptysis, and bowel or urinary symptoms.

Examination

'Unwell' patient Assess 'ABC', and look for shock, a pulsatile abdominal mass, peritonism, radial–femoral pulse discrepancies, or asymmetry.

'Well' patient Look for signs of weight loss, cachexia, anaemia, clubbing, or muscle wasting. Inspect the back for muscle spasm, scars, scoliosis, or other deformity. If possible, watch the patient walk, looking for spasm, abnormal posture, or limping. Palpate for tenderness over the spine, lower ribs, and renal angles. With the patient supine on a trolley, look for muscle wasting in the legs. Examine both sides:

- *Straight leg raise*: note the angle which reproduces pain (lumbar nerve root irritation).
- *Crossed straight leg raise*: nerve root symptoms reproduced by lifting the contralateral leg strongly suggests lumbar disc prolapse and nerve root entrapment.

Perform a neurological examination Check tone, power, sensation, and reflexes in the lower limbs:

- L4 covers sensation of the medial lower leg, quadriceps power, and knee jerk.
- L5 covers sensation of the lateral lower leg and great toe, extensor hallucis longus power, and hamstrings jerk.
- S1 covers sensation of the little toe and lateral foot, foot plantar flexor power, and ankle jerk. Always check perineal and peri-anal sensation.

Perform a rectal examination for anal tone, masses, or blood. Examine the abdomen for masses. Document peripheral pulses and perfusion.

Investigations

Check T° and urinalysis. X-ray is indicated for some patients aged >55y or those who are systemically unwell with a history of trauma (except clinical coccyx fracture) or where malignancy, infection, or HIV is suspected. In the latter cases, also check CRP, FBC, and U&E.

MRI scan for suspected cauda equina syndrome

Request an urgent MRI if there is any suspicion of cauda equina syndrome due to a central lumbar disc prolapse:

- Pain radiating down both legs.
- Difficulty passing urine or urinary incontinence.
- Weakness and/or numbness in both legs.
- Altered perineal sensation/reduced anal tone.
- Loss of sensation of rectal fullness.
- New impotence or priapism.

Treatment

Refer urgently patients with lower limb weakness, altered perineal or perianal sensation, and sphincter disturbance (this is strongly suggestive of *cauda equina syndrome* due to central lumbar disc prolapse). An MRI scan will confirm this diagnosis—in which case, urgent consultation with a neurosurgeon will allow emergency surgical decompression to be arranged as appropriate.

Refer patients with the following: age <20 or >55y, unremitting or ↑ symptoms, widespread neurological signs, weight loss, systemic illness, pyrexia, chronic corticosteroids, osteoporosis, or HIV +ve patients with thoracic pain.

Treat simple 'mechanical' back pain with regular simple analgesia and/or NSAID, and plan to discharge the patient. Avoid routine use of opioids. Small doses of benzodiazepines (eg diazepam 2–5mg tds) may be useful but tend to cause drowsiness. Advise the patient to aim to return to normal activity, even if some discomfort persists. Avoid bed rest. Expect recovery in 4–6 weeks. Nerve root symptoms mostly resolve over weeks to months with the above treatment, physiotherapy, or manipulation. In all cases, give written and verbal advice for immediate return if limb weakness, numbness, bladder, or bowel problems occur. Advise follow-up with the GP.

Occasionally, patients require admission to get pain under control, benefitting from input from the pain team and occupational therapist.

Acute arthritis: 1

Approach

Whenever a patient presents with a painful joint, try to distinguish whether the source of pain is articular or peri-articular. Painful joints of articular origin produce warmth, tenderness, and swelling about the entire joint, with painful movement in all directions. Pain of peri-articular origin (outside the joint capsule), such as bursitis/tendinitis, tends to result in tenderness and swelling localized to a small area, with pain on passive movement only felt in limited planes.

Consider a septic cause in every patient who presents with acute arthritis. Useful investigations include WBC, CRP (or ESR), and joint aspiration.

Joint aspiration

The most important diagnostic test in patients presenting with acute arthritis is examination of the synovial fluid. When joint aspiration is performed, ensure that an aseptic technique is employed. Avoid joint aspiration through an area of cellulitis. Send fluid for Gram staining, culture, crystal examination, and cell count (see Table 9.7). Remember that the absence of bacteria on Gram staining does not exclude septic arthritis.

Table 9.7 Joint aspirate findings

	Normal	Reactive	Infectious
Colour	Colourless/pale yellow	Yellow	Yellow
Turbidity	Clear, slightly turbid	Turbid	Turbid, purulent
Cell count/mm	200–1000	3000–10,000	>10,000
Predominant cell type	Mononuclear	Neutrophil	Neutrophil
Gram stain	None	None	+ve
Culture	–ve	–ve	+ve

Causes of polyarthritis

- RA.
- Ankylosing spondylitis.
- Reactive arthritis.
- Psoriatic arthritis.
- Arthritis associated with inflammatory bowel disease.
- Viral arthritis.
- Rheumatic fever.
- Gonococcal arthritis.
- Gout.

Septic arthritis

Pyogenic infection usually reaches a joint via the bloodstream but may also develop from adjacent osteomyelitis or external skin puncture wounds. Sepsis may progress to complete joint destruction within 24hr.

Infective agents *Staphylococcus aureus, Gonococcus, Streptococcus*, TB, *Salmonella*. *Haemophilus* was the most common organism in babies before *Haemophilus* immunization but is now rare. There is an ↑ incidence in patients with RA, those taking steroids, the immunosuppressed, and those at the extremes of age. Do not overlook septic arthritis superimposed on a non-infectious joint (eg gout, rheumatoid joints).

Presentation Typically only one joint is affected and is red, painful, and swollen. No movement is usually tolerated (but steroids and analgesics can mask many of the common features of septic arthritis). The joint is held in the position of most comfort, usually in slight flexion. There may be fever, shaking, and rigors. Note that hip joint infection may not produce obvious external findings due to its deep location. Do not overlook a septic joint with signs obscured by concomitant antibiotic use. IV drug users may have involvement of uncommon joints of the axial skeleton (eg sacroiliac, vertebral, and sterno-clavicular joints).

Investigations FBC, CRP (or ESR), blood cultures, and joint aspiration (see Table 9.7). X-rays may be initially normal or show only soft tissue swelling, with displacement of capsular fat planes. Later, features of bone destruction occur.

Treatment Commence IV antibiotics (eg flucloxacillin + gentamicin), according to local guidelines. Refer urgently to the orthopaedic team for joint irrigation/drainage, analgesia, and splintage of the joint.

Note: prosthetic joint infection can be difficult to detect, but pain is typically constant and present at rest. Early infection (within 3 months of surgery) may cause obvious wound inflammation. This is less likely to be apparent in delayed or later infections. There may be little in the way of systemic symptoms. Suggestive radiological features include widening and lucency of the bone–cement interface by >2mm, movement of the prosthesis, periosteal reaction, and fractures through the cement, although X-rays may be normal. Adopt a low threshold for suspecting prosthetic joint infections and referral to the orthopaedic team.

Traumatic arthritis

Joint pain, tenderness, ↓ range of movement, and haemarthrosis after injury imply intra-articular fracture. Note, however, that septic arthritis may occur in association with trauma, even in the absence of penetrating injury.

Osteoarthritis

Elderly patients with known OA may suffer acute 'flare-ups'. Constitutional symptoms are not a feature. X-rays may show asymmetrical joint space narrowing, osteophytes, and subchondral cyst formation. Advise NSAID and/or paracetamol, plus graduated exercises.

Acute arthritis: 2

Acute gout

Most often affects the first MTPJ or knee. Precipitated by trauma, diet, diuretics, renal failure, myeloproliferative disease, and cytotoxics. Ask about previous renal stones. Look for tophi. Joint aspiration reveals negatively birefringent crystals. Septic arthritis can occur with gout—ensure aspirates are Gram-stained and cultured. X-rays may show punched-out lesions in periarticular bone. Serum uric acid may be ↑ but can be normal. Advise rest and NSAID (eg diclofenac 75mg bd), or if NSAIDs are contraindicated, consider colchicine (500mcg bd initially, slowly ↑ to qds, as needed, for symptoms, with GP review). Do not alter the treatment of patients already on long-term gout therapy. Oral steroids (eg prednisolone 30mg od for 5 days) may help those who are unable to tolerate NSAIDs or are resistant to other treatments.

Acute pseudogout

Typically affects the knees, wrists, or hips of an elderly person, with arthritic attacks precipitated by illness, surgery, or trauma. Associated with: hyperparathyroidism, haemochromatosis, Wilson's disease, hypothyroidism, diabetes, and hypophosphataemia. X-ray shows calcification in joints, menisci, tendons, ligaments, and bursae. Aspiration reveals weakly +ve birefringent crystals on polarizing microscopy. Treat symptomatically with NSAID.

Rheumatoid arthritis

Presentation Persistent symmetrical, deforming peripheral arthropathy typically starts with swollen, painful, stiff hands and feet, which gradually get worse, with larger joints becoming involved. Other modes of presentation are: persistent or relapsing monoarthritis of different large joints, systemic illness with minimal joint problems, sudden-onset widespread arthritis, and vague limb girdle aches.

Hand signs Include MCPJ and PIPJ swelling, ulnar deviation and volar subluxation at MCPJs, and boutonnière and 'swan neck' finger deformities. Extensor tendon rupture may occur.

Neck problems Degeneration of the transverse ligament of the dens carries the risk of subluxation and cord damage.

Extra-articular features Subcutaneous nodules, vasculitis, pulmonary fibrosis, splenomegaly, anaemia, pleurisy, pericarditis, scleritis, kerato-conjunctivitis.

Rheumatoid factor +ve in 70% of cases.

X-rays Show soft tissue swelling, peri-articular osteoporosis, joint space narrowing, and bony erosions/subluxation.

Treatment Refer patients who are systemically unwell. Others may benefit from NSAID, splintage, and rheumatology clinic referral.

Viral arthritis

Rubella, hepatitis B, mumps, EBV, and enteroviruses may cause arthritis. In hepatitis B, arthritis usually affects PIPJs, MCPJs, or the knees and precedes the onset of jaundice. Rubella is associated with acute symmetrical arthritis and tenosynovitis.

Rheumatic fever (See ➔ Skin lesions in multisystem disease, pp. 688–9.)

This is a non-infectious immune disease which follows infection with group A β-haemolytic streptococci. Typically, migratory or additive symmetrical polyarthritis affects the knees, ankles, elbows, and wrists.

Diagnosis Based on revised Jones criteria: evidence of previous streptococcal infection (ie recent scarlet fever, +ve throat swab, or anti-streptolysin titre >200U/mL) plus two *major* or one *major* plus two *minor* criteria.

Major criteria Carditis (pericarditis, myocarditis, or endocarditis), migratory polyarthritis, chorea, subcutaneous nodules, rash (erythema marginatum).

Minor criteria ↑ ESR/CRP, arthralgia, fever, history of previous rheumatic fever (or rheumatic heart disease), ↑ PR interval on ECG.

Investigations Throat swab, ESR, CRP, and anti-streptolysin titre.

Treatment Refer for admission, rest, aspirin, benzylpenicillin, and splintage.

Sero-negative spondyloarthropathies

These have the following common features: involvement of the spine and sacroiliac joints; inflammation, then calcification, of bony tendon insertions; peripheral inflammatory arthropathy; and extra-articular manifestations such as uveitis, aortic regurgitation, and pulmonary fibrosis.

Ankylosing spondylitis Usually presents with chronic low back pain in men aged 15–30y. Progressive spinal fusion ultimately results in a fixed kyphotic spine (which is particularly prone to fracture after injury), hyperflexed neck, and restricted respiration. Hips, shoulders, and knees may be involved. Other features are: iritis, apical lung fibrosis, plantar fasciitis, and Achilles tendonitis. There may be normochromic anaemia and ↑ CRP. X-rays show 'bamboo spine' (squared vertebrae), eroded apophyseal joints, and obliterated sacroiliac joints.

Reactive arthritis A triad of urethritis, conjunctivitis, and sero-negative arthritis may follow infection (urethritis, cervicitis, or dysentery). It may cause large joint monoarthritis of a weight-bearing leg joint. Other features include: iritis, keratoderma blenorrhagicum, circinate balanitis, plantar fasciitis, Achilles tendonitis, and aortic incompetence. Joint aspirate yields inflammatory cells, with –ve culture. WCC and CRP are ↑ .

Psoriatic arthritis Arthritis rarely precedes skin involvement.

Enteropathic arthropathies Inflammatory bowel disease is associated with spondyloarthritis and large joint mono-arthropathy. There may also be migratory polyarthritis.

Gonococcal arthritis May present with fever, migratory tenosynovitis and polyarthralgia, arthritis (knee, ankle, or wrist), and skin rash. Genital infection may be silent, especially in women. Take swabs with special culture media, and refer for investigation.

Eponymous fractures

Correctly applied, the one or two words that comprise an eponymous injury convey succinctly an otherwise involved description of a complex fracture.

Aviator's astragalus

Fractures of the neck of the talus, previously commonly observed amongst World War II pilots who crash-landed their damaged planes on returning from bombing raids. The injuries resulted from the upward thrust of the rudder bar, causing dorsiflexion forcing the talus against the anterior tibia.

Bankart lesion

Avulsion of the joint capsule and glenoid labrum, resulting from anterior dislocation of the shoulder joint. It is implicated as a causative factor for recurrent dislocations.

Barton's fracture

First described by Barton in 1839, this complex distal radial fracture is intra-articular. Displacement of the distal radial fragment allows subluxation of the carpal bones. A rare variety is called a Lentenneur's fracture.

Bennett's fracture–dislocation

These intra-articular fractures of the base of the first MC are notorious for allowing the main MC fragment to slip into a poor position. If conservative treatment (POP) is preferred to internal fixation, careful follow-up will be needed to ensure a satisfactory outcome.

Boutonnière deformity

Rupture of the central slip of the extensor tendon at the PIPJ allows the base of the middle phalanx to 'button-hole' through. The remaining two parts of the extensor expansion slip along the side of the finger and act as flexors at the PIPJ, whilst still extending the DIPJ. This produces the characteristic deformity.

Boxer's fracture

Fracture of the neck of the little finger MC rarely occurs during formal boxing when gloves are worn. It is much more commonly seen following impromptu street or bar-room brawls—innocuous-looking overlying wounds are often compound human ('reverse fight') bites (see ➲ Specific bites and stings, pp. 422–3).

Bumper fracture

The height of the average car bumper renders the adult pedestrian (who is unfortunate enough to be knocked down) particularly vulnerable to a fracture through the lateral tibial condyle into the tibial plateau. There is often an associated tear to the medial collateral knee ligament.

Chance fracture

A horizontal fracture through a vertebral body, arch, and spinous process may follow a distraction and flexion injury. It typically involves the lumbar spine of car passengers restrained only by a lap belt in a crash.

Clay-shoveller's fracture

Resistance against neck flexion may produce an avulsion of the tip of a spinous process of the lower cervical or upper thoracic spine. The lesion typically affects C7.

Colles' fracture

Abraham Colles, Professor of Surgery in Dublin, described this common distal radial fracture in 1814. The classic dinner fork deformity results from posterior displacement and angulation of the distal fragment (see ➲ Colles' fracture, pp. 454–5).

Dashboard dislocation

A high-speed head-on road traffic collision causing the dashboard to impact upon the flexed knee often results in posterior dislocation of the hip.

Dupuytren's fracture–dislocation

A highly unstable ankle injury in which there is a fracture of the distal fibula shaft and disruption of the medial ankle ligament and posterior tibiofibular ligament. The result is gross diastasis and dislocation of the talus laterally.

Duverney fracture

This is an isolated fracture of the pelvis, involving one iliac wing. Unlike a number of other fractures affecting the pelvis, it is usually a relatively stable injury.

Essex–Lopresti fracture–dislocation

A heavy fall on an outstretched hand may cause a comminuted fracture of the radial head. It is associated with tearing of the interosseous membrane (diastasis), allowing subluxation of the distal ulna.

Galeazzi fracture–dislocation

Describes the combination of a fracture of the distal radial shaft with dislocation of the distal radio-ulnar joint (see ➲ Forearm fractures and related injury, pp. 460–1). A Moore's fracture–dislocation is a similar injury, except that the radial fracture involves the distal radius, not the shaft.

Gamekeeper's thumb

Rupture of the ulnar collateral ligament of the first MCPJ was originally described as an occupational injury amongst gamekeepers, sustained whilst breaking the necks of wounded rabbits. It is now most commonly seen as a result of skiing injuries, particularly on artificial slopes, when the thumb is caught in the diamond latticework matting. The injury requires prompt diagnosis and treatment in order to avoid the long-term complication of a weak pinch grip.

Hangman's fracture

Although no longer a part of modern life in the UK, executions were previously achieved by hanging. The victim was allowed to fall several feet before being arrested by a noose. This produced rapid death by severing the cervical spinal cord. The mechanism of injury is a combination of distraction and extension, causing an unstable (hangman's) fracture of the pedicles of the axis (C2) and disrupting the intervertebral disc between C2 and C3. The fracture may also result from extension and axial compression and may occur without neurological damage.

Hill–Sachs lesion

This is an impacted compression fracture of the humeral head, which occurs during anterior shoulder dislocation. It is produced by the recoil impaction of the humeral head against the rim of the glenoid as the former dislocates. It is believed by some to be an important causative factor for recurrent dislocation.

Horse rider's knee

Frontal impact at the level of the proximal tibiofibular joint may result in posterior dislocation of the fibular head. Reduction usually requires an MUA.

Hume fracture–dislocation

This refers to the combination of an olecranon fracture with dislocation of the radial head.

Hutchinson fracture

Also known as a 'chauffeur' fracture, this name is sometimes given to a radial styloid fracture. It is classically caused by forced radial deviation of the wrist when the starting handle of an old-fashioned motor car 'kicks back'.

Ice skater's fracture

Children aged 2–8y are susceptible to distal fibula stress fractures.

Jefferson fracture

An unstable 'blow-out' fracture of C1 follows an axial load. One-third are associated with a C2 fracture.

Jones fracture

This is a transverse fracture of the base of the fifth MT, just distal to the inter-MT joint. It is a more significant injury than an avulsion fracture at the insertion of the peroneus brevis, as it is prone to non-union (see ➲ Foot fractures and dislocations, pp. 504–5).

Le Fort facial fractures

Experiments by Le Fort in 1901 were followed by descriptions of facial fractures and classification into three anatomical types (see ➲ Middle third facial fractures, pp. 380–1), including the Guérin fracture (Le Fort I).

Lisfranc fracture–dislocation

Fracture–dislocation at the tarso-metatarsal joint is a significant injury. It is named after the surgeon who described the surgical operation of partial amputation of the foot at the level of the tarso-metatarsal joint.

Luxatio erecta

First described in 1859, this is an uncommon shoulder dislocation (inferior glenohumeral dislocation). The term is derived from Latin and describes the erect hyperabducted position of the arm after dislocation. The injury follows a hyperabduction force, most often after a fall. Axillary nerve damage occurs in 60%. Reduction of the dislocation may follow overhead traction or conversion to an anterior dislocation to which conventional techniques can be applied.

Maisonneuve injury

An unstable injury in which rupture of the medial ankle ligament is associated with diastasis and proximal fibula fracture.

Malgaigne's fracture

An unstable injury in which the pelvic ring is disrupted in two places: anteriorly (through both pubic rami) and posteriorly (sacroiliac joint disruption or fracture of the ilium or sacrum).

Mallet injury

Stubbing a finger may rupture the extensor tendon (or avulse its phalangeal attachment) at the DIPJ, causing a 'mallet deformity', in which the DIPJ is held flexed. The mechanism of injury is forced flexion of the extended DIPJ.

March fracture

This refers to a stress fracture of the (usually second) MT shaft after strenuous and unaccustomed exercise. Traditionally, it was observed after heavy marching in new army recruits.

Monteggia fracture–dislocation

Fracture of the proximal ulnar shaft is associated with dislocation of the radial head. The latter is relatively easy to miss. Never accept an ulnar fracture as an isolated injury without obtaining complete views of both forearm bones, including the elbow and wrist joints.

Nursemaid's elbow

Alternative name for a 'pulled elbow' in a preschool child (see ➲ Subluxation of the radial head ('pulled elbow'), p. 750).

Nutcracker fracture

Lateral force applied to the forefoot may cause the cuboid to be fractured, as it is compressed between the calcaneum and the base of the fourth and fifth MTs.

O'Donahue's triad

A torn medial meniscus, ruptured anterior cruciate ligament, and ruptured medial collateral ligament combine to produce a significant knee injury.

Pelligrini–Stieda's disease

Ossification of the medial collateral knee ligament may follow avulsion of the superficial part from its attachment to the medial femoral condyle.

Pilon fracture

These intra-articular fractures of the distal tibia are uncommon but may also be subdivided into three types.

Pipkin fracture–dislocation

This refers to a posterior hip dislocation in which part of the femoral head is avulsed by the ligamentum teres and remains attached to it within the acetabulum. The avulsed fragment is rarely large enough to be reattached.

Pott's fracture

This term has come to be applied indiscriminately to any ankle fracture, which may be simply subdivided into 'uni-', 'bi-', or 'trimalleolar'.

Rolando fracture

Essentially a comminuted Bennett's fracture, the classic description is of Y-shaped intra-articular fractures at the base of the first MC. Treatment is difficult.

Runner's fracture

Stress fractures of the tibia are particularly common amongst runners who chalk up many miles of running on roads each week.

Smith's fracture

The so-called 'reversed Colles' fracture' was first described by Smith in 1847.

Straddle fracture

Falls astride classically produce bilateral vertical pubic rami fractures.

Tillaux fracture

An avulsion fracture of the distal lateral tibia may occur due to the pull of the anterior tibiofibular ligament.

Toddler's fracture

Undisplaced spiral fractures of the tibial shaft in children <7y often follow minimal trauma and may not be visible on initial X-ray. Subperiosteal bone formation is usually apparent radiologically by 2 weeks (see ➲ Toddler's fracture, p. 754).

Chapter 10

Surgery

Approach to abdominal pain

First identify patients requiring resuscitation or urgent treatment. The need for resuscitation is apparent in emergencies with associated hypovolaemic and/or septic shock. Less obvious, but equally important, is recognition of patients requiring urgent treatment with no clinical evidence of shock (especially ruptured abdominal aortic aneurysm).

History

The pain Determine details of site, severity, radiation, shift, character, timing, precipitating and relieving factors.

Vomiting Record anorexia, nausea, and vomiting. Ask about the nature of vomit (blood, bile, etc.). Vomiting that follows the onset of abdominal pain tends to imply a surgical cause, whereas vomiting preceding pain is often non-surgical.

Bowel disturbance Enquire about recent change of bowel habit, particularly any rectal bleeding.

Other symptoms Do not forget that abdominal pain may be due to urological, respiratory, cardiovascular, or gynaecological disorders.

Past history Determine the nature of previous surgery, preferably by obtaining old notes.

Examination

Vital signs Pulse, BP, RR, SpO_2, GCS, and T° may indicate the need for immediate intervention.

Abdomen Note distension and old scars. Check hernial orifices. Palpate gently for areas of tenderness. It is unnecessary and unkind to attempt to elicit rebound tenderness—tenderness on percussion is ample evidence of peritonitis. Perform PR/per vaginam (PV) examination only if necessary.

General Look for evidence of dehydration and jaundice. Examine the respiratory and cardiovascular systems.

Investigations

Assessment of patients with abdominal pain in the ED usually depends upon history and examination, rather than sophisticated tests. However, the following investigations may prove useful:

- *BMG*: DKA may present with abdominal pain (see ➲ Hyperglycaemic crises, pp. 160–1).
- *Urinalysis*: abdominal pain may result from urinary stones or infection. Perform a urine pregnancy test on all women of child-bearing age.
- *Blood tests*: consider FBC, U&E, amylase, LFTs, CRP, coagulation screen, and cross-matching. If clearly unwell, check ABG (or venous gas), and lactate. Although FBC is frequently requested in patients with abdominal pain, the awaited WCC rarely alters initial management.
- *ECG*: especially in patients aged >55y who may be suffering from an atypical presentation of an acute medical problem, most notably acute MI.
- *Erect CXR*: can help to exclude chest pathology which may mimic abdominal conditions (eg congestive heart failure, basal pneumonia). It may also reveal free gas under the diaphragm.

- *Abdominal X-ray*: specific indications for abdominal X-ray include suspicion of intestinal obstruction, toxic megacolon, sigmoid volvulus, and GI perforation. X-rays are not indicated in patients with suspected uncomplicated appendicitis, UTI, 'simple' constipation, gastroenteritis, GI bleeding, and acute pancreatitis. They are not 'routinely indicated' in the investigation of abdominal pain. In severely ill patients requiring imaging, CT or USS is usually more appropriate than plain abdominal X-rays.
- *USS*: reveals gallstones, free peritoneal fluid, urinary stones, and aortic aneurysms. It is increasingly used in the ED but needs specific training.
- *CT scan:* may have a role in assisting with the diagnosis of certain conditions (eg acute appendicitis in less straightforward cases).

Treatment

Prompt resuscitation and provision of analgesia are integral components of the management of serious abdominal conditions. Ensure that patients who are very sick and/or hypotensive receive early IV fluids (caution if aneurysm suspected) and full monitoring (including measuring urinary output via a urinary catheter). Follow the guidelines outlined in ➲ Sepsis, pp. 62–3.

The traditional belief that analgesia should not be given because it might mask a serious diagnosis is incorrect and cruel. Diagnosis is often easier when pain is relieved and the patient can give a better history and co-operate with examination. The most appropriate form of analgesia is usually IV opioid (eg morphine) ± IV paracetamol.

It can be difficult to decide if admission is needed for a patient with abdominal pain. Adopt a low threshold for seeking senior help. In general, if doubt exists, refer to the surgeon, who may decide that it is prudent to admit the patient for observation and investigation.

Pitfalls

- Steroids, NSAIDs, or obesity may render physical signs less obvious.
- In elderly patients with peritoneal inflammation, a lack of abdominal musculature may make it difficult to elicit guarding.
- β-blockade may mask signs of shock.
- Absence of fever does not exclude infection, especially in the very old, the very ill, and the immunosuppressed.
- When severe abdominal pain is out of all proportion to the physical findings, consider mesenteric infarction, aortic rupture/dissection, acute pancreatitis, and torsion of an ovarian cyst.
- Splenic rupture may occur after relatively trivial trauma in patients with glandular fever or haematological disorders.
- Consider gynaecological causes of abdominal pain in any woman of child-bearing age—always perform a pregnancy test.
- WCC may be normal in established peritonitis/sepsis.
- Amylase may be normal in acute pancreatitis. Conversely, moderate amylase ↑ may occur in acute cholecystitis, perforated peptic ulcer, and mesenteric infarction.

Causes of acute abdominal pain

There is a wide range of causes of abdominal pain. The cause is often initially unclear. Remember that a patient is much more likely to have a common condition (perhaps with an atypical presentation), rather than a very rare condition. Thus, a patient presenting with atypical abdominal pain is more likely to have acute appendicitis than tabes dorsalis, lead poisoning, or acute intermittent porphyria. A number of conditions are seen relatively frequently (see Table 10.1).

Table 10.1 Causes of acute abdominal pain

Surgical	Gynaecological	Medical
• Non-specific abdominal pain • Acute appendicitis • Cholecystitis/biliary colic • Pancreatitis • Peptic ulcer disease • Ruptured aortic aneurysm • Mesenteric infarction • Diverticulitis • Large bowel perforation • Intestinal obstruction • Ureteric calculi • Urinary retention • Testicular torsion • Intussusception • Cancer (especially of the colon)	• Ectopic pregnancy • Pelvic inflammatory disease • Rupture/torsion of ovarian cyst • Endometriosis • Mittelschmerz	• MI • Pneumonia • PE • Aortic dissection • Acute hepatitis • DKA • UTI • Herpes zoster • Irritable bowel syndrome • Gastroenteritis • Inflammatory bowel disease

No cause for pain found

Many patients get better without any definite cause being identified ('non-specific' abdominal pain). If symptoms have improved and there are no worrying signs, it may be reasonable to consider discharging some patients, especially if blood tests and urinalysis are normal. Obtain senior review of patients aged >70y before discharge—there is a relatively high incidence of important missed pathology (eg ruptured aortic aneurysm—see ➲ Ruptured abdominal aortic aneurysm, pp. 536–7).

When discharging a patient, provide appropriate advice about when to return if symptoms recur/worsen.

Cancer causing abdominal pain

Unexplained abdominal pain in patients >50y may be caused by cancer, especially of the large bowel. The pain may result from transient or partial bowel obstruction. Ask about previous episodes of pain, weight loss, and change of bowel habit. If there is no indication for admission, consider referral to a surgical clinic for investigation or contact the GP to suggest this.

Acute appendicitis

This common cause of abdominal pain in all ages is particularly difficult to diagnose in the extremes of age and in pregnancy. However, the diagnosis of acute appendicitis is often missed initially at all ages.

History

The classic presentation is of central colicky abdominal pain, followed by vomiting, then shift of the pain to the right iliac fossa. Many presentations are atypical, with a variety of other symptoms (eg altered bowel habit, urinary frequency), partly depending upon the position of the tip of the inflamed appendix (retrocaecal 74%; pelvic 21%; paracaecal 2%; other 3%).

Examination

In the early stages, there may be little abnormal; in the late stages, the patient may be moribund with septic shock and generalised peritonitis. Between these extremes, there may be a variety of findings, including ↑ T°, tachycardia, distress, and fetor oris. There is usually a degree of tenderness in the right iliac fossa (± peritonitis). Rovsing's sign (pain felt in the right iliac fossa on pressing over the left iliac fossa) may be present. PR examination may reveal tenderness high up on the right, with inflammation of a pelvic appendix.

Investigations

The diagnosis of acute appendicitis is essentially clinical. Scoring systems (eg Alvarado score) have been developed but cannot be relied upon. X-rays are not helpful, but do perform urinalysis ± pregnancy test. WCC (and CRP) may be ↑, but this is not invariable. CT (or USS) may help the surgeon to make the diagnosis in certain circumstances.

Differential diagnosis

Depending upon the presentation, the potential differential diagnosis is very wide—consider urinary, chest, and gynaecological causes.

Treatment

Despite studies describing non-operative treatment of acute appenditicis with antibiotics, surgical intervention remains standard treatment.

- Obtain IV access and resuscitate as necessary. Commence IV fluids (eg 1000mL of 0.9% saline) if there is evidence of dehydration.
- Give IV opioid and antiemetic (eg slow IV metoclopramide 10mg).
- If acute appendicitis is likely, or even possible, keep 'nil by mouth' and refer to the surgeon. If appendicectomy is required, preoperative antibiotics (eg amoxicillin + gentamicin + metronidazole) ↓ infective complications.
- If a diagnosis of acute appendicitis seems very unlikely and the patient is going to be discharged (± after surgical review), ensure that (s)he is advised to return for review (or seek medical attention) if symptoms worsen.

Appendix mass

Untreated, acute appendicitis may proceed to perforation, with generalized peritonitis, or may become 'walled off' to produce a localized right iliac fossa inflammatory mass. There are many other causes of such a mass, including: caecal carcinoma, Crohn's disease, ovarian mass, pelvic kidney, ileocaecal TB, psoas abscess, actinomycosis, and Spigelian hernia. Refer to the surgeon for further investigation and management.

Acute pancreatitis

This is a relatively common serious cause of abdominal pain in the middle-aged and elderly, with an incidence of ~5 per 100,000/y.

Causes

Most are due to gallstones or alcohol. Many are idiopathic. Other causes include: hypothermia, trauma, infection (glandular fever, mumps, Coxsackie, and infectious hepatitis), hyperlipidaemia, hyperparathyroidism, drugs (steroids, azathioprine, thiazides, and statins), PAN, pancreatic cancer, post-endoscopic retrograde cholangiopancreatography (ERCP), and scorpion stings.

Symptoms

Typically, the complaint is of severe constant epigastric pain radiating to the centre of the back (possibly relieved by leaning forward), with associated nausea and vomiting.

Signs

The patient may be distressed, sweating, and mildly pyrexial. Look for evidence of shock—there may be a need for urgent resuscitation. Abdominal tenderness is likely to be maximal in the epigastrium ± guarding. The oft-quoted, but uncommon, bluish discoloration in the loins (Grey–Turner's sign) only develops after several days.

Investigations

- Check BMG and SpO_2.
- Serum amylase is likely to be grossly ↑ to >4 times upper limit of normal range (but if not diagnostically ↑, consider urinary amylase level). Note that mild ↑ amylase may occur in a wide range of other acute abdominal conditions.
- Serum lipase may be measured, instead of amylase.
- FBC may reveal ↑ WCC.
- U&E, Ca^{2+}, LFTs, glucose—hypocalcaemia is relatively common.
- Coagulation screen.
- CXR, ECG, ABG, including lactate.

Treatment

- Provide O_2.
- Obtain IV access and resuscitate with IV crystalloid fluids as necessary.
- Give IV analgesia (eg morphine titrated according to response—see Approach to abdominal pain, pp. 520–1).
- Give an antiemetic (eg cyclizine 50mg or metoclopramide 10mg slow IV).
- Insert a NG tube.
- Insert a urinary catheter and monitor urine output.
- Consider early involvement of the critical care team and possible insertion of a central venous line to monitor the CVP and guide IV fluid therapy in the seriously ill, particularly the elderly.
- Contact the appropriate specialist(s) and transfer to HDU/ICU.

Complications of acute pancreatitis

Acute pancreatitis has significant mortality. Early complications include ARF, DIC, hypocalcaemia, and ARDS. Later, pancreatic abscess or pseudo-cyst may occur.

Prognosis of acute pancreatitis

The risk of death may be predicted according to the number of prognostic indicators present (Glasgow Imrie scoring system).

Three or more of the following on admission and subsequent repeat tests over 48hr constitute severe disease:

- Partial pressure of O_2 (pO_2) <7.9kPa.
- Age >55y.
- Neutrophils ↑ (WCC >15 × 10^9/L).
- Corrected Ca^{2+} <2mmol/L.
- Raised blood urea >16mmol/L.
- Elevated enzymes (serum LDH >600U/L; AST >100U/L).
- Albumin <32g/L.
- Sugar ↑: fasting glucose >10mmol/L.

Chronic pancreatitis

The term chronic pancreatitis implies permanent pancreatic damage. The condition most often results from alcohol excess. Some patients with chronic pancreatitis present frequently to the ED requesting opioid analgesia (and, increasingly, cyclizine too). This can pose a difficult problem for the doctor who has not treated them previously. Patients with chronic pancreatitis can experience severe episodes of acute pancreatitis. Follow the approach outlined below and review previous hospital notes/letters early to help guide treatment. For patients with alcohol-related chronic pancreatitis, consider the need for IV thiamine (eg Pabrinex®) and diazepam or chlordiazepoxide (see ➲ Alcohol withdrawal, p. 640).

Admission or discharge?

Many patients with chronic pancreatitis can be successfully managed on an outpatient basis without admission to hospital. Refer for admission (usually under the medical team) patients with dehydration/shock, severe illness, uncontrolled pain, or vomiting.

Biliary tract problems

Most emergency biliary tract problems relate to gallstones. Both solitary cholesterol and multiple mixed gallstones are common amongst the middle-aged and the elderly. Pigment stones comprise a small proportion—they occur in hereditary spherocytosis, malaria, and haemolytic anaemia.

Complications of gallstones

Acute/chronic cholecystitis, biliary colic, obstructive jaundice, Mirizzi's syndrome, ascending cholangitis, mucocele, empyema, acute pancreatitis, gallstone ileus, gall bladder cancer.

Acute cholecystitis

History Impaction of gallstones with acute inflammation of the gall bladder usually manifests itself by right hypochondrial pain radiating to the right side of the back ± vomiting.

Examination Look for features of an acute inflammatory process. Fever is frequently present, combined with right hypochondrial tenderness (particularly felt on inspiration—Murphy's sign). There may be a palpable mass—this is also a feature of mucocele and empyema (the latter causing high fever, extreme tenderness, and septic shock).

Management

- Provide IV analgesia and antiemetic (see ➲ Analgesics: morphine, p. 286).
- Check FBC (WCC often ↑), U&E, glucose, amylase, and LFTs.
- CXR, ECG (in case pain is due to an atypical presentation of MI).
- USS will confirm the diagnosis (tenderness on pressing the USS transducer over the area where the thickened gall bladder containing stones is located is called the ultrasonic Murphy's sign).
- Commence antibiotics (eg cefotaxime 1g IV) and refer to the surgeon.

Biliary colic/chronic cholecystitis

Patients (sometimes with known gallstones) may present with short-lived recurrent episodes of epigastric/right hypochondrial pain ± radiation to the back. This pain of biliary colic/chronic cholecystitis may be difficult to distinguish from other causes, including peptic ulcer disease. If the pain has subsided and there are no residual abnormal physical signs, discharge the patient with arrangements for GP or surgical outpatient follow-up.

Common bile duct stones

Stones in the common bile duct can cause problems, including acute pancreatitis (see ➲ Acute pancreatitis, pp. 524–5), obstructive jaundice, and ascending infection.

Obstructive jaundice Biliary obstruction results in ↑ jaundice with pale stools and dark urine (± pain). Acute hepatitis and cholangio-/pancreatic carcinoma may present in a similar fashion. A palpable gall bladder implicates pancreatic carcinoma as the more likely diagnosis (Courvoisier's law: 'In the presence of jaundice, if the gall bladder is palpable, the cause is unlikely to be a stone').

Ascending cholangitis Biliary stasis predisposes to infection, characterized by Charcot's triad (abdominal pain, jaundice, and fever). The patient may be very ill and require resuscitation for septic shock (see ➲ Shock, pp. 64–5) and combination IV antibiotics (eg amoxicillin, gentamicin, and metronidazole).

Peptic ulcer disease

Perforated peptic ulcer

History Perforation of a gastric or duodenal ulcer is usually a severely painful sudden event. It may occur in those without known peptic ulcer disease, although close questioning may reveal recent symptoms attributed to 'indigestion'. Sudden localized epigastric pain spreads to the remainder of the abdomen—the pain is worse on coughing or moving and may radiate to the shoulder tip.

Examination Although distressed, patients often prefer to lie still, rather than roll about. However, some patients in extreme pain writhe or roll in agony and are unable to keep still for examination or X-rays until analgesia is given. Absent bowel sounds, shock, generalized peritonitis, and fever develop as time passes.

Investigations An erect CXR will demonstrate free gas under the diaphragm in ~75% of patients with perforated peptic ulceration. In those cases where the diagnosis is suspected, but not proven by X-ray, consider a contrast CT scan.

Other relevant investigations are: U&E, glucose, amylase (may be slightly ↑), FBC (WCC typically ↑), SpO_2, ABG, and ECG/troponin (ensure symptoms do not reflect MI, rather than peptic ulcer disease).

Treatment

- Give O_2.
- Provide IV analgesia (eg morphine titrated according to response).
- Give an antiemetic (eg slow IV metoclopramide 10mg).
- Resuscitate with IV 0.9% saline.
- Refer to the surgeon and give IV antibiotics (eg cefotaxime 1g and, in late presentations, metronidazole 500mg as well).

Other GI perforations

Perforations may affect any part of the GI tract, but the chief causes are peptic ulceration, trauma, diverticular disease, and colonic carcinoma. The emergency treatment principles are similar to those of perforated peptic ulcer (described in ➲ Perforated peptic ulcer, p. 527). Bowel perforation usually results in gas under the diaphragm on an erect CXR, but remember that there are other possible causes, including: recent surgery, peritoneal dialysis, gas-forming infections, and occasionally vaginal gas insufflation during waterskiing or oral sex.

Other presentations of peptic ulcer disease

Peptic ulcer disease may also present with upper GI haemorrhage (see ➲ Upper gastrointestinal bleeding, pp. 126–7) or pain from oesophagitis, gastritis, or duodenitis. If the presentation suggests inflammation of the upper GI tract and there is no evidence of serious complications, consider discharging the patient with an antacid and GP follow-up. It is not usually appropriate to initiate therapy with PPIs in the ED without an accurate diagnosis. *Note*: some patients require an urgent endoscopy arranged by the GP to exclude cancer (eg older patients with chronic pain, weight loss, and anaemia).

Mechanical intestinal obstruction

Intestinal obstruction may be *mechanical* or *paralytic* in nature. Causes of mechanical intestinal obstruction are shown in Table 10.2.

Table 10.2 Causes of mechanical intestinal obstruction

- Adhesions (most common)
- Obstructed hernia (commonly: inguinal, femoral, para-umbilical, incisional; rarely: obturator, Spigelian, lumbar)
- Tumours (gastric, caecal, or most commonly sigmoid)
- Peptic ulcer disease
- Pelvic kidney
- Enlarged gall bladder
- Intussusception (see ➲ Intussusception, p. 721)
- Volvulus (gastric, caecal, or most commonly sigmoid colon—see ➲ Large bowel emergencies, pp. 532–3)
- Inflammatory mass (eg diverticular, Crohn's)
- Gallstone ileus
- Common iliac artery aneurysm

History

Classic symptoms of intestinal obstruction are: abdominal pain, distension, vomiting, and constipation. The exact presentation depends upon the site of obstruction and the underlying cause. Ask about previous surgery. A history of severe pain suggests strangulation and developing ischaemia in a closed loop. The nature of the vomit (eg faeculent) may give a clue to the site of obstruction.

Examination

Check T°. Look for evidence of dehydration or shock. Carefully examine the hernial orifices (an obstructed femoral hernia is otherwise easily missed). Inspect for scars from old surgery. Note any distension and areas of tenderness (peritonism implies the surgical problem is advanced). Bowel sounds may be tinkling or absent. PR examination may reveal an 'empty' rectum.

Blood tests U&E, glucose, amylase, FBC, LFTs, clotting, group and save.

X-rays Request CXR and supine abdominal X-rays. If there is no convincing evidence of obstruction on the supine view, but still a high index of clinical suspicion, consider requesting a CT scan. X-rays may demonstrate distended loops of bowel (with multiple fluid levels visible on an erect abdominal view). The site and nature of distended bowel loops may suggest the site of obstruction—small bowel obstruction results in dilated loops of bowel with valvulae conniventes completely traversing the lumen, whereas large bowel haustra do not completely cross the lumen.

Note that although gallstone ileus is rare, X-rays may be diagnostic—the fistula between the bowel and the gall bladder allows gas into the biliary tree, which shows up as an abnormal Y-shaped gas shadow in the right hypochondrium (see Fig. 10.1).

ECG Obtain this if the patient is middle-aged or elderly.

ABG If the patient is shocked, check SpO_2, ABG, and lactate.

Old notes Review previous hospital case notes/letters.

Management of mechanical obstruction

- Insert an IV cannula and start IVI of 0.9% saline.
- If the patient is shocked, resuscitate with O_2 and IV fluids and insert a urinary catheter. Consider the need to insert a central venous line to guide resuscitation and involve ICU specialists at an early stage.
- Provide analgesia (eg IV morphine titrated according to response—see ➲ Analgesics: morphine, p. 286).
- Give an antiemetic (eg cyclizine 50mg).
- Insert an NG tube.
- Refer to the surgical team for ongoing care.

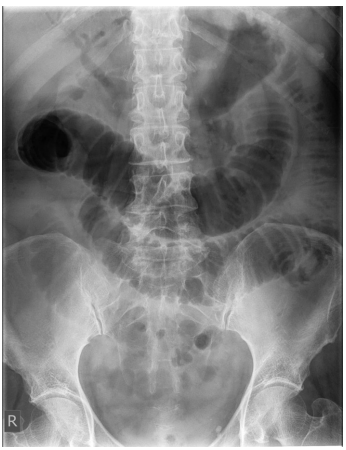

Fig. 10.1 Gallstone ileus. Note the combination of small bowel obstruction with gas in the biliary tree (appearing as a prominent Y-shaped shadow in the right upper abdomen).

Paralytic intestinal obstruction

This is relatively rare in the ED. Causes include postoperative ileus, electrolyte disturbance (eg hypokalaemia), and pseudo-obstruction.

Intestinal pseudo-obstruction

This condition results from chronic impairment of GI motility. Many of the patients affected are elderly and taking tricyclic antidepressants or other drugs with anticholinergic actions. Although pseudo-obstruction may involve any part of the GI tract, it typically presents with colonic distension. On rare occasions, this may be sufficiently severe to rupture the caecum or cause hypotension by compressing the inferior vena cava and blocking venous return. There may be a diagnostic X-ray appearance showing gas in the bowel all the way to the rectum, whereas in a classical, more proximal obstruction, gas will be absent from the rectum. Treatment of acute colonic distension from pseudo-obstruction is by decompression using a colonoscope.

Mesenteric ischaemia/infarction

Acute mesenteric infarction

Abrupt cessation of the blood supply to a large portion of gut results in irreversible gangrene of the bowel in a relatively short space of time. This is associated with high mortality. Unfortunately, the diagnosis can be difficult to make—the challenge therefore lies with making it early.

Pathophysiology

One or more of the following processes may be responsible:

- Mesenteric arterial embolism (often associated with AF).
- Mesenteric arterial thrombosis.
- ↓ mesenteric arterial blood flow (eg hypotension secondary to MI).
- Mesenteric venous thrombosis.

Most cases involve either arterial embolism or thrombosis.

History

Acute mesenteric infarction usually occurs in middle-aged or elderly patients. It is often heralded by severe, sudden-onset, diffuse abdominal pain. Typically, the severity of the pain initially far exceeds the associated physical signs. The pain may radiate to the back.

Some patients have a preceding history of chronic mesenteric ischaemia, with pain after meals and weight loss. There is often an associated history of vascular disease elsewhere (eg intermittent claudication).

Examination

Shock, absent bowel sounds, abdominal distension, and tenderness are late signs. Initially, there may be little more than diffuse mild abdominal tenderness. If the diagnosis is suspected, search carefully for evidence of an embolic source (eg AF, recent MI with a high risk of mural thrombus, aortic valve disease or valve prosthesis, recent cardiac catheter).

Investigations

- U&E, BMG, and laboratory blood glucose.
- Amylase may be moderately ↑.
- FBC may demonstrate ↑ WCC.
- Coagulation screen.
- Group and save.
- ABG typically reveals severe metabolic acidosis, and lactate may be ↑.
- X-rays may show non-specific dilatation of bowel loops and, in advanced cases, gas within the hepatic portal venous system.
- ECG may demonstrate AF.
- CT angiography may reveal the exact nature of the underlying problem and help to guide treatment.

Management

If the diagnosis is suspected:

- Resuscitate with O_2 and IV fluids.
- Provide analgesia (eg IV morphine titrated according to response).
- Consider broad-spectrum IV antibiotics.
- Refer urgently to the surgeon to consider heparin/revascularization.

Ischaemic colitis

Chronic arterial insufficiency to the bowel usually affects the mucosa and submucosa, typically in the region of the splenic flexure (the junction of the territory supplied by the superior and inferior mesenteric arteries).

The patient presents with abdominal pain, starting in the left iliac fossa. Loose stools with blood may be passed. The patient may have had previous similar episodes and exhibit evidence of cardiovascular disease. Examination may reveal low-grade pyrexia, tachycardia, and colonic tenderness, with blood PR.

Check FBC, U&E, group and save, ECG, and CXR. Plain abdominal X-rays may show 'thumb-printing' (submucosal colonic oedema), typically at the splenic flexure (see Fig. 10.2). Provide analgesia and IV fluids, and refer to the surgical team.

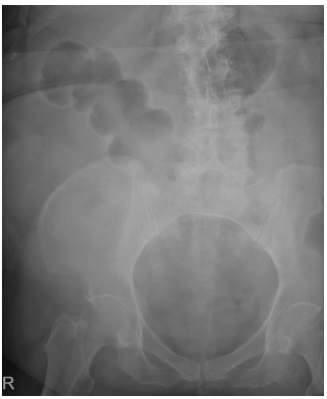

Fig. 10.2 Abdominal X-ray showing dilated transverse colon with mural oedema.

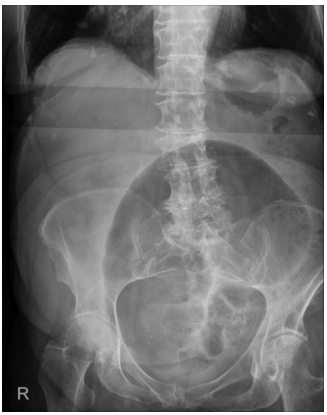

Fig. 10.3 Abdominal X-ray showing sigmoid volvulus.

Large bowel emergencies

Sigmoid volvulus

Usually occurs in the elderly with initially intermittent cramping lower abdominal pain and progressive abdominal distension, which may be spontaneously relieved by passage of large amounts of flatus/faeces. Some patients progress to complete obstruction—marked distension progressing to fever and peritonitis suggests strangulation.

Plain abdominal X-ray typically shows a large single dilated loop of colon (a 'bent inner tube') on the left side, with both ends down in the pelvis (see Fig. 10.3).

Refer to the inpatient surgical team for sigmoidoscopy/insertion of a flatus tube (if not strangulated) or surgery (if strangulated).

Caecal volvulus

Most common between the ages of 25 and 35y. Patients have symptoms of acute-onset small bowel obstruction.

Plain abdominal films usually show one large dilated segment of the colon in the mid-abdomen with distended small bowel loops and empty distal large bowel. Refer to the surgical team.

Acute diverticulitis

Diverticulosis is common in the middle-aged and elderly, particularly affecting the sigmoid colon. Without significant complications, there may be a change in bowel habit with passage of mucus.

Acute diverticulitis results from inflammation ± perforation of a diverticulum and may be confined to the colonic wall by the serosa. If this perforates, then inflammation may remain localized (pericolic abscess) or spread (frank peritonitis). Symptoms and signs reflect the extent of the infection—there may be lower abdominal dull constant pain and low-grade fever, with tenderness, rigidity, and occasionally a mass in the left lower quadrant. The elderly (the group most at risk of diverticulitis and its complications) and those on immunosuppressants may not manifest the expected pyrexia and signs of peritonitis.

Investigations Check FBC, U&E, CRP, group and save, and blood cultures. Plain abdominal X-rays may show non-specific changes and help to exclude perforation/large bowel obstruction. An erect CXR often shows copious subdiaphragmatic gas in free perforations. CT is the best investigation to delineate the extent of the problem and associated complications.

Treatment Give analgesia and IV fluids; keep fasted, and refer to the surgeon. Start broad-spectrum antibiotics (eg cefuroxime + metronidazole).

Complications

- Perforation: may be localized and walled off (forming an abscess) or generalized.
- Intestinal obstruction: both large and small (due to adherent loops).
- Massive PR bleeding.
- Fistulae to adjacent structures: small bowel, uterus, vagina, bladder.
- Post-infective strictures.

Ulcerative colitis

Severe acute colitis is characterized by the passage of >6 loose bloody motions per day, together with systemic signs (tachycardia, fever) and hypoalbuminaemia. There is a risk of haemorrhage, perforation, and toxic megacolon.

Resuscitate with IV fluids and give IV hydrocortisone (100–200mg), then refer to the inpatient gastroenterology service for aggressive medical therapy (IV and PR steroids, IV fluids) and joint review by medical and surgical teams. Surgery may be required for complications, especially toxic megacolon.

Suspect toxic megacolon if the colonic width is >5.5cm on abdominal X-ray (this sign is associated with a 75% risk of requiring colectomy).

Refer any patient who presents with suspected new-onset ulcerative colitis for investigation and control of the disease.

Crohn's disease

Colonic Crohn's disease may present as colitis with bloody diarrhoea, urgency, and frequency, similar to ulcerative colitis. Fibrosis may cause diarrhoea or obstructive symptoms.

Peri-anal disease with chronic anal fissure may be the first presenting symptom. Emergency surgery is indicated in acute fulminating Crohn's colitis with bleeding, toxic dilatation, or perforation.

Epiploic appendagitis

Primary inflammation of one of the hundreds of appendices epiploicae on the antimesenteric colonic border may present in a similar fashion to acute diverticulitis or appendicitis. However, T° and WCC are usually normal. Although often diagnosed at laparotomy, CT scan may be characteristic, allowing conservative treatment (including IV analgesia).

Irritable bowel syndrome

Patients are usually aged 20–40y with a prolonged history of intermittent symptoms—altered bowel function (diarrhoea, constipation, or diarrhoea alternating with constipation). Typically, the abdominal pain is crampy/aching and localized in the lower abdomen over the sigmoid colon. The patient may report that the pain is eased by the passage of stool or flatus. Examination fails to reveal any worrying features. The diagnosis is one of exclusion—be vigilant for clues that may point to other organic disease.

Mesenteric ischaemia/infarction

(See ➲ Mesenteric ischaemia/infarction, pp. 530–1.)

Anorectal problems

Any PR bleeding requires surgical follow-up to exclude malignancy.

Complications of haemorrhoids ('piles')

- *Bleeding*: haemorrhoids typically cause painless, bright red PR bleeding associated with defecation, but blood is not mixed with the stools. Check the abdomen and inspect the anus—if there is no prolapsed or external haemorrhoid, perform PR and arrange GP/surgical follow-up.
- *Prolapsed internal haemorrhoids (piles)* are acutely painful—treat conservatively with adequate analgesia (may need admission), bed rest, and stool softeners.
- *Thrombosed external pile* is due to rupture of a tributary of the inferior haemorrhoidal vein, producing a peri-anal haematoma. One or more dark blue nodules covered with squamous epithelium may be visible at the anus and a clot palpable. Refer to the surgeon to decide between incision and drainage under LA or conservative management.

Anal fissure

Typically causes severe pain on defecation and for 1–2hr afterwards. There may be blood on the toilet paper, but usually bleeding is minimal. The fissure is located just inside the anal orifice and is usually associated with the passage of hard stools. Most are located posteriorly in the midline. PR examination may be impossible due to pain, but the fissure is often visible with traction of anal skin.

Treatment Prescribe analgesia and stool softeners. Most heal spontaneously, but the presence of significant ulceration, hypertrophied tissue, or a skin tag suggests chronicity and the need for surgical follow-up. Be suspicious of those fissures not in the midline and those that are multiple (the differential diagnosis includes chronic inflammatory bowel disease, anal cancer, and adenocarcinoma of the rectum invading the anal canal).

Pruritus ani

Not strictly an emergency problem. There are numerous possible causes:

- Poor hygiene.
- Fissure, prolapsing piles, fistulae, rectal prolapse, anal cancer.
- Contact dermatitis due to local applications (especially LAs).
- Threadworms.
- Part of a general condition (eg obstructive jaundice, lymphoma, severe iron deficiency anaemia, uraemia, diabetes).
- Lichen sclerosis.
- STI (herpes, anal warts, HIV).

Treatment requires identification of the underlying problem—refer to the GP. In the meantime, advise avoidance of ointments and creams.

Pilonidal abscess

An infected pit in the natal cleft causes pain and/or offensive discharge.

Treatment Refer to the surgical team. Treatment may involve initial incision and drainage, followed by healing, then elective excision of the sinus.

Note: fissures, tears, or bruising around the anus of a child arouse suspicion of abuse. Refer to a specialist and avoid rectal examination.

Anorectal abscesses

Most begin with infection involving an anal crypt and its gland, from which it can spread between the external and internal sphincters to a variety of sites—these determine its symptomatology and mode of presentation.

Peri-anal and ischiorectal abscesses account for ~80% of cases. In 20%, there is a clear predisposing cause such as inflammatory bowel disease, anorectal cancer, or anal fissure (see Fig. 10.4).

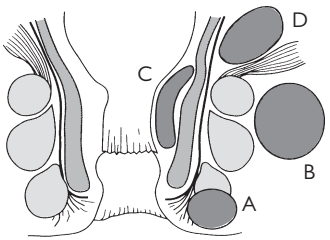

Fig. 10.4 Four types of anorectal abscesses: (A) peri-anal; (B) ischiorectal; (C) submucous; and (D) pelvi-rectal.

Clinical features Pain is a prominent initial feature of peri-anal and superficial ischiorectal abscesses, followed by local signs of inflammation. Patients complain of persistent dull, throbbing pain, made worse by walking and sitting and prior to defecation. Such symptoms are less evident with deep infections, which tend to develop slowly with pyrexia and systemic upset. Peri-anal abscesses produce localized fluctuant red, tender swellings close to the anus. With ischiorectal sepsis, the findings are more diffuse and fluctuance is a late finding. Deeper infections are less obvious—PR examination may reveal a mass or a tender area of induration.

Treatment Provide analgesia and refer to the surgical team for incision and drainage under GA.

Venereal proctitis

The organisms are similar to those transmitted by vaginal intercourse—assume >1 type of organism is present. Patients complain of pain, irritation, discharge, and bleeding. Consider gonococcus, *Chlamydia*, syphilis, and herpes simplex. Refer urgently to a GU specialist.

Rectal foreign bodies

X-rays may demonstrate the position and shape of FBs. More especially, look for the presence of any free air—perforation of the rectum or colon is the most frequent and most serious complication, in which case give IV antibiotics (eg cephalosporin + metronidazole). Refer the patient for removal of the FB by the surgical team.

Rectal prolapse

Complete prolapse of all bowel layers occurs particularly in elderly ♀ after excessive straining. If it is not reduced easily by gentle manipulation, refer to the surgical team.

Ruptured abdominal aortic aneurysm

Rupture of abdominal aortic aneurysm causes a large number of deaths, many of which occur suddenly out of hospital. Even when the patient reaches hospital alive, there is significant mortality. The best chance of survival lies with early diagnosis, prompt resuscitation, and rapid transfer to theatre. Most aneurysms are saccular and occur in the infrarenal aorta—haemorrhage after rupture is usually into the retroperitoneum and may be initially contained locally. Aneurysm extension to involve the renal arteries renders surgery more difficult and ↑ the risk of postoperative complications.

History

Presentation is highly variable, ranging from PEA cardiac arrest to painless sudden collapse of obscure origin, through to a classical history of central abdominal and lower back pain in a patient with a known aneurysm. Abdominal and/or back pain is usually a feature—typically sudden in onset and severe in nature.

Examination

The seriously ill patient may present a characteristic picture—distressed, pale, sweating, tachycardic, and hypotensive, with mottled skin of the lower body and a tender, pulsatile abdominal mass. One or both femoral pulses may be absent.

Diagnosis

Ruptured aortic aneurysm is not infrequently misdiagnosed as ureteric colic. Adopt a low threshold of suspicion in any middle-aged or elderly patient who presents with back pain, abdominal pain, or collapse. In some patients (eg the obese), it may be difficult to be certain about the presence of a pulsatile abdominal mass. In such cases, assume there is a ruptured abdominal aortic aneurysm and commence resuscitative measures, whilst appropriate experts are summoned and relevant emergency confirmatory investigations (eg USS or CT scan—see Fig. 10.5) are performed. It may be safer and quicker to perform USS in the ED, rather than transfer the patient for CT scan.

Management

- Provide high-flow O_2.
- Obtain venous access with two large-bore venous cannulae.
- Send blood for FBC, U&E, glucose, baseline coagulation screen, LFTs, and emergency cross-matching, as per the hospital's massive transfusion protocol.
- Give IV analgesia (eg morphine titrated according to response).
- Provide IV antiemetic (eg cyclizine 50mg).
- Avoid excessive IV fluid resuscitation. Treat major hypovolaemia, but accept moderate degrees of hypotension (systolic BP >90mmHg). In general, patients who are conscious and passing urine require minimal IV fluid therapy until theatre. Ensure O-negative blood is available if needed.
- Obtain a CXR.
- Insert a urinary catheter and a radial arterial line, and record an ECG.
- Call the vascular team early to consider open or endovascular repair.
- Ensure that other relevant staff (eg ICU, emergency theatre staff) are informed.

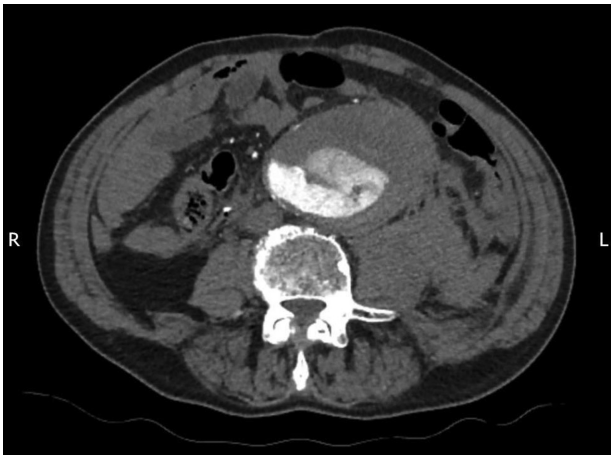

Fig. 10.5 CT showing a leaking abdominal aortic aneurysm.

Note that in patients with very poor renal function, there may be understandable concern about administering contrast. In this situation, consider a non-contrast CT scan which can still give useful information.

Pain after previous surgery for aortic aneurysm

If a patient presents with abdominal pain and has previously undergone aortic aneursym repair, consider complications of the surgery.

Infection of graft

This can be difficult to diagnose. It may present with evidence of sepsis and/or anastomotic leak. Take blood tests, including FBC and CRP; obtain a CT scan, and involve the vascular surgeon.

Anastomotic leak

A leaking anastomosis can produce a pseudoaneurysm and/or catastrophic haemorrhage. Pseudoaneurysms may produce a painful pulsatile mass—evaluate by CT, and involve the vascular surgeon.

Endoleak

Patients who have undergone endovascular repair may develop an 'endoleak' whereby blood flows outside the graft (but within the original aneurysm sac). Although many endoleaks may be managed conservatively, there is a risk of external rupture of the aneurysm sac.

Fistula

Aortoenteric and aortocaval fistulae can occur as primary problems associated with an untreated aortic aneurysm. An aortoenteric fistula may also develop after surgical repair, often with massive GI haemorrhage with an underlying associated graft infection.

Acute limb ischaemia

Clinical features

The cardinal features of acute limb ischaemia are shown in Table 10.3.

Table 10.3 Cardinal features of acute limb ischaemia (6 Ps)

Men	Women
• Pain	• Pulselessness
• Paraesthesiae (later anaesthesia)	• Paralysis (muscle damage)
• Pallor	• Perishing cold

Where acute arterial occlusion occurs in a previously normal limb, the features of ischaemia will be ↑ because of the absence of a developed collateral circulation. In the absence of a traumatic cause (either direct arterial injury or indirect injury such as compartment syndrome—see ➲ Crush syndrome, pp. 406–7), the most common causes are embolism or thrombosis.

Embolism Cardiac sources account for >80% (AF, post-MI, prosthetic valves, atrial myxoma, vegetations, and rheumatic heart disease). Acute embolic events affect legs more often than arms (especially artery bifurcations).

Risk factors Diabetes, smoking, hypertension, hypercholesterolaemia.

Past history Ask about previous TIA, stroke, and MI.

Examination A clear demarcation between normal and ischaemic skin suggests an embolic cause of an acutely ischaemic limb. Look for sources of emboli (irregular pulse, abnormal heart sounds, murmurs, valve clicks). Check all pulses in both the affected and contralateral limbs. The presence of normal pulses in the contralateral limb suggests an embolic cause, whereas absent contralateral pulses makes thrombosis more likely.

Investigations ECG, CXR, U&E, CK, FBC, coagulation screen, ABG, urinalysis (for myoglobin), cross-match. Cardiac and/or abdominal USS may be required, and if thrombosis *in situ* is suspected, angiography is indicated.

Thrombotic Thrombosis may develop acutely around atheroma. Previous intermittent claudication/vascular impairment is likely. The other limb may also have chronic vascular insufficiency (muscle wasting, hair loss, ulceration).

Treatment

- Give appropriate pain relief (usually IV opioid).
- Correct hypovolaemia and other causes of low-flow states.
- Revascularization is required within 6hr to avoid permanent muscle necrosis (and subsequent need for amputation) and metabolic effects (such as rhabdomyolysis and renal failure). If the cause is embolic, embolectomy is required. If thrombotic, angiography will define the site and extent of the lesion—thrombolysis ± reconstructive surgery is then undertaken.

Complications of varicose veins

Bleeding from varicose veins

Patients with chronic venous hypertension associated with varicose veins have a significant risk of haemorrhage from the dilated thin-walled veins which commonly surround the area of lipodermatosclerosis at the ankle. Haemorrhages may be profuse and sufficient to cause hypovolaemic shock. In extreme cases, this may even cause death.

Treatment

Control bleeding by elevating the leg and applying a non-adherent dressing and pressing firmly. Follow with appropriate bandaging, unless there is evidence of occlusive arterial disease (varicose veins and arterial disease frequently coexist in the elderly). Some patients may require resuscitation with IV fluids.

Refer for admission those who were shocked at presentation, those who have subsequently bled through the bandaging, those with occlusive arterial disease, and those who live alone. All patients require surgical outpatient follow-up—advise patients who are discharged about first-aid measures in the event of a rebleed.

Superficial thrombophlebitis

This occurs in those with varicose veins or prothrombotic states (eg underlying inflammatory/malignant conditions). It usually produces redness, tenderness, and induration along the course of the involved vein.

Treatment

Bed rest, elevation, and analgesia (NSAID). Pain typically ↓ over 1–2 weeks, and the patient is left with a hard thrombotic cord. Superficial thrombophlebitis is only rarely associated with DVT, but occasionally thrombosis spreads from the long saphenous vein to involve the femoral vein. If there is any question of deep vein involvement, request an USS. If the thrombotic process involves the sapheno-femoral junction or the ilio-femoral system, refer for anticoagulation (see ➲ Patients on anticoagulants, pp. 178–9).

Venous ulcers

Venous (varicose) ulcers tend to be chronic and recurrent. They typically occur on the medial side of the ankle. There is often associated dermatitis with surrounding brown discoloration, thickening of the skin, and leg oedema. There is often mixed venous and arterial disease, especially in the elderly. Although ischaemic ulcers tend to lie on the lateral aspect of the ankle, exclude ischaemic ulceration by checking the peripheral pulses (request Doppler in patients with oedematous legs). Look for areas suspicious of malignant change, which may rarely occur in chronic ulcers (Marjolin's ulcer).

Treatment

Clean the ulcer with saline, and dress it with either paraffin gauze or colloidal dressing. Follow with firm bandaging (unless there is coexisting arterial disease). Advise leg elevation when resting. Avoid topical antibiotics and indiscriminate use of oral antibiotics. Prescribe oral antibiotics (eg flucloxacillin) only if there is cellulitis. Liaise with the GP about surgical outpatient follow-up and to arrange for redressing by the district nurse.

Ureteric colic

►► *New-onset flank/back pain in the elderly may represent a leaking aortic aneurysm (even if haematuria is present).*

Causes

Calculi or blood clots may cause ureteric (or 'renal') colic. Colicky pain is produced by ureteric obstruction, ↑ intraluminal pressure, and muscle spasm. Calculi most commonly consist of calcium oxalate and/or calcium phosphate. Less common are magnesium ammonium phosphate (associated with UTIs and urea-splitting organisms such as *Proteus*), urate, and cystine stones.

Calculi are associated with hypercalcaemia, hyperoxaluria, and hyperuricaemia. 'Staghorn' calculi in the collecting system predispose to infections.

Calculi may form throughout the length of the renal tract. They vary in size from tiny particles to large 'stones' in the bladder. They cause symptoms from local obstruction and infection, and rarely they may ulcerate through the wall of the structure in which they are present. Smaller stones (<5mm diameter) are likely to pass spontaneously.

Clinical features

The most common presenting symptoms are pain from obstruction or UTI and/or haematuria. Constant dull, severe loin discomfort is associated with excruciating colicky pain, spreading to the respective iliac fossa, testis, tip of the penis, or labia. The pain may cause the patient to move, roll, or walk about. Nausea, vomiting, pallor, and sweating are common. There is frequently a previous history of stone disease—ask about this and whether there is any past history of renal disease. Ask about urinary and GI symptoms.

Apart from loin tenderness, abdominal examination is usually normal, but carefully check the haemodynamic status, pulses, bruits, and the abdominal aorta, as a ruptured aortic aneurysm can present in a similar fashion (see ➲ Ruptured abdominal aortic aneurysm, pp. 536–7). Pyrexia or rigors suggest associated infection. Microscopic (sometimes frank) haematuria is very common but not always present. Symptoms are usually relieved when the stone passes into the bladder, but larger calculi may then cause obstruction at the bladder neck or the urethra, producing acute urinary retention. Bladder calculi may present with symptoms of UTI and/or bladder irritation (frequency, dysuria, strangury, and haematuria).

Investigations

- Urinalysis and MSU: blood on stix testing is present in >80% of patients with proven stones. A pH >7.6 implies an associated infection with urea-splitting organisms.
- U&E, creatinine, glucose, Ca^{2+}, PO_4^{2-}, urate, amylase.
- FBC—↑ WCC is associated with infection.
- The traditional 'KUB' (kidneys, ureters, bladder) X-ray with subsequent IVU has now been replaced by CT. Plain CT (without contrast) has a high sensitivity and specificity and has the advantage of assisting the diagnosis of other causes of abdominal and/or loin pain. In addition to establishing the size and position of stones, CT will also identify obstruction and hydronephrosis.
- USS has a role in the investigation of pregnant patients with suspected ureteric colic but is much less reliable at identifying smaller stones than CT scan.
- Take specialist advice when considering imaging for patients who present with problems which are related to recurrent/known stones.
- Send any stone which has passed and been collected for laboratory analysis.

Treatment

See NICE guidance at https://cks.nice.org.uk/renal-or-ureteric-colic-acute

Analgesia

- Give diclofenac 75mg IM, repeated after 30min if necessary. If NSAIDs are not appropriate (eg allergy or renal impairment), give IV opioid titrated to effect (eg morphine 5–10mg).
- Consider adding IV paracetamol (1g).
- Provide an antiemetic (eg metoclopramide 10mg IM or IV).

Admission criteria

- Shock, fever, or other signs of systemic infection.
- ↑ risk of AKI (eg CKD, solitary kidney).
- Pregnancy.
- Dehydration and inability to take oral fluids due to vomiting.
- Uncertainty about the diagnosis.
- Continuing or early recurrence of severe pain.

Discharge advice

Aim to discharge patients (with arrangements for appropriate outpatient investigation) when symptoms have resolved or dramatically improved and in whom CT/IVU shows no obstruction. Provide NSAID analgesia (eg naproxen or diclofenac) plus antiemetic (eg metoclopramide). Advise the patient to maintain a normal oral fluid intake, with the aim of maintaining a normal urine colour. Explain that the stone may pass spontaneously—ideally, ask the patient to sieve the urine (eg using a tea strainer or nylon stocking) and retrieve the stone, so that its content can be analysed in the laboratory. Ask the patient to return to hospital if they develop severe pain, fever, or rigors. Ensure that the GP is informed in a timely fashion, so that further follow-up can be arranged as an outpatient.

Retention of urine

Causes of acute urinary retention

The common causes are shown in Table 10.4—men are affected more than women, most usually due to benign prostatic hyperplasia.

Table 10.4 Common causes of urinary retention

Men	Women
• Prostatic hyperplasia/cancer	• Retroverted gravid uterus
• Urethral stricture	• Atrophic urethritis
• Postoperative	• MS

Other causes include: acute urethritis, prostatitis, phimosis, urethral trauma, bladder blood clot, urethral calculus, prolapsed intervertebral disc/spinal cord syndromes, drugs (alcohol, antihistamines, anticholinergics, antihypertensives, tricyclics), faecal impaction, and anal pain.

Presentation

The diagnosis is usually obvious—the patient complains of inability to pass urine, combined with bladder discomfort. Remember, however, to consider the diagnosis in those patients unable to describe their symptoms (eg those unconscious after trauma).

Examination reveals a tender, enlarged bladder, with dullness to percussion well above the symphysis pubis. Search for the causes listed above. In particular, look for evidence of prolapsed disc/cord compression by checking lower limb power/reflexes and perineal sensation. Perform PR examination to assess anal tone and the prostate.

Initial management

Provided there is no contraindication (eg trauma or urethral stenosis), decompress the bladder by urethral catheterization. Use an aseptic technique. If urethral catheterization is impossible or contraindicated, consider a suprapubic catheter, but this requires a doctor experienced in the technique.

After bladder drainage, record the volume of urine obtained, then re-examine the abdomen for pathology that might have been previously masked. Test the urine and send an MSU for culture and sensitivity.

Check U&E and FBC. (Prostate-specific antigen is unreliable in retention.)

Further management

If blood tests are normal and there is no complication (eg bleeding) after urinary catheterization, discharge the middle-aged/elderly man with likely underlying benign prostatic hyperplasia, with plans to return to a Trial WithOut Catheter (TWOC) clinic, typically a week later.

Take specialist advice on starting an α-blocker (eg tamsulosin 400mcg daily), as this may ↑ the chance of successful removal of the catheter, although there are significant side effects and drug interactions.

Penile problems and prostatitis

Paraphimosis

Paraphimosis occurs when the foreskin is left retracted, thereby causing swelling of the glans, which results in difficulty replacing the foreskin to its proper position. Untreated, tissue necrosis may develop. Paraphimosis may be iatrogenic, occurring after urethral catheterization.

Treatment Initially attempt reduction by manual decompression, which may require the use of Entonox®, IV sedation, or LA (a small amount of topical 1–2% lidocaine gel or injection of 10mL of plain 1% lidocaine around the base of the penis). Digital pressure may allow the glans to ↓ in size, prior to the foreskin being delivered back into its usual position. If unsuccessful, refer to the surgical team for reduction under GA or dorsal slit of the prepuce followed by later circumcision.

Priapism

Persistent (and usually painful) penile erection has a number of causes:

- Iatrogenic (following intra-cavernosal injection of one or more of: papaverine, alprostadil, vasoactive intestinal polypeptide, and phentolamine for impotence).
- Others: leukaemia, myeloma, sickle-cell disease, spinal injury, drugs [eg sildenafil (Viagra®), phenothiazines, cannabis, cocaine), renal dialysis.

Management Priapism is a urological emergency. Refer urgently to the urology team. Initial emergency treatment of a prolonged (>6hr) artificial erection (ie following an intra-cavernosal drug injection or oral sildenafil) is to aspirate 50mL of blood from each corpus cavernosum through a 19G butterfly needle into a 50mL syringe with a Luer lock.

Urethritis

This usually presents with dysuria/urinary frequency, reflecting underlying STI. Refer for appropriate investigation, treatment, and follow-up.

Prostatitis

Inflammation of the prostate may be acute or chronic and presents in a variety of ways (fever, urgency, frequency, perineal pain, urethral discharge). PR examination reveals a tender prostate. Urinalysis demonstrates protein. Refer for further investigation and treatment.

Penile trauma

Minor superficial tears Relatively common. Most involve the frenulum. Patients report pain and bleeding following sexual intercourse. Bleeding usually responds to local pressure (if not successful, consider tissue glue or refer to the surgical team). Once bleeding has stopped, advise abstinence from sexual activity for ~10 days to allow healing to occur and prevent recurrence.

Fracture of the penis This occurs infrequently. It involves injury to the tunica albuginea of the erect penis. The result is penile tenderness and swelling. Refer to the urologist for urgent surgical exploration, evacuation of haematoma, and repair.

Testicular problems

Any pain of testicular origin may be initially referred to the abdomen.

Testicular torsion

Testicular torsion is most frequently seen in children and young adults. Any suspicion of testicular torsion should prompt immediate referral. The condition is covered fully in ➲ Inguinal and scrotal swellings, pp. 722–3.

Acute epididymitis

Causes For those aged <35y, infection with *Chlamydia* or gonococcus is commonly responsible. Acute epididymitis in those aged >35y is usually secondary to UTI and associated with underlying urinary tract pathology.

Clinical features There is typically a gradual onset of progressive testicular ache, with subsequent swelling of the epididymis and testis. There may be a history of dysuria or urethral discharge. The patient may be pyrexial. The epididymis is acutely tender, with the testis lying low in the scrotum. Advanced late cases may have progressed to abscess formation.

Investigations Send an MSU. Leave taking urethral swabs to the GU clinic.

Management The chief initial concern is to ensure that testicular torsion is not being missed—if there is any possibility of this (see ➲ Inguinal and scrotal swellings, pp. 722–3), refer urgently. Treatment of acute epididymitis comprises antibiotics (eg ciprofloxacin for 2 weeks), analgesia, and rest. Some patients require admission; others may be managed on an outpatient basis. Urology investigation and follow-up will be required, so involve the urologist early. For patients with suspected *Chlamydia* or gonococcus infection, refer to the GU clinic for appropriate advice, swabs, treatment, and contact tracing of sexual partners.

Orchitis

Orchitis may present as epididymo-orchitis, an extension of bacterial epididymitis (see ➲ Acute epididymitis, p. 544). Orchitis of viral origin may also occur—typically mumps, following ~5 days after parotitis. Mumps orchitis may be unilateral or bilateral and can occur in the absence of overt parotitis. Rarely, orchitis is secondary to TB or syphilis.

Treatment All patients with orchitis require analgesia and follow-up. If there is any possibility of bacterial infection, antibiotics are indicated (see ➲ Acute epididymitis, p. 544).

Scrotal/testicular lumps

Causes include: hydrocele, inguinal hernia, epididymal cyst, epididymitis, orchitis, and testicular tumour. Many patients require referral back to the GP or clinic. Be wary of an apparent epididymo-orchitis which failed to respond to antibiotics—it could be an atypical presenting testicular tumour.

Testicular trauma

See ➲ Scrotal, and testicular trauma, p. 361.

Cellulitis and erysipelas

Cellulitis

(See ➲ Infected wounds and cellulitis, p. 419.)

Cellulitis reflects bacterial skin infection (usually streptococcal, occasionally staphylococcal). It can occur in association with a skin wound acting as a portal of entry for infection (eg athlete's foot), but it may also occur without any obvious breach in the skin. Ascertain whether or not there is evidence of systemic upset or any background problems such as immunodeficiency, diabetes, or steroid therapy.

The area of affected skin is red and warm to touch, with poorly defined margins. Check T° and look for lymphangitis and/or lymphadenopathy.

Mark the limits of the cellulitis with a permanent marker pen to enable future judgements on improvement/deterioration.

Treatment

Treatment depends upon the nature and extent of clinical findings, as follows (see https://cks.nice.org.uk):

- Treat patients who have localized limb infection and no evidence of systemic upset with oral antibiotics (either flucloxacillin ± phenoxymethylpenicillin or co-amoxiclav or clarithromycin), and arrange follow-up in 36–48hr. Advise patients to return sooner for review if symptoms significantly worsen.
- Admit patients who are systemically unwell or have spreading infection (eg lymphangitis extending above the knee from an area of cellulitis on the foot). Obtain venous access; take blood cultures and start IV antibiotics (either benzylpenicillin + flucloxacillin or co-amoxiclav).
- Consider admission for IV antibiotics for patients who are systemically well but have a significant comorbidity (eg chronic venous insufficiency, morbid obesity, peripheral arterial disease). Note, however, that where systems exist to support it, some of these patients ('Eron class II') may be safely managed in the community by IV antibiotics and regular review.
- Admit patients with cellulitis of the face (particularly around the eye), unless very minor. They are at risk of significant intracranial complications (notably cavernous sinus thrombosis)—start IV antibiotics, and refer for admission to the ophthalmology team.

Erysipelas

This streptococcal infection is limited to the more superficial parts of the skin, resulting in an area of redness and heat, with clearly defined margins. Treat with antibiotics, as outlined for cellulitis above, except that phenoxymethylpenicillin alone (500mg PO qds for 7 days) suffices in most cases.

Necrotizing fasciitis

(See ➲ Streptococcal infections, p. 244.)

Abscesses

An abscess is a localized collection of pus, resulting in a painful soft tissue mass that is often fluctuant, but surrounded by firm granulation tissue and erythema. The cause is usually bacterial, resulting from minor trauma to the epithelium/mucosa or blockage of apocrine glands. A history of a previous lump at the site suggests infection of a sebaceous cyst. Check BMG in all patients.

For patients with recurrent abscesses, check for signs of hidradenitis suppurativa, diabetes, inflammatory bowel disease, and malignancy. Ask about steroid use.

Treatment

Incision and drainage A general surgical principle is that a collection of pus requires drainage. On occasions, depending upon local policy, it may be appropriate to do this in the ED. Some abscesses (eg face, breast, perineum, paediatric) require specialist attention. Regional, parenteral, or general anaesthesia may be needed to supplement LA, which works poorly in this situation.

Technique Incise along the length of the fluctuance and deep enough to enter the cavity. An elliptical incision will prevent premature closure and re-accumulation of pus. Send pus for culture. Ensure that loculi in the cavity are gently broken by the use of a curette. Consider inserting a loose antiseptic wick in the cavity to ensure drainage and prevent premature closure.

Antibiotics Not indicated in patients with normal host defences as long as the abscess is localized. Evidence of surrounding or spreading infection may warrant antibiotics (eg co-amoxiclav or penicillin + flucloxacillin) and, on occasions, admission (see below).

Refer the following

- Those who are systemically unwell (pyrexia, tachycardia, rigors), the immunocompromised, and those not responding to treatment.
- Abscesses secondary to IV drug misuse.
- Those with infection in certain anatomical sites: face (↑ risk of cavernous sinus thrombosis), those potentially involving the airway (sublingual abscesses, Ludwig's angina), and axillary, groin, retropharyngeal, perineal, and breast abscesses.
- Those with extensive or progressing cellulitis/lymphangitis.

These patients may require IV antibiotics (eg flucloxacillin + penicillin or co-amoxiclav IV), analgesia, and surgical drainage. Take blood for FBC, clotting studies, and blood culture. Treat sepsis/septic shock where necessary (see ➲ Sepsis, pp. 62–3 and ➲ Shock, pp. 64–5).

Breast infection

Lactational breast abscess These are usually peripherally located and due to *Staphylococcus aureus*. Local discomfort proceeds to painful swelling. Overlying skin may be red. Extreme cases may undergo necrosis and spontaneous discharge.

Technique If seen prior to frank abscess formation, consider antibiotic treatment alone—prescribe a penicillinase-resistant antibiotic. If there is any suspicion of an abscess, refer for needle aspiration—if pus is found, drainage will be needed. Encourage the infant to feed from the contralateral breast whilst the affected side is emptied of milk manually or by a breast pump.

Non-lactational breast abscess Typically affects the 30–60y age group, usually peri-areolar, recurrent, and related to duct ectasia/periductal mastitis. Refer for needle aspiration, culture, and antibiotics (metronidazole and flucloxacillin). Note that inflammatory breast cancer may mimic septic mastitis and breast abscess. Incision of neoplastic lesions may have disastrous results.

Perineal abscesses

(See ➲ Anorectal problems, pp. 534–5.)

Complications after surgery

All surgical procedures carry risks of complications. The most common and serious risks of a specific procedure are discussed with patients prior to the operation, as part of obtaining informed consent. Many patients experiencing problems soon after surgery will get in touch directly with the surgical team that performed the surgery or the ward staff that looked after them. However, many patients simply attend (or are advised to attend) the ED (sometimes at a different hospital), especially out of hours.

Approach

- Consider surgical complication as the cause of symptoms in any patient presenting within a few days of surgery.
- Try to establish exactly what surgery the patient has undergone.
- Remember that, irrespective of the nature of the operation, most surgeons will be very keen to be informed if a patient on whom they have operated develops a complication.
- Aim to involve the surgical team (or at least inform them) when treating a surgical complication.

Specific surgical complications

Haemorrhage relating to skin wounds

Apply pressure to a skin wound which is bleeding externally, and consider whether a further suture is needed and/or surgical referral. Haemorrhage developing deep to skin wounds can result in significant haematomas—manage according to the site, size, and situation. For example, significant haemorrhage relating to recent plastic surgery (eg 'facelift') may jeopardize the result, requiring urgent surgical intervention.

Internal haemorrhage

This may not be easy to identify—adopt a high index of suspicion.

Infection

Wound infection is usually apparent by localized pain, redness, and tenderness. Deeper infection may present in an atypical fashion—if there is any suggestion of this, check T°, WCC, and CRP and refer to the surgical team. Consider the possibility of intra-abdominal infection/collection in patients who have undergone recent laparotomy/laparoscopy.

Wound dehiscence

This may be superficial or deep and is often associated with infection. Cover any exposed viscera with saline-soaked swabs, and refer to the surgical team.

Misplaced gastrostomy tubes

(See ➲ Gastrostomy tube problems, p. 129.)

Inadvertent ('accidental') removal of percutaneous endoscopic gastrostomy (PEG) tubes requires a replacement tube to be inserted as soon as possible, so that the track does not close off. A temporary tube (eg lubricated urinary catheter) inserted gently will serve this purpose until a definitive replacement may be inserted. Refer to the relevant surgical/radiological interventionalist.

Chapter 11

Ophthalmology

Approach to eye problems

History

Always take a full ophthalmic history. Which eye is affected (are both)? What is the disturbance? Are there flashing lights or floaters? How quickly did the symptoms come on? How does it affect the patient's lifestyle (job, reading, watching TV)? Ask about prior ophthalmic/optician treatment, and take a full medical and drug history. Family history of glaucoma may also be relevant.

Always measure visual acuity in anyone presenting with an eye problem. Pointers to potentially serious pathology include those with:

- Sudden visual loss.
- Significantly ↓ VA.
- Penetrating eye injuries.
- Chemical burns of the eye (these require immediate treatment and specialist referral).

Have a low threshold for involving an ophthalmologist if a patient who is already blind in one eye presents with a problem with the 'good eye'.

Examination

Visual acuity This is the key to eye examination—measure this first.

Failure to document VA may constitute negligence.

Use a Snellen chart, and read at 6m for each eye separately. Allow patients to use glasses, if available; if not, employ a pinhole (made using a needle through a piece of card). Use of a pinhole eliminates refractive error.

VA is expressed as:

- *Distance from the chart in metres/number of lines on the chart (normal vision is 6/6),* eg a patient whose VA is recorded as 'Right eye 6/5; left eye 6/60' can read the bottom line with the right eye, but only the top line with the left eye. If patients read additional letters of the line below, record using + number of extra letters (eg 6/12 + 2).
- Bring patients unable to read the chart at 6m forward until they can read the chart (eg 3/60 = top line read at 3m). Very poor vision: try counting fingers or detecting hand movement at 1m, or light perception.
- A hand-held chart at 30cm is an alternative if a full Snellen chart is unavailable—ability to read small print implies normal VA for that eye. For patients who are illiterate, there is an alternative chart with various different versions of the letter 'E'—ask the patient to state in which directions the three limbs of the letter point.

Pupils Record pupil size, shape, and direct and consensual responses to light and accommodation.

Eye movements Check full range and for diplopia. Look for nystagmus.

Visual fields Check carefully in patients with visual loss.

Fundoscopy In a darkened room, first check for the red reflex. A lost or ↓ red reflex is abnormal, typically caused by vitreous haemorrhage, cataracts, or major corneal abrasions. Assess the optic discs and look for retinal haemorrhages and vessel abnormalities. Sometimes, there may be a poor view—leave the use of drops to dilate the pupil (eg tropicamide) to the ophthalmologist.

Direct assessment Under a bright light, look for inflammation or FBs.

Subtarsal examination If there is a possibility of an FB, evert the upper eyelid by pressing down lightly over the upper lid with a cotton bud or orange stick and rotating the lid upwards over it. Ask the patient to look down throughout.

Slit lamp examination Learn how to use a slit lamp. It allows a detailed view of the conjunctiva, cornea, and anterior chamber. Fluorescein staining reveals corneal abnormalities, particularly when viewed under blue light when abrasions appear yellow/green. Fluorescein is available either in drop form or dried onto a strip. Remember that fluorescein can permanently stain clothes and contact lenses.

Intra-ocular pressure Digital assessment is unreliable. Formal measurement of intra-ocular pressure is useful but requires training and is left to the eye specialist in many departments.

Temporal arteries Palpate for tenderness if temporal arteritis is a possibility.

LA drops to aid examination

Sometimes, blepharospasm prevents satisfactory examination. Consider LA drops (one or two drops of 1% amethocaine/tetracaine or 0.4% oxybuprocaine; 0.5% proxymetacaine causes less stinging and is useful in children). *Never discharge patients with a supply of LA drops.*

Notes on ophthalmological treatments

Antibiotic ointment and drops Apply to the lower fornix (between the lower eyelid and the sclera), then ask the patient to keep the eye shut for 1–2min. Ointment has the advantage over drops in that it lasts longer, eg chloramphenicol ointment needs to be given four times a day, whereas drops need to be given every 2hr initially. Theoretical concerns about aplastic anaemia are not well founded (see *BNF*).

Eye pads These were previously recommended following the administration of LA drops and for patients with corneal abrasions; they tend not to be useful.

Driving Advise patients not to drive until their vision has returned to normal (this particularly applies after the use of mydriatic agents). In addition, advise patients not to drive whilst wearing an eye pad. Document the advice given in the notes.

Blunt eye injury

Blunt injury to the face may result in injury to the orbit or its bony margins. Compression of the eye in an antero-posterior direction (eg squash ball or fist) can cause a *'blow-out' fracture* of the floor of the orbit.

Retrobulbar haematoma

This may lead to orbital compartment syndrome and blindness. Unless diagnosed and treated as an emergency, optic nerve ischaemia develops and the patient can lose sight in the affected eye within a few hours. Proptosis, reduced eye movements, reduced VA, and pain all point to a retrobulbar haematoma. There may be an afferent pupillary defect.

Assessment

Swelling around the eye can make assessment difficult, and this situation may worsen as swelling ↑, so try to examine the eye at the earliest opportunity.

- Look for proptosis.
- Check VA.
- Check pupillary reflexes.
- Check for enophthalmos and ↓ infra-orbital nerve sensation, both found in a blow-out fracture.
- Document the range of eye movements, looking, in particular, for entrapment of the extra-ocular muscles.
- Look for a hyphaema (a horizontal fluid level in the anterior chamber when the patient is upright). It can cause pain, photophobia, and blurred vision and can ↑ the intra-ocular pressure, causing nausea and vomiting.
- Stain the cornea and examine using the slit lamp for corneal abrasions.
- Ophthalmoscopic examination may reveal lens dislocation, hyphaema, and vitreous, subhyaloid, or retinal haemorrhage. Sometimes retinal oedema ('commotio retinae') may be seen as white patches with diffuse margins on the posterior pole of the eye.

X-ray if there is bony tenderness or clinical evidence of orbital or facial bone fracture.

Treatment

Any patient suspected of a retrobulbar haematoma requires an emergency lateral canthotomy and cantholysis. This should be performed by an ophthalmologist or a trained emergency physician, under LA in the ED, and it reduces the retro-orbital pressure.

Nurse patients with an obvious globe injury head up at 45°. Refer urgently. Consider prophylactic oral antibiotics (eg co-amoxiclav) for uncomplicated facial or orbital fractures, according to local policy, and arrange for maxillofacial follow-up, with advice to avoid nose-blowing in the meantime.

Penetrating eye injury

Suspect an *intra-ocular FB* if there is a history of hammering or work involving metal on metal. Establish if protective glasses were worn—remember that standard glasses (without a seal to the skin) do not provide full protection against an FB. Ascertain whether a small FB travelling at speed may have penetrated the orbit (eg during grinding, hammering, chiselling). Failure to suspect and diagnose these injuries can have serious consequences.

Assessment

- Check VA.
- Look for pupil irregularity.
- Look for puncture/entry wounds on both aspects of the eyelids, the cornea, and the sclera. Corneoscleral wounds are often situated inferiorly, due to upturning of the eyeball as the patient blinks.
- Examine the anterior chamber. There may be a shallow anterior chamber, air bubbles, a flat cornea, a deflated globe, and a positive Seidel's test (dilution of fluorescein by aqueous humour leaking from the anterior chamber).
- Look for a hyphaema.
- Look for vitreous haemorrhage on fundoscopy.

X-ray all patients with possible globe penetration (consider also CT or USS).

Give analgesia, tetanus prophylaxis, and IV antibiotics (eg cefuroxime 1.5g), and refer all patients with penetrating eye injuries immediately to an ophthalmologist, even if there are other major injuries needing attention at the same time.

Do not manipulate or try to remove embedded objects (eg darts).

Eyelid wounds

Lacerations (and incised wounds) to the eyelids may be a pointer to the presence of associated (more significant) globe damage. Most eyelid wounds can be closed under LA using small (6/0 or 7/0) interrupted nylon sutures—in most instances, this is more appropriately undertaken by a specialist.

Corneal trauma

Conjunctival FB

The typical history is of dust or grit blown into an eye by the wind. The FB usually gravitates into the lower fornix—remove it with a cotton bud, then check for associated subtarsal/corneal FBs.

Subtarsal FB

FBs may not gravitate into the lower fornix but may remain stuck under the upper eyelid. The patient reports pain on blinking. Fluorescein staining reveals characteristic vertical corneal abrasions (the cornea has been likened to an 'ice rink'). Evert the upper eyelid and remove the FB with a cotton bud. Discharge with topical antibiotic (eg chloramphenicol ointment qds or fusidic acid eye drops).

Corneal abrasions

These often result from a newspaper or fingernail in the eye. Irritation, photophobia, and lacrimation occur. Use LA drops and fluorescein staining to examine the cornea. Exclude FB or penetrating injury. Prescribe regular antibiotic ointment (eg chloramphenicol) and oral analgesia. Only consider an eye patch if the abrasion is very large (>1cm diameter). If the patient is very uncomfortable, instilling a drop of 1% cyclopentolate to dilate the pupil (this reduces iris spasm) or a drop of 0.1% diclofenac may help. Advise the patient not to drive until vision has returned to normal. Advise also to return for review if symptoms continue beyond 36hr.

Corneal FB

Instill LA and attempt removal with a cotton bud (moistening it and rolling it over the FB to pick it up may help). If unsuccessful, remove with a blue (23G) needle introduced from the side (ideally using a slit lamp). Ensure that the patient's head is firmly fixed and cannot move forward onto the needle—it can also help to attach the needle to a syringe and for the operator's hand to rest lightly on the patient's cheek to help to keep it steady. After complete removal of the FB, check that the anterior chamber is intact; instill and prescribe antibiotic ointment, and advise the patient to return if symptomatic at 36hr. Refer patients with large, deep, or incompletely removed FB or if a rust ring remains afterwards.

Arc (welder's) eye/'snow blindness'

Exposure to ultraviolet light can cause superficial keratitis. Climbers/skiers, welders, and sunbed users who have not used appropriate protective goggles develop pain, watering, redness, and blepharospasm several hours later. LA drops allow examination with fluorescein staining, revealing multiple punctate corneal lesions. Consider instilling a drop of 1% cyclopentolate or 0.1% diclofenac into both eyes. Discharge with an eye pad, oral analgesia, and advice not to drive until recovered. Anticipate resolution within 24hr. Do not discharge with LA drops.

Chemical eye burns

Chemical burns from alkali or acid are very serious. Triage urgently ahead; check TOXBASE® (https://www.toxbase.org), and irrigate the eye immediately with lukewarm normal saline for at least 20min or until the pH of tears has returned to normal (~7.4). A 1L bag of 0.9% saline with standard IV tubing is ideal. LA may be needed to enable full irrigation. Consider the need for protective clothing during irrigation. Try to identify the substance involved, and contact the Poisons Unit. Refer alkali and acid burns immediately to the ophthalmologist.

Superglued eyelids

Wash with warm water. The eye will open within 4 days. If the patient reports an FB sensation, this may represent a lump of glue, which may cause an abrasion if untreated—refer to the ophthalmologist.

Note that despite precautions, occasionally eyelids are glued together during the application of tissue glue to close a forehead wound (see Skin tissue glue, p. 415). If the eyelids remain closed despite simple measures, contact the ophthalmologist ± TOXBASE® (https://www.toxbase.org).

Contact lens problems

Contact lenses may be 'soft' (more comfortable) or 'hard'. Avoid using fluorescein with contact lenses, as permanent staining may occur.

'Stuck lens'

Most contact lens users are adept at removing their lenses. New users, however, can experience difficulty. Moisten soft lenses with saline, then remove by pinching between the finger and the thumb. Special suction devices are available to help remove hard lenses.

'Lost lens'

Patients may present concerned that they are unable to find their contact lens and cannot remember it falling out. Check under both eyelids carefully (evert the upper lid if the lens is not immediately apparent) and remove the offending lens, if present.

Hypersensitivity and overuse

Preservatives in lens-cleaning fluid cause itching and may evoke a reaction. Advise to stop using the lenses; give antibiotic ointment, and arrange ophthalmological follow-up.

Acanthamoeba keratitis

This protozoal infection of the cornea occurs in contact lens users, associated with poor lens hygiene or swimming whilst wearing contact lenses. The eye becomes painful and red. Corneal oedema and ulceration develop. If suspected, refer immediately for ophthalmological care.

Other problems related to contact lenses

Treat and refer conjunctivitis, corneal abrasions, or ulcers apparently related to contact lenses, as outlined in The red eye, pp. 558–9. Advise avoidance of use of both contact lenses until the problem has resolved, and arrange appropriate follow-up with the GP or an ophthalmologist.

Sudden visual loss

Sudden visual loss requires emergency assessment and treatment.

Amaurosis fugax

The patient describes temporary loss of vision in one eye, like a 'curtain coming down', with complete recovery after a few seconds to minutes. The cause is usually a thrombotic embolus in the retinal, ophthalmic, or ciliary artery, originating from a carotid atheromatous plaque, but it can also be a feature of giant cell arteritis (see ➲ Giant cell (temporal) arteritis, p. 557). Treat as for TIA (see ➲ Transient ischaemic attacks, p. 155), and involve the ophthalmology team (to exclude other 'eye' pathology).

Central retinal artery occlusion

The central retinal artery is an end artery. Occlusion causes an ischaemic stroke of the retina. It is usually embolic (check for AF and listen for carotid bruits), causing sudden painless ↓ VA to counting fingers or no light perception. The patient may have a history of amaurosis fugax. Direct pupil reaction is sluggish or absent in the affected eye, but it reacts to consensual stimulation (afferent pupillary defect). Fundoscopy reveals a pale retina, with a swollen pale optic disc and a 'cherry red macula spot' (the retina is thinnest here and the underlying choroidal circulation is normal). Retinal blood vessels are attenuated and irregular—there may be 'cattle-trucking' in arteries.

Treat by digitally massaging the globe (with the eye closed) for 5–15s, then release and repeat to dislodge the embolus, whilst awaiting urgent arrival of an ophthalmologist.

If there is any delay in the patient being seen by the ophthalmologist, discuss other options such as giving acetazolamide 500mg IV (to ↓ intra-ocular pressure and ↑ retinal blood flow). Note that current evidence does not support the use of thrombolytic agents in this situation.

Do reconsider the diagnosis. In particular, consider whether or not giant cell (temporal) arteritis (see ➲ Giant cell (temporal) arteritis, p. 557) is a possibility—ask about jaw claudication, headaches, and scalp tenderness.

Central retinal vein occlusion

This is a more frequent cause of sudden painless visual loss than arterial occlusion. Predisposing factors include: old age, chronic glaucoma, arteriosclerosis, hypertension, and polycythaemia. Examination reveals ↓ VA, often with an afferent pupillary defect. Fundoscopy reveals a 'stormy sunset' appearance—hyperaemia with engorged veins and adjacent flame-shaped haemorrhages. The disc may be obscured by haemorrhages and oedema. Cotton wool spots may be seen. Although the outcome is variable and there is currently no specific treatment, refer urgently as the underlying cause may be treatable, thus protecting the other eye.

Giant cell (temporal) arteritis

Inflammation of the posterior ciliary arteries causes ischaemic optic neuritis and visual loss. It is relatively common in those aged >50y and is associated with polymyalgia rheumatica. The other eye remains at risk until treatment is commenced. Rapid and profound visual loss may be preceded by headaches, jaw claudication, general malaise, and muscular pains—often these symptoms can worsen over several weeks or months. The temporal arteries are characteristically tender to palpation. Retinal appearances have been termed 'pale papilloedema'—the ischaemic disc is pale, waxy, and elevated and has splinter haemorrhages on it. If suspected, give oral prednisolone 60mg immediately; check ESR (typically >>40mm/hr but can be normal), and refer urgently.

Vitreous haemorrhage

This occurs in diabetics with new vessel formation and in bleeding disorders and retinal detachment. Small bleeds may produce vitreous floaters with little visual loss. Large bleeds result in painless ↓↓ VA, an absent red reflex, and difficulty visualizing the retina. Refer urgently. Meanwhile, elevate the head of the bed to allow blood to collect inferiorly.

Retinal detachment

This occurs in myopes, diabetics, and the elderly and following trauma. The rate of onset is variable—patients may report premonitory flashing lights or a 'snow storm', before developing cloudy vision. There may be a visual field defect. Macular involvement causes ↓ VA. The affected retina is dark and opalescent but may be difficult to visualize by standard ophthalmoscopy. Refer urgently for surgery and surgical reattachment or retinal laser photocoagulation.

Optic neuritis

This usually presents in a young woman. Optic nerve inflammation and demyelination cause visual loss over a few days. Pain on eye movement may occur. An afferent pupillary defect is associated with ↓ VA, ↓ colour vision (the colour red looks faded), and a normal/swollen optic disc. Most recover untreated; later some develop MS. Refer to the ophthalmologist (possibly to discuss steroid treatment—currently controversial and not proven).

Other causes

Patients with chronic visual loss due to a variety of conditions may present acutely (senile macular degeneration, glaucoma, optic atrophy, cataract, choroidoretinitis). Drugs which can cause painless visual loss include methanol (see Methanol poisoning, p. 211) and quinine (in overdose). Refer immediately all patients in whom an acute visual loss cannot be excluded.

The red eye

Refer all patients with new findings of ↓ VA, abnormal pupil reactions, or corneal abnormalities.

Orbital and preseptal cellulitis

(See ➲ Cellulitis, p. 545.)

This is a major infection of the orbital tissues. The infection most frequently spreads from the paranasal sinuses (ethmoid sinusitis), facial skin, or lacrimal sac. Occasionally, the infection follows direct trauma to the orbit or from haematogenous spread. Patients present with fever, eyelid swelling, erythema, and proptosis. Assess for signs of severe sepsis (see ➲ Sepsis, pp. 62–3 and ➲ Shock, pp. 64–5), and resuscitate as necessary. Obtain venous access; take blood for cultures, and commence IV antibiotics (eg co-amoxiclav) and fluids. Refer urgently to the ophthalmologist. Some aggressive infections may require surgical treatment. Cavernous sinus thrombosis and meningitis are potential complications.

Acute iritis (acute uveitis)

A relapsing condition of the young/middle-aged, associated with ankylosing spondylitis, ulcerative colitis, sarcoid, AIDS, and Behçet's syndrome.

Symptoms Include acute-onset pain, photophobia, 'floaters', blurred vision, and watering.

Signs ↓ VA, tender eye felt through the upper eyelid, circumcorneal erythema, small pupil (may be irregular due to previous adhesions). Shining a light into the 'good' eye causes pain in the other. Pain ↑ as the eyes converge and the pupils react to accommodation (Talbot's test). Slit lamp examination may reveal hypopyon and white precipitates on the posterior cornea.

Refer Refer urgently to the ophthalmologist for steroid eye drops, pupil dilatation, analgesia, investigation, and follow-up.

Acute closed angle glaucoma

Long-sighted middle-aged or elderly individuals with shallow anterior chambers are at risk. Sudden blocked drainage of aqueous humour into the canal of Schlemm causes intra-ocular pressure to ↑ from 10–20mmHg up to 70mmHg. This may be caused by anticholinergic drugs or pupil dilatation at night (reading in dim light).

Symptoms Include preceding episodes of blurred vision or haloes around lights due to corneal oedema. Acute blockage causes severe eye pain and nausea/vomiting.

Signs ↓ VA, hazy and oedematous cornea with circumcorneal erythema, and a fixed semi-dilated, ovoid pupil. The eye feels tender and hard through the upper eyelid. Measure the intra-ocular pressure if this facility is available.

Treatment Instill a 4% pilocarpine drop every 15min to produce ciliary muscle contraction and aqueous humour drainage. Apply prophylactic 1% pilocarpine drops into the other eye also. Give analgesia (eg morphine IV with antiemetic). Arrange an emergency ophthalmology opinion—consider giving acetazolamide 500mg IV (to ↓ intra-ocular pressure) in the meantime and/or mannitol 20% up to 500mL IVI over 1hr.

Conjunctivitis

This is caused by bacteria (*Streptococcus pneumoniae* or *Haemophilus influenzae*), viruses (adenovirus), or allergy. The sensation of FB may involve both eyes. The conjunctiva is red and inflamed, sometimes with eyelid swelling. VA and pupils are normal. Bacterial infection classically produces sticky mucopurulent tears, and viral infection copious watery tears (associated with photophobia and pre-auricular lymphadenopathy in the highly contagious adenoviral 'epidemic keratoconjunctivitis'). It is not possible to clinically distinguish viral from bacterial cases.

Advise not to share towels or pillows. Most cases settle relatively quickly with symptomatic measures—advise patients to see the GP if not better in 4 days or to return if significantly worse. Reserve a course of antibiotic eye drops or ointment (eg fusidic acid, chloramphenicol, or gentamicin) for patients whose symptoms last >5 days.

Ulcerative keratitis

Corneal ulceration causes pain with photophobia. It is apparent on fluorescein staining under a slit lamp.

- Hypopyon (pus in the anterior chamber) implies bacterial infection.
- Vesicles in the ophthalmic division of the trigeminal nerve occur with herpes zoster infection.
- Dendritic branching ulcers suggest herpes simplex. If misdiagnosed and steroid eye drops are given, ulceration can be disastrous.

Whatever the infective agent, refer corneal ulceration cases immediately.

Episcleritis

Inflammation beneath one area of the conjunctiva is usually associated with a nodule and a dull aching discomfort. VA, pupils, and the anterior chamber are normal. Prescribe oral NSAIDs, and advise GP review ± outpatient follow-up to consider steroid eye drops if there is no resolution.

Blepharitis

This chronic problem is quite common. Eyelashes are matted together and itchy. Ensure that there is no associated corneal ulceration; provide topical antibiotics (eg chloramphenicol), and refer for GP follow-up.

External hordeolum (stye)

Treat staphylococcal infections of eyelash roots with warm compresses.

Internal hordeolum (chalazion)

A chalazion is an inflammatory reaction in a blocked meibomian (tarsal) gland, which may get secondarily infected. Treat infected tarsal glands with topical antibiotics (eg chloramphenicol) plus oral antibiotics (eg co-amoxiclav). Refer patients who develop an abscess or nodule affecting vision.

Dacrocystitis (lacrimal sac infection)

This may follow nasolacrimal duct obstruction. Treat early infection with oral antibiotics (co-amoxiclav); later, refer for drainage.

Subconjunctival haemorrhage

This usually presents as a painless, well-defined area of haemorrhage over the sclera. It may result from vomiting or sneezing. Following trauma, consider orbital or base of skull fracture, and treat accordingly. Reassure the patient that subconjunctival haemorrhage will resolve in time.

Pupillary abnormalities

Pupillary examination may yield valuable information (see Table 11.1).

Table 11.1 Syndromes relating to examination of pupils

	Description	Common causes
Abducens nerve palsy	Diplopia, inability to look outwards	Trauma, tumour, Wernicke's syndrome
Adie pupil	Pupil is dilated and responds abnormally slowly to light	Commonly part of Holmes-Adie syndrome—cause unclear
Anisocoria	Unequal pupils	May be physiological or reflect disease process (eg third nerve palsy or Horner's syndrome)
Argyll Robertson pupil (usually bilateral)	Small pupils that accommodate, but do not constrict, to light	Pathognomonic of tertiary syphilis
Horner's syndrome (sympathetic nerve damage)	Miosis, ptosis, and anhidrosis (usually unilateral)	Lung cancer, trauma, lateral medullary syndrome, carotid artery dissection
Hutchinson's pupil	(Ipsilateral) pupil dilates and is unreactive to light	Compression of the oculomotor nerve can follow trauma or tumour often due to temporal lobe herniation
Marcus Gunn pupil (relative afferent pupillary defect)	Light shone in affected eye causes slow direct and consensual pupil reactions (light in normal eye gives brisk direct and consensual reactions)	Optic neuritis, giant cell arteritis, optic nerve trauma, retinal detachment
Miosis	Excessive pupillary constriction	Drugs such as opioids (bilateral miosis), Horner's syndrome (unilateral miosis)
Mydriasis	Excessive pupillary dilatation	Drugs, post-cardiac arrest
Oculomotor nerve palsy	Ptosis, mydriasis, and movement of the eyeball downward and outward (unopposed action of fourth and sixth nerves)	Trauma, tumour, aneurysm, demyelinating disorders, cavernous sinus thrombosis
Trochlear nerve palsy	Vertical diplopia; inability to look down and in	Trauma, haemorrhage, infarction

Chapter 12

Ear, nose, and throat

Ear, nose, and throat foreign bodies

Ear foreign bodies

Various FBs may become lodged in the external auditory canal, including beads, insects, and vegetable matter. FBs may cause pain, deafness, discharge, or, in the case of live insects, an irritating buzzing in one ear.

Diagnosis Depends upon direct visualization with the auroscope. In children, as with FBs elsewhere, there may be no history of FB available.

Removal (See Fig. 15.13 for positioning of the child.)

- Many FBs can be removed under direct vision with hooks or crocodile forceps. Manipulate gently to avoid damage or further impaction.
- Suction with a small soft plastic catheter may be successful.
- Drown live insects in 2% lidocaine first.
- Do not try to syringe out vegetable matter with water, as this may cause swelling and pain.
- If there is difficulty (especially with an unco-operative child), refer to ENT to consider removal under GA. Removing beads using an orange stick with a tiny amount of superglue on the end can work but carries some obvious dangers and requires complete patient co-operation.
- Check the tympanic membrane is intact after FB removal.

Embedded earrings The 'butterfly' piece of an earring may become embedded in the posterior part of the earlobe, causing inflammation or infection. The earrings are usually easily removed once adequate analgesia has been established—render the ear anaesthetized with a greater auricular nerve block (see ➲ Nerve blocks of forehead and ear, p. 310–11), or directly infiltrate LA into the lobe, remembering that this is a highly sensitive area. To release the butterfly, apply pressure in a posterior direction. Occasionally, forceps and a small posterior skin incision may be required to open up the track. If there is evidence of infection, prescribe oral antibiotics (eg flucloxacillin), and arrange GP follow-up. Advise the patient not to wear earrings until the symptoms have settled.

Nasal foreign bodies

These usually affect children, who present with offensive unilateral nasal discharge. They also occur in adults with psychotic illness or learning disabilities.

Removal Remove easily accessible anterior nasal FBs in the ED. However, there is a small risk of aspiration with any nasal FB, particularly in unco-operative patients. Refer such patients to an ENT surgeon for removal with airway protection. If co-operative, instruct the patient to blow their nose whilst occluding the unaffected nostril. If unsuccessful, consider attempting removal with a nasal speculum, hook, and forceps, as appropriate. A fine-bore tracheal suction catheter attached to wall suction can also work. One technique which can work in co-operative children is to ask a parent to blow into the child's mouth ('parent's kiss'), having first ensured a good seal and also occluded the normal nostril.

Nasal *button batteries* (see ➲ Button batteries, p. 220) can cause significant damage, so refer to ENT.

Inhaled foreign bodies

Aspiration causing complete upper airway obstruction is an emergency, requiring immediate intervention (see ➲ Airway obstruction: basic measures, pp. 334–5). FBs lodged in the larynx or tracheobronchial tree cause persistent coughing. There may not be a clear history—suspect an inhaled FB if a child presents following an episode of coughing/spluttering. Auscultation of the chest is often normal but may reveal wheezes or localized absence of breath sounds.

CXR May be normal or show a radio-opaque FB, with distal consolidation or hyperinflation (FB acting as a ball valve). A CXR in expiration may demonstrate this more clearly. Refer to a cardiothoracic surgeon.

Ingested foreign bodies

Various FBs, both radio-opaque (eg coins, rings) and non-radio-opaque (eg plastic pen tops, aluminium ring pulls) are frequently swallowed by children and by adults with psychiatric disorders. Provided the FB reaches the stomach, it is likely to pass through the rest of the GI tract without incident. Button batteries are an exception (see ➲ Button batteries, p. 220).

A hand-held metal detector may confirm a swallowed coin is below the xiphisternum and avoid the need for X-ray. For radio-opaque FBs, confirm with lateral neck X-ray and CXR that the FB is not impacted in the oesophagus. If there is doubt as to whether an FB is radio-opaque, consider X-raying a similar object if one is available (possibly by placing it on the patient's shoulder during the CXR).

Refer patients who are symptomatic, have impacted FBs, or have swallowed potentially dangerous items (button batteries, razor blades, open safety pins). Note that magnets can be dangerous if two or more are ingested, since they can attract each other through tissues and cause pressure necrosis/perforation of the bowel. Only discharge patients who are asymptomatic (with advice to return if they develop abdominal pain and/or vomiting), and arrange suitable follow-up. Unless the ingested FB is valuable or of great sentimental value, examination of the stools by the patient for the FB is unnecessary. It may take weeks to pass.

Impacted fish bones

Fish bones often become stuck in the pharynx or oesophagus. Direct visualization with a good light (head torch useful) and a wooden spatula as tongue depressor may reveal fish bones lodged in the tonsil or base of the tongue—remove with Tilley's forceps. If no FB is seen, obtain soft tissue lateral neck X-rays (look for prevertebral soft tissue swelling/fish bone, remembering not all are radio-opaque), then refer to ENT for endoscopy. A fish bone can scratch the pharynx, causing a sensation of FB to persist.

Oesophageal food bolus obstruction

This usually involves a lump of meat. Patients with complete obstruction are unable to swallow solids or liquids (including saliva). There may be retrosternal discomfort. Consider sips of fizzy drinks if obstruction is <24hr old and not complete. Refer patients with persistent obstruction for endoscopy. Glucagon and hyoscine butylbromide (eg Buscopan®) are used by some experts, but there is a lack of evidence to support their use and they carry a risk of side effects (vomiting, oesophageal rupture).

Facial nerve palsy

The facial (VII) nerve supplies the muscles of facial expression. Examination reveals whether VII nerve palsy is an upper or lower motor neurone type.

Upper motor neurone paralysis is usually due to a stroke (see ➲ Stroke, pp. 150–1), resulting in unilateral facial muscle weakness, but with sparing of the muscles of the forehead. If stroke is the cause, there may be additional evidence elsewhere (eg hemiparesis affecting the limbs).

Lower motor neurone paralysis results in weakness of the muscles of one side of the face. The facial nerve arises from its nucleus in the pons and emerges from the pons to travel past the cerebello-pontine angle, through the petrous part of the temporal bone, to emerge from the stylomastoid foramen and thence into the parotid gland where it divides into branches. During its passage through the petrous temporal bone, the facial nerve is accompanied by the chorda tympani (carrying taste fibres from the anterior two-thirds of one half of the tongue) and gives off the *nerve to the stapedius*. Lesions of the facial nerve in the temporal bone therefore produce loss of taste and hyperacusis (noise is distorted and sounds loud) on the affected side.

Causes of lower motor neurone facial palsy are shown in Box 12.1.

Box 12.1 Causes of lower motor neurone facial palsy

- Bell's palsy (most common cause)
- Pontine tumours
- Vascular events
- Acoustic neuroma
- Ramsay-Hunt syndrome
- Trauma
- Middle ear infection
- Cholesteatoma
- Sarcoidosis
- Parotid tumours, trauma, infection
- HIV

Bell's palsy

This is the most common cause of sudden-onset isolated lower motor neurone facial nerve palsy. Most follow a viral infection, producing facial nerve swelling in the temporal bone ± hyperacusis and loss of taste in the anterior two-thirds of one half of the tongue. The absence of involvement of other cranial nerves is a reassuring feature, helping to secure this clinical diagnosis.

Treatment Most recover completely over several months without treatment—a few are left with permanent weakness. Recovery is quicker if prednisolone is started within 72hr of onset of symptoms (prednisolone 60mg daily for 5 days, then 10mg less each day, total of 10 days of treatment OR 10 days of prednisolone 25mg bd). Antivirals are controversial and do not appear to help. Advise the use of artificial tears and an eye patch at night, to prevent corneal drying, and refer for GP follow-up.

Ramsay-Hunt syndrome

This is due to herpes zoster infection of the geniculate ganglion. Clinical features of Bell's palsy are present, together with (painful) herpetic vesicles present in the external auditory meatus and, occasionally, also the soft palate. Refer to an ENT specialist for aciclovir and follow-up.

Ear examination

Scope of the examination

Full ear examination includes assessment of the vestibulocochlear nerve and an auroscope examination. Check for mastoid or pinna tenderness. Look at the external ear canal for discharge or swelling, and examine the tympanic membrane for colour, translucency, bulging, and the cone of light.

Assessing hearing

Hearing can be assessed by asking the patient to place one finger in their ear. Stand a foot behind the patient's unoccluded ear, and whisper a two-syllable word. Ask the patient to repeat the word.

Weber's and Rinne's tests

Weber's test Strike a 512Hz tuning fork and place in the centre of the forehead. In conductive deafness, the sound localizes to the deaf ear; with sensorineural deafness, the sound localizes to the good ear.

Rinne's test Strike a 512Hz tuning fork and place it on the mastoid process. Ask the patient to tell you when they no longer hear the sound, then immediately place the tuning fork in front of the auditory meatus. In a normal ear, air conduction is heard for twice as long as bone conduction. In conductive deafness, bone conduction is heard for longer than air conduction. In sensorineural deafness, air conduction is heard longer than bone conduction.

Nystagmus

To complete the assessment of the vestibulocochlear nerve, examine for nystagmus. All forms of nystagmus can be associated with intracranial lesions, as well as peripheral causes; however, downbeat and upbeat nystagmus, in particular, signify a central cause. Tinnitus or deafness tends to suggest a peripheral cause. Peripheral nystagmus is exacerbated by gazing towards the side of the fast phase (Alexander's Law). Central nystagmus may change direction, depending on the side of gaze.

Cochlear implants

Cochlear implants consist of an implanted radio receiver and decoder package containing a magnet (above and behind the ear), together with a removable external microphone/radio transmitter. X-rays and CT do not damage this device, provided that the external microphone/transmitter is first removed and switched off. MRI can cause significant damage to the device and the patient. If there are concerns relating to a cochlear implant, refer to ENT. In particular, refer patients with:

- Significant direct trauma, including exposure by a scalp wound.
- Suspected otitis media of the implanted ear.

Earache

Otitis externa

Often caused by *Pseudomonas, Staphylococcus aureus, Streptococcus pneumoniae*, and *Escherichia coli*. Common in swimmers/surfers and after minor trauma. This causes intense itching and pain, which gradually ↑. Discharge and hearing loss may be present (profuse discharge implies middle ear disease). On examination, the external canal is inflamed and oedematous. Oedema and debris may obscure the tympanic membrane. Pain is induced by pressing on the tragus or pulling the pinna.

Management Prescribe topical antibiotics and topical steroids; advise against swimming, and arrange GP follow-up. In severe cases (eg if the drum is not visible), refer to an ENT surgeon for aural toilet to remove debris from the auditory canal.

Cellulitis or furunculosis of the ear canal

Cellulitis of the ear canal may be caused by scratching or by infection of hair follicles (furunculosis). *S. aureus* is the usual organism. Itching and a feeling of pressure are followed by pain in the ear, with deafness if the ear canal is occluded by swelling. Examination shows swelling and inflammation of the ear canal, with tenderness over the tragus and pain on movement of the ear.

Treatment Analgesia (eg NSAID) and antibiotics (eg flucloxacillin 500mg PO qds for 5 days). Arrange GP follow-up (or ENT in severe cases).

Acute otitis media

Most common in children aged 3–6y and may follow an URTI. The most common pathogens are *S. pneumoniae* and *H. influenzae*.

Presentation Earache may be accompanied by fever, deafness, irritability, and lethargy. Typically, hearing loss precedes pain. Examination of the tympanic membrane shows evidence of inflammation with loss of the light reflex and bulging of the drum. Eventual perforation results in purulent discharge, with some relief of pain. Look for associated swelling/tenderness over the mastoid—this implies secondary mastoiditis (see ➲ Acute mastoiditis, p. 567).

Treatment Prescribe oral analgesia. The use of antibiotics remains very controversial. Oral antibiotics (eg a 5-day course of amoxicillin or clarithromycin) are of questionable value but are frequently given. Prescribe oral antibiotics if the patient is very systemically unwell and/or is immunosuppressed or has significant comorbidities. Otherwise, consider antibiotics if there is no improvement within 72hr, or earlier if there is deterioration or perforation. If perforation has occurred (often heralded by a sudden ↑ in pain), arrange ENT follow-up and advise not to swim. Otherwise, arrange GP follow-up.

Acute mastoiditis

This is an uncommon, but important, diagnosis to make, because of the risk of intracranial spread of infection. Mastoiditis is a complication of acute otitis media—consider it if there is no response to therapy (eg discharging ear for >10 days). Suspect mastoiditis if there is pain, redness, swelling, or tenderness over the mastoid process. The pinna may be pushed forwards/outwards—swelling may mean that the drum is not visible. Refer urgently to the ENT surgeon for admission and IV antibiotics.

Cholesteatoma

This erosive condition affects the middle ear and mastoid. A cholesteatoma can result in life-threatening intracranial infection. There may be an offensive discharge, with conductive hearing loss, vertigo, or facial nerve palsy. Tympanic membrane examination shows granulation tissue and/or perforation with white debris. Refer to the ENT team.

Traumatic tympanic membrane rupture

This may result from direct penetrating injury, blast injury (see ➔ Blast injuries, p. 397), or basal skull fracture (see ➔ Signs of base of skull fracture, p. 369). Pain is associated with ↓ hearing. Perforation is visible on examination.

Treatment Most heal spontaneously with conservative measures and advice to keep out of water. Arrange GP (± ENT) follow-up and give prophylactic oral antibiotics according to local policy. Note that gentamicin or neomycin eardrops may cause sensorineural deafness because of ototoxicity when the tympanic membrane is ruptured.

Barotrauma

Sudden changes in atmospheric pressure with a blocked Eustachian tube can result in pain and hearing loss. This usually affects aircraft passengers and divers, especially if they have a cold (viral URTI). Pain is often relieved by the Valsalva manoeuvre (breathing out with the mouth closed, whilst pinching the nose). Decongestant nasal spray may help if the problem does not resolve spontaneously. Give analgesia (NSAID). Arrange ENT follow-up if the pain persists.

Referred pain causing earache

Earache may result from referred pain, as shown in Table 12.1.

Table 12.1 Causes of earache from referred pain

Dental origin	Non-dental origin
Dental caries or abscess	Cervical spine spondylosis
Impacted molar tooth	Ramsay-Hunt syndrome
Pharyngeal infection or tumour	Temporomandibular joint dysfunction

Epistaxis

Nasal bleeding may be idiopathic or follow minor trauma (eg nose picking). Haemorrhage can be severe when associated with hypertension and coagulation disorders. Epistaxis may follow nasal fracture and major facial injury.

Site of bleeding

Most nasal bleeding is from the anterior nasal septum in, or close to, Little's area. A few patients have posterior nasal bleeding, which may be brisk.

Equipment

Direct visualization of the anterior nasal cavity is aided by a headlamp (eg battery-operated head torch), a fine soft suction catheter, and a nasal speculum. Wear goggles to avoid blood splashes in the eyes.

Initial approach

Associated facial injury Assess ABC (especially pulse and BP), and resuscitate as necessary. Treat haemorrhagic shock (see ➲ Shock, pp. 64–5).

No associated injury Check airway, pulse, and BP. Treat hypovolaemia. Check coagulation status of patients on anticoagulants and treat appropriately (see ➲ Patients on anticoagulants, pp. 178–9). Sit the patient up, and instruct them to compress the fleshy part of their nose between their finger and thumb for 10min. If bleeding stops, the patient may be discharged after 30min observation.

Continuing bleeding after pressure

Adults Apply a cotton wool pledget soaked in lidocaine with phenylephrine. Then, with a headlamp and a nasal speculum, try to identify the bleeding point. Treat small anterior bleeding points with cautious cautery by applying a silver nitrate stick for 10–15s. Avoid excessive cautery, and never cauterize both sides of the septum—this may cause septal necrosis. If cautery stops the bleeding, observe for 15min, and discharge with GP follow-up. Advise avoidance of sniffing, picking, or blowing the nose in the meantime.

Children Applying a nasal antiseptic cream (eg Naseptin®) is as effective as cautery in stopping bleeding. The cream is relatively easy to apply, but avoid if there is a history of peanut, soya, or neomycin allergy.

Continuing bleeding despite cautery

Insert a compressed nasal tampon (eg Merocel®) or an inflatable pack (Rapid Rhino®)—follow specific product instructions on how to insert/inflate. Alternatively, pack traditionally with 1.25cm-wide ribbon gauze soaked in an oily paste (eg bismuth iodoform paraffin paste). Once packed, refer to ENT for observation, as the pack may dislodge and obstruct the airway.

Continuing bleeding despite packing

Refer to ENT. The bleeding site is likely to be posterior and can cause hypovolaemic shock. In this case, insert two large venous cannulae; send blood for FBC, coagulation screen, and cross-matching, and start an IVI. Posterior nasal bleeding usually responds to tamponade with a Foley catheter. Remove the nasal tampon, and insert a lubricated, uninflated Foley catheter through the bleeding nostril into the nasopharynx. Inflate the balloon with air, and gently withdraw the catheter, thus tamponading the bleeding site. Tape the catheter to the cheek, then re-insert the anterior tampon.

Nasal fracture

The prominent exposed position of the nose, combined with the delicacy of its bones, renders it relatively prone to injury.

Remember that the nose is part of the head, so nose injury = head injury (and potentially cervical spine injury also).

History

The nose is commonly broken by a direct blow (eg from a punch) or following a fall onto the face. Nasal fracture is usually accompanied by bleeding. Search for a history of associated facial/head injury (diplopia, loss of consciousness, etc.).

Examination

This is a clinical diagnosis based upon a history of injury with nasal swelling and tenderness. Having made the diagnosis, assess whether there is nasal deviation—it is useful to ask the patient to look in a mirror. Check and record whether the patient can breathe through each nostril. Look for an associated septal haematoma—this will appear as a smooth bulging swelling, which may obstruct the nasal passage. Children are at particular risk of septal haematoma, which predisposes to secondary infection and septal necrosis.

Assess for additional injuries to the head or face (eg tender mandible, diplopia, tender maxilla). Injury to the bridge of the nose may result in persistent epistaxis and/or CSF rhinorrhoea.

Investigations

Do not X-ray to diagnose a nasal fracture—the diagnosis is a clinical one. Obtain appropriate X-rays (eg OPG or facial views) if there is clinical suspicion of other bony injuries. Nasal fractures are often apparent on facial X-rays or CT scans.

Treatment

- Resuscitate and treat for associated head injury.
- Continuing nasal haemorrhage is uncommon—refer to an ENT surgeon to consider urgent MUA to stop the bleeding; meanwhile, insert a nasal tampon.
- Refer urgently to an ENT surgeon if there is a septal haematoma—this will require incision and drainage in order to prevent septal necrosis.
- Clean and close overlying skin wounds—Steri-Strips™ often allow good skin apposition. If there is significant contamination of the wound, start a course of prophylactic oral antibiotics (eg co-amoxiclav—one tablet PO tds for 5 days).
- Provide oral analgesia (eg ibuprofen 400mg PO tds).
- If the nose is deviated/distorted, or if there is too much swelling to judge, arrange for ENT follow-up at 5–7 days, so that MUA may be performed within 10 days. It is particularly important to ensure accurate reduction of fractures in children.
- Discharge with head injury instructions to the care of a relative.

Sore throat

Tonsillitis

Causes Acute pharyngo-tonsillitis may be caused by various agents:

- *Viral*: EBV, herpes simplex virus, adenoviruses.
- *Bacterial*: group A β-haemolytic *Streptococcus* (most common bacterial cause), *Mycoplasma, Corynebacterium diphtheriae*.

Features A sore inflamed throat often accompanies fever, headache, and mild dysphagia. Look for pus on the tonsils, and check for enlarged cervical lymph nodes—these are found in a variety of infections, but generalized lymphadenopathy (sometimes also with splenomegaly) may indicate glandular fever (infectious mononucleosis).

Diagnosis It is difficult to distinguish clinically bacterial from viral causes. Use the Centor criteria to help identify patients with a high chance of β-haemolytic streptococcal infection—three or more of: tonsillar exudate; tender anterior lymphadenopathy/lymphadenitis; absence of cough; and history of fever.

Investigations Consider throat swabs and anti-streptolysin titre in severe cases. If glandular fever is suspected, send blood for FBC and Paul–Bunnell (or Monospot) test.

Treatment Unless contraindicated, give paracetamol (1g PO qds PRN) and/or ibuprofen (400mg PO tds PRN) and discharge to the GP. Although frequently prescribed, oral antibiotics are rarely of benefit—a sensible approach is to limit their use for patients with any of the following: a history of valvular heart disease, immunosuppression, diabetes, marked systemic upset, peritonsillar cellulitis, known or suspected β-haemolytic streptococci (three or more Centor criteria—see above). In this case, prescribe phenoxymethylpenicillin (penicillin V) 500mg PO qds for 10 days (or clarithromycin 500mg PO bd for 5 days if allergic). Avoid ampicillin, amoxicillin, and co-amoxiclav, which cause a rash in patients infected with EBV.

Occasionally, patients with acute tonsillitis may be unable to swallow fluids (this is more commonly a feature of peritonsillar or retropharyngeal abscess). In this case, refer for IV antibiotics and IV fluids.

Complications Otitis media, sinusitis, retropharyngeal abscess, peritonsillar abscess.

Peritonsillar abscess (quinsy)

Typically preceded by a sore throat for several days, the development of a peritonsillar abscess is heralded by high fever, pain localized to one side of the throat, and pain on swallowing. Difficulty swallowing can result in drooling. Trismus may make inspection difficult, but if visualized, there is a tense, bulging tonsil, pushing the uvula away from the affected side. Group A β-haemolytic streptococci are frequently implicated.

Treatment Insert an IV cannula and give IV benzylpenicillin 1.2g (clarithromycin 500mg if allergic to penicillin), and refer immediately to an ENT surgeon for aspiration or formal drainage.

Retropharyngeal abscess

Spread of infection from adjacent lymph nodes may occasionally cause a retropharyngeal abscess, particularly in children aged <3y.

It is characterized by a sore throat, difficulty swallowing, fever, and dehydration. In children, cough is typically absent from the history (unlike in croup and other viral causes of upper airway obstruction). There may be evidence of airway compromise (stridor, neck hyperextension, signs of hypoxia). The differential diagnosis includes acute epiglottitis (see ➲ Acute epiglottitis, p. 693). Consider lateral neck X-ray which may show soft tissue swelling (preferably X-ray in the resuscitation room, rather than moving the patient to the X-ray department). Further imaging (CT or MRI) may be requested by the specialist team, especially if there is doubt about the diagnosis.

Treatment Get senior ED, ENT, and anaesthetic help. If the patient is a child with evidence of respiratory distress, do not upset them further. Airway obstruction may be precipitated by examination of the throat, so avoid this until appropriate staff and equipment are ready to cope with airway problems. The child can sit on mum's knee in the resuscitation room. On suspicion of a retropharyngeal abscess in an adult, insert an IV cannula; take bloods and blood cultures, and give IV fluids and IV antibiotics (eg ceftriaxone 2g IV daily + metronidazole 500mg IV tds OR if penicillin-allergic, clindamycin 600mg IV tds + gentamicin IV); refer immediately to an ENT surgeon.

Paranasal sinusitis

Bacterial infection of the paranasal sinuses (maxillary, ethmoid, frontal, and sphenoid) may result from direct spread from infected tooth roots or (more usually) be secondary to viral URTI. Most cases resolve spontaneously.

Clinical features

- Clear nasal discharge becoming purulent.
- Pain in (and often also tenderness over) the affected sinus.
- Fever.
- Headache and/or toothache.

Note that occasionally, sinusitis may become fulminant and spread to involve adjacent bones and soft tissues.

Management Provide analgesia, and suggest warm face packs and nasal decongestant (eg 1% ephedrine). Consider oral antibiotics (eg amoxicillin, doxycycline, or clarithromycin) if there is significant comorbidity or if symptoms are severe (eg high fever, severe localized unilateral pain, and purulent discharge) (see https://cks.nice.org.uk). Advise GP follow-up.

In severe cases, refer to ENT for IV antibiotics and admission.

Pharyngeal burns after cocaine use

Smoking cocaine can result in dangerous burns of the throat, since the drug acts as an LA. Swelling of the epiglottis may result in airway obstruction.

Vertigo

Vertigo is the impression or illusion of movement when there is none. It is often accompanied by nausea and/or vomiting. Distinguish vertigo from 'dizziness', which is often used to describe a feeling of light-headedness.

Causes of vertigo

Peripheral (ear) causes Typically sudden onset, severe, lasting for seconds or minutes (sometimes hours or days) and may be worsened by change in position and may be accompanied by auditory symptoms (hearing loss, tinnitus).

Central (brain) causes Typically result in milder nystagmus which is little affected by change in position, is accompanied by neurological findings, but not associated with auditory findings.
Causes of vertigo are shown in Table 12.2.

Table 12.2 Causes of vertigo according to origin

Peripheral	Central
• Benign positional vertigo	• Infection: meningitis, brain abscess
• Labyrithitis/neuronitis	• Post-traumatic
• Ménière's disease	• Subclavian steal syndrome
• Otitis media	• Vertebrobasilar insufficiency
• Wax or FB in the ear	• Stroke/cerebellar haemorrhage
• Acoustic neuroma	• MS

Note that whilst patients are often worried that their symptom of vertigo may be due to a stroke, this is not often the underlying cause, especially in the absence of other neurological signs.

Examination

- Perform a careful examination of the neurological system, including a search for cerebellar signs.
- Check the ears, including inspection of the tympanic membranes.
- Look for the characteristics of nystagmus—precipitation by changes in position strongly suggests a peripheral cause.
- Perform a Hallpike test—supporting the patient's head, with the eyes open, turn the head 45° whilst sitting upright, then lower the patient to lie supine, with the head lowered to 'hang' below the end of the trolley—ask if the manoeuvre has caused vertigo and check for nystagmus. Sit the patient back up. Repeat the test with the patient's head turned to the other side. A positive test results in vertigo (after a latent period of 5–10s), with nystagmus towards the affected side on lying down and with further transient vertigo (± nystagmus) on sitting up again.

Vertigo and driving

Advise patients not to drive whilst experiencing vertigo. Document this advice in the notes, and communicate it to the patient's GP.

Benign (paroxysmal) positional vertigo Common. Mostly results from posterior semicircular canal otoliths and can follow head injury. Sudden-onset vertigo, positional in nature, lasting for seconds or minutes at a time, but recurring, sometimes into the long term. There may be associated nausea and/or vomiting. Diagnosed with Hallpike's test (see Vertigo, p. 572). Drug treatment is not usually effective, but vestibular exercises or the Epley manoeuvre may work (see Fig. 12.1).

Acute labyrinthitis/vestibular neuronitis Usually follows a viral URTI. Vertigo is usually severe, positional in nature, and often accompanied by nausea/vomiting and sometimes hearing loss. Refer to ENT if there is hearing loss; otherwise treat symptomatically with cyclizine (50mg PO tds). Anticipate recovery within days/weeks.

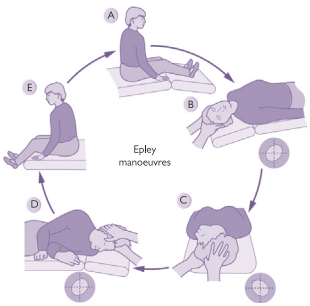

Fig. 12.1 Epley manoeuvre.
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Ménière's disease Characterized by the triad of vertigo, tinnitus, and deafness. Recurrent vertigo typically lasts for hours at a time. There may be nausea/vomiting. Try oral cinnarazine or buccal prochlorperazine. Refer to the ENT team.

Acoustic neuroma (or vestibular schwannoma) Presents with slow-onset deafness and tinnitus. Vertigo and cranial nerve lesions (V, VI, IX, X) develop over time.

Vertebrobasilar insufficiency May be associated with headache and/or neurological symptoms/signs (eg diplopia, weakness, ataxia, dysarthria). Refer to the medical team.

Stroke/cerebellar haemorrhage Sudden onset of headache, vertigo, ataxia, and/or other cerebellar signs.

Multiple sclerosis May present with vertigo ± nausea, vomiting, and eye signs.

Cause unclear The cause may be unclear, in which case refer to the medical/ENT team as appropriate.

Salivary gland problems

Saliva is a mixture containing water, various ions, mucin, and amylase, produced by the parotid, submandibular, and sublingual salivary glands. The problems most commonly affecting the salivary glands are infection and calculous disease.

Acute bilateral parotitis

Painful swelling of both parotid glands in children is most frequently due to mumps infection (see ➲ Childhood infectious diseases, pp. 230–1). In adults, painless bilateral parotid swelling may be due to Sjögren's syndrome, sarcoidosis, hypothyroidism, lymphoma, and drugs (eg oral contraceptive). In each of these cases, there are often other features, which will help in the diagnosis.

Acute unilateral parotitis

Painful unilateral parotid swelling may occur as part of mumps infection, but also in other circumstances (eg poor oral hygiene, postoperatively). Refer to an ENT surgeon for admission and IV antibiotics. Chronic painless unilateral parotitis is often neoplastic (mostly benign) in origin.

Calculous disease

Mechanical obstruction of the flow of saliva is most commonly due to salivary gland stones, usually affecting the submandibular gland, although they sometimes occur in the parotid duct system. Obstruction may also occur from neoplasms or strictures.

Features

Blockage of a salivary duct causes pain and swelling of the affected gland on eating. Bimanual palpation of the floor of the mouth may reveal a stone—occasionally, this may be visible intraorally at the duct orifice. If there is superimposed infection, it may be possible to express pus from the duct.

Investigations

Obtain X-rays of the floor of the mouth. If the patient presses down with the tongue when the X-ray is taken, the stone may be seen more easily below the mandible on a lateral view or OPG.

Treatment

Refer to an oral or ENT surgeon. If an immediate consultation is not available, discuss the use of antibiotics in the meantime (these are often reserved for situations where there is evidence of salivary gland infection).

Dental anatomy

Primary teeth erupt between 6 months and 2y and are replaced by permanent teeth which first appear at ~6y (see Table 12.3). There are 20 primary and 32 permanent teeth. The permanent teeth are made up of four quadrants of eight teeth. Each quadrant comprises two incisors, one canine, two premolars, and three molars (including the 'wisdom tooth') (see Figs. 12.2 and 12.3).

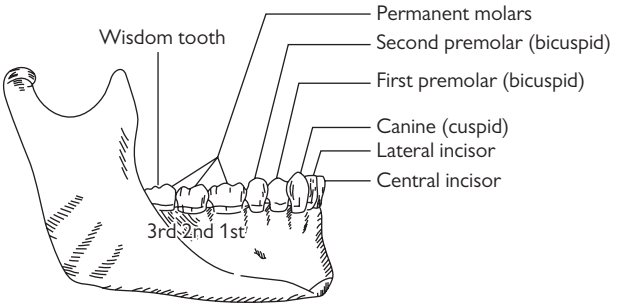

Fig. 12.2 Dental anatomy: lower jaw lateral view.

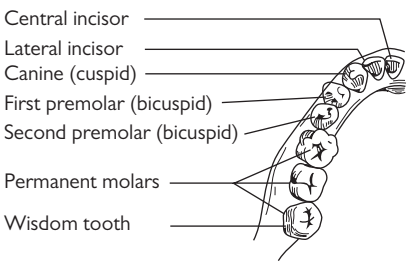

Fig. 12.3 Dental anatomy: upper jaw—view from below.

Table 12.3 Tooth eruption

	Deciduous	Permanent
Incisors	6–10 months	7–8y
Canine	16–20 months	11y
Premolars		11–13y
Molars	10–24 months	6–25y

Dental emergencies

Damaged teeth

Chipped teeth and crowns which have become dislodged Do not require immediate attention—redirect the patient instead to their dentist. Specialist 'sensitive teeth' toothpaste rubbed over the broken area of tooth may ↓ pain.

Tooth fractures which involve the pulp Present with a small area of bleeding and are exquisitely tender to touch. Refer to the on-call dentist.

Mobile teeth after trauma Need to be stabilized as soon as possible—advise the patient to avoid manipulating the tooth and to refer to the dentist.

Simple classification of tooth fractures

(See Fig. 12.4.)

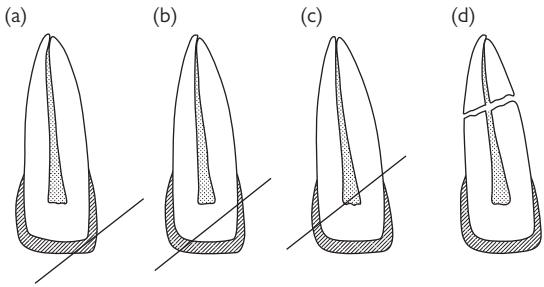

Fig. 12.4 Simple classification of tooth fractures. (a) Enamel only; (b) enamel and dentine; (c) enamel, dentine, and pulp; and (d) root fracture.

Avulsed teeth

Missing teeth Need to be accounted for (especially in the unconscious patient) in order to exclude the possibility of aspiration. Obtain a CXR to search for both the tooth and secondary problems such as pulmonary collapse and air trapping distal to the obstruction. Ensure adequate tetanus prophylaxis.

Avulsed permanent teeth Brought to the ED may be suitable for re-implantation. Avulsed primary teeth are usually not suitable. A history of rheumatic fever, valvular heart disease, or immunosuppressive treatment are contraindications to re-implantation. Milk is the best easily available transport medium to advise a patient to bring a tooth in. The best chance of success lies with early re-implantation (within the first few hours). Handle the tooth as little as possible. Hold it by the crown to clean it gently with 0.9% saline. Orientate the tooth, and then replace it within the socket using firm pressure (this may be easiest after LA—see ➲ Dental anaesthesia, p. 309). Refer immediately to the on-call dentist for stabilization and prophylactic antibiotics (eg clarithromycin). Ensure tetanus prophylaxis.

Post-extraction problems

Haemorrhage after tooth extraction May respond to simple measures. Ask the patient to bite on a rolled-up piece of gauze placed over the socket for 10min. If this is unsuccessful, consider stopping the bleeding by inserting a horizontal mattress suture (eg using 'Vicryl'), placed under LA using lidocaine with adrenaline (see Fig. 12.5). If bleeding continues despite these measures, apply direct pressure; send a coagulation screen, and refer to the on-call dentist.

Dry socket pain May follow tooth extraction (typically 3–8 days later) when bone is exposed in the empty socket. Gently irrigate the socket with warm saline. Prescribe oral antibiotics (eg penicillin or erythromycin) and analgesia, and refer to the dentist.

Fig. 12.5 Horizontal mattress suture in tooth socket.

Dental infection

Toothache is most often due to dental caries. In the absence of associated local or systemic symptoms/signs, pain usually responds to analgesia (eg ibuprofen 400mg PO tds with food ± paracetamol/codeine as necessary). Add antibiotics (eg penicillin or clarithromycin) if there is suspicion of local infection. Advise follow-up with a dentist.

Toothache with associated swelling, trismus, dysphagia, or systemic evidence of infection requires immediate referral to a maxillofacial surgeon for IV antibiotics and surgical drainage.

Temporomandibular dysfunction

Chronic pain and/or clicking in a temporomandibular joint is a relatively common problem but rarely presents to the ED. If there is no dislocation/fracture/infection, direct the patient back to the GP to consider elective referral to the maxillofacial team.

Chapter 13

Obstetrics and gynaecology

Gynaecological problems

The history

For gynaecological problems, always take a proper gynaecological history. This involves asking personal and sometimes sensitive questions, so privacy and confidentiality are of utmost importance. It is often best to interview the patient without other family members being present.

- Ask about the presenting problem. Always ask about abdominal pain, dyspareunia, and vaginal discharge.
- Take a detailed menstrual history, including the date of the last menstrual period, length of cycle, and description of the bleeding pattern.
- Obtain a full obstetric history, asking about children, pregnancies, miscarriages, terminations, and infertility treatment.
- Remember to ask about sexual activity and the type and number of partners in the past year. Also establish what form of contraception has been used.
- Ask if she has ever been treated for a sexually transmitted infection (STI).
- Find out when her last smear test was and what the result was.
- Consider differentials—ask about bladder and bowel function, plus any previous history of surgery (especially appendicectomy).

Examination procedure

Prior to performing a vaginal examination, explain the procedure to the patient and ensure you are in a private room. Allow the patient privacy to undress. Examine the patient in an unhurried manner, in the presence of a chaperone, who might usefully 'guard' the door to prevent sudden inadvertent interruption. Use a chaperone even when the patient is being examined by a ♀ member of staff. Document the name of the chaperone in the medical record. Full examination includes inspection of the external genitalia, digital bimanual palpation, and speculum vaginal examination, as well as taking necessary swabs. In certain circumstances (eg patients with painful vulval ulcers), this may not be appropriate in the ED. Vaginal examination in young children may require GA and should be undertaken by an expert. Adopt a low threshold for referring such patients. The appearance of the normal cervical os depends upon parity (see Fig. 13.1).

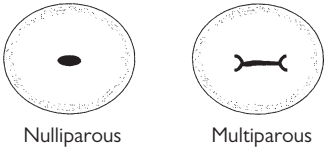

Fig. 13.1 Appearance of the cervical os.

Vulvovaginal pain

Distinguish between dysuria, dyspareunia (pain on vaginal penetration), and constant vulvovaginal pain/irritation. The latter is often associated with infection or ulceration. Enquire about other symptoms (abdominal pain, vaginal discharge, and bleeding).

Vulval ulcers

- *Herpes simplex virus* is sexually transmitted and usually due to type II virus but is increasingly due to type I virus (responsible for cold sores). Primary infection is extremely painful, lasting up to 3 weeks and sometimes causing urinary retention. Look for shallow yellow vulvovaginal or perineal ulcers with red edges. Cervical ulcers may also be present, although pain may prevent speculum examination. Liaise with the GU team (or the Gynaecology team out of hours) to treat primary infections immediately with aciclovir and analgesia and to exclude coexisting infection. Recurrent infections are less severe but may last up to a week. Treat with topical and oral aciclovir (200mg five times a day for 1 week), and arrange GU follow-up with advice to avoid sexual contact in the meantime. Leave the prescription of aciclovir in pregnancy to specialist teams and arrange for an obstetric opinion—the diagnosis may affect the fetus, as well as the mode of delivery.
- *Other STIs* may cause ulceration: syphilis (non-tender, indurated ulcers ('chancres') and lymphadenopathy), chancroid (painful ulcer caused by the streptobacillus *Haemophilus ducreyi*), lymphogranuloma venereum, and granuloma inguinale (see ➲ Sexually transmitted infections, p. 248). Refer to the GU clinic, and advise to abstain from sexual contact until treated.
- *Squamous carcinoma* causes indurated ulcers with everted edges, especially in the elderly. Refer urgently to gynaecology team.
- *Consider also*: Behçet's syndrome (arthritis, iritis, genital/oral ulceration), TB, and Crohn's disease.

Painful lumps

- *Bartholin's abscess*: infection of vestibular (Bartholin's) cyst/gland at the posterior part of the labium majus is usually due to staphylococci, streptococci, or *Escherichia coli*, but it may be due to *Neisseria gonorrhoeae*. Refer for incision and drainage and a full GU screen—likely all managed without admission unless there are signs of sepsis.
- *Infected sebaceous cysts*: may also require incision and drainage.
- *Urethral carbuncle*: this small, red, painful swelling at the external urethral meatus is due to urethral mucosal prolapse. It may cause dysuria. Refer to an appropriate clinic to consider excision or diathermy.

Pruritus vulvae

Vulval irritation may be caused by a generalized pruritic skin disorder (eg eczema), infection (particularly candidiasis), long-term skin conditions (eg lichen sclerosus), other causes of vaginal discharge (see ➲ Vaginal discharge, p. 582), urinary incontinence, threadworms, and vulval warts. Genital warts (including condylomata accuminata) are usually sexually transmitted and caused by human papillomavirus 6. Other STIs may coexist. Refer to the GU clinic.