3. Gallstones

Questions
- What are the complications of gallstones in the cystic duct?
- What are the complications of gallstones in the common bile duct?
- What tests would you perform to confirm gallstones?

Gallstones are very common, with prevalence at postmortem of 15–25%. However, 80% of stones remain asymptomatic.

Aetiology

A number of factors are associated with the occurrence of gallstones (the 'Fs': fat, fertile females over forty):

- gender (female to male 2:1) and age (> 40 years) are the most important determinants
- obesity: increases hepatic synthesis/secretion of cholesterol
- pregnancy: increased oestrogen causes choleostasis
- diabetes mellitus
- ileal disease or resection: leading to bile salt loss
- total parenteral nutrition: owing to gallbladder stasis.

There are three types of stone:

- mixed (80%): cholesterol is the main component, with bile pigments and calcium salts
- pigmented (10%): contain calcium bilirubinate, are associated with haemolytic disorders (haemolytic anaemia, malaria) and are rare in Western countries
- pure cholesterol (10%).

Investigations

Blood tests.   Liver function tests may be normal or show an obstructive picture (bilirubin, alkaline phosphatase, alanine aminotransferase or aspartate aminotransferase may be raised). Serum amylase excludes associated acute pancreatitis. Blood cultures identify septicaemia in the seriously ill.

Ultrasound.   The best test in the acute phase as it demonstrates gallstones (as an 'acoustic shadow') in over 90% of patients. (Only 10% of stones are radioopaque and so plain radiograph is unsuitable.) Ultrasound also will show wall thickening, pericholecystic collections, dilatation of common bile duct (CBD) and intrahepatic biliary tree.

Endoscopic retrograde cholangiopancreatography (ERCP). This allows visualization of the CBD and pancreatic duct via the ampulla of Vater; additionally any stones can be retrieved (Fig. 3.3.1; see Ch. 4).

Magnetic resonance cholangiopancreatography (MRCP). Contrast dye is secreted in bile, allowing a three-dimensional view of the entire biliary tree and stones.

Percutaneous transhepatic cholangiography.   This approach is only used when the above are unsuitable or have failed to visualize the stones; contrast dye is injected percutaneously into the liver and biliary tree, allowing direct visualization of stones on radiograph.

Complications of gallstones

Complications depend on the level at which the stones impact: in the **cystic duct** or (Fig. 3.3.2) in the **CBD**. Management options for each are dealt with in Ch. 4. Most stones (90%) remain in the gallbladder or cystic duct.

Stones in the gallbladder/cystic duct

1. *Biliary colic.* This is caused by a transient impaction in Hartman's pouch or the cystic duct. The patient often does not present to hospital. It is characterized by:
   - pain: episodic right upper quadrant or epigastric pain; it is not a 'colicky' pain but is progressive and then constant for around 1 to 4 h until the stone passes and pain is relieved; the pain radiates to the right shoulder (diaphragmatic irritation) and is provoked by fatty foods
   - nausea and vomiting
   - acute cholecystitis occurs if infection develops.
2. *Acute cholecystitis.* This is caused by an impacted stone in Hartmann's pouch with bacterial infection. There may be a previous history of biliary colic. Common infective

Fig. 3.3.1 Multiple gallstones in the common bile duct visualized with ERCP. Dye shows the biliary tree and a dilated proximal common bile duct.

organisms are Gram-negative gut flora (*Escherichia coli*), which reach the gallbladder via the blood or bile. Symptoms may occur as separate, repeated attacks:

■ pain with fever: epigastric and/or right upper quadrant pain radiating to the right shoulder tip

■ Murphy's sign: two fingers are placed in the right upper quadrant, pressing on the gallbladder; there is pain when the patient inhales that ceases when the patient breathes out (positive if no pain on left side)

■ nausea and vomiting, with tachycardia and pyrexia.

Chronic cholecystitis leads to a fibrosed and enlarged gallbladder wall and atrophic mucosa. **Acalculous cholecystitis** is rare and occurs without stones; it can be caused by burns, sepsis and diabetes.

3. *Mucocele and empyema.* A mucocele occurs when a stone impacts in Hartmann's pouch; continued mucus secretion behind the stones causing distension and tenderness. An infected mucocele **abscess** (empyema), although rare, may cause perforation of the gallbladder. **Mirrizi's syndrome** is an uncommon complication of a stone impacted in the cystic duct. This causes swelling of the gallbladder and cystic duct, which then compress the CBD and lead to obstructive jaundice.

Stones in the common bile duct

Of patients with gallbladder stones, 10% also have stones in the CBD, which have almost always migrated from the gallbladder (rarely formed in the duct itself). Many small stones pass into the duodenum, causing mild colic or mild jaundice; the larger stones may cause blockage.

4. *Obstructive jaundice.* Impaction of a stone in the CBD leads to obstructive jaundice. A stone in the CBD causes biliary colic or acute cholecystitis plus obstructive jaundice, since the bile drainage of the gallbladder and liver are blocked. It may recur in repeated attacks. Management is similar to acute cholecystitis. Obstructive jaundice involves conjugated bile acids (water soluble), leading to **dark urine** (dissolve in the urine) and **pale stools**.

5. *Ascending cholangitis.* When obstructive jaundice occurs, the stagnant bile above the stone may become infected, producing cholangitis and signs of **Charcot's triad**: rigors (fever and chills), obstructive jaundice and pain (upper right quadrant, radiates to right shoulder). This infection may spread to the intrahepatic ducts (hence 'ascending' cholangitis), and may cause a liver abscess or septicaemia. It is life threatening so the biliary system needs urgent decompression with ERCP or surgery.

6. *Gallstone pancreatitis.* If a stone impacts at or beyond the point of drainage of the pancreatic duct into the CBD, symptoms of obstructive jaundice plus acute pancreatitis are seen as bile refluxes into the pancreatic ducts.

Other

7. *Gallstone ileus.* This is small bowel obstruction caused by a stone which has perforated directly through the gallbladder wall into the duodenum via a **cholecystoenteric fistula**. Gas is visible in the biliary tree on plain abdominal radiography. Some stones may obstruct the ileocaecal valve, causing small bowel obstruction. This is a rare complication occurring mainly in the elderly.
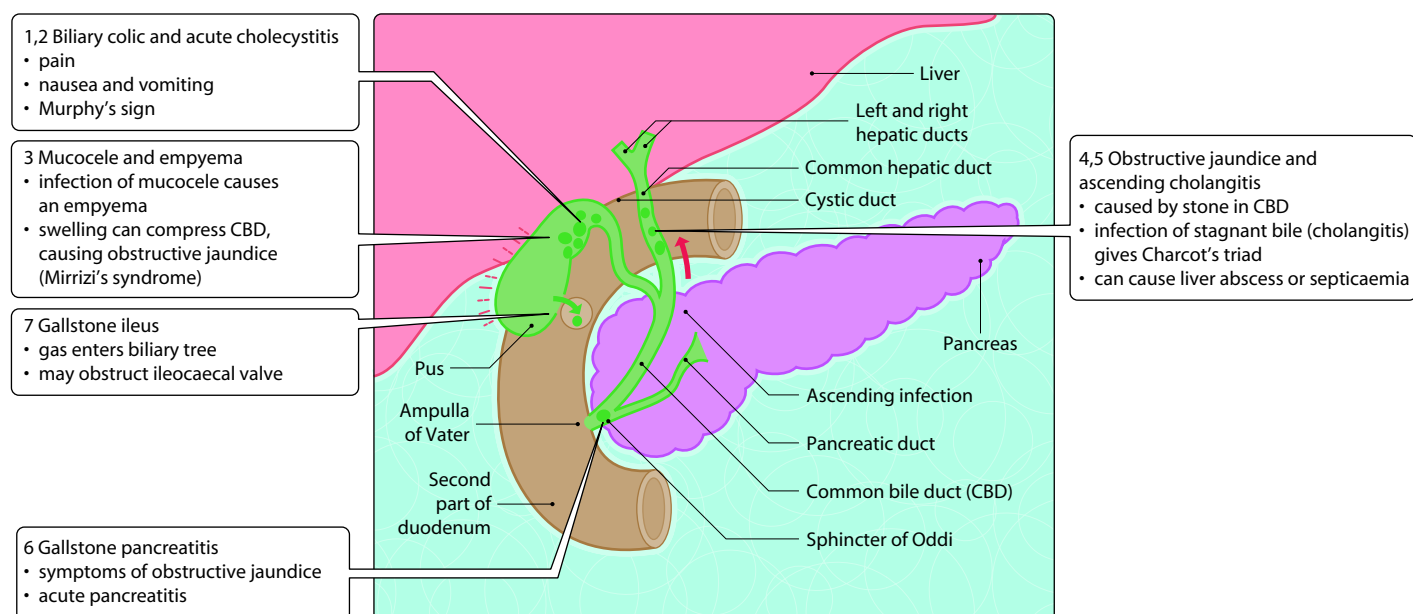

Fig. 3.3.2 The biliary tree and complications of biliary disease.

25. Aneurysms

Questions
- What are the different types of aneurysm?
- How is a patient with a ruptured abdominal aortic aneurysm initially managed?
- What are the complications of elective abdominal aortic aneurysm repair?

Aneurysms are permanent, localized dilatations in an arterial wall. They commonly occur in the abdominal aorta and popliteal arteries and are most common in males over 65 years (Fig. 3.25.1).

Aetiology

The most common cause is **atherosclerosis**. Other causes are connective tissue disorders (Marfan's syndrome, Ehler–Danlos syndrome), congenital (Berry aneurysms in the circle of Willis), or infective (e.g. syphilis; now rare).

◼ ABDOMINAL AORTIC ANEURYSM

Clinical features

Abdominal aortic aneurysms may present either as an asymptomatic pulsatile abdominal mass diagnosed incidentally or as an emergency with pain, distal embolization or rupture. Over 90% are **infrarenal**, which means surgical repair is easier;
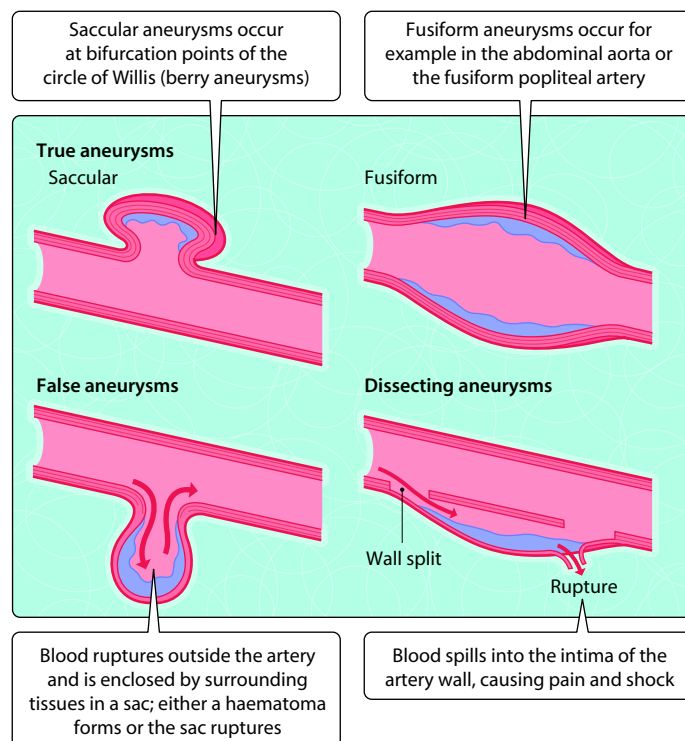

| Saccular aneurysms occur at bifurcation points of the circle of Willis (berry aneurysms) | Fusiform aneurysms occur for example in the abdominal aorta or the fusiform popliteal artery |

True aneurysms
Saccular

Fusiform

False aneurysms

Dissecting aneurysms

Wall split

Rupture

| Blood ruptures outside the artery and is enclosed by surrounding tissues in a sac; either a haematoma forms or the sac ruptures | Blood spills into the intima of the artery wall, causing pain and shock |

Fig. 3.25.1 Classification of aneurysms.

those above or involving the renal arteries are much more difficult to repair and have higher mortality.

Acute presentation:

- ruptured abdominal aortic aneurysm: pain is the most common symptom, which may be abdominal (with guarding) or sudden-onset back pain
- patients may collapse and be hypotensive with a tachycardia
- an acutely ischaemic limb: the aneurysm may 'spit out' emboli that block distal leg arteries (Ch. 23).

Non-acute presentation occurs in asymptomatic patients (e.g. during ultrasound scan or plain radiograph (showing calcification in the aortic wall) performed for another reason) or as a complaint of a mildly bloated abdomen, back pain or pulsation.

Investigations

Examination to detect an abdominal aortic aneurysm is described in Ch. 6. **Ultrasound** shows its diameter and should be repeated at regular intervals to monitor growth if it is below the threshold for repair (< 5.5cm). The use of ultrasound in screening for abdominal aortic aneurysm is currently being assessed. CT is performed preoperatively to check for renal artery involvement and assess suitability for endovascular repair (Fig. 3.25.2). In an emergency presentation, the only investigations that are performed preoperatively are FBC, clotting studies, cross-matching for 6–8 units of blood and electrocardiograph. If the patient is stable with back pain, there may be time for CT.

Management

Elective surgery is undertaken in the fit patient to prevent rupture for aneurysms > 5.5 cm in diameter, for those which are growing faster than 1 cm/year or if symptomatic (pain or emboli). Mortality associated with elective surgery is approximately 5%.

Emergency management of a ruptured abdominal aortic aneurysm

Initial management is ABC with oxygen, intravenous access (two large-bore cannulae in two large arm veins) and careful fluid management.

Fluid management. A haematoma may have formed around the aorta, and increasing the patient's blood pressure may dislodge it, causing the patient to bleed to death. Therefore, the patient's systolic blood pressure should be maintained carefully at or below 100 mmHg.

Surgery. The patient should be taken to theatre immediately where a conventional aneurysm repair is used (Fig. 3.25.3): 50% of those with a rupture reach hospital, and 50% of these patients survive emergency repair (overall mortality is 75–85%).

1. the patient is given intravenous heparin for anticoagulation
2. The aorta is cross-clamped above the aneurysm; the limbs are supplied by collateral (alternative) arteries
3. The aneurysm is cut open and the thrombus removed
4. A dacron graft is sewn inside the aorta, and the clamp removed to check for leaks
5. The rest of the aorta wall is sewn around the dacron graft.

In selected patients, an endovascular option exists, where the stent is placed within the aneurysm via the femoral artery. The complications are described on p. 8.

◼ OTHER ANEURYSMS

If one aneurysm is found, the rest of the peripheral vascular system should be carefully examined to identify others.

Popliteal aneurysms. These are the second most common and often bilateral. They may cause distal emboli, or may thrombose, presenting as an acutely ischaemic limb. If asymptomatic but > 3 cm in diameter, some advocate repair with a bypass graft.

Thoracic aortic aneurysms. These may be of the aortic arch or the ascending or descending thoracic aorta. They may present as chest pain, back pain, aorto-oesophageal fistula (with lethal haematemesis), obstruction of the superior vena cava or recurrent laryngeal nerve and tracheal compression. Assessment is with chest radiograph, CT and transoesophageal echocardiography. Under cardiac bypass, the aneurysm is partially excised and a synthetic graft inserted. These aneurysms may dissect (blood splits the intima of the artery wall), causing severe chest and upper back pain, and severe shock. Mortality is high and emergency surgery is required. Traumatic damage to the thoracic aorta by high-energy trauma is immediately life-threatening, requiring urgent surgery.

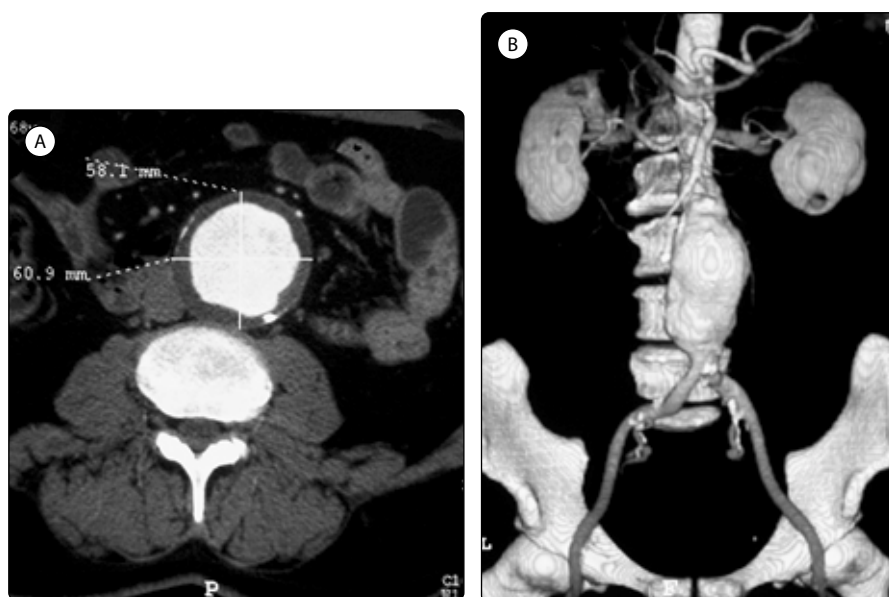*Femoral aneurysm.* One cause of a groin lump (Ch. 33).

The aneurysm is opened, the thrombus removed and the lumbar arteries are oversewn

The clamps are loosened to check for leaks *before* final closure of the aorta

A Dacron graft is stitched *inside* the aorta, proximal anastomosis first

Fig. 3.25.3 The repair procedure for an abdominal aortic aneurysm.

Fig. 3.25.2 An infrarenal abdominal aortic aneurysm, shown on conventional CT scan (A), and on three-dimensional CT reconstruction (B).
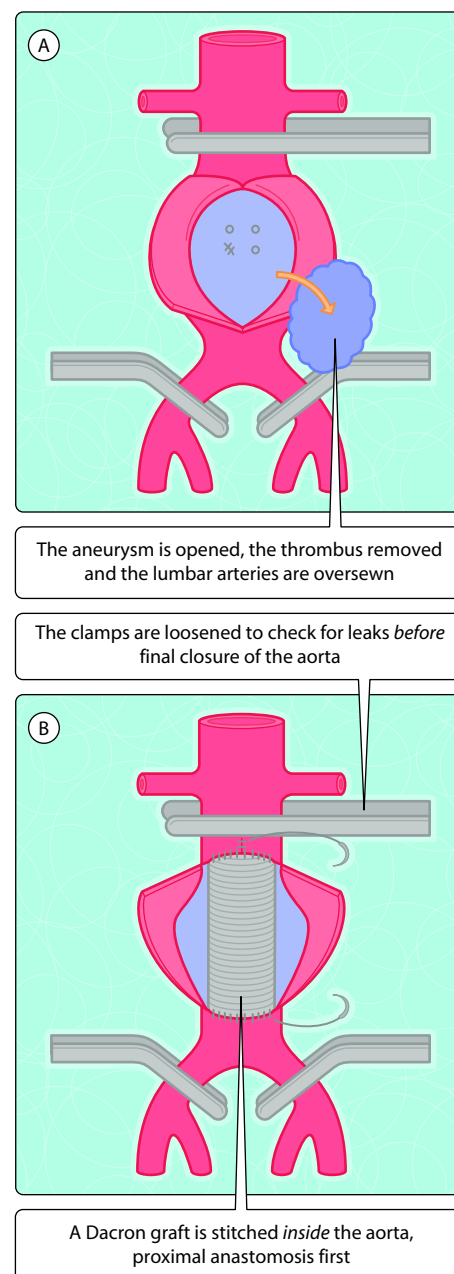
52. Paediatric fractures

Questions
- How do paediatric fractures differ from those in adults?
- What is a greenstick fracture?
- What is the main complication of a supracondylar fracture?

Paediatric fractures are common, especially in the forearm. They differ in terms of structure and repair from fractures in adults. Paediatric fractures are often greenstick fractures, heal faster and remodel to a greater degree; overgrowth occurs and growth plate injuries are important.

Fracture patterns

Growth plate injuries.   Injuries through a growth plate may result in complete or partial growth arrest, and thus limb length discrepancy, which may cause premature osteo-arthritis. Such injuries are classified by the Salter–Harris classification (Fig. 3.52.1), where severity increases with grade.

Greenstick fractures.   One side of the cortex breaks but the other stays intact (Fig. 3.52.2) as the child's bones are soft.

Buckling fractures.   One side of the cortex buckles; if the force continues, the bone breaks to form a greenstick fracture.

Overgrowth.   Paediatric fractures have a tendency to overgrow so some shortening is desirable when reducing the fracture.

Non-accidental injuries.   The key orthopaedic features are metaphyseal fractures, posterior rib fractures, fractures at different stages of healing, complex skull fractures and spiral long bone fractures. A parent who cannot produce a corroborative history for a fracture is suspicious. **Osteogenesis imperfecta** (blue sclera and brittle bones) and **calcium deficiencies** (rickets. hyperparathyroidism) are medical conditions that should be excluded.

Common fractures

The following are the most common fractures in children.

Distal radius

The distal radius is the most commonly fractured site in children, and is often a greenstick fracture. Even though a dinner-folk deformity forms, a Colles' fracture does not occur in
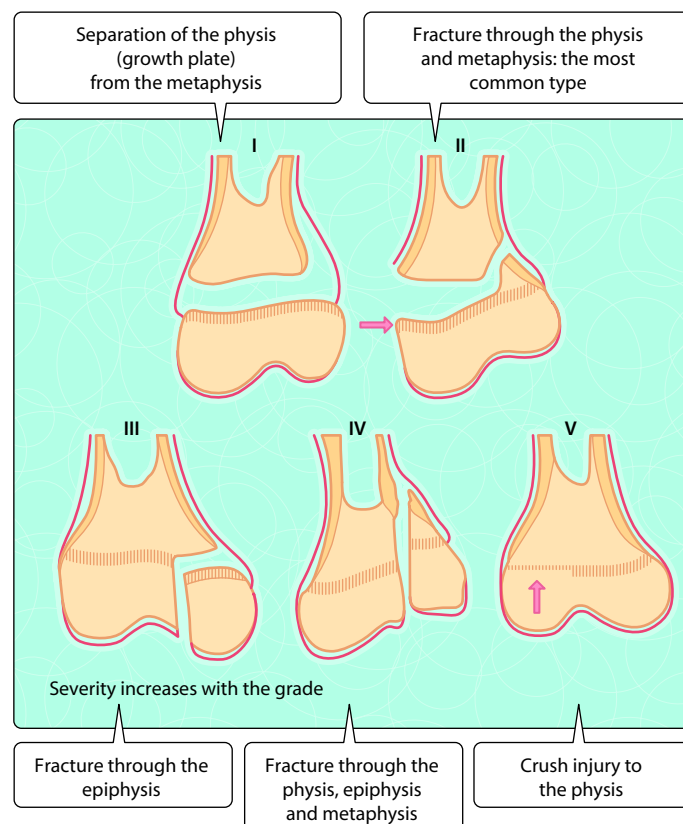

Separation of the physis (growth plate) from the metaphysis

Fracture through the physis and metaphysis: the most common type

I
II

III
IV
V

Severity increases with the grade

Fracture through the epiphysis

Fracture through the physis, epiphysis and metaphysis

Crush injury to the physis

Fig. 3.52.1 Salter–Harris classification of growth plate injuries.

Fig. 3.52.2 A greenstick fracture of the radius. There is buckling of the posterior surface and a fracture of the anterior surface (arrows). Note that the epiphyseal plates are still visible; these will later fuse when the child reaches skeletal maturity.

children; either the radial epiphysis has separated or a greenstick fracture of the distal radius has occurred. The fracture is treated conservatively with reduction and plaster immobilization. A certain amount of displacement is allowed, since a child's growing bones remodel, and subsequently the degree of angulation decreases with age (Table 3.52.1).

Supracondylar fracture

This fracture most commonly occurs as a result of a FOOSH and has a peak age of occurrence at 7 years. It must be recognized quickly as there are numerous serious associated complications.

Neurovascular damage. The displaced fractured bones may damage the **brachial artery** and **median nerve**, and so the neurovascular status of the limb must be checked at presentation and constantly reassessed.

Compartment syndrome. There is a high risk of a compartment syndrome (Ch. 53), as the brachial artery and median nerve can both be compressed by swelling in the anterior compartment.

Volkmann's ischaemic contracture. This can follow disruption to the blood supply or a delay in treatment of a compartment syndrome. The distal limb becomes fibrosed and the joints contract (especially the fingers), leading to flexion deformity and wasting, thus severe limitation of function.

Mal-union and myositis ossificans. Myositis ossificans is an aberrant reparative process where deposition of bone occurs in an area of muscle or soft tissue, here leading to elbow stiffness.

An undisplaced supracondylar fracture is treated by holding the limb in a plaster backslab. If the fracture is displaced, closed reduction is attempted under anaesthesia and the child admitted for observation (in case of compartment syndrome). If closed reduction cannot be achieved, ORIF with K-wires is required. Any lost median nerve function almost always recovers.

Femoral fracture

Closed femoral fractures in young children are treated non-operatively. In children under 3 years, both limbs are held up in traction using a **Gallow's splint**. Older children may require external fixation, intramedullary nails or a plate.

Pulled elbow

Pulled elbow typically occurs in a child under school age who has been tugged on the arm. The head of the radius slips out of the annular ligament but then returns to its normal position (but out of the ligament). Consequently, there is no clinical deformity but the elbow is generally tender and movement is restricted. Radiographs are not required. It is relocated by pulling on the arm while flexing the elbow and supinating the hand, where the radial head clicks back into the annular ligament.

Osteogenesis imperfecta

Osteogenesis imperfecta is a genetic condition affecting collagen production; it is characterized by blue sclera, deformities from fragile bones and a high susceptibility to fractures (Table 3.52.2). It is more common in Afro-Carribeans. Radiograph shows multiple fractures at different stages of healing (so it can be confused with a non-accidental injury). Treatment is to maximize safety (teach parental handling skills, inform school), and the fractures are treated individually. Osteotomies correct deformity and intramedullary stems fix long bones.

Table 3.52.1 DEGREE OF ANGULATION ALLOWED IN A FRACTURE OF THE DISTAL RADIUS

Age (years)	Degree of angulation
4–9	20
9–11	15
11–13	10
13+	5

Table 3.52.2 TYPES OF OSTEOGENSIS IMPERFECTA

Type	Diagnosis	Characteristics	Inheritance
I	Dominant, blue sclera	Type IA has brittle bones, blue sclera, normal teeth; type IB also has dentinogenesis imperfecta (discoloured and damaged teeth) and deafness	Autosomal dominant
II	Lethal perinatal	Deformed skeleton and multiple fractures (a lethal condition in utero)	Autosomal dominant
III	Progressive deforming	Multiple fractures at birth, progressive deformities and dentinogenesis imperfecta, normal sclera	Autosomal recessive
IV	Dominant, white sclera	Similar to type I, but with white sclera; difficult to diagnose	Autosomal dominant

60. Knee pain

Questions
- What is the management of a ligamentous injury?
- What are menisci and how are they damaged?
- What are the causes of knee pain in children?

Knee pain may present suddenly after injury or as a chronic pain, and it occurs in both adults and children.

Knee pain in adults

Ligament damage

Sporting injuries are a common cause of ligament damage. Strains or isolated tears may settle with rest, analgesia and plaster or bracing; acute tears and complex or chronic injuries often benefit from surgical reconstruction.

Anterior cruciate ligaments. A tear in an anterior cruciate ligament is a common sporting injury (Fig. 3.60.1) following a twisting motion or hyperextension. The patient reports feeling something break or give way and the joint swells rapidly. There is a positive anterior draw test and Lachman's test, and an effusion may be present. Initial management is conservative with aspiration of a haemarthrosis, analgesia and physiotherapy. One-third improve with this regimen; one-third manage with the decreased level of function, but one-third need arthroscopic repair, either to achieve a higher level of function (high-level contact sports) or for severe instability.

Posterior cruciate ligament. Injury occurs with the knee flexed, when the tibia is forced backwards; another ligamental injury is common (e.g. collateral ligament). The posterior draw sign is positive. Treatment is similar to anterior cruciate ligaments although repair is more difficult.

Medial collateral ligaments. Medical collateral damage usually occurs with an associated anterior cruciate ligament and medial meniscal injury (the 'unhappy triad'). Treatment is with either immobilization in a cast for 6 weeks or surgical reconstruction to restore stability.

Lateral collateral ligament. Damage here is rarely isolated, and instability is less common than with medial collateral ligament injury. Treatment is conservative.

Meniscal injuries

Meniscal tears are common and the majority will involve the medial meniscus. They are either traumatic or degenerative. Traumatic tears are common in those whose occupation involves crouching, kneeling, turning or trauma. The majority of degenerative tears are asymptomatic and are present in 65% of those over 60 years of age. There are different types of tear, classified by their appearance (Fig. 3.60.2).

Clinical features. Patients are usually fit, young and males; there is pain, joint line tenderness and positive McMurray's test. Symptoms may settle, but episodes may be repeated.

Investigations. MRI and arthroscopy confirm the nature and location of injury.

Management. Arthroscopy and partial meniscetomy are the mainstay of treatments, and meniscal repair can be attempted. Total meniscetomy is avoided as the risk of premature osteoarthritis is high.

Chronic knee pain

Osteoarthritis. The knee is a common site for osteoarthritis,
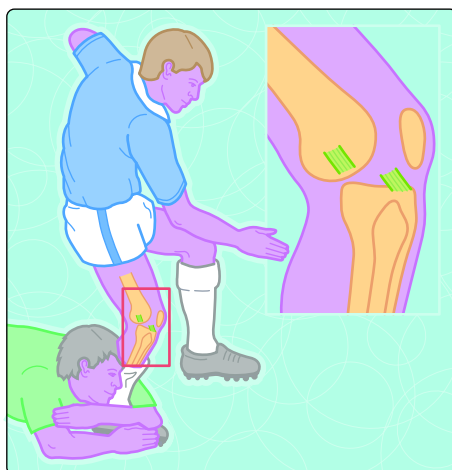

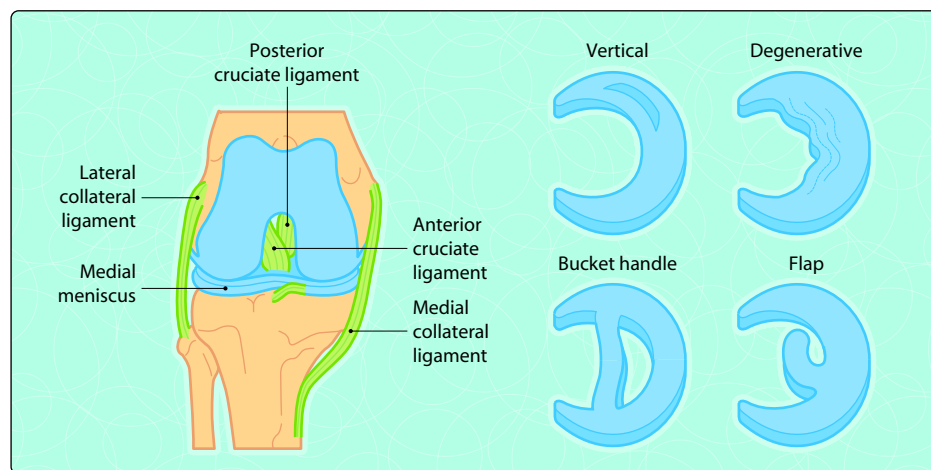
Fig. 3.60.1 Anterior cruciate ligament tear.

Fig. 3.60.2 Examples of meniscal tears.

which may be caused by previous injury or excessive use of the knee or an old meniscal tear. It results in pain, swelling, deformity and stiffness. General management options for osteoarthritis are discussed in Ch. 58. Knee osteoarthritis in young patients is managed conservatively for as long as possible. Steroid injections are avoided, and **osteotomies** (alteration of the joint line to allow a new-weight-bearing surface to be brought into use; Fig. 3.60.3) delay the need for knee replacement. Specific treatments for the knee include:

■ **arthroscopic washout** to remove loose particles of cartilage that are causing pain
■ **arthroplasty**: unicompartmental replacement is suitable for early disease, and total knee replacement (Fig. 3.60.4) for advanced 'tricompartmental' disease.

Rheumatoid arthritis. Rheumatoid arthritis causes effusions and a valgus deformity; surgical treatment includes arthroscopic washouts, synevectomies and total joint replacements.

Localized pain and swellings. There are several causes:

■ jumper's knee: occurs at the insertion of the patellar ligament onto the patella and is similar to tennis elbow
■ bursae*:* **prepatella bursitis** (housemaid's knee; leaning forward on the knees) and **infrapatella bursitis** (clergyman's knee; prolonged periods of kneeling); treatment is with activity modification, aspiration or excision
■ popliteal cysts (Baker's cysts) usually accompany rheumatoid arthritis and may burst spontaneously; they are mostly treated conservatively.

Knee pain in children

Osteochondritis dissecans

Osteochondritis dissecans is an idiopathic disease typically affecting boys aged 8–12 years; it is characterized by partial or complete detachment of a fragment of bone (Fig. 3.60.5). It most commonly affects the lateral surface of medial femoral condyle (the talus, femoral head and first metatarsal head may also be affected). Repeated minor trauma, ischaemic changes or genetic predisposition are all implicated. Many partially separated fragments reunite conservatively with rest, although loose bodies in the joint space require surgical removal.

Anterior knee pain

Anterior knee pain is most commonly seen in adolescent girls. It is caused by softening of the cartilage on the posterior aspect of the patella, caused by stresses around the knee known as **chondromalacia patella**. Treatment is conservative as the pain usually settles with skeletal maturity.

Osgood–Schlatter's disease

Osgood–Schlatter's disease is most common in boys aged 11–15 years. The tibial tuberosity is lifted off the tibia when young athletes exert too great a traction in their underdeveloped apophysis (**traction apophysitis**). There is pain on activity; swelling and a painful lump may be found. Radiographs may show fragmentation of the tubercle. Spontaneous recovery is usual but takes time (up to 2 years), and periods of rest may have to be reinforced with a plaster cast. Loose fragments in the joint require surgical removal.
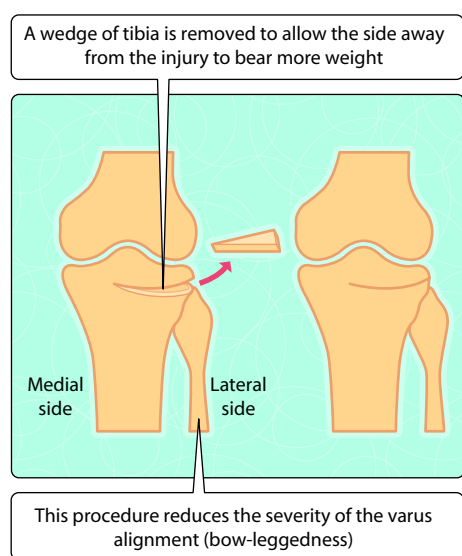

A wedge of tibia is removed to allow the side away from the injury to bear more weight

Medial side   Lateral side

This procedure reduces the severity of the varus alignment (bow-leggedness)

Fig. 3.60.3 Tibial osteotomy shown on an anterolateral view of the knee.

Fig. 3.60.4 Total knee replacement in lateral view. Infection has caused septic loosening around the prosthesis (arrows).
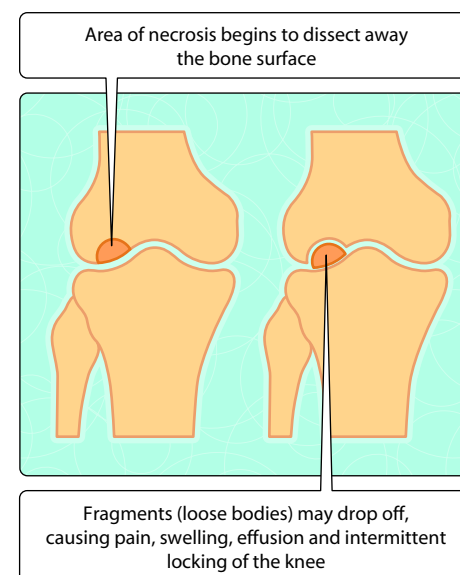

Area of necrosis begins to dissect away the bone surface

Fragments (loose bodies) may drop off, causing pain, swelling, effusion and intermittent locking of the knee

Fig. 3.60.5 The disease process in osteochondritis dissecans.