“A fracture is a soft tissue injury, complicated by a broken bone”. This is an important concept to remember when thinking about the potential complications, as many will be related to soft tissue rather than bony injury.

This chapter will be broken down into two sections; general complications and fracture specific complications.

General Complications

General complications refer to the things you must have in your mind when you assess any patient with a fracture. Orthopaedic surgeons are often accused of treating the bone, the whole bone and nothing but the bone. I would like to think this is not true! These are the things you should consider initially when assessing a patient:

Complications of distant tissue damage:
- haemorrhage and shock
- fat embolism syndrome
- muscle damage and rhabdomyolysis

Complications of prolonged bed rest:
- chest and urinary tract infection
- pressure sores and muscle wasting
- deep vein thrombosis and pulmonary embolism

Haemorrhage and Shock

Before looking at the fracture you must assess the patient using the ATLS® algorithm (see the previous section on ‘management the trauma patient’). It is imperative to remember that the patient may have multiple injuries or injuries unrelated to any obvious fracture.

In the traumatised patient carefully look for head, intra-abdominal or intra-thoracic injuries which will likely take priority in management over and above most fractures.

Fat Embolism Syndrome (FES)

Fat embolism can complicate long bone fractures and is caused when multiple small emboli are released into the circulation from the fracture site.

The symptoms of FES typically present 1 - 3 days post injury and may include:
- respiratory distress and hypoxia
- agitation and delirium
- anaemia and thrombocytopenia
- petechial rash (only present in a third of cases)

The management of the condition is supportive as there is no direct treatment. Oxygen should be used to counteract the hypoxia and coagulation abnormalities should be corrected. The best way of avoiding the condition is to reduce the fracture as soon as possible. Remember though that the condition is rare and other differential diagnoses such a pulmonary embolism must be considered.

Muscle Damage and Rhabdomyolysis

Rhabdomyolysis is a condition which occurs when skeletal muscle is rapidly broken down releasing myoglobin into the circulation. This is seen in patients who have suffered a crush injury and those who have been immobilised on the floor for a significant time period causing a pressure injury. A typical example would be an intoxicated patient who has fallen and remained on the floor overnight or an elderly patient with a neck of femur fracture who is unable to get up.

The release of myoglobin can cause acute renal failure to develop. The patient will also have local pain in the affected area and in severe cases compartment syndrome (see below) or pressure sores may develop.

All patients who could be considered to be at risk should have a serum creatinine kinase (CK) and urea and electrolytes (U&Es) checked. Very high levels of CK are often found and it continues to rise for up to 12 hours following injury. It will remain elevated for a number of days. Hyperkalaemia is commonly seen and should be expeditiously recognised and treated.

Treatment involves intravenous normal saline (0.9%) and the U&Es should be checked twice a day for deterioration. The patient should be catheterised to allow close monitoring of their fluid input/output balance. Their urine will look dark brown as the myoglobin is filtered by the kidneys. This is called myoglobinuria. The image below shows a urine sample from a patient with myoglobinuria.

Remember that the urine will remain dark brown despite good hydration due to persisting myoglobin. It is not necessarily a sign of dehydration.
Chest Infection and Urinary Tract Infection

Patients who sustain a fracture are often subjected to a period of prolonged immobility. This is particularly true of elderly patients with lower limb fractures. In the elderly group this period is commonly spent in hospital or a rehabilitation institution when the risk of hospital acquired infection (nosocomial infection) is high.

These patients are at high risk of developing respiratory tract infections such as hospital acquired pneumonia (HAP). A patient is considered to have HAP rather than community acquired pneumonia when they develop new symptoms over 48 hours after admission. HAP tends to be more serious than CAP as the causative organisms are often more resistant to antibiotics.

Urinary tract infection is also common in hospitalised orthopaedic patients. Up to 80% of hospital acquired UTIs are due to catheterisation. Patients are commonly catheterised pre- or intraoperatively for a range of reasons. Patients should have their catheter removed as soon as is practical following surgery.

Pressure Sores and Muscle Wasting

Pressure sores commonly occur in the elderly immobilised patient. They occur due to tissue ischaemia from prolonged pressure, usually over the bony prominences of the skull, sacrum and heels. The keys to prevention are careful nursing and early mobilisation of the patient. Once a pressure sore has developed it can be very difficult to treat.

Physiotherapy and early mobilisation are also key to prevent muscle wasting. It is remarkable how quickly muscle atrophy occurs with immobilisation. If you examine a young patient who has been immobilised for some weeks in an above knee plaster on one leg whilst remaining mobile with the other the difference between the two once the plaster is removed is usually significant.

Deep Vein Thrombosis and Pulmonary Embolus

Venous thromboembolism (VTE) is quoted as occurring most commonly at around day 6 post injury but there should always be a high index of suspicion for the condition in any orthopaedic patient with unilateral leg swelling or whose clinical condition has acutely deteriorated (as seen in pulmonary embolus).

Asymptomatic deep vein thrombosis (DVT) is estimated to occur in around 25% of all orthopaedic inpatients, although the condition is not usually clinically evident in the majority. The condition is caused by a blood clot forming in the deep venous system of the lower limb. There are many risk factors for the condition including smoking, obesity, previous DVT/PE, prothrombotic clotting disorders, infection, malignancy and oral contraceptive use.

The risk is exacerbated by the propensity for orthopaedic patients to be bed bound for a number of days, preventing the ‘calf pump’ from moving blood out of the lower limbs and by the fact that orthopaedic surgery on its own puts patients at increased risk.

All orthopaedic patients should have multimodal prophylaxis against VTE. This falls into two main groups; mechanical and chemical. Examples of mechanical prophylaxis are DVT stockings, which prevent pooling of blood in the deep venous system and intermittent pneumatic compression devices which act as a proxy for the dormant calf pump. Chemical prophylaxis in trauma patients is usually provided by daily low molecular weight heparin injections such as Enoxaparin which thin the blood reducing the risk of clotting. They do however increase the risk of bleeding complications and so are occasionally not appropriate.

Typical symptoms of a DVT are a swollen, painful, warm erythematous limb. The symptoms occur due to venous congestion with the clot preventing venous return from the affected limb. The following image shows a patient with a DVT in their right leg (arrow).

Investigation of DVT is with the use of lower limb vascular ultrasonography. The deep calf veins are imaged using an ultrasound probe which can identify the presence of a blood clot or thrombus.

Pulmonary Embolus (PE) is a life threatening condition which occurs when part of the thrombus in a DVT dislodges from its position in the deep calf veins and travels through the circulation to the vascular bed of the lungs. This causes obstruction of blood flow in the lung leading to a ventilation - perfusion mismatch. The result is dyspnoea, chest pain, haemoptysis, hypoxia and tachycardia. In my experience one of the most important signs is an unexplained tachycardia, even in the absence of the other signs. Have a low threshold for investigating these patients for PE.

Investigation of PE is with the use of a V/Q scan which looks for a ventilation/perfusion mismatch or more commonly now a CTPA (computed tomography pulmonary angiogram).
The image below shows a CTPA from a patient with multiple proximal pulmonary emboli, with the areas of thrombus (clot) shown by the arrows. They are seen in the proximal vessels.

Many people still use d-dimers as a first line investigation for VTE. In a post trauma or post surgery group it is a useless investigation as it will always be raised due to the trauma itself.

D-dimer is a small protein molecule released after a blood clot is degraded by fibrinolysis. Therefore when a very low level is detected in the plasma of a patient suspected of having a venous thrombosis it makes the diagnosis very unlikely.

However, in patients who have raised plasma levels of d-dimer the diagnostic implications are more problematic because of the poor specificity of the test. Recent research has indicated that for at least one week following trauma or lower limb orthopaedic surgery the d-dimer result will be raised and that there is no statistically significant difference in the result of the test between these patients with and those without a confirmed DVT.

Patients suspected of having a PE should have an ECG performed. This will usually show a sinus tachycardia. In some cases it will also show the changes classically associated with PE; S1 Q3 T3. This is the presence of a deep S wave on lead I and a deep Q wave on lead III with an inverted T wave also on lead III. Remember however this is not often the case and many patients with a PE do not exhibit these changes. The ECG below shows the changes in leads I and III demonstrated by the arrows.

Fracture Specific Complications

Immediate:
- haemorrhage
- vascular injury
- neurological injury
- visceral injury

Early:
- compartment syndrome
- infection (worse if associated with metalwork)

Late:
- mal-union
- delayed and non-union
- avascular necrosis
- CRPS (complex regional pain syndrome)
- myositis ossificans
- joint stiffness

Immediate Complications

Haemorrhage

A closed long bone fracture such as a femoral fracture can cause blood loss of up to one litre, which on its own may be enough to cause circulatory compromise. Remember that this is true of closed injuries; just because you cannot see the blood loss does not mean it is not there.

<table>
<thead>
<tr>
<th>Blood Loss (ml)</th>
<th>Class 1</th>
<th>Class 2</th>
<th>Class 3</th>
<th>Class 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 750</td>
<td>Normal</td>
<td>Increased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>750 - 1500</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1500 - 2000</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 2000</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BP</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse (bpm)</td>
<td>&lt; 100</td>
<td>100 - 120</td>
<td>120 - 140</td>
<td>&gt; 140</td>
</tr>
<tr>
<td>Respiratory Rate</td>
<td>14 - 20</td>
<td>20 - 30</td>
<td>30 - 40</td>
<td>&gt; 40</td>
</tr>
</tbody>
</table>

The table above shows the classes of shock, with the expected physiological response. As can be seen a single femoral fracture could lead to class 2 shock and multiple long bone fractures
could easily lead to class 4 shock which carries a high risk of circulatory collapse and cardiac arrest. More detail on haemorrhagic shock is provided in the previous section on managing the trauma patient.

**Vascular Injury**

It is uncommon for a complete transection of a blood vessel to occur with a fracture. Compression of blood vessels by a fracture or dislocation does occur more commonly, which is why it is critical to examine the limb distal to an injury for its vascular supply. In cases of a displaced fracture or dislocation, prompt manipulation of the injured part to its anatomic location often resolves the problem.

The image below is a 3D reconstruction of a CT angiogram showing a significantly displaced surgical neck of humerus fracture causing compression of the axillary artery.

Prompt open reduction and internal fixation resulted in restoration of the distal vascular supply. If surgery is not an immediate option, an attempt to manipulate the fracture into an anatomical position should be made as this will often also restore circulation.

**Neurological Injury**

Actual nerve transection is again rare, but stretching over a bony edge in a fracture or dislocation is more common. This leads to a condition called neuropraxia. Nerve injuries can be classified according to the Seddon classification, which has three types – neuropraxia, axonotmesis and neurotmesis.

Common examples of neuropraxies include axillary nerve palsy (dislocation of the shoulder), radial nerve palsy (fracture of the midshaft of humerus), ulnar nerve palsy (elbow dislocation), sciatic nerve palsy (posteriorly dislocated hip) and common peroneal nerve palsy (fracture of the neck of the fibula or knee dislocation). Neuropraxies are usually reversible, although it can sometimes take many months before the function starts to return to normal.

**Visceral Injury**

In orthopaedics, pelvic fracture carry the highest incidence of visceral injury, with the bladder and urethra being most at risk. In this group, care should be taken with catheterisation and should not be undertaken by anyone other than a urologist when blood is seen at the urethral meatus.

Rib fractures are another common cause of visceral injury, causing pneumothorax or haemopneumothorax when the lung is penetrated. These injuries should be expeditiously managed by the insertion of a large bore chest drain.

The x-ray above shows a patient with a large left sided pneumothorax. The lung edge is marked by the dotted line. Note the lack of lung markings lateral to the line in contrast to the right side. Remember that the rib fracture itself is often not seen on plain x-ray.

**Early Complications**

**Compartment Syndrome**

Compartment syndrome is defined as an increase in pressure within a closed osteofascial compartment. Muscles are divided into separate compartments by membranes that join the bone to the subcutaneous fascia. Swelling that occurs after a fracture can lead to an increase in the pressure within these compartments. As the pressure rises, the capillary blood flow to the tissues decreases. Ischaemia results when the capillary pressure is less than the compartment pressure. After about six hours, the lack of perfusion leads to tissue necrosis.

Patients classically have severe pain that is out of proportion to their clinical findings, paraesthesia and an unremitting tight throbbing sensation in the affected limb. The best and earliest sign of compartment syndrome is extreme pain on passive stretching of the muscles within the affected compartment. Compartment syndrome should not be confused with an ischaemic limb, as the limb is commonly warm and red rather than cold and pale.

The common sites for compartment syndrome are the lower limb (especially with tibial fractures) and the forearm, though it can occur in any myofascial compartment. If you are called to see a patient in whom you suspect compartment syndrome, your initial management should be to elevate the
limb, remove all dressings and split any plaster (right the way down to the skin). If this fails to relieve the pain, then you should remove the plaster completely.

It is possible to measure compartment pressures directly using pressure probes, although these can be unreliable and the diagnosis of compartment syndrome really is a clinical one. If a probe is used then a patient should have their compartments decompressed if the difference between the compartment pressure and diastolic blood pressure is less than 30mmHg.

Infection

Infection is a potential disaster in the presence of a fracture. It will be covered in detail in the following section on ‘complications of surgical management’ but it can nonetheless occur in fractures which are treated conservatively. It is a particular problem in open fractures, where the fracture haematoma communicates with the outside.

Late Complications

Malunion

Malunion occurs when a fracture does go on to unite but not in an anatomical position, leaving an unacceptable degree of angulation, rotation or shortening. It is caused by late presentation, failure of reduction or loss of maintenance of a good reduction.

The x-ray below shows a distal radius fracture which was treated conservatively in a plaster but was not followed up after 1 week. There is a clear malunion resulting in radial shortening, loss of radial inclination and dorsal tilt (see the chapter on distal radius fractures). This resulted in persisting pain and a poor functional outcome.

Malunion can be a difficult concept as the degree of malunion which is ‘acceptable’ varies widely between fractures. This will be discussed in the following chapters specific to the various injuries sustained. In general rotational deformity is not acceptable in any injury, whether in an adult or a child. However, in a child a more significant angular deformity can be accepted as it will remodel to a degree as the bone grows.

Delayed and Non-union

Delayed union occurs when a fracture takes longer than expected to heal for an injury of its type. For example a closed tibial fracture would be expected to heal within 12 weeks. If healing has not occurred within 24 weeks then it could be reasonably be said to have delayed union.

If the bone fails to unite after twice the expected healing time for that injury (after 24 weeks in the tibia example) then the fracture is considered to have gone into a state of non-union. There is no exact definition in terms of time when a fracture should be considered to have a delayed or non-union but this is the definition used in my centre.

The etiology of delayed union and non-union is not fully understood but there are a number of identified risk factors:

- age
- lower limb > upper limb
- open fractures
- infection
- diabetes
- smoking
- poor blood supply

There are two types of non-union, hypertrophic and atrophic. Each has a characteristic X-ray appearance. When the bone ends look enlarged and rounded (like elephant feet) and appear dense and sclerotic this is called hypertrophic non-union. In these cases there is plenty of new bone formation, but the two ends do not unite.

The following x-ray shows an example of hypertrophic non-union. The x-ray on the left shows a midshaft tibial fracture which has been fixed with an intramedullary nail. The x-ray was taken two weeks post-op. On the right is the same fracture at 6 months. There is plenty of new bone formation (callus) but there is a visible persisting fracture gap.

Less commonly, the fracture ends become osteopaenic with no evidence of new bone formation leading to an atrophic non-union.
The following x-ray shows a conservatively managed tibial fracture at the time of presentation on the left and at 6 months on the right.

Note that there is no evidence of fracture callus and that the bone ends have become relatively osteopaenic.

Sometimes a ‘joint’ may form between two non-united fracture ends which is referred to as a *pseudoarthrosis*. When an established non-union has developed then usually the only way to create union is to operate or re-operate on the fracture using an implant to gain good compression often using bone graft to aid healing.

**Myositis Ossificans**
This is a condition where new bone forms in soft tissues following injury or surgery. There does not necessarily need to be a fracture as it can be seen after a joint is dislocated. It causes restricted, painful movement. The commonest site for this is the elbow. The exact cause is unknown but is thought to be due to calcification and then ossification of blood that collects during the initial trauma. The new bone may be excised surgically at a later date if necessary.

The following x-ray shows a patient who developed significant heterotopic ossification following an acetabular fracture which was fixed with the three screws that can be seen. The two arrows indicate the ring of bone formation around the femoral neck. This patient subsequently underwent excision of the new bone and a total hip replacement for the secondary arthritis which developed due to the initial fracture.

**Joint Stiffness**
All joints are susceptible to stiffness following injury but certain joints are more prone than others. The knee, shoulder and elbow are the most susceptible of the large joints but the small joints of the hand are particularly at risk. The proximal interphalangeal joint of the fingers is probably the least tolerant to injury and immobilisation.

**Complex Regional Pain Syndrome**
This is a collection of symptoms, including persistent pain, swelling, redness and sweating, thought to be due to abnormal sympathetic response to injury.

It is often not noticed until after the plaster has been removed, several weeks after the injury. For example, a small proportion of patients following a Colles’ fracture have swelling of the hands and fingers, the skin is warm, pink and glazed in appearance, movement is decreased and the wrist and hand are painful to touch. It can also be seen in the lower limb.

Although the condition is usually self-limiting, some patients find it quite disabling and need multidisciplinary care including pain specialists, therapists and surgeons. Guanethidine nerve blocks and sympathectomy can help in some cases. Symptoms can take up to two years to improve. It is particularly important to make an early diagnosis in patients with upper limb fractures as poor function of one of the upper limbs can be very debilitating.

**Avascular Necrosis**
Avascular necrosis (AVN) occurs when a fracture causes disruption to the blood supply of the bone leading to ischaemia and bone necrosis. There are certain bones which are particularly susceptible due to the configuration of their blood supply. The two most commonly encountered are the
femoral head after displaced intracapsular fractures and the proximal scaphoid after a displaced waist fracture. The x-ray below shows a normal hip and a hip with established AVN. Note the collapse and flattening of the femoral head with sclerosis (whitening) of the bone.

AVN is also seen as a spontaneous condition in the absence of a fracture. Conditions associated with AVN include infection, sickle cell disease, Perthes’ disease, corticosteroid use and alcohol abuse.