Compartment syndrome of the lower limb: how to diagnose it, assess it and not to miss it

Introduction

Acute compartment syndrome occurs when the tissue pressure exceeds the perfusion pressure within a muscle compartment, resulting in progressive tissue, muscle and neurological ischaemia. The original description of compartment syndrome and the subsequent contractures and nerve injury secondary to rising intracompartmental pressure is attributed to Richard von Volkmann in his 1882 paper describing this after a supracondylar fracture.

Compartment syndrome can affect any compartment, and the incidence varies depending on the population and the aetiology. A retrospective study from Scotland, at the Royal Infirmary of Edinburgh, found that the annual incidence of acute compartment syndrome of an extremity was 7.3 per 100 000 for men and 0.7 per 100 000 for women. The study found that 69% of all acute compartment syndrome cases were associated with fractures and 23.2% were associated with blunt soft tissue injury (McQueen et al, 2000).

There are various forms of compartment syndrome such as abdominal, upper limb or lower limb compartment syndrome. This article focuses on acute compartment syndrome of the lower limb, including the anatomy of the lower limb compartments, the causes of compartment syndrome in the lower limb and the assessment and possible management of this plastic or orthopaedic surgical emergency.

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What is acute compartment syndrome?

Acute compartment syndrome is a surgical emergency which occurs in the presence of raised pressure in an enclosed fixed space such as an osseofascial compartment, exceeding perfusion pressure and compromising the circulation and function of the tissues within the compartment (Pearce et al, 2002). This reduces capillary flow as well as venous return leading to venous congestion, oedema and inadequate tissue oxygenation, eventually leading to an end point that can compromise the arterial supply to the compartment contents.

The sequelae of this raised pressure could be temporary or permanent ischaemia to the muscles and nerves, which also furthers the oedema and pressure rise. Acute compartment syndrome differs from chronic compartment syndrome, also known as exertional compartment syndrome. This commonly affects athletes and military personnel by causing recurrent predictable pain which is commonly associated with exertion but can also occur during normal activities. This is not a surgical emergency but may follow a similar mode of treatment (Brennan and Kane, 2003).

Where does compartment syndrome most often occur?

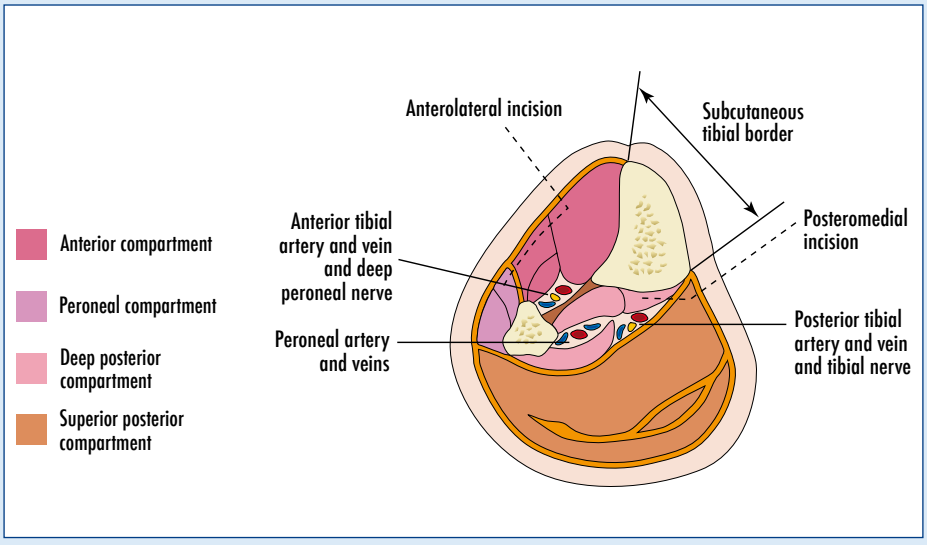
Although compartment syndrome can affect any compartment including the upper arm, forearm, hand, abdomen and buttock, it most commonly occurs in the lower limb, specifically the leg which has four compartments: anterior, lateral, superficial and deep posterior. The anterior compartment (the extensors) is most frequently affected (Simon and Koenigsknecht, 1982). The anterior compartment of the leg encloses the anterior tibial muscle, the extensor hallucis longus and the extensor digitorum longus muscles. Anterior compartment syndrome is characterized by anterior tibial pain, weakness of dorsiflexion of the ankle and toes and a variable degree of sensory loss over the deep peroneal nerve distribution (specifically reduced sensation in the first web space) (Rorabeck and Macnab, 1975).

Figure 1 shows the cross-sectional anatomy of the leg, and *Table 1* outlines the contents of the different compartments.

What are the causes of compartment syndrome?

Any internal or external factor that increases the pressure within a compart-

Figure 1. Cross-sectional anatomy of the leg with the four compartments and the surgical anatomy of performing a fasciotomy. Adapted from British Association of Plastic, Reconstructive and Aesthetic Surgeons/British Orthopaedic Association (2009).



ment can cause compartment syndrome. Internal causes include both open and closed fractures, usually secondary to high energy or crush injuries. Ischaemic reperfusion injury and rhabdomyolysis may also cause compartment syndrome. Some fracture configurations are more likely to lead to acute compartment syndrome than others.

A study by McQueen et al (2000) found that of 164 patients identified with acute compartment syndrome, 36% had a tibial diaphyseal fracture, which was the most likely type of fracture to lead to compartment syndrome. Furthermore, distal radial fractures comprised 10% of those affected by compartment syndrome, and diaphyseal fractures of the radius and ulna comprised 8%, while patients with femoral fractures and tibial plateau fractures each made up 3% of the compartment syndrome group in the study. Soft tissues injuries and crush syndrome combined made up 31% of the patients identified. The study also showed that men under the age of 35 years were at a significantly higher risk of developing compartment syndrome, probably because they were more likely to sustain high energy injuries, as well as having a larger muscle volume in a fixed fascial envelope that does not change after growth (McQueen et al, 2000). Haemorrhage secondary to direct trauma, anticoagulation therapy (Ebraheim et al, 1991), haemophilia or postoperative procedures on the limb can also lead to compartment syndrome.

Table 1. Compartments of the lower leg and their contents

Compartment	Contents
Anterior	Tibial nerve
	Sural nerve
	Tibialis anterior
	Extensor hallucis longus
	Extensor digitorum longus
	Peroneus tertius
	Anterior tibial artery
	Deep peroneal nerve
Lateral compartment	Peroneus longus
	Peroneus brevis
	Branches of the peroneal artery
	Superficial peroneal nerve
Posterior compartment: superficial	Gastrocnemius
	Soleus
	Plantaris
Posterior compartment: deep	Tibialis posterior
	Flexor digitorum longus
	Flexor hallucis longus
	Popliteus
	Posterior tibial artery
	Peroneal artery
	Tibial nerve
	Sural nerve

External causes of compartment syndrome include tight casts or splints, any external localized pressure or garment restriction, prolonged tourniquet application (Mubarak et al, 1978), insect or snake bites, and cases have been reported secondary to intravenous chemotherapy agents. Complications secondary to burn injuries can also lead to compartment syndrome (Shagden et al, 2010). Patient positioning during surgical intervention, e.g. in the lithotomy position or the lateral decubitus position (Smith et al, 1989; Simms and Terry, 2005; Kumar et al, 2007), may also increase the risk of acute compartment syndrome.

There is also exertional compartment syndrome which is associated with repetitive loading or microtrauma secondary to physical activity (Mouhsine et al, 2006). This is commonly a chronic compartment syndrome as opposed to the acute type dealt with in this article.

Pathophysiology

Osseofascial compartments have a fixed volume. An increase in fluid or external constriction can lead to an increase in pressure within these compartments with a resultant decrease in tissue perfusion until inadequate oxygen is available for cellular metabolism.

Tissue perfusion (TP) is equal to the difference between the capillary perfusion pressure (CPP) and the interstitial fluid pressure (IFP), represented as:

$$TP = CCP - IFP$$

Normal cellular metabolism requires an oxygen tension of 5–7 mmHg, which can be achieved with a capillary perfusion pressure of 25 mmHg and an interstitial tissue perfusion of 4–6 mmHg (Clayton et al, 1977). If the interstitial pressure exceeds the capillary perfusion pressure, the capillaries collapse, leading to muscle and tissue ischaemia.

A reduction in blood flow results in less oxygen to cells. This leads to release of vasoactive substances, e.g. histamine, serotonin, which increases endothelial permeability. This in turn causes continued fluid loss and continues to increase tissue pressure. This could eventually lead to reduced nerve conduction, a more acidic pH and may eventually lead to muscle necrosis secondary to prolonged ischaemia.

How is compartment syndrome diagnosed?

This is predominantly a clinical diagnosis. Clinically, the pain associated with compartment syndrome is disproportionate to the physical signs as well as the original injury. The pain is often described as burning, but also deep and aching that is worsening in nature. In anterior compartment syndrome the patient may complain of intractable pain in the front of the leg which is resistant to analgesia. Passive stretching of the affected muscles causes severe pain and there might be signs of paraesthesia. Paralysis, pulselessness and pallor are late signs. The four main signs of anterior compartment syndrome are:

1. Pain on passive plantar flexion of the foot
2. Pain increased by dorsiflexion of the foot against resistance
3. Reduced sensation in the first web space
4. Tenderness over the anterior compartment (Simon and Koenigsknecht, 1982).

The deep posterior compartment encloses the flexor digitorum longus, the tibialis posterior and the flexor hallucis longus. It also encloses the tibial nerve. The clinical picture of deep posterior compartment syndrome includes:

- Increased pain on passive extension of the toes
- Weakness of toe flexion and ankle inversion
- Reduced sensation over the sole of the foot (Simon and Koenigsknecht, 1982).

Although paraesthesia is an unreliable early symptom in compartment syndrome, loss of two-point discrimination is more relia-

ble and can be helpful in the diagnosis. Another important clinical sign is a firm, wooden feeling on deep palpation of the compartment. Furthermore, anticoagulation and bleeding disorders need to be delineated as they are a major contributing factor to the development of compartment syndrome.

It is important for the nurse performing observations to focus on several aspects. Particular attention should be paid to the pain score (noting any sudden changes) including pain associated with passive movement, colour and temperature, and sensation of the limb. The orthopaedic team need to be alerted if there are any changes or if the patient continues to take morphine-type analgesia with no resolution in the pain levels.

If needed, intracompartmental pressure can be measured (at the bedside) using a transducer or a needle attached to a manometer. A difference of 30 mmHg or less between the measured pressure and the diastolic pressure is a suggested threshold for surgical decompression (Whitesides et al, 1975; McQueen et al, 1996; British Association of Plastic, Reconstructive and Aesthetic Surgeons/British Orthopaedic Association, 2009). Measurement of intracompartmental pressure is not necessary if the clinical diagnosis is obvious. In practice it is reserved for uncooperative patients or equivocal cases, e.g. unconscious patient, children. The transducer measures the delta P value – the difference between diastolic pressure and the intracompartmental pressure. However, these measurements should always be taken into context of the clinical presentation.

The Stryker pressure monitor was first introduced in 1988 and has been demonstrated to yield accurate and reproducible results (Awbry et al, 1988). It is designed specifically for use with side-ported needles. When using this method, position the needle 45° to the site of insertion and apply pressure until the needle penetrates the skin. The tip of the needle should only be 1–3 cm (Seleno et al, 2012) below the surface of the skin for proper measurements. It comes with a pre-filled syringe and allows continuous pressure monitoring (Stryker, 2010).

An alternative method is to use central venous pressure monitors and intravenous pumps. Variations of this technique involve the use of directly connected central venous pressure monitors or intravenous pumps with pressure sensing capabilities (Seleno et al, 2012). Other commercial products that can be used to measure compartment pressure include the Compartmental Pressure Monitoring System manufactured by Synthes.

How would you treat compartment syndrome?

This is a surgical emergency. Split and remove the cast or plaster and bandages (if present) down to the skin to further assess the limb. The patient needs to be prepared for theatre for an urgent fasciotomy of all involved compartments (*Figure 2*).

The two incision technique is used to treat compartment syndrome. This is where double longitudinal incisions are made 2 cm from the anteromedial and anterolateral border of the tibia (the former to preserve the perforators at 5, 10 and 15 cm to allow for distally-based fasciocutaneous flaps). This technique allows access to all four compartments, does not compromise local flaps and also provides access to posterior tibial vessels (British Association of Plastic, Reconstructive and Aesthetic Surgeons/British Orthopaedic Association, 2009) (*Figure 3*).

- Decompress the superficial and deep posterior compartments through the medial longitudinal incision and the lateral and anterior compartments though the lateral longitudinal incision
- Leave open and revisit in 48 hours for inspection, debridement and primary

Figure 2. a. Schematic diagram of the pressure build-up in a muscle compartment during compartment syndrome (with specific focus on the neurovascular bundle) and (b) the release of pressure that occurs when a fasciotomy is performed to the specific compartment.

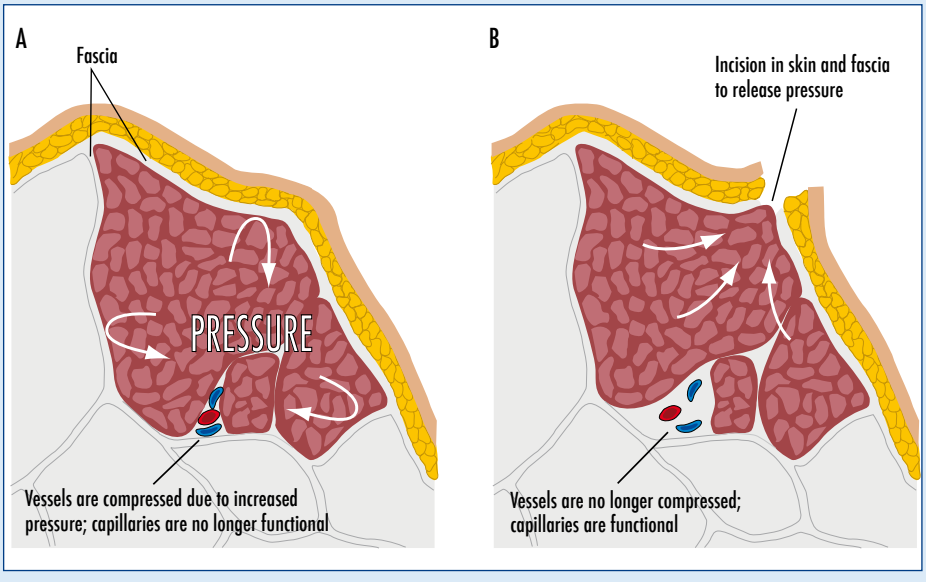
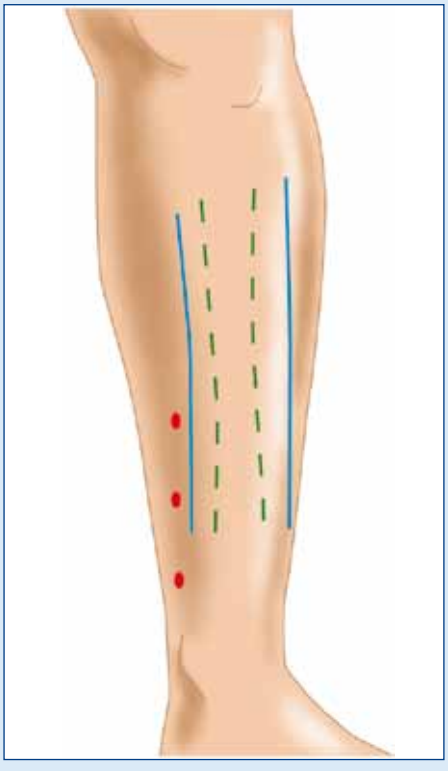


Figure 3. a. Incisions for a lower limb fasciotomy (marked in blue solid line) avoiding the three distal perforators of the posterior tibial vessels at 5 cm, 10 cm and 15 cm (marked in red) proximal to the medial malleolus. The margins of the subcutaneous border of the tibia are marked in the green dashed line.



delayed closure +/- skin grafting. Studies recommend application of a VAC dressing onto the wound which results in a higher rate of primary closure, decreases the time of hospitalization, allows early rehabilitation and increases patient satisfaction (Zannis et al, 2009).

Complications

If left untreated, compartment syndrome can lead to ischaemic necrosis of the muscles and nerves within that particular compartment, which leads to muscle and limb contractures, called Volkmann's ischaemic contracture. Established myoneural deficits seldom recover after late fasciotomies (Finkelstein et al, 1996). Patients can also experience chronic pain, significant scarring (some requiring flaps and reconstructive surgery) and nerve palsies. Infection is a further serious complication of compartment syndrome which can lead to significant morbidity and slower recovery. Severe cases of compartment syndrome can lead to amputation, rhabdomyolysis, renal failure, multiorgan failure and death.

Prognosis

The outcome of compartment syndrome relates to establishing the diagnosis and the time from injury to intervention. Fasciotomies performed within 6 hours achieved full functional limb recovery in a study by Rorabeck and Macnab (1975). When they are performed within 12 hours of acute onset, 68% achieved normal limb function, but only 8% did when the fasciotomy was performed after 12 hours (Sheridan and Matsen, 1976). In general, early diagnosis and the initiation of appropriate treatment results in a good limb outcome.

Conclusions

Acute compartment syndrome occurs when the tissue pressure exceeds the perfusion pressure within a muscle compartment, resulting in progressive tissue, muscle and neurological ischaemia. It is essential to identify it early and refer to the appropriate specialty in order to avoid the associated complications. BJHM

Conflict of interest: none.

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KEY POINTS

- Compartment syndrome is a surgical emergency.
- Compartment syndrome is predominantly a clinical diagnosis. The pain associated with compartment syndrome is disproportionate to the physical signs.
- A difference of 30 mmHg or less between the measured pressure and the diastolic pressure is a suggested threshold for surgical decompression.
- Early diagnosis and initiation of appropriate treatment results to good outcome.
- Urgent fasciotomy is the surgical intervention of choice.