Acute compartment syndrome

S. Singh\textsuperscript{a,}\textsuperscript{*}, S.P. Trikha\textsuperscript{b}, J. Lewis\textsuperscript{c}

\textsuperscript{a}7 Ardmay Gardens, Surbiton, Surrey, KT6 4SW, UK
\textsuperscript{b}Flat 3, 9 Grange Road, Kingston-upon-Thames, Surrey, KT1 2QU, UK
\textsuperscript{c}Worthing and Southlands NHS Trust, West Sussex, UK

Summary  Compartment syndrome can occur in any myofascial muscle compartment. If left untreated it can lead to ischaemic contractures and severe disabilities. A high index of suspicion is required in at risk cases. Compartment pressure monitoring is a useful adjunct in the diagnosis of raised compartment pressure especially when clinical assessment is difficult. The key to a successful outcome is early diagnosis and decompression of affected compartments.

Introduction

Compartment syndrome has been defined as 'a condition in which the circulation and function of tissues within a closed space are compromised by an increased pressure within that space'.\textsuperscript{1} The muscles and nerves of the extremity are enclosed in fascial spaces or compartments and are therefore susceptible to this condition. It is a surgical emergency which if not recognised and treated early can lead to ischaemic contractures, neurological deficit, amputation, renal failure and even death. Richard von Volkmann was the first to report this complication.\textsuperscript{2} He reported post-traumatic muscle contracture of acute onset with increasing deformity despite splinting and passive exercises.

Compartment syndrome is most commonly seen following trauma, but may occur after ischaemic reperfusion injuries,\textsuperscript{3} burns\textsuperscript{4} and positioning during surgery.\textsuperscript{5} Fractures of the tibial shaft and the forearm account for 58% of compartment syndromes.\textsuperscript{6} A high index of suspicion is required and early decompression of all at risk compartments is the treatment of choice.\textsuperscript{7-9}

Pathophysiology

The common pathogenic factor in compartment syndrome is increased pressure within a fascial compartment. Three theories have been proposed to explain the development of tissue ischaemia:

1. The increased compartmental pressure may lead to arterial spasm.\textsuperscript{10}

\textsuperscript{*}Corresponding author. Tel.: +44 07968 013803; fax: +44 208 390 7029.\hfill
\textsuperscript{E-mail addresses: sameer.singh@virgin.net (S. Singh), ptrikha@doctors.org.uk (S.P. Trikha), mrlewis@totalise.co.uk (J. Lewis).}

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The critical closing pressure theory states that because of the small luminal radius and the high mural tension of arterioles there must be a significant transmural pressure difference (arteriolar pressure minus tissue pressure) to maintain patency. If tissue pressure rises or arteriolar pressure drops so that this critical pressure difference does not exist then the arterioles will close.11

If tissue pressure rises then the veins will collapse due to their thin walls. If blood continues to flow from the capillaries the venous pressure will rise until it exceeds tissue pressure and patency of the veins is re-established. This leads to an increase in venous pressure and therefore reduces the arteriovenous gradient and as a result reduces tissue blood flow.12

The response of skeletal muscle to ischaemia or trauma is similar regardless of the mechanism of injury.13 When muscles become anoxic histamine-like substances are released and these dilate the capillary bed and increase endothelial permeability. Transudation of plasma occurs into the intramuscular compartment and this increases the pressure within the muscular compartment. To compensate the lymphatic drainage increases, however when this reaches a maximum the intracompartmental pressure (ICP) causes collapse of lymphatic vessels.14 Due to the high pressure in the arterial system there is continuing blood flow into the compartment and this increases the swelling and oedema. It is only in the late stages of compartment syndrome that arterial flow into the compartment is compromised.

The amount of pressure required to produce a compartmental syndrome depends on many factors, including the duration of pressure elevation, metabolic rate of tissues, vascular tone and the mean arterial pressure.

The data on effects of ischaemia to tissues are derived from research in which sudden, total ischaemia was imposed. Neural tissues demonstrate functional abnormalities (parasthesia and hyperesthesia) within 30 min of the onset of ischaemia, and irreversible functional loss after 12 h.12,15–17 Muscle shows functional changes after 2–4 h and irreversible changes beginning at 4–12 h.16–18 Ischaemia of 4 h gives rise to significant myoglobinuria, reaching a maximum at about 3 h although it can persist up to 12 h.19–22 Compartment syndromes lasting longer than 12 h produce chronic functional deficits, such as contractures, motor weakness and sensory disturbance.23,24 Any cause of increased compartmental pressure can result in a compartment syndrome. See Table 1 for a list of aetiologies as described by Matsen1.

### Diagnosis

#### Clinical

The key to successful treatment of acute compartment syndrome is early diagnosis and decompression of the affected compartments.1,7,9,23,24

The classical signs of impending compartment syndrome are pain, pallor, parasthesia, paralysis and pulselessness (The 5 p’s). However by the time all these symptoms have developed (especially pulselessness) the limb will be non-viable.

A high index of suspicion is required to make the diagnosis. Clinical diagnosis is made on a combination of physical signs and symptoms. These include pain out of proportion to the stimulus, pain on passive stretch of the affected muscle compartment, altered sensation, muscle weakness and tenderness over the muscle compartment. The symptoms and signs which are the most reliable in making the diagnosis are increasing pain, and pain on passive stretching of the muscle within the affected compartment.25–28 However these symptoms are subjective and impossible to elicit in the unconscious, non-cooperative patient and those

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**Table 1** Aetiology of compartment syndrome as described by Matsen.

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased compartment size</td>
<td>Closure of fascial defects, Tight dressings, Localised external pressure, Increased compartment content</td>
</tr>
<tr>
<td>Increased capillary permeability</td>
<td>Bleeding, Vascular injury, Bleeding disorder</td>
</tr>
<tr>
<td>Increased capillary pressure</td>
<td>Post Ischaemic swelling, Exercise, Seizure and eclampsia, Trauma, Burns, Orthopaedic surgery, Increased capillary pressure, Exercise, Venous obstruction, Muscle hypertrophy, Infiltrated infusion, Nephrotic syndrome</td>
</tr>
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who have had regional blocks. There has been concern raised with the use of patient controlled analgesia and regional anaesthesia in high risk cases.\textsuperscript{28,29}

**Intracompartmental pressures**

Pain can be unreliable especially in the trauma patient. It can range from being mild to severe, and in the unconscious patient important clinical symptoms and signs can be difficult to elicit. Techniques have been developed to measure ICPs.

**Technique for monitoring intracompartmental pressures**

Whitesides\textsuperscript{30} introduced a method for measuring ICP that required simple equipment available in most hospitals (Fig. 1). Using a needle, plastic tubing filled with saline and air attached to a mercury manometer they established tissue pressure measurement criteria as determinants of the need for fasciotomy. However this technique involved the injection of saline into the compartment and this may aggravate an impending compartment syndrome.

The slit and wick techniques require a polyethylene tubing connected to a pressure transducer. The tubing is filled with water and it is important that there are no air bubbles present within the tubing. The wick and slit catheter allow continuous monitoring of compartments, and have been shown to be more accurate than the needle manometer technique.\textsuperscript{31} However the end of the tubing in the fascial compartment may become blocked with blood leading to inaccurate readings. Due to these potential draw backs a solid-state transducer (STIC) intracompartment catheter has been developed.\textsuperscript{32} This has a multiperforated polyethylene tip with a STIC which can remain patent for up 16 h. The STIC catheter has been shown to be functionally superior to conventional systems (needle, wick and slit) and easier to assemble, calibrate, maintain and interpret.

The wick, slit and STIC methods require specialised equipment which may not be readily available, while the needle system as proposed by Whitesides\textsuperscript{30} can be constructed from equipment which is readily available in most hospitals.

If on clinical examination an obvious compartment syndrome is present pressure measurement may not be necessary. However it can be useful adjunct in the diagnosis of compartment syndrome especially in children, unconscious patients and those with equivocal clinical findings.

**At what pressure to decompress?**

The normal tissue pressure within closed compartments is about 0–10 mmHg. This pressure markedly increases in compartment syndrome. There is inadequate perfusion and relative ischaemia when the tissue pressure within a closed compartment rises to within 10–30 mmHg of a patient's diastolic blood pressure. Whitesides believed that fasciotomy is indicated when the tissue pressure rises to 40 mmHg in a patient with a diastolic pressure of 70 mmHg. Using these criteria no functional deficits developed in patients, and all showed conclusive

![Figure 1](https://example.com/figure1.png)
evidence of compartment syndrome at the time of operation. McQueen recommended a differential pressure (diastolic pressure minus ICP) of 30 mmHg as a threshold for fasciotomy in tibial fractures.

ICPs between 30 and 50 mmHg have been suggested that a fasciotomy should be performed. The lower level of 30 mmHg is most commonly used as when the tissue pressure rises above this the capillary pressure is insufficient to maintain capillary blood flow. It has also been shown that fascial compliance decreases sharply at an ICP of 33 mmHg as the fascia has reached its maximum stretch.35

It is important to state that tissue viability is dependant on adequate perfusion and blood flow within the microcirculation. Setting an absolute pressure ignores the role that blood pressure plays in maintaining adequate blood flow within a compartment. It has been shown that muscle damage occurring at a specific level relative to the blood pressure is more consistent that relying on a fixed compartment pressure.36

The diastolic pressure minus the ICP is called the delta pressure. The critical level has been found to range from 10 to 35 mmHg. The most commonly used delta pressure is 30 mmHg or less. In tibial fractures it has been shown that by using a delta pressure of 30 mmHg unnecessary fasciotomies can be avoided. No clinically significant complications were identified in patients with a delta pressure greater than 30 mmHg.

The ICP or delta pressure at one point in time does not necessarily confirm that a compartment syndrome is present. During intramedullary nailing there are short increases in ICP, however these are not always associated with clinical signs of compartment syndrome. The higher the ICP and the longer it is maintained the greater the muscle damage, however an ICP of 30 mmHg maintained for 8 h caused significant muscle necrosis in canines and biochemical changes have been observed in the experimental situation with a delta pressure of 20 mmHg for 4 h. When the delta pressure approached zero these changes were present in 2 h.38

As stated earlier the sooner the decompression the better the outcome. If decompression is delayed for more than 12 h permanent disability may occur, however if decompression is performed under 6 h of making the diagnosis a full recovery can be expected. However confirming the exact time of the start of compartment syndrome can be difficult.

Problems with interpreting pressures

The majority of compartment syndromes occur in the lower limb and hence the majority of the clinical studies relate to this region of the body. The site at which the compartment pressure is measured should be within a few centimetres of the maximal pressure as it cannot be assumed that the ICP equilibrates throughout the compartment. The pressure is always highest 5 cm from the fracture in tibial fractures, and therefore it is recommended that ICP should be measured as close to the site of injury as possible.

In the lower leg there are four fascial compartments and one or all of these may be involved in compartment syndrome. The highest pressures are recorded in the anterior compartment then the deep posterior compartment. It would seem logical therefore in tibial fractures to measure the pressure within 5 cm of the fracture and to monitor the pressure in the anterior tibial compartment. However other compartments may need to be monitored depending on the clinical picture.

Within the UK practices for monitoring ICP vary. In a postal questionnaire 46% of trauma centres had equipment available for monitoring compartment pressures, and 42% of respondents were unsure what ICP they would perform fasciotomies. Only 9% used a delta pressure of 30 mmHg as a guide to perform fasciotomies as suggested by Whitesides and McQueen. Failing to diagnose and treat a compartment syndrome urgently can be disastrous for patients. Pressure monitoring can be a useful adjunct to help confirm the diagnosis. McQueen et al. suggest monitoring all patients at risk as an aid to clinical diagnosis. Others have suggested that this can lead to over treatment. Certainly pressure monitoring should be used in unconscious patients, those who are difficult to assess and when equivocal clinical findings are present. All centres involved in trauma should have equipment available for monitoring compartment pressures and clinicians involved in trauma need to be aware of interpretation of these results.

Other methods for measuring compartment pressures

Near-Infrared Spectroscopy (NIRS)

NIRS is an optical technique that allows tracking of variations in the oxygenation of muscle tissue. The technique involves monitoring the absorption of light transmitted through muscle tissue at two distinct wavelengths. A change in the oxygenation state of haemoglobin results in opposite changes in the absorption of light. By calculating the differences in the absorption signal the device provides a continuous index of tissue oxygenation. It can be of
use in investigating chronic compartment syndrome in adults, as it can detect changes in relative oxygenation, but it is of little value in acute compartment syndrome as changes in the relative oxygenation may have already occurred.46

Laser Doppler flowmetry
This uses a flexible fibre optic wire which is introduced into the muscle compartment. The signals from this wire are recorded on a computer. It can be used as an adjunct in the diagnosis of chronic compartment syndrome,47 however it was suggested that further work needs to be carried out into the pathophysiology of chronic compartment syndrome and laser Doppler flowmetry needs analysis in larger population groups.

Treatment
Raised ICP threatens the viability of the limb and this represents a true management emergency. As stated earlier early diagnosis is the key to a successful outcome.

Removal of all dressing down to skin, followed by open extensive fasciotomies with decompression of all muscle compartments in the limb is the treatment of choice.

Experimental evidence shows that the circular cast can substantiate the adverse effects of raised ICP.48 Splitting of the cast on one side led to an average fall in ICP 30%, and 65% if split on both sides. Splitting of the padding led to a further fall in ICP by 10%. Complete removal of the cast reduced the pressure by another 15%.

In patients whom the diagnosis is being considered and in those in whom resuscitation is proceeding the following steps should be performed.14

1. Ensure the patient is normotensive, as hypotension reduces perfusion pressure and facilitates tissue injury,
2. Remove any circumferential or constricting bandages as these may increase ICP,
3. Maintain the limb at heart level as elevation reduces the arterio-venous pressure gradient.
4. Give supplemental oxygen to ensure optimal saturation.

Fasciotomies
If the tissue pressure remains elevated despite the above, and the clinical scenario indicates increased ICP adequate decompressive fasciotomies should be performed.

Several surgical approaches have been tried in the leg. The surgical goal is the prevention of permanent disability, and the adequacy of decompression should not be compromised by cosmesis or the number and lengths of incisions. It is essential to decompress all compartments at risk.

In the lower limb fibulectomy via a single lateral incision has been suggested, however this only allows limited views and an adequate release may not be achieved. A two incision approach allows safe access to all four compartments of the lower leg and is the treatment of choice. The deep posterior compartment has been neglected in descriptions of fasciotomies however this is the 2nd most commonly involved compartment and access can be gained behind the posteromedial border of the tibia in the distal third of the leg where the belly of flexor digitorum longus is exposed.

The technique of double incision fasciotomy is described below. It is important to perform a complete decompression and incisions less than 15 cm may result in inadequate decompression.49 In the emergency treatment of compartment syndrome there is no place for short cosmetic incisions.

Lower limb fasciotomy (Fig. 2)

Anterolateral incision. This incision allows approach to the anterior and lateral compartments of the leg. A 15–20 cm incision is placed halfway

![Figure 2](image_url) The safe incisions. These are designed to avoid the perforating arteries. The antero-lateral incision is 2 cm lateral to the medial border of the tibia. The posteromedial incision is 1 or 2 cm posterior to the medial border of the tibia.
between the fibula and the tibial crest. The skin edges are undermined. A short longitudinal incision is made over the muscle bellies allowing palpation of the intramuscular septum between the anterior and lateral compartments. By identifying the septum the superficial peroneal nerve can be identified adjacent to the septum where it crosses the junction of the middle and distal thirds of the leg. The anterior compartment fascia is opened throughout the leg by extending the first incision in the fascia (Fig. 3). It is important not to damage the superficial peroneal nerve in the distal third of the wound. The peroneal compartment is decompressed by incising the fascia in line with the fibular shaft posterior to the intermuscular septum. Proximally the incision is directed to the fibular head and distally to the lateral malleolus remaining posterior to the superficial peroneal nerve.

Figure 3 Anterior and peroneal compartment decompression (ac—anterior compartment; lc—lateral compartment). The fascia is shown in dark grey.

Posteromedial incision. This incision is used to decompress the superficial and deep posterior compartments of the leg. It is placed 2 cm posterior to the posterior tibial margin and is about 15–20 cm long. Care should be taken to avoid damage to the saphenous nerve and vein and they should be retracted anteriorly. The superficial posterior compartment is decompressed first, and the fascia is incised throughout its length (Fig. 4). The Achilles tendon helps to identify this compartment. The fasciotomy is extended distally as far as the medial malleolus. The deep posterior compartment is then released by incising the fascia distally and then proximally under the bridge of soleus. It may be necessary to detach the soleus from the back of the tibia.

A technique for forearm fasciotomy is now described.

Forearm fasciotomy

A single incision can be used to decompress the volar aspect of the forearm (Fig. 5). It is similar to the volar approach to the radius as described by Henry. It begins 1 cm proximal and 2 cm lateral to the medial epicondyle. It is carried obliquely across the antecubital fossa and over the volar aspect of the mobile wad of three muscles (brachioradialis, extensor carpi radialis longus and extensor carpi radialis brevis). It is curved medially reaching the midline at the junction of the middle and distal third of the forearm. It is continued straight distally to the proximal skin crease over palmaris longus. The incision is curved across the wrist crease to the mid palm area. The median nerve should be decompressed at the carpal tunnel. In cases of median nerve symptoms the median nerve should also be explored in the proximal forearm. The median nerve can be constricted at the proximal end of pronator teres and at the proximal edge of flexor digitorum superficialis.

The dorsal muscle compartment can be released by a single incision. This begins 2 cm distal to the lateral epicondyle and carried distally to the wrist. The skin edges are undermined and the dorsal fascia incised directly in line with the skin incision.

Foot fasciotomies

Excessive bleeding and oedema can produce compartment syndromes in the closed spaces of the foot. Foot compartment syndrome should be suspected in all crushing and high energy foot
injuries. With crush injuries of the foot Myerson found acute compartment syndrome in 16 of 58 patients. Tense swelling of the foot should alert the clinician to this possibility, particularly because pain on passive stretch of the toes and the presence or absence of pedal pulses are less reliable indications of compartment syndrome in the foot. There are a number of different approaches to decompress foot compartments. A dorsal approach along the 2nd and 4th metatarsals is simple to perform and provides effective decompression of all four compartments. Associated Lisfranc injuries and metatarsal fractures can also be stabilised via this approach.

Closure of fasciotomy wounds

After decompression of fascial compartments the wounds are left open and sterile dressings are applied. Delayed primary closure can be performed when swelling has subsided, however this may be difficult due to skin retraction and oedema. If the wound edges cannot be approximated without tension, skin grafting may be required.

Various methods have been described using the elastic properties of the skin to aid fasciotomy closure.

An elastic vessel shoelace can be applied with the staples at the side of the wound. This can be gradually tightened without the need for anaesthesia, providing gradual closure of the fasciotomy wound. Closure of the wound takes about 10 days. There are some commercially available devices to aid fasciotomy closure. The Suture Tension Adjustment Reel (STAR) is placed parallel to the wounds at the time of fasciotomy, and when the swelling has subsided the reels are tightened to gradually close the wound. This method requires 2–4 days of bedside tightening for wound closure.

Split skin grafting can lead to a poor cosmetic result, with insensate skin and donor site morbidity. Delayed primary closure using the skin’s elasticity provides a more cosmetically acceptable outcome for the patient but requires greater nursing care. However a poor cosmetic result is preferable to the outcome of a missed compartment syndrome.

Intramedullary nailing

Over the last 2 decades intramedullary nailing of tibial fractures has increased. Initially there was concern that nailing may increase ICPs and precipitate compartment syndrome and it was thought that nailing should be delayed for up to 7 days to allow the swelling to subside. Further research in this area has shown that during reaming the pressure may rise to 180 mmHg, however this high ICP fell back to normal after removing the reamer. The application of traction also increases ICPs but these immediately dropped with release of the traction. Despite high pressures being reached during the reduction of tibial fractures and during reaming no patients in the study developed any sequelae of compartment syndrome. Transient increases in compartment pressures seem to be well tolerated and return back to normal after the stimulus is removed.

Controversy still exists if monitoring should be performed during intramedullary nailing. McQueen advocates routine monitoring of all patients with tibial fractures if facilities are available. Others have suggested that this may lead to over treatment and unnecessary fasciotomies.

Conclusion

Compartment syndrome can have disastrous consequences if not recognised and treated appropriately. In conscious patients the diagnosis can be made by careful examination of the patient. Invasive monitoring is a useful adjunct especially in unconscious patients and those who are difficult to assess. As the tissue pressure rises the viability of the cells are threatened. The tissue pressure level at which perfusion threatens cell viability varies
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according to the age and circulatory status of the patient. A delta pressure (Diastolic pressure-Tissue pressure) of 30 mmHg or less is an accepted level that fasciotomy should be performed.

References