

# Acute limb compartment syndromes

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## Key points

Compartment syndrome (CS) is a medical emergency, which needs to be recognized by those involved with the provision of anaesthesia, critical care, and pain management.

Interventions by anaesthetists have the potential to mimic or mask the signs and symptoms of a CS.

Delayed diagnosis and treatment leads to devastating long-term problems for the patient, and is a potential source of litigation.

Clinicians need knowledge of this condition, awareness of at risk patients, and a high index of suspicion to make an early diagnosis.

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Compartment syndrome (CS) is a serious limb-threatening, and rarely life-threatening, condition, which can cause significant disability if not treated early. It is a challenging diagnosis requiring a *high index of suspicion* and should be anticipated after any limb injury or surgery that predisposes to CS. Despite clinical signs being unreliable, such assessment remains the key trigger to considering further investigations or interventions. It requires prompt diagnosis and urgent management as delays in treatment can result in limb damage, amputation, and even death.

## Definition

A CS is a condition in which increased pressure within a closed compartment compromises the circulation and function of tissues within that space.

Acute limb CS refers to acutely raised pressures in an osseofascial compartment of a limb, which is the focus of this article. It is most commonly seen in the calf or forearm, but occasionally occurs in the upper arm, thigh, buttock, foot, or hand. Other clinically relevant 'compartments' include the cranium, eye, spinal column, abdomen, chest, and pericardium. These are not further discussed here, but similar principles of management apply.

Hippocrates first described the dangers of raised compartment pressures and its sequelae in 400 BC. Volkmann recognized this syndrome in 1881, and described the ischaemic contracture that commonly occurs in untreated patients.

There is also a chronic exertional CS, which mainly affects athletes and is characterized by muscle pain on vigorous exercise with measured elevated pressures within affected compartments. Elective closed fasciotomy (i.e. via a small incision) is an effective treatment.

## Aetiology

A CS can be caused by either an increase in tissue volume within a compartment, thereby

increasing the pressure, or externally applied pressure compressing a compartment. The commonest cause is trauma, usually after a fracture, in male patients <35 yr old.<sup>1</sup>

A CS can be classified by causation, although in many patients a combination of factors contributes (Table 1).

## Trauma

Trauma resulting in limb injury, with or without a fracture, particularly after accidents with long extraction times, can lead to a CS. Both closed and open fractures are implicated, as the compartment may not necessarily be decompressed through an open wound. High energy fractures of the tibial shaft, radius, and ulna are the most commonly associated bony injuries.<sup>1</sup> In the leg, the anterior compartment is most commonly affected, whereas in the forearm it is the flexor compartment as these anatomical spaces have less potential to accommodate tissue swelling. Excessively tight bandages or plaster casts may also cause a CS.

Soft tissue injury without a fracture can result in a CS and is particularly likely after crush injuries and burns. Traumatic rhabdomyolysis can be caused by prolonged mechanical compression of skeletal muscle resulting in hypoperfusion and hypoxia of the muscle. The risk of developing a CS is increased in patients taking anticoagulants or those with a bleeding disorder.<sup>1</sup>

## Vascular

Both arterial and venous injuries can lead to CS. Any procedure that results in delayed limb reperfusion, such as embolectomy, thrombolysis, and bypass surgery, can cause CS, especially after prolonged periods of ischaemia. The increased inflammation that follows successful reperfusion of the ischaemic limb may lead to sufficient tissue swelling and oedema to result in CS. This is more common in patients of advanced age, with acute ischaemia and in the presence of hypotension. Haemorrhage into

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**Table 1** Common and rarer causes of a CS

Orthopaedic	Fractures and orthopaedic surgery
Soft tissue	Crush injury Circumferential burns
Vascular	Ischaemia–reperfusion injury Arterial and venous injuries Phlegmasia caerulea dolens (venous gangrene)
Iatrogenic	Drug or fluid extravasation Surgical positioning, especially prolonged lithotomy Large bore femoral vessel cannulation (e.g. intra-aortic balloon pumps or bypass circuits) Pneumatic anti-shock garments
Other causes	Recreational drugs and prolonged immobility resulting in ischaemia–reperfusion of limb or through intra-arterial injection Electrical injuries Venomous snakebite Heat stroke Sepsis, DIC e.g. purpura fulminans in meningococcal sepsis

a compartment, especially in patients on (excess) anticoagulants can also cause CS. The severe venous occlusion that occurs in phlegmasia caerulea dolens (venous gangrene) may result in sufficiently raised compartment pressures to cause a CS.

### Iatrogenic

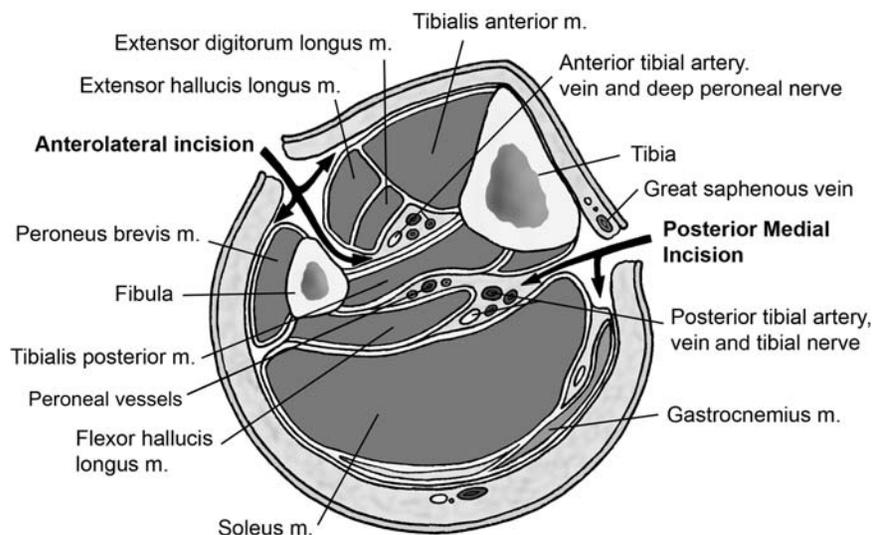
A CS may complicate a variety of medical interventions. Compartment pressures may be increased from accidental infiltration of fluid from misplaced IV cannulae, intraosseous needles, or joint irrigation systems. The use of arterial tourniquets may cause a CS after excessive inflation pressures and prolonged ischaemic times.<sup>2</sup> The mechanism of ischaemia–reperfusion is also responsible for a CS after prolonged surgical procedures in the lithotomy (Lloyd–Davies or Trendelenburg) position. Prolonged leg elevation in this position, especially in the presence of hypotension, may result in low-grade calf ischaemia that on reperfusion results in a

CS. Similarly, large cannulae within the femoral arteries required for bypass surgery or aortic balloon pumping are unusual, but well-recognized causes. Extrinsic compression from pneumatic anti-shock garments or a surgeon's weight may also play a role. Although there are reports of calf intermittent pneumatic compression garments causing CS, in these cases there were multiple risk factors. It is possible that they may help prevent it because of the redistribution of pressure points with the inflation–deflation cycle.

A CS complicating orthopaedic or vascular surgical procedures is generally well recognized by members of the healthcare team and is usually detected promptly. However, in the rarer causes of CS, which present to clinicians unfamiliar with the problem, there are frequently delays in recognition and treatment (e.g. leg CS after prolonged immobility after collapse, where recognition may be delayed as staff have less experience of this condition and a lower index of suspicion).

### Pathophysiology

Muscles, nerves, and blood vessels lie within fascial compartments (Fig. 1). After direct injury, ischaemia–reperfusion or fluid extravasation (see causes above) the pressure within these compartments may rise, reducing perfusion, and leading to local ischaemia of muscles and nerves. The ischaemia results in tissue membrane damage and leakage of fluid through capillary and muscle membranes, with a subsequent increase in tissue pressure. This raised tissue pressure results in venous outflow obstruction and increased venular pressure (normally 4–7 mm Hg). The consequent increase in capillary pressure induces a vicious cycle of increased fluid transudation, greater tissue swelling, and rise in intra-compartmental pressure. Eventually, as intra-compartmental pressure approaches capillary pressure, microcirculatory perfusion



**Fig 1** Cross-section of mid calf, showing osseofascial compartments with sites of fasciotomy incisions indicated.

ceases and, unless compartmental pressure is relieved, tissue infarction commences.

If reperfusion occurs, tissue damage initiated in the ischaemic phase is continued. The damaged membranes continue to leak, increasing oedema formation and thus pressure in the enclosed compartment. If the pressure is not relieved within a few hours of onset, irreversible changes will occur with muscle necrosis and contracture, and nerve and vessel damage. The time required to develop irreversible injury to nerves and muscle varies depending on the site, but within the leg may occur as early as 4 h after the onset of injury.

### Prevention

Limb ischaemia is preventable in some situations (e.g. by restricting the time legs are elevated in the lithotomy position, or release of a tourniquet). Legs should be lowered from lithotomy and the tourniquet deflated intermittently (every 2–3 h). Careful patient positioning and avoidance of perioperative hypotension should further reduce the risk of a CS developing. This requires effective communication and co-ordinated action between anaesthetists, surgeons, and other theatre staff.

### Clinical diagnosis

Severe *pain* over the affected compartment, often disproportionate to the apparent injury, is the cardinal symptom of a CS. This is aggravated by passive stretching of the involved muscles. *Paraesthesia*, especially loss of two-point discrimination in the distribution of the nerves traversing the compartment, is cited as characteristic. Weakness or *paralysis* of the limb is a late sign. However, pain is subjective and variable, so may be an unreliable symptom, and may be absent in an established CS.

The signs include tense and tender swelling over the compartment and dysfunction of the nerves traversing the compartment. *Pulselessness* is uncommon and implies a late stage as by the time compartment pressures have risen to occlude the traversing arteries, extensive muscle and nerve injury will have occurred. A high index of suspicion is needed, and repeated assessment and observation are required. It should be appreciated that not all of these signs are required for the diagnosis to be made, and the clinical findings can change as the syndrome progresses.

### Pitfalls in diagnosis

Many of the above findings may not be present until late in the disease process when damage is irreversible. Data from clinical studies demonstrate that clinical findings have a low sensitivity for diagnosing CS, suggesting that clinical features are more useful by their absence in excluding the diagnosis than their presence in confirming it.<sup>3</sup>

A high degree of suspicion is essential if an early diagnosis is to be made. Diagnosis can be especially challenging in children

**Table 2** Common causes for diagnostic confusion in CS

Type	Causes
Patient-related	Extremes of age Acute confusional state Spinal cord injury Multiple injuries Drug or alcohol overdose
Critical care-related	Sedation/analgesia
Anaesthetic-related	Recovery from general anaesthesia Regional anaesthesia Analgesia (e.g. PCA)

and patients with neurological compromise or altered mental status (Table 2).

Increasing analgesic use and increasing or breakthrough pain and pain remote to the surgical site have been identified as important early warning signs of an impending CS with a working epidural.<sup>4</sup> In sedated ICU patients, difficulties with pain control or sedation may be the only clinical indicator in this group. Obtunded patients after drug or alcohol overdose and prolonged limb compression may have soft tissue injury and develop a CS.

In a recent review of the effect of postoperative analgesia on the diagnosis of a CS, in 32 out of 35 patients the signs and symptoms described above were present in the presence of epidural analgesia. Eighteen patients had also documented breakthrough pain.<sup>5</sup> Similarly, in a national Paediatric Epidural Audit, four incidents of a CS were reported in 10 633 epidurals, but the presence of a working epidural did not mask or delay the diagnosis.<sup>6</sup> A recent review of paediatric patients showed no clear evidence that an epidural delayed the diagnosis of a CS.<sup>4</sup> Patient-controlled analgesia (PCA) with morphine has been implicated in a delayed diagnosis of a CS in several case reports, but to date there have been no case reports implicating peripheral nerve blocks.<sup>5</sup> Nevertheless, caution needs to be taken with all these analgesic techniques that could potentially mimic or mask developing signs of a CS.

### Making an early diagnosis

Awareness among medical and nursing staff of the possibility of the disorder in at risk groups and its clinical presentation are the most important factors contributing to an early diagnosis. It should be actively considered and excluded in any patient at risk, especially if obtunded. Those suspected of having CS should have prompt compartmental pressure monitoring.<sup>1</sup> Post-operative monitoring and documentation may be formalized by the use of a designated orthopaedic observation chart, including pain score, analgesic requirements, and neurovascular signs. Risk assessment tools could help identify those with impending CS, along with protocols identifying triggers for urgent medical review and management. The presence of increasing pain or pain remote to the surgical site should be a 'red flag' to medical staff.

Newer prolonged operative procedures such as robot-assisted laparoscopic prostatic surgery have been associated with CS of the

calves after prolonged lithotomy. Consequently, such patients should routinely be checked for calf tightness and tenderness.

It has been previously advocated that regional anaesthesia should be avoided in patients at risk of developing a CS, as analgesia may mask early signs of the syndrome. However it would seem that there is no convincing evidence for this *providing* patients are adequately monitored. Epidural anaesthesia should also be supervised by an acute pain service to help identify potential complications of its use. Avoidance of dense sensory or motor block and unnecessary sensory blockade of areas remote to the surgical site allow full assessment of the patient and limit the potential for delay in the diagnosis of a CS.

## Investigations

The diagnosis of CS is usually a clinical one, although compartmental pressure monitoring has been recommended for use in high-risk patients as an adjunct.<sup>1</sup> The normal pressure in the muscle compartments is <10–12 mm Hg. Compartmental pressure monitoring can be performed by using a pressure transducer attached to a needle or cannula which is placed into the suspect compartment and the transducer zeroed to the level of the needle. Other methods of measurement are documented and all are liable to inaccuracies, although none are associated with any significant complications.<sup>7</sup> All compartments in a limb suspected of having CS should be measured. The pressure threshold above which a diagnosis can be made is controversial, but it is the compartmental perfusion pressure which is deemed important. This is the difference between the diastolic arterial pressure and the intra-compartmental pressure. It has been recommended that the diagnosis is confirmed and fasciotomy is required if the compartmental perfusion pressure is  $\leq 30$  mm Hg.<sup>8</sup> However, muscle compartment pressures have also been advocated with fasciotomy recommended if the compartment pressure is  $>30$  mm Hg.<sup>9</sup> Other investigations show promise in monitoring for compartmental ischaemia and clearly a technique for early and reliable diagnosis is required. However, these methods are not yet widely available and all have limitations. They include near-infrared spectroscopy, infrared imaging,<sup>10</sup> ultrasound, and MRI.<sup>8</sup> Serum creatine phosphokinase or myoglobinuria has been used as an indicator of CS as it reflects muscle necrosis, but is not helpful for early diagnosis and may reflect muscle damage from one or more sites around the body.

## Treatment

CS must be treated urgently and surgical decompression is the mainstay of therapy. The goals of treatment are to decrease tissue pressure, restore blood flow, and minimize tissue damage and related functional loss. While it is difficult to identify the precise time at which CS develops, the incidence of complications is related to the time from diagnosis to fasciotomy. While surgery is organized, external pressure should be released by removing all constricting bandages, occlusive dressings, or casts encircling the

limb. The limb should be kept at the level of the heart and elevation avoided as this may further decrease perfusion below critical levels. Patients require urgent decompression, and any resuscitation should be performed during the procedure rather than delay this. Intra-operatively, hypotension should be avoided.

Open fasciotomy, incising both skin and fascia, is the most reliable method for adequate compartment decompression (Fig. 1). Muscle will be seen to bulge out of released compartments (Fig. 2). Obvious dead muscle will need debridement.

After this, the limb should be splinted to prevent contractures and any fracture stabilized to prevent further bleeding. Further inspection of exposed muscle and aggressive debridement of necrotic muscle may be required to ensure maximal salvage of function. Bleeding may be heavy during the procedure and continue into the postoperative period. Other local complications after fasciotomy include further muscle necrosis, nerve injury, and occasionally soft tissue and bone infection. The fasciotomy wounds may be later closed directly once the swelling has receded, left to heal by secondary intention, or covered with split skin grafts. The patient may also develop the systemic effects of massive rhabdomyolysis with hyperkalemia, myoglobinuria, acute renal failure, and a systemic inflammatory response syndrome with cardiovascular and respiratory failure.<sup>11</sup> Early amputation may be life-saving in this situation.

Hyperbaric oxygen therapy shows promise as an adjunct to fasciotomy or when immediate surgical treatment is not possible. However, this needs further evaluation and is seldom practicable locally. Mannitol may reduce the incidence of CS after revascularization by acting as a free-radical scavenger and reducing oedema. Again, while it may be beneficial, more human studies are required.

In phlegmasia caerulea dolens (venous gangrene), early aggressive limb elevation is essential along with anticoagulation.



**Fig 2** Lateral and medial fasciotomy incisions of the calf after a motorcycle accident. The diagnosis was suspected by intensive care staff after assessment of pressure by manually squeezing the calf, and confirmed and treated by vascular surgeons. There was an open femoral fracture, which had been externally fixed. This procedure was performed 4 days after the initial injury. Note bulging muscles which were viable and survived intact. Later skin grafting was required for closure. Reproduced with patient's permission.

Delayed diagnosis is likely to result in major functional morbidity for the patient from permanent nerve injury, poor muscle function, and ischaemic contractions. Limbs with extensive muscle infarction may ultimately require amputation. It should be noted that perceived delays in recognition and surgical management are a frequent cause of litigation.

### Conclusion

Although primarily a surgical diagnosis, all involved with anaesthesia, critical care, and acute pain services should be aware of this condition and have a low threshold for its potential diagnosis and early referral to an appropriate surgical speciality (typically vascular, orthopaedic, or plastic surgery).

### Conflict of interest

None declared.

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Please see multiple choice questions 20–22.