Emergency surgery 3

Diagnosis and treatment of acute extremity compartment syndrome

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Acute compartment syndrome of the extremities is well known, but diagnosis can be challenging. Ineffective treatment can have devastating consequences, such as permanent dysaesthesia, ischaemic contractures, muscle dysfunction, loss of limb, and even loss of life. Despite many studies, there is no consensus about the way in which acute extremity compartment syndromes should be diagnosed. Many surgeons suggest continuous monitoring of intracompartmental pressure for all patients who have high-risk extremity injuries, whereas others suggest aggressive surgical intervention if acute compartment syndrome is even suspected. Although surgical fasciotomy might reduce intracompartmental pressure, this procedure also carries the risk of long-term complications. In this paper in *The Lancet* Series about emergency surgery we summarise the available data on acute extremity compartment syndrome of the upper and lower extremities in adults and children, discuss the underlying pathophysiology, and propose a clinical guideline based on the available data.

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See Editorial page 1212

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Introduction

Acute extremity compartment syndrome is a surgical emergency for which timely diagnosis is essential. Although described around 130 years ago,[1] this disorder remains challenging to diagnose and treat effectively. Acute extremity compartment syndrome is defined similarly to many other compartment syndromes: an increase in intracompartmental pressure causing a decrease of perfusion pressure, leading to hypoxaemia of the tissues. Decreased tissue perfusion can lead to irreversible necrosis that might result in functional impairment, loss of limb, and, in rare cases, death. Acute extremity compartment syndrome is most frequently seen after a traumatic event, but in up to 30% of cases occurs without any evidence of fracture.[2,3] Other disorders that can cause acute extremity compartment syndrome are thermal injuries (especially when circumferential), lithotomy positioning during surgery, or constricting casts or wraps. Acute extremity compartment syndrome has also been documented in association with nephrotic syndrome,[4] rhabdomyolysis,[5] bleeding disorders,[6] and iatrogenic factors, such as accidental pressurised intravenous or extravenous infusion of an agent.[7] Furthermore, infections, especially with *Streptococcus* spp, can cause acute extremity compartment syndrome.[8,9] Therefore, almost any physician could see a patient with acute extremity compartment syndrome.[10–13]

Treatment with fasciotomy is well accepted, but delays in surgical or non-surgical treatment can result in permanent disability. All health-care professionals must, therefore, be familiar with the current standard of diagnosis and principles of the treatment of acute extremity compartment syndrome.[14]

Pathophysiology

The major muscle groups and neurovascular structures in the extremities are separated into compartments by dense connective tissue called fascia. The biomechanical functions of fascia are providing attachment sites for muscles, maintaining the position of muscle groups during motion, and improving the mechanical advantage of muscle during contraction.[15] Deep investing fascia is innervated and might also play a part in muscle coordination and proprioception.[16] The dense fibrous nature of fascia creates a defined anatomical space with low compliance.

Acute extremity compartment syndromes can generally be classified as primary (direct limb-related injury) or secondary (non-limb-related injury). Medical management of underlying causes of secondary acute extremity compartment syndrome with adequate crystalloid-sparing resuscitation, haemorrhage control, or both, might be crucial in preventing its development. Primary and secondary causes affect hydrostatic pressure within a compartment (figure 1) and are frequently seen in combination.

Search strategy and selection criteria

We searched PubMed, Embase, and Cochrane Library for articles published within the previous 10 years. We used the search terms "acute compartment syndrome" and "fasciotomy" plus optional terms "treatment outcome", "delayed diagnosis", and "pathophysiology". Reports providing high-level evidence were preferably selected. Dependent on the number of results retrieved from each database, the respective search strategy was modified to include more terms to narrow or broaden the desired results. We reviewed article titles and abstracts for relevance, and manually searched the reference lists of selected articles to identify commonly referenced and seminal older articles. Comments from peer reviewers of the report were also considered in the selection of relevant articles.

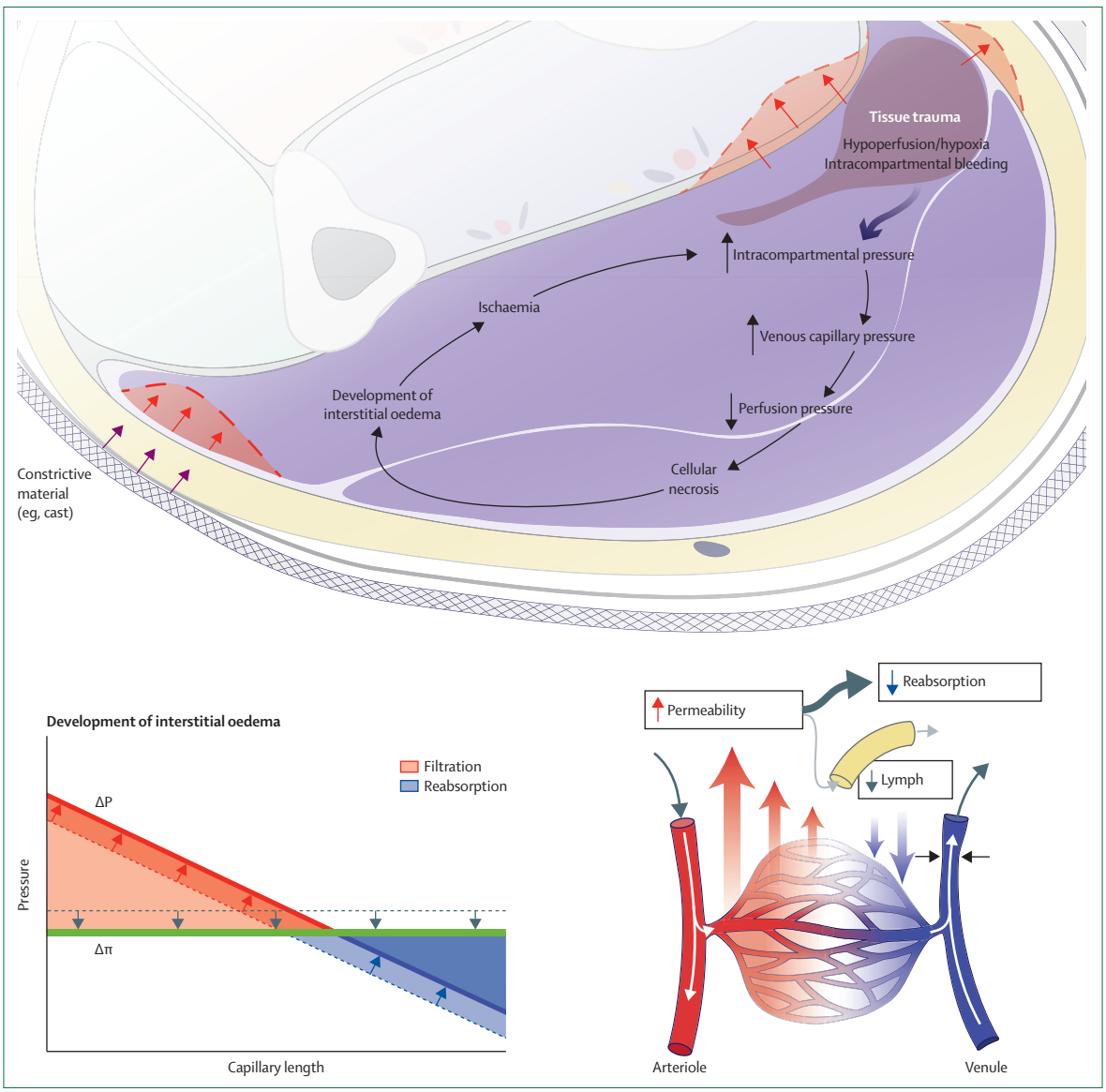

Figure 1: **Pathophysiology of acute extremity compartment syndrome**
ΔP=hydrostatic pressure, defined by the difference of the capillary pressure – the interstitial pressure. Δπ=colloid osmotic pressure.

Intrinsic causes of acute extremity compartment syndrome are tissue injury caused by a direct traumatic event or tissue ischaemia and reperfusion.[17] Group A streptococcal infections can be another cause, although the mechanisms are not fully understood. Local swelling due to pyrogenic exotoxin that functions as a super antigen is thought to lead to direct muscle injury.[9] Precapillary vasodilation in the arteriole system caused by muscle injury, along with collapsing venules and increased permeability of the capillary bed, leads to increased net filtration and raised interstitial fluid pressure in traumatised tissues. Interstitial fluid pressure is normally lower than 10 mm Hg. As it increases, adequate perfusion to tissue becomes decreased. Once perfusion reaches pressure a critically low level, tissue hypoxaemia ensues (figure 1).

The combination of hypoxia, increase in oxidant stress, and development of hypoglycaemia in tissue cause cell oedema due to a shortage of ATP and shutdown of the sodium–potassium ATPase channels that maintain physiological cellular osmotic balance. The subsequent loss of cell-membrane potential results in an influx of chloride ions, which leads to cellular swelling and cellular necrosis (figure 2). The resulting increase in tissue swelling further worsens the hypoxic state and creates a positive feedback loop.

Another cause of acute extremity compartment syndrome and compromised function is reperfusion injury. Once vascularity is restored after an extended period of ischaemia, the production of oxygen radicals, lipid peroxidation, and calcium influx leads to

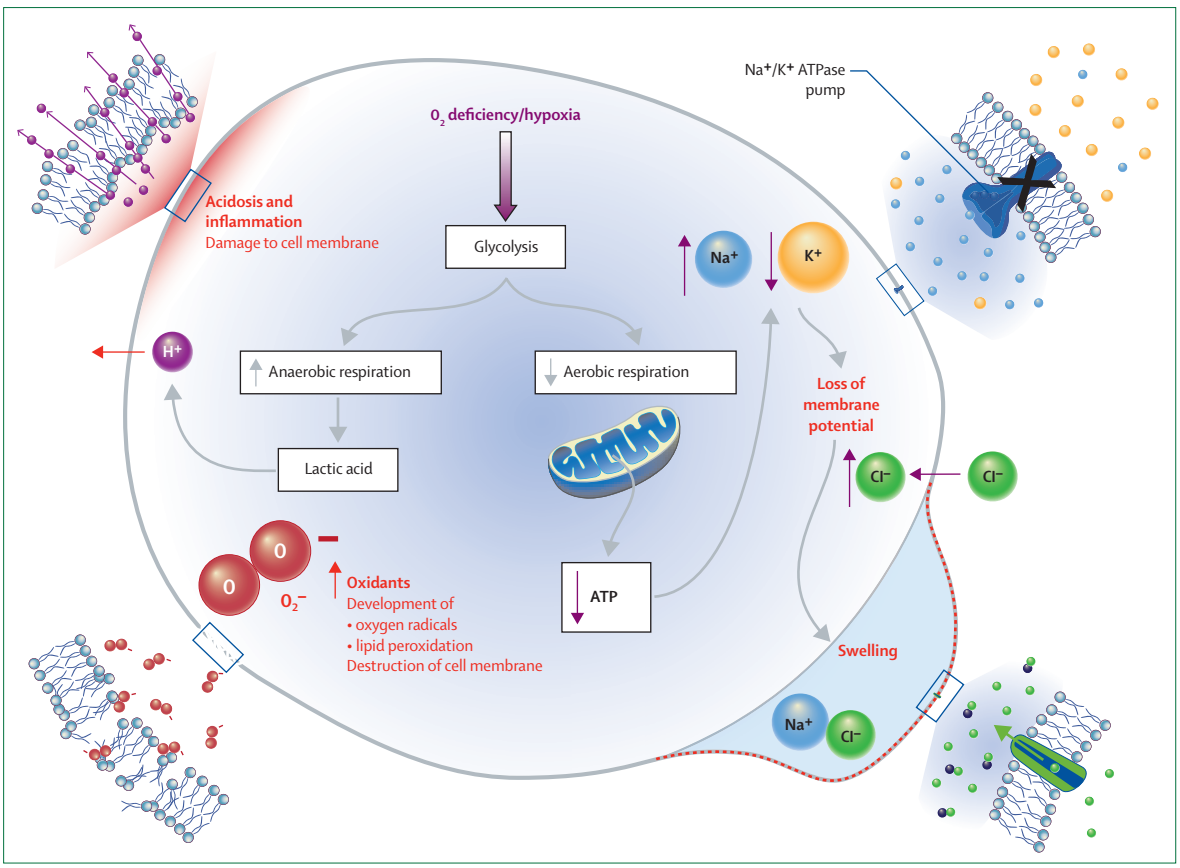

Figure 2: **Cellular pathophysiology of acute extremity compartment syndrome**

disturbances of mitochondrial oxidative phosphorylation and, ultimately, cell-membrane destruction. The subsequent release of hyperkalaemic and acidic blood might also lead to kidney failure, cardiac arrhythmias, and, in severe cases, multiple organ damage that could cause death.[18,19]

The time from the initiating event to acute extremity compartment syndrome can vary from minutes to hours. Total ischaemia time and reduction of aerobic metabolism correlate with irreversible changes in various tissue types. Peripheral nerve tissue is affected early in acute extremity compartment syndrome. Ischaemia of 1 h can lead to reversible neurapraxia, and irreversible axonotmesis has been suggested to occur as early as 4 h.[20] When ischaemia due to acute extremity compartment syndrome persists for more than 6 h, irreversible changes are likely to occur,[21] initiating an irreversible, inflammatory cascade that results in fibrosis in necrotic muscle tissue, which causes further functional impairments, such as contractures.[22]

The clinical sequelae of untreated acute extremity compartment syndrome depend on the anatomical compartment affected. In the leg, the anterior and lateral compartments are most frequently affected and, if untreated, ankle and foot contractures, dysaesthesias in the deep and superficial peroneal nerve distributions, and foot drop might develop.[23,24] In the forearm, Volkmann's contracture is a possible complication where muscle fibrosis leads to decreased hand and wrist motion, diminished strength, and clawing of the fingers.[25]

Diagnosis

Acute extremity compartment syndrome can be diagnosed on the basis of clinical symptoms, intracompartmental pressure, or both.

History and clinical symptoms

Some of the first clinical signs that should raise the suspicion of acute extremity compartment syndrome are severe pain out of proportion to the known injury, and pain that does not improve with adequate analgesia. Resting pain and pain on passive stretching of the affected muscles might be seen. The signs and symptoms of acute extremity compartment syndrome generally evolve progressively and, therefore, the diagnosis is usually made over a period of time unless it is strongly suspected at the initial presentation.[26]

Paraesthesia in the affected extremity might be one of the first signs of hypoxia to nerve tissue within a compartment. For example, altered sensation in between the first two toes could indicate deep peroneal nerve

ischaemia resulting from acute extremity compartment syndrome in the anterior compartment of the leg. Similarly, paresis of the extensor hallucis longus could result from sustained ischaemia to the deep peroneal nerve. However, clinicians must be very careful not to rule out compartment syndrome on the basis of absent neurological signs; motor nerves have some resistance to ischaemia, and objective motor deficits might develop late. Moreover, patients who have had extremity trauma can be difficult to examine clinically, and anxiety, other distracting injuries, and altered mental status might impede assessment.

Fullness or distension of the affected compartment should alert clinicians to the risk of acute extremity compartment syndrome. Subjective assessments of compartments deemed tense or distended, however, are unreliable even when judged by clinicians experienced in trauma care and, therefore, are insufficient to make a diagnosis.[27]

The commonly accepted clinical signs of acute extremity compartment syndrome, pain, pain on passive stretch, paraesthesia, and paresis, were shown in a systematic review to have low sensitivity but high specificity for diagnosis, giving them poor predictive value.[28] A combination of three or more of these clinical findings in a patient at risk of acute extremity compartment syndrome might increase the sensitivity. Of note, though, muscle paresis alone might be a late sign of acute extremity compartment syndrome.

The diagnosis is further complicated when there are communication barriers between clinical staff and patients, the patient has impaired awareness, or when patient-controlled analgesia, regional anaesthesia, or epidural pain catheters are used. Epidural pain catheters in particular carry important risks for masking compartment syndrome and should be avoided in high-risk patients.[29]

We emphasise that the use of the five P mnemomic (pallor, pain out of proportion, pulselessness, paraesthesia, and paralysis) to assess compartment syndrome is misleading. These signs are more often signs of arterial ischaemia than acute extremity compartment syndrome. Instead, if the patient is awake, the five Ps to consider are pain, pain, pain, pain, and pain. Acute extremity compartment syndrome should be at the top of the list of differential diagnoses for any patient with excessive limb pain; pain and paraesthesia are frequently seen in patients presenting with acute extremity compartment syndrome, but pallor, paralysis, and pulselessness might not be present at all or could be very late signs. Rarely, compartment syndrome has been reported in awake and alert patients without severe pain.[30] Therefore, it is important to maintain suspicion of acute extremity compartment syndrome in high-risk patients even when they do not report excessive pain.

The initial insult in compartment syndrome results in impaired venous outflow. As the pressure within an anatomical compartment increases, the capillary perfusion pressure is reached well before the systolic blood pressure, which leads to tissue ischaemia. Blood flow through large arteries is preserved. Distal pulses might not be affected at all or only when the compartment pressure rises above systolic blood pressure. At that stage, irreversible muscle damage is likely to have already occurred. Therefore, use of pulselessness as a primary sign to assess compartment syndrome can cause delays in diagnosis.

In children, who are not small adults, some of the clinical features discussed might not be applicable. Children who cannot provide clear verbal expression of symptoms might show signs of agitation, anxiety, and continually increasing need for analgesic pain medication (the three As).[31]

Measurement of pressure

If the clinical diagnosis is equivocal, measurement of intracompartmental tissue pressure might help to make the differential diagnosis.[32] The physiological compartment pressures in adults are around 8 mm Hg and in children are 10–15 mm Hg.[32,33]

Several techniques have been used to obtain absolute pressure values.[21,26,34,35] Of these, arterial line transducer systems with side-port needles, slit catheters, and self-contained measuring systems are the most accurate.[36,37] Due to differences in pressures within compartments,[38,39] intracompartmental measurements should be obtained roughly within 5 cm of the site of fracture.

Pressure measurements should be obtained in all compartments of the extremities involved to avoid missing the development of acute extremity compartment syndrome in a neighbouring compartment. The anterior compartment is the most common site for acute extremity compartment syndrome in the calf, followed by the lateral compartment. Measurement of all compartments in the distal aspects of the extremities, such as the hand and the foot, should also be attempted, being vigilant of the high number of compartments. Local anaesthesia or even conscious sedation might be helpful when measuring pressure in adults and children.

Absolute pressure greater than 30 mm Hg is thought to be an indication of impaired tissue perfusion in adults and children and, therefore, of the need for emergency surgical fasciotomy.[40,41] The use of an absolute value, however, has been questioned because the perfusion pressure necessary for oxygenation is partly dependent on the patients' blood pressure[40,42,43] and, therefore, could lead to unnecessary fasciotomies. Some researchers have suggested the use of differential pressure (Δp=diastolic blood pressure−intracompartmental pressure), with a proposed threshold of 30 mm Hg.[43] McQueen and Court-Brown[26] were among the first to question absolute cutoff values. They assessed 116 patients with diaphyseal tibia fractures by use of continuous measurement of intracompartmental pressure and found that the absolute intracompartmental pressure was more than 30 mm Hg

in 53 (46%) patients, more than 40 mm Hg in 30 (26%), and more than 50 mm Hg in four (3%). Only three patients, however, had differential pressures less than 30 mm Hg and underwent emergency fasciotomy. No patients had sequelae associated with acute extremity compartment syndrome. A subsequent prospective study of 101 patients by the same group confirmed that Δp had more diagnostic value than absolute intracompartmental pressure.[44] Animal studies of intracompartmental pressure measurements support avoidance of absolute values to indicate compartment release. The data also suggest that irreversible tissue necrosis correlates directly with the difference between intracompartmental and perfusion pressures over time.[38,45]

Rates of diagnosis of acute extremity compartment syndrome and emergency fasciotomy vary substantially. Some centres use continuous pressure monitoring to assess all high-risk patients,[26] whereas others rely on repeated clinical assessments of awake and coherent patients.[46] Intracompartmental pressure during surgery might be reduced due to transient diastolic hypotension associated with anaesthesia.[47] Therefore, measurements should be repeated after surgery to confirm complete release.

Continuous measurement of intracompartmental pressure can be made by attaching a catheter to an arterial transducer. Although the technical learning curve for this approach is slightly greater than that for other methods, it might reduce the risk of missed compartment syndrome.[26,48–50] Continuous measurement might be particularly beneficial in patients with impaired awareness or consciousness in whom physical examination is not possible or in those who cannot report symptoms of pain and paraesthesia.[51] Some studies, however, have suggest that the use of continuous measurement can lead to unnecessary fasciotomy,[52] which carries its own risks and complications,[53] such as long-term hospital stay, infection, delayed wound healing, and, potentially, delayed bone healing.[54]

Most studies of measurement of intracompartmental pressure have been done in patients with compartment syndrome in the leg. The findings have been extrapolated to other extremities and to children, despite the variability in diastolic blood pressures and anatomy.[55]

Diagnostic tools under investigation

Near-infrared spectroscopy has been introduced into clinical practice as a new tool to measure tissue oxygenation, and follows the principles of pulse oximetry.[56] Human tissue oxygenation is assessed by comparing the concentrations of venous blood oxyhaemoglobin and deoxyhaemoglobin to a depth of around 3 cm in soft tissue.[57] In theory, near-infrared spectroscopy can monitor patients at risk of acute extremity compartment syndrome by indirectly measuring decreased tissue perfusion due to raised intracompartmental pressures. Near-infrared spectroscopy has been studied in small case series[58] and some evidence from animal and basic science studies support positive findings,[59,60] but its broader clinical applicability has yet to be assessed in large trials.[61]

Specific compartment syndromes

Patients who are at risk of developing acute extremity compartment syndrome must be identified promptly. The incidence of acute extremity compartment syndrome is reported to be 7·3 per 100 000 of the general population for men and 0·7 per 100 000 for women.[3] A large single-centre study in a level 1 trauma centre showed that acute extremity compartment syndrome is associated with fractures of the tibial shaft in up to 36% of cases.[3] Other associated causes are soft-tissue injuries of the extremities, distal radius fractures, crush injuries, diaphyseal fractures of the radius and ulna, femoral fractures, and tibial plateau fractures (table).[3] In up to 30% of cases, however, acute extremity compartment syndrome develops from soft-tissue injury without a fracture.[2]

Lower extremities

The calf

The lower leg consists of four compartments: anterior, lateral, superficial posterior, and deep posterior (figure 3). The anterior intermuscular septum separates the lateral muscles from the anterior muscles, and the posterior intermuscular septum separates the lateral muscles from the posterior muscles. The interosseous membrane spans the gap between the tibia and fibula, separating the anterior and deep posterior compartments. The transverse intermuscular septum separates the musculature of the superficial and deep posterior compartments.

The lower leg is the most common location of acute extremity compartment syndrome, with the anterior and lateral compartments most frequently affected. Diaphyseal fractures of the tibia are mostly commonly associated with acute extremity compartment syndrome of the lower leg. An open injury does not exclude the possibility of developing acute extremity compartment syndrome.[62] High-energy tibial plateau fractures and

	Proportion of cases (%)
Fracture	
Tibial diaphyseal fracture	36%
Distal radius fracture	10%
Diaphyseal forearm fracture	8%
Femoral diaphyseal fracture	3%
Tibial plateau fracture	3%
Soft tissue	
Soft-tissue injury	23%
Crush syndrome	8%
Other	9%

Table: Incidence of fractures and other disorders associated with acute extremity compartment syndrome

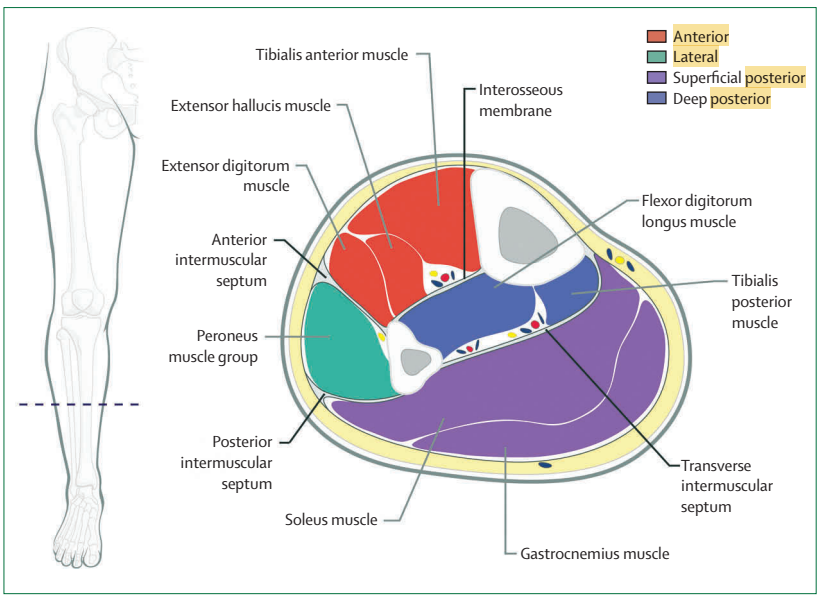

Figure 3: **Cross-sectional anatomy of the calf**

fracture-dislocations of the knee are also commonly associated with acute extremity compartment syndrome of the lower leg.[63]

Initially, non-surgical measures, such as removal of constrictive dressings or splitting of cast material and elevation of the limb, can be used along with repeated clinical assessments. The definitive treatment for acute extremity compartment syndrome of the lower leg is emergency surgical release and four-compartment fasciotomy. The release of all four compartments is typically achieved with two incisions, one incision centred over the intermuscular septum laterally and the other 2 cm posterior to the subcutaneous border of the tibia.[64] A single-incision technique has also been described[64] for which similar results have been reported.[65] Therefore, the choice of technique should be based on the surgeon's experience and the patient's condition.[65] Irrespective of the method, wide release of the compartments is crucial. The wounds should not be closed, but rather left open and dressed sterilely. Early closure of fasciotomy wounds has been associated with recurrence of acute extremity compartment syndrome.[64]

Despite the lower leg being the most common location for acute extremity compartment syndrome to occur, few studies have been done to assess the functional outcomes in treated patients.[66–68] Most patients who undergo emergency fasciotomy seem to return to the activity level before injury. Clinical outcomes are improved when fasciotomy is done within 12–24 h of acute extremity compartment syndrome developing,[17,21,69] although assessment of the exact time of onset is often difficult. Many patients have substantial muscle damage by the time of fasciotomy release. As clinically anticipated, patients who have had acute extremity compartment syndrome of the leg report significantly worse health outcomes and quality of life than controls, as measured with EQ-5D.[70] The degree of subsequent functional disability is generally associated with severity of the primary soft-tissue trauma.[23] Whether a patient has associated injuries does not seem to affect long-term outcomes.[46]

After surgical decompression of the affected compartment, the skin edges can be approximated with either elastic bands or vessel loops in a crisscross fashion. Moist dressings are used to cover the open wound to protect the tissue from drying and retraction, and these dressings improve secondary wound closure. Vacuum-assisted wound closure with negative pressure can also be used. Repeat debridement is usually necessary after 48–72 h. When primary wound closure is not possible, split-thickness skin grafts may be used at a ratio of 1·0:1·5. Passive stretching exercises and splinting might help to avoid shortening of muscle groups and the development of joint contractures in some types of injury.

When acute extremity compartment syndrome is missed or treatment is delayed, late functional disabilities are mainly caused by limited dorsiflexion of the ankle joint, reduced functional strength of the foot extensors, contractures of the foot flexors, abnormal superficial sensibility, and wound complications associated with the fasciotomy site. Amputation might be required if adequate perfusion and function cannot be restored.[71]

The thigh

The thigh has three compartments: anterior, medial, and posterior (figure 4). Separating the anterior compartment from the medial and posterior compartments are two well defined fascial thickenings, the medial and lateral intermuscular septa, which attach to the femur and are associated with the fascia lata. The medial compartment is separated from the posterior compartment by a thin fascial plane. The vastus intermedius is sometimes deemed to be a separate compartment.[69]

Femur fractures resulting from road traffic accidents are the injuries most commonly associated with the development of acute extremity compartment syndrome in the thigh.[72] The diagnosis of acute thigh compartment syndrome is difficult because it is frequently associated with multiple high-energy traumatic injuries and patients might have impaired awareness and distracting injuries. Mortality in patients with acute thigh compartment syndrome is reported to be as high as 47%,[73] although deaths are most frequently related to concomitant injuries. In a case series of 23 patients with acute extremity compartment syndrome in the thigh, mortality was 17%.[74] In the same series, eight (35%) patients had ischaemic changes in the musculature at the time of fascia release and four (17%) developed wound complications requiring amputation.[74]

The standard treatment of acute compartment syndrome of the thigh is emergency fasciotomy. Single-incision or two-incision techniques may be used.

Patients who undergo decompression within 8 h of injury have significantly better outcomes and thigh muscle strength and a significantly lower prevalence of long-term functional impairment than those who are treated later.[69] Thigh compartment fasciotomy results in large scars, and wound infection is seen in up to 67% of cases, which is associated with a significant risk of death.[75,76]

Long-term functional deficits are seen in 57% of patients with acute thigh compartment syndrome, of which neurological deficits seem to be the most frequent (16%).[72] Although the data on functional outcome are sparse, patients younger than 40 years seem to have significantly better outcome scores than older patients.[69]

The feet

The actual number of compartments and the treatment of acute compartment syndrome in the foot are controversial. Although three compartments were initially described—medial, lateral, and superficial—more were identified later in cadaveric studies by Manoli.[77] Each foot is now believed to have nine compartments: medial, lateral, four interossei, and three central. How diagnosis of acute extremity compartment syndrome by measurement of intracompartmental pressure and the need for fasciotomy are affected by the number of compartments is not well understood.[78]

The natural history of non-surgical management of acute compartment syndrome in the foot includes the potential development of ischaemic contractures, neuropathy, deformity, and chronic pain.[79,80] The ischaemic insult to the nerves might result in decreased proprioception and sensation, which can lead to ulceration. Lesser-toe deformities (claw toes) and cavus foot deformity are common sequelae.[79,80] Ischaemic contracture of the quadratus plantae muscle due to high compartment pressures can also be the cause of toe flexion deformities, especially in calcaneal fractures.[81]

There is some debate about the role of emergency fasciotomy of acute foot compartment syndrome. Although emergency fasciotomy is indicated in practically all other acute extremity compartment syndromes, its recommendation in the foot lacks consensus.[82] Some surgeons prefer to manage the sequelae of untreated compartment syndrome rather than make multiple large incisions on swollen and injured feet. The lack of high-level evidence, therefore, prevents development of specific guidelines.[82]

Complication rates associated with early fasciotomy for acute foot compartment syndrome are lower than those for untreated compartment syndrome, but this treatment remains controversial.[77,83] Fasciotomy can lead to severe scarring in some patients, and other long-term sequelae are paraesthesia and skin necrosis that necessitates skin grafts.[84] Myerson[85] reported on 12 patients treated with fasciotomy for acute compartment syndrome in the foot. Four patients returned to work and resumed exercise activities at preoperative levels. For six patients normal shoe wear was troublesome and three patients developed contractures with claw toe despite fasciotomy. In a prospective study of 14 patients, 11 returned to work after fasciotomy. Two patients developed claw deformity, and three had sensory abnormalities.[86] Shorter time to fasciotomy, younger age, the absence of bone injuries and other concomitant injuries, and low-velocity crush injuries were positive predictors of better functional outcomes among these patients.

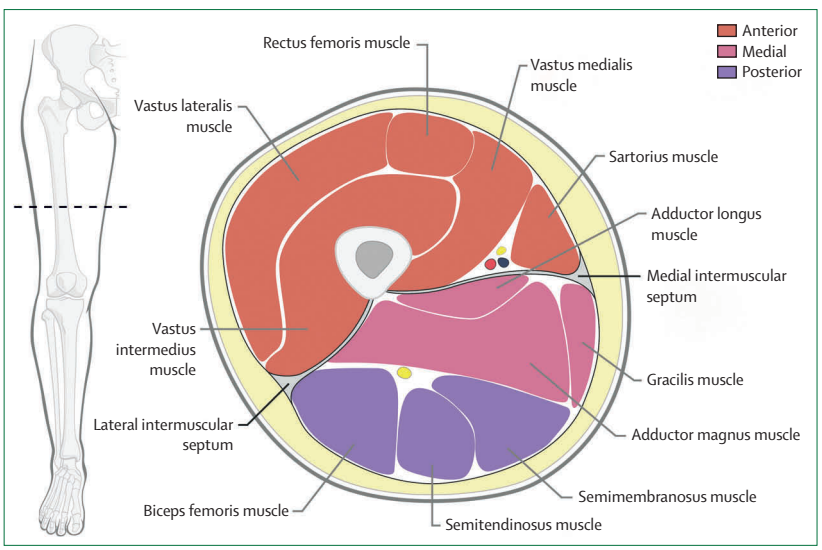

Figure 4: **Cross-sectional anatomy of the thigh**

Upper extremities

The upper arm contains two compartments, the forearm three, and the hand ten, all of which are at risk of acute extremity compartment syndrome. In the digits, the fascial compartments might also be susceptible to acute compartment syndrome and need release. Few cases of acute compartment syndrome in the upper extremities have been reported and, therefore, the true incidence is difficult to assess. In a systematic review of acute compartment syndrome in the forearm, 35% of cases were linked to fractures, 10% to narcotic overdoses, and 8% to intravenous infiltrations.[87] In a series of 164 cases of traumatic acute extremity compartment syndrome, 16 (10%) were associated with distal radius fractures and 13 (8%) with diaphyseal forearm fractures.[3] Patients are at particular risk of developing acute extremity compartment syndrome when they sustain a distal radius fracture and simultaneous ipsilateral elbow injury,[88] or a distal radius fracture with translation of more than 35%[89] or associated brachial artery injury.[90]

Acute compartment syndrome in the upper extremities is most often diagnosed by clinical examination and measurement of intracompartmental pressures, as described earlier in this paper. Good hand function depends on supple joints, precise sensibility, and fine motor control. Missed acute compartment syndrome in

the forearm or hand, therefore, will have severe functional consequences. Diagnosis might be delayed in patients who are uncooperative or have impaired awareness, which could lead to debilitating functional outcomes. Late sequelae range from skin changes to contractures, neurological deficits, infection, amputation, or death. Similar to acute compartment syndrome in the lower extremities, time to diagnosis is crucial. The optimum timing of fasciotomy is suggested to be within 8 h of development of acute extremity compartment syndrome. Whether fasciotomy is useful 8–24 h after onset has been questioned because muscle necrosis has generally already occurred. Fasciotomy for delayed acute compartment syndrome of the upper extremity might not improve outcomes and has been associated with the need for multiple surgical procedures to debride non-viable muscle tissue.[64,91]

Acute extremity compartment syndrome in children

The diagnosis of acute extremity compartment syndrome in children is particularly difficult and is often delayed because the classic signs commonly cited in adults are not helpful in children.[31] Escalating pain, pain with passive stretch of affected muscles, and diminished perfusion are not straightforward to characterise, particularly in acutely injured, non-verbal, or developmentally delayed children. For this reason, clinical diagnosis of acute extremity compartment syndrome in paediatric patients should be based on the three As: anxiety, agitation, and increasing analgesic requirement.[31]

Acute extremity compartment syndrome in children develops most frequently in association with fractures, especially of the lower extremities.[31] In a study of 212 children with open or closed tibial fractures, Shore and colleagues[92] showed that the incidence of acute extremity compartment syndrome was 11·6%. The time to fasciotomy was delayed (longer than 12 h after injury) in 110 (52%) patients, but, surprisingly, was not associated with significantly worse outcomes than earlier treatment. 195 (92%) patients regained preinjury functional levels, which suggests that children tolerate increased intracompartmental pressure for longer periods of time than adults before tissue necrosis becomes irreversible. Flynn and colleagues[93] assessed 42 children with acute traumatic compartment syndrome treated with fasciotomy and followed up for an average of 1 year. Only two patients had permanent functional impairment, and in both the time from injury to fasciotomy exceeded 80 h. Flynn and colleagues highlighted the subtleties of diagnosis in paediatric patients and noted that presentation of compartment syndrome might be delayed.

The largest case series of acute compartment syndrome in the upper extremities of children identified supracondylar humerus fractures as one of the most frequently associated injuries, followed by intravenous infiltrates, crush injuries, and tight casts.[41,94–96] A review found no correlation between the time from diagnosis to fasciotomy and final functional outcomes.[97]

Acute compartment syndrome in the foot is rare in children.[98] Silas and colleagues[99] have reported outcomes in the largest series of children with acute compartment syndrome in the feet and found that it most commonly occurs due to crush injuries or Lisfranc's fracture dislocations.

The most important point to remember is that presentation of acute extremity compartment syndrome in children differs from that in adults, particularly in terms of increasing need for analgesia after fracture. With timely diagnosis and appropriate fasciotomy, however, children can regain function and have favourable outcomes. Thus, a high index of suspicion should be maintained to avoid missed diagnosis and unfavourable outcomes.

Missed compartment syndrome

The optimum treatment of acute extremity compartment syndrome is emergency fasciotomy immediately after diagnosis. Animal studies suggest that tissue necrosis occurs within 6–12 h of onset of hypoxaemia.[21,100] Thus, better clinical outcomes have been reported when fasciotomy is performed within this timeframe.[50,92,101–103] However, since acute extremity compartment syndrome is an evolving disorder with intracompartmental pressure varying throughout its course, the exact time of onset can be difficult to define. The time from admission has been suggested as a surrogate start point as it is closest to the time of injury.[26,48] In some studies, though, researchers have chosen not to include an exact time of onset as it cannot be clinically established.[51]

Crush syndrome might lead to acute extremity compartment syndrome. Vigilant monitoring is needed to identify metabolic disturbances, such as shock in association with renal failure, in patients who have crush injuries to skeletal muscle to reduce the risk of rhabdomyolysis, which might lead to acute extremity compartment syndrome. Adequate medical resuscitation treatment is of the utmost importance to counteract hypotension, hypocalcaemia, hyperkalaemia, acidosis, and renal failure, especially in patients at risk of reperfusion syndrome.

If irreversible muscle necrosis has occurred before surgical release, patients are at risk of seeding of necrotic muscle carrying bacteria that could lead to systemic effects. If necrotic muscle becomes infected, repeated debridement is needed, and amputation might be necessary if the infection cannot be controlled. This complication, therefore, creates a clinical dilemma of whether or not fasciotomy should be performed if time of onset of acute extremity compartment syndrome is unclear.

Missed compartment syndrome might have legal consequences. Bhattacharyya and Vrahas[104] identified several risk factors for unsuccessful defence against

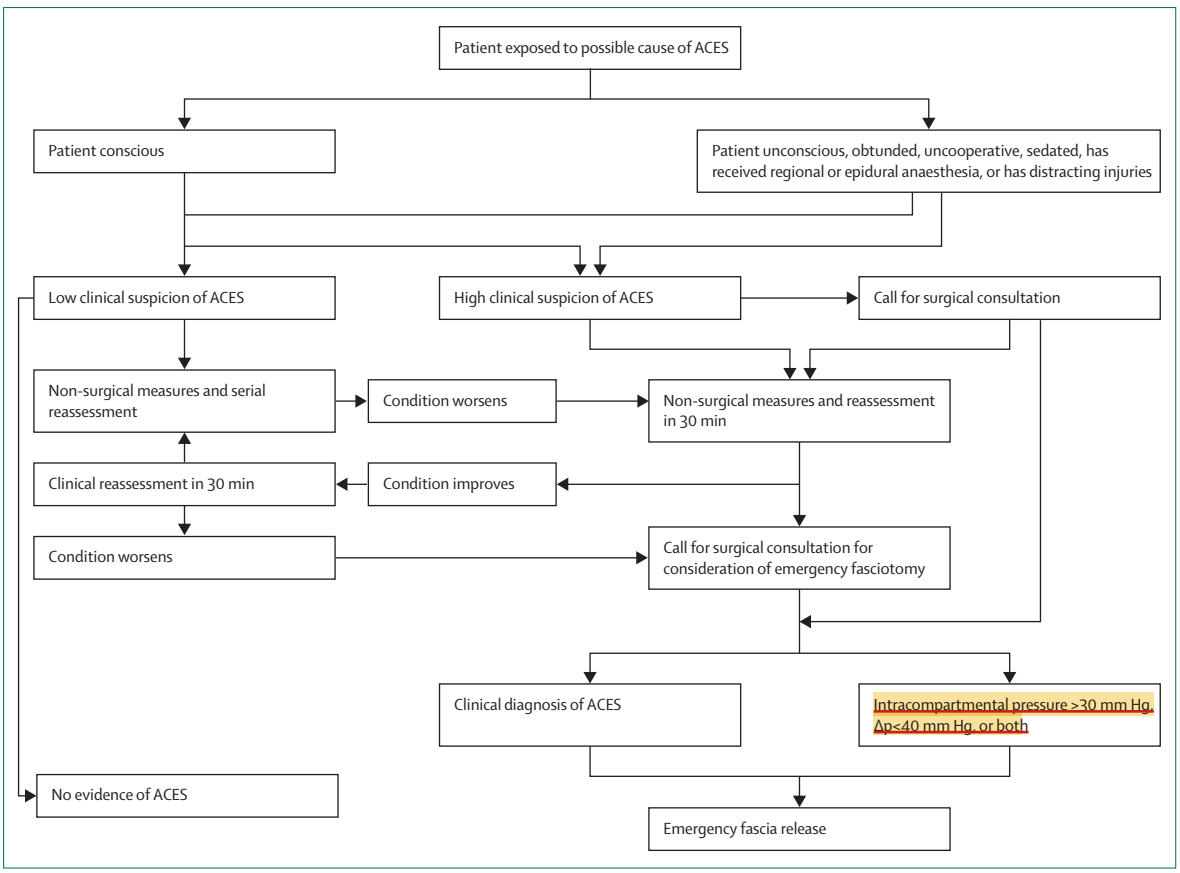

***Figure 5*: Proposed clinical treatment guidelines**
ACES=acute extremity compartment syndrome. Δp=diastolic blood pressure – intracompartmental pressure.

malpractice claims, including documentation of neurological compromise without subsequent action, poor communication between doctors, the number of cardinal signs, and increased time to fasciotomy. Common sense might lead physicians and patients to believe that a delay in diagnosis is due to abnormal presentation and symptoms, but inadequate training and poor communication seem to represent important risk factors for missed diagnosis, although no prospective research has been done to confirm the role of any factors.

The potential to salvage any viable muscle often outweighs the risks of fasciotomy in patients with delayed presentation of acute extremity compartment syndrome. Only if acute extremity compartment syndrome has clearly been missed and symptoms have been present for longer than 24–48 h without evidence of muscle function in the affected compartments should non-surgical management be applied. In such cases, care should comprise supportive measures, such as adequate hydration, pain control, and splinting, to counteract the development of fibrosis and contractures.

The systemic effects of missed acute extremity compartment syndrome with associated muscle necrosis might include notable increase in myoglobin concentrations. Renal failure might develop due to acute tubular necrosis in response to severe myoglobinuria.[105] In severe cases, hyperkalaemia and the development of metabolic acidosis can lead to cardiac arrhythmia or failure, which could ultimately result in loss of life. In these cases, urgent debridement of necrotic muscle is important to minimise metabolic insult and prevent further systemic complications.

Conclusions and future areas of research

Acute extremity compartment syndrome is a surgical emergency with potentially devastating sequelae. Accurate and timely diagnosis is of the utmost importance. Intracompartmental pressure should be measured in patients thought to be at risk of acute extremity compartment syndrome or in those who are non-compliant or unconscious (figure 5). Scientific investigations should focus on improving diagnostic tools for acute extremity compartment syndrome, as at present only indirect measures of tissue hypoxaemia and impending tissue necrosis are available. Future research should also aim to identify thresholds for treatment in adults and children with acute extremity compartment syndrome to eliminate the occurrence of late-onset and long-term sequelae while minimising the risk of overtreatment.

Contributors
AGvK and MSV planned the paper, and AGvK drafted the manuscript framework and wrote the abstract. MSV summarised the relevance of the research about present care and assessed the evidence and reviewed the whole paper. MJW helped with writing and reviewing of the sections on pathophysiology, lower-extremity compartment syndrome, and missed compartment syndrome. GSMD wrote and reviewed the section on upper-extremity compartment syndrome. PTA wrote the clinical diagnosis and future developments sections. DSB wrote the section on paediatric compartment syndrome. MH did the literature search, assessed the evidence, analysed and interpreted the data and, reviewed the sections on pathophysiology and lower-extremity compartment syndrome. JBJ reviewed the section on missed compartment syndrome and assessed the evidence in the whole paper.

Declaration of interests
We declare no competing interests.

References

1 von Volkman R. Die ischämischen Muskellähmungen und Kontracturen. *Centralblatt Chirurg* 1881; **8:** 801–03.
2 Hope MJ, McQueen MM. Acute compartment syndrome in the absence of fracture. *J Orthop Trauma* 2004; **18:** 220–24.
3 McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome. Who is at risk? *J Bone Joint Surg Br* 2000; **82:** 200–03.
4 Chim H, Soltanian H. Spontaneous compartment syndrome of the forearm in association with nephrotic syndrome and transient bacteremia. *J Surg Case Rep* 2012; **2012:** 11.
5 Ridha A, Khan A, Al-Abayechi S, Puthenveetil V. Acute compartment syndrome secondary to rhabdomyolysis in a sickle cell trait patient. *Lancet* 2014; **384:** 2172.
6 Wang KL, Li SY, Chuang CL, Chen TW, Chen JY. Subfascial hematoma progressed to arm compartment syndrome due to a nontransposed brachiobasilic fistula. *Am J Kidney Dis* 2006; **48:** 990–92.
7 Belzunegui T, Louis CJ, Torrededia L, Oteiza J. Extravasation of radiographic contrast material and compartment syndrome in the hand: a case report. *Scand J Trauma Resusc Emerg Med* 2011; **19:** 9.
8 Toney J, Donovan S, Adelman V, Adelman R. Non-necrotizing streptococcal cellulitis as a cause of acute, atraumatic compartment syndrome of the foot: a case report. *J Foot Ankle Surg* 2015; published online May 14. DOI:10.1053/j.jfas.2015.02.010.
9 Kleshinski J, Bittar S, Wahlquist M, Ebraheim N, Duggan JM. Review of compartment syndrome due to group A streptococcal infection. *Am J Med Sci* 2008; **336:** 265–69.
10 Barney BM, Gillette BP, Garces YI, Ding W, Cheville JC, Rose PS. Myeloid sarcoma of the upper extremity causing compartment syndrome: a case report. *Pract Radiat Oncol* 2011; **1:** 135–38.
11 Bedri MI, Khosravi AH, Lifchez SD. Upper extremity compartment syndrome in the setting of deep venous thrombosis and phlegmasia cerulea dolens: case report. *J Hand Surg Am* 2009; **34:** 1859–63.
12 Beraldo S, Dodds SR. Lower limb acute compartment syndrome after colorectal surgery in prolonged lithotomy position. *Dis Colon Rectum* 2006; **49:** 1772–80.
13 Clasper JC, Aldington DJ. Regional anaesthesia, ballistic limb trauma and acute compartment syndrome. *J R Army Med Corps* 2010; **156:** 77–78.
14 Balogh ZJ, Butcher NE. Compartment syndromes from head to toe. *Crit Care Med* 2010; **38** (suppl)**:** S445–51.
15 Garfin SR, Tipton CM, Mubarak SJ, Woo SL, Hargens AR, Akeson WH. Role of fascia in maintenance of muscle tension and pressure. *J Appl Physiol* 1981; **51:** 317–20.
16 Stecco C, Macchi V, Porzionato A, Duparc F, De Caro R. The fascia: the forgotten structure. *Ital J Anat Embryol* 2011; **116:** 127–38.
17 Matsen FA 3rd. Compartmental syndromes. *N Engl J Med* 1979; **300:** 1210–11.
18 Odeh M. The role of reperfusion-induced injury in the pathogenesis of the crush syndrome. *N Engl J Med* 1991; **324:** 1417–22.
19 Blaisdell FW. The pathophysiology of skeletal muscle ischemia and the reperfusion syndrome: a review. *Cardiovasc Surg* 2002; **10:** 620–30.
20 Hargens AR, Romine JS, Sipe JC, Evans KL, Mubarak SJ, Akeson WH. Peripheral nerve-conduction block by high muscle-compartment pressure. *J Bone Joint Surg Am* 1979; **61:** 192–200.
21 Rorabeck CH, Clarke KM. The pathophysiology of the anterior tibial compartment syndrome: an experimental investigation. *J Trauma* 1978; **18:** 299–304.
22 Huard J, Li Y, Fu FH. Muscle injuries and repair: current trends in research. *J Bone Joint Surg Am* 2002; **84-A:** 822–32.
23 Vandervelpen G, Goris L, Broos PL, Rommens PM. Functional sequelae in tibial shaft fractures with compartment syndrome following primary treatment with urgent fasciotomy. *Acta Chir Belg* 1992; **92:** 234–40 (in Dutch).
24 Zwipp H. Reconstructive surgery of sequelae of compartment syndrome of the lower leg and/or foot. Presentation of a new classification. *Unfallchirurg* 2008; **111:** 776–82 (in German).
25 Mubarak SJ, Carroll NC. Volkmann's contracture in children: aetiology and prevention. *J Bone Joint Surg Br* 1979; **61-B:** 285–93.
26 McQueen MM, Court-Brown CM. Compartment monitoring in tibial fractures. The pressure threshold for decompression. *J Bone Joint Surg Br* 1996; **78:** 99–104.
27 Shuler FD, Dietz MJ. Physicians' ability to manually detect isolated elevations in leg intracompartmental pressure. *J Bone Joint Surg Am* 2010; **92:** 361–67.
28 Ulmer T. The clinical diagnosis of compartment syndrome of the lower leg: are clinical findings predictive of the disorder? *J Orthop Trauma* 2002; **16:** 572–77.
29 Harrington P, Bunola J, Jennings AJ, Bush DJ, Smith RM. Acute compartment syndrome masked by intravenous morphine from a patient-controlled analgesia pump. *Injury* 2000; **31:** 387–89.
30 O'Sullivan MJ, Rice J, McGuinness AJ. Compartment syndrome without pain! *Ir Med J* 2002; **95:** 22.
31 Bae DS, Kadiyala RK, Waters PM. Acute compartment syndrome in children: contemporary diagnosis, treatment, and outcome. *J Pediatr Orthop* 2001; **21:** 680–88.
32 Shadgan B, Menon M, O'Brien PJ, Reid WD. Diagnostic techniques in acute compartment syndrome of the leg. *J Orthop Trauma* 2008; **22:** 581–87.
33 Staudt JM, Smeulders MJ, van der Horst CM. Normal compartment pressures of the lower leg in children. *J Bone Joint Surg Br* 2008; **90:** 215–19.
34 Hammerberg EM, Whitesides TE Jr, Seiler JG 3rd. The reliability of measurement of tissue pressure in compartment syndrome. *J Orthop Trauma* 2012; **26:** e166.
35 Mubarak SJ. A practical approach to compartmental syndromes. Part II. Diagnosis. *Instr Course Lect* 1983; **32:** 92–102.
36 Wong JC, Vosbikian MM, Dwyer JM, Ilyas AM. Accuracy of measurement of hand compartment pressures: a cadaveric study. *J Hand Surg Am* 2015; **40:** 701–06.
37 Boody AR, Wongworawat MD. Accuracy in the measurement of compartment pressures: a comparison of three commonly used devices. *J Bone Joint Surg Am* 2005; **87:** 2415–22.
38 Matava MJ, Whitesides TE Jr, Seiler JG 3rd, Hewan-Lowe K, Hutton WC. Determination of the compartment pressure threshold of muscle ischemia in a canine model. *J Trauma* 1994; **37:** 50–58.
39 Heckman MM, Whitesides TE Jr, Grewe SR, Rooks MD. Compartment pressure in association with closed tibial fractures. The relationship between tissue pressure, compartment, and the distance from the site of the fracture. *J Bone Joint Surg Am* 1994; **76:** 1285–92.
40 Mubarak SJ, Owen CA, Hargens AR, Garetto LP, Akeson WH. Acute compartment syndromes: diagnosis and treatment with the aid of the wick catheter. *J Bone Joint Surg Am* 1978; **60:** 1091–95.
41 Matsen FA 3rd, Veith RG. Compartmental syndromes in children. *J Pediatr Orthop* 1981; **1:** 33–41.
42 Matsen FA 3rd, Winquist RA, Krugmire RB Jr. Diagnosis and management of compartmental syndromes. *J Bone Joint Surg Am* 1980; **62:** 286–91.
43 Whitesides TE, Haney TC, Morimoto K, Harada H. Tissue pressure measurements as a determinant for the need of fasciotomy. *Clin Orthop Relat Res* 1975; **Nov–Dec:** 43–51.
44 White TO, Howell GE, Will EM, Court-Brown CM, McQueen MM. Elevated intramuscular compartment pressures do not influence outcome after tibial fracture. *J Trauma* 2003; **55:** 1133–38.
45 Heckman MM, Whitesides TE Jr, Grewe SR, Judd RL, Miller M, Lawrence JH 3rd. Histologic determination of the ischemic threshold of muscle in the canine compartment syndrome model. *J Orthop Trauma* 1993; **7:** 199–210.

46 Frink M, Klaus AK, Kuther G, et al. Long term results of compartment syndrome of the lower limb in polytraumatised patients. *Injury* 2007; **38:** 607–13.

47 Kakar S, Firoozabadi R, McKean J, Tornetta P 3rd. Diastolic blood pressure in patients with tibia fractures under anaesthesia: implications for the diagnosis of compartment syndrome. *J Orthop Trauma* 2007; **21:** 99–103.

48 McQueen MM, Christie J, Court-Brown CM. Acute compartment syndrome in tibial diaphyseal fractures. *J Bone Joint Surg Br* 1996; **78:** 99–104.

49 Allen MJ, Stirling AJ, Crawshaw CV, Barnes MR. Intracompartmental pressure monitoring of leg injuries. An aid to management. *J Bone Joint Surg Br* 1985; **67:** 53–57.

50 McQueen MM, Court-Brown CM. Early diagnosis of compartment syndrome: continuous pressure measurement or not? *Injury* 2010; **41:** 431–32.

51 Harris IA, Kadir A, Donald G. Continuous compartment pressure monitoring for tibia fractures: does it influence outcome? *J Trauma* 2006; **60:** 1330–35.

52 Janzing HM, Broos PL. Routine monitoring of compartment pressure in patients with tibial fractures: beware of overtreatment! *Injury* 2001; **32:** 415–21.

53 Al-Dadah OQ, Darrah C, Cooper A, Donell ST, Patel AD. Continuous compartment pressure monitoring vs. clinical monitoring in tibial diaphyseal fractures. *Injury* 2008; **39:** 1204–09.

54 Reverte MM, Dimitriou R, Kanakaris NK, Giannoudis PV. What is the effect of compartment syndrome and fasciotomies on fracture healing in tibial fractures? *Injury* 2011; **42:** 1402–07.

55 Mars M, Hadley GP. Raised compartmental pressure in children: a basis for management. *Injury* 1998; **29:** 183–85.

56 Garr JL, Gentilello LM, Cole PA, Mock CN, Matsen FA 3rd. Monitoring for compartmental syndrome using near-infrared spectroscopy: a noninvasive, continuous, transcutaneous monitoring technique. *J Trauma* 1999; **46:** 613–16.

57 Mancini DM, Bolinger L, Li H, Kendrick K, Chance B, Wilson JR. Validation of near-infrared spectroscopy in humans. *J Appl Physiol (1985)* 1994; **77:** 2740–47.

58 Shuler MS, Reisman WM, Whitesides TE Jr, et al. Near-infrared spectroscopy in lower extremity trauma. *J Bone Joint Surg Am* 2009; **91:** 1360–68.

59 Cathcart CC, Shuler MS, Freedman BA, Reno LR, Budsberg SC. Correlation of near-infrared spectroscopy and direct pressure monitoring in an acute porcine compartmental syndrome model. *J Orthop Trauma* 2014; **28:** 365–69.

60 Breit GA, Gross JH, Watenpaugh DE, Chance B, Hargens AR. Near-infrared spectroscopy for monitoring of tissue oxygenation of exercising skeletal muscle in a chronic compartment syndrome model. *J Bone Joint Surg Am* 1997; **79:** 838–43.

61 Shuler MS, Reisman WM, Kinsey TL, et al. Correlation between muscle oxygenation and compartment pressures in acute compartment syndrome of the leg. *J Bone Joint Surg Am* 2010; **92:** 863–70.

62 Moehring HD, Voigtlander JP. Compartment pressure monitoring during intramedullary fixation of tibial fractures. *Orthopedics* 1995; **18:** 631–35.

63 Stark E, Stucken C, Trainer G, Tornetta P 3rd. Compartment syndrome in Schatzker type VI plateau fractures and medial condylar fracture-dislocations treated with temporary external fixation. *J Orthop Trauma* 2009; **23:** 502–06.

64 Olson SA, Glasgow RR. Acute compartment syndrome in lower extremity musculoskeletal trauma. *J Am Acad Orthop Surg* 2005; **13:** 436–44.

65 Bible JE, McClure DJ, Mir HR. Analysis of single-incision versus dual-incision fasciotomy for tibial fractures with acute compartment syndrom. *J Orthop Trauma* 2013; **27:** 607–11.

66 Fitzgerald AM, Gaston P, Wilson Y, Quaba A, McQueen MM. Long-term sequelae of fasciotomy wounds. *Br J Plast Surg* 2000; **53:** 690–93.

67 Finkelstein JA, Hunter GA, Hu RW. Lower limb compartment syndrome: course after delayed fasciotomy. *J Trauma* 1996; **40:** 342–44.

68 Sheridan GW, Matsen FA 3rd. Fasciotomy in the treatment of the acute compartment syndrome. *J Bone Joint Surg Am* 1976; **58:** 112–15.

69 Mithoefer K, Lhowe DW, Vrahas MS, Altman DT, Erens V, Altman GT. Functional outcome after acute compartment syndrome of the thigh. *J Bone Joint Surg Am* 2006; **88:** 729–37.

70 Giannoudis PV, Nicolopoulos C, Dinopoulos H, Ng A, Adedapo S, Kind P. The impact of lower leg compartment syndrome on health related quality of life. *Injury* 2002; **33:** 117–21.

71 Rorabeck CH. The treatment of compartment syndromes of the leg. *J Bone Joint Surg Br* 1984; **66:** 93–97.

72 Ojike NI, Roberts CS, Giannoudis PV. Compartment syndrome of the thigh: a systematic review. *Injury* 2010; **41:** 133–36.

73 Schwartz JT Jr, Brumback RJ, Lakatos R, Poka A, Bathon GH, Burgess AR. Acute compartment syndrome of the thigh. A spectrum of injury. *J Bone Joint Surg Am* 1989; **71:** 392–400.

74 Kanlic EM, Pinski SE, Verwiebe EG, Saller J, Smith WR. Acute morbidity and complications of thigh compartment syndrome: a report of 26 cases. *Patient Saf Surg* 2010; **4:** 13.

75 Masini BD, Racusin AW, Wenke JC, Gerlinger TL, Hsu JR. Acute compartment syndrome of the thigh in combat casualties. *J Surg Orthop Adv* 2013; **22:** 42–49.

76 Mithöfer K, Lhowe DW, Vrahas MS, Altman DT, Altman GT. Clinical spectrum of acute compartment syndrome of the thigh and its relation to associated injuries. *Clin Orthop Relat Res* 2004; **Aug:** 223–29.

77 Manoli A 2nd. Compartment syndromes of the foot: current concepts. *Foot Ankle* 1990; **10:** 340–44.

78 Frink M, Hildebrand F, Krettek C, Brand J, Hankemeier S. Compartment syndrome of the lower leg and foot. *Clin Orthop Relat Res* 2010; **468:** 940–50.

79 Botte MJ, Santi MD, Prestianni CA, Abrams RA. Ischemic contracture of the foot and ankle: principles of management and prevention. *Orthopedics* 1996; **19:** 235–44.

80 Santi MD, Botte MJ. Volkmann's ischemic contracture of the foot and ankle: evaluation and treatment of established deformity. *Foot Ankle Int* 1995; **16:** 368–77.

81 Mittlmeier T, Mächler G, Lob G, Mutschler W, Bauer G, Vogl T. Compartment syndrome of the foot after intraarticular calcaneal fracture. *Clin Orthop Relat Res* 1991; **Aug:** 241–48.

82 Dodd A, Le I. Foot compartment syndrome: diagnosis and management. *J Am Acad Orthop Surg* 2013; **21:** 657–64.

83 Brey JM, Castro MD. Salvage of compartment syndrome of the leg and foot. *Foot Ankle Clin* 2008; **13:** 767–72.

84 Ojike NI, Roberts CS, Giannoudis PV. Foot compartment syndrome: a systematic review of the literature. *Acta Orthop Belg* 2009; **75:** 573–80.

85 Myerson MS. Management of compartment syndromes of the foot. *Clin Orthop Relat Res* 1991; **Oct:** 239–48.

86 Han F, Daruwalla ZJ, Shen L, Kumar VP. A prospective study of surgical outcomes and quality of life in severe foot trauma and associated compartment syndrome after fasciotomy. *The J Foot Ankle Surg* 2015; **54:** 417–23.

87 Roberts CS, Gorczyca JT, Ring D, Pugh KJ. Diagnosis and treatment of less common compartment syndromes of the upper and lower extremities: current evidence and best practices. *Instr Course Lect* 2011; **60:** 43–50.

88 Hwang RW, de Witte PB, Ring D. Compartment syndrome associated with distal radial fracture and ipsilateral elbow injury. *J Bone Joint Surg Am* 2009; **91:** 642–45.

89 Dyer G, Lozano-Calderon S, Gannon C, Baratz M, Ring D. Predictors of acute carpal tunnel syndrome associated with fracture of the distal radius. *J Hand Surg Am* 2008; **33:** 1309–13.

90 Kim JY, Schierle CF, Subramanian VS, et al. A prognostic model for the risk of development of upper extremity compartment syndrome in the setting of brachial artery injury. *Ann Plast Surg* 2009; **62:** 22–27.

91 Ouellette EA, Kelly R. Compartment syndromes of the hand. *J Bone Joint Surg Am* 1996; **78:** 1515–22.

92 Shore BJ, Glotzbecker MP, Zurakowski D, Gelbard E, Hedequist DJ, Matheney TH. Acute compartment syndrome in children and teenagers with tibial shaft fractures: incidence and multivariable risk factors. *J Orthop Trauma* 2013; **27:** 616–21.

93 Flynn JM, Bashyal RK, Yeger-McKeever M, Garner MR, Launay F, Sponseller PD. Acute traumatic compartment syndrome of the leg in children: diagnosis and outcome. *J Bone Joint Surg Am* 2011; **93:** 937–41.

94 Blakemore LC, Cooperman DR, Thompson GH, Wathey C, Ballock RT. Compartment syndrome in ipsilateral humerus and forearm fractures in children. *Clin Orthop Relat Res* 2000; **Jul:** 32–38.

95 Prasarn ML, Ouellette EA, Livingstone A, Giuffrida AY. Acute pediatric upper extremity compartment syndrome in the absence of fracture. *J Pediatr Orthop* 2009; **29:** 263–68.

96 Grottkau BE, Epps HR, Di Scala C. Compartment syndrome in children and adolescents. *J Pediatr Surg* 2005; **40:** 678–82.

97 Kanj WW, Gunderson MA, Carrigan RB, Sankar WN. Acute compartment syndrome of the upper extremity in children: diagnosis, management, and outcomes. *J Child Orthop* 2013; **7:** 225–33.

98 Bibbo C, Lin SS, Cunningham FJ. Acute traumatic compartment syndrome of the foot in children. *Pediatr Emerg Care* 2000; **16:** 244–48.

99 Silas SI, Herzenberg JE, Myerson MS, Sponseller PD. Compartment syndrome of the foot in children. *J Bone Joint Surg Am* 1995; **77:** 356–61.

100 Rorabeck CH, Macnab I. The pathophysiology of the anterior tibial compartmental syndrome. *Clin Orthop Relat Res* 1975; **Nov–Dec:** 52–57.

101 Matsen FA 3rd, Mayo KA, Sheridan GW, Krugmire RB Jr. Continuous monitoring of intramuscular pressure and its application to clinical compartmental syndromes. *Bibl Anat* 1977; **(15 Pt 1):** 112–15.

102 Wall CJ, Lynch J, Harris IA, et al, and the Liverpool (Sydney) and Royal Melbourne Hospitals. Clinical practice guidelines for the management of acute limb compartment syndrome following trauma. *ANZ J Surg* 2010; **80:** 151–56.

103 Prasarn ML, Ouellette EA. Acute compartment syndrome of the upper extremity. *J Am Acad Orthop Surg* 2011; **19:** 49–58.

104 Bhattacharyya T, Vrahas MS. The medical-legal aspects of compartment syndrome. *J Bone Joint Surg Am* 2004; **86-A:** 864–68.

105 Tollens T, Janzing H, Broos P. The pathophysiology of the acute compartment syndrome. *Acta Chir Belg* 1998; **98:** 171–75.