Chapter 20
Pathophysiology and Management of Limb Compartment Syndromes

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Key Learning Points

- Acute extremity compartment syndrome is a surgical emergency associated with significant morbidity if not expeditiously managed.
- The most important tool in diagnostics is to maintain a high level of clinical suspicion.
- Patients with classical clinical signs of compartment syndrome do not need any further investigation, and should undergo urgent fasciotomy.
- Incisions in skin and fascia need to be long enough to make tissues loose and allow for postoperative swelling.
- After compartment syndrome has resolved, delayed primary closure of the fasciotomy incisions is performed, with the intracutaneous suture method recommended.

20.1 Introduction

Acute extremity compartment syndrome is a surgical emergency associated with significant morbidity if not expeditiously managed. It requires prompt diagnosis and early treatment with compartment decompression for good clinical outcome.

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The definition of acute extremity compartment syndrome is an increased pressure within the compartmental space leading to decreased perfusion pressure and hypoxaemia of the tissues. This may lead to irreversible ischaemic necrosis of the muscles and nerves in the compartment causing functional impairment, limb amputation, multiple organ failure, or death [1, 2].

Acute limb compartment syndrome (ACS) in vascular surgery is mostly related to ischemia-reperfusion (I/R) injury associated with acute ischaemia, vascular trauma, and phlegmasia cerulea dolens [3–5]. Iatrogenic causes with accumulation of blood or contrast fluid within the compartment after catheter/wire perforation have also been described [3, 4]. Most extremity compartment syndromes result from internal compartment expansion due to fractures or crush injuries but also external compression from burns, tight plaster cast or bandage may occur [6]. The aim of this chapter is to summarise the pathophysiology and management of upper and lower limb compartment syndromes.

20.2 Epidemiology

No comprehensive review of the prevalence of extremity compartment syndrome has been published due to the variation of causes [7]. The incidence of lower extremity compartment syndrome after revascularization of acute ischaemic limbs is approximately 10–20% [5, 8, 9], but in contrast, the need for fasciotomy after elective vascular surgery is very low, from 0.15 to 0.45% [10]. Patman found that 32% of extremities with arterial injuries and only 2% with embolic occlusions underwent fasciotomy. In a retrospective series of vascular surgery patients (107 patients; 113 limbs) undergoing lower extremity fasciotomy, 72% of limbs underwent revascularization for acute limb ischaemia, 6.2% of limbs were related to acute aortic disease, and 20% of limbs had undergone elective vascular surgery [2]. In total, 57% of limbs had signs of ACS and a therapeutic fasciotomy was performed, while 43% fasciotomies were prophylactic. Data from the US National Trauma Data Bank showed that patients sustaining lower extremity arterial trauma required a fasciotomy in up to 42% of cases [11].

20.3 Pathophysiology

Skeletal muscles, nerves, and vessels in the upper and lower extremities are arranged into compartments surrounded by fascia. The upper arm contains two compartments (anterior and posterior) and the forearm has three (volar, lateral, and dorsal) compartments. In the thigh, there are three compartments (anterior, medial, and posterior), whereas there are four in the lower leg (anterior, lateral, deep posterior, and superficial posterior) [6, 12]. Compartments within the forearm and lower leg are especially limited in their ability to accommodate tissue oedema and are therefore

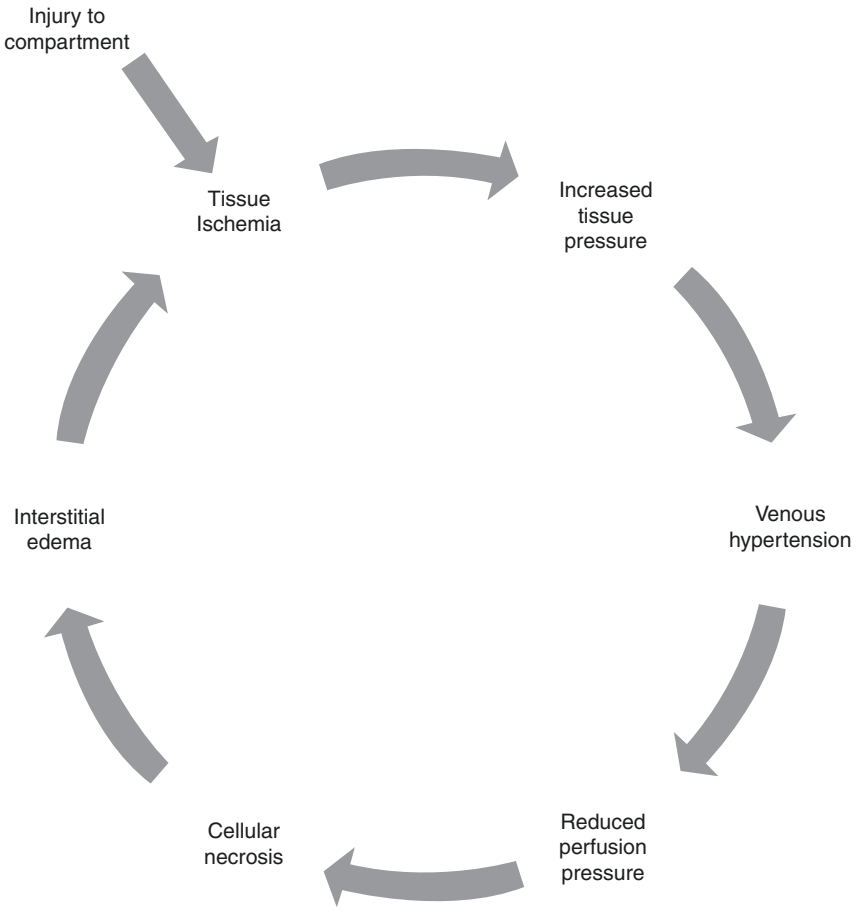

Fig. 20.1 Pathophysiology of acute extremity compartment syndrome [1, 14–16]

more prone to develop increased compartment pressures [13]. The lower leg is the most common location of acute extremity compartment syndrome, with the anterior and lateral compartments most frequently affected [12].

The pathophysiology of acute extremity compartment syndrome involves an external compression or an internal expansion within the compartment that leads to increased tissue pressure, reduced capillary blood flow, local tissue hypoxia and local tissue necrosis (Fig. 20.1) [1, 14]. Intrinsic causes of acute extremity compartment syndrome are tissue injury caused by a direct traumatic event or tissue ischaemia and reperfusion [1, 15]. The most common cause of compartment syndrome in vascular surgery is tissue oedema due to ischaemia-reperfusion injury caused by limb revascularisation. A traumatic vascular injury is often accompanied by fractures, so that ischaemia–reperfusion and haematoma formation may both contribute to the limb compartment syndrome [16]. The rising tissue pressure

impairs venous outflow by compressing the veins. This increase in venous pressure reduces the arteriovenous pressure gradient, resulting in diminished local perfusion [1, 15, 16].

The combination of increased pressure in the interstitial and intercellular fluid spaces shuts off transcapillary movement. The resultant cellular ischaemia leads to muscle and nerve damage. Interstitial oedema develops from tissue necrosis and further worsens hypoxaemia and compartmental swelling. During acute limb ischaemia, the combination of decreased oxygen supply and congestion of red blood cells within the capillaries triggers a complex cascade of metabolic, inflammatory, and prothrombotic pathways [17, 18]. The cell shifts energy metabolism from an aerobic to anaerobic mechanism, producing lactic acid. Continued ischaemia causes depletion of energy-rich adenosine triphosphate (ATP), leading to leakage of extracellular calcium into the muscle cells, which ultimately results in dysfunction and cell death [19].

Reperfusion injury represents the response to tissue injury when the blood flow is restored after ischaemia. This contributes to a systemic inflammatory, metabolic, and thrombotic response. Microvascular dysfunction mediates many of the local and systemic consequences of ischaemia-reperfusion injury with a plethora of changes specific to arterioles, capillaries, and venules [17, 20]. This includes impaired vasodilation, decreased perfusion and fluid leakage, and increased permeability.

Ischaemia-reperfusion injury leads to microcirculatory changes due to activation of inflammatory mediators. Increased arterial resistance leads to decrease perfusion in the capillaries. The permeability results in an increased rate of transcapillary fluid leakage. Impaired tissue perfusion and oedema will further raise the intracompartmental pressure [17, 20]. The no-reflow phenomenon is another factor in this response relating to vascular congestion in the arterioles and capillaries [13, 20]. Reactive oxygen species cause damage including injury to DNA, oxidation of fatty acids and lipids, and oxidation of proteins and co-factors necessary for enzymatic function [21]. This leads to capillary leakage, resulting in additional oedema and rising compartment pressure. (see Chap. 18, Pathophysiology of Ischaemia-Reperfusion injury).

The duration of ischaemia correlates with the onset of irreversible changes in various tissue types. The critical ischaemic time in different tissues at normal temperature can be defined as the maximum ischaemic time interval that a tissue can tolerate and still remain viable [22]. In the extremities, muscle tissue has the highest risk for ischaemia-reperfusion injury [17].

An ischaemic time of 4–6 h predisposes the patient to the development of a compartment syndrome [23]. Large-animal studies have demonstrated the neuromuscular ischaemic threshold of the limb to be less than 5 h, and similarly recommended restoration of flow within 3 h of injury for optimal functional recovery. Haemorrhagic shock also worsens the impact of ischaemia on the neuromuscular structures of the limb and reduces the ischaemic threshold to as little as 1 h [13].

20.4 Clinical Presentation

Acute limb compartment syndrome occurs after a precipitating causal event, most commonly trauma. In vascular surgery, compartment syndrome is also commonly seen following reperfusion after muscular ischaemia due to acute arterial occlusion or reduced distal perfusion during endovascular procedures or extracorporeal membrane oxygenation (ECMO) due to large catheters inserted into proximal arteries. Other contributing factors are fractures, haematomas, pressure injuries, and oedema. The time from insult to compartment syndrome development may vary from minutes to hours. The most sensitive tissues to ischaemia are unmyelinated nerve fibres followed by myelinated nerve fibres, skeletal muscle, skin and then bone. As an example, the first sign might be an altered sensation between digit 1 and 2 in the foot—corresponding to deep peroneal nerve ischaemia in the anterior compartment. Pulses will generally be preserved for a long time into the syndrome, well beyond the indication for acute surgery. Patients who are alert and awake normally complain of severe pain, often resistant to analgesics, sometimes pain "out of proportion"—meaning that it does not correspond to the severity of the injury sustained. Some of the other classical "P-signs" of compartment syndrome might also be present (pain, pain on passive stretch, paraesthesia, paralysis). Commonly foot drop occurs in compartment syndrome affecting the lower leg, due to the involvement of anterior and lateral calf compartments. In general, the sensitivity of each symptom is low but the specificity quite high. Absence of symptoms should therefore not be used to rule out compartment syndrome. But additionally, if three or more of the classical "4-P" symptoms are present, this increases the likelihood of the presence of a compartment syndrome [24].

Unfortunately many patients are not fully awake and alert when the syndrome occurs. This might be due to anaesthetics used in surgery, dementia, concomitant CNS or peripheral nerve injuries or drugs and alcohol. For this group of patients, firmness/tenderness of the compartments relative to the unaffected limb might be a helpful observation. The assessment of distended muscles alone is not sufficient for diagnosing increased compartment pressure [25]. In children, the adult symptoms described above are unreliable; signs of increasing need of analgesics, agitation and anxiety may be the clues to diagnosis and treatment [26, 27]. It should be noted that the syndrome usually evolves over some time, which is why repeated, serial clinical examinations over a short period of time sometimes makes it easier to diagnose. A sensory deficit is sometimes the first sign of compartment syndrome, but, in vascular surgery, the preceding ischaemic event may already have caused sensory loss, reinforcing why it is important not to rely only on a single clinical sign.

Clinical signs suggestive of irreversible ischaemic damage include fixed, non-blanching skin staining or gangrene. In these cases, urgent amputation should be considered to prevent systemic complications. Factors associated with post-ischaemic compartment syndrome in non-traumatic acute limb ischaemia are inadequate backflow, high serum creatine kinase (CK) level, positive fluid balance after

admission, and advanced-stage acute limb ischaemia [28]. In cases in which muscle swelling is highly likely, a prophylactic fasciotomy may be indicated. It may reasonable to perform a prophylactic fasciotomy in severe acute ischaemia exceeding 4–6 h, especially if there is inadequate collateral flow, and also in association with vascular trauma [9, 28].

20.5 Diagnostic Testing

Any suspicion of acute limb compartment syndrome (both in conscious and unconscious patients), must lead to either prompt treatment or immediate further diagnostic testing. Clinical assessment of suspected compartment syndrome is difficult; the majority of symptoms and signs are only reliably assessed in a fully conscious patient. Also, many patients may have some of these clinical signs present due to the injury that has caused the compartment syndrome such as the pulseless, painful, paraesthetic limb of acute ischaemia. The most important tool in diagnostics is to maintain a high level of clinical suspicion. A clinical situation with long-standing ischaemia, all the classical "P signs", and a firm and tense calf does not need any further investigations, and should undergo urgent fasciotomy. On the other hand, an unconscious patient in the intensive care unit with slightly firm calf after a short period of ischaemia should be worked-up more thoroughly before going ahead with immediate fascia release. The medicolegal and litigation situation in some areas of the world makes it wise to try to confirm the diagnosis if possible. The most commonly used method for confirmation of the diagnosis is pressure measurement in the respective compartment with multiple needle-sticks. All compartments in the affected limb should be measured. If any fracture is present, measurement should be done within five centimetres of the fracture. Some sedation or local anaesthetic might be helpful, especially in children. Most accurate results are reported with a side-port needle or a slit catheter connected to an arterial line or the Stryker manometer [29]. The normal compartment pressures in adults are around 8 mm Hg and in children 10–15 mmHg [1]. As a diagnostic cut-off, the perfusion pressure has been proven to be more accurate than an absolute value. The differential pressure (ΔP = diastolic blood pressure − intracompartmental pressure) of 30 mmHg is the most commonly used threshold [30, 31]. Collecting blood from the ipsilateral femoral vein and measuring lactate has also been shown to correlate with the risk of compartment syndrome but the clinical usefulness is not clear [32].

The dynamics of the syndrome is of importance; any measurement during surgery and anaesthesia should be repeated postoperatively if suspicion exists. Regional anaesthesia such as epidural catheters should be used with caution since they may mask symptoms and delay diagnosis [33]. Techniques with indwelling catheters allow continuous monitoring of limbs at risk, but these techniques are more complex and require prior training in their use [34].

Other methods to diagnose acute compartment syndrome have been proposed as well. Near-infrared spectroscopy (NIRS) has been useful in some case series [35]

but according to a recent review, the false-negative rate is too high to rely solely on NIRS in diagnosing a syndrome that needs timely treatment [36, 37]. In clinically advanced cases, the resulting inflammation may be associated with a leucocytosis or C-reactive protein (CRP) elevation. If tissue necrosis has developed, serum creatinine phosphokinase and myoglobin will be elevated, together with a metabolic acidosis. When rhabdomyolysis occurs, the urine will develop a brown discolouration, sometimes referred to as "tea-coloured". The colour stems from myoglobin in the urine which the kidneys are trying to excrete.

20.6 Management

20.6.1 Lower Extremity Operative Techniques

Acute limb compartment syndrome should be treated with fasciotomy of all affected compartments. In patients who present acutely (roughly within 12 h of onset of compartment syndrome), it should be performed immediately. Fasciotomy performed longer than 12 h after the onset of compartment syndrome results in a significantly poorer functional outcome [37, 38]. If the presentation or diagnosis is delayed, consideration must be given as to whether the limb is unsalvageable. If so, a fasciotomy may lead to significant morbidity without improving functional outcome. Beyond 36 h, rates of amputation, infection, neurological injury and death increase. Early amputation rather than futile attempts at limb salvage should be considered [39, 40].

Preoperative non-surgical measures such as removal of casts and dressings and elevation of the affected limb, should be performed during evaluation or waiting time for surgery. In the lower limb, the fasciotomy procedure should release any tissue that could constrict the respective compartment. The skin and fasciae should be released along the entire length of the compartments. The lower limb consists of the anterior, lateral (peroneal), superficial and deep posterior compartments. The easiest and most common way to decompress the compartments is through two incisions (Fig. 20.2). One posteromedial incision (to open the superficial and deep

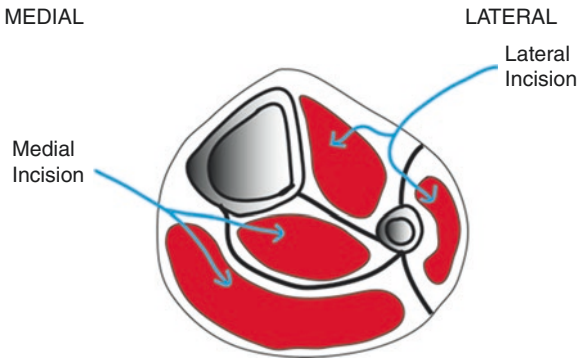

Fig. 20.2 Fasciotomy of the lower limb to decompress the four compartments (anterior; lateral; superficial and deep posterior) with two incisions (anterolateral and posteromedial)

posterior compartments) is performed two centimetres behind the medial border of the tibia; taking care not to injury the great saphenous vein. The superficial fascia is then opened along the length of the skin. Secondly, the soleus muscle attachment on the tibial border is sharply released to expose the deep posterior fascia which is then opened. The second anterolateral incision (to open the anterior and lateral compartments) is done one fingerbreadth anterior to the fibula from the fibular head to the ankle over the intermuscular septum between the anterior and lateral compartment, taking care not to injure the common and superficial peroneal nerves. The intermuscular septum can usually be seen as a white longitudinal line in the fascia. If any uncertainty exists, a short transverse incision can be used to confirm the location of the septum. The anterior and lateral (peroneal) compartments are then opened along the length of the incision. A single incision technique to treat lower limb compartments has also been used with similar outcomes but it is not recommended by the authors [41]. Irrespective of techniques used, incisions in skin and fascia need to be long enough to make the tissues loose and allow for postoperative swelling [42]. Any dressing material should be sterile and loosely attached. A simple and cheap way to prepare for later closure is to suture a running intracutaneous shoelace suture along the whole incision. The suture can be a 2/0 resorbable monofilament suture or a non-resorbable polypropelene suture. It is important to leave the sutures really loose; normally two sutures are needed to cover a lower limb incision.

Compartment syndrome in the thigh is rare, most commonly seen after blunt trauma. The thigh consists of three compartments (anterior, posterior and medial) and is usually decompressed medially and/or laterally (Fig. 20.3). No good data on the optimal method is available although a single-incision technique is reported more frequently [43].

20.6.2 Upper Extremity Operative Technique

In the upper extremity, the forearm is the most common site of development of compartment syndrome [1, 4]. Decompression of the volar and the lateral compartments is achieved through a curvilinear incision that begins proximal to the antecubital fossa, medial to the biceps tendon, extends to the radial side of the forearm,

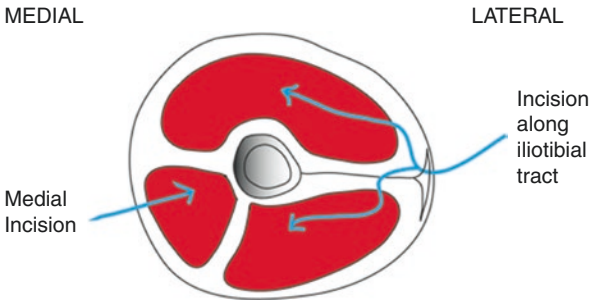

Fig. 20.3 Fasciotomy of the three compartments (anterior, posterior and medial) in the thigh with decompression from a medial and lateral approach

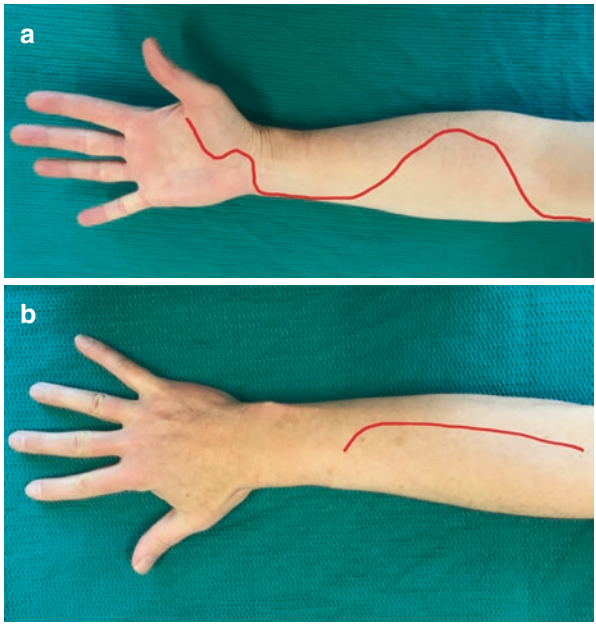

Fig. 20.4 Fasciotomy of the forearm with decompression of the volar and lateral compartments (**a**) and the dorsal compartment (**b**)

where it goes distally along the medial border of the brachioradial muscle, and finally across the carpal tunnel along the thenar crease (Fig. 20.4a) [3, 4, 6, 12]. The decompression of the dorsal compartment is achieved through an incision extending from just distal to the radial head to the midportion of the wrist (Fig. 20.4b). In the upper arm, the anterior compartment is incised just distal to the deltoid muscle to just above the elbow. The posterior compartment is incised over the triceps muscle in the midline to above the elbow [14].

20.7 Postoperative Care

The postoperative limb should be held elevated and a foot pump may be used if available. Physiotherapy should be started immediately to activate dorsiflexion of the ankle. Pain medication should be administered as needed. There are a few options for dealing with the fasciotomy wounds after the compartment syndrome has resolved. Delayed primary closure, vacuum-assisted closure, secondary closure, skin grafts, and healing by secondary intention are all options. Delayed primary closure with the vessel loop shoelace technique is often referred to and works well [44] but the authors would recommend the pre-positioned intracutaneous suture method described above which is easier, quicker and gives a better cosmetic outcome. Studies with vacuum-assisted closure have shown conflicting results, with low complication rates but the need for skin grafting has been quite frequent, as well

as high costs associated with the technique [45–47]. If the running sutures, described in the operative technique above, are used; the same sutures can be used to start closing and approximating the skin edges. If the fasciotomy was done prophylactically and no swelling is present, complete skin closure can be performed within 24–48 h. If a true compartment exists with muscular swelling, the successive closure process can usually start after 48–72 h. This can normally be done in the ward without the need to go back to the surgical theatre unless there is a need for surgical debridement or haemostasis. Most patients tolerate careful pulling of the sutures every 24–48 h. When the skin edges are closed, the suture is tied on the outside and left in situ for 3 weeks.

Some adjunctive non-operative management methods have been described but there are no standard methods in use. Free radical scavengers such as mannitol [48] and hypertonic saline [13] have been shown to be of some benefit in compartment syndrome caused by ischaemia-reperfusion injury. Hyperbaric oxygen therapy has also been shown to improve the outcome of reperfusion injuries [49]. The addition of sodium bicarbonate to the resuscitation fluids is beneficial, as it will correct metabolic acidosis, hyperkalaemia and also keeps the urine alkaline (urinary pH >6.5), thus decreasing the toxic effect of myoglobin on renal tubules. Forced diuresis with furosemide and mannitol may also help protect the kidneys [50]. If rhabdomyolysis occurs, continuous renal replacement therapy may be used, although a Cochrane analysis found that the supporting evidence is weak [51].

20.8 Outcome

Whilst fasciotomy wounds are associated with a moderate degree of morbidity [52, 53], fasciotomy does not appear to have any effect on long-term calf muscle pump function [54]. If fasciotomy is performed early for acute compartment syndrome, the outcome in terms of preventing limb loss, systemic complications and long-term functional disability is good [31]. Failure to prevent and promptly treat compartment syndrome risks the development of systemic complications such as multiorgan failure (including hyperkalaemia, hypocalcaemia, coagulopathy, myoglobinuria, and renal failure) with a correspondingly high risk of death. It is recommended that a fasciotomy should be performed as soon as possible once the diagnosis has been made, preferably within 6 h [55]. Fasciotomy performed early, less than 12 h after the onset of the compartment syndrome, resulted in normal function in 68% of the extremities. However, only 8% of those having late fasciotomy had normal function following decompression [38].

To study the outcome of compartment syndrome alone is difficult, since simultaneous vascular or traumatic injury may affect the outcome. The syndrome does have a high incidence of nerve deficit and foot drop (15–32%), [2, 56] and mortality rates of between 15 and 23% have been reported [2, 57]. In a retrospective study from 1980 to 1994, 53 patients had fasciotomies following surgical revascularization [5]. At discharge, 11 (21%) patients had undergone amputation and six (11%) patients

had died. In another retrospective review, lower extremity fasciotomies were performed for compartment syndrome after acute ischaemia and revascularization in patients with vascular trauma or arterial occlusive disease [53]. Fourteen (11%) amputations were required for refractory limb ischaemia and 18 (15%) patients died of cardiopulmonary or multisystem failure. After lower extremity trauma with compartment syndrome and fasciotomy, nearly 13% of patients required leg amputation [56]. In this study by Lollo et al., 67% of amputees had associated vascular injuries. At long-term follow-up, 10% of patients reported moderate lower extremity pain and 69% had returned to work. Neuromuscular sequelae, as well as long-term functional outcome, has been emphasized as important factors for patient expectation and satisfaction [13, 23]. Most children experience full recovery after fasciotomy but the literature is limited [58]. In a case series, persistent Volkmann's contracture as a post-traumatic complication occurred in one of 23 children (4.2%) [58].

20.9 Conclusion

Extremity compartment syndrome in vascular surgery is a limb-threatening condition requiring emergent fasciotomy. The management strategy is to expeditiously decrease tissue pressure, minimize tissue damage and functional loss. There are significant complications related to lower extremity compartment syndrome including neuromuscular dysfunction and amputation. The fasciotomy wounds per se cause minimal morbidity and can normally be closed with a variety of techniques. The role of inflammatory and metabolic biomarkers for compartment syndrome and whether they can guide therapy needs further evaluation. Future research should focus on early and improved diagnostics of compartment syndrome and refined treatment regimens for ischaemia-reperfusion injury.

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Further Reading

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