Chapter 8
Compartment Syndrome of the Lower Extremity

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Introduction

Acute compartment syndrome is a surgical emergency that can threaten life and the limb. Moreover, lower extremity compartment syndrome is most commonly associated with high-energy mechanisms of injury; however, a high index of suspicion should be maintained with low-energy or penetrating trauma, vascular or crush injuries, and prolonged periods of immobility. Rare presentations are even documented in association with diabetes mellitus, hypothyroidism, malignancy, viral-induced myositis, nephrotic syndrome, and bleeding disorders [1]. Most practitioners associate lower extremity compartment syndrome with the leg, but other sites including the buttock, thigh, and foot can develop the same pathology. Serial physical examinations by an experienced provider remain the best tool for accurate diagnosis, while intramuscular compartment pressure measurements are best used as an adjunct especially when a complete physical examination is not possible. Compartment syndrome, unlike many musculoskeletal conditions, is much easier to treat than to accurately diagnose. Prompt fasciotomies with release of all involved muscular compartments prevent the life- and limb-threatening sequelae of a missed compartment syndrome. Although fasciotomies are associated with increased blood loss and elevated risk of infection and commonly require split-thickness skin grafts in lieu of closure, they prevent irreversible ischemic tissue loss and potential for lifetime disability. We hope the reader uses this chapter to assist in diagnosis and treatment of patients with potential compartment syndrome.

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Pathophysiology

Compartment syndrome is the result of fascial compartment pressures surpassing perfusion pressure, causing tissue ischemia and eventual necrosis [2]. After a local insult, traumatic or others, volume increases to a compartment from bleeding or inflammation, leading to the onset of local tissue edema as a result. Fascial compartments in the body have finite volumes with limited ability for elastic expansion, so pressure levels correspondingly increase. Heightened tissue pressure corresponds with an increase in venous pressure, thus decreasing the arteriovenous gradient. As this gradient decreases, microvascular flow through capillaries falls, and inadequate perfusion of the tissue eventually results, causing ischemic changes to the tissues. Permanent damage to muscle tissue may result shortly after 4–8 hours of ischemia [3, 4]. Anoxic damage to endothelial cells results in further increases to vessel wall permeability that, along with decreased venous outflow, perpetuates the local edema and pressure increases. Eventual muscle necrosis leads to the release of myoglobin into the blood with associated metabolic acidosis and hyperkalemia. Severity is based on extent of muscle compartments involved and duration of the ischemic changes. The worst of cases may potentiate cardiac arrhythmias, renal failure, shock, or hypothermia. Fasciotomies remove the volume limitations to the compartment, drastically altering the pressure gradients with the goal of restoring tissue perfusion.

Medical Management and Missed Compartment Syndromes

All patients presenting after high-energy mechanisms of injury should be promptly evaluated using Advanced Trauma Life Support (ATLS) protocols to identify and treat life-threatening injuries [5]. Patients presenting after prolonged immobilization should be evaluated according to their signs and symptoms. Ischemic reperfusion events can occur following vascular injuries or prolonged compression events. Bywater's or crush syndrome is a traumatic rhabdomyolysis where cellular necrosis from a crush injury results in increased serum myoglobin and potassium, leading to acidosis and kidney failure [6]. Fluid resuscitation with consideration of added sodium bicarbonate helps to dilute increased myoglobin and urea concentrations from muscle necrosis and counteract the associated metabolic acidosis, thereby limiting acute tubular necrosis and renal dysfunction [6].

A Foley catheter is recommended to ensure adequate fluid resuscitation and monitor renal failure. Dark, tea-colored urine is suggestive of myoglobinuria and ongoing rhabdomyolysis [6]. Serial serum myoglobin levels may also be of benefit for diagnosis and guidance of need for ongoing fluid resuscitation [7]. Myoglobin levels may be elevated in trauma patients without acute compartment syndrome, especially those with muscle injury at multiple sites throughout the body. Increasing levels of myoglobin with declining renal function should alert the provider that

muscle necrosis is ongoing and prompt fasciotomy with debridement of necrotic muscle, while decreasing myoglobin levels can be reassuring to the provider.

A type and screen for potential blood transfusion is recommended as patients who undergo fasciotomy often bleed from the injured tissues, open wounds, and/or negative pressure wound therapy (NPWT). Blood transfusion should also be an early component of the resuscitation of the trauma patient in shock [8].

Missed compartment syndromes provide a treatment conundrum for the surgeon as opening a closed necrotic compartment may introduce a significant risk for deep infection and its sequelae. These are usually patients who have had ongoing pain for several hours that spontaneously resolves without fasciotomy. Neurologic and potential vascular compromise results in soft tissue death and potential limb loss. However, it is difficult for the surgeon to know if some muscle in the compartment can be salvaged without direct intraoperative examination of the muscle. Computed tomography (CT) scanning provides soft tissue windows that might alert the surgeon for any abnormalities in the musculature and the extent of myonecrosis. Worsening renal function, despite adequate fluid resuscitation, also forces the surgeon's hand toward fasciotomy for debridement of necrotic musculature which is a source of myoglobin, potassium, and tissue thromboplastin [6]. Complications as a result can vary based on the extent of necrosis but can include cardiotoxicity, disseminated intravascular coagulation, renal failure, and sepsis, leading to multiorgan failure or death. Observation without fasciotomy should be reserved for patients without signs of sepsis or worsening renal function that present to the surgeon with no ongoing pain.

Intracompartmental Pressure Measurements and Continuous Monitoring

The use of intracompartmental pressure measurements and continuous monitoring remains controversial. For the awake and alert patient, serial physical examinations remain the best diagnostic methods with the "one P," pain, being the hallmark symptom of compartment syndrome. An awake and alert patient with signs and symptoms of compartment syndrome concerning enough to undergo pressure measurements should probably just be taken to the operating room for emergent fasciotomy.

Intracompartmental pressure monitoring is especially useful in patients obtunded due to illicit substances or traumatic brain injuries; however, these measurements remain imperfect and lack specificity. Measurements can be made with commercially available devices with a side-port needle catheter or with an arterial line setup [9]. Thresholds for absolute pressure and ΔP (diastolic blood pressure – absolute compartment pressure) have been set to prevent any missed compartment syndrome but may lead to unnecessary fasciotomy in many patients. In a prospective study, patients with tibia fractures undergoing planned intramedullary nailing were

evaluated with four compartment preoperative pressure measurements. There was no clinical suspicion for compartment syndrome for any of these patients up to the time of surgery. However, measurements meeting an accepted threshold for fasciotomy at absolute pressure >= 40 mm Hg or ΔP <= 30 mmHg were present in 35% of patients. These patients were followed for 6 months with no signs of missed compartment syndrome [10]. This point is reiterated in another prospective study of diaphyseal tibia fractures with continuous pressure monitoring where an absolute pressure threshold of 30 mmHg or 40 mmHg would have led to 43% and 23% of unnecessary fasciotomies, respectively. This study recommended the ΔP <= 30mmHg as the best indication for fasciotomy and also highlighted one-time measurements do not preclude subsequent development of a compartment syndrome [11]. Continuous monitoring may address this limitation but requires many hospital resources that may include an intensive care or intermediate care bed and may not change the ultimate outcomes. Challenges with pressure measurements include low adherence to proper technique and substantial decreases in accuracy of measurement even with small errors in technique [12]. Overall, intracompartmental pressure monitoring should not be used not only as a screening tool but also to give evidence to confirm clinical suspicion as needed [2].

Gluteal Compartment Syndrome

The gluteal region is a rare anatomic location for the development of compartment syndrome. Most cases will result from prolonged immobilization secondary to heavy drug/alcohol use or surgical positioning [13]. Thorough examination of all extremities should be performed in patients presenting to the hospital after prolonged immobilization. These patients may be obtunded secondary to alcohol and illicit drug use or potentially due to neurodegenerative disorders, thereby limiting a full history and physical examination. Prolonged surgeries in the lateral decubitus or lithotomy are the most implicated surgical positions, causing a gluteal compartment syndrome [14]. Traumatic injury and gluteal compartment syndrome comprise approximately 20% of cases [13]. These usually result from a crushing mechanism to the lower lumbar spine, pelvis, and buttocks area. Prolonged extrication time and/or crushing mechanism with heavy objects should alert the provider for any potential development. Additional causes may include vascular injury, epidural analgesia after total hip arthroplasty, anticoagulation, overuse or exertion, and necrotizing fasciitis infections [13]. The gluteal compartment syndrome is also frequently associated with the previously discussed crush syndrome, so holistic management to prevent renal failure and systemic complications of rhabdomyolysis is of paramount importance.

There are three compartments in the gluteal region: tensor fasciae latae (TFL), gluteus medius and minimus, and gluteus maximus. The gluteus maximus is the largest of the three and supplied by the inferior gluteal nerve and vessels. This muscle is the main extensor and external rotator of the leg, originating on the posterior

ilium and dorsal sacrum extending over the gluteus medius to join the posterior iliotibial tract. Deep and superolateral to the maximus resides the gluteus medius muscle. The medius originates on the ilium and inserts on the greater trochanter overlaying the gluteus minimus. The superior gluteal nerve and vessels supply the gluteus medius and minimus, and these muscles together form a single compartment lying between the maximus and TFL. The TFL is in its own compartment that originates on anterior iliac crest and anterior superior iliac spine blending distally with the iliotibial band in the proximal thigh. Although not directly contained within these compartments, the sciatic nerve is at risk for a compressive neuropathy due to excessive swelling of the gluteal muscles or traumatic hematoma [14].

Alert patients will present with severe pain in the gluteal regions and may complain of lower extremity paresthesias. Physical examination will reveal tense and painful buttocks to touch, possibly ecchymoses, and/or Morel-Lavallee lesions. Passive motion of adduction and flexion of the hip would exacerbate pain in the examinable patient as it counters the typical movements of the gluteal musculature decreasing compartment volumes.

As gluteal compartment syndrome is rare and examinations are occasionally limited, the surgeon may choose to use intracompartmental pressure measurements more frequently than other locations in the lower extremity. Optimal needle placement for compartment pressure measurements was studied using cadavers and found to be clear of neurovascular bundles [14]. The gluteus maximus needle should be placed 2 cm inferior and lateral to the posterior superior iliac spine. Advance the needle until contact of the iliac wing and withdraw approximately 4 mm to assure localization within the muscle belly. The needle directed toward the medius/minimus compartment is placed 2 cm inferior to the iliac crest in the middle third of the iliac wing. A similar advance and withdrawal technique is performed. Needle entry for the tensor fasciae latae compartment is placed 2 cm anterior and 3 cm distal to the tip of the greater trochanter. Penetration of the deep fascia should be easily felt, and additional 4 mm advancement ensures the needle is within the muscle belly.

Decompression can be performed with a posterolateral approach to the hip including a Kocher-Langenbeck or a Gibson. The Kocher-Langenbeck approach involves a curvilinear or angular incision beginning just caudal of iliac crest and lateral to PSIS, extending over the tip of the greater trochanter below along the anterolateral border of the femur. The Gibson approach differs in that the proximal portion of the incision is not directed as posteriorly and develops the interval between TFL and gluteus maximus muscles and allows for easier access to the TFL. All three gluteal compartments can be visualized and released with these approaches (Fig. 8.1). On release, evaluation of the muscle for color, contractility, consistency, and capacity to bleed should guide further debridement decisions. These approaches also allow for exploration and neuroplasty of the sciatic nerve, which should be performed in each case, especially in a patient with preoperative paresthesias or motor dysfunction in its distribution. Plans typically include additional operations for repeat inspections, debridements, and delayed closure.

Delay in diagnosis and decompression can result in permanent disability. Urgent surgical decompression can drastically improve chances for a full recovery, but

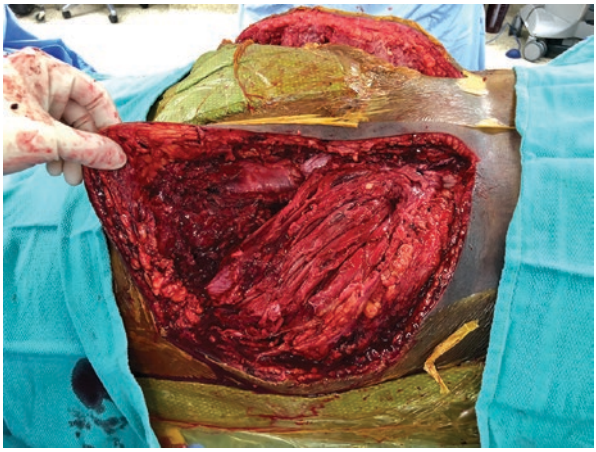

Fig. 8.1 Debridement of necrotic gluteal muscle, sequelae from a gluteal compartment syndrome after pelvic trauma. (Photo courtesy of Dr. Chip Routt)

long-term outcomes include chronic hip abductor weakness with Trendelenburg gait or potentially sensory and motor changes to the foot [13]. The treatment of a missed gluteal compartment syndrome is controversial but should depend more upon the systemic condition of the patient, rather than the presence of dead musculature in the buttock. Debridement is not mandatory if the patient does not have septic myonecrosis or rhabdomyolysis and renal insufficiency.

Thigh Compartment Syndrome

Thigh compartment syndrome is typically the result of blunt trauma with motor vehicle and motorcycle collisions being the most frequent causes. In a 2010 review, they found 90% of cases attributed to blunt trauma with 44% having associated to femur fractures [15]. Other causes include gunshot wounds, arterial injuries, coagulopathies or anticoagulant therapy, burns, overexertion, reperfusion swelling, or external compression.

The thigh has three anatomical compartments: anterior, posterior, and medial. The anterior compartment includes the sartorius and quadriceps, which are all innervated by the femoral nerve. The proximal portions of the femoral artery and vein also pass through this compartment, deep to the sartorius muscle, until they pass through Hunter's canal distally. The posterior compartment includes biceps femoris, semimembranosus, semitendinosus, and the sciatic nerve. The popliteal vascular bundle passes from medial to posterior in the distal third of the thigh. The medial compartment is composed of adductors longus, magnus, and brevis as well as the gracilis muscle and the obturator neurovascular bundle.

Diagnosis of compartment syndrome in alert patients generally only needs physical examination. Physical findings are similar to other body parts including pain out of proportion, tense compartments, pain with passive stretch, and associated

neurovascular changes. Muscle compartments can be tested individually with passive movements to decrease compartment volume, thereby exacerbating pain symptoms. Passive hip abduction tests the adductor compartment, knee flexion the anterior compartment, and knee extension the posterior compartment. Some surgeons may choose to use compartment pressure measurements to confirm the diagnosis in an alert patient or to make the diagnosis in an obtunded patient.

Treatment of a thigh compartment syndrome is a fasciotomy of the compartments. The anterior compartment is the most commonly affected compartment, with medial compartment involvement being more rare. The fasciotomy can be performed through a single lateral incision, allowing access for the anterior and posterior compartments. The technique for the fasciotomy involves an extensive longitudinal incision from the greater trochanter of the femur to the lateral condyle of the distal femur. The iliotibial band is incised the length of the incision, and the vastus lateralis is reflected anteriorly from the intermuscular septum releasing the anterior compartment. Incising the intermuscular septum releases the posterior compartment; however, this incision should be made away from the femur to avoid compromise of the perforating arteries traveling near the bone. The medial compartment rarely requires release once the anterior and posterior compartments have been decompressed; however, if the medial compartment remains tense, a separate anteromedial incision must be made.

A systematic review of thigh compartment syndrome revealed the majority, 59%, of thigh fasciotomy wounds were able to be closed by delayed primary closure; however, approximately 25% required skin grafting [15]. Most thigh fasciotomy wounds require multiple debridements before soft tissues are stabilized for delayed closure or split-thickness skin grafting; the average in this review was 5 days after index procedure for eventual wound closure.

Significant rates of mortality and morbidity are associated with thigh compartment syndrome. Mortality rates approach 50% due to polytraumatized or infected patients, with overall complication rates are as high as 78% in one systematic review [16]. The diagnosis also has a high association with the development of renal failure due to crush syndrome. Over half of the fasciotomy wounds in surviving patients became infected in this review. Many patients have persistent sensory deficits, motor weakness, decreased range of motion, or chronic pain of the extremity. A study examining functional outcomes found that worse results were associated with time to surgical decompression of greater than 8 hours, age over 30 years, femur fractures, high initial injury severity scores, and presence of myonecrosis at time of fasciotomy [17]. A rate as high as 40% of patients have permanent quadriceps impairment after a femur fracture, with this study finding greater than 80% with persistent thigh weakness in patients with a femur fracture and thigh ACS. The majority of patients will never recover full thigh muscle strength and have long-term functional deficits [17]. Heterotopic ossification may also be frequently visible after ACS in the thigh, although its clinical impact varies depending upon the severity and location. Decompression within 8 hours led to significantly better outcomes with strength and functional testing further giving evidence for the role of prompt fasciotomies.

Leg Compartment Syndrome

Acute compartment syndrome of the lower leg is the most frequently encountered of any area on the body. Greater than one-third of compartment syndrome cases are attributed to tibial shaft fractures [18]. It can result from both high and low-energy trauma or even atraumatic causes. Motor vehicle- and motorcycle-related injuries are the most common culprit; however, crushing injuries, burns, falls, sporting injuries, penetrating trauma, exertion, and circumferential compression can all result in ACS. Sporting events such as football and soccer have shown a strong association with development of ACS, despite being considered lower-energy trauma [19]. The rationale being that a significant local injury and inflammatory response is inflicted on younger patients with higher muscle mass who are already in a state of exertion. These injuries may not be enough to disrupt the fascial boundaries of the leg and may place them at a higher risk of an ACS. Although open fractures do not prohibit the development of a compartment syndrome, the concept of "autodecompression" is suggested by studies that report a decreased risk of ACS with high-grade open tibia fractures [18]. Regardless of the mechanism, all patients with tibial fractures should be carefully monitored with serial examinations for the potential development of an acute compartment syndrome.

The lower leg has four fascial compartments: anterior, lateral, superficial posterior, and deep posterior. The anterior compartment is very commonly involved in ACS and contains the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and the deep peroneal nerve. The anterior tibial artery enters the anterior compartment through the interosseous membrane just distal to the proximal tibiofibular joint with a recurrent branch directed proximally near the tibial tubercle. The lateral compartment contains the peroneus (fibularis) longus and brevis muscles and the proximal portion of the superficial peroneal nerve, which becomes extrafascial in the middle or distal third of the leg. The superficial posterior compartment contains the medial and lateral heads of the gastrocnemius, the soleus, and plantaris muscles. The gastrocnemius muscles receive blood supply from sural branches of the popliteal artery, while the soleus is supplied by the popliteal, posterior tibial, and peroneal (fibular) arteries. The deep posterior compartment is home to the posterior tibial, flexor hallucis longus, and flexor digitorum muscles along with the posterior tibial and peroneal vessels and the tibial nerve.

Fasciotomies for ACS are often performed for all four compartments and may be done with a dual or single incision approach. The dual incision technique is most frequently used and includes anterolateral and posteromedial incisions. Regardless of the technique chosen, the anterolateral incision should be performed from the level of the proximal tibiofibular joint to the level of the distal tibiofibular joint to permit complete release and full visualization. The anterolateral incision is longitudinal and often 2–5 cm anterior to the fibular shaft or midway between the tibial crest and fibular shaft. It provides access to the anterior and lateral compartments. With the creation of subcutaneous soft tissue flaps, the intermuscular septum must be identified to ensure both compartments are released. A transverse incision may

be made that allows for excellent visualization of the septum prior to longitudinal release of both of the compartments. This process is often performed in the proximal third of the leg to minimize risk of damage to the superficial peroneal nerve. The anterior compartment is released along the entire length of the compartment halfway between the septum and the tibial crest. The lateral compartment is incised posterior to the septum in line with the fibular shaft and should continue distally until the tendinous portion of the peroneal muscles is visualized. Care must be taken to protect the superficial peroneal nerve as it exits the fascia in the middle or distal third of the exposure and identification and dissection prior to anterior and lateral compartment release is recommended (see Video 8.1).

The posteromedial incision is conducted approximately 2 cm posterior to the posteromedial border of the tibia. Again the longitudinal dissection is carried out throughout the length of the leg, and the superficial posterior compartment is released initially, with exception of the lateral head of the gastrocnemius muscle. The soleal bridge, located near proximal metadiaphyseal junction of the tibia, must be completely released to adequately expose and decompress the deep posterior compartment. The deep posterior compartment is then released from the back of the tibia and is the most commonly "missed" compartment when fasciotomies are performed. The surgeon may use a Cobb elevator along the posterolateral aspect of the tibia to release this compartment and visualization of the deep posterior compartment musculature ensures it has been released (see Video 8.2).

A treatment algorithm with primary release of the anterior and lateral compartments followed by intraoperative reassessment of the superficial and deep posterior compartmental pressures has been suggested to reduce the need for four compartment releases in every case [20]. Patients presenting with compartment syndrome were initially treated with a single full-length anterolateral incision with standard release of both anterior and lateral compartments. After release, the intracompartmental pressures of both the superficial and deep posterior compartments were rechecked. Using preoperative diastolic blood pressure values, patients with ΔP values greater than 30 mmHg failed to undergo additional fasciotomies of the posterior compartments. Close postoperative observation of the patients in this study revealed no sequelae of a missed posterior compartment syndrome.

Alternatively, a single lateral incision to release all four compartments can be performed. The parafibular incision is made from the head of the fibula to the ankle, with larger subcutaneous tissue flaps created. Initial dissection is superficial to the lateral compartment, which should be followed anteriorly where the anterior intermuscular septum is identified. The anterior and lateral compartments are released similar to the anterolateral approach from the double incision technique. Next, by mobilizing the peroneal muscles anteriorly, the posterior intermuscular septum is identified which separates lateral and superficial posterior compartments. The posterior intermuscular septum joins with the transverse intermuscular septum, inserting on the posterolateral border of the fibula. Incision of these membranes and blunt elevation of the flexor hallucis longus from the posterior fibula lead to the release of the deep posterior compartment. This fascia should be completely opened and confirmed by passively moving the great toe, which can be felt in the muscle

belly of the dissection. Lastly, the superficial compartment is released either by incising the posterior intermuscular septum between soleus and peroneal muscles or by retraction of a posterior subcutaneous tissue flap and direct release of the fascia covering the soleus.

Advocates for the single incision release support the decreased insult to the tenuous anteromedial skin over the tibia as well as decreased stripping of soft tissues around the tibia [21]. However, this approach is more technically challenging, and criticisms stem from concerns over adequate access to and release of the deep posterior compartment of the leg. The dual incision approach is popular due to the ease of performance and excellent exposure and is most often recommended.

A retrospective comparison of infection and nonunion rates after single versus dual incision fasciotomies did not find any statistically significant differences between the approaches, although higher numbers of infection were seen with plates versus intramedullary devices [21]. This was the first study comparing the two methods of fasciotomy for complications and was admittedly underpowered to detect potentially small differences in infection rates. In a separate investigation, Blair et al. compared groups with tibia fractures and those with tibia fractures requiring fasciotomy for acute compartment syndrome for rates of delayed union, nonunion, and infection. Their results yielded a 5-week increase in time to union, fourfold greater risk of nonunion, and fivefold greater risk of infection in tibia fractures requiring fasciotomies [22]. There are also substantial increases in the length of hospital stay and total cost associated with the need for fasciotomies to treat ACS [23]. The selection of approach may be best determined by the treating surgeon, with the goal of full release and restoration of tissue perfusion.

Wound closure is typically performed with delayed primary closure or skin graft coverage 3–7 days after fasciotomy. With the dual incision fasciotomy, priority is given to closure of the posteromedial incision due to its proximity to the tibia. Skin grafts are not aesthetically appealing and are insensate, so different methods are employed to push toward delayed primary closure. In many cases, skin grafts are inevitable, especially for the lateral wound. A vessel loop technique can be used to minimize skin retraction by interlacing the loops across the incision with staples holding at the side, gradually tensioning them to help avoid skin grafting [24]. Negative pressure wound therapy may be used for temporary coverage of fasciotomy wounds as it creates a seal decreasing contamination from hospital microorganisms, promotes wound granulation, decreases tissue edema, and improves local perfusion [25].

Foot Compartment Syndrome

Compartment syndrome of the foot is an uncommon and controversial topic. Foot compartment syndrome as a whole was underrecognized prior to the 1980s when investigation of fixed foot deformities as a result of severe foot trauma echoed

similarities to Volkmann's ischemic contracture of the hand [26]. Typically, this presentation results from high-energy mechanisms such as crush injuries, motor vehicle or motorcycle collisions, or falls from height. Potential injuries vary but include isolated soft tissue trauma, forefoot fractures, Lisfranc or Chopart fracture dislocations, and calcaneal fractures. The latter, high-energy calcaneal fractures, has historically been reported to develop a foot compartment syndrome in up to 10% of cases, yet a more recent study suggests the actual incidence is lower finding only 1% of patients with an isolated calcaneal fracture underwent fasciotomy for suspected compartment syndrome [27]. Nevertheless, any patient presenting after a higher-energy mechanism, especially a crush, should be evaluated with a heightened suspicion. The development of a secondary foot compartment syndrome from more proximal injury is also described due to a communication between the deep posterior compartment of the leg and the deep central or calcaneal compartment in the foot [28].

There are considerable debate and no real consensus regarding the number of fascial compartments of the foot. Many of these studies were performed in cadavers and cannot reliably reproduce physiologic conditions [29]. At least nine compartments of the foot have been identified: three spanning the entire length of the foot (medial, lateral, and superficial), five forefoot compartments (adductor and four interossei), and a single hindfoot compartment (calcaneal) [26]. The calcaneal compartment contains the quadratus plantae muscle, lateral plantar neurovascular bundle, the posterior tibial nerve and vessels, and in some patients the medial plantar neurovascular bundle.

Diagnosis is again a combination of physical examination of the clinical presentation with an option for intracompartmental pressure monitoring. Symptoms of pain out of proportion, pain despite immobilization that is unrelieved by progressive doses of analgesics, and paresthesias are frequently seen. Signs include tense compartments and pain with passive range of motion. Passive dorsiflexion of toes decreases the volume of the interosseous compartments, thereby intensifying pain [26]. The most sensitive sensory indicators are decreased light touch and two-point discriminatory sensations, especially those with relative decreases over serial examinations [26]. Strength and pulses are poor indicators.

There is no consensus of a firm recommendation on the use of compartment measuring to the foot. This likely stems from disagreement regarding the true number of foot fascial compartments and debate about the potential of these compartments to develop pressures sufficient to cause a compartment syndrome. Continuous or even repeat monitoring of the compartments is not practical. However, it can provide further objective data helping in diagnosis. There is no firm consensus on the number of, or which, compartments that should be measured, but as the calcaneal compartment is frequently implicated as having the highest pressure readings in studies, increased attention should be paid to this compartment [28, 29]. The technique for accessing this compartment is insertion of the device 6 cm distal to the most prominent portion of the medial malleolus with insertion depth of approximately 24 mm [28]. An absolute compartment pressure >30 mmHg is generally considered an indication for emergent decompression.

The most frequently recommended approach for foot fasciotomies combines a dorsal two-incision approach with a medial plantar approach [28]. The dorsal medial incision is made just medial to the second metatarsal and allows access of the first two interosseous compartments, as well as the adductor compartment located deep to the first interosseous compartment. The dorsal lateral incision is made just lateral to the fourth metatarsal. It can be used to release the third and fourth interosseous, the lateral, and central compartments. Longitudinal dissection of the dorsal fascia is performed on both medial and lateral sides of the metatarsals with the central compartments entered after incision of the interosseous fascia. Advantages of these incisions provide exposure for fixation of midfoot trauma [29]. A separate medial plantar approach is added due to concerns of access to the calcaneal compartment from the dorsal approach [26]. This medial incision follows the length of the inferior border of the first metatarsal, a 6 cm incision beginning approximately 4 cm from the posterior border of the heel and 3 cm superior to plantar surface [26]. The abductor hallucis is retracted cranially, and the intermuscular septum is identified. Incising this septum releases the deep (calcaneal) compartment, and the quadratus plantae should bulge from the incision. Care should be taken with this step since the lateral plantar neurovascular structures are immediately deep to the septum [26]. Release of the distal tarsal tunnel through proximal extension of this medial incision may be necessary for adequate release of the calcaneal compartment [26]. Retracting this medial compartment superiorly exposes the superficial compartment, which is released longitudinally, decompressing the flexor digitorum brevis (FDB). The FDB is retracted inferiorly, and the medial fascia of the lateral compartment is visualized. Decompression of the lateral compartment is complete when the abductor digiti quinti and flexor digiti minimi are exposed [29].

Treatment with fasciotomies is not without complication, as the incisions are risks for wound infections and frequently require skin grafting. Secondary closure of wounds is typically delayed 5–7 days after fasciotomy, with skin grafting alternatively covering wounds not amenable to closure. A systematic review found that 65% of cases required skin grafts after fasciotomy [30]. Forefoot and midfoot fractures can be stabilized definitively acutely, provided primary wound closure is possible over the implants. Calcaneal fractures, however, are recommended to undergo delayed fixation 10–14 days after fasciotomy to allow swelling to decrease. Shoewear selection after soft tissue reconstruction poses another challenge that may limit long-term function. Patients commonly have residual pain and stiffness with only 10% able to return to their pre-injury state after fasciotomy [30].

There are many experienced surgeons who argue for managing the compartment syndrome conservatively, with delayed treatment of sequelae including nerve decompression, soft tissue releases, tendon transfers, osteotomies, or fusions. Although a systematic review reported that complications rates were lower for those treated with fasciotomies than those untreated, overall data comparing the two groups is lacking [30]. The treating surgeon must make the decision based upon the evaluation of the patient and their best judgment. Loss of distal perfusion should be an almost absolute indication for decompression.

An alternative approach to fasciotomies of the foot involves a "pie crusting" technique, where multiple stab incisions are made over the foot followed by blunt dissection with a hemostat [28]. The goal is to reduce pressure on the soft tissues and decrease the need for secondary soft tissue coverage; however, critics would cite the risk of inadequate release of muscular compartments.

Complications from a missed compartment syndrome include sensory alterations and the development of ischemic foot deformities. Claw toes are the most common complications, resulting from compression of the medial and lateral plantar bundles in the calcaneal compartment [28]. This ischemic insult to the quadratus plantae and interosseous muscles leads to their overpowering of these intrinsic muscles by the extrinsic muscles to the foot. Cavus foot deformities can develop as well. Neurologic complications include chronic pain, neuropathic pain, numbness, allodynia, and hyperalgesia. Ulcerations can develop secondary to the deformities, altered gait mechanics, and neuropathic changes, creating lifelong problems for some patients. Amputation is a final, but effective, treatment option in the most severe cases [28].

Summary

Compartment syndrome is a pathologic condition where intrafascial pressures increase and ultimately cause irreversible cell death if fasciotomies are not urgently performed. Diagnosis remains a challenge for all physicians and requires vigilance and frequent physical examinations. Intracompartmental measurements can be used to aid in diagnosis of obtunded patients when complete clinical examination is not possible, but measurements lack specificity and are of variable accuracy with technical errors common. Once diagnosed, urgent surgical management is simple and only requires a surgeon with anatomic knowledge and a scalpel.

References

1. Woolley SL, Smith DR. Acute compartment syndrome secondary to diabetic muscle infarction: case report and literature review. Eur J Emerg Med. 2006;13(2):113–6.
2. Garner MR, Taylor SA, Gausden E, Lyden JP. Compartment syndrome: diagnosis, management, and unique concerns in the twenty-first century. HSS J. 2014;10(2):143–52.
3. Olson SA, Glasgow RR. Acute compartment syndrome in lower extremity musculoskeletal trauma. J Am Acad Orthop Surg. 2005;13(7):436–44.
4. Whitesides TE, Heckman MM. Acute compartment syndrome: update on diagnosis and treatment. J Am Acad Orthop Surg. 1996;4(4):209–18.
5. Kortbeek JB, Al Turki SA, Ali J, Antoine JA, Bouillon B, Brasel K, et al. Advanced trauma life support, 8th edition, the evidence for change. J Trauma. 2008;64(6):1638–50.
6. Malinoski DJ, Slater MS, Mullins RJ. Crush injury and rhabdomyolysis. Crit Care Clin. 2004;20(1):171–92.

7. Kasaoka S, Todani M, Kaneko T, Kawamura Y, Oda Y, Tsuruta R, et al. Peak value of blood myoglobin predicts acute renal failure induced by rhabdomyolysis. J Crit Care. 2010;25(4): 601–4.
8. Holcomb JB, Tilley BC, Baraniuk S, Fox EE, Wade CE, Podbielski JM, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA. 2015;313(5):471–82.
9. 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(11): 2415–22.
10. Whitney A, O'Toole RV, Hui E, Sciadini MF, Pollak AN, Manson TT, et al. Do one-time intracompartmental pressure measurements have a high false-positive rate in diagnosing compartment syndrome? J Trauma Acute Care Surg. 2014;76(2):479–83.
11. McQueen MM, Court-Brown CM. Compartment monitoring in tibial fractures. The pressure threshold for decompression. J Bone Joint Surg Br. 1996;78(1):99–104.
12. Large TM, Agel J, Holtzman DJ, Benirschke SK, Krieg JC. Interobserver variability in the measurement of lower leg compartment pressures. J Orthop Trauma. 2015;29(7): 316–21.
13. Henson JT, Roberts CS, Giannoudis PV. Gluteal compartment syndrome. Acta Orthop Belg. 2009;75(2):147–52.
14. David V, Thambiah J, Kagda FH, Kumar VP. Bilateral gluteal compartment syndrome. A case report. J Bone Joint Surg Am. 2005;87(11):2541–5.
15. Ojike NI, Roberts CS, Giannoudis PV. Compartment syndrome of the thigh: a systematic review. Injury. 2010;41(2):133–6.
16. 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(3): 392–400.
17. 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(4): 729–37.
18. McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome. Who is at risk? J Bone Joint Surg Br. 2000;82(2):200–3.
19. Wind TC, Saunders SM, Barfield WR, Mooney JF 3rd, Hartsock LA. Compartment syndrome after low-energy tibia fractures sustained during athletic competition. J Orthop Trauma. 2012;26(1):33–6.
20. Tornetta P 3rd, Puskas BL, Wang K. Compartment syndrome of the leg associated with fracture: an algorithm to avoid releasing the posterior compartments. J Orthop Trauma. 2016;30(7):381–6.
21. Bible JE, McClure DJ, Mir HR. Analysis of single-incision versus dual-incision fasciotomy for tibial fractures with acute compartment syndrome. J Orthop Trauma. 2013;27(11): 607–11.
22. Blair JA, Stoops TK, Doarn MC, Kemper D, Erdogan M, Griffing R, et al. Infection and nonunion after fasciotomy for compartment syndrome associated with tibia fractures: a matched cohort comparison. J Orthop Trauma. 2016;30(7):392–6.
23. Crespo AM, Manoli A 3rd, Konda SR, Egol KA. Development of compartment syndrome negatively impacts length of stay and cost after tibia fracture. J Orthop Trauma. 2015;29(7): 312–5.
24. Asgari MM, Spinelli HM. The vessel loop shoelace technique for closure of fasciotomy wounds. Ann Plast Surg. 2000;44(2):225–9.
25. Kanakaris NK, Thanasas C, Keramaris N, Kontakis G, Granick MS, Giannoudis PV. The efficacy of negative pressure wound therapy in the management of lower extremity trauma: review of clinical evidence. Injury. 2007;38(Suppl 5):S9–18.
26. Fulkerson E, Razi A, Tejwani N. Review: acute compartment syndrome of the foot. Foot Ankle Int. 2003;24(2):180–7.

27. Thakur NA, McDonnell M, Got CJ, Arcand N, Spratt KF, DiGiovanni CW. Injury patterns causing isolated foot compartment syndrome. J Bone Joint Surg Am. 2012;94(11):1030–5.
28. Dodd A, Le I. Foot compartment syndrome: diagnosis and management. J Am Acad Orthop Surg. 2013;21(11):657–64.
29. Frink M, Hildebrand F, Krettek C, Brand J, Hankemeier S. Compartment syndrome of the lower leg and foot. Clin Orthop Relat Res. 2010;468(4):940–50.
30. Ojike NI, Roberts CS, Giannoudis PV. Foot compartment syndrome: a systematic review of the literature. Acta Orthop Belg. 2009;75(5):573–80.