



# Compartment Syndrome and Orthopedic Surgery: Diagnosis and Management

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## Objectives

- To define compartment syndrome and understand its etiology and incidence
- To explain how to identify and diagnose compartment syndrome
- To elucidate the spectrum of compartment syndromes
- To discuss treatment options and timing to surgical intervention
- To describe the most common post-treatment complications and patient outcomes
- To describe differences between adult and pediatric patients in terms of diagnosis, timing of fasciotomy, and outcomes in acute compartment syndrome

## Key Points

- Compartment syndrome is defined as an elevation of intracompartmental pressure to a level that impairs arterial flow to muscles, nerves, and other local tissues.

- Compartment syndrome of the upper and lower extremities can have multiple etiologies, including traumatic, exertional, and iatrogenic in the perioperative setting.
- Early identification and diagnosis enabling prompt intervention is essential to providing patients the best possible outcomes.
- In cases of acute compartment syndrome, emergent fasciotomy is generally indicated. Delayed fasciotomies more than 12–24 h after onset of symptoms are not recommended as they increase morbidity and mortality; however, it is often difficult to establish a time zero for onset or irreversibility.
- Even with timely treatment, multiple surgeries are often necessary to ensure adequate wound debridement, appropriate soft tissue coverage and satisfactory wound closure. Long-term sequelae range from cosmetic concerns secondary to wound complications, the use of skin grafts, limb deformity, amputation, or systemic complications associated with rhabdomyolysis.
- Compartment syndrome may be more difficult to diagnosis in the pediatric patient, but fortunately, outcomes are generally better than in the adult population, even following delayed fasciotomy.

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## Introduction

Compartment syndrome is defined as an increase in intracompartmental pressure sufficient to impair the micro and/or macrovascular circulation to a level that can cause ischemia and necrosis of local tissue, especially muscle [1]. A group of muscles bound by fascia are considered a compartment in the extremities, although paraspinous compartment syndrome has been described [2–6]. Quantitatively, relative ischemia of

muscle begins when tissue pressures rise to within 30 mmHg of the patient's diastolic pressure. Experimental studies have shown significant muscle necrosis at sustained absolute pressures of 30 mmHg [7–12]. Diagnostic values vary based on institutional preference and surgeon experience, but our threshold for the diagnosis of acute compartment syndrome is a  $\Delta P$  (diastolic blood pressure – intracompartmental pressure) of less than 30 mmHg in one or more compartment.

Compartment syndrome exists on a spectrum and ranges from acute to chronic. Despite a variety of causes, including burns, vascular injuries, and those that occur after surgical procedures, the vast majority of acute compartment syndromes seen by orthopedic surgeons are diagnosed in the setting of blunt trauma. Based on a study of 164 patients from the UK, tibial shaft fractures account for 36% of compartment syndromes associated with acute injuries [13]. Fractures in the upper extremity, hand, and foot account for the majority of other clinical scenarios where compartment syndrome is an important concern (Table 29.1). The same study reports the average annual incidence in men to be 7.3 per 100,000 and 0.7 per 100,000 in women, a tenfold increased risk of acute traumatic compartment syndrome for males. Nonetheless, for individuals presenting with acute tibial shaft fractures, a recent study out of Canada has found that the patient's sex does not predict the likelihood of acute compartment syndrome and open and closed tibial shaft fractures confer a statistically equivalent risk of acute compartment syndrome. Meanwhile, certain patient factors are in fact implicated in acute compartment syndrome, as the same study found that young adults with tibial shaft fractures are at higher risk of developing acute compartment syndrome when compared to older adults with similar injuries [14]. However, for those who care for orthopedic patients on a regular basis, it is imperative to keep in mind that treatment modalities such as surgical fixation of fractures and casting

can also result in compartment syndrome. For this reason, vigilance in the post-injury as well as postoperative period is essential.

It is well documented that the primary cause of poor outcomes and failed treatment in compartment syndrome is delayed diagnosis [15–18]. A missed compartment syndrome may lead to additional surgical procedures, medical expenses, and patient morbidity, which often results in legal ramifications for those involved. Bhattacharyya and Varhas retrospectively reviewed 19 closed malpractice claims and found the following factors to be associated with “poor legal outcome”: documentation of abnormal neurologic examination but no action, poor physician communication (i.e., disregarding telephone calls), and delay in fasciotomy after initial presentation. Furthermore, the number of cardinal signs of compartment syndrome (pain out of proportion, pallor, paresthesias, paralysis, and pulselessness) was linearly associated with the dollar amount of payment ( $p < 0.001$ ,  $R = 0.74$ ) and an increased number was associated with an increased chance of indemnity payment ( $p < 0.02$ ) [19]. Within this cohort, 11 patients required an average of 3.5 additional procedures. Sixteen cases were settled without trial over an average of 5.5 years. The decision ratio was 9:7 (patient/surgeon) with an average indemnity payment of \$426,000. Three cases went to trial with all three verdicts favoring the treating surgeon. The average defense cost of these cases was \$29,500. Overall, the most common sequelae alleged by the patients were need for additional procedures, loss of motion, foot drop, chronic pain, and difficulty walking.

To limit the patient morbidity and legal sequelae associated with compartment syndrome, early and accurate diagnosis is essential. Despite modern diagnostic tools, history and clinical examination remain the primary means of diagnosing compartment syndrome. All providers caring for the orthopedic patient, including nursing assistants, registered nurses, nurse practitioners, physician assistants, residents, and attending surgeons, should be aware of the diagnostic criteria and have a thorough understanding of injuries and surgical procedures that put patients at risk for compartment syndrome.

Primary and follow-up assessment of all traumatic injuries should include specific attention to the cardinal signs or the “Ps” of compartment syndrome (Table 29.2). Although pain out of proportion to examination (or increasing analgesic

**Table 29.1** Blunt trauma conditions in which compartment syndrome is diagnosed

Underlying condition	% of cases
Tibial shaft fracture	36
Soft tissue injury	23.2
Distal radius fracture	9.8
Crush syndrome	7.9
Diaphyseal forearm fracture	7.9
Femoral diaphyseal fracture	3.0
Tibial plateau fracture	3.0
Hand fracture(s)	2.5
Tibial pilon fractures	2.5
Foot fracture(s)	1.8
Ankle fracture	0.6
Elbow fracture dislocation	0.6
Pelvic fracture	0.6
Humeral diaphyseal fracture	0.6

Data from: McQueen et al. [13]

**Table 29.2** Cardinal signs or the five “Ps” of compartment syndrome

“P”	Description
Pain	Pain associated with injury or necrosis; typically seen early
Pallor	Loss of normal skin tone and/or capillary refill
Poikothermia	Loss of body heat in area of injury
Paresthesias	Numbness or tingling sensation; typically seen late
Pulselessness	Loss of pulses distal to site of injury

requirements in younger patients) is considered to be the first indication of an impending compartment syndrome, patients may present with any combination of signs or symptoms.

Work by Bae and coauthors has shown that the traditional five “Ps” of compartment syndrome are unreliable in the pediatric population, with the exception of pain as inferred from an increasing analgesia requirement. All patients in their study with access to patient-controlled analgesia (PCA) demonstrated increasing analgesia requirements. Agitation/restlessness and anxiety were also noted in their study population. Their findings have led the adoption of a separate mnemonic for pediatric compartment syndrome known as the three “As” for agitation, anxiety, and analgesia (short-hand for increasing analgesia requirement) [20].

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## Perioperative Considerations

### Acute Assessment

As stated previously, despite advances in quantitative diagnostic devices, history and physical examination are essential to diagnosing acute compartment syndrome.

In our institution, serial physical exams are performed every 2–4 h on all patients deemed to be at high risk. Such patients include tibial shaft and plateau fractures, crush injuries, and any patient with a concerning physical exam at presentation (i.e., significant swelling, pain out of proportion to exam, etc.). It is also very important to recognize that specific operations such as intramedullary nailing and osteotomies may lead to postoperative compartment syndrome, and thus these procedures mandate serial exams for a minimum of 24 h postoperatively. Lastly, to avoid iatrogenic compartment syndrome, all postoperative immobilization is performed with splints or bivalved casts to allow for tissue expansion and easy, rapid removal if necessary.

The use of catheter insertion to measure compartment pressure has become more common since the initial use of needle manometry in 1975 [7], but those using such devices should be aware that tissue pressures will vary based on distance from the site of injury with peak pressures being encountered within a few centimeters of fractures [21]. Often, such quantitative measures are used in the operating room to confirm a clinical diagnosis rather than to make a diagnosis.

Recent studies have focused on new diagnostic modalities for the detection of acute compartment syndrome. Cathcart and colleagues showed that near-infrared spectroscopy detects changes in oxygenation in muscle tissue in response to compartment syndrome induced in a porcine model, including a return of oxygenation to the muscle following fasciotomy [22]. Tissue ultrafiltration catheters have been explored for both their diagnostic potential to measure

compartment pressures and biomarkers associated with compartment syndrome such as lactate dehydrogenase and creatine kinase as well as their therapeutic capacity to remove fluid and thus decrease intracompartmental pressures [23]. Lastly, implantable microchip pressure sensors and transmitters have been proposed as minimally invasive, portable continuous compartment pressure monitors [24]. Nevertheless, these new technologies still need to be validated in the clinical setting, and clinical examination remains the mainstay of diagnosis.

### Anesthetic Considerations

Advances in regional anesthetic techniques over the past several decades have allowed for excellent perioperative pain control while limiting excessive narcotic use. A combination of spinal anesthesia for lower extremity procedures and short- and long-acting peripheral nerve blocks in both the upper and lower extremities is increasingly common. However, patients with suspected impending compartment syndrome or those undergoing high-risk surgical procedures should not be administered long-acting peripheral blocks under any circumstances. Such anesthetic techniques can mask pain associated with increased compartment pressures and severely limit a practitioner’s assessment [25]. Any spinal or peripheral anesthetic used should be either short acting or easily titrated down to zero so that a formal assessment of pain and neurologic status can be obtained rapidly and accurately.

### Sign-Out/Documentation

Orthopedic practice has seen a rapid increase in patient volume. Simultaneously, new regulations, such as residency work hour restrictions, have led to an increase in the number of care providers involved with a patient’s care. The number of “sign-outs” is only increasing, with patients often changing hands several times each day. The potential for error, due to a failure of communication is great [26].

Given that compartment syndrome is one of the few, true orthopedic emergencies, any patient at risk for developing this condition should receive special attention during sign-out sessions. The outgoing team must personally relay the information to the person who will be assuming care of the patient. E-mail, a common form of communication in the healthcare field today and one that is frequently used as a sign-out tool at many institutions, is neither appropriate nor adequate when transferring care of a patient, especially one at risk for developing a compartment syndrome. Further, in such situations where a patient will be receiving compartment checks from more than one practitioner over a given

time period, every attempt should be made for both individuals to see the patient together at the time care is transferred to establish an accurate baseline examination by the practitioner who is assuming care.

Given the medical-legal implications of delayed diagnosis and/or missed diagnosis of a compartment syndrome, timed documentation has become a point of emphasis for patients being monitored for a possible compartment syndrome. Each “compartment check” should be carefully documented and attention paid to both the patient’s subjective complaints and objective findings. The patient should be asked specifically about their pain, subjective tightness, as well as any emerging neurologic symptoms such as decreased sensation and dysesthesias. Objective findings and subjective complaints should be compared with prior exams.

The physical exam of a patient with possible compartment syndrome is fourfold. First, careful palpation of each compartment should be conducted, although findings are entirely subjective and have been shown unreliable. Shuler and colleagues showed that in a cadaveric model, palpation of compartments had a sensitivity of only 54% for detection of elevated compartment pressures [27]. Next, the muscle groups of each compartment should be stretched passively. If compartment pressures are significantly elevated, muscle stretching within that compartment should elicit significant pain. Passive stretch is perhaps the earliest objective finding and arguably the most important component of examination. Third, a careful neurologic examination including both motor and sensation should be conducted. It is essential to include all potential nerve distributions, especially in the splinted patient where particular distributions may be more difficult to access. Lastly, vascular status should be assessed with palpation of pulses, skin temperature, and capillary refill. Patients who are sedated, intubated, or otherwise unresponsive (including the pediatric patient) and cannot express their symptoms may require manometric monitoring and a lower threshold for intervention.

As with any physical examination, that of a patient with compartment syndrome can vary widely with each subsequent exam. Cascio and colleagues retrospectively reviewed 30 consecutive patients undergoing fasciotomy for acute compartment syndrome over a 10-year period and found 90% to be lacking in documentation of a complete physical exam (Table 29.3). Of the 30 patients, ten had permanent sequelae [28]. As stated previously, documentation of an abnormal neurologic examination and failure to act upon those findings is associated with indemnity payments during malpractice cases [19]. For these reasons, accurate documentation of a physical examination at the time it is performed, along with any actions taken at that time is crucial.

The rise of electronic medical records highlights certain medical-legal pitfalls in documenting “compartment checks.” Some practitioners choose to document all com-

**Table 29.3** Of 30 consecutive patients undergoing fasciotomy for acute compartment syndrome over a 10-year period, 90% are found to be lacking in documentation of a complete physical exam

Core H&P findings	Patients with inadequate documentation (n = 30)
Tenselessness	3 (10%)
Pain	5 (17%)
Compartment pressures	6 (20%)
Pulses	7 (23%)
Motor examination	8 (27%)
Sensory examination	9 (30%)
Pain on passive stretch	10 (33%)
Paresthesias	11 (37%)
Diastolic blood pressure	16 (53%)
Pallor	28 (93%)
Overall (excluding pallor)	21 (70%)

Data from: Cascio [28]

partment examinations in one note at the end of their shift to increase efficiency. The authors strongly discourage against this practice and recommend immediate documentation of each compartment check individually. Similarly, back-dating of notes undermines the practitioner’s legal credibility. If a practitioner wishes to clarify or correct an existing note in the chart, the authors recommend documenting this information in a timed addendum rather than attempting to edit the existing text of a note, as alterations to the original note are tracked in the electronic medical record and may appear suspect.

### Informed Consent/Patient Expectations

Perhaps one of the most overlooked issues surrounding the diagnosis and is treatment of compartment syndrome centers around the topic of informed consent. For any patient suspected of having an acute compartment syndrome, an impending compartment syndrome, or a surgical procedure associated with a high risk of compartment syndrome, it is the responsibility of the treating surgeon and team to discuss with the patient the risks associated with the diagnosis, the treatment options, and the possible long-term sequelae associated with both the diagnosis itself and the treatment (i.e., fasciotomy). Proper expectations must be set. Consent should be obtained for possible fasciotomy in such patients. If proper consent is obtained and the patient has a thorough understanding of possible outcomes, sequelae can be looked at as expectations rather than a complication.

The importance of early fasciotomy as treatment for acute, traumatic compartment syndrome is well-documented dating back as early as 1914 [29]. Any compartment in question should undergo early fasciotomy, and in many cases prophylactic fasciotomies are performed on neighboring compart-



ments – for example being the tibial shaft fracture with elevated intracompartmental pressures in the anterior compartment that is treated with a four compartment fasciotomy. All nonviable tissue is debrided at the time of fasciotomy. Surgeons often use the mnemonic of the four Cs – color, contractility, consistency, and capacity to bleed – as indices of muscle viability. However, the four Cs may not be reliable markers of muscle viability based on a recent study correlating intra-operative appearance and histology [30].

After fasciotomy and debridement, treating surgeons are frequently left with two issues, the first being fracture fixation, as the majority of compartment syndromes occur in the setting of osseous injury [31], and the second being wound closure. In order to decompress the compartments and allow for soft tissue swelling to subside, fasciotomy wounds are left open, frequently with negative pressure dressings (VAC). Delayed primary wound closure is typically attempted after 48 h, assuming there is viable muscle coverage of the underlying osseous structures and a tension-free closure can be achieved. Split thickness skin grafting or gradual closure techniques are indicated if the wound is under tension. For more severe cases, patients may require rotational or free muscle flap coverage and experience donor site morbidity, or they may require fitting of a prosthetic if amputation was required. The patient should be aware prior to fasciotomy that repeat procedures and possible plastic surgery intervention may be required, and, in some cases, amputation may be necessary.

Systemic complications of acute compartment syndrome should also be discussed with the patient and include sepsis stemming from infection of necrotic tissue and rhabdomyolysis with resulting renal failure. It is important to monitor serum CPK levels and renal function in patients suspected of compartment syndrome. Patients should also be counseled regarding cosmesis following wound closure, and muscle weakness secondary to necrosis and debridement. Recent murine studies demonstrate that much of the damage caused by acute compartment syndrome is mediated by inflammatory processes, and these pre-clinical studies also suggest that anti-inflammatory medications such as indomethacin may be protective if administered prior to or even after the onset of acute compartment syndrome [24].

It has been our experience that compartment syndromes of the foot is unique in that fasciotomy often results in poor functional outcomes. For these reason, we believe that select patients may be observed clinically, provided both the surgeon and patient are prepared to address the sequelae, which are often treatable with minor surgery and more tolerable than those associated with fasciotomy. Toe-clawing and contracture, fibrosis, stiffness and aching, atrophy of intrinsic muscles, and sensory disturbances [32] should all be discussed with the patient at length and the conversation documented before the decision is made to observe a diagnosed compartment syndrome.

## The Spectrum of Compartment Syndrome

As we have discussed, the criteria for diagnosis of compartment syndrome are predominately clinical. In the cases of acute compartment syndrome in a patient with a high-risk injury and rapid diagnosis, intervention is clear and well defined. The actual onset of compartment syndrome is often unknown, however. As is the case with many conditions in medicine and orthopedics, compartment syndrome frequently exists in a spectrum ranging from the classic acute presentation to delayed timing to diagnosis to the late or “missed” case.

The delayed or late compartment syndrome is a particularly important consideration in the orthopedic patient, especially in patients unable to convey their pain or symptoms (pediatric or ICU patient) or the patient transferred from an outside hospital facility hours or days following injury. Rorabeck and Clarke demonstrated that muscle function irreversibly deteriorated when fasciotomies were performed greater than twelve hours after the onset of acute compartment syndrome in a dog model, suggesting some degree of myonecrosis by this point [33]. While some studies indicate no increased risk with late fasciotomies [18], others suggested that fasciotomy-related morbidity, particularly with regard to infection, increases with delay in diagnosis as necrotic muscle exposed to the outside environment at the time of surgery is highly susceptible to bacterial pathogens [34, 35]. In fact some authors have reported increased morbidity and mortality in patients treated with fasciotomies more than 24 h after diagnosis [36]. Sheridan and Matsen noted that early fasciotomy patients had a complication rate of only 4.5%, while those treated with late fasciotomies were exposed to a 54% morbidity rate – half of which ultimately proceeded to amputation [35]. Finkelstein and colleagues reported a case series of five patients with closed lower extremity injuries who underwent late fasciotomies, more than 35 h after injury (average 56 h) [37]: one patient died from sepsis while the other four ultimately required amputations (3 secondary to infection and 1 secondary to lack of function). Prior to the era of renal dialysis, death from crush/compartment syndrome occurred most commonly from renal failure. The authors contend, however, that with modern means of dialysis, death from these injuries is predominately due to infection. They conclude that late fasciotomies convert a closed fracture into an open injury and put the patient at risk of overwhelming infection and related morbidity and mortality. Further to this end, Ritenour and colleagues more recently reported a two-fold increase in amputation and threefold increase in mortality in trauma patients treated with delayed fasciotomies [38].

Recent data, however, suggest that delayed treatment of compartment syndrome in the pediatric population may allow for acceptable results with a low risk of infection. Flynn and colleagues retrospectively reviewed 43 cases of acute traumatic compartment syndrome of the lower leg treated at two

institutions with fasciotomy [39]. Of the 43 cases, nine had fasciotomies beyond 24 h post-injury (up to 118 h). 7/9 had excellent outcomes, 2/9 had fair outcomes with fasciotomies at 82.5 and 86 h (weakness with dorsiflexion) and there were no cases of infection. Pediatric upper extremity acute compartment syndrome similarly had favorable outcomes following fasciotomy in a study conducted by Wadji and coauthors, with an average time from injury to fasciotomy of 32.8 h and no effect of time to fasciotomy on final outcomes [40].

Compartment syndrome following hip and knee arthroplasty is relatively rare. Lasanios and colleagues reviewed the literature for cases in which compartment syndrome complicated total joint arthroplasty and identified 41 such cases, with nearly a 50/50 split between hip and knee arthroplasty [41]. The most common site of compartment syndrome following total hip arthroplasty was gluteal, accounting for nearly 73%. Not surprisingly, the most common site of compartment syndrome following total knee arthroplasty was the calf (61%), but gluteal compartment syndrome occurred with relative frequency (17%). The mean time to diagnosis was 26 h and the mean time to surgical intervention was 53 h. Gluteal compartment syndrome was almost exclusively attributed to body habitus and prolonged positioning either intraoperatively or postoperatively.

Gluteal compartment syndrome most commonly is atraumatic in etiology. This is of particular importance when considering the obese orthopedic patient who undergoes a prolonged procedure. Henson and coauthors performed a systematic review of seven publications including 28 patients diagnosed with gluteal compartment syndrome [42]. They noted that the most common cause of gluteal compartment syndrome was prolonged immobilization in men with an average age of 45 years. The patient's body weight was connected with the condition in 50% of the cases studied. Of the cases, 21% occurred in the contralateral (down side) of postoperative total joint arthroplasty patients. Trauma was identified as the causative source in less than one quarter of patients. Less than half of the patients were diagnosed with quantitative pressure assessments with the remainder diagnosed based on history and physical examination. Only 71% of diagnosed gluteal compartment syndromes were treated with surgical decompression. Of those treated without surgical intervention, the majority of cases included delayed presentation or diagnosis. Patient outcomes are variable based upon the compartments involved, extent of damage, and chronicity of diagnosis.

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### Special Consideration in the Perioperative Patient

While the most common etiology of compartment syndrome is trauma, it is crucial to recognize other potential causes in the perioperative orthopedic patient. Iatrogenic compartment

syndromes may be prevented with attention to patient positioning, selection, appropriate tourniquet use, and careful application of immobilization devices.

It was initially thought that intramedullary nailing increased compartment pressures and thus increased the risk of postoperative compartment syndrome. Two studies, however, refute this notion. Tornetta and French prospectively evaluated 56 tibial shaft fractures without compartment syndrome preoperatively, each case being treated within 72 h of incident injury [43]. They performed continuous pressure monitoring of the anterior compartment and found transient increase in intracompartmental pressures highest during manual reduction (34 mmHg) and undreamed nail passage (26 mmHg), but noted immediate return to baseline pressures following nail passage. Nassif and colleagues reported on 49 tibial shaft fractures treated with intramedullary nailing within 72 h of injury [44]. They measured anterior and deep posterior compartment pressures and compared reamed and unreamed techniques. Their pressure measurements were similar to those found by Tornetta and French, noting rapid return to baseline. Further, they found no significant difference in reamed versus unreamed nailing on anterior compartment pressures, but statistically significant lower pressure in the deep posterior compartment for reamed nails. In each case, there were no cases of postoperative compartment syndrome.

The use of modern pneumatic tourniquets during orthopedic surgery is commonplace and allows improved visualization in a relatively bloodless operative field and reduced surgical blood loss. Temporary stoppage of blood flow to a limb results in tissue hypoxia and acidosis [45]. Inappropriate use, both pressure and duration, however, can lead to postsurgical complications including compartment syndrome. More than 2 h of sustained extremity ischemia may lead to post-tourniquet syndrome including pallor, swelling, and stiffness without neurologic symptoms due to myocyte injury [46, 47]. Post-tourniquet syndrome typically resolves within 1 week [46]. In extreme cases, however, extended tourniquet duration or excessive pressure can lead to frank compartment syndrome. Current guidelines for tourniquet use include duration less than 2 h [48]. In prolonged surgical cases, requiring greater than the recommended 2 h tourniquet time, Townsend and coauthors recommends a 30-min interval off tourniquet prior to reinflation [49]. The magnitude of tourniquet inflation should be 50–75 mmHg above preoperative systolic pressure for upper extremity surgery and 100–150 mmHg for lower extremity surgery [48]. Even with proper tourniquet use, however, compartment syndrome can occur. Hypervigilance and an open differential diagnosis are critical to recognition.

Compartment syndrome has also been reported in the well leg of patients undergoing orthopedic procedures on the trac-

tion table [50–53]. Development of this pathology is thought to be associated with direct compression of the lateral calf on the supportive post and the relative hypoperfusion of the limb in the elevated position if lithotomy position is used. The well leg may be dropped down into a scissor position to theoretically decrease the risk of hypoperfusion relative to positioning the well leg in lithotomy. Hypoperfusion of the limb may also be exacerbated in the patient undergoing regional anesthesia.

Application of pre- and postoperative splints and bandages must be undertaken with care and caution as over constriction of a limb may lead to the development of iatrogenic compartment syndrome [54]. Hinderland and coauthors reported a case of iatrogenic isolated lateral lower leg compartment syndrome in a 44-year-old man caused by ill-fitting compression stockings placed for DVT prophylaxis [55]. Others have reported cases of IV infiltration leading to compartment syndromes of the forearm and foot [56].

Compartment syndrome of the calf is most common, accounting for 36% of cases. This pathology can occur in any fascial bound muscle group including the foot, hand, and gluteal region. Regardless of the location, diagnosis and management occurs in a similar fashion. With regard to the less common regions, however, the most important diagnostic factor is a high clinical suspicion and inclusion of compartment syndrome on the differential diagnosis of pain. Roberts and coauthors looked at several of the less common compartment syndromes and noted that compartment pressures were performed in 64% of patients with compartment syndrome of the foot and less than 50% in the other less common areas such as the forearm, gluteal compartment, and the thigh [57].

Ojike and coauthors reviewed compartment syndrome of the foot in a systematic review and note the most common etiologies to be crush injuries, falls from height, and motor vehicle accidents in 28%, 26%, and 34% of cases, respectively [58]. Calcaneal fractures and Lisfranc fracture dislocations accounted for nearly half of the cases studied. While we have not found reports of foot compartment syndrome following elective corrective osteotomies, these are certainly to be considered a potential source of foot compartment syndrome. There exists significant debate as to the necessity of surgical decompression for treatment of foot compartment syndrome. Advocates argue that fasciotomies decrease the incidence of sequelae such as claw toes, impaired mobility, stiffness, and sensory deficits. Others argue, however, that the morbidity associated with surgical intervention may outweigh the morbidity of observation and later corrective procedures.

Sequelae of treated and untreated compartments syndrome have significant functional and aesthetic ramifications. Nerve deficits and stiffness are the most common

sequelae following compartment syndrome regardless of the location. Ultimately, a timely and accurate diagnosis provides patients with the most optimal circumstances for recovery.

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## Summary

Compartment syndrome has devastating implications for surgeons and patients alike. The sequelae include patient morbidities, both functional and cosmetic. Failure to identify and document findings can have profound ramifications. The most effective treatment is early diagnosis.

Diagnosis of compartment syndrome is overwhelmingly clinical. Members of the medical or surgical care team must pay particular attention to the earliest findings – pain out of proportion, increasing analgesic requirements, and pain with passive stretch of muscles in a suspect fascial compartment. Other findings such as palpation for fullness are subjective and have been linked with poor interobserver reliability. Further the other traditional findings (pallor, paresthesias, paralysis, and pulselessness) are late findings. The most reliable findings in the pediatric population are increasing analgesia requirement (such as in cases with PCA), agitation, and anxiety. Newer technologies are being assessed for their ability to diagnose and potentially even treat compartment syndrome, but these technologies are still in early phases.

Compartment syndrome exists on a spectrum ranging from acute to delayed to late recognition. An acute compartment syndrome should undergo immediate fasciotomies. Some more recent literature points to observation in cases of late compartment syndrome, as exposing necrotic muscle dramatically increases the risk of infection. Unfortunately, there is often an unclear distinction between these phases of compartment syndrome. As such, the authors urge fasciotomy in any case where there is a question as to the timing to onset of the condition. Furthermore, while delayed fasciotomies in the pediatric population tend to produce more acceptable outcomes than the severe morbidity seen in the adult population, immediate fasciotomy is best in all cases of acute compartment syndrome.

While compartment syndrome is traditionally thought of as occurring in the setting of trauma (fracture or crush), there are a number of other etiologies including iatrogenic ones. As a medical community, we have the opportunity to limit these risks by paying particular attention to details such as positioning, placement of stockings, and splints. Further, we have an obligation to identify those at particular risk, perform appropriate examination, communicate with colleagues, and take immediate action as a patient's condition changes.

### Summary Bullet Points

- Compartment syndrome is typically seen in the setting of acute trauma and osseous injury; however, patients undergoing specific operative interventions are at risk in the perioperative period, along with those treated with restrictive dressings (i.e., casts).
- A timely diagnosis of acute compartment syndrome can be difficult with patients experiencing a wide range of signs and symptoms but is essential to allowing for the best clinical outcomes. Compartment pressure measurements may be helpful, but serial clinical examinations are still the most important diagnostic tool.
- In most cases, urgent fasciotomy with adequate release of elevated compartment pressures will allow for the best possible clinical outcomes; however, all patients should be made aware that they may require multiple procedures and of the complications associated with both compartment syndrome and its treatment.

## Case Studies

### Case 1

A 43-year-old male underwent a right-sided wide excision of a supra-acetabular chondrosarcoma with subsequent reconstruction using an allograft-prosthetic composite total hip replacement. The patient was noted to have diminished dorsalis pedis and posterior tibial pulses on the right side on immediate postoperative assessment. Overnight the patient developed increasing pain in the right lower extremity. Compartments remained soft, but the patient's pulses were no longer palpable or dopplable on examination the next morning.

The vascular surgery service was consulted, and angiography was recommended. The patient was taken to the operating room by the vascular surgery service for angiography which demonstrated a thrombus in the right common femoral artery, and the patient underwent open thrombectomy and common femoral artery reconstruction on postoperative day 1. Dorsalis pedis and posterior tibial pulses were again palpable. In the recovery room, the patient complained of increasing pain and swelling in the right leg despite elevation and ice applied to the affected region. On examination, the patient had clinically worsening of swelling and tense compartments circumferentially in the right leg. Passive extension of the ankle and great toe elicited severe pain. Over the next several hours, the patient began experiencing sensory changes in the foot, ankle, and leg.

The patient was taken back to the operating room later in the day for four-compartment fasciotomies of the right leg. The patient was noted to have bulging but red, robust, contractile muscle with a capacity to bleed in all four compartments. Following the fasciotomies, a negative pressure dressing was applied to the fasciotomy wounds and primary closure deferred.

Over the next several days, the patient continued to complain of right lower extremity pain and progressive loss of sensation in the right lower extremity, eventually resulting in frank numbness below the mid-shin and calf level. The patient's right leg continued to feel clinically tense, and the patient still demonstrated considerable pain with passive extension of the ankle and toes. The patient's urine became a dark cola color, and the patient's laboratory values indicated that the patient was experiencing acute kidney injury from rhabdomyolysis. Ultimately, the patient required hemodialysis.

The patient was again taken back to the operating room for revision fasciotomies by the vascular surgery service and was noted to have bulging muscle with a much less robust appearance than at prior surgery, particularly in the anterior and lateral compartments. The fasciotomies were nevertheless extended, and brownish-gray necrotic muscle was debrided. The wounds were thoroughly irrigated, and again a negative pressure dressing was applied.

The patient remained in the hospital for several weeks. He underwent subsequent revision irrigation and debridement procedures of his fasciotomy sites due to recurrent bleeding in the setting of venous thromboembolism prophylaxis and for concern of infection as the patient remained intermittently febrile for over a week. Once the patient had overcome his recurrent fevers and the wound bed appeared healthy enough to accept a graft, the patient's fasciotomy wounds were covered using split-thickness skin graft from the anterolateral thigh. The patient's kidney function eventually recovered such that he no longer required dialysis. Despite best efforts with bracing and physical therapy to maintain a supple foot and ankle, the patient's right ankle developed an equinus contracture, and claw toes developed. Even several months after the index procedure, the patient had not regained sensation in the right lower extremity below the mid-leg.

This case underscores the importance of a high index of suspicion for compartment syndrome in the setting of revascularization. The patient was taken back to the operating room for fasciotomy, but it was unclear to what degree and exactly how long the patient's right leg had been ischemic. Compartment syndrome must be addressed surgically as soon as possible to afford the best outcomes. Additionally, this patient may have suffered additional sequelae of compartment syndrome due to an incomplete initial release of the anterior and lateral compartments, further delaying effective intervention. A complete release of all affected compartments is essential. A surgeon who is uncomfortable with the anatomy



of the leg may perform an incomplete release due to fear of injuring the superficial peroneal nerve. Regardless of whether vascular or orthopedic surgery performs the fasciotomies, the surgeon must be comfortable with the relevant anatomy to thoroughly decompress all the affected compartments.

## Case 2

An 87-year-old female with mild dementia was admitted for a revision of a primary total knee arthroplasty due to pain and prosthetic loosening. Preoperatively, the patient had full motor strength and intact sensation in all nerve distributions. A revision left total knee arthroplasty was performed under combined spinal/epidural anesthesia and with use of a tourniquet. The tourniquet was let down after 2 hours, but the overall case lasted approximately three-and-a-half hours and ended late in the evening. Two hours postoperatively, the patient had no motor function or sensation below the knee of the left leg when the on-call orthopedic provider made rounds. The left lower extremity was edematous below the compressive postoperative dressing, and distal pulses were faintly palpable on the operative side. Compartments were firm, but the patient was comfortable with passive ankle dorsiflexion. The patient denied issues with pain control. When questioned, the nurse in the recovery room reported that the patient was maxing out her epidural PCA pump. The patient's son was at bedside and admitted to pushing the PCA button for his mother several times because he "didn't want her to suffer and didn't think she understood how to use the pain pump."

The orthopedic provider, following institutional policy, requested that the epidural PCA pump be discontinued, and the on-call anesthesiologist agreed. The resident also loosened the postoperative dressing, flexed the patient's knee, and made a note to check back in an hour. One hour later, the patient's neurological exam had not improved at all, and the provider grew increasingly concerned. He did not want to disturb the attending surgeon overnight, but per hospital policy, the resident immediately escalated his findings to the attending surgeon.

The attending surgeon advised the resident to make the patient NPO in anticipation of possible revision surgery and requested immediate evaluation by the neurology and vascular surgery services. The neurology service recommended a lumbar MRI which was unrevealing and showed no evidence of epidural hematoma. Vascular surgery was also consulted, and at this point the patient had lost palpable distal pulses. An MRI/MRA of the affected leg was performed, revealing a popliteal thrombus and poor perfusion. A thrombectomy, four compartment fasciotomy, and deep dorsalis pedis artery bypass were performed at that time. The anterior and lateral compartments were found to be bulging at the time of fasciotomy, and all visible necrotic muscle was debrided.

The patient underwent one more incisional irrigation and debridement procedure to remove a small amount of additional necrotic muscle. At the next procedure, the medial fasciotomy wound was closed primarily, and a split-thickness skin graft was performed laterally over a clean, viable muscle bed. Neurology continued to follow the patient through her recovery, and the patient spent several months in physical therapy. Ultimately, the patient required an ankle-foot orthosis for ambulation.

Again, this case highlights the risk of compartment syndrome associated with specific surgical procedures, including those with extended tourniquet times. Further, the use of spinal and/or epidural anesthesia can inhibit a postoperative examination, and there should be a low threshold for removing pain catheters if there is any question of a compartment syndrome. Great care must be exercised when running an epidural or peripheral pain catheter, especially in patients whose understanding or ability to comply with a history and exam – such as this patient with mild dementia – may be compromised.

Furthermore, this case illustrates the power of thoughtfully designed institutional policy to influence and often improve postoperative care. This provider followed institutional policy and called the attending surgeon after hours, setting off a chain of events that led to a more timely surgical intervention than would have otherwise resulted. Our institution has implemented an algorithm for all arthroplasty patients affected by neurologic deficits in the early postoperative phase. Nurses, physical therapists, physician assistants, nurse practitioners, residents, fellows, and attending surgeons at our institution have undergone training in this algorithm to better detect and manage postoperative foot drop, as described by Derman and coauthors [59]. Promising early results of this initiative have shown that more orthopedic providers in our hospital are consulting with anesthesia regarding the use of pain management strategies such as epidural PCAs that might mask a potentially serious complication, delaying timely intervention. Algorithms designed to rapidly detect such serious complications and promote early communication and appropriate escalation may become increasingly important as the population ages, orthopedic surgical demand increases, and hospitals face financial pressures to decrease postoperative length of stay.

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## References

1. McQueen MM. Acute compartment syndrome. In: *Fractures in adults*. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2010. p. 689.
2. Sava J, Moelleken A, Waxman K. Cardiac arrest caused by reperfusion injury after lumbar paraspinous compartment syndrome. *J Trauma*. 1999;46:196–7.

3. Carr D, Gilbertson L, Frymoyer J, Krag M, Pope M. Lumbar paraspinal compartment syndrome. A case report with physiologic and anatomic studies. *Spine (Phila Pa 1976)*. 1985;10:816–20.
4. Ferreira J, Galle C, Aminian A, et al. Lumbar paraspinal rhabdomyolysis and compartment syndrome after abdominal aortic aneurysm repair. *J Vasc Surg*. 2003;37:198–201.
5. Minnema BJ, Neligan PC, Quraishi NA, Fehlings MG, Prakash S. A case of occult compartment syndrome and nonresolving rhabdomyolysis. *J Gen Intern Med*. 2008;23:871–4.
6. Paryavi E, Jobin CM, Ludwig SC, Zahir H, Cushman J. Acute exertional lumbar paraspinal compartment syndrome. *Spine (Phila Pa 1976)*. 2010;35:E1529–33.
7. Whitesides TE, Haney TC, Morimoto K, Harada H. Tissue pressure measurements as a determinant for the need of fasciotomy. *Clin Orthop Relat Res*. 1975;113:43–51.
8. 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.
9. 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;37:50–8.
10. McQueen MM, Christie J, Court-Brown CM. Compartment pressures after intramedullary nailing of the tibia. *J Bone Joint Surg Br*. 1990;72:395–7.
11. 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;55:1133–8.
12. Ovre S, Hvaal K, Holm I, Stromsoe K, Nordsletten L, Skjeldal S. Compartment pressure in nailed tibial fractures. A threshold of 30 mmHg for decompression gives 29% fasciotomies. *Arch Orthop Trauma Surg*. 1998;118:29–31.
13. McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome. Who is at risk? *J Bone Joint Surg Br*. 2000;82:200–3.
14. Shadgan B, Pereira G, Menon M, Jafari S, Darlene Reid W, O'Brien PJ. Risk factors for acute compartment syndrome of the leg associated with tibial diaphyseal fractures in adults. *J Orthop Traumatol*. 2015;16(3):185–92.
15. McQuillan WM, Nolan B. Ischaemia complicating injury. A report of thirty-seven cases. *J Bone Joint Surg Br*. 1968;50:482–92.
16. Matsen FA 3rd, Clawson DK. The deep posterior compartmental syndrome of the leg. *J Bone Joint Surg Am*. 1975;57:34–9.
17. Rorabeck CH, Macnab L. Anterior tibial-compartment syndrome complicating fractures of the shaft of the tibia. *J Bone Joint Surg Am*. 1976;58:549–50.
18. Rorabeck CH. The treatment of compartment syndromes of the leg. *J Bone Joint Surg Br*. 1984;66:93–7.
19. Bhattacharyya T. The medical-legal aspects of compartment syndrome. *J Bone Joint Surg Am*. 2004;86-A:864.
20. Bae DS, Kadiyala RK, Waters PM. Acute compartment syndrome in children: contemporary diagnosis, treatment, and outcome. *J Pediatr Orthop*. 2001;21(5):680–8.
21. 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.
22. 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(6):365–9.
23. Odland RM, Schmidt AH. Compartment syndrome ultrafiltration catheters: report of a clinical pilot study of a novel method for managing patients at risk of compartment syndrome. *J Orthop Trauma*. 2011;25(6):358–65.
24. Harvey EJ, Sanders DW, Shuler MS, Lawendy A, Cole AL, AlQahtani SM, et al. What's new in acute compartment syndrome? *J Orthop Trauma*. 2012;26(12):699–702.
25. Hyder N. Compartment syndrome in tibial shaft fracture missed because of a local nerve block. *J Bone Joint Surg Br*. 1996;78:499.
26. 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.
27. Shuler FD. Physicians' ability to manually detect isolated elevations in leg intracompartmental pressure. *J Bone Joint Surg Am*. 2010;92:361.
28. Cascio BM. Documentation of acute compartment syndrome at an academic health-care center. *J Bone Joint Surg Am*. 2005;87:346.
29. Murphy JB. Myositis. *JAMA*. 1914;63:1249.
30. Sassoon A, Riehl J, Rich A, Langford J, Haidukewych G, Pearl G, et al. Muscle viability revisited: are we removing normal muscle? A critical evaluation of dogmatic debridement. *J Orthop Trauma*. 2016;30(1):17–21.
31. Twaddle BC, Amendola A. Compartment syndromes. In: *Skeletal trauma*. 4th ed. St. Louis: Saunders Company; 2008. p. 359.
32. Fulkerson E, Razi A, Tejwani N. Review: acute compartment syndrome of the foot. *Foot Ankle Int*. 2003;24:180–7.
33. Rorabeck CH, Clarke KM. The pathophysiology of the anterior tibial compartment syndrome: an experimental investigation. *J Trauma Acute Care Surg*. 1978;18(5):299–304.
34. Rollins DL, Bernhard VM, Towne JB. Fasciotomy: an appraisal of controversial issues. *Arch Surg*. 1981;116:1474–81.
35. Sheridan GW, Matsen FA 3rd. Fasciotomy in the treatment of the acute compartment syndrome. *J Bone Joint Surg Am*. 1976;58:112–5.
36. Reis ND, Michaelson M. Crush injury to the lower limbs. Treatment of the local injury. *J Bone Joint Surg Am*. 1986;68:414.
37. Finkelstein JA, Hunter GA, Hu RW. Lower limb compartment syndrome: course after delayed fasciotomy. *J Trauma*. 1996;40:342–4.
38. Ritenour AE, Dorlac WC, Fang R, Woods T, Jenkins DH, Flaherty SF, et al. Complications after fasciotomy revision and delayed compartment release in combat patients. *J Trauma*. 2008;64:S153–61. Discussion S161–2.
39. Flynn JM. Acute traumatic compartment syndrome of the leg in children: diagnosis and outcome. *J Bone Joint Surg Am*. 2011;93:937.
40. 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(3):225–33.
41. Lasanianos NG, Kanakaris NK, Roberts CS, Giannoudis PV. Compartment syndrome following lower limb arthroplasty: a review. *Open Orthop J*. 2011;5:181–92.
42. Henson JT, Roberts CS, Giannoudis PV. Gluteal compartment syndrome. *Acta Orthop Belg*. 2009;75:147–52.
43. Tornetta P 3rd, French BG. Compartment pressures during nonreamed tibial nailing without traction. *J Orthop Trauma*. 1997;11:24–7.
44. Nassif JM, Gorczyca JT, Cole JK, Pugh KJ, Pienkowski D. Effect of acute reamed versus unreamed intramedullary nailing on compartment pressure when treating closed tibial shaft fractures: a randomized prospective study. *J Orthop Trauma*. 2000;14:554–8.
45. Wilgis EF. Observations on the effects of tourniquet ischemia. *J Bone Joint Surg Am*. 1971;53:1343–6.
46. Ward CM. Oedema of the hand after fasciotomy with or without tourniquet. *Hand*. 1976;8:179–85.
47. Macfarlane MG, Spooner SJ. Chemical changes in muscle during and after ischaemia. *Br J Exp Pathol*. 1946;27:339–48.
48. Wakai A, Winter DC, Street JT, Redmond PH. Pneumatic tourniquets in extremity surgery. *J Am Acad Orthop Surg*. 2001;9:345–51.

49. Townsend HS, Goodman SB, Schurman DJ, Hackel A, Brock-Utne JG. Tourniquet release: systemic and metabolic effects. *Acta Anaesthesiol Scand*. 1996;40:1234–7.
50. Carlson DA, Dobozi WR, Rabin S. Peroneal nerve palsy and compartment syndrome in bilateral femoral fractures. *Clin Orthop Relat Res*. 1995;(320):115–8.
51. Cascio BM, Buchowski JM, Frassica FJ. Well-limb compartment syndrome after prolonged lateral decubitus positioning. A report of two cases. *J Bone Joint Surg Am*. 2004;86-A:2038–40.
52. Anglen J, Banovetz J. Compartment syndrome in the well leg resulting from fracture-table positioning. *Clin Orthop Relat Res*. 1994;(301):239–42.
53. Mathews PV, Perry JJ, Murray PC. Compartment syndrome of the well leg as a result of the hemilithotomy position: a report of two cases and review of literature. *J Orthop Trauma*. 2001;15:580–3.
54. Danner R, Partanen K, Partanen J, Kettunen K. Iatrogenic compartment syndrome, a follow-up of four cases caused by elastic bandage. *Clin Neurol Neurosurg*. 1989;91:37–43.
55. Hinderland MD, Ng A, Paden MH, Stone PA. Lateral leg compartment syndrome caused by ill-fitting compression stocking placed for deep vein thrombosis prophylaxis during surgery: a case report. *J Foot Ankle Surg*. 2011;50:616–9.
56. Talbot SG, Rogers GF. Pediatric compartment syndrome caused by intravenous infiltration. *Ann Plast Surg*. 2011;67:531–3.
57. 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.
58. Ojike NI, Roberts CS, Giannoudis PV. Foot compartment syndrome: a systematic review of the literature. *Acta Orthop Belg*. 2009;75:573–80.
59. Derman PB, Iyer S, Garner M, Orr S, Felix KJ, Goldberg A, et al. An initiative to standardize the identification of and acute response to postoperative lower-extremity neurological deficits: effects on provider knowledge, confidence, and communication skills. *J Bone Joint Surg Am*. 2016;98(23):e105.