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Clinical Review

EVALUATION AND MANAGEMENT OF ACUTE COMPARTMENT SYNDROME IN THE EMERGENCY DEPARTMENT

Brit Long, MD,* Alex Koyfman, MD,† and Michael Gottlieb, MD, RDMS‡

*Department of Emergency Medicine, Brooke Army Medical Center, Fort Sam Houston, Texas, †Department of Emergency Medicine, The University of Texas Southwestern Medical Center, Dallas, Texas, and ‡Department of Emergency Medicine, Rush University Medical Center, Chicago, Illinois

Reprint Address: Brit Long, MD, Department of Emergency Medicine, Brooke Army Medical Center, 3841 Roger Brooke Drive, Fort Sam Houston, TX 78234

☐ **Abstract—Background: Acute compartment syndrome (ACS) is a time-sensitive surgical emergency caused by increased pressure within a closed compartment. ACS can lead to significant morbidity and mortality if it is not rapidly identified and treated. Objective: This article provides an evidence-based review of the diagnosis and management of ACS, with focused updates for the emergency clinician. Discussion: ACS is the result of decreased perfusion within a compartment and is associated with a number of risk factors, but it occurs most commonly after fractures or trauma to the involved area. It can present with a variety of findings, including pain out of proportion to the injury, paresthesias, pain with passive stretch, tenseness or firmness of the compartment, focal motor or sensory deficits, or decreased pulse or capillary refill time. Pain is typically the earliest finding in patients with ACS. Unfortunately, history and physical examination are typically unreliable and cannot rule out the diagnosis. Measurement of intracompartmental pressures using a pressure monitor is the most reliable test, though noninvasive means of diagnosis are under study. Treatment involves surgical consultation for emergent fasciotomy, as well as resuscitation and management of complications, such as rhabdomyolysis. Conclusion: ACS is a dangerous medical condition requiring rapid diagnosis and management that can result in significant complications if not appropriately diagnosed and treated. Emergency clinician awareness and knowledge of this condition is vital to appropriate management. Published by Elsevier Inc.**

☐ **Keywords—compartment syndrome; pressure; fasciotomy; orthopedics**

INTRODUCTION

Acute compartment syndrome (ACS) is a surgical emergency caused by excessive pressure within a fascial compartment resulting in decreased perfusion within the compartment (1–3). The incidence has been estimated at 0.7–7.3 cases per 100,000 people (2). Morbidity can be significant, with many patients having sustained neurovascular deficits after the injury, if not treated immediately (4). ACS also carries a significant medicolegal risk. One study of legal claims for ACS found that 23% of cases were due to misdiagnosis, and an additional 32% of cases were the result of a delay to definitive treatment, with a mean payout of $574,680 per case (5).

ACS is most common among patients younger than 35 years of age due to stronger fascial structures, increased muscle bulk, and a greater incidence of high-energy injuries (2,6). Older patients may have reduced muscle

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mass, as well as hypertension, which can increase perfusion pressures (7). ACS has also shown a male predominance, occurring 10 times more frequently in males than females (2). While ACS most commonly occurs within 24–48 h of the injury, it can be delayed up to several days (8). The most common cause of ACS is fractures, with tibial fractures having the highest associated risk (2,6,9–11). In fact, lower-leg ACS has been reported in 2–9% of all tibial fractures (12). Importantly, the presence of an open fracture does not exclude compartment syndrome, as the small fascial tears associated with open fractures often do not adequately decompress the compartment (12–15). Of note, compartment syndrome is not always associated with trauma or injury, as in some cases there is no history of injury. Other factors that are associated with developing ACS are listed in Table 1.

METHODS

The authors searched PubMed and Google Scholar for articles using a combination of the keyword *compartment syndrome* and Medical Subject Heading "compartment syndrome." The literature search was restricted to studies published in English. Authors decided which studies to include for the review by consensus. A total of 150 resources were selected for inclusion in this review.

DISCUSSION

Anatomy and Pathophysiology

Compartment syndrome is caused by increased pressure within a fascial or osteofascial compartment (5,50,51)

Table 1. Risk Factors Associated with the Development of Compartment Syndrome

Risk Factors	References
Blunt soft tissue injuries	(16,17)
Burns	(18,19)
Casts	(20)
Deep venous thrombosis	(21)
Electromyography	(22)
Exercise	(23,24)
Extravasation of contrast media	(25,26)
Fractures	(2)
Hematologic diseases	(27–30)
Incorrect patient position during surgery	(31,32)
Infections	(33)
Insect bites	(34,35)
Intramuscular hematomas	(36)
Intraosseous infusions	(37,38)
Intravenous infusions	(39,40)
Osteotomies	(41)
Prolonged immobilization	(42)
Punch biopsies	(43)
Skin and skeletal traction	(44,45)
Snake bites	(46,47)
Vascular procedures	(48,49)

This may be due to either increased volume within a fixed compartment size (e.g., edema, hematomas) or decreased size of the compartment (e.g., extrinsic compression from body positioning, tight casts, or wound dressings) (52). This increased pressure initially compromises the microcirculatory perfusion (5,50,51). However, as the pressure continues to build, lymphatic, capillary, and small venule flow decrease, followed by compromise of the venous and subsequently arterial blood flow, leading to tissue ischemia and necrosis (5,50,51). If the condition is not treated rapidly, this can lead to fibrous tissue degeneration, followed by neurologic damage, contractures, and even amputation (5,6,50,51). As vascular flow decreases, intracompartmental pressures continue to increase, resulting in a vicious cycle of worsening edema. Necrosis can occur rapidly, with one study finding that up to one-third of cases had necrosis of muscle within 3 h after the injury (53). Another study found that neurologic injury was reversible if the fasciotomy was performed within 4 h, while injury was irreversible if the fasciotomy was delayed until after 12 h (54).

The most common location of ACS is the anterior compartment, or anterior tibial compartment, of the lower extremity (55,56). The leg contains five total compartments: anterior, lateral, superficial posterior, deep posterior, and tibialis posterior. Specifically for the anterior compartment, the borders include the tibia, fibula, interosseous membrane, and anterior intermuscular septum, and muscles include the tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius (Figure 1). If there is concern for ACS within the proximal leg or thigh, physicians should monitor the anterior compartment of the thigh due to its more frequent involvement (4,16,58,59), though isolated posterior compartment involvement has been reported in the literature (60). If concern for ACS within the foot is present, the interosseous intracompartmental pressure should be obtained, and for hindfoot injuries, the calcaneal compartment should be monitored (61–67). In the upper extremity, ACS is most common in the volar compartment of the forearm, though isolated dorsal ACS has been reported (64–68). Physicians should also monitor the arm anterior compartment and interosseous compartments of the hand (64,69–71).

History and Physical Examination

It is important to ascertain historical features surrounding the injury, including any risk factors (Table 1) and change in symptoms. Initial findings are often subtle, and early findings may only be detected in conscious patients (2,3,7). Patients with altered mentation, traumatic

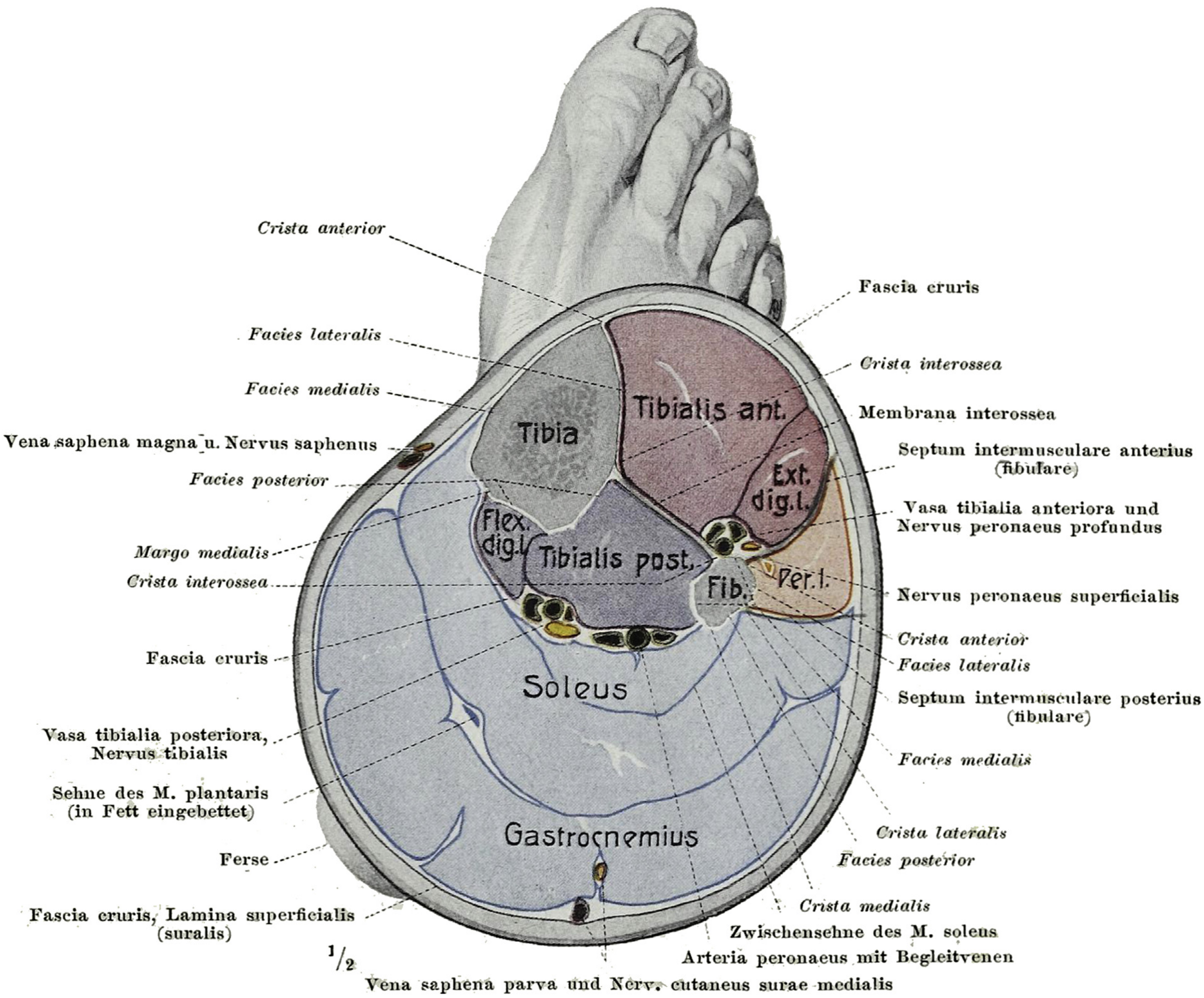

Figure 1. Cross-sectional diagram of the leg (57).

injury, heavy sedation, substance abuse, and at the extremes of age create difficulty in diagnosis. In these settings, the patient may not be able to describe symptoms, or the presentation may be confused with other conditions (15). Severe pain, often out of proportion to the examination, is the earliest symptom (9,72,73). Patients often describe this pain as severe, burning, deep, and worse with passive stretching (3). Unfortunately, pain is typically subjective and has poor sensitivity (9). Later symptoms can include paresthesias, sensory deficits, and focal motor weakness (9,50,51). Pain may be absent in later stages of compartment syndrome (15). On examination, the patient may have pain with palpation, pain with passive stretch, tenseness or firmness of the compartment, swelling of the affected limb, focal motor or sensory deficits, or decreased pulse or capillary refill time (9,50,51). However, paralysis and pulselessness are rare, and the firmness of the compartment is also not reliable (3,55). Visible, palpable swelling is common, occurring in more than half of patients (7). However, the degree of swelling can be subjective; measuring limb circumference and comparing to the contralateral side may reduce subjectivity, but requires study. Examinations should be performed serially and, when possible, by the same clinician each time (14,52). Importantly, digital palpation of the compartment is unreliable, with 49% sensitivity and 79% specificity for ACS of the hand (74) and 24% sensitivity and 55% specificity for ACS of the leg (75). Pulse oximetry may suggest poor limb perfusion if abnormal, but a normal pulse oximetry reading does not exclude compartment syndrome (7,9,15). Table 2 includes the sensitivity, specificity, positive predictive value, and negative predictive value for the most common findings (9).

While signs and symptoms can be suggestive of the diagnosis, they are not definitive (9,76). The literature suggests that a combination of signs and symptoms can

Table 2. Diagnostic Accuracy of Clinical Findings (9)

Finding	Sensitivity, %	Specificity, %	PPV, %	NPV, %
Pain	19	97	14	98
Paresthesia	13	98	15	98
Pain with passive stretch	19	97	14	98
Paresis	13	97	11	98

NPV = negative predictive value; PPV = positive predictive value.

increase the sensitivity for the diagnosis but cannot be used to exclude the diagnosis. The combination of pain with passive stretch, paresthesias, and pain with rest is associated with a sensitivity of 93% for diagnosis, while the addition of paresis to these three findings increases sensitivity to 98% (9). However, using clinical signs or symptoms in isolation is not recommended due to their poor sensitivity. Delay in diagnosis can result in poor patient outcomes, including infection, muscle necrosis, rhabdomyolysis, renal failure, muscle contractures, neurologic injury, chronic pain, fracture, amputation, and death (52,58,65,66,76–84). Clinician inexperience, patient sedation, polytrauma, soft tissue injury, and reliance on clinical signs and symptoms alone are associated with missed diagnosis (9,58,77,85–90).

Diagnostic Testing

Laboratory assessments including creatine kinase (CK), renal function, urinalysis, and urine myoglobin are recommended. CK levels >1000 U/mL or the presence of myoglobinuria are suggestive of ACS, and CK levels will continue to increase during the course of ACS (7,9,15,91). Rhabdomyolysis is present in >40% of traumatic ACS patients (91–93). The renal function panel may demonstrate evidence of renal injury and hyperkalemia, most often due to associated rhabdomyolysis (7,9,15,91). Imaging studies are not typically helpful in diagnosis; however, radiographs are normally obtained to evaluate for fractures and other potential underlying etiologies (52,58,77).

Diagnosis of ACS requires evaluation of the intracompartmental pressure, which is most commonly assessed with direct, invasive monitoring (7,9,50,94). Invasive means of evaluating intracompartmental pressures include needle manometry, the wick catheter, the Whitesides' method, and the solid-state transducer intracompartmental catheter (STC) device (Table 3) (7,50,51,94). One of the first techniques involved a needle manometer placed into the compartment and then connected to a column of saline solution. This technique has been modified by infusing saline into the compartment, but this is not recommended, as the volume may worsen ACS (95–99). Whitesides' method utilizes several i.v. extension tubes, a mercury manometer, three-way stopcock, 20-mL syringe, several 18-gauge needles, and a container of normal saline to produce a closed system that measures the pressure within the compartment by assessing the height of the meniscus in the manometer. More current methods utilize an arterial line transducer system with side-port needles, self-contained measuring systems, or slit catheters (7,9,50,51). This system has demonstrated excellent correlation with the actual intracompartmental pressure. A wick catheter is a modification of the needle catheter and involves fibrils protruding from the bore of a catheter, providing a larger surface area for the assessment of the intracompartmental pressure (97,100). A slit catheter method increases the surface area for measurement by adding an axial cut through the end of the catheter (98,101,102). The slit catheter has demonstrated higher accuracy when compared with the infusion method and similar accuracy to the wick catheter method (101,102) An STC device utilizes a pressure transducer directly within the catheter lumen and does not need a fluid column (103–105). One of the most common versions of this is the Stryker™ monitor (Stryker, Kalamazoo, MI) (7,9,50,51). This device has been shown to be very accurate, especially when compared to other techniques (such as the Whitesides' method), with a sensitivity of 94% and specificity of 98% (73,106,107). However, data evaluating the use of STC monitors in trauma patients are limited (50,51,108).

When evaluating intracompartmental pressures, physicians should place the catheter within 5 cm of the fracture level, with the transducer secured at the level of the measured compartment (77,109–111). Placement of the catheter tip within the fracture may falsely elevate readings (7,112). Additionally, if the transducer is not placed at the same height of the catheter tip, then it may read falsely high or low, depending on the position (77,109–111). Clinicians should target the compartments that correspond to specific muscle and nerve areas. If these pressures are normal but clinical concern remains, then clinicians should obtain repeat compartment pressures and consider obtaining pressures in the surrounding compartments, as well (7,9,50,51,77).

Noninvasive techniques are available, but further studies are required prior to routine use. Near-infrared spectroscopy evaluates the oxygen saturation of tissue by using a monitor that is placed on the skin (7,113). Studies in healthy volunteers have found that this correlates well with intracompartmental pressure (7,114,115). Ultrasound has also been utilized to assess the effect of increased intracompartmental

Table 3. Measuring Intracompartmental Pressures

STC Monitor Instructions	Intravenous Transducer Instructions
1. Obtain consent and prepare a sterile field. Clean and prepare the area. 2. Mark the entry site and anesthetize skin, but avoid injecting into deep tissue. 3. Turn on the Stryker device (left upper portion of the device). 4. Remove the diaphragm unit and needle from the sterile pouch. 5. Connect a prefilled 3-mL syringe to the diaphragm, then attach the needle to the other end of the diaphragm. 6. Open the lid on the device by lifting the blue latch at the bottom corner of the unit, then place the needle and syringe into the unit and secure the lid. 7. Point the device upwards and gently flick air bubbles out of the syringe. 8. Zero the device by holding it perpendicular to the site of entry and pressing the blue button. This will result in "00" appearing on the monitor screen. 9. Remove the needle protective cover and advance the needle 1–3 cm into the skin, while holding the device perpendicular to the entry site. 10. Insert 0.3 mL of saline by gently pressing the syringe hub. 11. Hold the device while the pressures equilibrate, and a number should appear on the device. 12. Remove the needle and device from the site. 13. Repeat for other compartments.	1. Obtain consent and prepare a sterile field. Clean and prepare the area. 2. Mark the entry site and anesthetize skin, but avoid injecting into deep tissue. 3. Attach an 18-gauge needle to i.v. extension tubing and a four-way stopcock. 4. Fill half the tubing with sterile saline, and ensure that no air is present within the tubing. 5. Attach a second i.v. extension tube to the four-way stopcock, with the opposite end attached to a blood pressure manometer or other device capable of measuring pressures. 6. Connect a 20-mL syringe filled with air to the middle attachment of the four-way stopcock 7. Place the 18-gauge needle into the compartment, while keeping the apparatus at the level of the needle. 8. Turn the stopcock so that the syringe is open to both extension tubes. 9. Compress the plunger of the syringe, which slowly increases the pressure in the system. 10. Closely watch the saline/air meniscus. Once the pressure in the closed system has surpassed the tissue pressure within the compartment, the meniscus will move. 11. When the column moves, stop pushing the plunger and read the manometer. This number is the tissue pressure in mm Hg. 12. Remove the needle and device from the site. 13. Repeat for other compartments.

STC = solid-state transducer intracompartmental catheter.

pressure on the arterial pulse waveform, with a sensitivity of 77% and specificity of 93% for correlation with intracompartmental pressures in healthy patients (116). However, few studies have evaluated these modalities in critically ill patients with hypotension.

Treatment

Management includes immediate surgical consultation. The initial treatment of ACS involves removal of constrictive dressings and limb elevation to the level of the heart (7,9,14). Removal of external compressive devices can reduce pressure by 65–85% (50,51,117,118). The limb should be maintained at the level of the heart. It is also important to reduce any displaced fractures, as this can decrease the associated edema (50,51,77). Physicians should treat the patient's pain with analgesics, but regional blocks are not recommended, as they can create difficulty in monitoring the patient's symptoms (31,85,119–122). Resuscitation and restoration of circulating volume are recommended for patients who are hypotensive to restore perfusion pressures (7,9,50,51).

There is significant controversy regarding the critical pressure threshold for performing a fasciotomy in suspected ACS, as well as whether to use intracompartmental pressure alone or differential pressure (ΔP) (7,9,50,51,77,123). The normal resting intracompartmental pressure within muscle is approximately 8–10 mm Hg in adults and 10–15 mm Hg in children (124–127). Prior literature recommended using an absolute intracompartmental pressure of 30–40 mm Hg as a threshold for fasciotomy (70,95,100,123,128–130). However, individuals can vary widely with respect to their intracompartmental pressures (7,9,15,100). Additionally, different compartments have different pressure thresholds (50,51). For example, an intracompartmental pressure > 15–20 mm Hg in the hand is a relative indication for fasciotomy (7,73). Moreover, the perfusion pressure, which is more important than the intracompartmental pressure, can also vary, based on numerous factors (e.g., age, pre-existing hypertension, vascular disease, medications) (13,94,100,106,131–133). Differential pressure is defined as the diastolic blood pressure minus the intracompartmental pressure (7,9,50,51). Several studies have suggested that differential pressures of $\leq$ 30 mm Hg are an indication for fasciotomy, though critical ΔP may be higher in muscle undergoing trauma or ischemia (7,13,56,101,110,134–136). Of note, if vascular injury is present, fasciotomy is recommended before vascular exploration (50,51,77).

Table 4. Indications for Fasciotomy

Indications
Clinical signs or symptoms strongly suggestive of ACS
Intracompartmental pressure > 30 mm Hg or ΔP < 20 mm Hg with concern for ACS (Note: ΔP < 30 mm Hg is a relative indication)
Intracompartmental pressure > 20 mm Hg in the presence of hypotension
Interruption in arterial perfusion for ≥ 4 h

ACS = acute compartment syndrome; ΔP = differential pressure.

The timing of intracompartmental pressure assessments is important to ensure rapid diagnosis and intervention. Earlier fasciotomy is associated with improved patient outcomes, including decreased muscle injury, nerve injury, and death (7,53,54,56,81,136). Once a motor nerve deficit has occurred, patients rarely recover fully after fasciotomy (3,7,77,81). However, there is a dynamic relationship among blood pressure, intracompartmental pressure, and duration of time (3,7,9,50,51,136). Higher intracompartmental pressures cause faster and more severe damage, while lower intracompartmental pressures can be sustained for longer periods of time before similar damage occurs (3,7,50,51). There is variation among clinicians with respect to the approach to evaluating for ACS. Some use repeat clinical assessments, while others use continuous monitoring with a catheter attached to an arterial transducer in patients for whom assessment is difficult (3,7,13,137–139). Single compartment measurements may lead to overdiagnosis and overtreatment (3,7,73,77). Several protocols have evaluated the timing of measurements, comparing continuous pressure monitoring with serial monitoring (3,7,73,77). For continuous compartment measurements, clinical symptoms in combination with ΔP (<30 mm Hg) resulted in a sensitivity of 61% and specificity of 97% for ACS, while using ΔP in isolation resulted in a sensitivity of 89% and specificity of 65% (3,7,139). Therefore, basing the diagnosis and

Table 5. Approaches for Fasciotomy (147,148)

Compartment	Incisions
Foot	Typically includes two dorsal incisions over second to fourth metatarsals for forefoot decompression; medial incision to decompress calcaneal, medial, and superficial compartments; and lateral incision starting at the lateral malleolus extending to the forefoot between the fourth and fifth metatarsals.
Lower leg	Anterolateral fasciotomy may include single lateral incision or lateral and medial incisions together, 15–20 cm in length. Lateral and medial incisions are recommended for adequate decompression. If tibial fracture is present, single-incision technique can be considered (double incisions reduce soft tissue support and reduce fracture stability).
Thigh	Fasciotomy typically includes single incision, though double incisions can be used. Single incision can decompress all three compartments, but two parallel fascia lata incisions can be used to prevent muscle herniation. For lateral and posterior ACS, incisions begin at intertrochanteric line and extend to lateral epicondyle.
Forearm	Decompress using two incisions with the volar and dorsal incisions; these are usually formed concurrently.
Hand	Carpal tunnel requires decompression, as well as incisions to decompress interossei. Mid-lateral incisions on noncontact or nondominant side can be used for digital decompression.

ACS = acute compartment syndrome.

Figure 2. Fasciotomy procedure of lower leg (146).

intervention on single measurements can potentially result in unnecessary fasciotomies, with false-positive rates approaching 35% (138). One study demonstrated that monitoring intracompartmental pressures over 2 h and using a ΔP < 30 mm Hg over that time as diagnostic criterion for ACS displayed a sensitivity of 94% and specificity of 98.4% (13,73,77). We recommend using a single measurement for patients who are clinically evaluable, with repeat assessment if the first is normal and clinical suspicion for ACS remains, while using continuous monitoring over at least 2 h for those patients who are not clinically evaluable (3,7,50,51). Performing continuous monitoring for > 2 h in patients who are alert and may be clinically observed for worsening symptoms may reduce overtreatment (7,9,13). When using ΔP, a ΔP < 20 mm Hg is a definitive indication for fasciotomy, with < 30 mm Hg a relative indication (3,7,13,56,134,135). Using ΔP may also assist in diagnosing ACS in trauma patients with hypotension (7,9,13,73,77). Clinical signs of ACS and absolute intracompartmental pressures > 30 mm Hg also require definitive therapy (3,7,50,51). Table 4 displays a summary of the indications for performing a fasciotomy. Unfortunately, if ACS has been present for 3–4 days, fasciotomy is unlikely to be beneficial (7,9,140–145). In these cases, the compartment should not be opened, and instead it should be allowed to form scar tissue (3,7,9).

Table 6. Leg Fasciotomy: Double- and Single-Incision Techniques (147,148)

Two-incision technique*
Anterolateral incision: 1. Obtain consent if able. 2. Ensure patient is supine with small bump under the affected extremity's hip. 3. Sterilely prepare and drape the extremity. 4. Perform one longitudinal 15–20 cm incision along anterolateral surface to decompress anterior and lateral compartments. 5. The skin incision is made at the center between the tibial crest and fibula shaft. The proximal landmark is approximately 3 cm distal of the tibial tuberosity, and the lateral malleolus is the distal landmark. 6. Dissect the subcutaneous tissue to expose the fascia, and then identify the intermuscular septum between the anterior and lateral compartments. 7. Make a small transverse incision over the fascia, centered on the intermuscular septum, which extends over both compartments. Do not cut through the superficial peroneal nerve. 8. To release the anterior compartment, incise the fascia over the anterior compartment by extending the transverse fascia incision longitudinally. Scissors can be used for incising fascia. 9. To release the lateral compartment in line with the fibula shaft, incise the fascia over the lateral compartment by extending the transverse fascia incision longitudinally. Scissors can be used for incising fascia. Proximally, stop the incision 5 cm distal to the head of the fibula, and distally, cut in the direction of the lateral malleolus. This minimizes damage to the peroneal nerves. 10. Cover with wound dressing. Do not close the fascia or skin in the emergent setting.
Posteromedial incision: 1. Perform one longitudinal 15–20 cm incision along the tibia posteromedial border. 2. The skin incision is made 2 cm posterior of the tibia posteromedial border. The proximal landmark is approximately 3 cm distal to the tibia tuberosity, and the medial malleolus is the distal landmark. 3. Dissect the subcutaneous tissue to expose the fascia over the superficial and deep posterior compartments. Protect the saphenous nerve and vein. 4. To release the superficial posterior compartment, make a small transverse incision over the fascia 2 cm posterior to the skin incision. This incision is extended longitudinally over the length of the gastrocnemius–soleus component. Scissors can be used for incising fascia. 5. To release the deep posterior compartment, dissect the superior posterior compartment from the tibia posteromedial border. Release the fascia over the soleus. Incise the fascia longitudinally over the flexor digitorum longus muscle (and tibialis posterior muscle if required). Scissors can be used for incising fascia. Do not cut through the posterior tibial neurovascular bundle. 6. Cover with wound dressing. Do not close the fascia or skin in the emergent setting.
Single-incision technique 1. Obtain consent if able. 2. Ensure patient is supine with small bump under the affected extremity's hip. 3. Sterilely prepare and drape the extremity. 4. Start the lateral incision at the head of the fibula, followed by extension distally along the fibula to the ankle. 5. Dissect the subcutaneous tissue to expose the fascia, and then identify the intermuscular septum between the anterior and lateral compartments. Protect the superficial peroneal nerve. 6. Start 1 cm anterior to the intermuscular septum to release the anterior compartment. 7. Start 1 cm posterior to the septum to release the lateral compartment. 8. Perform a fasciotomy of the superficial posterior compartment, which is over the gastrocnemius–soleus complex. 9. Move the lateral compartment anteriorly and superficial peroneal compartment posteriorly, which should allow access to the deep posterior compartment. 10. Release the deep posterior compartment by identifying the interosseous membrane and the fibula posterior aspect. 11. Cover with wound dressing. Do not close the fascia or skin in the emergent setting.
Equipment: Sterile gloves and drapes, scalpel, dissecting scissors, soft tissue retractors, electrocautery if available, wound dressing (preferably wound vacuum-assisted closure)

* Lateral and medial incisions are recommended for adequate decompression in almost all situations.

Definitive therapy is compartment release through fasciotomy (Figure 2) (91,143,144). Specific techniques for release depend on the affected extremity and the number of compartments (Table 5) (143–148). A general overview of the technique for fasciotomy of the leg is detailed in Table 6 (147,148).

Hyperbaric oxygen (HBO) is a potential adjunctive treatment, as it can promote hyperoxic vasoconstriction, which may reduce edema and improve tissue oxygen (149,150). HBO may also assist in the survival of marginally viable tissue. However, this should not delay fasciotomy and early decompression (149,150). Further studies are needed to determine how HBO can fit in the treatment algorithm.

Disposition

Patients require emergent consultation with a surgeon, preferably an orthopedic or plastic surgeon, with transfer to the operating room and admission for monitoring (3,7,9,77). A patient at risk for ACS should be admitted for close monitoring due to the potential for poor outcome with missed diagnosis and potential requirement for emergent fasciotomy (3,7,9).

CONCLUSIONS

ACS is a surgical emergency caused by increased pressure within a closed compartment. ACS is a time-sensitive condition and can lead to significant morbidity and mortality if it is not rapidly identified and treated. ACS is associated with a number of risk factors but occurs most frequently after a fracture or trauma to the involved area. Findings can include pain out of proportion to the injury, paresthesias, pain with passive stretch, tenseness or firmness of the compartment, focal motor or sensory deficits, or decreased pulse or capillary refill time. Pain is the earliest finding in patients with ACS; however, findings on history and physical examination should not be used to rule the diagnosis in or out. Measurement of intracompartmental pressures using a pressure monitor is the most reliable test. Noninvasive means of measurement require further study. Treatment involves resuscitation if hypotensive, providing analgesia, removing constrictive dressing, placing the limb at heart level, and surgical consultation for emergent fasciotomy, which depends on the specific compartment affected, along with management of complications such as rhabdomyolysis.

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REFERENCES

1. Matsen FA 3rd, Krugmire RB Jr. Compartmental syndromes. Surg Gynecol Obstet 1978;147:943–9.
2. McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome. Who is at risk? J Bone Joint Surg Br 2000; 82:200–3.
3. Donaldson J, Haddad B, Khan WS. The pathophysiology, diagnosis and current management of acute compartment syndrome. Open Orthop J 2014;8:185–93.
4. Schwartz JT Jr, Brumback RJ, Lakatos R, et al. Acute compartment syndrome of the thigh. A spectrum of injury. J Bone Joint Surg Am 1989;71:392–400.
5. Marchesi M, Marchesi A, Calori GM, et al. A sneaky surgical emergency: acute compartment syndrome. Retrospective analysis of 66 closed claims, medico-legal pitfalls and damages evaluation. Injury 2014;45(suppl 6):S16–20.
6. Lollo L, Grabinsky A. Clinical and functional outcomes of acute lower extremity compartment syndrome at a major trauma hospital. Int J Crit Illn Inj Sci 2016;6:133–42.
7. Duckworth AD, McQueen MM. The diagnosis of acute compartment syndrome. JFJS Rev 2017;5(12):e1.
8. Ferlic PW, Singer G, Kraus T, Eberl R. The acute compartment syndrome following fractures of the lower leg in children. Injury 2012;43:1743–6.
9. Ulmer T. The clinical diagnosis of compartment syndrome of the lower leg: are clinical findings predictive of the disorder? J Orthop Trauma 2002;16:572–7.
10. Erdös J, Dlaska C, Szatmary P, et al. Acute compartment syndrome in children: a case series in 24 patients and review of the literature. Int Orthop 2011;35:569–75.
11. Bodansky D, Doorgakant A, Alsousou J, et al. Acute compartment syndrome: do guidelines for diagnosis and management make a difference? Injury 2018;49:1699–702.
12. Mauser N, Gissel H, Henderson C, et al. Acute lower-leg compartment syndrome. Orthopedics 2013;36:619–24.
13. McQueen MM, Court-Brown CM. Compartment monitoring in tibial fractures. The pressure threshold for decompression. J Bone Joint Surg Br 1996;78:99–104.
14. Taylor RM, Sullivan MP, Mehta S. Acute compartment syndrome: obtaining diagnosis, providing treatment, and minimizing medicolegal risk. Curr Rev Musculoskelet Med 2012;5:206–13.
15. Via AG, Oliva F, Spoliti M, Maffulli N. Acute compartment syndrome. Muscles Ligaments Tendons J 2015;5:18–22.
16. Mithöfer K, Lhowe DW, Altman GT. Delayed presentation of acute compartment syndrome after contusion of the thigh. J Orthop Trauma 2002;16:436–8.
17. Gorman PW, McAndrew MP. Acute anterior compartmental syndrome of the thigh following contusion. A case report and review of the literature. J Orthop Trauma 1987;1:68–70.
18. Brown RL, Greenhalgh DG, Kagan RJ, Warden GD. The adequacy of limb escharotomies-fasciotomies after referral to a major burn center. J Trauma 1994;37:916–20.
19. Li X, Liang D, Liu X. Compartment syndrome in burn patients. A report of five cases. Burns 2002;28:787–9.
20. Muchow RD, Flannery WD, Miedaner JA, et al. Compartment syndrome after serial casting in spastic diplegic cerebral palsy: a case report. Arch Phys Med Rehabil 2010;91:653–5.
21. Rahm M, Probe R. Extensive deep venous thrombosis resulting in compartment syndrome of the thigh and leg. A case report. J Bone Joint Surg Am 1994;76:1854–7.
22. Vaienti L, Vourtsis S, Urzola V. Compartment syndrome of the forearm following an electromyographic assessment. J Hand Surg Br 2005;30:656–7.
23. McKee MD, Jupiter JB. Acute exercise-induced bilateral anterolateral leg compartment syndrome in a healthy young man. Am J Orthop (Belle Mead NJ) 1995;24:862–4.
24. Mendes AF Jr, Neto JDM, Heringer EM, et al. Hyperbaric oxygen therapy as treatment for bilateral arm compartment syndrome after CrossFit: case report and literature review. Undersea Hyperb Med 2018;45:209–15.

25. D'Asero G, Tati E, Petrocelli M, et al. Compartment syndrome of the hand with acute bullous eruption due to extravasation of computed tomography contrast material. Eur Rev Med Pharmacol Sci 2010;14:643–6.
26. Belzunegui T, Louis CJ, Torrededia L, Oteiza J. Extravasation of radiographic contrast material and compartment syndrome in the hand: a case report. Scand J Trauma Resusc Emerg Med 2011; 19:9.
27. Dincer HE, Raza T. Compartment syndrome and fatal rhabdomyolysis in sickle cell trait. WMJ 2005;104:67–71.
28. Way A, Ganesan S, McErlain M. Multiple limb compartment syndromes in a recruit with sickle cell trait. J R Army Med Corps 2011;157:182–3.
29. Jones G, Thompson K, Johnson M. Acute compartment syndrome after minor trauma in a patient with undiagnosed mild haemophilia B. Lancet 2013;382(9905):1678.
30. Rodriguez-Merchan EC. Acute compartment syndrome in haemophilia. Blood Coagul Fibrinolysis 2013;24:677–82.
31. Pollard RL, O'Broin E. Compartment syndrome following prolonged surgery for breast reconstruction with epidural analgesia. J Plast Reconstr Aesthet Surg 2009;62:e648–9.
32. Rao MM, Jayne D. Lower limb compartment syndrome following laparoscopic colorectal surgery: a review. Colorectal Dis 2011;13: 494–9.
33. Jamil W, Khan I, Robinson P, Thalava R. Acute compartment syndrome of the forearm secondary to infection within the space of Parona. Orthopedics 2011;34:e584–7.
34. McKnight AJ, Koshy JC, Xue AS, Shetty M, Bullocks JM. Pediatric compartment syndrome following an insect bite: a case report. Hand (N Y) 2011;6:337–9.
35. Hardwicke J, Srivastava S. Volkmann's contracture of the forearm owing to an insect bite: a case report and review of the literature. Ann R Coll Surg Engl 2013;95:e36–7.
36. Rööser B, Bengtson S, Hägglund G. Acute compartment syndrome from anterior thigh muscle contusion: a report of eight cases. J Orthop Trauma 1991;5:57–9.
37. Galpin RD, Kronick JB, Willis RB, Frewen TC. Bilateral lower extremity compartment syndromes secondary to intraosseous fluid resuscitation. J Pediatr Orthop 1991;11:773–6.
38. Moen TC, Sarwark JF. Compartment syndrome following intraosseous infusion. Orthopedics 2008;31:815.
39. Erickson BA, Yap RL, Pazona JF, Hartigan BJ, Smith ND. Mannitol extravasation during partial nephrectomy leading to forearm compartment syndrome. Int Braz J Urol 2007;33: 68–71.
40. Talbot SG, Rogers GF. Pediatric compartment syndrome caused by intravenous infiltration. Ann Plast Surg 2011;67:531–3.
41. Pinkowski JL, Weiner DS. Complications in proximal tibial osteotomies in children with presentation of technique. J Pediatr Orthop 1995;15:307–12.
42. Drey IA, Baruch H. Acute compartment syndrome of the calf presenting after prolonged decubitus position. Orthopedics 2008;31: 184.
43. Faulhaber J, Ehmke H, Koenen W, Weiss B, Goerdt S, Schneider SW. Compartment syndrome of the lower leg after punch biopsy. Dermatol Surg 2012;38:277–8.
44. Naito M, Ogata K. Acute volar compartment syndrome during skeletal traction in distal radius fracture. A case report. Clin Orthop Relat Res 1989;Apr(241):234–7.
45. Janzing H, Broos P, Romnens P. Compartment syndrome as complication of skin traction, in children with femoral fractures. Acta Chir Belg 1996;96:135–7.
46. Vigasio A, Battiston B, De Filippo G, Brunelli G, Calabrese S. Compartmental syndrome due to viper bite. Arch Orthop Trauma Surg 1991;110:175–7.
47. Pietrangiolillo Z, Frassoldati R, Leonelli V, et al. Compartment syndrome after viper-bite in toddler: case report and review of literature. Acta Biomed 2012;83:44–50.
48. Busch T, Sîrbu H, Zenker D, Dalichau H. Vascular complications related to intraaortic balloon counterpulsation: an analysis of ten years experience. Thorac Cardiovasc Surg 1997;45:55–9.
49. Mesfin A, Lum YW, Nayfeh T, Mears SC. Compartment syndrome in patients with massive venous thrombosis after inferior vena cava filter placement. Orthopedics 2011;34:229.
50. Schmidt AH. Acute compartment syndrome. Orthop Clin North Am 2016;47:517–25.
51. Schmidt AH. Acute compartment syndrome. Injury 2017;48(suppl 1):S22–5.
52. Mabvuure NT, Malahias M, Hindocha S, et al. Acute compartment syndrome of the limbs: current concepts and management. Open Orthop J 2012;6:535–43.
53. Vaillancourt C, Shrier I, Vandal A, et al. Acute compartment syndrome: how long before muscle necrosis occurs? CJEM 2004;6: 147–54.
54. Rorabeck CH, Clarke KM. The pathophysiology of the anterior tibial compartment syndrome: an experimental investigation. J Trauma 1978;18:299–304.
55. Al-Dadah OQ, Darrah C, Cooper A, et al. Continuous compartment pressure monitoring vs. clinical monitoring in tibial diaphyseal fractures. Injury 2008;39:1204–9.
56. McQueen MM, Christie J, Court-Brown CM. Acute compartment syndrome in tibial diaphyseal fractures. J Bone Joint Surg Br 1996; 78:95–8.
57. Wikimedia Commons. File:Braus_1921_293.png. Available at: https://commons.wikimedia.org/wiki/File:Braus_1921_293.png. Accessed May 14, 2018.
58. Mithofer K, Lhowe DW, Vrahas MS, et al. Clinical spectrum of acute compartment syndrome of the thigh and its relation to associated injuries. Clin Orthop Relat Res 2004;425:223–9.
59. Mithoefer K, Lhowe DW, Vrahas MS, et al. Functional outcome after acute compartment syndrome of the thigh. J Bone Joint Surg Am 2006;88:729–37.
60. Mallo GC, Stanat SJ, Al-Humadi M, Divaris N. Posterior thigh compartment syndrome as a result of a basketball injury. Orthopedics 2009;32:923.
61. Frink M, Hildebrand F, Krettek C, et al. Compartment syndrome of the lower leg and foot. Clin Orthop Relat Res 2010;468:940–50.
62. Myerson M. Diagnosis and treatment of compartment syndrome of the foot. Orthopedics 1990;13:711–7.
63. Myerson M, Manoli A. Compartment syndromes of the foot after calcaneal fractures. Clin Orthop Relat Res 1993;290:142–50.
64. Tollens T, Janzing H, Broos P. The pathophysiology of the acute compartment syndrome. Acta Chir Belg 1998;98:171–5.
65. Kalyani BS, Fisher BE, Roberts CS, Giannoudis PV. Compartment syndrome of the forearm: a systematic review. J Hand Surg Am 2011;36:535–43.
66. Eaton RG, Green WT. Volkmann's ischemia. A volar compartment syndrome of the forearm. Clin Orthop Relat Res 1975;113:58–64.
67. Stockley I, Harvey IA, Getty CJ. Acute volar compartment syndrome of the forearm secondary to fractures of the distal radius. Injury 1988;19:101–4.
68. Hwang RW, de Witte PB, Ring D. Compartment syndrome associated with distal radial fracture and ipsilateral elbow injury. J Bone Joint Surg Am 2009;91:642–5.
69. Prasarn ML, Ouellette EA. Acute compartment syndrome of the upper extremity. J Am Acad Orthop Surg 2011;19:49–58.
70. Halpern AA, Greene R, Nichols T, Burton DS. Compartment syndrome of the interosseous muscles: early recognition and treatment. Clin Orthop Relat Res 1979;140:23–5.
71. Diminick M, Shapiro G, Cornell C. Acute compartment syndrome of the triceps and deltoid. J Orthop Trauma 1999;13:225–7.
72. Shadgan B, Pereira G, Menon M, et al. Risk factors for acute compartment syndrome of the leg associated with tibial diaphyseal fractures in adults. J Orthop Traumatol 2015;16:185–92.
73. McQueen MM, Duckworth AD, Aitken SA, Court-Brown CM. The estimated sensitivity and specificity of compartment pressure monitoring for acute compartment syndrome. J Bone Joint Surg Am 2013;95:673–7.
74. Wong JC, Vosbikian MM, Dwyer JM, Ilyas AM. Accuracy of measurement of hand compartment pressures: a cadaveric study. J Hand Surg Am 2015;40:701–6.

75. Shuler FD, Dietz MJ. Physicians' ability to manually detect isolated elevations in leg intracompartmental pressure. J Bone Joint Surg Am 2010;92:361–7.
76. Gourgiotis S, Villias C, Germano S, et al. Acute limb compartment syndrome: a review. J Surg Educ 2007;64:178–86.
77. McQueen MM. Acute compartment syndrome. In: Bucholz RW, Court-Brown CM, Heckman JD, Tornetta P III, eds. Rockwood and Green's Fractures in Adults. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2010:689–708.
78. Gelberman RH, Zakaib GS, Mubarak SJ, et al. Decompression of forearm compartment syndromes. Clin Orthop Relat Res 1978; 134:225–9.
79. Holden CE. The pathology and prevention of Volkmann's ischaemic contracture. J Bone Joint Surg Br 1979;61-B:296–300.
80. Finkelstein JA, Hunter GA, Hu RW. Lower limb compartment syndrome: course after delayed fasciotomy. J Trauma 1996;40:342–4.
81. Sheridan GW, Matsen FA 3rd. Fasciotomy in the treatment of the acute compartment syndrome. J Bone Joint Surg Am 1976;58: 112–5.
82. 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.
83. Mullett H, Al-Abed K, Prasad CV, O'Sullivan M. Outcome of compartment syndrome following intramedullary nailing of tibial diaphyseal fractures. Injury 2001;32:411–3.
84. Hope MJ, McQueen MM. Acute compartment syndrome in the absence of fracture. J Orthop Trauma 2004;18:220–4.
85. Mubarak SJ, Wilton NC. Compartment syndromes and epidural analgesia. J Pediatr Orthop 1997;17:282–4.
86. Harrington P, Bunola J, Jennings AJ, Bush DJ, Smith RM. Acute compartment syndrome masked by intravenous morphine from a patient-controlled analgesia pump. Injury 2000;31:387–9.
87. Richards H, Langston A, Kulkarni R, Downes EM. Does patient controlled analgesia delay the diagnosis of compartment syndrome following intramedullary nailing of the tibia? Injury 2004;35:296–8.
88. Davis ET, Harris A, Keene D, et al. The use of regional anaesthesia in patients at risk of acute compartment syndrome. Injury 2006;37: 128–33.
89. Mar GJ, Barrington MJ, McGuirk BR. Acute compartment syndrome of the lower limb and the effect of postoperative analgesia on diagnosis. Br J Anaesth 2009;102:3–11.
90. 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.
91. Raza H, Mahapatra A. Acute compartment syndrome in orthopedics: causes, diagnosis, and management. Adv Orthop 2015; 2015:543412.
92. Tsai WH, Hunag ST, Liu WC. High risk of rhabdomyolysis and acute kidney injury after traumatic limb compartment syndrome. Ann Plast Surg 2015;74:S158–61.
93. Lima RS, da Silva Junior GB, Liborio AB, et al. Acute kidney injury due to rhabdomyolysis. Saudi J Kidney Dis Transpl 2008; 19:721–9.
94. Matsen FA 3rd, Winquist RA, Krugmire RB Jr. Diagnosis and management of compartmental syndromes. J Bone Joint Surg Am 1980;62:286–91.
95. Matsen FA 3rd, Mayo KA, Sheridan GW, Krugmire RB Jr. Monitoring of intramuscular pressure. Surgery 1976;79:702–9.
96. Whitesides TE Jr, Haney TC, Harada H, et al. A simple method for tissue pressure determination. Arch Surg 1975;110:1311–3.
97. Mubarak SJ, Hargens AR, Owen CA, et al. The wick catheter technique for measurement of intramuscular pressure. J Bone Joint Surg Am 1976;58:1016–20.
98. Rorabeck C, Castle G, Hardie R, Logan J. Compartmental pressure measurements: an experimental investigation using the slit catheter. J Trauma 1981;21:446–9.
99. Barnes MR, Gibson MJ, Scott J, et al. A technique for the long term measurement of intracompartmental pressure in the lower leg. J Biomed Eng 1985;7:35–9.
100. Mubarak SJ, Owen CA, Hargens AR, et al. Acute compartment syndromes: diagnosis and treatment with the aid of the wick catheter. J Bone Joint Surg Am 1978;60:1091–5.
101. Moed BR, Thorderson PK. Measurement of intracompartmental pressure: a comparison of the slit catheter, side-ported needle, and simple needle. J Bone Joint Surg Am 1993;75:231–5.
102. Shakespeare DT, Henderson NJ, Clough G. The slit catheter: a comparison with the wick catheter in the measurement of compartment pressure. Injury 1982;13:404–8.
103. McDermott AG, Marble AE, Yabsley RH, Phillips MB. Monitoring dynamic anterior compartment pressures during exercise. A new technique using the STIC catheter. Am J Sports Med 1982;10:83–9.
104. McDermott AG, Marble AE, Yabsley RH. Monitoring acute compartment pressures with the S.T.I.C. catheter. Clin Orthop Relat Res 1984;190:192–8.
105. Willy C, Gerngross H, Sterk J. Measurement of intracompartmental pressure with use of a new electronic transducer-tipped catheter system. J Bone Joint Surg Am 1999;81:158–68.
106. Large TM, Agel J, Holtzman DJ, et al. Interobserver variability in the measurement of lower leg compartment pressures. J Orthop Trauma 2015;29:316–21.
107. Boody AR, Wongworawat MD. Accuracy in the measurement of compartment pressures: a comparison of three commonly used devices. J Bone Joint Surg Am 2005;87:2415–22.
108. Collinge C, Kuper M. Comparison of three methods for measuring intracompartmental pressure in injured limbs of trauma patients. J Orthop Trauma 2010;24:364–8.
109. 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.
110. Matava MJ, Whitesides TE Jr, Seiler JG 3rd, et al. Determination of the compartment pressure threshold of muscle ischemia in a canine model. J Trauma 1994;37:50–8.
111. Saikia KC, Bhattacharya TD, Agarwala V. Anterior compartment pressure measurement in closed fractures of leg. Indian J Orthop 2008;42:217–21.
112. Harris IA, Kadir A, Donald G. Continuous compartment pressure monitoring for tibia fractures: does it influence outcome? J Trauma 2006;60:1330–5. discussion 1335.
113. Garr JL, Gentilello LM, Cole PA, et al. Monitoring for compartmental syndromeusing near-infrared spectroscopy: a noninvasive, continuous, transcutaneous monitoring technique. J Trauma 1999; 46:613–8.
114. Arbabi S, Brundage SI, Gentilello LM. Near-infrared spectroscopy: a potential method for continuous, transcutaneous monitoring for compartmental syndrome in critically injured patients. J Trauma 1999;47:829–33.
115. Gentilello LM, Sanzone A, Wang L, et al. Near-infrared spectroscopy versus compartment pressure for the diagnosis of lower extremity compartmental syndrome using electromyography-determined measurements of neuromuscular function. J Trauma 2001;51:1–8. discussion 8–9.
116. Lynch JE, Lynch JK, Cole SL, et al. Noninvasive monitoring of elevated intramuscular pressure in a model compartment syndrome via quantitative fascial motion. J Orthop Res 2009;27: 489–94.
117. Mars M, Hadley GP. Raised compartmental pressure in children: a basis for management. Injury 1998;29:183–5.
118. Hoover TJ, Siefert JA. Soft tissue complications of orthopaedic emergencies. Emerg Med Clin North Am 2000;18:115.
119. Cometa MA, Esch AT, Boezaart AP. Did continuous femoral and sciatic nerve block obscure the diagnosis or delay the treatment of acute lower leg compartment syndrome? A case report. Pain Med 2011;5:823–8.
120. Fowler SJ, Symons J, Sabato S, Myles PS. Epidural analgesia compared with peripheral nerve blockade after major knee surgery: a systematic review and meta-analysis of randomized trials. Br J Anaesth 2008;2:154–64.

121. Uzel AP, Steinmann G. Thigh compartment syndrome after intramedullary femoral nailing: Possible femoral nerve block influence on diagnosis timing. Orthop Traumatol Surg Res 2009;4:309–13.
122. Hyder N, Kessler S, Jennings AG, De Boer PG. Compartment syndrome in tibial shaft fracture missed because of a local nerve block. J Bone Joint Surg Br 1996;3:499–500.
123. Rorabeck CH. The treatment of compartment syndromes of the leg. J Bone Joint Surg Br 1984;66:93–7.
124. Giannoudis PV, Tzioupis C, Pape HC. Early diagnosis of tibial compartment syndrome: continuous pressure measurement or not? Injury 2009;40:341–2.
125. Shadgan B, Menon M, Sanders D, et al. Current thinking about acute compartment syndrome of the lower extremity. Can J Surg 2010;53:329–34.
126. Shadgan B, Menon M, O'Brien PJ, Reid WD. Diagnostic techniques in acute compartment syndrome of the leg. J Orthop Trauma 2008;22:581–7.
127. Staudt JM, Smeulders MJ, van der Horst CM. Normal compartment pressures of the lower leg in children. J Bone Joint Surg Br 2008;90:215–9.
128. Blick SS, Brumback RJ, Poka A, et al. Compartment syndrome in open tibial fractures. J Bone Joint Surg Am 1986;68:1348–53.
129. Hargens AR, Akeson WH, Mubarak SJ, et al. Fluid balance within the canine anterolateral compartment and its relationship to compartment syndromes. J Bone Joint Surg Am 1978;60: 499–505.
130. Allen MJ, Stirling AJ, Crawshaw CV, Barnes MR. Intracompartmental pressure monitoring of leg injuries. An aid to management. J Bone Joint Surg Br 1985;67:53–7.
131. Matsen FA 3rd, Veith RG. Compartmental syndromes in children. J Pediatr Orthop 1981;1:33–41.
132. 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.
133. Heckman MM, Whitesides TE Jr, Grewe SR, et al. Histologic determination of the ischemic threshold of muscle in the canine compartment syndrome model. J Orthop Trauma 1993;7:199–210.
134. White TO, Howell GE, Will EM, et al. Elevated intramuscular compartment pressures do not influence outcome after tibial fracture. J Trauma 2003;55:1133–8.
135. Ozkayin N, Aktuglu K. Absolute compartment pressure versus differential pressure for the diagnosis of compartment syndrome in tibial fractures. Int Orthop 2005;29:396–401.
136. Matsen FA 3rd. Compartmental syndrome. An unified concept. Clin Orthop Relat Res 1975;(113):8–14.
137. Frink M, Klaus AK, Kuther G, et al. Long term results of compartment syndrome of the lower limb in polytraumatized patients. Injury 2007;38:607–13.
138. Whitney A, O'Toole RV, Hui E, et al. Do one-time intracompartmental pressure measurements have a high false-positive rate in diagnosing compartment syndrome? J Trauma Acute Care Surg 2014;76:479–83.
139. Janzing HM, Broos PL. Routine monitoring of compartment pressure in patients with tibial fractures: beware of overtreatment!. Injury 2001;32:415–21.
140. Howard JL, Mohtadi NGH, Wiley JP. Evaluation of outcomes in patients following surgical treatment of chronic exertional compartment syndrome in the leg. Clin J Sport Med 2000;10:176–84.
141. Ritenour AE, Dorlac WC, Fang R, et al. Complications after fasciotomy revision and delayed compartment release in combat patients. J Trauma 2008;2(suppl):S153–61. discussion S161–2.
142. Rollins DL, Bernhard VM, Towne JB. Fasciotomy: an appraisal of controversial issues. Arch Surg 1981;11:1474–81.
143. Pearse MF, Harry L, Nanchahal J. Acute compartment syndrome of the leg Fasciotomies must be performed early, but good surgical technique is important. BMJ 2002;325(7364):557–8.
144. Malik AA, Khan WS, Chaudhry A, Ihsan M, Cullen NP. Acute compartment syndrome–a life and limb threatening surgical emergency. J Perioper Pract 2009;19:137–42.
145. Ojike NI, Roberts CS, Giannoudis PV. Compartment syndrome of the thigh: a systematic review. Injury 2010;41:133–6.
146. Wikimedia Commons. File: compartment_syndrome_with_fasciotomy_procedure_02.jpg. Available at: https://commons.wikimedia.org/wiki/File:Compartment_syndrome_with_fasciotomy_procedure_02.jpg. Accessed May 14, 2018.
147. Campbell WC, Canale ST, Beaty JH. Campbell's Operative Orthopaedics. Philadelphia, PA: Mosby/Elsevier; 2008:2737–43.
148. Masquelet AC. Acute compartment syndrome of the leg: pressure measurement and fasciotomy. Orthop Traumatol Surg Res 2010; 96:913–7.
149. Undersea and Hyperbaric Medical Society. Indications for hyperbaric oxygen therapy. Available at: 2003. http://www.uhms.org/Indications/indications.htm. Accessed May 17, 2018.
150. Wattel F, Mathieu D, Nevière R, Bocquillon N. Acute peripheral ischaemia and compartment syndromes: a role for hyperbaric oxygenation. Anaesthesia 1998;53(suppl 2):63–5.

ARTICLE SUMMARY

1. Why is this topic important?

Acute compartment syndrome (ACS) is the result of increased intracompartmental pressure and requires rapid diagnosis and management.

2. What does this review attempt to show?

This review seeks to provide evidence-based recommendations for the emergency department evaluation and management of ACS.

3. What are the key findings?

ACS results from decreased perfusion within a compartment and is associated with a number of risk factors, most commonly trauma to the involved area. The condition may present with a variety of signs and symptoms including pain out of proportion to the injury, paresthesias, pain with passive stretch, tenseness of the compartment, focal motor or sensory deficits, or decreased pulse or capillary refill time. However, history and physical examination are unreliable and cannot exclude the diagnosis. Intracompartmental pressure measurement is needed for diagnosis, though noninvasive means of diagnosis are under study. Treatment involves surgical consultation for emergent fasciotomy, as well as resuscitation and management of complications.

4. How is patient care impacted?

ACS requires rapid diagnosis and management to avoid patient morbidity and mortality. Knowledge of this condition can improve emergency department care of this dangerous disease.