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Acute compartment syndrome of the extremities

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INTRODUCTION — The muscle groups of the human limbs are divided into sections, or compartments, formed by strong, unyielding fascial membranes. Compartment syndrome occurs when increased pressure within a compartment compromises the circulation and function of the tissues within that space [[1,2](#)].

Compartment syndrome may occur acutely, often following trauma, or as a chronic syndrome, seen most often in athletes, that presents as insidious pain. Acute compartment syndrome (ACS) is a surgical emergency.

The pathophysiology, risk factors, diagnosis, and management of ACS in the extremities are reviewed here. Chronic exertional compartment syndrome, fasciotomy techniques and postsurgical care, and abdominal compartment syndrome are discussed separately. (See "[Chronic exertional compartment syndrome](#)" and "[Lower extremity fasciotomy techniques](#)" and "[Abdominal compartment syndrome in adults](#)".)

EPIDEMIOLOGY AND RISK FACTORS — Acute compartment syndrome (ACS) most often develops soon after significant trauma, particularly involving long bone fractures. However, ACS may also occur following minor trauma or from nontraumatic causes. In brief, any condition that decreases the capacity of a compartment or increases the volume of fluid within a compartment raises intracompartmental pressure and places the patient at risk for developing compartment syndrome. Common sites include the leg and forearm [[2,3](#)]. ACS can also occur in the foot, thigh, and gluteal region.

ACS is seen more often in patients under 35 years of age [[4,5](#)]. Young men appear to have the highest incidence, particularly after fractures of the tibial diaphysis and distal radius. This may be explained by the relatively larger muscle mass of men contained within fascial compartments that do not change in size once growth is complete.

Long bone fracture — Fractures account for approximately 75 percent of cases of ACS [[2,6,7](#)]. Risk increases with comminuted fractures [[7](#)]. The tibia is involved most often, with ACS developing in approximately 1 to 10 percent of such fractures [[2,8-10](#)]. The bones of the forearm are involved second most often. Among children, supracondylar fractures are a common cause. (See '[Pediatric considerations](#)' below and "[Evaluation and management of supracondylar fractures in children](#)".)

In one case series of 164 patients with ACS, fracture was the cause in 69 percent of cases [4]. Fractures of the tibial diaphysis (36 percent) and the distal radius (9.8 percent) accounted for the largest share. According to this case series, approximately 20 percent of tibial diaphyseal fractures that lead to ACS are sustained during sports [4]. Soft tissue injury without a fracture was the cause in 23 percent of patients. (See "[Overview of tibial fractures in adults](#)" and "[Distal radius fractures in adults](#)".)

Another case series reported on 113 patients with ACS over an eight year period [11]. The authors found that cases of ACS without a fracture were at significantly greater risk for delayed diagnosis and treatment (ie, fasciotomy). At fasciotomy, 20 percent of patients without a fracture had muscle necrosis requiring debridement, compared with 8 percent of patients with a fracture.

Both closed and open fracture treatment can increase compartment pressure and the risk for ACS [2]. Treatment of fractures should be performed as soon as possible, but clinicians should monitor patients for signs of ACS following these procedures.

Closed fracture reduction decreases the volume and alters the configuration of tissue compartments increasing compartment pressures. According to one prospective observational study involving distal radius fractures, pressures peak immediately after reduction and before a cast is split [12]. After splitting, pressures drop precipitously. A second pressure peak is seen approximately four hours after reduction and dissipates more gradually over several hours. Overly constrictive casts placed at any time during fracture management can lead to ACS. In addition, even non-constricting casts can contribute to ACS if there is significant swelling after the reduction.

Open fracture reduction and fixation also lead to increases in compartment pressures. Pressures during intramedullary nailing of the tibia appear to peak during the procedure and decrease over the following 36 hours [13]. Compartment pressures following volar plating of distal radius fractures appear to diminish substantially during the 24 hour period following surgery [14]. The risk of ACS increases with the duration of the procedure.

Trauma without fracture — Other forms of trauma not involving a fracture can predispose a patient to ACS. Possible causes include forceful direct trauma to a tissue compartment (eg, crush injury), severe thermal burns, overly constrictive bandages (usually circumferential), penetrating trauma, injury to vascular structures in the extremities, and in some cases, even minor injuries. Patients with a bleeding diathesis and those who continue to use an injured limb are at increased risk.

Thermal injuries, particularly full-thickness burns, can cause ACS secondary to tissue constriction, eschar, and edema [15]. The large fluid shifts associated with major burns also contribute. In such cases abdominal compartment syndrome is a major concern. (See "[Emergency care of moderate and severe thermal burns in adults](#)" and "[Abdominal compartment syndrome in adults](#)".)

Unconscious or obtunded patients with prolonged limb compression, either during surgery or due to recreational drug abuse, can develop ACS secondary to soft tissue injury and swelling [16,17]. Victims of penetrating extremity trauma are also susceptible to developing ACS [18]. Patients who develop rhabdomyolysis for any reason are at increased risk for ACS. (See "[Clinical manifestations and diagnosis of rhabdomyolysis](#)".)

Vascular, particularly arterial, injury is an important cause of ACS [15,19]. Arterial bleeding increases compartment pressures and muscle deprived of arterial blood flow becomes ischemic and prone to reperfusion injury, which in turn causes swelling and a further increase in compartment pressures. In addition, muscle that has sustained a previous ischemic insult is less tolerant of increased tissue pressure [20]. Venous injury (eg, traumatic deep vein harvest, direct vein trauma) is also associated with an increased risk of ACS [21,22].

Reports of minor trauma or repetitive trauma leading to ACS include cases involving the peroneal compartment following a minor ankle inversion injury [23-25], compartments of the foot after an inversion injury [26], and the

upper extremity after it was struck by a baseball [27]. Intramuscular hemorrhage following minor trauma in patients taking anticoagulants also increases the risk for developing ACS. Some researchers believe that relatively larger muscle volumes may be a risk factor for ACS, which explains why men are generally more susceptible [28].

Nontraumatic causes — ACS from nontraumatic causes occurs less frequently. It may be related to ischemia-reperfusion injury, thrombosis, bleeding disorders, vascular disease, nephrotic syndrome (or other conditions that decrease serum osmolarity), certain animal envenomations and bites, extravasation of IV fluids, injection of recreational drugs, and prolonged limb compression (eg, following severe drug or alcohol intoxication or poor positioning during surgery) [2,29-32].

Revascularization procedures and treatments, such as extremity bypass surgery, embolectomy, and thrombolysis, increase the risk for ACS [33]. This phenomenon is known as postischemic compartment syndrome and is due to tissue swelling from reperfusion. The syndrome can occur from a few hours following the procedure up to several days later [34]. Residual effects from anaesthesia and postoperative sedation can make early detection of ACS more difficult immediately after surgery.

Anticoagulation following surgery, such as prophylaxis against deep vein thrombosis, may contribute to ACS [35]. In addition, a number of surgical procedures involving the leg (eg, saphenous vein harvest) have been associated with ACS in case reports [36-38]. Symptoms can mimic postoperative pain making the diagnosis difficult. Iatrogenic injury of arteries or veins in anticoagulated patients is another potential cause [39]. The use of intraaortic balloon pumps has been associated with ACS [40].

Drug abusers can develop ACS following intravenous or inadvertent intraarterial injection of drugs [41,42].

ACS may arise as a complication of underlying disease. As examples, there are case reports of ACS in a 13-year-old boy with a large osteochondroma arising from the fibula that compromised blood flow in the lateral and posterior leg compartments [43] and in other children following episodes of myositis and fasciitis [30]. Group A streptococcus infections of muscle can be complicated by ACS [44]. There are case reports of patients who developed ACS from muscle ischemia secondary to McArdle disease, spontaneous bleeding due to acquired factor VIII inhibition, toxic shock and infectious pyomyositis, and prolonged exposure to subatmospheric pressure [45,46]. (See "[Invasive group A streptococcal infection and toxic shock syndrome: Epidemiology, clinical manifestations, and diagnosis](#)" and "[Necrotizing soft tissue infections](#)".)

Intramuscular hemorrhage in patients treated chronically with anticoagulants can progress to ACS [47-49]. In rare cases, phlegmasia cerulea dolens has been associated with ACS. (See "[Clinical presentation and diagnosis of the nonpregnant adult with suspected deep vein thrombosis of the lower extremity](#)".)

Among athletes there are reports of ACS developing in association with rhabdomyolysis from strenuous physical activity and possibly from dietary supplements [50-52].

PATHOPHYSIOLOGY — Multiple explanations for the complex pathophysiology of acute compartment syndrome (ACS) exist [53]. In all cases, the final common pathway is cellular anoxia [15]. A prerequisite for the development of increased compartment pressure is a fascial structure that prevents adequate expansion of tissue volume to compensate for an increase in fluid.

Perhaps the most widely believed hypothesis for the pathophysiology of ACS is the arteriovenous pressure gradient theory [2]. The premise of this theory is that ischemia begins when local blood flow cannot meet the metabolic demands of surrounding tissue. As compartment pressure rises, venous outflow is reduced and venous pressure rises, leading to a decrease in the arteriovenous pressure gradient. Ultimately arteriolar pressure is insufficient to overcome compartment pressure and blood is shunted away from intracompartmental tissues. Arterioles collapse when tissue pressure exceeds end-arteriolar pressure [54]. Inadequate venous

drainage results in tissue edema and a rise in interstitial pressure. Lymphatic drainage may compensate in part initially, but is soon overwhelmed.

Compartment pressures capable of compromising perfusion develop when they rise to within 10 to 30 mmHg of diastolic pressure; muscle oxygenation decreases as tissue pressure approaches mean arterial pressure [15,55,56]. Therefore, ACS develops based upon both compartment and systemic blood pressures. As an example, compared to a normotensive patient, a patient with hypotension is less likely to tolerate any given increase in tissue pressure.

ANATOMIC COMPARTMENTS AND RELATED CLINICAL SIGNS — Acute compartment syndrome (ACS) can occur in any distinct anatomic compartment bound by unyielding fascial membranes. ACS has been described in the upper extremity (primarily the forearm), lower extremity (primarily the leg), hand, foot, buttock, abdomen, thorax, and orbit (table 1) [26,53,57-59]. Abdominal compartment syndrome is discussed separately. The compartments of the extremities are discussed below. (See "[Abdominal compartment syndrome in adults](#)".)

The lower leg is a common site for ACS and is comprised of four compartments (figure 1). These compartments are the anterior, lateral, deep posterior, and superficial posterior. Below are included descriptions of possible neurologic findings associated with ACS, but it is important to note that nerve injuries proximal to the affected compartment may also account for such deficits.

The anterior compartment of the leg is the most common site for ACS. It contains the four extensor muscles of the foot, the anterior tibial artery, and the deep peroneal nerve. Signs of ACS affecting the anterior compartment include loss of sensation between the first (ie, great) and second toes and weakness of foot dorsiflexion. Late sequelae include foot drop, claw foot, and deep peroneal nerve dysfunction. (See '[Clinical features](#)' below.)

The lateral compartment of the leg contains the muscles responsible for foot eversion and some degree of plantar flexion (ie, peroneus brevis, peroneus longus), the superficial peroneal nerve, and the proximal portion of the deep peroneal nerve. Increased pressure in the lateral compartment may produce a deep peroneal nerve deficit, which manifests as weakness in dorsiflexion and inversion of the foot and sensory loss in the web space between the great toe and the adjacent toe. The superficial peroneal nerve also travels through this compartment and supplies sensation to the lower leg and the dorsum of the foot.

The deep posterior compartment contains muscles that aid in foot plantar flexion, as well as the posterior tibial artery, peroneal artery, and the tibial nerve. Increased pressure in this compartment may cause plantar hypesthesia, weakness of toe flexion, and pain with passive extension of the toes.

The superficial posterior compartment contains the major muscles of plantar flexion (ie, gastrocnemius, soleus). No major arteries or nerves travel in this compartment. Therefore, of the four leg compartments, the superficial posterior is least likely to develop ACS. Pain and a palpably tense and tender compartment suggest the diagnosis.

The forearm has four compartments: the deep and superficial volar compartments, the dorsal compartment, and the lateral compartment (figure 2). The volar compartment contains the digital flexors and the dorsal compartment contains the digital extensors. The volar compartments are at highest risk for developing ACS following trauma. The most frequent injuries associated with ACS in the forearm are supracondylar humerus fractures in children and distal radius fractures in adults [9,55,60,61]. (See '[Pediatric considerations](#)' below.)

The deep volar compartment usually develops the highest interstitial pressures with ACS of the forearm and thus the flexor digitorum profundus (responsible for distal interphalangeal joint flexion) and the flexor pollicis longus (responsible for interphalangeal joint flexion of the thumb) muscles are most often affected [60]. The flexor digitorum superficialis (responsible for proximal interphalangeal joint flexion) and pronator teres are affected less

often, while the wrist flexors and extensors and the brachioradialis are least likely to be involved. (See ["Finger and thumb anatomy"](#).)

The arm has two relatively large compartments, the anterior and posterior. These compartments tolerate relatively large fluid volumes, thereby limiting the rise in compartment pressure and minimizing the risk of ACS. The anterior compartment contains the elbow flexor muscles (biceps brachii, brachialis) and the ulnar and median nerves. The posterior compartment contains the elbow extensor muscles (triceps) and the radial nerve.

ACS rarely develops in the thigh, but may do so following major trauma [62]. The thigh has three large compartments — the anterior, posterior, and medial ([figure 3](#)):

The anterior compartment contains the knee extensors ([figure 3](#)). ACS of the anterior compartment may manifest as pain with passive knee flexion; inability to extend the knee; or sensory deficits involving the lateral, anterior, or medial thigh (cutaneous branches of femoral nerve) or involving the medial calf and foot (saphenous nerve).

The posterior compartment contains the knee flexors. ACS of the posterior compartment may manifest as pain with passive knee extension; inability to flex the knee, plantar flex the ankle, or dorsiflex the great toe; or sensory deficits involving either the dorsum or plantar surface of the foot or the great toe web space (peroneal nerve).

The medial compartment contains the hip adductors. ACS of the medial compartment may manifest as pain with passive hip abduction; inability to adduct the hip; or sensory deficits at the proximal medial thigh (obturator nerve).

The compartments of the foot are described separately ([figure 4](#)). Compartments in the hand and gluteal region are also susceptible to ACS, but such cases are uncommon. (See ["Lower extremity fasciotomy techniques"](#), [section on 'Foot'](#) and ["Lower extremity fasciotomy techniques"](#), [section on 'Buttock'](#).)

CLINICAL FEATURES — The signs and symptoms of acute compartment syndrome (ACS) generally appear in a stepwise fashion, although the timing of the appearance of specific findings varies [2,15,63,64]. Important clues to the development of ACS include rapid progression of symptoms and signs over a few hours and the presence of multiple findings consistent with the diagnosis in a patient at risk. Therefore, serial evaluation is of great importance in patients at risk for ACS. However, the limitations of the physical examination for identifying ACS must be emphasized; **any** tense painful muscle compartment represents a possible ACS. When the diagnosis of ACS is suspected on clinical grounds, it is often confirmed by measuring compartment pressures. As part of the initial assessment, a careful and complete neurologic examination of the extremity should be performed and documented. (See ['Epidemiology and risk factors'](#) above and ['Measurement of compartment pressures'](#) below.)

Symptoms of ACS can include the following:

- Pain out of proportion to apparent injury (early and common finding)
- Persistent deep ache or burning pain
- Paresthesias (onset within approximately 30 minutes to two hours of ACS; suggests ischemic nerve dysfunction)

Examination findings suggestive of ACS include the following:

- Pain with passive stretch of muscles in the affected compartment (early finding)
- Tense compartment with a firm "wood-like" feeling
- Pallor from vascular insufficiency (uncommon)

- Diminished sensation
- Muscle weakness (onset within approximately two to four hours of ACS)
- Paralysis (late finding)

The classic findings associated with arterial insufficiency are often described as signs of ACS, but this is inaccurate [65]. Of the five classic signs of arterial insufficiency (five P's: pain, pallor, pulselessness, paresthesias, poikilothermia [cold skin temperature]), only pain is commonly associated with compartment syndrome, particularly in its early stages. Paresthesias may also occur.

Many authors describe the importance of the common clinical symptoms and signs described above in diagnosing ACS. However, data supporting the accuracy of these findings is limited, and each may be unreliable in some circumstances [2,15]. A systematic review of four studies of ACS associated with tibial fracture found common clinical findings (eg, pain, paresthesia, pain with passive movement) to have poor sensitivity and specificity [66]. Therefore, compartment pressure measurements are often important for diagnosis.

Pain "out of proportion to injury" is often described as an early and sensitive sign of ACS [9,15,63]. Nevertheless, pain can be nonspecific [2]. Most patients at risk for ACS have sustained trauma, and a fracture or an injury to a nerve or soft tissue may be the source of pain. Severe injuries can also distract patients from pain that stems from an ACS. Furthermore, young children, obtunded or critically ill patients, and those emerging from general anesthesia or who were treated with nerve blocks cannot clearly convey the presence of severe or increasing pain.

Pain in response to passive stretching of muscles within the affected compartment is widely described as a sensitive early sign of ACS, but it too may be unreliable in some patients [67]. Palpable tenseness is a crude indicator of increased compartment pressure and cannot be used to assess the deep posterior compartment of the leg. A study using fresh cadavers found that palpation of leg compartments was neither sensitive nor specific for detecting substantial elevations in compartment pressures [68].

The presence of neurologic symptoms, such as paresthesias can be confusing because peripheral nerve injury may result directly from trauma or from an ACS [15]. Sensory deficits typically precede motor deficits and manifest distal to the involved compartment. Some feel that the loss of two-point discrimination is more typical in ACS than in raised intracompartmental pressure alone without ACS [21,69]. Again, the limited accuracy of the physical examination for identifying ACS should be emphasized here. In the presence of a painful tense muscle compartment, obtaining immediate surgical consultation and possibly compartment pressure measurements is far more important than discerning degrees of relative weakness or sensory loss.

Muscle weakness can be difficult to assess in the traumatized patient, patients receiving sedating medications, and patients with altered mental status, and may be attributable to pain, fracture, direct soft tissue injury, peripheral or central nerve injury, or ACS. Of note, muscle weakness or paresis is a late finding of ACS and suggests permanent muscle damage.

Several common misconceptions exist pertaining to the clinical diagnosis of ACS [2,65]. Clinicians should be aware that ACS can occur in the presence of open fractures as these may **not** necessarily decompress elevated compartment pressures [8]. Moreover, ACS can occur without a fracture (or a crush injury). The diagnosis is often delayed because clinicians fail to consider ACS in patients without a fracture. Arterial pulses and normal capillary refill can persist despite the presence of a prolonged, severe ACS. Pulse oximetry is an insensitive instrument for diagnosis and should not be relied upon [70,71].

Left untreated, ACS can result in muscle contracture, sensory deficits, paralysis, infection, fracture nonunion, and possibly limb amputation [4]. Rhabdomyolysis may occur with muscle ischemia, resulting in myoglobinuria and

possible renal failure necessitating dialysis. (See ["Clinical manifestations and diagnosis of rhabdomyolysis"](#) and ["Clinical features and diagnosis of heme pigment-induced acute kidney injury"](#), section on 'Clinical manifestations'.)

LABORATORY STUDIES — Acute compartment syndrome (ACS) is diagnosed on the basis of clinical findings and in many cases the measurement of compartment pressures. Laboratory values are not used for diagnosis. If the diagnosis is suspected, surgical consultation, possibly including the measurement of compartment pressures, should not be delayed in order to obtain a laboratory result.

Nevertheless, as ACS develops and muscle breakdown ensues, lab abnormalities develop, including elevations in the serum creatine kinase (CK) [72], (conversely, CK rises in patients with rhabdomyolysis, which may go on to cause ACS). Myoglobinuria can develop within four hours of the onset of ACS. (See ["Clinical manifestations and diagnosis of rhabdomyolysis"](#) and ["Trauma without fracture"](#) above.)

MEASUREMENT OF COMPARTMENT PRESSURES

Indications and general approach — Compartment pressure measurements are an important adjunct in the diagnosis of acute compartment syndrome (ACS). Whenever possible, the surgeon responsible for determining whether to perform fasciotomies should also determine the need for compartment pressure measurements and obtain them. In remote areas and hospitals with limited surgical coverage, this approach may not always be possible but it is preferable. Compartment pressures are not required for diagnosis, and the surgeon may opt to take the patient to the operating room on the basis of the history and examination findings alone without obtaining measurements [73]. Conversely, the surgeon may measure compartment pressures in an effort to avoid unnecessary fasciotomies in patients with suggestive findings. A pressure in the normal range allows such patients to be observed, often using serial measurements to guide care. (See ["Interpretation of measurements"](#) below.)

Compartment pressures can be measured as needed whenever a clinician suspects ACS based upon the patient's risk factors and clinical findings, namely a painful tense muscle compartment. Measuring compartment pressures in patients at risk for developing ACS entails no major complications, while not doing so may lead to a missed diagnosis and permanent deformity or dysfunction of the extremity [69]. Compartment pressure testing may be unnecessary if the diagnosis is clinically obvious, or conversely repeat measurements may be necessary in some patients as clinical conditions change (eg, worsening hypotension). Clinical context is essential for interpreting pressure measurements correctly. (See ["Clinical features"](#) above and ["Interpretation of measurements"](#) below.)

In some cases, it can be especially difficult to determine whether the patient is developing signs of ACS. Young children, obtunded or critically ill patients, and those emerging from general anaesthesia can neither cooperate with a physical examination nor can they clearly convey the presence of severe or increasing pain. Critically ill trauma patients appear to be at particularly high risk and should be monitored closely [74]. If ACS is suspected in such patients on the basis of risk factors and clinical findings, most authors agree that compartment pressure measurements should be obtained [75]. Measurement may also be needed in patients with regional or epidural nerve blocks or equivocal clinical findings who are at risk for ACS.

Direct measurement techniques — Although multiple techniques for direct compartment pressure measurement have been described [55,76-78], three methods have been used most frequently: a handheld manometer (eg, Stryker device), a simple needle manometer system, and the wick or slit catheter technique. Both manometer methods involve injecting a small quantity of saline into a closed compartment and measuring the resistance from tissue pressure. The hand-held manometer method is used most often because it is portable, simple, and relatively accurate [79]. The slit catheter technique involves inserting a catheter into the compartment and monitoring the pressure via a transducer connected to a pressure amplifier and recorder [80].

According to an unblinded observational study, these methods produce similar results but may not be reliable in all settings [81]. The authors of this study emphasized the importance of interpreting measurements in light of clinical findings. A laboratory study found that simple 18-gauge needles, side-ported needles, and slit catheters all provide equivalent measurements of intracompartmental pressure [82].

A retrospective study of 1184 patients investigating the use of continuous intracompartmental pressure monitoring for the diagnosis of ACS reported an estimated sensitivity of 94 percent, specificity of 98 percent, and negative predictive value of 99 percent, using intraoperative clinical findings as the gold standard [83].

Eighteen-gauge needles can be attached easily to an arterial pressure monitor to measure compartment pressures. This technique can prevent potential delays in diagnosis if for example a Stryker hand-held monitor cannot be located

Whichever method is used to measure compartment pressures, accuracy depends upon proper calibration of the measuring device and placement of the needle or pressure sensor close to the site of fracture or injury. Accuracy decreases the further the measurement is taken from the fracture site [84]. Compartment pressures are not uniform but exist along a gradient, with pressures highest near the site of trauma. The catheter or device tip should lie within approximately 5 cm of the level of the fracture to obtain the peak measure of intracompartmental pressure. However, tip placement immediately adjacent to the fracture itself may result in inaccurately elevated pressure readings due to the effects of fracture hematoma. In addition, the level of the transducer should be secured at the same level as the compartment being measured (not angled above or below) because of changes in the reading with height [85].

Interpretation of measurements — The normal pressure of a tissue compartment falls between 0 and 8 mmHg [86]. Clinical findings associated with ACS generally correlate with the degree to which tissue pressure within the affected compartment approaches systemic blood pressures:

- Capillary blood flow becomes compromised when tissue pressure increases to within 25 to 30 mmHg of mean arterial pressure [87].
- Pain may develop as tissue pressures reach between 20 and 30 mmHg.
- Ischemia occurs when tissue pressures approach diastolic pressure [88,89].

These values are approximations; the pressure necessary for injury varies depending upon clinical circumstance. As examples, higher compartment pressures may be necessary before injury occurs to peripheral nerves in patients with systemic hypertension [90], while ACS may develop at lower pressures in those with hypotension or peripheral vascular disease [65,91]. Traumatized tissue has increased metabolic demands and is therefore more susceptible to further injury from ischemia secondary to ACS [92]. In one case series involving 18 patients with confirmed ACS of the leg, preoperative tissue measurements ranged from 28 mmHg to 47 mmHg [93].

Many surgeons involved in trauma care use a threshold based upon the difference between systemic blood pressures and compartment pressures to confirm the presence of ACS. These experts believe that if the difference between the diastolic or mean arterial pressure and the compartment pressure falls below a specific value, perfusion pressure becomes compromised and ACS can develop. In addition, many believe the use of absolute measurements leads to unnecessary fasciotomies, or in some cases failure to perform needed fasciotomies [94,95].

We concur with this approach and suggest that a difference between the diastolic blood pressure and the compartment pressure (delta pressure) of 30 mmHg or less be used as the threshold for diagnosing ACS [94-96]. The critical delta pressure may be increased in traumatized or ischemic muscle [85]. The delta pressure is

found by subtracting the compartment pressure from the diastolic pressure. Many clinicians use the delta pressure of 30 mmHg to determine the need for fasciotomy, while others use a difference of 20 mmHg [15,84].

In summary:

- ACS delta pressure = diastolic blood pressure – measured compartment pressure
- ACS delta pressure <20 to 30 mmHg indicates need for fasciotomy (we use <30 mmHg)

Several studies support the use of delta pressure to diagnose ACS:

- In a prospective study of 116 patients with diaphyseal fractures of the tibia, the authors performed continuous blood pressure and compartment pressure monitoring to assess for ACS [94]. A delta pressure of 30 mmHg was used as the diagnostic threshold. Three patients were diagnosed with ACS immediately, one patient during the first 12 hours of treatment, and two patients during the second 12 hours. At six month follow-up, no patient had sequelae of ACS. Of note, 37 patients had isolated compartment pressure measurements over 40 mmHg during the first 24 hours.
- Another observational study evaluated 101 patients with tibial fractures using continuous compartment pressure monitoring [95]. Although 41 patients had compartment pressures above 30 mmHg continuously for six hours, no patient whose delta pressure remained above 30 mmHg developed ACS.
- In a similar observational study of 25 consecutive patients with closed tibia fractures continuously monitored for the development of ACS, 75 percent of the patients who did not meet the criteria for fasciotomy (delta pressure of 20 mmHg) demonstrated absolute tissue pressures above 30 mmHg but none developed ACS [84].

The authors of these studies emphasize the **importance of following trends in compartment and delta pressures**. When compartment pressures are trending downward, it is often safe not to intervene emergently, provided the delta pressure is also improving.

Once a consistent trend of decreasing compartment pressures becomes apparent over a minimum of six hours of observation, monitoring may be discontinued. Conversely, a single normal compartment pressure reading, which may be performed early in the course of the disease, does **not** rule out ACS. Serial or continuous measurements are important when patient risk or clinical suspicion is high.

Studies of the test characteristics of compartment pressure measurements are limited, and the true sensitivity and specificity of the test are not known [73]. In addition, most studies have been performed in patients following surgical treatment of a tibial fracture, and it is difficult to extrapolate these results to patients with other injuries or conditions. It bears emphasis that pressure measurements must be interpreted in the context of the history and examination findings, preferably by the surgeon charged with deciding whether to perform fasciotomies. Isolated measurements may be misleading, and a single abnormal measurement does not necessarily indicate ACS [97], particularly in patients without a history and examination findings consistent with the diagnosis.

Some surgeons continue to use absolute compartment pressure measurements to diagnose ACS. Traditional recommendations for decompression include absolute pressure readings above 30 mmHg [76] or above 45 mmHg [1]. We do not recommend this approach for the reasons alluded to above (eg, increased rate of unnecessary fasciotomies).

Continuous invasive monitoring of alert patients who are able to convey their symptoms and who are closely observed for clinical findings of ACS is unlikely to improve outcomes. This was demonstrated in a trial of 200 consecutive patients with tibia fractures randomized to continuous invasive compartment pressure monitoring or clinical observation [98]. No cases of ACS were missed in the clinical observation group and at the six month

follow-up there was no significant difference in outcome between groups. An observational study noted similar findings [99].

Investigational techniques — Several alternative noninvasive methods of measuring compartment pressures are under study [63]. Potential approaches include ultrasound with a pulsed phase-locked loop, laser Doppler flowmetry, and near-infrared spectroscopy [100].

DIAGNOSIS — Acute compartment syndrome (ACS) of an extremity is diagnosed on the basis of the history, examination findings, and often the measurement of compartment pressures, although this is not required. ACS most often develops soon after significant trauma, particularly involving long bone fractures. Other possible causes include crush injury, severe thermal burns, penetrating trauma, injury to vascular structures, and less often a number of nontraumatic conditions. Important clues to the development of ACS include rapid progression of symptoms and signs over a few hours and the presence of multiple findings consistent with the diagnosis in a patient at risk. Notable findings include pain out of proportion to apparent injury, pain with passive stretch of muscles in the affected compartment, and a tense compartment with a firm "wood-like" feeling. In patients with suggestive clinical features, a difference between the diastolic blood pressure and the compartment pressure (delta pressure) of 30 mmHg or less strongly suggests the diagnosis of ACS.

MANAGEMENT — Perhaps the most important aspect of diagnosis is to maintain a high index of suspicion among patients at risk for acute compartment syndrome (ACS) [4]. Frequent serial examinations are important in such patients. Immediate surgical consultation should be obtained if ACS is suspected. In remote areas and hospitals with limited surgical coverage, the patient should be transferred immediately to a hospital where compartment pressures can be measured and fasciotomies performed. With early diagnosis and appropriate treatment, the complications of ACS can be prevented and normal function of the extremity maintained. (See '[Epidemiology and risk factors](#)' above.)

Immediate management of suspected ACS includes relieving all external pressure on the compartment. Any dressing, splint, cast, or other restrictive covering should be removed. The limb should neither be elevated nor placed in a dependent position. Placing the limb level with the heart helps to avoid reductions in arterial inflow and increases in compartment pressures from dependent swelling, both of which can exacerbate limb ischemia [101].

Analgesics should be given and supplementary oxygen provided. Hypotension reduces perfusion, exacerbating tissue injury, and should be treated with boluses of intravenous isotonic saline.

Fasciotomy to fully decompress all involved compartments is the definitive treatment for ACS in the great majority of cases ([figure 5A-B](#)). Delays in performing fasciotomy increase morbidity, including the need for amputation [102,103]. The performance of fasciotomy procedures is described separately. (See "[Lower extremity fasciotomy techniques](#)", [section on 'Outcomes'](#).)

Fasciotomy is occasionally not indicated or may not be necessary. As an example, it should be avoided when the muscle is already dead. Fasciotomy in such instances provides no benefit and can increase the risk of infection. Definitive treatment for such injuries often involves amputation. In specific, low-risk circumstances, fasciotomy **may** not be necessary, but this decision must be made by the treating surgeon [104,105]. (See "[Lower extremity fasciotomy techniques](#)", [section on 'Contraindications'](#).)

Hyperbaric oxygen has been described as adjunct treatment for ACS [71,106,107]. Further research is needed to determine the appropriate role of hyperbaric oxygen therapy. (See "[Hyperbaric oxygen therapy](#)".)

PEDIATRIC CONSIDERATIONS — Although the criteria for diagnosis are the same as in older patients, making the diagnosis of acute compartment syndrome (ACS) in young children can be challenging. Trauma is the cause in most cases, but as in adults ACS can develop from a number of conditions [30]. As described above for

adults, compartment pressures should be measured when a definitive diagnosis is needed, such as in children with a depressed level of consciousness, possible nerve injury, or those unable to cooperate and at high risk. (See ['Measurement of compartment pressures'](#) above.)

A retrospective review of 42 skeletally immature children with ACS, sustained primarily in association with leg fractures following a motor vehicle collision, reported that most patients had good outcomes despite a relatively long period between injury and fasciotomy (average 20.5 hours) [108]. The authors emphasize the importance of vigilant monitoring in this age group following injury.

Supracondylar fractures occur primarily in children and may be complicated by ACS [109]. If an ipsilateral forearm fracture is also present, the risk of developing ACS increases. Children with supracondylar fractures should be closely monitored and compartment pressures measured if there is any doubt concerning the possibility of ACS. (See ["Evaluation and management of supracondylar fractures in children"](#).)

DISEASE COURSE AND PROGNOSIS — Early diagnosis and appropriate treatment of acute compartment syndrome (ACS) generally produce good functional and cosmetic results. However, mortality rates in patients with ACS requiring fasciotomy may reach as high as 15 percent, and can be much higher in patients with severe trauma. (See ["Patient management following extremity fasciotomy"](#), [section on 'Mortality'](#).)

Morbidity following fasciotomy may be significant. Skin grafts may be needed for incisions and muscle weakness can persist in the affected limb. (See ["Lower extremity fasciotomy techniques"](#), [section on 'Outcomes'](#).)

Current understanding of how long muscles can tolerate ischemia is based primarily upon extrapolations from experimental models. According to these models, muscles can tolerate up to three hours of complete warm ischemia before the onset of necrosis.

The most important determinant of a poor outcome from ACS is a delayed or missed diagnosis. ACS can result in muscle contracture, sensory deficits, paralysis, infection, fracture nonunion, and possibly limb amputation. (See ["Patient management following extremity fasciotomy"](#), [section on 'Complications'](#).)

Rhabdomyolysis may occur with muscle ischemia, resulting in myoglobinuria and possible renal failure necessitating dialysis. (See ["Clinical manifestations and diagnosis of rhabdomyolysis"](#) and ["Clinical features and diagnosis of heme pigment-induced acute kidney injury"](#), [section on 'Clinical manifestations'](#).)

SOCIETY GUIDELINE LINKS — Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See ["Society guideline links: Extremity compartment syndrome"](#).)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topics (see ["Patient education: Rhabdomyolysis \(The Basics\)"](#) and ["Patient education: Acute compartment syndrome \(The Basics\)"](#))

SUMMARY AND RECOMMENDATIONS

- Acute compartment syndrome (ACS) occurs when increased pressure within a compartment bounded by unyielding fascial membranes compromises the circulation and function of the tissues within that space ([figure 2](#)). ACS is a surgical emergency. (See ['Pathophysiology'](#) above.)
- ACS most often develops soon after significant trauma, particularly involving long bone fractures of the lower leg or forearm. ACS may also occur following penetrating or minor trauma, or from nontraumatic causes, such as ischemia-reperfusion injury, coagulopathy, certain animal envenomations and bites, extravasation of IV fluids, injection of recreational drugs, and prolonged limb compression. (See ['Epidemiology and risk factors'](#) above.)
- The accuracy of the physical examination for diagnosing ACS is limited. Early symptoms of ACS include progressive pain out of proportion to the injury; signs include tense swollen compartments and pain with passive stretching of muscles within the affected compartment. Important clues to the development of ACS include rapid progression of symptoms and signs over a few hours and the presence of multiple findings consistent with the diagnosis in a patient at risk. Close observation and serial examinations in patients at risk for ACS are of great importance. Motor deficits are **late** findings associated with irreversible muscle and nerve damage. (See ['Clinical features'](#) above.)
- Immediate surgical consultation should be obtained whenever ACS is suspected based upon the patient's risk factors and clinical findings. Whenever possible, the surgeon should determine the need for measuring compartment pressures, which can aid diagnosis. A single normal compartment pressure reading, which may be performed early in the course of the disease, does **not** rule out ACS. Serial or continuous measurements are important when patient risk is moderate to high or clinical suspicion persists. (See ['Measurement of compartment pressures'](#) above.)
- The normal pressure of a tissue compartment falls between 0 and 8 mmHg. Signs of ACS develop as tissue pressure rises and approaches systemic pressure. However, the pressure necessary for injury varies. Higher pressures may be necessary before injury occurs to peripheral nerves in patients with systemic hypertension, while ACS may develop at lower pressures in those with hypotension or peripheral vascular disease. (See ['Interpretation of measurements'](#) above.)
- When interpreting compartment pressure measurements in patients with clinical findings suggestive of ACS, we suggest using a difference between the diastolic blood pressure and the compartment pressure of 30 mmHg or less as the threshold for an elevated compartment pressure. (See ['Interpretation of measurements'](#) above.)
- Immediate management of suspected ACS includes relieving all external pressure on the compartment. Any dressing, splint, cast, or other restrictive covering should be removed. The limb should be kept level with the torso, **not** elevated or lowered. Analgesics should be given and supplementary oxygen provided. Hypotension reduces perfusion and should be treated with intravenous boluses of isotonic saline. (See ['Management'](#) above.)
- Fasciotomy to fully decompress all involved compartments is the definitive treatment for ACS in the great majority of cases. Delays in performing fasciotomy increase morbidity. (See ['Management'](#) above.)

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