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# Acute compartment syndrome: An orthopedic emergency

***Abstract: Acute compartment syndrome (ACS) is a true orthopedic emergency. NPs in all settings should be able to recognize ACS to ensure swift surgical intervention is not delayed. This article highlights a systematic approach to exam and diagnosis.***

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Acute compartment syndrome (ACS) occurs when increased pressure within a closed fascial space causes decreased perfusion to the tissues within the space. Orthopedic injury, specifically fracture of a long bone, is the most common cause of this critical rise in compartment pressure.[1-4] Without immediate surgical intervention, ACS can have disastrous consequences.[5] In addition to poor patient outcomes, delayed diagnosis also has been associated with increased healthcare costs and high rates of medical litigation.[4] This makes early diagnosis of ACS imperative for NPs caring for orthopedic patients in both inpatient and outpatient settings. This article will review the pathophysiology, clinical presentation, diagnosis, and treatment of ACS, highlighting a systematic approach to exam and diagnosis. Although this article focuses on ACS, it is important to note that compartment syndrome can also stem from a chronic exertional mechanism. In chronic exertional compartment syndrome, exercise increases blood flow to active muscles, causing their volume to expand by as much as 20%. This is most commonly seen in the lower extremities of young athletes, especially runners.[6]

**Keywords: "6 Ps," acute compartment syndrome, compartment syndrome, fasciotomy, tibial fracture**

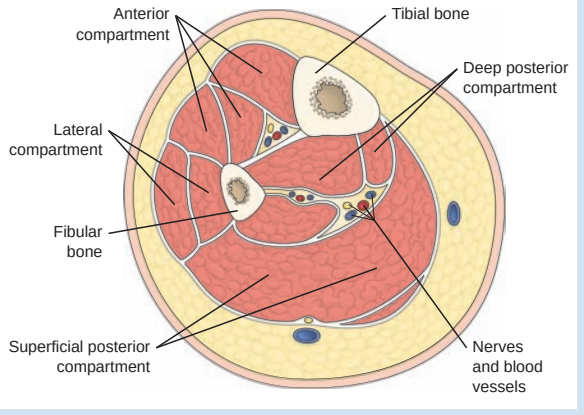
## Pathophysiology

A prerequisite for developing any type of compartment syndrome within a space is the presence of an unyielding fascial membrane. Fascia acts as a container for the structures within the extremities, which makes the arms and legs particularly susceptible to compartment syndrome. There are different explanations for the pathophysiology causing ACS, the most widely accepted being the arteriovenous pressure gradient theory. As pressure within a defined fascial space rises, venous outflow is reduced, arterioles collapse when arterial pressure is not sufficient to overcome compartment pressure, and local hypoxia results.[3,5] Simply, venous blood cannot get out and oxygenated arterial blood cannot get in, causing hypoxia and tissue death. This cell death initiates a vicious cycle of increased capillary permeability, which leads to further increases in swelling.[4]

***Overall, two-thirds of all ACS instances are caused by fractures, the most common being tibial fractures.***

Systemic manifestations may be seen in ACS. Muscle necrosis releases myoglobin into circulation. Myoglobin can cause acute kidney injury and kidney failure if not appropriately managed. Associated rhabdomyolysis also causes ischemic cells to flood into circulation, causing cytokine release, and ultimately a further increase in swelling.[4] This potentiates the domino effect of ACS.

### Compartments in the lower leg

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## Causes and risk factors

ACS can be caused by both traumatic and nontraumatic insults. Examples of traumatic causes are fracture, crush injuries, penetrating injuries, burns, and snake bites. Some nontraumatic causes are I.V. infiltration/extravasation, ischemia-reperfusion injury, thrombosis, and hemorrhage.[3] Patients receiving anticoagulants are especially at risk for ACS related to hemorrhage.[3,4,7] In orthopedics, tibial fracture and distal radius fracture are the fractures most commonly associated with ACS.[1-3]

Overall, two-thirds of all ACS instances are caused by fractures, the most common being tibial fractures.[3,4,8] The anterior and deep posterior compartments of the lower leg are directly adjacent to the tibia, so tibial trauma can cause soft-tissue trauma and swelling in these spaces. This makes the anterior and deep posterior compartments the most commonly affected by ACS.[3,8] The volar compartment of the forearm is the most common site for ACS in the arm, which is frequently caused by distal radial fracture.[9] It is important to identify patients with high-risk injuries and to monitor vigilantly for ACS.

Although fractures are the most common cause of ACS, there are nontraumatic orthopedic causes that are also worth noting. The first is circumferential dressings. Casts, splints, and wraps are common treatments for orthopedic injuries, but there is an associated risk with these generally benign treatments. When a circumferential extremity dressing becomes overly tight because of swelling, it acts like the unyielding membrane of the fascial compartments, restricting venous outflow and preventing arterial inflow, and thus causing ACS.[3,5] According to one study, applying a cast to a noninjured leg increases the pressures in the anterior and deep posterior compartments up to sevenfold.[10] Valving, or splitting casts, on one or both sides, helps reduce this pressure by up to 50%.[5]

Another nontraumatic cause of compartment syndrome is intraoperative patient positioning, which is particularly important in orthopedic surgery patients.

**Exam findings: Lower leg compartments**[5,14]

| Compartment | Contents | Exam finding |
|---|---|---|
| Anterior | • Four extensor muscles of the foot (tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus tertius)<br>• Anterior tibial artery<br>• Deep peroneal nerve | • Decreased sensation between great and second toes<br>• Weakness in dorsiflexion of foot<br>• Most likely to develop compartment syndrome |
| Lateral | • Peroneus longus and brevis<br>• Peroneal artery<br>• Superficial peroneal nerve | • Decreased sensation on dorsal foot<br>• Weakness in inversion and dorsiflexion of the foot |
| Deep posterior | • Muscles that enable plantar flexion of the foot (flexor digitorum longus, posterior tibialis, flexor hallucis longus, popliteus)<br>• Posterior tibial and peroneal arteries<br>• Tibial nerve | • Decreased sensation on plantar aspect of foot<br>• Weakness of toe flexion<br>• Pain with passive extension of toes |
| Superficial posterior | • Gastrocnemius and soleus<br>• No major arteries or nerves | • Least likely to develop compartment syndrome |

While patients are under anesthesia, they become unaware of pain that may be caused by prolonged malpositioning. Extremity positioning devices, such as well-leg holders, can constrict the compartments of extremities, causing an unexpected postoperative ACS.[3,11,12]

Other factors that put patients at risk for developing ACS include age younger than 35, male gender, anticoagulation or clotting abnormalities, high-energy trauma, and skeletal traction.[3,4,7,13] In general, any condition or situation that either increases the volume of a compartment or decreases the compartmental ability to accommodate that volume leads to higher pressures and an increased risk of compartment syndrome.

## Clinical manifestations

The "6 Ps" (pain, paresthesia, poikilothermia, pallor, pulselessness, and paralysis) are often described as the hallmark findings of ACS, but this is misleading.[4] Pain, especially pain that is disproportionate to the patient's injury or pain that is refractory to pain medication, is the earliest and most common manifestation of ACS.[1,3,9] Distal paresthesia may be present as swelling progresses and nerve conduction is affected but is considered a late finding. Poikilothermia, pallor, pulselessness, and paralysis are considered late findings.[1,3] Although the "6 Ps" are all results of ACS, they cannot be relied on for diagnosis.

When considering clinical manifestations, it is also important to consider conditions and situations that may mask the clinical manifestations of ACS. Because pain is the hallmark symptom, any condition that could conceal pain from the examiner should be approached with caution in high-risk patients. Some examples are sedation, withdrawal, altered mental status, neuraxial anesthesia, paralysis, neuropathy, and distracting injuries. Understanding the risk factors, clinical presentation, and circumstances that can mask presentation of ACS are the first steps in achieving early diagnosis.

## Physical exam

A consistent methodical physical exam is the next step in diagnosing a patient with suspected ACS. NPs must have a thorough understanding of the anatomy of the extremity they are examining. Having a systematic process for exam is also important. A physical exam should include assessments of pain at rest, sensation in distal nerve distributions, motor function, distal perfusion, pain with passive stretch of the muscle compartment involved, and direct palpation of swelling in the compartments.[3-5] Although each of these exam tools alone have a fairly low sensitivity for diagnosis, using them together helps build a clear clinical picture.[1,4]

As tibial fracture is by far the most common cause of ACS, NPs must be comfortable with a focused exam of the lower leg. The lower leg has four discrete compartments: anterior, lateral, deep posterior, and superficial posterior (see *Compartments in the lower leg*). As ACS develops and perfusion is compromised, the function of the neurovascular structures housed in the compartments is affected. (See *Exam findings: Lower leg compartments.*)

In patients at risk for ACS, focused "compartment-check" exams should be performed regularly for the first 24 to 36 hours following injury or surgery. Thorough documentation of serial exams provides a clear picture of swelling progression and subtle exam changes over time. Sensation in deep peroneal, superficial peroneal, and tibial nerve distributions should consistently be included. Motor function of the extensor hallucis longus, flexor hallucis longus, tibialis anterior, and gastrocnemius should also be included (see *Distal motor exam* and *Distal sensory exam*).

***Direct compartment pressure measurement is considered the gold standard for diagnosis.***

## Diagnosis

Failure to properly diagnose ACS can be devastating and lead to poor patient outcomes.[4,15] Early diagnosis is the key to preventing necrosis and permanent damage. Often, clinical exam alone is used to confirm diagnosis. Imaging, although used to visualize fractures, is not helpful in the diagnosis of compartment syndrome. If ACS is suspected and physical exam is equivocal, direct compartment pressure measurement is recommended and considered the gold standard for diagnosis.[2-4,8,16] There are several invasive devices used for direct pressure measurement, including needle manometer, wick catheter, and slit catheter. The most commonly used method is performed using a needle manometer device.[1] When interpreting pressure measurements, the compartment pressure must be viewed in comparison with the patient's BP at the time of measurement. Early orthopedic literature recommended diagnosis solely on an absolute compartment pressure threshold of 30 to 40 mm Hg.[1] Subsequently, it was recognized that tolerance of this absolute pressure varied greatly among patients, which was attributed to differences in perfusion pressure.[1] This is when the use of the differential delta pressure was introduced in 1975.[17] The absolute compartment pressure is subtracted from the diastolic BP, which yields a delta pressure. As the difference between diastolic and compartment pressure narrows, the delta pressure decreases. It is important to note that a lower delta pressure indicates a higher compartment pressure. A delta pressure less than 30 mm Hg is currently accepted as diagnostic for compartment syndrome.[1,3,4,16] There have been recommendations for continuous compartment pressure monitoring in high-risk patients, but this has not been widely adopted.[1,4]

Although direct compartment pressure measurement is a useful tool, it is contraindicated in patients who have clear clinical evidence of compartment syndrome because pressure measurement may delay operative intervention. When a diagnosis of ACS is made, immediate surgical intervention is indicated. Supplemental oxygen and neutral positioning of the extremity encourage tissue perfusion in the meantime.[18]

### Distal motor exam[14]

| Muscle group | Function |
|---|---|
| Extensor hallucis longus | Extension of great toe |
| Flexor hallucis longus | Flexion of great toe |
| Tibialis anterior | Dorsiflexion of foot |
| Gastrocnemius | Plantarflexion of foot |

### Distal sensory exam[14]

| Nerve | Distribution |
|---|---|
| Deep peroneal | Web between great and second toes |
| Superficial peroneal | Dorsal foot |
| Tibial | Plantar foot |
| *Saphenous | Medial foot |
| *Sural | Lateral foot |

*Sensation in the saphenous and sural nerve distributions is often included as part of a thorough neurovascular exam; however, they are extracompartmental cutaneous nerves that are not located within the compartments of the lower leg.

## Treatment

Complete compartment release with prompt fasciotomy is the only effective treatment for ACS.[19] Incising the restrictive fascia allows the swollen muscle to expand, relieving pressure on the vessels, and restoring blood flow.[3] Fasciotomy does not reverse damage that may have already been done but does prevent further tissue necrosis. Late fasciotomy in missed ACS is associated with extremely high rates of infection and

amputation.[4,20] According to Glass and colleagues, for lower extremity missed ACS in selected cases, late fasciotomy is not recommended because research findings suggest that a nonoperative management approach in selected stable patients may help prevent ischemia and reperfusion injury, infection, and possibly amputation.[20] Rather, the lower extremity is generally kept in a neutral position to encourage adequate blood flow to the tissues and intervention is deliberately delayed, allowing swelling to subside prior to debridement of necrotic tissue.

Fasciotomy, although necessary, is associated with its own risks. Morbidity rates have been reported as high as 95% and include infection, chronic pain, chronic numbness, decreased muscle strength, edema, reduced range of motion, contracture, cosmetic concerns, and limb loss.[4,21-24] After the initial fasciotomy procedure, incisions are covered with an expandable temporary construct (for example, negative pressure wound therapy and "shoelace" closures), providing time for tissue edema to resolve.[19,25,26] When tissue edema resolves, soft tissues and skin are closed primarily or with some type of graft.[16,21,26,27]

In addition to soft-tissue healing, underlying fractures must also be repaired, often surgically, to promote bone healing. The desired outcome is fracture union with soft-tissue healing. In the past, fasciotomy following tibia fracture was thought to increase the risk of postoperative infection and fracture nonunion. However, one recent study suggests that performing early decompression may not significantly increase the risk of infection or nonunion in tibial fractures.[28]

## Rehabilitation

As with any procedure, patient education following fasciotomy plays an important role in achieving positive outcomes. Following fasciotomy closure, patient education should center around pain control, infection prevention, and regaining function in the affected limb. Most patients will require an assistive device for ambulation and physical therapy postoperatively.[5] Rehabilitation time frames vary, depending on many factors. Weight-bearing status also varies depending on the type of fracture fixation and whether primary soft-tissue coverage was achieved. Orthopedic follow-up is needed for suture removal and serial imaging to confirm fracture alignment and union. After patients have been released from orthopedic care, primary care providers should be aware of the long-term effects of compartment syndrome and fasciotomy. Although there is little risk of recurrent ACS, it has been documented in athletes, which has been attributed to scarring.[5] Common long-term sequelae include chronic pain, persistent neurologic deficits, and dissatisfaction with scarring.[5,29]

## Conclusion

ACS is a surgical emergency that is most often associated with fracture of the tibia in orthopedics. As a challenging clinical diagnosis, there is significant variation between providers.[30] Delay in diagnosis and treatment can lead to poor outcomes and significant morbidity. To ensure prompt diagnosis and intervention, it is important for NPs to be aware of risk factors and to vigilantly monitor high-risk patients for clinical signs and symptoms of compartment syndrome. Because symptom development can be variable and unpredictable, a consistent systematic exam is critical.[31,32] NPs play an integral role in the early diagnosis of ACS, helping their patients achieve the best possible outcomes. NP

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