Injury 56 (2025) 112773

Contents lists available at ScienceDirect

Injury

journal homepage: www.elsevier.com/locate/injury

Diagnosis of acute compartment syndrome: current diagnostic parameters☆

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ARTICLE INFO

Keywords:
Compartment syndrome
Diagnosis
Management
Monitors
Sensors

ABSTRACT

Acute Compartment Syndrome (ACS) is a time-critical, limb-threatening condition best characterized by increased intracompartmental pressure that compromises tissue perfusion, leading to ischemia, hypoxia, and ultimately irreversible necrosis. Fractures to the extremities account for >80 % of all ACS cases, and those involving the tibia account for more than two-thirds of all ACS cases. Open fractures and those secondary to high-energy trauma and penetrating injuries like gunshots are at higher risk of ACS. Despite decades of research and technological advancement, early diagnosis has remained a significant clinical challenge due to the nonspecific symptoms and the absence of a definitive diagnostic gold standard. This review aims to provide a comprehensive overview of the pathophysiology, risk factors, diagnostic modalities, and current challenges associated with ACS. It emphasizes the importance of shifting the diagnostic paradigm from binary criteria toward objective outcome-based clinical decision-making. ACS should be redefined as a pathophysiological continuum rather than a binary diagnosis. Accurate, early recognition, and timely intervention are crucial for minimizing long-term morbidity. Future diagnostic approaches should prioritize objective markers of tissue health and clinical outcomes over static thresholds. Several learned bodies have recommended continuous pressure measurement, which is seen in the newer literature as highly accurate. Continued research is needed to develop standardized classification systems or treatment protocols.

Introduction

Acute Compartment Syndrome (ACS) is a potentially devastating condition that occurs when increased pressure within a closed myofascial compartment compromises the circulation and function of the tissues within that space [1,2]. It is most commonly observed following trauma, such as fractures, especially of the tibia or crush injuries. Still, it may also result from vascular injury or revascularization, burns, or tight cast immobilization [3]. The pathophysiology of ACS centers on the principles of capillary perfusion pressure and interstitial fluid dynamics [4,5]. Within a healthy compartment, capillary blood flow maintains the supply of oxygen and nutrients to muscle, nerves, and soft tissue. When compartmental pressure rises due to hematoma, edema, or external compression, it exceeds venous pressure, impairing outflow [6]. Notably, in early ACS, different muscle regions react temporally separately. This is why other muscle areas have been seen as having different biomarker values. This process, however, sets off a cycle of increasing compartmental pressure, reduced perfusion, and progressive ischemia. This progresses from local areas of high pressure and cell compromise to eventual whole compartment pressure rises, causing muscle death globally.

The ischemic cascade results in cellular hypoxia and anaerobic metabolism, accumulating lactate, reactive oxygen species, and inflammatory cytokines. Muscle cells begin to swell and break down, releasing myoglobin, potassium, and creatine kinase into the circulation [7]. This may happen first in high metabolic demand muscles, which are more sensitive to oxygen gradients. Eventually, the entire muscle and its contained elements are affected as pressure trends slowly increase. Nerve conduction also fails rapidly due to compression and reduced blood supply. Without timely intervention, which currently consists of surgical decompression via fasciotomy, this process can lead to irreversible damage within 4 to 6 h [8] after whole compartment involvement.

Understanding the underlying pathophysiology is critical, not only for early recognition but also for targeted diagnostic strategies. Previous reliance on clinical signs is inaccurate [9,10]. As clinical signs can be

☆ This paper is part of a Supplement supported by the Osteosynthesis and Trauma Care Foundation (OTCF) through a research grant from Stryker.
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https://doi.org/10.1016/j.injury.2025.112773
Accepted 22 September 2025
Available online 22 September 2025

subtle and nonspecific early on, incorporating this physiological insight of pressure trends into diagnostic models can help drive the use of adjunct monitoring technologies and prevent long-term morbidity such as muscle necrosis, limb loss, nerve damage, and chronic pain.

Causes and risk factors

Acute Compartment Syndrome (ACS) remains one of the most controversial and high-stakes, time-sensitive emergencies in orthopedic trauma care. Despite decades of improvements in clinical management, modern diagnostic tools, and extensive research, the condition continues to challenge surgeons with its vague symptoms, lack of an accurate diagnostic test, and the potential devastating consequences of both under- and over-treatment. To effectively anticipate and prevent complications, it is essential to understand the key clinical scenarios and conditions that predispose patients to developing ACS.

Fractures to the extremities account for >80 % of all ACS cases, and those involving the tibia account for more than two-thirds of all ACS cases [11]. Vascular injuries substantially increase the rate of fasciotomies, but it is difficult to assess how many would have associated traumatic ACS. Open fractures as well as crush injuries lead to a higher rate of fasciotomy [11–14]. Similarly, high-energy trauma and penetrating injuries like gunshots are at a higher risk of ACS [15–17]. One study, which described ACS in patients sustaining gunshot wounds (GSWs), did not find that there was a significant association between GSWs, motor vehicle injuries, and falls and the development of ACS. However, other factors found to significantly contribute to ACS, including young age and diaphyseal location, were significantly associated with ACS. Given that mechanism alone may not be a predictor of ACS development, surgeons should consider the location of the fracture, energy (high- vs. low-velocity gunshot wounds), patient age, and potentially other factors when estimating the potential for the development of ACS [17].

Comminuted and simpler fractures in specific locations, such as the tibial plateau or shaft, are at a higher risk [12]. Non-traumatic events can also lead to ACS. For instance, revascularisation after ischemia, vascular clamping, or tourniquet release is linked to ACS. Tight splints, bandages, and increased external compressive force can also lead to ACS [18–21]. Circumferential (full-thickness) burns will disrupt venous return, potentially causing an increase in pressure, requiring urgent escharotomy [22]. Strenuous exercise, such as CrossFit and military training, can also lead to rhabdomyolysis and ACS [23–25]. Hematological dysfunction, such as hemophilia or anticoagulant therapy, can lead to large muscular compartment hematomas and eventually ACS [26]. ACS has been seen in the so-called found-down presentation [27].

Understanding ACS risk factors is essential for clinicians to identify at-risk patients early and intervene with adequate monitoring or surgery [28] before irreversible damage occurs. Prompt recognition, a high index of suspicion, and timely fasciotomy can significantly reduce the morbidity associated with this orthopedic emergency.

Diagnostics

Non-invasive methods

Most clinicians still rely on physical examination to rule in or out ACS. Pain with passive stretch or pain out of proportion to the injury was traditionally considered the best approach; however, their subjective nature was demonstrated to be unreliable in more recent studies looking at ACS Surgery results [10] – in particular, the use of the Ps for diagnosis. Multiple studies have also demonstrated the unreliability of clinical exams [9,10,29].

There have been multiple attempts at assessing the state of the muscle using several imaging modalities, such as plain radiography and CT scan [30], and more recently, MRIs [31,32]. MRIs have been found to have limited efficacy, as edema alone was not sufficiently informative to establish a treatment decision. Near infrared spectroscopy (NIRS) in preclinical trials had promising results. However, the data acquisition was limited to a depth of 3 cm and was affected by darker skin tones, skin damage, hematomas, and serous fluid collections. NIRS also required a secondary measurement site on a control compartment [33–35]. All of these factors make the applicability of the technology difficult and unreliable. Ultrasound has also been investigated as a non-invasive diagnostic tool. In fact, shear wave elastography [36] and pulsed phase-locked loop ultrasound [37] have both been attempted, but with limited accuracy.

Direct measurement of tissue hardness has been tested but shown to be limited by its discontinuous measurement approach. It was also heavily influenced by the sex of the patient and the fat deposition around the muscles [38–41]. Preclinical studies of the leg, forearm, and hand have shown that experts cannot diagnose a tight compartment [29].

Invasive methods

Invasive methods require the introduction of a catheter or sensor through the skin and into the muscle compartment. Catheter fluid-based models require the introduction of a catheter connected to an external sensor with a column of water, which transfers the forces between the two. They require injecting a small quantity of fluid inside the compartment, which can greatly affect their accuracy [9,42,43]. Ensuring a fluid bleb is at the same pressure as the surrounding muscle is difficult. Though capable of measuring the pressure continuously, this measurement modality is impractical because of the inability to ensure a constant fluid pressure in the column and the possibility of blood clotting within the catheter tip or line [18,42–45].

To overcome the limitation of fluid-based models, MEMS-based devices were developed. These function by introducing the sensor directly in the muscle compartment to allow for direct pressure measurement in an array of physical environments without needing to balance fluid columns. This technique removes the need to transfer fluid pressure forces outside the patient's body [18,43,46] and provides continuous data points [28,43,47,48].

Diagnostic modalities

Without a gold standard, the combination of physical exam and continuous MEMS intra-compartmental pressure (ICP) has established itself as the main approach to confirm or rule out ACS. The study of ACS diagnosis had been complicated further by the lack of a classification system for the clinical outcomes. A recent panel of experts has validated a classification method for ACS [49]. Hopefully, its use in ongoing studies will allow for comparing ACS outcomes using a more standard method. There is no confirmatory diagnosis method and a strong bias for surgical interventions, and this complicates the differentiation between prophylactic and therapeutic fasciotomies. Additionally, no specific criteria exist for ACS. McQueen et al. proposed muscle escape as a sign of positivity [16]. The inability to close the fasciotomy has also been suggested [50] as well as the presence of sequelae [50]. The validated study above examined the extent of muscle death, the timing of closure, and the closure method for post-surgical wounds, which were validated. The use of histopathology has been limited to animal models [51].

Clinicians have relied on various P's (Pain, Pallor, Poikilothermia, Pulselessness, Paresthesia, Paralysis, Pressure Palpation) to diagnose compartment syndrome. But recent machine learning approaches have shown that using >3Ps increases the diagnostic accuracy only marginally. Although the presence of pain is the most commonly used marker, it is a poor predictor for ACS [10]. Digital palpation of the compartment is the strongest predictor of ACS despite its lack of reproducibility [45]. However, the late occurrence of positive pressure palpation in the clinical evolution of ACS makes it impractical and dangerous, as late fasciotomies lead to increased morbidity and likelihood of sequelae [52, 53]. Cadaver and clinical studies show physicians cannot accurately diagnose ACS [44,45]. Additionally, in comatose, sedated, distracted, or intoxicated patients, assessment of the full 7P's is not possible.

Pressure measurement using catheter-based models was limited to single-point measurement and required serial measurement throughout the clinical evolution of ACS. The difficulty of the setup meant that a third of the physicians used it correctly, a second third made lesser errors in technique, and nearly a third made catastrophic errors. Only 60 measurements with the correct technique were within 5mm Hg of the standard pressure [44]. Given the limitation in measurement resolution inherent to catheter-based devices, clinicians were tempted to use them in series. The strategy significantly reduced the percentage of false positives compared to those who received only one measurement [54]. A recent comparative study showed that using a MEMS-based sensor (model REF: MYO-00119) directly inside the compartment showed significantly higher accuracy and less bias when compared to catheter-based models (Stryker/C2DX compartment pressure monitor, compartmental pressure monitoring system from Synthes) [43].

Decompression threshold

The literature is replete with threshold pressure numbers for decompression of compartments. Much of the issue with these threshold numbers is using older technology to determine a threshold. Placement of the measuring devices as a single point in a non-constant part of the compartment has led to much confusion. Studies have indicated that normal ICP pressures have various values. One study showed close to 10 mm Hg [55], while early studies in the 1970s and 1980s suggested a critical pressure around 30 to 40mm Hg for initiating a fasciotomy [56–60]. This latter value made more sense, as it is usually slightly above capillary pressure. Some suggested fasciotomies are required at much higher pressures [61]. More recent work from McQueen et al. incorporated blood pressure (diastolic) to aid in determining muscle release. They showed that patients could tolerate different ICPs, depending on their blood pressure [62–64], and recommended a muscle release when the delta pressure (ICP- diastolic) was below 30 for over 2 h. Furthermore, they showed that using continuous pressure measurement in conjunction with physical exams led to superior clinical outcomes compared to clinical exams alone. In fact, on average, fasciotomies were performed 16 h earlier, with a 91 % lower likelihood of sequelae and even a lower non-union rate [62]. However, their setup was still catheter-based, making it difficult to use and reproduce across hospital centers. Implementing continuous pressure monitoring using MEMS technology showed impressive performance when combined with a physical exam, compared to pre-existing technologies [28]. This technology is user-agnostic. The increased specificity drastically reduced the number of false positives, thus reducing the number of unnecessary fasciotomies and their possible complications and economic costs. The financial model showed that they could drastically reduce the average price of treatment of lower leg fractures, reduce the length of stay, and make peripheral nerve block a possibility in the future for lower leg fractures.

Summary

Acute compartment syndrome (ACS) is a progressive pathophysiological process characterized by an initial local increase in cellular pressure and death. This can be tracked objectively with continuous data points. This leads to compromised perfusion, followed by tissue ischemia, hypoxia, and ultimately necrosis. Given this continuum, establishing a universal threshold for surgical intervention is inherently challenging, as individual patients may exhibit variable tolerance to degrees of circulatory compromise. Regardless of personalized limits, pressure trends can be tracked, and the point of no return can be visualized in graphic form with new MEMS-based technology. ACS should be conceptualized less as a binary entity and more for its physiological sequelae and progression. Diagnostic technologies, clinical approaches, and management guidelines should be evaluated primarily through the lens of patient-centered outcomes rather than on the presence or absence of predefined threshold diagnostic criteria.

CRediT authorship contribution statement

Yasser Bouklouch: Writing – review & editing, Writing – original draft. **Theodore Miclau:** Writing – review & editing, Writing – original draft, Conceptualization. **Edward Harvey:** Writing – review & editing, Writing – original draft, Conceptualization.

Declaration of competing interest

This paper is part of a Supplement supported by The Osteosynthesis and Trauma Care Foundation (OTCF), which receives funding from the Stryker Corporation.

Potential conflicts of interest and funding sources

B.Y. and EJH were employees of MY01 Inc., which produces pressure sensors to diagnose compartment syndrome. TM is a consultant for MY01 Inc.

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