Chapter 14
Compartment Syndrome in Polytrauma Patients

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Background

Diagnosing compartment syndrome in the awake and oriented patient is difficult, and the diagnosis becomes even more problematic in the polytrauma patient. Many clinicians argue that the clinical signs and symptoms are the most important components in identification. Prompt diagnosis and treatment are just as critical in polytrauma patients for the prevention of long-term sequelae, including possible amputation. Unfortunately polytrauma patients who are often obtunded, intubated, and unable to cooperate with an examination, combined with painful high-energy injury to the limbs, create a very difficult environment for recognition of compartment syndrome. It is imperative to identify high-risk injuries and patients and to maintain a high level of clinical suspicion in those patients unable to participate in a clinical examination, and even then the possibility of a missed compartment syndrome is very real.

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**Recommendations**

Prompt diagnosis of compartment syndrome remains the most integral factor to a successful outcome of compartment syndrome in polytrauma patients as it is in patients without polytrauma. Delay in diagnosis, and ultimately treatment of compartment syndrome, has been associated with permanent sensory and motor deficits, contractures, infection, and at times, amputation of the limb [1–3].

**Physical Examination**

Compartment syndrome has often been referred to as a clinical diagnosis, with various signs and symptoms postulated to be the most important or earliest presenting indicator. However, all of these clinical signs and symptoms require an alert and conscious patient, with a review of the literature suggesting a frequent delay in reaching the diagnosis of compartment syndrome as symptoms can be masked by other injuries in polytrauma patients [4–8].

Polytrauma patients have numerous risk factors for possible delay in diagnosis of compartment syndrome, including distracting injuries and altered consciousness. In the study by Frink et al., patients with multiple injuries and an Injury Severity Score > 16 had a mean time between admission and fasciotomy of 38 hours, in comparison with those patients with isolated injuries at 13 hours [9]. In those patients where altered mental status or pain evaluation is difficult to evaluate, the clinical signs of compartment syndrome become less helpful. Determining pain out of proportion that is expected for the injury, pain on passive stretch, paralysis and motor changes, and paresthesias requires a conscious and cooperative patient. Even in the setting of an alert patient with multiple injuries, determining the appropriate level of pain for a specific type of fracture is difficult. Pain can be influenced by psychosocial factors including anxiety, is of variable intensity, and is almost universal following any injury [10–12]. Furthermore, while increased analgesic requirements are important to assess, this is less reliable in the setting of multiple injuries and cannot be utilized in the presence of an unconscious patient. Paresthesia and paralysis are generally considered late clinical findings of compartment syndrome and cannot be evaluated in the unconscious patient.

The difficulty in performing a physical examination on an intubated patient is evident to all clinicians who have attempted this daunting task on a polytrauma patient. Take, for example, an attempt at physical inspection of swelling. Palpable and visible swelling are almost universally seen signs with acute compartment syndrome, but remain highly subjective even in an awake patient with an isolated injury. Assessment is routinely inadequate in polytrauma patients due to overlying splints and bandages and being inadequate to evaluate the deep compartments. Although sensitivity for this clinical finding is higher than other clinical signs and symptoms at 54%, the specificity (76%) and negative predictive value are far inferior (63%).
Furthermore, a physician’s ability to manually detect isolated elevations in leg intracompartmental pressure has been identified as poor. In the study by Schuler et al., the frequency with which an anterior compartment fasciotomy was recommended was 19% when the pressure was 20 mm Hg, 35% when it was 40 mm Hg, 45% when it was 60 mm Hg, and 56% when it was 80 mm Hg. In the deep posterior compartment, it was 19% when the pressure was 20 mm Hg, 19% when the pressure was 40 mm Hg, 56% when it was 60 mm Hg, and 64% when it was 80 mm Hg. When asked to qualify clinical interpretations of firmness as soft, compressible, or firm, participants described the compartment as firm in only 45% of the cases in which the pressure was 80 mm Hg [16].

**Risk Factor Assessment**

As clinical signs and symptoms are of questionable utility in the setting of the intubated, unalert, or sedated patient, a high index of clinical suspicion is even more critical in the evaluation process of compartment syndrome in a polytrauma patient. This can begin with recognizing demographic factors and mechanisms of injury that place patients at increased risk for compartment syndrome. Perhaps the strongest predictor of compartment syndrome after a tibial diaphyseal fracture is youth, with the prevalence of compartment syndrome in adolescents and young adults 50 times greater than in those older than 60 years. Additionally, in the study by McQueen et al., the highest prevalence of compartment syndrome was between 12 and 19 years and 20 and 29 years [17]. This has previously been thought to be due to the relative size of the compartment and the muscle contained within it [17, 18]. In the studies by Court-Brown et al. and Shore et al., they identified adolescents sustaining high-energy tibia fractures as at-risk patients for compartment syndrome [19, 20].

Location of the injury can also help allocate patients into high-risk and low-risk groups. Compartment syndrome is most classically associated with tibia fractures, with rates ranging from 2.7% to 15% in the literature [18–26]. In particular, high-energy tibial plateau fractures have been associated with a greater risk of compartment syndrome, with associated fibular fracture increasing the risk [27–29]. The proposed reasons behind the relative increased risk of compartment syndrome in comparison with other aspects of the tibia include increased muscle proximally, the location of the nutrient vessel, and the robust venous supply around the knee. Additionally, in regard to tibia fractures, fracture length greater than 20% of the tibial length was found to be a risk factor for compartment syndrome in the study by Allmon et al. [27].

Furthermore, radiographic predictors of compartment syndrome can become very useful in polytrauma patients unable to participate in the clinical examination. In the study by Ziran et al., the displacement of the tibial anatomic axis from the femoral anatomic axis divided by the width of the femur at its widest point was a predictor of compartment syndrome in tibial plateau fractures. They found that a
ratio of greater than 10% tripled the risk of developing compartment syndrome [30]. Additionally, ballistic fractures of the fibula and tibia have been associated with compartment syndrome. In particular, ballistic fractures of the proximal third of the tibia and fibula were at greatest risk for compartment syndrome among ballistic injuries [31].

**Serum Markers**

The use of other screening tools including specific biologic markers has also been explored in their utility in diagnosing or identifying an at-risk patient population for compartment syndrome. These objective measurements may be of particular use in the polytrauma patient, whose mental status may prevent clinical evaluation. In the prospective observational study by Kosir et al., patients who met high-risk criteria including pulmonary artery catheter-directed shock resuscitation, open or closed tibial shaft fracture, major vascular injury below the aortic bifurcation, abdominal compartment syndrome, or pelvic or lower extremity crush injury underwent a compartment syndrome screening protocol at admission and every 4 hours thereafter for the first 48 hours of admission. This screening included a comprehensive physical examination including lower leg circumference measurement, pain assessment, and vascular and neurologic examination, with any suspicious or unreliable physical examination findings mandating compartment pressure monitoring. No missed compartment syndrome was observed in the patients involved in this study, and the authors found during the first 24 hours of admission statistically greater base deficits (12.9 ± 5.9 mEq/L versus 7.5 ± 5.0 mEq/L), greater lactate levels (13.0 ± 5.2 mmol/L versus 5.4 ± 2.8 mmol/L), and greater PRBC requirements (28.4 units vs. 9.3 units) in those that developed compartment syndrome [32]. Other biological markers that have been associated with compartment syndrome include creatine kinase (CK) and lactate dehydrogenase. In patients treated with isolated limb perfusion, CK values exceeding 1000 IU/L after the first post isolated limb perfusion treatment day was correlated with compartment syndrome. LDH values peaked 2.9 days after CK values and was found to be less useful [33, 34]. In the study by Valdez et al., maximum CK greater than 4000 U/L, chloride levels greater than 104 mg/dL, and BUN less than 10 mg/dL were associated with the development of compartment syndrome. When all variables were absent, no patients had compartment syndrome. When one, two, or three of these variables were present, the percentage of patients with compartments syndrome was 36%, 80%, and 100%, respectively [35]. However, this research was a retrospective study with limited patient numbers, with future studies needed to validate these findings and correlate them with clinical examination. Furthermore, CK values may be of limited in utility in polytrauma patients, as they can be elevated due to multiple injuries rather than the presence of compartment syndrome.
**Intracompartmental Pressure Measurements**

In addition to altered consciousness and other factors that interfere with history taking and assessment of physical signs, polytrauma patients, for various reasons, may have lowered diastolic pressures. This scenario could place these patients at increased risk for compartment syndrome as it can occur at relatively lower threshold pressures. Given the lack of clinical examination, the use of invasive intracompartmental pressure monitoring is appealing in this patient population. One of the first invasive measurement techniques used was a needle manometer, placed within the compartment and connected to a column filled with a mixture of saline and air, with the pressure calculated through the accompanying manometer [36–38]. The Stryker intracompartmental pressure monitor has been frequently used in North America, with current data suggesting optimal placement of the device within 5 cm of the level of the fracture but not at the level of the fracture [39, 40]. The anterior and deep posterior compartments of the lower leg have been most commonly advocated for measurement, as the anterior compartment is most frequently involved and the deep posterior compartment at increased risk for neglect [14, 22, 25, 39]. The threshold for decompression has undergone extensive deliberation, with the debate centered on using either the intracompartmental pressure alone or the differential pressure ($\Delta P$). Studies have recognized that individual tolerance to absolute intracompartmental pressure varies widely and appears to be intrinsically associated with the systemic blood pressure or perfusion pressure [7, 15, 23, 37, 41–43]. As such, differential pressure has gained favor in determining thresholds for compartment syndrome. Clinical evidence and experimental data have suggested that a pressure difference of $\leq 30$ mm Hg between intracompartmental pressures and diastolic pressure prior to anesthesia application should be a safe threshold for fasciotomy [25, 44–46].

However, the utilization of single intracompartmental pressure measurements may lead to overtreatment and unnecessary fasciotomies. In the study by Prayson et al., 84% of patients had differential pressures of 30 mm Hg with no clinical evidence of compartment syndrome [47]; however, this sample was small with disparate issues. In the study by Whitney et al., a consecutive cohort of 48 patients with tibia fractures and no clinical evidence of compartment syndrome at presentation found 35% of patients with differential pressures of 30 mm Hg [48], validating the general concern brought up by Prayson for a high false-positive rate with single pressure measurements. The Edinburgh protocol, which involved continuous pressure measurement and employing a $\Delta P$ of $\leq 30$ mm Hg over a 2-hour period as the threshold for fasciotomy has been suggested as a means to reduce the time to fasciotomy while not significantly raising fasciotomy rates [25]. However, while clinical data seems to indicate that no compartment syndrome will be missed using a $\Delta P$ of $\leq 30$ mm Hg as a threshold, this does not necessarily mean that this value signifies the presence of compartment syndrome.

Ultimately, current best practice includes high clinical suspicion and awareness. As polytrauma patients present unique challenges in the diagnosis of compartment syndrome,
syndrome, it is imperative to recognize at-risk patient factors and to understand the clinical tools available in conjunction with one another to diagnose compartment syndrome but also to understand the limitations of our current diagnostic tools in polytrauma patients. As has been shown, the use of isolated clinical exams, laboratory markers, and compartment pressure monitoring may all yield high false-positive results in polytrauma patients and perhaps lead to unnecessary fasciotomies. However, missed compartment syndrome is a potentially very serious situation. Hence, the diagnosis of compartment syndrome in polytrauma patients remains a difficult challenge.

**Limitations and Pitfalls**

With varying conscious states, limited participation in the clinical examination, and distracting injuries, polytrauma patients present a unique clinical challenge when diagnosing compartment syndrome. The drawback with using biologic markers and compartmental pressures with minimal clinical correlation is that these objective markers may be most useful in telling clinicians who does not need fasciotomy rather than who does. The inability to distinguish among traumatized limbs with true ischemic compartment syndrome in its early stages before tissue necrosis has occurred, those with impending compartment syndrome, and those with no compartment syndrome are in large part responsible for the lack of consensus on how to manage at-risk patients.

While some have advocated for continuous compartment pressure monitoring in the unalert, sedated, or intubated patient, continuous pressure monitoring remains controversial and infrequently used in North America. In the study by McQueen et al., the ability to close fasciotomy wounds at 48 hours was used as an indicator for unnecessary fasciotomy [49]. However, this remains a somewhat subjective and unvalidated way to determine if compartment syndrome was truly present. Most orthopedic trauma surgeons have experienced cases of complex fractures that are difficult to close but have no suspicion of compartment syndrome and evident compartment syndrome that can be closed immediately if they were released early. Thus, the utility of this definition to define true compartment syndrome remains open. It additionally appears that the use of continuous pressure monitoring may lead to increased rates of fasciotomy [50]. However, in a patient with distracting injuries and other factors that obscure the clinical picture, this may be one of the most reliable tools in preventing late diagnosis of compartment syndrome. Unfortunately, the most reliable indicator of compartment syndrome remains unknown, and currently surgeons must balance for themselves the possible risk of overtreatment with unnecessary fasciotomy against the potential clinical and medicolegal consequences of missed compartment syndrome.

An important limitation that applies to all human research in the field of compartment syndrome is the lack of a solid definition of compartment syndrome. The literature almost universally uses the performance of a fasciotomy as synonymous
with compartment syndrome which creates great potential for research error given
the known disagreement between surgeons on which patients have compartment
syndrome [51]. This limitation is rarely discussed but is a major flaw affecting all of
the human work in this domain.

**Future Directions**

Future directions for the diagnosis of compartment syndrome in polytrauma patients
have focused on clinical labs or new sensors to diagnose and prevent compartment
syndrome. Multiple new techniques to diagnose compartment syndrome are cur-
rently being developed and investigated in prospective trials [52].

One future avenue that has been explored was introduced by Odland et al. [53].
In this pilot study, a novel compartment monitoring system (CMS) catheter has two
components: (1) one measures intramuscular pressure and (2) another removes
excess tissue fluid. These catheters were inserted in the operating room after fixa-
tion of isolated tibial shaft fractures treated with intramedullary fixation in ten
patients. This was done in conjunction with conventional Stryker catheters con-
nected to the Stryker Intra-Compartmental Pressure Measuring Device. Intramuscular and blood pressure readings were recorded hourly for all catheters
over a 24-hour observation period. They concluded that in comparison to conven-
tional Stryker catheters, the CMS catheters were safe, had reasonable agreement in
intramuscular pressure values with a high degree of correlation ($R^2 = 0.8$), and
allowed for early and sustained reduction of intramuscular pressure with an average
ultrafiltrate removal of $1.9 \pm 0.2$ mL (1.2–2.7 mL). Additionally, the ultrafiltrate
that was removed was analyzed for LDH and CK levels and was found to have a
positive correlation between intramuscular pressure and enzyme level and a nega-
tive correlation between pulse pressure and enzyme level. Serum levels of CK and
CK and LDH have been shown to be elevated but are not diagnostic for compart-
ment syndrome [24, 25], and although low serum levels may mean no injury, low
levels may also occur with severe injury and no perfusion. However, technology that
will provide information about focal cellular metabolism or degree of cellular injury
would be a significant advancement in the diagnosis and management of compart-
ment syndrome.

Biomechanical markers have additionally been explored as a means of diagnos-
ing compartment syndrome. Glucose, lactate, and pyruvate levels can detect muscle
ischemia in situations of arterial occlusion, venous hypertension, and hypoperfusion
[54, 55], and tissue glucose concentration was shown to detect ischemia within
15 minutes of vessel occlusion [56]. Glucose levels, as it relates to compartment
syndrome, was studied in a canine model. In this study, interstitial glucose monitors
were inserted into 12 canines, and acute compartment syndrome was created with
mean compartment pressures of 74 mm Hg. Within 15 minutes of compartment
syndrome, glucose concentration and oxygen tension were significantly decreased,
and intramuscular glucose concentrations of less than 97 mg/dL was found to be
100% sensitive for the presence of compartment syndrome [57]. However, this has yet to be studied in traumatized human tissue, and the intramuscular component of the probe is too short to reach into a human tibial compartment. Nevertheless, this is one future direction that may allow for objective data to confirm the presence of compartment syndrome when clinical diagnosis is not possible.

A noninvasive avenue that does not require an alert and conscious patient has focused on measuring tissue oxygenation with use of near-infrared spectroscopy to determine the presence of compartment syndrome. Near-infrared spectroscopy utilizes differential light absorption properties to solve for the concentrations of oxygenated and deoxygenated hemoglobin through the use of the Beer–Lambert law [58–62]. Similar to conventional pulse oximetry, near-infrared spectroscopy utilizes light to solve for the percentage of oxygenated hemoglobin, although near-infrared spectroscopy can sample tissue as deep as 3 cm below the skin [58, 60, 63–66]. An initial animal study utilizing an infusion compartment syndrome model in pigs demonstrated an inverse relationship between near-infrared spectroscopy values and compartment syndrome [67]. In the study by Schuler et al. among 26 patients, six patients with unilateral tibial fractures in the absence of compartment syndrome had injured and uninjured limbs measured with near-infrared spectroscopy and compared these to uninjured control subjects. Results of this study showed a predictable increase in oxygenation of injured limbs by 15.4% points compared to matched uninjured contralateral limbs, demonstrating the body’s increase in blood flow in response to injury [67]. In the subsequent study by Schuler et al., 14 patients enrolled after diagnosis of compartment syndrome both clinically and with intracompartmental pressure measurements were evaluated with near-infrared spectroscopy. Near-infrared spectroscopy values decreased by an average of 10.1%, 10.1%, 9.4%, and 16.3% in the anterior, lateral, deep posterior, and superficial posterior compartments, respectively. The authors postulated that these results suggest the clinician to be concerned about impaired blood flow to the injured limb should hyperemia in a patient with lower extremity trauma or fracture be absent [68]. Near-infrared spectroscopy could offer a means to evaluate the presence of absence of compartment syndrome in the intubated, unresponsive polytrauma patient. However, near-infrared spectroscopy values vary depending on skin pigmentation, and its applicability could be limited in patients with bilateral extremity injuries, and high-quality studies have recently been completed and await peer review publication to see if this technique is of clinical use [52].

**Take-Home Message**
The diagnosis of compartment syndrome remains a particularly challenging clinical entity in polytrauma patients. It has been well established that prompt diagnosis and surgical management of compartment syndrome provides the most optimal outcome for the patient. The diagnosis can become even more challenging in the polytrauma patient, where participation in the clinical examination can be limited due to altered consciousness, and
distracting injuries can complicate the clinical picture. Recognizing at-risk patients remains a critical first step. In particular, youth, especially between ages 12 and 29 years, appears to be a key factor for developing compartment syndrome, with tibia fractures being the most common precipitating injury.

As regards injury, high-energy tibial plateau fractures with tibial anatomic axis deviation greater than 10% in comparison with the femoral anatomic axis, and ballistic injuries to the proximal tibia and fibula, remain high-risk fractures. Clinical markers including elevated lactate, large base deficits, elevated CK and LDH levels, and greater PRBC requirements should raise clinical suspicion in those patients unable to participate in the clinical examination. Finally, the use of serial compartment measurements in conjunction with the aforementioned findings can be helpful in the timely diagnosis of compartment syndrome. Future work is investigating measures of oxygen levels, glucose, lactate, and other local measures within the limb, but these are not yet in widespread clinical use and await validation. The authors currently advocate for the combined use of these clinical tools to diagnose compartment syndrome in the polytrauma patient, with the recognition that individual use of these tools can misdiagnose or overdiagnose compartment syndrome.

References

1. Muullett H, Al-Abed K, Prasad CV, et al. Outcomes Of compartment syndrome following intramedullary nailing of tibial diaphyseal fractures. Injury. 2001;32:411–3.
2. Prasarn ML, Ouellette EA, Livingstone A, et al. Acute pediatric upper extremity compartment syndrome in the absence of fracture. J Pediatr Orthop. 2009;29:263–8.
3. 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.
4. Garfin SR, Mubarak SJ, Evans KL, et al. Quantification of intracompartmental pressure and volume under plaster casts. J Bone Joint Surg Am. 1981;63:449.
5. Lee BY, Brancato RF, Park IH, Shaw WW. Management of compartmental syndrome: diagnosis and surgical considerations. Am J Surg. 1984;148:383.
6. Rorabeck CH. A practical approach to compartmental syndromes: part III. Management Instr Course Lect. 1983;32:102.
7. Rorabeck CH. The treatment of compartment syndromes of the leg. J Bone Joint Surg Br. 1984;66:93.
8. Sundararak GD, Mani K. Management of Volkmann’s Ischemic contracture of the upper limb. J Hand Surg Br. 1985;10:401.
9. Frink M, Klaus AK, Kuther G, et al. Long term results of compartment syndrome of the lower limb in polytraumatised patients. Injury. 2007;38:607–13.
10. Matsen FA 3rd, Krugmire RB Jr. Compartmental syndromes. Surg Gynecol Obstet. 1978;147(6):943–9.
11. Eaton RG, Green WT. Volkmann’s ischemia. A volar compartment syndrome of the forearm. Clin Orthop Relat Res. 1975;113:58–64.
12. Vranceanu AM, Barsky A, Ring D. Psychosocial aspects of disabling musculoskeletal pain. J Bone Joint Surg Am. 2009;91(8):2014–8.
13. Ulmer T. The clinical diagnosis of compartment syndrome of the lower leg: are clinical findings predictive of the disorder? J Orthop Trauma. 2002;16(8):572–7.
14. Matsen FA 3rd, Clawson DK. The deep posterior compartmental syndrome of the leg. J Bone Joint Surg Am. 1975;57(1):34–9.
15. Mubarak SJ, Owen CA, Hargens AR, Garetto LP, Akeson WH. Acute compartment syndromes: diagnosis and treatment with the aid of the wick catheter. J Bone Joint Surg Am. 1978;60(8):1091–5.
16. Schuler FD, Dietz MJ. Physicians’ ability to manually detect isolated elevations in leg intracompartmental pressure. J Bone Joint Surg Am. 2010;92:361–7.
17. McQueen MM, Duckworth AD, Aitken SA, Sharma RA, Court-Brown CM. Predictors of compartment syndrome after tibial fracture. J Orthop Trauma. 2015;29:451–5.
18. McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome. Who is at risk? J Bone Joint Surg Br. 2000;82:200–3.
19. Court-Brown CM, Byrnes T, McLaughlin G. Intramedullary nailing of tibial diaphyseal fractures in adolescents with open physes. Injury. 2003;34(1):781–5.
20. Shore BJ, Glotzbecker MP, Zarakowski D, Gelbard E, Hedequist DJ, Matheney TH. Acute compartment syndrome in children and teenagers with tibial shaft fractures: incidence and multivariable risk factors. J Orthop Trauma. 2013;27(11):616–21.
21. Mullett H, Al-Abed K, Prasad CV, O’Sullivan M. Outcomes of compartment syndrome following intramedullary nailing of tibial diaphyseal fractures. Injury. 2001;32(5):411–3.
22. Al-Dadah OQ, Darrah C, Cooper A, Donell ST, Patel AD. Continuous compartment pressure monitoring vs. clinical monitoring in tibial diaphyseal fractures. Injury. 2008;39(10):1204–9.
23. Blick SS, Brumback RJ, Poka A, Burgess AR, Ebraheim NA. Compartment syndrome in open tibial fractures. J Bone Joint Surg Am. 1986;68(9):1348–53.
24. Finkemeier CG, Schmidt AH, Kyle RF, Templeman DC, Varecka TF. A prospective, randomized study of intramedullary nails inserted with and without reaming for the treatment of open and closed fractures of the tibial shaft. J Orthop Trauma. 2000;14(3):187–93.
25. McQueen MM, Court-Brown CM. Compartment monitoring in tibial fractures. The pressure threshold for decompression. J Bone Joint Surg Br. 1996;78(9):99–104.
26. Williams J, Gibbons M, Trundle H, Murray D, Worlock P. Complications of nailing in closed tibial fractures. J Orthop Trauma. 1995;9(6):476–81.
27. Allmon C, Greenwell P, Paryavi E, Dubina A, O’Toole RV. Radiographic predictors of compartment syndrome occurring after tibial fracture. J Orthop Trauma. 2016;30:387–91.
28. Barei DP, Nore SE, Mills WJ, et al. Complications associated with internal fixation of high-energy bicondylar tibial plateau fractures utilizing a two-incision technique. J Orthop Trauma. 2004;18:649–57.
29. Egol KA, Tejwani NC, Capla EL, et al. Staged Management of High-Energy Proximal Tibia Fractures (OTA types 41): the results of a prospective, standardized protocol. J Orthop Trauma. 2005;19:448–55.
30. Ziran BH, Becher SJ. Radiographic predictors of compartment syndrome in tibial plateau fractures. J Orthop Trauma. 2013;27:612–5.
31. Meskey T, Harcastle J, O’Toole RV. Are certain fractures at increased risk for compartment syndrome after civilian ballistic injury? J Trauma. 2011;71(5):1385–9.
32. Kosir R, Moore FA, Selby JH, et al. Acute lower extremity compartment syndrome (ALECS) screening protocol in critically ill trauma patients. J Trauma. 2007;63(2):28–75.
33. Shields RW, Root KE, Wilbourn AJ. Compartment syndromes and compression neuropathies in coma. Neurology. 1986;36:1370–4.
34. Vrouenraets BC, Kroon BB, Klaase JM, et al. Value of laboratory tests in monitoring acute regional toxicity after isolated limb perfusion. Ann Surg Oncol. 1997;4:88–94.
35. Valdez C, Schroeder E, Amdur R, Pascual J, Sarani B. Serum creatine kinase levels are associated with extremity compartment syndrome. J Trauma Acute Care Surg. 2013;74(2):441–7.
36. Matsen FA 3rd, Winquist RA, Krugmire RB Jr. Diagnosis and management of compartmental syndromes. J Bone Joint Surg Am. 1980;62(2):286–91.
37. Matsen FA 3rd, Mayo KA, Sheridan GW, Krugmire RB Jr. Monitoring of intramuscular pressure. Surgery. 1976;79(6):702–9.
38. Whitesides TE Jr, Haney TC, Harada H, Holmes HE, Morimoto K. A simple method for tissue pressure determination. Arch Surg. 1975;110(11):1311–3.
39. 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(9):1285–92.
40. Harris IA, Kadir A, Donald G. Continuous compartment pressure monitoring for tibia fractures: does it influence outcome? J Trauma. 2006;60(6):1330–5.
41. 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.
42. Hargens AR, Akeson WH, Mubarak SJ, Owen CA, Evans KL, Garetto LP, Gonsalves MR, Schmidt DA. Fluid balance within the canine anterolateral compartment and its relationship to compartment syndromes. J Bone Joint Surg Am. 1978;60(4):499–505.
43. 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(1):53–7.
44. McQueen MM, Christie J, Court-Brown CM. Acute compartment syndrome in tibial diaphyseal fractures. J Bone Joint Surg Br. 1996;78(1):95–8.
45. White TO, Howell GE, Will EM, Court-Brown CM, McQueen MM. Elevated intramuscular compartment pressures do not influence outcomes after tibial fracture. J Trauma. 2003;55(6):1133–8.
46. Ozkayin N, Aktuglu K. Absolute compartment pressure versus differential pressure for the diagnosis of compartment syndrome in tibial fractures. Int Orthop. 2005;29(6):396–401.
47. Prayson MJ, Chen JL, Hampers D, Vogt M, Fenwick J, Meredick R. Baseline compartment pressure measurements in isolated lower extremity fractures without clinical compartment syndrome. J Trauma. 2006;60(5):1037–40.
48. Whitney A, O’Toole RV, Hui E, et al. Do One-Time intracompartamental pressure measurements have a high false-positive rate in diagnosing compartment syndrome? J Trauma. 2014;76(2):479–83.
49. 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(8):673–7.
50. Janzing M, Broos PL. Routine monitoring of compartment pressure in patients with tibial fractures: beware of overtreatment! Injury. 2001;32(5):415–21.
51. O’Toole RV, Whitney A, Merchant N, Kim T, Sagebien C. Variation in diagnosis of compartment syndrome by surgeons treating tibial shaft fractures. J Trauma. 2009;67(4):735–41.
52. Schmidt AH, Bosse MJ, Frey KP, et al. Predicting acute compartment syndrome (PACS): the role of continuous monitoring. J Orthop Trauma. 2017;31S(1):S40–7.
53. Odland RM, Schmidt AH. Compartment syndrome ultrafiltration catheters: report of a clinical pilot study of a novel method for managing patients at risk of compartment syndrome. J Orthop Trauma. 2011;25:358–65.
54. Setala L, Korvenoja EM, Harma MA, Alhava EM, Uusaro AV, Tenhunen JJ. Glucose, lactate, and pyruvate response in an experimental model of microvascular flap ischemia and reperfusion: a microdialysis study. Microsurgery. 2004;24:223–31.
55. Setala L, Joukainen S, Uusaro A, Alhava E, Harma M. Metabolic response in microvascular flaps during partial pedicle obstruction and hypovolemic shock. J Reconstr Microsurg. 2007;23:489–96.
56. Sitzman TJ, Hanson SE, King TW, Gutowski KA. Detection of flap venous and arterial occlusion using interstitial glucose monitoring in a rodent model. Plastic Reconstr Surg. 2010;126:71–9.
57. Doro CJ, Sitzman TJ, O’Toole RV. Can intramuscular glucose levels diagnose compartment syndrome? J Trauma Acute Care Surg. 2014;76(2):474–8.
58. Arimoto H, Egawa M, Yamada Y. Depth profile of diffuse reflectance near-infrared spectroscopy for measurement of water content in skin. Skin Res Technol. 2005;11:27–35.
59. Fadel PJ, Keller DM, Watanabe H, Raven PB, Thomas GD. Noninvasive assessment of sympathetic vasoconstriction in human and rodent skeletal muscle using near-infrared spectroscopy and Doppler ultrasound. J Appl Physiol. 2004;96(4):1323–30.
60. Kim MB, Ward DS, Cartwright CR, Kolano J, Chlebowski S, Henson LC. Estimation of jugular venous O2 saturation from cerebral oximetry or arterial O2 saturation during isocapnic hypoxia. J Clin Monit Comput. 2000;16:191–9.
61. Mancini DM, Bolinger L, Li H, Kendrick K, Chance B, Wilson JR. Validation of near-infrared spectroscopy in humans. J Appl Physiol. 1994;77:2740–7.
62. Murkin JM, Adams SJ, Novick RJ, Quantz M, Bainbridge D, Iglesias I, Cleland A, Schaefer B, Irwin B, Fox S. Monitoring brain oxygen saturation during coronary bypass surgery: a randomized, prospective study. Anesth Analg. 2007;104:51–8.
63. Boushel R, Pott F, Madsen P, Radegran G, Nowak M, Quistorff B, Secher N. Muscle metabolism from near infrared spectroscopy during rhythmic handgrip in humans. Eur J Appl Physiol Occup Physiol. 1998;79:41–8.
64. Cui W, Kumar C, Chance B. Time-resolved spectroscopy and imaging of tissue. SPIE (The International Society for Optical Engineering). 1991:431:180–91.
65. Kaufman J, Almodovar MC, Zuk J, Friesen RH. Correlation of abdominal site near-infrared spectroscopy with gastric tonometry in infants following surgery for congenital heart disease. Pediatr Crit Care Med. 2008;9:62–8.
66. Schuler MS, Reisman WM, Whitesides TE Jr, Kinsey TL, Hammerberg EM, Davila MG, Moore TJ. Near-infrared spectroscopy in lower extremity trauma. J Bone Joint Surg Am. 2009;91:1360–8.
67. Garr JL, Gentilello LM, Cole PA, Mock CN, Matsen FA 3rd. Monitoring for compartmental syndrome using near-infrared spectroscopy: a noninvasive, continuous, transcutaneous monitoring technique. J Trauma. 1996;46:613–8.
68. Schuler MS, Reisman WM, Kinsey TL, Whitesides TE Jr, Hammerberg EM, Davila MG, Moore TJ. Correlation between muscle oxygenation and compartment pressures in acute compartment syndrome of the leg. J Bone Joint Surg. 2010;92:863–70.

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