JB&JS OPEN ACCESS

Percutaneous Forefoot Decompression in a Foot Compartment Syndrome Model

Drew E. Schupbach, MD*, Mohamad Nasser Eddine, MD*, Yazan Honjol, MD, Geraldine Merle, PhD, and Edward J. Harvey, MD

Investigation performed at McGill University Health Centre Research Institute, Montreal, Quebec, Canada

Background: Acute compartment syndrome of the foot is a controversial topic. Release of the foot has been seen as complicated because of large incisions and postoperative morbidity, and there has been debate over whether this procedure is actually effective for releasing all areas of increased pressure. New sensor technology affords the opportunity to advance our understanding of acute compartment syndrome of the foot and its treatment. The purpose of the present study was to determine whether percutaneous decompression could be performed for the treatment of compartment syndrome in a forefoot model.

Methods: The present study utilized a validated continuous pressure sensor to model compartment syndrome in human cadaveric feet. We utilized a pressure-controlled saline solution infusion system to induce increased pressure. A novel percutaneous release of the forefoot was investigated to assess its efficacy in achieving decompression.

Results: For all cadaveric specimens, continuous pressure monitoring was accomplished with use of a continuous pressure sensor. There were 4 discrete compartment areas that could be reliably pressurized in all feet. The average baseline, pressurized, and post-release pressures (and standard deviations) were 4.5 ± 2.9, 43.8 ± 7.7, and 9.5 ± 3.6 mm Hg, respectively. Percutaneous decompression produced a significant decrease in pressure in all 4 compartments ($p < 0.05$).

Conclusions: With use of continuous compartment pressure monitoring, 4 consistent areas were established as discrete compartments in the foot. All 4 compartments were pressurized with a standard pump system. With use of 2 small dorsal incisions, all 4 compartments were successfully released, with no injuries identified in the cutaneous nerve branches, extensor tendons, or arteries. These results have strong implications for the future of modeling compartment syndrome as well as for guiding clinical studies.

Clinical Relevance: A reproducible and accurate method of continuous pressure monitoring of foot compartments after trauma is needed (1) to reliably identify patients who are likely to benefit from compartment release and (2) to help avoid missed or evolving cases of acute compartment syndrome. In addition, a reproducible method for percutaneous compartment release that minimizes collateral structural damage and the need for secondary surgical procedures is needed.

Acute compartment syndrome (ACS) is a condition in which elevated pressures within a fascial compartment in the body compromise its blood supply, leading to death of the contained tissues[1-3]. The estimated incidence of extremity ACS is 3.1 per 100,000 people/year (representing 1% to 9% of reported lower-extremity fractures), with a strong male predominance[4-6]. The incidence of isolated foot compartment syndrome is highest following crush mechanisms either with (18%) or without (14%) a forefoot fracture. Only 1% of patients with an isolated calcaneal fracture are treated for ACS[7]. Currently, there is no reliable method to diagnose and treat foot compartment syndrome[8,9]. While seemingly a simple concept from a physics standpoint, the clinical presentation, diagnosis, and treatment of ACS present a more complex picture. The diagnosis of compartment syndrome is made primarily on the basis of clinical symptoms of ischemia, with pressure measurement being used as an adjunct. The treatment of ACS usually involves emergency release of the pressure through fasciotomy[10]. In the foot, the most common complications of ACS are persistent neurologic deficits, claw toes,

*Drew E. Schupbach, MD, and Mohamad Nasser Eddine, MD, contributed equally to this work.

Disclosure: The **Disclosure of Potential Conflicts of Interest** forms are provided with the online version of the article (http://links.lww.com/JBJSOA/A349).

JBJS Open Access • 2021:e21.00040. openaccess.jbjs.org

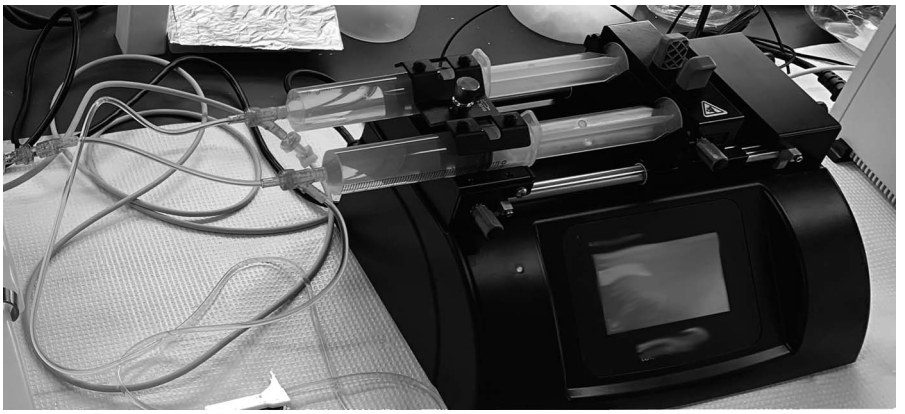

Fig. 1

A PHD ULTRA Syringe Pump Series (Harvard Apparatus) was used with an inline Hugo-Sachs Elecktronik APT300 pressure sensor (Harvard Apparatus).

amputations, and skin-healing problems[6]. However, release of the foot typically is done through large incisions with attendant morbidity[11], and some surgeons perform late tendon releases if needed[5,12,13].

The present study had several aims. First, the study aimed to evaluate the use of new modeling, measurement, and treatment techniques. To that end, recommendations from the American Academy of Orthopaedic Surgeons (AAOS) for continuous pressure monitoring were incorporated into our model[14]. New technological advances in pressure sensors allowing continuous measurements have provided us with the opportunity to better study and potentially manage this condition[15]. Another aim of the study was to define discrete foot compartments for release and to validate a modified minimally invasive release with the use of indwelling continuous pressure sensors[16]. Our hypothesis was that we could successfully model compartment syndrome in cadaveric feet and determine the success of percutaneous decompression.

Materials and Methods

Eight fresh-frozen human cadaveric legs that had been amputated above the knee were allowed to equilibrate at room temperature. The legs were examined to exclude any signs of systemic disease or surgical scars that could suggest compromised anatomy. Bolsters were placed under the knee and ankle to minimize disturbance of the compartments. There is not a clear consensus on the number of compartments in the forefoot, but it is generally accepted that there is a lateral compartment, a central or superficial compartment, a medial compartment, and an adductor compartment; these were the compartments used for the present study. The 4 dorsal interossei can be thought of as separate compartments[17-19] but can be reached from the 2 small incisions described in the Methods section or by adding a third incision between the third and fourth metatarsals.

One infusion line catheter was placed in a proximal-to-distal fashion in each of the 4 forefoot compartments (medial, adductor, superficial/central, lateral). The sensors and lines were placed in the muscle of the compartment but were separated as much as possible according to the limitations of the compartment. The model utilized a pressure-controlled infusion pump and multi-compartment pressure monitoring setup. A PHD ULTRA Syringe Pump Series (Harvard Apparatus) was selected, along with an inline Hugo-Sachs Elecktronik APT300 pressure sensor (Harvard Apparatus) (Fig. 1). The pump was loaded with four 60-mL syringes filled with normal saline solution (0.9% NaCl) and was programmed to adjust the infusion rate to maintain a set pressure as measured by the inline sensor. Syringes were connected with intravenous (IV) tubing that merged into 1 line at the inline pressure sensor before splitting into 4 individual infusion lines with stopcocks and terminating as 14-G catheters (Fig. 2). One sensor device (MY01 Muscle Sensor; MY01) was inserted per the device protocol into each compartment

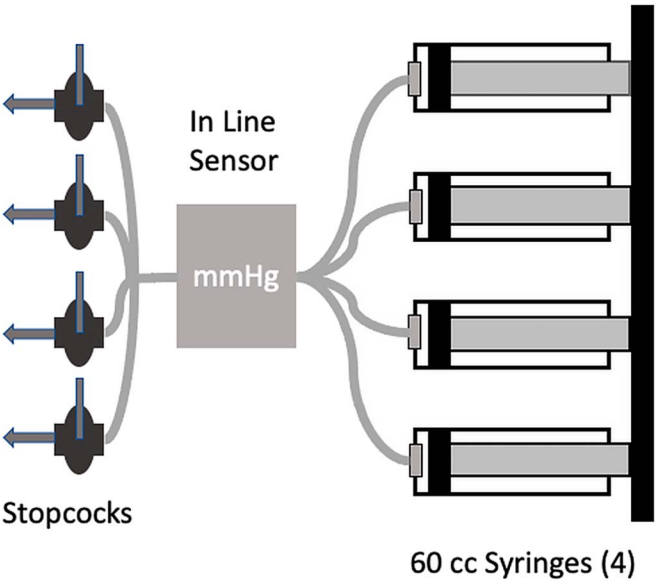

Fig. 2

Syringes were connected with IV tubing that merged into 1 line at the inline pressure sensor before splitting into 4 individual infusion lines with stopcocks and terminating as 14-G catheters.

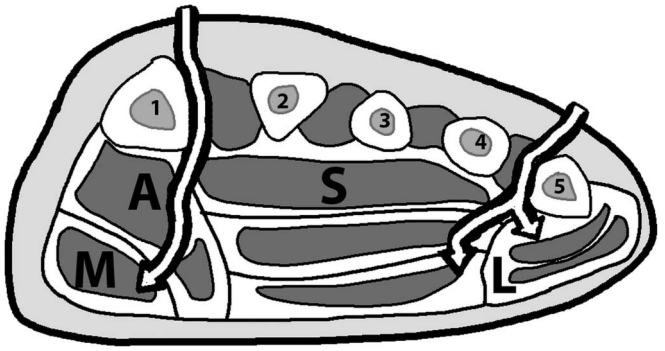

Fig. 3

Release of the 4 compartments through two 1-cm incisions on the dorsum of the foot. The metatarsals are numbered. A medial small incision is placed between the proximal first and second metatarsals, and a lateral incision is placed between the proximal fourth and fifth metatarsals, with each incision centered 4 cm proximal to its respective web space. The arrows show the dissection directions. The 2 dissection planes shown by the arrows allow full release of the 4 compartments. M = medial, A = adductor, S = superficial/central, L = lateral.

centrally. The pressure sensor was zeroed according to the device instructions before each infusion. Continuous pressure monitoring of the compartments was done with use of the MicroElectroMechanical System (MEMS) pressure sensor (MY01). This sensor places the measurement and analysis sensor unit directly in the compartment or muscle to be monitored. Placement of both infusion catheters and sensor tips was confirmed with use of an ultrasound device (Lumify L12-4 Transducer; Philips). The infusion pump and pressure sensor were set to record pressure and volume once per second. Data were recorded continuously by the sensor and continuously during infusion by the pump.

After ultrasound confirmation of placement, baseline pressures were noted, and all compartments were infused until stable pressure was achieved for a minimum of 5 minutes. Sequential infusion illustrated discrete compartments. The pump pressure setpoint was 30 mm Hg[14]. This setpoint was chosen as an extrapolation from lower-leg studies[3,9]. The variance in pressure from pre-infusion to pressurized and then to release was tracked. Some variance with the more accurate clinical sensor[16] being used to monitor compartment pressure was expected. Once all compartments were pressurized, infusion was stopped, and dorsal percutaneous decompression was performed through two 1-cm incisions. A medial incision was made between the proximal first and second metatarsals, and a lateral incision was made between the proximal fourth and fifth metatarsals, with each incision centered 4 cm proximal to its respective web space (Fig. 3). The incisions were made on the lateral side of the first metatarsal and the medial side of the fifth metatarsal. Purple marker dye was used in the incision to mark deep structures in relation to the skin incision in order to allow for the measurement of proximity to any vital structures. Blunt dissection was carried down on the bone to avoid neurovascular damage. Once the dissection plane was carried down to the metatarsal, the interosseous fascia was opened longitudinally, and the interosseous muscle was separated away from the bone along the entire length of the bone with blunt dissection. This created a safe access to the plantar compartments during release of the interossei. Medially, the adductor compartment was palpable, and the medial compartment was encountered as a fascial layer just past the muscle slips. Laterally, release of the central compartment and lateral compartment was accomplished with blunt dissection medially and laterally (Fig. 4). This technique was based on described fasciotomy techniques[18,19], ultrasound-guided dissections, and the clinical experience of the primary investigator. Once pressures had stabilized after release, a surgical dissection was performed to evaluate for any soft-tissue damage and proximity to incisions (Fig. 5). Structures that were identified and examined included cutaneous nerve branches, extensor tendons, and the dorsalis pedis artery. Any identifiable injuries and proximity to vital structures were noted. The medial terminal branch of the deep peroneal nerve at the first interosseous space divides into 2 dorsal digital nerves, which supply the adjacent sides of the toes. An incision was made >1 cm distal to this bifurcation. Before the nerve divides to become digital nerves, it gives off an interosseous branch to the first web space, which supplies the metatarsophalangeal

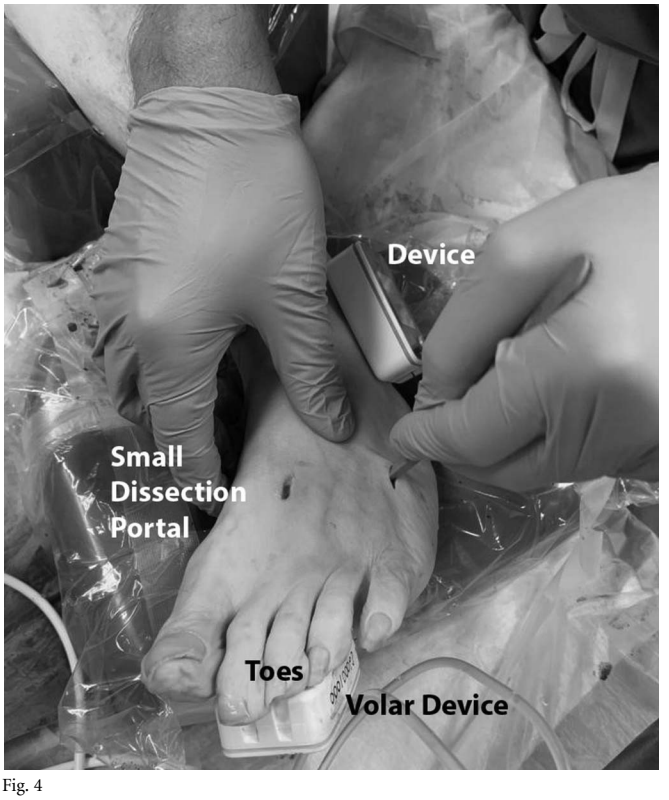

Fig. 4

Performance of forefoot release. Small, 1-cm incisions allowed for release, with a decrease in pressure to normal levels. Two of the 4 devices that were used are visible in this image: 1 on the dorsal side (Device) and 1 on the volar side (Volar Device). The other 2 devices are not visible in this image.

JBJS Open Access • 2021:e21.00040.

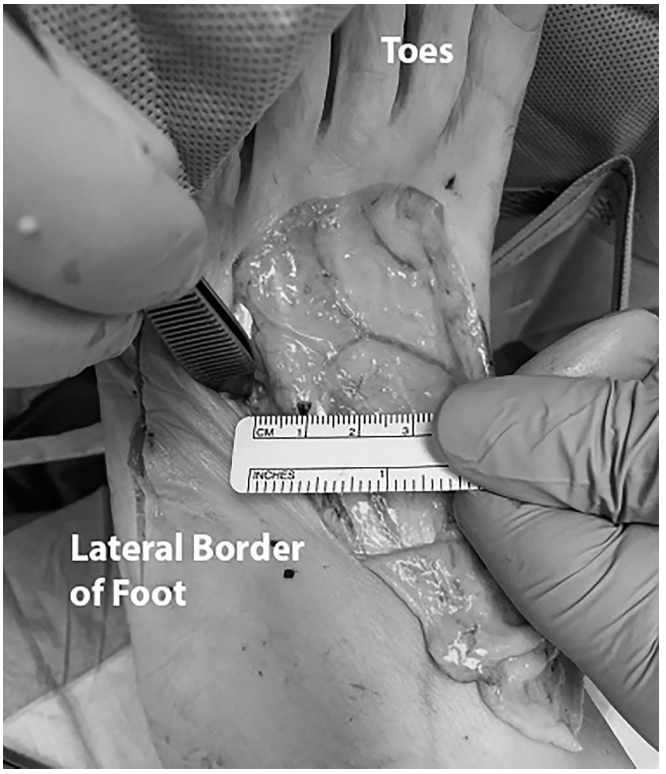

Fig. 5
Post-fasciotomy examination of the release paths to determine if any cutaneous nerve branches, tendons, or arteries were injured.

joint of the great toe and may supply the first interosseous muscle. The surgical dissection was performed between the muscle and the bone and did not affect this nerve. As the nerve was inside the muscle, it was not visible during the dissection and therefore was not recorded.

Baseline, infusion, and post-fasciotomy pressures was reported as the mean and the standard deviation. Significant differences were determined with use of the Student t test, with the level of α set at 0.05. Confidence intervals were reported using 95% limits. Power calculation for nonparametric and parametric testing revealed that 7 specimens were needed if all testing functioned as expected. Eight feet were used in order to have 1 redundant sample. All statistical analyses were performed with use of Microsoft Excel.

Source of Funding
The U.S. Army Medical Research Acquisition Activity, 820 Chandler Street, Fort Detrick MD 21702-5014 is the awarding and administering acquisition office. This work was supported by the Office of the Assistant Secretary of Defense for Health Affairs through the FY18 Defense Medical Research and Development Program, endorsed by the Department of Defense, through the FY18, DMRDP JPC-6/CCCRP Precision Trauma Care Research Award under Award No. W81XWH1920010. Opinions, interpretations, conclusions, and recommendations are those of the authors and are not necessarily endorsed by the Department of Defense.

Results

For all 8 cadaveric specimens, continuous pressure monitoring was accomplished with use of a continuous pressure sensor (MY01). The compartment pressures at baseline, during infusion, and after release corresponded to target values, and the averages are shown in Table I. The average volume infused into each foot (and standard deviation) was 178 ± 35.7 mL. Pressure rises to >30 mm Hg were achieved in the central/superficial compartment, medial compartment, and adductor compartment in all specimens, with return to a physiological baseline after release (average, 9.5 ± 3.6 mm Hg). The pressure measured by the inline sensor on the infusion apparatus was not found to accurately represent the compartment pressure when compared with the intracompartmental MY01 device. This finding is consistent with the literature[16]. However, the infusion pump was able to adjust the infusion rate based on the pressure measured by the inline sensor and to maintain the setpoint pressure during the duration of all infusions. The Pearson correlation coefficient between the setpoint pressure and all pressure measurements across infusion was 0. The differences between the stable infusion pressure and pump setpoint are shown in Table II.

Percutaneous decompression produced a significant decrease in pressure in all 4 compartments to well below compartment syndrome levels in all samples ($p < 0.05$). The average pressure decrease was 34.6 ± 7.7 mm Hg; this decrease was significant ($p < 0.05$). The average post-fasciotomy pressure was 9.5 ± 3.6 mm Hg. Post-fasciotomy dissection did not reveal any injuries of the cutaneous nerve branches, extensor tendons, or dorsalis pedis artery. Proximity measurements revealed that all extensor tendons and arteries were remote from the incisions. Dissection revealed no injuries involving the cutaneous branches, although 3 of 8 terminal branches were within 1 cm of the incision.

TABLE I Average ACS Model Pressures

	Pressure *(mm Hg)*			
Time	Average	Standard Deviation	Maximum	Minimum
Baseline	4.5	2.9	11	1
Infusion	43.8	7.7	60	31
Post-fasciotomy	9.5	3.6	16	0

TABLE II Difference Between Pump Setpoint Pressure and Stable Infusion Compartment Pressure

Pressure *(mm Hg)*		
Setpoint	Average*	Standard Deviation
25	19.1	7.7

*Infusion pressure – setpoint pressure.

Discussion

The ease and reproducibility achieved in this model are promising for future studies seeking to investigate ACS with use of continuous pressure monitoring. As live human models will always be impossible, the necessity of good modeling techniques will remain paramount. Furthermore, the discrepancy between inline pressure measurements and compartment pressure measurements provides insight into devices used in modeling compartment syndrome. This factor did not affect the results of the current study as there was a pressure sensor in each of the compartments. Some studies have utilized an inline pressure sensor as representative of the compartment pressure[20,21]. The results of the present study are consistent with the literature and suggest that pressure readings measured inline with the infusion may not always be representative of the compartment pressure. The discrepancies noted could be reflective of the varying resistance encountered between the inline sensor and the compartment sensor (e.g., IV tubing, muscle tissue). In the present study, the inline sensor was only utilized for the purpose of providing feedback regarding the maintenance of a steady pump infusion.

The successful percutaneous decompression of the forefoot in a cadaver model is promising for the development of less-invasive treatments for compartment syndrome and is the next logical step in trying to avoid complications. Following decompression with use of a standard dorsal approach, the infection rate has been shown to be as high as 20%, with an average of 3 additional procedures being needed to close the wound[22]. Successful percutaneous releases have been described previously in cases of chronic compartment syndrome of the lower leg and when used for single-compartment release[20,23]. Expansion of minimally invasive techniques to the foot was the next logical model.

While the experiments described in the present report were designed to be as robust as possible, the study had several limitations. Modeling compartment syndrome itself is challenging because of its heterogenous presentation and complex physiology. Cadaveric models are limited to the infusion of saline solution or colloid to increase intercompartmental pressure due to their lack of a physiological response. Animal models most commonly utilize ischemia-reperfusion through tourniquet direct pressure or the infusion of saline solution to induce pressure[24,25]. Both methods have shown success in recreating compartment syndrome-level pressures. One of the main problems both clinically and in research is the lack of availability and reliability of pressure-measuring techniques. Recent advances in microfabrication have produced miniaturized sensors that are revolutionizing many different fields, including medicine. A continuous pressure sensor utilizing MEMS technology[16,26] has been approved for clinical use and was utilized in the present study. The lack of a physiologic response in the cadaveric model does impose some limitation on clinical translatability. The cadaveric model is also limited in that it does not allow for the determination of whether swelling will continue after successful release. The absolute danger level for pressure in the foot is yet to be determined, so 30 mm Hg was chosen as a setpoint to allow monitoring of compartment release. Incision placement was determined by 3 surgeon consultants: a resident measured the incision placements, which were then verified by a trauma fellow and then by a trauma staff surgeon before any dissection was carried out. No placements were changed after the initial measurement. Finally, the academic-corporate relationship between the primary investigators and MY01 could introduce a source of bias, but the objective of this project was to create a surgical model, with the sensor results only proving the efficacy of release.

Conclusions

The main objective of the present study was to create a foot model of ACS and to gauge the success of a minimally invasive release. The continuous pressure analysis was able to illustrate successful model. The study demonstrated that the described percutaneous technique can adequately lower pressure within the compartments of the foot. The success of percutaneous release in the foot model bolsters the push toward minimally invasive treatments of ACS. These results have implications for the future of modeling compartment syndrome as well as for guiding clinical studies to improve the management of ACS. ■

Drew E. Schupbach, MD[1]
Mohamad Nasser Eddine, MD[2]
Yazan Honjol, MD[1]
Geraldine Merle, PhD[2,3]
Edward J. Harvey, MD[2,4]

[1]Experimental Surgery, Faculty of Medicine, McGill University, Montreal, Quebec, Canada

[2]Department of Surgery, Division of Orthopedic Surgery, McGill University, Montreal, Quebec, Canada

[3]Department of Chemical Engineering, Polytechnique Montréal, Montreal, Quebec, Canada

[4]Injury Repair and Recovery Program, McGill University Health Centre Research Institute, Montreal, Quebec, Canada

Email for corresponding author: Edward.harvey@mcgill.ca

References

1. Blaisdell FW. The pathophysiology of skeletal muscle ischemia and the reperfusion syndrome: a review. Cardiovasc Surg. 2002 Dec;10(6):620-30.

2. Harvey EJ, Sanders DW, Shuler MS, Lawendy AR, Cole AL, Alqahtani SM, Schmidt AH. What's new in acute compartment syndrome? J Orthop Trauma. 2012 Dec; 26(12):699-702.

3. Schmidt AH. Acute compartment syndrome. Injury. 2017 Jun;48(Suppl 1): S22-5.

4. Perry MD, Manoli A 2nd. Foot compartment syndrome. Orthop Clin North Am. 2001 Jan;32(1):103-11.

5. Perry MD, Manoli A 2nd. Reconstruction of the foot after leg or foot compartment syndrome. Crit Care Nurs Clin North Am. 2012 Jun;24(2):311-22.

6. Lutter C, Schöffl V, Hotfiel T, Simon M, Maffulli N. Compartment Syndrome of the Foot: An Evidence-Based Review. J Foot Ankle Surg. 2019 Jul;58(4):632-40.

7. Thakur NA, McDonnell M, Got CJ, Arcand N, Spratt KF, DiGiovanni CW. Injury patterns causing isolated foot compartment syndrome. J Bone Joint Surg Am. 2012 Jun 6;94(11):1030-5.

8. Gajanan K, Kannan RY, Brotherston MT. Foot compartment syndrome. J Trauma. 2010 May;68(5):E117.

9. Large TM, Agel J, Holtzman DJ, Benirschke SK, Krieg JC. Interobserver variability in the measurement of lower leg compartment pressures. J Orthop Trauma. 2015 Jul;29(7):316-21.

10. Shadgan B, Menon M, Sanders D, Berry G, Martin C Jr, Duffy P, Stephen D, O'Brien PJ. Current thinking about acute compartment syndrome of the lower extremity. Can J Surg. 2010 Oct;53(5):329-34.

11. Manoli A 2nd. Compartment syndromes of the foot: current concepts. Foot Ankle. 1990 Jun;10(6):340-4.

12. Lintz F, Colombier JA, Letenneur J, Gouin F. Management of long-term sequelae of compartment syndrome involving the foot and ankle. Foot Ankle Int. 2009 Sep; 30(9):847-53.

13. Bedigrew KM, Stinner DJ, Kragh JF Jr, Potter BK, Shawen SB, Hsu JR. Effectiveness of foot fasciotomies in foot and ankle trauma. J R Army Med Corps. 2017 Oct;163(5):324-8.

14. Schmidt AH, Osborn CP, Johnson AJ, Balsamo L, Philip M, Gitajn IL, Greer R, Jevsevar D, Samora JB, MacKenzie EJ, Murray J, Mullen K, Sevarino KS, Shores P, Woznica A, DeMars M, Sliwka P, Chang J, Cummins DS. American Academy of Orthopaedic Surgeons Evidence-Based Clinical Practice Guideline on the Management of Acute Compartment Syndrome. American Academy of Orthopaedic Surgeons; 2018. p. 20.

15. Honjol Y, et alet al. Current view and prospect: Implantable pressure sensors for health and surgical care. Med Devices Sens. 2020;3(3):e10068.

16. Merle G, Comeau-Gauthier M, Tayari V, Kezzo MN, Kasem C, Al-Kabraiti F, Laverdiere C, Xereas G, Harvey EJ. Comparison of Three Devices to Measure Pressure for Acute Compartment Syndrome. Mil Med. 2020 Jan 7;185(Suppl 1):77-81.

17. Frink M, Hildebrand F, Krettek C, Brand J, Hankemeier S. Compartment syndrome of the lower leg and foot. Clin Orthop Relat Res. 2010 Apr;468(4): 940-50.

18. Fulkerson E, Razi A, Tejwani N. Review: acute compartment syndrome of the foot. Foot Ankle Int. 2003 Feb;24(2):180-7.

19. Myerson MS. Experimental decompression of the fascial compartments of the foot—the basis for fasciotomy in acute compartment syndromes. Foot Ankle. 1988 Jun;8(6):308-14.

20. Vandergugten S, Zemmour L, Lengelé B, Nyssen-Behets C. A cadaveric model of anterior compartment leg syndrome: Subcutaneous minimally invasive fasciotomy versus open fasciotomy. Orthop Traumatol Surg Res. 2019 Feb; 105(1):167-71.

21. Meier C, Contaldo C, Schramm R, Holstein JH, Hamacher J, Amon M, Wanner G, Trentz O, Menger MD. A new model for the study of the abdominal compartment syndrome in rats. J Surg Res. 2007 May 15;139(2):209-16.

22. Brink F, Bachmann S, Lechler P, Frink M. Mechanism of injury and treatment of trauma-associated acute compartment syndrome of the foot. Eur J Trauma Emerg Surg. 2014 Oct;40(5):529-33.

23. Knight JR, Daniels M, Robertson W. Endoscopic compartment release for chronic exertional compartment syndrome. Arthrosc Tech. 2013 May 23;2(2): e187-90.

24. Kalns J, Cox J, Baskin J, Santos A, Odland R, Fecura S Jr. Threshold model for extremity compartment syndrome in swine. J Surg Res. 2011 May 1;167(1): e13-9.

25. Mortensen SJ, Vora MM, Mohamadi A, Wright CL, Hanna P, Lechtig A, Egan J, Williamson PM, Wixted JJ, Rutkove SB, Nazarian A. Diagnostic modalities for acute compartment syndrome of the extremities: A systematic review. JAMA Surg. 2019 Jul 1;154(7):655-65.

26. Merdassi A, Allan C, Harvey EJ, Chodavarapu VP. Capacitive MEMS absolute pressure sensor using a modified commercial microfabrication process. Microsyst Technol. 2017;23(8):3215-25.