Abstract

Chronic exertional compartment syndrome (CECS) has been hypothesised, following clinical observations, to be the result of abnormal biomechanics predominantly at the ankle. Treatment of CECS through running re-education to correct these abnormalities has been reported to improve symptoms. However no primary research has been carried out to investigate the movement patterns of those with CECS. This study aimed to compare the running kinematics and muscle activity of cases with CECS and asymptomatic controls.

20 men with bilateral symptoms of CECS of the anterior compartment and 20 asymptomatic controls participated. Barefoot and shod running 3D kinematics and muscle activity of the left and right legs; and anthropometry were compared.

Cases displayed less anterior trunk lean and less anterior pelvic tilt throughout the whole gait cycle and a more upright shank inclination angle during late swing (peak mean difference 3.5° , 4.1° and 7.3° respectively). Cases demonstrated greater step length and stance time, although this was not consistent across analyses. There were no consistent differences in Tibialis anterior or Gastrocnemius medialis muscle activity. Cases were heavier (mean difference 7.9kg, p=0.02) than controls with no differences in height (p>0.05)

These differences only partially match the clinical observations previously described. However, no consistent differences were found at the ankle joint suggesting that current running re-education interventions which focus on adjusting ankle kinematics are not modifying pathological aspects of gait. The longer step length is a continuing theme in this population and as such may be a key component in the development of CECS. Keywords: exercise-induced leg pain; chronic exertional compartment syndrome; biomechanics; anthropometry; military training.

Introduction

Chronic exertional compartment syndrome was first described in 1956 [1]. It is an overuse condition presenting as pain in the lower limb, associated with the muscles contained within the myofascial compartments of the shank. The anterior compartment is most frequently affected [2]. While numerous studies have tried to understand the pathophysiology of CECS [3-6], few studies have tried to identify potential risk factors for CECS. The higher reported incidence of CECS in the military compared to civilian practice makes this population ideal for testing potential factors.

CECS is commonly defined as a condition where elevated intramuscular compartment pressure (IMCP) during exercise impedes local blood flow leading to ischaemia and impaired neuromuscular function within the compartment [7,8]. Two systematic reviews recently questioned the role of IMCP and the validity of its use in diagnosis [9,10]. However, we have since reported much improved diagnostic criteria for CECS using continuous IMCP measurement during exercise, thus confirming the intrinsic role of IMCP in this condition [11].

IMCP can be increased through changes in compartment compliance, compartment content or muscle activity [3,12,13]. We recently reported the finding that IMCP in patients is elevated on standing prior to exercise. This suggests that a structural component, presumably increased fascial stiffness, results in reduced compartment compliance [11]. Biomechanical factors have been considered to play a role in the development of CECS for a long time [14]. More recently CECS has been hypothesised, following clinical observations, to be the result of abnormal biomechanics predominantly at the ankle [15]. However, only one other group has investigated the

role of movement patterns and muscle activity in the pathology and aetiology of CECS [16]. This study was focussed on skiing biomechanics and had a very limited sample (n=5 cases); limiting the applicability to the wider population.

Conservative treatment through gait modification has recently been promoted as a viable option for CECS [15,17,18]. Forefoot running was first described as a possible treatment in a case report by Cunningham [19] that may reduce the anterior compartment muscle activity [20] and therefore pain. This has since been followed up by further case reports and a case series of ten US military patients [21-23].

We recently reported the kinematic and kinetic differences between CECS patients and controls during walking and marching [24]. Patients had greater ankle plantarflexion at toe-off and generated lower ankle inversion moments than healthy controls. However, patients typically also complain of pain during running; indeed running is the most common cause of pain within civilians [25]. All of our military patients describe their pain as stopping them from either marching or running; while 30% of these individuals describe pain stopping them from only one of these activities (unpublished data). We therefore aimed to identify the differences in the running biomechanics between patients with CECS and healthy controls.

Methods

20 male cases with symptoms consistent with CECS of the anterior compartment of the leg and 20 asymptomatic controls were recruited. The diagnosis of CECS was established from typical symptoms, with clinical examination and MRI excluding alternative pathologies. Controls were recruited from the UK armed forces. All participants gave informed consent. Cases were recruited from the Lower Limb Pain clinic at the Defence Medical Rehabilitation Centre prior to the provision of any gait advice. Ethical approval was granted by the MOD Research Ethics Committee. The inclusion criteria were: Male; Aged 18-40 (representing the typical age-range of UK military service personnel); BMI<35; and no lower limb length discrepancy >2cm. Cases required the following: symptoms of exercise-induced leg pain consistent with a diagnosis of anterior compartment CECS; a negative MRI of the affected limb(s); no diagnosis other than anterior CECS more likely, and the ability to run for short periods without pain limiting performance. All patients were assessed in a multidisciplinary clinic by a consultant in sport and exercise medicine and senior physiotherapist. This specialist clinic was specifically for patients presenting with exercise induced leg pain. Detailed history taking, including direct questioning and physical examination were used to determine the exact localisation of the patients' pain. This often included a symptom provocation test on a treadmill. Patients were only included in the study if their symptoms were purely localised to the anterior myofascial compartment. Controls were included when they were able to run for at least 20 minutes and had: no lower limb pain in the previous 12 months; no current pain at any site, including during exercise activities; and no reliance on orthotics.

Measurements of leg length, height and body mass were performed using a tape measure, stadiometer (SECA, UK) and medical grade scales (SECA, UK) respectively.

Kinematics and electromyography

Retro-reflective markers were placed on specific anatomical landmarks to form 15 body segments including the feet, shank, thigh, pelvis, trunk, head, upper arm, forearm and hand by the same operator. The head, upper arm, forearm and hand were not analysed as part of this study; these were not considered further. Data were collected using a 10 camera (4xT160, 4xT40-S, 2xT10) 3D motion analysis system (Vicon MX system, Oxford Metrics Ltd., Oxford, England) at a sampling frequency of 120 Hz. A static calibration trial was first collected.

Participants walked barefoot on the treadmill for familiarisation, once happy the participant directed a member of the research team to increase the speed until they were at a comfortable running pace that they felt could be sustained for 15-30 minutes under normal circumstances. Once at the chosen speed this was maintained for a further 2 minutes. Only the final minute was used for analysis in order to allow gait to normalise to the running environment as much as possible. Five trials of five seconds of data were collected at five-second intervals in accordance with a similar previous study [26]. This process was repeated with participants provided with military issue training shoes (Hi-Tec Silver Shadow). Orthotics were not used during testing. A recorded trial was deemed suitable if it had minimal marker dropout and no major gait inconsistency on the part of the subject as judged by an observer, e.g. stopping or stumbling.

The pelvis and thigh segments were defined according to Wu [27], the shank segments were defined according to Peters [28] and tracked using the marker cluster recommended by Manal [29], the feet segments were a modified version of the foot flat option defined by Pratt [30]. The thorax was defined according to Gutierrez [31].

Electromyographic (EMG) data were collected using 4 wireless Trigno (Delsys Inc., Boston, MA, USA) sensors (16-bit Resolution; four 5mm x 1mm silver contacts; fixed 10mm inter electrode distance) at a sampling frequency of 1200Hz. Hair was removed from the EMG testing locations using a surgical razor. In order to reduce skin impedance, the skin was cleaned using an alcohol wipe and lightly rubbed so that the skin went light red [32]. EMG activity of the Tibialis anterior and Gastrocnemius medialis were recorded bilaterally during all movement trials and sensors placed according to the guidelines by Sacco [33] and the SENIAM project [34]. The Tibialis anterior EMG sensor locations were determined by measuring 1/3 of the way along a line drawn from the fibular head to the medial malleolus. The Gastrocnemius medialis was defined as halfway along a line drawn from the medial margin of the popliteal fossa to the medial insertion of the Achilles tendon at the calcaneus. In all cases the lower left corner of the electrode was placed at this point in order to improve standardisation. The sensors were attached using double sided adhesive tape along the longitudinal axis of the muscle. Correct placement and satisfactory signal quality was confirmed by performance of a maximum voluntary contraction of the individual muscle with observation of changes on the monitor in line with guidelines provided by Hislop [35].

Data processing and statistical analysis

Gaps smaller than 14 frames in the raw marker data were interpolated using a 3rd order least squares fit [36]. In the case of larger gaps the whole segment was excluded from analysis at these time points. The marker data was then filtered using a 6Hz low pass bidirectional Butterworth filter [37]. Gait data were normalised to leg length as recommended by Hof [38] and Pierrynowski [39].

Kinematic and kinetic data were normalized to 100% of the gait cycle and stance phase respectively. Bootstrapped t-tests on each individual normalised time point were carried out to identify regions within the gait cycle that were significantly different [40].

A custom MATLAB (R2015b,Mathworks, Natick,MA) script was used to carry out a wavelet analysis [41], normalise the result to 100% of the gait cycle, and perform bootstrap t-tests on each wavelet and time point to identify regions within the gait cycle that were significantly different [40]. A wavelet analysis allows the power of the EMG signal to be described in both the frequency and time domain. The R statistical software (v3.2.2, The R Foundation for Statistical Computing, Vienna, Austria) script published by Armstrong [42] was called by MATLAB to calculate the intensity of 11

wavelets using the EMG-specific parameters defined by von Tscharner [41]. The centre-frequency of these wavelets ranged from 7Hz to 395Hz.

Running speed was self-selected by the participants. Consequently there were variations in speed between participants. ANCOVA was therefore used to cross-check that controlling for the variations in speed would not alter the interpretation of the original analyses. Alpha for all analyses was set to 0.05. SPSS (v18; SPSS Inc, USA) or Matlab (v2014a; MathWorks, USA) were used for all analyses.

Results

Cases ranged in age between 18-37 (M=27.5, sd=5.2); controls between 18-36 (M=25.0, sd=6.1). No significant pain was reported during testing.

Cases (M=84.1kg, sd=10.0kg) were heavier (p=0.02) than controls (M=76.2kg, sd=11.4kg). There were no significant differences in height (Cases: M=1.77m, sd=0.05m; controls: M=1.79m, sd=0.09m). Cases (M=26.9, sd=2.7) also had a larger body mass index (BMI; p<0.001) than controls (M=23.6, sd=2.5).

Self-selected speed was slightly faster (p<0.05) for cases (M=11.0 kph, SD=1.1 kph) than controls (M=10.1 kph, SD=1.2 kph). When speed was controlled for, no consistent differences were seen in the temporal-spatial variables. As such there were no consistent differences in normalised step time, stance time, swing time or flight time. The faster speed necessitated a significantly longer step length (relative to leg length), as shown in the uncorrected data, for cases. This difference was generally no longer significant when speed was controlled for; although was still significant in the left shod condition. These differences are summarised in Table 1.

Normalisation to the gait cycle resulted in 101 individual tests for each movement plane where heel strike (HS) occurs at time points 0 and 100 and toe-off occurs between 32-35% of the gait cycle for both the barefoot and shod conditions. All participants had initial contact with the heel. The mean position of Toe-Off (TO) of 33% is marked on all gait curves. Key kinematic variables are presented graphically highlighting regions of data that differ significantly (p<0.05) between the two groups (Figure 1).

Cases displayed less anterior trunk lean and less anterior pelvic tilt throughout the whole gait cycle. During the late swing phase, cases had a more upright shank inclination angle. There was also some evidence that cases had less hip flexion during early swing phase (and terminal stance); although these differences were not apparent when speed was controlled for. A summary of the significant differences for kinematic data is presented in Table 2. Four different combinations of experimental condition were defined (Left / Right; Barefoot / Shod).

In view of the consistency of the results (with the exception of the hip angle) reported in Table 2, graphs of the original data (i.e. unadjusted for speed) are shown (Figure 1). Graphs for the left-sided shod condition are shown as there were no differences between left and right-sided data. The magnitudes of the differences in angular measurements are presented in Table 2 and summarised in Figure 2.

There were no consistently significant differences in any of the wavelet intensities or time points of the gait cycle for Tibialis anterior or Gastrocnemius medialis (Figure 3).

Discussion

This study demonstrates a number of key differences in biomechanical variables between CECS cases and asymptomatic controls. Anthropometric findings contrast with our recent study that demonstrated that cases in that cohort were on average 10cm shorter than controls; with no significant differences in mass [11] or BMI (unpublished data). As such, we have previously suggested that smaller stature may be a risk factor for CECS in the military. The results of the current study suggest that a prospective study is now needed to provide more robust data on this theory.

Our findings are the first to demonstrate that patients with CECS run with a different gait pattern to asymptomatic controls. The results complement the differences found in our earlier study [24] demonstrating that CECS patients have different ankle mechanics during walking and marching; but no differences occur further up the kinetic chain. The localisation of these differences to the planes of motion controlled by Tibialis anterior suggested that this muscle is functionally disadvantaged in these patients. However, during running, the main differences occur at the trunk with these differences then appearing to be propagated but diluted down the kinetic chain. It is feasible that the reduced requirements of Tibialis anterior during running. Studies directly testing the strength, endurance and mechanical properties of Tibialis anterior are therefore required. It is also possible that there is a separate aetiology for CECS developing for an athletic/running population versus a fast walking/marching population. Comparisons between the biomechanics of civilian and military populations are therefore required.

Modifications to running style that have been used in an attempt to reduce the anterior compartment muscle activity of CECS patients have typically encouraged greater forward lean; along with changing from a heel strike to a forefoot strike, increasing cadence and reducing step length [18]. Clinical observations have also suggested that patients have abnormally high levels of ankle dorsiflexion throughout the gait cycle and 'reduced heel lift during swing phase' [15]. While a reduced heel lift was observed during swing phase in the current study (differences in shank inclination), evidence for any differences at the ankle joint was not found. Similarly there was no evidence of

altered ankle muscle activity. This is surprising as an expected consequence of less heel lift would be increased ankle dorsiflexion during the swing phase to allow foot clearance.

The similarity in ankle joint kinematics observed between cases and controls suggests that it is unlikely that running technique alone is the sole cause of CECS development. Thus current running re-education interventions which focus on adjusting ankle kinematics do not focus on modification of pathological aspects of gait; yet have been reported to reduce CECS complaints in soldiers for up to one year [11]. Given the reports of reduced anterior compartment muscle activity in forefoot runners in healthy populations [44], these interventions may simply be an option for activity modification that has not previously been explored. A resulting reduction in anterior compartment muscle activity would also explain the differences in IMCP observed after exercise following the intervention [21,45]. The ability of these interventions to provide a lasting solution to military personnel that also need to regularly walk and march at a fast pace while carrying load is unclear.

The lack of angular differences at the ankle joint suggests that there is no difference in mechanical load of the anterior compartment musculature (and therefore fascia through myofascial force transmission) during running in this population. This is confirmed to some extent with the EMG analyses. Military personnel typically experience pain that causes them to stop both marching and running. Although the pain from CECS is more prevalent and of greater intensity during marching than running activities [46]. Replication of these results within the civilian athletic population is therefore warranted.

The temporal-spatial results in the current study are very similar to those reported in our walking/marching study [24]. The increased stride length (relative to leg length) during walking/marching was suggested to reflect ingrained changes induced by military training; whereby all personnel are required to move at a uniform cadence and speed. During running, cases also ran with a significantly longer step length (relative to leg length), although this was only significant in the left shod condition when speed was controlled for. Ingrained movement strategies are believed to explain the differences in gait seen between trained distance runners and sprinters running at the same speed [47]. We believe that a similar mechanism is occurring in this population whereby the increased stride length ingrained during walking and marching becomes translated into running gait.

Three prior studies comparing body mass and/or BMI between cases and controls have reported mixed findings [48-50]. The reasons for this are not clear, although some of the differences may be due to study design. For example, the comparisons in two of these studies may not be entirely valid as they were made between groups that consisted of both male and female subjects [48,49]. The study by Varelas [50] is also not directly comparable as they recruited an all-female cohort [50]. It is unclear whether the greater body mass observed in military studies is a result of deconditioning following the development of CECS or a risk factor for the condition itself.

One inherent limitation of our study was the inclusion of an all-male sample that might limit its translation to females. Cases were selected based on a clear clinical history rather than IMCP measurement due to strong evidence that IMCP testing had poor diagnostic validity at the start of this study [9]. A recent study now reports that IMCP can only provide an accurate diagnosis when it is measured during exercise to the limits of pain tolerance [11]. Future studies would therefore ideally use this new diagnostic method for case selection. The differences in speed selected also could have affected the results. It was therefore reassuring that both the bootstrapped t-tests and ANCOVA tests gave predominantly the same results. In order to prevent the onset of pain during testing, the familiarisation period at the selected speed could not be as long as generally recommended. However, even after just two minutes of familiarisation, the measurements can be considered to have high reliability [51]. In light of this, and due to the constancy of this period for all participants, we do not believe that this would have adversely affected our results. However this condition did not allow the investigation of the biomechanics of muscle activity during a longer time period and after the onset of fatigue when the development of this condition may be expected to occur. Finally, to differentiate between cause and association it is acknowledged that these results would ideally be confirmed in a prospective longitudinal study.

In summary, this study demonstrates differences in the running biomechanics of cases with CECS that are present prior to the onset of notable symptoms. These differences match to some extent the clinical observations previously described [15]. However the lack of differences in ankle kinematics and anterior compartment muscle activity suggests that current running re-education interventions do not focus on modifying pathological aspects of gait. The increased step length is a continuing theme in this population and as such we believe this may be a key component in the development of the condition.

References

[1] Mavor GE. The anterior tibial syndrome. J Bone Joint Surg Br 1956;38-B2:513-7.
[2] Reneman RS. The anterior and the lateral compartmental syndrome of the leg due to intensive use of muscles. Clin Orthop Relat Res 1975;(113)113:69-80.

[3] Turnipseed WD, Hurschler C, Vanderby R,Jr. The effects of elevated compartment pressure on tibial arteriovenous flow and relationship of mechanical and biochemical characteristics of fascia to genesis of chronic anterior compartment syndrome. J Vasc Surg 1995;215:810,6; discussion 816-7.

[4] Hurschler C, Vanderby R, Jr, Martinez DA, Vailas AC, Turnipseed WD. Mechanical and biochemical analyses of tibial compartment fascia in chronic compartment syndrome. Ann Biomed Eng 1994;223:272-9.

[5] Barbour TD, Briggs CA, Bell SN, Bradshaw CJ, Venter DJ, Brukner PD. Histology of the fascial-periosteal interface in lower limb chronic deep posterior compartment syndrome. Br J Sports Med 2004;386:709-17.

[6] Evers B, Odemis V, Gerngross H. Intramuscular oxygen partial pressure in patients with chronic exertional compartment syndrome (CECS). Adv Exp Med Biol 1997;428:311-6.

[7] Zhang Q, Jonasson C, Styf J. Simultaneous intramuscular pressure and surface electromyography measurement in diagnosing the chronic compartment syndrome. Scand J Med Sci Sports 2011;212:190-5.

[8] Styf J, Korner L, Suurkula M. Intramuscular pressure and muscle blood flow during exercise in chronic compartment syndrome. J Bone Joint Surg Br 1987;692:301-5.

[9] Roberts A, Franklyn-Miller A. The validity of the diagnostic criteria used in chronic exertional compartment syndrome: A systematic review. Scand J Med Sci Sports 2012;225:585-95.

[10] Aweid O, Del Buono A, Malliaras P, Iqbal H, Morrissey D, Maffulli N, Padhiar N. Systematic review and recommendations for intracompartmental pressure monitoring in diagnosing chronic exertional compartment syndrome of the leg. Clin J Sport Med 2012;224:356-70.

[11] Roscoe D, Roberts AJ, Hulse D. Intramuscular compartment pressure measurement in chronic exertional compartment syndrome: new and improved diagnostic criteria. Am J Sports Med 2015;432:392-8.

[12] Hile AM, Anderson JM, Fiala KA, Stevenson JH, Casa DJ, Maresh CM. Creatine supplementation and anterior compartment pressure during exercise in the heat in dehydrated men. J Athl Train 2006;411:30-5.

[13] Sporrong H, Styf J. Effects of isokinetic muscle activity on pressure in the supraspinatus muscle and shoulder torque. J Orthop Res 1999;174:546-53.

[14] Bates P. Shin splints--a literature review. Br J Sports Med 1985;193:132-7.

[15] Franklyn-Miller A, Roberts A, Hulse D, Foster J. Biomechanical overload syndrome: defining a new diagnosis. Br J Sports Med 2014;486:415-6.

[16] Federolf P, Bakker E. Muscle activation characteristics in cross-country skiers with a history of anterior compartment pain. Sports Biomech 2012;114:452-63.

[17] Franklyn-Miller A. Running Re-education. Aspetar Sports Medicine Journal 2014;32.

[18] Helmhout PH, Diebal AR, van der Kaaden L, Harts CC, Beutler A, Zimmermann WO. The Effectiveness of a 6-Week Intervention Program Aimed at Modifying Running Style in Patients With Chronic Exertional Compartment Syndrome Results From a Series of Case Studies. Orthopaedic Journal of Sports Medicine 2015;33:2325967115575691.

[19] Cunningham A, Spears IR. A successful conservative approach to managing lower leg pain in a university sports injury clinic: a two patient case study. Br J Sports Med 2004;382:233-4.

[20] Jerosch J, Geske B, Castro WH, Hille E. Compartment pressure of the anterior tibial area in jogging. Z Orthop Ihre Grenzgeb 1989;1271:56-64.

[21] Diebal AR, Gregory R, Alitz C, Gerber JP. Forefoot running improves pain and disability associated with chronic exertional compartment syndrome. Am J Sports Med 2012;405:1060-7.

[22] Diebal AR, Gregory R, Alitz C, Gerber JP. Effects of forefoot running on chronic exertional compartment syndrome: a case series. Int J Sports Phys Ther 2011;64:312-21.

[23] Gibson AR. Chronic Exertional Compartment Syndrome and Forefoot Striking: A Case Study. Int J Athl Ther Train 2013;186:24-6.

[24] Roberts AJ, Roscoe D, Hulse D. Biomechanical differences between cases and controls with chronic exertional compartment syndrome and asymptomatic controls during walking and marching. 2016.

[25] Detmer DE, Sharpe K, Sufit RL, Girdley FM. Chronic compartment syndrome: diagnosis, management, and outcomes. Am J Sports Med 1985;133:162-70.

[26] Loudon JK, Reiman MP. Lower extremity kinematics in running athletes with and without a history of medial shin pain. Int J Sports Phys Ther 2012;74:356-64.

[27] Wu G, Siegler S, Allard P, Kirtley C, Leardini A, Rosenbaum D, Whittle M, D'Lima DD, Cristofolini L, Witte H, Schmid O, Stokes I. ISB recommendation on definitions of joint coordinate system of various joints for the reporting of human joint motion--part I: ankle, hip, and spine. J Biomech 2002;354:543-8.

[28] Peters A, Sangeux M, Morris ME, Baker R. Determination of the optimal locations of surface-mounted markers on the tibial segment. Gait Posture 2009;291:42-8.

[29] Manal K, McClay I, Stanhope S, Richards J, Galinat B. Comparison of surface mounted markers and attachment methods in estimating tibial rotations during walking: an in vivo study. Gait Posture 2000;111:38-45.

[30] Pratt EJ, Reeves ML, van der Meulen JM, Heller BW, Good TR. The development, preliminary validation and clinical utility of a shoe model to quantify foot and footwear kinematics in 3-D. Gait Posture 2012;363:434-8.

[31] Gutierrez EM, Bartonek A, Haglund-Akerlind Y, Saraste H. Centre of mass motion during gait in persons with myelomeningocele. Gait Posture 2003;182:37-46.

[32] Konrad P. The ABC of EMG. 2005.

[33] Sacco IC, Gomes AA, Otuzi ME, Pripas D, Onodera AN. A method for better positioning bipolar electrodes for lower limb EMG recordings during dynamic contractions. J Neurosci Methods 2009;1801:133-7.

[34] Stegeman D, Hermens H. Standards for surface electromyography: the european project "surface emg for non-invasive assessment of muscles (seniam). Línea).Disponible en: http://www.med.uni-jena.de/motorik/pdf/stegeman.pdf [Consultado en agosto de 2008] 2007.

[35] Hislop H, Montgomery J. Daniels and Worthingham's Muscle Testing: Techniques of Manual Examination. 6th ed. Philadelphia: WB Saunders; 1995.

[36] Howarth SJ, Callaghan JP. Quantitative assessment of the accuracy for three interpolation techniques in kinematic analysis of human movement. Comput Methods Biomech Biomed Engin 2010;136:847-55.

[37] Winter DA, Sidwall HG, Hobson DA. Measurement and reduction of noise in kinematics of locomotion. J Biomech 1974;72:157-9.

[38] Hof AL. Scaling gait data to body size. Gait Posture 1996;43:222-3.

[39] Pierrynowski MR, Galea V. Enhancing the ability of gait analyses to differentiate between groups: scaling gait data to body size. Gait Posture 2001;133:193-201.

[40] Lenhoff MW, Santner TJ, Otis JC, Peterson MG, Williams BJ, Backus SI. Bootstrap prediction and confidence bands: a superior statistical method for analysis of gait data. Gait Posture 1999;91:10-7.

[41] von Tscharner V. Intensity analysis in time-frequency space of surface myoelectric signals by wavelets of specified resolution. J Electromyogr Kinesiol 2000;106:433-45.

[42] Armstrong WJ, Beck TW, Welch JD, Borg F. Technical Note: Clinical Application of the Intensity Analysis Using the R Open Source Software. Clinical Kinesiology (Online Edition) 2011;653.

[43] Hreljac A. Determinants of the gait transition speed during human locomotion: kinematic factors. J Biomech 1995;286:669-77.

[44] Yong JR, Silder A, Delp SL. Differences in muscle activity between natural forefoot and rearfoot strikers during running. J Biomech 2014;4715:3593-7.

[45] Hargens AR, Parazynski S, Aratow M, Friden J. Muscle changes with eccentric exercise: implications on Earth and in space. Adv Myochem 1989;2:299-312.

[46] Verleisdonk EJ, Schmitz RF, van der Werken C. Long-term results of fasciotomy of the anterior compartment in patients with exercise-induced pain in the lower leg. Int J Sports Med 2004;253:224-9.

[47] Bushnell T, Hunter I. Differences in technique between sprinters and distance runners at equal and maximal speeds. Sports Biomech 2007;63:261-8.

[48] Birtles DB, Minden D, Wickes SJ, M Puxley KP, A Llewellyn MG, Casey A, Rayson MP, Jones DA, Newham DJ. Chronic exertional compartment syndrome: muscle changes with isometric exercise. Med Sci Sports Exerc 2002;3412:1900-6.

[49] Rorabeck CH, Bourne RB, Fowler PJ, Finlay JB, Nott L. The role of tissue pressure measurement in diagnosing chronic anterior compartment syndrome. Am J Sports Med 1988;162:143-6.

[50] Varelas FL, Wessel J, Clement DB, Doyle DL, Wiley JP. Muscle function in chronic compartment syndrome of the leg. J Orthop Sports Phys Ther 1993;185:586-9.

[51] Lavcanska V, Taylor NF, Schache AG. Familiarization to treadmill running in young unimpaired adults. Hum Mov Sci 2005;244:544-57.