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Achilles tendinopathy alters stretch shortening cycle behaviour during a sub-maximal hopping task

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Abstract

Objectives

To describe stretch shortening cycle behaviour of the ankle and lower limb in patients with Achilles tendinopathy (AT) and establish differences with healthy volunteers.

Design

Between-subjects case-controlled

Methods

Fifteen patients with AT (mean age 41.2 ± 12.7 years) and eleven healthy volunteers (CON) (mean age 23.2 ± 6.7 years) performed sub-maximal single-limb hopping on a custom built sledge-jump system. Using 3D motion analysis and surface EMG, temporal kinematic (lower limb stiffness, ankle angle at 80 ms pre-contact, ankle angle at contact, peak ankle angle, ankle stretch amplitude) and EMG measures (onset, offset and peak times relative to contact) were captured. Data between AT and CON were compared statistically using a linear mixed model.

Results

Patients with AT exhibited significantly increased lower limb stiffness when compared to healthy volunteers ($p<0.001$) and their hopping range was shifted towards a more dorsiflexed position ($p<0.001$). Furthermore, ankle stretch amplitude was greater in AT compared with healthy volunteers ($p<.001$). A delay in muscle activity was also observed; soleus onset ($p<.001$), tibialis anterior peak ($p=0.026$) and tibialis anterior offset ($p<.001$) were all delayed in AT compared with CON.

Conclusions

These findings indicate that patients with AT exhibit altered stretch-shortening cycle behaviour during sub-maximal hopping when compared with healthy volunteers. Patients with AT hop with greater lower limb stiffness, in a greater degree of ankle dorsiflexion and have a greater stretch amplitude. Likewise, delayed muscle activity is evident. These findings have implications in terms of informing the understanding of the pathoaetiology and management of AT.

Keywords

Achilles tendon

Tendinopathy

Overuse injury

Plyometric exercise

Stretch shortening cycle

Hopping

Main Text

Introduction

Achilles tendinopathy (AT) is a common clinical syndrome experienced by active individuals, characterised by a combination of pain, diffuse or localised swelling, and impaired performance arising from overuse. Whilst AT is a challenging condition to manage and evidence supports a conservative approach [1], frequent reports of sub-optimal clinical outcomes (e.g. van der Plas, de Jonge, de Vos, van der Heide, Verhaar, Weir, Tol [2]) suggest our understanding of the condition is incomplete.

The pathoaetiology of AT is complex [3] and whilst multiple factors clearly interact in the development of AT, mechanical factors dominate where the cumulative load placed upon the tendon exceeds its mechanical capacity, resulting in a 'failed loading response' [4]. Given the central role that the Achilles tendon plays in the stretch-shortening cycle (SSC), it seems reasonable that aberrations in SSC behaviour and AT may be related.

The stretch shortening cycle (SSC) is a phenomenon that describes the natural pre-activation of a musculotendinous unit, followed by an eccentric phase and a subsequent concentric phase [5]. Its role is to simplify the motor control of locomotion and optimise locomotor efficiency [6]. It has been suggested that aberrations in SSC performance may result in injury [7] and evidence exits describing how AT is associated with alterations in measures indicative of altered SSC behaviour [8-10]. Unfortunately, these studies collectively provide an incomplete picture of the biomechanical changes that occur in the presence of Achilles tendinopathy. In particular kinematic evaluation of the ankle in the sagittal plane, and the associated activity of key agonist/antagonist muscles during a SSC task have yet to be explored in this population. Given that the principal plane of motion for the SSC during running is sagittal, a greater depth of understanding at this level would provide further insight into the relationship between AT and SSC behaviour.

In this study we compared SSC behaviour during sub-maximal single limb hopping in individuals with AT and in a group of healthy volunteers (CON). We hypothesised that in the AT group

alterations in SSC behaviour would be observed. Specifically, we hypothesised that when compared with CON, the AT group would exhibit decreased lower limb stiffness, hop in a greater degree of dorsiflexion and have a greater stretch amplitude. Likewise, we hypothesised that in AT, delayed muscle activity in both the agonist and antagonist muscles would be observed. To test these hypotheses, we measured SSC behaviour during a sub-maximal hopping task on a sledge-jump system (SJS). The utility of such a system is its capacity to limit variability of movement and mitigate fatigue; such systems have been used in the past to explore a variety of conditions influencing SSC behaviour [11].

Achilles tendinopathy is a common injury and challenging to treat in part due to our incomplete understanding of the pathoaetiological drivers of the condition. The findings of this study may have connotations in both deepening our understanding of the mechanical pathoaetiology of AT, and potentially informing the development and refinement of therapeutic interventions for AT.

Methods

This study employed a between-subjects case-controlled design and included 15 patients with AT (mean age 41.2 ± 12.7 years; 9 male: 6 female; affected side 4 left: 11 right) and 11 CON volunteers (mean age 23.2 ± 6.7 years; 5 male: 6 female). CON volunteers were recruited from the local university community and AT patients were recruited from local medical practices in Perth, Western Australia. AT inclusion criteria included a >3 month history of unilateral mid-portion Achilles tendon pain, a VISA-A score <80/100, with mid-portion pain and thickening identified on palpation. Exclusion criteria for both groups included an absence of co-existing lower quadrant musculoskeletal pathology or other visual/motor impairment(s). Informed consent approved by the Human Research Ethics Committee of Curtin University was obtained from all participants prior to testing (HR28/2010).

Retro-reflective markers were fixed to the skin of participants according to a customised marker set and model for the lower quadrant (see Figure 1b), set according to an established cluster-based method [12]. This established set-up enabled determination of anatomically-relevant ankle, knee and hip

joint axes of rotation and joint centres [12], and subsequent motion capture was performed using a 14-camera Vicon MX motion analysis system (Vicon, Oxford Metrics, Oxford, UK) operating at 250 Hz.

Temporal measures of soleus and tibialis anterior muscle activity were recorded using an AMTI-8 (Bortec Biomedical Ltd) surface EMG (sEMG) system. Bipolar differential surface electrodes (Ag / AgCL) were placed on the belly of each muscle with the reference electrode on the medial malleolus. Skin impedance (< 15kOhms) was achieved with skin preparation and signals were pre-amplified, analogue filtered (10 – 500Hz band pass) and then digitised using an 18 bit A-D card with a sampling rate of 1000Hz. All data was temporally synchronised and recorded on dedicated hardware running a customised Labview program (National Instruments, Austin, Texas, 2011).

Participants attended on a single testing occasion. They were instructed to continue with their normal everyday activities but to refrain from undertaking unfamiliar activities in the week prior to testing. In addition, they were instructed to avoid vigorous physical activity in the 24 hour period prior to data collection. AT participants were further instructed to not receive novel therapeutic interventions in the 2 weeks prior to testing.

Participants were instructed on the performance of sub-maximal hopping on a custom-built SJS (see Figure 1a). The task involved continuous sub-maximal single-limb hopping on the SJS keeping the knee fully extended; participants hopped on their affected (AT) or dominant (CON) limb for 15 seconds trials, before a 30 s rest period. Five trials were repeated.

Data was processed using Vicon Nexus motion analysis software (Vicon, Oxford Metrics, Oxford, UK). Kinematic data were filtered using a fourth order Butterworth filter operating at a frequency cut-off of 20 Hz for the marker trajectories and 50 Hz for the ground contact data as determined by residual analysis [13]. All lower limb anatomical and joint coordinates were calculated in accordance with the standards outlined by the International Standards of Biomechanics and have been previously described [12]. Data was exported from Nexus for further analysis using a customised LabVIEW program (National Instruments, Austin, Texas, 2011). For each hopping trial, the following ankle kinematic measures were calculated; ankle angle 80 ms prior to ground contact, ankle angle at ground

contact, peak ankle angle and ankle stretch amplitude. In addition, lower limb stiffness was calculated using the method described by Dalleau, Belli, Viale, Lacour, Bourdin [14] (figure 1c). In addition, temporal measures of muscle activity for soleus and tibialis anterior were calculated relative to ground contact; onset, peak and offset.

The EMG signal was full wave rectified and onsets detected using an integrated protocol [15]. Trial linear envelopes (LE) were created using a fourth-order, zero-lag Butterworth low-pass filter (10 Hz) and temporally synchronised to (T=0) foot contact.

Statistical analysis was conducted using SPSS version 20 (SPSS, Chicago, Il, USA). Descriptive statistics were used to establish mean values for all variables in each group (AT vs. CON). A linear mixed model was used for all statistical comparisons between groups. Age, gender height and body mass were input as covariates and adjusted for within the model. A fixed main effects model was fitted, with a type III sum of squares used to assess statistical significance. Parameter estimates were utilised, and main effects were compared as pairwise comparisons using a Bonferoni correction. The residuals were tested for normality as required by the linear mixed model.

Results

Mean (and standard deviation) values for our biomechanical measures are presented in Table 1. Patients with AT exhibited increased lower limb stiffness when compared to CON ($p<0.001$) and their hopping range was shifted towards a more dorsiflexed position ($p<0.001$). Ankle stretch amplitude was greater in AT compared with CON ($p<.001$). A delay in muscle activity was observed in soleus onset ($p<.001$), tibialis anterior peak ($p=0.026$) and tibialis anterior offset ($p<.001$) in AT compared with CON

Discussion

This is the first study to describe SSC-behaviour during a sub-maximal hopping task in patients with AT with a detailed focus on sagittal plane behaviour. AT is a common injury whose pathoaetiology is unclear and as a result management remains sub-optimal. Whilst we have some understanding of the changes in SSC behaviour that correspond with the pathology, our understanding of sagittal plane

SSC behaviour has been to this point somewhat limited. In this comparative study, we found that when compared to healthy volunteers, individuals with AT exhibit altered SSC behaviour during a sub-maximal hopping task. This has been demonstrated in the following ways. Firstly, individuals with AT hopped with increased lower limb stiffness. In addition they hopped in greater dorsiflexion, and with greater overall stretch amplitude. We also found that soleus onset, tibialis anterior peak, and tibialis anterior offset timing is delayed in AT (see Table 1). Whilst changes in SSC behaviour has been investigated in AT [8-10], this is the first study that has isolated SSC performance to the ankle in such a manner that has enabled detailed examination of sagittal plane ankle behaviour in this manner.

Contrary to our hypothesis and the existing literature, lower limb stiffness was increased in AT. Lower limb stiffness has only been previously measured in individuals with AT on limited occasions, and in all studies stiffness was found to be reduced. For example, Maquirriain [10] measured lower limb stiffness during an upright hopping task in athletes with AT, observing reductions in stiffness of the affected, compared with the unaffected leg. Arya, Solnik, Kulig [16] conducted the only study to date where stiffness has been compared with a healthy control group, which they did using an upright hopping model. They found that overall lower limb stiffness reduced, achieved by shifting to a knee strategy. As such, the most likely explanation for our findings is that in the presence of AT, the change in behaviour is done so with the aim of limiting exposure of the tendon to the painful stimulus. Assuming the strategy used to do so is task-dependent, our participants were likely attempting to reduce overall ankle load by limiting both ground contact time and reaction forces. Our findings, combined with those of Arya, Solnik, Kulig [16] suggest that one possible solution is that ankle stiffness increases to increase lower limb stiffness whilst knee stiffness reduces to reduce peak loading. If this theory is correct, our findings of increased stiffness could therefore be explained by the fact that our experimental model largely removes the ability of participants to redistribute a stiffness strategy to knee. Other less likely possible explanations for the increased stiffness values observed include the novel nature of the task, the absence of pain due to unloading, and the lack of perceived threat due to the secure nature of the task.

Consistent with our hypothesis, the AT group hopped in greater dorsiflexion at all recorded time points including; 4.3° at contact and 7.4° at peak. Our findings are consistent with those of Ryan, Grau, Krauss, Maiwald, Taunton, Horstmann [17], who investigated ankle range of motion in patients with AT compared with healthy volunteers, finding that runners with AT had comparatively increased dorsiflexion range of motion. Whilst we observed an increase in dorsiflexion stretch amplitude, Ryan, Grau, Krauss, Maiwald, Taunton, Horstmann [17] found similar findings on observation of eversion stretch amplitude. When combined with the findings of increased stiffness during the hopping task, it might be suggested that whilst increasing stiffness as a strategy to limit exposure to ground contact, individuals with AT lack the structural apparatus to achieve this in the most effective manner. During SSC tasks, elongation of the TA occurs in the presence of a 'quasi-isometric' plantarflexion contraction [18]. However, it has been reported that AT is associated with reduced tendon stiffness [19, 20], so the increased stretch amplitude may be viewed as an indicator of reduced tendon stiffness.

Our findings on temporal muscle activity partially supported our hypothesis with 3 of our 6 measures demonstrating delays in AT (see Table 1). The most likely explanation for this observation is that the pain experienced during the contact phase of the SSC can trigger inhibition of neuromuscular activity [21] resulting in delays or reductions in EMG activity [9]. Alternatively, it is possible that the earlier offsets observed are a manifestation of a learnt behaviour, adopted in response to chronic changes in muscular performance or alterations in sensory input secondary to changes in tendon compliance [22], which in turn affect feedforward muscle activity. Likewise, this sensory input may also be negatively influenced by the increased compliance observed in patients with AT [20]. Our findings are consistent with those of Azevedo, Lambert, Vaughan, O'Connor, Schwellnus [23], who observed reduced muscle activity in the tibialis anterior of runners with AT performing a running task. Likewise, Baur, Muller, Hirschmuller, Cassel, Weber, Mayer [9], investigating neuromuscular control of tibialis anterior, fibularis and gastrocnemius muscles in runners with AT reported that whilst no differences were observed in pre-activation of any muscles studied when compared with controls, gastrocnemius activity was reduced during the eccentric phase of the SSC. Finally, Wyndow, Cowan, Wrigley, Crossley [24] observed a delay in soleus offset in AT compared to controls during a running task in the

order of 18 (± 22) ms. The delay observed in tibialis anterior offset might suggest a global strategy of muscle delay in AT. The global nature of this strategy supports the findings of Smith, Honeywill, Wyndow, Crossley, Creaby [25] who observed delayed activation of gluteus medius and gluteus maximus in runners with AT.

Some methodological issues require consideration. The descriptive nature of this study limits causative interpretations; it is not possible to elucidate the temporal relationship between altered SSC behaviour, AT symptoms and pathology. Whilst age and gender matching was not ideal, the utilisation of the linear mixed model accounted for by its inclusion as a covariate in the model. We did not match participants for activity levels, which would have improved the homogeneity of groups. However, benefits exist in terms of external validity when using a heterogeneous group, which nevertheless remained reflective of the AT population. Finally, in this study, participants hopped rather than ran. In doing so, this enabled us to make a detailed exploration of SSC behaviour and whilst this is the first study to have conducted such an analysis in AT, its use in other experimental models does exist [11].

We speculate that many of our findings support the theory that biomechanical changes result in altered tendon loading and may be pathogenetic for AT. During SSC tasks, the plantarflexors control ankle dorsiflexion eccentrically and the shift in operating range towards greater dorsiflexion may increase the task demands of the plantarflexors. This may be further magnified by the possibility of a shift in the angle to peak torque that has been observed in other lower limb conditions [26]. This is an area of enquiry that justifies further exploration.

It has been speculated that muscle (pre-)activation is the strategy employed to increase stiffness to absorb impact forces [27], and the delays in muscle activity observed in our study could indicate an increase in tendon loading during the eccentric phase of the SSC. Although our findings require further confirmation, it is possible that differential stress generated by altered muscular activation generate altered intratendinous loads and may be associated with the pathogenesis of AT, as suggested by Wyndow, Cowan, Wrigley, Crossley [24].

Since no prospective data exists, we can only speculate if the observed changes in SSC behaviour in AT is a cause or consequence of the condition. Regardless of whether or not the observed changes in SSC behaviour are causative or not, they do inform clinical applications. It seems clear that focus should be placed on developing interventions to optimise SSC behaviour in line with modifying the impairments identified in this and other studies as a key factor to improving patient-centred functional outcomes.. In particular, our recommendations would include strategies to encourage regulation of stiffness strategies, reductions in dorsiflexion during ground contact and appropriate enhancement of agonist/antagonist timing around the ankle.

When considering these descriptive findings, it is recommended that prospective studies are undertaken to further explore whether the altered SSC behaviour observed in patients with AT is a consequence or a predisposing factor. In line with the clinical applications suggested by our findings, further study is also recommended exploring therapeutic interventions that modify SSC behaviour, such as strength training and plyometric training, developed with the relevant precautions required for this population in mind.

Conclusions

Our observation of SSC behaviour changes in Achilles tendinopathy showed relevant changes in lower limb stiffness, ankle joint kinematics and muscle activity. These findings support the theory of a mechanical pathoaetiological mechanism contributing to the development of Achilles tendinopathy and support the use of therapeutic interventions designed to optimise SSC behaviour in this patient population. Although these findings support these theories, further prospective studies are recommended to clarify causality.

Practical implications

- The shift in ankle mechanics during sub-maximal hopping towards a more dorsiflexed position with larger stretch amplitude and the associated global delay in muscle activity is likely to result in excessive load being placed on the Achilles tendon.

- The observed alterations in stretch shortening cycle behaviour in patients with Achilles tendinopathy lend support to the theory that failed loading is a pathoaetiological component of the condition.
- Clinicians should consider applying therapeutic interventions that optimise SSC behaviour in patients with AT.

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References

1. Magnussen RA, Dunn WR, Thomson AB. Nonoperative Treatment of Midportion Achilles Tendinopathy: A Systematic Review. *Clin J Sport Med.* 2009; 19(1):54-64.

2. van der Plas A, de Jonge S, de Vos RJ, et al. A 5-year follow-up study of Alfredson's heel-drop exercise programme in chronic midportion Achilles tendinopathy. *Br J of Sports Med.* 2012; 46(3):214-218.

3. Fu SC, Rolf C, Cheuk YC, et al. Deciphering the pathogenesis of tendinopathy: a three-stages process. *Sports Med, Arthrosc, Rehabil, Ther Technol.* 2010; 2:30.

4. Neviaser A, Andarawis-Puri N, Flatow E. Basic mechanisms of tendon fatigue damage. *J Shoulder Elbow Surg.* 2012; 21(2):158-163.

5. Taube W, Leukel C, Gollhofer A. How neurons make us jump: The neural control of stretch-shortening cycle movements. *Exerc Sport Sci Rev.* 2012; 40(2):106-115.

6. Bobbert MF, Richard Casius LJ. Spring-like leg behaviour, musculoskeletal mechanics and control in maximum and submaximum height human hopping. *Phil Trans R Soc Lond B Biol Sci.* 2011; 366(1570):1516-1529.

7. Butler RJ, Crowell HP, 3rd, Davis IM. Lower extremity stiffness: implications for performance and injury. *Clin Biomech.* 2003; 18(6):511-517.

8. Williams DS, Zambardino JA, Banning VA. Transverse-plane mechanics at the knee and tibia in runners with and without a history of achilles tendonopathy. *J Orthop Sport Phys Ther.* 2008; 38(12):761-767.

9. Baur H, Muller S, Hirschmuller A, et al. Comparison in lower leg neuromuscular activity between runners with unilateral mid-portion Achilles tendinopathy and healthy individuals. *J Electromyogr Kinesiol.* 2011; 21(3):499-505.

10. Maquirriain J. Leg stiffness changes in athletes with Achilles tendinopathy. *Int J Sport Med.* 2012; 33(7):567-571.

11. Kuitunen S, Kyrolainen H, Avela J, et al. Leg stiffness modulation during exhaustive stretch-shortening cycle exercise. *Scand J Med Sci Sports.* 2007; 17(1):67-75.

12. Besier TF, Sturnieks DL, Alderson JA, et al. Repeatability of gait data using a functional hip joint centre and a mean helical knee axis. *J Biomech.* 2003; 36(8):1159-1168.

13. Winter D. *Biomechanics and motor control of human movement*, New York, NY, John Wiley & Sons; 1990.

14. Dalleau G, Belli A, Viale F, et al. A simple method for field measurements of leg stiffness in hopping. *Int J Sport Med.* 2004; 25(3):170-176.

15. Allison GT. Trunk muscle onset detection technique for EMG signals with ECG artefact. *J Electromyogr Kinesiol.* 2003; 13(3):209-216.

16. Arya S, Solnik S, Kulig K. Influence of achilles tendinopathy on lower extremity joint stiffness strategies during single-legged hopping. *J Biomech.* 2006; 39(S1):S502-S502.

17. Ryan M, Grau S, Krauss I, et al. Kinematic analysis of runners with achilles mid-portion tendinopathy. *Foot Ank Int.* 2009; 30(12):1190-1195.

18. Lichtwark GA, Wilson AM. Interactions between the human gastrocnemius muscle and the Achilles tendon during incline, level and decline locomotion. *J Exp Biol.* 2006; 209(Pt 21):4379-4388.

19. Child S, Bryant AL, Clark RA, et al. Mechanical Properties of the Achilles Tendon Aponeurosis Are Altered in Athletes With Achilles Tendinopathy. *Am J Sports Med.* 2010.

20. Arya S, Kulig K. Tendinopathy alters mechanical and material properties of the Achilles tendon. *J App Physiol.* 2010; 108(3):670-675.

21. Henriksen M, Aaboe J, Graven-Nielsen T, et al. Motor responses to experimental Achilles tendon pain. *Br J Sports Med.* 2011; 45(5):393-398.

22. Lichtwark GA, Cresswell AG, Newsham-West RJ. Effects of running on human Achilles tendon length-tension properties in the free and gastrocnemius components. *J Exp Biol.* 2013; 216(Pt 23):4388-4394.

23. Azevedo LB, Lambert MI, Vaughan CL, et al. Biomechanical variables associated with Achilles tendinopathy in runners. *Br J Sports Med.* 2009; 43(4):288-292.

24. Wyndow N, Cowan SM, Wrigley TV, et al. Triceps surae activation is altered in male runners with Achilles tendinopathy. *J Electromyogr Kinesiol.* 2013; 23(1):166-172.

25. Smith MM, Honeywill C, Wyndow N, et al. Neuromotor Control of Gluteal Muscles in Runners with Achilles Tendinopathy. *Med Sci Sports Exerc.* 2013.

26. Brockett CL, Morgan DL, Proske U. Predicting hamstring strain injury in elite athletes. *Med Sci Sports Exerc.* 2004; 36(3):379-387.

27. Gollhofer A, Kyrolainen H. Neuromuscular control of the human leg extensor muscles in jump exercises under various stretch-load conditions. *Int J Sports Med.* 1991; 12:34-40.

28. Gibson W, Campbell A, Allison G. No evidence hip joint angle modulates intrinsically produced stretch reflex in human hopping. *Gait Posture.* 2013; 38(4):1005-1009.

Tables

Table 1. Mean (standard deviation) values for participant characteristics and biomechanical derived variables

Participant Characteristics	**Achilles Tendinopathy**	**Healthy Volunteers**
n = (male: female)	15 (9:6)	11(3:8)
Age (years)	41.2 (± 12.7)[a]	23.2 (± 6.7)
Height (cm)	174.1 (± 9.6) [a]	170.1 (± 8.2)
Mass (kg)	82.0 (± 12.2) [a]	70.7 (± 13.3)
VISA-A (0-100)	64.7 (± 12.7) [a]	100
Tendinopathy duration (months)	12.06 (±8.24)	na
Stiffness		
Lower Limb Stiffness (kNm^{-1})	8.8 (1.3) [a]	4.5 (2.6)
Ankle Kinematics		
Ankle Angle 80 ms pre-contact (° dorsiflexion)	-15.3 (9.97) [a]	-19.5 (8.97)
Ankle Angle at contact (° dorsiflexion)	-12.9 (10.0) [a]	-17.0 (9.3)
Peak Ankle Angle (° dorsiflexion)	18.4 (7.65) [a]	10.9 (9.97)
Ankle Stretch Amplitude (°)	29.9 (8.87) [a]	26.1 (6.82)
Muscle Activity		
Soleus Onset (ms)	82 (62) [a]	72 (66)
Soleus Peak (ms)	245 (69)	241 (72)
Soleus Offset (ms)	346 (67)	342 (66)
Tibialis Anterior Onset (ms)	46 (113)	38 (130)
Tibialis Anterior Peak (ms)	212 (114) [a]	201 (154)
Tibialis Anterior Offset (ms)	371 (74) [a]	347 (77)

[a] Significant difference between AT and CON means ($p<0.05$) adjusted for age, gender, height, mass and tendinopathy duration

VISA-A- Victorian Institute of Sport Assessment- Achilles

Figure Legends

Fig 1. (a) Custom-built low-friction sledge jump system (adapted with permission from Gibson, Campbell, Allison [28]; (b) 3D motion analysis marker set configuration and; (b) lower limb stiffness derivation [14]