Effects of intermittent claudication due to arterial disease on pain-free gait

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Abstract

Introduction:

Studies of intermittent claudication gait report inconsistent outcomes. Changes in gait are often attributed to degradation of calf muscles, but causation has not been proven through real-time electromyographic data. Neither have effects of walking speed been fully considered. This study aimed to investigate the effect of intermittent claudication on kinematics, kinetics and muscle activity during pain-free gait.

Methods:

18 able bodied individuals and 18 with intermittent claudication walked at their preferred speed while lower limb kinematic, kinetic and electromyography data were collected.

Findings:

People with intermittent claudication walk slower and with reduced step length. Internal ankle plantarflexion moment (P=0.004, effect size=0.96) and ankle power generation (P<0.001, effect size=1.36) in late stance were significantly reduced for individuals with intermittent claudication. Significant moment and power reductions at the knee and power reduction at hip occurred in early stance, with similar reductions in early and late stance for ground reaction forces. Peak electromyography of soleus activity was significantly reduced in late stance (P = 0.01, effect size = 1.1, n=13). Effects were independent of walking speed.

Interpretation:

Reductions in ankle plantarflexion moments and power generation were consistent with reduced soleus electromyography activity and reduced peak vertical ground reaction forces during late stance. These

effects are not due to a reduced walking speed. Changes in knee and hip function are also unrelated to walking speed. These outcomes provide a platform for the design and evaluation of interventions that seek to restore normal walking and improve pain-free walking distances for people with intermittent claudication.

Key words: intermittent claudication, gait, moment, pain-free

1. Introduction

Peripheral arterial disease affects 202 million people worldwide [1]; 20 million people in Europe, 8-10 million in north America, 10% of individuals over 65 years of age and 20% of those over 80 [2]. It is caused by atherosclerosis resulting in stenosis or occlusion of peripheral arteries which reduces the supply of blood and thereby oxygen and nutrients to peripheral tissues. During walking, blood flow in skeletal muscles increases to supply the higher oxygen demand. In peripheral artery disease, this increase cannot be achieved due to arterial occlusion resulting in lack of oxygen in muscles during walking. This causes cramping pain in the leg, most commonly in the calf. This pain typically occurs after walking short distances (e.g. less than 200m) and requires the individual to halt. Pain resolves after a few minutes rest but returns in a repeatable pattern during further walking, and is thus called intermittent claudication (IC). This affects 50% of individuals with arterial disease [3] impairs mobility and quality of life [4] and warrants surgery in many cases [5-6].

Several studies [7-13] have attempted to characterise gait in patients with IC. . However, despite the underlying physiological mechanism in IC being muscle activity and the associated demand for blood, fewer studies have reported muscle activity during gait for this patient group [13,14]. Gommans et al (2016), reported that tibialis anterior and medial gastrocnemius activity were the same as in healthy age-matched controls [13]. This finding appears to be in contradiction with the reductions in moment at the ankle and knee reported by Koutakis et al [11,12]. A similar contradiction appears between kinetic and kinematic studies and Bartolo et al (2019) who identified an increase in EMG muscle activity in many major lower limb muscles including the gastrocnemius and tibialis anterior [14]. In stationary studies, King et al (2015) identified that the soleus muscle contributes more to plantarflexion in both claudicating and asymptomatic limbs of individuals with PAD-IC compared to controls [15]. Although this is supported by muscle fibre changes observed in this group, it still does not provide a direct link to the kinetic results observed in other studies. The present study aimed to more uniformly investigate the link between muscle activity and the kinetics and kinematics of pain-free PAD-IC gait. It also aims to add to the existing research so that a more robust picture of PAD-IC gait and its potential musculature-based causes can be established.

Identifying any changes in gait, and especially muscle activity, is important because it should help inform the purpose of physical therapy interventions, such as exercise programs and therapeutic footwear. For example, footwear and orthoses that reduce calf muscle activity have been advocated for people with IC [6-17], but optimising these designs is hampered by an incomplete description of gait in IC.

As a forerunner to future work on gait interventions for IC, this study aimed to investigate the effects of IC on gait kinematics, kinetics and muscle activity.

1. Methods

Following ethical approval (14/LO/0382), 18 individuals with IC were recruited from local health services and 18 healthy and aged matched controls recruited from the same settings and the University community. Participants with IC were aged 50 years or over had a formal diagnosis confirmed by a Consultant Vascular surgeon (at least 3 months prior to participation). Diagnosis was based on colour-flow duplex scan, medical history, absence or reduction in foot pulses, and an ankle brachial pressure index (ABPI) of less than 0.8. They were able to walk for a minimum of 100m or perform 2 minutes of continuous walking unaided (Fontane scale II) [18]. Exclusion criteria were an active or a prior foot ulcer, significant foot deformities necessitating use of foot orthoses, complete neuropathy in their feet, surgery in the previous six months, pain in their lower limbs or back with a cause unrelated to peripheral arterial disease, painful knee, ankle or hip osteoarthritis, total reliance on walking aids, prior lower limb joint replacement, and morbid obesity (BMI>35). Healthy participants were excluded if they presented with any of the following: significant pain in the legs when walking, prior injury to the legs or spine, diabetic neuropathy, foot deformities (e.g. club foot, amputation of toes), prior joint replacement or major orthopaedic surgery.

2.1 Data collection

Participants wore shorts and t-shirt, and sites for EMG electrodes over tibialis anterior, medial gastrocnemius, lateral gastrocnemius and soleus muscles were prepared according to SENIAM guidelines [19]. Electrodes were connected to a Noraxon Telemyo system (TeleMyo 2400T G2, Noraxon U.S.A. Inc) and wires secured with bandages. Reflective markers were placed on the lower limb and pelvis following a CAST marker set-up (fig 1). Shoes were standardised and of an oxford style.

Figure 1: Marker set up: 2nd toe, 1st and 5th distal metatarsal head,1st (1PMT) and 5th proximal metatarsal head, 3 heel markers, medial and lateral malleolus, cluster of 4 shank markers, medial and lateral femoral tuberosity, cluster of 4 thigh markers, right and left greater trochanter, right and left anterior and posterior superior iliac spine.

Participants practised walking and their speed was recorded using timing gates to derive mean speed and a +/-5% tolerance. Participants then walked, at their preferred speed, as kinematic, (15 camera Qualysis system, Gothenburg, Sweden), ground reaction (400x600, AMTI Watertown, MA, USA) and EMG data was collected. Five successful walks were recorded for each participant. A walk was successful if the participant placed their feet on the force plates (the most affected limb only for IC participants) and walking speed remained within +/-5% of their mean speed. Healthy counterparts did not walk at speeds matched to the IC participants but speed was accounted for statistically.

2.2 Data processing

Markers were defined within Qualysis Track Manager and processed alongside force plate data in Visual 3D. The CAST marker set technique [20] was used. Rigid clusters of four non-orthogonal markers were placed over the lateral shank, lateral thigh and sacrum to track kinematics of each segment in six degrees of freedom. Eight retroreflective markers (fig 1) were placed onto each shoe type using double sided tape. The foot was modelled as a rigid, single segment. A static calibration trial was collected, for each experimental condition, in which retroreflective markers were placed on bony landmarks to specify the location of the lower limb joints in relation the clusters and to approximate joint centres. Ankle and knee joint centres were calculated as midpoints between the malleoli and femoral epicondyles, respectively. The hip joint centre was calculated using the regression model based on the anterior and posterior superior iliac spine markers [21]. In Visual 3D (C-Motion, Rockville, Maryland, USA), joint kinematics were calculated using an X–Y–Z Euler rotation sequence equivalent to the joint coordinate system [22] and joint kinetic data were calculated using three-dimensional inverse dynamics. Moments were then normalised to body mass. Marker data was filtered using a 4^{th} order Butterworth filter with a cut-off of 12 Hz. Kinetic data was low pass filtered with a cut-off of 25Hz. The data for each curve was then exported and ordered in Matlab (Mathworks, Natick, Massachusetts, USA) and exported to excel (Microsoft Excel 2013, Redmond, Washington, USA).

EMG signals were exported in their raw form to Matlab. Within Matlab, the signals were full-wave rectified, high pass filtered at 20 Hz, low pass filtered at 500 Hz and then filtered using an RMS envelope with a time window of 80. The methodology of filtering and cut-off values were as indicated by DeLuca (2010) [23]. The signal was synchronised with the gait cycle, using a manual sync pulse which was activated during data collection. During Matlab processing, this pulse was used to synchronise the muscle signals with the duration of the gait cycle and the signal that was captured outside the gait-cycle of interest was deleted. Matlab processing was made using a custom script. The relevant signal for each muscle was then exported to excel. Within excel, the average mean was calculated for each muscle and all trials were normalised to this mean value for each muscle and each participant, during stance phase as in Burden & Barlett (1999) [24].

For each participant the mean peaks and troughs for each outcome measure were computed as the average of 5 trials. The outcome measures were: maximum and minimum angle, moment and power at the ankle, knee and hip of the stance limb, peak ground reaction force (GRF) in the anterior/posterior, medial/lateral and vertical directions and maximum EMG activity during stance for the medial and lateral gastrocnemius, the soleus and tibialis anterior muscles, normalised to the mean stance activity of each muscle.

2.3 Statistical analysis

Independent t-tests were implemented via SPSS (Version 23, IBM Corporation, Chicago, USA) to investigate any difference between individuals with IC and healthy counterparts. If data were found not to follow a normal distribution (normality test in SPSS) a non-parametric Wilcoxon test was used. A univariate analysis of variance was conducted with speed as a covariate, to determine the effect of speed on the kinematic, kinetic and EMG characteristics of IC gait.

2. Results

Demographics and temporal gait characteristics of both groups are detailed in table 1. Average speed (*P*<0.001) and step length (*P*=0.019) were significantly reduced by 20% and 12.7% respectively for individuals with IC.

Table 1: Participant demographics and temporal-spatial parameters.

EMG data was available from 13 of the 18 participants with IC and 13 controls. In the remaining participants there was occasional EMG system failure and too few trials to use This is due to the study results being part of a larger data collection session which included the use of an orthotic. In patients where EMG signals were compromised due to electrode movement whilst wearing the orthotic, all EMG data for that participant was discounted. This has led to a lower EMG sample size compared to the sample size for kinematic/kinetic data.

3.1 Ankle

The peak internal ankle plantarflexion moment in late stance was reduced in cases of IC (*P*=0.006). Ankle power was significantly reduced for participants with IC both in late (*P*<0.001) and early (*P*=0.018) stance (fig 2a). After adjusting for speed, only the internal ankle plantarflexion moment and ankle power in late stance remained significantly reduced (*P*=0.030; *P* =0.022), and speed did not explain a significant amount of the variance (*P*=0.241; *P*=0.092) (table 2).

*Figure 2: 2a) Internal ankle moment of individuals with IC (dotted line) and healthy individuals (solid line) during stance. (+ve moment = internal dorsiflexion moment) 2b) Internal knee moment in IC (dotted line) and healthy (solid line) groups during stance. (+ve moment = internal extension moment) 2c) Internal hip in IC (*dotted line) *and healthy* (solid line) *groups during stance. (+ve moment = internal extension moment)*

Table 2: Mean, standard deviation and P values for ankle angle (°), ankle moment (Nm/Kg) and ankle power (W/Kg) in IC and healthy groups. * = Significant difference when adjusted for speed.

3.2 Knee

The internal knee flexion moment was significantly reduced at initial contact in the IC group (P=0.009) (fig 2b). Knee power absorption was reduced in both late and early stance (P=0.002) (p P=0.001) and knee power generation in mid-stance was also significantly reduced (P=0.047). When adjusted for speed, only knee power absorption in early stance was significantly reduced compared to healthy controls (P=0.022) and walking speed did not explain a significant amount of the variance (P=0.225) (table 3).

Table 3: Mean peaks, standard deviation and P values for knee angle (°), knee moment (Nm/Kg) and knee power (W/Kg) in IC and healthy groups. * = Significant differences when adjusted for speed.

3.3 Hip

Both the internal extension moment in early stance and the internal flexion moment in late stance were reduced in individuals with IC (fig 2c). Hip power absorption in early stance ($P<0.001$) and power generation in early stance ($P=0.024$) were also significantly reduced compared to healthy controls.

After adjusting for speed, only hip power absorption in early stance was significantly reduced ($P<0.001$) and speed did not explain a significant amount of the variance ($P=0.372$) (table 4).

Table 4: Mean peaks, standard deviation and P values for hip angle (°), hip moment (Nm/Kg) and hip power (W/Kg) in IC and healthy groups. * = Significant differences when adjusted for speed.

3.4 EMG

The increase from mean to maximum EMG activity, during stance, was reduced for lateral gastrocnemius (by > 22%) ($P=0.016$) and soleus (by > 19%) ($P=0.010$) in the 13 participants with IC , (table 5) (fig 3) (un-normalised IC means were lower than Healthy counterparts). When adjusted for speed, only soleus activity remained significantly reduced (adjusted means Healthy: 3.58 times of avg. mean, IC: 2.67 times of avg. mean; $P=0.26$).

Figure 3: 3a) Average lateral gastrocnemius activity in IC (dotted line) and healthy (solid line) groups during stance. EMG normalised using the mean dynamic method. 3b) Average soleus activity in IC (dotted line) and healthy (solid line) groups during stance. EMG normalised using the mean dynamic method.

Table 5: Mean, standard deviation and P value of peak EMG (expressed relative to each group's EMG mean) in IC and healthy groups. * = Significant differences when adjusted for speed

3.5 Ground Reaction Force

Maximum posterior (P<0.001) and anterior (P<0.001) GRF were significantly reduced in IC gait compared to the healthy group . Maximum lateral GRF in early stance (P = 0.002) and maximum vertical GRF in early and late stance were also significantly reduced (P=0.002; P=0.045; P<0.001) (table 6).

Table 6: Mean peak, standard deviation and P value for GRF (*body weight) in in IC and healthy groups. * = Significant differences when adjusted for speed.

Once adjusted for speed, late stance vertical GRF, maximum posterior and anterior GRF, and lateral GRF in early stance all remained significantly lower for individuals with IC (table 6). Speed was not found to explain a significant amount of the variance for any GRF variables (P>0.05).

3. Discussion

When taken in conjunction with previous literature, statistically significant findings (after adjusting for walking speed) confirm that the key points of difference between gait affected by IC and that of healthy older adults are:

- Reduced internal ankle plantarflexion moment and ankle power generation in late stance
- Reduced internal knee flexion moment at initial contact and knee power absorption in early stance
- Reduced hip power absorption in early stance
- Reduced peak posterior and lateral ground reaction force in early stance
- Reduced peak anterior and vertical ground reaction force in late stance
- Reduced increase in EMG activity of soleus muscle in late stance.

Other variables were significantly different in IC gait but only when not accounting for walking speed. Ankle power absorption in early stance, internal knee flexion at initial contact, knee power absorption in mid-stance and late stance, internal extension moment in early stance and the internal flexion moment in late stance, increase in maximum EMG activity of lateral gastrocnemius, and all ground reaction forces all ceased to be statistically significant. This highlights the importance of changes in walking speed on gait in this clinical group.

In accordance with previous literature the maximum internal ankle plantarflexion moment was reduced in late stance in individuals with IC [11-12].This agrees with Koutakis et al who tested unilateral [12] and bilateral cases [11] and thus the change in internal plantarflexion moment seems independent of uni/bi lateral nature of the arterial disease. Along with the reduced step length (table 1), ankle power generation and absorption throughout stance, and second peak in vertical GRF, this result indicates a reduction in internal force production in propulsion, specific to the calf muscles. This is further supported by the reduction in soleus EMG (>19%). This concurs with the clinical presentation of pain in the calf because insufficient blood and oxygen supply would lead to impaired muscle function and force production. The soleus has been found to have a significantly higher percentage of type I or slow twitch muscle fibres (70%) than the gastrocnemius (50%) [25-26]. These muscle fibres are more resistant to fatigue but produce weaker contractions than fast twitch (type II) muscle fibres. Furthermore, in the calf muscle of those with IC, the percentage of slow twitch (type I) muscle fibres is increased, compared to healthy controls [27-28]. This would mean a further increase in the slow twitch muscle fibres in the soleus, which would, in turn, reduce its peak force production.

Individuals with IC walked with 2.9 times their mean soleus activity compared to 3.6 for healthy counterparts. This effect was not due to speed but could perhaps relate to the reduced step length (-12.7%), since this would reduce the external forces and external joint moments the soleus opposes. However, any effects of step length would logically affect all plantarflexor muscles and a similar reduction was not found for the medial or lateral gastrocnemius. This selective effect could relate to the greater percentage of type I fibres in the soleus muscles [26-28] which would reduce its peak force production disproportionately compared to gastrocnemius.

The important reduction in ankle moments may be caused by the low walking speed in individuals with IC. Indeed, Wurdeman et al [8] found no such difference when they compared individuals with IC who walked at the same speed as healthy controls. However, the average speed of individuals with IC in Wurdeman’s study was considerably higher than that of individuals with IC in other studies, including the present one. Although IC severity was not described, the higher walking speeds reported suggest that the IC was less severe than in this and previous studies. This would also explain the absence of significant difference in internal ankle plantarflexion moment in late stance. Furthermore, recognising that people with IC do walk slower due to their generally poorer health and arterial disease status, the current study sought to account for the effect of walking speed statistically. Most changes at the ankle remained significant even accounting for speed and are thus related to the effects of IC.

Adjustments in walking speed, step length and calf muscle contraction could be concurrent strategies to reduce the demand for oxygen in the calf musculature and increase pain free walking distance. Muscle degeneration is present in individuals with IC [29]and this, coupled with the aforementioned change in muscle fibre type, would reduce force generating capacity. This is supported by previous findings of reduced peak isokinetic and concentric ankle torque produced by the calf muscles in individuals with IC [30,31]. This reduced physiological capacity may lead to a shorter step length since propulsion is less effective but also because it will reduce the external ankle dorsiflexion moments in late stance that the calf muscles must overcome. Ultimately there are a range of approaches that the body might adopt to better match muscle work done with muscle oxygen supply and thereby sustain pain free walking for longer.

The reduction in internal flexion moment at the knee at initial contact, internal extension moment at the hip in early stance, and reduced power absorption at both joints, indicate that individuals with IC have reduced eccentric function of the quadriceps. Likewise, the reduced power production in mid-stance, indicates a reduced effectiveness of the quadriceps to concentrically contract and support movement of the femur over the tibia. Similar to the results for calf muscles, this suggests reduced capacity of lower limb musculature in people with IC, as supported by similar literature findings in kinetics [11,31] and lower muscle strength in knee flexion (−14.0%) [31].

Peripheral arterial disease is a multilevel and multi-stage disease and this may lead to different gait effects. Changes at the level of the ankle are however almost unanimous across studies and different IC categories. This is perhaps not surprising given that the calf muscles act mainly around the ankle and this is the location of the clinical presentation.

Overall, findings suggest that if footwear or orthotic interventions are to benefit those with IC, they must affect external moments so that the force required by the calf muscles can be reduced. A study on the effect of orthotics and footwear on gait has explored the possibility of altering ankle moments and calf muscle activity [32]. Many studies have also assessed the effect of footwear as a calf muscle exercise tool [33-37]. Further research would be needed to investigate a footwear design with the potential to assist in the management of IC gait.

Limitations

4. Limitations

There are a range of limitations to this study that need to be considered. The sample size of the study was lower compared to other similar studies due to difficulty in recruitment. However, post-hoc power calculations (G*power) for the main outcome measures of ankle moment and ankle power indicated powers of 0.84 and 0.98 respectively, which are sufficient to support the findings. Sample size for EMG data was small and potentially not sufficient to detect changes at the calf muscles. This said, the significant findings for the soleus muscle activity, despite the low sample size, alongside the kinetic data, suggest intrinsic structural changes to the muscle lead to loss of force generating capacity. These changes are corroborated by previous research [31].

Furthermore, both individuals with unilateral and bilateral claudication were included in this study and this assumes gait effects are consistent between the two. However, the current research design is consistent with previous studies [7-8] and the purpose of this study was to investigate differences in gait in the wider IC population rather than focus on sub-populations.

Similarly, in the present study, participants were not chosen according to occlusion level. The level of occlusion has been shown to affect severity of patient symptoms and can significantly affect gait parameters. However, this study was part of a larger study on individuals with PAD-IC with a clinical focus on footwear to reduce symptoms. Therefore, it was deemed more appropriate to include participants of all occlusion levels but of the same scale of pain.

All participants wore Oxford style shoes. While this is not a limitation, the shoes were not those habitually worn by participants which could have affected their gait. This is standard practice in gait studies and ensures that gait patterns are not subject to the effects of the wide array of shoe types worn by participants.

5. Conclusion

People with IC walk slower and with reduced step length. There are reductions in ankle plantarflexion moments and power generation that are consistent with reduced soleus EMG activity and reduced peak vertical ground reaction forces during late stance. These effects are not due to reduced walking speed. There are also changes in knee moments and power indicative of reduced quadriceps capacity and reduced hip power absorption, which are unrelated to reduced walking speed. These outcomes provide a platform for the design and evaluation of interventions that seek to restore normal walking and improve pain free walking distances for this clinical population.

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Tables

	IC	Healthy	*P* value
Male	12	11	n/a
Female	6	7	n/a
Mean Age (yrs)	67.5(7.9)	61.3 (5.1)	0.246
Mean Height (m)	1.70 (SD = 0.08)	1.65 (SD = 0.08)	0.422
Mean Mass (kg)	75.3 (SD = 12.4)	69.2 (SD = 10.6)	0.182
Mean Walking Speed (m/s)	1.00 (SD = 0.25)	1.25 (SD = 0.23)	<0.001
Step length (m)	0.62 (SD = 0.07)	0.71 (SD = 0.13)	0.019
Diabetes	2	n/a	n/a
Unilateral claudication	7	n/a	n/a
Bilateral claudication	11	n/a	n/a

Table 1: Participant demographics and temporal-spatial parameters.

Table 2: Mean, standard deviation and *P* values for ankle angle (°), ankle moment (Nm/Kg) and ankle power (W/Kg) in IC and healthy groups. * = Significant difference when adjusted for speed.

Outcome measure	**IC**		**Healthy**			
	Mean	**SD**	**Mean**	**SD**	***P***	***Adjusted P***
Max ankle plantarflexion ***(°)***	-5.69	2.82	-7.23	2.29	0.081	>0.05
Max ankle dorsiflexion ***(°)***	-19.5	3.14	-18.9	3.18	0.559	>0.05
Max internal ankle dorsiflexion moment early stance (Nm/Kg)	0.25	0.11	0.28	0.06	0.271	>0.05
Max internal ankle plantarflexion moment late stance (Nm/Kg)	1.15	0.56	1.55	0.14	**0.004***	0.030
Max ankle power absorption early stance (W/Kg)	-0.55	0.26	-0.76	0.25	**0.018**	>0.05

Max ankle power absorption mid-stance (W/Kg)	-1.29	0.27	-1.57	0.66	0.112	>0.05
Max ankle power generation late stance (W/Kg)	1.90	0.49	2.80	0.80	**<0.001***	p =0.022

Table 3: Mean peaks, standard deviation and *P* values for knee angle (°), knee moment (Nm/Kg) and knee power (W/Kg) in IC and healthy groups.

* = Significant differences when adjusted for speed.

Outcome measure	**IC**		**Healthy**			
	Mean	**SD**	**Mean**	**SD**	***P***	***Adjusted P***
Max knee flexion early stance *(°)*	15.29	5.31	16.99	5.46	0.81	>0.05
Max knee extension *(°)*	3.13	4.66	3.10	5.71	0.559	>0.05
Max internal knee flexion moment initial stance (Nm/Kg)	-0.18	0.08	-0.25	0.08	**0.009**	>0.05
Max internal knee extension moment early stance (Nm/Kg)	0.50	0.22	0.59	0.19	0.184	0.030
Max internal knee flexion moment late stance (Nm/Kg)	-0.30	0.12	-0.33	0.11	0.354	>0.05
Max internal knee extension moment terminal stance (Nm/Kg)	0.15	0.06	0.18	0.06	0.136	>0.05
Max knee power absorption early stance (W/Kg)	-0.54	0.40	-1.03	0.38	**0.001***	0.022
Max knee power generation (W/Kg)	0.53	0.22	0.52	0.26	**0.047**	0.030
Max knee power generation mid-stance (W/Kg)	0.40	0.13	0.49	0.23	0.147	>0.05
Max knee power absorption late stance (W/Kg)	-0.81	0.33	-1.15	0.31	**0.002**	>0.05

Table 4: Mean peaks, standard deviation and *P* values for hip angle (°), hip moment (Nm/Kg) and hip power (W/Kg) in IC and healthy groups. * = Significant differences when adjusted for speed.

Outcome measure	**IC**		**Healthy**			
	Mean	**SD**	**Mean**	**SD**	***P***	***Adjusted P***
Max hip flexion ***(°)***	34.87	9.67	35.17	8.33	0.922	>0.05
Max hip extension ***(°)***	-5.66	11.47	-6.55	10.65	0.810	>0.05
Max internal hip extension moment early stance (Nm/Kg)	-0.55	0.16	-0.74	0.25	**0.012**	>0.05
Max internal hip flexion moment late stance (Nm/Kg)	0.61	0.19	0.77	0.25	**0.032**	0.030
Max hip power generation early stance (W/Kg)	0.52	0.26	0.85	0.53	**0.022**	>0.05
Max hip power absorption early stance (W/Kg)	-0.09	0.22	-0.52	0.28	**<0.001***	<0.001
Max hip power generation late stance (W/Kg)	0.98	0.35	1.18	0.43	0.136	>0.05
Max hip power absorption late stance (W/Kg)	-0.62	0.44	-0.72	0.37	0.482	>0.05

Table 5: Mean, standard deviation and *P* value of peak EMG (expressed relative to each group's EMG mean) in IC and healthy groups. * = Significant differences when adjusted for speed

Outcome measure	**IC**	**Healthy**		

	Mean	SD	Mean	SD	*P*	***Adjusted P***
Tibialis anterior	2.94	0.74	3.37	0.56	0.112	>0.05
Medial Gastrocnemius	3.91	1.38	3.98	1.00	0.888	>0.05
Lateral Gastrocnemius	3.30	0.86	4.21	0.92	**0.016**	>0.05
Soleus	2.89	0.62	3.57	0.60	**0.010***	**0.026**

Table 6: Mean peak, standard deviation and *P* value for GRF (*normalised to body weight) in IC and healthy groups.

* = Significant differences when adjusted for speed.

Outcome measure	**IC**		**Healthy**			
	Mean	**SD**	**Mean**	**SD**	***P***	***Adjusted P***
Maximum posterior GRF early stance *GRF (*normalised to body weight*	0.16	0.03	0.22	0.04	**<0.001***	**0.009**
Maximum anterior GRF late stance *GRF (*normalised to body weight*	-0.15	0.04	-0.20	0.03	**<0.001***	**0.005**
Maximum lateral GRF early stance *GRF (*normalised to body weight*	-0.03	0.02	-0.06	0.03	**0.002***	p = 0.017
Maximum lateral GRF late stance *GRF (*normalised to body weight*	-0.01	0.01	-0.01	0.01	0.193	>0.05
Maximum vertical GRF early stance *GRF (*normalised to body weight*	1.08	0.09	1.16	0.15	**0.045**	>0.05
Minimum vertical GRF mid-stance *GRF (*normalised to body weight*	0.78	0.04	0.74	0.12	0.170	>0.05
Maximum vertical GRF late stance *GRF (*normalised to body weight*	1.06	0.06	1.17	0.1	**<0.001***	0.004