Abstract

Stair negotiation poses a substantial physical demand on the musculoskeletal system and this challenging task can place individuals at risk of falls. Peripheral arterial disease (PAD) can cause intermittent claudication (IC) pain in the calf and results in altered gait mechanics during level walking. However, whether those with PAD-IC adopt alternate strategies to climb stairs is unknown. Twelve participants with PAD-IC (six bilateral and six unilateral) and 10 healthy controls were recruited and instructed to ascend a five-step staircase whilst 3D kinematic data of the lower-limbs were recorded synchronously with kinetic data from force plates embedded into the staircase on steps two and three. Limbs from the unilateral group and both limbs from the bilateral claudicants were categorised as claudicating (N=18), asymptomatic (N=6) and control (N=10). Claudicants walked more slowly than healthy controls (trend; P=<.066). Both claudicating- and asymptomatic-limb groups had reduced propulsive GRF (P=.025 and P=.002, respectively) and vertical GRF (P=.005 and P=.001, respectively) compared to controls. The claudicating-limb group had a reduced knee extensor moment during forward continuance (P=.060), ankle angular velocity at peak moment (P=.039) and ankle power generation (P=.055) compared to the controls. The slower gait speed, irrespective of laterality of symptoms, indicates functional capacity was determined by the limitations of the claudicating limb. Reduced ankle power generation and angular velocity (despite adequate plantarflexor moment) implies velocity-dependent limitations existed in the calf. The lack of notable compensatory strategies indicates reliance on an impaired muscle group to accomplish this potentially hazardous task, highlighting the importance of maintaining plantarflexor strength and power in those with PAD-IC.

Introduction

Peripheral arterial disease (PAD) is a chronic atherosclerotic disease of the peripheral arteries, primarily affecting the legs in older adults, with prevalence increasing with advancing age[1]. The disease negatively impacts on quality of life[2] and functional ability[3], with walking endurance declining with disease progression[4]. Alterations in gait biomechanics have been reported in proximal muscle groups (i.e. knee and hip extensors) as well as the plantarflexors[5,6], which are also a frequently reported site of intermittent claudication pain[7]. Ambulation over level ground is functionally important, however, the ability to negotiate stairs is also vital to maintaining functional independence[8]. There is a high incidence of falls among the elderly during stair walking[9,10], and balance, alongside lower limb strength, are vital pre-requisites to perform this task[11,12]. Given the previously reported impairments in balance[13,14], and reduced lower limb strength[15-17] of claudicants, stair negotiation likely poses a highly physically challenging and potentially hazardous task for this group of older adults.

Not only does stair negotiation place more demands on the lower limbs compared to level gait[12,18], but specific age-related adaptations also occur. The requirement to develop muscular force during stair ascent compared to the muscular capabilities is much greater for the healthy elderly than the young. The knee extensors in older adults work at 75% of their maximum capacity (quantified through isokinetic dynamometry) compared to 53% in younger adults[19] and, in some cases, operate in excess of their maximum measured strength at the knee[20]. A similar effect is evident at the ankle (elderly 93% vs young 85%)[19] with both the soleus muscle[21] and the gastrocnemii[22] playing important roles in raising the body to the next step in forward continuance.

It is evident that the gastrocnemii, in particular, are impaired in claudicants as there are signs of fibre type adaptations[23,24], infiltration of intra-muscular fat[25], neuromuscular impairments[26] and, more recently, adaptations in the structure of the gastrocnemii muscle and Achilles tendon[17,27]. Moreover, there is clear dysfunction in level gait with reduced plantarflexor moments and subsequently smaller power generation at push-off, which worsens in the presence of claudication pain[5,6,28]. However, it remains unknown how those with PAD-IC actually cope with the increased demands of stair ascent.

The purpose of the study was to determine whether individuals with PAD-IC adopt altered biomechanical profiles during stair ascent. This was achieved by comparing discrete parameters within the gait cycle to a control group consisting of healthy older adults, and exploring relationships between gait parameters and disease severity. It was hypothesised that claudicants would have reduced peak plantarflexor moment and ankle power generation compared to controls and adopt compensatory strategies at the knee and/or hip. Our second hypothesis was that reduced plantarflexor function would be associated with a reduced ankle brachial pressure index (ABPI), an indicator of disease severity in patients with PAD-IC.

Methods

Participants

Ethical approval was granted by the National Health Service Research Ethics Committee (REC reference: 11/YH/0335). Twenty-two participants were recruited, consisting of 12 individuals with PAD-IC (six unilateral, six bilateral) and 10 healthy controls (Table 1). PAD-IC patients were recruited from a local outpatient vascular clinic. Male and female participants aged

between 55-80 years diagnosed with Grade 1 Chronic Limb Ischemia[29] with an arterial narrowing of the superficial femoral artery were considered for inclusion. Healthy aged-matched controls were recruited from the local community. Individuals deemed to have severe or acute cardiovascular, musculoskeletal or pulmonary illness, history of neurological disorders, stroke, myocardial infarction or life-limiting diseases were excluded along with those with a previous lower-limb joint replacement and observable gait abnormalities or who required a walking aid.

Disease severity

Disease severity was determined using the ankle brachial pressure index (ABPI). ABPI measures for both lower limbs were taken pre- and post- a standardised exercise protocol performed on a motorised treadmill (5 minutes at 2.5km/h at 10% incline). Systolic blood pressure was measured in the posterior tibial and dorsalis pedis arteries of each leg and the brachial pressure of both arms, separately, using a sphygmomanometer cuff and a handheld Doppler instrument (Parks Medical Electronics Inc, Oregon, USA). In accordance with standard protocol, the ABPI for both legs was then calculated as the higher of the two leg artery pressures normalised to the higher brachial pressure of the two arms[7]. The post-exercise ABPI was used to identify the 'claudicating-limb' group (N=12 providing a total of 18 limbs; 12 from bilateral claudicants and 6 from unilateral claudicants) and the 'asymptomatic-limb' group (N=6 providing a total of 6 limbs from the unilateral claudicants). Whilst this creates a statistical imbalance between groups, to truly understand movement patterns within this cohort, potential adaptations in the asymptomatic limb as a compensatory mechanism for the more painful symptomatic limb must be explored. This method also assumes the asymptomatic limb is independent of the claudicating limb and although this contradicts the statistical rule of

independent samples, as the disease is unilateral in nature, the clinical presentations are different. The functional consequences may also differ therefore we believe this method of investigation is justified. Control participants also undertook the exercise protocol to determine ABPI values, to confirm the absence of PAD-IC. For brevity, only the dominant limb of the control participants, determined through a ball-kicking task, is presented.

Experimental protocol

Ten Qualisys Oqus 400 cameras (Qualisys, Gothenburg, Sweden) and 2 Kistler force plates (model 9286AA, Kistler, Winterthur, Switzerland), sampling at 100Hz and 1000Hz, respectively, were synchronised and collected kinematic and kinetic data. A total of 47 retro-reflective passive markers (14mm diameter) were positioned according to the six Degrees-of-Freedom marker set[30]. Functional movements were used to define the hip joint centre[31]. Participants were asked to ascend a custom-made five-step wooden staircase (step height; 20cm, step tread; 25cm, step width; 80cm) in a step-over-step manner. The staircase was instrumented with force plates embedded into steps two and three (step 5 being the top landing), and the top landing of the staircase was 1 metre in length. A 3m walkway in-front of the staircase allowed for steady-state level gait speed to be reached prior to stair ascent. The 1m top landing also allowed approximately two steps to be taken after participants ascended the staircase to avoid participants stopping on the last step. The staircase was equipped with a safety handrail and participants were instructed to use it only when necessary. Even light handrail influences lower limb kinetics during stair ascent[32] therefore, trials in which the participant used the handrail were excluded from further analysis. Our staircase has previously been shown to be rigid with negligible artefact or power lost from the force plate signals at physiologically relevant frequencies[33].

Data analysis

3D coordinate data were tracked using Qualysis Track Manager (V2.8, Qualysis, Gothenburg, Sweden) then exported for further processing in Visual 3D (V4, C-motion, Rockville, MD, USA). Coordinate data were interpolated using a cubic spline algorithm and both marker and kinetic data were filtered using a 2nd order low-pass Butterworth filter with a cut-off frequency of 6Hz for kinematic data and 15Hz for kinetic data. The focus of the present study was continuous, steady state stair ascent therefore one gait cycle was defined from foot contact on step 2 to the subsequent foot contact of the ipsilateral limb on step 4; and from foot contact on step 3 to the subsequent foot contact of the ipsilateral limb on step 5 for the contralateral limb. Lead limb preference for ascending the stairs was explored; no preferential strategy was evident for all participants and therefore not assessed further. Relevant gait events were identified (foot strike and foot off) using vertical ground reaction force (≥20 N and ≤20 N threshold for foot strike and foot off, respectively), and were normalised to 100% gait cycle for kinetic parameters and 100% stance phase for ground reaction forces. Sub-phases of gait were defined according to McFaydon & Winter (1998)[21]: weight acceptance, pull-up, forward continuance, foot clearance and foot placement. Variables of interest included temporal-spatial parameters, sagittal plane angular velocities, 3-dimensional ground reaction forces (GRF), sagittal plane kinetics (peak joint moments and powers), and angular velocities at the instant of peak moment for the hip, knee and ankle joints. Walking speed was determined over one gait cycle using gait events previously determined within Visual 3D. Angular velocities at the instant of peak moment occurred during weight acceptance for the hip and knee, and during forward continuance for the ankle. Positive angular velocities indicate changes in joint angle towards

ankle dorsiflexion, knee flexion and hip flexion. Data are expressed as mean and standard deviation.

Statistical analysis

Data were exported into SPSS v21.1 (SPSS Inc., Chicago, IL, USA), assessed for normality violations using Shapiro-Wilk's test for normality and assessed for outliers through box plot analysis. Demographic data were non-parametric so an independent samples Kruskal-Wallis test was performed with subsequent pairwise comparisons where appropriate. To avoid violating the assumption of independent samples, only the bilateral claudicants were included in the claudicating-limb group and unilateral claudicants in the asymptomatic-limb group for between-group analysis of walking speed and time spent in double support. As groups differed in walking speed between the bilateral and unilateral claudicants compared to healthy controls, a univariate analysis of variance was performed between control, claudicating limb and asymptomatic limb groups for joint kinetics and GRF with walking speed as a covariate. Where a significant interaction effect was observed, a Sidak post-hoc comparison was performed. A Pearson's partial product-moment correlation was performed to assess relationships between disease severity (as assessed by ABPI and controlled for the influence of age) and gait parameters for the claudicant group only. For all statistical tests, significance was accepted at $P \leq .05$ and trends were accepted at $P < .10$. For correlation analyses, a moderate relationship was accepted as R=.40–.59, a strong relationship was accepted as R=.60–.79 and a very strong relationship was accepted as R=.80–1[34].

Results

No significant differences were found between groups in age (P=.148), height (P=.230), or mass (P=.167) (Table I). Due to the bilateral nature of determining walking speed and to avoid violating the assumption of independent samples, walking speed and double support time were compared between bilateral and unilateral claudicants and healthy controls. Compared to the control group, trends towards slower walking speed than controls were evident in both the bilateral claudicant (0.71±0.09m/s vs 0.60±0.10m/s, P=.051) and the unilateral claudicant (0.71±0.09m/s vs 0.60±0.12m/s, P=.066) groups. Furthermore, unilateral claudicants spent significantly longer in double support compared to healthy controls (28.7±4.7% vs 20.2±6.2%, P=.018) with bilateral claudicants demonstrating similar trends (27.4±4.1 vs 20.2±6.2%, P=.088).

Table I. Participant characteristics. Data are presented as group mean (SD) unless otherwise stated

	Claudicating-limb	**Asymptomatic-limb**	**Control**
#	12	6	10
Limbs for analysis	18	6	10
% Males	83	67	40
Age (years)	64.7 (7.1)	67.3 (7.5)	61.6 (3.6)
Height (m)	1.72 (0.08)	1.70 (0.11)	1.66 (0.09)
Mass (Kg)	83.3 (18.8)	83.9 (22.6)	72.3 (10.9)
BMI (Kg/m^2)	28.0 (5.2)	28.8 (5.2)	26.1 (3.7)
ABPI pre-exercise	0.80 (0.21)	1.00 (0.12)	1.01 (0.09)
ABPI post-exercise	0.56 (0.20)	0.91 (0.08)	1.00 (0.16)
% Hypertension	50	50	10
% Hypercholesterolemia	58	67	20
% past smokers	50	50	30
% present smokers	50	50	0

BMI – Body mass index, ABPI – Ankle brachial pressure index.

Ground reaction forces and joint kinetics

Both the claudicating-limb and asymptomatic-limb groups had significantly reduced propulsive GRF (P=.025 and P=.002, respectively) and vertical GRF (P=.005 and P=.001, respectively) in forward continuance compared to healthy controls (Table II and Figure 1). Furthermore, the claudicating-limb group demonstrated trends towards increased braking force in early stance (P=.087), reduced knee extensor moment in forward continuance (P=.060) and ankle power generation (P=.055) compared to healthy controls. The claudicating-limb group had significantly reduced ankle angular velocity at the instant of peak plantarflexor moment (P=.039) compared to healthy controls (Table III).

Table II. Peak group mean (SD) ground reaction forces. * represent between-group differences reaching significance (P≤.05) and ^ represent those demonstrating trends towards significance (P<.10). ^{Con} = vs healthy control group. Positive values indicate medial, anterior and vertical force

GRF (N/BW)	Claudicating-limb	Asymptomatic-limb	Control
Medial during weight acceptance	0.60 (0.14)	0.49 (0.14)	0.54 (0.03)
Medial during forward continuance	0.55 (0.15)	0.47 (0.12)	0.60 (0.23)
Posterior during weight acceptance	-0.97 (0.33)$^{\wedge Con}$	-0.76 (0.26)	-0.71 (0.18)
Anterior during forward continuance	0.57 (0.14)^{*Con}	0.44 (0.12)^{*Con}	0.76 (0.10)
Vertical during weight acceptance	1.06 (0.13)	1.05 (0.11)	1.11 (0.13)
Vertical during forward continuance	1.15 (0.10)^{*Con}	1.14 (0.22)^{*Con}	1.53 (0.28)

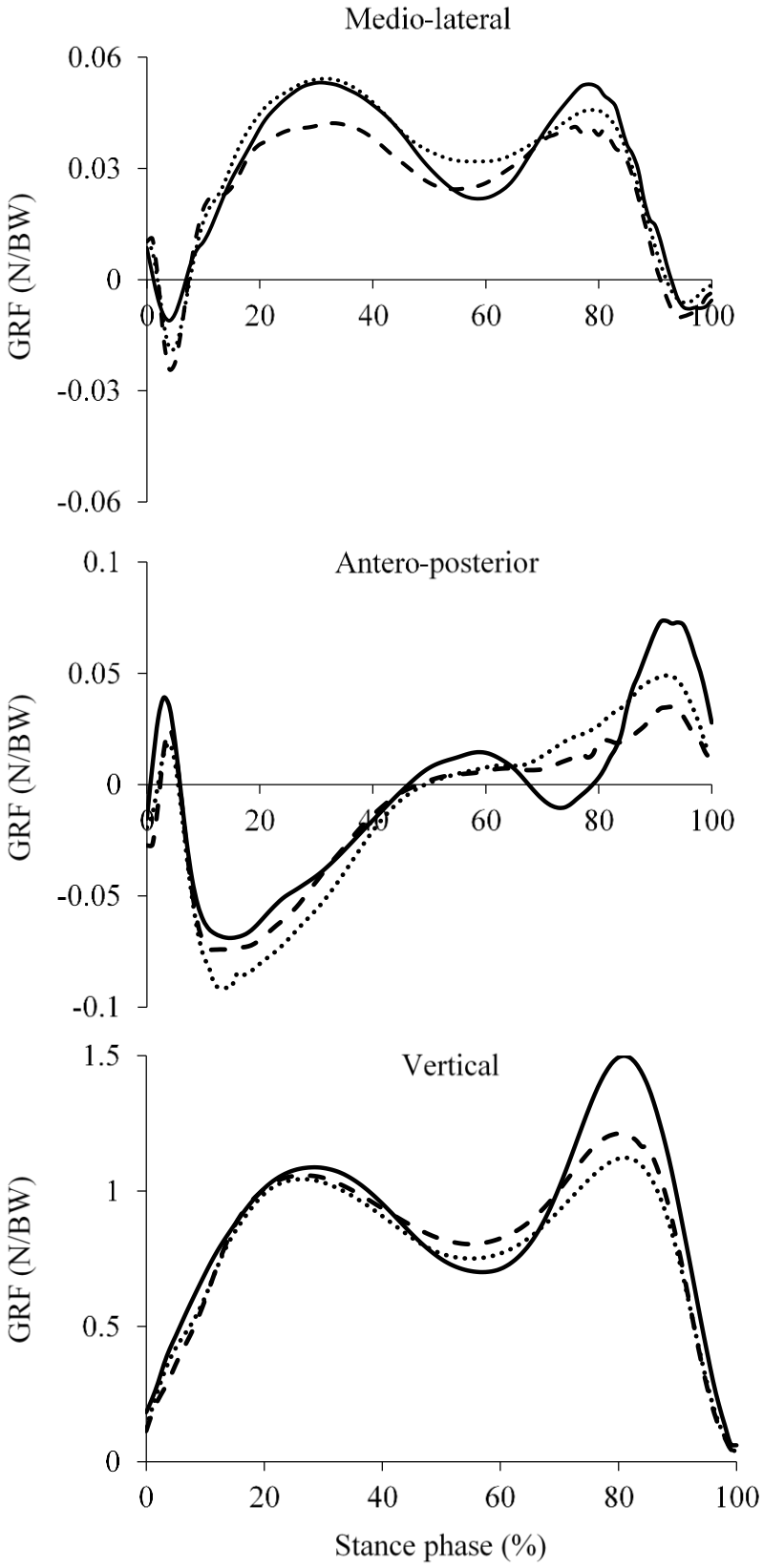

Figure 1. Group mean ground reaction forces per body weight (N/BW) for the claudication group (dotted line), asymptomatic limb group (dashed line) and control group (solid line) normalised to 100% stance phase. Positive direction indicates medial, anterior (propulsive) and vertical force.

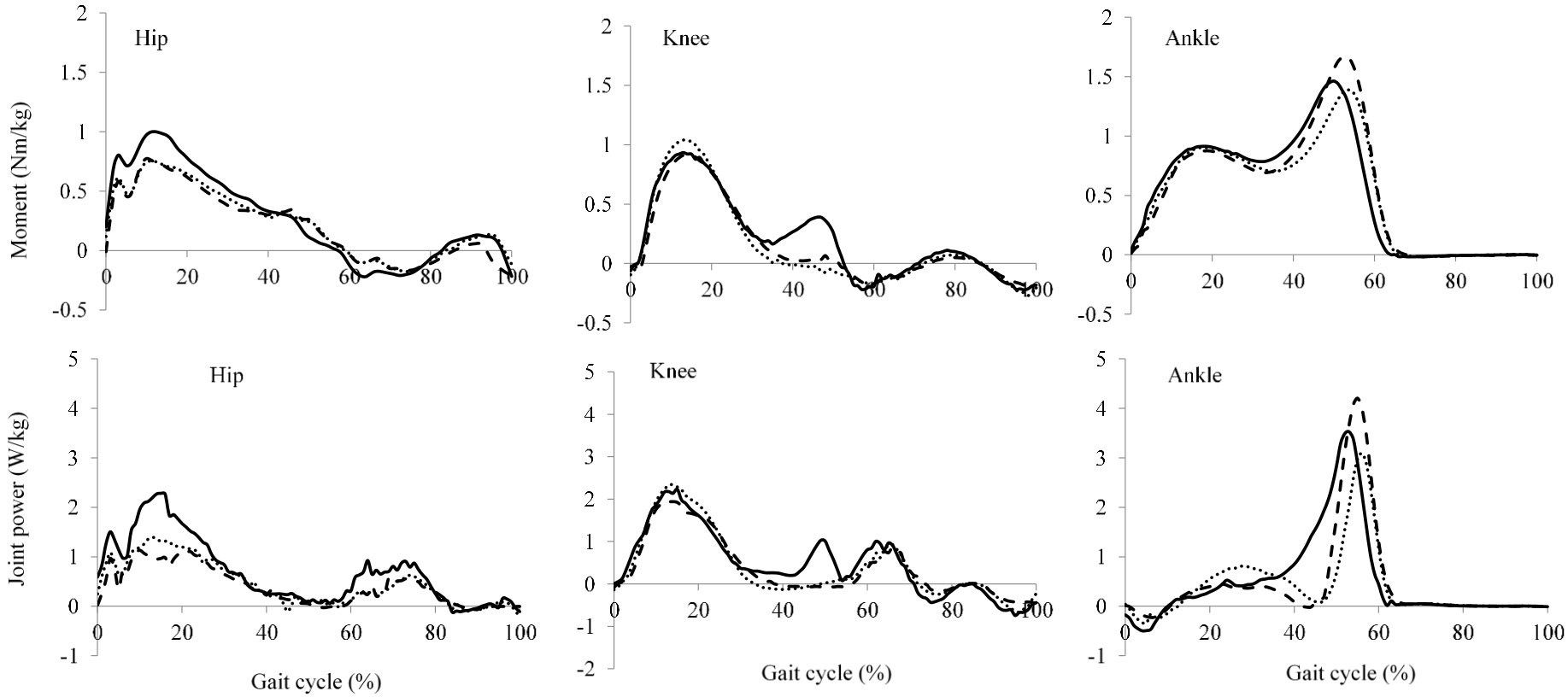

Figure 2. Group mean internal joint moment (top row) and joint power (bottom row) for the ankle, knee and hip across 100% gait cycle for claudicating-limb (dotted), asymptomatic-limb (dashed) and healthy controls (solid). Positive values indicate hip and knee extensor and ankle plantarflexor internal joint moments and power generation. Between 0-25% gait cycle was deemed the weight acceptance phase and between 40-65% deemed forward continuance.

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Table III. Peak group mean (SD) sagittal plane joint kinetics and angular velocities. Peak moments occurred during weight acceptance for the hip and knee, and during forward acceptance for the ankle. * represent between-group differences reaching significance (P≤.05) and ^ represent those demonstrating trends towards significance (P<.10). ^{Con} = vs healthy control group. Positive values indicate hip extensor, knee extensor, ankle plantarflexor and power generation.

Sagittal plane joint moments (Nm/kg) and angular velocities (°/s)	**Claudicating-limb**	**Asymptomatic-limb**	**Control**
Hip moment weight acceptance	0.91 (0.53)	0.84 (0.44)	1.16 (0.43)
Peak angular velocity	245.1 (57.8)	237.8 (51.1)	257.2 (23.7)
Angular velocity at peak moment	-103.5 (25.0)	-101.8 (19.2)	-137.2 (61.4)
Knee moment weight acceptance	1.05 (0.28)	1.07 (0.32)	1.03 (0.23)
Knee moment forward continuance	-0.14 (0.15)^{*Con}	-0.07 (0.20)	0.51 (0.20)
Peak angular velocity	213.0 (41.4)$^{\wedge Con}$	199.5 (32.3)$^{\wedge Con}$	281.2 (68.1)
Angular velocity at peak moment	122.7 (34.0)	127.6 (14.0)	139.6 (33.9)
Ankle moment weight acceptance	0.93 (0.48)	0.88 (0.23)	0.96 (0.40)
Ankle moment forward continuance	1.44 (0.31)	1.69 (0.39)	1.66 (0.43)
Peak angular velocity	198.4 (35.5)	231.1 (60.2)	229.5 (54.9)
Angular velocity at peak moment	-61.0 (22.4)^{*Con}	-82.4 (25.2)	-93.3 (28.1)
Joint powers (W/kg)	**Claudicating-limb**	**Asymptomatic-limb**	**Control**
Hip power weight acceptance	2.11 (1.36)	1.97 (1.45)	2.90 (1.03)
Knee power weight acceptance	2.48 (1.05)	2.50 (0.69)	2.55 (0.40)
Ankle power forward continuance	3.36 (1.19)$^{\wedge Con}$	4.43 (1.13)	5.28 (1.93)

Disease severity correlations

Increased disease severity was associated with increased peak medial GRF (R=-.630, P=.038) and trends towards reduced peak propulsive GRF during forward continuance (R=.601, P=.051), reduced knee extension moment during weight acceptance (R=.540, P=.086), and reduced peak knee angular velocity (R=-.554, P=.077). No association existed between overall walking speed of unilateral and bilateral claudicants, and disease severity (R=-.236, P.511).

Discussion

This study has, to the best of the authors' knowledge, investigated stair ascent biomechanics in PAD-IC for the first time. The findings suggest that claudicants walk more slowly than healthy controls, irrespective of whether the arterial stenosis and claudication symptoms are present in a single limb or bilaterally. In partial support of our first hypothesis, plantarflexor power generation in late stance (forward continuance), alongside propulsive and vertical GRF, were smaller in the claudicating-limb group compared to healthy controls. The reduced power generation in the claudicating-limb group appears to result from a slower ankle joint velocity necessary to achieve adequate peak plantarflexor moment in late stance.

The observed trends towards reduced ankle power generation in the forward continuance phase in the claudicating-limb group coincides with reduced angular velocity at peak moment, and reduced propulsive and vertical forces. This could be explained by the previous observation of reduced plantarflexor strength at high velocities[17]. Previous investigations into level gait mechanics have reported reduced plantarflexor moments in claudicants compared to healthy controls[5,28,35]. However, those studies also observed slower walking speeds in those with PAD-

IC compared to healthy controls, therefore those previous findings may also reflect walking-speed related gait differences and not solely reduced plantarflexor strength. A previous study on level gait mechanics compared claudicants to speed-matched controls[6] and the present study statistically controlled for variance in walking speed, both of which observed reduced joint powers in claudicants, not reduced moments. Therefore, velocity-dependent limitations in claudicant plantarflexors appear a genuine adaptation. It seems that claudicants possess adequate levels of strength when moving more slowly but are unable to exert that strength when moving more quickly. Therefore, it could be suggested that the slower walking speed is a strategy to allow claudicants to operate within safer limits relative to their maximal strength capacity. Further investigation following a similar experimental protocol to Reeves *et al.* (2009)[19] would be required to confirm these inferences. Nonetheless, these findings highlight the importance of maintaining plantarflexor strength, specifically plantarflexor power, in those with PAD-IC. Reductions in maximum strength capacity, without the adoption of adequate compensatory strategies, such as walking more slowly, would place excessive demands on the functionally-limited plantarflexors, thus jeopardising the ability to accomplish this task safely.

It has previously been documented that healthy elderly adopt strategies to increase knee extensor moment in late stance (forward continuance) just prior to peak ankle moment[19]. Those authors suggested that was a mechanism to transfer energy from the proximal knee joint to the distal ankle joint via the bi-articular gastrocnemii muscles, enabling a greater peak ankle moment to be generated. A similar profile was exhibited in the present study by the healthy elderly group (Figure 2) but not by the claudicants (either claudicating-limb group or asymptomatic-limb group), indicating a functionally useful mechanism was not being utilised by the PAD-IC patients. An alternative compensation could be an increase in contralateral hip

extensor activity during the mid-stance pull-up phase. However, no such increase in hip moments or powers were observed for either the claudicating-limb or asymptomatic-limb groups (Figure 2). The reasons for this are unclear, however reduced hip extensor strength has previously been associated with increased disease severity and reduced functional mobility in claudicants[36] and it may be that they lack adequate capacity to utilise such a compensation irrespective of the disease presence uni- or bi-laterally. Alternatively, it may simply be that the claudicants evade increased muscular effort in any lower limb muscle group as an attempt to avoid earlier onset of claudication pain and increased metabolic cost[37]. The lack of any observable compensatory strategy may also contribute to the aforementioned slower walking speed in claudicants as they are relying on the smaller, weaker and frequently symptomatic plantarflexor group to perform this task.

Given the identified functional decline of the ankle musculature in the claudicating limbs, it may have been expected that, in unilateral claudicants, the asymptomatic limb would demonstrate compensatory hip and/or knee strategies. Interestingly however, the asymptomatic limb demonstrated similar adaptations to the claudicating limb with reduced propulsive and vertical forces, reduced peak knee angular velocity and a (non-significant) 16% reduction in ankle power generation compared to healthy controls. These findings are analogous to those investigations of unilateral claudicants during level gait[5,6] and suggest that unilateral claudicants are functionally limited by the claudicating limb. This further highlights the importance of improving the strength and function of the claudicating limb, regardless of whether the disease is present in a single limb or both.

The present study investigated pain-free stair ascent only. Plantarflexor function during level gait deteriorates further in the presence of claudication pain[5,38]. It would be reasonable to assume that adaptations highlighted in the present study may be exacerbated detrimentally and extrapolated further during painful locomotion, however future investigation is needed to confirm this speculation. Whilst we statistically controlled for the influence of walking speed in between-group analyses, a more vigorous investigation comparing claudicants to speed-matched controls is required to fully explore the velocity-dependent limitations in the gastrocnemii. Previous research has also demonstrated light handrail use influences gait biomechanics[32] and the present study excluded trials in which participants used the handrail as the extent of handrail use and upper limb kinetics/force could not be quantified with our staircase design. However, given the reported balance deficits in the population[13,14], handrail use, particularly in the presence of claudication pain for example, may play a larger compensatory role and requires further investigation.

Conclusions

This study provides evidence for specific limitations of the plantarflexor muscles during stair ascent in claudicants with peripheral arterial disease. There was a lack of notable hip or knee strategies in the claudicating limb as a compensatory mechanism for the clear, velocity-dependent limitations of the plantarflexors. Similarly, in unilateral claudicants, there were no observable compensations from the asymptomatic limb. Thus, the stair climbing function of those with PAD-IC seems to be determined by the limitations of the claudicating limb, highlighting the importance of maintaining or improving strength and power in the plantarflexor muscle group.

References

1. Roger VL, Go AS, Lloyd-Jones DM, Adams RJ, Berry JD, Brown TM, et al. Heart disease and stroke statistics--2011 update: a report from the American Heart Association. Circulation. 2011;123(4):e18-e209.

2. Spronk S, White JV, Bosch JL, Hunink MG. Impact of claudication and its treatment on quality of life. Semin Vasc Surg. 2007;20(1):3-9.

3. McDermott MM, Greenland P, Liu K, Guralnik J, Criqui MH, Dolan NC, et al. Leg Symptoms in peripheral arterial disease. Associated clinical characterstics and functional impairment. JAMA. 2001;286(13):1599-606.

4. McDermott M, Liu K, Greenland P, Guralnik JM, Criqui MH, Chan C, et al. Functional Decline in Peripheral Arterial Disease Associations With the Ankle Brachial Index and Leg Symptoms. Journal of American Medical Association. 2004;292:453-61.

5. Koutakis P, Johanning JM, Haynatzki GR, Myers SA, Stergiou N, Longo GM, et al. Abnormal joint powers before and after the onset of claudication symptoms. J Vasc Surg. 2010;52(2):340-7.

6. Wurdeman SR, Koutakis P, Myers SA, Johanning JM, Pipinos II, Stergiou N. Patients with peripheral arterial disease exhibit reduced joint powers compared to velocity-matched controls. Gait & Posture. 2012;36(3):506-9.

7. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG, et al. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). Eur J Vasc Endovasc Surg. 2007;33 Suppl 1:S1-75.

8. Ariza-Vega P, Jimenez-Moleon JJ, Kristensen MT. Change of residence and functional status within three months and one year following hip fracture surgery. Disabil Rehabil. 2014;36(8):685-90.

9. Hemenway D, Solnick SJ, Koeck C, Kytir J. The incidence of stairway injuries in Austria. Accid Anal Prev. 1994;26(5):675-9.

10. Startzell JK, Owens DA, Mulfinger LM, Cavanagh PR. Stair Negotiation in Older People - A Review. Journal of American Geriatric Society. 2000;48:567-80.

11. Verghese J, Wang C, Xue X, Holtzer R. Self-reported difficulty in climbing up or down stairs in nondisabled elderly. Arch Phys Med Rehabil. 2008;89(1):100-4.

12. Nadeau S, McFadyen BJ, Malouin F. Frontal and sagittal plane analyses of the stair climbing task in healthy adults aged over 40 years: what are the challenges compared to level walking? Clin Biomech (Bristol, Avon). 2003;18(10):950-9.

13. Mockford KA, Mazari FA, Jordan AR, Vanicek N, Chetter IC, Coughlin PA. Computerized dynamic posturography in the objective assessment of balance in patients with intermittent claudication. Ann Vasc Surg. 2011;25(2):182-90.

14. Gohil RA, Mockford KA, Mazari F, Khan J, Vanicek N, Chetter IC, et al. Balance impairment, physical ability, and its link with disease severity in patients with intermittent claudication. Ann Vasc Surg. 2013;27(1):68-74.

15. McDermott MM, Criqui MH, Greenland P, Guralnik JM, Liu K, Pearce WH, et al. Leg strength in peripheral arterial disease: associations with disease severity and lower-extremity performance. J Vasc Surg. 2004;39(3):523-30.

16. Camara LC, Ritti-Dias RM, Meneses AL, D'Andrea Greve JM, Filho WJ, Santarem JM, et al. Isokinetic strength and endurance in proximal and distal muscles in patients with peripheral artery disease. Ann Vasc Surg. 2012;26(8):1114-9.

17. King S, Vanicek N, O'Brien TD. Dynamic muscle quality of the plantar flexors is impaired in claudicant patients with peripheral arterial disease and associated with poorer walking endurance. J Vasc Surg. 2015. 62(3): 689-697

18. Tiedemann AC, Sherrington C, Lord SR. Physical and psychological factors associated with stair negotiation performance in older people. J Gerontol A Biol Sci Med Sci. 2007;62(11):1259-65.

19. Reeves ND, Spanjaard M, Mohagheghi AA, Baltzopoulos V, Maganaris CN. Older adults employ alternative strategies to operate within their maximum capabilities when ascending stairs. Journal of Electromyography and Kinesiology. 2009;19(2):e57-e68.

20. Samuel D, Rowe P, Hood V, Nicol A. The biomechanical functional demand placed on knee and hip muscles of older adults during stair ascent and descent. Gait Posture. 2011;34(2):239-44.

21. McFadyen BJ, Winter DA. An integrated biomechanical analysis of normal stair ascent and descent. J Biomech. 1988;21(9):733-44.

22. Spanjaard M, Reeves ND, van Dieen JH, Baltzopoulos V, Maganaris CN. Gastrocnemius muscle fascicle behavior during stair negotiation in humans. J Appl Physiol. 2007;102(4):1618-23.

23. Steinacker JM, Opitz-Gress A, Baur S, Lormes W, Bolkart K, Sunder-Plassmann L, et al. Expression of myosin heavy chain isoforms in skeletal muscle of patients with peripheral arterial occlusive disease. Journal of Vacular Surgery. 2000;31:443-9.

24. McGuigan MRM, FBronks R, Newton RU, Sharman MJ, Graham JC, Cody DV, et al. Muscle fiber characteristics in patients with peripheral arterial disease. Medical Science in Sports Exercise. 2001;33(12):2016-21.

25. Raval Z, Liu K, Tian L, Ferrucci L, Guralnik JM, Liao Y, et al. Higher body mass index is associated with more adverse changes in calf muscle characteristics in peripheral arterial disease. Journal of Vascular Surgery. 2012;55(4):1015-24.

26. Garg PK, Liu K, Ferrucci L, Guralnik JM, Criqui MH, Tian L, et al. Lower extremity nerve function, calf skeletal muscle characteristics, and functional performance in peripheral arterial disease. J Am Geriatr Soc. 2011;59(10):1855-63.

27. King SL, Vanicek N, O'Brien TD. Gastrocnemius muscle architecture and Achilles tendon properties influence walking distance in claudicants with peripheral arterial disease. Muscle Nerve. 2015.

28. Chen SJ, Pipinos I, Johanning J, Radovic M, Huisinga JM, Myers SA, et al. Bilateral claudication results in alterations in the gait biomechanics at the hip and ankle joints. J Biomech. 2008;41(11):2506-14.

29. Rutherford RB, Baker D, Ernst C, Johnston KW, Porter JM, Ahn S, et al. Recommended standards for reports dealing with lower extremity ischemia: Revised version. J Vasc Surg. 1997;26:517-38.

30. Cappozzo A, Catani F, Della Croce U, Leardini A. Position and orientation in space of bones during movement: anatomical frame definition and determination. Clin Biomech (Bristol, Avon). 1995;10:171-8.

31. Leardini A, Cappozzo A, Catani F, Toksvig-Larsen S, Petitto A, Sforza V, et al. Validation of a functional method for the estimation of hip joint centre location. J Biomech. 1999;32(1):99-103.

32. Reeves ND, Spanjaard M, Mohagheghi AA, Baltzopoulos V, Maganaris CN. Influence of light handrail use on the biomechanics of stair negotiation in old age. Gait Posture. 2008;28(2):327-36.

33. Chesters T, Alcock L, O'Brien TD, Vanicek N, Dobson CA. Frequency domain characteristics of kinetic data from force platforms mounted in instrumented stairways. Gait Posture. 2013;39:S118-S9.

34. Taylor R. Interpretation of the Correlation Coefficient: A Basic Review. Journal of Diagnostic and Medical Sonography. 1990;6(1):35-9.

35. Koutakis P, Pipinos II, Myers SA, Stergiou N, Lynch TG, Johanning JM. Joint torques and powers are reduced during ambulation for both limbs in patients with unilateral claudication. Journal of Vascular Surgery. 2010;51(1):80-8.

36. Parmenter BJ, Raymond J, Dinnen PJ, Lusby RJ, Fiatarone Singh MA. Preliminary evidence that low ankle-brachial index is associated with reduced bilateral hip extensor strength and functional mobility in peripheral arterial disease. J Vasc Surg. 2013;57(4):963-73.

37. Mockford KA, Vanicek N, Jordan A, Chetter IC, Coughlin PA. Kinematic adaptations to ischemic pain in claudicants during continuous walking. Gait Posture. 2010;32(3):395-9.

38. Scott-Pandorf MM, Stergiou N, Johanning JM, Robinson L, Lynch TG, Pipinos, II. Peripheral arterial disease affects ground reaction forces during walking. J Vasc Surg. 2007;46(3):491-9.