

1 **Sagittal plane joint kinetics during stair ascent in patients with peripheral arterial disease**
2 **and intermittent claudication**

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17 **Category:** Original article

18 **Word Count:** 2780 excluding references

19 **Short title:** Stair ascent in PAD-IC

20

21 **Acknowledgements:** We wish to thank Dr Risha Lane and Professor Ian Chetter for their
22 invaluable assistance in patient recruitment

23

24 **Conflicts of Interest:** None

25

26

1 **Abstract**

2

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

1 **Introduction**

2 Peripheral arterial disease (PAD) is a chronic atherosclerotic disease of the peripheral arteries,
3 primarily affecting the legs in older adults, with prevalence increasing with advancing age¹.
4 The disease negatively impacts on quality of life² and functional ability³, with walking
5 endurance declining with disease progression⁴. Alterations in gait biomechanics have been
6 reported in proximal muscle groups (i.e. knee and hip extensors) as well as the plantarflexors^{5,6},
7 which are also a frequently reported site of intermittent claudication pain⁷. Ambulation over
8 level ground is functionally important, however, the ability to negotiate stairs is also vital to
9 maintaining functional independence⁸. There is a high incidence of falls among the elderly
10 during stair walking^{9,10}, and balance, alongside lower limb strength, are vital pre-requisites to
11 perform this task^{11,12}. Given the previously reported impairments in balance^{13,14}, and reduced
12 lower limb strength¹⁵⁻¹⁷ of claudicants, stair negotiation likely poses a highly physically
13 challenging and potentially hazardous task for this group of older adults.

14

15 Not only does stair negotiation place more demands on the lower limbs compared to level
16 gait^{12,18}, but specific age-related adaptations also occur. The requirement to develop muscular
17 force during stair ascent compared to the muscular capabilities is much greater for the healthy
18 elderly than the young. The knee extensors in older adults work at 75% of their maximum
19 capacity (quantified through isokinetic dynamometry) compared to 53% in younger adults¹⁹
20 and, in some cases, operate in excess of their maximum measured strength at the knee²⁰. A
21 similar effect is evident at the ankle (elderly 93% vs young 85%)¹⁹ with both the soleus
22 muscle²¹ and the gastrocnemii²² playing important roles in raising the body to the next step in
23 forward continuance.

24

1 It is evident that the gastrocnemii, in particular, are impaired in claudicants as there are signs
2 of fibre type adaptations^{23,24}, infiltration of intra-muscular fat²⁵, neuromuscular impairments²⁶
3 and, more recently, adaptations in the structure of the gastrocnemii muscle and Achilles
4 tendon^{17,27}. Moreover, there is clear dysfunction in level gait with reduced plantarflexor
5 moments and subsequently smaller power generation at push-off, which worsens in the
6 presence of claudication pain^{5,6,28}. However, it remains unknown how those with PAD-IC
7 actually cope with the increased demands of stair ascent.

8

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

17

18 **Methods**

19 *Participants*

20 Ethical approval was granted by the National Health Service Research Ethics Committee (REC
21 reference: 11/YH/0335). Twenty-two participants were recruited, consisting of 12 individuals
22 with PAD-IC (six unilateral, six bilateral) and 10 healthy controls (Table 1). PAD-IC patients
23 were recruited from a local outpatient vascular clinic. Male and female participants aged
24 between 55-80 years diagnosed with Grade 1 Chronic Limb Ischemia²⁹ with an arterial

1 narrowing of the superficial femoral artery were considered for inclusion. Healthy aged-
2 matched controls were recruited from the local community. Individuals deemed to have severe
3 or acute cardiovascular, musculoskeletal or pulmonary illness, history of neurological
4 disorders, stroke, myocardial infarction or life-limiting diseases were excluded along with
5 those with a previous lower-limb joint replacement and observable gait abnormalities or who
6 required a walking aid.

7

8 *Disease severity*

9 Disease severity was determined using the ankle brachial pressure index (ABPI). ABPI
10 measures for both lower limbs were taken pre- and post- a standardised exercise protocol
11 performed on a motorised treadmill (5 minutes at 2.5km/h at 10% incline). Systolic blood
12 pressure was measured in the posterior tibial and dorsalis pedis arteries of each leg and the
13 brachial pressure of both arms, separately, using a sphygmomanometer cuff and a handheld
14 Doppler instrument (Parks Medical Electronics Inc, Oregon, USA). In accordance with
15 standard protocol, the ABPI for both legs was then calculated as the higher of the two leg artery
16 pressures normalised to the higher brachial pressure of the two arms⁷. The post-exercise ABPI
17 was used to identify the ‘claudicating-limb’ group (N=12 providing a total of 18 limbs; 12 from
18 bilateral claudicants and 6 from unilateral claudicants) and the ‘asymptomatic-limb’ group
19 (N=6 providing a total of 6 limbs from the unilateral claudicants). Whilst this creates a
20 statistical imbalance between groups, to truly understand movement patterns within this cohort,
21 potential adaptations in the asymptomatic limb as a compensatory mechanism for the more
22 painful symptomatic limb must be explored. This method also assumes the asymptomatic limb
23 is independent of the claudicating limb and although this contradicts the statistical rule of
24 independent samples, as the disease is unilateral in nature, the clinical presentations are

1 different. The functional consequences may also differ therefore we believe this method of
2 investigation is justified. Control participants also undertook the exercise protocol to determine
3 ABPI values, to confirm the absence of PAD-IC. For brevity, only the dominant limb of the
4 control participants, determined through a ball-kicking task, is presented.

5

6 *Experimental protocol*

7 Ten Qualisys Oqus 400 cameras (Qualisys, Gothenburg, Sweden) and 2 Kistler force plates
8 (model 9286AA, Kistler, Winterthur, Switzerland), sampling at 100Hz and 1000Hz,
9 respectively, were synchronised and collected kinematic and kinetic data. A total of 47 retro-
10 reflective passive markers (14mm diameter) were positioned according to the six Degrees-of-
11 Freedom marker set³⁰. Functional movements were used to define the hip joint centre³¹.
12 Participants were asked to ascend a custom-made five-step wooden staircase (step height;
13 20cm, step tread; 25cm, step width; 80cm) in a step-over-step manner. The staircase was
14 instrumented with force plates embedded into steps two and three (step 5 being the top landing),
15 and the top landing of the staircase was 1 metre in length. A 3m walkway in-front of the
16 staircase allowed for steady-state level gait speed to be reached prior to stair ascent. The 1m
17 top landing also allowed approximately two steps to be taken after participants ascended the
18 staircase to avoid participants stopping on the last step. The staircase was equipped with a
19 safety handrail and participants were instructed to use it only when necessary. Even light
20 handrail influences lower limb kinetics during stair ascent³² therefore, trials in which the
21 participant used the handrail were excluded from further analysis. Our staircase has previously
22 been shown to be rigid with negligible artefact or power lost from the force plate signals at
23 physiologically relevant frequencies³³.

24

1 *Data analysis*

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

25

1 *Statistical analysis*

2 Data were exported into SPSS v21.1 (SPSS Inc., Chicago, IL, USA), assessed for normality
3 violations using Shapiro-Wilk's test for normality and assessed for outliers through box plot
4 analysis. Demographic data were non-parametric so an independent samples Kruskal-Wallis
5 test was performed with subsequent pairwise comparisons where appropriate. To avoid
6 violating the assumption of independent samples, only the bilateral claudicants were included
7 in the claudicating-limb group and unilateral claudicants in the asymptomatic-limb group for
8 between-group analysis of walking speed and time spent in double support. As groups differed
9 in walking speed between the bilateral and unilateral claudicants compared to healthy controls,
10 a univariate analysis of variance was performed between control, claudicating limb and
11 asymptomatic limb groups for joint kinetics and GRF with walking speed as a covariate. Where
12 a significant interaction effect was observed, a Sidak post-hoc comparison was performed. A
13 Pearson's partial product-moment correlation was performed to assess relationships between
14 disease severity (as assessed by ABPI and controlled for the influence of age) and gait
15 parameters for the claudicant group only. For all statistical tests, significance was accepted at
16 $P \leq .05$ and trends were accepted at $P < .10$. For correlation analyses, a moderate relationship was
17 accepted as $R = .40-.59$, a strong relationship was accepted as $R = .60-.79$ and a very strong
18 relationship was accepted as $R = .80-1$ ³⁴.

19

20 **Results**

21 No significant differences were found between groups in age ($P = .148$), height ($P = .230$), or
22 mass ($P = .167$) (Table I). Due to the bilateral nature of determining walking speed and to avoid
23 violating the assumption of independent samples, walking speed and double support time were
24 compared between bilateral and unilateral claudicants and healthy controls. Compared to the

1 control group, trends towards slower walking speed than controls were evident in both the
 2 bilateral claudicant ($0.71\pm 0.09\text{m/s}$ vs $0.60\pm 0.10\text{m/s}$, $P=.051$) and the unilateral claudicant
 3 ($0.71\pm 0.09\text{m/s}$ vs $0.60\pm 0.12\text{m/s}$, $P=.066$) groups. Furthermore, unilateral claudicants spent
 4 significantly longer in double support compared to healthy controls ($28.7\pm 4.7\%$ vs $20.2\pm 6.2\%$,
 5 $P=.018$) with bilateral claudicants demonstrating similar trends (27.4 ± 4.1 vs $20.2\pm 6.2\%$,
 6 $P=.088$).

7

8 **Table I.** Participant characteristics. Data are presented as group mean (SD) unless otherwise
 9 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/m²)	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

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

11

12

1 *Ground reaction forces and joint kinetics*

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

10

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

15

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) ^{^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)

16

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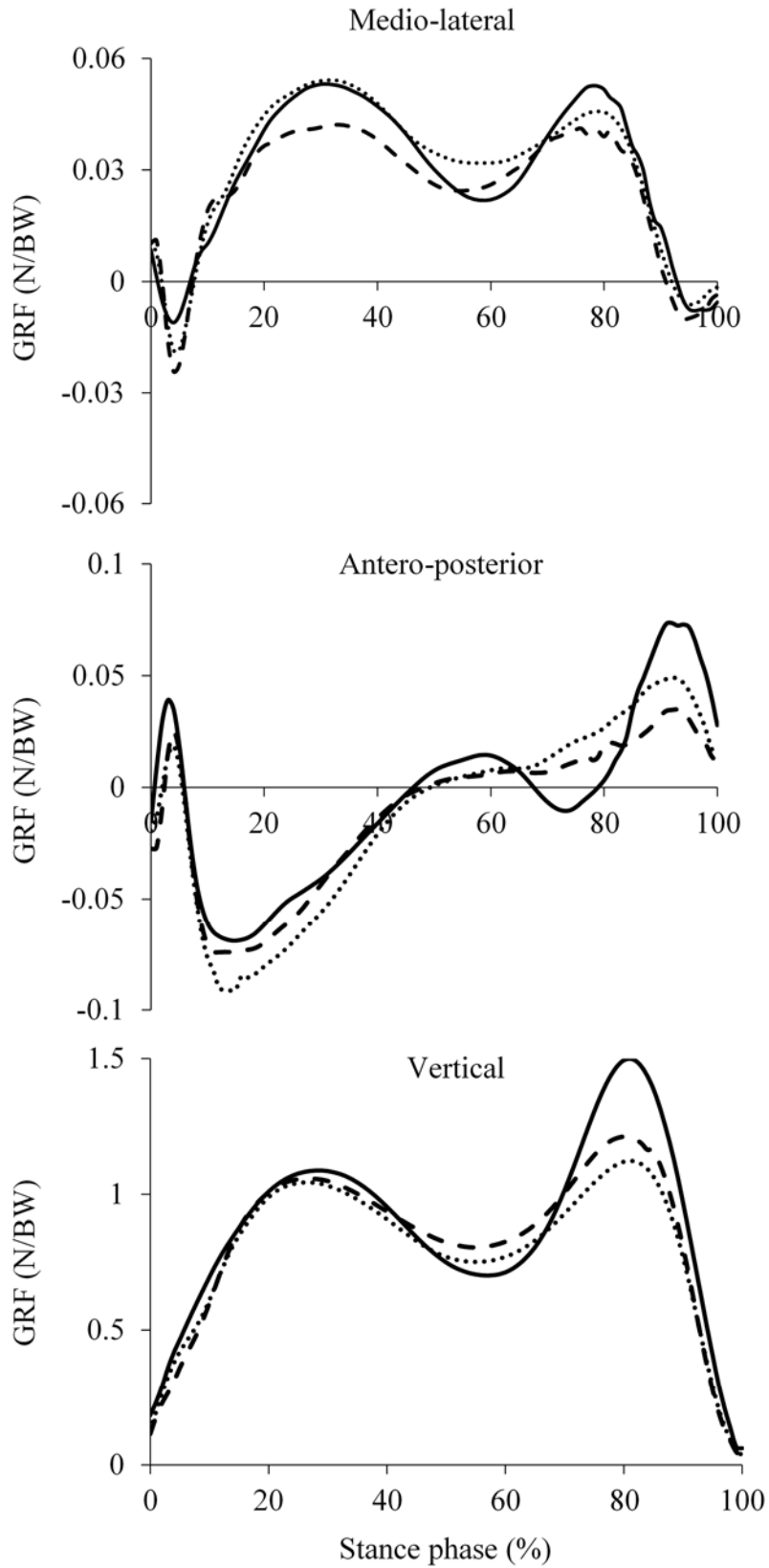


Figure 1. Group mean ground reaction forces per body weight (N/BW) for the claudication group (dotted line), asymptomatic limb group (dashed line) and control group (solid line) normalised to 100% stance phase. Positive direction indicates medial, anterior (propulsive) and vertical force.

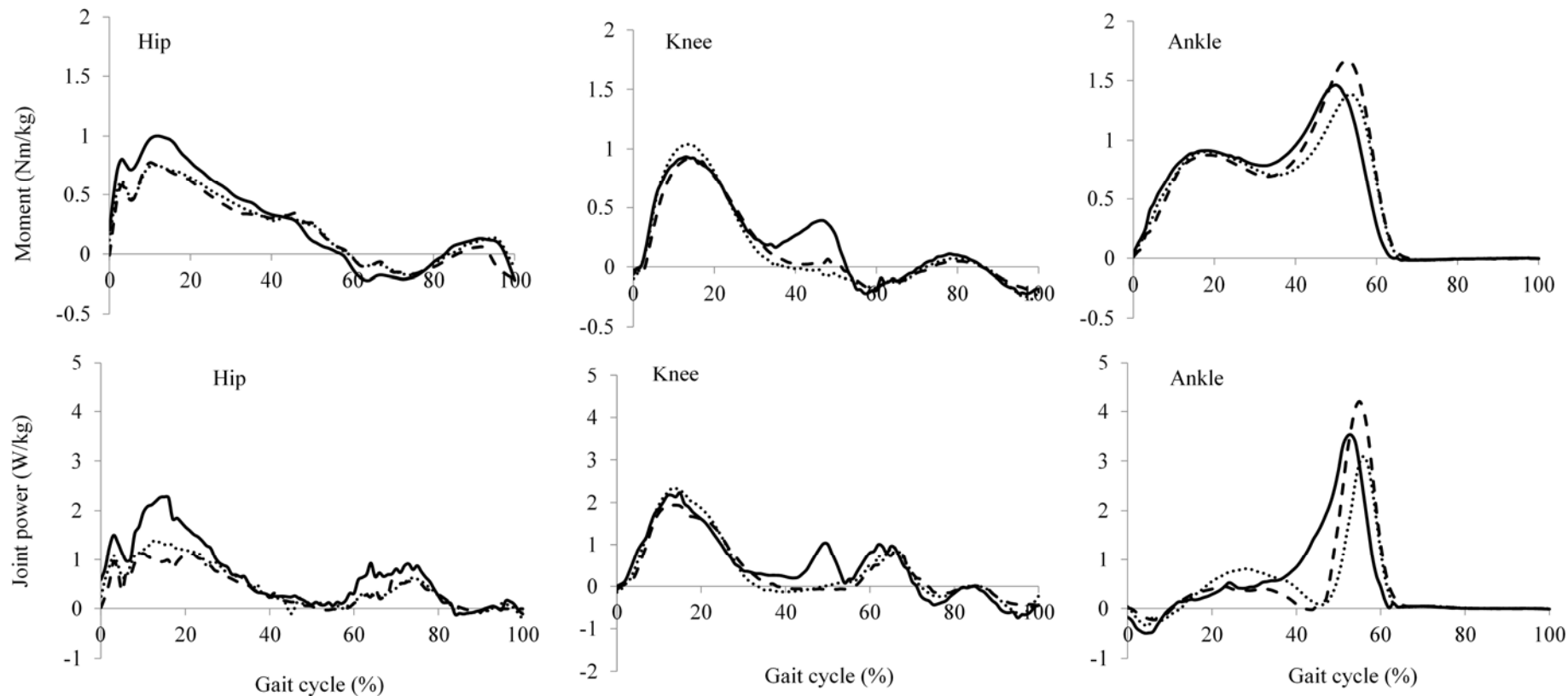


Figure 2. Group mean internal joint moment (top row) and joint power (bottom row) for the ankle, knee and hip across 100% gait cycle for claudicating-limb (dotted), asymptomatic-limb (dashed) and healthy controls (solid). Positive values indicate hip and knee extensor and ankle plantarflexor internal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

1
2

1 *Disease severity correlations*

2 Increased disease severity was associated with increased peak medial GRF ($R=-.630$, $P=.038$)
3 and trends towards reduced peak propulsive GRF during forward continuance ($R=.601$,
4 $P=.051$), reduced knee extension moment during weight acceptance ($R=.540$, $P=.086$), and
5 reduced peak knee angular velocity ($R=-.554$, $P=.077$). No association existed between overall
6 walking speed of unilateral and bilateral claudicants, and disease severity ($R=-.236$, $P=.511$).

7

8 **Discussion**

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

17

18 The observed trends towards reduced ankle power generation in the forward continuance phase
19 in the claudicating-limb group coincides with reduced angular velocity at peak moment, and
20 reduced propulsive and vertical forces. This could be explained by the previous observation of
21 reduced plantarflexor strength at high velocities¹⁷. Previous investigations into level gait
22 mechanics have reported reduced plantarflexor moments in claudicants compared to healthy
23 controls^{5,28,35}. However, those studies also observed slower walking speeds in those with PAD-
24 IC compared to healthy controls, therefore those previous findings may also reflect walking-

1 speed related gait differences and not solely reduced plantarflexor strength. A previous study
2 on level gait mechanics compared claudicants to speed-matched controls⁶ and the present study
3 statistically controlled for variance in walking speed, both of which observed reduced joint
4 powers in claudicants, not reduced moments. Therefore, velocity-dependent limitations in
5 claudicant plantarflexors appear a genuine adaptation. It seems that claudicants possess
6 adequate levels of strength when moving more slowly but are unable to exert that strength
7 when moving more quickly. Therefore, it could be suggested that the slower walking speed is
8 a strategy to allow claudicants to operate within safer limits relative to their maximal strength
9 capacity. Further investigation following a similar experimental protocol to Reeves *et al.*
10 (2009)¹⁹ would be required to confirm these inferences. Nonetheless, these findings highlight
11 the importance of maintaining plantarflexor strength, specifically plantarflexor power, in those
12 with PAD-IC. Reductions in maximum strength capacity, without the adoption of adequate
13 compensatory strategies, such as walking more slowly, would place excessive demands on the
14 functionally-limited plantarflexors, thus jeopardising the ability to accomplish this task safely.

15

16 It has previously been documented that healthy elderly adopt strategies to increase knee
17 extensor moment in late stance (forward continuance) just prior to peak ankle moment¹⁹. Those
18 authors suggested that was a mechanism to transfer energy from the proximal knee joint to the
19 distal ankle joint via the bi-articular gastrocnemii muscles, enabling a greater peak ankle
20 moment to be generated. A similar profile was exhibited in the present study by the healthy
21 elderly group (Figure 2) but not by the claudicants (either claudicating-limb group or
22 asymptomatic-limb group), indicating a functionally useful mechanism was not being utilised
23 by the PAD-IC patients. An alternative compensation could be an increase in contralateral hip
24 extensor activity during the mid-stance pull-up phase. However, no such increase in hip
25 moments or powers were observed for either the claudicating-limb or asymptomatic-limb

1 groups (Figure 2). The reasons for this are unclear, however reduced hip extensor strength has
2 previously been associated with increased disease severity and reduced functional mobility in
3 claudicants³⁶ and it may be that they lack adequate capacity to utilise such a compensation
4 irrespective of the disease presence uni- or bi-laterally. Alternatively, it may simply be that the
5 claudicants evade increased muscular effort in any lower limb muscle group as an attempt to
6 avoid earlier onset of claudication pain and increased metabolic cost³⁷. The lack of any
7 observable compensatory strategy may also contribute to the aforementioned slower walking
8 speed in claudicants as they are relying on the smaller, weaker and frequently symptomatic
9 plantarflexor group to perform this task.

10

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

21

22 The present study investigated pain-free stair ascent only. Plantarflexor function during level
23 gait deteriorates further in the presence of claudication pain^{5,38}. It would be reasonable to
24 assume that adaptations highlighted in the present study may be exacerbated detrimentally and

1 extrapolated further during painful locomotion, however future investigation is needed to
2 confirm this speculation. Whilst we statistically controlled for the influence of walking speed
3 in between-group analyses, a more vigorous investigation comparing claudicants to speed-
4 matched controls is required to fully explore the velocity-dependent limitations in the
5 gastrocnemii. Previous research has also demonstrated light handrail use influences gait
6 biomechanics³² and the present study excluded trials in which participants used the handrail as
7 the extent of handrail use and upper limb kinetics/force could not be quantified with our
8 staircase design. However, given the reported balance deficits in the population^{13,14}, handrail
9 use, particularly in the presence of claudication pain for example, may play a larger
10 compensatory role and requires further investigation.

11

12 **Conclusions**

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

21

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