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Patients with peripheral arterial disease exhibit reduced joint powers compared to velocity-matched controls

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ABSTRACT

Previous studies have shown major deficits in gait for individuals with peripheral arterial disease before and after the onset of pain. However, these studies did not have subjects ambulate at similar velocities and potential exists that the differences in joint powers may have been due to differences in walking velocity. The purpose of this study was to examine the joint moments and powers of peripheral arterial disease limbs for subjects walking at similar self-selected walking velocities as healthy controls prior to onset of any symptoms. Results revealed peripheral arterial disease patients have reduced peak hip power absorption in midstance (p = 0.017), reduced peak knee power absorption in early and late stance (p = 0.037 and p = 0.020 respectively), and reduced peak ankle power generation in late stance (p = 0.021). This study reveals that the gait of patients with peripheral arterial disease walking prior to the onset of any leg symptoms is characterized by failure of specific and identifiable muscle groups needed to perform normal walking and that these gait deficits are independent of reduced gait velocity.

Keywords:

Gait, Joint moments, Locomotion, Biomechanics, Vascular diseases

1. Introduction

Peripheral arterial disease (PAD) is the result of thickening and hardening of the arterial walls [1]. Intermittent claudication (IC) is the most common symptom of PAD, characterized by pain, cramping, aching and tiredness [2]. It is exacerbated by activities such as walking and relieved upon rest [2,3]. IC is associated with decreased physical activity, poor health outcomes, and increased dependence [1,4,5]. Spatial and temporal

measures of gait in PAD patients are abnormal. Specifically, PAD patients walk slower, take shorter and wider steps, and spend more time in double support than their healthy counterparts [3,6–11]. Recently, our group has shown that PAD patients walk with altered gait kinematics and kinetics prior to the onset of pain [3,6,7,12]. Specifically, the ankle takes longer to reach maximum dorsiflexion in late stance [8]. This limits the time for plantar flexion during propulsion. In addition, the ankle is unable to generate the power burst needed during push-off [3,7]. Decreases in ground reaction forces [6,13] as well as peak ankle plantar flexor moments and powers [3,14] have been documented, providing evidence of the inability of PAD patients to propel themselves at the end of the gait cycle. Further alterations in the gait cycle [9] demonstrate the significant gait impairment of PAD patients prior to the onset of pain, even in the unaffected legs of patients with unilateral disease [3,7].

These studies have provided valuable insight into the gait of PAD patients. However, these studies had PAD patients and healthy controls walking at their self-selected walking velocities, which were different between groups [3,6,7,13,14]. This provided the benefit of capturing the mechanics that the subjects would typically ambulate with, however, since walking velocity was significantly reduced for PAD patients in all of these studies, the true effect of walking velocity is not known. It is well established that the biomechanics of gait are dependent on the walking velocity [15–17]. Consequently, it is not entirely clear whether the alterations found in PAD gait are due to actual impairments in the lower limbs or an effect of a reduced walking velocity.

A similar issue has been present in the aging related research where elderly individuals walk with altered moments and powers at the hip, knee, and ankle joints compared to their healthy, younger counterparts [17,17–22]; but they also ambulate at slower velocities [20,23]. Devita and Hortobagyi [17] addressed the issue of walking velocity as a possible confounder of elderly gait alterations by having subjects walk at a controlled velocity. They found that ankle kinetics were reduced while the hip kinetics had slight increases, demonstrating a redistribution of forces from distal to proximal musculature. However, they had all subjects walk at a controlled velocity, which likely would not have been the natural speed for all individuals, thus causing possible altered mechanics from their natural gait.

The purpose of this study was to compare joint moments and powers of healthy controls and PAD patients walking at a similar self-selected walking velocity. We hypothesized that despite ambulating at the same velocity as healthy controls, differences in peak joint moments and powers in PAD patients would persist.

2. Methods

2.1. Subjects

Eighteen subjects (Table 1) diagnosed with PAD were recruited through the clinics of local medical centers. From the 18 PAD patients, twelve individuals had bilateral diagnosis and six had unilateral diagnosis. This resulted in 30 PAD affected

limbs included for analysis. In addition, 16 healthy age-, body mass-, and height-matched individuals (Table 1) were recruited through the community. PAD patients and healthy controls were screened for inclusion by a board-certified vascular surgeon. Screening evaluations included resting ankle-brachial index measurements; levels below 0.9 and symptomatic claudication were necessary for inclusion as a PAD subject. Ankle-brachial index is the ratio of systolic blood pressure at the dorsalis pedis and tibialis posterior arteries over the systolic pressure in the brachial artery. Detailed history, physical examination, visual observation and assessment of walking impairment were also performed. Those subjects with any cardiac, pulmonary, neuromuscular, or musculoskeletal conditions affecting gait were excluded. Subjects also experiencing any pain during ambulation other than IC were also excluded. No PAD patient had a history of previous revascularization. Control subjects underwent similar screening to the PAD patients. All subjects signed informed consent forms consistent with guidelines set forth by the Institutional Review Boards at the respective medical centers.

2.2. Experimental design and procedures

For data collection, all subjects came to the gait laboratory and wore a tight fitting spandex uniform to allow for precise motion capture. Retro-reflective markers were placed on anatomical locations on bilateral lower limbs and the pelvis such that a minimum of 3 markers were located on a single segment. This allowed calculations outlined by Vaughan et al. [24]. All PAD subjects were tested in a ''pain-free'' condition (prior to onset of any claudication symptoms in their legs). Three dimensional marker positions were recorded in real time with eight high-speed cameras (Motion Analysis Corp, Santa Rosa, CA) sampling at 60 Hz. In addition, ground reaction forces were collected with an embedded force platform (Kistler Instrument, Winterthur, Switzerland) sampling at 600 Hz. Subjects walked across a 10 m walkway for five successful trials for each limb. A trial was considered successful if only the single foot landed in the center of the force platform. Subjects were required to take a 1 min resting period between trials to assure no leg symptoms (claudication pain) occurred during trials. Throughout data collection trials, subjects were consistently asked if they were experiencing any pain or discomfort. Any such symptoms would result in an extended resting period until symptoms subsided. Following collection of data from PAD patients, healthy controls were selected from our database used for two recent studies examining PAD gait that utilized the same collection procedures (Table 1) [3,14]. The controls were matched to the PAD patients based on their self-selected walking velocities. This was done by calculating the PAD group average velocity. We then selected controls with a self-selected walking velocity 20% of the PAD group average velocity. This resulted in a range of control velocity of 1.00–1.51 m/s for the controls, which had similar distribution and range as the PAD group (range: 1.12–1.48 m/s). This allowed the study to control for walking velocities while allowing analysis of subjects' gait at self-selected velocity.

Table 1
Characteristics of healthy controls and peripheral arterial disease (PAD) patients suffering from symptomatic intermittent claudication.

Clinical characteristic	Control (n=32 limbs) Mean (SD)	PAD (n=30 limbs) Mean (SD)	p-Value
Age (yrs)	63.2 (13.2)	62.6 (9.8)	0.87
Body mass (kg)	83.8 (25.3)	79.3 (17.9)	0.54
Height (cm)	172.3 (7.6)	172.6 (7.3)	0.91
Body mass index	27.9 (6.8)	26.5 (5.1)	0.5
Ankle brachial index	n/a	0.54 (0.20)	n/a

2.3. Data analysis

Data from the three dimensional marker positions and ground reaction forces were combined to calculate joint moments and powers for three specific periods of the gait cycle: early stance (weight acceptance phase), mid stance (weight transfer phase), and late stance (weight propulsion phase). A low-pass fourth-order Butterworth filter with a 7 Hz cutoff frequency was used to smooth the marker position data. An inverse dynamics technique was implemented utilizing the kinematic data captured from the marker position and the kinetic data from the ground reaction forces [24]. Joint moments and powers were normalized to body weight and percentage of stance phase, i.e. heel strike (0% stance) to toe off (100% stance). Peak moments and powers were also identified. All calculations were done through custom software in Matlab (Matlab 2007, Mathworks Inc., Concord, Mass). Group differences were tested for significance using independent t-tests (a = 0.05).

3. Results

In early stance, PAD patients had a significantly lower amount of peak knee power absorption (p = 0.037; K1; Table 2). In midstance, PAD patients ambulated with significantly decreased peak hip power absorption compared to healthy controls (p = 0.017; H2; Table 2). In late stance, the PAD patients had significantly reduced peak power generation at the ankle (p = 0.021; A2; Table 2), as well as lower values for peak power absorption at the knee (p = 0.020; K3; Table 2).

4. Discussion

This study was the first to conduct a detailed biomechanical analysis of the kinetics of the lower extremities in PAD patients that walked at similar self-selected velocities as healthy matched controls. While other studies have successfully shown differences in gait kinetics between healthy individuals and PAD patients [3,6,9], those studies did not control for differences in walking velocity between groups, which has

been shown to affect biomechanical gait parameters and thus may have affected results. We hypothesized that despite ambulating at the same speed as healthy controls, differences in peak joint moments and powers in PAD patients would persist. Our results partially supported our hypothesis. PAD patients exhibited altered peak joint powers at the ankle, knee, and hip in different periods throughout the gait cycle despite walking at the same velocity as healthy matched controls. Peak joint moments, however, were not statistically different.

Table 2
Group means and standard deviations for peak joint moments and powers. Significant differences were found in peak joint powers at the ankle, knee, and hip at different times in stance phase of gait between PAD patients and healthy controls. ADM, ankle dorsiflexor moment; APM, ankle plantar flexor moment; KEM, knee extensor moment; KFM, knee flexor moment; HEM, hip extensor moment; HFM, hip flexor moment; A1, peak ankle power absorption early stance; A2, peak ankle power generation late stance; K1, peak knee power absorption early stance; K2, peak knee power generation early stance; K3, peak knee power absorption late stance; H1, peak hip power generation early stance; H2, peak hip power absorption midstance; H3, peak hip power generation late stance. Moments units: N m/kg; powers units: W/kg; velocity units: m/s.

	Control Mean (SD)	PAD Mean (SD)	p-Value
Moments			
ADM	−0.343 (0.103)	−0.376 (0.089)	0.195
APM	1.400 (0.270)	1.387 (0.190)	0.826
KEM	0.750 (0.225)	0.740 (0.210)	0.865
KFM	−0.132 (0.125)	−0.155 (0.154)	0.515
HEM	0.873 (0.236)	0.882 (0.202)	0.882
HFM	−1.056 (0.230)	−0.990 (0.279)	0.310
Powers			
A1	−0.470 (0.254)	−0.474 (0.225)	0.943
A2	2.998 (0.601)	2.677 (0.447)	0.021*
K1	−0.933 (0.373)	−0.760 (0.258)	0.037*
K2	0.473 (0.237)	0.402 (0.204)	0.214
K3	−0.899 (0.333)	−0.729 (0.211)	0.020*
H1	0.581 (0.235)	0.540 (0.197)	0.469
H2	−0.950 (0.270)	−0.788 (0.245)	0.017*
H3	0.689 (0.229)	0.695 (0.223)	0.914
Velocity	1.267 (0.124)	1.253 (0.104)	0.632

* Significance at $p < 0.05$.

The current study demonstrated that PAD patients had reduced peak power absorption at the knee (K1) during early stance compared to healthy controls (Table 1). The quadriceps provide power absorption in early stance by resisting the external flexor moment from the forward progression of the shank [25,26]. Reduced early stance peak

knee power absorption is consistent with our previous findings of excessive ankle plantarflexion in early stance [6,7], a mechanism that will reduce forward progression of the shank and thus reduce the need for power absorption. After the contralateral foot leaves the ground at toe-off, the individual is now in single support phase of gait and entering midstance. In midstance, PAD patients have reduced peak hip power absorption (H2). Hip power absorption in midstance is the result of the eccentric contraction of the hip flexors to control forward motion of the trunk.

We identified decreased peak ankle power generation (A2) and decreased peak knee power absorption (K3) during push-off, the period in the gait cycle when the contralateral leg becomes the leading leg and is now accepting the transfer of the person's weight. Our results are consistent with PAD findings during this phase as Scott-Pandorf et al. [13] reported a significantly decreased propulsion impulse in PAD patients. Similar to Koutakis et al. [3], the current study found decreased peak ankle power generation and decreased peak knee power absorption during push-off. Wurdeman et al. [30] similarly found decreased energy output during push-off in PAD patients.

Concerns have existed that previous findings of decreased gait function for patients with PAD were dependent upon differences in self selected walking velocities. The current study directly addresses this issue, confirming that decreased ankle power in PAD patients persists when matching velocities of ambulation between PAD patients and control subjects. The major finding from this study is that PAD patients ambulate with insufficient peak ankle power generation during push-off. This finding is consistent with previous studies where comparisons were performed between groups that did not walk at matched velocities [3,6,7,14]. The persistence of this finding further bolsters the severity of the problem. Walking models have shown that a weak or poorly timed push-off will result in an increased magnitude of negative work during weight acceptance of the contralateral limb which would then need to be reconstituted later in the gait cycle at the expense of an increased metabolic cost [27–29]. Our previous study has found this relationship to be true in PAD patients [30].

Although this study did not find differences in moments at the ankle, knee and hip, it is important to note that the peak moments do not necessarily occur at the same time as the peak powers. The calculation of moments and powers makes it possible that the peak moment could theoretically occur at the same time that power is zero if the angular velocity is zero. As such, differences in moments should still be considered as a possible cause of differences in peak power. However, future work needs to determine whether the peak power deficits are occurring as a result of reduced joint moments or angular velocities. The lack of significant differences in peak joint moments may offer insight into the mechanism behind typical slower walking velocities for PAD patients. Specifically, the diminished peak joint moments may be the reason PAD patients typically ambulate at slower velocity compared to healthy controls, and thus when the PAD patients are velocity matched to the small population of healthy controls that also

ambulate at slower velocities the difference no longer persists. In other words, if PAD patients could increase their peak joint moments then they would walk at velocities typical to healthy controls.

It is important to point out that our findings were found while PAD patients were walking in a "pain-free" condition. Thus, prior to onset of pain, patients are already experiencing problems. In previous studies, gait parameters such as peak joint moments and powers as well as kinematics and gait variability worsened with the onset of pain [6,7,12–14]. Future work needs to determine if gait deficits during "pain-free" walking highlight major neuro-muscular dysfunction of the locomotor system or if they are a possible adaptation to reduce muscle activity and oxygen need by certain muscle groups to prevent ischemic pain.

Our results are presented with the understanding of certain limitations to our study. First, we have included 18 PAD patients for analysis. Twelve of these individuals had bilateral diagnoses and both limbs were included for analysis, whereas six individuals had unilateral PAD diagnoses and only the affected limb was included. The inclusion of limbs from bilateral and unilateral diagnoses may influence results since patients with unilateral diagnosis have a leg that is considered not affected. This may provide an opportunity for compensations from this leg. However, our previous studies have shown deficits in the non-affected limb similar to the affected limb in unilateral PAD patients [3,30], which may limit the potential compensatory mechanisms from the non-affected limb. Another limitation to consider is the possibility for selection bias among our control subjects. Specifically, to match our controls' self-selected walking velocities with our PAD patients, the healthy controls selected from our previous studies were the "slower" walkers in our database of healthy controls. This fact, combined with our previous findings without matched velocities [3,14] adds to the speculation that diminished joint moments in PAD patients seems to be a cause of generally slower preferred walking velocities.

In conclusion, our results confirm our previous studies demonstrating significant reduction in ankle power in late stance for PAD patients that are independent of walking velocity. As the primary contributor to push-off, the biomechanical abnormality measured at the ankle joint should be the main target of clinical attempts at restoration of gait and provides a clear indicator for measuring interventions directed at PAD patients. Restoration of ankle plantar flexor function could result in improved closed chain kinetics throughout the entire stance limb that may actually alleviate deficits occurring at more proximal joints. Finally, future studies should detail the mechanisms that are being utilized by PAD patients to maintain similar velocities despite altered joint Powers.

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Conflict of interest statement

The authors do not have any potential conflicts of interest.

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