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

Shane R. Wurdeman [a,b], Panagiotis Koutakis [c], Sara A. Myers [a], Jason M. Johanning [c], Iraklis I. Pipinos [c], Nicholas Stergiou [a,b,*]

[a] Nebraska Biomechanics Core Facility, University of Nebraska at Omaha, Omaha, NE, United States
[b] College of Public Health, University of Nebraska Medical Center, Omaha, NE, United States
[c] Department of Surgery, University of Nebraska Medical Center and Veterans Affairs Medical Center, Omaha, NE, United States

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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.

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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

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* Corresponding author at: Nebraska Biomechanics Core Facility, University of Nebraska at Omaha, Omaha, NE 68182-0216, United States. Tel.: +1 402 554 3247.
E-mail address: nstergiou@unomaha.edu (N. Stergiou).

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, and 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.

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 ($\alpha = 0.05$).

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 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

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 $p < 0.05$.