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Abnormal joint powers before and after the onset of claudication symptoms

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Objective: **Claudication is the most common manifestation of peripheral arterial disease, producing significant ambulatory compromise. Our study evaluated patients with bilateral lower limb claudication and characterized their gait abnormality based on advanced biomechanical analysis using joint torques and powers.**

Methods: **Twenty patients with bilateral claudication (10 with isolated aortoiliac disease and 10 with combined aortoiliac and femoropopliteal disease) and 16 matched controls ambulated on a walkway while 3-dimensional biomechanical data were collected. Patients walked before and after onset of claudication pain. Joint torques and powers at early, mid, and late stance for the hip, knee, and ankle joints were calculated for claudicating patients before and after the onset of claudication pain and were compared to controls.**

Results: **Claudicating patients exhibited significantly reduced hip and knee power at early stance (weight-acceptance phase) due to decreased torques produced by the hip and knee extensors. In mid stance (single-limb support phase), patients had significantly reduced knee and hip power due to the decreased torques produced by the knee extensors and the hip flexors. In late stance (propulsion phase), reduced propulsion was noted with significant reduction in ankle plantar flexor torques and power. These differences were present before and after the onset of pain, with certain parameters worsening in association with pain.**

Conclusions: **The gait of claudication is characterized by failure of specific and identifiable muscle groups needed to perform normal walking (weight acceptance, single-limb support, and propulsion). Parameters of gait are abnormal with the first steps taken, in the absence of pain, and certain of these parameters worsen after the onset of claudication pain. (J Vasc Surg 2010;52:340-7.)**

Intermittent claudication is the most common clinical manifestation of peripheral arterial disease (PAD), presenting as exercise-induced leg muscle pain and gait dysfunction.[1] Claudication and its associated ambulatory impairment produce impaired quality of life,[2] physical dependence,[3] and poor health outcomes.[4] Previous work suggests that PAD patients walk slower, with decreased cadence, increased stance time, shorter stride length, and a narrower step width than healthy controls.[5,6] However, these changes alone are unable to describe in sufficient detail the locomotor impairments of claudicating patients or aid in our understanding of its underlying pathophysiology.

A more detailed quantitative evaluation of gait can be obtained using advanced biomechanical analysis that includes joint torques and powers.[7,8] Although muscles produce linear forces, motions at joints are all rotary. The rotary torque is a measure of the tendency of a force to rotate the limb around a joint and is calculated as the product of the muscle force and the distance from the joint center that the force is being applied. The net muscle torque does not represent any one particular muscle but rather describes the net activity of all the muscles acting across a joint.

Joint power can be defined as the rate of work produced by muscles contracting to move a joint and is determined as the product of the net torque (moment) of the muscles acting across a joint and the resulting angular velocity of the joint. Joint powers have been used extensively to identify the mechanisms responsible for pathologic gait in populations such as elderly patients and those with knee and hip arthritis and arthroplasty and anterior cruciate ligament reconstruction.[8-10] Joint powers can also assess and guide successful rehabilitation strategies.[10] Similar insights can be gained from patients with PAD by using this approach.

Previous studies of claudicating PAD patients from our laboratory using basic biomechanical analysis[7,11-13] suggested a potential weakness of the posterior compartment muscles of the hip and calf as key components of PAD gait impairment. The purpose of the current study was to use joint torques and powers to isolate and identify the individual muscle compartments responsible for the gait impairment of claudicating patients.

METHODS

This study was approved by the Institutional Review Boards of the Western Iowa and the University of Nebraska

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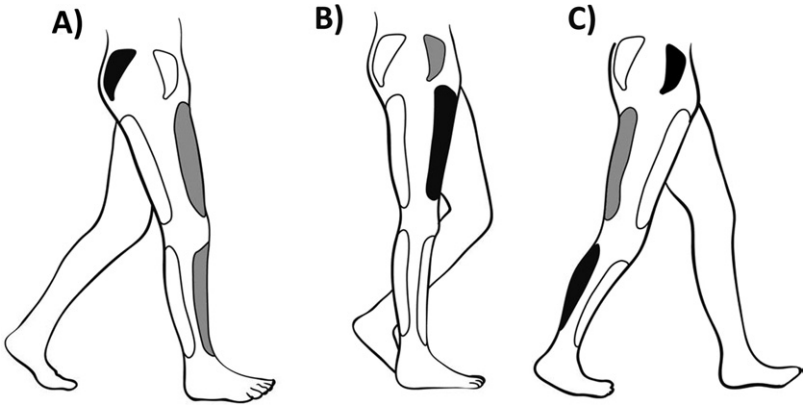

Fig 1. An illustration of the three phases of walking with the dominant flexor and extensor muscle groups that are involved in each phase based on the joint torques generated. The dominant muscle groups are identified in *black* if they contract concentrically (muscle shortens as it contracts) and in *grey* if they contract eccentrically (muscle lengthens as it contracts). **A,** Weight-acceptance phase, also known as early stance, initial contact, and heel-strike phase, lasts from ipsilateral heel strike to contralateral toe-off, thus covering the first double-support phase (initial 20% of stance). The right leg is accepting most of the weight of the body as it descends from previously being in single support on the left leg. The right hip extensors concentrically contract to extend the hip (reflected in the HET torque and H1 power in Tables III and IV), the knee extensors eccentrically contract to allow the knee to bend (reflected in the KET torque and K1 power in Tables III and IV), and the ankle dorsi flexors eccentrically contract to maintain ankle dorsi flexion (reflected in the ADT torque and A1 power in Tables III and IV). **B,** Single-limb support phase, also known as the mid-stance phase, lasts from contralateral (here left) toe-off until contralateral heel strike. During single support, the center of mass of the body is at its highest point and over the extended ipsilateral (here right) leg. The body has maximum potential energy getting ready to fall forward for the next double support. Limited muscular contractions are needed during this phase except its early part where the knee extensors contract concentrically to extend the knee and straighten the leg (reflected in the KET torque and K2 power in Tables III and IV) and the late part where the hip flexors contract eccentrically to control the movement of the pelvis (reflected in the HFT torque and H2 power in Tables III and IV). **C,** Propulsion phase, or the late stance or toe-off phase, lasts from contralateral heel strike to ipsilateral toe-off. It is the last 20% of stance and the second-double support phase. The body is propelled forward onto the extended left leg mainly by the action of the right ankle plantar flexors (posterior calf compartment muscles, the most important of which are the gastrocnemius and soleus). Functionally, these muscles contract concentrically (reflected in the APT torque and A3 power in Tables III and IV) and accelerate the trunk forward and upward over the left leg (ie, providing forward progression and weight support). In this phase, the knee flexors eccentrically contract, controlling the knee movement (reflected in the KFT torque and K3 power in Tables III and IV) and the hip flexors concentrically contract (reflected in the HFT torque and H3 power in Tables III and IV) to assist the ankle plantar flexors to accelerate the trunk forward and lift the leg over the ground into the swing phase.

Medical Centers. All individuals signed an inform consent before they participated in the study.

Key events during gait stance. The gait cycle (from heel touchdown to heel touchdown) consists of a stance and a swing period. The stance phase is the most important segment of the gait cycle because the ambulating limb accepts, supports, and propels forward the weight of the body and is the only portion of the gait cycle that can be accurately evaluated for joint moments and powers. The stance segment can be divided in three distinct phases: the weight-acceptance, the single-limb support, and the propulsion phases (Fig 1).

Participant inclusion and exclusion criteria. The study recruited 20 patients diagnosed with moderate arterial occlusive disease and bilateral claudication (Fontaine stage II and Rutherford grade 1 categories 2 and 3)[14] from the vascular surgery clinics of the VA Nebraska and Western Iowa and University of Nebraska Medical Centers. All 20 PAD patients had aortoiliac occlusive disease (10 with isolated aortoiliac disease and 10 with femoropopliteal disease.) The diagnosis of the level of disease was made with the use of computed tomography angiography and ultrasound evaluation.

In addition, 16 healthy controls were recruited, matched for gender, age, body mass, and height. The controls were matched to PAD patients by group/frequency matching. Patients and controls were screened and evaluated by two board-certified vascular surgeons.

The study excluded PAD patients with ambulation-limiting cardiac, pulmonary, neuromuscular, or musculoskeletal disease, or those who experienced pain or discomfort during walking for any reason other than claudication (ie, arthritis, low back pain, musculoskeletal problems, and neuropathy). Patient evaluation included resting ankle-brachial index (ABI; a measurement <0.90 was present in all claudicant patients), detailed history, physical examination, and direct assessment and observation of the patient's walking impairment. A vascular surgeon observed the pa-

tient walking and recorded all symptoms and signs affecting ambulation to ensure limitation was secondary to claudication pain.

Controls had a resting ABI >0.90 and no subjective or objective ambulatory dysfunction. Controls were screened in a similar fashion as PAD patients and were excluded for the same ambulation-limiting problems or if pain was experienced during walking. The gait of all recruited participants was tested in the Biomechanics Laboratory of the University of Nebraska at Omaha.

Experimental procedure and data collection. Before data collection, reflective markers were placed at specific anatomic locations of each participant's lower limb using the modified Helen Hayes marker set.[12] Each participant was directed to walk with a self-selected pace over a 10-meter pathway, while 3-dimensional marker trajectories (kinematics) and ground reaction forces (kinetics) were simultaneously collected. The marker trajectories were captured with an eight high-speed real-time camera system (EvaRT 5.0, Motion Analysis Corp, Santa Rosa, Calif) sampling at 60 Hz. The ground reaction force data were acquired with a Kistler force platform sampling at 600 Hz.

Each patient was tested before ("pain-free" condition) and after ("pain" condition) the onset of claudication pain. During pain-free testing, mandatory rest occurred between the walking trials to ensure that all trials were in a pain-free condition. Once patients completed all pain-free trials, the pain trials were performed. Each patient was asked to walk on an inclined treadmill with 10% grade at a speed of 0.67 m/s[15] until claudication pain was established. The patients were then immediately returned to the collection walkway to acquire the data for the pain condition without the mandatory resting periods. Controls completed only the pain-free condition trials. Five walking trials were collected from each leg of the participants for each condition.

Data analysis. Joint kinetics and kinematics were calculated for the sagittal plane during the stance phase of walking. An inverse dynamic solution was performed to calculate joint muscle torques and powers from the joint kinetics and kinematics.[8] Joint muscle power (P_j) is calculated as the product of the net torque of force at a joint (T_j) and the relative joint angular velocity (ω_j) or $P_j = T_j \times \omega_j$ (Joules/s or Watts). Power combines kinetic (forces) and kinematic (angles and velocities) information and can be expressed positively or negatively. Positive power indicates that energy is being generated and negative power that energy is being absorbed by the muscle group under study. Thus, positive joint muscle power is associated with concentric muscular contractions, and negative power is associated with eccentric muscular contractions.[8]

Joint torques and joint muscle powers were normalized with respect to the participant's body mass and expressed as a percentage of the stance phase. The peak values for extensor and flexor torques were identified for the ankle, knee, and hip joints.[8] The variables identified were the ankle dorsi flexor torque (ADT) in early stance and the ankle plantar flexor torque (APT) in late stance for the ankle, the knee extensor torque (KET) in early stance and the knee flexor torque (KFT) in late stance for the knee, and the hip extensor torque (HET) in early stance and the hip flexor torque (HFT) in late stance for the hip.

The peak values for power absorption (eccentric contraction) and generation (concentric contraction) were identified for the ankle, knee, and hip joints.[8] The power variables identified were the power absorption in early (A1) and mid (A2) stance and the power generation in late stance (A3) for the ankle; the power absorption in early stance (K1), the power generation in the early part of mid-stance (K2), and the knee power absorption in late stance (K3) for the knee, and the power generation in early stance (H1), the power absorption in mid stance (H2), and the power generation in late stance (H3) for the hip joint.[8] Custom-made Matlab (Matlab 2008b, Mathworks Inc, Concord, Mass) software was used to calculate the joint torques and powers.

Statistical analysis. Data were summarized by group means and standard deviations of the peak joint torques and powers. These were calculated for each testing condition for controls and for pain-free and pain conditions for claudicating patients by first averaging the limb measurements for each individual and then averaging the obtained averages overall for the 20 patients in the claudicating group and, respectively, for 16 controls.

When the effects of predictors on response variables were modeled, repeated measures models were developed where the unit of analysis was the limb (not participant), and the individual's two limbs were "nested" in him or her to account for possible correlation between the two limbs in a person. Three separate comparisons were performed: (1) Controls vs claudicating patients in pain-free condition; (2) controls vs claudicating patients in pain condition; and (3) claudicating patients in pain-free condition vs claudicating patients in pain condition. For comparisons 1 and 2, linear models with repeated measures on variable leg (left vs right) were used; whereas for comparison 3, linear models with doubly repeated measures on variables leg (left vs right) and pain status (before pain vs after pain) were used.

In addition to the main effects of group and leg for 1 and 2, or pain status and leg for 3, first-order interaction terms were also included in the models. Additionally, in all models we controlled for variable ABI and, particularly for comparison 3, we also controlled for variable level of disease (aortoiliac only vs aortoiliac plus femoropopliteal).

The concrete values for P were always reported. The level of significance was set to 0.05. The procedure MIXED in the SAS 9.2 software (SAS Institute, Cary, NC) was used to develop all linear models.

RESULTS

Temporal and spatial gait measurements. The baseline clinical characteristics of patients and healthy controls are reported in Table I. No significant differences were found between groups for age, body mass, and height. When compared with controls, PAD patients had significantly decreased gait velocity, stride length, and step

Table I. Baseline characteristics of peripheral arterial disease patients and healthy controls

Clinical characteristics	*PAD (n = 20)*	*Control (n = 16)*
Gender		
Male	19	15
Female	1	1
Age, mean ± SD, y	60.25 ± 7.21	62.81 ± 12.01
Body mass, mean ± SD, kg	82.55 ± 18.05	81.79 ± 20.99
Body height, mean ± SD, m	1.73 ± 0.07	1.73 ± 0.08
Disease duration, mean ± SD, y	4.01 ± 2.18	0
ABI, mean ± SD	<0.9	>0.9
Right limb	0.58 ± 0.22	1.11 ± 0.05
Left limb	0.57 ± 0.18	1.10 ± 0.04
Smokers, No. (%)	15 (75)	0 (0)
Hypertension, No. (%)	14 (70)	2 (12.5)
Diabetes mellitus, No. (%)	1 (5)	0 (0)
Hyperlipidemia, No. (%)	15 (75)	2 (12.5)
BMI, mean ± SD, kg/m^2	27.72 ± 5.35	27.11 ± 5.32

ABI, Ankle-brachial index; *BMI,* body mass index; *PAD,* peripheral arterial disease; *SD,* standard deviation.

length, and increased step width (Table II). The differences for these parameters remained significant when the patients walked, experiencing muscle pain in the pain condition (Table II). A comparison of temporal and spatial gait measurements before and after onset of claudication showed a significant decrease in gait velocity (Table II).

Weight-acceptance phase. Compared with the controls, patients in the pain-free condition generated significantly decreased torque by the hip extensors (HET) and by the knee extensors (KET), which translated to significantly decreased power at the hip (H1, reduced concentric contraction of the hip extensors) and at the knee (K1, reduced eccentric contraction of the knee extensors; Tables III and IV). Decreased (although not significant) power absorption was produced at the ankle (A1, reduced eccentric contraction of the ankle dorsi flexors). In the pain condition, the results for the knee extensor torque (KET), the concentric contraction of the hip extensors (H1), and the eccentric contraction of the ankle dorsi flexors (A1) remained significantly different compared with controls (Table III).

Single-limb support or mid-stance phase (all the body weight on one limb). Compared with the controls, patients in pain-free and pain conditions generated significantly decreased torque by the knee extensors (KET) and the hip flexors (HFT), which then translated to significantly decreased knee joint power generation in the early part of the mid-stance phase (K2, reduced concentric contraction of the knee extensors) and decreased hip power absorption in the late part of mid-stance (H2, reduced eccentric contraction of the hip flexors; Tables III, IV).

Propulsion or late-stance phase. Compared with the controls, patients in pain-free and pain conditions generated significantly decreased torque by the ankle plantar flexors (APT) and the hip flexors (HFT), which translated to significantly less power at the ankle (A3, reduced concentric contraction of the ankle plantar flexors) and the hip (H3, reduced concentric contraction of the hip flexors in the pain condition; Tables III, IV). Patients in both conditions also absorbed significantly less power at the level of the knee flexors (K3, reduced eccentric contraction of the knee flexors). Once in the pain condition, patients generated significantly decreased ankle plantar flexor torque (APT) than in the pain-free condition. This significant decrease in torque (reduced concentric contraction of the ankle plantar flexors) translated into further decrease of power generation at the ankle (A3).

DISCUSSION

The purpose of this study was to use joint torques and powers to characterize and provide an in depth understanding of the gait impairment of claudicating patients. Joint torques and powers were measured while patients walked, with and without claudication pain, and were compared with those of healthy controls matched for gender, height, body mass, and age. Our results from the temporal and spatial gait parameters demonstrate that the character of the PAD gait is overall "sluggish and tired." Patients with claudication have decreased gait velocity, decreased stride, and step length, and increased step width. These findings are in agreement with previous studies and unequivocally document the abnormal temporal and spatial gait parameters of claudicating PAD patients.[5,6]

By using advanced biomechanical analysis in the form of joint torques and powers, we were able to isolate and describe the specific muscle group impairments that operate to produce the gait deficit in claudicating patients. Our data demonstrate a decreased ability of the knee and hip extensors to control weight acceptance and of the ankle dorsi flexors to eccentrically control the lowering of the foot to the ground after heel strike in early stance. In mid stance we found a decreased ability of the knee extensors to concentrically extend the knee and of the hip flexors to eccentrically control the movement of the pelvis. Finally, in late stance we demonstrated a decreased ability of the ankle plantar flexors and of the hip flexors to concentrically propel the body forward and of the knee flexors to eccentrically control knee hyperextension as the trunk is accelerated forward.

Decreased weight acceptance. Trunk support in early stance is provided by the hip extensors concentrically contracting to extend the hip (H1), the knee extensors eccentrically contracting to allow the knee to flex (K1), and the ankle dorsi flexors, which eccentrically control the movement of the foot towards full contact with the ground (A1).[16] Our findings in PAD patients demonstrate that all three muscle groups involved in the weight-acceptance phase produce less power than in controls. Specifically PAD patients have decreased power generation by the hip extensors (gluteus muscles, H1) and the knee extensors (quadriceps) in early stance (K1), indicating decreased ability to support the body weight. These power results are sup-

Table II. Group means and standard deviations for the temporal and spatial gait measurements for peripheral arterial disease *(PAD)* and control groups

Measurement	*Control (N = 16)*	*PAD (N = 20)*				
		PAD-PF	P[a]	*PAD-P*	P[b]	P[c]
Gait velocity, m/s	1.28 ± 0.13	1.14 ± 0.10	.0051	1.09 ± 0.13	.0007	.042
Stride length, m	1.47 ± 0.11	1.30 ± 0.13	<.001	1.27 ± 0.11	<.001	.526
Cadence, steps/min	106.41 ± 7.45	101.03 ± 8.44	.053	101.58 ± 7.82	.051	.256
Step length, m	0.68 ± 0.05	0.64 ± 0.06	.013	0.61 ± 0.05	.011	.132
Step width, m	0.13 ± 0.03	0.15 ± 0.03	.045	0.15 ± 0.04	.050	.385
Stance phase, % of gait cycle	62.25 ± 4.45	61.80 ± 4.26	.356	63.20 ± 4.10	.367	.236
Swing phase, % of gait cycle	37.75 ± 2.11	38.20 ± 3.70	.678	36.80 ± 3.33	.308	.448
Double support, % of gait cycle	12.60 ± 2.91	12.93 ± 1.76	.453	13.04 ± 2.23	.274	.499

PAD-P, Pain condition; *PAD-PF,* pain-free condition.
[a]Differences between PAD-PF and control ($P < .05$ is significant).
[b]Differences between PAD-P and control ($P < .05$ is significant).
[c]Differences between PAD-PF and PAD-P ($P < .05$ is significant).

Table III. Group means and standard deviations (N × m/kg) for joint torques of the ankle, knee, and hip joint for peripheral arterial disease *(PAD)* and control groups

Variable	*Control (N = 16)*	*PAD (N = 20)*				
		PAD-PF	P[a]	*PAD-P*	P[b]	P[c]
ADT	−0.361 ± 0.101	−0.332 ± 0.103	.3559	−0.317 ± 0.087	.1526	.3920
APT	1.356 ± 0.138	1.289 ± 0.129	.0348	1.225 ± 0.143	.0038	.0416
KET	0.746 ± 0.186	0.514 ± 0.291	.0059	0.569 ± 0.241	.0167	.2367
KFT	−0.137 ± 0.128	−0.197 ± 0.241	.3349	−0.185 ± 0.221	.4149	.4720
HET	0.901 ± 0.248	0.756 ± 0.157	.0218	0.810 ± 0.159	.1402	.1009
HFT	−1.061 ± 0.231	−0.875 ± 0.285	.0033	−0.851 ± 0.177	.001	.2078

ADT, Ankle dorsi flexor torque in early stance; *APT,* ankle plantar flexor torque in late stance; *HET,* hip extensor torque in early stance; *HFT,* hip flexor torque in late stance; *KET,* knee extensor torque in early stance; *KFT,* knee flexor torque in late stance; *PAD-P,* pain condition; *PAD-PF,* pain-free condition.
[a]Differences between PAD-PF and control ($P < .05$ is significant).
[b]Differences between PAD-P and control ($P < .05$ is significant).
[c]Differences between PAD-PF and PAD-P ($P < .05$ is significant).

Table IV. Group means and standard deviations (in W/kg) for joint powers of the ankle, knee, and hip joint for peripheral arterial disease *(PAD)* and control groups

Variable	*Control (N = 16)*	*PAD (N = 20)*				
		PAD-PF	P[a]	*PAD-P*	P[b]	P[c]
A1	−0.650 ± 0.295	−0.412 ± 0.210	.061	−0.375 ± 0.192	.0171	.321
A2	−0.550 ± 0.143	−0.558 ± 0.171	.4584	−0.549 ± 0.308	.4378	.648
A3	2.957 ± 0.686	2.437 ± 0.445	.0045	2.178 ± 0.510	.0001	.022
K1	−0.986 ± 0.387	−0.645 ± 0.369	.0066	−0.731 ± 0.452	.0647	.273
K2	0.527 ± 0.305	0.293 ± 0.213	.0059	0.337 ± 0.244	.0321	.145
K3	−0.882 ± 0.324	−0.622 ± 0.206	.0028	−0.580 ± 0.249	.0015	.376
H1	0.604 ± 0.252	0.458 ± 0.161	.0316	0.478 ± 0.165	.023	.493
H2	−0.937 ± 0.263	−0.665 ± 0.207	.0003	−0.699 ± 0.205	.001	.338
H3	0.706 ± 0.237	0.603 ± 0.182	.1065	0.569 ± 0.175	.0332	.128

A1, Ankle power absorption in early stance; *A2,* ankle power absorption in mid stance; *A3,* ankle power generation in late stance; *H1,* hip power generation in early stance; *H2,* hip power absorption in mid stance; *H3,* hip power generation in late stance; *K1,* knee power absorption in early stance; *K2,* knee power generation in early mid stance; *K3,* knee power absorption in late stance; *PAD-P,* pain conditions; *PAD-PF,* pain-free condition.
[a]Differences between PAD-PF and control ($P < .05$ is significant).
[b]Differences between PAD-P and control ($P < .05$ is significant).
[c]Differences between PAD-PF and PAD-P ($P < .05$ is significant).

ported by our joint torque findings, which showed significantly decreased torque development by the hip (HET) and knee (KET) extensors.[12] The demonstrated weakness of the hip and knee extensors in early stance results in diminished ability for weight acceptance and control of forward momentum when a claudicating patient walks. The demonstrated weakness of the ankle dorsi flexors in early stance is in agreement with findings our group has previ-

ously published showing that PAD patients have a "foot drop" upon heel strike.[11]

Decreased weight support during the mid-stance phase. In the early part of the mid-stance phase, the knee extensors concentrically contract to extend the knee joint, and in the late part, the hip flexors contract eccentrically to control the movement of the pelvis during single-leg support. To maintain the energy required for walking,[16,17] it is necessary to straighten the leg and stabilize the pelvis to maximize the ability to generate potential energy at the highest point of the body's center of mass during the gait cycle. Our work shows that both muscle groups (knee extensors [K2] and hip flexors [H2]) involved in single-limb support produce less power in PAD patients than in controls.

Decreased forward propulsion. In late stance, the body is propelled forward mainly by the action of the ankle plantar flexors. Functionally, these muscles contract concentrically and accelerate the leg and the trunk forward to initiate swing, while decelerating the downward motion of the trunk (ie, providing forward progression and support).[18] Our results in the PAD patients demonstrate that power generation by way of concentric contractions of the ankle plantar flexor muscles in late stance (A3) is decreased in the pain-free condition and worsens in the pain condition. This hypothesis is supported by previous findings from our and other laboratories demonstrating that PAD patients have significantly decreased ankle plantar flexor torques[7,12] and strength.[13] Our additional findings of decreased knee and hip flexor powers further demonstrate the failure of the PAD limb to appropriately use these muscle groups to assist the ankle plantar flexors to accelerate the trunk forward.

Potential clinical implications for the observed gait abnormalities. Our findings for the temporal and spatial gait parameters provide definitive evidence of abnormal temporal and spatial parameters in patients with PAD and confirm the generally accepted thought that claudicating patients have an abnormal gait[5] that leads to increased energy cost and earlier fatigue.[19] More importantly, our advanced analysis with joint torques and powers provides a more detailed delineation of the gait disturbance than that previously documented by our group and others using spatial (joint angles)[11,12,20,21] and temporal (velocity, cadence, and step/stride characteristics) parameters.[5,6,20]

When the abnormalities of joint torques and powers in PAD are compared with other conditions, our values are in line with those of healthy elderly patients and elderly patients with osteoarthritis.[22-25] In contrast, however, the gait biomechanics of PAD patients appear to be significantly worse than healthy elderly individuals and those with severe arthritis. Specifically, our data demonstrate that from the first few steps they take and before they experience any muscle pain, claudicating patients walk with a 26% decrease (vs controls) of their ankle plantar flexor power compared with a 13% for elderly osteoarthritis patients.[26] Arthritis patients compensate for the 13% decrease in the power of their ankle plantar flexors by increasing the power of their knee and hip extensors by 13% and 28%, respectively.[23] In marked contrast to the arthritic patients, claudicating patients demonstrate a drop of compensatory power in these muscle groups by 39% and 14%, respectively.[23] From a biomechanical standpoint, claudication produces a considerably worse functional limitation than osteoarthritis. Our data support the findings that claudicating patients typically gather around the extreme low end of the physical activity spectrum[27] and experience a severe decline in all domains of physical function.[6,28]

Potential mechanisms for the observed gait abnormalities. The present data demonstrate significant abnormalities in the gait of claudicating patients that are present at the initiation of ambulation and before the onset of pain. Work of other investigators evaluating PAD patients has shown that tissue oxygen levels and blood flow in the leg during the first few steps of walking, are very similar to (and sometimes even higher than) those of controls.[29-31] These results together with our finding of gait impairments present within the first few steps taken by the patient suggest that a mechanism other than blood flow is responsible for a significant portion of the limb dysfunction of claudication. This conclusion is supported by the work of McDermott et al,[32] who measured muscle strength in PAD patients using a 5-second maximal isometric strength assessment. This test, which is independent of blood flow, demonstrated that muscle strength in PAD patients is significantly reduced compared with controls, again indicating that blood flow is not the only determinant of the dysfunction of claudicating muscle.

These baseline biomechanical impairments likely reflect a muscle metabolic myopathy and an axonal polyneuropathy in the lower extremities of patients with PAD.[33-35] Specifically, a number of reports have documented a metabolic myopathy in the PAD muscle that is related to defective mitochondrial bioenergetics, oxidative damage, and inflammation in the skeletal muscle[34,36-39] Furthermore, there is accumulating evidence suggesting that chronic ischemia in patients with PAD results in a consistent pattern of electrodiagnostic abnormalities indicating axonal nerve loss.[35,37]

Our data further demonstrate that certain baseline impairments worsen after the onset of claudication pain. This reflects exercise-induced ischemia and increased workload, restricting lower extremity bioenergetics and producing muscle pain. Because of this ischemia-induced neuromyopathy, patients with claudication become severely debilitated and adopt a sedentary lifestyle characterized by limited use of the myopathic limb, which may exacerbate the neuromyopathy. Fig 2 illustrates a proposed pathway linking these basic pathophysiologic mechanisms (exercise-associated ischemia-reperfusion, myopathy, neuropathy) with the specific biomechanic deficits identified in this work. The involvement by each one of these mechanisms and the way they are related to the clinical biomechanical findings of leg dysfunction should be the focus of intense future investigation and may hold the key to understanding PAD pathophysiology.

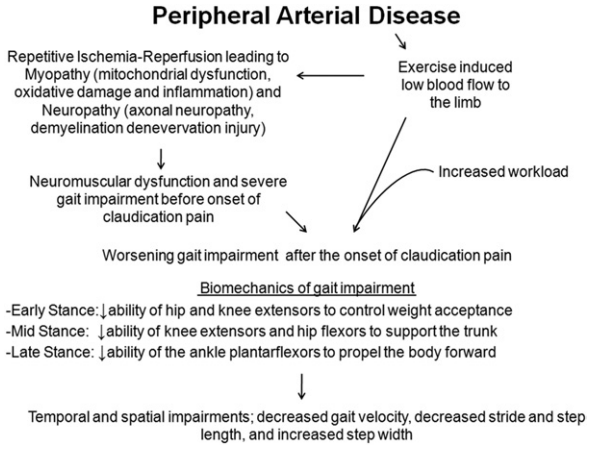

Fig 2. Proposed pathway for the pathogenesis of gait impairment in patients with peripheral arterial disease (PAD). The primary problem in claudicating patients is the presence of atherosclerotic blockages in the arteries supplying their legs. At rest, claudicating patients have adequate leg perfusion and experience no symptoms. During walking, however, the increased metabolic needs of the limb cannot be met, and as the exercise continues, the limb becomes progressively ischemic and painful, eventually forcing the patient to stop and rest. During rest, the metabolic demands of the limb return to baseline, and the leg is reperfused. Repeated cycles of ischemia-reperfusion, occurring with basic daily activities as simple as walking, initiate a combination of mitochondrial dysfunction, oxidative damage, and inflammation that eventually produces a myopathy and axonal polyneuropathy in the claudicating limbs. We propose that the gait impairments we have identified at baseline (in the first few steps taken and before the onset of muscle pain) reflect the effects of this myopathy and neuropathy in the function of the PAD limbs. Several of these biomechanic impairments get worse after onset of claudication symptoms when exercise-induced ischemia and increasing workload produce progressively worsening ischemic muscle pain and restriction of the lower extremity bioenergetics.

On a clinical level, only recently have studies with large sample sizes of 700 to 2000 participants been able to demonstrate the long-held assumption that claudicating patients have significantly reduced muscle strength.[32,40] By using advanced biomechanical analysis, our study has allowed us to confirm these large-scale studies with a limited number of patients and to implicate the involved muscle groups. Advanced biomechanical techniques thus provide a new avenue for evaluation, treatment, and rehabilitation of the PAD patient.

CONCLUSIONS

Biomechanical analysis using joint torques and powers demonstrates significant abnormalities in the gait of claudicating patients with bilateral PAD. These abnormalities are present at the onset of ambulation and worsen with the pain of claudication. Our work points to a failure of major muscle groups to optimally perform weight acceptance, transfer, and propulsion, the sequence of functions that characterize normal gait. In patients with occlusive disease affecting the proximal arterial tree, the muscle groups most affected by the chronic ischemia are the hip flexors and extensors, the knee extensors, and the ankle plantar flexors. These findings introduce new insights into the pathophysiology of the claudicating gait. In the future, these advanced biomechanical techniques will provide for detailed objective and quantitative evaluation of the gait deficit of the claudicating patient, allowing for evaluation of new treatment and rehabilitation strategies.

AUTHOR CONTRIBUTIONS

Conception and design: IP, PK
Analysis and interpretation: PK, GH, NS
Data collection: PK, SM
Writing the article: PK, IP, GH
Critical revision of the article: NS, IP, PK ML
Final approval of the article: IP, PK, NS
Statistical analysis: GH, PK
Obtained funding: PK, IP, NS, JJ
Overall responsibility: IP, JJ, NS

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