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Peripheral arterial disease affects ground reaction forces during walking

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Objective: Claudication is the most common manifestation of peripheral arterial disease (PAD), producing significant ambulatory compromise. The gait of claudicating patients has been evaluated using primarily temporal and spatial parameters. With the present study, we used advanced biomechanical measures to further delineate the ambulatory impairment of claudicating patients. We hypothesized that the claudicating legs of PAD patients have an altered kinetic gait pattern compared with normal legs from control subjects.
Methods: Ambulation kinetics (ground reaction forces) were evaluated in claudicating patients and compared with age-matched healthy controls. Forces were analyzed in the vertical, anterior–posterior, and medial–lateral directions. Time from heel touch-down to toe-off (stance time) and time spent in double-limb support were also evaluated.
Results: The study recruited 14 PAD patients (age, 58 ± 3.4 years; weight, 80.99 ± 15.64 kg) with femoropopliteal occlusive disease (ankle-brachial index [ABI], 0.56 ± 0.03) and five controls (age, 53 ± 3.4 years; weight, 87.38 ± 12.75 kg; ABI, ≥1.00). Vertical force curve evaluation demonstrated significant flattening in claudicating patients resulting in a lower and less fluctuant center of mass when ambulating. In the anterior–posterior direction, claudicating patients demonstrated significantly decreased propulsion forces. In the medial–lateral direction, they had significantly increased forces consistent with wider steps and an inability to swing their legs straight through. Claudicating patients demonstrated a greater stance time and time in double limb support compared with healthy controls. Most importantly, gait abnormalities were present before the onset of claudication, with gait worsening after the onset of claudication.
Conclusion: Claudicating patients demonstrate significant gait impairments that are present even before they experience any limb discomfort. These alterations may make them feel more stable and secure while attempting to minimize use of the affected limb. Advanced biomechanical analysis, using ambulation kinetics, permits objective and quantitative evaluation of the gait of claudicating patients. Such evaluation may point to new rehabilitation strategies and provide objective measurement of functional outcomes after medical and surgical therapy.

Introduction

Peripheral arterial disease (PAD) is the result of lower limb arterial atherosclerosis and affects more than 8.4 million people in the United States.[1,2] The most common presentation of PAD is intermittent claudication. Patients with claudication are unable to ambulate normally due to tightness, pain, and fatigue produced by decreased circulation to the exercising leg muscles. In healthy individuals, exercise induces a marked increase in skeletal muscle metabolism that results in a 20-fold to 40-fold increase in skeletal muscle blood flow and oxygen delivery.[3] In patients with claudication, a deficient

oxygen supply compared with the demand causes the exercising muscle to become progressively more ischemic and painful. Thus, the patient walks initially without pain, subsequently limps (claudication), and is eventually forced to stop walking.

The evaluation of gait in PAD patients has been largely limited to time and distance measurements such as stride, step length, stance time, speed, and cadence obtained by inspection, and timing of patients' walking performances.[4-7] These initial studies found PAD patients walk slower[5-8] and have a shortened step length[5,8] and a reduced cadence.[5,7,8] Furthermore, patients appear to have impaired balance, as indicated by a shorter unipedal stance time, a higher prevalence of ambulatory stumbling, and an increased prevalence of falling compared with healthy controls.[8,9]

A limited attempt at a more detailed analysis of claudicating limb gait was also attempted with the use of pressure- sensing insoles during treadmill walking. The work demonstrated progressively decreasing pressure values as claudicating patients walk.[10] The studies to date document significant ambulation abnormalities in claudicating patients, but give little insight into the exact biomechanical mechanisms producing the abnormal findings.

To fully understand the ambulation abnormalities in claudicating patients, our group has pursued advanced biomechanical evaluations of people with and without claudication. The biomechanical measures include an evaluation of lower extremity kinetics. This measures the forces exerted by the subject's weight-bearing limb on the ground, also known as ground reaction forces, a direct application of Newton's third law of motion concerning action-reaction.[11] The ambulating individual pushes the ground with a force, and the ground exerts an equal and opposite force. For clinical analysis, this force is resolved into three components orthogonal to each other (vertical, anterior–posterior, and medial–lateral) along a three-dimensional system (Fig 1). The magnitude and direction of these forces can be collected using a standard piezoelectric force plate.
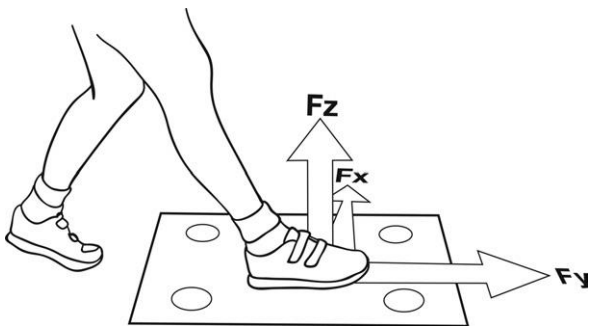

Fig 1. Lower extremity kinetic evaluation measures the forces exerted by the subject's weight-bearing limb on the ground, also known as ground reaction forces. The magnitude and direction of these forces can be collected using a standard piezoelectric force plate embedded in the ground. For clinical analysis, these forces are resolved into three components orthogonal to each other (vertical or Fz, anterior–posterior or Fy, and medial–lateral or Fx) along a three-dimensional system.

Although a common practice in many other clinical domains, no studies, to our knowledge, have examined claudicating patients with advanced biomechanical analysis.[12-14] Biomechanical analysis has quantified the efficacy of surgical procedures to improve gait in children with cerebral palsy, including guiding postoperative rehabilitation strategies.[15-17] Joint replacement operations have been evaluated extensively for biomechanical properties, longevity, and patient satisfaction.[18,19] In patients with anterior cruciate ligament deficiency, advanced biomechanical evaluations identified specific gait alterations currently being addressed through precise rehabilitation methodologies to restore normal gait.[20,21] Furthermore, rehabilitation strategies for patients with stroke and amputation have undergone thorough biomechanical analysis to identify objective gains in mobility.[22-24] In contrast to the progress made in these fields, Gardner and Montgomery9 correctly state that very little has been done to understand the underlying biomechanical gait abnormalities produced by PAD.[9]

This investigation used advanced kinetic biomechanical testing to evaluate and characterize the ambulatory pattern of PAD patients. We hypothesized that the claudicating legs of PAD patients have an altered kinetic gait pattern compared with normal legs from control subjects. This study provides initial data on the gait abnormalities of claudicating patients that can be used as the necessary foundation for the development of new rehabilitation strategies and the initiation of studies that may optimally quantify the effects of drugs and revascularization for patients with PAD.

METHODS

Subjects. Appropriate Institutional Review Board approval was obtained before the study was initiated, and all subjects provided informed consent. Patients with clinically diagnosed femoropopliteal PAD presenting with unilateral or bilateral calf claudication were recruited from our vascular surgery clinic. Patients were free of any confounding variables that could limit or alter their gait, including significant cardiac, pulmonary, neurologic, diabetic, or musculoskeletal disease. Specifically, subjects were excluded if they had recent myocardial infarction or unstable angina and ambulation-limiting heart failure, angina, or pulmonary disease. Subjects were also excluded if they had ambulation-limiting neurologic or musculoskeletal disease such as paresis, sciatica, diabetic neuropathy, or arthropathy.

Subjects were evaluated by history and physical examination, and femoropopliteal disease was verified by noninvasive testing and by computed tomography scanning, magnetic resonance imaging, or invasive angiography. These assessments were used to establish limbs with occlusive disease and typical symptoms as "claudicating limbs" and select them for biomechanical analysis.

Control subjects were recruited from the community, with a detailed history, physical examination, and noninvasive testing performed to establish absence of PAD, claudication, and confounding variables. Controls were selected to have similar age, body mass index (BMI), and physical activity level with the PAD subjects. Each leg of these individuals was used as "control limb." Height and weight were measured for all participants and BMI was calculated. To eliminate variability in gait due to shoes, all subjects wore the same standard laboratory shoes (Cross Trekkers, Payless Shoes, Topeka, Kan).

Lower extremity kinetics. The magnitude and direction of the ground reaction forces were collected using a standard piezoelectric force plate (Kistler Force Platform, Amherst, NY) with a sampling frequency of 600 Hz.[11] As already mentioned, ground reaction forces are those exerted by the subject's weight-bearing limb on the ground and are a direct application of Newton's third law of motion concerning action-reaction.[11] The ambulating individual pushes the ground with a force and the ground exerts an equal and opposite force. For clinical analysis, this force is resolved into three components orthogonal to each other along a three-dimensional system (Fig 2). The components are labeled: F_z, vertical (up–down) component; F_y, anterior–posterior (forward–backward) component; and F_x, medial-lateral (side-side) component.[11]

The patients walked on the force plate, with each leg undergoing five trials (10 trials per person). Because of claudication, ambulation was limited to three strides before and after striking the force plate, thus eliminating the possibility of pain in the affected limbs. All patients were required to rest in a chair for several minutes before and between trials to ensure "before onset of claudication" measurements were pain free. Kinetic data were collected on the force platform, with heel contact to toe-off representing one entire stance cycle. Determination of first leg to be collected was randomized.
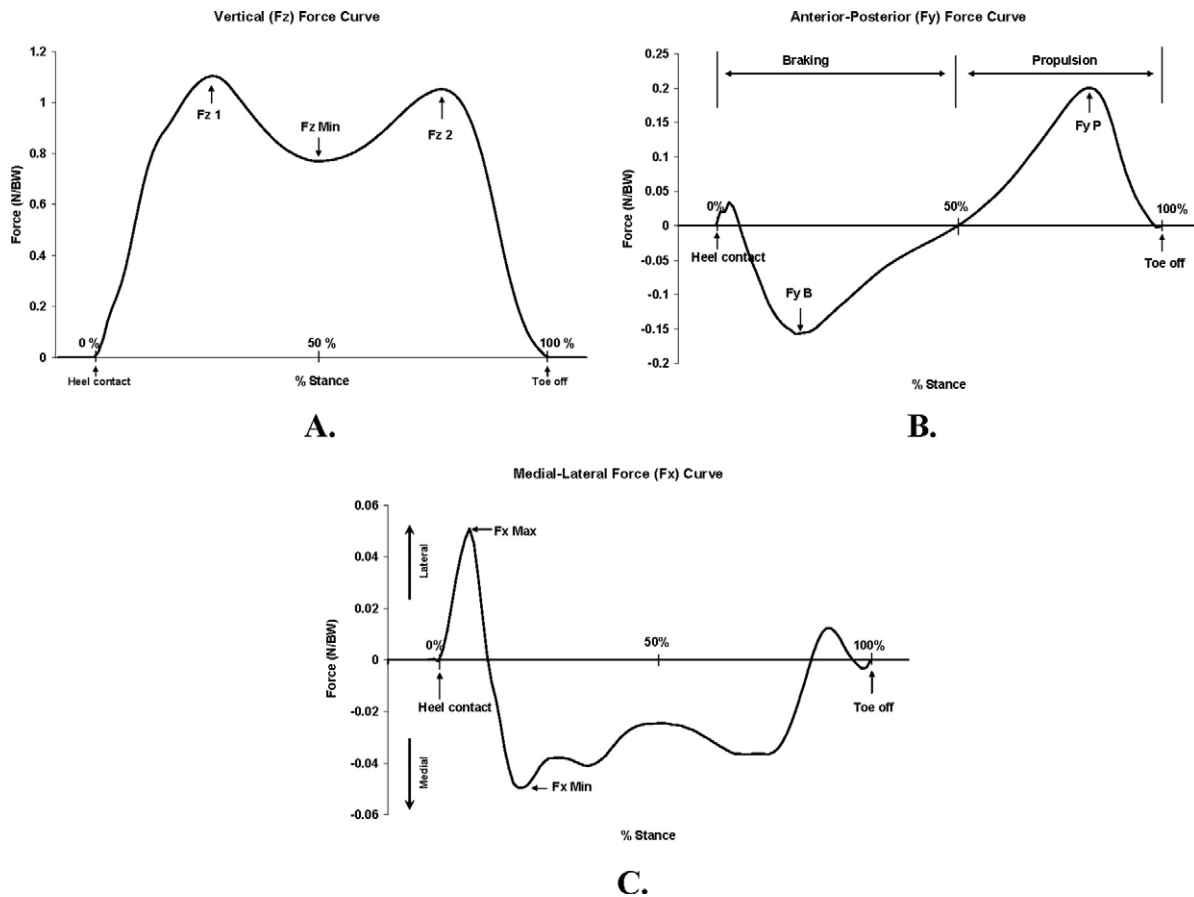
Fig 2. Typical force plate data from a control subject walking at a normal speed are shown plotted against time (percent of stance). Note that all forces are normalized over body weight and are expressed as multiples of body weight. A, The vertical force (F_z) is a representation of the vertical oscillation of the body's center of gravity and is expressed as percentage of body weight. It is very characteristic in that it shows a rapid rise after heel contact (center of mass accelerated upward) to reach a value (F_z1) in excess of body weight as full weight-bearing takes place. Then as the knee flexes during midstance, the center of mass is accelerated downward and the force drops below body weight, reaching a minimum at midstance (F_zmin). At push-off, the plantar flexors are active and again accelerate the body mass upwards, generating a second peak that is greater than body weight (F_z2). Finally, F_z drops to 0 as the contralateral limb takes up the body weight. B,The anterior–posterior force (F_y)has an initial negative component immediately after heel contact (braking force), indicating a backward horizontal friction between the ground (force plate) and the foot. The peak of the breaking component is named braking peak (F_yB). The area under the F_y anterior–posterior force curve between touch-down and zero-crossing at midstance is called "braking impulse." The most important F_y component is the positive one, reflecting the action of the leg muscles (mainly plantar flexors) causing the foot to push back against the ground, thus generating forward movement (propulsion force). The peak of the propulsion component is named propulsion peak (F_yP), and the area under the F_y anterior–posterior force curve between zero-crossing at midstance and toe-off is called "propulsion impulse." C, The medial–lateral force (F_x) has an initial, short positive (lateral force) component immediately after heel contact because of initial foot adduction. It then becomes negative (medial force component) as the foot abducts for toe-off. The peak of the lateral force component is named "lateral force maximum" (F_xmax) and the area under the F_x medial-lateral force curve between touch-down and zero-crossing is called "lateral F_x impulse." Similarly, the peak of the medial force component is named "medial force minimum" (F_xmin), whereas the area under the F_x medial-lateral force curve between zero-crossing and toe-off is called "medial F_x impulse."

Once PAD patients completed "before onset of claudication" pain-free trials, they ambulated until claudication pain was well established and "after onset of claudication" data were collected. To accomplish this, patients walked on a treadmill at a 10% grade (0.67 m/s) until claudication was first felt (usually patients become symptomatic after 1 to 3 minutes on the treadmill) and then for approximately 45 additional seconds. Patients were then returned to the force plate, where five trials were collected for each leg. Claudication pain was present in the PAD group throughout the second gait test. Healthy controls followed a similar protocol. Because they experience no claudication symptoms, no "after onset

of claudication" data were collected.

Data analysis. The force platform was interfaced with the Peak Motus system (Peak Performance Technologies, Englewood, Colo). Raw analog force data were then extracted, and the data files were further analyzed by using custom software created with Matlab (Mathworks Inc, Natick, Mass). The medial–lateral (F_x), anterior–posterior (F_y), and the vertical (F_z) forces were retained for further analysis. The variables identified from these forces and statistically considered are summarized in Table I and graphically depicted in Fig 2.

Table I. List of dependent variables examined and their description

Variable	Description
Vertical force (F_z)	
F_z1	First maximum for the F_z vertical force curve
F_z2	Second maximum for the F_z vertical force curve
$F_z$min	Local minimum between maximums for the F_z vertical force curve
$F_z1 - F_z$min	Difference between F1 and Fmin
$F_z2 - F_z$min	Difference between F2 and Fmin
Anterior–posterior force (F_y)	
F_yB	Minimum of the F_y anterior–posterior force curve (braking peak)
F_yP	Maximum of the F_y anterior–posterior force curve (propulsion peak)
Braking impulse	Area under the F_y anterior–posterior force curve between touch-down and zero-crossing at mid-stance
Propulsion impulse	Area under the F_y anterior–posterior force curve between zero-crossing at mid-stance and toe-off
Medial–lateral force (F_x)	
F_x max	Maximum of the F_x medial-lateral force curve (lateral force maximum)
F_x min	Minimum of the F_x medial-lateral force curve (medial force minimum)
Impulse F_x total	Area under the F_x medial-lateral force curve
Impulse F_x medial	Area under the F_x medial-lateral force curve between touch-down and zero-crossing (lateral F_x impulse)
Impulse F_x lateral	Area under the F_x medial-lateral force curve between zero-crossing and toe-off (medial F_x impulse)
Time parameters	
Stance	Time of stance
Time to F_z1	Percentage of stance time to F_z1
Time to F_z2	Percentage of stance time to F_z2
Time to $F_z$min	Percentage of stance time to $F_z$min
Time to F_yB	Percentage of stance time to F_yB
Time to F_yP	Percentage of stance time to F_yP
Time to $F_x$max	Percentage of stance time to $F_x$max
Time to $F_x$min	Percentage of stance time to $F_x$min

Times from heel touch-down to the minimums and maximums of the medial–lateral (F_x) and anterior–posterior (F_y) curves were calculated. Average curves were also developed to illustrate the average differences between groups. These average curves were calculated as follows:

- A cubic spline algorithm was used to normalize the data from each trial to 100 points for the stance period.[25]
- The curves for each trial were averaged point-by-point to generate an average curve for each subject for each condition.
- These resulting curves are plotted in Fig 3 to assist in the explanations of our results.
- The normalization procedure occurred after maximums and minimums and the other variables were determined to ensure that normalization did not distort these values.
- Impulse (calculated as area under the curve; Fig 2) parameters were normalized to stance time to allow comparisons between populations.[26]
- Stance time was determined based on the F_z force frequency and the number of points output from heel touch-down to toe-off.

Statistical analysis. Statistical analysis was performed using SigmaStat (Systat Software Inc, Point Richmond, Calif). Subject and group means were calculated for all dependent variables. Comparisons between claudication subjects and control subjects were conducted using unpaired t tests. In PAD

subjects, parameters before onset of claudication were compared with those after onset of claudication using paired *t* testing. Correlations between PAD severity and kinetic parameters were conducted using the Pearson correlation. Results were expressed as mean values ± standard error unless otherwise specified.

RESULTS

The study recruited 14 PAD patients (age, 58 ± 3.4 years; weight, 80.99 ± 15.64 kg; height, 172.12 ± 6.78 cm) with clinically diagnosed femoropopliteal occlusive disease (ankle-brachial index [ABI], 0.56 ± 0.03; range, 0.45 to 0.65). All patients had moderate claudication or category 2 symptoms in the Rutherford classification,[27] and 80% were hypertensive, 70% were smokers, 60% had dyslipidemia, and 30% were obese. All patients were treatment naïve and were leading a sedentary lifestyle. From these 14 patients, we identified 20 PAD legs that were included for evaluation in the present study. Five control subjects with absence of claudication (age, 53 ± 3.4 years; weight, 87.38 ± 12.75 kg; height, 178.78 ± 4.32 cm; ABI ≥ 1.00) were also included. Two had dyslipidemia and one had hypertension. BMI values were 28.5 ± 0.98 for PAD patients and 27.3 ± 1.5 for control subjects. All control subjects were leading a sedentary lifestyle. No significant differences were noted between the two groups for age and BMI.
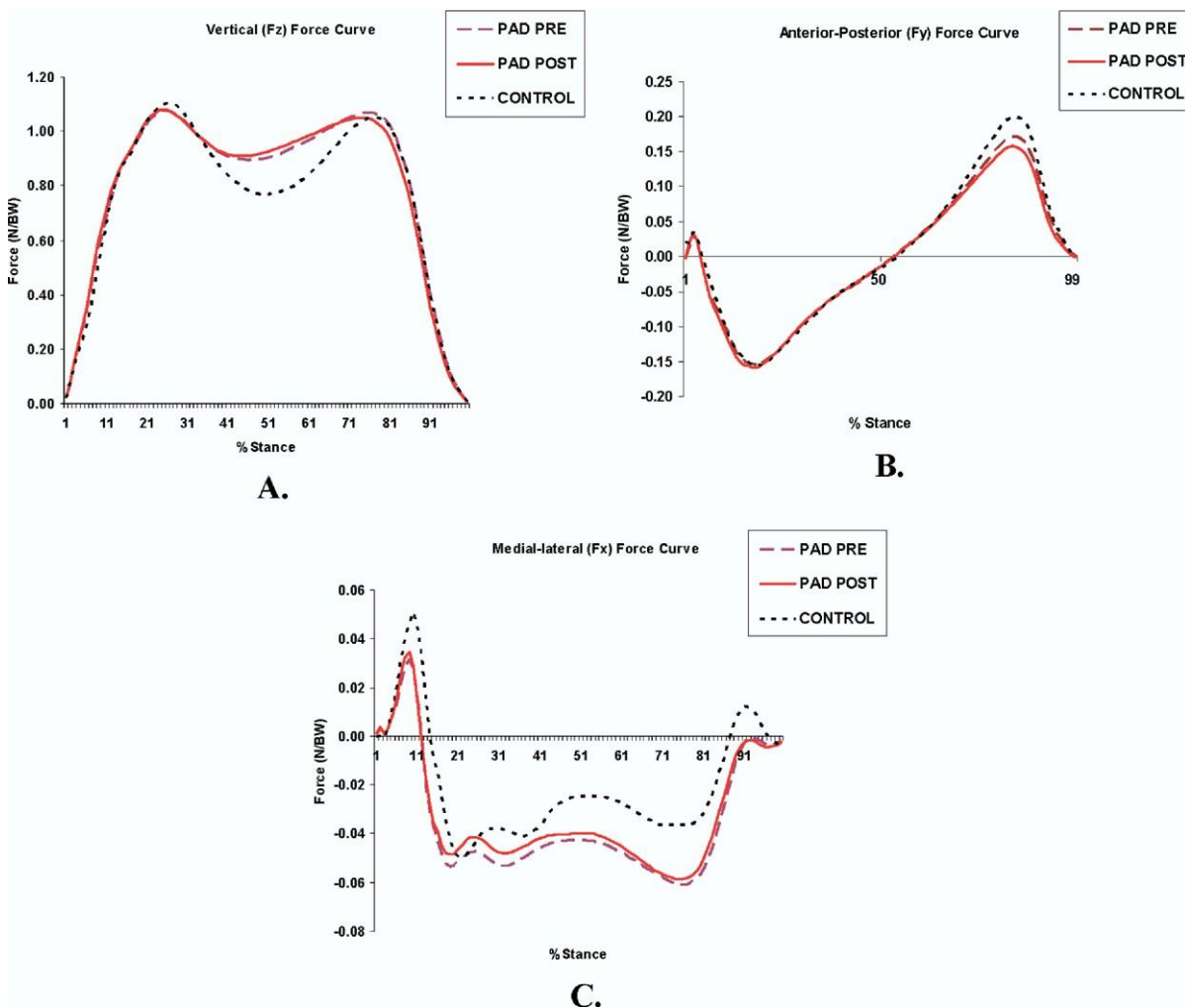

Fig 3. The mean curves for (A) the vertical forces, (B) anterior–posterior forces, and (C) medial–lateral forces. PAD PRE, Peripheral arterial disease patients before onset of claudication; PAD POST, peripheral arterial disease patients after the onset of claudication.

Gait is impaired before and after onset of claudication. Claudicating patients demonstrate significant gait alterations both before and after the onset of claudication (Fig 3, Table II). Evaluation of the vertical force demonstrates claudicating patients have a significantly higher midstance minimum ($F_z$min) and a significantly decreased difference between the second maximum and the midstance minimum ($F_z2 - F_z$min) compared with controls ($P < .05$). These results are reflected as a significant flattening of the vertical force curve for the claudicating patients (Fig 3). A more striking finding is the presence of these abnormalities in PAD limbs even before the onset of claudication pain.

Table II. Comparison of different gait parameters in controls and peripheral arterial disease patients both before and after onset of claudication*

	Control	PAD PRE	P[†]	PAD POST	P[‡]	P[$]
Vertical (F_z)						
F_z1	1.131 ± 0.070	1.123 ± 0.156	NS	1.131 ± 0.158	NS	NS
F_z2	1.058 ± 0.051	1.081 ± 0.128	NS	1.065 ± 0.127	NS	<.05
$F_z$min	0.752 ± 0.059	0.870 ± 0.121	<.05	0.879 ± 0.117	<.05	NS
$F_z1 - F_z$min	0.379 ± 0.126	0.252 ± 0.181	NS	0.251 ± 0.188	NS	NS
$F_z2 - F_z$min	0.307 ± 0.096	0.211 ± 0.129	<.05	0.186 ± 0.126	<.05	<.05
Anterior-posterior (F_y)						
F_yB	−0.168 ± 0.023	−0.172 ± 0.072	NS	−0.174 ± 0.075	NS	NS
F_yP	0.205 ± 0.024	0.170 ± 0.054	NS	0.157 ± 0.057	<.05	<.05
Braking impulse	0.044 ± 0.008	0.035 ± 0.011	NS	0.037 ± 0.013	NS	NS
Propulsion impulse	0.045 ± 0.005	0.032 ± 0.009	<.05	0.030 ± 0.009	<.05	<.05
Medial-lateral (F_x)						
F_x max	0.057 ± 0.024	0.038 ± 0.017	<.05	0.042 ± 0.005	<.05	NS
F_x min	−0.058 ± 0.011	−0.075 ± 0.016	<.05	−0.073 ± 0.005	<.05	NS
Impulse F_x total	0.029 ± 0.004	0.040 ± 0.004	<.05	0.0377 ± 0.003	<.05	NS
Impulse F_x medial	0.024 ± 0.004	0.038 ± 0.009	<.05	0.035 ± 0.003	<.05	NS
Impulse F_x lateral	0.0042 ± 0.002	0.0022 ± 0.001	<.05	0.0024 ± 0.0003	<.05	NS
Time parameters						
Stance	0.738 ± 0.065	0.822 ± 0.065	<.05	0.841 ± 0.0160	<.05	<.05
Time to F_z1	25.054 ± 2.496	27.156 ± 6.480	NS	26.487 ± 0.985	NS	NS
Time to F_z2	76.857 ± 1.228	74.094 ± 3.363	<.05	71.904 ± 1.005	<.05	<.05
Time to $F_z$min	50.199 ± 3.039	46.472 ± 5.792	NS	45.293 ± 0.956	<.05	NS
Time to F_yB	18.338 ± 1.228	16.885 ± 2.507	NS	16.930 ± 0.564	NS	NS
Time to F_yP	84.476 ± 0.651	84.118 ± 1.125	NS	83.239 ± 0.481	<.05	<.05
Time to $F_x$max	11.190 ± 6.030	11.300 ± 10.645	NS	8.771 ± 0.903	NS	NS
Time to $F_x$min	38.082 ± 21.880	39.853 ± 17.833	NS	44.704 ± 19.163	NS	<.05

PAD PRE, Patient before claudication onset; *PAD POST*, patient after claudication onset; *NS*, statistically not significant.
*For the description of each one of the gait parameters evaluated, please see Table I. Data are presented as means ± standard deviation.
[†]Control vs PAD PRE.
[‡]Control vs PAD POST.
[$]PAD PRE vs PAD POST.

Anterior–posterior force evaluation revealed claudicating patients have significantly lowered peak propulsion (F_yP). PAD patients demonstrate decreased propulsion force both before and after the onset of claudication ($P < .05$; Fig 3).

Medial–lateral force curve evaluation shows claudicating patients have significantly decreased forces in the lateral direction (early part of the stance), with increased forces in the medial direction (later part of stance, Fig 3). The difference in the medial forces becomes largest just before toe-off. These findings are also seen in a significantly increased area under the curve (ImpF_x) for the claudicating patients compared with controls ($P < .05$).

A significant increase in stance time, defined as time from heel contact to toe-off, ($P < .05$) was noted for claudicating patients before and after the onset of claudication compared with controls. Detailed time analysis of their curves demonstrates significant reduction in time to reach F_z2, $F_z$min, and F_yP. These changes in claudicating patients reflect a decrease in time spent in single-limb support, with faster transition into double-limb support. Again, most of these adaptations are displayed both before and after the onset of claudication.

Gait parameters worsen after the onset of claudication. After the onset of claudication, PAD patients demonstrated significant worsening in multiple kinetic parameters. Evaluation of the vertical

force curve demonstrated further flattening of the force curve, especially in the second half of stance, reflected in a significantly decreased difference between the second maximum and the midstance minimum (F_z2 - F_zmin). The anterior– posterior curve evaluation showed that after onset of claudication, PAD patients had a further significant drop in their propulsion peak (F_yP). In addition, evaluation of the time parameters demonstrated claudication results in prolongation of stance time, with corresponding short- ening of the time to Fz2 and FyP. Because double-limb support starts just before Fz2 and FyP, these findings taken together indicate that the onset of claudication is associated with prolongation of time spent in double- limb support. Of note, the control subject's gait was also evaluated after a 10-minute treadmill session (10% grade, 0.67 m/s) to determine possible effects of fatigue on biomechanical parameters. The assessment demonstrated that controls have similar gate characteristics before and after exercise (data not included).

DISCUSSION

Evaluation of claudicating patients by using advanced biomechanical measures (ground reaction forces) provides a new appreciation of the complexity of their ambulatory impairment. Ground reaction forces can be resolved into three components that are orthogonal to each other (vertical, anterior– posterior, and medial–lateral).[11] The vertical ground reaction force curve is significantly flatter in claudicating patients, both before and after onset of claudication, compared with controls. The flatter configuration in claudicating patients is the result of the absence of the normally deep midstance "valley" (Fig 2 and Fig 3; reflected in a higher F_zmin).

Because the vertical force curve mirrors the rise and fall of the subject's center of mass, this alteration indicates that claudicating patients have significantly less vertical movement of their center of mass compared with controls. Decreased vertical fluctuation, especially in the second half of stance, can be explained by an increase in time spent in double-limb support and an inability to fully extend the limb at single-limb support. Overall, the changes seen in the vertical forces indicate PAD patients adopt a walking pattern that limits the natural up-and-down body motion seen with normal ambulation. This may be the result of dysfunction at the muscle level, decreased fine neural motor control, or muscle pain inhibiting the ability to generate force.

In the anterior–posterior direction, the second half of stance is the indicator of impaired gait by the PAD patient. This is the point of propulsion of the foot into the swing phase. Even before the onset of claudication, PAD patients demonstrate a tendency towards a smaller propulsion peak, which becomes significantly smaller at the onset of claudication. Similarly, claudicating patients demonstrate decreased propulsion impulse both before and after the onset of claudication. These findings indicate that claudicating limbs have decreased ability to push off. Such a reduction in forward impulse indicates a lack of strength and dysfunction in the propulsive muscles of the lower extremity or can potentially be the result of purposeful action (ie, neural control) to reduce the use of the painful limb.

Our force data for the medial–lateral direction, both in the before-and-after onset of claudication conditions, demonstrated PAD patients apply significantly higher forces in the medial direction. This greater medial–lateral force fluctuation by the claudicating subject is probably the result of increased step width with reduction in single-limb support time (increased time spent in double-limb support). A larger step width creates increased forces in the medial–lateral direction to maintain the wider stance.[28] This possibility is further supported by our vertical curve data, where the PAD patients maintain a lower center of gravity throughout stance. As a result, during ambulation the limbs need to be swung laterally to clear the ground through to the next step. In adopting wider steps, greater medial forces are then created.

In the case of reduction in time spent in single support, PAD patients are pushing medially (generating the greater medial forces found) to help shorten the swing phase of the contralateral leg. As a result, the contralateral leg regains contact with the ground more quickly and provides increased double-limb support for the patient. Such adaptations may be secondary to the PAD patient who seeks security in his or her impaired gait. Adopting a wider step and maintaining double-leg support for most of the gait cycle, with less vertical fluctuations of the center of mass, may contribute to a feeling of safety

during locomotion.

Previous work has determined that elderly patients and other pathologic populations such as those with Parkinson disease or peripheral neuropathy walk slower.[14,29] It has been speculated that this change may be due to a need to increase stability.[30] Works from other laboratories as well as our present data (longer stance time, increased double support time) demonstrate that like the elderly, PAD patients adopt a slower walking pattern.[4-7] Investigations of elderly patients have reported stability and security gains from walking with increased double support time and slower velocity.[30,31] This may be the underlying etiology for the current findings in the PAD gait.

Walking slower, however, has been found to increase the metabolic cost of walking[32] and may not offer more stability.[33] For a PAD patient, metabolic efficiency is vital to functional mobility. A metabolically draining task will decrease the time a patient can walk because the occluded arteries cannot supply the needed energy to maintain the task and pain will likely occur sooner. In addition, Gardner and Montgomery[8] described a higher incidence of self-reported falling in PAD patients. If walking slower offers stability for the patient, theoretically, they should not be falling more often. Future investigations should further evaluate the apparent clinical significance of these findings. In the future, gait analysis may point to new rehabilitation strategies that could correct the abnormal gait patterns, allowing for greater exercise tolerance and improved balance in PAD patients.

When claudication pain is present, the vertical and anterior–posterior force changes became more apparent. The vertical force curve became even flatter, indicating further reduction of the natural vertical fluctuation seen with normal ambulation, and propulsion drops further, suggesting that the function of muscles supporting propulsion worsens. Analysis of the time parameters shows prolongation of stance time with less time spent in single-limb support (shortening of the time to F_z2 and F_yP). These findings indicate that the patient experiencing claudication pain probably does not want to stand on the painful, potentially weak and unsteady-feeling leg without alternative support.

As indicated in the results, a definite change occurs in the gait of the PAD patient during the second half of the stance phase. Further biomechanical analysis using kinematic data that evaluates the angles of the hip, knee, and ankle joints may offer more insight into the specific propulsion compromise experienced by the ambulating claudicating patient. As noted in our results, no change in gait over time was noted when the control subject's gait was evaluated after a 10-minute treadmill session. Therefore, it is highly unlikely that the degradation of kinetic parameters seen after the onset of claudication is the result of normal exercise-related fatigue.

An unexpected finding in these series was that PAD patients had evidence of significant ambulatory impairments even when not experiencing any claudication pain. In other words, the lower extremity neuromuscular system of PAD patients appears to be dysfunctional at baseline, before increased activity induces any appreciable ischemia and pain. This finding is consistent with previous reports that demonstrate a muscle metabolic myopathy[34] and an axonal polyneuropathy[35] in the lower extremities of PAD patients. Specifically, a number of reports have documented a metabolic myopathy in PAD muscle that appears to be secondary to defective mitochondrial bioenergetics. Mitochondria in PAD muscle have abnormal ultrastructure,[36] damaged DNA,[37] altered enzyme expression and activity, and abnormally high intermediates of oxidative metabolism.[3] Most importantly, evaluation of the mitochondrial bioenergetics of claudicating muscle demonstrates specific defects in the complexes of electron transport chain, with associated compromised mitochondrial respiration (oxidative phosphorylation) and adenosine triphosphate production[38-41] that is very similar to those seen in mitochondrial myopathies.[34,38]

In addition, accumulating evidence suggests that chronic ischemia in PAD patients results in a consistent pattern of electrodiagnostic abnormalities indicating axonal nerve loss.[35,42,43] Therefore, the impairments we have identified at baseline may be reflecting a combination of myopathy and neuropathy in the PAD limbs that gets worse when exercise-induced ischemia produces pain and further restriction of the lower extremity bioenergetics. Alternatively, repeated episodes of exercise-induced ischemic pain may lead to the establishment of a maladaptative gait in claudicating patients and such adaptations may persist even in the absence of pain.

CONCLUSION

Future research should build on the findings of this investigation. Our current work describes the gait kinetic alterations that occur in claudicating patients with femoropopliteal disease. Evaluation of patients with aortoiliac occlusive disease or combined femoropopliteal and aortoiliac occlusive disease should be the subject of similar future investigation. In addition, more detailed biomechanical analysis may include simultaneous evaluation of kinetics (ground reaction forces) and kinematics (joint angles) in the form of joint moments and powers. Such evaluation will improve our understanding of the action of the muscles across the joints of interest during gait, indicating specific muscle group failure that can be correlated with the individual patient's level of occlusive disease and type of ambulatory handicap. Measuring the muscular action across a joint in the PAD patient's current gait style, then rehabilitating gait into becoming more like the healthy control, and then evaluating again could offer important understanding of the adaptations by the PAD patient and, furthermore, may determine the altered gait to be maladaptive.

The functional handicap of claudicating patients cannot be fully understood without detailed studies of their movement. The goal of this investigation was to characterize the gait of claudicating PAD patients by using detailed kinetic biomechanical analysis. The results indicate clear abnormalities in the gait of claudicating patients. These abnormalities are present both before and after the onset of claudication, with several of them becoming worse after claudication onset. Our data indicate biomechanical analysis can objectively quantify the ambulatory handicap of PAD patients. Such analysis may facilitate the identification of optimal rehabilitative regimens that could correct the abnormal gait patterns, allowing for greater exercise tolerance. In addition, biomechanical evaluation may provide a firm foundation for optimal clinical decision-making and, more importantly, assist in an objective measurement of functional outcomes after medical and surgical therapy.

AUTHOR CONTRIBUTIONS

Conception and design: IP, NS, MS, JJ
Analysis and interpretation: MS, NS, JJ, LR, TL, IP Data collection: MS, LR, IP
Writing the article: MS, IP, NS
Critical revision of the article: JJ, LR, TL
Final approval of the article: MS, NS, JJ, LR, TL, IP Statistical analysis: MS, IP
Obtained funding: MS, NS Overall responsibility: IP, NS

REFERENCES

1. Cronenwett JL, Warner KG, Zelenock GB, Whitehouse WM Jr, Graham LM, Lindenauer M, et al. Intermittent claudication. Current results of nonoperative management. Arch Surg 1984;119:430-6.

2. Rosenbloom MS, Flanigan DP, Schuler JJ, Meyer JP, Durham JR, Eldrup-Jorgensen J, et al. Risk factors affecting the natural history of intermittent claudication. Arch Surg 1988;123:867-70.

3. Lundgren F, Bennegard K, Elander A, Lundholm K, Schersten T, Bylund-Fellenius AC. Substrate exchange in human limb muscle during exercise at reduced blood flow. Am J Physiol 1988;255:H1156-64.

4. Gardner AW, Forrester L, Smith GV. Altered gait profile in subjects with peripheral arterial disease. Vasc Med 2001;6:31-4.

5. McDermott MM, Ohlmiller SM, Liu K, Guralnik JM, Martin GJ, Pearce WH, et al. Gait alterations associated with walking impairment in people with peripheral arterial disease with and without intermittent claudication. J Am Geriatr Soc 2001;49:747-54.

6. Newman AB. Commentary on "the effects of peripheral vascular disease on gait." J Gerontol A Biol Sci Med Sci 1999;54:B295-6.

7. Scherer SA, Bainbridge JS, Hiatt WR, Regensteiner JG. Gait characteristics of patients with claudication. Arch Phys Med Rehabil 1998;79: 529-31.

8. Gardner AW, Montgomery PS. Impaired balance and higher prevalence of falls in subjects with intermittent claudication. J Gerontol A Biol Sci Med Sci 2001;56:M454-8.

9. Gardner AW, Montgomery PS. The relationship between history of falling and physical function in subjects with peripheral arterial disease. Vasc Med 2001;6:223-7.

10. Ayzin Rosoky RM, Wolosker N, Muraco-Netto B, Puech-Leao P. Ground reaction force pattern in limbs with intermittent claudication. Eur J Vasc Endovasc Surg 2000;20:254-9.

11. Winter DA, editor. Biomechanics and motor control of human movement. 3rd ed. Hoboken, NJ: Wiley; 2004. pp 86-116.

12. Chambers HG, Sutherland DH. A practical guide to gait analysis. J Am Acad Orthop Surg 2002;10:222-31.

13. Kaufman KR, Shaughnessy WJ, Noseworthy JH. Use of motion analysis for quantifying movement disorders. Adv Neurol 2001;87:71-81.

14. Alexander NB. Using technology-based techniques to assess postural control and gait in older adults. Clin Geriatr Med 1996;12:725-44.

15. Kay RM, Dennis S, Rethlefsen S, Reynolds RA, Skaggs DL, Tolo VT. The effect of preoperative gait analysis on orthopaedic decision making. Clin Orthop Relat Res 2000:217–22.

16. Lee EH, Goh JC, Bose K. Value of gait analysis in the assessment of surgery in cerebral palsy. Arch Phys Med Rehabil 1992;73:642-6.

17. Ounpuu S, Gage JR, Davis RB. Three-dimensional lower extremity joint kinetics in normal pediatric gait. J Pediatr Orthop 1991;11:341-9.

18. Borden LS, Perry JE, Davis BL, Owings TM, Grabiner MD. A biomechanical evaluation of one-stage vs two-stage bilateral knee arthroplasty patients. Gait Posture 1999;9:24-30.

19. Otsuki T, Nawata K, Okuno M. Quantitative evaluation of gait pattern in patients with osteoarthrosis of the knee before and after total knee arthroplasty. Gait analysis using a pressure measuring system. J Orthop Sci 1999;4:99-105.

20. Andriacchi TP. Functional analysis of pre and post-knee surgery: total knee arthroplasty and ACL reconstruction. J Biomech Eng 1993;115: 575-81.

21. Andriacchi TP, Mundermann A, Smith RL, Alexander EJ, Dyrby CO, Koo S. A framework for the in vivo pathomechanics of osteoarthritis at the knee. Ann Biomed Eng 2004;32:447-57.

22. Kerrigan DC, Todd MK, Della Croce U, Lipsitz LA, Collins JJ. Biomechanical gait alterations independent of speed in the healthy elderly: evidence for specific limiting impairments. Arch Phys Med Rehabil 1998;79:317-22.

23. Olney SJ, Griffin MP, Monga TN, McBride ID. Work and power in gait of stroke patients. Arch Phys Med Rehabil 1991;72:309-14.

24. Skinner HB, Effeney DJ. Gait analysis in amputees. Am J Phys Med 1985;64:82-9.

25. Giakas G, Baltzopoulos V. Acomparison of automatic filtering techniques applied to biomechanical walking data. J Biomech 1997;30:847-50.

26. Miller DI. Biomechanics of distance running. Champaign, Ill: Human Kinetics; 1990.

27. Rutherford RB, Becker GJ. Standards for evaluating and reporting the results of surgical and percutaneous therapy for peripheral arterial disease. J Vasc Interv Radiol 1991;2:169-74.

28. Donelan JM, Kram R, Kuo AD. Mechanical and metabolic determinants of the preferred step width in human walking. Proc Biol Sci 2001;268:1985-92.

29. McGibbon CA. Toward a better understanding of gait changes with age and disablement: neuromuscular adaptation. Exerc Sport Sci Rev 2003; 31:102-8.

30. Winter DA, Patla AE, Frank JS, Walt SE. Biomechanical walking pattern changes in the fit and healthy elderly. Phys Ther 1990;70:340-7.

31. Maki BE. Gait changes in older adults: predictors of falls or indicators of fear. J Am Geriatr Soc 1997;45:313-20.

32. Malatesta D, Simar D, Dauvilliers Y, Candau R, Borrani F, Prefaut C, et al. Energy cost of walking and gait instability in healthy 65- and 80-yr-olds. J Appl Physiol 2003;95:2248-56.

33. Buzzi UH, Stergiou N, Kurz MJ, Hageman PA, Heidel J. Nonlinear dynamics indicates aging affects variability during gait. Clin Biomech (Bristol, Avon) 2003;18:435-43.

34. Brass EP, Hiatt WR. Acquired skeletal muscle metabolic myopathy in atherosclerotic peripheral arterial disease. Vasc Med 2000;5:55-9.

35. Weber F, Ziegler A. Axonal neuropathy in chronic peripheral arterial occlusive disease. Muscle Nerve 2002;26:471-6.

36. Marbini A, Gemignani F, Scoditti U, Rustichelli P, Bragaglia MM, Govoni E. Abnormal muscle mitochondria in ischemic claudication. Acta Neurol Belg 1986;86:304-10.

37. Bhat HK, Hiatt WR, Hoppel CL, Brass EP. Skeletal muscle mitochondrial DNA injury in patients with unilateral peripheral arterial disease. Circulation 1999;99:807-12.

38. Kemp GJ. Mitochondrial dysfunction in chronic ischemia and peripheral vascular disease.

Mitochondrion 2004;4:629-40.

39. Pipinos I, Shepard AD, Anagnostopoulos PV, Katsamouris A, Boska MD. Phosphorus 31 nuclear magnetic resonance spectroscopy suggests a mitochondrial defect in claudicating skeletal muscle. J Vasc Surg 2000;31:944-52.

40. Pipinos II, Judge AR, Zhu Z, Selsby JT, Swanson SA, Johanning JM, Baxter BT, Lynch TG, Dodd SL. Mitochondrial defects and oxidative damage in patients with peripheral arterial disease. Free Radic Biol Med 2006;41:262-9.

41. Pipinos II, Sharov VG, Shepard AD, Anagnostopoulos PV, Katsamouris A, Todor A, Filis KA, Sabbah HN. Abnormal mitochondrial respiration in skeletal muscle in patients with peripheral arterial disease. J Vasc Surg 2003;38:827-32.

42. Koopman JP, de Vries AC, de Weerd AW. Neuromuscular disorders in patients with intermittent claudication. Eur J Surg 1996;162:443-6.

43. Laghi Pasini F, Pastorelli M, Beermann U, de Candia S, Gallo S, Blardi P, Di Perri T. Peripheral neuropathy associated with ischemic vascular disease of the lower limbs. Angiology 1996;47:569-77.