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Background: **Patients with intermittent claudication (IC) tend to walk at a slower pace, have less lower leg muscle strength, and consume approximately 40% more oxygen during walking compared with healthy individuals. An unfavorable locomotion pattern has been suggested to explain this metabolic inefficiency. However, knowledge on gait patterns in IC is limited. Muscle activity patterns during walking measured using surface electromyography (EMG) have not been investigated in this patient population.**

Methods: **In this cross-sectional study, gait pattern of patients newly diagnosed with IC and age-matched controls were evaluated using kinematic parameters and medial gastrocnemius (MG) and tibialis anterior (TA) muscles activity patterns. The protocol included pain-free and painful (only IC patients) treadmill walking sessions.**

Results: **A total of 22 IC patients and 22 healthy control subjects were included. Patients walked 1.4 km/h slower (3.2 km/h vs 4.6 km/h; $P < .001$) than control subjects, coinciding with a 10% slower cadence (110 steps/min vs 122 steps/min; $P < .001$). The kinematic analysis resulted in a patient's ankle plantar flexion reduction of 45% during the propulsion phase, and ankle dorsal flexion reduction of 41% at initial contact. No additional kinematic changes were observed when claudication pain presented. Interestingly, kinematic differences did not influence the muscle activity duration during walking, because equal duration of muscle activity was found in IC patients and healthy controls. However, the amount of muscle activity in microvolts did significantly increase in IC patients when claudication pain presented (TA: Δ23%; $P < .001$; MG: Δ54%; $P = .007$).**

Conclusions: **Patients with IC show significant kinematic changes during walking. These alterations did not affect EMG activity duration of MG and TA muscles. However, EMG amplitude of both muscles did significantly increase during painful walking in IC patients. (J Vasc Surg 2016;63:664-72.)**

Peripheral arterial disease is a chronic disease of the lower extremities caused by systemic atherosclerosis, affecting more than 12% of the population older than 65 years of age.[1] Intermittent claudication (IC) is the most common manifestation of peripheral arterial disease and is characterized by limb pain during exercise that resolves after a short period of rest. Patients with IC showed reduced walking performance[2] and revealed a greater prevalence of falls[3] compared with non-IC patients. Studies have demonstrated several mechanisms for these impairments[4,5]; patients were found to have less peak propulsion force, and a reduced ability to swing their legs forward.[6]

Other studies have suggested that the energy cost of locomotion is almost 40% greater in IC.[7] Various mechanisms might be responsible for this higher cost of locomotion. For instance, a maladapted and unfavorable gait pattern on the basis of IC induced neuromuscular dysfunction. Patients with IC might use the same muscles, but with a different sequence of fiber recruitment,[7] as a result of chronic fiber changes after exercise-induced ischemia.[8] Previous studies already found that lower extremity ischemia has a direct, negative effect on calf muscle diameter, density and muscle fiber types.[8,9]

Surface electromyography (EMG) is a noninvasive technique used to measure muscle action potentials that occur by muscle contraction during performance of motor tasks, such as walking.[10] The electrical activity is recorded by surface electrodes that are placed on the skin overlying a muscle or group of muscles.[11] It is a widely used method to determine activation patterns of different muscles (ie, timing, 'on-off pattern') and it allows study of normal and pathological motor strategies.[12] Surface EMG has been shown to be a feasible and valid method to record muscle activity.[13] A recent study in patients after total hip arthoplasty also included EMG as an adjunctive tool that complements gait analysis.[14] To our knowledge, surface

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EMG analyses during walking was not previously performed in patients with IC. This method might yield valuable information in IC patients as a means to investigate the previously suggested neuromuscular dysfunction. Chronic ischemia, as present in IC, might lead to alterations in motor unit recruitment (EMG amplitudes) and/or muscle coordination patterns. EMG evaluation examined together with kinematics (joint angles) provides a thorough insight into muscle performance and their role in accomplishing motor tasks.[10]

The primary aim of the current study was to determine lower extremity joint angles and EMG patterns in patients with IC and in healthy control subjects during continuous treadmill walking. Besides, the effect of claudication pain on these gait parameters was studied. We hypothesized that patients with IC would show less range of ankle and knee motion and generate a different pattern of muscle activity compared with healthy control subjects.

METHODS

Participants. Patients were recruited from a population who presented between October 2014 and March 2015 with new-onset manifestations of IC at the Vascular Surgery outpatient departments of three regional Dutch hospitals (Catharina hospital Eindhoven, Maxima Medical Center Veldhoven, and St. Anna Hospital Geldrop). According to standardized institutional protocols, potential patients underwent an ankle-brachial index (ABI) measurement during rest and after exercise. Individuals who showed a resting ABI <0.90 and/or an ABI decrease >0.15 after treadmill exercise[15] and typical symptoms of claudication in at least one leg, were eligible for this study. Additional inclusion criteria were presence of symptoms for more than 4 weeks and the ability to walk on a treadmill for $\geq$1 minute. Patients were excluded if they suffered from a comorbid condition that might possibly influence gait pattern, such as lower extremity amputation, severe osteoarthritis, knee or hip prosthesis, severe cardiopulmonary problems (ie, chronic obstructive pulmonary disease Gold classification $\geq$ III and/or New York Heart Association classification $\geq$ III), neurological diseases such as peripheral polyneuropathy (according to history and eventual physical examination), Parkinson disease, or stroke, previous lower extremities surgery (eg, bypass, percutaneous vascular intervention), use of walking aids, orthopedic shoes, or medication that might influence walking pattern (eg, psychotropic medication). Patients who experienced rest pain, suffered from chronic wounds, or had received more than five sessions of supervised exercise therapy (SET) during the preceding 3 years were also excluded.

Age-matched control subjects were recruited from family, friends, and hospital personnel. They exhibited an ABI at rest of >0.90 and did not experience any pain of the limbs or limitations during walking. Other exclusion criteria were similar as for the group of patients. Participants who were willing to participate were counseled before providing written informed consent. All procedures were approved by the Medical Ethical Committee of the Catharina Hospital Eindhoven, The Netherlands.

Study protocol. Two researchers (L.G. and A.S.) were responsible for data collection. Height and weight measurements allowed calculation of the body mass index. Leg length was determined with a tape measure with the subject in standing position and defined as the distance from the major trochanter of the hip to the inferior aspect of the lateral malleolus.

After anthropometric measurements, participants walked for 10 minutes (for IC patients, with intervals of rest if necessary) on a treadmill system to get familiarized with the system[16] and to determine a 'preferred' walking pace, defined as 'walk at a pace that is comfortable and comparable with your outside walking pace.'[16] The treadmill was started at 2.4 km/h and speed was increased in 0.1 km/h increments until this comfortable walking speed was achieved. Speed was then further increased until a rapid pace was attained, and decreased until the comfortable speed was reached once again. Gait measurements were obtained at this final, self-selected comfortable walking pace.

After a short period of rest, participants received reflective markers that were placed at specific anatomical locations of each participants' lower limb (Fig 1). The two-dimensional marker trajectories were captured with a high-speed real-time camera (Basler, 1394a/b, GigE) and its software system (Simi Aktysis version 1.4.1.2; Simi Reality Motion Systems GmbH, Unterschleissheim, Germany) sampling at 100 samples per second (hertz, Hz). Before placement of surface EMG probes, skin sites were cleaned with 70% isopropyl alcohol and hair was removed if necessary. The wireless EMG probes (BTS FreeEMG; BTS Bioengineering Corp, Brooklyn, NY) were attached to the skin via pregelled disposable Ag-electrodes (Kendall ECG electrodes H124SG) and positioned over the medial gastrocnemius (MG) muscle and tibialis anterior (TA) muscle following the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles recommendations.[11] MG and TA were assessed because these muscles are considered main actuators of the ankle.[17] Besides, the MG muscle is the most common site of ischemic pain that limits exercise tolerance in IC.[18] Recording muscle electrical activity using surface EMG during gait has been found to show a sufficient repeatability, reliability, and consistency.[19] Data were captured at 1000 Hz and synchronized with the kinematic data using a trigger box. Data were real-time stored and converted into digital signals by the EMG-Analyzer software program (BTS Bioengineering Corp).

After marker and electrode application, subjects had 20 minutes of rest. During 1 minute of painless walking, gait parameters were obtained. When IC patients experienced claudication pain determined using a 4-point claudication pain scale, gait parameters were again captured for 1 minute. Control subjects underwent similar measurements just once (ie, pain-free trial). All participants walked in bare feet on a flat treadmill.

Kinematic data analysis. The reflective markers provided a proper two-dimensional lower extremity segment

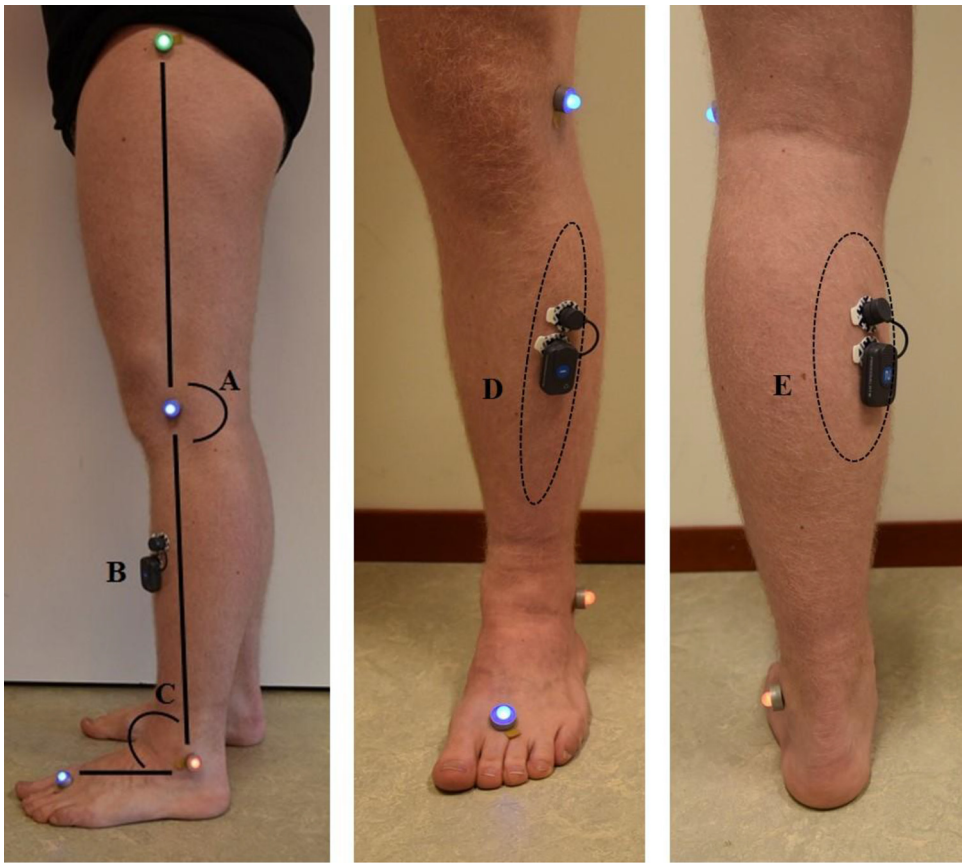

Fig 1. Measurement set up. **Left,** Light emitting diode (LED) lights are positioned on the bony landmarks of the lower limb, to determine the knee and ankle joint angles, respectively. *A* indicates knee joint angle; *B* indicates the electromyography (EMG) device on the tibialis anterior (TA) muscle; and *C* indicates the ankle joint angle. The current positions are set at zero degrees. **Middle,** *D* indicates the position of the EMG device on the TA muscle; the *dashed circle* indicates the muscle belly. **Right,** *E* indicates the position of the EMG device on the medial gastrocnemius (MG) muscle; the *dashed circle* indicates the muscle belly.

orientation and allowed for knee and ankle joint angles during walking. The maximal forward trajectory of the lateral malleolus reflective marker in the sagittal plane was used as an indication of heel strike. All kinematic parameters were normalized to 100% of the gait cycle (eg, 100 points represented heel strike to the following heel strike) using a cubic spline interpolation technique. Forty representative cycles were selected for each participant to determine the following (averaged) parameters: range of motion (ROM), maximum and minimum joint flexion and extension angles, and joint angles at the moment of heel strike. All signal processing and calculations were performed using a customized program that was created in Matlab R2012a (Mathworks Inc, Natick, Mass).

EMG data analysis. After digitalization of the raw EMG signals, further processing and analysis were performed in a customized Matlab program. The EMG signal was high-pass filtered (cutoff frequency of 20 Hz) to suppress potential movement artifacts, full-wave rectified by subtracting the mean offset value from the absolute signal values, and then low-passed filtered (cutoff frequency of 6 Hz) to create linear envelope EMG signals. A power spectrum analysis confirmed that chosen cutoff settings were appropriate. EMG data were normalized to a 100% gait cycle using the kinematic data of the lateral malleolus marker. Similarly, 40 gait cycles were used to calculate the ensemble average percentage of muscle activity and peak performance (in microvolts) for the MG and TA muscles per gait cycle. The threshold for muscle onset was on the basis of a 25% of the mean peak value of the EMG signal during walking.[20]

Statistical analysis. Differences in kinematic and EMG parameters of the index leg between IC patients and control subjects were studied. In IC patients, the most symptomatic leg was considered the index leg. In control subjects, the index leg was randomly selected. Evaluation within the patient group was performed by comparing the values of the pain-free and painful trials, respectively. A Pearson χ^2 test was used for analysis of differences between categorical variables, whereas an independent samples *t* test and paired *t*-test analyzed continuous variables. Variables that lacked normal distribution were expressed as median and interquartile

Table I. Demographic characteristics of study groups that underwent gait analysis

Characteristic	*IC patients (n = 22)*	*Control subjects (n = 22)*	P
Male sex	17 (77)	16 (73)	.73
Mean age (SD), years	69 (6.6)	69 (6.6)	.80
Mean height (SD), m	1.70 (0.08)	1.70 (0.08)	.84
Mean weight (SD), kg	80 (12)	76 (12)	.33
Mean leg length (SD), cm	81 (4.3)	81 (5.8)	1.00
Index leg: left[a]	11 (50)	10 (46)	.76
Mean ABI: Index leg (SD)			
Rest	0.64 (0.2)	1.1 (0.2)	<.001
After treadmill walking	0.32 (0.13)	-	
Mean duration of symptoms (SD), months	19 (17)	-	
Unilateral complaints[b]	9 (41)	-	
Mean BMI (SD)	27.3 (3.4)	26.3 (3.5)	.30
Smoking			
Current smoker	12 (55)	2 (9)	.03[c]
Former smoker	8 (36)	11 (50)	
Never smoker	2 (9)	9 (41)	
Hypertension	15 (68)	6 (27)	.01
DM	6 (27)	1 (5)	.10[d]
Cardiac disease[e]	6 (27)	2 (9)	.24[d]
Transient ischemic attack	2 (9)	1 (4.5)	1.00[d]
COPD	3 (14)	2 (9)	1.00[d]
Osteoarthritis	3 (14)	1 (5)	.61[d]

ABI, Ankle-brachial index; *BMI,* body mass index calculated as weight (kg)/height (m^2); *COPD,* chronic obstructive pulmonary disease; *DM,* diabetes mellitus; *IC,* intermittent claudication; *SD,* standard deviation.
Data are presented as number (%) unless otherwise indicated.
[a]Index leg, most symptomatic leg in IC patients, randomly selected for control subjects.
[b]IC complaints of a single leg. Remaining patients had bilateral complaints of IC.
[c]Fisher exact test, calculation was based on never smoker and former smoker vs current smoker.
[d]Fisher exact test.
[e]Cardiac diseases included coronary artery disease, arrhythmia, pacemaker.

range. A Mann-Whitney *U* test and a Wilcoxon signed rank test were used to study these variables. Pearson correlation coefficients (ρ) were calculated to study associations between kinematic data and EMG data. Multivariate regression analyses were performed to study the effect of walking speed on kinematic and EMG outcomes. Statistical significance was applied at $P < .05$. SPSS software version 20.0 (IBM Corp, Armonk, NY) was used for the statistical analysis.

Sample size calculation was on the basis of duration of muscle activity per gait cycle, which was considered the primary outcome. Because data of IC patients were not available, data from patients after total hip arthroplasty instead were used.[14] Calculation (using an $\alpha = .05$ and a $\beta = .80$) revealed that 16 subjects per group were required to attain a statistical significant difference of 5% gastrocnemius activity.

RESULTS

Study population. The study population consisted of 22 IC patients and 22 age-matched control subjects. Baseline characteristics were similar, except for current smoking and hypertension, which were more prevalent among IC patients (Table I). ABI measurements of patients during rest and after treadmill walking were 0.64 and 0.35, respectively (Table I). A total of 9 patients (41%) had unilateral IC complaints. IC patients walked at a 1.4 km/h slower pace and demonstrated a significantly lower cadence and shorter step length compared with the control subjects (Table II).

Kinematic parameters in IC. Except for a significant difference in total knee ROM (Δ1.3°; $P \leq .019$), no differences were found in a comparison of pain-free and painful walking in patients with IC (Table II). However, evident differences were present between IC patients and control subjects. Patients with IC demonstrated approximately 7° less knee flexion after heel strike (IC: 10.9° vs control subjects: 18.4°; $P \leq .001$; Fig 2). There was also a significant difference in total ankle ROM (Δ7°; IC: 18.3° vs control subjects: 25.3°; $P \leq .001$), predominantly because of a decreased peak plantar flexion (Δ8.3°; IC: −10.2° vs control subjects: −18.5°; $P \leq .001$; Fig 3). Moreover, IC patients were found to exhibit less ankle ROM from initial heel contact to the flat foot position (Δ2.3°; IC: 3.3° vs control subjects: 5.6°; $P = .001$). No differences in variability of total ROM for the knee and ankle joints between IC patients and controls are shown in Table II.

In control subjects, a significant correlation was found between walking speed and peak plantar flexion ($\rho = -0.709$; $P \leq .001$). In contrast, this correlation was not found in IC patients ($\rho = -0.388$; $P = .074$). Multivariate regression analyses revealed a significant contribution of walking speed to the difference in maximal ankle plantar flexion between IC and control subjects ($P \leq .001$), accounting for 64% of the explained variance.

Surface EMG in IC. Analysis of the EMG signals included 19 control subjects (86%) and 18 IC patients (82%) because EMG data of 3 control subjects and 4 IC patients were corrupted and therefore not used.

Fig 4 shows the EMG activity of the MG and TA muscles in a typical IC patient in the present study. In IC patients, no differences were found in MG muscle activity in a comparison of pain-free and painful walking. The TA muscle activity duration, however, decreased significantly when claudication pain occurred (pain-free: 58% activity of the gait cycle, painful: 49% activity of the gait cycle; $P = .005$). In addition, a significant increase of peak muscle activity was found for both muscles during painful ambulation (MG, +54%; $P = .007$; TA, +23%; $P < .001$). For the comparison between patients and control subjects no differences were found for the MG muscle. Both groups used the MG muscle for approximately 43% of the gait cycle. Differences for the TA muscle tended to be more distinct, but were not significantly different (Table III).

For control subjects, walking speed was significantly correlated to the position of peak MG muscle activity, as a percentage of the gait cycle ($\rho = 0.543$; $P = .020$), whereas this correlation was not found in IC patients ($\rho = 0.068$; $P = .789$). Furthermore, no correlations

Table II. Gait parameters in intermittent claudication (*IC*) patients (n = 22) and control subjects (n = 22)

	IC patients					
Variable	*Pain-free*	*Painful*	P[a]	*Control subjects*	P[b]	P[c]
Walking speed, km/h	3.2 (0.6)			4.6 (0.8)	<.001	
Cadence, steps/min	110 (7.1)			122 (9.2)	<.001	
Step length, cm	48.2 (8.7)	48.8 (9.0)	.758	62.4 (10.6)	<.001	<.001
Knee heel strike[d]	12.0 (6.9)	11.2 (7.5)	.213	7.3 (5.2)	.014	.054
Knee heel strike to weight support ROM[e]	10.5 (5.7)	10.9 (6.0)	.238	18.4 (4.6)	<.001	<.001
Knee total ROM	56.2 (6.0)	57.5 (6.7)	.019	59.5 (5.2)	.056	.269
Knee SD total ROM	1.6 (0.2)	1.7 (0.3)	.138	1.6 (0.4)	.606	.231
Ankle plantar flexion	−10.6 (4.5)	−10.2 (3.8)	.372	−18.5 (6.5)	<.001	<.001
Ankle dorsiflexion	7.7 (4.2)	8.1 (3.9)	.156	6.7 (4.0)	.462	.265
Ankle heel strike ROM[f]	3.4 (2.0)	3.3 (2.2)	.643	5.6 (1.9)	.001	.001
Ankle total ROM	18.3 (3.5)	18.3 (3.6)	.961	25.3 (5.6)	<.001	<.001
Ankle SD ROM	1.2 (0.2)	1.2 (0.3)	.995	1.2 (0.3)	.446	.484

ROM, Range of motion; *SD,* standard deviation.
Data are presented as mean (± SD).
[a]Paired *t*-test between pain-free and painful trial.
[b]Independent samples *t*-test: pain-free vs control.
[c]Independent samples *t*-test: painful vs control.
[d]Knee angle during heel strike (initial contact).
[e]ROM between heel strike and complete weight support.
[f]ROM of the ankle between heel strike and flat foot position.

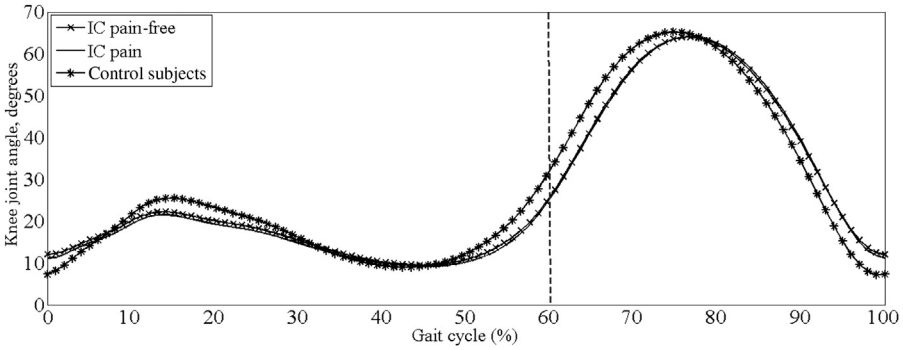

Fig 2. Knee joint angles in the sagittal plane during the complete gait cycle (ie, heel strike to heel strike) for intermittent claudication (*IC*) patients during pain-free walking (*x*), painful walking (-), and control subjects (*). The *dashed vertical line* separates the stance phase (approximately 60%) and swing phase (approximately 40%) of the gait cycle.

were found between EMG parameters and kinematic outcomes.

DISCUSSION

The purpose of this study was to evaluate kinematic gait parameters and muscle activity assessed via EMG in patients with IC and in age-matched controls. To our knowledge, this is the first study to investigate EMG and kinematic parameters using this type of methodology in this patient population. Patients with IC showed a substantially reduced ankle joint displacement during initial contact (−41%) and the propulsion phase (−45%), which resulted in a significantly decreased total ankle ROM compared with control subjects. Interestingly, no additional differences were found in the ankle joint angles when claudication pain presented. The surface EMG analysis revealed no significant differences between IC patients and control subjects regarding muscular activity duration of the MG and TA muscles. However, painful walking did result in an increased amount of muscle activity (ie, EMG amplitude) within the group of IC patients. These findings suggest that IC pain due to ischemia coincides with an increased recruitment of motor units and muscle fatigue.

Limb movements are induced by muscles acting over a joint. The lesser ankle ROM found in the present study might be considered a compensatory mechanism to minimize the requested duration of muscle contraction (ie, reduced workload). This adaptation might be beneficial during painful walking in particular, because limited blood

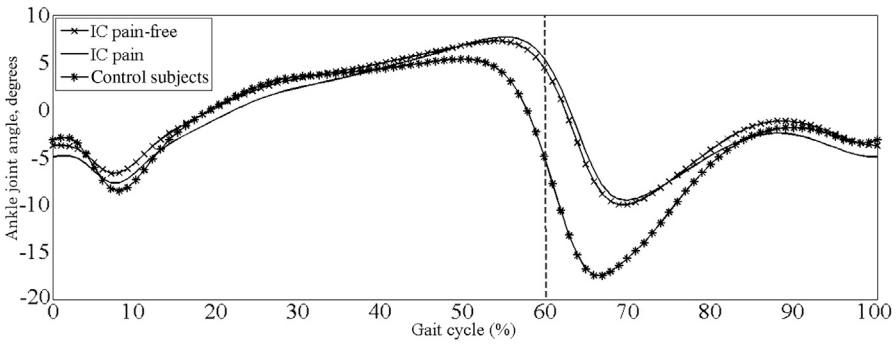

Fig 3. Ankle joint angles in the sagittal plane during the complete gait cycle (ie heel strike to heel strike) for intermittent claudication (*IC*) patients during pain-free walking (*x*), painful walking (-), and control subjects (*).The *dashed vertical line* separates the stance phase (approximately 60%) and swing phase (approximately 40%) of the gait cycle. Plantar flexion angles are expressed as negative values, and dorsal flexion angles as positive values.

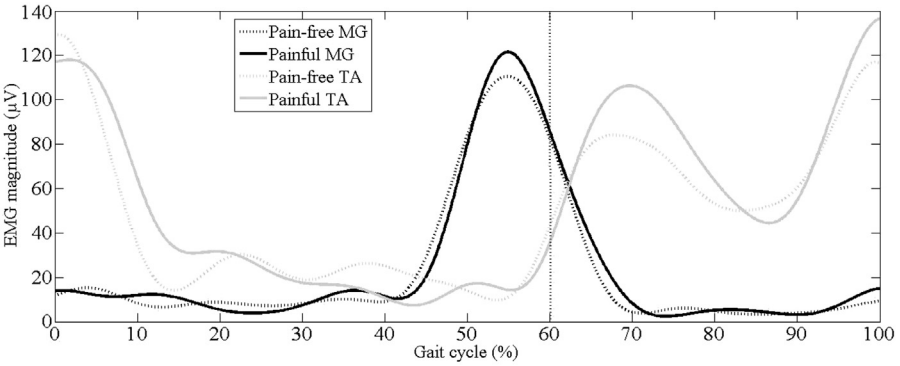

Fig 4. Graphs showing the level of muscle activity of a typical intermittent claudication (IC) patient of the present study. Data of the medial gastrocnemius (*MG*; *black solid* and *dashed line*) and tibialis anterior (*TA*; *gray solid* and *dashed line*) muscles, during the complete gait cycle for respectively, pain-free and painful ambulation. The *dashed vertical line* separates the stance phase (approximately 60%) and the swing phase (approximately 40%) of the gait cycle.

flow and oxygen (ie, ischemia) are thought to hinder muscle contraction. Our data, however, indicated that IC patients use their lower limb muscles to a similar percentage of the gait cycle as do control subjects. Therefore, evidence for our assumption that such data are strongly coupled is not yet provided. These findings, however, support previous studies that also found no changes in calf muscle activity despite evident changes in ankle movements.[21]

A small number of studies on gait patterns in patients with IC have reported conflicting results. For instance, one group described reduced plantar flexion and total ankle ROM, in line with our results.[22] A second group found an increased ankle ROM[23]; however, they did not include peak plantar flexion during the start of the swing phase, a moment when the largest ankle plantar flexion is generally found (Fig 3). It must be appreciated that the present study was unique in its assessment of gait parameters during continuous treadmill walking. Previous research groups based their conclusions on just a limited number of steps[22,23] and a larger number has been recommended.[24] On the basis of this optimized and standardized methodology, we are confident that the results of the present study reflect true alterations in the gait pattern of IC patients.

Walking in IC patients is known to occur at a much greater cost of transport (ie, walking economy). As a consequence, IC patients consume more oxygen and/or energy per unit walking distance then matched control subjects.[7,25] This phenomenon was judged by some as a consequence of an altered mechanical gait pattern,[7] because slower walking speeds are, for instance, known to decrease metabolic efficiency.[26] We observed evident kinematic differences in the ankle joint that can in part be explained by the difference in walking velocity between IC and control subjects. Causality could not be tested because of the cross-sectional nature of the present study, but results imply that both variables are related. Correction of the observed gait abnormalities might therefore contribute to an improved metabolic efficiency and will possibly allow IC patients to continue walking, whereas ischemic pain will likely be postponed.

In the present study we found that gait abnormalities became evident before the onset of claudication pain.

Table III. Electromyographic analysis in intermittent claudication (*IC*) patients (n = 18) and healthy control subjects (n = 19)

	IC patients					
Variable	*Pain-free*	*Painful*	P[a]	*Control subjects*	P[b]	P[c]
Gastrocnemius activity, %[d]	43.7 (7.0)	42.6 (9.6)	.497	43.1 (7.0)	.829	.773
Tibialis anterior activity, %[d]	58.0 (12.1)	49.0 (7.9)	.005	53.5 (11.6)	.268	.127
Gastrocnemius peak activity, μV[e]	67 (46-97)	103 (83-134)	.007[c]	91 (57-156)	-	-
Tibialis anterior peak activity, μV[e]	105 (50-127)	129 (101-185)	<.001[c]	135 (98-169)	-	-
Gastrocnemius peak position, %[f]	45.9 (11.4)	44.6 (14.1)	.604	45.3 (11.8)	.870	.866

SD, Standard deviation.
Data are presented as mean (± SD) or median (interquartile range).
[a]Paired *t*-test between pain-free and painful trial, except for where noted with footnote 'e'.
[b]Independent samples *t*-test: pain-free vs control.
[c]Independent samples *t*-test: painful vs control.
[d]Duration of muscle activation as percentage of the complete gait cycle.
[e]Wilcoxon signed-rank test.
[f]Position of the peak muscular activity in percentage of the complete gait cycle.

Therefore, mechanisms other than restricted blood flow and IC pain might be responsible for the observed ambulatory dysfunction. For instance, reduced calf muscle strength,[27] as oxidative damage due to exercise-induced ischemia, might contribute to metabolic myopathy in the lower extremity muscles.[28,29] Alternatively, increased fatigability and predominance of type II muscle fibers (ie, anaerobic and fatigue-prone) of the MG were also found in IC patients.[30] Muscle weakness and fatigue led to an increased recruitment of motor units and an increase in EMG amplitude when similar work load (ie, constant walking speed) was applied. These alterations might be reflected by significantly higher EMG amplitudes during painful ambulation, as found in this study. Because fatigue is generally considered an important control parameter for muscular training, current results provide evidence for the beneficial effect of SET at its intensity near maximal pain level, as recommended.[31]

The current research might fuel the search for novel training modalities of SET aimed to improve gait pattern. Because plantar flexion is strongly associated with walking speed,[32] one might first focus on improvement of plantar flexion during the preswing phase. It has been suggested that a reduced plantar flexion and subsequent inability to propel the body forward also contribute to the increased risk of falling in elderly.[33] Therefore, restoration of gait patterns might be beneficial from this perspective as well. Moreover, the effect of strength training on gait parameters in this patient population might be promising. A recent meta-analysis in IC already concluded that progressive resistance training with high intensities is a most effective exercise modality for improvement of gait speed.[34] Furthermore, lower limb strength training was shown to improve gait kinematics in elderly women.[35]

Some limitations of the present study need to be addressed. First, only participants without additional comorbidities (eg, osteoarthritis or previous lower limb surgery) were included thereby factors that could potentially influence gait pattern were excluded. The present findings were obtained in patients who were in a relatively good condition, and results might be overestimated in favor of a relatively normal gait pattern. However, participants with diabetes mellitus (DM) were not excluded. One study reported lower ankle generative mechanical work in patients with DM, even in the absence of diabetic neuropathy.[36] Because DM was more prevalent among patients with IC, reduced joint ankle ROM was possibly overestimated in the IC group. Second, data as captured during treadmill walking might not necessarily be similar to data that are obtained during over ground walking. However, very equal characteristics of gait were reported,[37] despite slight differences in stance duration and muscle activation.[38] It is thus reasoned that movement analysis during treadmill walking reveals outdoor day-to-day situations, all the more so when other advantages of treadmill-based protocols are taken into account. However, caution should be taken when comparing the present EMG data with results acquired from over ground walking protocols. The same might be true regarding bare-foot walking compared with walking using common footwear, because footwear is found to influence kinematics during walking. To exclude differences in gait pattern because of variable footwear, we decided to have all participants walk without shoes.[39] Third, we only studied two lower limb muscles. Although these muscles are considered main actuators of the ankle[17] and being the 'functional end organs' in the lower extremity ischemia,[40] it would be of interest to include additional muscles in future research. It must also be appreciated that surface EMG registration inherently has certain restrictions. EMG refers to the collective electric signal from muscles produced during contraction and the amplitude of an EMG signal is known to be dependent on several factors, such as the properties of the overlying tissue (eg, thickness of overlying skin and adipose tissue). It is therefore not possible to compare raw EMG amplitudes across subjects. So, surface EMG should be used for studying muscle activation patterns and activity duration only. In contrast, muscle force is not reflected by this method. Last, we did not

examine passive range of joint motion. Therefore, kinematic differences might possibly be attributed in part to a difference in passive ROM. However, participants with a history of orthopedic complaints, functional loss in ROM, or observational abnormities during walking were excluded.

CONCLUSIONS

Patients with IC showed significant changes in kinematic parameters during walking, even before the onset of claudication pain. However, these changes did not coincide with differences in EMG muscle activity duration of the medial gastrocnemius and anterior tibialis muscles.

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AUTHOR CONTRIBUTIONS

Conception and design: LG, FB, KM, JT
Analysis and interpretation: LG, AS, FB, EvD, KM
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Writing the article: LG, AS, MS, KM
Critical revision of the article: MS, EvD, FvdL, KM, JT
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REFERENCES

1. Hirsch AT. Treatment of peripheral arterial disease—extending "intervention" to "therapeutic choice". N Engl J Med 2006;354:1944-7.
2. Lauret GJ, Fokkenrood HJ, Bendermacher BL, Scheltinga MR, Teijink JA. Physical activity monitoring in patients with intermittent claudication. Eur J Vasc Endovasc Surg 2014;47:656-63.
3. 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.
4. Mockford KA, Vanicek N, Jordan A, Chetter IC, Coughlin PA. Kinematic adaptations to ischemic pain in claudicants during continuous walking. Gait Posture 2010;32:395-9.
5. Gardner AW, Forrester L, Smith GV. Altered gait profile in subjects with peripheral arterial disease. Vasc Med 2001;6:31-4.
6. Scott-Pandorf MM, Stergiou N, Johanning JM, Robinson L, Lynch TG, Pipinos II. Peripheral arterial disease affects ground reaction forces during walking. J Vasc Surg 2007;46:491-9.
7. Marconi C, Ferretti G, Anchisi S, Catalano M, Scandale G, Antico A, et al. Energetics of walking in patients with peripheral arterial disease: a proposed functional evaluation protocol. Clin Sci (Lond) 2003;105:105-11.
8. Koutakis P, Weiss DJ, Miserlis D, Shostrom VK, Papoutsi E, Ha DM, et al. Oxidative damage in the gastrocnemius of patients with peripheral artery disease is myofiber type selective. Redox Biol 2014;2:921-8.
9. Regensteiner JG, Wolfel EE, Brass EP, Carry MR, Ringel SP, Hargarten ME, et al. Chronic changes in skeletal muscle histology and function in peripheral arterial disease. Circulation 1993;87:413-21.
10. Benedetti MG, Agostini V, Knaflitz M, Bonato P. Muscle activation patterns during level walking and stair ambulation. Chapter 8. In: Steele C. Applications for EMG in clinical and sports medicine. InTech; January 11, 2012. ISBN 978-953-307-798-7.
11. Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. J Electromyogr Kinesiol 2000;10:361-74.
12. Spulak D, Cmejla R, Bacakova R, Kracmar B, Satrapova L, Novotny P. Muscle activity detection in electromyograms recorded during periodic movements. Comput Biol Med 2014;47:93-103.
13. Chowdhury RH, Reaz MB, Ali MA, Bakar AA, Chellappan K, Chang TG. Surface electromyography signal processing and classification techniques. Sensors (Basel) 2013;13:12431-66.
14. Agostini V, Ganio D, Facchin K, Cane L, Moreira Carneiro S, Knaflitz M. Gait parameters and muscle activation patterns at 3, 6 and 12 months after total hip arthroplasty. J Arthroplasty 2014;29:1265-72.
15. Gardner AW, Skinner JS, Cantwell BW, Smith LK. Progressive vs single-stage treadmill tests for evaluation of claudication. Med Sci Sports Exerc 1991;23:402-8.
16. Van de Putte M, Hagemeister N, St-Onge N, Parent G, de Guise JA. Habituation to treadmill walking. Biomed Mater Eng 2006;16:43-52.
17. He H, Kiguchi K, Horikawa E. A study on lower-limb muscle activities during daily lower-limb motions. Int J Bioelectromagn 2007;9:79-84.
18. Askew CD, Green S, Walker PJ, Kerr GK, Green AA, Williams AD, et al. Skeletal muscle phenotype is associated with exercise tolerance in patients with peripheral arterial disease. J Vasc Surg 2005;41:802-7.
19. Bogey R, Cerny K, Mohammed O. Repeatability of wire and surface electrodes in gait. Am J Phys Med Rehabil 2003;82:338-44.
20. Ozgunen KT, Celik U, Kurdak SS. Determination of an optimal threshold value for muscle activity detection in EMG analysis. J Sports Sci Med 2010;9:620-8.
21. Forghany S, Nester CJ, Richards B, Hatton AL, Liu A. Rollover footwear affects lower limb biomechanics during walking. Gait Posture 2014;39:205-12.
22. Crowther RG, Spinks WL, Leicht AS, Quigley F, Golledge J. Relationship between temporal-spatial gait parameters, gait kinematics, walking performance, exercise capacity, and physical activity level in peripheral arterial disease. J Vasc Surg 2007;45:1172-8.
23. Celis R, Pipinos II, Scott-Pandorf MM, Myers SA, Stergiou N, Johanning JM. Peripheral arterial disease affects kinematics during walking. J Vasc Surg 2009;49:127-32.
24. Di Nardo F, Ghetti G, Fioretti S. Assessment of the activation modalities of gastrocnemius lateralis and tibialis anterior during gait: a statistical analysis. J Electromyogr Kinesiol 2013;23:1428-33.
25. Gardner AW, Ritti-Dias RM, Stoner JA, Montgomery PS, Khurana A, Blevins SM. Oxygen uptake before and after the onset of claudication during a 6-minute walk test. J Vasc Surg 2011;54:1366-73.
26. 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 (1985) 2003;95:2248-56.
27. Chen SJ, Pipinos I, Johanning J, Radovic M, Huisinga JM, Myers SA, et al. Bilateral claudication results in alterations in the gait biomechanics at the hip and ankle joints. J Biomech 2008;41:2506-14.
28. Pipinos II, Sharov VG, Shepard AD, Anagnostopoulos PV, Katsamouris A, Todor A, et al. Abnormal mitochondrial respiration in skeletal muscle in patients with peripheral arterial disease. J Vasc Surg 2003;38:827-32.
29. Koutakis P, Myers SA, Cluff K, Ha DM, Haynatzki G, McComb RD, et al. Abnormal myofiber morphology and limb dysfunction in claudication. J Surg Res 2015;196:172-9.
30. Gasparini M, Sabovic M, Gregoric ID, Simunic B, Pisot R. Increased fatigability of the gastrocnemius medialis muscle in individuals with intermittent claudication. Eur J Vasc Endovasc Surg 2012;44:170-6.
31. Merry AH, Teijink JA, Jongert MW, Poelgeest A, van der Voort SS, Bartelink ME, et al. [KNGF-guideline symptomatic peripheral arterial disease (Author's translation)]; 2014.
32. Winter DA. Biomechanical motor patterns in normal walking. J Mot Behav 1983;15:302-30.

33. McGibbon CA. Toward a better understanding of gait changes with age and disablement: neuromuscular adaptation. Exerc Sport Sci Rev 2003;31:102-8.
34. Van Abbema R, De Greef M, Craje C, Krijnen W, Hobbelen H, Van Der Schans C. What type, or combination of exercise can improve preferred gait speed in older adults? A meta-analysis. BMC Geriatr 2015;15:72.
35. Persch LN, Ugrinowitsch C, Pereira G, Rodacki AL. Strength training improves fall-related gait kinematics in the elderly: a randomized controlled trial. Clin Biomech (Bristol, Avon) 2009;24:819-25.
36. Ko SU, Stenholm S, Chia CW, Simonsick EM, Ferrucci L. Gait pattern alterations in older adults associated with type 2 diabetes in the absence of peripheral neuropathy—results from the Baltimore Longitudinal Study of Aging. Gait Posture 2011;34:548-52.
37. Riley PO, Paolini G, Della Croce U, Paylo KW, Kerrigan DC. A kinematic and kinetic comparison of overground and treadmill walking in healthy subjects. Gait Posture 2007;26:17-24.
38. Lee SJ, Hidler J. Biomechanics of overground vs. treadmill walking in healthy individuals. J Appl Physiol (1985) 2008;104:747-55.
39. Franklin S, Grey MJ, Heneghan N, Bowen L, Li FX. Barefoot vs common footwear: a systematic review of the kinematic, kinetic and muscle activity differences during walking. Gait Posture 2015;42:230-9.
40. Koutakis P, Pipinos II, Myers SA, Stergiou N, Lynch TG, Johanning JM. Joint torques and powers are reduced during ambulation for both limbs in patients with unilateral claudication. J Vasc Surg 2010;51:80-8.

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