

The effect of diabetic neuropathy and previous foot ulceration in EMG and ground reaction forces during gait

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Abstract

Background. We aimed at investigating the influence of diabetic neuropathy and previous history of plantar ulcers on electromyography (EMG) of the thigh and calf and on vertical ground reaction forces during gait.

Methods. This study involved 45 adults divided into three groups: a control group ($n = 16$), diabetic neuropathic group ($n = 19$) and diabetic neuropathic group with previous history of plantar ulceration ($n = 10$). EMG of the right vastus lateralis, lateral gastrocnemius and tibialis anterior were studied during the stance phase. The peaks and time of peak occurrence were determined and a co-activation index between tibialis anterior and lateral gastrocnemius. In order to represent the effect of the changes in EMG, the first and second peaks and the minimum value of the vertical ground reaction force were also determined. Inter-group comparisons of the electromyographical and ground reaction forces variables were made using three MANCOVA (peaks and times of EMG and peaks of force) and one ANCOVA (co-activation index).

Findings. The ulcerated group presented a delayed in the time of the lateral gastrocnemius and vastus lateralis peak occurrence in comparison to control's. The lateral gastrocnemius delay may be related to the lower second vertical peak in diabetic subjects. However, the delay of the vastus lateralis did not cause any significant change on the first vertical peak.

Interpretations. The vastus lateralis and lateral gastrocnemius delay demonstrate that ulcerated diabetic neuropathic patients have a motor deficit that could compromise their ability to walk, which was partially confirmed by changes on ground reaction forces during the push-off phase.

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1. Introduction

Diabetic neuropathy is the most common chronic complication associated with Diabetes Mellitus, affecting 20–50% of diabetic patients 10 years after their diagnosis (Cavanagh et al., 1993). Diabetic neuropathy leads to a progressive loss of somatosensory sensitivity, proprioception and distal muscle function (Pirart, 1979; Pickup and Williams, 1981; Yavuzer et al., 2006), especially in the

lower limbs, which may cause an alteration of the motor control during gait and during static posture (Mueller et al., 1994; Shaw et al., 1998; Katoulis et al., 1997; Beek et al., 1998; Abboud et al., 2000; Sacco and Amadio, 2000, 2003; Yavuzer et al., 2006). These alterations may increase the risk of falling in diabetic neuropathic patients (Cavanagh et al., 1992; Simmons and Richardson, 2001).

The sensory and motor diabetic neuropathy modifies the amount and quality of sensory information necessary for motor control. Consequently, there is a higher instability during gait and static posture (Richardson et al., 1992), that previously has been considered to be due to muscular weakness (Courtemanche et al., 1996). Gait in humans is

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considered the result of harmonic interplay neural and muscular actions coordinated with skeletal function (Katoulis et al., 1997), and changes in this harmonic relationship will cause important alterations in the locomotor pattern. These alterations are considered the result of a loss of sensitivity and may result in biomechanical alterations of gait, such as plantar pressures changes, usually with higher pressures on the forefoot (Caselli et al., 2002; Gefen, 2003; Perry et al., 2002), differences in kinematic patterns (Fernando et al., 1991; Mueller et al., 1994; Katoulis et al., 1997; Shaw et al., 1998; Sauseng and Kastenbauer, 1999; Sacco and Amadio, 2000; Kwon et al., 2003; Menz et al., 2004; Petrofsky et al., 2005; Yavuzer et al., 2006), differences in kinetic patterns with modified ground reaction forces (Katoulis et al., 1997; Shaw et al., 1998; Sacco and Amadio, 2000), and altered muscle activity (Abboud et al., 2000; Sacco and Amadio, 2003; Kwon et al., 2003).

As diabetic neuropathy develops, the somatosensory inputs diminish, and the motor outputs become progressively more impaired, which may accentuate the changes in the locomotor pattern of diabetic neuropathic subjects. The biomechanical changes during the gait of diabetics, in conjunction with diabetic autonomic impairments, may lead to foot ulcerations. In this way, we may conclude that the presence of foot ulceration may be an indicator of the worsening of diabetic neuropathy. Neuropathic ulceration is the most prevalent type of long-term chronic injury for diabetic subjects (Piaggese, 2004).

So far, the main findings from the study of electrical muscle activity in diabetic neuropathic subjects, are that the electrical muscle activity present a delayed activation of the gastrocnemius, soleus, peroneus brevis and longus muscles, and most importantly, of the tibialis anterior muscle, during gait, which may be associated with an earlier forefoot contact with the ground (Abboud et al., 2000; Sacco and Amadio, 2003). Still, a delayed activation of the vastus lateralis is mentioned during the initial contact of gait that may be indicative of the presence of a deficiency in the shock attenuation mechanisms (Sacco and Amadio, 2003). Furthermore, it has been found that there is a premature activation of the soleus and medial gastrocnemius, and a prolonged tibialis anterior activity, leading to a co-contraction of these muscles during mid-stance in a possible attempt to improve foot stability. This also causes an earlier contact of the forefoot, and a decrease of shock absorption at the time of heel strike, which may lead to an altered plantar pressure distribution, and may also be related to plantar ulceration on the forefoot (Kwon et al., 2003).

Changes in muscle activity could lead to other alterations during gait, besides the plantar pressure distribution. It is already described that diabetic neuropathic subjects present modified ground reaction forces (GRF) during gait (Shaw et al., 1998; Sacco and Amadio, 2000; Santos and Barela, 2002). These alterations could be related to the slower walking velocity they adapt (Katoulis et al., 1997). What is still unknown is if the diabetic neuropathy itself

is in fact leading to significant GRF changes, in addition to a reduction in gait velocity.

Investigations of muscle activity alterations during gait in diabetic neuropathic subjects are still very scarce. The results in literature are controversial; and it is not yet clear how peripheral diabetic neuropathy affects muscle activity and the kinetic responses during gait. Whether muscle activity patterns change along with the evolution of the disease and what consequences altered muscle activity has on the kinetics of diabetes gait function in regard to ground reaction forces, remains unclear. Previous studies have not distinguished between the degrees of neuropathy in their experimental groups; therefore, it has not been possible to identify differences in gait patterns between the early and advanced stages of the disease. The hypothesis of the present study was that late stages of diabetic neuropathy, which is represented here by at least one occurrence of plantar ulceration in the patient's clinical evolution, would lead to greater biomechanical alterations during gait.

So, the purpose of this study was to investigate and compare the electromyographical activity of the thigh and calf muscles during gait among non-diabetic subjects and diabetic neuropathic patients in two stages of the disease: those with and those without previous experience of ulcers in their clinical history. This study also attempted to verify if the changes in EMG do, in fact, cause any alteration in GRF during gait. Within these descriptions of gait characteristics, we speculate about the influence of diabetic peripheral neuropathy and its progression in gait as well as the possible dynamic mechanisms developed to compensate for sensory and motor deficits.

2. Methods

2.1. Subjects

This prospective study involved 45 volunteer adult males and females divided into three groups: the control group (CG) ($n = 16$), a diabetic neuropathic group (DG) ($n = 19$) and a diabetic neuropathic group with previous history of plantar ulceration in the last two years (UDG) ($n = 10$). Ethics approval was obtained from the local institution (Protocol No. 1268/05).

All neuropathic subjects (DG and UDG) were diagnosed by physicians. The following inclusion criteria were adopted for patients: at least 5 years post-onset of Type 2 diabetes; presentation of at least two plantar areas with tactile insensitivity to the 10-g monofilament (Frykberg et al., 1998; Armstrong and Lavery, 1998; Perry et al., 2002), and a minimum score of 6 on the Michigan Neuropathy Screening Instrument-questionnaire (MNSI-q) (Feldman et al., 1994). The MNSI-q is a validated instrument for screening the symptoms related to diabetic neuropathy. In addition, the UDG subjects had at least one previous plantar ulcer in the last two years. The exclusion criteria adopted for all experimental groups included: age over 65 years, partial or total amputation, Charcot arthropathy

or any other major orthopedic foot alteration confirmed by radiography, presence of peripheral or central neurological disease not caused by diabetes, a history of alcohol abuse, the presence of plantar ulcers at the time of the evaluation, and an inability to walk independently without pain or the use of an assistive device.

Furthermore, we used a modified MNSI form in attempt to investigate the foot inspection looking for evidence of excessively dry skin, callous formation, fissures, frank ulceration or deformities such as flat feet, hammer toes, overlapping toes, halux valgus, joint subluxation, and prominent metatarsal heads. The ankle reflexes (Achilles tendon reflex) were also examined by the MNSI form using an appropriate reflex hammer. The original MNSI form includes vibration perception that was excluded from our screening due to lack of equipment. Thus, the score on the MNSI form was based on a total of 6 points. We also excluded the diabetics with a MNSI form score higher than 4, in order to exclude subjects with substantial foot deformities. The arch index (Cavanagh and Rodgers, 1987) was also evaluated in order to excluded major arch alterations (equinus planus feet and extra cavus feet) that could interfere in gait mechanics.

2.2. Experimental procedures

The procedures were explained to the subjects, and they signed an informed consent form before the beginning of the study. A preliminary investigation was completed in order to verify participation criteria including anthropometric and demographic measures (age, body mass, height and body mass index) and clinical data (glycemia level and length of time since diagnosis of diabetes). After the preliminary examination was completed, the subjects were assigned to one of the three experimental groups.

As one of the inclusion criteria, the tactile perception test was made in order to quantify how many areas present a sensory deficit. From a total of 10 areas from both feet, the number of areas in which the subjects did not feel the touch of the 10-g monofilament was counted.

The electrical activity of the right vastus lateralis (VL), lateral gastrocnemius (LG) and tibialis anterior (TA) muscles were measured and evaluated during the stance phase of barefoot gait. The motor behavior studied in the present paper is basically a symmetric locomotor task (Winter, 1991; Perry, 1992) that is not influenced by limb dominance. The peripheral diabetic neuropathy can be bi or unilateral, and can thus affect one or both sides independently of the limb side or the limb dominance. This fact depends purely on the laws of probability. Therefore, only the muscles from the right lower limb were collected. Thus, Sacco and Amadio (2000) found no differences between right and left lower limb biomechanical responses during the gait of diabetic neuropathic patients. The subjects were requested to walk barefoot at a self-selected cadence across a 10 m walkway with a force plate embedded in its center. The EMG activity was recorded during three trials using

the EMG System do Brasil (Sao José dos Campos, Brazil). The bipolar surface electrodes were placed according to SENIAM recommendations (SENIAM, 2006). The diameter of the electrodes was 10 mm and the inter-electrode distance was 25 mm. The electrodes were attached to the skin using Transpore adhesive tape and an elastic band after shaving the area and cleansing it with alcohol.

The electromyographical data were collected simultaneously at a sampling frequency of 1000 Hz and synchronized to the vertical GRF for periods of 6 s. The force plate data were acquired using an AMTI force plate (Watertown, USA). Besides the acquisition of the vertical forces, the GRF data were also used to determine the stance phase for the EMG signal of all subjects.

Three trials were recorded for each subject. The sessions lasted about 1 h each and the subjects could rest if they felt tired during the data collection. The motor skill performed during the study (to walk barefoot in a flat walkway) is a well-learned motor skill, because it is part of a motor pattern achieved during human development and it is the locomotor task used most in daily living activities. Therefore, we assumed that during data acquisition, the subjects would not present any signs of fatigue, and this, was in fact, observed later during data collection procedures.

2.3. Numerical and statistical analysis

EMG activity was represented through linear envelopes performed after a few steps: after the offset removal from the raw EMG, the signals were full-wave rectified, filtered through a zero lag 4th order Butterworth low pass filter with cutoff frequency at 5 Hz, normalized by the EMG signal mean, and time base normalized by the support time, which was determined using the GRF curve as a reference. The GRF data were also mathematically treated. After the offset removal, the signals were also filtered through a zero lag low pass Butterworth 4th order with a cutoff frequency of 100 Hz, then normalized by each subject's body weight and time base normalized by the support time (0–100% of stance phase).

After that, two EMG variables from each muscle for each step within each trial per subject (3 values for each variable per subject) were determined, using a mathematical function written in Matlab software. These variables were the peak value of the linear envelope curve of each muscle and the time, from 0 to 100% of stance phase when this peak occurred (Fig. 1). The variables were represented as their means and standard deviations for the three DG, UDG and CG groups. To ensure the validity of the computer derived EMG and GRF variables, each trace was also visually inspected to ensure that any movement artifacts or any other interference was not identified incorrectly as a muscle activity signal or force interference.

The GRF variables that were determined automatically by Matlab function were the first and second peak of force, and the minimum value of force between the two peaks. The EMG variables calculated from the linear envelope

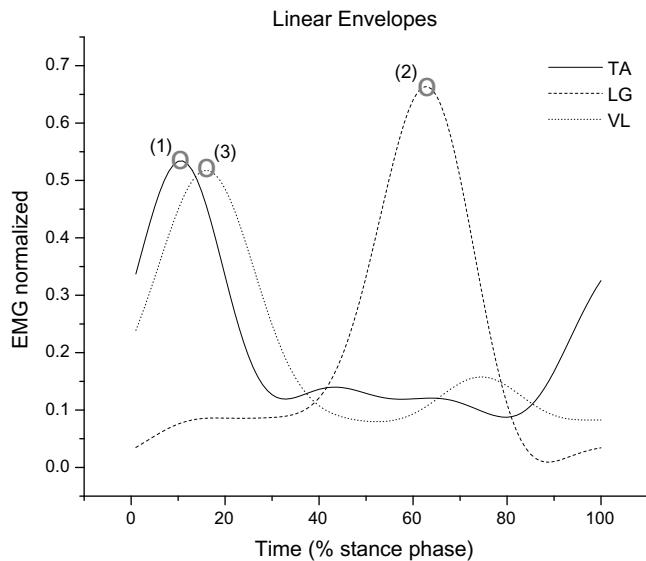


Fig. 1. Representation of the linear envelopes variables of the TA, LG and VL muscles during the stance phase: (1) time of peak occurrence of TA (x label – % of stance phase) and maximum peak of TA (y label – normalized by the mean of each subject), (2) time of peak occurrence of LG (x label – % of stance phase) and maximum peak of LG (y label – normalized by the mean of each subject), (3) time of peak occurrence of VL (x label – % of stance phase) and maximum peak of VL (y label – normalized by the mean of each subject).

curves were the maximum peaks and the time of peak occurrence for the TA, VL and LG muscles, and a co-activation index (Falconer and Winter, 1986) calculated according to the ratio between the TA and LG IEMG, in the period from the time of the heel strike to the peak of the horizontal anterior–posterior GRF. We assumed that this period represents the phase in the gait cycle that requires the maximum stability of the foot and ankle complex, in order to better accommodate loads at the beginning of the stance phase, and to ensure a better stability at the start of propulsion phase. The co-activation index was calculated using the following mathematical equation, which was adapted from Falconer and Winter (1986):

$$\text{co-activation} = 2 \cdot \frac{\text{IEMG}_{\text{TA}}}{\text{IEMG}_{\text{TA}} + \text{IEMG}_{\text{LG}}} \quad (1)$$

where IEMG means the integral of the rectified electromyographical sign which quantifies the amount of electrical muscle activity, TA is the abbreviation for the tibialis anterior muscle and LG is the abbreviation for lateral gastrocnemius.

The descriptive statistics of EMG and GRF variables and of the anthropometric and clinical data of the subjects were expressed as means and standard deviations. Data were tested according to normal distribution using the Shapiro–Wilk’s test, and confirmed the Gaussian pattern of distribution. Also, the homoscedasticity of each studied variable was checked by Levene’s test. As the total time of stance phase was different among groups, according to an ANOVA test performed earlier, inter-group comparisons

of the EMG and GRF variables were made using three MANCOVAs (EMG peaks, time of EMG peak occurrence and vertical GRF variables), and one ANCOVA for the co-activation index, using as a covariate the total contact time, in order to include this variable as an intervenient factor and to evaluate its influence on the biomechanical variables. After that, ANCOVA tests were performed for each variable within each MANCOVA, and if there were any statistical differences, the Tukey post hoc test was performed. Inter-group comparisons of the clinical data were performed using the *t*-test for independent samples, and the Mann–Whitney test for the MNSI score. The number of areas with tactile deficit was compared between the three groups using a Kruskal Wallis test and the Mann–Whitney post hoc test. We adopted an error level of 5%. Statistics were performed using SPSS software (SPSS, Inc.). Mathematical functions were carried out using Matlab Version 6.5 (Mathworks) and Origin Version. 6.0 (Microcal Software).

3. Results

The experimental groups’ demographic and clinical data are described in Table 1. The groups did not differ in terms of age or gender, but both diabetic groups (DG and UDG) had a significantly higher body mass index (BMI) when compared to the CG; however, the DG and UDG were similar in BMI value, time of diagnosis of diabetes, glycemia level, and their MNSI score. According to Table 1, their glycemia level was higher than what is expected for good diabetes control (140 mg/dl) (American Diabetes Association, 1996). Subjects from the CG did not present any tactile deficits on the plantar surface, as expected, but the UDG subjects presented a median of 8 plantar areas with tactile deficits, and these deficits were significantly worse than the DG deficit (a median of 2.5 plantar areas) ($P = 0.000$) However, *t*-tests showed that the mean number of areas with tactile deficit between the left (DG = 1; UDG = 2) and right (DG = 0.5; UDG = 4) foot was similar in both diabetic groups ($P > 0.05$). The groups

Table 1

Mean and standard deviation (SD) of age, height, body mass index (BMI), time of diagnosis of diabetes mellitus (DM), last glycemia level, and median of the MNSI-questionnaire score

	CG (n = 16)	DG (n = 19)	UDG (n = 10)	<i>P</i>
Age (years)	51.1 (8.3)	57.6 (8.5)	53.8 (7.9)	0.092 ^a
Gender (% female)	50	42	50	
BMI (kg/m ²)	23.9 (2.9) ^c	26.6 (4.2)	27.8 (4.6)	0.029 ^a
Time of DM (years)	–	12.6 (5.3)	16.4 (8.5)	0.151 ^a
Glycemia (mg/dl)	–	164.4 (58.8)	180.1 (63.6)	0.521 ^a
MNSI score	–	7	7.5	0.917 ^b

^a One-way ANOVA.

^b Mann–Whitney test.

^c The significantly different group.

Table 2

Mean values (SD) of the magnitude and time of the activation peak of the right vastus lateralis (VL), tibialis anterior (TA) and lateral gastrocnemius (LG) muscles, and of the co-activation index on CG, DG and UDG

	CG (n = 16)	DG (n = 19)	UDG (n = 10)	MANCOVA		P-value (ANOVA)
				P-value	P Wilk's Lambda	
TA peak (% mean)	2.85 (0.73)	3.04 (0.67)	2.78 ± 0.62	$F(6, 78) = 0.67, P = 0.67$	0.93	0.48
LG peak (% mean)	2.72 (0.49)	2.60 (0.51)	2.42 (0.44)			0.36
VL peak (% mean)	2.49 (0.70)	2.61 (0.60)	2.48 (0.47)			0.79
TA peak time (% support)	6.05 (2.15)	6.10 (1.68)	4.64 (1.59)	$F(6, 78) = 2.27, P = 0.04$	0.26	0.76
LG peak time (% support)	63.53 (3.65)	62.84 (5.06)	68.00 (4.78) ^a			0.06
VL peak time (% support)	10.82 (3.33)	11.97 (2.31)	14.83 (3.53) ^a			0.02
Co-activation index	0.96 (0.05)	0.93 (0.05)	0.93 (0.08)			0.14
Fz1 (BW)	1.05 (0.09)	1.07 (0.07)	1.05 (0.06)	$F(6, 78) = 2.27, P = 0.00$	0.00	0.64
Fz2 (BW)	1.09 (0.07)	1.05 (0.06)	1.02 (0.06)			0.01
Fzmin (BW)	0.80 (0.07)	0.78 (0.08)	0.82 (0.05)			0.90

^a The group statistically different.

also presented similar distributions (Chi-square = 1.535, df = 4, P = 0.820) of low, normal and high longitudinal arch (CG: normal – 48%, low – 18%, high – 35%; DG: normal – 57%, low – 17%, high – 27%; UDG: normal – 56%, low – 19%, high – 25%).

Table 2 and Figs. 2, 3, and 4 present the results of EMG analysis of the muscles studied in the groups CG, DG and UDG. There were no significant differences in the magnitude or in the time of peak activation of the TA (Fig. 2).

The first MANCOVA compared the peak values of each linear envelope among groups and it showed that the co-variable total contact time had no effect on these variables (Wilks' Lambda > 0.05) and it also showed that there were no differences among groups ($F(6, 78) = 0.67; P > 0.05$).

The second MANCOVA compared the time of peak muscle activity occurrence of the three muscles among

groups and it showed that the co-variable total contact time had no effect on these variables (Wilks' Lambda > 0.05) and that there were differences among groups ($F(6, 78) = 2.27, P = 0.04$). In order to verify which of the variables were different considering only the neuropathy as an interfering factor, ANCOVAs were performed. According to the ANCOVA, the LG time of peak occurrence of the ulcerated diabetic neuropathic subjects was delayed when compared to the control subjects (4.5% of delay in stance phase) and to the diabetics without any history of foot ulceration (5.2% of delay in stance phase) ($P = 0.06, F(2, 41) = 3.06$) as it can be seen in Fig. 3. According to the next ANCOVA, the VL time of peak activation of the ulcerated diabetic neuropathic subjects was also delayed (Fig. 4) when compared to the control subjects (4% of delay in stance phase) and to the diabetics without any history of foot ulceration (3% of delay in stance phase)

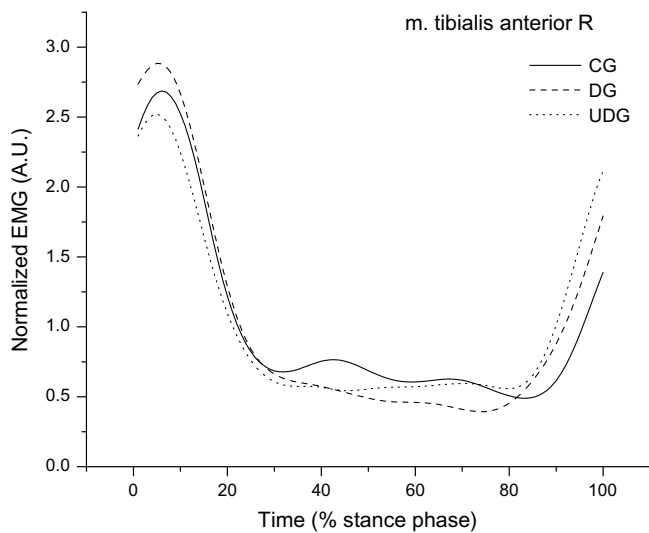


Fig. 2. Mean linear envelopes of the right tibialis anterior (TA), normalized by the mean, of the control (CG), diabetic (DG) and ulcerated diabetic (UDG) groups.

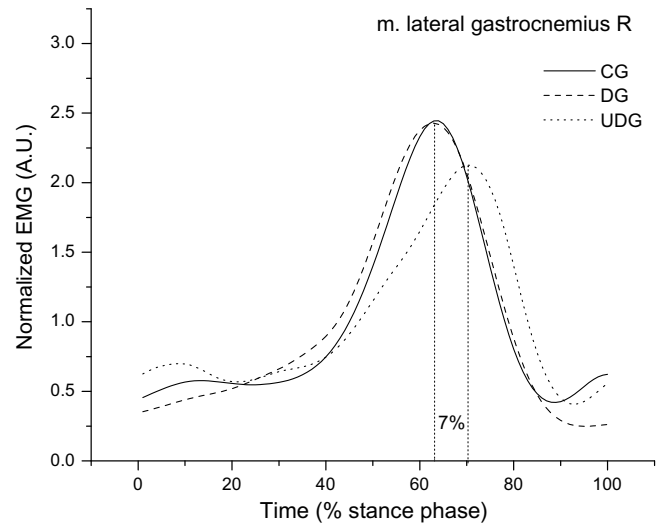


Fig. 3. Mean of the linear envelopes of the right lateral gastrocnemius muscle (LG) normalized according to the mean of the control (CG), diabetic (DG) and ulcerated diabetic (UDG) groups. Note the 7% of delay of UDG.

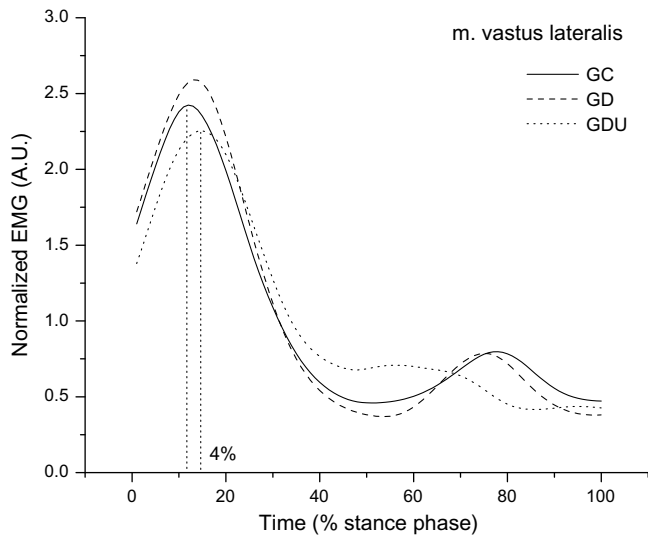


Fig. 4. Mean of the linear envelopes of the right vastus lateralis muscle (VL) normalized according to the mean of the control (CG), diabetic (DG) and ulcerated diabetic (UDG) groups. Note there is a delay of 4% of UDG in comparison to CG.

($P = 0.02$, $F(2,41) = 4.57$). There were no differences in the co-activation index among groups, according to the ANCOVA test ($P = 0.14$, $F(2,41) = 1.99$). Analyzing the coefficient of variation (CV) of the linear envelope curve, we observed lower intra-subject variability among trials (3) comparing to Winter's CV values (Winter, 1991). For the TA, the CV for CG, DG and UDG were, respectively, 15.2%, 25.2% and 21.3%. For the GL, the CV were 16.8%, 21.1% and 22.0%. For the VL, the CV were 16.6%, 18.3% and 20.7%.

Table 2 and Fig. 5 present the results of the GRF for all groups. The MANCOVA used to compare groups considering the vertical GRF showed that there is an effect of the total contact time on these variables (Wilks' Lambda < 0.01) and that they were similar among groups ($F(6,48) = 1.94$, $P > 0.05$). In spite of that, the following ANCOVAs showed that only the second GRF vertical peak was different among groups excluding the effect of the co-variable ($P = 0.01$, $F(2,41) = 6.03$). Post hoc tests showed that both diabetic groups present a lower second peak when compared to the control group [DG ($P = 0.003$) and UDG ($P = 0.01$)].

4. Discussion

Although there were no differences regarding the length of time since diabetes diagnosis and in the MNSI score, in agreement with Mueller et al. (1994) and Yavuzer et al. (2006), diabetic groups showed a decreased tactile plantar sensitivity with a larger number of plantar areas with sensory deficits. The sensitivity data confirmed that the history of ulceration indicates a more developed state of neuropathy in diabetic neuropathic subjects who have already presented a history of foot ulceration. A symmetry in neuropathy impairment was observed, as there was no

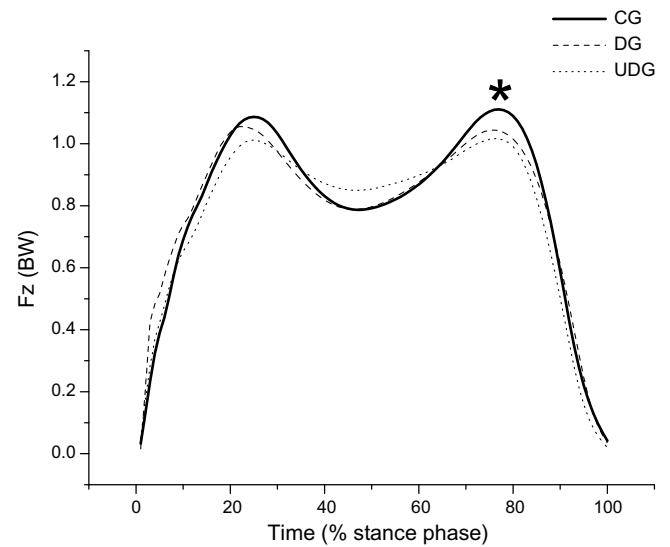


Fig. 5. Mean of GRF curves normalized according to each subject's body weight of the control (CG), diabetic (DG) and ulcerated diabetic (UDG) groups. * means the statistically different variable ($P < 0.05$) between the CG and UDG independently of the total contact time.

difference in the median number of areas with tactile insensitivity between the left and right feet in both diabetic groups. Despite the findings of symmetry, the association of autonomic neuropathy complications may itself have led to plantar ulceration in these subjects.

As for the electromyographical activity, there were no differences in the peak activation of any muscle, but the time of peak occurrence of the LG and VL were delayed in the UDG compared to the other two groups.

The motor system will generate motor responses according to the sensitivity inputs and mechanical loads received by the foot in order to attenuate load at the initial contact of the gait. Considering that, the diabetic subjects studied have a lack of sensitivity in their feet, especially UDG subjects, we can speculate that the output of muscular and joint controls responsible for load attenuation could be altered in these patients. Therefore, we expected a somewhat delayed muscular activation pattern during gait, especially at the time of initial contact, and mainly with respect to the muscles related to shock attenuation: quadriceps femoris and tibialis anterior. Confirming part of our hypothesis, a delay was observed in vastus lateralis activation at the beginning of the stance phase, and a delay of lateral gastrocnemius activation during push-off phase, and this was worse in subjects who had a history of plantar ulceration (UDG).

The delay of the vastus lateralis activation is in agreement with Sacco and Amadio (2003), who found an important postponement and decrease of the VL activation during treadmill gait in diabetic neuropathic subjects. The knee extensor muscles decelerate knee flexion during load reception, working as a shock absorber by transferring part of the impact to the thigh muscle mass. Therefore, a delayed activation of this muscle may be indicative of a

deficiency in the mechanism of shock absorption during the initial heel contact phase, when the lower limb receives the load. This same pattern was found in another study of diabetic neuropathic gait, showing higher minimum vertical forces of the ground reaction force during the stance phase (Sacco and Amadio, 2000). The delayed VL activation may predispose the subjects to heel ulceration, and also lead to joint structure overload of the lower limbs, that may cause biomechanical compensations in the gait of these subjects, such as a slower speed or instability, which are changes that have already been described in the literature (Giacomozzi et al., 2002).

Although there was a VL delay at the beginning of the stance phase, changes on the first GRF peak were not observed. When compared to other locomotor tasks, the GRF during gait (such as walking on inclined walkways or to go up and down stairs), can be considered a biomechanical variable with low impact, because its peak is around 1.1 times the body weight (Winter, 1991). Therefore, the changes in VL activity are not yet causing many effects on GRF during gait. We studied a discrete EMG variable and the study of EMG temporal series during gait might reveal more relationships between these two biomechanics parameters: EMG and GRF.

The delayed activity of the lateral gastrocnemius in the UDG may be indicative of propulsion inefficiency in gait motion of the diabetic neuropathic subjects. This fact is confirmed by the lower second GRF found among the diabetics who have already suffered foot ulceration. Lower ankle extensor moments in neuropathic subjects have already been described in the literature (Mueller et al., 1994; Katoulis et al., 1997; Kwon et al., 2003) and it may be due to changes in ankle extensor activity, which may be represented by the LG delayed peak activity during push-off phase found in the present study.

The propulsion inefficiency could also lead to a longer contact time between foot and ground, which increases the exposure time of the plantar surface to loads. This higher exposure could be a predisposing factor for foot ulceration.

There were no differences in the tibialis anterior time of peak activation or magnitude, among groups. This finding contradicts the results of Sacco and Amadio (2003) and Abboud et al. (2000) who observed a delayed activation of this muscle in the neuropathic groups studied, as well as lower magnitudes, similar to the findings of Eils et al. (2004). The TA is responsible for the impact reduction over the forefoot during the flat foot phase in gait (Winter, 1991), and a delay in its activation may lead to forefoot overloads. A lower and delayed activation of the TA would be expected (Abboud et al., 2000; Sacco and Amadio, 2003; Eils et al., 2004) because of the lower flexor moment at the moment of heel strike during gait that was observed in other studies (Mueller et al., 1994; Katoulis et al., 1997; Kwon et al., 2003; Eils et al., 2004). But the results of the present work are in accordance to the results from Kwon et al. (2003). These authors did not find any delay in TA

activity at the beginning of stance phase. Perhaps there is no delay in the TA activity during neuropathic gait, because it can be considered a usual locomotor task that has already made adaptations and therefore does not reveal any significant changes in the TA activity.

The qualitative analysis of the linear envelopes also shows no differences in the time of peak occurrence of the TA, but a higher peak activity may be seen in the DG, and a lower activity in the UDG, when compared to the CG. It may be suggested that there was an attempt to decelerate during the flat foot phase in the DG, in order to better control the impact on forefoot that caused a reduction of the plantar pressure. The subjects from the UDG were probably unable to adopt that particular strategy due to the advanced stage of their neuropathy.

Although both diabetic groups had significantly larger BMI, changes in gait biomechanics caused by obesity (Spyropoulos et al., 1991), if present, should not have been significant, as the diabetic groups studied are not included in the obesity level according to BMI classification. The low variability of EMG data is an indicative that the larger BMI did not influence the biomechanical variables analyzed.

It was part of the intention of this work to add new information for clinical prevention of foot ulcers in diabetic subjects. From the results of the present paper, we could assume that the peripheral neuropathy and previous foot ulceration are not causing any changes in TA activity that would lead to forefoot ulceration. However, the disease together with previous foot ulceration and a loss of tactile sensitivity could already be causing changes in motor control, especially in VL and GL activity because of changes in input information from the lower limbs, altering the ground reaction forces transmitted to the foot and lower limbs. Also, the changes in EMG activity are leading to a poor propulsion which could cause changes in gait functionality, increasing the risk of foot ulceration or its recurrence by increasing the contact time and exposure to loads (Shaw et al., 1998).

Perhaps in more demanding locomotor tasks such as walking on inclined walkways, or going up or down stairs, peripheral neuropathy may cause more biomechanical changes. Nonetheless, the findings of the present study are important for a better understanding of the biomechanical changes that affect the gait function of diabetic neuropathic subjects. Once the changes of muscle activity and its consequences in GRF caused by this disease have become clear, it will be possible to intervene with appropriate prevention and rehabilitation programs. In line with the outcomes of this study, it may be useful to focus on muscle activity training during function, in this case, for example, during gait, or even during more demanding tasks such as walking faster, or walking on irregular surface to optimize the output responses to different input information. Work on strengthening the knee extensors and ankle dorsiflexors in an attempt to promote shock absorption training during gait and especially during more difficult locomotor tasks may be another promising investigation. In addition, it is

important to focus on strengthening ankle extensors, in order to improve the propulsion during gait to restore its function. Further studies of other locomotor skills are necessary to confirm such assumptions regarding this matter.

5. Conclusion

The present study has shown that long-term neuropathic deficits, represented by a clinical history of at least one foot ulcer in the last two years, caused a delayed activation of the vastus lateralis and lateral gastrocnemius and a decreased propulsion of vertical ground reaction force during barefoot gait. The EMG time activation and GRF were influenced by the progression and the worsening of plantar sensitivity caused by diabetic neuropathy. Because of the vastus lateralis delay at the moment of initial heel contact, we suggest that the mechanisms of loading and shock attenuation might be weakened at this stage of diabetic neuropathy, even with no changes in the first GRF peak. The lateral gastrocnemius activation delay in ulcerated diabetic subjects can significantly alter the propulsion function during the push off phase of gait, confirmed in the present study by the lower second GRF peak, which may affect gait functionality. These findings are important for the definition of rehabilitation and prevention programs for these patients, but further studies are necessary to clarify the reasons for muscle activity pattern alterations and their relationship to plantar ulceration and diabetic neuropathy itself, especially during more demanding locomotor skills.

Conflict of interest

There is no conflict of interests in terms of financial or personal relationship with other people or organization that could inappropriately influence (bias) their work.

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