

Influence of the diabetic neuropathy on the behavior of electromyographic and sensorial responses in treadmill gait

I.C.N. Sacco^{a,b,*}, A.C. Amadio^b

^a Department of Physical Therapy, Speech and Occupational Therapy, School of Medicine of the University of São Paulo, R. Cipotânia, 51, Butantã, Sao Paulo 05360-000, Brazil

^b Laboratory of Biomechanics, School of Physical Education and Sport of the University of São Paulo, Av. Prof. Mello Moraes, Cidade Universitária, 65, Butantã, Sao Paulo 05508-900, Brazil

Received 25 June 2002; accepted 20 February 2003

Abstract

Objective. We describe and interpret self-cadence treadmill walking by neuropathic diabetic subjects under biomechanical and somatosensorial considerations.

Design. EMG variables during stance phase of neuropathic diabetic subjects were acquired and analyzed. We also evaluated sensorial and motor aspects of the feet and legs.

Methods. The experimental procedures are divided as follows: (a) determination of the sensitive cronaxie and pain tolerance in selected plantar areas, (b) determination and description of temporal aspects of EMG patterns of the vastus lateralis, tibialis anterior and lateral gastrocnemius of both sides during treadmill walking. We analyze and compare the results of the sensitive cronaxie, pain tolerance and the EMG parameters obtained by two experimental groups: diabetic neuropathic ($n = 20$) and non-diabetic control subjects ($n = 20$).

Results. The somatosensorial responses and pain tolerance threshold in the diabetic neuropathic group were significantly higher and considered far from the normal patterns. The EMG responses of the thigh and leg muscles in the diabetic neuropathic group were delayed if compared to the normal recruitment pattern, especially the tibialis anterior and vastus lateralis.

Conclusions. These findings lead us to conclude that probably central and/or peripheral control mechanisms of the gait of neuropathic diabetic patients are altered due to somatosensorial and motor deficits. The mechanism of load reduction during walking was considered inefficient because of the activation delay of the vastus lateralis and tibialis anterior. We have concluded that the peripheral diabetic neuropathy damages not only somatosensorial and motor sources but also intrinsic mechanisms of motor control leading to alterations in the ankle efficiency in gait. This resulting distal inefficiency compromises some of the principal requirements for gait, such as progression and balance.

Relevance

This investigation is based on an innovating thematic approach involving the diabetic peripheral neuropathy. This innovation concerns the use of EMG and an instrumented treadmill in a clinical application to study and interpret the motor control during gait in neuropathic diabetic patients.

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Keywords: Biomechanics; Gait; Peripheral diabetic neuropathy; Electromyography; Treadmill

1. Introduction

Human walking can be considered a result of a harmonic correlation between neural and muscular coordinated action with skeletal function (Katoulis et al., 1997). Important changes in the locomotor pattern will take place should any disturbances in this harmonic relationship occur. These pattern changes can bring neuromuscular and balance consequences to the subject's posture and to his gait.

* Corresponding author. Address: Laboratory of Biomechanics, School of Physical Education and Sport of the University of São Paulo, Av. Prof. Mello Moraes, 65, Cidade Universitária, Butantã, Sao Paulo 05508-900, Brazil.

E-mail address: icnsacco@usp.br (I.C.N. Sacco).

The multicentric study of the diabetes mellitus prevalence in Brazil has estimated that 7.6% of the Brazilian population aged 30–69 years have diabetes. In general there are 33.8 diabetics per 1000 inhabitants.¹ Lately, we have verified a great increase in the concern about health problems caused by diabetes in the population, not only those related to its prevalence and incidence, but also those related to the social and economic repercussions due to premature deaths, disability to work or expenses with treatment and control.

The peripheral diabetic neuropathy is the most insidious chronic complication of diabetes. It usually leads the patients to a progressive loss of their somatosensorial sensitivity, proprioception and muscular function (Pirart, 1979). The disease is initially characterized by a reduction in somesthetic sensitivity due to the sensitive nerve damage (sensory neuropathy) and this sensorial reduction can be tested by several techniques to quantify neurological deficits. Kimura (1989), using a universal pulse generator, has highlighted electrodiagnostic evaluations including the sensitive cronaxie which is not an invasive method and is less expensive than any other traditional nerve conduction study. These electrophysiological tests can predict the actual physiological state of peripheral nerve fibers and has already been reported in the literature (Kimura, 1989; Sacco and Amadio, 2000).

With the progression of the diabetic neuropathy, motor nerves are damaged and dysfunctions and atrophy are the results of the motor component of the neuropathy. The fibular nerve, together with the n. sural and the n. plantar medial nerves, are the nerves which present more abnormalities in electrophysiological tests in neuropathic patients (Dick et al., 1985). Therefore, it can be expected that the fibular muscles—tibialis anterior and gastrocnemius—will also have their functions damaged. It is necessary to emphasize the importance of the sensorial and kinesthetic information (spindles) of the lower extremities, especially of the ankle, which is crucial for a better control of gait and posture (Simoneau et al., 1996).

The sensory and motor diabetic neuropathy modify the amount and the quality of the sensorial information necessary to motor control. Consequently, there is an increase in instabilities during gait and static posture (Richardson et al., 1992), which were previously considered to be due to muscular weakness (Courtemanche et al., 1996).

Considering (i) that the peripheral sensorial information diminishes due to the injured peripheral nerves (sensory neuropathy); (ii) that this loss starts in the lower extremities; and (iii) that the muscle spindles of

these extremities are also damaged in diabetic motor neuropathy; the amount of information that comes from the ankle will be drastically reduced resulting in changes of gait and posture strategies (Van Deursen, 1997). As a consequence of the sensory and motor neuropathy, diabetic subjects will try to compensate for the small ankle activity and sensorial information by raising the muscular activity of the hip (Mueller et al., 1994). It is important to observe that other studies (Van Deursen, 1997; Delbridge et al., 1988; Mueller et al., 1989) have also observed smaller ankle mobility during specific ankle movements as well as in gait in neuropathic diabetic patients.

Mueller et al. (1989) have observed that diabetic neuropathic patients show a diminished dynamic ankle function during gait and probably compensate for it in some way by other body segment. Otherwise the results of this diminished function were smaller gait cadences and velocities and smaller step and stride lengths.

Mueller et al. (1994) have actually verified that gait velocity, step lengths, amplitude of ankle movement, ankle moments of force, power and anteroposterior ground reaction force variables were smaller in diabetic neuropathic subjects. However, the authors have observed bigger hip moments of force and power. They concluded that diabetic neuropathic patients have important foot and ankle deficits causing them to conduct the leg to the front of the body during the swing phase in gait using their hip flexors muscles—hip strategy—instead of their ankle extensors muscles to propel the body.

It is important to determine the relationship between the neuropathy severity and the adoption of these hip and ankle locomotor strategies. There is evidence that shows that the more severe the neuropathy is, the bigger the contribution of the hip strategy in gait. Considering therapeutic aspects, it has been suggested to strengthen the hip and knee flexors to compensate for the inefficiency of the ankle during neuropathic gait (Mueller et al., 1994).

Abboud et al. (2000) studied plantar pressure distribution and electromyographic activity of tibialis anterior, peroneus brevis, peroneus longus and soleus muscles during gait in diabetic neuropathic patients. All the studied muscles presented an important delay in their recruitment, the tibialis anterior in particular. As a consequence, diabetic patients' feet contact the ground in mid-stance earlier than what has been verified for healthy subjects. Furthermore, the forefoot keeps longer contact with the ground during stance phase and presents higher peak pressure in comparison with other plantar areas. The authors discuss that the primary cause of the formation of plantar ulcer in neuropathic subjects may not be the peak pressure itself but the dysfunction of the tibialis anterior that cannot prevent the abrupt contact of the forefoot with the ground during gait.

¹ Taxa de prevalência da diabetes mellitus. Available on Internet URL: <http://datasus.saude.gov.br/cgi/idb98/d09.htm> 1999.

Along with Mueller et al. (1994), Abboud et al. (2000) have identified a longer support phase in neuropathic gait because of a flexor dysfunction of the tibialis anterior during the flat foot phase and because of the typical delay of the recruitment of tibialis anterior, peroneus brevis and longus and soleus.

The importance of the present study lies in our proposal to investigate muscular recruitment patterns of the thigh and leg muscles during gait in diabetic subjects in order to identify motor control strategies adopted to compensate for motor and sensorial deficits. Some of those recruitment patterns would probably be already altered due to the diabetic neuropathy. Further intentions are to use these biomechanical results to interfere effectively in therapeutic and prophylactic conducts in these subjects, as well as to discuss old neuromuscular rehabilitation protocols.

The present investigation aims at studying sensorial deficits in neuropathic patients using the sensitive cronaxie and the pain tolerance threshold. We have described the gait in neuropathic diabetic patients using electromyographic responses. We have studied electromyographic responses of the thigh and calf muscles during treadmill gait. We have compared the results obtained in the investigation of neuropathic patients to the results obtained in the investigation of a non-diabetic group. Within these gait characteristic descriptions, we speculate about the influence of the diabetic peripheral neuropathy in gait and possible dynamic mechanisms developed to compensate for sensorial and motor deficits.

2. Methods

2.1. Subjects

Thirty-six voluntary adults of both sexes from the University Hospital of the University of Sao Paulo were divided by the University Hospital's medical staff into two groups: a diabetic neuropathic group (DG) with neuropathy confirmed by symptomatology and clinical investigation by the hospital team ($n = 16$); and a non-diabetic control group (CG) ($n = 20$). The diabetic neuropathic subjects had approximately 11.9 years of diabetes diagnosis. The control group was selected in order to match the diabetic group characteristics, such as: age, mass and sex. The exclusion criterion for both groups were: age over 65, plantar ulcers at the moment of the evaluation, vision impairment, use of walking-stick, peripheral vascular disease, vestibulopathy history, or any neurological, muscular or rheumatic diseases out of the diabetes etiology, history of abusive alcohol intake, partial or total amputation (Katoulis et al., 1997; Courtemanche et al., 1996; Mueller et al., 1994; Abboud et al., 2000; Bergin et al., 1995).

2.2. Experimental procedures

The Ethical Committee of the University Hospital of the University of Sao Paulo approved the following experimental procedure. The experimental protocol took approximately 1 h and it was divided into two stages: (a) electrodiagnostic evaluation: determination of the sensitive cronaxie, pain tolerance threshold and motor cronaxie; (b) gait analysis in treadmill. The procedures were fully explained to the subjects and their written consent was obtained before the beginning of the study.

A preliminary investigation was undertaken to verify clinical aspects of diabetes and neuropathy, diary habits of alcohol intake, foot care and presence of callus, history of ulceration and other relevant aspects. A questionnaire based on Redmond et al. (1992) was used and validated for the present aim. Besides, a clinical examination and investigation was accomplished by the hospital team using clinical criteria based on their daily practice to better characterize and diagnose the diabetic neuropathy. The hospital team uses validated instruments and questionnaires to diagnose their diabetic patients and investigate associated symptoms, associated diabetes chronic complications, history of ulceration, somatosensorial responses by semmes-weinstein monofilaments, and history of blood exams. We use the Redmond et al. (1992) questionnaire and the hospital clinical examination described above to evaluate the diabetic neuropathy.

In the first stage, sensitive cronaxie and pain tolerance threshold were determined in plantar surface, and motor cronaxie was determined over the motor points of the selected muscles (Kimura, 1989; Sacco and Amadio, 2000). Subjects were asked to lie down on a clinical table in prone position with their feet rested on a cushion. Using a universal pulse generator, Omni Pulsi-901 (Quark, Piracicaba, Brazil), we applied electrical pulses with twice the rheobase intensity in order to find the minimum pulse duration that the subjects could perceive in selected plantar areas—sensitive cronaxie. After that, we applied monopolar electrical pulses with 1000 milliseconds (ms) of duration and progressive intensity raise until the pain tolerance in the subjects had settled in the selected areas. We selected five areas to investigate sensitive cronaxie and pain tolerance following anatomic dissection studies of the plantar superficial innervation. The areas were: hallux; medial forefoot, concerning 1st and 2nd metatarsal heads (medial plantar n.); lateral forefoot, concerning 3rd, 4th and 5th metatarsal heads (lat. plantar n.); middle-foot (principal ramification of lat. plantar n.) and heel (calcaneous n.).

With the subjects still lying down, we evaluate the motor cronaxie (Kimura, 1989). We applied electrical pulses with twice the rheobase intensity in order to find the minimum pulse duration that could produce a slight contraction on motor points of the vastus lateralis, tibialis anterior and lateral gastrocnemius.

In the second stage, electromyographic activity (EMG) was acquired during walking on a treadmill. Subjects were requested to walk initially over a walkway of 20 m at a self-selected cadence in order to determine the velocity corresponding to the self-selected cadence to be imposed in the treadmill. After that, the subjects were requested to walk over an instrumented treadmill *Gaitway* Type 9810S1x (Kistler, Winterthur, Switzerland) and the habituation process was done for 10 min. After the habituation process, subjects were requested to walk three times over the *Gaitway* treadmill at the selected velocity for 12 s. They executed approximately 10 steps per trial of each right and left side resulting in 30 steps approximately for each side.

Each subject wore the same sample of a standard lightweight footwear (60–70, SD 6.5 g) throughout. The *Gaitway* treadmill provides a static calibration of the force plates. Each subject was asked to stand over one force plate to register his body weight.

The electrical activity of the right and left lateral gastrocnemius, tibialis anterior and vastus lateralis were accessed using the Bagnoli 8 EMG System (Delsys, Boston, USA). The surface electrodes were placed over the motor points of the selected muscles.

The electromyographic variables were collected simultaneously and synchronically with the ground reaction force, and were sampled at 1000 Hz for periods of 12 s. The studied variables of the right and left vastus lateralis were: (1) first activation peak, (2) second activation beginning, (3) second activation peak. The electromyographic variables of the right and left tibialis anterior were: (4) first activation peak, (5) second activation beginning, (6) second activation peak. The electromyographic variables of the right and left lateral gastrocnemius were: (7) activation beginning, (8) activation peak and (9) end of activation (Fig. 1). Considering that the EMG was simultaneously and synchronically acquired, its data could be interpreted in the same time base as the ground reaction force, which defined the stance phase. Because of that, these EMG variables were determined manually for each step within each trial per subject, and then they were represented by their means per group.

All studied variables (electrophysiological and electromyographical) were described for both sides separately (right and left) in order to investigate asymmetries in the subjects and describe the behavior of each limb during gait.

2.3. Numerical and statistical analysis

We represented the EMG activity through linear envelopes (Winter, 1991; Arsenault et al., 1986). The linear envelopes were obtained after a few steps: off-set remove from the raw EMG, full wave rectification, low-pass filtering (butterworth de 4th order 5 Hz), normalization

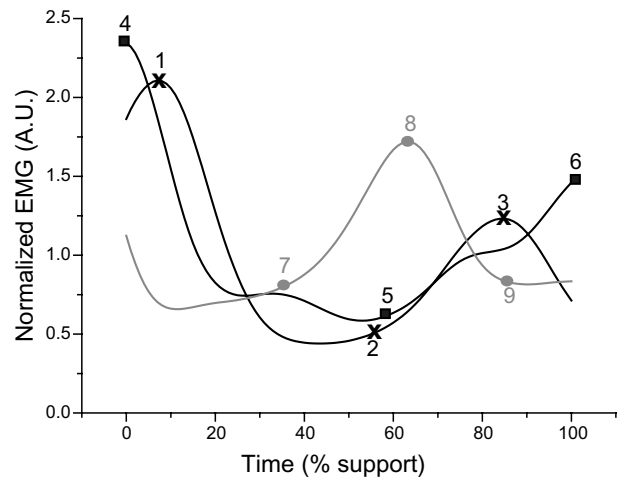


Fig. 1. Linear envelopes of vastus lateralis, tibialis anterior and lateral gastrocnemius representing the studied variables. For the vastus lateralis: (1) first activation peak, (2) second activation beginning, (3) second activation peak. For the tibialis anterior: (4) first activation peak, (5) second activation beginning, (6) second activation peak. For the lateral gastrocnemius were: (7) activation beginning, (8) activation peak and (9) end of activation.

of the data amplitude by its mean, and time base normalization by the support time which was determined using the ground reaction force curve as a reference. After that, we determined manually nine EMG variables (Fig. 1) for each step within each trial per subject (approximately 30 values for each variables per subject) and represented them by their means and standard deviations of the whole DG and CG groups. We also calculated the coefficient of variation (CV) as the mean standard deviation divided by the range of the mean data values, multiplied by 100. Descriptive statistics of the electrophysiological variables were also expressed in means and standard deviations.

Inter-groups comparisons of each anthropometric, electrophysiological and electromyographic variables were made through the use of parametric test (*t* test) and non-parametric test (Mann–Whitney test), considering the results of the tests of normality of each variable. Age, weight, height, electrophysiological variables and some of the EMG variables were compared between groups using the *t* test; and for the rest of the EMG variables, we use the Mann–Whitney test. We considered the differences as statistically significant for $P \leq 0.05$. Statistics was done in Excel (*Microsoft*), Primer v. 1.0 (*McGraw Hill*) and Statistica v.5.1 (*Statsoft Inc.*). Maths was done in Matlab v.5.3 (*Mathworks*) and Origin v.5.0 (*Microcal Software*).

3. Results

The experimental groups' demographic data is described in Table 1. Groups were significantly different in

Table 1
Anthropometric characteristics (mean (SD)), age, and sex of the control group (CG) and diabetic group (DG)

	<i>n</i>	Age (yr.)	Weight (kg)	Height (cm)	Male (%)	Female (%)	Diabetes diagnostic	Last glicaeamia (mg/dl)
CG	20	40.1 (10.4)*	66.1 (10.9)	167.3 (7.6)	60	40		
DG	16	51.5 (11.2)*	70.6 (14.2)	165.6 (10.3)	50	50	11.9 (10.4)	200.4 (62.4)

*Means statistically different.

age ($P = 0.0033$), but not different in weight ($P = 0.2871$) and height ($P = 0.5577$) (Table 1).

Eighty-seven percent of the DG subjects were type 2 diabetics. According to Table 1, glicaeamia was higher than what is expected for a good diabetes control (by 140 mg/dl). Six patients (37.5%) had already had a history of plantar ulceration due to the diabetic neuropathy and two of them (13.5%) had an ulcer on the fifth toe at the moment of evaluation, which, probably did not interfere in the process of gait, as we could visually certify. Typical neuropathic symptoms highly discussed in the literature (Thomas, 1991) such as burning, pain, distal numbness, tingling, pricking, boots feeling were also present in 50% of the subjects, especially during night rest. All subjects in the DG were diagnosed as neuropathic diabetic patients according to the clinical criteria of the hospital team based on their daily practice using validated instruments and questionnaires in order to investigate associated symptoms, associated diabetes chronic complications, history of ulceration, somato-sensorial responses by semmes-weinstein monofilaments test, and history of blood exams.

3.1. Plantar and motor sensitivity results

Table 2 shows mean data of the sensitive cronaxie and pain tolerance for both groups. The DG patients showed higher but not significant cronaxie responses in

all plantar areas, except for the right medial and lateral forefoot, and significantly higher responses for the left medial forefoot corresponding to the plantar medial nerve ($P = 0.007$), and left lateral forefoot corresponding to the plantar lateral nerve ($P = 0.020$). The sensitive cronaxie and pain tolerance of the control subjects were considered normal responses (between 0 and approximately 0.30 ms) (Sacco, 1997).

The DG patients showed significantly higher pain tolerance thresholds than the CG subjects in most of the areas (Table 2). The differences were statistically significant between groups for the right heel ($P = 0.010$), right middle-foot ($P = 0.0177$), right medial forefoot ($P = 0.0432$), right hallux ($P = 0.0269$) and left hallux ($P = 0.0082$). The value for the left heel of the neuropathic group was also high considering normal reference values (Sacco, 1997), but was not significantly different.

The motor cronaxie for both experimental groups was normal for all studied muscles, considering reference normal values—by 0.50 ms (Ervilha and Araujo, 1997) and there were no significant differences between groups.

3.2. EMG results

Table 3 and Fig. 2–4 present the results of the temporal pattern of activation of the studied muscles in both groups.

Table 2
Mean values (SD) of sensitive cronaxie (ms) and pain tolerance threshold (mA) in the 5 plantar areas for both experimental groups: control (CG) and diabetic (DG)

Plantar areas	Foot	Sensitive cronaxie (ms)			Pain tolerance (mA)		
		CG	DG	<i>P</i>	CG	DG	<i>P</i>
Heel	L	0.25 (0.07)	0.38 (0.37)	0.1812	12.73 (9.27)	17.72 (9.36)	0.0885
	R	0.32 (0.15)	0.64 (0.56)	0.0672	9.75 (5.93)	19.16 (9.06)	0.0010*
Middle-foot	L	0.28 (0.11)	0.32 (0.14)	0.3477	8.43 (5.81)	10.41 (5.96)	0.0582
	R	0.30 (0.14)	0.39 (0.25)	0.1613	8.83 (5.66)	10.97 (5.39)	0.0177*
Medial forefoot	L	0.23 (0.08)	0.85 (1.39)	0.0065*	8.63 (4.14)	9.69 (5.89)	0.5994
	R	0.32 (0.35)	0.30 (0.11)	0.1661	7.50 (3.01)	10.16 (4.85)	0.0432*
Lateral forefoot	L	0.24 (0.09)	0.37 (0.23)	0.0201*	8.48 (5.33)	8.81 (6.29)	0.6558
	R	0.29 (0.10)	0.28 (0.10)	0.5666	8.00 (4.14)	9.75 (3.94)	0.0582
Hallux	L	0.23 (0.08)	0.32 (0.33)	0.4640	4.73 (1.47)	6.69 (2.68)	0.0082*
	R	0.21 (0.06)	0.28 (0.16)	0.1265	4.75 (1.56)	6.38 (2.31)	0.0269*

*Means statistically different.

Table 3

Temporal pattern of muscle activation (mean (SD)): right and left vastus lateralis, tibialis anterior and lateral gastrocnemius

Muscles		Variables	CG (% support)	CV (%)	DG (% support)	CV (%)
Vastus lateralis	R	1st activation peak (1)	7.40 (3.30)*	44.58	10.27 (4.27)*	41.56
		2nd activation beginning (2)	59.13 (7.38)	12.47	59.31 (8.29)	13.98
		2nd activation peak (3)	85.47 (4.81)	5.63	86.85 (4.67)	5.38
Vastus lateralis	L	1st activation peak	8.10 (4.27)	52.67	8.40 (4.00)	47.58
		2nd activation beginning	57.79 (7.38)	12.77	58.93 (5.66)	9.61
		2nd activation peak	87.00 (94.88)	5.61	85.79 (3.58)	4.17
Tibialis anterior	R	1st activation peak (4)	1.35 (2.43)**	180.29	8.20 (10.61)**	129.41
		2nd activation beginning (5)	62.68 (11.98)	19.11	59.07 (14.17)	24.00
		2nd activation peak (6)	85.00 (12.01)	14.13	85.47 (11.96)	14.00
Tibialis anterior	L	1st activation peak	3.00 (4.95)***	165.08	9.73 (10.38)***	106.66
		2nd activation beginning	63.42 (11.38)	17.94	61.93 (9.62)	15.53
		2nd activation peak	87.42 (12.24)	14.00	86.20 (9.97)	11.57
Lateral gastrocnemius	R	Activation beginning (7)	24.40 (14.12)	57.86	28.13 (14.92)	53.04
		Activation peak (8)	64.30 (6.59)	10.26	67.00 (12.43)	18.55
		End of activation (9)	86.41 (6.46)	7.48	88.33 (9.06)	10.26
Lateral gastrocnemius	L	Activation beginning	26.30 (13.66)	51.93	18.73 (12.66)	67.60
		Activation peak	64.25 (8.45)	13.15	61.53 (8.45)	13.73
		End of activation	86.00 (4.68)	5.44	84.57 (7.74)	9.16

Differences between CG and DG **t* test $P = 0.0317$; **Mann–Whitney U test $P = 0.0423$; ***Mann–Whitney U test $P = 0.0215$ (*, ** and *** means statistically different).

The first activation peak of the right vastus lateralis of the diabetic subjects was significantly delayed (10.3% support time) when compared to the control subjects (7.4% support time) ($P = 0.0317$). The first activation peak of the right tibialis anterior of the diabetic subjects was significantly delayed (8.2% support time) when compared to the control subjects (1.4% support time) ($P = 0.0423$). The first activation peak of the left tibialis anterior of the diabetic group was significantly delayed (9.7% support time) when compared to the control group (3% support time) ($P = 0.0215$).

4. Discussion

Although there was a difference in age between groups, we observed within groups a high variation in age of the subjects and also the same range of age (minimum and maximum): minimum mean age was 25.9 for the CG and 25.5 for the DG; and maximum age in CG was 66.5 and 68.6 in DG. But, we could not neglect the potential influence of this factor on the results of our study.

According to the information given by the diabetic subjects, their glycaemia was higher (approximately 200 mg/dl) than expected for controlled diabetic patients. Scarlet and Blais (1989), Thomas (1991), and Cattaline and Cancian (1994) discussed that glycaemic taxes that are kept high for a while can cause vascular and neurological injuries in diabetic patients or can worsen pathological conditions already installed. Thus, the high

glycaemic taxes observed in the diabetic subjects, the frequent symptoms revealed by the questionnaire, and the clinical evidence of the diabetic neuropathy investigated by the hospital team could indicate a more severe picture of the diabetes and of the neuropathy.

The most injured plantar areas in the diabetic subjects are related to the plantar lateral and medial nerves, and sural nerve, which are the first nerves to be degenerated due to the peripheral diabetic neuropathy (Dick et al., 1985; Richardson et al., 1992). The sensitive cronaxie responses and the pain tolerance responses were different in the diabetic subjects considering the side and the plantar areas (Table 2). This fact can be attributed to the differences in the perception mechanisms of tact and pain sensations. Their receptors, afferent fibers, and paths to central system are different (Crutchfield and Barnes, 1984; Kandel et al., 2000).

Besides these observed differences in sensibility responses, the diabetic subjects presented higher values in the heel for both sensitivity modalities, although some of them were not significantly different. This plantar area is the first to touch the floor during gait and plays an extremely important role in supplying the nervous system with pressure and proprioception information. The motor system will generate motor responses according to the mechanical loads received by the foot in order to attenuate the load in this initial contact in gait. Considering that the studied diabetic subjects have less sensibility in the foot, we can conclude that the muscular and joint controls responsible for the load attenuation are altered in these patients. As a consequence, we

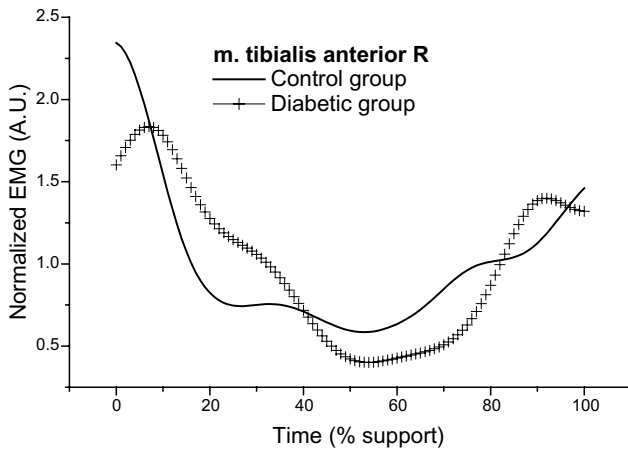


Fig. 2. Mean linear envelopes of the right tibialis anterior, normalized by the mean, for control ($n = 20$) and diabetic group ($n = 16$) during treadmill gait.

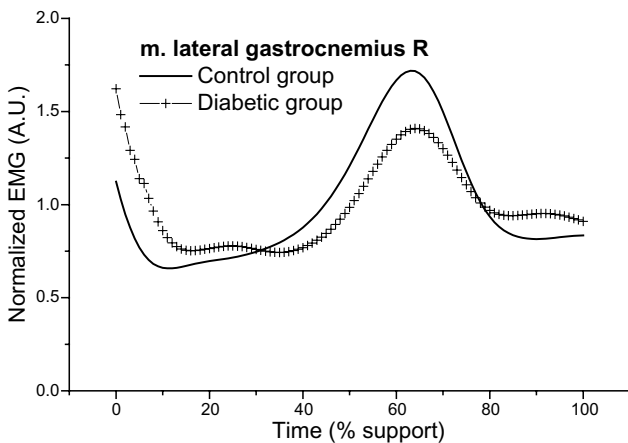


Fig. 3. Mean linear envelopes of the right lateral gastrocnemius, normalized by the mean, for control ($n = 20$) and diabetic group ($n = 16$) during treadmill gait.

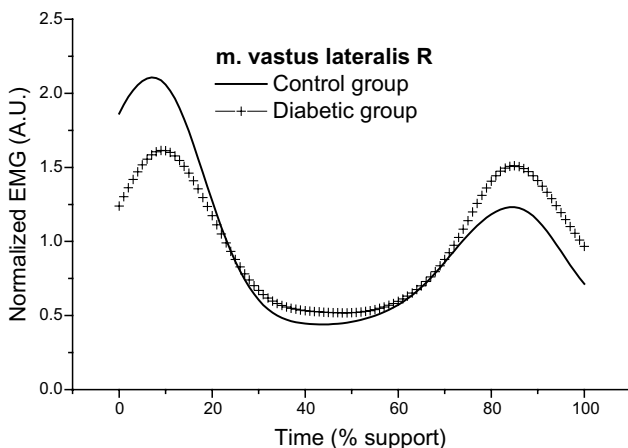


Fig. 4. Mean linear envelopes of the right vastus lateralis, normalized by the mean, for control ($n = 20$) and diabetic group ($n = 16$) during treadmill gait.

expected a muscular activation pattern somewhat delayed during gait, especially in the initial contact, and mainly with respect to muscles related to the shock attenuation: quadriceps femoral and tibialis anterior.

The values of the pain tolerance and sensitive craniocaxial of the medial forefoot and hallux of the diabetic patients were higher than the values of the control subjects. Considering that these areas are related to the propulsion phase of gait, if there is any alteration in the sensitivity functions of these areas, we should expect that the motor responses generated to propel the body in the final phase of gait would fail, in particular those of the muscles related to this phase of gait: *m. gastrocnemius*. But it is important to note for future studies that the activation of the lateral gastrocnemius presented a tendency to delay in the diabetic group, although this delay was not statistically significant in the present study.

The peak activation of the right and left tibialis anterior delayed significantly in the neuropathic subjects. Abboud et al. (2000) found the same results in their study. According to Richardson et al. (1992), the first nerve to show electrophysiological alterations due to diabetic motor neuropathy is the fibular nerve. This degeneration brings motor and sensorial consequences to the subjects. The tibialis anterior, which is innervated by the fibular nerve, is thus one of the first muscles to show alteration in the activation pattern, as was observed in the present study. Considering the tibialis kinesiological function, we could expect as a consequence that the ankle would not flex satisfactorily during the toe clearance and the foot would fall down uncontrolled during the initial contact phase.

The tibialis anterior has a fundamental role in the initial contact phase of gait. Its function is to reduce the initial shock of the forefoot during the flat foot phase (Winter, 1991). If a delay in the tibialis anterior activation occurs, the shock attenuation mechanism will fail, and higher loads on the forefoot will be generated in the neuropathic subjects. There has been much discussion about the possibility of relating this alteration in the load reduction mechanism to ulcer formation in the forefoot of diabetic neuropathic subjects (Abboud et al., 2000; Bevans, 1992). Bevans (1992) justified the higher prevalence of ulcers in the forefoot as a consequence of the higher plantar pressures generated by the foot drop in the initial phase of gait due to the delay in the tibialis anterior activation. Other authors have discussed that the forefoot ulcers prevalence can be attributed to the reduction of the foot and ankle mobility (Mueller et al., 1989; Delbridge et al., 1988; Van Deursen, 1997).

Considering the impairment of the distal extremities observed in diabetic neuropathic subjects, Mueller et al. (1994) proposed that these patients change the ankle strategy to the hip strategy during gait in order to compensate for the smaller ankle moments of force due

to the peripheral degeneration. In another study, it has been shown that the mechanical power of the ankle diminished during gait in ill elderly people while there was an increase in the hip (McGibbon et al., 2001). In the present study, the EMG activity of the tibialis anterior was smaller and delayed compared to the control group and this could represent an alteration in the contribution of the ankle during gait.

Alterations in the tibialis anterior activation during gait in neuropathic subjects were observed bilaterally, even though the sensorial impairment was unilateral in some cases. This could be explained if we considered a central mechanism of sensorial deficits compensation which is bilateral, even if the sensitivity impairment is unilateral. This way the system can anticipate any unbalanced gait response generated by the asymmetries in the diabetic locomotor apparatus. These bilateral compensations in neuropathic subjects have already been cited by Dingwell et al. (2000).

The same behavior observed for the tibialis anterior was observed for the right lateral gastrocnemius of the diabetic neuropathic subjects. The beginning of its activation and its activity peak were delayed when compared to the control subjects, even though this delay has not been statistically significant. The right and left vastus lateralis of the diabetic group has its first activation peak delayed when compared to the control group. We also observed smaller activation peaks of the vastus lateralis in diabetic group. These findings were also observed by Abboud et al. (2000). Deficits in sensorial information in the initial contact phase of gait can put off the activation of the vastus lateralis which plays an important role in the mechanical load attenuation during weightbearing. Mechanical loads in joints, ligaments and muscles during gait in these subjects maybe higher than normally expected and could progressively damage tissues.

Considering that both vastus lateralis and tibialis anterior play an important role in reducing mechanical loads during specific phases of gait, alterations in the activation patterns of those muscles could imply a deficit in the shock reduction mechanisms in ankle and knee joints during the support phase.

The EMG coefficient of variation was lower for both groups in comparison to the reported data for ground walking (Winter, 1991). Dingwell et al. (1999) have discussed that restrictive environments like a treadmill impose certain conditions for the subjects during walking and result in a more reproducible and less variable gait pattern, as well as observed in the present study. Although CV's were in general lower during walking, tibialis anterior presented higher values in both groups. We could speculate that this may be a unique characteristic of its activation independently of the group characteristics, as discussed by Lobo da Costa (1995).

5. Conclusion

The present study has shown that long-term sensorial and motor deficits have altered muscle activation patterns during neuropathic gait in treadmill. Besides the pathological responses of sensitive craxie and pain tolerance, diabetic neuropathic patients presented important delays in the activation of the tibialis anterior bilaterally and vastus lateralis during gait in treadmill. Although sensorial impairments were unilateral in most cases, when muscle responses were altered in gait, they were bilaterally and this fact could indicate a probable alteration in the gait control mechanism. Because of these delays, we also interpreted that mechanisms of mechanical loads and shocks attenuation could be collapsed.

The activation delay of the tibialis anterior in diabetic subjects can significantly alter the ankle function during gait. Authors (Mueller et al., 1994) have demonstrated that diabetic neuropathic subjects compensate for their deficit in distal extremities by changing the ankle strategy to the hip strategy in gait. In the present study, we could observe an abnormal contribution of the ankle muscles in the load reduction and also in the propulsion of the body during gait, but we did not study and consequently could not conclude in the present paper if there is indeed a greater contribution of the hip as suggested by Mueller et al. (1994).

Further clinical studies could be conducted in order to investigate new treatment and rehabilitation strategies to recover distal and specially proximal skeletal function in gait considering the findings presented in this and other related papers.

Acknowledgement

We thank FAPESP for the important financial support for this project (# 98/09992-4).

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