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MUSCLE DYNAMICS DURING GAIT OF DIFFERENT DEGREES OF DIABETIC NEUROPATHY SEVERITY CLASSIFIED BY A FUZZY EXPERT MODEL

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SUMMARY
The aim of this study was to investigate EMG patterns during gait in diabetic individuals with different stages of diabetic sensorimotor polyneuropathy (DSP) severity, classified by a fuzzy system. In this study, 147 subjects were divided into a control group and four diabetic groups: absent, mild, moderate and severe neuropathy, classified by a fuzzy expert model. The EMG activity of the vastus lateralis (VL), tibialis anterior (TA), and gastrocnemius medialis (GM) were measured during gait. Temporal and relative magnitude variables were compared among groups using ANOVA tests. Muscle EMG is altered even before neural involvement, with a delay in VL peak and lower TA relative magnitude, suggesting an impaired ankle joint shock absorption mechanism, with a dynamic compensation at the knee. Increased VL activity in mild and severe neuropathy groups may indicate an aggravation of alterations in this muscle activation. TA onset at terminal stance was anticipated in all diabetic groups and at higher degrees of neuropathy, the GM exhibited a reduction in its activity and a peak delay. Thus, the degree of severity of DSP must be taken into account when analyzing diabetic patients' biomechanical patterns of locomotion. We recommend the use of a fuzzy expert model in the classification of the stages of this disease stages.

INTRODUCTION
Electromyography (EMG) alterations during gait, supposedly caused by DSP, are subtle and still inconsistent, possibly due to difficulties in defining homogeneous experimental groups with a clear definition of disease stages. The use of a fuzzy expert model could be an interesting approach to enable a better distinction among different stages of DSP and to detect when changes in muscle activation during gait start occurring. The purpose of the present study was to investigate the lower limb EMG patterns of patients with different degrees of DSP severity, classified with a fuzzy expert system. Our theses were that the use of fuzzy logic would enable a better distinction among the different stages of the disease and that muscle activation would be altered even before the onset of DSP, with increased effects in the more severe degrees of neuropathy.

METHODS
In this study, 147 adult volunteers of both genders were divided into a control group of non-diabetic subjects (C; n=30) and four diabetic groups, classified by means of a fuzzy expert system: non-neuropathic diabetic group (D; n=43) and diabetic individuals with mild (MiN; n=30), moderate (MoN, n=16), and severe (SN; n=28) neuropathy. The fuzzy expert system, based on Picon et al.[1], used vibratory perception (128Hz tuning fork), tactile sensitivity (10g Semmes-Weinstein monofilament), and symptoms assessment (based on *MNSI questionnaire*) as the system´s inputs, and the combination among them determined the final "neuropathy degree score" by the center of area defuzzification process. This value was sorted into the disease classes with the following division, with x being the score value: (i) $x \leq 2.5$: absent neuropathy; (ii) $2.5 < x < 5.0$: mild neuropathy; (iii) $5.0 \leq x < 8.0$: moderate neuropathy; (iv) $x \geq 8.0$: severe neuropathy.
The EMG activity of VL, TA, and GM were measured during gait, with an EMG system (model 800C; EMG System do Brasil, São José dos Campos, Brazil). A signal amplification factor of 1000 was used. Disposable Ag/AgCl circular electrodes (10 mm diameter) were placed over each muscle with a center-to-center interelectrode distance of 20 mm, following the recommendations of Sacco et al.[2] for placement location. This signal was synchronized to the ground reaction force (model OR61000; AMTI, Watertown, MA) at a sampling rate of 2 kHz (A/D Board DT3002, 12 bits, AMTI), and five trials for each participant were collected. Temporal (time to peak activity of all muscles and onset time – TA at terminal stance and GM) and relative magnitude variables (maximum activity/minimum activity of all muscles and final activity/minimum activity of TA) were compared among groups using ANOVA tests.

RESULTS AND DISCUSSION
The main findings indicate that diabetes mellitus and DSP are related to changes in muscle activity, although the alterations did not follow a distal to proximal order, nor did they progress in the same manner from mild to severe stages. All the diabetic groups, except MoN, had a tendency to delay the peak activity of VL, although only Groups D and SN showed a significant difference (approximately 26%) compared to the other groups (Table 1). This delayed pattern was also described by earlier studies[3], and it may indicate a difficulty in producing proper shock absorption at

early stance, with more participation of the knee in this function. The greater use of this proximal joint seems to be more pronounced in SN and MiN groups, which presented a higher relative magnitude of VL in addition to the delayed peak (Table 1).

The TA relative magnitude was lower for all diabetic groups compared to C, suggesting an impaired function at the heel strike phase when this muscle plays an essential role in controlling forefoot contact with the ground and in attenuating the initial impact. This poor TA function at heel strike can be associated with the previously described delayed VL pattern at this specific gait phase, in both the D and SN groups. The possibility of a compromised shock absorption mechanics was also formerly discussed because of a delayed peak activity of the same muscle[4].

The activation onset of TA at late stance occurred earlier in the diabetic groups, both with and without neuropathy, and it was more evident in Groups D and SN (average delay of 28%) (Table 1). This could be related to the commonly observed slower gait velocity, cadence, and smaller stride length[5]. Nevertheless, the anticipation of TA onset could explain the reported lower extension moments at late stance[6]. The reduced relative magnitude and delayed peak activity of GM (6%) in SN group (Table 1) could also contribute to the aforementioned impaired role of propulsion at late stance. This temporal GM delay was previously described, even with imposed gait velocities[7], and has occurred only in individuals with a history of plantar ulceration[3], an indication of a worse state of DSP, which corresponds with the more severe diabetics in our study.

CONCLUSIONS

The activity levels of lower limb muscles during gait changed at distinct severity degrees of DSP. There is a delay in VL and a reduction in TA relative magnitude that are present even before a neurological involvement, suggesting an impairment in the shock absorption mechanism at the ankle and a higher dependence on the knee´s absorptive function in the weight acceptance phase of gait in the early stages of the disease. With the onset of neuropathy, this proximal compensation continues, with an increase in VL relative magnitude. At late stance, TA onset is anticipated even in the absence of DSP, with an intensification of this pattern in severe neuropathy state, along with peak delay and lower activation of GM in the moderate and severe groups.

DSP severity degree must be taken into account when analyzing the biomechanics of locomotion of diabetic patients, and we recommend the use of a fuzzy system to assess the state of disease, not only for research purposes, but also in the healthcare system.

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Table 1: – EMG temporal and relative magnitude variables (mag) for vastus lateralis (VL), tibialis anterior (TA) and gastrocnemius medialis (GM)

		Control (n=30)	Diabetes (n=43)	Mild Neuropathy (n=30)	Moderate Neuropathy (n=16)	Severe Neuropathy (n=28)	p
VL	**Time to peak (%stance)**	9.7 ± 3.2*	12.1 ± 2.3*§	11.0 ± 3.3	9.7 ± 2.5§	13.5 ± 3.6[a]	< 0.001 [1]
	Relative magnitude	8.3 ± 4.0[b]	11.0 ± 6.5	13.6 ± 10.0*	6.8 ± 3.1[c]	17.1 ± 15.6*	0.014 [2]
TA	**Time to peak (%stance)**	3.7 ± 2.0	4.2 ± 2.4	3.6 ± 2.1	2.2 ± 2.0	3.3 ± 2.6	0.055 [1]
	Relative magnitude	60.9 ± 55.5[d]	21.3 ± 12.8	24,7 ± 15.7	22.7 ± 20.6	33.4 ± 31.0	0.043 [2]
	Onset at push off (%stance)	90.4 ± 6.1†	72.7 ± 10.8*	79.4 ± 12.5*	76.7 ± 10.5	68.8 ± 4.9[a]	< 0.001 [2]
	Relative magnitude at push off	6.0 ± 2.7*	8.1 ± 2.7*	7.1 ± 2.8	7.6 ± 2.0	7.1 ± 2.2	0.014 [1]
GM	**Time to peak (%stance)**	60.0 ± 6.4	60.5 ± 5.1	61.3 ± 3.9	59.3 ± 4.3	64.0 ± 5.6[e]	0.015 [1]
	Relative magnitude	52.8 ± 48.8	33.7 ± 24.0	37.9 ± 24.5	19.2 ± 13.5[f]	16.0 ± 13.4[f]	< 0.001 [2]
	Onset (%stance)	40.4 ± 5.4	37.3 ± 5.4	38.7 ± 8.5	34.8 ± 9.2	38.1 ± 6.1	0.089 [1]

[1] ANOVA; [2] Kruskal-Wallis test; *, § statistically significant difference between groups; † statistically different group; [a] statistically different from control, mild and moderate; [b] statistically different from mild and severe; [c] statistically different from diabetes, mild and severe; [d] statistically different from diabetes, mild and moderate; [e] statistically different from control, diabetes and moderate; [f] statistically different from control, diabetes and mild.