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MULTICHANNEL EMG-BASED ESTIMATION OF FIBER CONDUCTION VELOCITY DURING ISOMETRIC CONTRACTION IN DIABETIC PATIENTS WITH DIFFERENT SEVERITY DEGREES OF NEUROPATHY CLASSIFIED BY A FUZZY EXPERT SYSTEM

[1] Marco K. Butugan, [1] Anice C. Pássaro, [1]Ricky Watari, [1]Cristina D. Sartor and [1] Isabel C. N. Sacco
[1] Universidade de São Paulo, School of Medicine, São Paulo, Brazil
email: krab23@gmail.com , web: www.usp.br/labimph

SUMMARY

This study compares muscle fiber conduction velocities during isometric maximal voluntary contraction in different degrees of diabetic neuropathy classified by a fuzzy expert model. Sixty-seven diabetic patients of both sexes were classified by a fuzzy expert system in four groups according to the neuropathy degree: absent, mild, moderate and severe. Average muscle fiber conduction velocities of gastrocnemius medialis, tibialis anterior, vastus lateralis and biceps femoris were assessed using linear array surface electrodes and one-way ANOVA was used to compare groups. Muscle fiber conduction velocities were significantly increased in the moderate group for the gastrocnemius medialis and decreased in the same group for the vastus lateralis. In the severe group, there was again a significant decrease for the gastrocnemius medialis. These results indicate that different mechanisms predominate in distal and proximal muscles at different degrees of disease severity, where axonal loss and muscle fiber diameter compensatory increase could be responsible for increased muscle fiber conduction velocity distally and proximally reinnervation of previously deinnervated fibers could be responsible for the reduction in the conduction velocity observed.

INTRODUCTION

As diabetic peripheral neuropathy progresses, losses in the neuromuscular system can be observed both in neural tissue, with endplate disruption and axonal retraction, and muscle fibers with contractile capacity impairment and selective fiber type damage[1,2]. These losses lead to changes that can be assessed by the spatial electromyographic potential distribution revealing that indeed fewer motor units are active for the same level of activity in the vastus lateralis muscle [3].

Muscular performance impairment has also been associated with muscle fiber conduction velocity independent of motor nerve conduction velocity changes for both human vastus lateralis muscle and the ankle joint muscles [4,5]. Muscle fiber conduction velocity has also been tested invasively in early diabetic neuropathy showing decrease for the slowest muscle fibers and also on the mean muscle fiber conduction velocity (MFCV) [6].

Noninvasive MFCV estimation can be used to assess the average muscle fiber conduction velocity of the innervated fibers and this data could be used to understand different mechanisms underlying the progression of diabetic neuropathy for different muscles.

For this study, we tested if different diabetic neuropathy degrees classified by a fuzzy expert model have distinct muscle fiber conduction velocities in four lower limb muscles during isometric contractions.

METHODS

In this study, 67 diabetic patients of both sexes (58.6±5.3yrs, 28.0±5.3kg/m^2) were divided into four groups with different neuropathy severity degrees classified by a fuzzy expert system: absent group (n=26), mild neuropathy (MiN; n=20), moderate neuropathy (MoN, n=10), and severe neuropathy (SN; n=10). Patients were assessed by a trained physical therapist, who evaluated the (i) vibratory perception (128Hz tuning fork), (ii) tactile sensitivity (10g Semmes-Weinstein monofilament), and (iii) presence of typical neuropathy symptoms (based on MNSI questionnaire [7]). These three group of variables were used as input in a fuzzy expert system, based on Picon et al.[8]. Glycated Hemoglobin levels were also assessed and compared among groups with a HbA1c monitor (A1CNow SELFCHECK, Bayer LLC Diabetes Care, New York, USA) (table 1).

Before the EMG signal acquisition, the innervation zone position for each muscle was determined by visual inspection of the signal obtained from an 8-channel dry linear array (SA8/10, OT Bioelettronica, Torino, Italy) [9] and 4-channel linear array electrodes with 10 mm interelectrode distance (ELSCH004, OT Bioelettronica, Torino, Italy) were placed between the tendon and the most distal innervation zone after skin cleaning and abrasion. The EMG activity of vastus lateralis(VL), biceps femoris(BF), tibialis anterior (TA), and gastrocnemius medialis (GM) were assessed during three maximum isometric voluntary contractions with a multi-channel surface electromyography acquisition system (EMG-USB2 16 channels, OT Bioelettronica, Torino, Italy) at 2048 Hz and band-pass filtered at 10-800 Hz with a zero-lag 4th order butterworth digital filter. MFCV of each muscle were estimated using the maximum likelihood algorithm proposed by Farina et al.[10] with a gaussian window with 30ms deviation

positioned in the center of the 500ms interval corresponding to the maximum root mean square observed during the maximum isometric voluntary contraction. Between groups comparison of MFCV and HbA1c was performed by one-way ANOVA with significance level set at 0.05.

RESULTS AND DISCUSSION

A significant increase in MFCV was observed for GM in the MoN group following a trend for increase from the absent and mild neuropathy groups. Paradoxically, an opposite pattern was seen for VL where the MFCV decreased significantly in the same individuals (Figure 1).

HbA1c levels where lower in the non-neuropathic group and higher in the MoN and SN groups (table 1) showing that the fuzzy expert system was accurate in discriminating and classifying diabetic patients within neuropathy degrees, since as the disease progresses, the HbA1c control gets worse.

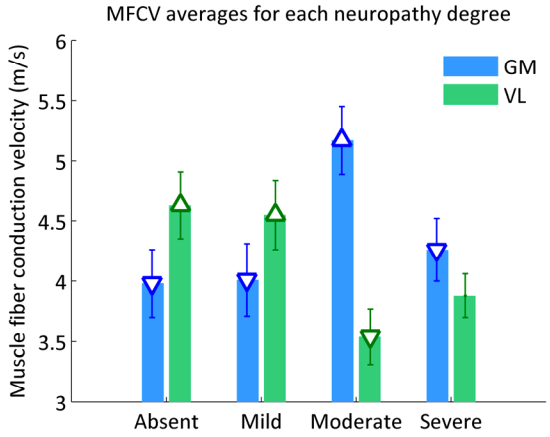

Figure 1: MFCV of gastrocnemius medialis and vastus lateralis muscles during isometric contraction. Significant differences (Neuman Keuls post-hoc $p<0.05$ after one-way ANOVA) are marked with Δ for higher values and ∇ for lower values (comparisons between neuropathy degrees).

These findings suggest that there are different mechanisms acting on the motor neurons innervating distal and proximal muscles on account of axonal length and neuropathy severity.

For the GM, the longer axons are more susceptible to damage and an increase in MFCV can be caused by neurogenic lesions due to denervation and subsequent compensatory increased diameter [6] or the change in fiber unit type ratio with decreased type I fiber proportion [1].

For the VL, muscle fiber slowing can be the result of the activation of muscle fibers with smaller diameters either as a sign of greater conservation of type I oxidative fibers or due to the reinnervation of previously deinnervated atrophic fibers [6, 11].

In the severe neuropathy group, there is a new decrease in MFCV in the GM muscle indicating that the compensatory increase in fiber diameter of the surviving motor units that was maintaining a higher conduction velocity is offset by muscle fiber atrophy [12] along with the increase in severity of neuropathy symptoms.

CONCLUSIONS

MFCV during isometric contractions are different for proximal and distal muscles and for different neuropathy severity degrees indicating that local factors and disease progression influences the degree of reinnervation and muscle fiber quality in different patterns.

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Table 1: HbA1c level and MFCV of tibialis anterior (TA), gastrocnemius medialis (GM), vastus laterais (VL) and biceps femoris (BF) assessed during isometic maximum voluntary contraction.

Group	HbA1c	MFCV TA	MFCV GM	MFCV VL	MFCV BF
Absent	8.02±0.32	4.24±0.28	3.98±0.28	4.63±0.28	5.26±0.37
Mild	8.86±0.35	3.94±0.29	4.01±0.30	4.55±0.29	5.13±0.41
Moderate	9.45±0.30	4.41±0.28	5.17±0.28	3.54±0.23	4.9±0.27
Severe	9.84±0.44	4.94±0.26	4.26±0.26	3.88±0.18	4.74±0.36
p; F*	0.003; 5.097	0.093; 2.202	0.017;3.556	0.006; 4.413	0.730; 0.433

*one-way ANOVA