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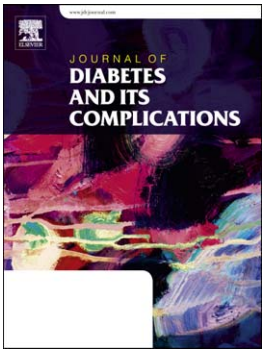

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Assessment of lower leg muscle force distribution during isometric ankle dorsi and plantar flexion in patients with diabetes: a preliminary study

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Abstract:

Aim: The aim of this study was to evaluate the differences in ankle muscle strength using hand-held-dynamometry and to assess difference in the isometric muscle force distribution between the people with diabetes and control participants. Methods: The maximal muscle strength of ankle plantarflexion, dorsiflexion, eversion, inversion, lesser toes flexors and extensors, hallux flexors and extensors was assessed in 20-people with diabetes and 20-healthy participants using hand-helddynamometry.

The maximal isometric ankle plantarflexion and dorsiflexion were imported to OpenSim software to calculate 12-individual muscle (8-plantarflexors and 4-dorsiflexors) forces acting on ankle joint. Results: A significant reduction in ankle strength for all measured actions and significant decrease in muscle force for each of the 12-muscles during dorsi and plantar flexion. Furthermore the ratios of agonist to antagonist muscle force for 6 of the muscles were significantly different between the healthy and people with diabetes. Conclusions: It is likely that the muscles for which the agonist/antagonist muscle force ratio were significantly different for the healthy and the people with diabetes, could be more affected by diabetes.

Keywords: muscle force; diabetic; musculoskeletal model; biomechanics

1. Introduction

Type 2 diabetes (DM2) is accompanied by a wide range of impairments. Previous investigations have shown that DM2 is associated with a loss of mobility (Orr et al. 2006; Lalli et al. 2013) and reduced muscle strength (Andreassen et al. 2006). Several studies have also described impairment of gait (Brach et al. 2008; Raspovic, 2013), foot ulceration (Raspovic, 2013), and increased risk of falling (Lalli et al. 2013) in neuropathic diabetic patients. Furthermore, a reduced walking speed along with a compromised static and dynamic balance have also been observed in older diabetic patients with neuropathy (Lalli et al. 2013)). In addition, Andersen et al. (2004) showed that DM2 is associated with loss of muscle strength around the ankle and knee joint and Mueller et al. (1994) revealed that diabetic neuropathic patients were unable to generate sufficient ankle joint moment, with a consequent reduction in the dynamic function during walking, resulting in a smaller step length and stride, reducing gait speed and cadence.

Whilst, neuropathy has been associated with impaired mobility, loss of muscle strength and decreased HR-QoL, as reviewed elsewhere (Van Schie, 2008), several factors could be responsible for this limited mobility and decreased muscle strength in diabetic patients; such as intrinsic abnormalities in diabetic muscle, impaired capillary recruitment, peripheralarterial disease and diabetic polyneuropathy (Andersen et al., 2004; Van Schie, 2008, Lalli et al. 2013).

Although, most in vivo studies have analysed muscle performance under isokinetic conditions (both active (Hatef et al., 2014) or passive (Hajrasouliha et al., 2005)), a simple, widely used and objective tool in a clinic for measuring muscle strength is hand-held-dynamometer (Abizandaet al. 2012). Hand-held-dynamometers have been shown to be reliable for testing a number of muscle groups including those of the ankle (Wang et al., 2002;

Burns et al. 2005), but this device does not give any information about the individual muscle forces distribution. Since muscle forces cannot be measured invasively (Pandy, 2001), these quantities are determined using indirect methods combining kinematic and kinetics analysis. Muscle force distribution problem within biomechanics deals with the determination of the internal forces acting on the musculoskeletal system using the known resultant inter-segmental forces and moments. The force distribution across human joints is typically represented with an indeterminate set of system equations; this means there are more unknowns than the number of equations that are most often used for calculating the muscle, ligament, and bone forces acting in and around joints. The analysis of muscle forces distribution is currently one of the major issues raised in biomechanics, requiring the use of sophisticated optimization models (Delp, et al., 2007).

There has been a paucity of studiesthat investigatethe individual muscle force distributions in people with diabetes. In light of the lack of such data, the aim of this pilotstudy was to evaluate differences in foot and ankle isometric muscle strength and to assess the difference in individual muscle force distributions between the people with diabetes and healthy controls.

2. Material and Methods

2.1. Participant recruitment and preparation

48 people with diabetes and severe neuropathy with a mean age of 59 ± 8.02 years, height of 1.66 ± 0.1 m and weight of 74.8 ± 7.23 kg participated in the study. Following a statistical analysis (detailed section 2.4.1) a subset of 20 of the 48 diabetic patients with mean age of 59 ± 9.84 years, height of 1.63 ± 0.1 m, weight of 71.6 ± 12.1 kg and average duration of diabetes 14 ± 7.8 years were selected for analysis. The diagnostic criteria for composing

the groups with signs and symptoms of neuropathy were based on the measurement of VPT at the Hallux, first, third or fifth metatarsals. The voltage was slowly increased at the rate of 1 mV/sec and the VPT value was defined as the voltage level that produced a vibration that was sensed by the subject. The mean of the four records was calculated and neuropathy was diagnosed if the average was more than 25mV (Young et al, 1994). . Twenty healthy volunteers with mean age of 60.7 ± 7.5 years, height of 1.64 ± 0.6 m and weight of 73.2 ± 6.12 kg were screened and included in the study. In both groups, the number of men and women were the same - 10 in each. A t-test was performed and showed no significant age differences between the healthy and diabetic group. The ethical approval was sought and granted by the local research ethics committee and all volunteers provided full informed consent.

2.2. Instrumentation and data collection

Isometric muscle strength was measured using a Citec hand-held-dynamometer (CIT Technics, Haren, Netherlands). The manufacturer's data state the device was factory calibrated to a sensitivity of 0.1% and a range of 0–500 N. The hand-held-dynamometer (HHD) measures the peak force produced by a muscle as it contracts while pushing against an object. A recent systematic review of HHD for assessment of muscle strength in the clinical setting found the instrument to be a reliable and valid tool (Stark et al., 2011). Isometric muscle strength was assessed using the 'make test', whereby the examiner held the HHD stationary while the participants actively exerted a maximal force. All tests were performed with the participants in a supine position with hips and knees extended and the lower limb stabilized proximal to the ankle joint as directed by (CIT Technics, Haren, Netherlands). The HHD was positioned against the lateral border of the foot distal to the base of the 5th metatarsal head to measure eversion; to the medial border of the foot, near the base of the 1st

metatarsal head to measure inversion; against the metatarsal heads on the plantar surface of the foot to measure plantarflexion, and on the dorsal aspect of the foot proximal to the metatarsal heads to measure dorsiflexion and over the interphalangeal joint of the hallux for hallux plantarflexion and dorsiflexion. For testing of the lesser digits, the dynamometer was placed on the plantar surface of the digits. Moreover, for testing both the hallux and lesser toe strength, the ankle was passively placed in maximum plantar flexion to prevent co-contraction of the ankle plantar flexor muscles influencing the result.

Each participant performed submaximal test movements for familiarization prior to testing. Testing of each muscle group required a contraction of 3-5 seconds. Three repetitions were obtained for each muscle group, for each leg with a minimum rest period of 10 seconds between each contraction. The average of the three contractions was used for analysis as mean values have been shown to be more reliable than maximal values (Van den Beld et al., 2006). Verbal encouragement was given during each contraction. To assess repeatability ofmeasurements, coefficients of variation (CVs) were calculated, which expresses between-trial variability as a percentage. It has been suggested that CV values of 0.60 and greater indicate poor repeatability, 0.4–0.60 fair repeatability, 0.20 – 0.40 good repeatability and 0.20 and less excellent repeatability (Krysicki et al., 2006). All measured with HHD parameters achieved good and excellent repeatability.

2.3. Musculoskeletal model

A generic musculoskeletal model with 19 degrees-of-freedom and 92 musculo-tendon actuators was used to generate the simulation in OpenSim 2.4 (Stanford, USA) (Delp et al., 2007). The model was dimensioned to represent a subject with a body mass of 72.6 kg. The feet of each subject were scaled to match the anthropometry, which was measured before the experiment. An inverse kinematics problem was solved to calculate the joint angles of the musculoskeletal model that the best reproduce the experimental kinematics of the subject,

what was distributed with OpenSim software. Following this step, individual muscle forces were computed using the computed muscle control (CMC) tool. CMC is an optimization based control technique designed specifically for controlling dynamic models that are actuated by redundant sets of actuators whose force generating properties may be nonlinear and governed by differential equations. The purpose of (CMC) is to compute a set of muscle excitations that will drive a dynamic musculoskeletal model to track a set of desired kinematics in the presence of applied external forces (Thelen et al., 2003). The OpenSim force data file was modified to allow simulations. For each subject plantarflexion force measured with HHD was put as a vertical force applied to toes as a body force and for each subject dorsiflexion force measured with HHD was applied as a vertical force with the same line as plantarflexion force but opposite direction also applied to toes as a body force. While the anterio-posterior and medio-lateral components of the ground reaction force are important during gait, in an isometric contraction we made sure that the measuring head of the dynamometer was held perpendicular to the plantar surface (in plantarflexion) and to the dorsal surface (in dorsiflexion). In this condition only the vertical component of the force causes a moment around the centre of rotation of the joint. Since the lever arm was perpendicular to the line of action of the force, hence the measured force by the dynamometer was the only component that exist during isometric dorsi and plantar flexion. For each person from the control and diabetic groups, muscle force distribution for each of the 12 muscles (8 ankle plantarflexors: Flexor Digitorum, Flexor Hallucis, Gastrocnemius Lateral Head, Gastrocnemius Medial Head, Peronus Brevis, PeronusLongus, Soleus, Tibialis Posterior and 4 ankle dorsiflexors: Extensor Digitorum, Extensor Hallucis, Peroneus Tertius, Tibialis Anterior) acting on the ankle joint were calculated.

2.4. Statistical analysis

2.4.1. Outliers and Extremes

In order to achieve equinumerosity of the analysis groups (20 persons in each group) and in order to further simulation of muscle force distribution in the OpenSim software, the number of persons in the diabetes group was reduced. To do this the Statistica 8.0 software (StatSoft, PL) was used and analysis of outliers and extremes was applied. Analysis of outliers and extremes was applied for the following parameters: foot dorsiflexors, foot plantarflexors, foot inversion, foot eversion, lesser toes flexors, lesser toes extensors, hallux flexors, and hallux extensors which were measured using HHD device. Extreme values are the lowest and highest values in a given data set, while outliers are values that are significantly higher or lower than the remainder of the data. In order to be an outlier, the value must be:

• larger than quartile 3 by at least 1.5 times the interquartile range, or

• smaller than quartile 1 by at least 1.5 times the interquartile range (Aggarwal, 2013).

All participants with extreme values at both ends were excluded from further analysis.

2.4.2. Differences between groups

The ratio of agonist to antagonist (Ago/Ant) for each individual muscle was calculated in order to eliminate the fact that healthy persons applied more dorsiflexion and plantarflexion force using following formula:

$$Ago/Ant = \frac{F_{individual\,plantarflexion\,muscle}\ for\ PF_{action}}{F_{individual\,plantarflexion\,muscle}\ for\ DF_{action}} \quad \text{or}$$

$$Ago/Ant = \frac{F_{individual\,dorsiflexion\,muscle}\ for\ DF_{action}}{F_{individual\,dorsiflexion\,muscle}\ for\ PF_{action}} \quad (1)$$

In order to assess the groups of dorsiflexion and plantarflexion muscles as a sum of individual muscle force contribution under applied plantarflexion and dorsiflexion measured force for both participants group the following ratios have been applied:

$$RPF = \frac{\sum_{n=1}^{8} F_{PF_n} \ for\ PF_{action}}{\sum_{n=1}^{8} F_{PF_n} \ for\ DF_{action}} \qquad RDF = \frac{\sum_{n=1}^{4} F_{DF_n} \ for\ DF_{action}}{\sum_{n=1}^{4} F_{DF_n} \ for\ PF_{action}} \qquad (2)$$

where: $\sum_{n=1}^{8} F_{PF_n} \ for\ PF_{action}$ means the sum of forces of the eight individual plantarflexors when measured by HHD plantarflexion force was applied during simulation. The same explanation applies to the other components of the formula (1).

Normality of measured, simulated and calculated data distribution was assessed using the Shapiro-Wilk test. Non parametric U Mann–Whitney test was used to determine statistical significance between the diabetic and control group for all parameters. All data were analysed using Statistica 8.0 with the alpha level set at 0.05.

3. Results

3.1. *Outliers and Extremes*

An outlier and extreme are observations that lies an abnormal distance from other values in a random sample from a population. For all collected data for the diabetic group the box-and-whisker plots were completed in order to determine outliers and extremes points (Fig. 1).

Insert figure 1 here.

Through further analysis,people with more than two extremes were eliminated, which could be any combination of experimentally measured values. In the plantarflexion group values which were less than 90N and more than 190N were considered extremes. For the dorsiflexors group extreme values were below 70N and more than 170N. For inversion and lesser toes extensors group extreme values were below 50N and more than 110N. Similar condition was found for foot eversion and lesser toes flexors 60N and 120N. Extremes values for hallux flexorswere 60N and 130N, and for hallux extensors 40N and 90N.

3.2. *Healthy and diabetes comparison*

The Shapiro-Wilk test indicated the measured and simulated datawas not normally distributed ($P < 0.05$). Thus, in order to determine statistical significance between the diabetic and control group for all parameters U Mann–Whitney test was used.

Insert table 1 here.

Results presented in Table 1 demonstrated significant difference between diabetic and controls group for all of measured parameters. Moreover, all measured parameters values in the healthy control group were almost 1.5 times larger than in diabetic group.

Insert table 2 here.

Results of simulation of individual muscle force distribution for plantarflexion and dorsiflexion acting force are presented in Table 2. Similar to the results from the HHD testing

we observed significant differences between diabetic and controls group for all of the individual muscle forces. Mean force for all muscles is almost 1.19 times higher for the control group during isometric plantarflexion and 1.11 during isometric dorsiflexion, when compared to the diabetic group.

Calculating the ratio of agonist to antagonistmuscles using formula (1), the fact that the healthy controls applied more force than the patients with diabetes (as measured by the HHD) was eliminated. For Ago/Ant radio we found that half the number of dorsiflexors (Extensor Digitorum, Tibialis Anterior) and half the number of plantarflexors muscle (Flexor Digitorum, Flexor Hallucis, Peronus Longus, Tibialis Posterior) shows no statistically significant difference ($P > 0.05$) between the group of healthy subjects and patients.

Insert figure 2 here.

Formula (2) was applied in order to assess the groups of dorsiflexion and plantarflexion muscles as a sum of individual muscle force contribution under applied plantarflexion and dorsiflexion measured force for both participants groups. Figure 2 is a box-and-whisker plot showing the median and interquartile ranges of ratios for control and diabetic group. Significant differences were found between the ratio calculated for control group during application of plantarflexion force and all other ratios for control and diabetic group ($P = 0.00$). Moreover, a significant difference was found between the ratio calculated for the control group during application of dorsiflexion force and the diabetic group during application of plantarflexion force ($P = 0.01$).

4. Discussion

The aim of this study was to evaluate differences in foot and ankle muscle strength between patients with diabetes and control participants using hand-held-dynamometry. The subsequent aim focused on the assessment of differences in individual muscle force distribution between the groups based on data from hand-held-dynamometry. This study has shown a significant reduction in plantarflexion, dorsiflexion, inversion and eversion, lesser toes flexors, lesser toes extensors, hallux flexors and hallux extensors muscle strength in patients with diabetes. Consequently it was also found that individual muscle force for each of the 12 muscles (8 ankle plantarflexors and 4 ankle dorsiflexors) acting on the ankle joint were significantly less in diabetic group in comparison to the control group.

Duration of diabetes and poor metabolic control are well-known risk factors for the development of muscle weakness (Andersen et al., 2004; Harbo et al., 2012). Weakness evaluated by manual testing has been reported to be an independent risk factor for the development of foot ulcers, probably because muscle weakness at the ankle and knee in diabetic neuropathy leads to abnormal application of pressure at the sole of the foot during walking (Andersenet al., 1996). The results of this study is in line with (Park et al., 2006) who reported that muscle quality, defined as muscle strength per unit regional muscle mass, was significantly lower in men and women with diabetes than those without diabetes in both upper and lower extremities. Andreassen et al. (2006) observed a certain worsening in muscle performance in patients with peripheral neuropathy. Ijzermanet al. (2012) examined patients with and without polyneuropathy. In both group patients leg muscle strength was reduced by 30-50% compared to healthy subject. We found a lower reduction in muscle strength measured by HHD in our patients 28-37% compared to healthy. Giacomozzi et al.(2008) reported significant reduction of ankle mobility. They showed that dorsal-flexing torque were significantly reduced in all patients and in all foot positions, the highest reduction - 28% being for diabetes patients without neuropathy and 37% for patients withneuropathy. Since the

torque depends on both the force and the distance from the axis of rotation, and considering that for isometric conditions this distance has a constant value, we can see that our result show a 37% reduction for dorsiflexors and a 30% reduction for plantarflexors. In summary, results presented in Table 1 demonstrate that all values measured parameters by hand-held-dynamometry including: plantarflexion, dorsiflexion, inversion and eversion, lesser toes flexors, lesser toes extensors, hallux flexors and hallux extensors in the healthy control group were almost 1.5 times larger than in diabetic group.

Actual estimates of muscle forces can only be obtained with computational models in which the skeleton and muscles are both represented. Implemented in a variety of forms, musculoskeletal models have been used in conjunction with non-invasive measurements to obtain individual muscle forces during a number of movement tasks. Until now, simulation of muscle force distribution was applied for measured kinematics and kinetics parameters during gait for healthy and disabled persons (Anderson and Pandy, 1999; Wang and Gutierrez-Farewik, 2014). Within the current article, based on the data from a simple device which is the hand-held-dynamometer, individual muscle force could be estimated. Results for the simulation of individual muscle force distribution for plantarflexion and dorsiflexion acting force (Table 2) show a significant decrease in muscle force for each of the 12-muscles. Mean force for all muscle is almost 16% lower for diabetes group during isometric plantarflexion action and 10% lower during isometric dorsiflexion action. We need to note that the current study examined the muscle force contribution of Diabetic neuropathic patients; hence the observed difference between the groups is attributed to the combined effect of neuropathy and diabetes. Giacomozzi et al (2008),when examining groups of diabetic and diabetic neuropathic patients against healthy participants, attributed the overall decrease in the ankle moment to muscle atrophy as a results of muscle tissue glycation and damage in addition the muscle atrophy as a result of impaired nerve conduction. These differences together with

alterations of cartilages, ligaments and tendons as a result of glycation (Worbel and Najafi, 2010; Giacomozzi et al, 2005) could explain the deterioration in the muscle force contribution during isometric plantar and dorsi flexion contractions. Although the dynamometer is traditionally used for quantifying the agonists' moment generating capacity, co-activation of antagonists can complicate interpretation of results essential for evaluating the effectiveness of a structured rehabilitation program. Co-activation of the antagonist during a contraction of the agonists results in a negative moment in relation to the moment developed by agonists, reducing the net resultant moment output. Very few studies have reported antagonistic co-activation during agonistic maximal isometric contraction (Carolan and Cafarelli, 1992; Grabiner et al., 1992). In the current paper the ratio agonist to antagonist (Equation 1) was calculated for each individual muscle force (Table 2). It was found that this ratio was statistically significant different ($P < 0.05$) between the healthy and diabetic group for two dorsiflexors (Peroneus Tertius, Extensor Hallucis) and four plantarflexors muscle (Gastrocnemius Medial Head, Gastrocnemius Lateral Head, Soleus, Peronus Brevis). These results have implications and relevance to the area of gait dysfunction in diabetic patients. Although Kwon and colleagues (2003) indicated that when compared to the healthy controls, patients with Diabetic neuropathy show more co-contractions of agonist and antagonist ankle muscles during the stance phase of gait. This agonist –antagonist co-contractions was deemed to facilitate a safer and more stable gait pattern to compensate for diminished foot sensation. For example, Hohne et al., (2012) reported an increased tibialis anterior and decreased gastrocnemius medialis muscle activity during foot flat to mid-stance phase of gait during a simulated sensory neuropathy using in tradermal anaesthetic injections. This decreased eccentric muscle activity of the gastrocnemius medial head during this phase of gait over time could lead to a decrease in muscle strength when the muscles act as agonist during maximum voluntary ankle plantar flexion.

Based on this study the higher antagonist muscle force was expected since this group of muscles get activated as co – contractors during plantarflexion action. The antagonist muscle force for dorsiflexion action does not seem to reach the same magnitude as that of healthy individuals. On the other hand the fact that antagonist muscle force for plantarflexion muscle group in Diabetic patients is higher as compare to healthy controls can be attributed to neuropathy and to the fact that group of patients activate their plantarflexion muscles during dorsiflexion action to stabilize their joints. This may indeed be considered as the main reason for the observed increased antagonist muscle force for dorsiflexors group during isometric plantar flexion in diabetic patients as compared to healthy controls that is observed in the current study.

The results of this study could be further explained in the sense that Diabetic patients have learning effect and experience in activation dorsiflexion muscles as antagonist during stance phase of walking. Hence the Diabetic patients can effectively deactivate the antagonist muscle during isometric plantar flexion (dorsi flexor group) when there is no need for increasing balance or joint stiffness (i.e. when sitting and applying force to the dynamometer). On the other hand, Diabetic persons does not have experience of performing this task and with neuropathy and motor neural impairments they cannot deactivate these muscles. These individuals find deactivation antagonist plantar flexor muscles more challenging as compare to deactivating antagonist dorsi flexor muscles for which they train during stance phase of walking or in standing still.

The ratio of the sum of agonist to the sum of antagonist muscle forces (Equation 2) during dorsi-flexion and plantar-flexion were significantly different in the healthy group, while this ratio for plantar-flexion was significantly different for the healthy group compared to the diabetic counterpart (Figure 1).

In summary, while the ankle muscle strength seem to be consistently and significantly different between the diabetic and healthy participants, the agonist/antagonist muscle force ratio seem to be only significantly different for half of the muscles involved in ankle plantar/dorsi-flexion actions. Because the central nervous system regulates the level of co-contraction of agonist to antagonist muscles, it is likely that motor dysfunction as a result of diabetes and neuropathy may be more pronounced for the muscles for which the agonist/antagonist muscle force ratio were significantly different between the two groups. The ratio between agonist/antagonist muscle forces can be considered as a parameter showing the effectiveness of muscles in producing a high agonist isometric contraction and a low antagonist muscle contraction. While this ratio may be considered as a measure of neuro-muscular capability of individual muscle, the results of this study have implications in quantifying this capacity in diabetic patients.

The results of this study are in line with Mueller et al. (1994) who revealed that diabetic neuropathic patients were unable to generate sufficient ankle joint moment, with a consequent reduction in the dynamic function during walking, resulting in a smaller step length and stride, reducing gait speed and cadence. In the present work it has been shown that the force of gastrocnemius-soleus muscle group as a strong ankle plantarflexors of diabetic patients is reduced by 30% compare to healthy subjects, under isometric plantarflexion. While the force of tibialis anterior, peroneus tertius, extensor digitorum and extensor hallucis was reduced by 26%, under isometric dorsiflexion force. The findings of this study can help in qualified prediction of each individual patient's distal muscle strength. This information can then be used to design interventions at the early phase of the disease which could prevent the accelerated loss of strength and improve quality of life in these patients.

4.1 *Limitation of our study*

The identification of individual muscle contributions tobody support was possible through a detailed analysis of a computer simulation. Some limitations of musculoskeletal modeling and simulation generation and analysis have been described (Neptune et al., 2001; Zajac et al., 2002). Moreover in this paper, the CMC method was used based on 'healthy' model and it is documented in the literature (Orr et al. 2006; Andreassen et al. 2006; Lalli et al. 2013) important alterations in the muscle fibers histology and neurophysiology, as well in passive tissue properties in diabetic population. Therefore, using a healthy model to compute individual muscles force in diabetic individuals will definitely add some errors in the computation. But in the absence of the possibility to measure the individual muscles force, even estimation is good, in particular, when the report is presented first time in the literature. Therefore we believe that this is a challenge an open problem, but proposed in this paper research methodology applies only to static conditions, so this means that it can be used in the general diagnosis of the maximum muscle force loss in diabetics.

5. Conclusions

This preliminarystudy adds to the limited amount of published information on foot and ankle muscle strength in patients with diabetes and increases the knowledge base on the individual muscle force distribution. The results indicate that patients with diabetes have reduced muscle strength in foot and ankle plantarflexion, dorsiflexion, eversion and inversion, lesser toes flexors, lesser toes extensors, hallux flexors and hallux extensors muscle strength. Consequently it was also found that muscle force for each of the 12 muscles (8 ankle plantarflexors and 4 ankle dorsiflexors) acting on the ankle joint were significantly less in the diabetic group in comparison to the control group. It is likely that the muscles for which the agonist/antagonist muscle force ratio were significantly different between the healthy and

diabetic groups, during ankle plantar/dorsi-flexion actionswere more affected by diabetes and may need more attention during rehabilitation programmers. Results from this study provide information for future research in this area.

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Table 1 The mean and standard deviation (SD) for muscle strength testing measured with HHD, for diabetic and control group where: * indicates significance at the $P < 0.05$ level for U Mann-Whitney test.

Table 2 The mean and standard deviation (SD) for individual muscle force distribution for measured PF and DF action force and ratio agonist to antagonist (Ago/Ant) for diabetic and control group, where: * indicates significance at the $p < 0.05$ level for U Mann-Whitney test.

Fig 1 Example of outliers and extremes for dorsiflexors data collected in group of 48 diabetic patients.

Fig. 2 Box and whisker plots (median and interquartile range) for ratios (RDF and RPF) for control and diabetic group, where RDF, RPF - ratio of sum of agonist to sum of antagonist.

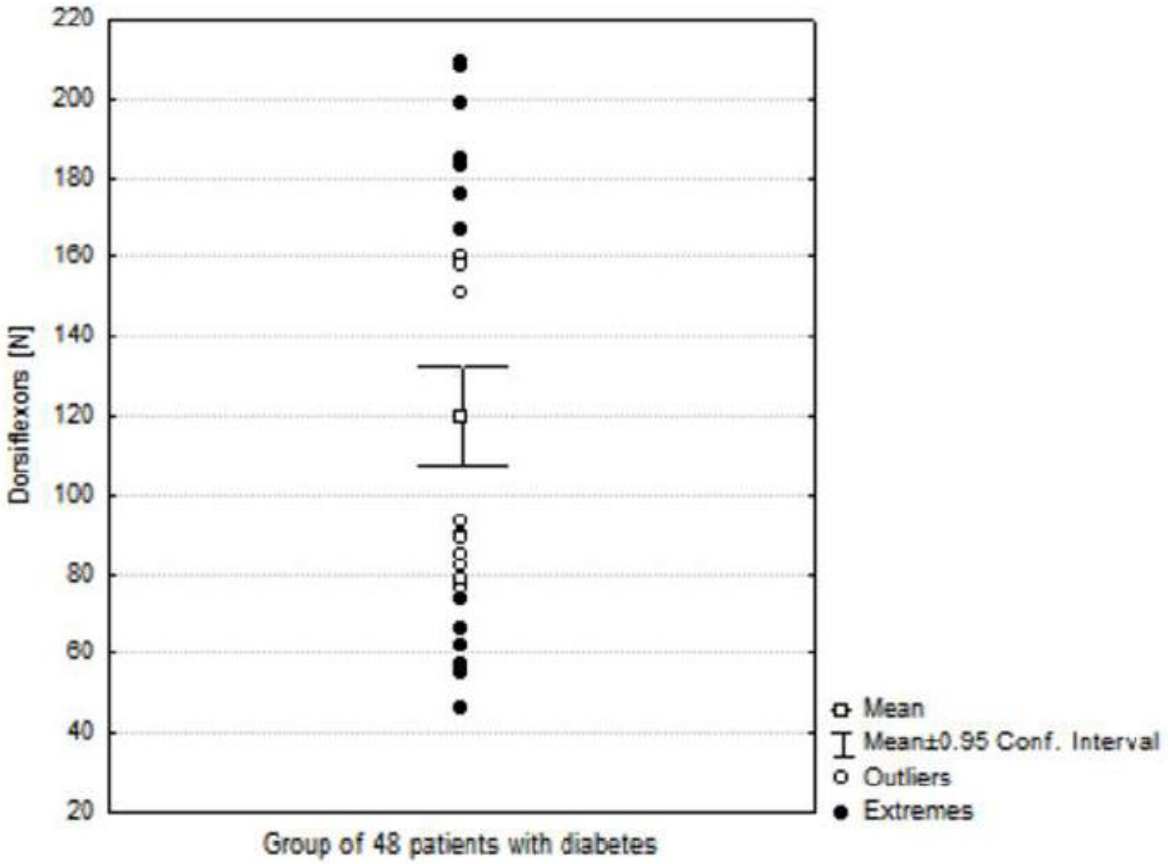

Fig. 1

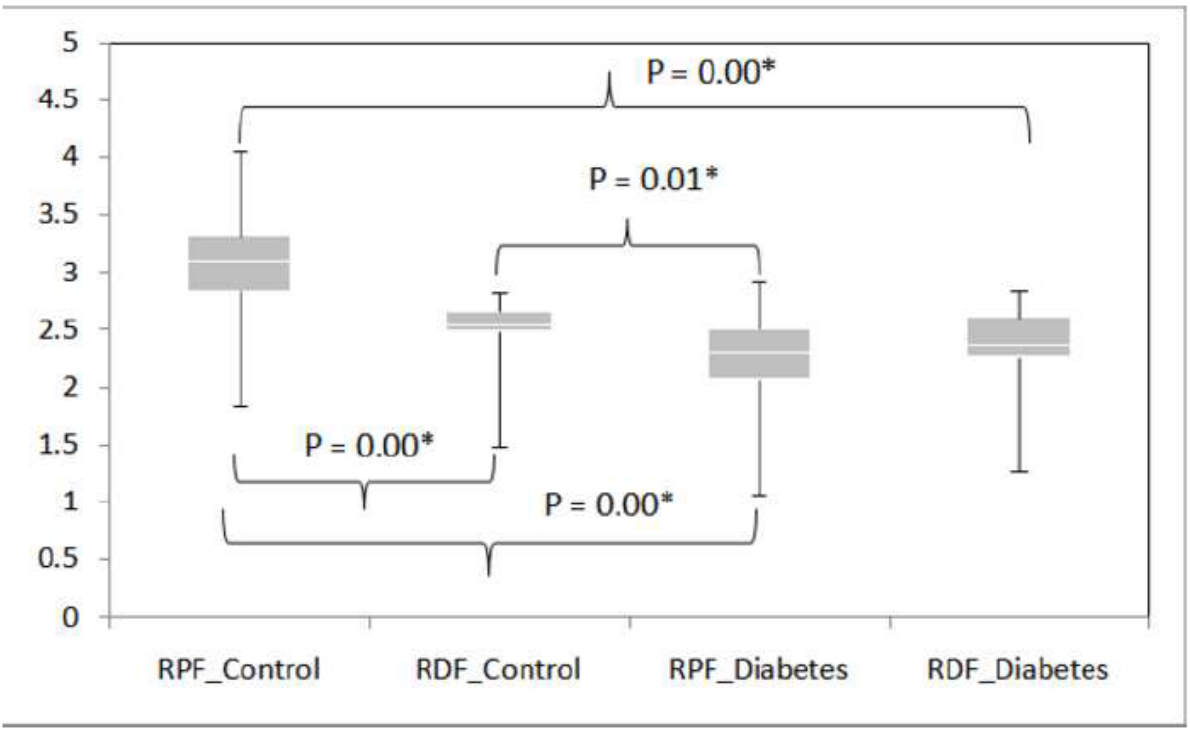

Fig. 2

Table 1 The mean and standard deviation (SD) for muscle strength testing measured with HHD, for diabetic and control group where: * indicates significance at the $P < 0.05$ level for U Mann-Whitney test.

Movement	Diabetic group mean (SD)	Control group mean (SD)	P - value
Plantarflexion [N]	142.2 (27.87)	203.68 (32.7)	0.000*
Dorsiflexion [N]	112.83 (24.75)	178.73 (27.41)	0.000*
Inversion [N]	77.7 (16.42)	114.7 (26.69)	0.000*
Eversion [N]	83.98 (13.15)	123.83 (25.88)	0.000*
Lesser toes flexors [N]	90.08 (17.24)	128.65 (33.05)	0.000*
Lesser toes extensors [N]	71.65 (13.1)	105.83 (27.8)	0.000*
Hallux flexors [N]	97.1 (19.93)	140.65 (34.68)	0.000*
Hallux extensors [N]	63.38 (10.6)	88.38 (28.54)	0.001*

Table 2 The mean and standard deviation (SD) for individual muscle force distribution for measured PF and DF action force and ratio agonist to antagonist (Ago/Ant) for diabetic and control group, where: * indicates significance at the $p < 0.05$ level for U Mann-Whitney test.

	Diabetic group mean (SD)			Control group mean (SD)			P-value		
Individual muscle force	PF action	DF action	Ago/ Ant	PF action	DF action	Ago/ Ant	PF action Diabetic vs. Control	DF action Diabetic vs. Control	Ago/Ant Diabetic vs. Control
Gastrocnemius Medial Head [N]	359.88 (55.16)	48.52 (0.15)	7.42 (1.18)	458.15 (44.55)	48.28 (0.09)	9.49 (0.94)	0.000*	0.000*	0.000*
Gastrocnemius Lateral Head [N]	64.71 (10.98)	23.29 (3.51)	2.84 (0.6)	84.34 (8.9)	22.4 (0.04)	3.77 (0.4)	0.000*	0.000*	0.000*
Soleus [N]	146.09 (40.07)	94.23 (0.61)	1.55 (0.44)	266.94 (75.31)	93.45 (0.29)	2.86 (0.81)	0.000*	0.000*	0.000*
Tibialis Posterior [N]	79.39 (0.09)	78.88 (0.09)	1.01 (0)	79.53 (0.06)	78.77 (0.04)	1.01 (0)	0.000*	0.000*	0.279
Flexor Digitorum [N]	9.29 (0.01)	9.24 (0.01)	1.01 (0)	9.31 (0.01)	9.23 (0.00)	1.01 (0)	0.000*	0.000*	0.417
Flexor Hallucis [N]	9.23 (0.01)	9.14 (0.01)	1.01 (0)	9.25 (0.01)	9.13 (0.01)	1.01 (0)	0.000*	0.000*	0.297
Tibialis Anterior [N]	172.17 (22.46)	421.71 (56.3)	2.46 (0.21)	223.84 (27.75)	569.32 (60.1)	2.55 (0.19)	0.000*	0.000*	0.085
Peronus Brevis [N]	15.56 (0.01)	15.49 (0.01)	1.01 (0.01)	15.58 (0.01)	15.47 (0.01)	1.01 (0)	0.000*	0.000*	0.030*
Peronus Longus [N]	35.91 (0.03)	35.74 (0.03)	1.01 (0.01)	35.95 (0.02)	35.7 (0.01)	1.01 (0)	0.000*	0.000*	0.058
Peroneus Tertius [N]	6.58 (0.03)	10.83 (1.7)	1.65 (0.26)	6.55 (0.13)	14.91 (1.68)	2.28 (0.26)	0.000*	0.000*	0.000*
Extensor Digitorum [N]	52.34 (6.95)	126.74 (15.91)	2.44 (0.32)	68.65 (8.81)	170.8 (21.04)	2.5 (0.23)	0.000*	0.000*	0.180
Extensor Hallucis [N]	5.38 (0.47)	13.3 (1.68)	2.47 (0.27)	6.78 (0.86)	17.84 (2.08)	2.64 (0.22)	0.000*	0.000*	0.025*