

Influence of dopaminergic treatment on resting elbow joint angle control mechanisms in patients with Parkinson's disease – a preliminary report

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Purpose: Heightened tonic stretch reflex contributes to increased muscle tone and a more-flexed resting elbow joint angle (EJA) in patients with Parkinson's disease (PD). Dopaminergic medication restores central nervous system (CNS) functioning and decreases resting muscle electrical and mechanical activities. This study aimed to evaluate the effects of dopaminergic medication on parkinsonian rigidity, resting EJA, resting electrical activity (electromyography, EMG) and mechanical properties (myotonometry, MYO) of elbow flexor muscles and the associations of EJA with these muscles resting electrical activity and mechanical properties in PD patients. We also evaluated a relationship between dopaminergic treatment dose and these outcome measures values. *Methods:* Ten PD patients (age 68 ± 10.1 years; body mass 70 ± 16.8 kg; height 162 ± 6.6 cm; illness duration 9 ± 4.5 years) were tested during medication on- and off-phases. Resting EJA, myotonometric muscle stiffness (S-MYO) and root mean square electromyogram amplitude (RMS-EMG) were recorded from relaxed biceps brachii and brachioradialis muscles. Based on the above parameters, we also calculated the EJA/S-MYO ratio and EJA/RMS-EMG ratio. Parkinsonian rigidity was assessed using the motor section of the Unified Parkinson's Disease Rating Scale. *Results:* EJA, EJA/S-MYO ratio, and EJA/RMS-EMG ratio were increased and S-MYO, RMS-EMG, and parkinsonian rigidity were decreased during the medication on-phase compared with the off-phase. In addition, the dopaminergic treatment dose was negatively correlated with S-MYO and RMS-EMG, and positively correlated with EJA/S-MYO and EJA/RMS-EMG ratios. *Conclusions:* We conclude that dopaminergic medication-induced improvements in resting elbow joint angle in tested patients with PD are related to changes in their muscle electrical and mechanical properties.

Key words: Parkinson's disease, electromyography, myotonometry, motor control, joint angle, medication

1. Introduction

Pathologic changes in the central nervous system (CNS) in Parkinson's disease (PD) include disturbances in the functioning of extrapyramidal dopamine-dependent neural structures [10] influencing motor control [22]. These structural and functional alterations in CNS lead to an increased spinal tonic

stretch reflex associated with higher electrical muscle activity and consequently greater resting muscle stiffness and rigidity in patients with PD [3], [5], [8], [9], [18], [22], [24], [25], [27], [31]. The fact that the tonic stretch reflex is heightened in flexor more than extensor muscles in PD [3] is an important factor regulating resting joint angle, leading to the relatively flexed posture of patients with parkinsonism [18], [31]. Recent studies proposed assessing muscle and joint stiff-

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ness in PD based on the combined clinical rating of rigidity (subjective) and dynamometry and/or electromyography (EMG) (objective) during passive movement [3], [5], [9], [22], [24], [25], [31]. Using myotonometry and goniometry, we previously reported increased rigidity-related muscle stiffness in the biceps brachii (BB) muscle at rest, accompanied by a tendency towards a more flexed elbow joint in PD patients compared to healthy controls [18]. Myotonometry is an objective, reliable, noninvasive and easy to use technique with potentially wide applications for investigating muscle properties in areas such as scientific research, medicine, sport, and rehabilitation [1], [2], [4], [6], [7], [11]–[13], [15], [17], [20], [23], [26]–[29]. The muscle stiffness [N/m] measured by myotonometer is determined as a ratio of low mechanical shock force (transmitted via testing end to skin surface overlying tested muscle and not inducing muscle reflex activity) to depth of the tissue deformation [13]. This method is an ideal alternative to invasive biomechanical methods for an assessment of mechanical properties of soft tissues in humans [14] and to other technically complicated non-invasive methods [3], [5], [8], [9], [22], [24], [25], [31]. Compared to dynamometry, myotonometry has the advantage of being easier to use in everyday practice and allows the mechanical properties of single muscles to be measured, while dynamometry measures the joint torque produced by a set of agonistic and antagonistic muscle groups. Furthermore, our conclusions regarding muscle tendon unit stiffness in PD based on myotonometric measurements at rest [18] were similar to those based on dynamometric measurements during passive movement [31], (i.e., PD patients have a more flexed elbow joint angle [EJA] related to increased elbow flexor muscle stiffness), indicating the potential use of myotonometry for investigating muscle tendon unit mechanical properties in patients with PD [18], [27].

Dopaminergic medication restores CNS activity in patients with PD [16], [30] and alleviates the increased spinal neuron activity [16], leading to decreased resting muscle electrical activity [5], [16], [30] and muscle stiffness [8], [9], [22], [25]. This suggests that the medication-induced improvements in the CNS might be associated with normalization of the resting joint angle (less-flexed position) related to a simultaneous decrease in muscle stiffness. However, no studies have yet investigated the relationship between medication-induced changes in resting EJA (measured by goniometry) and resting flexor muscle functional state, in terms of myotonometric muscle stiffness and electromyographic muscle activity assessment in PD.

This information would extend our understanding of PD and may also have clinical implications in terms of diagnosis and therapeutic monitoring. This study therefore aimed to measure resting EJA using goniometry, resting elbow flexor (BB and brachioradialis [BR]) muscle stiffness with myotonometry (S-MYO), and electromyographic amplitude. We also tested the relationships among these measured parameters during medication on- and off-phases in PD patients. We hypothesized that dopaminergic medication would increase resting EJA to achieve a less-flexed position, associated with decreased resting electromyographic activity and myotonometric stiffness in the BB and BR muscles, and decreased parkinsonian rigidity of the upper extremities. We also hypothesized that this medication-induced benefit would be positively correlated with a dose of the dopaminergic treatment.

2. Methods

2.1. Subjects and experimental procedures

Ten patients (six women, four men, mean age 68 ± 10.1 years; body mass 70 ± 16.8 kg; height 162 ± 6.6 cm; illness duration 9 ± 4.5 years) (Table 1) diagnosed with PD participated in our study. All patients were tested clinically according to the Hoehn and Yahr scale and Unified Parkinson's Disease Rating Scale (UPDRS), and biomechanically by goniometry, myotonometry, and electromyography. All goniometric, myotonometric, and electromyographic measurements were performed on the affected or more-affected upper extremity. To evaluate the influence of medication on clinical and biomechanical outcomes, patients were tested during their medication on-phase (first session) and off-phase (second session) with a 1-week interval between sessions. On-phase was defined as a period of beneficial effects of anti-parkinsonian medication, which was mainly *L*-dopa (monopharmacotherapy), or *L*-dopa with piribedil or ropinirol (polypharmacotherapy) in some patients (Table 2). Off-phase testing was carried out after withdrawal of anti-parkinsonian drugs for 12 hours overnight. According to the Hoehn and Yahr scale, all tested patients had either mild or mild-to-moderate PD, except for one patient who had severe disease (Table 2). According to the motor section of the UPDRS, upper-extremity rigidity during the medication on-phase ranged from absent (one subject), through slight (six subjects) and mild-to-

Table 1. Anthropometric characteristics of tested Parkinson's disease patients

Patient No.	Age [year]	Sex F/M	Body mass [kg]	Height [cm]
PD#1	76	M	69	159
PD#2	51	F	49	162
PD#3	72	F	64	161
PD#4	54	M	108	174
PD#5	58	M	80	170
PD#6	75	F	72	154
PD#7	66	M	82	168
PD#8	80	F	61	154
PD#9	68	F	54	159
PD#10	76	F	64	162

Abbreviations: No. – number, PD – Parkinson's disease, F – female, M – male.

Table 2. Clinical characteristics of tested Parkinson's disease patients

Patient No.	Hoehn and Yahr stage	Rigidity (UPDRS, item 22)		Medication dose [mg/day]
		On phase	Off phase	
PD#1	2.5	1	2	ΣL (L+LED) = 750 L:600; P:150 (LED:150)
PD#2	2.5	0	1	L:1050
PD#3	4	1	2	L:1100
PD#4	3	1	2	ΣL (L+LED) = 640 L:600; R:2(LED:40)
PD#5	2.5	2	2	ΣL (L+LED) = 1070 L:950; R:6(LED:120)
PD#6	3	1	2	ΣL (L+LED) = 280 L:200; R:4(LED:80)
PD#7	2.5	3	3	ΣL (L+LED) = 480 L:400; R:4(LED:80)
PD#8	3	1	2	ΣL (L+LED) = 850 L:700; P:150(LED:150)
PD#9	3	2	3	L:350
PD#10	2.5	1	1	L:600

Abbreviations: No. – number, PD – Parkinson's disease, UPDRS – Unified Parkinson's Disease Rating Scale, On-phase – disease status when parkinsonian symptoms are alleviated after medication, Off-phase – disease status when parkinsonian symptoms are intensified after withdrawal of medication, L – levodopa (monopharmacotherapy), P – Piribedil, R – Ropinirol, LED – levodopa equivalent dose (when in polypharmacotherapy the R and P were administered), ΣL – the sum of L and LED in polypharmacotherapy.

-moderate (two subjects), to marked rigidity (one subject) (Table 2). During the medication off-phase, rigidity ranged from slight (two subjects), through mild-to-moderate (six subjects), to marked rigidity (two subjects) (Table 2). This study was approved by

the Ethics Committee at the University School of Physical Education in Wrocław (Poland) and performed in accordance with the Helsinki Declaration. All patients gave their written consent prior to participation in the study.

2.2. Goniometric measurements

Three measurements of resting EJA were taken in the standing position for each subject. The subject stood comfortably with their back leaning against the wall and their upper extremities relaxed along the trunk. EJA measurements were performed using a hand-held, stainless steel, two-armed (each 25 cm long) goniometer (Saehan Corporation, Daegu, South Korea), with a 360° scale (marked in 1° increments). The bony landmarks used to determine resting EJA were: goniometer centered on the lateral epicondyle, stationary arm pointing at the tip of acromion process, and mobile arm pointing at the ulnar styloid process.

2.3. Myotonometric measurements

Resting muscle stiffness measurements were performed using a Myoton-3 device (Müomeetria AS, Tallinn, Estonia) over the center of the bellies of the BB and BR muscles, with the subjects in the supine position, their upper extremities along the trunk, and their forearm positioned between pronation and supination. After instructing the subject to relax their muscles, an experimenter placed the testing end of the Myoton-3 on the skin surface overlying the relaxed muscle belly and performed standard myotonometric measurements [6], [11], [15] with 20 consecutive records for the BB followed by the BR muscle, respectively. Based on the myotonometric parameters, we analyzed muscle stiffness (S-MYO; N/m), which describes the ability of biological tissue to resist an external force that changes its shape. Higher values of S-MYO represent the need for greater force to modify the transverse shape of the tissue. During contraction, muscle stiffness in healthy, young people usually increases proportionally with increasing contraction force up to 80% of maximal voluntary contraction [13]. We used this parameter because it is sensitive to changes in muscle stiffness in relation to changed muscle activation [11]–[13], [15], or as a consequence of changes in CNS activity in PD [27].

2.4. Electromyographic recordings and analysis

Prior to myotonometric measurements, resting surface electromyography (EMG) was recorded (three 3-s trials) from the same location over the BB and BR muscle bellies, with the patient in the same position, using a custom-made bipolar EMG probe. The probes were attached to the surface of the skin using a double-sided adhesive washer, and their locations were marked with a waterproof marker. The EMG electrode was 4 mm in diameter with an inter-electrode distance of 25 mm. The details of the custom-made EMG system have been described elsewhere [19]. The EMG signals were recorded and stored on the hard disc of a personal computer after analog-to-digital conversion. The sampling rate was 10 kHz for each of the two EMG channels. A programmable gain amplifier was used to amplify the EMG signals, with the amplifier gain adjusted to obtain the best signal-to-noise ratio (signal-to-noise ratio for EMG was about 8.0×10^3 , common mode rejection ratio of the amplifier was 100 dB). The recorded EMG signals were then filtered using software band-pass filters of 20–500 Hz. Online visual inspection and offline analysis (root mean square, RMS) of this signal were performed to check whether the measurements were made without the influence of parkinsonian resting tremors, and to estimate the influence of medication on tonic muscle electrical activity.

2.5. Statistical analysis

The EJA ($^{\circ}$) was calculated as the average of three measurements. S-MYO was expressed as the mean of 20 consecutive records, and RMS-EMG (μV) was calculated from three resting trials. We then calculated the angle-related ratios EJA/S-MYO ($^{\circ}/\text{N}/\text{m}$) and EJA/RMS-EMG ($^{\circ}/\mu\text{V}$), based on the S-MYO and RMS-EMG values.

The Shapiro–Wilk test was used to determine if the parameters satisfied the conditions for a normal distribution. Wilcoxon's signed-rank test and Student's *t*-test were used to test the significance of differences between the medication on- and off-phase sessions, as appropriate. Pearson's (*r*) correlation coefficient was used to test the relationships between L-dopa dose (monopharmacotherapy) or ΣL dose (the sum of L-dopa dose and L-dopa equivalent dose in polypharmacotherapy) and goniometric, myotonometric, or electromyographic parameters, and between myoto-

nometric and goniometric or electromyographic parameters. The Spearman's (ρ) correlation coefficient was used to test relationships between L-dopa dose (monopharmacotherapy) or ΣL dose (polypharmacotherapy) and parkinsonian rigidity. A value of $\alpha \leq 0.05$ was considered significant for all analyses.

3. Results

3.1. Myotonometric stiffness and EMG amplitude

S-MYO was significantly decreased by 8% in BB ($P = 0.048$) and 13% in BR muscles ($P = 0.033$), and RMS-EMG was significantly decreased by 20% in BB ($P = 0.035$) and 40% in BR muscles ($P = 0.004$) during the on-phase, compared with the off-phase.

3.2. EJA, EJA/RMS-EMG, and EJA/S-MYO

EJA (Fig. 1A; $P < 0.05$), EJA/RMS-EMG BR ratio (Fig. 1C; $P < 0.05$), and EJA/S-MYO ratio for both BB (Fig. 1D) and BR muscles (Fig. 1E) ($P < 0.05$) were significantly greater during the on-phase compared with the off-phase. EJA/RMS-EMG BB ratio also tended to be greater during the on-phase, but the difference was not significant (Fig. 1B; $P > 0.05$).

3.3. Clinical rating of parkinsonian rigidity

The mean rigidity score improved significantly (i.e., decreased) during the medication on-phase compared with the off-phase (2.0 ± 0.7 versus 1.4 ± 0.8 , respectively; $P = 0.008$).

3.4. Relationships between dopaminergic treatment dose and tested parameters

There was a tendency towards a positive correlation between dopaminergic treatment dose and EJA ($P > 0.05$; Table 3), but the result was not significant. However, dopaminergic treatment dose was positively correlated with EJA/RMS-EMG BB and EJA/RMS-EMG

BR, and with EJA/S-MYO BB and EJA/S-MYO BR ratios ($P < 0.05$; Table 3). There was a significant negative relationship between dopaminergic treatment dose and RMS-EMG and S-MYO for both the BB and BR muscles ($P < 0.05$; Table 3). Furthermore, there was a significant negative relationship between dopaminergic treatment dose and parkinsonian rigidity ($P < 0.05$; Table 3).

Pearson's correlation coefficients indicated a significant negative relationship between S-MYO BR and EJA ($P < 0.05$; Table 3), and a slight, but not significant, tendency towards a negative correlation between S-MYO BB and EJA ($P > 0.05$; Table 3). There was a significant positive relationship between RMS-EMG and S-MYO for both the BB and BR muscles ($P < 0.05$; Table 3).

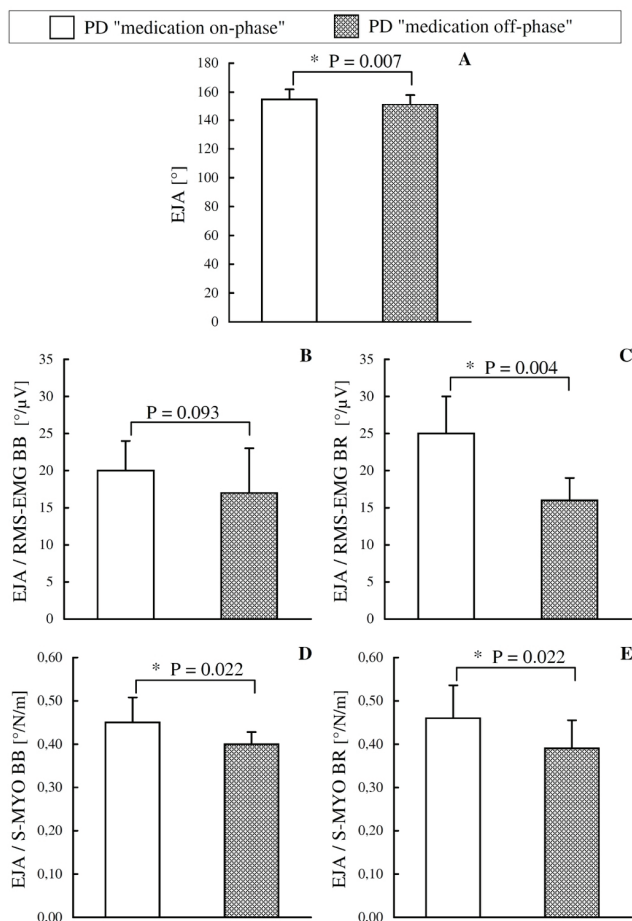


Fig. 1. Elbow joint angle (panel A) and ratios of elbow joint angle to electromyogram amplitude (panels B and C) and to myotonometric stiffness (panels D and E) during medication on- and off-phase.

Abbreviations: EJA – elbow joint angle, RMS-EMG – electromyogram amplitude, BB – biceps brachii muscle, BR – brachioradialis muscle, S-MYO – myotonometric measurements of stiffness. * – $P \leq 0.05$ for comparison between medication on-phase and off-phase

Table 3. The correlation's values of (1) the dose of dopaminergic treatment with: elbow joint angle, elbow joint angle ratios, electromyogram amplitude, myotonometric stiffness and parkinsonian rigidity scores; (2) myotonometric stiffness with elbow joint angle; and (3) electromyographic amplitude with myotonometric stiffness

Correlated variables	Correlation	
	ρ / r	P
L or Σ L with EJA	$r = 0.252$	$P = 0.141$
L or Σ L with EJA/RMS-EMG BB	$r = 0.487$	$P = 0.017 *$
L or Σ L with EJA/RMS-EMG BR	$r = 0.583$	$P = 0.003 *$
L or Σ L with EJA/S-MYO BB	$r = 0.591$	$P = 0.003 *$
L or Σ L with EJA/S-MYO BR	$r = 0.441$	$P = 0.026 *$
L or Σ L with RMS-EMG BB	$r = -0.411$	$P = 0.036 *$
L or Σ L with RMS-EMG BR	$r = -0.567$	$P = 0.005 *$
L or Σ L with S-MYO BB	$r = -0.545$	$P = 0.006 *$
L or Σ L with S-MYO BR	$r = -0.438$	$P = 0.027 *$
L or Σ L with RIGIDITY _{UPDRS}	$\rho = -0.448$	$P = 0.024 *$
S-MYO BB with EJA	$r = -0.143$	$P = 0.273$
S-MYO BR with EJA	$r = -0.381$	$P = 0.049 *$
RMS-EMG BB with S-MYO BB	$r = 0.424$	$P = 0.031 *$
RMS-EMG BR with S-MYO BR	$r = 0.433$	$P = 0.028 *$

Abbreviations: L – levodopa (monotherapy), Σ L – the sum of levodopa dose and levodopa equivalent dose when in polypharmacotherapy the Piribedil or Ropinirol were administered, EJA – elbow joint angle, RMS-EMG – electromyogram amplitude, BB – biceps brachii muscle, BR – brachioradialis muscle, S-MYO – myotonometric measurements of stiffness, UPDRS – Unified Parkinson's disease scale, ρ – Spearman's correlation coefficient, r – Pearson's correlation coefficient. * – $P \leq 0.05$ for correlations.

4. Discussion

The results of this study demonstrated that dopaminergic anti-parkinsonian agents improved EJA, decreased elbow flexor muscle electrical activity and stiffness, increased the ratios of EJA to muscle electrical activity and stiffness, and decreased parkinsonian rigidity in the upper extremities. Furthermore, the dopaminergic treatment dose correlated negatively with resting elbow flexor muscle electrical activity and stiffness and upper-extremity parkinsonian rigidity, and positively with the ratio of EJA to muscle electrical activity and stiffness.

4.1. Medication effects on EJA, elbow flexor muscle state, and parkinsonian rigidity

Watts et al. [31] indicated that the relaxed EJA was more flexed in patients with PD compared to healthy

people, as a result of greater passive stiffness in the flexor muscle tendon units, leading to changes in the length–tension curves in the elbow joint muscles. Similarly, we previously [18] showed a tendency to lower (more-flexed) EJA, higher BB myotonometric stiffness, and lower ratio of EJA to myotonometric stiffness in the BB muscle in PD patients during the medication on-phase, compared to healthy controls. These studies [18], [31] suggest that trends of EJA, elbow flexor S-MYO, and EJA/S-MYO ratio would be reversed during the medication off-phase. The results of the present study support this hypothesis, given that EJA was increased (i.e., less-flexed) in PD patients tested after medication intake, with a simultaneous decrease in elbow flexor muscle stiffness and increase in EJA/S-MYO ratio. These findings might thus provide an explanation that accounts for the previously identified [18], [31] interaction between elbow flexor muscle stiffness and EJA. Namely, the medication-induced decrease in flexor muscles stiffness (S-MYO) may change the length–tension relationship of the elbow flexors, consequently increasing the EJA. This hypothesis is supported by the observed medication-induced increase in EJA/S-MYO ratio, whereby the increased ratio during the medication on-phase results from an increase in EJA and simultaneous decrease in S-MYO under these respective conditions. EJA/S-MYO ratio might thus be a useful parameter for describing the role of muscle mechanical properties in regulating the joint angle.

The medication-induced decrease in elbow flexor muscle stiffness may be related to the electromyographic activity of these muscles at rest, which decreased during the medication on-phase. RMS-EMG depends on the level of motor-unit recruitment [21], which is governed by CNS activity. The medication-induced decrease in RMS-EMG values detected in this study may thus reflect changes in CNS activity after medication intake in our patients. Such changes in CNS drive might normalize spinal cord reflex activity (via shift of the tonic stretch reflex threshold towards longer muscle-tendon unit length [24]), thus decreasing the spindle reaction and causing decreased muscle activity [5], [8], [9], [22], [25], while simultaneously decreasing muscle stiffness and increasing EJA. The beneficial medication-induced effect on the relationship between EJA and electrical flexor muscle activity is reflected in the EJA/RMS-EMG ratio, with greater EJA and simultaneously lower RMS-EMG resulting in higher values of EJA/RMS-EMG during the on- versus the off-phase. This explanation is also supported by the results of correlation analyses, which showed a positive relationship between RMS-EMG and S-MYO, and

a negative relationship between EJA and S-MYO, indicating that muscle stiffness accordingly decreased with medication-induced decrease in flexor muscle electrical activity (positive correlation), while EJA increased with decreased S-MYO (negative correlation).

These above medication-induced changes in RMS-EMG, S-MYO, and EJA were in accordance with the improvements in rigidity after medication, supporting the idea that dopaminergic anti-parkinsonian agents cause positive alterations in the CNS [5], [16], [27], [30] in PD patients.

4.2. Relationships between dopaminergic treatment dose and biomechanical and clinical parameters

Marchand-Pauvert et al. [16] showed that L-dopa restored the enhanced group II spinal reflex excitation to a normal level in patients with PD, suggesting that L-dopa dose might correlate with the scale of response in terms of clinical and biomechanical outcomes related to medication-induced effects on spinal reflex activity. The clinical profiles of our PD patients were relatively consistent, with L-dopa or LED doses ranging from 200–1100 mg/day. This might explain why we were able to detect a negative relationship between dopaminergic treatment dose and flexor muscle RMS-EMG, S-MYO, and parkinsonian rigidity, and a positive relationship between L-dopa dose and EJA/RMS-EMG and EJA/S-MYO ratios. These results suggest that an higher dopaminergic treatment dose might decrease muscle electrical activity, muscle stiffness, and parkinsonian rigidity, and simultaneously increase the EJA towards a less-flexed position.

4.3. Significance of the results

The clinical and biomechanical outcomes of the present and previous studies enable us to propose a possible mechanism to explain the medication-induced improvement in EJA in patients with PD. The dopaminergic treatment intake improves the neural drive from the CNS [5], [16], [27], [30], which normalizes spinal reflex activity [5], [9], [16], [22], [25] leading to lower tonic muscle activity, reflected in lower resting muscle electrical activity (RMS-EMG) and muscle mechanical properties (S-MYO). The changes in muscle function then cause normalization of the resting elbow flexed position (increase of: EJA, EJA/RMS-EMG and EJA/S-MYO).

This information furthers our understanding of the mechanisms controlling EJA in PD. It is also valuable in terms of the diagnosis (in which reaction on dopaminergic treatment forms part of the diagnosis) and treatment of PD, given that an objective assessment of abnormal joint control (goniometry, myotonometry, electromyography) in PD is needed to assess an efficacy of various treatment approaches.

4.4. Limitations of the study

An important limitation of this study is the relatively small sample size. Nevertheless, an appropriate statistical analysis applied by us (for comparison of medication on- vs. off-phase and for correlations calculations) was able to reveal an effect of anti-parkinsonian medication on clinical and biomechanical outcomes even in this relatively small sample size. However, the results of this study cannot be generalized for the whole PD patients population due to this relatively small sample size and for the same reason the findings from this study have rather a preliminary character. However, the effects of dopaminergic medication on elbow joint angle and muscle state that we observed based on present comparative and correlation analyzes, warrant further study with a larger sample size to enable a more discerning analyzes.

5. Conclusions

Our findings indicate that an anti-parkinsonian dopaminergic medication improves resting elbow joint angle in patients with PD, and that this improvement is related to changes in the electrical and mechanical properties of elbow flexor muscles.

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