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Enhancement of parkinsonian rigidity with contralateral hand activation ☆

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HIGHLIGHTS

- Enhancement of rigidity by contralateral activation may aid in early detection of the disease.
- Medication reduced rigidity with and without contralateral activation.
- Increased rigidity with contralateral activation is not due to ipsilateral muscle activation.

ABSTRACT

Objective: Quantify the enhancement of parkinsonian rigidity associated with a contralateral activation maneuver.
Methods: Twelve subjects with PD and eight controls participated in the study protocol. Subjects' tested hand was displaced by a servo-motor throughout wrist flexion and extension motions of 60° without and with a concurrent gripping activation in the contralateral hand, referred to as Passive and Active conditions, respectively. Subjects with PD were tested in both OFF-MED and ON-MED states. Rigidity was quantified by integrating torque with position during both flexion and extension (torque resistance). ANOVA was performed to assess the effect of contralateral activation on rigidity.
Results: PD patients had significantly (0.038) enhanced torque resistance in OFF-MED compared to healthy controls and ON-MED. In the Active condition, differences in torque resistance were magnified ($p = 0.002$). Medication substantially reduced differences in torque resistance between controls and PD patients in the Passive and Active conditions.
Conclusions: A contralateral activation maneuver substantially increases rigidity in patients with PD, specifically the OFF-MED state. Rigidity is reduced with the application of dopaminergic medication, even with the presence of a contralateral activation maneuver.
Significance: These data support the use of a contralateral activation maneuver as a tool in the diagnosis of PD.

1. Introduction

Parkinsonian rigidity is described as increased, uniform resistance to passive limb movement throughout the range of motion (Fung and Thompson, 2002). Rigidity is a cardinal symptom of Parkinson's disease (PD), thereby used as a diagnostic criterion. According to the Motor Examination of the unified Parkinson's disease rating scale (UPDRS), assessment of rigidity, in clinic, "*is judged on slow passive movement of major joints with the patient in a relaxed position and the examiner manipulating the limbs and neck. If no rigidity is detected, ask the patient to perform an activation maneuver (e.g., tapping fingers, fist opening–closing or heel tapping in a limb not being tested)*" (Fahn and Elton, 1987). The rationale for the incorporation of an activation maneuver lies in that activation maneuver performed by the contralateral hand may enhance the degree of rigidity, as rigidity is not easily detectable in the early stage of the disease. However, the notion about the effect of contralateral activation on parkinsonian rigidity is empirical because its effect has never been quantitatively examined. The current study was conducted to quantify the effect of contralateral activation on parkinsonian rigidity. The wrist joint was chosen because it is commonly evaluated for rigidity in clinical practice (Fung and Thompson, 2002).

In addition to being used for a diagnostic purpose, rigidity is also used as a means of evaluating the efficacy of treatment, because it generally responds positively to therapeutic interventions. Dopaminergic medication and deep brain stimulation have been shown to reduce movement impairments associated with PD, such as rigidity, and directly affect the supraspinal coordina-

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tion and planning of movement (Benabid et al., 2009; Schapira et al., 2009).

The bilateral effects of unilateral muscle contraction have been well documented (Scripture et al., 1894; Hortobagyi et al., 1997, 1999, 2003; Hortobagyi, 2005). It has long been known that unilateral practice improves contralateral strength (Hortobagyi et al., 1997, 1999) and accuracy (Scripture et al., 1894; Zhou, 2000). This phenomenon, known as cross education, has been investigated using a variety of techniques (Hortobagyi et al., 1997, 1999, 2003; Zhou, 2000) and has proven beneficial in the rehabilitation paradigm. Specifically, unilateral exercise in the homologous muscle group in the uninvolved limb can lead to functional improvements in the injured limb (Stromberg, 1986; McCartney et al., 1988; Strens et al., 2003). However, it has also been suggested that dysfunction associated with injury or disease can also be transferred to the healthy limb (Fuchs et al., 1999; Koltzenburg et al., 1999). Previous research has shown that crossed effects of unilateral contractions are mediated by central pathways (Zhou, 2000; Hortobagyi et al., 2003; Hortobagyi, 2005) including spinal and supraspinal mechanisms; however, evidence exists suggesting cross education may also be mediated by peripheral factors including afferent input from muscle spindle fibers (Hortobagyi et al., 1997, 1999).

Though the phenomenon of cross education provides a mechanism for improved function in a rehabilitation paradigm, it describes the effect of contralateral training rather than the immediate effect of contralateral muscle contraction on ipsilateral function. The crossed effect, or the immediate effect of activation of the contralateral, homologous muscle group on ipsilateral function, has not been studied in people with PD. Therefore, the primary purpose of the current study was to determine the effect of activation of the contralateral wrist and hand muscles on rigidity of the wrist joint being examined. The secondary purpose was to quantify the effects of dopaminergic medication on rigidity with and without contralateral activation. Subjects with PD and control subjects were tested in the study. It was hypothesized that increased wrist rigidity associated with a contralateral activation maneuver: (1) would be present in people with PD and control subjects, (2) would be greater in people with PD while on dopaminergic medication in comparison to control subjects and (3) would be further enhanced in people with PD when withdrawn from medication.

2. Patients and methods

2.1. Informed consent

The experimental protocol was approved by the Institutional Review Board of Creighton University, Omaha, Nebraska, USA and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained prior to the participation of each subject in the current study.

2.2. Subjects

Twelve patients with idiopathic PD (7 M, 5 F) and eight healthy controls (4 M, 4 F) participated in the current study. The average age was 64 (±8.9) years for subjects with PD and 60 (±8.9) years for control subjects, respectively. Subjects' clinical characteristics are listed in Table 1.

2.3. Inclusion and exclusion criteria

Verbal medical history and the Motor Section (Part III) of the UPDRS were used to screen all patients for inclusion in the current study (Fahn and Elton, 1987). The inclusion criteria for people with PD were: (1) age between 30 yrs and 75 yrs, (2) treated using dopaminergic medication and (3) presence of clinical rigidity (>2, mild to moderate, or marked) in one or both arms when dopaminergic medication was withdrawn (4) minimal tremor (<1, slight and infrequently present) in the tested arm. Subjects with PD were excluded if cognitive impairments prevented the informed consent, understanding instructions or providing adequate feedback. Control subjects were age- and gender-matched to subjects with PD and had no history of neurological disorders. Any subject was excluded that had insufficient wrist range of motion (flexion or extension < 45°) or a history of an upper extremity condition that would affect wrist motion.

3. Experimental procedure

A detailed experimental procedure has been described previously (Xia et al., 2006). In summary, each subject was initially evaluated with the Motor Section (Part III) of the UPDRS (Fahn and Elton, 1987). Subjects were placed in a height-adjustable seat and the arm exhibiting more severity of rigidity was placed in the device via a manipulandum. With the shoulder and forearm in neutral position and the elbow in mid-flexion, the ulnar aspect of the subject's wrist was aligned with the center of rotation of the device and the forearm was stabilized with a vacuum bag splint preventing pronation and supination. The metacarpal restraints of the manipulandum restricted the motion of the wrist to flexion and extension.

Subjects were instructed to relax completely while the servomotor moved the wrist through a 60° range of motion. Each trial began at 30° of extension and moved to 30° of flexion then returned to 30° of extension at an angular velocity of 50°/s. Trials were conducted with and without a voluntary contralateral grip contraction equal to 20% of maximal grip force. Maximal grip force and contralateral contraction intensity were monitored using an instrumented hand dynamometer (Vernier Software & Technology, OR, USA) and LabView 2009 (National Instruments, TX, USA) which provided a graphic display of the contractile force. Subjects viewed the graph of grip contraction and matched their force to the visual display. Surface electromyography (EMG) signals were recorded from the skin overlying the bellies of wrist and finger flexors (flexors carpi radialis, flexor carpi ulnaris and flexor digitorum superficialis) and extensors (extensor carpi radialis, extensor carpi ulnaris and extensor digitorum communis) using a 16-channel surface EMG system (Delsys, Inc., MA, USA). Surface EMG electrode placements followed previously published recommendations (Perotto, 1994) and were confirmed via manual muscle testing. EMG signals were amplified (×10 k) and band-pass filtered (20–450 Hz) before being sampled at 1000 Hz for each EMG channel. Four trials were recorded under each experimental condition. EMG and torque signals were visually inspected after each trial to ensure that extrinsic wrist and hand musculature in the tested hand was electrically quiescent during Passive testing. Trials in which extrinsic muscle activation was observed or with a sudden increase in torque profile were discarded and repeated. All trials were followed by a period of rest to minimize fatigue.

Subjects were first tested after an overnight withdrawal of dopaminergic medication (OFF-MED) for at least 12 h (Jahanshahi et al., 2010) when the majority of the beneficial effects of dopaminergic therapy was eliminated (Defer et al., 1999). After the Off-Med tests were completed, the subject's regular dose was administered in the laboratory followed by a 30–60-min period of rest. Medication efficacy was validated verbally by the PD participants. After the rest period, the testing protocol was repeated in the on-medication (ON-MED) state.

Angular position of the wrist joint was measured using an emulated encoder output from the servomotor controller (SC904 series,

Table 1
Patients' clinical information.

Patient	Age (years)	Disease duration (years)	Sex	Arm tested	Rigidity (UPDRS)[a]		Medication[b]
					Off	On	
#1	62	5.5	F	L	3	2	R 1.0 mg (3×); S 5.0 mg (2×); C/L 25/100 (3×)
#2	74	2	F	L	2	1	C/L 25/100 (3×)
#3	71	5	F	R	2	1	C/L 25/100 (3×)
#4	55	10	M	L	3	1	R 1.0 mg (4×); C/L 25/250 (4×)
#5	57	13	M	L	3	3	Am 200 (3×); E 200 mg (3×); C/L 25/100 (3×)
#6	73	0.5	M	L	3	2	C/L 25/100 (3×)
#7	48	1.5	F	L	2	1	C/L 25/100 (3×); Pra 1.5 mg
#8	56	6.5	M	L	2	0	E 200 (3×); R 1 mg (3×); S 1.0 mg (1)
#9	63	12	M	R	2	0	Am 100 (1×); C/L 25/100 (2×)
#10	65	3	M	L	2	1	C/L 25/100 (3×)
#11	77	1	F	L	2	0	C/L 25/100 (3×)
#12	67	10	M	R	2	1	E 200 (4×); C/L 25/100 (4×)

[a] UPDRS (unified Parkinson's disease rating scale). Rigidity: 0, absent; 1, slight; 2, mild to moderate; 3, marked; 4, severe.
[b] Am: amantadine; E, entacopone; R, ropinirole; S, selegiline; Pra, pramipexole; C/L, carbidopa/levodopa.

Pacific Scientific, USA). The joint torque was measured using a strain gauge torque transducer (TRT-200, Transducer Techniques, USA). The position signal was sampled at 100 Hz while the torque signal was sampled at 1000 Hz. Data capture was controlled using LabView 2009 (National Instruments, TX, USA).

3.1. Data analysis

Customized software written in MatLab R2008a (Mathworks, MA, USA) was used to quantify rigidity of the wrist in healthy individuals as well as PD patients. To quantify rigidity, the torque signal was integrated with respect to joint angle (Nm deg) for a complete cycle of movement, including flexion and extension (Fung et al., 2000; Xia et al., 2006), referred to as rigidity score (Fig. 1). Flexion and extension movements were grouped together in order to more closely simulate a clinical assessment. Inertial components of the resistance torque, just after movement onset, were excluded from these analyses.

EMG signals were full-wave rectified and low-pass filtered with a cutoff frequency of 20 Hz. EMG signals for each muscle were averaged within the movement duration for flexion and extension, respectively, and then normalized to the background EMG activity by dividing by the mean EMG amplitude during the 100 ms prior to the onset of each movement. Mean normalized EMG signals were grouped by function (flexors and extensors) and represented by the sum of the EMG signals (i.e. Flexors = FCR + FCU + FDS; Extensors = ECR + ECU + EDC). The mean EMG of stretched muscles was calculated as the average of normalized EMG of the extensors during flexion and the averaged EMG of the flexors during extension (Xia et al., 2009).

In addition to stretch reflex muscle activity, EMG ratios were also calculated. The normalized EMG activity of stretched muscles was divided by the normalized EMG activity of shortened muscles during each movement. For example, during the imposed flexion movement the extensor muscles were stretched while the flexor muscles were shortened. Thus the normalized mean EMG in the stretched extensor muscles was divided by the normalized mean EMG in the shortened flexor muscles obtaining an EMG ratio for the flexion movement. EMG ratio has been previously used to characterize the interaction of the stretch reflex and shortening reaction and has been shown to be a robust index for characterizing parkinsonian rigidity (Meara and Cody, 1992; Xia et al., 2009).

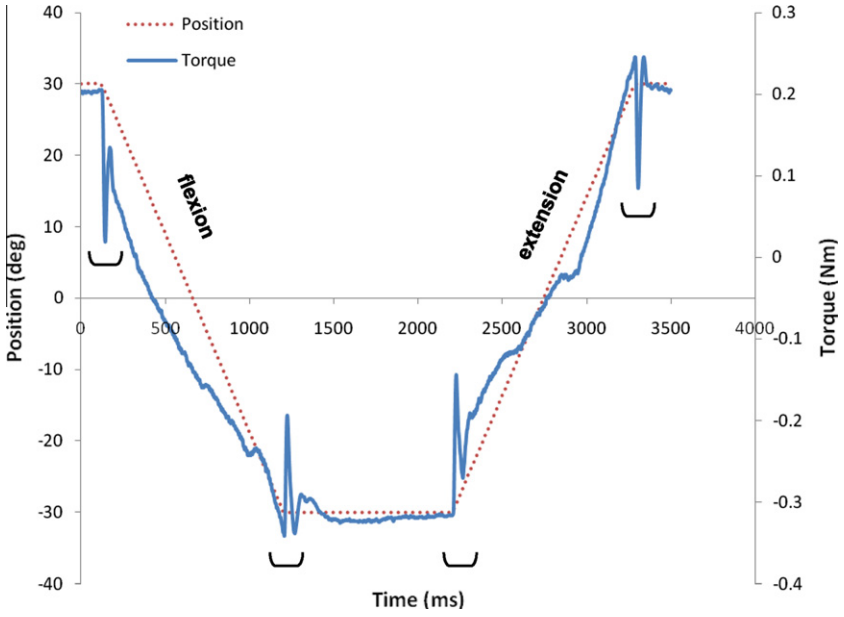

Fig. 1. A representative sample of joint position (dotted) and torque (solid) signals recorded during the imposed wrist flexion and wrist extension movements at 50°/s under Passive condition in a subject with PD. Rigidity scores were calculated for the period of flexion and extension movements without including the inertial components of torque indicated by brackets.

3.2. Statistical analyses

The calculated rigidity scores were analyzed using three repeated measures analyses of variance (ANOVA). A repeated measures ANOVA compared effect of contralateral contraction (Passive *vs.* Active) between the On- and Off-Medication states. Two further repeated measures ANOVAs compared the effect of contralateral contraction (Passive *vs.* Active) between the Control subjects and Off-Medication and On-Medication, respectively. The EMG response associated with the contralateral contraction was analyzed using three further repeated measures ANOVAs. Specifically, a repeated measures ANOVA compared the effect of contralateral contraction (Passive *vs.* Active) on mean EMG amplitude between the Off- and On-Medication states. Another two repeated measures ANOVAs compared the effect of contralateral contraction (Passive *vs.* Active) on mean EMG amplitude of control subjects and the Off- and On-medication states during flexion and extension. In the presence of a group by contraction interaction, a post hoc test was conducted using *t*-tests. For all statistical tests, differences were considered significant when $p < 0.05$. The statistical analysis was performed using SAS 9.2 (SAS Institute Inc., Cary, NC, USA).

4. Results

Fig. 2 shows joint position and EMG signals during the imposed extension movement and visualizes the stretch reflex and shortening reaction when a representative patient was tested in the Off-Med (A) and On-Med (B) conditions. In this representative example, the stretch reflex was observed in the wrist flexors, and the shortening reaction, defined as an anomalous muscular contraction in passively shortened muscles, was recorded in the wrist extensors. Both stretch reflex and shortening reaction were evident in the Off-Med condition. However, the phenomena were remarkably diminished with the administration of dopaminergic medication (On-Med).

Table 2 presents the results of EMG quantifications, including both EMG values of the stretched muscles and EMG ratios of stretched muscles over shortened muscles, obtained from all participants. The contralateral contraction was not associated with any changes in mean EMG amplitude of the stretched muscles (PD OFF-MED: $p = 0.262$; PD ON-MED: $p = 0.057$; control: $p = 0.154$). Furthermore, there was no effect of medication (PD OFF-MED *vs.* PD ON-MED: $p = 0.824$) on mean EMG amplitude. Results obtained from control subjects were not significantly different than those in the OFF-MED ($p = 0.723$) or ON-MED condition ($p = 0.571$). Similar to the mean EMG of the stretched muscles, no statistical differences in EMG ratio were associated with the contralateral contraction (PD OFF-MED: $p = 0.721$; PD ON-MED: $p = 0.598$; Control: $p = 0.864$). Medication did not alter the EMG ratio (OFF-MED vs. ON-MED: $p = 0.914$) and the controls were not significantly different than PD subjects in the OFF-MED ($p = 0.447$) or ON-MED condition ($p = 0.574$).

Table 2
Mean normalized EMG of the stretched musculature (EMGStretched) and the EMG ratio of stretched-shortened muscles during imposed flexion and extension movements with (Active) and without (Passive) a contralateral activation maneuver under both medication conditions in all PD and control subjects.

Group	EMGStretched		EMG ratio	
	Passive	Active	Passive	Active
PD OFF-MED	1.99 (0.18)	1.79 (0.41)	0.54 (0.07)	0.54 (0.14)
PD ON-MED	1.87 (0.37)	1.82 (0.41)	0.48 (0.17)	0.53 (0.13)
Control	2.03 (0.07)	1.88 (0.19)	0.55 (0.13)	0.58 (0.09)

There were no significant differences between the PD subjects and Controls as well as no effect of medication on mean EMG amplitude of the stretched muscles or EMG ratio during flexion and extension movements. Data shown in the table are mean (SD).

Torque resistance was elevated by the presence of contralateral activation (Active condition) as compared to the Passive condition. Fig. 3 illustrates torque–angle traces of the entire cycle of flexion and extension movements under both conditions when a subject with PD was tested in the Off-Med condition. There is an obvious difference in the contained area of torque–angle plots between the Passive and Active conditions.

The contralateral contraction was associated with increased rigidity score in patients as well as healthy controls ($F = 4.80$; $p = 0.036$; Fig. 4) at 50°/s (presented as mean ± SD: OFF-MED Pas-

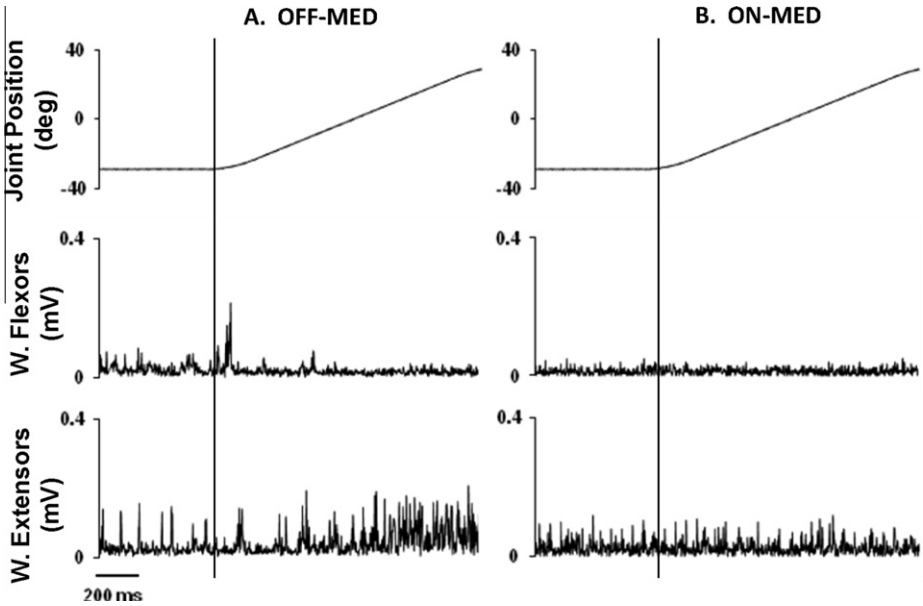

Fig. 2. Representative trials from a subject with PD showing EMGs of wrist flexors and extensors during Passive extension movement through a central 60° range of motion. In the OFF-MED condition (A), stretch reflex was observed in the wrist flexors and shortening reaction recorded in the wrist extensors. These phenomena were markedly reduced in the ON-MED condition (B). The vertical lines indicate where the movement onsets occur. Top panel: joint position (degree); middle panel: averaged EMG of flexor muscles; lower panel: averaged EMG of extensor muscles.

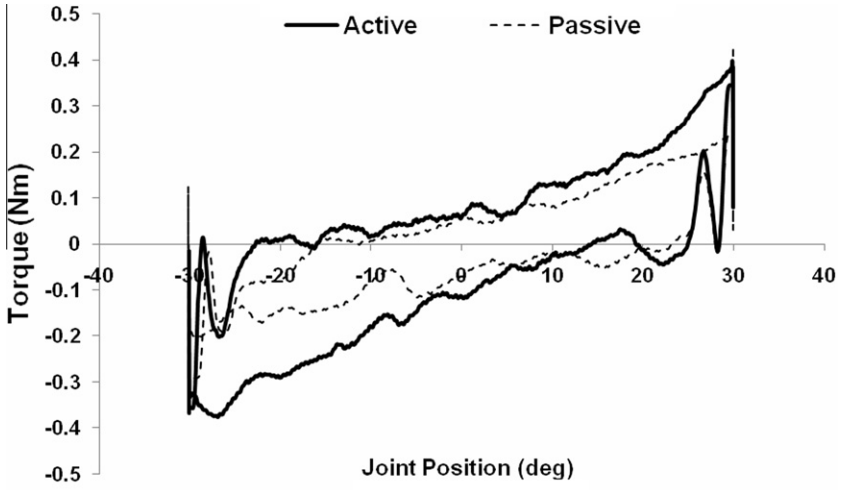

Fig. 3. Comparison of torque–angle traces between the Passive (dashed) and Active (solid) conditions illustrated in a subject with PD under the OFF-MED condition. The rigidity score, calculated as the integral of the torque with respect to position for the entire cycle of flexion and extension movements, was enhanced under the Active condition (i.e., the presence of a contralateral activation). Upper traces are associated with the extension movement and the lower ones with the flexion movement.

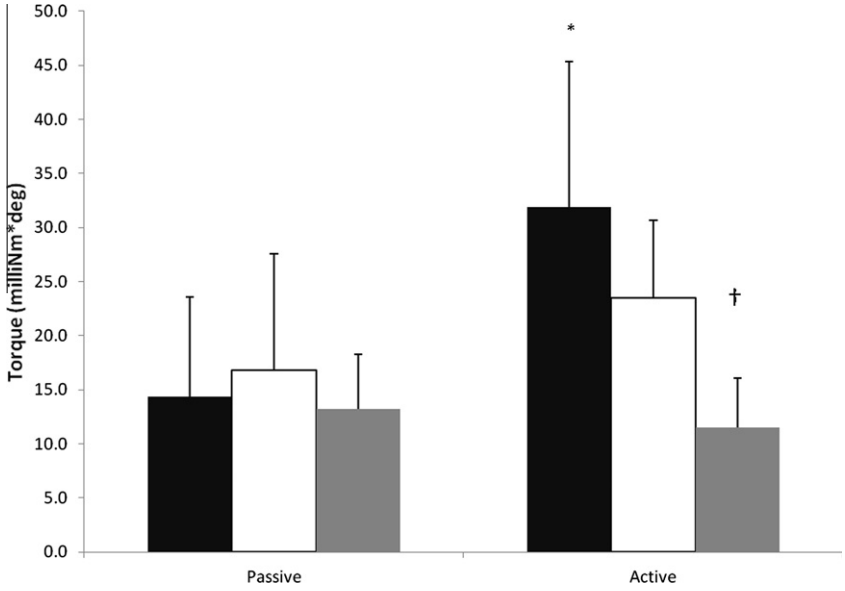

Fig. 4. Mean rigidity scores obtained from the patient group in both the OFF-MED (black) and ON-MED (white) states, and from the Control (gray) group under Passive and Active conditions. The rigidity score of the PD subjects in the OFF-MED state under the Active condition was significantly ($p = 0.007$) enhanced, compared to the PD subjects in the OFF-MED state under the Passive condition, as denoted by (∗), and was also significantly ($p = 0.004$) greater than Control subjects in the Active condition (denoted by †). Error bars are shown as standard deviation.

sive: 14.2 ± 9.2; OFF-MED Active: 24.9 ± 13.44; ON-MED Passive: 16.8 ± 10.8; ON-MED Active: 18.0 ± 7.2; CONTROL Passive: 13.6 ± 5.1; CONTROL Active: 12.2 ± 4.6). Specifically, the contralateral contraction was associated with a twofold increase in rigidity score compared to baseline when patients were in the OFF-MED state ($F = 9.36$; $p = 0.007$). The contralateral contraction was not associated with a statistically significant increase in rigidity score in patients when in the ON-MED state ($F = 0.10$; $p = 0.757$) or in healthy controls ($F = 0.08$; $p = 0.776$). Medication status was not associated with a significant decrease in rigidity score ($F = 1.08$; $p = 0.308$). The contralateral contraction was associated with a trend of increased rigidity score between medication states ($F = 3.92$; $p = 0.057$) as well as between the OFF-MED state and healthy controls ($F = 9.73$; $p = 0.004$). These differences were not present without the contralateral contraction (OFF-MED *vs*. ON-MED: $F = 0.45$; $p = 0.508$; OFF-MED *vs*. Control: $F = 3.15$; $p = 0.884$). The contralateral contraction did not produce differences between the ON-MED state and healthy controls (Passive: $F = 0.92$; $p = 0.344$; Active: $F = 2.40$; $p = 0.130$).

5. Discussion

A novel finding of the current study is the quantification of the disproportionate increase in rigidity associated with a concurrent gripping contraction in the contralateral limb. Though the assessment of parkinsonian rigidity during a contralateral activation maneuver has been included in the updated UPDRS, no previous research has addressed the magnitude of the associated change

in parkinsonian rigidity. In addition to this novel finding, the current study also presents findings that support previous research by demonstrating that patients with PD exhibit greater Passive rigidity than unimpaired subjects (Fung et al., 2000; Lee et al., 2002) and that parkinsonian rigidity is attenuated by dopaminergic interventions (Schapira et al., 2009). Previous research studies have investigated the mechanical and neurophysiological changes associated with contralateral activation and training (Hortobagyi et al., 1997; Zhou, 2000; Hortobagyi, 2005). It has been revealed that the crossed effect of ipsilateral activation is mediated by cortical, spinal and peripheral mechanisms (Delwaide and Pepin, 1991; Cramer et al., 1999; Muellbacher et al., 2000; Stinear et al., 2001; Hortobagyi, 2005).

Physiological mechanisms underlying the unique patterns of rigidity in PD, such as increased and uniform resistance, include abnormal responses to muscle stretch as well as the presence of the shortening reaction (Andrews et al., 1972; Berardelli et al., 1983; Xia and Rymer, 2004; Xia et al., 2009). It has been shown that patients with PD have an anomalous reaction in passively shortened muscles commonly referred to as the 'Westphal phenomenon' (Westphal, 1880) or 'shortening reaction' (Sherrington, 1909). The quantification of parkinsonian rigidity is determined by a resultant torque resistance, which is a summation of individual contributions of stretched muscles minus the contributions of shortened muscles (Xia et al., 2009). In the present study, patients with PD exhibited increased rigidity in the presence of a contralateral contraction, suggesting an increased contribution of the long-latency stretch reflex or a reduced contribution of the shortening reaction or a combination of both.

There are a few possible explanations for this observation. Firstly, the long-latency stretch reflex may well be mediated *via* a transcortical pathway (Capaday et al., 1991; Petersen et al., 1998; Lewis et al., 2004) although there was much debate as to its origin that spindle group II afferents play a pivotal role in the genesis of the delayed components (Matthews, 1984; Lourenco et al., 2006). A contralateral activation likely enhanced reflex responses to the stretch. Secondly, it has previously been demonstrated that H-reflex evoked in human leg muscle can be facilitated by the concurrent activation of other body part (such as making fists, or clenching teeth), i.e., a physiological phenomenon known as Jendrassik maneuver (Pasztor, 2004; Tuncer et al., 2007). A weak or missing reflex could be evoked by the presence of this "maneuver". In the present study, the effect of activation was explored in the homologous muscle groups. However, parkinsonian rigidity may also be enhanced if the activation maneuver was produced in the non-homologous muscles, as heel tapping of the contralateral limb is also used as an activation maneuver for assessment of rigidity (Goetz et al., 2008). There may be some common grounds in the underlying mechanisms between the effect of Jendrassik maneuver and the current investigation. Thirdly, previous research has revealed acute effects of contralateral contraction on unilateral motor neuron function (Hortobagyi et al., 2003). The investigators reported that H-reflex amplitude was progressively diminished with increasing intensity of contralateral muscle contraction. The results suggested that the diminished H-reflex amplitude with contralateral contraction might be the result of limb-specific increases in pre-synaptic inhibition of spinal afferent neurons diminishing the input of the muscle spindle to motor neurons. In the present study it is postulated that an increase in spinally modulated pre-synaptic inhibition associated with the contralateral contraction resulted in modulated Ib afferent input and a diminished shortening reaction within the passively flexed musculature of the wrist.

The findings of the current study also demonstrated a medication-related reduction of parkinsonian rigidity with a contralateral contraction. These results support previous research findings that suggest the crossed-effects of muscle contraction are partially mediated by a cortical component (Muellbacher et al., 2000; Stinear et al., 2001; Hortobagyi, 2005). Dopaminergic medication supports the release of dopamine and affect several cortical pathways including the nigrostriatal pathway which is involved in the production of coordinated movement (Benabid et al., 2009; Schapira et al., 2009). Dopaminergic medication has been shown to modulate voltage-gated sodium, potassium and calcium channels which directly alter neostriatal neuronal excitability (Tepper et al., 2007). Furthermore, research has demonstrated that a contralateral contraction alters motor cortical excitability (Cramer et al., 1999; Muellbacher et al., 2000; Hortobagyi et al., 2003). It is widely believed that patients with rigidity have difficulty in remaining relaxed to a certain extent. Magnitude of the stretch reflex is broadly correlated with the background muscle activity level, which is usually decreased by the dopaminergic medication therapy.

Quantification of muscle activities showed no differences in normalized EMG data between the Passive and Active conditions and between the Off and On-medication states. Considering a few reasons, these observations may not be surprising. In this study, the mean EMG amplitude within the movement duration was normalized to the background EMG activity. The movement duration lasted 1200 ms (in the range of 60°; velocity at 50°/s) whereas duration of the long-latency stretch-reflex is usually for 70–90 ms in forearm muscles. In addition, patients with parkinsonian rigidity exhibit increased background EMG activity (Burke et al., 1977; Marsden, 1982). Given the aforementioned situation, the normalized EMG values are not expected to reflect changes in rigidity assessed by torque resistance due to the effect of contralateral activation. Under the influence of medication therapy, both the reflex EMG activity and the background EMG level are reduced. This potentially explains why the normalized EMG data was not significantly reduced in the On-medication state. Moreover, reflex EMG activity contributes to the neural component of rigidity. Previous study suggested that visco-elastic properties of muscle also contribute to rigidity, in addition to enhanced EMG responses (Dietz et al., 1981).

The findings of the present study show that dopaminergic medication reduces the measured rigidity associated with PD, including in the presence of the contralateral contraction. Several mechanisms may propagate the observed decrease in parkinsonian rigidity. It has been shown that dopaminergic medication improves connectivity within the motor cortex and transcortical pathways (Jahanshahi et al., 2010). Previous research has revealed that dopaminergic medication reduces excessive inhibitory output by the basal ganglia and increases activation coupling of movement related centers (Jahanshahi et al., 2010). Furthermore, dopaminergic medication has been shown to improve both direct and indirect striatal pathways promoting movement coordination (Kreitzer and Malenka, 2008; Zhou et al., 2009; Zhou, 2010) and reducing aberrant reflex activity including the shortening reaction and stretch reflex.

The current research findings have clinical implications in diagnosis and treatment. These data reveal that rigidity is disproportionately increased in the presence of a contralateral contraction and the increased rigidity is exacerbated in the absence of dopaminergic medication. The current study provides novel insight into the effect of contralateral contractions on parkinsonian rigidity however it has some limitations. This study did not directly investigate the underlying mechanisms of the increased rigidity which may include any combination of segmental, spinal and supraspinal components. Future research should address these components in patients with PD as previous research has focused on healthy individuals. We acknowledge that a small sample size is a limitation of the current study and an expanded subject population would better generalize our findings.

6. Conclusions

The current study is the first to have investigated and quantified the effect of a contralateral activation maneuver on parkinsonian rigidity. The findings of the current study show that a contralateral contraction increases ipsilateral wrist rigidity. The activation maneuver for assessing rigidity is included in the updated MDS-UPDRS (Goetz et al., 2008) and may aid in earlier detection of Parkinson's disease. These data also provide a foundation for further exploration of the mechanisms by which an acute contraction results in altered contralateral neuromuscular function in people with neuro-pathology.

7. Financial disclosure

None.

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