Effect of Medication on EMG Patterns in Individuals with Parkinson’s Disease

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Abstract: Individuals with Parkinson’s disease show dramatic improvements in their ability to move when medicated. However, the neural cause of this improvement is unclear. One hypothesis is that neural activation patterns, as measured by surface electromyography (EMG), are normalized by medication. We tested this hypothesis by investigating the effect of medication on the electromyographic (EMG) patterns recorded when individuals with idiopathic Parkinson’s disease performed elbow flexion movements over three movement distances while off and on antiparkinsonian medication. When the subjects were off medication, they lacked the ability to modulate the agonist EMG burst duration with changes in movement distance. The ability to modulate agonist EMG burst duration is characteristic of the EMG patterns observed in healthy subjects. Also, multiple agonist bursts were exhibited during the acceleration phase. As expected, medication diminished the clinical signs of Parkinson’s disease, increased movement speed, and increased the magnitude of the first agonist burst. Medication did not restore agonist burst duration modulation with movement distance, did not change the frequency of agonist bursting, and did not alter the timing of the antagonist activation. These results show that medication does not alter the temporal profile of EMG activation. © 2002 Movement Disorder Society

Key words: Parkinson’s disease; medication; EMG patterns; movement control

The dramatic clinical effect of antiparkinsonian medication on reducing motor impairment in subjects with Parkinson’s disease is clear. However, the specific neural changes caused by medication that lead to the improvement in motor function are not clear. Motor control testing has been used to document improvement in motor performance along with associated changes in electromyographic (EMG) parameters during isometric contractions, movement tasks, and postural control. Moreover, although some authors have observed that medication causes EMG patterns during rapid point-to-point movements to become qualitatively similar to those of normal subjects, to our knowledge, the hypothesis that medication normalizes abnormal EMG patterns in the control of movement distance has not been explicitly tested.

In contrast to the EMG pattern seen in individuals with Parkinson’s disease, the EMG pattern seen in healthy individuals making point-to-point movements has been clearly described. Rapid movements are made with a biphasic or triphasic pattern of muscle activation characterized by an initial agonist burst, an antagonist burst, and frequently a second agonist burst. Increases in movement distance are accomplished by increasing the magnitude and duration of the initial agonist burst, while the timing of the antagonist burst is increasingly delayed. Buchman and colleagues compared the EMG pattern for older (mean age, 69 years) and young (mean age, 26 years) healthy individuals during rapid elbow flexion movements across 36, 54, and 72 degree movement distances. They noted that age and gender did not affect the following EMG parameters: the initial
rate of rise of the agonist EMG, magnitude of the first agonist burst, magnitude of the antagonist burst, and the timing of the main antagonist burst. Furthermore, even though not reported, their Figure 2 clearly shows that agonist burst duration systematically increases with increases in movement distance.\textsuperscript{12}

Differences have been identified between the EMG patterns of unmedicated subjects with Parkinson’s disease and healthy subjects during rapid, single-joint, point-to-point movements. For example, Baroni and associates demonstrated that in all untreated parkinsonian individuals, multiple agonist bursts of low intensity were present, while observing that the agonist burst frequency was similar to tremor at rest. They noted the presence of multiple agonist bursts in individuals who exhibited no resting tremor.\textsuperscript{7} Furthermore, Pfann and coworkers demonstrated that, in rapid elbow movements performed across increasing movement distances (36, 54, and 72 degrees), there was impaired agonist burst duration modulation in individuals with mild to moderate Parkinson’s disease.\textsuperscript{16}

Anti-parkinsonian medication has been shown to lead to an increase in peak velocity (or decrease in movement time),\textsuperscript{7–9} fewer agonist EMG bursts,\textsuperscript{7,9} agonist EMG bursts of greater magnitude,\textsuperscript{7,6} no change in agonist burst duration,\textsuperscript{9} and decreased antagonist activity during movement initiation\textsuperscript{7} during point-to-point movements. Furthermore, Baroni and colleagues concluded that the EMG patterns for parkinsonian patients on medication performing arm abduction movements are similar to those observed in normal subjects.\textsuperscript{7}

Although studies have shown that medication increases the amplitude of the first agonist burst, it remains to be established whether or not medication normalizes the temporal pattern of the EMG across distances. One study, in which the effect of medication on the temporal EMG pattern was examined during wrist flexion movements over distances from 15 to 60 degrees, found qualitatively similar modulation of EMG patterns both off and on medication.\textsuperscript{9} However, even off medication, the subjects with Parkinson’s disease in that study did not exhibit any deficit in prolonging the agonist burst with increased movement distance, a deficit that has been found in other studies (elbow flexion movements from 5 to 72 degrees).\textsuperscript{16} As a consequence, the effect of medication has not been studied in a group of subjects with Parkinson’s disease whose pattern of EMG modulation during the early part of the movement exhibit clear qualitative differences from those of healthy subjects performing rapid single-joint movements.

The purpose of this study was to determine whether antiparkinsonian medication is associated with a normalization of the neural control of volitional movement. Subjects were asked to perform rapid, single-joint elbow flexion movements over three movement distances (36, 54, and 72 degrees). This task was chosen because the EMG patterns for rapid single-joint elbow movements are well understood in neurologically healthy subjects\textsuperscript{12–15} and because there are known deficits in the modulation of the EMG pattern with movement distance during these movements in subjects with Parkinson’s disease.\textsuperscript{16} Therefore, specific changes in neural control can be predicted if medication is normalizing the EMG pattern in movements of different distances.

**METHODS AND MATERIALS**

**Subjects**

Eight subjects with Parkinson’s disease were tested according to University-approved protocols. Eligibility for inclusion in the study was restricted to subjects who (1) had a diagnosis of idiopathic Parkinson’s disease,\textsuperscript{17} (2) were taking levodopa, (3) had no other known neurological disorder as determined by history, and (4) had no known injury or other disease that might interfere with motor function. Table 1 describes the individual subject information. The most impaired limb of the subject was tested. Five subjects used their right arm and

<table>
<thead>
<tr>
<th>Subject no.</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>Disease duration (yr)</th>
<th>Motor UPDRS</th>
<th>Hoehn &amp; Yahr</th>
<th>Dyskinesias</th>
</tr>
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<td>Off</td>
<td>On</td>
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<td>1</td>
<td>65</td>
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<td>8</td>
<td>19</td>
<td>20</td>
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<td>2</td>
<td>75</td>
<td>M</td>
<td>20</td>
<td>46</td>
<td>12</td>
<td>IV</td>
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<td>3</td>
<td>71</td>
<td>M</td>
<td>16</td>
<td>37</td>
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<td>4</td>
<td>66</td>
<td>M</td>
<td>12</td>
<td>34</td>
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<td>5</td>
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<td>8</td>
<td>57</td>
<td>M</td>
<td>2</td>
<td>21</td>
<td>19</td>
<td>III</td>
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</table>

L–C, levodopa–carbidopa; Per., pergolide; Sel., selegiline HCl; Bro., bromocriptine; Pra., pramipexole; UPDRS, Unified Parkinson’s Disease Rating Scale.
three used their left arm when performing the movement task.

**Experimental Protocol**

All subjects underwent a clinical evaluation, which consisted of the motor (part III) subclass of the Unified Parkinson’s Disease Rating Scale (UPDRS),\(^{18}\) classification according to the Hoehn and Yahr scale,\(^{19}\) and dyskinesia ratings, which was followed by motor control testing. The protocol was first performed by subjects off medication (12-hour overnight withdrawal of antiparkinsonian medications) and then performed on medication after intake of the subject’s regular medication was resumed. The on trials were performed from 45 minutes to 1.5 hours after medication intake. The variable time period was due to the fact that patients respond differently to the effects of their medication. All subjects were tested in the morning to minimize the effect of diurnal variation.

**Experimental Setup**

Subjects were seated with their arm abducted 90 degrees. The forearm was strapped to a rigid, lightweight manipulandum that could freely rotate only in the horizontal plane. The axis of rotation was aligned with the elbow. Subjects viewed a computer monitor that displayed a vertical cursor (equivalent to 1 degree of elbow angular displacement), which corresponded to the angle of the elbow joint. A broad marker was located as a target at the desired angular distance. The width of the broad marker corresponded to 6 degrees of angular elbow rotation. For the right elbow, full extension was defined as 90 degrees; elbow flexion was in the negative direction. The initial position for the experiments was 35 degrees. For the left limb, full extension was defined as −90 degrees; elbow flexion was in the positive direction. Joint angle was measured by a capacitative transducer mounted on a shaft at the axis of rotation. Joint angle was digitally differentiated to generate joint velocity. Joint acceleration was measured by a piezoresistive accelerometer mounted 47.6 cm from the center of rotation. Surface electrodes were used to record electromyograms (EMGs) from biceps and the triceps (lateral head). The EMG signals were amplified (gain 1,600) and band-pass filtered (60–300 Hz). All signals were digitized at 1,000 Hz with 12-bit resolution. Data were collected for 2 seconds.

**Task**

Subjects were asked to perform elbow flexion movements over three distances (36, 54, 72 degrees) “as fast as possible.” The order of target presentation was randomized across subjects. A tone signaled the subject to initiate the movement. The cessation of the tone 2 seconds later signaled the end of the trial, at which time the subject returned to the initial position.

At the start of each test session, subjects performed 30 practice trials at the first distance to be tested. Before testing the two subsequent distances, subjects performed 10 practice trials. Ten consecutive trials were recorded at each distance.

**Data Analysis**

Data processing was performed offline. The angle and acceleration traces were low-pass filtered with a second-order Butterworth filter with a cut-off frequency of 20 Hz. The EMG signals were full-wave rectified. The onset and offset of the agonist bursts were marked based on visual inspection, and the number of agonist bursts before the time of peak velocity were counted. The onset and cessation of the agonist bursts was determined by visual inspection as the time at which the EMG signal went above and then returned to baseline, respectively. Trials were rejected for the following reasons: (1) technical problems (e.g., signal lost), (2) movement not completed in the allotted time because movement initiation was dramatically delayed; (3) movement initiated in the wrong direction, (4) movement not initiated from the correct initial position, (5) movement terminated outside of the 6 degree target, and (6) inability to determine the agonist offset. With the exception of data from Subject 2, 9% of the off and 5% of on medication trials were rejected. When off medication, Subject 2 exhibited such low levels of agonist activity that bursts could not be consistently marked; therefore, the data for Subject 2 were rejected for all measures that involved the calculation of agonist burst duration.

The following dependent measures were calculated from the individual trials during movement tasks. The mean of each parameter for each subject was used for further analysis, except for the duration of the first agonist burst in which the median was used. The median response was used, since this measure of central tendency is less affected by extreme measures.

1. **Peak velocity**: the maximum absolute value of the velocity signal.

2. **Duration of the first agonist burst**: the time difference between the visually marked onset and offset of the first agonist burst.

3. **Time to peak velocity**: the time period from where the absolute value of the velocity signal exceeds 5% of peak velocity until the maximum value of the velocity signal.

4. **Number of agonist bursts during the acceleration...
phase: the number of bursts in the agonist EMG before the time of peak velocity.

(5) Frequency of agonist bursting during the acceleration phase: the number of agonist bursts during the acceleration phase divided by the time to peak velocity.

(6) $Q_{ag,1}/T$: magnitude of the first agonist burst: the integral of the agonist EMG during the first agonist burst divided by the duration of the first agonist burst. This parameter characterizes the average magnitude of the first agonist burst.

(7) $Q_{ag,2}/T$: magnitude of the second agonist burst: the integral of the agonist EMG during the second agonist burst divided by the duration of the second agonist burst. This parameter characterizes the average magnitude of the second agonist burst. This burst (if present) represents agonist burst activity that occurs after the initial burst but before peak velocity. This parameter is thought, like the first agonist burst, to be involved with movement initiation.

(8) $Q_{ratio}$: the ratio of the area of the first agonist burst to the area of the antagonist EMG during the time interval of the first agonist burst. This parameter characterizes the ratio of agonist to antagonist EMG activity during the initiation of the movement.

(9) $Q_{ant}$: area of the antagonist EMG to the end of the movement: the integral of the antagonist EMG from the time of agonist onset to the end of the movement (where the time of the end of the movement is defined as the time at which the value of the velocity signal drops to 5% of peak velocity).

(10) $Q_{ant}/mt$: average magnitude of the antagonist EMG: the integral of the antagonist activity from the marked agonist onset to the end of the movement divided by the duration of the integration interval (where the time of the end of the movement is defined as the time at which the absolute value of the velocity signal drops to 5% of peak velocity). This measure reflects the average magnitude of antagonist activity during the movement.

Statistical Analysis

The data were analyzed using one- and two-way repeated measures analysis of variance. All levels of significance were designated at $P \leq 0.05$. The independent variables were medication and target distance while the dependent measures were the UPDRS along with the measures (1-10) delineated above. As previously mentioned, the data from Subject 2 were excluded from all analyses involving agonist burst duration.

RESULTS

Motor UPDRS Scores

There was a statistically significant effect of medication on the motor (part III) subsection of the UPDRS scores ($F(1,7) = 15.06; P = 0.006$). The mean off medication UPDRS score was 25.8 and dropped to 13.6 when subjects were medicated. Our subjects showed similar changes between their off and on medication UPDRS scores, when compared with previous studies.5,20 Because medication had a significant clinical effect on motor performance, this group was appropriate to study the effect of antiparkinsonian medication on the neural control patterns during motor tasks (see Table 1). Even though two subjects (S1 and S8) demonstrated little or no clinical improvement, they did demonstrate improvements in their movement speed. As such, we included their data in the analysis. We did perform the analyses excluding these subjects but the results were the same.

Time Series Patterns for Movements over Three Different Distances

In Figure 1, angle, velocity, acceleration, agonist, and antagonist EMG signals are shown for movements over three distances for a representative subject with Parkinson’s disease both off (Fig. 1a) and on (Fig. 1b) antiparkinsonian medication. Similar to that seen in healthy subjects (as shown for comparison in Fig. 1c), peak velocity and the magnitude of the first agonist burst increased with movement distance in subjects with Parkinson’s disease both off and on medication. However, the subject with Parkinson’s disease achieved lower peak velocities than the healthy subject. In contrast to that seen in the healthy subject, the agonist EMG signal consists of agonist bursts of fixed duration where the number, not duration, of the agonist bursts during the acceleration phase increases with movement distance. Furthermore, the onset of the antagonist EMG occurs nearly simultaneously with the onset of the first agonist EMG burst and is independent of movement distance. Seven of eight subjects in this study had antagonist EMG patterns that differed in magnitude and/or temporal profile from that characteristic of healthy subjects. Moreover, the acceleration traces in the subject with Parkinson’s disease show multiple peaks during the acceleration phase instead of a smooth single-peaked signal as seen in the healthy subject. This finding is most clearly seen off medication in Figure 1a.

Comparing Figure 1a and 1b, one can see that the modulation of EMG and kinematic signals with movement distance is qualitatively similar off and on medication. However, for movements at a given distance, peak velocity is greater, the number of agonist bursts during the acceleration phase is lower, and agonist magnitude is greater in the on medication condition compared with the off medication condition. At the same time, the temporal...
patterning of the agonist and antagonist EMG signals does not change with medication.

Kinematic Parameters: Peak Velocity During Movements over Three Distances

Peak velocity was analyzed to confirm that movements were faster in the on medication condition. Figure 2 shows group mean peak velocities for each target distance. The figure shows that peak velocity increases with both distance and medication. These were statistically significant main effects (Table 2). The time to peak velocity occurred later for the longer movements (both off and on medication) and was earlier when the subjects were medicated (Table 2).

EMG Parameters During Movements over Three Distances

Duration Modulation of the First Agonist Burst

We analyzed the duration of the first agonist burst to test whether or not agonist EMG duration scaled with
movement distance and whether medication exhibited any influence on agonist duration modulation. Figure 3 shows the group means of the duration of the first agonist burst for each target distance. There was no suggestion of duration modulation with changes in target distance (Table 2). In addition, there was no medication effect on duration modulation (Table 2). The figure clearly shows that medication does not reverse the loss of duration modulation with changes in target distance. The ANOVA results show no medication effect on agonist duration modulation. Figures 3 and 4 show the group means of the duration of the first agonist burst for each target distance. There was no suggestion of any influence on agonist duration modulation. Figure 3 shows the group means of the duration of the first agonist burst for each target distance. Figure 4 shows the linear relationship between time to peak velocity and number of agonist bursts (r^2 = 0.98) for 36, 54, and 72 degree movements. There are fewer bursts on medication, and these movements have shorter times to peak velocity. These findings suggest that changes in the time to peak velocity, not medication or distance, is the most influential factor in determining the number of agonist bursts. In accordance with this finding, we chose to analyze the frequency of agonist bursting to test whether or not the number of agonist bursts during the acceleration phase and time to peak velocity. Additional analysis showed a strong linear relationship (r^2 = 0.98) between the absolute number of bursts during the acceleration phase and time to peak velocity.

**FIG. 3.** Group mean duration of the first agonist burst. Duration of the first agonist burst for 36, 54, and 72 degree movements is shown for the off medication (circles, dashed line) and on medication (squares, solid line) conditions with standard error bars. Duration of the first agonist burst decreases with both decreases in distance and medication.

**FIG. 4.** Linear relationship between number of agonist bursts during the acceleration phase and the time to peak velocity. The number of agonist bursts during the acceleration phase (time to peak velocity) for 36, 54, and 72 degree movements is shown for the off medication (circles) and on medication (squares) conditions. There is a strong linear relationship (r^2 = 0.98) between the absolute number of bursts during the acceleration phase and time to peak velocity. Additionally, the number of agonist bursts during the acceleration phase decreases with both decreases in distance and medication.

**TABLE 2. ANOVA results: Movement task measures**

<table>
<thead>
<tr>
<th>Dependent measures</th>
<th>Medication</th>
<th>Distance</th>
<th>Medication × distance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak velocity</td>
<td>F(1,7) = 25.17</td>
<td>F(2,14) = 38.00</td>
<td>F(2,14) = 1.26</td>
</tr>
<tr>
<td>Time to peak velocity</td>
<td>F(1,6) = 9.3</td>
<td>F(2,12) = 12.06</td>
<td>F(2,12) = 0.51</td>
</tr>
<tr>
<td>Duration of the 1st agonist burst</td>
<td>F(1,6) = 1.38</td>
<td>F(2,12) = 0.81</td>
<td>F(2,12) = 1.51</td>
</tr>
<tr>
<td>Number of agonist bursts during the acceleration phase</td>
<td>F(1,6) = 2.68</td>
<td>F(2,12) = 5.94</td>
<td>F(2,12) = 1.32</td>
</tr>
<tr>
<td>Frequency of agonist bursting during the acceleration phase</td>
<td>F(1,6) = 0.15</td>
<td>F(2,12) = 0.01</td>
<td>F(2,12) = 0.30</td>
</tr>
<tr>
<td>Qag1/T, magnitude of 1st agonist burst</td>
<td>F(1,6) = 9.32</td>
<td>F(2,12) = 6.63</td>
<td>F(2,12) = 2.22</td>
</tr>
<tr>
<td>Qag2/T2, magnitude of 2nd agonist burst</td>
<td>F(1,6) = 7.40</td>
<td>F(2,12) = 3.96</td>
<td>F(2,12) = 1.53</td>
</tr>
<tr>
<td>Qratio, ratio of agonist and antagonist area during 1st agonist burst</td>
<td>F(1,6) = 8.40</td>
<td>F(2,12) = 2.53</td>
<td>F(2,12) = 0.84</td>
</tr>
<tr>
<td>Qant, area of antagonist EMG until the end of movement</td>
<td>F(1,7) = 0.19</td>
<td>F(2,14) = 6.8</td>
<td>F(2,14) = 0.83</td>
</tr>
<tr>
<td>Qanttime, average antagonist EMG until the end of movement</td>
<td>F(1,7) = 0.75</td>
<td>F(2,14) = 5.58</td>
<td>F(2,14) = 0.48</td>
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</table>

ANOVA, analysis of variance.
*Statistically significant, P ≤ 0.05.
pattern of bursts was directly related to the time period studied. When the number of bursts were normalized by the time to peak velocity (i.e., the frequency of bursting), neither the effect of distance nor medication approached statistical significance (Table 2, Figure 5). As shown in Figure 5, regardless of medication, agonist bursts occur at a fixed frequency (approximately 9 Hz).

Magnitude of the First and Second Agonist Bursts, $Q_{ag1/T}$ and $Q_{ag2/T2}$

We analyzed $Q_{ag1/T}$ and $Q_{ag2/T2}$ to test whether or not medication changes the magnitude of both of the agonist bursts. Figure 6 shows that the magnitude of the first and second agonist bursts scaled with distance and that medication was associated with greater burst magnitudes. The distance and medication effects were statistically significant (Table 2).

Ratio of EMG Signals During the First Agonist Burst, $Q_{ratio}$

We analyzed $Q_{ratio}$ (ratio of agonist to antagonist EMG activity during the first agonist burst) to test whether or not medication changes the relative amount of coactivation during the control of movement initiation. As shown in Figure 7, there was a significant effect of medication on $Q_{ratio}$ (Table 2), which shows that there is relatively greater agonist compared to antagonist activity on medication. There was no effect of movement distance.

We further analyzed the data to determine whether the medication effect was due to changes in agonist and/or antagonist EMG activity. As previously established, there was a significant effect of medication and distance on initial agonist EMG activity ($Q_{ag1/T}$), with greater activity associated with the on medication condition and with increases in movement distance. Furthermore, an analysis of the antagonist EMG activity during this same time period (time period of the first agonist burst) showed no significant main effects for either distance ($F_{2,12} = 1.22; P = 0.32$) or medication ($F_{1,6} = 0.198; P = 0.67$). Therefore, the changes in the $Q_{ratio}$ with respect to medication, were primarily due to changes in agonist and not antagonist EMG activity.
Measures of Antagonist Activity: Area of the Antagonist EMG to the End of the Movement, $Q_{\text{ant}}$; Average Antagonist EMG to the End of the Movement, $Q_{\text{ant/mt}}$

We analyzed $Q_{\text{ant}}$ and $Q_{\text{ant/mt}}$ to test whether medication changed the overall antagonist activation and average antagonist activation for the entire movement. There was a significant distance effect with both measures of antagonist EMG activity. However, medication had no statistically significant effect on $Q_{\text{ant}}$ or $Q_{\text{ant/mt}}$ (Table 2). As shown in Figure 8, this finding occurred because medication did not have a consistent effect on the magnitude of the antagonist signal across subjects. In general, medication did not change the temporal pattern of the antagonist EMG with respect to the timing of the agonist EMG. (Note that, in Subject 2, medication did lead to some bursting in the antagonist where no bursting and very little activation was observed when Subject 2 was off medication.)

DISCUSSION

We have shown clear changes in motor performance and EMG measures with the use of antiparkinsonian medication during movements over increasing distances in subjects with Parkinson’s disease. We confirmed previous findings that the use of medication leads to improvement in motor function, including a decreased clinical rating of motor impairment (Table 1)\textsuperscript{6,20} and

![Group Mean Diagram](image-url)

**FIG. 8.** Antagonist electromyography (EMG) measures. Area of the antagonist EMG until the end of movement ($Q_{\text{ant}}$) is shown in the left panels (a,c), and the average antagonist EMG during the movement ($Q_{\text{ant/mt}}$) is shown on the right panels (b,d). Group mean measures for all three distances are shown in the upper panels (a,b), and the individual subject means for the 72 degree movements are shown in the bottom panels (c,d). For each subplot, the measure is shown for the off medication condition (shaded bars) and the on medication condition (hatched bars). Medication had no statistically significant effect on $Q_{\text{ant}}$ or $Q_{\text{ant/mt}}$.

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faster movements (Figs. 1a,b, 2). Because medication had a clear effect on motor performance, this was an appropriate group in which to study the effect of antiparkinsonian medication on the modulation of the EMG pattern during movements over increasing distances. It is important to note that the order of the medication condition was not randomly assigned. This strategy was deliberate for the purpose of ensuring optimum electrode placement between the medication conditions. Therefore, the off medication state was tested first, which allowed all testing to be done on the same day. This raises the question as to whether or not there might be a practice effect. We reduced the likelihood of a practice effect by giving the subjects 30 practice trials prior to testing to ensure that the task itself was well understood. Additionally, if a practice effect was present, then the likelihood of reporting an effect of medication would increase. This is contrary to the findings of no change in either agonist burst duration or the frequency of agonist bursting.

Medication Results in Neural Compensation but Not Normalization of the EMG Pattern

If antiparkinsonian medication helps to normalize movement by normalizing the underlying neural control patterns, we would expect to see a single agonist EMG burst in which there is a systematic increase in agonist EMG magnitude and duration with increasing movement distance during the acceleration phase. In addition, we would expect to observe a clear antagonist burst that is increasingly delayed with increasing movement distance and that occurs after the first agonist burst. There is a clear effect of dopaminergic medication on the magnitude of the agonist bursts during the acceleration phase. However, medication does not result in any shift toward systematic modulation of the duration of the first agonist burst with increasing movement distance. In addition, there are multiple agonist bursts of muscle activation with the bursting frequency remaining unaltered by medication (Fig. 5). Moreover, medication does not affect the timing of the antagonist EMG burst relative to the first agonist burst, and it has an inconsistent effect on the antagonist EMG measures across subjects. Medication appears to elicit its effect on movement speed by leading to an increase in agonist EMG magnitude and a concomitant increase in the Q<sub>ratio</sub> during movement initiation.

Our findings also showed that medication does not eliminate the presence of multiple agonist bursts. This bursting activity on medication has also been reported in studies conducted at the elbow and shoulder muscles in individuals with Parkinson’s disease during rapid movements. A study by Brown and coworkers on elbow extensor muscles also showed that antiparkinsonian medication did not alter the frequency or absolute amplitude of the 8 to 12 Hz action tremor. Furthermore, they demonstrated that medication increased the magnitude of the agonist (triceps) EMG activity. Our results are consistent with this finding in that medication increased the magnitude of the agonist EMG, leaving the frequency of the action tremor unaltered. Therefore, we suggest that antiparkinsonian medication does not alter the frequency of the postulated neuronal oscillator, and this may have limited our subject’s ability to modulate agonist burst duration with movement distance.

We conclude that the inability to prolong agonist EMG burst duration and delay the timing of the antagonist EMG for longer movements are consequences of changes in how the circuitry of the basal ganglia functions to modify parameters of movement in individuals with Parkinson’s disease. Our findings are consistent with other studies that we have conducted in which both
stimulation of the VIM nucleus and pallidotomy have failed to normalize the EMG patterns associated with the control of movement distance in individuals with Parkinson’s disease.\textsuperscript{25,26} Furthermore, levodopa has been shown to only partially reverse and normalize cortical motor functions.\textsuperscript{27}

In conclusion, antiparkinsonian medication does not reinstate burst duration modulation and, therefore, one of the mechanisms used to generate force in rapid movements is lost in the parkinsonian nervous system. Antiparkinsonian medication leads to a change in the magnitude of the EMG waveform but it does not normalize modulation of the EMG pattern with changes in movement distance. The partial failure of dopaminergic medication to normalize function of the basal ganglia could be attributed to the way exogenous dopamine is used by the remaining neurons, or it may be due to fundamental changes in the organization of the basal ganglia–thalamo-cortical circuit in Parkinson’s disease.

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