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# Voluntary control of facial musculature in Parkinson's disease

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ABSTRACT

Aside from being measured in the context of producing facial expressions of emotion, the ability to voluntarily control a range of facial muscles in Parkinson's disease (PD) has not been systematically measured. We used in three enrollment phases an adaptation of the Upper and Lower Face Apraxia test, a measure of the ability to make voluntary movements of the upper and lower face in PD patients and healthy controls. Errors were scored due to (1) pauses prior to movement initiation, (2) loss of individuation, (3) impoverished movement, (4) no movement at all, or (5) content errors (likened to ideational apraxia errors). The results show impaired voluntary control of facial musculature in most but not all with PD (with large effect sizes) which correlated positively and highly with disease severity. Errors by PD patients were predominantly due to impoverished movement and individuation loss whereas those made by controls were predominantly due to individuation loss. Patients committed more errors than controls due to impoverishment and no movement, with negligible differences between groups in other errors. In summary, similarly to spontaneous and voluntary emotional expressions, voluntary non-emotional facial movements are impoverished in PD; impoverishment of all movement types will likely contribute to the mask-like facial appearance that is seen with disease progression. These findings also illustrate the utility of an adapted Face Apraxia test as a practical and sensitive measure of voluntary facial musculature control in PD. The test can be used to supplement clinical observations and as a research tool.



## 1. Introduction

A "masked" expressionless face is an important clinical sign of Parkinson's disease (PD) [3,13,21]. In a now classic and influential review on facial expression, Rinn [21] proposed that the impression of a mask-like face in PD originates from reduced spontaneous emotional expression, with voluntary emotional expression remaining intact. Following Rinn's proposal, studies found that PD patients were impaired in spontaneous emotional expressions during conversation or in response to emotionally evocative stimuli [14,20,24–26]. These findings have informed our understanding of facial masking in PD as a reduced spontaneity in facial expression of emotion. However, in contrast to Rinn's proposal, PD patients have also been shown to be impaired in voluntarily expressing emotions in response to verbal command (e.g. "look happy") [5,6,12,16,24,25]. In these studies, responses have either been scored by blind raters using Likert scales, the facial action coding system (FACS) [9] which codes observable facial movements as "action units" or action unit combinations, or by digital imaging analyses [6] and kinematic techniques [16]. These findings that were in contrast to earlier propositions [21] spurred the more recent conclusion [6] that facial masking in PD is not limited to spontaneous facial expressions of emotion, but also involves voluntary facial expressions of emotion.

It is reasonable to suspect that non-emotional facial movement is also impaired in PD, thereby contributing with diminished emotional expressions to facial masking. Nevertheless, voluntary non-emotional facial movements remain less systematically explored than voluntary emotional facial movements in PD. Studies on non-emotional facial movement in PD have been limited to specific facial areas, with impairment evidenced in voluntary, spontaneous, and reflex blinking rate and amplitude [1,4,15] and amplitude of jaw and upper lip movement during speech [7]. The group of Simons et al. [24,25] is the only group that has measured, using the FACS, voluntarily imitating a limited set of non-emotional facial movements and found these movements to be impaired in PD. Although the FACS procedure is sensitive to compare action unit patterns with requested patterns, it is not always a practical tool available to clinicians and researchers; FACS certification requires extensive and costly training. There is only one item that assesses facial expressivity on the Movement Disorders Society-sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS) [10]. On this item, PD patients are rated for their facial expressivity for 10 s without moving or speaking. Although this item may pick up some loss of facial musculature control (e.g. reduced control in keeping mouth closed, reduced blinking), the item does not measure the ability to control a range of facial muscles. Therefore, not only is there limited understanding of voluntary facial musculature control in PD, there is

*Abbreviations:* PD, Parkinson's disease; FACS, Facial action coding system; MDS-UPDRS, Movement Disorders Society-sponsored revision of the Unified Parkinson's Disease Rating Scale; MOCA, Montreal Cognitive Assessment; GDS, Geriatric depression scale; LED, Daily levodopa dose equivalent; CI, Confidence interval.

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no sensitive and practical measure of this ability in PD. As a result, the symptom of facial masking and its understanding remains restricted to impaired emotional expression, and mainly assessed by subjective judgment in clinical contexts.

We measured voluntary facial musculature control in PD in three enrollment phases with an adaptation of the Upper and Lower Face Apraxia Test [2], a test of the ability to make upper and lower facial movements. Initial observations of typical errors committed by both groups guided the process of modification of the scoring structure for a more refined classification of errors committed by PD and control participants. The overarching aim of this study was to explore a range of lower and upper non-emotional facial movements in persons with PD and healthy controls; in doing so, we also aimed to determine the utility of an adaptation of the Face Apraxia Test [2] as a sensitive and practical measure of voluntary facial musculature control in PD.

## 2. Materials and methods

### 2.1. Participants

Sixty-six participants (Control $n = 32$, PD $n = 34$), 49 participants (Control $n = 24$, PD $n = 25$), and 40 participants (Control $n = 17$, PD $n = 23$) took part in Enrollment Phases 1, 2, and 3 respectively. There were 22 participants unique to Phase 1 (Control $n = 12$, PD $n = 10$), four unique to Phase 2 (Control $n = 3$, PD $n = 1$), five unique to Phase 3 (Control $n = 2$, PD $n = 3$), and 23 and 26 participants who took part in two or three phases, respectively (two: Control $n = 13$, PD $n = 10$; three: Control $n = 10$, PD $n = 16$). The enrollment phases were approximately 1 year apart. The successive enrollment phases gave the opportunity to establish the reliability of the adapted Face Apraxia Test, by testing some patients in multiple phases; it also gave the opportunity to increase the number of data points in a notoriously variable group of subjects, thereby increasing the reliability of our measures of voluntary facial musculature in PD. PD and control groups were well matched in each phase on demographic and clinical characteristics (Table 1). Patients were diagnosed following clinical evaluation by a neurologist or geriatrician and were under care for PD by a local neurologist or geriatrician at the time of testing. Table 1 also shows PD-related characteristics, MDS-UPDRS-III motor scores, MDS-UPDRS-IV motor complication scores, years since diagnosis, and levodopa dose equivalent (LED) [30]. With the exception of two patients in Phase 1, one patient in Phase 2, and one patient in Phase 3, all patients were on dopamine replacement therapy (see Appendix Table A1 for a list of medications taken by patients for PD management). The local institutional ethics committee approved the study procedures and all participants gave written informed consent.

### 2.2. Stimuli and procedures

For PD patients, each of the enrollment phases was conducted approximately 1.5 h before their next scheduled dopamine replacement medication intake; we did not have ethical approval to compare patients that were on dopamine replacement therapy with those that were depleted from dopamine replacement therapy; patients were not asked to suspend their dopamine replacement therapy overnight, and thus were not in a practically defined off state. At the start of each phase, patients were assessed for disease severity, using the motor subscale (III) of the MDS-UPDRS [10], depressive symptoms (geriatric depression scale; GDS) [22], and general cognitive functioning (Montreal Cognitive Assessment; MOCA) [18]. The ability to voluntarily control facial musculature was measured using an adaptation of the Upper and Lower Face Apraxia Test [2] in which participants were required to make 9 upper and 29 lower facial movements. In the original test, the examiner gives a verbal instruction and demonstration for each item, and the participant's response follows immediately. For standardization purposes in our study, the instructions and demonstrations of each item were recorded in a video-clip lasting 7 min and watched by all participants on an 11-in. laptop. For scoring purposes, participants' faces were filmed with each face in full frontal view. Participants were instructed to reproduce the intensity and duration of each demonstration as accurately as possible.

Two independent raters, one blind to the study, scored the accuracy of each reproduction as correct or incorrect, with set criteria that determine an item as failed. For scoring purposes, the videorecording of each participant's set of reproductions was viewed alongside the video-clip of instructions and demonstrations. Initial observations revealed that errors by participants did not necessarily fit the error criteria in the original test. Facial movements by patients were often impoverished, with reproductions that were of lower amplitude than the demonstrated movement. Observations of impoverished facial movement are also commonly reported in clinical contexts [28,29]. We also observed loss of individuation of movement in PD and control groups, where the requested movement was temporally coupled with uninstructed movement. Individuation loss has also been reported with manual movements in healthy aging [11,17,23] and in PD [31]. Guided by these typical errors, we scored errors due to impoverished movement and individuation loss separately, allowing for a refined error analysis. We also introduced a category for content errors, which might indicate the presence of ideational apraxia. We observed reproductions by participants at times resembled the demonstrated movement but were incorrect in their content e.g. placing the tongue in the cheek when asked to puff out the cheek. Incorrect items were assigned to one of five error categories: (1) the reproduction was preceded by a pause during which unsolicited movements might have been present; (2) there was a loss of

**Table 1**
Demographic and clinical characteristics of participant groups in Enrollment Phases 1, 2, and 3.

| | Phase 1 | | Phase 2 | | Phase 3 | |
|---|---|---|---|---|---|---|
| | Control | PD | Control | PD | Control | PD |
| Age (years) | 66 (51–79) | 66 (46–80) | 67 (55–80) | 67 (53–81) | 70 (53–80) | 68 (58–82) |
| Males (females) | 11 (21) | 20 (14) | 12 (12) | 17 (8) | 11 (6) | 16 (7) |
| Education (years) | 15 (7–22) | 12 (7–20) | 16 (8–22) | 12 (7–20) | 16 (11–21) | 13 (9–19) |
| MOCA | 27 (21–30) | 28 (22–30) | 28 (25–30) | 27 (15–30) | 27 (22–30) | 27 (19–29) |
| GDS | 1 (0–5) | 2 (0–13) | 1 (0–5) | 2 (0–10) | 1 (0–4) | 1 (0–10) |
| Years diagnosed | – | 5 (1–19) | – | 7 (1–20) | – | 8 (2–21) |
| MDS-UPDRS-III | – | 38 (10–56) | – | 41 (19–56) | – | 40 (19–57) |
| MDS-UPDRS-IV | – | 6 (0–14) | – | 5 (0–13) | – | 6 (0–13) |
| LED | – | 788 (0–2046) | – | 916 (0–2312) | – | 1057 (0–2662) |

Note. Values (except male and female numbers) are expressed as median (range); MOCA scores range from 0 to 30, a score ≥26 reflects normal cognitive functioning; GDS scores range from 0 to 15, a score of ≥6 suggests depression warranting assessment; MDS-UPDRS-III scores range from 0 to 132 (most severe); MDS-UPDRS-IV scores range from 0 to 24 (most severe). LED = daily levodopa dose equivalent [30].

individuation: the instructed movement was executed, but was unfocused and accompanied by uninstructed movement that was intermittently or continuously present, or by an increase in the number of elements in a sequential requested movement; (3) the reproduction was executed but impoverished, either by reduction of amplitude of movement or by reduction in the number of elements of a sequential movement; (4) there was no movement at all; or (5) there was a content error, where the reproduction resembled the demonstrated movement but was incorrect in its content.

We report the Cronbach's alpha and the percent agreement between raters [27] by adding the number of cases that received the same rating by raters and dividing that number by the total number of cases rated. We report the mean of both raters' scores in each of the enrollment phases. Unless otherwise stated, we report pooled percent error scores on the Face Apraxia Test across enrollment phases with percent error scores for those unique to each of the enrollment phases and averaged percent error scores for those who did two or three enrollment phases. As recommended by Cumming, Fidler, Kalinowski and Lai [8] in their paper on statistical recommendations for the American Psychological Association, measures of effect size (Hedges' $g$) are reported to quantify overall differences between control and patient groups, and Pearson correlation coefficients and 95% confidence intervals (CI's) are reported for correlation analyses, to provide a more informative analysis of empirical results than statistical significance testing.

## 3. Results

The adapted Face Apraxia Test showed good inter-rater reliability [19] with Cronbach's alphas of .98, .97, and .92 for Enrollment Phases 1, 2, and 3 respectively for total scores. Table 2 shows that Cronbach's alphas for each of the five error categories for all phases were also acceptable. The proportion correct consensus estimate was .91 for Phase 1, and .90 for Phases 2 and 3. The adapted test also demonstrated good test–retest reliability with large positive correlations (Control $r = .85$, 95% CI: .67, .93; PD $r = .85$, 95% CI: .69, .93) between first-time (Phase 1 Control $n = 20$, PD $n = 24$; Phase 2 Control $n = 3$, PD $n = 2$) and second-time performance (Phase 2 Control $n = 18$, PD $n = 22$; Phase 3 Control $n = 5$, PD $n = 4$).

The box-and-whisker plots in Fig. 1 show that the PD group on average made more errors than the control group on the adapted Face Apraxia Test in all enrollment phases (Interquartile Range: Phase 1 Control: 15.8, PD: 20.3; Phase 2 Control: 12.5, PD: 18.0; Phase 3 Control: 17.5, PD: 20.0). The mean percent error rate was higher in the PD ($M = 38.8$, $SD = 14.7$) than control group ($M = 26.6$, $SD = 12.2$), and the effect size ($g = .89$) is considered large by conventional standards. Mean percent of errors was higher in the PD than control group for upper (Control $M = 27.1$, $SD = 14.4$; PD $M = 40.3$, $SD = 15.1$; $g = .89$) and lower facial movements (Control $M = 25.1$, $SD = 12.4$; PD $M = 33.9$, $SD = 20.7$; $g = .51$).

Table 3 shows that the majority of errors committed by both groups were due to loss of individuation and impoverished facial movements. The percent of individuation loss errors was higher in the control than PD group whereas percent of impoverishment errors was higher in the PD than control group, indicating a shift towards impoverished facial movements with the additional burden of PD. Table 3 also shows

**Table 2**
Cronbach's alpha for error assignment by two raters to each of the error categories on the adapted version of the Upper and Lower Face Apraxia Test for Enrollment Phases 1, 2, and 3.

| | Phase 1 | Phase 2 | Phase 3 |
|---|---|---|---|
| Pauses | .85 | .64 | .90 |
| Individuation loss | .96 | .94 | .93 |
| Impoverished movement | .95 | .94 | .93 |
| No movement at all | .95 | .94 | .95 |
| Content errors | .93 | .94 | .98 |

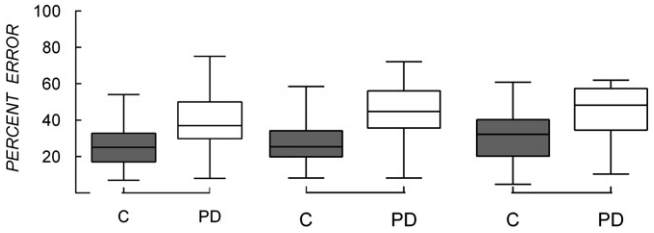


**Fig. 1.** Box-and-whisker plots showing percent error scores for control (filled boxes) and PD (open boxes) groups on the adapted Upper and Lower Face Apraxia Test in Enrollment Phase 1 (left), Enrollment Phase 2 (middle), and Enrollment Phase 3 (right).

that patients committed more errors due to impoverishment and 'no movement at all' than controls, as demonstrated by large and moderate effect sizes respectively. Negligible effect size for errors due to other categories indicate that patients made no more errors than controls due to individuation loss, pauses, and content errors.

The error profile in Fig. 2 of patients at varying stages of the disease (as indicated by the MDS-UPDRS-motor score) shows an increase in the total error number with disease progression ($r = .71$, 95% CI: .51, .84). The figure also shows that the number of errors due to impoverishment increased with disease progression (slope = .19) more so than the number of errors due to individuation loss (slope = .08), no movement (slope = .08), pauses (slope = .03), and content errors (slope = .02). We also calculated correlations between first- and second-time performance for the most predominant error types in PD, impoverishment and loss of individuation, for those who participated in at least two phases ($n = 26$). Correlations between first- and second-time performance were positive and moderate for the numbers of errors made due to impoverishment ($r = .61$, 95% CI: .29, .81) and individuation loss ($r = .60$, 95% CI: .28, .80). After removing the common variance shared with MDS-UPDRS-motor scores, there was little to no correlations between percent errors on the Face Apraxia Test, MOCA scores ($r = -.25$, 95% CI: −.52, .07) and depressive symptoms (measured by the GDS, $r = .18$, 95% CI: −.14, .47).

No relationship was found between MDS-UPDRS motor scores and total errors on the Face Apraxia test when correlating difference scores for individuals between two enrollment phases on the MDS-UPDRS motor subscale and on the Face Apraxia test ($r = .07$, 95% CI: −.33, .45) for those who participated in multiple phases; this near-zero correlation is likely a function of a limited range on both variables, with small changes in both MDS-UPDRS motor and Face Apraxia Test scores between enrollment phases (MDS-UPDRS motor score difference between phases: $M = 2.5$, $SD = 9.5$; Face Apraxia Test error score difference between phases: $M = 1.0$, $SD = 3.1$).

## 4. Discussion

Our results show the utility of the adapted Face Apraxia Test [2] in PD. The adapted test includes standardized video-recorded instructions and demonstrations and three new categories for error classification (individuation loss; impoverished movements; content errors) for an

**Table 3**
Mean percent errors and mean number of errors (with standard deviation in parentheses) for participant groups for each error category with pooled data from those unique to one phase and mean scores for those who completed two or three phases. Hedges' $g$ quantifies group differences.

| | Mean percent of errors | | | Mean number of errors | | |
|---|---|---|---|---|---|---|
| | Controls | PD | g | Controls | PD | g |
| Pauses | 3.5 (4.4) | 2.4 (3.3) | .26 | .4 (.6) | .4 (.8) | .06 |
| Loss of individuation | 59.2 (16.9) | 45.1 (15.6) | .86 | 6.2 (3.6) | 6.6 (3.2) | .11 |
| Impoverishment | 29.2 (17.7) | 44.3 (17.3) | .86 | 2.6 (1.4) | 6.5 (3.3) | 1.51 |
| No movement all | 2.2 (4.0) | 3.3 (4.5) | .27 | .2 (.5) | .6 (.8) | .52 |
| Content errors | 6.0 (7.1) | 4.9 (4.8) | .18 | .6 (.9) | .7 (.7) | .01 |

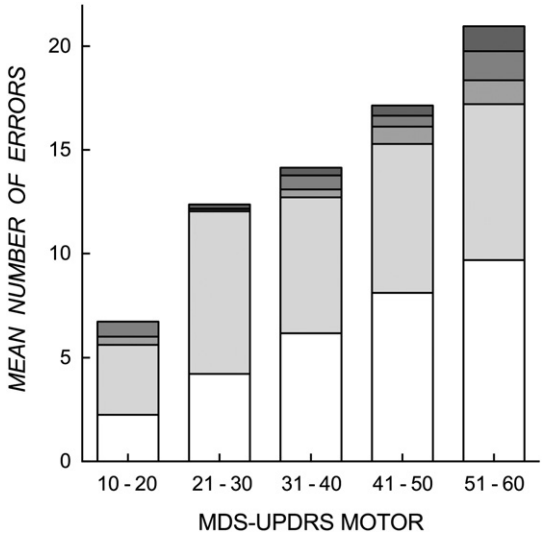


**Fig. 2.** Mean errors due to impoverishment (clear), individuation loss (lightest gray), no movement (medium gray), content (dark gray), and pauses (darkest gray) for patients assigned to each of five severity stages (10–20: $n = 5$; 21–30: $n = 5$; 31–40: $n = 12$; 41–50: $n = 13$; 51–60: $n = 5$).

improved resolution of voluntary facial musculature control in PD. With good inter-rater and test–retest reliability, this measure showed impaired upper and lower facial movements without emotional content in PD. Most errors by patients were due to individuation loss and impoverishment, with a marked increase in percent of impoverishment errors and a marked decrease in percent of individuation loss errors relative to controls. The mean number of errors due to impoverishment and 'no movement at all' was higher in the patient than control group, whereas group differences were negligible in mean number of errors due to individuation loss, pauses, and content errors. There was a strong positive correlation between total number of errors and disease severity, where impoverishment errors increased more so with disease progression than other error types.

The mask-like facial appearance in PD has historically been conceptualized as diminished spontaneous emotional facial expressions [21]. Our data show that PD patients are also impaired in a range of upper and lower non-emotional facial movements, which likely contribute, with blunted emotional expressions, to facial masking in PD. That impaired voluntary non-emotional facial movement was predominantly due to impoverished movement in PD further strengthens the claim that it too contributes to facial masking. The largest difference in number of errors between groups was those due to impoverished facial movements and 'no movement at all'; the latter might be considered the most pronounced case of impoverishment, which might give rise to the most pronounced case of facial masking. Clinical observations of facial masking, which have been shown to affect practitioners' impressions of patient personality, mood, and cognitive competency [28,29], are likely a product of impoverished spontaneous and voluntary emotional expressions, and as we show here, of voluntary non-emotional facial movements. Simply put, less movement of any kind on the face might give rise to the impression of a mask-like face in PD.

The total number of errors correlated positively and highly with disease severity, where impoverished voluntary facial movement increased more than other error types with disease progression. We showed this shift towards impoverishment with a sample of patients in early to moderate stages of disease progression. We expect that impoverishment of facial movement to become even more pronounced in the more advanced stages of the disease. We interpret with caution the negligible difference between groups in the number of errors due to pauses, given that this error type might have been underestimated. It was relatively difficult to capture errors due to pauses because the video-recorded data did not include a time-marker of windows in which participants were instructed to respond. Bowers et al. [6] have shown with sophisticated computer imaging methodology that voluntary emotional movements of the face are slower to initiate in PD than controls, which suggests at least some role of bradykinesia to impaired voluntary control of facial musculature, and facial masking. There was little difference between groups in content errors, which suggests ideational apraxia of the face is no more common in early to moderate stages of PD than that in healthy age-matched controls. These findings are also congruent with the evidence summarized by Zadikoff and Lang [32] of no ideational apraxia of the upper limbs in PD. The low incidence of content errors and the positive correlation with motor severity but not with general cognitive functioning suggest that impaired voluntary facial musculature control in PD is more a part of the motor symptoms than the non-motor symptoms of the disease.

Our results demonstrate that the adapted Face Apraxia Test [2] is a reliable measure of voluntary facial musculature control in PD, which can be used in research and clinical settings to study the presence and extent of facial masking. There are several advantages to the use of this test in PD. The test is practical, free, and easy to administer and score. Unlike the FACS, it does not require intensive and costly training. The test is also comprehensive, whereas previous PD research on non-emotional facial movements has been limited to specific facial areas [1,4,7,15] or to a small set of facial movements [24,25]. The revised error categories allow identifying a range of errors that are not otherwise captured by other measures of facial musculature control in PD. Finally, the test is sensitive to detect between- and within-group differences in voluntary facial musculature control in PD and controls. A limitation of the present study is that we did not compare voluntary facial musculature control in patients that were on dopamine replacement therapy with those that were depleted from dopamine replacement therapy. Therefore, the role of dopamine replacement therapy in voluntary facial movement cannot be determined. However, others have found little to no improvement in upper [1,4] and lower facial movements [16] in patients that were on—than off—dopamine replacement therapy, except for an improvement in spontaneous blinking rate in patients on—than off—dopamine replacement therapy [4].

In addition, similar to all measures of voluntary facial musculature control that provide a demonstration for each item, the Face Apraxia Test requires imitating demonstrated movements. It is unknown whether movement imitation inflates or deflates impairment in voluntary facial musculature control in PD. In summary, the work described here adds to the current literature on facial musculature control in PD by showing that a range of upper and lower voluntary non-emotional movements of the face are impaired. Furthermore, that these voluntary facial movements were impaired mainly due to impoverishment makes it likely to give rise, with impoverished emotional expressions, to the impression of the mask-like facial appearance that is seen with the progression of the disease.

## Conflict of interest

There is no conflict of interest.

## Acknowledgments

We are very grateful to the people with PD and those from the healthy control group who have kindly given their time by committing to this study. We would also like to thank ParkC at Curtin University (formerly at Edith Cowen University) and Parkinson's Western Australia for assisting with participant recruitment. We would also like to acknowledge the funding to the first author: Australian Postgraduate Award, Jean Rogerson Postgraduate Scholarship, University of Western Australia PhD Completion Scholarship, and a Parkinson's Western Australia Research Grant.

## Appendix A

**Table A1**
Therapeutic regimen for the management of PD of patients in Enrollment Phases 1, 2, and 3.

| | Phase 1 | Phase 2 | Phase 3 |
|---|---|---|---|
| Dopamine replacement therapy | | | |
| Levodopa–carbidopa | 12 (2) | 9 (2) | 12 (5) |
| Levodopa–carbidopa–entacapone | 3 | | 7 |
| Levodopa–benserazide | 19 | | 14 |
| Dopamine agonists | | | |
| Pramipexole | 20 (12) | 14 (7) | 14 (10) |
| Cabergoline | 3 | 1 | 1 |
| Rotigotine | 1 | 0 | 0 |
| Other | | | |
| Entacapone | 5 | 2 | 3 |
| Rasagaline | 0 | 0 | 1 |
| Amantadine | 1 | 1 | 1 |

*Note.* Values are the number of participants (with the number of those taking extended release type medications in parentheses).

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