Published in final edited form as:
*Parkinsonism Relat Disord*. 2010 March ; 16(3): 177–180. doi:10.1016/j.parkreldis.2009.11.002.

# Mild cognitive impairment in Parkinson's disease: Subtypes and motor characteristics☆

**Ann B. Sollinger***, **Felicia C. Goldstein**, **James J. Lah**, **Allan I. Levey**, and **Stewart A. Factor**
Department of Neurology, Emory University School of Medicine, Atlanta, GA, USA

## Abstract

The aims of this project were to determine the risk factors for and clinical characteristics of mild cognitive impairment (MCI) in Parkinson's disease (PD). We performed a retrospective record review of 72 non-demented PD patients (age: 57.79 ± 10.57, duration of PD: 7.32 ± 4.97) who completed a standardized neurological assessment, including a full neuropsychological battery, as part of their diagnostic work-up. Of these participants, 47.2% were cognitively normal and 52.8% met criteria for MCI. The majority of MCI patients had single domain MCI (23/38), the affected domains being memory ($n$ = 9), executive function ($n$ = 6), visuospatial skills ($n$ = 6), and language ($n$ = 2). The MCI group had longer duration of disease and higher postural instability and gait disorder subscale scores than the cognitively normal group. This report provides further support for use of the concept of MCI in PD research. There may be certain disease characteristics that could alert practitioners to the emergence of cognitive changes in patients. Future studies should focus on additional risk factors for MCI subtypes and their possible progression to frank dementia.

### Keywords

Parkinson's disease; Mild cognitive impairment; Dementia; Motor dysfunction; Cognition

Parkinson's disease (PD) is a progressive neurodegenerative disorder typically characterized by motor symptoms including tremor, rigidity, bradykinesia, and postural instability and gait dysfunction. It has become apparent, however, that non-motor features also play a key role, with dementia representing the greatest unmet need. The prevalence of dementia in PD as defined by neuropsychological testing has been estimated to be between 37 and 44% [1], and PD carries an approximately six-fold increased risk for dementia compared to the general population [2]. Dementia in PD is associated with older age and greater overall motor dysfunction [3]. Additionally, motor symptoms less responsive to dopaminergic drugs, particularly postural instability and gait disorder, appear to have a higher association [4].

While dementia is frequently a late feature of PD, subtle cognitive dysfunction can be found early in the disease course. Cognitive impairment not reaching the level of dementia is typically characterized by executive dysfunction, and these subtle changes have been reported in approximately 30% of non-demented PD patients [5]. While some suggest that cognitive impairment may be predictive of the development of later dementia [6], it is

☆The review of this paper was entirely handled by an Associate Editor, Robert Rodnitzky.



*Corresponding author at: Emory University, Wesley Woods Health Center, 1841 Clifton Rd, N.E., Atlanta, GA 30329, USA. Tel.: +1 404 728 6909; fax: +1 404 728 6685. asollin@emory.edu (A.B. Sollinger).

unknown whether these early difficulties represent frontal and/or subcortical processing changes that might be stable and part of the spectrum of dopaminergic dysfunction or whether they are the prodrome of a progressive dementing syndrome. This is an important question to answer as it could lead to prediction and prevention of dementia in PD.

The concept of mild cognitive impairment (MCI) as defined by Petersen proved fruitful in furthering our understanding of early phases of dementia, particularly in Alzheimer's disease (AD) [7]. Although originally focused on memory impairment as a risk factor for AD, the definition of MCI has since been expanded to include other domains such as language, executive functioning, and visuospatial skills. Different subtypes of MCI are now hypothesized to represent the prodromal phase for several different disorders (e.g., FTD, vascular dementia) [7].

Whether the application of this MCI concept to PD is appropriate is largely unknown, but the idea has sparked much recent interest. Several studies now label the early cognitive change in PD as MCI by the Petersen definition. Estimates of MCI in PD vary widely, ranging from 18 to 52% [8–10]. Caviness et al. found, in their brain bank population, that a transitional state between normal cognition and dementia could be defined in PD just as in AD [8]. The majority of their cases had single domain MCI (67%) with either executive dysfunction (39%) or amnestic deficit (22%). Janvin and colleagues found that single non-memory domain (44.7%) (mostly executive dysfunction) followed by multiple impaired domains (39.5%), and then single memory domain MCI (15.8%) were the most prevalent subtypes in PD [10]. MCI was a risk factor for development of dementia within four years, and the results suggested that MCI represents the initial stage of a progressive cognitive decline in PD that leads to frank dementia [10]. Recently, Mamikonyan and colleagues noted that even in a cohort of PD patients with intact global cognitive functioning as measured by the Mini Mental State Examination (MMSE), almost 30% were classified as MCI through neuropsychological testing [9].

The above studies represent important first steps in applying the concept of MCI to PD, but further study is required. Only one published study to date has utilized an extensive neuropsychological battery assessing all cognitive domains to characterize subtypes of MCI [8]. Also, no study has explored the relationship between MCI subtypes as defined by Petersen and their relationship to subsets of motor symptoms in PD. In the current investigation, we utilized a neuropsychological battery assessing all cognitive domains and examined the clinical characteristics associated with PD–MCI. Of particular interest was whether the relationship between postural instability and gait disorder (PIGD) and tremor and dementia in PD would also be found in PD–MCI. We hypothesized that MCI affecting a single domain involving executive functioning would be the most prevalent subtype. In addition, based on previous findings linking clinical characteristics to dementia in PD [2,6], we hypothesized that PD–MCI would be associated with longer duration of PD, greater overall severity of motor symptoms, and greater non-tremor symptomatology.

## 1. Methods

### 1.1. Participants

The Emory University IRB approved the research protocol, and all participants signed a consent document. We reviewed the records of 103 PD patients who were invited to participate in a research study investigating cognition in neurological disorders and who completed neuropsychological testing. Patients were consecutively recruited from a movement disorders clinic independent of whether they had cognitive complaints. All were English-speaking individuals diagnosed with PD by a movement disorder neurologist based on the presence of 2 of 3 cardinal features and absence of features suggestive of atypical

parkinsonism. We excluded individuals with a history of other neurological (e.g., stroke, head injury, $N = 9$) or psychiatric disorders (e.g., current major depressive episode, $N = 4$) that may have affected cognitive functioning. Additionally, we excluded patients with a diagnosis of Lewy body dementia ($N = 1$) defined as those with documentation of onset of cognitive and motor symptoms within one year of the other. Seventeen other participants were excluded because they did not have complete data for each neuropsychological domain.

### 1.2. Clinical assessment

Patients completed an extensive clinical evaluation, including review of medical history, informant interview, assessment of functional abilities, neurological examination, and cognitive evaluation. Data analyzed from these assessments included demographic information (duration of illness, age at onset, age at testing), motor ratings using a modified, shortened, version of the motor portion (Part 3) of the Unified Parkinson's Disease Rating Scale (UPDRSm) [11,12], scores on neuropsychological tests, and functional information. As hallucinations have been linked to dementia in PD, we assessed the presence of hallucinations by reviewing a research questionnaire that documented the presence/absence of this symptom as reported by the patient and informant.

**1.2.1. Cognitive and functional assessment—**The standard battery included cognitive tests in the following domains: visuospatial skills, language, attention, executive functioning, and memory. Visuospatial skills were assessed by having patients determine the angular orientation of lines (Judgment of Line Orientation) and copy the intersecting pentagons on the MMSE [13,14]. Language was examined via the Boston Naming Test and completion of letter and semantic verbal fluencies [15,16]. Attention was evaluated by having patients repeat digits forward and by the number of seconds needed to sequence numbers using a pencil (Trails A) [17]. For the evaluation of memory, participants completed either the CERAD Word List or the Hopkins Verbal Learning Test-Revised (HVLT-R) [18,19], and we also used their three-item recall from the MMSE. Executive functions were measured by ability to sequence numbers and letters using a pencil (Trails B) and by ability to spell the word "world" backwards.

Forty-two patients completed this standard battery with the CERAD Word List, while an additional 30 were identified who completed the Hopkins Verbal Learning Test-Revised (HVLT-R) due to a change in the cognitive battery. Comparisons between the proportion of patients considered normal or MCI did not differ significantly between those patients receiving the CERAD or the HVLT-R ($\chi^2 = 0.006$, df = 1, $p > 0.05$), and thus the results reported include all 72 participants. Standardized, published normative datasets were used as comparative references to determine impairments [16,19–22]. As suggested by Petersen [7], we did not rely solely on a strict cut-off score to determine impairment but made a clinical judgment as to whether performance was deficient for an individual in the context of their estimated premorbid abilities.

For determination of cognitive status, two neuropsychologists (ABS, FCG) provided independent diagnoses classifying patients as cognitively normal or MCI. Discrepancies were resolved via a consensus conference. Study partners (family/friends serving as informants) provided information on the patient's functional status, which was assessed through Lawton and Brody's self-maintenance and independent activities of daily living questionnaire [23] and/or a clinical history form assessing problems with everyday activities (e.g., impairment in hobbies, impairment in finances, etc). A clinician interview was also conducted to further probe for functional difficulties and to discriminate the extent to which any decline was attributable to cognitive, not motor, impairment. We defined mild cognitive

impairment according to Petersen criteria, including subjective (either participant or informant report) and objective cognitive impairment in the absence of associated functional limitations [7]. In addition, in order to document consistent impairment, we required deficits on at least two measures in a domain before that domain was considered abnormal. For example, we considered visuospatial functioning to be impaired in individuals with both deficient performance on Judgment of Line Orientation and their copy of interlocking pentagons from the MMSE. We further divided participants with MCI into four categories: amnestic MCI-single domain, amnestic MCI-multidomain, non-amnestic MCI-single domain, and non-amnestic MCI-multidomain based on the pattern of cognitive results [7].

**1.2.2. Motor assessment—**Sixty-nine patients had a modified UPDRS Part III [12]. This modification of the UPDRS included items assessing tremor, rigidity, gait, rising from a chair, stability, bradykinesia (face and body), posture, and speech, with scores ranging from 0 to 76. We combined single item scores from the UPDRSm based on clinical symptomatology to create two subsets of motor symptoms: postural instability and gait impairment (PIGD; rise from a chair, posture, gait, and postural stability) and tremor (head, upper and lower extremity tremor at rest, and postural/action tremor of the upper extremities) as described in Williams et al. [24].

### 1.3. Statistical analysis

Patients were divided into two groups, normal cognition and MCI. We compared the groups in terms of demographic (e.g., age, education, etc), clinical (e.g., age at PD onset, PD duration, etc.), and motor characteristics (e.g., UPRDSm total and subscales). We utilized parametric statistics (independent *t*-tests) to compare groups on continuous variables when the data were normally distributed and nonparametric statistics (Mann-Whitney U) when the data followed a non-normal distribution. Chi-square tests were used to compare categorical variables.

## 2. Results

Seventy-two participants fulfilled study criteria and were included in the analysis. We classified 34 (47.2%) as cognitively intact (CI) and 38 (52.8%) as MCI. Table 1 displays the demographic and general clinical characteristics of the groups.

Significant overall differences were found between subgroups in duration of PD ($t = -2.56$, df = 70, $p < 0.05$) and MMSE scores (Mann-Whitney $U = 273.5$, $p < 0.001$). There were no differences in education, age at testing, age at PD onset, gender, or handedness. There were no differences between groups in use of levodopa, dopamine agonists, antipsychotic medications, antidepressant medications, anticholinergic medications, or use of amantadine. Data on hallucinations were available on 70 participants, with 5/38 of the MCI group and 1/32 of the CI group displaying hallucinations. This comparison was not statistically significant.

Within the MCI group, participants displayed impairment most frequently in the domains of memory ($n = 18$) and executive functioning ($n = 17$), followed by visuospatial skills ($n = 15$), language ($n = 12$), and attention ($n = 5$). The majority met criteria for single domain MCI (Table 2), the affected domains being memory ($n = 9$), executive functions ($n = 6$), visuospatial skills ($n = 6$), and language ($n = 2$).

In terms of motor functioning, UPDRSm and tremor subscale scores did not differ significantly between the groups. Differences were seen, however, in PIGD subscores, (Mann-Whitney $U = 393.5$, $p < 0.05$) (Table 3) with the MCI group showing higher scores on this measure than the CI group.

We further divided the MCI group into the following different clusters: single domain ($N$ = 23) and multi-domain ($N$ = 15) MCI and amnestic MCI ($N$ = 18) and non-amnestic MCI ($N$ = 20). Comparisons of these groups did not reveal any statistically significant differences in demographic or motor characteristics.

## 3. Discussion

Our results support the notion that a state of cognitive impairment intermediate between normal cognition for age and dementia can be identified in PD using Petersen criteria [7]. In this sample with a neuropsychological battery assessing all cognitive domains, we found that 52.8% of cases met criteria for PD–MCI. Of the individuals with MCI, the mean duration of disease was <8 years and was as short as 1.5 years for some patients, suggesting that these subtle cognitive difficulties may be present very early in the disease course. Several factors, including definition of impairment, number of domains assessed, and the specific tests comprising the neuropsychological battery, are likely to significantly affect the proportion of PD patients found to have MCI in any particular sample. Although our techniques differed in these aspects from several other authors, the rate of MCI in our sample was within the range of that found in prior studies. Specifically, our findings are consistent with two reports of Janvin et al. who found that 52% and 55% of their samples respectively had cognitive impairment without dementia [10,25]. Similarly, when Caviness et al. required an abnormality of only a single test in a domain, they found that 51% of their sample met criteria for MCI [8]. Finally, using a more limited battery, Mamikonyan found 29.2% of their large cohort showed evidence of MCI [9]. Thus, as has been previously suspected, MCI is prevalent in PD and would benefit from a unified definition and system of categorization.

In terms of the cognitive characterization of PD–MCI, the most prevalent subtype was non-amnestic MCI-single domain, with frequent executive and visuospatial dysfunction. Executive dysfunction has formerly been demonstrated in PD patients without dementia and was frequently seen in our MCI sample (44.7%) [26]. However, we found a slightly higher rate of memory disturbance than reported in previous studies investigating application of Petersen MCI criteria [8,10]. This variance may represent differences in the definition of MCI (statistical cut-offs vs. clinical determination of impairment), measurements used, and/or population characteristics. On the other hand, some previous studies of early cognitive dysfunction in PD have documented similar high rates of memory dysfunction [27]. The fact that in our study memory disturbance was largely accompanied by executive dysfunction may suggest that PD memory impairment is secondary to executive difficulties. It has been shown, however, that memory impairment in PD is variable, with some studies demonstrating subcortical patterns potentially arising from executive dysfunction, and others showing rapid forgetting and poor recognition memory, a pattern much more often associated with cortical dementias such as AD [28]. Further study is needed to determine whether these subgroups of memory dysfunction can be replicated, whether they are stable, and if they have different outcomes and pathology.

We found that, despite the occurrence in some patients early on, longer duration of PD was associated with MCI categorization, and this has been previously reported [2]. Additionally, we found that a subtype of motor dysfunction, PIGD, was associated with increased cognitive dysfunction, as the PIGD subscores were higher in the MCI group relative to the CI group. Several investigators have reported that patients whose motor phenotype is predominantly characterized by symptoms less responsive to dopaminergic drugs, such as PIGD, have greater risk for development of dementia [4,6]. While visual hallucinations have been associated with dementia in PD, we did not find a statistically significant relationship between MCI categorization and hallucinations. This may have been due to reduced power

given the small sample size as 13% of the MCI group had hallucinations and only 3% of the CI group reported this symptom.

In relation to MCI, subtypes have been hypothesized as representing the early stages of different dementia processes [7]. Hypotheses regarding the pathological basis of PDD have included three main possibilities: 1) pathology directly related to PD in the brainstem, 2) cortical Lewy bodies, and 3) Alzheimer's disease pathology. It may ultimately be found that PD–MCI subtypes reflect these different pathological processes, with their diverse cognitive outcomes and clinical courses. Further investigation is required.

This study has several limitations, including retrospective nature of the data collection and the use of a truncated form of the UPDRS. Additionally, there may be some concern that we did not utilize a strict psychometric cut-off when defining MCI, and thus may have introduced bias in defining the groups. As there are no agreed upon cut-offs for defining MCI, we chose to apply clinical judgment based on the recommendations of several work groups focusing on the diagnosis of MCI [29,30]. Moreover, we required impairments in two tests comprising a domain, as well as a consensus diagnosis of two neuropsychologists to reduce the possibility of bias. In reviewing the data, all participants judged to have MCI fell at least one standard deviation or more below the mean on tests that were considered impaired, thus providing some objective support for our clinical impressions. Finally, the fact that the study was conducted in a university setting specifically investigating cognitive difficulties in neurological diseases may have inflated the proportion of patients with cognitive deficits, although this seems unlikely given that the rates found in this study are consistent with those previously reported. Nevertheless, this study offers further evidence of the utility of the concept of PD–MCI and the possibility that subtypes exist. Future work will require further attention to the motor characterization of subtypes and longitudinal follow-up to investigate the outcome and potential pathological correlates of each.

## Acknowledgments

This research was supported by the Emory Alzheimer's Disease Research Center (NIH-NIA 5 P50 AG025688), The Close to the Cure Foundation, and the Sartain Lanier Family Foundation. We thank Rachel Weissman, B.A. for her help in data extraction and organization.

## References

1. Emre M. What causes mental dysfunction in Parkinson's disease? Mov Disord. 2003 Sep; 18(Suppl 6):S63–71. [PubMed: 14502658]
2. Aarsland D, Andersen K, Larsen JP, Lolk A, Nielsen H, Kragh-Sorensen P. Risk of dementia in Parkinson's disease: a community-based, prospective study. Neurology. 2001 Mar 27; 56(6):730–6. [PubMed: 11274306]
3. Levy G, Schupf N, Tang MX, Cote LJ, Louis ED, Mejia H, et al. Combined effect of age and severity on the risk of dementia in Parkinson's disease. Ann Neurolo. 2002 Jun; 51(6):722–9.
4. Alves G, Larsen JP, Emre M, Wentzel-Larsen T, Aarsland D. Changes in motor subtype and risk for incident dementia in Parkinson's disease. Mov Disord. 2006 Aug; 21(8):1123–30. [PubMed: 16637023]
5. Foltynie T, Brayne CE, Robbins TW, Barker RA. The cognitive ability of an incident cohort of Parkinson's patients in the UK. The CamPaIGN study. Brain. 2004 Mar; 127(Pt 3):550–60. [PubMed: 14691062]
6. Verbaan D, Marinus J, Visser M, van Rooden SM, Stiggelbout AM, Middelkoop HA, et al. Cognitive impairment in Parkinson's disease. J neurology Neurosurg Psychiatry. 2007 Nov; 78(11): 1182–7.
7. Petersen RC. Mild cognitive impairment as a diagnostic entity. J Internal Medicine. 2004 Sep; 256(3):183–94.

8. Caviness JN, Driver-Dunckley E, Connor DJ, Sabbagh MN, Hentz JG, Noble B, et al. Defining mild cognitive impairment in Parkinson's disease. Mov Disord. 2007 Jul 15; 22(9):1272–7. [PubMed: 17415797]
9. Mamikonyan E, Moberg PJ, Siderowf A, Duda JE, Have TT, Hurtig HI, et al. Mild cognitive impairment is common in Parkinson's disease patients with normal Mini-Mental State Examination (MMSE) scores. Parkinsonism Relat Disord. 2008 Jun 30.
10. Janvin CC, Larsen JP, Aarsland D, Hugdahl K. Subtypes of mild cognitive impairment in Parkinson's disease: progression to dementia. Mov Disord. 2006 Sep; 21(9):1343–9. [PubMed: 16721732]
11. Fahn, S.; Elton, RL. UPDRS development committee. In: Fahn, S.; Marsden, CD.; Calne, DB.; Goldstein, M., editors. Recent developments in Parkinson's disease. Florham Park, NJ: Macmillian; 1987. p. 153-63.
12. Clark CM, Ewbank D, Lerner A, Doody R, Henderson VW, Panisset M, et al. The relationship between extrapyramidal signs and cognitive performance in patients with Alzheimer's disease enrolled in the CERAD Study. Consortium to Establish a Registry for Alzheimer's Disease. Neurology. 1997 Jul; 49(1):70–5. [PubMed: 9222172]
13. Benton, A.; Varney, NR.; Spreen, O. Contributions to neuropsychological assessment: a clinical manual. New York: Oxford University Press; 1983.
14. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975 Nov; 12(3):189–98. [PubMed: 1202204]
15. Kaplan, EF.; Goodglass, H.; Weintraub, S. The Boston naming test: experimental edition. 2. Philadelphia: Lea & Febiger; 1983.
16. Heaton, RK.; Miller, SW.; Taylor, MJ.; Grant, I. Revised comprehensive norms for an expanded halsteadreitan battery: demographically adjusted neuropsycological normal for African American and Caucasian adults. Lutz, FL: PAR; 2004.
17. Army, U. Army individual test battery. Washington, DC: War Department, Adjutant general's office; 1944.
18. Morris JC, Heyman A, Mohs RC, Hughes JP, van Belle G, Fillenbaum G, et al. The Consortium to Establish a Registry for Alzheimer's disease (CERAD). Part I. Clinical and neuropsychological assessment of Alzheimer's disease. Neurology. 1989 Sep; 39(9):1159–65. [PubMed: 2771064]
19. Brandt, J.; Benedict, RH. Hopkins verbal learning test-revised. Odessa, FL: PAR; 2001.
20. Crum RM, Anthony JC, Bassett SS, Folstein MF. Population-based norms for the mini-mental state examination by age and educational level. JAMA. 1993 May 12; 269(18):2386–91. [PubMed: 8479064]
21. Welsh KA, Butters N, Mohs RC, Beekly D, Edland S, Fillenbaum G, et al. The Consortium to Establish a Registry for Alzheimer's disease (CERAD). Part V. A normative study of the neuropsychological battery. Neurology. 1994 Apr; 44( 4):609–14. [PubMed: 8164812]
22. Wechsler, D. Wechsler memory scale-revised. San Antonio, Texas: The Psychological Corporation; 1987.
23. Lawton MP, Brody EM. Assessment of older people: self-maintaining and instrumental activities of daily living. Gerontologist. 1969; 9:179–86. [PubMed: 5349366]
24. Williams LN, Seignourel P, Crucian GP, Okun MS, Rodriguez RL, Skidmore FM, et al. Laterality, region, and type of motor dysfunction correlate with cognitive impairment in Parkinson's disease. Mov Disord. 2007 Jan; 22(1):141–5. [PubMed: 17089386]
25. Janvin C, Aarsland D, Larsen JP, Hugdahl K. Neuropsychological profile of patients with Parkinson's disease without dementia. Dement Geriatr Cogn Disord. 2003; 15(3):126–31. [PubMed: 12584427]
26. Woods SP, Troster AI. Prodromal frontal/executive dysfunction predicts incident dementia in Parkinson's disease. J Int Neuropsychol Soc. 2003 Jan; 9( 1):17–24. [PubMed: 12570354]
27. Zakharov VV, Akhutina TV, Yakhno NN. Memory impairment in Parkinson's disease. Neurosci Behav Physiol. 2001 Mar-Apr;31(2):157–63. [PubMed: 11388367]

28. Weintraub D, Moberg PJ, Culbertson WC, Duda JE, Stern MB. Evidence for impaired encoding and retrieval memory profiles in Parkinson disease. Cogn Behav Neurol. 2004 Dec; 17(4):195–200. [PubMed: 15622014]

29. Winblad B, Palmer K, Kivipelto M, Jelic V, Fratiglioni L, Wahlund L, et al. Mild cognitive impairment – beyond controversies, towards a consensus: report of the International working group on mild cognitive impairment. J Intern Med. 2004; 256:240–6. [PubMed: 15324367]

30. Portet F, Ousset PJ, Visser PJ, Grisoni GB, Nobili F, Scheltens P, et al. Mild cognitive impairment (MCI) in medical practice: a critical review of the concept and new diagnostic procedure. Report of the MCI Working Group of the European Consortium of Alzheimer's Disease. J Neurol Neurosurg Psychiatry. 2006; 77:714–8. [PubMed: 16549412]

**Table 1**

Characteristics of the PD groups.

| | Cognitively Intact (*N* = 34) | MCI (*N* = 38) |
|---|---|---|
| Age at onset of PD | 58.2 (9.9) | 57.4 (11.2) |
| Age at testing | 63.7 (9.5) | 66.0 (10.0) |
| Education | 16.0 (2.5) | 15.4 (2.5) |
| Duration of PD[a] | 5.8 (4.3) | 8.7 (5.2) |
| % Male | 67.6% | 81.5% |
| MMSE[b] | 29.0 (1.2) | 27.1 (2.1) |

[a] $p < 0.05$.

[b] $p < 0.001$.

**Table 2**

Subtypes of MCI.

| | ***N*** **(% of MCI)** |
|---|---|
| Amnestic MCI-Single Domain | 9 (23.7) |
| Amnestic MCI-Multiple Domain | 9 (23.7) |
| Non-amnestic MCI-Single Domain | 14 (36.8) |
| Non-amnestic MCI-Multiple Domain | 6 (15.8) |

**Table 3**

Motor ratings of the two groups.[a]

| Motor rating (range) | Cognitively intact (*N* = 32) | MCI (*N* = 37) |
|---|---|---|
| Total modified UPDRS (0–76) | 10.7 (6.6) | 13.7 (6.0) |
| PIGD subscale score (0–16) | 2.1 (2.3) | 3.2 (2.3)[b] |
| Tremor subscale score (0–28) | 2.0 (2.0) | 1.8 (2.2) |

[a] Sixty-nine of the seventy-two participants had available UPDRSm scores.

[b] $p < 0.05$.