Cognitive Functioning in Movement Disorders

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Definitions

- Cognition
  - Thinking and memory
  - Includes both verbal (language) and nonverbal (spatial) functions
- Dementia
  - Decline in cognitive function without alteration of consciousness (alertness)
  - Impairment (more than normal for age) in memory, plus
    - language, spatial cognition, "executive function" (planning, reasoning, problem-solving)
  - Functional impairment

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Causes of Dementia

- CORTICAL
  - Alzheimer's Disease
  - Frontotemporal Dementia
    - Pick's Disease
    - Motor-Neuron Disease Inclusion Body Dementia
  - Multi-Infarct Vascular Dementia
  - Dementia with Lewy Bodies
- SUBCORTICAL
  - Huntington's Disease
  - Parkinson's Disease
  - Subcortical Vascular Dementia
  - Normal Pressure Hydrocephalus
  - Cerebral HIV Infection

Subcortical Dementia

- Related to damage in structures below the cortex (e.g., basal ganglia, brainstem nuclei, thalamic nuclei, cerebral white matter, cerebellum)
- Has many more causes than cortical dementia
  - Depression
  - Vascular dementia
  - Parkinson's disease
  - Normal pressure hydrocephalus
  - Huntington's disease
  - Progressive supranuclear palsy
  - Systemic lupus eurythematosis
  - Multiple sclerosis
  - Acquired immune deficiency syndrome

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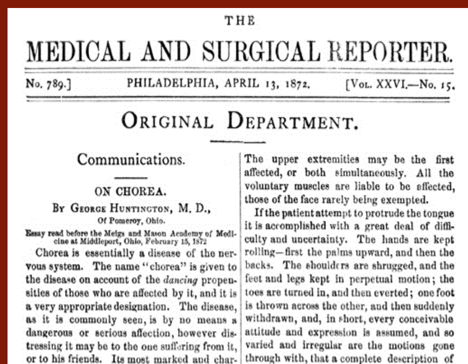
Huntington's Disease



THE

MEDICAL AND SURGICAL REPORTER.

No. 789.] PHILADELPHIA, APRIL 13, 1872. [Vol. XXVI.—No. 15.

ORIGINAL DEPARTMENT.

Communications.

ON CHOREA.

By George Huntington, M. D.,
Of Pomeroy, Ohio.

Essay read before the Meigs and Mason Academy of Medicine at Middleport, Ohio, February 15, 1872

Chorea is essentially a disease of the nervous system. The name "chorea" is given to the disease on account of the *dancing* propensities of those who are affected by it, and it is a very appropriate designation. The disease, as it is commonly seen, is by no means a dangerous or serious affection, however distressing it may be to the one suffering from it, or to his friends. Its most marked and char-

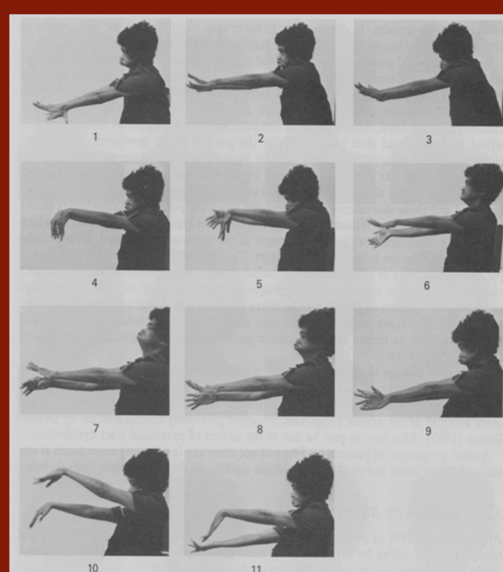
The upper extremities may be the first affected, or both simultaneously. All the voluntary muscles are liable to be affected, those of the face rarely being exempted.

If the patient attempt to protrude the tongue it is accomplished with a great deal of difficulty and uncertainty. The hands are kept rolling—first the palms upward, and then the backs. The shoulders are shrugged, and the feet and legs kept in perpetual motion; the toes are turned in, and then everted; one foot is thrown across the other, and then suddenly withdrawn, and, in short, every conceivable attitude and expression is assumed, and so varied and irregular are the motions gone through with, that a complete description of

"… The tendency to insanity, and sometimes that form of insanity which leads to suicide, is marked. I know of several instances of suicide of people suffering from this form of chorea, or who belonged to families in which the disease existed. As the disease progresses the mind becomes more or less impaired, in many amounting to insanity, while in others mind and body both gradually fail until death relieves them of their sufferings."

Huntington's Disease

- Insidious onset of abnormal movements in mid-life
  - Involuntary movements, esp. chorea and athetosis
  - Impaired voluntary movements and dystonia
- Subcortical dementia
  - Prominent attention and executive dysfunction
- Emotional/motivational disorder
  - Especially irritability, apathy and depression
- Progresses to death in approximately 15 years
- Autosomal dominant inheritance



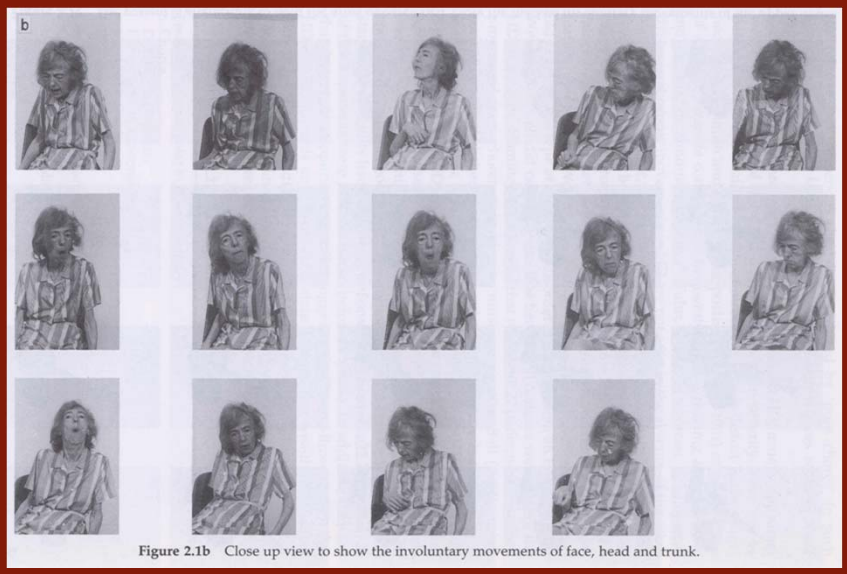
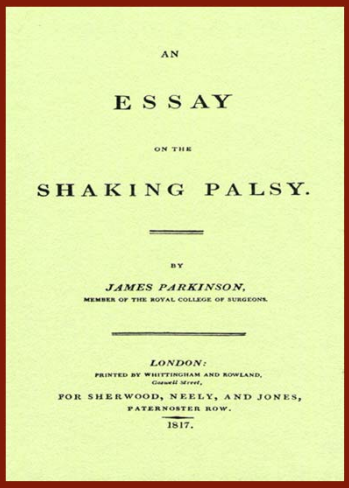


Figure 2.1b Close up view to show the involuntary movements of face, head and trunk.

Huntington's Disease

The apparent enlargement of the ventricles seen here is due to atrophy of the head of the caudate from neuronal loss in Huntington's disease, an autosomal dominant condition characterized clinically by choreiform movements.

Parkinson's Disease



AN

ESSAY

ON THE

SHAKING PALSY.

BY

JAMES PARKINSON,

MEMBER OF THE ROYAL COLLEGE OF SURGEONS.

LONDON:

PRINTED BY WHITTINGHAM AND ROWLAND,

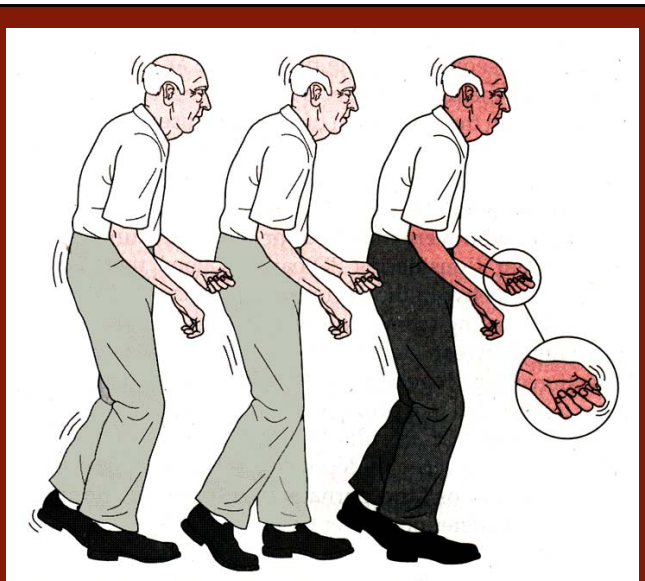
Goswell Street,

FOR SHERWOOD, NEELY, AND JONES,

PATERNOSTER ROW.

1817.

" ...involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace, the senses and the intellects uninjured."

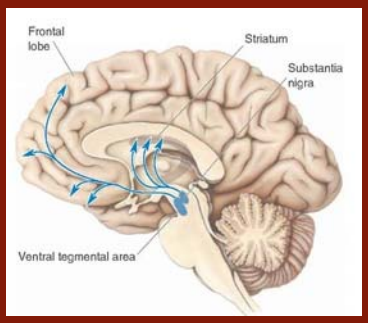


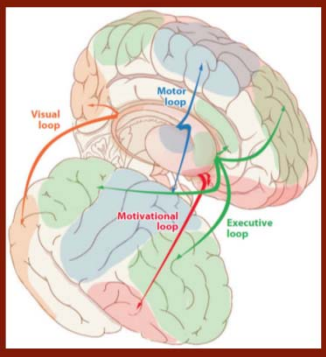
20.2 CLASSIC SYMPTOMS OF LATE-STAGE PARKINSON'S DISEASE, including a stooped and rigid posture, shuffling gait, tremor, a masklike facial appearance, and "pill rolling" (*inset*). (After Markey, 1986.)

Parkinson's Disease

- Progressive loss of dopaminergic neurons in substantia nigra

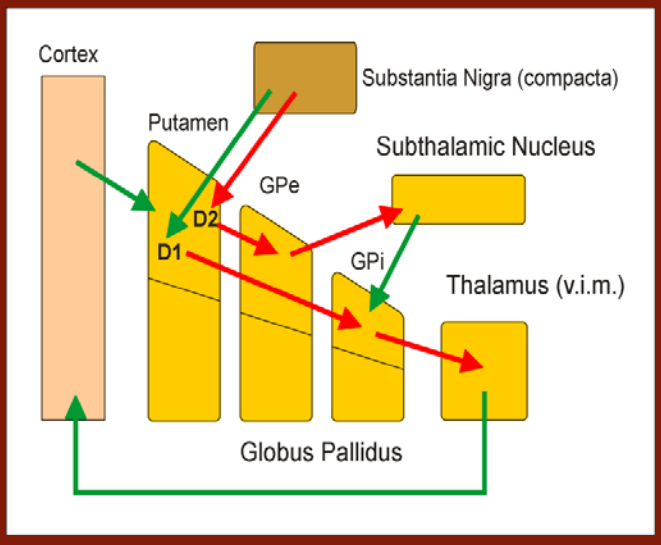
Cognition in Parkinson's Disease



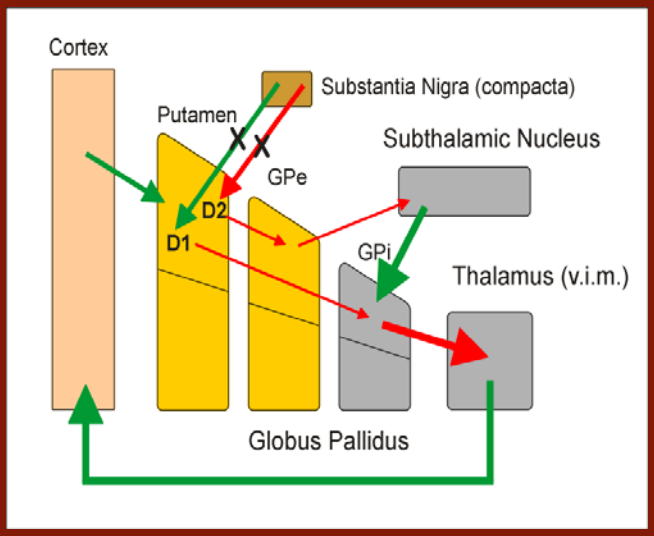


- Dopamine-producing neurons in the substantial nigra send signals to the striatum, a part of the basal ganglia
- Dopamine-producing neurons in the ventral tegmental area send signals to the frontal lobes
- The striatum and cerebral cortex have reciprocal connections; they send information back and forth
- Different parts of the striatum are connected to different parts of the cortex with specialized functions (visual, motor, executive, motivation)

Extra-Pyramidal Motor System



Parkinson's Disease



Subcortical Dementia

- A syndrome characterized by
  - Slowed mentation (bradyphrenia)
  - Deficits in sustained attention and working memory
  - Forgetfulness
  - Impaired planning and judgment
  - Changes in drives and/or mood states (with apathy, irritability, and depression being most common)
- Because subcortical dementia is often caused by diseases causing movement disorders, can also see
  - Dysarthric speech and psychomotor slowing
  - Problems with motor set acquisition and switching

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Distinguishing Dementia Syndromes

- **CORTICAL**
  - Amnesia (memory)
  - Aphasia (language)
  - Agnosia (recognition)
  - Apraxia (purposeful movement)
  - Acalculia (calculations)
- **SUBCORTICAL**
  - Bradyphrenia (slowed thinking)
  - Impaired attention and concentration
  - Inefficient retrieval from memory
  - Executive dysfunction
    - Impaired formation and disengagement of mental sets
    - Poor planning and forethought

Cognition in Parkinson's Disease

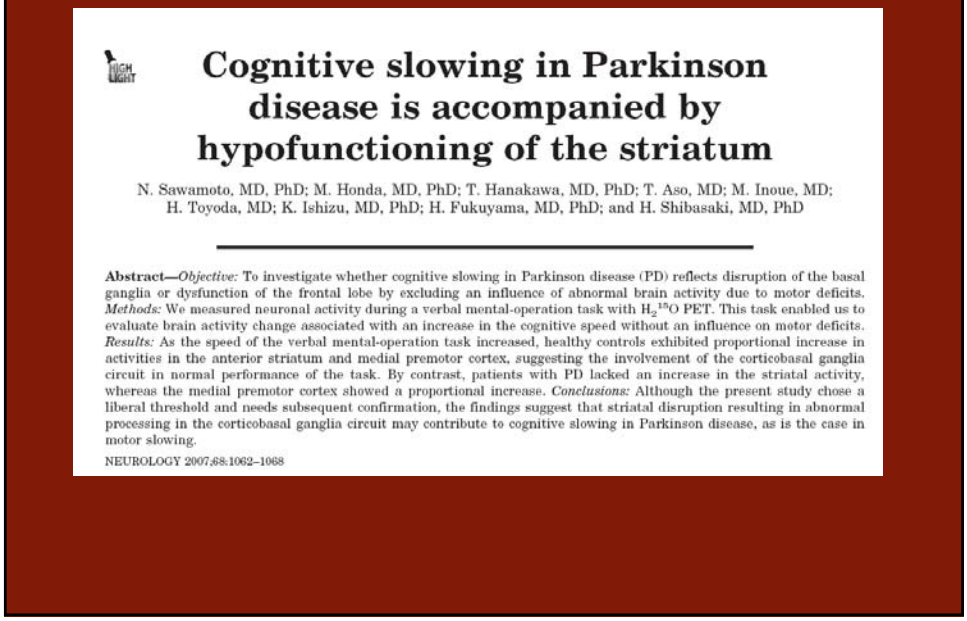
- Over 50% of PD patients experience some form of cognitive impairment
- Difficulties are typically in:
  - Attention
  - Episodic ("short-term") memory
  - Spatial cognition (perception, navigation, calculation)
  - executive functioning (problem-solving, multi-tasking)

Cognition in Parkinson's Disease

- It is common to have some word-finding difficulties and trouble multi-tasking even very early on in Parkinson's.
- Need to distinguish these from normal aging and other neurodegenerative diseases (such as Alzheimer's disease)
- Later on, some PD patients develop more severe problems with thinking and memory
  - This is what we mean by "dementia"

Three Types of Cognitive Disorder in Parkinson's Disease

- Uncomplicated PD
  - Mild subcortical cognitive impairment
  - Problems with rapid processing, memory retrieval, and multi-tasking
- Parkinson's Disease Dementia (PDD)
  - Subcortical-type dementia occurring long after the movement disorder
- Dementia with Lewy Bodies (DLB)
  - Cortical-type dementia at or near onset of movement disorder
  - Typically have AD pathology as well



Cognitive slowing in Parkinson disease is accompanied by hypofunctioning of the striatum

N. Sawamoto, MD, PhD; M. Honda, MD, PhD; T. Hanakawa, MD, PhD; T. Aso, MD; M. Inoue, MD; H. Toyoda, MD; K. Ishizu, MD, PhD; H. Fukuyama, MD, PhD; and H. Shibasaki, MD, PhD

Abstract—*Objective:* To investigate whether cognitive slowing in Parkinson disease (PD) reflects disruption of the basal ganglia or dysfunction of the frontal lobe by excluding an influence of abnormal brain activity due to motor deficits. *Methods:* We measured neuronal activity during a verbal mental-operation task with $H_2{}^{15}O$ PET. This task enabled us to evaluate brain activity change associated with an increase in the cognitive speed without an influence on motor deficits. *Results:* As the speed of the verbal mental-operation task increased, healthy controls exhibited proportional increase in activities in the anterior striatum and medial premotor cortex, suggesting the involvement of the corticobasal ganglia circuit in normal performance of the task. By contrast, patients with PD lacked an increase in the striatal activity, whereas the medial premotor cortex showed a proportional increase. *Conclusions:* Although the present study chose a liberal threshold and needs subsequent confirmation, the findings suggest that striatal disruption resulting in abnormal processing in the corticobasal ganglia circuit may contribute to cognitive slowing in Parkinson disease, as is the case in motor slowing.

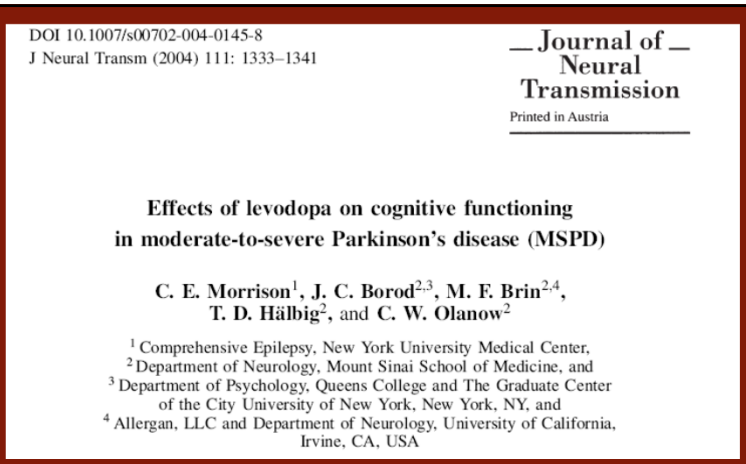
NEUROLOGY 2007;68:1062–1068

Prevalence of PDD

- Prevalence of PD
  - 500,000 Americans currently believed to have PD
  - Approximately 50,000 additional cases diagnosed each year
- Prevalence of PDD
  - Cross-sectional prevalence of dementia ranges from 24% to 40% in patients with PD
- Risk of developing dementia is 4 to 6 times higher in PD compared with similar age people without PD

Cognitive Profile in PDD

- Impaired memory retrieval
  - Benefit from external cues
  - Preserved recognition
- Executive dysfunction
  - Conceptual reasoning, problem solving, set shifting
  - Planning and sequencing
  - Behavioral self-regulation
- Attention impairment
  - Long reaction times
  - Diminished vigilance
  - Fluctuations
- Visuospatial deficit
  - Visuospatial analysis and orientation
- Bradyphrenia



DOI 10.1007/s00702-004-0145-8
J Neural Transm (2004) 111: 1333–1341

Journal of Neural Transmission
Printed in Austria

Effects of levodopa on cognitive functioning in moderate-to-severe Parkinson's disease (MSPD)

C. E. Morrison[1], J. C. Borod[2,3], M. F. Brin[2,4], T. D. Hälbig[2], and C. W. Olanow[2]

[1] Comprehensive Epilepsy, New York University Medical Center,
[2] Department of Neurology, Mount Sinai School of Medicine, and
[3] Department of Psychology, Queens College and The Graduate Center of the City University of New York, New York, NY, and
[4] Allergan, LLC and Department of Neurology, University of California, Irvine, CA, USA

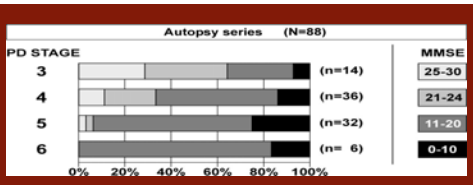
There are generally no adverse or beneficial effects of levodopa therapy on cognition in moderate-to-severe Parkinson's disease patients.

Cognitive status correlates with neuropathologic stage in Parkinson disease

H. Braak, MD; U. Rüb, MD; E.N.H. Jansen Steur, MD; K. Del Tredici, MD, PhD*; and R.A.I. de Vos, MD*

Abstract—*Objective:* To study the association of cognitive status with the stages of a published neuropathologic staging procedure for sporadic Parkinson disease (PD) in a cohort of 88 patients with PD from a single neurologic unit. None had received the clinical diagnosis of dementia with Lewy bodies (DLB). *Methods:* The authors assessed Lewy neurites/bodies (LNs/LBs) immunoreactive for α-synuclein semiquantitatively in sections from 18 brain regions. In silver-stained sections and sections immunostained for tau and β-amyloid protein, the authors semiquantitatively evaluated comorbidities potentially contributing to cognitive decline, e.g., Alzheimer disease (AD), argyrophilic grain disease (AGD), and cerebral vascular disease. The authors analyzed four Mini-Mental State Examination (MMSE) subgroups ranging from marginally impaired cognition to severe dementia using nonparametric tests. *Results:* It was possible to assign all patients to one of the PD stages. MMSE scores correlated with neuropathologic stages ($p < 0.005$) and this association showed a linear trend ($p < 0.025$). Median MMSE test scores for women were lower than those for men. Cognitively impaired individuals displayed higher stages of AD-related neurofibrillary pathology ($p < 0.05$) and β-amyloid deposition ($p < 0.05$) than cognitively unimpaired persons. MMSE scores did not correlate significantly with AGD, disease duration, age at disease onset, or age at death. Hoehn and Yahr scores, however, correlated with PD stages ($p < 0.0005$) and MMSE scores ($p < 0.0005$). *Conclusions:* The decrease in median Mini-Mental State Examination scores between PD stages 3 to 6 indicates that the risk of developing dementia increases with disease progression. In some individuals, however, cognitive decline can develop in the presence of mild Parkinson disease–related cortical pathology and, conversely, widespread cortical lesions do not necessarily lead to cognitive decline.

NEUROLOGY 2005;64:1404–1410



Dementia with Lewy Bodies (DLB)

- Also called:
  - Lewy body dementia (LBD)
  - Cortical Lewy body disease
  - Lewy body variant of Alzheimer's disease
- Similar in many ways to Alzheimer's disease
- Distinctive features:
  - fluctuating cognition
  - recurrent visual hallucinations
  - spontaneous parkinsonism early
    - rigidity and bradykinesia
    - frequent falls
  - sensitivity to medications (especially neuroleptics)
- More rapid course?

Dementia with Lewy Bodies (DLB)

- Shares features with Parkinson's and Alzheimer's diseases
- Same neuropathology (Lewy bodies) as PD, but extends into the cortex
- Unlike PDD, dementia develops at around the same time as parkinsonism
- On testing, visuospatial impairment is prominent and fluctuating attention is often observed

J Neurol Neurosurg Psychiatry 2001;70:483–488

Pentagon copying is more impaired in dementia with Lewy bodies than in Alzheimer's disease

T A Ala, L F Hughes, G A Kyrouac, M W Ghobrial, R J Elble

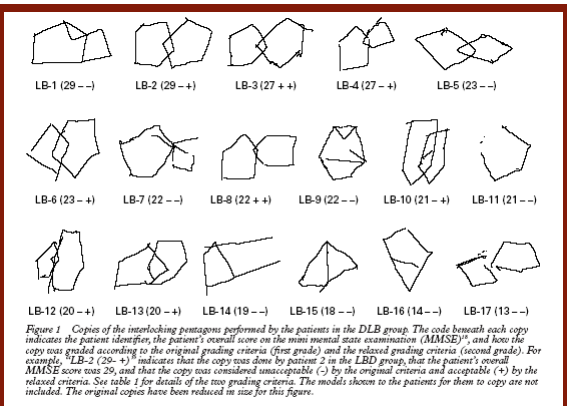


Figure 1 Copies of the interlocking pentagons performed by the patients in the DLB group. The code beneath each copy indicates the patient identifier, the patient's overall score on the mini mental state examination (MMSE)[14], and how the copy was graded according to the original grading criteria (first grade) and the relaxed grading criteria (second grade). For example, "LB-2 (29- +)" indicates that the copy was done by patient 2 in the LBD group, that the patient's overall MMSE score was 29, and that the copy was considered unacceptable (-) by the original criteria and acceptable (+) by the relaxed criteria. See table 1 for details of the two grading criteria. The models shown to the patients for them to copy are not included. The original copies have been reduced in size for this figure.

DLB and PDD boundary issues

Diagnosis, treatment, molecular pathology, and biomarkers

C.F. Lippa, MD; J.E. Duda, MD; M. Grossman, MD; H.I. Hurtig, MD; D. Aarsland, MD; B.F. Boeve, MD; D.J. Brooks, MD; D.W. Dickson, MD; B. Dubois, MD; M. Emre, MD; S. Fahn, MD; J.M. Farmer; D. Galasko, MD; J.E. Galvin, MD, MPH; C.G. Goetz, MD; J.H. Growdon, MD; K.A. Gwinn-Hardy, MD; J. Hardy, PhD; P. Heutink, PhD; T. Iwatsubo, MD, PhD; K. Kosaka, MD, PhD; V.M.-Y. Lee, PhD; J.B. Leverenz, MD; E. Masliah, MD; I.G. McKeith, MD; R.L. Nussbaum, MD; C.W. Olanow, MD; B.M. Ravina, MD; A.B. Singleton, MD, PhD; C.M. Tanner, MD, PhD; J.Q. Trojanowski, MD, PhD; and Z.K. Wszolek, MD; for the DLB/PDD Working Group*

Abstract—For more than a decade, researchers have refined criteria for the diagnosis of dementia with Lewy bodies (DLB) and at the same time have recognized that cognitive impairment and dementia occur commonly in patients with Parkinson disease (PD). This article addresses the relationship between DLB, PD, and PD with dementia (PDD). The authors agreed to endorse "Lewy body disorders" as the umbrella term for PD, PDD, and DLB, to promote the continued practical use of these three clinical terms, and to encourage efforts at drug discovery that target the mechanisms of neurodegeneration shared by these disorders of α-synuclein metabolism. We concluded that the differing temporal sequence of symptoms and clinical features of PDD and DLB justify distinguishing these disorders. However, a single Lewy body disorder model was deemed more useful for studying disease pathogenesis because abnormal neuronal α-synuclein inclusions are the defining pathologic process common to both PDD and DLB. There was consensus that improved understanding of the pathobiology of α-synuclein should be a major focus of efforts to develop new disease-modifying therapies for these disorders. The group agreed on four important priorities: 1) continued communication between experts who specialize in PDD or DLB; 2) initiation of prospective validation studies with autopsy confirmation of DLB and PDD; 3) development of practical biomarkers for α-synuclein pathologies; 4) accelerated efforts to find more effective treatments for these diseases.

NEUROLOGY 2007;68:812–819

Criteria for Probable DLB

- **Progressive cognitive decline, combined with 2 of 3 "core" features:**
  - **pronounced "fluctuations" in alertness and attention, such as frequent drowsiness, lethargy, lengthy periods of time spent staring into space, or disorganized speech**
  - **recurrent visual hallucinations**
  - **parkinsonian motor symptoms, such as rigidity and the loss of spontaneous movement**
- **Or 1 core feature with 1 suggestive feature:**
  - **Severe neuroleptic sensitivity**
  - **REM sleep behavior disorder**

Cognitive Impairment in Very Mild HD

Patients with QNE ≤ 19 and MMSE ≥ 29

Means (± SD)

	Huntington's (N=17)	Normal (N=110)	*p*
Age	49.68 (11.09)	37.07 (9.62)	<.001
Quantified Neurologic Exam	13.82 (4.00)	2.44 (2.39)	<.001
Education	15.06 (3.13)	15.05 (2.54)	.99
Mini-Mental State Exam	29.47 (0.51)	29.60 (0.49)	.61
Est. WAIS-R I.Q. (2-subtest)	110.06 (14.16)	108.68 (10.58)	.56
Brief Test of Attention	14.35 (3.14)	17.13 (2.90)	.001*
Hopkins Verbal Learning Test, delayed recall	9.80 (1.40)	11.42 (1.00)	.022*
Hopkins Verbal Learning Test, recognition discrimination	10.71 (1.26)	11.53 (0.75)	.002*
Trail Making Test, part A	38.12 (20.54)	24.59 (8.31)	.001*
Trail Making Test, part B	91.06 (39.30)	58.21 (18.837)	.001*
Stroop word reading T-score	42.35 (8.24)	48.35 (7.42)	.001*
Stroop color naming T-score	42.35 (8.82)	47.51 (7.90)	.016*
Stroop color-word T-score	42.88 (6.32)	49.15 (7.56)	.006*
Stroop interference T-score	48.59 (5.78)	50.13 (6.70)	.862*

** Age covaried*

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Archives of Clinical Neuropsychology 25 (2010) 396–409

Archives of CLINICAL NEUROPSYCHOLOGY

Episodic Memory in Dementia: Characteristics of New Learning that Differentiate Alzheimer's, Huntington's, and Parkinson's Diseases

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E-mail address: earetou1@jhmi.edu (E. Aretouli).

Accepted 4 May 2010

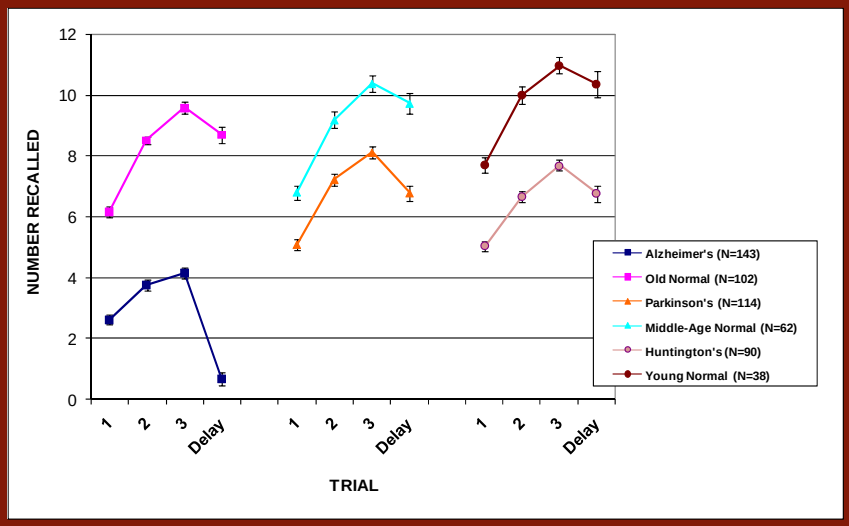
Table 1. Clinical and demographic characteristics of Study 1 participants. Means (*SD*)

	Alzheimer's disease ($n = 135$)	Huntington's disease ($n = 90$)	Parkinson's disease ($n = 111$)	*p*
Age (years)	74.64 (8.47)	50.56 (13.16)	65.78 (10.34)	<.001
Education (years)	12.83 (3.97)	14.82 (2.81)	16.57 (2.96)	<.001
Sex ratio (M:F)	55:80	40:50	75:36	<.001
Disease duration (years)	3.91 (2.82)	6.97 (4.37)	9.59 (6.62)	<.001
MMSE	19.72 (4.98)	26.13 (3.02)	27.47 (2.48)	<.001
Functional ability*	0.95 (0.49) (range = 0.50–3.00)	14.64 (10.50) (range = 0–39)	12.56 (5.90) (range = 8–30)	
Motor abnormalities**	N/A	29.91 (16.08) (range = 6–88)	26.14 (14.35) (range = 3–71)	

*AD: Clinical Dementia Rating Scale; HD: Huntington's Disease Activities of Daily Living Questionnaire; PD: Instrumental Activities of Daily Living (IADL) Scale.
**HD: Quantified Neurologic Exam; PD: Unified Parkinson's Disease Rating Scale-Part III.

Hopkins Verbal Learning Test-Revised in Neurodegenerative Disorders

education-adjusted means ± SEs



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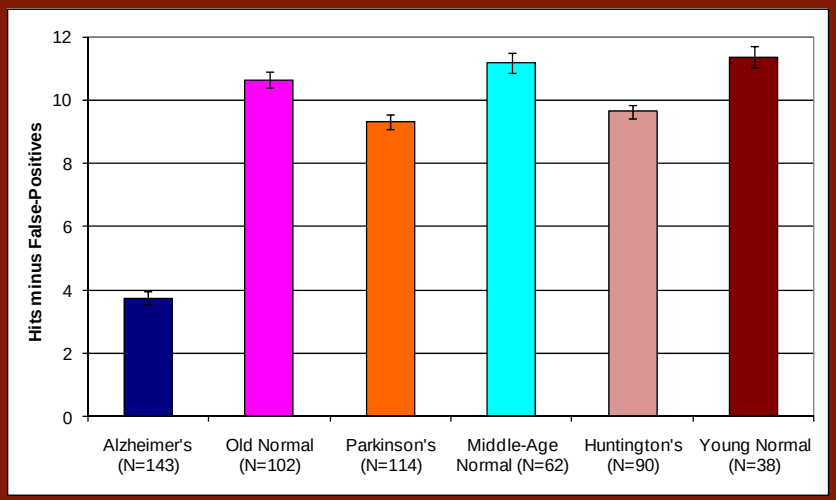
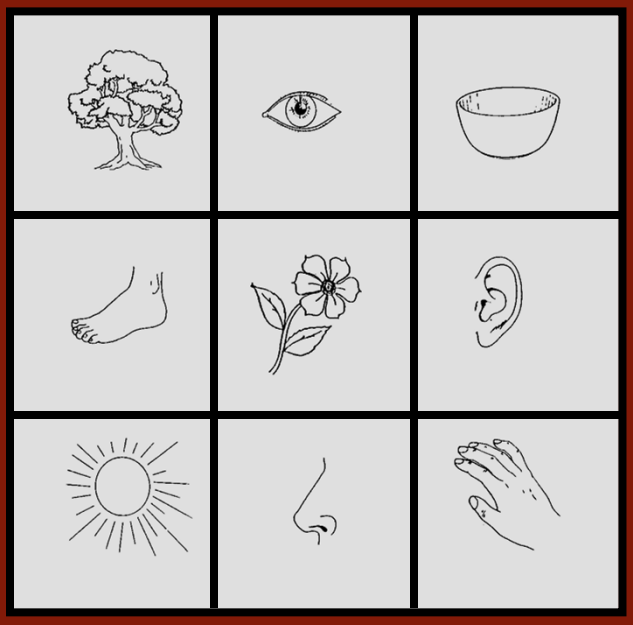


The Hopkins Board

A Culture-Fair Test of Verbal and Spatial Learning and Memory

- Naming
  - "What are these?"
- Learning
  - "Try to remember where I place these pictures."
- 20' Delayed Recall of Items
  - "What were those pictures?"
- 20' Delayed Recall of Locations
  - "Where were they?"



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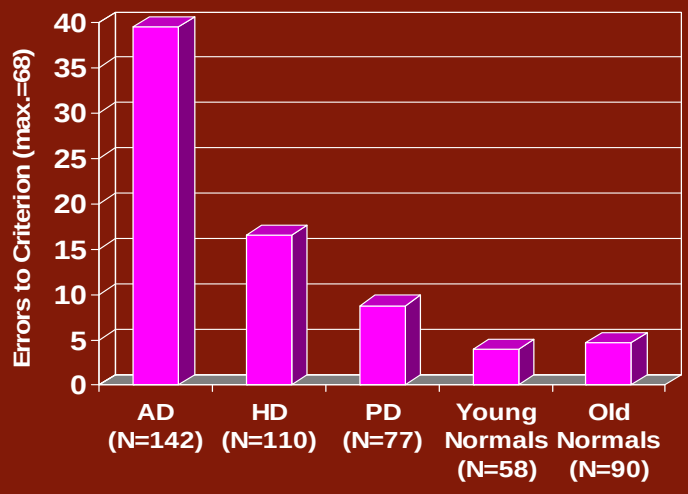
Learning Spatial Locations in Dementia

Brandt et al. (2005)



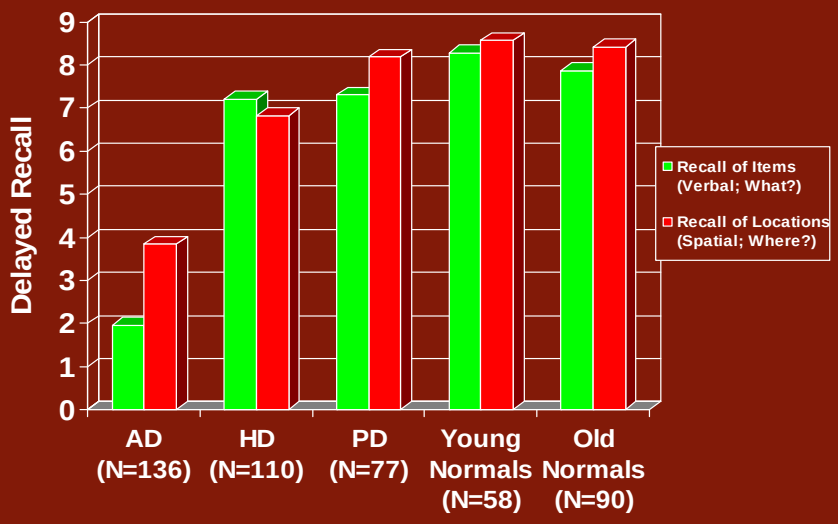
The Hopkins Board

A Culture-Fair Test of Verbal and Spatial Learning and Memory

- Naming
  - "What are these?"
- Learning
  - "Try to remember where I place these pictures."
- 20' Delayed Recall of Items
  - "What were those pictures?"
- 20' Delayed Recall of Locations
  - "Where were they?"

Verbal and Spatial Memory in Dementia

Brandt et al. (2005)



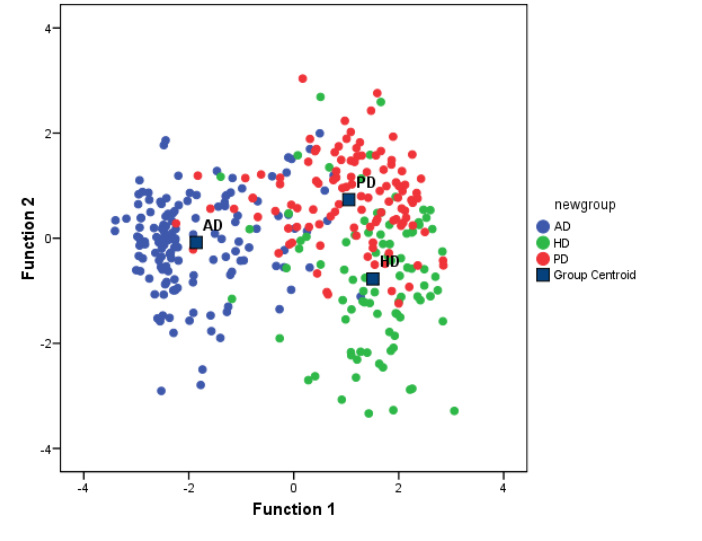
Discrimination of AD, HD and PD Using the HVLT-R and the Hopkins Board

- Age, education, and MMSE scores were entered first
- Then 3 HVLT-R variables and 5 HB variables allowed to enter stepwise (to minimize Wilk's *lambda*)
- Criterion for entry was F<.05; criterion for removal was F>.10
- Five variables were entered and retained:
  - HB delayed item recall, HVLT-R delayed recall, HB naming, HB trials to criterion, and HVLT-R sum of immediate recall trials (in that order)

Table 4. Predictors, standardized canonical discriminant function coefficients, and significance of the disease-specific discriminant analysis at each step

Step	Variable	Standardized canonical coefficients (Function 1)	Standardized canonical coefficients (Function 2)	Wilks' Lambda	*p*
1	HB delayed recall of items	0.638	0.148	0.362	<.001
2	Age (years)	−0.329	0.912	0.257	<.001
3	HVLT-R delayed recall	0.523	0.200	0.230	<.001
4	Education (years)	0.150	0.403	0.217	<.001
5	HVLT-R total learning	−0.256	0.160	0.211	<.001

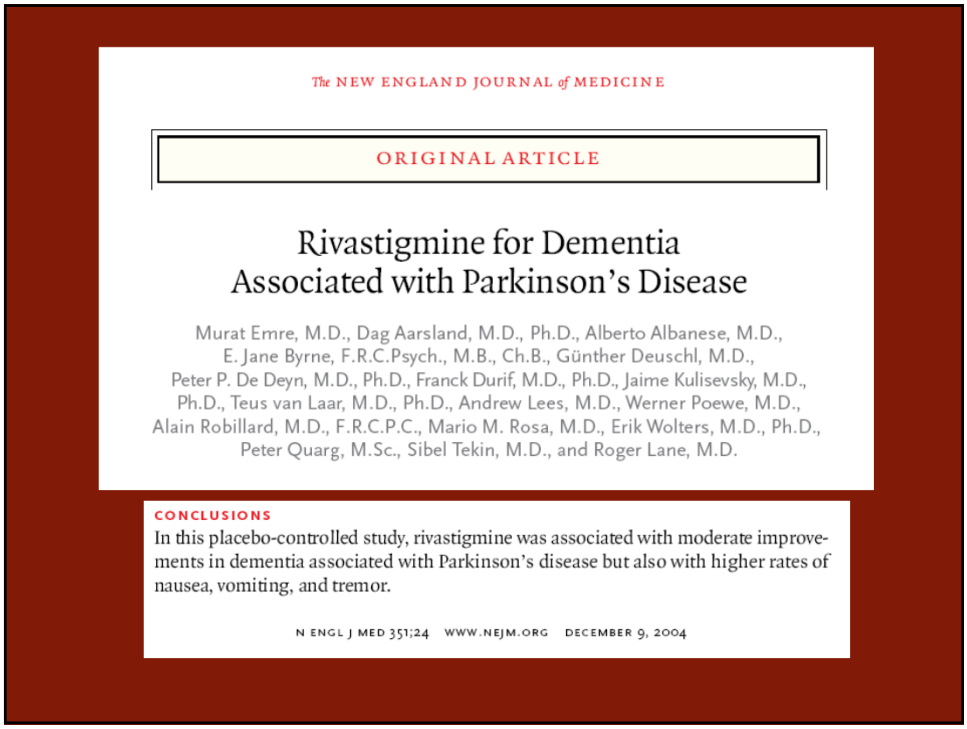
Scatterplot of AD, HD and PD Cases on Two Canonical Discriminant Functions



Discrimination of AD, HD and PD Using the HVLT-R and the Hopkins Board

		Predicted Group Membership		
		Alzheimer	Huntington	Parkinson
Original Sample	Alzheimer	122 (90.4%)	4	9
	Huntington	2	42 (66.7%)	19
	Parkinson	6	10	59 (78.7%)
Cross-Validation (hold-one-out)	Alzheimer	122 (90.4%)	4	9
	Huntington	3	38 (60.3%)	22
	Parkinson	8	14	53 (70.7%)

81.7% of original grouped cases correctly classified



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Rivastigmine for Dementia Associated with Parkinson's Disease

Murat Emre, M.D., Dag Aarsland, M.D., Ph.D., Alberto Albanese, M.D., E. Jane Byrne, F.R.C.Psych., M.B., Ch.B., Günther Deuschl, M.D., Peter P. De Deyn, M.D., Ph.D., Franck Durif, M.D., Ph.D., Jaime Kulisevsky, M.D., Ph.D., Teus van Laar, M.D., Ph.D., Andrew Lees, M.D., Werner Poewe, M.D., Alain Robillard, M.D., F.R.C.P.C., Mario M. Rosa, M.D., Erik Wolters, M.D., Ph.D., Peter Quarg, M.Sc., Sibel Tekin, M.D., and Roger Lane, M.D.

CONCLUSIONS

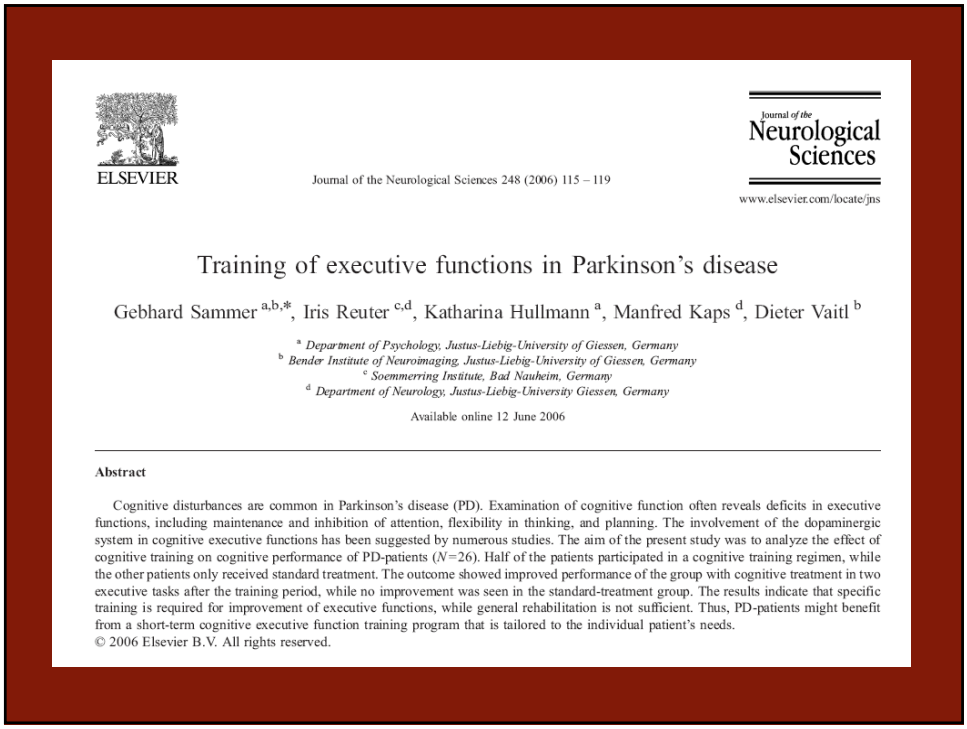
In this placebo-controlled study, rivastigmine was associated with moderate improvements in dementia associated with Parkinson's disease but also with higher rates of nausea, vomiting, and tremor.

N ENGL J MED 351;24 WWW.NEJM.ORG DECEMBER 9, 2004

Cholinesterase Inhibitors for Parkinson's Disease Dementia

Cochrane Database of Systematic Reviews , 2007

- Rivastigmine appears to improve cognition and activities of daily living in patients with PDD.
- *This results in clinically meaningful benefit in about 15% of cases.*



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Training of executive functions in Parkinson's disease

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Available online 12 June 2006

Abstract

Cognitive disturbances are common in Parkinson's disease (PD). Examination of cognitive function often reveals deficits in executive functions, including maintenance and inhibition of attention, flexibility in thinking, and planning. The involvement of the dopaminergic system in cognitive executive functions has been suggested by numerous studies. The aim of the present study was to analyze the effect of cognitive training on cognitive performance of PD-patients (N=26). Half of the patients participated in a cognitive training regimen, while the other patients only received standard treatment. The outcome showed improved performance of the group with cognitive treatment in two executive tasks after the training period, while no improvement was seen in the standard-treatment group. The results indicate that specific training is required for improvement of executive functions, while general rehabilitation is not sufficient. Thus, PD-patients might benefit from a short-term cognitive executive function training program that is tailored to the individual patient's needs.
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