

**Dementia in Parkinsonism**  
**Prepared for Michigan Dementia Coalition by**  
**The Professional Advisory Board of the Michigan Parkinson Foundation**  
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Parkinson's disease is considered the second most common neurodegenerative disorder, following Alzheimer's disease. While it is a neuromuscular movement disorder characterized by stiffness of the muscles (rigidity), slowness of movement (bradykinesia), resting tremor and problems with balance, it often presents with complex affective, psychiatric, and cognitive disorders. These include depression; anxiety, panic disorders, psychoses and dementias.

There is a growing recognition that non-motor symptoms of Parkinson's disease [PD], including dementia, play a significant role in the management and quality of life of individuals with PD and their families. When dementia develops, it is a major cause of caregiver stress, disability and mortality. (Barbas (2006), Aarsland (2007)). Most epidemiological studies suggest that PD reduces life expectancy despite different management methodologies (Mortality hazard ratios are between 1 to 5 and 2 to 7). Dementia, according to Lonneke (2006), seems largely responsible for the reduced life expectancy of patients with PD.

The prevalence of Parkinson's disease is reported as 300 cases per 100,000 population.(1) In Michigan, this translates to about 30,000 individuals. (Dr. Albin: Gutmann (4), Nebraska registry). The risk of developing dementia among individuals with Parkinson's disease is 4-6 times higher than the rest of the population. This risk is higher among persons older than 70 who have a 40-70% greater chance of developing dementia. Based on conservative estimates, about 25-40% of people diagnosed with PD will develop dementia. In Michigan, therefore, approximately 10,000 people with PD also have a significant dementia. This may well be an under-estimation. (see discussion: Dr. LeWitt; Lonneke – Appendix).

This Michigan Parkinson Foundation Professional Advisory Board Report will discuss the impact of dementia in Parkinsonism disorders, suggest future directions in the provision of medical and rehabilitation services, and recommend increased interagency cooperation in the State of Michigan.

(1) This figure is used by the Michigan Parkinson Foundation on the advice of its Professional Advisory Board. NINDS statistics list the rate as 172 per 100,000. It is the opinion of the PAB that this is an underestimate and that the extrapolation MPF should use is based on more current information and research. The Michigan Dementia Coalition reports base their statistics on a rate of between 150 and 200 per 100,000, suggesting a prevalence estimate of 20,000 in Michigan.

## **Dementias in Parkinsonism**

There are at least three dementia syndromes associated with PD: Alzheimer's disease [AD], Diffuse Lewy Body disease [DLBD], and Parkinson's disease dementia [PDD]. Alzheimer's disease is the leading cause of dementia in the general population and can also be seen in persons with Parkinson's disease. Alzheimer's dementia may develop in persons with Parkinson's disease and tends to occur among the elderly later in the course of the movement disorder. Diffuse Lewy Body Disease [DLBD] is considered the second most common cause of dementia in the general population and is often confused with Parkinson's disease due to its Parkinsonian symptoms. A diagnosis of Lewy Body Dementia [DLBD] should be considered if the movement disorder and the cognitive disorder develop within one year of each other. Features of Lewy body dementia [DLBD] also include visual hallucinations with day to day fluctuation of symptoms.

When the clinical features of Parkinsonism and dementia coexist, the diagnosis of dementia can be complex because of the significant overlap among the disease. (Barbas, 2006). For example, persons with vascular dementia can develop a gait disorder similar to Parkinson's. Other conditions with different etiologies yet similar symptoms include Normal Pressure Hydrocephalus [NPH] and the Parkinson's Plus Syndromes [PPS]. The Parkinson's Plus syndromes include progressive supranuclear palsy [PSP], multisystem atrophy [MSA], corticobasal ganglionic degeneration [CBD] and dentatorubical pallidolusian atrophy [DPA]. (see Appendix for descriptions). Persons with Parkinson's Plus Syndromes have a more severe course, fewer available treatment options, and reduced longevity.

### **Differentiating Parkinson's Disease Dementia [PDD] from Alzheimer's Dementia [AD]**

Parkinson's disease dementia [PDD] is a subcortical dementia that occurs more than one year after the diagnosis of Parkinson's disease. The following lists the chief differences of PDD and other dementias:

1. Type of cognitive changes
  - AD is a cortical dementia. The outer layers of the brain, concerned with processing information and connecting functions such as language and memory (are involved and) ...there are characteristic problems with memory, the inability to recall words and as the disease progresses the ability to understand what others are saying (aphasia)." (About.com. Alzheimer's)
  - PDD is a subcortical dementia which results from structures involved below the cortex. There is a less severe loss of intellectual and memory function and a lack of aphasia, agnosia, and apraxia typical of the cortical dementias
2. The profile of neuropsychiatric symptoms common in PDD differs from that in other dementias.
  - Less memory impairment compared to AD
  - Inattention
  - Visuospatial abnormalities
  - More apraxia, psychosis and motor problems
  - Frequent visual hallucinations
3. More visual hallucinations due to medications in PDD
  - management of hallucinations
    - i. reduction in PD medication
    - ii. addition of atypical antipsychotics.

1. PD symptoms worsen significantly
2. risk of death is greater
4. Wandering tendency is reduced in PDD vs AD
5. Verbal cueing is more helpful in PDD than AD
6. PDD has greater difficulties with sleep, orthostatic hypotension and falls.
7. Although people with AD can have parkinsonian symptoms, these are never apparent in the early and mid disease.
8. LBD can exhibit early parkinsonian symptoms, but dementia is present before or shortly after diagnosis as opposed to late in the disease.
9. Medication management differs significantly in Parkinson's disease dementia compared to other dementias.

## **Management of Dementia in Parkinsonism**

The recognition, care, and pharmacologic treatment of people with dementia in Parkinson's disease can be significantly different than dementia resulting from other conditions. The following areas are problematic:

1. Diagnosis and misdiagnosis of Parkinson's disease and concomitant neuropsychiatric conditions.
  - lack of inclusion of testing to improve early recognition of neuropsychiatric disorders. Improvement can result in early treatment (pharmacologic and non-pharmacologic) monitoring and prevention of further disabilities.
  - need to rule out other dementing illnesses
2. Complex pharmacologic treatment
  - inappropriate medication regimen (inadequate use of antiparkinsonian drugs)
  - need for continual monitoring and change in PD treatment (may need to reduce medications as opposed to addition) – this is different from other dementing illnesses
  - inappropriate use of contraindicated medications
  - addition of appropriate atypical neuroleptics – different from other dementing illnesses
  - need for treatment of depression and psychosis
3. Symptom management
  - sleep disorders
  - balance problems
  - management of non-motor symptoms
4. Need for additional non-pharmacologic testing, treatment and care
  - lack of referrals to psych
  - cognitive rehabilitation
  - patient education – coping strategies
  - psychological and psychiatric counseling
  - rehabilitative therapies
  - benefits of exercise, stress management, other
  - note: difficulty in moving results in rigidity and reduced cooperation
  - Other complicating factors in PDD: dehydration, constipation, minor infections, improper use of drugs

Clinically, PDD, LBD and AD with parkinsonism can be understood as a spectrum of disorders with overlapping symptomatology. The Professional Advisory Board of the Michigan Parkinson Foundation believes that these related disorders of Parkinson's disease will play a prominent role in its future programs

## **Role of Public Associations and the State Government**

There are number of specific areas of programming that can be provided by public associations and State agencies to improve our understanding and clinical services in Parkinsonism related dementias. Some of these areas include:

1. Need to improve patient and family knowledge and support
  - a. Education re: disease, treatment, family and patient responsibilities, medication management, what dementia is, resources available, care and living options
  - b. Specific training in skills to manage neuropsychiatric symptoms in addition to motoric symptoms; how to deal with lack of cooperation in performance of activities of daily living.
  - c. Support for family, including lack of appropriate respite care options
2. Need to improve public information about PDD
  - a. Few programs available for support groups
  - b. Limited information in public domain
3. Need to provide education regarding patient care for hospitals, respite and long term care facilities
  - a. Families have noted that nursing homes and respite care facilities that are "dementia specific" have difficulty dealing with specific cognitive problems in addition to cardinal PD symptoms.
  - b. There is a need to educate patients and providers about the appropriate use of palliative care and hospice. These services are probably underutilized in persons with advanced Parkinson's.
4. Need for more study regarding the difference in management of dementia in PD and AD

## **Discussion**

To our knowledge, there will not be grant funding in the fiscal year of 2008 for this project. The Michigan Parkinson Foundation recommends that, regardless, there be more information about Lewy Body Dementia and Parkinson's Disease Dementia in Dementia Network materials, and that further study be done to examine the emerging recognized differences and needs between the dementias.

We believe that there is a need to explore more ways to collaborate with other organizations to promote awareness of Parkinson's Disease Dementia and the other dementias and to provide services and programs sensitive to the provision of care for individuals with the non-Alzheimer's dementias.

We would also recommend addressing the following priority areas, should a grant of \$15,000 become available:

- 1) The Primary Care Physician Network to work with the Michigan Parkinson Foundation Professional Advisory Board and other neurologists with such expertise to begin to incorporate information and training for the primary care physician that will help practitioners to identify non-Alzheimer's dementias and initiate appropriate treatment and referrals.
- 2) Development of educational materials and programs specific to those individuals and families confronting Parkinson's Disease Dementia and Lewy Body Dementia, as well as the other non-Alzheimer's dementias (Parkinson's Plus and other conditions) to assist in understanding and managing the changes occurring with dementia.

### **Conclusion**

The Professional Advisory Board of the Michigan Parkinson Foundation recommends that we work with the **Dementia Coalition**, the **Michigan Public Health Institute** and the **Geriatric Education Center of Michigan** to improve the recognition, diagnosis and care for persons and their families who have Parkinsonism disorders with dementia in Michigan.

The following programs and services are needed:

1. Physician education (diagnosis and management)
2. Multidisciplinary team approach (to include mental health resources, caregiver assessment and supportive treatment)
3. Hospital, home care, respite and nursing home education
4. Patient and family education
5. Neuropsychiatric testing
6. Psychiatric and other mental health services and support
7. Training programs for care givers in extended care and long term care facilities and hospitals.
8. Study difference in management of dementia in PD and AD in care settings (non-pharmacologic as well as pharmacologic)
9. Identification of appropriate resources for people with PDD vs other dementias.- Survey of Long Term Care, respite, etc. that are trained in the management of PD and PDD in addition to AD.
10. Programs focusing on promotion of health for patient and family members (exercise, stress management, sleep hygiene, nutrition, coping, others)

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## **Appendices:**

### ***Prevalence:***

Prevalence of PD is 300/100,000 (Gutmann's study – Ontario; Nebraska registry data). This would give about 30,000 in Michigan, 10,000 demented (R. Albin).

Having followed this literature over the years (and discovering that Bruce Schoenberg's door-to-door prevalence studies in Copiah County, MS, haven't been much improved upon), I would agree that we have a ball park estimate of how frequently PD appears in the general population – but a few points are worth considering:

- 1) Given the typical age of symptom onset (approximately 58 years in clinics throughout Western Europe and the US), the “baby boomer” generation will impart a bulge to the prevalence data that will not be a linear projection from the past decade.
- 2) Diffuse Lewy body disease can develop in the absence of obvious Parkinsonism, imparting dementia (and sometimes only later Parkinsonism). Such cases can have pathological features found in typical PD cases (but not at the threshold for causing motor symptomatology). Such cases would also be the target for neuroprotective therapeutics, despite not being diagnosed PD.
- 3) Parkinsonian signs in an aging population are more common than the diagnosis of PD by conservative criteria in the same population, (see for example: Bennett DA, Beckett LA., Murray AM, Shannon KM, Goetz CG, Pilgrim DM, Evans DA. Prevalence of parkinsonian signs and associated mortality in a community population of older people. *N Engl J Med* 1996; 334:71-76 (and correspondence *N Engl J Med* 1996;334:1611) hence, prevalence data could well be major underestimates.

### ***Types of Dementia***

a. Alzheimer's Disease (AD) is a progressive and fatal brain disease, affecting more than 5 million Americans. Alzheimer's destroys brain cells, causing problems with memory, thinking and behavior severe enough to affect work, lifelong hobbies or social life. Alzheimer's gets worse over time, and it is fatal. Today it is the seventh-leading cause of death in the United States. AD is the most common form of dementia, a general term for the loss of memory and other intellectual abilities serious enough to interfere with daily life. Symptoms include: Memory loss, difficulty performing familiar tasks, problems with language, disorientation to time and place, poor or decreased judgment, poor abstract thinking, misplacing things, changes in mood or behavior including rapid mood swings, changes in personality, and loss of initiative.

b. Diffuse Lewy Body Dementia – 2<sup>nd</sup> most common progressive dementia of old age, often attended by Parkinsonism. Symptoms include: well-formed visual hallucinations, non-visual hallucinations (auditory), fluctuations in cognition, vigilance (systematized delusions). Dementia is visual-perceptual and attentional-executive. Depression, when present, can be treated. The individual is intolerant to neuroleptics, and may have syncope and falls. The age of onset is generally 60-68 and the duration of disease, 6-7 years. It is more frequent in men than women. Depression can be treated.

Parkinson Plus syndromes – A key to identifying these syndromes is that individuals generally lack a good response to dopaminergic treatment and have additional clinical features than the usual Parkinson's symptoms. The course of the disease is usually of shorter duration, leading to death. These syndromes are difficult to diagnose and treat. Most of those diagnosed have symptoms that start when the individual is in his 60's..

- a. Progressive Supranuclear Palsy (PSP) – Dementia occurs later in disease, The prevalence is 6.4 cases per 100,000. the average age of onset: 63 years, with it occurring in men more frequently than women. Survival is 6-7 years after diagnosed. The signs include: early broad-based and stiff gait disorder with backward falls and supranuclear gaze palsy with slow vertical saccades and difficulty looking down. There is more axial than limb rigidity, nuchal dystonia, facial spasticity with exaggerated nasolabial folds, dysarthria, dementia and poor levodopa response. Dementia is experienced in over half of those diagnosed. Behavioral changes include apathy, irritability, and labile mood. Psychosis is rare but there may be a mild depression and anxiety. Sleep disturbances are common.
- b. Multisystem Atrophy (MSA) – Attention needs to be paid to Parkinsonism vs cerebellar dysfunction. This complex of disorders are sporadic, progressive disorders of adult onset. Parkinsonism is seen in the 3 principal types.
  - Striatonigral degeneration (SND, MSA-P) – Parkinsonism negligibly responsive to levodopa.
  - Striatonigral degeneration – bradykinesia, rigidity, poor levodopa response and early autonomic dysfunction. Hyper-reflexia, anterocollis, various tremors besides resting tremor, sleep apnea and respiratory stridor. Rapid progression. Cognitive impairment has been observed: visuospatial organization, poor frontal lobe attention tasks
  - Shy Drager Syndrom (SDS, MSA-A) – Parkinsonism with prominent autonomic disturbance: Orthostatic syncope, impotence, urinary bladder dysfunction, Parkinsonism, dysarthria, incontinence and constipation. Pyramidal signs are present in 50% with eventual development of emotional lability, vocal cord stridor, obstructive sleep apnea. There generally is no cognitive disorder. Depression may be seen initially. REM Behavioral Disorder is common.
  - Olivopontocerebellar atrophy (OPCA, MSA-C) – prominent upper motor neuron and cerebellar signs. This disorder exhibits Parkinsonism with cerebellar ataxia. Prominent ataxia with relatively mild Parkinsonism, but progresses to involve impaired eye movements, dysarthria, dysphagia, incontinence, upper motor neuron signs, and lower motor neuron signs. Horizontal nystagmus, impaired upgaze and convergence and loss of vestibule-ocular reflex. Auditory hallucinations, paranoia, and depression. Dementia is possible.

Dysautonomia is characterized by urinary and erectile dysfunction, RBD. Diagnosis is established when at least 6 of the following are present: sporadic adult onset, autonomic signs, Parkinsonism, cerebellar features, pyramidal signs, lack of levodopa response, lack of cognitive dysfunction, lack of downward gaze palsy.

- c. Cortico-basal Ganglionic Degeneration (CBD) – gradually progressive condition with unilateral akinesia and rigidity responding poorly to levodopa, and apraxia. It is associated with cortical reflex myoclonus, limb dystonia, alien limb sign and occasional cortical sensory loss. Prevalence 4.9 to 7.3 cases per 100,000. Mean age of onset 63 with a 7.7 year course. Early frontal dysfunction. Aggressive behavior, disinhibition and Kluver-Bucy syndrome.
- d. Normal pressure hydrocephalus (NPH) is an abnormal increase of cerebrospinal fluid (CSF) in the brain's ventricles, or cavities. It occurs if the normal flow of CSF throughout the brain and spinal cord is blocked in some way. This causes the ventricles to enlarge, putting pressure on the brain. Normal pressure hydrocephalus is rare, but occurs most often in the elderly population. Symptoms of NPH include progressive mental impairment and dementia, problems with walking, and impaired bladder control leading to urinary frequency and/or incontinence. The person also may have a general slowing of movements or may complain that his or her feet feel "stuck."
- e. The most common type of vascular dementia, multi-infarct dementia (MID), includes vascular dementia due to lacunar lesions. Lacunar lesions are small infarcts resulting from blockage of blood flow in penetrating arteries. Penetrating arteries are found in several places in the brain including the diencephalon, where the basal ganglia are located. The basal ganglia are key structures in the neuronal circuitry affected in Parkinson's disease, and multiple lacunar lesions in the basal ganglia can give rise to the same symptoms as Parkinson's disease.
- f. Huntington disease (HD) is inherited as an autosomal dominant disease that gives rise to progressive, selective (localized) neural cell death associated with choreic movements and dementia. The disease is associated with increases in the length of a CAG triplet repeat present in a gene called 'huntingtin' located on chromosome 4p16.3. Juvenile-onset Huntington disease, 5 to 7% of all HD cases, typically shows rigidity, hypokinesia and seizures. It is usually transmitted from an affected father, and is associated with very large CAG repeat sizes (60 or more) in the HD gene.
- g. Dentatorubral-pallidoluysian atrophy is caused by an expanded trinucleotide repeat in the DRPLA gene, and causes a syndrome of myoclonic epilepsy, dementia, ataxia, and choreoathetosis. At autopsy, major neuropathologic changes consisted of combined degeneration of the

dentatorubral and pallidolusian systems. Inheritance is autosomal dominant. Three clinical forms of DRPLA have been described: the ataxo-choreoathetoid form, the pseudo-Huntington form, and the myoclonic epilepsy form. Patients with fewer numbers of trinucleotide repeats can present with a late onset ataxia and dementia that may appear Parkinsonian.

#### Footnotes:

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(Please note that Barbas' work provided significant content to this paper)

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