CASE STUDY: Nutritional Considerations in Patients with Parkinson’s Disease

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INTRODUCTION

I selected Parkinson’s disease (“PD”) and its nutritional implications because of my keen interest in neuromuscular conditions in general, and specifically due to the fact that the illness has recently struck one of my closest friends. In my previous career as a general dentist, with a practice focused on older adults, I unfortunately witnessed first-hand the decline of a handful of my dental patients who were stricken with PD. In my recent brief but productive and enlightening rotation at St. Francis Country House in Upper Darby, Pennsylvania, I interacted with and assessed a gentleman who was in the long-term care section of the facility and whose primary admitting diagnosis was PD.

In choosing PD as my case study topic, I wanted to learn more about the nutritional implications of the disease and perhaps how a patient’s nutritional requirements may change over time due to the pathogenesis of the disease process. During my research in the peer-reviewed literature, books, brochures, and websites, I was hoping to discover whether or not this disease had any unique features, compared to other neuromuscular and neurodegenerative conditions, which would trigger a commensurate nutritional therapy response.

Like many other neurodegenerative diseases, there is no known cure for PD, only presently a containment strategy involving: 1) medications; 2) exercise; 3) physical, speech, and/or occupational therapies when warranted; and, 4) a sound nutritional regimen including proper hydration. From a dietitian’s perspective, I am hoping to discover a greater understanding into the nutritional requirements of patients with Parkinson’s disease, and in the process, perhaps ultimately being able to help my good friend to live a more productive and healthy life, for as many years possible.
ABSTRACT

PD is the second leading age-related disease in the world behind only Alzheimer’s disease\(^1\), and is the second-most common neurodegenerative disease, behind only multiple sclerosis, in high-income nations\(^2\). It is characterized by a progressive degeneration of dopamine-producing neurons in the brain, which results in both cognitive and motor impairment, and ultimately dementia.

One of the most significant nutritionally-related consequences for many Parkinson’s patients is weight loss due to malnutrition, which is often exacerbated by the medications used to treat the disease\(^1\). Against the backdrop of an actual PD patient – more precisely, a long-term resident in a local neuro-rehabilitation facility - whom I had the privilege of assessing, this case study explores the disease from many perspectives. The epidemiology, etiology, pathophysiology, clinical manifestations, staging, treatment modalities, research, and nutritional implications of PD are discussed in detail.

Medical nutrition therapy, as it addresses both the disease process itself, with its clinical signs and symptoms, as well as the implications of the medications often employed to combat the progression of the disease, is outlined using the approach of the Nutrition Care Process. Topics in nutrition-based medical research are unveiled, discussing the possible use of micronutrient supplementation as an adjunct to pharmaceutical, surgical, and dietetic strategies.

As the baby-boomer generation enters retirement, the number of Americans diagnosed with PD will undoubtedly increase, and thus it is incumbent upon the dietitian to be familiar with the disease and how to assist the sufferer in maintaining a high quality of life through good nutrition.
DISCUSSION OF MEDICAL CONDITION

PD is a chronic progressive neuromuscular condition which has an unknown etiology and presently no known cure. In high-income countries, neurodegenerative diseases including PD are the seventh leading cause of death and a major contributor to disability. Aging appears to be a major risk factor, with 80-85% of all cases occurring over the age of 60, and the incidence increases more than five-fold for individuals in the 70-79 age range. The number of men over the age of 70 diagnosed with PD is fifty percent greater than in women of the same age.

Approximately one percent of Americans over the age of 70, more than one million individuals, are afflicted with this condition, and the average life expectancy after diagnosis is approximately 13 years. The older the person at the age of diagnosis of PD, the more rapid the progression of the disease.

Although the etiology of PD remains somewhat nebulous to this day, the most commonly postulated causes of this affliction include: 1) genetic mutation; 2) exposure to pesticides and environmental toxins over a long period of time; 3) oxidative stress; 4) protein malformations, known as prions, within dopaminergic neurons; 5) mitochondrial dysfunction; 6) altered inflammatory response in the brain; 7) neurotransmitter imbalance; and, 8) decreased ability of the body to reproduce dopamine-producing neurons.

Genetic mutations and single-gene defects are responsible for some cases of PD. The functions of the proteins from the affected genes differ widely, and as expected the clinical features of the protein/gene expression vary as well, affecting the age of onset, the type of dementia, psychiatric features, and motor disturbances. The actions of the proteins transcribed from genetic mutations mimic those of environmental neurotoxins, and thus studying the genetic
mutations in cases of familial or inherited PD has opened up new vistas of research into the causes of PD. Prolonged exposure to pesticides and environmental toxins have been reported to contribute to the destruction of dopaminergic neurons, as well as being linked to many of the motor deficits seen in PD. Paraquat and rotenone, two potent pesticides, as well as organic toxins such as toluene, cyanide, and carbon disulfide, are theorized to negatively interact with gene expression, perhaps modulating the expression of mutated genes, although presently the mechanism of this interaction remains unclear.

Unique biochemical features of the substantia nigra render this structure more vulnerable to oxidative stress due to the enhanced presence of iron. In its ferrous form, iron acts as a cofactor in the creation of free radicals from the oxidation of dopamine by the enzyme complex known as monoamine oxidase B. The proliferation of vast quantities of free radicals can wreak havoc on the dopaminergic neurons in the substantia nigra through oxidative stress, and this pathological process may be the most important etiologic factor in the progression of PD. Post-mortem examinations of the brains of PD patients reveal substantially elevated levels of oxidative stress.

Protein malformations, known as prions, are evident on the cellular level within dopaminergic neurons in patients with PD. Specifically, proteins such as alpha-synuclein and beta-amyloid, according to some studies, may have a causal role in the progression of PD when they become misfolded. The inability to perform their respective functions lead to cellular necrosis and the ultimate loss of dopaminergic neurons.

Mitochondrial dysfunction is another suspected etiological factor in the progression of PD. Neurotoxins such as MPTP are known to inhibit normal functioning of the electron transport chain in mitochondria, which leads to ATP depletion and accumulation of reactive oxygen
species, a particularly virulent type of free radical whose reduced form serves as an agent for oxidative phosphorylation in the healthy individual.

An altered and exaggerated inflammatory response is often seen in the brains of PD patients. Special support cells in the brain known as micarglia are activated, which produce immune signaling substances known as cytokines. The cytokines act as pro-inflammatory agents which can produce necrosis of dopaminergic neurons.

Neurotransmitter imbalance occurs when levels of the excitatory neurotransmitter dopamine become depleted, which produces a relative preponderance of the inhibitory neurotransmitter gamma amino benzoic acid (GABA). Since the maintenance of slow coordinated muscle movement is contingent upon a balance of these neurotransmitters, the destruction of dopaminergic neurons produces the wide spectrum of motor and cognitive impairment seen in most PD patients.

Decreased dopamine-producing neurons naturally occurs with the aging process but PD patients suffer from the disproportionate loss of dopaminergic neurons due to the variety of factors cited above. The pathophysiology of the disease emanates from the destruction of dopaminergic neurons in the substantia nigra portion of the brain, which is anatomically imbedded within the midbrain, deep within the cerebral hemispheres and beneath the cerebral cortex. This part of the brain is responsible for directing the body’s coordinated muscle movements and balance. In normal physiology, there is a balance between excitatory and inhibitory neurons that ensures slow, predictable, coordinated muscle movement, posture, and balance. The progressive loss of dopamine, the excitatory neurotransmitter, creates an imbalance with the inhibitory neurotransmitters, most notably gamma amino benzoic acid (GABA), and thus the cluster of motor symptoms of PD are manifested.
The substantia nigra is more vulnerable to oxidative stress than other portions of the brain, partly due to its higher iron content. Dopamine, the neurotransmitter that facilitates the complex coordinated process of movement and balance, can be oxidized by a monoamine oxidase enzyme in the presence of ferrous iron, thus producing large quantities of hydroxyl free radicals which damage the dopaminergic neurons. In later stages of the disease, a form of dementia with Lewy bodies – aggregations of proteins resulting from disruption of their tertiary structures within the neuronal cellular cytoplasm - can appear, producing clinical unresponsiveness and sluggishness. Autopsies on the brains of deceased PD patients often reveal elevated levels of oxidative stress; on a cellular level, the abnormalities in PD are evidenced by dysfunction of the mitochondria and oxidative stress, as defects in the electron transport chain can yield increased free radical-induced cellular damage and destruction of energy pathways.  

There is a pathognomonic set of signs and symptoms that often is used to assist in the diagnosis of Parkinson’s, and the acronym “TRAP” can be used to remember them: tremors, rigidity, akinesia (loss of movement), and postural instability. In addition, bradykinesia (slowed movement), mask-like facial features, shuffling gait, difficulty stopping and turning, and stooped posture are often seen. It has been reported that movement-related symptoms occur when approximately 60-80% of the dopaminergic neurons have been destroyed. The cumulative effect of these movement and balance disturbances increase the risk for falls and bone fractures.

Non-motor symptoms are often seen prior to the onset of motor disturbances, and they may include:

- cognitive dysfunction including memory loss and inability to complete tasks and recall information;
- anxiety and depression;
• sensory dysfunction;
• sleep disturbances.

PD has been staged according to two systems, the Hoehn & Yahr, and the Unified Parkinson’s Disease Rating Scale [UPDRS], which evaluate behavior, mood, cognition, motor skills, and activities of daily living. In the Hoehn and Yahr model, there are 5 progressive stages of PD with specific symptoms and corresponding nutrition concerns:

• Stage 1: mild, unilateral, non-disabling symptoms where some changes are noted in the patient’s facial expressions, posture, and gait. Nutritional concerns are focused on medication-induced constipation, nausea, and diminished taste and smell perception.
• Stage 2: bilateral physical symptoms, minimal disability but noticeable gait and posture regression. Unintentional weight loss, GERD, and gastroparesis join the list of nutritional concerns.
• Stage 3: significant bradykinesia, problems with equilibrium and balance, and generalized dysfunction symptomology. Dysphagia becomes another nutritional concern at this stage.
• Stage 4: severe symptoms, including limited walking and independence, and muscle rigidity. Pronounced decrease in manual dexterity resulting in reduced self-feeding and possible dehydration become concerns to be addressed by the dietitian.
• Stage 5: patient may become confined to a bed or wheelchair, with continual nursing care and inability to stand and walk. Cachexia is often seen at this stage, and nutrition support is often required.

Symptoms of PD more specifically related to diet and nutrition, which will be discussed in greater detail in the next section of the paper, can be classified into two main categories:
1. GI motility: gastroparesis and slowed peristalsis of the gastrointestinal tract with delayed transit time through the colon, leading to hemorrhoids, fecal impaction, and constipation.

2. Muscle dysfunction: decreased tongue movements, incomplete closure of the lips, and weakening of the lower esophageal sphincter, which can produce dysphagia, choking, aspiration, GERD, Barrett’s esophagus, and delayed medication absorption.\(^{10}\)

From a research perspective, primarily owing to the potential interaction of toxic environmental chemicals on gene expression and mutation, gene mapping and genetic analysis is currently a major area of research into PD. Nearly a dozen single gene defects have been identified in causing a number of PD cases. Other theorized causes include the long-term use of certain medications such as Reglan and some tranquilizers, but no direct cause-and-effect has ever been proven. The investigation of the potential neuro-protective effects of nutraceuticals via free-radical scavenging, inhibition of inflammatory pathways, regulation of cell-signaling pathways, and iron chelation, is another major focus of research efforts.\(^{8}\)

Current medical treatment modalities for PD patients are classified as either primarily pharmaceutically based, or surgical intervention. Since the symptoms of PD are directly caused by an inadequate supply of dopamine, the aim of pharmaceutical therapy is to somehow improve the supply of dopamine to the brain. Medications form the central strategy in managing the symptoms of PD, and they fall into several categories based on method of action.\(^{5,6,10}\) Note that some medications are used in combination, which has become the gold standard of therapeutic treatment today.

- L-dopa: the biochemical precursor to dopamine in the central nervous system, it provides some compensation for the limited availability of dopamine seen in PD patients. However, only 1-5% will enter the neurons in the brain because of the action
of the dopa-decarboxylase enzyme complex. Vitamin B6 is required for the conversion of L-dopa to dopamine. Motor fluctuations may occur with levodopa therapy, noted as “off periods,” during which PD patients may have greater anxiety, fatigue, or apathy. Because L-dopa competes with amino acids for transport molecules that facilitate its absorption into the bloodstream from the small intestines, limiting overall protein intake to 0.5-1.0 g/kg/day and by distributing the protein throughout the meals, or limiting overall protein intake during waking hours, may enhance the therapeutic effect of L-dopa. Taken 30-60 minutes prior to meals, it is most effective in managing the external motor symptoms of the disease, but little improvement is seen with cognitive deficits in PD patients, nor is dysphagia markedly improved. Medications that are taken for GERD, depression, and hypertension may interact unfavorably with L-dopa. Drug-nutrient interactions may include nausea, vomiting, decreased appetite, fatigue, taste alteration, xerostomia, and weight loss resulting in low BMIs.

• Decarboxylase inhibitors: include benserazide and carbodopa, aimed at limiting the breakdown of L-dopa outside the blood-brain barrier to enhance the efficacy of L-dopa. Drug-nutrient interactions may include abdominal pain, xerostomia, decreased appetite, constipation, diarrhea, and flatulence.

• Dopamine agonists: include Requip (ropinirole) and Mirapex (pramipexole), which bind to and activate dopamine receptor sites in the brain, thus acting as dopamine-mimetics and maximizing the bioavailability of dopamine. Medications such as Zofran, antipsychotics, and antihypertensives may interact unfavorably with these dopamine agonists. Drug-nutrient interactions may include nausea, vomiting, orthostatic
hypotension, reflux, constipation, polyuria, xerostomia, insomnia, hallucinations, and fatigue.

- Anticholinergic medications: include trihexyphenidyl and benztropine, which enhance dopamine release from dopaminergic neurons and indirectly produce a mild anti-PD effect by decreasing acetylcholine, which acts antagonistically to dopamine. Antihistamines can interfere with these anticholinergic medications. Drug-nutrient interactions may include xerostomia, constipation, urinary retention, blurred vision, nausea, and vomiting.

- COMT inhibitors: include COMTan (entacapone) and Tasmar (tolcapone), which inhibit COMT, one of the two enzymes involved in the breakdown and metabolism of L-dopa, thus resulting in higher and more prolonged concentrations of L-dopa. Confusion and dyskinesia are common side effects. Drug-nutrient interactions may include constipation, abdominal pain, nausea, diarrhea, fatigue, and reflux.

- Combination pharmaceuticals: include Sinemet, which contains both levodopa and carbodopa, the latter of which prevents the premature metabolism of L-dopa by inhibiting dopa decarboxylase before L-dopa reaches its destination in the dopaminergic neurons. It is best to take Sinemet 30-60 minutes before a meal to maximize its absorption and bioavailability and to avoid competition with other amino acids for carrier molecules. “On-off” fluctuations occur when taking this medication because of the rise and fall of the body’s response to dopamine levels. Note that Sinemet has side effects that impact the nutritional status of the PD patient, such as nausea, vomiting, decreased appetite, xerostomia, abdominal pain, and liver toxicity.
• MAO inhibitors: such as Anipryl (selegiline) and Azilect (rasgiline), help to reduce the symptoms of PD by inhibiting the actions of enzymes that breakdown the body’s naturally occurring dopamine stores. Antidepressants, decongestants, and narcotic analgesics can interfere with the efficacy of MAO inhibitors. The main side effect is stomatitis, but nausea, insomnia, dyspepsia, dizziness, edema, headache, and agitation have been reported as well.

• Neuroprotective therapy: includes the medication selegiline along with Vitamin E, aimed at slowing the progression of the disease by reducing oxidative stress.

Surgical modalities for treating PD, focused on improving the quality of life of some PD sufferers, have targeted gene therapy, deep brain electric stimulation, and implantation of embryonic dopaminergic cells into the substantia nigra. More radical brain surgical procedures used to control tremors include pallidotomy and thalotomy, which destroys certain cell clusters in the globus pallidus and thalamus, respectively. These surgical procedures have substantial inherent risks and unfortunately limited success.
DISCUSSION OF MEDICAL NUTRITION THERAPY

In order to comprehend the rationale behind the medical nutrition therapy, it is imperative to understand the physiological changes that often beset the Parkinson’s sufferer. PD patients may present with an elevated risk of malnutrition because of the symptomology manifested during disease progression and the untoward side effects of the pharmaceuticals used to manage the disease. A major goal in nutrition therapy for PD patients is to maximize the absorption of medications and minimize the length of time that inadequate levels of pharmaceuticals are present.1

Risk factors for poor nutrition in community-dwelling older adults are more pronounced in PD patients, and include advancing age, solitary living, polypharmacy, dementia, co-morbidities, anxiety and depression, struggles with activities of daily living, anorexia, and gastrointestinal disturbances which may include dysphagia, gastroparesis, and constipation. Depression and constipation are particularly significant predictors of protein-energy malnutrition in PD patients.6

In general, protein-energy malnutrition, specifically a deficit in these entities, is associated with diminished quality of life, higher morbidity and mortality, delayed wound healing, increased risk of falling and osteoporosis, as well as lengthier hospitalizations, all of which can affect PD patients. Compared to age-matched control subjects, PD patients often have significantly lower BMI and body weight, lower percent ideal body weight, lower triceps skin fold thicknesses, as well as faster progression of age-related sarcopenia as early as two to four years prior to diagnosis. Fifty-two to sixty-five percent of PD patients experience weight loss, according to some estimates.1

The symptoms of PD result in an increased loss of weight and lean body mass as well as a greater risk for decreased nutritional intake. Moreover, because of the deterioration in
coordinated muscle movements and fine motor skills, PD patients often are incapable of independently carrying out routine activities of daily living such as shopping, food preparation, and self-feeding, due to a limited capacity for ambulation. Decreased food intake and weight loss are often exacerbated in PD sufferers due to the cluster of neuropsychiatric symptoms associated with disease progression, such as anxiety, depression, confusion, apathy, and dementia. Nausea, early satiety, gastroparesis, constipation, dysphagia, and alterations in taste and smell all further contribute to a diminished nutrition status in PD patients.\textsuperscript{13}

Unintentional weight loss, which is seen in higher rates among PD patients, is a major nutritional concern in the progression of Parkinson’s disease, and may be a precursor to the end-stage progression of the disease. It manifests itself as a double-edged sword, of sorts: there is an augmentation in energy needs for these patients due to the increase in muscular tremors and the dyskinesia syndrome often seen; however, there is also a marked decrease in energy intake, due to the following factors:

- Decreased sense of smell and taste, which diminishes appetite;
- Cognitive impairment, which impedes the patient’s awareness of nutritional needs;
- Depression – both situational and neurochemical (from a reduction in serotonin levels) - and anxiety, which contribute to a lack of desire to eat and drink;
- Oropharyngeal dysphagia, resulting from poor muscle control, along with diminished masticatory efficiency, which together directly impacts the physical characteristics of the foods to be ingested;
- Medications used to treat Parkinson’s and co-morbidities, whose side-effects and interactions often produced dry mouth, nausea, vomiting, decreased appetite, constipation, and ultimately weight loss;
• Fatigue, which itself reduces the patient’s energy and willingness to eat sufficiently.6

Using the “ADIME” approach in the nutrition care process, a thorough assessment would include:

• Anthropometrics, including weight and BMI;
• Biological data, including current laboratory values which can assess hydration status and liver function;
• Clinical data, including medications and their side effects, the stage of PD, ability to self-feed, presence or absence of dysphagia, gastroparesis, appetite, co-morbidities, level of physical activity;
• Dietary data: food preferences, allergies, aversions, and need for dietary modifications

Nutritional therapy in managing the progression of PD is aimed at addressing six main nutritional diagnoses:

1. Inadequate oral intake
2. Involuntary weight loss
3. Consumption and motility dysfunction (chewing and swallowing difficulties, constipation, bowel impaction)
4. Diminished capacity for independent food preparation and self-feeding
5. Nutrient-medication interactions
6. Inadequate fluid intake and dehydration12

With the emphasis on providing PD patients a high-fiber, plant based diet - including whole grains, fruits and vegetables, calcium-rich foods, and smaller portions of high-protein foods - nutrition interventions designed to improve the nutritional status of PD patients may include any or all of the following aggregately for the above diagnoses:
• Determining calorie and protein needs and food preferences to promote a stable weight goal.

• Increasing nutrient density through fortification with additional calories from fats and/or carbohydrate sources, along with possible supplementation.

• Adjusting the quality, quantity, moisture content, and texture of foods, as well as the timing and duration of meals, and encouraging small, frequent, low-fiber, low-fat meals. This dysphagia nutrition therapy is accomplished by working closely with speech therapists and following their recommendations based on swallow studies and other diagnostic tests.

• Creating a supportive environment around eating, including proper lighting, noise monitoring, odor control, and proper seating support.

• Encouraging PO intake.

• Determine the need for the administration of nutrition support.

• Dental examination of the oral cavity with particular attention to caries, periodontal disease, occlusal trauma, and prosthetic and restorative considerations, and addressing xerostomia from medication-induced reduction in salivary flow.

• Providing the patient with adaptive equipment such as special flatware, cups, and plates, working with occupational therapists, to promote independence and self-feeding.

• Providing safe access to food sources and cooking facilities if needed.

• Scheduling frequent oral intakes of fluid in small amounts but incorporating IVF if/when clinically appropriate. Four to eight glasses of water, along with other
beverages and water-containing foods, are recommended for PD patients. Consideration may be given to providing higher calorie beverages as well.

- Adjustment of the timing of medications around meals to maximize absorption and effectiveness of nutrients and drugs and limitation of drug-nutrient interactions. Redistributing protein intake to evening meals and snacks may enhance the efficacy of daytime L-dopa administration.

- Addressing constipation and fecal impaction, which is a multifactorial problem emanating from decreased GI motility, dysfunction of the anal sphincter, inadequate fluid intake, medications and their side effects and interactions, and a reduction in the number of dopaminergic neurons located in the colon. Increased fiber, stool softeners, laxatives, and/or prokinetics are often required. Patient education is very important, especially in this matter. Twenty-five to thirty-five grams of daily fiber is recommended for PD patients.\(^\text{13}\)

In addition to the above-mentioned interventions, possible supplementation with the following micronutrients and phytochemicals may have some therapeutic value:

- Calcium, magnesium, and Vitamins D and K, to address osteoporosis.
- B6, B12, and folate for support of metabolic pathways.
- Vitamins C and E for their antioxidative properties.
- L-tyrosine, the biochemical precursor of L-dopa and dopamine, to possibly augment the availability of dopamine.
- Coenzyme Q10, which has a stimulatory effect on the cellular mitochondrial complex.
- Iron sulfate, which is a cofactor in L-dopa biosynthesis.
• Caffeine, whose increased intake at levels of 4-5 cups of coffee per day may
decrease the risk of acquiring Parkinson’s disease;
• Black Tea, which is made by oxidizing green tea, according to one study may also
decrease the risk of Parkinson’s, perhaps through the production of antioxidants;
• Essential fatty acids, whose contribution to cellular membranes may have a
protective benefit;
• Vitamin D, since Parkinson’s patients often appear to be deficient in this important
micronutrient.\(^8\)

The following parameters would be included in the monitoring and evaluation of the nutrition
interventions cited above:

• Weight
• PO intake
• Appetite and satiety
• Food preferences and textures
• Feeding assistance and adaptive equipment
• Laboratory values, especially for visceral protein
• Medications
• Skin integrity
• Gastrointestinal issues, including:
  • Nausea
  • Vomiting
  • Diarrhea
  • Constipation and fecal impaction
- Abdominal pain
- Dysphagia
- Gastroparesis
- GERD
- Need for nutrition support
- Adequate oral hydration
- Dental status and oral health
- Activities of daily living

Some cohort studies have shown that the overall quality of the diet or specific food groups is not a good predictor of occurrence for PD, concluding that there does not appear to be a strong correlation between the consumption of fruits and vegetables, nor intakes of antioxidant vitamins, and the risk of acquiring PD. It was postulated that perhaps farming pesticides and herbicides may offset the benefits of consumption of fruits and vegetables. One study that included more than 2,000 women test subjects did note a correlation between high milk consumption and an increased risk of PD, and although this finding is corroborated in other studies, no definitive explanation was presented as to the mechanism by which this most likely occurs.

Other studies have countered the above-cited cohort studies, proposing that nutrients with anti-oxidant properties may indeed be able to prevent or delay the progression of PD by protecting mitochondrial function.

- Epidemiological studies have suggested that consuming foods that are rich in the antioxidant vitamins C and E - which are cytosolic free-radical scavengers and cell membrane peroxidation inhibitors, respectively - are associated with a diminished risk
level of acquiring PD. In the case of Vitamin C, enhanced absorption and efficacy of L-dopa may occur.

- Vitamin D appears to have a neuroprotective effect, owing partly to its ability to both regulate dopamine levels and increase the levels of the powerful antioxidant glutathione; not coincidentally, the substantia nigra is a region of the brain which is rich in Vitamin D receptors as well as one-alpha-hydroxylase, the enzyme responsible for the biological activation of Vitamin D.

- Coenzyme Q, a fat-soluble vitamin-like substance with potent antioxidant properties, appears to have some protective effect on the dopaminergic neurons in the substantia nigra, thus minimizing the loss of these important neurons.

- Creatine, because of its ability to increase intracellular levels of phosphate-containing compounds needed to counter depletion of ATP stores, is being investigated for possible use in the prevention and treatment of PD.

- Omega-3 polyunsaturated fatty acids may protect against dopamine loss by stabilizing the lipid membranes of dopaminergic neurons, and deficient levels of mono- and polyunsaturated fatty acids are strongly associated with impaired brain function.

- In one study limited in scope, naturally occurring L-dopa from seed sources seems to have greater bioavailability than synthetic L-dopa, with a more rapid onset and longer duration of action of the therapeutic response in some of the PD patients studied.

- Polyphenolic compounds, such as those found in a variety of plants and green teas, have antioxidant properties and may play a role in inhibiting the aggregation of misfolded protein seen in the Lewy body formations in the dementia of PD patients.
- Resveretrol, a well-known antioxidant contained in a number of fruits and vegetables, and its chemical cousin oxyresveretrol, may help to protect dopaminergic neurons from the harmful effects of environmental toxins through the suppression of proinflammatory genes.
- Phytoestrogens found in soy, grains, and nuts, such as genistein, seem to also have a neuroprotective effect through the upregulation of antioxidative genes, and may partly explain the lower incidence of PD in women versus men.
- Supplementation with B vitamins folate, B6, and B12 can help mitigate against increased homocysteine levels often seen in PD patients receiving L-dopa treatment, since homocysteine has been linked to an increased risk of CVD, dementia, and cognitive impairment.

The above-mentioned nutraceuticals and dietary supplements may be a potentially important adjunct to current therapeutic modalities in treating PD patients, through prevention, diminution of pharmaceutical side effects, retardation of disease progression, and/or enhancement of the bioavailability of L-dopa. Vitamin C may improve the efficacy of L-dopa by enhancing its absorption.

Since there appears to be many uncertainties with regard to the pharmacodynamics of nutraceuticals and supplements, further research is needed to determine important factors such as dosing, therapeutic goals, efficacy, and practicality.

Another developing area of medical research involves the use of ketogenic diets, which are already used in controlling some seizure disorders, particularly in children. It is thought that the reduction of dietary protein would reduce the competition between dopamine and other amino
acids for binding sites and absorption, while the higher fat content may impart some neuroprotective function through cell membrane stabilization.

Although the evidence-based literature does not contain randomized controlled studies regarding a definitive link between PD and gluten intolerance, it is worth noting that there are some intriguing parallels between the neurological manifestations of each condition. Some individuals with gluten sensitivity have been known to exhibit ataxia, tremors, lack of coordination, gait abnormalities, diminished peripheral sensory ability, and muscle rigidity, all of which have been well documented in the progression of PD, and perhaps this may be an area of research worth exploring in greater depth.
PRESENTATION OF PATIENT

JM is an 82-year-old Caucasian male, born on October 2, 1931, who is a resident in the long-term care pavilion at the St. Francis County Home in Upper Darby, Pennsylvania, a neuro-rehabilitation facility. A lifetime resident of Philadelphia, JM retired as a truck driver seventeen years ago, and he and his wife Rose – with whom he shares room 243-B in Pavilion #2 at St. Francis – have 2 children, 7 grandchildren, and 2 great-grandchildren, most of whom still live in or in close proximity to Philadelphia. In my interviews with JM between February 4th and February 25th, 2014, I learned, among other things, that he is a passionate Phillies’ fan, greatly enjoys big band music, and likes watching game shows on TV with Rose. He prefers to interact with his wife rather than attend structured activities. Parkinson’s disease and subsequent dementia have affected JM’s ability to effectively communicate verbally, as he tended to mumble, but I was able to comprehend him adequately to extract information helpful to creating this case study.

JM was first admitted to St. Francis on April 3, 2007, with a primary diagnosis of Parkinson’s disease. Prior to admission, he was diagnosed with PD approximately ten years earlier, based on MRI studies and other diagnostic tests, as well as symptomology which included tremor in his hands, difficulty buttoning his shirts, sporadic walking difficulty, and occasional numbness in his toes, but at that point the disease was manageable at home. His past medical history includes dementia with Lewy bodies, anemia, osteoporosis, hyperlipidemia, cardiac pacemaker, syncope, hypothyroidism, GERD, abdominal aneurysm without evidence of rupture, gait abnormality, paralysis agitans, altered mental status, atrial fibrillation, hypertension, gout, and oropharyngeal dysphagia. Due to his mental status, JM is not permitted to leave the facility except for appointments with extramural healthcare professionals.
His current list of medications is as follows:

- Aricept 10 mg qd (dementia)
- Aspirin 81 mg qd (syncope)
- Calcium 600 mg + Vitamin D b.i.d (osteoporosis)
- Colace 150 mg b.i.d. (constipation)
- Dilantin 100 mg b.i.d. (convulsions)
- Folic Acid 1 mg qd (anemia)
- Gabapentin 250 mg t.i.d. (seizures)
- Levothyroid 150 mcg (hypothyroidism)
- Magnesium Oxide 400 mg qd (laxative)
- Multivitamin with iron qd (anemia)
- Nasonex 50 mcg spray prn b.i.d. (rhinitis)
- Nexium 40 mg qd (GERD)
- Phos-NaK 280-160-250 mg t.i.d. (mineral supplement)
- Senna-Gen 8.6 mg qd (constipation)
- Seroquel 25 mg qhs (dementia)
- Simvastatin 10 mg qd (hypercholesterolemia)
- Sinemet 25/250 (carbodopa 25 mg/levodopa 250 mg) q.i.d. (Parkinson’s Disease)
- Vitamin B12 500 mg qd (vitamin supplement)
- Acetaminophen 160 mg q.i.d. prn (pain)
- Atrovent Nasal 0.03% t.i.d. (rhinitis)
- Dulcolax Suppository 10 mg prn (constipation)
- Duoneb Inhaler q6h prn (rhinitis)
• Milk of Magnesia Oral Suspension 400 mg qd prn (constipation)
• Robitussin DM syrup q6h prn (rhinitis)
Due to the length of stay for JM at St. Francis, I have limited my discussion of the medical/surgical course of treatment to the most pertinent and generally more recent, especially as it pertains to nutrition.

- April 3, 2007: admission to St. Francis Country House with primary diagnosis of Parkinson’s disease, but concurrent diagnoses included hypertension, a non-ruptured abdominal aneurism, gout, and atrial fibrillation.
- October 1, 2007: additional medical conditions were diagnosed, which included convulsions, anemia, hypothyroidism, GERD, spinal stenosis, psychosis, syncope, and paralysis agitans.
- January 4, 2011: hyperlipidemia was noted.
- March 25, 2011: osteoporosis was noted.
- June 14, 2011: Nursing assessment on this date noted the following conditions: (1) chronic back pain related to osteoporosis, to be managed with pain medication, comfort measures, and diversional activities; (2) the potential for constipation due to decreased mobility and diminished fluid intake; (3) alteration in skin integrity related to decreased mobility, and two small skin tears were noted; (4) aspirin therapy may cause an increase in bruising and bleeding; (5) risk for decreased cardiac output related to hypertension and cardiac pacemaker.
- November 28, 2012: full nutrition assessment performed, at which time it was noted that he was following a regular CCD diet and hydration program; his meal completion was generally 76-100% with a reported good appetite; and his weight has been stable for the past 30, 90, and 180 days.
• December 26, 2012: restorative nursing care noted potential for muscle contracture and the need for range of motion therapy; impaired mobility resulting from decreased strength and the need for restorative ambulation 6 times per week, related to the progression of PD.

• March 7, 2013: Dietetic assessment noted inadequate oral intake related to therapeutic and texture-modified diet as evidenced by weight loss of 9.2 pounds (6.2%) X 30 days.

• April 18, 2013: CT brain scan: to evaluate seizures and altered mental status.

• April 25, 2013: upper endoscopy and PEG placement due to malnutrition, dehydration, and decreased PO intake.

• May 6, 2013: speech therapy assessment noted continued risk for aspiration related to poor oral control and poorly coordinated mastication, leading to recommendations of mechanically soft, regular diet with chopped meats and thin liquids, due to PD progression.

• November 1, 2013: Quarterly nutritional assessment was performed. His diet was mechanical soft with a fortified food program (FFP) and hydration program (additional 8 oz. fluid offered per meal). He was also receiving one can of Boost once per day, but a stable weight for the past 180 days prompted a recommendation to discontinue the Boost. Water flushed of 250 mL once per day continued through the PEG. He had good PO intakes and his BMI was calculated to be 25.

• November 12, 2013: speech therapy evaluation – confirms oropharyngeal dysphagia related to Parkinson’s disease, paralysis agitans, dementia with Lewy bodies, lack of coordination, and GERD.

• January 14, 2014: TSH 0.23 (slightly low; normal range=0.27-4.62).
• January 17, 2014: At his monthly weigh-in, JM was 140 pounds, a loss of 9.2 pounds or a 6.2% change in body weight; his UBW is 148.

• January 22, 2014: most recently updated monthly progress note, during which time there were no observed changes in his mental, functional, or health status and his appetite was reported to be general 50-75% at each meal. An 8-pound weight loss from the previous month as attributed to low levels of TSH and the appropriate downward adjustment to his levothyroxine was made. All lab values on this date were within normal limits.

• February 6, 2014: Dilantin 13.4, WNL (normal range=10-20).

• February 7, 2014: dental exam to evaluate oral mucosa and fit of full dentures, the outcome of which pronounced the resident in good dental health with properly fitting dentures.

• February 10, 2014: most recent psychiatric evaluation, noting Parkinson’s disease with psychosis, dementia with Lewy bodies, and an overall regressed state.

Other pertinent information gleaned from JM’s medical records include the following:

• JM receives yearly influenza vaccinations.

• He is presently non-ambulatory and wheel-chair bound.

• He has a peg tube, through which he receives certain medications and supplements only (Aricept, calcium, gabapentin, Nexium, Phos-NaK, and acetaminophen).

• He wears well-fitting full upper and lower dentures which were present in his mouth during all visits, and he sees a dentist yearly for examinations.

• His standing lab/test orders are as follows: every 6 months-B-12, BMP, Dilantin, LFTs, fasting lipid panel, pacemaker, TSH; every month-CBC.

• Ongoing physical, occupational, speech, and restorative nursing care therapies.
DISCUSSION OF NUTRITION CARE

Long-term care residents at St. Francis, such as JM, have nutrition assessments scheduled at the following four times: quarterly; annually; if/when significant changes occur; and if/when residents require hospitalization.

The most current quarterly full nutrition assessment was scheduled to be completed in February 2014, fortuitously coinciding with my clinical rotation at St. Francis Country House.

The results of the assessment are as follows:

A. Demographic/anthropometric data
   1. Current weight: 140.3 lbs., 63.6 kg
   2. Height: 64 inches, 162.56 cms
   3. UBW/goal weight: 135-145 lbs, 61.4-65.9 kg
   4. BMI: 24
   5. Resident’s gender: male
   6. Resident’s age and date of birth: 82 years old, 10/2/1931
   7. Calorie needs (method): 25-30 kcals/kg/day, 1590-1908 kcals/day
   8. Protein needs: 1 g/kg, 64 g protein/day
   9. Fluid needs: 1 mL/kcal, 1590-1908 mL/day
   10. Diet order: mechanical soft, hydration program (an additional eight ounces of fluid at each meal), fortified food therapy (additional calories from fats and carbohydrate sources which are infused into soups, mashed potatoes, puddings, and cereals)
   11. Supplements: house supplement, Resource, 4 oz, q.i.d., which provides 2 kcal/mL and 0.8 g protein/mL
   12. Adaptive equipment required: none
13. Enteral/parenteral order: none

14. Flush order (via PEG): 250 mL t.i.d. @ 12 am, 6 am, 12 pm, and 50 mL 9 am, 1 pm, 5 pm

15. Food allergies: NKFA

16. Chewing or swallowing disorders: none

17. Ethnic/cultural/religious food preferences: none

18. Weight status; gain or loss: BMI=24, in range of 21-27, no weight change

19. Weight history: February 12, 2014: 140.3
    February 6, 2014: 140.1
    January 29, 2014: 140.0
    January 22, 2014: 140.0
    January 15, 2014: 140.3
    January 8, 2014: 139.0
    December 6, 2013: 148.2
    November 5, 2013: 149.3
    October 1, 2013: 145.9

B. Oral Intake: Food: intake meets 76-100% of estimated needs

C. Oral Intake: Fluids: pt accepts fluids well, consumed almost all liquids in past 3 days.

    Water flushes 250 mL q.i.d. and 50 mL before and after meds

D. Relevant conditions and diagnoses: PMH: Parkinson’s, anemia, infection, CVA, fracture, UTI, COPD, edema, surgery, osteoporosis, GI bleed, poor circulation, constipation, diarrhea, GERD, anorexia, acute gastritis, altered mental status, afib, Lewy body dementia, syncope, HTN

E. Medications; nutrition-related; drug-nutrient interactions: 5 or more drugs/day. Current meds: Aricept, calcium 600+, Dilantin 100 mg b.i.d., levothroid 150 mg qd, magox 400 mg qd, MVI qd, Vit B-12 500 mcg qd, simvastatin, Senna-gen 8.6 mg qd, Seroquel 25 mg qd, Phos-NaK oral packet 280-160-250 tid, Sinemet qid, Nexium 40 mg qd
F. Physical and mental functioning: out of bed with assistance; motor agitations (tremors, wandering); limited feeding assistance; supervision while eating; chewing or swallowing problems; teeth in poor repair; ill-fitting dentures or refusal to wear dentures; edentulous; taste and sensory changes; unable to communicate needs.

G. Lab values: albumin and other nutrition-related lab values WNL

H. Skin condition: intact

I. Risk Level: medium

J. Assessment: Annual review-No significant weight change X 30 X 90 X 180 days. BMI 24 WNL. PO intakes variable but generally 76-100% of meals consistently consumed. Appetite good. House supplement provided, resident consumes 100%. Diet is mechanical soft with hydration and fortified food programs tolerated without difficulty. Albumin 1/2/14 3.8. Current thyroid panel WNL and weight is stable. Dehydration risk assessment low. No therapeutic diet necessary at this time; mechanically altered diet is appropriate. Resident still capable of feeding himself but requires more assistance and needs help with meal set-up. Skin intact, no wounds noted. No new recs at this time. Resident enjoys meals in his room with wife Rose.
SUMMARY

The role of the dietitian, as a member of a multidisciplinary health care-providing team, is central to the well-being and sustenance of individuals afflicted with Parkinson’s disease. Proper assessment, diagnosis, interventions, and monitoring and evaluating nutritional therapeutic approaches will help to manage the symptoms of the disease and allow the sufferer to live the highest quality of life that is possible.

With regard to an update on JM, based on the most recent assessment he appears to be faring quite well. His weight has remained stable, and his appetite and PO intakes remain excellent, suggesting in part that the current diet order is appropriate and meeting at least most of his estimated energy needs. He has not developed any GI issues and his skin integrity has remained healthy. Although there has been a general, steady decline in both his motor and non-motor symptoms, he is still functioning at a high level and enjoys some independence.
MEDICATION BIBLIOGRAPHY

Aricept (Donepezil) 10 mg qd (dementia); can cause N/V/D.
Aspirin 81 mg qd (syncope); can cause GI bleeding and N/V.
Calcium 600 mg + Vitamin D b.i.d (osteoporosis); need to insure adequate hydration.
Colace 150 mg b.i.d. (constipation); can cause N/D.
Dilantin (Phenytoin) 100 mg b.i.d. (convulsions); can cause N/V/C and dysphagia.
Folic Acid 1 mg qd (anemia); metabolism inhibited by Vitamin B-12, Vitamin C, and iron.
Gabapentin 250 mg t.i.d. (seizures); can cause N/V/D/C, dry mouth, and dyspepsia.
Levothyroid (Levothyroxine) 150 mcg (hypothyroidism); can cause weight loss.
Magnesium Oxide 400 mg qd (laxative); can cause N/V/D and cramps.
Multivitamin with iron qd (anemia)
Nasonex 50 mcg spray prn b.i.d. (rhinitis); can cause N/V and diminished taste sensation.
Nexium 40 mg qd (GERD); can cause N/D and abdominal pain.
Phos-NaK 280-160-250 mg t.i.d. (mineral supplement); can cause increased thirst, weight gain.
Senna-Gen 8.6 mg qd (constipation); can cause N/V/D and cramps.
Seroquel 25 mg qhs (for dementia); can cause abdominal pain, constipation; avoid grapefruit.
Simvastatin 10 mg qd (hypercholesterolemia); can cause N/D/C and abdominal pain; avoid alcohol and grapefruit.
Sinemet 25/250 (carbodopa 25 mg/levodopa 250 mg) q.i.d. (Parkinson’s Disease); can cause N/V/D/C, dry mouth, taste loss, dysphagia, and competition with aromatic amino acids for absorption.
Vitamin B12 500 mg qd (vitamin supplement); can cause mild diarrhea.
Acetaminophen 160 mg q.i.d. prn (pain); avoid alcohol; caffeine can increase absorption.
Atrovent Nasal 0.03% t.i.d. (rhinitis); can cause N, dyspepsia, dry mouth and throat.
Dulcolax Suppository 10 mg prn (constipation); can cause N/D and cramps.
Duoneb Inhaler q6h prn (rhinitis); can cause altered taste.
Milk of Magnesia Oral Suspension 400 mg qd prn (constipation); can cause N/D and cramps.
Robitussin DM syrup q6h prn (rhinitis); can cause N/V/D.
REFERENCES


3. Life with Parkinson’s: Diagnosis and Symptoms; The Michael J. Fox Foundation; [www.michaeljfox.org](http://www.michaeljfox.org); date accessed 3/9/14.


14. Patient’s medical records from St. Francis Country House, Upper Darby, PA; accessed February 4-25, 2014