Parkinson’s disease is classified as a progressive neurodegenerative disease. As time progresses symptoms get worse. From day-to-day the Parkinson’s disease patients may not realize things are getting worse. But, when they stop and reflect on how things were last year or five years ago it reveals a course of degeneration. A course that will accelerate in the future.

The typical neurologist sees the average Parkinson’s disease patient in clinic once every six months. During these visits prescriptions are refilled plus progression and degeneration of the patient’s symptoms are documented in the medical record. Nothing is done to address reversible aspects of nutritional collapse which are causing symptoms.

The clinical course may vary greatly from one patient to the next. While there are patients who function on a high level for ten or fifteen years after being diagnosed, this is rare. Many patients are severely debilitated to the point of being in a wheelchair or nursing home within five to seven years of being diagnosed.

Recognizing progression and degeneration of Parkinson’s disease. While symptoms can be better or worse from one day to the next, the best evidence of progression and degeneration of Parkinson’s disease is when the symptoms get worse to the point that the daily dosing value of the drugs need to be increased or new drugs need to be started. The stable patient is on the same daily dose of pills for years without symptoms getting worse.

What is not being managed. Over time, deterioration of the Parkinson’s disease symptoms is much more than progression of the brain damage causing disease symptoms. The symptoms of nutritional deficiency associated with Parkinson’s disease and drugs can be identical to Parkinson’s disease symptoms. The standard care provided by medicine wrongly views symptoms caused by nutritional deficiency as being caused by the irreversible permanent brain damage, when in fact these symptoms are reversible.

Our doctors discovered then published in the peer-reviewed medical journals how Parkinson’s disease and drugs can induce 29 causes of nutritional deficiency (see the following pages). Some nutritional deficiency symptoms are identical to Parkinson’s disease symptoms. Other nutritional deficiencies cause side effects thought to be untreatable in the past. In many cases the progression of symptoms is not due to permanent brain damage, it is from reversible nutritional deficiencies.

PARKINSON’S DISEASE CARE IN A LEAGUE OF ITS OWN

Stabilization involves gaining optimal control of Parkinson’s disease symptoms while preventing progression of symptoms caused by nutritional deficiencies.

Correcting the nutritional collapses associated with Parkinson’s disease and its care takes time (see pages 2 and 3). The typical new patient requires 20 to 30 weeks of weekly visits.

There are two ways to receive care from our doctors:

1. Face-to-face in clinic
2. By telemedicine

Telemedicine is the use of a secure internet computer link with voice and cameras to provide medical care. If you are not within close driving range to one of our doctors, telemedicine is a viable option. The American Medical Association has published papers documenting that Parkinson’s disease medical care provided with telemedicine is just as effective as face-to-face in clinic care. All you need is a computer, then our technicians help you set up.

There is only one approach that is most effective for managing Parkinson’s disease symptoms, the naturally occurring amino acid nutrient L-dopa, which is optimal for addressing the relative nutritional deficiency of dopamine precursors found in all Parkinson’s disease patients.

There is only one approach that optimally manages the 29 causes of relative nutritional deficiency symptoms associated with Parkinson’s disease or caused by drugs used to treat Parkinson’s disease, this approach.

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*Parkinson Clinics is a department of the Morgan Park Medical Clinic
1150 88th Ave W Duluth, MN 55808. Licensed medical doctors from many states have come together to provide these services.
The relative nutritional deficiencies of Parkinson’s disease

A relative nutritional deficiency occurs when an optimal diet does not meet the nutritional needs of the body.

Dopamine concentrations determine the severity of Parkinson’s disease symptoms. The body makes dopamine from the naturally occurring nutrient L-dopa. In the normal patient nutrient intake allows the body to make enough dopamine. For the Parkinson’s disease patient, an optimal diet will not allow the body to make enough dopamine, a relative nutritional deficiency of the nutrients that dopamine is made from exists.

The most effective nutrient for managing the primary relative nutritional deficiency associated with Parkinson’s disease is the naturally occurring amino acid nutrient L-dopa which the body needs to make dopamine.

Management with Parkinson drugs other than the nutrient L-dopa is not associated with optimal relief of symptoms. The patient must be supplemented with the naturally occurring amino acid nutrient L-dopa for the body to make levels of dopamine which facilitates optimal relief of symptoms.

Twenty-nine causes of nutritional deficiency are associated with Parkinson’s disease, L-dopa, and carbidopa or benserazide (which has the same effects as carbidopa). None of these nutritional deficiencies are addressed by doctors not using this approach. The standard medical approach induces nutritional collapse.

These nutritional deficiencies can cause Parkinson’s disease symptoms to progress, deteriorate, and accelerate the permanent brain damage causing Parkinson’s disease, or display as side effects that appear to be irreversible.

Administering carbidopa or benserazide is associated with making Parkinson’s disease symptoms worse secondary drug-induced nutritional deficiency. Read more on the next page. Symptoms of some nutritional deficiencies getting worse are identical to Parkinson’s disease symptoms getting worse.

The 29 primary causes of relative nutritional deficiency induced by Parkinson’s disease, L-dopa, and carbidopa (benserazide)

Each line between the left boxes and the list on the right represents a potential cause of relative nutritional deficiency which can deplete items listed on the right.

Source: Parkinson’s disease managing reversible neurodegeneration; Neuropsychiatric Disease and Treatment 2016:12 763–775

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Self-treatment with this approach should not be attempted. Experienced medical doctors with Parkinson’s disease, who have cared for many patients, are unable to achieve optimal results with self-treatment. Many decisions required for optimal results tend to be counter-intuitive (the opposite of common sense).
Parkinson’s disease pitfalls

The naturally occurring amino acid nutrient L-dopa is widely recognized as the most effective Parkinson’s care option.

There are two classes used for Parkinson’s disease symptom management:
1. L-dopa, a naturally occurring amino acid nutrient
2. Drugs which are not as effective as L-dopa.

All prescription forms of L-dopa contain the drug, carbidopa or benserazide.
1. 89% of Parkinson’s disease patients take these combinations.
2. Carbidopa or benserazide do nothing to improve Parkinson symptoms.
3. Their only benefit is management of L-dopa-induced nausea caused by improper management of L-dopa inducing a nutritional deficiency.

We have quit prescribing carbidopa and benserazide. The doctors that developed this form of optimized medical care offer the most effective Parkinson’s disease care available using L-dopa. Since we have found a better way, we do not use carbidopa or benserazide them.

Carbidopa or Benserazide deplete vitamin B6
1. Carbidopa or benserazide do the same thing in the human body. This causes vitamin B6 depletion.
2. Carbidopa and benserazide bind irreversibly to vitamin B6, this causes a nutritional deficiency.
   a. Symptoms of severe vitamin B6 depletion are identical and confused with progression and worsening of Parkinson’s symptoms.
   b. The increasing Parkinson’s death rate has been linked to carbidopa and benserazide. The Center of Disease Control notes that Parkinson’s disease deaths have quadrupled since 1976, the first full year that carbidopa become available (see left).
   c. Depletion of vitamin B6 depletes glutathione which protects against the fat-soluble neurotoxins that can cause Parkinson’s disease brain damage.
   d. Vitamin B6 depletion by carbidopa or benserazide is the cause of treatment associated dyskinesias and choreiform movement disorders. In the past these problems were thought to be permanent with no treatment available. Our doctors effectively manage these nutritional deficiency related problems that were wrongly attributed to L-dopa in the past.
   e. Vitamin B6 depletion negatively affects over 300 enzymes and proteins.
   f. Vitamin B6 depletion cannot be addressed while the patient is taking carbidopa or benserazide.
3. Twenty-nine causes of nutritional deficiency can be induced by Parkinson’s disease, L-dopa when used alone and carbidopa or benserazide. All need to be corrected for patients to achieve optimal results.

Giving vitamin B6 with carbidopa or benserazide is not effective, these drugs need to be stopped.

Clinical Pharmacology: Advances and Applications
The Parkinson’s disease death rate: carbidopa and vitamin B6

OUR 2014 PEER-REVIEWED PAPER NOTES: “Systemic vitamin B6 concentrations inversely correlate with mortality induced by coronary artery disease, colorectal cancer, stroke, heart failure, and atherosclerosis. We hypothesize that if carbidopa significantly depletes B6, then an increased death rate will be observed. During the first 15 years of prescribing L-dopa (1960–1975) it was administered without carbidopa, a practice that was associated with a decreasing death rate. ... Between 1976 and present, there has been an increase in the general Parkinson’s disease death rate.”

Data Sources: Center for Disease Control

In 2003, the CDC placed Parkinson’s disease on its top of 15 causes of death list at number 14, pneumonia dropped from the list.

Since 1976, the Parkinson’s disease death rate has increased by 356%. Recent peer-reviewed medical papers have linked the increasing death rate to carbidopa.

Prior to 1976, when L-dopa was used without carbidopa the death rate was trending downward.

1976, the first full year of carbidopa sales

Bibliography
Full text of each can be obtained free of charge with a Google search
3. Parkinson’s disease-associated melanin steal Neuropsychiatric Disease and Treatment Dec 2014
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