

LE Magazine June 2004

REPORT

Natural Prescriptions For Parkinson's Disease

By Bruce Scali

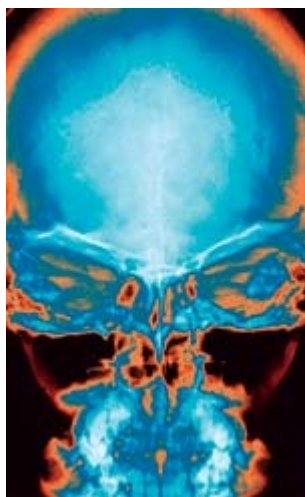
After 200 years of investigation, researchers and clinicians have identified neither the cause nor the cure for Parkinson's disease, the condition first described by James Parkinson in 1817. Yet with the baby-boom generation on the cusp of retirement, the need for definitive answers has never been more urgent.

Neurodegenerative symptoms such as tremor and a shuffling gait, also known as paralysis agitans, have been observed and recorded by physicians since ancient times. Much has been learned since about the characteristics of Parkinson's disease, but much remains unknown. Not only do the cause and cure of Parkinson's continue to elude us, but doctors still cannot identify those in early stages of the disease. Consequently, a definitive program for prevention remains uncertain.

On the positive side, much is known about Parkinson's disease, the factors that contribute to its development, and medical treatments and natural alternatives that relieve its symptoms and may even slow its progression. And avenues of further inquiry have shown great promise.

Parkinson's disease is correlated with age, and up to 40% of those with the disease experience serious mental decline.¹ Diagnostic tools such as the P300 brainwave speed and voltage test can identify early cognitive loss and may help detect early-stage Parkinson's disease as well.²

According to the National Parkinson Foundation, there are approximately 1.2 million people with Parkinson's disease in the US and Canada. Parkinson's disease afflicts 1% of adults aged 60 or older and 2% of adults aged 70 or older.³ As the population ages and the average life span grows, researchers estimate that 1.5 million Americans will be afflicted with Parkinson's in the near future.



The Pathology of Parkinson's

Just as plaque is evident in the brains of Alzheimer's patients, distinct physical representations can be found in the brains of Parkinson's sufferers. A loss of dopaminergic neurons is seen, primarily in the substantia nigra pars compacta and locus coeruleus regions in the midbrain. These cells produce the neurotransmitter or biochemical that helps regulate the nervous system and body. Particularly problematic about Parkinson's disease is that symptoms do not present until 80% of the brain's dopamine is lost. Also characteristic is the presence of cytoplasmic inclusion bodies (Lewy bodies, clear cells) in the brain, which take the place of the dead neurons.⁴

Epidemiological factors play a part in Parkinson's disease. Males are more likely to develop the disease than females and its incidence in Caucasians seems higher than in other races.^{5,6} The prevalence of Parkinson's disease differs geographically, but when allowances are made for age distribution, diagnostic criteria, and varying access to health care, the results are not conclusive.^{7,8} It is worthwhile noting, however, that three genes have been identified that give rise to the Parkinsonian phenotype, and Parkinson's disease occurs more frequently in identical twins.⁹

Genetic studies involving families, twins, and single genes also have not been definitive.¹⁰⁻¹² Data too often have depended on the subjects' memories, and Parkinson's disease could be explained by diet and environmental factors that also are shared by the subjects. Regardless, if Parkinson's disease were strictly a genetic trait, we would see far more occurrences of it in families than at present.

Toxins such as carbon monoxide, herbicides, methanol, and insecticides have been linked to Parkinson's disease, so both industrial and farm populations are affected. Exposure to infections such as encephalitis, measles, influenza, and sexually transmitted diseases also has been associated with Parkinson's.¹³

Determining cause becomes a question of whether something specific is responsible or whether one's genetic composition

predisposes susceptibility to environmental factors linked to Parkinson's disease. In the absence of a specific cause, the consensus is that Parkinson's disease results from both genetic and environmental factors.



Symptoms and Diagnosis

Complicating matters further is that dozens of common pharmaceuticals (including pain medications and selective serotonin reuptake inhibitors, or SSRIs), medical procedures such as dye studies and bone marrow transplants, and conditions such as head trauma, liver disease, tumors and lesions, vascular disease, and other central nervous system disorders can all cause Parkinson's disease symptoms.¹³ Parkinson's disease status progresses from clinically possible, to probable, to definite, based on the number and combination of motor symptoms present. Tremor, rigidity, or bradykinesia (hesitation or slowness) makes Parkinson's disease possible. Any two of resting tremor, rigidity, bradykinesia, and impaired postural reflexes (such as extended arm tremor or difficulty with handwriting) indicates probable Parkinson's disease. Any combination of three of the four previous symptoms results in a definitive diagnosis of Parkinson's disease.¹⁴

In the absence of clinical certainty, a physician can use methods of exclusion to eliminate Parkinson's disease as a diagnosis. Blood tests identify toxic poisoning and hypoparathyroidism that could explain symptoms. Magnetic resonance imaging (MRI), computed tomography (CT), positron emission tomography (PET), and single photon emission computed tomography (SPECT) scans uncover structural, vascular, and metabolic abnormalities that cause Parkinson's disease. Patient history reveals previous infections and drug therapies. Because motor deficits appear and advance gradually in Parkinson's, rapid onset or progression of symptoms would argue against a diagnosis of the disease. Exclusion also is attributed to non-responsiveness to dopamine therapies.¹⁵ Eliminating the possibility of Parkinson's disease helps physicians to focus on effective treatments for the actual condition presented by patients.

Parkinson's disease symptoms

Any three of these four symptoms confirm Parkinson's disease:

1. Resting tremor
2. Rigidity
3. Bradykinesia (slow movement)
4. Impaired postural reflexes

Accurate diagnosis is a significant issue with Parkinson's disease. A post-mortem study showed that 25% of those diagnosed with Parkinson's disease had another cause of the symptoms presented.¹⁶ Inaccurate diagnoses can lead to wasteful, ineffective, or even harmful treatments for patients, as well as provide faulty data that make finding a cure for Parkinson's even more difficult.



Pathogenesis

Brain cell death is undisputed in Parkinson's disease, and advances in molecular science have opened up new avenues of investigation for its cause and treatment. While previous research focused on oxidative stress, other factors are now receiving attention. Neurodegeneration may involve mitochondrial abnormalities, excitotoxicity, trophic factors, and inflammatory cytokines.¹⁷ Determining which of these, if any, is the primary cause of the death of brain cells and how these are interrelated have yet to be discerned. Nonetheless, this new knowledge about cell biology has had a positive impact on treatment.

Oxidative stress results when an excess of free radicals overwhelms the body's defenses. In their search for an electron to pair with a free radical, molecules cause damage to the donor molecules. The resulting chain reactions damage lipids, protein, DNA, and, ultimately, cells.¹⁸ Free radicals also can increase intracellular calcium, causing apoptosis (slow cell death, as opposed to necrosis, a more rapid cell death). Metals such as iron, copper, and aluminum catalyze free radical reactions, and studies have shown an excess of these metals in the brains of Parkinson's disease patients.¹⁹

The exploration of mitochondrial abnormalities in Parkinson's disease patients resulted from the discovery of the effect of MPTP (a toxin found in heroin and pesticides) on mitochondria exclusively in the SNc region of the brain.²⁰ This finding was not duplicated in patients with other neurodegenerative conditions. It is not known how mitochondrial damage contributes to neuron degeneration, but research is concentrating on a malfunctioning immune system, increased free radical reactions, and how this damage might initiate apoptosis.²¹

Contributing Factors to Neuron Degeneration

- Oxidative stress
- Mitochondrial abnormalities
- Excitotoxicity
- Trophic factors
- Cytokines

Excitatory amino acids such as glutamate also have been shown to cause cell damage.²² The mechanism is the mediation of an influx of calcium across cell membranes causing damage to proteins, membrane lipids, and DNA.

Inadequate trophic (nutritional) factors contribute to cell death.²³ Experiments have shown that neurons die when they are disconnected from target tissue such as muscle.²⁴

Finally, cytokines are small proteins produced as a result of microglia brain cells reacting to damaged neurons. Cytokines produce other cytokines, starting a cascade of events that causes harmful inflammation in brain tissue. In some cases, however, the inflammation causes further damage. Cytokines' contribution to cell death is not fully understood, but a large number of glial cells have been observed post-mortem in the brains of Parkinson's victims.¹³

These complex cellular processes are both challenging and exciting. New information complicates the discovery of a cure for Parkinson's disease, but also has led to effective strategies for mitigating Parkinson's symptoms and slowing the disease's relentless progression.

Treatment

Because of its frightening motor deficits, co-morbid conditions, multi-factorial cell death, and slow progression, Parkinson's disease requires a multi-modal approach. This includes a disease onset strategy, cognitive-deficit treatment, pharmaceutical and natural dopamine support, and surgery as a last resort. Studies have shown that specific therapies both improve Parkinson's symptoms and slow their progression.^{25,26}



Disease Onset Strategy

The first step in confronting disease onset is an emotional assessment of the patient, with professional counseling if indicated. A support system involving family and peers helps the patient cope with motor symptoms. Legal, financial, and occupational counseling can provide the patient with a sense of control when symptoms progress.²⁷

Doctors usually concentrate on treating motor disorders that are disturbing to a person who has never experienced tremors, slow limb movements, or rigidity. While the focus is on treating these symptoms, mention must be made of the cognitive issues that arise with Parkinson's disease.



For Parkinson's patients, cognitive degeneration is a serious issue, with a profound impact on their quality of life. A thorough assessment of cognitive status is necessary whenever Parkinson's disease is first diagnosed. This includes memory, attention, intelligence quotient (IQ), and psychological testing for normative comparisons and baselines, which can be used later to quantify declines and indicate treatment.

While Parkinson's disease itself does not have an early-warning marker, dementia does. Cognitive assessments and functional tests such as a quantitative electroencephalograph (QEEG) can pinpoint a deficit early enough to allow the use of therapies proven to preserve mental faculties.

Diet is paramount at every stage of Parkinson's. A certified nutritionist should be an early recruit in the battle. Organic fruits, vegetables, and poultry, wild salmon, red beets, green beans, carrots, turnips, spinach, and red onions are especially beneficial in avoiding toxins. Unheated extra-virgin olive oil and herbs for flavoring should be used. Coffee has been associated with a lower risk of Parkinson's disease, likely as a result of caffeine's dopaminergic properties.²⁸ Sugars and fats must be avoided, as high-sugar diets have been correlated with a threefold risk for developing Parkinson's, and high-fat diets with a fivefold risk.^{29,30} Others to avoid include dairy, wheat, and

gluten products, margarine, fried foods, polyunsaturated oils (especially canola), sweetened foods, artificial sweeteners, processed food (such as deli meats), monosodium glutamate (MSG), alcohol (except red wine), chlorinated and fluoridated water, and microwaved foods.

Finally, weight-bearing exercise has been associated with an increase in testosterone that raises dopamine levels. Combined with the inherent benefit of improved body tone and coordination, an exercise program is indispensable in treating Parkinson's disease.

REPORT

Natural Prescriptions For Parkinson's Disease

By Bruce Scali

Cognitive Deficit Treatment

Cognitive assessment and treatment have been covered at length in previous issues of Life Extension (see, for example, "Inflammation and the Aging Brain," September 2003, and "Life Extension for the Brain," March 2004). Treatment of cognitive degeneration focuses on cholinergic medications and supplements. Typical medications for cognitive degeneration include Aricept®, Prostigmin®, tacrine, and Exelon®. Supplement support should include choline, phosphatidylserine, vinpocetine, and quercetin, available in formulations such as Cognitex.

Dietary support includes food rich in choline such as eggs, wheat germ, blueberries, broccoli, almonds, and caviar.

Pharmaceutical Support

Knowledge of the cellular processes involved in Parkinson's has opened new fronts in the battle against the disease. Pharmaceutical advances have had marked positive effects in Parkinson's disease patients, but side effects, subtherapeutic effects, the need for increased dosages, and eventual drug ineffectiveness after long usage temper drug efficacy. Pharmaceutical intervention is usually delayed until absolutely necessary.

When Parkinson's disease progresses, levodopa increases dop-amine, which improves motor symptoms. Bradykinesia and rigidity are more likely to ameliorate than tremor, while there is usually no improvement in postural stability, mental state, or autonomic nervous system dysfunction. Side effects include nausea and vomiting, which can be mitigated with a decarboxylase inhibitor such as carbidopa. Dyskinesias (abnormal involuntary movements) also can occur, with additional effects on speech, swallowing, respiration, and balance.³¹ Levodopa invariably presents dosage issues, as too much causes side effects and too little causes an early "wearing off" with a return of motor dysfunctions.

Pharmaceutical approaches now include the use of dopamine agonists before levodopa is used. Agonists improve dopamine receptor activity that facilitates the transport of dopamine. Drugs such as ropinirole are less likely than levodopa to lead to motor fluctuations and dyskinesias, but their effectiveness wanes after three years.²⁶ At that time, a combination therapy using levodopa and an agonist is initiated. This protocol allows lower dosages of levodopa, and shows positive motor effects.³²

Catechol-O-methyltransferase has been linked to motor fluctuations and dyskinesias, so its inhibition can extend the therapeutic effects of levodopa. Entacapone and tolcapone have been shown to reduce both motor fluctuations and levodopa "off" time.³³⁻³⁵

To address oxidative stress, selegiline (Eldepryl®), a monoamine oxidase (MAO) inhibitor, is prescribed in Parkinson's disease and can delay the evolution of disability.³⁶ Early use of an agonist could also have a neuroprotective effect.³⁷

Doctors prescribe drugs such as benztropine and amantadine to counteract dyskinesias and relieve tumors. Because their adverse effects include cognitive impairment, however, these drugs should be used with caution in patients over the age of 70.

Excitotoxicity is mitigated by the use of antigitamatergic agents such as N-methyl-D-aspartate (NMDA) receptor antagonists and calcium channel blockers such as Procardia®.³⁸ Common medications such as Celebrex® and Vioxx® inhibit the cyclooxygenase type 2 (COX-2) enzyme, which has been implicated directly with the inflammation associated with Parkinson's disease.³⁹

Natural Approaches

Readily available natural alternatives address many of the cell death processes related to Parkinson's disease and have shown dramatic results (see case study on p. 72). Such approaches are particularly efficacious in the early stages of the disease, when symptoms first appear but drugs are not yet indicated. Moreover, natural supplements are well tolerated, without the side effects associated with prescription medications.

Tyrosine and phenylalanine are amino acid precursors to dopamine, available from protein food sources and supplements.⁴⁰ Because protein interferes with levodopa absorption, its intake should be limited to one meal when a course of medication commences. Vitamin B6, zinc, and the adrenal hormone DHEA also have been shown to increase dopamine formation in the brain.^{41,42}

Natural Prescriptions for Parkinson's Disease

Dopamine support

Tyrosine, phenylalanine, vitamin B6, zinc, DHEA

Antioxidants

Vitamins C and E, polyphenols, bioflavonoids, proanthocyanidins, tocotrienols, curcumin, glutathione

If levodopa (Sinemet®) is used, vitamin B6 should be taken 3-4 hours after the last dose of levodopa, since vitamin B6, in some cases, may cause levodopa to convert to dopamine in the blood before it reaches the brain. "There are so many cellular factors involved with Parkinson's that we have to enlist every possible means of support to fight it," notes Eric Braverman, MD, director of New York City-based PATH Medical and an expert on brain-related illnesses. "Nutrition cannot be underestimated, and there's a whole dopamine diet available to Parkinson's patients. Even when physicians didn't know what dop-amine was, they treated Parkinson's symptoms with a diet rich in fava beans (a natural source of levodopa), and it helped."

A major focus in treating Parkinson's disease is reducing oxidative stress, and alternative approaches are instrumental in this regard. Intravenous infusion of chelators eliminates from the brain iron and other toxins that contribute to the formation of free radicals.

Antioxidants also act as chelators, and best results are achieved when a combination is used. Alternatives include vitamins C and E, polyphenols found in green and black teas, bioflavonoids that provide the red, pink, and purple colors in flowers, fruits, and vegetables, proanthocyanidins from grape seed extract, tocotrienols from palm oil, and curcumin.⁴³⁻⁴⁵



Of particular interest in Parkinson's disease is the role of glutathione, a metabolite of the essential amino acid methionine. Glutathione is contained in the cells of all living organisms. A billion years before life appeared on earth, when the atmosphere was gaseous and toxic, cells had to incorporate antioxidants like glutathione to survive. Glutathione also assists with the transport of amino acids across cell membranes.⁴⁶

Glutathione is not readily obtainable from food sources, but is available in supplements and can be introduced directly via an IV infusion. Adequate levels of glutathione in the body depend on cysteine, glycine, and glutamic acid. Of these, only cysteine ever seems to be in short supply. Cysteine is derived from methionine, an essential amino acid that transports methyl groups and sulfur into the body to form proteins. To ensure adequate glutathione, one may eat foods rich in sulfur, such as egg yolks, red peppers, and onions. Another way to raise glutathione levels is to supplement the body's cysteine with N-acetylcysteine or L-cysteine.⁴⁶

Metabolic support for mitochondria has been demonstrated with phosphatidylserine, acetyl-L-carnitine, and coenzyme Q10.⁴⁷ A recent study links creatine, an amino acid compound of methionine, glycine, and arginine, with mitochondria metabolism.⁴⁸

As for nonprescription anti-inflammatory alternatives, the options are many. Over-the-counter non-steroidal anti-inflammatory drugs (NSAIDs) such as aspirin, ibuprofen, and Motrin® have proven efficacy in Parkinson's disease.⁴⁹ Green and black teas also reduce swelling, as do herbs such as thunder god vine and holy basil.⁵⁰ Phellodendron amurense extracts have been used in traditional Chinese medicine and have been proven to inhibit COX-2 activity.⁵¹ These extracts are available in Nexrutine®.

Magnesium and the amino acid tryptophan, found in turkey, bananas, and milk, mitigate the dyskinesia that can occur with dopamine therapies.⁵²

Finally, as with any neurological complaint, gentle stimulation introduced with cranial electrical stimulation (CES) or transcranial magnetic stimulation (TCMS) units can keep circuits open and mitigate motor disconnect symptoms.

With so many natural alternatives for relief of Parkinson's disease symptoms, more serious medical approaches can be postponed until absolutely necessary, as described below.

Metabolic support

CoQ10, phosphatidylserine, L-carnitine, creatine

Anti-inflammatories

NSAIDs, polyphenols, holy basil, thunder God vine, Nexrutine®

Anti-cholinergics

Tryptophan, magnesium

Parkinson's Disease Treatment Plan

- Counseling
- Nutritional support
- Cognitive assessment
- Conventional approaches
- Natural alternatives



Surgery

When Parkinson's disease has progressed such that it can no longer be managed using supplements and medications, two options remain.

The first, ablative surgery, disconnects areas in the brain responsible for muscle movement. Destructive surgery relieves rigidity, bradykinesia, and tremors. Symptom relief occurs in less than 90% of patients, but is complete and unilateral. Surgical complications include cerebral infarction, difficulty swallowing, cognitive impairment, and visual field defects.⁵³ Thus, ablative surgery is indicated only in patients with a long history of tremor that cannot otherwise be controlled.

The second option, deep brain stimulation, involves placing an electrode into the thalamus with a connection to a pulse generator implanted in the chest. Deep brain stimulation regulates muscle movement. Its advantages are that it is reversible and will not preclude the benefit of future remedies; its disadvantages include its cost, discomfort, risk of infection, and need for replacement.⁵⁴

Future Promise

New drugs for Parkinson's disease are constantly being evaluated. Among the clinical trials under way at the National Institutes of Health are those related to cognitive decline, depression, and sleep disorders. A new transdermal patch could provide more controlled drug delivery so that the problems of overdosing or "wearing off" can be avoided.

Information related to Lewy bodies (abnormal structures found in certain areas of the brain) and glutathione may prove to be significant for developing an early-warning marker for Parkinson's disease. Patients with incidental Lewy bodies do not have Parkinson's symptoms but are thought to have early Parkinson's disease. These same patients have reduced levels of glutathione, so perhaps this combination will lead to a test that can identify those most at risk.⁵⁵

Further research into the importance of trophic factors may have a major impact on the treatment of Parkinson's disease.^{56,57} These factors, such as glial-derived neurotrophic factor and brain-derived neurotrophic factor, are important for neuron protection and nutrition. Before they can be clinically used, however, issues relating to their delivery to specific brain regions, dosages, and factor selection must be resolved.

Finally, two highly experimental Parkinson's disease treatments now under investigation are transplanted grafts of adrenal or other tissue from fetuses or other species into the SNc to promote dopamine production, and gene therapies for maintaining the integrity of mitochondria cell membranes. Stem cells that would replace dopamine-producing neurons offer great promise.



Moving Forward

For all that remains unknown about Parkinson's disease, much can be done to counter its effects. First is the proper diagnosis and identification of contributing factors related to each case. Next is proper counseling and education. Finally, a global plan that incorporates lifestyle, diet, and treatment options, with initial and continuing emphasis on natural alternatives, must be formulated, administered, and monitored by experienced health care personnel. With such a plan, the Parkinson's patient can successfully fight a stalling action until new research results in a definitive cure.

REPORT

George A. — A Parkinson's Case Study

“George A.” (a pseudonym) is a 70-year-old male who first experienced motor dysfunction symptoms nine years ago. He visited the Mayo Clinic in Jacksonville, FL, a few times but remained undiagnosed and untreated.

When his symptoms persisted and deteriorated, he looked elsewhere for help. He was referred to Eric Braverman, MD, a specialist in complementary medicine and an acknowledged expert on brain-related diseases.

George A. was interviewed while being treated with an IV infusion in Dr. Braverman’s office at PATH Medical in New York City. A brief case review with Dr. Braverman follows the interview.



Life Extension: What were your initial symptoms?

George A.: It started when I was skiing, and my legs wouldn’t go where I wanted them to go. There was a delay, like a weight was on them. It was the strangest feeling, so I went to the Mayo Clinic.

LE: What did they do?

George A.: They had me walk down a hallway and they would say, “Turn . . . turn . . . turn . . .,” and they watched the way I turned. But they said they didn’t know what it was.

LE: What other tests did they do?

George A.: Blood work, MRI. I saw a urologist, an internist, even a psychiatrist. Nobody found anything.

LE: Did they ever mention Parkinson’s disease?

George A.: No.

LE: What did they tell you when they were finished? Did they give you anything?

George A.: All they said was to exercise, watch my diet, keep my cholesterol down, and I would be fine.

LE: Were you?

George A.: No. My symptoms got worse. So I went back to get some answers. But they didn’t give me any. What finally got me was I started to fall asleep at traffic lights. The beeping horns woke me up. That’s when my wife said, “They’re not doing anything for you.”

LE: What happened next?

George A.: A secretary in another doctor’s office told me about Dr. Braverman, that he specializes in this type of problem. So I called him.

LE: How long ago was that?

George A.: Three years, maybe a little more.

LE: How were your symptoms then?

George A.: They were worse. I was much more tired, my legs got very heavy, and my balance was off. My whole general feeling was not good.

LE: Did you have any tremors?

George A.: No. I never got tremors. But Dr. Braverman knew right away.

LE: What kinds of tests did he do?

George A.: Everything. Blood tests, brain tests, ultrasound.

LE: What did he find?

George A.: He said I had Parkinson's disease. And his neurologist said the same thing. He found a lot of things wrong with me. But the main thing was the heavy metals. He had me come here two days a week for drips.

LE: Chelation?

George A.: And vitamins. Over time, my tests were better.

LE: What do you take?

George A.: All kinds of things. Sinemet®. Cardura.® Glutathione.

LE: What supplements do you use?

George A.: I just started on 1000 milligrams of CoQ10, which is supposed to help. Glucosamine, selenium, creatine, zinc, multivitamin, methionine, antioxidants, carnitine, milk thistle, fish oil. I take quite a bit.

LE: How long after Dr. Braverman started treating you did you see an improvement?

George A.: Probably three, four months.

LE: Did your fatigue go away?

George A.: The fatigue went away.

LE: And the heavy legs?

George A.: They went away. Sometimes they come back, if I miss a treatment.

LE: And your balance?

George A.: The balance is still . . .

LE: Sometimes it goes?

George A.: I have to be careful on ice.

LE: We have to be careful on ice, too. Do you have any idea how you were exposed to toxins?

George A.: I worked as a plumber with my dad for 10 years. Lead was the water pipe of the world. Everyone had a lead main, and then copper pipes. What I dealt with for many years was asbestos. I used to do boilers. He had it.

LE: Who had what?

George A.: Parkinson's. My dad had it. But we didn't know that he had it. He was always falling down.

LE: Why did you go last year to the Motor Control Clinic at the National Institutes of Health?

George A.: Dr. Braverman felt that I had accomplished something. He wanted them to look at me and see if his treatment had



made me the way I am now. They had about 12 doctors sitting around, questioning, and they touched my arms and spoke to me. Everybody had a chance to ask me questions, how I sat, how I stood . . . very nice doctors, very knowledgeable. But nobody commits to anything. [Laughs]

LE: You volunteered for that?

George A.: I wanted to find out if what Dr. Braverman was doing could elevate me to another level, which is what happened. As far as I'm concerned, I stopped the progress of the disease. But I have to stay with the regimen I'm on because I'm afraid that if I stop, my legs will get heavy again.

LE: You're having your IV drip as we talk. What are your feelings about your health issues over the last decade?

George A.: I'm a little disappointed that I got sick. I realize now that it came from my father.

LE: Genetics?

George A.: Positively. Now I know that when he used to shake and fall down, he had Parkinson's.

LE: How do you feel right now?

George A.: I feel fortunate to have Dr. Braverman. He has something there. When he's exposed to other doctors, they flock around him like bees to a flower. And I'm fortunate that he takes an interest. I guess he does with everyone. I just feel he takes a special interest in me.

LE: What's your outlook for the future?

George A.: Hopefully, I can keep the dogs at bay. If I start to slip, I'm sure there are ways for Dr. Braverman to get me back on track. He doesn't like to use medication, but when he has to, he does.

LE: Are you comfortable if you have to do this [I.V. infusion] for the rest of your life?

George A.: I could live with it.

George A.'s case has many of the classic components of Parkinson's disease: multiple symptoms, multiple factors (toxins, genetics), slow progression, response to dopamine medication, multiple treatment modalities. The following is a brief case review with Dr. Braverman.

LE: George had Parkinson's disease?

Braverman: Yes. We have to educate the public that Parkinson's is a huge cluster of neuropsychiatric problems associated with a loss of motor control of all degrees. From incontinence, to shuffling gait, to droopy face.

LE: What happened at the National Institutes of Health?

Braverman: Parkinson's is not supposed to be a reversible disease, so they redefined his diagnosis. I've seen the same thing with multiple sclerosis. Seven years ago, I told the chairman of one of the major university medical centers that antidepressants, anticonvulsants, GABA agents, and nutrients could slow the multiple sclerosis progression. He hung up the phone on me. Now he's just published a paper saying that multiple sclerosis can be reversed with antidepressants.

LE: Glutathione seems to play an important role in treating Parkinson's disease.

Braverman: Yes, along with many other things. The Life Extension approach of using supplements and nutrients works for a lot of illnesses, not just Parkinson's. Like cognitive decline, for example.

LE: You did a cognitive assessment of George?

Braverman: That's standard PATH protocol. He had a 15 out of 30 on the Mini-Mental State [MMSE] when we first saw him. He was headed for dementia. We used all the cholinergic meds and supplements to treat that while we addressed his Parkinson's. He went to 30 out of 30 in one year. His brain age [speed] shows a dramatic improvement. Cognitive degeneration is common in neurodegenerative disease, but often overlooked. George lost what I call the "edge effect"—the connection between

his brain and his body.

LE: There is still a lot we don't know about the cause and treatment of Parkinson's, right?

Braverman: That's why we sent George to NIH. We were trying to secure a grant to study alternative approaches to Parkinson's. We can do all the studies we want, but we have to help real people like George now. We've got to use anything and everything that might help. We did that with him. Restored his edge.

REPORT

Natural Prescriptions For Parkinson's Disease

By Bruce Scali

The Parkinson's Prescription: Natural Treatments for Multiple Causes			
Causative Factor	Diagnostic Test	Medical Treatment	Natural Approach
Dopamine loss	P300 brainwave- brain voltage	Levodopa, ropinirole, COMT inhibitors	Tyrosine, vitamin B6, zinc, DHEA, phenylalanine
GABA and serotonin loss	Brain map (QEEG)	Paxil®, Effexor®, other antidepressants	L-theanine, vitamin B12, GABA, inositol, St. John's wort, tryptophan
Inorganic toxins/ heavy metals (iron, manganese, copper)	EDTA challenge, RBC, serum heavy metals lead, cadmium, (zinc)	D-penicillamine, BAL	IV chelation, zinc, CoQ10
Organic toxins (MPTP, pesticides, hydrocarbons)	Pesticide levels, fat biopsy	(None established)	Glutathione, N-acetylcysteine, methionine, cysteine, sulfur
Oxidative stress	Homocysteine, vitamin B12, serum vitamin E and C, selenium, beta-carotene	Selegiline, antidepressants (e.g., Zoloft®, glutathione, polyphenols, Wellbutrin®)	Super Alpha Lipoic Acid with Biotin, curcumin, bioflavonoids, tocotrienols
Inflammation	CRP, ESR, TH/TS, interleukin 6 and 8	COX-2 inhibitors (e.g., Vioxx®, Celebrex®)	NSAIDs (e.g., aspirin, ibuprofen), green and black tea, Nexrutine®
Diminished vascular flow, stenosis, cholesterol, mini-strokes	MRI, MRA, PET scan, ultrasound, ABI	Statins (e.g., Zocor®, Lipitor®)	Aspirin, niacin, red yeast, chelation, IV HDL, policosanol
Hormonal deficiencies	DHEA, testosterone, estrogen, progesterone	HGH injections, adrenal hormone, testosterone, estrogen, progesterone	Super MiraForte, HGH secretagogues, DHEA
Petrification: calcium in the brain and blood vessels with amyloid, mini-strokes	Ionized calcium, calcitonin, para-thyroid, progesterone, bone density studies	NMDA antagonists, calcium channel blockers (e.g., Procardia®)	Calcium, boron, strontium, calcitonin, vitamin D
Electrical loss or pause	BEAM (QEEG), PET scan	ECT	CES, TCMS
Metabolic loss	P300 brainwave, PET and SPECT scans	(None established)	CoQ10, creatine, phosphatidylserine, acetyl-L-carnitine

References

1. Mohr E, Mendis T, Grimes JD. Late cognitive changes in Parkinson's disease with an emphasis on dementia. *Adv Neurol.* 1995;65:97-113.
2. Scali B. Life extension for the brain. *Life Extension.* March, 2004:46-7.
3. Available at: <http://www.parkinson.org/encarta99htm>. Accessed March 23, 2004.
4. Bernheimer H, Birkmayer W, Hornykiewicz O, Jellinger K, Seitelberg F. Brain dopamine and the syndromes of Parkinson and Huntington. *J Neurol Sci.* 1973 Dec;20(4):415-55.

5. Kessler II. Epidemiologic studies of Parkinson's disease: II. A hospital-based survey. *Am J Epidemiol.* 1972 Apr;95(4):308-18.
6. Schoenberg BS, Osuntokun BO, Adeuja AOG, et al. Comparison of the prevalence of Parkinson's disease in the biracial population of Copiah County, Mississippi. *Neur.* 1985;35:841-845.
7. Zhang ZX, Roman GC. Worldwide occurrence of Parkinson's disease: an updated review. *Neuroepidemiology.* 1993;12(4):195-208.
8. Betemps EJ, Buncher CR. Birthplace as a risk factor in motor neurone disease and Parkinson's disease. *Int J Epidemiol.* 1993 Oct;22(5):898-904.
9. Tan LC, Tanner CM, Chen R, et al. Marked variation in clinical presentation and age of onset in a family with a heterozygous parkin mutation. *Mov Disord.* 2003 Jul;18(7):758-63.
10. Duvoisin RC. The genetics of Parkinson's disease. *Adv Neurol.* 1993;60:306-15.
11. Duvoisin RC, Eldridge R, Williams A, Nutt J, Calne D. Twin study of Parkinson disease. *Neurology.* 1981 Jan;31(1):77-80.
12. Smith CA, Gough AC, Leigh PN, et al. Debrisoquine hydroxylase gene polymorphism and susceptibility to Parkinson's disease. *Lancet.* 1992 Jun 6;339(8806):1375-7.
13. Riley DE. Secondary Parkinsonism, in *Parkinson's Disease and Movement Disorders, Third Edition.* Edited by Jankovic J, Tolosa E. New York, LW&W. 1998;317-328.
14. Tröster AI, Fields JA, Koller WC. Parkinson's disease and Parkinsonism, in *Textbook of Geriatric Neuropsychiatry, Second Edition.* Edited by Coffey CE, Cummings JL. Washington, D.C., American Psychiatric Press, 2000;562.
15. Tröster AI, Fields JA, Koller WC. Parkinson's disease and Parkinsonism, in *Textbook of Geriatric Neuropsychiatry, Second Edition.* Edited by Coffey, CE, Cummings JL. Washington, D.C., American Psychiatric Press, 2000;563.
16. Hughes AJ, Daniel SE, Kilford L, Lees AJ. Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinicopathological study of 100 cases. *J Neurol Neurosurg Psych.* 1992 Mar;55(3):181-4.
17. Olanow CW, Jenner P, Tatton N, Tatton W. Neurodegeneration and Parkinson's disease, in *Parkinson's Disease and Movement Disorders, Third Edition.* Edited by Jankovic J, Tolosa E. New York, LW&W. 1998;67-103.
18. Wolff SP, Garner A, Dean RT. Free radicals, lipids and protein degradation. *Trends Biol Sci.* 1986;11:27-31.
19. Hirsch EC, Brandel JP, Galle P, Javoy-Agid F, Agid Y. Iron and aluminum increase in the substantia nigra of patients with Parkinson's disease: an x-ray microanalysis. *J Neurochem.* 1991 Feb;56(2):446-51.
20. Singer TP, Castagnoli N Jr, Ramsay RR, Trevor AJ. Biochemical events in the development of parkinsonism induced by 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine. *J Neurochem* 1987 Jul;49(1):1-8.
21. Mizuno Y, Matuda S, Yoshino H, Mori H, Hattori N, Ikebe S. An immunohistochemical study on a-ketoglutarate dehydrogenase complex in Parkinson's disease. *Ann Neurol.* 1994 Feb;35(2):204-10.
22. Choi DW. Glutamate neurotoxicity and diseases of the nervous system. *Neuron.* 1988 Oct;1(8):623-34.
23. Winkler J, Thal LJ. Clinical potential of growth factors in neurological disorders. *CNS Drugs.* 1994;2:465-478.
24. Moller JC, Sautter J, Kupsch A. Potential of neurotrophic factors in therapy of Parkinson's disease. *J. Neural Transm.* 1996;48:103-112.
25. Parkinson Study Group: Effect of deprenyl on the progression of disability in early Parkinson's disease. *N Engl J Med.* 1989 Nov 16;321(20):1364-71.
26. Adler CH, Sethi KD, Hauser RA, et al. Ropinirole for the treatment of early Parkinson's disease. *Neurol.* 1997 Aug;49(2):393-

27. Olanow CW, Koller WC. An algorithm (decision tree) for the management of Parkinson's disease: treatment guidelines. *Neurol.* 1998 Mar;50(Suppl 3):S1-S57.
28. Benedetti MD, Bower JH, Maraganore DM, et al. Smoking, alcohol, and coffee consumption preceding Parkinson's disease: a case-control study. *Neurology.* 2000 Nov 14;55(9):1350-8.
29. Hellenbrand W, Boeing H, Robra BP, et al. Diet and Parkinson's disease. II: A possible role for the past intake of specific nutrients. Results from a self-administered food-frequency questionnaire in a case-control study. *Neurology.* 1996 Sep;47(3):644-50.
30. Logroscino G, Marder K, Cote L, Tang MX, Shea S, Mayeux R. Dietary lipids and antioxidants in Parkinson's disease: a population-based, case-control study. *Ann Neurol.* 1996 Jan;39(1):89-94.
31. Tanner CM. Drug-induced movement disorders, in *Extrapyramidal Disorders in Handbook of Clinical Neurology*, Vol 5. Edited by Vinken PJ, Bruyn GW, Klawans HL. Amsterdam. Elsevier Science. 1986;185-204.
32. Brooks DJ, Torjanski N, Burn DJ. Ropinirole in the symptomatic treatment of Parkinson's disease. *J Neural Trans Suppl.* 1995;45:231-8.
33. Martinez-Martin P, O'Brien CF. Extending levodopa action: COMT inhibition. *Neur.* 1998 Jun;50 (Suppl 6):S27-S32.
34. Waters CH, Kurth M, Bailey P, et al. Tolcapone in stable Parkinson's disease: efficacy and safety of long-term treatment. *Neurology.* 1997 Sep;49(3):665-71.
35. Rajput AH, Martin W, Saint-Hilaire MH, Dorflinger E, Pedder S. Tolcapone improves motor function in parkinsonism patients with the "wearing off" phenomenon: a double-blind, placebo-controlled, multicenter trial. *Neurology.* 1997 Oct;49(4):1066-71.
36. Parkinson Study Group. Effectiveness of l-dopa on the progression of disability in early Parkinson's disease. *Ann Neurol.* 1996 Jul;40(1):99-107.
37. Olanow CW. Attempts to obtain neuroprotection in Parkinson's disease. *Neurology.* 1997 Jul;49(Suppl 1):S26-S33.
38. Schapira AH, Olanow CW. Neuroprotection in Parkinson's disease. *JAMA.* 2004 Jan 21;291(3): 358-64.
39. Teismann P, Tieu K, Choi DK, et al. Cyclooxygenase-2 is instrumental in Parkinson's disease neurodegeneration. *Proc Natl Acad Sci USA.* 2003 Apr 29;100(9):5473-8.
40. Growdon JH, Melamed E, Logue M, Hefti F, Wurtman RJ. Effects of oral L-tyrosine administration on CSF tyrosine and homovanillic acid levels in patients with Parkinson's disease. *Life Sci.* 1982 Mar 8;30(10):827-32.
41. Sandyk R, Pardeshi R. Pyridoxine improves drug-induced parkinsonism and psychosis in a schizophrenic patient. *Int J Neurosci.* 1990 Jun;52(3-4):225-32.
42. Li H, Klein G, Sun P, Buchan AM. Dehydroepiandrosterone (DHEA) reduces neuronal injury in a rat model of global cerebral ischemia. *Brain Res.* 2001 Jan 12;888(2):263-266.
43. Fahn S. An open trial of high-dosage antioxidants in early Parkinson's disease. *Am J Clin Nutr.* 1991 Jan;53(Suppl 1):S380-S382.
44. Levites Y, Amit T, Youdim MB, Mandel S. Involvement of protein kinase C activation and cell survival/cell cycle genes in green tea polyphenol (-)-epigallocatechin 3-gallate neuroprotective action. *J Biol Chem.* 2002 Aug 23;277(34):30574-80.
45. Lim GP, Chu T, Yang F, Beech W, Frautschy SA, Cole GM. The curry spice curcumin reduces oxidative damage and amyloid pathology in an Alzheimer transgenic mouse. *J Neurosci.* 2001 Nov 1;21(21):8370-77.
46. Braverman ER. *The Healing Nutrients Within Third Edition.* North Bergen. Basic Health Pub. 2003;118-130.
47. Schulz JB, Henshaw DR, Mathews RT, Beal MF. Coenzyme Q10 and nicotinamide and a free radical spin trap protect

against MPTP neurotoxicity. *Exp Neurol.* 1995 Apr;132(2):279-83.

48. Beal MF. Mitochondria, oxidative damage, and inflammation in Parkinson's disease. *Ann N Y Acad Sci.* 2003 Jun;991:120-31.

49. Dzenko KA, Weltzien RB, Pachter JS. Suppression of A beta-induced monocyte neurotoxicity by antiinflammatory compounds. *J Neuroimmunol.* 1997 Dec;80(1-2):6-12.

50. Li RW, David Lin G, Myers SP, Leach DN. Anti-inflammatory activity of Chinese medicinal vine plants. *J Ethnopharmacol.* 2003 Mar;85(1):61-7.

51. Mori H, Fuchigami M, Inoue N, Koda A, Nishioka I. Principle of the bark of *Phellodendron amurense* to suppress the cellular immune response. *Plant Med.* 1994 Oct;60(5):445-9.

52. Sandyk R. L-tryptophan in Neuropsychiatric disorders: a review. *Int J Neurosci.* 1992 Nov-Dec;67(1-4):127-44.

53. Hammerstad J, Hogarth P. Parkinson's disease: surgical options. *Curr Neurol Neurosci Rep.* 2001 Jul;1(4):313-9.

54. Pollak P, Benabid A-L, Krack P, Limousin P, Benazzouz A. *Deep Brain Stimulation in Parkinson's Disease and Movement Disorders*, Third Edition. Edited by Jankovic J, Tolosa E. New York, LW&W. 1998;1085-1101.

55. Dexter DT, Sian J, Rose S, et al. Indices of oxidative stress and mitochondrial function in individuals with incidental Lewy body disease. *Ann Neur.* 1994 Jan;35(1):38-44.

56. Chaturvedi RK, Agrawal AK, Seth K, et al. Effect of glial cell line-derived neurotrophic factor (GDNF) co-transplantation with fetal ventral mesencephalic cells (VMC) on functional restoration in 6-hydroxydopamine (6-OHDA) lesioned rat model of Parkinson's disease: neurobehavioral, neurochemical and immunohistochemical studies. *Int J Dev Neurosci.* 2003 Nov;21(7):391-400.

57. Hoffer BJ, Hoffman A, Bowenkamp K, et al. Glial cell line-derived neurotrophic factor reverses toxin-induced injury to midbrain dopaminergic neurons in vivo. *Neurosci Lett.* 1994 Nov 21;182(1):107-11

All Contents Copyright © 1995-2009 Life Extension Foundation All rights reserved.

LifeExtension®

These statements have not been evaluated by the FDA. These products are not intended to diagnose, treat, cure or prevent any disease. The information provided on this site is for informational purposes only and is not intended as a substitute for advice from your physician or other health care professional or any information contained on or in any product label or packaging. You should not use the information on this site for diagnosis or treatment of any health problem or for prescription of any medication or other treatment. You should consult with a healthcare professional before starting any diet, exercise or supplementation program, before taking any medication, or if you have or suspect you might have a health problem. You should not stop taking any medication without first consulting your physician.