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Science is spoken here.

Maine PD News

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Exercise and Parkinson's disease

by Michael Kleinman, D.O.

The treatment of Parkinson's disease (PD) is often thought of as just medications, or in advanced cases, surgical treatment. There are in fact many non-medication treatments for PD, and exercise is one of them. Exercise has been shown in studies to improve not only the movement related symptoms, but also many non-motor symptoms of Parkinson's. Starting an exercise regimen may not seem to be as important when one initially receives a diagnosis of PD as the symptoms are often no more than a minor nuisance at that point. However, getting started early is vital, as it becomes harder to commit to making exercise a routine part of life as one's condition progresses. Participating in regular exercise can seem daunting to someone who has lived with PD for years. There are many different ways to go about getting exercise and these can be tailored to an individual's needs and abilities.

Getting Started

Exercise and physical activity are vital to an early treatment regimen in PD. By participating in exercise, one takes an active role in the fight against the disease. It can also give a person with PD a sense of empowerment over the course of their condition. In early stage PD, it is most important to choose an exercise regimen or activity that one will have a high probability of participating in regularly, rather than choosing one specific exercise over another. The ideal regimen will include activities that are enjoyable, and that also help control PD symptoms.

Identifying and addressing barriers to exercise will help overcome the

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***Announcing* Dr. Sarah Dodwell**, *who joined MMP Neurology in Scarborough, Maine this summer. She completed movement disorders fellowship in the Harvard University Neurology Program at Beth Israel Deaconess Medical Center.*

daunting nature of beginning a new fitness regimen. In later stages of PD, one of these major barriers is apathy.

Apathy is a troublesome non-motor symptom of PD that involves a lack of motivation and reduction of emotional expression, and can be difficult to treat. In my experience, starting an exercise regimen before apathy becomes problematic may even prevent this symptom from becoming a factor in reducing one's quality of life.

A referral to a physical therapist experienced with PD is often helpful in getting started with an exercise regimen. Periodically revisiting courses of physical therapy as PD progresses is important in order to customize and adjust one's exercise program to fit one's needs and capabilities. Home-based physical therapy programs are also an option for those who have a difficult time adhering to outpatient therapy programs. Exercise classes can be a good way of building social interactions both within and outside the PD community. The care partner also plays a vital role in providing motivation to stay active. Exercising with a family member can have a positive effect on relationships.

Exercise as a Symptomatic Treatment
Numerous research studies have found that various exercise regimens improve many bothersome symptoms of PD, including reducing stiffness, increasing strength, and improving balance, gait and mobility. Research on exercises aimed at improving gait and balance have shown a consistent improvement in balance with a reduction in the number of falls. Exercise has also been shown to improve overall quality of life.

What Types of Exercises are Good for PD?
Although there have been many studies which have demonstrated the positive effects of exercise, there is little data to say that one exercise program is superior to another. In general, a physical activity program that incorporates a variety of movements involving a large range of motion are preferred over performance of a single repetitive exercise. Ideally, an exercise routine would include lighter intensity activities focusing on stretching and balance, as well as higher intensity activities. Some specific examples of exercises for PD include:

- Brisk walking
- Aerobics classes
- Resistance exercises (light weight)
- Participation in sports
- Biking
- Dancing
- Yoga
- Tai Chi

Many patients have been referred to participate in the LSVT-BIG program for physical therapy through local outpatient rehabilitation centers.

This is an intensive program focusing on promoting high amplitude movements. LSVT-BIG has been shown to improve motor functioning in PD, as well as increase walking speed. We are now fortunate to have practitioners experienced in this program at several different locations throughout Maine. It is very important to remember that performance of the exercises at home after the completion of the course will maximize the likelihood of experiencing a sustained benefit.

The Effects of Exercise on the Non-Motor Aspects of PD
For many patients with PD, the most troublesome symptoms are related to the non-motor aspects of PD. These may include

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problems with fatigue, constipation, insomnia, anxiety, depression, and cognitive dysfunction. In many cases, medications to address these

symptoms are of limited effectiveness, but exercise can help. In one series, patients who started exercise early rather than later in their illness were found to have fewer symptoms of depression.

Cognitive symptoms, problematic in PD, include slowed processing speed, difficulties with multi-tasking, and impaired decision-making skills. These cognitive domains are referred to as frontal executive functions, impairment of which is seen in a significant number of people with PD, even relatively early on the course of the condition. Research on exercise in PD has shown an improvement in executive function skills, with the effects noted up to six months into treatment.

The Effects of Exercise on the Brain in PD
It is known that the movement symptoms of PD relate to a loss of the dopamine producing cells in a structure called the midbrain. Research has been performed on the effects of exercise on mice treated with a substance that mimics the effects of PD on the human brain. The mice that performed treadmill exercise were found to have lost fewer dopamine producing cells, when compared with the mice that did not perform any exercise. The exact mechanism for this protection of dopamine producing cells is unclear, but is thought to be due to a reduction of inflammation in the brain.

Mice with Parkinsonism were also found to have increased connections with other cells within the movement center of the brain, the basal ganglia, after being exposed to an exercise program. This suggests that exercise can enhance neuroplasticity, the brain's ability to form new connections and pathways. Studies of the levels of certain substances that are thought to promote neuroplasticity have found an increase in the level of these substances in humans with PD after the completion of exercise.

Structural imaging studies of the brain have also demonstrated positive changes after exercise in people with PD, with increased volumes of certain parts of the brain noted. Similar effects on brain volume have also been seen outside of the PD population in older patients, underscoring the importance of physical activity for overall brain health as we age.

How Much Exercise Should One be Doing?
There is no single number to describe the frequency and duration of exercise that applies to every person with PD. If you are not initially an active person, or you have physical limitations, then it is advised that you start with a shorter duration of exercise and gradually increase with time. Those who are younger or are in the mild stage of PD should be able to tolerate longer durations of exercise and perform higher intensity exercises. For most people with PD, a goal of 30 minutes of exercise 3-4 times a week is achievable. Many experts believe that more intensive programs may confer a higher degree of benefit.

In conclusion, exercise plays a vital role in maximizing one's functional abilities and quality of life across the spectrum of PD, should be incorporated into the treatment plan at the time of diagnosis, and continued throughout the course of the condition.

Dr. Kleinman is a neurologist specializing in movement disorders at MMP Neurology.

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The Neuropsychological Evaluation for Deep Brain Stimulation

by Tom Miller, Ph.D.

A neuropsychological evaluation is a method for examining the quality of brain function and for determining a patient's cognitive strengths and any limitations. It involves several steps:

- a clinical interview to obtain information from the patient and his or her spouse or other family members about daily functioning, details of ongoing problems, and any concerns about cognitive functioning (e.g. problems with attention, memory, mental processing,etc.);
- obtaining additional background information from medical records and reports, review of any previous testing, and other relevant information;
- the administration of various tests to examine cognitive functioning in a number of areas, including intellectual abilities, attention, language, learning and memory, visuospatial abilities, sensory-motor functioning, executive function, emotional status, and personality.

These areas of cognition involve different regions of the brain, and a person's performance on testing can reveal the relative efficiency or impairment in these brain regions.

A neuropsychological evaluation is scheduled before a deep brain stimulation procedure (DBS) for several reasons:

- to establish of pre-surgical baseline of functioning in these areas of cognition;
- to determine whether there are any difficulties that may be exacerbated by surgery;
- to determine whether there are any difficulties that may interfere with adjustment after surgery.

DBS involves implanting electrodes into brain regions that regulate attention, aspects of language, and memory retrieval. Parkinson's disease can contribute to difficulties in these areas of functioning. It is important to determine the extent of any difficulties before making a decision whether to proceed with DBS.

Memory impairment can make it difficult for a patient to follow recommendations and adhere to a medication schedule, and significant memory impairment may indicate early signs of dementia.

It is important to determine whether problems with memory are beyond the ordinary occasional memory lapses that many of us encounter from time to time. Testing is necessary to determine the nature and extent of memory problems: distinguishing between:

- "ordinary" and occasional lapses of memory (no interference with functioning); inefficiencies in memory (may be annoying at times but without much interference);
- mild memory impairment (difficulties with remembering details of events occurring in recent weeks or months);
- moderate memory impairment (inability to recall many details of events, conversations, appointments occurring more recently; needing frequent reminders, increasing interference with daily functioning);

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- severe memory impairment (inability to recall information within minutes, requiring multiple repetition of instructions or requests, inability to follow conversation, etc);
- lapses of attention and memory (difficulties with memory may actually be related to lapses of attention, distractibility, or inability to concentrate effectively).

Memory testing helps to determine whether difficulties are related to initial encoding of new information; the process of storing new information; or the retrieval of information from memory. These stages of memory involve different areas of the brain – and information about performance provides your doctor with information about functioning in specific brain regions.

It is also important to examine emotional status and personality functioning. Acute anxiety or deep depression interferes with thinking, planning, and problem solving. An inability to function effectively in these areas might impair judgment to an extent that the process of managing DBS becomes overly complicated and the advantages of DBS become difficult to see. Personality functioning itself may suffer, and a person may find themselves overly anxious and worried, or without interest or motivation in their lives.

It is possible to master bouts of anxiety or hopelessness by learning and practicing various mental or behavioral strategies – sometimes referred to as cognitive-behavior therapy, or CBT. The use of these tools can help restore emotional balance along with a sense of confidence and ease. If significant emotional distress is a factor, a referral can be made for therapy to help improve overall functioning.

Dr. Tom Miller practices at Maine Medical Center Department of Psychiatry, Geriatric Outpatient Psychiatry, 66 Bramhall Street, Portland, ME 04102

PD Pearls

Manganese toxicity does not cause PD, but may cause a static, unchanging parkinsonism.

The "Parkinson personality" is one of avoiding risk, caffeine, nicotine, and alcoholism, and is often present for many years prior to the onset of the motor symptoms of PD.

PD can occur at any age, but is most common in older people. Based on U.S. Census Bureau count of the number of Mainers over 65 and over 80 in 2015, along with the incidence of PD as people age, one can estimate at least 3500 elderly Mainers currently have PD.

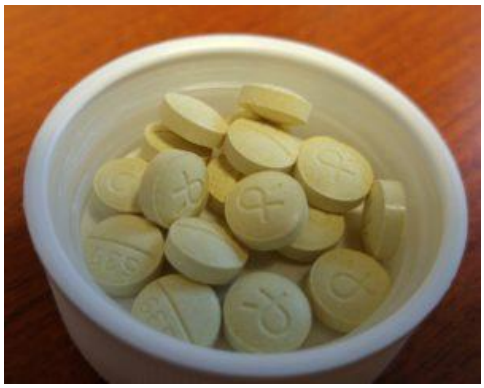
"ON" time is when medications are working and symptoms are lessened.

"OFF" time is when medications are not working and symptoms are present or worse.

Immediate release carbidopa/levodopa can cause allergic skin reactions

by Bill Stamey, M.D.

Sinemet (carbidopa-levodopa) 25/100 immediate release (IR) tablets contain a yellow dye, D&C Yellow #10 Lake. Per the prescribing information, rash is a possible side effect (1).



carbidopa/levodopa 25/100

There are a few case reports to support rash as a possible side effect in two publications. The first paper to cite this effect noted five patients who experienced rash associated with carbidopa/levodopa 25/100, and authors concluded the yellow dye in this formulation was responsible (2). Substitution with carbidopa/levodopa 25/250 or 10/100, formulations that do not contain the yellow dye, did not cause a rash.

The second paper reported two patients with rashes resulting from IR carbidopa/levodopa 25/100 (3). In both cases, stopping the drug resolved the rash. In one case, compounded pure levodopa and Stalevo 100 (carbidopa 25 mg/levodopa 100 mg/entacapone 200 mg), both of which are made without the yellow dye, were given in successive trials with no rash.

These are old reports, and searches for any more current information come up dry. The incidence is apparently very low, and the phenomenon not well known. I have seen this in patients and tend to try the CR, ER, or SA formulations, which do not contain the yellow dye, as alternatives.

Anecdotally, I have heard of allergic gastritis with IR carbidopa/levodopa, but have not seen this reported in literature. Some patients may also experience nausea with the IR, but not other formulations of carbidopa/levodopa.

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Dietary Supplements

by Bill Stamey, M.D.

It is estimated that over 50% of U.S. citizens use a daily supplement, whether an herbal, plant-based, amino acid, or a vitamin. Many PD patients take one or more of these compounds. My experience is that there is often an assumption that these products are natural, and therefore good for you, and the supply in stores is safe because the FDA will protect us from harmful substances. These points should be taken separately.

Are supplements good for you?

The issue is complex. Some might be, and some are not. Consider vitamins, for example. Vitamins are by definition required for good health, but the vast majority of us don't require vitamin supplements because we get the very small amounts we need from foods in a healthy diet. Most of the time, if a person does not have a documented vitamin deficiency, taking over the counter vitamins does not improve health at all. In fact, many well-designed studies involving hundreds of thousands of people have found no evidence that vitamins prevent heart disease, dementia, or cancers (1-3). Experts in medicine have called for patients to stop taking unnecessary vitamins (4). Thus, taking a vitamin to "make sure" you have enough in your system is not recommended. Complicating matters, there are studies indicating PD patients may be deficient of vitamins B6, B12, and D. And, there is marketing all around telling consumers to take vitamins. The best approach is, if indicated, for a qualified doctor to check a level and advise you on whether or not you should take a vitamin. If taking a vitamin for a specific medical reason, stick to the dose directed by the doctor. There are certain medical conditions that may call for a higher than normal dose of a vitamin. However, in general, taking a "more is better" approach can lead to serious health problems, especially when one takes megadoses (doses many times larger than usual or recommended). Some vitamins may actually be toxic in high doses commonly available in stores. Vitamin B6 toxicity can cause neuropathy or dermatitis. Acute vitamin D toxicity may cause confusion, excess urination, excess thirst, loss of appetite, vomiting, and muscle weakness. Chronically high levels of vitamin D may result in kidney stones, loss of bone mineralization, and pain. Vitamin E supplementation was found to be unhelpful for PD in the DATATOP trial of the 1990s, and is also known to be associated with health risks. A meta-analysis (a review of multiple studies) including nearly 136,000 people showed that use of vitamin E in doses at or above 400IU daily may increase all causes of death (5). The bottom line: take vitamins only if directed by a doctor familiar with the guidelines.

And vitamins are not the only supplements. The use of CoQ10 is widespread, though in a large, multi-center trial by the Parkinson Study Group, the supplement showed no clinical benefit (6). In other words, it did not help symptoms or improve scores on PD rating scales. CoQ10 also did not slow down disease, as had been hoped. Similar negative results have been shown in trials of CoQ10 for several other neurologic diseases.

Plants hold a vast potential for medicine. Plant-based supplements however, are not always effective, and some may interact with or block the effect of prescription medications. Some plant or herb supplements may cause harm. Nature is potent, and produces trees that can be used to make chemotherapy, bark that can

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be made into aspirin, and clover that is used to make the blood thinner warfarin, to name just a few. Doctors know that these products are potentially as dangerous as they are helpful. For example, the molecules used to make warfarin are also used as a poison found in pesticides. When used as a medicine warfarin has to be adjusted very carefully. The same care should be taken with any ingested compound, especially if there is the potential for danger. Simply designating a supplement safe because it comes from a plant is a dangerous and irrational position. For example, the supplement kava (extracted from the pepper plant *Piper methysticum*), may actually worsen PD symptoms, and if combined with the drug alprazolam, may result in a coma-like state (7). There are many other examples. Plants have had millions of years to evolve defenses against animals, some of which are very complex. Plants are potent for good and bad, and that is about as natural as you can get. This is one very important reason why it is important to make sure the compounds you take are safe.

Does the FDA protect us from harmful substances getting into supplements?

The FDA does not necessarily protect us from the potential dangers of supplements. By federal law, they are actually quite limited in this regard. To be clear, it was Congress which defined vitamins and other supplements as "dietary supplements" in 1994 (8), restricted the FDA, and created a law in which the supplement industry was to police itself. To quote the FDA (9):

"The U.S. Food and Drug Administration (FDA) does not have the authority to review dietary supplement products for safety and effectiveness before they are marketed. The manufacturers and distributors of dietary supplements are responsible for making sure their products are safe before they go to market."

Why is there a law preventing the FDA from regulating supplements?

In 1993, Dr. David Kessler, then commissioner of the FDA, attempted to regulate supplements. He was quoted in the June 15, 1993 edition of the New York Times to say, "The dietary supplement industry is pushing hard for deregulation of their products…There are no assurances that these products are appropriately manufactured, that what's on the label is actually in the bottle, that they bear adequate directions for use to insure safety or that basic safety data has been collected and reviewed" (10). This was in the face of numerous cases of injury or death resulting from the use of supplements. However, according to Dr. Kessler on the program Frontline, January 2016 (11), "What happened was the dietary supplement industry recognized that the standard that we set — significant scientific agreement — would require it, before it could make a claim, to have a scientific basis. And they just couldn't make any claim. And they saw, literally, billions of dollars at stake, and they unleashed a lobbying campaign that was second to none." This included a national television commercial (12) presenting the FDA in a fictitious middle-of-the-night, special ops-style raid on the home of actor Mel Gibson because he had a bottle of vitamin C. The commercial included dark lighting, ominous sound effects, and a voice over with Mel urging, "If you don't want to lose your vitamins, make the FDA stop. Call the U.S. Senate and tell them you want to take your vitamins in peace." The commercial ended with a written instruction to "Protect your

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right to use vitamins and other supplements. Write Congress Now." Several other actors spoke out publically, making similar straw man arguments, and Americans seemed to side with them, rather than the federal agency staffed with scientists and doctors, which had been established to protect them. The call to action by those with no medical or scientific training, along with organized efforts in health food stores around the country, resulted in massive letter writing. Per Frontline, Congress received over two million letters on this topic. In 1994, Congress passed the Dietary Supplements Health and Education Act (DSHEA), which limited then, and continues to limit now, the FDA as above. The key author was Senator Orin Hatch of Utah. According to author Dan Hurley in his 2006 book *Natural Causes* (13), supplements were the focus of Utah's third leading industry, Hatch owned over 35,000 shares of stock in a company that manufactured supplements, and Hatch's son was a lobbyist for the supplement industry. Hatch was further noted to be the recipient of large campaign contributions from the supplement industry dating back to the 1980s.

Is anyone evaluating supplements?

Federal laws restricting the FDA have not stopped independent investigators from analyzing supplements to see if they truly contain what their labels state. Multiple studies have shown outright substitution of herbal supplements in which the product contains something other than the stated substance (14,15). Other studies have demonstrated that traditional supplements manufactured overseas may contain contaminants including toxic heavy metals such as lead, mercury, and arsenic (16-22). The U.S. manufacture of supplements containing these toxins has also been demonstrated (23). Per the World Health Organization (24) "It is well known that there are many contaminants and residues that may cause harm to the consumers of herbal medicines." There are many reports in which prescription drugs have been added to supplements without reporting this on the label. Thus, it is of the utmost importance to make sure that the product one is using is safe. How that will happen is unclear. Experts call for reform and proper oversight of the supplement industry (25). Writers in lay press point to the lack of oversight and the politics behind the DSHEA law in less flattering terms (26, 27). Consumer Reports (CR) dedicated its September 16, 2016 issue to supplements and safety. The article "Supplements, a complete guide to safety," notes, "Pills and capsules make promises they can't keep in a marketplace with a profound lack of oversight." The article notes that since DSHEA became law, supplement products have grown in number from about 4,000 in 1994 to over 90,000 today. CR cites an industry currently generating $40 billion a year.

But this is a safe supplement, right?

Finally, many patients tell me that they believe some supplement is superior because a healthcare provider sold it to them directly in the office. The American Medical Association (AMA) Code of Physician Conduct (28) notes that the sale of health-related products by physicians raises ethical concerns about financial conflict of interest, undue pressure on the patient, erosion of trust, undermining of the primary obligation of physicians to serve the interests of their patients before their own, and such sales "demean the profession of medicine." The Maine Board of Licensure in Medicine also reflects this language.

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B6, friend or foe?

by Bill Stamey, M.D.

Pyridoxine, also known as vitamin B6, is a supplement that many people take, either by itself or in a multivitamin. It is available over the counter, no prescription required. Most of the patients taking B6 with whom I have spoken, however, have very little idea what B6 does, and other than a general recommendation they have been given at some point, are not very clear about whether they should take it at all.

It is true that B6 deficiency can lead to illness. It is also true that taking too much B6 can be dangerous. Here, I am going to briefly explain the basics of B6 and take apart the common issues PD patients may face with this vitamin.

B6 is classified as a coenzyme. Coenzymes are small molecules that assist enzymes in chemical reactions. There are thousands upon thousands of these reactions going on all the time in your body. B6 is involved in over 100 enzyme reactions with proteins, amino acids, carbohydrates, and lipids. B6 helps to maintain homocysteine levels in the blood; is a participant in immune function; and is involved the development of cognition through the formation of neurotransmitters - those chemicals like dopamine that allow one nerve to communicate with another. Deficiency of B6 is therefore associated with many different conditions such as anemia, dermatitis, cheilosis (scaling of lips, and cracks at the corners of the mouth), immune dysfunction, glossitis (swollen tongue), confusion, irritability, seizures, neuropathy, and depression. In short, it is important to have normal B6 levels.
We get B6 by eating a healthy diet. It is found in fruits, grains, fish, poultry, beef liver and other organ meats, potatoes, and some other starchy vegetables. Absorbing nutrients from foods is not always easy, however, and this leads some to think that they should supplement B6 and other vitamins to "make sure" they are getting enough. This should not be done blindly.

It is true that some vitamins and minerals are absorbed by a special process, which may take several steps in the body. A failure of any one of the multiple biochemical steps required may cause poor absorption of the vitamin, and thus a deficiency. This is the case with B12, for example, but it is not the case for B6. B6 is absorbed passively in the jejunum, a part of the small bowel. When a vitamin is absorbed passively, absorption is not an energy-requiring process, and when one has good health, there is usually no obstacle to B6 moving freely into the blood stream. It would therefore seem easy to maintain a normal level of B6 in the body.

However, a small percentage of people will have B6 deficiency for a variety of reasons, such as kidney disease; celiac disease, Crohn's disease, ulcerative colitis, and other malabsorption syndromes; autoimmune disease such as rheumatoid arthritis; alcohol dependence; and exposure to certain medications such as antiepileptic drugs. There is some data, also, that PD patients who take large amounts of levodopa may have low B6 levels (1, 2). Most of that data comes from European case reports of patients receiving continuous carbidopa/levodopa infusion via the Duodopa pump (not to be confused with Duopa in the United States), but it is also known that PD patients taking high doses of oral carbidopa/levodopa have a higher prevalence of chronic, sensory, axonal

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B6, continued…

polyneuropathy (3), in other words, nerve damage. It should be noted that some studies point to a deficiency of B12 in these patients, and apparently B6 was not always measured. There is mounting evidence for both. It is not entirely clear how a B6 deficiency is happening in these patients. One possible mechanism involves carbidopa, which is meant to block an enzyme in the body called dopamine decarboxylase, so that levodopa makes it to the brain. However, carbidopa also inhibits the action of B6. It may also be that absorption of B6 is somehow blocked by carbidopa/levodopa, or that downstream biochemical reactions deplete B6 and B12. According to the prescribing information of carbidopa/levodopa (Sinemet), B6 and carbidopa/levodopa may be given safely together (4). Though there is no formal guideline recommending testing, it has been suggested by some authors that it might be worthwhile to check one's blood level, especially if one is taking moderate to high doses of levodopa, or suffering from any of the above-mentioned disease states (1). Anecdotally, in my clinic I have diagnosed multiple PD patients taking carbidopa/levodopa with B6 deficiencies, where no other clear cause is evident.

The good news is that measuring a B6 level is done with a simple blood test, and replacement may be given with over-the-counter tablets. However, as with any supplement or medication, care should be taken in replacing B6, and to be clear, I re-emphasize that I am not recommending anyone take a B6 supplement without knowing one's own blood level first. Ideally, a qualified physician should interpret the test and tell you whether you need B6 supplementation, and if so, how much you should take. The United States Recommended Daily Allowance (USRDA) daily dose for men over 50 is 1.7mg, and for women of the same age 1.5mg (5). In most individuals, this amount is readily obtainable with consumption of a healthy diet. Higher doses are sometimes recommended for short periods, for specific conditions. You should know that many over the counter vitamin supplements carry amounts of B6 that are far higher than this, sometimes into or over the 500% or "megadose" range. The Food Intake Board of the Institute of Medicine has established that for men or women over 50, the tolerable daily upper intake level of B6 is 100 mg (6), a dose commonly found in grocery stores. B6 at this dose is 59 times higher than USRDA for women and 67 times higher than USRDA for men.

Overdosing B6 can be dangerous for several reasons. Merck Pharmaceuticals has stated that B6 in doses of 10 mg to 25 mg may actually reverse the effects of levodopa by increasing the rate of the enzyme activity that carbidopa is meant to inhibit (4). In other words, levodopa is depleted in the body before it can get to the brain, where it is needed to work against PD. There is no mention of the effect on the same enzyme when B6 is given at 100, 500, even 1000mg (all doses which are available in some health food, drug, and grocery stores), though one would suspect it is even more potent in driving down levodopa, and thus worsening the symptoms of PD. In addition, much like the case when B6 levels in the body are too low, excess B6 is well documented to cause neuropathy, or nerve damage, as well as a disfiguring skin condition (7, 8,9). The Weill Cornell Neuropathy Center evaluated all new neuropathy consultations from July 2014 until June 2015 and found that 7% had elevated B6 levels; whereas only 1.5% combined had either B6, or B12 deficiencies (10). Among the total group studied abnormal levels of nutritional factors were implicated in 24%. Likewise, the

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B6, continued…

Peripheral Neuropathy Center at Columbia University evaluated patients over a 10 year period ending in 2012 (11). These were new referrals with an existing diagnosis of idiopathic neuropathy, meaning another physician, typically a neurologist, had not yet determined the cause of the neuropathy. Among these patients, B6 toxicity accounted for 2.5%; whereas B6 deficiency was 0.3%, and B12 deficiency 1.4%.

In summary, some PD patients may have a high or low B6 level, and either may be harmful. There is some concern that carbidopa/levodopa may indirectly drive B6 levels down. B6 supplements may contain doses that are far too strong for daily use, and toxicity has been linked to neuropathy, and possible negative impact on PD symptoms. If you have concerns about your B6 level, have a qualified doctor check your level and advise you about how and whether to take B6.

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A brief history of the PD non profit groups in Maine

by Bill Stamey, M.D.

In the summer issue we ran a story about the New England Parkinson's Ride, an annual event which donates proceeds to the Michael J. Fox Foundation. This issue I will focus on two other groups which have raised funding for either national or local PD efforts.

The Maine Parkinson Society (MEPS) is currently headquartered in Bangor and is a 501(c)(3) charitable organization with a mission "to improve the quality of life for an estimated 7,000 Maine people affected by Parkinson's disease (PD). This is achieved through educational programs, by providing respite care services to health care workers and caregivers who work with people with PD, and by funding other programs that work to find the cure."

MEPS has worked to spread information since its beginning. The Secretary of State accepted the group's articles of incorporation in July 1998. Per the Lewiston Sun Journal, at the time, PD was known as the "disease of the invisible," and interviewees noted that funding for PD research ranked 32nd, "just ahead of sleep disorders." They stated that amount of money was equivalent to only six percent of the total directed to AIDS research. The article reported, "The Maine Parkinson Society is intended to provide greater visibility and to improve service to victims of the disease."

At that time, MEPS was based in Portland and the original president of the group was Gregory Leeman, a human resource director of Blue Rock Industries, who was quoted as saying there were then no movement disorder neurologists in Maine. He despaired as a PD sufferer, "the variety of drugs used for treatment adds to the confusion It is difficult, even when you keep daily charts as most of us do, to find a smooth drug regimen. It all adds up to a roller coaster ride that leaves patients in an emotionally drained state."

Vice president of that first board was Karen Bardo of Alna, who worked as an administrative secretary in special education. She had been diagnosed just two years prior and was reported to be the glue that had kept the effort to form MEPS going. Ms. Bardo kept up that enthusiasm for several years, and would later serve as MEPS president. Other initial members included treasurer Leonard Kaminow, M.D. (then and now in the practice of neurology in Scarborough), and secretary Harold Jones of Augusta. Of note, Mr. Jones, the father of Karen Bardo, would also go on to become president of MEPS for a time, and passed away due to complications of heart disease in December 2008. A touching portrait of his life was published in the Kennebec Journal, and in the winter 2009 edition of the Maine Parkinson's Pages.

MEPS hosted many activities over the years to raise both awareness of PD and funds for the mission of the group. Often, MEPS worked in collaboration with the already-established Maine Chapter of the American Parkinson Disease Association (APDA), which had been a long-time fundraiser and helped to organize support groups throughout the state.

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At first, however, it was not clear the two groups would work together. The Maine APDA president for 25 years was Carl Barker, who retired from that role in 2009. Barker, interviewed by phone, looks back on those days and notes, "at first I didn't see the need for the two programs, and I thought that they ought to join us. But, when it became clear that they wanted their money to remain in the state, to help people with respite and other projects here, we worked together." Barker notes "people who wanted to give money for research were encouraged to give to the APDA as MEPS had no channel for that."

That they did work together seems evident. Each organization hosted a variety of their own fundraisers such as bowl-a-thons, walk-a-thons, auctions, and the like. For several years, they also collaborated on events such as the seminar for Parkinson Disease Awareness Day each April. In 2000, for example, the Bangor Daily News reported on the seminar, which was held in Augusta. Speakers included general neurologist Dr. Bernie Vigna of Bath (who passed away last year), and Dartmouth-Hitchcock neurosurgeon, Dr. David Robert, who covered surgical options including deep brain stimulation. Quotes by Parkinson disease sufferer Viola Macomber were moving. She had expressed uneasiness about attending but apparently found the meeting very helpful when she stated, "This is incredible! He's describing me right down to my fingers locking, that I've blamed on arthritis."

That Spring, the Lewiston Sun Journal reported another collaboration: the APDA and MEPS were "working together to bring a full time PD specialist to Maine so that patients will no longer have to drive to Boston for treatment." To attract a movement disorders doctor, the groups applied for grant money to set up an information and referral center. In July 2001, the Bangor Daily News announced the opening of the American Parkinson Disease Association Information and Referral Center (IRC) at Maine Health's Maine Medical Center facility in Scarborough. Lillian Scenna, LSW, would become program manager that first year, and the IRC would eventually move to Bucknam Road in Falmouth for the duration of its existence. The office, primarily through the efforts of Lillian Scenna, would provide information about PD to the general public; coordinate educational classes; cultivate support groups; publish the Parkinson's Pages newsletter; maintain a lending library of books; provide assistance using the internet to research health-related questions; maintain a specialized list of community resources for people with PD; manage the administration of the Respite Care Reimbursement Program; and many other outreach efforts to increase awareness and proper care for Maine people with PD. The IRC also fielded a lot of phone calls. Lillian Scenna estimated she took over a thousand calls per year from PD patients, and the top three questions were related to provider referrals, support group referrals, and medication side effects. She was a member of the boards of MEPS and APDA, and was active in the planning and execution of most PD related events in Maine.

MEPS and APDA also found success in 2002, when Ed Drasby, D.O., brought the specialty of movement disorders to Maine His practice evolved over time to include wellness in PD and participation in multiple clinical trials as a Parkinson Study Group site. Studies included, for example,

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a trial of the drug pimavanserin, which would go on to become FDA-approved for psychosis in PD April 2016. Dr. Drasby has been on the MEPS board of directors since coming to Maine, and was a board member of the Maine chapter of APDA. He was instrumental in building a PD community in Maine and, as an aside, very welcoming and helpful in getting me here. From the beginning, he asked me to be a part of these organizations. His help and introductions were much appreciated. Dr. Drasby retired from practice in October 2014, and though there are other movement disorders doctors in the State, I frequently hear from patients that he is missed. I suppose patients worry that I will feel diminished after such praises, as they are usually followed by something sheepish along the lines of, "no offense." Rest assured, none taken.

Regrettably, due to cessation of funding in December 2013, the Maine APDA chapter ended and the IRC in Falmouth closed, taking with it the role of Lillian Scenna. These days, she is working with people with disabilities, helping them find jobs in her role as social worker at the Pine Tree Society. Though she is not directly involved in Parkinson disease, she notes her services might be helpful for people with young onset PD. "I still remember those calls," she says.

MEPS is still going, with a full board presided over by long-time member Gary Cole. This April a fundraiser was an event was called STRIDE - WALK - JOG - RUN for Parkinson Disease, which took place at the indoor track of the Boothbay Harbor Region YMCA. Participants exercised any distance they chose, and endurance athlete Hunter De Garmo completed a 50K run. According to the Boothbay Register, Hunter's mother has PD.

For more information about how to help MEPS, please visit their website at http://www.maineparkinsonsociety.org/ or on Facebook.

Parting thoughts

I want to extend another thanks to all those who contributed in articles, interviews, or insights. Also, I would like to thank readers. I have again heard from many of you personally, and it seems like this is a good resource. As I am still new to building and working with websites, please forgive any clumsiness.

Visit us online at www.mainepdnews.org to see upcoming events. And, if you know of a PD event in Maine, please let me know.

Be well, and stay out of trouble,

Bill Stamey, M.D.