Connecticut Parkinson's Working Group Newsletter April 2004

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Support for this newsletter comes from **Pfizer Corporation** and the donors to **CPWG**.
Editor: Stan Wertheimer stan.wertheimer@gmail.com **Design: Alyssa Wertheimer**
Interview Editor: Jeff Lincoln jeffrey.lincoln at yale.edu
Distribution: Pam Lincoln, Pat Gerace

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PD Research A discovery beneficial those with PD Judy Monchuk, CP

January 30, 2004(The London Free Press) - CALGARY -- University of Calgary neuroscientists have discovered how the brain tells the body to move, research that could give new hope to people with PD. The researchers say they have greater understanding of how the brain processes sensory "cues" that prepare the body for action, which may lead to new or different treatment for the degenerative nerve disorder. "The next step is to develop new drugs or therapeutic stimulation, and to modulate these cueing cells to trigger motor response," says Dr. Bin Hu of the university's faculty of medicine.

Hu, scientific director of the Movement Disorder and Therapeutic Brain Stimulation Program, has spent the past decade studying the thalamus, a part of the brain where signals for hearing, vision, touch and movement converge. Neurons in the thalamus decide what part of the brain gets signals and when, exerting tremendous influence on how people think, memorize and react to the outside world.

The research team found that cue cells take in sensory information and cognitive signals from the cortex. When those signals collide, they create a burst that alerts the body for action. The team believes the cue cells are controlled by a chemical messenger called acetylcholine, which can synchronize the collision of signals. The discovery may explain why PD patients have considerable difficulty controlling their movements, yet are able to move smoothly when stimulated by strobe lights or music, says Hu.

The information was published Jan 6 in Proceedings of the National Academy of Science. There are no immediate plans to test Hu's theory on human PD patients. "The more important thing at this stage is that we have to have a thorough understanding of this cueing system, then we can revisit the current PD model," Hu said.

[*It is a bit of serendipity that this very topic was the focus of a long discussion at our January meeting, precipitated by Mike O'Brien's comments. Clearly the research has a long way to go, but this illustrates the important point that there seems to be no end to the ways we can affect the progression of PD and its symptoms.* **Stan**]

Magnetic Stimulation for PD

January 30, 2004- BETHESDA, Md. (Ivanhoe Newswire) -- In the United States, more than one million Americans have PD. It is a neurological condition that can cause slowness and instability, but a new type of therapy can improve both of these problems. Larry Criner not only takes nature walks for pleasure; he's made a life out of capturing nature on film. "For me, it's a place where I can come and find quietude," he tells Ivanhoe. For nearly 15 years, Criner has dealt with PD. He says, "It affects me. It really does. I mean this is a disease of the brain, of the mind, and it plays very big games on your head."

Criner recently joined a new study with neurologist Mikhail Lomarev, Ph.D., M.D. He's testing TMS. "We think that TMS (transcranial magnetic stimulation) makes patients' brains more sensitive to the medication he or she is already taking," says Dr. Lomarev, of

National Institutes of Health in Bethesda, Md. A pulse of induced electrical current targets areas of the brain that control movement. Dr. Lomarev says: "Our goal is to improve stiffness in patients. To make them move faster than they did it before, without magnetic stimulation."

They have achieved that, but results have been short-lived. Criner doesn't know whether this will help him long- term. Until then, he's optimistic and will continue to do what he can. This therapy has been tested and used in depressed patients for more than a decade. Doctors say the study with PD patients will likely continue for several years.

{As the title suggests, this is the second of two articles by Steve. He has candidly and clearly given us an insight into what has happened to his life since the implantation. This is surely one of the best articles we have ever presented. Stan}

Deep Brain Stimulation – Part 2

Steve Holahan

In Part 1 of my article on my deep brain stimulation (DBS) surgery I covered the facts and mechanics of what happened to me. In this second part I want to cover the tougher topic of the life impacts DBS is having on me. The physical changes are dramatic as those of you who have seen me at out meetings can attest! Gone are the drug-induced dyskenesias, the tremor, the rigidity and freezing, and the on-offs. I have not fallen once since the DBS systems were turned on, before 2-3 falls per day were not uncommon. Before I had practically given up driving except very short distances, now I drive all the time. I even drove 11 hours straight this past summer going to visit my son in Michigan. I can sleep 6-8 hrs through the night, before I rarely slept more than 2 hours at a time. My walking gait is still not fluid and both my back and legs get tired if I walk too much, but before when I was off, a walk of even few feet was a feat. Eating is no longer a shaky-handed chore and I have gained needed weight back. My body's thermostat seems to work much better now. Before DBS I was very sensitive to the cold, and in the heat I would sweat uncontrollably at the slightest exertion.

People often ask about the care I have to take of the equipment itself or any discomfort it may cause. I have had no real discomfort from my DBS and Medtronic who makes the hardware gives a very complete book of dos and don'ts. The biggest problem is magnetic fields accidentally turning a stimulator off. I did this once using an electric hand tool too close to my chest. Otherwise any non-contact (you would not want to be struck in the chest where the stimulators are implanted) activity including swimming is fine. My ping-pong game is coming back after a long dormancy!

On the drug front, I continue to take the same amount of Tasmar (100mg 3/day), amantadine (100mg 2/day), and Mirapex (0.25mg 6/day) as I did before surgery. But the carbo/levodopa (Sinemet) has been cut to less than half of my pre-surgery levels, including completely eliminating the CR or extended release form. Lastly without the daily plunge into offs, I no longer needed the trial "escape" agonist, apomorphine. A nice side effect of all this drug reduction has been a return to a near normal digestive cycle.

Before moving on to the emotional changes since surgery, I want to say a few things about the surgery itself. The first surgery or brain implantation is the tough one. You must be fully conscious and off all medicines for the duration. It is a long tiring and uncomfortable day but not painful in any way. There seemed to be long periods of waiting between steps and in retrospect I wish I had been exposed to meditation as a way to cope. I would urge anyone else in the group who goes for DBS surgery to spend some time talking to Gunilla Norris, who leads the meditation sessions before the CPWG meetings.

Well, I am finally to the part that is, for me, the hardest to come to grips with, the emotional impact of the DBS surgery. I know that it sounds incredible that I would have to come to grips with what appears to be a medical miracle. So to be clear on this point I am not questioning my decision to have DBS or its results. Why is then that I am still not

comfortable with this "miracle"? Why is my mind still stuck, while my body is free of the worst of PD's impacts?

I think that the answers lie partly in the emotional effects of PD, but also in my personality and reaction to living with this chronic incurable illness for the past 22 years. I have always been a keep-busy, task and goal oriented person so coping with PD had always been tough. It took me a long time to learn to take life one day at a time. Now suddenly I can again live for tomorrow and next week, and even next year. But I still have PD and in many ways still think like I did pre-DBS. But I tend to forget that my pre-DBS persona included 35 years of life without any thought of PD. So in some respects in the past year I have been rewinding the emotional tape of my 21 pre-DBS years with PD. But this time at the end of the tape it is not a young man without PD, but rather a 57-year-old man who still has PD. The effect of this rapid change is that I often feel like a retired high wire walker, suspended above a swirling abyss of PD's worst symptoms. I'm darn glad I'm not down there but not sure I remember all of the old tricks well enough yet to fully relax and enjoy life up in the light and fresh air.

The key word is balance and it is balance of movement, of muscle tension and flexibility. and of emotion that PD upsets. DBS has restored a great deal of physical balance to my life. The mental and emotional balance has been a struggle for me, in part because I just didn't prepare for it. Having said that I am not sure how I would have prepared. Like many of life's journeys this may just have required me to put on the boots and make that hike. It is not over yet but I do sense more balance and control in my life. A life made much more livable because of DBS therapy and which, I hope, will prove how wrong the French playwright, Moliere was when he said, "Nearly all men die of their remedies, and not of their illnesses."

(Author's Note: As of this writing I am aware of some medical reports of cases of aggravated mood and personality changes in a very few post DBS patients. I suggest checking with a medical expert for more exact information.)

Who Do You Believe?

Stan Wertheimer

Recently Lou Stevens sent me, as well as several other people (including trusted medical doctors), two copies of a newsletter called "Second Opinion", whose sole contributor seems to be a doctor Robert Jay Rowen. In particular my attention was directed to the lead articles in both newsletters.

In the first article, "Monster Epidemic!" Rowen describes what he considers a cause of myriad diseases, including PD, ALS, MS, and Alzheimers - Lyme Disease. In the second "The Incredible Healing Action of One Simple Herb!" he names ***cat's claw*** as the wonder herb. He also tells of a foolproof, quick test for Lyme Disease. Conclusion: Anybody with one of the diseases he mentions as probably caused by Lyme Disease should get the foolproof test and adhere to the incredible healing of cat's claw.

I also know of PWP following other regimens which are said to be palliative or even curative. One is Forceless Spontaneous Release (FSR) which is practiced by a woman in California; she shares her ideas, methods and suggestions with anyone who is interested, mainly by means of several well written, if a bit wordy, manuals and position papers which make sense, unfortunately at least to me, only up to a point. She maintains after five years you will experience a cure to PD while gradually cutting your doses of medication. The central treatment is manipulation of the feet. She says to expect things to worsen as you diminish medication, but to have faith.

Another regimen is a combination of carefully chosen supplements and intravenous injections of glutathione several times a week, conducted by a medical doctor in Florida.

He claims to reduce symptoms significantly and has a clinic devoted to the practice of his treatment.

There is a chiropractor in Boston who manipulates only ones cervical vertebrae twice a week and claims relief from many PD symptoms. He makes lots of sense. There are also proponents of treating the brain with electromagnetism (see following article).

Then there are thousands of medical doctors throughout the world who say that they have a treatment which comprises carefully selected doses of chemicals on a daily basis. They can almost guarantee success for a while, with the chance that you will develop uncontrolled movements after five or six years, or other nasty side effects. In addition they have surgical treatments which require cutting into the brain and may result in death, but often alleviate troublesome symptoms, at least for a while. They can tell you why they think their regimen works, but not what the cause of PD is.

Most of us adhere to the last regimen. Some of us have tried the others, and ones not mentioned here. The practitioners of all of the above are usually passionate, well meaning people, except a few of those in the last group. The question that I have is: ***Who do you believe?***

The answer you come up with will surely affect your life greatly and most likely that of others who love and care for you. Therefore how you answer it is crucial. There are many ways to go about seeking an answer, including acceptance of the western medicine solution without question because that is the system you have always adhered to and most of the time it works pretty well. My guess is that this is the route a majority of us take.

After thinking about this for a while I came to the conclusion that I was asking the wrong question. In my life as a mathematician I found this to be a common happening and was often the one who made the diagnosis. I then sat down with the person who was seeking my mathematical advice and we would hash out what the question, or questions really were. Once we did that the solution was often immediate, required that they do some extra work, or did require that I get involved. I often got profuse thanks for doing nothing more that helping to determine the question they meant to ask – without doing any mathematics at all! I see now that I have become the client, asking the wrong question.

I know many PWP who believe many of the purveyors of solutions to their problems. I respect these people and can find no fault with the answers they have chosen. So the original question must be the wrong one to ask. Each of us would come up with a different set of "most important" questions. Let me tell you mine:

As I get to understand a treatment, which of its protocols am I comfortable with?
What do I want a protocol to do for me?
Under what conditions do I stop following a protocol?
Is the effort in following a protocol worth the possible benefit?
Are my choices consistent with each other?

Thus I don't have to consider the question of trust, except as it relates to trusting myself. I can select, as in a buffet, from any treatment I wish as long as the questions above are answered satisfactorily. For example, I might find that I benefit from having my feet massaged, and I feel better when my spine is straightened, and I have found my symptoms worsen more slowly when I take anti-oxidants, and sinemet in minimum doses helps me greatly in moving around, and tai chi improves my balance. So I select all of the above because they are consistent and worth my effort.

This is not a great revelation – I think most of us do something very close to this. There are, however, some of us who depend almost exclusively on only one regimen to the exclusion of others, either because the purveyor says that is what is required, or we impose that restriction ourselves. In the case of a disease whose mechanisms are not known this can be a costly path to follow. If one depends only on drugs as prescribed by a single western doctor for a complete treatment, or only cervical vertebrae manipulation, or only

glutathione you might be like a person at a feast who eats only the bread while neglecting the other foods on the table.

I have come to the conclusion that I want the opinion of more than one MD, it is a good idea to investigate reasonable food supplements to take, exercisei is a real benefit even if I can only do a little, the services of a good physical therapist when needed can be a great help, and drugs can improve my quality of life if chosen carefully and in consultation with a caring MD.

I urge each of us to sample from the whole range of protocols available and to consider what questions we want to answer.

[*This next article is included for its obvious relationship to PD. We know that PD is often misdiagnosed. FXTAS may be yet another candidate for what is diagnosed as PD. If we can divide the "diagnosed PWP" into groups that have closely related but different ailments we might eventually wind up with a much more cohesive group of actual PWP; this may help figure out what causes it. Also, why only men??* **Stan**]

Genetic Screening Recommended To Detect New Neurodegenerative Disorder In Men Over Age 50

Common and small mutation in the fragile X ***gene, once thought to have no health effects in male carriers, now linked to tremors, balance problems and dementia.***

SACRAMENTO, Calif. 27-Jan-2004 - A team of researchers, led by physicians at the UC Davis M.I.N.D. Institute (UCDMIND), have discovered a new, progressive neurodegenerative disorder that predominantly affects men over age 50 and results in tremors, balance problems and dementia that become increasingly more severe with age. M.I.N.D. stands for Medical Investigation of Neurodevelopmental Disorders.

A significant but currently unknown number of adults with these tremor and balance problems are being diagnosed with normal aging, PD, senile dementia and Alzheimer's disease when their condition may be accurately and easily identified with a standard DNA blood test ordered by their doctor. The discovery is published in the Jan. 28 issue of the Journal of the American Medical Association. Known as *fragile X* -associated tremor/ataxia syndrome, or FXTAS (pronounced fax-tass), the disorder affects older men who are carriers of a small mutation (premutation) in the same gene that causes fragile X syndrome, the most common cause of inherited mental retardation. Nearly 1 in 800 men in the general population carries this premutation in the fragile X gene, and UC Davis research suggests that as many as 30 percent of carriers -- roughly 1 in 3,000 men -- may develop FXTAS later in life.

"FXTAS may be one of the most common causes of tremor and balance problems in the adult population, yet it is being misdiagnosed because neurologists who see adults with movement disorders are not aware that they need to look for a family history of fragile X in grandchildren or to check for the presence of the premutation in the fragile X gene," said Randi Hagerman, medical director of the UCDMIND.

Screening for the gene mutation in men who have tremor and balance problems is important regardless of their family history, especially when accompanied by other signs such as parkinsonism (rigidity in movement), short-term memory loss and dementia. Family genetic counseling can help those affected with FXTAS, as well as future generations that may inherit fragile X syndrome. Research studies also are under way to specifically determine which medications are best suited to alleviate FXTAS-related problems, and whether other therapies, such as surgery to disable nerve tracks, may actually exacerbate balance problems.

Hagerman, a developmental and behavioral pediatrician who has specialized in the diagnosis, research and treatment of fragile X for more than 20 years, began looking for a

connection between children and their grandfathers because the mothers of her fragile X patients were worried about their own fathers, who were falling down, becoming forgetful and experiencing other neurological problems. Hagerman, along with her husband, Paul, a professor of biological chemistry at UC Davis School of Medicine, led the team of researchers from UCDMIND, University of Colorado Health Sciences Center, and RUSH-Presbyterian-St. Luke's Medical Center in the JAMA study.

The researchers looked at 192 individuals whose families belong to the Northern or Southern California Fragile X Associations or who were family members of patients seen at the UCDMIND. While only 17 percent of the men in their 50s had FXTAS, the percentage of individuals with tremors and balance problems increased with each decade of life, to 38 percent of men in their 60s, 47 percent of men in their 70s, and 75 percent of men in their 80s. The study also showed that the majority of older males carriers of the premutation will develop at least mild symptoms of FXTAS.

FXTAS is characterized by tremors, balance problems and dementia that become increasingly more severe with age. Initial signs of the disorder may include difficulty writing, using eating utensils, pouring water and walking. These initial symptoms progress over years or even decades, until carrying out many of the tasks of daily living and walking without assistance becomes difficult or impossible. Other features include short-term memory loss, anxiety, decreased sensation in the lower extremities to touch and vibration, lower-limb muscle weakness and parkinsonism.

"FXTAS is an enigma," said Hagerman, who also holds the Tsakopoulos-Vismara Endowed Chair in Pediatrics at the UC Davis School of Medicine and Medical Center. "The disorder appears later in life in men who are generally healthy throughout childhood and early-to-mid-adulthood and have normal to above-average intelligence, yet is caused by a defect in a gene known to cause mental retardation usually diagnosed in early childhood."

The underlying cause of FXTAS is a change, or mutation, in the fragile X mental retardation 1 gene, or FMR1. Under normal conditions, this gene produces a protein that maintains the proper functioning of nerve cells in the brain. The gene causes both fragile X syndrome and FXTAS when a particular segment of DNA is repeated too many times. The repetition informally is called a "CGG repeat" because it contains the same trio of DNA building blocks -- cytosine, guanine, and guanine in the same repetitive order.

The average person has 30 CGG repeats in the FMR1 gene. When an individual has 200 or more CGG repeats in the FMR1 gene, the individual makes little or no FMR1 protein and has fragile X syndrome. With 55 to 200 CGG repeats, an individual is considered a carrier of the premutation, which can lead to FXTAS later in life and to fragile X (the full mutation) in the next generations. Male carriers are at high risk to develop FXTAS, as well as for passing on the gene mutation to all of their daughters, who in turn are at risk to have children with fragile X syndrome.

Tissue and postmortem studies of brains from FXTAS patients, led by Paul Hagerman and UC Davis assistant professor of pathology Claudia Greco, showed accumulations of abnormal cellular material in the form of inclusion bodies in the nuclei of brain cells, (specifically neurons and astrocytes) throughout the cortex and brainstem regions. The greatest densities were found in the hippocampus and frontal cortical regions, areas of the brain that control movement and are important in learning, memory and emotion.

"The formation of inclusion bodies in the nuclei of nerve cells offers an important clue about the cause of the disorder, one that may ultimately help with the development of therapies for both FXTAS and fragile X syndrome," said Paul Hagerman. In 2001 the Hagerman team reported the first cases of FXTAS in men and suggested that the neurological dysfunctions could be due to the elevated levels of messenger RNA from the FMR1 gene mutation, which are consistently observed in the blood of premutation carriers.

As a result, they proposed the hypothesis that FXTAS results from an RNA toxic gain-of-function.

"Further study of these cellular processes can lead to a better understanding of the mechanisms leading to fragile X syndrome and may offer new targets for developing treatments," Paul Hagerman said. In addition, because some carriers of the premutation develop FXTAS while others appear protected, other factors also may play a role in disease development, offering additional clues to the origin of this disorder.

Classification of PD - PD and Parkinsonism

PD is named for Dr. James Parkinson, who in 1817 first described the features of this illness. Features of PD include tremor, slow movement (bradykinesia), and rigid muscles (rigidity). The syndrome of parkinsonism refers to people who have these features but do not have true PD.

Other conditions and diseases that cause parkinsonism may also cause symptoms that are not seen with PD. These conditions may be treated differently than PD. Unlike PD, many conditions that cause parkinsonism are reversible.

• PD-plus syndromes are a group of disorders characterized by the degeneration of nerve cells in different parts of the brain. They include progressive supranuclear palsy (PSP), Huntington's disease, and multiple system atrophy, among others. PD-plus syndromes have parkinsonian features as well as features not associated with PD. These syndromes usually do not respond to levodopa or dopamine agonists.

• Secondary or symptomatic parkinsonism describes the syndrome of parkinsonism when it occurs as the result of an identifiable cause. For example, certain medications, brain tumors, strokes, infections (such as encephalitis), and toxins (such as carbon monoxide or manganese) can cause secondary parkinsonism.

Stages of PD

It may be helpful for people with PD and their families to be familiar with some of the ways the disease is described. Experts describe symptoms and stages of the disease differently.

PD sometimes is described as early, moderate, or advanced.[1]

• **Early** disease describes the stage when a person has a mild tremor or stiffness but is able to continue work or other normal daily activities. This often refers to a person who has been newly diagnosed with PD.

• **Moderate** disease describes the stage when a person begins to experience limited movement. A person with moderate PD may have a mild to moderate tremor with slow movement.

• **Advanced** disease describes the stage when a person is significantly limited in his or her activity, despite treatment. Daily changes in symptoms, medication side effects that limit treatment, and loss of independence in activities of daily living are common. A person with advanced PD may have significant changes in posture and movement, speech problems, and frequent changes in movement.

PD may also be described by five stages:

• **Stage I (mild or early disease)**: Symptoms affect only one side of the body.

• **Stage II**: Both sides of the body are affected, but posture remains normal.

• **Stage III (moderate disease)**: Both sides of the body are affected, and there is mild imbalance during standing or walking. However, the person remains independent.

• **Stage IV (advanced disease)**: Both sides of the body are affected, and there is disabling instability while standing or walking. The person in this stage requires substantial help.

• **Stage V**: Severe, fully developed PD is present. The person is restricted to bed or chair.

Medical professionals may refer to this scale when discussing the disease and decisions about treatment.

Comments on the Next Meeting - May 15th

The speaker will be Whitney M. Lewendon, Esq. from the law firm of Coan, Lewenden, Gulliver & Miltenberger, in New Haven; he will speak at 10:00 on "Planning Ahead: Legal Issues When Faced With Chronic Illness." Attorney Lewenden ha extensive experience in this aspect of the law.

After Atty. Lewendon, Terry Deshefy-Longhi will give us a brief introduction to a fascinating topic that has arisen in her doctoral research - facial expression differences in the PWP when on and when off.

Don't forget that Gunilla Norris at 9:15 when she conducts a meditation session.

A reminder: Skip Komisar will be looking for comments and questions on the website he is designing for us. The current address (test version) is

http://198.63.55.180/cpwg2000/

If you have access to the Internet, please participate by going to the site and trying out the options. Send Skip your comments.

Putting A Patch On Parkinson's Disease

Mary Ellen Egan (Forbes), 04.23.04

NEW YORK - Mornings were the worst part of the day for Michelle Lane. In June 2000, the 41-year-old mother of three was diagnosed with PD, a fatal [?? – Stan], progressive disease that attacks the central nervous system. As the disease progressed, Lane lost the ability to button her shirts or brush her daughter's hair. Even more troubling were the mornings Lane would awaken paralyzed and have to wait up to an hour before her medication kicked in so she could get out of bed.

But a new drug has changed the way Lane approaches her day. The drug, called rotigotine, works by tricking certain receptor cells into producing dopamine, a neurotransmitter believed to be lacking in PD patients. This type of drug is called a dopamine agonist. The three most prescribed versions on the market are **Eli Lilly**'s Permax, **Pfizer**'s (Pharmacia) Mirapex and **GlaxoSmithKline**'s Requip. But rotigotine has a distinct advantage: It's the first dopamine agonist to be delivered via a patch, thereby allowing patients to have a consistent dose of the drug in their systems at all times.

Over 1 million Americans suffer from PD (PD), and they spend over $2 billion on medications each year, including $400 million to $500 million for dopamine agonists. Patients are diagnosed by four primary symptoms: tremor, rigidity, postural imbalance and the slowing or freezing of voluntary movement.

For over 30 years, levodopa, the chemical precursor of dopamine, has been the primary medication for PD patients. It is still the most efficacious drug, but long-term use can result in dyskinesia (jerky, involuntary movements) and levodopa can lose its beneficial effects over time. Since dopamine agonists have lesser side effects, they are currently the first line of defense against PD.

Two years after Lane was diagnosed, her doctor put her on Mirapex. Lane says she stayed on the drug for three months because it made her "extremely sleepy." At that time, her doctor, Jayaraman Rao, told Lane about a clinical trial for a new PD drug. In May 2002, she enrolled in the rotigotine trial.

The drug was developed by Aderis Pharmaceuticals, a privately held biotech based in Hopkinton, Mass., and Schwarz Pharma, the North American affiliate of Germany-based Schwarz Pharma AG. The two companies joined forces in August 1998 to finish the work on the agonist molecule that had begun in the late 1980s. Their goal was to have rotigotine delivered via a patch, since one of the biggest problems plaguing PD patients is keeping the right amount of medication in their systems throughout the day. Too much medication can cause dyskinesia, and too little can result in paralysis.

"Some patients set the alarm clock for 3:00 A.M. or so, and then take a pill so they can get out of bed in the morning. With the patch, patients can now sleep through the night," says Aderis Chief Executive Peter Savas.

Rotigotine comes in four patch sizes and dosages. Lane, whose clinical trial ended in November 2002, opted to stay on the medication and now wears two patches a day. "Mornings are now the best part of my day. I can wake up, clean the house, and get my kids off to school on my own again," she says.

Schwarz is currently compiling the data from three separate rotigotine trials--two on early stage patients and one on late-stage patients--and the company expects to file for U.S. Food and Drug Administration approval by this fall. If all goes well, the PD patch could be on the market by 2006.

Connecticut PD Working Group
132 Highwoods Drive
Guilford CT 06437

DISCLAIMER: Articles in this newsletter are for information only. Any questions of treatment should be discussed with your physician.

WRITE! your representatives in congress.

Christopher Dodd
Russell Senate Office Building
Washington, D.C. 20510

Joseph Lieberman
Hart Senate Office Building
Washington, D.C. 20510