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Alzheimer's and Parkinson's sufferers receive boost from Israeli research

Susan Goodman

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At the Hebrew University, cutting-edge research has already given some sufferers from Alzheimer's and Parkinson's diseases medical interventions to help control and alleviate the symptoms of these diseases. Now ground-breaking research is pointing the way to new understanding and novel therapies.

The brains of patients who suffer from advanced Alzheimer's disease are wasted and shrunken. Under the microscope it becomes clear that large numbers of neurons die and are coated with a protein-like substance known as amyloid plaques. Within the cells a tangle of proteins can be seen.

One approach is to investigate these tangles and plaques. But Professor Marta Weinstock-Rosin, incumbent of the Dr. Leon and Dr. Mina Deutsch Chair in Psychopharmacology at the HU School of Pharmacy, sees the problem differently. "If we are going to produce a drug that can impact and slow the progress of Alzheimer's then we must be able to replicate its earliest stages," she says. Careful experiments using an animal model that mimics the early symptoms of Alzheimer's provide her with the testing ground for a new generation of drugs to treat the disease.

Weinstock-Rosin certainly has the credentials for success. It was her research over 20 years ago that produced RA7, now sold worldwide under the trade name Exelon - one of the first Alzheimer's medications. Unique among drugs used to treat

Alzheimer's, Exelon targets only specific parts of the brain and doesn't have a deleterious effect on other parts of the body.

In June 2006 Exelon received further FDA approval for treating mild-to-moderate dementia associated with Parkinson's disease. Although Exelon can do much to improve the quality of life for about one-third of all Alzheimer's patients by slowing the progress of the disease and improving memory skills, it remains effective for only a few years.

Weinstock-Rosin's latest research focuses on a novel drug called ladostigil and which combines Exelon with Rasagiline, a Parkinson's disease treatment developed by Professor Moussa Youdim of the Technion-Israel Institute of Technology.

Following a chance conversation between them in 1994, Weinstock and Youdim realized that by combining their expertise and amalgamating the active components of their respective drugs, they might produce a more powerful new drug for combating and controlling the progress of Alzheimer's. Their research has been funded by Israel's leading drug company, Teva Pharmaceuticals.

Using an animal model to test the drug, some of the earliest stages of Alzheimer's disease have been elucidated, and insights have also been provided into the workings of the normal brain.

The brain is comprised of two chief types of cells - neurons and glial cells. Neurons are responsible for all the mental processes of understanding; the glial cells provide the support and protection essential for normal neuron function. The glial cells consist of different types of cells including astrocytes which reach out with long fibrous structures and cling to neurons,

supplying them with nutrition and oxygen. There are also microglia which act as the brain's local immune response, devouring intruders such as viruses or bacteria that might disturb the healthy function of the brain.

The brain, even a sleeping brain, requires a great deal of energy which is provided through metabolism of glucose in the astrocytes. Unfortunately, this metabolic process produces highly reactive "free radicals" and as we get older the brain fails to produce enough of the enzymes needed to neutralize them. Instead these oxidative free radicals rampage through the brain, attaching themselves to cell membranes and various proteins.

Recognizing these free radicals as dangerous intruders, the microglia spring into action and a full-blown immune response is initiated with special chemicals called cytokines released. The cytokines attack the astrocytes, and glucose can no longer be metabolized. The energy supply to the brain is thus decreased, the neurons can no longer function properly, and memory begins to fail. Alzheimer's has claimed another victim.

Having developed a rat model that replicates these early features of the disease, Weinstock-Rosin has been able to test the drug ladostigil. The results are impressive: the drug is able to combat the initial stages which lead to degeneration. It plays a key role in restoring normal metabolic processes in the brain's support system - the astrocytes return to their normal status, neuron function is restored, and memory skills return.

Now nearing completion of clinical trials Phase II, ladostigil has so far been proven a safe drug. The experiments with animal models demonstrate that the drug can not only slow the loss of cognitive function in Alzheimer's and Parkinson's diseases but also help alleviate symptoms of major depression. Weinstock-Rosin is convinced that these three debilitating forms of attack on normal brain function have common elements.

Stressed Out

Diseases like Alzheimer's and Parkinson's do not have a single, simple cause. In an organ as complex as the brain many processes seem to lead to neurodegeneration.

It has become clear through the research of Professor Hermona Soreq, Dean of the Faculty of Science and a member of the Department of Biological Chemistry in the Alexander Silberman Institute of Life Sciences and

the University's multidisciplinary Eric Roland Center for Neurodegenerative Diseases, that stress, both psychological and chemical, has an impact on the progress of neurodegenerative diseases and even affects neuromuscular and blood cell diseases.

Soreq has pioneered the development of new techniques to explore the underlying processes which produce damage, and she has successfully identified the mechanism whereby anxiety and stress exacerbate neurological diseases such as Alzheimer's. She is now progressing with the development of highly innovative treatment.

Soreq is driven by a two-fold quest. "By improving our understanding of the diseased brain, we can develop better ways to prevent and treat," she says. "However, if we study the changes that take place in a system pushed to the extreme, then we also learn how it functions normally - this is a basic research goal."

In the brain a large family of neurotransmitters moves between neurons. These chemicals form the web of communication between neurons which is the very essence of normal brain function. The normal brain produces a large family of neurotransmitters. They are secreted by one brain cell, move across to another, latch on to a receptor and thereby stimulate processes within the second cell. Cascades of neurotransmitters create a whole web of communication between different cells in the brain.

In Alzheimer's disease this network is disrupted. The amount of an essential neurotransmitter, acetylcholine, drastically declines; the connection between brain cells is broken, and memory and other brain functions eroded.

The disappearance of this neurotransmitter under stress has been shown by Soreq to be due to the stress-induced overproduction of acetylcholinesterase (AChE). This enzyme actively destroys too much acetylcholine, leaving the brain lacking a neurochemical essential for its normal cognitive, emotional and psychological functioning.

To prevent the progress of Alzheimer's disease it is necessary to prevent this excess elimination of acetylcholine. While existing drugs inactivate the destructive enzyme AChE, Soreq and her team have taken a highly original approach. They have designed a drug which prevents AChE from even being produced by the brain. AChE, like all enzymes, is manufactured by instructions contained in the genetic code on a stretch of DNA in the nucleus of a cell. It took Prof. Soreq's team five years to identify the actual

gene responsible for the manufacture of this particular enzyme. This pioneering work involved developing completely new research tools to carry out these technically innovative procedures.

Having identified the gene, the scientists made an inverse copy of it. This mirror image of the gene could then lock onto the original puzzle stretch of the gene, rather like two pieces of a jigsaw fitting together perfectly. The new DNA-based drug clips on to the exposed gene, making it a sealed unit and creating an unfamiliar package in the cell. The cell immediately identifies it as an unwelcome intruder and destroys it, just as if it were an invading virus.

This protective drug, known as Monarsen (for Prof. Soreq's nickname "Mona") has been developed through the start-up biopharmaceutical company, Ester Neuroscience. It is already being administered to a group of patients as part of Phase II clinical trials under the approval of the US Food and Drug Administration (FDA), which has designated it as an 'orphan drug' for the treatment of the rare muscle-wasting autoimmune disease myasthenia gravis.

Monarsen will soon be poised to enter final Phase III clinical trials. If the drug continues to prove its efficacy, it will receive the essential FDA approval that permits it to enter the pharmacopoeia available to the prescribing physician. "The development of a new drug costs well over \$500 million; most of the investment goes into this last phase," says Soreq, who is optimistic that Ester Neuroscience will find a strategic partner for this potentially powerful new weapon against the misery and debilitation caused by Alzheimer's and other inflammatory diseases such as myasthenia gravis.

This article was forwarded by Linda Chen. Thanks, Linda.

Deep Brain Stimulation (DBS)

Thomas M. King,

March 9, 2007 - When I had my DBS operation at Kaiser Hospital in Sacramento on March 31, 2004, I had no idea how electronic stimulation of the brain might work.

Since 1996, when I was first diagnosed with Parkinson's disease, my condition declined over a period of eight years. Slowly, then more rapidly, the amount of medicine required to suppress the symptoms increased. Fortunately, I could still sleep at night without medicines before my long-awaited operation. The amount of levodopa that replaces

natural dopamine in the brain of Parkinson's disease patients has an upper limit for many people. I was approaching mine.

After the operation, the nurses placed me on half my daily medicine. I asked why. Why not a third or a fourth? No answer. But at only 1.0 mg of Mirapex per day, I may never know the joy of compulsive gambling or shopping, or of going to sleep at the wheel of a motor vehicle, results that some people report after taking Mirapex.

Despite the fact that this operation was pioneered in Grenoble, France, in the early 1990s, and gained approval in the U.S. in 1996, no one seemed to **know** why one half of the previous medicine was appropriate. I do not relate this story to attack or belittle the staff, surgeons, or nurses. The DBS operation is NEW. And, the one fact that every Parkinson's disease person agrees on is that no two Parkinsonians display the same symptoms.

There is still no way to diagnose Parkinson's in a live patient, no way to stop its progression, and no cure. DBS is not a cure. It is the next best thing, in 2007.

I was almost desperate and willing to try this operation. My experience was positive, despite some problems. No major infections, no broken wires, and no bad stimulator programming.

I woke up on April Fools Day, April 1, 2004, having been flat on my back, sleeping during the 12 to 14 hours of surgery. Kaiser's Dr. Pappas installed both probes in my brain at the same time. The recovery room attendant expected to see another person struggling for survival. I felt fine! No more shaking. The temporary effect of the operation had begun.

In May 2004, Dr. Pappas installed in my right breast my battery-powered **Kinetra Stimulator**, and attached it to the two probe wires on the right side of my head. The area on my head where the wires were attached to my Kinetra never healed. On July 27, 2005, the surgeon operated again to bury the wire connector in my skull. This time it seems to have healed.

When the battery was hooked up, the "fun" began. People are learning how to program the stimulators. I am among the first to experience the process. After all, the operation, even in 2007, is less than 20 years old. Today DBS is being used for certain other conditions: Tourette's Disease, restless leg, and depression.

LEFT SHOULDER: My first experience, even before electric stimulation, was wonderful. During the operation, which began about 5 A.M. with an MRI of

my brain, the medical attendants woke me up and asked how “that” felt. “That” was the placement of the probe in the best place in my brain. My left shoulder no longer had constant pain. I no longer bent over all the time. I could sit up. DBS has maintained my freedom from left shoulder pain and rigidity.

FOG: My experience with stimulation has sometimes been frightening and confusing. Sometimes it resulted in a “fog” over my head. I knew it was not real, or there. But it was not until 2007 that I met a nurse who did not just say, “It’s not there.” This one said, “I know what is wrong,” and very soon the imagined fog lifted. Now I think it was the result of over-stimulation.

BOWELS: After the operation, they worked better, but deteriorated during stimulation. When the stimulation node was changed, the bowels returned to functioning almost normally.

WALKING: Stimulation has made my left foot land off center. It is a small thing. As I limp through five-mile hikes, I keep reminding myself of the gains from the operation.

MANIC: The most spectacular and upsetting effect from stimulation, for me, has been what I named, my “Jim Carrey effect”, or manic behavior. I do not normally feel excited constantly. But, at some level of stimulation, I become manic. It is probably an effect most Parkinsonians experience, called dyskinesia. I have extra movements that do no harm, but seem silly or strange to me, because of the mental confusion common to Parkinsonians. I am reluctant to share this effect of DBS stimulation.

VOICE: The following is an example of the Catch 22 of Parkinson's Disease DBS. Today my voice is very soft. People often must ask me to talk louder. I had no problem before my operation. After the DBS operation my voice deteriorated. Until recently, I had almost no voice and spoke too rapidly. The soft voice is the result of a change in the stimulation. It is better than no voice.

Loss of the voice is a common Parkinson's disease patient's experience. One nurse suggests that to talk I turn off the stimulator. After talking, I can stop shaking by turning the stimulator back on.

RIGIDITY: I do not have rigidity in my left arm because of DBS stimulation. But my right arm is becoming more rigid. It pops when I move it. Unlike the left side, my right shoulder did not hurt and cause me to bend over in 2004, during the operation. Now, three years later, it hurts. Now, sometimes I want to bend over to stop the pain.

CONCLUSION: Through stimulation, nurses and doctors can control the tremor, shoulder pain, bowels, walking, emotional feelings, voice, and rigidity.

Deep Brain Stimulation (or DBS) can control many of the effects of Parkinson's disease and allow me to live a more normal life. But no one completely understands DBS.

Some days I feel like the man who jumped out of the window of the tall building. As he flew past the 10th floor going down, he remarked, "So far so good!"

Note: At the time of the operation I took two 50/200 CR (controlled release) Sinemet pills, plus two of the new agonists, Mirapex, 0.25 mg (small white) pills EVERY FOUR HOURS. That is, for the mathematicians who like to add, a total of eight CR pills and eight Mirapex (or Pramipexal). More math: eight pills of 50/200 each CR Sinemet (50 carbadopa, 200 levadopa) makes 200/800 mg CR per day. The same calculations work for the agonist, Mirapex: $0.25 \times 2 \times 4 = 2.0$ mg per day.

Alcohol Consumption Can Damage Hearing

Researchers believe that alcohol can cause varied damage to different parts of the brain.

A new study uses brain currents called brainstem auditory evoked potentials (BAEPs) to examine the effects that cumulative, life-long alcohol consumption may have on hearing.

Results show that heavy alcohol consumption can lead to brainstem damage, resulting in hearing degradation.

Although alcohol researchers believe that drinking can cause brain damage, the quantity of alcohol and the length of time needed to accomplish this remain unknown. In the March issue of *Alcoholism: Clinical & Experimental Research*, scientists in Germany specify the damage that cumulative, life-long alcohol consumption can inflict on central auditory pathways, which is reflected as hearing loss.

"The main problem with identifying alcohol-related brain damage has been to identify those lesions caused by alcohol itself versus those caused by other common alcohol-related factors, such as thiamin deficiency," said Elisabeth Stephanie Smith, a member of the Ear, Nose and Throat Clinic at the University of Ulm, Germany, and first author of the study.

For example, she said, some alcoholics lose white matter, which can lead to overall brain shrinkage, and

may be partially reversible. Alcohol-related damage has also been noted in specific regions of the cerebral cortex hypothalamus and cerebellum, and possibly in the hippocampus, amygdala and locus ceruleus. "Many of the regions that are normal in alcoholics are damaged in those who have developed the Wernicke-Korsakoff syndrome," she added. "While documented dendritic and synaptic changes in alcoholics, together with receptor and transmitter changes, may explain the functional changes and cognitive deficits that precede more severe structural neuronal changes."

For this study, researchers recruited two groups of males from the University of Ulm: 19 head- and neck-tumour patients, representative of heavy drinkers according to previous research; and 19 age- and nicotine-matched plastic-surgery patients, representative of social drinkers according to previous research. All participants were given a standardized questionnaire regarding alcohol use, blood tests, and a hearing examination. Recordings and evaluation of brainstem auditory evoked potentials (BAEPs) were used to measure brain damage in the subjects.

Deviations from standard BAEP latencies and amplitudes generally reflect various diseases affecting the auditory nerve and central auditory pathways. "BAEPs are diffuse currents circulating in the brain," explained Smith. "When an acoustic stimulus on the brain is presented, a particular current response is activated. This response can be detected by electrodes and displayed as a kind of time-dependent voltage. Diseases like cerebellar angle tumours or multiple sclerosis cause defects in the transmission of brain currents, affecting the amplitude and/or latency of the current response, which can in turn be detected by means of BAEP."

The results indicate that cumulative life-long alcohol consumption affects BAEP latencies, which reflect damage to central auditory pathways, which means hearing loss.

"Not only does chronic alcohol consumption cause the already mentioned brain shrinkage," said Smith, "but it also leads to defects of the central auditory tracks, which causes delays in neurotransmission time. These defects cannot be detected by commonly used listening tests for hearing performance."

Interestingly, the study also found that the BAEP latencies of social drinkers appear to be significantly more sensitive to the increase of cumulative life-long alcohol consumption than do those of moderate and heavy drinkers.

"The finding of this article does not necessarily imply that social drinkers' hearing is at more risk of damage than that of moderate and heavy drinkers," said Tilman Keck, assistant clinical professor in the department of Otorhinolaryngology at the University of Ulm. "In fact, drinkers with lower life-long alcohol consumption still have a normal amount of healthy nerves in the brain whereas drinkers with high life-long alcohol consumption have a much larger amount of defective nerves." It is an issue of "saturation," he noted.

"For each unit of further alcohol consumption, the absolute amount of nerves damaged for both kinds of drinkers is the same," he said. "However, the relative change of brain damage and subsequent further degradation of the hearing performance in the brainstem due to alcohol consumption will be significantly higher for drinkers with lower life-long alcohol consumption than for those with high life-long alcohol consumption."

"In other words," said Smith, "if nearly all the nerves are damaged, no further nerve damage and thus no more degradation of the hearing performance can be possible." The bottom line, she added, is that "even an amount of alcohol consumption which is normally accepted by society can have a damaging effect on the auditory system."

Smith said that future studies may examine the relationship between chronic alcohol consumption and distorted speech.

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Know Stroke. Know the Signs. Act in Time.

Know Stroke

Stroke is the third leading cause of death in the United States and a leading cause of serious, long-term disability in adults. About 600,000 new strokes are reported in the U.S. each year. The good news is that treatments are available that can greatly reduce the damage caused by a stroke. However, you need to recognize the symptoms of a stroke and get to a hospital quickly. Getting treatment within 60 minutes can prevent disability. What is a stroke? A stroke, sometimes called a "brain attack," occurs when blood flow to the brain is interrupted. When a stroke occurs, brain cells in the immediate area begin to die because they stop getting the oxygen and nutrients they need to function.

What causes a stroke?

There are two major kinds of stroke.

The first, called an ischemic stroke, is caused by a blood clot that blocks or plugs a blood vessel or artery in the brain. About 80 percent of all strokes are ischemic. The second, known as a hemorrhagic stroke, is caused by a blood vessel in the brain that breaks and bleeds into the brain. About 20 percent of strokes are hemorrhagic.

What disabilities can result from a stroke? Although stroke is a disease of the brain, it can affect the entire body. The effects of a stroke range from mild to severe and can include paralysis, problems with thinking, problems with speaking, and emotional problems. Patients may also experience pain or numbness after a stroke.

Know the Signs

Because stroke injures the brain, you may not realize that you are having a stroke. To a bystander, someone having a stroke may just look unaware or confused. Stroke victims have the best chance if someone around them recognizes the symptoms and acts quickly.

What are the symptoms of a stroke?

The symptoms of stroke are distinct because they happen quickly:

Sudden numbness or weakness of the face, arm, or leg (especially on one side of the body)

Sudden confusion, trouble speaking or understanding speech

Sudden trouble seeing in one or both eyes

Sudden trouble walking, dizziness, loss of balance or coordination

Sudden severe headache with no known cause

What should a bystander do? If you believe someone is having a stroke – if he or she suddenly loses the ability to speak, or move an arm or leg on one side, or experiences facial paralysis on one side – call 911 immediately.

Act in Time Stroke is a medical emergency. Every minute counts when someone is having a stroke. The longer blood flow is cut off to the brain, the greater the damage. Immediate treatment can save people's lives and enhance their chances for successful recovery.

Why is there a need to act fast?

Ischemic strokes, the most common type of strokes, can be treated with a drug called t-PA that dissolves blood clots obstructing blood flow to the brain. The window of opportunity to start treating stroke patients is three hours, but to be evaluated and receive treatment, patients need to get to the hospital within 60 minutes.

What is the benefit of treatment?

A five-year study by the National Institute of Neurological Disorders and Stroke (NINDS) found that some stroke patients who received t-PA within three hours of the start of stroke symptoms were at least 30 percent more likely to recover with little or no disability after three months.

What can I do to prevent a stroke?

The best treatment for stroke is prevention. There are several risk factors that increase your chances of having a stroke:

High blood pressure

Heart disease

Smoking

Diabetes

High cholesterol

If you smoke – quit. If you have high blood pressure, heart disease, diabetes, or high cholesterol, getting

them under control – and keeping them under control – will greatly reduce your chances of having a stroke.

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Emory Participates in Study to Slow Progression of Parkinson's Disease

5/24/2007 (Dental Plans.Com) - Emory University is participating in one of the largest ever Parkinson's disease (PD) clinical trials to determine if the nutritional supplement creatine can slow the symptom progression of this disorder.

PD is a degenerative disorder of the brain in which patients develop tremor, slowness of movements and stiffness of muscles. It affects at least one million people in the U.S. Currently, there are a number of effective treatments to mask the symptoms but none to slow their progression.

Emory is among 51 medical centers in the U.S. and Canada recruiting the 1,720 participants with early-stage PD required to complete this study. The double-blind, placebo-controlled, phase III study is the first large national study following a series of smaller clinical trials sponsored by the National Institutes of Health (NIH).

"The premise for this important research study is supported by a large body of laboratory data and the promising results of an earlier smaller clinical study of creatine in Parkinson's disease," says Dr. Jorge Juncos, primary investigator at Emory University.

"If positive, the results will have a lasting impact in the treatment of all stages of this illness," says Dr. Juncos. "Our goal is to offer enhanced therapies to patients with Parkinson's that will improve their quality of life and slow the progression of symptoms."

Creatine is marketed as a nutritional supplement, but is not an approved therapy for PD or any other condition. Studies have suggested that it can improve energy production by the mitochondria, the "powerhouse" of cellular metabolism. In persons without PD and in patients with PD, it can improve exercise performance so long as the person is also exercising regularly. It is important to note that creatine is no substitute for exercise and its short-term use in PD does not directly affect motor symptoms, says Dr. Juncos.

Creatine also acts as an antioxidant to prevent damage from compounds that are harmful to cells in the brain. In mouse model studies of PD, creatine was able to prevent the loss of the dopamine cells in the brain, the same cells that are also affected in PD.

"This study is an example of our commitment to Parkinson's research," says Story C. Landis, PhD, director of the National Institute of Neurological Disorders and Stroke (NINDS), the NIH institute leading the study. "We are trying to explore every possible option for reducing the burden of this disease."

The study will enroll people who have been diagnosed with PD within the past five years and who have been treated for two years or less with levodopa or other drugs that improve dopamine transmission in the brain. Many of the symptoms of PD result from the loss of dopamine, a brain chemical that helps to control movement. Half of the participants will receive creatine and half will receive a placebo. Neither the participants nor their doctors will know which treatment to which the participants have been assigned.

The investigators will measure disease progression using standard rating scales that measure quality of life, cognitive function, walking and the ability to carry out other activities of daily living.

People in the Atlanta area interested in participating in this study can obtain more information by calling 404-778-7777 or visiting www.parkinsontrial.ninds.nih.gov.

Pain in Parkinson's Disease

from Parkinson's Disease Foundation
publication date: 2005

For most people with Parkinson's disease (PD), the most serious concern is with the motor system: stiffness, slowness of movement, impaired handwriting and co-ordination, poor mobility and balance. Descriptions of PD do not generally include the mention of pain. And yet, when carefully questioned, more than half of all people with Parkinson's disease say that they have experienced painful symptoms and various forms of physical discomfort. Most people experience aching, stiffness, numbness and tingling at some point in the course of the illness. For a few of them, pain and discomfort are so severe that they overshadow the other problems caused by the disease. This article will address these overlooked painful symptoms of PD, and describe an approach to diagnosing and treating the various pain

syndromes that may occur.

Pain is described in textbooks as an unpleasant experience associated with physical injury or tissue damage. Pain can arise from anywhere in the body, of course. It goes without saying that people with Parkinson's are subject to all of the painful conditions — cardiac, gastroenterological, rheumatological, among others — that can affect people without PD. This discussion will focus on pain that is directly related to PD itself.

Pain syndromes and discomfort in Parkinson's usually arise from one of five causes: (1) a musculoskeletal problem related to poor posture, awkward mechanical function or physical wear and tear; (2) nerve or root pain, often related to neck or back arthritis; (3) pain from dystonia, the sustained twisting or posturing of a muscle group or body part; (4) discomfort due to extreme restlessness and (5) a rare pain syndrome known as "primary" or "central" pain, arising from the brain.

It takes diagnostic skill and clinical experience to determine the cause of pain in someone with PD. The most important diagnostic tool is the patient's history. Where is the pain? What does it feel like? Does it radiate? When does it occur during the day? Does it occur in relation to any particular activity or medication? Perhaps the most important task for people with Parkinson's who experience pain is to describe as accurately as they can whether their medications induce, aggravate or relieve their pain. To help your physician in diagnosing pain, refer to the questions listed on the back at the end of this article on page 9.

Musculoskeletal pain

Aching muscles and joints are especially common in PD. Rigidity, lack of spontaneous movement, abnormalities of posture and awkward mechanical stresses all contribute to musculoskeletal pain in PD. One of the most common musculoskeletal complaints is shoulder stiffness, sometimes called a frozen shoulder (this may in fact be the first sign of PD). Hip pain, back pain and neck pain are all common painful complaints in PD. With prolonged immobility of a limb, band-like tendons, termed contractures, may occasionally develop, usually in the hands or feet; one example is the clenched fist contracture that may occur with prolonged flexion of a hand.

An accurate diagnosis of musculoskeletal pain is based on a careful history and a physical examination that takes into account posture, limb and trunk rigidity and

gait. It can occasionally be challenging to distinguish between back pain due to PD and that caused by arthritis or scoliosis. Occasionally, further testing — including x-rays, bone scans, ultrasound and rheumatologic or orthopedic consultation — will be needed. The proper treatment of musculoskeletal pain in PD depends upon the cause of the pain. If the pain is the result of excessive immobility or rigidity, a physician may prescribe dopaminergic therapy, physical therapy and an exercise program. If the treatment is successful, patients should continue with an exercise program that strongly emphasizes range of motion, to prevent the development of further musculoskeletal problems.

Radicular and neuritic pain

Pain that occurs close to a nerve or nerve root is described as neuritic or radicular pain. The classic root-pain syndrome is sciatica, caused by compression or inflammation of the L5 lumbar root. Patients usually describe root pain as a sharp, lightning-like sensation that radiates towards the end of a limb. Of course, any nerve or root may be subject to injury or compression, and a careful neurological assessment is needed for the diagnosis. Electrodiagnostic studies and neuroimaging are occasionally required to confirm the location of the involved nerve or root, and to determine the cause of the problem. Radicular pain can usually be successfully treated with a mobility program and pain medication and rarely requires surgery.

Pain associated with dystonia

Dystonic spasms are among the most painful symptoms that a person with PD may experience. The pain arises from the severe, forceful, sustained twisting movements and postures that are called dystonia. This type of muscle spasm is quite different from the flowing, writhing movements described as dyskinesias, which are not painful. Dystonia in PD may affect the limbs, trunk, neck, face, tongue, jaw, swallowing muscles and vocal cords. A common form of dystonia in PD involves the feet and toes, which may curl painfully. Dystonia may also cause an arm to pull behind the back, or force the head forward towards the chest.

The most important step in evaluating painful dystonia is to establish its relationship to dopaminergic medication. Does the dystonia occur when the medication is at peak effect? Or does it occur as a "wearing-off" phenomenon, when the benefits of medication are waning? The answers to these questions will usually clarify the nature and timing of the dystonia, and determine its treatment. Most painful dystonia represents an "off" parkinsonian

phenomenon, and occurs early in the morning or during wearing-off spells. In uncertain cases, the neurologist should observe the patient in the office over a period of several hours in order to appreciate the relationship of the dystonia to the medication-dose cycle.

In terms of treatment, early-morning dystonia is typically relieved by physical activity, or by the first dose of dopaminergic medication, whether it be levodopa (Sinemet®) or a dopamine agonist. When dystonia occurs as the medications wear off, the problem can be corrected by shortening the “off” period. In some patients, the dystonia is so severe that subcutaneous injections of apomorphine, with its onset of action in minutes, may be necessary. Individuals with intractable dystonia may benefit from deep brain stimulation, a neurosurgical procedure that involves implanting and activating electrodes in the brain.

A few patients experience dystonic spasms as a complication of their medications. When they take their levodopa, these patients experience dystonic facial grimacing or uncomfortable limb posturing. The standard treatment approach for these individuals is to reduce the amount of dopamine medication, sometimes by substituting a less potent agent, or adding a medication for dystonia, such as amantadine.

Akathisia

No discussion of physical discomfort in PD is complete without a mention of akathisia, or restlessness, a frequent and potentially disabling complaint. Some patients with parkinsonian akathisia are unable to sit still, lie in bed, drive a car, eat at a table or attend social gatherings. As a result of akathisia, patients may lose sleep or become socially isolated. In about half of the cases of parkinsonian akathisia, the symptom fluctuates with medications and may often be relieved by additional dopaminergic treatment.

Central pain syndromes

The most alarming pain syndrome in patients with PD is also one of the rarest: “central pain.” This affliction — which is presumed to be a direct consequence of the disease itself, not the result of dystonia or a musculoskeletal problem — is described by patients as bizarre unexplained sensations of stabbing, burning and scalding, often in unusual body distributions: the abdomen, chest, mouth, rectum or genitalia. The treatment of central pain in PD is challenging, and usually begins with dopaminergic agents. Conventional pain-killers, opiates, antidepressants and powerful drugs for psychosis, such as clozapine, may also be helpful treatments for central pain.

Depression and pain

It has long been known that chronic pain can induce depression, and depressed patients often experience pain. People who have PD are themselves at a higher-than-average risk for developing depression, which occurs in some 40 percent of patients at some point during the illness. It is therefore important that any assessment of pain in an individual with PD take into account the potential contributing role of depression, which may also require treatment.

Many patients with PD experience pain at some point during the illness. The complaint is often overlooked because PD is primarily a motor disorder. Yet, for a minority of patients, pain and discomfort can be so debilitating that they dominate the clinical picture. It is therefore important that individuals who experience pain discuss the problem with their neurologist. A careful history and examination — including, in some cases, additional diagnostic testing — can usually determine the cause of the pain. Depending on the category of painful complaint — musculoskeletal, root or nerve pain, dystonic muscle spasm, akathisia or central pain — it is usually possible for the physician to design an effective treatment plan.

10 Questions your doctor will ask you about pain:

- Where is your pain located?
- What does your pain feel like?
- Does the pain radiate anywhere?
- When does the pain occur?
- Do you have pain continuously, or only at certain times?
- Does the pain occur in relation to any particular activity?
- What relieves the pain?
- What makes the pain worse?
- Do your anti-Parkinson’s medications relieve your pain?
- Do you have arthritis?

This article was forwarded by Robin Riddle, the leader of Aptical Parkinson’s Support Group in San Mateo. Thanks, Robin.

Log on to www.ppsg.org

***This newsletter was assembled by
The Morgan Center. Thank You!***

PPSG SUPPORT GROUPS

---SOUTHERN REGION---

Berkeley 3rd Wed, 1-3, North Berkeley Senior Center, 1901 Hearst Av, Roddy Raikow 510-231-1998 or Mitzi Cahn 510-527-9075 **Fremont** 4th Mon 7:00 pm Fremont Senior Center 40086 Paseo Padre Parkway, Lettie Webb 510-656-6393 **Fremont Caregivers** Contact Nancy Rothschild, Caregiver Project Coordinator, 510-574-2035 **Marin County** 4th Tue most mo., 2-4 Redwoods Auditorium 40 Camino Alto, **Mill Valley**, Gloria Rashti 415-381-6680. Redwoods 415-383-2741 **Mt. Diablo Parkinson's Network General Meetings** 2nd Sat 10-12, Grace Presbyterian Church, 2100 Tice Valley Blvd, **Walnut Creek**, Nancy Walls, 510-236-7065, Philip Wheeler, 510-527-3588, Margy Hansell, 925-939-4210, or Ronalee Spear, 925-284-2189 **Oakland** 1st Thur 1:30-3:30 Easter Seals Bay Area, 180 Grand Av, Suite 300, Karen & Jim Eagan, 510-763-4492 **Petaluma** Last Sat 1:30-3:30 Sunrise of Petaluma, 815 Wood Sorrel Dr, John & Mamie Strong 707.763.3522 **Pleasanton Tri-Valley** 2nd Sat 10-12, Senior Center, 5353 Sunol Blvd, Norm & Jackie Bardsley 925-244-1231 or 925-831-9940 **San Leandro** 1st Thur (except Jul & Aug) 10-11:30, (NEW LOCATION) **San Lorenzo** Community Church, 945 Paseo Grande, Norma Zeff, 510-663-6435 **Sonoma County** 1st Sat (not Jan, Jul, Sep) 1-3, First Congregational Ch, 2000 Humboldt St, **Santa Rosa**, Ron & Colleen Trowse 707-526-4373 **Vallejo** 3rd Mon (except 2nd Mon, Jan & Feb) 2:00 Kaiser Medical Center, 975 Sereno Drive, Evelyn Fox 707-644-3390

---PENINSULA REGION---

Daly City 1st Tue 3-4 Doelger Senior Center, 101 Lake Merced Blvd, Leonard Ke 415-587-1285 **Los Altos Young Parkinson's Support Group** 2nd Sat 10-12, United Methodist Ch/Los Altos, Foothill at Magdalena, Dean Prescott 408-738-2505 or dean53@yahoo.com **Magnolia-Peninsula** 2nd Thur 1:30 main conference room Magnolia Apart, 201 Chadbourne Av, **Millbrae**, Leon Rosenthal, 650-348-3480 **Palo Alto** 2nd Wed 2:00-3:30 Avenidas Senior Center dining room, 450 Bryant St, 650-289-5400 **Redwood City Positive People Against Parkinson's** 3rd Fri 1-2:30, (No meetings Aug, Nov, Dec) Sequoia Hospital, Health & Wellness Ctr, 749 Brewster Ave, Tom Constantino 650-366-7166, or David Shein, 650-367-5998 **(NEW) San Francisco Caregivers** Thur (varies) 12-12:50 Veterans Affairs Med Ctr, Parkinson's Ctr conf room, Bldg 203 Room 1B26A, Susan Heath 415-379-5530 or Aliza Benditsky 415-221-4810 X 4741 **San Mateo Atypical Parkinsonism (PSP, LBD, MSA, CBD) Bay Area Caregivers** Sundays 5-7 about every 6 weeks, Mimi's Café 2208 Bridgepointe Parkway, San Mateo, Robin Riddle 650-233-9277 or rriddle@stanfordalumni.org **San Mateo Caregivers** 1st Wed 2:30-4:30 Ellsworth Room 100 San Mateo Dr., Call Ann Sasaki, Mills Health Center 650-696-4741 **Sunnyvale** 2nd Wed 1-3 First United Methodist Ch, 535 Old San Francisco Rd, Phyllis & Henry Ng 408-733-5648 **YOPD** (Young Onset Parkinson's Disease) 2nd Tue 6:30-8:00, Board Room, Lucile Packard Child Hosp, 725 Welch Road, **Palo Alto**, Martha Gardner, 866-250-2414.

Hollister 1st Tue 1:30-3:30 First Presbyterian Ch, 2066 Cienega Road, Shirley Kennedy 831-637-3839 or John Skinner 831-637-6755 **Monterey** 3rd Mon 2:30-4:00 SHARE Room, Monterey Adult School, 200 Coe Av, **Seaside**. Helen Garrett 831-657-4241 or Kathy Warthan 831-372-7510 **Salinas** 4th Wed 2:00-3:30 Salinas Adult School, 20 Sherwood Place, Sherry Whitcomb, 831-796-6920 **San Jose-Berryessa** 1st Wed 1:00-2:30 Berryessa Community Center, 3050 Berryessa Rd, Bob & Jane Pomeroy 408-263-8485 **San Jose Caregivers** usually 4th Wed 1:30-3:30 St Francis Episcopal Church, 1205 Pine Ave, Charmaine Eng 408-723-8116 **San Jose-Willow Glen** 1st Fri 10-12 St Francis Episcopal Church, 1205 Pine Ave, Betty Havens 408-227-8593, or Joan Lorentson, 408-997-7009 **Santa Cruz** 1st Wed 12:30-2:00 St. Stephen's Lutheran Church, 2500 Soquel Ave, David Donahoe 831-479-4485 **Saratoga** 3rd Tue 2-4 19449 Via Real, Lois McPherson 408-867-1807

---CENTRAL VALLEY REGION---

Fresno, Greater 2nd Sat 10 at San Joaquin Valley Rehab Hosp 7173 N. Sharon Ave, Max Robinson, 559-226-2673 **Merced** 4th Thur 10AM (Nov 17, Dec no meeting) Mission Gardens 1450 E. 27th St, Amie Marchini 209-384-3300 **Modesto** 3rd Wed 1:30-3:00 Centenary United Methodist Ch, Fireside Room 1911 Toyon Av, JoAnn & David Ryan 209-529-5643 or davejoann@sbcglobal.net **(NEW) Pine Grove** 1st & 3rd Thur 2-4 Calvary Chapel Patio Bldg 18400 Ridge Road, Sarah Johnson 209-296-2575 **Roseville** 1st Tues 1:30-3:00 Roseville Maidu Comm Ctr, 1550 Maidu Drive, Linda Krisa 916-261-1321 **Tulare-Kings** 1st Fri 10:30 **Visalia** United Methodist Church, 5200 W. Caldwell Av, Mary Dickerson 559-622-9044, Church Office 559-627-1660 **(FORMING) Turlock** Donald Jackson 209-606-9127 November 22, 2006

EXERCISE CLASSES

Berkeley: Vista College, Joan Nielsen, 510-981-2800

Berkeley: Mon. 10:30-11:30 & 1-2:30, John Argue 510-985-2645

Daly City: Tue./Thur. 1-2, Doelger Sr. Ctr. Pat Armstrong 650-991-8012

Gilroy: Gavilan College, Dave Ellis, 408-848-4878

Hayward: Kaiser Permanente, Wed. 10-11:30, John Argue 510-985-2645

Kensington: Tue. 1:30-3:00, John Argue 510-985-2645

Marin Cty: Tue. 10-11:30; 12-1:30. Osher Marin JCC, San Rafael. 415-479-2000

Monterey: Monterey Peninsula College, Mark Clements, 831-646-4231

Orinda: Tue. & Fri. 1:00-2:30, In Forma Gym. Dean Dallman 925-283-5019

Palo Alto: CAR, Aquatic Therapy, 650-494-1480

Palo Alto: Mon. & Fri., 9:15-10:15, Sr. Ctr. 450 Bryant St. 650-289-5400

Redwood City: Canada College, 4200 Farm Hill Blvd. Barbara McCarthy 650-306-3473

Salinas: Hartnell College, Melissa Stave, 831-755-6876

Saratoga: Mon. – Fri. 9-12; 1:30-3, W. Valley Comm. Coll. Joan 408-741-2420

San Bruno: Mon. & Wed. 1:10-2:30, Tue. & Thur. 12:35-1:50, Skyline Coll. Bess 650-738-4286

San Francisco: Fri. 11-12, SFSU, Marsha Melnick 415-338-1360.

San Jose: Mon. & Wed. 10:30-11:15 at Camden; Tue. & Thur. 10-11:15 at Evergreen; contact Dominic or Donna at 408.369.6438.

San Jose: Easter Seals Comm Ctr. Aquatic Exercise programs, 408-295-0228

San Jose: Evergreen Valley College, Rich Wagner, 408-274-7900 X 6447

San Mateo: College of San Mateo, 1700 W. Hillsdale Blvd., John Hogan, 650-574-6469

Sunnyvale: Tue. & Thur. 9-10, Sr. Ctr. 550 Remington Dr. Ruth Hanes 408-864-8873

Sunnyvale: Wed. 10-12 noon, The Parkinson's Institute, 1170 Morse Ave., Marilyn Basham: 408-734-2800.

Exercise Classes – New Addition

Palo Alto

Tai Chi/Chi Kung for Parkinson's in the Atrium at Stanford Medical Center

Every Saturday 10:00 am – 11:30 am (short break in between).

Mwezo & Jane

Kujiweza Healing Arts Institute

Call Jane: (408) 315-1179

Email: Kujiweza@sjyogataichi.com

San Jose

The Villages Golf & Country Club

Parkinson's Exercise Program (PEP)

Balance, gait, posture, Tai Chi/Chi Kung

Mondays 11:30 – 12:30 pm

Optional 3-day program

Mwezo & Jane

Kujiweza Healing Arts Institute

Call Jane: (408) 315-1179

Email: Kujiweza@sjyogataichi.com

Los Gatos

The Terraces of Los Gatos

Parkinson's Learning Lifelong Useful Skills (PLLUS)

Balance, gait, posture & Tai Chi/Chi Kung

Mon. 2:00 pm & Wed. 10:30 am

Mwezo & Jane

Kujiweza Healing Arts Institute

Call Jane: (408) 315-1179

Email: Kujiweza@sjyogataichi.com

If you would like to receive our newly-assembled caregivers' packet, please call or write to PPSG.

If you would like to be removed from our mailing list or know someone who would like to be included, please take a minute, call us at **408.734.1593**, or e-mail ppsginfo@yahoo.com, and let us know. Thank you.

Thank you so much for your donations! Please use return address labels, to help us acknowledge your donations properly. Your generous contributions go to support newsletters, education and community awareness of Parkinson's disease.

If you would like to be removed from our mailing list or know someone who would like to be included, please take a minute, call us at **408.734.1593**, or e-mail ppsginfo@yahoo.com, and let us know.

PPSG Board Meetings

The Parkinson's Institute And Clinical Center

On September 1, 2007, we will be moving to an exciting, newly Remodeled and larger facility located just a few minutes away.

Our new address will be

**675 Almanor Avenue
Sunnyvale, CA 94085**

Directions from the current site... take Hwy.101 North to Mathilda exit. Go west Mathilda, make first right onto Almanor Avenue. Travel to new site at 675 Almanor Avenue on right side of road.
STOP BY and Look!

You are welcome to drop by our board meetings and share ideas with us! We meet on the **3rd Monday** of the month between **1:30 and 3:30 PM** at the Parkinson's Institute. To confirm meeting dates and time, please call us at 408.734.1593. If you are planning to attend, please call Charmaine Eng at 408.723.8116 (dial *82 before the number).

Board Members

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Charmaine Eng

Vice Chair:

Dean Prescott

Secretary:

Carla Gwosden

Treasurer:

Allan Daily

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Le Sotir



Parkinson's Patients Support Groups, Inc.
1170 Morse Avenue
Sunnyvale, CA 94089-1605
408.734.1593

ADDRESS SERVICE REQUESTED

July/August 2007