

## UT Southwestern Parkinson's Disease Patient and Caregiver Symposium 2010

by Cindy Weatherall, DAPS Board Member

Several DAPS members were among more than 150 attendees at the Parkinson's Disease Patient and Caregiver Symposium in April. The free-of-charge seminar was sponsored by the UT Southwestern Medical Center Department of Neurology and the Office of Public Education, and was held on-site at UTSW.

Three well-known Department of Neurology faculty members, Shilpa Chitnis, MD, PhD; Richard B. Dewey, MD; and Padraig O'Suilleabhain, MD, provided up-to-date information on topics including possible causes of PD, medical and surgical options for treatment, and current areas of research. A question-and-answer period followed the formal presentations.

While it is not possible to summarize the entire symposium for this newsletter, there are several points that should be mentioned. Although the progress against PD may seem very slow to those who are dealing with the disease now, it is important that any new treatments are subjected to rigorous scientific standards. From the basic laboratory experiments that may shed light on the disease by testing small groups of cells, to the actual drug or surgical treatments that may eventually become common, patients must be protected by practices that carefully test safety and effectiveness. It can be encouraging to hear stories (**anecdotal evidence**) of patients who have benefited from certain treatments, but those stories are not enough, without further investigation in numerous settings, to warrant changing established treatments for large numbers of PD patients.

The **etiology** (study of the origin or causes of a disease) of PD is an area of much research. As the average human lifespan continues to increase, the lifespan of the average neuron is not changing, so we might expect to see greater numbers of people with PD and other neurodegenerative diseases. One question that is receiving attention is "Why are *particular types* of nerve cells affected in PD, versus, for example, those in Alzheimer's disease?"

One hypothesis is that neurons with relatively long axons (the "transmission line" between a neuron and its target cell) and with smaller amounts of protective myelin cover around them may be more vulnerable to the damage caused by alpha-synuclein. This type of neuron is present in areas that would account for many of the symptoms of PD. The substantia nigra (movement control), olfactory center (sense of smell), and medulla (REM sleep disorders) are parts of the brain containing these vulnerable neurons. Outside the brain, this type of neuron is found in the bowel wall, and when they are affected by PD degeneration, may result in constipation.

**Medical and surgical treatments** provide several potential ways to treat PD, and researchers are working to develop disease-modifying (neuroprotective) and restorative (gene therapies, growth factors) treatments to stop the progression of disease and repair damage already done. (See *DAPS Newsletters for April and May 2010 online at [www.daps.us](http://www.daps.us) for more detailed articles.*)

**Deep-Brain Stimulation surgery** (DBS) is performed at UT Southwestern. Not all PD patients are good candidates for this option, which involves a 2-stage surgical procedure to implant electrodes in the subthalamic nucleus of the brain and to place a battery under the collarbone and connecting it to the electrode to provide electrical impulses. Patients most likely to be good candidates have the common PD, where no particular cause (toxins, brain injury) is known. The patient should be

responsive to levodopa treatment, although the patient may be finding that he or she is experiencing increasing “off” periods, when the medication is not working well. People who are NOT good candidates would include those with secondary forms of Parkinsonism (for example, a stroke patient); those with Parkinson’s-Plus syndromes; those with uncontrolled depression; patients with dementia; those with a history of falls that do not respond to levodopa treatment; and patients with other significant medical problems.

One of the most promising agents available to **reduce or halt the underlying process of degeneration** is the medication **rasagiline (Azilect)**. In a study called TEMPO, some patients with early-stage PD were given rasagiline to start, while others were given a placebo (inactive). Later in the trial, the groups were switched. It was found that those who received rasagiline in the delayed group were never able to “catch up” to the benefits experienced by those who received the drug at the beginning of the study. This result, which has been consistent in small groups over about 6 years, indicates that rasagiline may, in fact, alter the course of PD. (For a technical discussion of this study, see [http://www.medscape.com/viewarticle/532116\\_7](http://www.medscape.com/viewarticle/532116_7) )

All studies of **gene therapy** to date have involved open observation with no placebo controls, making unbiased evaluation very difficult. There have also been serious side effects in some of the studies. **Stem cell** therapy is being used in very small studies, and, so far, the techniques are not producing impressive results.

The naturally-occurring chemical **inosine** is being studied as a way to increase urate in the body; people with higher blood urate seem to be less likely to develop PD. However, high urate levels are associated with gout, kidney stones, high blood pressure, and coronary artery disease, so this treatment would have to be very closely monitored.

**Creatine**, a naturally-occurring compound that is marketed as an inexpensive nutritional supplement, is being studied in a large group of about 1800 PD patients in a clinical trial that will last 5 to 7 years. Because the effects of creatine in an older population are not known, and because scientists are not yet sure why creatine may help in PD, it is not recommended as a supplement unless a patient is in the study.

**Exercise** has been recommended to help PD patients, and there are some medical studies attempting to quantify the benefits. In a small trial at the Cleveland Clinic, 10 patients were recruited to participate in a stationary cycling program for 1 hour, 3 times per week, for 8 weeks. Some of the patients rode the cycles at their own pace; others were paired with a trainer on a tandem cycle to attain 30% higher output compared to the others. The trainer generated about 75% of the effort, and the patient about 25%. At the end of the 8 weeks, those patients who were forced to pedal faster showed marked improvement in several areas of PD symptoms; those patients who pedaled on their own exhibited no changes. Four weeks after the end of treatment, however, the benefits in the tandem group began to subside.

The symposium provided a broad overview of the progress being made against PD and offered much more information than appears here. The DAPS members who attended would encourage our readers in the DFW area to consider participating in next year’s presentation. **You may call the UTSW Office of Public Education at 214-648-3138 and ask to be added to the Parkinson’s Disease Symposium mailing list.**

## **Questions and Answers from the UTSW Parkinson's Disease Symposium**

*At the conclusion of the symposium, Drs. Dewey, O'Suilleabhain, and Chitnis took questions from the audience. I have edited the information in the interest of clarity and space. C.W.*

### **What help is available to caregivers to have a more ordinary life?**

It is very important for caregivers to make time for themselves. Support groups are often valuable. **(DAPS offers free caregiver support groups in several locations; they are listed in this newsletter.)**

### **Regarding Deep-Brain Stimulation Surgery:**

The implanted battery has an expected life of 3 to 5 years. Some adjustments to the electrical output may need to be made after the surgery.

The oldest patient receiving the surgery was 84, and had Stage III PD.

There is no evidence of a link between DBS and dementia, except as part of the progression of PD.

A patient with the DBS device must be sure to notify the staff when being scheduled for an MRI exam to be sure the particular machine is compatible with the implant.

### **Who gets PD? How will PD progress?**

There are currently about 1 million people in the US with PD, with the number expected to triple in the next 50 years as the population ages. At age 50, about 10 per 100,000 people have PD; by age 80, there are 200 people with PD per 100,000.

It is impossible to predict the clinical outcome of an individual person. The Hoehn and Yahr Scale is used to classify symptoms and assign stages to PD (Stages 1 through 5, with 1 being the mildest symptoms). As a very broad generalization, about one-third of PD patients will reach H-Y Stage 4 or 5 in five years after diagnosis; another one-third after a period of 5 to 15 years; and another one-third will not reach Stage 4 or 5 until more than 15 years after diagnosis.

### **What are the causes of death in PD patients?**

Complications of PD that can result in death include aspiration pneumonia; falls causing hip fractures and head injuries; infections of pressure sores; deep-vein thrombosis and other blood clots. Many people also have other medical conditions unrelated to PD that cause death.

### **Does stress contribute to the progression of PD?**

While stress can aggravate symptoms of PD, it is unlikely to affect the underlying progression of the disease.

### **Will hypnosis, guided imagery, or yoga help?**

While some patients may find benefits, there are no well-designed studies to prove an effect.

***Disclaimer:** This information is provided for informational purposes only and is not intended as medical advice or as a treatment guide for patients. Please check with your personal physician for any specific questions regarding your medical care.*