PD and Medication: What’s New?

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Consultant/Speaker for Abbvie, Acadia, Accorda, Adamas, Kyona Kirin, Lundbeck, MERZ, Sunovion, Teva, US World Meds


Drug in Kind for Research: Novartis
"Shaking palsy" first described by James Parkinson in 1817

- Involuntary tremor
- Lessened muscular power
- Tendency to bend forward
- Tendency to pass from walking to running pace
- Senses and intellect uninjured

### Motor Signs
- Bradykinesia
- Tremor at rest
- Rigidity
- Postural instability

### Clinical Manifestations
- Decreased arm swing
- Hypomimia
- Hypophonia
- Micrographia

Pathology of Parkinson’s Disease
The Parkinson’s Complex

Evolution of Lewy Body Pathology

PD-related Lewy body pathology evolves in predictable stages. According to the staging system of Braak, Lewy bodies (LB) first form within the olfactory bulb and dorsal motor nucleus of the vagal nerve (Stage 1). In Stages 2 and 3, LB pathology expands from these induction sites into additional brain stem nuclei (e.g. locus coeruleus and substantia nigra) and then into the amygdala. In Stages 5 to 6, the pathology extends into the cerebral cortex. Clinical symptoms arise during Stages 4 to 6, when the pathology involves significant regions of the substantia nigra and related brain areas.

Non-motor Features of PD

• Neuro-psychiatric and cognitive:
  - Depression
  - Anxiety
  - Psychosis
  - **Dementia**
  - Apathy
  - Fatigue
  - Sleep disturbance

• Autonomic:
  - Constipation
  - Hyperhidrosis
  - Urinary dysfunction
  - Sexual dysfunction
  - Sialorrhea
  - Orthostatic Hypotension

• Sensory:
  - Pain
  - Smell loss
Prodromal Phases of PD Reflect Neuronal Loss

*Olfactory dysfunction may predate clinical PD by at least 4 years

A New Definition of PD: A 3-Phase Disease

| Phase 1 | Preclinical PD | PD-specific pathology assumed to be present | • Asymptomatic, but will need to be supported by:  
|• Molecular markers (α-synuclein, DJ-1, LRRK2, parkin, PINK1 mutations)  
|• Imaging markers (transcranial sonography, PET, SPECT, MIBG SPECT, α-synuclein imaging) |
|---|---|---|---|
| Phase 2 | Premotor PD | Presence of early nonmotor signs and symptoms due to extranigral PD pathology | • Premotor features commonly occur before the emergence of motor signs (olfaction abnormalities; constipation; cardiac involvement; neurobehavioral symptoms) |
| Phase 3 | Motor PD | PD pathology involves substantia nigra leading to dopamine deficiency sufficient to cause classic motor manifestations followed by later nonmotor features | • Traditionally diagnosable symptoms (bradykinesia, tremor, rigidity)  
|• May progress to include late PD features (dysautonomia, sensory symptoms, cognitive decline) |

Adapted from Stern MB, Lang, A, Poewe W. Toward a redefinition of Parkinson’s disease. *Mov Disord.* 2012;27(1):54-60, with permission from Copyright Clearance Center on behalf of John Wiley and Sons. Published online in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/mds.24051.
Classification of Parkinson’s Syndromes

Idiopathic PD ~ 85% of all PS
Neuroleptic-induced parkinsonism (DIP) 7-9%
MSA ~2.5%
PSP ~ 1.5%
Vascular Parkinsonism ~3%
MPTP, CO, MN, recurrent head trauma is extremely rare
No New cases of postencephalitic parkinsonism since 1960’s
Epidemiology of PD

PD is the second most common neurodegenerative disorder after Alzheimer’s disease.

Affects 0.3% of worldwide population
– 1%-2% of people aged >60 years

Approximately 1 million people have PD in the US.

Prevalence predicted to almost double in US from 2005-2030 in individuals aged >50 yrs.

The Incidence of PD Increases With Aging

Causes of PD

PD is heterogeneous
– Unlikely there is a single etiology
Contributing factors may include:
– Environment
– Genetics
– Combination of both
Abnormal aggregation of α-synuclein may play a role in the development of PD

Risk Factors for PD

Relative Risk (95% CI) of PD Diagnosis

CCB indicates calcium channel blocker; NSAID, nonsteroidal anti-inflammatory drug.

Genetics and PD

PD is primarily a sporadic or idiopathic disorder. The Human Genome Project has helped to better define the gene association:

- Up to 20% of patients with PD have the familial variety
- Causal and susceptibility genes discovered for PD
- Monogenic forms account for only a very small portion of patients with PD

## Genes Identified for Familial PD

<table>
<thead>
<tr>
<th>Name</th>
<th>AD/AR</th>
<th>Prevalence</th>
<th>Lewy Bodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>SNCA (PARK1, PARK4)</td>
<td>AD</td>
<td>Very rare</td>
<td>LB</td>
</tr>
<tr>
<td>LRRK2 (PARK8)</td>
<td>AD</td>
<td>5% familial Caucasian; 1%-2% of sporadic cases</td>
<td>LB</td>
</tr>
<tr>
<td>PRKN (PARK2)</td>
<td>AR</td>
<td>Most prevalent in early-onset (&lt;45 years) but relatively uncommon</td>
<td>Nigral degeneration; rare LB</td>
</tr>
<tr>
<td>PINK1 (PARK6)</td>
<td>AR</td>
<td>Rare cause of recessively inherited, early-onset Parkinsonism</td>
<td>Unavailable</td>
</tr>
<tr>
<td>DJ-1 (PARK7)</td>
<td>AR</td>
<td>&lt;1% of early-onset PD</td>
<td>Unavailable</td>
</tr>
</tbody>
</table>

### Causal Genes and Loci

AD indicates autosomal dominant; AR, autosomal recessive.

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HUGO (Human Genome Organization). HUGO Genome Nomenclature Committee. genenames.org/genefamilies/PARK.
<table>
<thead>
<tr>
<th>Additional Genes Associated With PD</th>
</tr>
</thead>
<tbody>
<tr>
<td>UCH-L1</td>
</tr>
<tr>
<td>POLG</td>
</tr>
<tr>
<td><strong>GBA</strong></td>
</tr>
<tr>
<td>UNKNOWN (PARK3, 10, 12)</td>
</tr>
<tr>
<td>NR4A2/NURR1</td>
</tr>
<tr>
<td>ATP13A2</td>
</tr>
<tr>
<td>PLA2G6</td>
</tr>
<tr>
<td>FBXO7</td>
</tr>
<tr>
<td>GIGYF2</td>
</tr>
<tr>
<td>Synphilin-1</td>
</tr>
<tr>
<td>OMI/HTRA2</td>
</tr>
</tbody>
</table>
DaTScan™ (labeled I 123 Ioflupane Injection), is a radiopharmaceutical agent recently approved by the FDA for striatal dopamine transporter (DaT) visualization using single photon emission-computed tomography (SPECT) imaging.

DaTScan differentiates between patients with and without a dopaminergic deficit.

DaTScan is a potential adjunct in the diagnosis of Parkinsonian symptoms.

DaTScan does not differentiate Parkinsonian Syndromes.

FDA indicates United States Food and Drug Administration.
DaTScan

Normal or Essential Tremor

PD or PD related disorders
DO MEDICINES CHANGE THE COURSE OF PARKINSON’S DISEASE?
Survival Prior to L-dopa

FIGURE 1. Obs = Observed survival in patients. Exp = Expected survival in general population. Observed survival is 215 Parkinson's patients who had onset and were first evaluated at Movement Disorder Clinic before 1/1/74 (Group IA) compared to expected survival in the general population matched for age, sex and the year of birth.

TIMELY LEVODOPA PROLONGS SURVIVAL IN PARKINSON'S DISEASE

FIGURE 2. Obs = Observed survival in patients. Exp = Expected survival in general population. Observed survival is 563 Parkinson's patients who had symptomatic onset after December 31, 1973 (Group IIb) compared to the expected survival in general population matched for age, sex and the year of birth.
Effects of Levodopa on Motor Function in Early PD

Pramipexole Improves Motor Function in Early PD

Mean change (%) from baseline at 31 weeks in UPDRS III (motor) scores

- Pramipexole (n = 163): 25*%
- Placebo (n = 170): -6.9%

*P<.0001

Risk of Dyskinesia Depending on Initial Treatment

TEMPO: Maintenance of Effect on Total UPDRS over 6 month period

Mean Change from baseline in Total UPDRS

1 mg vs placebo
$P < 0.001$

* $P = .01$

Treatment Options

Preventive treatment
– No definitive treatment available

Symptomatic treatment
– Pharmacological
– Surgical

Non-motor management

Restorative—experimental only
– Transplantation
– Neurotrophic factors
Drug Classes in PD

Dopaminergic agents
- Levodopa
- Dopamine agonists

COMT inhibitors

MAO-B inhibitors

Anticholinergics
Amantadine

A2A Antagonist (newest Class)
Anticholinergics

Dopaminergic depletion → cholinergic overactivity
Initially used in the 1950s
Effective mainly for tremor and rigidity
Common agents (Start low, go slow):
- Trihexyphenidyl: 2-15 mg/day
- Benztropine: 1-8 mg/day
- Ethopropazine: 10-200 mg/day

Side effects:
- Dry mouth, sedation, delirium, confusion, hallucinations, constipation, urinary retention
Amantadine

Antiviral agent; PD benefit found accidentally
Tremor, bradykinesia, rigidity & dyskinesias
Exact mechanism unknown; possibly:
- enhancing release of stored dopamine
- inhibiting presynaptic reuptake of catecholamines
- dopamine receptor agonism
- NMDA receptor blockade

Side effects — autonomic, psychiatric
200-300 mg/day
NOW Long Acting Formulations: Gocovri, Osmolex
Carbidopa/Levodopa (Sinemet)

Most effective drug for parkinsonian symptoms
First developed in the late 1960s; rapidly became the drug of choice for PD
Large neutral amino acid; requires active transport across the gut-blood and blood-brain barriers
Rapid peripheral decarboxylation to dopamine without a decarboxylase inhibitor (DCIs: carbidopa, benserazide)

Side effects: nausea, postural hypotension, dyskinesias, motor fluctuations
Diagram of LD Metabolism

www.wemove.org
# Levodopa/Carbidopa Formulations

<table>
<thead>
<tr>
<th>Formulation</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Immediate Release</strong></td>
<td>20-40 min</td>
<td>2-4 hr</td>
</tr>
<tr>
<td>10/100, 25/100, 25/250,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parcopa 10/100,25/100</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| **Controlled Release**      | 30-60 min | 3-6 hr   |
| 25/100, 50/200              |           |          |

| **“Liquid levodopa”**       | 10-20 min | 0.5-1 hr |
| (dissolved tablets)         |           |          |
Carbidopa/Levodopa

IPX066 (Rytary)- True long acting

Cabidopa/Levodopa Enteral Suspension – (CLES or DUOPA)
Inhaled L-Dopa

On Demand therapy for Off Episodes

Can be used up to 5 X a day

In clinical studies patients used on average 2X a day

99% of patients were able to use to deliver therapy in the Off State

Bypasses the GI system
Selegiline

- Irreversible MAO-B inhibitor
- Clinically active by inhibiting dopamine metabolism in brain
- Dosage: 5 mg at breakfast and lunch
- **Side effects:** insomnia, hallucinations, nausea (rarely), orthostatic hypotension
- Potential interactions with tricyclics and SSRI antidepressants (cheese reaction): uncommon
Rasagiline

• 1mg daily more potent?
• Safer?
• Increases on time up to 2hrs daily?
• Delay need for Levodopa?
• Neuronal Protection?? : TEMPO, ADAGIO TRIALS
Safinamide

- Reversible MAO-B inhibitor
- NMDA Blockade for Dyskenesias??
- Once Daily
COMT Inhibitors

- Newer class of antiparkinsonian drugs: tolcapone, entacapone (Opicapone only available in Europe)
- MOA similar to dopa decarboxylase inhibitors
- Potentiate LD: prevent peripheral degradation by inhibiting catechol O-methyl transferase
- Reduces LD dose necessary for a given clinical effect
- Helpful for both early and fluctuating Parkinson’s disease
- May be particularly useful for patients with “brittle” PD, who fluctuate between off and on states frequently throughout the day
Diagram of LD Metabolism
Tolcapone

- First COMT inhibitor licensed in the U.S.
- 100 mg TID or 200 mg TID
- Reduced LD dosage by 12%, improved motor fluctuations by 14% in non-fluctuating pts
- Reduced LD dosage by 30%, and on time increased from 1.7 to 2.9 hrs/day in fluctuating pts
- **Side effects:** Diarrhea, OH, dyskinesias, confusion
- Acute fulminant hepatic necrosis
  - 3/60,000+
  - FDA warning prevents use unless alternative therapy unsuccessful
  - liver monitoring every 2 weeks for a year and less frequently thereafter
Entacapone (Comtan®)

- Dosage: 200 mg w/each levodopa dose
- Parkinson’s Study Group 1997: Increased on time by 5%, more in pts w/least on time
- Rinne et al., 1998: Increased on time by ~10%; decreased levodopa
- Diarrhea, dopaminergic SEs
Levodopa/carbidopa/entacapone

- Dosage: 50/12.5/200, 100/25/200, 150/50/200
- Dosing is similar to taking traditional levodopa/carbidopa and entacapone
- Do not give more than one tablet of triple combination (TC) therapy at one time
- Side effect profile similar to these agents alone
- Appropriate for de novo and advancing PD patients less dyskinesias later on?
Dopamine Agonists: Distinguishing Features

• Directly stimulate dopamine receptors
• No metabolic conversion; bypasses nigrostriatal neurons
• No absorption delay from competition with dietary amino acids
• Longer half-life than levodopa
• Monotherapy or adjunct therapy
• May delay or reduce motor fluctuations & dyskinesias associated with levodopa??
## DAs: Receptor Effects

<table>
<thead>
<tr>
<th></th>
<th>D1</th>
<th>D2</th>
<th>D3</th>
<th>D4</th>
<th>D5</th>
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<tbody>
<tr>
<td><strong>Ergot</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Bromocriptine</td>
<td>-</td>
<td>++</td>
<td>++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Cabergoline</td>
<td>0</td>
<td>+++</td>
<td>?</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>Lisuride</td>
<td>+</td>
<td>++</td>
<td>?</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>Pergolide (Permax)</td>
<td>+</td>
<td>+++</td>
<td>++++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Non-Ergot</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pramipexole (Mirapex)</td>
<td>0</td>
<td>++</td>
<td>++++</td>
<td>++</td>
<td>?</td>
</tr>
<tr>
<td>Ropinirole (Requip)</td>
<td>0</td>
<td>++</td>
<td>++++</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>Apomorphine</td>
<td>++</td>
<td>++</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Neurology* 1998; 50(suppl 3)
## DA Pharmacokinetics and Dosage

### (monotherapy)

<table>
<thead>
<tr>
<th>Drug</th>
<th>T1/2</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bromocriptine (Parlodel)</td>
<td>6 hr</td>
<td>7.5-30 mg/day</td>
</tr>
<tr>
<td>Cabergoline</td>
<td>65+ hr</td>
<td>2-5 mg/day</td>
</tr>
<tr>
<td>Lisuride</td>
<td>2-4 hr</td>
<td>1-5 mg/day</td>
</tr>
<tr>
<td>Pergolide (Permax)</td>
<td>12-27 hr</td>
<td>1.5-12 mg/day</td>
</tr>
<tr>
<td>Pramipexole (Mirapex)</td>
<td>8 hr</td>
<td>1-4.5 mg/day</td>
</tr>
<tr>
<td>Ropinirole (Requip)</td>
<td>4 hr</td>
<td>3-24 mg/day</td>
</tr>
<tr>
<td>Apomorphine injectable</td>
<td>40 m</td>
<td>1-10 mg/day</td>
</tr>
<tr>
<td>Rotigotine (Neupro)</td>
<td>24 hr patch</td>
<td>4-8 mg/day</td>
</tr>
</tbody>
</table>
DAs: Common Adverse Effects

Nausea, vomiting
Dizziness, postural hypotension
Headache
Dizziness
Drowsiness & somnolence
Dyskinesias
Confusion, hallucinations, paranoia
Erythromelalgia; pulmonary & retroperitoneal fibrosis; pleural effusion & pleural thickening; Raynaud’s phenomena. May be more common with ergotoline DA’s
Apomorphine

- D1/D2 agonist
- Parenteral delivery - **Subcutaneous injection only in US** (s.c., i.v., sublingual, intranasal, rectal)
- Rapid “off” period rescue (10-20 minutes)
  - 2-10 mg s.c.; pen injection systems
- Treatment of unpredictable, frequent motor fluctuations, Morning Akenesia
  - continuous s.c. infusion via mini-pump
- SE: nausea, vomiting, hypotension
  - trimethobenzamide 300 mg t.i.d.
  - domperidone 20 mg t.i.d.; not available in U.S.
Sublingual Apomorphine

Still in Clinical Trials: Near future?
On Demand treatment for “Off Episodes”
Not FDA approved yet
Pumps

LCIG/CLES

Apomorphine Pump (Not in US)
Dopamine Agonist Patch

- Rotigotine transdermal patch
- Once a day patch (24hrs)
- Same side effect profile as DA
- Long acting DA, continuous
- For PD and RLS
Istradefyline 20mg or 40mg once daily

Increase On Time and Decrease Off Time

Good Tolerability

Works by Inhibiting Indirect Pathway

Side effects: Dyskenesias, Dizziness, Constipation, Nausea, hallucinations, insomnia
NON-MOTOR Treatments

Orthostatic Hypotension (nOH or Neurogenic Orthostatic Hypotension)
- Droxidopa (Northera)
- Off label: Midodrine, fludrocortisone, pyridostigmine
- Conservative measures (Water, salt, compression)

Hallucinations and Delusions In Parkinson’s Disease Psychosis
- Pimavanserin (Nuplazid)
- Clozaril
- Off label: Seroquel, etc

Sialorrhea (Drooling)
- Xeomin and Myobloc (Botulinium Toxins FDA Approved)
NON-MOTOR Treatments

Mild Cognitive Impairment or Dementia
- Rivastigimine (Patch)
- Donezepil

Rem Behavioral Sleep Disorders (RBD)
- Clonazepam
- Melatonin

Constipation, Urinary Urgency, Sexual Dysfunction
- Challenge and multifactorial
- need more studies
Deep Brain Stimulation (DBS)

High frequency, pulsatile, bipolar electrical stimulation
Stereotactically placed into target nucleus
Can be activated and deactivated with an external magnet
Exact physiology unknown, but higher frequencies mimic cellular ablation, not stimulation
Medtronic, Abbot and Boston Scientific
– Significant advances in Neuromodulation therapy
Motor Fluctuations

“Wearing off”: re-emergence of symptoms prior to the next scheduled levodopa (LD) dose

“On/off” phenomenon: unpredictable fluctuations of periods of good mobility and function followed by periods of poor symptom control

Delayed-“on” responses: dose takes longer to improve symptoms than previously

Dose failure: dose does not provide usual improvement in symptoms
<table>
<thead>
<tr>
<th>Motor</th>
<th>Nonmotor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tremor</td>
<td>Abdominal discomfort</td>
</tr>
<tr>
<td>Bradykinesia</td>
<td>Akathisia (uncontrollable motor restlessness)</td>
</tr>
<tr>
<td>Muscle cramping</td>
<td>Anxiety</td>
</tr>
<tr>
<td>Difficulty getting out of a chair</td>
<td>Cloudy mind, dullness of thinking</td>
</tr>
<tr>
<td>Reduced dexterity</td>
<td>Drenching sweats</td>
</tr>
<tr>
<td>Stiffness</td>
<td>Drooling</td>
</tr>
<tr>
<td>Balance problems</td>
<td>Dysphagia</td>
</tr>
<tr>
<td>Weakness</td>
<td>Dyspnea</td>
</tr>
<tr>
<td>Slowness in early morning/during the night</td>
<td>Facial flushing</td>
</tr>
<tr>
<td></td>
<td>Fatigue</td>
</tr>
<tr>
<td></td>
<td>Irritability</td>
</tr>
<tr>
<td></td>
<td>Mood changes</td>
</tr>
<tr>
<td></td>
<td>Numbness</td>
</tr>
<tr>
<td></td>
<td>Pain</td>
</tr>
<tr>
<td></td>
<td>Tightening sensations</td>
</tr>
<tr>
<td></td>
<td>Tingling sensations</td>
</tr>
</tbody>
</table>

Stacy M et al. Mov Disord. 2005;20:726-733
Future

New Definition for PD
Gene therapies
- AV Viruses GAD, AADC, BDNF, GDNF
Subcutaneous pumps L-Dopa
Opicapone: COMT Inhibitor
Sublingual Apomorphine
Protein Kinase Inhibitors
  - Nilotinib
  - Bosutinib
  - K0706 and others
New formulations of L-Dopa
  - IPX023, Accordin Pill
Constipation
  - On going studies Enterin
CONCLUSION

- We have **multiple medications** to help improve quality of life of individuals with PD.
- “Polypharmacy” is common but it **may help** with some of the motor complications and **minimize extremes** of more than one medication.
- **Every patient is different**, what works for one may not necessarily work for another.
- **Team effort approach** between patient and physician to optimize therapy and quality of life.
- Medications are only part of a **multi-disciplinary approach** necessary in order to treat Parkinson’s disease.
SLIDES
You can download slides via the link in the CHAT.

CEU
For Health Professionals interested in 1 free CEU, you can click on the link in the CHAT to access the application.
Valid until May 14, 2020
Every day with Parkinson’s disease can feel different.

We want to encourage the PD community to take actionable steps to #Plan4PD.

Download this calendar and more at Parkinson.org/Awareness.
For Tomorrow: Mapping the Future

PD GENEration: Mapping the Future of Parkinson’s Disease is the first-of-its-kind national initiative that offers free genetic testing for clinically relevant PD-related genes and free genetic counseling to help participants better understand their results.

To learn more, visit: Parkinson.org/PDGENEration
2020 Care Partner Summit | Cumbre Para Cuidadores

Planning for the Unpredictable Path of Parkinson's Caregiving

An Online Event | May 16th, 2020
12:00pm – 3:30pm EST

For more information and to register:
Parkinson.org/Summit
New Parkinson’s Foundation & AARP Webinar Series

Answering Your PD Questions with AARP: From Newly Diagnosed to Caregiver Resources

Thursday, April 30th
3pm – 4pm ET

Register here!

Amy Goyer
AARP’s national family and caregiving expert and author

Nina Browner, MD
Bryson Distinguished Associate Professor of Neurology and Director at the University of North Carolina
Expert Briefing Series

Newly Diagnosed: Living Your Best Life with Parkinson’s

Tuesday, June 9th | 1:00pm ET
Register [here](#)!
Thank You to our Sponsor!
Resources

National Helpline
Specialists answer calls about all aspects of Parkinson’s in addition to helping you locate your local PD trained allied health professional therapist.
1-800-4PD-INFO
Helpline@Parkinson.org
Mon- Friday 9 am to 8 pm ET

Podcast: Substantial Matters
New episodes every other Tuesday featuring Parkinson’s experts highlighting treatments, techniques and research.
Parkinson.org/Podcast

Episode 34: Dr. Michael Okun
New Pathways and Drug Development

Fact Sheets and Publications
Get the resources and information you need to start living a better life with Parkinson’s.
Parkinson.org/Library

Aware in Care Kit
Includes tools and information for people with PD to share with hospital staff during a planned or emergency hospital stay.
Parkinson.org/Awareincare