Innovations in the Treatment of Parkinson's Disease and Demystifying Deep Brain Stimulation (DBS)

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Best Practice Update: Parkinson's Disease Treatment Options

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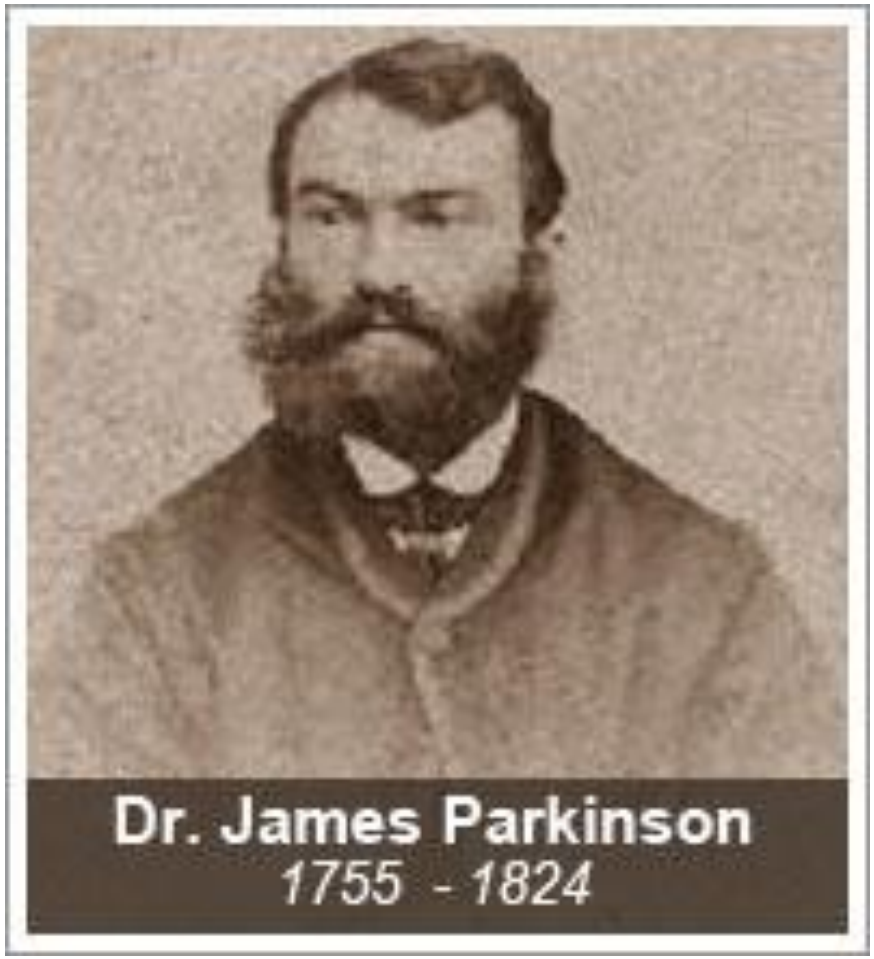
Objectives

1. To review the history and epidemiology of PD
2. To learn the cardinal motor symptoms of PD
3. To review the UK Brain Bank criteria for diagnosing PD
4. To appreciate the Pre-Motor phase of PD, and its involvement of diverse systems
5. To provide an update on medical management of PD
6. To appreciate the multitude of non-motor symptoms and learn strategies for clinical management to improve QOL
7. To learn the most common forms of genetic parkinsonism
8. To review the 10 Quality Measures for PD put forth by the American Academy of Neurology

Parkinson’s Disease

Essay on the Shaking Palsy, 1817



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SHAKING PALSY.

CHAPTER I.

DEFINITION—HISTORY—ILLUSTRATIVE CASES.

SHAKING PALSY. (*Paralysis Agitans.*)

Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace: the senses and intellects being uninjured.

The term Shaking Palsy has been vaguely employed by medical writers in general. By some it has been used to designate or-

Epidemiology

- Affects 1-2 million people in the United States
- Likelihood of developing PD increases with age
- Average age of onset is 62.4 years
- Age is the strongest risk factor
- Men > Women, but only for age >60
- Cultural Disparity: Higher incidence in whites than African Americans or Hispanics
  - Due to true biological differences or barriers to healthcare (education, cultural beliefs about health and aging)?

Taylor KS, Cook JA, Counsell CE. Heterogeneity in male to female risk for Parkinson's disease. J Neurol Neurosurg Psychiatry 2007;78(8):905-906.

Dahodwala N, Siderowf A, Xie M, et al. Racial differences in the diagnosis of Parkinson's disease. Mov Disord 2009;24(8):1200-1205.

Parkinson's Motor Symptoms

- Rest tremor
- Bradykinesia and slowness of ADL's
- Rigidity and freezing in place
- Stooped, shuffling gait
- Decreased arm swing while walking
- Difficulty arising from a chair
- Micrographia
- Hypomimia (lack of facial expression)
- Difficulty turning in bed
- Postural instability

UK PD Society Brain Bank Clinical Diagnostic Criteria

- Inclusion Criteria (need 2 of these 4)
  - **Bradykinesia** (and at least one of the following):
  - Muscular rigidity
  - 4 Hz to 6 Hz tremor
  - Postural instability (not caused by primary visual, vestibular, cerebellar, or proprioceptive dysfunction)

UK PD Society Brain Bank Clinical Diagnostic Criteria

- Exclusion Criteria
  - Hx of repeated strokes with stepwise progression of parkinsonian features
  - Hx of repeated head injury
  - Hx of definite encephalitis
  - Oculogyric crisis
  - Neuroleptic treatment at onset of symptoms
  - More than one affected relative
  - Sustained remission
  - Strictly unilateral features after 3 years

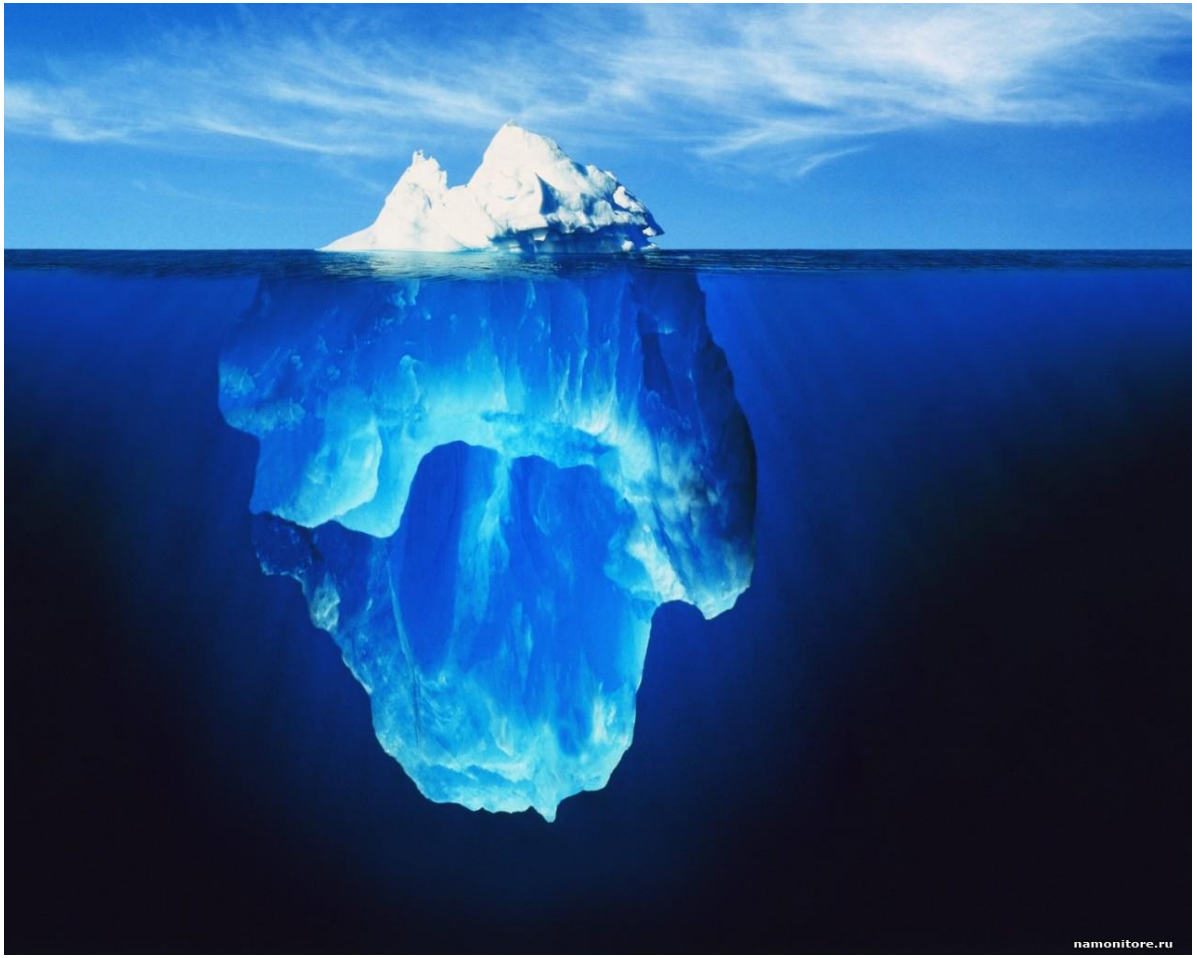
UK PD Society Brain Bank Clinical Diagnostic Criteria

- Exclusion Criteria (continued)
  - Supranuclear gaze palsy
  - Cerebellar signs
  - Early, severe autonomic involvement
  - Early, severe dementia or apraxia
  - Babinski sign
  - Presence of cerebral tumor or communicating hydrocephalus on CT scan
  - Negative response to large doses of levodopa
  - MPTP exposure

UK PD Society Brain Bank Clinical Diagnostic Criteria

- Supportive Criteria (need 3 or more for diagnosis of “Definite” PD)
  - Unilateral onset
  - Rest tremor present
  - Progressive disorder
  - Persistent asymmetry
  - Excellent response (70% to 100%) to levodopa
  - Severe levodopa-induced dyskinesias
  - Levodopa response for 5 years or more
  - Clinical course for 10 years or more

Parkinson’s Disease



Parkinson's Non-Motor Symptoms

- Anosmia (decreased sense of smell)
- Depression / anxiety
- Drooling (dysphagia)
- Blepharitis
- Hypophonia (low vocal volume)
- Postural lightheadedness (orthostatic hypotension)
- Sudomotor dysregulation (abnormal sweating)
- Sleep disturbance (RBD, OSA, RLS, PLMS, fragmentation)
- Constipation
- Urinary frequency / urgency
- Male erectile dysfunction
- Painful foot cramps (dystonia)
- Bursitis, "frozen shoulder"

Braak and Braak Pathologic Staging

- Clinicopathologic staging system for Lewy body disease-associated changes.
- Predictable topography of progression of Lewy body disease in the CNS
- Begins in olfactory structures and medulla, progresses rostrally from the medulla to the pons, then to midbrain and substantia nigra, limbic structures, and neocortex.
- Symptoms resulting from degeneration of olfactory and pontomedullary structures begin many years before prominent nigral degeneration and the typical Parkinsonian features.

Braak H, Del Tredici K, et al. 2003. Staging of brain pathology related to sporadic Parkinson's disease. Neurobiol Aging 24 (2):197-211.

Pre-Motor Phase of PD

Pre-Motor Symptoms	Brain Structures Involved
Olfactory Loss - Hyposmia in 90%, impairment in odor detection, identification, discrimination	Olfactory bulb, anterior olfactory nucleus, amygdala, perirhinal cortex
Dysautonomia -GI: gastroparesis, **constipation** (yrs prior) -Urinary: frequency and urgency -Sexual: erectile dysfunction	Dorsal nucleus of the vagus, sympathetic ganglia, enteric and abdominopelvic plexuses (amygdala, intermediolateral column of spinal cord)
Mood -Depression -Anxiety	Locus ceruleus, raphe nuclei (amygdala, mesolimbic, mesocortical cortex)
Sleep -**REM behavior disorder (most common)** -Excessive daytime sleepiness -Insomnia / sleep maintenance	Nucleus subceruleus, pedunculopontine nucleus (thalamus, hypothalamus)

Update on the Medical Management of PD

- Levodopa (various formulations)
- Catechol-o-methyltransferase Inhibitors
- Monoamine Oxidase Type B Inhibitors
- Dopamine Agonists
- Other agents

Formulations of Levodopa

- Mainstay of antiparkinsonian therapy since the late 1960's
- Combined with a peripheral aromatic amino acid decarboxylase inhibitor to block conversion of dopamine outside the CNS
  - In the US: Carbidopa (Lodosyn when sold separately)
  - Overseas: Benserazide

Levodopa Effects

- Positive effects: dramatic reduction in PD symptoms within a few days
- Negative effects:
  - Early stage
    - Nausea, vomiting
    - Drowsiness
    - Dizziness, hypotension
  - Later stages
    - Hallucinations
    - Dyskinesias

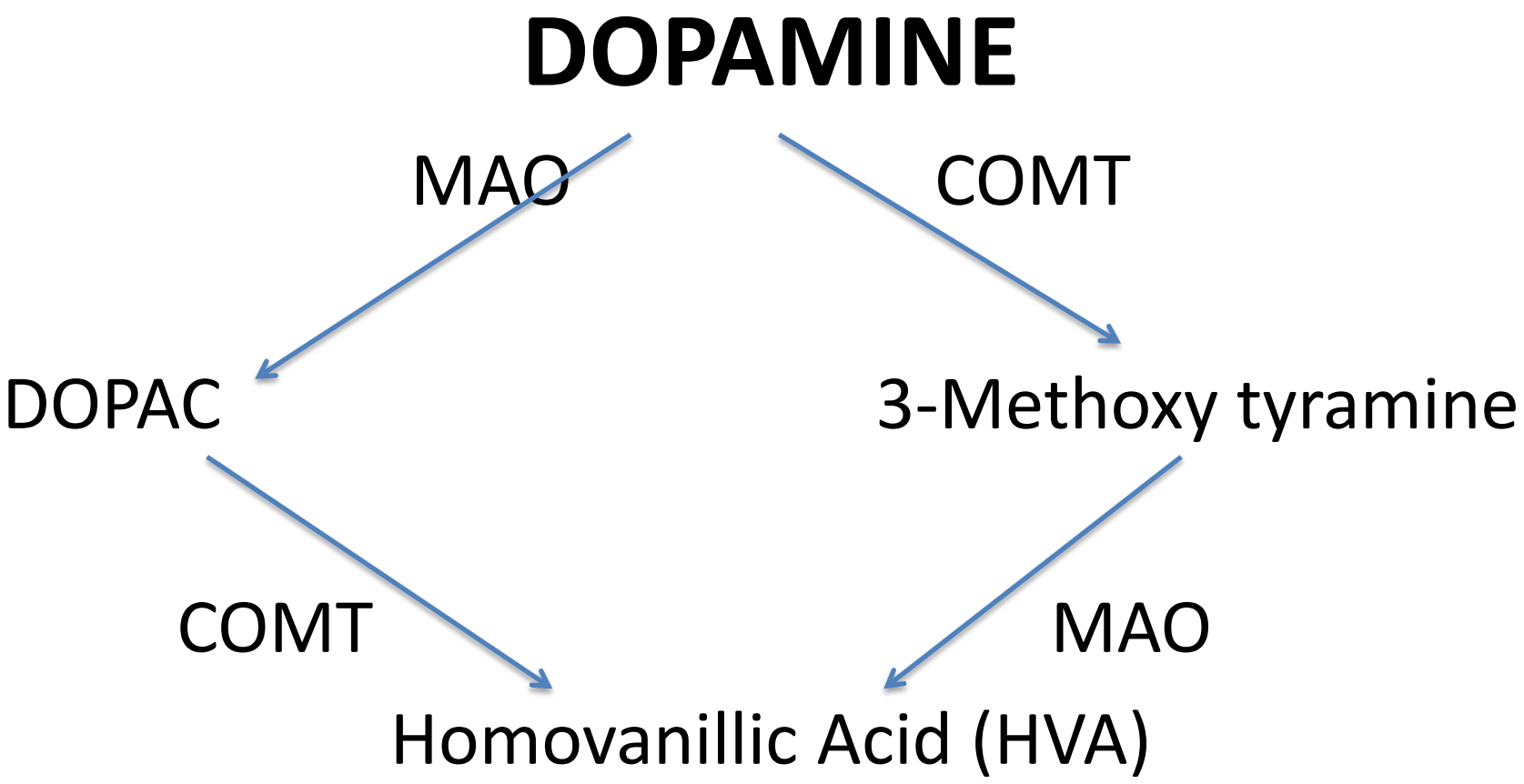
Long-Term Variability with Levodopa

- Levodopa must be converted by neurons to dopamine
- These neurons degenerate over time
- Disease progression results in decreased predictability of dopamine levels

Levodopa Complications

- Motor Fluctuations
  - Dyskinesias
  - Wearing off (shortened duration of effect)
- Strategies to combat these:
  - Shorten dosing interval
  - Add an inhibitor of levodopa/dopamine catabolism
- Non-motor complications
  - Treat each directly

Dopamine Degradation



Catechol-o-methyltransferase (COMT) Inhibitors

- COMT converts levodopa to the inactive 3-O-methyldopa
- Blocking COMT increases brain levels of levodopa and dopamine, and provides longer duration of action
- TOLCAPONE (Tasmar)
  - Risk of fatal hepatic failure
- ENTACAPONE (Comtan)

Entacapone (Comtan)

- Shown to reduce OFF time (Parkinson's Study Group, 1997)
- 200mg, given with each dose of carbidopa/levodopa
- Maximum daily dose of entacapone: 1600mg
- Combination formulation of entacapone + levodopa + carbidopa = Stalevo
- Side effects: orange discoloration of urine and bodily fluids, diarrhea, levodopa potentiation

Monoamine Oxidase Type B Inhibitors

- Increase the half-life of dopamine by blocking catabolic pathways
- Type B enzyme is in the brain, so preferentially affects PD
- SELEGILINE (Eldepryl, Deprenyl) 5mg BID
- SELEGILINE orally disintegrating tab (Zelapar)
  - Less first pass hepatic metabolism
  - Dosed 1.25 to 2.5mg per day

Monoamine Oxidase Type B Inhibitors

- RASAGILINE (Azilect) 1 mg per day
  - Studied extensively to show a reduction in disease progression, but unclear how much of any putative disease-modifying effect is related to improvement in symptoms
- Side effects of MAO-B inhibitors
  - Potentiating dopamine side effects
  - Tyramine effects (potentially fatal tachycardia and hypertensive crisis)
    - Limit tyramine in diet (fermented foods)
    - Do not combine with decongestants (pseudoephedrine, phenylephrine) and certain narcotics

Dopamine Agonists

- Ergot Derived (vascular toxicities)
  - ~~Bromocriptine (Parlodel)~~
  - ~~Pergolide (Permax), linked to valvular heart disease~~
- Non-Ergot Derived
  - Pramipexole (Mirapex)
  - Ropinirole (Requip)
  - Rotigotine transdermal (Neupro)

Two large multiyear studies showed a reduced incidence of dyskinesias with initial agonist therapy compared with initial levodopa TID therapy (Parkinson's Study Group, 2000; Rascol et al, 2000)

Benefits of Dopamine Agonists

- Longer half-lives than levodopa
  - More convenient dosing in some situations
  - Fewer motor complications
- FDA approved for restless legs syndrome (RLS)
  - Often co-existent in PD
- Once-a-day formulations are available
  - Requip XL
  - Mirapex ER
  - Neupro patch

Side Effects of Dopamine Agonists

- Somnolence, Sleep Attacks
- Compulsive behaviors
  - Impulse Control Disorder
- Peripheral edema
- Nasal congestion
- Potentiating levodopa effects
  - Hypotension
  - Dyskinesias

A Unique Dopamine Agonist

- Apomorphine (Apokyn)
  - For use in advanced Parkinson's disease
  - Injected subcutaneously by the patient
  - For treatment of acute off periods despite treatment with existing antiparkinsonian therapy
  - Benefit lasts 1 hour

Other Agents

- Amantadine (Symmetrel)
  - Partial dopamine agonist and partial NMDA receptor antagonist
  - Dosed as 100 mg 1-4 times per day
  - Usefulness has been replaced by levodopa
  - Good anti-dyskinetic effect in advanced PD
  - Side Effects
    - Livedo reticularis
    - Confusion, hallucinations, depression, anxiety

Other Agents

- **Anticholinergics**
  1. Trihexyphenidyl (Artane)
  2. Benztropine (Cogentin)

  - Used to treat PD since the 1940's
  - Preferentially treat tremor
  - Side effects: USE WITH CAUTION in the elderly!
    - Cognitive dysfunction
    - Constipation, urinary retention
    - Blurry vision
    - Dry mouth

Duodopa

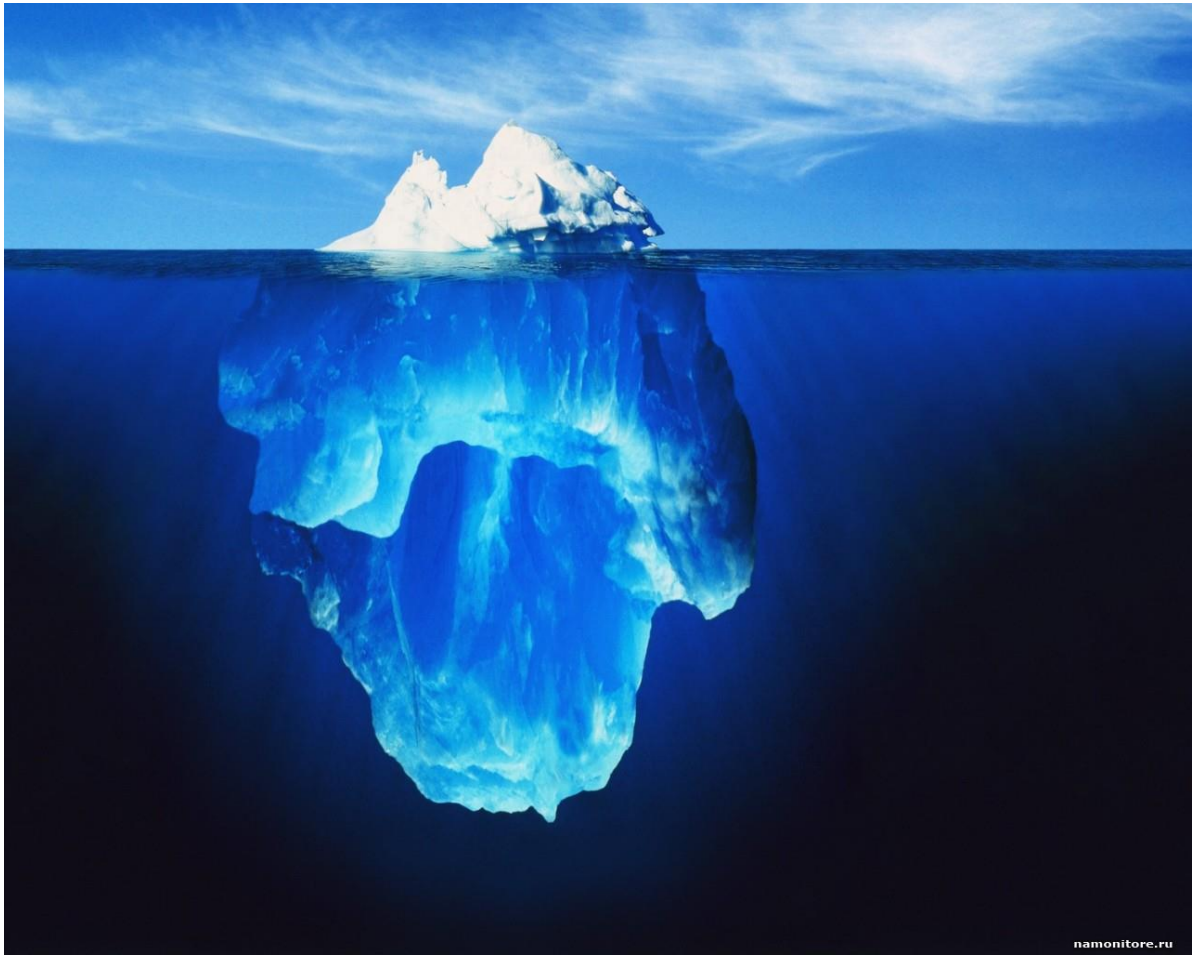
- Not yet available in the US
- New gel formulation of carbidopa/levodopa
- Delivery via novel intra-intestinal pump
- Surgically inserted and programmed to deliver doses at specific times (like insulin pump)
- External controller makes dose adjustments non-invasively
- More constant blood levels minimize levodopa motor complications

Exercise in PD

- Critical component: non-negotiable
- Components of daily regimen:
  1. Stretching
  2. Conditioning: stationary bicycle, walking outdoors, treadmill (caution re: fall risk), walking pool laps, elliptical, tai chi, yoga, dance (www.danceforparkinsons.org) .
- Physical Therapy, LSVT BIG
- Symptom management
  - Improvement in gait, balance, flexibility, coordination
  - Decrease in falls
- Neuroprotection: slowing disease progression

Ebersbach G, Comparing exercise in Parkinson's disease: the Berlin LSVT BIG study. Mov Disord, V25:12 2010, 1902-1908.

Non-Motor Symptoms



Improving Quality of Life

Depression
Anosmia, Weight Loss
Blepharitis
Shoulder Pain
Dystonia
Sleep Disturbance
Sexual Dysfunction
Constipation, Bladder dysfunction
Hypophonia, Dysphagia
Dementia, Psychosis
Orthostatic Hypotension

Many of the drugs we will discuss are off-label for these symptoms.

Mood Disorders in PD

- Depression affects 50-75% of PD patients
- Secondary to underlying neuroanatomical degeneration, rather than a reaction to psychosocial stress and disability.
- Based on changes in central serotonergic function and specific cortical and subcortical pathways.
- Depression, anxiety precede motor symptoms of PD by ~6 years (correlates with PET studies).

McDonald WM, Richard IH, DeLong MR. Prevalence, etiology, and treatment of depression in Parkinson's disease. Biol Psychiatry. 2003 Aug 1;54(3):363-75.

Rickards H. Depression in neurological disorders. J Neurol Neurosurg Psychiatry 2005; 76:i48-i52.

Depression in PD

- Challenges to diagnosis include similarities in clinical signs to PD itself.
- Look for:
  - pervasive low mood with diurnal variation for at least two weeks
  - Early morning awakening
  - Pessimistic thoughts about the world, themselves, the future
  - Suicidal ideation

Rickards H. Depression in neurological disorders. J Neurol Neurosurg Psychiatry 2005; 76:i48-i52.

Management of Depression in PD

- Rating scales have reduced validity due to emphasis on somatic / vegetative symptoms.
- Medication Management:

1. Tricyclic Antidepressants
   - Amitriptyline and Nortriptyline
   - Review of the literature by Miyasaki et al, 2006
   - Use with caution due to anticholinergic effects, which are problematic in elderly

Management of Depression in PD

2. SSRI's

- Fluoxetine, paroxetine, sertraline, fluvoxamine, citalopram, escitalopram
- Side effects: nausea, GI disturbance, sexual dysfunction, exacerbation of RBD, withdrawal syndrome in elderly.

3. SNRI

4. NDRI

Potential for Serotonin Syndrome

- There is a potential for Serotonin Syndrome when combining MAO inhibitors with any antidepressant.
- Risk is lower with MAO-B than MAO-A, but must discuss with patients.
- Symptoms: acute mental status changes, autonomic dysfunction, myoclonus, hyperreflexia.
- Requires proper clinical judgment, patient education, and close monitoring.

Anosmia and Depression

Loss of sense of smell → Loss of taste

Decreased appetite

Decreased PO intake

Weight loss

Blepharitis

- “Dry Eye,” chronic eyelid inflammation and conjunctival injection
- Increased bacteria on surface of dry eyes
- Blepharitis in PD is a function of decreased blink rate (facial bradykinesia)
- Blepharitis often predates PD diagnosis by many years
- Management: High viscosity lubricant eye drops at bedtime (ex: Systane Gel Drops)

Shoulder Pain

- Often the presents prior to the diagnosis of PD
- Typically unilateral, on the side with more prominent Parkinsonism
- Rigidity and bradykinesia (including decreased arm swing) leads to immobility and subsequent shoulder dysfunction and discomfort.
- Bursitis, tendonitis, frozen shoulder, rotator cuff injury
- Physical Therapy
- BIG arms gait training

Stamey WP, Jankovic J. Shoulder pain in Parkinson's disease. Mov Disord 2007; 22:S247-8.

Dystonia

1. Unrelated to treatment
   - Kinesiogenic foot dystonia is a hallmark of early onset PD. Typically involves tibialis posterior and toe flexors. Painful and impairs gait/balance.
   - Blepharospasm, Torticollis
   - Management: targeted botulinum toxin injection
2. Related to therapy
   - Peak-dose, diphasic, and off-dystonia

Tolosa E, Compta Y. Dystonia in Parkinson's disease. J Neurol 2006 Dec; 253 Suppl 7:V117-13.

Sleep Disturbance

- **REM Behavior Disorder**
  - Rapid eye movement (REM) sleep behavior disorder
  - Parasomnia with vivid dreams and dream enactment behavior during REM sleep.
  - Findings from animal and human studies suggest that dysfunction in REM sleep and motor control circuitry in pontomedullary structures cause RBD symptoms
  - Degeneration of these structures might explain the RBD years or decades before the onset of motor symptoms in people who develop PD

Boeve BF. Idiopathic REM sleep behaviour disorder in the development of Parkinson's disease. Lancet Neurol. 2013 May;12(5):469-82.

REM Behavior Disorder

- Sequela of untreated RBD
  - Excessive daytime sleepiness
  - Physical injury to patient or bed partner
  - Chronic sleep deprivation leads to exacerbation of daytime motor symptoms
- Management of RBD
  - Clonazepam (Klonopin) 0.25-1.0 mg QHS: off label
  - Melatonin 3-5mg tabs, up to 10mg QHS

Sleep Disturbance

- Sleep Fragmentation
  - Clonazepam (Klonopin) 0.25-1.0 mg QHS: off label
  - Melatonin 3mg (1-3 tabs) QHS
- Insomnia
  - Mild sedatives are well-tolerated in the non-demented patient
  - Zolpidem (Ambien), Zaleplon (Sonata), Eszopiclone (Lunesta), Ramelteon (Rozerem)
  - None are FDA-approved for use in PD

Sleep Disturbance

- Obstructive Sleep Apnea
  - CPAP, BiPAP
- Restless Legs Syndrome (RLS) &

  Periodic Limb Movements in Sleep (PLMS)
  - Dopamine agonists
- Diagnosis: Formal Nocturnal Polysomnography
  - Must specify application of EMG leads on the extremities in PD (RBD, RLS, PLMS)

Excessive Daytime Sleepiness

- Identify underlying sleep disorder and treat directly
- Daytime Sleep Restriction: minimize naps
- Sleep hygiene
  - Fixed bedtime and awakening time
  - Avoid alcohol, caffeine, or heavy/spicy/sugary foods 4-6 hours before bedtime
  - Bed is for sleeping (not eating, reading, office work)
- Modafinil (Provigil)
  - FDA-approved “to improve wakefulness in adult patients with excessive sleepiness associated with narcolepsy, OSA, and shift-work sleep disorder;” NOT PD.

Sexual Dysfunction

- Not correlated with disease severity
- Decreased libido
- Erectile dysfunction
  - Sildenafil (Viagra)
    - Watch for hypotension
  - Dopamine Agonists
- Hypersexuality
  - Associated with dopaminergic treatment
  - Linked to inhibition of prolactin secretion

Constipation

- One of the earliest signs of autonomic dysfunction
- Affects majority of PD pts (even described in the original 1817 essay)
- Stools become less frequent and difficult to pass due to delayed gastric emptying and slowed intestinal transit time
- Lewy bodies are found in intestines post-mortem

Management of PD Constipation

- Optimize hydration
- Well-balanced, high fiber diet (fruits, vegetables, prunes, bran cereal)
- Regular exercise
- Fiber supplements, bulk formers
- Stool softeners: Daily Colace
- Laxatives: Miralax 17g packet daily or QOD

Bladder Dysfunction

- Overactive bladder
  - Abnormal central control of urinary sphincter
  - Urgency, frequency, incontinence, nighttime urination
  - Depends
  - Comodes or urinals at bedside to prevent falls
  - Sphincter relaxants: Ditropan, Detrol, Vesicare
- Urinary Retention: Incomplete bladder emptying
  - Bladder sphincter dyssinergia or contractile weakness
  - Weak urinary stream, dribbling, leaking
  - Recurrent UTI's

Speech Disturbances

- Hypophonia
  - Limited vocal / pitch range
  - Low voice and volumes
  - Diminished respiratory support and coordination
- Speech deficits
  - Imprecise articulation
  - Accelerated rate
  - Decreased intelligibility

Speech Therapy

- Lee Silverman Voice Treatment (LSVT LOUD)
  - Best to initiate early
  - Goal is to think loud and “Speak LOUD!”
  - Systematic hierarchy of exercises stimulates largyngeal muscles and speech mechanism
  - improves respiratory, laryngeal, and articulatory function to maximize speech intelligibility
  - Intensive: 16 sessions per month

Dysphagia

- Drooling
  - Not due to excessive salivary production (salivary output in PD is normal or decreased)
  - A result of difficulty transporting saliva to posterior pharynx + decreased frequency of swallowing
  - Often exacerbated by forward-flexed posture
  - Socially embarrassing but not dangerous
  - Gum and hard candy trigger swallow reflex
  - Risks of anticholingerics are not worth the benefit
  - Atropine eye drops on the tongue minimizes systemic effects
  - Targeted botulinum toxin injection of the parotids

Dysphagia

- Difficulty swallowing in 50-80% of pts
- 90-100% show impaired swallowing on MBS or FEES
- Impaired pharyngeal peristalsis
- Restricted opening of the upper esophageal sphincter
- Lingual tremor
- Increased risk of aspiration and pneumonia
- Primary cause of mortality in PD
- Throat clearing, sensation of food “sticking” in the chest
- Often not improved by dopaminergic meds

Quigley E. Dysphagia in Parkinson’s disease. In: Parkinson’s disease: diagnosis and clinical management. Factor SA, Weiner WJ, editors. Demos Medical Publishing, 2002.

Dysphagia

- Speech and Swallow Therapy
  - Swallow techniques
    - Second swallow
    - Chin tuck
    - Straws
  - Food consistency
    - Thickened liquids
    - Softer food texture

Nausea and Bloating

- Levodopa effect
  - Peripheral dopaminergic stimulation
  - Treat with supplemental dopa-decarboxylase inhibitor
  - Lodosyn (carbidopa) 25mg with each Sinemet dose to achieve higher carbidopa:levodopa ratio
- Gastroparesis
  - Management of constipation
  - Small, more frequent meals

PD Dementia

- Acetylcholinesterase Inhibitors
  - Rivastigmine (Exelon)
    - FDA approved for PD dementia
    - Tablet, liquid, transdermal patch
  - Donepezil (Aricept)
    - FDA approved for Alzheimer's disease only
  - Galantamine (Razadyne)
    - FDA approved for Alzheimer's disease only
- Memantine (Namenda)
    - Chemically similar to Amantadine
    - FDA approved for Alzheimer's, under study for PD dementia

Psychosis in PD

- Hallucinations are typically visual, not auditory
- Paranoia
- Avoid CNS dopamine receptor antagonists
- No antipsychotics are FDA approved for hallucinations in PD
- FDA warns against use of antipsychotics in pts with dementia due to increased risk of death

Psychosis in PD (con't)

- Clozapine (Clozaril)
  - 12.5 mg to 25 mg BID
  - Risk of agranulocytosis requires frequent monitoring of WBC count
  - Somnolence
- Quetiapine (Seroquel)
  - Atypical neuroleptic with some antipsychotic efficacy data in PD clinical trials
  - 25 mg to 75 mg QD-BID; higher doses may worsen parkinsonism
  - Somnolence

Orthostatic Hypotension (OH)

- Feature of advanced PD, and some atypical parkinsonian syndromes (Multiple System Atrophy)
- Use CAUTION when using dopaminergic agents which can worsen OH
- Compression stockings
- Increase water and salt intake
- Rise slowly
- Raise head of bed
- Watch for SUPINE HYPERTENSION
- Monitor orthostatic vitals at every visit

Orthostatic Hypotension (OH)

- Midodrine (ProAmantine)
  - Agonist at peripheral alpha-1 adrenergic receptors
  - Increases systemic vascular resistance
  - 2.5 mg to 5 mg TID
- Fludrocortisone (Florinef)
  - 0.1 mg to 0.3 mg daily
  - Watch for excessive hypertension, edema

Prodromal Dysautonomia in PD

- Patients with RBD were followed annually in a prospective cohort established in 2004.
- Urinary, orthostatic, erectile, and constipation symptoms, and SBP drop from lying to standing were assessed annually.
- Estimated onset of autonomic dysfunction is ~11-20 years before diagnosis of PD. (SBP drop: 20.4 yrs, constipation: 15.3 yrs)
- SBP drop + ED + constipation = correct identification of PD 5 yrs prior to motor symptom diagnosis with sensitivity of 50-90%.

Postuma RB, Gagnon JF, Pelletier A, Montplaisir J. Prodromal autonomic symptoms and signs in Parkinson's disease and dementia with Lewy bodies. Mov Disord. 2013 May; 28(5):597-604.

Young-Onset Parkinson's Disease

- Onset before age 30 is rare
- Up to 10% of cases begin by age 40
- 10-15% have a strong family history
- Dystonia may be a presenting symptom

Familial Parkinsonism

- Autosomal Dominant PD
  - PARK 1 and PARK 4 account for 2% of AD PD
    - Mutations of the alpha-synuclein gene
  - PARK 8 (10% of familial cases)
    - Mutations in leucine-rich repeat kinase 2 gene (LRRK2)
- Autosomal Recessive PD
  - PARK 2 (50% of familial, 20% of “sporadic” YOPD)
    - Mutations in parkin gene, encoding ubiquitin E3 ligase
    - High incidence of dystonia

10 Quality Measures for PD Care

- Quality improvement in neurology: AAN Parkinson's disease quality measures. Report of the Quality Measurement and Reporting Subcommittee of the American Academy of Neurology. EM Cheng, S Tonn, R Swain-Eng, SA Factor, WJ Weiner, CT Bever, for the AAN PD Measure Development Panel. *Neurology* 2010; 75: 2021-2027.

The 10 Measures (and frequency of inquiry) are:

1. Annual PD diagnosis review (annually)
2. Psychiatric assessment (annually)
3. Cognition assessment (annually)
4. Query autonomic dysfunction (annually)
5. Query sleep disturbances (annually)
6. Query about falls (every visit)
7. PD rehab therapy options (annually)

The 10 Quality Measures (con't)

8. PD related safety issues counseling (annually)
9. Query about PD medication-related motor complications (every visit)
10. Review of PD medical and surgical treatment options (annually)

Questions?

- Thank You!