Shake, Rattle and Roll: Optimizing Care for Movement Disorders

UCDAVIS HEALTH SYSTEM

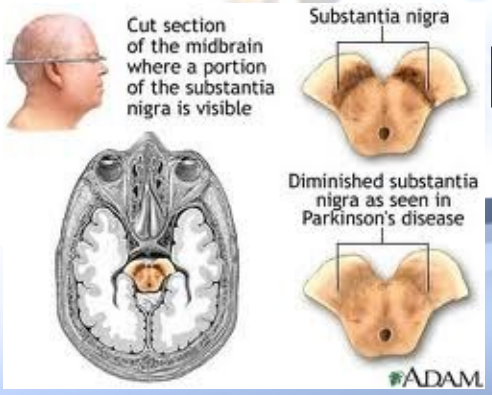
WORLD FEDERATION OF NEUROSCIENCE NURSES

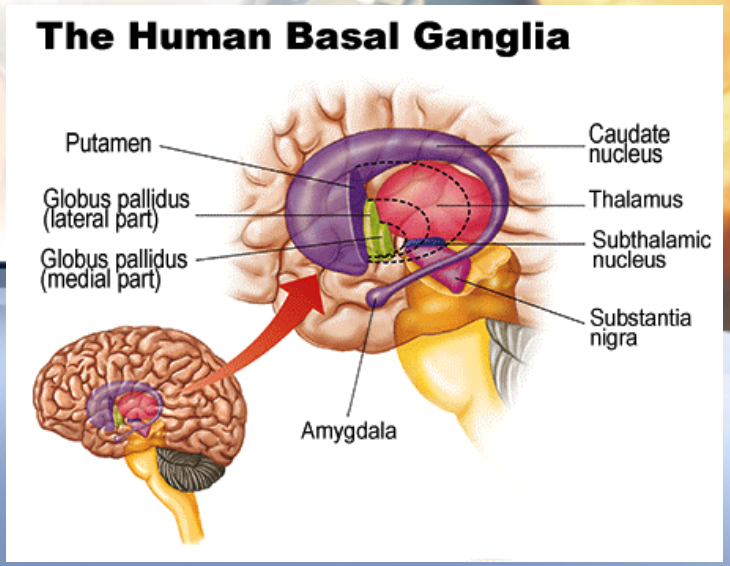
Parkinson's Disease

Objectives

1. To learn the cardinal motor symptoms of PD
2. To provide an update on medical and surgical management of PD
3. To appreciate the multitude of non-motor symptoms and learn strategies for clinical management to improve QOL
4. To review the 10 Quality Measures for PD put forth by the American Academy of Neurology

Pathology



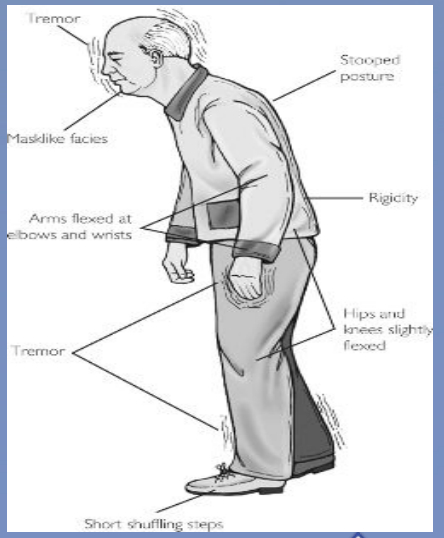


- Caused by slow, progressive depletion of dopamine-producing cells in the substantia nigra (SN)
  - Nonmotor sx secondary to loss of these neurons outside the SN involving dopamine and acetylcholine

- 60% loss these neurons by the time clinical manifestations emerge.
  - Pre-motor symptoms can occur months-years before motor symptoms:
    - Constipation, REM behavior disorder, depression, decreased smell

Cardinal features

- Resting tremor (>70%)
  - Pill-rolling rest tremor (re-emergent), intermittent, sensation of internal tremulousness, initially unilateral with asymmetry over time. Involve legs, lips, jaw, tongue, arms, rarely head. Worse with anxiety or stress.
- Bradykinesia: Slowness of movement (most common feature of PD)
  - Major cause of disability. Difficulty with manual dexterity of fingers.
- Rigidity (90%)
  - Passive movement (arms, legs, neck). Initially unilateral. Cogwheeling *if* tremor present. Lead-pipe rigidity.
- Postural instability (later stages)
  - Stooped posture. + retropulsion test. Frequent falls.



Parkinson's Motor Symptoms

- ***Rest tremor***
- ***Bradykinesia and slowness of ADL's***
- ***Rigidity and freezing in place***
- ***Postural instability***
- Shuffling gait; freezing, festination
- Decreased arm swing while walking
- Difficulty arising from a chair
- Micrographia
- Hypomimia (lack of facial expression)
- Camptocormia (anterior flexion of thoracolumbar spine)
- Decreased eye blink; Impaired gaze and eyelid opening
- Difficulty turning in bed
- Hypophonia
- Dysphagia
- Sialorrhea (excessive salivation)
- Dystonia
- Striatal hand or toe

Off Medications, Pre-DBS

Treatment

- **Goal** = maintain function and quality of life while avoiding drug – induced complications.
- Bradykinesia, tremor, and rigidity respond well
- Cognitive symptoms, autonomic dysfunction, and imbalance respond poorly

Non-pharmacologic

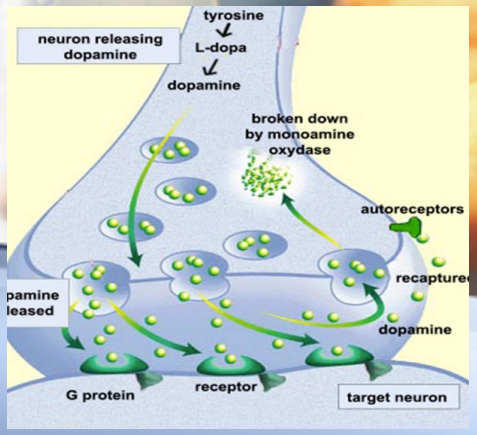
- PT/OT/ST (low threshold and ongoing)
- Specialist referrals (Urology, ENT, Pulmonary, etc)
- Fall Precautions
  - Assisted devices early to avoid falls
  - Get rid of throw rugs
  - Grab bars and shower chairs
  - Cues or tricks to overcome freezing (marching to command, stepping over an object, walking to music, rocking, metronome, etc)
- Nutritional counseling
- Education
- Volunteer activities, Support groups, Leisure activities

Exercise in PD

- Critical component
- Symptom management
  - Improvement in gait, balance, flexibility, coordination, pain
  - Decrease in falls
- Neuroprotection: slowing disease progression

- 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 (www.lsvtglobal.com)
  - LSVT BIG Tx (pre/post): www.youtube.com/watch?v=wElz9jNrqns
  - LSVT BIG Program (example): www.youtube.com/watch?v=nPmPCa1H3hU

Medical Management of PD



- **Levodopa (various formulations)**
  - Dopamine precursor (L-dopa) increase availability of dopamine
  - Most effective treatment in improving motor sx and quality of life
- **Catechol-o-methyltransferase (COMT) Inhibitors**
  - Blocking metabolism of dopamine in the brain and periphery
- **Monoamine Oxidase Type B (MAOB) Inhibitors**
  - Block catabolism of dopamine at the synapse
- **Dopamine Agonists**
  - Stimulate postsynaptic dopamine receptors directly
  - Often first agents used due to lower risk of motor complications (dyskinesia's and motor fluctuations)
- **Other agents**
  - More mechanisms emerging: anticholinergics, Adenosine A 2 R agonists, etc
  - Targeted botulinum toxin injection (dystonia)

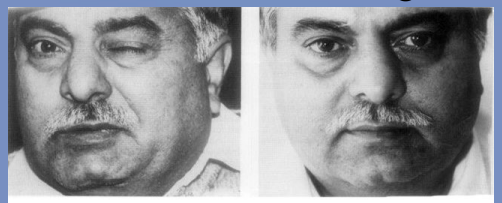


Fig. 3 - Patient before (left) and 4 weeks after injection of botulinum A exotoxin into left perioral and periocular muscles for hemifacial spasm.

L-dopa

- L-dopa is a precursor to the catecholamine neurotransmitters dopamine, norepinephrine and epinephrine
- L-dopa crosses the blood-brain barrier where it is converted into dopamine
- Conversion into dopamine in the peripheral nervous system results in many of the side effects (nausea, vomiting, orthostasis)
- Thus, it is common practice to combine L-dopa with a peripheral decarboxylase inhibitor (carbidopa or benserazide) to prevent the peripheral synthesis of dopamine from L-dopa

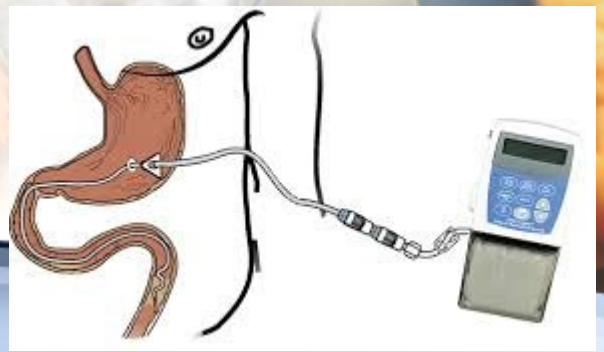
Levodopa Side Effects

- Early stage
  - Nausea, vomiting
  - Drowsiness, confusion
  - Dizziness, hypotension, headaches
- Later stages
  - Hallucinations, Delusions, Psychosis
  - Dyskinesias
  - Motor fluctuations
  - Dystonias

Strategies to combat these:

- Shorten dosing interval
- Add an inhibitor of levodopa/dopamine catabolism
- Take 1 hour before or 2 hours after a protein-rich meal to minimize impact on absorption

Duodopa/ Duopa



- FDA Approved in U.S. (“Duopa”) 1/12/2015, available outside of U.S. (“Duodopa”) for many years
- 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

Dopamine Agonist

Mimics dopamine, readily crosses blood brain barrier, long lasting with more uniform stimulation

Monotherapy or adjunct

Effective in treating bradykinesia, tremor, and gait; Less potent < L-dopa

PRAMIPEXOLE (MIRAPEX)

- Renally cleared, at least 2x stronger than others
- 0.125 mg TID and increase q week
- 0.5mg-1.5 mg TID (therapeutic at 3mg daily)
- XL: 0.375mg daily, 0.75 mg, 1.25 mg, 2 mg (therapeutic at 1-1.5 mg daily)

ROPINIROLE (REQUIP)

- 0.25 mg TID, 0.5 mg, 0.75 mg, 1 mg TID
- XL: 2mg, 4 mg, 6 mg (therapeutic dose), 8 mg

ROTIGOTINE TRANSDERMAL (NEUPRO)

- 2 mg, 4 mg, 8 mg q 24 hours

Side Effects: Caution with elderly!

- Somnolence, sleep attacks, daytime sleepiness
- Psychiatric symptoms: Visual hallucinations, confusion
- Peripheral edema
- Impulse Control Disorder (6-10%): punding, compulsions
- Nasal congestion
- Potentiating levodopa effects (nausea)
- Postural hypotension
- Dyskinesias (< Levodopa)

A Unique Dopamine Agonist

- Apomorphine (Apokyn)
  - Injected subcutaneously by the patient
  - “Rescue” drug: For treatment of acute off periods despite treatment with existing antiparkinsonian therapy
  - Short half-life: Benefit lasts 1 hour
  - Take with trimethobenzamide, anti-nausea therapy, prior to injection for first several weeks
  - May use up to 5 times/day

Impulse-Control Disorder

- Impulse-Control: excessive dopaminergic stimulation
  - Prevalence approximately 6% (Voon, et al., 2006)
  - Gambling, compulsive shopping, hypersexuality, hobbyism, punding (excessive, repetitive, purposeless movements: shuffling papers; reordering bricks, sorting handbags), compulsive medication use (in excess of dose required to alleviate motor symptoms)
- Distressing to patient and can be devastating to families

- Intervention:
  - Discontinue Dopamine Agonists
  - Pending actual concern, access to finances, medications etc. may need to be limited.

Catechol-o-methyltransferase (COMT) Inhibitors:

Block enzyme that breaks down levodopa: administer with levodopa

TOLCAPONE (Tasmar)

- Risk of fatal hepatic failure
- Check LFTs q 2 weeks X 1 year then q 1 months thereafter
- Administer 100-200 mg TID

ENTACAPONE (Comtan)

- Administered 200 mg along with each dose of carbidopa/levodopa
- Maximum daily dose 1600 mg
- Stalevo (carbidopa, levodopa, entacapone) combined

Side effects:

- Orange discoloration of urine and bodily fluids,
- Levodopa potentiation (sleep disturbances and dyskinesias),
- Confusion, hallucinations,
- Nausea,
- Orthostatics,
- Diarrhea

Monoamine Oxidase Type B (MAO-B) Inhibitors

Increases the half-life of dopamine by blocking breakdown of dopamine at the synapse

Monotherapy or adjunct treatment

RASAGILINE (Azilect) 1 mg/day

SELEGILINE (Eldepryl, Deprenyl) 5mg BID

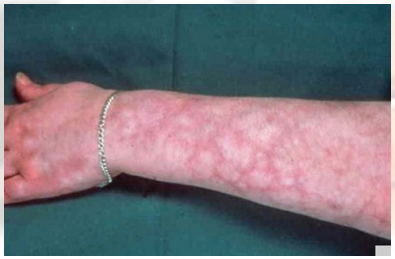
SELEGILINE orally disintegrating tab (Zelapar) 1.25 to 2.5mg/day

- Less first pass hepatic metabolism

Side effects (well tolerated)

- Most common: mild nausea, dry mouth, lightheadedness, constipation
- Tyramine effects (potentially fatal tachycardia and hypertensive crisis)
  - Limit tyramine in diet (fermented foods: aged cheese, cured meats, soy sauce, beer on tap, red wine
- Potential for rare serotonin syndrome
  - Caution with indirectly acting sympathomimetics (tyramine, ephedrine, pseudoephedrine), SSRI, TCA, etc

Other Agents



Amantadine

- Treats drug-induced dyskinesias in advanced PD by 70%
- Adjunct to levodopa
- Dosed as 100 mg 1-4 times per day
- Good initial effect but wanes in weeks to months

Side Effects: Caution with elderly or renal impairment

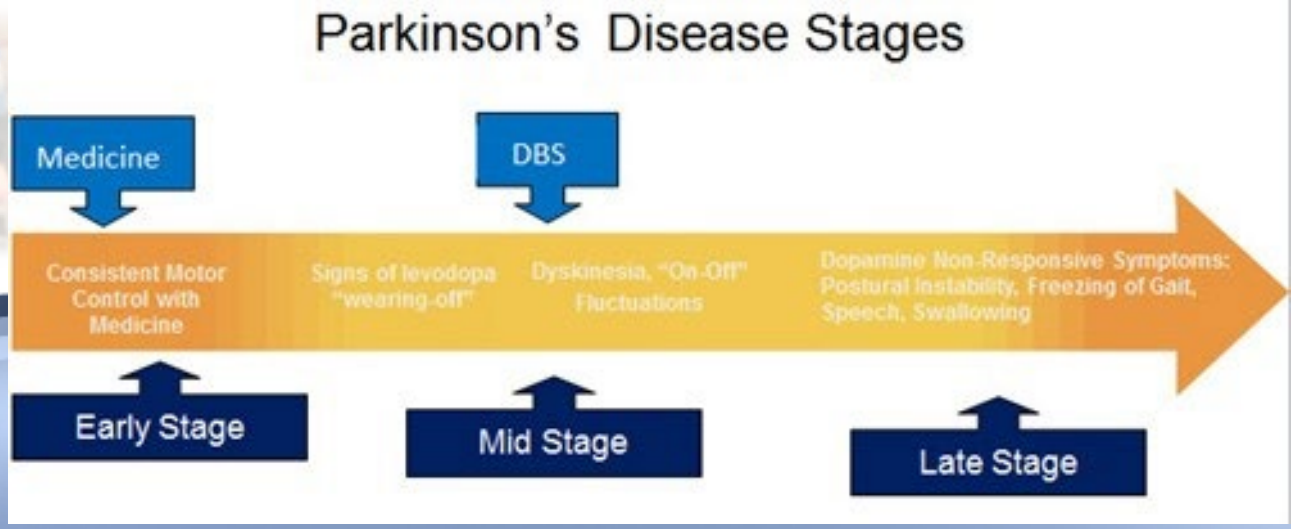
- Common: nausea, headaches, edema, erythema, dry mouth, lightheadedness, insomnia, confusion and pyschosis/hallucinations (elderly)
- Rare: urinary retention, Livedo reticularis

Anticholinergics

- Preferentially treats rest tremor and dystonia
- Monotherapy or adjunct
- TRIHEXYPHENIDYL (ARTANE)
- BENZTROPINE (COGENTIN)

Side effects: USE WITH CAUTION in the elderly!

- Cognitive dysfunction, confusion, hallucinations
- Constipation, urinary retention
- Blurry vision
- Dry mouth



Progression

- Preclinical phase
- Diagnosis
- Onset of therapy
- Honeymoon period: 0-3yrs
- Motor complications: 3 yrs
- Resistant symptoms: 8 yrs
- Cognitive decline: 15 years
- Death: 20 years
- THERE IS A SPECTRUM!

Management of Motor Fluctuations in PD

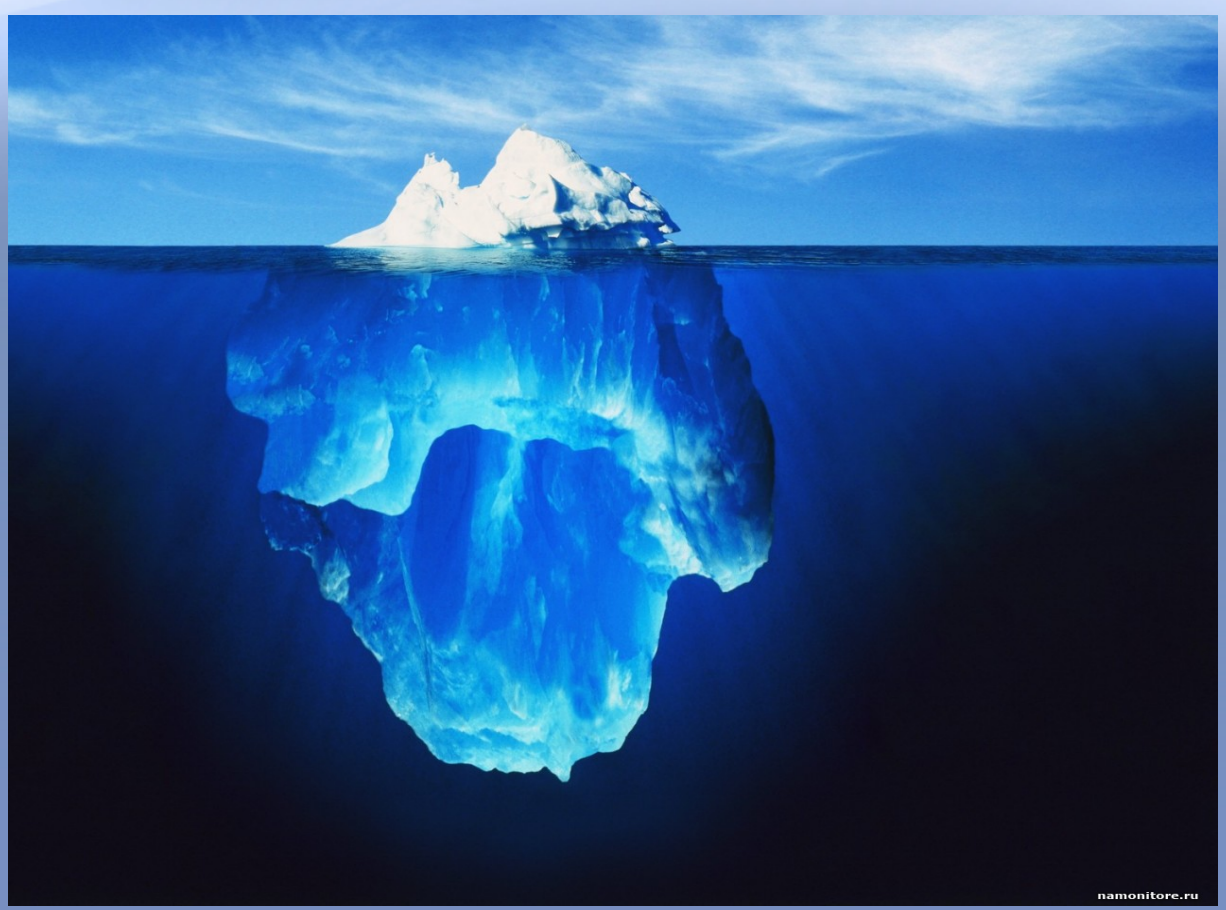
- Characterize fluctuations
- Adjust levodopa
  - Change incremental dose and interval
  - Different preparation
- Add adjunctive agent
  - Amantadine
  - MAO-B inhibitor
  - COMT inhibitor
  - Dopamine agonist
    - injectable apomorphine as a “rescue” drug

Management of Dyskinesia

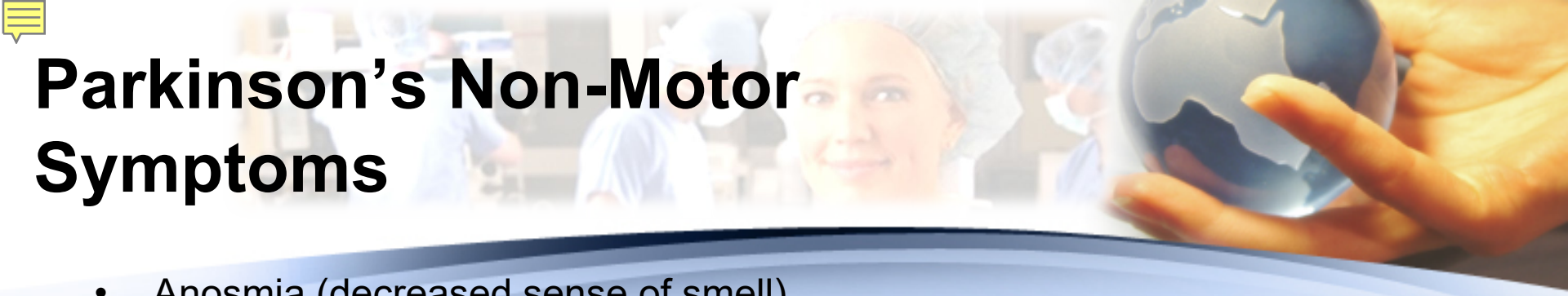
- Adjust levodopa dose and interval
- Decrease adjunctive medications
- Amantadine 100 mg BID

Pre-DBS On Medication

Non-Motor Symptoms



Parkinson's Non-Motor Symptoms



- Anosmia (decreased sense of smell)
- Drooling (dysphagia)
- Blepharitis
- Hypophonia (low vocal volume)
- Sleep disturbance (RBD, OSA, RLS, PLMS, fragmentation)
- Painful foot cramps (dystonia)
- Bursitis, "frozen shoulder"
- Cognitive dysfunction and dementia
- Psychosis and hallucinations (PDD vs DLB vs drugs)
- Mood changes (depression, anxiety, apathy)
- Autonomic dysfunction
  - Postural lightheadedness (orthostatic hypotension), ,Sudomotor dysregulation (abnormal sweating)
  - Constipation, urinary difficulties, male erectile dysfunction,
  - Olfactory dysfunction
- Pain and sensory disturbance (dystonia or "off")
- Seborrhea

Cognition

- Cognitive changes can start early in the disease
  - Generally correlated with disease severity but considerable variability
- Pathology
  - Cortical Lewy bodies
  - Alzheimer's disease
- Lead to reduced job performance and contribute to loss of functional abilities (e.g. driving, cooking safely)
- The cognitive and secondary functional consequences can also create stress for families of affected individuals

Cognitive Deficits found in PD

- Bradyphrenia – slowing of cognitive processes
- Apathy
- Attention and concentration
- Short-term Memory problems
- Visuospatial Deficits
- Executive Deficits
- Language production

Mild Cognitive Impairment (PD-MCI)	Parkinson's disease Dementia (PDD)
Point prevalence 20-30%	Point prevalence ~30%
• Diagnosis of PD • Gradual cognitive decline in the context of PD • Cognitive deficits on either formal neuropsychological testing or global cognitive screen • Cognitive deficits **not** sufficient to result in loss of independence	• Diagnosis of PD (motor symptoms clearly proceed dementia*) • Dementia syndrome with insidious onset and slow progression • Impairment in more than 1 cognitive domain • Deficits severe enough to impair independent fx

Coping with cognitive loss: Compensatory strategies

- Use ‘external aids’
  - Use a daily ‘To Do’ list (helps to ‘getting going’ and what order to tackle things)
  - Break big jobs into little steps
  - Use a calendar (place in highly visible location)
  - Use alarms and reminders on smart phones
  - Keep a routine schedule
- Minimize distractions
  - Do only one thing at a time (limit radio and/or talking while driving, limit conversations and other distractions while cooking)
  - Work in a quiet location
  - One question at a time! (minimize stimulus overload!)
- Allow for extra time

Activities associated with improving or maintaining brain health

- Remain mentally engaged
  - Currently unknown which activities or ‘games’ are best
  - Active ingredients: learning new skills, moderately challenging
- Remain as physically active as possible
  - Many studies demonstrate benefits of physical exercise to brain structure and function
  - Consider physical activities that have a strong cognitive component (e.g. adapted tango class improved spatial cognition in PD, as well as executive function and balance)
- Remain socially engaged
  - Cognitive stimulation
  - Emotional support

PD Dementia: Pharmaceutical Interventions

- 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
- Glutamate Antagonists
  - 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
  - “sundowning”
- Paranoia, illusions, delusions, agitation
- Risk Factors
  - Age, severe disease, cognitive impairment
- Predicts nursing home placement and early death
- Avoid CNS dopamine receptor antagonists (metoclopramide, Antipsychotics)
- 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

Pharmacologic Treatment of Psychosis in PD

- Clozapine (Clozaril)
  - 12.5 mg to 25 mg BID
  - Risk of agranulocytosis requires frequent monitoring of WBC count
  - AE: 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
  - AE: Somnolence
- Discontinuation of Dopamine Agonists, if appropriate

Mood Disorders in PD

- Secondary to underlying neuroanatomical degeneration, rather than a reaction to psychosocial stress and disability.
- Prevalence:
  - Anxiety 40%; Depression 50-70%
- Depression, anxiety precede motor symptoms of PD by ~6 years
- Inquire re: pattern of sx onset:
  - Disease progression and “wearing off” can mimic depression (e.g., anxiety, sense of impending doom, dysphoria)
  - Motor symptoms, balance and gait disturbance, freezing episodes, increased fall risk can contribute to anxiety
  - Optimize dopaminergic therapy, decrease “off” time

Mood Disorder: Interventions

- **Nonpharmaceutical Interventions:**
  - Good nutrition, sleep hygiene, moderate cardiovascular exercise, PD support group, social interaction, stimulating leisure activities
  - Psychotherapy (individuals and couples): CBT
  - Meditation, massage, yoga, exercise, acupuncture, biofeedback
- **Pharmaceutical Interventions:**
  - Depression: SSRI, SNRI, NDRI, TCA
  - Anxiety: SSRI, Buspirone (NDRI), Benzodiazepines
- Serotonin Syndrome : antidepressants & MAO-B inhibitors
  - Risk is lower with MAO-B < MAO-A inhibitors
  - Sx: acute mental status changes, autonomic dysfunction, myoclonus, hyperreflexia
  - Educate patient and family, close monitoring

NMS	Interventions
Blepharitis	High viscosity lubricant eye drops at bedtime (ex. Systane gel drops)
Shoulder Pain	Physical Therapy LSVT BIG arms gait training Muscle relaxants
Dysphagia	Speech and Swallow Therapy • Swallow techniques: Second swallow, Chin tuck, Straws • Food consistency: Thickened liquids, Softer food texture
Sialorrhea (drooling)	Gum and hard candy trigger swallow reflex Anticholingerics: not worth the side effects Atropine eye drops on the tongue (dec AE) Targeted botulinum toxin injection of the parotids
Nausea, Bloating • **Levodopa effect** • **Gastroparesis**	Treat with supplemental dopa-decarboxylase inhibitor: Lodosyn (carbidopa) 25mg with each Sinemet dose Management of constipation Small, more frequent meals
Speech Deficits • **Hypophonia** • **Imprecise Articulation, accelerated rate, decreased intelligibility**	Lee Silverman Voice Treatment (LSVT LOUD) • Lsvtglobal.com • https://www.youtube.com/watch?v=gNIdxYjGVV8

Sleep Disturbances

- **Referral to Sleep Specialist**
- **Diagnosis**: Formal Nocturnal Polysomnography
  - Must specify application of EMG leads on the extremities in PD (RBD, RLS, PLMS)

- **Treatment**:
  - Treat underlying sleep disorder
  - 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)
    - » Limit naps

Sleep Disturbances	
Rapid Eye Movement (REM) Sleep Behavior Disorder (RBD)	Clonazepam (Klonopin) 0.25-1.0 mg QHS: off label, low dose Melatonin 3-5mg tabs, up to 10mg QHS Extra supplemental doses of carbidopa/levodopa at night
Sleep Fragmentation	Clonazepam (Klonopin) 0.25-1.0 mg QHS: off label Melatonin 3mg (1-3 tabs) QHS
Obstructive Sleep Apnea (OSA)	CPAP, BiPAP
Restless Legs Syndrome (RLS) & Periodic Limb Movements in Sleep (PLMS)	Dopamine agonists Extra nighttime dose of DA or levodopa
Excessive Daytime Sleepiness	Identify underlying sleep disorder and treat directly Modafinil (Provigil): FDA-approved "to improve wakefulness in adult patients with excessive sleepiness associated with narcolepsy, OSA, and shift-work sleep disorder" • fewer side effects < stimulants Methylphenidate (Ritalin): can increase daytime alertness and wakefulness; off-label • Side Effects: palpitations, high blood pressure, confusion, psychosis, insomnia
Insomnia	Mild sedatives are well-tolerated in the non-demented patient : Zolpidem (Ambien), Zaleplon (Sonata), Eszopiclone (Lunesta), Ramelteon (Rozerem) • None are FDA approved in PD

Autonomic Dysfunction

Sexual Dysfunction • **Decreased libido, motor sx, impaired expressiveness** • **Male erectile dysfunction** • **Hypersexuality**	Rule out other etiology Refer to urology/ gynecology **Erectile dysfunction:** • Sildenafil (Viagra), Tadalafil, Vardenafil (Watch for hypotension) • Dopamine Agonists **Hypersexuality:** • Associated with dopaminergic treatment (DA)
Constipation	Optimize hydration Well-balanced, high fiber diet (fruits, vegetables, prunes, bran cereal) Regular exercise Fiber supplements, bulk formers Stool softeners: Daily Docusate Sodium Laxatives: Polyethylene Glycol (Miralax) 17g packet daily or QOD
Overactive Bladder	Protective undergarments Commodes or urinals at bedside to prevent falls and accidents Urology referral Sphincter relaxants/anticholinergics: Ditropan, Detrol, Vesicare • Monitor for side effects
Urinary Retention	Urology referral Evaluate medication side effects

Autonomic Dysfunction: Orthostatic Hypotension (OH)

Initial Interventions:

- **Compression stockings**
- **Increase water, salt and caffeine intake**
- **Rise slowly**
- **Raise head of bed, elevate legs when sitting**
- **Monitor orthostatic vitals at every visit**
- **Decrease dose of DA or levodopa**
- **Adjust bp meds if necessary**
- **Rule out other etiology**
- **Watch for SUPINE HYPERTENSION**

Pharmacologic Interventions:

Midodrine (ProAmantine): alpha-1 adrenergic receptors agonist

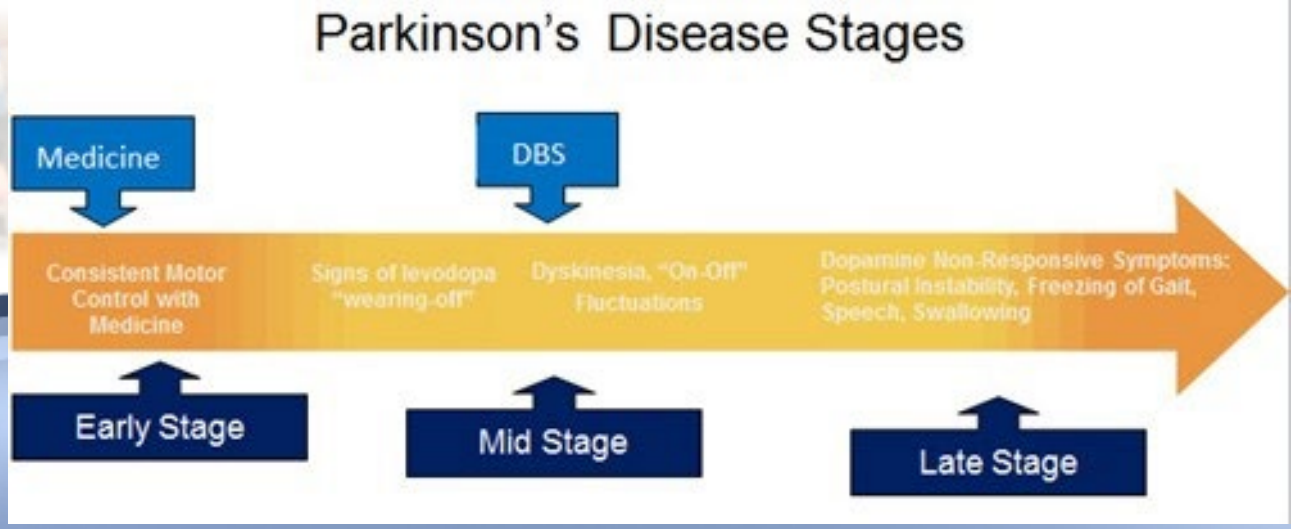
- Increases systemic vascular resistance
- 2.5 mg to 5 mg TID
- Black Box Warning: supine hypertension (> Fludrocortisone)

Fludrocortisone (Florinef): systemic corticosteroid that increases salt retention -> increasing blood volume

- 0.1 mg to 0.3 mg daily
- Watch for excessive supine hypertension, edema

Droxidopa (Northera): Increases level of norepinephrine and epinephrine in peripheral nervous system => tachycardia and hypertension

- Side effects: tachycardia, hypertension, nausea, vomiting, headache, migraine
- Black Box Warning: supine hypertension
- Elevate HOB when sleeping
- Start with 100 mg TID and titrate in increments of 100 mg TID every 24-48 hours.
- Monitor supine bp prior to initiation and after each dose increase



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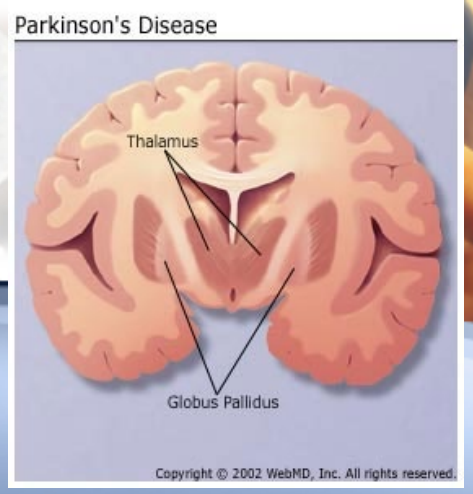
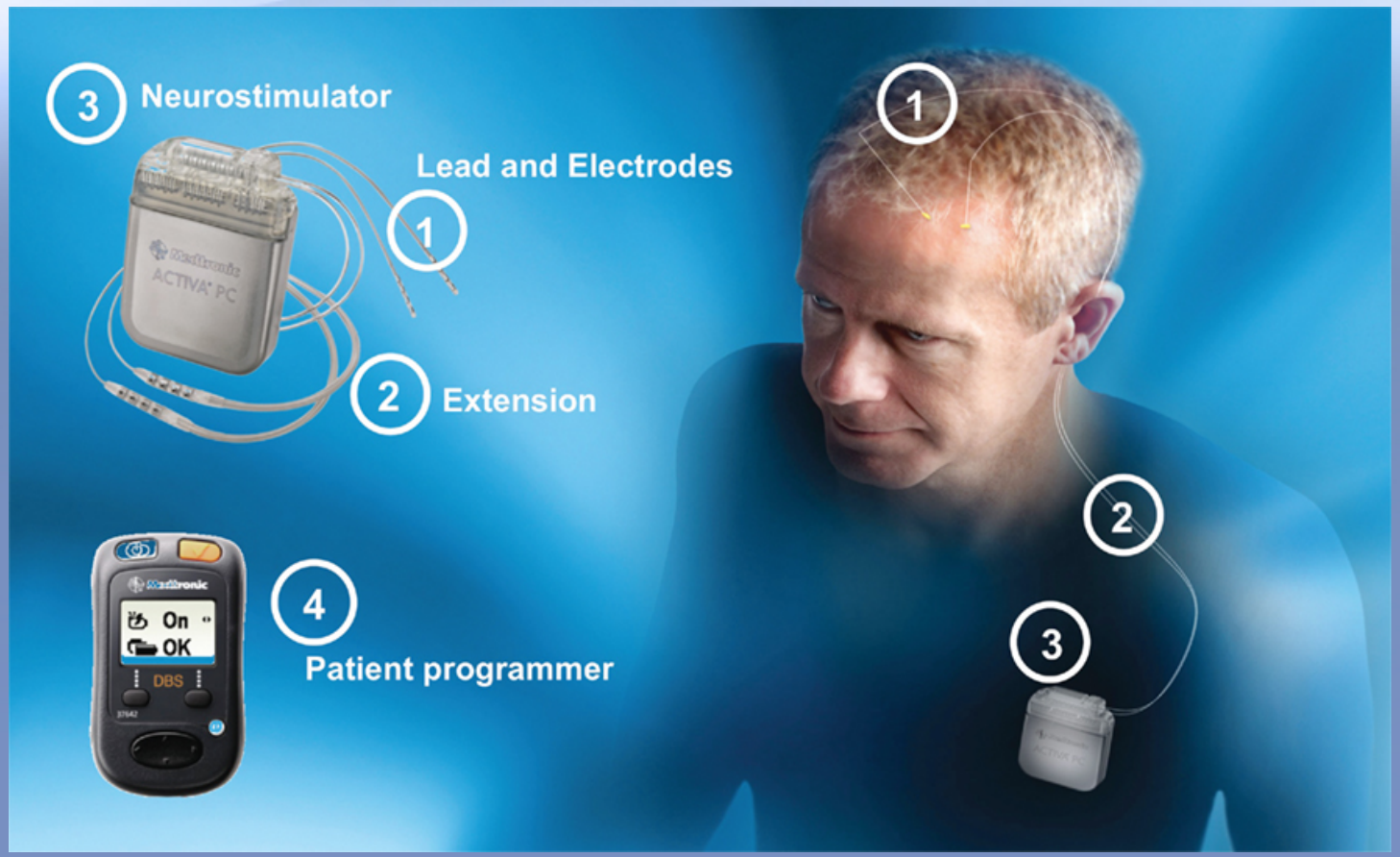
Surgical Treatments for PD



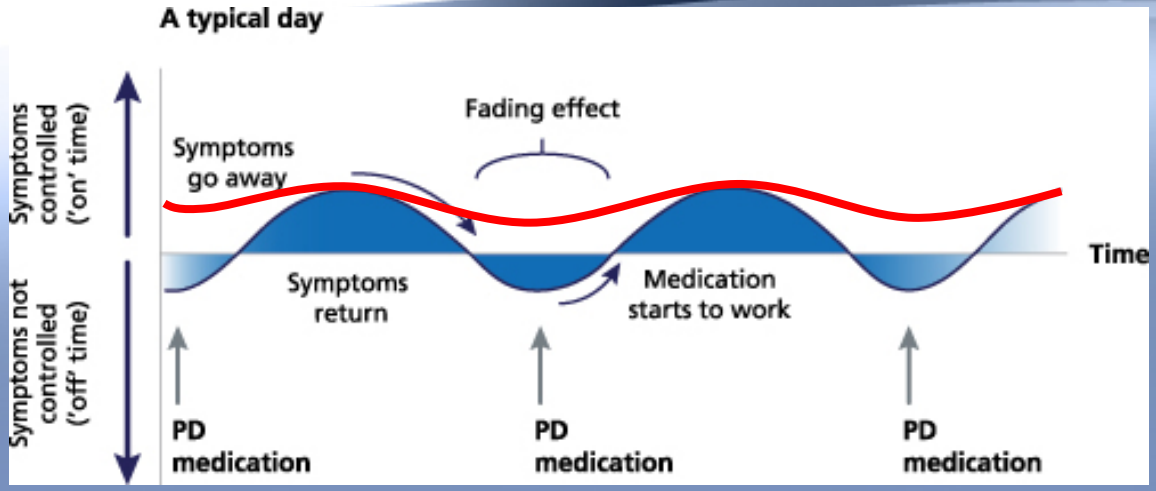
- Pallidotomy:
  - Surgery permanently destroys the overactive globus pallidus to lessen the symptoms of Parkinson's disease
- Thalamotomy:
  - Surgery destroys part of the thalamus to block the abnormal brain activity from reaching the muscles and causing tremor.
  - It only targets tremors DBS
- Deep Brain Stimulation (DBS)
  - Adjustable, reversible surgical intervention
  - Targets: Subthalamic Nucleus (STN) or internal aspect of Globus Pallidus (Gpi)

DBS Hardware



Benefits of DBS for PD

DBS is typically *as effective* as "best" dopamine response…



~ 30% improvement in motor scores

~ 40% improvement in ADL scores

~ 50% reduction in PD medication needs (STN)

Likely to improve:

- ✓ Tremor
- ✓ Rigidity (tightness)
- ✓ Bradykinesia (slowness)
- ✓ Dystonia
- ✓ Dyskinesia*

Unlikely to improve:

- Gait instability / falls
- Freezing of gait
- Speech
- Swallow
- Cognitive deficits

1 year Post DBS Surgery

DBS Safety Issues

- **MRI:** fields can induce tissue damage
  - MRI of neck or body is contraindicated
  - Specific DBS protocol must be used – head coil MRI < 1.5T
  - Depending on the DBS device, the IPG needs to be turned off or re-programmed to factory settings before MRI by DBS provider
- **Diathermy:** contraindicated
  - It can heat up the leads resulting in stroke or death and can damage DBS system
- **Bipolar electrocautery** only. DO NOT USE UNIPOLAR DEVICE.
  - Prior to procedure, turn off DBS and set amplitude settings to "0"
  - Ground lead should be placed on a LE
- **Cardiac pacemaker**: must be 10 inches away from DBS device
- **Lithotripsy** is not recommended unless only medical option
  - If needed, use protective shielding over neurostimulator and turn system off and to "0"
- **Dentist**: do not place electric drills/ cleaning tools over DBS system
- Avoid exposure to high voltage electrical and/or magnetic fields (i.e welding)

AAN Parkinson's disease Quality Measures (2010)

Annually:

1. Annual PD diagnosis review
2. Psychiatric assessment
3. Cognition assessment
4. Query autonomic dysfunction
5. Query sleep disturbances
6. PD rehab therapy options
7. PD related safety issues counseling
8. Review of PD medical and surgical treatment options

Each Visit:

9. Query about falls
10. Query about PD medication-related motor complications

University of California, Davis Deep Brain Stimulation Team

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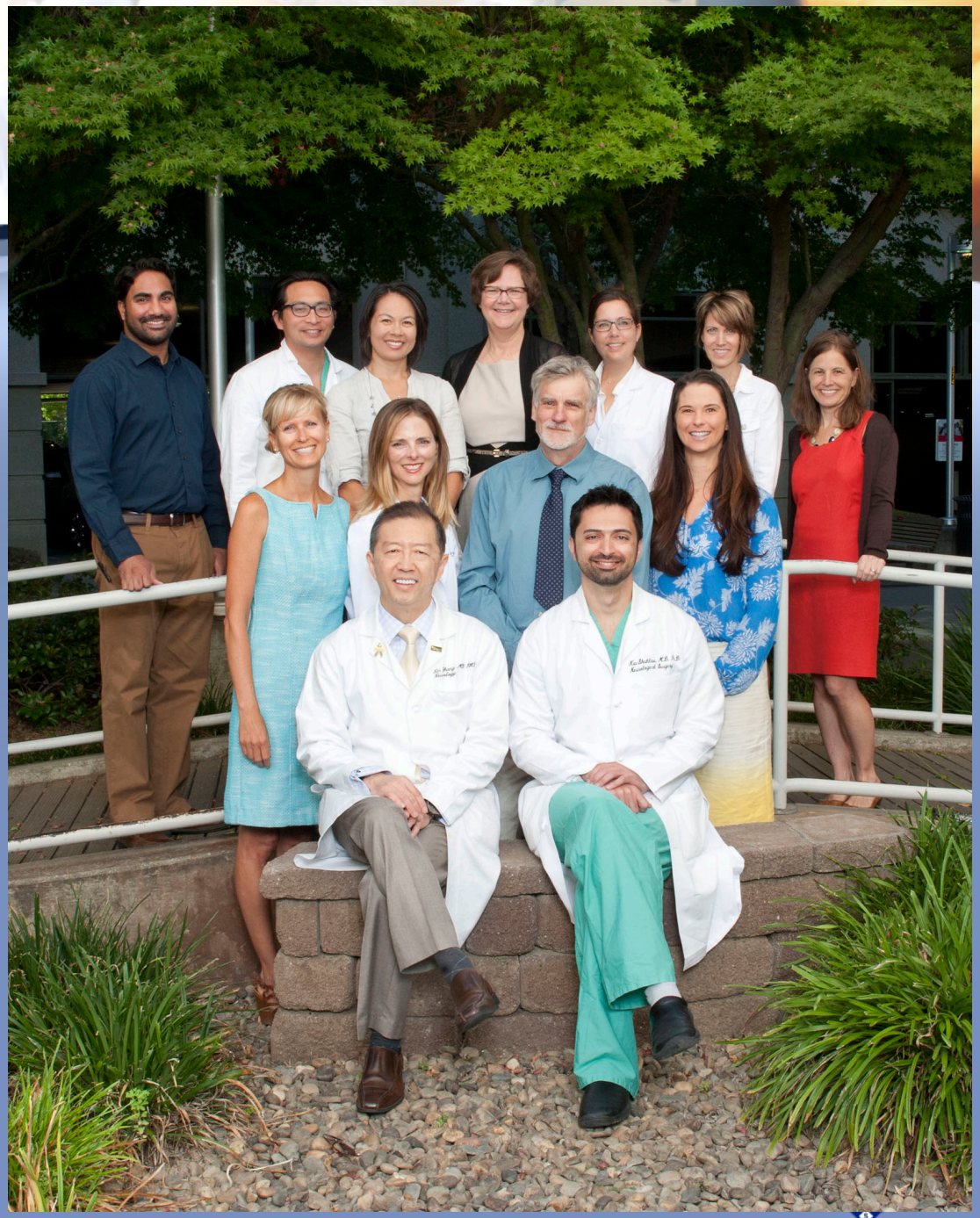

Questions?

- Thank You!

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