Parkinson’s Disease and the Parkinsonisms: Recognition and treatment strategies specific to condition

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Outline and objectives

Participants will be able to:

• Identify relevant physiologic changes that occur in PD and each of the main Parkinsonisms.

• Identify the evidence on objective testing of each impairment

• Apply recent evidence in clinical interventions to each of the classifications or presentations of PD and Parkinsonisms.

Timeline

• Introduction and epidemiology
• Pathophysiology of Parkinson’s Disease and the variant subtypes
• Pathophysiology and presentations of Parkinsonisms
• Translating the evidence with practical intervention strategies across the conditions
• Case Studies
• Bonus material

PD – statistics and trends

• Incidence
  — 4-6 million affected directly
  — 60,000 newly diagnosed cases/year
  — Frequency of PD will increase 4 fold by 2040

• Prevalence
  — 0.3% in general US population
  — Prevalence increases to 4-5% in those >85 years
  — Average age of onset 60 years
Parkinson’s Disease

- Progression
  - Can be rapid or slow
  - Progression in symptom severity and number of symptoms
- Complicating definitive diagnosis
  - Variable presentations, given affected neurons
  - Many related conditions imitate PD
  - Clinical diagnosis only – no definitive test

Making a diagnosis in PD

Brainstem structure: the substantia nigra – source of dopamine

Clinically detectable after loss of 60-80% of neurons

Presence of eosinophilic intracytoplasmic inclusions

Binding protein: α-synuclein – creating plaques

Making a diagnosis in PD

- Clinical examination to arrive at diagnosis of PD
- Imaging likely normal: CT, MRI, diffusion-weighting*
- Autopsy can confirm
- Diagnosis from clinical presentation: “rule-in”
  - Asymmetrical onset and good response to dopamine
  - Clinical indicators
    - Resting tremor
    - Rigidity
    - Loss of postural reflexes
    - Dyskinesia
    - Gait impairment
    - Bradykinesia
    - Flexed posture
    - Frequent falls

The Parkinsonisms

- Multiple System Atrophies: (MSAs)
- Alzheimer’s Disease (AD)
- Fronto-temporal Dementia (FTD)
- Lewy-Body Disease (LBD)
- Stroke, toxicity, trauma (basal ganglia or SN)
- Cortico-Basal Ganglia Degeneration (CBGD)
- Progressive Supranuclear Palsy (PSP)
Abbreviations

• PD = Parkinson’s Disease
• PRD or PD+ = Parkinsonism/Related Disorders/Parkinson Plus
• PWP= Persons with PD
• YOPD= Young Onset PD
• PSP = Progressive Supranuclear Palsy
• AD = Alzheimer’s Disease
• FTD = Fronto-Temporal Dementia
• MSA = Multiple Systems Atrophy (subtypes A, C, P)
• HD = Huntington’s Disease
• CBGD or CBD = Cortico Basal Ganglia Degeneration

Differential Diagnosis

Three Phenotypes of PD
1) Young onset (YOPD)
2) Tremor
3) Rigidity/Freezing of Gait

Parkinsonisms and related disorders (PD+)
• PSP: Progressive Supranuclear Palsy
• CBGD: Cortico-Basal Ganglia Degeneration
• MSAs (3): Multiple Systems Atrophy
• HD*: Huntington’s Disease
• AD and FTD: Alzheimer’s and Fronto-Temporal Dementia

Movement Disorders Society (MDS) clinical diagnostic criteria for Parkinson’s disease (Postuma et al.2015).

Clinical examination features: The bedside differential diagnostics

Bradykinesia is the core feature of PD and PD+.

Slowness of initiation of voluntary movement with progressive reduction in speed and amplitude of repetitive movements. The emphasis on the decrement in amplitude or speed in repetitive movements.

NOT present in most PSP.

Clinical examination features: The bedside differential diagnostics

Rigidity: Velocity-independent resistance to passive movement. Typically of unilateral onset.

Asymmetry in PD, MSA-P, CBD, and PSP-P
Clinical examination features: The bedside differential diagnostics

Parkinson syndromes, a slow (4–6 Hz) tremor in the fully resting limb, which is suppressed during initiation of a movement.

Mainly observed in PD and virtually rules out MSA and CBD.

Clinical examination features: The bedside differential diagnostics

Myoclonus is not observed in PD, DLB, and PSP, but frequent in CBD and MSA.

Clinical examination features: The bedside differential diagnostics

Postural instability, as evidenced with a pathological pull-test, without identifiable cause, occurring within 3 years after symptom onset and leading to recurrent falls - favors atypical Parkinson syndromes over PD.

Clinical examination features: The bedside differential diagnostics

Freezing, i.e., a sudden stop or hesitation in locomotion

Early-onset is typically seen in vascular Parkinsonism or PSP/PSP with predominant gait freezing (PSP-PGF).
Clinical examination features: The bedside differential diagnostics

Dystonia: asymmetric dystonic posturing of a foot during off-periods is common in PD.

Dystonia in MSA often develops rapidly into fixed dystonic contractions of hands + feet

PSP, dystonia is also typically not fluctuating

Clinical examination features: The bedside differential diagnostics

Abnormal postures are also very frequently observed in PD and PD+.

Camptocormia and Pisa syndrome + antecollis

Retropulsion is suggestive of PSP

Differential diagnostics: Response to Dopamine

Response is NOT an outright differential, however:

PD should present with the highest responsiveness
NOT PD if no response in moderate severity sx
MSA, PSP, CBS may respond early, then fleeting

Hoglinger, G. et al. J Neural Transm. 2017
Disease Specifics: Late onset PD

- Slower rapid disease progression (varied)
- Three subtypes:
  - Tremor
  - Rigidity
  - Dyskinesia*

*LATE ONSET dyskinesia likely medication-induced

Disease Specifics: Parkinsonism

- Variable presentations given etiology
  - Stroke, multiple concussions, encephalitis
- Typically imbalance > tremor and rigidity
- Typically less freezing
- DIFFERENTIAL: Limited to no responsiveness to dopamine meds in most conditions

Disease Specifics: PSP

- Rapid progression – live 6 yrs after diagnosis
- Posterior loss of balance tendencies (retropulsion)
- Cognitive changes
- Loss of superior gaze
- Similar to other prion diseases (Creutzfeldt-Jakob disease)
- Downbeat nystagmus and gaze-evoked nystagmus

*DIFERENTIAL: Loss of superior gaze AND no bradykinesia

Disease Specifics: CBGD or CBD

- Rapid progression and late onset
- Greater apraxia and some aphasia
- Clinical diagnosis
- May have cognitive and psychiatric changes

*DIFFERENTIAL with AD, LBD: extrapyramidal symptoms

Similar to:
- Dementia with Lewy Bodies, PSP, AD
AD + FTD (fronto temporal dementia)

- Impaired declarative (facts) and episodic (events) memories
- NO impact on procedural memories (automatic, overlearned tasks)
- DIFFERENTIAL: early cognitive signs outside of dual-tasking efforts

Multiple System Atrophy (MSA)

- MSA-A (Autonomic)
- MSA-P (Parkinsonian)
- MSA-C (Cerebellar)

Typical onset early 60s
Survival at diagnosis +/- 8 years

Multiple System Atrophy (MSA)

- Autonomic dysfunction (Shy-Drager) with hypotension MSA-a
- Parkinsonistic: (muscle rigidity +/- tremor and slow movement) MSA-p
- Cerebellar: ataxia - gait and extremities MSA-c

*DIFFERENTIAL: progression of other symptoms.
* Sense of smell MAY be spared compared to PD

Swallowing
Often dry-mouth
Retropulsive

PROGNOSIS: Sx orthostatic hypotension + Urinary incontinence

Suzuki, M. BMC Neurol. 2011
Multiple System Atrophy (MSA)

- MSA-P (Parkinsonian)
  
  Typical onset is high frequency of falls
  Limited to no response to Dopamine
  Orthostatic hypotension
  
  “Striatonigral Degeneration”
  Diffusion-Tensor imaging MAY be + for MSAs

Multiple System Atrophy (MSA)

- MSA-C (Cerebellar)
  
  Rare late-onset ataxia without stroke or SCA*
  Sx progression
  Orthostatic hypotension
  
  *SCA = Spinocerebellar Ataxia (familial) 8+ subtypes

Neurophysiology of PD

Functional correlates:

Motor planning
Procedural memories
Sensory processing
Emotion
Selective + divided attention

Pathophysiology of Parkinson’s Disease

Structures involved:

Substansa Nigra
Basal Ganglia: Putamen
Globus Pallidus
Striatum
Dorsolateral Prefrontal Cortex
Neurophysiology of Parkinsonisms

Stroke (BG or substantia nigra)
Trauma (pugilistica)
Toxicity (mercury, other)
Degenerative (MSA, CBGD)
Infectious (encephalitis)
Familial (Essential tremor, others)

Pathophysiology of variant subtypes (phenotypes)

1) Primary dyskinesia
2) Primary tremor and rigidity
3) Freezing of gait; posture; and festination
4) Cognitive changes

Primary dyskinesia

- Structures – Globus pallidus
- Incidence – Young onset, estimated <10%
  Higher incidence with heavy Levodopa use
- Presentation – Whole body, progressive
  Increases with stress, fatigue, distractions
Primary tremor and rigidity

Structures

- Ventral intermedius n. (VIM) of the thalamus
- Subthalamic n. (STN) between BG and SN

- Incidence 80%
- Presentation

Freezing of gait; posture; and festination

- Structures – Putamen (automaticity)
- Incidence
- Presentation

(Freezing of gait – abbreviated as FoG)

Procedural Memories...

- Automaticity is lost in this form of PD (+ FoG)
- Overlearned tasks (procedural memories) are built through repetition and ALSO stored in:
  - Cerebellum
  - Supplementary Motor Area (SMA)
  - Premotor Cortex

Two forms of Long Term Memory

Explicit (declarative)

- Facts
- Events

Medial Temporal Diencephalic System

Implicit (nondeclarative)

- Neocortex
- Striatum
- Amygdala
- Cerebellum
Types of learning

Explicit Learning:

- Declarative, conscious verbalizable knowledge of facts and events supported by declarative memory
- Memory for words, scenes, faces, stories
- Assessed by conventional tests of recall and recognition

Implicit Learning:

- Nondeclarative, procedural, abstract knowledge without verbalizable or conscious awareness
- Supported by procedural memory
- Changes in performance as a result of experience
- Skill learning, habit formation, classical conditioning, priming

Procedural (Implicit) Memory System:

- Not dependent on awareness or cognitive processes
- Accumulates through repetition over many trials
- Expressed by improved performance on certain tasks
- Difficult to express in declarative form
- Includes perceptual and motor skills
- Includes learning of procedures and rules

For example:

Procedural knowledge of riding a bicycle:

- Physicists know the rule: turn the handlebars so that the curvature of the bike’s trajectory is proportional to the angle of its imbalance, divided by the square of its speed.
- Most bicyclists do not “know” this rule stated as such; however, at some level the information summarized in the rule is embodied in the neural networks that allow cyclists to stay erect while cycling.

Squire, 1986
TRANSLATING THE EVIDENCE WITH PRACTICAL INTERVENTION STRATEGIES ACROSS THE SUBTYPES

Individualizing PD programs

- Resources and impairments in the respective phenotypes or “Parkinson’s Diseases”.
- Underlying pathologies are not all the same
- Functional presentations are not the same
- Impairments are not the same
- Interventions SHOULD NOT be the same

Disease Specifics: YOPD

Dyskinetic movement

- INTERVENTION: maximize flexibility and conditioning, pain in overuse from dyskinesia
- Improve kinesthesia
- Improve core strength
- Improve dual task tolerance* (cognition often spared)

Disease Specifics: Late onset PD

Slower rapid disease progression (varied)

- Three subtypes:
  - Tremor
  - Rigidity
  - Dyskinesia

- Late onset dyskinesias are MOST likely medication-induced
- INTERVENTION: Conditioning, power, balance, compensation
Disease Specifics: Parkinsonism

- Variable presentations given etiology
  Stroke, multiple concussions, encephalitis
- Typically imbalance > tremor
- Less freezing NOT progressive
- Limited to no responsiveness to dopamine
- INTERVENTION is more aligned to stroke than high-intensity PD.

Disease Specifics: PSP

INTERVENTION: Compensatory, safety, conditioning

REMINDER:
- Rapid progression – live 6 yrs after diagnosis
- Posterior loss of balance tendencies
- Loss of superior gaze
- Cognitive changes
- Similar to other prion diseases (Creutzfeldt-Jakob disease)

Disease Specifics: CBGD

INTERVENTION: Assistive devices, core strength, conditioning, caregiver training.

Reminder:
- Rapid progression and late onset
- Greater apraxia and some aphasia
- Clinical diagnosis
- May have cognitive and psychiatric changes

Multiple System Atrophy (MSA)

INTERVENTION: Based on presentation ($p$ or $c$). Strength, endurance, orthostatic hypotension.

Reminder:
- Autonomic dysfunction (Shy-Drager) with hypotension $MSA-a$
- Parkinsonism (muscle rigidity +/- tremor and slow movement) $MSA-p$
- Cerebellar Ataxia - gait and extremities $MSA-c$
AD + FTD (fronto temporal dementia)

Intervention:
- Practical, functional treatment environments
- Caregiver education
- Blocked practice, USE preserved procedural
  - Impaired declarative (facts) and episodic (events) memories
  - NO impact on procedural memories (automatic, overlearned tasks)

CURRENT EVIDENCE FOR TREATMENT WITHIN EACH IMPAIRMENT AND FUNCTIONAL LIMITATION:

POWER
COORDINATION
DYSKINESIA

Commonalities in PD

THE cumulative cycle of INTENSITY leads to good things!!

- Attention
- Strength
- Endurance
- May include BDNF/GDNF
- +...more intensity!!
Common balance-related impairments in PD

- Musculoskeletal impairments
  - Posture
  - Muscle flexibility
  - Muscle performance (strength, power, endurance)
  - Tone (hypo- or hypertonicity)
- Voluntary control deficits (e.g., bradykinesia, freezing)
- Poor sensory integration
- Difficulty switching tasks in movement sequences
- Impaired cognitive processing and dual-tasking

Barriers to safe balance training in PD

High fall risk in treatment given:
- Rigidity
- Attention
- Anxiety
- Motor control (freezing, bradykinesia)
- BWSTT to the rescue??

Future research in PD: Learning Based Exercise Training

- Challenging neural networks/connections with attended learning based activities can improve timing and sequencing across sensory modalities
- Modify the “topographical” and “functional” brain representations of sensation, movement and task performance
  (Jenkins et al, 1984; Merzenich et al, 1985-2011; Byl et al, 1996; Blake et al 2002; Niewboer 2016)

Potential for Neuroprotective benefits in PD

Glial-derived neural protective factor (GDNF)
Stimulated or activated through:
- High intensity aerobic activities
- Skill-based speed and coordination activities
- Sport and competition with intensity
- Group programs of dance, balance
- More studies and practical applications...
Evidence in aerobic, dual task activities

- Dancing, high speed cycling, higher-intensity aerobic classes can enhance:
  - learning
  - mobility
  - fine motor skills in the UE + agility
  - mood/counter depression


Commonalities and future trends

- High intensity resistance exercise
- Task specific endurance exercise
- Postural control (reactive and pro-active)
- Feedforward/Feedback (APA and responsive)
- No one has too much strength
- Combine fun with exercise
- Combine personal interest with exercise

Proven Benefits of Intense Aerobic Exercise

- Increased brain volume (Colcombe et al, 2006)
- Improved efficiency of Beta blockers
- Improved BMI
- Improved psychosocial function (Kohut et al, 2010)
- Increased endorphines (Cohen et al, 2003)
- Upregulation of dopamine (Villar-Cheda et al, 2009)
- Increased brain growth factors; Zigmund et al, 2009; Vucckovic et al, 2010)
- Increases walking speed, stride length and endurance (Miyai et al, 2000;2002)
- Improved cardiovascular fitness ( Tillerson et al, 2003)

Barriers to aerobic exercise in PD

Challenge to achieve sustained heart rate given:

- Imbalance
- Rigidity
- Attention
- Anxiety
- Motor control (freezing, bradykinesia)

- BWSTT to the rescue??
Body weight support (BWS)

- Functional task
- Many patients have NEVER been on a treadmill
- Ensured safety, reducing anxiety
- Intensity and duration easier to record, replicate
- May induce balance challenges, overload
- Reduces dual task interference

BWST Training for PD

BWSTT vs. traditional care

- Greater outcomes in QOL, gait speed, balance, endurance
- Responses higher with H/Y patients 1.0 to 3.0

(M) Herman et al 2007, Miyai et al 2002

Motor learning in PD

- Can we use procedural learning?
- Can we introduce dual tasking?
- Can we generalize across all types of PD?
- Let’s investigate...

Procedural learning in the clinic

- Transfers
- W/C setup
- Bed mobility
- Dressing
- Walking
- Use of an assistive device
- Negotiating stairs
Dual tasking in PD and PD+

Reasonable to expect improvements given impairments in THE MAIN attention center (DLPFC)?

Yes, because...

1) Pre-building dual task tolerance
2) Need to compensate in distracting environments
3) Neuroplasticity = supply in the face of demand

Evidence-Based Practice

Intense Body-weight supported treadmill training
Intense balance challenges: speed, movement, surface and dual-task considerations - the "BIG" movement
Role of VOLUME regulation in all aspects of function
Make them comfortable with the world BEHIND them
Provide PROTECTED practice in changing directions
Postural changes and improving muscular balance
Compensatory efforts with visual, verbal rehearsal cues

The brain CAN change for the positive in PD...neuroplasticity

Tasks must promote: protection, competition, improvement
The brain will not change...
If there is no challenge
If there is no chance
If there is no expectation
If there is no success

Even with peripheral loss, there can be central changes
PD and PD+ POTENTIAL

- Muscular strength
- Muscular endurance
- Cardiovascular endurance
- Somatosensory neuroplasticity*
- Motor control neuroplasticity*
- PSYCHOLOGICAL

*PD+ neuroplasticity - requires BDNF. How?

Lessons in PD and PD+s

- Control all variables that you can...
- Consistent and lifelong exercise, activity
- No one ever comes to therapy with too much strength, or...too much endurance
- Balance exercises are a daily routine

Point by point...how you intervene

- Muscular strength*
- Muscular endurance*
- Cardiovascular endurance*
- Somatosensory neuroplasticity
- Motor control neuroplasticity
- PSYCHOLOGICAL

* Slides for you to review in BONUS section
Point by point...how you intervene

**Sensory neuroplasticity:**

- Sensory changes...with PD?...with PD+?
- Remove/alter sensory strengths
- Dual tasking - automaticity
- Vision
- Somatosensation
- Daily +

3/27/2017

Point by point...how you intervene

**Motor control neuroplasticity and BDNF:**

- As discussed – demand and supply
- Task specific
- Repetition-based
- MUST be challenged...and see progress

* Viable for WHICH Parkinsonisms?

3/27/2017

Point by point...how you intervene

**PSYCHOLOGICAL**

- Understand that the brain can change
- Understand that I can improve with PD
- SEE that I have improved – USE measurements
- Know that challenge = opportunity to improve
- Knowing, “I will do no harm with this activity”

3/27/2017

OUTCOME MEASURES IN PD AND PD+
Common outcome measures

- Four square step test
- 2/6 min walk test and gait velocity
- Modified Dynamic Gait Index and FGA
- Timed Up & Go
- BEST + Mini BEST
- Berg Balance
- Sit to stands
- Activity-specific Balance Confidence (ABC scale)
- Freezing of Gait (FOG)

Frail PD and PD+ patients:
MEASUREMENT

- Objective recordings that can be reproduced to prove real changes within a patient’s case
- Bed mobility
- 5x sit to stand
- Minimal Chair Height Stand Test (MCHST)
- Unassisted sit to stand height
- 10’ w/c propulsion
- Standing endurance
- QUIP* (see BONUS section)

Frail patient considerations

- Psychology of rehabilitation
- Nutritional considerations
- Evidence and recommendations: ACSM
- Provide body weight support to allow for endurance improvements
- Build RESOURCES, then function
Deep Brain Stimulation: Severe Symptoms PD

• Surgery (stimulation Putamen or Globus Pallidus) may improve function
  – Does not stop progression
  – May still need to be on medications
  – Improves level of function to best PD medication control
  – Risks of surgery and battery management
  – Interference with pacemakers

Evidence in exercise and PD

Resistance exercises can increase strength and ↓ falls
ROM exercises improve flexibility
Balance exercises improve balance and ↓ falls
Aerobic exercises improve cardio-pulmonary↓ pain, improve cognition and decrease depression.
Auditory, visual, rhythmic and learning based exercises improve sensory processing and cognition

Have we been INTENSE enough?
(Keus et al, 2009; Niewboer 2016; Horak 2016; Morris 2014)
Principles of Motor Learning in DT

- Task specificity
- Variability
- Feedback (prioritization)
- Intensity
- Increasing levels of difficulty


Compensatory efforts: Adaptive

- Walkers
- Canes
- Walking poles/trekking poles

Compensatory efforts: Sensory Cues

- Environmental changes: Flooring, doorways, transitions, etc.
- Visual: Virtual, mobile, stationary
- Auditory: Metronome, music, other
The Immediate Effect of Attentional, Auditory, and a Combined Cue Strategy on Gait During Single and Dual Tasks in Parkinson’s Disease

1) Attentional Focus (single) on step length
2) Attentional Focus (single) on following the auditory cue
3) Attentional Focus (dual) on step length with auditory cue

Conclusion: Improved in 1, 2, and no additional benefit in the combination (3) when tested under single and DT conditions


A Training Program to Improve Gait While Dual Tasking in Patients With Parkinson’s Disease: A Pilot Study

1. Usual walk baseline
2. Verbal fluency: Max words beginning with a predefined letter, during 1 minute of walking
3. Serial 3 subtractions
4. Mindstreams +/- 4 in simple arithmetic
5. Complex conversation DT in retention test
6. Usual walking with no DT: fatigue assessment

Can people with Parkinson's disease improve dual tasking when walking?

High intensity interventions

Training for:

- Power
- Direction change
- Gait speed
- Reactive balance
- Available range of motion
- Endurance
- Motivation and neuroplasticity

Equipment considerations

- Hydrotherapy
- Passive exercycle/theracycle
- Visual aids and assistive devices
- Training for power and speed
- Body Weight Supported Treadmill Training
- BioStep Sit to Stand

Future research and trends in PD

Motivation and learning: OPTIMAL

Optimizing
Performance
Through
Intrinsic
Motivation
Attention
Learning
Meaningful Goals:

- Thorndike (1911) “A connection is significantly modified only if its activation is associated with outcomes important to the animal's behavior.”
  *Law of Effect*
- Important outcomes are those that are meaningful to the patient
- ...you will not get patients to participate in intensive practice unless their participation is meaningful

Questions

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BONUS material

These slides are intended to extend your experience:

- Further interest in a particular area
- Future courses, research
- Answering unanswered questions

More on physiology and networks
Reward systems (dopaminergic)

Meaningful Goals: OPTIMAL

- Optimizing
- Performance
- Through
- Intrinsic
- Motivation (and)
- Attention (for)
- Learning

Meaningful Goals: OPTIMAL

- Enhanced expectancies for future performance
- Support for client autonomy
- External focus of attention
- Pairing motor practice with conditions that boost confidence and outcome expectations
- These conditions align thoughts, motivation, attention, and neural and neuromuscular systems to the performer’s goals.
Meaningful Goals: OPTIMAL

- Dopaminergic reward loop based on expectations and previous experiences

- Consider pathophysiology – WHICH PD+ conditions is this viable for?

Parkinson’s Specific: Forced vs. Voluntary Aerobic Ex

- **BOTH**: VO2 max improved 17% for FE and 11% for VE

- **ONLY Forced Ex. improvements**
  - Improved cardinal motor signs for FE (41% in rigidity)
  - Improvement in tremor (38%)
  - Improvement in bradykinesia (28%)
  - Bimanual motor dexterity (grip coupling), grip force and digit placement for maintained for 4 weeks
  - UPDRS rating improved by 35% for FE and no improvements for VE (p<0.002)
Is moderate intensity enough in PD?

- General moderate aerobic exercise maintains mobility and balance in patients with PD
  (Smidt et al, 2005; Goodwin et al, 2008; Suchowersky et al, 2006; Suchowersky et al, 2008; Dibble et al, 2009)
- Reduced fall rates
- Increases dopamine synthesis in available dopaminergic cells (Sutoo, 2003)

Point by point... how you intervene

STRENGTH

- Function and falls
- Resistance tolerated 8-12 reps
- 2-3 sets
- 3-4 days/week
- Expect soreness
- Perceived exertion drives intensity

Muscular endurance

Resistance 15-20 repetitions

- Multiple sets
- 3-4 days/week
- The art of cumulative effects
- Consecutive order for sets?
- Perceived exertion drives intensity
Point by point...how you intervene

Cardiovascular endurance:

- Sustained activity, whole body as able
- 30 minutes
- 10 minutes, 3 +/-day acceptable (cumulative)
- 4-7 days/week
- The art of cumulative effects
- Interest, experience, music...what drives your intensity?

NON-MOTOR SYMPTOMS IN PD

Non-Motor Symptoms

- Cognition
- Psychiatric
- Autonomic
- Sensory
- Sleep

Non-Motor Symptoms

- Cognition – Attention, memory, and problem-solving/safety
- Psychiatric
- Autonomic
- Sensory
- Sleep
Non-Motor Symptoms: Cognition

- Attention leading to functional difficulties in:
  - Memory
  - Reaction speed
  - Dual task tolerance
  - Problem-solving/safety

Integrative Dual Task Aerobic PD Exercise Program

- Neurofit activities
- Running and gliding on BWSTT
- Coordination and balance exercises
- Game type activities: basketball, boxing, badminton
- Integrating technology: neuromuscular stimulation

Cognitive Changes

- **Structures** – SN, caudate and the DLPFC
- **Incidence** – 30% of PD population
- **Presentation** - Higher in those with flat affect, hearing loss, retropulsion, and higher fall rates

Cognitive Changes

- Attention
- Memory
- Dual task capacity
- Psychiatric: paranoias, depression
Non-Motor Symptoms

- Cognition
- Psychiatric – Paranoia, depression
- Autonomic
- Sensory
- Sleep

Non-Motor Symptoms: Psychiatric

- Paranoia, depression
- Leading to caregiver distrust and combativeness
- Medication noncompliance
- Reduced exposure to high intensity exercise

Non-Motor Symptoms

- Cognition
- Psychiatric
- Autonomic – Urinary incontinence, Hypotension – MORE common in PD+
- GI, endocrine
- Sensory
- Sleep

Non-Motor Symptoms: Autonomic

- Urinary incontinence
- Orthostatic Hypotension*
- GI, endocrine slowing
Non-Motor Symptoms

- Cognition
- Psychiatric
- Autonomic
- Sensory - pain, tingling and burning, olfactory loss, hypersensitivity (pressure, DOMS)
- Sleep

Non-Motor Symptoms: Sensory Dysfunction

- Sensory abnormalities and losses:
  - Smell
  - Foveal color vision/contrast sensitivity
  - Verticality/perception of upright
  - Pain
    - Higher incidence and risk of shoulder pain: 21x more common than age matched subjects

Non-Motor Symptoms: Sleep

- Nightmares acted-out, restlessness
- Initial sx with many
- RLS
- Medications
- Prognostics
Unified Parkinson’s Disease Rating Scale

• Part I: Non-motor Aspects of Experiences of Daily Living
  — Cognition, sleep, GI, fatigue, pain
• Part II: Motor Experiences of Daily Living
  — Speech, ADL’s, tremor, freezing, gait speed, balance
• Part III: Motor Examination
  — Speech, rigidity, gait, freezing, posture, tremor

• Items score 0-4 (0=normal, 4=severe)  
  Goetz, 2007

Hoehn and Yahr Scale

Stage 0  No Symptoms
Stage 1  Unilateral symptoms only; mild symptoms
Stage 1.5 Unilateral + axial involvement; friends note change in posture/locomotion/facial
Stage 2  Bilateral symptoms; no change in balance; minimal disability
Stage 2.5 Mild bilateral disease; posture/gait affected, recovers on pull test
Stage 3  Mild/mod. bilateral disease; some postural instability; physically independent; 1 slowness
Stage 4  Severe disability; still can walk/stand unassisted; no longer able to live alone
Stage 5  Wheelchair or bed-bound unless assisted  
  Gancher, 2002

Unified Multiple System Atrophy Rating Scale

Part I: Functional score of symptoms and ability to undertake activities of daily living

Part II (neurological motor evaluation)

Composite Autonomic Symptoms Scale (COMPASS): a measure of autonomic symptoms and autonomic functional status
Frail patient measurement: QUIPS

QUIPS =

QUantifying
Independence
Performance
Scale

Time x assistance for BASIC mobility

Quantification of Independence and Performance Scale

Supine to sit
Sit to supine
Stand pivot transfer

Time (in seconds)

x Assistance = FIM scale

Total score

Studer, M – in publication, 2017