

Autonomic Symptom Identification, Management and Treatment in Parkinson's Disease and Parkinsonism

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AMDAPP
Association of Movement Disorder Advanced Practice Providers

Katie Henke – Relevant Financial Relationships

- No disclosures to report.

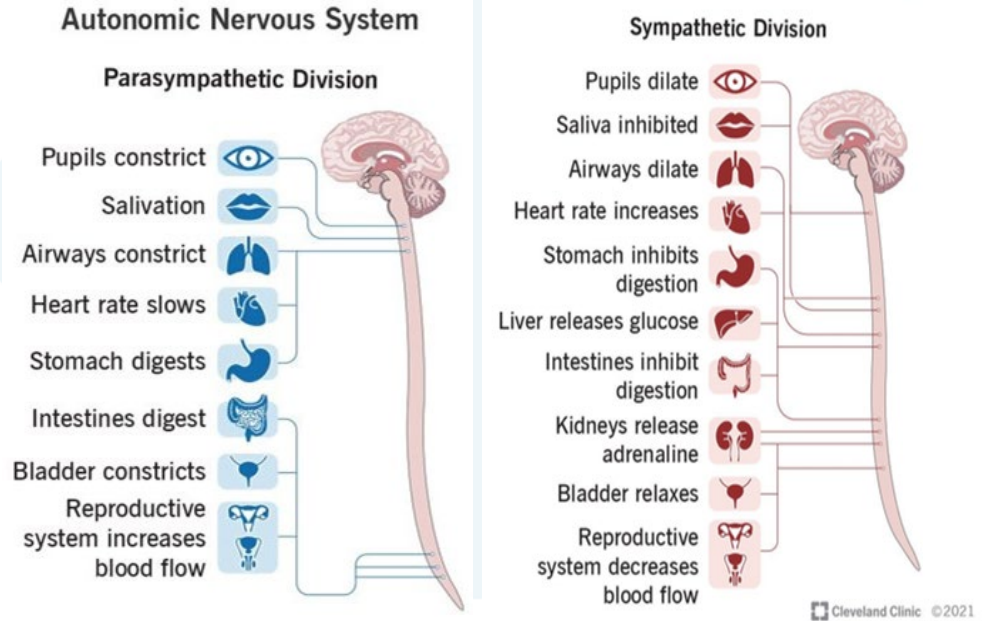
All relevant financial relationships have been mitigated

Objectives

- Review the autonomic nervous system (ANS)
- State the prevalence of autonomic dysfunction in Parkinsonism
- Highlight the importance of autonomic dysfunction detection in Parkinsonism
- Describe how to test and measure the autonomic nervous system
- Explore the most affected systems to differentiate manifestations in patients with Parkinsonism
- Identify non-pharmacological and pharmacological treatments options
- Differentiate the autonomic involvement in multiple system atrophy (MSA), progressive supranuclear palsy (PSP), and Parkinson's Disease

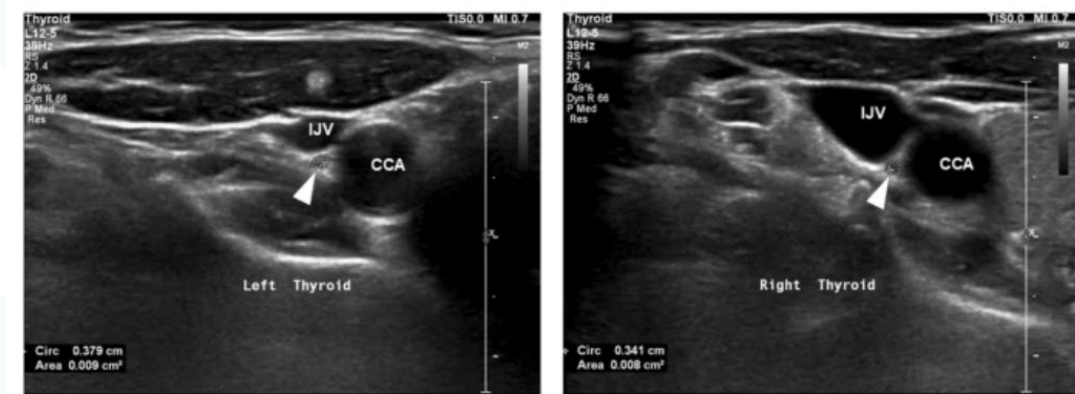
Autonomic Nervous System

- Central Nervous System
- Peripheral Nervous System
 - Sympathetic (fight or flight)
 - Parasympathetic (rest and digest)
 - Enteric (digestion regulation)



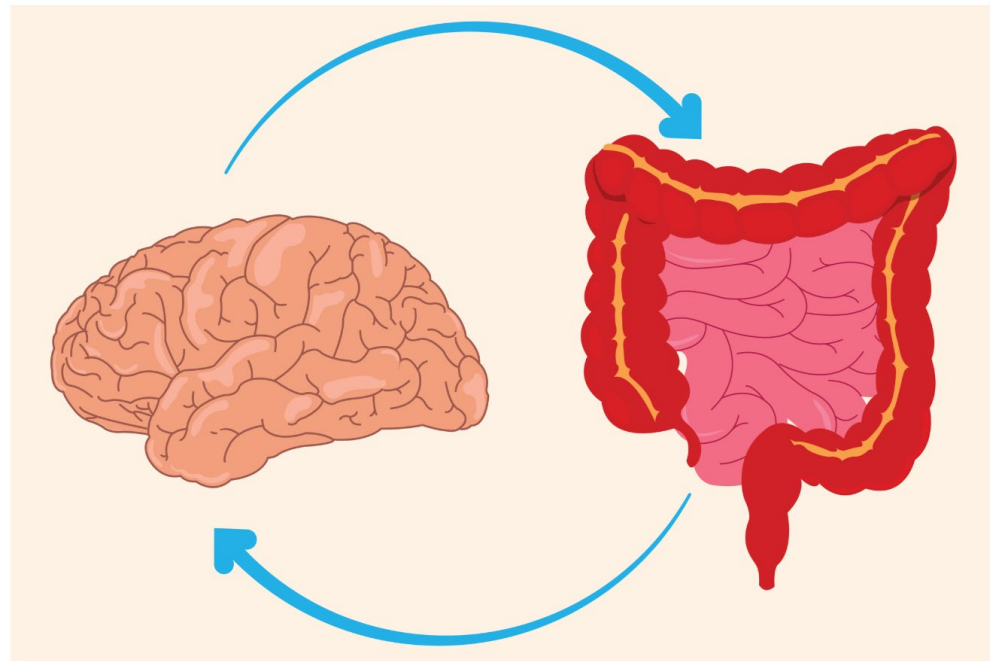
The Vagus Nerve

- The **10th cranial nerve (CN X)** and the **longest cranial nerve**
- Part of the **parasympathetic nervous system** (“rest and digest”)
- Extends from the **brainstem** to the **neck, chest, and abdomen**
- Links the brain with major organs: **heart, lungs, stomach, intestines, liver, pancreas**
- Key pathway of the **gut–brain connection**



The Vagus Nerve in Parkinson's Patients

- Shown by ultrasonography to be **atrophic** in recent studies
 - Small right vagus nerve CSA is associated with early parasympathetic dysfunction
- Suggested to be a **pathway** for alpha-synuclein in PD (gut-brain axis)
- Vagus nerve ultrasound may serve as a non-invasive biomarker for prodromal autonomic involvement



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Autonomic Dysfunction in Parkinsonism

Prevalence of Autonomic Dysfunction in Parkinsonism

- Autonomic symptoms are among the most common non-motor manifestations in PD
- Highly prevalent, affecting 63-91% of patients
- They severely impact quality of life
- Increase morbidity and mortality
- Autonomic dysfunction typically worsens progressively, with overall severity increasing by approximately 20-23% annually in the first years after diagnosis, though progression may plateau in later years

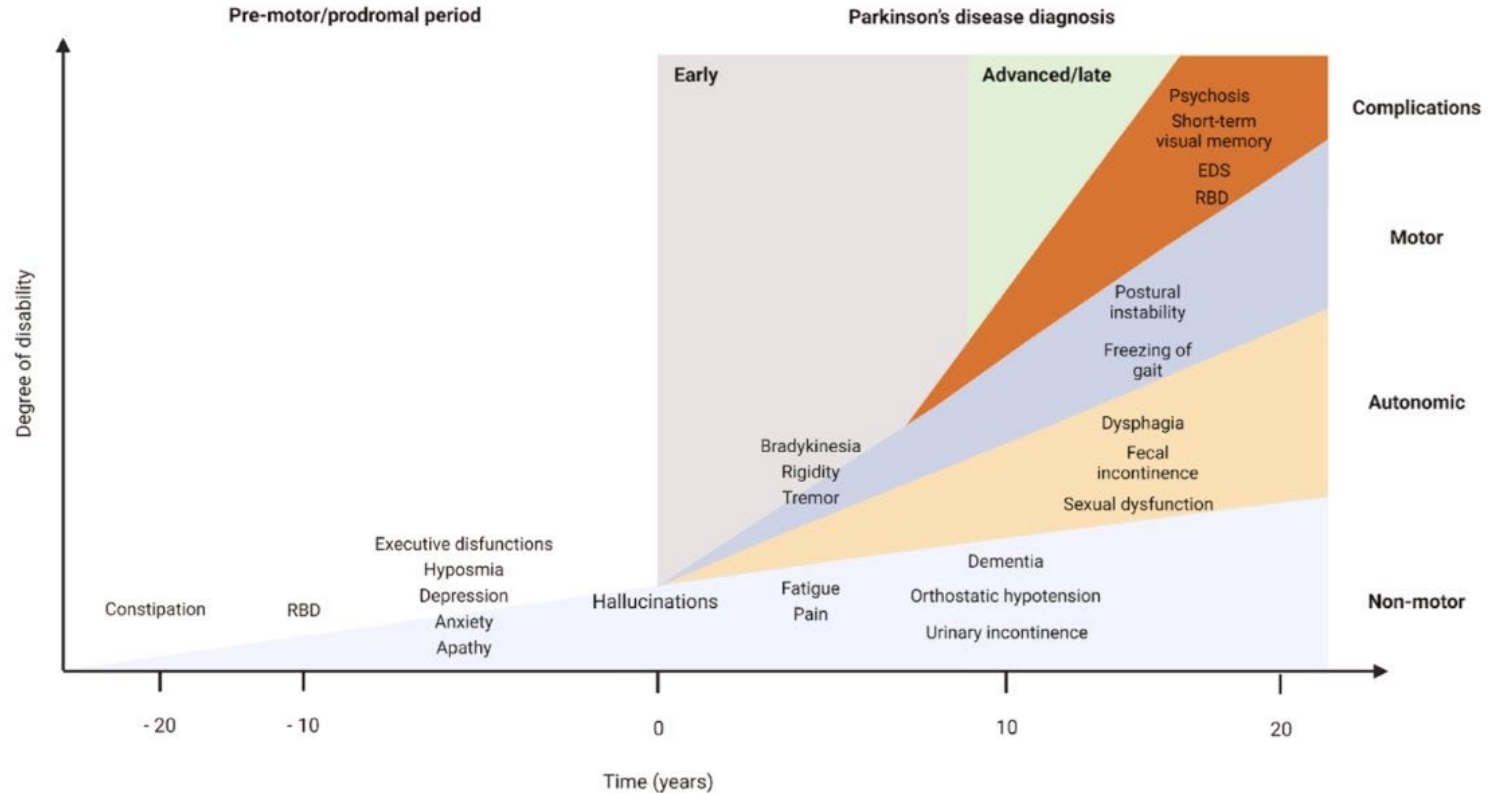
Parkinson's Disease Symptom Onset and Progression

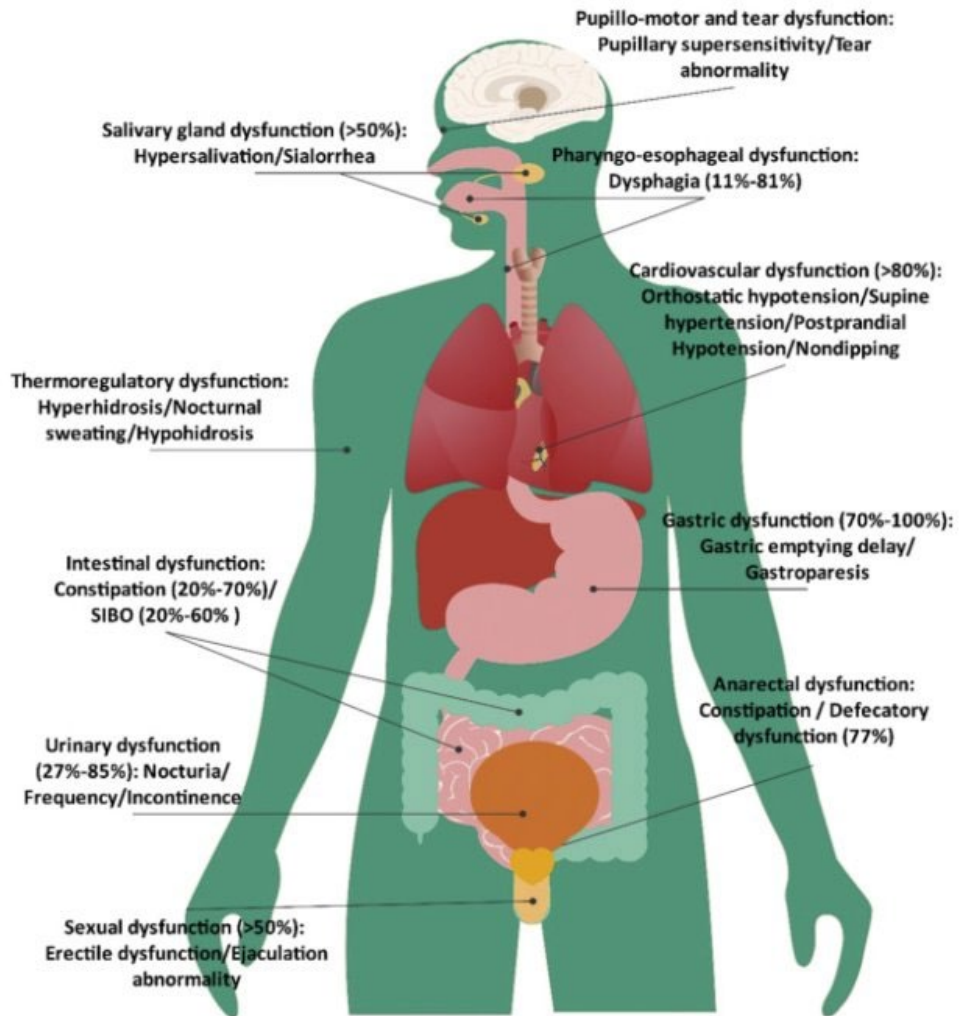
Table 1

Description of the premotor, nonmotor and motor symptoms of Parkinson disease.

Premotor Symptoms	Nonmotor Symptoms: Neuropsychiatric	Nonmotor Symptoms: Cognitive	Nonmotor Symptoms: Autonomic	Nonmotor Symptoms: Sleep Disorders	Nonmotor Symptoms: Sensory Abnormalities	Cardinal Motor Symptoms
Constipation	Depression	Executive dysfunction	Orthostatic hypotension	Insomnia	Anosmia	Tremor
Anosmia	Anxiety (mood disorders)	Memory loss	Constipation	Somnolence	Pain	Rigidity
Rapid eye movement (REM) sleep behavior disorder	Apathy	Dementia	Fecal incontinence	Excessive daytime sleepiness	Ageusia	Bradykinesia (or akinesia)
Depression	Impulsive control disorder		Nausea	Restless legs syndrome	Numbness	Postural instability
	Psychosis		Vomiting	Sleep attacks	Paresthesia	Gait disorder
	Anhedonia		Drooling	Periodic limb movements of sleep		
	Hallucinations		Urinary incontinence and urgency	REM sleep behavior disorder		
	Abulia		Sexual dysfunction	Vivid dreaming		
	Attention deficit disorder		Altered cardiac reflexes			
	Panic attacks		Olfactory dysfunction			

Chronology of Parkinsonism





Testing Autonomic Dysfunction in Parkinsonism

Autonomic Testing

Very few autonomic centers and testing facilities in the nation

There are still many other ways...

- Heart rate
- Heart rate variability
- Tilt table testing
- Catecholamine testing
 - Baseline serum, but supine to standing is more specific
- Orthostatic vital sign measurement
- Gastric emptying study
- Exercise tolerance testing
- Swallowing evaluation
- Urodynamic studies
- Post-void residual studies



Scales for Testing Autonomic Dysfunction

Table 1

The scales for the evaluation of autonomic dysfunction in PD.

Autonomic dysfunction	Scales	References
Global evaluation	SCOPA-AUT	Evatt et al. (2009)
Sialorrhea	Non-motor Symptoms Questionnaire	Evatt et al. (2009)
	Drooling Severity and Frequency Scale	
	Drooling Rating Scale	
Dysphagia	Sialorrhea Clinical Scale for PD	Evatt et al. (2009)
	Swallowing Disturbance Questionnaire	
	Dysphagia-Specific Quality of Life scale Swallowing Clinical Assessment Score	
Constipation	Rome III criteria or Rome II criteria	Evatt et al. (2009)
Orthostatic hypotension	SCOPA-AUT	Pavy-Le Traon et al. (2011)
	Composite Autonomic Symptom Scale	
	Orthostatic Grading Scale	
	Novel Non-Motor Symptoms Scale	
Urinary dysfunction	Danish Prostatic Symptom Score	Pavy-Le Traon et al. (2018)
	International Consultation for Incontinence Questionnaire for Male Lower Urinary Tract Symptoms	
	Overactive Bladder Questionnaire (OABq)/OABq Short Form/8-item OABq score/OAB Symptom Score	
Sexual dysfunction	Quality of Sexual Life Questionnaire	Moore et al. (2002)
	Arizona Sexual Experiences Scale	
	Sexual Dysfunction Inventory	

Scales for Outcomes in Parkinson's disease - Autonomic Dysfunction (SCOPA-AUT)

Acronym: SCOPA-AUT

Authors: Visser M, Marinus J, Stiggebout AM, Van Hilten JJ

Year Published: 2004

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Estimated Time to Complete: 10-15 minutes

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International Parkinson and
Movement Disorder Society

SCOPA-AUT

Scales for Outcomes in Parkinson's disease - Autonomic Dysfunction

Most Common Autonomic Symptoms in Parkinsonism

Most Common Autonomic Symptoms in Parkinsonism

Cardiovascular

- Orthostatic and postprandial
- hypotension
- Supine hypotension/hypertension

Gastrointestinal

- Dysmotility
- Gastroparesis
- Dysphagia
- Constipation

Urinary

- Storage and voiding problems
- Urgency and nocturia

Sexual

- Erectile dysfunction
- Vaginal dryness
- Difficulty achieving orgasm

Cardiovascular Autonomic Dysfunction in Parkinsonism

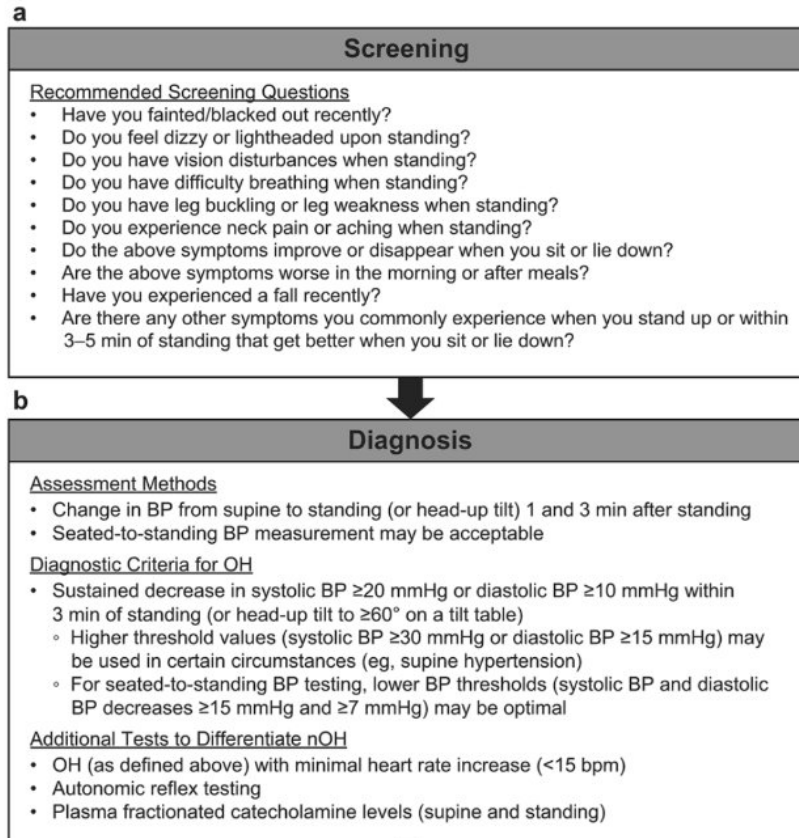
Cardiovascular Dysfunction –Orthostatic hypotension

- Orthostatic hypotension is present in 35.8% of patients with PD
- Defined by a reduction of systolic blood pressure of ≥ 20 mmHg and/or diastolic BP (DBP) of ≥ 10 mmHg within 3 mins of standing or head-up tilt from a supine position
- One of the most disabling dysautonomias in Parkinson's
- Associated with more rapid motor, non-motor and cognitive decline
- Premature mortality
- Infrequently reported by patients

Table 1 Neurogenic orthostatic hypotension and variants [5, 9, 10, 13–19]

Parameters	Classical		Temporal	
	nOH	Non-neurogenic OH	Initial OH	Delayed OH
Definition	Sustained BP decrease (systolic ≥ 20 mmHg or diastolic ≥ 10 mmHg) within 3 min of standing		Transient BP decrease (systolic ≥ 40 mmHg or diastolic ≥ 20 mmHg) that occurs within 15 s of standing and resolves within 30–60 s	BP decrease (systolic ≥ 20 mmHg or diastolic ≥ 10 mmHg) > 3 min after standing
Clinical features	Lightheadedness, fatigue, weakness, visual disturbances, head/neck pain, syncope		Lightheadedness, visual disturbances occurring a few seconds after standing and lasting < 30 s	Prolonged prodrome of lightheadedness, fatigue, weakness, visual disturbances, syncope
Mechanisms	Inadequate compensatory SVR increase (due to ANS impairment) upon assuming an upright position, causing blood pooling in the lower body and leading to diminished CO	Inadequate compensatory BP regulation reflex adjustments (i.e., increased SVR and CO) upon assuming an upright position owing to severe volume depletion	Transient imbalance between CO and SVR when moving to a standing position	Slow, progressive impairment of SVR with no appreciable change in CO upon assuming an upright position
Causes	Sympathetic noradrenergic failure associated with neurodegenerative diseases (e.g., Parkinson disease, multiple system atrophy, pure autonomic failure) and peripheral neuropathies	Medications (e.g., diuretics, vasodilators) Hypovolemia (e.g., dehydration) Cardiac pump failure	Young individuals with asthenic features Medications (e.g., vasoactive or psychiatric agents)	Mild or early sympathetic adrenergic failure

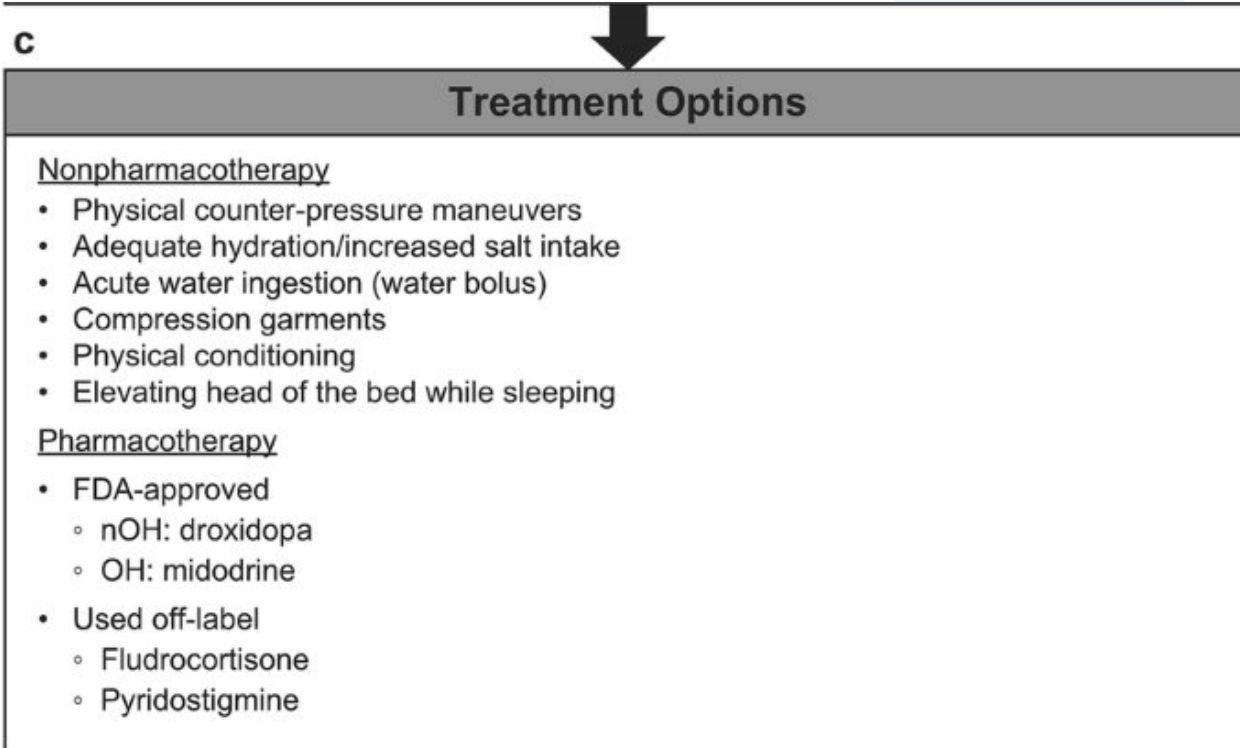
Cardiovascular Dysfunction –Orthostatic hypotension



Orthostatic Vital Sign Measurement

	Blood Pressure	Heart Rate	Time	Symptoms
Supine (after 5 mins)				
Immediately upon standing				
1 min				
3 mins				
5 mins				
7 mins				
10 mins				

Cardiovascular Dysfunction –Orthostatic Hypotension Treatments



Medications for Orthostatic Hypotension

Midodrine 2.5 mg-15 mg PO TID

- vasoconstrictive properties
- Helpful for nOH
- Do not give within 4-5 hours of bedtime
- Side effects: scalp tingling, supine hypertension

Droxidopa 100-300 mg PO TID

- Sympathomimetic
- Helpful for nOH
- Side effects: hypertension, racing heart
- Expensive but financial assistance program for commercial insurance
- Available on CostPlus drugs
- Can add to Midodrine, but higher risk of supine HTN

Do not take 4-5 hours before rest

Atomoxetine (off-label use) 40-80 mg PO daily.

- Non-stimulant ADHD medication
- Must have catecholamine testing with evidence of norepinephrine levels >250 pg/mL
- Side effects: nausea, decreased appetite, tachycardia and hypertension
- Can help fatigue, brain fog

Fludrocortisone 0.1-0.2 mg PO daily

- Mineralocorticoid
- Easy dosing schedule
- Side effects: bloating, fluid retention
- Should have serial potassium levels drawn

Pyridostigmine 30-60 mg PO up to QID

- Peripheral acetylcholinesterase inhibitor
- Helpful for orthostatic intolerance and constipation
- Side effects: abdominal cramping, diarrhea, subjective weakness, sweating

Cardiovascular Dysfunction –Orthostatic Hypotension Clinical Pearls

- Waist trainer, shapewear or triathlon suits
- Counter-maneuvers
- Breathing out while standing up
- Acid reflux wedge for mattress
- Compression stockings must be at least thigh high or not helpful
- Oral fluid bolus of 16-20 ounces quickly
- Keep an abdominal compression device in clinic to demonstrate with patient the difference
- PT can be helpful for lower extremity strengthening and imbalance
- Oral rehydration salts
- Shower chairs



Medication-Induced Orthostatic Hypotension

Table 2 Common medications used to treat Parkinson disease that may exacerbate neurogenic orthostatic hypotension

Mechanism of action	Medication	Magnitude of systolic BP drop (mmHg)	Magnitude of diastolic BP drop (mmHg)
Dopamine precursor	Levodopa [29, 88, 89, 91, 92, 100]	8.0 to > 20.0	2.1–5.0
Dopamine agonist	Bromocriptine [93, 97]	16.1	2.8
	Pramipexole [95, 96]	Unknown	Unknown
	Ropinirole [90, 99]	4.2	Unknown
Monoamine oxidase inhibitor	Selegiline [93, 94, 101]	12.5–19.0	5.0–5.2
	Rasagiline [98]	Unknown	Unknown
<i>N</i> -Methyl-D-aspartate receptor antagonist	Amantadine [102–104]	Unknown	Unknown

Start with reduction of dopamine agonists since they most often have other side effects

Cardiovascular Dysfunction - Supine Hypertension

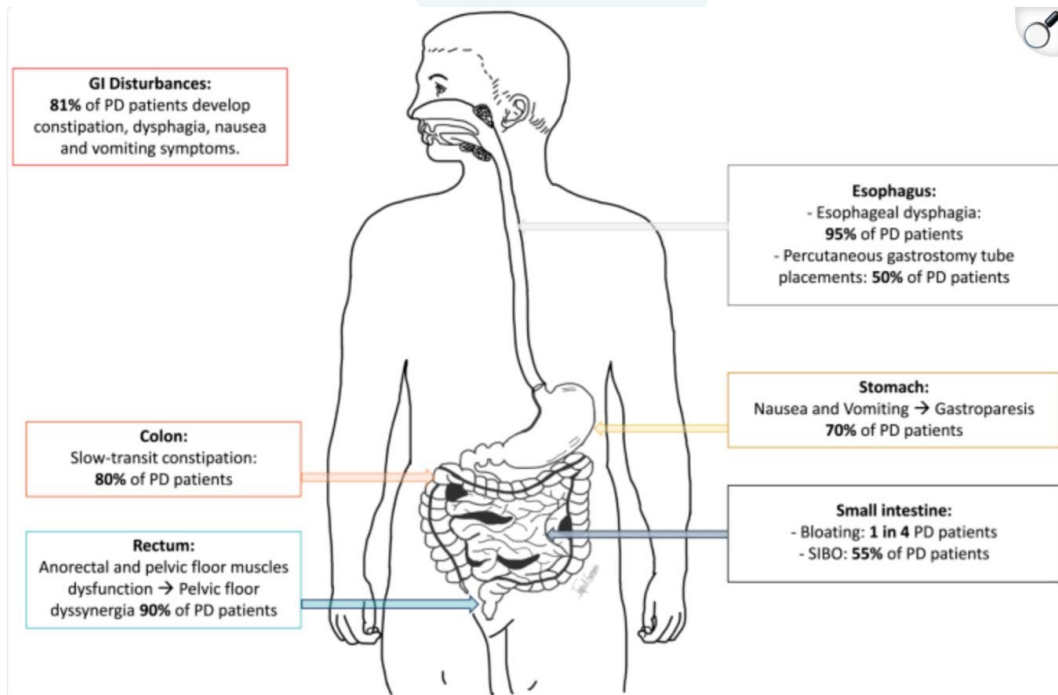
- Estimated to be present in approximately one-third to one-half patients with nOH related to parkinsonism
- Supine hypertension has been defined by consensus criteria as a systolic BP of ≥ 140 mmHg and/or a diastolic BP of ≥ 90 mmHg after ≥ 5 min of supine rest
 - Morbidity and mortality impacts are not nearly as well understood as OH
 - Renal impairment
 - Left ventricular hypertrophy
 - Arterial stiffness
 - Essential hypertension
- Avoid supine position at rest
- Patient should not lie flat for at least 3-4 hours after any dose of midodrine or droxidopa. Not the case for atomoxetine, fludrocortisone or pyridostigmine
- **A heating pad applied to abdomen when supine can significantly reduce hypertension**
- Short-acting anti-hypertensive
 - Clonidine PO
 - Hydralazine
 - Losartan
 - Nitroglycerin patch

Extra caution upon waking

Gastrointestinal Autonomic Dysfunction in Parkinsonism

Gastrointestinal Dysfunction

"The frequency of gastrointestinal symptoms is very high in PD, even during the premotor phase of the disease. It has been reported that 88.9% of PD patients will develop gastrointestinal symptoms prior to the onset of Parkinsonian motor symptoms." (Chen et al., 2020)



Gastrointestinal Dysfunction

Heartburn, nausea, vomiting

- Central preganglionic AD
- Peripheral postganglionic parasympathetic dysfunction
- Neurodegenerative changes in the peripheral autonomic ganglia and enteric nervous system

Constipation

- Central preganglionic AD
- Peripheral postganglionic parasympathetic dysfunction
- Neurodegenerative changes in the peripheral autonomic ganglia and enteric nervous system

*Drugs with anticholinergic effect

- Anticholinergics
- Tricyclic antidepressant
- Opiates
- L-dopa can exacerbate some symptoms, such as delayed gastric emptying, by slowing down motility through its effect on dopaminergic enteric receptors

FIGURE 4.

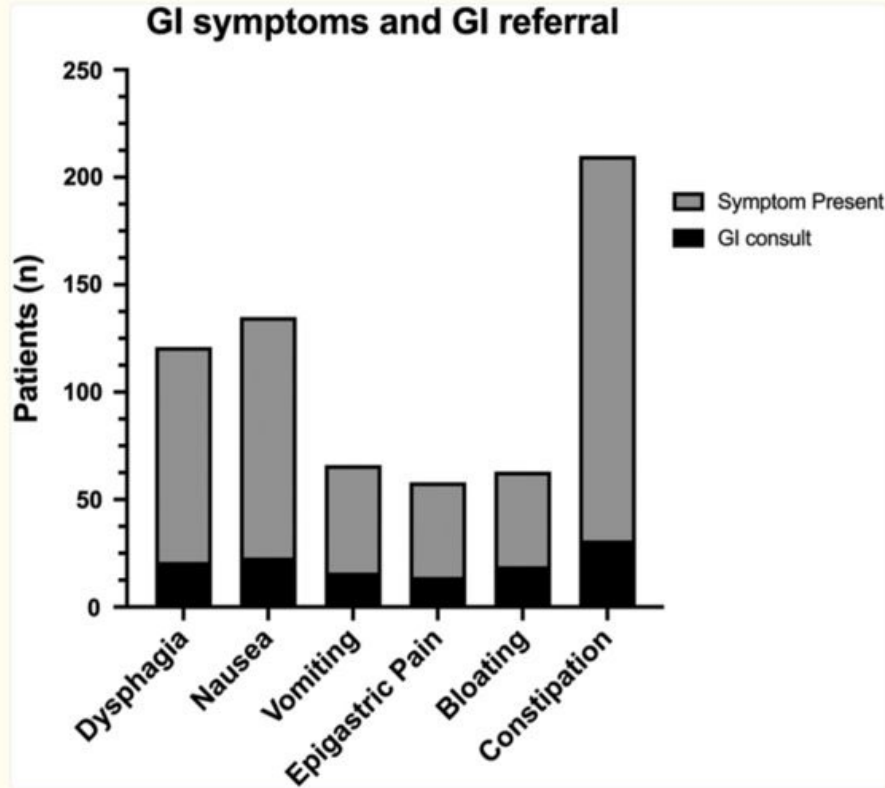



TABLE 1.

Common GI Medications Contraindicated in Parkinson's Disease

Medication	Mechanism of action	Purpose
Prochlorperazine (Compazine)	Blocks central D2 receptors	Anti-emetic
Promethazine (Phenergan)	Blocks central D2 receptors	Anti-emetic
Metoclopramide (Reglan)	Blocks central D2 receptors	Anti-emetic
Buspirone (Buspar)	5HT1A receptor partial agonist, weak D2 antagonist	Anxiolytic, used off-label for nausea
Haloperidol (Haldol)	Blocks central D2 receptors	Typical antipsychotic, used off-label for nausea
Olanzapine (Zyprexa)	Blocks central D2 receptors and serotonin receptors. Can dissociate more quickly than typical antipsychotics.	Atypical antipsychotic, used off-label for nausea
Amoxapine (Asendin)	Blocks central D2 receptors in addition to cholinergic, muscarinic, and histaminergic receptors.	Tricyclic antidepressant, used off-label for chronic abdominal pain, visceral hypersensitivity

Gastrointestinal Dysfunction - Treatments

▶ J Clin Gastroenterol. 2024 Jan 4;58(3):211–220. doi: [10.1097/MCG.0000000000001961](https://doi.org/10.1097/MCG.0000000000001961) 

Management of Gastrointestinal Symptoms in Parkinson's Disease

A Comprehensive Review of Clinical Presentation, Workup, and Treatment

[Trisha S Pasricha](#) ^{*,†,✉}, [Ingrid L Guerrero-Lopez](#) [‡], [Braden Kuo](#) ^{*,†}

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PMCID: PMC10855995 PMID: [38260966](https://pubmed.ncbi.nlm.nih.gov/38260966/)

Goal is to alleviate symptoms, correct malnutrition and manage fluid intake

- Diet high in fiber
- Diet low in fats
- Small frequent meals
- Regular exercise
- Abdominal massage
- Remove drugs that worsen condition

Medication options

- Probiotics
- Fiber supplements
- Stool softeners
- Per GI consultation (magnesium, lactulose)
- Fecal microbiota transplantation

(Palma & Thijs, 2023; Pasricha et al., 2024)

Urinary Autonomic Dysfunction in Parkinsonism

Urinary Dysfunction

- 27-87% of patients with parkinsonism experience urinary dysfunction
- *"Detrusor overactivity is the most common urinary problem in PD and is characterized by urinary frequency, urgency, and nocturia, with a sensation of bladder fullness and an urge to void before appropriate bladder filling has occurred"* (Pfeiffer, 2020).
- Lower urinary tract
 - Nocturia*
 - Urgency
 - Incontinence
- Post-void residual bladder volume can diagnose incomplete bladder emptying
- Urodynamic studying for detrusor overactivity
- Management is crucial to avoid the risk of urosepsis and death

Urinary Dysfunction

- The non-pharmacologic goal is to alleviate urinary dysfunction
 - Self-monitoring with a diary
 - Daily pelvic floor muscle exercises
 - Minimize caffeine and alcohol intake
 - Treat constipation
 - Head up sleeping can reduce nocturia
 - Bedside commode or urinal
 - Assessment for fluid restriction in patients with nOH
 - Urge suppression
 - Distraction techniques
- Pharmacologic treatments
 - Anticholinergic drugs acting on muscarinic receptors in the bladder
 - Nonselective and selective anticholinergic drugs

Urinary Dysfunction - Treatments

Table 1
Treatment options for overactive bladder symptoms including urinary urgency, frequency, and nocturia²⁷

Medication	Mechanism of Action	Dosing Instructions	Side Effects
Mirabegron	Adrenergic beta 3 agonist	Start 25 mg once daily. After 8 wk can increase to 50 mg once daily Can be used concomitantly with solifenacin 5 mg once daily	Dizziness, diaphoresis, hypertension, irregular heart rate, abdominal or pelvic pain
Solifenacin	Anticholinergic	Start 5 mg once daily. Can increase to 10 mg once daily	Xerostomia, constipation, blurred vision, urinary retention, nausea, dyspepsia
Oxybutynin	Anticholinergic	Immediate release: 2.5 mg BID up to 5 mg QID Extended release: 5–15 mg once daily	Xerostomia, constipation, blurred vision, urinary retention, nausea, dyspepsia
Tolterodine	Anticholinergic	Immediate release: 2 mg BID Extended release: 2–4 mg once daily	Xerostomia, constipation, blurred vision, dyspepsia, dizziness, urinary retention
Fesoterodine	Anticholinergic	4–8 mg once daily	Xerostomia, constipation, blurred vision, dyspepsia, dizziness, urinary retention
Trospium	Anticholinergic	Immediate release: 20 mg BID Extended release: 60 mg once daily	Xerostomia, constipation, dry eyes, headache, urinary retention
Posterior tibial nerve stimulation (PTNS)	Neuromodulation	Unilateral stimulation	
Onabotulinum toxin A	Blocks presynaptic release of acetylcholine	100–200 units total every 12 wk	Urinary retention

(Margolesky et al., 2020)

Autonomic Sexual Dysfunction in Parkinsonism

Sexual Dysfunction

- Frequently overlooked and not discussed
- Not well-studied
- Negatively affects quality of life for patients and their partners

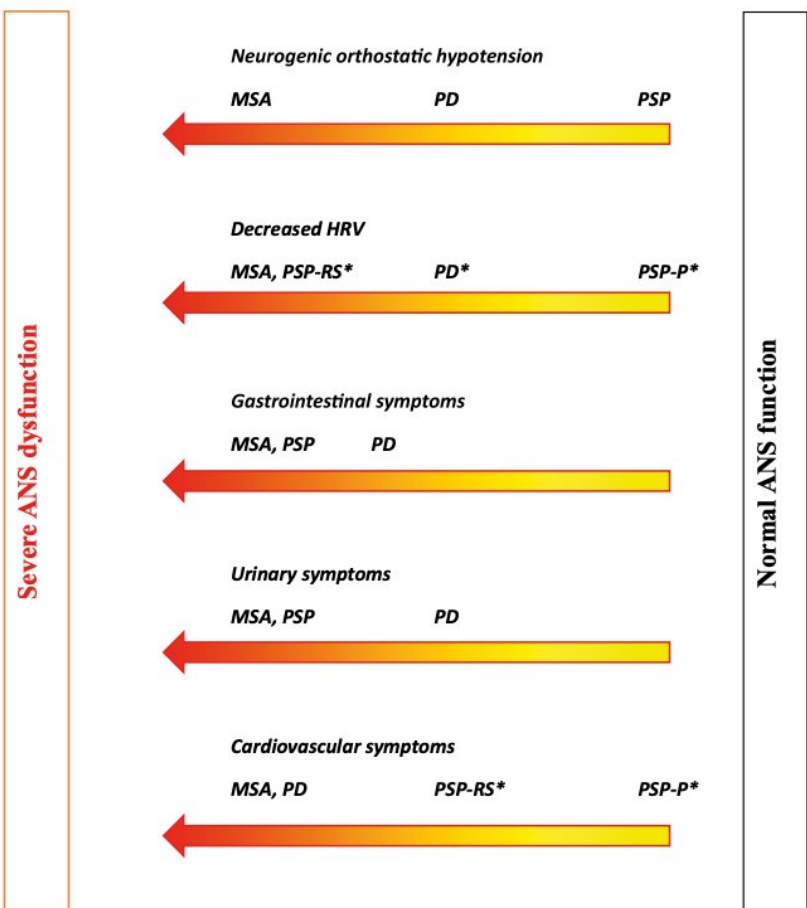
- Causes are multifactorial
 - Challenges with sexual expression due to motor, nervous, sensory and autonomic symptoms
 - Hormonal, psychological, and cognitive aspects
 - Prescribed and recreational drugs
 - Physiological events such as aging

- "PD medications should be optimized to maximize the motor requirements of sexual activity" (Margolesky et al., 2020)

Sexual dysfunction - Treatments

Table 2 Treatment of sexual symptoms				
Symptom	Medication	Mechanism of Action	Dosing Instructions	Side Effects
Erectile dysfunction	Sildenafil	PDE5 inhibitor	Start 50 mg once. May increase up to 100 mg up to once daily.	Hypotension, headache, flushing, dyspepsia, visual symptoms
	Tadalafil	PDE5 inhibitor	Start 10 mg once. May increase up to 20 mg up to once per 24 h. Alternative, for daily dosing: start 2.5 mg once daily. May increase to 5 mg once daily.	Hypotension, headache, flushing, dyspepsia, visual symptoms
	Vardenafil	PDE5 inhibitor	Start 10 mg once. May increase up to 20 mg up to once per 24 h.	Hypotension, headache, flushing, dyspepsia, visual symptoms
	Alprostadil	Prostaglandin E1	Start 1.25 mcg once. After 1 h may give 2.5 mcg once. After 24 h may give 5 mcg once. Maximum 2 doses per 24 h; maximum dose 60 mcg. May use more rapid titration for mixed causes (vascular/psychogenic/neurologic).	Priapism
	Mechanical devices	Mechanical/Structural	Varies based on device	Bruising, skin edema
Sexual dysfunction (women)	Sildenafil	PDE5 inhibitor	Start 50 mg once. May increase up to 100 mg up to once daily.	Hypotension, headache, flushing, dyspepsia, visual symptoms
	Flibanserin	Serotonin 5-HT1A agonist. Dopamine D4 and serotonin 5-HT2A, 5-HT2B, 5-HT2C antagonist.	Start 100 mg nightly. In premenopausal women, stop after 8 wk if no response.	Dizziness, nausea, insomnia, somnolence, anxiety, dry mouth
Sexual desire (men)	Testosterone	Androgen	30 mg every 12 h	Skin reactions, benign prostatic hypertrophy, testicular atrophy, hypertension, increased hematocrit, emotional lability

Autonomic Dysfunction in Parkinsonism vs PSP vs MSA



Severe ANS dysfunction

Normal ANS function

PD	MSA	PSP
Gradual onset of autonomic symptoms but can be up to 20 years prior to diagnosis	Severe autonomic dysfunction with rapid progression. Sporadic and fatal	No orthostatic hypotension but more postural instability
Alpha-synucleinopathy	Alpha-synucleinopathy	Tauopathy
SCoPA-AUT scores are similar to PSP	Non-responsive parkinsonian treatments	All domains of autonomic dysfunction
Demetia and psychiatric symptoms	Tends to have more constipation vs diarrhea	More dysphagia and constipation
	Very prominent urinary symptoms early on	

Questions?



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Thank you!



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