Title

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Journal

Parkinsonism & Related Disorders

ISSN

1353-8020

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Publication Date

2026-07-01

DOI

10.1016/j.parkreldis.2026.108905

Parkinsonism and Related Disorders xxx (xxxx) xxx

Contents lists available at ScienceDirect

Parkinsonism and Related Disorders

journal homepage: www.elsevier.com/locate/parkreldis

A framework for multidisciplinary management of autonomic dysfunction in Parkinson disease

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ARTICLE INFO

Keywords:
autonomic
Collaborative
Multidisciplinary
Parkinson

ABSTRACT

Autonomic dysfunction (AD) is present in nearly all people with Parkinson disease (PD), contributing to tremendous morbidity and mortality. Highly variable presentations including cardiovascular, gastrointestinal, urogenital, and thermoregulatory dysfunction can substantially affect daily function, safety, medication tolerance, and quality of life. Although autonomic symptoms are frequently encountered in neurologic practice, their management frequently extends beyond the traditional scope of neurologic care and may require input from multiple disciplines. Despite the increasing complexity of PD care and the need for coordinated multidisciplinary involvement, there are currently limited practical frameworks to guide specialist collaboration. Consequently, people with PD and their caregivers are often left to navigate fragmented care systems, conflicting recommendations, and uncertainty regarding which clinician should guide management. To help address these gaps, we provide a framework for the possible indications for specialist referrals and the potential roles of different healthcare practitioners in evaluating and treating AD. The manuscript also discusses the intersection between autonomic and neuropsychiatric manifestations in PD and offers practical clinical guidance for addressing this overlap. By providing a framework informed by coauthors in movement disorders, autonomic disorders, cardiology, nephrology, gastroenterology, urogynecology, physical therapy, and psychiatry, we hope to encourage neurologists – who often serve as the central point of contact – to collaborate closely and actively engage multidisciplinary team members in the management of these complex patients.

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https://doi.org/10.1016/j.parkreldis.2026.108905
Received 10 June 2026; Received in revised form 23 July 2026; Accepted 25 July 2026
Available online 29 July 2026

1. Introduction

Autonomic dysfunction (AD) of the nervous system is pervasive in Parkinson disease (PD) [1]. Just as abnormal aggregation of phosphorylated alpha-synuclein in the substantia nigra is associated with motor features, synuclein pathology throughout the peripheral and central autonomic nervous system is associated with AD in PD [1,2]. Given involvement of multiple organ systems, AD manifests with heterogeneous cardiovascular (CV), gastrointestinal (GI), urogenital, and thermoregulatory signs and symptoms. Furthermore, many of these features may begin years if not decades prior to the development of motor dysfunction. Signs and symptoms include orthostatic hypotension (OH), supine hypertension (SH), GI symptoms ranging from dysphagia to constipation, urinary frequency and urgency, sexual dysfunction, and heat and cold intolerance. As PD progresses, these features worsen, complicating management and leading to adverse outcomes like falls, hospitalizations, and increased mortality. Although disease-modifying therapies for AD are not currently available, symptomatic management can improve functional status, quality of life, and clinical outcomes [1,3,4]. Given the multisystemic involvement of AD, treatment decisions are greatly influenced by comorbidities and polypharmacy, which can lead to conflicting management priorities. Consequently, care frequently involves multiple simultaneous subspecialty consultations with parallel treatment plans, often creating confusion and barriers for effective multidisciplinary care coordination among patients, caregivers, and healthcare professionals.

2. Framework for multidisciplinary care

The aim of this paper is to highlight the complexity of AD and provide a framework for potential indications for specialist referral and the complementary roles different specialists can play in the multidisciplinary management of these complex patients. This topic was developed through the Parkinson Study Group Other Non-Motor Working Group (PSG-ONMWG), which brought together experts from multiple disciplines as co-authors, including movement disorders, autonomic disorders, gastroenterology, cardiology, nephrology, urogynecology, psychiatry, and physical therapy to collaboratively develop this practical framework for multidisciplinary care of AD symptoms in people with PD. In order to address multisystemic presentations of AD in PD, care is best delivered with the neurologist serving as the central "point person" for longitudinal care (Fig. 1). This role in the multidisciplinary team is critical since neurologists can best recognize when symptoms are PD-related versus attributable to comorbid or alternative etiologies. Importantly, since these symptoms may even develop before the diagnosis of PD, patients may be unaware of the association with their disease, requiring proactive screening and education from neurologists. Timely referral to other specialists is essential when autonomic features require sub-specialized expertise, testing, and/or interventions in managing refractory symptoms or co-morbid conditions. Some symptoms such as blood pressure (BP) management may be so complex that they require input from several subspecialists including cardiologists and nephrologists. It is critical to realize that no one physician can do it all; it requires a team. We hope that this framework will empower neurologists to effectively engage the expertise of other team members for the best care for this patient population.

Initial management strategies employed by the neurologist may include adjustment of dopaminergic and selected neuropsychiatric medications to reduce their impact on autonomic manifestations such as OH and GI symptoms [5,6]. Neurologists may also implement lifestyle interventions including dietary modifications, together with first-line pharmacologic therapies, early in the treatment course. When symptoms are refractory, complicated by comorbidities, or accompanied by specific red flag features, multidisciplinary collaboration may be indicated (Table 1, Fig. 2) [4,6–8]. The following sections describe the roles of various clinicians organized by AD domain (cardiovascular, gastrointestinal, urogenital), with an additional discussion of the implications of the overlap between autonomic and neuropsychiatric manifestations of PD. Key takeaways from each section are provided in Table 2.

3. Cardiovascular autonomic dysfunction

CV AD is often a disabling non-motor feature of PD, with both immediate and long-term consequences. Key clinical manifestations include neurogenic OH, SH, and postprandial hypotension (PPH) [9]. The reported prevalence of neurogenic OH in people with PD varies

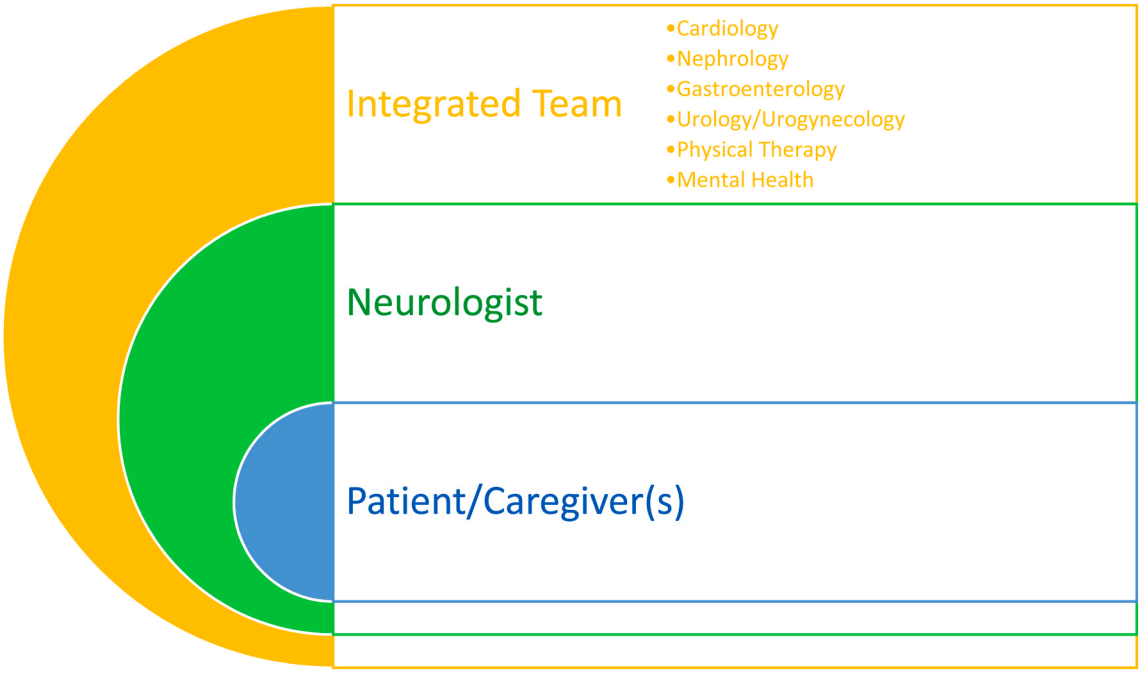

Fig. 1. Proposed model for collaborative patient-centered management of AD in PD. Neurologist serves as the point person, integrating collaboration with subspecialists & maintaining longitudinal oversight.

Table 1
Clinical scenarios that may warrant collaborative management of autonomic dysfunction in Parkinson disease.

Clinical Scenario	Collaborating Discipline(s)	Indication(s) for Involvement
Symptomatic OH	PT	1. Counterpressure training 2. Educate on abdominal binders, compression garments 3. Exercise regimen adaptations (for OH/chronotropic incompetence)
OH with CV or renal disease[a]	Cardiology, nephrology	1. Adjust medications for comorbidities that exacerbate OH 2. Advise on medications contraindicated in OH
Supine or nocturnal hypertension	Cardiology	1. Prescribe antihypertensive medications 2. Define appropriate BP goals
Orthostatic intolerance or syncope with features atypical of neurogenic OH[b]	Cardiology	1.Further work-up (e.g., electrocardiogram)
Esophageal dysphagia	Gastroenterology	1. Further work-up (e.g., esophagram, esophageal manometry, upper endoscopy)
Symptoms of gastroparesis with red flag features[c]	Gastroenterology	1. *H pylori* testing/treatment as appropriate 2. Further testing (e.g., upper endoscopy) 3. Medications (e.g., proton pump inhibitor for dyspepsia)
Constipation refractory to conservative measures & initial medications	Gastroenterology, PT	1. Additional medications (e.g., prokinetic or prosecretory agents, rectal enemas) 2. Pelvic floor and/or biofeedback therapy
Urinary frequency/urgency with red flag features[d]	Urology/urogynecology	1. Evaluate PVR and other urodynamic testing 2. Consider intermittent self-catheterization or suprapubic catheter 3. Consider antibiotic for recurrent UTI
Urinary frequency/urgency refractory to initial medications	Urology/urogynecology	1. Interventions (e.g., botulinum toxin)
Urinary stress or defecatory incontinence	Urology/urogynecology, PT	1. Interventions (e.g., pessary device, sling surgery) 2. Pelvic floor therapy
Vaginal dryness/dyspareunia due to low estrogen	Urology/urogynecology	1. Prescribe medications (e.g., topical or oral estrogen)
Erectile dysfunction refractory to initial medications	Urology	1. Interventions (e.g., intra-cavernosal injections, penile prosthesis)

Abbreviations: BP, blood pressure; CKD, chronic kidney disease; CV, cardiovascular; GI, gastrointestinal; HF, heart failure; MI, myocardial infarction; OH, orthostatic hypotension; PT, physical therapy; PVR, postvoid residual.

[a] Prior MI, HF, cardiomyopathy, arrhythmia, cardiac conduction disease, CKD.

[b] Lightheadedness or syncope in the supine or seated position, syncope with exertion or without warning.

[c] Prominent weight loss or diarrhea, evidence of GI bleeding.

[d] Urinary retention, urinary incontinence, urinary bleeding, recurrent UTI.

across studies but is estimated to be approximately 30%, with prevalence increasing with disease duration [10]. SH is present in approximately 30-50% of people with PD with neurogenic OH [1].

Symptoms of OH and PPH may include lightheadedness, dizziness, visual changes, and syncope; however, some people with PD may remain asymptomatic. SH may cause elevated nocturnal pressure natriuresis, contributing to nocturia and volume depletion, which in turn may worsen morning OH [9]. Recent evidence also links CV AD with cognitive dysfunction, fluctuating mood, more severe motor symptoms, and end-organ damage with resultant disability [11–14].

OH due to AD is neurogenic, characterized by a BP drop without reflex tachycardia [9]. However, people with PD may also experience unrelated cardiogenic causes of OH [14]. For example, OH in heart failure (HF) ranges from 8% among community-living individuals to 83% in elderly hospitalized patients [15]. CV AD may also be complicated by chronic kidney disease (CKD), which can exacerbate CV AD through several mechanisms. Activation of the renin-angiotensin-aldosterone system increases sympathetic nervous system activity, while vascular remodeling increases arterial stiffness, potentially worsening SH [16].

3.1. Neurology

The management of CV AD is currently symptomatic and not disease-modifying; CV physiology will likely not normalize completely despite best medical management [9]. Lifestyle interventions for OH include increased fluid and salt intake, intermittent water boluses, compression garments (especially abdominal binders), exercise, and counterpressure maneuvers such as leg crossing to prevent hypotension and increase venous return (important for patients with premonitory symptoms of syncope) [17]. If these measures are inadequate, pharmacologic therapy is initiated. Available medications include peripheral vasoconstrictors (e.g., midodrine), volume expanders (e.g., fludrocortisone), norepinephrine precursors (e.g., droxidopa), and cholinesterase inhibitors (e.g., pyridostigmine). In cases of severe or refractory symptomatic OH, multiple antihypotensive agents are often required. The management of SH begins with lifestyle interventions such as head-of-bed elevation, heated mattresses or blankets to promote peripheral vasodilation, and continuous positive airway pressure. These measures may have the added benefit of reducing nocturia and morning OH by reducing pressure natriuresis [9]. If these strategies prove inadequate, short-acting antihypertensive drugs (e.g., clonidine, losartan, nifedipine) may be administered at bedtime [9].

Neurologists may refer patients with CV AD for cardiopulmonary exercise testing to identify chronotropic incompetence, which may impact goals for exercise and physical rehabilitation (see *Physical therapy* below) [18].

3.2. Cardiology

Reasons for referral to a cardiologist can include (1) red flag features that are atypical for CV AD (e.g., orthostatic vital signs or autonomic function testing that support a non-neurogenic pattern in a patient with OH, or syncope occurring in the supine or sitting position), (2) significant CV comorbidity, and (3) refractory or high-risk clinical presentations.

For red flag features, cardiologists may perform an expanded physical examination to assess for cardiac disease, especially HF (e.g., pedal edema, raised jugular venous pressure), and begin with basic investigations such as an electrocardiogram, echocardiogram, or ambulatory heart rate monitoring.

If a patient either (1) has a history of myocardial infarction (MI), HF, cardiomyopathy, conduction system disease, arrhythmia, (2) is taking medications for these problems, or (3) has a pacemaker or implantable cardioverter-defibrillator, a cardiologist's opinion is recommended before adjusting or adding new medications to treat OH. For example, fludrocortisone may exacerbate congestive HF via fluid retention. Similarly, in patients with history of MI or stroke where strict BP control

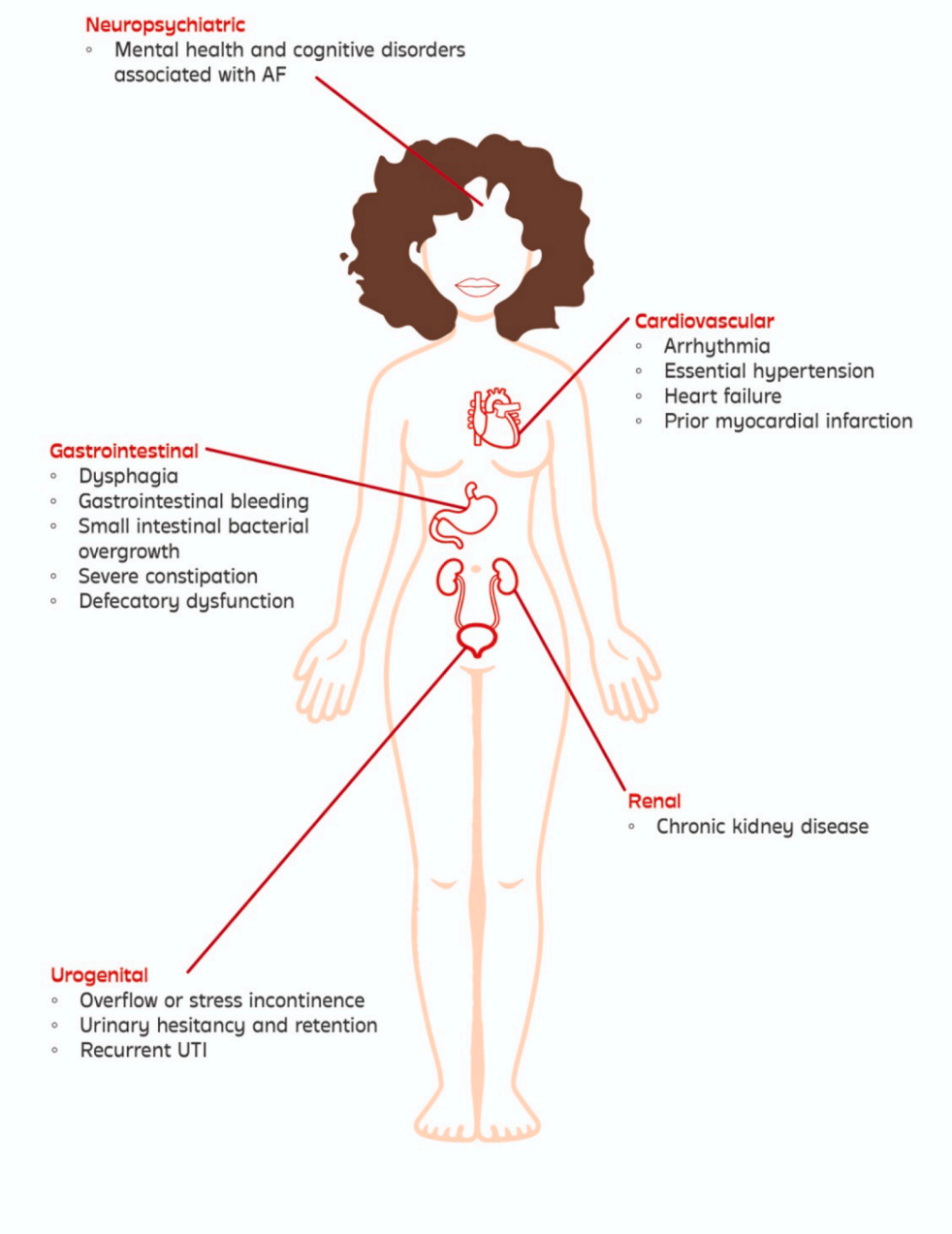

Fig. 2. Potential indications for specialist referral for AD by system.

is a priority, antihypotensive medications that may result in SH, such as fludrocortisone, midodrine, and droxidopa, should be prescribed in consultation with a cardiologist. Lifestyle recommendations, including liberal fluid and salt intake, may be inappropriate in patients with HF or severe hypertension; cardiology guidance is essential to individualize these interventions.

Cardiology input is invaluable when balancing AD-related BP lability and selecting appropriate nighttime antihypertensive therapy when first-line management is inadequate. Neurologists have limited formal training in the management of multiple vasoactive and cardioactive agents. When there is concern for undertreated SH in the setting of neurogenic OH, ambulatory BP monitoring can help identify 24-h BP ranges and may facilitate collaborative decision-making on BP goals. Data from the Systolic Blood Pressure Intervention Trial in adults >50 years with essential hypertension and OH indicates that a goal systolic BP < 120 mmHg is not associated with higher risk of OH-related adverse events compared to goal <140 mmHg [19]. However, for people with PD with significant neurogenic OH, consensus guidelines indicate that pharmacologic management for SH may be deferred unless systolic BP reaches 160-180 mmHg [17]. In complex cases with significant BP lability or CV comorbidity, collaboration with cardiology can improve safety and may decrease long-term CV risk in people with PD.

3.3. Nephrology

Reasons for referral to a nephrologist for patients with CV AD and CKD can include (1) to help define individualized BP treatment targets, (2) to optimize fluid and sodium balance while mitigating polypharmacy, and (3) to determine dialysis regimens and goals in patients with end-stage renal disease.

The 2025 American Heart Association guidelines recommend a seated systolic BP target of <130 mmHg to reduce all-cause mortality for adults with CKD not on dialysis [20]. While more intensive systolic BP targets have been encouraged in CKD and for patients with higher CVD

Table 2
Clinical pearls from each domain of autonomic dysfunction.

AD Domain	Key Takeaways
Cardiovascular	1. ? cardiogenic OH if reflex tachycardia on orthostatic vitals/autonomic testing. 2. ? cardiogenic causes if syncope occurs in seated or supine position. 3. Defer Rx for SH with severe nOH for systolic BP < 160-180 mmHg. 4. Liberalize systolic BP target in CKD to >120 mmHg with severe nOH. 5. Avoid volume expanders (e.g., fludrocortisone) in HF and CKD. 6. ? modifying target weights for people with PD on dialysis with nOH. 7. Screen for chronotropic incompetence in patients with nOH on PT. 8. Prescribe exercise for people with PD with chronotropic incompetence based on perceived exertion. 9. PTs can modify exercise for people with PD with symptomatic OH. 10. PTs can educate on counterpressure training & compression garments.
Gastrointestinal	1. Neurologists can consider barium swallow, esophagram, gastric emptying study prior to GI eval for relevant sx. 2. Red flags for causes other than AD: GI bleeding, unintentional weight loss, prominent diarrhea 3. ? empiric treatment for SIBO for prominent diarrhea/ unintentional weight loss. 4. Pelvic floor dyssynergia may contribute to impaired defecation & benefit from PT.
Urogenital	1. Rule out pelvic floor dysfunction and outlet obstruction in AD. 2. Urinary retention can contribute to UTIs, & ? With worsening confusion or hallucinations 3. Neurologists can initiate lifestyle recs and first-line meds for LUTs and sexual dysfunction. 4. Urology/urogyn can manage toxin injections, catheterization, urethral bulking agents, prostate reduction, sling surgery. 5. Behavioral therapy and PT can empirically improve LUTS.
Neuropsychiatric Care	1. AD may exacerbate &/or mimic neuropsychiatric symptoms (e.g., anxiety, cognitive impairment). 2. CBT can help patients cope & live better with AD.

Abbreviations: AD, autonomic dysfunction; CBT, cognitive behavioral therapy; CKD, chronic kidney disease; CV, cardiovascular; GI, gastrointestinal; LUTS, lower urinary tract symptoms; nOH, neurogenic orthostatic hypotension; OH, orthostatic hypotension; PD, Parkinson disease; Rx, prescription; SIBO, small intestinal bacterial overgrowth; ?, consider.

risk [21], supporting data to target SBP <120 mmHg is limited, and aggressive targets are often unsafe in patients with severe OH [22]. Accordingly, the guidelines suggest less intensive BP targets in individuals who are frail or have a high risk of falls, limited life expectancy, or symptomatic OH. Intensive BP lowering trials have also largely excluded patients with advanced CKD (estimated glomerular filtration rate <30 mL/min) and those with neurogenic OH, which makes weighing the risks and benefits of aggressive management difficult. In people with PD with severe symptomatic OH and/or advanced disease, the consequences of untreated OH may be more clinically significant than hypertension, warranting prioritization of OH management and liberalization of BP targets.

In patients with neurogenic OH, increased dietary salt intake is often recommended as a nonpharmacologic strategy to expand intravascular volume [17]. However, Kidney Disease: Improving Global Outcomes (KDIGO) guidelines advise a dietary sodium intake of <2 g/day in patients with hypertension and CKD [21], highlighting a clinical tension between autonomic and renal management priorities. Clinicians should weigh the short-term benefits of treating OH, such as reducing orthostatic intolerance, preventing falls, and improving functional status, against the long-term risks of sustained hypertension and increased extra-cellular fluid volume such as worsening renal function and CV disease. Furthermore, nephrologists may need to down-titrate or discontinue medications for CKD, such as angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, SGLT2 inhibitors, and diuretics, if they exacerbate OH. Certain medications for OH such as fludrocortisone should be avoided in advanced CKD given the increased risk of fluid retention and SH. There is limited information available on use of many other medications for OH in the setting of CKD and should be introduced and titrated cautiously in collaboration with a nephrologist [23–25].

In end-stage renal disease, appropriate dialysis to control extracellular volume is critical for reducing hypertension and CV risk. Conversely, excessive sodium and fluid removal exacerbates OH. Therefore, patients are often unable to meet their true target weights on dialysis. Therapies with increased dialysis time and slower fluid removal rates (e.g., peritoneal dialysis, frequent home hemodialysis) may improve hypertensive control while reducing hypotension. Though these therapies may be preferred in some people with PD, they may increase caregiver burden and should be considered on a case-by-case basis.

3.4. Physical therapy

Reasons for referral to physical therapists (PTs) can include (1) education on adaptations to exercise if limited by symptomatic OH, and (2) education on lifestyle interventions to counter OH. PTs usually spend more time longitudinally observing patients performing a wider range of activities than other practitioners. While patients are typically referred to physical therapy (PT) for management of motor dysfunction, PTs can screen for and monitor signs and symptoms of OH as part of routine practice.

Identification of CV AD at PT sessions may help triage patients to other providers for pharmacologic intervention and also has important implications for rehabilitative management of motor dysfunction. Some people with PD may exhibit a reduced heart rate (HR) due to CV AD, so that HR goals during aerobic exercise may need to be modified [18]. Therefore, cardiopulmonary exercise testing should be considered for all patients with OH in physical rehabilitation programs in order to objectively confirm an individual's peak HR. Individuals with chronotropic incompetence should have exercise prescribed based on their rate of perceived exertion [26]. Chronotropic incompetence does not negatively affect the benefits of endurance exercise on oxygen consumption or disease progression, at least in early PD [27]. BP increases during exercise may be blunted in individuals with OH, which can create a mismatch between oxygen delivery to working muscles and increased metabolic demands. PTs should be aware of this blunting, which can ultimately lead to premature muscle fatigue and exercise intolerance. Patients with OH may also need to modify activities to prevent fainting, such as cautiously transitioning from horizontal exercises (e.g., recumbent cycling, rowing machine) to upright activities. Consuming 500 mL of water immediately before exercise may prevent acute exercise-induced OH [28]. Careful monitoring for orthostatic intolerance at all sessions is prudent, as patients with AD can experience exercise-induced hypotension and exaggerated post-exercise hypotension [29].

PTs can educate on and reinforce lifestyle interventions that address CV AD. Counterpressure training for OH, which can increase systemic vascular resistance and reduce blood pooling in the lower body, should be incorporated into sessions [29]. Training on the use of abdominal binders and other compression garments, combined with increased fluid and salt intake, are first-line strategies to manage OH that can reduce fall risk [8].

4. Gastrointestinal autonomic dysfunction

GI AD is the most common non-motor feature of PD, reported in up to

80% of patients [30]. It can impact all functions of the GI system, including swallowing, gastric emptying, small bowel motility, and colonic motility, leading to a wide range of symptoms including drooling, dysphagia, nausea, vomiting, weight loss, bloating, abdominal distension, and constipation [30]. GI AD in PD not only affects quality of life, but can interfere with optimal medication absorption and response to treatment [31]. Of note, certain dopamine-blocking antiemetic/prokinetic agents that may worsen motor symptoms, such as metoclopramide, should be avoided in people with PD [6]. In severe cases, emergency admissions and hospitalizations can occur due to severe swallowing dysfunction, aspiration, bowel obstruction, and constipation [30]; these complications may be lessened with appropriate collaborative management.

4.1. Neurology

Evaluation and management of GI AD in people with PD can begin with the neurologist utilizing comprehensive symptom assessment and targeted testing. History should screen for dysphagia, symptoms of gastroparesis including nausea, vomiting, early satiety, erratic response to levodopa, and constipation [30,31]. Given the wide range of symptoms, validated disease-specific questionnaires including the Gastrointestinal Dysfunction Scale for PD can be considered to screen for GI AD [32]. Ancillary testing for select scenarios may include the following: (1) a modified barium swallow study for patients with suspected oropharyngeal dysphagia or prominent drooling (particularly to evaluate for silent aspiration), (2) an esophagram for patients with suspected esophageal dysphagia (e.g., patients reporting a sensation of food "getting stuck in the chest", or with dysphagia and a normal modified barium swallow), and (3) a gastric emptying study for patients with significant dyspepsia or an erratic response to levodopa [6]. Both lifestyle interventions (e.g., dietary modifications) and initial pharmacologic management may be discussed and/or prescribed by the neurologist [6].

4.2. Gastroenterology

Reasons for referral to a gastroenterologist can include (1) red flag features that may not be expected in PD alone, and (2) patients with prominent GI symptoms that are refractory to initial interventions [6]. Red flag features that are atypical for GI AD in PD include prominent esophageal dysphagia, diarrhea, and weight loss, as well as evidence of GI bleeding (e.g., hematochezia, melena, unexplained iron deficiency anemia).

Endoscopic evaluation including upper endoscopy and/or colonoscopy may be employed to evaluate red flag features and to further tailor treatment. Small bowel dysmotility can predispose to small intestinal bacterial overgrowth (SIBO), which manifests as abdominal bloating, distension, diarrhea, and unintentional weight loss [33]. The breath test available for SIBO diagnosis has low sensitivity, so gastroenterologists should guide antibiotic management decisions [33].

Gastroenterologists may be best equipped to consider the risks and benefits of specific prokinetic or prosecretory agents and rectal enemas for patients with severe constipation refractory to stool softeners and laxatives, as well as consideration for more invasive options [6].

4.3. Physical therapy

PT also plays a critical role in the management of constipation in PD by identifying and addressing pelvic floor dyssynergia, a common contributor to impaired defecation. Targeted interventions, including pelvic floor muscle training, biofeedback, and abdominal massage, can restore coordinated defecation and improve bowel evacuation [34].

5. Urogenital autonomic dysfunction

Lower urinary tract symptoms (LUTS) are common in people with PD, with reported prevalence ranging from 24 to 96%; underreporting may in part be due to associated stigma [35,36]. The most common symptoms include urinary urgency and frequency, for which patients may restrict water intake, leading to dehydration. Nighttime trips to the toilet due to nocturia increase risk of falls and associated injuries. Less common manifestations include an underactive bladder, presenting as a sense of incomplete bladder emptying and/or weak stream, and urinary retention, which can lead to an increased risk of urinary tract infection (UTI) and resultant hospitalization [37,38]. In people with PD, new onset or substantial worsening of hallucinations and confusion may be the only sign of a UTI [38].

Comorbid conditions unrelated to urogenital AD may contribute to LUTS in people with PD. Nocturia may result from pressure natriuresis due to SH and sleep fragmentation due to other conditions like obstructive sleep apnea and rapid eye movement sleep behavior disorder. Pelvic floor dysfunction, which can lead to pelvic floor prolapse, overactive bladder, and bowel obstruction, is more common in women, but is often only reported when highly bothersome [39]. Pelvic floor prolapse may contribute to stress incontinence, or leakage of urine with coughing, sneezing, and lifting heavy items. Constipation, prostatic enlargement, and vaginal prolapse can block flow of urine, contributing to urinary hesitancy and retention.

In addition to LUTS, sexual dysfunction is a common complication of urogenital AD, present in approximately 80% of men and women with PD [1]. This includes reduced libido, difficulty with erection and/or ejaculation in men, and dyspareunia and vaginal dryness in women.

5.1. Neurology

Prior to evaluation by the urologist or urogynecologist, a review of comorbid conditions that affect the bladder may reveal contributing factors, such as constipation, prostatic enlargement, uterovaginal prolapse, and pelvic floor weakness. In addition to a detailed history, specific clinic maneuvers may identify the type of dysfunction. For example, the cough test can provoke urine leakage, signaling stress incontinence.

A neurologist can guide discussions of lifestyle measures that may ameliorate urinary urgency, including limiting caffeine and alcohol intake and avoiding excess fluid intake right before bed [40]. Neurologists should also recognize and treat LUTS that are sequelae of other features of PD. For example, treating nocturnal SH may reduce nocturia by reducing pressure natriuresis, while adjusting dopaminergic medications may reduce LUTS resulting from on-off fluctuations. Beta-3 agonists may be considered first-line pharmacotherapy for urinary urgency and frequency. Mirabegron may increase BP and should be avoided in patients with uncontrolled hypertension, in which case vibegron could be used [40]. Anticholinergic medications are associated with elevated fall risk, dry mouth, confusion, and dementia risk, and therefore should be avoided when possible [41]. For erectile dysfunction, phosphodiesterase-5 inhibitors such as sildenafil remain first line agents [42]. Patients with OH should be advised that these medications may worsen hypotension and orthostatic intolerance, and hence the lowest effective dose should be prescribed. For premature ejaculation, daily or on-demand serotonin specific reuptake inhibitors may be used [42]. For vaginal dryness and dyspareunia, vaginal lubricants and vaginal estrogen cream may be considered.

5.2. Urology/urogynecology

Reasons for referral to a urologist or urogynecologist can include (1) severe symptoms of LUTS or sexual dysfunction that are refractory to initial interventions, (2) red flag symptoms of LUTS (e.g., urinary retention and incontinence, hematuria), and (3) recurrent UTI.

An initial investigation to better characterize the type of urinary

dysfunction may include urodynamic testing to assess urine storage and bladder emptying, including postvoid residual, pressure flow, and cystometric testing [40]. Once the patient's urinary dysfunction is characterized, the urologist/urogynecologist may then guide additional treatment. In men with prostate enlargement-associated urinary retention, tamsulosin may be used, although it can worsen OH and may require input from other practitioners prior to use. Prophylactic antibiotic treatment for UTI may be prescribed by the urologist, possibly in collaboration with an infectious disease specialist. In women, vaginal estrogen cream may reduce annual UTI risk by 50% [43].

Procedural interventions are available for severe LUTS due to urogenital AD, LUTS due to comorbidities, as well as sexual dysfunction. For urinary frequency and urgency, botulinum toxin injections administered in the bladder muscles can improve urine leakage and urgency [44]. Urologists can best identify patients with retention who may benefit from catheterization. Several interventions are available for women with stress incontinence, including a pessary device to support the bladder, urethral bulking injections for women with intrinsic sphincteric deficiency, and sling surgeries to support the bladder and urethra [44]. In addition, artificial urinary sphincters and balloon devices may be effective for stress incontinence in men [45]. Other evidence-based modalities to improve bladder control in patients with LUTS include sacral neuromodulation and percutaneous tibial nerve stimulation [44]. Intracavernosal injections and prosthetics may be considered for erectile dysfunction refractory to medications [42,46].

5.3. Physical therapy

Referral for PT should be considered for all people with PD with LUTS. Kegel exercises are a well-established treatment for sequelae of pelvic floor dysfunction including retention and stress incontinence. Evidence suggests that behavioral therapies including pelvic floor muscle training, bladder training, and urge control techniques may also benefit LUTS associated with urogenital AD in PD [47].

6. Neuropsychiatric care

Mental health practitioners including psychiatrists, psychologists, and social workers serve an important role in collaboration with neurologists to address scenarios in which autonomic and neuropsychiatric symptoms may potentiate or even mimic one another. There is a well-established link between the limbic and central autonomic systems, in which emotional processing influences autonomic regulation [48,49]. Growing evidence suggests an overlap between neuropsychiatric and autonomic dysregulation in PD via shared pathophysiologic mechanisms [13,50,51]. Dopaminergic medication-associated fluctuations in anxiety and cognition may be due to underrecognized changes in BP [52], while impaired cognition in the upright position may be an overlooked presentation of OH [53,54]. In these scenarios, the first-line approach for addressing neuropsychiatric symptoms may involve identifying and treating AD. If mood-stabilizing medications are introduced, psychiatrists should avoid agents that worsen autonomic symptoms, such as tricyclic antidepressants and certain neuroleptics [55]. While cholinesterase inhibitors may worsen nausea and diarrhea, rivastigmine in particular may have an antihypotensive effect and be preferred when cognitive symptoms are associated with OH [56]. Cognitive behavioral therapy and strategies to assist with coping can help people with PD to live better with a chronic illness that has unpredictable symptoms.

Another ongoing area of investigation that may have important management implications for AD in PD is interoception, described as "*the sensing, interpreting, and integrating of internal physiological signals through the trafficking of information between internal organ systems and the peripheral and central nervous systems, creating moment-by-moment neural representations, both conscious and non-conscious, of the internal organismal state*." [57,58] People with PD may have impaired interoception and therefore may not report symptoms of AD such as lightheadedness with OH or abdominal discomfort with GI dysmotility [58]. Similarly, objective swallowing dysfunction and aspiration can occur even when the patient is unaware of dysphagia subjectively [58]. Whether specific biofeedback or other therapeutic techniques can improve interoception and thereby prevent adverse sequelae of unrecognized AD requires further study [58,59]. Given the potential for impaired interoception in people with PD, clinicians should proactively incorporate objective measures for identifying AD instead of relying only on patients' report of symptoms, as described in other sections of this review.

7. Conclusion

As PD advances, AD can complicate management and lead to significant impairment in quality of life and function, safety issues, hospitalizations, and increased mortality. The patient and caregiver can have tremendous stress navigating all the moving parts, with simultaneous subspecialty consultations, parallel management plans, and potentially conflicting therapeutic recommendations. Neurologists often serve as the central point of contact and can help guide patients and caregivers through this complexity. This role can be strengthened by integrating cardiology, nephrology, gastroenterology, urology/urogynecology, physical therapy, and mental health practitioners to deliver coordinated, patient-centered care. This paper highlights the complexity of AD and provides a framework outlining potential indications for specialist referral and the complementary roles that these different disciplines may play in the evaluation and management of AD. Specialists from these various disciplines lent their viewpoints as coauthors to offer multidisciplinary perspectives and practical clinical insights. We hope this work emphasizes the value of a collaborative, team-based approach in which neurologists coordinate care while leveraging the expertise of other team members to optimize outcomes for people with PD and AD.

CRediT authorship contribution statement

Cameron Miller-Patterson: Writing – original draft, Writing – review & editing. **Delaram Safarpour:** Conceptualization, Writing – original draft, Writing – review & editing. **Katherine Longardner:** Writing – original draft, Writing – review & editing. **Guillaume Lamotte:** Writing – original draft, Writing – review & editing. **Abhishek Lenka:** Writing – original draft. **Abhimanyu Mahajan:** Writing – original draft. **Sarah Diamond:** Writing – original draft. **Ankita Gupta:** Writing – original draft. **Julie Hershberg:** Writing – original draft. **Sahib Khalsa:** Writing – review & editing. **Brian Olshansky:** Writing – review & editing. **Gregory M. Pontone:** Writing – review & editing. **Robert Rope:** Writing – review & editing. **Ronald F. Pfeiffer:** Writing – review & editing. **Indu Subramanian:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

The authors would like to thank Giulia Barone for designing Fig. 2. The authors did not receive financial support for the publication of this article.

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