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Case Study

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Activity-Dependent Upright Gait Failure With Home-Documented Delayed Hypotension A Patient-Authored Clinical Observation of Delayed Activity-Provoked Orthostatic Intolerance

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Abstract

Objective: *To describe delayed, activity-dependent upright gait dyscontrol with preserved recumbent cycling in association with symptom-linked systemic hypotension.*

Methods: *A 74-year-old patient-author compiled contemporaneous symptom records and home arm-cuff blood-pressure and heart-rate measurements during ordinary upright activity in August 2026. Cerebral blood flow was not measured.*

Results: *Walking initially remained normal but became unsafe after approximately 10-20 minutes, with head fog, visual blurring, disequilibrium, lateral instability, 1 fall, and 4 near-falls; recumbent cycling remained substantially better tolerated. On August 19, blood pressure/heart rate changed from 121/72 mm Hg and 78 after 3 minutes standing without symptoms to 107/53 and 85 after approximately 30 minutes of activity with head fog; standing pressure remained 103/56 after seated rest. On August 23, blood pressure/heart rate changed from 135/75 and 65 before a 50-minute brisk walk to a repeated standing value of 85/45 and 85 immediately afterward, with improvement during feet-elevated recumbent rest. On August 24, symptoms began approximately 20 minutes into a 50-minute walk. Postwalk measurements were 143/65 and 64 standing, 180/78 and 71 sitting, and 123/61 and 77 on re-standing.*

Interpretation: *These observations support delayed activity-provoked orthostatic intolerance with home-documented symptomatic hypotension and postexercise orthostatic susceptibility. They are compatible with, but do not formally establish, delayed orthostatic hypotension. Possible cerebral underperfusion remains an inference. Prolonged symptom-linked beat-to-beat blood-pressure and rhythm monitoring would be required for more definitive physiological characterization.*

Keywords: Delayed Orthostatic Hypotension, Gait, Falls, Dysautonomia, Cerebral Perfusion, Postexercise Hypotension

1. Introduction

Delayed orthostatic hypotension is defined by a sustained qualifying blood-pressure fall occurring beyond 3 minutes of standing or head-up tilt and may therefore be missed by brief office measurements [1-3]. Presentations can include cognitive slowing, visual disturbance, unexplained falls, and gait impairment [2,4,5]. This note reports a patient-generated longitudinal observation in which sustained walking produced delayed head and gait symptoms with documented systemic hypotension, whereas recumbent cycling remained substantially preserved. The account deliberately distinguishes measured systemic hemodynamics from the unmeasured possibility of cerebral underperfusion.

2. Case Report

A 74-year-old man had a history of chikungunya infection in 2008, orthostatic symptoms, high-burden ventricular ectopy, and a

2021 left ventricular outflow tract ablation complicated by cardiac tamponade, emergency sternotomy, cardiopulmonary bypass, and ventricular free-wall repair. Subsequent manifestations included marked supine or episodic hypertension with orthostatic falls, gastrointestinal dysmotility, pancreatic exocrine insufficiency, eosinophilic esophagitis, and bladder-emptying difficulty. The patient-author used the working formulation of multifactorial secondary autonomic dysfunction, this had not been independently established as a discrete specialist diagnosis. Reported medications included tamsulosin, losartan, spironolactone, and intermittent furosemide. During 2024-2025, autonomic surges ceased, thermoregulation improved, and heart-rate responsiveness during exercise returned. In August 2026, however, unsupported walking became unreliable after initially normal movement. Symptoms included head fog or pressure, visual blurring, disequilibrium, lateral veering, and loss of automatic step rhythm without loss of consciousness. One fall occurred after approximately 10 minutes of standing and walking on Pitt Street, followed by 4 near-falls. A shopping trolley improved stability. Recumbent cycling remained substantially better tolerated.

Home observations captured 3 complementary events (Table 1). On August 19, blood pressure/heart rate was 121/72 mm Hg and 78 after 3 minutes standing without immediate symptoms, then 107/53 and 85 after approximately 30 minutes of walking and activity with head fog and reduced steadiness. After 30 minutes seated rest, standing pressure remained 103/56 and heart rate 78, with incomplete symptom recovery. On August 23, prewalk blood pressure/heart rate was 135/75 and 65. Immediately after a 50-minute brisk walk, while still standing, an Omron monitor recorded 85/45 and 85 during head fog, impaired balance, and general unwellness, a repeat within 1-2 minutes was unchanged. With feet-elevated recumbent rest, systolic pressure returned to 120-130 mm Hg over approximately 10-15 minutes and heart rate settled near 75. On August 24, prewalk blood pressure/heart rate was 165/85 and 68. Symptoms began approximately 20 minutes into another 50-minute walk and worsened before return home. Postwalk blood pressure/heart rate was 143/65 and 64 standing, 180/78 and 71 after approximately 2 minutes sitting, and 123/61 and 77 approximately 15 seconds after re-standing. The 57/17 mm Hg postural fall was accompanied by only a 6-beat/min heart-rate increase. Although the calculated delta-heart rate/delta-systolic blood-pressure ratio was 0.11, this nonstandard postexercise maneuver cannot establish neurogenic orthostatic hypotension [6-10].

3. Discussion

The phenotype is best described before assigning mechanism: delayed, threshold-dependent upright gait dyscontrol with falls, accompanied by home-documented symptomatic hypotension. The August 19 series is compatible with delayed orthostatic hypotension but lacks a standardized supine baseline. The August 23 event documents severe postwalk standing hypotension, whereas the August 24 sequence demonstrates marked postexercise orthostatic susceptibility. Intermittent cuff readings cannot identify the precise nadir or separate upright pooling from exercise-related vasodilation [2,6,7,9]. A two-stage model may account for the observed delay. The patient reported a usual preactivity range of 165-195/85-95 mm Hg and an immediate systolic fall of approximately 30-50 mm Hg on standing. The early fall may reduce available pressure reserve while residual vasoconstriction, cardiac output, and the leg muscle pump initially preserve walking. With continued upright activity, gravitational pooling, exercise vasodilation, medication or volume effects, and the neural demands of balance may exceed compensatory reserve. When walking stops, loss of muscle-pump support with persistent vasodilation may further lower pressure, brief sitting can restore central volume, and re-standing can expose a large postexercise fall [2,6,7,9].

Preserved recumbent cycling supports a postural hemodynamic contribution because recumbency reduces the hydrostatic column and gravitational pooling, maintains preload, and removes most balance demands. It does not exclude arrhythmia, vestibular disease, sensory ataxia, posterior-circulation disease, or another neurologic cause. Cerebral blood flow was not measured, head, visual, balance, and gait symptoms therefore cannot be presented as direct evidence of cerebral underperfusion [4,5]. The fall and near-fall cluster warrants prompt injury-focused assessment [8]. The highest-yield characterization would extend monitoring beyond the usual symptom threshold and combine continuous beat-to-beat blood pressure, ECG, symptom marking, and a supervised walking-equivalent protocol through walking cessation, sitting, re-standing, and recovery. Neurologic, vestibular, and cerebrovascular evaluation is required if focal signs occur or systemic hemodynamics do not adequately explain the gait disturbance.

Date/event	Activity and posture	Blood pressure and heart rate	Symptoms and interpretation
August 19	3-minute stand, then approximately 30 minutes walking/activity, repeat stand after seated rest	121/72, HR 78, then 107/53, HR 85, after rest 103/56, HR 78	No initial symptoms, later head fog and reduced steadiness, incomplete recovery
August 23	Before and immediately after 50-minute brisk walk, postwalk measurement while standing	135/75, HR 65 before, repeated 85/45, HR 85 after	Head fog and impaired balance, BP recovered during feet-elevated recumbence
August 24	Before 50-minute walk, symptoms at approximately 20 minutes, standing, sitting, and re-standing after walk	165/85, HR 68 before, 143/65, HR 64 standing, 180/78, HR 71 sitting, 123/61, HR 77 re-standing	Progressive head and balance symptoms, 57/17 mm Hg sit-to-stand fall with HR +6
Task comparison	Sustained unsupported walking vs recumbent cycling	No continuous BP recorded during cycling	Walking became unsafe, recumbent cycling remained substantially better tolerated

Table 1: Symptom-Linked Home Observations

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Competing Interests
The author reports no disclosures relevant to this manuscript.

Author Contribution
Bruce H. Knox had the major role in acquisition of the patient-generated data, study concept and design, analysis and interpretation of data, drafting and revision of the manuscript for intellectual content, and final approval of the manuscript.

Consent and Ethics
The patient and author are the same person and has elected to disclose the clinical information contained in this manuscript for scholarly dissemination. This is a patient-authored, noninterventional case narrative, institutional ethics approval was not sought.

Data Availability
All data supporting the principal observations are presented in the article and Table 1. Additional longitudinal patient-generated records may be available from the corresponding author on reasonable request, subject to protection of identifiable health information.

AI-Assisted Editorial Disclosure
OpenAI ChatGPT (GPT-5.6, accessed August-September 2026) was used for language editing, condensation, and formatting support. The tool did not generate the clinical observations. The author reviewed the source records, citations, analysis, and final text and accepts full responsibility for the manuscript.

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