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Research Article

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Walking Fails, Cycling Persists
Delayed Upright Gait Dyscontrol, Recurrent Falls, and Suspected Cerebral Under-Perfusion in Multifactorial Secondary Autonomic Dysfunction

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Abstract

Background: *Delayed orthostatic intolerance may emerge only after sustained standing or walking and can be missed by brief office measurements. Delayed orthostatic hypotension (dOH) is a haemodynamic subtype in which a qualifying blood-pressure fall occurs beyond three minutes upright; delayed orthostatic intolerance is the broader symptom phenotype. In this paper, possible cerebral under-perfusion is inferred from systemic blood-pressure readings and contemporaneous head, visual, balance, and gait symptoms; cerebral blood flow was not measured [1-5].*

Case: *A 74-year-old man with a history of chikungunya infection presents with multifactorial secondary autonomic dysfunction. He has consulted with both cardiology and gastroenterology specialists. His medical history includes severe arrhythmia and an ablation procedure performed in 2021, which was complicated by cardiac tamponade, requiring emergency sternotomy and cardiopulmonary bypass. Following these events, he has experienced significant supine hypertension accompanied by major orthostatic falls. His reported usual pre-activity blood pressure was 165-195/85-95 mmHg, with an immediate systolic reduction of approximately 30-50 mmHg on standing. In August 2026, sustained upright activity produced delayed head disequilibrium and unreliable step control while recumbent cycling remained substantially better tolerated. The pattern escalated to a fall after approximately 10 minutes of standing and walking on Pitt Street and four subsequent near-falls. On 19 August, BP/HR changed from 121/72 mmHg and 78 after three minutes standing without immediate symptoms to 107/53 and 85 after approximately 30 minutes of activity with head fog and reduced steadiness; standing pressure remained 103/56 after seated rest. On 23 August, BP/HR fell from 135/75 and 65 before a 50-minute brisk walk to a repeated standing value of 85/45 and 85 immediately afterwards, with head fog, impaired balance, and improvement during feet-elevated recumbent rest. On 24 August, symptoms began approximately 20 minutes into another 50-minute brisk walk. After returning home, BP/HR was 143/65 and 64 standing, 180/78 and 71 sitting, and 123/61 and 77 on re-standing—a 57/17 mmHg postural fall with only a 6-beat heart-rate increase.*

Interpretation: *The record suggests two orthostatic time scales: an immediate systolic fall from a markedly hypertensive starting range, followed by delayed head and gait symptoms as upright and exercise-related stresses accumulate. The 19 August series links delayed symptoms with a fall in systemic pressure; the 23 August event documents severe post-walk standing hypotension with recumbent recovery; and the 24 August event demonstrates marked post-exercise orthostatic susceptibility. Together, these observations support the descriptive formulation of delayed activity-provoked orthostatic intolerance with home-documented delayed symptomatic hypotension, compatible with dOH but not formally diagnostic of it. Preserved recumbent cycling supports a postural haemodynamic contribution but does not exclude arrhythmia, vestibular disease, or neurological and cerebrovascular causes [1-19]. Greater predictability of symptom onset may reflect improved warning or adaptation, but is not by itself evidence of recovered autonomic haemodynamic function.*

Conclusion: *A fall, four near-falls, and an apparent shortening of the upright threshold from approximately 20 minutes to approximately 10 minutes represent a clinically important escalation. Prompt fall-focused assessment and prolonged symptom-linked monitoring of beat-to-beat blood pressure, heart rate, and rhythm are warranted; cerebral-perfusion, neurological, or vestibular assessment should be added when clinically indicated.*

Music Introduction: The link below opens The Lament of the Upright Traveler, a lament that gives musical form to the substance of this paper: the lived experience of remaining upright, moving forward, and repeatedly encountering the limits imposed by delayed orthostatic failure. Readers are invited to click the link and listen to the music before continuing into the clinical narrative.
https://heyzine.com/flip-book/768a2e478c.html

Key Findings.

- The case demonstrates delayed, threshold-dependent upright gait dyscontrol in the setting of multifactorial secondary autonomic dysfunction and marked blood-pressure lability.
- Walking appears to begin after an immediate 30-50 mmHg systolic orthostatic pressure fall from a markedly hypertensive usual pre-activity range of 165-195/85-95 mmHg.
- Home observations on 19, 23, and 24 August 2026 provide convergent symptom-linked evidence of delayed hypotensive deterioration, severe post-walk standing hypotension, and post-exercise orthostatic susceptibility.
- The 23 August event provides the clearest paired capture: BP/HR changed from 135/75 and 65 before walking to a repeated standing value of 85/45 and 85 immediately after a 50-minute brisk walk, followed by recumbent recovery.
- The 24 August event shows delayed walking symptoms followed by a 57/17 mmHg sit-to-stand fall with limited heart-rate compensation.
- The most defensible formulation is delayed upright-activity intolerance with home-documented delayed symptomatic hypotension, compatible with dOH, together with superimposed post-exercise orthostatic susceptibility.
- Possible cerebral under-perfusion remains an inference from systemic BP data and patient-described head, visual, balance, and gait symptoms; cerebral blood flow was not directly measured.
- The fall and near-fall cluster marks a clinically important transition requiring prompt assessment, preventive mobility measures, and prolonged symptom-linked haemodynamic and rhythm monitoring.

Keywords: Delayed Orthostatic Intolerance, Delayed Orthostatic Hypotension, Suspected Cerebral Under-Perfusion, Gait Dyscontrol, Falls, Dysautonomia, Supine Hypertension, Recumbent Cycling, Case Study

Central formulation. The sequence begins from a reported pre-activity range of 165-195/85-95 mmHg, includes an immediate 30-50 mmHg systolic fall on standing, and culminates in delayed gait failure during sustained upright activity. The 19, 23, and 24 August observations provide convergent home evidence of delayed symptomatic pressure decline, severe post-walk standing hypotension, and marked post-exercise orthostatic susceptibility. Cerebral blood flow was not measured, any proposed under-perfusion remains an inference from systemic BP data and contemporaneous head, visual, balance, and gait symptoms.

1. Introduction

Orthostatic disorders are often conceptualised as immediate events occurring within seconds of standing. That model is incomplete. Blood pressure may initially remain adequate and deteriorate only as upright stress continues, symptoms suggesting reduced cerebral reserve may also emerge late, but this paper does not measure cerebral blood flow. Delayed orthostatic hypotension (dOH) is defined by a sustained fall meeting orthostatic hypotension criteria beyond three minutes of standing or head-up tilt. Delayed orthostatic intolerance (dOI) is a broader descriptive phenotype: disabling symptoms emerge late during standing or upright activity, whether or not a qualifying pressure fall has yet been recorded [1-5]. The distinction is essential in this case. The principal disability is not simply light-headedness or syncope, but a delayed loss of reliable step control accompanied by head pressure or disequilibrium, lateral veering, and the need to consciously manage each step. Recumbent cycling remains substantially more tolerable. This task dissociation raises a focused question: does accumulated upright haemodynamic load temporarily reduce the functional reserve required for safe walking while supported horizontal exercise remains possible?

The paper interprets possible cerebral under-perfusion as an inference from systemic blood-pressure readings and contemporaneous head, visual, balance, and gait symptoms. It does not claim that cerebral blood flow was directly measured. The August 2026 Pitt Street fall and four subsequent near-falls materially change the clinical significance. The phenomenon is

no longer an isolated subjective disturbance: symptom-linked home readings support a delayed hypotensive component, while the recurrent motor disturbance presents an immediate threat to physical safety and requires formal characterisation within a broad differential diagnosis.

2. Case Presentation

2.1. Longitudinal Clinical Context

The patient-author is a 74-year-old man from Auckland, New Zealand. His autonomic and cardiovascular history began after chikungunya infection in November 2008. From 2009 to 2015, orthostatic dizziness, visual dimming, heat and exercise intolerance and orthostatic hypotension were prominent. Later, high-burden ventricular ectopy developed, at times reported at approximately 15,000 premature ventricular contractions per 24 hours. On 15 October 2021, an attempted left ventricular outflow tract premature ventricular contraction ablation was complicated by cardiac perforation and tamponade, requiring emergency sternotomy, cardiopulmonary bypass and ventricular free-wall repair. During 2022-2023, the phenotype included severe supine or episodic hypertension alternating with major orthostatic falls, initially restricted heart-rate compensation and autonomic surges. Gastrointestinal dysmotility, pancreatic exocrine insufficiency, eosinophilic oesophagitis, bladder emptying difficulty and earlier thermoregulatory disturbance contributed to a multisystem pattern.

Several domains improved during 2024-2025: severe autonomic storms ceased, thermoregulation became more stable and heart-rate responsiveness during walking and exercise returned. In 2026, however, upright vascular control remained unreliable. The patient could complete stairs, household work, driving, cycling and short shopping trips, yet after sustained walking he developed head pressure, visual or spatial disturbance, fatigue, veering and loss of automatic step rhythm. Recumbent rest for 30-60 minutes often restored function while he remained awake, suggesting haemodynamic relief rather than sleep alone. The working label of multifactorial secondary autonomic dysfunction is a patient-authored explanatory formulation, not a universally established diagnostic entity and not independent proof of a specific lesion. It is used here to describe a distributed acquired syndrome with cardiovascular, gastrointestinal, urinary and thermoregulatory involvement following several possible secondary insults. The case does not establish primary autonomic failure, neurodegeneration or classical afferent baroreflex failure.

2.2. Haemodynamic Starting Point and Documented Blood-Pressure Lability

The starting point is clinically important. The patient reports a usual pre-activity blood-pressure range of 165-195/85-95 mmHg. Although customary for him, this is markedly hypertensive rather than physiologically normal. On standing, systolic pressure ordinarily falls immediately by approximately 30-50 mmHg. Walking therefore begins only after a substantial orthostatic pressure cost has been incurred, even when initial movement remains normal. Historical home and clinical records demonstrate genuine orthostatic blood-pressure instability, but the usual range and the recent delayed walking events were not captured as one continuous standardised series. Selected measurements are summarised below. Timing and posture labels reproduce the patient record, they should not be treated as a standardised autonomic laboratory protocol.

Approximate period	Starting measurement	Upright measurement	Interpretive limit
Usual 2026 pre-activity pattern	Before walking or cycling: 165-195/85-95	Standing: immediate systolic fall approximately 30-50 mmHg	Reported recurrent starting pattern, not a same-session paired baseline for the 45-min walk.
2022	Supine 215/105, HR 55	30 s 145/85, 1 min 150/80, 3 min 160/85, HR approximately 58	Large early pressure fall with little heart-rate increase, not the later walking episode.
July 2026, approximately 3 pm	Supine 131/59, HR 51	Standing 87/51, HR 73	Clear symptomatic-risk range, exact stand duration not independently verified.
July 2026, approximately 10 pm	Seated 189/94, HR 59	Standing 158/89, HR 67	Large relative systolic fall from a hypertensive starting pressure.
August 2026, after 45-min walk	Pre-walk pressure not recorded	Post-walk BP reported below 105/75, HR not recorded	Supports concern but cannot distinguish dOH from post-exercise hypotension or identify pressure at the fall threshold.
19 August 2026, 10:03 to 10:30 am	Standing 3 min: 121/72, HR 78, no immediate drop	After approximately 30 min walking/activity: 107/53, HR 85, head fog and "out of kilter"	Delayed symptomatic decline, including a 19 mmHg diastolic fall from the 3-min standing value.
19 August 2026, 11:00 am	After 30 min seated rest	Standing 103/56, HR 78, symptoms persisted	Incomplete haemodynamic and symptomatic recovery.
23 August 2026, 9:15 am	9:15 am pre-walk BP/HR 135/75 and 65, post-walk value obtained after a 50-min brisk dog walk, while still standing.	Omron BP 85/45, HR 85, head fog, impaired balance and feeling unwell	Repeat value unchanged, recumbent feet-elevated recovery to SBP 120-130 over 10-15 min.

24 August 2026, after 5 pm walk	Symptoms began after approximately 20 min of a 50-min brisk dog walk, post-walk standing 143/65, HR 64	After approximately 2 min sitting: 180/78, HR 71, re-standing: 123/61, HR 77	Paired post-exercise fall 57/17 with HR +6, symptoms persisted approximately 15 min. No pre-walk baseline or exact stand timing.
24 August 2026, 7:00 pm	Sitting 171/72, HR 65 after rest, a meal and household activity	Standing 152/63, HR 71	Residual 19/9 postural fall with HR +6, below conventional 20/10 threshold by 1 mmHg in each component.

Table 1: Selected Blood-Pressure Observations Showing Immediate Orthostatic Change, Delayed Activity-Related Symptoms, and Post-Exercise Orthostatic Susceptibility

The sequence is therefore not a simple transition from normal pressure to hypotension. It begins from a markedly hypertensive reported range, includes an immediate 30-50 mmHg systolic fall on standing, and may remain functionally compensated for approximately 10-20 minutes before head and gait symptoms emerge. The 19 August series shows why a brief standing measurement can appear acceptable while later pressure declines with symptoms. The 23 August event captures severe post-walk standing hypotension at 85/45 mmHg, the 24 August event captures delayed symptoms followed by a 57/17 mmHg post-exercise sit-to-stand fall. The variable recorded nadirs make posture, symptom timing, relative change, absolute pressure, and recovery more informative than any single brachial value [1,2,6,8,11,12].

2.3. Medication and Volume Context

The reported medication context included tamsulosin, losartan, spironolactone, and intermittent furosemide. Alpha-1 blockade may reduce peripheral vasoconstrictor reserve, while antihypertensive and diuretic effects may lower vascular resistance or circulating volume. These are plausible amplifiers, not demonstrated causes. Because severe supine hypertension coexists with orthostatic falls, medication withdrawal or dose changes cannot be inferred safely from this narrative. Clinician-led review should consider indication, dose, timing, renal function, electrolytes, fluid balance, and the timing of symptoms [11,14].

3. Evolution of the Walking Disturbance

3.1. Initial August 2026 Threshold Episode

On 17 August 2026, the patient completed breakfast and coffee, climbed stairs, drove to a landscaping store and walked in the store without difficulty. At home he then worked on a trailer, requiring standing, bending, position changes and manual attention to the trailer brakes. For approximately 20 minutes, function remained stable. Thereafter, over nearly two hours, head disequilibrium and impaired step control developed and fluctuated, forcing repeated rest breaks. Each step required conscious management because the head and legs no longer felt automatically coordinated. After rest and coffee, the disturbance eased. He then cycled without significant recurrence. Later, mild instability reappeared during unsupported supermarket walking, but holding a trolley made continued movement more manageable. No blood pressure, heart rate, rhythm, glucose, neurological examination or cerebral-flow measurement was captured during the symptomatic period.

3.2. Pitt Street Fall and Near-Fall Escalation

Later in August 2026, the patient fell after approximately 10 minutes of combined standing and walking on Pitt Street. He subsequently recorded four near-falls while walking. He described a recurring sensation of inadequate blood supply or pressure in the head whenever upright movement continued beyond approximately 10 minutes. This wording records the lived sensation only, it is interpreted alongside the systemic blood-pressure data but is not a direct measure of cerebral blood flow. The previously observed threshold appeared to shorten from approximately 20 minutes to approximately 10 minutes, although task, temperature, meal timing, sleep, fluid status, medication timing, and starting pressure were not controlled. After a separate 45-minute walk, he recorded a brachial pressure below 105/75 mmHg. Relative to his usual pre-activity range, this suggests a large fall, but it is not a paired calculation because no contemporaneous pre-walk BP or heart rate was recorded. The measurement cannot identify the nadir or determine whether the fall developed during walking, after walking stopped, or through both processes. The clinical significance lies in the convergence of findings: a potentially large relative fall from a hypertensive starting range, reproducible delayed head and gait symptoms, one actual fall, and four near-falls. Falls in older adults with cardiovascular disease are commonly multifactorial, orthostatic hypotension, arrhythmia, medication effects, neurological disease, gait and balance impairment, and environmental hazards may interact [7,12,16,17].

3.3. Symptom-Linked Home Monitoring on 19 August 2026

A structured morning of ordinary home activity was recorded as a "living laboratory". At 10:00 am, after gardening and while standing, pressure was 126/68 mmHg with heart rate 80. At 10:03 am, after an extended three-minute stand, it was 121/72 with heart rate 78 and there was no immediate drop or clear symptom. By 10:30 am, after approximately 30 minutes of walking and activity, pressure had declined to 107/53 with heart rate 85, coinciding with head fog, feeling "out of kilter" and reduced steadiness. At 11:00 am, after 30 minutes seated rest, the standing pressure remained 103/56 with heart rate 78 and symptoms had not fully cleared. The same record showed variability rather than a simple monotonic fall: later gardening was associated with 143/73 and heart rate 88, followed by 130/62 during continued activity. After lunch, sitting pressure was 160/80, on standing it fell to 117/59 while heart rate rose only from 70 to 75. Taken together, the 19 August data show initial short-duration tolerance, later pressure decline aligned with symptoms, incomplete recovery and intermittent rebound. They

are stronger evidence than an isolated post-walk reading, although they remain patient-conducted home observations rather than a controlled autonomic test.

3.4. Symptom-Linked Home Monitoring on 23 August 2026

At 9:15 am on 23 August 2026, before walking, BP/HR was 135/75 mmHg and 65. Immediately after a 50-minute brisk walk with the dog, while still standing, he recorded 85/45 mmHg and 85 using an Omron monitor with a small cuff on the right arm, the elbow bent, and the arm held at heart level. Contemporaneous symptoms included head fog, impaired balance, and a general feeling of being markedly unwell. A repeat measurement within one to two minutes was unchanged, reducing the likelihood that the finding reflected a single isolated reading. He then rested with his feet elevated. Over approximately 10-15 minutes, systolic pressure returned to the 120-130 mmHg range, rising by approximately 10 mmHg every five minutes, while heart rate settled at approximately 75 bpm. This temporal sequence links severe post-walk standing hypotension with symptomatic improvement during recumbence.

3.5. Delayed Walking Symptoms and Post-Exercise Orthostatic Fall on 24 August 2026

At 5:00 pm on 24 August 2026, pre-walk BP/HR was 165/85 mmHg and 68. The patient then began a 50-minute brisk walk with the dog. After approximately 20 minutes, his head became light and blurry and felt acutely 'out of kilter'. He continued cautiously for the remaining 30 minutes because of concern about falling, and the symptoms worsened by the time he reached home. While still standing, BP/HR was 143/65 mmHg and 64. Approximately two minutes after sitting, it was 180/78 and 71. On re-standing, it fell to 123/61 and 77: a paired postural reduction of 57 mmHg systolic and 17 mmHg diastolic with only a 6-beat heart-rate increase. Estimated arm-level mean arterial pressure fell from approximately 112 to 82 mmHg, this derived value is not a measurement of cerebral perfusion. Head disequilibrium continued for approximately 15 minutes while he sat and relaxed. He subsequently ate a meal, stacked the dishwasher and resumed ordinary household activity. At 7:00 pm, BP was 171/72 with HR 65 sitting and 152/63 with HR 71 standing, a smaller 19/9 mmHg postural difference with the same 6-beat heart-rate rise. The later pair sits just below the conventional 20/10 mmHg orthostatic criterion and indicates substantial recovery from the immediate post-walk lability, although a residual posture-related difference remained.

The immediate post-walk re-standing fall meets conventional orthostatic BP-change thresholds if sustained. However, this was a post-exercise home manoeuvre rather than a formal active-stand test: it began from sitting after prolonged walking, and the re-standing measurement was obtained approximately 15 seconds after standing. The calculated delta-HR/delta-SBP ratio was approximately 0.11 beats/min/mmHg. Although a ratio below 0.5 may support a neurogenic mechanism during controlled testing, it should not be used here as a stand-alone classification. The finding is best treated as a strong signal for formal autonomic characterization [1,2,11,19].

Phase / time point	Observed function or measurement	Clinical meaning
Starting point and standing	Before walking or cycling BP is usually 165-195/85-95, standing immediately lowers systolic pressure by approximately 30-50.	Walking starts after a substantial early orthostatic reduction, while recumbent cycling avoids most of that vertical transition.
Short upright exposure	Stairs, driving, brief shopping and initial walking may be normal.	Baseline strength and movement capacity remain available.
After approximately 20 min	Trailer work provoked head disequilibrium and impaired step control.	Accumulated upright and task demand crossed a functional threshold.
Later August 2026, approximately 10 min	Fall on Pitt Street after standing and walking.	Threshold appeared shorter, the phenotype became injury-producing.
Subsequent walking	Four near-falls and continuing head-perfusion sensation beyond approximately 10 min.	Recurrent unsafe gait, not a single anomalous event.
After 45-min walk	Brachial BP reported below 105/75, HR and pre-walk BP unavailable.	Objective concern, but timing cannot separate orthostatic from post-exercise effects.
19 August living laboratory	121/72 at 3 min without symptoms, 107/53 after 30 min with head symptoms, 103/56 after rest.	Direct home evidence that pressure declines as delayed symptoms emerge and recovery may be incomplete.
23 August, 50-min brisk walk	Standing BP 85/45 and HR 85 at 9:15 am with head fog, impaired balance and feeling unwell, recumbent recovery followed.	Independent severe symptom-linked hypotensive event after prolonged upright walking.
24 August, 50-min brisk walk	Symptoms began at approximately 20 min, post-walk standing 143/65, then 180/78 sitting and 123/61 re-standing, HR 64, 71 and 77.	Delayed symptomatic event followed by a paired 57/17 post-exercise orthostatic fall with limited HR compensation.

Recumbent cycling	Substantially better tolerated.	Supported horizontal exercise imposes different gravitational and balance demands.

Table 2: Functional Sequence Linking Upright Exposure, Observed Walking Disturbance, and Clinical Meaning

4. Clinical Interpretation

4.1. Phenotype before Mechanism

The most defensible descriptive diagnosis is delayed, threshold-dependent upright gait dyscontrol with falls. It is delayed because initial function may be normal, threshold-dependent because it appears only after accumulated upright activity, posture-related because standing and walking are more provocative than recumbent cycling, and a gait disorder because step placement, balance, and directional control become unreliable before consciousness is lost. Within the established autonomic history, the leading physiological formulation is delayed upright-activity intolerance with home-documented delayed symptomatic hypotension. The measurements link systemic hypotension with head fog, an 'out of kilter' sensation, visual disturbance, and impaired balance. They provide a clinically meaningful basis for considering cerebral under-perfusion while preserving the distinction between inference from systemic BP data and direct specialist measurement of cerebral blood flow [2,6,8-10,12].

Formal dOH classification requires a sustained fall of at least 20 mmHg systolic or 10 mmHg diastolic beyond three minutes upright, with supine hypertension, a systolic threshold of at least 30 mmHg may be more appropriate. On 19 August, BP was 121/72 after three minutes standing without immediate symptoms and 107/53 after approximately 30 minutes of activity when head symptoms appeared. Because this was not measured from a standardised supine baseline, it is compatible with, but not formally diagnostic of, dOH. On 23 August, severe standing hypotension was recorded while symptomatic after 50 minutes of walking. On 24 August, delayed symptoms began during walking and were followed by a 57/17 mmHg post-exercise sit-to-stand fall with limited heart-rate compensation. The most precise formulation is therefore delayed upright-activity intolerance with home-documented delayed symptomatic hypotension, compatible with dOH, and superimposed post-exercise orthostatic susceptibility [1-5,11,18,19].

4.2. A Two-Stage Sequence: Immediate Pressure Cost, Delayed Walking Failure

The reported starting pressure changes the interpretation. Activity commonly begins from 165-195/85-95 mmHg, followed by an immediate systolic reduction of approximately 30-50 mmHg on standing. The absence of immediate collapse does not make that transition benign: the early fall may consume part of the available haemodynamic reserve while compensatory mechanisms preserve consciousness and short-duration walking. The distinctive failure occurs later, when compensation no longer sustains stable head function and automatic gait. Standing redistributes blood into the legs and splanchnic circulation and reduces central blood volume. Normal compensation requires sustained vasoconstriction, adequate circulating volume, appropriate heart-rate and contractility responses and effective skeletal-muscle and respiratory pumps. A system with limited reserve may initially compensate after the early pressure fall and then become insufficient as pooling, active-muscle vasodilation, heat, meals, fatigue, medication effects and time upright accumulate [2,5,11,14].

The 24 August sequence also shows why pressure during walking, immediately after walking, and after re-standing may differ. Rhythmic leg contractions can support venous return during movement. When walking stops, this muscle-pump assistance falls away while exercise-related vasodilation may persist. Sitting can restore central volume and raise pressure, re-standing before the vasodilatory state has resolved can then expose a larger orthostatic fall. This is a plausible explanation for the rise from 143/65 standing to 180/78 sitting and subsequent fall to 123/61 on re-standing, but venous return and vascular resistance were not measured directly [2,14,18]. Walking adds a second layer of demand. Safe gait requires continuous integration of vision, vestibular input, proprioception, attention, executive control and motor output. Cerebral oxygenation studies in older adults show associations between impaired orthostatic cerebral recovery and slower gait or falls, although these population data do not establish the mechanism in this individual and do not substitute for a direct cerebral blood-flow measurement here [7-10,12,17]. The phrase 'baroreflex fatigue' should be avoided unless a specific mechanism is demonstrated. The more accurate formulation is failure of the integrated compensatory system to sustain vascular resistance, venous return, cardiac output, and—potentially—cerebral perfusion across time.

4.3. Integrated Physiological Explanation

The overall pattern is most coherently explained by a posture- and duration-dependent reduction in circulatory reserve. Normal orthostatic compensation requires sustained vasoconstriction, adequate circulating volume, appropriate cardiac responses, and effective skeletal-muscle and respiratory pumps. In this case, these mechanisms appear sufficient for brief upright tasks but may become inadequate as standing and walking continue. The initial orthostatic pressure cost may leave less reserve for later demands. Venous pooling, exercise-related vasodilation, medication effects, meal or fluid status, heat, fatigue, and the continuous neural demands of balance and gait may then accumulate. Once the available reserve is exceeded, systemic pressure may enter a symptomatic range. This model explains why stairs, short shopping trips, driving transitions, or the beginning of a walk may remain possible while symptoms emerge after 10-30 minutes. The 23 August fall from 135/75 before walking to a repeated standing value of 85/45 after 50 minutes provides the clearest home example of a delayed activity threshold being crossed.

The limited heart-rate response on 24 August is also notable. BP fell from 180/78 sitting to 123/61 on re-standing while heart rate rose only from 71 to 77. This pattern may reflect impaired baroreflex compensation, limited autonomic vasoconstriction, medication or volume effects, or a combination. It is a clinical signal for formal autonomic testing, not proof of a single mechanism. Head, visual, balance, and gait symptoms are physiologically plausible during systemic hypotension because safe walking depends on stable perfusion and rapid integration of visual, vestibular, proprioceptive, and motor information. Cerebral blood flow was not measured, so under-perfusion remains a reasoned possibility rather than a demonstrated finding. Recumbent cycling fits the same model: recumbency reduces the vertical hydrostatic column and gravitational pooling, supports venous return, and removes much of the balance and foot-placement demand imposed by walking. The post-walk readings add a final component. When walking stops, muscle-pump support diminishes while exercise-related vasodilation may persist, brief sitting can raise pressure, and re-standing can then reveal marked post-exercise orthostatic susceptibility.

4.4. Why Recumbent Cycling can Remain Possible

Preserved recumbent cycling is physiologically coherent and does not make the walking symptoms less real. Walking and cycling may begin from the same usual hypertensive pre-activity range, but they do not impose the same transition. Recumbent cycling avoids or markedly reduces the immediate 30-50 mmHg vertical pressure cost. Recumbency reduces the hydrostatic column and gravitational pooling, supports central blood volume and preload, lowers the centre of mass and removes most balance and foot-placement demands. Rhythmic leg contractions can assist venous return. Horizontal exercise is deliberately used in orthostatic-intolerance rehabilitation because it permits cardiovascular work while avoiding the upright posture that provokes symptoms. The supporting rehabilitation literature is largely based on postural tachycardia syndrome and is used here only as a positional physiological analogy, not as evidence that this patient has POTS [15]. A shopping trolley or rollator provides a partial parallel benefit: it adds external support, reduces postural-control demand and offers immediate access to stability. The benefit of support does not identify the cause and does not prove that prolonged walking is safe.

4.5. What the 23 and 24 August events add

Earlier observations established the recurring phenotype but left gaps in timing and pairing. The 19 August series improved symptom linkage by showing 121/72 without symptoms after three minutes standing, 107/53 with head symptoms after approximately 30 minutes of activity, and 103/56 after seated rest. The 23 August observation is stronger again: BP/HR changed from 135/75 and 65 before walking to a repeated standing value of 85/45 and 85 after 50 minutes of brisk walking, during head fog and impaired balance, followed by recumbent recovery. Exercise can itself produce a transient reduction in pressure through sustained vasodilation, particularly in hypertensive individuals [18]. Consequently, the measurement could reflect upright pooling, exercise vasodilation, impaired autonomic compensation or a combination. The decisive data would be continuous or repeated pressure and heart-rate measurements across baseline, the first 10 minutes, symptom onset, the fall threshold and recumbent recovery. The 24 August event adds a different capture.

The first post-walk standing value of 143/65 was obtained after symptoms had already been present for approximately 30 minutes and therefore cannot reconstruct the earlier trajectory. Persistent symptoms at that point remain clinically important but do not establish ongoing cerebral under-perfusion. The rise to 180/78 sitting and fall to 123/61 on re-standing demonstrate marked positional lability after exercise. The accompanying heart-rate increase from 71 to 77 was small relative to the 57 mmHg systolic fall and warrants formal characterization [19]. The two events are complementary rather than contradictory. On 23 August, the captured endpoint was severe absolute hypotension at 85/45, on 24 August, it was a large relative postural fall from a hypertensive seated pressure, with a higher standing value of 123/61. Intermittent arm-cuff readings need not map perfectly to symptoms. A safely supervised walking-equivalent protocol with continuous beat-to-beat BP, rhythm, and symptom marking would clarify the systemic nadir, the effect of stopping movement, and the recovery pattern. Specialist cerebral-perfusion testing would be relevant only if clinically indicated or if systemic haemodynamics did not explain the disturbance.

4.6. Predictability, Adaptation, and the Meaning of Improvement

This document examines whether increasing predictability of the walking-related event reflects physiological improvement, or whether it is better understood as improved warning, adaptation, and self-management. The orthostatic-hypotension literature generally defines and follows the disorder through blood-pressure behaviour, upright tolerance, symptom burden, falls or syncope, functional capacity, and response to treatment. Recognition of a reproducible warning period can be valuable because it permits earlier sitting or recumbence and may reduce injury risk. However, symptom awareness or a more stereotyped latency is not, by itself, an established marker that vascular autonomic control has recovered. Improvement in the underlying physiology would be more convincingly supported by smaller or less sustained upright pressure falls, longer safe upright tolerance, fewer symptomatic episodes or falls, improved compensatory heart-rate and vascular responses, or improved findings on standardised autonomic testing [1-5,11,14].

In the present record, the ability to recognise that something is wrong after approximately 10-20 minutes of walking may therefore represent improved interoceptive recognition, adaptation, or a more reproducible threshold. It may also make the condition more manageable. Those functional gains should not be conflated with resolution of the haemodynamic abnormality. Significant classical orthostatic falls remain present, while delayed walking-related hypotension and post-exercise orthostatic susceptibility are relatively new or newly documented features. The fall,

four near-falls, repeated 85/45 mmHg post-walk pressure, and 57/17 mmHg post-exercise postural fall do not independently support physiological recovery. The literature also treats delayed orthostatic hypotension as a clinically meaningful orthostatic phenotype rather than as evidence of recovery from classical orthostatic hypotension. Delayed and classical forms can differ in timing and clinical profile, and delayed hypotension requires prolonged upright observation because brief measurements may miss it. Accordingly, the new delayed walking component should be characterised rather than interpreted automatically as improvement [2-5].

The most balanced formulation is that predictability may indicate improved warning, adaptation, or day-to-day control, while the available home data continue to show substantial autonomic haemodynamic impairment and an evolving activity-related phenotype. Determining whether there has been physiological improvement requires comparison with prior standardised measurements, ideally using prolonged symptom-linked beat-to-beat blood pressure, heart rate, rhythm, and functional endpoints. The relevant clinical question is therefore: does 'improvement' refer to better recognition and manageability, or to demonstrably improved autonomic blood-pressure regulation?

4.7. Why the New Fall Cluster Changes Urgency

The Pitt Street fall and four near-falls transform the issue from symptom explanation to injury prevention. Cardiovascular falls may arise from hypotension, arrhythmia, reduced cardiac output or medication effects, while neurological, sensory, gait and environmental factors may coexist [16]. Beat-to-beat evidence of impaired orthostatic pressure recovery has been associated with subsequent unexplained and injurious falls in older adults [17]. Although those findings cannot predict this individual's outcome, the recurrence and apparent shortening threshold justify prompt assessment and immediate mobility precautions.

5. Evidence-Graded Formulation

- Observed by repeated home measurement: the usual living or pre-activity pressure before walking or cycling is 165-195/85-95 mmHg, with an immediate systolic fall of approximately 30-50 mmHg on standing.
- Observed: initially preserved movement followed by delayed head disequilibrium, lateral instability and impaired step control during sustained upright activity.
- Observed: one fall on Pitt Street after approximately 10 minutes of standing and walking, followed by four near-falls while walking.
- Observed: a post-walk brachial pressure below 105/75 mmHg after 45 minutes, without a paired baseline or heart-rate value.
- Observed on 19 August 2026: 121/72 with HR 78 after three minutes standing and no immediate symptoms, followed after approximately 30 minutes of activity by 107/53 with HR 85 and head fog, then 103/56 with HR 78 after seated rest with persistent symptoms.
- Observed on 23 August 2026 at 9:15 am: pre-walk BP was 135/75 with HR 65, after a 50-minute brisk walk with the dog and while still standing, BP was 85/45 with HR 85 during head fog, impaired balance and general unwellness, a repeat within one to two minutes yielded the same result and recumbent feet-elevated recovery followed.
- Observed for the 23 August event: the arm was held at heart level, the repeat value was unchanged, systolic pressure recovered by approximately 10 mmHg every five minutes over 10-15 minutes, and heart rate fell to approximately 75 bpm during recovery.
- Observed on 24 August 2026: symptoms began after approximately 20 minutes of a 50-minute brisk walk and worsened, after returning home, standing BP was 143/65 with HR 64, sitting BP approximately two minutes later was 180/78 with HR 71, and re-standing BP was 123/61 with HR 77.
- Derived from the 24 August post-walk pair: a 57/17 mmHg sit-to-stand fall, an estimated mean arterial pressure reduction from approximately 112 to 82 mmHg and a delta-HR/delta-SBP ratio of approximately 0.11, these derived measures are clues, not cerebral-flow measurements or a formal neurogenic classification.
- Observed at 7:00 pm on 24 August: 171/72 with HR 65 sitting and 152/63 with HR 71 standing, indicating substantial but incomplete reduction of the earlier postural lability.
- The 24 August event contributes walking start time, symptom latency, sequential post-walk BP/HR values, and an approximately 15-second re-standing interval. Further supervised monitoring could add repeat confirmation, continuous rhythm and pressure tracing, arm-position standardisation and pressure measurement at symptom onset.
- Observed: substantially better tolerance of recumbent cycling and improved walking stability with external support.
- Strongly suggested: a posture- and duration-dependent physiological threshold rather than continuous global muscle weakness.
- Supported as a home-observed phenotype: delayed symptomatic hypotension compatible with dOH, but not formally classified by a standardised laboratory protocol.
- Plausible associated mechanism: reduced circulatory reserve with possible cerebral under-perfusion inferred from systemic hypotension and head, visual, balance, and gait symptoms, cerebral blood flow was not directly measured.
- Specialist characterisation could clarify the haemodynamic mechanism, heart-rate and baroreflex responses, and formal reproduction of dOH, cerebral blood-flow or oxygenation assessment would be added only when clinically indicated.
- Not excluded: intermittent arrhythmia, structural or functional cardiac limitation, transient cerebral ischaemia, vestibular dysfunction, sensory ataxia, metabolic disturbance, medication effect or another neurological disorder.

6. Differential Diagnosis and Red Flags

The autonomic formulation is coherent because of the delayed threshold, documented pressure lability, recovery with recumbence, preserved cycling and benefit from external support. Nevertheless, recurrent loss of gait control is neurologically consequential. Orthostatically provoked ataxia has rarely been reported with

vertebrobasilar insufficiency, illustrating why focal or posterior-circulation signs should not be absorbed automatically into a dysautonomia label [13].

The differential diagnosis includes:

- delayed orthostatic hypotension or another form of orthostatic intolerance,
- possible orthostatic cerebral under-perfusion, which is not measured in this home record and would require specialist assessment before it could be stated as a finding [6].
- post-exercise hypotension superimposed on orthostatic vulnerability [18].
- intermittent bradyarrhythmia, tachyarrhythmia or ectopy-related haemodynamic compromise,
- transient ischaemic attack, posterior-circulation insufficiency or another neurological disorder,
- vestibular dysfunction, sensory neuropathy or proprioceptive impairment,
- volume depletion, anaemia, electrolyte abnormality, hypoglycaemia or infection, and
- medication-related vasodilation or volume reduction in the context of supine hypertension.

Emergency assessment is required for abrupt unilateral weakness or numbness, facial asymmetry, dysarthria, diplopia or sustained visual loss, a new severe headache, chest pain, palpitations with collapse, loss of consciousness, persistent inability to walk, or a significant head injury. A new focal event should be treated as possible stroke or TIA until assessed.

7. Proposed Diagnostic Pathway

The investigation must reproduce the patient's actual latency safely rather than stop at the conventional three-minute office measurement. Subject to the treating clinicians' judgement, the highest-yield sequence is:

- Prolonged active stand or head-up tilt extending beyond the usual 10-minute threshold, with continuous beat-to-beat blood pressure, heart rate, symptom marking and immediate tilt-back or seating capability [2,5].
- Continuous ECG during provocation and ambulatory rhythm monitoring if the event is not captured, to assess intermittent arrhythmia or ectopy-related haemodynamic compromise.
- A supervised activity protocol approximating the walking trigger, because passive tilt and active walking do not impose identical muscle-pump, vasodilatory and balance demands. Monitoring should continue through cessation of walking, quiet standing, brief sitting, re-standing and recovery so that post-exercise and orthostatic components can be separated.
- If symptoms occur without an explanatory systemic pressure fall, specialist assessment could consider transcranial Doppler cerebral blood-flow velocity or near-infrared cerebral oxygenation monitoring, ideally with end-tidal carbon dioxide. Such testing is outside the scope of the present home record [6,8,9,12].
- Focused neurological, vestibular and gait examination before and during symptoms, documenting laterality, eye movements, cerebellar signs, proprioception, strength, stride rhythm, veering and the effect of external support.
- Medication and volume review including timing of tamsulosin, losartan, spironolactone and furosemide, renal function, electrolytes, haemoglobin, hydration, meal timing and nocturnal pressure profile [11,14].
- Brain and vascular imaging when events are abrupt, focal, stereotyped, progressive or accompanied by posterior-circulation symptoms [13].
- A structured diary recording time upright, task, meals, fluids, medication timing, ambient temperature, symptoms, falls or near-falls, laterality, safely obtained BP/HR and recovery after sitting or lying.

8. Immediate Safety Implications

Once step control becomes unreliable, the activity has crossed a safety threshold even if consciousness is preserved. The current record supports using the rollator from the beginning of walking rather than waiting for symptoms, avoiding unsupported prolonged walking or self-provocation, remaining close to seating, walking with another person when possible and stopping at the first head, visual, directional or step-control warning. These measures reduce injury risk but do not diagnose or treat the underlying cause. Medication, salt and fluid changes should not be undertaken independently. The coexistence of supine hypertension and orthostatic falls makes simple pressure-raising or pressure-lowering strategies potentially hazardous and requires individual clinical management [11,14]. Any head strike from the Pitt Street fall warrants a low threshold for medical review, particularly if accompanied by loss of consciousness, confusion, vomiting, worsening headache, anticoagulant use or new neurological symptoms.

9. Patient Perspective

The defining experience is not merely feeling faint. After a period of normal movement, the head and the next step no longer seem to work together automatically. Walking becomes deliberate and unsafe. The later ability to use a recumbent bicycle does not cancel the event, it demonstrates that function changes sharply with posture, duration and task. The Pitt Street fall and four near-falls have made this unpredictability more than an inconvenience. They have reduced confidence in unsupported walking and established a practical need for protection before the threshold is crossed.

10. Limitations

This is a single-person narrative based on patient-kept longitudinal records and selected medical history. The August 2026 fall and near-falls are home-observed events supported by symptom-linked cuff measurements and recovery patterns. The 19, 23, and 24 August observations add clinically useful data on posture, activity, symptoms, BP/HR change, and recumbent recovery, but they do not provide continuous beat-to-beat pressure, ECG, neurological examination, glucose assessment, or cerebral blood-flow measurement. The 23 August record includes a same-session pre-walk BP/HR, activity duration, standing post-walk posture, symptoms, repeat 85/45 measurement, monitor and cuff details, arm position, and recumbent recovery. The 24 August record

includes pre-walk BP/HR, walking duration, symptom latency, sequential post-walk values, an approximately 15-second re-standing interval, and approximate symptom recovery. Specialist assessment could standardise posture and timing, capture pressure and rhythm at symptom onset, and document medication, meal, fluid, temperature, and injury context. These limitations constrain mechanistic and formal subtype claims but do not erase the central observation: clinically significant, symptom-linked systemic pressure instability has been captured at home.

11. Conclusion

The case is most coherently described as a two-stage orthostatic sequence: a reported usual pre-activity pressure of 165-195/85-95 mmHg falls immediately by approximately 30-50 mmHg systolic on standing, after an initially functional period, sustained upright activity produces delayed hypotension and gait dyscontrol. In August 2026, this escalated to one fall and four near-falls. The 19 August symptom-linked decline, the 23 August fall from 135/75 and HR 65 before walking to a repeated standing value of 85/45 and HR 85 after a 50-minute brisk walk, and the 24 August delayed symptomatic walk followed by a 57/17 mmHg post-exercise sit-to-stand fall materially strengthen the formulation. The phenotype is best described as delayed upright-activity intolerance with home-documented delayed symptomatic hypotension compatible with dOH and superimposed post-exercise orthostatic susceptibility. The evidence does not directly demonstrate cerebral under-perfusion or identify a single autonomic mechanism. It demonstrates systemic blood-pressure instability, severe hypotensive episodes, and symptom-linked disturbance of head sensation, vision, balance, and gait in a home setting. Formal characterisation should use prolonged symptom-linked beat-to-beat pressure, heart-rate, and rhythm monitoring, neurological, vestibular, cerebrovascular, or cerebral-perfusion assessment should be added when clinically indicated or when systemic haemodynamics do not adequately explain the motor disturbance.

The patient's increasing ability to anticipate the walking-related event is a meaningful functional advantage for injury avoidance, but the literature does not establish predictability alone as a marker of autonomic recovery. In this case, persistent classical orthostatic falls and the newer delayed walking-related hypotensive phenotype support separating improved recognition from improvement in the underlying haemodynamics. The decisive new development is the transition from symptom explanation to fall-risk management. The Pitt Street fall and recurrent near-falls require prompt clinical assessment and immediate preventive mobility measures while the mechanism is investigated.

Declarations

Consent. The patient and author are the same person and consent to the use of this clinical narrative and the identifying author information in the working paper.

Ethics. This is a patient-authored narrative case study and not an interventional investigation. Journal-specific ethics and consent requirements should be confirmed before external submission.

Funding and Conflicts of Interest: No external funding or competing interest is reported for this working paper.

Editorial Assistance: Language-model assistance was used as an accessibility, communication and editorial support tool. The author reviewed and approved the clinical content, interpretations and final wording.

Assistive Technology Declaration: Assistive technology, including language-model support, was used to assist with drafting, organisation, editing, formatting, and accessibility of this working paper. The author retained responsibility for the clinical observations, interpretation, accuracy, and final approval of the text.

Plain-Language Summary

This case study examines a delayed, posture-dependent walking disorder in which upright walking progressively becomes unsafe while recumbent cycling remains relatively preserved. Walking begins after an immediate fall in blood pressure on standing and may remain possible for a limited period before head symptoms, balance, and step control deteriorate. Recumbent cycling avoids much of the gravitational pooling and postural-control demand that appear to provoke the problem. The exact mechanism remains unproven, but the pattern is compatible with failure of the integrated circulatory compensation needed to sustain blood pressure during prolonged upright activity. Possible contributors include autonomic dysfunction, impaired cardiovascular reflexes, limited vascular constriction, medication or volume effects, residual cardiovascular injury, or a combination. The 23 and 24 August events provide complementary evidence: one captured severe standing hypotension after prolonged walking, while the other captured a large post-exercise fall on re-standing. Transient cerebral under-perfusion is plausible but remains an inference from systemic blood-pressure data and symptoms rather than a directly measured finding. Being able to predict the onset more reliably may improve safety and day-to-day control, but does not by itself show that autonomic blood-pressure regulation has recovered.

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