ISSN: 2690-909X

Research Article

Advances in Neurology and Neuroscience

The Measure of What Remains An 18-year Case Study of Autonomic Reserve, Systemic Collapse, Partial Recovery, and Life within a Changing Physiological Threshold

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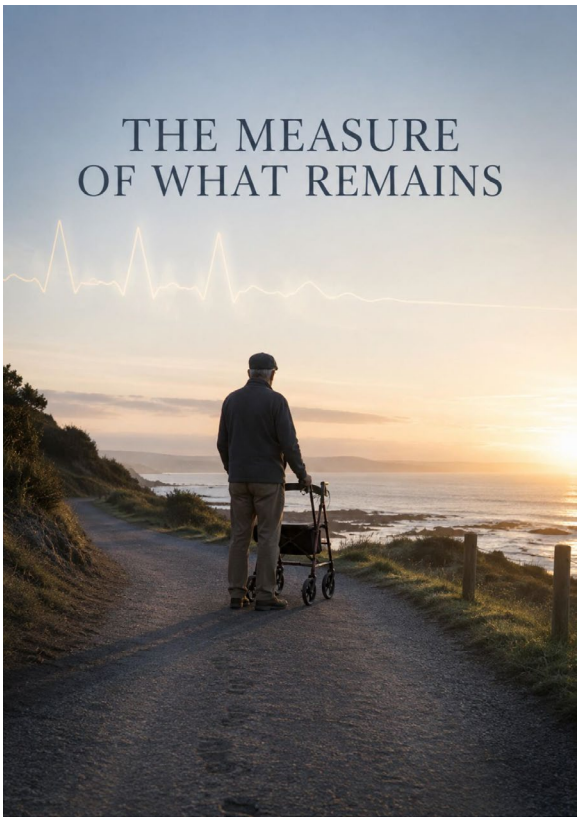
Submitted: 2026, Aug 12; **Accepted:** 2026, Sep 11; **Published:** 2026, Sep 22

Citation: Knox, B. H. (2026). The Measure of What Remains An 18-year case study of autonomic reserve, systemic collapse, partial recovery, and life within a changing physiological threshold. *Adv Neur Sci, 9*(3), 01-15.

Central narrative. My autonomic reserve has not behaved like a battery that was once full and then emptied. Instead, it has behaved like a changing margin: injured by cumulative events, overwhelmed in crisis, partly rebuilt through recovery and adaptation, yet still insufficient for some of the ordinary demands that matter most.

The narrative distinguishes recorded events from physiological interpretation and is not a substitute for diagnosis or treatment. It should be read alongside Supplementary Appendix A, which provides the evidence-informed medical and physiological foundation for its principal interpretations.



Abstract

This first-person case study describes the changing shape of autonomic reserve across an 18-year illness trajectory. Before illness, I was physically active and professionally accustomed to sustained planning, analysis and responsibility. Chikungunya infection in Bandung in November 2008 was followed by years of orthostatic intolerance. A later burden of frequent ventricular ectopy culminated on 15 October 2021 in an ablation for left-ventricular-outflow-tract ectopy complicated by left-ventricular free-wall perforation, cardiac tamponade, emergency sternotomy, cardiopulmonary bypass and ventricular repair. During 2022-2023, cardiovascular instability widened into multisystem collapse involving blood-pressure lability, thermoregulation, gastrointestinal function, elimination, fatigue and cognitive slowing. Meaningful but asynchronous improvement emerged during 2024-2025: autonomic storms ceased, sweating and temperature control returned, exercise heart-rate responsiveness improved, and participation expanded. Yet by 2026, orthostatic control, bowel and bladder function, postprandial endurance and sustained upright walking remained impaired or had worsened. In August 2026, delayed head and gait symptoms, a fall, repeated near-falls and symptom-linked hypotensive readings made the residual deficit unmistakable. The concept of autonomic reserve provides a disciplined language for this mixed history. It does not explain every symptom or establish the original cause. It does, however, describe how residual capacity can support one task, posture or organ system while failing another; how recovery and deterioration can coexist; and why a person can appear capable at the beginning of a walk yet become unsafe after a reproducible period upright. The accompanying Supplementary Appendix A examines the physiological plausibility, published support, alternative explanations and evidentiary limits of this account.

Pragmatic Conclusion. The evidence supports severe, fluctuating autonomic dysfunction, including documented orthostatic pressure loss and supine hypertension, together with genuine recovery in selected domains. It does not establish a single lesion, a validated global reserve score, permanent baroreflex damage, cerebral hypoperfusion during every event, or a settled prognosis.

Author's Note: how I am Telling this Story

I am both the person who lived this history and the investigator attempting to make it intelligible. That position provides an unusually detailed record, but it also creates risks: memory is selective; crisis magnifies; improvement can invite hopeful over-interpretation; and repeated self-measurement can generate its own diagnostic momentum. Narrative medicine treats lived experience as clinically relevant evidence while insisting that it be interpreted rather than romanticised [1-18]. The CARE framework likewise asks case reports to make chronology, uncertainty and limitations visible. Accordingly, this narrative distinguishes four evidentiary levels. Dated procedures and recorded blood pressures are treated as observations. Symptoms and functional thresholds are treated as first-person data. Mechanisms such as impaired vasoconstriction, inadequate venous return, chronotropic limitation or reduced cerebral perfusion are treated as hypotheses unless directly measured. Umbrella terms such as multifactorial secondary autonomic dysfunction and autonomic reserve remain explanatory constructs rather than consensus diagnoses. Supplementary Appendix A applies the same discipline to the medical literature and sets out a claim-to-evidence concordance for the major conclusions advanced here.

Prologue: before Reserve had a name

For most of my working life, reserve was something I used without noticing. In senior educational leadership, quality assurance and academic research, I relied on being able to remain upright, think for long periods, move between settings, travel, make decisions and recover from exertion without consciously planning the physiology of each step. I was physically active. My body was not frictionless, but it was sufficiently dependable that intention usually became action.

Illness changed that relationship gradually, then catastrophically. The central loss was not simply strength, but reliability. Standing, eating, walking, heat, sleep, bowel function and bladder emptying ceased to be background processes and became variables to manage. The body that once carried my plans became a system I had to supervise.

The Longitudinal Arc

Period	Dominant phase	What happened	Meaning for reserve
2008	Infectious breach	Severe chikungunya infection in Bandung.	Probable initiating contributor; chronic causation remains unproven.
2009-2015	Orthostatic phase	Recurrent light-headedness, hypotension and upright intolerance.	Early evidence that cardiovascular compensation was less reliable.
2016-2021	Cardiac burden	Frequent ectopy, at peak about 15,000 PVCs in 24 hours, and rhythm treatment.	Confirmed cardiac load; plausible amplifier of autonomic vulnerability.
15 Oct 2021	Major insult	Ablation perforation, tamponade, emergency sternotomy, bypass and ventricular repair.	A profound acute physiological insult to a system already carrying earlier burden.

2022-2023	Systemic collapse	Pressure storms, hypotension, heat and sweating failure, gastrointestinal dysfunction, fatigue and cognitive slowing.	Reserve became insufficient across several domains, and ordinary life lost predictability.
2024-2025	Selective recovery	Storms ceased; thermoregulation, heart-rate response and participation improved.	Some pathways recovered or were compensated for; the whole system did not normalise.
2026	Mixed state	Persistent pressure instability, bowel and bladder dysfunction, postprandial crashes, and delayed upright gait failure.	A better overall life than at the nadir, but with a narrow and task-specific upright margin.

Table 1: Timeline Derived from the Longitudinal Case Record. Causal Confidence is Deliberately lower than Confidence in Dated Events and the Observed Phenotype.

1. The First Breach: Bandung, 2008

In November 2008, I contracted chikungunya in Bandung, Indonesia. In retrospect, this became the first major landmark in the story because orthostatic symptoms followed and persisted. Yet a careful case study must resist the simplicity of a single-cause narrative. Chikungunya is associated with neurological complications, including peripheral nervous-system involvement, but the literature does not establish that one infection caused my chronic autonomic disorder across the following eighteen years. The most defensible formulation is therefore temporal and probabilistic: the infection was a probable initiating contributor, not a proven complete explanation. Between 2009 and 2015, recurrent light-headedness and hypotensive episodes made standing and movement unreliable. At that stage, the pattern was narrower than the illness that followed. The system still functioned, but the available margin appeared reduced. Reserve, though unnamed, was already becoming visible in the gap between what I could begin and what I could sustain.

2. Accumulating Load: the Cardiac Years, 2016-2021

From 2016, frequent ventricular ectopy added a different burden. At its peak, approximately 15,000 premature ventricular contractions were recorded in 24 hours. The ectopy is established; its relationship to autonomic dysfunction is less certain. Autonomic tone can influence ventricular excitability; conversely, rhythm disturbance, symptoms, medications, altered activity and haemodynamic stress can affect autonomic regulation. In my case, the cardiac burden is established. Its precise contribution to the later dysautonomia cannot be calculated. This distinction matters because cumulative illness invites hindsight. Once the whole pattern is visible, each earlier event can be made to look inevitable. It was not. The years before 2021 contained vulnerability, but they also contained work, movement, competence and adaptation. Reserve was not absent; it was being asked to carry more.

3. The Day the System was Overwhelmed: 15 October 2021

On 15 October 2021, an ablation for left-ventricular-outflow-tract ectopy was complicated by perforation of the left-ventricular free wall and cardiac tamponade. Emergency sternotomy, cardiopulmonary bypass and ventricular repair followed during an operative course of approximately six hours. This was not a metaphorical shock; it was a documented major physiological insult involving the heart, circulation, surgery, anaesthesia, inflammation, pain, immobility and critical care. Studies show that cardiac surgery can acutely impair baroreflex and cardiovascular autonomic measures, although improvement often occurs over weeks or months [16,17]. These studies do not prove that surgery caused my long-term multisystem dysfunction. They support a narrower statement: the event was biologically capable of disturbing autonomic cardiovascular control and was a probable amplifier of vulnerability already present. The evidence cannot separate the effects of tamponade, haemorrhage, bypass, direct repair, anaesthesia, postoperative inflammation, medication and deconditioning.

A cautious causal formulation. Chikungunya may have opened the vulnerability; years of orthostatic and cardiac burden may have narrowed the margin; and the 2021 catastrophe may have overwhelmed it. That sequence is coherent, but it is not laboratory proof of a single causal chain.

4. The Low Point: 2022-2023

The period after surgery did not settle into a single stable deficit; dysfunction widened across systems. Blood pressure could surge to approximately 180/110 mmHg or higher and at other times fall below 90 mmHg systolic. I came to describe the paroxysms as autonomic storms: tremor, sweating or disturbed temperature control, head symptoms, weakness, internal adrenergic intensity and profound fatigue. A later recorded example showed the paradox plainly: 215/105 mmHg supine, then 145/85 within 30 seconds of standing, with heart rate changing only from 55 to 58 beats per minute. High pressure did not imply abundant usable reserve; it could coexist with an inability to buffer the change of posture [5,13]. At the lowest point, I described myself as a 'complete dysfunctional disaster'. This did not mean that every organ had failed; it meant that the coordination of ordinary life had failed. Standing could abruptly remove function. Meals could produce depletion. Heat could become intolerable. Sweating and temperature control were unreliable. Fatigue could make the body unavailable despite intact intention. Cognitive processing slowed. The day was no longer organised primarily by purpose; it was organised by physiological risk.

5. One Body, Many Systems

5.1. Cardiovascular Regulation

Supine or seated hypertension coexisted with orthostatic

hypotension. These states are not contradictory. In autonomic failure, impaired buffering can permit pressure to overshoot while recumbent and collapse when upright [5,13]. For me, this opposition made management inherently difficult: measures that might protect standing pressure could worsen recumbent hypertension, while aggressive pressure reduction could narrow the margin for walking or rising.

5.2. Gastrointestinal Burden

Delayed gastric emptying, early satiety, nausea, bloating, reflux and oesophageal dysfunction altered nutrition and daily timing. Eosinophilic oesophagitis and pancreatic exocrine insufficiency required disease-specific recognition; pancreatic enzymes and gastric peroral endoscopic myotomy addressed important components. It would be inaccurate to label every gastrointestinal diagnosis 'autonomic'. Delayed motility can be compatible with autonomic or enteric dysfunction, whereas eosinophilic inflammation and exocrine insufficiency have distinct mechanisms. The cumulative lived effect was nevertheless substantial: food itself became a physiological load.

5.3. Bowel and Bladder

Constipation, difficult evacuation and episodes of impaction came to dominate parts of the day. Urinary hesitancy, incomplete emptying and retention progressed to intermittent self-catheterisation, commonly morning and evening. Catheter passage sometimes seemed more difficult when the bowel was loaded and easier after evacuation. This observation warrants investigation of pelvic mechanical, outlet, sensory and reflex interactions; it does not prove a single shared autonomic lesion.

5.4. Cognition, Fatigue and Participation

Cognitive change was experienced primarily as slowed processing rather than a dominant memory syndrome. Fatigue was not ordinary tiredness, but a reduction in available function, often intensified after meals or prolonged upright activity. Observational research associates orthostatic hypotension with cognitive impairment, but association cannot determine the cause of an individual's cognitive profile [19,20]. Sleep, medication, nutrition, metabolic factors, cerebrovascular regulation and the burden of constant self-management remain alternative or interacting contributors.

What the low point took away. The deepest loss was bodily trust. Every plan required an exit route: somewhere to sit, a meal strategy, a temperature strategy, access to a toilet, medication timing and a judgement about whether a symptom was familiar or dangerous.

6. The Highs: Recovery Returns in Pieces, 2024-2025

Recovery did occur, but it did not announce itself as cure. It appeared through the return of small but meaningful functions. The paroxysmal pressure storms ceased. Sweating and temperature regulation progressively normalised. Heart rate again responded to walking and exertion, often reaching about 100 beats per minute during walking and 115-120 with greater effort after a period in which the response had seemed markedly constrained. Activity tolerance and participation expanded. These were genuine highs because they restored not only physiological functions but choices. A walk completed without collapse, appropriate sweating, a better-tolerated meal or a heart rate that rose with effort represented evidence that the system was not fixed at its worst state. The body had retained some capacity for repair, compensation, retraining, or some combination of all three. The most important shift was from waiting for a cure to building conditional agency: the ability to do valued things within known constraints. Pacing, recumbent exercise, fluid and salt strategies within cardiovascular limits, compression, head-up sleeping, smaller meals, muscle-pump manoeuvres, heat avoidance and equipment became part of that work. Using a tricycle, trolley or rollator was not a declaration of defeat; each was a tool for extending participation without pretending that the risk had disappeared.

The high was not normality. It was the return of options: movement, thought, participation and the ability to recognise a pattern before it became a crisis.

7. Recovery did not Move in a Straight Line

By 2026, neither 'recovered' nor 'progressively failing' accurately described the whole person. Thermoregulation had improved. The storms had stopped. Exercise heart-rate responsiveness and participation were better than at the nadir. At the same time, orthostatic blood-pressure control remained abnormal; severe colonic dysfunction and urinary retention had become more prominent; postprandial crashes and slowed processing persisted; and walking acquired a new delayed threshold. This is asynchronous recovery. The autonomic nervous system is a distributed network, not a single cable. Baroreflex sensing, central integration, sympathetic vascular control, cardiac chronotropy, sweat fibres, enteric circuits, pelvic parasympathetic pathways, skeletal-muscle pumps, renal volume regulation and learned behavioural strategies can change on different timelines. Improvement in one domain does not certify recovery of the others, and deterioration in one domain does not erase authentic gains elsewhere. There is an emotional cost to this mixed trajectory. Improvement raises hope; renewed deterioration can feel like a betrayal. The evidence supports a steadier account: recovery can be real without being global, and decline can be serious without proving that the whole system is inevitably degenerating.

8. The New Low: Walking Fails while Recumbent Cycling Persists

During August 2026, the limits of remaining cardiovascular reserve became visible through a striking dissociation. I could often stand, climb stairs, drive, begin shopping or start a walk normally. After approximately 10-20 minutes upright, however, light-headedness, blurred or greyed vision, pressure in the head, fatigue and loss of automatic gait control emerged. Stride rhythm deteriorated. I veered. Foot placement required conscious attention. Sitting or lying down helped. Recumbent cycling remained substantially better tolerated. On 17 August, ordinary morning activity, driving and initial work on a trailer were tolerated. After about 20 minutes of standing, bending and moving around the trailer, head disequilibrium and impaired step control emerged and fluctuated for nearly two hours.

After rest and coffee, I could cycle without the same recurrence. Later, unsupported supermarket walking produced mild instability, while holding a trolley improved function.

Later that month, I fell on Pitt Street after approximately 10 minutes of combined standing and walking. Four subsequent near-falls followed. The previously recognised window appeared to have shortened. That shortening may represent true deterioration, although temperature, task, meal timing, sleep, hydration, medication timing and starting pressure were not controlled. What is established is the functional change and the associated injury risk.

9. When the Numbers Met the Story

Home measurements cannot substitute for continuous laboratory data, but several August observations brought objective measurements into closer alignment with the lived pattern. On 19 August, a three-minute standing value of 121/72 mmHg did not coincide with a clear immediate symptomatic event. After approximately 30 minutes of walking and activity, pressure had fallen to 107/53 as head fog, a sense of being 'out of kilter', and reduced steadiness appeared. After 30 minutes of seated rest, standing pressure remained 103/56 and recovery was incomplete. On 23 August, immediately after a 50-minute brisk walk with the dog and while still standing, I recorded 85/45 mmHg with heart rate 85 during head fog, impaired balance and a general feeling described in my notes simply as 'feeling yuk'. Lying down with my feet elevated was followed by improvement over roughly 10-15 minutes. The reading was not paired with a same-session pre-walk baseline, and cuff position introduced measurement limitations. Nonetheless, it documented severe absolute hypotension during a delayed symptomatic event. On 24 August, symptoms began about 20 minutes into another 50-minute brisk walk and worsened on the way home. Immediately afterwards, standing pressure was 143/65 with heart rate 64. After about two minutes seated, it was 180/78 with heart rate 71. On standing again, it fell to 123/61 with heart rate 77: a postural reduction of 57/17 mmHg with only a six-beat heart-rate rise. This was not a standard diagnostic active-stand test and does not establish what pressure did during the walk. It does, however, demonstrate marked post-exercise orthostatic susceptibility.

What these observations establish. There is delayed, threshold-dependent upright gait dyscontrol with falls and home-documented symptom-linked hypotension. The pattern is compatible with delayed orthostatic and post-exercise hypotension. The formal mechanism and classification remain open. Appendix A explains why this combination is physiologically coherent while preserving the distinction between compatibility and diagnosis.

10. The Anatomy of my Reserve Threshold

The reserve model explains why the beginning of a walk can look deceptively normal. Standing immediately shifts blood toward the legs and splanchnic circulation. My usually high pre-activity pressure may then fall by 30-50 mmHg without immediate collapse. Residual vasoconstriction, heart-rate response, cardiac contraction, circulating volume and the leg-muscle pump may still preserve consciousness and movement. That initial success consumes part of the available margin [1-3]. Walking then adds metabolic demand and active-muscle vasodilatation to the gravitational load. Heat production, skin blood flow, sweating, meal-related splanchnic demand, medication effects or low starting volume may narrow the margin further. In sympathetic denervation, vascular resistance can fall excessively during and after exercise, and blood-pressure recovery can be impaired [6,7]. If heart-rate or stroke-volume reserve is limited, cardiac output may not compensate [8]. A threshold may then be crossed at which stable pressure, cerebral perfusion, attention and automatic gait can no longer all be maintained.

This is why I avoid the phrase 'baroreflex fatigue'. A baroreflex is not an isolated muscle shown to tire after ten or twenty minutes. The more defensible account is failure of the integrated compensatory system to sustain vascular resistance, venous return, cardiac output and cerebral perfusion under cumulative load. The integrated components, competing loads and alternative mechanisms are detailed in Supplementary Appendix A. Recumbent cycling changes that equation. It shortens the hydrostatic column, reduces pooling, supports venous return, lowers balance demand and preserves the rhythmic leg pump. The fact that I can cycle when walking has become unsafe does not establish normal exercise capacity. It does show that posture is a major component of the load and that my reserve is task-specific.

11. The Paradox of High Pressure and Low Reserve

My case repeatedly demonstrates that high pressure at rest is not equivalent to usable upright reserve. A system with impaired buffering may overshoot while supine or seated and still fail to constrict appropriately when upright [5,13]. Overnight or prolonged recumbent hypertension may also contribute to pressure natriuresis and reduce morning volume. Treatment therefore cannot be reduced to normalising a single clinic reading. This also explains why a cuff reading after a walk may miss the most important moment. Walking, stopping, sitting and re-standing each alter the muscle pump, venous return and vascular demand. The clinically decisive evidence would capture the sequence of workload, posture, beat-to-beat pressure, heart rate, rhythm, symptoms and recovery together.

My reported medication regimen has included tamsulosin, losartan, spironolactone and intermittent furosemide. Alpha-1 blockade can reduce peripheral vasoconstrictor support, while antihypertensive and diuretic effects may lower vascular resistance or circulating volume. These medicines are therefore plausible modifiers of the threshold, not demonstrated causes of it. Because the opposing problem - severe supine hypertension - is also present, medication timing or dose changes require clinician-led review rather than simple withdrawal.

12. What I can say - and what I cannot

Evidence level	Defensible statement	Boundary
Established	Recurrent orthostatic pressure loss, supine/seated hypertension, severe upright symptoms, falls and multisystem functional burden.	Home measurements vary in protocol and do not replace continuous testing.
Strongly supported	Limited, time-dependent cardiovascular autonomic reserve with a posture-exercise dissociation.	Autonomic reserve is an integrative construct, not a validated global measurement.
Plausible	Delayed orthostatic hypotension, exercise-induced or post-exercise hypotension, limited vasoconstrictor or chronotropic reserve, and episodic cerebral underperfusion.	Each proposed mechanism requires symptom-linked physiological confirmation.
Unproven	One baroreflex lesion, small-fibre neuropathy, formal neurogenic classification, a single post-chikungunya mechanism, or permanent global autonomic failure.	A secondary formulation must remain open to cardiac, medication, volume, neurological, structural, metabolic, immune and age-related contributors.

Table 2: Evidence Discipline for Interpretation of the Individual Case

13. Does Predictability mean Recovery?

A predictable warning interval has practical value. It creates time to stop, sit, use support and avoid injury. It may indicate that early compensation remains available. Compared with a period of immediate or chaotic failure, a longer window may represent partial preservation, improved conditioning, better management or selective recovery. However, predictability alone is not a recovery biomarker. Delayed orthostatic hypotension can be clinically serious and has progressed in selected referral cohorts [4]. Those findings should not be transferred mechanically to my secondary, multifactorial case, but they caution against treating delay as benign. In August 2026, the shift from roughly twenty minutes towards ten minutes, together with a fall and repeated near-falls, is more consistent with a narrower functional margin than with uncomplicated improvement. The most informative longitudinal test is comparative: under similar conditions, does symptom-free upright time lengthen; does the pressure fall lessen; does heart-rate and blood-pressure recovery improve; do near-falls decrease; and does day-to-day variability narrow? Until these outcomes are measured, the threshold is useful for safety but remains ambiguous for prognosis.

14. Pragmatic Implications

My reserve deficit should be investigated during the activity that exposes it. The most informative next assessment is not another isolated seated blood-pressure reading. It is extended, symptom-annotated monitoring: supine-to-standing measurements beyond three minutes; beat-to-beat blood pressure and ECG during standing, tilt or supervised walking; a recumbent-cycle comparator; formal exercise assessment where chronotropic limitation is suspected; ambulatory pressure monitoring linked to posture, meals, medication and sleep; and evaluation of falls, gait and non-haemodynamic contributors [2,5,8,12,14]. Appendix A translates this principle into a proposed symptom-linked investigation framework. Safety takes priority over demonstration. Once vision greys, gait loses rhythm, veering begins or presyncope develops, continuing solely to prove the threshold turns information gathering into injury risk. Support devices reduce fall exposure; they do not correct the underlying circulation. Fluid, salt, compression and medication choices require individual review because heart disease, supine hypertension, renal and urinary issues mean that apparently simple advice can help one pole while harming another [5,13,14]. The same discipline applies beyond blood pressure. Worsening bowel and bladder function should not be absorbed automatically into the dysautonomia narrative. They warrant direct gastrointestinal, pelvic-floor, urological, structural and neurological evaluation. A coherent whole must not erase the distinct mechanisms of its parts.

15. My Reserve Now

My present reserve is neither empty nor dependable. It remains demonstrably available under some conditions. I can begin many tasks. I can often think, drive, climb stairs, shop briefly, cycle recumbently and participate. The return of sweating and temperature control, the improvement in exercise heart-rate responsiveness, and freedom from autonomic storms are not imagined; they are part of the record. The central limitation is sustainability. Upright walking combines the demands my system handles least reliably: gravity, changing venous return, active-muscle vasodilatation, balance, head movement, continuous sensory integration and the absence of an immediate seat. The result is a body that may appear normal at minute one and become unsafe at minute ten or twenty. That is the shape of what remains: not a simple decline, not a complete recovery, and not a story that can be compressed into a single blood-pressure number. It is a changing relationship between burden, resilience, reserve and demand. The highs are real; the lows are real. The task is to honour both without allowing either to distort the evidence.

Closing judgement. I have recovered enough reserve to regain agency, but not enough to assume safety. The scientifically defensible position is hopeful without being falsely reassuring, cautious without being fatalistic, and committed to measuring the physiology at the moment my lived function changes.

How to use Supplementary Appendix A

The personal narrative and Appendix A serve distinct but complementary roles. This document is the primary first-person record of events, symptoms, function and meaning. Supplementary Appendix A provides the medical physiology, maps the principal narrative claims to published evidence, identifies reasonable alternatives, and states what further testing would be required. The appendix supports a coherent interpretation but does not convert that interpretation into a confirmed diagnosis or settled prognosis.

Key Messages

- Autonomic reserve is the spare, sustainable capacity of several interacting systems; it is not a single measurable tank.
- The case supports severe, partial and fluctuating dysfunction, not proven global or irreversible autonomic failure.
- Recovery has been asynchronous: thermoregulation, autonomic storms and exercise heart-rate responsiveness improved, while orthostatic, bowel and bladder limitations persisted or worsened.
- Delayed walking failure with preserved recumbent cycling is physiologically coherent because posture and venous return materially alter circulatory load.
- Predictability improves safety planning but does not prove recovery; the August 2026 pattern of falls and near-falls warrants objective symptom-linked assessment.
- The most useful clinical question is not simply whether the autonomic nervous system is better or worse, but which reserve component fails, during which task, after how long, and with what measurable haemodynamic signature.

Declarations and Scope

Patient authorship and consent: Bruce H. Knox is both the author and the person described in this first-person case study. Source base: prior case manuscripts, home haemodynamic records as previously reported, and Supplementary Appendix A: Medical and Physiological Foundation. Epistemic scope: this is a narrative case study, not a diagnostic opinion, validated prognostic model or treatment prescription. Home observations should be interpreted alongside source records and, where clinically relevant, confirmed through standardised testing before clinical reliance. Use of artificial intelligence: generative AI assisted with synthesis, drafting, evidence organisation and document production. The author retains responsibility for verifying the clinical record, interpretation and references, and for any subsequent use.

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Supplementary Appendix A

Medical and Physiological Foundation for The Measure of What Remains

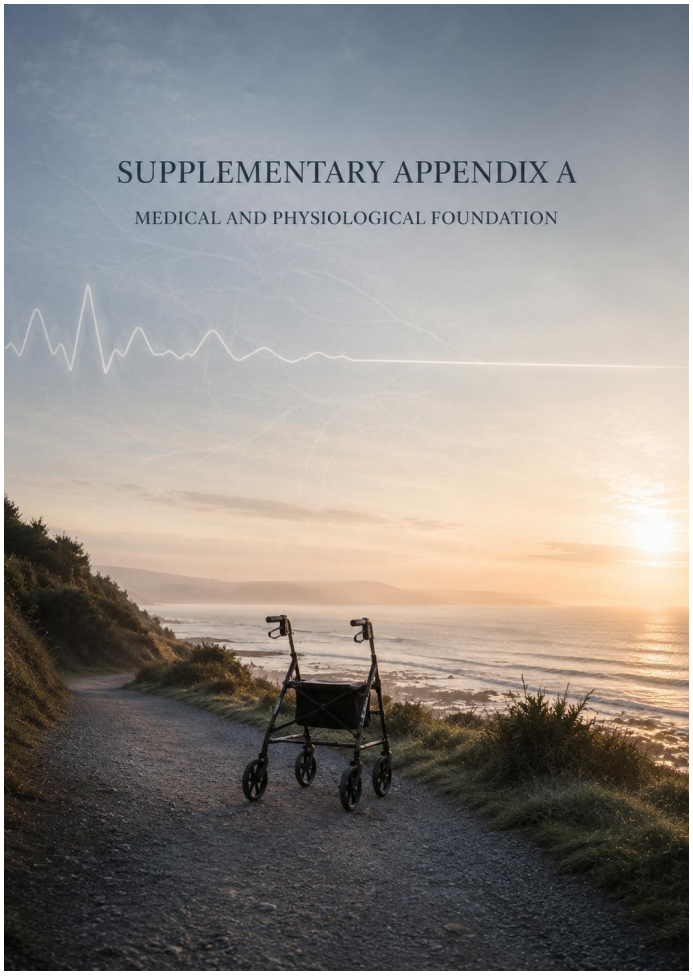
An evidence-informed framework supporting the personal case narrative of autonomic reserve

Purpose. This appendix provides the medical and physiological foundation for The Measure of What Remains. It explains why the reported pattern is physiologically coherent, identifies the literature supporting each major interpretation, and marks the boundary between documented observation, physiological plausibility and unproven mechanism.

Evidence-Informed Supplementary Medical Review

Companion appendix for author and clinical review; not a diagnosis or individualised treatment plan

Scope: This is a narrative physiological appendix, not a systematic review, an independent verification of the case record, a diagnostic opinion or a treatment prescription. "Autonomic reserve" is used as an integrative explanatory construct rather than as a recognised standalone diagnosis or validated global score. Evidence derived from primary autonomic failure cannot be transferred automatically to a secondary, mixed or recovering disorder.



Executive Summary

Autonomic nervous system reserve is a clinically useful way of describing the additional regulatory capacity that remains after resting needs have been met. The term is used in the physiological literature in related forms—such as sympathetic reserve, baroreflex reserve, chronotropic reserve and vasoconstrictor reserve—but it is not a single, universally standardised diagnosis, biomarker or quantity [8, 21-24]. It is therefore more accurate to speak of an integrated reserve made up of several partly independent components.

- At rest, only part of the available control capacity is usually required. Standing, walking, exercise, meals, heat, dehydration, illness and emotional stress increase the demand.
- The system does not normally "run out" like a battery. Rather, one or more compensatory responses reach a ceiling, respond too slowly, cannot be sustained, or are opposed by a stronger vasodilating or volume-lowering force.
- When total demand exceeds available compensation, arterial pressure and organ perfusion become unstable. The brain is especially sensitive; symptoms may include light-headedness,

dimmed or blurred vision, cognitive slowing, weakness, impaired gait control, presyncope or syncope [2,12].

- Delayed symptom onset is physiologically plausible: initial compensation may be adequate, but the reserve margin can progressively narrow as venous pooling, exercise vasodilatation, heat loss, sweating, relative hypovolaemia or medication effects accumulate.
- A repeatable time-to-symptom threshold may make risk more predictable, but predictability alone does not prove that autonomic function is improving or deteriorating. Longitudinal physiological measurements are needed.
- Improvement can be uneven. Heart-rate responsiveness, gastrointestinal function or immediate standing tolerance may improve while sustained vasoconstrictor or blood-volume reserve remains limited.

Key Conclusion. "Reserve exhaustion" means that the integrated response has become insufficient for the current load. It does not, by itself, identify the cause, prove permanent neuronal damage, or establish a degenerative autonomic disorder.

How this Appendix Supports the Personal Story

The companion narrative is the primary account of what happened and how function changed. This appendix asks a narrower medical question: Are the narrative's interpretations consistent with established autonomic and cardiovascular physiology, and where does the evidence stop? The answer is that the central pattern is physiologically coherent and clinically important, but several proposed mechanisms remain hypotheses requiring symptom-linked testing.

Three evidentiary layers are used throughout:

- Observed case data: dated procedures, reported diagnoses, home blood-pressure and heart-rate readings, symptoms, falls, functional thresholds and recovery patterns supplied by the author.
- Literature-supported interpretation: established physiology or published findings that make an observation medically plausible or clinically recognisable.
- Unconfirmed mechanism: an explanation that fits the pattern but has not been demonstrated in this individual by continuous haemodynamic, autonomic, cardiac, cerebral or neurological testing.

Case Observations Requiring Explanation

- Orthostatic symptoms followed chikungunya infection in 2008; the temporal association is clear, but chronic causation is not established [15].
- A major cardiac procedural complication in October 2021 involved tamponade, emergency sternotomy, cardiopulmonary bypass and ventricular repair; cardiac surgery can acutely disturb cardiovascular autonomic measures, but the literature cannot attribute this individual's long-term syndrome to one operative mechanism [16,17].
- Supine or seated hypertension has coexisted with large postural pressure falls and a relatively limited heart-rate response, a pattern recognised in impaired autonomic blood-pressure buffering [1,2,5,13].
- During 2024-2025, autonomic storms and thermoregulatory problems improved and heart-rate responsiveness returned, while orthostatic, gastrointestinal, bowel, bladder and postprandial limitations remained uneven.
- In August 2026, walking produced a delayed threshold of head and gait symptoms, falls or near-falls and symptom-linked hypotension, whereas recumbent cycling remained better tolerated [4,6-8,12].
- Home readings included 85/45 mmHg with heart rate 85 after a symptomatic brisk walk and a post-exercise seated-to-standing reduction from 180/78 to 123/61 mmHg with only a six-beat-per-minute heart-rate rise. These are clinically concerning observations but not standardised laboratory measurements.
- Tamsulosin, losartan, spironolactone and intermittent furosemide are plausible threshold modifiers through vascular or volume effects, but the contribution of medication has not been isolated and must be weighed against severe supine hypertension [2,5,22,23].

1. What the Autonomic Nervous System does

The autonomic nervous system (ANS) continuously regulates functions that must adapt without conscious control: heart rate, cardiac contractility, vascular tone, blood-pressure buffering, breathing pattern, sweating and temperature control, gastrointestinal motility and secretion, bladder function, pupillary responses and aspects of metabolic and immune regulation. Its sympathetic and parasympathetic divisions are not simply an accelerator and a brake; they work with sensory receptors, brainstem and hypothalamic networks, hormones, the heart, blood vessels, kidneys, lungs and skeletal-muscle pumps to preserve internal stability. For cardiovascular control, arterial pressure is determined approximately by cardiac output multiplied by systemic vascular resistance. Cardiac output is heart rate multiplied by stroke volume. The ANS therefore has several ways to defend pressure: increase heart rate, increase myocardial contractility, constrict resistance vessels, constrict venous capacitance beds to return blood to the heart, and coordinate these responses with breathing, muscle activity and slower hormonal volume regulation [2,3,21].

2. What "Autonomic Reserve" means

A useful working definition is the difference between the regulatory capacity currently being used and the maximum effective, sustainable response available to meet an additional physiological challenge. Four properties matter:

- Magnitude — how much additional change can be produced, such as a rise in sympathetic vasoconstriction, heart rate or contractility.
- Speed — whether the response begins quickly enough to prevent a transient fall in pressure or perfusion.
- Sustainability — whether the response can continue for minutes or hours without fading or being overtaken by other demands.
- Coordination — whether the heart, arteries, veins,

blood volume, muscle pump, cerebral circulation and thermoregulation act together rather than working at cross-purposes.

Because these components can be impaired to different degrees, a person can have adequate reserve for one task and inadequate reserve for another. Resting blood pressure can be normal or high while upright reserve is poor. A normal brief standing test can coexist with delayed orthostatic hypotension. A reasonable heart-rate rise can coexist with inadequate vascular resistance, and preserved recumbent cycling can coexist with failure during upright walking [2,4,6-8].

3. The Components of Integrated Reserve

Reserve component	Normal contribution	What limitation can look like
Baroreceptor sensing and central integration	Detects changes in arterial stretch and coordinates rapid reflex responses.	Slow, small or poorly buffered responses; large pressure swings.
Sympathetic vasoconstrictor reserve	Constricts arteriolar and venous beds, especially within the splanchnic and dependent circulations.	Pooling, falling vascular resistance, narrow tolerance for standing, meals or exercise.
Cardiac reserve	Raises heart rate and contractility to preserve cardiac output.	Chronotropic limitation, low stroke volume or inadequate output during demand.
Volume and hormonal reserve	Maintains plasma volume and recruits renin-angiotensin-aldosterone and vasopressin responses.	Greater sensitivity to dehydration, diuresis, blood loss, anaemia or overnight natriuresis.
Mechanical reserve	Leg and abdominal muscle pumps return venous blood during movement.	Worse with immobility, weak calf pump, venous disease or unsupported standing.
Cerebral autoregulatory reserve	Buffers cerebral blood flow across a range of perfusion pressures.	Neurological symptoms at pressures tolerated by another person; this may vary by posture and illness.

Table 1: Autonomic Reserve is an Integrated Set of Capacities, not a Single Pool.

4. What Reserve does During Common Challenges

Standing

On standing, gravity rapidly shifts a substantial volume of blood towards the legs and splanchnic venous circulation. Venous return and stroke volume fall. Baroreceptor unloading should rapidly withdraw cardiac vagal influence, increase sympathetic output, accelerate the heart modestly, constrict arterioles and veins, and support venous return. The skeletal-muscle pump and respiratory pump assist. These mechanisms are normally so effective that upright pressure is maintained despite the gravitational challenge [2,3,5,21].

Walking and Exercise

Exercise adds a competing requirement: active muscle must dilate its vessels to receive oxygenated blood, while arterial pressure must be maintained. The baroreflex is not switched off; it is reset to operate around the higher pressure and flow demands of exercise. Heart rate, stroke volume and cardiac output should rise, while sympathetic restraint limits excessive vasodilatation in inactive, splanchnic and cutaneous beds [8,21]. In autonomic failure, exercise-induced vasodilatation may not be matched by adequate vasoconstriction or cardiac output. Studies of sympathetic denervation show that blood pressure can fall during exercise and remain low afterward because vascular resistance falls excessively, splanchnic constriction is delayed or inadequate, and leg hyperaemia persists [6,7]. Chronotropic incompetence—the inability to raise heart rate in proportion to demand—can further limit cardiac output and exercise tolerance [8].

Meals and Heat

A meal increases blood flow to the gastrointestinal circulation. Healthy compensation maintains systemic vascular resistance and cardiac output; people with dysautonomia and postprandial hypotension may fail to do so [10]. Heat dilates skin vessels, shifts blood away from the central circulation and may add sweating and volume loss. Heat stress therefore reduces orthostatic tolerance even in healthy people and can be particularly important when reserve is already limited [9].

5. What Happens when Reserve Becomes Insufficient

The phrase “runs out” is shorthand for a threshold event. It may be represented conceptually—not as a validated clinical equation—as:

Reserve margin at time t = available integrated compensatory capacity at time t minus total haemodynamic demand at time t.

While the margin remains positive, pressure and perfusion can be maintained, although the body may already be using a high level of sympathetic drive. As the margin approaches zero, blood pressure may become more variable and symptoms can appear with small additional loads. When demand exceeds capacity, one or more of the following occurs:

- Systemic vascular resistance is too low because arteriolar or venous constriction is inadequate.
- Venous return and stroke volume fall because of pooling or low circulating volume.
- Heart rate or contractility does not rise enough to preserve cardiac output.

- Cerebral perfusion falls below the individual's autoregulatory or symptom threshold.12,22
- Competing regional demands—for exercising muscle, skin cooling or digestion—overwhelm the remaining circulatory margin [6,9,10].

The resulting manifestations may include light-headedness or head pressure, blurred or grey vision, reduced concentration, fatigue, neck or shoulder discomfort, weakness, altered proprioceptive confidence, gait deterioration, presyncope, falls or syncope. Symptoms reflect impaired perfusion or instability but are not specific to autonomic disease; cardiac rhythm disorders, structural heart disease, vestibular disease, neurological disease, metabolic disturbance and medication effects must also be considered [2,5].

6. Why Reserve can "run out"

Reserve failure usually reflects an interaction between reduced capacity and increased load. The major mechanisms are best grouped by where they act.

A. Reduced Neural Capacity

- Afferent baroreceptor impairment: pressure changes are not sensed accurately or transmitted effectively.
- Central integration impairment: brainstem or central autonomic networks do not generate an appropriately scaled response.
- Efferent sympathetic failure: noradrenergic signals to arteries and veins are reduced.
- Peripheral autonomic neuropathy or impaired adrenergic responsiveness: signals arrive, but target vessels respond inadequately [2,3,5].

B. Reduced Cardiac Capacity

- Chronotropic limitation from sinus-node dysfunction, conduction disease, cardiac disease, autonomic impairment or rate-limiting medication.
- Limited stroke-volume or contractile reserve from impaired filling, ventricular dysfunction, valve disease, ischaemia or a stiff cardiovascular system.
- Arrhythmia or pacing constraints that prevent the required rise in effective cardiac output [8].

C. Reduced Vascular or Volume Capacity

- Low plasma volume from dehydration, reduced intake, diarrhoea, vomiting, sweating, fever or diuretic therapy.
- Anaemia or blood loss, which reduces oxygen delivery even if pressure is partly maintained.
- Venous pooling, impaired venous constriction, varicosities, a weak abdominal or calf-muscle pump or prolonged deconditioning.
- Age-related vascular stiffening and lower cardiovagal baroreflex sensitivity can reduce the ability to buffer rapid changes [11].

D. Increased Competing Demand

- Prolonged upright posture, especially standing still or walking after a period of sitting.
- Exercise vasodilatation, especially when workload, duration or active muscle mass increases [6,7].
- Warm environments, heavy clothing, fever or hot showers [9].
- Meals, especially large or carbohydrate-heavy meals in susceptible people [10].
- Pain, infection, sleep loss, travel, emotional stress or cumulative fatigue, which can alter vascular tone, hydration and autonomic demands.

E. Medication and Timing Effects

Diuretics, alpha-adrenergic blockers, nitrates, vasodilators, some antihypertensives, dopaminergic drugs, sedatives and other medicines can lower pressure, reduce volume, blunt heart-rate responses or worsen postural symptoms. Conversely, attempts to raise upright pressure may worsen supine hypertension. Medication timing, meal timing and overnight pressure natriuresis can therefore change the available reserve from hour to hour [2,5,22,23]. Medication changes require clinician review, particularly when both hypertension and hypotension occur.

7. Why Symptoms may be delayed rather than Immediate

Delayed symptoms do not mean gravity starts late. Gravity acts immediately. The delay means that compensation initially succeeds but cannot be sustained indefinitely, or that additional loads accumulate over time. A plausible sequence during upright walking is:

- Initial upright shift: central blood volume and stroke volume fall, but available baroreflex, vascular and cardiac reserve stabilise pressure.
- Early walking: the calf-muscle pump helps venous return, while active muscle vasodilatation and higher metabolic demand increase the circulatory task.
- Progressive load: heat production, skin vasodilatation, sweating, ongoing dependent or splanchnic pooling, low starting volume, medication effects or an inadequate heart-rate response narrow the reserve margin.
- Threshold Crossing: vascular resistance and/or cardiac output can no longer maintain adequate pressure and cerebral perfusion. Symptoms may then worsen progressively until the person sits or lies down.

This framework is consistent with delayed orthostatic hypotension, defined as a sustained orthostatic pressure fall that occurs after the first three minutes of standing or tilt [1,4]. However, a delayed decline during walking is not automatically identical to delayed orthostatic hypotension. It may include exercise-induced hypotension, post-exercise hypotension, chronotropic limitation, impaired cerebral autoregulation or another cardiac or neurological cause. Testing must reproduce the provoking activity and record the relevant physiology.

8. Why Recumbent Cycling may remain possible

Recumbency shortens the hydrostatic column between the heart and brain and reduces gravitational pooling in the legs and abdomen. Venous return is generally better supported, and cycling activates a rhythmic leg-muscle pump. This can preserve stroke volume and

cerebral perfusion even when upright walking exceeds available reserve. The contrast is therefore physiologically meaningful, but it is not diagnostic by itself. Importantly, exercise itself still dilates active muscle vessels. In severe sympathetic failure, even supine cycling can lower pressure [6,7]. Preserved recumbent cycling therefore suggests that posture and venous return are important contributors; it does not prove that exercise vasodilatation or cardiac limitations are absent.

9. Reserve, Recovery and Progression

Autonomic recovery is rarely all-or-nothing. Different networks and effectors can recover at different rates. A person may show better heart-rate responsiveness, temperature control, gastrointestinal function or immediate postural adaptation yet continue to have limited sustained vasoconstrictor reserve. Conversely, worsening hydration, medication burden, anaemia, cardiac filling or physical conditioning can reduce the functional margin even if neural recovery is continuing.

A more predictable symptom onset is clinically valuable because it improves pacing and fall prevention. Nevertheless, it is not, by itself, evidence of physiological improvement. It could reflect a stable threshold, better symptom recognition, a more reproducible activity load, or a narrower but consistent reserve margin. Evidence of improvement would ideally include longer symptom-free upright time, a smaller pressure fall, better cardiac-output or vascular-resistance responses, improved cerebral blood-flow preservation, fewer near-falls, and reduced day-to-day variability under comparable conditions. Delayed orthostatic hypotension has been described in some cohorts as an early or milder manifestation of autonomic failure and may progress over time.4 That finding is important but must not be overgeneralised: it was derived from selected referral populations and does not establish that every delayed or activity-dependent episode is neurodegenerative. Secondary, medication-related, volume-related, cardiac and mixed mechanisms remain possible [2,5].

10. Why High Supine or Seated Blood Pressure does not disprove Low Upright Reserve

Autonomic failure can produce both hypertension and hypotension because the core defect may be impaired buffering rather than a uniformly low sympathetic state. When baroreflex control is poor, pressure can overshoot while supine and collapse when upright. Supine hypertension can also promote pressure natriuresis overnight, lowering morning circulating volume and worsening orthostatic tolerance. Thus, a high reading in one posture is not evidence that adequate reserve will be available during prolonged upright activity [5,22,23]. An arm-cuff reading obtained after the person stops walking may also miss the pressure nadir. Sitting, standing still, muscle contraction, anxiety, pain and recovery can rapidly change pressure. Intermittent cuff pressure is therefore less informative than beat-to-beat pressure recorded during the actual provoking task when the clinical question concerns a delayed, dynamic event.

11. How reserve can be Investigated Clinically

There is no single "autonomic reserve test." Evaluation combines a reproducible challenge with measurements that identify which component fails and when.

- Standardised supine-to-standing blood-pressure and heart-rate measurements at baseline, 1 minute and 3 minutes, extended to 5, 10, 15 or more minutes when symptoms are delayed [1,2].
- Beat-to-beat blood pressure during active standing, tilt, treadmill walking or a carefully supervised corridor-walk protocol that reproduces the actual symptoms.
- Heart-rate response interpreted alongside the blood-pressure fall and medication effects; cardiopulmonary exercise testing when chronotropic incompetence or limited cardiac output is suspected [8].
- Valsalva manoeuvre, heart-rate response to deep breathing, sudomotor testing and plasma norepinephrine in selected autonomic laboratories.
- Ambulatory blood-pressure monitoring and a structured diary linking posture, walking duration, meals, temperature, hydration, symptoms and medication timing.
- ECG/rhythm monitoring, echocardiography and relevant laboratory assessment, including full blood count, electrolytes, renal function and other tests guided by the clinical context.

Transcranial Doppler or other cerebral haemodynamic assessment in specialised settings when symptoms and arm blood pressure do not align [12].

Interpretation principle. The most useful measurement is not simply the lowest blood pressure. It is the sequence linking workload, posture, heart rate, pressure, symptoms, recovery and medication timing.

12. Application to the Delayed Upright Walking Pattern in the Companion Narrative

The companion narrative describes the ability to stand, begin walking and undertake some ordinary tasks normally, followed after approximately 10-20 minutes by light-headedness or head pressure, visual disturbance, loss of automatic gait rhythm, veering and falls or near-falls. Sitting, lying down or using support helps, while recumbent cycling is substantially better tolerated. That posture-task dissociation is consistent with an initially compensated but unsustainable haemodynamic response. The reported 85/45 mmHg reading during a delayed symptomatic event and the post-exercise seated-to-standing fall of 57/17 mmHg provide objective support for clinically significant hypotensive and orthostatic susceptibility. They do not reconstruct pressure during the whole walk, establish neurogenic causation, or show that every gait event was caused by cerebral hypoperfusion. Cuff timing, arm position, movement and the absence of matched baselines limit precision.

Clinical meaning of the "reverse" pattern. Walking can fail while recumbent cycling persists because upright walking adds a longer hydrostatic column, greater dependent and splanchnic pooling, balance demand and a sustained competition between active-

muscle vasodilatation and pressure maintenance. Recumbency reduces several of those demands and supports venous return. This makes posture a material part of the mechanism, but it does not exclude cardiac, vestibular, neurological, musculoskeletal or medication-related contributors.

13. Claim-to-Evidence Concordance

Narrative claim	Medical and physiological support	Boundary
The observed phenotype is clinically significant, severe and fluctuating.	Repeated postural pressure loss, absolute hypotension, supine/seated hypertension, falls and multisystem functional burden form a clinically significant phenotype [1,2,5,13].	Severity is supported; one cause or lesion is not established.
High resting pressure can coexist with low upright reserve.	Impaired buffering can permit supine hypertension while upright pressure falls; nocturnal pressure natriuresis may further reduce daytime volume [5,13,22,23].	Formal neurogenic classification requires standardised testing.
Walking may fail only after a delay.	Compensation may initially maintain pressure, then be overtaken by pooling, exercise vasodilatation, heat, volume loss or limited cardiac response [2,4,6-9,24].	Walking-related decline is compatible with, but not identical to, delayed orthostatic hypotension.
Recumbent cycling may remain possible.	Recumbency reduces the hydrostatic burden and pooling and supports venous return while rhythmic leg movement preserves a muscle pump [6-8].	This localises posture as important; it does not prove the precise lesion.
Head and gait symptoms can emerge near the threshold.	Falling pressure or cerebral-flow instability can produce visual, cognitive, presyncopal and motor-control symptoms; TCD can assess cerebral haemodynamics [2,12].	Symptoms are not specific and require cardiac, vestibular and neurological alternatives to be assessed.
Recovery can be genuine but uneven.	Autonomic control is distributed across distinct reflex, cardiac, vascular, sudomotor, enteric and pelvic pathways; functional domains need not change together.	Asynchronous change does not itself prove neural repair or rule out progression.
Predictability does not necessarily mean improvement.	A stable warning interval may reflect residual compensation, improved recognition, a reproducible load or a narrowed but consistent threshold [4].	Improvement requires comparative outcomes, not time-to-symptom alone.
Chikungunya and the 2021 cardiac event are plausible contributors.	Chikungunya has recognised neurological complications, and cardiac surgery can alter autonomic cardiovascular measures [15-17].	The sequence is biologically plausible but does not prove a single cumulative causal chain.
Gastrointestinal, bowel, bladder, fatigue and cognitive problems add to the burden.	The ANS participates in motility, elimination and cardiovascular support; orthostatic hypotension is associated with cognitive impairment in observational evidence [20].	Each organ diagnosis may have distinct structural, inflammatory, metabolic, medication or age-related causes.
The phenotype does not establish a degenerative autonomic disease.	Secondary, medication-related, volume-related, cardiac, post-infectious and mixed explanations remain viable [2,4,5].	Longitudinal examination and objective autonomic testing determine classification and prognosis.

Table 2: Medical support and evidentiary boundaries for the principal claims in the personal story

14. Interpretation of the Longitudinal Course

The most defensible longitudinal interpretation within this narrative is a secondary, multifactorial autonomic phenotype with changing reserve, rather than a proven single-lesion or primary degenerative disorder. Chikungunya may have been an initiating contributor; the years of orthostatic and cardiac burden may have reduced resilience; and the 2021 catastrophic procedural course may have amplified an existing vulnerability. This is a coherent three-stage interpretation, not a demonstrated chain of causation [15-17]. The later coexistence of improvement and persistent limitation is also medically credible. The cessation of storms, return of sweating and improved heart-rate responsiveness are valid recovery markers in their respective domains. Persistent orthostatic instability, delayed walking failure, bowel and bladder dysfunction and postprandial depletion show that recovery was not global. No single feature, including a predictable warning interval, is sufficient to define prognosis. The appendix therefore supports the personal story's central judgement: meaningful recovery can coexist with a

dangerously narrow reserve for sustained upright walking. It does not support claims of proven permanent baroreflex damage, universal cerebral hypoperfusion, a validated global reserve score or inevitable neurodegeneration.

15. Safety and Clinical Implications

The purpose of recognising a reserve threshold is to prevent injury, not to test how far beyond it the body can be pushed. When symptoms include visual greying, loss of gait rhythm, veering, presyncope or previous falls, the person should stop promptly and sit or lie down rather than attempting to complete the walk. A rollator or other support may reduce fall exposure, but it does not correct the underlying haemodynamic or non-haemodynamic cause of the event. Fluid, salt, compression, meal modification, exercise design and medication changes can be helpful in appropriate patients, but require individual review when there is supine hypertension, heart disease, kidney disease, urinary problems or diuretic/antihypertensive treatment. Evidence-based reviews support non-pharmacological measures as foundational and emphasise individualised treatment goals centred on symptoms and function rather than a conventional seated blood-pressure target [5,22,23]. Urgent medical assessment is warranted for syncope with injury, new focal neurological deficit, chest pain, severe breathlessness, sustained palpitations, a new severe headache, black or bloody stools, or a prolonged episode that does not improve with recumbency.

16. Conclusion

Autonomic reserve is best understood as the available, integrated capacity to maintain circulation and organ perfusion when physiological demand rises. It is built from sensing, central integration, sympathetic vascular control, heart-rate and contractile responses, circulating volume, mechanical venous return and cerebral autoregulation. It "runs out" when the combined demand of posture, exercise, heat, meals, medication effects or low volume exceeds the effective and sustainable response available at that moment. The resulting threshold can be immediate or delayed, stable or variable, and task-specific. A delayed threshold does not mean gravity is delayed, and a predictable threshold does not by itself prove recovery. The clinically useful question is not simply "Is the autonomic nervous system better or worse?" but "Which reserve component is limiting, under what load, after how long, and with what objective haemodynamic signature?"

Scope, Limitations and Authorship

This appendix is an evidence-informed narrative review prepared to accompany a first-person case study. It does not independently authenticate the source measurements, provide a diagnosis, replace specialist assessment or prescribe treatment. Home blood-pressure readings are clinically useful signals but remain sensitive to posture, cuff position, movement, timing and protocol. The claim-to-evidence table states concordance, not proof. Bruce H. Knox is the person described and retains responsibility for verification and subsequent use. Generative AI was used to assist with synthesis, editing, evidence organization and document production.