

When Reflexes Lose Their Timing: Baroreflex Dysfunction, Orthostatic Hypotension, and the Failure of Bowel Coordination — A Lived Experience of Autonomic Injury and Recovery

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Abstract

The baroreflex is a central autonomic control system responsible for maintaining moment-to-moment blood pressure stability and preserving organ perfusion [1-3]. When disrupted, the resulting instability—particularly orthostatic hypotension—can affect multiple reflex-dependent systems, including gastrointestinal and bowel function [2,4-7]. In this patient-perspective paper, I describe the lived experience of progressive autonomic dysfunction culminating in bowel reflex failure, drawing both on clinical understanding and on my own longitudinal experience of illness and recovery [8,9]. Through clinical discussions and reflective analysis, I came to understand this illness as a cumulative autonomic injury in which baroreflex dysfunction and recurrent orthostatic hypotension interacted to destabilise neural signalling, compromise perfusion, and impede recovery [1-6,8,9]. This paper argues that orthostatic hypotension is not merely a symptom but a central mechanism of ongoing injury, repeatedly compromising perfusion at moments of physiological demand [2,4-6]. Recovery is framed as a gradual process of neural recalibration dependent on sustained hemodynamic stability [1,2,5,8,9].

Keywords: Baroreflex Failure, Orthostatic Hypotension, Bowel Dysfunction, Dysautonomia, Autonomic Instability, Patient Experience

1. Introduction: The First Signs Were Positional

One of the earliest recognisable features of my illness was that the symptoms were not random—they were positional [8,9]. Standing changed everything [8,9]. What had once been automatic became effortful, with a delay between postural change and physiological response [8,9]. At times the body compensated too late; at other times it did not compensate adequately at all [8,9].

Clinically, this pattern is consistent with **orthostatic hypotension**, defined as a sustained reduction in systolic blood pressure of at least 20 mm Hg or diastolic blood pressure of at least 10 mm Hg within 3 minutes of standing or head-up tilt [4]. Orthostatic hypotension reflects failure of the normal compensatory responses that preserve perfusion during gravitational stress, responses in which the baroreflex plays a central role [1-4].

At the time, I experienced this simply as dizziness, weakness,

delayed clarity, and internal instability [8,9]. In retrospect, these were not isolated symptoms but early manifestations of impaired baroreflex-mediated circulatory control [1-4,8,9].

1.1. The Three-Hit Autonomic Framework

To make sense of how this level of autonomic dysfunction and blood pressure instability emerged and persisted, I came—through clinical discussions and longitudinal reflection—to understand my illness as a cumulative “three-hit” process affecting the baroreflex and wider autonomic system. The first “hit” appears to have been a systemic viral illness, specifically Chikungunya virus infection, which is recognised to have the potential to disrupt autonomic balance and alter baroreflex sensitivity, creating a state of physiological vulnerability rather than permanent structural injury [1,3,10]. The second “hit” occurred during a catheter ablation procedure undertaken to address rhythm disturbances that followed this illness, which was complicated by cardiac tamponade.

This event introduced acute hemodynamic compromise, during which reduced perfusion and mechanical stress may have disrupted afferent autonomic signalling pathways central to baroreflex function, leading to impaired timing and coordination rather than complete system failure [1,2,5]. The third “hit” was the subsequent emergency open-heart surgery required to stabilise the tamponade and preserve life. While lifesaving, this phase likely imposed further physiological stress, including periods of altered perfusion, inflammatory activation, and autonomic disruption, compounding the existing vulnerability of the system [1-3,5].

What followed was not recovery in a stable environment, but a prolonged period marked by recurrent orthostatic hypotension—repeated episodes of low perfusion occurring during everyday postural change—which may have continually interrupted neural recovery and reinforced dysfunction over time [4,5]. Within this framework, the persistence and longevity of symptoms can be understood not as the result of a single irreversible injury, but as a dynamic process in which initial autonomic vulnerability was compounded by sequential physiological insults and then sustained by ongoing hemodynamic instability. This interpretation, while grounded in lived experience, is consistent with current understanding of baroreflex dysfunction, orthostatic hypotension, and the dependence of autonomic recovery on stable perfusion [1-5].

2. The Baroreflex and the Orthostatic Challenge

The arterial baroreflex is one of the body’s principal short-term blood pressure control systems [1-3]. Baroreceptors in the carotid sinus and aortic arch detect arterial stretch and send afferent input via the glossopharyngeal and vagus nerves to the brainstem, especially the nucleus tractus solitarius, where autonomic responses are integrated [1-3]. Through coordinated sympathetic and parasympathetic output, the reflex helps stabilise blood pressure, heart rate, and effective perfusion during posture change, exertion, and daily physiological fluctuations [1-3].

Standing presents a normal circulatory challenge because venous pooling in the lower body reduces venous return and can lower arterial pressure unless compensated rapidly [2,4,5]. When the baroreflex does not respond effectively, orthostatic hypotension results, and repeated episodes of low-pressure perfusion can affect not only the brain but also peripheral autonomic pathways and other organ systems [2,4-6].

3. The Lived Experience of Orthostatic Hypotension

Orthostatic hypotension was not a single symptom for me but a repeated event that shaped daily life [8,9]. Every time I stood, I risked lightheadedness, weakness, slowed cognition, and a sense that the body had not yet caught up with the movement I had just made [8,9]. These episodes often seemed temporary in the moment, but their repetition became one of the defining features of the illness [8,9].

The clinical literature recognises that neurogenic orthostatic hypotension commonly occurs as part of broader autonomic

dysregulation and is frequently accompanied by dysfunction in other organ systems, including bowel and bladder disturbance [5]. Earlier physiological work also linked autonomic failure with gastrointestinal motility disturbance in patients with neurogenic orthostatic hypotension [7]. In that light, what I experienced across cardiovascular and bowel systems was not a coincidence but a coherent autonomic pattern [5,7-9].

4. From Blood Pressure Instability to Bowel Dysfunction

The bowel symptoms did not begin as the centre of the illness. They emerged gradually and initially seemed disconnected from the circulatory symptoms [8,9]. Over time, however, the pattern became harder to dismiss: loss of natural urge, delayed bowel response, incomplete evacuation, and an increasing need for conscious management of what had once been automatic [8,9].

Bowel function depends on a distributed interaction between the enteric nervous system, extrinsic autonomic pathways, central integration, and coordinated motor output [6,11,12]. The enteric nervous system is capable of substantial local control, but normal bowel function still depends on broader gut-brain-autonomic coordination [6,11,12]. Disorders of autonomic or neurologic regulation can impair motility, rectal sensation, and evacuation, producing constipation, ineffective emptying, or broader neurogenic bowel dysfunction [6,7,10-12].

This helped me reinterpret my experience. What I had first understood as a local bowel problem made more sense as a **downstream effect of impaired autonomic coordination and unstable perfusion** [5-12].

5. Why Orthostatic Hypotension Matters So Much

Orthostatic hypotension is not simply an unpleasant symptom. It is a recurrent physiological event in which perfusion pressure falls at the precise moment the body is required to adapt to gravitational stress [2,4,5]. In neurogenic disorders, these episodes may recur many times each day and can become cumulative in effect [5].

That cumulative aspect mattered in my experience [8,9]. Each episode was brief, but together they appeared to create a pattern of repeated physiological stress [8,9]. This interpretation is consistent with the broader understanding that autonomic disorders are often characterised not only by overt crises but by repeated failures of regulation across ordinary activities of daily life [1,2,5].

Where bowel function is concerned, such repeated hypotensive events are relevant because gastrointestinal function is closely linked to neural regulation and coordinated motility [6,11,12]. Repeated compromise of perfusion and autonomic control provides a plausible framework for why bowel signalling and timing can degrade over time in a patient with persistent orthostatic instability [5-7,10-12].

6. The Baroreflex–Bowel Connection

Through clinical discussion and reflection, I came to understand that the baroreflex is not merely a blood pressure reflex; it is part

of the body's wider infrastructure for maintaining conditions under which other reflex systems can function [1-3,8,9]. Bowel coordination depends on intact afferent sensation, central processing, autonomic modulation, and motor output [6,11,12]. It also depends on adequate perfusion to the neural tissues and muscular structures involved [6,11,12].

Autonomic dysfunction is increasingly recognised as relevant to gastrointestinal symptoms, and gastrointestinal dysmotility is well described in neurologic disease and autonomic failure [5,6,10,12]. Reviews of gastrointestinal autonomic dysfunction describe the gut as deeply integrated with both autonomic and central regulation rather than functioning as an isolated organ system [6,11,12].

This was consistent with my lived experience: the bowel did not seem to fail on its own. It failed within a body that had lost reliable autonomic timing [8,9].

7. A Three-Phase Understanding of How This Happened

Looking back, the illness makes most sense to me as unfolding in three overlapping phases [8,9].

Phase 1: Initial Physiological Stress

The first phase was characterised by a systemic stressor, likely inflammatory or otherwise physiologically destabilising, which appeared to alter autonomic stability and leave the system vulnerable [8,9]. Viral and inflammatory illnesses are increasingly recognised as possible precipitants or amplifiers of dysautonomia in susceptible patients [10].

Phase 2: Hemodynamic Instability

The second phase involved more obvious circulatory instability, including sustained or repeated hypotensive episodes and impaired postural tolerance [8,9]. Baroreflex dysfunction and orthostatic hypotension are both associated with impaired buffering of blood pressure change and instability of autonomic output [1-5].

Phase 3: Recurrent Orthostatic Hypotension and Interrupted Recovery

The third phase was dominated by repetition. Each orthostatic episode may have been individually transient, but the recurrence of low-perfusion states appeared to interrupt recovery and reinforce dysfunction [8,9]. In a condition where autonomic control is already impaired, repeated orthostatic hypotension is a reasonable explanatory mechanism for why improvement may be slow, fragile, and easily reversed [2,5,8,9].

8. Mechanisms by Which Bowel Function Was Affected

The bowel effects in this illness can be described through several interlocking mechanisms supported by the neurogastroenterology and autonomic literature [5-10].

First, sensory signalling may become unreliable. Neurogenic bowel dysfunction can involve altered rectal sensation, impaired

awareness of the need to evacuate, and disrupted reflex triggering [10,11]. That matches the lived experience of losing a reliable urge signal [8,9].

Second, central and autonomic coordination may degrade. Gastrointestinal motility disorders in neurologic disease commonly reflect disrupted extrinsic neural regulation, autonomic impairment, or broader gut-brain axis dysfunction [6,10,12].

Third, motor output may become poorly timed. Neurogenic bowel syndromes are characterised not only by slowed transit but also by impaired evacuation and discoordination of the anorectal response [10-12].

Fourth, repetition matters. Successful bodily reflexes are reinforced by successful execution; repeated failed or incomplete cycles may contribute to persistence of dysfunction, especially where autonomic control is already compromised [5,8-12].

9. Why This Persisted Despite Reassuring Tests

One of the most difficult aspects of the illness was that structural investigations were often reassuring while function remained clearly abnormal [8,9]. This mismatch produced uncertainty and, at times, self-doubt [8,9].

That pattern is not unusual in autonomic and neurogastroenterological disorders. Reviews of baroreflex dysfunction, orthostatic hypotension, and gastrointestinal autonomic dysfunction all describe syndromes in which the abnormality lies in regulation, signalling, and coordination rather than obvious structural destruction [1-3,5,6,9]. In such disorders, patients may have severe symptoms with limited structural findings because the pathology is functional, distributed, or dynamic [1-3,5,6,9].

This literature helped explain why the illness could be real, disabling, and physiologically coherent even when standard structural testing did not fully capture it [1-3,5,6,8,9,12].

10. The Current Situation

The current situation is not one of total recovery, but neither is it one of complete failure [8,9]. There has been gradual improvement in orthostatic tolerance, some reduction in blood pressure variability, and greater predictability in bowel function than during the worst phases of illness [8,9]. The system remains vulnerable, especially during physiological stress or illness, but the overall direction has been forward rather than backward [8,9].

That pattern is consistent with the clinical reality that disorders of autonomic regulation may improve slowly and incompletely, especially when the goal becomes stabilisation rather than cure [1,2,5]. In practical terms, maintaining hemodynamic stability, reducing the orthostatic burden, and working with rather than against bowel timing have become central parts of ongoing management [5,8-12].

11. What Assisted Recovery

What helped was not a single intervention but a change in framework [8,9]. Management became less about chasing isolated symptoms and more about protecting the conditions under which recovery might occur [8,9].

This included attention to volume status, blood pressure support, avoidance of unnecessary orthostatic stress, and regularity in bowel routine [4,5,8-12]. In neurogenic bowel management, conservative structured approaches—timing, positioning, facilitation, and consistency—are widely recognised as core strategies [10,11]. In autonomic disorders, reducing orthostatic stress and supporting hemodynamic stability are equally central [4,5].

Within my own experience, these were not simply convenience measures. They appeared to reduce instability and create a more favourable environment for reflex function to re-emerge [8,9].

12. Conclusion

This illness is best understood as a disorder of autonomic timing and coordination, centred on baroreflex dysfunction and amplified by recurrent orthostatic hypotension [1-5]. The bowel consequences were not isolated; they were part of a wider pattern of impaired autonomic regulation and downstream reflex failure [5-7,10-12].

The lived experience recorded in the attached source documents supports a three-part account of what happened: initial autonomic destabilisation, subsequent hemodynamic injury, and then prolonged interruption of recovery through repeated orthostatic stress [8,9]. The literature supports the plausibility of that model, particularly the links among baroreflex dysfunction, orthostatic hypotension, multisystem autonomic disturbance, and gastrointestinal dysregulation [1-7,10-12]. The most important conclusion remains this: stability is not the reward of recovery; it is the prerequisite for recovery [1,2,5,8,9].

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