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Research Article

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Cumulative Autonomic Injury and Secondary Multifactorial Dysautonomia: A Lived Case Integrated with a Central Autonomic Regulatory Model

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Abstract

Background: *Dysautonomia arising from cumulative physiological insult remains poorly characterised, particularly when involving both chronic post-viral injury and acute cardiovascular trauma.*

Objective: *To integrate a lived patient narrative with a physiologically grounded model of central autonomic dysregulation and long-term recovery.*

Methods: *A longitudinal first-person clinical narrative is combined with a structured three-layer autonomic model and supported by current literature in autonomic neuroscience and cardiovascular physiology.*

Results: *The clinical presentation is best explained by central autonomic dysregulation with impaired gain control rather than structural autonomic failure. A three-hit framework—post-viral priming, cardiac tamponade, and surgical insult—accounts for both collapse and delayed recovery.*

Conclusion: *This case demonstrates that complex dysautonomia may represent regulatory instability following cumulative injury. Recovery occurs through delayed neuroplastic adaptation, resulting in stabilisation with persistent limitations rather than full restoration.*

Keywords: Dysautonomia, Autonomic Nervous System, Orthostatic Hypotension, Fatigue, Neuroplasticity, Cardiac Surgery, Patient Narrative, Autonomic Recovery

Musical Narrative

Please follow the link to find a series of limericks set to music and performed, which capture the totality of this story.
https://heyzine.com/flip-book/67560a117c.html

1. Introduction

Dysautonomia is increasingly recognised as a disorder of **regulation rather than destruction,** particularly when arising in the context of cumulative physiological stressors [1,2]. This paper presents a combined **clinical narrative and physiological model** describing the development, collapse, and partial recovery of autonomic function over more than a decade.

The illness trajectory began following Chikungunya virus infection in 2008, a pathogen associated with recognised neurological and post-infectious sequelae [3,4]. Over time, this evolved into a compensated but unstable autonomic state.

The addition of **cardiac tamponade and open-heart surgery in 2021** precipitated a transition from compensated dysfunction to **multisystem autonomic collapse,** reflecting compounding injury rather than isolated pathology.

This paper proposes that the condition is best understood as:
Secondary multifactorial dysautonomia driven by central autonomic regulatory instability

2. Lived Experience: When the System Stops Agreeing with Itself

There was no single moment when everything failed. Instead, the body adapted for years—quietly compensating for dysfunction that remained largely unrecognised.

Following the initial viral insult, the dominant experience was not collapse, but inconsistency. This aligns with recognised post-infectious autonomic dysfunction, where symptoms may reflect impaired regulation rather than overt failure [1,2].

For years, the system held.

Until it could not.

The events of 2021 did not introduce a new disease—they exposed a vulnerable system. Cardiac tamponade and surgical intervention are both known to disrupt autonomic balance, including measurable reductions in autonomic variability and regulatory precision [4,5].

What followed was not simply illness, but **physiological unpredictability**.

Blood pressure no longer corrected reliably. Orthostatic regulation failed. Supine hypertension emerged alongside hypotensive episodes—patterns consistent with impaired baroreflex function and autonomic instability [3,6,7].

This was not organ failure.

It was **loss of coordination across systems.**

3. Clinical Symptom Profile

3.1 Cardiovascular and Autonomic Features

- Episodic supine hypertension
- Orthostatic hypotension
- Blunted heart rate response
- Oscillatory instability
- Reduced buffering capacity
- Delayed recovery after exertion

These findings align with impaired autonomic regulation and reduced baroreflex sensitivity [3,6].

3.2 Gastrointestinal Dysfunction

- Dysmotility during instability
- Functional collapse during autonomic stress

Autonomic dysfunction is well recognised in gastrointestinal motility disorders [8-10].

3.3 Motor System Involvement

- Sustained muscular guarding
- Reduced inhibitory control

Reflecting broader central autonomic network involvement [4].

3.4 Effort–Activation–Recovery Disturbance

- Rapid escalation with stress
- Delayed recovery
- Post-exertional crash

Consistent with impaired autonomic recovery mechanisms [11,12].

4. Fatigue as a Defining Feature

Fatigue in this context is not proportional to exertion and is not resolved by rest.

Autonomic disorders are associated with impaired recovery, including delayed parasympathetic reactivation and altered cardiovascular control following exertion [8,9]. The clinical expression of this is:

- Energy is accessible but not reliably recoverable
- Effort produces delayed physiological cost
- Rest stabilises but does not restore

This reflects impaired homeostatic regulation rather than simple energy depletion.

5. The Three-Hit Pathophysiological Framework

Hit 1: Viral Priming

- Post-infectious autonomic dysfunction [1,2]

Hit 2: Cardiac Tamponade

- Acute haemodynamic collapse [6]

Hit 3: Open-Heart Surgery

- Surgical autonomic disruption [5]

These are **synergistic,** not additive, producing sustained instability.

6. The Three-Layer Autonomic Model

Layer 1 – Baroreceptor Function

- Intact but reduced sensitivity
- Blunted physiological responses

Layer 2 – Central Autonomic Integration (Primary Dysfunction)

- Elevated sympathetic tone
- Reduced inhibitory modulation
- Impaired gain control

Likely involving:

- Brainstem nuclei
- Hypothalamic regulation

Layer 3 – Recovery Dysfunction

- Delayed parasympathetic reactivation
- Incomplete physiological reset

Result:

Oscillatory instability rather than fixed failure

7. Multisystem Autonomic Collapse

Following the 2021 events:

- Cardiovascular instability
- Gastrointestinal dysfunction
- Thermoregulatory failure
- Loss of exercise tolerance

Consistent with severe dysautonomia affecting multiple organ systems [4,6].

8. The Latency Phase (2021–2023)

This period appeared as non-recovery but is better understood as:

- Neural recalibration
- Receptor resetting
- Resolution of inflammatory processes

Autonomic recovery may occur over prolonged timeframes rather than acutely [5].

9. Onset of Recovery (2024–2025)

Recovery emerged gradually:

- Improved heart rate responsiveness
- Reduced BP instability
- Better functional tolerance

Reflecting partial reintegration of autonomic control [4–6].

10. Four-Year Outcome: Stabilisation with Residual Deficits

10.1 What Has Improved

- Reduced orthostatic collapse
- Stable cardiovascular function
- Controlled gastrointestinal symptoms
- Improved cognitive clarity

10.2 What Persists

Reduced Autonomic Reserve

- Limited tolerance to stressors
- Vulnerability to heat, illness, exertion

Persistent Fatigue

- Increased metabolic cost
- Delayed recovery
- Bladder Dysfunction
- Voiding inefficiency
- Autonomic–somatic dyssynergia

Bladder control represents a higher-order autonomic function and may recover incompletely [13].

11. Reframing the Condition

The illness was initially perceived as progressive autonomic failure.

It is now better understood as:

Cumulative autonomic injury with partial recovery

This distinction is critical:

- It changes prognosis
- It removes the expectation of degeneration
- It reframes management toward stability and adaptation

Secondary autonomic dysfunction is recognised across multiple systemic conditions [4,7].

12. Prognosis

Likely:

- Continued stability
- Incremental improvement
- Sustained independence

Unlikely:

- Neurodegenerative progression
- Escalating autonomic failure

Recovery is:

Maintained, not completed

13. Conclusion

This case demonstrates that:

- Dysautonomia may arise from cumulative injury across time
- The defining pathology is **regulatory instability,** not destruction
- Recovery may occur after prolonged latency

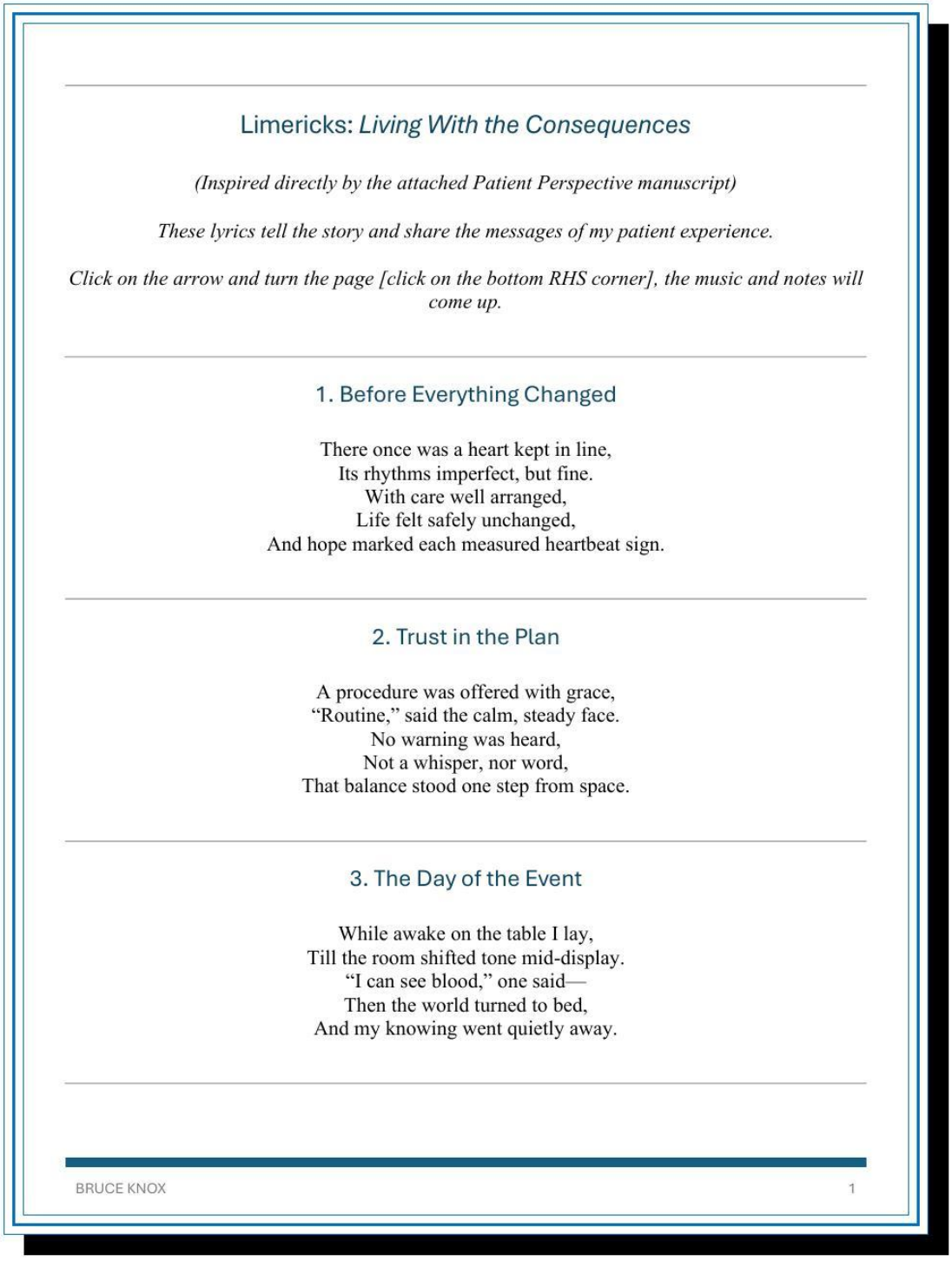
The autonomic nervous system in this case was not lost.

It was disrupted, overwhelmed, and partially restored.

What remains is not failure.

It is adaptation.

Musical Link



Limericks: *Living With the Consequences*

(Inspired directly by the attached Patient Perspective manuscript)

These lyrics tell the story and share the messages of my patient experience.

Click on the arrow and turn the page [click on the bottom RHS corner], the music and notes will come up.

1. Before Everything Changed

There once was a heart kept in line,
Its rhythms imperfect, but fine.
With care well arranged,
Life felt safely unchanged,
And hope marked each measured heartbeat sign.

2. Trust in the Plan

A procedure was offered with grace,
"Routine," said the calm, steady face.
No warning was heard,
Not a whisper, nor word,
That balance stood one step from space.

3. The Day of the Event

While awake on the table I lay,
Till the room shifted tone mid-display.
"I can see blood," one said—
Then the world turned to bed,
And my knowing went quietly away.

BRUCE KNOX 1

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