

Why Autonomic Dysfunction Is Commonly Assumed to Be Neurodegenerative: A Bias-Based and Evidence-Informed Analysis

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Abstract

Autonomic dysfunction is frequently interpreted within a neurodegenerative framework at the point of clinical recognition, particularly in cases of severe or multi-system involvement. This assumption persists despite well-established evidence that dysautonomia arises from a broad spectrum of reversible and non-degenerative causes, including immune-mediated, infectious, metabolic, and iatrogenic factors. This paper examines the structural, cognitive, and clinical drivers underlying the tendency to default toward neurodegenerative diagnoses such as pure autonomic failure and multiple system atrophy. Using a longitudinal case trajectory involving severe autonomic collapse followed by recovery and reclassification as secondary dysautonomia, this paper demonstrates that the neurodegenerative assumption is often a product of diagnostic heuristics, training bias, and limitations in current testing modalities. The findings highlight the need for a paradigm shift toward probabilistic, time-dependent diagnosis and emphasise the importance of maintaining diagnostic openness in autonomic medicine.

The following link is to a set of music that I have crafted, which communicates the joy of understanding the outcomes from this autonomic dysfunction pathway.

<https://heyzine.com/flip-book/0d032e9066.html>

1. Introduction

Autonomic dysfunction presents one of the most complex diagnostic challenges in modern medicine. When identified—particularly in its severe or generalised form—it is frequently interpreted as indicative of **neurodegenerative disease**, most notably within the spectrum of α -synucleinopathies.

However, this assumption is often made **before sufficient longitudinal or mechanistic evidence is available**.

This paper addresses a critical question:

Why does the identification of autonomic dysfunction so often lead to an assumption of neurodegeneration?

Drawing on clinical evidence, diagnostic theory, and a longitudinal case demonstrating reversal of severe dysautonomia, this paper argues that the assumption is not purely evidence-based, but is influenced by **cognitive bias, structural limitations in medicine, and historical framing of autonomic disorders**.

2. Clinical Context

2.1. Autonomic Dysfunction: A Non-Specific Clinical Signal

Autonomic dysfunction refers to impairment across domains including:

- Cardiovascular (e.g. orthostatic hypotension)
- Gastrointestinal (motility dysfunction)
- Thermoregulation
- Genitourinary function

Crucially, these features are non-specific and may arise from multiple aetiologies [1].

3. Neurodegenerative Framing of Dysautonomia

Historically, autonomic dysfunction has been strongly associated with:

- Pure autonomic failure (PAF)
- Multiple system atrophy (MSA)
- Parkinson's disease

These are grouped under α -synucleinopathies, characterised by progressive neurodegeneration [2].

Because of this association, **autonomic failure—particularly when severe—is often interpreted as evidence of underlying degeneration.**

4. The Diagnostic Assumption

4.1. The Clinical Leap

In practice, the following reasoning frequently occurs:

- Autonomic dysfunction is identified
- Severe or multi-system involvement is observed
- Neurodegenerative causes are considered most likely
- A working diagnosis such as PAF is assigned

This sequence represents a **logical but incomplete inference**, as it prioritises **pattern recognition over aetiological confirmation.**

5. Why This Assumption Occurs

5.1. Historical Anchoring in Neurodegenerative Models

The conceptual development of autonomic medicine has been heavily influenced by studies of neurodegenerative disease.

- Early landmark work focused on Parkinson’s disease and MSA
 - Autonomic failure became a recognised prodromal or core feature
- As a result, clinicians are trained to associate dysautonomia with degeneration.

This creates an anchoring bias, where initial interpretation is disproportionately influenced by familiar disease models [3].

5.2. Severity Bias: “The Worse It Is, The More It Must Be Degenerative”

Severe autonomic failure—particularly when involving multiple systems—appears clinically dramatic.

This leads to an implicit assumption:

Severe = irreversible = neurodegenerative

However, evidence contradicts this:

- Autoimmune autonomic ganglionopathy can produce profound, diffuse autonomic failure
- Acute autonomic neuropathies may present with near-total system shutdown
- Recovery, partial or substantial, is documented in multiple cases [4]

Thus, severity reflects **extent of dysfunction**, not necessarily **irreversibility.**

5.3. Lack of Immediate Alternative Explanations

At presentation, many secondary causes are:

- Difficult to detect
- Multifactorial
- Temporally remote (e.g. prior infection, procedural injury)

This creates a diagnostic vacuum.

In the absence of a clear alternative, clinicians default to:

- The most recognisable framework
- The most structured diagnostic category

Often, this is neurodegenerative disease.

5.4. Limitations of Diagnostic Testing

Autonomic testing provides:

- Quantification of dysfunction
- Distribution across systems

But does **not determine cause** [5].

Therefore:

- A patient with autoimmune dysautonomia
- A patient with PAF

may produce **similar test results**

This reinforces the illusion that the observed pattern reflects a single disease process.

5.5. Time Compression in Clinical Decision-Making

Diagnosis is often made under conditions of:

- Limited consultation time
 - Fragmented specialist input
 - Pressure to provide clarity
- However, autonomic disorders are inherently **time-dependent.**

- Neurodegenerative conditions progress
 - Secondary conditions may stabilise or improve
- Without longitudinal observation, clinicians are forced to **infer trajectory from a single snapshot.**

5.6. Cognitive Bias in Clinical Reasoning

Diagnostic reasoning is vulnerable to well-described biases:

- **Anchoring bias** – reliance on initial impression
- **Availability bias** – preference for familiar diagnoses
- **Premature closure** – failure to revisit initial conclusions

The National Academies of Sciences, Engineering, and Medicine highlights that diagnostic error frequently arises from these cognitive processes within complex clinical systems [6].

5.7. The Problem of “Pure Autonomic Failure” as a Provisional Label

By definition, PAF is:

- Diagnosed in the absence of other neurological features
- Based on current observation

However:

- Up to one-third of patients later develop broader neurodegenerative disease [7]

- Others remain stable or show atypical trajectories

Thus, PAF is often a **diagnosis of exclusion and temporality**, not certainty.

6. Case-Based Insight

The longitudinal trajectory underpinning this analysis demonstrates:

6.1. Initial Interpretation

- Severe, progressive autonomic failure
- Consistent with PAF

6.2. Subsequent Development

- Stabilisation and recovery
- Functional improvement across multiple systems

6.3. Final Interpretation

- Multifactorial secondary dysautonomia
- Injury-based rather than degenerative

This sequence reveals a critical truth:

The initial neurodegenerative assumption was reasonable—but but ultimately incorrect.

The error did not arise from negligence, but from:

- Structural limitations
- Pattern recognition bias
- Lack of longitudinal data

7. Reframing the Diagnostic Approach

7.1. From Deterministic to Probabilistic Diagnosis

Instead of:

“This is neurodegenerative autonomic failure”

A more accurate approach is:

“This is severe autonomic dysfunction of uncertain aetiology, requiring longitudinal evaluation”

8. Key Principles for Improvement

- Maintain diagnostic openness
- Prioritise trajectory over snapshot
- Actively consider reversible causes
- Reassess diagnosis over time
- Recognise severity does not equal irreversibility

9. Discussion

The assumption that autonomic dysfunction is neurodegenerative is not purely evidence-driven. It is shaped by:

- Historical disease models
- Clinical training patterns
- Cognitive biases
- Limitations in diagnostic tools

Importantly, this assumption can have consequences:

- Psychological burden for patients
- Premature prognostic conclusions
- Missed opportunities for treatment

This paper argues that autonomic medicine requires a **shift in paradigm**:

From static diagnosis → to dynamic, evolving interpretation

10. Conclusion

Autonomic dysfunction is frequently assumed to be neurodegenerative because:

- It is historically framed within degenerative disease models
- Severe presentations mimic neurodegeneration
- Diagnostic tools lack specificity for aetiology
- Clinical reasoning is influenced by cognitive bias
- Time-dependent disease evolution is underappreciated

This case demonstrates that such assumptions, while understandable, are not always correct. A more accurate model recognises dysautonomia as a **heterogeneous, evolving condition, requiring ongoing reassessment and diagnostic humility.**

Four Years Recovery [An anthem that conveys the four year journey and where I have ended up]

<https://heyzine.com/flip-book/0d032e9066.html>

There was a time when the ground gave way
When standing felt like a gamble each day,
Heart and head in a wild, unsure race,
Every small step taken on borrowed grace.
But storms don't last forever, they say,
And slowly, slowly, the fog rolled away,
The chaos hushed, the noise drew thin,
And the body learned how to hold again. Oh sing it loud, four years on!
We're not the same — but we're still strong!
The cliffs are gone, the lights are on,
And we're dancing where the fear once shone!
Hey!
The heart found rhythm, the pressure stayed,
No more falling off the edge unmade,
Still a wobble here, a pause or two,
But corrections come — and they see me through.
The gut found peace, the fire grew tame,
Stopped dragging the whole wide system aflame,
Not perfect, no — but steady and kind,
A working truce with the body and mind. Oh sing it loud, four years on!
We're not restored — but we're still strong!
No cliff-edge days, no siren songs,
Just steady feet and carried-on!
Hey!
Now energy's dear — not free to spend,
It likes a plan, it likes a friend,
You spend it wise, you earn it back,
You rush it hard — it cuts you slack (...later).
Some systems lag, they take their time,
Precision's slow — but that's just fine,
The pipes might grumble, the timing's odd,
But we laugh and say, “Sure isn't that God?” Oh sing it loud, four years on!
Thin margins, aye — but the fear is gone!
We know the signs, we read them well,
And live our lives inside the swell!
Hey!
Was it failure?
Choir (harmonised):
Stability first... reserve comes slow...
And wisdom grows where fear once went...
We once got told the road went down,
That every year would steal more ground,
But we learned the truth — the kinder way:
This wasn't decline — just damage in play.
Three hard hits, then time stood still,
And time — that quiet healer's will —

Did what it does when given space:
It taught the system a brand-new grace. Oh sing it loud, four years on!
Not fixed, not frail — just carrying on!
The noise is low, the map is clear,
And life is wide when fear's not here!
Hey! Hey! Four years on... and still we sing... Four years on... and everything...
Four years on — we're standing strong...

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