

Case Study

Advances in Neurology and Neuroscience

My Health Story 2008 to 2026

One Step Forward Followed by Another Downturn within a Published, Literature-Grounded MSAD Hypothesis

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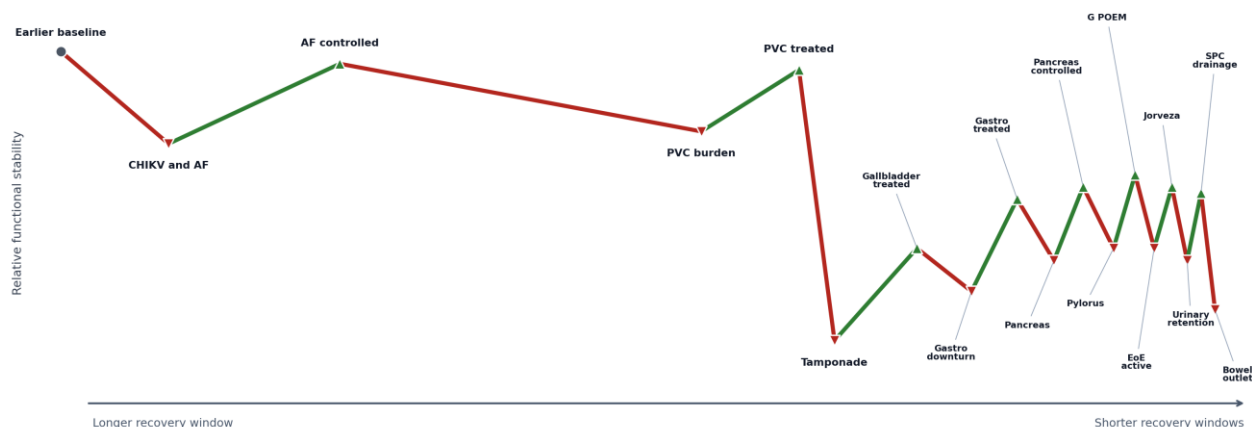
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Purpose

This document holds the whole story in one place. Its central pattern is repeated progress followed by a new downturn, with the period between recovery and the next problem appearing to shorten over time. The sequence is interpreted through a literature-grounded framework of Multifactorial Secondary Autonomic Dysfunction, or MSAD. The framework treats the autonomic nervous system as a distributed coordinating network whose different components can be affected and recover differently. This interpretation is not being introduced for the first time in this narrative: it has been developed across my published, peer-reviewed papers on MSAD, autonomic vulnerability, visceral dysautonomia, EoE, recovery, upright gait failure, autonomic reserve and visual dysfunction [1-

35]. MSAD is used here as a proposed organising formulation, not as a formally recognised diagnosis. It does not claim that autonomic dysfunction caused every illness. It asks whether continuing regulatory vulnerability, interacting with local organ disease, procedures, medicines, ageing and reduced physiological reserve, offers a coherent explanation for the repeated pattern. Publication and peer review establish that this is a documented scholarly hypothesis; they do not prove that it is correct in this individual case or that each condition was autonomically caused. The narrative therefore distinguishes documented facts, established physiology, published hypothesis, case-specific interpretation and unproven causal links. The figure is conceptual and does not represent a measured clinical score.



Conceptual MSAD overview Each improvement is real but the next downturn arrives sooner

Document Map

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1. The Central Account

The clearest way I can describe my health is one step forward followed by two steps back. A problem is identified, treated and brought under control. For a time there is relief and genuine recovery. Then another problem appears, often in a different organ system, and the work begins again. More recently, the period of stability between recovery and the next downturn appears to be getting shorter. The sequence began with chikungunya, myocarditis and atrial fibrillation. The AF was brought under control and did not remain the active problem. Premature ventricular contractions then became dominant. The PVC ablation treated the rhythm problem but caused ventricular perforation, tamponade and the major downturn of October 2021. After that came a succession of gallbladder, gastrointestinal, pancreatic and pyloric problems. After G-POEM improved the pyloric dysfunction, eosinophilic oesophagitis became clinically active and required Jorveza treatment before it came under control. Urinary and now bowel-outlet problems followed. Each intervention achieved something important; none restored the whole system to its former reserve. I describe the overall pattern cautiously as Multifactorial Secondary Autonomic Dysfunction, or MSAD. Published autonomic literature recognises that dysautonomia can be primary or secondary, can affect one component or several, and can produce cardiovascular, gastrointestinal, thermoregulatory and urogenital manifestations [1,16]. My own peer-reviewed papers have already proposed MSAD and autonomic vulnerability as a disease-modifying substrate across this course [20-29,35]. MSAD is my proposed name for the way these established features appear to converge in this case; it is not itself an accepted diagnostic category.

The central conclusion is not that treatment has failed or that disease has moved from one organ to another. It is that each successful intervention has removed an important local problem while a broader vulnerability in regulation, signalling, compensation and physiological reserve may have remained. Different autonomic components can respond differently to illness and stress [17]. One function can therefore improve while another becomes the next limiting point. The published papers establish this as a previously articulated, literature-informed hypothesis. They do not convert the complete causal synthesis into proof.

2. The Autonomic Nervous System and Autonomic Dysfunction

The autonomic nervous system, or ANS, is the body's largely involuntary system for maintaining internal stability while conditions continually change. It is not a single nerve or a simple on-off switch. It is a distributed network linking the brain, brainstem, spinal cord, peripheral ganglia, sympathetic and parasympathetic pathways, visceral sensory input and closely related enteric and pelvic circuits. The central autonomic network integrates information about the internal state of the body and coordinates cardiovascular, respiratory, neuroendocrine, digestive, thermoregulatory and sphincter responses [14,15]. In practical terms, the ANS helps adjust heart rate and cardiac force, constrict or dilate blood vessels, preserve blood pressure when standing, redirect blood flow after a meal or during exertion, regulate sweating and skin blood flow, influence gastrointestinal motility and secretion, and coordinate bladder storage and emptying. These functions depend on feedback loops rather than isolated commands. Autonomic cardiovascular control is therefore dynamic, involving rapid sensing, central integration and an appropriate response through the heart and circulation [18].

3. What Autonomic Dysfunction Means

Autonomic dysfunction means that one or more parts of this regulatory system are not sensing, integrating or responding normally [1,16]. The disturbance may be too little activity, too much activity, an abnormally timed response or poor coordination between components. It need not be total autonomic failure, and it need not affect every organ at the same time or to the same degree. The ANS has parallel, state-dependent pathways, and its components can be differentially involved in disease and stress [15,17]. Symptoms may therefore fluctuate with posture, walking, heat, meals, sleep, pain, emotion, infection, surgery, medication and volume status. The word secondary indicates that the dysfunction is understood as arising in association with another acquired illness, injury, procedure, medicine or systemic condition rather than being assumed to be a primary inherited or neurodegenerative autonomic disorder. In my case, the term is descriptive and hypothesis-generating. The chronology after chikungunya, myocarditis, cardiac disease, tamponade and major surgery supports a secondary formulation, but chronology alone cannot establish which event caused which later abnormality.

Domain	Recognised autonomic role	Relevance and evidential boundary in this journey
Cardiovascular	Heart rate, cardiac force, vascular tone, venous return and baroreflex responses.	Repeated large postural BP falls, limited or variable HR compensation, hypertensive starting pressures, delayed walking failure and preserved recumbent cycling are the strongest directly autonomic features. AF and PVCs are documented cardiac disorders but are not automatically proved to be autonomic in origin.
Gastrointestinal	Splanchnic blood flow, motility, secretion, gastric emptying and coordinated sphincter activity.	Post-meal crashes and gastroparesis are autonomically relevant. Eosinophilic oesophagitis, gallbladder disease and pancreatic insufficiency may be local or mixed conditions and should not be relabelled autonomic without evidence.
Thermoregulatory	Sweating, skin blood flow and heat dissipation.	Heat intolerance, altered sweating and later improvement in thermoregulation fit an autonomic phenotype but are not specific to one lesion.
Urinary and pelvic	Bladder storage and emptying through integrated autonomic, somatic and sensory pathways.	Retention and difficult evacuation make neural coordination relevant, but obstruction, detrusor underactivity, pelvic-floor dysfunction, catheter effects and local disease still require objective assessment.
Sensory and glandular	Pupil size, accommodation, lacrimal and salivary secretion, and vascular or neural modulation of sensory organs.	Dry eye and light-adaptation symptoms have plausible autonomic pathways. Hearing and taste links are more indirect and weaker; ophthalmic, retinal, audiological, oral, medication, nutritional and age-related explanations remain essential.
Demand and recovery	Moment-to-moment matching of circulation and organ function to posture, exertion, meals and stress.	The walking-cycling contrast, post-meal decline and fluctuating tolerance suggest impaired adaptation to demand. Slower tissue healing is a separate observation and is not, by itself, proof of autonomic causation.

4. The Strongest Autonomic Evidence in this Story

The most persuasive autonomic evidence is not simply the number of organs involved. It is the recurrent, measured and posture-dependent disturbance of blood-pressure control; the sometimes limited heart-rate compensation; the coexistence of hypertensive and hypotensive states; the delayed failure during upright walking despite preserved recumbent cycling; and the associated meal, heat and sweating patterns. The gastrointestinal, urinary and pelvic features add coherence because these domains are neurally regulated, but they are less specific and must remain open to organ-specific explanations.

5. The Literature Grounded MSAD Framework

The framework is deliberately descriptive. It places three processes in the same clinical picture: a shared vulnerability in autonomic regulation; discrete local diseases or mechanical problems that require their own treatment; and background modifiers such as ageing, medicines, volume state, sleep, inflammation, surgery and deconditioning. It asks whether this combined model explains the timing, multisystem distribution and partial recovery more coherently than either a single lesion or a series of wholly unrelated events. This layered formulation has been developed in my published MSAD and disease-modifying-substrate papers [20-26]. It does not assume that every later disorder was caused by autonomic dysfunction.

MSAD element	Literature grounded meaning	Status in this case
Multifactorial	Several contributors may interact: infection, cardiac disease, procedural injury, surgery, ageing, medication, volume state, deconditioning and organ disease.	The model does not require every event to have one cause. The size of each contribution in this case is not yet measured.
Secondary	Autonomic dysfunction can accompany or follow acquired systemic disease and other physiological insults [1,16].	The chronology is compatible with a secondary process but does not prove a particular initiating cause.
Autonomic	The ANS is a distributed network with parallel, state-dependent and differentially affected components [15,17].	The measured haemodynamic and multisystem phenotype fits; formal testing is needed to define the site, extent and mechanism.
Dynamic and reserve limited	Different functions can recover at different rates, while ageing and accumulated stress can reduce homeostatic reserve [12,17].	Selective recovery is observed. Reduced reserve and shortening recovery intervals remain working interpretations rather than measured biomarkers.

6. How one Recovery can be followed by Another Downturn

- A discrete problem becomes dominant and is treated: rhythm, gallbladder, oesophageal inflammation, pancreatic insufficiency, pyloric resistance or urinary drainage.
- The treatment produces a genuine local gain. That success should be preserved in the narrative rather than dismissed because another problem later appears.
- The intervention may not correct the wider regulatory vulnerability. Autonomic compensation, vascular control and physiological reserve may remain incomplete even when the treated organ is working better.
- A later demand such as prolonged standing, a meal, heat, surgery, inflammation, sleep loss, medication change or pelvic irritation may expose another vulnerable control loop or a separate organ disorder.
- The next weakest or most stressed domain becomes clinically dominant. This can look as though illness has moved from system to system, although the more cautious interpretation is a changing bottleneck within a vulnerable network plus local disease.
- If the recovery intervals are truly shortening, cumulative burden or reduced reserve is one plausible explanation. Increased surveillance, treatment effects, ageing and coincidence are competing explanations that must also be considered.

7. The Coincidence Question

The succession of problems cannot be declared non-coincidental simply because it forms a striking story. Its clinical significance comes from the continuous background phenotype that persists across the separate episodes: abnormal postural blood-pressure control, variable heart-rate compensation, heat and sweating disturbance, post-meal deterioration, exercise-position dependence and fluctuating recovery. These recurring regulatory features make a shared autonomic contribution more plausible than the organ sequence alone would make it. MSAD therefore functions as an overarching hypothesis, not a claim that the ANS directly caused the gallbladder, eosinophilic oesophagitis, pancreatic

insufficiency, procedural perforation or every pelvic symptom. It proposes that acquired autonomic vulnerability may have shaped the body's response to these events, limited compensation after them and helped determine which function became the next clinical bottleneck. This is the level at which the coincidence can be understood without overstating causation.

8. The Published Unifying Hypothesis

Across my peer-reviewed publications, I have proposed that viral illness, cardiac injury, haemodynamic collapse, surgery and later local disease may cumulatively reduce autonomic flexibility and physiological reserve [20-29]. In this model, secondary dysautonomia is not one additional organ diagnosis. It is the shared regulatory terrain within which separate diagnoses, procedures and recoveries occur. That terrain may help explain why a local treatment can succeed while the whole-body pattern continues and another vulnerable function becomes the next bottleneck. The proposed mechanism is disease modifying rather than universally disease causing. Autonomic dysregulation may alter timing and coordination of motility, secretion, vascular perfusion, inflammatory thresholds, organ-to-organ signalling, stress responses and compensation without being the sole origin of gallbladder disease, pancreatic insufficiency, pyloric dysfunction, EoE, urinary retention or sensory symptoms [22-26]. This distinction allows the conditions to be physiologically connected without erasing their independent pathology. The evidential boundary is critical. Peer review and publication show that the hypothesis has been stated transparently, placed in relation to existing literature and judged suitable for scholarly discussion. They do not establish that the hypothesis is correct in me, prove that one event caused the next or substitute for objective testing. Several papers arising from the same longitudinal case and conceptual programme are repeated scholarly examination of one model, not independent replication of that model.

9. Physiological Linkage Across Systems

Domain	Physiological bridge	Case-specific evidential boundary
Gallbladder and biliary tract	Gallbladder filling, contraction and biliary coordination reflect enteric, vagal, sympathetic and hormonal influences [30].	Autonomic dysregulation could contribute to impaired motility or timing, but gallbladder disease and response to treatment do not identify autonomic causation.
Pancreas	Pancreatic exocrine secretion is neurally regulated, including vagal stimulation and sympathetic modulation [31].	Disordered autonomic signalling is physiologically plausible as a modifier of secretion, but structural, metabolic, medication-related and idiopathic causes remain possible.
Stomach and pylorus	Gastric accommodation, emptying and pyloric coordination depend on vagal-enteric control; autonomic abnormalities occur in gastroparesis cohorts [7,8].	G-POEM treated a downstream pyloric bottleneck. Its success does not by itself establish why that dysfunction developed.
Oesophagus and EoE	EoE is an antigen-driven inflammatory disease. The published hypothesis proposes autonomic effects on motility, clearance, epithelial integrity and neuroimmune thresholds as possible modifiers [19,24-26].	The autonomic model does not replace the inflammatory diagnosis or make dysautonomia the proven primary cause.
Eye and night vision	Pupil control, accommodation and lacrimal secretion have autonomic components [32,33,35].	Dry eye and light-adaptation symptoms fit the hypothesis, but retinal dark adaptation and other ocular mechanisms require independent ophthalmic assessment.

Hearing and taste	Autonomic activity may interact with auditory physiology, while salivary and cephalic reflexes influence the oral environment in which taste is perceived [33,34].	These links are indirect and comparatively weak. Age, medication, ear disease, dental or oral disease, smell, nutrition and neurological causes must remain in the differential.
Bladder, bowel and sphincters	Storage, emptying and outlet relaxation use integrated autonomic, somatic and sensory pathways, with recognised bladder-bowel cross-signalling [9-11].	Neural coordination is relevant, but the specific SPC-to-anal-outlet hypothesis remains unproved and requires objective urological and anorectal assessment.

10. What the Framework Validates

The independent literature supports the coherence of the component pattern: the ANS is a distributed coordinating network dysautonomia may be secondary and multisystem different autonomic components may be affected differently orthostatic hypotension and supine hypertension have consensus definitions; autonomic abnormalities occur in gastroparesis cohorts; biliary and pancreatic functions have neural regulation lacrimal, pupillary and salivary functions have autonomic components and bladder and sphincter control use integrated neural pathways [1-3,7,9,14-18,31-34]. My published papers then bring these components together as an explicit MSAD and disease-modifying-substrate hypothesis [20-29,35].

11. What the Framework does not prove

Neither the independent literature nor my publications prove the full causal chain: that chikungunya caused the later course, cardiac surgery caused every symptom, local organ disease was autonomic, the SPC caused bowel dysfunction, one anatomical lesion explains the whole course, sensory changes were autonomically driven, or recovery intervals are shortening. Repeated publication of a hypothesis developed from the same case is not independent confirmation. These propositions require testing against dated records, formal autonomic studies, medication and volume review, organ-specific investigation, urodynamics, anorectal physiology and competing diagnoses.

12. Timeline at a Glance

Period	Event	Meaning in the story
Before 2008	Physically and professionally active	No established history of orthostatic intolerance, gastrointestinal dysmotility, bladder dysfunction or exercise limitation.
2008 to 2015	Chikungunya, myocarditis and atrial fibrillation	The AF was brought under control and no longer remained the active rhythm problem, but autonomic vulnerability persisted.
2016 to 2021	PVC burden increased to about 15,000 in 24 hours	A new rhythm problem replaced the earlier one and ultimately required ablation.
15 Oct 2021	PVCs treated but ablation caused perforation and tamponade	The step forward in rhythm control produced the largest downturn: emergency sternotomy, bypass and ventricular repair.
2022	Gallbladder problem treated	One discrete problem settled, but wider multisystem instability continued.
2022 to 2023	Gastrointestinal dysfunction treated	Gastric and oesophageal problems were progressively managed while heart rhythm and blood pressure remained under continuing review.
2023	Pancreatic insufficiency brought under control	Enzyme support improved digestion, but another gastrointestinal control point remained.
2024	Pyloric dysfunction treated by G POEM	Gastric emptying improved after treatment of the muscular outlet at the bottom of the stomach.
After G POEM	Eosinophilic oesophagitis became clinically active	Jorveza, an oesophagus-targeted budesonide treatment, brought the EoE under control.
2024 to 2026	Heart rhythm and blood pressure continually managed	Overall capacity improved, but delayed upright deterioration and regulatory instability remained.
2026	Urinary retention managed with an SPC	Bladder drainage improved, but bowel evacuation then deteriorated as the anal outlet failed to relax normally.

13. Health before 2008

Before late 2008, I understood myself as physically and professionally active. I did not live with the orthostatic intolerance, gastrointestinal dysmotility, urinary dysfunction or exercise limitation that later came to define so much of my health. This matters because the later pattern represented a change from my prior baseline, not simply the continuation of a lifelong condition.

14. The first hit in 2008 and 2009

14.1. Chikungunya in Bandung

In late 2008, while in Bandung, Indonesia, I contracted laboratory-confirmed chikungunya. The acute illness included fever, rash, severe joint pain and profound fatigue. The obvious infection passed, but I did not return completely to the person I had been. Persistent fatigue and joint symptoms were followed by dizziness

on standing, poorer heat tolerance, fluctuating blood pressure and reduced exercise resilience.

14.2. Myocarditis and the Beginning of a Longer Pattern

In 2009, a cardiologist at North Shore Hospital diagnosed myocarditis, and this remains documented in my medical record. Published reports show that chikungunya can involve the cardiovascular system, including myocarditis, arrhythmia and hypotension [4]. That evidence makes the temporal link biologically plausible, but it does not prove virological causation in my individual case. This distinction matters: the myocarditis is documented; the specific causal link remains a reasoned attribution. From that point onward, my body increasingly behaved as though cardiovascular and autonomic regulation had become vulnerable. Symptoms did not remain dramatic every day, but the system no longer felt completely dependable.

14.3. The Long Period of Vulnerability from 2009 to 2021

Between 2009 and 2021, I lived through a long compensated phase. Atrial fibrillation was brought under control and did not continue as the active problem. That was a genuine step forward. Orthostatic light-headedness, presyncope, heat intolerance, blood-pressure variability and delayed recovery from exertion nevertheless continued in the background. The next major problem was ventricular ectopy. Premature ventricular contractions became increasingly frequent and reached approximately 15,000 PVCs in 24 hours. I could still function at a high level, but the rhythm burden showed that one corrected cardiac problem had been followed by another.

14.4. The Cardiac Catastrophe on 15 October 2021

On 15 October 2021, I underwent an intended LVOT PVC ablation. The procedure treated the PVC problem, but it also perforated the left ventricular wall. Blood accumulated around the heart, producing acute cardiac tamponade and haemodynamic collapse. The step forward and the major downturn occurred in the same intervention. A sternotomy was performed. Cardiopulmonary bypass was established and the ventricular injury was repaired during an operation lasting approximately six hours. The intervention saved my life. It was also a profound cardiovascular, surgical, inflammatory and psychological event. I now understand this day as the major dividing line in my later health: the body before 15 October 2021, and the body after it. Prospective studies

show that cardiac surgery can acutely impair baroreflex sensitivity and alter sympathovagal balance, while other work documents at least partial heart-rate-variability recovery over subsequent weeks [5,6]. This supports surgery as a plausible autonomic stressor, not as proof of permanent injury. My working interpretation is that the tamponade and emergency repair amplified a vulnerability that already existed.

14.5. The Broadest Dysregulation in 2022 and 2023

The widest disturbance emerged after the 2021 event. My body no longer behaved as a collection of separate organs. Blood pressure, heart rate, digestion, bladder function, temperature control, fatigue, balance and cognition appeared to fluctuate as parts of one poorly coordinated regulatory system.

The principal features were:

- Severe orthostatic hypotension together with periods of marked supine or resting hypertension.
- Little or delayed heart-rate compensation during some major blood-pressure falls.
- Profound fatigue, poor exercise tolerance and slow recovery after exertion.
- Autonomic storms, temperature dysregulation, altered sweating and poor heat tolerance.
- Gastroparesis and wider gastrointestinal dysmotility, including constipation and difficult evacuation.
- Oesophageal disease and swallowing or reflux symptoms, including eosinophilic oesophagitis.
- Pancreatic exocrine dysfunction requiring enzyme support.
- Urinary hesitancy, retention and an increasing need for catheter-based management.
- Cognitive slowing, head pressure when upright, presyncope, gait instability and falls risk.

One illustrative early pattern was a supine blood pressure of 215/105 mmHg falling to 145/85 within 30 seconds of standing, with heart rate changing only from 55 to 58 beats per minute. Consensus literature recognises orthostatic hypotension as a haemodynamic finding and separately recognises the coexistence of supine hypertension with autonomic failure [2,3]. The pattern is therefore autonomically relevant, but formal assessment is required before labelling it neurogenic or assigning a specific lesion.

15. Illustrative Haemodynamic Observations

Date	Position	BP mmHg	HR bpm	Interpretive value
2022 example	Supine to standing at 30 seconds	215/105 to 145/85	55 to 58	Large rapid pressure fall with very limited immediate heart-rate response.
July 2026	Supine to standing	131/59 to 87/51	51 to 73	Marked orthostatic fall despite a clearer heart-rate response.
July 2026	Seated to standing	189/94 to 158/89	59 to 67	Hypertensive starting pressure with a significant postural fall.
25 Aug 2026	Resting to standing	211/97 to 166/78	62 to 62	45 mmHg systolic fall with no measured heart-rate increase.
27 Aug 2026	Resting to standing	166/83 to 126/71	80 to 86	Reproducible 40 mmHg systolic fall with limited compensation.

These are selected observations rather than a complete physiological study. They demonstrate the pattern that has shaped my daily life but do not, alone, determine whether the mechanism is neurogenic, cardiac, vascular, medication-related, volume-related or mixed [2].

16. The Gastrointestinal Story

After the cardiac catastrophe, the sequence moved through the digestive system. A gallbladder problem was identified and treated. That issue settled, but the wider gastrointestinal system then became one of the longest-lasting expressions of the disorder. I experienced delayed gastric emptying, early satiety, nausea, bloating, reflux, oesophageal dysfunction and chronic constipation. Multicentre research in gastroparesis has found autonomic abnormalities and associations between parasympathetic dysfunction, delayed emptying and symptom severity [7]. This establishes a plausible autonomic contribution, not a complete explanation for every digestive diagnosis. The digestive problems were addressed in stages. Pancreatic exocrine insufficiency was brought under control with Creon. The muscular outlet at the bottom of the stomach, the pylorus, remained resistant; G-POEM in 2024 relieved that outlet dysfunction and improved the gastroparesis component. A sham-controlled trial supports G-POEM as an effective treatment for severe gastroparesis, but response to G-POEM does not by itself identify the original cause of the pyloric dysfunction [8]. The physiological bridge across these events is regulation rather than a claim of one disease spreading from organ to organ. Gallbladder motility, pancreatic secretion, gastric emptying and pyloric coordination all have neural as well as local and hormonal control [30,31]. My published visceral-dysautonomia papers propose that impaired autonomic timing, signalling and compensation may modify how these separate conditions emerge or recover [24,25]. This is a coherent mechanism to investigate, not retrospective proof that dysautonomia caused the gallbladder, pancreas or pylorus to fail.

After the G-POEM improvement, eosinophilic oesophagitis became clinically active and emerged as the next significant problem. It required treatment with Jorveza, an orodispersible formulation of budesonide designed to act on the oesophagus, before the EoE came under control. Randomised trial evidence supports budesonide orodispersible tablets for inducing remission

in active EoE [19]. EoE is a distinct inflammatory disease and is not being relabelled as autonomic; its importance here is chronological, as another substantial problem becoming dominant after the preceding one had improved. In the published EoE hypothesis, I have proposed a narrower relationship: autonomic vulnerability may modify oesophageal motility, clearance, epithelial conditions or neuroimmune thresholds while EoE remains an inflammatory disease in its own right [26]. That formulation permits an interaction without claiming that autonomic dysfunction is the established primary cause of EoE. Each of these was a meaningful step forward: the gallbladder problem settled, pancreatic support improved digestion, G-POEM improved gastric emptying, and Jorveza brought the EoE under control. Yet improvement in one part did not end the larger story. Heart rhythm and blood-pressure regulation still needed continual attention, and the next major system to fail was urinary. By 2026, there were signs that gastrointestinal and oesophageal stability had improved; at one point the eosinophilic oesophagitis remained quiet through approximately eight weeks without medication. Constipation and outlet evacuation, however, again became prominent during the more recent urinary catheter period. Meals remain an autonomic load. A characteristic evening pattern has been a marked crash roughly two hours after dinner. I understand this as an observation about timing and demand, not proof of a single mechanism.

17. Sensory and Glandular Features

My wider writing also records dry eye, difficulty or delay when adapting to changes in light and night vision, changes in hearing, altered taste and other smaller complications. These features matter because a unifying hypothesis should account for the full phenotype, not only the largest organ events. They do not all carry the same evidential weight. The eye provides the clearest physiological bridge in this group. Pupil diameter, accommodation and lacrimal secretion are autonomically regulated [32,33]. My published paper on visual dysfunction placed dry eye, photophobia, visual fatigue and light-adaptation symptoms within the MSAD hypothesis [35]. However, night vision also depends on the retina, lens and other ocular mechanisms that are not simply autonomic. An autonomic contribution is therefore plausible, but ophthalmic causes remain independently important. The proposed links with hearing and taste are more tentative. Limited research connects parasympathetic measures with hearing impairment,

but the relationship is not established as causal [33]. Taste is influenced indirectly by salivary secretion and cephalic reflexes under autonomic control, yet taste change may also arise from medication, oral or dental disease, smell disturbance, nutritional deficiency, infection or neurological disease [34]. These symptoms extend the hypothesis-generating pattern; they should not be presented as equally strong evidence of dysautonomia.

18. The Urinary and Pelvic Story

Urinary dysfunction evolved from hesitancy and incomplete emptying to retention requiring regular self-catheterisation. By 2026 I was catheterising in the morning and evening. Bladder storage and emptying depend on coordinated brain, spinal, peripheral autonomic and somatic pathways [9]. That physiology makes autonomic involvement possible, but retention also requires assessment for obstruction, detrusor underactivity, medication effects and local urological disease. A suprapubic catheter, or SPC, restored dependable drainage and brought the immediate emptying problem under practical control. It was another real step forward, but the period before the next downturn was short.

19. The September 2026 Catheter Complications

On 6 September 2026, the suprapubic catheter tubing was accidentally cut during dressing removal. The balloon deflated, the catheter fell out, and the tract closed before successful reinsertion could be achieved. Subsequent catheter management was complicated by bright red bleeding, urethral leakage or incontinence, bladder spasms and considerable uncertainty about what was normal during healing. A particularly important observation was that bleeding appeared or increased as the bladder filled, while the urine could clear rapidly when the SPC leg bag was allowed to drain freely. My working interpretation is that decompression reduces stretch, pressure and mechanical disturbance at an injured area. That interpretation is plausible but still requires clinical correlation, because bleeding can also reflect catheter irritation, infection, granulation tissue, stones, mucosal injury or other urological causes.

20. The Bowel Outlet Pattern

Soon after the urinary problem had been brought under practical control with the SPC, the bowel stopped working normally. The urge to defecate remained and stool appeared to reach the rectum, yet the anal outlet would not relax. Repeated attempts, incomplete emptying and the sensation that the outlet was holding shut suggested an outlet disorder more than a simple failure of the colon to move stool forward. I have developed a specific hypothesis: even in a continuously drained and largely decompressed bladder, the catheter tip or balloon may continue to provide mechanical and sensory input. Experimental and clinical literature supports neural cross-talk and cross-sensitisation between bladder and bowel pathways [10]. This makes altered pelvic signalling plausible, but the evidence does not establish that an SPC causes anal dyssynergia. Symptoms alone also cannot diagnose a defecatory disorder; anorectal manometry, balloon expulsion testing and, when indicated, defecography provide the relevant objective assessment [11].

21. Walking Failure with Preserved Riding my Adult Trike.

A revealing feature in 2026 has been the contrast between walking and recumbent cycling. I can often cycle in a supported recumbent position, yet sustained upright walking can produce a delayed failure after approximately 15 to 30 minutes or more. Blood pressure may fall by 50 to 80 mmHg. Head pressure, light-headedness, visual narrowing, gait dyscontrol and near-falls can follow. This contrast tells me that I retain substantial capacity to generate work. The additional problem is gravitational and regulatory: upright walking requires sustained venous return, vasoconstriction and beat-to-beat pressure control. Delayed orthostatic hypotension is a recognised autonomic phenotype, but its cause and prognosis cannot be inferred from timing alone [13]. Preserved cycling therefore argues against a simple global loss of fitness, while the walking pattern identifies a serious posture-dependent constraint and continuing falls risk.

22. Healing and Reduced Repair Reserve

At age 74, I have become increasingly aware that my body still heals but does so more slowly. My own phrase is simple: my bounce is not as quick as it used to be. The change became particularly noticeable after the 2021 cardiac catastrophe. A shoulder injury and repair, the major sternotomy and later bladder or catheter-related tissue injury have each required more time and more careful pacing than I once expected. Ageing offers the strongest established explanation for slower repair. Geriatric literature describes reduced physiological reserve as increased vulnerability to incomplete or delayed restoration of homeostasis after a stressor [12]. I use that concept cautiously: it supports the language of reserve, but it does not diagnose frailty in me. My additional working idea is reduced repair reserve under autonomic constraint. This remains a hypothesis, not proof that orthostatic hypotension directly impairs a particular wound.

23. Selective Recovery from 2024 to 2026

Recovery has been real, but repeatedly interrupted. During 2024 to 2026 the autonomic storms diminished. My heart rate began to respond more appropriately to activity. Thermoregulation and sweating became more dependable. I regained meaningful cognitive and occupational work, protected exercise and wider participation in life. In 2022 and 2023 my heart rate rarely rose above about 70 beats per minute even when exertion produced severe breathlessness and fatigue. By late 2025 it could reach about 100 during walking and approximately 120 when pushed, with far less exercise intolerance. The difficult change is temporal. Earlier in the story, a treatment could be followed by a comparatively long period before the next major problem. More recently, the interval appears shorter: urinary drainage improved with the SPC, and bowel outlet dysfunction followed soon afterwards. This perception needs continued longitudinal recording, but it is now one of the central features of my lived experience. The fact that several independent functions improved over time supports my working view that this remains a recovery-capable process. It does not, however, exclude a progressive disorder; delayed orthostatic hypotension has progressed in some longitudinal cohorts [13]. The non-degenerative interpretation therefore remains provisional and

should continue to be tested against neurological examination, formal autonomic findings and the direction of change over time.

24. Postoperative Reserve and Re-Stabilisation in September 2026

Following general surgery in late August 2026, I appeared to have less accessible reserve. From 11 to 13 September an unexpected emotional trigger produced a rapid fight-or-flight response; later there was generalised nocturnal sweating, exhaustion and the sense of reaching a physiological brick wall. By late 14 September, physical, cognitive and emotional function had improved

markedly. Two later emotional triggers were contained without the earlier spread or escalation. I interpret this sequence as a temporary narrowing of available reserve rather than proof of new structural injury. The improvement over several days and better containment of later events support the possibility that reserve can be drawn down and then reconstituted. Other explanations, including infection, medication effects, endocrine or metabolic disturbance, sleep loss, volume change and postoperative complications, remain clinically important.

25. My Current Health Picture

Domain	Working summary
Function that has returned	Sustained thinking and professional work; improved heart-rate response; better thermoregulation; fewer autonomic storms; meaningful recumbent exercise; wider participation in daily life.
Function that remains vulnerable	Upright blood-pressure control; delayed walking tolerance; falls risk; urinary emptying and catheter dependence; bowel outlet relaxation; post-meal stability; tissue-repair speed.
Common stressors	Prolonged standing or walking; heat; large or evening meals; surgery and inflammation; poor sleep; emotional salience; cumulative physical and cognitive demand.
Warning pattern	Head pressure, light-headedness, visual narrowing, gait change, near-fall, sudden exhaustion, marked BP change, disproportionate sweating or cross-system symptom spread.
Protective strategies used	Fluids and salt as clinically appropriate; compression; head-of-bed elevation; pacing; smaller meals; supported or recumbent exercise; walking stick; bowel support; catheter routines; specialist review.
Current framing	Each treatment produces a real step forward, but another system becomes vulnerable before reserve is fully rebuilt. Recovery windows appear to be shortening.

26. What Recovery Now means to me

Recovery is no longer defined as a complete return to the body I had before 2008 or before 2021. Every successful intervention matters: AF controlled, PVCs treated, gallbladder settled, gastrointestinal and pancreatic problems managed, pyloric dysfunction relieved by G-POEM, EoE brought under control with Jorveza, and urinary drainage restored by the SPC. These are not imaginary gains. They are genuine steps forward. The harder truth is that each gain has been followed by another downturn. The current challenge is not only to recover from the latest problem, but to understand why a new one appears so soon and whether the interval is truly shortening. A breakthrough event does not cancel earlier recovery, but it may show that the body has not yet rebuilt enough reserve to absorb the next demand.

27. The Three Hit Model and its Limits

I organise the story using a three-hit model:

- Late 2008 chikungunya and the probable post-viral initiation of autonomic and cardiovascular vulnerability.
- The cardiac perforation, haemopericardium and tamponade during PVC ablation on 15 October 2021.

- Emergency sternotomy, cardiopulmonary bypass and direct ventricular repair as a further major haemodynamic, inflammatory and surgical load.

This sequence is coherent and biologically plausible. Chikungunya has documented cardiovascular manifestations, and cardiac surgery can produce measurable early autonomic disturbance [4-6]. Those findings support the proposed pathway at the level of plausibility. They do not prove that chikungunya caused the later arrhythmias, that cardiopulmonary bypass caused every autonomic symptom, or that one anatomical lesion accounts for the whole course. Medication, ageing, volume status, deconditioning, cardiac and vascular factors, concurrent disease and individual organ pathology may all contribute. The value of the model is therefore not that it closes the case. Its value is that it holds the sequence together, protects the complexity of the story and generates testable questions. The correct evidential wording is literature-consistent and hypothesis-generating, not literature-proven for this individual case.

28. Facts Observations and Interpretations

Evidence level	What belongs here
Documented or directly measured	Laboratory-confirmed chikungunya; documented myocarditis; high PVC burden; 15 October 2021 procedural perforation, tamponade, sternotomy, bypass and repair; serial BP and HR readings; diagnosed gastrointestinal and urinary conditions; procedures and catheter events.
Lived longitudinal observations	Postural symptoms, fatigue, thermoregulatory failure, post-meal crashes, walking-cycling contrast, slowed healing, triggers, recovery intervals and changes in daily function.
Published working hypotheses	MSAD formulation; three-hit model; autonomic vulnerability as a disease-modifying substrate; systemic visceral dysautonomia; autonomic priming in EoE; changing autonomic reserve; possible autonomic contribution to visual dysfunction [20-29,35].
Not yet established	MSAD as a formal diagnosis; a single causal pathway; exact site of autonomic injury; classical baroreflex failure; cerebral hypoperfusion during individual episodes; autonomic causation of each organ or sensory disorder; confirmed SPC-caused dyssynergic defecation; a measured biomarker of autonomic or repair reserve.

29. Current Clinical Priorities

- Protect against falls and injury during delayed upright deterioration.
- Clarify the mechanism of marked blood-pressure variability and limited compensation using appropriate formal autonomic and haemodynamic assessment.
- Monitor persistent or recurrent haematuria and ensure that catheter position, infection, stones, granulation tissue and other urological causes are considered.
- Distinguish colonic delay from pelvic-floor outlet non-relaxation if difficult evacuation continues.
- Preserve conditioning through safe supported or recumbent exercise while upright activity is extended below the failure threshold.
- Review the interaction of medicines, volume status, meals, sleep, postoperative recovery and blood-pressure patterns.
- Treat recovery as a multidomain trajectory rather than judging it from one clinic reading or one organ system.

30. Questions I am Carrying Forward

- Do the dated records confirm that the intervals between recovery and the next downturn are becoming shorter?
- Are the successive organ problems independent conditions, or different expressions of a shared loss of autonomic and physiological reserve?
- Which part of the baroreflex or vascular response best explains the coexistence of severe orthostatic falls and hypertensive surges?
- Can upright cerebral blood flow be measured during the delayed walking-related failure?
- How much of the current urinary dysfunction is autonomic, obstructive, postoperative, catheter-related or mixed?
- Does objective anorectal testing show pelvic-floor dyssynergia or another outlet disorder?
- Does changing catheter position, balloon volume or bladder irritation alter the bowel outlet pattern?
- Which visual, hearing and taste changes track with autonomic state, and which have independent ophthalmic, audiological, oral, medication-related or nutritional causes?
- Which measures best show that my safe physiological

boundary is moving outward?

- What signs would distinguish a temporary loss of accessible reserve from a new medical problem requiring urgent investigation?

31. Literature Supporting the Framework

References 1-19 and 30-34 provide independent physiological and clinical context. References 20-29 and 35 document my published development and application of the unifying hypothesis. Publication supports scholarly plausibility and traceability; it does not establish MSAD as a recognised diagnostic entity, provide independent replication or prove the complete causal interpretation in this individual case.

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Authors Personal Blog

Closing reflection

I survived an event that required urgent and extraordinary medical care. Survival, however, was the beginning of another story: AF controlled, then PVCs; PVCs treated, then tamponade; cardiac survival followed by gallbladder, gastrointestinal, pancreatic and pyloric problems; G-POEM followed by clinically active EoE requiring Jorveza; urinary drainage restored, then a bowel outlet that would not relax.

Every correction has mattered. The pattern becomes visible only when the whole course is held together: each specialist may see one successful intervention or one organ; I have lived the increasingly short interval before the next downturn. The MSAD interpretation is that the corrected function ceases to be the principal bottleneck while shared regulatory vulnerability and local organ risks may remain.

The final word is still repair under pressure. MSAD is now a published and peer-reviewed hypothesis rather than an after-the-fact label. That makes it a coherent model worthy of testing; it does not prove every link or make every diagnosis autonomic. My task is to protect what has returned, measure the changing recovery interval, define the autonomic phenotype objectively, test competing explanations and keep recording the steps forward and back.

Medical and interpretive notice

This document is a personal working health narrative. It does not replace medical records, examination, diagnosis or treatment. Mechanistic explanations are presented as hypotheses unless clearly identified as documented findings. New or worsening bleeding, urinary obstruction, chest pain, fainting, neurological symptoms, infection signs or severe blood-pressure disturbance require appropriate clinical assessment.

Why I Kept Asking Questions After 2021

One step forward, two steps back—and the search for a framework that could hold the whole story

Before 2021, I believed I had a reasonable understanding of what had happened to my health.

I knew that I had been infected with chikungunya virus—CHIKV—and I knew from the medical literature that this virus could, in some people, have consequences extending beyond the initial infection. These could include cardiac involvement and disturbances of heart rhythm. When rhythm problems subsequently emerged, therefore, I was concerned, but not entirely surprised. There appeared to be a medically plausible line connecting the infection with at least some of what followed.

That understanding changed profoundly after the cardiac tamponade and the emergency surgery required to save my life.

I entered the procedure with a recognisable cardiac problem. I emerged into something much more difficult to describe: a body in which the usual systems of regulation no longer seemed to work together as they once had.

When I say that the “complete system broke down,” I do not mean that every organ failed simultaneously. I mean that the body’s

capacity to coordinate its different functions appeared to have been fundamentally disturbed. Problems no longer seemed to arise as isolated events. Cardiovascular regulation, blood-pressure control, digestion, temperature regulation, energy, vision, hearing, taste, and bladder and bowel function appeared to fluctuate, interact and, at times, deteriorate together.

That was the beginning of a much harder journey—not only medically, but cognitively and emotionally.

The need to understand

Over the past two years, recovery has rarely followed a straight line.

There have been genuine breakthroughs: procedures that helped, treatments that improved particular symptoms, periods of greater stability, and moments when I believed that I had finally turned a corner. Yet these improvements were often followed by setbacks or by the appearance of a different form of dysfunction.

It became a pattern of one step forward and two steps back.

A treatment might improve one part of the system while another problem emerged. A successful intervention could resolve an immediate obstruction or dysfunction without restoring the broader pattern of physiological regulation. At times, a setback appeared to erase months of progress. At other times, the improvement remained, but a new and seemingly unrelated problem took its place.

Emotionally, this was extraordinarily difficult. Hope repeatedly had to coexist with uncertainty. Relief could not be enjoyed without the fear that it might be temporary. Each new symptom created another question: Was this an independent condition, a complication of treatment, the consequence of surgery, a continuation of the earlier illness, or part of a broader regulatory disturbance?

I persisted in asking these questions because I needed more than a list of diagnoses. I needed a way of understanding why so many different problems had appeared within the same person and within the same evolving clinical history.

This was not an attempt to dismiss individual diagnoses or to claim that every problem had a single cause. Nor was it a rejection of the clinicians who treated particular organs and conditions. Many of those interventions were necessary, and some were life-changing. My difficulty was that the organ-by-organ explanations did not appear to capture the behaviour of the whole system over time.

From separate diagnoses to a unifying hypothesis

I could not find a description in the medical literature that adequately captured the complete scenario as I had experienced it: an initial infectious insult, subsequent cardiac involvement, a catastrophic procedural complication and major surgery, followed by a prolonged and fluctuating pattern of multi-system dysfunction. This led me to develop the term Multifactorial Secondary Autonomic Dysfunction, or MSAD.

Each part of that name matters.

“Multifactorial” recognises that there may not have been one solitary cause. The original infection, cardiac disease, cardiac procedures, tamponade, emergency surgery, cardiopulmonary bypass, medications, repeated physiological stress and the effects of ageing may all have contributed.

“Secondary” recognises that the autonomic dysfunction appeared within the context of acquired illnesses and medical events rather than necessarily representing a primary autonomic disorder.

“Autonomic dysfunction” identifies the possible common physiological terrain: disturbance of the systems responsible for regulating circulation, digestion, glandular secretion, temperature, pupillary adaptation, bladder and bowel function, and many other processes that normally occur without conscious control.

MSAD therefore became a unifying hypothesis and a possible disease-modifying substrate. It offered a framework for asking whether autonomic dysregulation might alter the onset, severity, expression or recovery of otherwise distinct conditions.

It did not make the individual diagnoses disappear. It provided a way of examining how they might be connected.

The physiological bridges

Over time, I explored possible physiological bridges between autonomic dysfunction and the various problems documented throughout my medical history.

These included gallbladder dysfunction, pancreatic insufficiency, pyloric dysfunction and the subsequent G-POEM procedure, eosinophilic oesophagitis, and disturbances involving bladder–bowel coordination. They also included changes that can be more easily overlooked: impaired night vision and visual adaptation, alterations in hearing, changes in taste, and other sensory or glandular complications.

The autonomic nervous system has physiological relationships with each of these areas. It contributes to gastrointestinal motility, sphincter behaviour, gallbladder contraction, pancreatic secretion, vascular regulation, pupillary responses, glandular function and bladder and bowel coordination. Autonomic pathways may also influence the environment in which inflammatory, sensory and metabolic processes are expressed.

These bridges make an association physiologically plausible. They do not prove that autonomic dysfunction caused each condition.

That distinction is essential. I am not claiming that MSAD has been demonstrated as the cause of my gallbladder disease, pancreatic problems, eosinophilic oesophagitis, sensory changes or every other complication. Some conditions may have separate causes. Others may have been modified, amplified or made more difficult to recover from by an underlying disturbance of autonomic regulation.

The hypothesis is that these events may share a regulatory context—not that every event can be reduced to one explanation.

From a private framework to a published hypothesis

MSAD did not remain only a private term that I used to make sense of my experience. The hypothesis and its associated physiological arguments have now been articulated through multiple peer-reviewed publications.

That provides a stronger scholarly basis for discussing the idea. It means that the hypothesis has been described, examined and placed within the published medical conversation. In the formal paper, the proposed physiological bridges are supported by references to the relevant clinical and physiological literature, including a dedicated consideration of sensory and glandular involvement.

Publication, however, is not the same as proof.

Several papers arising from the same longitudinal case can deepen

the analysis, improve the theoretical framework and demonstrate that the hypothesis has been subjected to peer review. They do not constitute independent replication. They remain different examinations of one person’s evolving clinical history.

I am one person. My experience cannot establish causality, determine prevalence or demonstrate that the same framework applies to other patients. A single longitudinal case cannot, by itself, create generalisable clinical knowledge.

It can, however, reveal a pattern. It can generate a hypothesis. It can identify questions that conventional diagnostic categories may not yet answer. It can also provide a sufficiently detailed account for other patients, clinicians and researchers to decide whether they recognise something similar.

Why I have continued

My persistence has partly been intellectual. When the available explanations did not encompass the entire course of events, I felt compelled to continue observing, documenting and searching for a more coherent interpretation.

But the motivation has also been deeply personal.

Living through repeated dysfunction without an adequate explanatory framework can be frightening and isolating. It is difficult to convey what it means to make progress and then lose it, or to recover from one serious problem only to confront another. The absence of a unifying explanation can make a person feel that each new symptom begins the story again from the beginning.

Developing the MSAD hypothesis gave me a language through which the story could be considered as a whole. It did not provide certainty, but it provided structure. It allowed me to distinguish more carefully between what I had observed, what established physiology might plausibly explain, and what remained hypothetical.

Most importantly, it allowed me to transform bewilderment into questions that could be examined by others.

I am not presenting this framework as a verdict. I am offering it as an invitation.

Perhaps MSAD will ultimately prove incomplete. Perhaps future evidence will separate conditions that I have tentatively brought together. Perhaps a different and better explanation will emerge. That is how scientific understanding develops.

But if other patients have experienced a similar sequence—an initial illness or major physiological insult followed by fluctuating, multi-system dysfunction—then this hypothesis may help those experiences become more visible and more systematically investigated.

That is why I have continued writing.

The journey since 2021 has been medical, cognitive and emotional. It has involved survival, uncertainty, disappointment, persistence and moments of genuine recovery. It has repeatedly followed the pattern of one step forward and two steps back.

I cannot prove that autonomic dysfunction connects every part of this history. I can only show why the connection is physiologically plausible, describe the evidence and limitations honestly, and place the hypothesis before others for consideration.

For one person, that may be the most responsible contribution possible: not to claim certainty, but to document the pattern

carefully enough that someone else may recognise it, test it and take the next step forward

Assistive Communication Disclosure

ChatGPT by OpenAI was used only to organise and refine the author's notes and prior writing, mitigating dyslexia-related

sequencing barriers and cognitive fatigue. The lived experience, facts, hypotheses, interpretations, judgements and conclusions are the author's own; he reviewed the document and accepts responsibility. AI did not generate research data, make clinical decisions or replace clinical or scholarly judgement; it served as a bridge for expression, not a replacement for authorship.

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