

Narrative Case Study

Advances in Neurology and Neuroscience

Longitudinal Health Symptom, Treatment and Control Summary (2008–2026) A Longitudinal Synthesis of Symptom Burden, Autonomic Linkage, Intervention, Recovery and Present Level of Control

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Purpose and Scope

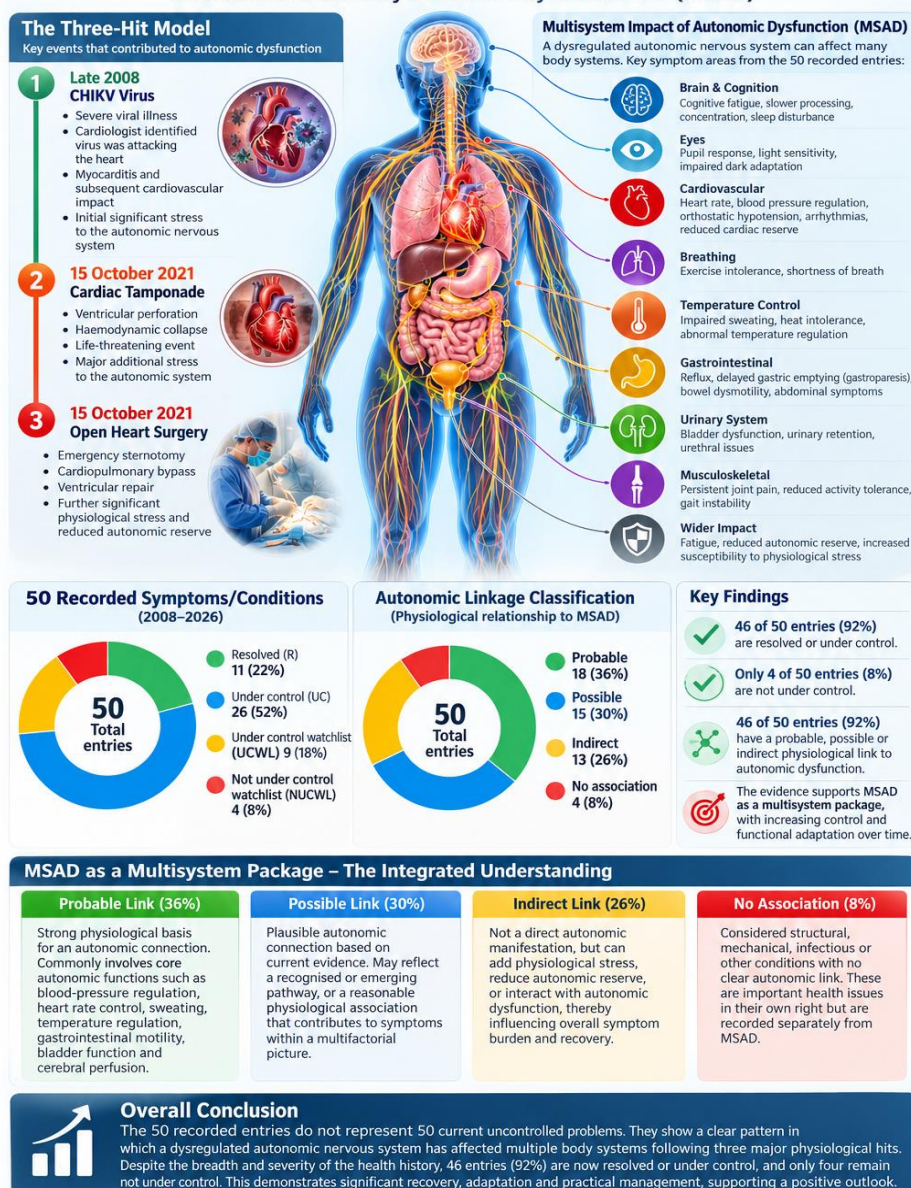
This document brings together 50 recorded health conditions or symptom complexes across the period 2008–2026. Its purpose is to show, in one longitudinal record, what occurred, how each problem may or may not relate physiologically to secondary autonomic dysfunction/MSAD, what treatment or management was used, what outcome followed, and the present level of control. The table is therefore both a chronology and a functional account of recovery, adaptation and continuing clinical burden. This is, fundamentally, a very personal medical log, arising from my own recordings, observations and continuing analysis of a complex health journey since 2008. The account has been developed in

dialogue with the medical team that has supported me, whose clinical assessments and care have helped validate its central observations, and it has also been tested against relevant findings reported in the medical literature. I wrote it primarily to help myself understand the sequence, interrelationships and changing significance of what has happened over nearly two decades. It is therefore offered with the understanding that it is personal, candid and deliberately honest—not as a substitute for individual medical advice, but as a carefully considered lived-experience record that may help others facing a similar situation gain a greater sense of confidence, context and understanding in making sense of their own journey.

My Health Journey and Autonomic Dysfunction

A 2008–2026 Longitudinal Summary of Symptoms, Treatment and Control

Understanding a complex multisystem condition through the lens of
Multifactorial Secondary Autonomic Dysautonomia (MSAD)



1. What has been done

- Each row is anchored to a period and a two- or three-word symptom summary, allowing the reader to identify the issue immediately before reading the detailed explanation.
- Column 2 records the condition or symptom complex and then, in *italics*, evaluates the MSAD/autonomic linkage using four mutually exclusive categories: **PROBABLE**, **POSSIBLE**, **INDIRECT** or **NO ASSOCIATION**. The colour-coded word in each row identifies the linkage category immediately.
- The linkage analysis is based on recognised physiology,

the documented timing and pattern of symptoms, and the cumulative autonomic-reserve framework developed through the author's MSAD work. It is intended to express biological plausibility, not to claim that every symptom was caused by dysautonomia.

- Key sources are identified within the cells and hyperlinked so that the underlying reference material can be opened directly.
- Columns 3–5 then show management, observed outcome and a current status classification: Resolved (R), Under Control (UC), Under Control Watchlist (UCWL), or Not Under Control Watchlist (NUCWL).

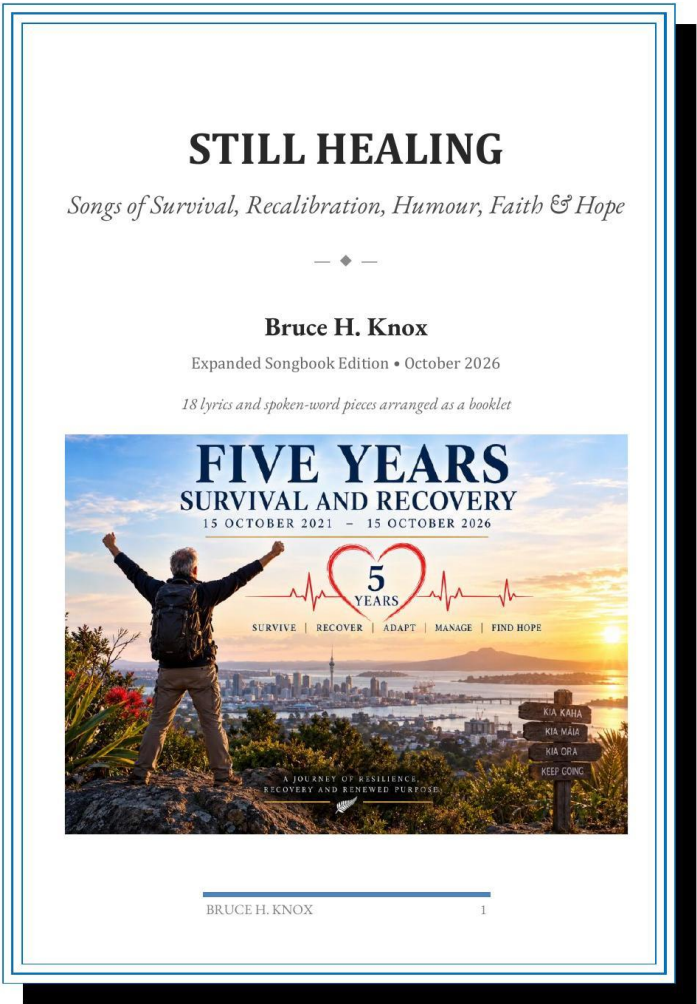
2. Current Position at a Glance

50	46	4	92%
recorded entries	resolved or under control	not under control	resolved or controlled

The statistical summary records 11 resolved entries (22%), 26 under control (52%), 9 under control but retained on the watchlist (18%), and only 4 not under control and remaining on the watchlist (8%). Thus, 46 of 50 entries (92%) are either resolved or presently controlled, although some continue to require active monitoring. UC and UCWL together account for 35 entries (70%) that remain under active management or control.

Principal conclusion: the record is not a picture of 50 current uncontrolled problems. It is a record of substantial cumulative illness followed by meaningful recovery, adaptation and management. Sufficient progress has been achieved for the great majority of recorded problems to be resolved or controlled; only four entries are currently classified as not under control.

A selection of musical presentation can be accessed in this summary findings below.
flipbook. These lyrics capture much of the story outlined in the



3. How the Table Should Be Read

Method, interpretive framework and meaning of the status categories

- Longitudinal Method**
The table uses a retrospective longitudinal synthesis. It follows the documented sequence of illness, procedures, symptoms, interventions and response over time rather than treating each problem as an isolated event. This makes it possible to distinguish conditions that preceded the major autonomic deterioration from symptoms that appeared, intensified or became more functionally important after the later cardiovascular events.
- Three-Hit Physiological Framework**
The table is interpreted against a three-hit working model:

Hit 1 is the late-2008 CHIKV illness, described in the clinical history at the time as the virus “attacking the heart”, followed by myocarditis and subsequent cardiovascular difficulties; Hit 2 is the ventricular perforation, cardiac tamponade and haemodynamic collapse during LVOT PVC ablation on 15 October 2021; Hit 3 is the same-day emergency sternotomy, cardiopulmonary bypass and ventricular repair. The model proposes that repeated viral-inflammatory, haemodynamic and major-surgical stress may have cumulatively reduced autonomic and physiological reserve. It is a biologically plausible explanatory framework, not proof of a single causal chain.

- How Autonomic Linkage is Expressed**

Autonomic linkage status	Meaning in this table
PROBABLE	Recognised autonomic mechanism with a strong fit to the documented pattern.
POSSIBLE	Biologically plausible relationship, but the symptom is not specific to autonomic dysfunction.
INDIRECT	The condition may amplify symptom burden, reduce cardiovascular or physiological reserve, or complicate autonomic compensation without itself being an autonomic manifestation.
NO ASSOCIATION	No direct autonomic association is identified; the condition is retained as a separate structural, inflammatory, mechanical or other diagnosis. Any indirect reserve effect is described separately where relevant.

- Meaning of the Current Status**

Code	Interpretation	Current total
R	Resolved	11 22%
UC	Under control	26 52%
UCWL	Under control, but retained on watchlist because significant care, specialist review or close monitoring remains necessary	9 18%
NUCWL	Not under control and retained on watchlist	4 8%

<i>PROBABLE</i> — 18 36%	<i>POSSIBLE</i> — 15 30%	<i>INDIRECT</i> — 13 26%	<i>NO ASSOCIATION</i> — 4 8%
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- Overall Interpretation: MSAD as a Multisystem Package**
Taken together, the linkage classifications show a coherent system-level pattern rather than a collection of 50 unrelated problems. Of the 50 recorded entries, **18 (36%) are PROBABLE, 15 (30%) are POSSIBLE, and 13 (26%) are INDIRECT. Only 4 (8%) are classified as NO ASSOCIATION.** Thus, 46 of 50 entries (92%) have at least a probable, possible or indirect physiological relationship to the wider autonomic picture, while four are best retained as separate diagnoses. This does not mean that MSAD caused all 46 conditions; rather, it shows that autonomic dysfunction provides a plausible integrating framework for understanding how many of the symptoms arose, interacted, were amplified, or became harder to compensate for. In this synthesis, MSAD is therefore best understood as a package: an umbrella description of an autonomic nervous system whose automatic regulatory functions became dysregulated across several domains at the same time. The table documents strong or plausible autonomic involvement in blood-pressure and baroreflex control, heart-rate and vascular responses

to standing and exertion, sweating and temperature regulation, gastrointestinal motility, bladder filling and emptying, pupillary responses, and the fatigue/cerebral-perfusion burden of upright activity. Other structural, inflammatory, cardiac or mechanical conditions sit around that core picture as indirect contributors because they can add physiological load, reduce reserve, or make autonomic compensation more difficult. The three-hit chronology offers a coherent temporal context for this cumulative loss of reserve, while remaining a working physiological model rather than proof of a single causal chain.

The positive conclusion is equally important. A dysregulated autonomic system is not necessarily a permanently failing system. Across this record there is substantial evidence of recovery, compensation, treatment response and learned management. Independently of the linkage classifications above, 46 of the 50 entries (92%) are now either resolved or under control, including those retained on the watchlist; only

four entries (8%) remain classified as not under control. Taken as a whole, the longitudinal pattern is therefore most consistent with multifactorial secondary dysautonomia that has been extensive but increasingly contained: the system remains vulnerable and imperfectly regulated, but the dominant direction is toward adaptation, restored function and practical control rather than relentless deterioration.

Important interpretive limit: this remains a longitudinal single-case synthesis. The linkage categories describe physiological plausibility and interaction, not proof that autonomic dysfunction caused every recorded condition. Structural, inflammatory, medication-related and other diagnoses remain identified separately where appropriate.

4. Longitudinal Health Symptom, Treatment and Control Summary (2008–2026)

Highlighted entries are those identified from the table wording as involving significant ongoing care, specialist review, or close monitoring.

Status: R = Resolved | UC = Under control | UCWL = Under Control Watchlist | NUCWL = Not Under Control Watchlist
Amber highlight = UCWL/NUCWL: [not] / under control, but

requiring significant ongoing care, specialist review, or close monitoring

Autonomic-linkage colour key used below: PROBABLE = recognised autonomic mechanism with a strong fit to the documented pattern; POSSIBLE = biologically plausible but not specific; INDIRECT = may amplify burden, reduce physiological reserve or modify autonomic compensation; NO ASSOCIATION = no direct autonomic association identified and the condition is retained separately. These linkage categories express physiological plausibility, not proof of causation, and are separate from the R/UC/UCWL/NUCWL clinical-control status in the final column.

Three-hit framework: Hit 1 = late-2008 CHIKV illness, described in this clinical history by the treating cardiologist as the virus 'attacking the heart', followed by myocarditis/cardiovascular difficulties; Hit 2 = ventricular perforation with cardiac tamponade and haemodynamic collapse during LVOT PVC ablation on 15 October 2021; Hit 3 = same-day emergency sternotomy, cardiopulmonary bypass and ventricular repair. The model proposes cumulative loss of autonomic/physiological reserve from repeated viral-inflammatory, haemodynamic and major-surgical stress. It is a working physiological hypothesis based on temporal sequence and biological plausibility, not proof of causation.

Period	Medical condition or symptom complex/how this is related to MSAD	Treatment or management approach	Outcome or level of control achieved	Status
2008 Urinary outlet obstruction	Prostate/urethral problems, later including urethral stricture and obstructed emptying <i>MSAD linkage (POSSIBLE): structural outlet disease is not autonomic, but bladder filling sensation, detrusor activity and coordinated emptying depend on autonomic pathways. In this history, urethral obstruction and possible autonomic bladder dysfunction can therefore coexist and compound incomplete emptying. Three-hit context: later autonomic bladder impairment may have been superimposed on pre-existing structural outlet disease, especially after Hits 2-3.</i> <i>Key source: National Institute of Diabetes and Digestive and Kidney Diseases, “Autonomic Neuropathy.”</i>	Prostate surgery in 2008; intermittent self-catheterisation; urethroplasty in 2019; another urethral procedure in November 2022	Structural repair and catheterisation provided management; later urinary emptying difficulties persisted.	UC
2008, late Acute CHIKV illness	Acute chikungunya—CHIKV: fever, rash, severe joint pain and profound fatigue. The clinical understanding at the time was that a virus was attacking your heart, with no clear prediction of which way the outcome would go. <i>MSAD linkage (POSSIBLE): major viral illness can be followed by persistent autonomic disturbance through post-viral inflammatory, immune and neural mechanisms. Within the cumulative-injury/autonomic-reserve framework developed in the MSAD work, CHIKV is therefore a biologically plausible early 'hit', although the later dysautonomia cannot be attributed to it alone. Three-hit context: this is Hit 1, the proposed initiating viral-inflammatory/cardiac stressor in the model.</i> <i>Key source: National Institute of Neurological Disorders and Stroke, “Postural Tachycardia Syndrome (POTS).”</i>	Hospital admission in April 2009 with critical-care monitoring and supportive management for the acute viral illness and cardiac involvement	You survived the acute illness. Persistent joint symptoms, fatigue and subsequent cardiovascular difficulties became part of the longer health journey.	R
2008 onwards Persistent joint pain	Persistent or recurrent joint pain following CHIKV <i>MSAD linkage (INDIRECT): persistent post-CHIKV joint pain is not itself an autonomic sign, but chronic pain and inflammatory stress can increase sympathetic load, reduce activity and deconditioning reserve, and consume physiological reserve. It may therefore amplify MSAD burden without being caused by MSAD. Three-hit context: persistent post-CHIKV pain added physiological load after Hit 1 and may have reduced later reserve.</i> <i>Key source: Blitshteyn, “Dysautonomia: a common comorbidity of systemic disease,” 2025.</i>	Ongoing symptom monitoring, pacing and activity adjustment during recovery from CHIKV	Persistent joint pain remained part of the longer recovery; pacing and activity adjustment supported day-to-day function, with the degree of resolution remaining variable.	UC

2009 <i>Myocarditis cardiac injury</i>	Myocarditis, diagnosed at North Shore Hospital <i>MSAD linkage (INDIRECT): myocarditis is a cardiac inflammatory injury rather than an autonomic diagnosis, but it can reduce cardiovascular reserve and increase the work required of autonomic compensation. In a cumulative-injury model it is a biologically plausible contributor to reduced reserve, not proof of autonomic causation. Three-hit context: post-Hit 1 myocarditis represents an early documented cardiac injury within the cumulative-reserve model.</i> <i>Key source: Blitshteyn, "Dysautonomia: a common comorbidity of systemic disease," 2025.</i>	Cardiology assessment, cardiac monitoring and continuing management following the myocarditis diagnosis	Cardiac function stabilised sufficiently for substantial professional activity to continue, although the myocarditis-specific contribution to later symptoms cannot be isolated.	UC
2009 <i>onwards</i> <i>Coronary artery disease</i>	Coronary artery disease requiring coronary stent placement <i>MSAD linkage (INDIRECT): coronary artery disease is a separate structural vascular disorder, but reduced cardiac/perfusion reserve and vasoactive treatment can materially limit the body's ability to compensate for standing and exertion. It can therefore magnify autonomic symptoms without being attributed to MSAD.</i> <i>Key source: Juraschek et al., AHA Scientific Statement on orthostatic hypotension in adults with hypertension.</i>	Cardiac stenting recorded in 2008 and March 2022; another account mentions 2010	Coronary stenting treated the identified arterial disease; symptom-specific benefit is difficult to separate from the broader cardiac history, and the procedural chronology varies between accounts.	UC
2009 <i>onwards</i> <i>Persistent hypertension</i>	Persistent hypertension, sometimes difficult to control <i>MSAD linkage (PROBABLE): blood-pressure stability depends heavily on baroreflex-mediated sympathetic and parasympathetic responses. Persistent hypertension alternating with orthostatic pressure falls is physiologically consistent with impaired baroreflex/autonomic regulation, while medication, renal and vascular factors may modify the pattern. Three-hit context: later worsening after Hits 2-3 is compatible with cumulative baroreflex/autonomic vulnerability, without proving a single cause.</i> <i>Key source: Juraschek et al., AHA Scientific Statement on orthostatic hypotension in adults with hypertension.</i>	Spironolactone introduced; renal ablation for BP control in October 2012; later reported medicines include losartan, spironolactone and Lasix	A measure of control achieved, but substantial BP variability continued. The contribution of each intervention is not separately recorded.	NUCWL
2009 <i>onwards</i> <i>Recurrent skin rash</i>	Recurrent cutaneous irritation and rash <i>MSAD linkage (INDIRECT): rash is not a recognised core manifestation of dysautonomia. Inflammatory or medication-related skin disease can, however, increase systemic stress and autonomic demand, so it may add to overall MSAD burden while retaining a separate dermatological explanation.</i> <i>Key source: Blitshteyn, "Dysautonomia: a common comorbidity of systemic disease," 2025.</i>	Identification and avoidance of suspected irritants or medication triggers, with skin-directed symptomatic care as required	Management centred on avoiding suspected triggers and treating irritation when present; the longer-term pattern of recurrence remains unclear.	UC
2011 <i>Atrial fibrillation</i>	Atrial fibrillation: abnormal heart rhythm <i>MSAD linkage (POSSIBLE): atrial fibrillation is an electrophysiological arrhythmia, but atrial rhythm is influenced by sympathetic-parasympathetic balance and autonomic reflexes. Autonomic dysregulation can therefore modify susceptibility or symptom expression, although AF itself does not establish MSAD. Three-hit context: AF followed Hit 1; the timing is compatible with altered cardiac-autonomic vulnerability but is not proof of causation.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	AF catheter ablation; Pradaxa also appears in your later medication history	AF brought under control and ceased to be the dominant rhythm problem. Anticoagulation served a separate protective purpose.	UC
2011 and later <i>Reflux oesophageal pain</i>	Reflux, oesophageal or chest discomfort, nocturnal throat/oesophageal burning and pain <i>MSAD linkage (POSSIBLE): vagal and enteric pathways coordinate oesophageal peristalsis, sphincter function and upper-GI motility. Reduced autonomic motor reserve can therefore worsen reflux, transit or swallowing symptoms, while EoE and other structural/inflammatory causes remain independently important.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	GERD assessment and treatment; gastroscopy, acid monitoring and barium swallow; later G-POEM and EoE-directed treatment	Reflux reportedly resolved after G-POEM; later inflammatory oesophageal symptoms required separate treatment.	R

2014 and March 2021 Severe hip disease	Severe hip joint disease requiring total hip replacement <i>MSAD linkage (INDIRECT): hip disease is structurally musculoskeletal, not autonomic. Pain, reduced mobility and deconditioning nevertheless reduce the reserve available for upright compensation and can amplify orthostatic intolerance, gait limitation and falls risk within a multifactorial MSAD picture.</i> <i>Key source: Fedorowski et al., “Orthostatic Hypotension: Management of a Complex, But Common, Medical Problem.”</i>	Total hip replacements	Both diseased hip joints were surgically replaced, providing structural treatment and supporting mobility; the degree of pain relief is not separately quantified.	R
2017 onwards; severe episodes in 2022 and 2026 Falls gait instability	Falls, near-falls, gait instability and fall-related injuries, including broken bones <i>MSAD linkage (PROBABLE): documented orthostatic hypotension can directly produce dizziness, visual narrowing, presyncope, instability and falls through reduced cerebral perfusion. Musculoskeletal, vestibular and balance factors may coexist, but the autonomic mechanism is physiologically strong where episodes track upright BP deterioration. Three-hit context: falls began before 2021, but more severe later episodes are compatible with amplification after Hits 2-3.</i> <i>Key source: Fedorowski et al., “Orthostatic Hypotension: Management of a Complex, But Common, Medical Problem.”</i>	Hospital assessment when required; walking stick; activity modification and management of postural symptoms	The walking stick, activity modification and postural management support safer participation, although gait instability and falls risk persist.	NUCWL
2018 and after procedures in 2021–2022 Recurrent urinary infections	Recurrent or severe urinary infections, antibiotic resistance and sepsis reported in earlier history <i>MSAD linkage (POSSIBLE): autonomic bladder dysfunction can impair sensation and emptying, leaving residual urine that increases infection risk. Urethral obstruction and catheterisation provide additional non-autonomic pathways, so recurrent infection is plausibly the downstream result of interacting autonomic and structural factors.</i> <i>Key source: NIDDK, “Autonomic Neuropathy.”</i>	Antibiotic treatment and hospital care	Antibiotics and hospital care treated acute episodes, but recurrent infection, resistance and the earlier sepsis history continued to complicate urinary management.	UCWL
2019–2021 Frequent PVCs	Frequent premature ventricular contractions, reaching approximately 15,000 in 24 hours <i>MSAD linkage (POSSIBLE): PVCs are an electrophysiological rhythm disorder rather than a diagnostic sign of dysautonomia, but ventricular excitability and symptom perception are influenced by sympathetic-parasympathetic balance. Autonomic instability can therefore interact with the arrhythmia and with BP/HR regulation without being its sole cause. Three-hit context: PVCs preceded Hits 2-3; their ablation precipitated Hit 2, followed immediately by Hit 3.</i> <i>Key source: NIDDK, “Autonomic Neuropathy.”</i>	LVOT PVC ablation on 15 October 2021	PVC problem treated, but the procedure caused ventricular perforation and tamponade.	R
2019 onwards; worse after 2021 Heat intolerance	Heat intolerance and difficulty regulating body temperature <i>MSAD linkage (PROBABLE): sweating, cutaneous vasodilation/vasoconstriction and heat dissipation are autonomically regulated. Heat intolerance and impaired temperature control therefore have a direct physiological route from autonomic dysfunction, although medication, cardiac status and deconditioning can add to the burden. Three-hit context: pre-2021 heat intolerance worsening after Hits 2-3 supports amplification of an existing autonomic vulnerability.</i> <i>Key source: NIDDK, “Autonomic Neuropathy.”</i>	Activity and environmental adaptation; pacing	Temperature regulation became more dependable during recovery, although heat remains a stressor.	UC
2020 onwards; pronounced after 2021 Orthostatic hypotension	Orthostatic hypotension: dizziness, light-headedness, presyncope and occasional syncope on standing <i>MSAD linkage (PROBABLE): orthostatic hypotension is a cardinal manifestation of impaired autonomic cardiovascular compensation. Inadequate vasoconstriction and/or heart-rate adaptation on standing can reduce venous return and cerebral perfusion, producing the documented dizziness, presyncope and occasional syncope; medication and volume status may add secondary effects. Three-hit context: orthostatic symptoms pre-dated 2021 but became pronounced after Hits 2-3, strongly supporting a cumulative-reserve interpretation.</i> <i>Key source: Juraschek et al., AHA Scientific Statement on orthostatic hypotension in adults with hypertension.</i>	Fluids and salt as clinically directed; compression garments and abdominal support; positional adaptation; head-of-bed elevation; pacing and BP monitoring	Greater practical control and ability to function; postural pressure falls remain a major limitation.	NUCWL

2020–2021; <i>Gallbladder dysfunction</i>	Gallbladder dysfunction <i>MSAD linkage (POSSIBLE): gallbladder emptying is influenced by vagal, sympathetic and enteric-neurohormonal control, so autonomic dysfunction can plausibly modify biliary motility. Gallbladder disease, however, has many independent causes and should not be attributed to MSAD without specific evidence.</i> <i>Key source: Blitshteyn, “Dysautonomia: a common comorbidity of systemic disease,” 2025.</i>	Cholecystectomy reported in an earlier account	The gallbladder disorder was described as settled after cholecystectomy; the operation date differs between the available accounts.*	R
2020–21 <i>Biliary tract dysfunction</i>	Biliary tract dysfunction <i>MSAD linkage (POSSIBLE): biliary motility is partly under neural and neurohormonal control, and broader autonomic GI dysmotility can alter coordinated digestive function. This gives a biologically plausible connection, but biliary tract disease remains nonspecific and requires its own explanation.</i> <i>Key source: NIDDK, “Autonomic Neuropathy.”</i>	Clinical monitoring and evaluation of biliary symptoms, considered separately from the gallbladder disorder	Biliary symptoms remained under clinical evaluation and were considered separately from the gallbladder disorder; a distinct treatment response cannot be established from the available account.	UC
2021 onwards, <i>especially Impaired sweating</i>	Altered or impaired sweating <i>MSAD linkage (PROBABLE): sweat-gland output is controlled by autonomic sudomotor pathways. Altered or impaired sweating is therefore a direct physiological autonomic manifestation; later return of sweating is also consistent with selective recovery, compensation or recalibration of autonomic function. Three-hit context: the post-Hits 2-3 timing strengthens the fit with a broader multisystem autonomic deterioration.</i> <i>Key source: NIDDK, “Autonomic Neuropathy.”</i>	Temperature regulation, hydration, pacing and observation during autonomic recovery	Sweating returned and became more dependable.	R
2021 onwards; <i>described in 2026 Delayed pupillary response</i>	Delayed pupil response, light sensitivity and difficulty adapting between bright and dim conditions <i>MSAD linkage (PROBABLE): pupil size and light-dark adaptation depend on coordinated sympathetic and parasympathetic control. Delayed pupil response and impaired adaptation therefore have a direct autonomic pathway, although ocular or neurological causes should still be excluded. Three-hit context: this post-Hits 2-3 symptom fits the broader emergence of autonomic signs after the 2021 insults.</i> <i>Key source: NIDDK, “Autonomic Neuropathy.”</i>	Softer, predictable lighting and environmental adaptation	Provides practical accommodation; complete resolution not reported.	UC
2021 onwards; <i>continuing in 2026 Cognitive fatigue</i>	Cognitive fatigue, slower processing, difficulty finding words or making decisions under load <i>MSAD linkage (PROBABLE): cognitive slowing and fatigue can arise when orthostatic/autonomic failure reduces cerebral perfusion and increases the metabolic effort needed to maintain homeostasis. The close association with upright intolerance makes this physiologically plausible, while sleep, medication and other neurological factors remain possible contributors. Three-hit context: prominence after Hits 2-3 is consistent with reduced cerebral-perfusion/autonomic reserve in the post-2021 phase.</i> <i>Key source: NINDS, “Postural Tachycardia Syndrome (POTS).”</i>	Pacing cognitive work, rest and assistive communication, including LLM-supported writing	Sustained thinking, writing and professional work regained; processing speed and fatigue remain limiting at times.	UC
2021–2026, recurring <i>Trauma emotional exhaustion</i>	Trauma-related distress, fear, frustration, grief, emotional exhaustion and the burden of repeated setbacks <i>MSAD linkage (INDIRECT): emotional stress activates sympathetic, cardiovascular and endocrine stress pathways and can temporarily consume autonomic reserve. It can therefore amplify an established dysautonomia; this is a bidirectional physiological interaction and does not imply that the autonomic disorder is psychological in origin. Three-hit context: Hits 2-3 were themselves major physiological trauma; later emotional arousal can further tax autonomic reserve.</i> <i>Key source: Goldstein, “Linking the Extended Autonomic System with the Homeostat Theory,” 2024.</i>	Self-directed trauma processing through research, explanatory writing and musical expression, together with pacing and adjustment of daily demands	Research, writing and musical expression provided a framework for processing repeated setbacks, while pacing and reduced demands supported ongoing adjustment.	UC

15 October 2021 Cardiac tamponade	Cardiac tamponade and haemodynamic collapse following ventricular perforation MSAD linkage (POSSIBLE): haemodynamic collapse, emergency sternotomy and cardiopulmonary bypass constitute major cardiovascular, inflammatory and neural stress. Within the cumulative-injury/autonomic-reserve model, this event is a plausible major 'hit' capable of reducing autonomic reserve and preceding multisystem dysregulation, although causation cannot be proved from one case. Three-hit context: tamponade/haemodynamic collapse is Hit 2; the emergency sternotomy, CPB and ventricular repair constitute Hit 3. Key source: NINDS, “Postural Tachycardia Syndrome (POTS).”	Emergency sternotomy, cardiopulmonary bypass and ventricular repair	Life saved and immediate cardiac injury repaired. Recovery was followed by extensive multisystem difficulties.	R
October 2021 onwards Post-exertional fatigue	Severe fatigue, weakness, reduced endurance and slow recovery after exertion MSAD linkage (PROBABLE): exertion requires rapid autonomic adjustment of heart rate, vascular tone and cerebral perfusion. Blunted or delayed compensation can produce weakness, breathlessness, fatigue and prolonged recovery; cardiac recovery and deconditioning may add to, but do not exclude, this autonomic mechanism. Three-hit context: onset immediately after Hits 2-3 gives this fatigue/endurance syndrome a particularly strong temporal relationship. Key source: NINDS, “Postural Tachycardia Syndrome (POTS).”	Pacing, rest, protected exercise, recumbent cycling and treatment of individual cardiac, digestive and urinary problems	Meaningful recovery of activity and professional participation; fatigue and delayed recovery continue to fluctuate.	UC
2022–2026, especially BP regulation instability	Supine or resting hypertension alternating with low upright BP MSAD linkage (PROBABLE): the combination of supine or resting hypertension with low upright BP is a recognised pattern of impaired autonomic/baroreflex control. Failure to buffer pressure appropriately in opposite postures provides a strong physiological link, while cardiovascular disease and medication can further shape the observed values. Three-hit context: this post-Hits 2-3 BP pattern is a central expression of impaired baroreflex/autonomic reserve. Key source: <u>Juraschek et al., AHA Scientific Statement on orthostatic hypotension in adults with hypertension.</u>	Specialist-directed medication management, home BP readings, head-of-bed elevation and adjustment of daily activity	Partial control; the coexistence of high and low pressures remains difficult to manage.	NUCWL
2022–2023, especially Blunted heart-rate response	Inadequate heart-rate response to standing or exertion, with breathlessness and exercise intolerance MSAD linkage (PROBABLE): heart-rate adaptation to standing and exercise is partly autonomically mediated. A blunted chronotropic response with breathlessness and exercise intolerance therefore fits impaired autonomic cardiac regulation within the broader MSAD pattern. Three-hit context: post-Hits 2-3 chronotropic limitation is compatible with worsened autonomic cardiac responsiveness. Key source: <u>NIDDK, “Autonomic Neuropathy.”</u>	Cardiology review, heart-rate and symptom monitoring, graded activity within tolerance, and pacing to limit exertional breathlessness	Gradual improvement: exercise HR reportedly rose from rarely exceeding about 70 bpm to around 100 during walking and approximately 120 with greater effort by late 2025.	UC
2022–2024; later intermittent episodes Autonomic storms	Autonomic storms: abrupt widespread physiological disturbance and exhaustion MSAD linkage (POSSIBLE): abrupt multisystem episodes involving cardiovascular, thermoregulatory, gastrointestinal or exhaustion symptoms are physiologically compatible with unstable autonomic signalling and an exaggerated homeostatic stress response. 'Autonomic storm' remains a descriptive term rather than a separate diagnosis. Three-hit context: emergence after Hits 2-3 fits a temporarily unstable, low-reserve autonomic system. Key source: <u>Goldstein, “Linking the Extended Autonomic System with the Homeostat Theory,” 2024.</u>	Blood-pressure management, pacing, trigger reduction, self-monitoring and recovery during episodes	Storms ceased or markedly diminished during 2024–2026. Improvement cannot be attributed to one intervention alone.	UC
2022–2024, especially Delayed gastric emptying	Delayed gastric emptying/gastroparesis: early fullness, nausea, bloating and difficulty moving food through the stomach MSAD linkage (PROBABLE): gastric emptying depends substantially on vagal, enteric and pyloric coordination. Autonomic neuropathy can slow gastric transit and produce gastroparesis, making the documented delayed emptying a recognised GI-autonomic manifestation, while medication and local gastric factors may coexist. Three-hit context: clinically important gastroparesis emerged after Hits 2-3, matching the broader post-2021 GI-autonomic deterioration. Key source: <u>NIDDK, “Autonomic Neuropathy.”</u>	Gastrointestinal investigations; smaller meals; G-POEM in 2024 to address pyloric resistance	Improved gastric emptying and digestive function; meaningful improvement in quality of life.	UC
2022 diagnosis; treatment from 2023 Pancreatic insufficiency	Pancreatic exocrine insufficiency, impairing digestion MSAD linkage (INDIRECT): the exocrine pancreas is densely regulated by parasympathetic and sympathetic pathways, so autonomic dysfunction can plausibly alter digestive secretion and gut-pancreatic coordination. Established pancreatic exocrine insufficiency nevertheless requires its own pancreatic explanation; within MSAD it may further reduce nutritional and physiological reserve. Key source: <u>Bliishteyn, “Dysautonomia: a common comorbidity of systemic disease,” 2025.</u>	Creon with meals	Digestion improved and pancreatic insufficiency brought under practical control with ongoing enzyme support.	UC

2022 onwards Constipation evacuation difficulty	Constipation and difficult evacuation <i>MSAD linkage (PROBABLE): autonomic and enteric pathways regulate colonic propulsion, rectal sensation and internal-sphincter behaviour. Dysfunction can therefore contribute to constipation and difficult evacuation; pelvic-floor, medication and structural mechanisms may coexist, especially where outlet relaxation is impaired. Three-hit context: post-Hits 2-3 onset fits the wider post-2021 pattern of enteric/autonomic dysmotility.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Hydration, soluble fibre in earlier accounts, and Microlax when required	A measure of bowel control achieved; continued bowel management remained necessary.	UC
2022 onwards Dry eyes	Acute dry eyes <i>MSAD linkage (POSSIBLE): lacrimal secretion and ocular-surface homeostasis are influenced by autonomic parasympathetic and sympathetic pathways. Dry eyes can therefore form part of autonomic dysfunction, although local ocular-surface disease, medication and environmental factors are common alternative or additional causes. Three-hit context: post-Hits 2-3 timing is compatible with multisystem autonomic involvement but remains nonspecific.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Prescribed eye drops following University of Auckland Optometry Clinic assessment	Prescription eye drops provided a defined treatment pathway following optometry assessment; the extent and durability of symptom relief are not quantified.	UC
2022 Muscle dystonic episodes	Muscle tightening and dystonic episodes occurring with autonomic disturbance <i>MSAD linkage (POSSIBLE): dystonia or muscle tightening is not a core autonomic sign, but sympathetic surges, pain, altered perfusion and heightened physiological arousal can trigger or intensify abnormal muscle contraction. A primary neurological or musculoskeletal explanation must remain in consideration. Three-hit context: post-Hits 2-3 timing makes autonomic surges or altered perfusion plausible amplifiers, not proven causes.</i> <i>Key source: Goldstein, "Linking the Extended Autonomic System with the Homeostat Theory," 2024.</i>	Pacing, postural and autonomic symptom management, gentle mobility, and monitoring of muscle-tightening episodes	Pacing, gentle mobility and autonomic management provided a practical framework for episodes; their frequency, severity and response remain under observation.	UC
2023 Colonic polyps	Four colonic polyps identified during colonoscopic investigation <i>MSAD linkage (NO ASSOCIATION): colonic polyps are structural mucosal lesions and are not explained by autonomic dysfunction. Their importance is gastrointestinal surveillance rather than evidence for MSAD.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Colonoscopy and removal of four polyps	Polyps removed; a separate effect on bowel symptoms is not recorded.	R
2024, during oesophageal treatment Oesophageal medication irritation	Oesophageal irritation associated in your observations with taking Pradaxa <i>MSAD linkage (INDIRECT): medication-associated mucosal irritation is not autonomic, but impaired oesophageal motility or clearance could prolong local exposure and magnify symptoms. The observed relationship to Pradaxa remains clinical observation rather than proof of an autonomic cause. Three-hit context: post-Hits 2-3 dysmotility could plausibly have reduced oesophageal clearance and amplified a medication-related injury.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Taking the medicine with food, as you reported	Irritation reportedly settled. The proposed inflammatory mechanism remains an interpretation.	R
2024 Altered taste	Altered taste <i>MSAD linkage (INDIRECT): taste is not a defining autonomic function, but autonomic control of salivary secretion and oral perfusion can influence the environment needed for normal taste perception. Medication, oral and gastrointestinal causes remain more direct alternatives, so the MSAD connection is biologically plausible but weak.</i> <i>Key source: Blitshteyn, "Dysautonomia: a common comorbidity of systemic disease," 2025.</i>	Observation of taste changes, review of possible medication or gastrointestinal contributors, and dietary adaptation as needed	Taste changes were managed through observation, review of possible contributors and dietary adaptation; the subsequent course remains uncertain.	UC
2025 recurrences; clinically active after G-POEM Eosinophilic oesophagitis	Eosinophilic oesophagitis: swallowing difficulty, oesophageal discomfort and recurrent attacks <i>MSAD linkage (INDIRECT): EoE is an inflammatory oesophageal disease, but autonomic/enteric dysmotility can reduce oesophageal motor reserve while EoE reduces structural-mechanical reserve. The combined loss of reserve can plausibly lower the threshold for dysphagia or food-impaction symptoms without making MSAD the cause of EoE. Three-hit context: EoE remains independent, but post-Hits 2-3 dysmotility may have reduced oesophageal functional reserve.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Jorveza/ budesonide, followed at times by swallowed Flixotide/ fluticasone	Symptoms brought under control. Approximately eight weeks without treatment or flare were reported in 2026; later recurrence again required Jorveza.	UCWL

2025/26 <i>During recurrent EoE symptoms Suspected allergic trigger</i>	Attacks associated in your observations with dog exposure <i>MSAD linkage (INDIRECT): an environmental or allergic trigger is not an autonomic phenomenon, but inflammatory activation can increase sympathetic and homeostatic load and transiently reduce available physiological reserve. The dog-exposure association remains observational and does not establish either allergy or autonomic causation.</i> <i>Key source: Goldstein, "Linking the Extended Autonomic System with the Homeostat Theory," 2024.</i>	Grooming and bathing the dog; modifying close interaction	You reported elimination of that apparent trigger. This records your experience without establishing an allergy mechanism.	R
2026 <i>Post-meal exhaustion</i>	Post-meal exhaustion or "crash", particularly about two hours after dinner <i>MSAD linkage (PROBABLE): after a meal, blood is redistributed to the splanchnic circulation and healthy compensation requires increased vascular resistance and appropriate cardiac responses. When autonomic compensation is limited, this can produce postprandial hypotension, cerebral hypoperfusion and a delayed energy 'crash'; meal size and GI disease may intensify the effect. Three-hit context: this late post-Hits 2-3 symptom has a strong physiological route through impaired splanchnic vascular compensation.</i> <i>Key source: NINDS, "Postural Tachycardia Syndrome (POTS)."</i>	Smaller, more frequent meals; pacing around meals	Used to reduce the burden of eating; the evening crash remains a vulnerability. Its separate treatment response is not quantified.	UC
2026 <i>Blurred vision fatigue</i>	Fluctuating focus or blurred vision; visual fatigue during reading, screens and conversation <i>MSAD linkage (PROBABLE): blurred or narrowed vision can result from reduced cerebral and ocular perfusion during inadequate autonomic BP compensation. Refractive, ocular-surface and neurological causes remain possible, but the physiological link is strong when symptoms accompany upright pressure falls. Three-hit context: the post-Hits 2-3 timing is most meaningful when visual symptoms track upright BP/perfusion deterioration.</i> <i>Key source: NINDS, "Postural Tachycardia Syndrome (POTS)."</i>	Screen breaks, pacing visual work and managing lighting	Helps organise visual demands; symptoms remain variable.	UC
2026 <i>Visual motion sensitivity</i>	Visual motion sensitivity, disorientation in busy surroundings and night-vision difficulty <i>MSAD linkage (POSSIBLE): autonomic pupillary dysfunction can impair light-dark adaptation, while orthostatic cerebral hypoperfusion can worsen visual disorientation and motion sensitivity. Vestibular and ophthalmic disorders remain important alternative explanations, so the association is plausible rather than specific. Three-hit context: these later symptoms are compatible with post-Hits 2-3 pupillary and cerebral-perfusion vulnerability.</i> <i>Key source: NINDS, "Postural Tachycardia Syndrome (POTS)."</i>	Choosing environments carefully and adapting lighting	Practical management documented; specific improvement in night vision not recorded.	UC
2026 <i>Reduced hearing</i>	Reduced hearing acuity requiring audiological assessment <i>MSAD linkage (INDIRECT): hearing loss is not a recognised defining manifestation of MSAD and requires independent audiological assessment. Autonomic regulation of vascular tone and systemic BP can influence inner-ear perfusion, providing a biological route for interaction, but there is insufficient evidence to attribute this hearing loss to dysautonomia.</i> <i>Key source: Blitshteyn, "Dysautonomia: a common comorbidity of systemic disease," 2025.</i>	Audiology and hearing-aid care discussed in your history	Audiological review established a pathway for hearing-aid support; the fitting status and functional benefit are not yet clear from the available history.	UC
2026 <i>Reduced vocal endurance</i>	Reduced vocal strength and endurance during sustained conversation <i>MSAD linkage (INDIRECT): sustained speech depends on respiratory endurance, perfusion and energy reserve. Autonomic-related fatigue, breathlessness or reduced cardiovascular compensation can therefore limit vocal endurance, while laryngeal, pulmonary and age-related causes remain separate possibilities. Three-hit context: late post-Hits 2-3 voice fatigue may reflect reduced whole-body cardiovascular/autonomic reserve.</i> <i>Key source: NINDS, "Postural Tachycardia Syndrome (POTS)."</i>	Voice conservation, pacing of conversation, hydration and rest between periods of speaking	Voice conservation, hydration and paced conversation support continued communication, although vocal strength and endurance remain limited during sustained use.	UC
2026, especially <i>Delayed walking deterioration</i>	Delayed deterioration during walking: head pressure, light-headedness, visual narrowing and substantial BP falls after approximately 15–30 minutes or longer <i>MSAD linkage (PROBABLE): delayed deterioration during sustained upright walking, with head pressure, visual narrowing and measured BP falls, strongly fits failure of autonomic vascular compensation and progressive cerebral hypoperfusion. Relative preservation of recumbent cycling further supports an orthostatic rather than purely muscular exercise limitation. Three-hit context: this is a strong late post-Hits 2-3 expression of limited autonomic vascular reserve under sustained gravitational stress.</i> <i>Key source: Fedorowski et al., "Orthostatic Hypotension: Management of a Complex, But Common, Medical Problem."</i>	Compression, hydration and salt as directed; pacing; walking stick; supported or recumbent exercise	Recumbent cycling remains comparatively preserved. Sustained upright walking continues to be restricted.	UCWL

2026, <i>progressing</i> Urinary retention	Urinary hesitancy, incomplete emptying and retention <i>MSAD linkage (PROBABLE): bladder filling sensation, detrusor contraction, sphincter coordination and emptying are autonomically regulated. Neurogenic/autonomic dysfunction can therefore cause hesitancy and retention, while established urethral disease and medication provide concurrent structural or pharmacological contributors. Three-hit context: post-Hits 2-3 progression supports autonomic bladder impairment superimposed on longstanding structural urethral disease.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Tamsulosin, morning/ evening self- catheterisation, then suprapubic catheter—SPC	SPC restored dependable drainage and brought the immediate emptying problem under practical control.	UCWL
2026; after surgery/ injury Slow tissue healing	Slower healing and longer recovery from tissue injury <i>MSAD linkage (POSSIBLE): delayed healing is not a standard diagnostic feature of dysautonomia, but autonomic disturbance can plausibly affect regional perfusion, inflammatory signalling, stress responses and the physiological resources available for repair. The MSAD work therefore treats this as a hypothesis of reduced repair reserve, not established causation. Three-hit context: the cumulative Hits 1-3 underpin the reduced-repair-reserve hypothesis, while that relationship remains unproven.</i> <i>Key source: Goldstein, "Linking the Extended Autonomic System with the Homeostat Theory," 2024.</i>	Surgical repair where required, wound/catheter care, time and careful pacing	Healing continues, but more slowly. No specific treatment restoring repair speed is documented.	UCWL
6 September 2026 Accidental SPC loss	Accidental SPC loss after tubing was cut and the balloon deflated; tract subsequently closed <i>MSAD linkage (NO ASSOCIATION): accidental SPC loss and tract closure were mechanical complications. The autonomic relevance lies upstream: neurogenic or mixed-mechanism bladder dysfunction may have contributed to the need for catheter drainage, but it did not cause the tubing/tract event itself.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Attempted reinsertion and subsequent urological/catheter management	Initial reinsertion unsuccessful; later accounts describe functioning SPC drainage. Exact restoration procedure needs recording.	R
September 2026 Night sweating exhaustion	Generalised night sweating, exhaustion and a prolonged response following emotional triggers <i>MSAD linkage (PROBABLE): sweating is directly autonomically controlled and emotional triggers activate sympathetic stress pathways. Generalised sweating followed by prolonged exhaustion is therefore physiologically compatible with dysregulated autonomic arousal, although infection, endocrine and medication causes must remain excluded. Three-hit context: this late post-Hits 2-3 episode is consistent with prolonged recovery from sympathetic/sudomotor activation.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Recovery over several days; ongoing pacing and monitoring	Physical, cognitive and emotional function improved markedly by 14 September; subsequent triggers were better contained.	UC
September 2026 Urinary bleeding	Bright-red urinary bleeding, particularly as the bladder filled <i>MSAD linkage (NO ASSOCIATION): visible haematuria is not a recognised autonomic manifestation and requires independent urological assessment. Autonomic retention, bladder overdistension and catheter dependence may create the mechanical context in which bleeding occurs, but they should not be assumed to be the bleeding source.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Free drainage through the SPC leg bag; urological review and planned investigations	You observed urine clearing or bleeding reducing with continuous drainage. Persistent or recurrent bleeding was not fully resolved in the original account.	UCWL
September 2026 Bladder spasms leakage	Bladder spasms and urethral leakage/incontinence despite SPC <i>MSAD linkage (PROBABLE): autonomic bladder dysfunction can disturb storage pressure, detrusor activity and coordinated emptying, producing urgency, leakage or spasm-like symptoms. Catheter irritation, blockage or local bladder pathology can provoke the same symptoms, so autonomic and mechanical mechanisms can reasonably coexist. Three-hit context: post-Hits 2-3 autonomic bladder vulnerability and SPC irritation provide plausible simultaneous mechanisms.</i> <i>Key source: NIDDK, "Autonomic Neuropathy."</i>	Maintaining unobstructed SPC drainage, catheter review and urological assessment of bladder spasms and urethral leakage	Maintaining free SPC drainage supports bladder emptying, while spasms and urethral leakage remain active issues requiring catheter review and urological assessment.	UCWL

September 2026, after SPC placement Bowel outlet dysfunction	New bowel-outlet difficulty: urge preserved, stool apparently reaching the rectum, repeated attempts, incomplete emptying and a sense that the outlet would not relax <i>MSAD linkage (POSSIBLE): autonomic/enteric dysfunction can alter colonic propulsion, rectal sensation and internal-sphincter coordination, while non-relaxation of the external outlet points to pelvic-floor recruitment. The change after SPC placement supports a biologically plausible bladder-bowel interaction hypothesis, but the SPC-to-outlet pathway remains unproven. Three-hit context: this late post-Hits 2-3 problem arose after GI and bladder dysautonomia were already established, supporting systems interaction.</i> <i>Key source: NIDDK, “Autonomic Neuropathy.”</i>	Existing bowel support, including Microlax; investigation and development of a bladder–bowel hypothesis	Existing bowel support provided incomplete relief, and the new outlet difficulty remained under investigation; a relationship to SPC-related pelvic-floor recruitment remains a working hypothesis.	UCWL
September 2026 Incisional hernia bulge	Incisional hernia/bulge near the SPC site <i>MSAD linkage (NO ASSOCIATION): an incisional hernia is a structural surgical complication and should not be labelled an autonomic manifestation. Within the reduced-repair-reserve hypothesis, autonomic dysregulation could theoretically influence perfusion and recovery capacity, but this remains an indirect hypothesis rather than an explanation for the hernia itself. Three-hit context: the hernia is not caused by the model; any link is indirect through the reduced-repair-reserve hypothesis.</i> <i>Key source: Blitshteyn, “Dysautonomia: a common comorbidity of systemic disease,” 2025.</i>	Photographic monitoring, comparison over time and clinical assessment of the bulge near the SPC site	Photographic comparison and clinical assessment support monitoring of the bulge; the need for hernia-specific treatment remains to be determined.	UCWL

5. Statistical Summary of Recorded Health Entries

Measure	Number	% of all entries
Total entries	50	100.0%
Resolved (R)	11	22.0%
Under control (UC)	26	52.0%
Under Control Watchlist (UCWL)	9	18.0%
Not Under Control Watchlist (NUCWL)	4	8.0%
Interpretation: of 50 recorded entries, 11 are resolved (R: 22.0%), 26 are under control (UC: 52.0%), 9 are under control but remain on the watchlist (UCWL: 18.0%), and 4 are not under control and remain on the watchlist (NUCWL: 8.0%). These four categories are mutually exclusive and together account for 100% of entries. UC and UCWL together represent 35 of 50 entries (70.0%) that are currently controlled or actively managed.		

6. Additional Reading

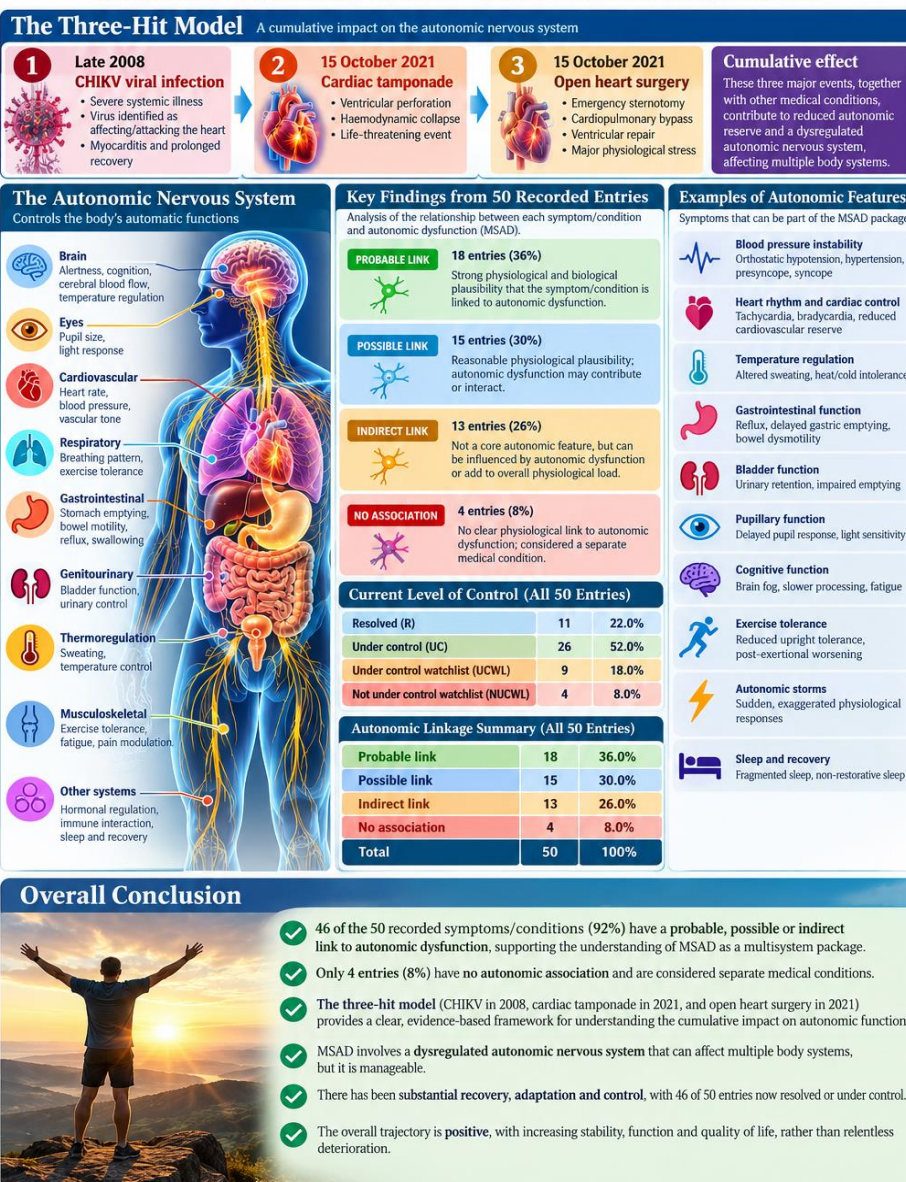
Issue(s) covered	Additional background reading
Urethral stricture, obstructed emptying, urinary retention	American Urological Association: Urethral Stricture Disease Guideline (2023 amendment) National Institute of Diabetes and Digestive and Kidney Diseases: Autonomic Neuropathy
Acute chikungunya and persistent post-CHIKV joint pain	Goupil and Mores: Review of chikungunya virus-induced arthralgia de Moraes et al.: Long-term arthralgia after chikungunya
Myocarditis and cardiac recovery	American College of Cardiology: Diagnosis and management of myocarditis American Heart Association: Myocarditis overview
Coronary artery disease, hypertension and orthostatic hypotension	Juraschek et al.: Orthostatic Hypotension in Adults With Hypertension—American Heart Association Scientific Statement Magkas et al.: Orthostatic hypotension—from pathophysiology to clinical applications
Atrial fibrillation and autonomic regulation	Vandenberk et al.: Autonomic nervous system in atrial fibrillation Qin et al.: Cardiac autonomic nervous system and atrial fibrillation
Reflux, oesophageal discomfort and swallowing symptoms	NIDDK: Autonomic Neuropathy—digestive-system manifestations

Hip disease, gait instability, falls and fall-related injury	New Zealand Falls Prevention Toolkit for Clinicians Detecting postural hypotension and falls risk
Urinary infections, catheter-associated infection and antibiotic resistance	EAUN: Catheter complications and catheter-associated urinary tract infection
Premature ventricular contractions and autonomic–arrhythmic interaction	Autonomic nervous system modulation in arrhythmias
Heat intolerance, sweating disturbance and temperature regulation	Mayo Clinic: Autonomic neuropathy symptoms and causes NIDDK: Autonomic Neuropathy—sweating and temperature regulation
Orthostatic hypotension, presyncope, syncope and delayed deterioration while walking	Merck Manual: Orthostatic Hypotension Torabi et al.: Classical and delayed orthostatic hypotension Cerebral blood flow in orthostatic intolerance
Gallbladder and biliary-tract dysfunction	Richmond: Biliary dyskinesia—controversies, diagnosis and management American College of Surgeons: Biliary dyskinesia management
Pupillary delay, light sensitivity and impaired dark adaptation	Mayo Clinic: Autonomic neuropathy and sluggish pupil response
Cognitive fatigue, slower processing, exercise intolerance and post-exertional weakness	Cerebral blood flow in orthostatic intolerance Merck Manual: Orthostatic intolerance symptoms and mechanisms
Emotional triggers, autonomic arousal and multisystem symptom exacerbation	Cardiovascular autonomic dysfunction and clinical guidance
Cardiac tamponade, ventricular perforation and complications of catheter ablation	American College of Cardiology: Procedure-related complications of catheter ablation Major complications after catheter ablation of cardiac arrhythmias
Supine hypertension alternating with low upright blood pressure	Hypertension and orthostatic hypotension: a challenging balance European hypertension guidance and cardiovascular autonomic dysfunction
Blunted heart-rate response to standing or exertion	Mayo Clinic: Autonomic neuropathy and exercise intolerance
Autonomic storms and abrupt widespread physiological disturbance	Autonomic dysfunction affecting several organ systems
Gastroparesis, delayed gastric emptying and G-POEM	American Gastroenterological Association: Management of gastroparesis AGA Clinical Practice Update on G-POEM
Pancreatic exocrine insufficiency and pancreatic enzyme replacement	AGA: Evaluation and management of exocrine pancreatic insufficiency
Constipation, difficult evacuation and bowel-outlet dysfunction	AGA: Evaluation and management of constipation AGA–ACG guideline: Pharmacological management of chronic idiopathic constipation
Dry eyes and autonomic–ocular-surface interaction	Kaido et al.: Autonomic nerve activity in dry eye with reduced tear stability Autonomic nervous activity and unstable tear-film dry eye
Muscle tightening and dystonic episodes	Multisystem manifestations of autonomic dysfunction
Colonic polyps and post-polypectomy surveillance	US Multi-Society Task Force: Follow-up after colonoscopy and polypectomy
Medication-associated oesophageal irritation	American College of Gastroenterology: Eosinophilic oesophagitis guideline summary
Altered taste	Mayo Clinic: Autonomic neuropathy symptoms and differential contributors
Eosinophilic oesophagitis, dysphagia and swallowed topical steroids	American College of Gastroenterology: Updated eosinophilic oesophagitis guideline ACG Clinical Guideline: Diagnosis and management of eosinophilic oesophagitis

Environmental or allergic triggers associated with oesophageal attacks	ACG: Eosinophilic oesophagitis diagnosis, treatment and monitoring
Post-meal exhaustion and postprandial orthostatic symptoms	Merck Manual: Meals as an aggravating factor in orthostatic hypotension
Blurred vision, visual narrowing, visual fatigue and cerebral hypoperfusion	Hypocapnia and cerebral hypoperfusion in orthostatic intolerance Cerebral blood flow in orthostatic intolerance
Visual motion sensitivity, disorientation and night-vision difficulty	Orthostatic intolerance and disequilibrium Mayo Clinic: Autonomic neuropathy and light–dark adaptation
Reduced hearing acuity and increased communication load	Autonomic dysfunction as a multisystem condition
Reduced vocal strength and conversational endurance	Merck Manual: Fatigue, breathlessness and orthostatic intolerance
Neurogenic or mixed-mechanism bladder dysfunction	NIDDK: Autonomic neuropathy and bladder dysfunction Managing a suprapubic catheter
Slow tissue healing and prolonged recovery	Mayo Clinic: Autonomic neuropathy, circulation and systemic contributors
SPC loss, tract closure and catheter replacement	British Association of Urological Surgeons: Suprapubic catheter practice guidelines Management after suprapubic catheter insertion
Night sweating and prolonged episodes after triggers	BMJ: Patients who report night sweats Persistent night sweats: Diagnostic evaluation
Visible urinary bleeding/haematuria	AUA/SUFU Guideline: Evaluation of haematuria
Bladder spasms, leakage and catheter complications	EAUN: Catheter complications Clinical guidance: Bladder spasm and suprapubic catheter management
Incisional hernia or bulge near the SPC site	European Hernia Society: Midline incisional hernia guidelines

Multifactorial Secondary Autonomic Dysfunction (MSAD) A Longitudinal Overview (2008 – 2026)

How three major health events, together with other medical conditions, can lead to a multisystem autonomic nervous system that is out of kilter – and how this has been managed over time.



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