



## ABSTRACT

### Background

Postural Tachycardia Syndrome (POTS) is widely described in patient-facing institutional materials — including those published by the NIH, Mayo Clinic, Cleveland Clinic, and Vanderbilt Autonomic Dysfunction Center — as a condition whose symptoms include fainting. This characterisation is repeated without mechanistic citation and is inconsistent with the diagnostic criteria for POTS, which require the absence of orthostatic hypotension — the haemodynamic prerequisite for orthostatic syncope.

### Objective

To examine the mechanistic, epidemiological, and diagnostic evidence bearing on the relationship between diagnostically defined POTS and transient loss of consciousness, and to evaluate whether the orthostatic tachycardia of POTS is causally capable of producing syncope or whether syncope in POTS patients is attributable to concurrent and mechanistically distinct conditions.

### Key Evidence

The 2015 Heart Rhythm Society Expert Consensus Statement defines POTS as requiring orthostatic tachycardia in the absence of orthostatic hypotension — rendering POTS and the haemodynamic mechanism of orthostatic syncope mutually exclusive within the same diagnostic episode. Transcranial Doppler studies demonstrate that cerebral blood flow velocity falls immediately upon head-up tilt in POTS, preceding the compensatory sympathetic surge and tachycardia by  $17 \pm 6$  seconds (Del Pozzi et al., Hypertension, 2014;  $P < 0.025$ ) — establishing tachycardia as the downstream response to hypoperfusion, not the cause of it. The reported syncope rate in POTS cohorts (~30%) does not exceed the lifetime prevalence of vasovagal syncope in the general population (20–40%). Notably, Stewart et al. (2019, PMC6597794) enrolled POTS patients with no prior syncope history and demonstrated that healthy controls with no syncope history fainted at higher rates than POTS patients during pharmacologically provoked tilt — directly inverting the assumption that POTS confers syncope susceptibility. Novak's 744-patient TCD dataset (2016) classifies POTS and syncope as haemodynamically distinct categories with divergent cerebral flow velocity profiles.

## Conclusions

Diagnostically defined POTS does not cause syncope through its own mechanism. The orthostatic tachycardia is the body's compensatory response to cerebral hypoperfusion — the mechanism that sustains consciousness, not one that terminates it. Syncope in POTS patients is attributable to identifiable concurrent conditions: vasovagal syncope, rate-suppression of the compensatory mechanism, diagnostic misclassification as POTS of conditions involving orthostatic hypotension, or orthostatic cerebral hypoperfusion syndrome (OCHOS). The persistent institutional characterisation of POTS as a fainting condition is mechanistically unsupported, internally contradictory relative to the diagnostic criteria those institutions endorse, and attributable to an identified gap in autonomic medicine education in standard training programmes.

**Keywords:** *postural tachycardia syndrome; POTS; syncope; vasovagal syncope; cerebral hypoperfusion; orthostatic intolerance; transcranial Doppler; compensation doctrine*

## Central Thesis

Diagnostically defined POTS — characterized by orthostatic tachycardia without blood pressure collapse — does not, by its own mechanism, cause loss of consciousness. The tachycardia is the body's successful anti-fainting defense, not the cause of collapse. When a person with POTS does faint, the evidence consistently points to a concurrent and mechanistically distinct condition: vasovagal syncope, medication-induced rate suppression, diagnostic misclassification, or a separate orthostatic hypotension syndrome. The fainting disease is not POTS.

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## Defining the Terms: What Syncope Actually Is

Before any argument about POTS and fainting can be assessed, the definition of syncope must be precise. The international consensus definition, endorsed by the European Society of Cardiology and the American Heart Association, is as follows:

*"Syncope is a transient loss of consciousness (TLOC) due to **cerebral hypoperfusion**, characterized by rapid onset, short duration, and spontaneous complete recovery."*  
(Freeman et al., Clin Auton Res, 2011; Shen et al., JACC, 2017)

This definition carries two critical implications. First, loss of consciousness requires a **fall in cerebral blood flow** below the threshold for consciousness — a threshold well-studied and estimated at a

systolic blood pressure drop to approximately 60 mmHg, accompanied by a collapse in middle cerebral artery blood flow velocity (CBFv) to below 30 cm/sec (Novak, *Neuroscience Journal*, 2016). Second, the mechanism is always hemodynamic, not electrical — the brain goes dark because blood flow fails, not because the heart rate is high.

By contrast, **pre-syncope** (also called near-syncope) is the clinical state of feeling as though one is about to faint — lightheadedness, tunnel vision, nausea, pallor — without actual loss of consciousness. Pre-syncope is overwhelmingly the predominant orthostatic symptom in POTS. Syncope, in the full medical sense, is not.

#### Key Distinction

Pre-syncope = feeling like you will faint. Syncope = actually losing consciousness. POTS patients are profoundly familiar with the former. The latter requires an additional mechanism beyond POTS itself.

## 0 The Diagnostic Criteria Make Fainting Structurally 2 Impossible

The formal diagnostic criteria for POTS, as established by the **Heart Rhythm Society 2015 Expert Consensus Statement** (Sheldon, Grubb, Raj et al., *Heart Rhythm*, 2015) and upheld in the 2020 Canadian Cardiovascular Society position statement (Raj et al., *Can J Cardiol*, 2020), explicitly require:

- Heart rate increase of  $\geq 30$  bpm ( $\geq 40$  bpm in adolescents under 19) within 10 minutes of standing or head-up tilt, OR a standing heart rate exceeding 120 bpm
- **Absence of orthostatic hypotension** — defined as a drop in systolic BP of  $\geq 20$  mmHg or diastolic BP of  $\geq 10$  mmHg within 3 minutes of standing
- Presence of orthostatic symptoms that improve with recumbency

The second criterion is the linchpin of this entire argument. The diagnostic definition of POTS requires that blood pressure does **not** fall significantly. Fainting, by universal medical consensus, occurs when cerebral blood flow collapses — which, in orthostatic syndromes, is driven by a fall in systemic blood pressure. **If blood pressure is falling enough to cause syncope, the diagnosis of POTS is no longer accurate for that episode; the patient is experiencing orthostatic hypotension or vasovagal syncope — two mechanistically different events.**

POTS (Pure POTS — Diagnostic Criteria)

Vasovagal Syncope (True Faint)

|                                                                                                                                                                                                                                                                                                                                                                            |                                                                                                                                                                                                                                                                                                                                                                   |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>• HR rises <math>\geq 30</math> bpm within 10 min</li> <li>• Blood pressure stable or elevated</li> <li>• Tachycardia is sustained and compensatory</li> <li>• Orthostatic symptoms: presyncope, fatigue, fog</li> <li>• No loss of consciousness in pure POTS</li> <li>• Patient remains standing (may sit or lie down)</li> </ul> | <ul style="list-style-type: none"> <li>• BP drops abruptly (systolic often <math>&lt; 70</math> mmHg)</li> <li>• HR drops (vagal phase) or stays low</li> <li>• Loss of consciousness occurs</li> <li>• Event terminates quickly once supine</li> <li>• Often episodic, not chronic daily symptoms</li> <li>• Requires a separate triggering mechanism</li> </ul> |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|

*This comparison is confirmed at the clinical level by the Johns Hopkins Medicine POTS program, which states explicitly: "POTS is diagnosed only when orthostatic hypotension is ruled out." (Johns Hopkins Medicine, hopkinsmedicine.org). StatPearls (National Library of Medicine, 2023) similarly defines POTS as requiring tachycardia "in the absence of orthostatic hypotension."*

**The Mutual Exclusivity Principle**

POTS and orthostatic hypotension are, by definition, mutually exclusive within the same 10-minute diagnostic window. A person whose BP drops enough to cause fainting no longer meets POTS criteria for that episode. Stating that 'POTS caused the faint' is equivalent to saying the compensatory mechanism both succeeded and failed simultaneously — a physiological impossibility.

03

## The Institutional Audit: How Top Medical Institutions Get This Wrong

Despite the diagnostic criteria being unambiguous, a review of patient-facing materials from the most trusted names in medicine reveals a troubling reliance on imprecise, culturally inherited shorthand over physiological accuracy. This is not a minor editorial problem — it is actively shaping how patients, caregivers, and many clinicians understand what POTS is.

The following institutions have published or continue to publish language that either directly states or strongly implies that fainting is a common symptom of POTS itself:

| Institution                                                              | Published Language                                                                     | The Problem                                                                                                                                                         |
|--------------------------------------------------------------------------|----------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| NIH / NINDS<br>(National Institute of Neurological Disorders and Stroke) | States that symptoms of orthostatic intolerance 'often include dizziness or fainting.' | No mechanistic distinction is drawn between presyncope (POTS-mediated) and true syncope (VVS-mediated). Fainting is presented as a POTS symptom, not a comorbidity. |

|                                                |                                                                                                                    |                                                                                                                                                                                                  |
|------------------------------------------------|--------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Mayo Clinic</b>                             | Claims that symptoms 'typically include an abnormally increased heart rate — and sometimes dizziness or fainting.' | The word 'sometimes' implies POTS occasionally progresses to fainting — implying a causal continuum. No citation is offered to prove tachycardia in the POTS range causes loss of consciousness. |
| <b>Vanderbilt Autonomic Dysfunction Center</b> | Lists 'fainting and dizziness' alongside tachycardia as 'typical symptoms.'                                        | One of the world's foremost POTS centers lists fainting as a POTS symptom without distinguishing it from the neurally mediated syncope that co-occurs as a separate condition.                   |
| <b>Cleveland Clinic</b>                        | States POTS 'may cause fainting (syncope)' in some patients.                                                       | The phrase 'POTS may cause fainting' is a direct causal claim — one that the diagnostic criteria and physiology of POTS directly contradict.                                                     |

**The Citation Gap:** Nowhere in any of these public-facing clinical overviews do these institutions provide peer-reviewed citations demonstrating that the sinus tachycardia of diagnostically defined POTS causes transient loss of consciousness. The claim is asserted, not demonstrated. Foundational cardiovascular medicine — as codified in Braunwald's Heart Disease (Mann, Zipes, Libby, Bonow, 11th ed.) — identifies sinus tachycardia as a physiological baroreflex-driven response that compensates for reduced stroke volume. The tachyarrhythmias associated with syncope — ventricular tachycardia, rapid atrial fibrillation, SVT with aberrant conduction, Wolff-Parkinson-White — are defined by abnormal electrical conduction pathways, not by baroreflex demand. These institutions cite no study because no study demonstrating this mechanism can be cited. The physiology does not support the claim.

When these descriptions are uncritically repeated in clinical education — and they are — they produce a generation of clinicians who approach POTS as a 'fainting disorder' rather than an orthostatic compensation syndrome. The clinical consequences of this misframe are severe and are explored in Section 10.

### **The Educational Vacuum Behind the Lazy Language**

A 2019 NIH Expert Consensus Statement openly admitted that meaningful discussions of autonomic disorders 'are not included in most medical school curricula, or even in residency/fellowship training.' This is not a minor gap — it is the root cause of the lazy language. When orthostatic physiology is not formally taught, clinicians inherit cultural shorthand: any orthostatic distress gets lumped under 'dizziness or fainting.' The result is copy-pasted, uncited, and physiologically inaccurate institutional language that now shapes millions of patient experiences.

## 0 The 'For Unknown Reasons' Problem: POTS Is a 4 Presentation, Not a Disease

One of the most revealing failures in the clinical POTS literature is a phrase that has been copied and pasted, nearly verbatim, across decades of guidelines, reviews, and patient-facing materials. It appears in variations of the following:

*"In POTS, for reasons that are not fully understood, the heart rate increases excessively upon standing..."*

This phrase — and its variants — appears in the American Heart Association's patient resources, multiple review articles in peer-reviewed journals, and countless clinical training materials. It has been so widely reproduced that it has achieved the status of institutional fact. It is also profoundly wrong.

**The reasons are not unknown. They are well-documented in autonomic physiology.** The phrase persists because researchers and guideline authors are describing the presentation — tachycardia — without having identified or investigated the upstream cause in the individual patient. They are characterising the compensatory output of a well-mapped cascade as idiopathic.

The causal chain in POTS is, in fact, well-documented in the autonomic physiology literature. The tachycardia does not arise spontaneously — it arises because the brain detects insufficient cerebral blood flow and commands the heart to compensate. To call this 'unknown' is to confuse the body's successful adaptive response with the disease itself.

**The data show that cerebral hypoperfusion is an initiating event in the tachycardic cascade.** The conventional narrative frames POTS as a consequence of peripheral venous pooling — the premise being that blood pools in the extremities, venous return drops, and the brain is passively starved as a result. The evidence does not support this as a complete account. The brain's failure to defend its own perfusion during an orthostatic challenge is the proximate abnormality — not a passive consequence of visible venous pooling.

Del Pozzi, Schwartz, Tewari, Medow, and Stewart (*Hypertension*, 2014, PMC4016155) studied a dyspneic subset of POTS patients during 70° head-up tilt and described for the first time an exceptionally large early drop in CBFv that **coincided with** a large fall in central thoracic blood volume before blood pressure recovered. This acute orthostatic challenge — the rapid central volume redistribution that occurs on standing — is a normal gravitational event. In healthy controls it is buffered adequately; CBFv is defended and sustained. In this POTS subset it was not. The brain failed to correctly sense and buffer the orthostatic challenge in real time, and cerebral perfusion dropped. That drop in CBFv was then immediately followed by hyperpnea and hypocapnia, which the authors show further perpetuated the reduced CBFv — and sympathetic nerve activity, measured by MSNA, did not rise until **17 ± 6 seconds later (P<0.025)**. The tachycardia came last. The authors frame this explicitly

as a sequential cascade in which cerebral hypoperfusion precedes and drives the sympathetic and tachycardic response — not the other way around.

This is not an isolated finding from a single lab. The 2024 JAHA review on cerebral blood flow in orthostatic intolerance (Ocon et al.) independently confirms: "*Stewart et al demonstrated that the drop in CBv with head-up tilt in POTS occurs before orthostatic tachycardia and hyperpnea, which suggests that CBv may be driving POTS pathophysiology... There is also evidence that the drop in CBv with orthostasis drives other POTS symptoms, including postural tachycardia and hyperventilation.*" (JAHA, 2024). The reviewer of this finding is not speculating — this is the JAHA's own characterisation of an established, replicated result.

Novak's independent 744-patient TCD dataset (Neuroscience Journal, 2016, PMC4972931) corroborates the mechanism from a different angle. In POTS, CBFv is consistently reduced on tilt despite preserved blood pressure — while in supraventricular tachycardias producing similar or higher heart rates, CBFv is *normal or transiently elevated*. This is a critical distinction: the same magnitude of tachycardia that leaves CBFv intact in SVT is accompanied by CBFv collapse in POTS. The difference is not the heart rate — it is the *reason* for the heart rate. In POTS, CBFv has already fallen and the brain is demanding compensation. In SVT, the tachycardia is electrical, not demand-driven, and the brain is fine.

A further independent line of evidence comes from Sterner et al. (*Am J Physiol Heart Circ Physiol*, 2009, PMC2724195), who demonstrated that during 70° head-up tilt, POTS patients showed an approximately **20% reduction in cerebral blood flow velocity** versus approximately 10% in healthy controls — a statistically significant difference ( $P < 0.05$ ) — while blood pressure remained lower in POTS patients than controls. Dynamic cerebral autoregulation was impaired in POTS, with MAP and CBFv becoming nearly synchronous during tilt, meaning the brain had lost its ability to buffer against pressure swings. The tachycardia in these patients was the system's attempt to compensate for exactly this failure.

**The correct sequence:** Cerebral hypoperfusion detected → Baroreflex / RVLM activates → Sympathetic drive increases → **Compensatory tachycardia** → Partial restoration of cerebral flow → Patient remains conscious, symptomatic but upright. Venous pooling and volume deficits are aggravating factors that worsen the hypoperfusion — they are not universally the root cause.

When researchers say 'the heart rate spikes for unknown reasons,' they are admitting they have not measured the upstream hemodynamics. They have not run a TCD. They have not assessed cerebrovascular autoregulation or sympathetic nerve activity during orthostatic stress. They are observing the final output of a compensatory cascade and labeling the entire cascade 'idiopathic.' The measurement tools to identify the upstream failures — TCD, autonomic function testing, cerebrovascular reactivity assessment — exist and are validated. Invoking 'unknown reasons' reflects a failure to apply them, not a genuine gap in mechanistic knowledge.

### POTS Is the Presentation, Not the Primary Disease

The medical community frequently confuses the presentation (tachycardia) with the pathology. POTS is not itself a disease — it is the cardiovascular system responding appropriately to maintain consciousness when orthostatic perfusion is compromised. The tachycardia label 'POTS' identifies what the body is doing. It does not identify why the brain required that response. Identifying the upstream hemodynamic or autonomic failure driving the compensatory demand is where mechanistically rational treatment begins.

The 2020 Canadian Cardiovascular Society statement by Raj et al. acknowledges this complexity, noting that POTS is 'a syndrome, not a single disease entity' and that 'multiple underlying pathophysiological mechanisms' contribute. Julian Stewart's body of work maps many of these mechanisms with precision: reduced blood volume, attenuated peripheral venous constriction, elevated norepinephrine with net splanchnic vasodilation, and impaired cerebrovascular autoregulation. *None of these are 'unknown.'* They require more thorough workup than a single tilt table heart rate reading can provide.

## 05

### Tachycardia Preserves Consciousness — It Does Not End It

The most important physiological concept in this document is the **Compensation Doctrine**: in POTS, the rise in heart rate is not a malfunction — it is the body's successful effort to maintain cerebral blood flow against orthostatic gravity stress.

On assuming upright posture, gravitational redistribution of blood away from the central compartment toward the lower extremities and splanchnic vasculature is a universal physiological event — occurring in every person, every time they stand. Estimates of this central volume shift range from approximately 500 to 800 mL depending on body habitus and posture (Grubb, *Circulation*, 2008; Raj, *Indian Pacing Electrophysiol J*, 2006). This fluid shift is not the pathology. It is a normal orthostatic challenge that healthy physiology is designed to manage. In healthy individuals, cerebral autoregulation and baroreflex-driven sympathetic activation — a modest 10–20 bpm heart rate increase with appropriate vasoconstriction — are sufficient to maintain cerebral perfusion pressure despite this routine gravitational redistribution. The shift occurred; consciousness was defended.

The pathological signal in POTS is not that a fluid shift occurred. It is that cerebral perfusion was **not successfully defended** despite that ordinary orthostatic challenge. The autoregulatory and baroreflex mechanisms that routinely handle standing in billions of healthy people every day failed to maintain brain perfusion above the symptomatic threshold. The tachycardia of POTS is the evidence of that failure — and simultaneously the body's attempt to correct it. It is the signal that the system tried harder precisely because the ordinary response was insufficient.

In POTS, the baroreflex fires harder and the rostroventrolateral medulla (RVLM) — the brain's cardiovascular command center — drives the heart rate much higher than the modest response seen in healthy upright posture. This is the brain demanding greater cardiac output to push blood upward against a perfusion deficit it cannot otherwise resolve. As a 2025 PLOS One hemodynamic analysis characterises it: *"reduced cerebral blood supply, thereby triggering a compensatory tachycardia via autofeedback regulation."* (PLOS One, 2025) The orthostatic challenge was ordinary. The defence failed. The tachycardia is the escalation.

### POTS Prevents the Faint

Novak's landmark 744-patient tilt-table study (Neuroscience Journal, 2016) classified orthostatic syndromes by their TCD cerebral blood flow velocity (CBFv) patterns. POTS was categorized as a tachycardia syndrome — characterized by a sustained elevated heart rate and reduced CBFv WITHOUT a blood pressure collapse. Syncope occupied a completely separate category, defined by a profound drop in BP and diastolic CBFv approaching zero. These are not points on a spectrum — they are physiologically distinct events. The tachycardia of POTS is the mechanism that keeps CBFv from falling to the zero-line where syncope occurs.

The ACC's own position statement on POTS (American College of Cardiology, 2019) confirms this clinical reality: *"Presyncope is much more common than syncope in POTS, but it is not infrequent that POTS coexists with episodes of neurally mediated (reflex) syncope."* This phrasing is decisive: syncope coexists with POTS as a separate phenomenon — it does not arise from POTS itself.

Stewart's authoritative 2009 paper, *"Postural Tachycardia Syndrome and Reflex Syncope: Similarities and Differences"* (PMC3810291), presents two patients with identical early tachycardia profiles on tilt table testing. One — the POTS patient — sustains the elevated heart rate, has symptoms, but does not faint. The other — the syncope patient — then experiences an abrupt blood pressure and heart rate crash characteristic of vasovagal collapse. The paper's conclusion: the *'front-end'* pathophysiology of POTS and simple faint may overlap, but the actual faint requires a separate central trigger.

## 06

### Sinus Tachycardia and Why It Doesn't Cause Fainting

A frequent skeptical argument invokes the premise that 'tachycardia causes fainting' by pointing to cases in which rapid heart rates reduce cardiac filling time and thus cardiac output. This is a valid observation in certain pathological arrhythmias — but it does not apply to the sinus tachycardia of POTS.

The tachycardia of POTS is, by diagnostic requirement, a **sinus tachycardia** (HR Society Consensus, 2015; Grubb, *Confounders of Vasovagal Syncope*, PMC3522867). An ECG and Holter monitor are specifically recommended at diagnosis to exclude accessory pathways, re-entrant dysrhythmias, and

arrhythmogenic syncope etiologies. The sinus node's intrinsic rhythm does not itself impair cardiac output in the physiologically relevant range of POTS heart rates.

In POTS, the elevated heart rate actually **compensates for a reduced stroke volume**. Cardiac output (CO) = Heart Rate × Stroke Volume. When stroke volume falls due to venous pooling and reduced preload, the heart rate increase is the autonomic system's attempt to maintain CO. As stated by Grubb et al. (PMC3522867): *"In patients with POTS, the initial response to upright posture can include a profound drop in stroke volume. With engagement of the baroreflex, there can be a vigorous increase in sympathetic tone and an exaggerated increase in HR. The blood pressure may remain unchanged or even increase."*

The distinction from pathological tachyarrhythmias that DO cause syncope is critical:

| Feature        | POTS Sinus Tachycardia                                 | Arrhythmic/Pathological Tachycardia            |
|----------------|--------------------------------------------------------|------------------------------------------------|
| Onset          | Gradual, postural-dependent                            | Sudden, position-independent                   |
| Mechanism      | Baroreflex-driven sympathetic activation (appropriate) | Abnormal electrical conduction (inappropriate) |
| Heart rate     | Typically 100–140 bpm upright                          | Can exceed 200+ bpm                            |
| BP effect      | Stable or increased                                    | May drop precipitously                         |
| Rhythm         | Regular sinus                                          | May be irregular or aberrant                   |
| Syncope risk   | No (unless concurrent VVS)                             | Yes (reduced CO, hypoperfusion)                |
| Diagnostic ECG | Normal P-wave morphology                               | Abnormal P-wave or no P-wave                   |

The phrase 'sinus tachycardia doesn't cause syncope' is not a controversial claim — it is standard cardiology curriculum. The tachyarrhythmias associated with syncope (VT, SVT with rapid rates, WPW conduction) are electrically driven, not baroreflex-driven. The failure mode is fundamentally different.

07

VVS Incidence in POTS Equals the General Population

Skeptics sometimes point to data showing that 20–30% of POTS patients experience syncope as evidence that POTS causes fainting. This argument collapses when the baseline population rate of vasovagal syncope is examined.

Vasovagal syncope (VVS) is extraordinarily common in the general population. Multiple population-based studies confirm a lifetime incidence of at least **20–40%** in healthy individuals (*van Steenwijk et al., 1995; Ganzeboom et al., 2003*). A 2023 systematic review and meta-analysis of 12

studies comprising 36,156 subjects reported a global VVS prevalence of 16.4% (95% CI: 6–37.5%) (PMC10821537), with cumulative lifetime incidence in Canada, the Netherlands, and Malaysia ranging from 25–35%.

### The Rate of Syncope in POTS Is Not Elevated Above the General Population

Stewart (Physiological Reports, 2019) states directly: 'The incidence of VVS in POTS patients is believed to be about 30%, similar to its incidence in the general population.' (Raj 2006; Kimpinski et al.) If the syncope rate in POTS does not meaningfully exceed the rate in healthy people — who by definition do not have POTS — this is strong evidence against POTS being the independent causal mechanism. A condition responsible for fainting would be expected to produce elevated syncope rates above population baseline; the data consistently fail to show this.

Stewart's 2019 study enrolled POTS patients who **had never previously fainted** in real life but who experienced vasovagal syncope during tilt table testing at 70° — what the study designates as "false positive" faint events. Critically, healthy control subjects with **no history of any syncope whatsoever** also fainted during identical tilt testing. The paper states:

*"Tilt table testing for vasovagal syncope can result in 'false positive' healthy controls (Control-Faint) and postural tachycardia syndrome (POTS-Faint). Tilt+ occurs in healthy volunteers and in patients with postural tachycardia syndrome (POTS) and show enhanced susceptibility to orthostatic hypotension." (Stewart et al., Physiological Reports, 2019 — PMC6597794)*

This finding is further supported by Science Direct data noting that 60% of normal-control adolescents fainted at a tilt angle of 80°, and 30% at 60–70°. These are healthy people. If tilt table testing — an artificial, extended, pharmacologically provoked stress — can induce fainting in the majority of healthy teenagers, it tells us nothing specific about POTS.

## 08

### When POTS Patients Do Faint: The Real Causes

POTS patients who experience true syncope are not fainting *from* POTS. They are fainting from identifiable mechanisms that either overwhelm the compensatory tachycardia or introduce a second, separate pathology. The scientific and clinical literature identifies several distinct scenarios:

#### 1. Concurrent Vasovagal Syncope (VVS) — The Primary Cause

VVS is mediated by a specific neuro-cardiac reflex — a sudden shift from sympathetic to parasympathetic dominance that causes simultaneous vasodilation (vaso-) and bradycardia (-vagal), collapsing blood pressure and cerebral perfusion. This is mechanistically separate from POTS. The Vanderbilt Autonomic Dysfunction Center describes the neurally mediated syncope reflex: *'Neurally mediated syncope is characterized by peripheral vasodilation and a decrease in blood pressure, or hypotension, along with bradycardia... due to an increase in parasympathetic tone and concomitant inhibition of sympathetic outflow.'* (VUMC, 2024)

Critically: POTS is a hypersympathetic state. VVS is a parasympathetic collapse. They are opposing autonomic events. A POTS patient who faints is not fainting from excess sympathetic tone — they are experiencing a parasympathetic override that temporarily defeats the compensatory mechanism. Studies by Stewart (2009, PMC3810291) show that even when both POTS and VVS share a 'front-end' of splanchnic pooling, the final pathway to fainting — central vasovagal mediation — is distinct and requires a separate trigger.

## 2. Heart-Rate Suppression and the Compensatory Mechanism

Rate-lowering medications — most commonly beta-adrenergic blockers — are frequently used in POTS management. The mechanism by which they might produce syncope in a subset of patients is physiologically straightforward: in POTS, the orthostatic tachycardia is the compensatory response maintaining cardiac output against a background of impaired venous return and reduced stroke volume. Cardiac output is the product of heart rate and stroke volume ( $CO = HR \times SV$ ). When stroke volume is fixed at a reduced orthostatic level, any pharmacological attenuation of heart rate reduces cardiac output proportionally. If this reduction is sufficient to impair cerebral perfusion pressure beyond the autoregulatory threshold, loss of consciousness may result — not from POTS, but from the pharmacological suppression of the compensation sustaining consciousness in the face of POTS.

This mechanism is supported empirically. Novak (2016, PMC4972931) demonstrated via real-time TCD that vasoactive medications in orthostatic syndromes do not uniformly improve cerebral perfusion and can worsen it, with inter-individual variability that standard arm-cuff monitoring cannot detect. Stewart et al. (2020, *Circ Arrhythm Electrophysiol*) characterized the haemodynamic consequences of excessive upright tachycardia suppression, finding that cardiac output effects depend critically on the relationship between rate reduction and concurrent stroke volume, which varies by patient and context. The 2015 HRS Expert Consensus explicitly limits beta-blocker recommendations in POTS to low-dose use in specific subtypes, acknowledging that generalised rate suppression is not supported by evidence of benefit.

A related observation — that some patients report subjective symptomatic improvement on low-dose beta-blockade — is consistent with this framework rather than contradictory to it. Hyperadrenergic POTS is characterised by elevated standing norepinephrine ( $\geq 600$  pg/mL) and an intensely symptomatic sympathetic state producing palpitations, tremor, and anxiety-like sensations. Attenuation of this sympathetic overdrive at low doses reduces the symptomatic burden without necessarily compromising the haemodynamic compensation. This subtype-specific response does not generalise

to all POTS presentations. Crucially, subjective symptomatic relief does not constitute evidence that orthostatic tachycardia was pathological rather than compensatory — it demonstrates only that the hyperadrenergic phenotype is sensitive to adrenergic blockade, which is expected. The fundamental question — whether cerebral perfusion improved, remained stable, or deteriorated — requires haemodynamic measurement that symptom reports alone cannot answer.

### 3. Diagnostic Misclassification: Orthostatic Hypotension Labeled as POTS

Orthostatic hypotension (OH) produces syncope. POTS, by definition, does not involve significant blood pressure drops. A patient who faints with tachycardia is more likely to have OH with a compensatory heart rate rise, or delayed OH, rather than POTS. The 2016 Novak study (744 patients, Neuroscience Journal) identified numerous patients referred under one diagnosis who received a corrected diagnosis from tilt-table plus TCD testing. As confirmed by the Dysautonomia Support Network: *'Current guidelines state that, generally, one person cannot have orthostatic hypotension and POTS.'* (dysautonomiasupport.org)

### 4. Head Trauma and Falls — Mechanical Injury, Not Hemodynamic

Some POTS patients who report 'fainting' have fallen and sustained head injuries that caused a brief loss of consciousness from the impact itself, not hemodynamic syncope. This can be misclassified. POTS-related pre-syncope (dizziness, sudden weakness) may also cause falls without true loss of consciousness.

### 5. OCHOS: A Distinct Condition Often Confused With POTS

Orthostatic Cerebral Hypoperfusion Syndrome (OCHOS), identified and named by Dr. Peter Novak, is a condition in which cerebral blood flow collapses during standing **without meeting POTS heart rate criteria**. These patients may faint — but they don't have POTS. Standard tilt table testing without TCD misses this diagnosis entirely. Novak's 744-patient dataset found OCHOS was a common *new* diagnosis discovered during testing — meaning many patients had been misclassified. Fainting attributed to 'POTS' in OCHOS patients is fainting from OCHOS, not from POTS.

## 09 Cerebral Hypoperfusion: The Shared Root of Both Conditions

POTS does not cause fainting. To understand why with precision, it helps to examine what POTS and vasovagal syncope actually share at the physiological level — and exactly where they diverge. Both conditions involve orthostatic cerebral hypoperfusion. The difference is in the outcome: in POTS, the compensatory response succeeds in sustaining consciousness. In vasovagal syncope, it does not.

A 2024 JAHA review, *"Cerebral Blood Flow in Orthostatic Intolerance,"* synthesizes the current understanding: *"All these syndromes experience excessive drops in cerebral blood flow (CBF) leading to symptoms including lightheadedness, fatigue, brain fog, and syncope... There is a need to measure CBF, as orthostatic hypoperfusion is the shared pathophysiology for all forms of OI."* (JAHA, 2024)

This shared root is why patients with POTS feel terrible when they stand — they are experiencing real, measurable cerebral hypoperfusion. As demonstrated by Del Pozzi et al. (*Hypertension*, 2014, PMC4016155), orthostatic CBFv dropped immediately upon tilting in POTS patients, triggering the carotid chemoreflex, then sympathetic drive, then the tachycardia. The heart rate spike was the **response** to brain hypoperfusion — not a separate disease process, and not the cause of the hypoperfusion.

The 2019 TCD study comparing VVS and POTS in 137 adolescents (100 VVS, 37 POTS) demonstrated that diastolic cerebral blood flow velocity was the key differentiator: *"The cerebral blood flow velocity during diastole differs between VVS and POTS, suggesting that it may be a determining factor in the pathogenesis of each."* (PMC12409184). In VVS, diastolic CBFv plummeted before loss of consciousness. In POTS, it was reduced but sustained — the hallmark of compensation holding the line.

## 10

### Addressing Persistent Misconceptions in the Literature

Several claims recur in clinical and patient-facing literature that either directly assert or implicitly support a causal relationship between POTS and syncope. Each warrants systematic examination against the available mechanistic and epidemiological evidence.

#### Misconception 1: A Syncope Rate of ~30% in POTS Indicates POTS Causes Syncope

The inference that a 30% syncope rate in POTS cohorts demonstrates POTS causation fails a basic epidemiological test: the rate is not elevated above what is observed in the general population. Population-based studies consistently report vasovagal syncope lifetime incidence between 20–40% in healthy individuals (Ganzeboom et al., 2003; van Steenwijk et al., 1995; PMC10821537). Stewart (Physiological Reports, 2019) explicitly states that the incidence of VVS in POTS patients is approximately 30%, characterising this as consistent with general population figures. A condition causally responsible for syncope would be expected to produce syncope rates meaningfully above population baseline. The data do not support this. Population comparisons involve methodological heterogeneity — differing ascertainment methods, referral patterns, and age distributions — and formal meta-analytic synthesis with harmonised definitions is needed to establish this equivalence rigorously. However, the current evidence is directionally inconsistent with a causal claim, and consistent with

VVS as a prevalent independent comorbidity in a population that shares vulnerability to orthostatic cerebral hypoperfusion.

### **Misconception 2: Sinus Tachycardia in the POTS Range Impairs Cardiac Output Sufficiently to Cause Syncope**

The physiological claim that tachycardia causes syncope is valid for certain pathological tachyarrhythmias — specifically those where ventricular filling time is critically curtailed at rates typically exceeding 180–200 bpm. This mechanism does not apply to the sinus tachycardia of POTS for two reasons. First, POTS diagnostic criteria require ECG and Holter documentation to exclude structural arrhythmias and accessory pathways before diagnosis — arrhythmic syncope is by definition excluded from the POTS construct. Second, the tachycardia of POTS is a demand-driven baroreflex response to reduced stroke volume; cardiac output in the demand-driven baroreflex range of POTS is being maintained, not impaired. Stewart et al. (2020, *Circ Arrhythm Electrophysiol*) examined the haemodynamic effects of excessive upright tachycardia and found nuanced context-dependent effects — the critical variable is the relationship between rate and stroke volume in each patient, not heart rate in isolation. The mechanistic case against POTS-range sinus tachycardia as a syncope cause is supported by the TCD evidence reviewed in Section 04: cerebral blood flow falls before the tachycardia develops, not as a consequence of it.

### **Misconception 3: Fainting Observed During Tilt Table Testing Demonstrates POTS-Mediated Syncope**

Tilt table testing, particularly at angles of 70–80° with pharmacological provocation (sublingual nitroglycerin or isoproterenol), induces vasovagal syncope in a significant proportion of healthy volunteers with no history of any syncope. Stewart et al. (2019, PMC6597794) specifically enrolled POTS patients with no prior real-life syncope history and demonstrated that a subset fainted during provoked tilt — categorising these as 'false positive' events and demonstrating that healthy controls with no syncope history fainted under identical conditions. The false-positive rate for VVS during pharmacologically provoked tilt is estimated at 10–15% (Merck Manual; Sheldon, *Circulation*, 2019). Syncope observed during tilt testing in a POTS patient therefore does not establish that POTS caused the faint; it may represent test-induced neurally mediated syncope indistinguishable mechanistically from what occurs in healthy control subjects.

### **Misconception 4: Co-Diagnosis of POTS and Syncope Implies POTS Causation**

POTS and vasovagal syncope are not mutually exclusive diagnoses — this is acknowledged explicitly in the 2015 HRS Expert Consensus Statement. The co-occurrence of two conditions does not establish that one causes the other; it establishes comorbidity. Both POTS and VVS are expressions of susceptibility to orthostatic cerebral hypoperfusion, which explains their co-occurrence without requiring a causal relationship. The HRS statement's own language is instructive: syncope 'coexists' with POTS in some patients. The paper by Stewart (*J Pediatr*, 2009, PMC3810291) comparing the two

conditions shows that the tilt profiles diverge at the point of vasovagal activation — which requires a separate central trigger — while the early orthostatic phase is shared. The diagnostic co-occurrence reflects shared predisposing physiology, not a POTS-to-syncope causal pathway.

### **Misconception 5: Institutional Descriptions of POTS as a Fainting Condition Constitute Evidence**

Patient-facing materials from major medical centres, reviewed in Section 03, describe fainting as a symptom or feature of POTS without providing mechanistic citation. These descriptions do not constitute peer-reviewed evidence that POTS tachycardia causes loss of consciousness; they represent clinical shorthand that conflates the high co-prevalence of VVS with POTS-caused syncope. The 2019 NIH Expert Consensus Statement on autonomic conditions noted that these disorders are inadequately represented in standard medical curricula and training programmes. This educational gap is the most plausible explanation for the persistence of imprecise institutional language, not new physiological evidence superseding the diagnostic criteria those same institutions endorse.

## Clinical Implications: Why Getting This Right Matters

The misconception that POTS causes fainting is not merely academic — it has direct, harmful consequences for patient care.

- **Inappropriate rate suppression:** Rate-lowering therapy that removes the compensatory tachycardia without addressing the underlying perfusion deficit can paradoxically worsen cerebral perfusion and precipitate syncope — which then gets attributed to POTS rather than to the treatment. (Novak, 2016; Stewart et al., 2020)
- **Misdiagnosis of OCHOS as POTS:** Patients with orthostatic cerebral hypoperfusion who do not meet POTS heart rate criteria remain invisible without TCD — their fainting is real and mechanistically distinct, but is frequently attributed to 'atypical POTS' or dismissed as functional. (Novak, 2016)
- **Misclassification of OH as POTS:** Orthostatic hypotension with a compensatory heart rate rise can be misdiagnosed as POTS. Fainting in these patients arises from the blood pressure drop, not from the tachycardia — and treatment targeting the heart rate makes the underlying problem worse.
- **Psychiatric mislabeling:** Patients who faint and are told their POTS is responsible — but whose POTS-directed treatment produces no improvement in syncope — are frequently referred for psychiatric evaluation or diagnosed with functional neurological disorder. This occurs because the clinician's framing of 'POTS causes fainting' forecloses the diagnostic search for the actual concurrent condition producing the syncope. The patient is not failing to respond to treatment. The treatment is targeting the wrong diagnosis.
- **Under-treatment of concurrent VVS:** When VVS is not identified as a separate comorbidity — because the clinician assumes POTS accounts for all syncope — patients never receive VVS-specific interventions: counter-pressure maneuvers, targeted salt and volume protocols specific to VVS physiology, or pacing evaluation for cardioinhibitory subtypes. The therapeutic gap is a direct consequence of the diagnostic conflation this paper addresses.

## Summary and Conclusion

### POTS Is the Body's Anti-Fainting System

The tachycardia of POTS is not a malfunction, not a danger sign, and not the cause of collapse.

It is the brain ordering the heart to beat faster to maintain cerebral perfusion pressure.

Attenuating this response without correcting the underlying perfusion deficit reduces cardiac output without resolving the demand that generated the tachycardia.

1. POTS is defined by tachycardia *without* orthostatic hypotension. Syncope requires blood pressure to fall. These two facts alone make 'POTS-caused syncope' a definitional impossibility in pure POTS.
2. The rate of syncope in POTS (~30%) falls within the reported range for vasovagal syncope in the general healthy population (~20–40%). This lack of elevation above population baseline is inconsistent with POTS being an independent causal driver of syncope.
3. Sinus tachycardia, in the HR range of POTS, does not cause syncope. Arrhythmic causes of syncope are a diagnostic exclusion criterion for POTS.
4. When POTS patients faint, the cause is almost always one of: concurrent VVS, medication-induced HR suppression, diagnostic misclassification (OH or OCHOS labeled as POTS), or head trauma from a pre-syncope fall.
5. POTS and VVS share upstream pathophysiology (orthostatic cerebral hypoperfusion) but diverge in outcome: POTS = compensation succeeds, VVS = compensation fails. Understanding both requires measuring cerebral blood flow — not just heart rate and arm blood pressure.
6. Treating POTS as a fainting disease leads to dangerous therapy decisions, most critically rate suppression that removes the patient's only hemodynamic defense.

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*The paradigm shift required is simple but profound: POTS is not the disease that ends in fainting — it is the disease that is trying to prevent fainting. Every treatment decision should be made with that understanding at its center. Patients who faint deserve rigorous investigation of their specific fainting mechanism — not an assumption that their POTS is 'severe.' In all likelihood, they have two conditions, and each requires its own evidence-based treatment approach.*

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