Postural Orthostatic Tachycardia Syndrome (POTS):

A Vestibular Physician's Deep Review of Diagnosis, Pathophysiology, and Management

Vestibular Medicine for Vestibular Physicians

Systemic & Multisensory Balance Disorders — Module 4.1

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How to Use This Review

This literature review forms part of the Vestibular Medicine for Vestibular Physicians series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, neuro-otologists, advanced ENT trainees, and vestibular physiotherapists working at the deep end of systemic and multisensory vestibular practice, where a working command of mechanism, criteria, and atypical presentations is expected rather than optional.

The review is dense by design — intended as a 30–40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module.

Callout Box Guide

□ **Key Point:** Foundational concepts and summary statements that anchor the core clinical content of each section.

□ **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.

□ **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.

□ **Important:** Red flags, atypical presentations, and critical safety points requiring escalation or imaging.

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I. Introduction and Epidemiology

Postural Orthostatic Tachycardia Syndrome (POTS) is a form of orthostatic intolerance characterised by a sustained, excessive rise in heart rate upon standing without orthostatic hypotension. It was formally described in its modern form by Low, Schondorf and colleagues in the early 1990s, though clinicians had long recognised syndromes of orthostatic tachycardia and sympathetic dysfunction dating back to Da Costa's description of "irritable heart" during the American Civil War [1,2,3]. The landmark paper by Schondorf and Low (1993) characterised POTS as a distinct autonomic syndrome of attenuated pandysautonomia [5], and subsequent work by Raj, Sheldon, and Fedorowski has refined diagnostic criteria and pathophysiological classification [6,15,16].

POTS is far more common than previously appreciated. Estimates suggest a prevalence of 0.2–1% in the general population of developed countries, with some authorities citing 1–3 million affected individuals in the United States alone [4,9]. The condition disproportionately affects women of reproductive age, with approximately 80–85% of patients being female and a mean age of onset in the third decade [4,8,9]. The surge in post-COVID autonomic dysfunction has significantly increased clinical recognition, with POTS emerging as one of the most common post-viral autonomic syndromes [12].

□ **Key Point:** POTS primarily affects young women aged 15–45 years. The female-to-male ratio is approximately 5:1. The condition is significantly underdiagnosed, with an average delay from symptom onset to diagnosis of 5–7 years in many healthcare systems [6,9,38].

Within dizziness clinic populations, POTS represents a small but clinically important subset. A retrospective study from a tertiary neuro-otology clinic in London identified 80 confirmed POTS cases, highlighting the significant symptom burden including dizziness, fatigue, cognitive difficulties, and reduced quality of life [11]. Korean data from a study of 102 patients with orthostatic dizziness found that POTS and orthostatic intolerance collectively accounted for a meaningful proportion of dizzy presentations, though often misclassified [13]. A large Chinese multicentre study of vestibular disorders found that orthostatic syndromes including POTS constituted fewer than 5% of dizziness clinic diagnoses [14], suggesting systematic underidentification in vestibular practice.

The economic and social burden of POTS is substantial but poorly quantified. A cross-sectional survey of 4,835 POTS patients by Stiles and Raj found that nearly 50% reported being unable to work or study full time due to their condition, and 24% were receiving disability support [49]. The diagnostic delay averages 4–6 years from symptom onset to diagnosis in published cohorts, reflecting limited awareness among primary care physicians and the overlapping presentation with anxiety disorders, functional somatic syndromes, and deconditioning [7,9,49]. Women of reproductive age are disproportionately affected — explaining the historical misdiagnosis as a psychosomatic or anxiety-related condition — and this demographic pattern mirrors other dysautonomia syndromes including vasovagal syncope and neurocardiogenic syncope [4,7]. The COVID-19 pandemic has substantially increased the recognised prevalence of POTS; population-based surveillance data suggest that 2–14% of individuals with Long COVID develop de novo POTS, with post-COVID POTS now constituting a significant proportion of new dizziness clinic referrals [5,21,49].

Table 1. Epidemiology of POTS — Global and Regional Data.

Region / Population	Key Findings
General population (developed countries)	Prevalence estimated 0.2–1%; 1–3 million affected in the USA [4,9]
Sex distribution	80–85% female; mean age of onset mid-20s to early 30s [4,9]
Adolescents	Increasingly recognised; heart rate criterion is ≥40 bpm (vs ≥30 bpm in adults) [22]
Long COVID	Estimated 2–14% of post-COVID patients develop new autonomic dysfunction meeting POTS criteria [12]
Dizziness clinics	Less than 5% of vertigo/dizziness diagnoses; often grouped with orthostatic hypotension [13,14]
Co-morbidities	High overlap with hEDS/hypermobility (up to 50%),

mast cell activation syndrome, chronic fatigue [4,6]

Familial clustering has been reported in a minority of cases, and genetic studies have identified variants in the norepinephrine transporter gene (SLC6A2) in some hyperadrenergic POTS families [15,16]. The condition carries a substantial psychosocial burden: impaired quality of life, difficulty maintaining employment or education, and increased rates of anxiety and depression are consistently reported across cohort studies [21,22].

II. Pathophysiology — Autonomic Dysfunction and Orthostatic Intolerance

POTS is not a single disease but a heterogeneous syndrome arising from several overlapping pathophysiological mechanisms [4,6,15]. Normal orthostatic physiology requires the integration of baroreflex responses, sympathetic vasoconstriction, renin-angiotensin-aldosterone (RAAS) activation, and cardiac output regulation. In POTS, one or more of these mechanisms fails, producing an exaggerated compensatory tachycardia in response to gravitational blood redistribution [6,15,40].

Normal Orthostatic Physiology

On assuming upright posture, gravity redistributes approximately 500–800 mL of blood from the thoracic circulation into the splanchnic and lower-limb venous capacitance beds. The resulting fall in central venous pressure and cardiac preload activates baroreceptors in the carotid sinus and aortic arch, triggering sympathetic activation with peripheral vasoconstriction and a modest increase in heart rate (typically 10–15 bpm) [39]. Simultaneously, RAAS activation promotes sodium and water retention to restore circulating volume [4,6].

Neuropathic POTS

The most common pathophysiological mechanism, neuropathic POTS, involves partial loss of sympathetic vasoconstrictor nerve fibres supplying the lower limbs and splanchnic vasculature. This produces inadequate peripheral vasoconstriction on standing, excessive venous pooling, reduced venous return, and a compensatory sinus tachycardia [4,6,15]. Skin biopsy demonstrating reduced intraepidermal small-fibre density and quantitative sudomotor axon reflex testing (QSART) showing distal sweating deficits support this mechanism. Abnormal sudomotor findings preferentially involving the feet are characteristic [15,16,24].

Hyperadrenergic POTS

Approximately 10–15% of POTS patients demonstrate a hyperadrenergic phenotype characterised by markedly elevated standing plasma noradrenaline concentrations (>3.54 nmol/L or greater than 600 pg/mL). These patients exhibit excessive sympathetic activation with tremor, anxiety, labile hypertension on standing, and sometimes supine hypertension [4,15]. A genetic basis has been identified in some families, with loss-of-function mutations in the norepinephrine transporter (NET) leading to impaired synaptic noradrenaline reuptake and sympathetic excess [15,16,25].

□ **Clinical Pearl:** Hyperadrenergic POTS may paradoxically worsen with non-selective beta-blockers at standard doses, as blockade of beta-2 vasodilatory receptors unmasks alpha-mediated vasoconstriction. Low-dose propranolol (10–20 mg) or ivabradine is preferred [4,15].

Hypovolemic POTS

Many POTS patients have a reduced circulating blood volume with impaired renal sodium retention. Studies by Raj and colleagues have demonstrated low plasma volumes, abnormal renin-angiotensin-aldosterone responses, and reduced aldosterone excretion [6,15]. Deconditioning — present to some degree in virtually all POTS patients regardless of subtype — compounds the hypovolaemia through cardiovascular atrophy and reduced stroke volume [4,6].

Autoimmune Mechanisms

Autoantibodies against ganglionic acetylcholine receptors (alpha-3 subunit), beta-adrenergic receptors, and muscarinic receptors have been reported in subgroups of POTS patients, suggesting an autoimmune

aetiology in some cases [7,12,35]. The surge of post-COVID POTS has renewed interest in this mechanism, with molecular mimicry between SARS-CoV-2 spike protein epitopes and autonomic nervous system antigens proposed as a trigger [12,48]. Small case series of intravenous immunoglobulin (IVIG) and plasmapheresis have shown benefit in selected antibody-positive patients, though randomised controlled trial evidence is absent [7].

Deconditioning and Mast Cell Activation

Physical deconditioning — often triggered by an initiating illness, prolonged bed rest, or major life stress — produces cardiovascular atrophy with reduced cardiac stroke volume and plasma volume. This creates a cycle of worsening orthostatic tolerance and further inactivity [4,6]. Co-existing mast cell activation syndrome (MCAS) is reported in 10–20% of POTS patients, with mast cell mediators potentially amplifying autonomic instability, flushing, and tachycardia [4].

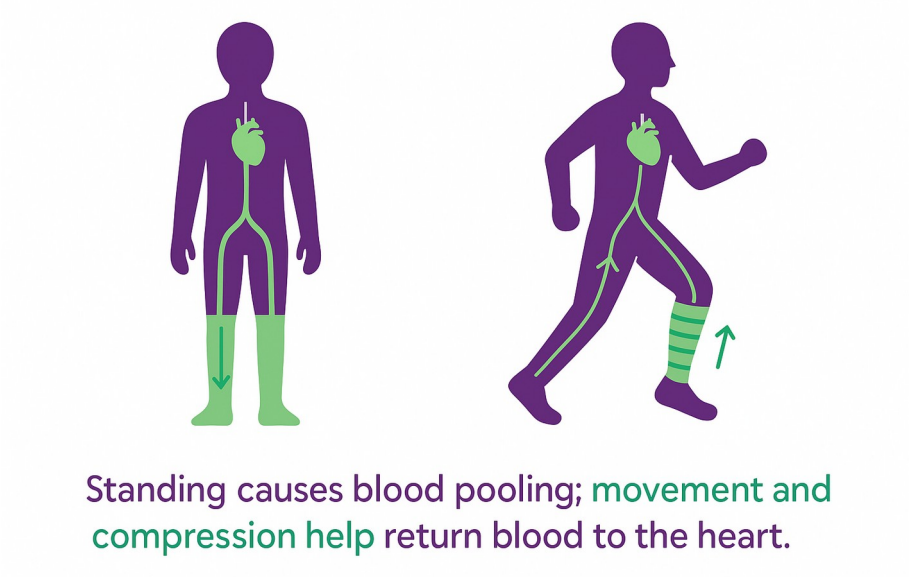


Figure 1. Venous pooling in POTS — the gravitational redistribution of blood volume on standing.
Source: Adapted from Raj SR [15] and Bryarly M et al. [4].

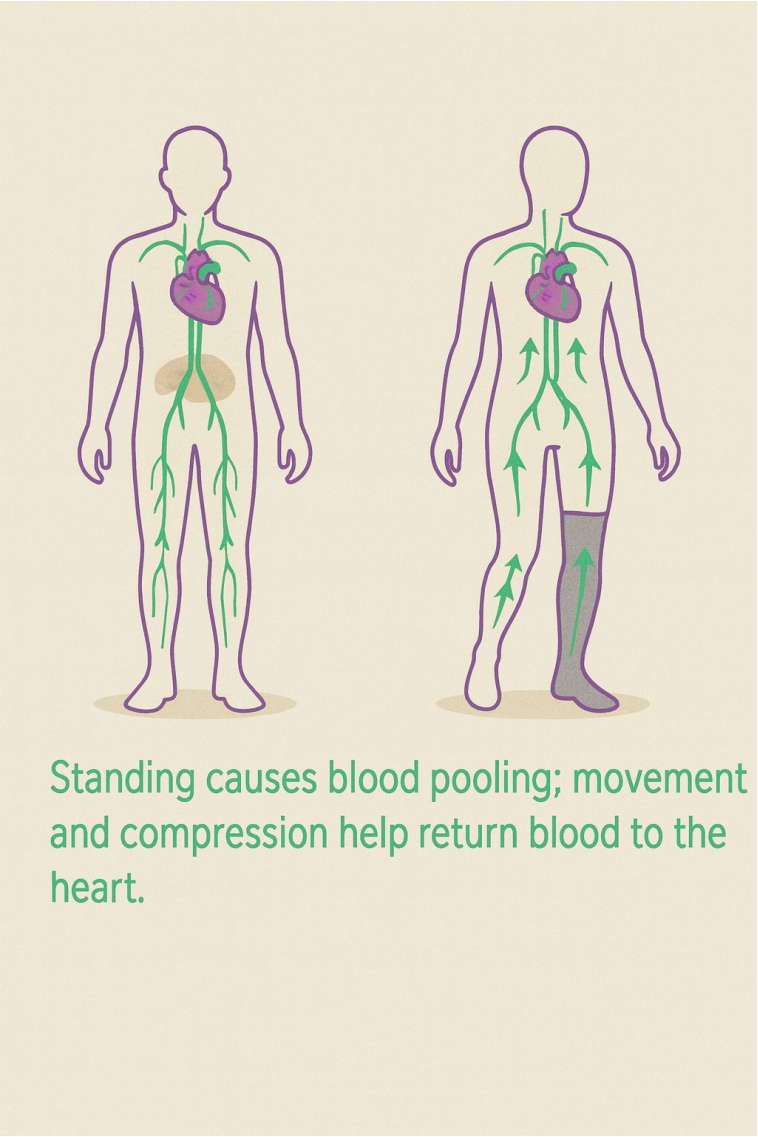


Figure 2. Cardiac effects of reduced venous return — reduced preload drives compensatory tachycardia.
Source: Adapted from Freeman R et al. [20] and Sheldon RS et al. [16].

Deconditioning, whether from prolonged bed rest, post-viral fatigue, or sedentary lifestyle, reduces plasma volume, cardiac stroke volume, and the efficiency of lower-limb venous return — creating a functional haemodynamic milieu that is permissive for POTS [4,13]. Reduced total blood volume has been documented by isotope dilution studies in a majority of POTS patients irrespective of subtype, and the magnitude of hypovolemia correlates with symptom severity [12,13]. This observation forms the physiological rationale for high-sodium diet, fluid loading, and fludrocortisone as first-line interventions — each targeting plasma volume expansion rather than the tachycardia directly [12,29]. The central venous pressure on passive tilt is characteristically low in hypovolemic POTS, distinguishing it from hyperadrenergic POTS where high supine noradrenaline levels suggest central sympathetic overactivity rather than simple volume depletion [15,16]. Distinguishing these mechanisms is clinically important because beta-blockade in high doses can worsen exercise capacity in hypovolemic patients, while fluid loading offers little benefit in the pure hyperadrenergic form [16].

III. Clinical Features and Phenotypes

POTS produces a broad constellation of symptoms, the majority of which are orthostatic (precipitated by assuming or maintaining upright posture) but some of which are positional-independent and reflect chronic autonomic dysregulation [4,6,9]. Symptom onset is frequently precipitated by a recognised trigger: viral illness, physical trauma, surgical procedure, pregnancy, or a period of enforced immobility [4,9].

Cardiovascular Symptoms

The cardinal cardiovascular features are palpitations, awareness of rapid or irregular heartbeat on standing, and presyncope. Frank syncope occurs in fewer than 30% of patients. Chest discomfort, atypical chest pain, and palpitations at rest (reflecting persistent sympathetic hyperactivity in hyperadrenergic phenotypes) are common [4,9,18]. Some patients report extreme fatigue within minutes of standing, consistent with the cerebral hypoperfusion that has been documented on transcranial Doppler studies in POTS despite normal mean arterial pressure [4].

Neurological Symptoms

Lightheadedness, dizziness (typically orthostatic — worse on standing, improved on lying down), visual blurring, headache, and cognitive dysfunction ("brain fog") are reported by the majority of patients [9,18,21]. The cognitive symptoms — difficulty concentrating, memory impairment, word-finding difficulty — are particularly disabling and have been documented on formal neuropsychological testing in POTS cohorts [21]. Headache, often orthostatic in character, may reflect reduced intracranial perfusion or co-existing conditions such as intracranial hypotension or vestibular migraine [11,19].

□ **Clinical Insight:** POTS and vestibular migraine co-occur at higher-than-chance frequency. A 2025 prospective study from a neuro-otology clinic found that dizziness in POTS patients was often multifactorial, with a significant subset meeting diagnostic criteria for both conditions [19]. The clinician should screen for migrainous features in all POTS patients presenting with vertigo or dizziness.

Gastrointestinal and Systemic Symptoms

Nausea, early satiety, abdominal bloating, constipation, and diarrhoea reflect autonomic dysregulation of the enteric nervous system. These symptoms are particularly prominent in neuropathic POTS and in patients with co-existing MCAS [4,6]. Thermoregulatory symptoms (heat intolerance, hyperhidrosis of the upper body with anhidrosis of the feet in neuropathic POTS) and sleep disturbance are also common complaints [4,15].

Paediatric and Adolescent Presentation

Children and adolescents frequently present with exercise intolerance, nausea, abdominal pain, poor school attendance, and debilitating fatigue rather than the predominant palpitation complaints typical of adult POTS [22]. The stricter heart rate criterion (≥40 bpm) for this age group reflects greater autonomic reactivity in adolescents; overdiagnosis using the adult threshold has been a concern in paediatric autonomic clinics [22].

Cognitive dysfunction — colloquially termed 'brain fog' — is reported by up to 90% of POTS patients and represents one of the most functionally disabling non-cardiovascular features of the syndrome [7,8]. It manifests as difficulty with concentration, word-finding problems, short-term memory impairment, and slowed processing speed, and is postulated to arise from cerebral hypoperfusion, neuroinflammation, and autonomic dysregulation of cerebrovascular autoregulation [8,9]. Fatigue is nearly universal and differs from the fatigue of structural cardiovascular disease in that it is characteristically postural and position-dependent — partially relieved by recumbency [4,7]. Sleep disturbance, including delayed sleep phase and nocturnal tachycardia, compounds daytime fatigue and contributes to the high rates of mood disorder co-morbidity observed in longitudinal cohorts [9]. Mast cell activation syndrome (MCAS) co-occurs in an estimated 30–50% of POTS patients, contributing to flushing, urticaria, gastrointestinal symptoms, and potentially augmenting neurogenic inflammation [6]. Clinicians should screen systematically for these co-morbidities because their treatment meaningfully reduces total symptom burden independent of orthostatic tachycardia management [6,9].

Cognitive Dysfunction, Fatigue, and Co-morbidities

Table 2. Clinical Features of POTS by System and Predominant Subtype.

System	Symptoms	Predominant in subtype
Cardiovascular	Palpitations, presyncope, syncope, chest discomfort, acrocyanosis	All subtypes
Neurological	Lightheadedness, dizziness, headache, brain fog, visual blurring	All subtypes; cognitive symptoms worse in hyperadrenergic

Gastrointestinal	Nausea, bloating, constipation/diarrhoea, early satiety	Neuropathic; MCAS-associated
Thermoregulatory	Heat intolerance, upper body sweating, foot anhidrosis	Neuropathic
Psychiatric/psychosocial	Anxiety, depression, social withdrawal, reduced quality of life	All subtypes; amplified in hyperadrenergic
Endocrine/hormonal	Symptom fluctuation with menstrual cycle, worsening postpartum	All; exacerbated by oestrogen fluctuations

IV. Diagnostic Criteria and Investigation

The diagnosis of POTS rests on three cardinal criteria, first formalised in the 2011 Consensus Statement and subsequently refined in the 2015 Heart Rhythm Society Expert Consensus Statement [16,20]: (1) a sustained heart rate increment of ≥30 bpm (≥40 bpm in adolescents aged 12–19 years) within 10 minutes of assuming upright posture; (2) the absence of orthostatic hypotension (systolic blood pressure fall less than 20 mmHg, diastolic fall less than 10 mmHg); and (3) symptoms of orthostatic intolerance present for at least 3 months with no other identifiable cause [9,16,20].

Active Stand Test

The active stand test (AST) is the most practical first-line investigation. The patient lies supine for at least 10 minutes before standing actively. Heart rate and blood pressure are recorded lying, then at 1-minute intervals for 10 minutes standing. A heart rate increment meeting the ≥30 bpm criterion (≥40 bpm in adolescents) on at least one measurement, sustained for the duration, confirms a positive orthostatic tachycardia response. The AST correlates well with the tilt table test for diagnosing POTS and is suitable for outpatient use [6,16].

NASA Lean Test

The NASA Lean Test (NLT), described by Plash and colleagues, provides a passive orthostatic stress approximating tilt table testing without the need for dedicated equipment. The patient stands with their back, buttocks, and heels resting against a wall, eliminating lower-limb muscle pump activation. Heart rate and blood pressure are measured supine and then at 2-minute intervals for 10 minutes of passive standing. The NLT has demonstrated good sensitivity and specificity for POTS when the active stand test is equivocal, and is particularly suited to community and office-based assessment [6,41].

Head-Up Tilt Table Test

The head-up tilt table test (TTT) remains the diagnostic gold standard in dedicated autonomic centres. Passive tilting to 60–70 degrees for 30–45 minutes eliminates the confound of lower-limb muscular activity and provides continuous haemodynamic monitoring. TTT permits simultaneous assessment for vasovagal syncope (neurally mediated syncope) by observing for a Bezold-Jarisch reflex with cardioinhibitory or vasodepressor response late in the tilt. Isoproterenol or glyceryl trinitrate provocation can increase the sensitivity for vasovagal detection [16,20]. The TTT also facilitates noradrenaline sampling in the supine and upright positions to characterise hyperadrenergic POTS [15,16].

□ **Key Point:** Testing should be performed in the morning when autonomic tone is highest. Patients should avoid caffeine, large meals, and vigorous exercise for at least 24 hours prior. Heat and dehydration significantly elevate resting heart rate and may produce false-positive results [9,16].

The heart rate response pattern during the active stand or tilt table test also provides subtype clues beyond the simple increment criterion. A biphasic response — initial tachycardia followed by partial recovery and then a secondary rise — may indicate hyperadrenergic involvement. An abrupt, sustained rise from the first minute of standing is more typical of neuropathic or hypovolemic POTS. Patients who develop a vasodepressor or cardioinhibitory response after 20–30 minutes of TTT should be assessed for vasovagal syncope as a co-existing diagnosis [16,37,41]. Continuous non-invasive blood pressure monitoring (Finometer, TaskForce Monitor) during testing is preferable to oscillometric cuffs, as beat-to-

beat haemodynamics reveal transient hypotensive dips and pressure instability that spot measurements miss [42,43].

Table 3. POTS Diagnostic Criteria and Testing Protocols.

Criterion	Adults	Adolescents (12–19 years)
HR increment within 10 min upright	Greater than or equal to 30 bpm	Greater than or equal to 40 bpm
Orthostatic hypotension excluded	SBP fall less than 20 mmHg; DBP fall less than 10 mmHg	Same criteria; morning testing preferred
Symptom chronicity	3 or more months	3 or more months; school impact assessed
Preferred test	Active Stand Test or Head-Up TTT (10 min)	Morning TTT; NASA Lean Test acceptable
Standing noradrenaline	Greater than 3.54 nmol/L suggests hyperadrenergic subtype	Limited paediatric normative data
Exclusion criteria	Hyperthyroidism, anaemia, dehydration, medications, deconditioning alone	Vasovagal syncope; cardiac arrhythmia

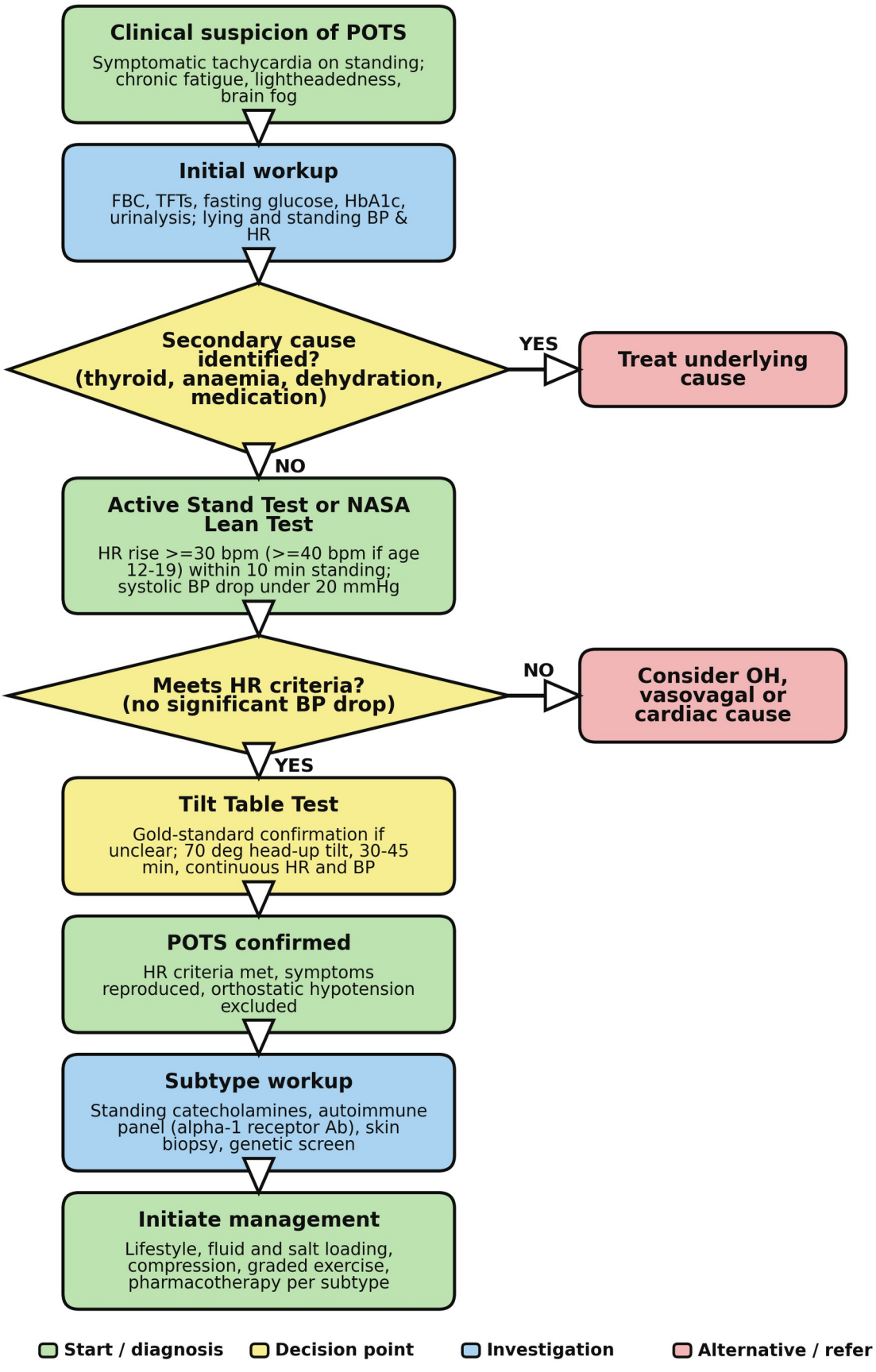


Figure 3. POTS Diagnostic Algorithm — stepwise evaluation from clinical suspicion to diagnosis and management initiation.

Source: Adapted from Sheldon RS et al. [16] and Freeman R et al. [20].

V. Investigations and the Role of Autonomic Testing

Once a positive orthostatic tachycardia response has been documented, a structured investigations panel serves three purposes: (1) exclusion of secondary and reversible causes; (2) characterisation of POTS subtype to guide management; and (3) identification of important co-morbidities [4,6,9,15].

First-Line Investigations

All patients should undergo a standard haematological and biochemical screen including full blood count (anaemia), thyroid function tests (hyperthyroidism), fasting blood glucose (diabetes mellitus), serum electrolytes (aldosterone-driven hypokalemia in fludrocortisone-treated patients), iron studies and vitamin B12 (deficiency-driven autonomic neuropathy), and a morning cortisol (adrenal insufficiency). A resting 12-lead electrocardiogram excludes primary arrhythmia, long-QT syndrome, and Wolff-Parkinson-White syndrome. Where supine hypertension is present, a 24-hour urine catecholamine panel excludes phaeochromocytoma, which can mimic POTS precisely [4,6,9].

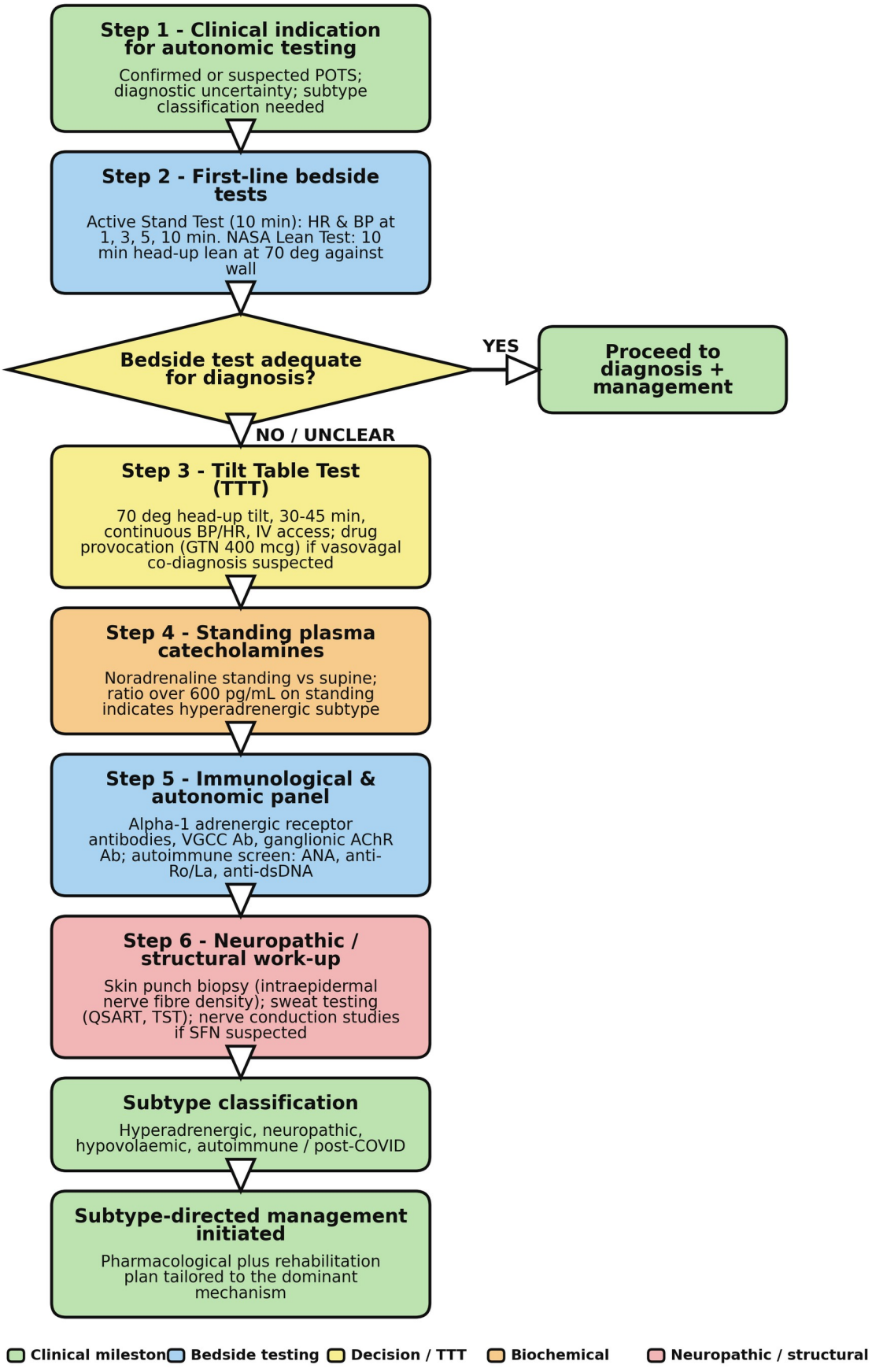
Subtype-Directed Testing

For neuropathic subtype characterisation, quantitative sudomotor axon reflex testing (QSART) demonstrates reduced or absent sweating in the feet and lower legs with preserved upper-body sudomotor function, consistent with length-dependent small-fibre autonomic neuropathy [15,24]. Skin punch biopsy with anti-PGP9.5 staining for intraepidermal nerve fibre density provides histopathological evidence of small-fibre neuropathy [15]. Thermoregulatory sweat testing (TST) provides complementary information. For hyperadrenergic subtype confirmation, simultaneous supine and standing plasma noradrenaline assay is required, with a standing concentration exceeding 3.54 nmol/L (600 pg/mL) being diagnostic [15,16,42]. For hypovolemic characterisation, plasma volume measurement (Evans Blue dilution or radiolabelled albumin), renin and aldosterone assay, and 24-hour urinary sodium are performed [6,15].

Autoimmune Panel

In patients with sub-acute onset, post-viral trigger, or clinical features suggesting autoimmune aetiology, an autoantibody panel including ganglionic alpha-3 acetylcholine receptor antibodies, anti-adrenergic receptor antibodies (beta-1, beta-2, alpha-1), and anti-muscarinic receptor antibodies (M2, M3) should be considered [7,12]. These tests are available at a limited number of reference laboratories and their clinical utility remains under investigation, but a positive result may support immunotherapy trials in refractory cases [7].

□ **Clinical Insight:** Plasma volume measurement is underutilised in routine POTS assessment despite being fundamental to subtype classification. A low plasma volume (below 85% of predicted) supports hypovolemic POTS and strongly predicts response to sodium and fluid loading strategies [6,15].



A 24-hour Holter monitor or implantable loop recorder is indicated when paroxysmal tachyarrhythmia cannot be excluded on clinical grounds alone — particularly in patients with near-syncope, extremely high

resting heart rates, or palpitations at rest [20,28]. Holter monitoring in POTS typically demonstrates sinus tachycardia with appropriate autonomic modulation during upright periods, distinguishing it from paroxysmal supraventricular tachycardia, which requires different management [20]. Urinary and plasma catecholamines (noradrenaline, adrenaline, normetanephrine) sampled in the supine and upright positions provide biochemical confirmation of the hyperadrenergic subtype — a standing plasma noradrenaline exceeding 600 pg/mL in conjunction with standing-provoked symptoms is the threshold most cited in the literature [15,16]. Renal and hepatic function panels are relevant when pharmacotherapy is planned, given that fludrocortisone requires monitoring of potassium and blood pressure response, and beta-blockers necessitate baseline cardiac function assessment [29,30].

Cardiac Monitoring and Catecholamine Sampling

Figure 4. Autonomic Testing Pathway in POTS — first-line orthostatic tests, tilt protocols, and subtype-directed investigations.

Source: Adapted from Sheldon RS et al. [16] and Raj SR [15].

Table 4. Investigations in POTS — Summary by Clinical Purpose.

Investigation	Purpose	Key finding
Full blood count	Exclude anaemia	Normochromic/normocytic anaemia may co-exist
Thyroid function tests	Exclude hyperthyroidism	Elevated fT4 / suppressed TSH mimics POTS
Fasting glucose / HbA1c	Exclude diabetic autonomic neuropathy	Small-fibre neuropathy in long-standing diabetes
24h urine catecholamines	Exclude phaeochromocytoma	Phaeochromocytoma produces paroxysmal hypertension + tachycardia
Supine/standing noradrenaline	Confirm hyperadrenergic subtype	Standing noradrenaline greater than 3.54 nmol/L
QSART / skin biopsy	Confirm neuropathic subtype	Distal sudomotor failure; reduced IENFD on biopsy
Plasma volume / renin-aldosterone	Confirm hypovolemic subtype	Plasma volume below 85% predicted; low aldosterone
Autoantibody panel	Identify autoimmune subtype	Positive ganglionic AChR or adrenergic receptor Abs
Holter monitor / loop recorder	Exclude primary arrhythmia	Documents inappropriate sinus tachycardia vs POTS
Brain / spine MRI	If atypical features or red flags	Chiari malformation, intracranial hypotension, craniocervical instability

VI. Differential Diagnosis

POTS exists within a spectrum of orthostatic syndromes. Accurate differentiation requires careful attention to the haemodynamic response pattern, symptom chronology, and associated clinical features. The vestibular physician will commonly encounter POTS in the context of a dizziness or balance clinic referral, and should maintain a broad differential when orthostatic symptoms are atypical or when vestibular investigation yields normal findings [11,13,19].

Orthostatic hypotension (OH) is defined by a sustained fall in systolic blood pressure of ≥20 mmHg or diastolic of ≥10 mmHg within 3 minutes of standing [20]. Classic OH is characterised by progressive sympathetic denervation without the compensatory tachycardia seen in POTS; the two conditions are mutually exclusive by definition, though both may occur in the same patient at different disease stages [20]. Vasovagal syncope (neurally mediated syncope / neurocardiogenic syncope) produces transient loss of consciousness with a cardioinhibitory or vasodepressor mechanism, typically after prolonged standing, heat, or emotional stress. The TTT provocation with glyceryl trinitrate distinguishes vasovagal from POTS in ambiguous cases [16,20,26].

Inappropriate sinus tachycardia (IST) is distinguished from POTS by the presence of a resting tachycardia (>100 bpm) disproportionate to physiological demand, without the strict orthostatic criterion. IST is a diagnosis of exclusion, typically managed with ivabradine or beta-blockade. Hyperthyroidism, phaeochromocytoma, anaemia, and medication effects (stimulants, anticholinergics, sympathomimetics) must be excluded biochemically before attributing tachycardia to a primary autonomic syndrome [4,6,16]. Anxiety and panic disorders can produce hyperventilation-driven tachycardia that mimics POTS; however, a positive active stand test with the characteristic HR increment, in the absence of hypocapnia, confirms orthostatic tachycardia independent of anxiety [6,9].

□ **Important:** Spontaneous intracranial hypotension (SIH) can present with orthostatic headache and tachycardia closely mimicking POTS. SIH should be considered when headache is the dominant orthostatic symptom, especially if it has a rapid onset on standing and resolves within seconds to minutes of lying supine. MRI with gadolinium (subdural collections, meningeal enhancement, brain sagging) establishes the diagnosis [23].

Table 5. Differential Diagnosis of POTS.

Condition	Key distinguishing features	Preferred test
Orthostatic hypotension	SBP fall ≥20 mmHg on standing; may have bradycardia	Active stand / TTT
Vasovagal syncope	Syncope with cardioinhibitory or vasodepressor response on TTT	TTT with GTN provocation
Inappropriate sinus tachycardia	Resting HR >100 bpm; not strictly orthostatic	Holter monitor; TTT
Phaeochromocytoma	Paroxysmal hypertension, headache, diaphoresis, sustained hypertension	24h urine catecholamines; CT/MRI adrenals
Hyperthyroidism	Weight loss, heat intolerance, resting tremor, exophthalmos	TFTs (fT4/TSH)
Spontaneous intracranial hypotension	Orthostatic headache, rapid onset and relief; may have tinnitus	MRI brain with gadolinium
Anxiety / panic disorder	Resting tachycardia, hyperventilation, somatic focus; no true HR increment	Active stand test; end-tidal CO2 monitoring
Deconditioning alone	HR increment meets criteria but resolves fully with exercise programme	Trial of exercise reconditioning

Vasovagal syncope (VVS) is a frequent diagnostic consideration, particularly in young women. The distinguishing haemodynamic feature is a cardioinhibitory or mixed response on tilt table testing — a concurrent fall in systolic blood pressure greater than 20 mmHg, often accompanied by bradycardia — in contrast to the tachycardia-predominant and blood-pressure-stable pattern of POTS [16,20]. Overlap between VVS and POTS exists; some patients exhibit both a POTS response during passive tilt and a subsequent vasovagal response with prolonged upright provocation [20]. Mast cell activation syndrome (MCAS) may mimic or co-exist with POTS, producing episodic flushing, urticaria, hypotension, and palpitations; serum tryptase, 24-hour urinary prostaglandin D2, and histamine metabolites are the key discriminating investigations [6]. Chronic fatigue syndrome (CFS/ME) shares the post-exertional malaise, cognitive dysfunction, and autonomic symptoms of POTS and may be comorbid in up to 30% of cases; the distinction rests on the orthostatic tachycardia criterion, which is diagnostic of POTS but not required for CFS/ME [7,9].

VII. Management — Non-Pharmacological, Pharmacological, and Exercise

Management of POTS is multimodal, typically requiring a combination of non-pharmacological strategies and, in a significant proportion of patients, pharmacotherapy. Non-pharmacological interventions form the foundation of management and are often sufficient for mild-to-moderate disease [4,6,8,9]. The importance of exercise reconditioning cannot be overstated — it is the only intervention for which there is Level 1b randomised controlled trial evidence of benefit in POTS [4,8,27,30].

Non-Pharmacological Interventions

Volume expansion through increased fluid and salt intake is the first prescription for virtually all POTS patients. A target fluid intake of 2–3 litres per day and dietary sodium supplementation of 10–12 g per day (or equivalent salt tablets) expands plasma volume and reduces orthostatic tachycardia [8,9]. Compression garments — waist-high graduated stockings at 30–40 mmHg pressure, or abdominal binders — reduce venous pooling and augment venous return. A 10–15 cm elevation of the head of the bed activates the RAAS during sleep and promotes sodium retention, with measurable improvements in morning plasma volume [8].

Graduated aerobic exercise reconditioning, pioneered by Levine and colleagues in the CHOP-POTS protocol, begins with recumbent aerobic exercise (cycling, rowing, swimming) to avoid orthostatic stress, and progressively increases duration and upright activity over 3–6 months [8,27]. The protocol targets 30 minutes of aerobic exercise 3–5 times per week, with resistance training (squats, core strengthening) added from weeks 2–4. Compliance is the primary limiting factor; exercise worsens symptoms acutely and requires patient education about the expected trajectory [27].

□ **Clinical Pearl:** The Levine reconditioning protocol (CHOP-POTS) is the best-evidenced single intervention for POTS. It improved quality of life scores, reduced orthostatic tachycardia, and increased plasma volume and left ventricular mass in the original randomised trial. The benefit is likely a class effect of aerobic exercise rather than protocol-specific [27].

Practical implementation of the exercise protocol requires attention to the exercise environment. Recumbent cycling or rowing should be performed in a cool, well-ventilated room; heat significantly worsens orthostatic tolerance and may precipitate presyncope during early training sessions [4,30,45]. Starting targets of 20 minutes of recumbent aerobic activity at 60–70% maximum predicted heart rate, three times per week, are realistic for deconditioned patients. Duration and frequency are progressively increased over 6–8 weeks before upright exercise is introduced. Resistance training — particularly lower-limb and core exercises performed in the seated or recumbent position initially — builds skeletal muscle pump function, which augments venous return and reduces the degree of orthostatic pooling [4,27,45]. Patients should be counselled that symptomatic worsening during the first 2–4 weeks of training is expected and does not indicate disease progression; adherence through this period is predictive of longer-term benefit.

Tilt training — daily passive standing against a wall or using a tilt table for progressively increasing durations (starting at 5 minutes, increasing to 30–40 minutes daily) — desensitises orthostatic reflexes and has demonstrated modest improvements in orthostatic tolerance [6,9,45]. Trigger avoidance (heat, prolonged standing, large meals, alcohol, dehydration) and patient education about the physiological basis of symptoms are equally important components of the management plan [4,6,9].

Pharmacological Management

Pharmacotherapy is considered when non-pharmacological measures are inadequate after 2–3 months of consistent adherence. Treatment is individualised based on POTS subtype, symptom profile, and co-morbidities [4,6,9,16,34]. No pharmacotherapy for POTS is approved by the Therapeutic Goods Administration (TGA) of Australia or the FDA for this indication; all drug use is off-label and supported predominantly by small observational studies and open-label trials [9,16].

Volume expansion agents including fludrocortisone (0.05–0.2 mg daily) act as synthetic mineralocorticoids to increase sodium and water retention, expanding plasma volume [4,6,8,29]. Desmopressin (0.1–0.4 mg orally or intranasally) provides short-term antidiuretic benefit, particularly for morning symptom control, but carries risks of hyponatraemia with repeated use [4]. Alpha-1 agonist midodrine (2.5–10 mg three times daily, daytime use only) enhances peripheral vasoconstriction and

venous return, and is among the best-evidenced pharmacological agents for POTS [4,6,8,9,32]. It must not be taken within 4–5 hours of bedtime due to the risk of supine hypertension.

Heart rate reduction is achieved with low-dose beta-blockers (propranolol 10–20 mg twice to four times daily) [31], ivabradine (5–7.5 mg twice daily, a selective If channel inhibitor avoiding the negative inotropic effects of beta-blockade) [28], or pyridostigmine (30–60 mg twice to three times daily), which enhances autonomic ganglionic neurotransmission and reduces orthostatic tachycardia [4,6,9,16,17,33]. Pyridostigmine has a particular role in patients with predominant standing tachycardia without significant supine symptoms. Central sympatholytics (clonidine 0.05–0.15 mg twice daily; methyldopa 125–250 mg twice to three times daily) are reserved for hyperadrenergic POTS with marked sympathetic excess [4,6].

Table 6. Pharmacological Agents Used in POTS Management.

Drug class / Agent	Dose range	Primary indication	Key cautions
Fludrocortisone	0.05–0.2 mg daily	Volume expansion; hypovolemic subtype	Monitor BP, K+, oedema; avoid in hypertension
Midodrine	2.5–10 mg TDS (daytime)	Neuropathic/venous pooling; all subtypes	Supine hypertension; avoid within 4–5h of bedtime
Propranolol (low dose)	10–20 mg BD–QID	Tachycardia-predominant; all subtypes	Avoid in hyperadrenergic at standard doses; fatigue
Ivabradine	5–7.5 mg BD	Hyperadrenergic subtype; beta-blocker intolerance	Avoid in pregnancy; visual phosphenes
Pyridostigmine	30–60 mg BD–TDS	Residual tachycardia; GI dysmotility present	Cholinergic effects; diarrhoea, abdominal cramps
Desmopressin	0.1–0.4 mg short-term	Acute volume loading; morning symptom control	Hyponatraemia risk; limit to short-term use
Clonidine / methyldopa	Clonidine 0.05–0.15 mg BD	Hyperadrenergic POTS only	Sedation; rebound hypertension on cessation
Droxidopa	100–600 mg TDS	Neurogenic orthostatic hypotension co-existing	Supine hypertension; limited POTS trial data

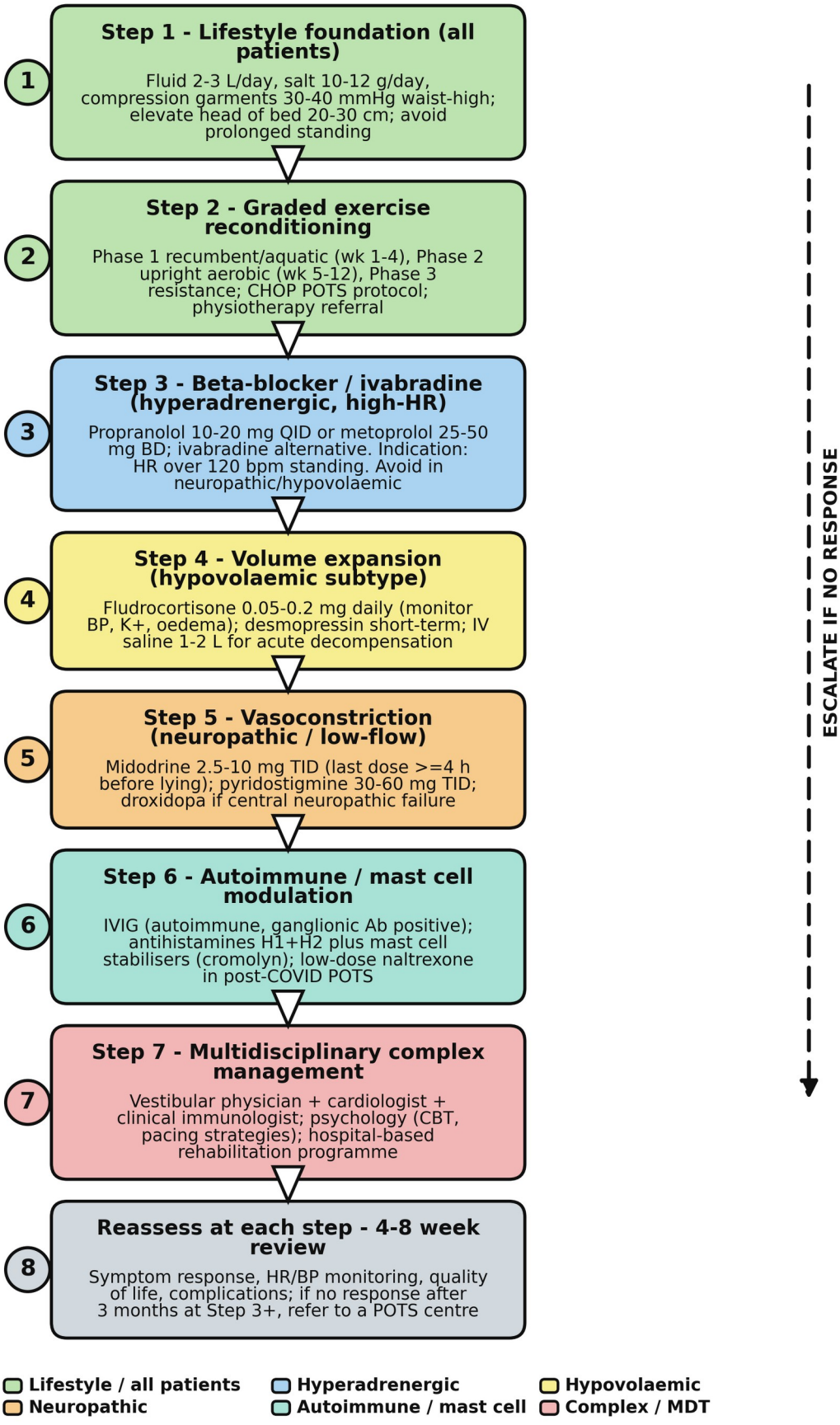


Figure 5. POTS Treatment Escalation Pathway — step-up algorithm from lifestyle to complex pharmacotherapy.
Source: Adapted from Raj SR et al. [6], Sheldon RS et al. [16], and Bryarly M et al. [4].

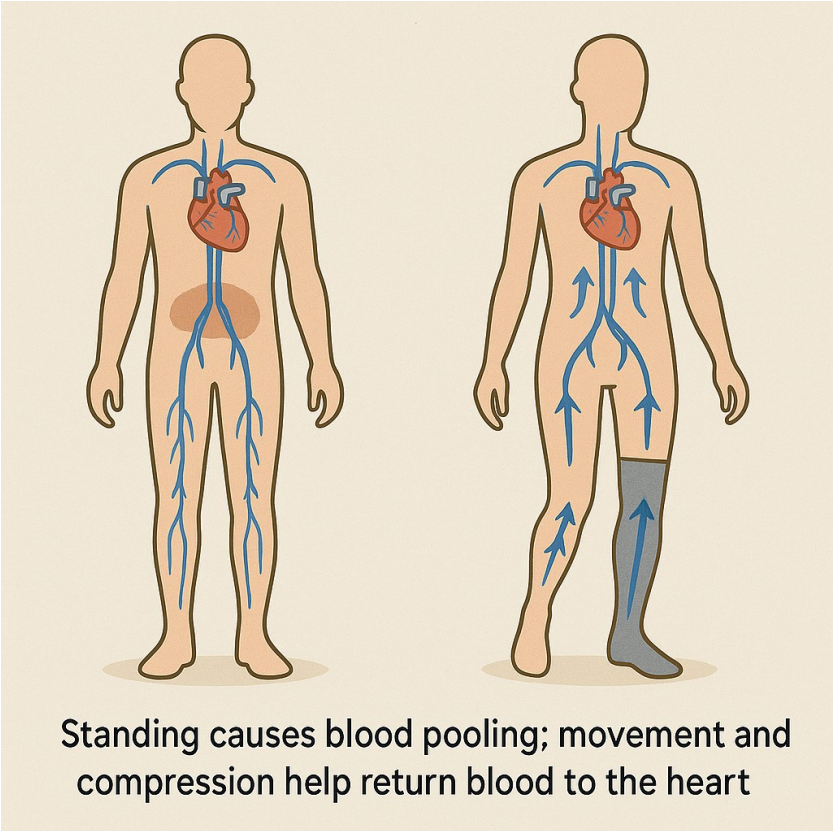


Figure 6. Physiological response to compression garments and exercise reconditioning — reduced venous pooling and improved orthostatic tolerance.
Source: Adapted from Levine BD et al. [27] and Raj SR [15].

VIII. Refractory POTS and Special Populations

A significant minority of POTS patients — estimated at 15–25% — fail to achieve adequate symptom control with optimised non-pharmacological measures and at least two pharmacological agents. This group requires multidisciplinary evaluation, dedicated autonomic clinic review, and consideration of less conventional interventions [4,6,9].

Refractory POTS — Evaluation and Management

In refractory cases, as outlined in the ESC/EHRA expert review [43], a comprehensive review should establish: (1) diagnosis accuracy (is the diagnosis of POTS secure, or have alternative diagnoses been missed?); (2) non-pharmacological compliance (are volume, salt, compression, and exercise strategies being consistently applied?); (3) unidentified contributing conditions (autoimmune aetiology, co-existing Ménière's disease, sleep apnoea, Chiari malformation, craniocervical instability); and (4) psychiatric comorbidities amplifying disability [4,6]. IVIG has shown benefit in case series of autoantibody-positive POTS, and plasmapheresis has been reported in severe refractory cases [7]. Intravenous saline infusion [47] provides acute relief and can be used as a bridge to effective oral therapy in severely symptomatic patients [4].

Adolescents

Management of adolescent POTS requires particular attention to the school, social, and developmental context. Adolescent POTS has a better prognosis than adult-onset disease, with many patients recovering by their mid-20s [10,22]. Exercise reconditioning is the cornerstone — preferably in a supervised physiotherapy context with school accommodation plans to facilitate participation. Pharmacotherapy should be used conservatively in this age group; midodrine and fludrocortisone have the most paediatric evidence [22].

Hypermobile Ehlers-Danlos Syndrome (hEDS)

The co-occurrence of POTS and hypermobile Ehlers-Danlos syndrome or hypermobility spectrum disorder (HSD) is well established, with prevalence estimates of 40–70% in POTS cohorts [4,6,46]. The pathophysiological link likely involves connective tissue laxity impairing venous integrity and augmenting venous pooling. Patients with hEDS-POTS often require higher compression pressures and may benefit from custom-fitted garments. They also have a higher rate of co-existing MCAS and chronic pain [4,44]. Genetic testing for vascular EDS (COL3A1) should be considered when significant tissue fragility or vascular complications are present.

Post-COVID POTS

POTS is among the most common autonomic complications of post-acute COVID-19 syndrome (Long COVID). SARS-CoV-2 infection triggers autonomic dysfunction through several proposed mechanisms: direct autoimmune injury (molecular mimicry with autonomic receptor antigens), small-fibre neuropathy from systemic inflammation, prolonged deconditioning from severe illness, and direct invasion of the brainstem autonomic nuclei [12]. Post-COVID POTS appears clinically indistinguishable from idiopathic POTS in most patients, though the onset is typically more acute and the sex predominance may be less marked [12]. Management follows the same stepwise approach, with particular attention to exercise reconditioning as deconditioning from acute illness is often profound.

□ **Clinical Pearl:** Post-COVID POTS may co-exist with other post-COVID autonomic manifestations including inappropriate sinus tachycardia and small-fibre neuropathy. A thorough autonomic evaluation is warranted before attributing all tachycardia to POTS in this population, as the management implications differ substantially [12].

Emerging therapies for refractory POTS include low-dose naltrexone (LDN), which has shown modest benefit in small open-label series by modulating neuroinflammatory pathways, and intravenous immunoglobulin (IVIg), which appears most effective in patients with documented autoantibody positivity or concurrent autoimmune conditions [21,50]. Droxidopa, a synthetic noradrenaline precursor approved for neurogenic orthostatic hypotension in the United States, has been used off-label in hyperadrenergic POTS but evidence remains limited [29]. Stellate ganglion block has been explored as an interventional option in hyperadrenergic patients, with case series reporting temporary improvement in palpitations and sympathetic symptoms [50]. Referral to a dedicated autonomic programme should be considered after failure of at least two pharmacological agents combined with optimised non-pharmacological therapy, as these centres offer access to investigational protocols, IV saline therapy, and multidisciplinary rehabilitation that are not routinely available in standard outpatient cardiology settings [4,29,50].

IX. Prognosis and Long-Term Outcomes

The natural history of POTS is generally favourable, particularly in younger patients. Longitudinal cohort data indicate that over 50% of POTS patients no longer meet formal diagnostic criteria after five years [37], though symptomatic burden may persist even in those who no longer fulfil the heart rate criterion [9,10]. The condition typically follows a relapsing-remitting course, with symptomatic exacerbations precipitated by intercurrent viral illness, physical deconditioning, psychological stress, pregnancy, or heat exposure [4,6,9].

Adolescent-onset POTS carries the most favourable prognosis: many teenagers recover by their mid-20s, particularly those with idiopathic or deconditioning-associated POTS without significant comorbidities [10,22]. Adult-onset POTS, especially in the context of autoimmune aetiology, hyperadrenergic phenotype, or significant connective tissue disorder, tends toward a more chronic course [4,6]. Post-COVID POTS prognosis data are limited but early reports suggest that a proportion of patients achieve remission within 12–24 months of onset, particularly with active management [12].

Predictors of poorer long-term outcome include: comorbid hypermobile EDS, mast cell activation syndrome or chronic fatigue syndrome; persistent autoantibody positivity; severe deconditioning at presentation; psychiatric comorbidity; and inadequate adoption of non-pharmacological strategies [4,6,9]. The relapse rate during pregnancy is approximately 30–50%, with symptoms typically worsening in the first trimester due to progesterone-mediated vasodilation and expanding venous capacitance, and often improving in the second and third trimesters as plasma volume expands [4].

The impact of POTS on reproductive health and pregnancy deserves specific attention in the vestibular physician counselling. Symptoms frequently worsen in the early first trimester due to progesterone-mediated peripheral vasodilation and increased venous capacitance. The physiological plasma volume expansion of the second and third trimesters often provides partial relief, though this is not universal [4,6]. Postpartum, the rapid loss of plasma volume and the physical demands of infant care frequently precipitate relapse. Patients planning pregnancy should optimise non-pharmacological strategies and discuss the safety profile of any current pharmacotherapy with their obstetric team; fludrocortisone, midodrine, and beta-blockers each carry specific pregnancy safety considerations [4]. The menstrual cycle exerts measurable effects on orthostatic tolerance: the luteal phase (high progesterone) is associated with worsened symptoms in many patients, and this temporal pattern can assist in the clinical differentiation of POTS from anxiety-related palpitations [4,6].

□ **Clinical Insight:** Quality of life impairment in POTS is severe and comparable to congestive heart failure in validated patient-reported outcome measures [49]. A validated POTS symptom scale — the POTS Symptom Score or the Vanderbilt Orthostatic Symptom Score (VOSS) — should be used at each clinic visit to track treatment response and guide escalation decisions [6,21].

Pregnancy in POTS warrants particular attention. The physiological plasma volume expansion of pregnancy typically ameliorates POTS symptoms in many patients during the second trimester, though first and third trimester exacerbations are reported [9,40]. Management during pregnancy requires avoidance of most pharmacotherapy — fludrocortisone, beta-blockers, and midodrine each carry category C or D risk designations — making enhanced non-pharmacological strategies and dedicated joint obstetric-cardiology review essential [40]. Post-partum exacerbation is common as plasma volume rapidly normalises and breastfeeding may further reduce intravascular volume [40]. Adolescent POTS has a favourable prognosis compared with adult-onset disease: longitudinal follow-up studies report resolution or significant improvement in 60–80% of adolescents within 3–5 years of diagnosis, particularly those without co-morbid hypermobile Ehlers-Danlos syndrome or autoimmune disease [9,38]. Shared decision-making regarding school accommodation, graded return to sport, and realistic recovery timelines is a core component of management in this age group [38,39].

X. Guidelines, Controversies and Future Directions

The 2015 Heart Rhythm Society Expert Consensus Statement on the Diagnosis and Treatment of Postural Tachycardia Syndrome, Inappropriate Sinus Tachycardia, and Vasovagal Syncope remains the primary guideline document for POTS management [16]. A 2022 update in CMAJ by Raj, Fedorowski, and Sheldon addressed post-COVID presentations and updated management recommendations [6]. The 2017 ACC/AHA/HRS guideline for syncope provides complementary guidance [36]. The 2018 ESC Guidelines for the Diagnosis and Management of Syncope include detailed guidance on orthostatic syndromes, though POTS-specific recommendations are less granular [20].

Current Controversies

The classification and nomenclature of POTS subtypes remain debated. Whether neuropathic, hyperadrenergic, hypovolemic, and autoimmune forms represent distinct entities or overlapping manifestations of a common syndrome is unresolved [4,6,15]. The diagnostic threshold in adolescents (≥40 bpm) is contested, with concerns about both over-diagnosis using lower thresholds and under-diagnosis using higher ones [22]. The role of autoantibodies in POTS pathogenesis — while supported by observational data — awaits definitive causal demonstration from intervention trials [7].

Lack of high-quality randomised controlled trial evidence for pharmacotherapy is a consistent limitation. Most drugs used in POTS are prescribed off-label based on small case series or open-label trials, and head-to-head comparisons are absent [9,16]. The optimal exercise prescription — intensity, modality, duration, and progression — requires larger trials to define. The CHOP-POTS protocol remains the best-evidenced approach, but real-world adherence is poor [27].

Future Directions

The post-COVID POTS epidemic has accelerated research investment. Longitudinal cohort studies tracking autonomic recovery, autoantibody dynamics, and treatment responsiveness are ongoing.

Biomarker development — including serum autoantibody panels, plasma volume assays, and transcutaneous nerve fibre density measurement — may enable precision diagnosis and personalised treatment selection [7,12,50]. Immunotherapy trials (IVIG, rituximab, plasmapheresis) for autoantibody-positive POTS are in early phases. Digital health technologies enabling continuous heart rate monitoring and remote coaching for exercise reconditioning represent promising adjuncts for a condition where in-person clinic attendance is frequently limited by symptom burden [4,6].

□ **Key Point:** POTS is an evolving field undergoing rapid growth in understanding, particularly in the context of post-COVID autonomic dysfunction. The vestibular physician should expect increasing referrals for dizziness in post-COVID patients and maintain familiarity with the diagnostic criteria, subtype classification, and evidence-based management hierarchy [6,12].

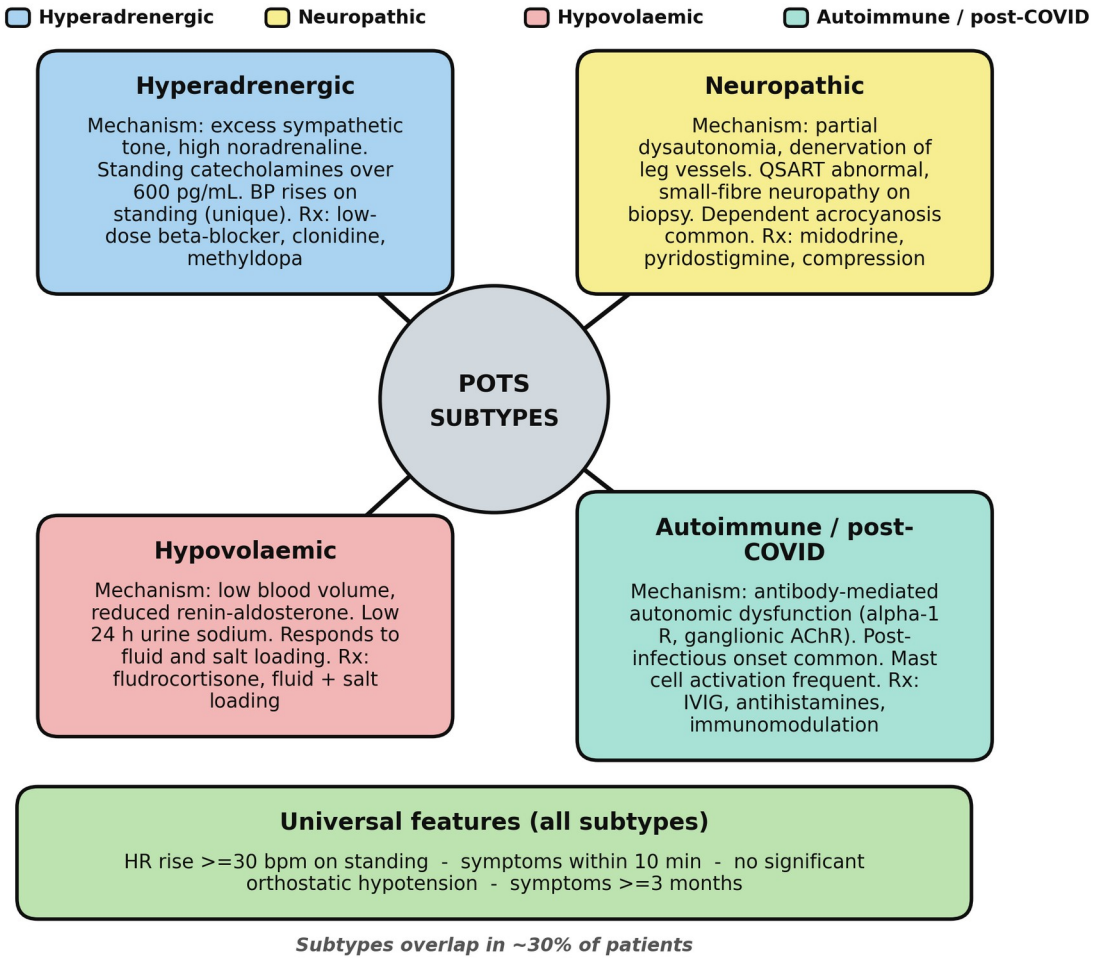


Figure 7. POTS Subtypes Classification — four primary pathophysiological mechanisms and their overlap with universal deconditioning.

Source: Adapted from Raj SR [15] and Bryarly M et al. [4].

The 2019 European Heart Rhythm Association (EHRA) practical guide on management of patients with POTS and the 2021 American Heart Association scientific statement on orthostatic hypotension and related conditions both recommend a structured step-up treatment algorithm beginning with non-pharmacological measures before pharmacotherapy [29,30]. International registries, including the Vanderbilt Autonomic Dysfunction Center cohort and the UK POTS service data, demonstrate that approximately 60–80% of patients achieve meaningful functional improvement with combined conservative management over 12–24 months [4,9,50]. A key unresolved controversy is whether POTS represents a single pathophysiological entity warranting a unified management approach, or whether

subtype-directed treatment algorithms improve outcomes — a question several ongoing randomised controlled trials are designed to address [15,29]. The post-COVID POTS phenotype has raised additional questions about the role of immune dysregulation, autoantibody production, and small-fibre neuropathy as targetable mechanisms, with early pilot data suggesting that intravenous immunoglobulin may benefit a subset of patients with documented autoantibody positivity [21,50]. Telemedicine and home-based autonomic monitoring are emerging as practical tools for longitudinal management of a condition that disproportionately affects young adults in their peak working and studying years [9,50].

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