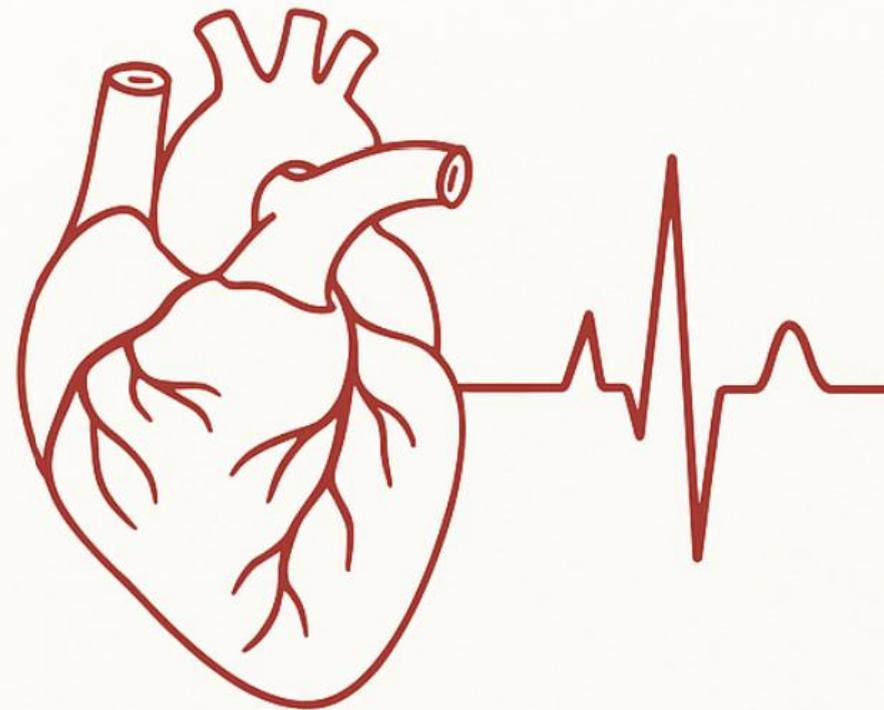


POTS and Dysautonomia Demystified:

A Cardiovascular Perspective for Primary Care



CARDIOLOGIST

UK HEALTHCARE



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November 1, 2025

Learning Objectives



Define POTS

Understand Postural Orthostatic Tachycardia Syndrome and broader dysautonomia spectrum



Recognize Presentations

Identify clinical manifestations and critical red flags requiring immediate attention



Understand Pathophysiology

Grasp cardiovascular mechanisms underlying autonomic dysfunction



Review Diagnostics

Master systematic evaluation approaches and appropriate testing strategies



Outline Management

Implement evidence-based therapeutic interventions for optimal outcomes



Identify Referral

Determine appropriate timing and process for cardiology consultation

What does the AFP say about Dysautonomia?

Review and Update of Dysautonomias

Print Comments

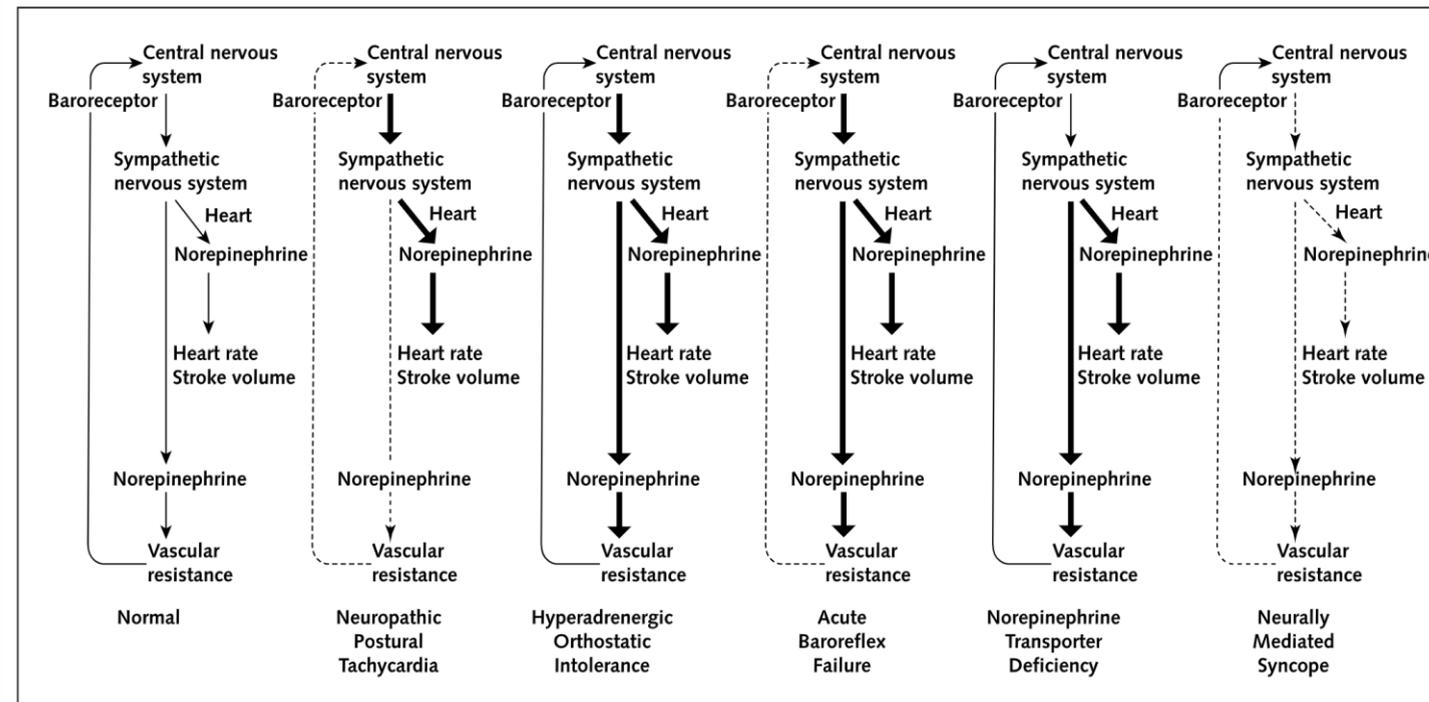
RICHARD SADOVSKY, M.D.

American Family Physician

Am Fam Physician. 2003;67(4):841-842

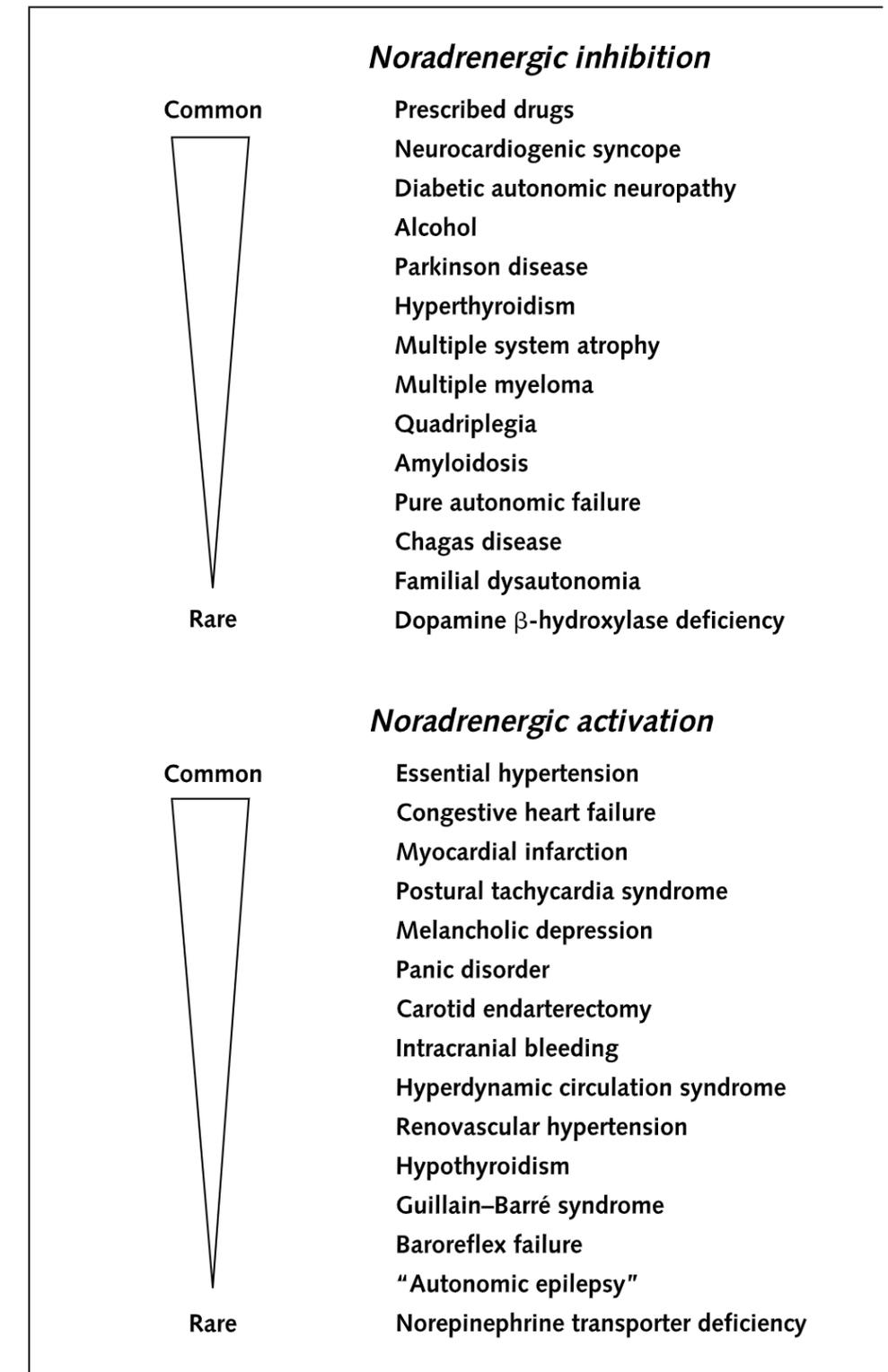
Dysautonomia is a condition in which abnormal autonomic function negatively affects a patient's health. Some conditions may be transient and mild, while others can result in progressive neurodegenerative diseases ([see accompanying table](#)). The autonomic system is the part of the nervous system mainly responsible for involuntary, unconscious functions within the body's organs, contrasting with voluntary, conscious skeletal-muscle activity. The autonomic system has sympathetic and parasympathetic portions that maintain body homeostasis. The main chemical messenger of the sympathetic nervous system is norepinephrine (noradrenaline), while that of the parasympathetic system is acetylcholine. A Clinical Staff Conference held on May 31, 2000, at the National Institutes of Health and moderated by Goldstein reviewed current knowledge about the spectrum of dysautonomias.

Figure 3. Regulation of sympathetic outflows to the heart and other parts of the body in orthostatic intolerance syndromes.



Thick lines represent increased activity, thin lines represent normal activity, and dashed lines represent decreased activity.

Figure 1. Dysautonomias featuring altered sympathetic noradrenergic function.



DYSAUTONOMIA SYMPTOMS

Head

- Dizziness
- Light-headedness
- Pre-Syncope
- Syncope
- Tinnitus (ringing in ears)
- Migraine/Headache
- Occipital Neuralgia
- Neck Pain
- Coat-hanger Pain

Sleep

- Insomnia
- Sleep-Disordered Breathing
- Sleep Paralysis
- Altered Sleep architecture

Cardiovascular

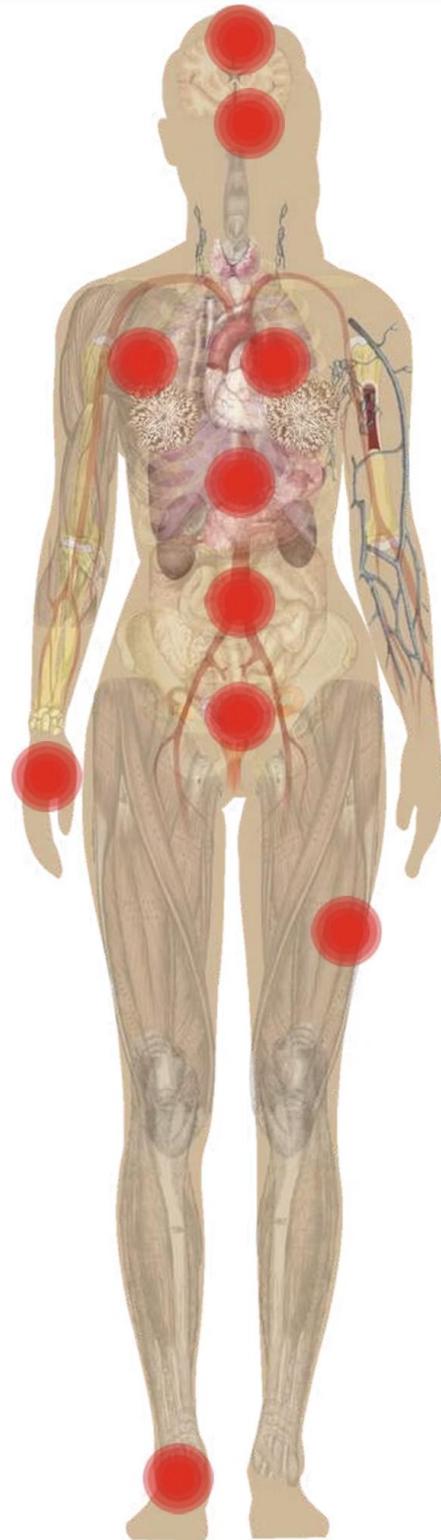
- Tachycardia
- Bradycardia
- Palpitations
- Hypertension
- Hypotension
- Large Swings in heart rate and blood pressure
- Chest Pain

Gastrointestinal

- Nausea/Vomiting
- Loss of Appetite
- Early Satiety
- Indigestion
- Constipation
- Diarrhea
- Abdominal Pain
- Difficulty Swallowing

Extremities

- Acral Coldness
- Raynaud Syndrome
- Hyperhidrosis
- Anhidrosis



Vision

- Sensitivity to Textures
- Sensitivity to Light
- Difficulty with Depth Perception

Mind

- Anxiety
- Attention Deficit
- Cognitive Impairment
- Brain Fog
- Mood Disturbances
- Sensitivity to Sound and Light

Respiratory

- Dyspnea
- Shortness of Breath
- Hyperventilation

Genitourinary

- Urinary Frequency
- Urinary Retention
- Urinary Incontinence
- Dysmenorrhea
- Endometriosis Pain
- Erectile Dysfunction
- Decreased Libido

Skin

- Cyanosis
- Blood Pooling
- Livedo Reticularis
- Sensitivity to Touch
- Flushing

Systemic

- Generalized Fatigue
- Early Exhaustion
- Exercise Intolerance
- Muscle Weakness
- Problems with Balance
- Sensitivity to Motion
- Tremulousness
- Temperature Dysregulation
- Paresthesias
- Heat Intolerance

Why This Topic Matters

Rising Prevalence

Increasing recognition and diagnosis rates, particularly post-pandemic with Long COVID associations

Diagnostic Challenges

Frequently misattributed to anxiety, panic disorder, or simple deconditioning, delaying appropriate care

Quality of Life Impact

Profound effects on daily functioning, employment, and psychological wellbeing requiring timely intervention

Primary Care Gateway

First point of contact for most patients, positioning you as critical diagnostic gatekeepers

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> J Intern Med. 2019 Oct;286(4):438-448. doi: 10.1111/joim.12895. Epub 2019 Apr 16.

The face of postural tachycardia syndrome - insights from a large cross-sectional online community-based survey

B H Shaw¹, L E Stiles^{2,3}, K Bourne¹, E A Green⁴, C A Shibus⁴, L E Okamoto⁴, E M Garland⁴, A Gamboa⁴, A Diedrich⁴, V Raj^{1,5}, R S Sheldon¹, I Biaggioni⁴, D Robertson⁴, S R Raj^{1,4}

Affiliations + expand

PMID: 30861229 PMCID: PMC6790699 DOI: 10.1111/joim.12895

Abstract

Background: Patients with postural tachycardia syndrome (POTS) experience chronic symptoms of orthostatic intolerance. There are minimal data detailing the demographics, clinical features and clinical course of this condition. This online, community-based survey highlights patients' experience with POTS. It consists of the largest sample of POTS patients reported to date.

FULL TEXT LINKS



ACTIONS



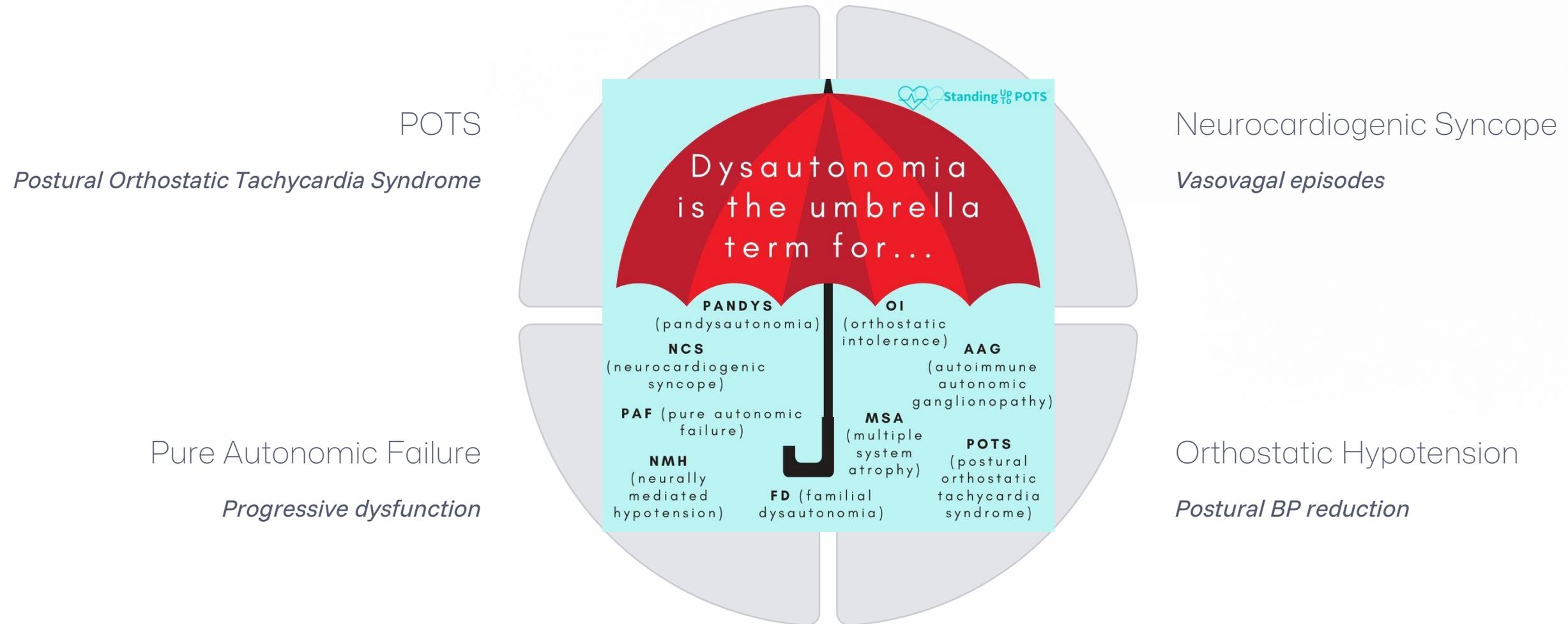
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Abstract

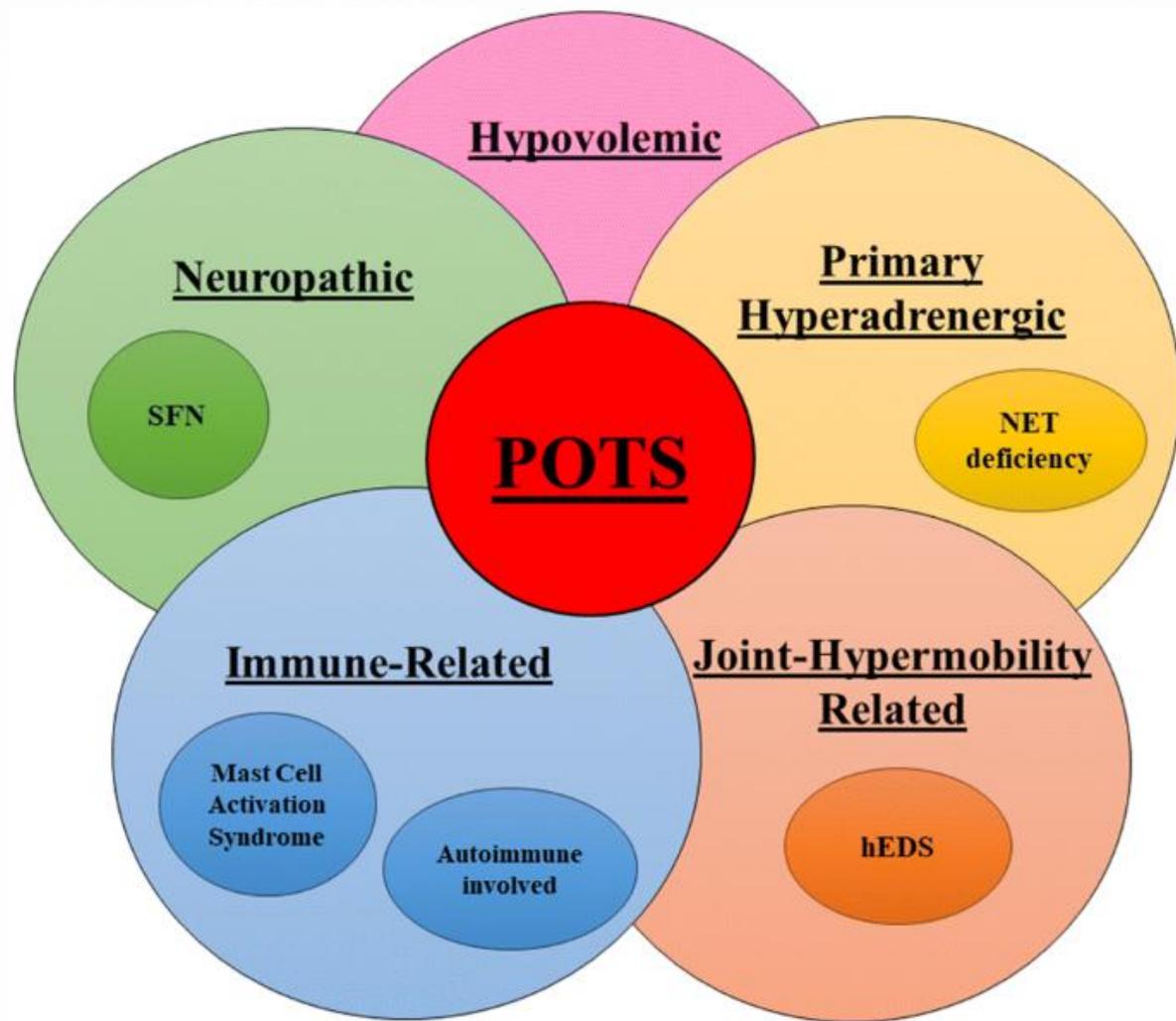
Defining Dysautonomia

Dysautonomia represents an umbrella term encompassing diverse disorders affecting the autonomic nervous system (ANS), which regulates involuntary physiological functions including heart rate, blood pressure, temperature control, and gastrointestinal motility.



📄 **Cardiovascular Relevance:** *These conditions manifest through abnormal heart rate responses, impaired blood pressure regulation, and compromised hemodynamic stability—all within the cardiovascular domain.*

What is POTS?



1

Diagnostic Criterion

Heart rate increase ≥ 30 bpm in adults (≥ 40 bpm in adolescents) within 10 minutes of standing, without orthostatic hypotension (BP drop $>20/10$ mmHg)

2

Cardinal Symptoms

Lightheadedness, palpitations, profound fatigue, cognitive impairment ("brain fog"), exercise intolerance

3

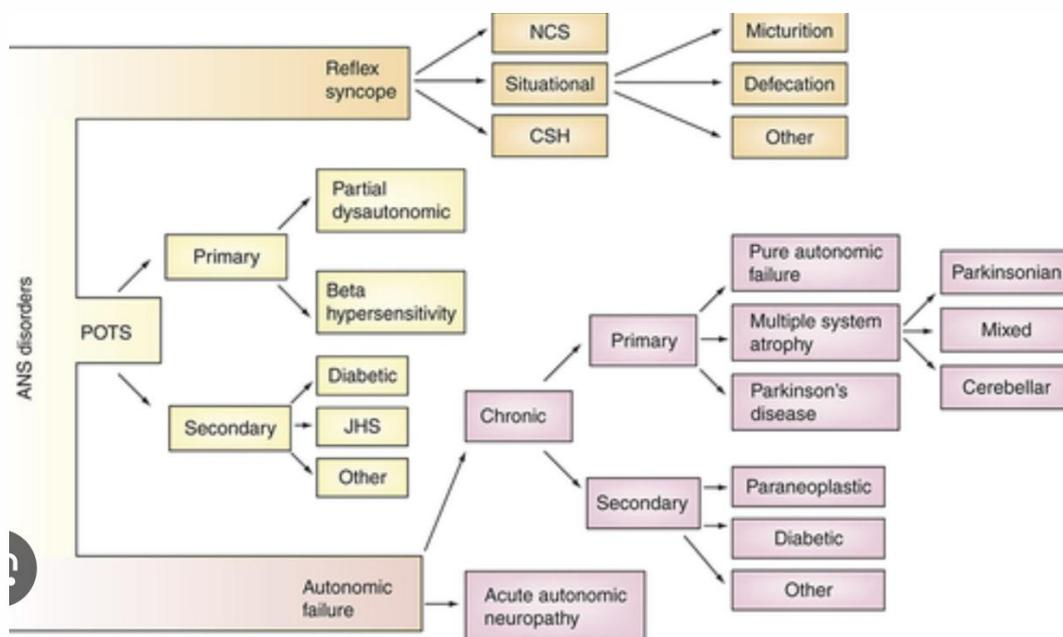
Chronicity Requirement

Persistent symptoms lasting $>3-6$ months, distinguishing from acute orthostatic responses

4

Demographic Pattern

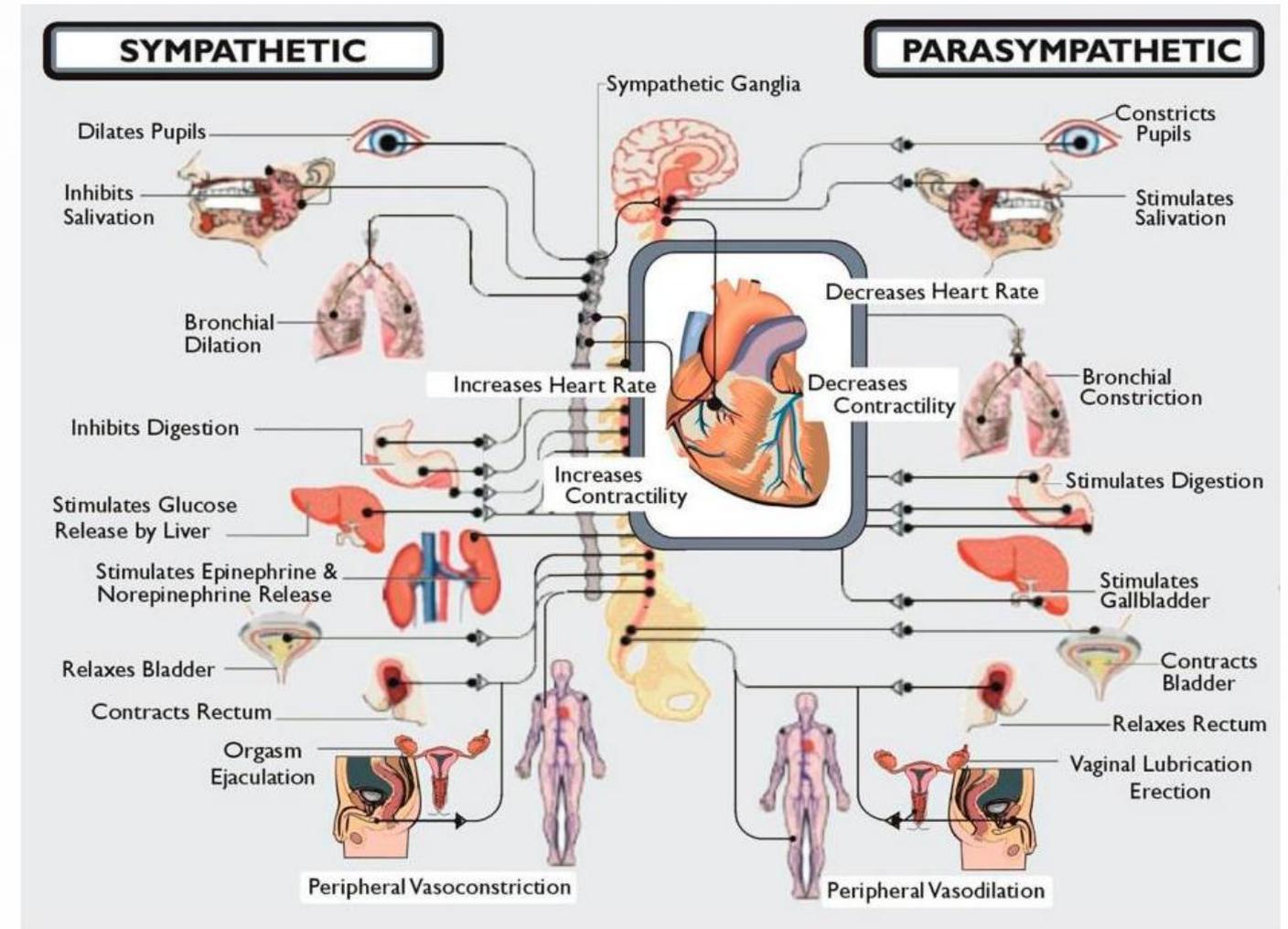
Predominantly affects women aged 15–50 years, though can occur across all demographics



Pathophysiology Overview

POTS represents a complex, multifactorial disorder involving intricate interactions between cardiovascular, neurological, and endocrine systems. Understanding these mechanisms informs targeted therapeutic approaches.

-  Autonomic Imbalance
Excessive sympathetic activation with inadequate parasympathetic counterregulation
-  Hypovolemia
Reduced circulating blood volume compromising venous return
-  Venous Pooling
Excessive blood accumulation in lower extremities upon standing
-  Baroreceptor Dysfunction
Impaired pressure sensor function disrupting cardiovascular reflexes
-  Deconditioning
Reduced fitness and small fiber neuropathy perpetuating symptoms



Cardiovascular Impact

Results in compensatory tachycardia, reduced venous return, diminished stroke volume, and inadequate cerebral perfusion—creating the characteristic symptom complex

Cardiovascular Physiology Refresher

Normal Standing Response

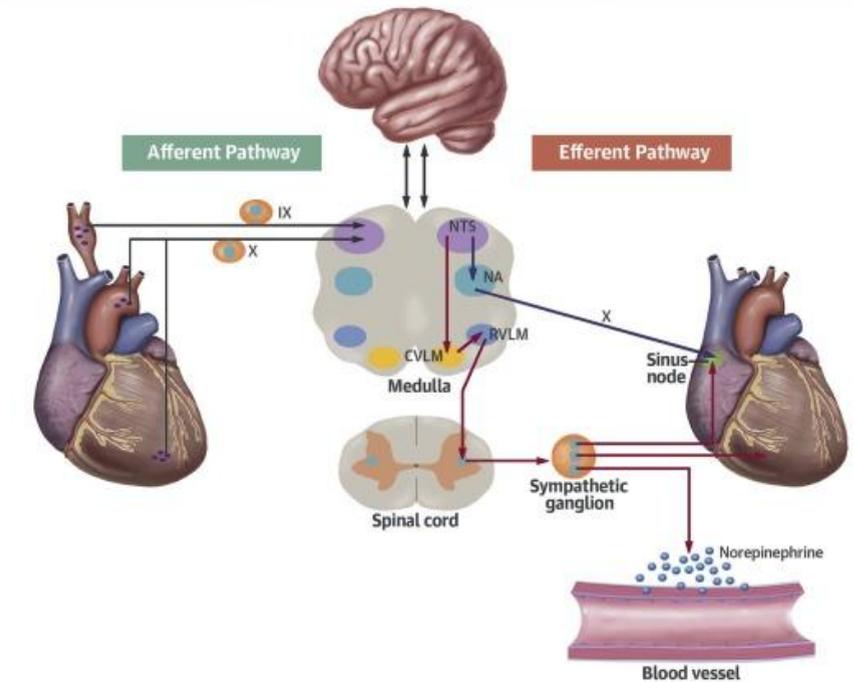
- Modest heart rate elevation (10–15 bpm)
- Minor systolic BP decrease (<10 mmHg)
- Rapid baroreceptor-mediated compensation
- Maintained cerebral perfusion pressure
- Brief gravitational blood redistribution (500–1000 mL to lower body)

Pathologic POTS Response

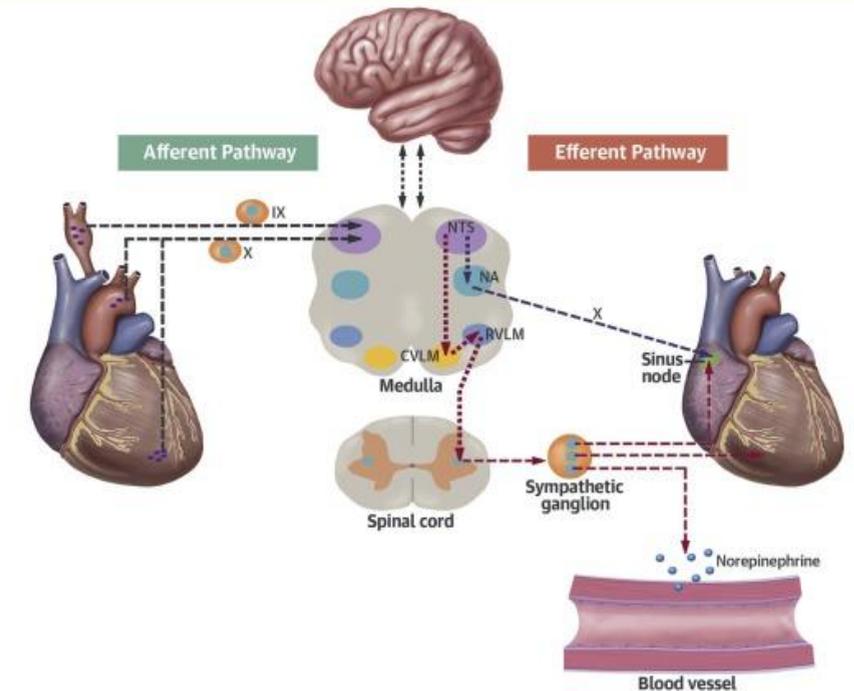
- Exaggerated heart rate increase (≥ 30 bpm)
- Inadequate peripheral vasoconstriction
- Excessive venous pooling
- Compromised stroke volume despite tachycardia
- Symptomatic cerebral hypoperfusion

CENTRAL ILLUSTRATION: Illustration of the Anatomy and Physiology of Blood Pressure Control in the Normal State and in Autonomic Failure

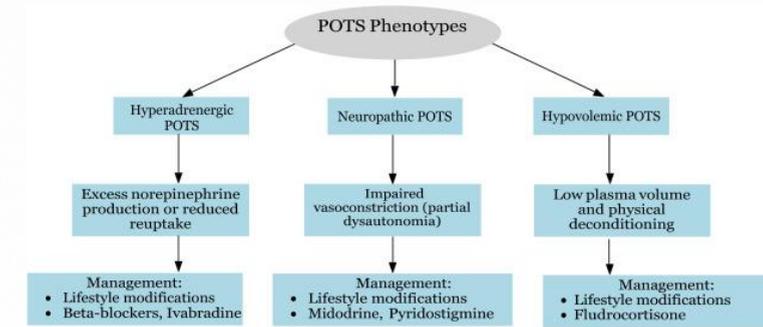
Neurophysiology of Normotension



Pathophysiology of Neurogenic Orthostatic Hypotension



Subtypes of POTS



Recognizing POTS subtypes enables targeted therapeutic strategies, as each variant responds differently to specific interventions. Patients may exhibit overlapping features across multiple subtypes.

Neuropathic POTS

Mechanism: Peripheral sympathetic denervation, predominantly affecting lower extremities

Features: Impaired vasoconstriction, acrocyanosis (bluish discoloration), reduced sweating in legs

Hyperadrenergic POTS

Mechanism: Excessive norepinephrine release or impaired clearance

Features: Elevated standing blood pressure, tremulousness, anxiety-like symptoms, increased plasma norepinephrine

Hypovolemic POTS

Mechanism: Abnormally low plasma volume (often 70% of predicted)

Features: Chronic dehydration, elevated hematocrit, rapid symptom response to volume expansion

Secondary POTS

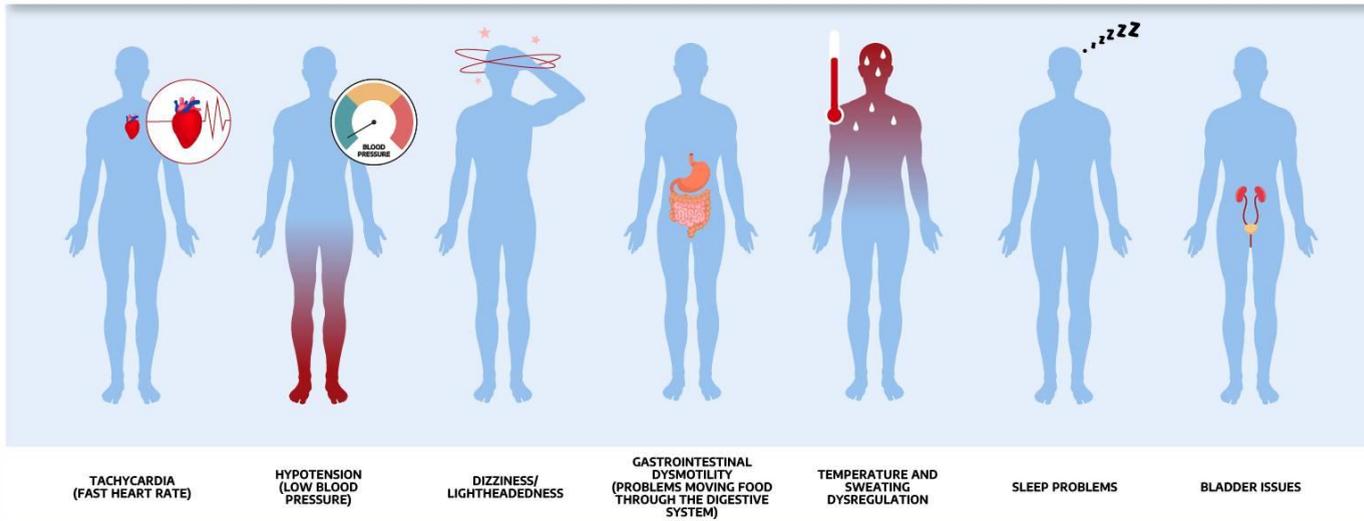
Mechanism: Dysautonomia resulting from underlying conditions

Associated with: Ehlers-Danlos syndrome, diabetes mellitus, autoimmune disorders, viral illness (including COVID-19)

Clinical Presentation



COMMON SYMPTOMS OF DYSAUTONOMIA



Symptom Constellation

- Orthostatic Intolerance

Dizziness, lightheadedness, presyncope (near-fainting) worsening with prolonged standing, particularly in warm environments or after meals

- Cardiac Symptoms

Palpitations, chest discomfort, awareness of rapid or forceful heartbeat, tremulousness

- Neurocognitive Effects

Profound fatigue, "brain fog" (impaired concentration and memory), mental slowness

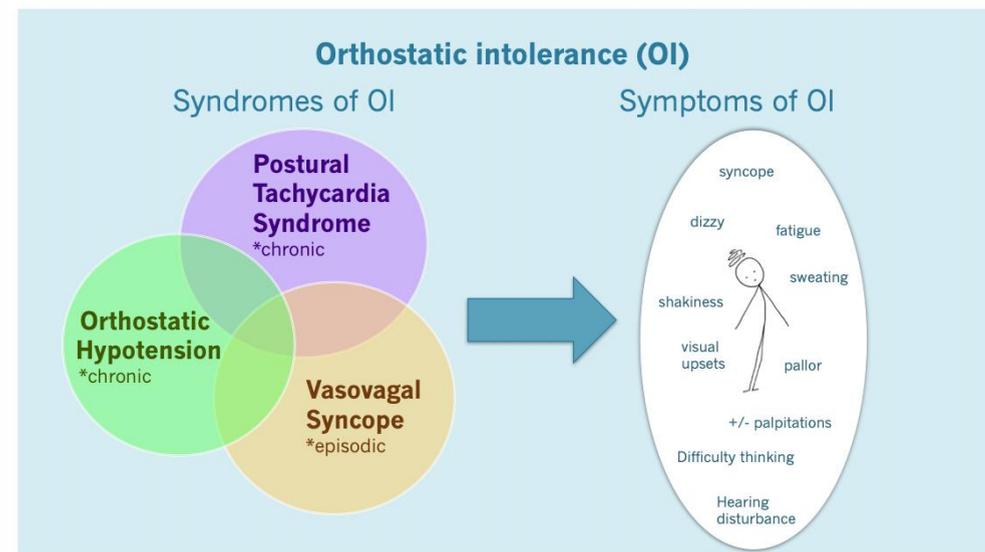
- Exercise Limitation

Post-exertional malaise, reduced functional capacity, prolonged recovery after physical activity

- Systemic Manifestations

Nausea, chronic headaches, temperature dysregulation, sleep disturbances, anxiety symptoms

Overlap between vasovagal syncope, orthostatic hypotension and PoTS



Common Misdiagnoses

POTS patients average 4–7 years and multiple physician consultations before accurate diagnosis. Recognition of these common misdiagnoses accelerates appropriate care and reduces patient frustration.

Anxiety or Panic Disorder

Tachycardia and palpitations easily mistaken for psychological etiology. However, symptoms are posturally triggered, not situational.

Chronic Fatigue Syndrome

Overlapping profound fatigue and post-exertional symptoms. Orthostatic tachycardia distinguishes POTS.

Fibromyalgia

Chronic pain, fatigue, and cognitive symptoms create diagnostic confusion. Many patients carry both diagnoses.

Simple Deconditioning

Exercise intolerance attributed to poor fitness. True deconditioning improves with activity; POTS may worsen.

Psychogenic Dizziness

Dismissal of symptoms as "medically unexplained." Objective orthostatic vital signs reveal physiologic abnormality.

Red Flags for Secondary Causes

⚠ Late-Onset Presentation

New symptoms after age 40 suggest acquired causes: diabetes, paraneoplastic syndromes, or neurodegenerative disease rather than primary POTS

⚠ Significant Weight Loss

Unintentional weight reduction may indicate malignancy, hyperthyroidism, or eating disorder with secondary autonomic effects

⚠ Neuropathy or Autoimmune Disease

Peripheral neuropathy symptoms or known autoimmune conditions (Sjögren's, lupus, celiac) suggest secondary dysautonomia

⚠ Medication-Induced

Recent initiation of α -blockers, vasodilators, diuretics, or psychotropic medications may produce iatrogenic orthostatic symptoms

⚠ Structural Heart Disease

Known cardiomyopathy, valvular disease, or arrhythmia requires exclusion before diagnosing primary POTS

Diagnostic Evaluation: Stepwise Approach

01

Comprehensive History & Physical

Document orthostatic symptoms, temporal patterns, triggers (heat, meals, exercise), family history, and functional impairment. Examine for signs of dehydration, Ehlers-Danlos features, or neuropathy.

03

12-Lead Electrocardiogram

Rule out underlying arrhythmia, conduction abnormalities, or structural heart disease indicators (LVH, ischemic changes).

05

Tilt Table Testing (Gold Standard)

Cardiology-performed diagnostic test providing controlled orthostatic challenge with continuous hemodynamic monitoring. Differentiates POTS from other dysautonomias.

02

Orthostatic Vital Signs

Measure HR and BP supine (after 5 min rest), then at 1, 3, and 10 minutes standing. Document symptoms at each interval. ≥ 30 bpm HR increase without significant BP drop confirms POTS.

04

Targeted Laboratory Testing

CBC (anemia), CMP (electrolytes, renal function), TSH (thyroid), morning cortisol (adrenal), vitamin B12, ferritin (iron stores). Consider hemoglobin A1c for diabetes screening.

06

Advanced Testing (Selected Cases)

Autonomic reflex testing (QSART, TST), plasma catecholamines (supine and standing norepinephrine), or additional specialized evaluations as indicated.

Recommendations - Investigation of POTS from Sheldon et al. 2015. Heart Rhythm 12(6): e44

A complete history and physical exam with orthostatic vital signs and 12-lead EKG should be performed on patients being assessed for POTS

Class **Level**

I **E**

Complete blood count and thyroid function studies can be useful for selected patients being assessed for POTS

IIa **E**

A 24-hour Holter monitor may be considered for selected patients being assessed for POTS, although its clinical efficacy is uncertain

IIb **E**

Detailed autonomic testing, transthoracic echocardiogram, tilt-table testing, and exercise stress testing may be considered for selected patients

IIb **E**

Recommendations - Treatment for POTS taken from Sheldon et al. 2015. Heart Rhythm 12(6): e44

A regular, structured, and progressive exercise program for patients with POTS can be effective

Class **Level**

IIa **B-R**

It is reasonable to treat patients with POTS who have short-term clinical decompensations with an acute intravenous infusion of up to 2 L of saline

IIa **C**

Patients with POTS might be best managed with a multidisciplinary approach

IIb **E**

The consumption of up to 2-3 L of water and 10-12g of NaCl daily by patients with POTS may be considered

IIb **E**

It seems reasonable to treat patients with POTS with fludrocortisone or pyridostigmine

IIb **C**

Treatment of patients with POTS with midodrine or low-dose propranolol may be considered

IIb **B-R**

It seems reasonable to treat patients with POTS who have prominent hyperadrenergic features with clonidine or alpha-methyl dopa

IIb **E**

Drugs that block the norepinephrine reuptake transporter can worsen symptoms in patients with POTS and should not be administered

III **B-R**

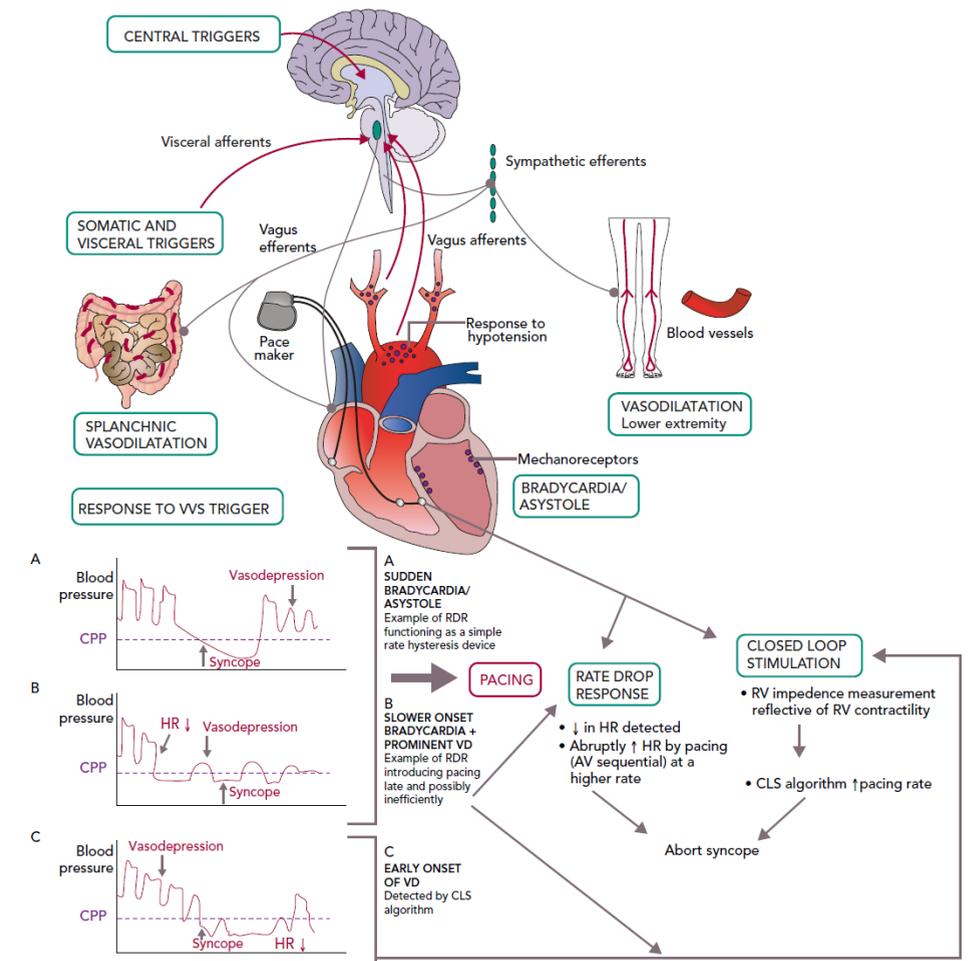
Regular intravenous infusions of saline in patients with POTS are not recommended in the absence of evidence, and chronic or repeated intravenous cannulation is potentially harmful

III **E**

Radiofrequency sinus node modification, surgical correction of Chiari malformation type I, and balloon dilation or stenting of the jugular vein are not recommended for routine use in patients with POTS and are potentially harmful

III **B-NR**

Figure 4: Pathophysiological Mechanisms in VVS Leading to Bradycardia and Hypotension, Role of Pacing and Currently Used Pacing Algorithms



There is generally considered to be a close relationship between systolic blood pressure and CPP with a critical level of 60-70 mmHg. CPP = cerebral perfusion pressure; CLS = closed loop stimulation; HR = heart rate; VD = vasodepression; RV = right ventricle; RDR = rate drop response.

Tilt Table Testing

The tilt table test is the gold standard for diagnosing orthostatic syndromes like POTS, providing objective data on your body's response to gravity.

Indications and Procedures for Head-Up Tilt Table Testing

Indications

High index of suspicion for orthostatic hypotension despite normal bedside orthostatic vital signs

Assessment of chronic orthostatic intolerance (e.g., postural orthostatic tachycardia syndrome)

Unexplained transient loss of consciousness

Inability to tolerate assessment of standing orthostatic vital signs because of motor issues or symptoms

Monitoring the disease course of an autonomic disorder to guide therapy

Protocol*

Environment and patient factors should be optimized according to the goal of testing and suspected disorder. Testing room should be quiet with a temperature of 68°F to 75°F (20°C to 24°C). Time of day of testing, proximity to meals, psychological state, and prescribed medications should be adjusted to yield the highest sensitivity.

Patient should have an empty bladder and be secured to a tilt table using straps. A supine position should be maintained for at least 10 minutes.

Continuous, noninvasive blood pressure and heart rate monitoring should be assessed for the duration of the test.

From a horizontal plane, the patient is slowly tilted upright between 60 and 80 degrees.

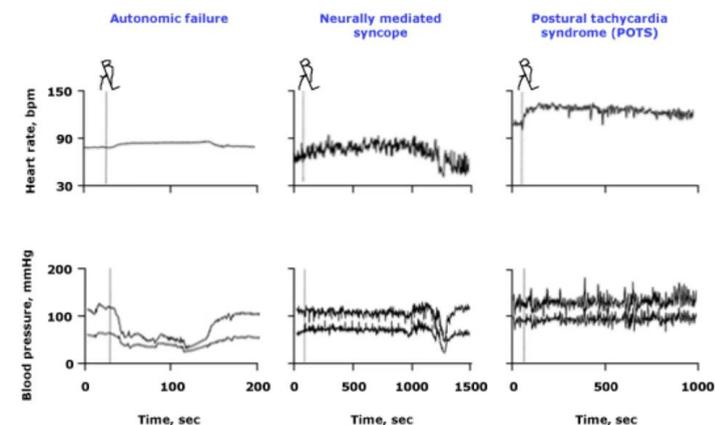
Duration of tilting varies by clinical question. For detecting orthostatic hypotension, the typical duration is five minutes.

The test should be terminated when diagnostic criteria are achieved, the patient becomes intolerant of the test (severe symptoms, adverse effects,† elective termination), or the protocol is completed.

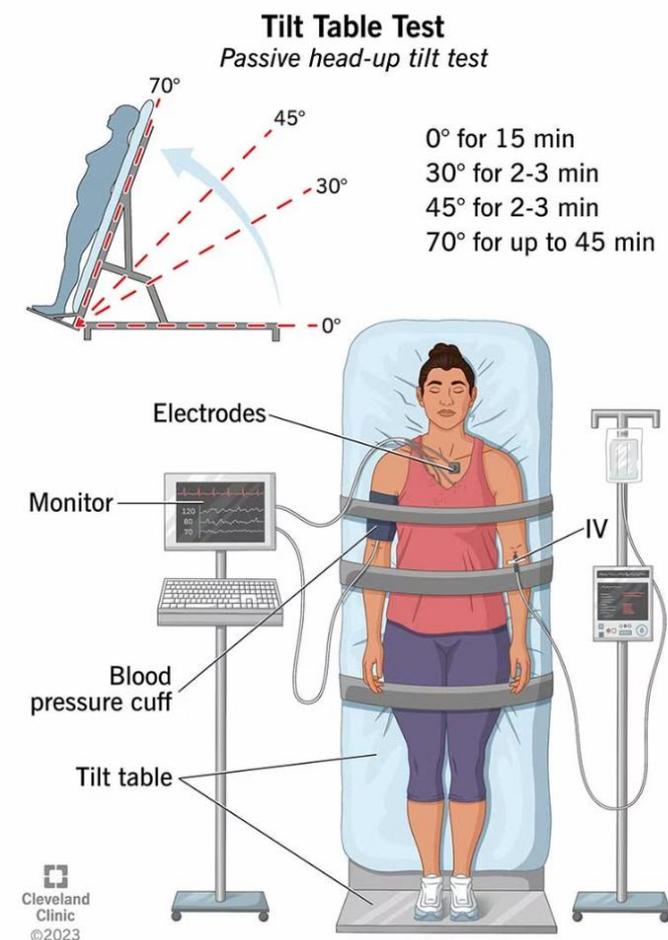
*—There is no universally standardized protocol, with notable variations (e.g., pharmacologic provocation) in each medical center. This protocol is provided as a general example.

†—Rare adverse effects include arrhythmia, syncope, chest pain, coronary vasospasm, and hypertensive crisis.

Adapted with permission from Lanier JB, Mote MB, Clay EC. Evaluation and management of orthostatic hypotension. Am Fam Physician. 2011;84(5):530, with additional information from references 33, 35, and 42.



Shown are the heart rate and blood pressure responses seen during tilt table testing in patients with various etiologies of syncope, including autonomic failure, neurally mediated syncope, and postural tachycardia syndrome (POTS). The absence of an appropriate reflex-induced increase in heart rate as the blood pressure falls is a useful clue to the presence of autonomic failure. During neurally mediated syncope, parasympathetic (vagal) activity increases as blood pressure declines, slowing the heart. With POTS, tilt table testing typically reproduces the clinical symptoms in association with a heart rate increase ≥ 30 beats/min or a maximum heart rate ≥ 120 beats/min within the first 10 minutes; these changes are not associated with hypotension.



Cardiovascular Differential Diagnosis

Not all tachycardia is POTS. Distinguishing primary cardiac etiologies from autonomic dysfunction prevents inappropriate treatment and identifies conditions requiring urgent intervention.

Inappropriate Sinus
Tachycardia (IST)

Persistent resting tachycardia (>100 bpm) not solely postural, often >90 bpm supine. Gradual HR increase unlike abrupt POTS response.

Supraventricular
Tachycardia

Paroxysmal narrow-complex tachycardia with abrupt onset/termination. Heart rate typically >150 bpm, compared to POTS range of 100–130 bpm.

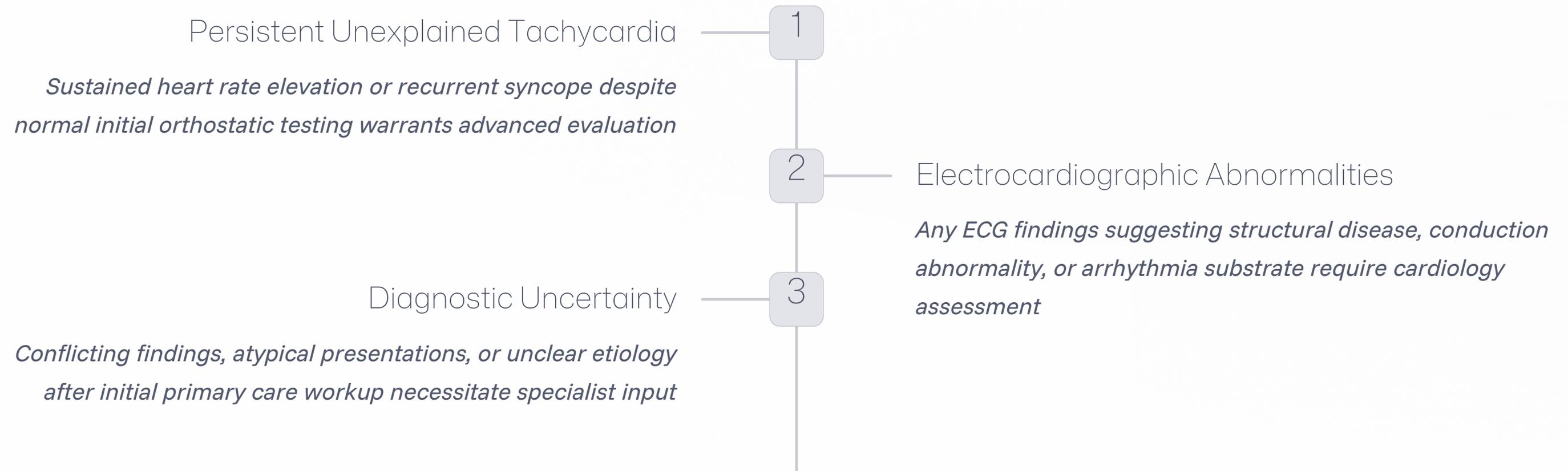
Atrial Arrhythmia

Irregularly irregular rhythm in atrial fibrillation or flutter. ECG distinguishes from regular sinus tachycardia of POTS.

Heart Failure or
Cardiomyopathy

Reduced ejection fraction, elevated BNP, structural abnormalities on echo. May coexist with compensatory tachycardia mimicking POTS.

Cardiac Testing: When to Refer



Recommended Cardiac Investigations

- **Echocardiogram:** Assess structure, function, valves
- **Holter Monitor:** 24–48 hour rhythm analysis
- **Event Monitor:** Extended monitoring for infrequent symptoms
- **Cardiac MRI:** If myocarditis suspected (post-viral cases)

Importance of Cardiology Referral

Why Cardiology Referral Matters

Early identification of autonomic cardiovascular abnormalities through specialized expertise. Access to advanced diagnostic tools including tilt table testing, autonomic reflex studies, and invasive hemodynamic monitoring when indicated.

Collaborative development of individualized management plans encompassing fluid optimization, targeted pharmacotherapy, and supervised exercise rehabilitation programs. Patient education and reassurance addressing the often-misunderstood nature of these symptoms.

Measurable Patient Impact

Improved diagnostic accuracy reduces time to treatment initiation. Decreased emergency department visits and unnecessary testing. Enhanced symptom control and functional status. Better long-term outcomes through coordinated specialty care and ongoing monitoring.

Multidisciplinary Benefit

Optimal POTS management requires integration across neurology (autonomic testing), physical therapy (reconditioning programs), psychology (coping strategies), and primary care (longitudinal support)—with cardiology serving as diagnostic and therapeutic hub.

POTS: A Look Beneath The Surface

She looks fine, but here's what she could be experiencing if she has postural orthostatic tachycardia syndrome (POTS).

reduced brain blood flow
trouble concentrating
lightheadedness
fainting
migraines
altered sleep phases
abnormally dilated pupils
sensitivity to light
esophageal dysmotility
nausea/bloating
diarrhea/constipation
peripheral neuropathy
blood pooling in the limbs



vertigo (dizziness)
sensitivity to noise
tachycardia (fast heart rate)
chest pain
shortness of breath
profound fatigue
iron storage deficiency
low blood volume
muscle weakness
bladder problems
reduced ability to sweat
heat/cold sensitivity
and more...

www.dysautonomiainternational.org/POTS





→ Diagnostic Complexity

Diagnostic uncertainty, conflicting findings, or inability to clearly differentiate POTS from primary cardiac conditions

→ Syncope or Presyncope

Recurrent episodes not adequately explained by bedside orthostatic vital signs alone

→ Significant Tachycardia

Resting heart rate consistently >100 bpm or excessive exertional tachycardia (>160 bpm with minimal activity)

→ Hyperadrenergic Features

Suspected hyperadrenergic POTS with standing hypertension, severe tremor, or anxiety-like symptoms

→ ECG or Rhythm Abnormalities

Any electrocardiographic findings or symptoms suggesting arrhythmia (palpitations with sudden onset/termination)

→ Treatment Failure

Inadequate response to first-line nonpharmacologic interventions after 8–12 weeks of adherent trial

→ Post-COVID Dysautonomia

Long COVID patients with persistent autonomic symptoms or suspected post-viral myocarditis involvement

→ Patient Request

Patient desire for specialist evaluation, particularly after prolonged diagnostic journey or severe functional impairment

UKY in Dysautonomia Research

University of Kentucky's Role

The University of Kentucky proudly serves as an active participant in the prestigious NIH-funded RECOVER-AUTO Trial, a landmark multicenter investigation examining autonomic dysfunction and POTS in post-acute sequelae of SARS-CoV-2 (PASC/Long COVID) patients.

This groundbreaking research advances our understanding of cardiovascular-autonomic recovery mechanisms, pathophysiologic endotypes, and optimal therapeutic strategies for virus-triggered dysautonomia.

Why It Matters for Your Practice

- Access to cutting-edge research protocols and evidence-based interventions
- UKY as a recognized referral center for complex dysautonomia cases
- Collaborative opportunities for community physicians
- Direct contribution to national knowledge base
- Clinical trial enrollment options for eligible patients



 En Español

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RECOVER-AUTONOMIC Clinical Trial

RECOVER-AUTONOMIC is focused on autonomic dysfunction symptoms related to Long COVID, such as fast heart rate, dizziness, and fatigue. These symptoms are related to damage to nerves that are part of the autonomic nervous system, which controls automatic body functions like heartbeat and blood pressure.



Nonpharmacologic Management

First-Line Interventions (Initiate Before Medications)

These lifestyle modifications form the foundation of POTS therapy. Even patients ultimately requiring pharmacotherapy benefit significantly from consistent application of these strategies. Emphasize that improvement requires 8–12 weeks of adherent implementation.

Volume Expansion

Fluids: 2–3 liters daily, spaced throughout day

Salt: 8–12 grams daily (3–5 teaspoons) unless contraindicated by hypertension or heart failure.
Bouillon, salt tablets, or heavily salted foods.

Compression Garments

Type: Waist-high compression stockings (30–40 mmHg)

Use: Don before standing in morning, wear throughout day

Alternative: Abdominal binders for patients intolerant of full-leg compression

Physical Countermeasures

Techniques: Leg crossing and tensing, calf muscle pumping, squatting, sitting with legs elevated

Timing: Use when symptoms develop or before prolonged standing

Reconditioning Exercise

Protocol: Recumbent or semi-recumbent aerobic exercise (rowing machine, recumbent bike, swimming)

Progression: Start 10–15 min/day, gradually increase to 30–45 min, 5 days/week

Resistance training: Add after establishing aerobic base

Trigger Avoidance

Minimize: Prolonged standing, hot showers/environments, rapid position changes, large carbohydrate-heavy meals, alcohol, dehydration

Pharmacologic Options

Drug*	Dosage	Comments	Cost†
Midodrine	Start at 2.5 mg three times per day orally, and titrate up to 10 mg three times per day	Dosage of 10 mg three times per day improves standing blood pressure measurements and symptom scores during a six-week course ⁵⁶⁻⁵⁸ Avoid three to five hours before bedtime ^{22,24}	10 mg: \$33 (—) for 60 tablets
Droxidopa (Northera)	Start at 100 mg three times per day orally, and titrate up to 600 mg three times per day	Improvement in symptom score based on validated assessment tool with medication titrated to symptom and blood pressure response over 14 days (mean dose = 430 mg) ⁶⁰ Avoid five hours before bedtime ²⁴ Effects may be blunted if coadministered with carbidopa ²²	Only available at specialty pharmacies
Fludrocortisone	Start at 0.1 mg once per day orally, and titrate up to 0.2 mg once per day	Improvement in symptoms based on validated assessment over three-week treatment period with 0.1 mg once per day orally ⁶² Use caution in heart failure Potassium level should be monitored ^{2,22,24}	0.1 mg: \$13 (—) for 30 tablets
Atomoxetine (Strattera)	18 mg once per day orally	Improvement in standing blood pressure and validated symptom scores ^{2,64,65}	18 mg: \$27 (\$392) for 30 tablets
Pyridostigmine (Mestinon)	60 mg once per day orally	Improved postural diastolic blood pressure reduction with 60-mg dosage ⁶³	60 mg: \$9 (\$508) for 30 tablets

*—Only midodrine and droxidopa are approved by the U.S. Food and Drug Administration for the treatment of orthostatic hypotension. The other medications are used off-label.

†—Estimated lowest GoodRx price for one month's treatment. Actual cost will vary with insurance and by region. Generic price listed first; brand name price listed in parentheses. Information obtained at <https://www.goodrx.com> (accessed August 11, 2021; zip code: 66211).

Key Principle: No single medication works universally. Sequential trials, combination therapy, and patience are often required. Coordinate medication selection with comorbidities.

Addressing Comorbidities

POTS rarely exists in isolation. Comprehensive management requires identifying and treating concurrent conditions that amplify symptoms or complicate therapy. Integrated care significantly improves overall outcomes.

Anxiety & Depression



Prevalence: Present in 30–50% of POTS patients—both as reaction to chronic illness and potentially sharing underlying mechanisms

Management: Cognitive behavioral therapy (CBT), acceptance and commitment therapy (ACT), stress reduction techniques. SSRIs/SNRIs if indicated (monitor for orthostatic effects).

Sleep Disorders



Issues: Difficulty initiating/maintaining sleep, non-restorative sleep, irregular sleep-wake patterns exacerbating fatigue

Management: Sleep hygiene optimization, consistent schedule, consider melatonin 3–5 mg at bedtime. Address sleep apnea if suspected.

Autoimmune Disease



Associations: Sjögren's syndrome, celiac disease, lupus, inflammatory bowel disease may present with secondary dysautonomia

Management: Rheumatology referral for evaluation and disease-specific therapy. Treat underlying autoimmune condition.

Ehlers-Danlos Syndrome



Connection: Hypermobile EDS strongly associated with POTS; joint laxity may contribute to venous pooling

Management: Physical therapy emphasizing joint stabilization, proprioception, and gentle strengthening. Compression garments particularly beneficial.

Patient Education and Support



Essential Messages for Patients

*"Your symptoms are **real and physiologic**—not psychological or 'all in your head.' POTS involves measurable cardiovascular dysfunction."*

"Improvement requires consistency with lifestyle measures. Changes won't be immediate, but most patients see meaningful benefit within 8–12 weeks."

"You're not alone. POTS affects millions, and support networks can provide valuable coping strategies and validation."

Actionable Self-Management

- *Teach postural precautions (gradual position changes, avoid sudden standing)*
- *Encourage HR/BP self-monitoring with home blood pressure device*
- *Provide symptom diary templates to identify triggers and track progress*

Recommended Patient Resources

- ***Dysautonomia International** (dysautonomiainternational.org)*
- ***Standing Up to POTS** (standinguptopots.org)*



Case Example #1: Classic POTS

Clinical Presentation

Patient: 23-year-old female graduate student

Chief Complaint: 6-month history of dizziness and palpitations, particularly during morning lectures

Orthostatic Vitals:

- Supine: HR 72, BP 110/70
- Standing (3 min): HR 125, BP 108/68
- Symptomatic with standing

Initial Workup:

- Normal ECG, CBC, CMP, TSH
- No history of syncope
- No cardiac risk factors

Diagnosis & Management

Testing: Tilt table test confirmed POTS (HR increase 58 bpm at 10 minutes, no hypotension)

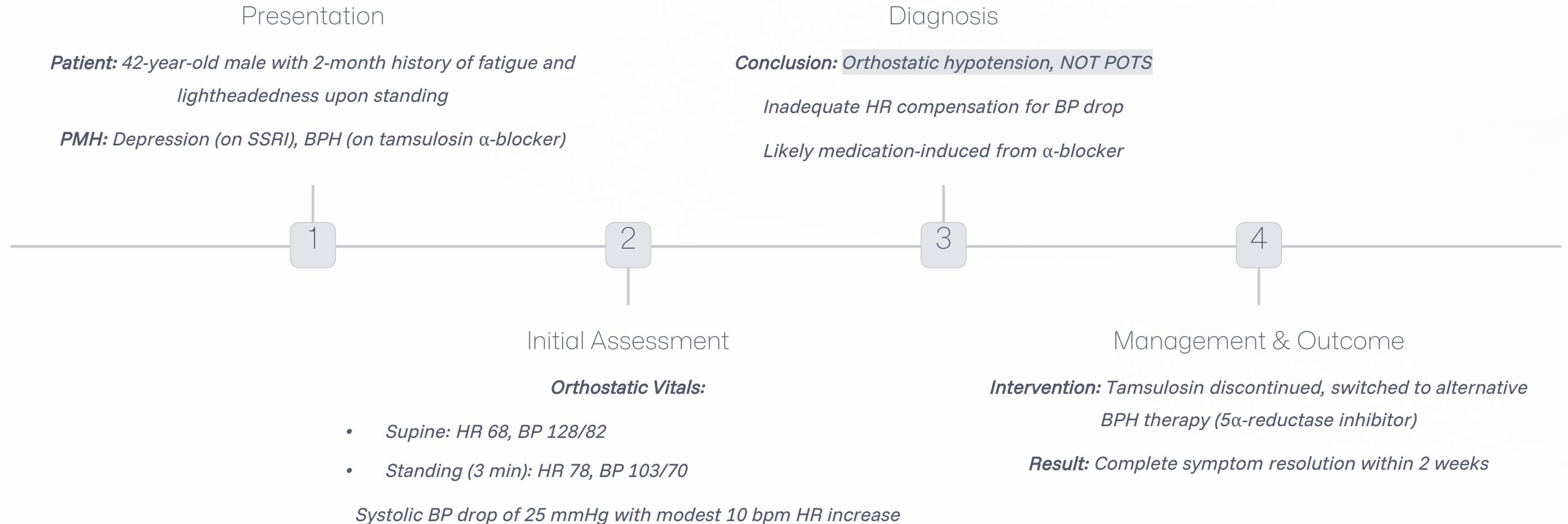
Initial Management:

1. Fluid intake to 2.5 L daily
2. Salt supplementation (10 g/day)
3. Waist-high compression stockings
4. Recumbent exercise program (rowing, swimming)
5. Low-dose propranolol 10 mg BID

3-Month Follow-Up Outcome:

- Standing HR reduced to 95 bpm
- Significant symptom improvement
- Returned to full academic activities
- Able to stand through lectures without presyncope

Case Example #2: Medication-Induced Orthostatic Hypotension



Clinical Pearl

***Always review medications** before diagnosing primary dysautonomia. Common culprits include α -blockers, vasodilators, diuretics, tricyclic antidepressants, and antihypertensives. Iatrogenic orthostatic symptoms are reversible with medication adjustment.*

Collaboration with Primary Care

Primary care physicians serve as the cornerstone of longitudinal POTS management. While specialists provide diagnostic confirmation and targeted therapy, the ongoing partnership between patient and primary care provider determines ultimate success.



Early Recognition & Validation

*First crucial step: **believe your patient**. Take orthostatic symptoms seriously, perform bedside vital signs, and validate the real physiologic nature of their experience. Early recognition accelerates diagnosis and prevents years of frustration.*



Exclude Secondary Causes

Conduct systematic evaluation ruling out mimics and underlying conditions. Medication review, basic laboratory testing, and ECG should precede specialty referral. Address easily correctable factors first.



Initiate Lifestyle Measures

Begin nonpharmacologic interventions immediately—don't wait for cardiology appointment. Hydration, salt supplementation, and patient education can be started in primary care, providing early symptom relief.



Coordinate Specialty Care

Facilitate referrals to cardiology, autonomic clinics, and ancillary services (physical therapy, psychology) as needed. Serve as care coordinator ensuring communication between specialists and preventing fragmented treatment.



Provide Long-Term Support

POTS is chronic, requiring ongoing monitoring, medication adjustments, and psychosocial support. Regular primary care follow-up sustains gains, addresses setbacks, and provides consistent therapeutic relationship through disease fluctuations.

Key Takeaways

Definition

POTS represents chronic orthostatic intolerance with excessive HR increase (≥ 30 bpm adults, ≥ 40 bpm adolescents) upon standing, lasting >3–6 months

Pathophysiology

Multifactorial cardiovascular and autonomic dysfunction involving sympathetic overdrive, hypovolemia, venous pooling, and baroreceptor impairment

Diagnosis

Clinical diagnosis supported by bedside orthostatic vitals; tilt table testing provides gold-standard confirmation and differentiates POTS from other dysautonomias

Management

Foundation: aggressive hydration (2–3 L/day), salt supplementation (8–12 g/day), compression garments, and graded exercise reconditioning. Medications as adjunct therapy.

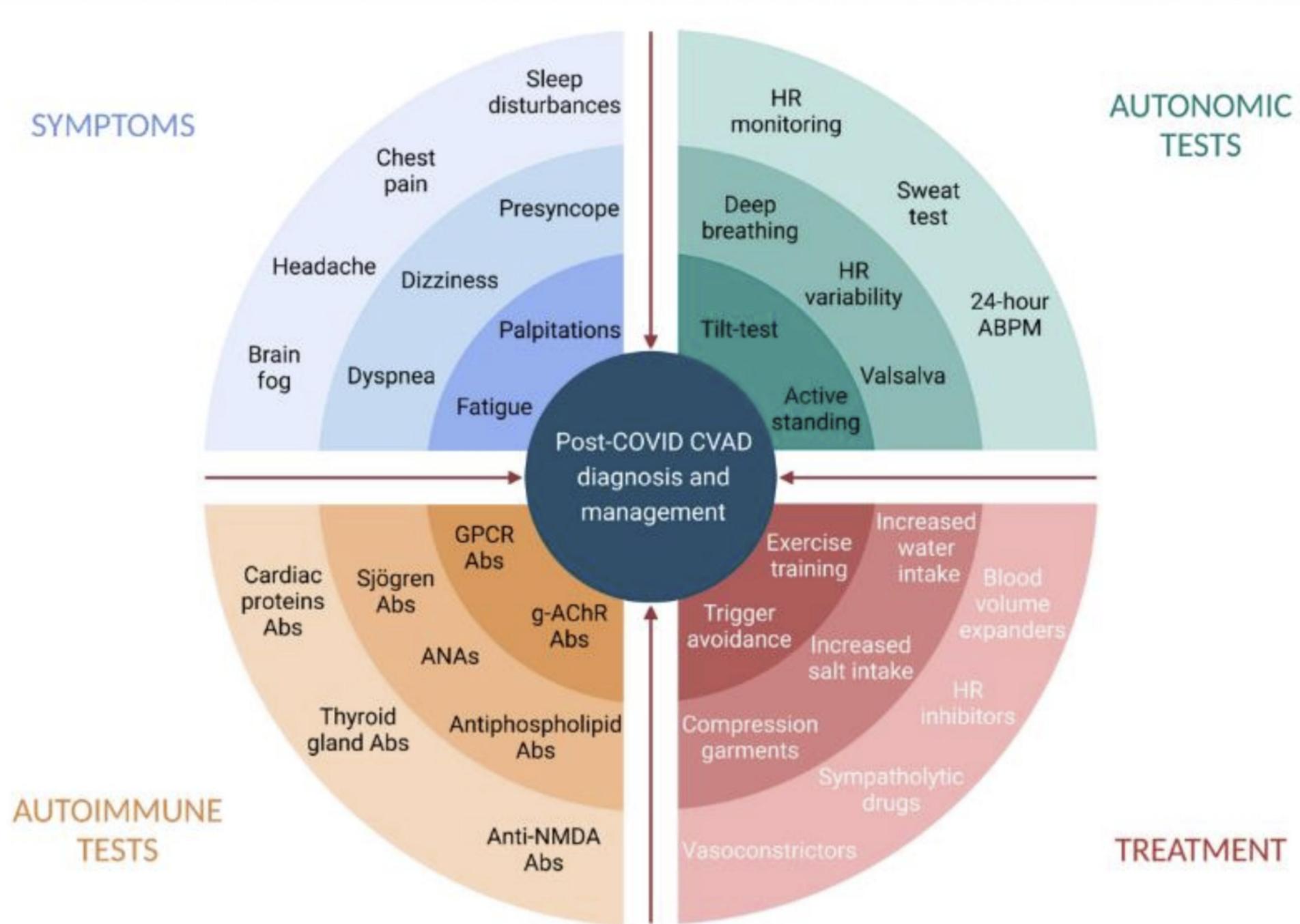
Collaborative Care

Optimal outcomes require partnership between primary care, cardiology, and multidisciplinary team (PT, psychology, nutrition)

UKY Excellence

University of Kentucky leading national research through NIH RECOVER-AUTO trial, serving as regional referral center for complex dysautonomia

From Tilt to Team: A Multidisciplinary Continuum of Care for the Chronicity of POTS and Dysautonomia



Questions & Discussion

Contact & Referral Information

University of Kentucky

Gill Heart & Vascular Institute

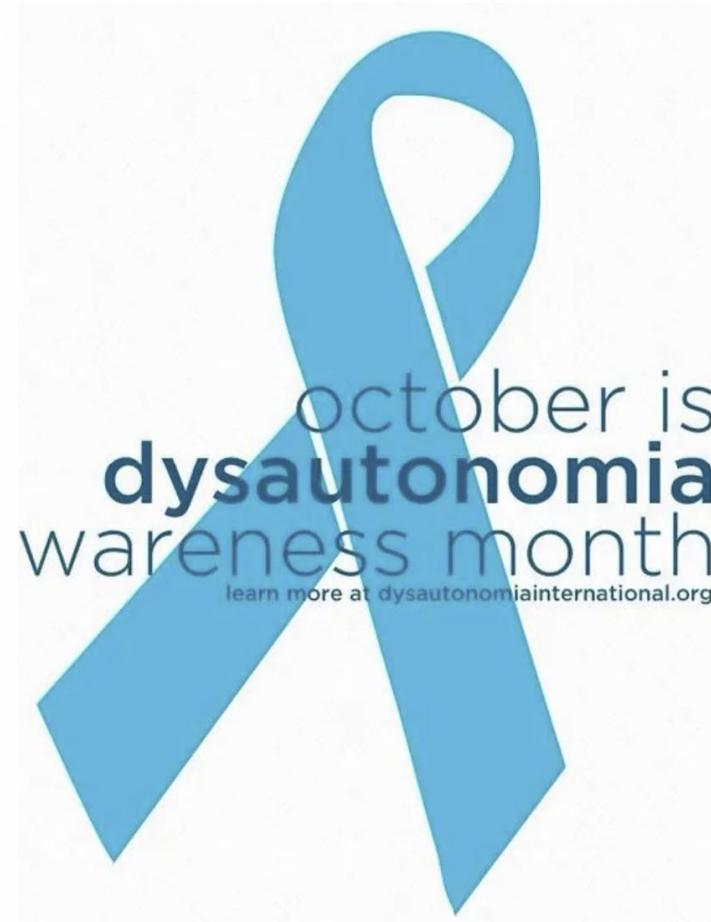
Additional Resources

For Patients:

- *Dysautonomia International: dysautonomiainternational.org*
- *Standing Up to POTS: standinguptopots.org*

For Clinicians:

- *NIH RECOVER Initiative: recovercovid.org*
- *ACC CardioSmart POTS Resources*



DYSAUTONOMIA

What is Dysautonomia?

Dysautonomia refers to a group of disorders where the autonomic nervous system (ANS) does not function as it should. The ANS helps control everything our body does automatically. This can involve dysfunction in any of the divisions of the ANS; the sympathetic, parasympathetic, or enteric nervous systems. Dysautonomia can affect every system in the body, sometimes in unpredictable ways. Symptoms may be mild or debilitating. They also may wax and wane in intensity, or be relentless.

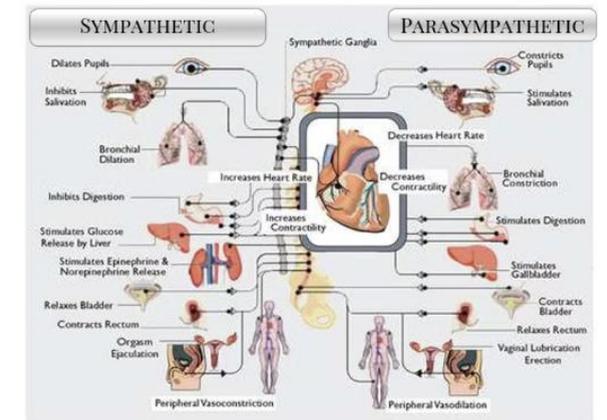
Depending on the type of dysautonomia and its cause, patients may deal with symptoms permanently or in some cases symptoms will improve and they will recover.

Forms of Dysautonomia

While some specific forms are rare, dysautonomia itself is not. Over 70 million people in the world are living with one or more forms of dysautonomia. Some of the forms include:

- Postural Orthostatic Tachycardia Syndrome (POTS)
- Vasovagal Syncope (VVS)
- Orthostatic Intolerance (OI)
- Autoimmune Autonomic Ganglionopathy (AAG)
- Pure Autonomic Failure (PAF)
- Multiple System Atrophy (MSA)
- and more.

In some cases, otherwise healthy patients can develop dysautonomia or the cause may be unknown. There are also patients who develop dysautonomia secondary to other underlying



conditions such as Ehlers-Danlos syndromes, Chiari malformation, mast cell activation disorder, Parkinson's disease, diabetes, autoimmune disorders, viruses, trauma, and more. Patients whose dysautonomia is caused by an underlying condition are more likely to deal with symptoms to some degree indefinitely. However, when possible, treating the underlying condition can sometimes improve patients' symptoms of dysautonomia.

Prognosis

There is currently no cure for dysautonomia, but ongoing research is building a better understanding of many forms of dysautonomia and offering new hope. Treatment involves assessing each individual's unique presentation and combining lifestyle changes and sometimes medication use to help improve symptoms. Proper medical care and patient education can help those affected by dysautonomia better manage their condition and improve their quality of life

