POTS CHEAT SHEET

# Postural Orthostatic Tachycardia Syndrome (POTS) — Cheat Sheet for Vestibular Physicians

*Anchor on the sustained heart-rate rise on standing without a blood-pressure fall. Reframe to orthostatic hypotension or vasovagal syncope when the haemodynamics do not fit.*

## ► Why POTS matters

A chronic disorder of orthostatic intolerance and the commonest cause of orthostatic tachycardia in young adults. Prevalence ~0.2–1%, ~80% female, peak onset 15–45 y, frequently post-viral with a marked post-COVID rise. A final common pathway — not one disease — reached by neuropathic, hyperadrenergic, hypovolaemic and autoimmune mechanisms. Dizziness and brain fog make it a frequent dizzy-clinic diagnosis, often after years of delay.

## Indications — when this pathway fits

### ► When to apply this work-up

► **Symptomatic orthostatic tachycardia — lightheadedness, palpitations, tremor, brain fog — worse standing, better lying.**

► **Worse with heat, meals, alcohol and deconditioning; a young patient, often female, often after a viral illness.**

► **Normal structural work-up; distinguish from orthostatic hypotension, vasovagal syncope, inappropriate sinus tachycardia and endocrine mimics.**

## Mechanism — why POTS happens

| Driver | Mechanism | Clinical relevance |
|---|---|---|
| Venous pooling | On standing ~500 mL pools in legs/splanchnic bed; vasoconstriction inadequate | Reduced venous return and stroke volume — the primary trigger |
| Compensatory tachycardia | Sympathetic surge defends cardiac output as filling falls | The defining ≥30 bpm rise; palpitations, tremor, brain fog |
| Deconditioning & low volume | Smaller, under-filled heart and reduced plasma volume | Amplifies the loop — why reconditioning is foundational |

***Pearl — Measure the rate, not just the dizziness. The sustained ≥30 bpm rise on standing (≥40 in adolescents) WITHOUT a blood-pressure fall is what separates POTS from orthostatic hypotension and vasovagal syncope.***

## Diagnostic criteria — heart-rate thresholds

| Tier | Required features |
|---|---|
| Adult POTS | HR rise ≥30 bpm within 10 min of standing; NO orthostatic hypotension; symptoms ≥3 months; reproduced on testing. |
| Adolescent (12–19 y) | HR rise ≥40 bpm within 10 min of standing; otherwise as for adult POTS. |

***Pearl — Exclude the reversible mimics before you label. A focused screen — FBC, TFTs, fasting glucose, electrolytes, morning cortisol — plus a medication review (stimulants, vasodilators, diuretics) precedes the diagnosis.***

## Investigations — anchored on the active stand test

| Test | Purpose | When to order |
|---|---|---|
| Active stand / NASA lean (10 min) | Confirm HR rise; no significant BP fall; reproduce symptoms | Bedside — every assessment |
| Head-up tilt-table 70° | Gold-standard confirmation if bedside equivocal | Equivocal or discordant cases |
| Standing plasma catecholamines | Identify hyperadrenergic subtype (noradrenaline over 600 pg/mL) | Suspected hyperadrenergic |
| QSART + skin biopsy | Confirm neuropathic subtype (small-fibre neuropathy) | Suspected neuropathic |
| Autoantibody panel + Holter | Autoimmune subtype; exclude primary arrhythmia | Post-viral / refractory cases |

***Pearl — Subtype the patient — it changes the drug. Standing catecholamines, QSART and autoantibodies separate hyperadrenergic, neuropathic and autoimmune POTS, each with a different first-line agent.***

## Differential diagnosis — high-yield mimics

| Mimic | Key distinguishing features |
|---|---|
| Orthostatic hypotension | Sustained BP fall ≥20/10 on standing, not isolated tachycardia. |
| Vasovagal syncope | Episodic reflex faint — later bradycardia with hypotension. |
| Inappropriate sinus tachycardia | Raised resting heart rate even when supine. |
| Thyrotoxicosis / anaemia / phaeo | Screen and exclude on bloods; treat the underlying cause. |
| Anxiety / panic | Frequently coexists and amplifies; rarely explains the full orthostatic physiology. |

► ***Red flags — Syncope with injury or on exertion · chest pain or exertional dyspnoea · tachycardia present when supine · new focal neurology · marked weight loss or drenching sweats. Each points away from simple POTS — investigate for cardiac, endocrine or central disease before locking the diagnosis.***

## Management — stepped care

| Step | Intervention | Practice principles |
|---|---|---|
| Foundation (all) | Fluid 2–3 L/day, salt 10–12 g/day, waist-high compression 30–40 mmHg, head-up bed tilt | Every patient; avoid prolonged standing, heat and large meals |
| Reconditioning | Graded exercise — recumbent/aquatic first → upright aerobic → resistance (CHOP) | Single most effective intervention; pace carefully or symptoms flare |
| β-blocker / ivabradine | Propranolol 10–20 mg or ivabradine | Hyperadrenergic / high-HR; avoid in neuropathic/hypovolaemic |
| Volume expansion | Fludrocortisone 0.05–0.2 mg or desmopressin | Hypovolaemic; monitor BP, $K^+$ and oedema |
| Vasoconstriction | Midodrine + pyridostigmine; droxidopa if refractory | Neuropathic / low-flow subtype |
| Autoimmune / mast cell | IVIG / immunotherapy; antihistamines + mast cell stabilisers | Antibody-positive or mast cell activation overlap |

***Pearl — Reconditioning beats any drug. Start recumbent or aquatic and progress slowly — rushing upright exercise flares symptoms. Medication is the add-on, chosen by subtype and reassessed every 4–8 weeks.***

## The four POTS subtypes — quick reference

| Subtype | Signature | First-line |
|---|---|---|
| Neuropathic | Partial sympathetic denervation of legs; abnormal QSART; SFN on biopsy | Midodrine, pyridostigmine, compression |
| Hyperadrenergic | Standing noradrenaline over 600 pg/mL; tremor; BP rises on standing | Low-dose β-blocker, clonidine |
| Hypovolaemic | Reduced plasma volume; low renin–aldosterone | Fludrocortisone, fluid + salt |
| Autoimmune / post-COVID | Adrenergic & ganglionic AChR antibodies; post-infectious; MCAS | IVIG / immunomodulation; treat MCAS |

## Counselling and follow-up

► **Generally good prognosis; most improve substantially over months to years with coordinated care.**
► **Post-viral and adolescent onset carry the most favourable outlook; many young patients recover.**
► **Secondary anxiety is common and treatable — do not relabel POTS as primary anxiety.**
► **Reassess every 4–8 weeks while titrating; track symptoms, HR/BP, quality of life and complications.**
► **Discharge with a relapse-prevention plan; relapse follows illness, inactivity or high stress.**

## Special populations — adolescents & pregnancy

Adolescents frequently improve markedly with growth and structured reconditioning. Pregnancy is usually well tolerated but requires medication review — fludrocortisone and beta-blockers need individualised decisions — with closer monitoring through the postpartum period. Strong overlaps with joint hypermobility (hEDS) and mast cell activation should be screened for and managed in parallel.

*Key references — Sheldon RS et al. Heart Rhythm 2015;12:e41–63 · Raj SR et al. Circulation 2005;111:2734–40 · Freeman R et al. Auton Neurosci 2011;161:46–8 · Bryarly M et al. J Am Coll Cardiol 2019;73:1207–28 · Vernino S et al. Auton Neurosci 2021;235:102828.*