ORTHOSTATIC HYPOTENSION CHEAT SHEET

Orthostatic Hypotension — Clinician Cheat Sheet

Vestibular Physician quick reference · Confirm with a properly performed active stand test (readings to 10 minutes) before attributing dizziness to OH.

► Why OH matters

A common, treatable, and frequently missed cause of dizziness, presyncope, and falls. Prevalence rises from ~5% under 50 to 20–30% over 70 and higher in residential care. An independent predictor of falls, fractures, cognitive decline, and all-cause mortality — and the multisensory mimic most often overlooked when the history is not deliberately positional.

► When to apply this work-up

- ► Dizziness, lightheadedness, presyncope, or unsteadiness that is worse on standing, after meals, in heat, or first thing in the morning.
- ► Unexplained falls or syncope in older or autonomically-vulnerable patients (Parkinson's, diabetes, MSA, amyloid).
- ► A blunted heart-rate rise, supine hypertension, or neurological signs — reclassify as neurogenic and broaden the work-up.

► Mechanism — the autonomic reflex arc

Layer	Mechanism	Clinical relevance
Gravitational challenge	500–1000 mL pools in the legs on standing; venous return and cardiac output fall	The stimulus every reflex must counter
Baroreflex arc	Carotid/aortic baroreceptors → NTS/RVLM → sympathetic activation + parasympathetic withdrawal	Restores BP within 3–5 s
Non-neurogenic failure	Intact arc overwhelmed — volume depletion, venous pooling, deconditioning, drugs	Compensatory tachycardia preserved
Neurogenic failure	Efferent sympathetic damage — central, peripheral, or ganglionic	HR rise blunted < 0.5 bpm/mmHg — the key discriminator

► Diagnostic criteria — the five orthostatic syndromes

Syndrome	Defining criteria	Timing / clue
Classical OH	SBP ↓ ≥ 20 or DBP ↓ ≥ 10 mmHg within 3 min of standing	Commonest type
Initial OH	Transient SBP ↓ ≥ 40 / DBP ↓ ≥ 20 mmHg within 15 s	Post-meal/exertional; needs beat-to-beat
Delayed OH	Same fall as classical but only after 3 min	Missed on short tests; elderly
Neurogenic OH	OH criteria + HR rise < 15 bpm (< 0.5 bpm/mmHg BP fall)	Autonomic efferent failure
POTS	HR rise ≥ 30 bpm (≥ 40 if < 19) in 10 min, no sustained BP fall	Younger, female predominance

Pearl — *Measure to 10 minutes. A 2-minute protocol misses delayed OH and under-diagnoses the condition.*

► Investigations — anchored to the question

Test	Purpose	When to order
Active stand test	Confirm OH; assign subtype by timing of the nadir	First-line, every patient
NASA lean test	Non-laboratory alternative; good yield for delayed / neurogenic	When AST equivocal or prolonged standing needed

Tilt-table + beat-to-beat BP	Symptoms without a bedside fall; syncope; initial OH	Discordant cases
Autonomic profiling (Valsalva, deep breathing, plasma noradrenaline)	Separate neurogenic vs non-neurogenic; localise lesion	Suspected neurogenic OH
Bloods · ECG · medication review	Reversible causes — anaemia, glucose, adrenal, arrhythmia, culprit drugs	All new presentations

Pearl — *The heart-rate-to-blood-pressure ratio is the single best bedside discriminator of neurogenic OH.*

► Differential diagnosis

Mimic	Key distinguishing features
Vasovagal syncope	Sudden later fall with bradycardia and a prodrome
POTS	Tachycardia ≥ 30 bpm without a sustained BP fall
Cardiac (arrhythmia, AS, HF)	Exertional, structural signs, abnormal ECG
Post-prandial hypotension	Symptoms 30–60 min after meals
PPPD / primary vestibular	Positional but not BP-dependent; frequently coexists

⚠ **Red flags —** New neurogenic OH without a cause → MRI brain/spine, full autonomic panel, paraneoplastic + amyloid screen. Syncope with exertion or cardiac features → cardiology. Rapid neurological progression → urgent neurology.

► Management — stepwise algorithm

Tier	Intervention	Practice principles
Step 1 — Reversible causes	Stop/replace culprit drugs; correct dehydration, anaemia, adrenal insufficiency, arrhythmia	Always first
Step 2 — Non-pharmacological (all patients)	Salt + fluid, 500 mL water bolus before triggers, head-up bed tilt, abdominal binder, counter-manoeuvres	Highest-yield; patient education prevents falls
Step 3 — First-line drugs	Fludrocortisone 50–100 mcg/day OR midodrine 2.5–10 mg TDS	Monitor electrolytes/supine BP; midodrine not before lying
Step 4 — Neurogenic add-on	Droxidopa 100–300 mg TDS (noradrenaline precursor)	For confirmed neurogenic OH
Step 5 — Refractory / complex	Autonomic neurology referral	Titrate to standing BP, symptoms, and falls

Pearl — *Treat standing BP and falls, not the number on the supine cuff. Accept mild supine hypertension as the trade-off.*

► Special populations & supine hypertension

Supine hypertension coexists with neurogenic OH in up to 50% of older patients — the central therapeutic dilemma. Treat standing pressure while accepting modest supine elevation; use short-acting nocturnal antihypertensives and head-up tilt. Beware polypharmacy and post-prandial hypotension; falls dominate the morbidity.

► Counselling & follow-up

► Educate on triggers (rising, heat, meals, alcohol) and counter-manoeuvres — the highest-yield falls prevention.

► Home lying/standing BP diary; review medicines at every visit.

► Discharge to GP once BP targets are stable and falls risk is controlled; shared care with autonomic neurology and geriatrics as needed.

Key references — Freeman R et al. Consensus statement on OH, POTS and reflex syncope. Auton Neurosci 2011 · Gibbons CH et al. The recommendations of a consensus panel for neurogenic OH. J Neurol 2017 · Kaufmann H et al. Neurogenic OH. Nat Rev Dis Primers 2017 · Fedorowski A. Orthostatic hypotension. J Intern Med 2019 · Sheldon RS et al. Syncope guideline. Heart Rhythm 2015.