

# Syncope & Transient Loss of Consciousness

A Clinical Diagnostic Blueprint

Based on Med2Date Clinical Guidelines

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Defining TLOC | Risk Stratification | Diagnostic Workup | Specific Management

# The Clinical Problem: Defining TLOC

**Transient Loss of Consciousness (TLOC)** is defined as a spontaneous, temporary **loss of consciousness** characterized by rapid onset, short duration, and complete spontaneous recovery.

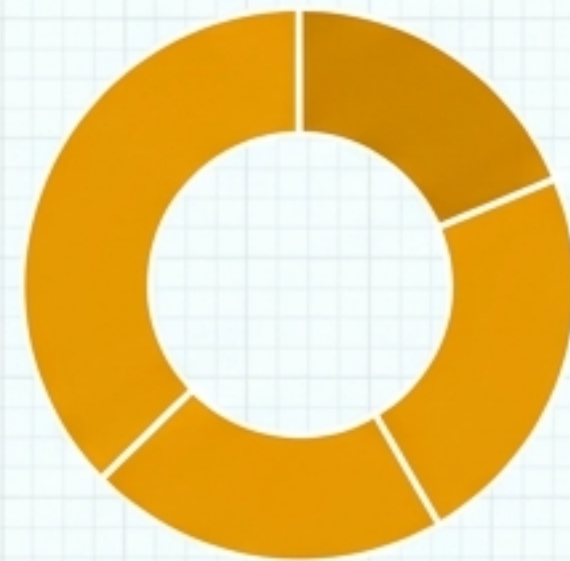
**Syncope** is TLOC specifically caused by global cerebral hypoperfusion.



**Scale:** >100,000 annual ED presentations in Australia. Accounts for 1-3% of all ED visits and up to 6% of acute medical admissions.

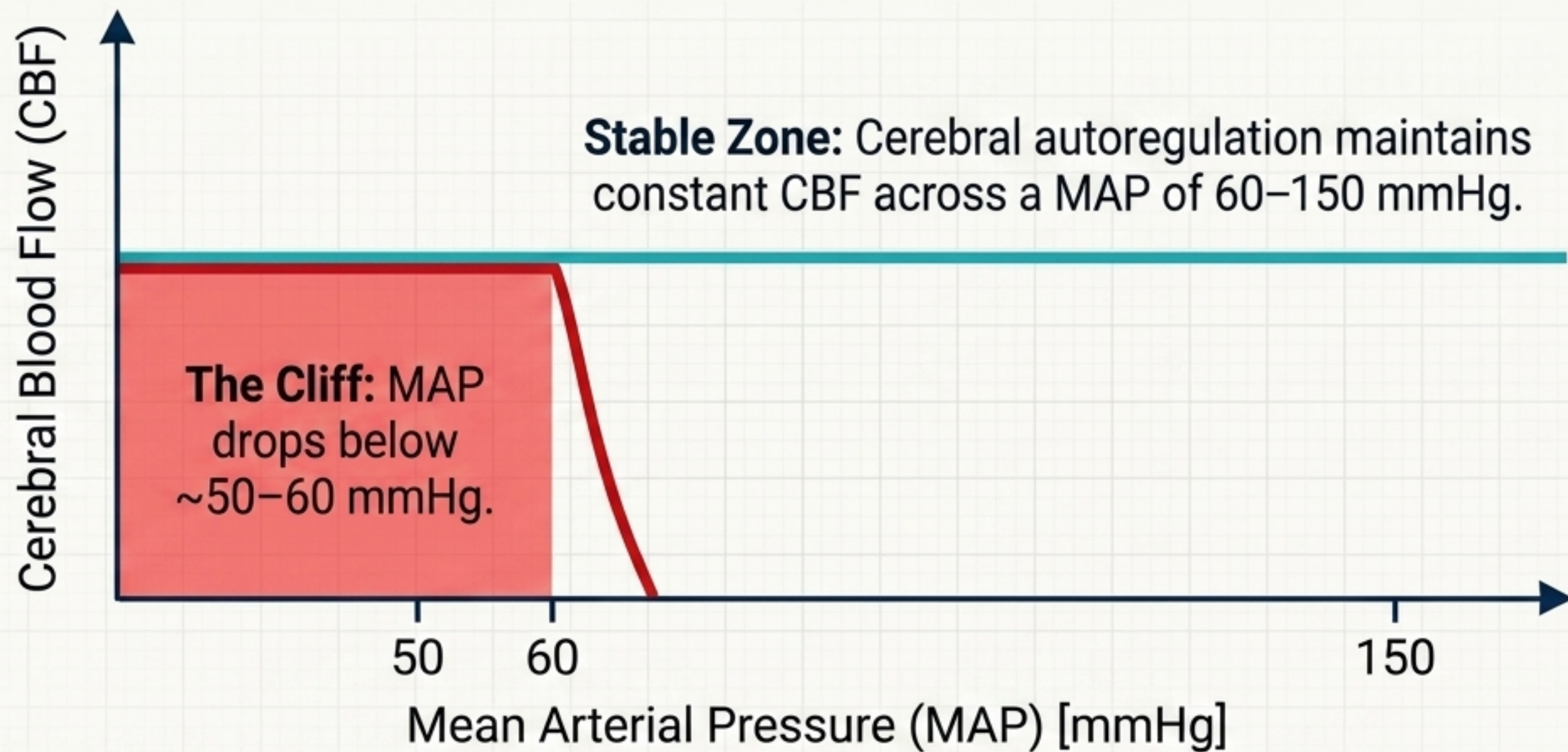


**Cost:** ~\$1.4 Billion annual direct healthcare cost.



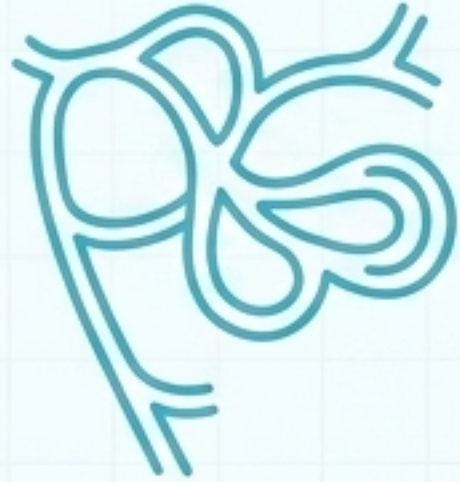
**The Diagnostic Pitfall:** 20% of patients initially diagnosed with syncope actually have an alternative diagnosis (seizure, concussion, intoxication). **40%** remain undiagnosed after initial ED evaluation.

# The Physiology of Collapse



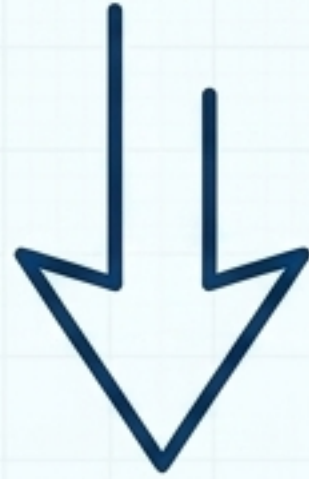
**The Consequence:** Global cerebral hypoperfusion lasting >6–8 seconds results in clinical syncope.

# The Three Pillars of Syncope



## Reflex (Vasovagal)

- **Mechanism:** Inappropriate vasodilation  $\pm$  bradycardia (Bezold-Jarisch reflex).
- **Proportion:** ~40–60%
- **Mortality Risk:** Very Low



## Orthostatic Hypotension

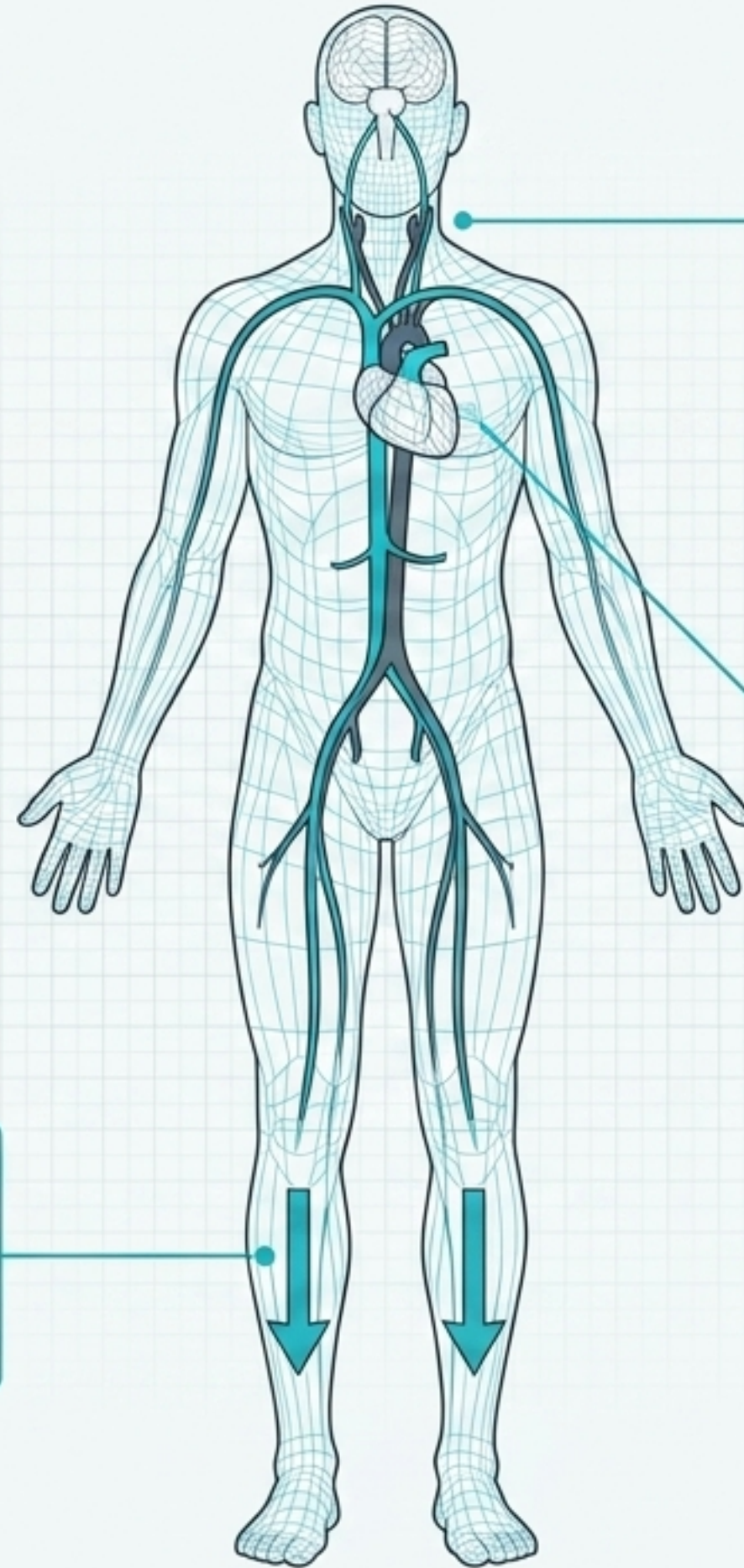
- **Mechanism:** Inadequate compensatory vasoconstriction on standing.
- **Proportion:** ~10–30%
- **Mortality Risk:** Low–Moderate



## Cardiac Syncope

- **Mechanism:** Arrhythmia, structural disease, or acute cardiac event reducing output.
- **Proportion:** ~10–20%
- **Mortality Risk:** High (Up to 30% at 1 year)

# The Hemodynamic Trap




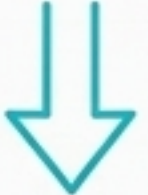

**Failure of the baroreceptors** in the carotid sinus and aortic arch to trigger sympathetic vasoconstriction results in sustained hypotension.

**Causes:** Autonomic neuropathy, volume depletion, vasodilatory meds.

**Reflex Mechanism:** Triggered by prolonged standing, pain, or emotion. The Bezold-Jarisch reflex mediates paradoxical bradycardia and vasodilation via vagal afferents.

**Orthostatic Mechanism:** Gravity-dependent pooling of 500–1000 mL of blood occurs in the lower limbs on standing, reducing venous return.

# The Clinical Presentation Matrix

	 <b>Reflex (Vasovagal)</b>	 <b>Orthostatic Hypotension</b>	 <b>Cardiac Syncope</b>
<b>Position</b>	Prolonged standing, sitting upright.	Immediately on standing.	Supine or during exertion.
<b>Prodrome</b>	Nausea, warmth, pallor, diaphoresis (>30s).	Gradual lightheadedness, visual greying.	None or brief palpitations.
<b>Onset</b>	Gradual.	Seconds after postural change.	Sudden.
<b>Recovery</b>	Rapid, no confusion.	Rapid once supine.	Prolonged if low output.
<b>Triggers</b>	Pain, emotion, warmth, venepuncture.	Dehydration, morning, medications.	Exertion, preceding chest pain.

# Diagnostic Red Flags: Syncope vs. Seizure

## Syncope

Lateral tip (rare)

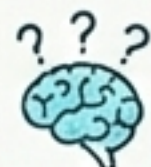
<30 seconds typically

Brief myoclonic jerks (<15 sec)

Rapid recovery, no confusion

Pallor

Rare (urinary only)



**Tongue Biting**

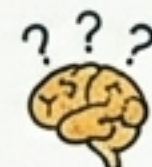
**Duration of LOC**

**Motor Movements**

**Postictal State**

**Complexion**

**Incontinence**



## Seizure

Lateral tongue (highly specific)

>1–2 minutes

Sustained >15 seconds, rhythmic tonic-clonic

Prolonged (>5 min) confusion or drowsiness

Cyanosis during event

Urinary ± faecal (more common)

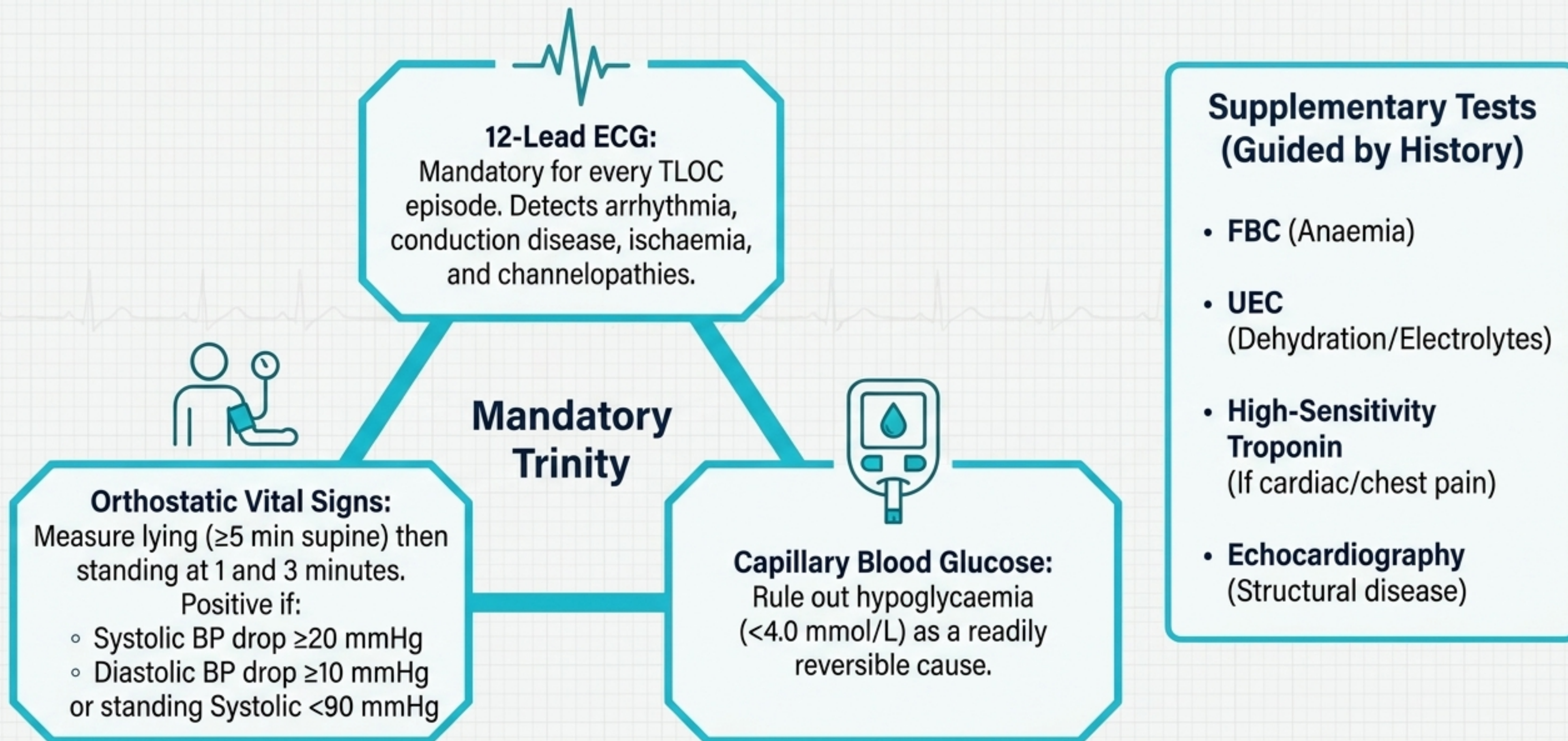


## High-Risk Clinical Features

**Admit and investigate urgently if ANY of the following are present:**

- ✓ Syncope occurring during exertion or while supine.
- ✓ Family history of sudden cardiac death at <40 years of age.
- ✓ New or unexplained dyspnoea.
- ✓ Persistent hypotension (systolic <90 mmHg).
- ✓ Syncope associated with chest pain or palpitations.
- ✓ Known severe structural heart disease (e.g., severe aortic stenosis, hypertrophic cardiomyopathy) or reduced ejection fraction.

# The Essential Basic Workup



# The ECG Mandate

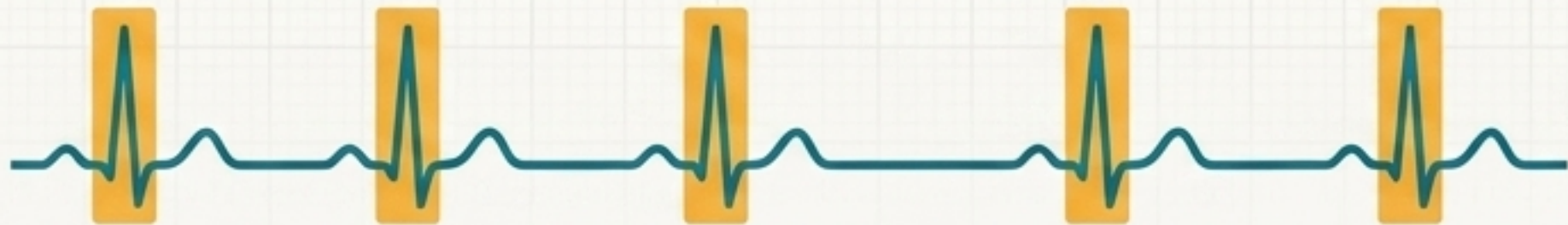
Abnormalities that mandate urgent cardiology referral.



**Prolonged QTc:** QTc >500 ms.  
(Risk of Torsades de Pointes).



**Brugada Pattern:** Coved  
ST-segment elevation in V1-V3.



**AV Block:** High-degree (Mobitz II  
or Complete Heart Block).



**Ventricular Pre-excitation:** Delta  
waves (Wolff-Parkinson-White).

Note: Also screen for sustained VT and Q waves suggesting prior infarction.

# Neuroimaging & EEG: Avoid Routine Use

**Core Rule:** Do **NOT** routinely order neuroimaging or EEG for syncope. Diagnostic yield is <1% without focal neurological signs.



When to Actually Order	
<b>CT Brain</b> (Non-contrast)	<ul style="list-style-type: none"><li>• Head injury from syncope</li><li>• Focal neuro signs</li><li>• Anticoagulated trauma</li><li>• Suspected SAH (Subarachnoid Hemorrhage)</li></ul>
<b>MRI Brain</b>	<ul style="list-style-type: none"><li>• Focal neuro deficit</li><li>• Suspected posterior fossa lesion</li><li>• Recurrent unexplained TLOC with seizure features</li></ul>
<b>EEG</b>	<ul style="list-style-type: none"><li>• Prolonged postictal confusion (&gt;5 min)</li><li>• Witnessed tonic-clonic activity &gt;15 sec</li><li>• Lateral tongue biting</li></ul> <p>(Never for classic vasovagal/orthostatic or brief myoclonic jerks)</p>

# Risk Stratification & Disposition Funnel

## Low Risk (Typical Vasovagal)

- **Features:** Clear prodrome, classic trigger, normal ECG, age <40, no injury. CSRS  $\leq -1$ .
- **Disposition:** Discharge with GP follow-up in 1–2 weeks.

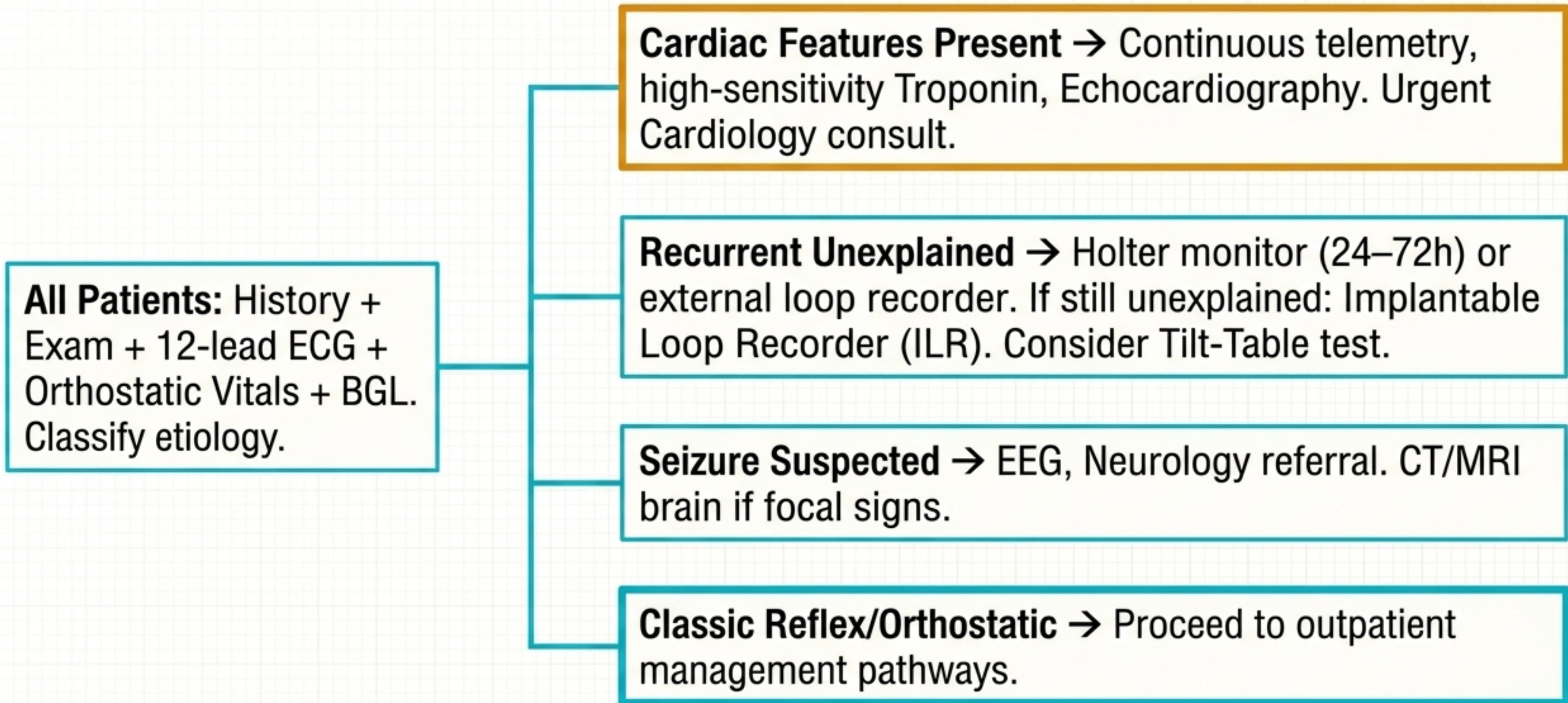
## Moderate Risk (Uncertain Aetiology)

- **Features:** Age >40, single non-specific ECG finding, medication-related, no high-risk flags. CSRS 0–3.
- **Disposition:** ED observation (6-12 hours)  $\pm$  short-stay unit, outpatient investigation (Holter).

## High Risk (Suspected Cardiac)


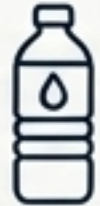


- **Features:** Exertional/supine syncope, abnormal ECG, structural disease, recurrent episodes. CSRS  $\geq 4$ .
- **Disposition:** Inpatient admission, telemetry bed, urgent cardiology consultation.

# The Diagnostic Algorithm




# Management Blueprint: Reflex (Vasovagal) Syncope

## Non-Pharmacological (First-Line)


- ✓ - **Education & Trigger avoidance** (prolonged standing, warm environments). 
- ✓ - **Fluid loading:** 2–3 L water/day (1.5 L bolus before standing). 
- ✓ - **Dietary Salt:** 6–10 g NaCl/day (if no contraindications). 
- ✓ - **Physical Counterpressure Manoeuvres (PCM):** Leg crossing with muscle tensing, squatting. 

## Pharmacological Options (For recurrent failure)



**Midodrine (Gutron®):**  
 $\alpha_1$ -agonist. 2.5–10 mg PO TDS.   
Note: PBS Authority Required (S100).  
Avoid within 4h of bedtime.



**Fludrocortisone:**  
Mineralocorticoid.   
0.1–0.2 mg PO mane.  
Note: PBS General Benefit.  
Monitor K<sup>+</sup> and fluid retention.

# Management Blueprint: Orthostatic & Iatrogenic Syncope

**Core Orthostatic Management:** Treat underlying cause, volume repletion, waist-high elastic compression stockings, elevate head of bed 10-20°, rise slowly.

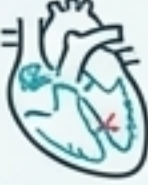
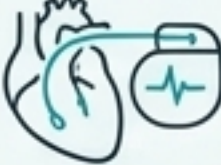
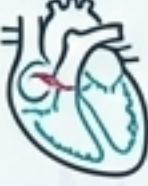







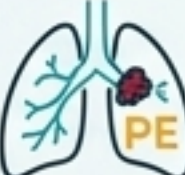

## The Medication Implication Matrix

Medication Class	Mechanism	Clinical Action
Antihypertensives (Diuretics, ACEi, CCBs)	Excessive vasodilation/volume depletion	Dose reduction; switch to shorter-acting agent.
Psychotropics (TCAs, SSRIs, Antipsychotics)	Orthostasis, QT prolongation	Review indication, monitor QTc.
Anti-anginals (Nitrates, $\beta$ -blockers)	Vasodilation, bradycardia	Adjust dose; avoid abrupt $\beta$ -blocker cessation.
Phosphodiesterase-5 inhibitors	Vasodilation	<b>Absolute contraindication</b> with nitrates.
Opioids	Vasodilation	Reduce dose, consider non-opioid analgesia.

# Management Blueprint: Cardiac Syncope





**Warning:** Cardiac syncope requires definitive treatment of the underlying cause. Empirical treatment without diagnosis is dangerous. Always involve cardiology.

Pathology			Definitive Intervention	
	Sick Sinus Syndrome / High-Degree AV Block	→		Permanent Pacemaker
	SVT (AVNRT/AVRT)	→		Catheter Ablation or rhythm control
	Ventricular Tachycardia (with EF $\leq 35\%$ )	→		ICD Implantation
	Hypertrophic Cardiomyopathy (High risk SCD)	→		ICD Implantation
	Severe Aortic Stenosis	→		Aortic Valve Replacement (Surgical/TAVI)
	Acute Pulmonary Embolism	→		Anticoagulation; Thrombolysis if massive

# Driving Restrictions & Occupational Hazards

Austroads Guidelines (2022)

	 <b>Private</b>	 <b>Commercial</b>
<b>Single Vasovagal</b>	No restriction.	1 month off.
<b>Recurrent Vasovagal</b>	Cease until controlled (min 1 month).	<b>Min 3 months off</b> (requires 12mo recurrence-free).
<b>Unexplained Syncope</b>	Min 1 month off.	<b>Min 6 months off</b> (requires specialist assessment).
<b>Cardiac Syncope</b>	Min 1 month off.	<b>Min 6–12 months off</b> (specialist review required).
<b>Orthostatic</b>	Cease driving until cause identified and treated.	



**Occupational Warning:** Safety-critical roles (pilots, heavy machinery, crane operators) require formal occupational health assessment before returning to work.

# Clinical Nuance in Special Populations

## Pregnancy:

**Supine hypotension syndrome**  
(2<sup>nd</sup>/3<sup>rd</sup> trimester aortocaval compression)



**Action: Left lateral positioning.**

⚠️ Exclude PE/amniotic fluid embolism.

⚠️ Avoid fludrocortisone.

## Paediatrics:

Vasovagal accounts for >80%.

⚠️ **Red Flags:** Exertional syncope, FHx SCD <40.

**Action:** ECG, BGL.

**Breath-holding spells** in toddlers are benign.

**Midodrine** requires specialist guidance.

## Elderly (>65):

Often multifactorial (polypharmacy, postprandial, autonomic).

May present as 'falls.'

**Action: Carotid sinus massage** (if >50, under ECG monitoring, no bruits).

Deprescribe using **STOPP/START criteria**.



## Renal Impairment:

**Uraemic autonomic neuropathy,** intradialytic hypotension (ultrafiltration >10mL/kg/h).

**Action:** Review dry weight.

⚠️ **Fludrocortisone high risk** (fluid/K<sup>+</sup> retention).

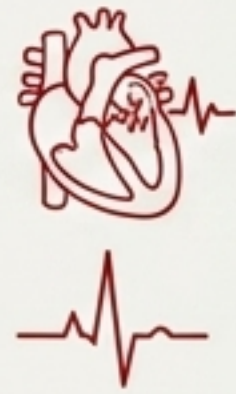
⚠️ Watch for **QT prolongation** from electrolyte shifts.



# Aboriginal & Torres Strait Islander Health Considerations

## Cardiovascular Disease Burden

- ✓ **Rheumatic Heart Disease (RHD):** Extremely high prevalence. Valvular disease (mitral stenosis) and **arrhythmias** are key cardiac causes of syncope. Early Echo mandated.
- ✓ **Diabetes: T2DM** prevalence is 3-4x higher; **autonomic neuropathy** causing orthostasis is heavily underrecognized.
- ✓ **Chronic Kidney Disease:** 4x higher risk. **Volume-related syncope** and **electrolyte disturbances** are frequent.



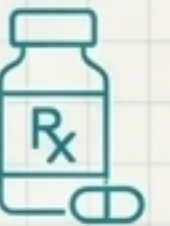
## Remote Access Barriers

- Lack of continuous ECG/Echo** in remote settings **necessitates aeromedical transfer** for moderate/high-risk patients.
- Ensure **culturally safe transport**.

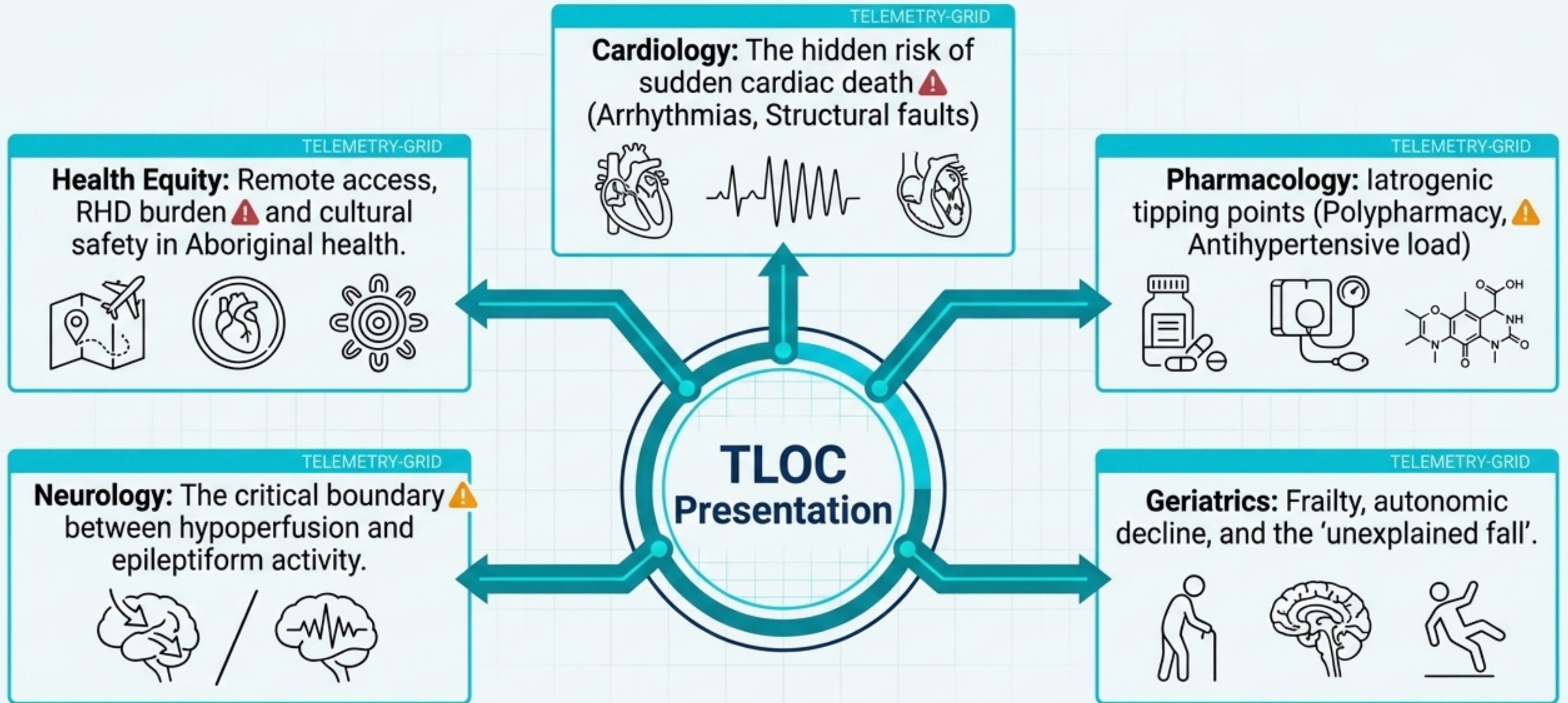


## Systemic Support

- Utilize **Section 100 Close the Gap** scripts for medication access (Midodrine/Fludro).
- Coordinate **ICD/Pacemaker follow-up** via **telecardiology** and Aboriginal community-controlled health services (ACCHS).



# Synthesis: The Syncope Ecosystem



**Core Insight: Syncope is not a final diagnosis—it is a gateway symptom demanding holistic, systemic medical thinking.**

# Key References & Clinical Guidelines

Med2Date Clinical Guidelines: Syncope & Transient Loss of Consciousness (Updated May 2026).

Shen W-K, et al. 2017 ACC/AHA/HRS guideline for the evaluation and management of patients with syncope. *Circulation*. 2017.

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Austroads. *Assessing Fitness to Drive*. 2022.