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Situational Syncope Across Diverse Triggers: A Diagnostic and Management challenge: Case Series

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ABSTRACT

Situational syncope is a subtype of neurally mediated reflex syncope triggered by specific activities such as micturition, defaecation, coughing, swallowing, or laughter, but remains under-recognised in clinical practice. We present a case series of six adults with syncope or presyncope associated with diverse triggers, including laughter, micturition, motion sickness, defaecation, swallowing, and coughing, highlighting the heterogeneity of presentation.

All patients underwent targeted evaluation with electrocardiography and echocardiography, with selective use of tilt-table testing. No structural heart disease or sustained arrhythmia was identified, supporting a reflex mechanism in all cases. Management was conservative, focusing on trigger avoidance, lifestyle modification, and patient education, with no recurrence observed during follow-up.

This series emphasises the central role of detailed history-taking in establishing the diagnosis of situational syncope and avoiding unnecessary investigations. Recognition of both common and less well-described triggers, alongside an understanding of the underlying autonomic mechanisms, enables accurate diagnosis and effective patient-centred management. Increased awareness of situational syncope may improve clinical confidence, reduce diagnostic uncertainty, and optimise outcomes in patients presenting with transient loss of consciousness.

Keywords: *Syncope, Trigger, Situational Syncope, Lifestyle Advice*

Introduction

Syncope, a transient loss of consciousness caused by global cerebral hypoperfusion, is a common presentation in both primary and secondary care. There are three main categories of syncope: reflex, orthostatic, and cardiac syncope.

Situational syncope represents an under-recognised subtype of reflex syncope characterised by specific physiological triggers such as urination, defecation, coughing or swallowing. 10-15% of all syncope cases are due to situational syncope (Sutton, 2017). Each trigger induces a transient vasovagal reflex leading to bradycardia and occasional hypotension (Freeman *et al*., 2011).

It is a generally benign condition but the lack of training and awareness in this relatively common presentation prompts several cardiac and neurological investigations, whilst the patients remain symptomatic with reduced quality of life (Mathias *et al*., 2001). The following six cases of situational syncope reflect its autonomic physiology, and the diagnostic power of the clinical history (Sutton, 2017).

Cases Presentation

We present six patients, who were evaluated in the cardiology outpatient clinic for transient loss of consciousness or presyncopal episodes triggered by identifiable physiological activities (Table 1).

Table 1: Summary of cases.

Case	Age/Sex	Trigger	Clinical features	Key findings	Management
1	46/M	Laughter	Brief LOC during intense laughter; full recovery	ECG: sinus rhythm, Echo: no structural abnormality	Hydration, reduce alcohol
2	50/F	Micturition	Dizziness/light-headedness post-voiding	ECG, Holter & Echo normal	Hydration, gradual standing
3	35/F	Motion sickness	LOC during travel with nausea, diaphoresis	ECG: sinus rhythm with minor non-specific T-wave inversion	Trigger avoidance, reassurance
4	70/F	Defecation	Syncope during straining; nausea, dizziness	ECG: anterior T-wave inversion; Echo: normal	Hydration, salt, stool softening
5	59/M	Swallowing	LOC after swallowing liquids	ECG: sinus rhythm, poor R progression	Behavioural modification
6	44/M	Cough	LOC during coughing fits	Tilt with cough provocation: vasodepressor response	Cough control, hydration

1. Laughter-Induced Syncope

Patient background: A 46-year-old man with no prior cardiovascular history presented following a syncopal episode.

Clinical findings: He collapsed during a bout of intense laughter at a social event. The episode lasted approximately 10 seconds and was followed by complete spontaneous recovery without confusion or convulsive activity. He reported occasional light-headedness during laughter. His history was notable for high alcohol intake (30–35 units/week) and inconsistent hydration.

Timeline: Symptoms occurred immediately during intense laughter, with rapid recovery within seconds.

Diagnostic assessment: Blood pressure was 128/80 mmHg and heart rate 72 bpm. Resting ECG demonstrated sinus rhythm (PR interval 186 ms, QTc 426 ms). Transthoracic echocardiography (TTE) showed preserved left ventricular systolic function (LVEF 71%) with no structural abnormalities. Forty-eight-hour Holter ECG monitoring confirmed sinus rhythm (mean HR 67 bpm), no pauses, sustained or non-sustained SVT or BCT with rare ectopic beats.

Therapeutic intervention: Lifestyle modification was advised, including improved hydration and reduction in alcohol intake.

Follow-up and outcomes: The patient remained asymptomatic with no recurrence at 6-month follow-up.

2. Micturition Syncope

Patient background: A 50-year-old woman with no cardiovascular risk factors presented with recurrent presyncopal episodes.

Clinical findings: She experienced dizziness and light-headedness immediately after voiding, particularly on rising from the toilet at night. She had not experienced complete loss of consciousness.

Timeline: Symptoms occurred immediately post-micturition, especially with rapid standing.

Diagnostic assessment: Clinical examination, ECG, TTE, and 24-hour Holter ECG monitoring were unremarkable.

Therapeutic intervention: She was advised to maintain adequate hydration (2.5–3 L/day), avoid alcohol and caffeine, and rise slowly after urination.

Follow-up and outcomes: She remained symptom-free at 8-month follow-up.

3. Travel Sickness Syncope

Patient background: A 35-year-old woman reported recurrent syncope since adolescence.

Clinical findings: Episodes were consistently triggered by travel (train, ferry, amusement rides) and began with nausea, diaphoresis, and visual dimming, followed by transient loss of consciousness lasting less than 30 seconds.

Timeline: Symptoms developed during motion exposure and resolved rapidly after the episode.

Diagnostic assessment: Blood pressure was 111/70 mmHg and heart rate 63 bpm. ECG demonstrated sinus rhythm (PR 152 ms, QTc 412 ms) with minor T-wave inversion, which was considered a non-specific finding in the absence of structural heart disease and not suggestive of underlying pathology. Given the long-standing, reproducible, and benign nature of symptoms without high-risk features, further investigations were deferred.

Therapeutic intervention: Management consisted of reassurance, education, and advice on trigger avoidance.

Follow-up and outcomes: She remained asymptomatic at 12-month follow-up.

4. Defecation Syncope

Patient background: A 70-year-old woman with a history of mitral valve repair presented with recurrent syncope.

Clinical findings: She described four syncopal episodes occurring during defecation over a 12-month period, one resulting in facial bruising. Prodromal symptoms included dizziness, nausea, and blurred vision. Her comorbidities included an incisional hernia and dyslipidaemia.

Timeline: Symptoms occurred during straining at defecation, with rapid recovery following the episodes.

Diagnostic assessment: Blood pressure was 129/84 mmHg and heart rate 89 bpm. ECG demonstrated sinus rhythm with QTc 406 ms and anterior T-wave inversion. TTE confirmed normal left ventricular function and satisfactory mitral valve repair, with no evidence of valve-related haemodynamic contribution to the syncopal episodes.

Therapeutic intervention: She was advised on adequate hydration (2–2.5 L/day), liberal salt intake, stool-softening diet, and avoidance of prolonged straining.

Follow-up and outcomes: No further episodes were reported at 6-month follow-up.

5. Swallowing (Deglutition) Syncope

Patient background: A 59-year-old man with a history of hypertension and hyperlipidaemia presented with recurrent syncope.

Clinical findings: He experienced four episodes of transient loss of consciousness immediately after swallowing liquids such as water or tea. Each episode was preceded by a choking sensation and lasted approximately 10–15 seconds.

Timeline: Symptoms occurred immediately following swallowing, with rapid recovery.

Diagnostic assessment: Physical examination was normal (BP 137/86 mmHg, HR 75 bpm). ECG showed sinus rhythm (PR 142 ms, QTc 404 ms) with poor anterior R-wave progression. Further investigations, including TTE and 48-hours Holter ECG monitoring, did not identify structural heart abnormality or arrhythmia. Otolaryngology and neurology assessments were unremarkable.

Therapeutic intervention: Medication review was undertaken, including assessment of atenolol use as a potential contributing factor; however, no adjustment was required as symptoms improved. Behavioural advice was provided, including slow ingestion of fluids in a seated position.

Follow-up and outcomes: No recurrence was observed at 4-month follow-up.

6. Cough Syncope

Patient background: A 44-year-old man with no significant past medical history presented with syncope.

Clinical findings: He developed transient loss of consciousness during bouts of coughing following a viral respiratory infection. Each episode lasted less than 10 seconds and resolved spontaneously.

Timeline: Symptoms occurred during coughing episodes and resolved rapidly.

Diagnostic assessment: Clinical examination showed normal cardiovascular findings (BP 124/78 mmHg, HR 82 bpm). TTE demonstrated no structural and functional heart disease. Head-up tilt testing with cough provocation reproduced presyncopal symptoms and a fall in systolic blood pressure from 163 mmHg to 84 mmHg, confirming a vasodepressor response.

Therapeutic intervention: He was advised on cough control, adequate hydration, and avoidance of excessive straining.

Follow-up and outcomes: Complete symptom resolution was achieved by 3-month follow-up.

Discussion

General Discussion on situational syncope

History and Pathophysiology:

We present six cases of situational syncope triggered by laughter, micturition, travel sickness, defecation, swallowing, and coughing, illustrating the heterogeneity of clinical presentation within a single diagnostic entity. Despite differing triggers, all cases demonstrated a consistent pattern of transient loss of consciousness or presyncope with rapid spontaneous recovery and no post-ictal features. Awareness of both common and less recognised triggers is essential for accurate diagnosis and effective lifestyle advice (Fig. 1). A detailed patient history should identify the specific situational trigger and associated prodromal symptoms such as nausea, diaphoresis, or light-headedness, which help distinguish situational syncope from other causes of transient loss of consciousness (Parry and Tan, 2010).

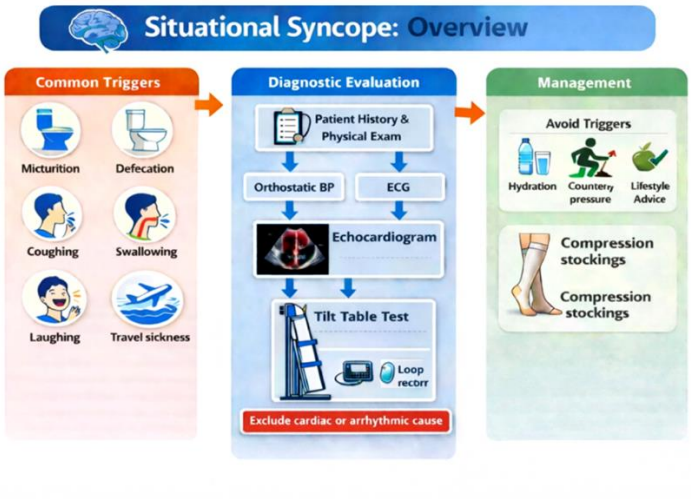


Figure 1:

Legend:

Schematic overview of situational syncope illustrating common triggers, recommended diagnostic evaluation, and management principles. Common triggers include micturition, defecation, coughing, swallowing, laughing, and travel sickness. Diagnostic evaluation begins with patient history and physical examination, followed by orthostatic blood pressure measurement and electrocardiogram (ECG). Echocardiography and tilt-table testing may be used in selected cases, with ambulatory rhythm monitoring (e.g., loop recorder) when arrhythmia is suspected. Management focuses on trigger avoidance, adequate hydration, physical counterpressure manoeuvres, lifestyle advice, and compression stockings in selected patients.

Situational syncope is a subtype of neurally mediated reflex syncope in which specific triggers such as micturition, defecation or coughing activate autonomic reflexes that abruptly alter cardiovascular control and lead to transient cerebral hypoperfusion and loss of consciousness (Freeman *et al*., 2011, Kapoor, 2000). The underlying mechanism involves abnormal integration of afferent autonomic signals within the brainstem, particularly the nucleus tractus solitarius (NTS) in the medulla. Afferent impulses travel primarily via the glossopharyngeal nerve (cranial nerve IX) and the vagus nerve (cranial nerve X) from baroreceptors and visceral mechanoreceptors (Je, 2016; Brignole *et al*., 2019). These inputs may be triggered by increased intrathoracic pressure (e.g., cough syncope), bladder or rectal distension (micturition/defecation syncope), or oesophageal stimulation (swallow syncope), leading to exaggerated reflex activation in susceptible individuals (Brignole *et al*., 2019; Benditt and Nguyen, 2009). Central processing within the NTS and adjacent medullary autonomic centers results in increased parasympathetic output and withdrawal of sympathetic tone (Je, 2016).

The efferent limb involves heightened activity in the vagus nerve, which releases acetylcholine at the sinoatrial (SA) and atrioventricular (AV) nodes, acting on M2 muscarinic receptors to reduce heart rate and atrioventricular conduction (cardioinhibitory response) (Je, 2016). Concurrently, inhibition of sympathetic outflow reduces norepinephrine release at peripheral vascular α_1-adrenergic receptors, producing vasodilation and decreased systemic vascular resistance (vasodepressor response). The combined bradycardia and hypotension result in transient cerebral hypoperfusion and loss of consciousness. In some cases, mechanisms similar to the Bezold–Jarisch reflex, mediated by vagal afferents from cardiopulmonary receptors, may contribute to the autonomic imbalance observed in reflex syncope (Je, 2016; Benditt and Nguyen, 2009).

Epidemiology and triggers:

Reflex syncope accounts for roughly 60 % of all syncope, with situational forms comprising 10–15 % (Sutton, 2017) Prevalence and mechanism vary by trigger (Table 2). The demographic characteristics of our patients broadly align with these patterns, including age-related susceptibility and trigger-specific variation.

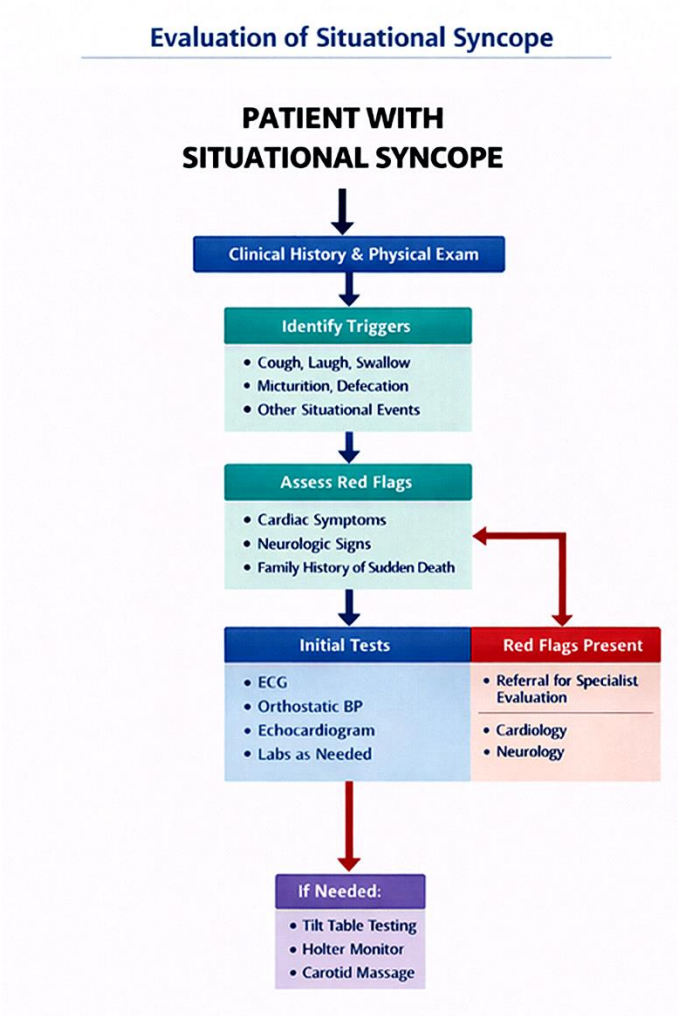
Table 2: The prevalence and Mechanism of Situational Syncope.

Trigger	Approx. prevalence (%)	Mechanism	Typical profile
Micturition	40–55	Valsalva reflex + vagal activation	Middle-aged men
Defecation	10–20	Vagal overactivity during straining	Older adults
Swallowing	5–15	Oesophageal–vagal reflex → bradycardia/asystole	Middle-aged/elderly
Cough/sneeze	4–10	↑ intrathoracic pressure → ↓ venous return	Obese men, COPD
Motion sickness	2–4	Vestibular–vagal reflex	All ages
Laughing/singing/blowing instruments	< 3	Valsalva-like manoeuvre	All ages

There are a few recognised high-risk or contributing factors include dehydration, alcohol excess, medications that impair autonomic compensation (e.g., beta-blockers or vasodilators), and comorbid cardiorespiratory disease (Kapoor, 2000; Brignole *et al*., 2019). Several of these factors were present in our cases, highlighting their potential role in increasing susceptibility to reflex syncope.

Diagnosis

Medical investigations play a crucial role in the assessment of syncope, particularly in distinguishing benign causes such as situational syncope from potentially life-threatening cardiac or neurological conditions, such as epilepsy. The diagnostic approach (Fig. 2) typically begins with a thorough history, physical examination, and ECG, which together can identify the cause in up to 50% of cases (Brignole *et al*., 2019). The presence of a clear trigger, brief duration, rapid recovery, and absence of post-ictal confusion or focal neurological deficit strongly supports a reflex mechanism (Brignole *et al*., 2019; Parry and Tan, 2010).



Figures 2: Diagnostic algorithm.

Legend:

Practical diagnostic algorithm to be followed in diagnosis of situational syncope. Identifying triggers and red flags followed by appropriate investigations are crucial.

The echocardiogram serves as an essential investigation to evaluate for structural heart disease, which is a major determinant of syncope prognosis. It is particularly indicated when the initial assessment or ECG suggests cardiac pathology, such as abnormal heart sounds, evidence of valvular disease, or left ventricular dysfunction. Echocardiography can identify conditions such as aortic stenosis, hypertrophic cardiomyopathy, pulmonary hypertension, or cardiac masses, all of which may predispose to exertional or arrhythmic syncope. However, in situational syncope, echocardiographic findings are typically normal, as the underlying pathophysiology is reflex and autonomic rather than structural. While a few case reports have described transient left ventricular outflow tract obstruction or increased intrathoracic pressure

during cough or micturition that may contribute to syncope, these are rare and not consistent echocardiographic markers (Brignole *et al*., 2019; Sutton and Petersen, 1995). Thus, the primary role of echocardiography in situational syncope is exclusionary—to rule out cardiac causes rather than to confirm the diagnosis itself.

A key diagnostic tool in evaluating reflex and situational syncope is the tilt-table test (TTT). This non-invasive investigation assesses cardiovascular responses to orthostatic stress in a controlled environment. By tilting the patient to 60–70 degrees while continuously monitoring ECG and blood pressure, abnormal reflex responses associated with hypotension, bradycardia, or both can be identified. Pharmacologic provocation (e.g., with nitroglycerin or isoproterenol) may be used to enhance sensitivity. In patients with suspected situational or vasovagal syncope, a positive TTT—characterized by symptom reproduction and hemodynamic changes—confirms a neurally mediated reflex mechanism. While not entirely specific, TTT provides valuable educational and diagnostic insights, helping clinicians differentiate between vasovagal, orthostatic, and psychogenic causes (Task Force for the *et al*., 2009; Shen *et al*., 2017).

Another important investigation in the evaluation of reflex-mediated syncope, particularly in older adults, is carotid sinus massage (CSM). Performed under continuous ECG and blood pressure monitoring, CSM assesses carotid sinus hypersensitivity (CSH)—an exaggerated baroreceptor response that can cause syncope due to excessive vagal activation or vasodilation. A diagnostic response is defined as asystole lasting ≥3 seconds (cardioinhibitory type), a systolic BP fall ≥50 mm Hg (vasodepressor type), or a combination of both (mixed type). The test should be performed cautiously, ideally after excluding significant carotid artery disease via auscultation or Doppler imaging, as it carries a small risk of cerebrovascular complications. CSM is especially useful when the history suggests reflex or unexplained falls in elderly patients, as it can unmask a treatable autonomic disorder contributing to syncope (Brignole *et al*., 2019; Task Force for the *et al*., 2009).

In our case series, all patients had normal baseline investigations, supporting a reflex mechanism and highlighting the importance of clinical history over extensive diagnostic testing.

Management:

Management of situational syncope focuses first on non-pharmacological strategies, as this subtype of reflex syncope is generally benign but can significantly affect quality of life if recurrent episodes occur. A core principle is education and avoidance of known triggers—for example, advising patients to void the bladder before rising from bed at night to reduce micturition syncope, to avoid prolonged straining, or to

minimize situations that provoke cough or swallowing episodes when possible. Increasing fluid and salt intake can help expand intravascular volume and reduce susceptibility to reflex hypotension in some individuals, although specific data in situational syncope are extrapolated from vasovagal management principles. Patients should also be taught physical counterpressure maneuvers (such as leg crossing, squatting, or buttock tensing) at the onset of prodromal symptoms to increase venous return and blood pressure, which has shown efficacy in preventing vasovagal fainting and is conceptually applicable to situational forms (Bayard *et al*., 2023).

In patients with frequent or severe episodes despite trigger avoidance and behavioural measures, pharmacological options may be considered on a case-by-case basis. Medications such as midodrine (an alpha-agonist that raises peripheral vascular tone) or fludrocortisone (a mineralocorticoid that promotes sodium and fluid retention) are sometimes used in recurrent reflex syncope, although evidence specifically in situational syncope is limited and treatment should be individualized after excluding other syncope etiologies (Sheldon *et al*., 2016). In rare and refractory cases where profound bradycardia is demonstrated and correlated with symptoms, cardiac pacing might be considered, particularly in older patients with significant cardioinhibitory responses, but this is uncommon and guided by specialist assessment (Parry and Tan, 2010).

All patients in this series were successfully managed with conservative measures alone, with no recurrence during follow-up, supporting current recommendations for trigger avoidance and lifestyle modification. Overall, the management pathway emphasizes accurate diagnosis, trigger avoidance, symptom-specific intervention, and shared decision-making to balance the typically benign prognosis with patient-reported impact.

Ethics Consideration

Formal ethics approval and written consent were not required for this retrospective case series of fully anonymised clinical cases in accordance with local institutional policy.

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