Risk Stratification of Patients Presenting with Transient Loss of Consciousness

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KEYWORDS

• Syncope • Risk stratification • Transient loss of consciousness • Syncope clinic

KEY POINTS

- Transient loss of consciousness (TLOC) has many possible causes, and syncope is among the most frequent.
- Important goals in initial evaluation of patients with TLOC include determining whether the episode was true syncope (vs other causes of TLOC, such as seizures or head trauma) and ascertaining the appropriate venue for subsequent care.
- At present, 30% to 50% of patients with syncope are admitted to hospital for evaluation, although many of these individuals could be safely and more cost-effectively managed outside of hospital.
- To reduce unnecessary admissions, several recent studies have focused on development of risk stratification tools to assist frontline clinicians in making a disposition decision.
- Although there is not yet consensus regarding an optimum decision process, the various available risk stratification recommendations provide direction in assessing short-term and long-term risks associated with syncope.
- Patients who have high short-term risk of adverse outcomes need prompt hospitalization for diagnosis and/or treatment, whereas others may be safely referred for outpatient evaluation (preferably at a clinic specializing in syncope evaluations).

INTRODUCTION

Syncope is a syndrome in which transient loss of consciousness (TLOC) occurs as a consequence of a self-limited, brief, and spontaneously self-terminating period of inadequate cerebral oxygen delivery.[1,2] The most common cause is a transient, but spontaneously reversible, decrease of systemic arterial pressure to a level less than the minimum needed to sustain cerebral blood flow (ie, less than the cerebrovascular autoregulatory range).[1–3] Other causes, such as acute hypoxemia (eg, abrupt aircraft decompression) or major, but transient,

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metabolic derangements affecting neuronal activation, are rare.

Whether the cause of transient systemic hypotension is innocent in nature (eg, the vasovagal [common] faint), or potentially life threatening (eg, torsades de pointes ventricular tachycardia), syncope may lead to physical injury, accidents, diminished quality of life, and economic loss. As a result, it is essential to recognize and evaluate patients with possible syncope, and differentiate true syncope from other nonsyncope causes of TLOC (eg, seizures, concussions, metabolic derangements), identify the specific causes(s) of the faints, and develop a treatment plan designed to prevent recurrences.[1,3]

CLASSIFICATION OF SYNCOPE

The classification of syncope is mainly based on the underlying mechanisms, which ultimately lead to global hypoperfusion. A table summarizing the diagnostic classification of the causes of syncope based on the European Society of Cardiology (ESC) syncope practice guidelines is given elsewhere in this issue.[1]

Reflex Syncope (Also Termed Neurally Mediated Reflex Syncope)

Reflex syncope includes several conditions in which neural reflex activity initiates a period of inappropriate vasodilation and relative or marked bradycardia. The most important, and the most common, among the reflex syncopes is vasovagal syncope (VVS), also known as the common faint. The second most frequently encountered form (although principally in older individuals and primarily in men) is carotid sinus syncope (CSS).[3–6] CSS must be distinguished from carotid sinus hypersensitivity, a finding that may be elicited by carotid sinus massage in many older individuals. CSS is only diagnosed if carotid sinus massage reproduces symptoms or, in the absence of other diagnoses, a sinus pause longer than 6 seconds is triggered by carotid sinus massage (previously 3 seconds was considered diagnostic, but that value is probably too nonspecific a criterion).

The other forms of reflex syncope tend to be classed as situational faints because they accompany certain specific activities (eg, syncope triggered by cough, micturition, or defecation).[1,3] In each of these cases, the reflex is triggered by the specific stimulus in a given patient. As in the case of VVS and CSS, the initial trigger event in situational faints (eg, micturition syncope, cough syncope) initiates either a slow heart rate (or at least slow for the degree of accompanying hypotension), or depressed vascular tone, or both. The result is systemic hypotension causing transient cerebral hypoperfusion and loss of consciousness. Lesser degrees of hypotension may cause disturbance of cerebral function without complete syncope. The resulting near-syncope symptoms may include nonspecific lightheadedness, visual spots or blinking lights or gray-out, and/or loss of hearing or abnormal hearing (eg, tinnitus), and often a vague sense of lightheadedness.

Orthostatic Syncope

Orthostatic hypotension leading to syncope occurs as result of the body's inability to maintain blood pressure and consequently adequate cerebral perfusion; this in turn results in TLOC.[1,7,8] The causes may be related to inadequate circulatory volume, as occurs with dehydration; excessive diuresis leading to volume depletion; or drug-induced vasodilation. Orthostatic hypotension may alternatively be a reflection of inadequate or delayed autonomic vasoconstrictor response.

Orthostatic syncope, as the name suggests, is usually initiated by a movement from supine or seated to an upright posture (although symptoms may be delayed for several minutes). Such a change in posture results in shift of 500 to 1000 mL of blood away from the chest to the venous capacitance system below the diaphragm. Absent an effective neurovascular constriction response, the sequestration of blood below the diaphragm reduces venous return to the heart with consequent reduction of cardiac filling pressure and stroke volume; if sufficiently severe, the resulting hypotension may lead to symptomatic cerebral hypoperfusion.

The human body has physiologic defenses against postural hypotension; these include baroreflex-initiated increase in heart rate, arterial and venous vasoconstriction (particularly in the splanchnic bed and lower extremities), and (albeit delayed) neuroendocrine adjustments operating through the renin-angiotensin-aldosterone system.[7] However, these defenses could be undermined if there is superimposed excessive volume depletion, or loss of cardiac chronotropic response, or impaired reflex vasoconstriction caused by autonomic dysfunction or medications such as β-blockers. Loss of skeletal muscle tone (a problem more often encountered in elderly individuals) may also contribute by reducing the effectiveness of muscle pump activity.

Physical counterpressure maneuvers such as leg crossing with tensing of thigh and buttock muscles may be helpful acutely for delaying symptoms by enhancing muscle pump activity and increasing venous return.[1] Longer-term physical rehabilitation should focus on restoring effective neurovascular reactivity to postural change.