Syncope in the Emergency Department: A Guide for Clinicians

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Contribution to Emergency Nursing Practice

- Syncope remains unexplained after ED evaluation, possible cardiac etiologies are crucial to identify because of an increased risk of serious adverse events. Existing guidance on risk stratification is limited and practitioners' risk aversion can lead to unnecessary low-risk admissions.
- A systematic approach to syncope that integrates a patient's history; examination and electrocardiogram; additional testing; risk stratification; and team-based, patient-centered care may help ED practitioners to rapidly and accurately identify patients classified as high risk.
- ED practitioners should be cognizant of the high-risk features of syncope, which increase the likelihood of cardiac etiology. Supplement clinical judgment with risk scores when no serious cause is evident. Engage patients in shared decision-making to arrange appropriate outpatient and follow-up care, observation, or admission.

Abstract

Syncope is a common presenting symptom to emergency departments, but its evaluation and initial management can be challenging for ED practitioners and particularly urgent in the presence of high-risk features that increase the likelihood of cardiac etiology. Even after thorough clinical evaluation, syncope may remain unexplained. In such instances, practitioners' clinical judgment and risk assessments are critical to guide further management. In this article, evidence-informed strategies are outlined to approach the diagnosis of syncope and provide an overview of syncope clinical decision rules and shared decision-making. By incorporating risk stratification and shared decision-making into syncope care, practitioners can more confidently engage patients and families in disposition decisions to organize appropriate outpatient and follow-up care, observation, or admission.

Key words: Syncope; Emergency department; Risk stratification; Shared decision-making

Introduction

Transient loss of consciousness (TLOC) is a frequent presentation to emergency departments, accounting for 0.6% to 1.0% of ED visits in North America,[1,2] and most commonly manifests in the form of syncope. All classifications of syncope result from cerebral hypoperfusion,[3] but the precise underlying cause can be challenging for ED practitioners to determine. The 3 general classifications of syncope include reflex syncope and syncope due to orthostatic hypotension (OH), which together make up approximately one-third of the ED diagnoses, and cardiac syncope, which makes up approximately 10% of the ED diagnoses.[3,4] Cardiac etiology is particularly imperative to

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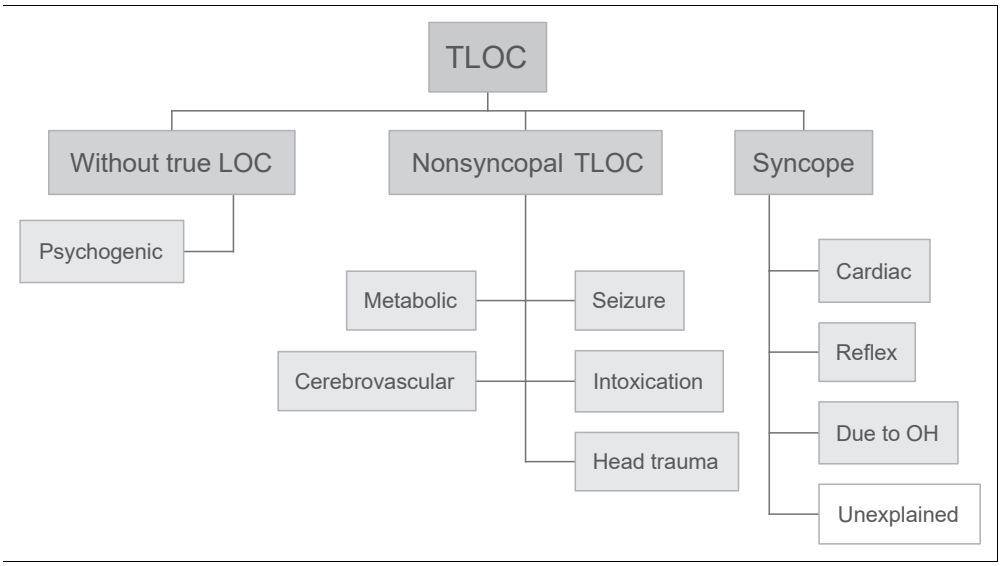

FIGURE 1

Differential diagnosis of TLOC. OH, orthostatic hypotension; TLOC, transient loss of consciousness. (Adapted from Williford and Olshansky.[17])

identify because of an increased risk of death and serious adverse events (SAEs) (eg, life-threatening arrhythmia or bleeding, sudden cardiac death (SCD), acute myocardial infarction, and stroke) and an increased need for procedural intervention.[4-7] Furthermore, even after thorough clinical evaluation, the underlying cause of syncope can remain unexplained in nearly one-third of the cases.[4] In these instances, licensed independent practitioners (including nurse practitioners, physicians, and physician assistants) must integrate clinical judgment and risk assessments to guide further management.

HERERetrospective studies estimate that hospitalization rates for syncope range from 25% to 35% in the United States.[1,8] For patients at low risk of SAEs and in the absence of serious medical conditions, hospitalization may be unnecessary because of its limited diagnostic value and potentially harmful outcomes.[3,6,9,10] Amid risk-averse contexts, varying risk perceptions, and occasional diagnostic uncertainty, ED practitioners are challenged with not only identifying patients at high risk for SAEs but also avoiding unnecessary hospitalizations.[11,12] Accordingly, researchers have called for more standardized and risk stratification–based approaches to syncope evaluation to improve practitioners' diagnostic confidence, decrease unnecessary admissions, and reduce costs associated with testing and hospitalization.[7,12]

Clinical decision rules (CDRs), which supplement risk assessments, and shared decision-making (SDM), which engages patients and families in the disposition decision, are 2 areas of recent innovation that have the potential to improve syncope evaluation and care experiences.[13,14] At a time of crowded emergency departments and disparities in access to primary care, ED advanced practice registered nurses are essential to increase underserved populations' access to, and experiences of, care.[15] The purpose of this article is to empower ED practitioners, and nurse practitioners in particular given their expertise in patient education and health promotion,[16] to incorporate CDRs and SDM into their practice. This article also outlines evidence-informed strategies to approach the diagnosis of syncope and discusses special considerations for older adults, syncope mimics, and rare presentations to augment practitioners' knowledge and clinical judgment.

Pathophysiology

TLOC is a state of real or apparent loss of consciousness characterized by amnesia, motor control abnormalities, and unresponsiveness, with numerous causes (Figure 1).[3,17] Syncope is a form of TLOC characterized by rapid onset and spontaneous recovery and specifically results from cerebral hypoperfusion.[3,17] Syncope must be differentiated from nonsyncopal TLOC (eg, seizure and head trauma) as well as mimics (eg, psychogenic pseudosyncope).[3,6] Presyncope refers to the symptoms preceding syncope (eg, nausea, vomiting, or sweating in reflex syncope, lightheadedness in OH, or palpitations in cardiac syncope).[3] European Society of Cardiology (ESC)

TABLE 1
Low- and high-risk features at index ED evaluation[3,6,31]

Assess	Low risk	High risk
Context of TLOC	• Features suggestive of reflex syncope • Prodrome (eg, lightheadedness, warmth, sweating, nausea, or vomiting) • Specific triggers (eg, fear, pain, or unpleasant smell) • Situational triggers (eg, micturition, deglutition, defecation, cough, or sneeze) • Being in crowded or hot spaces • Prolonged standing • Standing from supine or sitting position	• New-onset chest pain, dyspnea, abdominal pain, or headache • Syncope on exertion or while supine • Sudden-onset palpitations preceding syncope
Medical history	• Absence of heart disease • Long history of recurrent low-risk syncope similar to current syncope	• Severe structural heart disease or coronary artery disease (eg, heart failure, low LVEF, or previous myocardial infarction)
Family history	• No family history of SCD	• Family history of SCD
Physical examination	• Normal physical examination	• Unexplained SBP <90 mmHg • Evidence of bleeding (eg, gastrointestinal bleeding) • Persistent abnormal vital signs (eg, bradycardia in awake nonathletes) • Undiagnosed systolic murmur
ECG	• Normal ECG	• Abnormal ECG

ECG, electrocardiogram; LVEF, left ventricular ejection fraction; SBP, systolic blood pressure; SCD, sudden cardiac death; TLOC, transient loss of consciousness.

guidelines recommend that presyncope be evaluated and managed similarly to syncope because the 30-day risk of SAEs is comparable.[3,18]

The underlying mechanism of all 3 classifications of syncope is that it often starts with low cardiac output and decreased peripheral resistance, resulting in hypotension and cerebral hypoperfusion.[3] Reflex (neurally mediated) syncope has vasovagal or situational (eg, micturition) causes, whereas syncope due to OH can be caused by drugs (eg, vasodilators and diuretics), volume depletion (eg, hemorrhage), and primary or secondary autonomic failure.[3] Treatment for these classifications of syncope usually involves first-line education and lifestyle measures (eg, reassurance and awareness of triggers, situations, and prodromes) but may also extend to pharmacotherapy, drug discontinuation, and other therapies.[3] In cardiac syncope, arrhythmias, structural disease, and other less common causes (eg, acute coronary syndromes, pulmonary embolism, aortic dissection, and cardiac tamponade) are implicated in low cardiac output.[3] Cardiac syncope requires prompt treatment (eg, catheter ablation, device implantation, or surgical intervention) to address the underlying cause.[3]

Diagnostic Approach

Both ESC and American College of Cardiology/American Heart Association guidelines provide similar recommendations for the initial evaluation of syncope.[3,6] Key elements of the history, physical examination, and electrocardiogram (ECG) assist a practitioner in the diagnosis, risk assessment, and plan of care.

HISTORY

The history-taking in syncope has been referred to as history-building to emphasize its mutuality and diagnostic value.[19] The history should include the context of the TLOC, medical history, and family history to

TABLE 2
Features associated with classifications of syncope[3,6,22]

Cardiac syncope	LR+, 95% CI*	Reflex syncope	Syncope due to OH
AF	7.3 (2.4-22)	• History of recurrent syncope	• Prolonged standing
Severe structural HD	3.3-4.8	• Specific triggers (eg, fear, pain, or unpleasant smell)	• Postprandial hypotension
History of HF	2.7-3.4	• Situational triggers (eg, micturition, deglutition, defecation, coughing, sneezing, or laughing)	• Recent change in vasodepressive medications
Age >35 y	3.3 (2.6-4.1)	• Being in crowded or hot spaces	• Volume depletion (eg, hemorrhage, diarrhea, or vomiting)
On exertion	14-15	• Pallor, sweating, or nausea/vomiting	• Primary or secondary autonomic failure (eg, Parkinson disease, autonomic neuropathy)
Supine position	1.1-4.9		
Dyspnea	3.5 (1.5-9.1)		
Chest pain	3.4-3.8		
Palpitations	1.9 (0.86-4.5)		
Cyanosis	3.2 (1.6-24)		
Absence of prodrome	1.6 (1.0-2.6)		
HD and/or abnormal ECG	2.3 (1.7-3.0)		

AF, atrial fibrillation; ECG, electrocardiogram; HD, heart disease; HF, heart failure; LR+, positive likelihood ratio; OH, orthostatic hypotension.
* 95% CI for LR+ as reported in a systematic review on the detection of cardiac syncope by Albassam et al[22]

enable rapid triage on the basis of the presence of low- and high-risk features (Table 1). Syncope must be differentiated from nonsyncopal TLOC (Figure 1). For instance, features suggestive of seizure include the absence of a trigger; tongue-biting, head-turning, and unusual posturing; duration in minutes; and memory deficit.[3]

If syncope is suspected, the history may help differentiate cardiac syncope from reflex syncope or syncope secondary to OH (Table 2). Practitioners should note the association between the presence of high-risk features and greater likelihood of cardiac syncope.

CLINICAL EXAMINATION

Cardiac and pulmonary examinations should be performed for all patients, with close attention paid to the features that suggest the presence of structural heart disease (eg, murmurs, gallops, or rubs). A basic neurologic examination should also be performed. Because syncope generally presents without focal neurologic deficits, any identification of focal deficits requires further evaluation for cerebrovascular disease (eg, vertebrobasilar or carotid transient ischemic attacks or subclavian steal syndrome).[3,6] Practitioners should be aware that although rare, focal deficits and syncope may coexist; in this instance, treatment after a stroke misdiagnosis would aggravate hypotension.[20]

ELECTROCARDIOGRAM

A resting 12-lead ECG should be obtained for all patients presenting with syncope because of wide availability and utility in pinpointing arrhythmic syncope.[3,6] Practitioners should keep in mind that an arrhythmia may be intermittent or not recognized on an initial ECG and that a normal initial ECG cannot rule out 30-day serious cardiac arrhythmia.[21] High-risk ECG features that suggest a serious condition include abnormalities in rhythm and conduction, ventricular hypertrophy, changes consistent with ischemia, and several syndromes (eg, Wolff-Parkinson-White syndrome, Brugada syndrome, and long QT syndrome).[3]

FURTHER INVESTIGATIONS

If syncope remains unexplained after evaluation, further testing (eg, cardiac imaging and monitoring) may help clarify a diagnosis and prognosis when clinically indicated.[3,6] Routine laboratory testing in syncope is not well supported by evidence; however, recent studies have explored the utility of cardiac biomarkers (eg, B-type natriuretic peptide [BNP], N-terminal pro-BNP [NT-pro-BNP], and high-sensitivity cardiac troponins [high-sensitivity cardiac troponin T {hs-cTnT} and high-sensitivity cardiac troponin I]) in the detection of cardiac syncope and risk stratification.[22-24] Two recently developed CDRs, the Canadian Syncope Risk Score (CSRS) and the FAINT (heart failure,

arrhythmia, Initial ECG result abnormal, Elevated NT-proBNP, Elevated hs-troponin T) Score, include cardiac biomarkers as predictors.[25,26]

Special Considerations

COMPREHENSIVE GERIATRIC ASSESSMENT

Complex interactions exist between syncope and aging, multimorbidity, polypharmacy, frailty, and functional decline.[27] ESC guidelines recommend multifactorial evaluation and intervention for older adults with syncope, including potential discontinuation of hypotensive and psychotropic drugs, cognitive and physical assessments, and following the approach for unexplained syncope in the presentation of unexplained falls.[3]

SYNCOPE MIMICS AND CHAMELEONS

Syncope mimics are disorders that can seem similar to syncope, including seizures, metabolic disorders, stroke and transient ischemic attack, and psychogenic pseudosyncope.[28] Syncope chameleons are instances in which true syncope presents atypically, seeming to be similar to other disorders.[28] Chameleons include convulsive syncope, which resembles seizure activity, and syncope that resembles subclavian steal syndrome or subarachnoid hemorrhage. A thorough history and clinical examination are key to identifying life-threatening conditions and differentiating true syncope.

Rare Causes of Syncope

Although uncommon, multiple system atrophy (MSA) and inherited arrhythmia syndromes (IAS) can both cause syncope. These 2 particular causes are discussed here because they illustrate the multifactorial etiology of syncope and encourage practitioners to think critically.

MULTIPLE SYSTEM ATROPHY

MSA is a progressive neurodegenerative disorder thought to result from misfolded α-synuclein and includes both Parkinsonian (MSA-p) and cerebellar (MSA-c) variants.[29] MSA is characterized by autonomic failure and typically presents with early urogenital dysfunction followed by OH.[29] Autonomic studies and neuroimaging are central to evaluation, and management is directed toward addressing symptoms.

INHERITED ARRHYTHMIA SYNDROMES

IAS are genetic disorders that cause mutations in cardiac ion channel genes and may result in life-threatening arrhythmias and SCD.[30] IAS include long QT syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia. Features suggestive of arrhythmic syncope or a family history of SCD, particularly in younger patients, should prompt evaluation for IAS as well as cardiac imaging and testing.[30] Management includes pharmacotherapy (eg, β-blockers), implantable cardioverter-defibrillators, and avoidance of triggers (eg, exercise and stress).[30]

Risk Stratification

Risk stratification involves identifying a patient's risk of SAEs to guide further management.[3,6] By identifying patients at low risk of SAEs, many of whom can safely be discharged and receive outpatient follow-up,[3] health care service use is optimized and patients' quality of life is improved by avoiding unnecessary and prolonged hospitalization.

The prospective cohort Intermediate-Risk Syncope study found a low rate of 30-day SAEs in patients classified as being at intermediate risk of SAEs compared with those classified as high risk (0.8% vs 27.8%; $P < .01$).[31] Patients classified as being at intermediate risk did not meet all low-risk criteria nor present with any single high-risk feature (eg, family history of SCD, syncope on exertion or while supine, palpitations or chest pain, or marked ECG abnormalities). Notably, patients classified as being at intermediate risk possessed features such as stable cardiovascular disease and potentially related but stable comorbidities (eg, history of stroke or gastrointestinal bleeding, anemia, or Parkinson disease). In risk-averse contexts, these patients might be unnecessarily hospitalized despite being clinically stable. The Intermediate-Risk Syncope findings substantiate that generally, if patient education is provided and appropriate outpatient follow-up is arranged, patients classified as being at intermediate risk can safely be discharged after ED observation.

CLINICAL DECISION RULES

Numerous CDRs have been developed to predict short-term SAEs in patients presenting with syncope. ESC and American College of Cardiology/American Heart Association guidelines underscore that good clinical judgment continues to offer better prognostic yield than CDRs, and thus CDRs should merely supplement practitioners' clinical

TABLE 3
Canadian Syncope Risk Score[26]

Category	Points
Clinical evaluation	
Predisposition to vasovagal symptoms*	−1
History of heart disease†	+1
Any systolic pressure reading <90 or >180 mm Hg‡	+2
Investigations	
Elevated troponin level (> 99th percentile of normal population)	+2
Abnormal QRS axis (< −30° or >100°)	+1
QRS duration >130 ms	+1
Corrected QT interval >480 ms	+2
Diagnosis in emergency department	
Vasovagal syncope	−2
Cardiac syncope	+2
Total score (−3 to 11)	

The Canadian Syncope Risk Score was developed by Thiruganasambandamoorthy et al[26]

* Triggered by being in a warm, crowded place; prolonged standing; fear; emotion; or pain.

† Includes coronary or valvular heart disease, cardiomyopathy, congestive heart failure, and nonsinus rhythm (electrocardiogram evidence during index visit or documented history of ventricular or atrial arrhythmias or device implantation).

‡ Includes blood pressure values from triage until disposition from the emergency department.

judgment.[3,6] Meta-analyses have found that syncope CDRs are limited by varying ECG interpretation and definitions of syncope and arrhythmia; lack of external validation; and, if validated, poor sensitivity and specificity.[32-34] CDRs integrated into information technology systems, such as in clinical decision-support systems, have the potential to assist nurses and all practitioners in triage decision-making and the identification of high-risk conditions.[35]

Practitioners should keep in mind that the outcomes predicted by syncope CDRs are fundamentally associated with underlying disorders, of which syncope itself is a symptom.[3] Moreover, CDRs should be only used when no evident serious causes are identified during initial clinical evaluation.[33,36]

San Francisco Syncope Rule

The San Francisco Syncope Rule (SFSR) predicts the short-term risk of SAEs in syncope that remains unexplained after initial ED evaluation.[37,38] There are 5 risk factors that make up the SFSR: history of congestive heart failure, hematocrit <30%, abnormal ECG, shortness of breath, and systolic blood pressure <90 mmHg. A patient is considered to be at high risk of short-term SAEs if they have any 1 of the 5 risk factors. The SFSR derivation study found a sensitivity of 96% (95% CI, 92%–100%) and specificity of 62% (95% CI, 58%–66%).[38] Meta-analyses of external validation studies, however, have found lower sensitivity (87%; 95% CI, 79%–93%) and specificity (52%; 95% CI, 43%–62%) for the SFSR.[33,34] Considerable heterogeneity in sample and outcome definition may limit evidence for its generalizability.

Canadian Syncope Risk Score

The CSRS estimates the risk of 30-day SAEs not identified during initial ED syncope evaluation.[26] Nine top predictors (Table 3) were identified from an initial list of 43 candidate predictors through statistical analysis and predictive modeling of standardized presentation variables and outcomes during a prospective cohort study across 6 Canadian emergency departments (n = 4030). Importantly, the model was corrected for overfitting and internally validated through bootstrapping. The CSRS separates an abnormal ECG into individual predictor variables and further includes practitioners' diagnostic impression as a category, underscoring the value of clinical judgment. A score greater than or 4 confers a high or very high risk (>12%) of SAEs within 30 days.

The CSRS was externally validated in a prospective cohort study across 9 Canadian emergency departments (n = 3819).[36] The model demonstrated excellent calibration, with no statistically significant difference between predicted and observed risks, as well as excellent discrimination, with an area under the receiver operating characteristic curve of 0.91 (95% CI, 0.88–0.93). In this validation cohort, less than 1% of the patients classified as very low risk and low risk, 20% of those classified as high risk, and 50% of those classified as very high risk experienced 30-day SAEs. At a threshold score of −1 (low risk), CSRS sensitivity was 97.8% (95% CI, 93.8%–99.6%) and specificity was 44.3% (95% CI, 42.7%–45.9%).

Canadian Syncope Arrhythmia Risk Score

The Canadian Syncope Arrhythmia Risk Score (CSARS) is a CDR developed to predict the 30-day risk of arrhythmia unidentified during initial ED evaluation and death.[39] The 8 clinical predictors that make up the CSARS were derived from an additional prospective cohort study at 6 Canadian emergency departments (n = 5010) and are similar to CSRS predictors, although point values differ. Scores for the CSARS range from −2 to 8, with scores greater than or 4

conferring high or very high risk of arrhythmia or death within 30 days. Although the CSARS was internally validated through bootstrapping, it must be externally validated before it can be implemented in clinical settings. Once validated, it may help practitioners identify patients at low risk of arrhythmia who do not require admission, as well as guide follow-up care (eg, outpatient cardiac monitoring).

FAINT Score

The FAINT score is a CDR developed to rule out 30-day SAEs among older adults presenting to emergency departments with syncope.[25] Derived during a prospective cohort study at 11 emergency departments in the US (n = 3177), the FAINT score comprises 5 clinical predictors: history of heart failure, history of cardiac arrhythmia, initial abnormal ECG result, elevated NT-pro-BNP, and elevated hs-cTnT. Practitioners should keep in mind that the NT-pro-BNP and hs-cTnT assays may not be readily available in all emergency departments, although the researchers anticipate wider availability in the coming years. Although the FAINT score was internally validated through cross-validation, it must be externally validated before it can be implemented in clinical settings.

Shared Decision-Making

SDM is a means to alter power differentials in health care and requires practitioners to continually reflect on their language, communication, and ways of knowing during clinical encounters. In ED settings, SDM involves actively engaging patients and families, to the extent they desire and as clinically appropriate, in mutual information-sharing and consensus when a risk-benefit balance and several reasonable care options exist.[40,41] SDM aims to ensure that patients are well informed about their condition as well as the benefits, risks, and consequences of care options. Barriers to SDM implementation in emergency departments include the high-stakes, time-sensitive clinical situations of ED practice as well as the perceptions that patients would rather that practitioners make all the decisions.[13,41] SDM improves patients' knowledge and care experiences, provided that the proposed care options are well supported by evidence and that a risk-benefit balance exists.[13]

In syncope, SDM benefits patients at low to intermediate risk of SAEs or whose syncope remains unexplained after ED evaluation because multiple care options (eg, discharge with primary care or specialist follow-up vs observation vs admission) are made clear.[14,42] Outpatient management may even be indicated for select patients with suspected cardiac syncope in the absence of serious conditions.[6] For example, outpatient cardiac testing is an underused alternative to inpatient cardiac monitoring despite established safety and convenience.[6]

The disposition decision involves collaboration between a patient and practitioner that weighs the patient's condition, values and preferences, and life context and determinants of health.[14] Practitioners should specifically inquire into a patient's risk perceptions and tolerance, living circumstances (eg, support from informal or formal caregivers), and access to outpatient follow-up care if discharge is appropriate.[14,21] If observation or admission is indicated, a practitioner should inquire into a patient's socioeconomic status and implications of potentially missing work or other responsibilities.

SHARED DECISION-SUPPORT TOOLS

Shared decision-support tools (SDSTs) are aids (eg, paper- or computer-based tools and videos) that facilitate SDM between practitioners and patients and families.[13] Practitioners should tailor SDSTs to patients and families, which involves consideration of person-first language and patients' life circumstances, access to care, risk perceptions and tolerance, and literacy and numeracy (Figure 2). To ensure this, SDSTs may be supplemented to individualize care. For instance, Winokur et al[43] developed pictographs to improve patients' and families' comprehension of discharge instructions (eg, fever in children and gastroenteritis).

An SDST has recently been developed and tested to facilitate SDM in syncope. SynDA (Patient Decision Aid for Syncope) is a paper-based patient decision aid intended to meaningfully engage patients with unexplained syncope judged to be at low to intermediate risk of SAEs—but without any identified serious conditions—in disposition decisions (Figure 2).[42] In a randomized controlled pilot trial at 1 emergency department, SynDA demonstrated feasibility and showed promise in improving patients' active involvement in care and optimizing health care use.[44]

Implications for Emergency Clinical Practice

The initial management and risk assessment of syncope challenges many ED practitioners and often leads to unnecessary low-risk admissions, particularly in risk-averse contexts. At the same time, it is imperative that practitioners accurately identify the small but important subset of patients, primarily those with suspected cardiac syncope,

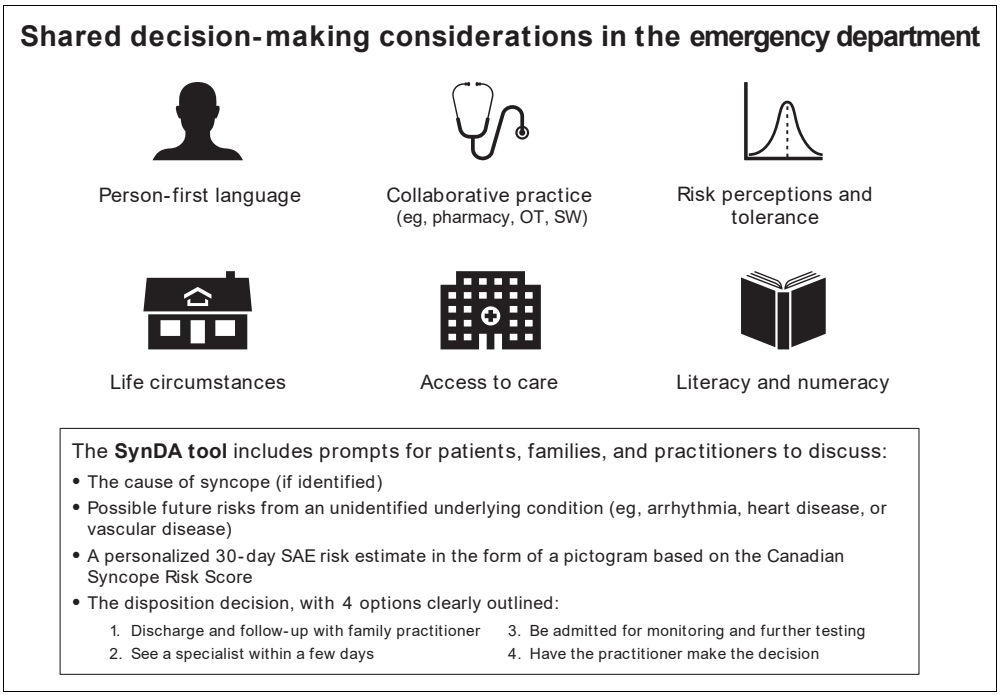

FIGURE 2

Shared decision-making considerations[14,21] and key aspects of the SynDA tool.[42] OT, occupational therapy; SAE, serious adverse event; SW, social work. (The SynDA tool was developed by Probst et al[42])

at high risk of SAEs. Moreover, syncope can often remain unexplained even after thorough clinical evaluation. In this article, we have presented 2 innovative, complementary, and evidence-informed strategies—risk stratification and SDM—with which practitioners can supplement their knowledge and clinical judgment to navigate complex clinical presentations of syncope. Practitioners can use the CSRS, a rigorously developed and validated CDR, to predict the risk of 30-day SAEs. To facilitate the disposition decision, the SynDA tool shows promise to engage patients at low to intermediate risk of SAEs in SDM.

Conclusions

TLOC and its manifestation of syncope are complex ED presentations. In this article, we briefly summarized the pathophysiology of syncope. Although reflex syncope and syncope due to OH generally entail a benign course, cardiac syncope confers an increased risk of SAEs. We outlined a diagnostic approach to discern the differential diagnosis of syncope and underscored the importance of a thorough history and clinical examination. When syncope remains unexplained and no serious causes are evident, practitioners' clinical judgment may be supplemented with CDRs to inform risk assessments. Finally, we highlighted the value of SDM in improving patients' active involvement in care decisions. Patient education, risk stratification, SDM, and appropriate follow-up care are pivotal to reduce unnecessary hospitalization as well as to improve outcomes and quality of life for patients with syncope. Incorporating these principles into practice will strengthen practitioners' knowledge and clinical judgment, and further empower them to provide safe, evidence-informed, and comprehensive care.

Author Disclosures

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