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Trauma and syncope: looking beyond the injury

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SUMMARY

Background 42% of the population experience syncope by the age of 70, accounting for up to 6% of hospital admissions that frequently present as falls. The etiologies of some falls are benign, and others, such as cardiac syncope, are associated with a greater mortality and must be identified.

Methods This review article aims to bridge the literature gap by providing a comprehensive practice review and critical summary of the current syncope guidance relating to the trauma patient.

Results The National Institute for Health and Care Excellence, the American College of Cardiology, and European Society of Cardiology published syncope risk stratification guidance. The inclusion of certain high-risk features represented in all three guidelines suggests their significance to identify cardiac syncope including heart failure, abnormal vital signs, syncope during exercise with little to no prodrome, family history of sudden cardiac death, and ECG abnormalities. Of 11 syncope risk stratification scoring systems based on these guidelines, only 2 are externally validated in the emergency department, neither of which are validated for major trauma use. Adherence to thorough history-taking, examination, orthostatic blood pressure recording, and an ECG can diagnose the cause of syncope in up to 50% of patients. ECG findings are 95% to 98% sensitive in the detection of serious adverse outcomes after cardiac syncope and should form part of a standardized syncope trauma assessment. Routine blood testing in trauma is often performed despite evidence that it is neither useful nor cost effective, where the screening of cardiac enzymes and D-dimer rarely influences management.

Discussion In the absence of a gold-standard clinical test to identify the cause of a syncopal episode, standardized syncope guidelines as described in this review could be incorporated into trauma protocols to analyze high-risk etiologies, improve diagnostic accuracy, reduce unnecessary investigations, and develop an effective and safer management strategy.

INTRODUCTION

Syncope is a common cause of trauma. There is insufficient published guidance and few validated tools for trauma teams to use in standardizing the investigation and management of syncope in the trauma setting. This review article seeks to provide a clear summary of the current syncope guidance relating to the trauma patient by exploring the harmful causes of syncopal falls that may be overlooked and discussing the optimal evaluation and safe management of these cases.

Falls are currently the leading mechanism of injury to major trauma patients, accounting for 39% of the major trauma in the UK with an estimated annual cost burden of £2.3 billion.[1 2] Despite 29% to 36% of hospital attendees with syncope suffering concomitant physical injury, syncope is a neglected cause of trauma.[3 4] Among elderly patients, 42% to 95% have no recollection of their loss of consciousness and can mistakenly attribute their syncope to an accidental or 'mechanical fall'.[5 6] These incorrectly labeled mechanical falls can be mistakenly characterized as benign.[7] This inappropriately obviates further investigation into potentially serious underlying health conditions. As such, there is growing evidence that all falls and near syncope should be investigated as for syncope.[8]

SYNCOPE AND THE TRAUMA TEAM

Syncope is common; 42% of the population experience syncope by the age of 70, accounting for up to 6% of hospital admissions.[9] A reduction in cerebral perfusion leads to a transient loss of consciousness (TLoC) and loss of muscular tone and, if upright, will precipitate a fall. Injuries associated with trauma are governed by impact energy and patient condition, where frailty factors such as age, sarcopenia, and osteoporosis influence the morbidity and mortality associated with trauma. Increased frailty in the elderly population can cause their falls from a standing height to incur significant trauma, including but not limited to fragility fractures, soft tissue injury, joint dislocation, and head injury. However, with falls increasingly dominating trauma presentations in an aging population, the burden of syncope care will continue to span across a breadth of specialities from general and emergency medicine practitioners to surgeons and medical physicians.

Syncope is under-reported, and the harm inflicted by failure of recognition and diagnosis can be significant. The Framingham Heart and Offspring studies followed up 7814 participants during an average of 17 years, identifying that those who suffered cardiac syncope had a 2.01 increased risk (HR, 95% CI 1.48 to 2.73) of death from any cause when compared with those who did not experience syncope.[10] This highlights a valuable opportunity for trauma teams to identify syncope as a cause of unexplained falls and to prevent significant future harm through the correct diagnosis and treatment of serious underlying disease.

DEFINITIONS

- Blackout or TLoC: a loss of consciousness characterized by rapid onset, short duration, and spontaneous recovery, irrespective of mechanism.
- Syncope: TLoC due to transient global cerebral hypoperfusion.

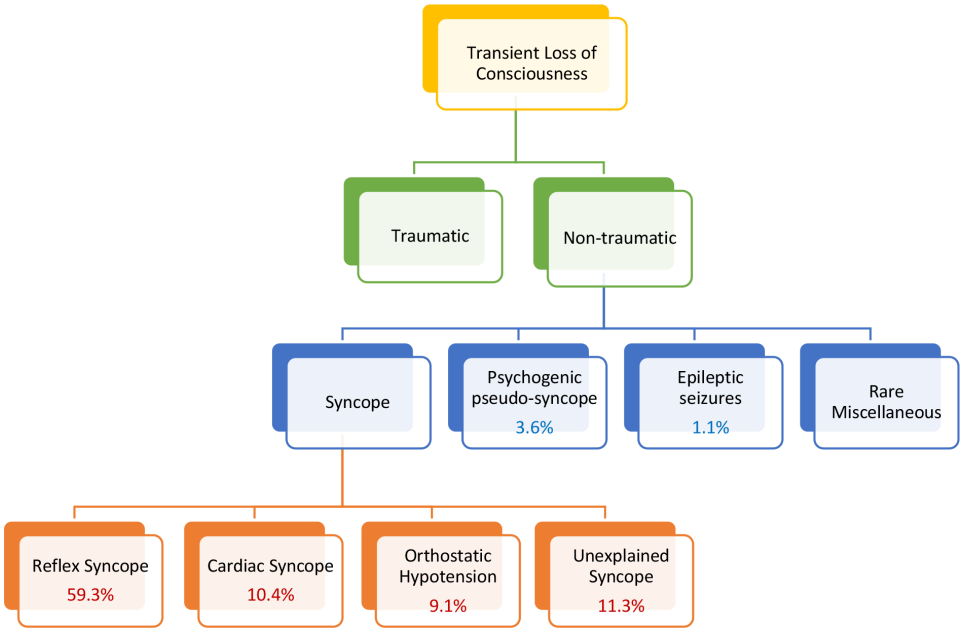

Figure 1 Classifications of TLoC and syncope. TLoC can be broadly categorized into a traumatic and non-traumatic loss of consciousness, with non-traumatic further subdivided into four groups: syncope, psychogenic pseudo-syncope, epileptic seizures, and rare miscellaneous causes. Syncope is categorized into four types: reflex, cardiac, orthostatic hypotension, and unexplained syncope. Their relative mean frequencies are stated.[11] TLoC, transient loss of consciousness.

CAUSES OF SYNCOPE

There are several different causes of TLoC as shown in figure 1. Some are benign and others, such as cardiac syncope, are associated with a greater mortality. It is therefore important to recognize the potential cause of the fall for risk stratification and management. The role of the trauma team is to stabilize and refer onward or safely discharge the patient with suspected syncope. After stabilization, the question trauma teams must answer is: was this fall due to a TLoC and what was the cause?

Syncope is probable where TLoC occurs with features present that are indicative of reflex syncope, cardiac syncope, or orthostatic hypotension (OH)-induced syncope (table 1), without any features suggestive of non-syncopal causes (table 2). A review of major research publications describing syncope in 5248 patients identified the mechanism as 59.3% reflex syncope, 10.4% cardiac syncope, 9.1% syncope due to OH, and 11.3% remained unexplained syncope.[11]

Reflex syncope occurs when a neural trigger causes vasodilation and/or bradycardia when activated inducing cerebral hypoperfusion. Vasovagal syncope, or fainting in common parlance, is the most common form of reflex syncope and is associated with diaphoresis, nausea, pallor, and warmth after upright posture or emotional triggers.[12] Reflex syncope also includes situational syncope, which is a faint triggered in a specific situation

Table 1 Causes of syncope

Pathophysiological origin	Causes
Reflex syncope	Vasovagal Situational Carotid sinus syndrome Non-classical forms
Cardiac syncope	**With obstructive structural heart disease:** Aortic stenosis Hypertrophic cardiomyopathy Cardiac masses Pericardial disease/tamponade Prosthetic valvular dysfunction Congenital coronary artery abnormalities **With arrhythmia:** Tachyarrhythmias Bradyarrhythmias **Cardiopulmonary and great vessels:** Pulmonary embolism Aortic dissection Pulmonary hypertension
Orthostatic hypotension	Medication induced Volume depletion Primary autonomic failure Secondary autonomic failure

The categorizations and causes of syncope, modified from the European Society of Cardiology.[32]

Table 2 Non-syncopal conditions presenting with collapse

Condition	Features that distinguish from syncope
Cardiac arrest	No spontaneous recovery from TLoC
Cataplexy	Loss of muscular tone and responsive, usually associated with narcolepsy
Coma	Longer duration of LoC without spontaneous recovery
Complex partial seizures, absence epilepsy	Unresponsiveness and amnesia without falls, with associated neurological features
Falls without TLoC	Absence of amnesia and unresponsiveness, clear cause for fall
Generalized seizures	Aura, flashing lights may trigger, longer duration of LoC, symmetrical rhythmic muscle movements, confusion after LoC lasting minutes (shorter with syncope)
Intoxication	Longer duration of LoC, consciousness often impaired rather than lost
Intracerebral or subarachnoid hemorrhage	Severe headache, neurological signs, and progressive LoC
Metabolic disorders (hypoglycaemia, hypoxia, hyperventilation with hypocapnia)	Longer duration of LoC, consciousness often impaired rather than lost
Psychogenic pseudosyncope	Increased frequency and longer duration of apparent syncope without true LoC, maintained hemodynamics, normal electroencephalogram
Subclavian steal syndrome	Upper extremity activity associated with focal neurological signs
Transient ischemic attack	Focal neurological features usually without TLoC. If LoC occurs, there is a longer duration of TLoC.

Conditions that may be incorrectly diagnosed as syncope are listed, modified from the American College of Cardiology and European Society of Cardiology and syncope guidelines.[12 32]
LoC, loss of consciousness; TLoC, transient loss of consciousness.

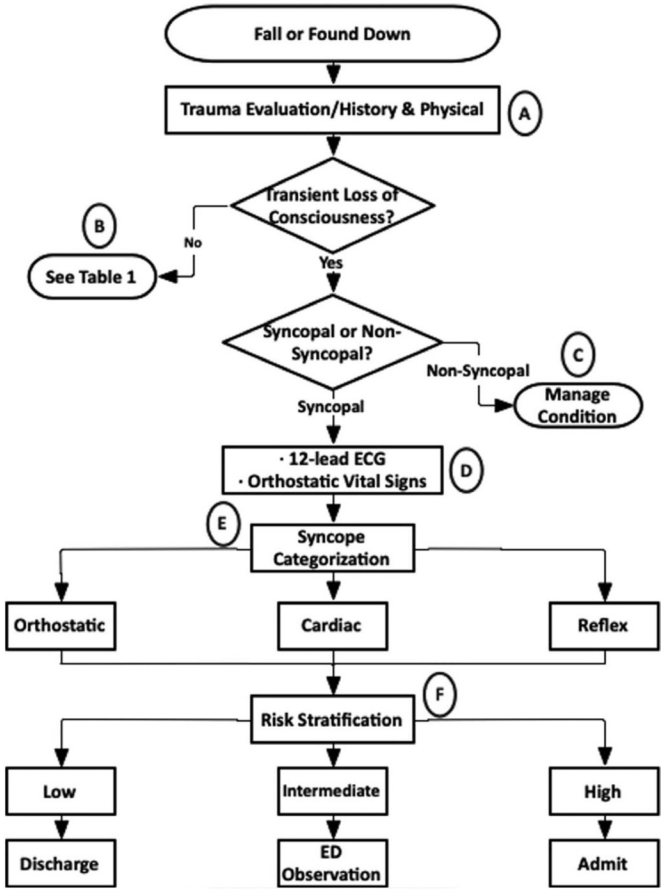

Figure 2 Algorithm for the management of falls and syncope evaluation. Credit to 'Syncope, "mechanical falls", and the trauma surgeon' by Biffl *et al*.[16] Consent for image use provided by Wolters Kluwer health (CC-BY-NC). The discussion points A–F and table 1 can be sourced from the original article.

including micturition, coughing, sneezing, defecation, laughing, swallowing, or stretching. Carotid sinus hypersensitivity can induce carotid sinus syndrome and is rare under the age of 40.[13] This is a condition where the blood pressure sensing system in the carotid arteries becomes oversensitive and induces a significant and inappropriate bradycardia and/or hypotension with carotid pressure. Major trauma is more frequently associated with carotid sinus syndrome (24.3%) than other syncope types (2.2-7.9%) presenting to the emergency department (ED).[14]

The cardiac causes of syncope must be identified as priority as they carry the greatest risk of sudden cardiac death (SCD), defined as death from a cardiac cause within 1 hour from the onset of symptoms.[15] Cardiac causes of syncope include arrhythmia, structural heart disease and conditions affecting the cardiopulmonary and great vessels. In general, these relate to the obstruction of cardiac output limiting blood supply to the brain. OH describes a drop in systolic blood pressure of ≥20 mm Hg or diastolic blood pressure of ≥10 mm Hg on assuming the upright position and can be associated with intravascular volume depletion, medication side effects, and primary and secondary autonomic failures.[12]

ASSESSMENT OF SYNCOPE

Biffl *et al* proposed a management algorithm for the assessment of traumatic falls and syncope (figure 2), adapted from the American and European cardiology societies' guidance.[16] The following sections will build on this framework though the analysis of the trauma evaluation in syncope with practical management considerations, the critical inclusion of UK blackout guidance in addition to European and American guidelines, and a discussion of the relevance of current syncope risk stratifications tools in trauma.

Adherence to thorough history-taking, examination, orthostatic blood pressure recording, and an ECG can diagnose the cause of syncope in up to 50% of patients.[9] However, in elderly patients, the task of distinguishing between syncopal and non-syncopal falls can be complicated by a patient's cognitive impairment or amnesia of the event; where the patient is unaware that they have fainted and often assumes that they have simply fallen.[17] As such, an accurate history from a collateral source that witnessed the event is of paramount importance and, where possible, should be included in the evaluation of trauma and syncope to help describe or validate the features.[17 18] This too can be difficult in the older population as up to 40% of syncopal episodes are unwitnessed.[19] Investigations into a form of reflex syncope noted that up to 95% of patients presenting with a fall had observed amnesia of the event.[5] A patient's age was an important indicator for the likelihood of syncopal amnesia in research by O'Dwyer *et al* where 42% of patients over 60 years old suffered amnesia after induced syncope compared with only 20% of patients under 60 years of age ($p=0.003$).[6]

Recurrent falls, defined as two or more falls in a year, are a familiar presentation to trauma teams. Syncope should be suspected in these patients, prompting a full falls review including a syncope workup.[20] An investigation of 5590 patients conducted by Furukawa *et al* identified that head injury was twice as likely in patients with arrhythmic syncope when compared with non-arrhythmic forms ($p<0.01$).[21] Furthermore, trauma patients presenting after a fall with a head injury with no injury to the hands or forearm demonstrate an absence of protective fall reflexes. This signals that a possible TLoC has occurred and that syncope investigations should be conducted.[22 23] Polypharmacy, defined as the use of five or more routine medications, raises suspicion of iatrogenic OH in patients presenting with falls.[24]

SHOULD INVESTIGATIONS BE PROTOCOL DRIVEN?

Prospective research demonstrated that abnormal ECG findings in combination with thorough history-taking was 95% sensitive in the detection of cardiac syncope or of serious adverse outcomes following cardiac syncope and, therefore, should form part of a standardized syncope assessment.[25 26] Routine blood testing in trauma is often performed despite evidence that it is neither useful nor cost effective.[27] Routine trauma panels typically include full blood count, urea and electrolytes, liver function tests, coagulation screen, blood group and crossmatch, ethanol level, and blood gas analysis including blood glucose and lactate. Though a controversial marker of acute blood loss, the recording of a hemoglobin level can identify severe anaemia, a reversible cause of syncope.[28] Severe electrolyte disturbances can be identified and corrected through electrolyte blood testing. However, a prospective, controlled, multicentre study observed that routine basic laboratory testing was rarely helpful in the diagnosis of syncope.[29]

The measurement of cardiac enzymes, including troponin and brain natriuretic peptide, can detect dangerous structural causes of cardiac syncope. However, blood testing directed by clinical need was shown to be preferable to routine collection of cardiac enzymes after syncopal events, where screening enzyme analysis only influenced management in 1% of patients.[30] Research

by Kelly *et al* identified that D-dimers were tested in 15.7% of 32 440 patients presenting to the ED with syncope.[31] The diagnostic yield was low; pulmonary embolism was detected in only 2.2% of patients after D-dimer testing.

Cardiac imaging is recommended once cardiac syncope is suspected or when there is previously known heart disease.[12 32] With increasing availability and widespread use, it is necessary to evaluate the role of echocardiography in screening for cardiac causes of syncope. Mendu *et al* investigated 2106 patients admitted to the hospital after syncope and identified that echocardiography revealed the cause of syncope in <5% of patients.[30] Regarding the role of preoperative echocardiography in patients without syncope and with syncope, a retrospective study of 264 823 patients demonstrated that echocardiography prior to non-cardiac operation was not associated with reduced hospital admission length or improved survival at 30 days and at 1 year.[33] When carotid sinus syndrome is suspected, a carotid sinus massage can be undertaken by a specialist. A diagnosis is confirmed if pressure to the common carotid artery bifurcation causes syncope associated with a ventricular pause lasting three seconds or more and/or a fall in systolic blood pressure over 50 mmHg. Contraindications include previous stroke, transient ischemic attack or myocardial infarction in the preceding 3 months, ventricular fibrillation, ventricular tachycardia, known carotid stenosis over 70%, or an adverse reaction to a previous carotid sinus massage. Trauma may necessitate a delay to assessing for carotid sinus syndrome due to the inability to stand patients during testing.

Ultimately, research findings corroborate that standardized routine syncope workups in trauma patients produce low yield results, recommending that diagnostic workup should be analyzed on an individual clinical basis.[30 34 35]

WHICH INTERNATIONAL SYNCOPE GUIDELINE TO FOLLOW?

Once syncope is suspected as the trigger for trauma, the priority of the trauma team is to risk stratify patients into those who require hospital admission for further investigation and those who can be safely treated in the community. The National Institute for Health and Care Excellence (NICE), the American College of Cardiology (ACC), and European Society of Cardiology (ESC) published guidance in 2014, 2017, and 2018, respectively, outlining the best practice for the risk stratification of TLoC. NICE and the ESC outline the high-risk features that are indicative of cardiac syncope requiring specialist assessment in under 24 hours, whereas the ACC guidelines identify short-term and long-term high-risk factors. The Venn diagram in figure 3 illustrates the overlap between these guidelines and highlights the features of common concern.

Consensus between guidance exists where syncope during exercise with little (<10 s) to no prodrome is strongly indicative of cardiac syncope. Palpitations preceding the syncopal episode

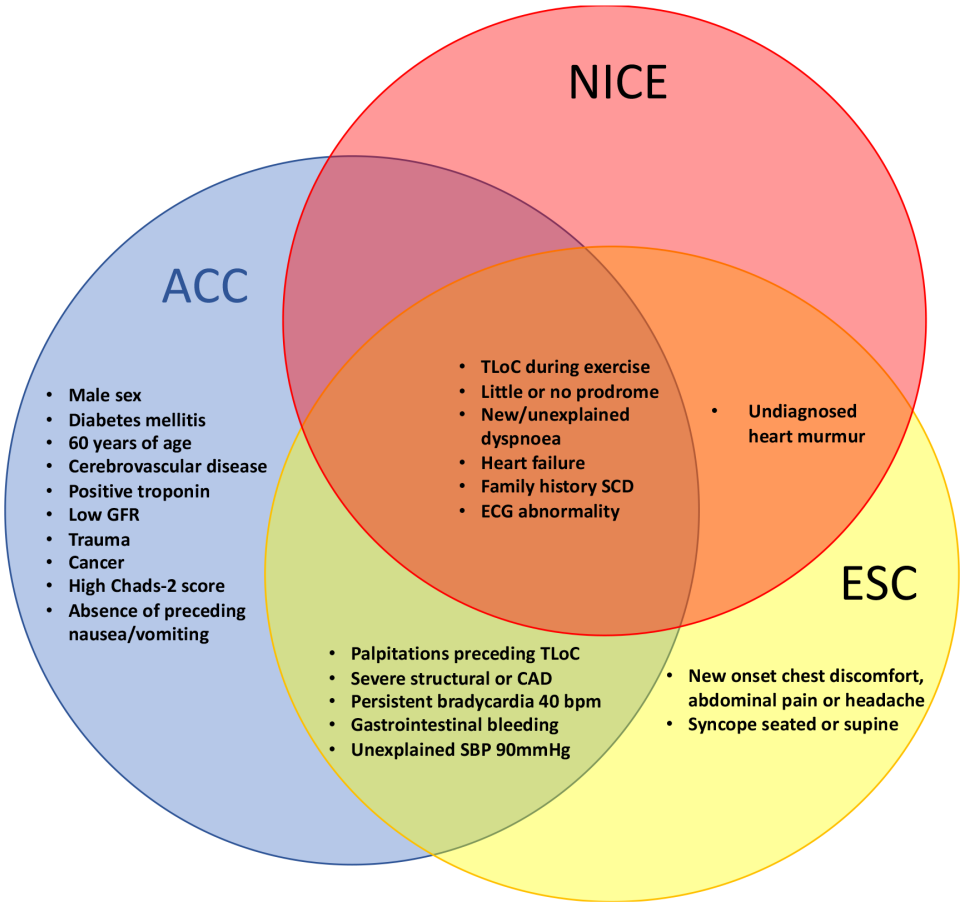

Figure 3 Venn diagram of the ESC, ACC, and NICE high-risk syncope features. A Venn diagram illustrating which high-risk features indicative of cardiac syncope are recognized by NICE, ACC, and ESC guidance. CHADS-2 estimates stroke risk in patients with atrial fibrillation; ACC, American College of Cardiology; BPM, beats per minute; CAD, coronary artery disease; ED, emergency department; ESC, European Society of Cardiology; GFR, glomerular filtration rate; NICE, National Institute for Health and Care Excellence; SBP, systolic blood pressure; SCD, sudden cardiac death; TLoC, transient loss of consciousness.

Table 3 Canadian Syncope Risk Score

Category	Risk factors	Points
Clinical evaluation	Predisposition to vasovagal symptoms	−1
	History of heart disease*	1
	Any systolic blood pressure reading <90 or >180 mm Hg	2
Investigations	Elevated troponin level (>99th percentile)	2
	Abnormal QRS axis (<−30° or >100°)	1
	QRS duration >130 ms	1
	Corrected QT interval >480 ms	2
Diagnosis in the ED	Vasovagal syncope	−2
	Cardiac syncope	2
Total score	Very low risk: −3 to −2	
	Low risk: −1 to 0	
	Medium risk: 1–3	
	High risk: 4–5	
	Very high risk: 6–11	

The risk factors included in the Canadian Syncope Risk Score and their associated points that categorize patients into groups from very low risk to very high risk of serious adverse outcomes after 30 days.
*History of heart disease includes coronary or valvular heart disease, cardiomyopathy, heart failure and non-sinus rhythm on ECG (ECG evidence during index visit or documented history of ventricular or atrial arrhythmias, or device implantation).
ED, emergency department.

is a high-risk feature incorporated by the ESC and ACC. The presence of heart failure and a family history of SCD are included as high-risk features in all guidance, with NICE recognizing the risk associated with a family history of SCD of relatives <40 years old. Further overlap of pre-existing cardiac disease indicative of high-risk cardiac syncope is represented by the inclusion of severe structural or coronary heart disease and undiagnosed heart murmur in two of the three guidelines.

Each set of guidelines references the higher risk associated with abnormal vital signs. All guidelines assign new or unexplained breathlessness as a high-risk feature, whereas persistent abnormal vital signs are also included as high-risk in ACC guidance. The ESC and ACC specifically identify unexplained systolic blood pressures of <90 mm Hg or persistent bradycardia <40 beats/minute while awake and in the absence of physical fitness training as high-risk of cardiac syncope. Of note, only the ESC guidance includes chest discomfort, palpitations, abdominal pain, and headache as high-risk features. Most notably, chest discomfort is a strong predictor of serious adverse outcomes due to association with structural cardiac disease.[25]

ECG abnormalities are considered high-risk, and further investigation is recommended by all three sets of guidelines for initial syncope evaluation and is frequently incorporated within standard trauma protocols. A comprehensive review of individual ECG abnormalities is beyond the scope of this review article, and specific high-risk ECG patterns can be sourced within each guideline. Ultimately, the inclusion of certain high-risk features in all three sets of guidance would suggest their significance to identify cardiac syncope.

RISK STRATIFICATION

When the cause of TLoC in the trauma patient remains unclear, risk stratification becomes necessary to guide accurate management. A 2022 literature review conducted by Sutton *et al* investigated the performance of syncope risk stratification tools available for use in the ED including the Martin-Kapoor score, the San Francesco Syncope Rule, the Osservatorio Epidemiologico sulla Sincope nel Lazio, the Risk Stratification of Syncope in the Emergency Department score, the Evaluation of Guidelines in Syncope Study score, the Boston Syncope Criteria, the Short-Term Prognosis of Syncope score, the FAINT score, the

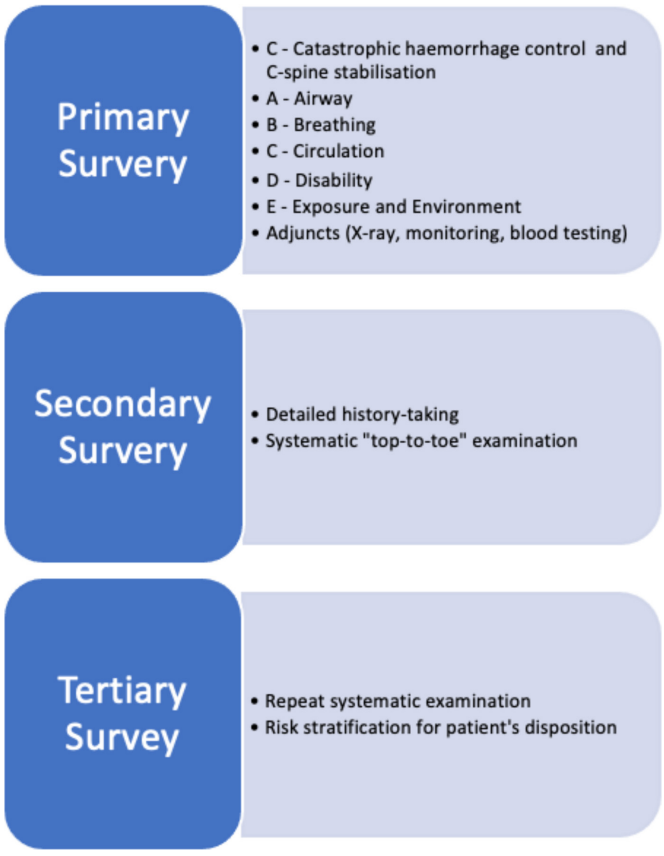

Figure 4 A systematic outline of ATLS principles. ATLS, advanced trauma life support.

Basel IX ECG ALERT-CS tool, the Canadian Syncope Risk Score (CSRS), and the Early Standardised Clinical Judgement for syncope score (ESCJ).[36]

All risk assessment tools employ a variety of factors including features from the history, examination, ECG, and serum biomarkers. Their findings suggest that the prognostic yield of most syncope risk stratification tools is no better than good clinical judgement. However, the CSRS and ESCJ have been externally validated to identify low-risk patients able to be discharged from the ED and high-risk patients requiring urgent investigation and potential admission to the hospital.

The ESCJ offers a standardized approach to syncope assessment including a syncope specific case report form which accumulated in the correct identification of cardiac syncope with 87% accuracy.[37] The CSRS approach (table 3) uses a rapid risk stratification score to guide the management of patients when the cause of syncope had not been identified during initial evaluation. This tool includes pertinent features of the history, examination and investigations that are routinely collected within a trauma evaluation. The CSRS was successfully validated in 2020, identifying that <1% of patients with very-low-risk and low-risk, 20% of high-risk and approximately 50% of very-high-risk patients experienced serious outcomes after 30 days.[38] These outcomes indicate that, in the absence of confounding disease requiring in-hospital investigation or treatment, very-low-risk and low-risk groups can be discharged with safety advice. The medium-risk group may benefit from admission or referral to a syncope unit, and the high-risk and very-high-risk groups require immediate admission and investigation for cardiac syncope. Notably, the CSRS excluded patients who suffered major trauma

as a result of syncope. This stratification tool requires validation regarding this patient cohort prior to inclusion in trauma protocols.

Between 2009 and 2018, the ESC guidance altered from an emphasis on risk stratification techniques in the management of syncope to recognizing that the diagnostic accuracy from risk stratification may only equal that of good clinical judgement. An investigation into syncope history-taking demonstrated that a variety of clinical judgment at initial assessment exists, where the diagnostic accuracy from assessment in the ED was 65% compared with 80% from independent syncope researchers with a standardized approach.[39] Importantly, no cardiac syncope was missed by either group. Furthermore, Sutton *et al* established a difference in diagnostic yield for syncope of 14% between the <60 years group (68%) and the >60 years group (54%).[11] As most trauma patients presenting with syncope are elderly and assessed in the ED, risk stratification can be a powerful tool for trauma teams.

In summary, after the initial syncope evaluation and management of the sequalae of trauma, a decision whether to admit the patient into the hospital, provide a short period of observation or discharge the patient to be managed in the community must be considered. As discussed, patients presenting with high-risk features should be admitted for specialty medical assessment in under 24 hours. Patients with low-risk features indicative of reflex syncope or OH can be discharged directly from the ED with ongoing community management. Patients with neither low-risk nor high-risk features require expert syncope opinion. Expert opinion obtained in an ED observational unit compared with admission to the hospital has shown decreased hospital admission times with no difference in serious adverse outcome rates.[40]

HOW CAN TRAUMA BE MANAGED DIFFERENTLY TO PREVENT RECURRENT SYNCOPE AND FUTURE TRAUMA?

Gold-standard trauma care evaluates patients in line with the advanced trauma life support (ATLS) programme. This section will examine the relationship between the evaluation of syncope (figure 2) and ATLS principles in the context of trauma (figure 4).

When undertaking the primary survey, patients with suspected traumatic cervical spine injury should be immobilized with spinal precautions in line with ATLS guidance. Horizontal immobilization aids venous return and reduces the risk of orthostatic syncopal events. Consideration on an individual basis should be given to patients with head injury and suspected intracranial hypertension as ideal head elevation of 30° reduces the risk of intracranial hypertension but may reduce cerebral perfusion pressures and cause syncope.[41 42] Furthermore, if patients are restricted from lying horizontally, strain on the blood pressure control system can continue and syncope may recur. Awareness and preventative measures aimed to resolve hemodynamic instability would help reduce the incidence of such events.[43] The recording of orthostatic blood pressures to identify OH within the initial syncope evaluation may not be possible due to immobilization, limb fracture, or severe soft tissue injury. In addition, OH in the acute setting may also be affected by the trauma presentation and later assessment will be important. A meta-analysis involving 121 913 patients provides strong evidence of an association between OH and all-cause mortality.[44] However, it is acknowledged that the comorbidities associated with OH are likely to cause this increase in mortality. Thus, if OH is suspected, orthostatic blood pressure recordings for diagnostic confirmation can be delayed until clinically appropriate.

All forms of syncope are exacerbated by circulatory volume depletion, particularly the orthostatic and reflex types that account for the majority of cases.[32] The application of hemostatic devices minimizing blood loss, reversal of anticoagulating medication, use of hemostatic agents, and adequate replacement of blood products will reduce syncopal events.[32 45] Minimizing the sight of blood or wounds where possible can further reduce the risk of vasovagal syncope. Compression stockings can be applied to aid venous return if not contraindicated. A single target resuscitative blood pressure in trauma remains controversial, particularly when complicated by premorbid syncope. Syncope occurs below systolic blood pressures of 50 mm Hg to 60 mm Hg at cardiac level. Target systolic blood pressures of over 100 are not always feasible in major trauma and lower systemic blood pressure may predispose patients to syncope.[32 46] A permissive or 'damage control' systolic blood pressure target of 80 mm Hg to 90 mm Hg in trauma without brain injury, and mean arterial pressure of over 80 mm Hg in trauma with brain injury was proposed in the European guidelines on management of major bleeding and coagulopathy after trauma.[47] However, it was concluded that permissive hypotension has not yet been proven effective in trauma resuscitation with consideration to patient age, the mechanism and severity of injury, or in the presence of shock.[46]

Trauma patients with suspected syncope without circulatory volume loss are routinely fasted until the need for intubation or operation can be excluded. To reduce further syncopal events in fasting patients, adequate hydration should be maintained with parental administration of 25 mL/kg/day of fluid with 1 mmol/kg/day of sodium, potassium, and chloride.[48] Caution should be exercised when hydrating patients with severe cervical spinal cord injury, as autonomic dysfunction risks overhydration resulting in pulmonary oedema and hyponatraemia.[49]

The tendency to vasovagal syncope due to severe pain may be exacerbated through the hypotensive side effects of frequently administered analgesic agents. A large systematic review and meta-analysis demonstrated a low risk of hypotensive adverse events in trauma patients who were administered ketamine (0%), morphine (0.5%), and fentanyl (1.6%).[50]

CONCLUSION

Syncope is an underdiagnosed cause of trauma and should be considered a potential cause of unexplained falls, regardless of whether TLoC was reported. Literature regarding the relationship between syncope and trauma is limited, with prospective research essential to investigate their interactions. Despite promising syncope risk stratification tools, they require further validation for major trauma patients prior to their inclusion in trauma protocols. In the absence of a gold-standard clinical test to identify the cause of a syncopal episode, standardized syncope guidelines as described in this review could be incorporated into trauma protocols to analyze high-risk etiologies, improve diagnostic accuracy, reduce unnecessary investigations, and develop an effective and safer management strategy.

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