Heart Sci J 2020; 1(4): 9-13

Contents list available at www.heartscience.ub.ac.id

Heart Science Journal

Journal Homepage : www.heartscience.ub.ac.id

Review Article

Clinical Management of Syncope in Emergency Department Based on Risk Stratification: A Literature Review

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ARTICLE INFO

Keywords:
Syncope;
Risk stratification;
TLOC

ABSTRACT

Background: Background Syncope is a frequent problem among patients who present to the ED, accounts for 3% of emergency department admission and 1% of hospitalization. It is characterized by a comparatively short and self-limited loss of consciousness, which is caused by temporary cerebral hypoperfusion.
Objective: Therefore, Risk stratification performed in the ED can guide triage decisions, and Risk-stratifying patients into low, moderate, and high-risk groups can assist medical decisions and determine the patient's disposition.
Discussion: The central point of syncope progression pathophysiology is the reduction of systemic blood pressure (BP) with a drop in global cerebral blood flow. Based on the European Society of Cardiology (ESC) syncope practice guidelines, syncope is classified into three categories, Neurally-mediated syncope (neural reflex syncope), Orthostatic hypotension, Cardiac syncope. Proper evaluation of syncope cases could in turn enable timely hospitalization and treatment by syncope experts. Assessment of a patient with syncope can be difficult, requiring a wide variety of medical testing with high health care costs. Sometimes, even after a careful examination, it may not be possible to determine a definitive etiology for syncope. Given these uncertainties, about one-third of emergency room (ER) syncope/collapse patients are referred for assessment to the hospital, including non to low-risk patients. establish the urgency of any further work-up.
Conclusion: Syncope assessment and treatment are very difficult, and syncope cases should be treated and dispositioned properly using proper risk stratification guidelines.

1. Introduction

Syncope is one of the most prevalent conditions in ED patients, comprising 1% of hospital admissions and 3% of referrals to the emergency room.[1] It is a condition characterized by a spontaneous, self-limited episode of lack of consciousness arising from a sudden disruption of the delivery of oxygen to the brain, which is nearly invariably triggered by a sudden absence of blood flow. Syncope is characterized by a fast onset, brief duration, and full spontaneous recovery.[2]

Reflex syncope (35-48 percent) accompanied by orthostatic hypotension (4-24 percent), cardiac (5-21 percent), non-syncope Transient Loss Of Consciousness (TLOC) are the most frequent triggers of syncope ED visit and ranges from 17 to 33 percent of presentation of syncope can remain unidentified.[3,4]

Clinical decision making can be challenging in the management of syncope patients admitted to ED.[5]

Given the resulting uncertainties, hospital admission remains a common practice in dealing with syncope. Half of these patients are rehospitalized for further examination; half of which will be released without a convincing diagnosis.[6]

When the cause of symptoms remains unclear after initial evaluation in ED, Assessing the probability of serious implications, such as the risk of major coronary events or sudden cardiac death, is essential. This risk stratification profile will help direct the future treatment and condition of the patient.[7]

2. Pathophysiology of Syncope

The central point of syncope progression pathophysiology is the reduction of systemic blood pressure (BP) with a drop in global cerebral blood flow. Systemic BP is the product of total peripheral resistance and cardiac output; syncope can result from a fall in either. However, to a ranging degree, both processes frequently work together in syncope.[8,9]

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https://doi.org/10.21776/ub.hsj.2020.001.04.03
Received 9 November 2020; Received in revised form 30 November 2020; Accepted 15 December 2020
Available online 25 December 2020

3. Classification of Syncope

The syncope classification is based largely on the fundamental pathways contributing to the occurrence of transient cerebral hypoperfusion. Based on the European Society of Cardiology (ESC) syncope practice guidelines, syncope is classified into three categories:[9]

(1) Neurally-mediated syncope (neural reflex syncope)

A loss of consciousness (LOC) associated with reflex vasodilation and bradycardia is defined by neurally mediated syncope as a response to certain triggers.[10] There are several conditions in this type of syncope. Vasovagal syncope is the most important and also the most common one in this category. Precipitating causes for this disorder include prolonged sitting posture or standing position, mental stress, pain, heat, venous puncture, alcohol consumption, dehydration, use of diuretics, and vasodilators. Prodromes, such as nausea, vomiting, stomach pain, diaphoresis, pallor, palpitations, and dizziness, are often preceded by vasovagal syncope and are most frequent in young adults.[8]

Carotid Sinus Syncope (CSS) is the second most common. It is a syncope-related autonomic nervous system condition that arises mainly in the elderly and mostly in men.[11] A clinical phenomenon precipitated by carotid sinus massage is called Carotid Sinus Hypersensitivity (CSH). As the CSH is a clinical finding and the CSS is a clinical manifestation, the former should be differentiated from the latter. CSS is identified only if carotid massage induces enough hypotension and/or bradycardia (usually 6 seconds) to replicate the symptom.[11,12]

The third type of reflex faint is situational syncope; it involves syncope caused by a variety of events such as defecation, swallowing, coughing, or micturition.[12] Identification of the trigger is of importance because of its therapeutic implications, with avoidance of the trigger possibly preventing further syncopal episodes.

(2) Orthostatic hypotension

Orthostatic hypotension is the second most common cause of syncope. Orthostatic syncope occurs as a result of the failure of the body to sustain sufficient cerebral perfusion blood pressure as the individual transitions to the upright position, resulting in TLOCC in turn.[8] Patients that more likely to have orthostatic syncope include the patients taking vasodilatory medications, pregnant women, and the elderly.

The postural change from lying down to standing transfers 500 to 800 mL of blood to the abdomen and lower limbs, allowing the venous return to the heart to suddenly decline. In turn, this transition contributes to reduced venous return to the heart and subsequent decrease in stroke volume and cardiac filling pressure due to cerebral hypoperfusion and hypotension.[8] Maneuvers with physical counterpressure such as muscle straining and leg-crossing have been shown to help improve venous return by increasing the activity of the muscle pump.[9]

(3) Cardiac syncope

The most frequent life-threatening cause of syncope is cardiac syncope. The most prominent cardiac sources of syncope are arrhythmias. A more frequent cause of syncope than tachyarrhythmia is bradyarrhythmia.[8] Symptomatic hypotension can occur in this group as a result of high-grade atrioventricular block, sinus pauses, or asystole occurring at the end of atrial arrhythmia.[13] Ventricular tachycardia (VT) is the most common syncope-causing tachyarrhythmia. Supraventricular tachycardia (SVT) can also induce syncope, although less extreme symptoms such as palpitations, dyspnea, and lightheadedness are found in most patients with supraventricular arrhythmias.

Another cause of cardiac syncope is structural heart disease. Blood flow obstruction, acute myocardial infarction, or pulmonary embolism can result in syncope. In these cases, cortical hypoperfusion is mostly attributed to the immediate hemodynamic effect of an anatomical disorder, as well as neurally regulated reflexes or lack thereof.[12]

4. Initial Evaluation of Syncope in the Emergency Department

Syncope's presentation and clinical situation help direct the extent of the examination of the ED. It allows a detailed determination of the origin of syncope in all patients.[7] There is several steps to follow in evaluating syncope in ED. First, the initial evaluation should begin by differentiating between non-syncopal transient loss of consciousness (TLOC), and syncope by asking the following: (1) Did the patient experienced complete LOC? (2) Was the LOC transient with a short duration and rapid onset? (3) Did the patient recover completely, spontaneously, and without sequelae? and (4) Did the patient lose postural tone? If one or more of the answer is negative, other non-syncopal causes of transient LOC should be suspected.[8,14]

Second, a comprehensive assessment comprising of history, physical examination (including standing and supine blood pressure), and electrocardiogram to obtain an aetiological diagnosis, including any additional tests.[14] In 66% of cases, it can provide an initial diagnosis, with a diagnostic precision of 88 percent.[13]

The last step is risk stratifications of patients with undetermined syncope. This step is to focus on determining whether the patient is at increased risk for a cardiovascular event or death and to guide the next approach for the patient.[14]

5. Risk Stratification of Syncope

Since the cause of syncope in the ED can be difficult to determine, risk stratification is an important part of decision-making by ED physicians.[15] The following course of action is evident in situations where the cause of TLOC is determined with certainty during the initial evaluation.

More frequently, though the diagnosis is uncertain and the responsible providers face the challenge of deciding between urgent hospitalization and prompt outpatient examination.[12] Historically, doctors have preferred a cautious course of action leading to hospital admission of many more patients than is required.

The function of ED risk stratification assessment is essential for the following reasons: (i) it helps to define the prognosis, (ii) affects the decision of triage, (iii) establishes urgency for further evaluations and expert assessment, and (iv) ensures sufficient conversations exist with patients.[16]

The 2018 Guidelines for the Diagnosis and Treatment of Syncope of the European Cardiology Society (ESC) recommend that ED risk stratification should be carried out using the following high-risk (suggestive of a critical condition) and low-risk (suggestive of a moderate condition) criteria of risk stratification in patients with syncope at the initial ED evaluation.[9,17]

After ED risk stratification, the ESC ED risk stratification flowchart shown in Figure 2 should be used to assess the management of the corresponding patient.[9,17] The last question that has to be answered by the attending clinician is whether to admit the patient to the hospital. In the case of recurrent syncope, a patient with only low-risk characteristics and no high-risk characteristics can be categorized as low-risk and can be easily discharged from the ED with fast-tracking to a Syncope Unit (SU). There is no need for further

Table 4. IMPROVE bleeding RAM: score ≥ 7 indicates high bleeding risk.[29]

Syncopal Event
Low Risk
i) Associated with prodrome typical of reflex syncope (e.g. light-headedness, feeling of warmth, sweating, nausea, vomiting); ii) After sudden unexpected unpleasant sight, sound, smell, or pain; iii) After prolonged standing or crowded, hot places; iv) During a meal or postprandial; v) Triggered by cough, defaecation, or micturition vi) With head rotation or pressure on carotid sinus (e.g. tumour, shaving, tight collars); vii) Standing from supine/sitting position
High risk (red flag)
Major: i) New onset of chest discomfort, breathlessness, abdominal pain, or headache; ii) Syncope during exertion or when supine; iii) Sudden onset palpitation immediately followed by syncope.
Minor (high risk only if associated with structural heart disease or abnormal Electrocardiogram): i) No warning symptoms or short (<10 s) prodrome; ii) Family history of Sudden Cardiac Death (SCD) at young age; iii) Syncope in the sitting position
Past medical history
Low Risk
i) Long history (years) of recurrent syncope with low-risk features with the same characteristics of the current episode; ii) Absence of structural heart disease.
High risk (red flag)
Major: Severe structural or coronary artery disease (heart failure, low left ventricular ejection fraction; LVEF or previous myocardial infarction)
Physical Examination
Low Risk
Normal Examination High Risk (Red Flag) i) Unexplained systolic blood pressure (BP) in the ED <90 mmHg; ii) Suggestion of gastrointestinal bleed on rectal examination; iii) Persistent bradycardia (<40 beats per minute; bpm) in awake state and in absence of physical training; iv) Undiagnosed systolic murmur
Low risk
Normal
High Risk (Flag)
Major i) ECG changes consistent with acute ischaemia; ii) Mobitz II second- and third-degree atrio-ventricular (AV) block; iii) Slow Atrial Fibrillation (AF) (<40 bpm); iv) Persistent sinus bradycardia (<40 bpm), or repetitive sinoatrial block or sinus pauses >3 seconds in awake state and in absence of physical training; v) Bundle branch block, intraventricular conduction disturbance,ventricular hypertrophy, or Q waves consistent with ischaemic heart disease or cardiomyopathy; vi) Sustained and non-sustained Ventricular Tachycardia (VT); vii) Dysfunction of an implantable cardiac device (pacemaker or implantable cardioverter defibrillator); viii) ST-segment elevation with type 1 morphology in leads V1−V3 (Brugada pattern); ix) QTc >460 ms in repeated 12-lead ECGs indicating long QT syndrome (LQTS).
Minor (high risk only if history consistent with arrhythmic syncope): i) Mobitz I second-degree AV block and 1° degree AV block with markedly prolonged PR interval; ii) Asymptomatic inappropriate mild sinus bradycardia (40-50 bpm), or slow AF (40- 50 bpm); iii) Paroxysmal supraventricular tachycardia (SVT) or atrial fibrillation; iv) Pre-excited QRS complex; v) Short QTc interval (≤340 ms); vi) Atypical Brugada patterns; vii) Negative T waves in right precordial leads, epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy.

Note; Sudden Cardiac Death= SCD, BP= blood pressure, ED= emergency department; LVEF= left ventricular ejection fraction; ECG= Electrocardiography; AV= atrio-ventricular; VT= Ventricular Tachycardia; LQTS= long QT syndrome

clinical examination in these patients in the ED and their possible diagnosis is reflex, situational, or orthostatic syncope. Reassurance or counseling may aid them.[17,18]

Patients would be categorized as intermediate-risk patients with no high or low-risk characteristics. It is difficult to work with these patients and many need specialist syncope opinions.[15] Instead of being discharged, these patients must be treated in the ED observation unit. A study from Shen et al. found that a dedicated ED syncope unit, where patients could remain up to 6 hours, dramatically increased ED diagnostic yield, and decreased hospital admission and overall hospital stay without impacting recurrent syncope and all-cause mortality among patients at intermediate risk.[19] Patients underwent constant cardiac monitoring, hourly control of vital signs, echocardiography (in patients with abnormal ECG or abnormal cardiovascular examination results), tilt table screening and if necessary, expert consultation.

Patients with any high-risk features should be identified as high-risk and should not be released from the ED because they need a prompt and comprehensive clinical approach and may need immediate care. This is likely to suggest that high-risk patients should be hospitalized for diagnostic or therapeutic reasons until patients can access immediately advanced investigations such as echocardiography, ECG evaluation, specialized cardiovascular evaluation, and syncope analysis by a syncope expert during either an extended ED stay or a syncope clinical decision/research unit.[20]

Recently, The Canadian Syncope Risk Score (CSRS), which includes clinical variables, ECG and elevated troponin (> 99th percentile of the average population) and suspected EDD diagnosis, was established as a new syncope risk score. When comparing the area under the curve (AUC), the CSRS performed better than not only cardiac biomarkers in forecasting mortality and adverse effects but also cardiac biomarkers in comparison with older risk ratings.[16]

6. Management of Syncope

Treating a syncope patient has three objectives: (1) prolong survival, (2) prevent traumatic injuries, and (3) preventing syncope recurrence. A syncope patient's response to treatment is largely dependent on the syncope's cause and mechanism.[8,9]

Patients with reflex syncope are generally treated with non-pharmacological therapy, such as education, alteration of lifestyle,

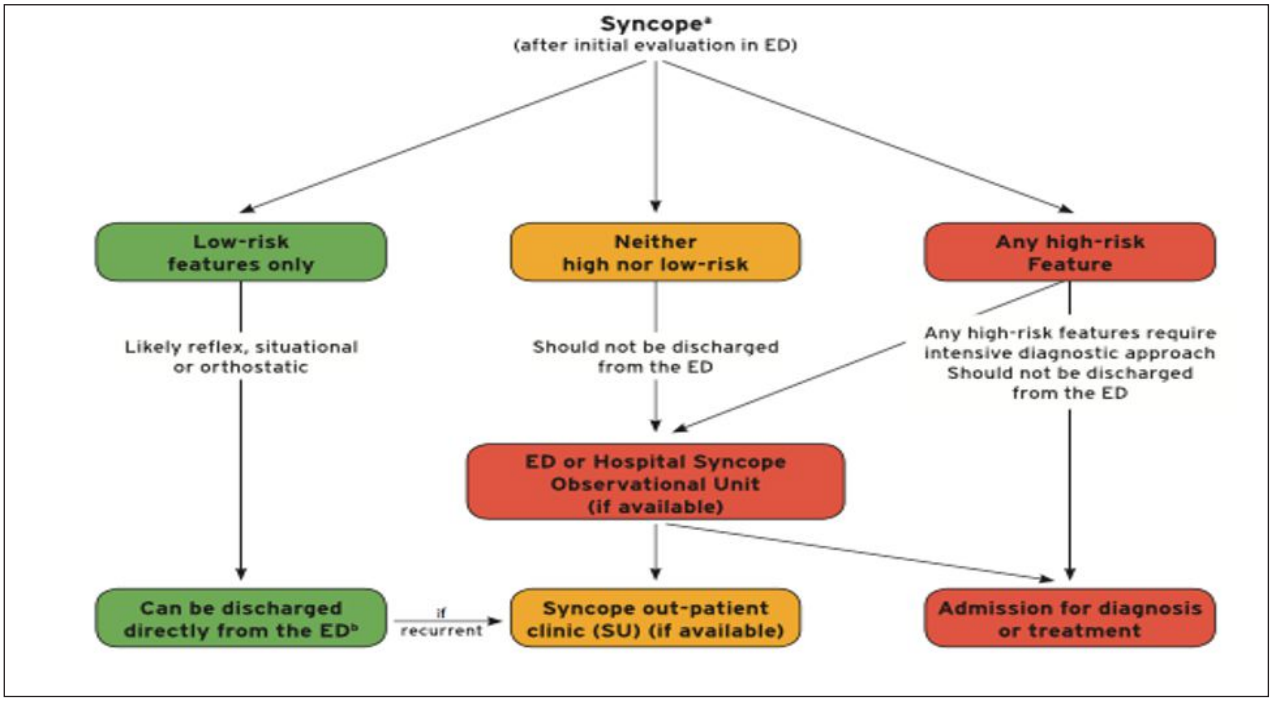

Figure 2. Emergency department risk stratification flow chart. ED = emergency department;SU = syncope unit. Reproduced from Brignole M, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. European Heart Journal(2018) 00, 1-69, doi:10.1093/eurheartj/ehy037.

and rereaasurance of the mild nature of the disease.[21] Another approach used for the treatment of neurally mediated syncope has resulted from the effectiveness of "physical" measures and maneuvers in the treatment of patients with this condition. Supplementary treatment may be warranted in patients with severe forms, such as when the syncope is very frequent and impair quality of life or when it occurs during high-risk activities (e.g. driving, machine operation, flying, etc.).[21] However, the pacing is considered not to be very effective in preventing syncope in most patients despite early favorable reports. On the other hand, the ISSUE-2 trial indicated that if ILR recording has been described with marked bradycardia during spontaneous syncope, then pacing could be required in recurrent fainters.[22]

Patients with carotid sinus syndrome who have frequent syncope or falls resulting from carotid sinus hypersensitivity in recommended to undergo dual-chamber pacemaker implantation.[8] But if the diagnosis of carotid sinus hypersensitivity is based on longer than a 3-second pause with carotid sinus massage without clear, provocative events, pacemaker implantation is less strongly recommended (class IIA)

Management for syncope due to arrhythmia is based on the underlying cardiac rhythm disturbance. A patient with syncope related to AV block would be a pacemaker in most situations. However, a patient with syncope secondary to heart block in the setting of an inferior wall myocardial infarction will not usually require a permanent pacemaker because the heart block usually resolves spontaneously. Similarly, heart block resulting from neurally mediated syncope does not generally require pacemaker implantation.[8]

Management of a syncope patient related to Wolff-Parkinson-White syndrome typically involves catheter ablation, and treatment of a patient with syncope related to VT or in the setting of ischemic or non-ischemic cardiomyopathy may involve implantation of a defibrillator.[23]

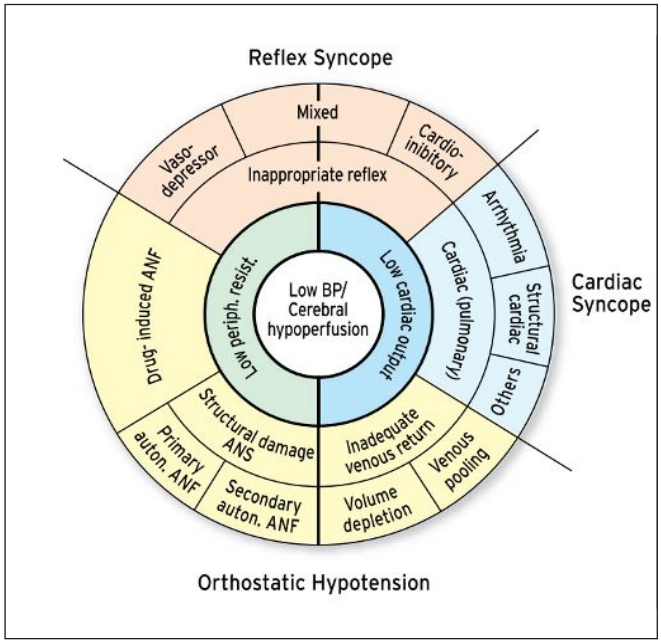

Figure 1. Pathophysiological basis of the classification of syncope. Reproduced from Brignole M, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. European Heart Journal(2018) 00, 1-69, doi:10.1093/eurheartj/ehy037

7. Conclusion

Syncope assessment and treatment are very difficult, and syncope cases should be treated and dispositioned properly using proper risk stratification guidelines. The ESC recommendations for the

first time include an entire section dedicated to the treatment of the syncope patient presenting to the ED. In the initial assessment of syncope patients, risk stratification is an essential element since it enables immediate hospitalization at imminent risk of life-threatening conditions as it could offer an ability to recognize and support syncope patients. When optimal, risk stratification also provides the ability to prevent needless patients from hospitalization at low risk, thus minimizing healthcare spending.

7. Declarations

7.1. Ethics Approval and Consent to participate
Patient has provided informed consent prior to involve in the study.

7.2. Consent for publication
Not applicable.

7.3. Availability of data and materials
Data used in our study were presented in the main text.

7.4. Competing interests
Not applicable.

7.5. Funding source
Not applicable.

7.6. Authors contributions
Idea/concept: PAK. Control/supervision: AR, NK, SW. Literature review: PAK. Writing the article: PAK. Critical review: AR, NK, SW. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

7.7. Acknowledgements
We thank to Brawijaya Cardiovascular Research Center.

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