

Clinical update no. 517

11 July 2018

Case: A 72yr-man is brought to hospital because the family observed him to become unresponsive while eating dinner. He looked pale and was about to fall out of his chair when they lowered him to the ground. There were no convulsive movements and he recovered fairly quickly back to normal. There was no chest pain, headache or other concerning symptoms. He has not had this before. He takes aspirin, perindopril for BP and a statin. There is no other history of cardiac disease. ECG is sinus rhythm with no diagnostic features. Examination and CXR are normal. What further workup is needed and does he need admission for monitoring?

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 ESC GUIDELINES
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2018 ESC Guidelines for the diagnosis and management of syncope

The Task Force for the diagnosis and management of syncope of the European Society of Cardiology (ESC)

<https://academic.oup.com/eurheartj/advance-article/doi/10.1093/eurheartj/ehy037/4939241>

Figure 1 What is new in the 2018 syncope Guidelines?

2018 NEW RECOMMENDATIONS (only major included)

Management of syncope in ED (section 4.1.2)

- **Low-risk:** discharge from ED
- **High-risk:** early intensive evaluation in ED, SU versus admission
- **Neither high or low:** observation in ED or in SU instead of being hospitalized

MANAGEMENT IN EMERGENCY DEPARTMENT:

- List of low-risk and high-risk features
- Risk stratification flowchart
- Management in *ED Observation Unit* and/or fast-track to *Syncope Unit*
- Restricted admission criteria
- Limited usefulness of risk stratification scores

A focus is on risk stratification from ED which has the potential to reduce unnecessary admission. About 1% of patients with syncope die within a month of their ED visit. About 10% have a serious outcome, with two

thirds of these occurring while in ED. Syncope risk-stratification scores perform no better than clinical judgment.

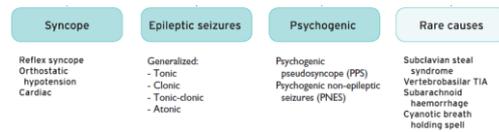
3.1 Definitions

- *Syncope* is defined as TLOC due to cerebral hypoperfusion, characterized by a rapid onset, short duration, and spontaneous complete recovery.

TLOC = transient loss of consciousness.

- The adjective *presyncope* is used to indicate symptoms and signs that occur before unconsciousness in syncope. Note that the noun *presyncope* is often used to describe a state that resembles the prodrome of syncope, but which is not followed by LOC.

Non traumatic TLOC is classified as follows:



Cardiac causes of syncope are mainly from arrhythmia (brady- or tachy-cardia); there are also structural causes (aortic stenosis, HOCM); ischaemia, PE, tamponade, congenital, other. Seizures and psychogenic are alternate causes of TLOC.

4. Diagnostic evaluation and management according to risk stratification

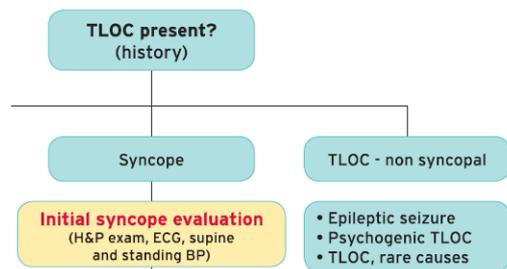


Table 6 High-risk features (that suggest a serious condition) and low-risk features

SYNCOPAL EVENT	
Low-risk	<ul style="list-style-type: none"> • Associated with prodrome typical of reflex syncope (e.g. light-headedness, feeling of warmth, sweating, nausea, vomiting)^{36,49} • After sudden unexpected unpleasant sight, sound, smell, or pain^{36,49,50} • After prolonged standing or crowded, hot places³⁶ • During a meal or postprandial⁵¹ • Triggered by cough, defaecation, or micturition⁵² • With head rotation or pressure on carotid sinus (e.g. tumour, shaving, tight collars)⁵³ • Standing from supine/sitting position⁵⁴
High-risk	<p>Major</p> <ul style="list-style-type: none"> • New onset of chest discomfort, breathlessness, abdominal pain, or headache^{26,44,55} • Syncope during exertion or when supine³⁶ • Sudden onset palpitation immediately followed by syncope³⁶ <p>Minor (high-risk only if associated with structural heart disease or abnormal ECG):</p> <ul style="list-style-type: none"> • No warning symptoms or short (<10 s) prodrome^{36,38,49,56} • Family history of SCD at young age⁵⁷ • Syncope in the sitting position⁵⁴

Low risk features include: prodrome, noxious stimulus, postural or with prolonged standing, during a meal/post prandial, trigger (cough, micturition), head rotation/pressure on neck.

High-risk

Major high risk features include chest discomfort, dyspnoea, and abdominal pain (? AAA/ other) or headache (? SAH).

Minor high risk features of concern *only* if structural heart disease or an abnormal ECG include no warning symptoms or a short <10 sec prodrome; family history of sudden death at a young age, syncope in a sitting position.

PAST MEDICAL HISTORY	
Low-risk	<ul style="list-style-type: none"> Long history (years) of recurrent syncope with low-risk features with the same characteristics of the current episode⁵⁶ Absence of structural heart disease^{27, 55}
High-risk	<p>Major</p> <ul style="list-style-type: none"> Severe structural or coronary artery disease (heart failure, low LVEF or previous myocardial infarction)^{26, 27, 35, 55, 59}

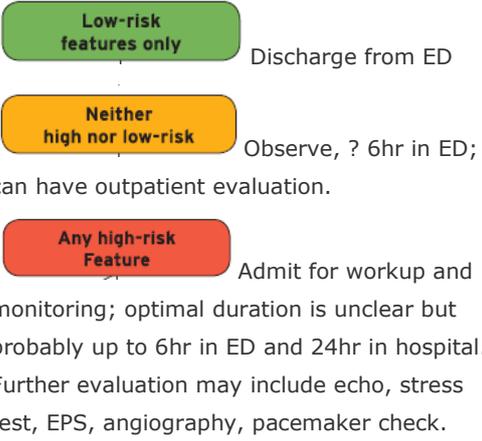
Recurrent syncope in the absence of structural heart disease is low risk. Structural or coronary artery disease/CCF/low LVEF are high risk features.

PHYSICAL EXAMINATION	
High-risk	
Major	<ul style="list-style-type: none"> Unexplained systolic BP in the ED <90 mmHg^{26, 59} Suggestion of gastrointestinal bleed on rectal examination⁴⁴ Persistent bradycardia (<40 b.p.m.) in awake state and in absence of physical training Undiagnosed systolic murmur⁶⁰

High risk exam features are unexplained SBP <90 mm Hg, possible GI bleed, persistent bradycardia <40 bpm and systolic murmur.

ECG*	
Low-risk	<ul style="list-style-type: none"> Normal ECG^{26, 35, 36, 55}
High-risk	<p>Major</p> <ul style="list-style-type: none"> ECG changes consistent with acute ischaemia Mobitz II second- and third-degree AV block Slow AF (<40 b.p.m.) Persistent sinus bradycardia (<40 b.p.m.), or repetitive sinoatrial block or sinus pauses >3 seconds in awake state and in absence of physical training Bundle branch block, intraventricular conduction disturbance, ventricular hypertrophy, or Q waves consistent with ischaemic heart disease or cardiomyopathy^{44, 56} Sustained and non-sustained VT Dysfunction of an implantable cardiac device (pacemaker or ICD) Type 1 Brugada pattern ST-segment elevation with type 1 morphology in leads V1-V3 (Brugada pattern) QTc >460 ms in repeated 12-lead ECGs indicating LQTS⁴⁶ <p>Minor (high-risk only if history consistent with arrhythmic syncope)</p> <ul style="list-style-type: none"> Mobitz I second-degree AV block and 1^o degree AV block with markedly prolonged PR interval Asymptomatic inappropriate mild sinus bradycardia (40-50 b.p.m.), or slow AF (40-50 b.p.m.)⁵⁶ Paroxysmal SVT or atrial fibrillation⁵⁰ Pre-excited QRS complex Short QTc interval (<340 ms)⁴⁶ Atypical Brugada patterns⁴⁶ Negative T waves in right precordial leads, epsilon waves suggestive of ARVC⁴⁶

High risk ECG features as described. Assess for ischaemia; rhythm disturbance on monitor; A-V block; conduction disturbance (e.g. bi- or tri- fascicular blocks that can be overlooked). Look specifically for features of HOCM, prolonged QT, type 1 Brugada, WPW, ARVC. Interpret as to whether history suggests a rhythm disturbance as the cause.



The **case vignette** described is low risk and can be safely discharged from ED. Syncope while seated without other concerning risk factors and a normal ECG is low risk.

Electrocardiographic monitoring	
Recommendations	
Indications	

Inpatient monitoring if high risk. Consider Holter monitor if frequent syncope; yield is low at 1 - 2% however symptoms without dysrhythmia suggests another cause. Consider external or internal loop recorder depending on risk and frequency;

Electrophysiological study	
Recommendations	
Indications	

Consider if otherwise unexplained with prior MI and other conditions with possible scarring, or with higher grade bundle branch block.

Echocardiography	
Recommendations	
Indications	

If suspect structural heart disease (HOCM, aortic stenosis); but not for syncope alone.

Exercise testing	
Recommendations	
Indications	

Not routinely required; of value if syncope related to exertion, and may show exercise induced hypotension or A-V block.

5. Treatment

There is a long discussion of treatment options as guided by the underlying cause. These updates are a review of current literature and are the views of Dr Brendon Smith, FACEM. Over time they will become outdated. They do not replace local treatment protocols and policy.