Review Article

Initial Evaluation of Patients with Presumed Syncope

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Syncope is a common clinical problem, but nevertheless is but one element of the broader issue of 'transient loss of consciousness' (TLOC). The first step is to ascertain whether the patient actually suffered a syncopal episode, and thereafter the goal must be to determine the basis of symptoms with sufficient confidence to assess prognosis and initiate an effective treatment strategy. The initial evaluation of these patients, which usually takes place in an emergency department (ED) or acute care facility, is challenging since patients are usually asymptomatic when they come for medical attention, may have little or no recall of the event, and witnesses, if any, often cannot provide reliable information. Given these circumstances, it is understandable that frontline physicians often tend to take a seemingly 'safe' approach, and admit both high-risk and intermediate-risk syncope patients to hospital. This strategy has many implications, including life-style and economic concerns for the patient, and health care management issues for physicians, hospital administrators and the overall health care system. The European Society of Cardiology (ESC) guidelines and several clinical studies provide helpful advice regarding "risk stratification" to help guide physicians in selecting patients for either early hospital admission or later oupatient subspeciality evaluation. The utility of syncope management units in the ED, and a guideline-based approach to the syncope patient, has tended to both diminish the number of undiagnosed cases and reduce the hospital admission rate. In this review, we have attempted to both highlight a cost-effective diagnostic pathway beginning with the initial evaluation of the patient with suspected syncope, and to provide criteria which may help frontline physicians better base their decisions regarding need for in-hospital versus outpatient clinic evaluation of syncope patients.
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Key words: syncope, loss of cosciousness, risk stratification

Introduction

Syncope is a syndrome characterized by a relatively sudden, temporary and self-terminating loss of consciousness; the causes may vary widely from patient to patient, but they have a common underlying pathophysiology; specifically, the transient loss of consciousness in syncope patients is the result of a temporary inadequacy of cerebral nutrient flow, most often triggered by a fall in systemic arterial pressure below the minimum needed to sustain cerebral blood flow.

Despite the fact that syncope is a relatively common clinical problem,[1–3] it is only one of many possible explanations for episodic transient loss of

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Table 1 Causes of real or apparent transient loss of consciousness (T-LOC)[1)]

Syncope	Non-syncope
• Neurally mediated (reflex) • Orthostatic hypotension • Cardiac arrhythmias as primary cause • Structural cardiac or cardiopulmonary disease • Cerebrovascular	• Disorders resembling syncope without any impairment of consciousness, e.g falls, psychogenic pseudo-syncope, etc • Disorders with partial or complete loss of consciousness, e.g seizure disorders, etc.

consciousness (TLOC) (**Table 1**). The differential diagnosis of TLOC is extensive. Consequently, the diagnostic evaluation should start not with the focus solely on 'syncope', but more broadly with consideration of a range of possible causes for real (e.g. seizures and concussion) or seemingly real (e.g. narcolepsy and certain psychogenic disturbances) TLOC (**Table 1**).[1)] Thus, epilepsy, concussions, metabolic disturbances and intoxications, and 'syncope mimics' need to be considered. In each of these cases, the pathophysiology of disturbance of consciousness differs from that of syncope, and therefore treatment would also be entirely different. For instance, seizures are a primary electrical disturbance of brain function, concussion is a disturbance of consciousness triggered by trauma, metabolic disturbances and/or intoxications may cause TLOC as the result of adverse toxic effects on cerebral function, while the basis for the so-called 'syncope mimics' varies and is poorly understood.

Among those patients in whom TLOC is deemed to be due to true syncope, the next step is thorough evaluation of the underlying cause. This is an important matter, since syncope, while perhaps relatively benign from a mortality perspective in the vast majority of cases, is nevertheless only infrequently an isolated event; syncope tends to recur, and physical injury resulting from falls or accidents, diminished quality-of-life, and possible restriction from employment or avocation are real concerns. Determining that certain individuals are at 'low mortality risk' is insufficient. The goal in every case should be to determine the cause of syncope with sufficient confidence to provide patients and family members with a reliable assessment of prognosis, recurrence risk, and treatment options.

In this review we summarize a strategy for undertaking the syncope evaluation. The objective is to determine, with as much certainty as possible, the basis for the patient's symptoms; only then is it possible to evaluate prognosis and define an effective treatment plan.

Initial evaluation

The initial medical evaluation of patients presenting with suspected syncope usually occurs in the Emergency Department (ED) or acute care clinic. After determining as best as possible that the patient has experienced true syncope, the initial evaluation should then focus on the following key points

- A detailed description of symptomatic events
- Ascertaining whether clinically important structural heart disease is present
- Clinical features of the history that suggest a diagnosis.

As a rule, the initial evaluation begins with a comprehensive medical history and physical examination, an electrocardiogram (ECG) and often an echocardiogram. In experienced hands, the cause of syncope can be established by these steps alone in approximately 60 percent of patients.[4,5)]

Even after it is concluded that 'syncope' was the most likely cause of TLOC, the subsequent clinical assessment remains challenging for a number of reasons. First, the affected individual is usually asymptomatic on arrival for medical attention. Consequently, it is rare to find any helpful physical findings or ECG observations. Second, the victim (especially if in an older age group) may not be able to provide a detailed history of the circumstances, and in other cases the reported history may not be reliable. Third, the event(s) may not have been witnessed, or even if witnessed, the observer is often unable to recollect details. The physician must spend considerable time elicting the story as best as possible. Finally, syncope has many possible causes (**Table 2**), and each needs to be carefully considered.

The medical history is the physician's most valuable tool in the initial syncope assessment. Not infrequently the history alone is diagnostic of the cause of syncope and no further testing is needed. The most common example is when the history is indicative of a 'classic' vasovagal faint or one of the so-called 'situational' neurally-mediated reflex

Table 2 Main causes of syncope[1)]

Neurally-mediated (reflex)
• Vasovagal
• Carotid sinus syncope
• Situational syncope (e.g micturition, post-micturition, swallowing, cough, defecation, etc)
• Glossopharyngeal neuralgia
Orthostatic hypotension
• Autonomic failure
• Drug (and alcohol)-induced orthostatic syncope
• Volume depletion
Cardiac arrhythmias as a primary cause
• Sinus node dysfunction
• Atrioventricular conduction system disease
• Paroxysmal supraventricular and ventricular arrhythmias
• Inherited syndromes (e.g long QT syndrome, Brugada syndrome)
• Implanted device malfunction
• Drug-induced pro-arrhythmias
Structural cardiac or cardiopulmonary disease
• Cardiac valvular disease
• Acute myocardial infarction or ischemia
• Obstructive cardiomyopathy
• Atrial myxoma
• Acute aort dissection
• Pulmonary embolus/pulmonary hypertension
Cerebrovascular
• Vascular steal syndromes

faints. In such cases, an experienced physician can feel comfortable that the basis for symptoms has been determined, and can proceed with appropriate treatment steps. On the other hand, if the careful history is suggestive but inconclusive, the physician has still made considerable progress; subsequent steps in the evaluation can be focused, efficient, and cost-effective (**Figure**). A 'shotgun' approach to diagnosis is to be avoided.

Among the most important factors to identify in the patient with suspected syncope is whether he/she has a history of or physical findings suggestive of underlying structural heart disease. In this regard, the inclusion of an echocardiogram as part of the initial evaluation of suspected syncope patients can be a valuable aid for the clinician. The presence of heart disease is an independent predictor of a 'cardiac cause' for syncope (i.e., a primary arrhythmic cause or a cause based on a structural cardiac abnormality leading to a transient hemodynamic disturbance), with a sensitivity of 95% and a specificity of 45%; by contrast, the absence of heart disease excludes a cardiac cause of syncope in 97% of the patients.[6)]

Syncope in conjunction with exertion raises special concerns. In particular, if the faint occurs in 'full flight', one must consider the possibility of structural and/or dynamic heart lesions producing a relatively 'fixed' cardiac output in the setting of vascular dilatation (e.g., severe aortic or mitral valvular stenosis, hypertrophic cardiomyopathy). However, syncope during or early following exercise (even moderate exertion such as climbing stairs) can also occur in patients with severe autonomic dysfunction (e.g., pure autonomic failure) in whom vascular control is unable to maintain adequate cerebral perfusion pressure. In addition, on rare occasions syncope accompanying exertion may occur as a consequence of a neurally-mediated reflex faint (i.e., post-exertional variant of vasovagal faint); however, in these latter cases the faint typically occurs shortly after completion of, not during, the exercise.

A comprehensive discussion of medical history taking in patients with suspected syncope lies beyond the scope of this review. However, we have included in the Appendix a brief summary of certain key features to be considered in this process. In brief,

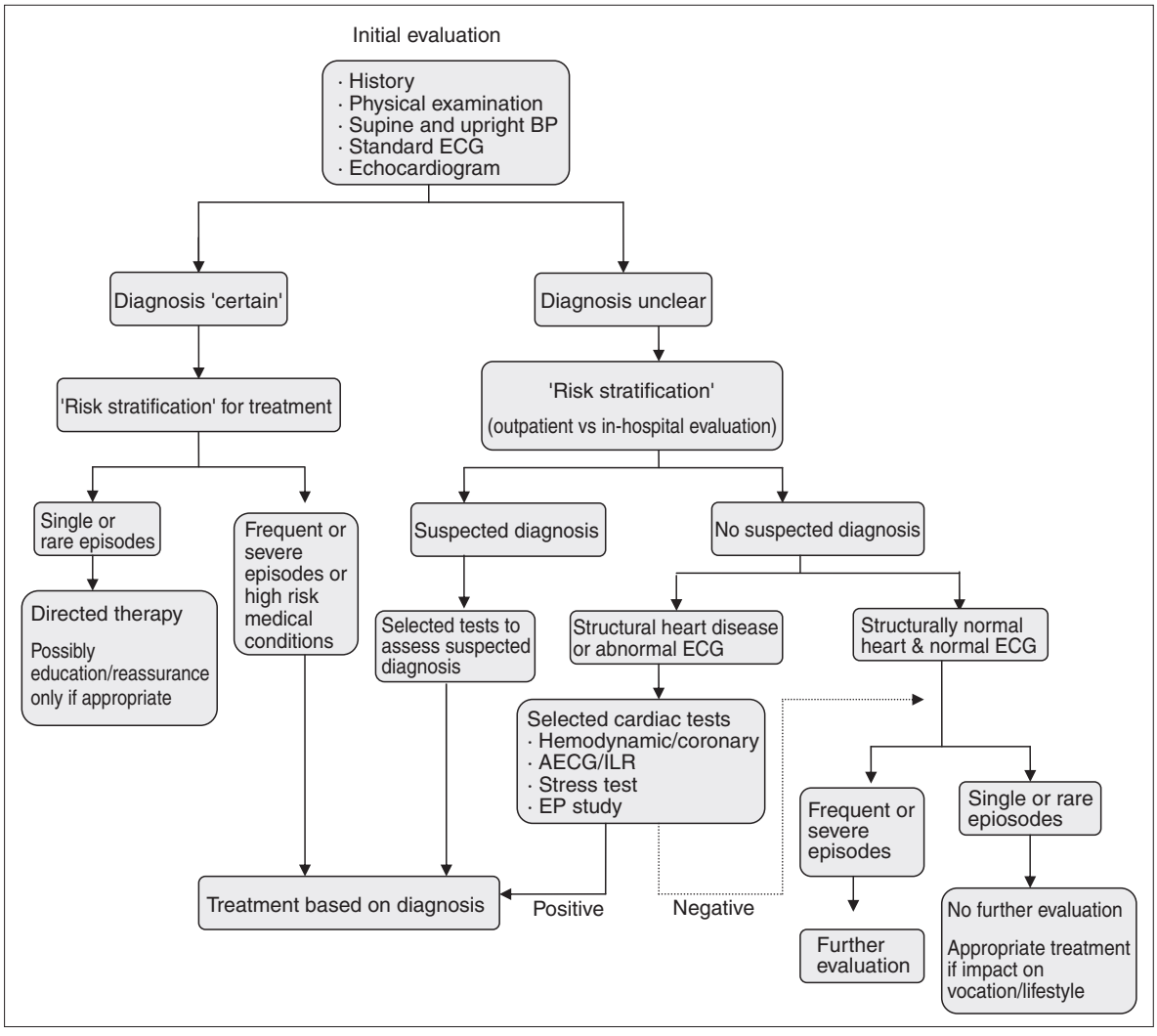

Figure Initial evaluation and further management strategy of syncope based on European Society of Cardiology Task Force Guidelines.[1)]

ECG, electrocardiogram; BP; blood pressure; AECG, ambulatory electrocardiogram; ILR, implantable loop recorder; EP, electrophysiologic.

it is essential that as many symptom events as possible be reviewed in detail, the preceding circumstances, premonitory symptoms (**Table 3**), and subsequent outcome should be documented, along with careful note being made of co-morbidities (for example diabetic neuropathy, autonomic dysfunction). If a pattern emerges regarding symptom events, the diagnosis may become evident without need for further tests or with the aid of only a few selected confirmatory tests. A pre-prepared patient questionnaire may prove helpful to save time and yet acquire the needed detail.

As alluded to earlier, based on a careful initial evaluation the physician may determine that a 'Certain' diagnosis has been established and treatment can be contemplated. However, in many instances the initial assessment may yield only a 'Suspected' cause necessitating selected further testing, or might be entirely unrevealing. In the latter case the diagnosis remains unclear or 'Unknown'. For each of these circumstances, the diagnostic flow pathway illustrated in **Figure** (modified after that devised by the European Society of Cardiology Syncope Guidelines Task Force [ESC guideline]) may prove helpful. Ultimately, the goal is to establish, in an efficient and cost-effective manner, the cause(s) of syncope with sufficient confidence to provide patients and families a reliable recommendation regarding prognosis and treatment options.

Risk stratification in the emergency department based on initial evaluation

Wherever the initial syncope evaluation leads,

physicians almost always face the question "Does this individual need in-hospital care for further evaluation and/or initiation of treatment?". The outcome of this decision has many implications, including life-style and economic concerns for the patient, as well as health care management issues (e.g. bed availability, hospital costs, and laboratory utilization) for the broader health care system.

In general, the driving force determining whether the patient with presumed syncope should be hospitalized is most often concern regarding the individual's immediate mortality risk. Secondary issues of importance include potential for physical injury (e.g., falls risk), and to a lesser extent the issue of whether certain treatments inherently require hospital monitoring for safe initiation. In any case, the current practice pattern for ED physcians and general practitioners is to take a presumably 'safe' approach and admit a large proportion of syncope patients to hospital. This strategy is mostly based on the presumption that syncope is commonly a harbinger of sudden death among patients. However, most studies indicate that syncope, by itself, is not an independent predictor of mortality. The real risk, in the vast majority of cases (exclusions may be the 'channelopathies'), is the risk associated with any evident coexisting cardiovascular disease.[7)]

In terms of the apparent preference for frontline physicians to admit syncope patients to hospital, Bartoletti et al.[8)] evaluated the frequency with which ED physicians who were specially trained with respect to ESC Guidelines (particularly with regard to hospital admission recommendations), elected in-hospital or outpatient evaluation for patients presenting with syncope. During the approximate 2-year enrollment, 1,124 patients were deemed to have true syncope and 440 of these (39%) had at least one marker supporting admission for evaluation; 393 of these 440 patients (89%) were admitted. In contrast, 684 patients met no evident admission criterion; 511 of 684 patients (75%) were appropriately discharged from the ED, but 25% were admitted. The high 25% admission rate in the low-risk patients despite being backed-up by guideline statement showed that among ED physicians there remain unresolved problems that need to be addressed in order to decrease potentially unnecessary hospital admissions.

In instances when the etiology of syncope is considered 'certain' after the initial clinical evalua-

Table 3 Clinical features suggestive of specific causes of real or apparent loss of consciousness[1)]

Neurally mediated syncope
• Absence of cardiac disease
• Long history of syncope
• After sudden unexpected sight, sound, smell or pain
• Prolonged standing or crowded, hot places
• Nausea, vomitting associated with syncope
• During the meal or in the absorptive period after a meal
• With head-rotation, pressure on carotid sinus (as in tumors, shaving, tight collars)
• After exertion
Syncope due to orthostatic hypotension
• After standing-up
• Temporal relationship with start of medication leading to hypotension or changes of dosage
• Prolonged standing especially in crowded, hot places
• Presence of autonomic neuropathy or Parkinsonism
• After exertion
Cardiac syncope
• Presence of definite structural heart disease
• During exertion or supine
• Preceded by palpitation
• Family history of sudden death
Cerebrovascular syncope
• Arm exercise
• Differences in blood pressure or pulse in the two arms

Note that the causes on the top of the list are more frequent and their likelihood of being the cause decreases as we move towards the bottom.

tion, the need for hospitalization depends in part on the immediate risk posed to the patient by the underlying problem, and in addition on the treatment proposed. Thus, for example, patients with syncope accompanying complete heart block, ventricular tachycardia, acute aortic dissection, or pulmonary embolism, should be admitted to the hospital and preferably to an ECG monitored unit. Similarly, if the initiation of antiarrhythmic drug therapy is contemplated, the proarrhythmia risk and potential for ameliorating that risk by in-hospital monitoring must be considered. On the other hand, most vasovagal fainters can be sent home after careful discussion of the nature of the problem and simple preventative maneuvers (e.g., hydration, avoidance of hot crowded environments, etc). Later clinic follow-up suffices in most of these cases.

For patients with syncope in whom the etiology remains 'suspected' or entirely 'unknown' after the initial evaluation, the need for hospitalization is currently less well defined; consequently, there has been interest in developing so-called "risk stratification" methods. The goal of such 'risk stratification' is to ascertain the relative risk for early adverse outcomes (particularly mortality, but also falls/injury risk) using patients' clinical features and presenting characteristics. Based on this assessment the physician determines if hospitalization is prudent. **Table 4** summarizes ED risk stratification criteria used in several recent clinical studies or reports: 1) Syncope Evaluation in the Emergency Department Study (SEEDS), 2) The San Francisco Syncope Rule (SFSR) study, 3) the European Society of Cardiology and American College of Emergency Physicians (ACEP) guidelines, and 4) The Evaluation of Guidelines in Syncope Study (EGSYS).[1,2,9,10] In each case, stratification uses clinical data that is readily accessible to the ED physician or general practitioner. These data include patient's symptoms, signs, basic laboratory results and clinical experience ('judgement'). The latter is, of course, a commodity that is almost impossible to assess.

The following provides an overview of common circumstances for which hospitalization is recommended or conversely is usually not needed.

Patients with 'high risk'

Several prognostic markers identify syncope patients who should be considered for in-hospital evaluation. Syncope associated with symptoms suggestive of acute myocardial ischemia, acute aortic dissection, signs of congestive heart failure, and/or suspicion of hemodynamically concerning underlying structural heart disease (e.g., valvular aortic or mitral stenosis, severe pulmonary hypertension) have the highest immediate mortality and recurrence risk. At similar high risk are syncope patients with certain ECG abnormalities, including high-grade atrioventricular (AV) block, cardiac rhythm pauses of 3 to 5 seconds or greater, pre-excitation syndromes (e.g Wolff-Parkinson White Syndrome), suspected arrhythmogenic right ventricular cardiomyopathy (ARVD) based on ECG or imaging evidence (although this is not generally available in the ED or clinic), idiopathic ventricular tachycardia, long or short QT syndromes (LQTS, Short QTS) or Brugada syndrome. Patients with syncope during exercise, and syncope causing motor vehicle accidents or severe injury should also be carefully considered for in-hospital evaluation unless the history strongly supports the diagnosis of exercise variant of neurally-mediated reflex syncope.

An additional troublesome risk marker in the syncope patient is a family history of premature sudden death. This history might be indicative of ischemic heart disease, but may also signal any of a variety of familial conditions that may first present as syncope (e.g LQTS, ARVD, Brugada syndrome etc). In the SEEDS study, a family history of unexpected death was considered to pose an intermediate risk to the patient with syncope, and consequently such patients were first observed in an ED-based syncope management unit instead of immediate hospital admission.[9] However, ESC guidelines strongly recommend hospital admission for the patients with family history of sudden death.[1]

In the Osservatorio Epidemiologico sula Sincope nel Lazio (OESIL) study, 4 syncope patient characteristics were associated with adverse outcome: age >65 years, a clinical history of cardiovascular disease, syncope without prodromal symptoms, and an abnormal ECG.[11] The presence of each characteristic scored one point. One year mortality has been shown to increase with increasing score (0% for a score of 0; 0.8% for 1 point; 19.6% for 2 points; 34.7% for 3 points; 57.1% for 4 points; $p < 0.0001$ for trend).

In the San Francisco Syncope Rule (SFSR) study, a high-risk was defined as those syncope patients having any of the following 5 risk factors: abnormal ECG (non-sinus rhythm or new abnormality), anemia (hematocrit <30%), a complaint of shortness of breath, systolic hypotension (<90 mmHg) and a history of congestive heart failure.[2] The SFSR criteria was found to be 96% sensitive and 62% specific for short-term serious outcomes (7 days of their initial visit) (**Table 4**). However, a recent prospective study to validate the SFSR found a much

Table 4 Risk Stratification Criteria From Several Studies

SEEDS[9]	ESC[1]	ACEP[10]	SFSR[2]	EGSYS score[15]	
High risk	Hospital admission strongly recommended for diagnosis	Admission recommended	High risk	Variable	Score
• Chest pain compatible with acute coronary syndrome • Signs of severe CHF • Moderate to severe valvular heart disease • History of ventricular arrhythmia • ECG/cardiac monitor signs of ischemia • Prolonged QTc (>500 ms) • Trifascicular block or pauses between 2 and 3 s • Third degree AV block • Persistent sinus bradycardia between 40–60 bpm • Atrial fibrillation or nonsustained ventricular tachycardia without symptoms • Pacemaker or defibrillator with dysfunction	• Suspected or known significant heart disease • ECG abnormalities suggestive of arrhyhmic syncope • Syncope during exercise • Syncope causing severe injury • Family history of sudden death	• Older age and associated comorbidities • Abnormal ECG (including acute ischemia, dysrhythmias, or significant conduction abnormalities) • Hematocrit <30% (if obtained) • History of presence of heart failure, coronary artery disease, or structural heart disease	• History of CHF • Shortness of breath • Anemia (hct <30%) • Sytolic hypotension (<90 mmHg) • Abnormal ECG (non-sinus rhythm or new abnormality	Palpitations preceding syncope	4
				Heart disease and/or abnormal ECG	3
				Syncope during effort	3
				Syncope while supine	2
				Precipitating and/or predisposing factors*	−1
				Autonomic prodromes §	−1
	Hospital admission strongly recommended for treatment				
	• Cardiac arrhythmias • Syncope due to cardiac ischemia • Syncope secondary to structural cardiac or cardiopulmonary diseases • Cardioinhibitory neurally mediated syncope when a pacemaker implantation is planned				
Intermediate risk	Occasionally may need to be admitted				
• Age >50 years • Previous history of CAD, MI, HF • CMP without active signs or symptoms on cardiac medications • Bundle branch block or Q wave without acute changes on ECG • Family history of premature unexplained sudden death (<50 years) • Symptoms not consistent with reflex-mediated or vasovagal cause • Cardiac devices without evidence of dysfunction • Physician judgement: A cardiac syncope is possible	• Patients without heart disease but sudden onset of palpitations shortly before syncope • Syncope in supine position • Frequent recurrent episodes • Patients with minimal or mild heart disease when there is a high suspicion for cardiac syncope			* warm, crowded place/ prolonged orthostatis/ fear, pain § Nausea/vomitting	
Low risk					
• Age <50 years • No previous history of cardiovascular disease • Normal cardiovascular examination • Symptoms consistent with reflex-mediated or vasovagal syncope • Normal ECG findings					

lower sensitivity (74%) and negative likelihood ratio (0.5) of the SFSR score to predict serious outcomes within 7 days of ED visit (death, myocardial infarction, pulmonay embolism, stroke, subarachnoid hemorrhage, significant hemorrhage, any condition causing or likely to cause a return ED visit and hospitalization for a related event).[12)]

A recent study, Short Term Prognosis of Syncope (STePS), evaluated the risk factors for both short-term and long-term severe outcomes in a group of 676 patients presenting with syncope. Severe outcomes included death, the need for major therapeutic procedures (cardiopulmonary resuscitation, pacemaker or defibrillator implant, and intensive care unit admittance, and acute antiarrhythmic therapy), and early (within 10 days) readmission to hospital. An abnormal electrocardiogram, concomitant trauma, absence of symptoms of impending syncope, and male gender were associated with a higher risk of severe outcome by 10 days. However, the positive predictive value was low (11%–14%) due to relatively low rate of these events. The long term (1 year) severe outcomes correlated with age >65 years, history of neoplasms, cerebrovascular diseases, structural heart diseases, and ventricular arrhythmias[13)].

Patients with intermediate risk

Syncope associated with age >50 years, history of structural heart disease but without signs of active consequences of disease, certain ECG abnormalities, family history of sudden death, cardiac devices without evidence of dysfunction, symptoms not consistent with vasovagal or reflex-mediated syncope and physician's judgement that a cardiac syncope is possible constitute the patient group with intermediate risk for adverse outcome (**Table 4**, SEEDS study). In SEEDS, patients with intermediate risk were placed in an ED-based syncope management unit where they received continuous cardiac monitoring for up to 6 hours, hourly vital signs, orthostatic blood pressure checks, and echocardiogram for patients with abnormal cardiovascular examination or ECG findings. During this evaluation, if patients developed high risk features they were admitted to the hospital, otherwise they were discharged to be followed-up in an outpatient clinic in <72 hours. For ED's without a well-developed syncope management unit, an observation unit similar to that often used for 'chest pain' assessment may prove useful, although careful additional staff training would be needed.

Patients with low risk

Patients in this group typically have no evidence of structural heart disease and have a normal baseline ECG. The syncope is considered of a 'relatively benign' nature; that is, the syncope is thought to be of neurally-mediated reflex or orthostatic cause. In this setting the risk of a life-threatening cardiac syncope is low, but 'falls risk' (and potential for physical injury) is still a consideration especially in older individuals. These patients can generally be stabilized in the ED or clinic. They can be reassured that they have a good prognosis in terms of survival, but must be warned regarding injury and accident risk. Consequently, before being sent home they should be provided cautionary advice regarding hydration, avoidance of provocative factors, driving concerns, avocation risk, and occupation risk given the real risk of recurrent events. Further, prompt follow-up in a 'syncope' or 'falls' clinic should be arranged so that definitive therapy is instituted in a timely manner.

Syncope management units

An as yet incompletely answered question is whether "syncope management units" (SMU) can help solve the problem of too many low- and intermediate-risk syncope patients being admitted to hospital where they often are submitted to unneeded expensive diagnostic tests. In this regard, two recent prospective observational studies demonstrated improved syncope management by use of a team of specially-trained personnel in a SMU.

The SEEDS study examined the utility of a SMU in the ED for patients with syncope who are considered at intermediate risk for adverse cardiovascular outcome.[9)] In this prospective, single-center, unblinded randomized study, 103 patients were randomized to 'standard care' or SMU care after initial assessment with a complete history, physical examination and ECG. The study found that in the ED, a presumptive diagnosis of the cause of syncope was significantly increased from 10% in the 'standard care' patients to 67% among those who underwent SMU evaluation; hospital admission was reduced from 98% among the 'standard care' patients to 43% among the SMU patients; the total length of patient-hospital days was reduced by >50% for patients in the SMU group.

The potential for the ESC Guidelines to facilitate management of syncope patients referred to ED's of 11 Italian general hospitals was investigated in the The Evaluation of Guidelines in Syncope Study (EGSYS-2).[14)] The application of guidelines to clinical circumstances was facilitated by use of purpose-designed software in addition to personnel

training at test sites. A definite diagnosis was established in 98% of cases, with the vast majority being either neurally-mediated or orthostatic faints. The initial evaluation (history, physical examination, and electrocardiogram) established a diagnosis in 50% of cases. The investigators further compared the outcomes of 745 patients managed with this aforementioned "standardized care" system to 929 patients managed with usual care. In the group designated to "standardized-care", hospitalizations were fewer, in-hospital stay was shorter, fewer tests were performed per patient, and cost per patient and mean cost per diagnosis were lower. An EGSYS score was derived from this study to predict cardiac syncope at initial ED evaluation. An abnormal ECG and/or heart disease, palpitations before syncope, syncope during effort or in supine position, absence of autonomic prodromes, and absence of predisposing and/or precipitating factors were found to be predictors of cardiac syncope. A score from +4 to −1 was assigned, and a score ≥3 identified cardiac syncope with 95% sensitivity and 67% specificity[15] (**Table 4**).

Conclusion

Syncope is a very common clinical problem that is often first evaluated by busy ED physicians and general practitioners. Delineating the underlying etiology and the risk of adverse outcome is often challenging. In the majority of the patients presenting with syncope a careful history, physical examination including orthostatic blood pressure measurements and ECG can often establish the diagnosis with substantial certainty. In other instances, when the initial evaluation is inconclusive, further evaluation is needed. In this setting, the studies should be carefully selected, and a 'shotgun' approach studiously avoided. The mere presence of an abnormal finding should not lead one to assume that the 'cause' has been found. In any event, whether the diagnosis is quickly established or not, the need for in-hospital care inevitably arises. In this regard, several potentially useful risk stratification criteria have been advocated with the goal of better guiding the decision-making process regarding immediate need for patient admission to hospital or safety of discharge from the ED with later clinic assessment. While none of these systems are perfect, they do offer reasonable strategies for identifying high-risk, intermediate-risk, and low-risk patients. High-risk patients need to be admitted to the hospital for further diagnosis and treatment. Intermediate-risk patients may warrent brief observation in ED-based SMU, but absent evolution of any 'high-risk' features, are best evalauted on an outpatient basis in syncope or falls clinic. More widespread development of such SMUs and syncope/falls clinics is strongly encouraged; together, they have been shown to reduce both hospital costs and number of undiagnosed cases. Finally, low-risk patients can be safely discharged from the ED with counselling, and later follow-up in an ambulatory care setting.

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Appendix: Medical History in Syncope Patients

Attention to each of the following key diagnostic components may prove helpful as part of the medical history taking in order to assure that a reported T-LOC event is true syncope. Thus, a careful history should focus on circumstances immediately before the attack, its onset, the attack, the end of the attack, and the patient's background.

Questions about the circumstances just prior to the attack

i. *Position*:
Prone or supine: Cardiac arrhythmia (tachy- or brady-arrhythmia)
Standing: Vasovagal syncope, Orthostatic hypotension
Immediately on standing: Orthostatic hypotension

ii. *Activity*
Exercise
Rest: Cardiac or cardiopulmonary causes (see **Table 2**)
Urination, defecation, cough, swallowing: Situational syncope (neurally-mediated syncope)

iii. *Predisposing factors*
Prolonged standing, hot or crowded places: Vasovagal syncope, orthostatic hypotension
Fear, intense pain, unpleasant sight or smell: Vasovagal syncope
Neck movements: Carotid sinus syndrome
New medication: Temporal relationship with start of medication leading to orthostatic hypotension

Questions about the onset of attack (e.g warning symptoms)

i. Nausea, vomiting, abdominal discomfort, feeling of cold or hot, sweating, blurred vision: Neurally-mediated syncope (e.g Vasovagal syncope)
ii. Aura: Seizure

Questions about the attack (eyewitnesses)

i. *Way of falling*: Loss of voluntary muscle control is an inherent part of loss of consciousness. Consequently, if standing, the fainter falls down; if seated he or she slumps over.
ii. *Rapid onset*: Generally, onset of syncope is rapid, being no more than 10 to 20 sec after onset of warning symptoms. However, many fainters either do not experience or are unaware of any premonitory symptoms. This lack of warning is particularly prevalent in older individuals.
iii. *Jerky movements*: In syncope, it is not uncommon for patients to exhibit jerky movements of the arms and legs for a brief period of time; non-expert bystanders may incorrectly interpret these movements as a "seizure" or a "fit". However, the jerky movements during a faint differ from those accompanying a grand mal epileptic seizure in several ways. They are of shorter duration, they tend to occur after the loss of consciousness has set in rather than before, and they do not have the "tonic–clonic" features of a true grand mal seizure.
iv. *Tongue biting*: Tongue biting, particularly if it is lateral has a high specificity for seizure disorder. Because of low sensitivity, absence of tongue bites has no diagnostic significance.[16] Midline tongue biting is usually seen in seizure disorders, but can also be seen in reflex-mediated syncope.
v. *Skin color*:
Pallor: Vasovagal syncope
Cyanosis: Cardiac or cardiopulmonary causes

Questions about the end of the attack

i. *A spontaneous, complete and prompt recovery* from the faint excludes a number of conditions which may cause T-LOC (e.g coma, intoxicated states, stroke etc). On the other hand, in

certain forms of syncope, particularly the vasovagal faint, recovery may be marked by fatigue with the need for sleep, and a general sense of diminished energy for some lengthy period of time (often hours in duration).

ii. *Confusion*: After an episode of syncope, patients may briefly appear disoriented or confused, but this resolves within moments and is often shorter than the post-ictal period associated with general seizures.

iii. *Urinary or fecal incontinence*: Seizure

Questions about the background

i. *Past medical history*: Previous cardiac disease, neurological history, metabolic disorders (e.g diabetes, etc)

ii. *Medications*: Many drugs prolong the QT interval and are associated with life-threatening dysrhythmias. Vasoactive drugs such as antihypertensives, vasodilators used for angina, and those used for erectile dysfunction can cause syncope. Drug related syncope is especially common in elderly patients taking multiple medications.[17)]

iii. *Family history*: A family history of premature sudden death should alert the physician to the possibility of serious congenital conduction abnormalities such as long-QT syndrome, preexcitation syndromes, Brugada syndrome, etc