Approach to Patients with Syncope in Emergeny Department - An Evidence-Based Review

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Abstract

Syncope is an important health problem, constituting 1%-5% of all emergency service admissions and up to 6% of all hospitalizations. Substantial experience with patient history and physical examination and time are required to diagnose syncope in patients presenting with transient loss of consciousness. In addition, only up to 50% of patients with syncope can be diagnosed with a final diagnosis, despite all efforts. Thus, management of syncope in emergency departments has shifted from reaching a final diagnosis and treatment to short-, moderate-, or long-term risk stratification systems, allowing decisions for outpatient management, including specialized branch care, or admission for further work-up. This review discusses the definition of syncope-related transient loss of consciousness, differential diagnosis of syncope, diagnostic methods and algorithms, and the main risk stratification studies. It also incorporates the recommendations of the American College of Emergency Physicians (ACEP) 2007 policy statement regarding patients with syncope. *(JAEM 2014; 13: 82-91)*

Key words: Emergency, syncope, transient loss of consciousness

Introduction

Syncope is a rapid-onset, short-duration, transient loss of consciousness (T-LOC) with spontaneous recovery due to global cerebral hypoperfusion (1). Although the final endpoint is global cerebral hypoperfusion and rapid loss of posture, syncope has a diverse pathophysiological and etiological spectrum with respect to triggers and mechanism of occurrence. Therefore, it is challenging to make a differential diagnosis, detect life-threatening conditions, and make important therapeutic decisions. Syncope constitutes 1%-5% of yearly emergency department visits and 6% of hospital admissions (2, 3).

This review aimed to discuss the differential diagnosis and risk stratification of syncope with a review of the relevant literature and make recommendations about making decisions on consultation, emergency intervention, hospital admission, or discharge in light of information obtained from the patient history and laboratory data in patients presenting to the emergency department with suspected loss of consciousness.

Syncope as a Symptom

Syncope is not a disease per se but rather a symptom that may arise during the course or as a result of a number of diseases. The self-limiting nature of syncope and full recovery after syncopal attacks indicate that syncope-related mortality and morbidity are not from the syncope itself but rather from the resulting trauma or the severity of underlying disease. Thus, the importance of syncope lies in being a warning sign or a cause of trauma rather than a true disease state.

It is of prognostic significance that emergency physicians urgently begin diagnostic tests and start to treat relatively rare life-threatening conditions (e.g., arrhythmias, aortic dissection, pulmonary embolism, acute coronary syndromes). Unfortunately, initial evaluation of patients in the emergency department is usually unrevealing. The most common and important limitations of emergency department evaluation are lack or inconsistent statements of witnesses regarding the syncopal event, post-syncopal fatigue or panic experienced by patients, leading to inability to give a reliable history, and limitation of history taking due to undulations in the consciousness level of patients traumatized by a syncopal episode.

Etiology of syncope: There are 3 main forms of syncope: reflex (neurocardiogenic), orthostatic, and cardiovascular. Reflex syncope is grouped as vasovagal, situational, carotid sinus syncope, and atypical forms. Syncope due to orthostatic syncope is due to primary or sec-

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ondary autonomic failure, drug effects, or volume loss (due to bleeding, dehydration, vomiting, or diarrhea). Cardiovascular syncope is generally secondary to cardiac causes and includes arrhythmic forms (bradycardia or tachycardia), structural cardiovascular disorders, or cardiorespiratory diseases (pulmonary embolism or pulmonary hypertension) (Table 1) (1).

In a considerable portion of patients, the syncope etiology can not be found. Such cases are said to have "unexplained syncope." Previous studies have shown that the etiology of syncope can be clarified in at best 20%-50% of patients, while 15%-30% of them remain undiagnosed despite extensive investigations (2, 4, 5). Thus, the decision of emergency physicians as to which patients require further diagnostic workup or monitorization is very important. The role of emergency physicians in the management of syncope has recently evolved from a diagnostic standpoint to a cooperative role with other departments in the form of risk stratification or decisions with respect to hospitalization (6).

In the Framingham Heart Study, 822 of a total of 7814 patients experienced syncope between 1971 and 1998. The rate of first reported syncope was 6.2 per 1000 patients. The syncope forms with identifiable etiology were neurocardiogenic syncope (21.2%), cardiogenic syncope (9.5%), and orthostatic syncope (9.4%). On the other hand, the largest group was unexplained syncope, which had a rate of 36.6%. According to this analysis, rate of death from any cause or cardiovascular reason was high. Patients with syncope of unknown origin were also at increased risk of mortality of any cause. In contrast, vasovagal syncope had a markedly benign course. Recurrence rates (overall 21.6%) have been found to be higher in patients with previous syncopal attacks or cardiac syncope (7).

Non-traumatic Transient Loss of Consciousness

Transient loss of consciousness (T-LOC) encompasses conditions with reversible, complete loss of consciousness, including syncope. As viewed from this aspect, all syncope cases fall into the T-LOC definition, but not all T-LOC cases can be included by the term syncope. T-LOC is a sine qua non for a diagnosis of syncope, and its absence should make one consider the possibility of cataplexy, falls, and psychiatric (psychogenic pseudosyncope) and neurologic (TIA of carotid origin) causes and move away from a syncope diagnosis. Therefore, it is imperative to obtain a thorough history from the patient or, if possible, from the witnesses of the episode to find out whether loss of consciousness was actually present. A transient, rapid-onset, short-duration loss of consciousness with full recovery makes the diagnosis of T-LOC (Figure 1).

Table 1. Syncope classification and major etiological factors

Reflex (neurally mediated) syncope
Vasovagal
- Emotional stress-induced: fear, pain, instrumentation, blood phobia, orthostatic stress-induced: prolonged standing or sitting
Situational
- Coughing, sneezing, gastrointestinal stimulation (swallowing, defecation, visceral pain), micturition (post-micturition), post-exercise, post-prandial, others (e.g., laughter, blowing into a brass instrument, weightlifting)
Carotid sinus syncope
Atypical forms (no specific trigger and/or atypical presentation)
Syncope due to orthostatic hypotension
Primary autonomic insufficiency
Pure autonomic insufficiency, multiple system atrophy, Parkinson+autonomic insufficiency, Lewy body dementia
Secondary autonomic insufficiency
- Diabetes mellitus, Amyloidosis, Uremia, Spinal cord injuries
Drug-induced orthostatic hypotension
- Alcohol, Vasodilators, Diuretics, Phenothiazines, Antidepressants
Volume loss
- Bleeding, diarrhea, vomiting
Cardiac Syncope (Cardiovascular)
Primary etiology is arrhythmia
Bradycardia
Sinus node dysfunction, atrioventricular conduction defect, dysfunction of implantable devices (PM, ICD, etc)
Tachycardia
- Supraventricular, ventricular
Structural Heart Disease
Valvular disease (native or prosthetic), myocardial disease (hypertrophic cardiomyopathy, restrictive myocardial disease, dilated cardiomyopathy, pericardial disease (tamponade, constrictive pericarditis) congenital coronary anomalies, cardiac mass, pulmonary embolism and hypertension, aortic dissection
(Modified from reference No. 1)

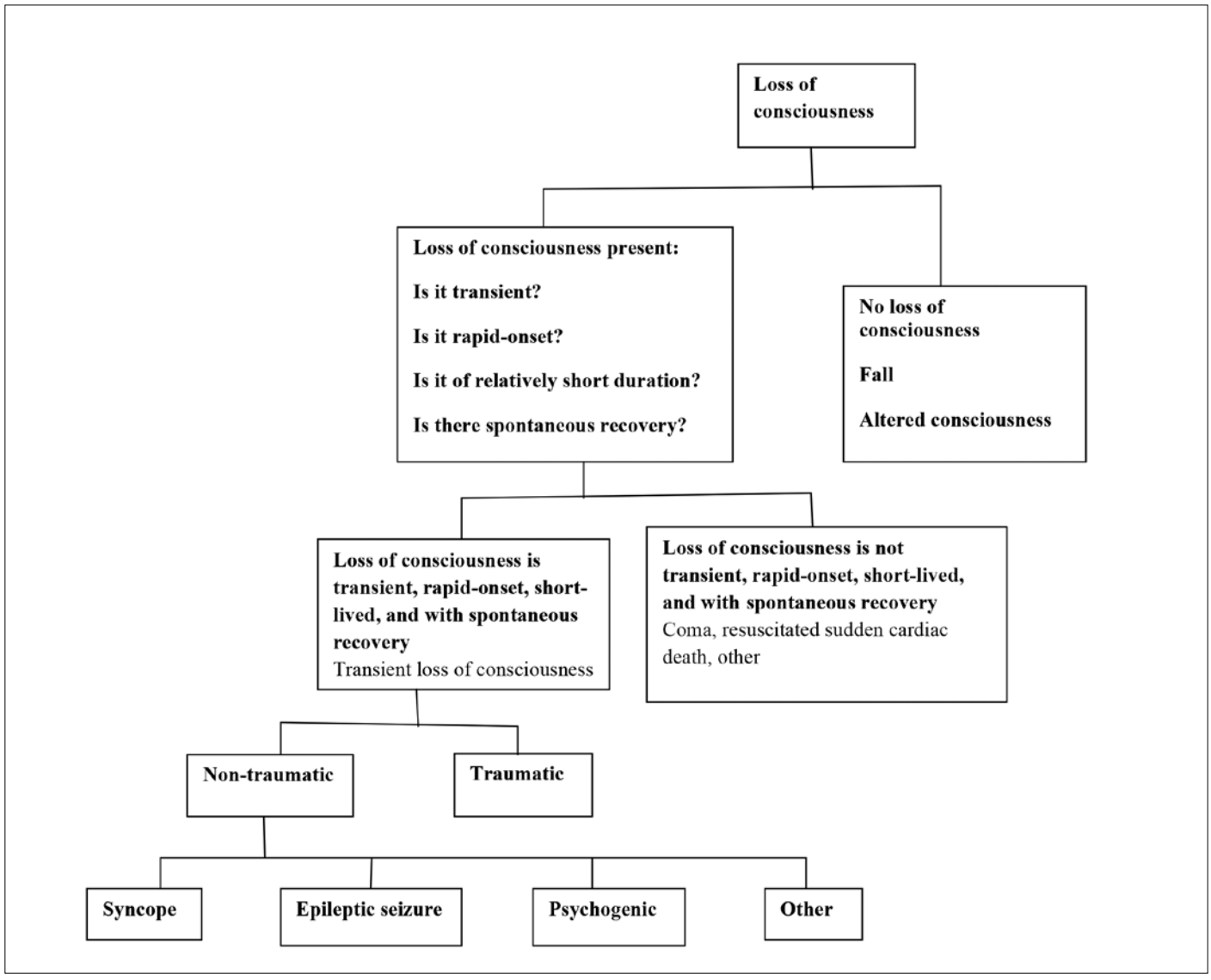

Figure 1. Algorithm for assessment of patients according to the presence and characteristics of loss of consciousness

Seizure vs. Syncope

Epileptic seizures are the most important conditions that require distinction from a syncopal episode. Emergency physicians often straddle making one of these diagnoses, leading to extended emergency department waiting times. Indeed, it is sometimes challenging to differentiate syncope from seizure activity. Some studies have attempted to devise various algorithms and scoring systems with the use of some historical variables as well as clinical clues for the differentiation of syncope and seizure. Crampton et al. reported that some elements in the patient history can be used in the differentiation of syncope from seizure. These include presence of previous presyncope or syncope attacks; prolonged sitting or standing as the initiating mechanism; rapid assumption of upright posture; dehydration; prodromal symptoms, such as nausea, palpitation, dyspnea, warmth sensation, dizziness, blurred vision, and sounds coming from a distance; paleness and sudden collapse during attacks; and recalling the onset of loss of consciousness during recovery (Table 2) (8).

Sheldon et al. (9) developed a scoring system for syncope versus seizure distinction based on elements of the patient history (Table 3). They assigned certain point scores to various historical variables. According to this scoring system, a total point score ≥1 was in favor of seizure, and <1 was in favor of syncope. The authors reported that the sensitivity and specificity of the scoring system were both 94%. In a review by McKeon et al. (10), it was reported that some features of the patient history may be beneficial for a distinction between syncope and seizure. According to this, dizziness, sweating, prolonged standing, certain precipitants and chest pain, palpitation, low heart rate, and low blood pressure point to a syncope episode, whereas deja vu and jamais vu, aphasia, olfactory aura, tongue biting, post-event delirium, and focal neurological signs are indicative of a seizure attack. Witness accounts pointing to a pale, sweating subject with low pulse and blood pressure indicate a syncopal origin, while presence of aphasia, delirium, head turning to the side, automatism, convulsion, and postictal delirium are more consistent with seizure activity.

Perhaps convulsive syncope is the condition that is most commonly confused with a seizure attack. This syncope variety is characterized by convulsions or tonic-clonic movements similar to those in epileptic seizure. A useful diagnostic clue in such patients is that

Table 2. Features of patient history in differentiation of syncope from seizure activity

	In favor of syncope	In favor of seizure activity
Past history	Previous presyncope*/syncope	Previous seizure attacks, cortical abnormalities
	Prolonged standing or sitting*, sudden assumption of erect posture, dehydration, vertigo, sweating, situational triggers (micturition, defecation, etc) chest pain, palpitation, low heart rate, low blood pressure	Stress*, sleep deprivation, drug withdrawal (alcohol, benzodiazepines), photic triggers, deja vu or jamais vu, aphasia, olfactory aura, epigastric sensation, tongue biting, post-event delirium, focal neurological signs
Prodrome	nausea*, dyspnea*, palpitation*, warmth sensation*, dizziness, blurred vision	With partial onset symptoms, points to a temporal, frontal, parietal or occipital focus
Attack	Pallor, collapse, low pulse or blood pressure	Tongue biting*, head deviation*, bizarre posture*, urinary incontinence*, cyanosis*, automatism, aphasia, delirium, convulsion
Recovery	Loss of consciousness recalled*	Confusion*, postictal delirium, headache*, event not recalled*

*Previously determined discriminators of syncope vs seizure attack
(Modified from reference No. 8)

Table 3. Scoring for syncope vs seizure differentiation

	Point
Waking up with tongue biting?	2
Deja vu or Jamais vu?	1
Emotional stress	1
Head turning	1
Unresponsive, uncommon posture, extremity movements, or amnesia? (any)	1
Post-event confusion	1
Dizziness attacks	-2
Pre-event sweating	-2
Loss of consciousness with prolonged standing or sitting	-2

≥1 point indicates high seizure likelihood, <1 point indicates high syncope likelihood
(Modified from reference No. 9)

myoclonus or convulsion occurs after pallor, sweating, and collapse, whereas this order is reversed in seizure attacks.

A patient history notable for structural heart disease (coronary artery disease, heart failure, left ventricular hypertrophy), diabetes, and presence of multiple risk factors for coronary artery disease may specifically direct clinicians to cardiac syncope.

Investigation of Syncope Etiology in the Emergency Department

The etiology of syncope may not readily be found in the emergency department. Solid evidence may be apparent in the patient history at the initial stage of the patient evaluation in some patients, while the diagnosis is only possible after an extensive workup, including complete blood count, biochemistry, ECG, cardiac biomarkers, chest x-ray, and thoracic tomography in others.

Initial Assessment

After determining that the transient loss of consciousness is due to a syncopal attack, an initial assessment should be rapidly done. This assessment includes evaluation of vital signs as well as taking a patient history and performing a thorough physical examination and ECG. The initial evaluation should specifically aim to reveal resting or orthostatic tachycardia or orthostatic hypotension. Points not to miss in the physical examination include evaluation of pulse characteristics (e.g., filiform pulse, pulsus paradoxus, pulsus alternans, pulsus bigeminus, pulsus bisferiens, pulsus parvus et tardus, or pulse deficit), neck veins, place of maximal apical impulse, additional heart sounds (S3-S4), certain auscultatory findings of aortic stenosis, hypertrophic cardiomyopathy, left atrial myxoma, or pericardial effusion/tamponade.

The diagnostic findings in the initial examination are listed in Table 4 (1). Vasovagal, situational, and orthostatic syncope; arrhythmic syncope; and syncope related to cardiac ischemia or some other cardiovascular disorders can be diagnosed with diagnostic clues obtained from history taking, physical examination, and ECG. Vasovagal syncope is the most common type after unexplained syncope. Some components of the patient history may lead to vasovagal syncope in the emergency department. A relatively young patient age, lack of previous history of cardiovascular diseases, rare syncopal episodes with relatively long inter-episode intervals, and the index episode being the very first syncope episode are all indicative of a benign etiology. In addition, the presence of typical stressors (prolonged standing or sitting in the unchanged position; waiting without changing posture in crowded, moist, poorly ventilated, hot environments; exposure to unwanted sounds, images, smells, or blood), and having warmth sensation, rapid pulse, nausea, and dizziness prior to syncopal episode are supportive of a vasovagal origin. Situational syncope is characterized by very typical, recurrent syncopal attacks always elicited by the same activity (vagally mediated maneuvers, such as blowing a musical instrument, defecation, micturition, sneezing, or swallowing); orthostatic syncope is related to advanced patient age, diabetes, neurological, renal, or hepatic diseases; disorders characterized by autonomic dysfunction; use of drugs, including diuretics, alpha blockers, dihydropyridine derivatives, or first doses of ACE inhibitors or ARBs; and reduced circulatory volume associated with diarrhea, blood loss, vomiting, excessive sweating, dehydration, or fluid loss to the third space. Orthostatic hypotension is diagnosed by a reduction of systolic blood pressure by 20 mm Hg and a reduction of diastolic blood pressure by 10 mm Hg 2 minutes after active standing.

Presence of arrhythmia (absence of a prodrome, syncope during exercise or supine position, a low ejection fraction or presence of clinical heart failure, or family history of sudden cardiac death or syncope) or typical features of ischemic episodes (typical angina pectoris or angina equivalents) along with typical ischemic ECG changes secures arrhythmic or ischemic syncope. On the other hand, angiotensin-converting enzyme I/D polymorphism episode associated

Table 4. Diagnostic findings and related diagnoses at first assessment

	Diagnosis
Syncope triggered by emotional orthostatic stress and associated with typical prodrome	vasovagal syncope
Syncope occurs during or immediately after specific triggers such as micturition, defecation, and swallowing	Situational syncope
Syncope takes place with standing up and orthostatic hypotension is documented	Orthostatic syncope
Any of the following:	Arrhythmic syncope
- Persistent sinus bradycardia < 40 bpm, sinusal pause ≥3 seconds, or recurrent sinoatrial block	
- Mobitz Type 2 or 3. degree AV block	
- Alternating right and left bundle branch block	
- VT or fast SVT	
- Short or long QT interval with episodes of polymorphic VT	
- PM/ICD dysfunction with cardiac pause	
Syncope with typical signs and symptoms of cardiac ischemia and ECG findings of acute ischemia with or without enzyme positivity	Cardiac ischemia-associated syncope
Syncope occurs with atrial myxoma, severe aortic stenosis, pulmonary hypertension, pulmonary embolism, and aortic dissection	Cardiovascular syncope
(Modified from reference No. 1)	

Table 5. ACEP Risk stratification

Class A	Defines patient management principles that are generally accepted and show highly clinical precision
Class B	Defines patient management principles with moderate clinical precision
Class C	Defines evidence that is precursory, imprecise, or controversial
(Modified from reference No. 6)	

with emotional or orthostatic stress preceded by a typical prodrome makes the diagnosis of vasovagal syncope.

Tests directed at the suspected etiology should be rapidly performed in cases having no clear diagnosis after the initial evaluation but that are suspected to have a specific etiology, including acute coronary syndromes, pulmonary thromboembolism, aortic dissection, pulmonary hypertension, left atrial myxoma, pericardial tamponade, hypertrophic cardiomyopathy, or aortic stenosis. These conditions may not show typical findings in the ECG, history, and physical examination in the initial evaluation. These possible diagnoses should be confirmed with use of cardiac biomarkers (BNP, troponins, CK-MB), chest x-ray, serial ECGs, echocardiography, thoracic CT, ventilation-perfusion scintigraphy, and coronary angiography whenever suspected (Figure 2).

Risk Stratification in Unexplained Syncope

Only some (20%-50%) patients presenting to the emergency department with syncope can be diagnosed with a specific etiology after the initial evaluation (2, 4, 5). Despite an extensive workup, approximately 30% of cases with syncope remain unexplained. The current role of emergency physicians in the evaluation of syncope has shifted from the point of making an exact diagnosis to the point of risk stratification (6).

The following are three critical questions an emergency physician should ask (6):

1. Which elements of the history and physical examination help in risk stratification?
2. Which diagnostic tests are helpful in risk stratification?
3. Which patients with unexplained syncope should be hospitalized?

History and physical examination play an important role in risk stratification of a patient with syncope. Risk stratification based on patient history and physical examination, as suggested by the 2007 ACEP consensus guidelines for syncope, is summarized in Table 5.

In the guideline mentioned above, a Class A recommendation was made for a search of typical signs and symptoms of heart failure with the history and physical examination to determine patients at higher risk of adverse events; a Class B recommendation was made for giving consideration to advanced age, structural heart disease, or coronary heart disease with respect to future risk of adverse events, and a Class B recommendation was also made for determining young persons who do not have exercise-induced syncope, any history of cardiovascular disease, or any family history of sudden cardiac death.

The primary objective of risk stratification in the emergency department is to predict short-term (days to weeks) and long-term (months to years) risk and to prevent adverse events, with death being in the first place, in high-risk patients. In the San Francisco syncope rule (3), which was originally devised to pick up low-risk patients, 5 parameters were identified that can be remembered as the CHESS mnemonic. According to this syncope rule, the presence of any of the following criteria puts a patient into the high-risk category: a history of heart failure, hematocrit <30, abnormal ECG, dyspnea, and triage blood pressure less than 90 mm Hg. The score had a sensitivity of 96% (95% CI 92%-100%) and a specificity of 62% (95% CI 58%-66%). A validation study of the scoring system, dated 2006, revealed a sensitivity of 98% (95% CI 89% to 100%) and a specificity of 56% (95% CI 52% to 60%), confirming that the scoring system functioned well (11).

However, an external validation of that scoring system, which was performed by Birnbaum et al. (12) in 2008, did worse with a sen-

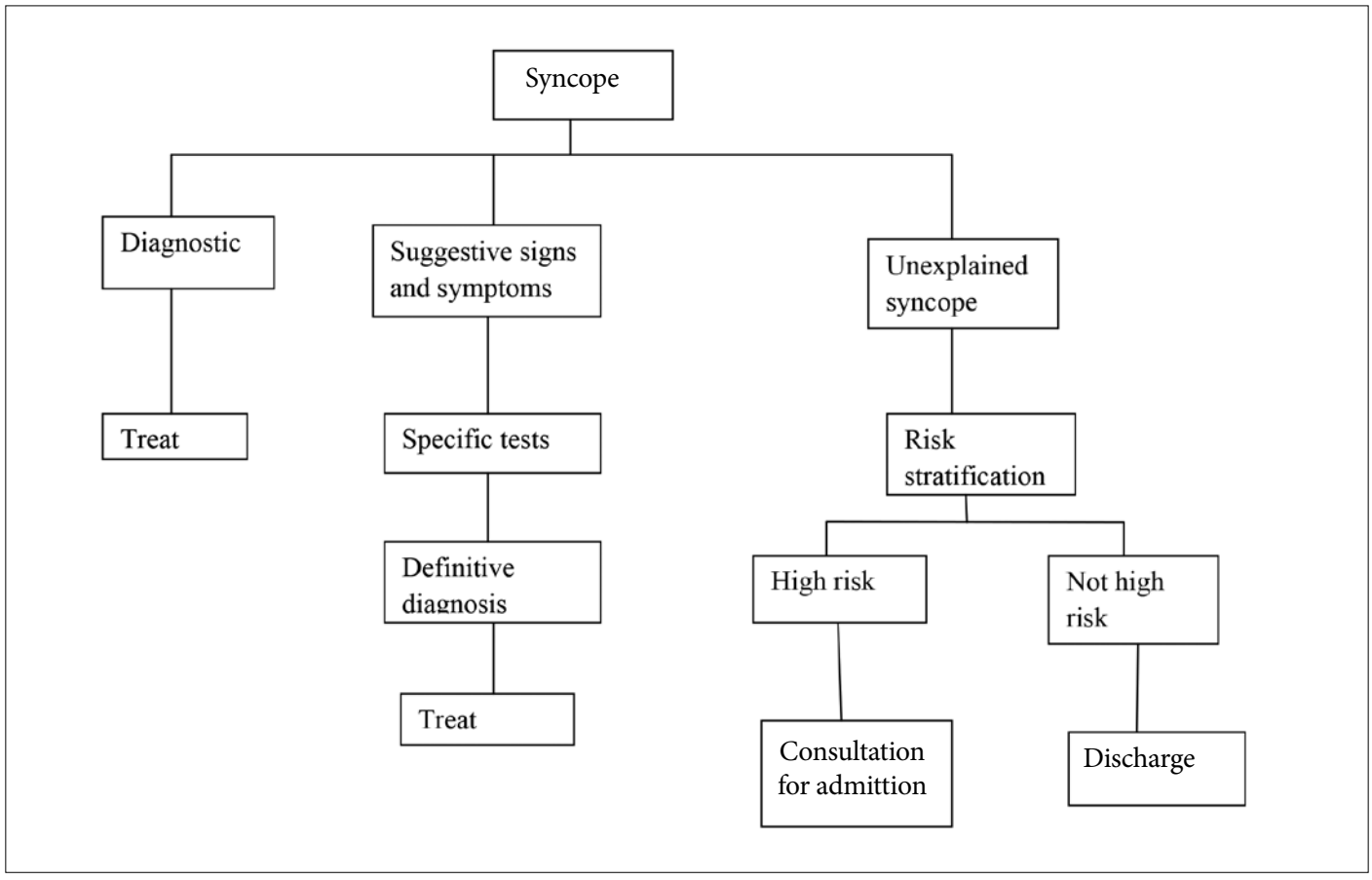

Figure 2. An algorithm for diagnostic assessment of syncope in emergency department

sitivity of 74% (95% CI 61% to 84%), specificity of 57% (95% CI 53% to 61%), negative likelihood ratio of 0.5 (95% CI 0.3 to 0.7), and a positive likelihood ratio of 1.7 (95% CI 1.4 to 2.0). However, a correspondence (13) written by the authors of the original study for criticism of the study by Birnbaum et al. stressed that a lower rate of syncope presentation (0.5% versus 1.2% to 1.5%) and a higher admission rate (86% versus 55%) in the study by Birnbaum et al. compared to previous studies were indicative of a selection bias. They also added that the authors misapplied the criteria for abnormal ECG and that the study ECGs were not reviewed by the primary physicians but rather by a separate physician in a retrospective manner, leading to possibly variable and inconsistent ECG interpretation. In reply to this criticism, Birnbaum et al. (14) replied that 88% of ECGs were reviewed by an emergency physician, and senior physicians reviewed ECGs for the purpose of standardization. They put forward geographic and ethnic differences of the study region for their study's different syncope and admission rates from the previous literature. In 2011, Saccilotto RT et al. published a meta-analysis of 12 studies containing 5316 patients. They reported a combined sensitivity of 87% (95% CI 79% to 93%) and a combined specificity of 52% (95% CI 43% to 62%) for the San Francisco syncope rule (15). Gabayan et al. (16) made a retrospective analysis of short-term (7-day) adverse cardiac events (cardiac death or hospitalization) in 39,943 admissions for syncope in 35,330 patients and reported that 3% of patients experienced adverse events, the positive predictors of which were age ≥60 years, male gender, congestive heart failure, ischemic heart disease, cardiac arrhythmias, and valvular diseases. In contrast, the following criteria were negative predictors for adverse events: history of a previous pacemaker or implantable cardioverter-defibrillator (ICD) implantation or a previous revascularization, both protecting against lethal arrhythmias or coronary ischemia; history of dementia, making the patient history unreliable for distinguishing syncope from fatigue, seizure, or drop attacks; or history of cerebrovascular disease, making a neurological etiology more likely than a cardiological etiology. In the latter study, it was stressed that the cardiac event rate was higher during the first 7 days and especially within the first 3 days. The authors, therefore, suggested that patients who are discharged from the emergency department after the initial tests fail to point to a specific etiology should be followed on an outpatient basis for the first 7 days. However, the patients analyzed in that study were younger than similar studies (mean age 60.1 years) and had a better health status, leading to lower event rates as compared to other trials (2.5% vs 8%-11%). Moreover, prediction of short-term events was negatively affected by reviewing comorbidity data from available records and a lack of assessment of ancillary instruments, such as symptoms or physical examination findings.

Another short-term risk stratification tool, the Boston Syncope Rule, introduced 8 criteria about the signs and symptoms for prediction of 30-day risk of adverse events or death (17). These include signs and symptoms of acute coronary syndromes, signs of conduction system disease, a positive cardiac history, valvular disease as determined with a history or physical examination, sudden death in the family, persistent abnormal signs in the emergency department, volume depletion (persistent dehydration, gastrointestinal bleeding, or

Hct <30), and a central nervous system event. The Boston rule could predict syncope with a sensitivity of 97%, a specificity of 62%, and an NPV of 99%. Implementation of the rule reduced the rate of hospitalization by 48% in the original study.

A recent study from our country compared the value of several syncope rules, including EGSYS, OESIL, and the San Francisco syncope rule, in predicting short-term serious events and additionally developed a new syncope rule, called the "Anatolian Syncope Rule" (18). In a total of 231 patients, dyspnea, orthostatic hypotension, a precipitating factor causing syncope, age >58 years, congestive heart failure, and ECG abnormalities (defined by the acronym DO-PACE) were predictive of short-term adverse events in the univariate analysis. According to that study, the newly developed "Anatolian Syncope Rule" had a sensitivity of 100% (0.66-1.0) for mortality compared to the OESIL score having a sensitivity of 90% (0.54-0.99), EGSYS score having a sensitivity of 80% (0.44-0.97), and San Francisco syncope rule having a sensitivity of 100% (0.66-1.0). The rules had a specificity of 78% (0.72-0.83), 76% (0.70-0.82), 80% (0.74-0.85), and 70% (0.63-0.76), respectively. The scoring systems had sensitivities of 97% (0.85-1.00), 70% (0.52- 0.82), 56% (0.40- 0.72), and 87% (0.72 - 0.95), respectively. Their specificities for any adverse event were 72% (0.64- 0.78), 82% (0.76 - 0.87), 84% (0.78 - 0.89), and 78% (0.71 - 0.83), respectively. The authors stated that the newly developed scoring system was considerably sensitive in predicting short-term serious events and was non-inferior to the previous scoring systems. Nevertheless, as the authors admitted, their study was a single-center study, and its results may not have reflected the entire Turkish patient population. The real-world performance of that scoring system will be clarified by prospective validation studies.

The ROSE (Risk stratification Of Syncope in the Emergency department) (19) risk stratification score includes risk predictors designated with the mnemonic BRACES. B stands for BNP ≥300 pg/mL, or bradycardia <50 bpm), R stands for occult blood in stool (in rectal examination/laboratory tests), A stands for anemia (Hb<9 mg/dL), C stands for chest pain preceding or concurrent with syncope, E stands for Q wave on the ECG (except for D3), and S stands for saturation <94% (in room air). Presence of at least one of these criteria puts patients at increased risk for serious adverse events (acute myocardial infarction, life-threatening arrhythmias, need for implantation of a pacemaker or implantable cardioverter-defibrillator, pulmonary embolism, stroke, and bleeding or surgical intervention) at 1 month. The sensitivity, specificity, and NPV of the rule were 87%, 65.5%, and 98%, respectively. Of them, the criterion that was most commonly associated with cardiac events was a BNP level greater than 300 pg/ml. EGSYS (20), OESIL (1), and the risk scoring system developed by Martin et al. (22) have been used for long-term risk stratification. While OESIL and the scoring system of Martin et al. predict rates of serious events at 1 year, the EGSYS score predicts 2-year risk. The EGSYS score has been reported to possess a two-aspect predictive value. One of them is prediction of long-term mortality, and the other is prediction of cardiac syncope. According to this scoring system, palpitation preceding syncope is assigned 4 points, heart disease and/or abnormal ECG is assigned 3 points, syncope during exercise is assigned 3 points, syncope in supine position is assigned 2 points, presence of a precipitating or predisposing factor is assigned -1 point, and presence of a prodrome with nausea and vomiting before the episode is assigned -1 point. A score ≥3 was reported to predict cardiac syncope with a sensitivity of 92% and a specificity of 69%, and patients with point(s) were reported to portend higher mortality in both the original and validation studies.

The OESIL risk score examined the role of the following criteria in the prediction of serious events at 1 year: abnormal ECG, history of cardiovascular disease, age >65 years, and lack of prodrome. The original study determined risk factors for mortality at 1 year in 270 patients and validated the results in another 328 patients (13). Each of the criteria (abnormal ECG, history of cardiovascular disease, age > 65 years, and lack of prodrome) was assigned 1 point. The mortality rates for 0, 1, and 2 points were 0%, 0.6%, and 14%, respectively. Patients with a score of 0-1 were accepted as low-risk, and those with a score equal to or greater than 2 were considered high-risk. There have been some inconsistencies between the original OESIL risk study and the subsequent validation studies. Martin et al. (14) attempted to devise a risk classification system for arrhythmias and mortality at 1 year in 252 patients. They also validated the system in a 374-patient cohort. The risk factors were ECG abnormalities, history of ventricular arrhythmias, history of congestive heart failure, and age > 45 years. The validation study reported a mortality rate of 0% at 1 year for patients with no risk factors and 27% for 3 or 4 risk factors. Advanced age, abnormal ECG, lack of prodrome, and a positive history of cardiovascular disease, especially ventricular arrhythmias being in the first place, appear to have predictive power for 1-year risk in both the OESIL risk score and the risk score of Martin et al. However, long-term risk stratification has a limited role in emergency departments, where short-term risk has a greater role in patient management. Long-term risk stratification is rather used by other departments, particularly cardiology.

A meta-analysis published in 2011 (23) analyzed studies investigating patients presenting to emergency departments between 1990 and 2010. The mean age of a total of 43,314 study participants studied by 11 trials was 63 years (range 60-64 years). The hospital admission rate was 44%. The mortality rate was low, albeit significantly increased (4.4%; 95% CI: 3.1-5.1). One-third of deaths were attributed to cardiovascular causes. One-third of the patients were undiagnosed; in the group where a diagnosis could be reached, the most common diagnoses were vasovagal, orthostatic, and situational syncope, which altogether were responsible for 29% of all episodes (95% CI: 12-47). The syncope etiology was a cardiac condition in 10.4% of cases (95% CI: 7.8-16), with bradyarrhythmias (4.8%; 95% CI: 2.2-6.4) and tachyarrhythmias (2.6%; 95% CI: 1.1-3.1) being the most common etiologic factors. The most powerful predictors of adverse cardiac events were palpitation before syncope, exercise-induced syncope, history of heart failure or ischemic heart disease, and active bleeding or a history of bleeding. A review of predictors of adverse events (death or hospital admission) separately for each of these 11 studies revealed that all studies reported heart failure, ischemic heart disease, and abnormal ECG; 6 studies reported age; 3 studies reported abnormal blood pressure (<90 mm Hg or >160 mm Hg); 3 studies reported male sex; and 2 studies reported signs of bleeding.

A systematic review and meta-analysis of the methodological quality and prognostic accuracy of clinical decision rules examined a total of 18 studies including 10,994 patients aged 50-79 years, of whom 55% were women. Five of these studies were derivation studies, 9 were validation studies, and 4 were derivation/validation studies. A total of 13 studies were 30-day studies, 1 was a 6-month study, and 2 were 1-year studies. One study did not specify any follow-up duration. Three to forty-one percent of the patients were hospitalized, and 13% had adverse events, a quarter of which could be di-

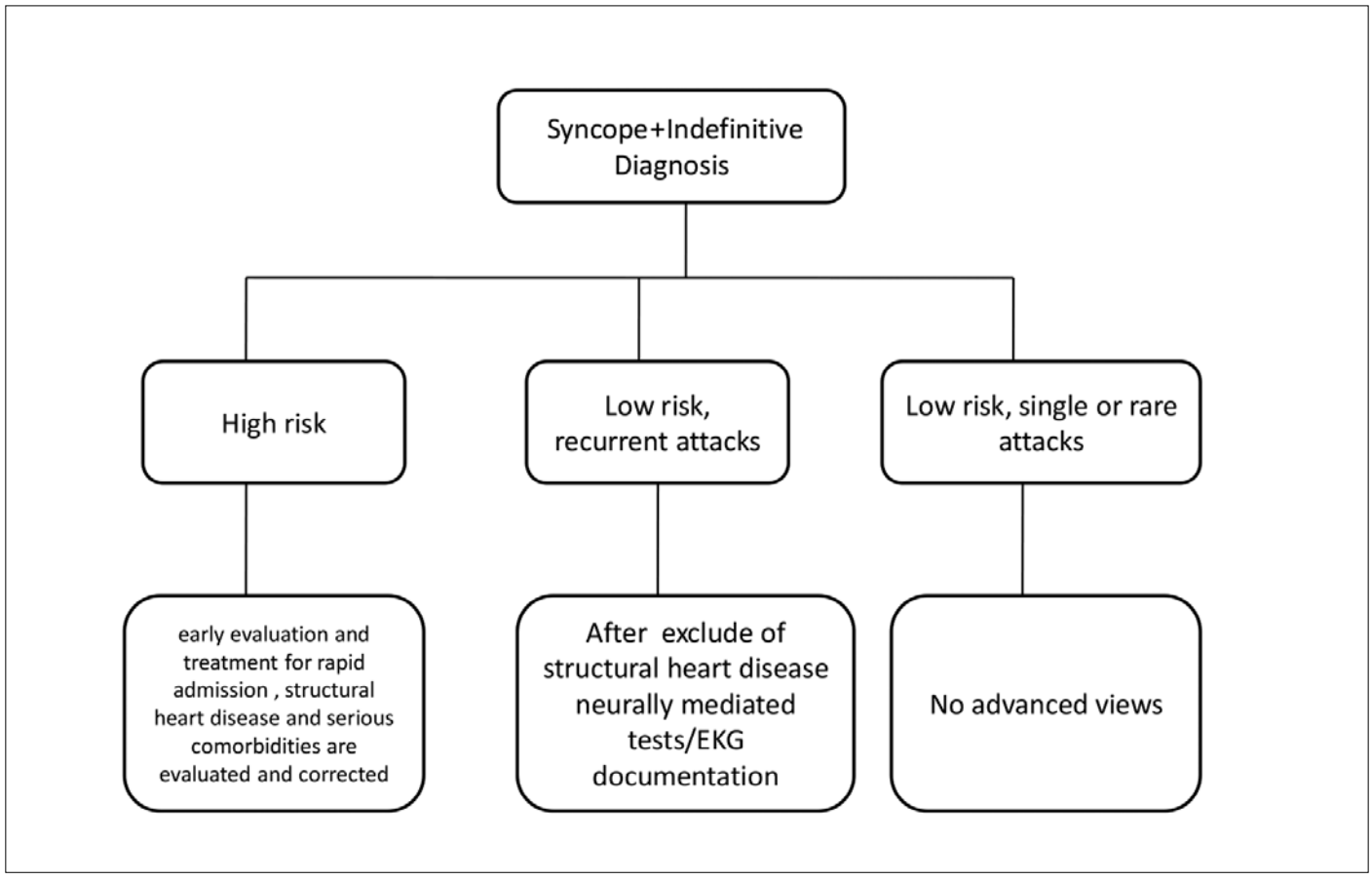

Figure 3. In case of undefined syncope patient management according risk classification

agnosed in the emergency department. That review suggested that the San Francisco syncope rule and OESIL risk score were validated in more than one setting (level 2 evidence) and therefore may be considered to be implemented in clinical practice (24).

Risk stratification before investigating the syncope etiology will miss serious diagnoses, such as an acute coronary syndrome, dysrhythmia, ectopic pregnancy, or subarachnoid bleeding. For example, a middle-aged man with chest pain and subsequent syncope will be labeled as low-risk unless an abnormality is detected in his vital signs and admission ECG. Therefore, one should search for identifiable causes of syncope first, reserving risk stratification to be performed for unexplained syncope after the initial evaluation. Another important point for clinicians to remember is that none of the available scoring systems for risk stratification is ideal. Therefore, a risk score should never preclude a clinician's common sense, no matter how comprehensive it is (25).

Position of ACEP and ESC Guidelines for Risk Stratification

There are several guidelines for risk stratification and management of patients presenting with syncope. Among these, the American College of Emergency Physicians (ACEP) guideline, dated 2007, and the European Society of Cardiology guideline, dated 2009, will be discussed here. Although the ESC guideline is not primarily intended for use by emergency physicians, it conveys important information related to the initial evaluation and urgent management of syncope.

The ACEP guideline (6) published in 2007 recommends use of signs and symptoms of heart failure as assessed with past history and physical examination to determine patients at higher risk (Level A recommendation). Level B recommendations consider structural heart disease or coronary artery disease as risk factors for adverse events. Level B recommendations also suggest considering young patients with no cardiovascular disease or comorbidities as low-risk. The same guideline mentioned that 12-lead ECG is helpful in risk stratification and recommended performing advanced tests, such as echocardiography or cranial CT, according to specific findings or results of the physical examination and ordering laboratory tests in the appropriate clinical context. It was specified that cardiac electrical monitorization may be useful in patients with strongly suspected arrhythmic syncope. Electrical monitorization beyond 24 hours does not add much to 24-hour monitoring. Despite referring to a report suggesting that rhythm monitoring for up to 72 hours might be beneficial in case of age >65 years, male gender, history of heart disease, or a non-sinus rhythm in the first ECG, the guideline states that patients in whom an arrhythmia is detected in the second and third 24-hour period remain asymptomatic (26).

The ESC guideline for management of syncope (1) that was published in 2009 recommends that an initial evaluation be made for risk stratification in patients with transient loss of consciousness suspected to have syncope. It also recommends rapid treatment of patients in whom a clear diagnosis can be made. As also admitted by the guideline, however, a definitive diagnosis can not be made in a considerable portion of cases, and risk stratification should inevitably be made. The ESC guideline recommends classifying patients as high- or low-risk and admitting and rapidly assessing high-risk pa-

Table 6. Quick hospitalization and requiring extensive research, which increases the risk of short-term high-risk criteria

Severe structural disease or coronary artery disease
Heart failure
Low EF
Prior MI
Clinic suggesting arrhythmic syncope or EKG characteristics
Syncope in the supine position/Exercise
During syncope, palpitations
Family history of sudden cardiac death
Discontinuous VT
Bifascicular block (LBBB, RBBB+LAHB/LPHB) or QRS>120 ms along with other interventricular conduction abnormalities
Without negative chronotropic medication or history of long-term physical exercise, inefficient sinus bradycardia or sinoatrial block
Preeksit of the QRS complex
Short or long QT interval
ST elevation of V1-V3 with RBBB (Brugada patern)
Negative T waves in right precordial leads, epsilon waves and ventricular late potentials (AVRD finding suggesting)
Serious co-morbidities
Serious anemia
Electrolytes abnormalities

EF: ejection fraction, MI: myocard infarction, LBBB: left bundle branch block, RBBB: right bundle branch block, LAHB: left anterior hemifascicular block, LPHB: left posterior hemifascicular block, ARVD: arrhythmogenic right ventricular displazisis

tients with at least one high-risk criterion that increases short-term risk and requires rapid admission and extensive workup, including echocardiography, stress tests, angiography, electrophysiologic procedures, and tests for neurally mediated syncope. Further evaluation of low-risk cases varies by whether there is one or recurrent syncopal episodes (Figure 3). Conditions specified by the ESC guideline that put patients into the high-risk category are listed in Table 6.

Decisions about Treatment and Hospitalization

Treatment of syncope patients with a definitive diagnosis includes treatment of underlying disorder(s) and trauma-associated injuries. In this context, a specific therapy in the emergency department or hospital admission is not needed in vasovagal syncope or situational syncope where the triggering event is no longer present and the patient has already fully recovered. On the other hand, fluids or blood products should be administered as needed in orthostatic syncope, and the offending drug should be stopped or a specific antidote be administered in drug-induced syncope. Specific treatment options are available in emergency departments when arrhythmic syncope is suspected. In bradyarrhythmic syncope, atropine or temporary transcutaneous or transvenous pacemaker therapy may permanently or transiently abolish the underlying cause. Syncope of tachyarrhythmic origin is managed by treating supraventricular or ventricular arrhythmias by antiarrhythmic drugs (class 1, 2, 3, and 4) or, when the patient is unstable or unresponsive to medications, cardioversion or defibrillation. Lastly, conditions, such as pulmonary embolism, pulmonary hypertension, aortic dissection, or acute cardiac ischemia, should be treated in collaboration with the relevant departments.

In patients with no clear diagnosis who could not be given specific or nonspecific therapy, the decision should be made about the setting (outpatient/inpatient) in which the tests and therapeutic interventions will be delivered. The main aim of the risk scores mentioned above is to pick up patients to be admitted to the hospital versus those to be followed on an outpatient basis. Patients labeled by the risk scores as high-risk should be hospitalized. However, there are some other high-risk criteria not found in available risk scores; patients with such criteria should also be admitted to the hospital. In addition, patients considered high-risk by a physician should be admitted to the hospital independently of the results of the risk scoring.

The ACEP guideline recommends hospitalization of patients with heart failure or structural heart disease as well as patients with high-risk features as detected by risk stratification schemes. High-risk patients typically have at least 1 of the following:

1. Advanced age
2. Abnormal ECG
3. Hct <30%
4. History of heart failure, coronary artery disease, or structural heart disease.

Conclusion

Syncope constitutes a considerable part of emergency service admissions. It is associated with medicolegal and ethical difficulties. Diagnosing transient loss of consciousness and distinguishing it from central nervous system events, especially from seizure, are the first steps towards a correct diagnosis. Unfortunately, the majority of patients continues to have unexplained syncope after completing the initial evaluation composed of a history, physical examination, and ECG. Emergency physicians should perform risk stratification before sending such patients home. Despite being far from ideal, available risk stratification tools now provide emergency physicians with tan-

gible risk stratification tools that were not present until the last decade. Nevertheless, no risk stratification tool can replace physician judgment and common sense. Physicians can avoid adverse events with good clinical practice.

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