CLINICAL PRACTICE

Syncope

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This Journal *feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.*

SUMMARY

Syncope is a common clinical problem caused by transient cerebral hypoperfusion and encompasses reflex, orthostatic, and cardiac mechanisms. Accurate diagnosis relies on careful history taking and physical examination, as well as targeted testing guided by risk stratification. Management of syncope is determined on the basis of the underlying mechanism and focuses on reducing recurrence and injury risk. In older patients, syncope may manifest as unexplained falls. Early identification of high-risk features is critical to prevent adverse cardiovascular outcomes.

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N Engl J Med 2026;395:582-91.
DOI: 10.1056/NEJMcp2517255
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CME

A 75-year-old retired man who lives alone presents to the emergency department with a zygomatic fracture after an unexplained fall. He has a history of hypertension, hyperlipidemia, and depression, for which he is receiving treatment. The incident occurred in the early afternoon while he was walking his dog. It was associated with urinary incontinence, and the patient has no recollection of the event. There were no witnesses. Over the preceding 6 months, he reports having had one similar fall and two episodes of transient loss of consciousness. These episodes occurred after he stood up after eating a meal or sitting for a prolonged period or during nocturnal urination. All events were unwitnessed. How should this case be managed?

THE CLINICAL PROBLEM

Syncope, or a fainting episode, is defined as a transient loss of consciousness accompanied by an inability to maintain postural tone, resulting from temporary cerebral hypoperfusion, which is followed by rapid and spontaneous recovery. Episodes are typically brief, usually lasting less than 1 minute.[1] Causes of syncope are classified as reflex syncope, cardiac syncope, and syncope due to orthostatic hypotension.

Syncope and falls frequently overlap, particularly in older adults, making the distinction between the two events clinically challenging.[2] The incidence of unexplained falls increases with age, rising from approximately 4% per year among adults 50 to 60 years of age to approximately 8% per year among adults 70 years of age or older, as compared an incidence of syncope of 4.4% per year across similar age groups. Approximately one in five events results in an injury leading to hospital evaluation.[2] Falls account for approximately 15% of emergency department visits among older adults,[3] and 20 to 30% of such falls may be unexplained, potentially reflecting underlying syncope.[3]

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Syncope is common, affecting one third to one half of the general population during their lifetime. Syncope is reported more frequently in women than in men, with prevalence estimates of 22% and 15%, respectively.[4] The cumulative incidence increases markedly with age, ranging from approximately 5% among women 20 to 29 years of age to nearly 50% among those 80 years of age or older, with the lifetime cumulative incidence among women being nearly twice that observed among men.[2,5] Recurrent syncope occurs in up to 20% of affected persons and is associated with greater risks of death (hazard ratio, 1.87; 95% confidence interval [CI], 1.26 to 2.77) and major adverse cardiovascular events (hazard ratio, 2.69; 95% CI, 2.02 to 3.59) than nonrecurrent syncope over a follow-up period of 24 months.[6]

Reflex syncope results from dysregulation of the autonomic nervous system leading to vasodilation, bradycardia, or both. It includes vasovagal syncope, situational syncope, and carotid sinus syndrome.[6] Reflex syncope accounts for more than 90% of cases among younger adults but only approximately 40% of cases among older adults.[5-7] Vasovagal syncope is the most common type of syncope and represents a reflex, neurally mediated faint that results from bradycardia (vagal excess), hypotension (withdrawal of sympathetic innervation), or most commonly, a combination of the two. Although vasovagal syncope generally carries a benign prognosis, injuries occur in more than 30% of episodes, with major injuries reported in approximately 15%. Older age and recurrent syncope or falls increase the likelihood of injury, as does the lack of a recognizable prodrome, which would allow patients to take preventive action (e.g., lying down) before the occurrence of syncope.[8] Situational syncope is reflex syncope due to an action or situation such as emotional stress, micturition, swallowing, or coughing. Carotid sinus syndrome occurs almost exclusively in older adults and accounts for 9 to 17% of syncopal events and up to 30% of otherwise-unexplained falls.[9]

Orthostatic hypotension also causes syncope and falls.[10] The prevalence of orthostatic hypotension increases with age, affecting less than 5% of persons younger than 50 years of age, more than 20% of persons older than 70 years of age, and up to 50 to 68% of older adults living in institutional settings.[11] Cardiac syncope is rare in younger adults, accounting for up to 5% of events, but the incidence increases with age, reaching 30% of syncopal events among older patients and leading to higher morbidity and mortality.[7] In older adults, multiple potential contributing mechanisms may coexist in up to one third of cases — a condition referred to as complex syncope.[9,10]

As diagnostic strategies and management guidelines have evolved over the past three decades, the percentage of syncopal events that are unexplained has decreased substantially (Fig. 1), declining from approximately 37% in early studies[12] to less than 10% in more recent cohorts in which structured diagnostic pathways and prolonged rhythm monitoring were used.[13] Consequently, tailored interventions are advocated and range from conservative management of triggers and deprescribing in hypotensive syncope to cardiac pacing and cardioneural ablation for problematic bradyarrhythmias or to the use of implantable cardiac defibrillators for serious ventricular arrhythmias.[7,13]

STRATEGIES AND EVIDENCE

EVALUATION

The starting point of the diagnostic evaluation of syncope is careful history taking about present and previous attacks, as well as eyewitness accounts, provided either in person or by means of telephone interview, followed by a physical examination, supine and standing blood-pressure measurements, and electrocardiography (ECG) (Fig. 2).[13] Details of triggers, symptoms, and signs before, during, and after the syncopal event, along with data about previous events and cardiac and neurologic history, will suggest a diagnosis (Table 1). The features associated with each cause of syncope — reflex, orthostatic, and cardiac — are presented in Table 1.

Vasovagal syncope results from vasodepressor hypotension and a systolic blood pressure of no more than 80 mm Hg, a heart rate of less than 40 beats per minute, or asystole lasting at least 3 seconds.[13] Carotid sinus syndrome is reflex syncope associated with carotid sinus hypersensitivity, which is defined by a pause lasting at least 3 seconds or by a decrease in the systolic blood pressure of at least 50 mm Hg during carotid sinus stimulation (massage over the carotid sinus).[9,13,14]

Orthostatic hypotension is defined by a re-

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duction in the systolic blood pressure of at least 20 mm Hg or in the diastolic blood pressure of least 10 mm Hg on standing.[7] It may be classified as initial orthostatic hypotension, which is a transient decrease in the blood pressure that occurs within 15 seconds after standing, is generally benign, and is detected by beat-to-beat recording of the blood pressure. Classic orthostatic hypotension is defined by a sustained decrease in the systolic blood pressure of at least 20 mm Hg (or of ≥30 mm Hg with supine hypertension) or in the diastolic blood pressure of least 10 mm Hg within 3 minutes after standing.[15] Delayed orthostatic hypotension occurs when the decrease in the blood pressure occurs after prolonged upright posture. Neurogenic orthostatic hypotension results from autonomic nervous system dysfunction due to central or peripheral neurologic disease.[15] Postprandial hypotension can also cause syncope and is defined by a decrease in the systolic blood pressure of at least 20 mm Hg within 2 hours after a meal owing to impaired cardiovascular compensation for splanchnic blood pooling during digestion; it often co-occurs with orthostatic hypotension.[16]

Cardiac syncope results from conditions that reduce cardiac output or cerebral perfusion. Such conditions include bradyarrhythmias, tachyarrhythmias, structural heart disease causing outflow obstruction, pulmonary hypertension, and acute vascular disorders such as pulmonary embolism or aortic dissection.[17,18]

SYNCOPE MIMICS

Syncope mimics are classified under nonsyncopal causes of transient loss of consciousness according to the 2018 European Society of Cardiology (ESC) guidelines regarding syncope.[13] The most common mimic of syncope that manifests as transient loss of consciousness is epilepsy. Discriminating between epilepsy and syncope from the patient's history alone may be difficult. For example, urinary incontinence does not distinguish between syncope and epilepsy and may occur in 10 to 20% of syncopal events, particularly in older adults.[19] Other features that are attributed to epilepsy such as tongue biting, jerking movements, and postevent drowsiness also occur in syncope.[19] Other causes of syncope mimics are listed in Table 2.

RISK STRATIFICATION

The severity of syncope depends on associated cardiac disease and the risk of clinically significant consequences, such as injury. Short-term prognosis (associated with outcomes in the emergency department and up to 30 days after syncope) is related mainly to the cause of syncope and

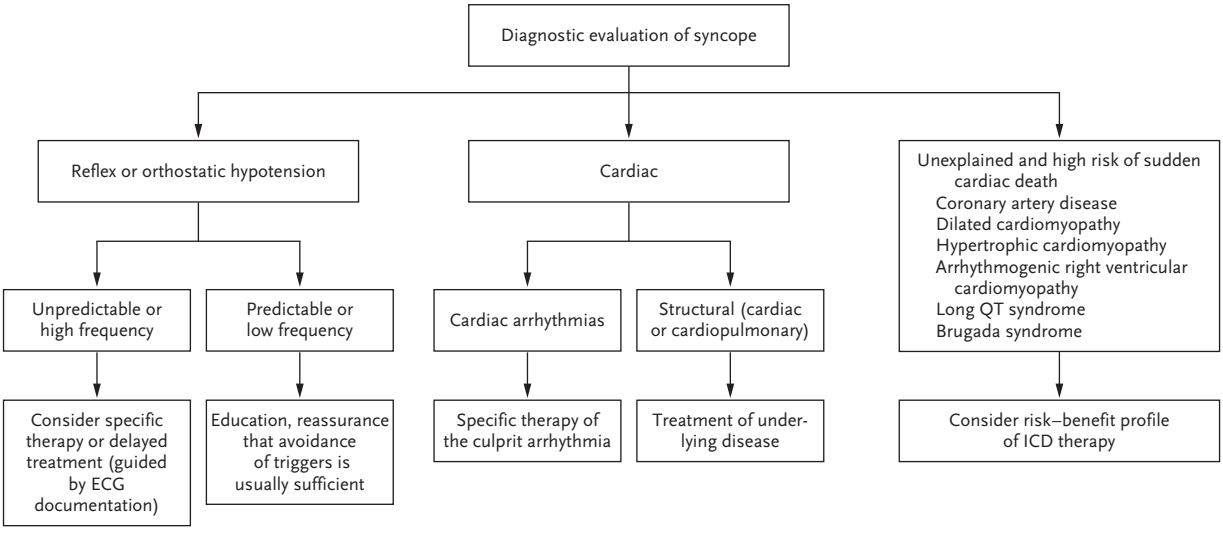


Figure 1. Treatment of Syncope Based on the Diagnostic Evaluation.

Adapted from Brignole et al.[13] The general framework of treatment is based on risk stratification and the identification of specific mechanisms when possible. ICD denotes implantable cardioverter–defibrillator.

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short-term reversibility of the underlying condition; long-term prognosis (up to 12 months of follow-up) is related to the effectiveness of therapy and the severity and progression of underlying diseases and likelihood of injuries.[18,20-23]

Features that increase the probability of an underlying cardiac cause include an age of more than 60 years, presence of cardiac disease (i.e., ischemic heart disease, valvular heart disease, structural heart disease, or underlying device malfunction), male sex, brief prodrome, palpitations or chest pain before syncope, sudden syncope, syncope during exertion or when seated or supine, a low number of syncopal episodes (one or two), abnormal cardiac examination, family history of inheritable conditions or premature sudden cardiac death (at <50 years of age), and known congenital heart disease.[24,25] ECG features of cardiac syncope are discussed in Table 1.

Features associated with an increased probability of injury include unexplained falls, recurrent falls, brief or absent prodrome, coexisting conditions such as osteoporosis and cognitive impairment, and polypharmacy. Culprit medications include diuretic, antianginal, psychotropic, anticholinergic, opioid, anticoagulant, and antiplatelet agents; the recent introduction of antihypertensive or antiarrhythmia medications is also associated with syncope.[26,27]

Additional examinations may be performed as indicated on the basis of these findings (Fig. 3). Immediate ECG monitoring is appropriate when there is a suspicion of arrhythmic syncope, as is an echocardiogram in patients with known heart disease or evidence suggestive of structural heart disease. The duration of longer-term external cardiac monitoring will depend on the frequency of symptoms and likelihood of arrhythmia detection during a monitoring period; for example, the detection of one or two events per month may require up to 4 weeks of monitoring. Symptoms of known or suspected cardiac disease warrant further cardiac investigations such as exercise stress testing and coronary-artery evaluation.

To assess for carotid sinus syndrome, carotid sinus massage should be carried out both while the patient is supine and while the patient is upright, with simultaneous heart-rate and blood-pressure monitoring. Carotid sinus massage should be done in patients older than 40 years of age with suspected reflex syncope, syncope on head turning, or unexplained falls. Head-up tilt-table testing is indicated when there is suspicion of reflex syncope. Ambulatory monitoring of the blood pressure is helping in determining circadian blood-pressure behaviors and hypotensive episodes including postprandial hypotension. In one cross-sectional diagnosis study involving patients with established reflex syncope, one or more daytime decreases in the systolic blood pressure to less than 90 mm Hg carried a 91% specificity and 32% sensitivity for the diagnosis of reflex syncope, as compared with controls undergoing ambulatory blood-pressure monitoring for non–syncope-related reasons (e.g., hypertension).[28] In this study, reflex syncope was diagnosed when the clinical features were consistent with a reflex mechanism and competing diagnoses had been ruled out. Tilt-table testing

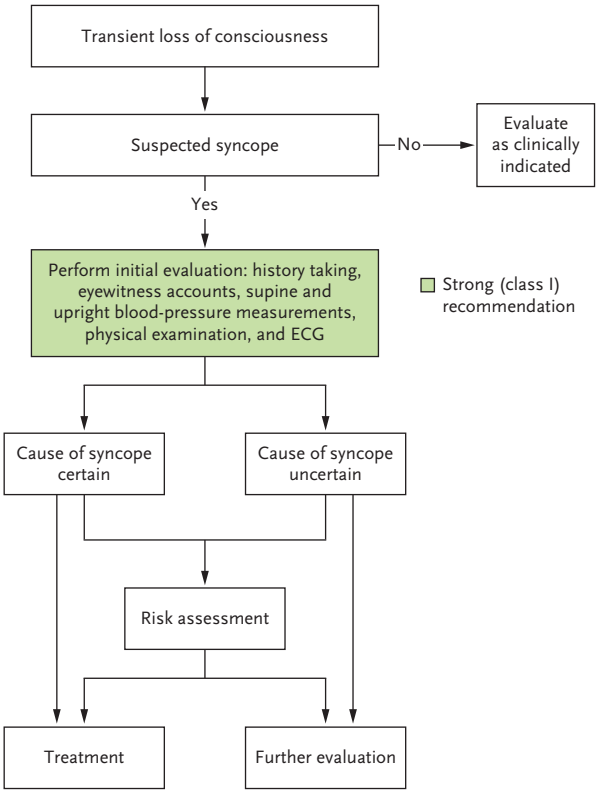


Figure 2. General Principles for Initial Evaluation of All Patients after an Episode of Syncope.

Adapted from the 2017 guidelines from the American College of Cardiology and American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society (ACC–AHA–HRS).[7] ECG denotes electrocardiography.

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Table 1. Clinical Features That Can Suggest a Diagnosis on Initial Evaluation.*

Feature
Reflex syncope
Long history of recurrent syncope, in particular occurring before the age of 40 yr
Occurrence after unpleasant sight, sound, smell, or pain
Occurrence with prolonged standing
Occurrence during meal
Occurrence in crowded or hot places
Autonomic activation before syncope: pallor, sweating, nausea, or vomiting
Occurrence with head rotation or pressure on carotid sinus (e.g., due to tumors, shaving, or tight collars)
Absence of heart disease
Syncope due to orthostatic hypotension
Occurrence shortly after standing
Occurrence with prolonged standing
Occurrence with standing after exertion
Postprandial hypotension
Temporal relationship with initiation or changes in dose of vasodepressive drugs or diuretics leading to hypotension
Presence of autonomic neuropathy or parkinsonism
Cardiac syncope
Occurrence during exertion or when supine
Sudden-onset palpitation immediately followed by syncope
Family history of unexplained sudden death at young age
Presence of pulmonary hypertension or structural heart, coronary artery, or pericardial disease
ECG findings suggesting arrhythmic syncope:
Bifascicular block (defined as either left or right bundle- branch block combined with left anterior or left posterior fascicular block)
Other intraventricular conduction abnormalities (QRS duration, ≥0.12 sec)
Mobitz type I second-degree atrioventricular block and first-degree atrioventricular block with markedly prolonged PR interval
Asymptomatic mild inappropriate sinus bradycardia (40–50 beats/min) or slow atrial fibrillation (40–50 beats/min) in the absence of negatively chronotropic medications
Nonsustained ventricular tachycardia
Preexcited QRS complexes
Long or short QT intervals
Early repolarization
ST-segment elevation with type 1 morphology in leads V1, V2, and V3 (Brugada syndrome)
Negative T waves in right precordial leads, epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy
Left ventricular hypertrophy suggesting hypertrophic cardiomyopathy

* Adapted from Brignole et al.[13] ECG denotes electrocardiography.

was performed to confirm the diagnosis when reflex syncope was suspected but not established after the initial assessment.

Blood tests should be carried out when clinically indicated. Evaluation of the hematocrit or hemoglobin level is warranted when hemorrhage is suspected, oxygen saturation and blood-gas analysis when hypoxemia is suspected, the troponin level when cardiac ischemia-related syncope is suspected, and the D-dimer level or computed tomography when pulmonary embolism is suspected.[7,13]

SYNCOPE IN THE OLDER PATIENT

The approach to the assessment and treatment of an older patient with syncope is similar to that in other age groups.[13,29-31] Because complex syncope occurs in more than one quarter of older patients with syncope, multifactorial evaluation and intervention is recommended.[10,29,30,32,33] Orthostatic measurements of blood pressure, carotid sinus massage, and head-up tilt-table testing can be performed even in frail patients with cognitive impairment.[9,31,32]

MANAGEMENT AND TREATMENT

In the absence of serious medical conditions or injury risk, treatment of patients with presumptive reflex-mediated syncope in the outpatient setting is reasonable.[7] In patients at intermediate risk with an unclear cause of syncope, the use of a structured emergency-department observation protocol with 6 to 12 hours of cardiac monitoring is effective in reducing the incidence of hospital admission.[24,34,35]

Hospital admission is indicated only in patients at high risk.[7,20,36] In a randomized trial involving 124 adults older than 50 years of age who presented with syncope, consecutive participants were randomly assigned to observation in the emergency department or to hospital admission. The rates of serious outcomes (tachyarrhythmia, unstable angina, pulmonary embolism, aortic dissection, and cerebral infarction) after hospital or emergency department discharge were similar in the two groups at both 30 days and 6 months. Index hospital costs in the observation group were also substantially lower than those in the admission group.[37,38]

A dedicated syncope management unit further enables a definitive diagnosis and addi-

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Table 2. Disorders That Can Cause Transient Loss of Consciousness and Mimic Syncope.*

Condition	Key Features	Distinguishing Points from Syncope
Epileptic seizure	Tonic–clonic movements, tongue biting (lateral), postictal confusion	Longer loss of consciousness, postictal phase, no rapid recovery
Psychogenic pseudosyncope	Apparent loss of consciousness, often prolonged, eyes closed	Normal blood pressure and heart rate during event, no true cerebral hypoperfusion
Hypoglycemia	Sweating, confusion, tremor	Low glucose, gradual onset, not sudden collapse
Vertebrobasilar TIA	Dizziness, ataxia, diplopia	Focal neurologic signs, loss of consciousness uncommon
Drop attacks	Sudden fall without loss of consciousness	Patient remains conscious
Cataplexy	Sudden loss of muscle tone triggered by emotion	Consciousness preserved, associated with narcolepsy
Intoxication with alcohol or drugs	Altered consciousness, unsteadiness	Persistent confusion, not transient
Concussion or head injury	Loss of consciousness after trauma	Trauma is cause, not consequence
Metabolic disturbances	Hypoxemia, electrolyte imbalance	Gradual or fluctuating course
Panic attack or anxiety	Hyperventilation, palpitations	Rarely a true loss of consciousness, situational context
Sleep disorders (e.g., narcolepsy)	Sudden sleep episodes	Occurs in sleep context, not circulatory

* Adapted from van Dijk et al.[19] TIA denotes transient ischemic attack.

tional testing such as carotid sinus massage while the patient is supine and upright, head-up tilt-table testing, and ambulatory cardiac and blood-pressure monitoring together with the introduction of a personalized treatment plan and follow-up. Implantable cardiac monitors (e.g., an implantable loop recorder) may detect arrhythmias during events or may suggest hypotensive or functional syncope when events are unrelated to arrhythmia. Four companies currently provide implantable monitoring devices: Abbott, Biotronik, Boston Scientific, and Medtronic. Given a battery life of up to 4 years in these devices, less-frequent events can be tracked.[37]

Because attributing a specific cause to syncope can be difficult unless symptoms are captured during blood-pressure or heart-rate monitoring, treatment of all possible causes is recommended. Therapy that is aimed at preventing the recurrence of syncope is determined largely by the mechanism of syncope rather than by its cause — that is, by targeting hypotension, bradycardia, or tachycardia.

The general treatment of hypotension in persons without known structural heart disease varies widely and includes increasing fluid and electrolyte intake, avoidance of syncope triggers, and discontinuation of possible culprit medications. Both blood-pressure–lowering and psychotropic medications cause reflex syncope and orthostatic hypotension.[39-41] A large systematic review of 78 studies involving older adults showed that of all antihypertensive agents, the lowest risks of hypotensive events were associated with angiotensin-converting–enzyme inhibitors (odds ratio, 0.85; 95% CI, 0.81 to 0.89) and calcium-channel blockers (odds ratio, 0.81; 95% CI, 0.74 to 0.90).[42]

In some cases, patients with reflex syncope or with syncope due to orthostatic hypotension may receive medications that enhance intravascular volumes or target venoconstriction, in addition to conservative measures and deprescribing. In a systematic review of the efficacy and safety of pharmacologic treatments for orthostatic hypotension (25 studies, including randomized, controlled trials and observational studies), droxidopa and midodrine therapy each consistently reduced orthostatic symptoms; atomoxetine and fludrocortisone each showed moderate efficacy; and pyridostigmine and octreotide, as combination therapy, showed potential for the treatment of refractory orthostatic hypotension, but robust evidence for use was

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lacking. In one double-blind, multicenter, randomized, placebo-controlled trial, after 2 weeks of dose stabilization, the investigators found a marginally nonsignificant lower risk of recurrent vasovagal syncope in the fludrocortisone group (hazard ratio, 0.69; 95% CI, 0.46 to 1.03; P=0.07).[42]

Cardiac pacing is the most effective therapy for symptomatic bradycardia, particularly in older patients. In meta-analyses of randomized trials of pacing in patients with recurrent reflex syncope (vasovagal syncope or carotid sinus syndrome) and documented bradycardia, pacemaker therapy was associated with a reduction of more than 50% in the risk of syncope recurrence.[43,44] Continued presyncope and syncope despite pacing was attributed to clinically significant hypotension independent of bradycardia.

Cardioneural ablation is a newer intervention for vagally mediated syncope in which ablation of the epicardial ganglionated plexuses reduces vagal activity; this procedure may be a promising tool for the treatment of bradycardia reflex syncope.[45,46] Randomized, controlled trials of cardioneural ablation are ongoing (ClinicalTrials.gov numbers, NCT03903744, NCT05573178, NCT05803148, NCT05461859, and NCT04642976).

Treatment of ventricular arrhythmia depends on the specific arrhythmia subtype (e.g., premature ventricular complexes, sustained ventricular tachycardia, or ventricular fibrillation), symptom severity, hemodynamic stability, and the underlying cause, such as myocardial ischemia, structural heart disease, electrolyte disturbances, or inherited channelopathies. Management may include correction of reversible causes, antiarrhythmic medications, catheter ablation, implantable cardioverter–defibrillator therapy, and emergency

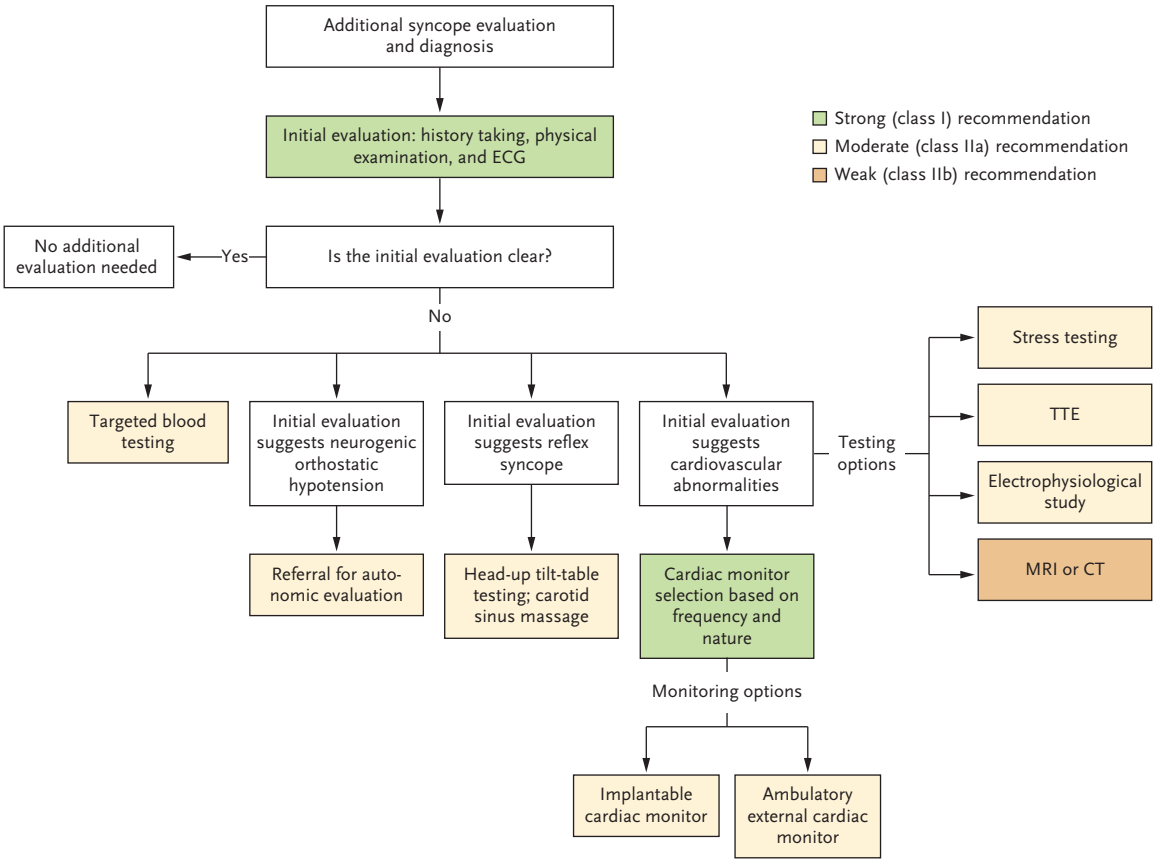


Figure 3. Additional Evaluation and Diagnosis of Syncope According to Current ACC–AHA–HRS Guidelines.

Adapted from the 2017 ACC–AHA–HRS guidelines.[7] CT denotes computed tomography, MRI magnetic resonance imaging, and TTE transthoracic echocardiography.

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KEY POINTS

SYNCOPE

- Syncope is caused by transient cerebral hypoperfusion and is classified as reflex, orthostatic, or cardiac.
- Initial evaluation with history taking, physical examination, electrocardiography, and orthostatic blood-pressure measurement identifies the cause in most patients.
- Risk stratification determines the appropriateness of hospitalization and further testing.
- Management is mechanism-specific and aims to reduce the risks of recurrence and injury.

defibrillation in life-threatening cases. Detailed recommendations for diagnosis, risk stratification, and treatment are provided in contemporary clinical practice guidelines.[47]

GUIDELINES

The two principal, most recently published guidelines for syncope are the ESC guidelines for the diagnosis and management of syncope[13] and the guidelines from the American College of Cardiology and American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society (ACC–AHA–HRS) for the evaluation and treatment of patients with syncope.[7] Both sets of guidelines include recommendations regarding overlap between syncope and falls, as illustrated by the case vignette, and align with the management strategy of the case. They differ in recommendations regarding diagnostic pathways and management strategies. In particular, the ESC guidelines strongly recommend establishing specialized syncope units for management and diagnosis, whereas the ACC–AHA–HRS guidelines do not. The ESC guidelines also recommend earlier use of implantable loop recorders for patients with unexplained syncope and unexplained falls. The ESC guidelines place a stronger emphasis on patient education and behavioral lifestyle changes for reflex syncope management. This review is more aligned with the ESC guidelines.

AREAS OF UNCERTAINTY

Additional data from clinical trials are needed to guide the management of unexplained falls and complex syncope in older adults.[13] Further studies are necessary to determine appropriate service models for syncope assessment and care. In addition, ongoing uncertainty remains regarding effective approaches to blood-pressure reduction in frail older patients with hypertension.[40] Although fludrocortisone is widely used for the treatment of orthostatic hypotension, the evidence of benefit is not definitive.[48]

CONCLUSION

In a patient such as the one described in the vignette, injuries are first managed while emergency causes of syncope are simultaneously ruled out. Because the events were sudden and injurious in an older man, I would deem him to be at intermediate-to-high risk and therefore would recommend monitoring in an emergency department observation unit where he would undergo ECG, echocardiogram, and orthostatic blood-pressure studies. If no arrhythmias or structural heart disease were noted but his blood pressure decreased to 80/55 mm Hg with symptoms of lightheadedness on standing, I would perform carotid sinus massage to evaluate for carotid sinus syndrome. Such a diagnosis would be established with a cardiac pause of at least 3 seconds during upright carotid sinus massage during which the patient would lose consciousness. Although more-prolonged cardiac monitoring, such as an implantable loop recorder, might capture symptom–rhythm correlation, given the patient's age and injuries, it would be reasonable to proceed directly to implantation of a dual-chamber cardiac pacemaker for syncope attributed to carotid sinus syndrome, coupled with modification of possible culprit medications (antihypertensive and antidepressant medications) to manage orthostatic hypotension. These recommendations align with the most recent U.S. and European guidelines for syncope.

Disclosure forms provided by the author are available with the full text of this article at NEJM.org.

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