Chapter 4
Echocardiography in Patients with Syncope

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Abstract Syncope is defined as a temporary, abrupt loss of consciousness with spontaneous return to baseline. This chapter offers a framework for a focused approach to syncope. A detailed history and physical examination establishes the pretest probability that the syncope is of cardiac origin. Although echocardiography is not indicated for many cases of syncope, it can be a valuable tool to risk stratify syncope when it is suspected to be of cardiac origin. Transthoracic echo (TTE) is an efficient means of rapidly identifying structural heart disease, which suggests a higher probability of cardiogenic syncope and places the patient in a higher risk category than a patient with a structurally normal heart.

Keywords Syncope · Arrhythmia · Cardiomyopathy

Introduction

Syncope is defined as a temporary, abrupt loss of consciousness with spontaneous return to baseline. It contributes to approximately 3% of emergency-department admissions as well as 6% of total hospital admissions in the United States [1]. The cost of medical care for this condition approaches two billion dollars annually [2]. Although it is a common medical problem, syncope is really an umbrella term representing a multitude of disorders and frequently presents a diagnostic challenge. Many institutions lack a standardized approach because there is no "gold standard" diagnostic test for the cause of syncope [3].

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Dedication: This chapter is dedicated to the memory of Dr. Farooq Chaudhry who was a cherished colleague in the Division of Cardiology at Mount Sinai Hospital. His untimely passing in 2017 has left a void in our hearts and minds.

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Mechanisms of Syncope

Syncope can be broadly classified based on the underlying mechanism [4]. They can be subdivided into neurally mediated syncope, orthostatic syncope, and cardiogenic syncope (Fig. 4.1). Frequently, a detailed history and physical examination are sufficient to identify the cause of syncope. When there is sufficient suspicion of cardiogenic syncope, an electrocardiogram and/or echocardiogram may be appropriate for further risk stratification.

Neurally Mediated Syncope

Neurally mediated or "reflex syncope" accounts for 45% of cases of known syncope [5]. It mainly presents with prodromal symptoms such as nausea, flushing, warmth, and diaphoresis. It is associated with prolonged standing, emotional stress, urination, coughing, swallowing, or carotid stimulation. It is the most common cause of syncope regardless of age and gender. Neurally mediated syncope is driven by imbalance in the autonomic nervous system leading to excess parasympathetic activation. It can manifest as a vasodepressor (vasodilatory, hypotensive) response or as cardioinhibitory (vagally mediated bradycardia). It is also one of the most common

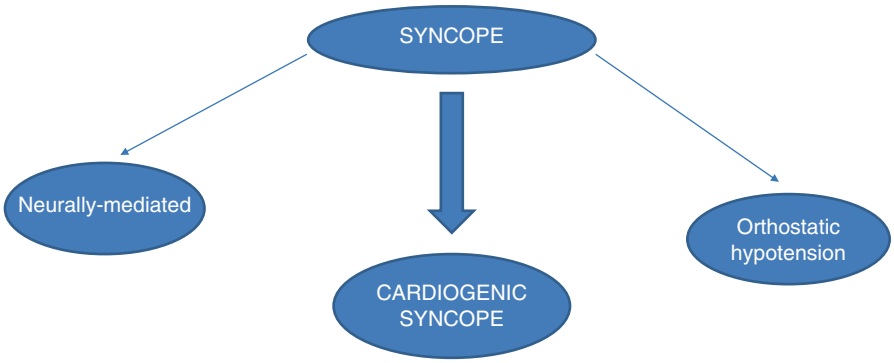

Fig. 4.1 Mechanisms of syncope

causes of recurrent syncope [6]. Regardless, the prognosis is very good and the mortality is low [7]. This cause of syncope typically does not warrant echocardiographic evaluation or inpatient admission.

Orthostatic Hypotension

Orthostatic hypotension is defined as a symptomatic drop in blood pressure with postural changes [3]. Similar to neurally mediated and cardiogenic syncope, there are a number of causes [8]. It is frequently seen in the elderly following postural changes due to age-related degeneration of the autonomic nervous system. Pathological conditions such as parkinsonism, multisystem atrophy, peripheral neuropathies, and diabetes can cause orthostatic hypotension due to alterations in the autonomic nervous system regulation and loss of compensatory mechanisms. It can also result from a number of medications such as diuretics, nitrates, antidepressants, and antipsychotics when it carries a favorable prognosis [9]. As with neurally mediated syncope, this diagnosis can be made by history and physical examination, and does not merit echocardiographic evaluation unless concomitant structural heart disease is suspected.

Cardiogenic Syncope

Cardiac disorders are the most common causes of syncope occurring in the critical care setting, and contribute to nearly 80% of intensive care unit admissions for syncope [10]. Identification and timely assessment of cardiogenic syncope are critical because the associated morbidity and mortality are higher compared to other causes of syncope [11]. Cardiac syncope can be broadly subdivided into arrhythmias or obstruction in blood flow (Fig. 4.2).

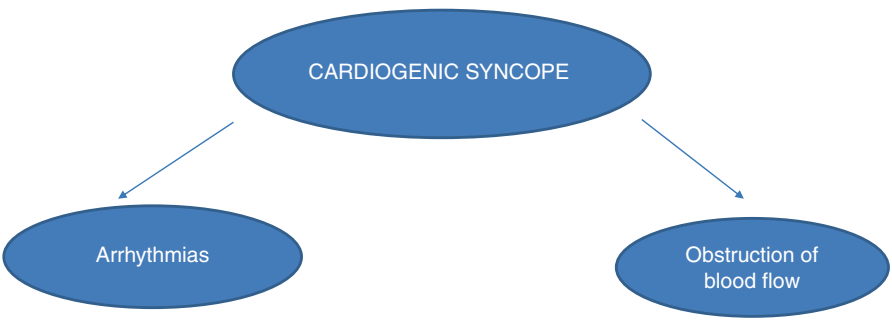

Fig. 4.2 General mechanisms of cardiac syncope

Disorders of Heart Rhythm

Cardiac arrhythmias are the most common cause of cardiogenic syncope [3]. Severe rhythm disturbances may mediate syncope by causing an acute drop in cardiac output due to abrupt decreases or increases in heart rate with resulting cerebral hypoperfusion. Among cardiac arrhythmias, severe bradyarrhythmia and asystolic pauses are frequently encountered. Common causes for bradyarrhythmia include sick sinus syndrome, Mobitz type II block, and complete heart block [3]. Tachyarrhythmias (ventricular and supraventricular) are the second most common cause of cardiac arrhythmia causing cardiogenic syncope [12]. Medications, particularly AV nodal blockers, QT interval prolonging drugs, and antiarrhythmics are important causes of arrhythmia [3]. Arrythmogenic syncope is considered an important risk factor for sudden cardiac death [13].

Obstruction of Blood Flow

Obstructive cardiac lesions presenting with syncope can be potentially life threatening but can be readily identified by transthoracic echo. For example, syncope is well established as a very high-risk presenting symptom in patients with severe aortic stenosis. Furthermore, other common conditions such as cardiac tamponade, hypertrophic cardiomyopathy, mitral stenosis, and massive pulmonary embolism may be lethal if not recognized and treated promptly. These findings are summarized in Table 4.1.

Table 4.1 High-risk cardiac lesions and their echocardiographic correlates

Structural heart disease which may present with syncope	Associated echo findings
Severe aortic stenosis	Calcified aortic valve with reduced excursion, elevated velocities across aortic valve
Hypertrophic obstructive cardiomyopathy	LV hypertrophy, elevated velocities in LV outflow tract, abnormal papillary muscles, early aortic valve closure
Cardiac tamponade	Early diastolic collapse of RV, systolic collapse of RA, enhanced ventricular interdependence, IVC plethora
Mitral stenosis	Thickened, domed mitral valve with elevated gradient
Massive pulmonary embolism	RV dilation, dysfunction
Anomalous origin of coronary arteries	Echo may be normal
Severe pulmonary hypertension	High-velocity tricuspid regurgitation jet, dilated pulmonary artery, early closure of pulmonic valve, elevated pulmonic regurgitant end-diastolic velocity
Arrhythmogenic right ventricular cardiomyopathy (ARVC)	Dilated RV, RV wall motion abnormalities

Diagnosis: History and Physical Examination

A proper and detailed medical history is paramount in distinguishing possible cardiogenic syncope from more benign causes of syncope. The historical features which distinguish cardiac syncope from neurally mediated or orthostatic syncope are summarized in Table 4.2.

Attention should be paid to eliciting the setting of syncope, prodromal symptoms, medications, associated medical conditions, and family history of malignant arrhythmia, structural heart disease, or sudden death. Many patients with cardiogenic syncope may present with loss of consciousness during exercising or lying flat [14]. These patients are typically asymptomatic prior to the event, although some may experience palpitations just prior to the event [15]. If the history does not clearly suggest a neurally mediated or orthostatic cause of syncope, then cardiogenic syncope should be considered, particularly in older patients. Any information regarding previous history of heart disease, cardiac surgeries, and family history of heart disease, medications, and comorbidities should be noted.

On physical exam, one can seek out clues suggesting structural heart disease such as murmurs, gallops, distended jugular veins, cyanosis, or clubbing.

How to Differentiate Noncardiac Syncope from Cardiogenic Syncope

Demographics are important in establishing the probability of cardiac syncope. Patients with cardiac syncope are more frequently male, typically greater than 60 years of age [16]. In patients with a history of heart disease or congenital heart

Table 4.2 Historical clues to distinguish cardiac from noncardiac causes of syncope

Features suggesting cardiac causes of syncope	Features suggesting noncardiac causes
Older age (greater than 60 years)	Younger age
Known coronary disease, reduced ventricular function, or structural heart disease	No known cardiac disease
Exertional or supine syncope	Syncope from standing position or after positional changes
Absent or minimal prodrome, usually palpitations	Prodrome of nausea, warmth, sweating
Family history of sudden death	Triggers such as pain, dehydration, emotional stress, coughing, laughing, swallowing, urinating
One or fewer prior episodes of syncope	Multiple, recurrent episodes of syncope
Abnormal cardiac physical examination	Normal physical examination

disease, they tend to be younger than 50 years. Patients with cardiac syncope usually do not have a history of recurrent syncope, and typically have only one or two prior episodes [16].

Patients with noncardiac syncope are usually of younger age and frequently have no history of cardiac disease. These patients may have a history of frequent and recurrent syncope. Syncope often occurs in standing position or in association with pain or emotional duress. They may also have a history of physical triggers such as coughing, laughing, and urination.

Role of ECG for Syncope

An ECG is essential in the evaluation of syncope. It should be the first diagnostic test performed for suspected cardiogenic syncope. It is a low-cost, easily available, and reproducible test that can detect the presence of arrhythmia and may provide clues to the presence of underlying structural heart disease. The gold standard for the diagnosis of cardiogenic syncope is when a relationship between symptoms and documented arrhythmia on ECG is seen [17]. However, some patients may have arrhythmias such as prolonged asystole (>3 s), rapid supraventricular tachycardias (SVTs) (>160 b.p.m. for >32 beats), or ventricular tachycardia seen on ECG when asymptomatic. This can also be considered diagnostic for arrhythmogenic syncope [18]. However, if a patient presents with syncope and a normal ECG, cardiogenic syncope cannot be ruled out. Continuous monitoring may be required in these cases. If cardiogenic syncope is suspected, echocardiography can provide valuable prognostic information, whether or not the initial ECG is abnormal.

Role of Echocardiography for Syncope

Transthoracic echocardiography should be considered in the setting of suspected cardiogenic syncope or structural heart disease in the setting of syncope [16]. If the history, physical, and ECG clearly point towards a noncardiac cause of syncope, then there is no role of echocardiogram as part of the evaluation (Fig. 4.3). If, however, the initial evaluation is equivocal and there are historical, exam, or ECG features suggesting cardiac syncope, then transthoracic echocardiogram is reasonable [16]. Given the noninvasive nature, low risk, and relatively low cost of TTE, it can be a valuable test for suspected cardiac syncope.

In certain conditions, echocardiography is instrumental in making the final diagnosis. Conditions such as severe aortic stenosis, cardiac tamponade, hypertrophic obstructive cardiomyopathy, mitral stenosis, or atrial myxoma can be definitively diagnosed by TTE. Other findings such as reduced LV ejection fraction can help inform prognosis, as discussed below.

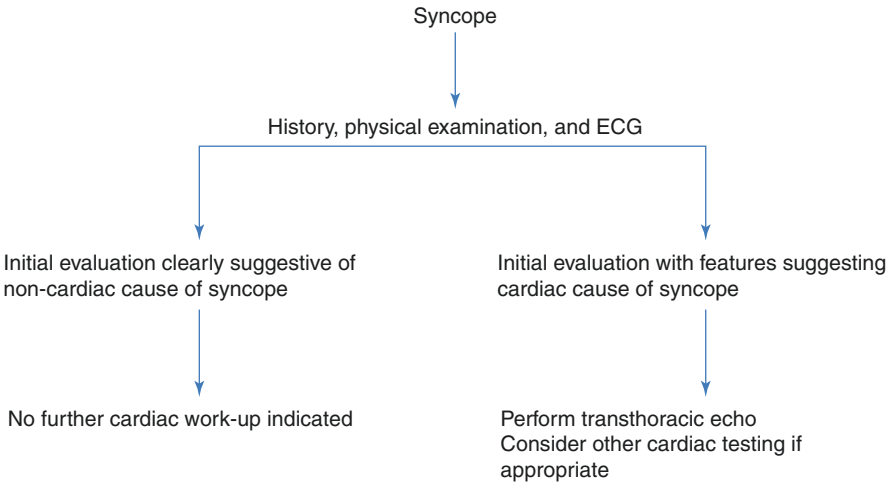

Fig. 4.3 A diagnostic approach to syncope

Echocardiography and Risk Stratification in Syncope

Although echocardiography may not always indicate a clear cause for syncope, echo remains an important tool for risk stratification in the setting of syncope. Patients with reduced left or right ventricular function and/or evidence of prior myocardial infarction may be at higher risk for adverse events in the short and long terms. Ejection fraction can also be used as a predictor of arrhythmia in certain subset of patients with an unremarkable initial ECG [19].

Quantifying risk in syncope is an area of ongoing research. There are a number of clinical risk scores which have been validated in cases of syncope, though the studies are heterogeneous and difficult to compare [16]. Syncope risk scores incorporating echocardiographic findings have not been well described.

In the setting of syncope and known pulmonary embolism, the presence of right ventricular dilation or dysfunction on transthoracic echo adds significant prognostic information and may help guide the decision to perform thrombolysis. Echocardiography is the best imaging study to detect right ventricular dysfunction during acute PE [20]. In cases of submassive pulmonary embolism, thrombolysis may be offered if RV dysfunction or dilation is present [20].

High-Risk Features Meriting Admission or Extended Observation

The 30-day mortality for syncope is less than 1% [20]. Despite the overall favorable prognosis, risk stratification is essential to determine which patients would benefit from higher levels of monitoring, including inpatient or intensive care admissions.

Table 4.3 European Society of Cardiology guidelines for cardiogenic syncope admission

Severe structural or coronary artery disease including:
• Heart failure • Reduced ejection fraction • Previous myocardial infarction
Clinical features suggesting arrhythmic syncope:
• Exertional syncope, syncope while supine, palpitations prior to syncope • Family history of sudden cardiac death • History of non-sustained ventricular tachycardia
High-risk ECG features:
• Bifascicular block (LBBB or RBBB with left hemi-block) • Prolonged QRS duration • ECG suggestive of pre-excitation, Brugada pattern • Arrhythmogenic ventricular cardiomyopathy
Other important comorbidities:
• Severe anemia • Electrolyte imbalance

Table 4.4 Canadian Cardiovascular Society criteria for syncope admission

Major risk factors (should have urgent, inpatient cardiac evaluation):
• Abnormal ECG (acute or old infarct, tachyarrhythmia, bradyarrhythmia, or conduction disease
• History of significant structural heart disease or arrhythmia
• Hypotension (systolic blood pressure < 90 mmHg)
Minor risk factors (can consider urgent inpatient evaluation):
• Age >60
• Associated dyspnea
• Anemia
• Hypertension
• Cerebrovascular disease
• Family history of sudden death
• Syncope while supine, exercising, or without prodrome

The presence of high-risk echocardiographic features is among some of the criteria which would generally merit expedited workup with inpatient admission for syncope, according to the guidelines set forth by the European Society of Cardiology (ESC) and Canadian Cardiovascular Society (CCS) [3, 21]. These features are delineated in Tables 4.3 and 4.4.

Role of Advanced Echocardiography in Syncope

Exercise stress testing can be considered in situations of exertional syncope. However, exercise stress echocardiography should be reserved for individual cases

in which exercise would be expected to provide incremental information above and beyond the resting echocardiogram, as may be seen in dynamic intracavitary or LV outflow tract obstruction.

Transesophageal echocardiography (TEE) is generally not indicated for the evaluation of syncope unless there are specific structural features that would be better identified on TEE, such as atrial myxoma or mitral valve pathology not well characterized on TTE.

Conclusion

The CCU should be equipped to provide rapid and immediate access to echocardiography for all patients with possible cardiogenic syncope. There are several "can't miss," life-threatening causes of cardiogenic syncope, most of which can be rapidly identified or excluded with a good-quality transthoracic echo. Even when the echocardiogram does not indicate a clear cause for the syncope, the presence or absence of high-risk features can help risk stratify the patient and aid in selecting optimal monitoring and follow-up.

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