Syncope
Electrocardiographic and Clinical Correlation

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KEYWORDS

• Syncope • Electrocardiogram • Arrhythmias

KEY POINTS

- Syncope is a frequent condition, owing to a transient global cerebral hypoperfusion, that may depend on a reduction of vascular total peripheral resistance and/or cardiac output.
- Cardiac syncope doubled the risk of death from any cause and increased the risk of nonfatal and fatal cardiovascular events.
- Arrhythmias are the most common cardiac causes of syncope. Both bradyarrhythmias and tachyarrhythmias may predispose to syncope.
- The first line evaluation relies on clinical history, physical examination, active standing test, 12-lead echocardiogram.
- The diagnostic yield of electrophysiological study in detecting the cause of syncope depends highly on the pretest probability.

EPIDEMIOLOGY

Syncope is a transient loss of consciousness owing to a transient global cerebral hypoperfusion and is characterized by a rapid onset, a short duration, and a spontaneous and complete recovery.[1] This condition is extremely common in the general population.[2] In the latest report of the Framingham Offspring study, 10% of the 7814 participants (mean age, 51 years; range, 20–96 years) reported at least 1 syncope. The prevalence of syncope is very high in patients between the age of 10 and 30 years, uncommon in adults with an average age of 40 years, and peaks again above the age of 65 years.[3]

CLASSIFICATION AND PATHOPHYSIOLOGY OF SYNCOPE

Box 1 provides a classification of the principal causes of syncope. The global cerebral hypoperfusion is responsible for an inadequate supply of oxygen and metabolic substrates to the brain[4] and is what differentiates syncope from other transient losses of consciousness without underlying cerebral hypoperfusion, such as epilepsy, hypoglycemia, and episodes of only apparent loss of consciousness, such as falls.[1]

Cerebral autoregulation maintains a constant blood flow within a wide range of pressures (systolic blood pressure between 60 and 190 mm Hg). When the systolic blood pressure decreases below this threshold, brain perfusion decreases slowly and progressively and, if this hemodynamic status lasts for 8 to 15 seconds, ischemia and ultimately loss of consciousness will follow.[5] Therefore, the main mechanism is a decrease in the systemic blood pressure (BP), which may depend on a reduction of vascular total peripheral resistance and/or cardiac output (CO).[1]

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Card Electrophysiol Clin 10 (2018) 371–386
https://doi.org/10.1016/j.ccep.2018.02.007

Box 1
Classification of syncope

Reflex (neurally mediated) syncope

Vasovagal

Orthostatic vasovagal: standing, or less common sitting

Emotional: fear, pain, instrumentation, blood phobia

Pain triggers: peripheral or visceral

Situational

Micturition

Gastrointestinal stimulation (swallow, defecation)

Cough, sneeze

Others (eg, laughing, brass instrument playing, weight lifting, after exercise)

Carotid sinus syncope

Syncope owing to orthostatic hypotension

Drug-induced orthostatic hypotension (eg, vasodilators, diuretics, phenothiazine, antidepressants)

Volume depletion (eg, hemorrhage, diarrhea, vomiting)

Primary autonomic failure (pure autonomic failure, multiple system atrophy, Parkinson's disease, dementia with Lewy bodies)

Secondary autonomic failure (diabetes, amyloidosis, spinal cord injuries, autoimmune autonomic neuropathy, paraneoplastic autonomic neuropathy, kidney failure)

Cardiac syncope

Arrhythmia as primary cause

Bradycardia

- Sinus node dysfunction (including bradycardia/tachycardia syndrome)
- Atrioventricular conduction system disease
- Implanted device malfunction

Tachycardia

- Supraventricular
- Ventricular (idiopathic, secondary to structural heart disease or to channelopathies)

Structural disease

Cardiac: Cardiac valvular disease, acute myocardial infarction/ischemia, hypertrophic cardiomyopathy, cardiac masses (atrial myxoma, tumors, etc)

Pericardial disease/tamponade, congenital anomalies of coronary arteries, prosthetic valves dysfunction

Cardiopulmonary and great vessels

Pulmonary embolus, acute aortic dissection, pulmonary hypertension

Adapted from Moya A, Sutton R, Ammirati F, et al. Guidelines for the diagnosis and management of syncope (version 2009): the task force for the diagnosis and management of syncope of the European Society of Cardiology (ESC). Eur Heart J 2009;30:2636; with permission.

Vascular total peripheral resistance may be decreased by an inappropriate reflex activity causing vasodilatation through withdrawal of sympathetic vasoconstriction (vasodepressive reflex syncope) or by functional and structural impairment of the autonomic nervous system.[1] A decrease in CO may be caused by reflex bradycardia (cardioinhibitory reflex syncope), cardiovascular causes (arrhythmia, structural disease including pulmonary embolism and pulmonary hypertension), inadequate