Syncope: Definition, Epidemiology, and Classification

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KEYWORDS

• Syncope • Transient loss of consciousness • Epidemiology • Cerebral hypoperfusion

KEY POINTS

- During the last 2 decades, major progress has been made in syncope as a consequence of the acceptance of a clear definition of this symptom.
- The strategy for diagnosis of syncope includes the following criteria: transient loss of consciousness must be established; rapid onset and short duration of transient loss of consciousness must be established; recovery must be "complete and spontaneous"; and head trauma and epilepsy must be ruled out.
- Awareness of this diagnostic strategy will help to avoid useless, costly, and painful examinations.

INTRODUCTION

For decades, in fact since the origin of medicine, syncope has not drawn much attention. For neurologists it was just a manifestation of epilepsy, for cardiologists, a manifestation of paroxysmal atrioventricular (AV) block, and for most physicians, a benign and not very positive expression of "weakness" (swoon, blackout, fainting). Interest in syncope arose in the mid-1980s when Kapoor and colleagues[1,2] published some articles in which, for the first time, syncope was not considered through the scope of one specialty but as a self-determining symptom not directed by an immediate diagnosis or therapy. A second major input occurred almost at the same time when Kenny and colleagues[3] reported the effectiveness of head up tilt test to reproduce syncope in patients with previous spontaneous episodes of loss of consciousness. These preliminary reports stimulated publications of many new findings on the subject, mostly by cardiologists, and finally in the late 1990s, syncope was considered sufficiently important in the area of cardiology to justify guidelines and recommendations directed by the European Society of Cardiology (ESC). A task force was therefore nominated and proposed the first ever published comprehensive document on syncope in 2001.[4] A complete revision of this first issue was published in 2009.[5] The present review of the definition, epidemiology, and classification of syncope makes ample reference to this latter document.

DEFINITION

One of the major contributions of the 2001 document and the subsequent 2009 version of the ESC guidelines is certain to have generated a precise and widely accepted definition of syncope.[4,5] Before that time, there was no widely accepted definition of syncope, which certainly helped explain the lack of interest in syncope observed for many years: everyone had their own definition, giving rise to conflicting and nonreproducible results and diagnosis.

In the 2009 version of the ESC guidelines,[5] syncope is defined as "a transient loss of consciousness (TLOC) due to transient global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery."

To satisfy this definition and to be classified as having syncope, a patient suspected of having this symptom must meet the following criteria: TLOC

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must be established; rapid onset and short duration of TLOC must be established; recovery must be "complete and spontaneous"; and head trauma and epilepsy must be ruled out.

Transient Loss of Consciousness

The first mandatory step is to establish that the patient had a TLOC. This information is easily obtained by questioning patients and/or eyewitnesses. However, some studies have recently demonstrated that almost 50% of patients during head up tilt test, whatever their age but predominantly older patients, denied any TLOC, although the loss of consciousness was confirmed by nurses and/or doctors.[6] In patients in whom TLOC remains uncertain, occurrence of loss of postural tone is a strong argument to consider that TLOC has really occurred.

In the absence of TLOC, syncope can be definitively excluded and an alternative diagnosis should be sought, such as falls, dizziness, cataplexy, vertigo, psychogenic pseudo-TLOC, or transient ischemic attack (TIA) of carotid origin. The latter diagnosis is frequently and erroneously considered to be the cause of syncope, but as stated by Van Dijk "in clinical practice TIA is characterized by focal neurological signs without TLOC, whereas syncope is characterized by TLOC without focal neurological signs" (Van Dijk, personal communication, 2010). How many useless, time-consuming, and expensive computed tomographic scans or even magnetic resonance images would have been avoided if this sentence had been displayed in every emergency room?

Is TLOC Characterized by Rapid Onset and Short Duration?

Once the occurrence of TLOC has been established, the next step is to answer to the following question: "Was TLOC characterized by rapid onset and short duration"? The term "rapid onset" is not very accurate because it can mean a few seconds or a few minutes. This ambiguity is certainly a minor limitation of the definition, but "rapid" could infer that the delay between the beginning of symptoms and TLOC is between a few seconds to less than 1 minute. The same reproach can be directed at the term "short duration." In that case, however, the overlap between "short" or "long" does not seem significant and it could be assumed that the term "short" excludes TLOC lasting more than a few minutes. When a TLOC exceeds this delay, it is not syncope but coma, definite stroke, or intoxication (alcohol, drugs). A common error leading to underestimate the real cause is to consider that syncope could be the consequence of hypoglycemia; however, hypoglycemia induces coma and not syncope.

Recovery

The third requirement before considering TLOC as a syncope is to establish that "recovery has been complete and spontaneous." This sentence has been added to exclude from the scope of syncope patients who had sudden death consecutive to malignant ventricular arrhythmias,[7] aborted as a result of resuscitation maneuvers and particularly external shocks. In that case the diagnostic strategy is totally different. However, that does mean, of course, that malignant ventricular arrhythmias when they are paroxysmal have to be excluded from the causes of syncope.

Rule Out Head Trauma and Epilepsy

Finally, even if the 3 preceding requirements have been satisfied, one is still missing to definitely consider a TLOC as syncope: "due to transient global cerebral hypoperfusion." This statement is certainly the most original contribution of the ESC definition but also the most crucial. The first 3 requisites included in the definition could also apply to 2 other conditions that are not syncope: TLOC due to head trauma and epilepsy. Without this addition, the 2 previous situations are considered syncope, which obviously could not be the case according to the experts selected in the task force of the ESC.[5]

For head trauma, TLOC is caused by cerebral concussion and not by global cerebral hypoperfusion. It is obviously diagnosed simply by questioning the patient. A rare situation could, however, be confusing: syncope could provoke head trauma as a consequence of loss of postural tone and fall. In this situation when patients are unable to describe which came first, trauma or TLOC, syncope should be consider the cause,[8] unless the alternative was proved.

Epilepsy is the result of cerebral neuronal discharge and not of global cerebral hypoperfusion. There are no simple methods to differentiate these 2 mechanisms: electroencephalography could be diagnostic when demonstrating clear abnormalities. However, it is not always indicative, particularly when the delay between the episode and the examination exceeds some days. Measurement of cerebral blood flow is only feasible in dedicated laboratories. Fortunately, syncope and epilepsy are sufficiently typical to be easily differentiated in most instances by questioning of the patient and/or witnesses.[9] If doubt persists, a multidisciplinary discussion is definitely indicated.