

PART 1

Syncope and TLOC overview

CHAPTER 1

Definition and classification of syncope and transient loss of consciousness

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Syncope is a common complaint responsible for up to 1% of admissions in emergency departments in Europe [1–4]. During the last two decades cardiologists have become the specialists most involved in developing the diagnosis and treatment strategies for patients with presumed syncope, but they are not alone; many other physicians of various specialities are interested in the management of patients with syncope, including neurology, internal medicine, and geriatrics among others.

In order to establish a uniform standard of care for syncope patients despite the participation of diverse specialities, it is essential that there be a common language. Perhaps most critical in this regard is a clear understanding of what “syncope” is, and why other conditions that cause real or apparent transient loss of consciousness (TLOC) are not classified as “syncope.” Unfortunately, a widely acceptable uniform definition does not currently exist.

The aim of this chapter is to develop a definition of syncope that can be defended and that might prove to be acceptable for use across multiple medical specialties. To this end, the subject is approached through a series of questions and responses.

Is syncope a symptom?

The word “symptom” is generally accepted to mean “a sensation or change in health function experienced by a patient.” This definition certainly applies to “syncope,” but this, of course, is insufficient to fully characterize the term “syncope.”

Syncope is a symptom, but is it the same as TLOC?

The notion of TLOC is certainly included in the etymology of the word “syncope” that is derived from an ancient Greek word, meaning “interrupt.”

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Therefore, it is reasonable to assume that TLOC must be an essential element of "true" syncope.

Determining whether TLOC actually occurred in a given clinical situation may not be easy; it can only be derived from careful evaluation of the history taken from the patient or from eyewitnesses. In the absence of TLOC the diagnosis of syncope should be excluded. However, the concept of TLOC is much broader than just "syncope." TLOC incorporates many other conditions that cause self-limited loss of consciousness but are not due to cerebral hypoperfusion (e.g., epilepsy, concussion, and intoxication). For example, a boxer who is "knocked out" can be considered to have experienced TLOC but cannot be considered as having had syncope. A patient with a toxic coma has TLOC but again cannot be considered as having had syncope. Some patients with psychogenic disorders mimic TLOC but they cannot be considered to have syncope.

Thus, we can at this stage conclude that syncope is a form of TLOC, but the two are not entirely the same. Something more should be added to this definition to fit with what is considered syncope by clinicians.

Syncope is a symptom encompassing a TLOC, but is it spontaneous?

Addition of the word "spontaneous" is necessary to exclude from the field of syncope patients with concussion (e.g., head trauma) or intoxication who require a completely different therapeutic strategy. However, in some cases, "real" syncope can lead to severe head trauma and sometimes it is difficult to determine the real primary cause. Observations by eyewitnesses may be helpful and should be sought.

Syncope is a symptom defined as a transient spontaneous loss of consciousness, but is the onset rapid?

The notion of rapid onset is ambiguous but perhaps it can be agreed that in the case of syncope the time between the onset of premonitory symptoms and the loss of consciousness is relatively brief (i.e., no more than a few dozens of seconds). Intoxications would be expected to take longer, whereas epileptic fits would be indistinguishable, in terms of abruptness of TLOC, from true syncope. Thus, the notion of rapid onset is per se insufficient to exclude some of the causes of TLOC and something more should be added to the syncope definition in order to be more precise.

Syncope is a symptom defined as a transient spontaneous loss of consciousness with a rapid onset, but is it self-limited and complete with usually a prompt recovery?

This addition is crucial. It includes two major concepts. The first is the notion of a "self-limited symptom," which means that patients recover their

consciousness without any external medical interventions, such as prolonged resuscitation maneuvers including electrical cardioversion. In this latter instance, patients should be classified as having had an aborted sudden death, which is not syncope. A second example is of coma, particularly hypoglycemia, which needs a medical intervention to reverse. Thus, the “self-limited” concept excludes some conditions that result in TLOC but are not syncope.

The second concept stated here is the notion of “complete and usually prompt recovery.” This element of the puzzle does not on its own totally discriminate between syncope and some types of TLOC, but it can help. For example, patients with epileptic seizures or coma usually recover slowly and in the case of certain seizures they may remain disoriented for a lengthy period of time.

The “complete and usually prompt recovery” addition tends to restrict the differential diagnosis to what “syncope” is generally considered to be in the broad medical community. But is it really enough? This definition excludes coma, concussion, resuscitated sudden death, “pseudo” TLOC, etc., but not epilepsy, which is not considered to be syncope by most medical practitioners. Something should be added to the definition to exclude this limitation.

Syncope is a symptom defined as a transient spontaneous loss of consciousness with a rapid onset, and a self-limited, complete, and usually prompt recovery in which the underlying mechanism is a transient global cerebral hypoperfusion

This is certainly the most difficult and controversial part of the definition. How could “global cerebral hypoperfusion” be documented in patients with syncope outside of a specially designed laboratory (which is not of course the usual situation)? It is obvious that we do not yet have at our disposal an ambulatory monitor capable of recording cerebral perfusion changes in free-living individuals; consequently, cerebral hypoperfusion can only be suspected based on indirect factors. On the other hand, at this stage of the evolving definition, there remain only two contenders that are consistent with the above-mentioned definition prior to adding the concept that “the underlying mechanism is a transient global cerebral hypoperfusion”; these two are “syncope” or “seizure.”

In the case of epileptic seizure, there is a self-terminated TLOC but the underlying mechanism is abnormal diffuse brain electrical hyperactivity. However, since we do not have a readily deployed long-term ambulatory electroencephalographic monitor, our ability to definitively distinguish between seizure and syncope is limited. Fortunately, in most cases, the clinical picture permits differentiating between syncope (the only TLOC due to global cerebral hypoperfusion) and seizure; in fact, confusion between the two entities is (arguably) relatively rare in everyday practice. The main clinical arguments for each of the two entities are summarized in the guidelines on syncope of the European Society of Cardiology [5].

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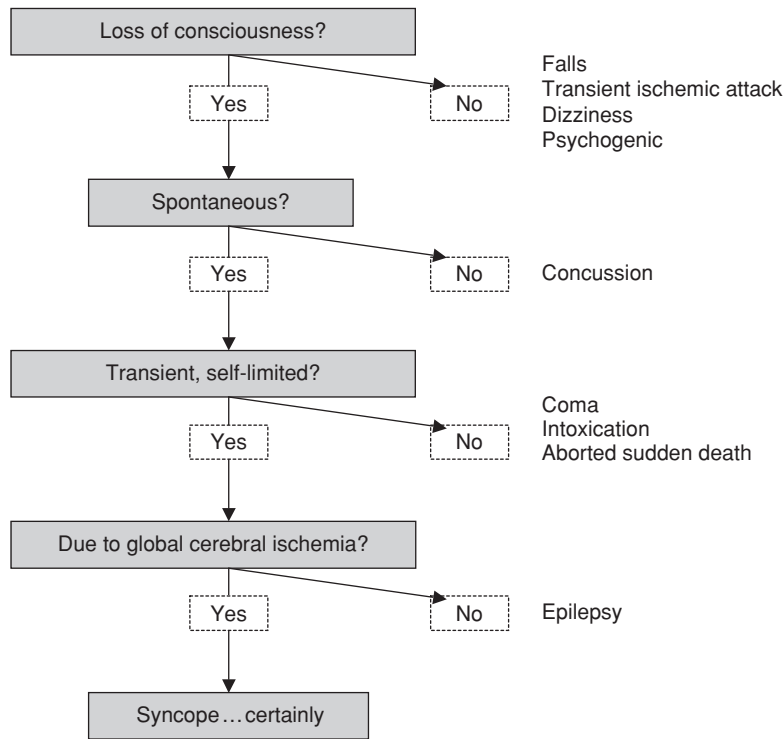


Figure 1.1 Flowchart summarizing the diagnosis strategy from TLOC to syncope.

Syncope is a symptom defined as a transient spontaneous loss of consciousness with a rapid onset, and self-limited, complete, and usually prompt recovery the underlying mechanism of which is a transient global cerebral hypoperfusion. Is this definition adequate?

This definition seems to correspond to the generally accepted view of syncope by the medical community, not just cardiologists. For example, neurologists in their vast majority do not consider that epilepsy is syncope [6].

This last iteration of the definition is very close to the one adopted by the Task Force of the European Society of Cardiology in charge of the guidelines on syncope [5]. The only concept that is not included in the current definition but present in the European Society of Cardiology's definition is "the loss of postural tone." In reality, this latter point is not very helpful, as all patients with TLOC (not exclusively those with syncope) have a "loss of postural tone."

It is evident that new data on pathophysiology of TLOC or new physiologic monitoring devices can modify the proposed definition of syncope, but it seems unlikely that it will be markedly changed. Unfortunately, however, there is a long way to go before there is acceptance of the importance of a careful definition. For example, in the recently published American College of

Cardiology / American Heart Association scientific statement [7], the definition of syncope is just limited to saying, "syncope is a symptom defined as a transient loss of consciousness." Therefore, in the eyes of the official bodies of US cardiology, TLOC and syncope are equivalent. This apparent failure to differentiate between TLOC and syncope clearly promotes long-standing confusion and leads to imprecise thinking with regard to patient management.

Conclusion

To develop an optimum uniform management strategy for any condition, a minimum requirement is clear understanding of what the condition encompasses. In the case of syncope, this level of understanding remains to be achieved. In this chapter the basic elements that characterize the syndrome of syncope have been examined. Based on consideration of these elements, a clinically applicable definition has been proposed. From this assessment it should be evident that syncope is only one of the many causes of TLOC (Figure 1.1) and that, before a final determination of the cause of a patient's symptoms can be offered, it is crucial to first ascertain whether syncope had indeed occurred or whether the apparent loss of consciousness was one of the many other conditions lying within the larger TLOC umbrella.

References

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