Editorials
Is a syncope a syncope?

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Recently, Soteriades et al.[1] provided an update regarding the incidence, specific causes, and prognosis of episodes of transient loss of consciousness (which they term as "syncope") among women and men participating in the Framingham Heart Study from 1971 to 1998. Of 7814 study participants followed for an average of 17 years, 822 reported "syncope". The incidence of a first report of syncope was 6.2 per 1000 person-years. Assuming a constant incidence over time, the authors calculated a 10-year cumulative incidence of syncope of 6%; this means a 42% prevalence of syncope during the life of a person living 70 years. However, the incidence was not constant, but increased more rapidly starting at the age of 70 years. The most frequently identified causes were vasovagal (21%), cardiac (9.5%) and orthostatic (9.4%); for 37% the cause was unknown. The participants with syncope due to any cause, as compared with those who did not have syncope, had a 1.31-fold increased risk for death from any cause, a 1.27-fold increased risk for myocardial infarction or death from coronary heart disease, and a 1.06-fold increased risk for fatal or nonfatal stroke. Persons with cardiac syncope were at an increased risk of death from any cause (hazard ratio of 2.1) and of cardiovascular events (hazard ratio of 2.66), and persons with syncope of unknown cause and neurologic syncope were at an increased risk for death of 1.32 and 1.54 respectively. There was no increased risk of cardiovascular morbidity or mortality associated with vasovagal (including orthostatic and medication-related) syncope.

Although the report by Soteriades et al.[1] provides a generally useful insight into the epidemiology and long-term outcome of syncope or syncope-like symptoms over a mean 17-year period, it is unfortunately burdened by serious limitations that make many conclusions questionable. The most important of these limitations is the uncertainty regarding whether true syncope had occurred and whether the diagnostic evaluation used to establish the basis for syncope was sufficiently definitive.

Establishing the basis for syncope (i.e., determining the "diagnosis") is a prerequisite to advising patients with regard to prognosis, and to developing an effective treatment strategy. However, arriving at the diagnosis can be difficult, and is often marked by the undertaking of costly and often fruitless diagnostic procedures. In view of the above, the development and evaluation of thoughtful, evidence-based (when possible) diagnostic guidelines for the evaluation of syncope patients is highly desirable[2]. However, since syncope is a temporary "state" and not a disease, establishing the true value of such pathways is challenging and will likely require careful assessment of the outcomes in individual patients.

The first step of the diagnostic evaluation is the differentiation of syncope from other conditions in which loss of consciousness may be real or seem to be real, and which thereby mimic "true" syncope. Examples of such conditions include certain types of seizures, sleep disorders, accidents and some psychiatric conditions (Fig. 1, Tables I and II). It is evident that the

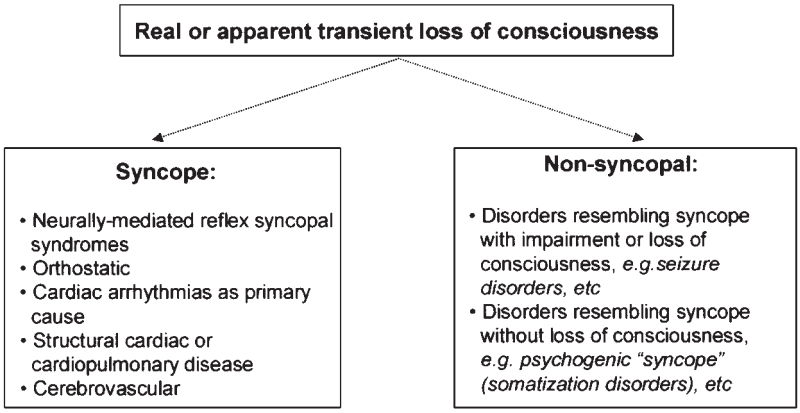

Figure 1. *Classification of transient loss of consciousness according to the guidelines of the European Society of Cardiology[2].*

Table I. Causes of syncope according to the guidelines of the European Society of Cardiology[2].

Neurally-mediated reflex syncopal syndromes
Vasovagal faint (common faint)
Carotid sinus syncope
Situational faint
- Acute hemorrhage
- Cough, sneeze
- Gastrointestinal stimulation (swallow, defecation, visceral pain)
- Micturition (post-micturition)
- Post-exercise
- Others (e.g., brass instrument playing, weight lifting, post-prandial)
Glossopharyngeal and trigeminal neuralgia
Orthostatic
Autonomic failure
- Primary autonomic failure syndromes (e.g., pure autonomic failure, multiple system atrophy, Parkinson's disease with autonomic failure)
- Secondary autonomic failure syndromes (e.g., diabetic neuropathy, amyloid neuropathy)
- Drugs and alcohol
Volume depletion
- Hemorrhage, diarrhea, Addison's disease
Cardiac arrhythmias as primary cause
Sinus node dysfunction (including bradycardia/tachycardia syndrome)
Atrioventricular conduction system disease
Paroxysmal supraventricular and ventricular tachycardias
Inherited syndromes (e.g., long QT syndrome, Brugada syndrome)
Implanted device (pacemaker, defibrillator) malfunction
Drug-induced proarrhythmias
Structural cardiac or cardiopulmonary disease
Cardiac valvular disease
Acute myocardial infarction/ischemia
Obstructive cardiomyopathy
Atrial myxoma
Acute aortic dissection
Pericardial disease/tamponade
Pulmonary embolus/pulmonary hypertension
Cerebrovascular
Vascular steal syndromes

Table II. Causes of nonsyncopal attacks (commonly misdiagnosed as syncope) according to the guidelines of the European Society of Cardiology[2].

Disorders with impairment or loss of consciousness
Metabolic disorders*, including hypoglycemia, hypoxia, hyperventilation with hypocapnia
Epilepsy
Intoxication
Vertebro-basilar transient ischemic attack
Disorders resembling syncope without loss of consciousness
Cataplexy
Drop attacks
Psychogenic "syncope" (somatization disorders)**
Transient ischemic attacks of carotid origin

* disturbance of consciousness probably secondary to metabolic effects on the cerebrovascular tone; ** may also include hysteria, conversion reaction.

Framingham study, perhaps due to the timeframe in which it was initiated (some 45 years ago) did not take this distinction into sufficient consideration.

In the report by Soteriades et al.[1], presumably following the now outmoded definitions in use at the time the Framingham study was initiated, defined "syncope" as a "sudden loss of consciousness associated with the inability to maintain postural tone, followed by spontaneous recovery". Similar definitions are not uncommon even in recent textbooks. However, we doubt that this wide view of syncope fits most physicians' concept of syncope. Based on this definition, many causes of temporary unconsciousness which are not "syncope", such as concussion and various types of epilepsy would be incorporated within the scope of the "incidence and prognosis" of syncope. Indeed, to demonstrate the importance of defining syncope properly, the authors erroneously included both "seizures" and "stroke" as a cause of syncope. Seizures may be considered as a form of transient loss of consciousness but are not usually considered to be syncope. Similarly "stroke" is not

syncope, although the patient may slump to the ground as a result of a stroke.

The more restrictive recent definition of the Task Force on Syncope of the European Society of Cardiology[2] stated that "syncope is a transient, self-limited loss of consciousness, usually leading to falling. The onset of syncope is relatively rapid, and the subsequent recovery is spontaneous, complete and relatively prompt. The underlying mechanism is a transient global cerebral hypoperfusion". Note that the restriction resides in the addition of a specific pathophysiology. Syncope by this definition is part of the larger entity "transient loss of consciousness" which, apart from syncope proper, includes disorders such as epilepsy.

Although the Framingham paper defined "syncope" in the transient loss of consciousness sense, the contents suggest that it was for the most part applied in the more restricted and we believe more appropriate sense[2]. For instance, there were only 48 cases of "syncope due to concussion", a figure which appears to be very low in view of the large number of person-years encompassed by the report. Their exclusion suggests a wish for restriction. The inclusion of seizures, stroke and transient ischemic attacks also points to a transient loss of consciousness-like usage, but again it is doubtful that all such cases were included. Furthermore, in the vast majority of cases transient ischemic attacks are not accompanied by unconsciousness, making it unclear how at that time physicians had interpreted "syncope".

A consequence of the lack of precision in terminology is that most of the prognostic data loose their attraction. It is no longer clear on which grounds patients were actually included. In addition, there appears to be little point in bundling disorders such as seizures and transient ischemic attacks to obtain a group of "neurological syncope", as these point to a wholly different pathophysiology. If inappropriate terminology is used by "experts", then the general physician has every right to be confused.

Further, it becomes difficult to have a high degree of reliance on the study's ability to distinguish among various causes of syncope, since more detail regarding diagnostic methods employed was not provided. Thus, it is unclear how the authors defined syncope of unknown cause. The authors report that it was the largest category among patients with syncope in their cohort, accounting for 37% of patients. Though this prevalence is similar to that reported in several studies performed in the 1980s, in more recent studies, in which more extensive tests were used, the cause of syncope could not be determined in only 17% of cases[2]. This suggests that the group with unknown syncope of the Framingham study was heterogeneous probably including patients with a benign cause of syncope as well as those with an undiagnosed cardiac cause. The consequence is that this group showed an intermediate risk between those of the cardiac and vasovagal groups. The challenge remains how to identify those who are at high risk of death.

In short, in this study the data regarding both the incidence and prognosis of "syncope" suffer from an unclear definition. The problem is by no means restricted to the report by Soteriades et al.[1]; the entire field of syncope and associated conditions suffers from the consistent application of a rational classification system such as that advocated by the European Society of Cardiology Task Force on syncope[2]. We suggest that the term "syncope" be used in a restricted sense[2], including a specific pathophysiology and implying that the cause must be sufficiently certain. A term such as "transient loss of consciousness" should be used when a major category such as syncope or epilepsy cannot be identified. By doing so, "syncope" becomes a clearer entity; this approach helps to avoid confusion, unnecessary investigations and false interpretations of associated risks.

References

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2. Brignole M, Alboni P, Benditt D, et al. Guidelines on management (diagnosis and treatment) of syncope. Eur Heart J 2001; 22: 1256-306.