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Clinical Classification of Syncope

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ABSTRACT

Syncope is a presenting symptom, and in itself is not a diagnosis. An etiology or a mechanism must be sought in all cases. Currently, most clinicians classify syncope on clinical grounds by attempting to ascertain its etiology. They then use this classification to guide further management. Using this approach, reflex syncope is the most common form of syncope, occurring in approximately 60% of syncope presentations. Orthostatic hypotension presents in around 15% with arrhythmic syncope in 10% and structural heart disease as the cause of syncope in 5%; in 10% of patients no diagnosis is made. An alternative classification system uses the mechanism of syncope derived from an implanted ECG loop recorder (ILR). While this approach may be of value for optimizing therapy, it cannot be considered as the primary classification since ILRs are not typically implanted early in the evaluation process of most patients. ILRs are usually placed after "risk stratification" in those deemed not to be at high risk but remain in the uncertain etiology category. Furthermore, there exists, in current ILR technology, lack of ambulatory blood pressure monitoring capability. Thus, vasodilation leading to hypotension, the main trigger of cerebral hypoperfusion other than bradycardia, cannot be detected and is currently unavailable for use in a mechanistic-based classification. Thus, the etiological classification remains the basis for both risk stratification and subsequent clinical management.

The etiological classification of syncope is made by clinicians to provide a basis for risk stratification and subsequent management. One of the major challenges of syncope management is that it involves a very broad range of physicians including cardiologists, neurologists, internists, emergency physicians, pediatricians and geriatricians.[1] Unfortunately, communication between these specialties is not always optimal; the result is often disparate views of the same condition, with adoption of advances in one subspecialty being delayed or overlooked in another.

Risk stratification is proving difficult to achieve at least in part because so many different and sometimes inexperienced physicians are involved at the outset of the diagnostic process, for example, in the emergency department. Excellent results can be achieved when patients are seen by experienced physicians but this is seldom feasible in everyday practice.[2]

Clinical classifications of syncope should provide logical separations of types of presentation, usually on etiological grounds. However, the classification must also offer clinical care pathways beginning with separation of syncope from other causes of transient loss of consciousness (Fig 1), continuing with risk stratification of syncope and progressing through diagnosis of specific causes and management.

Statement of Conflict of Interest: see page 343.

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Abbreviations and Acronyms

AV = atrioventricular

AVB = atrioventricular Block

BP = blood pressure

EEG = electroencephalogram

ESC = European Society of Cardiology

ILR = insertable loop recorder

ISSUE = International Study on Syncope of Uncertain Etiology

MI = myocardial infarction

TLOC = transient loss of consciousness

VPS = Vasovagal Pacemaker Study

VT = ventricular tachycardia

Today, classifications should also permit computer algorithm generation to spread expertise and improve care standards across countries, not merely in academic centers.[3]

Classification

Transient loss of consciousness (TLOC) (Table 1) is the inclusive initial diagnosis requiring assessment. Syncope is defined as a form of TLOC with relatively rapid onset, brief duration and usually rapid full recovery due to transient global cerebral hypoperfusion.

The most common form of syncope is reflex, which implies that it is neuraly mediated (Fig 2). Among those with reflex syncope seen in all age groups, the vasovagal faint is by far the most frequently encountered. Other forms of reflex syncope include carotid sinus syndrome, which is a presentation seen almost exclusively in older people (mostly males). It is thought to have a prevalence in the population of more than 40 per million.[4] Situational syncope is less prevalent than carotid sinus syndrome. There are many phenomena represented in this category. Included are those faints triggered by any of the following: cough, swallow, micturition, defecation, and so on. Some wish to include here syncope due to the sight of blood, post-exercise or the experience of pain but this author prefers to consider these as forms of vasovagal syncope. An expansion of this subclassification can logically be made based on involved body systems (Table 2).

Table 1 – Transient loss of consciousness (TLOC).

Trauma induced	Concussion
Not trauma induced	Syncope Seizure Intoxication Metabolic disorder
Not true LOC	TLOC mimics: Psychogenic Pseudosyncope/ Pseudoseizure Drop attacks Cataplexy

Abbreviation: LOC=loss of consciousness.

In terms of incidence, the second most important category is syncope associated with orthostatic hypotension (OH) or orthostatic syncope. Orthostatic syncope is common in older people and in those taking medical therapy, usually for hypertension. Rare causes of OH are neurological.

The third major syncope category is arrhythmic syncope caused either by bradycardia or by tachycardia. The latter group includes patients with genetically determined channelopathies. The final category is structural heart disease. Structural heart disease is often obstructive in nature, such as:

1. pulmonary embolism or arterial disease leading to pulmonary hypertension on the right side of the heart, or
2. aortic stenosis, mitral valve obstruction including by a left atrial ball thrombus or left atrial myxoma, hypertrophic cardiomyopathy, myocardial infarction or dissection of the aorta primarily affecting the left side of the heart.

All of these conditions reduce cardiac output causing cerebral hypoperfusion. However, many of these same conditions may also be associated with tachyarrhythmias. Other structural (whether they be obvious or molecular–biologic in nature) heart conditions are usually associated with tachyarrhythmias such as in arrhythmogenic (right) ventricular cardiomyopathy and most channelopathies (although conduction system disease and potential for bradyarrhythmia is associated with a few of these). It must also be borne in mind that vasovagal syncope is no less common in these patients with structural heart disease than in the general population and, furthermore, in some cases such as aortic stenosis and hypertrophic cardiomyopathy the tendency to reflex syncope may be enhanced, possibly triggered by mechanoreceptor activation initiated by elevated left ventricular chamber pressure.

There remain about 10% of patients with syncope for whom no diagnosis is made. In part, this difficulty is increased by the fact that when seen by a physician, the event has passed and there may be no evident physical abnormalities. This is particularly the case with reflex

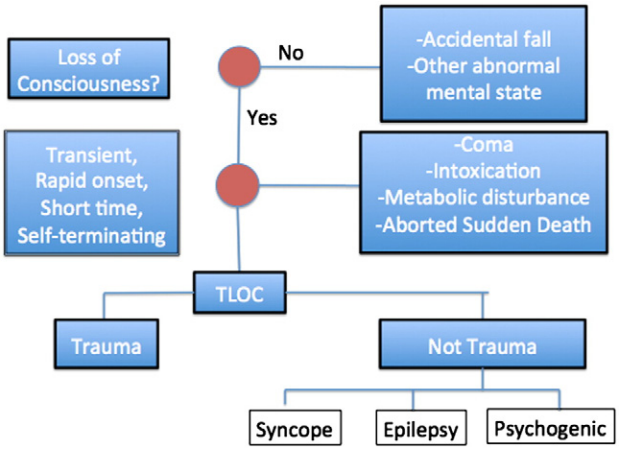

Fig 1 – Syncope and transient loss of consciousness (TLOC). Diagram illustrating an approach to the evaluation of patients presenting with transient loss of consciousness (TLOC). Syncope is a subset of this clinical presentation. Adapted from ESC guidelines.[9]