The evaluation and treatment of syncope

A handbook for clinical practice

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The evaluation and treatment of syncope

A handbook for clinical practice

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Contents

Section three: Specific diagnostic procedures

Section four: Causes of syncope and syncope mimics, and treatment

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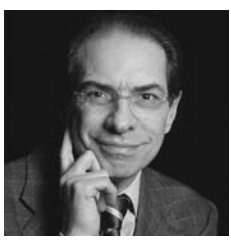

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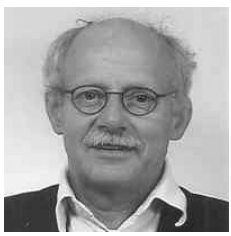

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Introduction

Michele Brignole

This handbook is based on *Guidelines on the Management (Diagnosis and Treatment) of Syncope* published by the European Society of Cardiology in 2001 and updated in 2004. The contributors are primarily comprised of Task Force members but other outstanding authorities in the field have also contributed to specific topics. The purpose was principally to provide a means for disseminating the *Guidelines* in a manner that was readily accessible to medical professionals and could be conveniently utilized in the office, clinic, and emergency department.

The Task Force was constituted in 1999 and the first edition of the *Guidelines* was published in 2001 (*Eur Heart J* 2001; **22**: 1256–1306). An updated edition of the *Guidelines* was published in 2004 (*Eur Heart J* 2004; **25**: 2054–2072 and *Europace* 2004; **6**: 467–537) and was the impetus for developing the second edition of this handbook.

The purpose of the ESC Syncope guidelines is to provide specific recommendations regarding the diagnostic evaluation and management of syncope. The creation of a panel of experts was justified by the fact that in this field, data from the literature are often not definitive, and there has been a lack of standardization regarding nomenclature, diagnostic procedures and their interpretation, and treatment strategies. There are several reasons for this. First, a major issue in the use of diagnostic tests is that syncope is a transient symptom and not a disease. Typically, patients are most often asymptomatic at the time of evaluation. The opportunity to capture a spontaneous event during diagnostic testing is rare. As a result, the diagnostic evaluation must focus on discerning susceptibility to physiological states that could cause loss of consciousness. This type of reasoning leads, of necessity, to uncertainty in establishing a cause. In other words, the causal relationship between a diagnostic abnormality and syncope in a given patient is often presumptive. Second, in the absence of documentation at the time of an event, the establishment of the cause of syncope depends critically on taking an accurate and detailed history. Currently, there is a great deal of variation in how physicians take the history and their knowledge base regarding the crucial information to be sought, and the interpretation of the findings. Third, since documentation of spontaneous syncope events is relatively rare, measurements of test sensitivity are not possible. Essentially there is lack of a 'gold standard' for most of the tests employed for this condition. Consequently, decisions have to be made based on the patient's history and abnormal

findings usually obtained during asymptomatic periods. To overcome the lack of a gold standard, the diagnostic yield of many tests in syncope has been assessed indirectly by evaluation of the reduction of syncopal recurrences after administration of the specific therapy suggested by the results of the test(s) that were diagnostic. In the absence of randomized controlled treatment trials, inferences derived from follow-up observations are inherently suspect.

Given these issues the objectives of the Task Force were to provide:

- criteria for diagnosis of the cause(s) of syncope from history and physical examination;
- guidelines for choosing tests and determining test abnormalities in the further evaluation of syncope;
- advice regarding how to use the results of diagnostic procedures in defining the most probable cause of syncope; and
- recommendations regarding the most appropriate treatment strategy

The methodology for writing the basic Guideline document consisted of literature reviews and consensus development by the panel. The recommendations provided in this book are directly derived from that development process. However, since the goal of the handbook is to provide practicable specific recommendations for diagnosis and management for practicing care givers, recommendations are often provided even when the data from the literature is not definitive. In fact, as remains the case in much of medical practice, most of the recommendations are based on consensus expert opinion.

In order to facilitate reading, the handbook provides neither levels of evidence for every recommendation, nor literature citations for each statement. Key goals for each section are noted at the beginning of each section. Additional reading for each section will be found at the end of each segment of the text. Further, a relatively complete literature source, divided into major interest areas (e.g. pathophysiology, history taking, tilt-table testing) is provided separately at the end of the book. The interested reader is referred to the European Society of Cardiology Guidelines document for statements of levels of evidence and detailed literature citations (you can download this document from the guidelines section of the ESC website: www.escardio.org).

In this handbook the reader will find practical consideration of all the important clinical aspects of syncope:

- What are the diagnostic criteria for causes of syncope?
- What is the preferred approach to the diagnostic work up in various subgroups of patients with syncope?
- How should patients with syncope risk be stratified?
- When should patients with syncope be hospitalized?
- Which treatments are likely to be effective in preventing syncopal recurrences?

In respect to the initial document, the following sections of the *Guidelines* were widely revised in the updated 2004 document and, every effort

has been made in this second edition to provide a parallel updated view of:

- classification of transient loss of consciousness;
- epidemiologic and prognostic considerations;
- initial evaluation and diagnostic flow;
- prolonged ECG monitoring;
- electrophysiological testing;
- ATP test;
- tools for risk stratification (e.g. signal averaged electrocardiogram, T-wave microvolt alternans);
- exercise testing;
- neurological and psychiatric evaluation;
- treatment of neurally mediated (reflex) syncope;
- syncope in the older adult;
- syncope in pediatric patients.

This book attempts to present the Guidelines information in a succinct form. It is directed toward practicing physicians who encounter syncope patients. Thus, we envision it being widely useful. It should be of particular value to practitioners in Emergency Medicine, Primary Care, Internal Medicine, Neurology, Pediatrics, and Cardiology.

Both the ESC Syncope Task Force Guidelines document and this handbook, owe their development to many individuals who planned the tasks, undertook the research, wrote the text, and provided the financial resources to bring these efforts to fruition. In particular, the authors very much appreciate the encouragement and support provided by the leadership and staff of the European Society of Cardiology and specifically the chairmen of the Committee for Practice Guidelines, Professor Jean Pierre Bassand (1998–2000), Professor Werner Klein (2000–2002), and Professor Silvia Priori (2002–2006), and their coordinating secretary, Ms Veronica Dean and her staff.

Section one: Definition, pathophysiology, epidemiology

CHAPTER 1
Syncope: definition, classification, and multiple potential causes

Jean-Jacques Blanc and David G. Benditt

Introduction

The term 'syncope' is derived from an old Greek word meaning 'to cut short' or 'interrupt'. In modern usage, syncope refers to a transient and spontaneously reversible interruption of global cerebral activity resulting in loss of consciousness (and by inference, loss of postural tone). However, in the clinic, most English-speaking patients do not use the word 'syncope'. More commonly they will use terms that are more common in everyday language such as 'fainting', 'blacking out', 'collapse', or 'passing out'. In former days, the term 'swoon' was used, but this is rare today. Additionally, syncope must be considered as part of the differential diagnosis for patients who present with an apparent self-limited 'fall' or 'collapse' (Figure 1.1), even if it is unclear whether they suffered loss of consciousness.

The '*sine qua non*' of syncope (faint) is transient global diminution of blood flow to the brain, such that a disturbance of cerebral function occurs

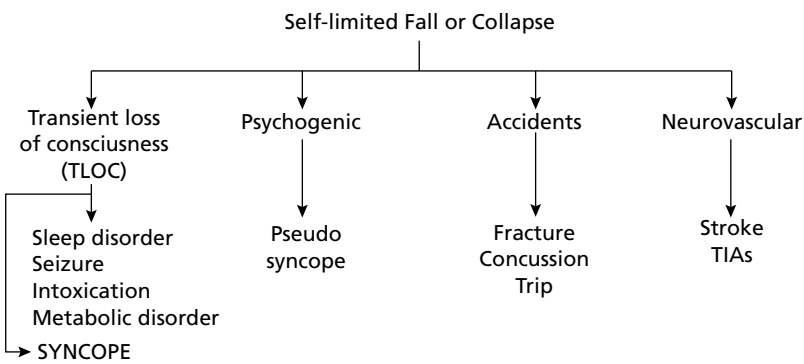

Figure 1.1 Scheme depicting differential diagnostic considerations for patients who present with a self-limited fall or collapse. Syncope is only one element of the differential, but is the primary focus of this book.

Table 1.1 Conditions often mistakenly considered to be syncope.

Dizziness
Vertigo
Drop attacks
Falls
Psychogenic syncope
Transient ischemic attack (TIA)

(see Chapters 3 and 4). As discussed below and elsewhere in this book, this definition eliminates many other conditions that are often mistakenly (even in the literature) considered to be syncope (Table 1.1).

In terms of a practical approach to the clinical problem, physicians are most often first confronted with a patient who has apparently experienced an episode of transient loss of consciousness, or (as in the case of an unexplained 'fall' – suspicion of transient loss of consciousness). In this scenario, one should not immediately assume that the event was 'syncope', since it is not the only explanation for such symptoms. The broader term 'Transient Loss of Consciousness (TLOC)' is a better starting point, since it has a much more diverse set of etiologies and thereby requires the physician to consider a wider range of possibilities. Only if TLOC is due to transient inadequacy of global cerebral blood flow should the differential diagnosis begin to focus on those conditions typically responsible for 'syncope'.

Goals

This chapter provides an introduction to the concept of syncope as a symptom with many potential causes. Indeed, multiple possibilities frequently coexist in the same patient, thereby complicating the diagnostic dilemma. Specifically, the objectives of this section are to:

- define syncope;
- provide a classification of the principal causes of syncope in a manner consistent with the most recent ESC Syncope Task Force guidelines; and
- highlight the possibility that multiple potential contributing factors need to be considered when evaluating syncope patients.

Definition

Syncope is a symptom defined as a transient, self-limited loss of consciousness, and as a consequence the concomitant loss of voluntary muscle tone. The underlying mechanism is transient global cerebral hypoperfusion. The onset of syncope is relatively rapid and the subsequent recovery is by definition

spontaneous, complete, and usually prompt. Residual symptoms (e.g. fatigue) may, however, persist for hours or longer in certain types of faints.

Elements of the definition of syncope

The definition of syncope incorporates five main components.

1 *Loss of consciousness.* This is a critical feature that has to be derived from the history taken from the patient or from those who witnessed the episode(s). If the history convincingly points to there not having been loss of consciousness associated with the patient's 'spell', the diagnosis of syncope is excluded – it is something else (for examples see Table 1.1). Beware, however, that the victim may deny (possibly due to memory deficit or embarrassment) having experienced loss of consciousness, and only careful interrogation of witnesses may determine the real state-of-affairs.

2 *Loss of voluntary muscle tone.* Loss of voluntary muscle control is inherent with loss of consciousness. Therefore, if standing, the fainter falls down; if seated he or she slumps over.

3 *Onset is relatively rapid.* As a rule, the onset of syncope is rapid, being no more than 10 to 20 s after onset of premonitory symptoms (if there are any such symptoms). Faints may be associated with any of a variety of warning symptoms (or none at all), and the nature of these (see Chapters 7 and 8 discussing the initial evaluation and medical history taking) may provide important clues as to the cause of the symptoms. On the other hand, many fainters either do not experience or are unaware of any premonitory symptoms. This lack of warning seems to be particularly prevalent in older individuals.

4 *Recovery is spontaneous, complete, and usually prompt.* This aspect of the definition excludes a number of conditions that may result in loss of consciousness, but which in fact do not reverse themselves to normal in the absence of medical intervention. Examples of such conditions are coma (e.g. hypoglycemia), intoxicated states (alcohol, narcotics, other drugs), stroke, or resuscitated 'sudden death' syndrome. Although states of intoxication usually reverse spontaneously, the relatively long time frame of the recovery distinguishes them from true syncope.

5 *Underlying mechanism is transient global cerebral hypoperfusion.* This element of pathophysiology differentiates 'true syncope' from loss of consciousness due to trauma (e.g. concussion) or seizures (epilepsy). Both trauma and epilepsy may lead to loss of consciousness with complete and spontaneous recovery, but their origins are not inadequacy of cerebral perfusion. With regard to epilepsy (see also Chapters 2, 17, and 23), perhaps the aspect that causes the most confusion is abnormal motor activity. In syncope, it is not uncommon for patients to exhibit jerky movements of the arms and legs for a brief period of time; nonexpert bystanders may incorrectly interpret these movements as a 'seizure' or a 'fit'. However, the jerky movements during a faint differ from those accompanying a grand mal epileptic seizure in several ways. They are

of shorter duration, they tend to occur after the loss of consciousness has set in rather than before, and they are jerkier and do not have the 'tonic–clonic' features of a true grand mal epileptic seizure.

Causes of syncope: classification and single versus multiple etiologies

Later chapters in this book provide a comprehensive discussion of the most important causes of syncope and their appropriate investigation. Only a brief

Table 1.2 Syncope classification.

Neurally mediated reflex syncopal syndromes
Vasovagal (common) faint
Carotid sinus syndrome
Situational faint
Acute hemorrhage
Cough, sneeze
Gastrointestinal stimulation (swallow, defecation, visceral pain)
Micturition (postmicturition)
Postexercise
Other (e.g. brass instrument playing, weightlifting, postprandial)
Glossopharyngeal and trigeminal neuralgia
Orthostatic
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)
Volume depletion
Hemorrhage, diarrhea, Addison's disease
Cardiac arrhythmias as primary cause
Sinus node dysfunction (including bradycardia/tachycardia syndrome)
AV conduction system disease
Paroxysmal supraventricular and ventricular tachycardias
Inherited syndromes (e.g. long QT syndrome, Brugada syndrome, short QT, arrhythmogenic dysplasia)
Implanted device (pacemaker, ICD) 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

overview is provided here. Specifically, we provide a classification (Table 1.2) of the causes of syncope beginning with the most frequently encountered conditions, the neurally mediated reflex faints. However, it should be borne in mind that even after a thorough assessment, it may not be possible to assign a single cause for fainting. Often, patients have multiple comorbidities and as a consequence they may have several equally probable causes of fainting. Thus, individuals with severe heart disease may faint due to transient tachyarrhythmias, high-grade atrioventricular (AV) block, or even as a consequence of being excessively medicated. Thus, the physician must not be lured into the trap of accepting an observed abnormality as either the certain cause or the sole cause of fainting in a given individual.

Neurally mediated reflex faints are of several different types, but the best known is the common or vasovagal faint. This is the so-called swoon often seen in films (usually triggered in the movies by a painful or emotionally upsetting event). The vasovagal faint can occur in both healthy persons as well as those with health problems; it is not indicative of nervous system disease and should not typically initiate neurologic studies. The patient experiencing a vasovagal type of reflex faint is very likely to feel nauseated and sweaty before fainting, and often appears pale and feels clammy. After the faint, they often feel tired; this sensation may last for hours or days. Other reflex faints include carotid sinus syndrome, or faints triggered by micturition or defecation. Coughing, swallowing, laughing, or even forcibly blowing into a wind instrument may also trigger a faint, presumably on a reflex basis.

Orthostatic (postural) faints are also common, and most often are associated with movement from lying or sitting to a standing position. Many healthy individuals experience a minor form of this faint when they need to support themselves momentarily as they stand up. However, the most dramatic postural faints occur in older frail individuals, those who have underlying medical problems (e.g. diabetes, certain nervous system diseases), or persons who are dehydrated from hot environments or inadequate fluid intake. Certain commonly prescribed medications such as diuretics, beta-adrenergic blockers, antihypertensives, or vasodilators (e.g. nitroglycerin) predispose to postural faints.

Cardiac arrhythmias may cause faints if the heart rate is too slow or too fast. Occasionally, such faints occur in otherwise healthy people such as at the onset of a paroxysmal supraventricular tachycardia (SVT) episode. However, individuals with underlying heart disease (e.g. previous myocardial infarction, valvular heart disease) are at greater risk. In either case the faint tends to occur at the onset of the rhythm problem, before compensatory vasoconstriction has a chance to respond and support the central systemic pressure. Faints may also occur when a rapid abnormal rhythm stops suddenly, and a pause ensues before the normal heart rhythm takes over again. If this is for more than 5 s,

Table 1.3 Causes of 'spells' commonly misdiagnosed as syncope.

Disorders with impairment or loss of consciousness
Metabolic disorders, including hypoglycemia, hypoxia, hyperventilation with hypocapnia
Epilepsy
Intoxication (drugs, alcohol)
Vertebrobasilar transient ischemic attack
Disorders resembling syncope without loss of consciousness
Cataplexy
Drop attacks
Psychogenic syncope (somatization disorders)
Transient ischemic attacks of carotid artery origin

the patient can experience lightheadedness or a faint (especially if they are in an upright position at the time).

Structural cardiopulmonary diseases are relatively infrequent causes of faints. The most common cause in this category is fainting associated with an acute myocardial infarction or ischemic event. The faint in this case is primarily caused by an abnormal nervous system reaction similar to the reflex faints. In general, faints caused by structural disease of the heart or blood vessels are particularly important to recognize as they are warning of potentially life-threatening conditions.

Cerebrovascular disease is rarely the cause of a faint. Perhaps subclavian steal is the best example in this class, but it is extremely uncommon. In the absence of clear-cut fixed or transient localizing neurologic signs during physical examination, cerebrovascular disease as a cause of syncope is unlikely. As a rule, this category should be considered only after all other 'causes' have been eliminated.

As noted earlier, certain clinical presentations are unfortunately often mislabeled as 'syncope' (Table 1.1). In other situations, however, the medical history mimics that of a faint (see also Chapter 23), and the most important of these are worth noting here primarily because they are commonly confused with 'true' faints (Table 1.3). As a consequence of this confusion (often aggravated by the manner in which even well-known investigators present their findings in the literature), the process needed to arrive at the correct etiologic diagnosis is impeded. The most common conditions in this category include: seizures, sleep disturbances, accidental falls, and some psychiatric conditions (e.g. anxiety attacks, severe hyperventilation and hysterical reactions). Inner ear problems causing dizziness (vertigo) are also frequently mislabeled as faints. Neurologic and metabolic disturbances (such as diabetes) are rarely the cause of true fainting.

Summary

The methods recommended to determine the most probable cause of syncope and ascertain which treatment direction is most appropriate are reviewed in subsequent chapters of this book. Here, we have attempted to provide an introductory overview, so that the reader will better appreciate the value of understanding the pathophysiology, the differential diagnosis, and the need for a thoughtful evaluation strategy. In the end, however, it is important to bear in mind that neurally mediated reflex syncope, orthostatic syncope, and cardiac arrhythmias account for approximately 60 to 70% of the recognized causes of syncope. Further, in 20% of patients the cause of syncope may remain unknown in spite of an extensive and well-planned evaluation. In some of this latter 20% there may be multiple possible causes and distinguishing among them in an effort to find a 'sole' cause may be both impossible and incorrect.

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CHAPTER 2
What is syncope and what is not syncope: the importance of definitions

J. Gert van Dijk and Adam P. Fitzpatrick

Introduction

The literature surrounding syncope is extensive. However, its interpretation is undermined by the most basic of deficiencies, namely a consistent operational definition of the term syncope.

Goals

- Define syncope and review the rationale for the definition accepted by the European Society of Cardiology (ESC) Syncope Task Force.
- Illustrate that the use of imprecise definitions impairs understanding of the clinical problem.

The definition and its understanding

The European Society of Cardiology Task Force on Syncope defines syncope as:

> 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 usually prompt. The underlying mechanism is a transient global cerebral hypoperfusion.

The first part of the definition is wholly clinical in nature, while the last sentence describes an underlying cause. This distinction may be felt to be unwise because often neither the cause nor the mechanism of transient of loss of consciousness (TLOC) is clear. How useful is a definition of which an important part cannot always be assessed? Why was it necessary to include a nonclinical element?

The simple answer is that within the concept of syncope it was essential to include some disorders and also to exclude others that most clinicians would never label as syncope. A vasovagal faint should be included as should a temporary loss of consciousness due to cardiac arrhythmia, or to orthostatic hypotension, because all these phenomena have in common that TLOC is due to global lack of blood flow to the brain. However, TLOC due to a subarachnoid hemorrhage, an epileptic seizure, or a brain concussion should clearly