The evaluation and treatment of syncope
A handbook for clinical practice
THE ESC EDUCATION SERIES

The evaluation and treatment of syncope
A handbook for clinical practice
Second edition

A publication based on the Guidelines on Management (diagnosis and treatment) of Syncope by the European Society of Cardiology (www.escardio.org/knowledge/guidelines)

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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 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...
Table 1.1 Conditions often mistakenly considered to be syncope.

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness</td>
</tr>
<tr>
<td>Vertigo</td>
</tr>
<tr>
<td>Drop attacks</td>
</tr>
<tr>
<td>Falls</td>
</tr>
<tr>
<td>Psychogenic syncope</td>
</tr>
<tr>
<td>Transient ischemic attack (TIA)</td>
</tr>
</tbody>
</table>

(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
Syncope: definition, classification, causes

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.

<table>
<thead>
<tr>
<th>Disorders with impairment or loss of consciousness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic disorders, including hypoglycemia, hypoxia, hyperventilation with hypocapnia</td>
</tr>
<tr>
<td>Epilepsy</td>
</tr>
<tr>
<td>Intoxication (drugs, alcohol)</td>
</tr>
<tr>
<td>Vertebrobasilar transient ischemic attack</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disorders resembling syncope without loss of consciousness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cataplexy</td>
</tr>
<tr>
<td>Drop attacks</td>
</tr>
<tr>
<td>Psychogenic syncope (somatization disorders)</td>
</tr>
<tr>
<td>Transient ischemic attacks of carotid artery origin</td>
</tr>
</tbody>
</table>

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.

Additional reading

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 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
be excluded from this definition of syncope. These latter disorders may also present as TLOC, but often otherwise differ in many clinical features and in their underlying pathogenesis. For example, loss of consciousness in epilepsy is due to inappropriate firing of cortical neurons, whereas concussion is less well characterized but is not due to inadequate perfusion.

**Various definitions of syncope**

In the past, many researchers and textbooks used definitions along the following lines: syncope is ‘a temporary self-limited loss of consciousness associated with loss of postural tone’. Such a definition is much broader than the one used in this book. Taken literally, such a broad definition encompasses both the disorders that were felt to be syncopal, but also the ones that most physicians would not now label as syncope. In fact, it is better used as a good definition of TLOC.

Readers may feel that these nomenclature distinctions are solely an academic question. Two examples are given in which a lack of precision was harmful:

1. A paper describing the prognosis associated with syncope was published in the *New England Journal of Medicine* in 2002. The report, part of the Framingham study, was retrospective in nature and spanned a period of 17 years. Syncope was defined as ‘a sudden loss of consciousness associated with the inability to maintain postural tone, followed by spontaneous recovery’. One may wonder whether the physicians who diagnosed syncope over this period had done so according to this definition. If so, the study should result in a sizable number of epileptic seizures, concussion cases, etc. If, in contrast, physicians had applied a concept of syncope based on cerebral perfusion, such disorders (i.e. concussions, seizures) should not show up at all, meaning that the study was about something different than that which was implied by the definition provided in the manuscript itself.

   Syncope was subclassified into various groups. For the present purpose only the group of ‘neurological syncope’ is relevant. In it, 47 cases of concussion are encountered, which seems a low number in view of the large number of person-years studied. These cases were not analyzed in the paper. The number of epileptic seizures also appears too low for the duration and size of the study. What is apparent from this paper is that some physicians had used a broad definition as stated in the paper, but most relied on another concept. The group of ‘neurological syncope’ also included cases of TIA’s and stroke. This is most surprising, as TIA’s almost never cause unconsciousness, and strokes are by definition not temporary. It is therefore apparent that many of the cases of syncope included in this study were cases of TLOC, and in this study syncope included TLOC with a wide variety of underlying causes, certainly not just cerebral hypoperfusion. The relative numbers of syncope due to an epileptic seizure, a concussion, a TIA, or a stroke must depend on the apparently rather individual concept of syncope used by individuals who entered patient data. What we can say with some confidence, is that different physicians used the
term ‘syncope’ in different ways, and the clinical features, investigations and outcomes of syncope in this paper cannot be quoted with confidence.

A relatively recent manuscript published in the British Medical Journal defined syncope as ‘a transient loss of consciousness, with loss of posture (that is, falling)’. This is also a broad definition, and again fits with a definition of TLOC, not of syncope. The authors divided syncope into cardiac, metabolic, psychiatric, and neurological groups. At first glance, this division appears to do justice to the definition, as consciousness may definitely be lost though a variety of means. But psychiatric mechanisms can only do so through circuitous mechanisms: some people can voluntarily evoke syncope through a Valsalva-like maneuver, but the cause of the unconsciousness is cerebral hypoperfusion, that is, it is true syncope. Somatization disorders and hyperventilation were stated as psychiatric causes of syncope. In the first, however, the brain keeps functioning so patients may look unconscious but are not. As for hyperventilation, it appears to be almost impossible to lose consciousness by hyperventilation (in subjects with autonomic failure it may worsen orthostatic hypotension, but if this contributes to unconsciousness, this too is true syncope). The group of ‘neurological syncope’ again includes epileptic seizures and TIA’s, as well as normal pressure hydrocephalus (the latter two conditions would rarely be included even in the differential diagnosis of TLOC). ‘Cardiac syncope’ contained the four groups of syncope recognized by the ESC Syncope Task Force as true syncope. However, the ESC did not regard ‘orthostatic syncope’ and ‘neurally mediated (i.e. reflex)’ syncope as cardiac in origin.

These two examples, illustrate how an imprecise concept of syncope, that does not include a definition of the pathogenesis, leads to the inclusion of disorders in which consciousness merely appears to have been lost, but is not actually lost (i.e. it is not true TLOC), disorders where there is TLOC but it is not due to transient global cerebral hypoperfusion, or some conditions in which consciousness does not even look lost, (i.e., not even apparent TLOC). Imprecise usage such as exemplified here cannot help understanding, and runs the risk of complicating in the physicians’ minds the most important distinction, that is the difference between syncope and epilepsy. Similar criticisms can be directed at many other published works, whose authors cannot be blamed for doing so in such a confused situation. However, henceforth it does mean that papers on syncope should be more precise in their nomenclature and should also be subjected to more critical reading. Readers must ask: was this really syncope?

Consequences of confusing TLOC and syncope

The inclusion of a pathogenesis in the definition of syncope has two important consequences. The first is that additional clinical features, not included in the definition, are needed to conclude syncope. An example is the presence of nausea and pallor in a young girl in whom TLOC was triggered by having
her ears pierced; this points to neurally mediated reflex syncope; another example concerns a man fainting repeatedly after reaching the top of a flight of stairs, suggesting either autonomic failure or perhaps a cardiac origin, such as ischemia or rate-dependent block.

The second consequence is that the word syncope should not be used when there is insufficient evidence that TLOC was due to transient global cerebral hypoperfusion. In such cases, epilepsy cannot be discounted, and a term is needed to describe this situation. TLOC can be used to describe this condition. As said, its definition is akin to that of syncope, but with the cause removed and one addition. This important addition is that the term TLOC should not be used when the transient loss of consciousness is due to traumatic head injury; concussion should not cause much confusion with either syncope or epilepsy. Note that both the broad and narrow definitions referred to above are recognized in this view. Both are not just useful for clinical and scientific purposes, but also critically important for an accurate diagnosis and prescription of the correct treatment to prevent recurrences.

A word on epilepsy

Epilepsy is discussed elsewhere in this book (see Chapters 17 and 23). Nevertheless, a few remarks may be made regarding terms encountered when dealing with epilepsy. ‘Seizures’ are usually understood to mean ‘epileptic attacks’, but the International League against Epilepsy does not in fact firmly restrict the term to epilepsy. Every kind of attack associated with abnormal movement may be called a ‘seizure’, and sometimes is. ‘Reflex anoxic seizures’ is used to describe attacks in young children who are startled, often by a bump on the head, cry, lose consciousness, and who then exhibit jerking movements. The word seizures here does not refer to epileptic attacks, but to the movements that may accompany many types of TLOC. These attacks are examples of reflex syncope (equivalent to neurally mediated reflex syncope) of the emotionally induced or vasovagal type (in infants this often leads to asystole). The attacks are also described as pallid breath-holding spells, a term that also does not convey that this is syncope (in the cyanotic type prolonged expiratory apnea does appear to play a role, but neither term implies voluntary breath holding). The jerking movements that may accompany syncope, and that should be distinguished from epileptic clonic movements, are sometimes called convulsions giving rise to convulsive syncope. Again, this does not imply an epileptic nature, since ‘syncope’ with or without convulsions means that TLOC was due to transient cerebral hypoperfusion.

In the United States, the term ‘seizure disorder’ appears to be preferred over epilepsy. Apparently, the word seizure is restricted to epilepsy for some, while it can also include convulsive syncope for others. It would be preferable to restrict it to one sense, and the one that is preferable is the most widely used one: seizures mean an epileptic attack and nothing else.
Chapter 2

Sometimes the term ‘non-epileptic attack disorder (NEAD)’ is used to indicate attacks that look like epilepsy but are not, and that are of a psychogenic nature. Syncope is obviously nonepileptic, but definitely not psychogenic. Moreover, there are also psychogenic attacks that resemble syncope (sometimes termed psychogenic pseudosyncope) more than they resemble epilepsy. Those working with syncope might choose to label this condition as ‘non-syncopal attack disorder’ but this would cause the same problems as NEAD. It might be better to label such attacks as psychogenic instead of emphasizing what they are not. Alternatives are pseudosyncope and pseudoseizures.

A wider framework

There is one further definition required, and that is for patients who have abrupt loss of postural tone, but in whom it is not certain whether consciousness was lost or not. Elderly patients may be completely unaware after the fact that they had temporarily lost consciousness. Such cases may present with falls of an undermined nature. Sometimes, the fall is clearly attributable to an external cause, such as a trip on an uneven floor and sometimes the cause appeared to reside in the patient, where the fall was actually precipitated by syncope. When consciousness was lost, one may conclude TLOC and then try to determine its cause. But when this is not the case, or the cause is uncertain, a term is needed that leaves all possibilities open, to stop physicians from jumping to conclusions, and forgetting the full range of possible causes. ‘Collapse of unknown cause’ may serve this purpose, but its use will depend on circumstances beyond those described in this book. This subject falls outside the scope of the ESC Task Force on syncope, and is discussed here only to broaden interest.

Summary

The syncope literature is extensive, but unfortunately it is characterized by substantial variability in the definition of what syncope really is. On first pass, this may seem to be an arcane academic concern, but as has been demonstrated in this chapter, nomenclature problems introduce real operational difficulties. The reader is encouraged to think of syncope in terms of the ESC Syncope Task Force definition provided above; specifically that subset of TLOC in which the pathophysiology is self-limited diminution of cerebral perfusion.

Additional reading
