Chapter

CARDIOGENIC SYNCOPE

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ABSTRACT

Syncope is a transient, brief and sudden loss of consciousness produced by a lessening of the flow of blood to the brain and therefore oxygen deprivation. It can be presented from different ways. Similarly, can be due to a great variety of causes, although the most frequent causes to produce an episode of syncope on any person are: the stroke (embolism and / or brain hemorrhage) and cardiovascular disorders. In many occasions, both are interlinked.

Patients suffer a complete loss of consciousness that may be associated to involuntary movements of the limbs. This may lead to confusion in diagnosis between epilepsy and syncope, while the treatment is quite different for both pathological entities. Unfortunately this confusion is too frequent, so that many cases are treated as epilepsy when in fact is another origin of syncope. Hence, the differential diagnosis should be essential in such events.

All syncope episode gives as result an inexplicable fall of the person who suffers from it.

The most important tool for all physicians for a first diagnostic impression is the patient medical history along with the eyewitness recollection of the incident, if possible. Physicians should be aware of the possibility that a patient or caregiver may inadvertently misinterpret the symptoms when trying to describe to them.

Today is well known that approximately 10% of patients diagnose with epilepsy are syncope sufferers. More than 25% of them have an origin in cardiac abnormalities (this percentage is 45-50% higher when the patient is 60 years or older, regardless of sex).

Any patient with several episodes of syncope or loss of consciousness should be subjected to a full investigation. This should include at least, a blood chemistry analysis, an electroencephalogram (EEF) and an electrocardiogram (ECG), which are fundamental components for evaluation. A normal ECG is a good prognostic feature in most cases.

From the cardiac viewpoint, strictly, the events that can raise concern are:

- Cardiac ischemia and myocardial infarction.
Any electrical disorder from the heart:

- Bradycardia: <40 palpitations per minute (with exception of athletes or pauses of 3 seconds or more).
- Atrioventricular block (2º and 3º degrees, especially).
- Complete left bundle branch block.
- Rapid supraventricular tachycardia (greater than 150 bpm).
- Ventricular tachycardia.
- Disorders from electrical cardiac systole
  3º Recording of some ECG patterns like a shortened of “PQ and QT –intervals” together in a same ECG record (Breijo’s pattern).
  4º Brugada Syndrome.
  5º Other disorders of electrical cardiac conduction.

An ECG without signs suggestive for cardiac pathology should make us think that the syncope is not severe and should be seen as reassuring.

**INTRODUCTION**

There are two distinct reasons for the evaluation of patients with syncope: one is to identify the precise cause that allows for an effective treatment that be specific for the production mechanism of syncope, the other is the identification of specific risk to the patient, which often depends from underlying disease rather than the mechanism itself syncope [1, 2, 3]. The patient’s backgrounds are fundamental to clinicians in order to avoid confusing the two concepts [4,5].

The main objective of this chapter is to develop a summary document concerned to proceed not just to cardiologists, but also to all physicians who are interested in this field. The most relevant actions to achieve these objectives are listed below:

- An update from the classification of syncope in a broader framework on transient loss of consciousness.
- New epidemiological data.
- A new focused diagnostic approach on risk stratification for sudden cardiac death and cardiovascular events after from the initial assessment, including recommendations for treating patients with syncope of unknown risk.
- A main emphasis for to achieve a better diagnostic strategy based on long-term monitoring, rather than the conventional approach based in laboratory tests only.

As we said in the "abstract", the syncope is a transient and brief loss of consciousness due to a transient global cerebral hypoperfusion and is characterized by a sudden onset, a short duration and a complete spontaneous recovery. In some forms, the syncope can have a prodromal period in which various symptoms (such as auras, nausea, sweating, weakness and visual disturbances) warn that syncope is imminent.

Any sudden and full cessation in cerebral blood flow of just 6-8 seconds has been demonstrated like a sufficient time to cause a complete loss of consciousness.
CLASSIFICATION AND PATHOPHYSIOLOGY OF SYNCOPE

The common feature of all pathophysiological mechanisms of syncope is a fall in systemic blood pressure, with an overall reduction in cerebral blood flow. Experience, with Tilt table, has shown that a decrease in systolic blood pressure of 60 mm Hg or below is associated with syncope.

Systemic arterial pressure is determined by cardiac output and full peripheral vascular resistance and a fall in either of these two factors can cause syncope, but often there is a combination of both mechanisms, although their relative contributions can vary considerably. The low or inappropriate peripheral resistance may be due to an inappropriate reflex activity. Other causes of low or inadequate peripheral resistance are functional and structural failure from autonomic nervous system induced by drugs, primary and/or secondary. In the autonomic nervous system dysfunction, the vasomotor sympathetic pathways are unable to increase total peripheral vascular resistance in response to the upright position. When there is a gravitational stress, combined with the vasomotor sympathetic dysfunction, venous blood accumulation under the diaphragm, causes a decrease in venous return, in cardiac output and, consequently to a reduction in cerebral blood flow with syncopal episode appearance [6, 7,8].

Table 1. Classification of Syncope

<table>
<thead>
<tr>
<th>Reflection (neuromediated) Vasovagal:</th>
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<tr>
<td>- Mediated by emotional distress, fear, pain, instrumentation, blood phobia</td>
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<tr>
<td>- Mediated by orthostatic stress</td>
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<tr>
<td>Facts:</td>
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<tr>
<td>- Coughing, sneezing</td>
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<td>- gastrointestinal stimulation (swallow, defecation, visceral pain)</td>
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<td>- micturition (post-micturition)</td>
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<td>- After exercise</td>
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<tr>
<td>- Postprandial</td>
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<tr>
<td>- Other (eg., laughing, playing wind instruments, lifting weights)</td>
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<tr>
<td>Carotid sinus syncope:</td>
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<tr>
<td>Atypical forms (without apparent triggers and / or presentation atypical)</td>
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<td>Syncope due to orthostatic hypotension</td>
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<tr>
<td>Primary autonomic dysfunction:</td>
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<tr>
<td>- pure primary autonomic dysfunction, multiple system atrophy, Parkinson's disease with autonomic dysfunction, dementia with Lewy corps</td>
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<tr>
<td>Secondary autonomic dysfunction:</td>
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<tr>
<td>- Diabetes, amyloidosis, uremia, spinal cord injury</td>
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<tr>
<td>Drug-induced orthostatic hypotension:</td>
</tr>
<tr>
<td>- Alcohol, vasodilators, diuretics, phenothiazines, antidepressants</td>
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<tr>
<td>Volume depletion:</td>
</tr>
<tr>
<td>- bleeding, diarrhea, vomiting, etc.</td>
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<tr>
<td>Cardiac syncope</td>
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Table 2. Clinical situations incorrectly diagnosed as syncope

<table>
<thead>
<tr>
<th>Disorders with partial or complete loss of consciousness but not global cerebral hypoperfusion:</th>
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<tr>
<td>- Epilepsy</td>
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<td>- Metabolic disorders, including hypoglycemia, hypoxia, hyperventilation with hypocapnia</td>
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<tr>
<td>- Poisoning</td>
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<tr>
<td>- Vertebro-basilar transient ischemic attack</td>
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<tr>
<th>Disorders with impaired consciousness:</th>
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<tr>
<td>- Cataplexy</td>
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<tr>
<td>- Drop Attacks</td>
</tr>
<tr>
<td>- Falls</td>
</tr>
<tr>
<td>- Functional (psychogenic pseudo-syncope).</td>
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<tr>
<td>- Transient ischemic attack of carotid origin.</td>
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</table>

The causes of transient low cardiac output are threefold. The first is a reflex bradycardia, known as reflex cardio-inhibitory syncope.

The second is from cardiovascular causes, due to structural or non-structural disease such as arrhythmias, arterial hypertension and pulmonary embolism. The third is an inadequate venous return, due to volume depletion or venous pooling.

The ending is three mechanisms: reflex syncope, syncope secondary to orthostatic hypotension and cardiovascular syncope.

In these cases, the differential diagnosis is usually obvious, but sometimes it can be difficult due to the absence of clinical history, recurrent circumstances or confusion about the definition of syncope. This distinction is important for all clinicians who facing to patients with sudden loss of consciousness (real or perceived) that may be due to causes not related to a decrease in the global cerebral blood flow, such as epileptic attacks or a conversion reaction.

**CAUSES OF CARDIOGENIC SYNCOPE**

The most frequent syncope’s episodes from Cardiogenic cause are:

**NO STRUCTURAL HEART DISEASE.**

The Arrhythmia (is the leading cause):

**Bradycardia:**

- Sinus node dysfunction (including the syndrome bradycardia / tachycardia).
- Disease of the atroventricular conduction system.
- Dysfunction of an implantable device

**Tachycardia:**

- Supraventricular.
- Ventricular (idiopathic or secondary to structural heart disease).
Any channelopathy type as well as any abnormality from electrical cardiac systole.
Bradyarrhythmia and tachyarrhythmia, drug-induced
STRUCTURAL HEART DISEASE.
Cardiac valvular disease.
Myocardial infarction / ischemia.
Hypertrophic cardiomyopathy.
Cardiac masses (atrial myxoma, tumors, etc.).
Pericardial disease / tampon.
Congenital anomalies of the coronary arteries.
Prosthetic valve dysfunction.
Other: Pulmonary embolism. Acute aortic dissection. Pulmonary hypertension.

1. Arrhythmias

Any type of cardiac arrhythmia accompanied by important haemodynamic deterioration is capable of cause syncopal episodes. Arrhythmias are the most common causes for cardiac syncope. It produces a haemodynamic deterioration, which can lead to a critical reduction in cardiac output and in cerebral blood flow. Nevertheless, the syncope often has multiple contributing factors, such as heart rate, type of arrhythmia (supraventricular or ventricular), left ventricular function and the adequacy of vascular compensation. This latter includes the neural baroreceptor reflexes and responses to orthostatic hypotension induced by arrhythmia.

Independently of these contributing factors, when the arrhythmia is the primary cause of syncope should be treated specifically.

In the intrinsic sinus node dysfunction, the sinus node is damaged, due to an abnormal automaticity or sinoatrial conduction abnormalities. In this situation, the syncope is caused by a prolonged pauses caused by sinus arrest or sinoatrial block and the failure of the escape mechanisms. These pauses occur often to the final of episodes of atrial tachyarrhythmia (tachycardia-bradycardia syndrome).

The most serious forms of atrioventricular block (AVB) acquired (Mobitz II block, "progressive blocking” and complete atrioventricular block) are related more closely to syncope. In these cases, heart rate may become dependent on pacemakers or subsidiary pacemaker (often unreliable). Syncope occurs, because the pause before these pacemakers begin to shoot is long [9,10]. Besides, these sites act as subsidiary pacemakers and typically have relatively slow frequencies (25-40 bpm). Bradycardia also prolongs ventricular repolarization and predisposes to polymorphic ventricular tachycardia, especially torsade de pointes. Several drugs can cause bradyarrhythmias and tachyarrhythmias. Many antiarrhythmic drugs may produce bradycardia as a result of its specific effect on sinus node function or conduction auriculoventricular (AV). Syncope due to torsade de pointes is not uncommon, especially in women and is caused by the drugs that prolong the QT interval. It is particularly common in patients with long QT syndrome. Drugs that prolong the QT interval belonging to different categories, such as antiarrhythmics, vasodilators, psycho-tropics, antibiotics, non-sedating antihistamines, etc. Much has been learned on the hereditary long QT syndrome from data collected in the international registry.
2. Structural Cardiac Disease

Structural heart disease can cause syncope when circulatory demand exceeds the limited capacity of the heart to increase cardiac output. Syncope is very worrying when it is associated with clinical situations in which there is a fixed or dynamic obstruction of left ventricular outflow. The basis for the occurrence of an fainting is inadequate blood flow due to mechanical obstruction. However, in some cases, the syncope is not merely the result of restricted cardiac output, but may be partly a reflection of inappropriate or orthostatic hypotension [9-13]. For example, in the context of valvular aortic stenosis, the syncope is not merely the result of restricted cardiac output, but may be due in part to inadequate reflex vasodilatation and / or arrhythmia primary heart. Therefore, the mechanism of syncope may be multifactorial. Recognize in the heart the cause of the problem is justified by the need to correct the underlying structural disease, when possible.

<table>
<thead>
<tr>
<th>Table 3. Data to be considered as a cardiogenic syncope</th>
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<tbody>
<tr>
<td>- Presence of structural heart disease confirmed</td>
</tr>
<tr>
<td>- Family history of sudden cardiac death of unknown cause or channelopathy.</td>
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<td>- During the exertion, or supine</td>
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<td>- Sudden onset of syncope, palpitations followed immediately.</td>
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<td>- Findings indicate the ECG arrhythmic syncope:</td>
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<td>- Block bifascicular (defined as blocking both the right and left arm, combined with left anterior fascicular block or left posterior)</td>
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<tr>
<td>- Other intraventricular conduction abnormalities (QRS duration ≥ 0.12 s)</td>
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<tr>
<td>- Second-degree atroventricular block Mobitz I and Mobitz II.</td>
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<tr>
<td>- Complete atroventricular block.</td>
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<tr>
<td>- Inadequate asymptomatic sinus bradycardia (&lt;50 bpm), sinoatrial block or sinus pause ≥ 3 s in the absence of negative chronotropic medications.</td>
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<tr>
<td>- Non-sustained ventricular tachycardia.</td>
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<tr>
<td>- QRS complexes preexcited</td>
</tr>
<tr>
<td>- Short, Long QT interval and Short PR interval: Wolff-Parkinson-White’s syndrome; Mahaim’s syndrome; Lown-Gagnon-Levine’s syndrome; Breijo Pattern.</td>
</tr>
<tr>
<td>- Early Ventricular Repolarization.</td>
</tr>
<tr>
<td>- A pattern of right bundle branch block with ST elevation in leads V1-V3 (Brugada’s syndrome).</td>
</tr>
<tr>
<td>- Alterations in electrical cardiac systole.</td>
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<tr>
<td>- Negative T waves in right precordial leads, epsilon-waves and ventricular late potentials consistent with arrhythmogenic right ventricular cardiomyopathy.</td>
</tr>
<tr>
<td>- Q-waves consistent with myocardial infarction.</td>
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</tbody>
</table>

**Epidemiology and Prognosis [1, 3,4]**

The prevalence of other causes of syncope is different depending on the clinical context in which it evaluates the patient and the patient's age. In addition, there are other differences that depend on the diagnostic definitions, geographical factors and local care pathways, which make comparison between different studies. However, it is possible to make some general comments:
Cardiogenic Syncope

- The reflex syncope is the most frequent syncope in any context.
- Syncope secondary to cardiovascular disease is the second most common cause.

The number of patients with a cardiovascular cause varies widely between studies; the higher frequencies are seen in emergency departments, especially in older patients and consultations to cardiology.

- The orthostatic hypotension is a rare cause of syncope on general population; it is common in very elderly patients.
- Many episodes are misdiagnosed as syncope. Syncopal episodes in the initial evaluation are more frequent in patients referred from emergency and reflect the multifactorial complexity of these patients. While on young people, the reflex syncope is by far the most common cause of transient loss of consciousness, the elderly patients often has multiple causes and medical history may be less reliable than in the young.

It is quite possible that the actual statistics of episodes of syncope can be much higher than today we know. This may be due to a misinterpretation of them. In the clinical practice, we have seen how many of the episodes classified as "epilepsy", in fact, have an etiology due to an electrical cardiac systole’s alteration. Especially the so denominated: Syndromes of Lown-Ganong-Levine, Mahaim and Breijo (short PR interval with short QT interval in the same person).

It is possible, therefore, that incidence and prevalence of "Syncope" is very underestimated and can be most important. Recent studies show a "frequency" constant and higher significantly of syncope in the emergency services in Europe, with an incidence of 1% (0.9% -1.7%) of all of cases attended.

In the Framingham study, the incidence of syncope shows a pronounced increase after 70 years of age, from 5.7 episodes/1000 person-years in Males aged 60-69 years to 11.1 / 1,000 in those 70-79 years.

However, in older adults and in elderly patients (> 60 years) the cumulative incidence of syncope throughout the life becomes increasingly difficult to obtain due to bias in data fainting episodes in the past decades. Regarding the prognosis (ie, risk stratification) associated with syncope, one must consider two important elements:

- The risk of death and events that endanger life.
- The risk of recurrence of syncope.

The structural heart disease and primary electrical disease are major risk factors for sudden cardiac death and overall mortality in patients with syncope. Orthostatic hypotension is associated with a risk of death from the severity of co-morbidities that is 2 times the overall population.

In contrast, young patients in who has been excluded the structural and an electrical heart disease, have an excellent prognosis.
The number of patients with a cardiovascular cause varies widely among studies; the higher frequencies are seen in emergency departments, especially in older patients as well as in oriented queries on cardiology. Most of the deaths and poor prognosis may be related to the severity of the underlying disease, rather than syncope itself. In population studies, approximately one third of patients have recurrence of syncope in 3-year follow-up. The number of episodes of syncope during the life is the strongest predictor of recurrence. As premise, we could say:

“All syncopal episodes that appear on persons under 15 or over 65 should be considered as cardiogenic syncope until proven otherwise”.

**DIAGNOSIS**

The two decision trees that separate the transient loss of consciousness of other clinical situations are based on whether or not unconsciousness and if present the four characteristics that define the presentation of transient loss of consciousness (temporary, quick start, short duration and spontaneous recovery). The transient loss of consciousness is divided in traumatic or no traumatic.

The commotion tends to cause a loss of consciousness; as the existence of traumatism is usually clear, the risk of confusion in diagnosis is small. The transient loss of consciousness is divided in non-traumatic syncope, epileptic attacks, psychogenic pseudo-syncope and a miscellany of more rare causes. Rare causes include both those that are rare (such as cataplexy) and those whose presentation was similar to other forms of transient loss of consciousness only in unusual circumstances (eg., excessive sleepiness during the day).

The initial evaluation of a patient presenting with transient loss of consciousness consists of a careful history, physical examination, determinations include orthostatic blood pressure and ECG [14-19]. Syncope is a common phenomenon in the overall medical practice. The first thing we must consider is that syncope is not a disease in itself but a symptom. Therefore, it is the specific treatment of primary disease the main aim of any treatment.

Likewise, PREVENTION of syncope episode must be based on prevention from pathology causing the syncope.

The recurrent vasovagal syncope is the most common diagnosis in this context. The diagnosis is based on a careful history and the context in which the incident took place. The physician can diagnose most of these common and typical fainting, and just need reassurance. We recommend an active search for worrisome symptoms of syncope as during exercise; syncope while the patient is lying; no external factors; and family history of sudden cardiac death or slow recovery from syncope. If the diagnosis is uncertain and there is a potential risk of harmful consequences, the patient should be referred to a cardiologist, internist, neurologist or psychologist / psychiatrist, as appropriate, or a specialized unit of syncope if available.
Table 5. Differential diagnosis between epilepsy and syncope

<table>
<thead>
<tr>
<th>Epilepsy can cause transient loss of consciousness: they are “non-responders”, who fall and then have amnesia.</th>
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<tbody>
<tr>
<td>This occurs in the atonic seizures, tonic-clonic, clonic and tonic, only. In the epilepsy with absence total on children and in complex partial epilepsy on adults, consciousness is altered but there is not a full loss; patients remain standing during attacks, unlike what happens in the transient loss of consciousness (or syncope).</td>
</tr>
<tr>
<td>A complete flaccidity during unconsciousness is against epilepsy. The only exception is the &quot;atonic attacks&quot;, but this is rare and occurs without a trigger, on children with pre-existing neurological disease.</td>
</tr>
<tr>
<td>Involuntary movements may occur both in epilepsy and in syncope. In epilepsy, the movements are 1 min. aprox. Whereas in syncope are seconds.</td>
</tr>
<tr>
<td>The epileptic seizures are large, usually rhythmic and synchronous, whereas syncope is usually asynchronous, small and non-rhythmic. Nevertheless, sometimes the synchronous seizures can occur in syncope and eyewitnesses can report the type of movements incorrectly.</td>
</tr>
<tr>
<td>The involuntary movements in syncope occur only after the onset of unconsciousness and after the fall, not so in epilepsy. Syncope is usually triggered, epilepsy, rarely. There are some triggers the reflex epilepsy, such as lights going on and off, different from those of syncope.</td>
</tr>
<tr>
<td>A typical aura is a growing feeling in the abdomen (epigastric aura). This growing sense in syncope rarely occurs.</td>
</tr>
<tr>
<td>Sweating and pallor are common in epilepsy and no in syncope.</td>
</tr>
<tr>
<td>The bite of the language is much more common in epilepsy and occurs on the sides of the tongue, while in syncope occurs at the tip. Urinary incontinence occurs in both.</td>
</tr>
<tr>
<td>Patients may feel confused after the attack for some time in epilepsy while syncope is usually retrieved immediately.</td>
</tr>
<tr>
<td>Headache, muscle pain and an increase of creatine kinase and prolactin are much more common after epileptic episode.</td>
</tr>
</tbody>
</table>

**MANAGEMENT OF SYNOCOPE IN THE OVERALL PRACTICE**

Syncope is a common phenomenon in the overall practice. The recurrent vasovagal syncope is the most common diagnosis in this context.

The diagnosis is based on a careful history and the context in which the incident took place. The physician can diagnose most of these common and typical fainting, and just need reassurance. We recommend an active search for worrisome symptoms of syncope as during exercise; syncope while the patient is lying; no external factors; and family history of sudden cardiac death or slow recovery from syncope. If the diagnosis is uncertain and there is a potential risk of harmful consequences, the patient should be referred to a cardiologist, internist, neurologist or psychologist / psychiatrist, as appropriate, or a specialized unit of syncope if available.

Based on the results of different tests, the additional examinations that may be performed [20,21]:

...
Carotid sinus massage in patients over 40 years.
- Echocardiogram when is been previously known cardiac disease or findings consistent with structural heart disease or syncope secondary to cardiovascular causes.
- ECG monitoring immediately when suspected arrhythmic syncope.
- Orthostatic tests (proof of inclination or Till test) when the syncope is related to the position vertical or there is suspicion of reflex mechanism.
- Other less specific tests, including neurological examination or blood tests are only appropriate when there is suspicion of transient loss knowledge in non-syncopal.

Management of Cardiac Arrhythmias as Primary Cause for Syncope

We repeat that syncope is not a disease in itself but a consequence of some pathology. In this section would be a result of any cardiac arrhythmia. The aims of treatment are to prevent the recurrence of symptoms, improve quality of life and prolong survival. The basis of syncope in these situations is multifactorial and is influenced by ventricular rate, left ventricular function and the adequacy of vascular compensation (including the potential impact of reflex neuro mediated) [20,21].

1. Sinus Node Dysfunction: In every cases , the cardiac pacemaker therapy is indicated and has proved very effective in patients with sinus node dysfunction when there is an ECG evidence for spontaneous, bradyarrhythmia, that is the cause of syncope or is a result of a recovery time sinus node abnormal. The permanent stimulation often relieves symptoms, but may have no effect on survival. Even when there is adequate stimulation, syncope recurs in 20% of patients in long-term monitoring. This is due to the frequent association of a vasodepressor reflex mechanism of sinus node disease. We recommended, pacing modes ventricular fibrillation with minimal stimulation have been recently developed as an alternative to conventional rate-dependent pacemaker dual chamber, in patients who mainly require headset support.

The removal of drugs that may exacerbate underlying susceptibility to bradycardia is an important element in preventing the recurrence of syncope. However, when replacement is not feasible may be necessary cardiac stimulation. Cardiac ablation techniques for atrial tachyarrhythmia control have become increasingly important in selected patients presenting as bradycardia-tachycardia, sick sinus syndrome, but are used only occasionally to prevent syncope.

2. Disease Atrioventricular Conduction System:
The pacing is the treatment of syncope associated with symptomatic atrio-ventricular block (AVB).

The indications and the preferential mode of stimulation in the AVB have been updated recently. The possible deleterious role of apical stimulation permanent on right ventricular also been stressed recently, but alternative sites of stimulation are still under discuss.

Stimulation should be considered in patients requiring biventricular pacing due to AVB, depressed Left ventricular ejection, heart failure and wide QRS.

3. Paroxysmal Tachycardias:
Supraventricular and ventricular tachycardia in patients with AV re-entry or typical atrial flutter associated with syncope, treatment of choice is catheter ablation. In these patients, the role of pharmacotherapy is to bridge to the ablation or used when the ablation has failed. In patients with syncope associated with atrial fibrillation or atypical left atrial flutter, the decision must be made individually. Syncope due to torsade de pointes is not uncommon, and is acquired as a result of treatment with drugs that prolong the QT interval. The treatment is to discontinue the drug immediately suspect.

Should be considered for catheter ablation or medical therapy in patients with syncope due to ventricular tachycardia, in the context of a normal heart or heart disease, with mild cardiac dysfunction.

ICDs are indicated in patients with syncope and cardiac function depressed, and tachycardia or ventricular fibrillation without a cause susceptible of correction. Although the ICD patients are usually not prevent recurrence of syncope, is indicated for reducing the risk of sudden cardiac death. Dysfunction of the implanted device 4 oddly shaped implantable stimulation systems have caused pre-syncope or syncope. However, more often than syncope in these patients has no relation with device.

4. Patients with primary electrical disease: The unexplained syncope is considered an ominous finding in patients with inherited disorders of cardiac ion channels. Should be considered carefully the use of an ICD in the absence of other diagnosis or cannot be excluded ventricular tachyarrhythmia as a cause of syncope. However, the mechanism of syncope can be heterogeneous and in some cases, can cause serious arrhythmias, but in many other cases may have a more benign origin, as reflex syncope. Therefore, in these contexts, syncope does not necessarily involve a high risk of major cardiac events that endanger life and is less sensitive than a history of documented cardiac arrest.

In the long QT syndrome, especially in those with LQTS2 and LQTS3, the number of cardiac events before age 18, very long QT intervals and females has a worse clinical outcome [21].

Patients with Brugada syndrome with a spontaneous type 1 ECG pattern have a worse outcome than those who have a pattern or type 2 induced by drugs.

The usefulness of ICD in patients with syncope is controversial and certainly more questionable than in survivors of cardiac arrest [19,20,21].

**SPECIAL TOPICS [22,23]**

SYNCOPE IN THE ELDERLY.

The most common causes:

- Orthostatic hypotension, reflex syncope, especially the carotid sinus syndrome and cardiac arrhythmias.

- Often different forms coexist in a patient, which makes diagnosis difficult.

- Hospitalization associated with orthostatic hypotension increase progressively with age: 4.2% in patients 65 to 74 years and 30.5% in patients over 75 years.

- Symptomatic patients, 25% had orthostatic hypotension related age", in the rest, orthostatic hypotension is mainly due to atrial fibrillation medication and primary or secondary.
Systolic hypertension supine often occurs in elderly patients with orthostatic hypotension and complicates treatment, as most of the drugs used to treat orthostatic hypotension exacerbate supine hypertension and vice versa.

SYNCOPE IN PAEDIATRIC PATIENTS
Two specific disorders occur early on in childhood:

1. Infantile reflexes syncopal attacks (also called anoxic apnea or seizures pale reflections) produced by a brief unpleasant stimulus, are due to cardiac vagal inhibition.

2. The transient loss of consciousness type apneic hypoxic (cyanotic apnea also called) is characterized by a cessation of breathing during expiratory crying, cyanosis and often produces transient loss of consciousness.

OTHER DISORDERS IN ELECTRICAL CARDIAC SYSTOLE AS CAUSE FORyncopal episodes

The electrical cardiac systole originates from the beginning of the P wave (atrial depolarization) to the end of the descending branch of the T wave (ventricular repolarization). Are included, therefore, the succession of P-QRS-T and its corresponding intervals and segments: PQ, ST and QT. The mathematical possibilities in the variation on length of electrical systole of the heart may be several. It is well documented and demonstrated that such changes in length can cause that be more vulnerable and unstable all myocardial cells, and can also cause serious cardiac arrhythmias, several syncope episodes and even sudden death for this motive. Even today, many of these disorders are poorly understood and, too many times, its clinical manifestations are categorized as "episodes of epilepsy"; other times (most) are classified within a "common sack" called "channelopathies", when -actually- is the alteration from electrical cardiac systole the true etiology of them. All these disorders can cause syncopal episodes.

The measures and lengths of the different components of electrical cardiac systole, considered for most authors as normal are these:

- PR-interval: 0.120-0.200 seconds.
- QRS complex: 0.08-0.120 seconds.
- QT-interval (corrected): 0.350-0.450 seconds. (Here, there is much disagreement among different authors). The most used methods for QT interval correction, since it is frequency-dependent, are Bazett, Fridericia.

When the PR-interval is lesser than 0.120 seconds, we call it a short PR-interval. In contrast, when is greater than 0.200 seconds, we call it a first-degree AV block. When the QRS complex is lesser than 0.08 seconds, we call it "narrow QRS" but when is greater than 0.120 seconds, we call it "wide QRS". Likewise, when the corrected QT-interval length is lesser than 0.350 seconds, we call it Short QT-interval and when is greater than 0.450 seconds, we call it a Long QTc-interval.

It is clear that there may be, in the same ECG recording, a combination of them all.
Some of these disorders, we will explain briefly below.
A/ Wolff-Parkinson-White’s Syndrome (WPWS).
Wolff-Parkinson-White syndrome (WPWS) is a congenital heart disease (PRKAG2. Genetic map 7q36) characterized by a premature ventricular depolarization caused by an abnormal atrioventricular accessory pathway, between the atria and ventricles, known as Kent’s bundle. However, even today, is called into question the real cause of Wolff-Parkinson-White, there are some authors who believe that, PRKAG2 mutations, are caused by a glycogen storage cardiomyopathy associated with WPWS, because the overwhelming majority of accessory pathways occur in individuals without structural heart disease, and probably without this mutation. The pathogenesis of accessory pathway formation in PRKAG2 may be completely different, and some authors believe it is due to an inflammation of myocardial cells that occur in the atrial-ventricular connections.

In fact, do not even know if the accessory pathways are mediated genetically or due to environmental exposures or randomly.

A short PR interval, a delta wave, a wide QRS complex (greater than 120 ms) and, occasionally, alterations in the ventricular repolarization are its main electrocardiographic characteristics on the ECG. Its incidence varies between 0, 1% and 3% in the general population.

It is essential to achieve the right differential diagnosis between

- Wolff-Parkinson-White’s syndrome or real ventricular pre-excitation.
- Lown-Ganong-Levine syndrome or accelerated atrioventricular conduction.
- Mahaim’s syndrome.
- “Short PR alongside short QT” intervals in the same person. (Breijo's Pattern).

Typical ECG image of the Wolff-Parkinson-White.

In this context of ECG recording, that has a normal heart rate, a short PR interval, a delta-wave and an early ventricular repolarization can be seen.

B/ Lown-Ganong-Levine Syndrome (LGL).

This syndrome was described in 1952 by Lown, Ganong, and Levine, forming the famous now used to describe it. It is considered a preexcitation syndrome.

We now know two types of pre-excitation syndrome:

- The Wolff-Parkinson-White or ventricular preexcitation true.
---
The Lown-Ganong-Levine or accelerated atrioventricular conduction.

- Short PR alongside short QT" intervals in the same person. (Breijo's Pattern).

LGL is a disease entity that is included within the more general condition called Short PR-Interval).

**Etiology** • Acquired . • Congenital :
- Inherited.
- Not inherited.

The familial form is inherited, as an autosomal dominant genetic trait has been associated with the PRKAG2 gene that encodes the activated AMP protein kinase, responsible for transport and store energy from the heart. A mutation in this gene could explain the susceptibility of the heart to the crises of tachycardia. Mutation has been identified on the long arm of chromosome 7 (7q34-q36).

The Lown-Ganong-Levine may affect approximately 1 in every 50,000 people.

Several structural abnormalities have been proposed as the possible basis for LGL, including the presence of James's fibbers, Mahaim's fibbers, Brechenmacher and underdeveloped anatomic sinus node (hypoplastic).

Each of these fibbers can only be identified histologically.

Thus, unless other studies demonstrate definitive structural or functional abnormalities, the diagnosis of LGL remains a clinical diagnosis.

In the absence of significant structural heart disease, the mortality rate appears to be very low.

Patients may present with an acute episode of tachycardia or a history of symptoms suggestive of paroxysmal tachycardia.

In diagnosis is necessary to make:

1. A standard test for tachycardia, including an ECG to document the rhythm.
2. Serum electrolytes, calcium, magnesium levels, and levels of serum thyroid hormone-stimulating hormone (TSH). Lithemia.
3. History suggestive of recurrent paroxysms of tachycardia,
4. A Holter monitor or event recorder may be useful to document the rhythm during acute symptomatic episodes.
5. An ergometric study.
6. In rare cases, an implantable monitor for pace may be helpful.
7. Family History. (Screening).

**Differential Diagnosis with Wolf-Parkinson-White**

Although apparently similar, there are differences, which, in our opinion, are critical with respect to drug treatment elective. The key differences are:
The LGL is a PR-interval shortened due to the presence of accessory pathway, prevents the AV node but normal QRS because the accessory pathway (James fibbers) binds directly to the sinus and depolarizes the ventricles not directly, but does so by typical pathway, by the Hiss-Purkinje system.

- Not displayed "Delta waves -" in D1, aVL, V5 and V6.
- The QRS complexes tend to be narrow because there is usually no interventricular conduction disturbance.
- It is not be as frequent the association of atrial fibrillation during concomitant crisis.

**Prognosis:**

No studies have shown an increased risk of sudden death or reduced survival for patients meeting the criteria for the diagnosis of LGL.

**Current Therapeutic Bases**

Rarely, the drug medical therapy can have failures usually, but there are patients in who there is not effective (for patients who continue to have recurrent and intolerable symptoms). In such extreme cases are used:

- Radiofrequency ablation (RF).
- The external pacemaker.
- The Implantable Cardioverter Defibrillators (ICDs).

This destroys the accessory pathway using a catheter (tube) inserted into the body to reach the heart. The success rate of this procedure ranges between 85 and 95% depending on the location of the extra or additional route.

Digoxin, verapamil and beta-blockers (other drugs commonly used to treat other types of tachycardia) can increase the frequency of episodes of tachycardia in some people with this syndrome. Beta-blockers may increase cardiac depression.

We can use drugs such as adenosine (Inpatient), and amiodarone to control or prevent episodes of tachycardia. For the control of tachycardia is usually proceed according to the severity of the implementation of vagal maneuvers carotid massage type and Valsalva maneuver (forced expiratory made with the nose and mouth closed).

Typical ECG image of the Lown-Ganong-Levine:
C/ Short PR-interval alongside short QT- interval on the same person. (Breijo’s Pattern) [24,25,26].

In 2006, Breijo-Marquez, Pardo Ríos et al. evaluated a series of young patients, who had had, since childhood, many episodes of nocturnal palpitations, chest pain, full loss of consciousness (syncope), and which were accompanied by tonic-clonic seizures. All had been diagnosed and treated as epileptic episodes. Treatment outcomes were null. They were always considered as normal, in every cardiac studies performed absolutely.

However, all these patients had an ECG recording common:

- A PR-interval lesser than 0.120 seconds with a QTc-interval equal to or lesser than 0.350 seconds.

That is, a pattern of short PR and QTc in the same person.

The correct treatment was begun (beta-blockers and, in some cases, an implantable cardio defibrillator, ICD.). Was removed all treatment from epilepsy.

The outcome to date is satisfactory.

Although we don’t know, with certainty, the etiology of this pattern of ECG to date, we know that there were two important confusions:

First. - The physicians mistook to syncopal episode, with an epileptic episode.

Second. - The syncopal episodes are due to a cardiac disorder (was a cardiogenic syncope due to a cardiac electrical systole’s alteration).

This ECG recording may be easily confused with a Lown-Ganong-Levine, since both have a short PR-interval. Nevertheless, in this type of ECG pattern there is also a short QTc-interval.

Unfortunately, both entities are confused with epileptic episodes too often.

Typical ECG image of the “Short PR alongside short QT” intervals in the same person. (Breijo’s Pattern):

![ECG Image](image_url)

This ECG recording was the first with 12 leads that was obtained from our Hospital from Boston, MA. The patient was a 17 years-old male. We can see a shortening of the PR and QT intervals (Bazett), especially in inferior and left precordial leads. PR-interval length is lesser than 0.120 seconds and QTc length is lesser than 0.350 seconds. Patient had the symptoms exposed previously. He was also diagnosed for epileptic episodes. However, he had syncopal episodes by cardiological disturbances.
D/ MAHAIM SYNDROME.
 Mahaim syndrome is characterized by:
 The PR- interval with a standard length. Presence of pseudo-delta wave in the initial phase of the QRS complex because the sinus stimulus enters to AV node where physiological suffers a delay and then depolarizes the ventricles by an abnormal way: Mahaim fibers. That is:
 PR-interval with a normal length.
 Wide QRS complexes.
 Typical ECG image of the Mahaim’s Syndrome

![Mahaim Syndrome ECG Image]

Differential diagnosis among various entities with alterations in electrical cardiac systole.

<table>
<thead>
<tr>
<th>ENTITY</th>
<th>PR-interval</th>
<th>QRS complex</th>
<th>QTc -interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.P.W</td>
<td>Short</td>
<td>Wide (δ-wave)</td>
<td>Normal</td>
</tr>
<tr>
<td>L.G.L</td>
<td>Short</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Mahaim</td>
<td>Normal or Short</td>
<td>Normal or wide</td>
<td>Normal</td>
</tr>
<tr>
<td>Breijo’s Pattern</td>
<td>Short</td>
<td>Normal</td>
<td>Short</td>
</tr>
</tbody>
</table>

Differential diagnosis, based on the characteristics from the different intervals and complex.

CONCLUSIONS

In any loss of consciousness, physicians should follow a protocol in order to know if a syncopal episode is or not.
1. Is This a Syncopal Episode?

On the initial presentation, the patient will not mention syncope but will use terms of common speech. They may say that they have ‘fallen’, ‘fainted’ or ‘had a blackout’. A witness may state that they have fitted. It is important to assist the patient to change these general terms into more exact medical language. Start at the point where they felt normal and take them through what happened from the onset of symptoms to full recovery. When did they realize that they were not well, why was this, were there disturbances of vision, sound or smell? Did they ‘fall’; do they remember the fall or just being on the floor? How quickly did they come round? How did they feel? Did they hurt themselves? Were they incontinent? Were there witnesses? Did they comment on duration, movement during unconsciousness and pallor?

2. Maybe This Is Not Syncope?

Through the history, there may be points that suggest that this is not syncope but a loss of consciousness due to another cause. The distinction between syncope and epilepsy is a difficult process and will influence the route of referral. If movements are tonic–clonic, continue for more than 15 seconds and involve the whole of a limb or limbs, this would suggest epilepsy. In comparison, irregular movements of short duration affecting only the distal parts of the limbs might suggest only cerebral hypoxia. If confusion following the attack lasts over 5–10 minutes, this may also be suggestive of an epileptic seizure. In older people with falls, a clear history may not be available. There may have been no witness of the incident and poor memory may mean events leading to the incident are unclear. Without a history of a stumble or fall then there should be a suspicion of syncope. Older people can experience syncope without realizing that it has happened. This renders the history unreliable without a witness. A full falls assessment in the patient's own environment is essential. People can collapse without losing consciousness due to other causes, such as paroxysmal benign positional vertigo and drop attacks. If there is a suspicion of collapse without loss of consciousness specialist consultation will be necessary with either an ear, nose and throat (ENT) surgeon or a neurologist. Careful definition of the term dizziness, meaning light-headedness, and giddiness, implying rotation, is important in assessment of these conditions.

3. What Has Gone Before?

Syncope needs to be put in the context of previous medical history. Is there known structural heart disease such as valvular disease or heart failure? Has the patient a past history of angina or myocardial infarction? Has the patient a past history of electrical cardiac disturbances? Has there been a previous history of blackout, especially in youth? When was this and was a diagnosis established at the time? What steps were taken? Has there been a previous diagnosis of epilepsy made? If so, how was this diagnosis made?

Not only does the sufferer need to be considered but family history also needs to be considered. Is there a history of sudden cardiac deaths? These can masquerade as unexpected
deaths that have been explained away, drowning or road traffic accidents. Is there a known family history of an inherited cardiac condition?

It is also of interest to know if their parents or other family members are fainters. It has been observed that people who faint are more likely to have come from parents who were also fainters.

4. Carry Out an Examination

A clinical examination is always vital and should be comprehensive.

Starting at the pulse, this should be assessed by manual palpation for rhythm and rate. Is there a bradycardia? Is the rhythm regular? A full assessment of blood pressure both supine and erect is necessary. The blood pressure should be taken after lying for 10 minutes and then several times in the few minutes after standing, where a fall of 20 mmHg in systolic or to < 90 mmHg, or a fall in diastolic of 10 mmHg are considered abnormal and strongly suggestive of orthostatic hypotension.

The cardiovascular examination should involve a detailed auscultation of the heart to identify valvular lesions or signs of heart failure. A general neurological assessment should be undertaken, seeking gross neurological deficit. The gait should be observed to see if there are abnormalities that make falls likely.

If there is a rotational or vertiginous element in the history, a simple Dix-Hallpike assessment should be performed to check for benign paroxysmal positional vertigo. If this is positive, then assessment may be required from an ENT specialist. It is important to note that benign paroxysmal positional vertigo is not associated with loss of consciousness, and if the history is suggesting blackout, then these findings may be coincidental.

5. Undertake Investigation

Any patient presenting with syncope or a disturbed period of consciousness should undergo simple investigations. These may include blood chemistry and blood count. An electrocardiogram (ECG) is a vital part of the assessment. A normal ECG is an excellent prognostic feature. Changes that should raise concern include:

- Bradycardia: < 40 beats per minute (except in athletes) or pauses >3 seconds
- Mobitz II, 2nd, 3rd degree atrioventricular (AV) block
- Alternating right and left bundle branch block
- Rapid supraventricular tachycardia
- Ventricular tachycardia
- Cardiac ischemia
- Myocardial infarction.
- Electrical cardiac systole’s disturbances:

ECG patterns mixed. Unless the attacks are happening on a daily or very frequent basis, 24-hour ECG monitoring reveals nothing. A normal 24-hour ECG without symptoms does not further the diagnosis at all and should not be seen as reassuring.

6. What Is the Prognosis?

Patients presenting with any problem want to know if they will come to any harm. This is such a complex area that it can be difficult to give assurances. However, in young healthy people with a normal ECG and a suggestion of neurally mediated (vagal) or rarely orthostatic hypotension the prognosis is excellent. The presence of structural heart disease, independent of the cause of syncope, reflects a poor prognosis.

SOME EXAMPLES OF ECG RECORDING WITH DIFFERENT PATTERNS THAT PRODUCE SYNCOPAL EPISODES. [27,28].

1°. Wolff-Parkinson-White and Prolonged "Q-T" Patterns in the Same Electrocardiographic Record

Wolff-Parkinson-White syndrome (WPWS) is a congenital heart disease (PRKAG2. Genetic map 7q36) characterised by a premature ventricular depolarisation caused by an abnormal atrioventricular accessory pathway known as Kent’s bundle. Prolonged QT syndrome (PQTS) consists of an abnormal prolongation of the QT interval on the ECG, which can be both inherited and acquired. This anomaly is known to favour the occurrence of malign cardiac arrhythmias, above all polymorphic ventricular tachycardia, ventricular fibrillation and “torsade de pointes”.

When taken separately, both syndromes have little incidence, which leads us to expect this incidence to be even lower when they are found on the same electrocardiogram. Incidentally, the current medical literature contains no publications on this topic. This clinical case aims to establish the existence of an electrocardiographic pattern characterised by WPW and a PQTS pattern on an ECG record. With a high susceptibility to crisis of tachycardia, especially at night, several episodes of syncope, even cardiac arrest.

The patient is a 24-old-years man. Since childhood, he has suffered from more than four tachycardia attacks, three documented syncope episodes, as well as two cardiac arrests recovered, for which he was treated with electric discharges. Afterwards, he was treated with radiofrequency ablation of Kent’s bundle, with permanent positive results so far.
We can see a typical ECG recording of an intermittent WPW and a Long QT-interval together in a patient with several syncopal episodes and a recovered cardiac arrest.

2°. ECG patterns with short PR interval together to a long QT (A) and first-degree AV block alongside a long QT (B: Increased of cardiac electrical systole). [25,27,29].

In this exposition, we present the ECG record of two patients with an obvious diversity and variability of alterations in the electrical system of heart. These electrical cardiac disturbances could explain completely the symptomatology from patients: nocturnal palpitations, several syncopal episodes. In Figure A, we can see the presence of a short PR-interval together to a Long QT-interval. In Figure B, we can also see an ECG recording with a Long PR-interval alongside a Long QT-interval.

ACKNOWLEDGEMENTS

Lourdes and Alejandro Breijo, for their unselfishness collaboration from Miami, Florida, USA.

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