emergencias 2007;19:273-282

Review Article

An approach to the diagnosis and treatment of syncope in the emergency department

J. Sagristá Sauleda

Servei de Cardiologia. Hospital Universitari Vall d'Hebron. Barcelona

ABSTRACT

Syncope, defined as a transient lost of conciousness with spontaneous recovery in a short period of time due to an acute diminution of cerebral blood flow, is a frequent cause of admission in the emergency room. Syncope has multiple etiologies with very different pronostic implications. The role of the emergency room physician is, firstly, to differentiate syncope from nonsyncopal causes of lost of consciousness, and, secondly, to try to establish the most probable etiological diagnosis and its pronostic relevance by means of relatively simple and easily available tools. In this paper we describe the pathophysiology of syncope, its diagnostic approach and the criteria for hospital admission. Finally, some therapeutic recommendations are provided.

Key Words: *Syncope. Emergency.*

RESUMEN

Abordaje diagnóstico y terapéutico del síncope en urgencias

El síncope, definido como un cuadro de pérdida completa y transitoria de la conciencia con recuperación espontánea en un breve intervalo de tiempo debido a disminución del flujo sanguíneo cerebral, es un motivo frecuente de consulta en los servicios de urgencias. El síncope puede ser debido a múltiples causas con significado pronóstico muy variable. El papel del médico de urgencias consiste, en primer lugar, en diferenciar el síncope de otras causas de pérdida de conciencia, y luego intentar establecer la etiología más probable y perfilar la gravedad pronóstica, lo cual se puede conseguir mediante métodos sencillos y fácilmente disponibles. En este trabajo se describen someramente los diferentes mecanismos del síncope, los métodos para llegar a un diagnóstico etiológico, los criterios de ingreso hospitalario y el enfoque terapéutico.

Palabras clave: *Síncope. Urgencias.*

DEFINITIONS AND GENERAL CONSIDERATIONS

Syncope is defined as the transient loss of consciousness and postural tone which is completely and spontaneously recovered after a short period of time. This is caused by an acute, critical and temporary reduction in cerebral blood flow. The patient suffering from syncope usually falls onto the floor if they are standing up or collapses if they are sitting down. The loss of consciousness may begin suddenly or may be preceded by warning symptoms. It last for a short time (seconds or a few minutes) and may occur alone or along with other symptoms of general discomfort or epigastic discomfort, nausea, vomiting, sweating, diarrhoea, incontinence or convulsions. Many patients with syncope are also affected by presyncopal episodes or milder syncopes which may manifest themselves in the same way and be just as clinically relevant.

Conceptually, only loss of consciousness caused by a temporary reduction in cerebral blood flow should be diagnosed as syncope, given that there are other states of loss of consciousness that are different and should not be classified as syncope. When a patient comes to the emergency department explaining that they have suffered a loss of consciousness, the first issue that should be considered is whether we are dealing

Correspondence: Dr. Jaime Sagristá Sauleda
Servei de Cardiologia
Hospital Universitari Vall d'Hebron
P. Vall d'Hebron 119-129
08035 Barcelona
E-mail: jsagrist@vhebron.net

Received: 1-6-2007
Accepted: 8-6-2007

with a real syncope (transient loss of consciousness caused by a reduction in cerebral blood flow) or a loss of consciousness caused by something else that might be confused with syncope[1,2].

Other causes of loss of consciousness, apart from having different medical characteristics, cannot be attributed to a reduction in cerebral blood flow. Metabolic disorders (hypoglycaemia, metabolic coma, hypoxia, hyperventilation with hypocapnia) occur within a certain context which generally makes them easy to diagnose. Hypoglycaemia normally appears in diabetic patients who are being treated with insulin or oral antidiabetic drugs. When this causes a loss of consciousness the onset is usually slow and preceded by weakness, sweating, trembling, feeling hungry and confusion. The heart rate is not altered and the patient is not affected by low blood pressure. Symptoms disappear when the patient eats something or when administered glucose intravenously. Psychogenic syncope[3,4] usually affects people with personality disorders, it may be prolonged and often the "syncopes" occur frequently, their characteristics are varied and in contrast with the circulatory syncope, those affected do not recover after lying down, their heart rate, blood pressure and colouring do not change and there is no trauma. The possibility of acute poisoning should always be considered and in these cases the index of suspicion and asking the correct questions is crucial. "Drop attack" symptoms (falling to the floor caused by ischemia/claudication) can be difficult to differentiate from real syncope, especially in older patients who do not respond clearly to questions. Symptoms of TIA (transient ischemic attack) may coincide with those of some focal neurologic deficits, however they do not cause a loss of consciousness and so therefore cerebrovascular diseases are not a recognised cause of syncope. Doubts are common when having to differentiate between syncope and an epileptic attack[5-9]. It is sometimes difficult to make the distinction given that syncope can include convulsions and incontinence.

The elements that help us to establish a differential diagnosis are the information that can be obtained from a detailed patient history and/or a witness account and the findings of a physical examination and ECG. Epileptic fits may be sensed by the patient early on; tonic-clonic seizure and automatism are the first symptoms and may occur even before a loss of consciousness. Often the patient bites their tongue, their face turns blue and they experience prolonged confusion after the attack. In contrast, syncope may be preceded or accompanied by vegetative symptoms (sweating, nausea); if convulsions occur they do so late on, there is no biting of the tongue or confusion although incontinence and a general feeling of weakness afterwards may be present.

Once all of these elements have been considered and the initial differential diagnosis has been made we can refer to these patients as patients with syncope (according to the definition previously outlined).

Syncope is a very common symptom[10-14]. Its prevalence among the general population varies between 3% and 50%. Approximately between 25% and 50% of patients who have suffered from syncope will experience at least one recurring episode. Firstly, syncope poses a diagnostic problem given that it is not possible to diagnose the cause with certainty after a full examination in an increased number of cases (30%-50%). Secondly, the prognosis of syncope can vary from the benign symptoms of a case of vasovagal syncope (although at times this can be debilitating) to a sign of sudden death in patients with severe heart disease. Another potentially serious aspect of syncope is the collapsing given that if the onset of syncope is sudden it can cause serious injuries.

PATHOPHYSIOLOGY

The common pathophysiological basis of syncope is the acute, transient reduction in cerebral metabolism caused by decreased cerebral blood flow. The mechanism that triggers it may vary according to the type of syncope involved, however the factors that have the greatest influence are a changing combination of a reduction in cardiac output and a reduction in peripheral resistance and consequently, low blood pressure. Both factors play a part in many types of syncope.

CLASSIFYING THE CAUSE OF THE DISEASE AND A CLINICAL DESCRIPTION OF SYNCOPES

Table 1 shows different syncopes classified according to their main cause.

Cardiac syncope

Cardiac syncope represents approximately between 10% and 40% of syncopes according to different series. They can be secondary to arrhythmias or obstructive.

1. Secondary to arrhythmias. Most cardiac syncopes are secondary to arrhythmias, whether it be a bradyarrhythmia (chronic or paroxysmal blockage of the ventricular artery, sinus arrest, etc.) or a tachyarrhythmia. Supraventricular tachyarrhythmias rarely cause syncope unless the patient already

TABLE 1. Classification of the causes of syncope

1. Syncope of cardiac origin
   - Caused by arrhythmias
   - – Bradyarrhythmias
     - A-V block
     - Sinus node disease
   - – Tachyarrhythmias
     - Supraventricular,
     - Ventricular
       - Sustained ventricular tachycardia
       - "Torsades de pointes" in patients with congenital or acquired long QT syndrome
       - Polymorphic tachycardia in patients with Brugada syndrome
   - Caused by obstructive heart disease (aortic stenosis, hypertrophic cardiomyopathy, pulmonary stenosis, pulmonary thromboembolism, pulmonary hypotension)
2. Syncope caused by orthostatic hypotension
3. Reflex syncope. Neuromediated syncope
   - Vasovagal syncope
   - Situation syncope (coughing, micturation, defecation, swallow)
   - Carotid sinus hypersensitivity syncope
4. Other types of syncope
   - Exercise-related syncope
   - Syncope caused by drugs
   - Syncope caused by a neurological disease

has heart disease or if the arrhythmia coincides with a very high ventricular rate. Ventricular arrhythmias (or ventricular fibrillation) are a recognised cause of syncope. The majority of these arrhythmias occur in patients with organic heart disease (ischemic heart disease, cardiomyopathy); however, sometimes they are solely a manifestation of disorders of the electrical system, such as long QT syndrome[15,16] and Brugada syndrome[17,18].

2. Obstructive syncope. This includes syncopes which occur in patients with conditions that can cause obstructions in the blood flow (atrial myxoma, atrial thrombus, left ventricle outflow tract obstruction, severe pulmonary stenosis, pulmonary thromboembolism[19], pulmonary hypertension, etc.).

The onset of cardiac syncopes is usually quite rapid and lasts a short time, with recovery often occursing very quickly. If syncope is prolonged, signs of severe cerebral anoxia may appear (generalised convulsions, incontinence, cyanosis and stertorous respiration) which are all symptoms that make up Stokes-Adams syndrome.

Syncope caused by orthostatic hypotension

Syncope caused by orthostatic hypotension is due to a rapid decrease in arterial pressure when standing up because of low blood volume or a problem with the reflexes that control arterial pressure. Orthostatic hypotension (the definition of this varies depending on different authors and can be defined as a drop in systolic blood pressure of 30 mm Hg and of over 15 mm Hg in diastolic blood pressure) is common in elderly patients and is often not associated with any clinical symptoms.

Therefore, a diagnosis of syncope cased by orthostatic hypotension should be based on indicative signs and symptoms which should be tested. Low blood volume irrespective of its cause and a long list of drugs (hypotensors, diuretics, vasodilators, tranquilisers, antidepressants, etc.) can cause or aggravate orthostatic hypotension. Hypotension can also be a sign of a series of endocrinological or neurological (Parkinson's disease) diseases or conditions affecting the nervous system[20].

Circulatory syncope. Neuromediated syncope or syncope caused by neural mediation

This section includes a series of syncopes that have an important element in common. The activation of a reflex arc plays a very an important role when it appears and involves the neurovegetative system along with vagus nerve stimulation and sympathetic nerve inhibition.

Vasovagal or vasodepressor syncope[21,22]. – Although there can be various triggers, the definitive way to identify vasovagal syncope is by detecting a decrease in the sympathetic tone which causes low blood pressure (vasodepressor component) and an increase in the vagal tone which impedes compensatory tachycardia and can even cause severe bradycardia and asystole lasting several seconds (cardioinhibitor component)[23] (Figure 1). The vasodepressor component is probably more important given that atropine or pace maker stimulation cannot prevent syncope even when bradycardia is avoided. The most common type of vasovagal syncope (fainting or faintness) occurs in situations where there is a reduction in the ventricular volume (an accumulation of venous blood after getting up or spending a long time standing, hot environments, sweating, giving blood, the use of vasodilators, etc.) or in situations involving a release of

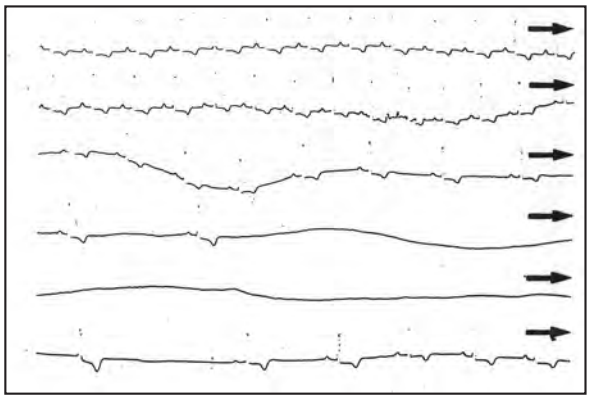

Figure 1. The two upper strips show normal sinus rhythm (slightly fast). In the third strip the onset of progressive sinus bradycardia begins which ends in asystole lasting several seconds (strips 4 and 5) and subsequently recovers (sixth strip). This recording corresponds to the tilt-table test carried out in a young patient with a history of vasovagal syncope. The asystole does not indicate the presence of sinus node illness but rather a cardioinhibitory response caused by vagal stimulation ("neuromediated syncope"). Despite the seriousness of this response the patient does not need a pace maker given that the prognosis of this condition is good.

adrenalin (fear, pain, intense emotional stimulation, etc.). Vasovagal syncope is the most common cause of syncope among the general population and usually begins at a young age[21,22]. Approximately 15%-25% of individuals have experienced syncope once or twice during adolescence. Clinically, vasovagal syncope usually has a slow onset with early signs and symptoms that last a few seconds or minutes including pallor, sweating, nausea, muscle weakness, etc. The loss of consciousness is not usually deep and it is recovered gradually after a few minutes while the individual remains pale, sweaty and weak. In contrast to this type of "benign vasovagal syncope", some patients experience syncopes with a rapid onset, without any triggering factors or early signs and symptoms. In these cases the tilt-table test also allows us to identify the vasovagal mechanism. These symptoms are collectively known as "malign vasovagal syncopes" and usually affect older individuals.

Situational syncope. - Cough syncope, micturation syncope, defecation syncope and swallow syncope are included in the group of syncopes known as situational or circumstantial syncopes. The causes and effects of these syncopes are mixed and can contribute to a reflex mechanism, Valsalva's manoeuvre applied to a certain extent which causes a decrease in venous return and low blood pressure when standing. Micturation syncope is more common among men. Swallow syncope may occur when individuals are affected by conditions related to the oesophagus (tumours, diverticula, spasms, etc.). Situational syncopes can be recurrent but in general they do not lead to sudden death.

Syncope caused by carotid sinus hypersensitivity[25,27].- Massage or compression of the carotid sinus can lead to limited sinus bradycardia and slightly low blood pressure in some people. When this condition becomes extreme it is known as carotid sinus hypersensitivity. The response to the stimulation of the carotid sinus is made up of two components: a cardioinhibiting component (ventricular activity stops for more than 3 seconds and this is caused by asystole or an atrioventricular block, it is defined as pathological) and a vasodpressor component (a drop of over 30 mm Hg in systolic pressure is defined as abnormal). The cardioinhibiting component is more common and occurs in about 70% of patients with this syndrome. Carotid sinus hypersensitivity is very common among older people. It is important to highlight that not all patients with carotid sinus hypersensitivity experience syncope. In fact, a diagnosis of syncope caused by carotid sinus hypersensitivity or carotid sinus syndrome should only be established when the syncope has been triggered by a manoeuvre that could have caused stimulation of the carotid sinus and a pathological response to carotid sinus massage has been identified. Carotid sinus hypersensitivity causes syncope in approximately 1%-5% of cases.

Other types of syncope

Exercise-related syncope.- Syncope may be triggered by exercise in patients with aortic stenosis, pulmonary stenosis and pulmonary hypertension.

Similarly, exercise can cause supraventricular and paroxysmal ventricular arrhythmias. In principle, a syncope that occurs during exercise is most likely to be related to a vasovagal reflex.

Syncope caused by drugs.- Some drugs can induce syncopes when they cause low blood pressure (hypotensors, diuretics, vasodilators, etc.), or when they cause supraventricular or ventricular arrhythmias (the "proarrhythmic effect" of certain antiarrhythmic drugs). Cocaine and marijuana may also cause syncope. Table 2 details the drugs that can cause syncope.

Syncope caused by a neurological disease.- Several neurological diseases can cause low blood pressure when standing and induce syncope this way. Some of these diseases are mentioned in the section that describes orthostatic syncope[20].

TABLE 2. Drugs that can cause syncope

Drug	Possible effect
Antiarrhythmic drugs	Lengthening of the QT interval (ventricular arrhythmias)
Quinidine	
Sotalol	
Amiodarone	
Vasodilatadors	Low blood pressure
Nitrates	
Calcioantagonists	
Antihypertensives	Low blood pressure
Beta blockers	Bradycardia. A-V block
Diuretics	Low blood volume. Alterations in electrolytes
Antidepressants	Arrhythmias. Orthostatic hypotension
Phenothiazines	Orthostatic hypotension
Levodopa	Orthostatic hypotension

A GENERAL EVALUATION OF PATIENTS WITH SYNCOPE AND THE GUIDELINES OF THE STUDY

Syncope is a syndrome that can be attributed to a number of causes (with different prognoses) and that should be evaluated methodically. The diagnosis of the causes of syncope is essentially based on three main elements which are available to any doctor: the medical history, a physical examination and an ECG. In the majority of cases where a cause can be established, it is done using simple instruments. When this is not possible, it is often difficult to make a diagnosis despite carrying out additional tests.

During the patient's initial general evaluation, the doctor should be able to identify potential chronic diseases (general debilitating diseases, anaemia, etc.) or acute breakthrough processes (haemorrhage, dehydration, taking medication, etc.) which may have a causal relationship with the syncope. In patients with risk factors we must have a high index of suspicion for pulmonary thromboembolism.

The medical history is fundamentally important and should be done meticulously. Questions should be asked about the position of the body and possible changes in position at the time of the syncope, the situation in which the syncope took place, triggers, early signs and symptoms, the speed of the onset, manifestations during the syncope and the type of recovery. It important to ask any witnesses questions since the patient may not be able to provide specific details about some aspects of the syncope (convulsions, skin colouring) while they were unconscious. Questions should be asked systematically about any medication the patient might be taking. In some cases the cause of syncope can be established from the medical history (coughing syncope, micturation syncope and other types of vasovagal syncope triggered by painful stimuli or medical manipulation). Syncope which takes place while patients are lying down and does not include a triggering factor, in principle exclude a vasovagal cause. Syncopes that appear very quickly and have limited characteristics in a patient with heart disease or pathological ECG suggest an arrhythmic cause.

Apart from the general aspects, the physical examination should include taking blood pressure from both arms, looking for carotid murmurs and measuring arterial blood pressure when lying down and when standing for a prolonged period of time (10 minutes). Low blood pressure is very common in elderly people and its role as a cause should be established after a full clinical evaluation and other tests.

Physical examination allows examination of important aspects such as pallor and tumours and may establish a diagnosis of aortic stenosis, hypertrophic cardiomyopathy or pulmonary stenosis.

The ECG only allows detection of the cause of syncope in 2%-10% of cases. At other times it may contribute information that increases suspicion (intraventricular conduction disorder, bradyarrhythmias, signs of preexcitation, etc.) which may indicate which mechanism is involved and which tests should be carried out. The anomalies that may suggest an arrhythmic syncope can be found in Table 3. These tests should be carried out in all patients that come to the emergency department for syncope as they allow a diagnosis to be established in a large number of cases.

It is important to emphasise that the only solid evidence of the cause of syncope can be found when there is a diagnostic discovery in a patient who is being monitored correctly (heart rate and blood pressure). This happens in exceptional circumstances and, most of the time, the diagnosis of the cause of syncope is based on indirectly related information. The diagnostic criteria that have been recommended (and allow a diagnosis to be established with an acceptable level of certainty) are the following:

Vasovagal syncope

Syncope which is clearly triggered by a precipitating cause (pain, fear, aggression or intense emotion) or which occurs in situations that favour the condition (prolonged standing, crowds, heat). Warning symptoms such as nausea or instability without any kind of trigger is not enough to establish a diagnosis of vasovagal syncope. If the cause of

TABLE 3. Anomalies in the ECG that suggest an arrhythmic syncope

- Bifascicular block (defined as a bundle block in the left branch or a block in the right branch plus anterior or posterior hemiblock).
- Other intraventricular conduction disorders (QRS length ≥ 0.12 seconds).
- Second degree Mobitz II arterioventricular block.
- Asymptomatic sinus bradycardia (< 50 bpm) or arterial sinus block.
- Ventricular preexcitation in the ECG.
- Prolonged QT.
- Right bundle branch block pattern with elevated ST in V1-V3 derivations (Syndrome de Brugada).
- Negative T waves in right precordials or epsilon waves suggestive of arrhythmogenic right ventricular dysplasia.

syncope is unknown, a diagnosis of vasovagal syncope can be established for patients who do not have structural heart disease and have a normal basal ECG, by obtaining a positive result from the tilt-table test (more information below). A diagnosis of vasovagal syncope can only be established for patients with heart disease or conduction alterations in the basal ECG when all other cardiogenic causes of syncope have been ruled out.

Situational syncope

A high probability diagnosis is usually made in the following situations. However, just because the syncope is linked to a particular set of circumstances described, that does not mean that other causes can be completely ruled out.

Cough syncope

When syncope occurs immediately after significant coughing without any other apparent cause.

Micturation syncope

When syncope occurs immediately before, during or after urinating without any other apparent cause.

Defecation syncope

When syncope occurs during or immediately after defecating without any other apparent cause.

Swallow syncope

When syncope is triggered by swallowing without any other apparent cause.

Carotid sinus hypersensitivity syncope

Syncope which is triggered by a manoeuvre that can cause stimulation of the carotid sinus (shaving, etc.) and pathological response to carotid sinus massage is identified (see below).

Syncope caused by orthostatic hypotension

Syncope which occurs when the patient adopts an upright position and low blood pressure is identified.

Syncope caused by bradyarrhythmia

Syncope caused by bradyarrhythmia can be confirmed when the ECG shows a complete A-V block, an A-V Mobitz II type block (Figure 2) or a block of the right branch alternating with a block of the left branch. The presence of intraventricular conduction disorders or severe sinus bradycardia is not enough to establish a diagnosis of syncope caused by bradyarrhythmia. However, these findings should prompt other tests to be carried out in order to rule out syncope caused by bradyarrhythmia.

Syncope caused by tachyarrhythmia

This can be diagnosed if we detect the presence of supreventricular paroxysmal or sustained ventricular tachycardia (Figure 3) during or immediately after syncope. Similarly the presence of heart disease, particularly ischaemic heart disease and cardiomyopathies, runs of unsustained ventricular tachycardia, preexcitation syndrome in the basal ECG, long QT interval or ST elevation in V1 V2 with the image of the block in the left branch (suggestive of Brugada syndrome), rule out syncope caused by tachyarrhythmia.

Apart from trying to establish a cause, an evaluation of the seriousness of the syncope should be carried out. The seriousness may be determined by the risk of sudden death or the risk of severe injuries in relation to how quickly the syncope occurs. The risk to life associated with syncope depends on the presence of heart disease and abnormalities in the ECG. In principle, a benign prognosis can be established when the ECG is normal and there is no heart disease.

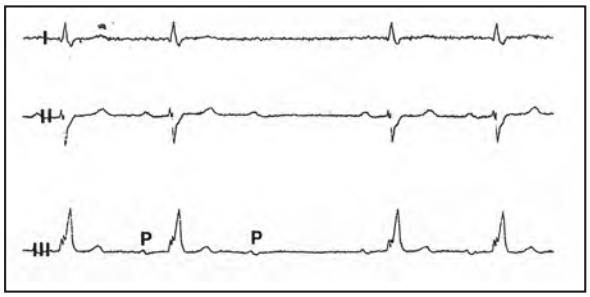

Figure 2. The ECG clearly shows a blocked P wave and a wide QRS complex which indicates intraventricular conduction disorder. Therefore, we can assume that this patient's syncopes have been caused by an arterioventricular block and the insertion of a permanent pacemaker is recommended.

In contrast, if the patient does have heart disease (ischaemic heart disease with myocardial infarction even in the past, any kind of cardiomyopathy) or a clear abnormality in the ECG, the prognosis may be serious or at least guarded. This evaluation should also be used to assess whether hospital admission is necessary and in order to establish the priority level for carrying out the rest of the diagnostic tests.

CRITERIA OF HOSPITAL ADMISSION

Some authors have already outlined the hospital admission criteria for walk-in patients[28]. Once the patient has come to the emergency department we believe that the following individuals should be admitted to hospital:

– Patients with a suspected severe left ventricular outflow tract obstruction.

– Patients with suspected bradyarrhythmia or tachyarrhythmia, especially if the syncope has a sudden onset.

– Patients with severe heart disease, especially those with a history of myocardial infarction or cardiomyopathy.

– Patients with pacemakers or implantable defibrillators.

In the following situations hospital admission should be considered on the basis of the individual:

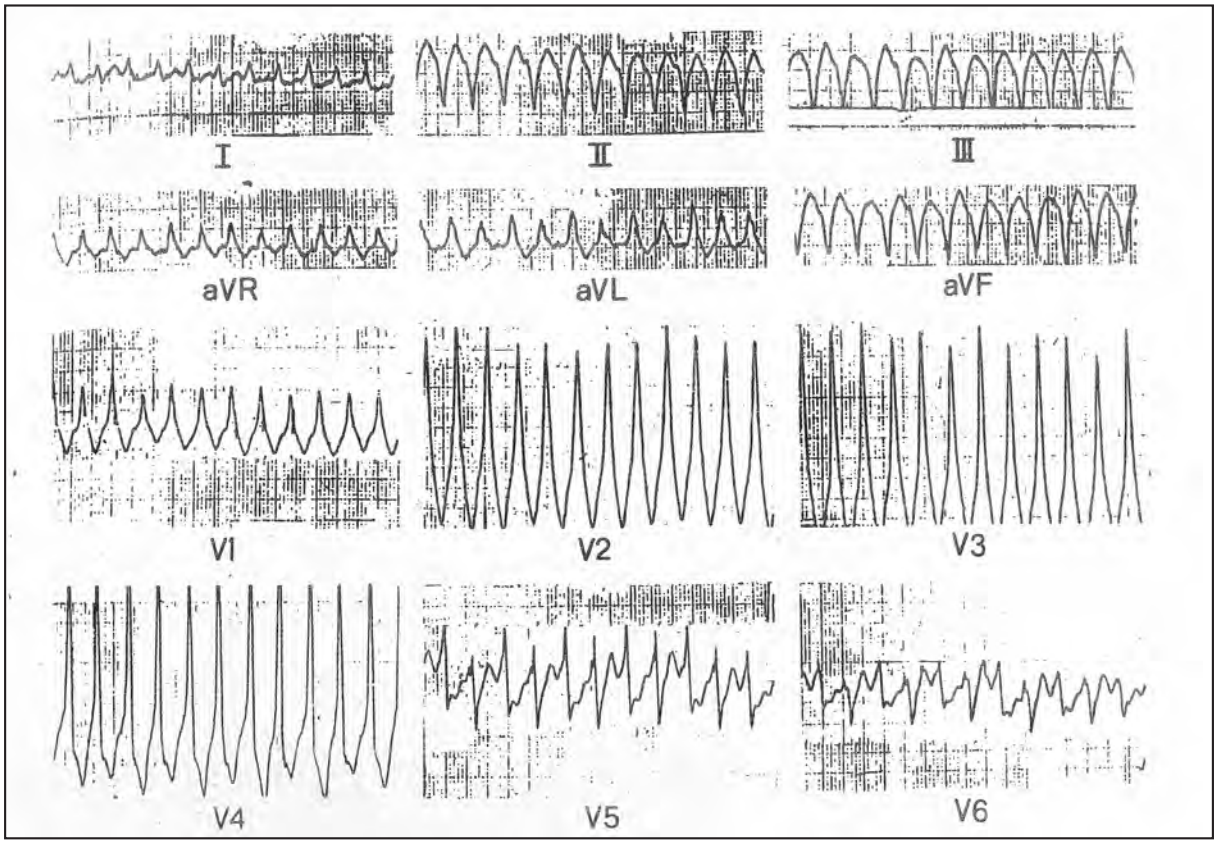

Figure 3. This trace belongs to a patient with a history of myocardial infarction with sudden syncopes. A very fast tachycardia with wide QRS complex corresponding to ventricular tachycardia, which is obviously the cause of the syncopes, can be observed.

– Patients with recurrent episodes of syncope which negatively affect their quality of life, irrespective of the cause of syncope.

– Patients with injuries.

– Syncopes which occur during exercise.

ADDITIONAL TESTS

Although the emergency physician's duties end once the patient is admitted into hospital, it is worth outlining the additional tests that can be carried out if the cause of syncope has still not been established.

The tilt-table test[29-33]

The aim of this test is to try and induce vasovagal syncope, so that if the test triggers a positive response and the patient's general background is indicative, a diagnosis of vasovagal syncope can be established. This test is recommended for patients with suspected vasovagal syncope with the aim of confirming the diagnosis, and for patients with syncope of unknown origin, no heart disease, normal ECG and little suspicion of syncope caused by arrhythmia. The tilt-table test is only be recommended for patients with a history of heart disease or suspected syncope caused by arrhythmia if all other tests fail to find an arrhythmic cause.

The tilt-table test is a passive standing test in which the patient is moved from a horizontal position to a 60º angle (with the head tilted upwards) and this is maintained for 20 minutes using a tilting couch with a platform for the patient's feet. This simple procedure may trigger a vasovagal response and the test is positive if the patient develops symptoms of syncope, or presyncopal manifestations caused by low blood pressure and/or associated bradycardia which may reproduce the symptoms of spontaneous syncope. The response may consist of symptoms of vasodepression with significant low blood pressure and no bradycardia or even a cardioinhibitory response with severe bradycardia and prolonged asystole, although the most common response is mixed (low blood pressure and bradycardia). The level of diagnostic accuracy of the tilt-table test is relatively low given that its degree of sensitivity and precision are around 60%-80%.

Electrophysiological exam[32]

The objective of the electrophysiological exam is to assess the function of the sinus node and the atrioventricular node and study the possible cause of arrhythmias that may explain the syncope. This is recommended for patients with suspected arrhythmic syncope, caused either by bradyarrhythmias or tachyarrhythmias. Therefore, this is carried out in patients with intraventricular conduction disorder (advanced bundle block in the right branch, advanced bundle block in the left branch, bifascicular block) and patients with organic heart disease (except aortic stenosis and myxoma). A description of the methodology and diagnostic criteria fall outside the aims of this study.

Carotid sinus massage

This is recommended for patients over the age of 50 who do not suffer from heart disease and have a normal ECG, especially when the tilt-table test has not established a diagnosis. It is recommended for patients with heart disease or abnormalities in the basal ECG when a cause is not established after carrying out the electrophysiological exam. This is not recommended for patients with carotid pulse alterations that suggest an obstruction or with carotid murmurs or a history of a cerebrovascular accident. The two carotid arteries should be examined (with a minimum time gap of two minutes between them); the ECG and blood pressure should also be monitored. Furthermore, the examination should ideally be carried out while the patient is lying down and when they are standing. The massage should last about 5 seconds and an asystole over 3 seconds or symptomatic low blood pressure is considered an abnormal response. An abnormal response alone is not enough to diagnose the cause given that positive results can be obtained from patients with syncopes that have other causes. In order for a diagnosis of carotid sinus hypersensitivity syndrome to be confirmed, not only must the response be positive but the syncope must also have the indicative clinical characteristics (syncope triggered by pressure applied to the neck or by neck movements).

Continuously monitoring the ECG

There are several continual ECG monitoring systems that are designed to record the heart rate during a spontaneous attack of syncope and therefore establish a diagnosis of arrhythmic syncope or rule out this possibility. The classic 24-hour Holter system is limited because of its quick recording time and therefore it is often not possible to register a syncope using this method. In fact, the performance results show that it diagnoses syncope in only 4% of cases[33,34]. Therefore, over the last few years different monitoring systems for implantable ECGs have been developed which allow much longer recording times.

REVEAL[23,35,36] is a recording system that is implanted in the subcutaneous tissue of the precordial area and lasts over 12 months. If syncope (or presyncope) occurs the patient can freeze the memory so that it can analyse the heart rate for around 40 minutes, in this way we can know the patient's heart rate immediately before and during the syncope. REVEAL is essentially meant for patients with heart disease or with intraventricular conduction disorder accompanied by one or more syncopes of unknown origin. Its use is only considered for patients who do not have heart disease and have a normal ECG when the syncope is recurrent and clinically severe (although the majority of these cases are vasovagal syncopes).

Echocardiogram

This is carried out in patients with heart disease or suspected heart disease in order to identify the condition except in cases of aortic stenosis or arterial myxoma. It rarely provides any definitive information on the cause of the syncope.

Exercise test

This is essentially carried out in patients with exercise-related syncope. It should also be carried out on patients with ischaemic heart disease. However, myocardial ischaemia on its own (without myocardial infarction) very rarely explains the symptoms of syncope.

Coronariography

The coronariography should not be carried out to establish the cause of syncope. Its use depends on the seriousness of the symptoms attributed to ischaemic heart disease.

Aortic arch branch Doppler ultrasound

This should be carried out in patients with arteriosclerosis of the aortic arch branches (carotid murmur, asymmetric pulses or arterial pressure in both arms) and in patients with a neurological focus. In any case, we have already mentioned that carotid artery disease causes cerebrovascular accidents rather than syncopes.

Consulting the Department of Neurology

If the aforementioned tests do not draw any conclusions regarding the cause of the syncope or if there are doubts about the possibility of the patient having epilepsy, a neurologist should assess the case. He to decide whether specific tests should be carried out or not. In these cases an electrocephalogram is necessary. Head CT scans and MRIs are not recommended as initial tests for patients with clear syncopes such as dose described earlier in this article.

Consulting the Department of Psychiatry

This should only be considered for patients strongly suspected of having psychiatric disease and only after ruling out organic causes of syncope.

PROGNOSIS

We have already highlighted that the prognosis of syncope basically depends on the presence of heart disease[1,2,10-12,37,38]. Mortality after 1 year stands at approximately 20% to 30% in patients with cardiac syncope, from 0% to 10% in patients with non-cardiac syncope and 5% in patients with syncope of unknown cause. Patients with syncope of unknown cause, without heart disease and with a normal ECG have an excellent prognosis and a survival rate similar to that of the general population.

TREATMENT

Some causes of syncope can be treated easily and effectively (for example, surgery in aortic stenosis or inserting a pacemaker for an atroventricular block). When the diagnosis of the cause of syncope is based on indirect information, such as, asymptomatic supraventricular or ventricular arrhythmias or an intraventricular conduction disorder, treatment should be personalised. It is important to give patients with vasovagal syncope instructions regarding how to avoid the factors that trigger syncope and how to identify the early signs and symptoms. Patients should be advised to eat foods which are high in salt (at least five times a day) and drink plenty of water especially in hot weather. If these methods are not effective different treatments such as ethylephrine, mineralcorticoids or beta blockers can be recommended, although none of these have proven to be significantly effective. The patient should be given information about counter-pressure manoeuvres (basically consisting of isometric exercises testing the arms and legs)[39] which raise blood pressure and can be done when the patient experiences the early symptoms of syncope. The manoeuvres can prevent or delay the appearance of the syncope

and consequently injuries can be avoided. The psychological support of a doctor who can reassure the patient with a good prognosis may be very helpful since treatment methods alone are often not very effective.

REFERENCES

1- Fitzpatrick AP, Cooper P. Prognosis and management of patients with blackouts. Heart 2006;92:559-68. doi: 10.1136/hrt.2005.068650.

2- Day SC, Cook EF, Funkenstein H, Goldman L. Evaluation and outcome of emergency room patients with transient loss of conciousness. Am J Med 1982;73:15-23.

3- Zaidi A, Crampton S, Clough P, Fitzpatrick A, Scheepers B. Head-up tilting is a useful provocative test for psychogenic non-epileptic seizures. Seizure 1999;8:353-5.

4- Grubb BP, Wolfe D, Gerard G. Syncope and seizures of psychogenic origin: identification with head-up tilt testing. Clin Cardiol 1992;15:834-42.

5- Sander JWAS, Sharvon SD. Epidemiology of the epilepsies. J Neurol Neurosurg Psychiatry 1996;61:433-43.

6- Hoefnagels WA, Padberg GW, Owerweg J, van der Velde EA, Roos RA. Transient loss of conciousness: the value of the history for distinguishing seizure from syncope. J Neurol 1991;238:39-43.

7- Benbadis SR, Wolgamuth BR, Goren H, Brener S, Fouad-Tarazi F. Value of tongue biting in the diagnosis of seizures. Arch Intern Med 1995;155:2346-9.

8- Zaidi A, Clough P, Cooper P, Scheepers B, Fitzpatrick A. Misdiagnosis of epilepsy: many seazure like attacks have a cardiovascular cause. J Am Coll Cardiol 2000;36: 181-4.

9- Sheldon R, Rose S, Ritchie D, Connolly SJ, Koshman ML, Lee MA, et al. Historical criteria that distinguish syncope from seizures. J Am Coll Cardiol 2002;40:142-8.

10- Kapoor WN, Karpf M, Wieand S, Peterson JR, Levey GS. A prospective evaluation and follow-up of patients with syncope. N Engl J Med 1983;309:197-204.

11- Kapoor WN. Evaluation and outcome of patients with syncope. Medicine 1990;69:160-75.

12- Soteriades ES, Evans JC, Larson MG, Chen MH, Chen L, Benjamin EJ, et al. Incidence and prognosis of syncope. N Engl J Med 2002;347:878-85.

13- Sagristá Sauleda J, Permanyer G, Moya A, Rius T, Soler Soler J. Síncope en la edad avanzada. Estudio etiológico en una serie prospectiva de 100 pacientes. Rev Esp Geriatr Gerontol 1994;29:287-91.

14- Brignole M, Alboni P, Benditt DG, Bergfeldt L, Blanc JJ, Bloch Thomson PE, et al. Guidelines on management (diagnosis and treatment) of syncope. Europace 2004;6:467-537.

15- Roden DM, Lazzara R, Rosen M, Schwartz PJ, Towbin J, Vincent GM. Multiple mechanisms in long QT syndrome. Current knowledge, gaps and future directions. Circulation 1996;94:1996-2012.

16- Priori SG, Schwartz PJ Schwartz PJ, Napolitano C, et al. Risk stratification in the long QT syndrome. N Engl J Med 2003;348:1866-74.

17- Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and suden cardiac death: a distinct syndrome and electrocardiographic syndrome. A multicenter report. J Am Coll Cardiol 1992;20: 1391-6.

18- Martínez Pérez J, Capdevila Bernardo D, Pérez Sánchez A. Síncopes de repetición en varón joven. Síndrome de Brugada. Emergencias 2004;16:23-9.

19- Querol Gutiérrez JJ, Morales Gordillo MA, Levy Naon A, Querol Gutiérrez JC, Vázquez Fuentes N, Laza E, et al. Mujer joven con síncope y tromboembolismo pulmonar bilateral. Emergencias 2005;17:152-3.

20- Smith AAJ, Halliwill JR, Low PA, Wieling W. Pathophysiological basis of orthostatic hypotension in autonomic failure. J Physiol 1999;519:1-10.

21- Chen-Scarabelli C, Scarabelli TM. Neurocardiogenic syncope. BMJ 2004;329:336-41.

22- Wieling W, Ganzeboom KS, Saul IP. Reflex syncope in children and adolescents. Heart 2004;90:1094-9

23- Moya A, Brignole M, Menozzi C, García-Civera R, Tognarini S, Mont L, et al. The mechanism of syncope in patients with isolated syncope and in patients with tilt-positive syncope. Circulation 2001;104:1261-7.

24- Kerr A, Derbes VJ. The syndrome of cough syncope. Ann Med Intern 1953;39:1240-53.

25- Kenny RA, Traynor G. Carotid sinus syndrome: clinical characteristics in elderly patients. Age Ageing 1991;20:449-54.

26- Brignole M, Menozzi C. Carotid sinus syndrome: diagnosis, natural history and treatment. Eur J Cardiac Pacing Electrophysiol 1992;4:247-54.

27- Kenny RA, Richardson DA, Steen N, Bexton RS, Shaw F, Bond J. Carotid sinus syndrome: a modifiable risk factor for nonaccidental falls in older adults (SAFE PACE). J Am Coll Cardiol 2001;38:1491-6.

28- Hernández García J, Salegi Etxebeste I, Marín Tamayo JM, Pérez Irazusta I. Criterios de derivación hospitalaria en los pacientes con síncope. Emergencias 2004;16:68-73.

29- Almquist A, Goldenberg IF, Milstein S, Chen MY, Chen XC, Hansen, et al. Provocation of bradycardia and hypotension by isoproterenol and upright posture in patients with unexplained syncope. N Engl J Med 1989;321:346-51.

30- Fitzpatrick AP, Theodorakis G, Ahmed R, Vardas P, Sutton R. Methodology of head-up tilt in the investigation of unexplained syncope. J Am Coll Cardiol 1991;17:125-30.

31- Moya Mitjans A, Permanyer Miralda G, Sagristá Sauleda J, Mont Girbau Ll, Rius Gelabert T, Soler Soler J. Análisis de las respuestas de la prueba en tabla basculante en función de las características clínicas de los episodios sincopales en pacientes sin cardiopatia aparente. Rev Esp Cardiol 1993;46:214-9.

32- Sagristà-Sauleda J, Romero B, Permanyer-Miralda G, Moya A, Rius-Gelabert T, Mont-Girbau L, et al. Clinical usefulness of head-up tilt test in patients with syncope and intraventricular conduction defect. Europace 1999;1:63-8.

33- Sagristà-Sauleda J, Romero-Ferrer B, Moya A, Permanyer-Miralda G, Soler-Soler J. Variations in diagnostic yield of head-up tilt test and electrophysiology in groups of patients with syncope of unknown origin. Eur Heart J 2001;22:857-65,doi:10.1053/euhj.2000.2398.

34- Gibson TC, Heitzman MR. Diagnostic efficacy of 24-hour electrocardiographic monitoring for syncope. Am J Cardiol 1984;53:1013-7.

35- Krahn Ad, Klein GJ, Yee R, Skanes AC. Randomized assessment of syncope trial: conventional diagnostic testing versus a prolonged monitoring strategy. Circulation 2001;104:46-51.

36- Deharo JC, Jego C, Lanteume A, Dijiane P. An implantable loop recorder study of highly sympomatic vasovagal patients. J Am Coll Cardiol 2006;47:587-93.

37- Kapoor WN, Hanusa B. Is syncope a risk factor for poor outcomes?. Comparison of patients with and without syncope. Am J Med 1996;100: 646-55.

38- Baron-Esquivas G, Errazquin F, Pedrote A, Cayuela A, Gomez S, Aguilera A, et al. Long-term outcome of patients with vasovagal syncope. Am Heart J 2004;147:883-9.

39- Krediet CT, van Dijk N, Linzer M, van Lieshout JJ, Wieling W. Management of vasovagal syncope: controlling or aborting faints by leg crossing and muscle tensing. Circulation 2002;106:1684-9.