Approach to syncope related to the sport

Aridane Cárdenes León[1], José Juan García Salvador[1], Clara A. Quintana Casanova[2], Alfonso Medina Fernández Aceytuno[1]

[1]Servicio de Cardiología. Hospital Universitario Dr. Negrín de Gran Canaria. [2]Unidad de Medicina Deportiva. Hospital Perpetuo Socorro. Las Palmas de Gran Canaria.

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Summary

The safety of physical activity in athletes who have presented a syncope is not well established. It differs in some aspects from the management of syncope in the general population. Although syncope in athletes is generally benign, inadequate assessment can have dire consequences. Syncope may be the prelude to episodes of sudden death. It is a frequent phenomenon in the general population, it is estimated that up to 50% of the population may present a syncopal episode throughout life, as well as relatively infrequent in the sports population. It is estimated that around 6% of athletes may experience syncope at 5 years of follow-up. An adequate differential diagnosis is essential. The etiology of these episodes can be very diverse, although in most cases, we are faced with benign cause syncopes that appear right after exercise. However, it is necessary to be systematic and rational when considering other diagnostic studies that allow us to rule out malignant cardiological pathologies such as cardiomyopathies, channelopathies, etc. Likewise, many doubts arise among professionals when it comes to establishing the best recommendations in relation to continuing physical activity at the professional level. It is really important to establish an algorithm of decisions about proper management of them. Particular caution should be taken when suspending physical activity in athletes who present syncopal episodes of benign etiology or treatable causes. Current research focuses mainly on the safety of sports activity in athletes with syncopal episodes and the fear that the persistence of the practice of physical activity at a competitive level can significantly increase the risk of adverse events, especially arrhythmic events and sudden death. In this review, we will analyze numerous studies and guidelines of clinical practice, in order to establish the recommendations for an adequate assessment of syncope of the athlete, as well as the restriction of sports activity in pathologies that can be potentially lethal.

Key words:
Syncope. Exercise. Athlete. Sporting activity. Competition.

Abordaje del síncope relacionado con el deporte

Resumen

La seguridad de la actividad física en deportistas que han presentado un cuadro sincopal no está claramente establecida y difiere en algunos aspectos del manejo del síncope en la población general. Aunque el síncope en deportistas es un cuadro por lo general benigno, un evaluación inadecuada puede tener consecuencias nefastas, ya que el síncope puede ser la antesala de episodios de muerte súbita. El síncope es un fenómeno frecuente en la población general, se estima que hasta un 50% de la población puede presentar un episodio sincopal a lo largo de la vida, así como relativamente poco frecuente en la población deportista. Se estima que en torno a un 6% de los atletas pueden experimentar un síncope a los 5 años de seguimiento. Es fundamental la realización de un adecuado diagnóstico diferencial. La etiología de estos episodios puede ser muy diversa; aunque en la mayoría de los casos, nos encontramos ante síncopes de causa benigna que aparecen justo después del ejercicio. Sin embargo, es necesario ser sistemáticos y racionales a la hora de plantear otros estudios diagnósticos que nos permita descartar con seguridad aquellas patologías cardiológicas malignas (miocardiopatías, canalopatías, etc.) Asimismo, surgen numerosas dudas entre los profesionales a la hora de establecer las mejores recomendaciones en relación a continuar la actividad física a nivel profesional y en el algoritmo de decisiones para establecer el manejo adecuado de los mismos. Se debe tener especial precaución a la hora de suspender la actividad física en deportistas que presentan cuadros sincopales de etiología benigna o de causas tratables. Las investigaciones actuales se centran principalmente en la seguridad de la actividad deportiva en deportistas con episodios sincopales, y el temor a que la persistencia de la práctica de actividad física a nivel competitivo pueda aumentar de forma significativa el riesgo de eventos adversos, especialmente de eventos arrítmicos y muerte súbita. En esta revisión, analizaremos numerosos estudios y guías de práctica clínica, con el fin de establecer las recomendaciones a la hora de realizar una adecuada valoración en el síncope del deportista, así como la restricción de la actividad deportiva en patologías que pueden resultar potencialmente letales.

Palabras clave:
Síncope. Ejercicio físico. Deportista. Actividad deportiva. Competición.

Correspondence: Aridane Cárdenes León
E-mail: aricardenes@gmail.com

Introduction

Syncope is a common phenomenon in all age groups, however, its assessment in competition-level athletes is little known to date.

These episodes predominantly occur after having performed intense exertions and are generally benign. However, syncope during exertion may be a symptom of structural heart disease or channelopathies that may trigger sudden death. To focus the diagnosis, it is key to carry out a detailed anamnesis, a thorough physical exploration as well as, occasionally, multiple diagnostic techniques (electrocardiographic monitoring, image techniques, etc.).

Despite the majority of syncopes in athletes being reflective and considered benign, such as, for example, neurologically mediated syncopes, if this occurs when the athlete is performing a high-risk sport (diving, motorcycling, etc.), it could potentially be lethal. It is estimated that in around 50% of cases, no definitive ethology is found for the syncope[1].

Recommendations for treatment and a potential restriction of physical activity constitute an important challenge for the clinic. We should consider that the long-term suspension of sporting activities may cause serious emotional and psychological problems in the athlete.

For these reasons, today it is considered essential to carry out a complete study of syncope in athletes so as to avoid unfavourable outcomes and to avoid undue sporting restrictions in healthy individual athletes. By means of this review, the assessment and handling of syncope in competition-level athletes will be discussed.

Definition

Syncope is defined as the transitory loss of conscience due to global cerebral hypoperfusion, with posterior spontaneous and complete recovery. In contrast, pre-syncope is defined as the presence of torpor or weakness without arriving at a loss of consciousness[2].

The differential diagnosis of the syncope is wide, though the majority present a benign aetiology. Only a small percentage is attributable to the presence of underlying structural heart disease[3].

Demographic

This clinical framework presents a prevalence of around 40% in the general population[4]. Athletes represent an exceptional population in the context of handling syncope, given the great controversy in the need to carry out screening tests in athletes, in both the elite and in those that carry out recreational physical activity.

Cases of syncope are more frequently due to vasovagal or orthostatic aetiology (at around 30%) with the presence of cardiogenic causes in around 9.5% of cases. A broad study describes that in 37% of cases, a definitive cause of the syncopal framework is not described[5].

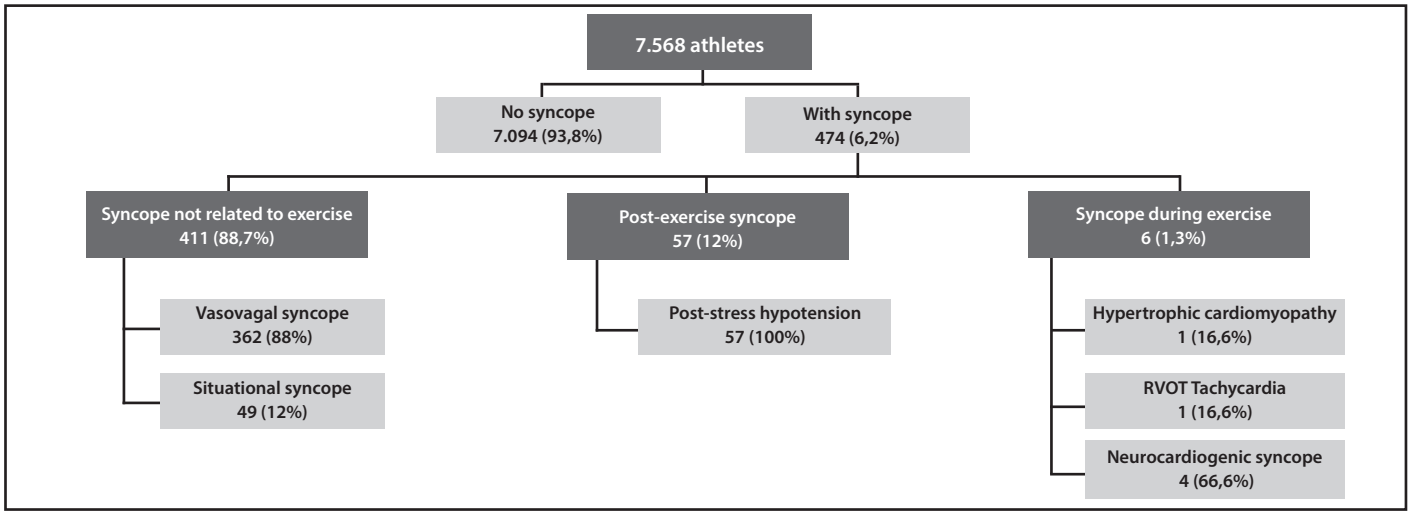
It is estimated in different publications that around 5-6% of athletes may experience a syncope in 5 years of follow-up[6]. In a cohort of 7,568 athletes[7], 474 (6.2%) of athletes presented syncopes in the 5 successive years. Of these, the vast majority of the cases were not related to exercise, 12% were post-exercise, and just 1.3% of cases were triggered during the exertion, with these latter being diagnosed as hypertrophic cardiomyopathy, right ventricle outflow tract tachycardia and neurologically mediated syncope (Figure 1). As such, the majority of the syncopes were neurologically mediated, also called reflex or vasovagal syncopes.

The syncope is currently much less frequent in athletes than in the general population[8]. Furthermore, according to diverse publications, the majority of syncopal episodes are not related to exercise[9].

Classification

We should take into account that in the study of syncope in athletes, we may find different situations, trying to clearly limit the time

Figure 1. Cohort of 7568 athletes, 6.2% of which presented syncopes in the 5 successive years. The figure describes their different aetiologies.



relationship with this clinical picture. In many cases, the syncope is clearly related to exercise (during or just after it), and on certain occasions it may be an individual that regularly practises sport, and that has presented a syncopal episode with no relationship at all to physical activity. In these cases, the characteristics of the clinical picture must also be thoroughly researched via a detailed anamnesis.

We can therefore classify athletes with syncope into two main groups depending on chronology of it:

Syncope related to exercise

On numerous occasions they may have a vasovagal or situational aetiology, etc. Dehydration and the reduction of the intravascular volume can induce a state of orthostatic hypotension and induce a pre-syncopal episode[10].

However, exercise-related syncopes are more worrying, and have been classically related to cardiovascular diseases[11]. For this reason, the athletes that lose consciousness during exercise require an exhaustive study to rule out underlying heart disease. Furthermore, we should assess whether the athlete should restrict his/her physical exercise until potentially malignant pathologies have been ruled out. In certain occasions, some carefully supervised training can be continued (monitored using a Holter, training vests, etc.), especially if we have immediate access to an automatic external defibrillator.

These kinds of syncopes may be the only symptom to precede sudden cardiac death[12]. The differential diagnosis should include numerous cardiological causes, such as: hypertrophic cardiomyopathy (HCM), congenital coronary abnormalities, right ventricle arrhythmogenic dysplasia (RVAD), channelopathies such as long QT syndrome (LQTS), or Brugada syndrome[13], myocarditis, *conmotio cordis,* etc. We should also take into account heat stroke or hydro-electrolytic alterations as aetiology of the exercise-related syncope.

Post-exercise syncope

We should distinguish this from the collapse framework associated with exercise, in which the athlete falls to the ground with no real loss of consciousness or cerebral hypoperfusion. In these cases, preferably non-cardiogenic causes should be ruled out, such as dehydration, hyponatremia, heat stroke, etc. When the syncope occurs immediately after exercise and is triggered whilst the athlete is standing, generally it is less worrying and usually has a benign aetiology.

Collapse associated with exercise can also be a symptom of exhaustion or may more commonly be a neuromediated syncope that has occurred after stopping quickly after exercise. Exercise leads to an increase in the heart rate (suppression of the para-sympathetic system and increase of sympathetic activity), an increase of contractibility and systolic volume, as well as the balance between sympathetic vasoconstriction in the inactive vascular beds and the increase of muscular-skeletal vasodilation of metabolic origin.

These physiological responses result in a marked increase of cardiac output, which is re-distributed to the active muscles. During exercise, the maintenance of the cardiac output increase will be pre-load-dependent and requires the peripheral muscle activity to return the venous blood to the heart. When exercise is stopped quickly, the pump stops working and the venous blood return to the heart is reduced, with the consequent decrease of end diastolic volume of the left ventricle, systolic volume, and therefore the cardiac output[14].

As such, neuromediated syncope is also frequent in these situations, though its mechanism is still little known today. One of these mechanisms described is the cardiac depressor reflex, also known as the Bezold-Jarisch reflex[15]. During the acute reduction of the pre-load and filling of the heart with the sustained elevation of the catecholamines, the increase of the myocardial contractility can lead to the activation of chemoreceptors and mechanoreceptors, and induce paradoxical bradycardia and hypotension.

In a study published several years ago regarding ultra-marathon races[16], it was estimated that 85% of athletes collapsed after the competition. Only a third of these runners had a medical condition behind this collapse (predominantly hydroelectrolytic disorders, heat stroke, etc.), whilst the rest quickly regained stability after being placed in the Trendelemburg position. On the other hand, all the runners that collapsed during the race had an unidentified medical condition.

Differential diagnosis

The differential diagnosis of syncope in athletes is vast, though currently we can divide them into two main categories: neurocardiogenic or cardiological syncope. Less frequent causes of syncope include eating disorders, substance abuse, chronic fatigue syndrome, psychiatric, neurological and metabolic disorders.

Neurocardiogenic syncope

This kind of syndrome consists in neurologically mediated events due to our body's sudden incapacity to maintain the blood pressure at the levels needed to maintain the brain flow. This term of neurocardiogenic syncope has been widely discussed by experts, including numerous aetiologies such as vasodepressor syncope, orthostatic syncope, cardioinhibitor syndrome, situational syndrome and carotid sinus syndrome[17]. Situational syncopes include episodes associated with urination, defecation, coughing, etc.

Although the exact mechanism of these clinical pictures is unknown, literature suggests that syncopal episodes are due to the fact that the organism is incapable of increasing the systematic vascular resistances efficiently in response to the significant reduction of the venous return.

Cardiogenic profile syncope

It is important to rule these kinds of pathologies out, given that to a large extend they determine whether the syncopal picture could be an important risk marker of sudden death. In turn we could

divide this kind of syncope into two subgroups, depending on if they are arrhythmic episodes or circulation disorders, or structural heart disease episodes.

Arrhythmic episodes or circulation disorders

Multiple arrhythmogenic pathologies are described that can cause syncopal events, and, in turn, that are potentially lethal: Brugada syndrome, long QT syndrome, Wolf Parkinson White[18], polymorphic ventricular tachycardia, idiopathic ventricular tachycardia, etc. As such, especially in athletes over 35 years, we must consider that ischemic heart disease may be a common cause of sudden death in athletes, also presenting a high arrhythmogenic load whilst physical exercise is being carried out. Likewise, we should take into account that we will frequently find athletes with different degrees of atrioventricular block (AV block), with the majority of them considered within the physiological alterations in the context of the heart of an athlete. Given the increase of vagal tone that they present, we frequently encounter findings such as sinus bradycardia, migratory pacemakers, 1st degree AV block, type I Mobitz 2nd degree AV block, pacing of the union, etc. As such, asymptomatic patients present pauses of less than 4 seconds, not requiring additional studies. However, we should rule out advanced circulatory disorders, such as significant pauses (greater than 4 seconds), type II 2nd Mobitz degree AV block or complete AV block, which could lead to a picture of dizziness or syncopal episodes.

Structural heart disease

The cardiogenic profile syncope can be related to those athletes that have structural cardiological pathologies, especially cardiomyopathies, coronary abnormalities and atherosclerosis. With suspected cardiological aetiology syncope, we should use image techniques to rule out the presence of myocardial disease, such as hypertrophic cardiomyopathy (most frequent cause of sudden death in the under 35s), right ventricle arrhythmogenic dysplasia, dilated cardiomyopathy, non-compacted cardiomyopathy, etc. In the event of this kind of pathology, a genetic study and family advice is recommended[19]. We should also rule out both coronary atherosclerosis and congenital coronary abnormalities. Other causes of structural heart disease, that may on rare occasions cause syncopal episodes or sudden death in athletes are: myocarditis, valvular heart disease, Marfan syndrome, etc.

The assessment of an athlete with syncope

Clinical history and anamnesis

In the assessment of an athlete that has revealed a syncopal picture, it is considered fundamental to carry out a clinical history and complete anamnesis, as they can identify the aetiology of the syncope. It is fundamental to distinguish if the syncope is related to exercise or immediately after performing exercise. It is also important to rule out the presence of relevant family antecedents. In the event that there is a significant family history of unexplained deaths or known genetic mutations, thorough research should be performed regarding the possible presence in family members of hypertrophic or dilated cardiomyopathy, long QT syndrome, right ventricle arrhythmogenic dysplasia, etc. On occasions it could be helpful for the athlete to provide a complete genealogy tree during the assessment to obtain a detailed and complete family history. Research should also establish if these athletes have consumed alcohol, pharmaceutical drugs, certain illegal drugs or substances that improve their physical performance and interfere in syncopal pictures.

It is important to assess the state of hydration and nutrition at the time of the event, environmental conditions, the activity of the patient immediately before the syncopal episode, the presence of auras or warning signs, etc. It should also be researched if the patient has presented prior symptomatology on any occasions: dizziness, nausea, pre-syncopal episodes, chest pain, palpitations or dyspnoea. As such, it is recommended to investigate the presence of cardiovascular risk factors that may suggest the existence of ischemic heart disease as a main diagnosis: smoking, arterial hypertension, dyslipidemia, *mellitus* diabetes, etc. We should take into account that athletes, occasionally, may mask or minimise symptoms so they are not excluded from carrying out professional physical activity.

On numerous occasions, athletes are not able to remember the events that occurred during the syncopal episodes, which is why it is very useful to question witnesses that saw the episode. If cardiopulmonary resuscitation has been carried out and an automatic defibrillator used, efforts should be made to obtain the records. It is recommendable to insist on the time frame of the clinical picture, reinforcing the clinical picture of the patient before, during and immediately after the syncopal episode. For example, certain pre-syncopal events may guide us to a differential diagnosis: a febrile condition in the days prior to the episode enables us to rule out the presence of Brugada syndrome, myocarditis, etc. The presence of a stress stimulus (including loud noises or getting into cold water) that precedes the syncope may suggest the diagnosis of a catecholaminergic tachycardia, long QT syndrome[20], etc.; whilst chest trauma could indicate a commotio cordis. Myoclonic tremors or the relaxing of sphincters could suggest certain convulsive activity, though patients with neuromediated syncopes often develop certain myoclonic movements that are confused with convulsive activity.

Physical exploration

It is essential to carry out a suitable physical exploration in the study of the syncope: neurological examination, cardiopulmonary auscultation (special manoeuvres can be carried out with the aim of ruling out specific pathologies, such as in the case of HCM or mitral valve prolapse), the presence of peripheral pulses (Table 1). This will help us to rule out predominantly different types of valvular heart diseases or aorta diseases[21].

The physical examination should also include an assessment of the vital signs, which predominantly includes heart rate and blood pressure.

Table 1. Suggestive findings in physical exploration with associated suspected diagnostics.

Findings in the physical examination	Suspected diagnosis
Episode triggered immediately after standing up	Orthostatic hypotension or orthostatic postural tachycardia
Heart murmur	Significant heart valve diseases
Dynamic abnormal heart exploration	Structural heart disease
Weak pulse in lower extremities Difference in pressure between upper and lower limbs	Coarctation of the aorta
Pulse in slow ascent	Aortic stenosis
Bisferious pulse Increase in the intensity of the murmur following the Valsalva manoeuvre	Hypertrophic cardiomyopathy
Chest deformity Joint hypermobility Kyphoscoliosis Palate with pronounced arch	Marfan Syndrome
Wheezing	Asthma, anaphylaxis

These measurements should be performed after 3-5 minutes of rest, both standing and in the supine position. Likewise, we should measure the blood pressure both in the upper extremities as well as the lower limbs so as to rule out coarctation of the aorta. It is important to carry out an appropriate inspection of the athlete, as well as, for example, phenotypical characteristics that could suggest the existence of Marfan syndrome: *pectus excavatum*, scoliosis, joint hyper-flexibility, etc.[22].

Resting electrocardiogram

It is key to perform a 12-lead electrocardiogram (ECG) on athletes that have presented a syncopal condition[23]. The majority of disorders associated with a greater risk of sudden cardiac death, such as cardiomyopathies and channelopathies, present abnormal findings in a basal electrocardiogram.

However, the interpretation of the ECG in athletes requires a careful analysis to properly distinguish the physical changes related to athletic training with suggestive findings of an underlying pathological condition.

The prevalence and significance of electrocardiographic alterations in the heart of athletes have been the motivation behind numerous studies and discussions. There are currently criteria that help discern whether or not these changes are related to physiological adaptations or are suggestive of structural heart disease. Some of these criteria often fall into a grey area, in which discerning the pathological from the normal can be complicated, which is why on occasions a strict follow-up is required as well as a certain degree of "un-training" so as to perform a suitable assessment. Both the Seattle criteria and current guidelines from the European Cardiology Society in the interpretation of electrocardiographic alterations of athletes classify these findings as those that are common among athletes and related to the adaptation to exercise, and findings that do not appear to be related to training and that require a more thorough study to exclude underlying heart pathologies. Recently, refined criteria have been developed, which integrate and improve both previous classifications; presenting even greater sensitivity and specificity; with a significantly lower rate of false positives[24].

We frequently encounter physiological adaptations in athletes that align with the vagal hypertonia that athletes usually present: sinus bradycardia, 1st degree AV block, Mobitz I 2nd degree AV block. However, these findings alone do not justify the presence of the syncopal condition, which is why other causes must be ruled out[25].

As we have previously commented, in the electrocardiographic assessment, we are going to encounter findings within normality or those that suggest physiological alterations. However, these findings should not give the doctor a false sense of security, as multiple potentially lethal pathologies (atherosclerosis, coronary abnormalities, non-compacted cardiomyopathy, etc.) can be clinically silent in the resting ECG. Likewise, the ECG in athletes may orientate us towards a diagnosis upon revealing data compatible with Wolff-Parkinson White Syndrome (WPW), supraventricular arrhythmias (atrial fibrillation, etc.), myocardiac ischemia, or channelopathies such as the Brugada syndrome or long QT syndrome.

Despite the sensitivity being very variable, various studies suggest that approximately half of cardiovascular diseases can be detected in asymptomatic athletes following an ECG.

Laboratory

Laboratory trials can prove highly useful, especially if there is a certain previous focus on the possible aetiology of the syncopal condition. It is convenient to carry out a haemogram if there is suspected anaemia, frequently in older patients with suspected active bleeding, or women during menstruation.

If the syncope has been triggered in a possible context of arrhythmia or extreme dehydration, the electrolyte levels or those of other metabolic alterations should be assessed, especially alterations of sodium, potassium and other ionic disorders. Myocardial damage markers should be required (creatine kinase, troponine, etc.) when faced with a suspected condition of acute myocarditis, acute coronary syndrome, etc. As such, it is important to consider that on multiple occasions we can encounter normal laboratory findings in the study of syncope, which is why it should merely be considered a tool to complement the diagnosis, and not useful in excluding primary cardiac disorders.

Transthoracic echocardiogram

Today, the echocardiogram is a fundamental test that largely helps us to establish the definitive diagnosis or to rule out the presence of structural heart disease.

This result is highly useful in confirming the findings suggested in the electrocardiogram, as well as in studying the dimensions of the heart cavities and the parietal thickness, ventricular function, dilation of aortic root, etc.[26]. Furthermore, it is currently considered to be the gold standard in establishing the diagnosis of different cardiomyopathies (hypertrophic cardiomyopathy, dilated cardiomyopathy, etc.) or significant heart valve diseases.

As such, if the syncopal condition is highly suggestive of neurological or vasovagal aetiology and the electrocardiogram is completely normal, performing an echocardiogram may not be strictly necessary.

Advanced imaging techniques (Cardiac magnetic resonance/Computerised tomography)

Advanced imaging techniques help us to study different types of structural heart disease more precisely. These non-invasive tests can be highly useful when defining the myocardium and coronary anatomy, especially if they cannot be correctly assessed in the echocardiogram.

A cardiac CT should be performed when faced with a suspected presence of congenital coronary abnormalities or the presence of ischemic heart disease with a low pre-test probability[27]. We should consider that this study exposes the athlete to high levels of radiation, which is why it is recommended to exercise precaution during follow-up.

Magnetic resonance is currently the gold standard in performing a definitive diagnosis of right ventricle arrhythmogenic dysplasia[28]. Via this technique, the myocardium and right cavities can be studied with better image resolution. As such, the different late enhancement models with gadolinium and the oedema sequences may help to establish the presence of myocardial fibrosis, myocarditis, non-compacted cardiomyopathy, etc.

Exertion test

The cardiac stress test may be a very useful tool in handling the syncope, with the carrying out of an echocardiogram prior to use with the aim of ruling out structural heart disease also being recommendable. It is important to carry out an exertion test in athletes that present recurring syncopes during exercise; given that monitoring during the exertion will help us to discern between different aetiologies, such as arrhythmic episodes, reflex syncopes, etc.

The habitual protocols used in clinical practice (Bruce, Naughton, etc.) are short-duration exercise protocols of low-intermediate intensity, for which on occasions they may not provoke the symptoms in the athlete. Currently, the performance of an exertion test is recommended, which simulates physical activity during which the syncopal episode occurs[29]; i.e. individualised protocols should be performed based on the sporting activity carried out or on the setting in which it develops. For example, athletes with a high aerobic component, such as long-distance runners or triathletes, should keep a constant or lightly ascending rhythm over a long period of time. Conversely; athletes with anaerobic predominance should carry out interval training with high-speed races and intermittent resting periods.

Therefore, the cardiac stress test may be highly useful, especially if we suspect arrhythmic aetiology of the syncopal event. The exertion test also helps us rule out ischemic heart disease, to assess the functional capacity of the athlete and the induction of arrhythmic events, whether during the exertion phase or the recovery phase.

Holter

The Holter test provides us with an electrocardiographic record for 24 hours, helping us rule out the presence of significant pauses as well as paroxysmal arrhythmic events.

It is recommendable to position this device when the athlete is going to perform his/her habitual sporting activity. In the event that competitive physical activity has been restricted, the performance of recreational physical activity could be recommended during the time that the electrocardiographic monitoring is being carried out[30].

However, the Holter has low sensitivity, and on most occasions we will find certain limitations when it comes to achieving significant findings during monitoring. For this reason, in cases in which there is still a high suspicion of cardiogenic syncope due to the presence of arrhythmias or circulatory alterations, it could be useful to position a more prolonged recording device or an implantable Holter. Furthermore, once the athlete returns to his/her habitual sporting activity after having ruled out underlying heart disease, it is recommended that eventual electrocardiographic monitoring continues during high intensity training sessions.

Tilt - test

The tilt test is a non-invasive study method which studies the changes that take place in the blood pressure and heart rate depending on the postural angles, predominantly during the prolonged standing position. It is mainly indicated in the study of athletes with suspected pre-syncopes, repeated syncopes of vasovagal aetiology, and in the study of other alterations to the autonomous nervous system.

In different studies, it has been demonstrated that this test has low sensitivity, and a high rate of false positives in athletes[31]; which is why

it should not be used as the main tool in establishing the diagnosis of the syncope in the athlete.

Electro-physiological study

The electro-physiological study (EPS) is a useful tool, though uncommon in the study of syncope; though today it is estimated that only 2% of patients with syncopes of unknown aetiology undergo an EPS[32].

This test is highly effective when it comes to inducing supraventricular arrhythmias and ventricular tachycardias. For this reason, the EPS may be used in athletes to confirm and treat arrhythmias targeted during the diagnostic study. This test is highly useful in patients with suspected Wolff-Parkinson White Syndrome, intranodal tachycardia, etc.; in which the excision of anatomical substrate is also attempted.

With regards to ventricular tachycardias (VT), the recovery rate after excision with radio-frequency is very high in idiopathic tachycardias in the absence of structural heart disease. However, in cases of VT in patients with structural heart disease[33], such as coronary disease and established myocardial scarring, excision may not be considered curative, given that they present a very high risk of recurrence and of presenting sudden death during follow-up.

Therapeutic attitude

Managing the syncope should fundamentally focus on establishing the safety of the athlete. These athletes should be remitted following the clinical picture, to doctors with experience in the diagnosis and treatment of syncope in athletes. These athletes should suspend sporting practice until the study has been completed. There are some centres that promote an organisation model of managing the syncope, via multidisciplinary approach units, as well as the importance of risk assessment scales following a syncopal condition[34].

The objectives of this assessment are fundamentally based on excluding underlying structural heart disease that could lead to sudden death, therefore enabling the athlete to return safely to physical activity. Patients with benign syncope aetiologies (orthostatic, vasovagal, etc.) may continue with sporting activity after establishing the suitable guidelines. However, in those that are still undiagnosed after the complete study is over, strict follow-up should be performed when it comes to assessing re-incorporation into habitual physical activity[35]. Patients that present a high risk of sudden death, unchangeable with treatment or therapeutic intervention, should restrain from competitive sport and limit physical activity to merely the recreational kind.

In athletes with neurocardiogenic profile syncope, it is important to avoid triggering factors. A suitable intravascular volume should be maintained, which is why good hydration is recommended, preferably with isotonic drinks. Certain simple manoeuvres may help avoid this kind of condition; usually involving the exercising of the lower extremity muscles: isometric contractions, crossing legs, Trendelemburg position, squatting position, etc. (Figure 2). However, these manoeuvres will only be useful in the event that athletes present warning signs or symptomatology prior to the syncopal episode to have time to perform them[36].

Although certain medications can be used (alpha-agonists, disopyramide, beta-blockers, etc.) in this kind of syncope, pharmacological treatment is not clearly recommended at the current time, given that it has not given significantly favourable results[32]. As such, high-risk sports should be avoided (motorcycling, cycling, etc.) in athletes that present recurring syncopal episodes, despite the aetiology being benign.

According to the 36th Bethseda Conference[37], the suspension of sporting activity is recommended in athletes that have presented potentially malignant aetiology syncopal episodes, especially with cardiological causes. In the case of athletes with an implantable automatic defibrillator (IAD), the clinical practice guidelines and current recommendations are quite restrictive regarding physical activity. However, recent data affirms that even competitive activity may be safe in those athletes, as long as a customised assessment is carried out of the patient and of the base pathology, and an activity level is adjusted to the programming of the device[38].

Conclusion

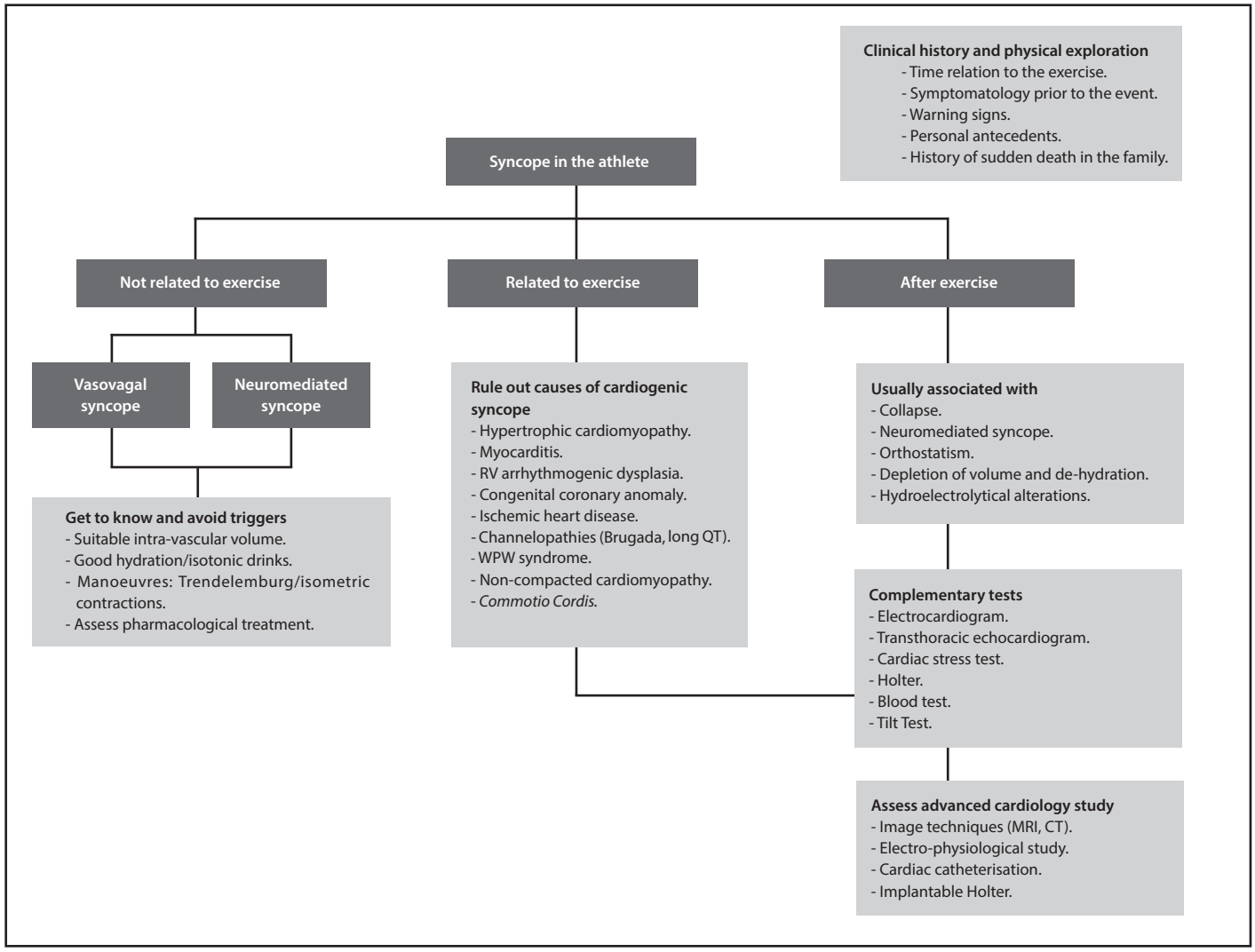
The appropriate assessment of the syncope continues to be a subject of debate, given the wide spectrum of circumstances, both benign and potentially dangerous, whose first revelation may be this symptom. The assessment of the symptomatology in the demographic of athletes also implies an added difficulty, as well as the conditions in which the syncope occurs, and the implications that ceasing sporting activity at competitive level entails. Today we frequently face the challenge of carrying out a suitable assessment in athletes that have presented syncopes. Cardiovascular adaptations through sport often overlap with initial states of heart diseases, whose cardinal symptom is the syncope, thus increasing the diagnostic difficulty. The amount of evidence currently available is wide; aside from a detailed clinical history, physical exploration and an electrocardiogram, it is not uncommon to require further tests to help establish a differential diagnosis. Carrying out these tests may follow a rational and phased sense, depending on the findings discovered during the study of the syncope. On occasions, despite having an exhaustive assessment, doubts may persist regarding the benign nature of the syncope, which is why follow-up and often ceasing physical activity may form part of the diagnosis.

Based on this, the main objective of the assessment of the athlete with syncope is to establish whether or not there is a potential risk of sudden death and to establish the safety of physical activity.

An error in the diagnosis may have devastating consequences, but on the other hand, the cost of diagnosing a benign condition as dangerous or potentially lethal can lead to the unnecessary restriction of physical activity, with negative consequences for the athlete.

To conclude, the assessment of syncope in athletes has certain characteristics that distinguish it from the general population. A careful and phased assessment can help us to not make errors in the diagnosis

Figure 2. Algorithmic assessment focus of an athlete with a syncopal picture.



and to not increase the anxiety linked to a pathology that in the large majority of cases is benign.

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