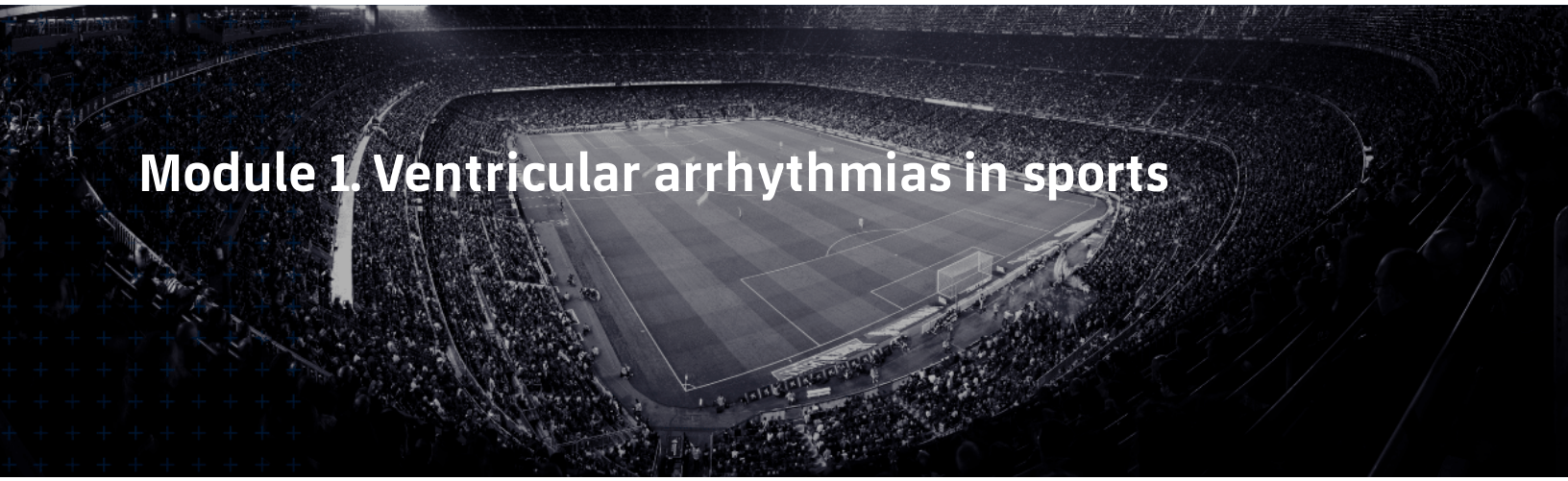

Module 1. Ventricular arrhythmias in sports

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Introduction

Just as in the general population, atrial and ventricular arrhythmias are documented with some frequency in athletes. They can present clinically with a variety of symptoms, including palpitations, syncope, or they may be completely asymptomatic. Occasionally, reduced performance or general discomfort may be reported. Syncope during exercise is critically important.

Sudden cardiac death (SCD) associated with athletic activity is a rare but devastating event. The victims can be young and seemingly healthy, and while many of these deaths are unexplained, a considerable number have an underlying, previously undiagnosed cardiovascular disease. Therefore, there is great interest in early identification of individuals at risk for whom appropriate activity restrictions can be implemented to minimize risk.

In this regard, a pre participation evaluation is important, based on age; in young athletes (←35 years old), the medical history, physical examination, and a 12 lead electrocardiogram (ECG) should be assessed, while in middle-aged and older athletes, established risk

scores (e.g., SCORE2 risk) should be used for evaluation. Although echocardiography has been shown to increase the sensitivity of structural heart disease detection, at present, it is not feasible as a routine test in mass screening due to accessibility issues. If pathological data are found in the initial evaluation, second line tests, such as echocardiography, 24 hour Holter recording, stress test and cardiac resonance, are performed.

It is essential to have an in depth knowledge of the electrocardiographic changes derived from sports practice in order to differentiate them from those pathological changes that reflect an underlying cardiac pathology. This will avoid unnecessary tests and allow for the early diagnosis of cardiac pathologies that could cause sudden cardiac death.

Most SCD events in athletes are the consequence of malignant arrhythmias, usually sustained ventricular tachycardia (VT) or ventricular fibrillation (VF). In individuals with certain cardiac disorders (e.g., hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, etc.), high intensity sport may increase the likelihood of VT/VF in two ways:

- In susceptible individuals (e.g., with structural heart disease, channelopathies, etc.), prolonged physical training may induce adaptive changes in cardiac

structure (e.g., interstitial fibrosis, alteration of normal myocardial architecture, right and left ventricular dilatation), which may influence the progression of a pathological arrhythmogenic substrate.

- The immediate physiological demands of intense sports (e.g., mechanical stress, increased myocardial oxygen consumption, hemodynamic overload, catecholamine release, electrolyte imbalance) can trigger malignant arrhythmias in susceptible individuals with underlying cardiac abnormalities.

After identifying the type of ventricular arrhythmia and the risk of SCD associated with it, the type of treatment to be performed will be determined, either pharmacological or the implantation of implantable cardioverter defibrillator (ICD). Additionally, individualized sports recommendations will be provided.

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1. Definitions

Subtypes of ventricular arrhythmia

- **Premature ventricular complex (PVC):** Premature appearance of an abnormally wide QRS complex (typically →120 ms duration, corresponding T wave typically wide and in the opposite direction to the main QRS deflection, with no preceding P wave). These can be the following:

  - Unifocal or monomorphic premature ventricular contractions (PVC): PVCs with a single QRS morphology.
  - Multifocal, multiform or polymorphic PVCs: PVCs with different QRS morphologies.
  - Short coupled PVCs: PVC interrupting the T wave of the preceding conducted beat.
  - Variable coupled PVCs.

- **Ventricular tachycardia (VT):** →3 consecutive beats with a rate >100 beats per minute originating in the ventricles, independent of atrial and atrioventricular (AV) nodal conduction.

- **Non sustained ventricular tachycardia (NSVT):** →3 consecutive ventricular beats persisting for 3 beats and less than 30 seconds.

- **Monomorphic ventricular tachycardia (MVT):** Same QRS morphology beat to beat.

- **Polymorphic ventricular tachycardia (PVT):** Continuous change of QRS morphology.

- **Sustained monomorphic/polymorphic ventricular tachycardia (SMVT/SPVT):** Continuous VT for at least 30 s or requiring intervention for termination.

- **Bidirectional ventricular tachycardia:** Beat-to-beat alternation of the frontal QRS axis.

- ***Torsades de Pointes* (TdP) ventricular tachycardia:** A subtype of polymorphic VT in the context of QT interval prolongation with QRS complexes that continuously change and appear to twist around the ECG baseline in a sinusoidal pattern, generally non sustained but frequently degenerating into ventricular fibrillation.

- **Ventricular fibrillation (VF):** A chaotic rhythm with undulations in timing and morphology, without discrete QRS complexes on the surface ECG.

- **Electrical storm:** VT occurring 3 or more times in 24 hours (separated by at least 5 min), each requiring termination by intervention.

- **Incessant VT:** Continuous sustained VT that rapidly recurs despite repeated intervention for termination over several hours (Zeppenfeld et al., 2022, p. 15).

Sudden cardiac death

- **Sudden cardiac arrest (SCA):** Sudden cessation of normal cardiac activity with hemodynamic collapse.

- **Sudden cardiac death (SCD):** Death presumed to be of cardiac cause occurring within one hour of the onset of symptoms in witnessed cases, and within 24 hours of last being seen alive when not witnessed. SCD in autopsied cases is defined as the natural unexpected death of unknown or cardiac cause.

- **Sudden unexplained death:** It occurs in an individual older than 1 year old.

- **Sudden arrhythmic death syndrome (SADS):** Sudden unexplained death occurring in an individual older than 1 year old with negative pathological and toxicological assessment. (Zeppenfeld et al., 2022, p. 15).

Syncope

- **Drop attack:** Loss of postural tone, without loss of consciousness.

- **Absence:** Loss of consciousness, without loss of postural tone.

- **Syncope:** Simultaneous loss of consciousness and postural tone.

- **Unexplained syncope:** Transient loss of consciousness due to cerebral hypoperfusion, characterized by a rapid onset, short duration, and spontaneous complete recovery, but unexplained after conventional workup.

- **Arrhythmic syncope:** As above, but highly suspicious for intermittent bradycardia, rapid supraventricular tachycardia (SVT) or ventricular arrhythmia (VA). (Zeppenfeld et al., 2022, p. 15).

Populations of athletes and the level of competition they participate in

- **Young athletes:** They are younger than 35 years old, in whom SCD is usually due to a variety of congenital heart diseases (Maron et al., 1996).

- **Master athletes:** Individuals aged →35 years in whom SCD is most commonly associated with coronary heart disease (Karam et al., 2018).

- **Elite competitive athletes:** They regularly participate in high level training and competition and may not have the will or judgment to limit their activity (Maron et al., 2004).

- **Recreational athletes:** They participate for health and/or fun and generally do not have the same pressures to excel. Activity levels can still be vigorous, and the distinction from competitive sport can be elusive in the individual case (Franklin et al., 2020).

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2. Epidemiology

According to D'Ascenzi et al. (2016), ventricular arrhythmia (i.e., >1 PVC on standard 12 lead ECG) is uncommon in athletes, and it is not different from the general population; it is present in more than 1% of athletes examined with ECGs. If more frequent premature ventricular complexes (PVC) are recorded on a standard ECG (10 seconds), the athlete is likely to have a high PVC burden on a 24-hour recording. While PVCs are likely to be benign in a highly trained athlete, their presence may be the hallmark of underlying heart disease and requires careful assessment (Biffi et al., 2004; Biffi et al., 2002).

The incidence of SCD in athletes increases with age, as it does in the general population(Pelliccia et al., 2021). In apparently healthy athletes (>35 years old), the estimated incidence of SCD ranges from 2 to 6.3 per 100,000 participant-years. In comparison, in young competitive athletes (←35 years old), the incidence of fatal events is significantly lower, 0.4-3 per 100,000 participant years (Risgaard et al., 2014). Female athletes have a low risk of SCD; on average, 1 in 14 cases of SCD occurs in female athletes (Rajan et al., 2022).

Incidence data are imprecise, as most are derived from retrospective analyses, and incidence varies depending on the intensity of exercise, the athletic population considered, the time period of observation, and whether the definition of athletic SCD encompasses SCD outside of sport/exercise (Phillips et al., 1986).

The magnitude of the problem and the inherent challenges of detection are illustrated in reports of arrhythmic events associated with major endurance sports events (Maron et al., 1996). Although the incidence of SCD among marathon runners is low (one death per 215,000 hours), it is higher than for other types of exercise, such as non competitive jogging (one death every 396,000 hours), cross country skiing (one death per 607,000 hours) or general non competitive exercise (one death per 375,000 hours) (Hillis et al., 1994).

3. Clinical presentation

When present, symptoms should be evaluated to determine if any action is necessary. Symptoms may be related to the arrhythmia itself (e.g., palpitations); to hemodynamic consequences (e.g., dyspnea, dizziness, syncope, presyncope, chest pain, SCD); most often, palpitations, which may be associated with diaphoresis, lightheadedness or dizziness; shortness of breath or chest discomfort due to increased pulmonary vascular pressure and/or congestion. The least common presentation is syncope and sudden cardiac arrest (Maron et al., 1996).

- **Palpitations:** Unpleasant perception of strong, rapid or irregular heartbeat. Occasionally, patients may describe the sensation as a rapid fluttering in the chest, a flip flop feeling in the chest or a feeling of palpitations in the chest or neck, and these descriptions can help to elucidate the cause of the palpitations (Hastings & Levine, 2012).

- **Syncope and presyncope:** Syncope (and presyncope, with lightheadedness or dizziness) in an athlete is an important symptom that requires a thorough evaluation. Syncope occurring during exertion suggests a life threatening arrhythmic etiology (e.g., aortic stenosis, hypertrophic cardiomyopathy [HCM], ventricular arrhythmia, etc.) and should be evaluated with great seriousness and urgency. On the other hand, syncope during the recovery phase after exertion (e.g., during the cool down period) is generally non arrhythmic and is more likely due to a vagal reflex (Sakaguchi et al., 1996; Shen et al., 2017).

- **Reflex syncope (neurally or vaso-vagally mediated):** A common cause in young athletes that is generally not associated with heart disease and indicates a benign clinical outcome (Sakaguchi et al., 1995; Calkins et al., 1995). It is due to mechanisms mediated by neural pathways. However, hypovolemia due to unreplenished fluid losses may contribute in athletes. Athletes (especially those participating in endurance disciplines) may be more susceptible to neurally mediated syncope because of the nature of their increased vagal tone (Hastings et al., 2012). Usually, they do not require further studies. However, if there are worrisome features of structural heart disease or cardiac cause of syncope, further evaluation is necessary (Colivicchi et al., 2002).

- **Cardiogenic syncope:** Causes include ventricular tachycardia associated with arrhythmogenic cardiomyopathies or obstruction resulting from HCM or aortic stenosis, and hypotension, due to vagally mediated vasodepression in patients with HCM (Shen, et al., 2017).

- **Exercise associated syncope:** It may also be related to hyponatremia or hyperthermia as a result of intense or prolonged exercise (Shen, et al., 2017).

- **Sudden cardiac arrest:** This is a rare and devastating event. Malignant arrhythmias, usually VT (ventricular tachycardia) or VF (ventricular fibrillation), are responsible for sudden cardiac arrest (SCA) in athletes (Colivicchi et al., 2004). Such arrhythmias usually occur in the context of underlying structural heart disease (e.g., HCM, arrhythmogenic cardiomyopathy, etc.) or previously undiagnosed primary electrical disease (e.g., Brugada syndrome, long QT syndrome, etc.) (Calkins et al., 1995). Pre-participation screening of athletes is largely aimed at identifying such underlying cardiac conditions (Hastings & Levine, 2012).

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4. Diagnosis and diagnostic evaluation

Pre-participation cardiovascular screening has the potential to allow for the identification of athletes at risk for cardiovascular disease prior to the onset of symptoms. The assessment protocol should be tailored to the athlete's age to account for age specific cardiovascular disease (Corrado et al., 1998; Baggish et al., 2010).

Pre-participation screening includes medical history, physical examination and ECG, these are effective in identifying cardiovascular disease in young athletes (←35 years old) by identifying relevant symptoms (e.g., exertional syncope) or ECG abnormalities compatible with inherited cardiomyopathies or channelopathies (Corrado et al., 2006; Steinvil et al., 2011). Although echocardiography can increase the sensitivity of detecting structural heart disease (SHD), since it is not universally available, it is not feasible as a routine screening test in mass detection. More cardiovascular diseases are identified through serial (annual) evaluations of adolescent athletes (Sarto et al., 2021; Tesch, 1985).

The prevalence of false-positive results is highly dependent on the criteria used to define an ECG as **abnormal** (Tesch, 1985; Sharma, 2018). In case of **abnormal** ECG findings, complementary tests will help in the diagnosis. Athletes diagnosed with clinically relevant cardiovascular disease are treated under the available clinical practice guidelines (Piepoli et al., 2016; Heidbuchel et al., 2021).

In master athletes, the most common cause of SCD is coronary artery disease (CAD) (Corrado et al., 2011). Before participating in vigorous physical activity, they must be assessed using risk scoring systems such as ESC SCORE2 (Pelliccia et al., 2021).

Medical history, directed anamnesis and physical examination

These elements should focus on warning signs, including characteristics of arrhythmic syncope, such as the absence of a vagal prodrome, and a family history of premature SCD (<40 years), including, for example, drowning or car accidents in long QT syndrome (LQTS) and catecholaminergic polymorphic ventricular tachycardia (CPVT) (Brignole et al., 2018). Subtle features suggesting hereditary causes include a family history of possible epilepsy, sudden infant death syndrome, congenital deafness (LQTS), heart failure, or pacemaker implantation before the age of 50. Characteristics of diseases related to proarrhythmic conditions include a midsystolic click in mitral valve prolapse (MVP) and murmurs in the outflow tract with Valsalva in HCM. Specific skin features may be relevant, e.g.,

lupus pernio, erythema nodosum in sarcoidosis, angiokeratoma in Fabry disease, xanthelasma/xanthoma and palmoplantar keratosis in arrhythmogenic cardiomyopathy (Zeppenfeld et al., 2022).

ECG in athletes

ECG findings in athletes may be physiological consequences of cardiovascular adaptation to regular physical training or may be the expression of pathological conditions (Maron et al., 2015). Therefore, there is a need for proper knowledge of what is normal and abnormal in an athlete's ECG (Brosnan et al., 2014). For practical purposes, these changes can be classified into 3 groups: the first is related to chronic training (normal ECG changes in athletes); the second is associated with expression of an underlying pathological condition (e.g., hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, etc.), which are associated with increased risk of SCD; and the third includes a gray or borderline area of ECG changes, left or right axis deviation, left or right atrial enlargement, and complete right bundle branch block (RBBB). The presence of a single borderline abnormality is likely to be unrelated to structural cardiac abnormalities (Sharma et al., 2018).

Additional tests

The primary focus of any additional testing is to document the presence (or absence) of underlying structural heart disease.

1. **Cardiac imaging:** Athletes with a known arrhythmia, or those with a high suspicion of arrhythmia, should undergo a transthoracic echocardiogram. If echocardiographic imaging is not considered diagnostic, additional imaging with cardiovascular magnetic resonance (CMR) should be performed.

2. **Stress test:** Exercise stress testing is warranted for athletes with symptoms suggestive of arrhythmia during exertion. In patients with symptoms of arrhythmic origin, it aims to assess the athlete's hemodynamic behavior during exercise (i.e., heart rate and blood pressure response to exercise) and the reproducibility of symptoms, as well as the potential recording of arrhythmia.

3. **Ambulatory ECG monitoring:** For athletes in whom an arrhythmia is highly suspected based on presenting signs and symptoms, but whose initial ECG is unrevealing, we perform ambulatory ECG monitoring. Ambulatory ECG, especially long-term recording (two to four weeks, for example, with the Nuubo™ or SpiderFlash™ systems), significantly increases the likelihood of capturing abnormal heart rhythms and confirming the diagnosis.

Electrophysiological studies

These studies:

They include the measurement of reference intervals (e.g., the atrial-His [AH] interval and the His-ventricular [HV] interval), programmed electrical stimulation (PES), and electroanatomic mapping, which can be used for diagnostic purposes and to guide therapy. The performance of an electrophysiological study (EPS) varies depending on the underlying heart condition and its severity, the presence or absence of spontaneous VT, concomitant pharmacological therapy, the stimulation protocol, and the stimulation site or sites. Typical protocols include stimulation of 2 right ventricle (RV) sites with 2-3 basic conduction cycles, introduction of 3 extrastimuli and administration of isoprenaline.

In the current era, programmed electrical stimulation is mainly employed to confirm the diagnosis of VT and induce mappable VAs with non inducibility being an ablation endpoint. In patients with structural heart disease and mildly reduced or preserved LVEF who present with unexplained syncope, induction of SMVT with programmed electrical stimulation can be helpful to

identify the underlying cause and to predict subsequent events. PVT/VF induction in structural heart disease is, in general, considered as a non specific finding (Zeppenfeld et al., 2022, p. 4015)

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5. Etiology of sudden death

It depends on whether there is structural heart disease or not.

1. **Structural heart disease:** SCD in athletes often occurs in the presence of a structural heart disease, although the underlying disorder is usually not detected until the arrhythmic event occurs. The etiology varies by age.

    - **Athletes younger than 35 years:** Several large series have evaluated SCD in athletes younger than 35 years (Maron et al., 1996; Maron et al., 2007; Maron et al., 2015). In most cases, structural heart disease was present, although contemporary data suggest an increase in arrhythmic sudden death with a structurally normal heart (Franklin et al., 2020; Finocchiaro et al., 2016). When the following are present: HCM, anomalous origin of a coronary artery, congenital aortic valve disease (CAVD), myocarditis, and coronary atherosclerosis, though with some

variation across different series (Finocchiaro et al., 2016).

- **Athletes →35 years:** Commonly referred to as master athletes, coronary artery disease is the predominant cause of SCD during exercise (Karam et al., 2018; Baggish et al., 2017).

Structural heart disease may increase the risk of SCD through one or more of the following mechanisms:

- The most frequent are VTs, which are usually caused by reentrant arrhythmias that develop in abnormal myocardium and/or areas of fibrotic tissue replacement in the myocardium.
- Rare mechanisms include bradyarrhythmia or asystole due to the pathological process extending into the conduction system, causing complete heart block without an adequate escape rhythm.
- Syncope, besides arrhythmic causes, can be due to outflow tract obstruction in HCM and aortic stenosis, as well as cyanosis during exercise in the context of certain congenital lesions with right-to-left shunts.
- Dissection of large vessels, as in patients with Marfan syndrome.

2

Primary electrical disease: Series of patients with SCD have shown an increasing number with normal autopsies, although there is concern that cardiac abnormalities have been missed. Several inherited arrhythmic syndromes predispose individuals to SCD, and usually, the heart is structurally normal. These syndromes can be the following: long QT syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, short QT syndrome, early repolarization syndrome.

In addition, in individuals with structurally normal hearts, arrhythmic events may be triggered by trauma or occur as sporadic/idiopathic phenomena, such as commotio cordis, where SCD results from a blow to the precordium by a projectile object like a baseball, hockey puck, football helmet, or fist.

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6. Ventricular arrhythmias

The general view on the association between sports and arrhythmias is that exercise sets the stage for an arrhythmia in the context of an underlying, pre existing condition, whether structural, electrical, hereditary, or acquired (Benito et al., 2011; Sawant et al., 2014). The relationship between sport and arrhythmias can be understood along three lines: sport as a trigger of arrhythmias on an underlying problem, sport as a promoter of the arrhythmic substrate, or sport as an inducer of the substrate. The athlete's heart, a heart that adapts so magically to cope with the demands of exercise, harbors many structural and functional changes that themselves predispose to the development of arrhythmias, at the atrial, nodal and ventricular levels. In essence, the athlete's heart is a proarrhythmic heart (Heidbuchel, 2018). Adolescents and young adults who participate in competitive sports activities have a risk of sudden cardiac death (SCD) that is three times higher than their sedentary counterparts. Sport, per se, was not a cause of increased mortality, but triggered SCD in athletes who were affected by cardiovascular conditions predisposing to life threatening ventricular arrhythmias during physical exercise (Corrado et al., 2003). This explains why recommendations for sports

participation in people with arrhythmogenic conditions are so complex.

"Managing sports participation in individuals with arrhythmogenic conditions is guided by three principles:

1. Preventing life-threatening arrhythmias during exercise.
2. Managing symptoms to allow sports.
3. Preventing potential sports-induced progression of the arrhythmogenic condition." (Pelliccia et al., 2021, p. 73).

PVCs/VT in patients without structural heart disease are defined as idiopathic, based on a negative history and a normal physical examination. 12-lead ECG and transthoracic echocardiography are the first important diagnostic steps followed to exclude underlying structural heart disease. Typically, 24-hour Holter ECG monitoring is performed to determine the PVC load. It is necessary to recognize multiform PVCs on prolonged ECG monitoring and subtle changes on ECG or echocardiography. CMR should be performed whenever ECG and echocardiography are inconclusive to rule out structural heart

disease, or the clinical presentation raises suspicion (Zeppenfeld et al., 2022).

Ventricular extrasystole/ventricular premature complexes (PVC) and NSVT

Frequent and complex PVC detected during cardiovascular screening of the athletic population may be a sign of underlying cardiovascular disease, causing risk of sudden cardiac death (SCD), but are also often recorded in trained athletes without cardiovascular abnormalities. The interpretation of PVCs could present a clinical dilemma in the general population, particularly in athletes. The initial evaluation should involve an algorithmic approach to risk stratification. While certain characteristics of premature ventricular beats (PVB) are considered common and benign, others are rare in athletes and may suggest underlying cardiovascular disease. Evaluation should begin with a complete medical history, exercise symptoms, family history, race, and ethnicity of athletes and their families, as well as origin from regions with endemic diseases that predispose individuals to heart conditions, arrhythmias, and sudden death (SCD), along with substance use or recreational drugs, and comorbidities. This is the initial step to suspect that these may lead to complex arrhythmias and SCD in athletes. Physical examination helps identify associated cardiac and non cardiac conditions, where the presence of PVC may raise suspicion of associated diseases and SCD risk. The PVC burden and morphology, along with imaging and exercise testing, can provide

information about diagnosis and risk stratification, while management and sports eligibility largely depend on symptoms and underlying etiology.

Epidemiology

The prevalence of PVCs is directly related to the study population, the detection method and the duration of observation. PVCs are more likely to be detected in older patients, patients with more comorbidities, and patients who are monitored for longer periods of time (Marcus, 2020). In patients without known heart disease, PVCs have been observed in approximately 1% of routine 12-lead ECGs of 30 to 60 seconds duration and in up to 6% of ECGs of 2 minutes duration (Simpson et al., 2002; Jouven et al., 2000). In comparison, when 24-hour ambulatory monitoring is used, up to 80% of apparently healthy people have occasional PVCs (Sobotka et al., 1981). The occurrence of frequent PVCs accounting for more than 20% of overall heartbeats is rare, observed in less than 2% of patients (Scorza et al., 2022).

There is an age-related increase in the prevalence of PVCs in both healthy individuals and those with underlying heart disease (Simpson et al., 2002; Glasser et al., 1979). The prevalence of PVCs increases with age and in the presence of other factors, such as a faster sinus rate, hypokalemia, hypomagnesemia, and hypertension (Simpson et al., 2002). Generally, a **normal number** of PVCs in an adult is considered to be >500 in a 24 hour period (Kostis et al., 1981).

It is unclear whether PVCs are observed more frequently in athletes than in their sedentary counterparts. In a group of 355 elite Italian athletes with VA and no underlying structural abnormalities, arrhythmias tended to decrease after detraining, and follow-up was uneventful, suggesting that PVCs may be a consequence of structural and neuroautonomic remodeling of the athlete's heart (Wolff et al., 1968). On the other hand, most studies comparing the prevalence of VA in 24-hour ambulatory ECG monitoring between healthy athletes and sedentary individuals have shown that only a minority of athletes present frequent or complex VA, with a prevalence that did not differ from that of their sedentary counterparts (Palatini et al., 1985; Pilcher et al., 1983).

Mechanisms

According to Marcus (2020), the mechanisms by which PVCs are generated include the following:

1. **Reentry:** This is a potential mechanism for PVCs, particularly in patients with structural heart disease, such as in the post-myocardial infarction (MI) setting. Reentrant PVCs occur with delayed conduction and unidirectional block, conditions typically observed in patients with healed myocardial infarction or evidence of myocardial fibrosis of any etiology.

2 **Abnormal automaticity:** This mechanism is more likely to occur with electrolyte abnormalities or acute ischemia, and is enhanced by catecholamines. These conditions tend to lower the diastolic transmembrane voltage, resulting in premature depolarization. The primary site of PVC development, due to abnormal automaticity, is the Purkinje fiber layer.

3 **Triggered activity:** Early (phase 3 of the action potential) or late (phase 4) afterdepolarizations may occur in Purkinje cells or the ventricular myocardium. This electrical activity can arise due to various conditions, including hypokalemia, ischemia, infarction, cardiomyopathy, calcium overload, and drug toxicity (such as digoxin or agents that prolong repolarization or the QT interval). If repetitive firing allows these afterdepolarizations to reach the threshold potential, PVCs will be generated and can become sustained if conditions are favorable (Marcus, 2020).

Classification

They can be classified in several ways, namely:

1. The absence (idiopathic) or presence of underlying structural heart disease.
2. Clinical presentation (symptomatic or asymptomatic).
3. ECG morphology: Complete right or left bundle branch block; unifocal with single morphology or multifocal with >1 morphology; interpolated when interposed within two sinus beats without a compensatory pause.
4. Relationship (or not) with exercise (i.e., exercise-induced or not).
5. Frequency of occurrence (PVC burden).
6. Prognosis (potentially **malignant**, for example, frequent PVCs in patients with structural heart disease or **idiopathic** short-coupled PVCs).

Characteristics of PVCs that confer a worse prognosis

The specific characteristics of PVCs, including their morphology (originating from the apex or free wall of the LV or RV), high burden, and complexity (e.g., couplets, triplets, or multifocal NSVT and/or an

increase in frequency during exercise), should raise concern about the possibility of an electrical, ischemic, or structural heart disease.

Burden

It is evaluated with a Holter ECG, which allows for determining the number and percentage of PVCs over a 24-hour period, along with other characteristics such as the tendency to form couplets, triplets, or NSVT. Patients can present frequent asymptomatic PVCs/VT. Only a minority of patients with >1000 PVCs per day will develop ventricular dysfunction after 5 years of follow-up (Lee et al., 2019). A PVC burden of 10% appears to be the minimum threshold for the development of LV dysfunction, with greater risk when the PVC burden exceeds 20% (Baman et al., 2010). Therefore, periodic evaluation of LVEF is indicated in this context. To date, there is no data supporting the benefit of arrhythmia treatment in asymptomatic patients with preserved ventricular function. Additionally, PVC burden often decreases spontaneously over time, particularly in children (Lee et al., 2019; Sharma et al., 2019).

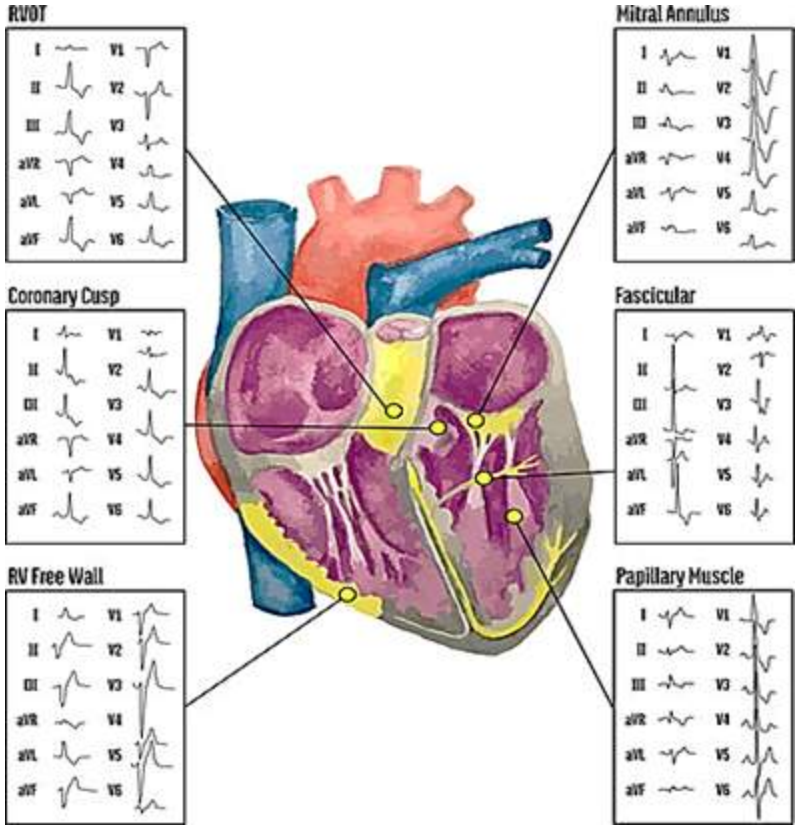
On the other hand, systematic research on SCD in young people and athletes from the Veneto region of Italy, along with retrospective evaluations of ECG tracings from SCD victims during their lifetime, demonstrated that the presence of a single PVC in a baseline pre participation ECG can be a warning sign of an underlying heart disease (such as arrhythmogenic cardiomyopathy or hypertrophic

cardiomyopathy) in an asymptomatic individual (Zipes et al., 2015). Current consensus standards for interpreting ECGs in athletes suggest that two or more PVCs in a resting ECG are required to initiate further investigation in an asymptomatic athlete, even a single PVC, particularly with a **high risk** QRS morphology (Drezner et al., 2017). Elite athletes with frequent PVCs (>2000/24 hours) and NSVT were reported to have a higher likelihood of underlying heart disease compared to athletes with fewer PVCs (Biffi et al., 2002). There is no absolute threshold for the number of PVCs that can be used as a cutoff point for diagnosing underlying disease. A study showed that in asymptomatic athletes with >2000 PVCs per day, there was a 30% chance of identifying underlying structural or cardiogenetic heart disease (Di Florio et al., 2021).

Morphology

The morphology of PVCs is likely to provide valuable diagnostic and prognostic information by allowing an approximate identification of their anatomical origin and the possible associated substrate of underlying disease (Figure 1). Most PVCs in athletes originate from the right or left ventricular outflow tract (RVOT/LVOT) or are fascicular in origin. These types of PVCs are generally benign in the context of a structurally normal heart (Figure 2A) (Di Florio et al., 2021).

Figure 1. Example 12-lead ECGs from common locations of premature ventricular complexes

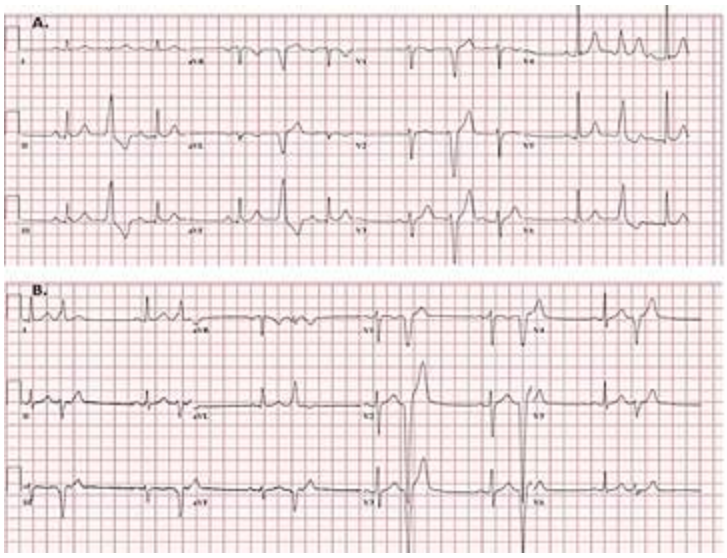


Source: Marcus, 2020, p. 1409

RV: right ventricle. RVOT: right ventricular outflow tract

Figure 2. (A) Typical PVCs from the RVOT, with LBBB pattern, precordial transition by V4, and an inferior axis. (B) Apical PVCs from the right ventricle in a patient with early ARVC. The PVC morphology shows LBBB with a very late transition and a superior axis. (PVC: premature ventricular complex; RVOT: right ventricular

outflow tract; LBBB: left bundle branch block; ARVC: arrhythmogenic right ventricular cardiomyopathy)



Source: Cantillon, 2013, p. 379

Also known as infundibular PVCs, PVCs originating from the outflow tract typically exhibit a characteristic left bundle branch block (LBBB) pattern with an inferior axis. Fascicular PVCs, which are also considered benign, display a typical right bundle branch block (RBBB) pattern. If they originate from the left posterior fascicle, they present a superior axis, while those from the left anterior fascicle show an inferior axis (Enriquez et al., 2019); this is illustrated in Table 1.

Conversely, PVCs with an LBBB pattern and an intermediate or superior axis should prompt investigation for ARVC or sarcoidosis (Figure 2B), while PVCs with a morphology similar to LBBB could

indicate an underlying cardiomyopathy, especially in cases with multiple morphologies (Di Florio et al., 2021; Corrado et al., 2020). Although these morphologies are uncommon in athletes, recognition of morphology is crucial for guiding diagnostic evaluation. For instance, PVCs aid in the differential diagnosis between idiopathic arrhythmias of the RVOT and early ARVC. An intrinsicoid deflection time >80 ms, QS pattern in V1, and QRS axis >90° have been identified as independent predictors of early ARVC (Figure 3) (Novak et al., 2017).

Table 1. PVC characteristics

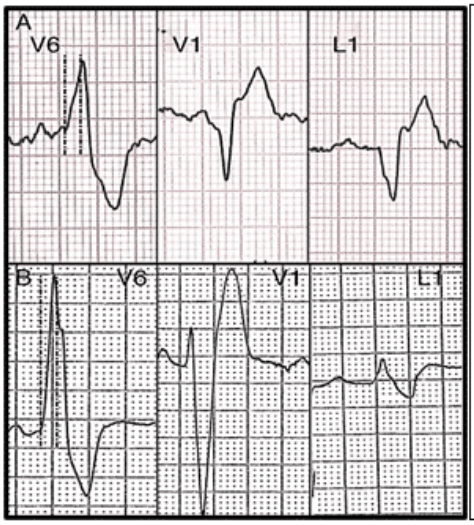
	BENIGNE	COMPLEX
MORPHOLOGY	• **RVOT:** LBBB pattern, inferior axis, transition →V4. • **LVOT:** LBBB pattern, inferior axis, transition <V4. • **Fascicular:** RBBB, QRS duration typically <130 ms, inferior axis (anterior fascicle), superior axis (posterior fascicle).	• **RV free wall or moderator band:** LBBB with intermediate or superior axis. • **LV cardiomyopathy:** RBBB-like morphology with wide QRS (>130 ms).
PATTERN	Monomorphic.	Polymorphic, repetitive.

RESPONSE TO EXERCISE	Disappear, may return in recovery.	Induced.
STRUCTURAL HEART DISEASE ASSOCIATION	Rare, further testing often not required.	Common, further testing required.

Source: Darden y Prutin, n. d., https://goo.su/V6gdz

RVOT: right ventricular outflow tract; LBBB: left bundle branch block; LVOT: left ventricular outflow tract; RBBB: right bundle branch block; RV: right ventricle.

Figure 3. PVC representative of ECG leads V6, V1, and 1 in a patient with early ARVC



(*A*) Patient with idiopathic RVOT arrhythmia. (*B*) In lead V6, the dotted lines identified an 80 ms interval after the beginning of the QRS. The intrinsicoid deflection time is >80 ms in the early ARVC patient, whereas it is ≤80 ms in the patient with idiopathic RVOT arrhythmia. In lead V1, there is a QS morphology in the early ARVC patient but not in the idiopathic RVOT arrhythmia patient. In lead 1, a Q wave is observed only in the early ARVC patient. The co-existence of all three factors has a sensitivity of 55% and a specificity of 91% for early ARVC.
ARVC: arrhythmogenic right ventricular cardiomyopathy. RVOT: right ventricular outflow tract.

Source: Novak et al., 2017, p. 626

Coupling interval

Short coupled PVCs or those PVCs that overlap with the preceding T wave (at its peak or earlier) should be considered a warning sign of myocardial electrical instability. This is due to early or non homogeneous ventricular repolarization, which may predispose the individual to ventricular fibrillation even in the absence of structural heart disease (i.e., idiopathic ventricular fibrillation) (Cipriani et al., 2019). Athletes with short-coupled PVC, particularly when associated with the inferolateral early repolarization pattern on the ECG, along with pronounced alteration of the terminal QRS, should be referred to a specialist for evaluation by electrophysiological study (Corrado et al., 2020).

Premature ventricular contractions and response to exercise

The reduction or resolution of PVCs with increased exercise load is typical of idiopathic and benign VAs, particularly those with an outflow tract morphology. Exercise-induced PVCs should be considered a **red flag**, because VAs associated with heart diseases often worsen with adrenergic stimulation. A higher prevalence of myocardial substrates (mainly non ischemic mid wall or subepicardial LV scars) was found in a CMR study among athletes with

exercise-induced PVCs compared to those with exercise-suppressed VA (56% vs. 21%) (Cipriani et al., 2019). It's important to note that isolated or repetitive exercise induced PVCs with multiple morphologies, especially with alternating beat-to-beat morphologies (the so-called **bidirectional** pattern), may be the expression of catecholaminergic polymorphic VT, which may degenerate into VF (Corrado et al., 2020).

Response to detraining

A previous study showed that, in most athletes with PVC and no underlying heart disease, the arrhythmia decreased or disappeared after a period of 3 to 6 months of detraining. While athletes with persistent PVCs were not considered eligible for competitive sports, those who had a reduction in PVC burden after detraining were able to resume competitive sporting activity, and had uneventful long-term follow-up. These findings suggest that the prognosis of exercise-induced PVCs that are not related to structural heart disease and resolve with detraining is favorable. Nevertheless, there other studies which showed contradictory results and questioned the prognostic value of detraining athletes with PVC (Corrado et al., 2020).

Biffi et al. (2002) demonstrated that healthy Olympic athletes with frequent PVCs most often show a reduction or disappearance of the arrhythmia with detraining. This finding has been interpreted as

supporting the idea that PVCs are part of the physiological spectrum of adaptive electrical and structural changes in the heart due to physical exercise (the so-called athlete's heart). On the other hand, these authors in later studies found the following: (1) retraining the athletes did not lead to a recurrence of PVCs, and (2) there was no correlation between arrhythmic burden and the degree of training-induced left ventricular hypertrophy. Delise et al. (2005) found no differences in VA behavior (persistence or reduction) during follow-up in a group of athletes who continued training versus a group of athletes who interrupted sporting activity.

Practical management

The most important task in individuals with PVC or NSVT who wish to participate in sports is to rule out underlying familial arrhythmogenic or structural conditions, as sporting activity can trigger sustained VT (Pelliccia et al., 2021; Corrado et al., 2020).

The study includes 12 lead ECG (with morphology suggestive of common and likely benign PVCs or rare and potentially malignant PVCs), 24-hour Holter monitoring possibly with a 12-lead system and including a sports session (PVC morphology, number and complexity), stress test (increase or decrease with exertion) and appropriate imaging studies (echocardiography and computed tomography and/or cardiac magnetic resonance imaging) and family history.

Finally, it may be necessary to repeat the evaluation after 6 months to 2 years (Heidbuchel et al., 2021).

According to Heidbuchel et al. (2021), decisions regarding the participation of athletes with PVCs should be individualized based on the assessment of underlying heart conditions and often involve shared decision-making.

Benign, asymptomatic PVCs do not require treatment if underlying heart disease is excluded. In symptomatic athletes, medical therapy with beta blockers (if permitted) or class 1 drugs may be considered, although ablation of the ectopic focus may provide a more definitive treatment option (Heidbuchel et al., 2021; Zipes et al., 2015).

How to evaluate athletes with PVCs

Table 2 classifies PVCs in athletes according to number, morphological pattern, complexity, response to exercise and clinical manifestations. This table provides criteria to distinguish **common** and benign PVCs from **uncommon** PVCs that are associated with an increased risk of cardiac pathology. This approach is fundamental to properly manage athletes with PVCs, to guide arrhythmic risk stratification and diagnosis, and to confirm (or rule out) underlying heart disease (Pelliccia et al., 2021; Heidbuchel et al., 2021). The presence of PVCs on an athlete's resting ECG or during an exercise test does not lead to a diagnosis of a cardiac disorder per se, but it should initiate a cascade

of additional cardiovascular evaluations to confirm (or rule out) cardiac pathology. International criteria for ECG interpretation in athletes suggested that further evaluation is warranted when PVCs PVC →2 are recorded on a 12 lead resting ECG. However, even a single PVB may merit attention, especially in the presence of one or more of these five characteristics: (1) positive family history of premature SCD or cardiomyopathy, (2) relevant symptoms, (3) associated ECG abnormalities, (4) uncommon PVC morphology (Table 2), and (5) short coupling interval (Pelliccia et al., 2021; Heidbuchel et al., 2021; Zipes et al., 2014).

Table 2. Classification and risk stratification of PVB in athletes

	COMMON	UNCOMMON
PVB characteristics		
Ectopic QRS morphology	LBBB/inferior axis, typical RBBB and narrow QRS (<130 ms)	LBBB/intermediate or superior axis, atypical RBBB and wide QRS (→130 ms)
Response to stress test	Decrease/suppression	Persistence/increase
Complexity of PVBs	Isolated, monomorphic	Repetitive‡, polymorphic

Short coupling interval*	No	Yes
CLINICAL FINDINGS		
Symptoms	No	Yes
Family history of premature SCD† or cardiomyopathy	No	Yes
Other ECG abnormalities	No	Yes
Imaging abnormalities	No	Yes

Source: Corrado et al., 2020, p. 5

*PVBs are superimposed on the preceding T wave peak or earlier (i.e., R on T).

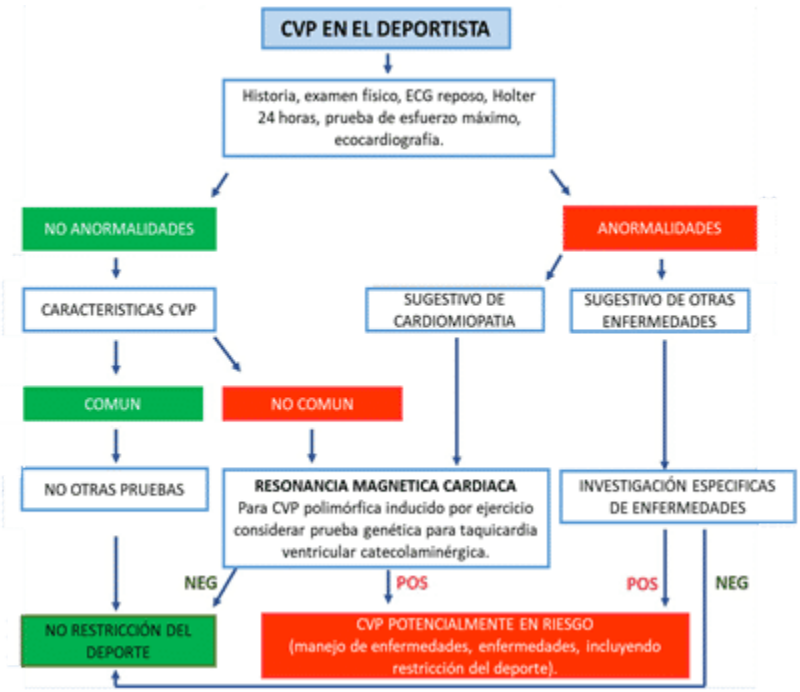
†Premature sudden cardiac death (SCD) is defined as that occurring before 40 years of age in men and before 50 years old in women.

‡ Couplets, triplets or non sustained ventricular tachycardia. LBBB: left bundle branch block; PVB:

premature ventricular beats; RBBB: right bundle branch block (Corrado et al., 2020, p. 5).

Figure 4 shows a practical flowchart for the clinical evaluation of athletes with PVCs. First-line tests include echocardiography, maximal stress test and 24 hour ambulatory ECG monitoring (ideally with a 12 lead configuration and inclusion of a training session). If these tests are abnormal, further testing depends on the suspected disease. The treatment of athletes with negative results from first-line tests is based on the characteristics of the PVBs. Athletes with a common PVC pattern (Table 2) do not require further testing and may be considered eligible for competitive sports, unless the clinical suspicion of disease remains high due to severe arrhythmic symptoms or a positive family history of SCD or cardiomyopathy.

Figure 4. Algorithm for the evaluation of athletes with PVC



Source: own source based on Heidbuchel et al., 2021, p. 148

CVP EN EL DEPORTISTA	PVC IN ATHLETES
Historia, examen físico, ECG reposo, Holter 24 horas, prueba de esfuerzo máximo, ecocardiografía.	History, physical examination, resting ECG, 24 hour Holter, maximal stress test, echocardiography.
NO ANORMALIDADES	NO ABNORMALITIES
CARACTERÍSTICAS CVP	PVC CHARACTERISTICS
COMÚN	COMMON
NO OTRAS PRUEBAS	NO FURTHER TESTING

NO RESTRICCIÓN DEL DEPORTE	NO SPORT RESTRICTION
ANORMALIDADES	ABNORMALITIES
SUGESTIVO DE CARDIOMIOPATÍA	SUGGESTIVE OF CARDIOMYOPATHY
NO COMÚN	UNCOMMON
RESONANCIA MAGNÉTICA CARDIACA	CARDIAC MAGNETIC RESONANCE IMAGING
Para CVP polimórfica inducido por ejercicio considerar prueba genética para taquicardia ventricular catecolaminérgica.	For exercise-induced polymorphic PVC, consider genetic testing for catecholaminergic ventricular tachycardia.
NEG	NEG
NO RESTRICCIÓN DEL DEPORTE	NO SPORT RESTRICTION
SUGESTIVO DE OTRAS ENFERMEDADES	SUGGESTIVE OF OTHER DISEASES
INVESTIGACIÓN ESPECÍFICA DE ENFERMEDADES	DISEASE-SPECIFIC INVESTIGATIONS
POS	POS
CVP POTENCIALMENTE EN RIESGO	PVC POTENTIALLY AT RISK

(manejo de enfermedades, enfermedades, incluyendo restricción del deporte).	(disease management, illnesses, including sport restriction).

"Ideally, 24-hour ECG monitoring should have a 12 lead configuration and include a training session. NEG: negative; POS: positive; PVB, premature ventricular beats" (Heidbuchel et al., 2021, p. 148).

> Athletes with an **uncommon** PVC pattern should undergo contrast-enhanced CMR (regardless of symptoms, family history, or results from first-line tests) to rule out an occult myocardial substrate that poses a risk for malignant arrhythmic events during sporting activities. Other tests such as coronary computed tomography or coronary angiography may be considered in selected middle aged and older athletes with exercise induced PVCs and a high coronary risk score. PVCs that occur during exercise test and become complex as the workload increases can be a sign of catecholaminergic polymorphic VT, whose definitive diagnosis (or exclusion) is based on molecular genetic testing for pathogenic mutations in the ryanodine receptor or calsequestrin genes (Priori et al., 2021).

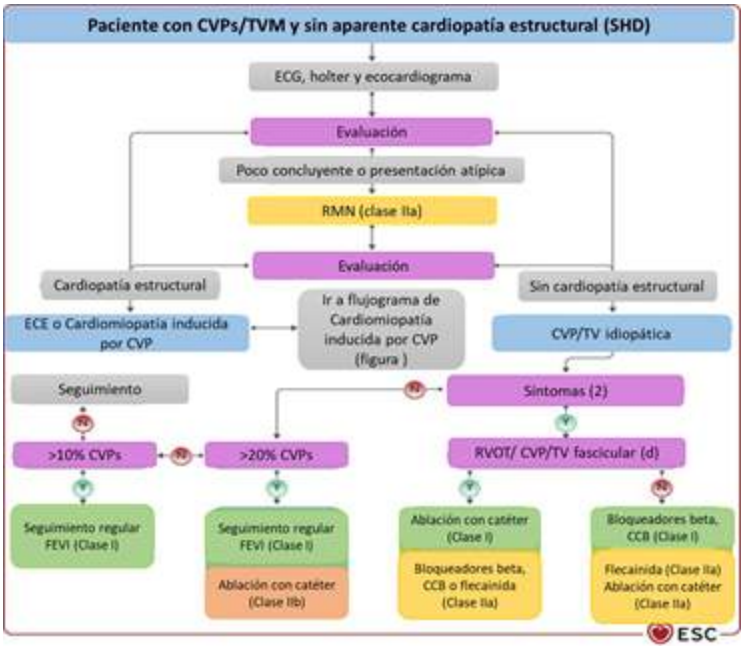
According to the proposed management flowchart for athletes with PVCs, additional diagnostic evaluation with sophisticated (and costly) imaging or molecular genetic testing is limited to the small subset of athletes with **uncommon** PVC characteristics, which may reflect a clinically hidden but potentially lethal heart disease that could be missed in routine tests. Conversely, the presence of more common PVCs, such as those with an **infundibular** or fascicular pattern, should provide reassurance for continued participation in competitive sports, as long as first line tests are normal, the athlete is asymptomatic, and family history of hereditary heart disease or premature SCD is negative.

If structural heart disease is identified, it should be managed according to the specific guidelines for each disease; if an idiopathic etiology is identified, the algorithm suggested by the **2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death** can be followed (Figure 4).

Generally, treatment is initiated when PVCs/TV are symptomatic or associated with cardiac function deterioration. The clinical course and responses to different treatments have been studied mainly in those

originating in the RVOT or left fascicles. Several drugs have been used to treat idiopathic PVCs/TV, such as beta blockers and calcium channel blockers; there is little evidence for the use of flecainide (Zeppenfeld et al., 2022).

Figure 5. Algorithm for the management of patients with idiopathic PVCs/TV without structural heart disease



Source: Zeppenfeld et al., 2022, p. 4051

CCB, calcium channel blocker; RMC, Spanish acronym for cardiac magnetic resonance (CMR); ECG, electrocardiogram; FEVI Spanish acronym for left

ventricular ejection fraction (LVEF); N, No; CVP, Spanish acronym for premature ventricular complex (PVC); TSVD, Spanish acronym for right ventricular outflow tract (RVOT); SHD, structural heart disease; TV, Spanish acronym for ventricular tachycardia (VT). Non-apparent SHD is defined by lack of significant abnormalities in physical examination, basal ECG, and echocardiogram. Atypical presentation: e.g. older age, right bundle branch block morphology, sustained monomorphic VT consistent with re-entry. Symptoms should be relevant and related to PVC/VT. Origin suspected by ECG or confirmed during electrophysiological evaluation (Zeppenfeld et al., 2022, p. 4051).

Medical therapy

β blockers or non dihydropyridine calcium channel blockers (diltiazem or verapamil) are considered first-line drugs for PVC. Both have a long history of safety in structurally normal hearts, and β blockers may have additional benefits in the setting of coronary artery disease or reduced LVEF.

β blockers are particularly effective for PVCs triggered by the sympathetic system, higher rates, or during exercise, with data demonstrating effectiveness specifically in outflow tract PVCs. Beta-

blockers should also be selected when a focal triggered activity mechanism is suspected (Zeppenfeld et al., 2022).

Non dihydropyridine calcium channel blockers have demonstrated similar efficacy in outflow tract PVCs and are considered particularly useful for fascicular ventricular arrhythmias. In patients with a structurally normal heart, it is reasonable to try a calcium channel blocker if a β blocker fails (and vice versa). Drug failure may be due to insufficient efficacy or intolerance to the medicine. The evidence for flecainide is limited. If these initial medicines fail, then catheter ablation should be considered (Cantillon, 2013).

Although data are lacking, beta-blockers or CCBs are considered first line treatment for ventricular extrasystoles (VE) originating outside the RVOT or the left fascicles, because flecainide may cause proarrhythmic side effects. Amiodarone is associated with severe systemic toxicity effects and should be used only if ablation or other drugs fail or cannot be used.

Since 1999, the International Olympic Committee founded the World Anti-Doping Agency (WADA), with the mission of governing medical procedures and performance-enhancing drugs to ensure fairness for professional athletes. If athletes require pharmacological treatment, caution must be exercised regarding their eligibility due to anti-doping policies. It is important to understand which medicines are on the list and when they are restricted. Beta-blockers reduce sympathetic

effects, such as increases in heart rate and blood pressure, which often rise during athletic competition. WADA explicitly bans beta blockers in sports that rely on limb stability, such as archery, racing, billiards, darts, golf, shooting, and fishing. All beta-blockers, including carvedilol, metoprolol, atenolol and propranolol, are included on the list. Beta-blockers are permitted in other sports but are banned in competition. The in-competition ban means they cannot be used from just before midnight (11:59 PM) the day before the competition until the end of the competition and the sample collection process (Heuberger & Cohen, 2019).

Beta-blockers can reduce heart rate by 30 to 35%; however, during maximal exercise, cardiac output is not equally reduced. Consequently, most studies have shown an increase in stroke volume after beta-blockade. Work capacity, reflected by the ability to perform short-term intense exercise or longer steady-state exercise, is also affected after beta-blockade. Beta-adrenoreceptors can be subdivided into beta 1 selective blockers and non selective blockers; they differ in their effect on exercise performance. Exercise performance capacity, regardless of the intensity and duration of the exercise, is more significantly affected after non selective beta blockade than with beta 1 selective blockade at equivalent reductions in heart rate.

This response results from a decreased energy flow through glycogenolysis during non selective beta blocker treatment. Therefore, individuals on beta blocker medication exhibit a greater adaptive

response to physical conditioning when using beta 1 selective blockers compared to non selective blockers. Neither psychomotor performance, nor muscle strength and power are negatively affected by beta blockade. However, the ability to perform sports events requiring high levels of motor control under emotional stress, but not high levels of aerobic or anaerobic energy release, is likely to improve during beta blockade (Tesch, 1985).

Athletes have a hypervagotonic profile, which involves a predisposition to asymptomatic sinus bradycardia. This, combined with the use of drugs like beta blockers, which also lower resting heart rate, can contribute to the development of sinus bradycardia. Therefore, caution should be exercised with dosing, and beta blockers are contraindicated in patients with symptomatic bradycardia. Beta-blockers also depress conduction through the atrioventricular (AV) node, which can lead to heart block. As a result, they can cause severe bradyarrhythmias in patients with underlying complete or partial AV conduction defects (i.e., second- or third-degree AV block), especially if the patient is also taking another drug that affects AV node conduction, such as digoxin or a calcium channel blocker (Lydtin, 1977).

Catheter ablation

In general, catheter ablation is more effective than medicines for treating PVCs, particularly when the target is predominantly

monomorphic. The success of PVC ablation procedures ranges from approximately 80% to 95%. Both the American Heart Association/American College of Cardiology/Heart Rhythm Society guidelines for the treatment of ventricular arrhythmias and the expert consensus statement on catheter ablation for ventricular arrhythmias from the Heart Rhythm Society/European Heart Rhythm Association/Asia Pacific Heart Rhythm Society/Latin American Heart Rhythm Society generally recommend either medicines or catheter ablation as first-line therapies for symptomatic PVCs or PVCs that are likely responsible for systolic dysfunction. Specifically, catheter ablation is classified as a Class I indication (a strong recommendation where the benefit greatly outweighs the risk) for treating PVCs if the patient does not tolerate medications, if the medicines are ineffective, or if the patient prefers ablation.

Complications of catheter ablation procedures for PVCs are observed in 0% to 2% of cases overall, primarily due to vascular access-related issues, including hematomas, pseudoaneurysms, or arteriovenous fistulas (which often resolve without intervention). Rarer but more serious complications include aortic dissection, atrioventricular block, myocardial infarction, cardiac tamponade, and stroke.

Expert guidelines on catheter ablation recommend ablation for PVCs from the outflow tract that originate on the left side of the heart, including the sinuses of Valsalva, as a Class II recommendation, with Class I indications reserved for PVCs in other locations. This is partly

because the left ventricular outflow tract is more complex and may require epicardial access more frequently (Cantillon, 2013).

> In general, the treatment of pediatric cases should be similar to that of adults. However, ablation should be deferred in young children due to the risk of complications and the relatively larger size of the ablation lesion compared to the child's heart. Verapamil is not recommended as first-line therapy in children under 1 year of age because it is associated with hypotension in some cases (Zeppenfeld et al., 2022, p. 57).

Implications for eligibility in competitive sports

The presence of underlying heart disease is a key prognostic factor and the most important determinant regarding eligibility recommendations for competitive sporting activity for athletes with PVCs. For this purpose, practical management guidelines for athletes should be followed, allowing for appropriate risk stratification for arrhythmias and determination of structural heart disease.

Subsequently, recommendations from the European guidelines, **SEC 2020 and ERHA 2021** as well as the American guidelines, **American**

Heart Association and American College of Cardiology - 2015, which agree on the following indications:

- If there is no evidence of underlying familial or structural disease and the athlete has PVCs at rest and during exercise (i.e., during stress test at a level comparable to the sport in which they compete) and they are not asymptomatic or are minimally symptomatic, they may participate in all competitive sports; all competitive and leisure time sporting activities are permitted.

- If PVCs increase in frequency during exercise or stress test to the point that the athlete develops symptoms of altered consciousness, significant fatigue or dyspnea, the athlete should be further evaluated. In the presence of structural heart disease, the athlete should be treated accordingly. If no cardiac abnormality is found, the athlete should receive medical treatment to alleviate symptoms and undergo close follow up. In both cases, the athlete may participate only in low-intensity sports.

- Athletes with a high prevalence of asymptomatic PVCs without structural heart disease should be reassessed annually to identify potential changes in arrhythmic burden and underlying cardiac condition.

- Catheter ablation should be offered to athletes with particularly frequent PVCs (>15%) that persist over time and are not reduced by medical treatment.

- Athletes with structural heart disease who are in high-risk groups and have PVCs should receive appropriate treatment and can only participate in low-intensity sports. (Pelliccia et al., 2021; Heidbuchel et al., 2021; Zipes et al., 2015).

In asymptomatic athletes with brief episodes (usually <8 to 10 consecutive ventricular beats) of non sustained monomorphic ventricular tachycardia (VT), rates usually <150 beats per minute and no structural heart disease established by non invasive and invasive testing, there is no apparent increased risk of sudden cardiac death. If the stress test (preferably by recording an ambulatory electrocardiogram [ECG] during the specific competitive activity) demonstrates suppression of VT or no significant worsening compared to baseline, participation in all competitive sports is permitted with regular follow-up (Zipes et al., 2015).

PVC-induced cardiomyopathy

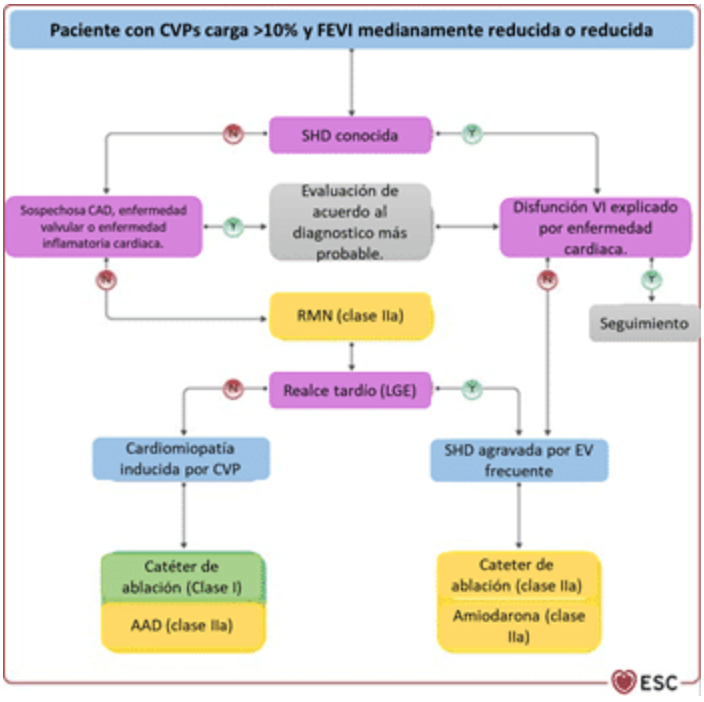
The importance of PVC-induced cardiomyopathy as a secondary and reversible cause of LV dysfunction in

patients without structural heart disease has been recognized. The patient's medical and family history, 12-lead ECG, Holter-ECG, and echocardiography form the cornerstones of the evaluation of patients with suspected PVC induced cardiomyopathy (Figure 6). PVC burden has been shown to be the strongest independent predictor of PVC induced cardiomyopathy in several studies. A PVC burden of at least 10% appears to be the minimal threshold for development of PVC induced cardiomyopathy, and the risk increases further with a PVC burden >20%. In patients with a PVC burden <10%, other cardiomyopathy etiologies should be suspected and further diagnostic work-up undertaken. Factors predicting adverse LV remodeling in patients with frequent PVCs include superior PVC axis, epicardial origin, NSVT, shorter coupling interval, and male gender.

Frequent PVCs can also aggravate LV dysfunction in patients with structural heart disease. CMR should be considered for patients suspected to have PVC induced cardiomyopathy to exclude subtle forms of SHD. In a patient with frequent PVCs, the presence of late gadolinium enhancement (LGE) suggests SHD with frequent PVCs rather than PVC induced cardiomyopathy, in which LGE is mostly absent. Given that PVCs with an RBBB morphology have been reported to show a stronger

association with LGE, those patients should be particularly considered for CMR. The diagnosis of PVC induced cardiomyopathy vs. PVC aggravated cardiomyopathy can be confirmed only after LVEF improvement/normalization (reverse remodeling) following suppression of the PVCs. Catheter ablation of the PVCs is very efficient, with reported success rates of 75 90%, and is considered first-line treatment for PVC induced cardiomyopathy (Zeppenfeld et al., 2022, p. 4052)

Figure 6. Algorithm for the management of patients with PVC induced cardiomyopathy



Source: Zeppenfeld et al., 2022, p. 4053.

AAD, antiarrhythmic drug; CAD, coronary artery disease; RMC, Spanish acronym for cardiac magnetic resonance (CMR); LGE, late gadolinium enhancement; VI, Spanish acronym for left ventricle (LV); FEVI, Spanish acronym for left ventricular ejection fraction (LVEF); N, No; CVP, Spanish acronym for premature ventricular complex (PVC); SHD, structural heart disease (Zeppenfeld et al., 2022, p. 4053).

Sustained ventricular tachycardia

According to Zeppenfeld et al. (2022), in athletes with sustained VT, the search for underlying heart disease is paramount. Athletes should be advised to stop participating immediately until further evaluation can be completed.

Documentation of sustained VT requires rigorous evaluation to distinguish idiopathic VT from life threatening monomorphic VT related to structural heart disease. Polymorphic VTs and VTs with alternating complexes (**bi directional VT**) during exercise are often associated with structural diseases or inherited electrophysiological disorders, and carry a high risk of malignant events. Idiopathic monomorphic sustained VTs are considered benign. However, symptoms (dizziness, presyncope) depend on VT cycle length (CL) and vascular tone (Zeppenfeld et al., 2022).

Evaluation of VT with 12-lead ECG allows for the site of origin identification. Most focal idiopathic VTs are due to triggered activity and arise from the endocardial outflow tract region (RVOT ⩾ LVOT) with a repetitive pattern at low exercise levels and suppression at higher levels. Occasionally, sustained exercise induced VT occurs. However, idiopathic focal VTs of the right and left ventricles without outflow tract have been recognized. Idiopathic epicardial VTs are due to a catecholamine sensitive mechanism and are often rapid under catecholaminergic stimulation and produce syncope. Verapamil sensitive idiopathic left fascicular reentrant VT (left posterior ⩾ left anterior ⩾ superior septal type) is recognized on the typical ECG and often presents as sustained VT. Many of these are also catecholamine-dependent (Zeppenfeld et al., 2022; Heidbuchel et al., 2021; Al-Khatib et al., 2018).

Evaluation of athletes with sustained ventricular tachycardia

According to Heidbuchel et al. (2021), the overall assessment of athletes with VA is equal to that of PVC/NVST. Late contrast-enhanced CMR should be performed for non fascicular VT morphologies, even if echocardiography is negative. It can be particularly challenging to distinguish idiopathic RVOT VT from early ARVC affecting the RVOT and exercise-induced arrhythmogenic remodeling (EI AR) with isolated subepicardial scarring of the RVOT can be particularly difficult. Suspicion of the latter is high if →2

distinct VT morphologies are observed with typically fast heart rates in high level endurance athletes.

ERHA 2021 and **American Heart Association and American College of Cardiology - 2015** agree on the following statements:

- Athletes with structural heart disease or channelopathies and sustained VT should not participate in intense recreational and competitive sports, regardless of acute therapeutic response to ablation/pharmacological treatment. Moderate and high intensity competition is contraindicated, regardless of whether VT is suppressed or ablated. Only low-intensity sports are allowed (Heidbuchel et al., 2021; Zipes et al., 2015).

- Sustained VT disqualifies for competitive sports, except in the particular case where all of the following apply: (i) absence of familial sudden death, (ii) absence of indication of any underlying structural pathology or channelopathy, (iii) a typical idiopathic VT focal or fascicular disease presentation, and (iv) absence of symptoms of hemodynamic compromise during VT with/without exercise (Heidbuchel et al., 2021; Zipes et al., 2015).

- Catheter ablation of symptomatic focal idiopathic RVOT VT and idiopathic left fascicular reentrant VT can be performed with high success rates (80 to 95%) and low complication rates, and is recommended in athletes to allow resumption of competitive sports (Liu et al., 2015).

- Athletes with idiopathic monomorphic VT, without hemodynamic compromise during exercise, can resume competitive or leisure time athletic activities where syncope does not increase the risk for the athlete or others (increased risk during, e.g., driving, climbing, diving) (Heidbuchel et al., 2021; Zipes et al., 2015).

- Athletes with idiopathic monomorphic VT who have undergone successful VT ablation and have no symptoms or other signs of recurrence (on Holter or stress test) during a 3 month follow up period may resume full competitive or leisure-time athletic activity (Heidbuchel et al., 2021; Zipes et al., 2015).

- Symptomatic athletes with →2 distinct VT morphologies or VT highly suspected to be reentrant as the underlying mechanism, with negative imaging studies including CMR, should undergo an invasive electrophysiological (EP) study to assess VT

inducibility and confirm the underlying mechanism (Heidbuchel et al., 2021; Zipes et al., 2015).

- Athletes with idiopathic monomorphic VT who choose to undergo pharmacologic treatment for suppression and are symptom free for a 3 month follow up period, which includes exercise testing or EP studies, may resume full or leisure-time athletic activity (Heidbuchel et al., 2021; Zipes et al., 2015).

- Ablation of idiopathic VT from non-fascicular, non RVOT, epicardial sources may involve greater procedural complexity, higher risks, and lower success rates, but should be considered depending on the athletes' preferences (Heidbuchel et al., 2021; Zipes et al., 2015).

Ventricular fibrillation

According to the approaches of Heidbuchel et al. (2021), as in the general population, athletes will have an indication for ICD implantation unless they have reversible, identifiable, and reversible causes encompassing the following:

1. Atrial fibrillation with rapid conduction over an accessory pathway that is successfully ablated.

2. Electrolyte imbalance due to a transient cause.
3. Proarrhythmic drug reactions (e.g., acquired QTc prolongation due to, for example, psychotropic medication).
4. Transient ischemia without myocardial infarction (MI), followed by complete revascularization.
5. Transient ischemia without MI due to coronary artery vasoconstriction in response to cocaine.
6. Acute myocarditis, followed by normalization of cardiac function, serum markers of inflammation/myocardial injury, and absence of frequent and complex ectopy (Heidbuchel et al., 2021).

If reversible causes are ruled out, the recommendations (**ERHA 2021** and **American Heart Association** and **American College of Cardiology 2015**) suggest the following:

- If there is no definitive assurance that a transient cause that led to resuscitated sudden death will not recur in athletes, then competitive sports are contraindicated.

- Athletes with conditions that lead to cardiac arrest, in the presence or absence of structural heart disease, are generally treated with an ICD and have traditionally been advised not to participate in any moderate or high intensity competitive sports.

However, in the current approach of shared decision-making, athletes with ICDs who have not experienced ventricular fibrillation episodes requiring device therapy for three months may participate in competitive sports, with an understanding of the potential risks involved (Heidbuchel et al., 2021; Zipes et al., 2015).

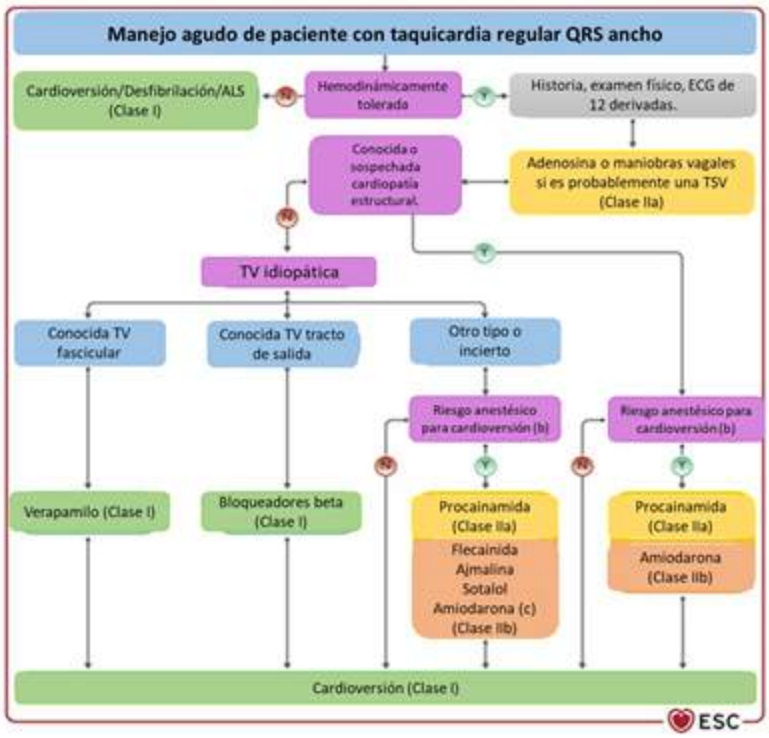
In patients with idiopathic ventricular fibrillation episodes, which obviously require admission to intensive care, the infusion of isoproterenol, verapamil, or quinidine is recommended (IIa) for the acute treatment of electrical storm or recurrent ICD shocks, and quinidine is recommended as chronic therapy to suppress electrical storm or recurrent ICD shocks in idiopathic VF (Zeppenfeld et al., 2022).

Acute management of SMVT and arrhythmic storm

Acute management of SMVT

Based on the contributions of Zeppenfeld et al. (2022), patients presenting with SMVT should be treated according to symptoms and etiology (Figure 7). Patients with hemodynamic instability require immediate synchronized cardioversion. If synchronization is not possible, a non synchronized shock should be used. Cardioversion is not indicated in patients with repetitive NSVT. Intravenous procainamide should not be used in patients with severe heart failure, acute myocardial infarction, or end-stage renal disease. The administration of other antiarrhythmic drugs (ajmaline, sotalol, and flecainide) may be considered in patients without significant heart disease.

Figure 7. Algorithm for the acute management of regular wide QRS complex tachycardia



Source: Zeppenfeld et al., 2022, p. 4029

ALS, advanced life support; ECG, electrocardiogram; N, No; TSV, Spanish acronym for supraventricular tachycardia (SVT); TV, Spanish acronym for ventricular tachycardia (VT). Besides SVT, adenosine may also terminate idiopathic VT, which then indicates triggered activity as the mechanism underlying the arrhythmia. The benefit of cardioversion should be weighed against risks related to anesthesia/sedation. Considering limited availability of the other antiarrhythmic drugs (Zeppenfeld et al., 2022, p. 4029).

Management of electrical storm and incessant ventricular tachycardia

An electrical storm is common in ICD patients and has been defined as three or more episodes of sustained ventricular arrhythmia occurring within 24 hours, requiring either anti-tachycardia pacing (ATP) or cardioversion/defibrillation, with each event separated by at least 5 minutes. Patients who experience an electrical storm are prone to psychological disorders, heart failure decompensation, and increased mortality.

Table 3. Management of electrical storm

Mild to moderate sedation is recommended in patients with electrical storm to alleviate psychological distress and reduce sympathetic tone.	I	C
Antiarrhythmic therapy with beta-blockers (non-selective preferred) in combination with intravenous amiodarone is recommended in patients with SHD and electrical storm unless contraindicated.	I	B
Intravenous magnesium with supplementation of potassium is recommended in patients with TdP.	I	C
Isoproterenol or transvenous pacing to increase heart rate is recommended in patients with acquired LQT syndrome and recurrent TdP despite correction of	I	C

precipitating conditions and magnesium.		
Catheter ablation is recommended in patients presenting with incessant VT or electrical storm due to SMVT refractory to AADs.	**I**	**B**
Deep sedation/intubation should be considered in patients with an intractable electrical storm refractory to drug treatment.	**IIa**	**C**
Catheter ablation should be considered in patients with recurrent episodes of PVT/VF triggered by a similar PVC, non-responsive to medical treatment or coronary revascularization.	**IIa**	**C**
Quinidine may be considered in patients with CAD and electrical storm due to recurrent PVT when other AAD therapy fails.	**IIb**	**C**
Autonomic modulation may be considered in patients with electrical storm refractory to drug treatment and in whom catheter ablation is ineffective or not possible.	**IIb**	**C**

Source: Zeppenfeld et al., 2022, p. 4034

AAD, antiarrhythmic drug; CAD, coronary artery disease; DC, direct current; LQT, long QT; PVC, premature ventricular complex; PVT, polymorphic VT; RVOT, right ventricular outflow tract; SHD, structural heart disease; SMVT, sustained monomorphic ventricular tachycardia; SVT, supraventricular tachycardia; TdP, Torsades de

Pointes; VF, ventricular fibrillation; VT, ventricular tachycardia (Zeppenfeld et al., 2022, p. 4034).

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