

Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 9: Arrhythmias and Conduction Defects A Scientific Statement From the American Heart Association and American College of Cardiology

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A broad range of variations in heart rates and rhythms, specific cardiac arrhythmias, and atrioventricular (AV) and intraventricular conduction disturbances are observed in athletes. Although most are common among nonathletes as well, the special circumstances and pressures related to athletic performance demand a high level of attention. The distinction between normal variants, often exaggerated by the specific physiology of the conditioned athlete, and arrhythmias that may be symptomatic or life-threatening may be significant challenges.

Bradycardia

Sinus Bradycardia

Sinus bradycardia, defined as a sinus rate <60 beats per minute (bpm), is common in the athlete.¹ Generally, it is attributed to enhanced vagal tone caused by conditioning and is thus physiological. Occasionally, heart rates can be as slow as 30 to 40 bpm at rest in the highly conditioned athlete and decrease to <30 bpm during sleep. Some athletes with marked

sinus bradycardia will exhibit periods of low atrial or junctional escape rhythms with rates of 40 to 60 bpm. This is a normal phenomenon, and these will become suppressed with exercise-induced increases in the sinus rate.

Evaluation of the athlete with sinus bradycardia includes a careful history to determine whether the athlete has symptoms related to the bradycardia. In addition, physical examination and an ECG are warranted, with selective use of additional tests such as an echocardiogram and exercise stress test if underlying structural heart disease is suggested. Stress testing can also be used to verify a normal rate response to exercise, if judged to be necessary. The same approach applies to the sinus arrhythmia commonly observed in the athlete. Generally, asymptomatic sinus pauses or sinus arrest (<3 seconds) are not considered clinically significant unless accompanied by symptoms. Pauses of longer duration may fall within the spectrum of physiological responses to athletic conditioning; however, when accompanied by symptoms, sinus bradycardia,

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sinoatrial exit block, and sick sinus syndrome with pauses at the termination of a supraventricular tachycardia (SVT) are considered abnormal. Athletes with symptoms potentially associated with these arrhythmias should have an ECG, 24-hour ambulatory monitoring, and an exercise test. Clinical assessment for structural heart disease and noninvasive assessment of sinus node function with ambulatory monitoring and stress testing are also appropriate in symptomatic patients or those with resting heart rates <30 bpm or pauses >3 seconds.

Invasive electrophysiology studies (EPS) play a very limited role in the assessment of sinus node function. An athlete with symptoms related to sinus bradycardia caused by high vagal tone related to training should restrict athletic training and have clinical reassessment of symptoms and sinus node function.¹ Patients with symptomatic bradycardia not responsive to other measures such as deconditioning or the withholding of nonessential medications that are contributing to the bradycardia may need to be treated with a permanent pacemaker, although this is very rarely needed in the athlete.^{2,3}

Recommendations

1. Athletes with sinus bradycardia, sinus exit block, sinus pauses, and sinus arrhythmia without symptoms can participate in all competitive athletic activities unless otherwise excluded by underlying structural heart disease or other arrhythmias (*Class I; Level of Evidence C*).
2. Athletes with symptomatic bradycardia should be evaluated for structural heart disease and be treated for the bradycardia, generally by an implanted pacemaker. They should be restricted from training and athletic competition while being evaluated. If treatment of the bradycardia eliminates symptoms, they can participate in athletic training and competition unless otherwise excluded by structural heart disease or other arrhythmias (*Class I; Level of Evidence C*).

AV Block

Athletes with AV block should be assessed for symptoms attributable to the block with a history and for any underlying structural heart disease with a cardiovascular examination and ECG. Other tests, including an echocardiogram, ambulatory monitoring, exercise stress test, and invasive EPS, should be used in a selective fashion.

First-Degree AV Block

In asymptomatic athletes with structurally normal hearts who have first-degree AV block identified on a preparticipation or other incidental ECG, the PR interval will shorten during a stress test in most cases. However, stress testing is rarely necessary for the evaluation of an athlete with a PR interval <0.3 second and a normal QRS duration. An echocardiogram is not necessary unless the cardiovascular examination or ECG suggests structural heart disease. If the QRS complex is abnormal, or the PR interval is excessively prolonged (≥ 0.3 second), an exercise stress test, 24-hour ambulatory monitor, and echocardiogram are warranted. EPS is rarely necessary but might be performed in selected cases, such as those with

exercise-induced AV block suspected of having type II AV block, to determine the site and duration of conduction delay (AV node or intra-His/infra-His) and ensure that the patient is not at risk for progression to higher-degree block that would cause symptoms. Patients with congenitally corrected transposition of the great arteries can exhibit first-degree AV block with very little else on physical examination.

Recommendations

1. Asymptomatic athletes with no structural heart disease and first-degree AV block (PR interval <0.3 ms) can participate in all competitive sports unless there are findings that indicate that the person is at risk for progression to higher-degree block that would cause symptoms (*Class I; Level of Evidence C*).
2. Asymptomatic athletes with first-degree AV block, in whom type I second-degree AV block appears with exercise, should be evaluated further for possible intra-His or infra-His block with EPS (*Class I; Level of Evidence C*).
3. If structural heart disease is present, athletic restrictions should be recommended as appropriate for the type of structural heart disease (*Class I; Level of Evidence C*).

Type I Second-Degree (Wenckebach) AV Block

Wenckebach type I AV nodal block can be present in otherwise normal, well-trained endurance athletes. Type I second-degree AV block (ie, Wenckebach) is observed more commonly during sleep in athletes than in the daytime when they are awake. Athletes should be assessed for symptoms attributable to the block and for any underlying structural heart disease with an echocardiogram. In asymptomatic or symptomatic athletes with Wenckebach block, a history, physical examination, ECG, echocardiogram, and exercise stress test may be considered. If the QRS complex is abnormal, or the shortest PR interval is excessively prolonged (≥ 0.3 second), 24-hour ECG recording or other ambulatory monitor is warranted. EPS is rarely necessary but might be performed in highly selected cases to determine the site and duration of conduction delay and ensure that the patient is not at risk for progression to higher-degree block that would cause symptoms.

Recommendations

1. Asymptomatic athletes with structurally normal hearts and Wenckebach AV block (type I second-degree AV block) with improvement in conduction with exercise or recovery can participate in all competitive sports (*Class I; Level of Evidence C*).
2. Asymptomatic athletes with structurally abnormal hearts with improvement in Wenckebach AV block with exercise can participate in all competitive sports, unless there are restrictions based on heart disease (*Class I; Level of Evidence C*).
3. Athletes with Wenckebach AV block that does not improve with exercise should be evaluated with an EPS for intra-His or infra-His block that may require pacemaker therapy (*Class I; Level of Evidence C*).

- 4. In athletes with Wenckebach AV block and coexisting bundle-branch block or with any indication that they are at risk for progression to higher-degree AV block, EPS should be performed to identify the presence of intra-His-Purkinje or infra-His-Purkinje block that may require pacemaker therapy (Class I; Level of Evidence C).**

Type II Second-Degree (Mobitz) AV Block

Type II second-degree (Mobitz) AV block is abnormal in athletes. Athletes with type II second-degree AV block should be assessed with a history, physical examination and echocardiogram regardless of symptoms. In addition, it is important to distinguish 2:1 Wenckebach physiology at the level of the AV node from true Mobitz type II AV block. This can usually be achieved by a stress test, but EPS may be required in rare cases. Generally, Mobitz type II second-degree AV block is considered an indication for a permanent pacemaker.^{2,3} The recommendations for evaluation and treatment of Mobitz type II second-degree AV block are the same as those for acquired complete heart block below.

Recommendations

- 1. Athletes with Mobitz type II second-degree AV block with a wide QRS, including isolated right bundle-branch block (RBBB) should receive a permanent pacemaker (Class I; Level of Evidence C). Restrictions for athletic participation for those with pacemakers are in the section on "Athletes With Permanent Pacemakers."**
- 2. Permanent pacemaker implantation is reasonable for athletes with asymptomatic Mobitz type II second-degree AV block with a narrow QRS (Class IIa; Level of Evidence C).**

Complete RBBB

Athletes with a complete RBBB should have a cardiac evaluation with a history, physical examination, ECG, echocardiogram, and stress test. Ambulatory monitoring and EPS can be used in a very selective fashion in patients with documentation of symptoms possibly attributable to progression to type II second-degree AV block or complete heart block.⁴ Progression is more likely if left anterior fascicular block accompanies the RBBB.

Recommendation

- 1. Athletes with RBBB, who do not develop periods of type II second-degree AV block or complete heart block spontaneously or during exercise and who have no symptoms or heart disease identified by appropriate testing that otherwise precludes participation, can participate in all competitive athletics (Class I; Level of Evidence C).**

Complete Left Bundle-Branch Block

Athletes with a complete left bundle-branch block (LBBB) should have a cardiac evaluation with a history,

physical examination, ECG, echocardiogram, and stress test. Ambulatory monitoring and EPS can be useful in patients with documentation of, or symptoms possibly attributable to, progression to type II second-degree AV block or complete heart block. Acquired LBBB may be associated with syncope from paroxysmal AV block. In patients with syncope or presyncope, an invasive EPS should be strongly considered to exclude intra-Hisian or infra-Hisian block. In contrast, rate-dependent LBBB in the absence of symptoms or structural heart disease may be benign, but long-term data are lacking. However, because rate-dependent LBBB, particularly if at slow rates, often occurs in the presence of structural heart disease, a more complete evaluation is necessary to exclude the latter.⁵

Recommendations

- 1. Athletes with permanent or rate-dependent LBBB who do not develop spontaneous type II second-degree AV block (Mobitz) or complete heart block and who have no symptoms or heart disease identified by appropriate testing that otherwise precludes participation, can participate in all competitive athletics (Class I; Level of Evidence C).**
- 2. In athletes with concerning symptoms, an EPS is recommended. An athlete with a normal HV interval and a normal AV conduction response to pacing can participate in all competitive sports unless otherwise restricted by their structural heart disease (Class I; Level of Evidence C).**
- 3. Athletes with abnormal AV conduction characterized by an HV interval >90 ms or a His-Purkinje block should have pacemaker implantation (Class I; Level of Evidence C).**

Congenital High-Grade or Complete Heart Block

Athletes with congenital complete heart block, and the rare cases of congenital advanced type II second-degree heart block, should be evaluated with a history, physical examination, ECG, echocardiogram, 24-hour ambulatory monitor, and exercise stress test. The exercise stress test protocol should be to maximum level of performance to assess ability to exercise to a level comparable to the relevant athletic activity. In recent years, there has been a trend to implant pacemakers in all patients with congenital complete heart block because of concern of evolution of left ventricular dysfunction and heart failure over time.^{6,7}

Recommendations

- 1. Asymptomatic athletes without heart disease who have a junctional escape rhythm that has a QRS duration <120 ms, resting ventricular rates >40 bpm that increase appropriately with exertion, and exercise capacity that approximates that of the relevant sport can participate in athletic activity without restriction (Class I; Level of Evidence C).**
- 2. Athletes with symptomatic heart block, resting ventricular rates <40 bpm, or ventricular escape rhythm with a QRS width >120 ms should have a pacemaker**

implanted before they participate in competitive sports. Before athletes are allowed to resume sports, an exercise test should be conducted to ensure patient safety and that the exercise capacity of the athlete is similar to that required for the relevant sport (*Class I; Level of Evidence C*).

3. Athletes with structural heart disease and congenital complete heart block should be restricted from, or allowed to participate in, competitive athletics based on the recommendations for the type of structural heart disease with or without a permanent pacemaker (*Class I; Level of Evidence C*).

Acquired Complete Heart Block

Athletes with acquired complete heart block should be evaluated with a history, physical examination, ECG, echocardiogram, and additional diagnostic testing as is clinically appropriate. Acquired complete heart block, unless caused by completely reversible factors, is an indication for placement of a permanent pacemaker.^{2,3}

Recommendations

1. Athletes with acquired complete heart block should have a permanent pacemaker placed regardless of symptoms, type of structural heart disease, and exercise capacity unless the heart block is attributable to completely reversible causes and resolves completely (*Class I; Level of Evidence C*).
2. Athletes with structural heart disease and acquired complete heart block should be restricted from, or allowed to participate in, athletic activities based on the recommendations for the type of structural heart disease (*Class I; Level of Evidence C*).
3. Before athletes with a permanent pacemaker are allowed to engage in athletic activities, an exercise test should be conducted to ensure that the exercise capacity of the athlete is similar to that required by the relevant sport (*Class I; Level of Evidence C*).

Athletes With Permanent Pacemakers

Many of the patterns of bradycardia and AV conduction variants observed in athletes do not require consideration of pacemaker therapy, but a few of the conditions described have clear indications. The presence of a pacemaker is not an automatic impediment to clearance for athletic participation. The presence or absence of underlying structural heart disease, level of pacemaker dependence, risk of damage to device, and symptoms are relevant modifiers.

Recommendations

1. Generally, athletes with permanent pacemakers should be cleared for athletic participation if there are no limiting structural heart conditions or symptoms (*Class I; Level of Evidence C*).
2. Athletes who are completely pacemaker dependent should not engage in sports in which there is a risk of collision that could result in damage to the pacemaker system (*Class I; Level of Evidence C*).

3. Athletes treated with a pacemaker who are not pacemaker dependent may participate in sports with a risk of collision or trauma if they understand and accept the risk of damage to the pacemaker system and they have no structural heart disease that precludes participation (*Class I; Level of Evidence C*).
4. For athletes with permanent pacemakers, protective equipment should be considered for participation in contact sports that have the potential to damage the implanted device (*Class I; Level of Evidence C*).

Supraventricular Tachycardia

SVTs are not more common in athletes than in the general population of a similar age distribution, with the possible exception of atrial fibrillation (AF).^{8,9} Treatment of these SVTs with catheter ablation is likely to achieve a permanent cure and in general is preferable to lifelong therapy with pharmacological agents. SVT-associated symptoms include palpitations, weakness, lightheadedness, and occasionally syncope, all of which may impair athletic performance, although the vast majority of SVTs are not life threatening. Symptoms do not distinguish between the different SVTs, and thus, a symptom-rhythm correlation is required. Rarely, a person with a sustained form of SVT, such as atrial flutter (AFL) or AF, or more commonly in young people, atrial or junctional tachycardias or the permanent form of junctional tachycardia, can present with a tachycardia-induced cardiomyopathy. The differential diagnosis of SVTs in the athlete includes sinus tachycardia, although this tachycardia should be relatively easily diagnosed by use of resting ECGs.¹⁰

Atrial Fibrillation

There are some data suggesting that athletes are at increased risk of AF, and in particular vagally mediated AF.^{8,9,11} Athletes may be particularly prone to AF because of the high vagal tone associated with extreme fitness, as well as cardiac remodeling, which includes changes in chamber size and pressure. Other causes, including fibrosis, inflammation, and sympathetic discharge, can also play a role. All athletes with AF should undergo a workup that includes thyroid function tests, ECGs, echocardiograms, and queries for drug use, including performance-enhancing agents and illicit drugs. Athletes with AF should be evaluated for hypertension and coronary artery disease. Further testing is warranted in some cases, including cardiac magnetic resonance imaging and stress testing. Patients with underlying cardiac disease such as dilated cardiomyopathies, hypertrophic cardiomyopathy, Brugada syndrome, and catecholaminergic ventricular tachycardia (VT) have an increased risk of AF. AF in a child or adolescent athlete is uncommon and should suggest a familial inheritance or the presence of an accessory pathway.

The management options for AF in athletes include rate control or rhythm control. Rate control, although an option, may not be ideal for competitive athletes because of the focus on performance and difficulty ensuring adequate rate control during an athletic performance. A rhythm control strategy is thus the preferred method of treatment in athletes. Rhythm control can be achieved with antiarrhythmic agents

or ablation procedures. Increasingly, ablation has shown a sustained benefit, particularly in those with paroxysmal AF in the presence of a normal heart, which would likely be most athletes with AF¹²; however, longer-term observations are necessary to determine benefits over many years. Antiarrhythmic drug therapy has efficacy and side effect concerns, including proarrhythmic risk. In some cases, withdrawal from competitive sports or attempts at deconditioning might be chosen. Conversely, some athletes may choose to avoid any therapy and still participate because they tolerate short episodes of AF during competition.

The other component of management is anticoagulation. Most athletes will have a low risk of systemic thromboemboli as manifested by a low CHADS₂ score or a CHA₂DS₂-VASC score of zero, and anticoagulation will rarely be necessary. If anticoagulation is used, athletes should be restricted from participation in high-impact contact sports because of the bleeding risk.

Recommendations

1. Athletes with AF should undergo a workup that includes thyroid function tests, queries for drug use, ECG, and echocardiogram (Class I; Level of Evidence B).
2. Athletes with low-risk AF that is well tolerated and self-terminating may participate in all competitive sports without therapy (Class I; Level of Evidence C).
3. In athletes with AF, when antithrombotic therapy, other than aspirin, is indicated, it is reasonable to consider the bleeding risk in the context of the specific sport before clearance (Class IIa; Level of Evidence C).
4. Catheter ablation for AF could obviate the need for rate control or antiarrhythmic drugs and should be considered (Class IIa; Level of Evidence B).

Atrial Flutter

AFL may also be more common in the athlete. The workup for AFL is identical to that of AF: thyroid function tests, queries for drug use, ECGs, and an echocardiogram. Anticoagulation and rate control are also similar to that of AF. However, given the high cure rates of ablation and the low complication risk, AFL ablation should be the rhythm control strategy of choice for those with typical cavotricuspid isthmus-dependent flutter.

Recommendations

1. Athletes with AFL should undergo an evaluation that includes thyroid function tests, queries for drug use, ECG, and echocardiogram (Class I; Level of Evidence B).
2. Catheter ablation for typical AFL has a high likelihood of success and should be considered (Class I; Level of Evidence B).
3. When anticoagulation, other than with aspirin, is indicated in an athlete, it is reasonable to consider the bleeding risk in the context of the specific sport before clearance (Class IIa; Level of Evidence C).

AV Nodal Reentry Tachycardia, AV Reciprocating Tachycardia, Atrial Tachycardia

These 3 tachycardias, AV nodal reentry tachycardia (AVNRT), AV reciprocating tachycardia (AVRT), and atrial tachycardia (AT), are considered together because of the many similarities they share,¹⁰ such as acute onset and termination, rates between 150 and 250 bpm, a regular ventricular rhythm, largely narrow QRS complex, and termination with adenosine. The latter is more likely to be effective in AVNRT and AVRT than AT. In addition, AT can exhibit a progressive rate increase at the onset and a gradual slowing before termination. The surface ECG may not reliably distinguish between these 3, and both acute and long-term treatments for these 3 SVTs are similar. AVNRT occurs because of dual AV nodal physiology; AVRT because of a bypass tract that allows conduction between the atria and ventricle other than via the AV node; and AT because of microreentrant circuits, automatic foci, and possibly triggered activity. The ECG in AT might be confused with the permanent form of junctional tachycardia or atypical AVNRT because of a long RP interval, but it is unlikely to be confused with AVRT and typical AVNRT, which have a short RP interval. If preexcitation is present on a surface ECG, then AVRT is likely; however, a definite diagnosis often requires an invasive EPS. Occasionally, these SVTs can present as a wide-complex tachycardia if a bypass tract is present or if there is aberrant ventricular conduction of RBBB or LBBB. Treatment options include β -adrenergic blocking agents, nondihydropyridine calcium channel antagonists, multiple antiarrhythmic agents, and catheter ablation. Given the high success rates of catheter ablation and the low complication rate, catheter ablation is the treatment of choice in this young healthy population.

There is no clear consensus regarding the asymptomatic athlete with an ECG that demonstrates preexcitation. There is concern regarding the increased but unquantifiable risk of sudden cardiac death (SCD), most notably among athletes with accessory pathways having short refractory periods that allow very rapid ventricular rates during AF. A few studies and opinions have advocated risk stratification for asymptomatic people with an ECG that shows preexcitation.¹³ A recent consensus statement, endorsed by the Heart Rhythm Society and the Pediatric and Congenital Electrophysiology Society, recommends that people aged <21 years undergo initial stress testing to determine whether there is sudden and complete loss of preexcitation during exercise, which would denote low risk because of an accessory pathway with a long refractory period.¹⁴ If a person cannot be ascertained as being at low risk by stress testing, then an invasive EPS is advocated, with ablation if the bypass tract has a high risk for SCD because of an effective refractory period \leq 250 ms.

Recommendations

1. Athletes with regular, acute-onset SVTs should undergo cardiac assessment with ECG and echocardiogram (Class I; Level of Evidence B).
2. The treatment of choice for athletes with regular, acute-onset, symptomatic SVTs should be catheter ablation (Class I; Level of Evidence C).

3. **Athletes with short refractory period bypass tracts capable of anterograde conduction and a history of paroxysmal AF should have an ablation of the accessory pathway before clearance for competitive sports because of risk for life-threatening arrhythmias (Class I; Level of Evidence B).**
4. **In athletes with asymptomatic preexcitation, it is reasonable to attempt risk stratification with stress testing to determine whether the preexcitation abruptly terminates at low heart rates. If low risk is unclear, it is reasonable to recommend invasive electrophysiological evaluation, with ablation of the bypass tract if it is deemed high risk for SCD because of a refractory period ≤ 250 ms (Class IIa; Level of Evidence B).**

Ventricular Arrhythmias

A variety of ventricular arrhythmias can occur in competitive athletes across the age spectrum relevant to this document. Generally, the appearance of any ventricular arrhythmia requires evaluation before clearance for participation in athletic activities, but the level of workup depends on the specific pattern of the arrhythmias, whether they are symptom-provoking or not, and whether they occur in the presence of structural, molecular, or inflammatory heart diseases.

Premature Ventricular Complexes

Premature ventricular complexes (PVCs) are most commonly benign, but their appearance requires at least a minimal level of evaluation before clearance. The major distinctions to be made are whether they are isolated or occur in the presence of a transient or chronic cardiac abnormality, as well as how they respond to exercise.¹⁵ The minimal level of testing to acquire prognostic information is a 12-lead ECG and exercise stress test.¹⁶ In most instances, an echocardiogram will also be performed to rule out a structural abnormality that cannot be identified by either the ECG or stress test. Other imaging studies can be considered, based on the circumstances of the specific arrhythmias noted. These include computed tomography and magnetic resonance imaging for disorders such as cardiomyopathies, anomalous coronary artery origins, and subclinical myocarditis. In addition, a 24-hour ambulatory monitor may be helpful in determining the frequency and pattern of the arrhythmias. PVCs recorded at a frequency of >2000 per 24 hours have a higher likelihood of association with underlying cardiac disease,¹⁵ estimated at 30% in this subgroup. It is reasonable to conclude that palpitations caused by PVCs in the absence of heart disease that occur at rest, are suppressed with exercise, and are not accompanied by periods of nonsustained VT (NSVT; at most, PVC couplets) are benign and should not limit full participation in competitive physical activities.¹⁷ For the purpose of this recommendation, multifocal/multiform single PVCs may be equivalent to uniform/unifocal PVCs in terms of risk assessment, as in the case of other clinical settings.¹⁸ PVCs that become more frequent or convert to runs of NSVT during exercise should lead to further evaluation, depending on findings on the initial noninvasive testing.¹⁹

PVCs observed in the conditioned athlete without heart disease may decrease on deconditioning and reappear with reconditioning. This pattern does not indicate independently heightened risk in the absence of other risk markers, and with continued training, the frequency of ectopy decreases.²⁰ There may be as yet unrecognized implications for higher risk of SCD associated with intense exercise in subjects in the general population who do not exercise regularly.²¹ This observation should be considered in deconditioned athletes who immediately begin a very intense conditioning program.

Disorders that should be considered are structural abnormalities such as occult coronary artery disease and coronary artery anomalies, including myocardial bridging, early evolution of hypertrophic cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy. Athletes with persisting frequent PVCs should remain under surveillance over time for early evidence of development of PVC-induced cardiomyopathy. Annual cardiological evaluation is required in athletes with PVCs >2000 per 24 hours.¹⁵ Contrast-enhanced cardiac magnetic resonance may detect subtle changes seen in hypertrophic cardiomyopathy and myocarditis.²² One study suggests that electroanatomic mapping in athletes with ventricular arrhythmias may identify evidence of subtle cardiomyopathies.²³ The small number of subjects studied predominantly had sustained or NSVT or very frequent PVCs. Molecular disorders possibly associated with increased PVCs that should be considered are the various channelopathies, including long-QT syndrome and catecholaminergic polymorphic VT, and transient disorders such as a viral myocarditis should be considered. If there is evidence for the latter, the athlete should be retested after resolution of myocarditis.

Recommendations

1. **Athletes with single PVCs and complex forms no greater than couplets at rest and during exercise testing without structural heart disease can participate in all competitive sports. The exercise testing protocol should be based on maximal performance rather than achieving 80% to 100% of the target heart rate to come as close as possible to the level of exertion achieved during their competitive sport (Class I; Level of Evidence C).**
2. **Athletes with PVCs at rest that increase in frequency during exercise or exercise testing and convert to repetitive forms should have further evaluation by appropriate imaging or monitoring strategies before clearance for participation in high-intensity sports. If uncontrollable exercise-induced arrhythmias produce symptoms of lightheadedness or near-syncope, fatigue, or dyspnea, the athlete should be limited to competitive sports below the level at which marked frequency increase or symptoms evolved during testing (Class I; Level of Evidence C).**
3. **Athletes with defined structural heart disease who are considered high risk based on the specific heart disease and who have PVCs with or without treatment should be limited to low-intensity class IA competitive sports. This statement applies whether or not**

PVCs in this setting are suppressed by drug therapy (Class I; Level of Evidence C). Some degree of risk can still be present during class IA sports, however, depending on the nature of the heart disease.

4. Ablation of PVCs may be considered in symptomatic patients with frequent PVCs resistant to medical therapy (Class IIb; Level of Evidence C).

Nonsustained VT

NSVT, defined as ≥ 3 consecutive PVCs up to a maximum duration of 30 seconds of repetitive activity that does not provoke cardiovascular collapse, has a higher probability of reflecting an underlying disorder than single PVCs.¹⁹ Nonetheless, short runs of NSVT may be normal, but the potential for significant abnormalities must determine the workup and decision making. NSVT may occur as monomorphic or polymorphic forms. In general, patterns that are monomorphic and tend to be slower (eg, <150 bpm) are more likely to be benign than those that are polymorphic and faster. In all cases, the minimum workup should include a 12-lead ECG and stress test, including echocardiography, either as part of the stress test or separately. A 24-hour ambulatory monitor should also be conducted, with the patient instructed to perform his or her usual levels of exercise with the monitor in place. The same limitations in regard to symptomatic worsening of the arrhythmias that are described for PVCs apply to NSVT as well. Athletes with NSVT at rest that is suppressed with exercise and who have no evidence of structural heart disease, molecular/genetic disorders, or transient abnormalities at the time of evaluation can be cleared for competitive athletics without limitations. If structural heart disease is identified, the athlete should be limited to class IA competitive sports.

Recommendations

1. Athletes with a structurally normal heart and no evidence of molecular/genetic or inflammatory disorders with suppression of the arrhythmia during exercise can participate in competitive athletics at any level. The exercise testing protocol should be based on maximum performance rather than achieving 80 to 100% of the target heart rate to come as close as possible to the level of exertion achieved during the athlete's competitive sport. Consideration of advanced therapy such as catheter ablation in an attempt to cure the runs of NSVT is optional (Class I; Level of Evidence C).
2. For athletes without structural heart disease who have NSVT that is suppressed by drug therapy, especially β -blockers, documentation of both ambient and exercise-induced NSVT should be required before general clearance for participation in higher-level competitive athletics. Specifically, the athlete should not compete in sports with a classification greater than IA unless it is documented by exercise testing or electrophysiological testing that the arrhythmia is no longer inducible under the circumstances in which it was induced before therapy (Class I; Level of Evidence C). β -Blockers might exacerbate exercise-induced asthma.

3. Athletes with structural disorders or active myocarditis and documented NSVT should only participate in low-intensity class IA sports. In the case of myocarditis, reevaluation is recommended after there is clinical and laboratory evidence of healing of the myocarditis, with return to athletics a minimum of 3 months after clinical resolution (Class I; Level of Evidence C).

Sustained Monomorphic VT

Sustained monomorphic VT may be a benign arrhythmia, but it has a higher probability of reflecting an underlying structural disorder. Generally, the benign forms of sustained monomorphic VT appear at low levels of exercise and are suppressed during higher levels, although catecholamine-dependent forms of right ventricular outflow tract tachycardia may occur with increasing physical stress. The forms that are present at rest or at low levels of activity and are suppressed with greater levels of activity do not require therapy if the patient is asymptomatic, whereas those that appear with exercise or appear to be catecholamine dependent often respond to β -blocker therapy. In the absence of structural heart disease, athletes with this pattern, particularly if relatively slow (<150 bpm during peak activity) and asymptomatic, can be cleared to participate in athletics without restrictions, but the workup to reach this level of recommendation must be thorough, including stress testing and appropriate imaging, particularly to exclude occult heart disease. The prognosis for these patterns occurring at faster rates (eg, >170 bpm) is less clear. Ablation is a reasonable therapy for idiopathic sustained monomorphic VT. If successful and there is no recurrence after a reasonable time interval (3 months), then return to play is allowed. For patients with structural, molecular, or inflammatory disorders who have sustained monomorphic VT at rest or exercise, athletic activity is prohibited. For acute forms of myocarditis, return to athletic activities is permissible if and when the disorder resolves.

Recommendations

1. Athletes with structurally normal hearts and monomorphic sustained VT amenable to catheter ablation who undergo ablation and remain free of spontaneous or induced VT at least 3 months after the procedure can resume full competitive activities (Class I; Level of Evidence C).
2. Athletes with structurally normal hearts and monomorphic sustained VT who elect to undergo drug suppression with pharmacological therapy should not compete in any sports for at least 3 months after the last VT episode. In the absence of clinical recurrences or inducibility of the arrhythmia by exercise/exercise testing or EPS, all competitive sports may then be permitted (Class I; Level of Evidence C).
3. For the athlete with structural heart disease and sustained monomorphic VT, moderate- and high-intensity competition is contraindicated regardless of apparent therapeutic response, although participation in low-intensity class IA competitive sports is permitted (Class III; Level of Evidence C).

Sustained Polymorphic VT, Ventricular Flutter, and Ventricular Fibrillation

Athletes who manifest these arrhythmias in the presence or absence of structural heart disease or defined molecular/genetic disorders generally receive implantable cardioverter-defibrillators (ICDs). Athletes who have these arrhythmias in the setting of transient inflammatory or electrolyte disorders may be an exception in that they may not receive ICDs, and if they remain free of episodes of these arrhythmias for 3 months after resolution of the inflammatory process, they may be considered for reevaluation of clearance to participate.

Recommendations

- 1. Athletes who have survived a cardiac arrest caused by ventricular fibrillation or VT or who have had documented symptomatic rapid VT associated with a defined nonreversible cardiac abnormality (structural or molecular) or unidentified cause should have an ICD placed. See “Athletes With ICDs” for recommendations regarding competitive sports participation after ICD implantation (Class I; Level of Evidence A).**
- 2. Class IIb athletes who have survived a cardiac arrest caused by ventricular fibrillation or VT or who have had documented symptomatic rapid VT associated with a defined reversible abnormality (eg, resolved acute myocarditis or a controllable electrolyte abnormality) may be considered for reinstatement of participation after reevaluation at 3 months (Class I; Level of Evidence C).**

Syncope

Syncope is a transient loss of consciousness caused by transient global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery.^{24–26} Syncope in the athlete can result from relatively benign causes such as cerebral hypoperfusion because of physiology similar to that found with the common faint or neurally mediated syncope.^{27–30} Less frequently, syncope results from serious cardiovascular conditions that result in transient loss of cerebral blood flow because of an obstruction or arrhythmias associated with underlying structural heart disease.³¹ Primary electrical disorders can result in syncope in the absence of any structural heart disease.³²

Syncope or presyncope in an athlete mandates a thorough evaluation by a qualified clinician.³³ The purpose of the evaluation is to determine the cause of syncope, with particular emphasis on detecting structural or electrical heart disease that may lead to sudden death. The evaluation should include a detailed history that includes specific details of the event and observations of witnesses when available. The distinction between syncope during exercise and postexertional syncope is clinically important. Most syncopal episodes that occur immediately after exercise are benign. This pattern is believed to be a result of transient postural hypotension caused by lower-extremity pooling of blood once the athlete stops the activity (from exercise-induced vasodilation) and the resultant impairment of cardiac

baroreflexes.³⁴ It may be potentiated by relative or absolute bradycardia attributable to a parasympathetic surge at the cessation of exercise. By contrast, syncope during exercise has a higher probability of being caused by serious underlying cardiovascular disease; however, neurally mediated syncope also can be induced by prolonged intense exercise.

The history should include asking about a family history of syncope, cardiovascular disease, and sudden death. A careful physical examination with particular attention to the cardiovascular examination should be performed in all athletes. Subsequent diagnostic testing in all patients should include an ECG and an echocardiogram, with selective use of additional cardiovascular tests. These tests may include a tilt table test, exercise stress test, ambulatory monitoring, and an implantable loop monitor. The sensitivity and specificity of tilt table testing for the diagnosis of syncope in the competitive athlete are lower than for the general population, and some experts believe there is not a role for tilt testing in the workup.³⁵ For those patients in whom the cause of syncope remains uncertain, especially if the syncope raises concern for arrhythmic causes, contrast-enhanced magnetic resonance imaging, cardiac computed tomography, coronary angiography, and invasive electrophysiological testing may be indicated. Provocative testing with stress testing, epinephrine, procainamide, or isoproterenol should be considered to identify otherwise concealed cases of long-QT syndrome, catecholaminergic polymorphic VT, and Brugada syndrome. Genetic testing may be clinically useful in selected cases.³⁶

Neurally mediated syncope is generally compatible with continued athletic participation once measures are taken to mitigate the syncope. The primary responsibility of the clinician is to definitively exclude structural heart disease or primary electrical disorders that may predispose to sudden death or recurrent syncope. In a significant minority of athletes, the cause of syncope cannot be established despite a thorough evaluation. Athletes with syncope of unknown cause should not participate in athletics in which the transient loss of consciousness can be hazardous.

Recommendations

- 1. Athletes with exercise-induced syncope should be restricted from all competitive athletics until evaluated by a qualified medical professional (Class I; Level of Evidence B).**
- 2. Athletes with syncope should be evaluated with a history, physical examination, ECG, and selective use of other diagnostic tests when there is suspicion of structural heart disease or primary electrical abnormalities that may predispose to recurrent syncope or sudden death (Class I; Level of Evidence C).**
- 3. Athletes with syncope caused by structural heart disease or primary electrical disorders should be restricted from athletic activities according to the recommendations for their specific underlying cardiovascular condition (Class I; Level of Evidence C).**
- 4. Athletes with neurally mediated syncope can resume all athletic activities once measures are demonstrated to prevent recurrent syncope (Class I; Level of Evidence C).**

5. Athletes with syncope of unknown cause, based on a ruling out of structural or molecular pathogenesis, should not participate in athletics in which transient loss of consciousness can be hazardous (Class III; Level of Evidence C).

Athletes With ICDs

As ICDs achieved recognition of efficacy for primary and secondary prevention of SCD, based on clinical trial and observational data, the specific question of participation of ICD recipients in competitive athletics arose. Although the various guideline documents have not addressed this issue directly, the 36th Bethesda Conference offered both general opinion³⁷ and several disease-specific recommendations that athletes with ICDs should limit competitive sports to class IA–level activities. This was based largely on reasoned notions, in the absence of observational data, concerning the effect of the physiology and biochemistry of high-intensity activities and underlying structural disease states on reliability of device therapy, the possibility of device malfunction, and the risk of injury to the athlete or damage to the device by trauma. Appropriate or inappropriate discharges were also cited as potential concerns. The recommendation against competition sports participation by athletes with ICDs is being reevaluated on the basis of reported practice patterns and recently generated observational data.^{38,39}

Recommendations

1. ICD indications for competitive athletes should not differ from those applicable to the general population with appropriate diagnoses and clinical profiles (Class I; Level of Evidence C).
2. Recommendations should be based on existing evidence for benefit and risk and should include discussions of potential impact on sport-specific participation and performance (Class I; Level of Evidence C).
3. Participation in sports classified as IA for athletes with an ICD is reasonable if they are free of episodes of ventricular flutter or ventricular fibrillation requiring device therapy for 3 months (Class IIa; Level of Evidence C).
4. Participation in sports with higher peak static and dynamic components than class IA may be considered if the athlete is free of episodes of ventricular flutter or ventricular fibrillation requiring device therapy for 3 months. The decision regarding athletic participation should be made with consideration of, and counseling of, the athlete regarding the higher likelihood of appropriate and inappropriate shocks and the potential for device-related trauma in high-impact sports (Class IIb; Level of Evidence C).
5. The desire of the athlete to continue athletic competition should not represent the primary indication for use of an ICD (Class III; Level of Evidence C).

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*Modest.

†Significant.

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*Modest.

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References

- Zipes DP, Ackerman MJ, Estes NAM 3rd, Grant AO, Myerburg RJ, Van Hare G. Task Force 7: arrhythmias: 36th Bethesda Conference: eligibility recommendations for competitive athletes with cardiovascular abnormalities. *J Am Coll Cardiol*. 2005;45:1354–1363. doi: 10.1016/j.jacc.2005.02.014.
- Epstein AE, DiMarco JP, Ellenbogen KA, Estes NA 3rd, Freedman RA, Gettes LS, Gillinov AM, Gregoratos G, Hammill SC, Hayes DL, Hlatky MA, Newby LK, Page RL, Schoenfeld MH, Silka MJ, Stevenson LW, Sweeney MO. ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) [published correction appears in *Circulation*. 2009;120:e34–e35]. *Circulation*. 2008;117:e350–e408. doi: 10.1016/CIRCULATIONAHA.108.189742.
- Tracy CM, Epstein AE, Darbar D, DiMarco JP, Dunbar SB, Estes NA 3rd, Ferguson TB Jr, Hammill SC, Karasik PE, Link MS, Marine JE, Schoenfeld MH, Shanker AJ, Silka MJ, Stevenson LW, Stevenson WG, Varosy PD, Ellenbogen KA, Freedman RA, Gettes LS, Gillinov AM, Gregoratos G, Hayes DL, Page RL, Stevenson LW, Sweeney MO. 2012 ACCF/AHA/HRS focused update of the 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society [corrected] [published correction appears in *Circulation*. 2013;127:e357–e359]. *Circulation*. 2012;126:1784–1800. doi: 10.1161/CIR.0b013e3182618569.
- Kim JH, Noseworthy PA, McCarty D, Yared K, Weiner R, Wang F, Wood MJ, Hutter AM, Picard MH, Baggish AL. Significance of electrocardiographic right bundle branch block in trained athletes. *Am J Cardiol*. 2011;107:1083–1089. doi: 10.1016/j.amjcard.2010.11.037.
- Fisch C, Zipes DP, McHenry PL. Rate dependent aberrancy. *Circulation*. 1973;48:714–724.
- Moak JP, Barron KS, Hougen TJ, Wiles HB, Balaji S, Sreeram N, Cohen MH, Nordenberg A, Van Hare GF, Friedman RA, Perez M, Cecchin F, Schneider DS, Nehme RA, Buyon JP. Congenital heart block: development of late-onset cardiomyopathy, a previously underappreciated sequela. *J Am Coll Cardiol*. 2001;37:238–242.
- Bordachar P, Zachary W, Ploux S, Labrousse L, Haissaguerre M, Thambo JB. Pathophysiology, clinical course, and management of congenital complete atrioventricular block. *Heart Rhythm*. 2013;10:760–766. doi: 10.1016/j.hrthm.2012.12.030.
- Furlanello F, Bertoldi A, Dallago M, Galassi A, Fernando F, Biffi A, Mazzone P, Pappone C, Chierchia S. Atrial fibrillation in elite athletes. *J Cardiovasc Electrophysiol*. 1998;9(suppl):S63–S68.
- Grimsmo J, Grundvold I, Maehlum S, Arnesen H. High prevalence of atrial fibrillation in long-term endurance cross-country skiers: echocardiographic findings and possible predictors: a 28-30 years follow-up study. *Eur J Cardiovasc Prev Rehabil*. 2010;17:100–105. doi: 10.1097/HJR.0b013e32833226be.
- Page RL, Joglar JA, Al-Khatib SM, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. [published online ahead of print September 23, 2015]. *Circulation*. doi: 10.1161/CIR.0000000000000312.
- Koopman P, Nuyens D, Garweg C, La Gerche A, De Buck S, Van Casteren L, Alzand B, Willems R, Heidbuchel H. Efficacy of radiofrequency catheter ablation in athletes with atrial fibrillation. *Europace*. 2011;13:1386–1393. doi: 10.1093/europace/eur142.
- Calkins H, Kuck KH, Cappato R, Brugada J, Camm AJ, Chen SA, Crijns HJ, Damiano RJ Jr, Davies DW, DiMarco J, Edgerton J, Ellenbogen K, Ezekowitz MD, Haines DE, Haissaguerre M, Hindricks G, Iesaka Y, Jackman W, Jalife J, Jais P, Kalman J, Keane D, Kim YH, Kirchhof P, Klein G, Kottkamp H, Kumagai K, Lindsay BD, Mansour M, Marchlinski FE, McCarthy PM, Mont JL, Morady F, Nademanee K, Nakagawa H, Natale A, Nattel S, Packer DL, Pappone C, Prystowsky E, Raviele A, Reddy V, Ruskin JN, Shemin RJ, Tsao HM, Wilber D; Heart Rhythm Society Task Force on Catheter and Surgical Ablation of Atrial Fibrillation. 2012 HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: recommendations for patient selection, procedural techniques, patient management and follow-up, definitions, end-points, and research trial design: a report of the Heart Rhythm Society (HRS) Task Force on Catheter and Surgical Ablation of Atrial Fibrillation. Developed in partnership with the European Heart Rhythm Association (EHRA), a registered branch of the European Society of Cardiology (ESC) and the European Cardiac Arrhythmia Society (ECAS); and in collaboration with the American College of Cardiology (ACC), American Heart Association (AHA), the Asia Pacific Heart Rhythm Society (APHRS), and the Society of Thoracic Surgeons (STS): endorsed by the governing bodies of the American College of Cardiology Foundation, the American Heart Association, the European Cardiac Arrhythmia Society, the European Heart Rhythm Association, the Society of Thoracic Surgeons, the Asia Pacific Heart Rhythm Society, and the Heart Rhythm Society. *Heart Rhythm*. 2012;9:632–696.e21. doi: 10.1016/j.hrthm.2011.12.016.
- Pappone C, Manguso F, Santinelli R, Vicedomini G, Sala S, Paglino G, Mazzone P, Lang CC, Gulletta S, Augello G, Santinelli O, Santinelli V. Radiofrequency ablation in children with asymptomatic Wolff-Parkinson-White syndrome. *N Engl J Med*. 2004;351:1197–1205. doi: 10.1056/NEJMoa040625.
- Cohen MI, Triedman JK, Cannon BC, Davis AM, Drago F, Janousek J, Klein GJ, Law IH, Morady FJ, Paul T, Perry JC, Sanatani S, Tanel RE. PACES/HRS expert consensus statement on the management of the asymptomatic young patient with a Wolff-Parkinson-White (WPW, ventricular preexcitation) electrocardiographic pattern: developed in partnership between the Pediatric and Congenital Electrophysiology Society (PACES) and the Heart

- Rhythm Society (HRS). Endorsed by the governing bodies of PACES, HRS, the American College of Cardiology Foundation (ACCF), the American Heart Association (AHA), the American Academy of Pediatrics (AAP), and the Canadian Heart Rhythm Society (CHRS). *Heart Rhythm*. 2012;9:1006–1024.
15. Biffi A, Pelliccia A, Verdile L, Fernando F, Spataro A, Caselli S, Santini M, Maron BJ. Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes. *J Am Coll Cardiol*. 2002;40:446–452.
 16. Steriotis AK, Nava A, Rigato I, Mazzotti E, Daliento L, Thiene G, Basso C, Corrado D, Bauce B. Noninvasive cardiac screening in young athletes with ventricular arrhythmias. *Am J Cardiol*. 2013;111:557–562. doi: 10.1016/j.amjcard.2012.10.044.
 17. Lampert R. Evaluation and management of arrhythmia in the athletic patient. *Prog Cardiovasc Dis*. 2012;54:423–431. doi: 10.1016/j.pcad.2012.01.002.
 18. Myerburg RJ, Castellanos A. Cardiac arrest and sudden cardiac death. In: Bonow RO, Mann DL, Zipes DP, Libby P, eds. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. Oxford, United Kingdom: Elsevier; 2010.
 19. Heidbüchel H, Corrado D, Biffi A, Hoffmann E, Panhuyzen-Goedkoop N, Hoogsteen J, Delise P, Hoff PI, Pelliccia A; Study Group on Sports Cardiology of the European Association for Cardiovascular Prevention and Rehabilitation. Recommendations for participation in leisure-time physical activity and competitive sports of patients with arrhythmias and potentially arrhythmogenic conditions, part II: ventricular arrhythmias, channelopathies and implantable defibrillators. *Eur J Cardiovasc Prev Rehabil*. 2006;13:676–686. doi: 10.1097/01.hjr.0000239465.26132.29.
 20. Biffi A, Maron BJ, Culasso F, Verdile L, Fernando F, Di Giacinto B, Di Paolo FM, Spataro A, Delise P, Pelliccia A. Patterns of ventricular tachyarrhythmias associated with training, deconditioning and retraining in elite athletes without cardiovascular abnormalities. *Am J Cardiol*. 2011;107:697–703. doi: 10.1016/j.amjcard.2010.10.049.
 21. Albert CM, Mittleman MA, Chae CU, Lee IM, Hennekens CH, Manson JE. Triggering of sudden death from cardiac causes by vigorous exertion. *N Engl J Med*. 2000;343:1355–1361. doi: 10.1056/NEJM200011093431902.
 22. Friedrich MG, Sechtem U, Schulz-Menger J, Holmvang G, Alakija P, Cooper LT, White JA, Abdel-Aty H, Gutberlet M, Prasad S, Aletras A, Laissy JP, Paterson I, Filipchuk NG, Kumar A, Pauschinger M, Liu P; International Consensus Group on Cardiovascular Magnetic Resonance in Myocarditis. Cardiovascular magnetic resonance in myocarditis: a JACC White Paper. *J Am Coll Cardiol*. 2009;53:1475–1487. doi: 10.1016/j.jacc.2009.02.007.
 23. Dello Russo A, Pieroni M, Santangeli P, Bartoletti S, Casella M, Pelargonio G, Smaldone C, Bianco M, Di Biase L, Bellocci F, Zeppilli P, Fiorentini C, Natale A, Tondo C. Concealed cardiomyopathies in competitive athletes with ventricular arrhythmias and an apparently normal heart: role of cardiac electroanatomical mapping and biopsy. *Heart Rhythm*. 2011;8:1915–1922. doi: 10.1016/j.hrthm.2011.07.021.
 24. Moya A, Sutton R, Ammirati F, Blanc JJ, Brignole M, Dahm JB, Deharo JC, Gajek J, Gjesdal K, Krahn A, Massin M, Pepi M, Pezawas T, Ruiz Granell R, Sarasin F, Ungar A, van Dijk JG, Walma EP, Wieling W. Guidelines for the diagnosis and management of syncope (version 2009). *Eur Heart J*. 2009;30:2631–2671.
 25. Freeman R, Wieling W, Axelrod FB, Benditt DG, Benarroch E, Biaggioni I, Cheshire WP, Chelmsky T, Cortelli P, Gibbons CH, Goldstein DS, Hainsworth R, Hiltz MJ, Jacob G, Kaufmann H, Jordan J, Lipsitz LA, Levine BD, Low PA, Mathias C, Raj SR, Robertson D, Sandroni P, Schatz I, Schondorff R, Stewart JM, van Dijk JG. Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. *Clin Auton Res*. 2011;21:69–72. doi: 10.1007/s10286-011-0119-5.
 26. Strickberger SA, Benson DW, Biaggioni I, Callans DJ, Cohen MI, Ellenbogen KA, Epstein AE, Friedman P, Goldberger J, Heidenreich PA, Klein GJ, Knight BP, Morillo CA, Myerburg RJ, Sila CA. AHA/ACCF scientific statement on the evaluation of syncope: from the American Heart Association Councils on Clinical Cardiology, Cardiovascular Nursing, Cardiovascular Disease in the Young, and Stroke, and the Quality of Care and Outcomes Research Interdisciplinary Working Group; and the American College of Cardiology Foundation. *Circulation*. 2006;113:316–327. doi: 10.1161/CIRCULATIONAHA.105.170274.
 27. Link MS, Estes NA 3rd. How to manage athletes with syncope. *Cardiol Clin*. 2007;25:457–466, vii. doi: 10.1016/j.ccl.2007.07.005.
 28. Grubb BP. Clinical practice. Neurocardiogenic syncope. *N Engl J Med*. 2005;352:1004–1010. doi: 10.1056/NEJMcp042601.
 29. Asplund CA, O'Connor FG, Noakes TD. Exercise-associated collapse: an evidence-based review and primer for clinicians. *Br J Sports Med*. 2011;45:1157–1162. doi: 10.1136/bjsports-2011-090378.
 30. Colivicchi F, Ammirati F, Santini M. Epidemiology and prognostic implications of syncope in young competing athletes. *Eur Heart J*. 2004;25:1749–1753. doi: 10.1016/j.ehj.2004.07.011.
 31. Khoo C, Chakrabarti S, Arbour L, Krahn AD. Recognizing life-threatening causes of syncope. *Cardiol Clin*. 2013;31:51–66. doi: 10.1016/j.ccl.2012.10.005.
 32. Krahn AD, Healey JS, Simpson CS, Chauhan VS, Birnie DH, Champagne J, Gardner M, Sanatani S, Chakrabarti S, Yee R, Skanes AC, Leong-Sit P, Ahmad K, Gollob MH, Klein GJ, Gula LJ, Sheldon RS. Sentinel symptoms in patients with unexplained cardiac arrest: from the Cardiac Arrest Survivors With Preserved Ejection Fraction Registry (CASPER). *J Cardiovasc Electrophysiol*. 2012;23:60–66. doi: 10.1111/j.1540-8167.2011.02185.x.
 33. O'Connor FG, Levine BD, Childress MA, Asplund CA, Oriscello RG. Practical management: a systematic approach to the evaluation of exercise-related syncope in athletes. *Clin J Sport Med*. 2009;19:429–434. doi: 10.1097/JSM.0b013e3181b732c3.
 34. Levine BD, Lane LD, Buckley JC, Friedman DB, Blomqvist CG. Left ventricular pressure-volume and Frank-Starling relations in endurance athletes: implications for orthostatic tolerance and exercise performance. *Circulation*. 1991;84:1016–1023.
 35. Hastings JL, Levine BD. Syncope in the athletic patient. *Prog Cardiovasc Dis*. 2012;54:438–444. doi: 10.1016/j.pcad.2012.02.003.
 36. Ackerman MJ, Priori SG, Willems S, Berul C, Brugada R, Calkins H, Camm AJ, Ellinor PT, Gollob M, Hamilton R, Hersheberger RE, Judge DP, Le Marec H, McKenna WJ, Schulze-Bahr E, Semsarian C, Towbin JA, Watkins H, Wilde A, Wolpert C, Zipes DP. HRS/EHRA expert consensus statement on the state of genetic testing for the channelopathies and cardiomyopathies: this document was developed as a partnership between the Heart Rhythm Society (HRS) and the European Heart Rhythm Association (EHRA). *Heart Rhythm*. 2011;8:1308–1339. doi: 10.1016/j.hrthm.2011.05.020.
 37. Maron BJ, Zipes DP. Introduction: eligibility recommendations for competitive athletes with cardiovascular abnormalities-general considerations. *J Am Coll Cardiol*. 2005;45:1318–1321. doi: 10.1016/j.jacc.2005.02.006.
 38. Lampert R, Cannon D, Olshansky B. Safety of sports participation in patients with implantable cardioverter defibrillators: a survey of Heart Rhythm Society members. *J Cardiovasc Electrophysiol*. 2006;17:11–15. doi: 10.1111/j.1540-8167.2005.00331.x.
 39. Lampert R, Olshansky B, Heidbüchel H, Lawless C, Saarel E, Ackerman M, Calkins H, Estes NA, Link MS, Maron BJ, Marcus F, Scheinman M, Wilkoff BL, Zipes DP, Berul CI, Cheng A, Law I, Loomis M, Barth C, Brandt C, Dziura J, Li F, Cannon D. Safety of sports for athletes with implantable cardioverter-defibrillators: results of a prospective, multinational registry. *Circulation*. 2013;127:2021–2030. doi: 10.1161/CIRCULATIONAHA.112.000447.

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Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 9: Arrhythmias and Conduction Defects: A Scientific Statement From the American Heart Association and American College of Cardiology

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