Task Force 7: Arrhythmias

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GENERAL CONSIDERATIONS

Guidelines for athletic participation are needed to reduce the risk for arrhythmia-related morbidity or mortality. However, it is often difficult to establish the importance of a cardiac rhythm disturbance in assessing an athlete's eligibility for competition. Few data exist that have been obtained prospectively from well-designed, scientifically acceptable studies to determine whether a particular rhythm disturbance predisposes an athlete to sudden death or to symptoms, such as syncope or presyncope, that could precipitate severe injury. Sudden unexpected cardiac death in the young is rare, estimated at less than 1% of that observed in adults. Nonetheless, a significant proportion of these deaths occur in relation to exercise (1). The deaths of several prominent athletes have focused attention on athletes with known arrhythmias.

Arrhythmias commonly are evanescent, often disappearing unpredictably for long periods of time, in some cases years. If they recur when the athlete is not exercising, the arrhythmia may not be noted or may not produce significant symptoms. The same arrhythmia may minimally affect a competitive golfer but severely incapacitate other athletes, such as cross-country skiers, performing at peak physical effort. The athlete may not develop the arrhythmia during each sporting event. Although the reasons for this are not known, factors related to the autonomic nervous system probably play a very important role in determining whether an arrhythmia occurs and its rate and effect on hemodynamic responses and symptoms. Autonomic "tone" probably varies greatly and perhaps unpredictably between and within athletic events and from one athlete to another. Mental stress during competition can produce important electrophysiologic and hemodynamic changes that are probably mediated through the autonomic nervous system.

It is important to understand the range of normal heart rate and rhythm for the trained athlete. Heart rates of 25 beats/min and sinus pauses lasting greater than 2 s may be found on 24-h Holter ambulatory electrocardiographic (ECG) recordings. Type I second-degree atrioventricular (AV) block and single uniform premature ventricular complexes each may occur in approximately 40% of athletes. Complex ventricular arrhythmias (multiform premature ventricular complexes, couplets, nonsustained ventricular tachycardia) are less common (2,3).

Many of our conclusions result from data obtained in non-athletes, from general perceptions, or experience and from a heavy input of "what seems reasonable." Decision-making based on this type of logic is often faulty but is the best available. Recommendations and guidance need to be balanced between an effort to avoid restricting activity unduly and the hope of reducing the risk of death and injury due to a rhythm disturbance.

Despite the lack of complete information, some firm conclusions can be reached. Certain arrhythmias, such as ventricular tachyarrhythmias, create symptoms and are dangerous in and of themselves regardless of the clinical situation in which they occur (4). These arrhythmias generally are characterized by very rapid or very slow heart rates that significantly compromise cardiac output, coronary or cerebral blood flow, or maintenance of blood pressure. Such arrhythmias may include atrial flutter or atrial fibrillation with uncontrolled ventricular rates of 200 to 300 beats/min, usually (but not exclusively) in athletes with Wolff-Parkinson-White (WPW) syndrome, rapid sustained ventricular tachycardias, and AV block, or sinus node disease with very slow ventricular rates. Certain persistent arrhythmias, such as chronic tachycardias, can worsen cardiac function by a process called "remodeling" (5). Other arrhythmias, such as AV nodal re-entrant tachycardia, generally well-tolerated in most people, may not produce important symptoms at rest but only during exercise in athletes with structural heart disease due, in part, to an increase in the tachycardia rate. Arrhythmias that might otherwise be innocuous and no more than a nuisance might, under conditions of participation in certain sports involving body contact or high speed, place the athlete at risk of injury or death because of transient impaired mental function causing loss of physical control. For example, it is possible that an athlete with supraventricular tachycardia participating in potentially dangerous sports, such as diving, downhill skiing, or auto racing, may be at greater risk because of dizziness, near syncope (a feeling of impending loss of consciousness), or syncope than if he or she were playing basketball or baseball.

The search for significant structural heart disease is an important element in evaluating athletes with arrhythmias prior to sports participation. Some athletes with coronary artery disease (CAD), hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy (ARVC), aortic stenosis, some inherited cardiac channelopathies like congenital long QT syndrome (LQTS) (6), and other forms of congenital heart disease, including repaired congenital heart disease, are probably at greater risk for cardiac arrest and sudden death during and, perhaps, just after exercise.

This is probably true whether or not arrhythmias have been recognized previously. In general, athletes with symptoms possibly related to cardiac arrhythmia, such as exertional- or auditory-triggered syncope, near syncope, and palpitations, should be carefully evaluated (see Task Force 2: Congenital Heart Disease) before being permitted to participate in competitive sports. A consideration of cardiac hemodynamic status is critical because right or left ventricular dysfunction is an additional important predictor of arrhythmic death. The presence of a significant rhythm disturbance, such as a rapid supraventricular or ventricular tachyarrhythmia in athletes with abnormal cardiac hemodynamic status (from any cause), itself is definitely incompatible with participation in all competitive sports. However, it is important to emphasize that some disease states, such as a myocarditis, can produce arrhythmias that can be self-limited, with subsequent full recovery.

In general, all athletes with significant cardiac arrhythmias being considered for athletic activity should have a careful cardiac examination, a 12-lead ECG, echocardiogram, exercise test, and, in some, a long-term 24-h Holter ambulatory ECG recording, if possible during the specific type of exercise being considered. Arrhythmias, as discussed in the various Task Force documents, are usually identified by exercise testing or some form of long-term monitoring (including ambulatory Holter and event recording). Arrhythmias precipitated during the specific type of exercise being considered can be important because a conventional exercise test may not replicate the specific clinical situation produced by actively participating in the sport. In this regard, exercise tests may need to be adapted specifically for the athlete; that is, to begin exercise at peak energy expenditure, as a sprinter in a race might, rather than with the slow increase in workload commonly used in testing athletes with CAD. Resuscitation equipment and trained personnel may be needed on a standby basis.

All athletes with an arrhythmia who are permitted to engage in athletics should be re-evaluated at 6 to 12 month intervals after they are trained, to determine whether the conditioning process affected the arrhythmia. It should also be stressed that athletes with arrhythmias controlled by antiarrhythmic drugs may stop taking these drugs for a variety of reason and, therefore, compliance with recommended therapy, as well as evaluation for recurrence of symptoms, must be established periodically. Abuse with drugs like cocaine or ephedra can precipitate life-threatening arrhythmias, and such considerations are an important part of the evaluation. Of note, the use of certain cardioactive drugs, such as beta-adrenergic blocking agents, is banned in some competitive sports (see the Introduction). In addition, it is important to realize that the catecholamines released during exercise may counteract the salutary effects of some antiarrhythmic agents. For some arrhythmias, an ablation approach, usually with a catheter and usually with radiofrequency energy, to eliminate the arrhythmia may be preferable to drug treatment. Other ablation energies, such as cryoablation, or other approaches such as surgery, can be used as indicated. After successful ablation of the arrhythmia, a return to athletics can be within days for those in whom repeated attempts at tachycardia induction during isoproterenol administration is unsuccessful, and in whom the tachycardia was easily induced prior to ablation. For those in whom such provocative testing is not performed, waiting two to four weeks seems advisable.

It may be difficult for team physicians and consultants from the local community to make objective decisions to restrict or proscribe sports for a competitive athlete. Borderline cases can be reviewed by non-biased experts for the purpose of helping make these decisions. Whether that approach can be practically implemented has not been determined. Alternatively, it has also been suggested that in such borderline cases, both appropriate emergency equipment and medical professionals versed in the operation of that equipment be present at all practices and games. However, there is considerable concern with regard to this practice because its safety and efficacy have not been established. Indeed, the practical implementation and reliability of this approach to reverse potentially lethal arrhythmias in out-of-hospital settings for the competing athlete is highly questionable.

SYNCOPE

Unexplained syncope in an athlete is a potentially important symptom that mandates a thorough evaluation. It may be due to a variety of causes, including cardiovascular disease; alternatively, it may not be associated with either structural heart disease or a primary electrical disorder but rather to mechanisms such as vasovagal syncope, which is a common finding in highly trained athletes. Although vasovagal syncope may be compatible with continued participation in all competitive sports, caution should be used in making this diagnosis in highly trained athletes without first definitively excluding underlying structural cardiovascular disease. A cardiac arrhythmia should be considered, particularly when syncope occurs during or immediately after exercise.

A cause of syncope can be established in approximately 50% of patients. Often, a careful history and physical examination will identify the etiology (7). When such an evaluation does not determine the cause, further testing in search of an arrhythmia is indicated. Ambulatory ECG recordings are often unrevealing but nevertheless probably worthwhile to obtain during the initial evaluation. Event or loop recorders or implantable devices can be used to increase the ECG sampling time. Tilt-table testing has been used to assess patients at risk for vasovagal syncope, but the lack of specificity of this test (particularly in endurance-trained athletes in whom false-positive results can occur) requires a particularly cautious interpretation of the results. Exercise testing is useful and is optimally performed while recording the ECG during the athletic activity in which the person

participates. Provocative catecholamine stress testing with either epinephrine, procainamide, or isoproterenol may be useful to unmask cases of concealed LQTS, Brugada syndrome (BrS), or catecholaminergic polymorphic ventricular tachycardia (CPVT) (8). Invasive electrophysiologic testing is most likely to identify an arrhythmia responsible for syncope in those patients with structural heart disease or an abnormal ECG but can be considered in other athletes when no other cause of the syncope has been identified, remembering that the diagnostic yield of invasive electrophysiologic testing is low in the absence of structural heart disease.

TYPES OF ARRHYTHMIAS

Disturbances of sinus node function. Sinus tachycardia and sinus bradycardia appropriate for the clinical situation are not considered abnormal, and no tests are necessary. Sinus arrhythmia and wandering pacemaker are generally considered normal, and no tests are necessary unless the arrhythmias result in inappropriately slow rates accompanied by symptoms. Sinus arrhythmia and sinus bradycardia are particularly common in the trained athlete.

Asymptomatic sinus pause or sinus arrest (less than 3 s) is probably of no significance. Longer symptomatic pauses, sinoatrial exit block, and sick sinus syndrome are considered abnormal, and athletes should have a 12-lead, 24-h ECG, and an exercise test. Mutations involving the cardiac sodium channel encoded by *SCN5A* has been demonstrated in some patients with progressive cardiac conduction disease and congenital sick sinus syndrome (9,10). In an occasional athlete experiencing syncope or near syncope, an electrophysiologic study may be indicated and reveal abnormal sinus node function, but in general, invasive electrophysiologic testing is not helpful. Echocardiography should be performed to exclude structural heart disease; other tests to evaluate ventricular or valvular function may be indicated.

Recommendations:

1. **Athletes with a normal or structurally abnormal heart in whom the bradycardic rate is increased appropriately by physical activity can participate in all competitive sports consistent with the limitations imposed by the structural heart disease. They should be reassessed periodically to determine that training does not aggravate the bradycardia.**
2. **Athletes with syncope or near syncope should not participate in sports where the likelihood of even a momentary loss of consciousness may be hazardous until the cause has been determined and treated, if necessary.**
3. **Athletes with symptoms such as impaired consciousness and fatigue clearly attributed to the arrhythmias should be treated and if asymptomatic for two to three months during treatment, they can participate in all competitive sports after physician re-evaluation.**
4. **Athletes with symptomatic tachycardia/bradycardia syndrome or inappropriate sinus tachycardia should be treated. If no structural heart disease and asymptomatic for two to three months, they can participate in all competitive sports.**
5. **Athletes with pacemakers should not engage in sports with a danger of bodily collision because such trauma may damage the pacemaker system. This restriction should clearly exclude activities where direct blows to the chest are a part of the sport, such as football, rugby, boxing, martial arts, hockey, and lacrosse. Protective padding for the device is advisable for other sports such as soccer, basketball, baseball, and softball where trauma is possible but less likely.**

Premature atrial complexes. In the absence of evidence of structural heart disease and in the absence of symptoms other than occasional palpitation, no evaluation other than a 12-lead ECG is necessary.

Recommendation:

1. **Athletes can participate in all competitive sports.**

Atrial flutter (in the absence of WPW syndrome). In the absence of an acute, limiting illness, sustained atrial flutter is an uncommon rhythm disturbance in athletes without structural heart disease. Therefore, an echocardiogram should be performed to evaluate cardiovascular structure and function. Because the potential for very rapid ventricular rates exists if the atrial flutter conducts 1:1 to the ventricles, ECG determination of the ventricular response during an exercise test or athletic event during treatment is essential. For some patients with paroxysmal atrial flutter, induction of the arrhythmia by electrical stimulation may be considered before the exercise test, recognizing the difficult logistics in doing this. A 12-lead ECG and, at times, long-term 24-h ECG may be necessary. Asymptomatic athletes who have transient episodes of atrial flutter lasting less than 10 s that do not increase in duration during exercise can participate in all sports.

Recommendations:

1. **Athletes with atrial flutter in the absence of structural heart disease who maintain a ventricular rate that increases and slows appropriately comparable to that of a normal sinus response in relation to the level of activity, while receiving no therapy or therapy with AV nodal blocking drugs, can participate in class IA competitive sports with the warning that rapid 1:1 conduction still may occur. However, full participation in all competitive sports should not be allowed unless the athlete has been without atrial flutter for two to three months with or without drug treatment. Note that the use of beta-blockers is prohibited in some competitive sports (see Introduction).**

2. **Athletes with structural heart disease who have atrial flutter can participate in class IA competitive sports only after two to four weeks have elapsed without an episode of atrial flutter.**
3. **Athletes without structural heart disease who have elimination of the atrial flutter by an ablation technique or surgery can participate in all competitive sports after two to four weeks without a recurrence, or in several days after an electrophysiologic study showing non-inducibility of the atrial flutter in the presence of bi-directional isthmus block.**
4. **Athletes in whom anticoagulation is deemed necessary cannot participate in competitive sports where the danger of bodily collision is present.**

Atrial fibrillation (in the absence of WPW syndrome). Atrial fibrillation is far more common than atrial flutter and may be present intermittently or chronically (11,12). Evaluation should include a search for the cause, such as thyrotoxicosis. More often atrial fibrillation occurs in association with diseases such as CAD or hypertension. Evaluation includes determination of the ventricular response during athletic activity or an exercise test comparable to the intended athletic competition. For some patients with paroxysmal atrial fibrillation, electrical induction of atrial fibrillation before the exercise test may be necessary, recognizing the difficult logistics in doing this. A 12-lead ECG is necessary, and long-term 24-h ECG recordings and an echocardiogram are helpful in establishing the presence of structural heart disease. Asymptomatic athletes who have episodes of atrial fibrillation of 5 to 15 s that do not increase in duration during exercise can participate in all sports.

Recommendations:

1. **Athletes with asymptomatic atrial fibrillation in the absence of structural heart disease who maintain a ventricular rate that increases and slows appropriately and is comparable to that of a normal sinus response in relation to the level of activity, while receiving no therapy or therapy with AV nodal-blocking drugs, can participate in all competitive sports. Note that the use of beta-blockers is prohibited in some competitive sports.**
2. **Athletes who have atrial fibrillation in the presence of structural heart disease who maintain a ventricular rate comparable to that of an appropriate sinus tachycardia during physical activity while receiving no therapy or therapy with AV nodal-blocking drugs can participate in sports consistent with the limitations of the structural heart disease.**
3. **Athletes who require anticoagulation should not participate in sports with danger of bodily collision (13).**
4. **Athletes without structural heart disease who have elimination of atrial fibrillation by an ablation technique, including surgery, may participate in all competitive sports after four to six weeks without a recurrence or after an electrophysiologic study has confirmed non-inducibility.**

Sinus node re-entry, inappropriate sinus tachycardia, and atrial tachycardia (in the absence of WPW syndrome). Sinus node re-entry, inappropriate sinus tachycardia, and atrial tachycardia should be evaluated as described for atrial flutter. Asymptomatic athletes who have episodes of tachycardia of 5 to 10 s that do not increase in duration during exercise can participate in all sports.

Recommendations:

1. **Athletes with sinus node re-entry, inappropriate sinus tachycardia, or atrial tachycardia in the absence of structural heart disease who maintain a ventricular rate that increases and slows appropriately and is comparable to that of a normal sinus response in relation to the level of activity, with or without therapy, can participate in all competitive sports.**
2. **Athletes with underlying structural heart disease can participate only in competitive sports consistent with the limitations of the heart disease.**
3. **Athletes without structural heart disease who have elimination of the atrial tachyarrhythmia by an ablation technique, including surgery, may participate in all competitive sports after two to four weeks without a recurrence or in several days after an electrophysiologic study showing non-inducibility.**

AV junctional escape beats/rhythm. Atrioventricular junctional escape beats and junctional rhythm are common in athletes. The clinical approach and final recommendations are the same as those given earlier for symptomatic athletes with disturbances of sinus node function.

Premature AV junctional complexes. If the athlete is asymptomatic except for occasional episodes of palpitations that do not suggest a sustained tachycardia, evaluation need include only a 12-lead ECG. In some athletes, a 24-h ECG recording (during athletic activity if possible), echocardiogram, and an exercise test may be indicated.

Recommendations:

1. **Athletes with a structurally normal heart and a normal heart rate response to activity without evidence of a sustained tachycardia can participate in all competitive sports.**
2. **Athletes with an abnormal heart, depending on the type and extent of the heart disease, can participate in competitive sports consistent with the limitations of the structural cardiac disease.**

Non-paroxysmal AV junctional tachycardia. A form of junctional tachycardia, so-called junctional ectopic tachycardia, can be found in a permanent form in infants less than six months old, whereas a transient form occurs mostly in children but occasionally in adults (13). Most adults have a

JACC Vol. 45, No. 8, 2005
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slower form of non-paroxysmal junctional tachycardia. Evaluation generally includes a 12-lead ECG, echocardiogram, exercise test, and 24-h ECG recording during activity. Invasive studies may be necessary for some symptomatic patients or for those with very rapid ventricular rates.

Recommendations:

1. **Athletes without structural heart disease or symptoms who have a controlled ventricular rate that increases and slows appropriately and is comparable to that of a normal sinus response in relation to the level of activity, with or without therapy, can participate in all sports.**
2. **Athletes who have no symptoms but who have structural heart disease or incompletely controlled ventricular rates can engage in class IA competitive sports depending on the nature and extent of the structural heart disease and the ventricular rate.**
3. **Athletes with inappropriately rapid ventricular rates, with or without structural heart disease, should be considered for treatment to control the ventricular rate before participating in any sports. Athletes whose tachycardia is controlled by therapy and verified by appropriate testing can participate in all competitive sports consistent with their cardiac status.**

Supraventricular tachycardia. Atrioventricular nodal reentrant tachycardia and AV re-entry over a concealed accessory pathway (with only retrograde conduction) (4,14) are included in this category. If the diagnosis of the supraventricular tachycardia cannot be made with certainty, and if other clinical therapeutic circumstances warrant it, invasive electrophysiologic studies might be indicated. It is important to identify the rate response of the supraventricular tachycardia during exercise. If the exercise does not induce the tachycardia, attempts to induce the supraventricular tachycardia (possibly with atrial or esophageal pacing) may be useful, followed once again by an exercise test performed by the athlete when the supraventricular tachycardia has been initiated. The logistics to accomplish this may be difficult. Asymptomatic athletes who have episodes of supraventricular tachycardia of 5 to 10 s that do not increase in duration during exercise can participate in all sports.

Recommendations:

1. **Athletes without structural heart disease who are asymptomatic and have reproducible exercise-induced supraventricular tachycardia prevented by therapy and verified by appropriate testing can participate in all competitive sports.**
2. **Athletes who do not have exercise-induced supraventricular tachycardia but experience sporadic recurrences should be treated. However, because of the unpredictable nature of the tachycardia, end points for adequate therapy may be difficult to achieve; but once established, these athletes can participate in all activities consistent with their cardiac status. Asymptomatic athletes who have episodes of supraventricular tachycardia of 5 to 15 s that do not increase in duration during exercise can participate in all sports consistent with their cardiac status.**
3. **Athletes with syncope, near-syncope, or significant symptoms secondary to arrhythmia or who have significant structural heart disease in addition to the arrhythmia should not participate in any competitive sports until they have been adequately treated and have no recurrence for two to four weeks (4). At that time they can participate in class IA competitive sports.**
4. **For those athletes with no structural heart disease who have had successful catheter or surgical ablation, are asymptomatic, and have no inducible arrhythmia on follow-up electrophysiologic testing, all competitive sports are permitted in several days. If no electrophysiologic testing is done, full participation is permitted if no spontaneous recurrence of tachycardia for two to four weeks after ablation.**

Ventricular pre-excitation (WPW syndrome). Required noninvasive tests include a 12-lead ECG, exercise test, and echocardiogram to exclude associated cardiovascular abnormalities. In some instances, a 24-h ECG recording during athletic activity may be indicated. Electrophysiologic studies are indicated in athletes with symptoms of impaired consciousness, long-lasting palpitations, or rapid rates in whom an ablation procedure is indicated.

In asymptomatic athletes with no history of palpitations or tachycardia and no evidence of structural cardiac abnormalities, further evaluation may not be necessary. However, the optimal management for these athletes is uncertain, and continues to be debated (15,16). Furthermore, the younger the patient, the less time the patient has had to develop symptoms, and so the distinction between symptomatic and asymptomatic WPW syndrome may be less meaningful in the pediatric population. Sudden death in athletes with pre-excitation is rare, and it appears to be confined largely to those with accessory pathways that have short refractory periods. Therefore, it may be advisable in selected asymptomatic athletes who anticipate moderate or high level activity to undergo electrophysiology study to determine the anterograde refractory period of the accessory pathway, the minimum RR interval between pre-excited complexes in atrial fibrillation, and the number of accessory pathways. Individuals with multiple accessory pathways or ventricular rates exceeding 240 beats/min should be offered catheter ablation of the accessory pathway (17,18). For those athletes with a history of palpitations, syncope, or near syncope, it is mandatory to assess the functional capabilities and electrophysiologic properties of the accessory pathway.

Recommendations:

1. **Athletes without structural heart disease, without a history of palpitations, or without tachycardia (particularly those 20 to 25 years old or more) can participate in all competitive sports. However, in younger age groups, a more in-depth evaluation including an electrophysiologic study may be recommended before allowing participation in moderate- to high-intensity competitive sports.**
2. **Athletes with episodes of AV reciprocating tachycardia should be treated as previously recommended (see section on Supraventricular Tachycardia). However, it should be appreciated that they can develop atrial fibrillation with rapid ventricular rates. Electrical induction of atrial fibrillation to determine the shortest QRS interval between two complexes conducted over the accessory pathway during isoproterenol administration or exercise is recommended. Those athletes in whom the shortest cycle length is less than 250 ms should undergo ablation of the accessory pathway.**
3. **Athletes with episodes of atrial flutter/fibrillation and syncope or near syncope whose maximal ventricular rate at rest (without therapy) as a result of conduction over the accessory pathway exceeding 240 beats/min should be considered for catheter ablation therapy of the accessory pathway prior to continuing competition. Those whose ventricular rate during isoproterenol administration is less than 240 beats/min and who have no episodes of syncope or near syncope appear to be at low risk for sudden cardiac death.**
4. **Athletes with no structural heart disease who have had successful catheter or surgical ablation of the accessory pathway, are asymptomatic, and have normal AV conduction and no inducible arrhythmia by follow-up electrophysiologic study can participate in all competitive sports in several days. Those without an electrophysiologic study and no spontaneous recurrence of tachycardia for two to four weeks after ablation can participate in all competitive sports.**

Premature ventricular complexes. Noninvasive tests recommended include a 12-lead ECG and exercise test. If there is evidence to suggest the presence of structural heart disease, an echocardiogram is indicated, and a 24-h ECG recording may be beneficial. Even without evidence of structural heart disease, if an increase in the number of premature ventricular complexes or complex ventricular arrhythmias occurs during exercise (with or without treatment, or substantial reduction or abolition following a period of deconditioning), further evaluation may be indicated. In some of these athletes thought to have a structurally normal heart, cardiac catheterization and angiography may reveal otherwise undetected abnormalities, including occult CAD, congenital coronary anomalies, ARVC, cardiac tumor, or evidence of cardiomyopathy. The more recently recognized channelopathy known as CPVT should be considered (see the following text).

Frequent and complex ventricular tachyarrhythmias are common in trained athletes; they are usually unassociated with underlying cardiovascular abnormalities and do not appear to convey increased risk (18). Deconditioning usually results in loss or diminution of these arrhythmias, providing evidence of their benign clinical nature (19).

Recommendations:

1. **Athletes *without* structural heart disease who have premature ventricular complexes at rest and during exercise, and exercise testing (comparable to the sport in which they compete) can participate in all competitive sports. Should the premature ventricular complexes increase in frequency during exercise or exercise testing to the extent that they produce symptoms of impaired consciousness, significant fatigue, or dyspnea, the athlete can participate in class IA competitive sports only.**
2. **Athletes *with* structural heart disease who are in high-risk groups and have premature ventricular complexes (with or without treatment) can participate in class IA competitive sports only. Such athletes with premature ventricular complexes that are suppressed by drug therapy (as assessed by ambulatory ECG recordings) during participation in the sport can compete in only class IA competitive sports.**

Ventricular tachycardia (VT). Nonsustained or sustained monomorphic or polymorphic VT is always a potentially serious occurrence. Noninvasive tests to be performed include a 12-lead ECG, exercise test, and echocardiography. In some patients, 24-h ECG recording during exercise may be indicated. Echocardiography, cardiac catheterization, and electrophysiologic study should be considered to verify that the heart is structurally normal and to establish the mechanism or location, or both, of the VT. A possible exception is the patient with accelerated idioventricular rhythm, in which the ventricular rate is similar to the sinus rate. In such patients, if they have no significant structural heart disease, the approach should be similar to that in individuals with premature ventricular complexes.

Recommendations:

1. **Athletes with a structurally normal heart and monomorphic nonsustained or sustained VT that can be localized to a specific site(s) in the heart are candidates for a catheter ablation procedure that may potentially offer a cure. Following such a successful ablation procedure, with subsequent failure to induce VT during electrophysiologic study (EPS) with/without isoproterenol when the VT was reproducibly induced before ablation, the athlete can resume full**

competitive activity within two to four weeks. A more conservative approach is recommended for the athlete who chooses drug suppression because catecholamines released during athletic activity can counter the suppressive effects of the drug, and the VT can re-emerge. In that situation, generally the athlete should not compete in any sports for at least two to three months after the last VT episode. If there have been no clinical recurrences, and the VT is not inducible by exercise/exercise testing or EPS, and the athlete has no structural heart disease, all competitive sports may be permitted. Because deconditioning can result in the loss or lessening of ventricular arrhythmias (19), a short period of deconditioning and retesting can be considered in some athletes.

2. **For the athlete with structural heart disease and VT, moderate- and high-intensity competition is contraindicated regardless of whether the VT is suppressed or ablated. Only class IA competitive sports are permitted.**
3. **An exception to this general recommendation is the asymptomatic athlete with brief (generally less than 8 to 10 consecutive ventricular beats) episodes of nonsustained monomorphic VT, rates generally less than 150 beats/min, and no structural heart disease established by noninvasive and invasive tests. These athletes do not appear to be at increased risk for sudden cardiac death. If exercise testing (preferably by ambulatory ECG recording during the specific competitive activity) demonstrates suppression of the VT or no significant worsening compared with baseline, participation in all competitive sports is permissible.**
4. **The desire of the athlete to continue athletic competition should not represent the primary indication for use of an implantable cardioverter-defibrillator (ICD). The efficacy with which these devices will terminate a potentially lethal arrhythmia under the extreme conditions of competitive sports, with the associated metabolic and autonomic changes, and possible myocardial ischemia, is unknown. In addition, sports with physical contact may result in damage to the ICD and/or lead, preventing normal function. For athletes with ICDs, all moderate and high intensity sports are contraindicated. Class IA sports are permitted.**

Ventricular flutter and ventricular fibrillation.

Recommendation:

1. **Athletes with conditions that result in cardiac arrest in the presence or absence of structural heart disease generally are treated with an ICD and cannot participate in any moderate- or high-intensity competitive sports. However, athletes with ICDs and who have had no episodes of ventricular flutter or ventricular fibrillation requiring device therapy for six months may engage in class IA competitive sports. Recommendations in the section on VT also apply.**

First-degree AV block. In asymptomatic athletes with structurally normal hearts, if the QRS complex is normal, no further evaluation other than a 12-lead ECG is necessary. If the QRS complex is abnormal, or the PR interval is excessively prolonged (0.3 s or more), an exercise stress test, 24-h ECG recording and echocardiogram may be indicated. Possibly an EPS might be necessary to determine the site and duration of conduction delay.

Recommendation:

1. **Asymptomatic athletes without evidence of structural heart disease, in whom the first-degree AV block does not worsen with exercise, can participate in all competitive sports. If underlying heart disease is present, its nature and severity can independently dictate alternative restrictions.**

Type 1 second-degree (Wenckebach) AV block. Wenckebach AV nodal block can be present in otherwise normal, well-trained endurance athletes (4). Recommended evaluations include a 12-lead ECG, exercise test, and echocardiogram. A 24-h ECG recording during athletic activity may be indicated in some athletes. In those athletes with type 1 second-degree AV block and co-existing bundle-branch block, EPS may be indicated to identify the presence of His-Purkinje Wenckebach block.

Recommendations:

1. **Asymptomatic athletes with a structurally normal heart and no worsening or actual improvement of AV block with exercise or recovery can participate in all competitive sports.**
2. **Asymptomatic athletes with a structurally abnormal heart in whom AV block disappears or does not worsen with exercise or recovery can participate in all competitive sports, as determined by the limitations of the cardiac abnormality.**
3. **Asymptomatic athletes in whom type 1 second-degree AV block initially appears or worsens with exercise or during the recovery period should be evaluated further (e.g., for possible intra- or infra-His block) and may require pacemaker therapy. Such athletes can participate in class IA competitive sports.**
4. **Athletes treated with pacemakers should not engage in competitive sports with a danger of bodily collision because such trauma may damage the pacemaker system.**

Type 2 second-degree (Mobitz) AV block. The evolution and treatment of this abnormality is considered to be the same as in acquired complete heart block. It should be treated with permanent pacing before any athletic activity. Athletes with pacemakers should not participate in compet-

itive sports that pose a danger of bodily collision because such trauma may damage the pacemaker system. Before allowing athletes to engage in these activities, an exercise test should be done at the level of activity demanded by the particular sport to be certain that the paced heart rate increases appropriately.

Congenital complete heart block. The clinical approach to evaluating the severity of the cardiovascular abnormality includes an echocardiogram, 12-lead ECG, 24-h ECG recording during exercise, and exercise stress test (exercise testing should be performed at the same exercise level as that during the sports activity).

Recommendations:

1. **Athletes with a structurally normal heart and normal cardiac function, with no history of syncope or near syncope, a narrow QRS complex, ventricular rates at rest greater than 40 to 50 beats/min increasing appropriately with exertion, no or only occasional premature ventricular complexes, and no VT during exertion can participate in all competitive sports.**
2. **Athletes with ventricular arrhythmia, symptoms of fatigue, near-syncope, or syncope should have a pacemaker implanted before they participate in competitive sports. Athletes with pacemakers should not participate in competitive sports when the danger of bodily collision exists because such trauma may damage the pacemaker system. Before allowing athletes to engage in these activities, an exercise test should be conducted at the level of activity demanded by the particular sport so as to be certain that the paced heart rate increases appropriately.**
3. **Athletes with abnormal hemodynamic status, as those with an intracardiac shunt, cannot participate in any competitive sports without a pacemaker. Restrictions are the same as those in recommendation 2.**

Acquired complete heart block.

Recommendations:

1. **Patients with acquired complete heart block should be treated with pacing before any athletic activity.**
2. **Athletes with a pacemaker should not participate in competitive sports posing a danger of bodily collision because such trauma may damage the pacemaker system.**

Complete right bundle-branch block. Evaluation includes a 12-lead ECG, exercise test, and echocardiogram. In some instances, a 24 h ECG recording may be indicated.

Recommendation:

1. **Athletes without ventricular arrhythmias who do not develop AV block with exercise and who have no symptoms can participate in all competitive sports consistent with their cardiac status. This also applies to athletes with associated left-axis deviation.**

Complete left bundle-branch block. Evaluation includes a 12-lead ECG, exercise test, and echocardiogram. In some instances, a 24-h ECG recording may be indicated. Because of the rarity of acquired left bundle-branch block in children and its association with syncope from presumed paroxysmal AV block, an invasive EPS should be considered in young patients.

Recommendations:

1. **Adult athletes with acquired left bundle-branch block should follow the recommendations under the section entitled Complete Right Bundle-Branch Block.**
2. **Athletes with a normal HV interval and a normal AV conduction response to pacing can participate in all competitive sports consistent with their cardiac status.**
3. **Athletes with abnormal AV conduction characterized by an HV interval greater than 90 ms or a His-Purkinje block should have pacemaker implantation. They should be restricted from competitive sports that hold a danger of bodily collision because such trauma may damage the pacemaker system.**

Inherited arrhythmia syndromes. LONG QT SYNDROME. The definitive clinical diagnosis of congenital long QT syndrome (LQTS) can be complex (20). Debate continues as to what QTc constitutes the upper limit of normal. An increasing proportion of asymptomatic individuals with genetically proven LQTS are found to have a normal resting ECG with a heart rate corrected QT interval (QTc) by Bazett's formula of less than 460 ms (genotype positive/phenotype negative LQTS). In addition, a QTc of 440 ms, used in the past as an upper limit of normal, is present in far too many normal individuals (greater than 25%) to serve as a meaningful upper limit cut-off value. In general, a QTc of 470 ms or more in males and 480 ms or more in females requires further investigation as to the presence of congenital (or acquired) causes of QT prolongation. A patient with LQTS and a resting QTc of 500 ms or more is generally considered at increased clinical risk for a significant arrhythmia (21). One approach to the diagnosis of congenital LQTS is to utilize the "Priori-Schwartz" score that incorporates QTc, T-wave morphology, symptomatic presentation, and family history into the diagnostic algorithm (21). A "Priori-Schwartz" score of 4 or more suggests high clinical probability for LQTS. In addition, genetic testing for the five cardiac ion-channel genes responsible for 75% of LQTS (LQT1, LQT2, LQT3, LQT5, and LQT6) is now available as a commercial diagnostic test (22).

Mutations involving the structural protein ankyrin-B underlie the rare LQT4 form (23). Mutations involving the *KCNJ2*-encoded IK1 potassium channel account for approximately one-half of Andersen-Tawil syndrome (ATS1)

characterized by abnormal U waves, a prolonged QU interval, periodic paralysis, and facial and skeletal dysmorphisms. The ATS1 has been annotated in the past as LQT7.

Physical exertion (particularly swimming) appears to be a common trigger for ventricular arrhythmias in LQT1, whereas individuals with LQT2 seem more at-risk to auditory/emotional triggers, and patients with LQT3 may be at greater risk during rest and inactivity (24,25). However, exceptions to these genotype-phenotype correlations hinder genotype-specific tailoring of competitive sports recommendations. The entire personal and family phenotype must be incorporated before any eligibility or disqualification decision is rendered.

Recommendations:

1. **Regardless of QTc or underlying genotype, all competitive sports, except those in class IA category should be restricted in a patient who has previously experienced either: 1) an out-of-hospital cardiac arrest, or 2) a suspected LQTS-precipitated syncopal episode.**
2. **Asymptomatic patients with baseline QT prolongation (QTc of 470 ms or more in males, 480 ms or more in females) should be restricted to class IA sports. The restriction limiting participation to class IA activities may be liberalized for the asymptomatic patient with genetically proven type 3 LQTS (LQT3).**
3. **Patients with genotype-positive/phenotype-negative LQTS (i.e., identification of a LQTS-associated mutation in an asymptomatic individual with a nondiagnostic QTc) may be allowed to participate in competitive sports. Although the risk of sudden cardiac death is not zero in such individuals, there is no compelling data available to justify precluding these individuals (who are being identified with increasing frequency) from competitive activities. Because of the strong association between swimming and LQT1, persons with genotype-positive/phenotype-negative LQT1 should refrain from competitive swimming.**
4. **LQTS patients with an ICD/pacemaker should not engage in sports with a danger of bodily collision because such trauma may damage the pacemaker system. The presence of an ICD should restrict individuals to class IA activities.**

SHORT QT SYNDROME. Individuals with the short QT syndrome (SQTS) (QTc less than 300 ms) have a short QT interval and ventricular refractory period, and at least some of them have "gain-of-function" abnormalities in either I_{Kr} (*KCNH2*) or I_{Ks} (*KCNQ1*) (26).

Recommendation:

1. **Until the phenotype of SQTS is better understood, a universal restriction from competitive sports with the possible exception of class IA activities seems to represent the most prudent recommendation (27).**

CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA (CPVT). Approximately one-half of patients with CPVT is pursuant to mutations involving the *RyR2*-encoded ryanodine receptor (sarcoplasmic reticulum calcium release channel). Such individuals are vulnerable to exercise-induced VT/ventricular fibrillation.

Recommendations:

1. **Symptomatic patients have a poor prognosis unless treated with an implantable cardioverter-defibrillator (ICD) (28) and all such patients are restricted from competitive sports with the possible exception of minimal contact, class IA activities. As with LQT1, patients with CPVT should be restricted from competitive swimming. Asymptomatic patients detected as part of familial screening with documented exercise- or isoproterenol-induced VT should refrain from all competitive sports except possible class IA activities. A less restrictive approach may be possible for the genotype-positive/phenotype-negative (asymptomatic, no inducible VT) athlete.**

BRUGADA SYNDROME (BrS). Brugada syndrome (29), characterized by an accentuated J-wave primarily in leads V_1 through V_3, with ST-segment elevation, often followed by a negative T-wave and an R prime, may be the cause of sudden unexplained death syndrome, typically during sleep. Only 15% to 20% of BrS is established as a channelopathy due to mutations involving the *SCN5A*-encoded alpha subunit of the cardiac sodium channel (30). Individuals with BrS and no previous cardiac arrests may have a high risk of sudden death if they have inducibility of ventricular arrhythmias and a previous history of syncope (28). Hyperthermia can potentially unmask the Brugada ECG pattern in patients with BrS, who can then display fever-induced polymorphic VT. Death often occurs with mild activity or during sleep.

Recommendations:

1. **Although a clear association between exercise and sudden death has not been established, and because of the potential impact of hyperthermia, restriction to participation in class IA sports seems advisable.**
2. **The presence of an ICD device warrants the same restrictions to class IA sports as previously outlined.**

doi:10.1016/j.jacc.2005.02.014

TASK FORCE 7 REFERENCES

1. Maron BJ. Sudden death in young athletes. N Engl J Med 2003;349:1064–75.
2. Zehender M, Meinertz T, Keul J, Just H. ECG variants and cardiac arrhythmias in athletes: clinical relevance and prognostic importance. Am Heart J 1990;119:1378–91.

3. Bjornstad H, Storstein L, Meen HD, Hals O. Ambulatory electrocardiographic findings in top athletes, athletic students and control subjects. Cardiology 1994;84:42–50.
4. Olgin JE, Zipes DP. Specific arrhythmias: diagnosis and treatment. In: Zipes DP, Libby P, Bonow RO, Braunwald E, editors. Heart Disease: A Textbook of Cardiovascular Medicine. Philadelphia, PA: Saunders, 2005:803–63.
5. Allessie M, Ausma J, Schotten U. Electrical, contractile and structural remodeling during atrial fibrillation. Cardiovasc Res 2002;54:230–46.
6. Antzelevitch C. Molecular genetics of arrhythmias and cardiovascular conditions associated with arrhythmias. J Cardiovasc Electrophysiol 2003;14:1259–72.
7. Calkins H, Zipes DP. Hypotension and syncope. In: Zipes DP, Libby P, Bonow RO, Braunwald E, editors. Heart Disease. A Textbook of Cardiovascular Medicine. Philadelphia, PA: Saunders, 2005:909–19.
8. Ackerman MJ, Khositseth A, Tester DJ, Hejlik JB, Shen WK, Porter CB. Epinephrine-induced QT interval prolongation: a gene-specific paradoxical response in congenital long QT syndrome. Mayo Clin Proc 2002;77:413–21.
9. Schott JJ, Alshinawi C, Kyndt F, et al. Cardiac conduction defects associated with mutations in SCN5A. Nat Genet 1999;23:20–1.
10. Benson DW, Wang DW, Dyment M, et al. Congenital sick sinus syndrome caused by recessive mutations in the cardiac sodium channel gene (SCN5A). J Clin Invest 2003;112:1019–28.
11. Nattel S, Erlich J. Atrial fibrillation. In: Zipes D, Jalife J, editors. Cardiac Electrophysiology: From Cell to Bedside. Philadelphia, PA: Saunders, 2004:512–22.
12. Furlanello F, Bertoldi A, Dallago M, et al. Atrial fibrillation in elite athletes. J Cardiovasc Electrophysiol 1998;9:S63–8.
13. Oral H, Strickberger SA. Junctional rhythms and junctional tachycardia. In: Zipes D, Jalife J, editors. Cardiac Electrophysiology: From Cell to Bedside. Philadelphia, PA: Saunders, 2004:523–7.
14. Lockwood D, Otomoto K, Wang Z. Electrophysiologic characteristics of atrioventricular nodal reentrant tachycardia. In: Zipes D, Jalife J, editors. Cardiac Electrophysiology: From Cell to Bedside. Philadelphia, PA: Saunders, 2004:537–57.
15. Pappone C, Santinelli V, Rosanio S, et al. Usefulness of invasive electrophysiologic testing to stratify the risk of arrhythmic events in asymptomatic patients with Wolff-Parkinson-White pattern: results from a large prospective long-term follow-up study. J Am Coll Cardiol 2003;41:239–44.
16. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. N Engl J Med 1979;301:1080–5.
17. Pappone C, Santinelli V, Manguso F, et al. A randomized study of prophylactic catheter ablation in asymptomatic patients with the Wolff-Parkinson-White syndrome. N Engl J Med 2003;349:1803–11.
18. Biffi A, Pelliccia A, Verdile L, et al. Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes. J Am Coll Cardiol 2002;40:446–52.
19. Biffi A, Maron BJ, Verdile L, et al. Impact of physical deconditioning on ventricular tachyarrhythmias in trained athletes. J Am Coll Cardiol 2004;44:1053–8.
20. Ackerman MJ. Cardiac channelopathies: it's in the genes. Nat Med 2004;10:463–4.
21. Priori SG, Schwartz PJ, Napolitano C, et al. Risk stratification in the long-QT syndrome. N Engl J Med 2003;348:1866–74.
22. Genaissance Pharmaceuticals, Inc. Genaissance Pharmaceuticals, Inc. (GNSC) launches its proprietary FAMILION test for genetic mutations associated with sudden cardiac death. Available at: http://www.biospace.com/news_story.cfm?StoryID=16229920&full=1. Accessed October 1, 2004.
23. Mohler PJ, Schott JJ, Gramolini AO, et al. Ankyrin-B mutation causes type 4 long-QT cardiac arrhythmia and sudden cardiac death. Nature 2003;421:634–9.
24. Schwartz PJ, Priori SG, Spazzolini C, et al. Genotype-phenotype correlation in the long-QT syndrome: gene-specific triggers for life-threatening arrhythmias. Circulation 2001;103:89–95.
25. Choi G, Kopplin LJ, Tester DJ, Will ML, Haaglund CM, Ackerman MJ. Spectrum and frequency of cardiac channel defects implicated in swimming-triggered arrhythmia syndromes. Circulation 2004;110:2119–24.
26. Brugada R, Hong K, Dumaine R, et al. Sudden death associated with short-QT syndrome linked to mutations in HERG. Circulation 2004;109:30–5.
27. Gaita F, Giustetto C, Bianchi F, et al. Short QT syndrome: a familial cause of sudden death. Circulation 2003;108:965–70.
28. Sumitomo N, Harada K, Nagashima M, et al. Catecholaminergic polymorphic ventricular tachycardia: electrocardiographic characteristics and optimal therapeutic strategies to prevent sudden death. Heart 2003;89:66–70.
29. Brugada J, Brugada R, Brugada P. Determinants of sudden cardiac death in individuals with the electrocardiographic pattern of Brugada syndrome and no previous cardiac arrest. Circulation 2003;108:3092–6.
30. Brugada P, Brugada R, Mont L, Rivero M, Geelen P, Brugada J. Natural history of Brugada syndrome: the prognostic value of programmed electrical stimulation of the heart. J Cardiovasc Electrophysiol 2003;14:455–7.

Appendix 1. Author Relationships With Industry and Others

Name	Consultant	Research Grant	Scientific Advisory Board	Stock Holder	Expert Witness Testimony
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Dr. Augustus O. Grant	None	None	None	None	None
Dr. Robert J. Myerburg	• Guidant • Procter & Gamble	None	• Procter & Gamble • Reliant Pharmaceutical	None	• 2000, Defense, Lewis vs. Mudge • 2002, Defense, Weiner vs. Vitello • 2005, Defense, Ephedra Multi-District Litigation
Dr. George Van Hare	None	• Medtronic • St. Jude Medical	None	None	None
Dr. Douglas P. Zipes	• Cardiofocus • Janssen • Medtronic	• Medtronic	• Medtronic	• MVMD	• 1996, Defense, Knapp vs. Northwestern