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NASPE CONSENSUS STATEMENT

# Report of the NASPE Policy Conference on Arrhythmias and the Athlete

N.A. MARK ESTES III, M.D.,[1] MARK S. LINK, M.D.,[1] DAVID CANNOM, M.D.,[2] GERALD V. NACCARELLI, M.D.,[3] ERIC N. PRYSTOWSKY, M.D.,[4,5] BARRY J. MARON, M.D.,[6] and BRIAN OLSHANSKY, M.D.[7]

From the [1]New England Medical Center Hospitals, Tufts University School of Medicine, Boston, Massachusetts; [2]Good Samaritan Hospital, Los Angeles, California; [3]Pennsylvania State University, Hershey, Pennsylvania; [4]Duke University Medical Center, Durham, North Carolina; [5]St. Vincent's Hospital, Indianapolis, Indianapolis; [6]Minneapolis Heart Institute Foundation, Minneapolis, Minnesota; and [7]Loyola University, Chicago, Illinois

**Arrhythmias and the Athlete.** ***Introduction:*** **This consensus statement summarizes the proceedings of The Expert Consensus Conference on Arrhythmias in the Athlete of the North American Society of Pacing and Electrophysiology (NASPE) on detecting, evaluating, and treating athletes with cardiovascular disorders that predispose to cardiac arrhythmias.**

***Methods and Results:*** **The participants in the open policy conference were selected by the codirectors (Drs. Estes and Olshansky) based on expertise and contributions to the literature. All participants provided a referenced summary of their presentation. The writing group used the information from all published scientific studies, clinical trials, registries, clinical experience, and expert opinion to make recommendations regarding screening, evaluation, management, eligibility for competition, and a range of other medical, social, and legal issues regarding the recreational and competitive athlete. The codirectors of the symposium synthesized the participants' reports for this and made revisions according to suggestions of all members of the writing committee. The manuscript was reviewed by four independent reviewers assigned by the NASPE Committee for the Development of Position Statements and NASPE Board of Trustees.**

***Conclusion:*** **Despite considerable advances in knowledge regarding the diagnosis, therapy, and mechanisms of arrhythmias in the athlete, much remains unknown. Continued basic, clinical, and epidemiologic research is needed. Current screening techniques to detect athletes lack sensitivity and specificity. Evaluation of standardized screening programs with tracking of long-term outcomes is needed. Officials from athletic, academic, medical, and legal institutions need to form strategic partnerships to develop policy related to assessment of risk and assumption of responsibility for athletic activities.** *(J Cardiovasc Electrophysiol, Vol. 12, pp. 1208-1219, October 2001)*

*arrhythmia, sudden death, syncope, athletics*

## Introduction

Evaluation and management of athletes with symptoms of or a clinically documented cardiac arrhythmia remain a challenge for the physician. At once there is the risk of not diagnosing an important cardiovascular condition that may predispose to a serious or life-threatening arrhythmia and the risk of unnecessary restriction of the athlete with a more benign condition. Evaluation of the athlete with a cardiac arrhythmia often is confounded by the complex psychological, social, and even economic ramifications of the diagnosis and treatment. Based on a number of high-profile deaths in athletes, the public and medical interest in medical, ethical, and legal issues related to cardiac arrhythmias and sudden cardiac death in the athlete has intensified. Guidelines for screening all competitive high school and collegiate athletes have been proposed as a method to identify individuals with underlying structural heart disease who may be at risk for life-threatening cardiac arrhythmias.[1,2] The Expert Consensus Conference on Arrhythmias in the Athlete held in conjunction with the 19th Annual Scientific Session of The North American Society of Pacing and Electrophysiology (NASPE) focused on a broad range of medical, legal, and ethical issues related to screening for, evaluating, and managing arrhythmias in the athlete. The impetus for this conference came from the recent advances in diagnosis, therapy, and understanding of the mechanisms and cardiovascular disorders associated with arrhythmias in athletes.[3-15]

Although this document focuses on the competitive athlete, which is defined as one who participates in an organized team or individual sport requiring systematic training and regular competition against others while placing a high premium on athletic excellence and achievement, many of the recommendations also are relevant to the recreational athlete and nonathlete.[16] When a definitive cardiovascular

Address for correspondence: N.A. Mark Estes III, M.D., Tufts University School of Medicine, Box 197, 750 Washington Street, Boston, MA 02111. Fax: 617-626-4586; E-mail: nestes@lifespan.org

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**TABLE 1**
ECG Alterations in Response to Physiologic Adaptations in the Athlete

| |
|---|
| Sinus pause |
| Sinus arrhythmia |
| Sinus bradycardia |
| Junctional rhythm |
| Atrial extrasystole |
| Ventricular extrasystole |
| PR prolongation (first-degree AV block) |
| Wenckebach phenomenon |
| Advanced AV block |
| Voltage criteria for left ventricular hypertrophy |
| Voltage criteria for right ventricular hypertrophy |
| J point elevation |
| ST segment evaluation |
| T wave abnormalities |

diagnosis is made, the consensus panel recommendations of the 26th Bethesda Conference provide guidelines for continued participation or disqualification from athletics.[16] When relevant, these guidelines, which focus on eligibility recommendations for competitive athletes with cardiovascular abnormalities, will be reviewed in this consensus statement. An objective of this NASPE conference was to review the available data regarding arrhythmias in the athlete and develop a consensus statement reporting the proceedings of the conference. Few data are available that have been obtained prospectively from rigorously designed scientific studies on arrhythmias or cardiovascular conditions that predispose to arrhythmias in the athlete.[16,17] This report uses the best available information including that from well-designed clinical trials, as well as registry data, clinical experience, and expert opinion, to form the basis for recommendations regarding evaluation and management of the athlete with cardiac arrhythmias and conditions that predispose to arrhythmias.

## Bradyarrhythmias

In the conditioned athlete, there is a broad spectrum of bradyarrhythmias and other ECG alterations due to the resting heightened vagal tone and withdrawal of sympathetic tone that accompanies physical conditioning[3,16-19] (Table 1). In the absence of symptoms due to sinus bradycardia, evaluation is not generally warranted. Extreme bradycardia with resting rates <30 beats/min or sinus pauses >3 seconds is seen most commonly in endurance athletes, such as long-distance runners, bicyclists, and long-distance swimmers. Symptomatic or extreme bradyarrhythmias can be evaluated with a history, physical examination, ECG, 24-hour ambulatory monitoring, and exercise tolerance test. When there is any indication of underlying structural heart disease based on history, examination, or ECG, an echocardiogram should be considered. In those without symptoms and without structural heart disease, there is no need to restrict athletic activity.[3,16-19] In the presence of structural heart disease, restriction based on the type of heart disease is recommended.[16] Rarely, it becomes necessary to limit training because of symptomatic resting bradycardias in well-conditioned athletes. In those athletes with or without structural heart disease, all competitive sports are allowed if heart rate increases appropriately with exercise. An exercise stress test or Holter monitor during exercise can be used for this evaluation. In individuals with symptoms such as syncope or presyncope, participation in athletics should be restricted unless there is a period of 3 to 6 months of definitive therapy for the arrhythmia.[3,16] When pacemaker therapy is required, restriction from contact sports is recommended.[3,16]

In some athletes, increased vagal tone contributes to first-degree AV block or Mobitz type I AV block (Wenckebach). In the absence of symptoms, worsening of AV block with exercise, or underlying structural heart disease, there is no need for therapy or restriction from athletic activity.[3,16] Uncommonly, Mobitz type II or complete heart block (third-degree heart block) is seen in the athlete.[3,16,18,19] For such individuals, permanent pacing is recommended if they have underlying heart disease or symptoms. For athletes without structural heart disease or congenital complete heart block, no symptoms, and resting rates >40 beats/min without ventricular arrhythmias during exercise, no restrictions in exercise are recommended.[3,16,17] Athletes treated with a permanent pacemaker are restricted to athletics having no danger of bodily collision.[3,16,17]

## Supraventricular Arrhythmias

Supraventricular arrhythmias in the athlete frequently are benign and may not even require therapy. However, they occasionally may be associated with more severe symptoms or cause hemodynamic collapse and require definitive treatment (Table 2). Premature atrial contractions may be symptomatic and detected by the athlete, or they may be detected by the physician. A careful history, physical examination, and 12-lead ECG are recommended.[3,16,17] In the absence of clinically important symptoms and structural heart disease, additional evaluation and therapy are not recommended. However, when more severe symptoms are present, such as frequent or severe palpitations or light-headedness, beta-blocker therapy could be considered. In the absence of any significant cardiac disease, athletes with premature atrial contractions are not restricted from any athletic competition.[3,16,17]

In the athlete, sustained supraventricular tachycardia is most commonly due to AV nodal reentry. Most authorities agree that the appropriate evaluation includes a history, physical examination, and invasive electrophysiologic evaluation to define the tachycardia mechanism. With success rates >95% in experienced laboratories and serious complication rates <1%, many consider a curative approach with radiofrequency ablation to be the preferred initial approach.[3,16,20,21] For individuals who elect for pharmacologic therapy, drug therapy should be proven effective for a period of 6 months before resuming competitive athletics based on the recommendations of the Bethesda Conference.[3,16] However, for selected patients at low risk for arrhythmia occurrence or severe symptoms, it may be appropriate to allow resumption of athletics earlier than 6 months. It would be reasonable to perform a stress test on individuals undergoing pharmacologic therapy to assess for arrhythmia recurrence, although no prospective data are available regarding the predictive value of this approach. For athletes undergoing successful catheter ablation, the Bethesda Conference guidelines allow resumption of all competitive athletics after 3 months.[3,16] However, for individuals at low risk for arrhythmia recurrence or severe symptoms, many experts would allow resumption of ath-

**TABLE 2**
Supraventricular Arrhythmias in the Athlete*

| Arrhythmia | Baseline ECG | Symptoms | Diagnosis | Treatment Options | Guidelines for Athletic Participation[17] |
|---|---|---|---|---|---|
| APCs | Often WNL | Palpitations | Monitor | Reassurance | No restrictions; beta-blocker if highly symptomatic |
| Atrial fibrillation | Often WNL | Palpitations | Monitor | Antiarrhythmics, anticoagulation, and rate control | Bodily contact prohibited with warfarin |
| Atrial flutter | Often WNL | Palpitations | Monitor | RFA, antiarrhythmics, rate control, and anticoagulation | Bodily contact prohibited with warfarin |
| Ventricular preexcitation (WPW) | Short PR, delta waves | Asymptomatic | Monitor, ECG, EPS | No therapy, RFA if high risk | Consider EPS to risk stratify |
| Ventricular preexcitation (WPW) | Short PR, delta waves | Palpitations | Monitor, ECG | RFA, antiarrhythmias | No restrictions after 3–6 months without symptoms |
| AVNRT | Normal | Palpitations | Monitor, EPS | RFA, antiarrhythmics | No restrictions after 3–6 months without symptoms |

*Modified from reference 3.
APC = atrial premature contractions; AVNRT = AV nodal reentrant tachycardia within normal limits; EPS = electrophysiologic study; RFA = radiofrequency ablation; WNL = within normal limits; WPW = Wolff-Parkinson-White syndrome.

letic activity earlier than 3 months. Routine follow-up electrophysiologic evaluation after institution of medical therapy or radiofrequency ablation is not warranted. However, in selected individuals with severe symptoms or those participating in high-risk sports where an arrhythmia recurrence may put them at risk (e.g., downhill skiing, automobile racing, swimming, biking, horseback riding), repeat electrophysiologic evaluation before participation in athletics may be warranted. For individuals with more severe symptoms related to the arrhythmia or for athletes involved in high-intensity competitive athletics who want to resume sports, catheter ablation technique would be preferred.[3,16]

Wolff-Parkinson-White (WPW) syndrome is found in approximately 3 of 1,000 individuals.[22-24] Evaluation of the athlete with asymptomatic ventricular preexcitation or WPW pattern on ECG remains controversial. Some experts recommending observation without restriction of athletic participation because the risk of sudden death is very low,[3,16,20-24] but many experts recommend electrophysiologic evaluation to define the properties and location of the bypass tract. If the bypass tract allows conduction to the ventricle at rate >240 beats/min, consideration should be given to radiofrequency ablation to eliminate the risk of future life-threatening arrhythmias.[3,16,20-24]

The athlete with symptomatic arrhythmias due to WPW may have AV reciprocating tachycardia, atrial fibrillation, or rarely ventricular fibrillation. A complete history and physical examination should be supplemented by an ECG, 24-hour ambulatory monitor, exercise testing, and echocardiogram to exclude any underlying structural heart disease.[3,16,20-24] In some individuals, manifest preexcitation is not present on the surface ECG, but a retrograde bypass tract is identified at the time of electrophysiologic evaluation. This "concealed" bypass tract allows retrograde conduction from the ventricle to the atrium during the tachycardia. With success rates >95% and very low rates of significant complications, cure with radiofrequency ablation generally is considered the preferred approach for manifest or concealed bypass tract.[3,16,20-24] Resumption of all athletic activities at 3 months is recommended with this approach. Many experts allow return to athletics in a period of a few weeks after successful ablation in patients at low risk for arrhythmia recurrence or severe symptoms. In contrast, when drug therapy is used, a period of 6 months without arrhythmia recurrence is recommended before resumption of athletics.[3,16] Recommendations for the resumption of athletic activities earlier than 3 months and the need for follow-up electrophysiologic evaluation are similar for WPW and AV node reentry.

Atrial fibrillation or atrial flutter in the athlete may be more common compared with an age-matched population of nonathletes.[3,25-29] Some experts believe that athletes are predisposed to atrial fibrillation due to the high vagal tone[25,29] that is present in many. For athletes with atrial fibrillation or flutter, a history, physical examination, ECG, echocardiogram, and thyroid function tests should be completed. The maximal exertional rate of the atrial fibrillation or flutter should be assessed with an exercise tolerance test, and an ambulatory monitor is warranted to assess maximal and minimal rates and the presence of any ventricular arrhythmias. The options for therapy include rhythm control by reestablishing and maintaining normal sinus rhythm or rate control during atrial fibrillation.[27] The strategy of rhythm control has the advantage of avoiding anticoagulation, but the potential disadvantage of side effects and cost of the antiarrhythmic drug therapy.[27] If the athlete has structural heart disease or other risk factors for embolic events, anticoagulation may be needed, which would preclude participation in any sport with a risk of bodily collision.[3,16] Control of the rate of ventricular response can be difficult in the athlete. In some competitive athletics, beta-blockers are prohibited.[3] For athletes with or without structural heart disease with atrial fibrillation who maintain a ventricular response during physical activity comparable to sinus rate with or without therapy, athletic participation is allowed as appropriate for their anticoagulation status and the limitations of their structural heart disease.[3,16] Currently, catheter-based approaches with radiofrequency ablation offer the potential for cure of atrial flutter, atrial tachycardia, and select forms of focal atrial fibrillation.[26] When successful, athletic participation could be allowed after 3 months without arrhythmia recurrence.[3,16]

**TABLE 3**
Ventricular Arrhythmias In the Athlete*

| Arrhythmia | Baseline ECG | Symptoms | Diagnosis | Treatment Options | Guidelines for Athletic Participation[17] |
|---|---|---|---|---|---|
| PVCs | WNL | Palpitations | Monitor | Reassurance, beta-blockers | No restrictions if no SHD |
| NSVT | WNL | Palpitations | Monitor | Assess for SHD; if no SHD, reassurance | No restriction if no SHD |
| Sustained VT/VF | WNL or SHD | Palpitations, syncope, cardiac arrest | Monitor, EPS | If SHD, further evaluation; RFA if no SHD; ICD or AAD if SHD | No restrictions if no SHD and successful RFA; low-intensity sports otherwise |

*Modified from reference 3.
EPS = electrophysiologic study; NSVT = nonsustained ventricular tachycardia; PVCs = premature ventricular contractions; RFA = radiofrequency ablation; SHD = structural heart disease; VF = ventricular fibrillation; VT = ventricular tachycardia; WNL = within normal limits.

## Ventricular Arrhythmias

In evaluating the athlete with ventricular arrhythmias, the presence of symptoms and structural heart disease is a critical factor in determining treatment and exercise restrictions (Tables 3 and 4). Premature ventricular contractions in the athlete are common and rarely cause symptoms sufficiently severe to warrant therapy. In the absence of any congenital or acquired structural heart diseases, there is no increased risk of life-threatening cardiac arrhythmias.[3,30] In athletes with premature ventricular contractions, the history, physical examination, and ECG should be supplemented by an echocardiogram. In the presence of congenital heart disease (such as long QT syndrome [LQTS], hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, or anomalous origin of the coronary arteries) or acquired heart disease (such as coronary artery disease or a dilated cardiomyopathy), further evaluation and therapy is warranted.[31-44] In the absence of structural heart disease, therapy with beta-blockers may decrease symptoms with or without a reduction in the frequency of premature ventricular contractions. However, beta-blockers are prohibited by many athletic organizations.[3] When not prohibited, they may impair performance. Drug therapy with other antiarrhythmic agents generally is not warranted unless the symptoms are severe and persist despite beta-blocker therapy. If the premature ventricular contractions do not cause symptoms with exertion or worsen with exercise, there are no athletic restrictions in the absence of structural heart disease.[3,16]

Nonsustained ventricular tachycardia in the absence of structural heart disease indicates no additional risk of sudden cardiac death.[3,16,32] Evaluation and management of these athletes is similar to that of athletes with premature ventricular contractions. However, athletes with nonsustained polymorphic ventricular tachycardia may be at higher risk for life-threatening ventricular arrhythmias, and therapy with beta blockers and athletic restriction should be considered.[33,34] Selected subsets of athletes with nonsustained polymorphic ventricular tachycardia may require more aggressive therapy, such as an implantable cardioverter defibrillator (ICD). However, there are not sufficient data to universally recommend such an approach.

Sustained ventricular tachycardia or prior episodes of ventricular fibrillation necessitate thorough evaluation of the athlete's cardiac status, with history, physical examination, ECG, echocardiogram, and selective use of the stress test, cardiac magnetic resonance imaging, cardiac catheterization, and electrophysiologic evaluation.[3,6,7,15,32,34] Sustained ventricular tachycardia originating from the right ventricular outflow tract or other regions of the right or left

**TABLE 4**
Cardiovascular Conditions Associated with Ventricular Arrhythmias

| Condition | Symptoms | ECG | VT Morphology | Treatment Options | Guidelines for Athletic Participation |
|---|---|---|---|---|---|
| HCM | Palpitations, syncope, SCD | LVH, pseudoinfarct pattern | PMVT/VF | BB, AAD, ICD, myomectomy | Low intensity only |
| ARVD | Palpitations, syncope, SCD | T wave inversion, epsilon wave | LBBB, inferior axis | AAD, ICD | Low intensity only |
| CAD | Palpitations, syncope, SCD | Infarction pattern, ischemic ST | RB, LB, VF | ICD, AAD | Low intensity only |
| IDCM | Palpitations, syncope, SCD | Often LBBB | RB or LB | Amiodarone, ICD | Low intensity only |
| LQTS | Palpitations, syncope, SCD | Long QTc | Torsades de pointes | BB, PPM, ICD | Low intensity only |
| Anomalous CAD | SCD | WNL | VF | CABG | No restrictions after CABG |
| Idiopathic LVT | Palpitations, LH, syncope | WNL | RB, left axis | RFA | No restrictions 3 months after RFA |
| Idiopathic RVOT | Palpitations, LH, syncope | WNL | LB, inf axis | RFA | No restrictions 3 months after RFA |

AAD = antiarrhythmic drugs; ARVD = arrhythmogenic right ventricular dysplasia; BB = beta blocker; CABG = coronary artery bypass surgery; CAD = coronary artery disease; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter-defibrillator; IDCM = idiopathic cardiomyopathy; LB = left bundle; LBBB = left bundle branch block; LH = lightheadedness; LQTS = long QT syndrome; LVH = left ventricular hypertrophy; LVT = left ventricular tachycardia; PMVT = polymorphic ventricular tachycardia; PPM = permanent pacemaker; RB = right bundle; RFA = radiofrequency ablation; RVOT = Right ventricular outflow tract; SCD = sudden cardiac death; VF = ventricular fibrillation; WNL = within normal limits.

ventricle (idiopathic ventricular tachycardia) in the absence of any identifiable structural heart disease is unusual but is important to document.[3] In this setting, programmed ventricular stimulation supplemented by isoproterenol infusion frequently can induce the arrhythmia, which would allow mapping and cure with radiofrequency ablation. Under such circumstances, these athletes can return to athletic competition in 3 months.[3,16]

Sustained ventricular tachycardia or ventricular fibrillation usually occurs in the presence of some underlying structural heart disease. In the athlete younger than 35 years, hypertrophic cardiomyopathy, right ventricular dysplasia, anomalous origin of the coronary artery, or other congenital abnormalities[3,6,7,15,34,37] most frequently are the underlying heart diseases that predispose to sudden cardiac death. In these athletes, a continued risk of arrhythmia recurrence may exist and justify antiarrhythmic therapy or an ICD. Most experts believe that the ICD confers superior protection for sudden death prevention in this patient population, but no prospective comparative data are available. In individuals older than 35 years, coronary artery disease is the most common cause of sudden cardiac death.[5,6,8,36] In athletes with underlying structural heart disease of any type, competitive sports generally are prohibited by current guidelines.[3,16,34] When an anomalous coronary artery is identified, competitive athletics can be resumed 6 months after definitive treatment with surgery.[3,16,34]

In many of the cardiovascular conditions known to predispose to sudden death, exercise is believed to exacerbate arrhythmias.[14,34,36,39] In both right ventricular outflow tract ventricular tachycardia and sustained monomorphic ventricular tachycardia associated with right ventricular dysplasia, the arrhythmias frequently are exercise induced.[39] With right ventricular outflow tract ventricular tachycardia (in the absence of structural heart disease), catheter-based cure frequently is possible.[43] Similarly, in patients with congenital abnormalities of the coronary arteries, exercise triggers the sudden death.[3,8,14,34,36] In idiopathic ventricular fibrillation, approximately 15% of individuals have their cardiac arrest while exercising.[3,14,34-36] In the most common form of underlying structural heart disease associated with sudden cardiac death, hypertrophic cardiomyopathy, the arrhythmias are induced by exertion in approximately one half of the individuals.[9,15] There is evidence that restriction of athletic activity in this athletic population is a successful strategy for prevention of sudden death.[31] In individuals with LQTS, sudden death generally is exertional or immediately postexertional.[34,38] In this condition, beta-blockers have been shown to decrease the frequency of syncope and sudden death. However, most experts believe that the ICD provides better protection against sudden cardiac death, particularly with syncope or ventricular arrhythmia recurrence while the patient is undergoing beta-blocker therapy.[3,16,34] Restriction from athletic activities is recommended even when effective therapy is identified.[3,16]

## Participation of Athletes with Life-Threatening Ventricular Arrhythmias and an ICD

Although ICDs have proven to be extremely effective for prevention of sudden cardiac death, their ability to terminate lethal arrhythmias under the conditions of vigorous physical activity associated with competitive athletics is unknown.[3,16,45] Many experts believe that the ICD should not be implanted for the explicit purpose of allowing continued participation in athletic activity for the individual at risk for life-threatening ventricular arrhythmias. However, this position is controversial because there are no prospective data on use of the ICD for this purpose. It is generally accepted that for individuals with an ICD, all moderate- and high-intensity sports are contraindicated.[3,16,45] Although there are no good data on the risk of athletic participation of athletes with ICDs, it is recommended that only low-intensity competitive sports, such as golf or bowling, which do not constitute a significant risk to the ICD, be allowed. These activities can begin 6 months after implant or after the last ventricular arrhythmia requiring intervention, including pacing, cardioversion, or defibrillation.[3,16] Nevertheless, patients should be advised that these activities may predispose to lead fracture or other complications. Therefore, moderation of the intensity of the activity is recommended.

## Commotio Cordis

Until recently, it has not been widely appreciated that nonpenetrating blunt trauma delivered without excessive force to the chest wall can lead to fatal cardiac arrhythmias, in the absence of preexisting heart disease or identifiable myocardial damage. The mechanism of commotio cordis, a cause of sudden cardiac death in the athlete resulting from an object striking the chest wall, recently has been elucidated.[46-49] With commotio cordis, an object (such as a baseball, hockey puck, or other firm object) results in instantaneous death in the absence of any acute injury or underlying heart disease at autopsy.[46-49] An experimental model of this condition recently demonstrated that a precordial blow at or near the peak of the T wave results in ventricular fibrillation.[46-49] Safety baseballs, which are softer than regulation, have been shown in this model to reduce the risks of ventricular fibrillation.[49] Given the proven decreased risk of head injury and the reduced risk of sudden death due to chest impact, age-appropriate safety baseballs should be advocated for use in youth sports. Whether chest wall protectors reduce the risk of sudden death is not yet clear; therefore, no recommendations regarding chest protectors can be given.

## Role of the Automatic External Defibrillator

The concept of public access to defibrillation has received more attention over the last several years as it has become clear that resuscitation of the cardiac arrest victim is most likely to be effective if a system is in place to ensure that cardiopulmonary resuscitation and early defibrillation are provided promptly.[50,51] This system of response both in the community and at athletic events includes prompt activation of the emergency medical services, bystander cardiopulmonary resuscitation, early defibrillation, and advanced cardiac life support. Over the last several years, development of the automatic external defibrillator (AED) has revolutionized the approach to out-of-hospital cardiac arrest. The available technology allows the application of pads to the arrest victim's chest wall, allowing analysis of the patient's cardiac rhythm and, if appropriate, delivery of defibrillation shocks. These devices contain both audio and visual prompts that direct the user through the process. These devices are small (approximately the size of a lap-top

computer), low maintenance, and fairly inexpensive, and they have a battery shelf life of several years. With a few hours of training, nonmedical or medical responders can be instructed on the use of the AED. This allows nontraditional responders, including police, firefighters, security personnel, team physicians, coaches, and trainers, to respond rapidly in emergency situations. With early availability of bystander defibrillation, the potential for a higher efficacy rate of resuscitation is greater than with shorter response times. Prior studies have documented that AEDs will assess the cardiac rhythm correctly and successfully defibrillate with an accuracy >95%.[52]

With respect to cardiac emergencies at athletic events, more than two thirds of the reported cardiac events occur at football or basketball games, and more than half of the events occur during training sessions or practice as opposed to competition.[50] It is evident that team athletic trainers or coaches are more likely to be present to respond immediately than a team physician. Approximately one third of cardiac arrests occur at athletic competitions, when a team physician is present. Based on analysis of the probability and frequency of cardiac arrests, it also is evident that emergencies may arise in some settings more frequently in spectators attending athletic events than in the athletes.[50]

The success of these programs for early-access defibrillation with the AED has been documented with multiple programs.[50] At the 1986 World's Fair, 18 million visitors attended over a 5-month period. There were 6 cardiac arrests, for a rate of 1 cardiac arrest per 3 million visitors. In instances in which ventricular fibrillation was the initial rhythm, AEDs were used successfully by trained security personnel to reestablish normal sinus rhythm.[50] Similar results have been reported at athletic events in the Kingdome in Seattle, Washington, and at the University of Washington Husky Stadium, where an arrest rate of 1 per 2 million attendees has been reported at athletic events.[50] Similar programs currently are in progress at Vanderbilt University, Nashville, Tennessee, and on most airlines.[50]

It is important that these programs using the AED be incorporated into a coordinated program of early response defibrillation with public bystander initiated cardiopulmonary resuscitation, use of the AED by trained personnel, and organized EMS response. Athletic organizations, educational institutions, coaches, trainers, sports medical physicians, and emergency response personnel should place the issue of incorporation of the AEDs into a coordinated response system with tracking of outcomes high on their agendas. More data on the cost-effectiveness of AEDs are needed.

Given the highly specific and sensitive algorithms for ventricular tachycardia and fibrillation detection and the encouraging preliminary observations with AEDs, it is probable that there will be an exponential use of these devices at athletic events and in the general community setting over the next several years. The legal barriers, which have been an obstacle to their widespread utilization, are being removed as individual states limit liability for the medical directors of the programs using the devices and the purchaser and trained users of AEDs.

## Cardiovascular Screening

Preparticipation screening of athletes for early detection of cardiovascular abnormalities that might predispose to sudden cardiac death has been recommended by a consensus panel of the American Heart Association and the American College of Cardiology[1,2,10-13,15,17] (Table 5). The purpose of screening is to provide medical evaluation for clearance to participate in competitive sports. Systematic evaluation intended to identify clinically relevant cardiovascular conditions that may increase the risks of athletic participation has been recommended before the initial engagement in organized high school grades 9 through 12 and collegiate sports.[1,2,10-13,15,17] This evaluation should be repeated every 2 years, with an interim history obtained in the intervening years. Recommended screening includes personal and family history and physical examination.[1,2,10-13,15,17] Routine ECGs and echocardiograms are not recommended, but it is acknowledged that they can be of value in selected individuals based on abnormal symptoms, history, or physical examination.

**TABLE 5**
Cardiovascular Conditions Associated with Arrhythmias and Sudden Death in the Athlete

| |
|---|
| Hypertrophic cardiomyopathy |
| Atherosclerotic coronary artery disease |
| Arrhythmogenic right ventricular dysplasia |
| Anomalous origin of coronary artery |
| Long QT syndrome |
| Wolff-Parkinson-White syndrome |
| Idiopathic ventricular fibrillation |
| Brugada syndrome |
| Myocardial bridge |
| Aortic valve stenosis |
| Subvalvular aortic stenosis |
| Pulmonary hypertension |
| Congenital heart disease |
| Myocarditis |
| Dilated cardiomyopathy |
| Marfan syndrome |
| Commotio cordis |

ECG in the athlete has a relatively low specificity as a screening test because of the high frequency of changes that occur in association with the normal physiologic adaptations to training in the athlete's heart. ECG has a low specificity because of the high frequency with which ECG alterations, including sinus bradycardia, first-degree AV block, voltage criteria for right and left ventricular hypertrophy, J point and ST segment elevation and T wave abnormalities, including peaked T waves and T wave inversions, are found in athletes. In the setting of preparticipation screening, it has been estimated that approximately 20% to 25% of athletes will have ECG patterns that are sufficiently unusual to warrant further evaluation with echocardiography.[17,18] There are a number of ECG abnormalities that, if detected, might identify a group of athletes at high risk for sudden death. These include ventricular preexcitation or WPW pattern on ECG, LQTS, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, and Brugada syndrome. However, these ECG abnormalities are sufficiently uncommon so that routine screening with ECGs is not recommended.[1,2,10-13,15,17] There remains no standardization among the athletic governing bodies or among the states regarding the history, physical examination, and other components of the evaluation. There is a clear need to develop a systematic national standard for preparticipation

medical evaluation with standardized history and physical examination recommendations. When a cardiovascular condition is diagnosed with a known genetic basis, clinical screening of family members should be performed. In the future, clinical markers of a cardiovascular condition associated with arrhythmias may be supplemented by genetic markers.

For athletes older than 35 years, it is recommended that a careful history for coronary risk factors, such as familial occurrence of premature coronary disease, hypertension, unfavorable lipid profile, tobacco use, or symptoms suggestive of possible coronary artery disease, be taken.[1,2,17] If an individual is identified as being at risk for coronary artery disease or if there are symptoms suggestive of ischemia, an exercise stress test should be considered. In addition, it is reasonable to consider performing exercising testing in males over age 40 years or females over age 50 years in whom coronary artery disease is suspected on the basis of at least two risk factors (other than age and gender) or one markedly abnormal finding.[1,2,17] However, in older trained athletes, the routine application of exercise testing to detect coronary artery disease is limited by its low specificity and pretest probability.[17]

Recent data from a national screening program for young athletes before participation in competitive athletics suggest that the identification and disqualification of affected athletes by screening may prevent sudden death due to hypertrophic cardiomyopathy.[31] This screening program included the recording of history, physical examination, ECG, and limited exercise testing in all individuals.[31] Subsequently, an echocardiogram, Holter monitoring, and submaximal exercise tests were performed in individuals who had positive test results. The cost-effectiveness, practicality, and utility of the screening process are limited by the uncommon occurrence in the general and athletic population of these relevant cardiovascular conditions that predispose to sudden death.[1,2,10-13,17] To date, the sensitivity and specificity of screening programs have been marginal. One retrospective study concluded that the history and physical examination were able to detect cardiovascular abnormalities in only 3% of high school and college athletes who ultimately died suddenly of cardiovascular disease.[10] Additionally, prospective studies in large athletic populations using ECG and echocardiography have shown a low yield of identifiable cardiovascular conditions that predispose to sudden death.[1,2,10-13,17]

Despite these limitations, an American Heart Association Consensus Panel recently recommended the concept of preparticipation cardiovascular screening for athletes based on medical and ethical considerations.[1] These recommendations included a personal and family history with a physical examination targeted to detect the cardiovascular conditions known to predispose to sudden cardiac death. The panel recognized the limitations of this approach but considered it the most practical and best available current strategy for screening large populations of young athletes.[1,2,17]

## Illicit Drug Use

The highly publicized sudden death of a number of athletes in association with the illicit use of drugs has heightened the awareness of the problem of substance abuse in the athlete. Athletes use drugs for a variety of reasons, including therapeutic indications, to enhance performance, for recreational or social reasons, or to mask the presence of other drugs during drug testing.[53-62] For example, probenecid is known to mask the presence of multiple drugs in the urine.[53,60]

Drugs that enhance exercise capacity or athletic performance are ergogenic.[53] By definition of the International Olympic Committee (IOC), an ergogenic drug is any substance foreign to the body or any physiologic agent taken in abnormal quantities or by abnormal entry into the body with the intent of increasing performance in competition by the athlete.[53,54] These agents include testosterone, other anabolic steroids (such as stanozol, oxandrolone, methandrostenolone, nandrolone, oxymetholone, and oxymesterone), the androgen precursor human growth hormone, erythropoietin, and amphetamines. Some athletes believe that cocaine is ergogenic.[53] Use of testosterone and synthetic derivatives is a major problem in the United States, with estimates of $>1$ million individuals having used or currently using anabolic steroids to promote athletic performance.[53,54] Although there is little systematic evaluation of their effects on the cardiovascular system, premature coronary artery disease and myocardial infarction,[54-58] cardiomyopathy,[57,58] and sudden death[58] have been reported in association with anabolic steroid use. Other mechanisms by which anabolic steroids can adversely affect the cardiovascular system include increase in aggregation of platelets, activation of the hemostatic system, hypertension, and worsening of atherogenic lipids.[54-58] Premature coronary artery disease and cardiomyopathy have been reported with human growth hormone use.[60] Sudden death, coronary artery spasm, myocardial infarction, ventricular arrhythmias, and cardiomyopathy have been reported in association with amphetamine use.[53] Recently, ma huang, which contains ephedra alkaloids, is gaining popularity as a performance-enhancing substance.[59] It also has been associated with adverse cardiovascular events, including myocardial infarction, sudden death, cerebrovascular events, and cardiac arrhythmias.[59]

In the athletic population, cocaine, alcohol, marijuana, smokeless tobacco, sedatives, and antidepressants are among the drugs that are used that may impair function.[53] Of these drugs, cocaine has received the most attention because of the high-profile deaths of Len Bias and Don Rogers.[53] Before the adverse cardiovascular effects of cocaine were recognized,[61] many individuals believed that cocaine was a harmless and nonaddicting substance. In the mid-1980s, it was estimated that 30 million Americans had used cocaine and 6 million were regular users.[53,61] It now is appreciated that cocaine use is associated with cardiac arrhythmias, myocardial infarction, pulmonary edema, ruptured aortic aneurysm, myocarditis, and dilated cardiomyopathy.[53] Cocaine acts as a local anesthetic agent by inhibiting the influx of sodium into cardiac cells.[53,60-62] Rhythm disturbances, including atrial and ventricular arrhythmias, are related to the local anesthetic and sympathomimetic properties of the substance.[53,60-62] Cardiovascular effects of cocaine are mediated by block of the reuptake of norepinephrine in both the central and peripheral nervous systems, thereby causing tachycardia, vasoconstriction, acute hypertension, ventricular arrhythmias, and seizures.[53,60-62]

In an effort to control the use of illicit drugs in athletes,

**TABLE 6**
Mechanisms of Syncope in the Athlete

| Mechanism | Associated Cardiovascular Conditions |
|---|---|
| Neurally mediated | Usually no structural heart disease |
| Exertional | |
| Postexertional | |
| Nonexertional | |
| Bradycardias | Physiologic adaptation to training |
| | Congenital complete heart block |
| | Conduction system disease |
| Supraventricular arrhythmias | Atrial fibrillation, atrial flutter |
| | AV nodal reentrant tachycardia |
| | AV reciprocating tachycardia |
| | Other supraventricular tachycardias |
| Ventricular arrhythmias | Hypertrophic cardiomyopathy |
| | Coronary artery disease |
| | Arrhythmogenic right ventricular dysplasia |
| | Long QT syndromes |
| | Dilated cardiomyopathy |
| | Myocarditis |
| | Valvular heart disease |
| | Congenital heart disease |
| | Idiopathic ventricular tachycardia |
| Reduced cardiac output | Aortic stenosis |
| | Atrial myxoma |
| | Pulmonary hypertension |
| | Dehydration |

a number of organizations have implemented drug testing programs. Substances of abuse are generally readily detected in the urine. The United States Olympic Committee was the first athletic organization to initiate drug testing of athletes by conducting testing. The list of substances prohibited includes stimulants, narcotics, anabolic agents, diuretics, peptide and glycoprotein hormones and analogues, alcohol, marijuana, local anesthetics, corticosteroids, and beta-blockers.[53] A program for drug testing at the collegiate level has been implemented by the National Collegiate Athlete Organization to test championship teams in all sports.[53] Since 1986, the percentage of samples testing positive for stimulants and anabolic steroids has stabilized at 0.37% and 1%, respectively.[53]

Although these programs have been in place for many years, there are little data regarding the results of the testing or the efficacy in preventing substance abuse. In screening athletes and evaluating athletes with symptoms of or having a documented arrhythmia, all health care professionals should be aware of the possible role of illicit substances. The efficacy of screening and education programs and the potential interaction of physical activity with the effects of illicit drugs merit further study.

## Syncope

By definition, syncope is sudden loss of consciousness and postural tone with spontaneous and complete recovery after a short duration. Athletes presenting with syncope present a particular challenge because the potential causes can range from the common faint or vasovagal spell with a benign prognosis to a life-threatening condition such as ventricular tachycardia[63] (Table 6). Although data exist regarding the frequency of syncope in a general population,[64] there are little or no data on the frequency of loss of consciousness in the athlete. Generally, syncope results from a sudden decrease in blood flow to the brain stem and reticular activating system, which may result from decreased cardiac output, decreased systemic vascular resistance, or both occurring simultaneously.[63-80]

The mechanisms and causes of syncope in the athlete include the common vasovagal spell or neurocardiogenic syncope.[77-82] Exercise-induced neurocardiogenic syncope generally occurs immediately after exercise and rarely occurs during exertion.[77-80] Ventricular arrhythmias as the cause of syncope generally occur in the setting of some underlying structural heart disease (such as hypertrophic cardiomyopathy), arrhythmogenic right ventricular dysplasia, cardiomyopathy, myocarditis, valvular heart disease (such as aortic stenosis), pulmonary hypertension, or congenital heart disease. In contrast, bradycardia as the cause of syncope can occur as a result of neurally mediated syncope and commonly is associated with a vasodepressor component resulting in peripheral vasodilation. Extreme sinus bradycardia or AV block due to heightened vagal tone as a training effect also can cause syncope, most commonly at rest. Underlying sinus node dysfunction, congenital or acquired conduction system disease, or supraventricular arrhythmias also must be considered in the differential diagnosis of syncope in the athlete.

The history and physical examination are the cornerstone of the initial evaluation of the athlete with syncope, with particular attention to the details of activities immediately preceding the episode of loss of consciousness. Witness observations may provide useful information regarding the abruptness, duration, and recovery from the loss of consciousness. Premonitory symptoms, such as nausea, diaphoresis, and yawning, may indicate a vasovagal spell. Syncope occurring without premonition, associated with injury, preceded by palpitations, or occurring during exercise mandates further evaluation to exclude underlying structural heart disease or an arrhythmic cause of syncope.

Physical examination should assess orthostatic hypotension, carotid sinus hypersensitivity, and signs of aortic stenosis, pulmonary hypertension, hypertrophic cardiomyopathy, or other forms of organic heart disease. In approximately 50% to 60% of patients, the likely cause of syncope can be identified from the history and physical examination. ECG should be part of the routine evaluation of the athlete with syncope and will give an additional diagnostic yield of up to 10%.[63] An echocardiogram should be performed in all athletes with syncope to exclude structural heart disease not detected by the history and examination. Usually, hypertrophic cardiomyopathy is not obstructive and, therefore, is not associated with a murmur or diagnostic ECG abnormalities, but it can be associated with serious cardiac arrhythmias.[4,6,9,48] A standard stress test will rarely reproduce symptoms but should be considered in athletes with exercise-induced syncope. It is reasonable to start at a high level of exercise, such as Bruce stage 4, to simulate the abrupt physiologic demands of athletics. In the absence of structural heart disease, it may be reasonable to perform either ambulatory monitoring or a loop monitor for a prolonged period of time to assess for any arrhythmias that might be associated with syncope. Tilt-table testing has been advocated as a marker of neurocardiogenic syncope but is associated with false-positive results in some athletes.[69] Invasive testing with cardiac catheterization should be reserved for patients in whom there is an indication based

on the presence of structural heart disease. Coronary artery disease is rare in those <40 years of age, but occasionally anomalous coronary origin is a cause of exercise-induced syncope in the younger athlete.[14] Anomalous origin of the coronary arteries can be detected in many athletes by transthoracic or transesophageal echocardiography.

Therapy for syncope is directed by the specific cause. Definitive therapy for bradyarrhythmias or tachyarrhythmias with pacemaker, pharmacologic, ablation, or ICD therapy should be accompanied by advice regarding eligibility for the athlete according to the Bethesda Guidelines.[16,76] Although syncope precedes sudden death in 17% to 50% of athletes and precedes exercise-related sudden death in 86% of individuals, in most instances the underlying cause of syncope is not due to life-threatening arrhythmias or structural heart disease.[67] Nonetheless, evaluation with noninvasive testing and selective use of invasive tests, including cardiac catheterization and electrophysiologic testing, are warranted to exclude or define these conditions before making therapeutic recommendations or allowing resumption of athletic activities.

## Legal Considerations

There are multiple legal principles and considerations that should be borne in mind by the physician evaluating the individual athlete. In general, the standard of care provided to the athlete by the physician should be consistent with the knowledge, skill, and care ordinarily possessed and used by members of his or her specialty in good standing considering the state of medical science at the time that such care is rendered.[81] In this regard, consensus guidelines have been developed regarding cardiovascular care of athletes.[16] These guidelines focus on issues related to medically clearing or excluding athletes from athletic participation once a cardiovascular abnormality has been identified.[16]

More recently, the American Heart Association has developed recommendations regarding preparticipation screening of athletes.[1,2,17] These guidelines use the best available knowledge regarding cardiovascular conditions in the athlete to make expert consensus recommendations regarding screening, evaluating, treating, and allowing continued athletic participation for the individual athlete by the physician. Such guidelines have the advantage of consolidating expert medical knowledge and experience to enable physicians to provide medical care to the athlete based on consensus recommendations rather than their own background and experience. To that extent, they are intended to enhance the quality of cardiovascular care to the individual athlete and to improve and protect the medical profession.[81]

However, the guidelines do not conclusively establish the standard of care with which every physician must comply.[81,83] The true legal standard of care is rather defined by the reasonable or accepted practice within a physician's specialty. Guidelines such as those noted earlier[16] are relevant judicially in resolving issues related to the standard of care.[81,83] Based on current law and legal precedent, such consensus guidelines established by medical organizations and based on the current medical knowledge represent admissible evidence of what constitutes good medical practice in matters related to malpractice litigation.[81,83] The American Heart Association recommendations on preparticipation physical examinations[1,2,17] and participation in athletics[16] now have been endorsed by five separate organizations, including those consisting of family physicians, pediatricians, orthopedic surgeons, and sports medicine physicians.[81,83] Endorsement of these recommendations provides strong legal evidence that adherence to them currently constitutes reasonable or acceptable medical practice in providing cardiovascular evaluation and medical clearance.[81,83]

All educational institutions and professional teams are required to use reasonable care in overseeing their athlete programs. However, there is no clear legal precedent regarding their duty to conduct preparticipation screening of athletes to attempt to detect medically significant abnormalities.[81,83] Importantly, a physician who has medically cleared an athlete to participate in competitive sports is not necessarily legally liable for an injury or death caused by an undetectable cardiovascular condition.[82] For medical malpractice liability to be present would require proof that a physician deviated from usual, customary, or acceptable medical practice in his or her specialty during performance of preparticipation screening, and that standard diagnostic criteria and techniques would have disclosed the condition.[82]

There is widespread acknowledgment of the limitations associated with any program for preparticipation screening. In the future, there will likely be development of more reliable diagnostic procedures that are practical, feasible, and cost-effective.[81-83] Currently, the American Heart Association recommendations for cardiovascular preparticipation screening of athletes[1,2] form the basis for the proper standard of medical care.[82] These recommendations and guidelines will establish the legal standard of care if generally accepted or customarily followed by physicians or if relied upon by courts to determine the nature and scope of legal responsibility of organizers of athletics.[82]

Based on these considerations, the individual physician should generally comply with the consensus guidelines that provide a medical and legal standard of care by following generally accepted good medical practice within his or her respective specialty as determined by reasonable and accepted practices and current state-of-the-art consensus guidelines.[81,83] The most likely potential areas of liability by physicians are in failing to conduct appropriate screening or diagnostic tests and misinterpreting test results that impact on fitness for participation. Additional areas in which liability may be incurred include providing improper treatment of a cardiac condition, improper clearance of an athlete with a cardiovascular condition that exposes the athlete to risk of an adverse outcome and/or inadequately disclosing the medical risks of participation in athletics, or failing to follow the generally accepted guidelines discussed earlier for cardiovascular care of the athlete.[81,83]

## Recommendations

Evaluation and management of athletes with symptoms of or having clinically documented arrhythmias is a complex multifaceted problem. Present methods of screening with history and physical examination are not sufficiently sensitive or specific to accurately identify which asymptomatic athlete is at risk to develop an arrhythmia or death. Further evaluation of standardized screening programs with long-term outcomes of the athletes restricted from or allowed to participate is needed. There is a clear need to

develop guidelines for a standard national preparticipation medical evaluation for all high school and college athletes, with tracking of outcomes. Detailed national and international registries of screening programs and athletes with cardiovascular conditions that predispose to cardiac arrhythmias are needed. Such registries can serve as an invaluable repository of patients for future analysis of the molecular and genetic bases of these cardiovascular conditions.

Athletic organizations, athletes, educational and legal institutions, coaches, trainers, team physicians, and lawyers need to develop strategic partnerships to shape policy on issues related to assessment of risk and assumption of responsibility for athletic activities. When differences of opinion exist among experts regarding the eligibility of an athlete for competition, an independent expert panel could be established to assess the risk and make recommendations.

Additional studies are needed to assess the efficacy and cost of availability of the AED at athletic events. Because early resuscitation and defibrillation of the cardiac arrest victim has been efficacious in other settings, consideration should be given to making AEDs available at athletic events, with a coordinated response system and tracking of outcomes.

Educational and enforcement programs for performance-enhancing and illicit drugs should be refined, with widespread implementation and reporting of its outcomes. Additional research is needed to clarify the safety of athletic participation of the athlete with an ICD. Continued basic and clinical research of the factors that can trigger and prevent arrhythmias in the athlete is needed. Further research also is needed to determine how genotype analysis can supplement clinical information and serve as a screening tool in individuals at risk for cardiovascular conditions associated with arrhythmias.[84] Cost-effective and practical preventive strategies for commotio cordis are needed. Finally, as more information becomes available regarding these and other issues related to arrhythmias in the athlete, it is important that there be timely updates of consensus statements by experts from a broad background of disciplines.

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