Cardiac arrhythmias in the athlete range from the benign and asymptomatic to the symptomatic and even those that are life threatening. Athletes—because of their extreme fitness and corresponding high vagal tone—may be more susceptible to certain arrhythmias, including bradyarrhythmias and atrial fibrillation (AF), and may be more prone to sudden cardiac death than the general population (1, 2). It is the task of the physician to determine which athletes with arrhythmias or symptoms consistent with arrhythmia require further workup and therapy and which ones can safely return to competitive play. This article discusses the different types of arrhythmias that may occur in athletes and focuses especially on the treatment of these arrhythmias (both supraventricular and ventricular) because much progress has been made in the treatment of cardiac arrhythmias during the last 5 years. The recommendations herein for participation in sports are based on those from the 1996 Bethesda Conference regarding athletic participation (3).

Bradyarrhythmias

Sinus Bradycardia

Because of the high resting vagal tone present in athletes, bradyarrhythmias are common in this population (Table 1) (4–7). Because vagal tone influences both sinus node and atrioventricular (AV) nodal function, bradyarrhythmias related to both may occur. Indeed, it has been suggested that sinus bradycardia occurs in direct relationship to the individual’s level of fitness. With the high vagal tone at rest that is present in competitive athletes, these individuals’ sinus rates can drop as low as 30/min during sleep (5, 7). However, with the withdrawal of vagal tone such as that which occurs when exercise ceases, peak heart rates are not affected. Therefore, sinus bradycardia is rarely an indication for further workup and therapy. The exception is the rare athlete, particularly if older than age 35, in whom sinus node rate during exercise does not appear to be normal for age and in whom symptoms of lightheadedness and exertional fatigue clearly are related to the bradycardia. This symptom can be suggested not by the resting bradycardia but by the blunted peak heart rate and a delayed heart rate rise in response to exercise. Peak heart rate in the trained athlete can rise to approximately 220/min minus the athlete’s age. In younger athletes, chronotropic incompetence (i.e., inability to increase the heart rate with activity) is rare and is thought to be due to a genetic predisposition to sinus node dysfunction (8). In older athletes, however, sinus node dysfunction becomes more common with age, and therefore chronotropic incompetence is observed more frequently in this group. The indications for treatment in patients with sinus bradycardia are based on symptoms of syncope, presyncope, and occasionally exercise limitation caused by chronotropic incompetence (9). Electrophysiologic studies are of little use for the assessment of sinus bradyarrhythmias (10).

Unfortunately, drugs are not effective in the management of sinus bradycardia; therefore, the only valid treatment is a permanent pacemaker. Current generation permanent pacemakers can increase the patient’s heart rate based on a sensor. In the past, sensor technology was based on the piezoelectric crystal, which increased the patient’s heart rate in correspondence with the vibration of the pacemaker. Some current pacemakers include not...
only the piezoelectric crystal but also a minute ventilation sensor that modulates the patient’s heart rate based on his or her respiratory rate. Because the ventilation sensor increases the patient’s heart rate in relation to the patient’s respiratory rate, the heart rate response is more physiologic with these pacemakers.

Restrictions on sports participation among patients with pacemakers are based on concerns about pacemaker lead integrity. In younger patients with less dense subcutaneous tissue, the pacemaker and leads are potentially more vulnerable to damage from bodily trauma. Thus, these patients’ participation in sports that involve intense physical impact is not advised (3). Older patients generally not only have more subcutaneous tissue but also are less likely to participate in trauma-related sports such as football. Exercise that involves repetitive movement of the clavicle over the ribs (e.g., the use of free weights) also puts the lead at increased risk for damage and therefore should be discouraged.

**Bradyarrhythmias Caused by Heart Block**

Wenckebach (or Mobitz I heart block) at rest also is common in athletes and, similarly to sinus bradycardia, is related to trained athletes’ high vagal tone (7, 11). Again, as in sinus bradycardia, Wenckebach at rest is resolved by the withdrawal of vagal tone as occurs with exercise. Therefore, for the athlete with Wenckebach that occurs during sleep or at rest and who has no symptoms of fatigue, syncope, or presyncope associated with Wenckebach, no treatment is necessary. If symptoms of syncope, presyncope, or Wenckebach during exertion are present, however, then a pacemaker generally is indicated, and the same restrictions on sports participation as those for the athlete with sinus bradycardia apply (9).

Higher degrees of heart block, such as Mobitz II or complete heart block, are pathologic in any individual, including trained athletes. The presence of Mobitz II or complete heart block signifies marked disease of the His-Purkinje system and generally is accepted as a Class I indication for permanent pacemaker implantation, even in the absence of symptoms (9). Occasionally, a 2:1 block may occur. In individuals with this condition, it is not clear whether the level of the block is at the AV node or in the His-Purkinje system. One clue to the location of the block is the width of the QRS complex. If the QRS complex is of normal duration, then it can be inferred that the His-Purkinje system is intact and that the level of block is at the AV node. If the QRS complex is widened, however, then it is more likely that the level of block is in the His-Purkinje system, and permanent pacing may be required. In occasional cases in which the level of block cannot be determined, an electrophysiologic study can be helpful in determining not only the level of block but also the indication for permanent pacemaker placement. If the block is in the AV node, then the athlete is treated similarly to patients with Wenckebach; if the level of block is in the His-Purkinje system, however, the athlete is treated as a Mobitz II patient.

**Supraventricular Arrhythmias**

Supraventricular arrhythmias (Table 2) are not particularly common in athletes, with the possible exception of AF, which may occur frequently because of athletes’ high vagal tone (2). Aided by the observations and insights of radiofrequency (RF) ablation, it has come to be appreciated that supraventricular tachycardias can be grouped into four or five categories instead of the multitude of types that were proposed in the past (12–14). The treatment of supraventricular tachycardias has been revolutionized by RF ablation, which allows actual cure in the majority of cases, removing the need for lifelong pharmacologic treatment. The most common supraventricular tachycardia is sinus tachycardia, which is a normal response to physiologic stresses such as exercise, fear, anger, or a multitude of pharmacologic agents. Sinus tachycardia consequently does not require therapy. Atrial premature contractions are observed in many individuals, including endurance athletes (15), and do not require treatment unless they are associated with symptoms. In those athletes with symptomatic atrial premature beats, beta-blockers or calcium channel blockers may provide some relief. However, beta-blockers are banned in many sports and are tested for in drug screenings.

**Atrial Fibrillation and Flutter**

Perhaps the most common abnormal arrhythmia in the athlete is AF. It is thought that athletes are more vulnerable to AF because of their high vagal tone and consequent bradycardia that allow for dispersion of atrial repolarization and more susceptible to AF (2, 16). During the past few years, it has become appreciated that AF that

---

**Table 1. Diagnostic and therapeutic approach for bradyarrhythmias**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Symptoms</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>Competitive athletics</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-degree HB</td>
<td>None</td>
<td>Echocardiogram</td>
<td>None</td>
<td>No restrictions</td>
</tr>
<tr>
<td>Wenckebach</td>
<td>LH, syncope</td>
<td>Monitor, ECG</td>
<td>PPM</td>
<td>No bodily collision</td>
</tr>
<tr>
<td>Wenckebach</td>
<td>None</td>
<td>Monitor, ECG</td>
<td>PPM</td>
<td>No bodily collision</td>
</tr>
<tr>
<td>Mobitz II or CHB</td>
<td>None</td>
<td>Monitor, ECG</td>
<td>PPM</td>
<td>No bodily collision</td>
</tr>
</tbody>
</table>

HB, heart block; ECG, electrocardiogram; LH, lightheadedness; PPM, permanent pacemaker; CHB, complete heart block.

Recommendations for athletic competition are based on the 26th Bethesda Conference.

Table 2. Diagnostic and therapeutic approach to supraventricular arrhythmias

<table>
<thead>
<tr>
<th>Condition</th>
<th>Symptoms</th>
<th>ECG</th>
<th>Diagnosis</th>
<th>Treatment options</th>
<th>Competitive athletics</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVNRT</td>
<td>Palpitations, LH,</td>
<td>Monitor, EPS</td>
<td>BB, Digoxin, Ca ch bland, RFA</td>
<td>After 3–6 months symptom-free</td>
<td></td>
</tr>
<tr>
<td>WPW</td>
<td>Asymptomatic</td>
<td>Short PR delta waves</td>
<td>No therapy, RFA if high risk</td>
<td>To compete, athletes should undergo an EPS to stratify risk of SCD.</td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>Palpitations, LH,</td>
<td>Short PR delta waves</td>
<td>Antiarrhythmics, RFA</td>
<td>After 3–6 months symptom-free</td>
<td></td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Palpitations</td>
<td>Often NL</td>
<td>Rate control, anticoagulation, antiarrhythmics, RFA if focal atrial tachycardia</td>
<td>If warfarin is used for anticoagulation, sports with bodily contact should be prohibited.</td>
<td></td>
</tr>
<tr>
<td>Atrial premature complexes</td>
<td>Palpitations Within NL limits</td>
<td>Monitor</td>
<td>Reassurance, BB if disabling symptoms</td>
<td>No restrictions</td>
<td></td>
</tr>
</tbody>
</table>

ECG, electrocardiogram; AVNRT, atrioventricular reentrant tachycardia; LH, lightheadedness; NL, normal; EPS, electrophysiologic study; BB, beta-blockers; Ca ch bland, calcium channel antagonists; RFA, radiofrequency ablation; WPW, Wolff-Parkinson-White syndrome; SCD, sudden cardiac death; PR, pulse rate.

Recommendations for participating in competitive athletics are based on the 26th Bethesda Conference on Recommendations for Determining Eligibility for Competition in Athletes with Cardiovascular Abnormalities.


occurs in an individual with a structurally normal heart (as defined by echocardiogram, cardiac catheterization, and stress testing) is most often due to atrial tachycardias that degenerate into AF (17). Many of these atrial tachycardias originate from pulmonary vein insertions into the left atrium, tissue that is physiologically similar to the sinus node. RF ablation of this arrhythmogenic atrial tachycardia tissue can cure AF (17, 18). Focal atrial tachycardia degenerating into AF must be distinguished from AF that occurs in association with chronic cardiac diseases, such as heart failure, hypertension, or coronary artery disease (CAD), in which AF is more likely due to stretch and irritation of the atria rather than to atrial tachycardias. Currently, RF ablation for idiopathic AF is in development and criteria for patient selection continue to evolve. In the trained athlete with a normal heart and idiopathic AF, however, consideration should be given to loop and Holter monitoring to ascertain whether an atrial tachycardia precedes AF. If that is the case, RF ablation of the atrial tachycardia focus may offer the athlete a cure for AF.

Atrial flutter is caused by a reentrant circuit around the tricuspid valve and can be associated with a dilated right atrium. Atrial flutter is an unusual arrhythmia in the trained athlete and generally is the result of an underlying cardiomyopathy. Occasionally, however, individuals present with idiopathic atrial flutter. In athletes with atrial flutter, cure rates with RF ablation approach 80% (19), and RF ablation should be considered for these individuals even before trials of antiarrhythmic agents.

In the athlete with AF and atrial flutter, if RF ablation is not undertaken, pharmacologic treatment should be considered. General principles of pharmacologic treatment include rate control, anticoagulation, and antiarrhythmic therapy. Rate control is accomplished with beta-blockers, calcium channel blockers, digoxin. Digoxin, though effective in controlling resting heart rates in AF, is not effective with exercise-related heart rates; therefore, particularly in the athlete patient, it should not be relied on as the sole therapeutic agent. Beta-blockers and calcium channel blockers should be the first lines of therapy to control the ventricular rate response in AF and atrial flutter. In addition to rate control, the issue of anticoagulation should be considered in individuals with AF and risk factors for thromboembolism, including abnormal structural hearts, heart failure, diabetes, and hypertension. In these individuals, anticoagulation with warfarin is generally recommended. In the individual with a normal heart and no history of hypertension or diabetes, the risk of thromboembolism is low and no clear consensus exists on anticoagulation. It is possible that these individuals have a very low incidence of thromboembolism and that anticoagulation with warfarin therefore would not be needed. Individuals with short, rare bursts of AF have a very low incidence of thromboembolism, and thus anticoagulation may not be needed. In individuals whose AF duration and incidence are at an intermediate level, anticoagulation with aspirin should strongly be considered. In the individual with chronic AF or long, frequent bouts of paroxysmal AF, warfarin should be considered. Further ongoing trials evaluating the use of warfarin in idiopathic AF will shed more light on its use for these conditions. Use of warfarin increases the patient's risk of bleeding, and therefore the
patient’s participation in sports with the risk of potential trauma, such as skiing, may not be advisable.

**Atrioventricular Nodal Reentrant Tachycardia**

Atrioventricular nodal reentrant tachycardia (AVNRT) is a condition attributable to a reentrant loop in the lower right atrium in areas subtended by the AV node. Abrupt onset and termination of symptoms marks these tachycardias. Generally, the tachycardia demonstrates a narrow complex QRS, and the atrial activity is not evident during the tachycardia (Fig. 1). Pharmacologic treatment for AVNRT includes beta-blockers, calcium channel blockers, and digoxin. These agents decrease the frequency and often the severity of AVNRT. Because RF ablation has the ability to cure >95% of these individuals, however, athletes should be considered for RF ablation even before pharmacologic treatment is considered (12, 20). The main complication of RF ablation is the requirement for a permanent pacemaker, but with the current techniques, the incidence of an unsuspected need for a pacemaker is <1%.

**Atrioventricular Reentrant Tachycardia**

Another rhythm in the athlete and in young individuals is atrioventricular reentrant tachycardia (AVRT) that is due to an accessory bypass tract. When the bypass tract is evident on the surface electrocardiogram (ECG), this condition commonly is referred to as Wolff-Parkinson-White syndrome (WPW). Often, however, the bypass tract conducts only in a retrograde fashion, and therefore a delta wave will not be present on the surface ECG. Arrhythmias in these conditions are dependent on antegrade or retrograde conduction through the AV node and corresponding retrograde or antegrade conduction through the bypass tract. Pharmacologic treatment for AVRT includes beta-blockers, calcium channel, and digoxin as well as Class IA, IC, or III antiarrhythmic agents. Because the rates of cure with RF ablation are >95% (21), however, and because patients with WPW who have symptoms may be at risk for sudden death, strong consideration should be given to early RF ablation, even before pharmacologic therapy in the setting of symptoms (12, 20, 22).

The athlete with a WPW pattern on ECG and no symptoms of palpitations, syncope, or presyncope represents a more difficult problem. It is thought that although these individuals have a lifelong risk of developing arrhythmias, the risk of developing a fatal arrhythmia is quite low; therefore, most experts do not advise routine electrophysiologic testing of these individuals (23–26).

---

**Fig. 1.** 12-lead electrocardiogram of an 18-year-old female who participated in competitive crew. This electrocardiogram demonstrates a wide complex tachycardia without apparent atrial activity. The wide complex is due to a rate-related right bundle branch block, and the lack of atrial activity is caused by simultaneous depolarization of the atrium and ventricles, such as those observed in atrioventricular nodal tachycardia (AVNRT). This woman was debilitated not only by her AVNRT but also by the beta-blocking agents used to treat her. She underwent successful ablation of the tachycardia.
Because of the extreme conditions present in competitive sports, however, others recommend characterization of the bypass tract with possible ablation for the asymptomatic athlete with a WPW electrocardiographic pattern (27, 28). If it is shown during an electrophysiologic evaluation that the bypass tract is able to sustain a rapid arrhythmia, particularly one that causes hemodynamic compromise, then an ablation should be performed. Athletic restrictions generally are not recommended for the asymptomatic individual with WPW (22).

Table 3. Ventricular arrhythmias in different forms of structural heart disease

<table>
<thead>
<tr>
<th>Condition</th>
<th>Symptoms</th>
<th>ECG</th>
<th>VT morphology</th>
<th>Treatment options</th>
<th>Competitive athletics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic LV VT</td>
<td>Palpitations, LH,</td>
<td>NL</td>
<td>RB, left axis</td>
<td>RFA, Ca chan ant, BB</td>
<td>No restrictions 3 months post-RFA</td>
</tr>
<tr>
<td></td>
<td>syncope</td>
<td></td>
<td></td>
<td>BB</td>
<td></td>
</tr>
<tr>
<td>Idiopathic RVOT VT</td>
<td>Palpitations, LH,</td>
<td>NL</td>
<td>LB, inferior axis</td>
<td>RFA, Ca chan ant, BB</td>
<td>No restrictions 3 months post-RFA</td>
</tr>
<tr>
<td></td>
<td>syncope</td>
<td></td>
<td></td>
<td>BB</td>
<td></td>
</tr>
<tr>
<td>HCM</td>
<td>Palpitations,</td>
<td>Q’s-ant, LV hypertrophy</td>
<td>Ventricular fibrillation</td>
<td>BB, AAD, myomectomy, ICD</td>
<td>Only low-intensity sports</td>
</tr>
<tr>
<td></td>
<td>syncope, SCD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARVD</td>
<td>Palpitations,</td>
<td>T-inv ant, RBBB, epsilon wave</td>
<td>LB, inferior axis</td>
<td>Sot or amio, ICD, RFA</td>
<td>Only low-intensity sports</td>
</tr>
<tr>
<td></td>
<td>syncope, SCD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>Palpitations,</td>
<td>Infarts, ischemic ST</td>
<td>RB or LB</td>
<td>Amio, ICD</td>
<td>Only low-intensity sports</td>
</tr>
<tr>
<td>disease</td>
<td>syncope, SCD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Idiopathic dilated cardiomyopathy</td>
<td>Palpitations,</td>
<td>Often LBBB</td>
<td>RB or LB</td>
<td>Amio, ICD</td>
<td>Only low-intensity sports</td>
</tr>
<tr>
<td></td>
<td>syncope, SCD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long-QT syndrome</td>
<td>Palpitations,</td>
<td>Long-QTc</td>
<td>Torsades</td>
<td>BB, PPM, ICD</td>
<td>Only low-intensity sports</td>
</tr>
<tr>
<td></td>
<td>syncope, SCD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anomalous coronary artery disease</td>
<td>Exertional chest</td>
<td>NL</td>
<td>Ventricular fibrillation</td>
<td>CABG</td>
<td>No restrictions post-CABG</td>
</tr>
<tr>
<td></td>
<td>pain, syncope, SCD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ECG, electrocardiogram; VT, ventricular tachycardia; LV, left ventricle; LH, lightheadedness; NL, normal; RB, right bundle; RFA, radiofrequency ablation; Ca chan ant, calcium channel antagonists; BB, beta-blocking agents; RVOT, right ventricle outflow tract; LB, left bundle; HCM, hypertrophic cardiomyopathy; SCD, sudden cardiac death; BB, beta-blockers; AAD, antiarrhythmic drugs; ICD, implantable cardioverter defibrillator; ARVD, arrhythmogenic right ventricular dysplasia; RBBB, right bundle branch block; LBBB, left bundle branch block; sot, sotalol; amio, amiodarone; PPM, permanent pacemaker; CABG, coronary artery bypass graft.


Fig. 2. 12-lead ECG of an asymptomatic 16-year-old swimmer with hypertrophic cardiomyopathy. Note the marked voltage and ST T-wave abnormalities, changes that are consistent with left ventricular hypertrophy. This young man’s septal thickness measured 4 cm. He was counseled not to swim competitively.
Ventricular Arrhythmias

Life-threatening arrhythmias rarely occur in the presence of a normal heart (Table 3). This is particularly true in the athlete population, in which >97% of those athletes who die suddenly have a defined underlying cardiac condition (1). In North America, the two most common cardiac causes of death are hypertrophic cardiomyopathy followed by congenital coronary artery abnormalities. In Italy, however, the most common underlying cardiac condition is arrhythmogenic right ventricular dysplasia (29). This article does not discuss the various cardiomyopathies because several other recent articles and reviews evaluate these conditions (30, 31). Treatment of individuals with underlying cardiomyopathy who have symptoms of syncope, presyncope, or cardiac arrest and care of asymptomatic athletes with structural heart disease known to put the patient at risk for sudden cardiac death are discussed next.

Athletes with Structural Heart Disease and Syncope or Sudden Cardiac Death

In the athlete with underlying structural heart disease such as CAD, anomalous coronary artery, dilated cardiomyopathies, hypertrophic cardiomyopathy (Fig. 2), and arrhythmogenic right ventricular dysplasia, the occurrence of resuscitated sudden death is an ominous sign, and these individuals require intensive treatment. In general, athletes (and all other patients) who experience resuscitated sudden death should undergo treatment with an implantable defibrillator, which has been shown to be superior to pharmacologic treatment for prevention of sudden death (32). This is true for individuals with hypertrophic cardiomyopathy (33), CAD (32), and dilated cardiomyopathies and may be true for those with arrhythmogenic right ventricular dysplasia (34) and long-QT syndrome as well (35). However, patients with anomalous coronary arteries can be cured by revascularization and these individuals do not necessarily require implantable cardioverter defibrillators.

Another subset of individuals who may not require an implantable cardioverter defibrillator are those with no structural heart disease and ventricular tachycardia. These patients generally are young and by definition have no CAD or myocardial disease. They can have idiopathic ventricular tachycardias originating from the right ventricular outflow or the right ventricular inflow tract or in the left posterior fascicle. Ablation can be curative for these individuals, and they may return to unrestricted athletic participation (36). Idiopathic ventricular tachycardia is an unusual condition, however, and structural heart disease must be ruled out by an echocardiogram, exercise stress testing, coronary angiography, and left and right ventriculograms.

In athletes with structural heart disease and syncope, the cause of the syncope should be presumed arrhythmic, and athletes should be treated similarly to those who have experienced resuscitated sudden death (unless another clear diagnosis that explains syncope can be made) (37). In athletes without known structural heart disease, syncope during peak exertion should be cause for an extensive workup. Exertional syncope may be arrhythmic in the presence of an occult and difficult-to-diagnose cardiac condition. Postexertional or nonexertional syncope is less likely to be arrhythmic, however, and an
Echocardiogram should suffice to rule out structural heart disease.

In the individual treated with an implantable defibrillator, participation in competitive sports should be restricted (22). The rationale for this recommendation is that exercise may provoke ventricular arrhythmias, and high catecholamine conditions may make ventricular defibrillation more difficult. Concern also is warranted for the integrity of the defibrillator and lead system with intense physical activity. Therefore, currently, athletes with an implantable defibrillator should be restricted to low-intensity sports such as golf. Those individuals with idiopathic ventricular tachycardias treated by ablation and those with anomalous CAD treated by revascularization can return to athletics following a 3- to 6-month period free of symptoms.

**Asymptomatic Athletes with Structural Heart Disease**

Like the patient with WPW who has no symptoms, the asymptomatic patient with a diagnosis of structural heart disease that is known to be associated with sudden cardiac death represents a difficult diagnostic and therapeutic dilemma. The treatment of these patients is related not only to the type of cardiac disease but also to the presence of risk factors. Evidence is accumulating that asymptomatic patients with hypertrophic cardiomyopathy and the presence of risk factors for sudden death may benefit from prophylactic treatment with implantable cardioverter defibrillators (33, 38). Evidence also exists that restriction from sports in asymptomatic athletes with hypertrophic cardiomyopathy can reduce risk (39). Currently, the treatment of asymptomatic athletes with arrhythmogenic right ventricular dysplasia should include restriction from competitive athletics; however, no evidence has been found that treatment with an implantable defibrillator is required.

In asymptomatic individuals with the long-QT syndrome, a family history of sudden death suggests a continued risk of ventricular arrhythmias. Beta-blockade and possibly more aggressive therapy with the implantable defibrillator should be considered (35). It is not clear whether the asymptomatic individual with long-QT syndrome should be restricted from sports. Electrophysiologic testing cannot prognosticate risk of sudden death in individuals with hypertrophic cardiomyopathy or long-QT syndrome but may play a limited role in the individual with arrhythmogenic right ventricular dysplasia (31). In the patient with CAD who has had no prior arrhythmic symptoms (if left ventricular ejection fraction is ≥40%) and the patient with idiopathic dilated cardiomyopathy and no prior arrhythmic symptoms, there is
little justification for prophylactic primary treatment of arrhythmias. In these individuals, athletic participation can be beneficial; however, vigorous competitive athletics generally should not be allowed for individuals with cardiac disease. Asymptomatic patients with CAD and left ventricular ejection fractions $\leqslant 35\%$ may benefit from electrophysiologic testing (40, 41).

**Sudden Cardiac Death in Individuals with Structurally Normal Hearts**

It has become appreciated in the past 10 years that several conditions of sudden cardiac death are not associated with structural heart disease. A significant subset of such individuals possess the Brugada syndrome, a specific genetic abnormality of the SCN5A channel (42, 43). Genetic transmission is usually autosomal dominant, and a predisposition for fatal arrhythmias is evident in affected individuals. Individuals can be screened for the presence of Brugada syndrome by the 12-lead ECG, in which incomplete right bundle branch block and ST segment elevation are present in the precordial leads (Fig. 3). Symptomatic individuals (i.e., those with syncope or resuscitated sudden death) with Brugada syndrome should be treated with the implantable defibrillator. Management of asymptomatic individuals with Brugada syndrome is presently unresolved, although some experts advocate defibrillator therapy even for this subgroup (44).

Another condition of sudden cardiac death in athletes with normal hearts is that of commotio cordis, in which sudden death in young individuals is produced by a nonpenetrating blunt impact to the chest wall (Fig. 4) (45–47). When arrhythmias are documented, they are ventricular fibrillation, and only 10% of individuals survive despite prompt resuscitation. In autopsies of these individuals, no evidence of acute or chronic cardiac disease is found. In an animal model of commotio cordis, we have shown that important variables of vulnerability to sudden death with chest-wall impact include timing, impact object hardness and speed, and location of the impact. Impact must occur within a 20-millisecond window on the upslope of the T wave (48). Baseballs thrown at 40 mph are more deadly than those thrown faster or slower (49). The harder the impact object, the greater the likelihood of a fatal arrhythmia—a finding that has implications for safety baseball use (48). Finally, the area of impact is critically important in that an impact at the center area of the heart is more deadly than one at the periphery of the heart (50). It is not clear whether individuals with resuscitated commotio cordis face a lifelong risk for recurrent ventricular fibrillation; however, currently it is thought that this is a chance occurrence and that implantable defibrillator therapy is not needed. Chest-wall protection should be advised for individuals who have survived a commotio cordis event.

**Conclusions**

 Bradyarrhythmias in the athlete are common and require treatment only if Mobitz II or complete heart or symptomatic Wenckebach is present. These pathologic bradyarrhythmias are very unusual in the trained athlete. Supraventricular tachycardias are no more common in the athlete than in the general population, with the possible exception of AF. Strong consideration should be given to early RF ablation because this treatment represents a cure for the tachycardia. Life-threatening ventricular tachyarhythmias in athletes generally are related to underlying heart disease, and in individuals with these symptoms, strong consideration should be given to implantable cardioverter defibrillators and restriction from competitive athletics.

Review of Wenckebach heart block in athletes.


Review of radiofrequency ablation for the supraventricular tachycardias.


Review of the mechanism and treatment of supraventricular arrhythmias.


Review of the mechanisms and treatment of supraventricular tachycardias.


Article describing the frequent occurrence of atrial and ventricular premature beats in athletes.


Vagal influences that are marked in athletes predispose some athletes to certain types of atrial tachycardias and AF.


Seminal work describing the initiation of AF from atrial fibrillation.


Describes radiofrequency ablation of atrial tachycardias that degenerate into AF; thus, AF in these individuals can be cured.


Atrial flutter can be cured in a majority of patients with radiofrequency ablation.


Review of radiofrequency ablation of supraventricular arrhythmias.


Seminal work describing radiofrequency ablation of accessory pathways.


Task Force of the Bethesda Conference on participation of athletes with cardiac disease, focusing on athletes with arrhythmias.


Article describing the usefulness of invasive electrophysiologic testing to separate the benign from the potentially life-threatening accessory pathways.


Long-term follow-up of patients with Wolff-Parkinson-White syndrome (WPW) in whom the risk of sudden death is low.


A review that discusses the risk of sudden death in patients with WPW.


A reasoned review of the risk of sudden death in the athlete with WPW.


Report that raised awareness of how exercise and isoproterenol can increase the conduction velocity of accessory pathways and thereby increase the risk of sudden death.


Personal perspectives on the asymptomatic patient with WPW in which the authors state their preferences for electrophysiologic evaluation.


A study conducted in Italy that found a high incidence of arrhythmogenic right ventricular dysplasia in athletes who die young.


Review of the underlying cardiac conditions in athletes who die at a young age.


Review of the underlying cardiac abnormalities in athletes who die at a young age and the implications for treatment in athletes with heart disease.


In patients with life-threatening arrhythmias, treatment with the implantable defibrillator is superior to treatment with amiodarone.


Article that describes multicenter experience in treating patients with hypertrophic cardiomyopathy with implantable defibrillators. In this study group, a high frequency of defibrillator therapy was observed even in those individuals without prior arrhythmic episodes.


Article that describes the authors’ experience in treating patients with arrhythmogenic right ventricular dysplasia using implantable defibrillators.


Data from the Long QT Registry on the long-term follow-up of patients with long-QT syndrome.


Article in which the site of origin of idiopathic ventricular tachycardia and the implications for radiofrequency ablation are discussed.

A high incidence of syncope was observed in soldiers in the weeks and months preceding sudden death.


Article in which the risk of sudden death in hypertrophic cardiomyopathy is directly related to the thickness of the ventricular septum.


Demonstration of efficacy of Italian screening program for detection of HCM—largely by virtue of routine use of standard 12-lead ECG.


Individuals with low left ventricular ejection fractions and nonsustained ventricular tachycardia that are induced into ventricular tachycardia by electrophysiologic testing have an improved survival rate when treated with implantable defibrillators.


Similar to (40), this study reports that individuals with low left ventricular ejection fractions, nonsustained ventricular tachycardia, and induced ventricular tachyarrhythmias have an improved survival when treated with implantable defibrillators.


An early description of Brugada syndrome.


Recent review of Brugada syndrome.


Brief book on the Brugada syndrome in which the authors discuss their preference for treating asymptomatic patients with the Brugada syndrome with implantable defibrillators.


Article describing the clinical and pathological characteristics of 25 commotio cordis victims.


Recent report from the Commotio Cordis Registry in which the clinical and pathological characteristics of 70 victims of commotio cordis are described.


Case report of a commotio cordis victim.


Experimental model of commotio cordis in which the mechanism of death is shown to be due to ventricular fibrillation caused by a blow from a baseball traveling at 30 mph during a narrow time segment of the cardiac cycle. In addition, the efficacy of safety balls is assessed in this model.


Abstract describing people’s vulnerability to sudden death when hit in the chest with baseballs traveling at different velocities.


Abstract describing the importance of impact location in commotio cordis.