ECG Findings in Active Patients
Differentiating the Benign From the Serious

N. A. Mark Estes III, MD; Mark S. Link, MD; Munther Homoud, MD; Paul J. Wang, MD

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In Brief: ECGs and cardiac rhythms of normal athletes can vary widely. The heightened vagal tone from athletic conditioning can result in variant ECG findings that may mimic serious disorders. ECG patterns of long-QT syndrome, arrhythmogenic right ventricular dysplasia, Wolff-Parkinson-White syndrome, and hypertrophic cardiomyopathy signal the need for further evaluation, therapy, and possible participation restriction. Radiofrequency ablation may be appropriate when symptomatic supraventricular arrhythmias or Wolff-Parkinson-White syndrome is present. Further research is needed to effectively distinguish normal ECG changes in the athlete from changes that underlie cardiac disease. Improvements in identifying athletes at risk of serious or life-threatening arrhythmias are also needed.

Changes in the surface electrocardiogram (ECG) are common in the athlete and reflect physiologic changes occurring in myocardial conduction, repolarization, and impulse formation from athletic conditioning and changes in autonomic tone. Because these changes can be interpreted as underlying cardiac disease, distinguishing normal from abnormal ECGs in the athlete can represent a challenge to the physician (table 1). The athlete may be subject to diagnostic evaluations (stress test, ambulatory monitoring) or invasive tests (electrophysiologic evaluation or cardiac catheterization) because of ECG abnormalities (1-40). In some instances, unnecessary restriction from exercise or unwarranted therapy may be recommended (41). In contrast, the ECG may manifest changes that indicate the potential for a life-threatening arrhythmia.
TABLE 1. Common ECG Findings in Athletes

- Sinus bradycardia
- Sinus arrhythmia
- First-degree AV block
- Second-degree Wenckebach AV block
- Incomplete right bundle branch block
- Notched P waves
- Right ventricular hypertrophy (by voltage criteria)
- Left ventricular hypertrophy (by voltage criteria)
- Repolarization abnormalities (including ST-segment elevation and depression)
- Corrected QT interval at the upper limit of normal
- Tall, peaked, and inverted T wave

Based on these considerations, it is particularly important to appreciate the ECG changes that can accompany athletic conditioning and those that signal underlying structural heart disease or vulnerability to cardiac arrhythmias. It is important to remember that an interpretation of an ECG or arrhythmia should always be evaluated in light of the patient's medical and family history, symptoms, physical examination, and, when appropriate, studies evaluating the presence of underlying structural heart disease (1-40).

**Sinus Bradycardia and Sinus Arrhythmia**

A broad spectrum of normal bradyarrhythmias exists in the conditioned athlete due to physical conditioning (1-19).

**Sinus bradycardia**

Among the most common findings in the athlete’s ECG is sinus bradycardia or a heart rate less than 60/min (figure 1). Generally, unless symptoms of sinus bradycardia are present, evaluation and therapy are not warranted.
Up to 91% of athletes will have sinus bradycardia at rest depending on the sport (1-12). In endurance sports such as long-distance running, bicycling, and swimming, the resting heart rates become lower as the level of conditioning increases (20). One study (21) of athletes documented average resting heart rates of 56/min in runners, 57/min in cyclists, 62/min in swimmers, and 51/min in wrestlers. Other studies showed that elite long-distance runners had a mean resting heart rate of 47/min (7), and one runner had an asymptomatic resting rate as low as 25/min (9). Sinus pauses longer than 2 seconds without symptoms are common in athletes (1). In runners, pauses have been observed up to 2.55 seconds while awake and 2.8 seconds while asleep (17).

Physicians may wish to assess the adequacy of sinus node function with an exercise stress test or an ambulatory monitor during exercise in those athletes with or without structural heart disease and resting heart rates less than 30/min. In the absence of symptoms from sinus bradycardia, the athlete need not be restricted from sports unless restriction is required based on underlying structural heart disease (41,42).

**Sinus arrhythmia**

This is common in the athlete and is defined as respiratory variation in heart rate with an increased heart rate during inspiration. The condition normally disappears with exercise (9,21,31-35).
Conduction Abnormalities

In 10% to 33% of athletes, ECGs reveal a delay in conduction through the atrioventricular (AV) node with first-degree AV block (defined as a PR interval >0.20) (2,14,15). Similar to sinus bradycardia, this phenomenon is generally attributed to enhanced vagal tone. First-degree AV and Wenckebach-type blocks in the athlete typically normalize with exercise or atropine as vagal tone is withdrawn (3,7,12,15,18). Review of 122,043 ECGs of healthy men in the military showed that 0.65% had first-degree AV block (17).

Mobitz type 1. Ambulatory monitoring has shown that up to 40% of athletes with first-degree AV block will also demonstrate Mobitz type 1 (Wenckebach-type) block with progressive PR prolongation before a nonconducted P wave followed by a PR-interval shortening (1,12). Among nonathletes, the prevalence of Mobitz type 1 block was 0.003% (17). This conduction disappears with exercise and with deconditioning.

Mobitz type 2. Among athletes, the frequency of Mobitz type 2 block and advanced AV block with more than one consecutive, nonconducted P wave is unknown, but it is rare compared with first-degree or Wenckebach-type block. Generally, Mobitz type 2 block should be considered a potential marker of underlying heart disease and an indication for further evaluation. Advanced AV block and concomitant structural heart disease or significant conduction system disease below the level of the AV node may be a marker of higher risk of progression to higher-degree AV block and often warrants permanent pacemaker implantation.

Complete heart block. This condition is extremely rare in athletes. Not uncommonly with congenital complete heart block, the junctional rhythm will accelerate to rates that allow athletic participation without symptoms.

Congenital complete heart block is most commonly asymptomatic and may be associated with certain types of structural heart disease. In individuals with symptoms such as presyncope or syncope, athletic participation should be restricted until they have undergone 3 to 6 months of definitive therapy for the bradycardia (41,42). Athletes who require a permanent pacemaker should not participate in contact sports (41,42). In the absence of symptoms, worsening of the AV block with exercise, or underlying structural heart disease, there is no need to restrict participation of athletes with first-degree AV block or Wenckebach block.

Intraventricular Conduction Delays

Incomplete right bundle branch block (RBBB) is the most common form of prolongation of intraventricular conduction and has been noted in up to 51% of athletes in some series (2,12,15,20). Prolongation of QRS conduction greater than 0.12 sec from a complete RBBB or left bundle branch block is extremely rare (1,16) and would prompt evaluation for underlying structural heart disease.

QRS Axis

Generally, the QRS frontal axis is between 0° and 90° (12,20). Left-axis deviation with an axis less than 0° is very uncommon and when present should be considered as a possible marker of underlying structural heart disease, particularly when accompanied by a bundle branch block.
P Wave

Multiple studies (2,3,7) have noted that the P-wave amplitude is greater in athletes than in age-matched nonathletes. The basis is unknown but may stem from atrial hypertrophy.

Ventricular Hypertrophy

It is common to find electrocardiographic criteria for either left ventricular hypertrophy (LVH) or right ventricular hypertrophy (RVH) in athletes (12,20). ECG evidence of LVH has been studied extensively in athletes and correlated with echocardiographic measurements of left ventricular wall thickness (12). LVH is seen in up to 76% of athletes by standard ECG criteria (2,3,5-7,13,15,25).

Sequential increases in voltage criteria occur with progressive training and regress with training cessation (20). When endurance athletes and sprinters are compared, endurance athletes have a significantly higher incidence of LVH than sprinters (44% vs 32%) (6). Both groups exhibit similar dimensions of left ventricle cavity dilatation, but the endurance athletes have greater wall thickness. No meaningful research has been done on what relationship exists between chest wall musculature and meeting the criteria for ventricular hypertrophy, but the general impression is that the thinner chest wall in endurance athletes is associated with ECG evidence of LVH. Nonetheless, athletes commonly meet the criteria for LVH, and the condition can be considered physiologic and within the normal spectrum for them.

Repolarization Changes in the Athlete

J-point elevation. J-point elevation, ST-segment elevation, and T-wave changes are reported with high frequency in athletes (figure 2) (1,12,18,43). It has been reported that these elevations normalize with exercise. J-point elevations may be difficult to distinguish from changes seen in ECGs of acute pericarditis because the elevation is commonly accompanied by ST-segment elevation. However, the clinical setting and the localization of the J-point changes to the inferior and anterior leads, in contrast to the global nature of the changes in pericarditis, can help differentiate the two.
ST depression and T waves. ST-segment depression is unusual in the athlete. Only one series (14) noted this finding in athletes; 3% of bicyclists had 0.1 mm J-point and ST-segment depression. In contrast, T-wave changes with inversions in the precordial or limb leads have been seen in up to 30% of endurance athletes (1,2,4,7,15,27,29). The T waves may also be peaked and tall (20), biphasic, or isoelectric. These T-wave changes have been observed to normalize with exercise or with isoproterenol infusion (18). Distinguishing such T-wave changes from metabolic or ischemic causes is based on the clinical setting.

Supraventricular Arrhythmias

Premature atrial contractions. Noted in a high percentage of long-distance runners (36,42), premature atrial contractions are generally asymptomatic but may be symptomatic or detected by the physician. When symptoms such as frequent or severe palpitations or light-headedness are present, beta-blockers are the first-line therapy. Because premature atrial contractions are frequently episodic, a reasonable approach is a few weeks’ therapy with a long-acting beta-blocker such as atenolol (25 to 50 mg daily). If the patient remains highly symptomatic, larger dosages can be given, or a long-acting calcium channel blocker such as verapamil (controlled-release, 120 to 240 mg daily) can be substituted. Without any significant cardiac disease, athletes with premature atrial contractions are not restricted from any sports (40-42).

Sustained supraventricular tachycardia and treatment. Sustained supraventricular tachycardia (SVT) in athletes generally stems from AV nodal reentry (AV nodal reentry tachycardia or AVNRT) (42). Typically, SVT starts and terminates abruptly. Triggers may include physical or emotional stress, caffeine, and alcohol. Valsalva maneuvers or intravenous (IV) adenosine or verapamil are commonly used to terminate the acute episode. IV adenosine (6 mg) is given as a rapid bolus over 1 to 2 seconds, followed by a fluid bolus. If the arrhythmia is not
terminated within 1 to 2 minutes, a 12-mg bolus can be given and repeated a second time if needed. Doses higher than 12 mg are not recommended.

IV verapamil may be given over 1 to 2 minutes. Second doses may be given later if no response is seen. A backup defibrillator should always be available. Verapamil is a vasodilator and may decrease blood pressure. During exercise, athletes may experience symptoms ranging from mild palpitations to syncope, depending on several factors, including the rate of the tachycardia and the blood pressure. Evaluation should include a history, physical examination, and invasive electrophysiologic evaluation to define the tachycardia mechanism. Occasionally, stress testing will provoke the arrhythmia or an episode can be documented with ambulatory or loop monitoring.

A curative approach with radiofrequency ablation is the preferred initial treatment (42-45), because it has a success rate greater than 95% and a low complication rate (<1%). Individuals electing pharmacologic therapy should have no episodes for 6 months before they resume competitive, low-intensity athletics (41-44). Drug therapy for patients who have recurrent SVT secondary to AVNRT includes beta-blockers, calcium channel blockers, digoxin, and, rarely, class 1A (quinidine, procainamide, disopyramide) or 1C (flecainide, propafenone) antiarrhythmic agents.

Rarely do physicians need to resort to therapy with class 1A antiarrhythmic or with class 1C antiarrhythmic agents, especially given these drugs’ potential for stimulating proarrhythmia. If used, 1A agents should be administered in the hospital with continuous ECG monitoring. A stress test to assess for arrhythmia recurrence or proarrhythmia is reasonable for individuals on pharmacologic therapy, though no prospective data have documented the approach’s predictive value. For athletes undergoing successful catheter ablation, competitive athletics may be resumed after 3 months (41-44). Radiofrequency catheter ablation is preferred for those with more severe arrhythmia-related symptoms or for athletes involved in high-intensity competitive sports.

**Wolff-Parkinson-White Syndrome**

Wolff-Parkinson-White (WPW) syndrome is found in approximately 3 in 1,000 individuals. This condition manifests on the surface ECG with a short PR interval (<0.12 sec) and a delta wave appearing as a slurred upstroke on the QRS complex from early ventricular myocardium activation (figure 3). The evaluation of the athlete who has asymptomatic WPW remains controversial, but many experts recommend observation without restriction of athletics since the risk of sudden death is very low (41-44).
**Characteristics.** The risk of sudden cardiac death in WPW syndrome is about 1 in 1,000 patient-years (40-42). The mechanism is considered to be atrial fibrillation with rapid conduction down the accessory pathway resulting in ventricular fibrillation. Symptomatic WPW patients should have an electrophysiologic study to establish the potential for the bypass tract to conduct rapidly to the ventricle. Attempts at determining noninvasive indices of the accessory bypass tracts' potential for rapid antegrade conduction have not been conclusive. A history of syncope, the presence of multiple bypass tracts, and an interval of 250 msec or less (equivalent to a heart rate <240/min) between two maximally preexcited QRS complexes during atrial fibrillation are considered markers for increased risk of sudden death. Intermittent preexcitation (abrupt loss of preexcitation and the lengthening of the PR interval) during a continuous ECG recording with minimal variation in the underlying sinus rate is considered a sign that the accessory bypass tract has a prolonged effective refractory period (41-43). Radiofrequency ablation is reasonable therapy if conduction to the ventricle exceeds 240/min (41-44).

**Causes.** Symptomatic arrhythmias from WPW may be due to AV reciprocating tachycardia, atrial fibrillation, or, rarely, ventricular fibrillation. Typically, the SVT in WPW is a narrow complex rhythm with P waves visible with a short RP interval during the tachycardia. Less commonly, the tachycardia proceeds antegrade through the bypass tract and retrograde via the AV node, resulting in a wide-complex preexcited tachycardia that can mimic ventricular tachycardia.

Manifest preexcitation may not be present on the surface ECG with a short PR interval and delta wave, but a retrograde bypass tract may still be identified at the time of electrophysiologic evaluation. This "concealed" bypass tract allows retrograde conduction from the ventricle to the atrium during the tachycardia and serves as the substrate for the reentrant circuit. Radiofrequency ablation is the preferred approach. All athletic activities can
be resumed 3 months after successful ablation; however, with drug therapy, resumption is allowed only after 6 months without recurrence (41-44).

The commonest arrhythmia associated with WPW syndrome is orthodromic atrioventricular reentrant tachycardia (AVRT). This manifests as a narrow-complex tachycardia and can be distinguished from AVNRT associated with dual AV pathways by the presence of retrograde P waves inscribed in either the ST segment or T waves of the tachycardia. Acute treatment of acute orthodromic AVRT is the same as for AV nodal tachycardia: IV adenosine, IV beta-blockers, or calcium channel blockers. If these agents fail or the patient is hemodynamically compromised, synchronized DC cardioversion should be employed.

**Treatment.** First-line drugs used in preventing recurrences are the class 1C antiarrhythmic agents flecainide acetate or propafenone. These drugs prolong the accessory bypass tract's refractory period and reduce the incidence of atrial and ventricular premature beats (SVT triggers). Although propafenone has not been approved for use in SVT, its beta-blocking effect may help limit recurrences. In patients with underlying structural heart disease, both drugs have the potential to exacerbate preexisting arrhythmias. Great care should be exercised in excluding underlying cardiac disease before initiating these agents in a monitored hospital setting. Although beta-blockers can be used in WPW patients, digoxin and calcium channel blockers are best avoided as chronic therapy. This limits their use to patients who have had electrophysiologic studies to confirm drug safety.

**Atrial Fibrillation and Atrial Flutter**

Atrial fibrillation or atrial flutter in the athlete may be more common than in age-matched nonathletes (45). One form of atrial fibrillation has been described in young patients without underlying structural heart disease but with paroxysmal atrial fibrillation. This proclivity to start during sleep and after meals may implicate a parasympathetic origin. Digoxin, a common treatment for atrial fibrillation, is not effective for this type and may make the condition worse. Self-limited atrial fibrillation in young individuals often occurs after heavy alcoholic beverage consumption.

The incidence of atrial fibrillation increases with age and with underlying heart disease. Patients younger than 65 with no underlying cardiovascular disease or hyperthyroidism are classified as having "lone atrial fibrillation." They have a relatively low risk of thromboembolism and constitute a distinct minority of patients. However, young athletes with atrial fibrillation are likely to be in this group. Management of recently diagnosed young patients should routinely include an ECG and an echocardiogram.

**Cardioversion.** Most cases of acute atrial fibrillation convert spontaneously within 24 hours of onset. However, cardioversion should not be delayed beyond the first 48 hours of onset because of the increased risk of developing left atrial thrombus. If atrial fibrillation exceeds 48 hours or the time of onset cannot be reliably determined, conversion to sinus rhythm should be deferred until after administration of 3 weeks of anticoagulation therapy.

**Other tests and therapies.** Athletes may be predisposed to atrial fibrillation due to the high vagal tone from training. A history, physical examination, ECG, echocardiogram, thyroid function tests, and tests for illicit
drug use should be completed. The maximal exertional rate of the atrial fibrillation or flutter should be assessed with an exercise tolerance test. An ambulatory monitor to assess maximal and minimal rates and the presence of any ventricular arrhythmias should also be performed. Options for therapy include rhythm control by reestablishing and maintaining normal sinus rhythm or rate control during atrial fibrillation. Rhythm control has the advantage of avoiding anticoagulation but the potential disadvantage of risks and cost of the antiarrhythmic drug therapy (45,46). Athletes with structural heart disease or other risk factors for embolic events may require chronic anticoagulation with warfarin sodium, and would then be prohibited from any sport having a risk of body collision.

**Ventricular Arrhythmias**

**Premature ventricular contractions.** These are common in athletes. Fortunately, premature ventricular contractions rarely cause symptoms that necessitate therapy. Without congenital or acquired structural heart disease, there is no increased risk of life-threatening cardiac arrhythmias. Athletes with premature ventricular contractions should have a history, physical examination, ECG, and echocardiogram. Further evaluation is needed for those with congenital heart disease such as long-QT syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, or anomalous origin of the coronary arteries—or with acquired heart disease such as coronary artery disease or dilated cardiomyopathy (46-57).

In the absence of heart disease, therapy with beta-blockers may decrease symptoms with or without reducing premature ventricular contraction. Other antiarrhythmic agents are not recommended unless the symptoms are severe and persist despite beta-blocker therapy. When the premature ventricular contractions do not cause symptoms with exertion or worsen with exercise, no athletic restrictions are needed in the absence of structural heart disease (41,42).

**Nonsustained ventricular tachycardia.** This is defined as ventricular tachycardia less than 30 seconds in duration and, unless structural heart disease is present, carries no additional risk of sudden cardiac death (42,56-58). Management is similar to that for 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 should be considered (58).

**Sustained ventricular tachycardia.** This condition or prior episodes of ventricular fibrillation require a comprehensive evaluation of cardiac status including selective use of the stress test, cardiac magnetic resonance imaging, cardiac catheterization, and electrophysiologic evaluation (41,42,46,47). In the absence of any identifiable structural heart disease, sustained ventricular tachycardia can originate from the right ventricular outflow tract or other regions of the right or left ventricle (idiopathic ventricular tachycardia). Programmed ventricular stimulation supplemented by isoproterenol infusion can usually induce the arrhythmia for mapping. When cured by radiofrequency ablation, these athletes can return to competition in 3 months (41,42). Sustained ventricular tachycardia or ventricular fibrillation usually occurs in patients who have structural heart disease. In athletes younger than 35, congenital abnormalities like hypertrophic cardiomyopathy most commonly predispose them to sudden cardiac death (47-58). Since a risk of arrhythmia recurrence exists, antiarrhythmic therapy or an implantable cardioverter defibrillator (ICD) should be used. In patients with an anomalous coronary
artery, competitive athletics can be resumed 6 months after bypass surgery (41). In athletes older than 35, coronary artery disease is the most common cause of sudden cardiac death. An ICD confers superior protection over drug therapy against sudden death in this group. Patients with ICDs are generally prohibited by current guidelines from competitive athletics.

**Exercise.** In many cardiovascular conditions that predispose to sudden death, exercise may trigger the arrhythmia (42,56,57). In both right ventricular outflow tract ventricular tachycardia and in the sustained monomorphic ventricular tachycardia associated with right ventricular dysplasia, arrhythmias are commonly exercise induced (42). Exercise can also trigger sudden death in patients with congenital coronary artery abnormalities (56). Approximately 15% of those with idiopathic ventricular fibrillation have cardiac arrest during exercise (46,59,60). In hypertrophic cardiomyopathy—the most common underlying structural heart disease associated with sudden cardiac death in the young—arrhythmias are exertionally induced in most patients. Restriction of athletic activity for these patients has been a successful strategy for preventing sudden death (61).

Sudden death in patients with long-QT syndrome is generally exertional or immediately postexertional (62). Beta-blockers have been shown to decrease the frequency of syncope and sudden death in this group, though ICDs provide better protection against sudden cardiac death. Restriction from athletic activities is recommended alongside therapy (41).

**Implantable cardioverter defibrillators.** No one knows how well ICDs can terminate lethal arrhythmias precipitated by vigorous competitive athletics (41). ICDs should not be placed to allow continued sports participation in patients at risk. Generally, for those with ICDs, all moderate- and high-intensity sports are contraindicated (41,42), and low-intensity competitive sports without a significant risk to the ICD should be restricted for at least 6 months after the last episode requiring intervention.

**Parting Comments**

Changes in autonomic tone and chamber hypertrophy that accompany athletic conditioning can cause normal physiologic changes that resemble ECG abnormalities. It is especially important for physicians to recognize on the ECG the specific abnormalities associated with the risk of serious or life-threatening arrhythmias.

**References**

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