ELECTROCARDIOGRAPHIC EVALUATION IN ATHLETES AND USE OF THE SEATTLE CRITERIA TO IMPROVE SPECIFICITY

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Abstract

The screening of athletes for cardiovascular disease prior to participation can be challenging. Sustained training often leads to anatomical changes in the heart that can translate into electrocardiographic (ECG) abnormalities. This can lead to a high false-positive rate that excludes healthy participants or results in costly workups. The Seattle Criteria applied to ECG interpretation has resulted in improved specificity without sacrificing sensitivity. At the Houston Methodist DeBakey Heart & Vascular Center in Houston, we have had much success using this tool to screen thousands of athletes ranging across all age groups and levels of expertise—from the middle school level to the professional athlete.

Introduction

Sudden cardiac death (SCD) on the playing field is a rare event. The incidence is only 1 to 2 in 200,000. This can vary depending on the sport and the person's race and gender. Basketball players have the highest incidence of sudden death in the United States. Males have a higher incidence than females, and black athletes have a higher incidence than non-black athletes.[1] When SCD does occur, however, it can be quite devastating for the family as well as the community. Many cases of SCD involve an underlying, pre-existing cardiac condition. In athletes younger than 35 years old, hypertrophic cardiomyopathy (HCM) accounts for 48% of SCD cases. Anomalous coronary anatomy, arrhythmogenic right ventricular dysplasia (ARVD), dilated cardiomyopathy, and channelopathies such as long QT syndrome make up the remainder of the causes.[2] In patients 35 years and older, coronary heart disease makes up > 80% of the causes of SCD.

The purpose of the preparticipation physical is to catch those at risk for SCD and other catastrophic events before they step onto the playing field or court. A cardiac etiology can be found in 75% of those athletes who die suddenly while exercising. This is made especially difficult with the fact that most of the victims of SCD have no symptoms leading up to the event. Therefore, the cardiac evaluation becomes the most important part of the exam. The current method of screening athletes involves taking a history, often in the form of a questionnaire filled out by the participant, and a cursory physical exam. The family history is perhaps the most important information, although many of the above conditions can go undetected in family members. The dismal fact is that the current screening process catches less than 5% of those who actually go on to have an event.[3]

The electrocardiogram (ECG) has been used to assist in the screening process since a substantial number of athletes with these conditions have abnormal ECGs. For example, 80% to 85% of patients with HCM and > 80% of patients with ARVD have abnormal ECGs.[4] The drawback of ECG analysis, however, is the lack of specificity. The anatomical changes that take place in the heart with athletic training lead to ECG changes that are interpreted as abnormalities, which in turn can lead to the elimination of healthy athletes or to unnecessary costly workups. The following discusses the effects of exercise on cardiac output and function and the role of the Seattle Criteria in improving ECG specificity when screening athletes.

Cardiac Adaptation to Exercise

With training, cardiac output can increase 5- to 6-fold during maximal exertion. Most of this can be explained with an increase in heart rate. However, stroke volume can increase with training as well. This is accomplished by an increase in end-diastolic volume or left ventricular chamber size as well as a sympathetically mediated decrease in end-systolic volume. The type of stress on the heart can determine how the heart adapts. Isotonic or endurance training is a volume load on all four chambers, whereas isometric or strength training increases systemic vascular resistance, which leads to a pressure overload on the heart. We know about these anatomical changes that take place in the heart largely from Pellicia et al., who performed echocardiographic studies on athletes.[5] Left ventricular end-diastolic dimensions varied from 38 mm to 66 mm in women and from 43 mm to 70 mm in men. Left ventricular end-diastolic diameter was > 60 mm in 14% of the cohort. They also reported on left ventricular (LV) wall thickness in these athletes. A substantial number of athletes had LV thickness in the 13-mm to 15-mm range. They all had concomitant LV dilation. These changes have resulted in a new term known as "athletic heart." It can be difficult at times to differentiate athletic heart from early pathologic conditions such as HCM or other malignant illnesses.

The Seattle Criteria

On February 13-14, 2012, a group of sports medicine physicians and cardiologists met in Seattle, Washington, to hold a summit on ECG interpretation in athletes.[6] The challenge was to identify and differentiate between those ECG changes that are adaptive and benign from those that indicate underlying pathology in order to improve the specificity of the ECG when used as a screening tool for athletes. That meeting resulted in the creation of two tables: one listing adaptive and benign training-induced cardiac changes that need no further workup (Table 1), and the other listing abnormalities that could indicate underlying pathology and therefore warrant further evaluation (Table 2).

Sinus bradycardia ≥ 30 bpm
Sinus arrhythmia
Ectopic atrial rhythm
Junctional escape rhythm
First-degree AV block (PR > 200 ms)
Mobitz I second-degree AV block
Incomplete right bundle branch block
Isolated voltage criteria for LVH (absence of left atrial enlargement, left axis deviation, ST depression, T-wave inversion, pathologic Q waves)
Early repolarization (ST elevation with T-wave inversion in V1-V4)

Table 1. Normal electrocardiogram findings in athletes. AV: atrioventricular; LVH: left ventricular hypertrophy.

Some common training-induced abnormalities seen in ECG evaluations of athletes are sinus bradycardia, sinus arrhythmia, first-degree atrioventricular block, early repolarization, incomplete right bundle branch block, and isolated QRS voltage criteria for left ventricular hypertrophy (LVH). The presence of LVH based on the Sokolow-Lyon index can occur in almost half of all athletes, making this an ineffective screening tool. Only 2% to 3% of patients with hypertrophic cardiomyopathy (HCM) meet voltage criteria for LVH as the only ECG finding.[6] If LVH exists with other abnormalities such as left atrial enlargement, left axis deviation, ST depression, or T-wave inversion, then it is considered abnormal. Otherwise, when interpreting an athlete's ECG, an increased QRS amplitude is not a reliable indicator of disease.

Variation in repolarization is perhaps the most common abnormality seen in an athlete's ECG. Diastolic function in endurance athletes is enhanced by increased early diastolic filling,[7] which can be interpreted as early repolarization and therefore perceived as an abnormality when seen on the ECG. This pattern can be seen in 50% to 80% of athletes.[8] There are two main variations in early repolarization. One is upward concavity in the ST segment followed by positive T waves (Figure 1). The other, which is very common in African-Caribbean athletes, is ST elevation with upward concavity followed by negative T waves in V1 through V4 (Figure 2). Both are normal variants and do not warrant further workups. Echo and tissue Doppler have greatly aided in the evaluation of diastolic function and in differentiating normal or enhanced diastolic filling from abnormal or strained diastolic filling. It should be noted, however, that LV strain pattern by echo is never a normal variant.

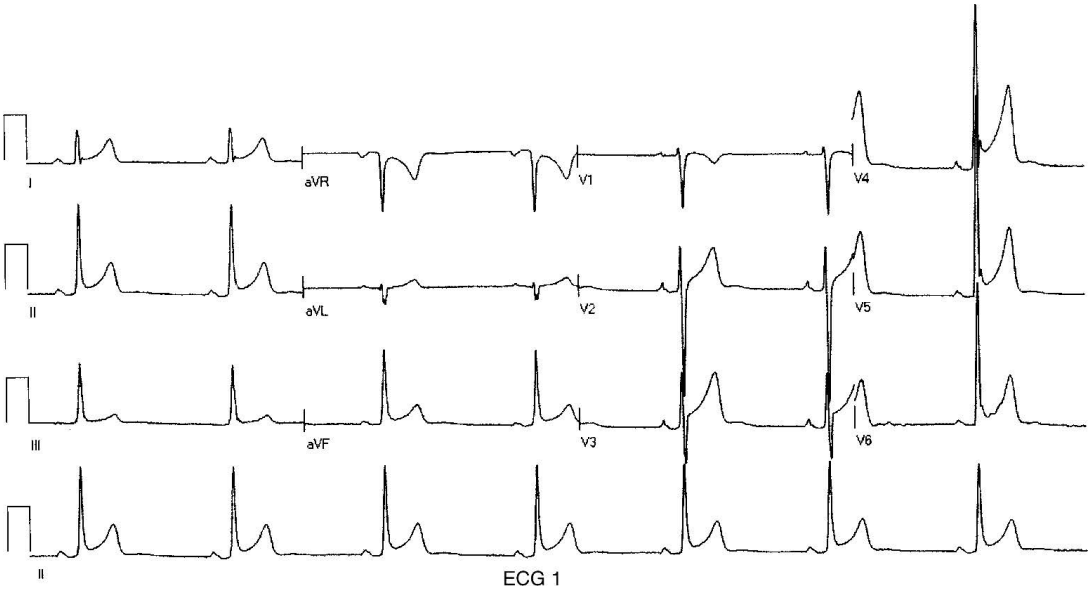
There are abnormalities that do warrant further workup, including T-wave inversion that goes beyond V1 and V2. This is especially the case if there is involvement of the inferior leads excluding lead III. ST depression is never a normal variant and warrants further workup. Pathologic Q waves, which are defined as > 3 mm in depth or > 40 ms in duration with the exception of lead III or aVR, are never normal. Right ventricular hypertrophy based on the Sokolow-Lyon criteria is also never a normal variant and warrants further workup.

Figure 3 is an ECG of a young athlete with HCM. There is deep T-wave inversion anteriorly and in the inferior leads that is very abnormal. Figure 4 is an ECG of a young athlete with HCM presenting with pathologic Q waves. As stated above, pathologic Q waves are never normal in the ECG evaluation of an athlete and definitely warrant further workup. Figure 5 is an ECG of an athlete with ARVD. Notice the T-wave inversions anteriorly and inferiorly.

The Seattle Criteria were tested on 1,078 elite Australian athletes who underwent ECG testing prior to participation. The European Society of Cardiology (ESC) recommendations were initially applied, and according to ESC guidelines, 17.3% had abnormal ECGs. Of those, only three athletes (0.3%) were found to have a cardiac abnormality. When the Seattle Criteria were applied, the number of ECGs classified as abnormal fell to 4.5% ($P < 0.001$). The

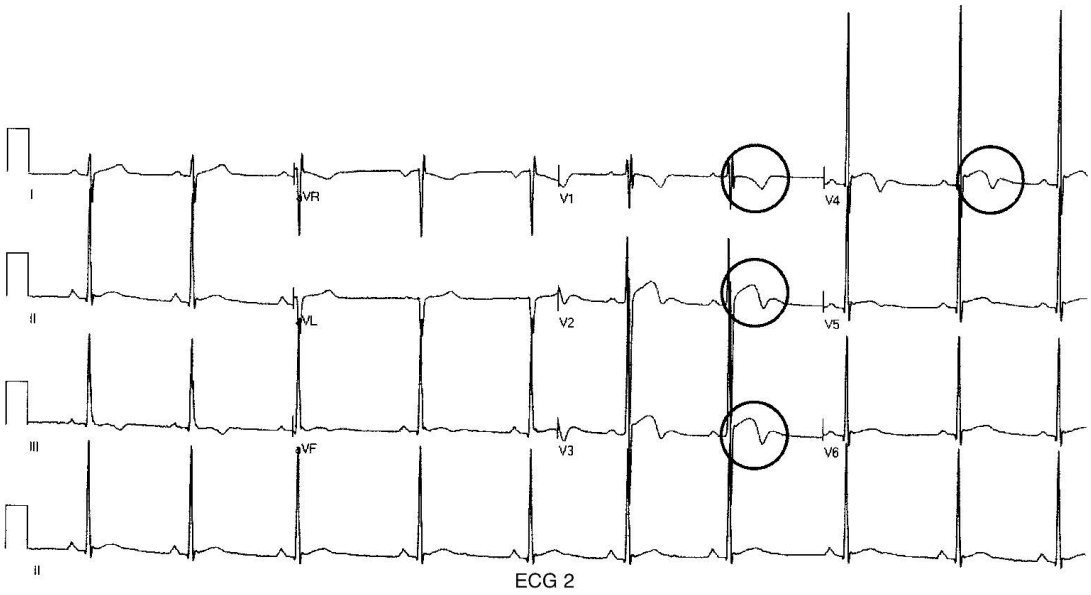
T-wave inversion (in two or more leads V2-V6, II, aVF, or I and aVL
ST depression (≥ 0.5 mm in two or more leads)
Sinus tachycardia
Pathologic Q waves (> 3 mm in depth or > 4 msec in duration in two or more leads except III and aVR)
Left bundle branch block
Left axis deviation (-30° to -90°)
Left atrial enlargement
Right ventricular hypertrophy (RV1 + SV5 > 10.5 mm and right axis deviation)
Ventricular pre-excitation
Brugada-like ECG pattern
Sinus bradycardia < 30 bpm
PVCs (≥ two PVCs per 10-second tracing or nonsustained ventricular tachycardia

Table 2. Abnormal electrocardiogram findings in athletes.



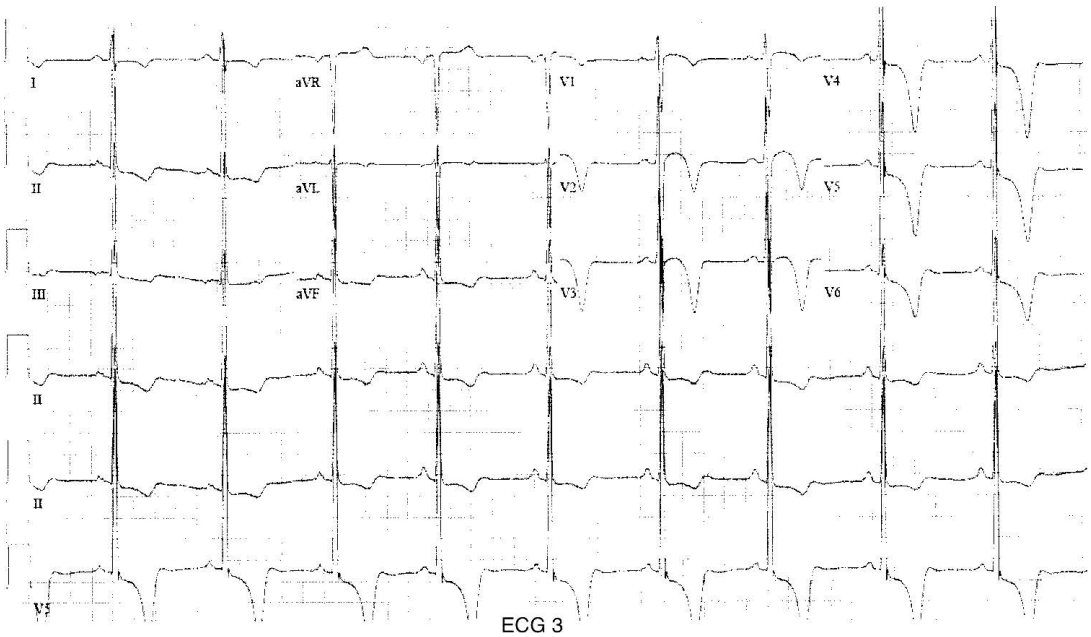
ECG 1

Figure 1. Electrocardiogram showing upward concavity in the ST segment followed by positive T waves.



ECG 2

Figure 2. Electrocardiogram showing ST elevation with upward concavity followed by negative T waves in V1 through V4.



ECG 3

Figure 3. Electrocardiogram of a young athlete with HCM; note the deep T-wave inversion anteriorly and in the inferior leads, which is abnormal.

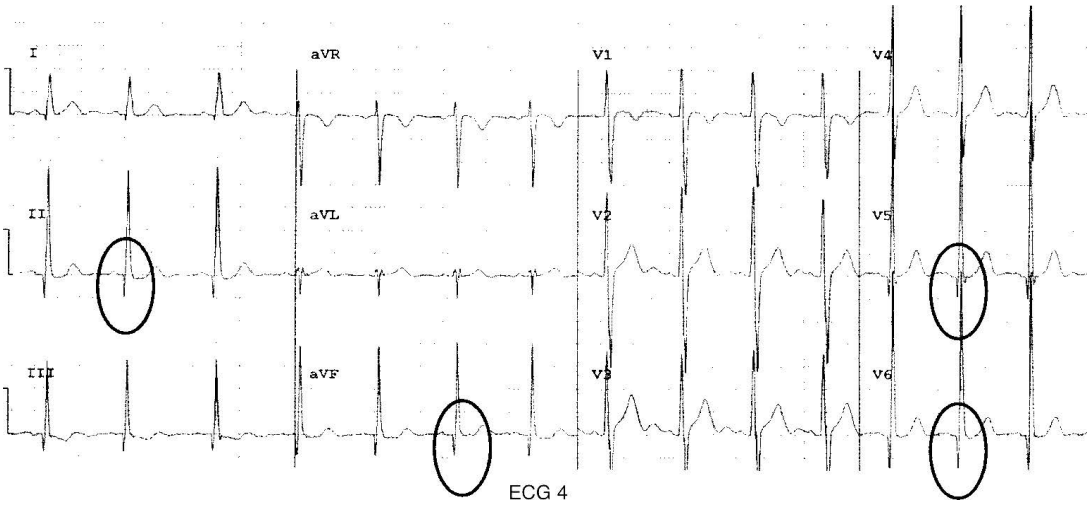


Figure 4. Electrocardiogram of a young athlete with hypertrophic cardiomyopathy presenting with pathologic Q waves.

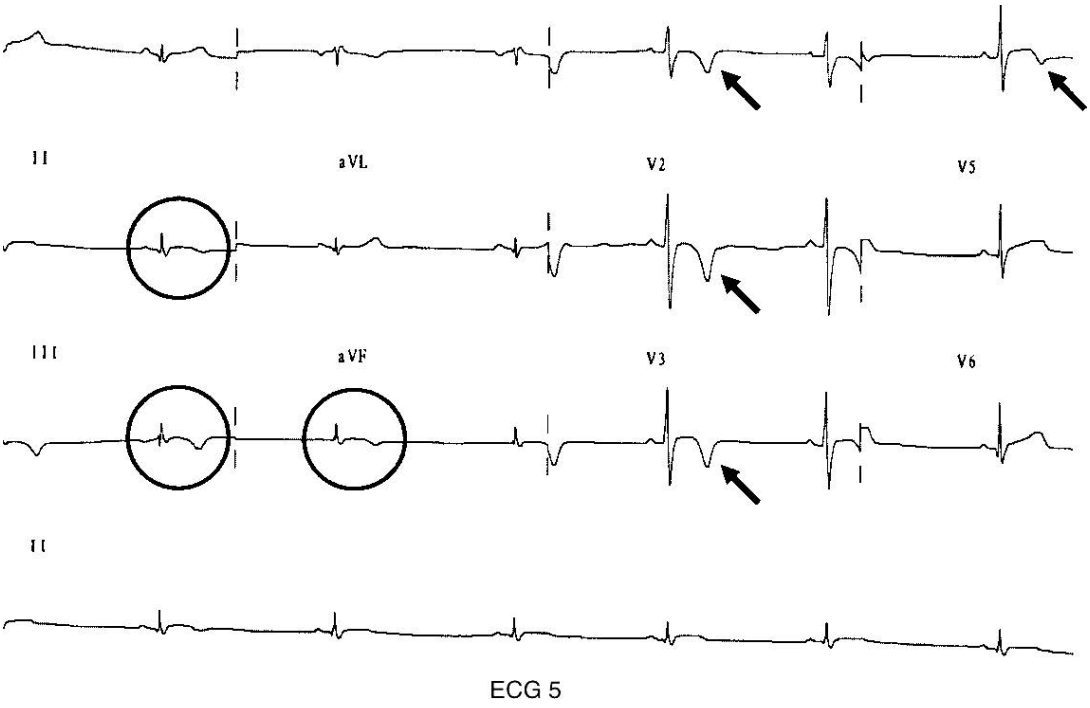


Figure 5. Electrocardiogram of an athlete with arrhythmogenic right ventricular dysplasia.

three with cardiac abnormalities were still identified.[9] The Seattle Criteria reduced the false-positive rate from 17% to 4.2% while still maintaining the sensitivity of the test.

Conclusion

We have screened thousands of athletes at all levels of competitive sports and have had much success applying the Seattle Criteria to our ECG interpretations. The ECG has been a valuable tool in screening athletes for serious cardiovascular diseases prior to participation, and it is important to understand the anatomic and physiological processes that lead to the athletic heart and how they can change an ECG. The advances made in imaging, including echo and tissue Doppler, specifically LV strain analysis, as well as MRI technology has greatly assisted in evaluating those athletes with equivocal ECG findings and in differentiating athletic heart from pathologic conditions.

Key Points:

When reviewing ECGs in athletes:

- T-wave inversion beyond V1, V2, and especially in the inferior leads is never normal.
- ST depression in two or more leads is never normal.
- Isolated voltage criteria for LVH can be normal except when associated with left atrial enlargement, ST depression, or T-wave inversion.
- Pathologic Q waves in two or more leads is never normal.

Conflict of Interest Disclosure: The author has completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords: athletic heart, Seattle Criteria, sudden cardiac death, cardiac adaptation

References

1. Maron BJ, Doerer JJ, Haas ST, Tiemey DM, Mueller FO. Sudden death in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation. 2009 Mar 3:119(8):1088-92.
2. Maron BJ. Sudden death in young athletes. N Engl J Med. 2003 Sep 11;349(11):1064-75.
3. Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. JAMA. 2006 Oct 4;296(13):1593-601.
4. Corrado D, Basso C, Schiavon M, Thiene G. Screening for hypertrophic cardiomyopathy in young athletes. N Engl J Med. 1998 Aug 6;339(6):364-9.
5. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med. 1991 Jan 31;324(5):295-301.
6. Drezner JA, Ackerman MJ, Anderson J, et al. Electrocardiographic interpretation in athletes: the Seattle Criteria. Br J Sports Medicine. 2013 Feb;47(3):122-4.
7. Caso P, D'Andrea A, Galderisi M, et al. Pulsed Doppler tissue imaging in endurance athletes: relation between left ventricular preload and myocardial regional diastolic function. Am J Cardiol. 2000 May 1;85(9):1131-6.
8. Uberoi A, Stein R, Perez MV, et al. Interpretation of the electrocardiogram of young athletes. Circulation. 2011;124:746-57.
9. Brosnan M, La Gerche A, Kalman J, et al. The Seattle Criteria increase the specificity of the preparticipation ECG screening among elite athletes. Br J Sports Med. 2014 Aug;48(15):1144-50.