Electrocardiograms of Collegiate Football Athletes

Address for correspondence:
Stephen F. Crouse, PhD, FACSM
4243 TAMU
Texas A&M University
College Station, Texas 77843, USA
s-crouse@tamu.edu

Stephen F. Crouse, PhD, FACSM,* Thomas Meade, MD,† Brent E. Hansen, MS,*
John S. Green, PhD,* Steven E. Martin, MS*
*Department of Health Kinesiology, Texas A&M University; †Scott and White Clinic, College Station, Texas, USA

ABSTRACT

Background: The prevalence of electrocardiogram (ECG) abnormalities in American collegiate football athletes is virtually unknown.

Purpose: The purpose of this study was to characterize the type and frequency of ECG abnormalities in a sample of football athletes entering National Collegiate Athletic Association (NCAA) Division I Football Bowl Subdivision university program.

Methods: Over a 4-y period, resting and exercise 12-lead ECG recordings were analyzed by a cardiologist from 68 freshmen and 9 transfer football athletes (n = 77; 54 African-Americans and 23 Caucasians, aged 18±1 y, height = 1.89±0.06 m, weight = 104.4±19.8 kg) as part of their entry physical examination.

Results: A total of 79% of the athletes demonstrated at least 1 abnormal ECG finnding, and significantly more African-America athletes (85%) than Caucasian (65%) athletes. Wolff-Parkinson-White (WPW) syndrome was found in 1 African-American player. Frequencies of various ECG abnormal findings in all athletes were: left ventricular hypertrophy = 64.5%, ST-T wave = 6.5%, interventricular conduction delay = 2.6%, sinus bradycardia = 9.1%, sinus arrhythmia = 15.6%, first-degree atrioventricular (AV) block = 11.7%, left atrial enlargement = 48.1%, early repolarization = 33.8%, and right axis deviation = 20.8%. Average values for the PR (0.17±0.03 s), QRS (0.08±0.02 s), and QT intervals (0.38±0.05 s), P-wave duration (0.10±0.02 s), and QRS axis (79.1±18.2 degrees) were normal. The ECG responses to maximal treadmill exercise stress tests were evaluated as normal without ischemia or arrhythmias.

Conclusion: Abnormal resting ECG findings are common in a sample of collegiate football athletes, exceeding the rate expected for their age, and are more frequent in African-American athletes as compared with Caucasian athletes.

Key words: electrocardiogram, exercise testing, sports medicine

Introduction

Athlete's Heart Syndrome has been described as a constellation of cardiac adaptations, including benign hypertrophy specific to training. The increase in left ventricular mass may average 46% above controls, and depends on the frequency, type, intensity, and level of training, as well as on the type of sport in which the athlete competes.[1–5] Correspondingly, the electrocardiogram (ECG) is altered in up to 40% of athletes. Elevated QRS voltage, QRS axis shifts, ST-T wave changes consistent with early repolarization, bradyarrhythmias, first-degree and Mobitz type I atrioventricular (AV) block, and inverted T-waves have been reported with some regularity in athletes. Deeply inverted T-waves have been linked to underlying cardiomyopathies and delayed adverse outcomes.[6] Specific ECG changes and the frequency of abnormal findings are dependent on the sport in which the athlete participates, as well as the nature of their training. More men than women athletes exhibit ECG abnormalities, and data are limited for African-American athletes.[4,7]

Most sport-related ECG research has been conducted on endurance athletes, and rarely have American football athletes been studied. American football players were not included in a recent review on cardiac adaptations in athletes.[4] Only professionals were included as subjects in the 2 ECG studies in football athletes published since 1984, with up to 55% showing at least 1 ECG abnormality.[8,9] To our knowledge, similar ECG studies have not been published for collegiate American football athletes. Thus, the rate of ECG abnormalities in this sizeable population of athletes is virtually unknown. It was our purpose to describe the type and frequency of ECG abnormalities in freshmen and transfer football athletes entering a National Collegiate Athletic Association (NCAA) Division I Football Bowl Subdivision university program.

Methods

From August 2003 to August 2006, we collected resting and exercise ECG data from 68 freshmen and 9 transfer football players (n = 77) who matriculated to our NCAA Division I Football Bowl Subdivision university program. The racial distribution was 54 African-American athletes and 23 Caucasian athletes. With rare exceptions, the players were tested in early August of their first year in the program, most within 1 wk of their screening physical examination and arrival on campus and just prior to fall 2-a-day practices. Most of the players had no formal physical training at an

Received: November 27, 2007
Accepted with revision: January 30, 2008

Published online in Wiley InterScience. (www.interscience.wiley.com)
DOI:10.1002/clc.20452

NCAA Division I university. This study was approved by the university's Institutional Review Board for Research with Human Subjects.

Upon arrival at the laboratory, a supine, resting 12-lead ECG was recorded from each player using the Mason and Likar[10] exercise stress testing lead configuration. Subsequently, the players completed a maximal exercise treadmill test using the standard Bruce protocol, with ECG recordings the last 15 sec of each stage and at maximal exertion. The cardiac axis (AXIS, normal range 0-degree to +90-degree), and the PR, QRS, and QT intervals were measured on the resting ECG by the same trained technician. Each resting ECG was evaluated by the same cardiologist for sinus arrhythmia; bradycardia; tachycardia; ventricular arrhythmias; left atrial enlargement (LAE); right atrial enlargement; right ventricular hypertrophy; voltage criteria for left ventricular hypertrophy (LVH), defined as the greatest measured depth of the S-wave in leads V_1 or V_2 added to the greatest height of the R-wave in leads V_5 or $V_6 \geq 35$ mm;[11], early repolarization; AV block; bundle branch block; interventricular conduction delay (IVCD); and nonspecific ST-segment and T-wave changes (ST-T). The cardiologist also evaluated each exercise ECG for arrhythmias and ST-T deviations from resting. Unless otherwise noted, the ECG interpretation is according to Chou.[12]

Data Analysis

Chi-square analysis was used to test for significance of racial differences in the frequency of selected ECG findings. Student's *t* tests were used to examine racial differences in measured ECG and demographic variables. Alpha was set at 0.05 for all statistical tests.

Results

The demographic characteristics of the athletes are presented in Table 1. Caucasian athletes were on average 3% taller and 12% heavier than their African-American counterparts ($p<0.05$). At least 1 abnormal ECG finding was found in 79% of the players, and more frequently in African-American athletes (85%) than in Caucasian athletes (65%) ($p<0.05$) (Figure 1). African-American players also demonstrated a significantly lower (9%) resting heart rate than did Caucasisa players (Table 2). Average PR, QRS, and QT intervals, P-wave duration, and AXIS were within normal ranges for all athletes (Table 2). The single abnormally prolonged QRS complex found in an African-American black athlete was accompanied by a short PR with a delta wave, thus diagnosed as Wolff-Parkinson-White (WPW) syndrome.

TABLE 1: Demographics of football athletes by race and total

Variable	African-America	Caucasian	Total
Age (y)	18±1	18±2	18±1
	(17–21)	(17–25)	(17–25)
Height (m)	1.87±.06 (1.75–2.01)	1.92±.06 (1.78–2.01)	1.88±.06 (1.75 — 2.01)
Weight (kg)	101.0±16.8 (77.3–145.5)	112.6±24.2 (77.7–154.1)	104.1±20.5 (75.7–154.1)

Data are means±standard deviation (range).

The frequencies of selected ECG abnormalities in our total sample and by race are presented in Table 3. No racial differences in these selected ECG variables were statistically significant. Of note, precordial voltages were relatively high compared with normal (Table 2), and nearly two-thirds of the athletes demonstrated voltage criteria for LVH. Early repolarization was noted in about one-third of the athletes, and LVH together with early repolarization occurred in 22% of the players. No evidence of deeply inverted T-waves suggestive of increased risk for cardiomyopathy[6] were found in any of our football athletes. None of the athletes showed evidence of significant atrial ectopy other than mild sinus arrhythmia, nor was there evidence of ventricular ectopy, bundle branch block, or second-degree or third-degree AV block. Moreover, all ECG responses to maximal treadmill exercise were evaluated as normal without evidence of ST-T wave ischemic changes or arrhythmias.

Discussion

It is well-known that athletes often exhibit cardiac hypertrophic changes in response to physical training, and that these changes appear to be benign.[1,3–5] Moreover, heart dimensions and cardiac function change throughout the training cycle, adapting to the seasonal demands of the specific sport.[2] The surface ECG reflects changes in cardiac morphology, and physically trained men and women often exhibit ECG patterns that would be considered abnormal in the untrained.[4,7] Defining the limits of ECG changes in healthy athletes engaged in various sports provides clinicians with criteria essential for an accurate diagnosis of health and disease.

We have documented in this study that ECG abnormalities are common in collegiate American football athletes. The athletes most likely to be successful in this sport are men of comparatively large body masses who also possess unusually high capabilities for the production of muscular power and speed. Training is naturally designed to improve these correlates of performance, and consists predominately of high intensity resistance training and activities that require short bursts of high-intensity, whole-body movements. As Maron and Pelliccia[4] point out in their thorough literature review, this type of physical training largely produces a pressure overload on the heart. Athletes in sports with similar physical demands, for example, weight lifting and wrestling, show cardiac remodeling characterized by increased left ventricular wall thickness with only small

Published online in Wiley InterScience. (www.interscience.wiley.com)
DOI:10.1002/clc.20452

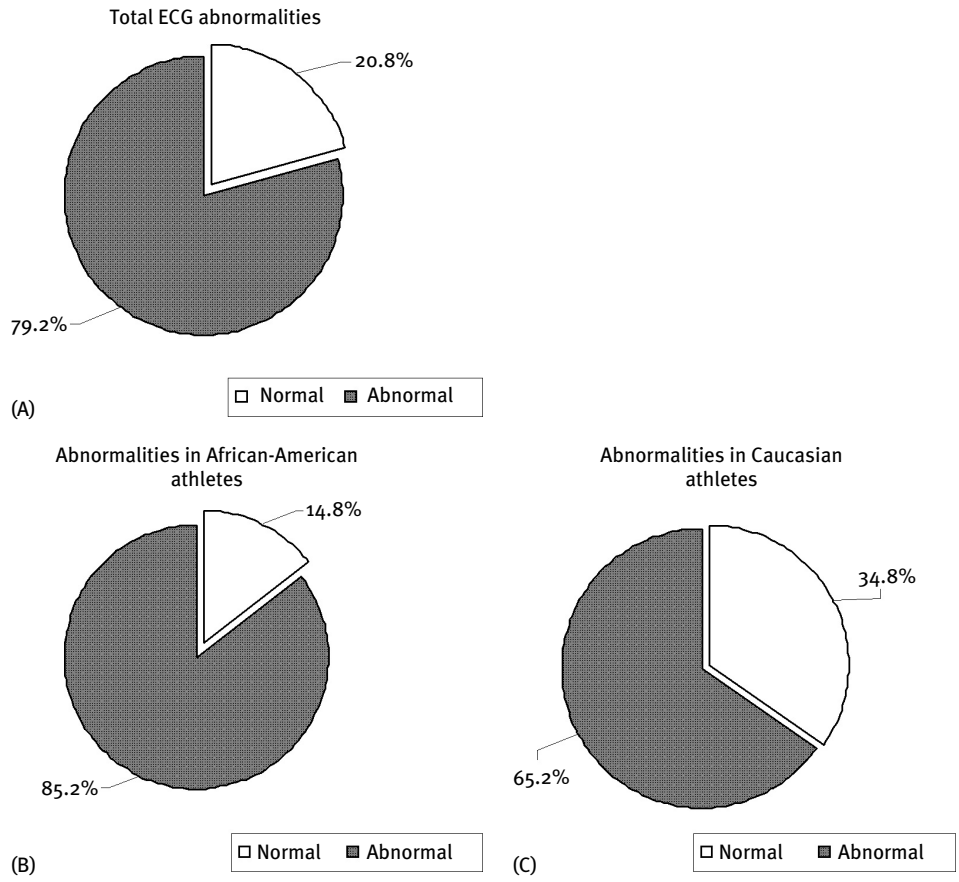

Figure 1: (A) Overall frequency of at least 1 ECG abnormality in all football athletes. (B) Abnormal ECG frequencies in African-American football athletes. (C) Frequency of abnormal ECG findings in Caucasian athletes. The higher frequency in African-American athletes was statistically significant ($p<0.05$).

increases in cavity size. The ECG changes reflect the cardiac remodeling that accompanies physical training.[4,7] Thus, cardiac adaptations and related ECG changes are expected as a consequence of sport-specific training by American football athletes.

A total of 79% of our sample of collegiate football athletes exhibited at least 1 ECG finding considered abnormal in a nonathletic population. A representative ECG recorded from 1 of our subjects, presented in Figure 2, clearly shows evidence of early repolarization and LVH, 2 of the most common abnormalities we documented (Table 3). Even though there was a racial disparity in our subject number, which could have negatively impacted the statistical power of our analysis, ECG abnormalities were found to occur significantly more frequently in African-American athletes (85%) than in Caucasian athletes (65%) ($p<0.05$) (Figure 1). This compares with an abnormal ECG prevalence of 1.6% to 3.4% in the general population of 25–34-y-old men.[13] The abnormal frequency we noted is also considerably higher than the 45% prevalence reported in a large sample of athletes in various sports that did not include American football players.[7] Since our data are descriptive, we cannot confirm that our abnormal ECG findings are caused by football participation, nor can we predict that sport termination would resolve them. However, the magnitude of the abnormal frequency (79%), coupled with the results of other studies in athletes,[2,4,7] supports the speculation that football participation is a contributing causal factor. Others have reported that the cardiac changes in athletes that accompany training are generally benign and are reversible when training ceases.[2,4] In contrast to this conclusion, recent evidence from a large pool of athletes in various sports suggests that deeply inverted T-waves ($\geq$2 mm) in at least 3 ECG leads may be predictive of serious, later-developing cardiomyopathies.[6] None of our football athletes showed this potentially serious abnormality. In light of the high frequency of abnormal resting ECG findings in our study, it is noteworthy that all maximal treadmill exercise stress tests were completed by the athletes without cardiovascular incident, and evaluated as normal with no evidence of ischemia or arrhythmias.

Published online in Wiley InterScience. (www.interscience.wiley.com)
DOI:10.1002/clc.20452

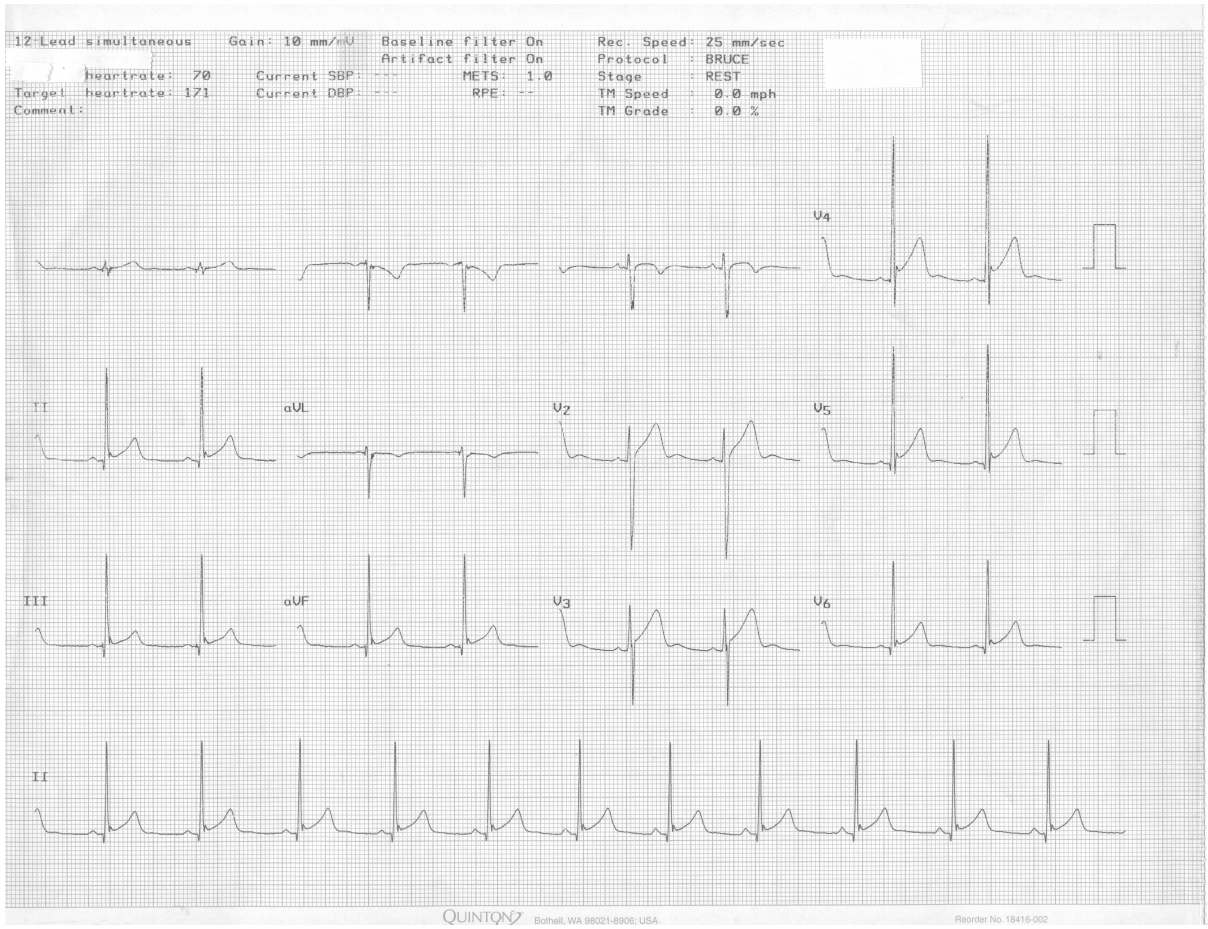

Figure 2: A representative ECG recording from an 18-y-old African-American football athlete in our study showing evidence of early repolarization and voltage criteria for left ventricular hypertrophy.

Our descriptive data also limit us from determining any cause of the racial differences in the frequency ECG abnormalities. In the general population, ECG amplitudes and amplitude combinations consistent with LVH are higher in African-American compared with Caucasians.[14] This suggests that ethnicity may contribute to the frequency of abnormal ECG findings, at least in nonathletic men. Therefore, higher frequencies of ECG abnormalities in African-American athletes in our study might reflect underlying racial differences in cardiac morphology accentuated by physical training. Additional research should address this hypothesis.

The preponderance of abnormal findings in our athletes was consistent with cardiac enlargement. Comparatively, the 64.5% LVH frequency in our study is higher than that reported by others in professional football players, or in the general population.[8,9,13] Choo et al.[9] reported high QRS voltages in professional players, but did not quantify the number of athletes or the average QRS voltage. Our voltage criteria value for LVH[11] averaged 39 mm, similar to the 37 mm reported for professional players.[8] Future studies should employ echocardiography, or some other imaging technology, to verify and characterize the nature of the football-specific cardiac hypertrophy.

Left atrial enlargement was found in 37 (45.1%) of the football athletes in our study (Table 3). This compares with 4% reported in a large sample of athletes from various other sports.[7] The African-American players in our study demonstrated a higher frequency of LAE, ST-T abnormalities, IVCD, first-degree AV block, and early repolarization than Caucasian players, but these differences were not statistically significant. Where comparative data are available, professional football athletes show an increased prevalence of ST-T abnormalities and IVCD, but a lower prevalence of first-degree AV block and early repolarization than our collegiate players; the frequency of right axis

Published online in Wiley InterScience. (www.interscience.wiley.com)
DOI:10.1002/clc.20452

TABLE 2: Means of electrocardiogram characteristics

Variable	African-American	Caucasian	Total
Resting heart rate (bpm)	69±9	76±9*	71±9
	(49–87)	(59–102)	(49–102)
Axis (degrees)	77±20 (8–110)	84±14 (54–113)	79±18 (8–113)
Intervals/ duration (sec)			
PR	0.17±0.03 (0.08–0.24)	0.16±0.02 (0.12–0.22)	0.17±0.03 (0.08–0.24)
QRS	0.08±0.02 (0.05–0.15)	0.09±0.01 (0.07–0.12)	0.08±0.02 (0.05–0.15)
QT	0.38±0.03 (0.30–0.48)	0.38±0.03 (0.31–0.44)	0.38±0.03 (0.30–0.48)
P	0.10±0.02 (0.06–0.14)	0.10±0.02 (0.06–0.12)	0.10±0.02 (0.06–0.14)
Amplitude (mm)			
P	1.5±0.5 (0.5–2.5)	1.7±0.5 (1.0–3.0)	1.6±0.5 (0.5–3)
R_{V1}	3.6±2.2 (0.5–12.2)	2.9±1.3 (1.0–6.0)	3.4±2 (0.5–12.2)
S_{V1-V2}	19.2±6.9 (5.0–36.0)	17.4±6.1 (6.0–30.0)	18.7±6.7 (5.0–36.0)
R_{V5-V6}	20.0±4.5 (12.0–28.0)	20.6±6.3 (9.0–34.0)	20.2±5.1 (9.0–34.0)

Data are means±standard deviation, (range).

TABLE 3: Frequency of ECG Abnormalities

ECG Abnormality	Black	White	Total
LVH	64.2% (34)	65.2% (15)	64.5% (49)
ST-T	7.4% (4)	4.4% (1)	6.5% (5)
IVCD	3.7% (2)	0% (0)	2.6% (2)
Sinus bradycardia	11.1% (6)	4.35% (1)	9.1% (8)
Sinus arrhythmia	14.8% (8)	17.4% (4)	15.6% (12)
First-degree AV block	14.8% (8)	4.4% (1)	11.7% (9)
Early repolarization	38.9% (21)	21.7% (5)	33.8% (26)
Left atrial enlargement	53.7% (29)	34.8% (8)	48.1% (37)
Right axis deviation	18.5% (10)	26.1% (6)	20.8% (16)

Data are given as percent and (number of subjects).

deviation was nearly identical between professional athletes and our collegiate football athletes.[8,9]

Routinely measured ECG wave intervals, durations, and the QRS axis on average were normal in our football athletes (Table 2). Sinus bradycardia was noted in about 9% of our subjects, and resting heart rates were significantly lower in African-American athletes. We measured resting heart rates from ECG recordings from athletes in the supine position 5 min prior to the exercise test. Therefore, we cannot rule out the possibility that anticipation of exercise affected our heart rate data. This may explain our lower rates of sinus bradycardia compared with those in professionals, reportedly ranging from 24.6% to 77%.[8,9] It is also likely that higher rates of sinus bradycardia in professional football athletes is a consequence of their superior cardiovascular fitness compared with our younger collegians.

Although considered a congenital defect, it is of clinical importance that WPW syndrome was diagnosed in 1 of the football athletes in our study. In this relatively small sample of football athletes, we cannot rule out chance in this single occurrence, yet the calculated 1.3% frequency is close to the prevalence rates reported for other athletes,[7] and is considerably higher than the 0.06% frequency in young healthy men.[13] Previous team physical examinations of our athlete with WPW conducted prior to his arrival at our university did not include electrocardiography, and therefore had missed the diagnosis. Follow-up questioning after our diagnosis revealed that this athlete had experienced symptoms consistent with tachycardia during sport participation. He subsequently underwent a successful ablation procedure, then returned to competition without further incident. This underscores the importance of recording the ECG as a part of the athletic participation physical examination to identify preexisting and potentially lethal cardiac conditions.

Conclusions

In summary, we found that resting ECG abnormalities were common in a sample of collegiate football athletes entering an NCAA Division I Football Bowl Subdivision program. African-American athletes showed ECG abnormalities at a significantly greater frequency than did their Caucasian counterparts. The ECG findings in these football athletes are consistent with myocardial adaptations to heavy physical training. Additional longitudinal studies will be required to

Published online in Wiley InterScience. (www.interscience.wiley.com)
DOI:10.1002/clc.20452

determine if participation in this sport causes the ECG changes, or if they will normalize after the athletes are no longer competing.

Acknowledgements

Partial support was provided by the Sydney and J. L. Huffines Institute for Sports Medicine and Human Performance at Texas A&M University (College Station, Tex., USA). The following are acknowledged for their valuable technical support: Grady E. Kaiser, MS, John W. Womack, MS, Ben Pollard, BS, and Greg S. Miller, MS.

References

1. Basilico FC: Cardiovascular disease in athletes. *Am J Sports Med* 1999;27:108–121
2. Crouse SF, Rohack JJ, Jacobsen DJ: Cardiac structure and function in women basketball athletes—seasonal-variation and comparisons with nonathletic controls. *Res Q Exerc Sport* 1992; 63:393–401
3. Maron BJ: Heart disease and other causes of sudden death in young athletes. *Curr Probl Cardiol* 1998;23:477–529
4. Maron BJ, Pelliccia A: The heart of trained athletes: Cardiac remodeling and the risks of sports, including sudden death. *Circulation* 2006;114:1633–1644
5. Morganroth J, Maron BJ: The athlete's heart syndrome: A new perspective. *Ann NY Acad Sci* 1977;301:931–941
6. Pelliccia A, Di Paolo FM, Quattrini FM, Basso C, Culasso F, et al.: Outcomes in athletes with marked ECG repolarization abnormalities. *N Engl J Med* 2008;358:152–161
7. Pelliccia A, Maron BJ, Culasso F, Di Paolo FM, Spataro A, et al.: Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation* 2000;102:278–284
8. Balady GJ, Cadigan JB, Ryan TJ: Electrocardiogram of the athlete: An analysis of 289 professional football players. *Am J Cardiol* 1984;53:1339–1343
9. Choo JK, Abernethy WB III, Hutter JAM: Electrocardiographic observations in professional football players. *Am J Cardiol* 2002;90:198–200
10. Mason RE, Likar I: A new system of multiple-lead exercise electrocardiography. *Am Heart J* 1966;71:196–205
11. Sokolow M, Lyon TP: The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. *Am Heart J* 1949;37:161–186
12. Chou T-C: *Electrocardiography in Clinical Practice*. Philadelphia: W. B. Saunders Company; 1996
13. De Bacquer D, De Backer G, Kornitzer M: Prevalences of ECG findings in large population based samples of men and women. *Heart* 2000;84:625–633
14. Rautaharju PM, Zhou SH, Calhoun HP: Ethnic-differences in ecg amplitudes in north-american white, black, and hispanic men and women—effect of obesity and age. *J Electrocardiol* 1994;27:20–31