Ethnicity and Cardiac Changes in Athletes

Relation of Race to Electrocardiographic Patterns in Elite American Football Players

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Objectives

The purpose of this study was to establish an electrocardiographic (ECG) profile in a biracial population of highly-trained American football players.

Background

Intense physical training can induce cardiac structural and functional changes (“athlete’s heart”), including 12-lead ECG alterations. That race might play a role in determining ECG patterns has been suggested, although not studied in a large athletic population comparing black and white athletes.

Methods

Electrocardiographic analysis of 1,959 elite male athletes attending the National Football League Invitational Camp from 2000 to 2005 was performed. Subjects were classified by race and player position and judged free of structural heart disease.

Results

Abnormal ECG patterns were present in 480 (25%) athletes and were significantly more common among black players (n = 396; 30%) compared with white players (n = 78; 13%) or other races (n = 6; 15%) (p < 0.0001). Distinctly abnormal ECG patterns, suggestive of cardiac disease, were also more common in blacks (n = 76; 6%) than whites (n = 11; 2%) (p = 0.0005). In multivariable analysis, black race was an independent predictor of abnormal ECGs (risk ratio [RR] 2.03, 95% confidence interval [CI] 1.56 to 2.64, p < 0.0001), including patterns judged distinctly abnormal (RR 2.59, 95% CI 1.18 to 5.67, p = 0.02). Abnormal ECGs were also related to player position: most frequent in wide receivers (n = 91; 35%) and least common in quarterbacks (n = 16; 14%) and place kickers (n = 8; 11%). Echocardiograms, obtained in 203 athletes (10%), did not show structural cardiac abnormalities.

Conclusions

Electrocardiographic abnormalities were 2-fold more common in black than in white highly-trained male American football players, with race an independent determinant of ECG pattern. These findings have important implications for pre-participation cardiovascular screening of athletes with ECGs. (J Am Coll Cardiol 2008;51:2250–5) © 2008 by the American College of Cardiology Foundation

Physical training can induce cardiac structural and functional changes including left ventricular (LV) cavity enlargement and modest increases in wall thickness and mass, collectively known as “athlete’s heart” (1–3). In competitive athletes, it is important to distinguish such normal physiological adaptations to training from pathological conditions such as hypertrophic or dilated cardiomyopathy (4). Mass pre-participation screening can identify or raise the suspicion of cardiovascular abnormalities known to cause sudden death in sports participants (5). Incorporation of the 12-lead electrocardiogram (ECG) into screening programs is practiced in Italy, promoted in Europe (4), and suggested in the U.S. (6). However, there are few systematic data assessing ECG patterns in athletes of different races free of cardiovascular disease. This study assessed ECGs in a large biracial cohort of highly-trained American football players.

Methods

Participating subjects. The study group consisted of collegiate football players (n = 1,959) participating in the annual National Football League (NFL) Invitational Camp (i.e., Scouting Combine) from 2000 to 2005. Each...
athlete was eligible to attend only once after entering the NFL draft. The primary objective of this event is to assemble medical information. Evaluations include comprehensive history, physical examination, and 12-lead ECG and also echocardiography selectively when clinically appropriate. All study subjects were judged free of structural heart disease. This study was approved by Saint Luke’s Hospital Institutional Review Board.

**Echocardiography.** Two-dimensional echocardiography was performed with Hewlett Packard Sonos 2500 and 5500 instruments (Andover, Massachusetts). Images were obtained in multiple cross-sectional planes with standard transducer positions. Cardiac dimensions were measured according to American Society of Echocardiography recommendations (8). Parameters of LV filling were obtained with pulsed Doppler echocardiography.

Echocardiograms were obtained selectively in 203 athletes (10%), including 33 with distinctly abnormal ECGs. Athletes were selected for echocardiography by virtue of clinical suspicion of cardiovascular disease on the basis of ECG pattern, heart murmur, or family history. Echocardiograms were interpreted without knowledge of player identity or ECG.

**Statistical analyses.** Variables were assessed with chi-square or Fisher exact tests for categorical variables expressed as proportions and the Student t test or analysis of variance for continuous variables expressed as mean ± SD. Dunnett’s multiple comparison adjustment was used to adjust p values for player position. Independent association of race, player position, body surface area (BSA), and age with abnormal ECG was assessed with multivariable modified Poisson regression models to obtain odds ratios, which were used to estimate adjusted relative risk (9). Covariates in the multivariable model included race, player position, BSA, and age. Statistical significance was defined as a 2-sided p value <0.05. Analyses were performed with SAS 9.1 (SAS Institute, Inc., Cary, North Carolina) and R 2.1.1 (R Foundation for Statistical Computing, Vienna, Austria).

**Table 1** **Criteria for Normal and Abnormal ECGs**

<table>
<thead>
<tr>
<th>Normal</th>
<th>Mildly Abnormal</th>
<th>Distinctly Abnormal</th>
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<tbody>
<tr>
<td>Patterns commonly associated with athlete’s heart syndrome and characterized by ≥1 of the following:</td>
<td>Patterns suggestive of cardiovascular disease and characterized by ≥1 of the following:</td>
<td>Patterns strongly suggestive of cardiovascular disease and characterized by ≥1 of the following:</td>
</tr>
<tr>
<td>1. Increased PR interval duration &gt;0.2 s</td>
<td>1. Increased R- or S-wave voltage (30–34 mm) in any lead</td>
<td>1. Increased R- or S-wave voltage ≥35 mm in any lead</td>
</tr>
<tr>
<td>2. Mild increase in R- or S-wave voltage (25–29 mm)</td>
<td>2. Q waves (2–3 mm) in depth and present in ≥2 leads</td>
<td>2. Q waves ≥4 mm in depth and present in ≥2 leads</td>
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<tr>
<td>3. Early repolarization (ST-segment elevation ≥2 mm in ≥2 leads exclusive of V2 and V3)</td>
<td>3. Repolarization patterns with flat, minimally inverted, or particularly tall (i.e., ≥15 mm) T waves in ≥2 leads exclusive of AVR</td>
<td>3. Repolarization pattern with inverted T-wave &gt;2 mm in ≥2 leads exclusive of AVR</td>
</tr>
<tr>
<td>4. Incomplete RBBB (rSr’ in V1 &lt;0.12 s)</td>
<td>4. Abnormal precordial R-wave progression with R &gt;S-wave only in V2 or V3</td>
<td>4. LBBB</td>
</tr>
<tr>
<td>5. Sinus bradycardia &lt;60 beats/min</td>
<td>5. RBBB (rSr’ ≥0.12 s in lead V1)</td>
<td>5. Marked left (≤ −30°) or right (≥110°) QRS axis deviation</td>
</tr>
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**Abbreviations and Acronyms**

- **BSA** = body surface area
- **CI** = confidence interval
- **ECG** = electrocardiogram
- **LV** = left ventricular
- **NFL** = National Football League
- **RR** = risk ratio

**Results**

**Demographic data.** The mean age of study subjects was 23 ± 0.9 years (range 20 to 29 years); all were male (Table 2). By race, 1,321 (67%) were black, 598 (31%) white, and 40 (2%) of other races. The BSA was 2.4 ± 0.3 m². Abnormal ECGs were identified in 480 (25%) athletes, of which 88 (5%) were regarded as distinctly abnormal and 392 (20%) mildly abnormal (see Fig. 1 for representative examples).

**Race and ECG.** Abnormal ECGs were significantly more common among black players (n = 396; 30%), compared with whites (n = 78; 13%) or other races (n = 6; 15%; p < 0.0001) (Fig. 2). The ECG patterns classified as distinctly abnormal and most suggestive of cardiac disease were also more common in black athletes (n = 76; 6%) than in whites (n = 11; 2%) or other races (n = 1; 3%; p = 0.0005) (Fig. 2).

Specific ECG patterns also showed a relation to race. T-wave inversion and increased R- or S-wave voltages ≥35 mm, usually in precordial leads, were also significantly more common in black players (Fig. 3). All 7 athletes with ≥1 distinctly abnormal ECG finding were black.

**Player position.** Significant associations between player position and abnormal ECG were evident (p < 0.0001) (Table 3). Abnormal ECGs were most common in wide
receivers (n = 91; 35%), defensive backs (n = 100; 30%), and running backs (n = 62; 29%) and least common in
quarterbacks (n = 16; 14%) and place kickers (n = 8; 11%). Black players who were defensive backs, linebackers, offensive
linemen, and quarterbacks more commonly had abnormal ECGs compared with whites at the same positions (p < 0.05) (Fig. 4).

Echocardiograms. Of 203 athletes with echocardiograms, 197 (97%) were judged within normal limits for trained
athletes (1–3). Ventricular septal thickness was 7 to 12 mm (mean 10 ± 1 mm), LV end-diastolic cavity dimension was
40 to 64 mm (mean 54 ± 4 mm), and ejection fraction was 50% to 75% (mean 60 ± 5%). The other 6 athletes (3%) had
septal thicknesses within the ambiguous zone of 13 to 14
mm, which were judged unlikely to represent hypertrophic
cardiomyopathy in these highly-trained athletes in the
absence of other echocardiographic abnormalities and with
normal Doppler inflow velocities (3).

Among 88 athletes with distinctly abnormal ECGs, 33
(38%) had echocardiograms, including only 1 with border-
line septal thickness of 14 mm. Systolic anterior motion of
the mitral valve (or any other feature of hypertrophic
cardiomyopathy), mitral valve prolapse, and evidence of
coronary anomalies of wrong sinus origin were absent in
each of the 203 athletes.

Univariate/multivariable analyses. Unadjusted predictors
of abnormal ECGs appear in Table 3. Compared with
white players, blacks were significantly more likely to have
abnormal ECGs (risk ratio [RR] 2.30, 95% confidence
interval [CI] 1.84 to 2.87, p < 0.0001), whereas athletes
with a larger BSA were less likely to have abnormal ECGs
(RR 0.93, 95% CI 0.90 to 0.96, p < 0.0001). After
adjustment for all other variables in multivariable analysis,
black race was the only independent predictor of an abnor-
mal ECG (RR 2.03, 95% CI 1.56 to 2.64, p < 0.0001),
including patterns regarded as most abnormal (RR 2.59,
95% CI 1.18 to 5.67, p = 0.02) (Table 4).

### Discussion

We evaluated the prevalence of ECG abnormalities in a large
biracial population of healthy elite American football players. Diverse ECG abnormalities were present in 25%, including
5% that were characterized as distinctly abnormal with patterns
likely to raise clinical suspicion of structural heart disease.

We also found that race was associated with different
frequencies of abnormal ECGs. First, a significantly higher
proportion of abnormal ECG patterns were present in black
compared with white athletes (by >2:1). Second, multiva-
riable analysis showed black race to be an independent
predictor of both mildly and markedly abnormal ECGs,
even after differences in ECG pattern by player position
were taken into account. Specific ECG abnormalities most
common in black athletes included increased precordial
voltages and diffuse T-wave inversion.

These findings are consistent with historical assertions
that ECG patterns might differ between healthy black and
white subjects. In the early 1950s, J-point and ST-segment
elevation in the right precordial leads was regarded as
characteristic of young black male individuals without heart
disease (10–12). More recently, several studies have dem-
onstrated a higher prevalence of ECG abnormalities in
young black men (13) or athletes (14) without systemic
hypertension or cardiac disease.

Xie et al. (13) reported substantial differences in preva-
ience of LV hypertrophy by ECG in young healthy black
(16 of 1,000) compared with white men (2.4 of 1,000).
Likewise, in a healthy, middle-age, biracial population,
Vitelli et al. (15) reported ECG abnormalities in 8% of black compared with 5% of white men. Finally, in a study population similar to ours, Choo et al. (14) found ST-T abnormalities more commonly in black compared with white football players. These prior observations and the present findings indicate that race is an important independent predictor of abnormal ECG patterns.

These observations have important implications for preparticipation screening strategies in populations of competitive athletes (5). For example, European investigators (4) and the International Olympic Committee (16) have recommended screening programs routinely incorporating 12-lead ECGs, a process employed in Italy over the last 25 years (17). Electrocardiograms are now commonly obtained in U.S. professional athletes (18), particularly those in football and basketball. Therefore, such ECG-based screening programs and those proposed for U.S. high school and college-age athletes (6) could expose black athletes to false positive screening, in which abnormal ECGs unnecessarily raise the suspicion of cardiac disease.

Prevalence of abnormal ECGs differed among the various positions played in professional football, even though all participants were highly-trained athletes involved in competitive sports for much of their lives. For example, abnormal 12-lead electrocardiographic (ECG) patterns in 5 black athletes characterized by (A) ST-segment depression in inferior leads, J-point elevation in right precordial leads, and diffuse T-wave inversions in a 21-year-old lineman; (B) deep symmetric T-wave inversions in V1 to V3 in a 21-year-old defensive back; (C) J-point elevation in right precordial leads, deep symmetric T-wave inversions in V3 to V6, and inferior T-wave inversions in a 22-year-old defensive back; (D) markedly increased voltage (>40 mm in V4) in a 21-year-old wide receiver; and (E) marked increased voltage (>40 mm in V4), J-point elevation in V2 to V3, and T-wave inversion in V2 to V4 in a 21-year-old wide receiver.
mal ECGs were more common at positions requiring sprinting and similar bursts of physical activity associated with abrupt elevations in heart rate (i.e., wide receivers, running backs, and defensive backs) but less common at positions with lower levels of exertion (e.g., linemen and place kickers). However, player position was not a strong multivariable determinant of ECG patterns.

Echocardiographic data available in a relatively small subset of athletes offer a measure of substantiation that altered ECG patterns reported here are not manifestations of cardiac disease, and differences identified between ECGs obtained in white and black athletes are based predominantly on race.

Conclusions

In conclusion, ECG abnormalities were present in 25% of highly-trained professional football players and were 2-fold more common in black compared with white athletes. Furthermore, black race proved to be the only multivariable predictor of an abnormal ECG. These data have important implications for cardiovascular screening programs routinely incorporating ECGs, suggesting the potential for a high rate of false positive results in black athletes.

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REFERENCES


