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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

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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

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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.

ECGs. Twelve-lead ECGs were recorded at 25 mm/s in the supine position during quiet respiration and interpreted by 1 investigator (A.M.) blinded to identity, race, and position. The ECGs were classified into subgroups as described by Pelliccia et al. (7) (Table 1).

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. Dichotomous outcomes were analyzed with logistic or 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).

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 m^2. 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

Table 1 Criteria for Normal and Abnormal ECGs

Normal	Mildly Abnormal	Distinctly Abnormal
Patterns commonly associated with athlete's heart syndrome and characterized by ≥1 of the following: 1. Increased PR interval duration >0.2 s 2. Mild increase in R- or S-wave voltage (25–29 mm) 3. Early repolarization (ST-segment elevation ≥2 mm in >2 leads exclusive of V_2 and V_3) 4. Incomplete RBBB (rsR' in V_1 <0.12 s) 5. Sinus bradycardia <60 beats/min	Patterns suggestive of cardiovascular disease and characterized by ≥1 of the following: 1. Increased R- or S-wave voltage (30–34 mm) in any lead 2. Q waves (2–3 mm) in depth and present in ≥2 leads 3. Repolarization patterns with flat, minimally inverted, or particularly tall (i.e., ≥15 mm) T waves in ≥2 leads exclusive of AVR 4. Abnormal precordial R-wave progression with R >S-wave only in V_5–V_6 5. RBBB (rsR' ≥0.12 s in lead V_1) 6. Right atrial enlargement (peaked p waves ≥2.5 mm in leads II, III, or V_1) 7. Left atrial enlargement (prolonged positive p wave in lead II and/or deep prolonged negative p wave in V_1) 8. Short PR interval ≤0.12 s	Patterns strongly suggestive of cardiovascular disease and characterized by ≥1 of the following: 1. Increased R- or S-wave voltage ≥35 mm in any lead 2. Q waves ≥4 mm in depth and present in ≥2 leads 3. Repolarization pattern with inverted T-wave >2 mm in ≥2 leads exclusive of AVR 4. LBBB 5. Marked left (≤−30°) or right (≥110°) QRS axis deviation 6. Wolff-Parkinson-White pattern 7. Atrial fibrillation

LBBB = left bundle branch block; RBBB = right bundle branch block.