

The Impact of ST Elevation on Athletic Screening

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INTRODUCTION

With the electrocardiogram (ECG) becoming an accepted part of preparticipation examination (PPE) for athletes, it is important to discern the ECG patterns of clinical pathology from normal variants.¹ ST elevation (STE) is a common finding among athletes and has multiple reasons for generating concern. Although noted as an occurrence in healthy individuals for more than 60 years, the benign nature of STE, and specifically early repolarization (ER), has been challenged by recent studies.^{2,3} In an asymptomatic individual, STE typically occurs in 2 patterns: (1) anterior (V₁, V₂, and V₃) lead STE, which can be part of the Brugada syndrome, and (2) inferior (II, III, and aVF) or lateral (V₄, V₅, V₆, and aVL) lead STE, which is traditionally referred to as ER.⁴ Although common among young athletes, STE can raise clinical concern because it occurs with acute myocardial infarction or injury, angina, pericarditis, and the Brugada syndrome.

Early repolarization has become controversial with new evidence, suggesting that its definition and clinical implications should be reconsidered.⁵ It was originally defined as an elevation of the ST segment from baseline exceeding 0.1 mV (1 mm), occurring in the lateral and inferior leads, usually accompanied with J waves or QRS slurring and tall T waves. In a seminal study, Haissaguerre et al² noted that in patients with idiopathic ventricular tachycardia/ventricular fibrillation, J waves and QRS notching or slurring were more common than those in age-matched controls. Subsequently, in population studies using the same criteria, Tikkanen et al³ and Sinner et al⁶ demonstrated a small but a significant risk for cardiovascular mortality. Based on their cellular physiology experiments and these 3 clinical studies, Antzelevitch and Yan⁵ have proposed including ER in the J wave syndrome along with ischemia, Osborn waves, and the Brugada syndrome.

Another problem encountered when using the ECG for screening is the inconsistent criteria used to define STE. Although STE in 2 adjacent leads is part of the guidelines for thrombolysis for acute myocardial infarction, it is remnant from the early days of electrocardiography when each lead was only viewed for several seconds and when respiratory variation and noise caused much inaccuracy. Currently, most physicians use computerized systems that average 10 seconds of data from all leads, making a finding in 1 lead more representative of the actual ST level in an area.

These controversies have complicated the addition of the ECG to the PPE of athletes because considering STE as abnormal could greatly increase the burden of false-positives.⁷ The aim of this article is to demonstrate the prevalence and

Objective: To demonstrate the prevalence and patterns of ST elevation (STE) in ambulatory individuals and athletes and compare the clinical outcomes.

Design: Retrospective cohort study. ST elevation was measured by computer algorithm and defined as ≥ 0.1 mV at the end of the QRS complex. Elevation was confirmed, and J waves and slurring were coded visually.

Setting: Veterans Affairs Palo Alto Health Care System and Stanford University varsity athlete screening evaluation.

Patients: Overall, 45 829 electrocardiograms (ECGs) were obtained from the clinical patient cohort and 658 ECGs from athletes. We excluded inpatients and those with ECG abnormalities, leaving 20 901 outpatients and 641 athletes.

Interventions: Electrocardiogram evaluation and follow-up for vital status.

Main Outcome Measures: All-cause and cardiovascular mortality and cardiac events.

Results: ST elevation in the anterior and lateral leads was more prevalent in men and in African Americans and inversely related to age and resting heart rate. Athletes had a higher prevalence of early repolarization even when matched for age and gender with nonathletes. ST elevation greater than 0.2 mV (2 mm) was very unusual. ST elevation was not associated with cardiac death in the clinical population or with cardiac events or abnormal test results in the athletes.

Conclusions: Early repolarization is not associated with cardiac death and has patterns that help distinguish it from STE associated with cardiac conditions, such as myocardial ischemia or injury, pericarditis, and the Brugada syndrome.

Key Words: electrocardiography, screening, ST elevation, preparticipation examination, cardiovascular death

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patterns of STE in ambulatory patients and collegiate athletes to help discriminate normal variants from STE associated with cardiac disease.

METHODS

Clinical Population

We performed a retrospective study of 45 829 ECGs from March 1987 to December 1999 at the Veterans Affairs Palo Alto Health Care System. Since 1987, the Veterans Affairs Palo Alto Health Care System has used a centralized computerized ECG system for collection, storage, and analysis of ECGs. Electrocardiograms were ordered by health care providers for clinical indications that included screening when first initiating care at the Veterans Affairs. We excluded ECGs with inpatient status (n = 12 319) and abnormalities, including atrial fibrillation or flutter, ventricular rates greater than 100 beats per minute (bpm), QRS durations greater than 120 milliseconds, paced rhythms, ventricular preexcitation, Q waves, ST depression, or T-wave inversion, leaving 20 901 patients (87% men, 14% African American; age, 53 ± 14 years). Race was determined by self-report at the time of ECG acquisition.

TABLE 1. Demographics in Athletes and Veteran Population With Normal ECGs

Variables	Athletes (n = 641)		Clinical Cohort (n = 21 485)	
	Males (n = 343, 54%)	Females (n = 298)	Males (n = 18 738, 87%)	Females (n = 2747)
Age, mean ± SD, y	20 ± 1.6	19 ± 1.3	52 ± 14	54 ± 17
Race, n (%)				
Whites/others	309 (87)	282 (94)	16 069 (86)	2488 (91)
African Americans	48 (13)	19 (6)	2669 (14)	259 (9)
Weight, mean ± SD, kg	85.7 ± 17.2	64 ± 9.5	84.4 ± 17.2	68 ± 15.4
Height, mean ± SD, m	1.85 ± 0.1	1.7 ± 0.1	1.75 ± 0.1	1.63 ± 0.1
BMI, mean ± SD, kg/m ²	25 ± 4	22 ± 3	27 ± 5	26 ± 6
Heart rate, mean ± SD, bpm	59 ± 10	60 ± 12	70 ± 12	69 ± 11

The athletes and clinical cohort differed significantly by age and gender (*P* < 0.01), with the clinical cohort being older and predominantly consisted of men. BMI, body mass index.

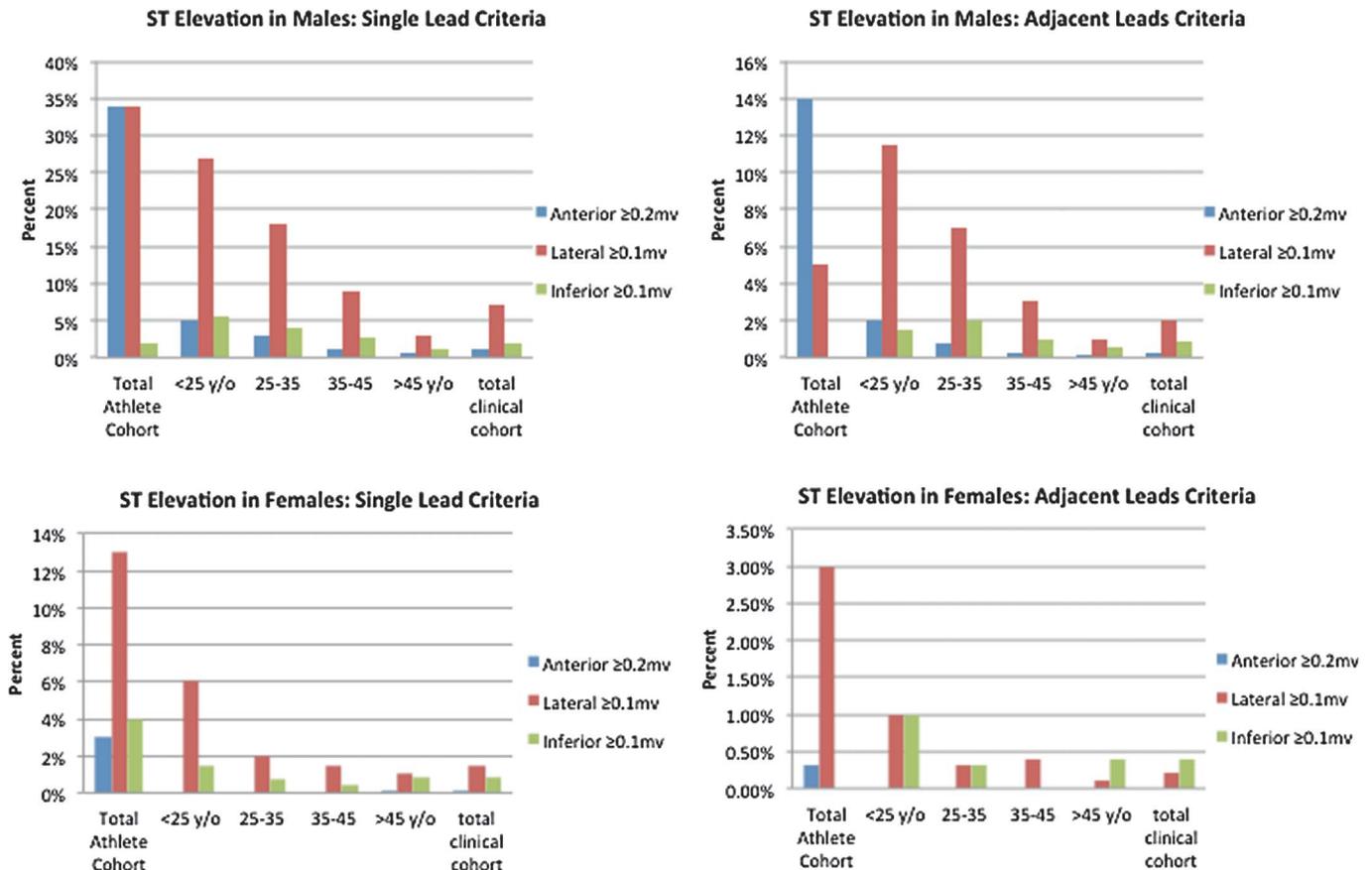


FIGURE 1. Overall percentage of STE with a normal ECG in the athletes by gender and in the clinical cohort by age and gender using the single lead/adjacent leads and the 0.2-mV criteria in the anterior leads (V₁, V₂, and V₃) and the 0.1-mV criteria in both the lateral (V₄, V₅, V₆, and aVL) and the inferior leads (II, III, and aVF).

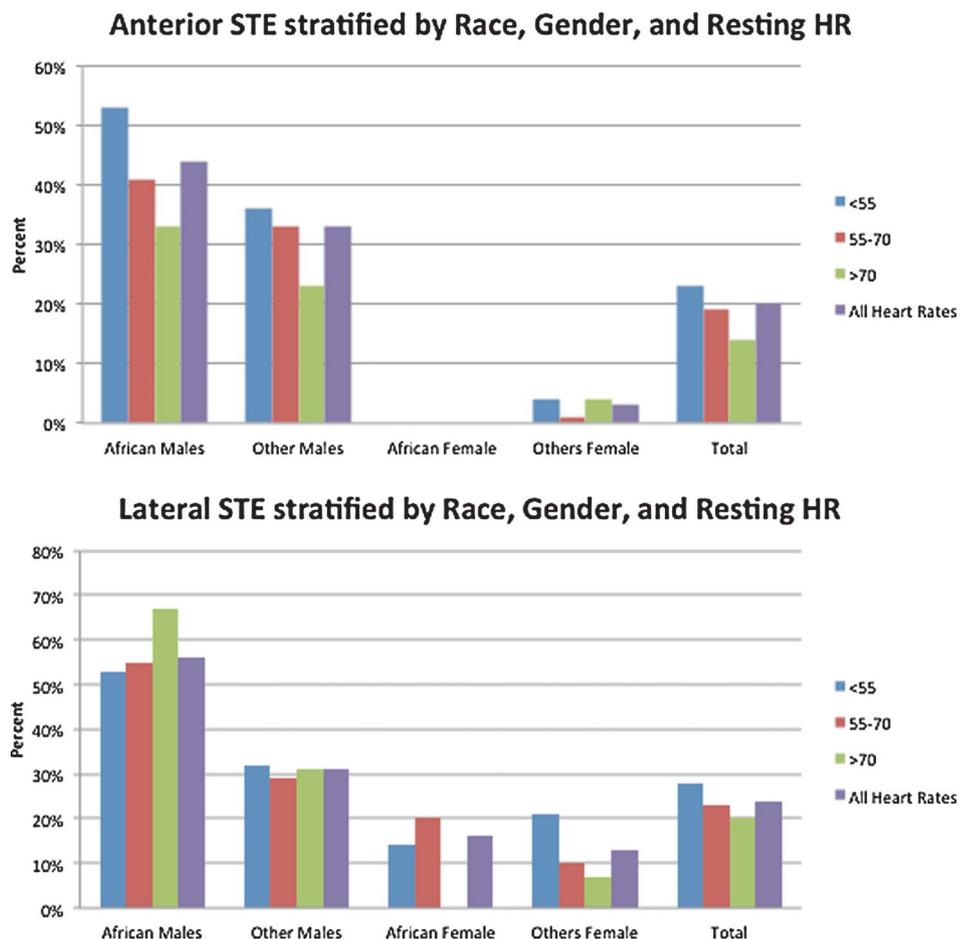


FIGURE 2. The prevalence of STE among athletes by resting HR, gender, and ethnicity using the single-lead criteria.

Athlete Population

During the PPE for the Stanford University varsity athletes in 2007, computerized 12-lead ECGs (Schiller AG, Baar, Switzerland) were obtained from 658 athletes. After excluding athletes with ECG abnormalities, our study included 641 athletes (350 men and 291 women, 10% African American; mean age, 19.5 ± 1.5 years). Complete description of this population has been previously reported.⁸

Clinical Electrocardiogram Interpretation

All ECGs with measurements by the 12-SL software (General Electric Healthcare, Wauwatosa, Wisconsin) consistent with STE were over read by 3 cardiologists and corrected when necessary. ST level was measured at the J point (QRS end, ST beginning). ST elevation was considered 0.1 mV or more of elevation at the end of the QRS complex. The criterion requiring 2 adjacent leads in an area lead group (inferior: II, III, and aVF; lateral: V₄, V₅, V₆, and aVL; anterior: V₁, V₂, and V₃) was applied, as well as a single lead in any distribution.

Athlete Electrocardiogram Interpretation

Electrocardiograms were recorded using Schiller ECG machines, and the digital recordings were entered into a database (SEMA; Schiller AG). Similar steps were taken as

described above, including physician over read. To validate the correlation of the Schiller and GE ECG-processing programs (General Electric, Milwaukee, Wisconsin), more than 1000 ECGs were processed with both the programs and by an independent program with onset/offset points of the QRS assessed for timing and amplitude, and no difference was demonstrated ($R > 0.95$).

Outcomes

The primary outcome variable was time to cardiovascular mortality. The California Health Department Service and Social Security Death Indices were used to ascertain the vital status of each patient as on December 31, 2002. Accuracy of causes of deaths was confirmed using the Veterans Affairs computerized medical records by 2 physicians blinded to the ECG results. There were no cardiovascular end points or abnormal test results in the athletes after 3 years of follow-up.

Statistical Analysis

NCSS software 2007 (NCSS, Kaysville, Utah) was used for all statistical analyses. Unpaired *t* tests were used for comparisons of continuous variables, and χ^2 tests were used to compare dichotomous and categorical variables. In the clinical population, survival analysis was performed using the method of Kaplan and Meier, and Cox proportional

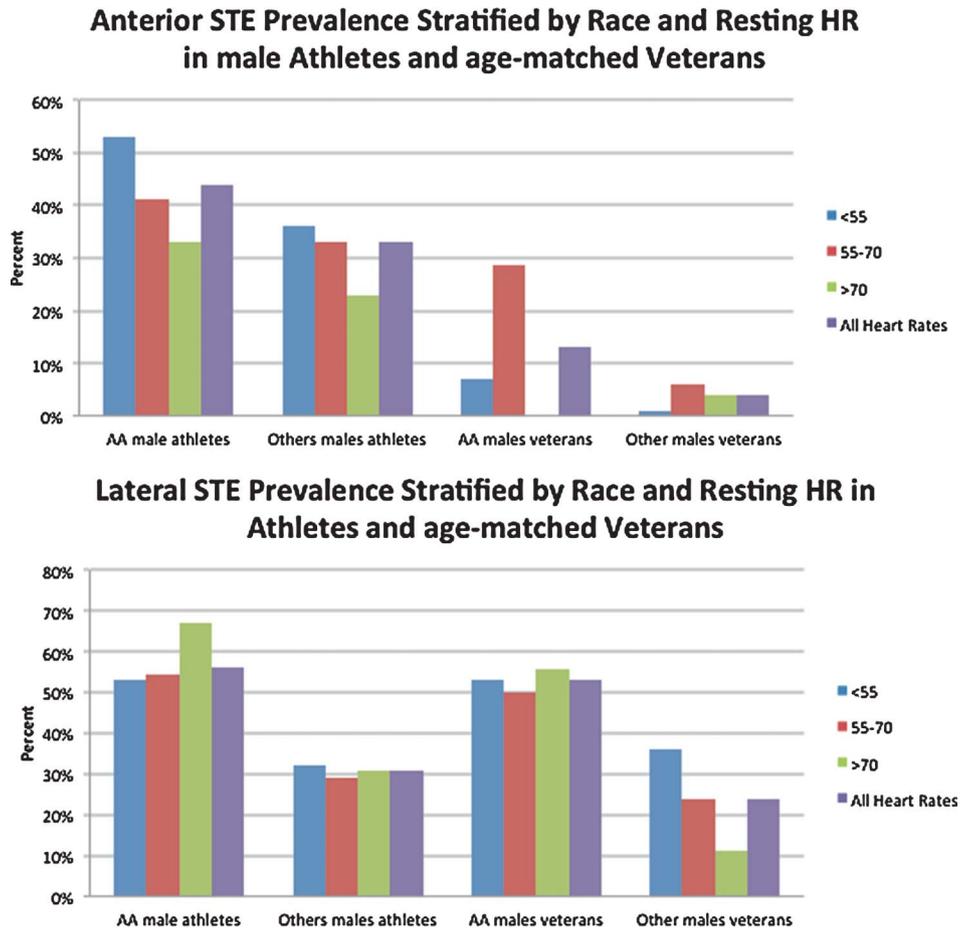


FIGURE 3. The prevalence of STE among male athletes and male veterans younger than 25 years stratified by HR and ethnicity using the single-lead criteria.

hazard ratios were used to determine associations between ECG patterns and cardiovascular death.

Ethical Considerations

All athletes signed the Stanford University Institutional Review Board–approved consent form, and the use of the patient database and follow-up was approved by the Stanford University Human Subjects Committee. All data were deidentified and encrypted per the requirements of the Health Insurance Portability and Accountability Act.

RESULTS

Table 1 summarizes the baseline demographics of the 2 cohorts; the athletes were younger and nearly evenly divided by gender, whereas the clinical cohort was older and predominantly consisted of men (as would be expected with a population from the Veteran’s Administration). There was a similar breakdown in race for men (athletes 13% vs cohort 14%); however, African American women were underrepresented in both the populations.

The prevalence of STE is summarized in Figure 1 using the anterior STE criteria of ≥ 0.2 mV and lateral or inferior lead criteria of ≥ 0.1 mV for both the single lead in an area (single-lead criteria) and the adjacent leads in an area (adjacent-lead criteria). J waves or slurring did not accompany STE in anterior

leads but accompanied STE laterally and inferiorly in 75% of those with STE, with some degree of slurring in the remainder. Lateral and inferior STE of ≥ 0.2 mV are not shown here because there were no instances of STE ≥ 0.2 mV in the inferior leads and it occurred in less than 0.5% of the ECGs in the lateral leads. ST elevation is 2 to 4 times more prevalent using the single-lead criteria compared with the adjacent-lead criteria, so the following figures will only show the results for the single-lead criteria. As shown, the prevalence of STE decreases with age, is more prevalent in athletes compared with age-matched outpatients, and is 2 to 10 times more prevalent in men.

Figure 2 summarizes the prevalence of STE by single-lead criteria in athletes by gender, ethnicity, and resting heart rate. Inferior STE of ≥ 0.1 mV is not presented because it was the least common STE pattern in athletes and occurred only using the single-lead criteria. It was the only pattern more common in female than in male athletes (4% vs 2%; $P < 0.01$). Notably, women have a lower prevalence of STE ($P < 0.01$ for either area). African Americans have a greater prevalence of STE, with less effect from resting heart rate.

Figure 3 summarizes the prevalence of STE by single-lead criteria in the male athletes and that of age-matched clinical cohort by ethnicity and resting heart rate. The prevalence of STE in the anterior leads is significantly greater in the athletes than in the clinical cohort ($P < 0.01$), whereas it is similar in the lateral leads. Resting heart rate seems to have

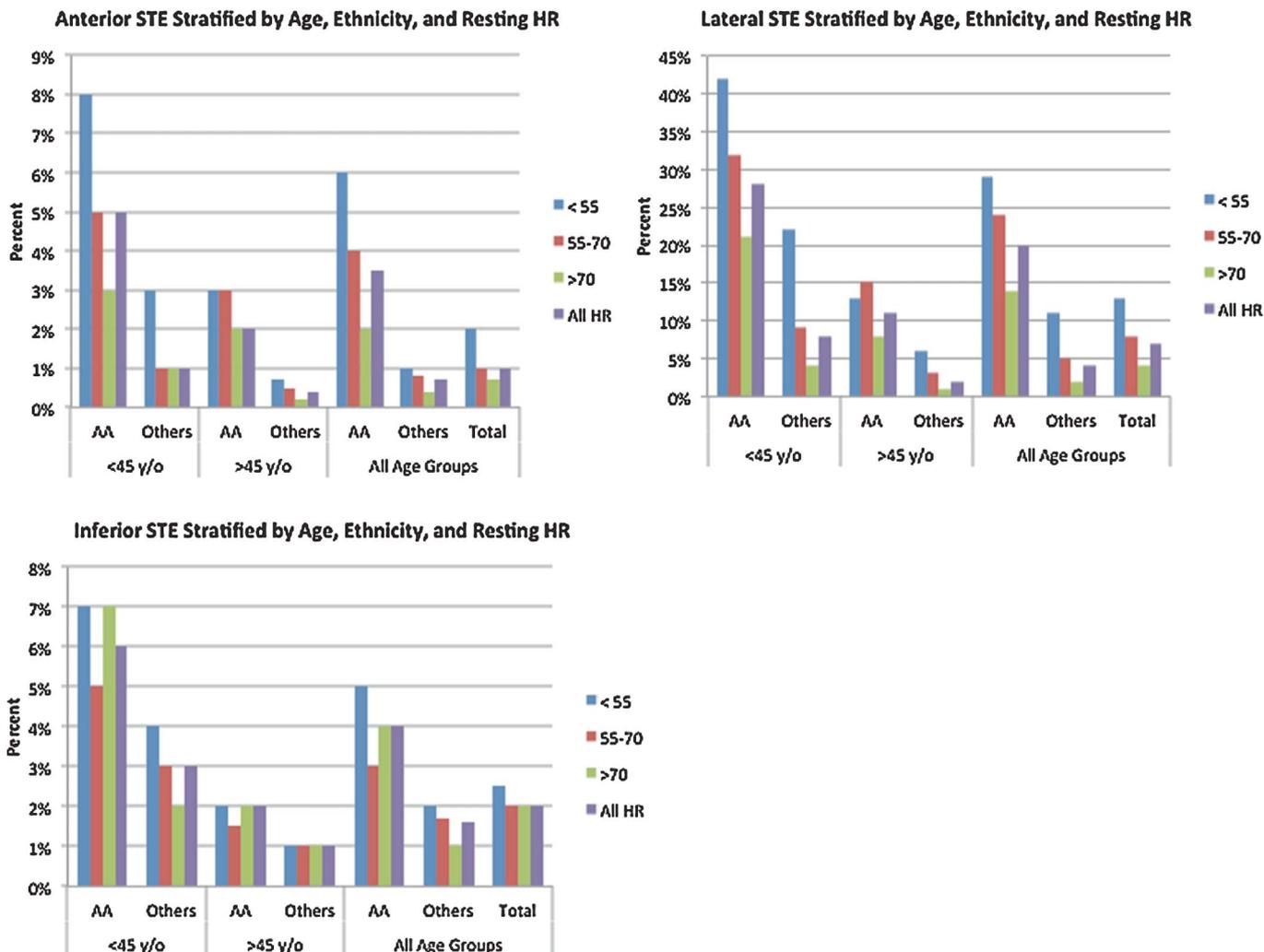


FIGURE 4. The prevalence of STE among male veteran clinical cohort by HR and ethnicity using the single-lead criteria.

less of an effect on the prevalence of STE in these younger subjects than in the older patients.

Figure 4 summarizes the prevalence of STE by the single-lead criteria in the male clinical cohort by age, ethnicity, and resting heart rate. Lateral STE is the most prevalent (7%, 2%, and 1% for lateral, inferior, and anterior leads, respectively; $P < 0.01$). African Americans have the highest prevalence in all 3 areas, but the difference is most striking in the lateral leads (20% vs 4% for non-African Americans; $P < 0.01$). The inverse relationship with resting heart rate is most striking in the lateral leads (13% for less than 55 bpm and 4% for greater than 70 bpm; $P < 0.001$).

Vital status and cause of death were determined on the entire clinical cohort as of December 2002; 4086 died [963 died of cardiovascular causes (17%)] after an average follow-up of 7.9 (± 3.7) years. The survival curves for these patterns in the clinical cohort are shown in Figure 5. Table 2 lists the Cox hazard results. There were no significant differences, and multivariate Cox models did not demonstrate a hazard for cardiovascular death. The pattern significantly associated with outcome was STE in the lateral leads; it was associated with

a decreased risk of CV death, but this was no longer present after age adjustment.

DISCUSSION

Our study sought to detail the prevalence and patterns of STE among athletes and an outpatient population of patients to further understand the clinical relevance of STE and its association with cardiovascular outcomes. We considered the effects of age, gender, heart rate, and ethnicity on STE in various lead groups using a threshold of 0.1 and 0.2 mV and investigated the risk of cardiovascular death due to STE in the clinical cohort.

Our analysis demonstrates that there are significant differences in the prevalence of STE according to sex and ethnicity, with STE more common in men and in African Americans. ST elevation fulfilling the 0.2-mV (2 mm) criterion was rare, with the exception in the anterior leads, which occurred in 34% of male athletes. In general, the prevalence of STE declined with age and increasing resting heart rate. Using single-lead criteria, STE was 2 to 4 times more prevalent than

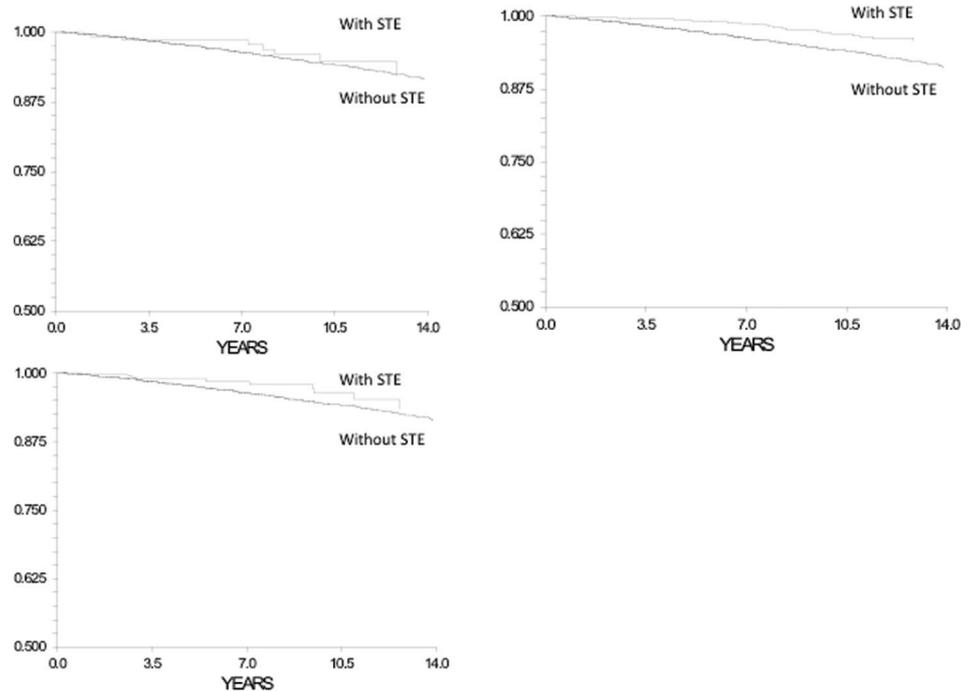


FIGURE 5. Kaplan–Meier survival curves for the clinical cohort. Anterior lead STE ≥ 0.2 mV (top left); lateral lead STE ≥ 0.1 mV (top right); inferior-lead STE ≥ 0.1 mV (bottom left). Lateral STE was associated with a decreased risk of death, but this was no longer present after adjustment (ie, the decreased mortality was due to the fact that the pattern occurred more frequently in the younger subjects).

using adjacent-lead criteria. Athletes had a higher prevalence of STE than our clinical population even when matched for age and gender. None of our athletes with anterior STE displayed inverted T waves in V_2 . It is interesting to note that 20% our female athletes exhibit T-wave inversion in V_2 , whereas it is rare in male athletes, but the converse is true for STE (present in 34% of men and none in women).⁸ Finally, STE, regardless of lead location, was not associated with cardiac death, events, or abnormal test results, including follow-up testing necessitated by the ECG that could lead to exclusion from participation.

ST elevation in the absence of acute infarction was first reported in the ECGs of healthy individuals in 1947 and titled “early repolarization” in 1951.^{9,10} This “normal RS-T segment elevation variant” was characterized as follows: (1) STE at the end of the QRS complex, (2) a distinct notch (J wave) or slur on the downslope of the R wave, (3) upward concavity of the ST segment, and (4) tall T waves in 1961.¹¹ In 1976, ER was reported as more common in young men, those of African ethnicity, and appeared typically in lateral and less in inferior leads.¹²

Recent studies suggesting an association between ER and cardiovascular death did not include STE in their definition; rather, they focused on the morphology of the downslope of the R wave. The seminal article by Haissaguerre et al² was based on a retrospective analysis of 206 individuals with idiopathic ventricular fibrillation but structurally normal hearts. The subjects were found to have a higher prevalence of J waves or slurring of the QRS downstroke compared with age-matched controls. Subsequently, the community epidemiological studies reported that J waves or slurring, particularly in the inferior leads, had an adjusted hazard of 2 to 4 for cardiac death.^{3,6} These studies were contrary to the previously upheld notion that ER was benign. Based on these 2 studies, cellular physiologists hypothesized that ER should be included in the pathological categories of J-wave syndromes and STE channelopathies.¹³

If these new observations and hypotheses are confirmed, they could have a profound impact on the PPE and on ECG screening in general because athletes, African Americans, and young men often exhibit a higher prevalence of STE than the general population. Specifically, the burden

TABLE 2. Unadjusted and Adjusted Cox Hazard Results for the Major STE Patterns in the Clinical Cohort With Early Repolarization

Cardiovascular Mortality	Univariate RR	P	Age- and Gender-Adjusted RR	P	Multivariate RR*	P
Anterior leads (V_1, V_2, V_3) using the single/adjacent lead and the 0.2-mv criteria	0.78/0.00	0.50/0.99	1.35/0.00	0.39/0.99	1.41/0.00	0.33/0.99
Lateral leads (I, aVL, V_4, V_5, V_6) using the single/adjacent lead and the 0.1-mv criteria	0.44/0.42	<0.01/0.007	0.88/0.94	0.51/0.85	0.96/1.03	0.82/0.92
Inferior leads (II, III, aVF) using the single/adjacent lead and the 0.1-mv criteria	0.62/0.84	0.14/0.66	1.1/1.44	0.76/0.37	1.07/1.34	0.82/0.44

The pattern significantly associated with outcome was ER in the lateral leads; it was associated with a decreased risk of death but this was no longer present after adjustment (ie, the decreased mortality was due to the fact that the pattern occurred more frequently in the younger subjects).

*Multivariate model was adjusted for age, gender, heart rate, and ethnicity.

RR, risk ratio.

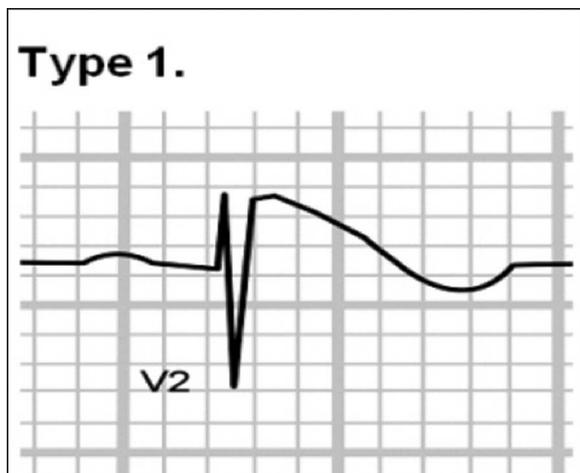


FIGURE 6. Most specific Brugada pattern (type I) for the syndrome; STE of 0.2 mV or more in V₂ occurred in one-third of our male athletes, although it was very rare in the female athletes; T-wave inversion in V₂ occurred in one-quarter of our female athletes but was very rare in the male athletes.⁸

of requiring further testing of this potentially large cohort with ER would be prohibitive.

Only 2 studies besides ours have included athletes. Rosso et al¹⁴ reported results in 45 patients with idiopathic ventricular tachycardia/ventricular fibrillation, 124 matched controls, and 121 athletes; they found that the prevalence of STE and QRS slurring was similar in all the 3 groups. Cappato et al¹⁵ studied 21 athletes with sudden cardiac death and 365 healthy athletes. J waves and/or QRS slurring was found more frequently among athletes with cardiac arrest/sudden death than in control athletes, but this difference was not statistically significant, suggesting that this ECG pattern seems not to confer a higher risk for recurrent malignant ventricular arrhythmias.

In our analyses, we focused on STE rather than the more recent definitions of ER that use slurring at the end of the QRS complex for several reasons: slurring does not lend itself to strict morphological classification, has not traditionally been included in algorithms for ER, and has not been defined in cardiology textbooks.^{16,17} The STEs seen in the Brugada syndrome are isolated to the right precordial leads,

often with right ventricular conduction delays that result in an R' with the same timing of a J wave (Figure 6).

As described above, recent studies have added to the controversy of ER and its use in cardiovascular screening. In defining ER, some reports have focused on the J waves and slurring at the end of the QRS complex and ignored the upright T waves and the STE characteristics of ER. With the available data, we see no reason to change the classic definition of ER and therefore have focused our attention on its critical component, STE, which has well-established clinical and pathological correlates. In our study, the ST level was measured at the J point (QRS end, ST beginning), which is much more qualitative than notching or slurring, and therefore, we focused on the single-lead criterion because it approximately doubled the number in each group.

Although other studies have had similar findings, our study has the largest clinical population ever studied specifically for STE and ER and the first to include a follow-up for cardiac events. Both our athletes and clinical population were studied using modern ECG technology and multiple criteria. Furthermore, the clinical population had a mean 7-year follow-up for cardiac events.

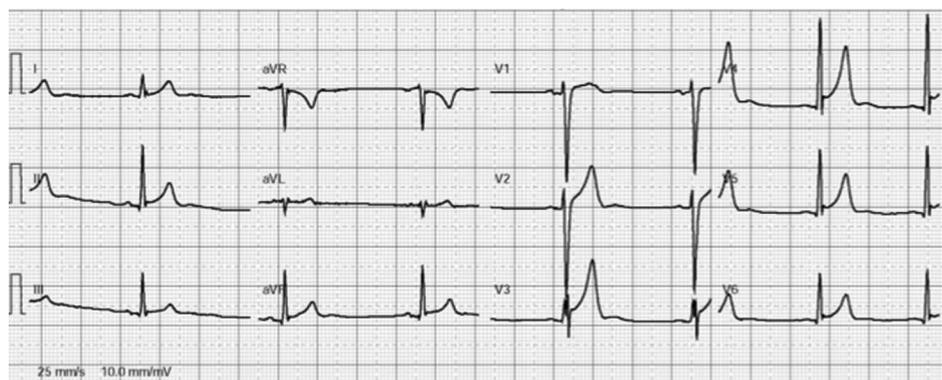
Limitations

Although our study has the largest clinical population studied to date, the small number of female patients makes it difficult to generalize to the entire population. This demographic breakdown is expected for the Veteran's Administration, and further investigation into a gender-balanced cohort would help strengthen our findings. Also, our main finding, the benign nature of STE in athletes, is mainly derived from the survival analysis of a nonathlete clinical cohort because the follow-up time line for athletes was only 3 years. A more definite conclusion would warrant a long-term follow-up of a sufficient number of athletes (both men and women) with STE; however, this would be a challenging study. Until sufficient data are available based on the follow-up of a large cohort of athletes with STE, our study provides the largest clinical study to support that it is benign.

Summary

Our study provides strong evidence that classical ER and anterior lead STE can be benign in apparently healthy

FIGURE 7. Electrocardiogram displaying STE of a healthy, 20-year-old, male, African American collegiate basketball player (heart rate, 46 bpm).



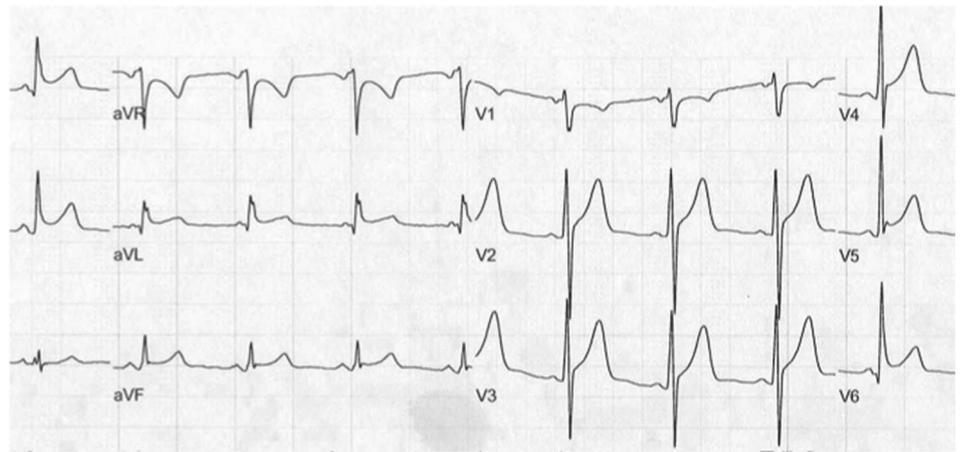


FIGURE 8. Electrocardiogram displaying STE of a sedentary, 62-year-old, white male with pericarditis (heart rate, 90 bpm).

asymptomatic individuals and need not mandate further evaluation before participation in sport because short-term follow-up showed no mortality risk. Appreciation of these findings should be helpful in distinguishing “benign” STE from that associated with pathological conditions when screening athletes. Based on our findings, the following characteristics suggest that the STE is a benign finding: (1) slower heart rate, (2) male gender, (3) younger age (<25 years), (4) STE less than 0.2 mV (2 mm), (5) African American, and (6) being athlete. The Brugada type 1 pattern did not occur in our populations but, when noted, requires evaluation by an electrophysiologist. When using the ECG for athlete screening purposes, the findings of the previous characteristics suggest that the athlete has no increased risk for cardiovascular events. Finally, these characteristics are useful in asymptomatic individuals; however, any athlete with CV symptoms should be investigated no matter what the ECG findings are. Figures 7 and 8 are representative ECGs displaying STE. The first is from a healthy, 20-year-old, male, African American, collegiate basketball player. The second is a sedentary, 62-year-old, white male with pericarditis. Although similar in appearance, demographic and clinical data can be used to differentiate the 2 using the characteristics described above.

Although highly prevalent in an athletic population, resting STE (including ER) was not associated with an increased risk of cardiovascular death in the short term in predominantly a male population and has patterns that help distinguish it from STE due to cardiac conditions, such as ischemia and injury or pericarditis. ST elevation in the anterior leads is not a practical screening criterion for the Brugada syndrome because STE greater than 0.2 mV (2 mm) occurs in one-third of male athletes. Using this knowledge and recognizing common patterns in athletes, the sports medicine physician can appropriately interpret the patterns of STE that commonly occur in athletes.

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