Early Repolarization Pattern in Competitive Athletes
Clinical Correlates and the Effects of Exercise Training

Peter A. Noseworthy, MD; Rory Weiner, MD; Jonathan Kim, MD; Varsha Keelara; Francis Wang, MD; Brant Berkstreser, MS, ATC; Malissa J. Wood, MD; Thomas J. Wang, MD; Michael H. Picard, MD; Adolph M. Hutter, Jr, MD; Christopher Newton-Cheh, MD, MPH; Aaron L. Baggish, MD

Background—Inferior lead early repolarization pattern (ERP) recently has been associated with sudden cardiac death. Although ERP is common among athletes, prevalence, ECG lead distribution, clinical characteristics, and effects of physical training remain uncertain. We sought to examine the nonanterior ERP in competitive athletes.

Methods and Results—ERP was assessed in a cross-sectional cohort of collegiate athletes (n=879). The relationship between ERP and cardiac structure were then examined in a longitudinal subgroup (n=146) before and after a 90-day period of exercise training. ERP was defined as J-point elevation ≥0.1 mV in at least 2 leads within a nonanterior territory (inferior [II, III, aVF] or lateral territory [I, aVL, V4-V6]). Nonanterior ERP was present in 25.1% (221/879) of athletes, including the inferior subtype in 3.8% (33/879). Exercise training led to significant increases in the prevalence of ERP and the inferior subtype, but there were no associations between ERP and echocardiographic measures of left ventricular remodeling. In a multivariable model, ERP was associated with black race (odds ratio [OR], 5.84; 95% CI, 3.54 to 9.61; \(P=0.001\)), increased QRS voltage (OR, 2.08; 95% CI, 1.71 to 2.52; \(P<0.001\)), and slower heart rate (OR, 1.54; 95% CI, 1.26 to 1.87; \(P<0.001\)).

Conclusions—Nonanterior ERP, including the inferior subtype, is common and has strong clinical associations among competitive athletes. The finding of increased ERP prevalence after intense physical training establishes a strong association between exercise and ERP. (Circ Arrhythm Electrophysiol. 2011;4:432-440.)

Key Words: exercise ■ electrocardiography ■ electrophysiology

Since its initial description nearly 75 years ago,1 the early repolarization pattern (ERP) has been considered a normal variant.2-4 However, emerging evidence from case-control5,6 and prospective cohort7 studies suggests that ERP in the inferior leads is associated with an increased risk of sudden cardiac death (SCD). The prevalence, clinical associations, and underlying mechanisms of this potentially malignant ERP subtype remains largely unstudied in young competitive athletes. Although SCD in athletes is relatively rare, it is an important clinical problem with a devastating impact on families and communities. Valuable athlete SCD registry data have shown that the majority of athlete SCD is attributable to cardiac causes but that an identifiable cardiac disorder is absent roughly one third of the time.8 Mechanisms and markers of electric instability in the absence of identifiable heart disease are lacking in this population.

Clinical Perspective on p 440

It is well established that ERP is more common among young athletes (prevalence estimates of 20% to 90%) than in the general population.6-9 However, the recently identified and potentially malignant inferior ERP has not been thoroughly examined in this population. Further, there are no available longitudinal data documenting the impact of exercise training on ERP. Accordingly, we conducted the current study to examine the prevalence, electrocardiography lead distribution, clinical characteristics, effect of physical training, and structural left ventricular (LV) parameters associated with ERP and the inferior subtype in young competitive athletes.

Methods

Study Design
We used cross-sectional and prospective longitudinal study designs to examine specific aspects of ERP in competitive athletes. A large cross-sectional cohort comprising competitive collegiate athletes was recruited to examine the demographic and clinical features associated with the presence of ERP. A subgroup of this cohort was studied in a prospective and longitudinal fashion to examine the effects of exercise training and exercise-induced cardiac remodeling on ERP.
Study Subjects
Athletes enrolled in the Harvard Athletics Initiative, an ongoing effort designed to address numerous aspects of student-athlete cardiovascular health, were studied. For the present study, male and female student athletes aged ≤18 years who were official participants in competitive intercollegiate athletics were enrolled between 2006 and 2010. Written informed consent was obtained from all participants before involvement. The Harvard University Institutional Review Board and the Partner’s Healthcare Human Research Committee approved the protocol before study initiation.

Study Protocol
The cross-sectional cohort consisted of newly matriculated student athletes who were recruited for participation in varsity-level (intercollegiate) competitive athletics. Participants were enrolled and studied during preparticipation screening sessions as previously reported.10 This study excluded individuals with any cardiovascular disease that was detected during screening or previously known. Height, weight, resting vital signs, medication use, and personal and family medical history were obtained. Exercise training history was elicited by questionnaires and confirmed by training logs. Exercise training history spanned the 8 weeks before enrollment and was quantified as hours per week engaged in either endurance or strength training. Exercise training history was blinded to all subject characteristics. In ambiguous cases, final adjudication was achieved by consensus with a third reviewer. Early repolarization within a single lead was defined as elevation of the J point (offset of QRS complex) of at least 0.1 mV. The amplitude of J-point elevation was semiquantitatively coded as 0.1 to <0.2 mV or ≥0.2 mV. Reviewers categorized the presence of ERP in each of the 12 leads separately and then by territory (anterior [V1 to V5], inferior [II, III, aVF], lateral [V6 to V8, I, aVL]). To be consistent with prior studies,6,11 anterior lead (V1 to V3) early repolarization was recorded but was not included in the definition of an ECG that was positive for ERP to avoid confusion with Brugada syndrome. The ECG was considered to exhibit the ERP if early repolarization was present in at least 2 leads within a nonanterior territory (inferior [II, III, aVF] or lateral [V4 to V6, I, aVL]).

The morphology of the J point was coded as notched (positive J deflection), slurred (terminal QRS slurring), discrete notched (notch observed after signal return to baseline), or discrete (early repolarization after signal return to baseline). The ST segment was coded as ascending (after the J point, signal ascends gradually until the peak of T wave), descending (after the J point, signal descends gradually until the T wave), or horizontal (before T wave, signal is horizontal). The predominant J point and ST-segment morphology were classified for each territory. Representative examples of these morphologies are shown in Figure 1.

Other ECG measurements included PR interval, QT interval, heart rate-corrected QT interval (Bazett correction), mean heart rate, and QRS duration. QRS amplitude was recorded as a continuous variable using the Sokolow-Lyon index (S in V1 +R in V5 or V6, whichever is greater).11 LV hypertrophy was recorded as a dichotomous variable as defined by a Sokolow-Lyon index ≥3.5 mV. Presence of right bundle branch block (defined as QRS complex ≥0.12 ms in duration with an rsR’ morphology in lead V1 and a QRS in lead V6), incomplete right bundle branch block (defined as a right bundle branch block morphology with a QRS duration of >0.1 ms and <0.12 ms), or left bundle branch block (defined as a QRS complex ≥0.12 ms in duration with QS or rS complex in lead V1 and a monophasic R wave in leads I and V6) were noted.

Echocardiography
Echocardiography was performed using a commercially available system (Vivid-i; GE Healthcare) with a 1.9- to 3.8-MHz phased-array transducer. Participants were imaged at rest ≥12 hours after the most recent training session. Imaging was performed to facilitate structural measurements and assessment for the presence of occult pathology of relevance to sport participation risk.10 Two-dimensional imaging was performed from standard parasternal and apical transducer positions with frame rates of 25 to 100 frames/s by trained sonographers. Each sonographer performed both baseline and poststudy imaging on the same individuals. Cardiac structural measurements were made according to current guidelines.11 LV mass was estimated using the area-length formula, and LV volumes were estimated using Simpson biplane technique. Relative wall thickness was calculated using the formula (septal thickness + posterior wall thickness)/LV internal diameter at end diastole. All data were stored digitally, and post hoc analyses were
performed with EchoPac version 7.0 (GE Healthcare) by cardiologists blinded to study time point.

Statistical Analysis

In the cross-sectional cohort, the prevalence with 95% CIs and clinical correlates of ERP and the inferior subtype were examined. A \( \chi^2 \) test was performed to examine whether the prevalence of ERP and the inferior subtype were distributed differently across sport types. Clinical characteristics were compared by ERP status (ERP positive versus ERP negative and inferior subtype ERP positive versus ERP negative) using Fisher exact test for dichotomous covariates and \( t \) test for independent samples for continuous variables. Univariable binary logistic regression models were used to test for association of clinical characteristics that differed based on ERP status (ERP positive versus ERP negative) using Fisher exact test for dichotomous covariates and \( t \) test for independent samples for continuous variables. Univariable binary logistic regression models were used to test for association of clinical characteristics that differed based on ERP status (ERP positive versus ERP negative and inferior subtype ERP positive versus ERP negative) using Fisher exact test for dichotomous covariates and \( t \) test for independent samples for continuous variables. Univariable binary logistic regression models were used to test for association of clinical characteristics that differed based on ERP status (ERP positive versus ERP negative) using Fisher exact test for dichotomous covariates and \( t \) test for independent samples for continuous variables. Univariable binary logistic regression models were used to test for association of clinical characteristics that differed based on ERP status (ERP positive versus ERP negative and inferior subtype ERP positive versus ERP negative) using Fisher exact test for dichotomous covariates and \( t \) test for independent samples for continuous variables.

Clinical Characteristics of Cross-Sectional Cohort

Characteristics of the athletes included in the cross-sectional cohort are shown in Table 1. This group comprised 879 athletes (64% men) and included representation from 20 sport types.

Prevalence and Characteristics Associated With ERP

ERP was present in 221 (35.1%) of 879 participants (95% CI, 21.4 to 28.9), and ERP was significantly more common among male athletes (33%) than among female athletes (11%, \( P<0.001 \)). The inferior ERP subtype was observed in 33 (3.8%) of the 879 participants (95% CI, 2.1 to 5.4), which accounted for 14.9% of all ERP (Figure 2). The most common morphology of ERP in the lateral leads was a discrete J point (discrete, 101/199 [51%]; discrete notched, 80/199 [40%]; notched, 14/199 [7%]; slurred, 4/199 [2%]).

Results

Clinical Characteristics of Cross-Sectional Cohort

Characteristics of the athletes included in the cross-sectional cohort are shown in Table 1. This group comprised 879 athletes (64% men) and included representation from 20 sport types.

Prevalence and Characteristics Associated With ERP

ERP was present in 221 (35.1%) of 879 participants (95% CI, 21.4 to 28.9), and ERP was significantly more common among male athletes (33%) than among female athletes (11%, \( P<0.001 \)). The inferior ERP subtype was observed in 33 (3.8%) of the 879 participants (95% CI, 2.1 to 5.4), which accounted for 14.9% of all ERP (Figure 2). The most common morphology of ERP in the lateral leads was a discrete J point (discrete, 101/199 [51%]; discrete notched, 80/199 [40%]; notched, 14/199 [7%]; slurred, 4/199 [2%]).
with an ascending ST segment (ascending, 197/199 [99%]; horizontal 2/199 [1%]). The most common morphology of ERP in the inferior leads was a notched J point (notched, 19/33 [56%]; slurred, 10/33 [30%]; discrete notched, 3/33 [9%]; discrete, 2/33 [6%]) with an ascending ST segment (ascending, 18/33 [55%]; horizontal, 15/33 [45%]). Marked J-point elevation (>0.2 mV) was observed in ≥2 leads within an inferior or lateral territory in 18 (2%) of the 879 athletes. Isolated marked J-point elevation in a single lead, however, was more common. For example, isolated marked J-point elevation was observed in lead V4 in 199 (22.6%) of the 879 athletes.

The prevalence of ERP varied by sport type, ranging from 0% to 63% as shown in Figure 3. In χ² analysis, the variability in ERP prevalence by sport type was driven by lateral ERP (P<0.001) because the comparatively uncommon inferior ERP did not differ significantly by sport (P=0.14).

Compared to ERP-negative athletes, those with ERP were more likely to be men, black, and taller; to have slower heart rates; and to have performed more previous exercise training (Table 2). In addition, those with ERP had longer QRS-complex duration and higher QRS voltage. Athletes with the inferior ERP subtype also were more likely to be black, to have lower heart rates, and to have increased QRS voltage compared with ERP-negative athletes. These variables were each considered separately in univariable logistic regression models and then collectively in a multivariable logistic regression model for association with ERP or the inferior subtype (Table 3). Multivariable regression demonstrated that male sex, black race, slower heart rate, and increased QRS voltage were significantly associated with ERP, whereas black race, slower heart rate, and increased QRS voltage were significantly associated with the inferior ERP subtype. The covariate with the largest effect (highest odds ratio) on ERP prevalence was black race with an odds ratio for ERP of 5.8 (95% CI, 3.54 to 9.61; P<0.001) and an odds ratio for the inferior subtype of 21.1 (95% CI, 9.1 to 49.1; P<0.001) (Table 3).
Characteristics of the Longitudinal Training Cohort

The longitudinal training cohort comprised male football players (n=78) and male rowers (n=68), as described in Table 4. Training during the 90-day study period (football, 19±2 hours/week; crew, 21±1 hour/week) was significantly higher than that experienced by the athletes during the 8 weeks before enrollment. The football training regimen consisted predominantly of strength training (17±1 hour/week), whereas the rowers engaged in mostly endurance training (19±1 hour/week). At completion of the training period, football players demonstrated weight gain (219±35 to 223±35 lb, P<0.001) and increased blood pressure (systolic, 116±9 to 125±13 mm Hg [P<0.001]; diastolic, 64±11 to 67±11 mm Hg [P=0.008]). In contrast, crew athletes demonstrated a drop in blood pressure (systolic, 114±9 to 112±8 mm Hg [P<0.001]; diastolic, 61±9 to 59±8 mm Hg [P=0.006]) and a drop in heart rate (62±6 to 56±4 beats/minute, P<0.001) (Table 4).

Both football players and rowers experienced significant exercise-induced LV remodeling during the 90-day study period (Table 5). Football players experienced concentric hypertrophy as demonstrated by increases in LV mass, wall thickness, and relative wall thickness. LV mass increased by a similar magnitude in rowers but was attributed to eccentric LV hypertrophy as characterized by LV chamber enlargement and unchanged relative wall thickness.

ERP and Exercise Training

The prevalence of ERP and the inferior subtype before and after exercise training is shown in Table 6. At the conclusion of the training period, the prevalence of ERP increased from 37.2% (55/148) to 52.7% (78/148) (P=0.003). This included a significant increase in the prevalence of the inferior ERP subtype from 4.1% (6/148) to 8.1% (12/148) (P=0.031). A representative pretraining and posttraining ECG is shown in Figure 4. There were no significant associations between LV parameters (baseline, posttraining, or change from baseline to posttraining) and either ERP subtype.

Clinical Follow-Up

All participants were followed for significant cardiac events for 21±13 months (range, 7 to 50 months) during organized...
intercollegiate sport participation. During this follow-up period, there were no cases of SCD, unexplained syncope, or hospitalization with a cardiovascular diagnosis.

Discussion

Recent studies suggest that ERP, specifically the inferior lead pattern, is associated with an increased risk of cardiac death. This observation is seemingly at odds with the fact that ERP is particularly common in young competitive athletes, with prevalence estimates ranging from 20% to 90%. To our knowledge, ERP lead distribution in athletes has been addressed in only 2 prior studies. Specifically, Rosso et al used a cross-sectional, case-control approach to show that ERP (both inferior and lateral) was more common in young athletes (n=132) than among sedentary controls but less common than among individuals with idiopathic ventricular fibrillation. Most recently, Junttila et al presented data documenting a nonanterior ERP prevalence of 30% in a sample of 503 collegiate athletes and demonstrated associations between ERP and both male sex and ECG correlates of LV mass.

The present study provides additive information about ERP in competitive athletes. Our data are in accord with the recent report by Junttila et al and confirm that a significant percentage of young competitive athletes (≈25% in the present study) show ERP in either the inferior or lateral electrocardiography leads. Although our data demonstrate that the inferior ERP subtype is comparatively uncommon, we found that it is associated with the same athlete characteristics (ethnicity, heart rate, etc) as the more common lateral subtype. Importantly, the current study is the first, to our knowledge, to demonstrate that exercise training is causal in the development of ERP. Specifically, both ERP and the inferior subtype increased in prevalence after intense physical training. Interestingly and somewhat surprisingly, neither ERP nor the inferior subtype was associated with the structural LV measures, including chamber volume or wall thickness, that are well-established components of the “athlete heart.” These data suggest that exercise-related ERP may be an isolated electric phenomenon that develops in parallel but not as a result of structural myocardial remodeling. In aggregate, our data suggest that ERP (including the inferior subtype) is a common finding in young, healthy, competitive athletes that is a direct result of exercise training.

Mechanistic explanations for the ERP are an area of active investigation, but at present, remain incompletely understood. In general, ERP is visible on the surface ECG when adjacent areas of myocardium repolarize at slightly different rates, causing intramyocardial current flow and upward displacement of the ST segment. ERP can be thought of as an electrocardiographic signature of repolarization dispersion and can arise in numerous conditions, including myocardial injury; extremes of temperature, such as hypothermia; local differences in I<sub>o</sub> current density; and primary heritable disorders (eg, mutations in KCNJ8, CACNA1C, CACNB2B, and CACNA2D1). In athletes, it has been proposed that parasympathetic modulation increases regional electrophysiological differences and repolarization dispersion, resulting in ST elevation, J waves, and prominent T waves.

Although the present study was not intended to examine mechanisms of ERP, several issues deserve mention. First,
the finding that ERP (and the inferior subtype) were more common in athletes with bradycardia supports a causal link between an alteration in the autonomic balance favoring parasympathetic dominance and these ERP subtypes. It should be noted, however, that other investigators have found that ST-segment elevation is not attenuated by concomitant atropine and propranolol administration. Second, ERP shared no significant associations with any aspect of structural LV remodeling. As such, it appears that although ERP may develop in parallel with structural features of the athlete’s heart, it is not clearly caused by exercise-induced LV hypertrophy or dilation.

Findings from the current study have important implications with respect to the care of the athletic patient. Our data demonstrate that nonanterior ERP, including the inferior subtype, is a common characteristic of the healthy athlete ECG and does not indicate an underlying structural abnormality. Thus, when found on an ECG that is obtained during preparticipation screening, preoperative clearance, or routine health maintenance physical examinations in an asymptomatic athlete, ERP should not necessarily mandate further costly diagnostic testing. In addition, practitioners should

Table 5.  Echocardiographic LV Measures Before and After Training

<table>
<thead>
<tr>
<th></th>
<th>Football (n=78)</th>
<th>Crew (n=68)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preseason</td>
<td>Postseason</td>
<td>P*</td>
</tr>
<tr>
<td><strong>LV function</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEF, %</td>
<td>65.9±6.7</td>
<td>65±6.6</td>
<td>0.757</td>
</tr>
<tr>
<td><strong>LV mass measurements</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass, g</td>
<td>222.5±28.6</td>
<td>248.1±35.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV mass index, g/m²</td>
<td>98.8±13.6</td>
<td>109.0±15.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IVS, mm</td>
<td>9.4±0.8</td>
<td>10.7±1.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IVS index, mm/m²</td>
<td>4.2±0.5</td>
<td>4.7±0.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PWT, mm</td>
<td>9.9±0.9</td>
<td>11.1±1.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PWT index, mm/m²</td>
<td>4.4±0.6</td>
<td>4.9±0.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>LV size/volume measurements</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVIDd, mm</td>
<td>51.4±3.7</td>
<td>51.9±3.7</td>
<td>0.26</td>
</tr>
<tr>
<td>LVIDd index, mm/m²</td>
<td>22.9±2.2</td>
<td>22.9±2.3</td>
<td>0.954</td>
</tr>
<tr>
<td>LVEDV, mL/m²</td>
<td>147.3±21.0</td>
<td>145.6±24.2</td>
<td>0.277</td>
</tr>
<tr>
<td>LVEDV index, mL/m²</td>
<td>65.5±10.2</td>
<td>64.0±10.2</td>
<td>0.042</td>
</tr>
<tr>
<td><strong>Composite measurements</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.37±0.042</td>
<td>0.42±0.050</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD. LV indicates left ventricle; LVIDd, LV end-diastolic volume; LVEF, LV ejection fraction; LVd, LV internal dimension in diastole; IVS, interventricular septal thickness; PWT, posterior wall thickness.

*Compared to preseason within a sport using a paired-sample t test.

Table 6.  Changes in ERP Prevalence With Training

<table>
<thead>
<tr>
<th></th>
<th>All Sports (n=148)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preseason</td>
<td>Postseason</td>
</tr>
<tr>
<td>All sports (n=148)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERP</td>
<td>55 (37.2)</td>
<td>78 (52.7)</td>
</tr>
<tr>
<td>Inferior ERP subtype</td>
<td>6 (4.1)</td>
<td>12 (8.1)</td>
</tr>
<tr>
<td>Football (n=78)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERP</td>
<td>28 (35.9)</td>
<td>35 (44.9)</td>
</tr>
<tr>
<td>Inferior ERP subtype</td>
<td>3 (3.8)</td>
<td>6 (7.7)</td>
</tr>
<tr>
<td>Crew (n=68)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERP</td>
<td>27 (39.7)</td>
<td>43 (63.2)</td>
</tr>
<tr>
<td>Inferior ERP subtype</td>
<td>3 (4.4)</td>
<td>6 (8.8)</td>
</tr>
</tbody>
</table>

Data are presented as n (%). Abbreviation as in Table 2.

*Compares preseason to postseason.

Figure 4.  Lateral (V4 to V6) and inferior (II, III, aVF) 12-lead ECG leads in a collegiate rower before (A) and after (B) 90 days of team-based exercise training, demonstrating the development of nonanterior early repolarization pattern (arrows).
recognize that ERP is a dynamic process that is more likely to be observed in healthy athletes at times of peak fitness.

Several limitations of the present study are noteworthy. First, it was not adequately powered to assess the relationship between ERP and SCD in athletes, and thus, we cannot make definitive conclusions about this important topic. However, it is noteworthy that no athlete with ERP, including those with the inferior subtype, experienced an adverse cardiovascular event during an average follow-up period of roughly 2 years. This follow-up period is short but included the critical period of safety concern during which athletes were participating in intercollegiate athletics. Second, although we detected no association between ERP and underlying cardiac structure, we acknowledge that the echocardiographic assessment was limited to basic LV parameters. It is possible that ERP may be a reflection of structural features not included in this protocol, such as those that define the right ventricle. Finally, we chose to study collegiate athletes, a fairly homogeneous population with respect to age, heart rate, blood pressure, and comorbid medical conditions, thereby limiting power to detect associations between clinical variables and ERP. However, we chose this group because it represents a large percentage of the real-world competitive athlete population.

In conclusion, nonanterior ERP, including the inferior subtype, is common among young competitive athletes. Importantly, the inferior and more common lateral ERP subtypes are associated with similar athlete characteristics, and both increase in prevalence with intense exercise training. These data establish a causal link between exercise training and both increase in prevalence with intense exercise training. Further investigation is warranted to fully characterize the prognostic implications of ERP in competitive athletes.

Sources of Funding

This work was supported by the Max Schaldach Fellowship in Cardiac Pacing and Electrophysiology (to Dr Noseworthy); the National Institutes of Health, National Heart, Lung, and Blood Institute (HL080025 to Dr Newton-Cheh); the Doris Duke Charitable Foundation (to Dr Noseworthy); the National Institutes of Health, National Heart, Lung, and Blood Institute (HL080025 to Dr Newton-Cheh); the Doris Duke Charitable Foundation (to Dr Newton-Cheh); the Burroughs Wellcome Fund (to Dr Newton-Cheh); and the American Heart Association (09FTF2220328 to Dr Baggish).

Disclosures

None.

References

Emerging evidence suggests that early repolarization pattern (ERP) on the 12-lead ECG, particularly when observed in the inferior leads, may be associated with increased risk of sudden cardiac death. Although it is well-known that ERP is particularly common among young athletes, its prevalence, morphology, clinical and echocardiographic correlates, and association with intense physical training remain unknown. The present study examined a large group of collegiate athletes to address these areas of uncertainty. In this cohort of nearly 900 competitive athletes, roughly one fourth were found to have ERP on the preparticipation screening ECG. The majority of athletes had ERP confined to the lateral leads, with inferior ERP present in only 4%. In a multivariable model, ERP was associated with black race, increased QRS voltage, and slower HR. There were no associations between ERP and echocardiographic measures of left ventricular remodeling. After a discrete period of intense physical training, the prevalence of ERP increased, suggesting that ERP in young athletes is a dynamic phenomenon related to the magnitude physical activity.
Early Repolarization Pattern in Competitive Athletes: Clinical Correlates and the Effects of Exercise Training

Peter A. Noseworthy, Rory Weiner, Jonathan Kim, Varsha Keelara, Francis Wang, Brant Berkstresser, Malissa J. Wood, Thomas J. Wang, Michael H. Picard, Adolph M. Hutter, Jr, Christopher Newton-Cheh and Aaron L. Baggish

Circ Arrhythm Electrophysiol. 2011;4:432-440; originally published online May 4, 2011; doi: 10.1161/CIRCEP.111.962852

Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2011 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/4/4/432

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Arrhythmia and Electrophysiology is online at:
http://circep.ahajournals.org/subscriptions/