Electrocardiographic Changes Induced by Endurance Training and Pubertal Development in Male Children

Flavio D'Ascenzi, MD, FESC[a,*], Marco Solari, MD[a], Francesca Anselmi, MD[a], Francesca Valentini, MD[a], Riccardo Barbati, MD[b], Paola Palmitesta, PhD[c], Marta Focardi, MD, PhD[a], Marco Bonifazi, MD[d], and Sergio Mondillo, MD[a]

Training-induced electrocardiographic changes are common in adult athletes. However, a few data are available on electrocardiogram (ECG) in preadolescent athletes and little is known about the potential changes induced by training on 12-lead electrocardiogram (ECG) at rest. Twelve-lead ECGs at rest and complete echocardiographic examinations were performed in 94 children (57 endurance athletes, 37 sedentary controls; mean age 10.8 ± 0.2 and 10.2 ± 0.2 years, respectively) at baseline and after 5 months of growth and training in athletes and of natural growth in controls. At baseline, athletes had lower heart rate at rest compared with controls ($p = 0.046$) and a further decrease was observed after training ($p < 0.0001$). An incomplete right bundle branch block was found in 19% of athletes and 15% of controls ($p = 0.69$) with no changes after training. Although none of the athletes showed negative T waves from V1 to V3, 6% of controls at baseline had T-wave inversion V1 to V3 with a decrease to 3% after 5 months ($p = 0.16$). The early repolarization pattern did not differ between athletes and controls and was correlated with Tanner's scale score in the overall population both at first and second evaluation ($R = 0.30$, $R = 0.27$, $p = 0.005$, $p = 0.012$, respectively). No correlations were found between ECG and echocardiographic data. In conclusion, 12-lead ECG at rest is not substantially affected by training in children, despite a physiological increase in cavity size. Thus, in preadolescent athletes, 12-lead ECG at rest does not reflect exercise-induced morphologic remodeling and seems to be influenced more by sexual maturation than by training. **(Am J Cardiol 2016;■:■−■)**

Electrocardiographic changes in athletes are common and usually reflect structural and electrical remodeling of the heart induced by training.[1−4] Electrocardiographic examination is an established tool in the evaluation of athletes, providing relevant diagnostic information on a variety of cardiovascular conditions that are associated with sudden cardiac death during sports activities, particularly in young subjects.[5] Unfortunately, physiological electrocardiographic changes observed in athletes may mimic findings of the diseased heart, causing concern to physicians who are asked to interpret the electrocardiographic findings in the setting of cardiovascular evaluation of athletes. Therefore, position statements and recommendations were published to help physicians to differentiate between physiological adaptive electrocardiographic changes and pathologic electrocardiographic abnormalities.[5−7] However, contrary to adult athletes, a few data are available on ECG in preadolescent athletes and studies longitudinally investigating the electrocardiographic changes induced by training are lacking. Therefore, the aim of the present study was to longitudinally investigate whether intensive training is able to change ECG at rest in preadolescent athletes.

Methods

Sixty-two male pre-adolescent endurance athletes practicing competitive swimming at regional level were enrolled in this study. The mean age was 10.8 ± 0.2 years. The swimmers trained once a day, over 5 or 6 days each week. The typical training started with 30 to 45 minutes of dry-land exercises (gymnastics and stretching) and continued with 75 to 90 minutes of swimming. The total training program consisted of 10% of warm-up exercises, 15% of technical training, and 75% of 3-staged aerobic exercises. The physical examination and electrocardiographic and echocardiographic evaluations were performed at the beginning of the season (September 2014, named hereafter "pre-training") and after 5 months of intensive and closely supervised training (February 2015, named hereafter "post-training"). Thirty-seven sedentary age-matched male subjects were used as controls. The mean age was 10.2 ± 0.2 years. Controls participated in recreational physical activities for less than 2 hours per week, and none was engaged in a regular training program. They were evaluated at the beginning of the study (named hereafter "baseline") and after 5 months of natural growth (named hereafter "second evaluation"). The inclusion and exclusion

[a]Division of Cardiology, Department of Medical Biotechnologies, [c]Department of Social, Political and Cognitive Sciences, and [d]Department of Medicine, Surgery, and Neuroscience, University of Siena, Siena, Italy; and [b]Division of Cardiology, Department of Cardiac, Thoracic, and Vascular Sciences, Santa Maria alle Scotte Hospital, Siena, Italy. Manuscript received September 27, 2016; revised manuscript received and accepted November 3, 2016.

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*Corresponding author: Tel/fax: 00390577585377.
E-mail address: flavio.dascenzi@unisi.it (F. D'Ascenzi).

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Table 1
Demographic characteristics of the study population

Variable	Controls (n = 37)		Endurance athletes (n = 57)		P value athletes vs. controls (baseline)	P value athletes pre - vs. post-training	P value controls baseline vs. 2nd evaluation
	Baseline	2nd evaluation	Pre-training	Post-training			
Resting HR (bpm)	77±12	78±11	72±9	67±9	0.046	<0.0001	0.63
Height (cm)	142±8	146±9	146±11	149±11	0.11	<0.0001	<0.0001
Weight (Kg)	41±12	44±12	41±10	43±10	0.94	<0.0001	<0.0001
BSA (m^2)	1.27±0.20	1.33±0.20	1.29±0.20	1.34±0.20	0.45	<0.0001	<0.0001

BSA = body surface area; DBP = diastolic blood pressure; HR = heart rate; SBP = systolic blood pressure.

Table 2
Electrocardiographic results observed at the beginning of the study and after 5 in athletes and controls

Variable	Controls (n = 37)		Endurance athletes (n = 57)		P value athletes vs. controls (baseline)	P value athletes pre- vs. post-training	P value controls baseline vs. 2nd evaluation
	Baseline	2nd evaluation	Pre-training	Post-training			
Resting HR (bpm)	77±12	78±11	72±9	67±9	0.046	<0.0001	0.63
Bradycardia	4%	4%	0%	11%	0.20	0.024	NA
PR interval (ms)	137±18	137±19	134±16	133±17	0.32	0.75	0.79
QRS duration (ms)	85±8	85±7	86±5	87±7	0.71	0.20	0.40
Incomplete RBBB	15%	15%	19%	19%	0.69	NA	NA
Complete RBBB	0%	0%	0%	0%	NA	NA	NA
Complete LBBB	0%	0%	0%	0%	NA	NA	NA
LAD	0%	0%	0%	0%	NA	NA	NA
QRS axis (degree)	48±28	46±32	57±26	56±28	0.15	0.80	0.41
Isolated QRS voltage criteria for LV hypertrophy (Sokolow-Lyon)	14%	11%	19%	24%	0.69	0.18	0.33
Isolated QRS voltage criteria for RV hypertrophy (Sokolow-Lyon)	0%	0%	0%	0%	NA	NA	NA
Pathological Q-waves	0%	0%	0%	0%	NA	NA	NA

LAD = left axis deviation; LBBB = left bundle branch block; LV = left ventricular; NA = not applicable; RBBB = right bundle branch block; RV = right ventricular.

criteria have been previously described.[8] According to these criteria, 3 athletes were excluded because of musculoskeletal injuries and 2 for evidence of cardiac heart disease (1 with atrial septal defect and 1 with patent *ductus arteriosus*), whereas none of the controls were excluded. Thus, the final population included 57 healthy athletes. Height, weight, body surface area (BSA),[9] and the biologic maturation, assessed by the Tanner's 5 stages of penile and testicular development,[10] were obtained both at the beginning of the study and after 5 months. The presence of cardiac symptoms, fatigue, or performance impairment was also investigated.

A standard 12-lead ECG was performed using an ESAOTE P8000 Power Light, recorded at 25 mm/s in a supine position during quiet respiration. ECG was interpreted by an expert cardiologist, blinded to study time and without any knowledge of the echocardiographic findings. Heart rate (HR) at rest, PR interval, QRS duration, QT interval, and QRS axis were calculated. Any form of ventricular pre-excitation or atrioventricular block was sought. Left axis deviation was defined as a QRS axis exceeding −30°, and right axis deviation was defined as a QRS axis exceeding +120°.[11] Incomplete right bundle branch block (RBBB) was defined as QRS duration 90-100 ms, with r′ or R′ wave in lead V1 or V2, an S wave of greater duration than R wave or greater than 40 ms in leads I and V6, and normal R peak time in leads V5 and V6 but greater than 50 ms in lead V1, while complete RBBB was defined as QRS duration >100 ms in presence of the criteria described earlier.[12] The presence of T-wave inversion in the peripheral leads and beyond V1 in the precordial leads was also explored.[13] The Sokolow-Lyon voltage criteria were applied for the definition of left ventricular hypertrophy (LVH) and right ventricular (RV) hypertrophy.[14,15] Left atrial enlargement, right atrial enlargement, and early repolarization (ER) were defined as recommended by the Seattle criteria.[16] A Q wave was considered abnormal or pathological if it exceeded 0.04 seconds in duration and/or the depth of the Q wave exceeded 25% of the height of the R wave.[11] The QT interval was corrected for HR (QT_c) at rest. A QT_c interval was considered abnormally prolonged if longer than 460 milliseconds.[13]

The echocardiographic examination has been previously described in detail.[8] In brief, echocardiography was performed by 1 cardiologist using a high-quality echocardiograph (Vivid 9; GE, Milwaukee, Wisconsin), equipped with a M4S 1.5-4.0 MHz transducer, and a one-lead ECG was continuously displayed. Off-line data analysis, from 3 stored cycles, was performed by 2 experienced readers, blinded to the study time-point, using a dedicated software