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FULL RESEARCH PAPER
Sports cardiology

Prevalence and determinants of low QRS voltages and QRS fragmentation in children and adolescents undergoing sports pre-participation screening

Francesca Graziano [1,2], Oscar Edoardo Genta[1,3], Laura Manfrin[1,4], Domenico Corrado [1], Laura Brusamolin[5], Franco Giada[6], Luigi Gerbino[6], Silvia Compagno[6], and Alessandro Zorzi [1]*

[1]Department of Cardiac, Thoracic and Vascular Sciences and Public Health, University of Padova, Via Giustiniani 2, 35128 Padova, Italy; [2]Department of Sports Medicine, Semmelweis University, Budapest, Hungary; [3]Cardiovascular Department, University of Milan, Milan, Italy; [4]Cardiovascular Department, University of Trieste, Trieste, Italy; [5]Sports Medicine Unit, AULSS6, Padova, Italy; and [6]Sports Medicine and Cardiovascular Rehabilitation Unit, AULSS3, Noale, Italy

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Aims Low QRS voltages (LQRSV) in limb leads and QRS fragmentation (FQRS) are possible electrocardiographic signs of myocardial fibrosis and cardiomyopathy, but they are not listed in current criteria for interpreting athlete's electrocardiogram (ECG). We investigated the prevalence and determinants of LQRSV and FQRS in a cohort of young apparently healthy athletes undergoing pre-participation screening (PPS).

Methods and results We analysed a consecutive series of 2140 ECG obtained during PPS of young athletes (mean age 12.5 ± 2.6 years, 7–18-year-old, 49% males). The peak-to-peak QRS voltage was measured in all limb leads, and LQRSV were defined when maximum value was <0.5 mV. Fragmented QRS morphologies were grouped into five patterns. Lead aVR was not considered. Maximum peak-to-peak QRS voltage in limb leads was 1.4 ± 0.4 mV, similar between younger and older athletes, but significantly lower in females than males (1.35 ± 0.38 mV vs. 1.45 ± 0.42 mV; $P < 0.001$). There was a weak correlation between maximal QRS voltages and body mass index (BMI), but not with type of sport or training load. Only five (0.2%) individuals showed LQRSV. At least one fragmented QRS complex was identified in 831 (39%) individuals but excluding the rSr′ pattern in V1–V2, only 10 (0.5%) showed FQRS in ≥2 contiguous leads. They were older than those without FQRS, but did not differ in terms of gender, BMI, type of sport, or training load.

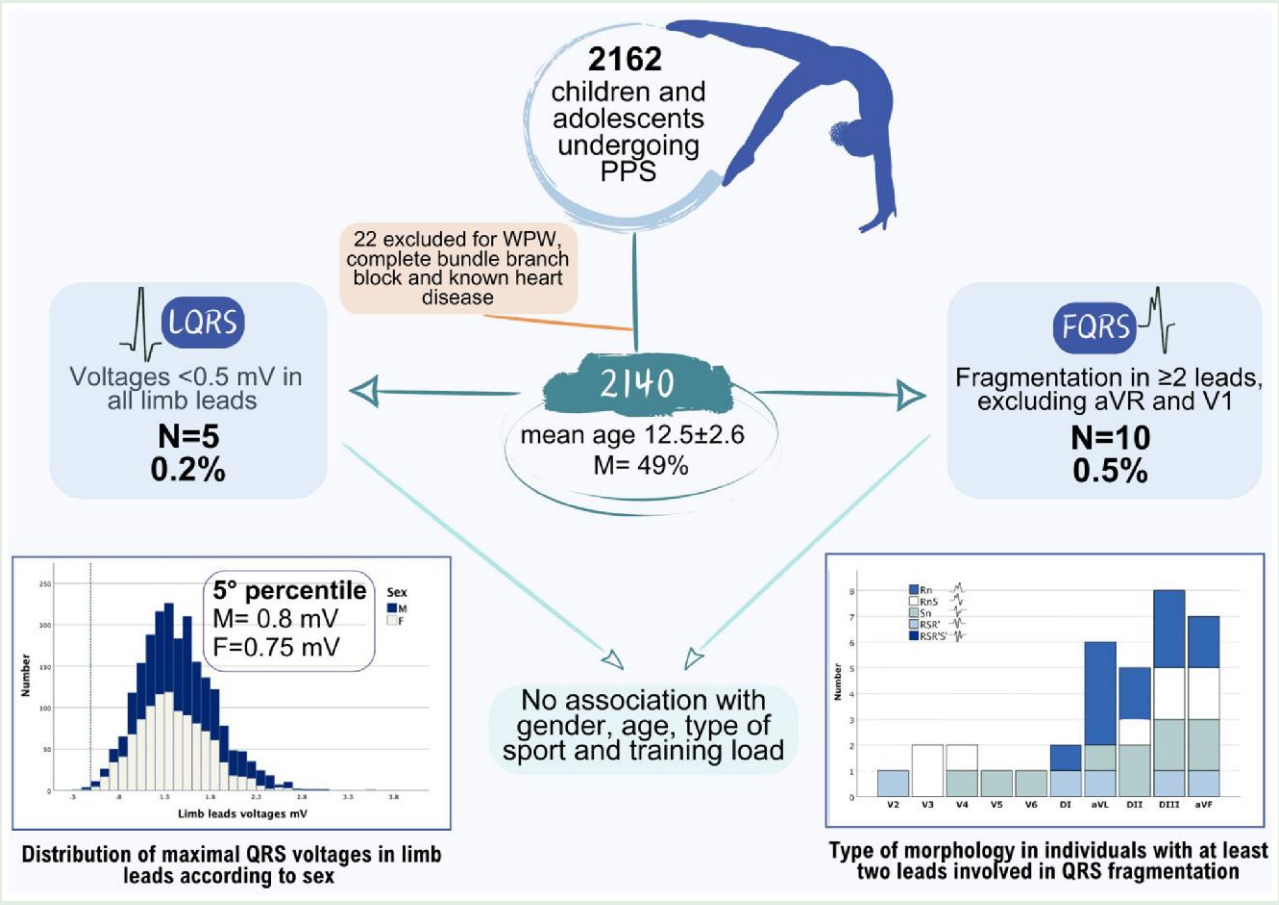
Conclusion Low QRS voltages in limb leads and FQRS in ≥2 contiguous leads excluding V1–V2 are rare in young apparently healthy athletes and are not related to the type and intensity of sport activity. Therefore, they may require additional testing to rule out an underlying disease particularly when other abnormalities are present.

Lay summary Low QRS voltages (LQRSV) in limb leads and QRS fragmentation (FQRS) are possible electrocardiographic signs of myocardial fibrosis and cardiomyopathy. In our study, we analysed the occurrence and characteristics of FQRS and LQRSV in young athletes undergoing pre-participation screening.

- We found a low prevalence of these abnormalities, with only 0.2% showing LQRSV and 0.5% displaying FQRS.
- These abnormalities were not associated with factors such as gender, age, type of sport, or training load.

* Corresponding author. Tel: +390498212322, Email: alessandro.zorzi@unipd.it

Graphical Abstract



Keywords Athletes • Children • Sports cardiology • Cardiomyopathy • Sudden cardiac death • Screening

Introduction

Pre-participation screening (PPS) offers the possibility to identify diseases at-risk of sudden cardiac death (SCD) early, before they become symptomatic.[1] A simple clinical tool such as the 12-lead electrocardiogram (ECG) has consistently demonstrated to increase the sensitivity of PPS compared to history and physical examination only.[2] However, structural and electrical remodelling secondary to training may cause the occurrence of abnormalities that resemble those observed in heart diseases, potentially reducing the specificity of the ECG.[3] To overcome this limitation, specific criteria for interpretation of the athlete's ECG have been developed.[4] They divide the ECG changes in three categories: group 1, including common and training-induced changes, which are very frequent in normal athletes and usually benign; group 2, including changes that are uncommon/untrained-related in athletes but unlikely to be the sign of a disease if found in isolation and group 3, including changes that not only are uncommon/non-training related in healthy athletes but also frequent in patients with underlying heart diseases. According to these recommendations, further examinations to rule out pathological conditions are required when ≥1 group 3 or ≥2 group 2 abnormalities are present.

Among the ECG changes that are neither discussed nor classified in one of the three categories, two have recently attracted attention as possible signs of cardiomyopathy: low QRS voltages (LQRSV) and fragmentation of the QRS complex (FQRS). The definition of LQRSV varies in the literature but most studies consider only the limb leads (that are less influenced by leads position and chest conformation) and define voltages as low when peak-to-peak QRS is <0.5 mV in all leads.[5,6] Recent reports showed that LQRSV may be particularly associated with left-ventricular (LV) scarring.[7–11] However, the prevalence of LQRSV in young athletes and whether the 0.5 mV cut-off should be modified remain to be established. Also, FQRS may be potentially a marker of an underlying LV scar, which alters the wave front of myocardial depolarization, but there are few data on their prevalence and association with training.[12–15]

The aim of our study was to assess the prevalence and determinants of LQRS and FQRS in a cohort of young, apparently healthy athletes undergoing PPS.

Methods

We retrospectively evaluated young athletes aged 7–18 years old of both genders who underwent annual PPS from 2019 to 2022 at the centres for sports medicine of the National Health System of Padua (AULSS6) and Noale-Venice (AULSS3), Veneto region, Italy. Athletes with overt cardiovascular diseases were excluded. The study was approved by the local Ethical Committee: because of the observational and retrospective nature of the study, consent from participants was waived. The data supporting this study are available from the corresponding author upon reasonable request.

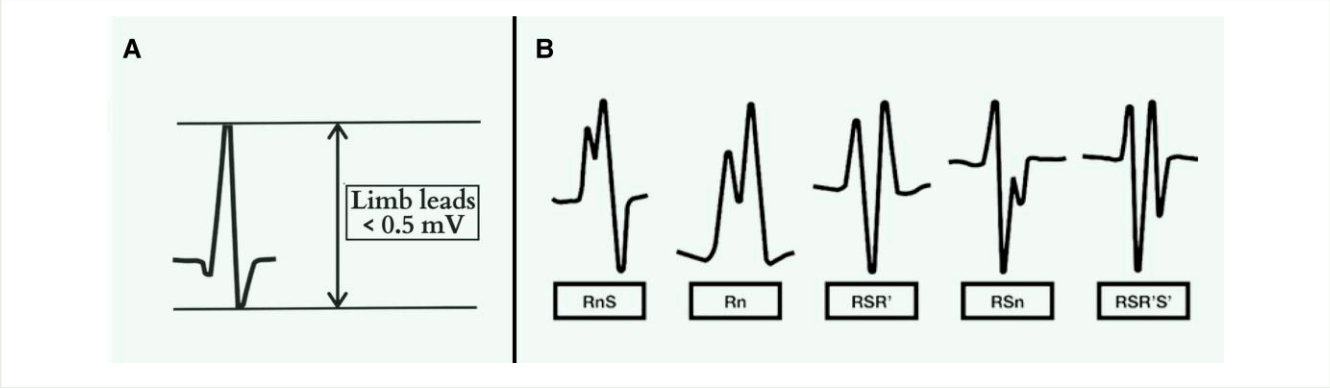


Figure 1 Peak-to-peak measurement of QRS voltage and definition of low voltages in limb leads (*A*). Classification of the five patterns of QRS fragmentation (*B*). The RSR′ pattern included any morphology with two positive waves and one negative wave. The R pattern with notch (Rn) included any notch of the R wave that did not cross the isoelectric line. This pattern could be followed by an S wave, resulting in the RnS pattern. Similarly, the S pattern with notch (RSn) included any notch of the S wave that did not cross the isoelectric line. Finally, the RSR′S′ pattern included any four-phase QRS morphology with two positive and two negative waves.

Pre-participation screening protocol

In Italy, PPS carried out by a physician with a postgraduate degree in Sports Medicine and physical exercise is mandatory for all non-professional competitive athletes. The age at which the PPS starts varies according to the sport discipline and is set by each sports federation depending on when official competitions begin. The screening protocol is established by the Italian law and regional regulations. It includes family and personal history, physical examination with blood pressure measurement, spirometry, urine dipstick, visual acuity test, resting 12-lead ECG, and limited stress testing particularly aimed at evaluating ventricular arrhythmias inducibility. Further examinations may be required in case of abnormal findings at first-line screening.

Classification of sports type and intensity

The sports practiced were classified into four categories according to the European Society of Cardiology recommendations: skill, power, endurance, and mixed.[16] According to the hours of training per week, the children athletes were arbitrarily classified as: *high training load* (>6 h per week); *moderate training load* (4–6 h of training per week); and *low training load* (<4 h per week).

Twelve-lead resting electrocardiogram analysis

Electrocardiograms were acquired in the supine position with the limb leads placed at the wrists and ankles at standard speed of 25 mm/s and at a standard gain of 1 mV/cm. Filters were set at 0.05 and 150 Hz. They were scanned at high definition, and QRS amplitudes were measured with digital callipers. Electrocardiogram was interpreted by experienced cardiologists according to the 2017 International Recommendation for ECG interpretation in athletes,[4] and discrepancies were solved by consensus.

The amplitude of the QRS complex from peak to nadir was measured in all limb leads. Low QRS voltages were defined as an amplitude of the QRS complex < 0.5 mV in each limb lead (*Figure 1A*). The FQRS morphologies were classified with a system inspired by the modified criteria of Haukilahti *et al.*[12] Using this system, fragmented morphologies were grouped into five different patterns (*Figure 1B*); aVR was excluded from the analysis because of the frequent presence of a rSr′ pattern in this lead ($n = 1448$, 68% in our study), which is considered physiological.

Athletes with known cardiac diseases including corrected congenital heart defects, ventricular pre-excitation, and complete bundle branch block were excluded as these conditions can alter the ECG.

Statistical analysis

Normal distribution of all continuous variables was examined using the Shapiro–Wilk test and Q–Q plot. Continuous variables are expressed as median with 25th–75th percentiles if non-normally distributed, or mean ± SD if normally distributed. Categorical variables are expressed as *n* (%) and were compared with the χ^2 test or the Fisher's exact test, as appropriate. For main prevalence data, 95% confidence intervals (CI) were calculated using the Binomial distribution. Continuous variables were analysed with Student *t*-test or Mann–Whitney *U* test as appropriate. Bonferroni correction was performed to correct multiple comparison among subgroups. A correlation analysis was conducted to assess the relationships between voltages and age, sex, body mass index (BMI), type of sport, and training load. Univariate linear regression models were applied to explore the associations between voltages and age, sex, BMI, type of sport, and level of training. The results from this model are presented as beta coefficients (β) with accompanying 95% CI. All models were adjusted for participant age and sex. A significance level of 0.05 from a two-tailed test was considered statistically significant throughout the study. Data were analysed with SPSS V.29 (IBM).

Results

Demographic and baseline characteristics

Between 2019 and 2022, 2162 athletes aged 7–18 years old underwent PPS. Of these, 22 were excluded from the analysis because of corrected congenital heart defects, ventricular pre-excitation, complete bundle branch block, or because they received a diagnosis of an underlying at-risk heart disease by PPS (none of the latter showed LQRSV or FQRS). The final population was composed of 2140 athletes, median age 12.5 ± 2.6 years, 49% males, and 98.5% Caucasian (*Table 1*).

Most of the enrolled athletes practiced mixed sports (61%), followed by endurance sports (29.1%) and skill sports (9.9%). The three most popular sports were football (18.6%), volleyball (14.7%), and basketball (13.5%). The *high training load* athletes resulted to be 25.6%, the *moderate training load* athletes 35%, and *low training load* athletes 39.4%.

Electrocardiographic parameters and diagnosis

Table 2 provides details about the ECG characteristics of the study sample. The most frequent normal ECG alterations were incomplete right bundle branch block (iRBBB), early repolarization, left ventricular hypertrophy, and right ventricular hypertrophy, all belonging to group 1 abnormalities (common and usually benign) according to the international recommendations.[4]

Table 1 Demographic characteristics of the study population

	Total population $n = 2140$	Males $n = 1048$ (49%)	Females $n = 1092$ (51%)	P
Age (years)	12 (11–14)	12 (11–15)	12 (10–14)	$P < 0.001$
Height (cm)	158 (148–167)	160 (151–173)	156 (144–163)	$P < 0.001$
Weight (kg)	57.5 (39–60)	62 (47–87.5)	54 (42–70)	$P < 0.001$
BSA (Mosteller, m^2)	1.47 (1.26–1.66)	1.51 (1.32–1.74)	1.43 (1.19–1.60)	$P < 0.001$
BMI (kg/m^2)	20 (17.3–21.9)	19.9 (17.65–22.15)	19.4 (16.85–21.67)	$P < 0.001$
Positive family history[a]	589 (27)	293 (28)	296 (27)	$P = 0.746$
Training time, h/week	4.5 (2–6)	4.5 (3–6)	4.5 (3–6)	$P = 0.20$
Years of training	2 (1–5)	3 (1–5)	2 (1–5)	$P < 0.001$

Data are given as number (percentage) or median (25th–75th percentile).
[a]For premature (<40 years old in men and <50 years old in women) sudden death, inherited cardiomyopathies, or coronary artery disease.

QRS voltages

Distribution according to age and sex

The maximum peak-to-peak QRS voltage in the limb leads was on average 1.40 ± 0.41 mV. There was no significant correlation between limb lead voltages and age ($r = -0.02$; $P = 0.2$) but maximum QRS voltage was significantly higher in males (1.45 ± 0.42 mV) than females (1.35 ± 0.38 mV; $P < 0.001$). In female athletes, a weak negative correlation resulted between maximum QRS voltage and age ($r = -0.081$, $P = 0.007$), demonstrating lower limb lead voltages in the older athletes. This correlation was not found in males ($P = 0.7$). *Figure 2* shows the distribution of limb voltages and mean values according to age in both sexes.

Distribution according to body mass index

There was a weak negative correlation between limb leads and BMI ($r = -0.047$; $P = 0.03$) in the entire population. As the BMI was significantly higher in male than female athletes (males = 20[18–24], kg/m^2; females = 19[17–22], kg/m^2; $P < 0.001$) and in order to compare the voltages values between males and females without BMI as a confounder factor, we divided the population into homogeneous tertiles of BMI [1°: BMI ≤ 18 kg/m^2 (38.2%), 2°: BMI 19–21 kg/m^2 (32.5%), 3°: BMI > 21 kg/m^2 (29.3%)]. The difference in voltages between male and female athletes remained significant (*Table 3*). Moreover, in male athletes, BMI was weakly negatively correlated with limb voltages ($r = -0.066$; $P = 0.03$), whereas in female, it was not ($P = 0.13$).

Distribution according to training level and type of sport

Because of the different voltages trend in males and females, the higher training level in males and the higher percentage of male athletes participating in mixed sport, we performed the analysis dividing the population according to sex. There was no significant association between the maximum QRS voltage in limb leads and level or type of sports both in males and females (males, $P = 0.128$; females, $P = 0.23$).

Regression analysis

A sex-adjusted univariate regression analysis was performed to test which variables among age, BMI, type of sport, and level of training influenced the QRS voltages. The only significant factor resulted to be BMI, with a low beta value (*Table 4*).

Athletes with low QRS voltages

Five athletes (0.2%, 95% CI 0.01–0.5%) showed LQRSV in limb leads (<0.5 mV): two of them were ≤12 years old, one male and one female; three of them were >12 years old, two males and one female. Together with LQRS voltages in limb leads, two athletes showed respectively left axis deviation and right axis deviation at the basal ECG, and one athlete had two premature ventricular beats (PVBs) with LBBB/inferior axis morphology during effort at the exercise test. Therefore, these three underwent additional investigations (echocardiography and 24 h Holter monitoring) that were unremarkable. Three of five athletes underwent a follow-up PPS in 2023: in two, LQRSV continued to be observed while in one, maximum peak-to-peak QRS voltage in the limb leads increased to 0.8 mV. No clinically relevant events occurred.

QRS fragmentation

In our population, 831 (39%) individuals showed at least one lead with FQRS at the basal ECG excluding aVR, in particular in leads V1 (521, 24.3%) and V2 (257, 12%). In most cases, the type of pattern was rSr′ consisting with a diagnosis of iRBBB. Ten (0.5%, 95% CI 0.2–0.9%) individuals showed FQRS pattern in ≥2 contiguous leads different from V1–V2: two in the anterior leads (V2–V4, 0.09%), one in the lateral leads (V5–V6, 0.04%), two in high lateral leads (DI–aVL, 0.09%), and eight in the inferior leads (DII–DIII–aVF, 0.4%) (*Figure 3*).

There were no statistically significant differences between young athletes with and without FQRS in terms of age ($P = 0.245$), sex ($P = 0.142$), BMI ($P = 406$), training level ($P = 0.712$), and type of sport ($P = 0.893$). Eight of them were males (80%), four (40%) were high intensity athletes, four (40%) were moderate intensity athletes, and two (20%) were low intensity athletes; seven (70%) practiced mixed sport, two (20%) endurance sports, and one (10%) skill sports. The ECG characteristics of these athletes are showed in *Table 5*.

Among the 10 individuals with FQRS in ≥2 contiguous leads, two underwent further examinations: (1) a 13-year-old male soccer player with a cardiac systolic click and murmur at the physical examination, and with the basal ECG characterized by FQRS with Sn morphology in V5–V6–aVL, underwent an exercise testing that was unremarkable and an echocardiography that showed a mitral valve prolapse with a mild regurgitation; and (2) a 16-year-old male gymnastic with both FQRS in the inferior leads with Rn pattern, and PVBs with a RBBB configuration at exercise testing, underwent a comprehensive work-up including cardiac magnetic resonance (CMR) that resulted to be normal. Five of 10 athletes underwent a follow-up PPS in 2023, and all continued to show FQRS although no clinically relevant events occurred.

Discussion

In this study, we analysed a consecutive series of ECG of young athletes undergoing PPS at two different Italian sports medicine centres and aimed to analyse the prevalence and determinants of LQRSV and

Table 2 Electrocardiographic parameters of the total population

ECG parameter	Total population $n = 2140$
HR, b.p.m.	75 (67–84)
P wave, ms	90 (80–100)
PR interval, ms	134 (120–155)
QRS, ms	90 (84–100)
QT interval, ms	360 (346–380)
QTc interval, Bazett	406 (389–423)
Group 1 abnormalities	
QRS axis, degree	75 (68–83)
Incomplete RBBB	488 (22.8)
Early repolarization	316 (14.8)
LVH (Sokolow–Lyon)	296 (13.8)
RVH (Sokolow–Lyon)	229 (10.7)
TWI V1–V3 < 16 years old	109 (5.1)
Sinus bradycardia	78 (3.6)
Junctional rhythm	2 (0.1)
Ectopic atrial rhythm	36 (1.7)
1° AV block	17 (0.8)
Group 2 abnormalities[a]	
LAD	2 (0.09)
LAE	7 (0.32)
RAD	6 (0.28)
RAE	22 (1.02)
Group 3 abnormalities[a]	
Pathological TWI	9 (0.37)
QRS ≥ 140 ms (LBBB morphology)	1 (0.4)
PVB ≥ 1[b]	6 (0.28)

Data are given as number (percentage) or median (25th–75th percentile). There are: the basal ECG parameters; the ECG characteristic classified respectively as normal (group 1 abnormalities), borderline (group 2 abnormalities), and abnormal (group 3 abnormalities) according to the International recommendation for ECG interpretation in athletes.[4]
AV, atrioventricular; b.p.m., beat per minute; ECG, electrocardiographic; HR, heart rate; LAD, left axis deviation; LAE, left atrial enlargement; LBBB, left bundle branch block; LVH, left ventricular hypertrophy (voltage criteria); ms, millisecond; PVB, premature ventricular beat; RAD, right axis deviation; RAE, right atrial enlargement; RBBB, right bundle branch block; RVH, right ventricular hypertrophy (voltage criteria); TWI, T-wave inversion.
[a]Athletes with complete bundle branch block or ventricular pre-excitation were excluded.
[b]Modified from International criteria.

FQRS. The main findings were the following: (1) maximum peak-to-peak limb lead QRS voltage was on average 1.40 ± 0.41 mV, significantly higher in males (1.45 ± 0.42 mV) than females (1.35 ± 0.38 mV; $P < 0.001$); (2) maximum limb lead QRS voltage was slightly influenced by BMI, but not by age, type of sport, or training intensity; (3) only five (0.2%) individuals fulfilled the definition of LQRSV (peak-to-peak < 0.5 mV in all limb leads), and the cut-off corresponding to the 5th percentile of the study sample was 0.8 mV; (4) excluding leads V1–V2 that often showed QRS fragmentation as a result of incomplete RBBB (rSr′ or rSn patterns), FQRS involving ≥2 contiguous leads was uncommon (10, 0.5%) with no differences from the rest of the population in terms of sex, age, BMI, type of sport, or training load; and (5) further echocardiographic examinations were performed in 5/15 athletes with LQRSV or FQRS because they showed other abnormalities at PPS and one showed a cardiac disease (mitral valve prolapse).

Classification of electrocardiogram abnormalities in the athletes

The main aim of PPS is to identify with very high sensitivity all heart diseases that may pose a threat to the athlete's health. On the other hand, PPS needs to be sustainable and the proportion of false positive findings triggering second-line (such as echocardiography) and potentially third-line (such as CMR) investigations should remain within acceptable values.[17] With this in mind, current criteria for interpretation of the athlete's ECG follow probability criteria and identify three groups.[4] The first includes abnormalities that needs to fulfil two requirements: (1) to be secondary to training (e.g. sinus bradycardia, increased in QRS voltages in the precordial leads…) and (2) to carry a low probability of an underlying disease. Those changes do not require additional investigations if found in isolation. Group 2 includes 'borderline findings' (such as atrial enlargement and QRS axis deviation) that have a low predictive value for an underlying disease but are also uncommon and not training related in athletes. According to current recommendations, group 2 changes require additional investigations only if two or more are found in the same ECG. Finally, group 3 include abnormalities with a higher predictive value for diseases and should always require further clinical work-up also when only one is present.

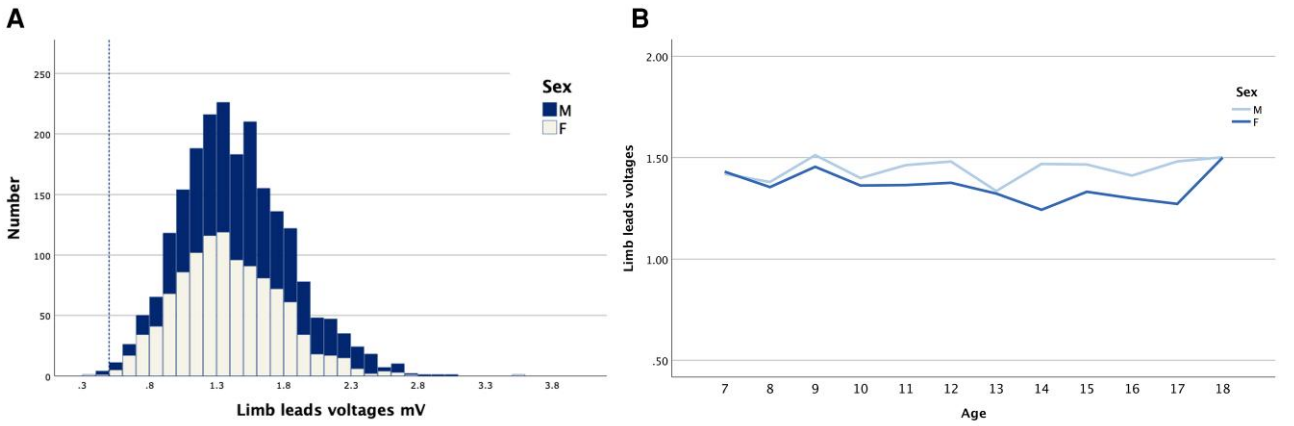
Low QRS voltages and FQRS are not classified in any category, because scientific data were lacking at the time of document writing. However, they may represent a potential sign of diseases characterized by ventricular scarring (such as arrhythmogenic cardiomyopathy), which is a potential substrate of sustained ventricular tachycardia during sports activity.[7–10,12] Understanding how common these ECG changes are in athletes and whether they are 'training-dependent' is fundamental for proper classification.

Low QRS voltages in limb leads

Low voltages in the limb leads can be the sign of a cardiomyopathy that increases the risk of SCD during sport.[5,6,18] Particularly, the presence of a non-ischaemic LV fibrosis as suggested by subepicardial/midmyocardial late gadolinium enhancement on CMR can cause a decrease in the limb lead voltages.[7–11] The non-ischaemic LV scar is a substrate of re-entrant ventricular tachycardia and can cause major arrhythmic events and SCD, particularly during sport.[8,19] It can be suspected based on ECG abnormalities (such as LQRSV or T-wave inversion in the lateral leads) or exercise-induced PVBs with a RBBB configuration (suggesting LV origin) at PPS.[20,21] Unfortunately, echocardiography has a low sensitivity for non-ischaemic LV scar because the fibrosis spares the subendocardial layer that mostly contributes to myocardial thickening.[22] Hence, the diagnosis requires tissue characterization by CMR, which is a more expensive and less available imaging modality.[23] Although prescription of CMR to athletes with LQRSV may appear justified, for a cost-effective use of the resources, there is a need to understand whether this pattern is common and training-related.

In a study by Mango *et al.*,[24] 4% of 503 Italian Olympic athletes showed LQRSV. Similar to our findings, age, sex, BMI, and type of sport were not correlated with the presence of LQRSV. Moreover, although systematic CMR study was not performed, athletes with LQRSV showed a higher prevalence of PVBs with a RBBB configuration potentially suggesting a possible underlying LV scar. More recently, Zorzi *et al.* examined 2229 subjects including 775 national and international elite athletes (mean age 23) and 1454 amateur/recreational athletes (mean age 18) of mixed ethnicities: the prevalence of LQRS was 2.2% and 0.5%, respectively. Predictors of LQRSV were higher age and elite athlete status. Of note, 33% of athletes with LQRSV showed PVBs during exercise testing and some of those who underwent CMR had an underlying non-ischaemic LV scar.[7]

In our study, which focused on children and adolescents (mean age 12 years), we found an even lower prevalence of LQRSV in limb leads



	Median	1°	5°	10°	25°	50°	75°	90°	95°	99°
Males	1.40	0.57	0.8	0.95	1.15	1.40	1.70	2.0	2.20	2.60
Females	1.30	0.60	0.75	0.90	1.10	1.30	1.60	1.85	2.0	2.40

Figure 2 Distribution of QRS voltages according to sex in panel (*A*), and sex and age in panel (*B*). In the table, the median value and the percentiles in male and female athletes. The values of the voltages reported in the limb leads are the maximal voltages measured in all limb leads. The dotted line marks the value of 0.5 mV (*A*).

Table 3 Comparison of limb lead voltages in mV between the homogeneous body mass index tertiles among male and female athletes

	Females	Males	*P*
BMI ≤ 18 kg/m^2	1.35 ± 0.39	1.46 ± 0.43	*P* < 0.001
BMI 19–21 kg/m^2	1.36 ± 0.39	1.43 ± 0.41	*P* = 0.014
BMI > 21 kg/m^2	1.33 ± 0.36	1.45 ± 0.42	*P* = 0.001

Data are given as mean ± standard deviation.
BMI, body mass index.

Table 4 Sex-adjusted limb lead voltages linear regression results for age, body mass index, type of sport, and level of training

	Beta	*P* value	CI (95%)
Age	−0.006	0.093	−0.012 to 0.001
BMI	−0.006	0.010*	−0.011 to −0.001
Type of sport	−0.019	0.183	−0.009 to 0.046
Level of training	0.019	0.130	−0.006 to −0.044

Metrics displaying beta coefficients and 95% confidence intervals for athletes.
*$P < 0.05$.

(0.2%). Moreover, 95% of the study sample showed a maximum peak-to-peak QRS voltage > 0.8 mV (thus representing the 5th percentile of our voltages distribution), suggesting that the 0.5 mV threshold may be too conservative in children. In addition, we also did not find any association between QRS voltages and type of sport/training load. Hence, the present and previous reports agree that LQRSV in limb leads cannot be classified as 'common and training related'.[6] However, the positive predictive value of such ECG abnormality could not be established neither by the current nor the previous studies because systematic CMR was not performed in young athletes with isolated LQRSV as it was not considered clinically justified at the time these studies were performed.

Fragmentation of the QRS complex

Fragmentation of the QRS in two contiguous leads may be caused by altered propagation of the depolarization wave front throughout a diseased myocardium.[12,25] A well-known form of QRS fragmentation in classical (right-dominant) arrhythmogenic cardiomyopathy is the so-called 'epsilon wave', which is defined as a fragmented positive wave at the end of the QRS complex in leads V1–V2 resulting from delayed depolarization of scarred right ventricular regions.[26] Calò *et al.*[11] recently assessed the value of the ECG in patients with the variant of arrhythmogenic cardiomyopathy with predominant LV involvement. They found that FQRS was present in 35% of patients vs. 11% of controls and were distinctively associated with more extensive non-ischaemic LV scarring (so-called 'rink-like pattern'). However, FQRS is not uncommon in normal individuals (prevalence up to 30%) due to benign conduction delay or anatomical variation in the His-Purkinje system.[12] Despite the potential diagnostic utility of FQRS in the setting of sports PPS, data are needed to assess whether this ECG sign is common and training related.

Some studies focused on QRS fragmentation in lead V1 in athletes, as a potential sign of conduction delay secondary to remodelling of the athlete's heart. Ollitrault *et al.*[15] found that a quadriphasic QRS complex in lead V1 was more frequent in athletes compared with non-athletes (22% vs. 5%) and independently correlated with age and right ventricular outflow tract dimensions. Similar findings were obtained by Vecchiato *et al.*,[13] who reported a prevalence of FQRS in V1 as high as 33%. This pattern correlated with higher exercise capacity and indexes of right ventricular remodelling. In our study on children and adolescents, we also found a high prevalence of FQRS in leads V1 and V2 (24% and 12%, respectively) with predominantly rSr′ or rSn patterns,

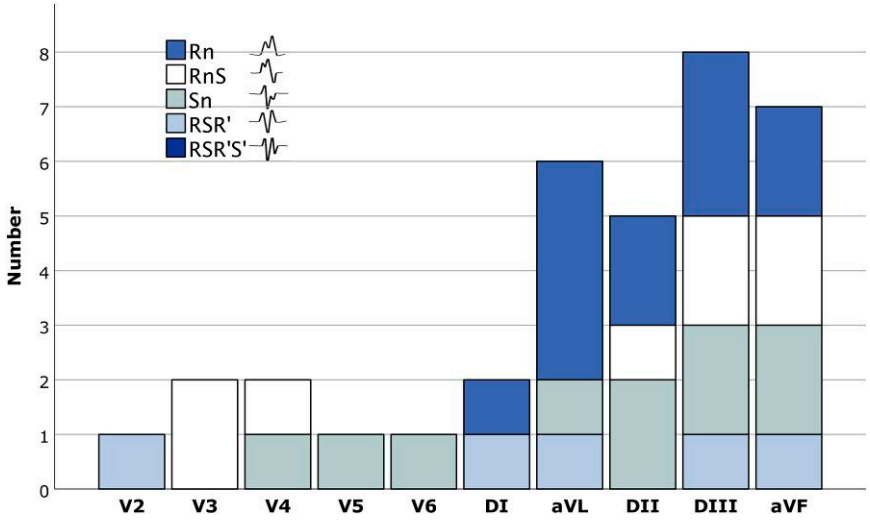


Figure 3 Type of morphology in individuals with at least two leads involved in QRS fragmentation.

Table 5 Electrocardiographic parameters of the individuals with QRS fragmentation in at least two contiguous leads

ECG parameter in FQRS individuals	Total population $n = 10$
HR, b.p.m.	71.5 (68.5–88.25)
P wave, ms	90 (83–103.75)
PR interval, ms	145 (120–170)
QRS, ms	106 (87.5–121)
QT interval, ms	347 (340–391.5)
QTc interval, Bazett	409 (378.1–433.9)
QRS axis, degree	66.5 (42.75–76.5)
Group 1 abnormalities	
Incomplete RBBB	1 (10)
Early repolarization	1 (10)
LVH	2 (20)
RVH	2 (20)
Group 2 abnormalities	
RAD	1 (10)
Complete RBBB	1 (10)
Group 3 abnormalities	
Pathological TWI	1 (10)

Data are given as number (percentage) or median (25th–75th percentile). There are: the basal ECG parameters; the ECG characteristic classified respectively as normal (group 1 abnormalities), borderline (group 2 abnormalities), and abnormal (group 3 abnormalities) according to the international recommendation for ECG interpretation in athletes.[4]
b.p.m., beat per minute; ECG, electrocardiographic; HR, heart rate; LVH, left ventricular hypertrophy; ms, millisecond; RBBB, right bundle branch block; RVH, right ventricular hypertrophy; TWI, T-wave inversion.

most likely representing the sign of an incomplete RBBB, which is known to be common and benign in the young athlete.[27] To the best of our knowledge, the only study that assessed FQRS beyond V1–V2 in athletes is that of Orlandi *et al.*,[14] who examined a population of athletes with a wide age range: they reported a prevalence of FQRS of 7.8% that was correlated with older age and higher degree of LV remodelling. However, information on distribution of FQRS across the ECG leads were not provided.

In our study, we found FQRS in two contiguous leads other than V1–V2 to be very rare, with a prevalence of only 0.5%. Moreover, no association between FQRS and characteristics of the athletes in terms of sex, BMI, type of sport, or training intensity were found. Although only two underwent further examinations, one received a diagnosis of mitral valve prolapse that is a valvular disease that may cause LV scarring.[28] These data suggest that FQRS in a young athletic population in leads other than V1–V2 may potentially be a pathological finding but, similar to LQSRV, its positive predictive value for underlying myocardial disease remains to be investigated.

Study limitations

First, this study is limited by its retrospective nature and the absence of a control group of sedentary subjects. The second limitation is the restricted ethnic diversity of our sample. Although ethnic background was not systematically collected at the time of PPS, we can estimate that the vast majority of enrolled individuals were Caucasian and thus our results may not be generalized to athletic cohorts of different ethnicities. The third is the lack of systematic clinical investigation of all athletes with LQRSV and FQRS. Particularly CMR, which is the only imaging modality that can reliably rule out an underlying LV scar, was performed in only one case. Similarly, thyroid hormones in athletes with LQRSV but no other clinical signs of hypothyroidism were not checked. However, as this was an observational study in a paediatric population, management of young athletes needed to follow the consolidated clinical practice and prescription of investigations only based on LQRSV or FQRS, which are not included among pathological ECG abnormalities by international and national guidelines, was not justified.

Conclusions

In conclusion, we found that maximum QRS < 0.5 mV in all limb leads and FQRS in two contiguous leads other than V1–V2 were rare in

young apparently healthy athletes. Moreover, neither the prevalence of LQRSV nor of FQRS was influenced by the type of sports activity or training load.

At the present state of knowledge, there is consistent evidence that LQRSV are rare and deserve clinical attention particularly when other abnormalities (such as positive family history, cardiovascular symptoms, other ECG abnormalities, or arrhythmias at exercise testing) are present.[6] Further studies are needed to better define the positive predictive value of isolated LQRSV in order to establish whether they warrant prescription of a comprehensive clinical work-up including CMR even when found in isolation. On the other hand, data on FQRS prevalence, predictors, and clinical significance in athletes are preliminary and do not allow to draw similar conclusions until more robust evidence obtained by evaluating other samples of athletes and non-athletes become available.

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Author contribution

A.Z., F.Gi., and F.Gr. conceived and designed the study. O.E.G., L.M., L.B., L.G., S.C., and F.Gr. acquired the data. F.Gr., O.E.G., L.M., D.C., A.Z., and F.Gi. analysed and interpreted the data. F.G., A.Z., and F.Gi. drafted the manuscript. F.Gr., O.E.G., and L.M. carried out the statistical analysis. All authors critically revised the manuscript for important intellectual content. All authors gave final approval and agreed to be accountable for all aspects of work ensuring integrity and accuracy.

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Data availability

The data that support the findings of this study are available from the corresponding author on reasonable request.

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