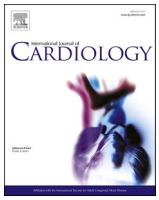
International Journal of Cardiology 447 (2026) 134149

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard



QRS fragmentation on the 12-lead electrocardiogram and its association to physiological cardiac adaptation in elite cyclists

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ARTICLE INFO

Keywords:
QRS fragmentation
Athlete's heart
Cardiac remodelling

ABSTRACT

Background: The relationship between electrocardiographic based QRS fragmentation (fQRS) and physiological cardiac adaptation is not fully understood. We aimed to determine the prevalence of fQRS in elite male and female cyclists, and relationship between fQRS and cardiac structure and function.
Methods: 181 Elite international cyclists underwent their national cycling team's pre-participation cardiac screening. This included a resting 12-lead electrocardiogram and transthoracic echocardiogram. Fragmented QRS was defined as an additional notch >1 mm deep from the peak of the wave within the QRS complex in any lead. We excluded RSR' or RSR'S' patterns in V1–2 to differentiate from bundle branch block.
Results: Fragmented QRS was observed in 73 (41 %) of the cyclists. The most common lead was V1, followed by lead III, aVL and aVF. Of those cyclists with fQRS it was observed in just one lead in 38 cyclists (52 %), two leads in 22 cyclists (30 %) and ≥ three leads in 13 cyclists (18 %). Cyclists with fQRS demonstrated greater left ventricular (LV) diastolic diameter index ($p = 0.008$), mean wall thickness ($p < 0.001$), LV mass index ($p = 0.001$), proximal right ventricular (RV) outflow diameter ($RVOT_{PLAX}$) index ($p = 0.040$), distal $RVOT_2$ index ($p < 0.001$), RV systolic area index ($p = 0.005$), and RV:LV ratio ($p = 0.008$). In addition, cyclists with fQRS had significantly lower RV fractional area change ($p = 0.002$).
Conclusion: Fragmented QRS in athletes is associated with indices of physiological LV and RV adaptation. In isolation, fQRS may not raise concern or initiate any further onward investigations.

1. Introduction

The 'athlete's heart' describes the unique changes in cardiac morphology and function in response to structured exercise training [1,2] and is often characterised by electrical adaptation [3]. An increase in chamber dimension and cardiac mass also occurs at high training volumes in those athletes with high cardio-respiratory fitness [4]. Such changes enable the athlete to generate and sustain a sufficient cardiac output during high performance exercise [5,6]. In this regard, elite cyclists, who engage in very high training volumes of isometric and isotonic exercise activity, often reveal the greatest magnitude of electrical, structural and functional adaptations in comparison to other sporting disciplines [7].

Electrocardiographic based QRS fragmentation (fQRS) is defined as an additional notch in the QRS complex in any of the 12-leads [8]. Our understanding of its relationship to cardiac adaptation in athletes is not fully understood [9]. The prevalence of fQRS appears to be greater in athletes compared to non-athletes, albeit these studies have assessed mixed discipline sports of variable cardio-respiratory fitness [10,11]. However, there is very limited data on differences in cardiac structure and function in athletes with and without fQRS [9,11]. Previous data in non-athletes with structural heart disease have highlighted a relationship between fQRS and myocardial scar with cardiac events [12–14]. Additionally, the visual similarity of an epsilon wave, a major diagnostic criterion specific for arrhythmogenic right ventricular cardiomyopathy (ARVC) [15], means it is often misinterpreted as fQRS [16]. This

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https://doi.org/10.1016/j.ijcard.2025.134149
Received 24 November 2025; Received in revised form 17 December 2025; Accepted 31 December 2025
Available online 1 January 2026

ambiguity stems from the fact that both electrocardiographic based morphologies exist on a spectrum of right ventricle (RV) depolarisation delay [14].

The study aimed to: 1) to determine the prevalence of fQRS in elite male and female cyclists with a 'normal' 12-lead electrocardiogram (ECG) and echocardiogram, and 2) to determine the nature of any relationship between fQRS and indices of cardiac structure and function.

2. Methods

We assessed 192 elite international cyclists who underwent their national cycling team's pre-participation cardiac screening between 2016 and 2024. Participants refrained from strenuous physical activity for 6 h prior to the examination and from consuming alcohol or caffeine within the preceding 24 h. The screening protocol included a health questionnaire, anthropometric measurements, systemic arterial blood pressure, a 12-lead ECG and a 2-D transthoracic echocardiogram that was conducted by British Society of Echocardiography (BSE) accredited sonographers (DO / JM) adhering to BSE national guidelines [17,18]. Participants were included in the study if they showed no evidence of cardiac pathology at the initial screening. This included no history of cardiovascular disease, no cardiovascular medications, a normal 12-lead ECG in line with the International Criteria [3] and a normal echocardiogram [17]. Following this, 181 cyclists were included in the final analysis which included age-matched males ($n = 105$; 23 ± 6 years) and females ($n = 76$, 23 ± 5 years). Ethics approval was obtained from the National Research Ethics Service at London – West London & GTAC Research Ethics Committee (IRAS 169429) with all cyclists providing written informed consent.

2.1. Anthropometry, blood pressure and training volume

Anthropometric assessment included height (meters) (Seca 217, Hannover, Germany) and body mass (kg) (Seca supra 719, Hannover, Germany). Body surface area (BSA) was calculated using the Mosteller equation [19]. Resting blood pressure was assessed with an automated sphygmomanometer (Dinamap 300, GE Medical systems, USA).

The cyclists were classified by their discipline as either endurance (track endurance, road, mountain bike, cyclocross, tandem pilot) or sprint (track sprint, tandem, kilo team, BMX). Training data were provided and utilised to calculate relative exercise intensity as defined by Metabolic Equivalent of Tasks (MET) hours per week in accordance with the 2024 Adult Compendium of Physical Activities [20].

2.2. ECG analysis

ECG assessment was conducted using the SECA CardioPad-2 (Hannover, Germany). Standard indices were extracted from the 12-lead ECG and included heart rate, QRS axis, PR interval, QRS interval and corrected QT interval. Corrected QT interval was calculated using Bazett's formula. Normal and borderline-training related ECG changes were defined in accordance with the International Criteria for Assessment of the 12-lead ECG in Athletes [3].

Fragmented QRS was defined as the presence of an additional notch within a narrow QRS complex (<120 ms), specifically excluding RSR' or RSR'S′ patterns in V1–2. Each qualifying notch was required to occur at a depth greater than 1 mm from the peak of the wave. This pragmatic approach allowed for differentiation of fQRS from incomplete or complete right or left bundle branch block morphologies. Examples of the types of fQRS morphologies are shown in Fig. 1. Fragmented QRS was categorised as either present or absent, the specific ECG lead(s) and the number of ECG leads (1, 2, ≥3) of its occurrence.

2.3. Echocardiography

Echocardiographic images were acquired with a commercially available ultrasound system (Vivid IQ or Vivid E95, GE Medical, Horten, Norway) and a 1.5–4 MHz phased array transducer. Data was stored in raw digital imaging and communications in medicine (DICOM) format and transferred to an offline workstation (EchoPAC, Version 204, GE Healthcare, Horten, Norway).

Left ventricular (LV) linear dimensions were assessed in the parasternal long-axis view in diastole and systole (LVIDd and LVIDs). LV volumes (LVEDV and LVESV) were measured using Simpsons biplane methodology from the apical 4-chamber view (A4C) and 2-chamber view and stroke volume (SV), cardiac output (CO) and ejection fraction (EF) were derived. Left atrial (LA) volume was measured at end systole (LAESV). LV wall thickness was measured at eight locations in the parasternal short-axis view at basal and mid-levels of the anteroseptum, inferoseptum, lateral and posterior walls and mean wall thickness was calculated as an average (MWT) [17]. Relative wall thickness was calculated as (2 x MWT) / LVIDd and LV mass was calculated using the ASE corrected equation [18]. The combination of indexed LV mass and RWT was used to define geometry and was classified as 'normal' if RWT was ≤0.42 in the presence of normal LV mass (≤110 g/m^2 in males, ≤99 g/m^2 in females). A normal LV mass with a RWT >0.42 defined 'concentric remodelling' whilst an increased LV mass with a RWT ≤0.42 defined 'eccentric hypertrophy' and an increased LV mass and a RWT >0.42 defined 'concentric hypertrophy'.

Measurements of RV inflow structure were taken from the modified A4C at end-diastole. These included the RV basal-level diameter (RVD_1), mid-level diameter (RVD_2), and length from the tricuspid annulus base to the apex (RVD_3). The RV:LV ratio was determined by measuring RV and LV end-diastolic diameters in the A4C view. The RV outflow tract (RVOT) was measured in diastole from both parasternal long ($RVOT_{plax}$) and short axis views proximal ($RVOT_1$) and distal ($RVOT_2$). The $RVOT_1$: RVD_1 ratio was calculated to assess the relative sizes of the outflow and inflow tracts. RV function was assessed by measuring RV end-diastolic area (RVDA) and end-systolic area (RVSA) to calculate RV fractional area change (RVFAC). Tricuspid annular plane systolic excursion (TAPSE) was measured using M-Mode in the A4C view. Right atrial (RA) size was measured at end ventricular systole (RAESV) in the A4C view

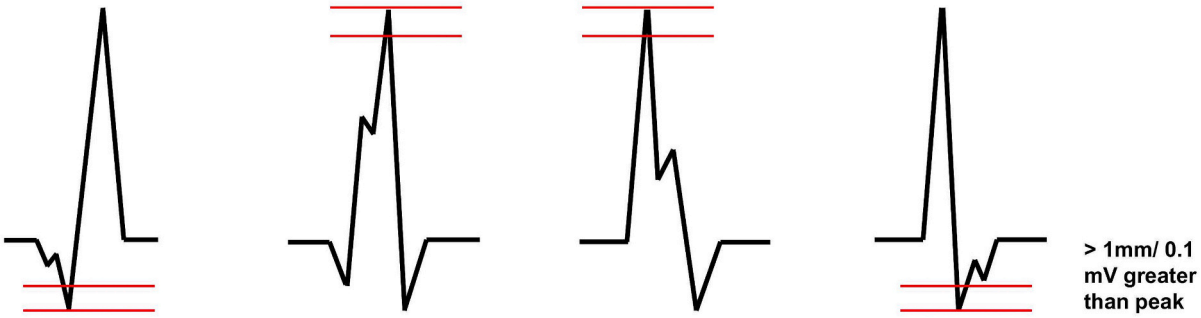

Fig. 1. Examples of different fQRS morphologies.

and the inferior vena cava (IVC) diameter was measured at its maximum in the subcostal view.

All structural measurements were scaled based on the principle of geometric similarity. Linear dimensions were scaled to $BSA^{0.5}$, areas directly to BSA and volumes to $BSA^{1.5}$.

2.4. Statistical analysis

Continuous data are presented as mean ± SD. The normality of distribution for all continuous parameters was assessed using the Kolmogorov–Smirnov test. Differences in continuous variables between cyclists with and without fQRS were subsequently analysed using an independent *t*-test for normally distributed data, and the Mann-Whitney *U* test for non-normally distributed data. For all statistical tests, the alpha value was set at $p < 0.05$. Between group differences in nominal variables (Sex, Normal Training Related Changes) were assessed using the chi-square test. The association between the number of fQRS occurring leads (1, 2, ≥3) and continuous variables were assessed by a one-way ANOVA for normally distributed data, and the Kruskal Wallis test for non-normally distributed data or nominal variables. Following the identification of a significant interaction, a post hoc pairwise comparison incorporating the Tukey or the Dunn's multiple comparison test was performed to investigate the specific differences and assess their statistical significance. Statistical analysis was performed using GraphPad Prism (Version 10.0.0 for Mac, GraphPad Software, Boston, Massachusetts, USA) software.

3. Results

3.1. Demographics

Demographics and training volumes are presented in Table 1. The cohort was predominantly of white ethnicity (96 %), with black (1 %) and mixed (3 %) ethnicity. The mean age for the whole cohort was 23 ± 5 years, with no difference between cyclists with and without fQRS. There was no difference in the number with fQRS in males compared to females ($p = 0.992$).

3.2. Training volume

Training volume was not different between cyclists with and without fQRS. Demographics and training volumes of cyclists categorised by the number of leads (1, 2, or ≥ 3) exhibiting fQRS are presented in Table 4. Training MET hours were significantly different between the different number of leads with fQRS ($p = 0.041$), although post hoc analysis revealed no significant pairwise differences between any of the groups.

3.3. Distribution of QRS fragmentation

There was no difference in the prevalence of fQRS in males compared to females ($p = 0.335$). The ECG characteristics of cyclists with and without fQRS are presented in Table 2. Cyclists with fQRS had significantly greater P-wave (107 ± 14 ms vs 101 ± 14 ms, $p = 0.009$) and QRS durations (100 ± 9 ms vs 92 ± 10 ms, $p < 0.001$) than those without fQRS. Additionally, cyclists with fQRS had a significantly greater occurrence of partial right bundle branch block and early repolarisation compared to cyclists without fQRS ($p < 0.001$).

Fragmented QRS was observed in 73 (41 %) of the cyclists. The most common lead with fQRS was V1 occurring in 43 cyclists (58 % of cyclists with fQRS), followed by lead III in 20 cyclists (27 %), aVL in 16 cyclists (22 %) and aVF in 14 cyclists (19 %) (Fig. 2). Fragmented QRS occurred most frequently in just one lead (52 % of cyclists with fQRS, $n = 38$), followed by two leads (30 %, $n = 22$) and ≥ 3 leads (18 %, $n = 13$).

Of the cyclists with fQRS in a single lead, V1–3 was most frequently displayed (71 %, $n = 27$) while 6 cyclists (16 %) had fQRS confined exclusively to the limb leads. Of the cyclists with fQRS in >1 lead, 15 cyclists (43 %) had fQRS in V1–3 and a limb lead(s), 5 cyclists (14 %) had fQRS in just V1–3 and 10 cyclists (29 %) had fQRS exclusively in the limb leads.

3.4. Association of QRS fragmentation with cardiac structure and function

Cardiac structure and function derived from echocardiography in those with and without fQRS are presented in Table 3. Cyclists with fQRS had significantly greater LVd Index ($p = 0.008$), MWT ($p < 0.001$), LV Mass Index ($p = 0.001$), $RVOT_{plax}$ Index ($p = 0.040$), $RVOT_2$ Index ($p < 0.001$), RVSA Index ($p = 0.005$), and RV:LV ratio ($p = 0.008$). In addition, cyclists with fQRS had lower RVFAC ($p = 0.002$). No differences in cardiac structure or function were observed amongst cyclists categorised by the number of leads (1, 2, or ≥ 3) (Table 3).

Left ventricular geometry derived from echocardiography in those with and without fQRS are detailed in Table 5. Eight (11 % of those with fQRS) cyclists had fQRS in 2 or more anterior leads, with 50 % of these cyclists displaying eccentric LV hypertrophy.

Table 1
Demographic, Anthropometric and Training Volume Characteristics of Elite Cyclists.

Variable		Total sample ($n = 181$)	fQRS ($n = 73$)	No fQRS ($n = 108$)	p value
Gender (n, %)	Male	105 (58)	46 (63)	59 (54)	0.335
	Female	76 (42)	27 (37)	49 (46)	
Age (years)		23 ± 5	22 ± 5	23 ± 6	0.682
Height (m)		1.74 ± 0.10	1.75 ± 0.10	1.74 ± 0.09	0.570
Weight (kg)		70.6 ± 10.6	71.9 ± 11.5	69.7 ± 9.9	0.169
BSA (m^2)		1.84 ± 0.18	1.86 ± 0.19	1.83 ± 0.17	0.227
Training	Duration (years)	12 ± 5	12 ± 5	12 ± 5	0.527
	Days (per week)	6 ± 1	6 ± 1	6 ± 1	0.131
	Hours (per week)	17 ± 4	17 ± 4	17 ± 4	0.619
	MET hours (per week)	221 ± 57	223 ± 56	219 ± 57	0.523

Data are presented as mean ± standard deviation.
Abbreviations: **fQRS,** Fragmented QRS**; BSA**, Body Surface Area; **MET**, Metabolic Equivalent of Task.

Table 2
Electrocardiogram Characteristics in Elite Cyclists with (n = 73) and without Fragmented QRS ($n = 107$).

	fQRS	No fQRS	p value
Electrocardiogram			
P duration (ms)	107 ± 14	101 ± 14	**0.009**
PR interval (ms)	167 ± 14	161 ± 23	0.095
QRS duration (ms)	100 ± 9	92 ± 10	**<0.001**
QT interval (ms)	420 ± 32	414 ± 38	0.181
QT corrected – Bazett (ms)	404 ± 27	403 ± 26	0.764
QRS axis (degree)	59 ± 27	65 ± 22	0.241
Normal training related changes (n, %)			
Sinus bradycardia	42 (58)	56 (52)	0.451
Sinus arrhythmia	6 (8)	4 (4)	0.192
1st degree AV block	4 (6)	5 (5)	0.796
Partial RBBB	15 (21)	0 (0)	**<0.001**
Early repolarisation	38 (52)	16 (16)	**<0.001**
Isolated LVH	22 (30)	35 (34)	0.444

Data are presented as mean ± standard deviation.
Bold Face = p < 0.05.
Abbreviations: **AV**, Atrioventricular; **RBBB**, Right Bundle Branch Block; **LVH**, Left Ventricular Hypertrophy.

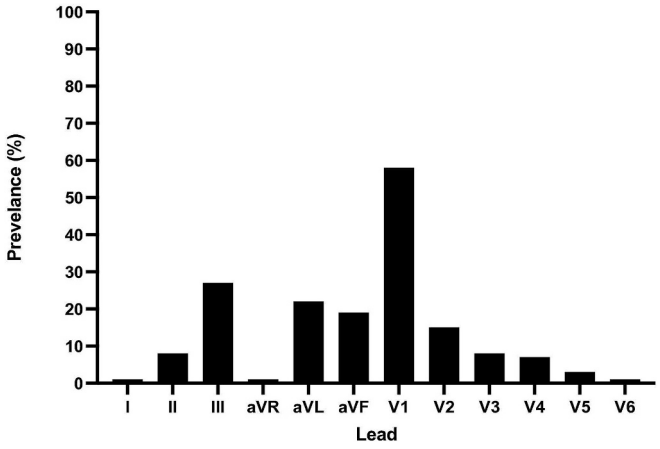

Fig. 2. Prevalence of QRS Fragmentation on a 12-Lead Electrocardiogram in Elite Cyclists.

4. Discussion

To the best of our knowledge this is the first study to describe the prevalence of fQRS and determine the association of fQRS and cardiac structure and function in an elite cyclist population. The main findings of the study were 1) 41 % of cyclists displayed fQRS in at least one lead, demonstrating a high prevalence amongst elite cyclists, 2) cyclists with fQRS had greater RV and LV structural adaptation, 3) cyclists with fQRS had lower RV function (albeit still within normal values) and 4) there were no significant differences in cardiac structure and function between cyclists with fQRS observed in 1, 2 or $\geq$ 3 leads albeit those with higher training intensity are more likely to present with fQRS in greater number of leads.

The absolute prevalence of fQRS within our cohort is similar to the 32–39 % that has been previously reported in a heterogeneous athlete group [9,21,22]. Our elite cyclist cohort represents the pinnacle of athletic adaptation and therefore supports the physiological nature of fQRS. Consistent with previous findings, no significant differences were observed in training volume between those with and without fQRS [9,11,21]. These findings may suggest that the presence of fQRS in athletes represents a more generalised physiological adaptation to sustained intense athletic training. This is supported by Christou and colleagues [10] who demonstrated the presence of fQRS in V1 to be significantly more in athletes compared to non-athletes without cardiac disease.

Similar to our findings, previous studies have highlighted the association of fQRS with greater RV structural adaptation in athletic populations [9,11]. Our findings support this with greater RV structural adaptation observed in elite cyclists with fQRS. Consistent with previous studies, the most common leads for fQRS were V1, lead III and aVF [9,10,21] with an association to increased RV:LV ratio. These findings collectively support the theory that fQRS in V1 arises from a localized conduction delay within the RV myocardium activation, potentially as a consequence of RV enlargement or an absolute slow conduction zone [10,11]. This is further supported by greater QRS duration in those cyclists with fQRS. We also demonstrate lower RVFAC (albeit with absolute values within normal limits) in those cyclists with fQRS providing further evidence to support this theory with lower function seen in those athletes with larger chambers and a potentially enhanced contractile reserve [10,11]. Cyclists with fQRS also presented with a greater prevalence of LV remodelling of both wall thicknesses and cavity size which is in-fitting with previous studies [8,9].

The presence of fQRS in different number of leads and its association to demographics, cardiac structure and function has yet to be explored in elite cyclists. Our findings highlight the presence of fQRS in 1 lead (52 %), 2 leads (30 %) or $\geq$ 3 leads (18 %). Previous research by Christou and colleagues [10] demonstrated an increase in the number of leads with fQRS in a heterogeneous group of athletes which was associated with an increased training age. This finding was not reproduced in our work albeit this may be reflective of the narrow age-range in our cohort. The association between training volume and number of leads with fQRS is an interesting finding and may be indicative of the impact of training intensity and duration as the potential stimulus for this type of electrical remodelling. Further longitudinal studies should aim to establish the

Table 3
Echocardiographic parameters in Elite Cyclists with and without Fragmented QRS.

	fQRS	No fQRS	p value	1 Lead	2 leads	≥3 leads	p value
MWT (mm)	8.5 ± 0.9	8.1 ± 0.9	**<0.001**	8.43 ± 0.87	8.51 ± 0.93	8.61 ± 0.88	0.940
LVIDd index (mm/(m^2)$^{0.5}$)	39.9 ± 2.8	38.8 ± 2.3	**0.008**	39.53 ± 2.65	40.08 ± 3.02	40.37 ± 2.92	0.587
LV mass index (g/m^2)	94.7 ± 18.7	85.8 ± 17.3	**0.001**	92.05 ± 16.94	97.20 ± 48.91	98.07 ± 20.95	0.548
Relative Wall thickness	0.32 ± 0.04	0.32 ± 0.04	0.593	0.32 ± 0.04	0.33 ± 0.04	0.32 ± 0.04	0.907
LVEDV index (mL/(m^2)$^{1.5}$)	58.9 ± 10.1	55.6 ± 9.9	**0.027**	58.07 ± 9.40	61.38 ± 11.95	57.02 ± 8.20	0.409
LV SV (mL)	87 ± 20	82 ± 20	0.086	86 ± 17	89 ± 24	85 ± 21	0.666
CO (L/min)	4.71 ± 1.13	4.58 ± 1.12	0.446	4.79 ± 1.04	4.60 ± 1.27	4.69 ± 1.24	0.833
LV EF (%)	59 ± 5	60 ± 5	0.096	59 ± 6	58 ± 5	59 ± 4	0.612
LAESV index (mL/(m^2)$^{1.5}$)	22.2 ± 5.9	21.8 ± 6.3	0.692	21 ± 6	23.56 ± 6.10	24 ± 6	0.089
RVOT$_1$/RVD$_1$ ratio	0.75 ± 0.11	0.74 ± 0.10	0.315	0.76 ± 0.11	0.74 ± 0.10	0.75 ± 0.12	0.787
TAPSE (mm)	23 ± 4	23 ± 3	0.682	23 ± 4	23 ± 3	22 ± 4	0.626
RV:LV ratio	0.90 ± 0.11	0.86 ± 0.09	**0.008**	0.91 ± 0.11	0.90 ± 0.13	0.89 ± 0.07	0.802
RVFAC (%)	42 ± 7	45 ± 6	**0.002**	43 ± 7	41.9 ± 6.6	40.2 ± 6.7	0.469
RVOT$_{plax}$ index (mm/(m^2)$^{0.5}$)	22 ± 3	21 ± 3	**0.040**	22 ± 2	22 ± 4	23 ± 2	0.328
RVOT$_1$ index (mm/(m^2)$^{0.5}$)	23 ± 3	22 ± 3	0.060	23 ± 2	24 ± 3	23 ± 3	0.743
RVOT$_2$ index (mm/(m^2)$^{0.5}$)	19 ± 2	18 ± 2	**<0.001**	19 ± 2	19 ± 2	19 ± 3	0.409
RVD$_1$ index (mm/(m^2)$^{0.5}$)	31 ± 3	30 ± 4	0.457	30 ± 3	32 ± 4	31 ± 2	0.177
RVD$_2$ index (mm/(m^2)$^{0.5}$)	22 ± 4	22 ± 3	0.355	23 ± 4	22 ± 4	22 ± 2	0.828
RVDA index (mm/m^2)	13 ± 2	13 ± 2	0.086	13 ± 2	14 ± 3	13 ± 2	0.730
RVSA index (mm/m^2)	8 ± 2	7 ± 1	**0.005**	8 ± 2	8 ± 2	8 ± 1	0.877
IVC diameter (mm)	23 ± 5	22 ± 4	0.096	23 ± 5	24 ± 6	23 ± 2	0.571
RAESV index (mL/(m^2)$^{1.5}$)	26 ± 9	24 ± 8	0.175	25 ± 9	27 ± 10	27 ± 6	0.346

Data present as mean ± standard deviation.
Bold Face = $p < 0.05$.
Abbreviations: **fQRS**, Fragmentated QRS; **MWT**, Mean Wall Thickness; **LVIDd**, Left Ventricular Internal Dimensions in Diastole; **LVESV**, Left Ventricle End Systolic Volume; **LVEDV**, Left Ventricle End Diastolic Volume; **SV**, Stroke Volume; **CO**, Cardiac Output; **EF**, Ejection Fraction; **LAESV**, Left Atrial End Systolic Volume; **RVFAC**, Right Ventricular Fractional Area Change; **RVOT**, Right Ventricular Outflow Tract; **PLAX**, Parasternal Long Axis View; **RVD**, Right Ventricle Diameter; **RVDA**, Right Ventricle End Diastolic Area; **RVSA**, Right Ventricular End Systolic Area; **IVC**, Inferior Vena Cava; **RAESV**; Right Atrial End Systolic Volume.

Table 4
Demographic, Anthropometric and Training Volume Characteristics of Elite Cyclists with Single Lead or Multiple Lead Fragmented QRS.

Variable		1 Lead (n = 38)	2 Lead (n = 22)	≥3 leads (n = 13)	*p* value
Gender (n, %)	Male	24 (63)	14 (64)	8 (62)	0.992
	Female	14 (37)	8 (36)	5 (38)	
Mean age (years)		24 ± 6	21 ± 4	21 ± 5	0.336
Height (m)		1.74 ± 0.10	1.75 ± 0.12	1.77 ± 0.07	0.404
Weight (kg)		72.0 ± 11.1	70.4 ± 10.9	74.3 ± 14.6	0.640
BSA (m^2)		1.86 ± 0.19	1.85 ± 0.20	1.91 ± 0.22	0.691
Training	Duration (years)	13 ± 6	11 ± 4	11 ± 3	0.192
	Days (per week)	6 ± 1	6 ± 1	6 ± 1	0.476
	Hours (per week)	17 ± 5	19 ± 3	16 ± 4	0.055
	MET hours (per week)	217 ± 63	245 ± 45	204 ± 47	**0.041**

Data are presented as mean ± standard deviation.
Bold Face = $p < 0.05$.
Abbreviations: **BSA,** Body Surface Area**; SD**, Standard Deviation; **MET**, Metabolic Equivalent of Task.

Table 5
LV Geometry in Elite Cyclists with and without Fragmented QRS.

	fQRS	No fQRS	1 Lead	2 leads	≥3 leads
Normal geometry	57 (77)	100 (93)	35 (85)	15 (68)	10 (77)
Eccentric hypertrophy	16 (23)	7 (6)	6 (15)	7 (32)	3 (23)
Concentric hypertrophy	0 (0)	1 (1)	0 (0)	0 (0)	0 (0)
Concentric remodelling	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

Data presented as total (%).
Abbreviations: **fQRS**, Fragmentated QRS.

specific nature of this relationship.

There was no difference in the overall prevalence of fQRS between sexes, contrary to prior research that identified significant differences in the prevalence of fQRS in males and females [8,9]. Furthermore, there was no difference in the number of leads in which fQRS was present between sexes. Although training-induced remodelling differs between the sexes [23], our data suggest that fQRS appears to be a manifestation of remodelling independent of sex. This challenges the expectation that fQRS would align with other known sex-specific manifestations, such as shorter QRS intervals and lower QRS voltages [24], thus warranting further investigation.

The cause and significance of fQRS amongst athletes is unknown. Fragmented QRS in two contiguous leads has been shown to have no relationship with ventricular ectopic burden on 24-h Holter monitoring in veteran athletes [25]. Athletes with fQRS in V1 have previously demonstrated no differences in the prevalence of any type of exercise-induced arrhythmias, especially in common ventricular arrhythmias consistent with origin from the RVOT, previously described in healthy athletes [9,26]. However, fQRS in at least two consecutive anterior leads has shown to be significantly more common amongst those who subsequently died suddenly in relation to exercise compared to those who suffered sudden cardiac death during rest, or amongst the non-athletic population [27]. Athletes with persistent fQRS in at least one lead upon follow up has also been associated with a higher prevalence of cardiac pathology compared to athletes without fQRS at the initial screening [28]. Fragmented QRS is frequently observed in patients with coronary artery disease, reflecting myocardial damage and serving as a prognostic indicator [29,30]. Ischemic heart disease, myocardial scarring, and cardiac hypertrophy have been identified as autopsy findings frequently associated with exercise-related sudden cardiac death [31]. Crucially, however, this study did not include any fQRS data pre mortem [31]. Although these data are concerning in those individuals harbouring cardiac disease our population of apparently healthy elite cyclists highlights the challenges associated with interpreting fQRS in pre-participation screening. Four of the eight cyclists with fQRS in at least two anterior leads had eccentric LV hypertrophy with no evidence of functional abnormalities. In addition, lateral lead fQRS is often associated with pathological substrates [14,32,33], yet our data corroborate previous findings suggesting a near absence of this in athletes [10,21]. These findings further support more marked physiological adaptation and are reassuring. However, this is only a small proportion of the overall cohort, therefore detailed assessments in larger populations should be a focus of future studies. The high prevalence of fQRS in our study raises questions of potential type I errors from those previous studies that have focused on small populations with high numbers of potentially confounding factors and the associated risk of co-linearity. Based on the lack of known sensitivity of fQRS for underlying disease, international guidelines for ECG interpretation in athletes do not identify this as borderline or abnormal and based on the physiological associations highlighted here we also recommend that the presence of fQRS in isolation in elite athletes should not raise concern or initiate any further onward investigations.

4.1. Limitations

All cyclists had a negative pre-participation cardiac screening in accordance with national and international guidelines. The fact that we are unable to assume 100 % sensitivity of cardiac screening, we can therefore not fully exclude quiescent cardiac disease. Screening was conducted during different training blocks therefore seasonal variation may confound this data. Additionally, we did not account for the influence of deep inspiration on the electrical axis of the heart, which results in the reduction of the number of leads with fQRS [10]. That aside, the high prevalence of fQRS and associations to physiological cardiac adaptation also supports fQRS as a physiological phenomenon. Further studies should aim to establish the association of fQRS to myocardial characteristics using cardiac magnetic resonance imaging. Furthermore, future longitudinal studies are warranted to assess changes in fQRS appearance on follow up and establish any subsequent alterations in cardiac structure and function.

Our cohort is homogenous for ethnicity and sporting discipline and although this should be considered a strength for the internal validity of our work it limits the external generalisability across other sporting and ethnicity.

5. Conclusions

Fragmented QRS is a frequent finding amongst elite cyclists and is associated with global physiological cardiac remodelling. Our findings support the interpretation of fQRS as a benign, physiological adaptation to exercise-induced cardiac remodelling. Given the lack of known association with underlying pathology and its exclusion from international guidelines, the presence of fQRS in isolation should not be a cause for concern or warrant further investigation in asymptomatic elite athletes but should raise the awareness of physiological adaptation in affected individuals.

CRediT authorship contribution statement

Michael Goodman: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Aneil Malhotra:** Writing – review & editing, Investigation. **Robert Cooper:** Writing – review & editing, Investigation. **Shaun Robinson:** Writing – review & editing. **Joseph Maxwell:** Writing – review & editing, Investigation. **Florence Place:** Writing – review & editing, Investigation. **Keith George:** Writing –

review & editing, Formal analysis. **Max Knights:** Writing – review & editing, Investigation. **Nigel Jones:** Writing – review & editing, Supervision. **David Oxborough:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Investigation, Data curation, Conceptualization.

Declaration of competing interest

No conflict of interest to declare.

Acknowledgements

We deeply appreciate the contributions of the cyclists and cyclist team of Great Britian.

References

[1] A. Pelliccia, S. Caselli, S. Sharma, C. Basso, J.J. Bax, D. Corrado, et al., European Association of Preventive Cardiology (EAPC) and European Association of Cardiovascular Imaging (EACVI) joint position statement: recommendations for the indication and interpretation of cardiovascular imaging in the evaluation of the athlete's heart, Eur. Heart J. 39 (2018) 1949–1969, https://doi.org/10.1093/eurheartj/ehx532.

[2] P. Naud, E. Guasch, S. Nattel, Physiological versus pathological cardiac electrical remodelling: potential basis and relevance to clinical management, J. Physiol. 588 (2010) 4855–4856, https://doi.org/10.1113/jphysiol.2010.202556.

[3] S. Sharma, J.A. Drezner, A. Baggish, M. Papadakis, M.G. Wilson, J.M. Prutkin, et al., International recommendations for electrocardiographic interpretation in athletes, J. Am. Coll. Cardiol. 69 (2017) 1057–1075, https://doi.org/10.1016/j.jacc.2017.01.015.

[4] D.L. Prior, A. La Gerche, The athlete's heart, Heart 98 (2012) 947–955, https://doi.org/10.1136/heartjnl-2011-301329.

[5] J.D. Maxwell, D. Oxborough, The athletes heart-from acute stimulus to chronic adaptation, Br. Med. Bull. (2025) 153, https://doi.org/10.1093/bmb/ldae021.

[6] B.D. Levine, VO2max: what do we know, and what do we still need to know? J. Physiol. 586 (2008) 25–34, https://doi.org/10.1113/jphysiol.2007.147629.

[7] B. Brown, J. Somauroo, D.J. Green, M. Wilson, J. Drezner, K. George, et al., The complex phenotype of the athlete's heart: implications for preparticipation screening, Exerc. Sport Sci. Rev. 45 (2017) 96–104, https://doi.org/10.1249/JES.0000000000000102.

[8] G. Orlandi, M. Corsi, L. Casatori, L. Stefani, Frequency of fragmented QRS in sports activity: a pilot study, J. Sports Med. Phys. Fitness 62 (2022) 1748–1753, https://doi.org/10.23736/S0022-4707.22.13435-3.

[9] M. Vecchiato, G. Quinto, N. Borasio, S. Palermi, G. Berton, F. Battista, et al., The fragmented QRS complex in Lead V(1): time for an update of the athlete's ECG? J. Cardiovasc. Transl. Res. 17 (2024) 24–32, https://doi.org/10.1007/s12265-023-10448-9.

[10] G.A. Christou, M.A. Christou, K.A. Christou, D.K. Christodoulou, D.N. Kiortsis, Physiological changes in QRS fragmentation in athletes and nonathletes without cardiac disease, J. Clin. Med. (2024) 13, https://doi.org/10.3390/jcm13102741.

[11] P. Ollitrault, A. Pellissier, L. Champ-Rigot, N. Junqua, M. Chequel, E. Reboursiere, et al., Prevalence and significance of fragmented QRS complex in lead V1 on the surface electrocardiogram of healthy athletes, Europace 22 (2020) 649–656, https://doi.org/10.1093/europace/euaa037.

[12] M.A. Haukilahti, A. Eranti, T. Kentta, H.V. Huikuri, QRS fragmentation patterns representing myocardial scar need to be separated from benign Normal variants: hypotheses and proposal for morphology based classification, Front. Physiol. 7 (2016) 653, https://doi.org/10.3389/fphys.2016.00653.

[13] H.K. Terho, J.T. Tikkanen, J.M. Junttila, O. Anttonen, T.V. Kentta, A.L. Aro, et al., Prevalence and prognostic significance of fragmented QRS complex in middle-aged subjects with and without clinical or electrocardiographic evidence of cardiac disease, Am. J. Cardiol. 114 (2014) 141–147, https://doi.org/10.1016/j.amjcard.2014.03.066.

[14] Y. Take, H. Morita, Fragmented QRS: what is the meaning? Indian Pacing Electrophysiol. J. 12 (2012) 213–225, https://doi.org/10.1016/s0972-6292(16)30544-7.

[15] F. Graziano, A. Zorzi, A. Cipriani, M. De Lazzari, B. Bauce, I. Rigato, et al., New diagnostic approach to arrhythmogenic cardiomyopathy: the Padua criteria, Rev. Cardiovasc. Med. 23 (2022) 335, https://doi.org/10.31083/j.rcm2310335.

[16] A.R. Perez-Riera, R. Barbosa-Barros, R. Daminello-Raimundo, L.C. de Abreu, J. Garcia-Niebla, M.J. de Deus Morais, et al., Epsilon wave: a review of historical aspects, Indian Pacing Electrophysiol. J. 19 (2019) 63–67, https://doi.org/10.1016/j.ipej.2019.02.003.

[17] D. Oxborough, K. George, R. Cooper, R. Bhatia, T. Ramcharan, A. Zaidi, et al., Echocardiography in the cardiac assessment of young athletes: a 2025 guideline from the British Society of Echocardiography (endorsed by cardiac risk in the young), Echo Res. Pract. 12 (2025) 7, https://doi.org/10.1186/s44156-025-00069-0.

[18] S. Robinson, B. Rana, D. Oxborough, R. Steeds, M. Monaghan, M. Stout, et al., A practical guideline for performing a comprehensive transthoracic echocardiogram in adults: the British Society of Echocardiography minimum dataset, Echo Res. Pract. 7 (2020) G59–G93, https://doi.org/10.1530/ERP-20-0026.

[19] R.D. Mosteller, Simplified calculation of body-surface area, N. Engl. J. Med. 317 (1987) 1098, https://doi.org/10.1056/NEJM198710223171717.

[20] S.D. Herrmann, E.A. Willis, B.E. Ainsworth, T.V. Barreira, M. Hastert, C.L. Kracht, et al., 2024 adult compendium of physical activities: a third update of the energy costs of human activities, J. Sport Health Sci. 13 (2024) 6–12, https://doi.org/10.1016/j.jshs.2023.10.010.

[21] F. Graziano, O.E. Genta, L. Manfrin, D. Corrado, L. Brusamolin, F. Giada, et al., Prevalence and determinants of low QRS voltages and QRS fragmentation in children and adolescents undergoing sports pre-participation screening, Eur. J. Prev. Cardiol. 31 (2024) 1535–1542, https://doi.org/10.1093/eurjpc/zwae180.

[22] T. Kramer, V. Ventovuori, A. Heinonen, J. Parkkari, M.T. Korhonen, A. Rovio, et al., Prevalence of electrocardiographic markers associated with myocardial fibrosis in masters athletes: a cohort study, BMJ Open Sport Exerc. Med. 10 (2024) e001988, https://doi.org/10.1136/bmjsem-2024-001988.

[23] B.J. Petek, E.H. Chung, J.H. Kim, R. Lampert, B.D. Levine, D. Phelan, et al., Impact of sex on cardiovascular adaptations to exercise: JACC review topic of the week, J. Am. Coll. Cardiol. 82 (2023) 1030–1038, https://doi.org/10.1016/j.jacc.2023.05.070.

[24] F. D'Ascenzi, F. Biella, E. Lemme, V. Maestrini, B. Di Giacinto, A. Pelliccia, Female athlete's heart: sex effects on electrical and structural remodeling, Circ. Cardiovasc. Imaging 13 (2020) e011587, https://doi.org/10.1161/CIRCIMAGING.120.011587.

[25] S. Fyyaz, J. Moon, K. Alfakih, S. Al-Turaihi, R. Bhatia, M. Tome, A. Ujeyl, P. Bulleros, M. Papadakis, G. Parry-Williams, S. Sharma, 202 Qrs fragmentation and low qrs voltages in veteran athletes, Eur. Heart J. (2023) 109, https://doi.org/10.1136/heartjnl-2023-BCS.202.

[26] G. Quinto, M. Vecchiato, N. Borasio, V. Baioccato, A. Gasperetti, F. Battista, D. Neunhaeuserer, A. Ermolao, Prevalence of exercise-induced arrhythmias in young athletes with fragmented QRS pattern in lead V1. Eur, J. Prev. Cardiol. (2022) 29, https://doi.org/10.1093/eurjpc/zwac056.262.

[27] T. Toukola, M.J. Junttila, L.T.A. Holmstrom, M.A. Haukilahti, J.T. Tikkanen, H. Terho, et al., Fragmented QRS complex as a predictor of exercise-related sudden cardiac death, J. Cardiovasc. Electrophysiol. 29 (2018) 55–60, https://doi.org/10.1111/jce.13341.

[28] K. Yamagata, C. Cowie, S. Sharma, A. Malhotra, 7-041 longitudinal follow-up of QRS fragmentation in athletes, Eur. Heart J. (2025) 11, https://doi.org/10.1136/heartjnl-2025-BCS.232.

[29] M.A.E. Haukilahti, L. Holmstrom, J. Vahatalo, J.T. Tikkanen, H.K. Terho, A. M. Kiviniemi, et al., Gender differences in prevalence and prognostic value of fragmented QRS complex, J. Electrocardiol. 61 (2020) 1–9, https://doi.org/10.1016/j.jelectrocard.2020.05.010.

[30] J. Mahenthiran, B.R. Khan, S.G. Sawada, M.K. Das, Fragmented QRS complexes not typical of a bundle branch block: a marker of greater myocardial perfusion tomography abnormalities in coronary artery disease, J. Nucl. Cardiol. 14 (2007) 347–353, https://doi.org/10.1016/j.nuclcard.2007.02.003.

[31] T. Toukola, E. Hookana, J. Junttila, K. Kaikkonen, J. Tikkanen, J. Perkiomaki, et al., Sudden cardiac death during physical exercise: characteristics of victims and autopsy findings, Ann. Med. 47 (2015) 263–268, https://doi.org/10.3109/07853890.2015.1025824.

[32] T. Yamamoto, S. Ogawa, Y. Ide, K. Miyazaki, A. Sunami, Y. Nambu, et al., Fragmented QRS in lateral leads on electrocardiography is associated with cardiac dysfunction and left ventricular dilation in Duchenne muscular dystrophy, Biomedicines (2025) 13, https://doi.org/10.3390/biomedicines13040804.

[33] M.K. Das, D.P. Zipes, Fragmented QRS: a predictor of mortality and sudden cardiac death, Heart Rhythm. 6 (2009) S8–14, https://doi.org/10.1016/j.hrthm.2008.10.019.