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ORIGINAL ARTICLE

Athlete's Heart or Heart at Risk? Cardiac Remodeling and Exercise-Induced Ventricular Arrhythmias in Elite Athletes

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BACKGROUND: Ventricular ectopic beats (VEBs) are frequently observed in athletes, but their clinical significance remains debated. We aimed to assess the prevalence, pattern of exercise-induced VEBs, and their association with exercise-induced cardiac remodeling (EICR) in elite athletes.

METHODS: We analyzed a large cohort of Olympic athletes who underwent comprehensive preparticipation screening, including exercise-electrocardiography test and echocardiography. VEB morphology was classified as common (left bundle branch block, with inferior axis, and fascicular) or uncommon, including polymorphic.

RESULTS: We enrolled 2525 athletes (mean age, 25.7±5.2 years; 45.1% female); 14.8% of athletes had exercise-induced VEBs, more frequently males (16.7% versus 12.4%; P=0.002), with no differences between sport disciplines (P=0.295). The VEB pattern was defined in 283 (ie, 76%), including 135 (48%) common and 148 (52%) uncommon, including polymorphic. Prevalence of common VEBs increased proportionally with the functional capacity (as W/kg), ranging from 16.3% in I quartile to 40% in IV quartile (P<0.0001), while no differences existed in those with uncommon VEBs (P=0.140). Moreover, athletes with common VEBs showed a greater EICR, including a larger right ventricle (with wider right ventricular outflow tract; P=0.014; right ventricular end-diastolic area; P=0.016) and left ventricle (greater left ventricular mass indexed; P=0.037; a higher prevalence of eccentric remodeling; P=0.019). On the contrary, no relationship with cardiac remodeling or exercise capacity was seen in athletes with uncommon VEBs and in those without VEBs.

CONCLUSIONS: Exercise-induced common VEBs in athletes seem to be associated with EICR and superior exercise performance and may represent a benign phenomenon, expression of the pathophysiologic consequences of EICR. Instead, uncommon VEBs were not related to the extent of EICR or the level of exercise performance, suggesting a nonphysiological nature.

GRAPHIC ABSTRACT: A graphic abstract is available for this article.

Key Words: arrhythmias, cardiac ■ athletes ■ cardiovascular diseases ■ exercise ■ ventricular premature complexes

The athlete's heart refers to a set of cardiac adaptations that occur in response to sustained and intensive exercise training.[1] These changes typically include a balanced enlargement of all cardiac chambers with modest increases in wall thickness and enhanced diastolic function.[2–5] In parallel with these structural changes, electrical remodeling is also observed, manifesting as sinus bradycardia, first-degree atrioventricular block, second-degree atrioventricular Mobitz type I, early repolarization patterns, and increased QRS voltage on the electrocardiography.[6,7] These findings are most commonly found in athletes engaged in endurance sports[6] and are aimed to improve cardiac efficiency during effort.[7]

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WHAT IS KNOWN?

- Ventricular ectopic beats are frequently observed in both recreational and elite athletes, yet their true clinical significance and long-term prognostic implications remain debated.
- Exercise-induced ventricular ectopy can present with different morphologies, with common forms often regarded as benign, while uncommon or polymorphic forms may raise greater concern.

WHAT THE STUDY ADDS

- In a large and systematically evaluated cohort of Olympic athletes, 14.8% demonstrated exercise-induced ventricular ectopic beats during preparticipation screening.
- Common ventricular ectopic beats were associated with more pronounced exercise-induced cardiac remodeling and with higher exercise performance, supporting the interpretation that they reflect a physiological adaptation to training.
- In contrast, athletes with uncommon ventricular ectopic beats showed no association with the extent of cardiac remodeling or with exercise capacity, suggesting that these forms are not directly related to the physiological consequences of athletic training and may warrant closer evaluation.

Nonstandard Abbreviations and Acronyms

EH	eccentric hypertrophy
EICR	exercise-induced cardiac remodeling
LBBB	left bundle branch block
LV	left ventricle
LVM	left ventricular mass
RBBB	right bundle branch block
RVOT	right ventricular outflow tract
VEB	ventricular ectopic beat

Ventricular ectopic beats (VEBs) are a common finding (5%–15%) at preparticipation cardiovascular screening in athletes and are of relevant clinical value because they may occasionally be the initial presentation of underlying cardiovascular disease, including arrhythmogenic cardiomyopathies at risk of sudden cardiac death.[8–12]

At present, the occurrence and clinical significance of VEBs during exercise in athletes remains a controversial issue because their prevalence is largely disproportionate to the actual prevalence of cardiovascular diseases at risk, representing, in most instances, a clinical challenge for their correct interpretation and management.[12]

Only a few studies suggest that VEBs may be part of the athlete's adaptation, deprived of clinical consequences, while the majority disregard the physiological nature and consider VEBs, especially those exercise-induced, to be associated with an increased risk of underlying pathological conditions.[9,13–15]

As a consequence, the occurrence of VEBs, specifically those exercise-induced, often prompts further diagnostic investigation, including cardiac magnetic resonance to rule out the presence of an underlying cardiac disease.[12,16]

In this study, we sought to assess the hypothesis that the morphofunctional cardiac changes in athletes may predispose them to the occurrence of VEBs. To this scope, we evaluated the prevalence and patterns of VEBs occurring during exercise in a large cohort of Olympic athletes, stratified by sporting category and their association with specific morphofunctional cardiac parameters.

METHODS

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

Setting

The Institute of Sport Medicine and Science in Rome is the medical institution overseen by the Italian National Olympic Committee whose primary responsibility is the medical evaluation of athletes selected to participate in international competitive events. The study design of the present investigation was evaluated and approved by the Review Board of the Institute of Sports Medicine and Science. All athletes included in this study were fully informed of the types and nature of the evaluation and signed the consent form, according to Italian Law and Institute policy. All clinical data assembled from the study population are maintained in an institutional database. The research methodology used in this investigation underwent scrutiny and approval by the Ethical Committee of USL Rome 1-Sapienza University of Rome (date of approval: 06/03/2024; IRB number: 0208/2024).

Study Population and Clinical Evaluation

We retrospectively enrolled 2525 elite athletes selected to participate in the Olympic Games from London 2012 to Paris 2024, including Summer and Winter Olympic Games. Specifically, athletes were engaged in the 48 different sporting disciplines, grouped according to the 2020 European Society of Cardiology Guidelines on Sport Cardiology[17] in the following.

1. Skill (n=330, 13.1%) including archery, equestrian, golf, shooting, figure skating, sailing, curling, diving, surfing, and equestrian sports.
2. Power (n=783, 31%) including weightlifting, Greco-Roman wrestling, judo, javelin, shot-putting, bob, skeleton, snowboard, swimming (<800 m), alpine skiing, athletics (speed races), and luge.
3. Mixed (n=899, 35.6%) with soccer, fencing, volleyball, basketball, tennis, water polo, rhythmic gymnastics, taekwondo, badminton, beach volleyball, and softball.
4. Endurance (n=513, 20.3%) with cycling, rowing, canoeing, triathlon, long-distance running, long-distance

swimming (>800 m), cross-country skiing, pentathlon, biathlon, Nordic combined, and long-distance skating.

Exclusion criteria included the following: athletes taking regular antihypertensive medications; athletes with known cardiac disease and arrhythmogenic conditions (ie, cardiomyopathies, channelopathies, and arrhythmogenic mitral valve prolapse); and athletes assuming chronic antiarrhythmic drug and ectopic ventricular beats at rest electrocardiography.

From an initial cohort of 2550 athletes, 6 athletes were excluded from the study for structural heart disease (1 arrhythmogenic cardiomyopathy, 3 isolated left ventricular (LV) scars, 1 hypertrophic cardiomyopathy, and 1 significant coronary artery disease). Moreover, 11 athletes were excluded due to ectopic ventricular beats at rest electrocardiography, 2 athletes due to antiarrhythmic drug treatments, 3 due to an arrhythmic mitral valve prolapse, and 3 due to antihypertensive treatments.

Athletes underwent a comprehensive, multidisciplinary preparticipation evaluation, which included a complete physical examination, a full panel of blood tests, resting electrocardiography, transthoracic echocardiography, cycle-ergometer exercise stress test, and 24-Holter electrocardiography monitoring and cardiac magnetic resonance or coronary computed tomography scan, when indicated.[16]

Of the enrolled 2525 athletes, those with VEBs at exercise stress test were submitted to second-level investigations, including 24-Holter electrocardiography monitoring and cardiac magnetic resonance or coronary computed tomography scan, according to clinical indications.[16] All 2525 athletes included in our study had no evident structural heart diseases after first- and second-level investigations and obtained sport eligibility according to Italian law and the specific cardiovascular evaluation protocols for Olympic athletes implemented in our program.

Body height and weight were obtained in each subject, and body mass index was calculated as weight (kg)/height (m^2). Body surface area was derived by the Mosteller formula.[18]

A standard 12-lead electrocardiography was conducted with the subject in a supine position, and interpretation was performed in accordance with international criteria for electrocardiography interpretation in athletes.[6]

VEB morphology was considered a common or uncommon pattern according to previous classification.[16]

Specifically, common VEBs were those with wide QRS (>130 ms), left bundle branch block (LBBB; or right bundle branch block [RBBB]), inferior axis, and fascicular pattern. Fascicular ectopic beats were defined as VEBs with a narrow (QRS, 120–130 ms) and typical RBBB and superior axis configuration, suggestive of the origin from the posterior fascicle of the left bundle branch and VEBs with a narrow QRS (120–130 ms) and typical RBBB and inferior axis configuration, suggestive of the origin from the anterior fascicle of the left bundle branch.[16] Uncommon morphology VEBs were those with large QRS (>130 ms), RBB or LBBB, and superior axis patterns, other than polymorphic or repetitive.[16]

Blood pressure was assessed, as advised, via noninvasive brachial cuff measurement, while the subject was in a supine position at rest.[19] All participants underwent maximal exercise testing on a bicycle ergometer (Cubestress XR400; Cardioline SpA, Milan, Italy) with an incremental protocol until exhaustion.[20] The athlete's functional capacity was expressed as W/kg and arbitrarily grouped into 4 quartiles as follows: I quartile (<2.88 W/kg), II quartile (between 2.88 and 3.30 W/kg), III quartile (between 3.31 and 3.79 W/kg), and IV quartile (>3.79 W/kg).

Echocardiographic Evaluation

The echocardiographic examinations were performed utilizing a GE Vivid E9 ultrasound system equipped with a 4Vc phased array probe (GE Healthcare Vingmed Ultrasound AS, Horten, Norway) or Philips EPIQ 7 (Philips Medical System, Andover, MA). A comprehensive 2-dimensional echocardiographic study was performed, wherein cardiac images were captured in various cross-sectional planes employing established transducer positions.

Measurements of end-diastolic and end-systolic LV cavity dimensions, interventricular septum, and posterior wall thickness were obtained according to the current recommendations.[21,22]

LV mass (LVM) was calculated by the Devereux formula.[9] LV ejection fraction was calculated with the biplane method of disks' summation, that is, the modified Simpson rule.[21] LV diastolic function was evaluated by pulsed-wave Doppler and tissue Doppler imaging, as recommended.[9,23]

Different shapes of LV remodeling were defined based on the measurements obtained, including normal geometry defined as LVM ≤115 g/m^2 in male or ≤95 g/m^2 in female and relative wall thickness ≤0.42; concentric remodeling as LVM ≤115 g/m^2 in male or ≤95 g/m^2 in female and relative wall thickness >0.42; concentric hypertrophy as LVM >115 g/m^2 in male or >95 g/m^2 in female and relative wall thickness >0.42; and eccentric hypertrophy (EH), as LVM >115 g/m^2 in male or >95 g/m^2 in female and relative wall thickness ≤0.42.

Interobserver variability for LV chambers quantification was assessed in a sample of 100 athletes, selecting randomly 1 in every 25 athletes on our database, independently of sex and sporting discipline. Two investigators (G.D.G. and A.P.) blinded measured the same exam. The interclass correlation coefficient was 0.92 (P<0.0001) for interobserver agreement.

Statistical Analysis

Categorical variables were expressed as frequencies, and percentages were compared using the Fisher exact test or the χ^2 test, as appropriate. Normality criteria were checked for any continuous variable, which were presented as mean±SD and compared using the Student t test for independent data if normally distributed. A P value of <0.05 was considered statistically significant. The Dunn test and the pairwise comparison method were used to focus on differences between sports and quartile comparison. The pooled P values for the comparison test of 4 categories were determined, and if <0.05, a pairwise test was performed. Pairwise comparisons were considered significant if the P value was <0.05. Intraclass correlation coefficients were calculated to assess interobserver agreement. Statistical analysis was performed with STATA Statistics for Windows (SE, version 17).

RESULTS

Characteristics of Overpopulation

The overall study population included 2525 Italian athletes, with a mean age of 25.7±5.2 years, 1138 females

(45.1%), mostly Whites (104 Afro-Caribbean, 4.1%), a mean body weight of 73.4±14.9 kg, and a mean body mass index of 23.2±3.1 kg/m^2; of them, 208 were active smokers (8.2%), while 314 (12.4%) had family history of cardiovascular disease. Athletes practiced different sporting disciplines grouped as follows: 330 (13.1%) skill, 783 (31%) power, 899 (35.6%) mixed, and 513 (20.3%) endurance.

At the exercise stress test, 373 (14.8%) had VEBs. Then, we have evaluated medical records to define VEB morphology, which were available in 283 (ie, 76% of all arrhythmic athletes), including 135 (48%) common and 148 (52%) uncommon patterns.

Characteristics of Athletes With VEBs

In those with common VEBs, virtually all, 111 (or 92%) had an LBBB inferior axis morphology, n=13 RBBB inferior axis, and the remaining 11 cases were fascicular. Uncommon morphologies included the following: LBBB horizontal (n=6) or superior axis (n=39) and RBBB superior axis (n=81). Finally, VEBs were polymorphic in 22 cases.

The overall VEB prevalence was higher in male athletes (232, 16.7%) compared with females (141, 12.4%; P=0.002).

Table 1 shows the prevalence of VEBs at exercise stress test, according to different sporting disciplines, types of LV geometry, and incremental functional capacity at exercise stress test, expressed as quartiles of W/kg.

No significant differences were also highlighted in the overall VEB prevalence among different sporting disciplines (P=0.295). However, higher VEB prevalence was found in athletes with cardiac remodeling (ie, EH) compared with those without (ie, normal geometry; 19.4% versus 13.4%; P=0.0003). Moreover, global prevalence of VEBs increased proportionally with a greater functional capacity, ranging from 12.1% in I quartile to 17.8% in IV quartile (P=0.023).

We then performed an individual analysis based on the VEB pattern, and we observed that uncommon (including polymorphic) VEBs presented no significant differences regarding prevalence among different sporting disciplines (P=0.290), different LV geometries (EH versus normal geometry; P=0.586), or different quartiles of exercise performance (W/kg; P=0.140).

On the contrary, distribution of common VEBs was largely different according to functional capacity, that is, W/kg quartiles, increasing significantly from 16.3% in the I quartile, 20.7% in the II quartile, 23% in the III quartile, and 40% in the last quartile (P<0.0001). Indeed, in athletes with common morphology, prevalence of cardiac remodeling (ie, EH) was higher compared with those with uncommon morphology (37% versus 23.6%; P=0.020).

Of relevance, the morphofunctional cardiac features varied according to the level of functional capacity, as expected, including mostly the presence and extent of EH, increasing from 9.2% (n=60) in I quartile to 14.6% (n=91) in II quartile, 22.1% (n=139) in III quartile, and 48.4% (n=301) in the IV quartile (P<0.0001; Table 1).

Table 1. Prevalence of VEB at Exercise Stress Test According to Different Sporting Disciplines, Type of Left Ventricular Geometry, or Functional Capacity, Expressed in Quartiles of W/kg at Exercise Stress Test

Sporting disciplines	Skill	Power	Mixed	Endurance	P_{pooled} value	$P_{pairwise}$ value
N, %	330 (13.1)	783 (31)	899 (35.6)	513 (20.3)		
VEB, n (%)	40 (12.1)	114 (14.5)	143 (15.9)	76 (14.8)	0.295	
LV geometry	**NG**	**CR**	**CH**	**EH**	**P_{pooled} value**	**$P_{pairwise}$ value**
N, %	1913 (75.8)	15 (0.6)	6 (0.2)	591 (23.4)		
Total VEBs, n (%)	256 (13.4)	1 (6.7)	1 (16.7)	115 (19.4)	0.002	NG vs EH, P=0.0003; NG vs CR, P=0.762; NG vs CH, P=0.235; CR vs CH, P=0.480; CR vs EH, P=0.213; and CH vs EH, P=0.172
Common VEB, n (%); n=135	85 (63)	1 (0.7)	0 (0)	49 (37)	<0.0001	
Uncommon VEB, n (%); n=148	112 (75.7)	0 (0)	1 (0.7)	35 (23.6)		
W/kg	**First quartile**	**Second quartile**	**Third quartile**	**Fourth quartile**	**P_{pooled} value**	**$P_{pairwise}$ value**
N, %	652 (25.8)	621 (24.6)	630 (25)	622 (24.6)		
Total VEBs, n (%)	79 (12.1)	84 (13.5)	99 (15.7)	111 (17.8)	0.023	I vs IV, P=0.004; II vs IV, P=0.045; I vs II, P=0.399; I vs III, P=0.062; II vs III, P=0.317; and III vs IV, P=0.312
Common VEB, n (%); n=135	22 (16.3)	28 (20.7)	31 (23)	54 (40)	<0.0001	I vs IV, P≤0.0001; II vs IV, P=0.0006; III vs IV, P=0.002; I vs II, P=0.347; I vs III, P=0.167; and II vs III, P=0.658
Uncommon VEB, n (%); n=148	33 (22.3)	29 (19.6)	43 (29)	43 (29)	0.140	

CH indicates concentric hypertrophy; CR, concentric remodeling; EH, eccentric hypertrophy; LV, left ventricle; NG, normal geometry; and VEB, ventricular ectopic beat.

Comparison of Athletic Cohorts: Common VEBs, Uncommon VEBs, and No VEBs

Finally, Table 2 shows the differences between athletes with common, uncommon, and no VEBs. Namely, athletes with common VEBs reached higher W/kg at exercise stress test (3.73±1 W/kg; P<0.0001) compared not only to athletes with uncommon morphology (3.44±0.8 W/kg) but also to those without VEBs (3.38±0.8 W/kg), while no differences were highlighted between those with uncommon morphology and those without VEBs (P=0.475).

Consistently, athletes with common VEBs showed a superior exercise capacity with a larger prevalence in the IV quartile of W/kg (38.5%) compared with those with uncommon VEBs and no VEBs (ie, 38.5% versus 29.7% and 23.7%, respectively, with P=0.0002 and P=0.099, respectively).

Athletes with common VEBs presented greater LV remodeling characterized by greater LVM indexed (99.5±23.1 versus 94.1±20 g/m² in uncommon and 93.3±20.1 g/m² in those without VEB; P=0.002), in association with a higher prevalence of EH (35.5% versus 22.9% in uncommon; P=0.019 and 22.3% in those without VEBs; P=0.0004). No differences in EH prevalence were instead noted between athletes with uncommon and those with no VEB (P=0.860).

Moreover, athletes with common VEBs consistently also showed right cardiac remodeling, characterized by larger right ventricular outflow tract (RVOT; wider RVOT diameter, 31.2±4.4 mm and P=0.014, compared with those with uncommon morphology; 29.4±4.3 mm and P=0.021, compared with those with no VEBs; 29.3±4.3 mm; larger right ventricular end-diastolic area, 22.4±5.3 mm² and P=0.016, compared with uncommon group: 20.9±4 mm²). No difference in RVOT diameter was instead observed between athletes with uncommon and without VEBs (P=0.878).

DISCUSSION

Our research provides novel evidence supporting the association between cardiac remodeling and the

Table 2. Main Clinical, Anthropometric, and Morphofunctional Parameters Differences Between Athletes With Common or Uncommon VEB

	Common	Uncommon	No VEB	P_{pooled} value	$P_{pairwise}$ value
N, %	135 (47.7)	148 (52.3)	2152		
Age, y	26.6±5	26.9±5.1	25.6±5.2	0.002	C vs UC, P=0.641; C vs no, P=0.032; and UC vs no, P=0.004
Females, n (%)	57 (42.2)	58 (39.2)	997 (46.3)	0.171	C vs UC, P=0.603
Afro-Caribbean, n (%)	8 (5.9)	8 (5.4)	81 (3.8)	0.303	C vs UC, P=0.850
Weight, kg	74.3±14.2	76.7±15.1	73±14.8	0.010	C vs UC, P=0.171; C vs no, P=0.324; and UC vs no, P=0.003
BMI, kg/m²	23.3±2.6	23.7±3.4	23.2±3.1	0.150	C vs UC, P=0.346
SBP, mm Hg	114.1±10.7	116.5±9.5	111.9±11.2	<0.0001	C vs UC, P=0.049; C vs no, P<0.0001; UC vs no, P<0.0001
Rest HR, bpm	60.6±13	58.6±11.4	63.9±13.6	<0.0001	C vs UC, P=0.171; C vs no, P=0.009; and UC vs no, P<0.0001
Endurance, n (%)	34 (25.2)	30 (20.3)	437 (20.3)	0.395	C vs UC, P=0.323
Mixed, n (%)	48 (35.5)	58 (39.2)	756 (35.1)	0.607	C vs UC, P=0.528
Power, n (%)	39 (28.9)	39 (26.3)	669 (31.1)	0.433	C vs UC, P=0.633
Skills, n (%)	14 (10.4)	21 (14.2)	290 (13.5)	0.561	C vs UC, P=0.329
W/kg	3.73±1	3.44±0.8	3.38±0.8	<0.0001	C vs UC, P=0.008; C vs no, P<0.0001; and UC vs no, P=0.475
W/kg, fourth quartile, n (%)	52 (38.5)	44 (29.7)	511 (23.7)	0.0002	C vs UC, P=0.118, C vs.no, P=0.0001; and UC vs no, P=0.099
IVS, mm	9.5±1.2	9.43±1	9.31±1.1	0.117	C vs UC, P=0.559
PWT, mm	9.1±1.3	8.79±1.2	8.88±1.2	0.088	C vs UC, P=0.045; and UC vs no, P=0.349
EF, %	62±5.3	62.8±5.4	63.7±5.3	0.0003	C vs UC, P=0.196; C vs no, P=0.0003; and UC vs no, P=0.049
LVMi, gr/BSA	99.5±23.1	94.1±20	93.3±20.1	0.002	C vs UC, P=0.037; C vs no, P=0.0005; and UC vs no, P=0.609
LVEDD, mm	53.7±4.8	53.6±4.5	52.3±4.4	<0.0001	C vs UC, P=0.806; C vs no, P=0.0004; and UC vs no, P=0.0008
LVEDV, mL	145.2±42.7	140.2±37.5	130.8±38.2	<0.0001	C vs UC, P=0.306; C vs no, P<0.0001; and UC vs no, P=0.004
LAVi, mL/BSA	22.4±6.8	22±6.6	21.4±6.8	0.179	C vs UC, P=0.627
EH, n (%)	48 (35.5)	34 (22.9)	481 (22.3)	0.002	C vs UC, P=0.019; C vs.no, P=0.0004; and UC vs no, P=0.860
RVOT lax, mm	31.2±4.4	29.4±4.3	29.3±4.3	0.072	C vs UC, P=0.014; C vs no, P=0.021; and UC vs no, P=0.878
RVEDA, mm²	22.4±5.3	20.9±4	21.9±5.3	0.056	C vs UC, P=0.016; C vs no, P=0.392; and UC vs no, P=0.028

BMI indicates body mass index; BSA, body surface area; EF, ejection fraction; EH, eccentric hypertrophy; HR, heart rate; IVS, interventricular septum; LAVi, left atrial volume indexed; LVEDD, left ventricular end-diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVMi, left ventricular mass indexed; PWT, posterior wall thickness; RVEDA, right ventricular end-diastolic area; RVOT, right ventricular outflow tract; SBP, systolic blood pressure; and VEB, ventricular ectopic beat.

occurrence of exercise-induced VEBs of common morphology in elite athletes.

First, the prevalence of VEBs during effort is not so rare in athletes, being observed in 14.8% of our large athlete cohort. This finding is in line with part of the available literature. Pizzolato et al[8] showed that the incidence of at least 1 VEB during exercise test in competitive athletes was 14.5%, that is, close to our observation.

In our cohort of healthy athletes, we showed that exercise-induced VEBs with common morphology (mostly LBBB and inferior axis morphology) are significantly associated with the features of the athlete's heart, such as superior functional performance, eccentric remodeling, and specifically RVOT enlargement, suggesting a causal relationship between RVOT remodeling and proclivity to generate these VEBs. This association was particularly true in male athletes, while, in female athletes, it was less evident. This observation reflects the well-established concept that female athletes undergo a less pronounced degree of cardiac remodeling compared with males[24] in response to training.

The concept that VEBs, especially if exercise-induced, might be integrated into the response to chronic endurance training is highly controversial. Previous studies have largely focused on resting VEBs, often including heterogeneous morphologies and different study populations. While some reports suggested that frequent VEBs might be an early indicator of hidden cardiomyopathy, others proposed that isolated, monomorphic, and suppressible ectopy, especially from RVOT, is not associated with underlying disease and is just benign in otherwise normal individuals, including well-trained athletes, and deprived of adverse clinical consequences.[16]

Graziano et al[25] studied 433 healthy competitive athletes, showing that the prevalence and the complexity of ventricular arrhythmias on 24-hour electrocardiography monitoring were similar between athletes and sedentary controls, with no association with sport type, training volume, or sex. Accordingly, no specific VEB pattern was regarded as clearly training-related in their athletic population. In another study by Zorzi et al,[26] the authors stated that the prevalence of VEBs at 24-hour ambulatory electrocardiographic monitoring did not differ between young competitive athletes and sedentary individuals and was unrelated to type, intensity, and years of sports practice. However, both of these studies included nonelite athletes[17] (<10 training hours per week) with only modest or absent extent of training-induced cardiac remodeling. Therefore, it can be inferred that the different prevalence of VEBs between sedentary individuals and elite athletes may be explained by the presence of an evident remodeled athlete's heart. Moreover, the study by Zorzi et al[26] did not analyze the prevalence of the different VEB morphologies, which is the crucial information of our study, where only VEBs with common morphology, but not all other patterns, were associated with features of the athlete's heart.

A previous study by Palatini et al[27] in a cohort of 40 well-trained healthy endurance athletes (20 cyclists and 20 runners) showed a higher prevalence of VEBs in athletes compared with sedentary counterparts, but information regarding the morphology of VEBs was not available. Furthermore, Verdile et al[28] in a previous investigation already suggested that the majority of VEBs occurring during exercise in elite athletes were with LBBB inferior axis morphology, anticipating the current results, without the support of a morphological (echocardiographic) evaluation. Finally, Biffi et al[29] further showed that athletic deconditioning has the effect of reducing or abolishing the presence of frequent and even complex ventricular arrhythmias in Olympic, well-trained athletes.

Importantly, in our study, the prevalence of exercise-induced VEBs increased in parallel with functional capacity and the prevalence of EH, suggesting a dose-response relationship between training load and cardiac remodeling, and may represent a benign phenomenon. This aligns with the pathophysiological hypothesis that chronic volume overload and increased wall stress may facilitate ectopic activity from the enlarged RVOT, without implying the presence of a structural disease. It is worth emphasizing that it is not the type of sport per se that influences the prevalence of arrhythmias but rather the degree of cardiac remodeling itself, highlighting that sport discipline alone is not the only factor capable of affecting cardiac remodeling.[30]

In contrast, polymorphic VEBs, which are generally considered a red flag for underlying arrhythmogenic cardiomyopathies, showed no association with training level, cardiac geometry, or performance indices in our cohort.[16,26,31,32] This reinforces their distinction from the benign RVOT pattern and confirms that such morphologies should not be considered part of the adaptation to training. Rather, they should prompt a patient-tailored investigation, regardless of athletic status. Therefore, exercise-induced VEBs with uncommon morphology, although not attributable to sport-related cardiac remodeling, in our selected population of elite athletes were not always associated with a patent underlying cardiomyopathy or structural heart disease, suggesting that pathological structural changes may occur later in time than arrhythmias, and other factors may be implicated, such as a subtle inflammatory process.[14,33] Therefore, the diagnostic and prognostic significance of exercise-induced uncommon arrhythmias in athletes with structurally normal hearts requires more long-term prospective studies to define the natural history and clinical implications and to determine whether continued surveillance or any restriction is warranted in this subgroup.

However, despite the evidence collected in our study, we do not claim that these arrhythmias with a common pattern have a benign prognosis based on our data alone. Our study is a cross-sectional study, and while our observations align with previous reports suggesting a benign nature of common pattern VEBs in well-trained athletes,[6,16,28] further longitudinal studies are needed to definitively establish their long-term benign clinical significance. The scope of our work is limited to demonstrate that certain arrhythmias, in particular common exercise-induced VEBs, are associated with the athlete's heart phenotype and from a clinical standpoint, and our study emphasizes the importance of morphological characterization and context in the evaluation of VEBs in athletes. Specifically, while common VEBs seem related to training-induced cardiac remodeling and generally may not require intensified surveillance beyond standard evaluation, uncommon VEBs deserve closer clinical attention. In particular, athletes with uncommon morphologies may benefit from tighter follow-up, including repeated echocardiographic examinations and exercise testing, to promptly identify the development of an early underlying cardiomyopathy.

Limitations

Our study presents several limitations. First, the cross-sectional design does not allow us to draw definitive conclusions about the temporal or causal relationship between training, cardiac remodeling, and VEB occurrence. Second, this investigation focused exclusively on exercise-induced VEBs, and thus, our findings cannot be extended to the broad spectrum of arrhythmias occurring at rest or over the total 24 hours, which may have different clinical pathophysiology and clinical implications. Finally, we evaluated Italian elite athletes including mostly Whites; further studies are needed to compare other global ethnic variants.

Conclusions

In conclusion, our study supports the hypothesis that exercise-induced VEBs of common morphology (mostly, LBBB inferior axis) are frequently observed in the setting of sport-induced eccentric cardiac remodeling and increased performance capacity. These arrhythmias may still represent a benign, physiological epiphenomenon of intensive training, integrated into the adaptive spectrum of the athlete's heart. Conversely, polymorphic or other uncommon VEBs are unrelated to training or cardiac remodeling and need closer follow-up monitoring. Further prospective studies are warranted to confirm the natural history and clinical implications and to determine whether surveillance or restriction is warranted in this subgroup.

ARTICLE INFORMATION

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