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Arrhythmias in Amateur Athletes: Warning Sign or Physiological Adaptation?

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Abstract:

The prevalence of arrhythmias detected in physically active adults has increased in parallel with the growing participation of amateur athletes in endurance and high-intensity sports and with widespread use of wearable ECG-capable devices. Exercise training induces autonomic and structural cardiac remodeling that may manifest as bradyarrhythmias or ectopy and is frequently benign. However, long-standing high-volume endurance exercise has also been associated with a higher burden of atrial fibrillation and, in selected individuals, ventricular arrhythmias, raising the clinical challenge of distinguishing physiological "athlete's heart" adaptations from arrhythmic phenotypes that may reflect underlying structural or electrical heart disease. This narrative review synthesizes current evidence regarding epidemiology, proposed mechanisms, ECG interpretation, diagnostic evaluation, detraining, management, and return-to-sport considerations for amateur athletes presenting with arrhythmias. Particular attention is given to atrial fibrillation, premature ventricular contractions, and ventricular tachyarrhythmias, as well as to the role of pre-participation evaluation and shared decision-making.

Keywords: athlete's heart; atrial fibrillation; premature ventricular contractions; ventricular tachycardia; sports cardiology; ECG interpretation; screening; detraining; amateur athletes.

1. Introduction

Exercise is a cornerstone of cardiovascular prevention; yet, a growing body of observational and mechanistic literature suggests that high cumulative exposure to intense endurance exercise may be associated with an increased burden of certain arrhythmias, most consistently atrial fibrillation (AF), and possibly specific forms of ventricular arrhythmias in selected subgroups [1,6-9,20]. The concept is not that exercise is broadly harmful, but rather that, beyond a certain threshold, the interaction between training-related remodeling, autonomic tone, and individual susceptibility may create an arrhythmogenic milieu [1,7,8,15].

Amateur athletes represent a heterogeneous group. Some train recreationally, while others accumulate near-elite training volumes without the medical surveillance typical of professional sports programs. This heterogeneity complicates interpretation of incidental arrhythmias and intensifies the need for practical, evidence-informed diagnostic pathways that differentiate benign adaptations from disease states such as cardiomyopathies or channelopathies [2,4,14,32-34]. This review addresses that challenge by focusing on: (i) physiological remodeling relevant to rhythm disturbances, (ii) common arrhythmias encountered in amateur athletes, (iii) "red flags" suggesting pathology, (iv) diagnostic evaluation and the role of detraining, and (v) management and return-to-sport considerations.

2. Methods: Narrative Review Approach

The cited articles include reviews, consensus statements, cohort/observational studies, and clinically oriented guidance focusing on athlete ECG interpretation, atrial fibrillation in athletes (especially endurance athletes), ventricular ectopy and ventricular arrhythmias, pre-participation evaluation, detraining, and sports participation in inherited arrhythmia substrates [1-34]. The synthesis emphasizes clinically actionable themes relevant to amateur athletes-namely symptom-guided evaluation, morphology- and context-based interpretation of ectopy, and individualized risk assessment incorporating shared decision-making where evidence is limited or uncertainty persists [2,4,14,16,22,27-30,32].

3. Defining the "Amateur Athlete" in a Clinical Context

There is no single universally accepted definition of an amateur athlete; clinically, the term usually refers to individuals engaging in structured training and/or competitions without professional status. This group spans adolescents in community sports, adults undertaking high-intensity interval training, and "masters" endurance athletes with decades of cumulative training exposure [25-28]. Importantly, amateurs may also have traditional cardiovascular risk factors-hypertension, obesity, or obstructive sleep apnea- that independently affect arrhythmia risk and interact with training-related remodeling [2,6,11,27].

Because screening intensity differs between elite and amateur cohorts, arrhythmia detection rates and diagnostic work-up thresholds may vary, and evidence from elite athletes can only be cautiously extrapolated to recreational or semi-competitive populations [12,25-28]. A practical approach is to conceptualize the amateur athlete along two axes: (i) current training load/intensity, and (ii) lifetime training exposure ("exercise dose"), both of which are relevant to AF risk and to interpreting cardiac remodeling [8,10,11,13,15,31].

4. Exercise-Induced Cardiac Remodeling and Arrhythmogenesis

4.1 Autonomic Adaptation and Conduction Phenotypes

Training increases parasympathetic tone and reduces resting sympathetic drive, producing sinus bradycardia and, commonly, first-degree atrioventricular block or Mobitz I (Wenckebach) patterns, particularly at rest or during sleep. These findings generally normalize with exercise due to sympathetic activation [2,14]. Such changes are typically physiological in asymptomatic athletes and in the absence of structural disease markers [2].

4.2 Structural Remodeling: Atrial and Ventricular Changes

Endurance training can enlarge cardiac chambers and increase myocardial mass. Most changes are adaptive and balanced; however, atrial dilation and mechanical stretch are of particular relevance to AF, while remodeling patterns may influence the clinical meaning of ventricular ectopy [7,8,15,31]. Concerns about "too much exercise" have centered on the possibility that, in a subset, chronic high-volume training may promote a cardiomyopathy-like phenotype or arrhythmogenic substrate via inflammation, microinjury, and fibrosis [1,20,21]. The implication for amateur athletes is not automatic restriction but rather a need for stratified evaluation when arrhythmias are complex, symptomatic, or linked to exertion [16,18,20,32].

5. Atrial Arrhythmias in Amateur Athletes

5.1 Epidemiology of AF and the Exercise-Dose Relationship

AF is repeatedly reported as the most common clinically significant arrhythmia associated with endurance sport participation, especially among middle-aged men with long-term exposure [5-8,10,11,13,15]. Observational data support a non-linear association: moderate exercise correlates with reduced AF risk, whereas prolonged, intense endurance training may increase AF incidence ("U-shaped" or "J-shaped" relationship) [6,8,11]. Cohort observations among veteran endurance athletes and older active men have been used to support this concept, emphasizing that risk appears to rise with cumulative exposure and may be influenced by age and sex [10,11].

In practice, amateur athletes may present with palpitations, reduced exercise tolerance, or incidental AF detected by wearable devices. The clinical significance of AF in athletes includes symptoms, performance impact, and long-term consequences such as stroke risk-though athletes often have fewer conventional stroke risk factors; nevertheless, risk assessment must follow standard clinical frameworks [6,8].

5.2 Mechanistic Framework: Triggers, Substrate, and Modulators

Mechanisms proposed for AF in athletes include heightened vagal tone, atrial ectopy, atrial enlargement, inflammation/oxidative stress, and atrial fibrosis [7,8,15]. Reviews describing "AF in endurance athletes" emphasize that both triggers (e.g., ectopy) and substrates (remodeling) are shaped by chronic training [5,7,8]. Additional modulators include alcohol intake, sleep-disordered breathing, hypertension, and stimulant-containing supplements, which may be particularly relevant in amateurs [2,5,6].

5.3 Clinical Phenotype and Presentation

Athlete-associated AF is frequently paroxysmal and may be vagally mediated, with episodes at rest, post-exercise, or at night [6,7,15]. Some athletes report symptoms only during high-intensity sessions due to loss of atrial contribution to ventricular filling or due to rapid ventricular response. The key clinical task is to assess symptoms, hemodynamic stability, and comorbidities while maintaining a high index of suspicion for alternative diagnoses when "atypical" findings appear (e.g., significant structural abnormalities, disproportionate atrial enlargement, or ventricular dysfunction) [2,5,6,31].

6. Bradyarrhythmias in Amateur Athletes: Physiologic vs Pathologic

Sinus bradycardia and benign conduction delays are common manifestations of training-related autonomic adaptation [2,14]. In asymptomatic amateur athletes, these findings often require reassurance rather than restriction. However, evaluation is warranted when bradyarrhythmias are associated with syncope, presyncope, unexplained fatigue, chronotropic incompetence, or high-grade atrioventricular block [2].

The distinction between physiological adaptation and intrinsic conduction system disease may be challenging in older athletes, where age-related fibrosis can coexist with training-induced vagal effects. Reviews addressing arrhythmogenesis in sport highlight conduction tissue disease as one of the arrhythmic phenotypes observed in athletes, emphasizing a careful symptom-based approach rather than reliance on heart rate alone [1,2,30].

7. Ventricular Ectopy and Ventricular Arrhythmias

7.1 PVCs in Athletes: How Common and What Matters?

PVCs are frequently observed in athletes and non-athletes alike, and their mere presence does not equate to pathology. Athletic cohorts assessed with ambulatory monitoring demonstrate that some ventricular ectopy is common, but distributions of burden and morphology help differentiate "common and benign" patterns from potentially abnormal phenotypes [17]. Reviews and proposed diagnostic algorithms emphasize that the clinical interpretation should incorporate: PVC burden, complexity (couplets, triplets, NSVT), morphology (site of origin),

response to exercise, associated symptoms, and the presence of structural abnormalities on imaging [16,18,20,22,23].
A recurring practical point is that morphology and context often matter more than absolute PVC count. For example, outflow tract patterns may be idiopathic, while atypical morphologies or exercise-induced ventricular arrhythmias warrant deeper evaluation [16,17,18,23].

7.2 When Ventricular Arrhythmias Suggest Underlying Substrate

Complex ventricular arrhythmias in athletes may be associated with structural conditions ranging from cardiomyopathies to myocardial scar. Reviews of ventricular arrhythmias in the setting of athletic remodeling discuss how training-related changes can overlap with or mimic pathology, underscoring the importance of advanced imaging (including CMR) when red flags are present [19-21]. Observational work in competitive athletes has examined prevalence and determinants of ventricular arrhythmias and links to underlying substrates, supporting structured evaluation pathways [18].
Clinical red flags include syncope (especially exertional), polymorphic ventricular ectopy, increasing ectopy with exercise, sustained ventricular tachycardia, abnormal ECG markers suggestive of cardiomyopathy, reduced ventricular function, or evidence of myocardial fibrosis [16,18-21].

7.3 Detraining and the PVC “Dilemma”

Detraining has been debated as a means to differentiate exercise-induced phenomena from underlying disease. Discussions specifically addressing whether to detrain in athletes with PVCs acknowledge that ectopy may decrease with reduced training load, but also that regression does not reliably exclude pathology and may not be acceptable or feasible for many athletes [22]. A broader review on detraining as a diagnostic clue similarly cautions that while reversal of adaptive changes can occur, detraining should not replace appropriate imaging and electrophysiological assessment when risk markers exist [30].

8. ECG Interpretation in Athletes: Avoiding Both Over- and Under-Diagnosis

Accurate ECG interpretation is a cornerstone of the athlete evaluation. Contemporary reviews emphasize that many ECG changes in athletes reflect physiological adaptation, and standardized interpretation criteria help reduce false positives while maintaining sensitivity for conditions associated with sudden cardiac death [2]. Practical guidance provides clinicians with structured frameworks to distinguish normal, borderline, and abnormal ECG findings and to decide when further investigations are indicated [14]. Consensus documents continue to refine definitions of abnormal athlete ECG findings and provide recommended downstream evaluation pathways to address diagnostic uncertainty [4].
The practical message for amateur athletes is that ECG interpretation should be performed using athlete-specific criteria and integrated with clinical context. Overdiagnosis can lead to unnecessary anxiety and testing, while under-recognition of truly abnormal patterns can miss cardiomyopathy or channelopathy [2,4,14,32-34].

9. Diagnostic Work-Up: A Stepwise, Context-Based Algorithm

Evaluation of arrhythmias in amateur athletes should be symptom-driven and morphology-driven, escalating from non-invasive assessment to advanced testing when risk markers appear.

9.1 History and Physical Examination

Key elements include symptom characterization (palpitations, presyncope/syncope, chest discomfort), exertional association, recovery-phase symptoms, family history of sudden death or inherited disease, medication/supplement use, alcohol/stimulants, and training “dose” (weekly hours, intensity distribution, lifetime exposure) [1,2,5,6,16,25].

9.2 Resting ECG and Exercise Testing

Resting ECG interpretation using athlete-specific criteria is essential [2,4,14]. Exercise testing helps determine whether ectopy suppresses with exercise (often reassuring) or increases/appears with exertion (more concerning) [16,18].

9.3 Ambulatory Monitoring

Ambulatory ECG monitoring (Holter or extended patch monitoring) quantifies ectopy burden, complexity, and temporal patterns. Multi-lead or 12-lead ambulatory monitoring can improve morphological classification and site-of-origin inference [17,18].

9.4 Cardiac Imaging

Transthoracic echocardiography evaluates chamber size, systolic/diastolic function, and valve pathology. Cardiac magnetic resonance imaging is considered when complex ventricular arrhythmias, abnormal ECG findings, or suspicious phenotypes suggest myocardial scar or cardiomyopathy [18-21,31].

10. Management Strategies and Return-to-Sport Decisions

10.1 Atrial Fibrillation in Amateur Athletes

Management of AF in athletes is challenging because symptom burden and performance goals often favor rhythm control. Reviews on AF in athletes highlight lifestyle and trigger modification (including evaluation for sleep apnea and blood pressure control), along with rhythm-control strategies and consideration of catheter ablation in symptomatic, recurrent AF [5,6,15]. Importantly, treatment should still follow established clinical principles for anticoagulation and stroke prevention based on validated risk assessment, recognizing that athletes may have lower baseline risk but are not risk-free [6,8].

Exercise prescription after AF diagnosis should be individualized. Some evidence and expert guidance suggest that moderating extreme endurance loads may reduce recurrence in some individuals, but rigid universal restrictions are not supported; rather, training should be adjusted based on symptom control and recurrence patterns [6,15,30].

10.2 Ventricular Ectopy and Ventricular Arrhythmias

For PVCs without red flags-especially when monomorphic, low burden, suppressed with exercise, and with normal imaging-reassurance and follow-up may be appropriate [16,17,22]. When arrhythmias are complex or suggest an underlying substrate, management may include targeted treatment and, in selected cases, electrophysiological study or ablation, guided by the suspected mechanism and athlete goals [18-21].

Return-to-sport decisions rely on etiology, arrhythmia severity, and risk of malignant events. Contemporary perspectives emphasize individualized assessment and, where evidence is limited, shared decision-making after thorough evaluation [32].

10.3 Arrhythmias in Cardiomyopathies and Channelopathies: Special Considerations
For athletes with known or suspected inherited arrhythmia substrates or cardiomyopathies, risk stratification and sports participation decisions have shifted from blanket prohibitions toward individualized counseling, risk mitigation, and shared decision-making [32,33]. Emerging literature discusses circumstances in which carefully managed sports participation may be feasible for selected individuals, though this requires specialist evaluation, tailored precautions, and clear communication of residual risk [33]. Channelopathy-focused discussions (e.g., Brugada syndrome and exercise) illustrate evolving perspectives and the need to avoid simplistic rules; the presence of such conditions in an amateur athlete with arrhythmia mandates specialty input [34].

11. Pre-Participation Evaluation and Screening in Amateur Athletes

Pre-participation cardiovascular evaluation aims to identify conditions associated with exercise-related sudden cardiac arrest or death. However, the appropriate scope of screening-especially in amateurs- remains debated [25–28]. Reviews of pre-participation screening discuss potential benefits of early detection but also the harms of false positives and downstream testing. [25,26]. Contemporary analyses emphasize current controversies and future directions, including how best to implement screening pragmatically and equitably [27]. More recent clinical guidance highlights a comprehensive approach combining history, examination, ECG (when appropriate), and follow-up testing guided by findings rather than indiscriminate testing [28]. The practical dilemma of whether athletes may continue training while awaiting resolution of abnormal screening findings is addressed in focused discussions, emphasizing risk stratification and shared decision-making rather than automatic restriction [29].

12. Practical "Red Flags" and a Clinical Heuristic

Across arrhythmia types, the following features should prompt escalation of evaluation in amateur athletes:

- Symptoms: exertional syncope/presyncope, unexplained dyspnea, chest pain during exercise, or persistent reduction in performance [16,18,25].
- ECG abnormalities: patterns classified as abnormal by athlete criteria or consensus statements (e.g., concerning repolarization changes, pathologic Q waves, pre-excitation) [2,4,14].
- Ventricular arrhythmia behavior: increasing ectopy with exercise, polymorphic ectopy, NSVT, or sustained VT [16-21].
- Imaging abnormalities: ventricular dysfunction, disproportionate dilation, or evidence suggestive of scar/cardiomyopathy [18-21,31].
- Family history: sudden cardiac death or known inherited disease [32,33].

This heuristic aligns with proposed algorithms for evaluating ventricular ectopy and supports structured escalation while avoiding unnecessary restriction for clearly benign adaptations [16,22,30].

13. Limitations of the Evidence Base and Future Directions

Much of the athlete arrhythmia literature focuses on elite athletes or specific endurance cohorts, often male-dominant, limiting generalizability to the diverse amateur population [6,10-13,15]. Definitions of training exposure vary, and causal inference is constrained by observational designs [8,11]. Further work is needed to clarify: (i) dose thresholds relevant to AF risk in different sexes and age groups, (ii) the prognostic meaning of specific ventricular ectopy morphologies in amateurs with normal imaging, and (iii) how wearable-detected arrhythmias should be triaged to avoid both under-treatment and over-medicalization [1,6,16,17,27,28].

14. Conclusions

Arrhythmias in amateur athletes span a spectrum from benign manifestations of training-related autonomic adaptation to clinically significant markers of underlying structural or electrical heart disease. Sinus bradycardia and simple conduction delays are often physiological, whereas AF- especially in endurance-trained, middle-aged athletes- may reflect a complex interaction between training-induced atrial remodeling, autonomic modulation, and modifiable risk factors [5–8,10,11,13,15]. Ventricular ectopy is common but requires interpretation grounded in morphology, complexity, exercise response, symptoms, and imaging findings [16-21,23,24].
A stepwise evaluation anchored in athlete-specific ECG interpretation, targeted ambulatory monitoring, and appropriate imaging allows clinicians to identify athletes who can be reassured versus those requiring further investigation or treatment [2,4,14,16- 21]. Management should integrate risk factor modification, arrhythmia-specific therapy, and individualized return-to-sport decisions, increasingly informed by shared decision-making- particularly when inherited substrates, cardiomyopathies, or persistent diagnostic uncertainty are present [29,32-34].

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