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Risk of Arrhythmias in Endurance vs Strength Sports: A Comparative Review

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ABSTRACT

Background. While physical activity is vital for cardiovascular health, high-intensity competitive training follows a "J-shaped" dose-response curve for arrhythmic risk, driven by complex structural and electrical adaptations.

Aim. To contrast the incidence, mechanistic pathways, and diagnostic patterns of arrhythmias in endurance versus strength-trained athletes.

Materials and Methods. Data were synthesized from master endurance athletes, "power" phenotypes, and reference cohorts. The review evaluated diagnostic efficacy across athlete-specific ECG criteria, Holter monitoring, exercise stress testing, and advanced imaging, specifically echocardiography and Cardiac Magnetic Resonance (CMR) with late gadolinium enhancement.

Results. Endurance athletes show a 2.5-fold higher risk of Atrial Fibrillation (AF) compared to controls, attributed to bi-atrial enlargement, fibrosis, and vagal dominance. Conversely, strength athletes exhibit a more benign supraventricular profile, with high muscle strength potentially buffering against bradyarrhythmias. While ventricular ectopy is common in both groups, endurance training is more closely linked to chronic right-ventricular (RV) remodeling and non-ischemic myocardial fibrosis. Modern ECG criteria provide high screening specificity, but CMR is the gold standard for identifying the "concealed scars" that provide a substrate for malignant events.

Conclusion. The "Athlete's Heart" is not a singular entity. Endurance training imposes an "atrial tax" of increased AF risk via volume-load adaptations, whereas strength-focused pressure-load training leads to concentric hypertrophy with fewer arrhythmic consequences.

Regardless of modality, non-ischemic myocardial fibrosis remains the most potent predictor of life-threatening ventricular events.

Keywords: endurance, strength training, athlete's heart, atrial fibrillation, ventricular arrhythmias

Content

1. Introduction

The cardiovascular benefits of regular physical activity are well-established, yet the relationship between high-intensity exercise and cardiac rhythm follows a complex, often "J-shaped" dose-response curve. While moderate exercise reduces cardiovascular mortality, lifelong participation in competitive sports—particularly high-volume endurance disciplines—is associated with a distinct set of structural and electrical adaptations that may paradoxically increase the risk of certain arrhythmias [1–3].

Contemporary research highlights a significant divergence in arrhythmic risk based on training modality. Endurance athletes, especially middle-aged and "master" competitors, exhibit a consistently higher incidence of atrial fibrillation (AF), with a risk approximately 2.5 times higher than non-athletic controls [2, 4]. This is largely attributed to a unique arrhythmogenic substrate characterized by bi-atrial enlargement, fibrosis, and profound parasympathetic (vagal) dominance [1, 5, 6].

In contrast, strength-dominant phenotypes appear to maintain a more benign supraventricular profile. Large-scale cohort data suggest that higher muscle strength may even be protective against clinically significant bradyarrhythmias and ventricular events, without the associated rise in AF risk seen in endurance-adapted hearts [2]. These differences are rooted in the distinct mechanical loads placed on the myocardium: the volume-load of endurance training leads to eccentric dilation, whereas the pressure-load of strength training promotes concentric hypertrophy [3, 7].

While simple ventricular ectopy is common across all athletic disciplines and often benign [8, 9], the identification of high-risk "malignant" substrates remains a clinical priority. In endurance athletes, intense training can lead to chronic right-ventricular (RV) remodeling and non-ischemic myocardial fibrosis, which significantly increases the risk of sustained ventricular tachycardia [10–12].

Because the "athletic heart" often mimics features of cardiomyopathy, current diagnostic protocols rely on a multi-modality approach. This includes:

- Resting ECG using modern athlete-specific criteria [13, 14].
    - Ambulatory Holter monitoring to assess ectopic burden and morphology [15].
- Exercise Stress Testing (EST) to differentiate between benign idiopathic ectopy and exercise-induced arrhythmias linked to concealed myocardial scar [16–18].

The following sections provide a detailed examination of the incidence, mechanisms, and diagnostic patterns of arrhythmias in both endurance and strength athletes, exploring how cumulative training volume and individual susceptibility interact to shape long-term cardiovascular health.

2. Materials and Methods

This comparative review synthesized data from a wide range of athletic populations to evaluate arrhythmic risk, primarily focusing on endurance athletes, such as middle-aged and master competitors in high-volume disciplines like cycling, triathlon, and ultramarathon running, as well as strength athletes representing "power" phenotypes, including weightlifters, powerlifters, and participants in throwing events. To provide a comprehensive perspective, comparative data were drawn from sedentary reference cohorts and large-scale population studies—such as the Swedish conscription cohort of approximately 1.1 million men—while also incorporating data from screening programs involving high-school and elite youth athletes aged 7–16 years to assess baseline physiological adaptations. The review analyzed several diagnostic modalities and criteria used to assess the "Athlete's Heart" and identify arrhythmogenic substrates, beginning with resting 12-lead ECG evaluations based on modern athlete-specific standards, including the International, Seattle, and ESC-2005 criteria, to distinguish physiological remodeling from pathological conditions. Furthermore, twenty-four-hour ambulatory ECG monitoring (Holter) was utilized to quantify ectopic burden and identify complex patterns like non-sustained ventricular tachycardia, while exercise stress testing was employed for risk stratification to differentiate benign idiopathic ectopy from exercise-induced arrhythmias associated with concealed myocardial scar. Cardiac imaging played a central role, with echocardiography used to measure left atrial volume and ventricular dimensions to contrast eccentric and concentric remodeling, and cardiac magnetic resonance with late gadolinium enhancement serving as the gold standard for identifying non-ischemic myocardial fibrosis and focal scar. Key metrics extracted for comparison between endurance and strength disciplines included structural parameters like left atrial volume index, left ventricular mass, and relative wall thickness, alongside electrophysiological markers such as corrected QT dispersion, QRS duration, and early repolarization patterns, as well as autonomic indices like heart rate variability and resting heart rate to assess vagal versus sympathetic dominance. Data collection was systematically organized to evaluate the relationship between training modality and outcomes through dose-response modeling, specifically assessing the "J-shaped" curve relating

training volume to atrial fibrillation risk and categorizing findings by mechanical load type—volume-load versus pressure-load. Finally, the review synthesized results from meta-analyses and longitudinal cohorts using a statistical threshold for clinical significance typically set at $p < 0.05$, utilizing various computational tools and software for meta-regression of epidemiological data and computational modeling to simulate the impact of anatomical changes on ECG leads.

3. Results

3.1. Arrhythmia Incidence in Endurance Athletes

3.1.1. Atrial fibrillation and supraventricular tachycardias

Endurance athletes, especially middle-aged and "master" competitioners, exhibit a consistently higher incidence of atrial fibrillation (AF) than non-athletic controls. A recent systematic review and meta-analysis found athletes have about a 2.5-fold higher likelihood of developing AF, with the greatest risk in younger (<55 years) athletes and in mixed-sport disciplines, although endurance sports also confer excess risk [2]. Cross-sectional imaging/Holter cohorts of master endurance athletes report AF prevalences around 30% versus 0% in non-athletic controls [4]. Former professional cyclists followed decades after retirement similarly show more AF/flutter and sinus node disease than matched controls, indicating that AF risk persists beyond the competitive years [19].

Mechanistically, endurance training promotes atrial remodeling (enlargement and fibrosis), heightened vagal tone and frequent atrial ectopy, all of which facilitate AF and other supraventricular tachycardias (SVTs) [1, 3]. Animal models demonstrate that long-term intensive exercise induces bi-atrial fibrosis and increases AF susceptibility independent of ventricular disease [1, 5]. In master ultramarathon runners, nearly all athletes display premature atrial beats over 24 hours, but sustained supraventricular tachycardias and exercise-induced AF during a single event are uncommon, suggesting a high background burden of atrial ectopy with relatively infrequent acute SVT episodes [20]. Observational data in athletes with paroxysmal AF show that recurrence is temporally clustered during and within 3 hours after training, implicating both sympathetic activation and post-exercise vagal rebound as triggers [6].

3.1.2. Ventricular ectopy and potential clinical significance

The incidence of ventricular ectopy and non-sustained ventricular tachycardia (NSVT) in well-screened endurance athletes is not clearly higher than in the general population, and much

of it appears idiopathic and benign. In a case-control Holter study of middle-aged competitive endurance athletes versus sedentary controls, ~25% in each group had frequent PVCs or complex ventricular arrhythmias, with no association between burden and training hours or years of activity. Morphologies were predominantly infundibular or fascicular, typical of idiopathic benign ectopy [8].

However, several cohorts highlight a subgroup with clinically important ventricular arrhythmias (VAs). Master endurance athletes show higher NSVT prevalence (≈9%) than non-athletic controls (≈1%) on Holter monitoring [4]. Prospective loop-recorder studies in veteran male cyclists/triathletes report VAs in about 20–24% of athletes, mostly NSVT; sustained VT occurred in ~3% [9, 10]. In the VENTOUX cohort, focal non-ischemic myocardial fibrosis on CMR was present in nearly half of asymptomatic athletes and was strongly associated with incident VAs (HR ≈4.7), including all cases of sustained VT [10]. Separate work links VAs in endurance athletes to right-ventricular dysfunction during exercise and chronic RV remodeling consistent with an "exercise-induced arrhythmogenic phenotype" [9, 11, 12]. These data suggest that while simple ectopy is common and usually benign, the combination of complex VAs, structural remodeling or fibrosis, and abnormal blood-pressure responses may identify athletes at higher risk of malignant arrhythmias and sudden cardiac death [9, 11, 12, 21].

3.1.3. Dose-response relationship with training volume

Epidemiological and experimental data support a J-shaped dose–response between endurance training volume and arrhythmia risk. Moderate physical activity is protective, but very high lifelong training loads increase AF incidence [1–3]. Meta-regression indicates that younger, high-level athletes and those in mixed or high-dynamic sports bear the greatest AF burden (8.). Observational endurance cohorts show AF prevalence rising with age and cumulative lifetime training hours, and remaining elevated even after retirement, despite partial reversal of ventricular remodeling [1, 4, 19].

Animal work in mice subjected to graded daily swim durations demonstrates a clear dose–response: prolonging daily intense exercise (≥180–240 min/day) progressively increases vagally mediated bradycardia, atrial hypertrophy, fibrosis, inflammation, and AF inducibility, while ventricular remodeling remains physiological and non-arrhythmogenic [5]. The exercise dose required to provoke AF vulnerability in this model parallels the MET-hours per week linked to AF in human endurance athletes [5].

For ventricular arrhythmias, current data are more equivocal. Holter and loop-recorder studies have not identified a simple linear relationship between prospective weekly training load and VA incidence or timing; athletes with and without VAs often report similar exercise volume and intensity [8, 22]. Instead, VA risk appears to depend on the interaction between cumulative exposure and individual susceptibility to adverse remodeling, particularly RV dysfunction and non-ischemic fibrosis [9–12].

A comparative overview of patterns and determinants of atrial and ventricular arrhythmias in endurance athletes is presented in Table 1.

Table 1. Patterns and determinants of atrial and ventricular arrhythmias in endurance athletes.

Aspect	**Main findings**	**Citations**
Atrial Fibrillation incidence	~2–5× higher in endurance/mixed-sport athletes vs controls; persists after retirement	[1, 2, 4, 19]
Supraventricular ectopy	Very common; sustained SVT and acute AF during single events relatively rare	[1, 4]
Ventricular ectopy burden	Similar in athletes and controls; often idiopathic infundibular/fascicular	[8, 19]
Clinically important Ventricle Arrhythmias	NSVT 9–22%, sustained VT ≈3%; strongly linked to myocardial fibrosis and RV dysfunction	[4, 9, 10]
Dose–response	J-shaped for AF with high lifetime volume; dose–VA link less linear, mediated by remodeling	[1, 2, 19]

3.2. Arrhythmia Incidence in Strength Athletes

Most available work on athletes and arrhythmias aggregates endurance, mixed, and power/strength sports, so data specific to pure strength athletes (e.g., weightlifting, powerlifting, throwing events) are limited. Where possible, this section highlights findings related to strength or "power" modalities or to muscle strength as a phenotype; when only mixed cohorts are available, this is stated explicitly.

3.2.1. Supraventricular arrhythmias and conduction changes

Evidence linking muscle strength (as a proxy of strength-oriented phenotype) to arrhythmic risk comes primarily from a very large Swedish conscription cohort (≈1.1 million men, median age 18.2 years) with >26 years' follow-up. Higher handgrip strength in late adolescence was associated with a lower long-term risk of overall arrhythmia, driven particularly by reduced risk of bradyarrhythmia and ventricular arrhythmia, while not increasing atrial fibrillation/flutter risk [23]. In contrast, very high cardiorespiratory fitness (a marker of endurance adaptation) showed a U-shaped association with overall arrhythmias, largely via higher atrial fibrillation/flutter [23]. These divergent patterns suggest that a strength-dominant profile is not associated with the marked supraventricular arrhythmia excess seen in high-volume endurance athletes.

Across cohorts of competitive athletes undergoing pre-participation screening, clinically relevant supraventricular arrhythmias are infrequently reported, and bradycardia or first-degree AV block are typically not counted as adverse outcomes [16, 24, 25]. In young athletes undergoing stress testing, the focus of additional evaluation was predominantly on ventricular rather than supraventricular arrhythmias [16]. Similarly, population-based analyses emphasize atrial fibrillation risk in endurance-adapted hearts, with bradyarrhythmia interpreted largely as a manifestation of augmented vagal tone [23]. Direct comparative data on atrial fibrillation incidence between strength-specialized and endurance athletes are lacking, but the large population-level finding that higher muscle strength is neutral for atrial fibrillation/flutter while protective for bradyarrhythmia is consistent with a relatively benign supraventricular profile in those whose training is strength-oriented rather than endurance-dominated [23].

Conduction changes associated with the athletic heart—sinus bradycardia, first-degree AV block, and early repolarization—have been described across adolescent and young adult athletes of mixed sport types [25, 26]. Early repolarization patterns, often linked to vagal predominance, are common (≈22% in adolescent competitors) but in that setting correlate

mainly with left-ventricular hypertrophy and wall thickness rather than with a higher burden of ventricular ectopy, and were not associated with adverse arrhythmic events during follow-up [26]. No study isolates pure strength athletes, but there is no indication that strength-focused training magnifies supraventricular arrhythmia or malignant conduction disturbance beyond what is seen in other sport profiles.

3.2.2. Ventricular arrhythmias and significant events

Most detailed characterizations of ventricular arrhythmias (VAs) in athletes involve endurance or mixed-sport cohorts, but they help frame expectations for strength athletes. In multiple Holter-based studies of young competitive athletes, the prevalence and complexity of VA (couplets, non-sustained VT) did not differ from sedentary controls and were not related to sport type, intensity, or years of training [24, 25]. In 433 competitive athletes and 261 sedentary controls, >90% in both groups had ≤10 premature ventricular beats (PVBs)/24 h and no complex VA; higher PVB burdens correlated with age, not training load, and VA morphology did not differ by sport type [25]. In 288 athletes vs 144 sedentary subjects, the proportion with >10 PVBs or ≥1 complex VA was similarly low and indistinguishable between groups; no association with type or intensity of sport was found [27]. These data indicate that mere participation in competitive sport—including power and mixed disciplines—does not by itself increase VA incidence in the absence of structural disease.

A comparative overview of patterns of the ventricular arrhythmias across athletic phenotypes is presented in Table 2.

Table 2. Patterns of ventricular arrhythmias across athletic phenotypes.

Context/Phenotype	Key Ventricular Arrhythmias/ Structural Features	Relevance for strength-type athletes	Citations
Young competitive athletes, mixed sports	Low VA burden; similar to controls; unrelated to sport type/intensity	Suggests no generalized VA excess purely from training	[24, 25]
High-dose athletes with VA	Impaired RV and LV function,	Phenotype described mainly in	[28, 29]

	myocardial fibrosis, high rate of life-threatening events	high-volume endurance; not clearly linked to strength sports	
Large conscription cohort (exercise capacity vs. muscle strength)	Higher strength → lower ventricular arrhythmia risk; high endurance capacity not protective	Strength-dominant phenotype appears VA-protective at population level	[23]

When VAs occur in athletes, prognosis depends critically on morphology and underlying substrate. In large cohorts of competitive athletes referred for complex VAs, those with "common" idiopathic outflow-tract or fascicular morphologies and no structural abnormalities had benign outcomes; all serious events occurred in those with "uncommon" morphologies and structural disease, particularly non-ischaemic LV scar on cardiac MRI [16, 28, 29]. Isolated non-ischaemic LV scar with a subepicardial/mid-myocardial stria pattern in competitive athletes—primarily involving the lateral wall—is strongly linked to malignant VAs and sudden death, whereas spotty insertion-point enhancement behaves more benignly (38.). Similarly, elite athletes with VAs and no identifiable genetic cardiomyopathy show impaired right-ventricular function and more myocardial fibrosis than healthy athletes, and more than half have experienced life-threatening ventricular events (aborted arrest, sustained VT, or appropriate implantable cardioverter-defibrillator (ICD) therapy); LV mechanical dispersion independently predicts these events [30].

Although these detailed phenotypes come predominantly from endurance or mixed-sport samples, they highlight that in any strength athlete with complex or exercise-worsened VAs, cardiac MRI to detect non-ischaemic scar and careful morphologic analysis of VA are crucial, because arrhythmic risk is dictated far more by substrate than by training mode per se [16, 28–30]. Population-based data suggesting lower ventricular arrhythmia risk with higher muscle strength [23] support the view that, in the absence of such substrate, pure strength training is unlikely to carry the same VA risk profile reported in high-dose endurance sport.

Significant events such as sudden cardiac death or aborted arrest remain rare among screened young competitive athletes of all sport types. In a large Italian pre-participation

programme (>22,000 children, mixed sports), only one resuscitated arrest occurred over ≈7.5 years, corresponding to ≈0.6 events per 100,000 athlete-years, despite active efforts to identify ventricular arrhythmogenic substrates including non-ischaemic LV scar [31]. These low event rates likely apply to strength athletes as well, though direct sport-specific incidence data are not available.

3.2.3. Differences in autonomic modulation patterns

Direct studies of autonomic modulation (e.g., heart-rate variability, baroreflex sensitivity) comparing strength vs endurance athletes in relation to arrhythmia are scarce. However, indirect inferences can be drawn from arrhythmic patterns linked to vagal and sympathetic tone.

High-level endurance training is strongly associated with resting sinus bradycardia and features of high vagal tone, which are often accompanied by increased atrial fibrillation and clinically significant bradyarrhythmias in observational cohorts [4, 23]. Large-scale data show a U-shaped relation between exercise capacity and arrhythmia, driven by higher atrial fibrillation/flutter and an elevated risk of clinically significant bradyarrhythmia at very high endurance capacity levels [23]. These observations are consistent with a pattern of pronounced parasympathetic predominance and atrial stretch/remodelling in endurance-adapted hearts.

In contrast, higher muscle strength is associated with lower long-term risk of bradyarrhythmia and ventricular arrhythmia, without an accompanying rise in atrial fibrillation/flutter [23]. This suggests that individuals with a strength-dominant phenotype may exhibit a more balanced autonomic profile and less atrial structural remodelling than high-endurance counterparts. Studies of early repolarization in adolescent athletes, a pattern often linked to heightened vagal tone, show that while early repolarization is common and associates with concentric LV remodeling and lower heart rate, it does not confer an increased VA burden in that population [26]. Because early repolarization and training-related bradycardia arise across several sport types, not exclusively endurance, they likely reflect shared features of athletic conditioning rather than a strength-specific autonomic state.

Moreover, pre-participation screening cohorts that stratify athletes by sport type do not find systematic differences in VA burden or morphology attributable to particular disciplines, including power/skill sports, after accounting for age and structural disease [21, 25, 31]. This again supports the idea that autonomic modulation patterns relevant to arrhythmogenesis are driven more by individual physiology and cumulative exercise dose than by the mere classification as a strength vs endurance athlete.

Overall, available population-level data imply that a strength-dominant phenotype is not associated with the atrial fibrillation–prone, high-vagal autonomic milieu typical of life-long high-volume endurance training, and may even confer a relative reduction in clinically important brady- and ventricular arrhythmias [23]. However, detailed heart-rate variability or autonomic studies specifically in strength athletes and directly linked to arrhythmic endpoints remain a clear research gap.

3.3. Mechanistic Insights

3.3.1. Structural remodeling

Endurance training produces marked atrial and eccentric ventricular enlargement. Elite and master endurance athletes show larger left atrial (LA) volumes and biventricular dilation with preserved or slightly reduced ejection fraction, consistent with eccentric LV remodeling driven by high-volume preload [4, 12, 32, 33]. LA volume index is significantly greater in endurance than in strength athletes, and training type and duration independently predict LA size [34]. Longitudinal data in formerly sedentary adults confirm that 1–2 years of high-intensity endurance training induces progressive LV and LA dilation, with LA enlargement outpacing LV changes over time [33, 35, 36]. LA enlargement often persists even after years of detraining, suggesting partially irreversible structural remodeling, potentially mediated by fibrosis [4, 37].

By contrast, strength athletes predominantly exhibit concentric LV changes with limited atrial dilation. Powerlifters and bodybuilders show increased LV mass, wall thickness, and relative wall thickness with only modest increases in LA size compared with endurance athletes, indicating pressure-load–driven concentric hypertrophy and smaller chamber expansion [7, 34]. Large sport-specific echocardiographic series report eccentric hypertrophy as typical of high-dynamic (endurance) sports, whereas concentric remodeling or hypertrophy is rare and mainly associated with sports with a substantial static component, aligning with the strength-training phenotype [27, 38].

3.3.2. Autonomic balance

Endurance training is associated with parasympathetic predominance. Resting sinus bradycardia, sinus arrhythmia, junctional escape rhythms, and first-degree AV block are common in endurance athletes and reflect high vagal tone [39]. Experimental models of chronic endurance exercise and human HRV studies show reduced low-frequency/high-frequency ratio and increased indices of vagal modulation, indicating sustained parasympathetic dominance at rest [5].

In strength athletes, autonomic balance appears more sympathetically weighted or mixed. Cross-sectional HRV assessment in elite track-and-field athletes found no major autonomic differences between endurance- and power-trained groups once overall training load was accounted for, suggesting less extreme vagal predominance in power/sprint disciplines [40]. Strength-trained cohorts often exhibit higher resting blood pressure and concentric LV geometry, compatible with greater sympathetic and pressure-load influences compared with endurance athletes [7, 38].

3.3.3. Electrophysiological Substrates

Endurance training can create a complex arrhythmogenic substrate involving fibrosis, inflammation, and repolarization changes. CMR studies show a higher prevalence of focal late gadolinium enhancement, typically at RV insertion/hinge points, and higher extracellular volume in heavily trained endurance athletes, consistent with diffuse myocardial fibrosis [4, 12, 32]. Animal and human data link cumulative high-intensity endurance exposure to atrial and right-ventricular fibrosis, local inflammation, and mechanical stretch, which modify conduction and repolarization and increase vulnerability to atrial fibrillation and ventricular arrhythmias [5, 9, 12, 41]. Endurance athletes frequently exhibit early-repolarization patterns, high LV voltages, and bradyarrhythmias on ECG, reflecting both autonomic and structural remodeling [9, 39].

Strength training yields a different electrophysiologic profile, dominated by concentric LV hypertrophy and altered repolarization heterogeneity. Strength-trained athletes have increased LV wall thickness and mass, with higher corrected QT dispersion that correlates with hypertrophy, indicating more inhomogeneous ventricular repolarization and a potential substrate for ventricular arrhythmias despite preserved systolic and diastolic function [7]. Computational modeling supports that concentric hypertrophy, even without intrinsic ionic remodeling, prolongs QRS duration and alters amplitudes across all 12 ECG leads, thereby increasing dispersion of depolarization and repolarization that could facilitate reentry [42].

Overall, endurance training favors dilated, fibrotic, and vagally modulated atrial and right-ventricular substrates, whereas strength training favors pressure-load–driven concentric LV hypertrophy with repolarization heterogeneity; both patterns may interact with individual predisposition to shape electrophysiologic risk [4, 5, 7].

3.4. Diagnostic Patterns

This chapter outlines how different diagnostic tools—resting ECG, ambulatory monitoring, and exercise testing—are used in athletes, with emphasis on sensitivity, specificity, and modality performance in distinct athlete populations.

3.4.1. Resting ECG in Athlete Screening

Large preparticipation screening programs consistently show that adding a 12-lead resting ECG to history and physical examination substantially improves detection of conditions associated with sudden cardiac death (SCD) compared with history/physical alone [13, 14]. In 3620 high-school athletes, ECG had sensitivity 87.5%, specificity 97.5%, and positive predictive value (PPV) 13.6% for SCD-associated disease, whereas the American Heart Association 14-point evaluation had sensitivity 18.8%, specificity 68.0%, PPV 0.3% [13]. In pediatric athletes (7–16 years), modern "International" ECG criteria achieved very high specificity (98%) but lower sensitivity (57%) compared with earlier ESC and Seattle criteria (specificity 64–95%, sensitivity 86%) [43]. Similar low prevalence of significant pathology (≈0.3%) and low abnormal ECG rates (≈2–3.5%) are reported in large pediatric and elite cohorts when contemporary criteria are used [14, 31, 44]. In master athletes (>35 years), the ESC-2005 criteria show the highest sensitivity for high-risk cardiovascular conditions, detecting 1.8% HRCC versus 1.3–1.4% with Seattle/International criteria [45].

3.4.2. Ambulatory ECG Monitoring

Twenty-four–hour ambulatory ECG (Holter) is typically reserved for athletes with symptoms, abnormal resting ECG, or exercise-induced ventricular arrhythmias (VA) [16, 31, 46]. In young athletes evaluated after SARS-CoV-2 infection, ambulatory monitoring revealed low-burden premature beats (<50/24 h) in over half of athletes and no malignant arrhythmias, supporting a high negative predictive value in this context [46]. Population studies of healthy athletes and matched controls show that the prevalence and complexity of VAs on 12-lead 24-h monitoring are similar between athletes and non-athletes and largely benign; age, but not training load, predicts higher ectopic burden [8, 25]. However, in referred cohorts with frequent or complex VA but normal ECG and echocardiography, Holter characteristics (multiple morphologies, non-infundibular patterns, repetitivity) strongly predict pathologic late gadolinium enhancement on cardiac magnetic resonance (CMR), with these markers independently associated with concealed scar [15, 17].

3.4.3.Exercise Testing

Exercise ECG is used both for screening and risk stratification. In large Italian preparticipation programs, adding exercise stress testing (EST) to history, exam, and resting ECG increased diagnostic yield of SCD-risk conditions from 0.28% to 0.49% (≈75% relative increase), but reduced PPV from 6.9% to 5.5% because of additional false positives [16]. Over serial annual evaluations, many at-risk conditions are first detected on repeat stress testing, particularly in athletes ≥12 years [31]. In asymptomatic middle-aged athletes, however, exercise ECG for ischemia has a very low PPV: among 1298 athletes, 4.1% had a positive test, yet 95% of those referred were false positives, with only 2 requiring coronary revascularization [47]. Thus, sensitivity for coronary disease is modest and specificity limited in low-risk older athletes.

In athletes with VA, exercise-induced arrhythmias are a powerful discriminator of underlying myocardial scar. In a multicenter cohort, complex exercise-induced VAs with right bundle-branch block or polymorphic morphology increased the odds of abnormal CMR fivefold, and overall 56% of athletes with exercise-induced VA had CMR abnormalities versus 21% with non–exercise-induced VA [17]. Reproducibility of the same VA pattern on two exercise tests yielded a PPV 83% and negative predictive value 98% for non-ischemic left ventricular scar [18]. In long-term outcome studies of athletes with complex VA, lack of VA suppression and non-sustained VT induction during stress testing are strongly associated with adverse events [28].

Collectively, these data indicate that resting athlete-specific ECG criteria provide high sensitivity and specificity for SCD-related disease in youth and young adults, EST adds incremental sensitivity at the cost of false positives, and ambulatory monitoring and exercise-induced VA patterns substantially improve specificity for concealed myocardial disease in selected high-risk athletes.

4.Discussion

The comparative analysis of arrhythmia incidence in athletes reveals two distinct physiological trajectories, often characterized as the "athletic heart" phenotypes. While both training modalities induce significant cardiac adaptation, the clinical implications for arrhythmic risk differ substantially. Endurance athletes, particularly middle-aged and "master" competitors, exhibit a consistently higher incidence of atrial fibrillation (AF) than non-athletic controls, with a recent meta-analysis suggesting a 2.5-fold higher likelihood of developing the condition [2].

This risk appears to follow a J-shaped dose-response curve where moderate activity remains protective, but high-volume, lifelong training creates a pro-arrhythmic environment [1, 3, 5].

Mechanistically, this endurance-driven risk is fueled by marked structural remodeling, including bi-atrial enlargement and fibrosis, which animal models suggest increases AF susceptibility independent of ventricular disease [1, 5, 33]. Furthermore, the endurance phenotype is defined by a state of parasympathetic predominance. High resting vagal tone leads to sinus bradycardia and frequent atrial ectopy, while post-exercise vagal rebound often serves as the temporal trigger for paroxysmal AF episodes [6, 39]. Beyond the atria, a subgroup of endurance athletes exhibits an "exercise-induced arrhythmogenic phenotype" involving right-ventricular (RV) dysfunction and non-ischaemic myocardial fibrosis, which is strongly associated with a higher prevalence of non-sustained ventricular tachycardia (NSVT) and even sustained VT [9–11].

In contrast, the arrhythmic profile of strength-trained athletes appears significantly more benign. Data from large-scale cohorts indicate that higher muscle strength is not associated with an increased risk of atrial fibrillation or flutter and may even be protective against bradyarrhythmias and certain ventricular arrhythmias [23]. Unlike the eccentric dilation seen in endurance sports, strength athletes predominantly exhibit pressure-load–driven concentric LV hypertrophy with limited atrial expansion [7, 34, 38].

While this concentric remodeling can lead to increased LV wall thickness and higher corrected QT dispersion—indicating a potential substrate for inhomogeneous repolarization—there is no clear evidence that strength training alone increases the incidence of complex ventricular arrhythmias in the absence of underlying structural disease [7, 25, 42]. In both populations, the prevalence of simple ventricular ectopy remains similar to sedentary controls, suggesting that training volume itself does not dictate ventricular risk [8, 24].

The diagnostic approach to these athletes highlights the critical role of substrate over training mode. While athlete-specific ECG criteria provide high specificity for screening young competitors, the detection of "concealed" myocardial disease in high-risk individuals requires more advanced tools [13, 14, 43]. The presence of non-ischaemic myocardial scar on cardiac MRI is the single most powerful predictor of malignant events, increasing the risk of incident ventricular arrhythmias nearly fivefold [10, 15]. Additionally, exercise stress testing serves as a vital discriminator; complex arrhythmias that are induced or fail to suppress during exercise are strongly linked to underlying scar and adverse outcomes [17, 18, 28]. Ultimately, while endurance training pushes the heart toward a dilated, fibrotic, and vagally-modulated state

prone to AF, strength training favors a stable, pressure-adapted morphology that appears to carry a lower overall arrhythmic burden.

5. Conclusions

The data clearly indicates that the "Athlete's Heart" is not a singular physiological entity but a divergent adaptation based on the primary training stimulus. While both endurance and strength training induce significant cardiac remodeling, their arrhythmic consequences are distinct in both mechanism and clinical presentation.

Long-term, high-volume endurance training carries a significant "atrial tax." The combination of atrial dilation, localized fibrosis, and heightened vagal tone creates a robust substrate for Atrial Fibrillation, particularly in middle-aged and master athletes. This risk follows a J-shaped curve, where the benefits of moderate activity are overtaken by the structural "wear and tear" of lifelong extreme volume overload.

In contrast, strength-dominant training—defined by pressure overload—favors concentric hypertrophy with a relatively neutral or even protective effect against supraventricular arrhythmias. While the "thickened" heart of a power athlete presents its own electrophysiological challenges (such as repolarization heterogeneity), it does not appear to trigger the same AF-prone milieu seen in the "stretched" heart of the marathoner.

For both cohorts, the presence of non-ischemic myocardial scar is the ultimate "red flag." Regardless of the sport, if an athlete presents with complex ventricular arrhythmias that persist during exercise, the focus must shift from training volume to structural substrate. Myocardial fibrosis remains the most potent predictor of malignant events, transcending training modality.

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