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Arrhythmias In Endurance Athletes: A Systematic Review of Epidemiology, Clinical Characteristics, and Management Strategies

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LIST OF ABBREVIATION

AED	Automated external defibrillator
AFEQT	Atrial Fibrillation Effect on Quality-of-Life
ARVC	Arrhythmogenic right ventricular cardiomyopathy
AUC	Area under the curve
AVNRT	Atrioventricular nodal reentrant tachycardia
CI	Confidence interval
ECG	Electrocardiogram
ESC	European Society of Cardiology
GPS	Global Position System
HR	Hazard Ratio
HRS	Heart Rhythm Society
ICD	Implantable cardioverter-defibrillators
IQR	Interquartile range
mEHRA	modified European Heart rhythm Association score
MeSH	Medical Subject Headings
MOCO	Motion corrected
ms	Milliseconds
NOS	Newcastle-Ottawa Scale
OR	Odds Ratio
p	P-value
PICO	Population, Intervention, Comparison, Outcome
PRISMA	Preferred Reporting Items for Systematic reviews and Meta-Analyses
PVB	Premature ventricular beats
RACER	Race Associated Cardiac Event Registry
RR	Relative Ratio
s	Seconds
SD-TPS	Standard deviation of time-to-peak strain
UK	United Kingdom
VO_2peak	Peak Oxygen Uptake

SANTRAUKA

Reguliarus fizinis aktyvumas yra viena veiksmingiausių širdies ir kraujagyslių ligų prevencijos strategijų. Tačiau lėtinės, didelio intensyvumo ištvermės treniruotės gali padidinti įvairių širdies aritmijų riziką. Tai buvo įrodyta keliuose neseniai atliktuose tyrimuose.

Tikslas: Šioje sisteminėje literatūros apžvalgoje nustatomi ir įvertinami dabartiniai įrodymai apie širdies aritmijas didelės ištvermės reikalaujančiuose sporto šakų sportininkuose, daugiausia dėmesio skiriant epidemiologijai, klinikiniams požymiams ir gydymo strategijoms.

Metodai: Keturiose duomenų bazėse (PubMed, ScienceDirect, Google Scholar ir Scopus) buvo ieškoma pirminių tyrimų, paskelbtų 2016–2026 m., laikantis PRISMA 2020 metais paskelbtomis gairėmis. Tyrimų kokybė buvo įvertinta naudojant Niukaslio-Otavos skalę. Dėl nustatytų pirminių tyrimų heterogeniškumo buvo atlikta naratyvinė sintezė.

Rezultatai: Iš viso buvo įtraukti 33 tyrimai, kuriuose dalyvavo daugiau nei milijonas dalyvių. Prieširdžių virpėjimas buvo dažniausia širdies aritmija. Paplitimas svyravo nuo 0,3 % tarp jaunų elitinių sportininkų iki daugiau nei 30 % tarp buvusių sportininkų. Tai rodo U formos dozės ir atsako ryšį su kaupiamuoju treniruočių kiekiu. Tiek vyrai, tiek moterys buvo paveikti ir jiems buvo nustatyta žymiai didesnė rizika. Stipriausias skilvelių aritmijų prognozavimo veiksnys buvo neišeminė miokardo fibrozė, kuri sudarė 48 % buvusių sportininkų. Bradiaritmijos taip pat buvo dažnos, tačiau dažniausiai gerybinės. Terapiškai reikšmingos bradiaritmijos atvejais buvusiems sportininkams dažnai reikia širdies stimuliatoriaus anksčiau nei bendrajai populiacijai. Išgyvenamumas po staigaus širdies sustojimo pastaraisiais dešimtmečiais padvigubėjo dėl pagerėjusios skubios pagalbos. Sportininkams, sergantiems supraventrikulinėmis tachikardijomis, buvo būdingi specifiniai elektrofiziologiniai požymiai ir dažnesni pasikartojimai po abliacijos. Kairiojo prieširdžio padidėjimas išliko ir nutraukus treniruotes, o tai rodo negrįžtamą remodeliaciją. Kateterinė abliacija buvo veiksmingos tiek supraventrikulinėms, tiek skilvelinėms aritmijoms, nepaisant didesnio pasikartojimo dažnio. Antikoaguliacinis gydymas prieširdžių virpėjimui buvo taikomas rečiau nei bendrojoje populiacijoje.

Išvada: Didelio ištvermingumo reikalaujančiose sporto šakų sportininkų širdies aritmijos pasižymi plačiu spektru, turinčiu specifinių epidemiologinių ir klinikinių savybių bei gydymo strategijų. Rekomenduojamas bendras sprendimų priėmimo procesas, pritaikytas individualiam sportininkui. Vis dar yra didelių tyrimų spragų, susijusių su sportininkėmis moterimis, genetiniu polinkiu, ilgalaike natūralia treniruočių sukeltos širdies remodeliacijos eiga ir optimaliomis gydymo strategijomis.

SUMMARY

Background: One of the most effective strategies for cardiovascular disease prevention is regular physical activity. But chronic high intensive endurance training may increase the risk of different cardiac arrythmias. This is demonstrated by several recent studies.

Aim: This systematic literature review identifies and evaluates the current evidence on arrhythmias in endurance athletes, focusing on their epidemiology, clinical characteristics, and management strategies.

Methods: Four databases (PubMed, ScienceDirect, Google Scholar, and Scopus) were screened according to the PRISMA 2020 guidelines for primary studies published between 2016 and 2026. The quality of the studies was assessed using the Newcastle-Ottawa Scale. Due to the heterogeneity of the primary studies identified, a narrative synthesis was conducted.

Results: A total of 33 studies were included, involving more than millions of participants. Across all studies, atrial fibrillation was the most common arrhythmia. The prevalence was ranging from 0.3% in young elite athletes to over 30% in former athletes. This suggests a U-shaped dose-response relationship with cumulative training volume. Both men and women were affected and have a significantly increased risk. For ventricular arrhythmias, the strongest predictor was non-ischemic myocardial fibrosis, accounting for 48% in former athletes. Bradyarrhythmias were also common, though they are typically benign. In therapeutic relevant bradyarrhythmia, former athletes often require a pacemaker earlier than the general population. The survival rate from sudden cardiac arrest has doubled in recent decades due to improved emergency response. Athletes with supraventricular tachycardias exhibited specific electrophysiological characteristics and more frequent recurrences following ablation. Left atrial enlargement persisted after cessation of training, suggesting irreversible remodeling. Catheter ablations were effective for both supraventricular and ventricular arrhythmias despite higher recurrence rates. Anticoagulation therapy for atrial fibrillation was used less frequently than in the general population.

Conclusion: Arrhythmias in endurance athletes encompass a broad spectrum with specific epidemiological and clinical characteristics, as well as management strategies. A shared decision-making process adapted to the individual athlete is recommended. Critical gaps in the research remain regarding female athletes, genetic predisposition, the long-term natural course of training-induced cardiac remodeling, and optimal treatment strategies.

Keywords

Atrial fibrillation, Ventricular arrhythmia, Myocardial fibrosis, Cardiac remodeling, Athletes heart, Sport cardiology

1. INTRODUCTION

Regular physical activity is one of the most well-established protective factors against cardiovascular disease (1,2). The World Health Organization guidelines (1) and the European Society of Cardiology (ESC) (2) recommend at least 150-300 minutes of moderate-intensity or 75-150 minutes of vigorous-intensity aerobic exercise per week for the general adult population. By following these recommendations, the risk of cardiovascular diseases such as coronary artery disease, heart failure, stroke, and overall cardiovascular mortality is reduced (1,2). Endurance athletes, such as marathon runners, cyclists, triathletes, cross-country skiers, rowers, and competitive swimmers, typically exceed the recommended training times. These athletes train intensively several hours per week over decades of their career. Endurance events including marathons, ultramarathons, long-distance cycling events, and triathlons are widespread, and in the United States, more than 29 million athletes completed a half marathon or marathon between 2010 and 2023 (3). While the cardiovascular benefits of regular exercise are well documented, recent studies show that chronic high-intensity endurance training may paradoxically increase the risk of certain cardiac arrhythmias. This leads to the idea of a potential "exercise paradox" in cardiovascular health (4).

The concept of the "athlete's heart" was first described in the late nineteenth century and became a central topic in sports cardiology. It describes the physiological cardiac adaptations like biventricular cavity dilatation, increased left ventricular wall thickness, enhanced diastolic filling, increased stroke volume, and resting sinus bradycardia. These occur in response to sustained hemodynamic stress caused by regular endurance training (5). The type of sport affects the pattern of remodeling. Strength-based sports predominantly cause pressure overload leading to concentric hypertrophy. In contrast endurance sports predominantly cause volume overload leading to eccentric hypertrophy with chamber dilatation (6). Differentiate between beneficial and harmful cardiovascular responses to exercise remains a major diagnostic challenge. It challenge arises from the phenotypic similarity between the electrophysiological and anatomical changes that occur during physiological exercise-induced cardiac remodeling and those observed in hereditary or acquired cardiomyopathies. This overlap matters most where atrial dimensions exceed expected physiological ranges, or where cardiac magnetic resonance shows myocardial fibrosis. These findings represent either extreme physiological adaptation or early pathological remodeling, which require further investigation (7). Atrial fibrillation is the most frequently investigated arrhythmia in the context of endurance sport. Multiple studies (8–11) have reported an increased prevalence of atrial fibrillation among endurance athletes compared to the general population. Especially male athletes with a long history of training are at the highest risk. The underlying pathophysiology is considered to be multifactorial. It involves structural and electrical changes in the atria caused by chronic hemodynamic overload, the development of interstitial fibrosis, and changes in the autonomic nervous system (4). There is a dose-dependent

relationship between lifetime training volume and the risk of atrial fibrillation. Current data suggest a U- or J-shaped relationship: while moderate exercise reduces the risk, extremely high levels of physical activity lead to an increased risk. This finding is important for sports medicine practice in order to provide athletes with targeted support in designing their training programs and to inform them individually about their risk (2,11).

Ventricular arrhythmias and myocardial fibrosis in endurance athletes have appears as potential contributors to adverse cardiac events. Myocardial fibrosis has been identified as a substrate for ventricular tachyarrhythmias in this population. Non-invasive visualization of this fibrosis is possible using cardiac magnetic resonance imaging with late gadolinium contrast (7). Cases of sudden cardiac arrest during endurance competitions show the clinical significance. The absolute risk for such an event remains low and the cardiovascular benefits of regular physical activity are well documented overall (2). Other potential serious consequences of chronic endurance training include bradyarrhythmias, supraventricular tachycardia, and a newly described form of exercise-induced arrhythmogenic cardiomyopathy (4).

The diagnosis and evaluation of cardiac arrhythmias in endurance athletes are complicated by several factors specific to this population. Physiological adaptations of the athlete's heart, including resting bradycardia, increased electrocardiographic voltages, early repolarization patterns, and enlarged cardiac chambers, can mimic findings associated with pathological conditions. This leads to diagnostic uncertainty (2). International guidelines have been developed for the interpretation of electrocardiograms in athletes. These distinguish between normal training-related changes and abnormal findings that require further evaluation (12,13). Because arrhythmias in athletes are often sporadic and paroxysmal, standard resting or short-term recordings can miss clinically significant episodes. For this reason, advanced monitoring strategies such as long-term electrocardiogram recording or implantable loop recorders may be necessary (7).

The clinical management of arrhythmias in endurance athletes departs in important way from the approach used in the general population. Standard treatment algorithms are not directly applicable to individuals with the distinct cardiac physiology of the endurance trained athlete. The decision to restrict or modify athletic activity has implications that reach beyond the medical. These are psychological and social as well as physical (14). Management strategies described in the current literature include a wide spectrum: Conservative approaches are exercise modification and training volume reduction, pharmacological therapy as antiarrhythmic drugs and anticoagulation and interventional strategies contain catheter ablation and implantable device therapy (2). The ESC guidelines on sports cardiology (2) underline the importance of a shared decision-making process between the athlete, sports cardiologist, and electrophysiologist. They balance the potential benefits

of continued athletic participation against the risks of arrhythmia recurrence and adverse outcomes (14).

Over the past decades a growing interest in exercise-associated arrhythmias has grown, but several important knowledge gaps persist (15). Most of available studies focus on atrial fibrillation in middle-aged to older male athletes. Only limited data is published on other arrhythmias (ventricular arrhythmias, bradyarrhythmias, and supraventricular tachycardias) (16,17). Female endurance athletes remain markedly understudied in this field (5). Whether genetic predisposition modifies an athlete's arrhythmia risk is a question only recently taken up by research (9). The significant heterogeneity of study designs, populations, diagnostic methods, and outcome definitions across the available literature makes it difficult to take a conclusions regarding the true prevalence, clinical significance, and optimal management of arrhythmias in this population (2). While several narrative reviews have addressed individual aspects of this topic, no systematic synthesis of the recent primary evidence covering the full spectrum of arrhythmias in endurance athletes, including their epidemiology, clinical characteristics, and management strategies (4,5).

1.1 Aim

The aim of this systematic literature review is to identify, appraise, and synthesize the current evidence on arrhythmias in endurance athletes, focusing on their epidemiology, clinical characteristics, and management strategies.

1.2 Objectives

I. To evaluate the prevalence and incidence of different types of arrhythmias among endurance athletes in studies published between 2016 and 2026.

II. To characterize the clinical presentation and diagnostic features of arrhythmias in endurance athletes, including the role of cardiac remodeling and myocardial fibrosis.

III. To identify and compare the management strategies (conservative, pharmacological, and interventional) described in the included studies.

IV. To assess the quality of the available evidence and identify gaps for future research.

2. METHODS

2.1 Study Design

This systematic literature review was conducted in accordance with the PRISMA 2020 guidelines (18). A review protocol was defined before the commencement of the literature search. The research question was structured using the PICO (Population, Intervention, Comparison, Outcome) framework (19) to ensure a systematic and reproducible approach.

2.2 Research Question and PICO Framework

The research question of this review is: What is the current evidence regarding the epidemiology, clinical characteristics, and management strategies of arrhythmias in endurance athletes?

The PICO framework (19) was applied as follows:

Population (P): Endurance athletes: individuals, who regularly participate in prolonged aerobic exercise. Examples: running (including marathon and ultramarathon), cycling, triathlon, cross-country skiing, rowing, and competitive swimming.

Intervention/Exposure (I): Chronic endurance training. This included competitive and recreational participation at moderate to high intensity over extended periods.

Comparison (C): Sedentary individuals or the general population, if available in the included studies. Not all studies had a comparison group.

Outcomes (O): Epidemiological data as prevalence, incidence, and risk factors of arrhythmias. Clinical characteristics as type of arrhythmia, symptoms, diagnostic findings, cardiac remodeling. Management strategies as conservative approaches, pharmacological treatment, catheter ablation, device therapy, and exercise modification.

2.3 Eligibility Criteria

2.3.1 Inclusion Criteria

Studies were eligible for inclusion if they met all of the following criteria: 1) primary studies with original data, including prospective and retrospective cohort studies, cross-sectional studies, case-control studies, randomized controlled trials, and case series with three or more patients; 2) published between January 2016 and 17 January 2026; 3) investigating cardiac arrhythmias in endurance athletes or populations engaged in regular endurance exercise; 4) reporting on at least one of the following outcomes: epidemiology (prevalence, incidence, or risk factors), clinical characteristics (arrhythmia type, symptoms, diagnostic findings), or management strategies; 5) published in English; and 6) full-text available.

2.3.2 Exclusion Criteria

Studies were excluded if they met any of the following criteria: 1) review articles, meta-analyses, editorials, commentaries, letters to the editor, or expert consensus statements without original data; 2) case reports with fewer than three patients; 3) studies exclusively investigating non-endurance athletes (e.g., strength-only or skill-based sports) without an endurance athlete subgroup; 4) animal or in vitro studies; 5) study protocols without published results; 6) studies with no arrhythmia-related outcome; 7) studies with populations that did not meet the PICO criteria (e.g., general population without differentiation of endurance exercise); 8) duplicate publications from the same cohort

reporting the same outcomes (in cases of overlapping cohorts with different outcomes, both studies were included and the overlap was transparently documented); and 9) studies published before 2016.

2.4 Information Sources and Search Strategy

A systematic literature search was conducted across four electronic databases: PubMed, ScienceDirect (Elsevier), Google Scholar, and Scopus. The search was performed on 17–18 January 2026. All records were subsequently screened by title and abstract, and eligible full-text articles were assessed and evaluated by the end of January 2026.

2.4.1 Search Terms

The search strategy was adapted to the specific syntax and capabilities of each database (APPENDIX A). The part of the search was conducted in PubMed using a combination of Medical Subject Headings (MeSH) terms and free-text keywords. The PubMed search string was as follows:

((("Endurance Training"[Mesh] OR "Athletes"[Mesh] OR "Sports"[Mesh] OR "endurance athlete*" OR "marathon runner*" OR cyclist* OR triathlete* OR rower*) AND ("Arrhythmias, Cardiac"[Mesh] OR "atrial fibrillation"[Mesh] OR "ventricular arrhythmia*" OR "bradycardia" OR "tachyarrhythmia") AND ("Epidemiology"[Mesh] OR "prevalence" OR "clinical characteristic*" OR "management" OR "treatment" OR "diagnosis"))

Additional PubMed searches were conducted using broader MeSH-based and Title/Abstract-based strategies to maximize sensitivity. For ScienceDirect, a simplified search string was used: "endurance athletes AND arrhythmia". For Google Scholar, the search was conducted using the allintitle function: "arrhythmia OR atrial fibrillation OR ventricular arrhythmia AND endurance athletes OR runner OR marathon OR cyclist". For Scopus, the following advanced search string was applied:

TITLE-ABS-KEY(("endurance athlete*" OR "marathon runner*" OR cyclist* OR triathlete* OR rower* OR "cross-country skier*" OR "long-distance runner*") AND (arrhythmia* OR "atrial fibrillation" OR "ventricular arrhythmia*" OR "supraventricular tachycardia" OR bradycardia* OR "premature ventricular")) AND PUBYEAR > 2015 AND PUBYEAR < 2027.

2.4.2 Filters Applied

All database searches were limited to publications from 2016 to 2026 and to articles with full-text availability. Only studies published between 2016 and 2026 were included in order to capture the most recent decade of evidence and to ensure that the review reflects the current state of research. German and English language filter were applied during the initial search, though only English-language articles were ultimately included due to availability constraints. The search was restricted to articles published in peer-reviewed journals.

2.5 Study Selection Process

The study selection process followed a standardized multi-step approach. First, all records from the four databases were compiled. Duplicate records were identified and removed. Second, the titles and abstracts of all remaining records were screened against the predefined eligibility criteria. Studies that clearly did not meet the inclusion criteria based on their title and abstract were excluded at this stage. Third, full-text versions of the remaining studies were obtained. Studies that could not be accessed in full text despite reasonable efforts were excluded and documented. Fourth, the final selection of included studies was made based on a thorough review of the full-text articles against all inclusion and exclusion criteria.

The reasons for exclusion at the full-text stage were systematically documented and categorized as follows: Inappropriate study design, Lack of relevant arrhythmia endpoints, overlapping cohorts, population not meeting PICO criteria, methodological/technical feasibility focus and others. The complete study selection process is presented in the PRISMA flow diagram (18) (APPENDIX B).

2.6 Data Extraction

Data were extracted from each included study using a standardized data extraction form. The following information was collected: first author and year of publication, journal, study design, sample size, participant characteristics (age, sex, sport type), type of arrhythmia investigated, key findings, management strategies described, and database. The extracted data were organized in a structured table and are presented in APPENDIX C.

2.7 Quality Assessment

The methodological quality of the included studies was assessed using the Newcastle-Ottawa Scale (NOS) (20). For cohort and case-control studies, the original NOS was applied. This evaluates three domains: Selection (representativeness of the cohort, selection of controls, ascertainment of exposure, and demonstration that the outcome was not present at baseline; maximum 4 stars), Comparability (control for confounders through design or analysis; maximum 2 stars), and Outcome (assessment method, follow-up duration, and follow-up adequacy; maximum 3 stars), yielding a maximum score of 9 stars. For cross-sectional studies, an adapted version of the NOS was used as described by Herzog et al. (2013) (21), which evaluates Selection (representativeness, sample size, non-response rate; maximum 3 stars), Comparability (maximum 2 stars), and Outcome (assessment method, statistical test; maximum 2 stars), yielding a maximum score of 7 stars. The single randomized controlled trial (McNamara et al. 2019) was assessed separately using the Jadad scale (22).

To ensure consistent and transparent scoring, a priori definitions were established for each NOS criterion before the assessment was conducted. For the Comparability domain, age was defined a

priori as the most important confounder. It is independently associated with both cumulative training volume and arrhythmia incidence. A first star was awarded if the study controlled for age through matching, stratification, or multivariable adjustment. A second star was awarded if the study additionally controlled for at least one further confounder. These were hypertension, body mass index, sex, or cardiovascular comorbidities. For sample size in cross-sectional studies, a star was awarded if n was 100 or greater for the primary analysis, or if a power calculation was performed. For follow-up adequacy in cohort studies, a star was awarded if the dropout rate was below 20 percent. Studies were classified as good quality (NOS 7-9 for cohort and case-control studies, or modified NOS 5-7 for cross-sectional studies), fair quality (NOS 4-6, or modified NOS 3-4), or poor quality (NOS below 4, or modified NOS below 3). Of the 33 included studies, 19 (57.6%) were rated as good quality, 13 (39.4%) as fair quality, and one (3.0%) was assessed separately as an RCT. No study was rated as poor quality. The quality of individual studies was considered when interpreting and synthesizing the results. The complete quality assessment scores are presented in APPENDIX D.

2.8 Data Synthesis

Due to the significant heterogeneity in study designs, populations, arrhythmia types, and outcome measures across the included studies, a quantitative meta-analysis was not feasible. Instead, a narrative synthesis approach was adopted. The results were organized thematically according to the three main domains of the research question: 1) epidemiology of arrhythmias in endurance athletes, 2) clinical characteristics and diagnostic findings, and 3) management strategies. Within each domain, findings were further grouped by arrhythmia type, including atrial fibrillation, ventricular arrhythmias, bradyarrhythmias, supraventricular tachycardias, and sudden cardiac arrest.

Where studies reported overlapping cohorts with different research questions or outcomes (e.g., Javed et al. 2025 and 2026, both from the VENTOUX cohort; Sørensen et al. 2022 and 2023, both from the Birkebeiner cohort; Boraita et al. 2018 and 2022, both from a Spanish elite athlete registry), this overlap was documented transparently, and the studies were treated as complementary rather than independent sources of evidence.

2.9 Ethical Considerations

As this study is a systematic review of previously published literature, no ethical approval was required. All data were extracted from publicly available, peer-reviewed publications.

3. RESULTS

3.1 Epidemiology of Arrhythmias in Endurance Athletes

The epidemiological findings from the 33 included studies revealed a broad spectrum of arrhythmias among endurance athletes. Atrial fibrillation was the most frequently investigated condition, followed by ventricular arrhythmias, bradyarrhythmias, supraventricular tachycardias, and sudden cardiac arrest. The reported prevalence rates varied substantially across studies. This depends on the population studied, the age and sex distribution of participants, the type and duration of endurance training, the diagnostic methods employed, and the definition of arrhythmia used. The following sections present the epidemiological findings organized by arrhythmia type.

3.1.1 Atrial Fibrillation

Atrial fibrillation was investigated in 22 of the 33 included studies. All of these consistently concluded that atrial fibrillation is the most prevalent arrhythmia in endurance athletes. The prevalence varied depending on the studied population. It ranges from 0.3% in a large cohort of 6,813 young elite athletes followed over 20 years (23) to 32% in lifelong master endurance athletes aged 40 years and older (8). This variation reflects the influence of age, sex, and cumulative training exposure on atrial fibrillation risk.

Svedberg et al. (24) investigated a large cohort from 736 102 individuals, including more than 208 654 Vasaloppet cross-country skiers, over a median follow-up of 8.8 years. A significant sex difference in the risk of atrial fibrillation was found. Male skiers had slightly higher risk after adjusting for comorbidities (HR 1.11) compared to non-skiers men. Female skiers had a significantly lower risk compared to non-skiing females (HR 0.55). Especially men who had the highest numbers of ski races (HR 1.21) and finished these the fastest (HR 1.24) had the highest risks. Consequently, there appears to be a dose-dependent relationship between exercise intensity and atrial fibrillation risk in men. This study shows that male skier with atrial fibrillation had lower mortality (HR 0.57) and stroke risk (HR 0.73) compared to non-skiers with atrial fibrillation (24).

Another study with skiers, aged 65 years and older, from Johansen et al. (11) found an atrial fibrillation prevalence of 28.5% compared to 17.8% in non-athletes with the same age. The adjusted relative risk was 1.88 (95% CI 1.49–2.37). The authors described a J-shaped dose-response relationship between physical activity levels and atrial fibrillation risk, with the highest risk observed in the most active group. In this study, the scientists also reported that the stroke prevalence among athletes was significantly lower (5.4% vs. 9.7%, RR 0.60, 95% CI: 0.37-0.95). Among athletes with atrial fibrillation, the stroke risk was 2.38-fold higher than in athletes without atrial fibrillation (95 % CI: 1,08 bis 5,24) (11). The third study with skiers from Myrstad et al. (25), a cohort study using self-reported atrial fibrillation data from 2626 Birkebeiner skiers, reported an atrial fibrillation

prevalence of 12.3%. Atrial fibrillation was strongly associated with older age, more cardiovascular risk factors, and longer history of endurance sport practice. Specifically in this study, both self-reported atrial fibrillation (adjusted OR 2.16, 95% CI 1.07–4.38) and adjudicated atrial fibrillation (adjusted OR 2.38, 95% CI 1.05–5.40) were significantly associated with increased stroke prevalence. There were no association in athletes younger than 65 years (25).

Schreiner et al. (10), a study among competitive swimmers over 60 years old, found a prevalence of atrial fibrillation of 26.5% compared to 7% in controls of the same age. With the mean swimming duration of 25.6 years and a weekly distance of 6.0 miles, the athletes were loaded. In the logistic regression analysis where age and diabetes were in the final model, swimming was the only significant predictor of atrial fibrillation (OR 8.74, 95% CI 2.29–33.34). The athletes had less hypertension (46.9% vs. 72%) and diabetes (2% vs. 32%) than the control group, but an increased atrial fibrillation risk despite this better overall cardiovascular risk profile (10).

A large online survey of 2819 masters runners, from Matsumura et al. (26), reported an atrial fibrillation prevalence of 2.4% (self-reported). The only statistically relevant sport-related factor was accumulated training years. Athletes with 10 or fewer training years of running were affected by 1.2% with atrial fibrillation and the athletes with more than 30 years were with 6.1% affected (OR 1.455 per decade). In the multivariable analysis, age (OR 1.077 per year) and hypertension (OR 2.116) were also independent predictors (26).

A prospective case-control study by Calvo et al. (27) of 115 patients with lone atrial fibrillation and 57 matched controls, characterized the dose-response relationship between exercise volume and atrial fibrillation. It identified a U-shaped relationship. Cumulative intense training exceeding 2000 lifetime hours was associated with a significantly increased risk of lone atrial fibrillation (OR 3.88, 95% CI 1.55–9.73). Training volumes below this threshold were cardioprotective (OR 0.38, 95% CI 0.12–0.98). Other risk factors described were obstructive sleep apnea and increased waist circumference. Sleep apnea was present in 22% of lone atrial fibrillation cases and carried a five-fold increased risk (OR 5.01). Increased waist circumference had an OR of 1.06 per cm. Endurance sport was associated with a higher atrial fibrillation risk than team sport participation (OR 5.68). At least one emerging risk factor was identifiable in 76% of patients with lone atrial fibrillation. This raised doubts that these cases are truly “lone” atrial fibrillation (27).

Elliott et al. (28), in a prospective analysis of 402406 UK Biobank participants with the age between 40-69 years, found that physical activity generally reduced the risk of atrial fibrillation. The protective effect was stronger in women (HR 0.80 at 5000 MET-min/week) than in men (HR 0.90 at 1500 MET-min/week). Very virgious physical activity in men was associated with a 12% increased risk of atrial fibrillation (HR 1.12) (28). This is consistent with the U-shaped relationship described by Calvo et

al. (27). According to Elliot et al. physical activity reduced the risk of ventricular arrhythmias by 11–22% in both sexes (28).

Boraita et al. (23) investigated the atrial fibrillation incidence in young elite athletes (endurance athletes and mixed-exercise athletes). They reported that only 21 of 6813 Spanish national-level athletes (0.3%) developed atrial fibrillation over a 20-year follow-up period. Only one case was a female athlete. The age (OR 1.07 per year), the number of competition years (OR 1.14 per year), and the left atrial anteroposterior diameter (OR 1.21 per mm) were independently associated with atrial fibrillation risk. A left atrial diameter of 41 mm or greater turned out to be a potential risk threshold. Endurance athletes were significantly overrepresented in the atrial fibrillation group (52.4%) compared to the overall cohort (25.4%). This underlines the association between endurance sports and atrial fibrillation. 18 out of 21 cases of atrial fibrillation were paroxysmal, one was persistent and two were long-persistent (23).

The first significant evidence of an increased atrial fibrillation risk in female endurance athletes was provided by Drca et al. (29). These authors conducted a retrospective matched cohort study of 228 female marathon runners, cyclists, and 10,000 m track runners. They were compared to 1368 matched controls from the Swedish population register. Atrial fibrillation developed in 4.4% of female athletes compared to 1.7% of controls in a mean follow-up of 28.8 years. This corresponds to a hazard ratio of 3.67 (95% CI 1.71–7.87) after adjustment for hypertension (29). This finding challenged the earlier connection that exercise-related atrial fibrillation mainly affects male athletes. It points out the importance of conducting sex-specific research (29).

In Apelland et al. (16), 43 non-elite endurance athletes (40 men and three women) with paroxysmal atrial fibrillation using implantable cardiac monitors over a mean monitoring period of 50 days were examined. Only 0.18% (IQR 0–2.6%) was the median atrial fibrillation burden. But 67% of athletes experienced at least one episode during monitoring. 30% of athletes perceived a reduced quality of life, which was assessed by the AFEQT score (total score <80). Episodes lasting longer than 60 minutes were significantly associated with lower quality of life scores (mean AFEQT 78 vs. 90, p=0.001). The majority of athletes (93%) were classified as mild to moderately symptomatic (mEHRA 2a/2b). 74% of these continued to participate in competitive sport (16). D'Ambrosio et al. (8), a cross-sectional analysis, found no significant difference in atrial fibrillation prevalence between active (32%) and retired (34%) lifelong endurance athletes. This suggests that the arrhythmogenic substrate persists after cessation of training. About two-thirds of athletes with non-sustained ventricular tachycardia also had atrial arrhythmias, mainly atrial fibrillation. This points to a global proarrhythmic predisposition rather than just atrial vulnerability (8).

Flannery et al. (9), explored the role of genetic factors in exercise-associated atrial fibrillation. The study examined 121 elite rowers and 11,495 controls and demonstrated a 6.8-fold higher prevalence

of atrial fibrillation in rowers. According to this study a polygenic risk score makes a contribution to the increased risk of atrial fibrillation. This is the first evidence for a gene-exercise interaction in the development of sport-associated atrial fibrillation. The authors showed with this study that the individual genetic susceptibility may partly explain why some athletes develop atrial fibrillation while others with similar training exposure do not. This could lead to future changes in personal risk assessment (9).

3.1.2 Ventricular Arrhythmias

Ventricular arrhythmias were considered in 10 of the 33 included studies. These involved premature ventricular complexes, non-sustained ventricular tachycardia, sustained ventricular tachycardia, and sudden cardiac death. Across multiple studies myocardial fibrosis turned out to be the strongest and most consistent predictor of ventricular arrhythmias in endurance athletes.

The VENTOUX study by Javed et al. (30) monitored 106 veteran male cyclists and triathletes, with age over 50 years, using implantable loop recorders over a median follow-up of 720 days. Cardiac magnetic resonance imaging with late gadolinium enhancement detected myocardial fibrosis in 47.2% of athletes. The fibrosis was predominantly (88%) located in a non-ischemic pattern involving the basal inferolateral left ventricular segment. During the monitoring period, 21.7% of athletes developed ventricular arrhythmias. 20 of these cases were non-sustained ventricular tachycardia and 3 cases were sustained ventricular tachycardia. Myocardial fibrosis was the strongest predictor for ventricular arrhythmia occurrence (HR 4.7, 95% CI 1.8–12.8, p=0.002). It was followed by indexed left ventricular end-diastolic volume. A common finding in these endurance athletes was late gadolinium enhancement at the right ventricular insertion point. But this was not associated with ventricular arrhythmias and only a benign hemodynamic adaptation rather than a pathological substrate (30).

In a complementary analysis of the same VENTOUX cohort, Javed et al. (15) examined the temporal relationship between exercise and ventricular arrhythmias. The rate of exercise-associated ventricular arrhythmias was 0.4 per 1,000 hours of exercise compared to 0.014 per 1,000 hours at rest. This results in a relative risk of 28.6. All episodes of sustained ventricular tachycardia occurred during exercise. The athletes had both myocardial fibrosis and prior episodes of non-sustained ventricular tachycardia. There were no significant differences in training volume, intensity, or modality between athletes who developed ventricular arrhythmias and those who did not. This suggests that the arrhythmia risk is determined primarily by the underlying substrate and acute exercise acting as a trigger (15).

Similar findings were reported by Farooq et al. (31) in a cross-sectional study of 50 veteran cyclist and triathletes. These athletes had a median age of 56 years and the control group constated of 26

sedentary individuals. Myocardial fibrosis was present in 48% of athletes compared to 15% of controls (p=0.005). All fibrotic lesions were located in a characteristic mid-myocardial pattern in the basal lateral wall of the left ventricle. Athletes with fibrosis had a significantly higher burden of premature ventricular beats (p=0.03) and ventricular couplets and triplets (p=0.02). The T2-weighted cardiac magnetic resonance imaging demonstrated elevated signal intensity in fibrotic segments. It indicates an ongoing myocardial inflammation or edema even in the absence of acute symptoms. The resting myocardial blood flow was reduced in the fibrotic areas, which points to a potential microvascular dysfunction (31).

Claessen et al. (32) investigated the clinical and genetic overlap between exercise-induced cardiac remodeling and dilated cardiomyopathy in 281 young elite endurance athletes (mean age 22 years, 79.7% male). A reduced ejection fraction was found in 15.7% of athletes. It was defined as left ventricular ejection fraction below 50% and/or right ventricular ejection fraction below 45%. These athletes had significantly more premature ventricular beats on 24-hour Holter monitoring (13.6% vs. 3.8% with >100 PVBs/24h, p=0.008) and lower left ventricular global longitudinal strain (-17% vs. -19%, p<0.001). No significant difference in the prevalence of non-sustained ventricular tachycardia between athletes with reduced and preserved ejection fraction (0% vs. 1.3%, p=0.45) was discovered. In athletes with reduced ejection fraction, a polygenic risk score for left ventricular end-systolic volume was significantly higher (0.57±0.13 vs. 0.51±0.14, p=0.009). It is linked to with genetic susceptibility to dilated cardiomyopathy. The athletes in the highest ten percent of this score had an 11-fold increased risk of reduced ejection fraction. Male sex and higher polygenic risk score were the only significant predictors in the multivariable analysis. One sudden cardiac death occurred in the median follow-up time of 4.4 years. Both, reduced right ventricular ejection fraction and a polygenic risk score above the 95th percentile were present in this case (32).

The retrospective study of Venlet et al. (33) described a novel entity of isolated subepicardial right ventricular outflow tract scar. 57 patients with sustained ventricular tachycardia were examined. 11 of them were cyclists and runners with a mean age of 42 years. Neither Task Force criteria for arrhythmogenic right ventricular cardiomyopathy were met, nor genetic testing revealed pathogenic mutations. The ventricular tachycardia in athletes with this isolated scar pattern had a significantly shorter cycle length (257 ms) compared to patients with subtricuspid scars (predominantly ARVC) (328 ms). This indicates a smaller reentrant circuit within the confined scar area. Electroanatomic voltage mapping demonstrated that the scar was exclusively subepicardial and confined to the right ventricular outflow tract region (33).

Cavigli et al. (34) monitored 68 master athletes during a 50-km ultramarathon with single lead Holter recorders. 69% of the athletes were men and the mean age was 47.9 years. The recorders registered in 7% isolated premature ventricular beats during the race and no episodes of non-sustained or

sustained ventricular tachycardia. Echocardiography examinations showed post-race no changes. Examined were left and right ventricular systolic function, speckle-tracking analysis of global longitudinal strain and right ventricular free wall strain. Prominent were the significantly prolonged QTc interval post-race (406±18 ms to 423±20 ms, $p<0.001$) and the increased R-wave amplitude in V1. The clinical significance remains uncertain (34).

Boraita et al. (35) performed 24-hour Holter monitoring in 654 elite athletes, who were selected from a larger cohort of 6579 athletes. The median age was 24 years and 72% were men. The scientists found premature ventricular complexes in 39.4% of athletes. In most of these cases less than 100 extrasystoles per 24 hours occurred. Non-sustained ventricular tachycardia was recorded in only 1.5% and atrial fibrillation in less than 1%. In echocardiography no significant association between ventricular arrhythmias and structural cardiac disease could be identified. In some cases mild mitral regurgitation was present and was associated with higher arrhythmia burden. Overall, these results point to a benign nature of ventricular arrhythmias in young elite athletes (35).

Only one case of ventricular tachycardia was identified in Vanova et al. (36), who screened 280 athletes. The individual was successfully treated with radiofrequency catheter ablation (see section 3.1.4) (36).

Herm et al. (37) examined 107 marathon runners during their races with a continuous ECG monitoring. The median age was 48 years and 76% were male sex. Non-sustained ventricular tachycardia was detected in 9.4% and atrial fibrillation in 0.9%. Increasing age was the only independent predictor of abnormal ECG findings (OR 1.11 per year, 95% CI 1.01–1.23). In 16.8% of runners the high-sensitivity troponin T was elevated post-race. Association was given with ST-T segment deviation (OR 9.9, 95% CI 1.9–51.5). Cardiac magnetic resonance imaging was performed in ten athletes with abnormal findings. It revealed no structural pathology or late gadolinium enhancement. No cardiovascular events occurred during one-year follow-up (37).

New insights into the temporal dynamics of exercise-associated arrhythmias were provided by Wundersitz et al. (38). The authors conducted a 9-day continuous Holter monitoring protocol in 34 recreational cyclists. 70.6% were male and the mean age was 52 years. Abnormal arrhythmias were found in 68% of participants on at least one monitoring day. Most frequently the arrhythmias occurred on the training day compared to both the pre-training baseline days and the first post-training recovery day ($p<0.05$). The relative risk of abnormal arrhythmias on the training day increased proportionally with the number of pre-training days on which arrhythmias were already present. It was increased up to five-fold (RR 5.0, $p<0.001$) in athletes with arrhythmias on three or more of the preceding days. Throughout the monitoring period, serum concentrations of calcium, potassium, magnesium, and sodium remained stable. This shows that a structural or autonomic substrate would be more likely to

trigger the arrhythmias. In the study a multi-day pre-exercise arrhythmia screening for older recreational athletes before high-volume training sessions is recommended (38).

3.1.3 Bradyarrhythmias

Bradyarrhythmias were reported in five of the included studies and represented the most common electrocardiographic finding in endurance athletes.

Boraita et al. (35) performed 24-hour Holter monitoring in 654 elite athletes and found sinus bradycardia in 96% of the study population. Extreme sinus bradycardia, below 30 beats per minute, was rare. From these, only in 0.9% of athletes occurred during daytime and in 8.6% during nighttime. There were no clinically significant pauses recorded, which was defined as >3 seconds. No symptoms were indicated during these extreme bradycardia episodes. In a small number of athletes, a first-degree atrioventricular block appeared. Under 1% of athletes had a high-degree atrioventricular block (35).

Kaleta et al. (39) evaluated 40 male amateur marathon runners before and after a marathon race using serial 12-lead electrocardiography. The mean age was 39 years. In 92.5% of these athletes there was at least one abnormality in resting electrocardiography. Sinus bradycardia was the most common finding (62.5%). 30% of athletes had voltage criteria for left ventricular hypertrophy and 20.4% showed early repolarization. Only in 5% of cases an incomplete right bundle branch block was present. Training-associated electrocardiographic variants were significantly more prevalent at rest (82.5%) than immediately after the marathon (42.5%). This reflects the expected physiological shift from vagal to sympathetic predominance during maximal exercise. Immediately after the race, signs of right atrial enlargement developed in 42.5%. This finding points to a transient right-sided cardiac loading during prolonged endurance exercises. Pathological electrocardiographic variants were identified in 15% of runners and make further investigations necessary. In complete this study shows the importance of interpreting electrocardiographic findings in athletes within the context of their training status and the temporal relationship to exercise (39).

A cross-sectional comparison of 22 competitive triathletes and 7 healthy non-athletic controls by Climstein et al. (40), with the mean age of 20.6 years and 50% women, showed in 68.2% of athletes sinus bradycardia and in zero of non-athletes. The athletes had a significantly lower mean resting heart rate (53.8 vs. 72.1 bpm, $p=0.04$). First-degree atrioventricular block was detected in 13.6% of triathletes. Echocardiographic assessment revealed that peak aerobic capacity (VO_2peak) was strongly correlated with left ventricular mass ($r=0.68$, $p<0.003$). It was not correlated with resting heart rate. This suggests that the training-associated bradycardia is mediated primarily by autonomic mechanisms and not by the degree of structural cardiac adaptation. The authors recommended echocardiography in addition to electrocardiography for assessing left ventricular hypertrophy in

athletes, because voltage-based ECG criteria underestimated the prevalence of structural remodeling in this cohort (18.2% vs. 81.8%) (40).

The long-term clinical significance of training-associated bradyarrhythmias was highlighted by Bondarev et al. (41), who studied 361 patients requiring permanent pacemaker implantation for sinus node dysfunction or atrioventricular conduction disease at a single center. This cohort study included 9 former professional endurance athletes and 9 former professional strength/mixed athletes. Former endurance athletes were diagnosed with clinically significant conduction disorders at a significantly younger mean age (64 vs. 72 years, $p<0.01$). They received pacemaker implantation earlier (mean age 65 vs. 73 years, $p<0.05$) than other athletes or the other patients without a professional sport background. Second- and third-degree atrioventricular block was the predominant indication for pacing in 78% of endurance athletes. In only 44% of former strength and mixed sports athletes these were the indication for pacing. These findings show that long-term endurance training can lead to lasting changes in vagal tone and the cardiac conduction system, especially at the atrioventricular node. Even after training cessation, these changes may persist (41).

Both analyses of Elliott et al. mention bradycardia (28,42). Elliott et al. reported one clinically significant case of nocturnal bradycardia among 99 recreational endurance athletes, involving an 8-second sinus pause that resolved completely with detraining (42). In the same UK Biobank cohort, Elliott et al. (28) found no significant overall association between physical activity levels and the incidence of bradyarrhythmias in either sex. In women, vigorous physical activity was associated with a 9–18% lower risk of bradyarrhythmias. This shows a potential sex-related difference in the cardiac conduction system response to chronic exercise (28).

3.1.4 Supraventricular Tachycardias

Supraventricular tachycardias, specifically atrioventricular nodal reentrant tachycardia (AVNRT), were investigated in three studies.

Miljoen et al. (17), a retrospective analysis of 504 patients, including 85 athletes, investigated the impact of athletic cardiac remodeling on the clinical presentation of atrioventricular nodal re-entrant tachycardia (AVNRT). Individuals were classified as athletes if they trained ≥3 hours per week for ≥5 years. These athletes mainly performed in high dynamic sports like cycling and their mean age was 30.9 years with predominantly male sex (78.8%). They were compared to the mean age of 44.1 years and 21.7% male sex. A reversal of genders can be observed in comparison. There was a clear shift in the distribution of arrhythmia subforms of athletes compared to non-athletes. Non-athletes predominantly exhibited the typical "slow-fast" AVNRT (approximately 80–84%). Almost half of the athletes presented with atypical subforms, such as slow-slow or fast-slow varieties (45–48% vs. 16–20% in non-athletes, $p<0.001$). Multivariate analysis showed that both male gender and sports

participation were independent predictors for the presence of atypical subforms. Age was no significant factor. The authors suspected that these variants resulted from athlete's heart remodeling and the resulting changes in electrophysiological properties of the slow atrioventricular nodal pathways (17).

Vanova et al. (36) performed a cardiovascular screening in 280 athletes with the mean age of 29.4 years and 86.8% were male. The authors identified significant cardiac pathology in 5.3% (n=15). 14 athletes showed arrhythmias. Diagnosed were three athletes with AVNRT, two athletes with ventricular tachycardia, two athletes with premature ventricular complexes, two athletes with second-degree atrioventricular block (Mobitz type II) and five athletes with atrial fibrillation. All five cases of paroxysmal atrial fibrillation occurred exclusively in endurance runners with the mean age of 52 years. Structural heart disease was identified in three subjects. These were hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy and bicuspid aortic valve (36).

142 endurance athletes, predominantly runners and cyclists, were evaluated by Gajda et al. (43). The mean age were 26 years and 73% were male. These athletes showed suspected exercise-induced arrhythmias. In only one case a true AVNRT (0.7%) was detected. 45% of high heart rate alerts turned out to be motion artifacts. 39% showed concordant normal findings. A 15% discordance rate was observed where Holter ECG detected extrasystoles that the heart rate monitors failed to capture (43).

3.1.5 Sudden Cardiac Arrest

Sudden cardiac arrest during endurance events was investigated by Kim et al. (3) using the RACER registry. It registered over 29.3 million marathon and half-marathon finishers in the United States between 2010 and 2023. 176 cardiac arrests were recorded. This results in an overall incidence of 0.60 per 100,000 participants. The incidence rate remained stable throughout the study period. The fatality rate declined from the 2000–2009 period with 71% to 34% in the 2010–2023 period. It results in an overall survival rate of 66%. Coronary artery disease was the most identified underlying cause (40% of confirmed etiologies). The other etiologies were hypertrophic cardiomyopathy and other structural heart conditions. Male participants had a higher incidence of cardiac arrest (1.12 per 100,000) compared to female participants (0.19 per 100,000). Full marathon distance was associated with approximately doubled risk compared to half-marathon distance (1.04 vs. 0.47 per 100,000). The independent predictors of survival were an initial shockable rhythm, as ventricular tachycardia or ventricular fibrillation, and shorter time from collapse to initiation of cardiopulmonary resuscitation. The higher survival rate over time could be concluded to better emergency action planning at race events (3).

3.2 Clinical Characteristics and Diagnostic Findings

The included studies provided detailed insights into the structural, functional, and electrophysiological changes underlying arrhythmias in endurance athletes. In the next sections, findings on atrial remodeling, myocardial fibrosis and ventricular substrate, and the diagnostic methods employed for arrhythmia detection in this population are presented.

3.2.1. Atrial Remodeling

Left atrial enlargement was a consistent finding across multiple studies. It turned out to be a central mechanism underlying the increased susceptibility to atrial fibrillation in endurance athletes. McNamara et al. (44) provided a strong evidence for a relationship between endurance training and atrial remodelling. They showed it through a randomized controlled trial with 53 previously sedentary middle-aged adults (mean age 52 years). A part of these participants underwent a 24 months of progressive endurance exercise training and the other part were the control group. Left atrial volumes increased by 55% in the exercise group. The left ventricular end-diastolic volume increased by only 17% and reached a plateau after approximately 10 months. The left atrium to left ventricle volume ratio increased by 32% in the exercise group. These volume ratio levels were observed in 14 masters endurance athletes included as a cross-sectional comparison cohort. This disproportionate atrial dilatation supports the “dam-wall” hypothesis. This proposes that the thin-walled atrium is more vulnerable than the muscular ventricle to the chronic pressure and volume loading by endurance training. While left ventricular volumes plateaued after the initial 10 months of peak training, the left atrium continued to significantly dilate throughout the subsequent 14-month maintenance phase. Left atrial ejection fraction remained stable, showing a physiological adaptation and no pathological. No electrical remodeling was detected, and no episodes of atrial fibrillation were identified in a two-week telemetry monitoring. This means that structural remodeling starts first and after a longer endurance exercise exposure, electrical remodeling follows (44).

Complementary data were found by the Birkebeiner cross-country skiing cohort. Sørensen et al. (45) studied 302 male participants (mean age 70.8) with speckle-tracking echocardiography. They compared athletes with and without paroxysmal atrial fibrillation to non-athletes with and without atrial fibrillation. Left atrial reservoir strain was more effective than traditional volume measurements in identifying participants with paroxysmal atrial fibrillation. By achieving an area under the receiver operating characteristic curve of 0.740. Left atrial reservoir strain was significantly reduced in all participants with atrial fibrillation, with no difference between athletes and non-athletes. There was an overlap in left atrial volumes between non-athletes with atrial fibrillation and athletes without atrial fibrillation. This shows the limited discriminatory value of volume-based assessment alone (45).

In another analysis from the same cohort, Sørensen et al. (46) evaluated left atrial mechanical dispersion (SD-TPS) as a marker of atrial dyssynchrony. The authors found that it was significantly associated with paroxysmal atrial fibrillation ($p<0.001$) but not with athlete status itself ($p=0.173$). By including both parameters in one model, left atrial mechanical dispersion did not provide significant incremental diagnostic value over left atrial reservoir strain ($p=0.056$). Reservoir strain remains the most useful single echocardiographic parameter for identifying veteran athletes with paroxysmal atrial fibrillation (46).

Several studies examined the persistence and reversibility of atrial remodeling after training cessation. Aaroee et al. (47) followed 50 former elite endurance athletes approximately 7 years after the end of their competitive careers. The mean baseline age was 23 years and 70% were male athletes. For comparison, maximal left atrial volume index was measured. After training cessation, weekly training volume was reduced by approximately 60% and peak aerobic capacity declined by 20%. Left atrial dilatation persisted at levels significantly above normal reference values. Left ventricular volumes and wall thickness normalized to levels comparable to the general population. E/e' ratio increased, which suggested a worsening diastolic function with aging. The researchers observed a significant increase in left atrial contraction strain and the left atrial stiffness index. This leads to hypothesis of compensating the age-related increase of left ventricular filling pressure through enhanced active emptying. One new diagnosis of atrial fibrillation was made during the follow-up assessment (47).

This observation was confirmed by D'Ambrosio et al. (8). The authors compared active and retired lifelong endurance athletes in a cross-sectional study. They found significantly enlarged left atrial indexed volumes in retired athletes compared to controls (approximately 45 mL/m² vs. approximately 31 mL/m²). Ventricular dimensions had normalized after cessation (8).

Elliott et al. (42) reported a graded relationship between lifetime training hours and left atrial volume. Moderate to severe left atrial dilatation presented in 19.4% of athletes with more than 6000 training hours. 12.9% of those presented with had 3000 to 6000 hours. 0% of athletes presented moderate to severe left atrial dilatation with fewer than 3000 hours ($p=0.006$ after age adjustment) (42).

Significantly larger left atrial anteroposterior diameters in athletes, who developed atrial fibrillation (43±9 mm), were reported by Boraita et al. (23). This was compared to those athletes who did not develop atrial fibrillation (34±5 mm, $p<0.001$). The authors identified a diameter of ≥41 mm as a potential threshold for increased risk (23).

3.2.2 Myocardial Fibrosis and Ventricular Substrate

Myocardial fibrosis detected by late gadolinium enhancement on cardiac magnetic resonance imaging was a prominent finding in studies of veteran endurance athletes. It was consistently associated with ventricular arrhythmias. Javed et al. (30) found late gadolinium enhancement in 47.2% of veteran

cyclists and triathletes. The fibrosis was predominantly located in a non-ischemic pattern in the basal inferolateral segment. Fibrosis was the strongest predictor of ventricular arrhythmias (HR 4.7). Late gadolinium enhancement at the right ventricular insertion point, commonly observed, showed no association with arrhythmia risk. Exercise-induced premature ventricular contractions were an independent predictor of future arrhythmias (HR 3.9), especially when combined with myocardial fibrosis (30). Similar prevalence in veteran endurance athletes (48%) was reported by Farooq et al. (31). This was compared to 15% in sedentary controls (p=0.005). All fibrotic lesions were located in the mid-myocardial basal inferolateral segments. Elevated T2 signal in the fibrotic areas could point to possible ongoing myocardial inflammation and reduced resting myocardial blood flow in these segments (31). D'Ambrosio et al. (8) detected late gadolinium enhancement in 40% of endurance athletes compared with 18% of controls. Non-ischemic left ventricular scars were equally prevalent in active and retired athletes. This suggests that if fibrosis is established, it does not regress with detraining (8). The role of genetic predisposition in ventricular remodeling was already described in section 3.1.2. Claessen et al. (32) showed that a polygenic risk score for left ventricular end-systolic volume was the main predictor of reduced ejection fraction in young elite athletes. Training volume and intensity did not play a significant role. This means that a gene-exercise interaction is likely the main factor determining who develops adverse ventricular remodeling and who does not (32).

3.2.3 Diagnostic Methods and Monitoring

The included studies showed a wide range of diagnostic modalities for arrhythmia detection. They reflect two practical challenges: The intermittent nature of many arrhythmias in endurance athletes and the confounding effects of physiological cardiac adaptation. Implantable loop recorders provided the broadest surveillance.

Javed et al. (15,30) used these devices for continuous single-channel electrocardiographic monitoring over a median of 720 days. The authors synchronized these devices with GPS-based exercise tracking, which allowed direct correlation between arrhythmia episodes and physical activity patterns. The objective tracking shows a significant discrepancy in clinical data collection and athletes' self-reported training volumes (mean 11.9 vs. 7.2 hours per week). It highlights the importance of prospective, device-based quantification of exercise exposure for correct risk assessment (15,30).

Apelland et al. (16) similarly used implantable cardiac monitors in 43 athletes with paroxysmal atrial fibrillation. They documented detailed atrial fibrillation burden data over an average of 50 monitoring days that would not have been captured by standard intermittent monitoring approaches (16).

The value of extended ambulatory monitoring was demonstrated by Wundersitz et al. (38). Their 9-day continuous Holter protocol revealed that the daily incidence of arrhythmias varied in individual athletes. 68% of athletes showed abnormal arrhythmias on at least one day but not consistently across

all days. Single-day Holter assessments would substantially underestimate the true prevalence of arrhythmias in recreational athletes. This may explain the wide variation in arrhythmia prevalence reported across studies using different monitoring durations. Multi-day monitoring protocols have to be used for a more accurate characterization of arrhythmia burden in these symptomatic athletes (38).
Gajda et al. (43) evaluated wearable heart rate monitors critically. They recorded simultaneous recordings with a commercial heart rate monitor and a medical long-term ECG (Holter-ECG) on 142 athletes during physical exertion. 45% of high heart rate alerts generated by the wearable device during exercise were motion artifacts caused by rhythmic body movements. 15% of true arrhythmias detected by the Holter were missed entirely by the heart rate monitor. The authors proposed a structured diagnostic algorithm. All apparent arrhythmias detected by consumer wearable technology should be verified using standard 12-lead or Holter electrocardiography. Afterwards further cardiac evaluation would be initiated, avoiding unnecessary and potentially costly investigations in athletes with false-positive alerts. To reliable distinguish between supraventricular and ventricular arrhythmias, information regarding QRS morphology and atrial signals needs to be present, which is only possible with an electrocardiogram (43).
Advanced echocardiographic techniques showed good potential for non-invasive risk stratification (23,45). For identifying athletes with paroxysmal atrial fibrillation, Sørensen et al. (45) reported that left atrial reservoir strain measured by speckle-tracking echocardiography performed better than conventional left atrial volume measurements (AUC 0.740). It offers a more functional assessment of atrial myocardial health (45).
Boraita et al. (23) identified a left atrial anteroposterior diameter of 41 mm as a potential echocardiographic threshold for increased atrial fibrillation risk in young athletes (23).
The primary diagnostic tool for detecting and quantifying myocardial fibrosis, in the studies by Javed et al., Farooq et al., D'Ambrosio et al., and Claessen et al. (8,30–32), was cardiac magnetic resonance imaging with late gadolinium enhancement. A higher sensitivity in this technique is achieved by applying motion-corrected (MOCO) and dark-blood late gadolinium enhancement sequences. This allows identification of small areas of scar with high diagnostic certainty. T2-weighted imaging for inflammation detection and myocardial perfusion mapping, as demonstrated by Farooq et al. (31), provided additional diagnostic dimensions beyond scar detection. The integration of structural imaging with exercise testing was essential useful for the risk assessment, especially for exercise-induced atypical premature ventricular contractions (30).

3.3 Management Strategies

Management strategies for arrhythmias in endurance athletes were described in the majority of the included studies. Some studies specifically evaluated the long-term outcomes of therapeutic

interventions in prospective designs. The reported approaches ranged from conservative management with exercise modification to pharmacological therapy, catheter ablation, implantable device therapy, and organized emergency response at endurance sporting events.

3.3.1 Conservative Management and Exercise Modification

Exercise modification, as training volume reduction, intensity modification, and temporary or permanent detraining, was the most commonly described conservative approach across the included studies. With that, the hemodynamic stimulus is reduced and cardiac remodeling may slow, stop or be reversed.

Elliott et al. reported the resolution of clinically significant nocturnal sinus pauses with detraining in one recreational athlete (42). D'Ambrosio et al. (8) noted that the atrial fibrillation prevalence did not differ between active and retired endurance athletes (8). Aaroee et al. showed that left atrial dilatation persisted 12 years after career cessation despite a 60% reduction in training volume and a 20% decline in aerobic capacity (47). These findings show that some bradyarrhythmias may reverse with detraining, but the atrial fibrillation substrate, once established, may not be fully reversible.

Apelland et al. (16) reported that none of the 43 athletes with paroxysmal atrial fibrillation in their cohort were receiving regular antiarrhythmic medication. 74% continued to participate in competitive sport despite their diagnosis. This points to a general preference for conservative management with symptom monitoring in this population. However, more than 50% of these athletes were classified as mEHRA Class 2b, a symptom level increasingly recognized as a threshold for considering rhythm-control interventions like catheter ablation. 30% of athletes reported reduced quality of life despite a low median atrial fibrillation burden of only 0.18%. This shows that even infrequent episodes can have a disproportionate impact on highly active individuals (16).

Wundersitz et al. (38) proposed multi-day pre-exercise arrhythmia screening for older recreational athletes before high-volume training sessions. The authors based this recommendation on their finding that arrhythmias on preceding days were the strongest predictor of arrhythmias on training days (38).

3.3.2 Pharmacological Therapy

Pharmacological therapy was reported variably across the included studies and covered antiarrhythmic drugs, rate-control agents, and anticoagulation for stroke prevention. Athletes with atrial fibrillation appeared to use antiarrhythmic medications less frequently than non-athletes.

Sørensen et al. (46) noted that flecainide use was lower in athletes with paroxysmal atrial fibrillation (3.5%) compared with non-athletes with atrial fibrillation (11.5%). The suspected reasons for that are

concerns about the effect and side effects on the exercise capacity and a generally lower symptom burden in athletes (46).

The side effects of rate controlling drugs as lethargy and the direct negative impact on maximal exercise capacity is demonstrated by Apelland et al. (16). The athletes in this cohort preferred the pill-in-the-pocket approach to manage symptomatic episodes (16). Regarding anticoagulation for stroke prevention in athletes with atrial fibrillation, the results from the included studies indicate both appropriate guideline adherence and potential undertreatment.

Myrstad et al. (25) found that only two-thirds of athletes with atrial fibrillation and a CHA_2DS_2-VASc score of 2 or higher were receiving oral anticoagulation. Despite the clear guideline indication for anticoagulation therapy, the reasons against it could be the increased bleeding risk in athletes during contact or fall-prone sports (25).

In cross-country skiers with atrial fibrillation, Svedberg et al. (24) reported higher anticoagulation rates (58%) compared to non-skiers with atrial fibrillation (52.3%). This could reflect a greater health awareness and more frequent medical contact in the athletic population (24).

Vanova et al. (36) reported that five athletes with newly diagnosed paroxysmal atrial fibrillation and a CHA_2DS_2-VASc score of 0 were appropriately managed without anticoagulation according to ESC guideline (36).

3.3.3 Catheter Ablation

Catheter ablation outcomes were reported in three studies. The results were generally positive. In two studies the therapy is additionally mentioned. Miljoen et al. (17) evaluated radiofrequency catheter ablation for AVNRT in 85 athletes and 419 non-athletes. In athletes this procedure was more challenging. It required more radiofrequency energy applications and the use of long deflectable sheaths to achieve adequate catheter contact. The enlarged and remodeled posteroseptal right atrial region made the procedure technically difficult. The initial success was comparable between athletes and non-athletes. During follow-up, athletes had a significantly higher recurrence rate (10% vs. 4%, $p=0.029$). This could be attributed to the ongoing remodeling of the atrioventricular nodal region as a result of continued intensive training. No cases of major complication occurred, which confirms the overall safety (17).

Excellent outcomes of catheter ablation for isolated subepicardial right ventricular outflow tract scars were presented by Venlet et al. (33). The ablation success rate was 91% using an epicardial approach. This is significantly higher than the 57% success rate in patients with genetic arrhythmogenic cardiomyopathy. The higher ablation success rate may be explained by the shorter ventricular tachycardia cycle length in athletes (257 vs. 328 ms). This points to a smaller, more focal reentrant

circuit within the limited epicardial scar. For this specific athlete phenotype, epicardial catheter ablation appears to be a highly effective treatment strategy (33).

Vanova et al. (36) reported successful radiofrequency catheter ablation in three athletes with AVNRT and one with ventricular tachycardia. All four athletes returned to competitive sport within three months without complications or arrhythmia recurrence. This shows that a rapid return to competitive sport is possible after successful ablation (36).

For atrial fibrillation ablation, Apelland et al. (16) reported that 6 of 43 athletes (14%) had undergone prior catheter ablation for atrial fibrillation. Two of them had undergone repeat procedures and other athletes were awaiting ablation at the time of enrollment (16).

In the Birkebeiner cross-country skiing cohort, Sørensen et al. (45,46) reported that approximately 35% of veteran athletes with atrial fibrillation had undergone prior catheter ablation. This indicates that ablation is a commonly accepted treatment option among athletes. The high rate of repetition may also reflect incomplete efficacy of single procedures in athletes with extensively remodeled atria (45,46).

3.3.4 Implantable Device Therapy

Several studies described on implantable device therapy, including permanent pacemakers and implantable cardioverter-defibrillators (ICDs). D'Ambrosio et al. (8) reported the implantation of three pacemakers and three ICDs in their cohort of 266 athletes and controls. During a median follow-up of 5.5 years, no adverse cardiac events occurred in athletes with non-sustained ventricular tachycardia. This is reassuring for the intermediate-term safety of conservative monitoring in this subgroup (8).

In the study by Bondarev et al. (41), all former endurance athletes with clinically significant sinus node dysfunction or atrioventricular conduction disease received dual-chamber pacemakers. These were implanted at a significantly younger age (mean 65.0 years) compared to patients without a professional athletic background (mean 75.2 years). The pacing stimulation burden was low (mean 5.0%), meaning that the underlying conduction disease was mostly intermittent, even though permanent device support was needed (41).

Javed et al. (15) reported one ICD implantation in a veteran cyclist who experienced presyncope during sustained ventricular tachycardia. A second athlete was referred for electrophysiological study. A third athlete experienced sustained ventricular tachycardia during exercise and was advised to stop competitive sport but declined further diagnostic (15).

Vanova et al. (36) reported one ICD implantation in an athlete with arrhythmogenic right ventricular cardiomyopathy, which was found during pre-participation screening and led to career cessation. Another athlete with a bicuspid aortic valve received an aortic valve replacement (36). Looking at

ICD implantations across all studies, they were mainly done for secondary prevention. The athletes had either survived an out-of-hospital cardiac arrest or had symptomatic sustained ventricular tachycardia. In many of these cases, ventricular function was preserved and no myocardial scar was found (8,36).

Claessen et al. (32) took a different approach. For asymptomatic elite athletes with reduced ejection fraction but no high-risk features like syncope, sustained arrhythmias, or a very high polygenic risk score, they recommended continued competitive sport with regular clinical, echocardiographic, and Holter follow-up (32).

3.3.5 Emergency Response at Endurance Events

Kim et al. (3) delivered the most detailed data on emergency response effectiveness at endurance sporting events. The RACER registry demonstrated that the implementation of organized emergency action planning was the most important factor contributing to the halving of cardiac arrest mortality during marathon and half-marathon events over the past decade. These plans included universal bystander cardiopulmonary resuscitation training, systematic deployment of automated external defibrillators along race courses, and the presence of trained medical personnel at regular intervals. The overall survival rate improved from 29% in the 2000–2009 period to 66% in the 2010–2023 period. This represents a clinically meaningful and statistically significant improvement. The authors observed that most cardiac arrests occurred during the last quartile of the race. This causes the suggestion to strategically concentrated medical resources and AED deployment in these final stages. Survival rates with these protocols are now comparable to other high-performance settings with public-access defibrillation, such as airports and casinos, pointing to the effectiveness of race-day planning. An initial shockable cardiac rhythm and shorter time from collapse to initiation of cardiopulmonary resuscitation were the strongest independent predictors of survival. This shows that the survival outcomes can be dramatically improved through systematic and well-rehearsed emergency response protocols (3).

4. DISCUSSION

4.1 Summary of Main Findings

This systematic literature review included the findings of 33 primary studies published between 2016 and 2026. These examined arrhythmias in endurance athletes, covering over 30 million participants across diverse study designs, and endurance sport disciplines. The review addressed three primary domains as defined by the study objectives: the epidemiology of arrhythmias, their clinical characteristics and diagnostic findings, and the management strategies described in the current literature.

The epidemiological findings confirmed that atrial fibrillation is the most prevalent therapeutic relevant arrhythmia in endurance athletes. The reported prevalence is ranging from 0.3% in young elite athletes (23) observed over 20 years to over 30% in veteran lifelong athletes (8). A consistent dose-response relationship was identified across multiple studies. The dose factors were cumulative training volume and athletic career duration both contributed independently to atrial fibrillation risk. This relationship follows a U-shaped or J-shaped curve (2,11). Moderate physical activity confers protection against atrial fibrillation while very high training volumes, particularly exceeding approximately 2000 lifetime hours of intense exercise, are associated with a significantly elevated risk (27).

Male sex, advancing age, left atrial enlargement and the presence of obstructive sleep apnea were identified as the most consistent risk factors (8,23,27). This review also identified evidence indicating that female endurance athletes are at increased risk of atrial fibrillation (29), and that polygenic risk scores may contribute to individual susceptibility (9).

Ventricular arrhythmias were the second most commonly investigated arrhythmia type. It represented a clinically distinct challenge from atrial fibrillation. The strongest predictor of ventricular arrhythmias was non-ischemic myocardial fibrosis, detected by late gadolinium enhancement on cardiac magnetic resonance imaging (30,31). In 48% of veteran endurance athletes it was identified (31) and led to an approximately four- to five-fold increase in risk (30). Exercise was associated with a markedly higher rate of ventricular arrhythmia occurrence compared to rest (RR 28.6 during physical activity) (15). A newly recognized clinical entity of an isolated subepicardial scar located in the right ventricular outflow tract was documented. It was differentiated from genetic arrhythmogenic cardiomyopathy and responds very well to catheter ablation (33). Fibrosis at the right ventricular insertion point, which was a common finding on cardiac magnetic resonance in athletes, was not associated with arrhythmia risk. This appears to be a benign hemodynamic adaptation (30).

Bradyarrhythmias, particularly sinus bradycardia, were present in up to 96% of elite athletes on Holter monitoring. These were generally considered benign physiological adaptations (35). The literature suggests that former endurance athletes may develop clinically significant conduction system disease which requires pacemaker implantation at substantially younger ages than in the general population (41).

Supraventricular tachycardias, as atrioventricular nodal reentrant tachycardia, demonstrated distinct electrophysiological characteristics in athletes. Structural remodeling of the posteroseptal atrial region leads to significantly higher prevalence of atypical subtypes and higher recurrence rates after catheter ablation (17).

Sudden cardiac arrest at endurance events remained rare (0.60 per 100000 participants). The survival rates have more than doubled over the past decade due to improved emergency response infrastructure (48).

Regarding management, conservative approaches including exercise modification and detraining were the most commonly used strategies. They demonstrated limited effectiveness for reversing established atrial remodeling (8,47). Catheter ablation showed favorable acute outcomes for both supraventricular and ventricular arrhythmias in athletes. High success rates were found for isolated right ventricular outflow tract scar. After time the recurrence rates were higher than in non-athletes for supraventricular tachycardias (17). Implantable device therapy was reserved for high-risk patients. The evidence from the Scandinavian skiing cohorts consistently underlined potential undertreatment with anticoagulation among athletes who met the guideline criteria for stroke prevention therapy (25). Left atrial reservoir strain measured by speckle-tracking echocardiography turned out to be superior to conventional volume measurements for detecting paroxysmal atrial fibrillation in athletes (45,46). This technique offers a more functional assessment of atrial myocardial health for clinical risk stratification.

4.2 Comparison with Existing Literature

The findings of this review are broadly consistent with the existing knowledge on exercise-associated arrhythmias as summarized in recent narrative reviews, meta-analyses, and clinical guidelines (2,4,7,49). A recent meta-analysis by Newman et al. (50) summarized data from 13 studies involving 6816 athletes and 63662 controls. The authors reported that the overall risk of atrial fibrillation was significantly higher in athletes compared to non-athletes (OR 2.46, 95% CI 1.73–3.51). The present review confirms this elevated risk and provides additional details by demonstrating the wide variation in prevalence (0.3–32%). These depend on age, training history, and sport type (50). The dose-response relationship with the threshold of approximately 2000 cumulative lifetime hours of intense exercise (27), is in line with the U-shaped model proposed in earlier reviews by Merghani et al. (51). It also aligns with the clinical counseling framework recommended in the 2020 ESC guidelines on sports cardiology (2).

Heidbuchel (49) proposed the concept that the athlete's heart is naturally a "proarrhythmic heart". The author argued that the same structural and functional adaptations that enable superior athletic performance also create substrates for arrhythmia. The findings of this review provide substantial support for this concept. A continuum between physiological adaptation and pathological vulnerability seems to arise from the nearly universal bradycardia in athletes, the higher prevalence of atypical AVNRT subtypes, the association between left atrial enlargement and atrial fibrillation, and the relationship between myocardial fibrosis and ventricular arrhythmias. This framework affects

how clinicians evaluate and manage arrhythmias in athletes. Arrhythmias in this population may not always show the same disease process as in sedentary individuals (49). The present review extends this literature in several important areas.

First, the finding by Drca et al. that female endurance athletes have a significantly elevated risk of atrial fibrillation (HR 3.67). This is the first robust evidence addressing a critical gap in the previous male evidence base (29). Prior meta-analyses, such as Newman et al., were unable to perform sex-specific subgroup analyses due to the lack of female athletes data (50). The 2020 ESC guidelines on sports cardiology explicitly acknowledged the lack of sex-specific evidence as a major limitation (2). The evidence on sex differences in exercise-associated atrial fibrillation risk is not uniform. Svedberg et al. (24) reported that female cross-country skiers had a substantially lower risk of atrial fibrillation compared to female non-skiers (HR 0.55). Male skiers showed a slightly increased risk (HR 1.11) (24). This contrasts with the findings of Drca et al. (29), who revealed a significantly elevated risk in female marathon runners and cyclists (HR 3.67). The apparent discrepancy may be explained by differences in study populations, exercise intensity, and follow-up duration. In the Svedberg cohort recreational skiers with a wide range of fitness levels were included (24), while the Drca cohort specifically studied competitive female endurance athletes over nearly three decades (29). These contrasting findings highlight the complexity of the sex-exercise-atrial fibrillation relationship and shows that the type, and duration of endurance training may influence atrial fibrillation risk differently in women compared to men (29). The finding by Drca et al. directly addresses this. They suggest that the previously held assumption of female sex as a protective factor against exercise-associated atrial fibrillation requires revision (29).

Second, polygenic risk scores appear to play a role in modulating the relationship between exercise and arrhythmia development. Claessen et al. (32) showed it for ventricular remodeling and Flannery et al. (9) for atrial fibrillation. These findings gives a new dimension to research on exercise-associated arrhythmias. Based on their results, exercise-associated arrhythmias may not be a purely acquired condition from chronic hemodynamic overload. It is more a complex interaction between genetic predisposition and cumulative exercise exposure. This matches the findings of the observation that many highly trained athletes with similar training volumes never develop arrhythmias, while others develop them relatively early in their careers. Polygenic risk assessment could eventually enable personalized exercise recommendations based on an individual's genetic susceptibility profile.

Third, the characterization of myocardial fibrosis as a key predictor of ventricular arrhythmias in veteran endurance athletes. The VENTOUX study (30) and Farooq et al. (31) demonstrate the growing recognition that fibrotic substrate is centrally involved in exercise-associated ventricular arrhythmogenesis. This review work adds clinically important meaning by showing that not all patterns of late gadolinium enhancement have the same arrhythmogenic potential. The specific

finding that fibrosis at the right ventricular insertion point in the VENTOUX cohort was not associated with an increased risk of ventricular arrhythmias resolves a common clinical dilemma, as this pattern is frequently seen in cardiac magnetic resonance imaging of athletes and has led to diagnostic uncertainty in the past (30). If validated in larger cohorts, this finding can help clinicians avoid unnecessary activity restrictions and further invasive examinations in athletes whose only abnormality is this seemingly benign pattern.

Fourth, the finding that atrial remodeling persists after training cessation (8,47). This challenges the traditional view that the athlete's heart is fully reversible upon detraining. Ventricular dimensions appear to normalize relatively rapidly after training cessation (8,47). But the left atrium may represent a structural 'point of no return' after which the dilation becomes permanent. This observation has important clinical implications for the timing of exercise modification. It may be more effective to prevent excessive atrial enlargement instead of attempting reversal of established remodeling. The concept of differential reversibility between cardiac chambers is consistent with the "dam-wall" hypothesis proposed by McNamara et al. (44), which attributes the disproportionate atrial vulnerability to the thinner myocardial wall of the atrium compared to the ventricle. In this context, the finding by Sørensen et al. that left atrial reservoir strain was superior to conventional volume-based assessment for identifying athletes with paroxysmal atrial fibrillation (AUC 0.740) is extremely relevant (45). Since left atrial volumes overlap considerably between athletes without atrial fibrillation and non-athletes with atrial fibrillation, strain-based functional parameters may offer a more reliable approach to distinguishing physiological from pathological atrial remodeling in endurance athletes (45,46).

Fifth, Venlet et al. (33) presented an isolated subepicardial scar in the right ventricular outflow tract with a new clinical entity. This expands the recognized spectrum of exercise-associated cardiac conditions beyond the previously described exercise-induced arrhythmogenic cardiomyopathy as originally proposed by Heidbuchel et al. (49). The absence of genetic mutations, the failure to meet Task Force criteria for arrhythmogenic right ventricular cardiomyopathy, and the remarkably high ablation success rate in this entity distinguish it clearly from genetic arrhythmogenic cardiomyopathy. All of these support the importance of expert evaluation by clinicians with specific expertise in sports cardiology when assessing endurance athletes. The findings from La Gerche et al. about the challenges of differentiating physiological from pathological right ventricular phenotypes in athletes, provide additional context for understanding this entity (52).

Sixth, stroke risk in athletes with atrial fibrillation. The Scandinavian skiing cohorts (11,24,25) showed that endurance athletes with atrial fibrillation have lower stroke rates than non-athletes with atrial fibrillation. This is an observation that was not specifically addressed in the 2020 ESC guidelines on sports cardiology (2) or in the 2020 ESC guidelines for the diagnosis and management

of atrial fibrillation (53). Several hypotheses may explain this paradox. Athletes have a more favorable cardiovascular risk factor profile. Regular exercise also has anti-inflammatory and antithrombotic effects. Myrstad et al. (25) found that atrial fibrillation remains a significant stroke risk factor in athletes aged 65 years and older. A substantial proportion of these athletes are not receiving guideline-indicated anticoagulation. The authors suggest that the protective effect of athletic fitness may diminish with advancing age and comorbidity accumulation. This is why systematic stroke risk assessment and guideline-adherent anticoagulation are so important in older athletes with atrial fibrillation.

4.3 Clinical Implications

The findings of this systematic review have several important implications for clinical practice in sports cardiology and sports medicine. This affects screening, diagnosis, management and emergency preparedness.

For screening and risk stratification, several studies suggest a dose–response relationship between cumulative training volume and the risk of atrial fibrillation (27). Athletes with very high lifetime training exposure appear to be particularly affected. Especially, those exceeding approximately 2,000 hours of intense exercise, as described by Calvo et al. (27), may benefit from periodic cardiac rhythm monitoring. Boraita et al. (23) and Sørensen et al. (45) described echocardiographic tools that may help identify athletes at increased risk of atrial fibrillation. A left atrial anteroposterior diameter of 41 mm or greater was detected as a potential risk threshold (23). Left atrial reservoir strain by speckle-tracking echocardiography showed better diagnostic performance compared to conventional volume measurements (AUC 0.740) (45). It may be preferred as a screening parameter, as it captures functional atrial impairment that precedes or accompanies the volumetric changes seen in athlete's heart (45,46).

For ventricular arrhythmia risk assessment in veteran endurance athletes, cardiac magnetic resonance imaging with late gadolinium enhancement appears essential, especially when athletes present with ventricular ectopy or symptoms suggestive of ventricular arrhythmias (29,30). The presence and pattern of myocardial fibrosis carries strong prognostic value. The finding that right ventricular insertion point fibrosis is not associated with arrhythmia risk may help practitioners avoid unnecessary activity restriction in athletes with this common and likely benign imaging finding (30). The earliest evidence on the role of polygenic risk scores and their change of the relationship between exercise and arrhythmia risk, raises the possibility of genetic-based risk stratification in endurance athletes (9,32). In the future, exercise recommendations could possibly be personalized based on an individual's genetic susceptibility to exercise-associated cardiac remodeling. Combining genetic data

with traditional risk factors like age, sex and training volume could lead to better risk prediction models. The evidence is not yet mature enough for clinical implementation.

For diagnostic evaluation, the evidence from this review strongly supports the use of extended monitoring strategies in athletes with suspected arrhythmias. Resting electrocardiography and short-term ambulatory monitoring may not be sufficient to capture the intermittent and exercise-induced arrhythmias in the athletic population. Implantable loop recorders provide the most long-term surveillance but are invasive and expensive. A 9-day continuous monitoring protocol offers a non-invasive alternative for initial evaluation (38). It captured more arrhythmia events than a standard 24-hour Holter monitoring. To extended rhythm monitoring, clinicians should integrate maximal exercise stress testing into the diagnostic workup of symptomatic athletes. Javed et al. showed that atypical exercise-induced premature ventricular contractions with structural imaging findings together, help to identify athletes at high risk for sustained events during competition (15). The high rates of false-positive arrhythmia alerts from consumer wearable devices found by Gajda et al. (43) are also worth considering. These are relevant in the current era of widespread wearable technology adoption among athletes. Wearable alerts should therefore always be verified with standard electrocardiographic methods before starting further cardiac workup.

For management decisions, the evidence from this review supports the individualized and shared decision-making approach. This is recommended in the 2020 ESC guidelines on sports cardiology (2) and the 2024 HRS expert consensus statement on arrhythmias in the athlete (14). Persistent atrial remodeling after training cessation shows that exercise restriction alone may not eliminate the arrhythmia risk and should be carefully balanced against the well-documented cardiovascular, metabolic and psychological benefits of continued physical activity (8,47). Catheter ablation appears to be an effective and safe treatment option for athletes with both supraventricular and ventricular arrhythmias (33). The high success rate of catheter ablation for isolated right ventricular outflow tract scar provides a particularly encouraging therapeutic option for this specific clinical phenotype (33). In contrast, the higher recurrence rate of AVNRT after ablation in athletes needs to be communicated to patients as part of the informed consent process (17). The potential undertreatment with anticoagulation in older athletes with atrial fibrillation deserves attention. The clinical challenge is complicated by the “stroke paradox” observed in Svedberg et al. (24). Endurance athletes with atrial fibrillation show lower absolute stroke rates than non-athletes with the same diagnosis. This may create a false sense of security and contribute to undertreatment (24). Based on the current evidence, anticoagulation should be used in athletes with atrial fibrillation and elevated CHA_2DS_2-VASc score (25) or changed since 2024 CHA_2DS_2-VA score (53).

For race-day safety, Kim et al. (3), documented a significant improvement in survival from sudden cardiac arrest at endurance events (29% to 66%). These data show the effectiveness of organized

emergency action planning. These results are directly relevant to race organizers and event medical directors. It supports the continued investment in automated external defibrillators along racecourses, systematic bystander cardiopulmonary resuscitation training programs, and standardized medical coverage protocols at endurance sporting events. Emergency preparedness needs sustained attention and resources as the most impactful intervention for reducing exercise-related cardiac mortality (3).

4.4 Strengths and Limitations

This systematic review has several strengths. First, the literature search was conducted across four electronic databases. By using a comprehensive and reproducible search strategy, with a priori defined inclusion and exclusion criteria, 33 primary studies were identified. These studies include over 30 million participants as combined study population. With this, it is one of the broadest systematic syntheses on the topic of arrhythmias in endurance athletes at this point. Second, the present review covers the full spectrum of arrhythmias in endurance athletes. It includes atrial fibrillation, ventricular arrhythmias, bradyarrhythmias, supraventricular tachycardias, and sudden cardiac arrest. This broad scope allows a wide understanding of the cardiac rhythm consequences of chronic endurance exercise. It demonstrates the common underlying mechanisms across different arrhythmia types. Third, the methodological quality of all included studies was formally assessed using the Newcastle-Ottawa Scale (20), adapted NOS (21) or the Jadad scale (22) for cohort, case-control, and cross-sectional studies, with a prior defined criteria for each quality domain. This provides a standardized and reproducible quality evaluation. Fourth, the restriction of the inclusion period to 2016–2026 ensures that the review captures the most recent evidence. This includes new findings on genetic risk factors and advanced diagnostic techniques, as well as the description of novel clinical entities. These are isolated right ventricular outflow tract scar, and evolving management strategies.

Several limitations of this review should be acknowledged. First, the study selection and data extraction were performed by a single reviewer. This may lead to selection or extraction bias. Predefined inclusion and exclusion criteria were used to mitigate this risk. A second, independent screening was not feasible within the scope of this master's thesis, as recommended by the PRISMA 2020 guidelines (18).

Second, the significant heterogeneity in study designs represents a challenge for evidence synthesis in this field. The included studies covered diverse populations, ranging from young elite athletes to veterans, varied sport disciplines from swimming to cross-country skiing, and a wide range of diagnostic approaches from self-report to implantable loop recorders. This heterogeneity made direct comparison difficult, and a quantitative meta-analysis was not possible. As an example, the wide range of reported atrial fibrillation prevalence, from 0.3% to 32%, illustrates this heterogeneity.

Prevalence estimates should therefore always be interpreted in the context of the specific population studied. Some studies relied on retrospective recall of lifetime training history (25,26). This method has lower validity and reliability compared to objective tracking data.

Third, several included studies relied on self-reported arrhythmia diagnoses. This may underestimate the true prevalence of arrhythmias due to the frequently asymptomatic nature of paroxysmal atrial fibrillation and other intermittent rhythm disturbances. Especially the studies, where only hospital-diagnosed arrhythmias were recognized, were potentially missing subclinical episodes.

Fourth, the majority of the included studies investigated predominantly male athletes of white European ethnicity. These facts limit the generalizability of the findings to female endurance athletes and other ethnicities.

Fifth, three pairs of included studies shared overlapping cohorts: the VENTOUX cohort (15,30), the Birkebeiner cross-country skiing cohort (45,46), and the Spanish elite athlete registry (23,35). These overlapping cohorts were transparently documented in the methods section. Each study addressed a different research question and outcomes. But this overlap should be considered when assessing the independence and weight of the evidence. The range of evidence is restricted with this overlapping, but the study depth in these defined athletic groups is increased. Sixth, some potentially relevant studies identified during the Scopus search could not be included because full-text articles were not accessible despite reasonable efforts. It may introduce a degree of publication access bias. Finally, the restriction of the search to English and German-language articles may have excluded relevant studies published in other languages.

4.5 Recommendations for Future Research

Based on the findings and limitations found in this systematic review, the following priorities for future research can be identified.

First, there is a clear need for large-scale prospective studies specifically investigating arrhythmia risk and cardiac remodeling in female endurance athletes. A first step was taken by Drca et al. (29) with a small sample size and limited to two sport disciplines. Future studies should include women across a broader range of endurance sports and training intensities. These should examine whether the dose-response relationship, risk factors, and underlying mechanisms for arrhythmias differ between sexes. The low proportion of female participants in the included studies shows a broader issue in sports cardiology research that needs to be addressed.

Second, further investigation is needed for the role of genetic factors in exercise-associated arrhythmias. The polygenic risk score approaches described by Claessen et al. (32) for ventricular remodeling and Flannery et al. (9) for atrial fibrillation were applied in relatively small and predominantly white European cohorts of athletes. Larger, multi-ethnic studies are needed to validate

these findings. These should determine whether genetic risk stratification can be translated into clinically useful tools for personalized exercise prescription. This future genomic research should concentrate on common genetic background variation, as recent evidence suggest that these are the dominant genetic driver for atrial fibrillation (9). The specific interaction between genetic predisposition and cumulative training volume in finding the arrhythmia risk is an important and currently unanswered question. Understanding this interaction could help identify athletes who may benefit from more conservative training recommendations or more intensive monitoring.

Third, the natural history and long-term clinical prognosis of myocardial fibrosis in endurance athletes requires further clarification through longitudinal studies with serial imaging. The currently available evidence is largely cross-sectional. This allows no definitive conclusions about whether fibrosis in athletes is progressive, stable, or potentially reversible with training modification. Long-term prospective studies which include serial cardiac magnetic resonance imaging in athletes with and without fibrosis at baseline would be particularly valuable. Such studies could also examine the finding of ongoing myocardial inflammation in fibrotic segments. These were mentioned by Farooq et al. (31) and more information of the clinical development and potential response to anti-inflammatory interventions would be therapeutically relevant. In these future studies the isolated subepicardial right ventricular outflow tract scar could examine in detail for better therapeutic approaches (33).

Fourth, there is still no clear evidence for the optimal management of atrial fibrillation in endurance athletes. Catheter ablation is widely used in clinical practice and detraining is frequently recommended, but there is no randomized controlled trial which directly compared these strategies in an athletic population. The ongoing NEXAF detraining trial, reported in Apelland et al. (16), may provide the first randomized evidence on the effectiveness of structured exercise reduction as a therapeutic intervention for atrial fibrillation in athletes. Randomized trials comparing different management strategies are needed to establish evidence-based treatment recommendations specific to the athletic population. These should include the pill-in-the-pocket approach, rhythm control with antiarrhythmic drugs, catheter ablation, and exercise modification.

Fifth, the standardization of arrhythmia definitions, diagnostic protocols, and outcome measures across studies would facilitate future evidence synthesis. It would improve the comparability and clinical applicability of research findings. The wide variation in reported prevalence rates across the studies is partly attributable to differences in monitoring duration, monitor technology (ranging from resting ECG to implantable loop recorders), arrhythmia detection thresholds, and classification systems. Developing uniform monitoring protocols, consensus-based arrhythmia definitions, and standardized outcome reporting frameworks for athletic populations would be a major step forward. Future studies should focus on the development and clinical validation of motion-resistant detection

algorithms for consumer wearables (43). This would minimize the false-positive alerts and the resulting psychological impact on healthy athletes. The validation of speckle-tracking echocardiography parameters, particularly left atrial reservoir strain, as standardized screening tools for atrial fibrillation risk in athletes across different age groups and ethnic backgrounds represents another important research priority.

Sixth, organized emergency action planning has significantly improved survival from sudden cardiac arrest at endurance events (3). This provides a strong case for continued investment in race-day medical infrastructure. Future research should evaluate the cost-effectiveness of different emergency response models, determine the optimal density and placement of automated external defibrillators. It should also explore if targeted pre-race screening protocols can identify athletes at the highest risk of sudden cardiac arrest. In these studies, care should be taken to avoid an unacceptable burden of false-positive findings and as a result unwarranted activity restriction. The right balance between effective screening and avoiding unnecessary harm to healthy athletes is still one of the biggest challenges in preventive sports cardiology.

5. CONCLUSIONS

Based on the systematic review of the 33 primary studies published between 2016 and 2026, the following can be concluded in relation to the four objectives of this thesis:

1. Atrial fibrillation is the most prevalent arrhythmia in endurance athletes. It follows a U-shaped dose-response relationship with cumulative training volume. Male sex, older age, left atrial enlargement, and obstructive sleep apnea are the most consistent risk factors. Recent evidence confirms that female athletes are also at significantly increased risk. Ventricular arrhythmias affect up to 22% of veteran athletes with myocardial fibrosis. Bradyarrhythmias are nearly universal but generally benign. Sudden cardiac arrest survival at endurance events has more than doubled over the past decade.

2. Disproportionate left atrial enlargement is the most consistent structural correlate of atrial fibrillation in endurance athletes. It persists after training cessation. Non-ischemic myocardial fibrosis is the strongest predictor of ventricular arrhythmias. Fibrosis at the right ventricular insertion point appears benign. Polygenic risk scores and left atrial reservoir strain by speckle-tracking echocardiography are seem to be tools for risk stratification and diagnosis.

3. Management requires an individualized and shared decision-making approach. Catheter ablation is effective for both: Supraventricular and ventricular arrhythmias. Particularly high success rates are registered for isolated right ventricular outflow tract scar. Exercise modification alone has limited effectiveness for reversing established atrial remodeling. Anticoagulation may be underutilized in

older athletes with atrial fibrillation. Organized emergency action planning is the most effective intervention for reducing cardiac arrest mortality at endurance events.

4. The quality of the available evidence is predominantly good to fair. Because of the heterogeneity across studies a quantitative meta-analysis was not possible. Important evidence gaps include the underrepresentation of female athletes, the need for validation of genetic risk stratification tools, the lack of randomized trials comparing management strategies in athletes, and the lack of standardized arrhythmia definitions and monitoring protocols for athletic populations.

5.1 Practical Recommendations

1. Veteran endurance athletes with high cumulative training volumes should consider a periodic cardiac rhythm monitoring, which should include echocardiographic assessment of left atrial dimensions and left atrial reservoir strain.

2. Cardiac magnetic resonance imaging with late gadolinium enhancement should be performed in veteran athletes presenting with ventricular ectopy. With paying attention to the right ventricular insertion point fibrosis alone should not cause an activity restriction.

3. Extended multi-day ambulatory monitoring should be preferred over standard 24-hour Holter for initial arrhythmia evaluation. Wearable device alerts should be verified with standard electrocardiographic methods.

4. Management decisions should follow a shared decision-making model that balances arrhythmia risk with the cardiovascular, metabolic, and psychological benefits of continued athletic exercising.

5. Older athletes with atrial fibrillation should undergo systematic stroke risk assessment. The guideline-indicated anticoagulation should not be withheld based on athletic status alone.

6. Organizers of endurance events should organize an extensive emergency action planning, which should include automated external defibrillator deployment and bystander cardiopulmonary resuscitation capability.

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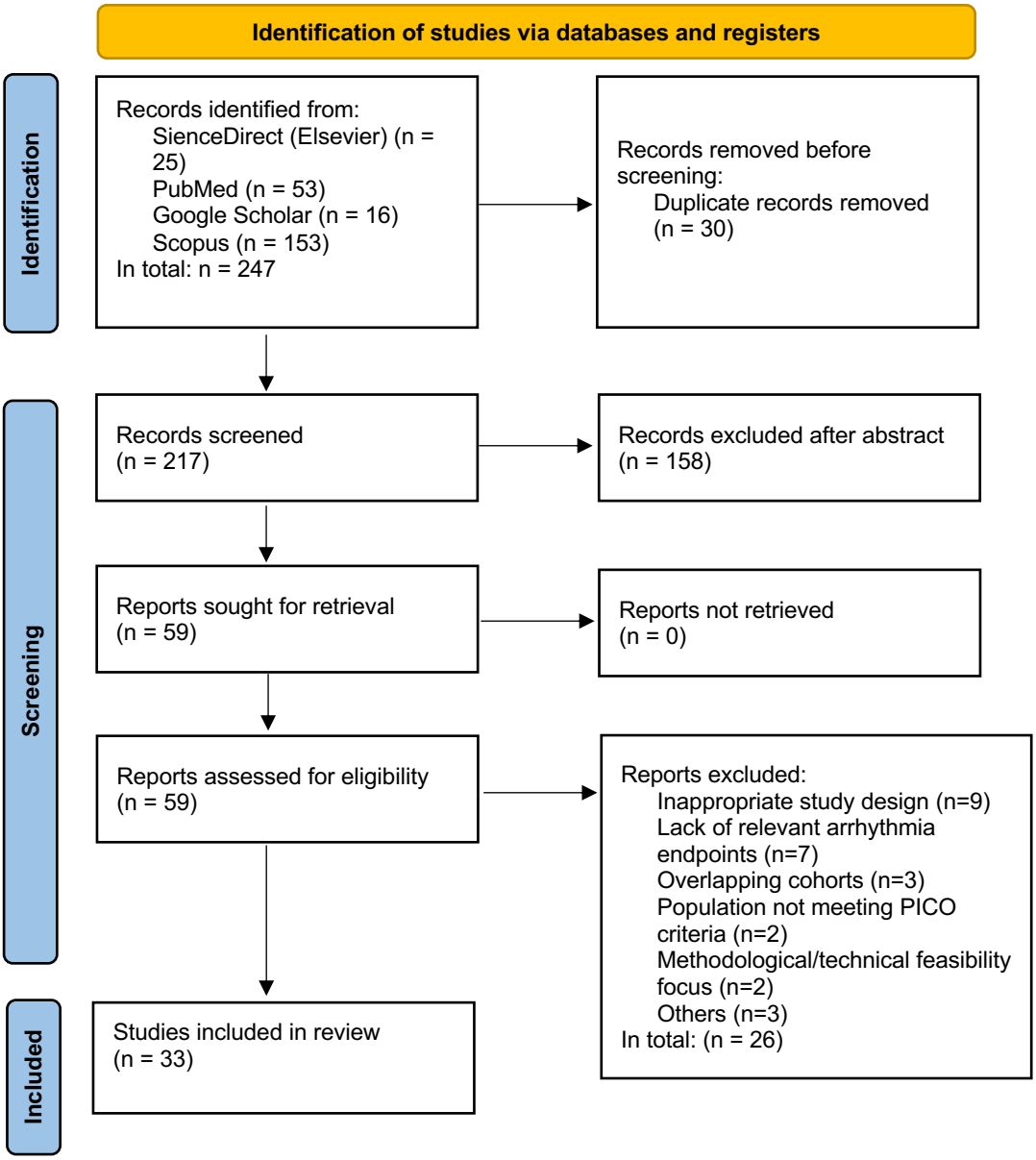
APPENDICES

APPENDIX A

Search Strings

Database	Search Strings
PubMed	(("Endurance Training"[Mesh] OR "Athletes"[Mesh] OR "Sports"[Mesh] OR "endurance athlete*" OR "marathon runner*" OR cyclist* OR triathlete* OR rower*) AND ("Arrhythmias, Cardiac"[Mesh] OR "atrial fibrillation"[Mesh] OR "ventricular arrhythmia*" OR "bradycardia" OR "tachyarrhythmia") AND ("Epidemiology"[Mesh] OR "prevalence" OR "clinical characteristic*" OR "management" OR "treatment" OR "diagnosis"))
ScienceDirect	endurance athletes AND arrhythmia
Google Scholar	Allintitle: arrhythmia OR atrial fibrillation OR ventricular arrhythmia AND endurance athletes OR runner OR marathon OR cyclist
Scopus	TITLE-ABS-KEY(("endurance athlete*" OR "marathon runner*" OR cyclist* OR triathlete* OR rower* OR "cross-country skier*" OR "long-distance runner*") AND (arrhythmia* OR "atrial fibrillation" OR "ventricular arrhythmia*" OR "supraventricular tachycardia" OR bradycardia* OR "premature ventricular")) AND PUBYEAR > 2015 AND PUBYEAR < 2027

APPENDIX B



PRISMA 2020 Flow Diagram
Source: (18)

APPENDIX C
Study characteristics

N°	Author (Year)	Journal	Study Design	Cohort size (n)	Age (Years)	Male (%)
1	Elliott et al. (2020)	European Heart Journal	Prospective cohort	402406	40-69	47.6
2	Schreiner et al. (2016)	Sports Medicine - Open	Cross-sectional	149	72.6 ± 4.9	NR
3	Javed et al. (2025)	Circulation: Cardiovascular Imaging	Prospective cohort	106	≥50	100%
4	Javed et al. (2026)	European Journal of Preventive Cardiology	Prospective cohort	106	≥50	100%
5	Miljoen et al. (2019)	EP Europace	Retrospective	504	30.9±14.6	78.8%
6	Svedberg et al. (2019)	Circulation	Retrospective registry	736102	37.3	60.6%
7	Herm et al. (2017)	BMJ Open	Prospective observational	108	48	76%
8	Calvo et al. (2016)	Europace	Prospective case-control	172	46±10	87%
9	Kim et al. (2025)	JAMA	Observational cohort	29.3 Million,	NR	72%
10	Elliott et al. (2018)	Clinical Cardiology	Cross-sectional	99	45-54	Predom-inantly male
11	D'Ambrosio et al. (2025)	Journal of Sport and Health Science	Cross-sectional	266	≥40	79-96%
12	Apelland et al. (2025)	BMJ Open	Prospective multicentre	43	57±10	93%
13	McNamara et al. (2019)	Circulation	Randomized Control Trial + Cross-sectional	61+14	53±5	56.6%
14	Sørensen et al. (2023)	Echocardiography	Cross-sectional	293	70.7±6.7	100%
15	Boraita et al. (2018)	JAMA Cardiology	Retrospective cohort	6813	22±7	65%
16	Gajda et al. (2018)	Scandinavian Journal of Medicine & Science in Sports	Prospective diagnostic	142	26±6.9	73%
17	Matsumura et al. (2019)	Sports	Cross-sectional survey	2819	49.1	68.2%
18	Kaleta et al. (2018)	Advances in Clinical and Experimental Medicine	Prospective observational	40	39±8	100%

19	Climstein et al. (2025)	Sports	Cross-sectional	29	20.6±4.1	50%
20	Drca et al. (2023)	British Journal of Sports Medicine	Retrospective matched	228+1368	32	0%
21	Bondarev et al. (2024)	Journal of Clinical Medicine	Prospective observational	361	69±13	NR
22	Venlet et al. (2017)	Journal of the American College of Cardiology	Retrospective cohort	57	48±16	83%
23	Boraita et al. (2022)	Frontiers in Cardiovascular Medicine	Retrospective cohort	654	24	72%
24	Farooq et al. (2023)	Scientific Reports	Cross-sectional	50+26	56	100%
25	Sørensen et al. (2022)	European Heart Journal - Cardiovascular Imaging	Cross-sectional	302	70.8±6.7	100%
26	Aaroee et al. (2025)	BMJ Open Sport & Exercise Medicine	Prospective longitudinal	50	23±5	70%
27	Claessen et al. (2024)	Circulation	Prospective cohort	281	22±8	79.7%
28	Vanova et al. (2022)	Journal of Physical Education and Sport	Prospective cohort	280	29.4±9.14	86.8%
29	Johansen et al. (2022)	Open Heart	Prospective cohort	2372	≥65	100%
30	Myrstad et al. (2020)	European Journal of Preventive Cardiology	Retrospective cohort	2626	64-66	Predom-inantly male
31	Cavigli et al. (2022)	European Heart Journal - Cardiovascular Imaging	Prospective observational	68	47.9±7.8	69%
32	Wundersitz et al. (2026)	Journal of the American Heart Association	Prospective cohort	34	52.0±12.7	70.6%
33	Flannery et al. (2025)	European Heart Journal	Prospective cohort	121+ 11495	62	74.4%

N°	Author (Year)	Sport	Arrhythmia	Key findings	Management	Database
1	Elliott et al. (2020)	Self-reported physical activity	AF, VA, Brady	Intense physical activity +12% AF risk	Not described	PubMed
2	Schreiner et al. (2016)	Swimming	AF	AF 26.5% vs 7%, Odds ratio 8.74	Not described	PubMed
3	Javed et al. (2025)	Cycling, Triathlon	VT, NSVT	47.2% fibrosis, 21.7% VA, HR 4.7	ICD, Electro-physiological	PubMed

					study, training restriction	
4	Javed et al. (2026)	Cycling, Triathlon	VT, NSVT	23.5% VA, Relative risk 28.6	ICD, Electro-physiological study, training restriction	PubMed
5	Miljoen et al. (2019)	Variety	AVNRT	45% atypical in athletes, 10% vs 4% recurrence	Radiofrequency ablation	PubMed
6	Svedberg et al. (2019)	Skiing	AF	Female skiers lower AF HR 0.55, Lower stroke risk	Anticoagulation	PubMed
7	Herm et al. (2017)	Marathon	NSVT, AF	nsVT 9.4%, AF 0.9%	cMRI	Google Scholar
8	Calvo et al. (2016)	Cycling, Marathon	Lone AF	U-shaped: ≥2000h OR 3.88, OSA OR 5.01	OSA screening	PubMed
9	Kim et al. (2025)	(Half-) Marathon	SCA	0.60/100,000, mortality halved, CAD 40%	AED, emergency plans	PubMed
10	Elliott et al. (2018)	Variety endurance	AF, PAC, PVC, Brady	LA larger >6000h	Detraining, Ablation	PubMed
11	D'Ambrosio et al. (2025)	Rowing, Cycling, Triathlon	AF, NSVT, Brady, SVT	AF 32%, no difference active vs retired athletes	Pacemaker, ICD	Science Direct
12	Apelland et al. (2025)	Cycling, Skiing, Running	Paroxysmal AF	AF burden 0.18%, 30% reduced quality of life	Ablation, Pill-in-pocket	Science Direct
13	McNamara et al. (2019)	Endurance	AF	LA volume +55% after 24 months, no AF detected	Not described	Science Direct
14	Sørensen et al. (2023)	Skiing	Paroxysmal AF	Left atrial mechanical dispersion associated with pAF	Ablation	Google Scholar
15	Boraita et al. (2018)	Variety	AF	0.3% AF/20y, age, competition years & LA size predictive	Left atrial monitoring	Science Direct
16	Gajda et al. (2018)	Running, Cycling	SVE, VE, AVNRT	39% no arrhythmia, 45% HRM artifacts, 1 AVNRT	ECG, Holter ECG, Echo, further follow up	Scopus
17	Matsumura et al. (2019)	Running	AF	2.4% AF, training years OR 1.455/decade	Not described	Scopus
18	Kaleta et al. (2018)	Marathon	Brady	92.5% ECG abnormal, 62.5% sinus bradycardia	Further investigations	Scopus

19	Climstein et al. (2025)	Triathlon	Brady, AVB	68.2% sinus bradycardia, 13.6% 1st AVB	Echo	Scopus
20	Drca et al. (2023)	Marathon, Cycling	AF	AF 4.4% vs 1.7%, HR 3.67, first female study	Not described	Scopus
21	Bondarev et al. (2024)	Variety	SND, AVB	Endurance = earlier diagnosis 64 vs 72y	Pacemaker	Scopus
22	Venlet et al. (2017)	Cycling, Running	SVT	RVOT scar, 91% ablation success	Epicardial ablation, ICD	Scopus
23	Boraita et al. (2022)	Variety	Brady, PVC, NSVT, AVB	96% bradycardia, PVC 39.4%, NSVT 1.5%	Monitoring athletes with mitral valve regurgitation	Scopus
24	Farooq et al. (2023)	Cycling, Triathlon	PVC, couplets	48% fibrosis vs 15%, higher PVC, T2 signal elevated	cMRI	Scopus
25	Sørensen et al. (2022)	Skiing	Paroxysmal AF	LASr better, AUC 0.740	Ablation	Scopus
26	Aaroee et al. (2025)	Endurance	AF	LA dilation persisted, LV normalized	Not described	Scopus
27	Claessen et al. (2024)	Cycling, Rowing	PVC, NSVT, SCD	15.7% reduced EF, polygenic risk score associated, 1 SCD	Follow up	Scopus
28	Vanova et al. (2022)	Variety	AVNRT, VT, PVC, AF	5.3% pathology, AF only in endurance athletes	Radiofrequency ablation, ICD	Scopus
29	Johansen et al. (2022)	Skiing	AF	AF 28.5% vs 17.8%, lower stroke RR 0.60	Not described	Scopus
30	Myrstad et al. (2020)	Skiing	AF	AF 12.3%, stroke in ≥65y adjusted OR 2.16	Anticoagulation	Scopus
31	Cavigli et al. (2022)	Ultramarathon	PVB	7% exercise PVBs, no NSVT, QTc prolonged	Not described	Scopus
32	Wundersitz et al. (2026)	Cycling	PVC, NSVT, AF, SVT	68% abnormal, VA higher on training day	Screening over days	Scopus
33	Flannery et al. (2025)	Rowing	AF	6.8x AF prevalence, polygenic risk score	Not described	Scopus

Abbreviations: AF: Atrial fibrillation, VA: Ventricular arrhythmia, Brady: Bradyarrhythmia, VT: Ventricular tachycardia, NSVT: Non-sustained ventricular tachycardia, AVNRT: Atrioventricular nodal re-entrant tachycardia, SCA: Sudden cardiac arrest, PAC: Premature atrial contraction, PVC: Premature ventricular contraction, SVT: Sustained ventricular tachycardia, SVE: Supraventricular extrasystole, VE: Ventricular extrasystole, AVB: Atrioventricular block, SND: Sinus node dysfunction, SCD: Sudden cardiac death, OR: Odds ratio, RVOT: Right ventricular outflow tract

APPENDIX D

Quality Assessment:

Newcastle-Ottawa Scale:

No.	Author (Year)	Study design	S1	S2	S3	S4	C	O1	O2	O3	Total (/9)	Quality
1.	Elliott (2020)	Prospective Cohort	⋆	⋆	⋆	⋆	⋆⋆	⋆	⋆	⋆	9	Good
2.	Javed (2025)	Prospective Cohort	⋆	⋆	⋆	⋆	⋆	⋆	⋆	⋆	8	Good
3.	Javed (2026)	Prospective Cohort	⋆	⋆	⋆	⋆	⋆	⋆	⋆	⋆	8	Good
4.	Miljoen (2019)	Retrospective	⋆	⋆	⋆	⋆	⋆	⋆	⋆		7	Good
5.	Sevdberg (2019)	Retrospective registry	⋆	⋆	⋆	⋆	⋆⋆	⋆	⋆	⋆	9	Good
6.	Herm (2017)	Prospective Cohort	⋆		⋆	⋆		⋆	⋆	⋆	6	Fair
7.	Calvo (2016)	Prospective case control	⋆	⋆	⋆	⋆	⋆⋆	⋆	n/a	n/a	7	Good
8.	Kim (2025)	Observational cohort	⋆		⋆	⋆		⋆	⋆		5	Fair
9.	Apelland (2025)	Prospective multicentred cohort	⋆		⋆	⋆		⋆	⋆	⋆	6	Fair
10.	Boraita (2018)	Retrospective cohort	⋆		⋆	⋆	⋆	⋆	⋆	⋆	7	Good
11.	Gajda (2018)	Prospective diagnose	⋆		⋆	⋆		⋆		⋆	5	Fair
12.	Kaleta (2018)	Prospective observation	⋆		⋆	⋆		⋆		⋆	5	Fair
13.	Drca (2023)	Retrospective matched	⋆	⋆	⋆	⋆	⋆⋆	⋆	⋆	⋆	9	Good
14.	Bondarev (2024)	Prospective observation	⋆	⋆	⋆	⋆	⋆	⋆			6	Fair
15.	Venlet (2017)	Retrospective cohort	⋆	⋆	⋆	⋆	⋆	⋆	⋆	⋆	8	Good
16.	Boraita (2022)	Retrospective cohort	⋆		⋆	⋆	⋆	⋆			5	Fair
17.	Aaroee (2025)	Prospective longitudinal	⋆		⋆	⋆		⋆	⋆	⋆	6	Fair
18.	Claessen (2024)	Prospective Cohort	⋆		⋆	⋆	⋆	⋆	⋆	⋆	7	Good
19.	Vanova (2022)	Prospective Cohort	⋆		⋆	⋆	⋆	⋆	⋆	⋆	7	Good
20.	Johansen (2022)	Prospective Cohort	⋆	⋆	⋆	⋆	⋆⋆	⋆	⋆		8	Good
21.	Myrstad (2020)	Retrospective cohort	⋆		⋆	⋆	⋆			⋆	5	Fair
22.	Cavigli (2022)	Prospective observation	⋆		⋆	⋆		⋆		⋆	5	Fair

23.	Wundersitz (2026)	Prospective Cohort	⋆		⋆	⋆		⋆	⋆	⋆	6	Fair
24.	Flannery (2025)	Prospective Cohort	⋆	⋆	⋆	⋆	⋆ ⋆	⋆	⋆	⋆	9	Good

Source: (20)
Abbreviations:
S1: Representativeness of the exposed cohort
S2: Selection of the non-exposed cohort
S3: Ascertainment of exposure
S4: Demonstration that outcome of interest was not present at start of study
C: Comparability of cohorts on the basis of design or analysis (max. 2: first for age, second for additional confounders)
O1: Assessment of outcome
O2: Was follow-up long enough for outcome to occur
O3: Adequacy of follow-up of cohorts
Quality: 0-3 = Poor, 4-6 = Fair, 7-9 = Good

Modified Newcastle-Ottawa Scale, Cross-sectional studies:

No.	Author (Year)	S1	S2	S3	C	O1	O2	Total (/7)	Quality	
1.	Schreiner (2016)	⋆			⋆⋆		⋆	4	Fair	
2.	Elliot (2018)	⋆		⋆	⋆	⋆	⋆	5	Good	
3.	D'Ambrosio (2025)	⋆	⋆	⋆	⋆⋆	⋆	⋆	7	Good	
4.	Sørensen (2023)	⋆	⋆	⋆	⋆⋆	⋆	⋆	7	Good	
5.	Matsumura (2019)	⋆	⋆		⋆		⋆	4	Fair	
6.	Climstein (2025)	⋆		⋆	⋆	⋆	⋆	5	Good	
7.	Farooq (2023)	⋆		⋆	⋆⋆	⋆	⋆	6	Good	
8.	Sørensen (2022)	⋆	⋆	⋆	⋆⋆	⋆	⋆	7	Good	

Source: (21)
Abbreviations:
S1: Representativeness of the sample
S2: Sample size justified or adequate
S3: Non-respondents described or response rate adequate
C: Comparability of subjects (max. 2: first for age, second for additional confounders)
O1: Assessment of outcome using validated method
O2: Appropriate statistical test with confidence intervals reported
Quality: 0-2 = Poor, 3-4 = Fair, 5-7 = Good

Jadad scale (22):
McNamara (2019) is a Randomized Control Trial
Score: 3/5 - randomized, described as randomized, no blinding due to exercise intervention nature
Quality = Good