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MITRAL ANNULAR DISJUNCTION IN ATHLETES: AN UNDERRECOGNIZED ARRHYTHMOGENIC SUBSTRATE AT THE INTERFACE OF SPORTS CARDIOLOGY AND SUDDEN CARDIAC DEATH PREVENTION

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ABSTRACT

Background: Mitral annular disjunction (MAD) is a structural abnormality of the mitral valve apparatus characterized by atrial displacement of the leaflet hinge point away from the left ventricular myocardium. Once considered an incidental finding, MAD is now recognized as a component of the arrhythmic mitral valve prolapse phenotype and a potential substrate for ventricular arrhythmias and sudden cardiac death, particularly in physically active individuals.
Objectives: To summarize current evidence regarding the anatomy, epidemiology, arrhythmogenic mechanisms, multimodality imaging, risk stratification, and management of MAD, with emphasis on its relevance in athletes.
Methods: A narrative review of the literature was performed using PubMed/MEDLINE and major cardiology and sports medicine journals. Publications from 1999 to 2024 were reviewed using the terms “mitral annular disjunction”, “arrhythmic mitral valve prolapse”, “athlete”, “exercise”, “sudden cardiac death”, “eligibility”, and “return to play”. Priority was given to recent guidelines, expert consensus documents, systematic reviews, large cohort studies, and landmark publications.
Results: Physiological cardiac remodeling in athletes may obscure the diagnosis of MAD, while exercise-related hemodynamic and autonomic changes may facilitate ventricular arrhythmias in susceptible individuals. Current recommendations support individualized risk assessment and shared decision-making rather than universal sports restriction.
Conclusions: MAD should be considered during cardiovascular evaluation of athletes with bileaflet mitral valve prolapse, unexplained ventricular arrhythmias, or a family history of sudden cardiac death. Further studies are needed to establish athlete-specific risk stratification and long-term management strategies.

KEYWORDS

Mitral Annular Disjunction; Mitral Valve Prolapse; Sports Cardiology; Ventricular Arrhythmia; Sudden Cardiac Death; Athlete's Heart

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1. Introduction

Sudden cardiac death (SCD) in athletes remains a rare but devastating event that continues to drive research into arrhythmogenic substrates unique to, or unmasked by, intensive exercise. Attention in sports cardiology has traditionally focused on hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, coronary artery anomalies, and, increasingly, on wearable-detected atrial fibrillation. A comparatively underappreciated entity is mitral annular disjunction (MAD), an anatomical separation between the atrioventricular junction and the basal left ventricular myocardium that disrupts the normal mechanical continuity of the mitral annulus [1].

MAD was first described pathologically decades ago but has only recently re-entered clinical prominence following the identification of a distinct “arrhythmic mitral valve prolapse” (AMVP) phenotype, in which bileaflet myxomatous prolapse, MAD, complex ventricular ectopy, and papillary muscle or inferobasal fibrosis coexist and confer a disproportionate risk of malignant ventricular arrhythmia [2,3]. Because this phenotype tends to affect young to middle-aged individuals, often women, with structurally near-normal hearts and preserved ejection fraction, it is precisely the population that also populates recreational and competitive sport [4].

The purpose of this review is to consolidate current knowledge on MAD as it pertains specifically to athletes: its anatomy and pathophysiology, its epidemiological overlap with exercise-associated cardiac remodeling, the mechanisms by which exertion may precipitate malignant arrhythmia, the diagnostic pathway across imaging modalities, current risk stratification tools, and the practical question that dominates the clinic — whether, and under what conditions, an athlete with MAD can safely continue competitive or recreational sport.

2. Aim and Methods of the Review

This is a systematically informed narrative review. Relevant literature was identified through structured searches of PubMed/MEDLINE and major cardiology and sports-medicine journal databases using combinations of the terms “mitral annular disjunction”, “arrhythmic mitral valve prolapse”, “athlete”, “exercise”, “sudden cardiac death”, “eligibility”, and “return to play”. Priority was given to expert consensus statements, systematic reviews, large cohort studies, and imaging-based validation studies published within the last decade, supplemented by seminal earlier work establishing the arrhythmic MVP phenotype. Case reports were included selectively to illustrate diagnostic or management dilemmas specific to athletes. The review was based on publications published between 1999 and 2024, with particular emphasis on studies from the last five years and current international guidelines. Given the narrative design, no formal risk-of-bias assessment or meta-analytic synthesis was performed; the aim is to provide clinicians and sports cardiologists with a coherent, clinically oriented synthesis rather than an exhaustive systematic appraisal.

3. Definition, Anatomy and Pathophysiology of Mitral Annular Disjunction

Anatomically, the mitral annulus should represent a continuous fibrous attachment between the atrial and ventricular myocardium and the base of the mitral leaflets. In MAD, the hinge point of the posterior, and occasionally the anterior, leaflet is displaced atrially, creating a gap between the leaflet insertion and the crest of the left ventricular myocardium that becomes most apparent in systole [1]. This disjunction is frequently, though not invariably, associated with mitral valve prolapse (MVP), and is considered one of several “red flag” features that mark a minority of MVP patients as being at increased arrhythmic risk [5].

Mechanistically, the disjunction alters the normal transmission of mechanical stress across the annulus. During systole, the unrestrained annular segment and the prolapsing leaflet impart excessive traction on the papillary muscles and adjacent basal inferolateral wall, producing a distinctive “systolic curling” motion of the myocardium [6]. Repetitive mechanical stretch of this kind is thought to promote localized myocardial fibrosis, most consistently identified in the posteromedial papillary muscle and inferobasal wall on late gadolinium

enhancement cardiac magnetic resonance (CMR) imaging [7,8]. This fibrotic substrate, combined with mechanically triggered premature ventricular contractions (PVCs) arising from the papillary muscles or fibrous tissue, is currently regarded as the leading explanation for the malignant ventricular arrhythmias observed in this population, following a classic substrate-plus-trigger model of arrhythmogenesis [4].

An important refinement in the recent literature is the distinction between “true” MAD, representing genuine separation of the atrioventricular junction, and “pseudo-MAD”, an echocardiographic mimicker related to annular flattening and systolic curling in the setting of significant mitral regurgitation without true anatomical disjunction. Because pseudo-MAD is far more prevalent and correlates with regurgitant severity rather than with the arrhythmic substrate itself, its recognition is essential to avoid overdiagnosis and unwarranted restriction of athletes whose annular appearance is a secondary consequence of volume overload rather than a primary arrhythmogenic lesion.

4. Epidemiology: From the General Population to Athletic Cohorts

Mitral valve prolapse affects approximately 2–3% of the general population and, in the large majority of individuals, follows a benign course [9]. Reported MAD prevalence varies considerably according to the imaging modality used and the population studied, ranging from roughly one-fifth of patients on transthoracic echocardiography to substantially higher figures when transesophageal echocardiography or CMR are used, reflecting the superior spatial resolution of these techniques for identifying subtle annular separation. Only a small minority of individuals with MVP and MAD, generally estimated at well under 2% per year, will ever experience a life-threatening ventricular arrhythmia or SCD [10].

Dedicated epidemiological data on MAD specifically within athletic populations remain sparse. Most available evidence derives from case series, single-center cohorts, and case reports of athletes or individuals in physically demanding occupations, such as military aviators, who present after an aborted cardiac arrest and are subsequently found to have bileaflet MVP with MAD. These reports, while inherently subject to ascertainment bias, are instructive precisely because they demonstrate that the arrhythmic MVP/MAD phenotype is not confined to sedentary, older populations but can present catastrophically in young, highly conditioned individuals with no prior cardiac symptoms. This observation underscores the need for greater awareness of MAD within pre-participation cardiovascular evaluation programs, which have historically prioritized cardiomyopathies, ion channelopathies, and coronary anomalies over valvular-annular pathology.

5. The Overlap Between MAD and Physiological Athletic Cardiac Remodeling

Long-term, high-volume endurance and mixed-discipline training produces well-described physiological adaptations of the heart, including chamber dilation, increased wall thickness, and altered diastolic filling patterns collectively termed the “athlete's heart” [11]. These adaptations can complicate the echocardiographic assessment of the mitral valve apparatus in several ways. First, physiological annular dilation associated with chronic volume loading may itself produce mild degrees of leaflet billowing that can be mistaken for pathological MVP if standard diagnostic thresholds are not applied rigorously. Second, exercise-induced left ventricular enlargement can alter the geometric relationship between the papillary muscles, the mitral leaflets, and the annulus in ways that are not yet fully characterized in longitudinal athlete cohorts.

Conversely, and of greater clinical concern, subtle degrees of true MAD may be overlooked in athletes precisely because abnormal annular or leaflet motion is too readily attributed to benign remodeling. The 2020 European Society of Cardiology (ESC) guidelines on sports cardiology emphasize that echocardiographic findings in athletes should always be interpreted in the context of training status, sport discipline, and body habitus, but they also caution against reflexively normalizing structural findings that would be considered pathological in a non-athletic individual [12]. Distinguishing physiological remodeling from an early arrhythmogenic MAD/MVP phenotype therefore requires a systematic approach that integrates leaflet morphology, annular measurements at multiple points, systolic curling assessment, and, where indicated, tissue characterization by CMR, rather than reliance on any single echocardiographic parameter.

6. Arrhythmogenic Mechanisms Specific to the Exercising Heart

Exercise imposes acute hemodynamic and autonomic changes that may specifically interact with the MAD/AMVP substrate. Dynamic exertion increases heart rate, sympathetic tone, and catecholamine levels, all of which lower the threshold for triggered activity from the papillary muscles and fibrotic myocardial segments implicated in AMVP. Increased preload and inotropy during exercise can also transiently accentuate leaflet prolapse and annular traction, thereby amplifying the mechanical stimulus that is thought to drive PVC generation in this condition [3].

Direct evidence that exercise itself provokes clinically relevant ventricular arrhythmia in MVP patients has begun to accumulate. A prospective evaluation of exercise-induced ventricular ectopy in MVP patients using symptom-limited exercise testing with continuous electrocardiographic monitoring found that a meaningful proportion of patients developed severe forms of ventricular arrhythmia specifically during or immediately after peak exertion, reinforcing exercise as a proximate trigger rather than merely a bystander activity. Complementary work using long-term ambulatory monitoring in patients with established arrhythmic mitral valve syndrome has shown that ventricular ectopic burden and complex arrhythmia are not uniformly distributed across the day but cluster around periods of physical exertion and heightened sympathetic activity [13].

7. Diagnostic Pathway: Electrocardiography, Echocardiography, CMR and CT

The diagnostic pathway for suspected MAD in an athlete typically begins with a resting 12-lead electrocardiogram (ECG) and a comprehensive transthoracic echocardiogram (TTE), consistent with standard pre-participation and symptom-driven cardiovascular evaluation. Electrocardiographic clues associated with the arrhythmic MVP/MAD phenotype include repolarization abnormalities such as negative or biphasic T waves in the inferior leads, and frequent or complex premature ventricular complexes, particularly those with a right bundle branch block morphology suggesting a left ventricular papillary muscle or fascicular origin [5].

On TTE, MAD is best visualized in the parasternal long-axis view as a discrete separation between the posterior mitral annulus and the adjacent ventricular wall at end-systole, ideally measured at its maximal longitudinal extent. However, TTE has recognized limitations in sensitivity relative to transesophageal echocardiography and CMR, both of which allow more precise delineation of the extent of disjunction and its relationship to leaflet prolapse and to systolic curling of the inferolateral wall [6]. CMR additionally provides tissue characterization through late gadolinium enhancement, enabling identification of the papillary muscle and inferobasal fibrosis that constitutes the arrhythmogenic substrate rather than merely documenting the anatomical lesion. Cardiac computed tomography has emerged as a further complementary tool, particularly useful for detailed annular geometry and for pre-procedural planning when catheter ablation or surgical repair is considered. Current expert guidance suggests reserving CMR for athletes with phenotypic risk features such as bileaflet prolapse, moderate or greater mitral regurgitation, unexplained syncope, complex ventricular ectopy, or a family history of unexplained sudden death, rather than applying it indiscriminately to all athletes with incidental mild prolapse.

8. Risk Stratification: The “Malignant” Phenotype and Red Flags

Risk stratification in AMVP and MAD relies on the integration of clinical, electrocardiographic, and imaging “red flags” rather than any single decisive test. Recognized high-risk features include bileaflet myxomatous prolapse, female sex, a personal history of unexplained syncope, a family history of premature or unexplained SCD, complex ventricular ectopy (multifocal PVCs, couplets, non-sustained or sustained ventricular tachycardia), inferior T-wave inversion, and demonstrable myocardial fibrosis of the papillary muscles or inferobasal wall on CMR [2,14,15]. A longer measured MAD distance and the presence of pronounced systolic curling have both been associated with a greater likelihood of ventricular arrhythmia in cohort studies of MVP patients with echocardiographically confirmed MAD.

Importantly, isolated mild MAD in the absence of prolapse, ectopy, fibrosis, or symptoms appears to carry a comparatively low absolute arrhythmic risk, and current expert consensus cautions against equating the mere anatomical presence of disjunction with high-risk disease. This distinction is particularly relevant to sports cardiology, where the consequence of overdiagnosis is unnecessary disqualification of an asymptomatic athlete, while the consequence of underdiagnosis is a missed opportunity to identify a genuinely malignant substrate before a catastrophic event occurs. A staged, phenotype-driven approach to escalating investigation, beginning with ECG and TTE and reserving ambulatory monitoring, exercise testing, and CMR for those with red-flag features, currently represents the most rational compromise [16].

9. Exercise as a Trigger: Dose–Response Relationships and the Exercise Paradox

A central and still incompletely resolved question in this field is whether habitual high-volume exercise itself increases arrhythmic risk in individuals with MVP and MAD, independent of its acute triggering effect during a single bout of exertion. A retrospective cohort study quantifying lifetime exercise dose in metabolic equivalent task hours per week among MVP patients found that, contrary to the assumption that higher lifetime training volumes would proportionally increase risk, patients who experienced severe ventricular arrhythmia were not uniformly those with the highest cumulative exercise exposure; several severe arrhythmic events occurred during only mild or moderate intensity activity, and a proportionally higher risk of arrhythmia was observed during exercise relative to the resting state [17].

These findings, together with data suggesting that athletes with MVP may have a broadly similar incidence of clinical events to non-athletic MVP populations, have led some authors to question whether blanket exercise restriction is justified for all MVP or MAD patients irrespective of phenotype. Current exercise guidelines nonetheless continue to recommend that patients with MVP who additionally demonstrate arrhythmic risk factors should limit high-intensity exercise, while allowing more liberal participation for those without such features, an approach that again foregrounds the importance of individualized, phenotype-based rather than diagnosis-based restriction [12].

10. Sports Eligibility, Return-to-Play and Shared Decision-Making

Sports eligibility assessment for athletes with known or suspected MAD remains, by the acknowledgment of the specialist societies themselves, an area with limited high-quality evidence and considerable practice variation between national frameworks. The 2020 ESC sports cardiology guidelines and national frameworks such as the Italian guidelines for competitive sports eligibility recommend a first-line evaluation comprising history, physical examination, resting ECG, and comprehensive echocardiography, escalating to maximal exercise stress testing, ambulatory ECG monitoring incorporating a representative training session, and CMR when phenotypic risk features are present [12].

The 2024 Heart Rhythm Society expert consensus statement on arrhythmias in the athlete marks a notable philosophical shift in this field, reframing the central clinical question from whether an athlete may return to sport toward how return to sport can be safely facilitated, emphasizing shared decision-making between athlete and clinician and individualized risk communication over blanket restriction [16]. Applied to MAD and AMVP, this framework favors a structured, phenotype-driven pathway: athletes without high-risk features may reasonably continue competitive activity with periodic reassessment, whereas those with sustained ventricular tachycardia, aborted cardiac arrest, or a high-burden “red flag” phenotype require more definitive risk mitigation, which may include activity modification, pharmacological therapy, catheter ablation, or device therapy, before any return-to-play decision is finalized. Because the underlying evidence base in athletes specifically remains limited, such decisions are best made in specialized sports cardiology centers with access to multimodality imaging and electrophysiological expertise.

11. Therapeutic Strategies: Catheter Ablation, Device Therapy and Surgery

For athletes with symptomatic or high-burden ventricular ectopy localized to a papillary muscle or fascicular focus, catheter ablation has emerged as an effective option for reducing arrhythmic burden and, in carefully selected cases, permitting resumption of competitive sport once repeat ambulatory monitoring and maximal exercise testing confirm absence of complex or exercise-induced ventricular arrhythmia. Left-sided ablation procedures typically require a period of oral anticoagulation, which itself necessitates a temporary interruption of contact or collision sports until treatment is complete [16].

Implantable cardioverter-defibrillator (ICD) therapy follows the same general indications in AMVP/MAD as in other arrhythmogenic substrates, being reserved principally for secondary prevention after documented sustained ventricular tachycardia, ventricular fibrillation, or aborted cardiac arrest without another clearly reversible cause, and considered on an individualized basis for primary prevention in patients with a particularly high-risk phenotype [5,7,16]. Surgical mitral valve repair, addressing both the regurgitant lesion and, where feasible, the annular disjunction itself, has been associated in case-based and cohort literature with favorable reverse cardiac remodeling and reduction in arrhythmic burden postoperatively, suggesting that correction of the mechanical substrate may itself be arrhythmia-modifying rather than purely valve-restorative [2,4,6]. Nonetheless, robust randomized or large-scale prospective data specifically evaluating these interventions in athletic populations are currently lacking, and management decisions continue to rely heavily on extrapolation from broader MVP/MAD cohorts [10,12,16].

12. Research Gaps and Future Directions

Several priority areas emerge from this review. First, there is a clear need for prospective, athlete-specific cohort studies quantifying the true prevalence of MAD across different sporting disciplines and training volumes, ideally incorporating standardized multimodality imaging protocols to reduce diagnostic heterogeneity. Second, validated risk-stratification models specifically calibrated to athletic populations, rather than extrapolated from general cardiology cohorts, are needed to avoid both unnecessary disqualification and missed high-risk cases. Third, longitudinal outcome data following catheter ablation, device therapy, and surgical repair in athletes with AMVP/MAD would materially strengthen return-to-play guidance, which currently rests largely on expert opinion and small case series. Finally, given the described female predominance of the arrhythmic MVP/MAD phenotype, sex-specific data in athletic cohorts, an area explicitly flagged as understudied by recent consensus statements, represent an important and currently underexplored research priority [16,18].

13. Conclusions

Mitral annular disjunction is an increasingly recognized, mechanically driven arrhythmogenic substrate that can affect structurally young, highly conditioned hearts and therefore merits specific consideration within sports cardiology. Its diagnostic overlap with physiological athletic remodeling and with the more benign "pseudo-MAD" appearance associated with mitral regurgitation makes accurate identification of the genuinely high-risk phenotype clinically demanding. A structured, phenotype-driven approach, integrating clinical history, ECG, echocardiography, and selectively applied CMR, together with individualized, shared decision-making regarding sports participation, currently represents the most defensible clinical strategy. Given the paucity of athlete-specific outcome data, clinicians should maintain a high index of suspicion in athletes presenting with bileaflet prolapse, unexplained complex ventricular ectopy, syncope, or a relevant family history, and should refer such individuals to specialized sports cardiology services for comprehensive risk stratification before eligibility decisions are made.

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Conceptualization, S.J., J.S. and T.W.; methodology, J.S.; formal analysis, J.S. and M.Ś.; investigation, T.W., A.N.O., and M.Ś.; resources, T.W., A.N.O., and M.Ś.; data curation, T.W., A.N.O., and M.Ś.; writing-original draft preparation, T.W., A.N.O., M.Ś. and S.J.; writing-review and editing, S.J. and J.S.; supervision, S.J., J.S., and M.Ś.; project administration, S.J. All authors have read and agreed to the published version of the manuscript.

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