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SPECIAL ARTICLE
Epidemiology, prevention, and health care policies

Masters athletes with abnormal cardiovascular findings: a clinical consensus statement of the European Association of Preventive Cardiology of the ESC and the American College of Cardiology

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Abstract

Exercise training improves cardiovascular health and reduces the risk for future cardiovascular events and mortality. However, emerging evidence suggests that Masters athletes may have a higher prevalence of cardiovascular abnormalities, such as atrial arrhythmias, coronary atherosclerosis, aortic dilatation, and myocardial fibrosis, compared to their less active peers. The clinical management of Masters athletes may be challenging as available guidelines for such conditions are generally based on data derived from symptomatic sedentary patients, limiting their applicability to highly trained individuals. Other unique challenges in the clinical assessment of Masters athletes include differences in symptomatic presentations compared to sedentary individuals, potential

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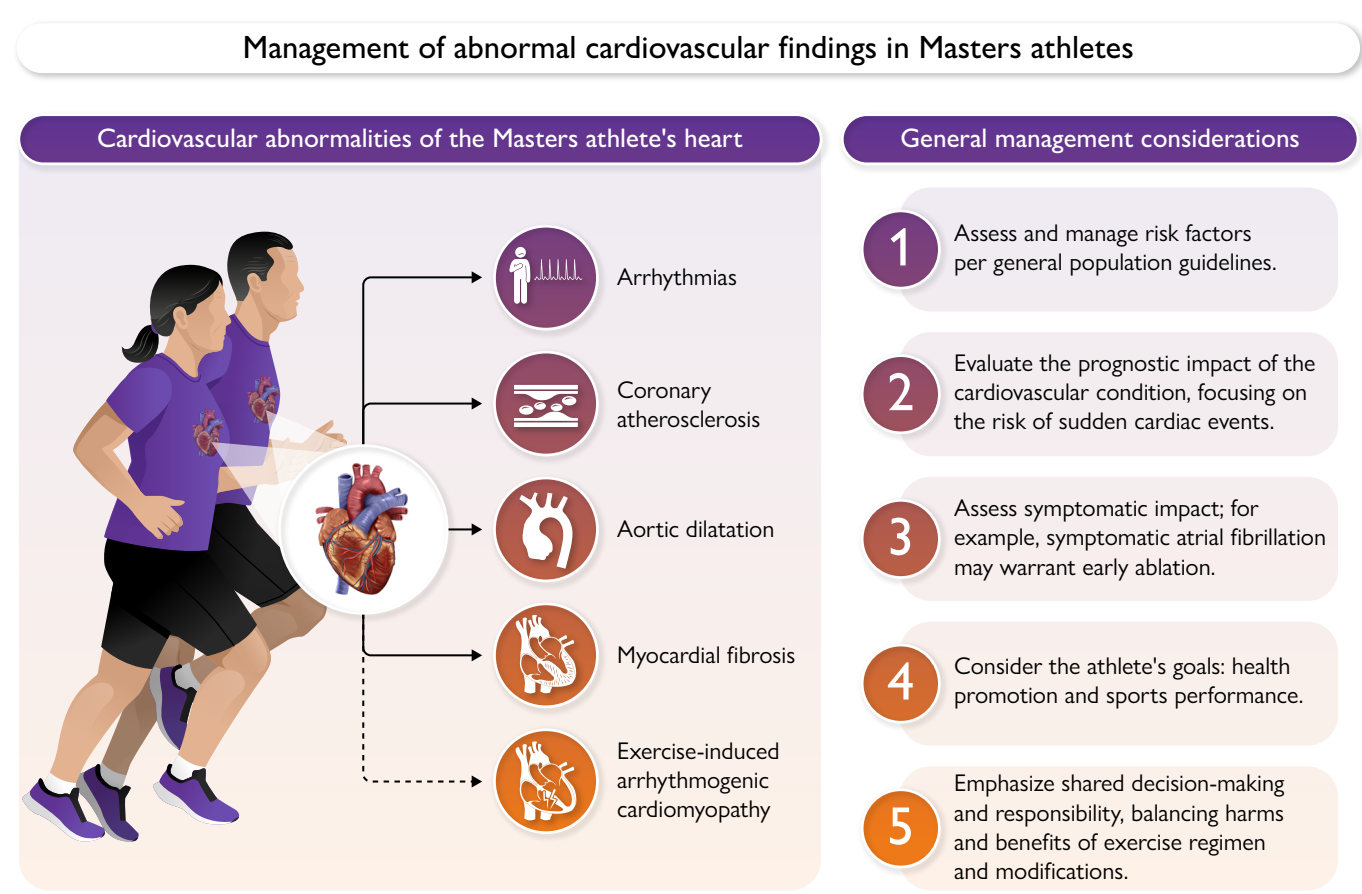
resistance to the initiation of pharmacologic treatment, and the increasing availability of consumer wearable health data that may provide relevant information on their cardiovascular health status. The purpose of this joint EAPC/ESC and ACC Clinical Consensus Statement is to provide an in-depth update on the current state of knowledge on abnormal cardiovascular findings in Masters athletes. We present an expert-based approach on the diagnostic assessment, management, and prognosis of (i) atrial fibrillation, (ii) bradyarrhythmias, (iii) ventricular arrhythmias, (iv) coronary atherosclerosis, (v) aortic dilatation, (vi) myocardial fibrosis, and (vii) exercise-induced arrhythmogenic cardiomyopathy. Clinical challenges, areas of ongoing controversy, and uncertainty and the potential underlying mechanisms are discussed. We also present future perspectives and research directives to further refine current best practice strategies. This includes the need for clinical outcome studies, dedicated randomized controlled trials in athletes, and international registries with diverse populations and longitudinal follow-up to evaluate the natural history of cardiac abnormalities and facilitate development of evidence-based approaches in the clinical management of Masters athletes with cardiovascular abnormalities.

Graphical Abstract



Overview of cardiovascular abnormalities encountered in Masters athletes, including arrhythmias, coronary atherosclerosis, aortic dilatation, myocardial fibrosis, and exercise-related arrhythmogenic phenotypes, and key principles guiding their clinical management. Decision-making integrates cardiovascular risk assessment, prognostic implications, symptom burden, and the athlete's goals, emphasizing shared decision-making to balance cardiovascular safety with the health and performance benefits of lifelong exercise.

Keywords Exercise • Cardiovascular risk • Atrial fibrillation • Myocardial fibrosis • Coronary atherosclerosis • Aortopathy • Cardiomyopathy • Athlete's heart

Introduction

Habitual physical activity and regular exercise training reduce the risk of cardiovascular disease (CVD) and all-cause mortality.[1,2] The complex association between physical activity dose and health benefits appears to be curvilinear,[3] with the largest risk reductions with small amounts of exercise, and additional benefits with higher exercise volumes.[4] These observations have contributed to the belief that 'more exercise is better'. As a result, the number of people performing regular exercise training and participating in competitive sports during adulthood have markedly increased over recent decades.[5]

Masters athletes are defined as individuals aged 35 years or older that perform habitual exercise training to improve fitness levels and regularly participate in competition (*Figure 1*). Masters athletes' exercise habits consistently exceed contemporary global physical activity recommendations[6] and are primarily driven by performance or competitive goals rather than health goals. Data have shown that those who participate in organized endurance events have a lower mortality risk compared to peers from the general population with greater risk reductions for runners and cyclists compared to long-distance walkers, suggesting an important role for exercise intensity.[7] However, high cardiorespiratory fitness does not exclude the possibility of developing cardiovascular (CV) risk factors and CVD.[8] Emerging evidence suggests that some CV abnormalities are more prevalent in Masters athletes compared to sedentary peers.[9] For example, individuals performing long-term exercise training have an increased prevalence of atrial arrhythmias,[10,11] coronary calcification,[12–14] and aortic dilatation.[15] Furthermore, increased myocardial late gadolinium enhancement (LGE), indicative of myocardial fibrosis (MF), has been reported in lifelong athletes.[16]

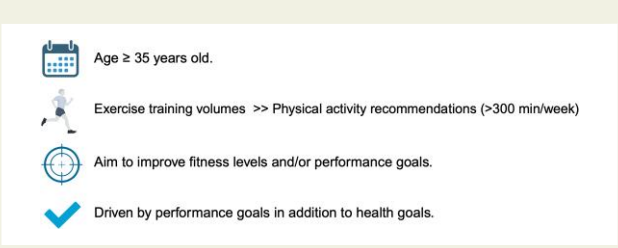


Figure 1 Masters athletes are defined as individuals aged 35 years or older with exercise habits that consistently exceed physical activity recommendations, aimed to improve fitness and achievement of performance goals rather than health goals

Current guidelines for management of common CV conditions are almost exclusively based on data derived from symptomatic sedentary patients with CVD and/or sedentary asymptomatic patients with medium to high risk for adverse CV outcomes.[17] Accordingly, contemporary strategies delineating diagnostic assessment, treatment options, exercise prescription and potential exercise restriction have limited generalizability to Masters athletes with abnormal CV findings. Clinical management in this context is therefore highly variable and often based on small single-centre studies, opinions, and anecdotal experiences of the treating clinician.

This Clinical Consensus Statement provides an in-depth update on the current state of knowledge on abnormal CV findings in Masters athletes (*Graphical Abstract*). We present an expert consensus-based approach to the diagnostic assessment, management, and prognosis of several key cardiac abnormalities observed among Masters athletes with emphasis on clinical challenges and areas of ongoing controversy and uncertainty.

Methodological approach

The writing group was assembled in alignment with the European Society of Cardiology (ESC) and the American College of Cardiology (ACC) gender policy and with efforts to include early career researchers. For the writing process, teams of four to five international experts were assigned to each content section and online sessions were organized to discuss the content and reach consensus on the considerations presented in the tables. The leadership team (T.E., J.K., A.B., and G.C.) harmonized all sections and recommendations and drafted the manuscript. The draft manuscript was subsequently circulated to all co-authors for additional suggestions and feedback, particularly directed to the clinical considerations. This process was repeated until consensus was reached. The strength of advice for clinical management considerations, as dictated by ESC policy, is summarized in *Figure 2*.

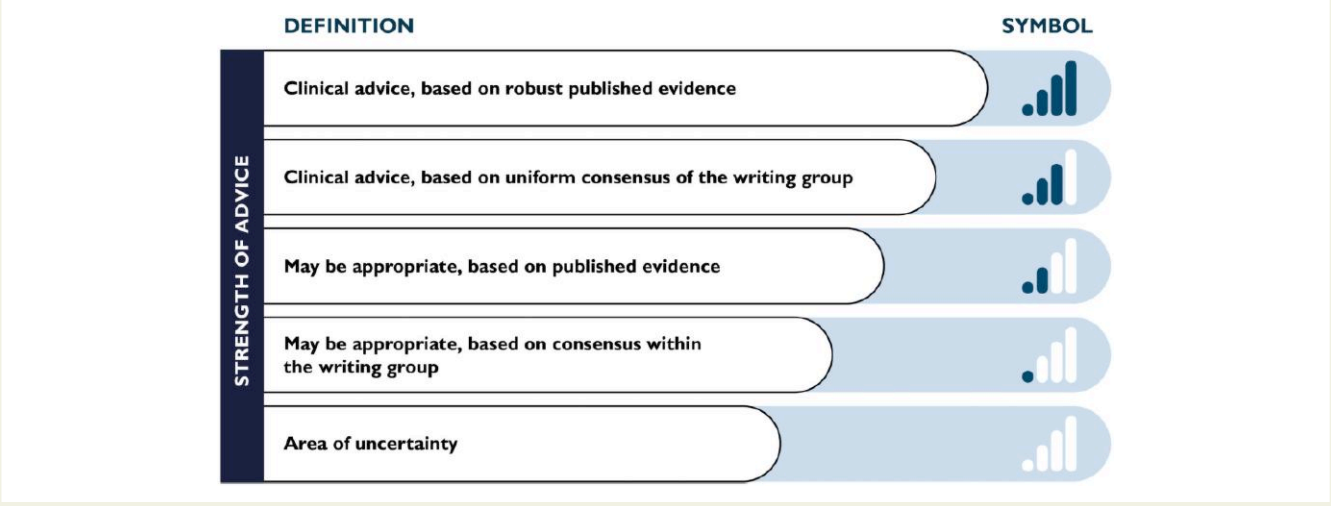


Figure 2 Explanation of 'Strength of advice' categories for Clinical Consensus Statements as per ESC Scientific Document Policy. Clinical advice: where there is evidence or general agreement that a given measure is clinically useful and appropriate, or evidence and general agreement that a given measure is harmful and not appropriate. May be appropriate: where there is evidence or general agreement that a given measure may be clinically useful and appropriate. Areas of uncertainty: no data available, solely based on expert opinion

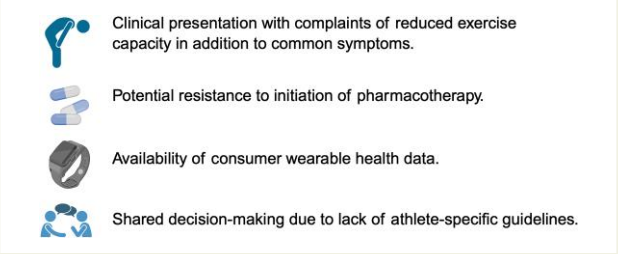


Figure 3 The clinical assessment of Masters athletes includes a number of unique challenges, such as (i) a different clinical presentation, (ii) resistance to pharmacotherapy, (iii) availability of wearable data, and (iv) a shared decision-making approach

Clinical considerations for the masters athlete

The clinical assessment of Masters athletes aligns with routine CV practice, but a number of challenges unique to this population are noteworthy (*Figure 3*). First, Masters athletes often present with a chief complaint of reduced exercise capacity in isolation or in addition to common symptoms (i.e. chest pain, palpitations, etc.). This complaint raises a specific differential diagnosis inclusive of incident CVD, non-CVD systemic pathology, ageing, and physiologic detraining. It is important to quantify reductions in exercise capacity using the individual Masters athlete as his or her own point of reference, as impaired exercise capacity among Masters athletes often substantially exceed normal reference levels of the general population. The potential impact of ageing on exercise capacity deserves attention. In general, age-related declines in exercise capacity, which are inevitable, occur in a gradual fashion over years. Sudden reductions in exercise capacity are not suggestive of an ageing effect and should raise the suspicion for underlying abnormalities. Second, Masters athletes are often resistant to the initiation of pharmacologic treatment. The misconception that routine high-level exercise obviates the need for medication coupled with concerns about the impact of drugs on exercise capacity, particularly statins and agents with negative chronotropic effects (i.e. beta-blockers), are common. Nonetheless, drug therapy coupled with lifestyle modifications are often necessary to address common CV risk factors including hypertension and dyslipidemia in Masters athletes. Third, Masters athletes are increasingly using commercially available fitness devices which provide information that may be relevant to CV health. Clinicians working with Masters athletes are encouraged to develop a basic familiarity with the common metrics provided by these devices (i.e. exercise heart data, heart rate variability scores, acute workload trajectory data, etc.) to effectively integrate this potentially useful information into diagnostic assessment.[18] Fourth, there are scant primary data to guide therapeutic intervention in Masters athletes. Management decisions should account for the limitations of relying on data derived from non-athletic patients and should present what is known and what is unknown in the context of shared decision-making (SDM). SDM includes an explanation of the risks and benefits of potential clinical management options and integrates the clinician's opinion about the risk-benefit balance for each potential management strategy. Finally, the use of performance enhancing drugs should be evaluated in the assessment of the Masters athlete as they may explain presenting symptoms and exacerbate the common underlying forms of abnormal CV findings discussed in this document.

Atrial fibrillation

Atrial fibrillation (AF) is an age-related arrhythmia resulting in part from longstanding underlying atrial cardiomyopathic remodelling. Masters athletes have a lower prevalence of classical AF risk factors (e.g. hypertension, obesity, or diabetes) but a higher prevalence and incidence of AF.[10,11] Among Masters athlete Nordic skiers, the prevalence of AF is approximately twice that among comparable members of the general population.[19] This is true both in men and women, although the absolute AF rates are lower in female than in male athletes.[10] Adjusted hazard ratios for AF prevalence in Masters athletes are estimated to range from 2.5 to 4,[20] with increasing exercise duration and intensity associated with increased risk.[21]

Multiple mechanisms have been hypothesized to explain the increased incidence of AF in Masters athletes.[20] These include (i) structural changes in the atrium, with dilatation caused by pressure and volume overload, (ii) autonomic changes, with heightened vagal tone at rest and higher sympathetic tone during exercise, (iii) electrical changes, with slowing of conduction with increased electrical dispersion, and (iv) development of interstitial fibrosis in the atrium due to possible longstanding low grade inflammation.[22] These effects of remodelling of the athlete's heart on the propensity for AF may extend beyond the active sports career. A recent study showed a four-fold higher risk for AF among former elite athletes with a high polygenic risk score.[23] On the other hand, the polygenic contribution to AF risk was similar in athletes and non-athletes (odds ratio 3.7 vs 2.0, $P = .37$). These findings emphasize the complex interplay between genetics, exercise training and AF, which may additionally be mediated through non-inherited mechanisms.[24]

Given the increased prevalence of AF in Masters athletes, a high clinical suspicion should lead to a low threshold for diagnostic evaluation among individuals presenting with symptoms, such as palpitations, particularly among those with elevated CHA_2DS_2-VA scores. The clinical assessment of suspected AF in athletes is not different compared to non-athletes,[25] but treatment decisions should consider symptoms and quality of life in the context of sports performance (*Table 1*). A rate control strategy for symptomatic Masters athletes is often ineffective. Beta-blockers (prohibited in certain sports), calcium antagonists, and digoxin are infrequently tolerated due to side effects both at rest and during exercise.[26] Thus, rhythm control is usually the preferred management option. Antiarrhythmic drug therapy remains a viable option, but catheter ablation has emerged as an additional and reasonable first-line option. Prior concerns that pulmonary vein isolation (PVI) may be less efficacious in athletes than non-athletes have been disproven in several ablation series showing similar efficacy in both groups.[27–29] Therefore, PVI as a first-line treatment of AF also applies to Masters athletes.[25,30] SDM in this context remains important as complications (which still occur in 1% to 2% of PVI cases) may have an impact on athletic performance. Current data on the impact of PVI on exercise tolerance is limited and warrants further study. One retrospective study comparing exercise treadmill test results before and after PVI found no significant difference.[29] However, only a

Table 1 Clinical consensus statements for Masters athletes with atrial fibrillation

#	Consideration	Strength of the advice
1	Endurance sports-related cardiovascular changes may promote AF at higher training loads, but moderate intensity physical activity should be encouraged given the many physiological and psychological health benefits.	
2	Masters athletes presenting with AF should undergo evaluation for and treatment of standard risk factors (hypertension, obstructive sleep apnea, weight control, alcohol use, and hyperthyroidism).	
3	Anticoagulation for Masters athletes should follow standard guidelines, including the use of anticoagulants for Masters athletes with a CHA_2DS_2-VA score $\geq$2.	
4	Among Masters athletes that meet criteria for anticoagulation, the type of sport and risk of collision/trauma should be integrated with SDM regarding future sports participation.	
5	Medical management should aim for safe sports participation, including adequate rate control during exercise and/or avoidance of exercise for two half-lives after pill-in-the-pocket treatment with Class 1C antiarrhythmics.	
6	Rhythm control is generally preferred over rate control in Masters athletes with AF to optimize performance and enhance quality of life.	
7	With SDM, pulmonary vein isolation is reasonable as an early treatment option in athletes, taking possible complications and the unknown impact on exercise tolerance into account.	
8	It is unknown whether prescribed reduction of endurance sports activity leads to lower rates of AF recurrence.	
9	Left atrial appendage occlusion devices should not be implanted for the sole purpose to avoid anticoagulation in Masters athletes with AF.	

minority of participants underwent testing both pre- and post-procedure. Another study of highly symptomatic athletes with AF, also limited in size, showed improved exercise capacity after radiofrequency catheter ablation.[27] Procedural success after ablation seems comparable to non-athletes, with slow attrition rates at longer follow-up. While high volumes of endurance exercise are a well-established risk factor for AF, it remains under investigation whether prescribed reductions in exercise (detraining) leads to more effective rhythm control.

Small studies suggest that athletes with AF may have lower stroke risk than non-athletes,[11,19] but there are no data supporting athlete-specific anticoagulation algorithms, and stroke risk assessment and prevention in athletes should follow validated risk scores such as the CHA_2DS_2-VA.[25,31] For athletes participating in sports with risk of trauma (i.e. collision or bodily impact), SDM around continued participation is required.[32] Similar to the general population, left atrial appendage occlusion may be appropriate only for Masters athletes with definitive medical contraindications to anticoagulation. There are no data to support the use of left atrial appendage occlusion for the sole purpose of avoiding anticoagulation for Masters athletes without medical contraindications,[25,30] so it is recommended to follow guideline-directed management.

Bradyarrhythmias

Bradyarrhythmias and marked sinus bradycardia are frequent in endurance sport Masters athletes. Sinus bradycardia with rates <40 bpm, first-degree atrioventricular block, and second-degree atrioventricular block type I may occur among healthy asymptomatic Masters athletes under resting conditions.[33] Bradyarrhythmias occur due to a combination of exercise-induced increases in vagal tone and molecular changes of ion channel proteins that lead to an attenuated sinoatrial impulse generation and conduction.[34,35] Importantly, most athletes with sinus bradycardia and benign forms of heart block (i.e. first degree and type-1 second degree) demonstrate appropriate heart rate augmentation during exercise.

In the evaluation and management of Masters athletes, it is essential to determine whether symptoms, such as dizziness, presyncope, isolated exercise intolerance, or fatigue, are linked to clinically significant bradyarrhythmias. The diagnostic approach should include a thorough medical history, exercise testing, and ambulatory rhythm monitoring to assess the chronotropic response to exercise. While specific thresholds are currently unknown for Masters athletes, chronotropic incompetence is defined as a significantly lower than predicted

Table 2 Clinical consensus statements for Masters athletes with bradyarrhythmias

#	Consideration	Strength of the advice
1	The clinical evaluation of Masters athletes presenting with symptoms, that may be attributable to bradyarrhythmias (i.e. dizziness, presyncope, intermittent exercise intolerance, etc.), should include a careful history, exercise testing and ambulatory rhythm monitoring to evaluate chronotropic response to exercise.	
2	In asymptomatic Masters athletes with sinus bradycardia, first degree AV block, or Mobitz 1 AV block, no intervention is required. In contrast in Masters athletes with pathological bradyarrhythmias (type second degree and third degree heart block) decision making regarding pacemaker placement should follow standard clinical guidelines.	
3	It is unknown whether prescribed reductions in sports activity among Masters athletes with chronotropic incompetence reduces the necessity for future pacemaker implantation.	
4	Among Masters athletes with subjectively or objectively reduced exercise capacity and chronotropic incompetence, it is unknown whether pacemaker implantation with rate-response functionality improves exercise capacity.	

heart rate during maximal effort exercise testing. Diagnostic efforts should be made to determine the relationship between maximal achieved heart rate and exercise performance. Currently, it is unknown whether reductions in sports activity for Masters athletes with chronotropic incompetence decreases the likelihood of future pacemaker implantation or if pacemaker implantation with rate-response functionality effectively enhances exercise capacity.

While bradyarrhythmias are most often asymptomatic and reversible, emerging data indicate that long-term endurance exercise may also increase the likelihood of pathological sinus node and conduction system changes.[36] A dose–response relationship was observed in a study of cross-country skiers with those participating in more races having a higher risk of sinus node disease or third-degree atrioventricular block.[37] In a follow-up study, the long-term incidence of bradycardia and the need for pacemaker implantation were, respectively, 19% and 17% more frequent in male skiers compared to the general population and most notably among the best-performing athletes and those competing in more events.[38] However, there is currently no evidence to support the necessity of routine long-term follow-up in these individuals in the absence of symptoms (*Table 2*).

Ventricular arrhythmias

The occurrence of ventricular arrhythmias in Masters athletes is generally not a normal training-related finding and requires clinical attention as this may be the first sign of an underlying cardiac disorder. Asymptomatic premature ventricular contractions (PVCs) or other ventricular arrhythmias are commonly detected in endurance athletes. Previous studies reported a prevalence of 7% in ultra-marathon finishers[39] and 34% among middle-aged and older athletes undergoing preparticipation stress testing.[40] The prevalence of ventricular arrhythmias on Holter monitoring does not appear to differ between Masters athletes and the general population.[41] PVCs are more likely to have higher-risk characteristics in Masters athletes compared to younger athletes,[40] but most prior studies have predominantly focused on younger athletes.[42] The term high-risk PVCs, used to denote a series of morphologic features that increase the pretest probability of disease, has been summarized in previous clinical consensus statements for athletes[25,32,43,44] and includes (i) PVCs with atypical morphology (i.e. those originating from locations other than the left/right ventricular outflow tract or fascicles), (ii) PVCs that are polymorphic, repetitive, or with short coupling interval (<360 ms), (iii) PVCs in combination with other abnormal ECG features for the general population, (iv) non-suppression, new emergence of PVCs with exercise, or increased burden of PVCs during exercise, and (v) symptoms of sudden onset of exercise intolerance associated with emergence of arrhythmias. A high PVC burden in isolation, whether observed on 12-lead ECGs, exercise testing, or 24-h Holter monitoring, has been suggested as a potential high-risk marker.[45] However, it is important to note that thresholds for PVC burden are not yet clearly defined, and clear cut-points linked to clinical outcomes are unavailable in athletic cohorts, complicating the clinical interpretation and application of this finding. Nevertheless, in athletes with a PVC burden >10% by 24-h Holter monitoring, an assessment of cardiac structure and function is reasonable to exclude PVC-induced left ventricular dysfunction.

To what extent PVCs in asymptomatic Masters athletes reflect the presence of underlying disease and/or carry an unfavourable prognosis has not been well defined. While moderate exercise is protective against arrhythmias, excessive and prolonged exercise may induce structural and electrical remodelling of the heart, increasing the risk of ventricular arrhythmias.[46,47] As such care should be taken when evaluating Masters athletes as some of the features that would be flagged as high risk in young athletes may be more prevalent in ostensibly healthy Masters athlete population.[48] Available European[43] and American[25] guidelines only provide recommendations for the younger athlete, so high-risk features need to be defined for Masters athletes in future studies. Among the Masters athlete

Table 3 Clinical consensus statements for Masters athletes with ventricular arrhythmias

#	Consideration	Strength of the advice
1	In asymptomatic Masters athletes with isolated rare (1 on a resting ECG) PVCs of typical morphology (i.e. originating from the outflow tract or fascicles), no further clinical work-up is required.	
2	For Masters athletes with >1 typical PVC, at least 1 atypical PVC on resting ECG, or symptomatic PVCs, the clinical work-up should include family and personal history, echocardiography, exercise testing, ambulatory rhythm monitoring (including a training session) and individualized longitudinal surveillance.	
3	Among Masters athletes with PVC characteristics consistent with higher risk (e.g. atypical morphology, short PVC coupling interval, repetitive PVCs, >10% burden, emergence during exercise) more extensive evaluation, including cardiac magnetic resonance imaging, ischaemic evaluation, and/or invasive electrophysiology study, should be considered.	
4	Management of Masters athletes with high-risk PVCs and/or complex ventricular arrhythmias should be based on the underlying pathology according to disease-specific guidelines.	
5	Individualized longitudinal surveillance is reasonable in Masters athletes with high-risk PVCs and no clearly defined etiology during initial evaluation.	

with PVC characteristics consistent with higher risk, we suggest a clinical work-up (*Table 3*) including family and personal medical history, resting ECG, echocardiography, exercise testing, and 12-lead ambulatory ECG recording. This monitoring should encompass at least one or more training sessions to accurately assess the PVC burden and behaviour during physical exertion. It is currently unknown whether the burden of atypical PVCs is associated with clinical risk. Further investigations including cardiac magnetic resonance (CMR) imaging, electrophysiology studies, and genetic testing, may be appropriate on an individualized basis depending on the results of first-line testing.[25,43,49]

Coronary atherosclerosis

Regular exercise training improves atherosclerotic risk factors, including blood pressure, inflammatory status, lipid levels, and glucose tolerance.[50] However, there is emerging evidence that coronary calcification and atherosclerosis are more common in male,[12–14,51,52] but not female,[53] Masters endurance athletes compared to less active peers. A non-linear association between lifelong exercise volumes and the prevalence of a coronary artery calcium score (CACS) > 100 Agatston units (AU) has been repeatedly observed.[54] Initial studies demonstrated atherosclerotic plaques were predominantly calcified,[12,13] suggesting that exercise training may promote plaque stabilization by increased calcification, similar to the effect of statins.[55] In contrast, the Master@Heart study found that the prevalence of calcified plaques was similar in lifelong athletes vs controls.[14] Nonetheless, the relatively high fitness status of the control group may explain this finding.[56]

Mechanisms underlying coronary atherosclerosis in male Masters athletes remain uncertain. Traditional CV risk factors, such as prior tobacco use, arterial hypertension, and a family history of premature atherosclerotic disease are highly prevalent among middle-aged and older athletes[8,57] and should be assessed in all Masters athletes.[58] Exercise physiology factors including sustained increases in blood pressure and heart rate, mechanical coronary stress, disruption of laminar intra-coronary flow, and exercise-induced inflammation may synergize with traditional CV risk factors to the development and progression of coronary calcification and atherosclerosis.[50] The magnitude of these exercise-induced mechanisms appear to be directly related to exercise intensity,[59] as suggested by data from the MARC-2 study.[60] Additional mechanisms including exercise-induced changes in lipid metabolism and calcium homeostasis,[61,62] the consumption of an atherogenic diet, and undefined genetic factors may also contribute to the increased prevalence of coronary calcification and atherosclerosis in Masters athletes.[50,54] Future studies are warranted to elucidate the role of these proposed pathways and to allow targeted treatment and preventive countermeasures.

Prospective outcome data in Masters athletes with coronary calcification and atherosclerosis are currently unavailable. However, findings from the Cooper Center Longitudinal Study, a general population cohort, provide important insights. Men with CACS <100 AU who engaged in high exercise volume (>3000 MET-min/week) had significantly lower all-cause mortality risk compared to less active peers with CACS <100 AU, but no differences in event rate were observed in high- vs low-volume exercisers with CACS ≥100 AU (*Figure 4*).[52] In another study, those with the highest fitness levels had the lowest CV risk for each defined CACS category.[63] However, even among the fittest subgroup, the CV event rate was higher in those with CACS >400 AU vs CACS = 0. In summary, higher amounts of exercise and improved cardiorespiratory fitness appear to reduce the risk of coronary artery calcification[52,63,64] but do not negate the effect of higher CACS on CV event risk.

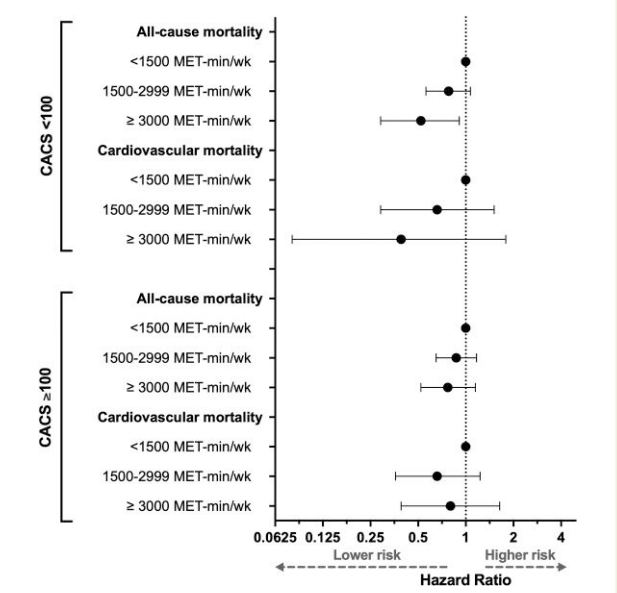


Figure 4 All-cause and cardiovascular mortality risks in male participants of the Cooper Center Longitudinal Study (n = 21 758) with distinct physical activity patterns and stratified for a CACS <100 AU vs ≥100 AU. Data were previously published,[52] presented as hazard ratios with 95% confidence intervals, and indicate that weekly exercise volume does not attenuate event rate in men with CACS ≥100 AU

The clinical work-up of Masters athletes with coronary calcification and/or atherosclerosis should consist of CV risk factor management, delineation of symptoms, and risk stratification (*Table 4*). In the absence of outcomes data in Masters athletes, CV risk factor management should be similar to the general population.[17] Statins are the primary treatment for preventing CV events in athletes with elevated CACS and universal cut-off values for CACS and low-density lipoprotein goals can be used.[65] Data from the multinational CONFIRM registry revealed that individuals with CACS >300 AU face an equivalent risk of major adverse CV events compared to those with a prior diagnosis of myocardial infarction, stroke or peripheral artery disease,[66] and such risks are even higher among those with CACS >1000 AU.[67] Hence, aggressive treatment of CV risk factors should be addressed in athletes with high CACS. Similar to the general population, low-dose aspirin therapy and lipid-lowering therapy are reasonable for Masters athletes with high-risk plaque characteristics.[65]

It is reasonable to utilize risk scores that integrate CACS, such as those provided by MESA[68] or Astro-CHARM,[69] to estimate incident CV events and the potential benefit from medical therapies. However, it should be recognized that available risk scores fail to capture important factors that may contribute to overall risk in Masters athletes including habitual physical activity characteristics (i.e. total exercise dose, rest/recovery patterns and annual periodization), levels of cardiorespiratory fitness, dietary macronutrient intake, and psychosocial stress. Masters athletes with coronary calcification, but no evidence of lipid-containing atherosclerosis (i.e. mixed- or low-attenuation plaques on non-invasive imaging) and no traditional CV risk factors may be seen in clinical practice. Accordingly, routine screening of CACS among low-risk Masters athletes is currently not recommended by either the ESC Sports Cardiology Guidelines or the recent American Heart Association/American College of Cardiology (AHA/ACC) Clinical Considerations for the Care of Competitive Athletes.[17,32] For Masters athletes deemed intermediate or high-risk by traditional CV risk estimates, further clinical risk stratification is reasonable and may include assessment of CACS and plaque phenotype using computed tomographic angiography scanning.

Lifestyle is an important risk modifier for Masters athletes with coronary atherosclerosis as previous studies have shown that higher volumes of exercise and improved cardiorespiratory fitness reduce the risk of adverse health outcomes across all CACS categories compared to less trained/fit individuals.[52,63,64] Based on current evidence, there should be no restrictions to exercise (e.g. intensity, duration, frequency) in asymptomatic athletes with high CACS in the absence of inducible myocardial ischaemia. It is imperative for Masters athletes and their medical providers to appreciate that optimal CV risk reduction is maximized at exercise levels well below the typical training volume of Masters athletes and that no amount of exercise can be considered a substitute for guideline-driven pharmacotherapy.

In asymptomatic Masters athletes deemed to be at intermediate or high risk, including those with the presence of coronary calcification, further risk stratification is reasonable. Options include exercise stress testing to evaluate for silent ischaemia and/or coronary computed tomography angiography. Exercise stress testing defines objective exercise capacity, can provoke exercise-induced arrythmias, and can elicit ischaemic electrocardiographic ST-changes with exercise. These advantages are at least partially offset by the suboptimal sensitivity and specificity of the exercise ECG. Computed tomography angiography enables quantification of the degree, location and morphology of stenoses, but offers no information about functional capacity. In clinical practice these tests are often performed in parallel as they provide synergistic information to inform targeted risk factor management and the role of revascularization with SDM.

Symptoms of coronary artery disease (CAD) in Masters athletes may not be confined to typical angina, but can also present as unexplained declines in exercise performance or the development of exertional dyspnoea. Symptomatic Masters athletes and those with obstructive coronary atherosclerosis should be evaluated for inducible ischaemia in accordance with current guidelines. Exercise, a physiological stimulus, is preferred over pharmacological stimuli for stress testing in athletes.[17]

Multiple studies have demonstrated that acute thrombosis is not solely responsible for sudden cardiac arrest among men with CAD.[70–72] This suggests that stable high-grade CAD may precipitate demand-mediated ischaemic arrhythmia. The ISCHEMIA trial demonstrated that early invasive intervention has no appreciable mortality benefits over medical therapy among individuals with stable obstructive CAD.[73] However, it is important to note that Masters athletes are not comparable to the ISCHEMIA study population, since they may routinely exercise to intensities exceeding ischaemic threshold and are also exposed to additional precipitants of plaque destabilization including dehydration, hypercoagulability and increased coronary flow shear forces. These factors likely increase susceptibility to adverse CV events during exercise. As such and regardless of

Table 4 Clinical consensus statements for Masters athletes with coronary atherosclerosis

#	Consideration	Strength of the advice
1	Masters athletes with established cardiovascular risk factors should receive primary preventive (cardiac) care measures including lifestyle counseling and pharmacological therapy (i.e. anti-hypertensive, lipid-lowering, etc.) for risk factor modification as dictated by general population guidelines.	
2	High cardiorespiratory fitness is associated with improved overall mortality and cardiovascular outcomes. In the absence of signs or symptoms of ischaemia, exercise habits (intensity, duration, and frequency) do not require restrictions or reductions in the presence of traditional cardiovascular risk factors, including the presence of a high coronary artery calcium score.	
3	For asymptomatic Masters athletes with low-risk cardiovascular risk profiles, routine screening with CT-derived coronary artery calcium scoring should not be performed.	
4	For asymptomatic Masters athletes deemed moderate or high-risk, including those with documented coronary calcification, further risk stratification is reasonable. Options including maximal effort-limited exercise stress testing to evaluate for silent ischaemia and/or a coronary CT angiography to evaluate the degree, location, and morphology of stenoses to inform targeted risk factor management, may be appropriate.	
5	Diagnostic ischaemic evaluation with functional testing and/or coronary CT angiography should be performed for Masters athletes with symptoms suggestive of ischaemia.	
6	Masters athletes with confirmed myocardial ischemia should receive individualized counselling on lifestyle modifications and initiation of guideline-based pharmacotherapy.	
7	Revascularization of stable coronary stenoses responsible for symptoms may be appropriate to reduce symptom burden and the potential risk of ischemic ventricular arrhythmias during high-intensity physical exertion.	
8	In the absence of randomized controlled data, revascularization of high-grade stenoses in asymptomatic Masters athletes with evidence of myocardial ischemia on functional or imaging testing may be appropriate given the potential risk of ischemic ventricular arrhythmias during high-intensity physical exertion.	
9	Similar to the general population, low-dose aspirin therapy and lipid-lowering therapy are reasonable for Masters athletes with high-risk plaque characteristics or high CACS.	
10	There is no evidence to support the initiation of prophylactic aspirin therapy prior to competition among Masters athletes without established cardiovascular risk factors.	

the presence of symptoms, for Masters athletes with high-risk lesions (i.e. > 50% left main stenosis, > 50% proximal left anterior descending coronary artery stenosis, tow-three vessel disease with >50% stenosis and >90% stenosis in a vessel), as per ESC[74] and AHA/ACC[32] guidelines, and who intend to engage in vigorous exercise, coronary revascularization can be considered after judicious SDM.[17] This remains a key area for future research.

Aortic dilatation

The impact of vigorous exercise on the aorta remains an area of ongoing research. A 2013 meta-analysis revealed 3.2 mm larger aortic diameters in athletes compared to controls, suggesting a potential effect of exercise exposure on aortic dimensions.[75] Studies in young athletes (≤25 years old) indicate that aortic dilatation, typically defined as ≥40 mm in males and ≥34–38 mm in females, is observed in only ~1–2% of athletes.[76–79] Data among older athletic populations remain limited and report substantial variation in the prevalence of aortic dilatation. For example, mild aortic dilatation (40 to 45 mm) was present in ~20% of Masters runners and rowers,[15] whereas prevalences up to 41% have been reported in former elite strength athletes, including American football[80] and rugby players[81] (*Figure 5*). These findings suggest that sport-specific long-term exercise training, perhaps in combination with traditional risk factors, idiopathic aortic diseases, and/or prohibited substance use may contribute to increased aortic dimensions and potentially associated risk in Masters athletes.

A bicuspid aortic valve (BAV) is found in ~1% of athletes (as in the general population),[83] and general population data suggest that up to 50% will develop aortic enlargement.[84] Risk factors for aortic dilatation include ageing and hypertension,[85,86] and it has been suggested that marked exercise-induced acute and transient increases in systolic blood pressure may be a risk factor

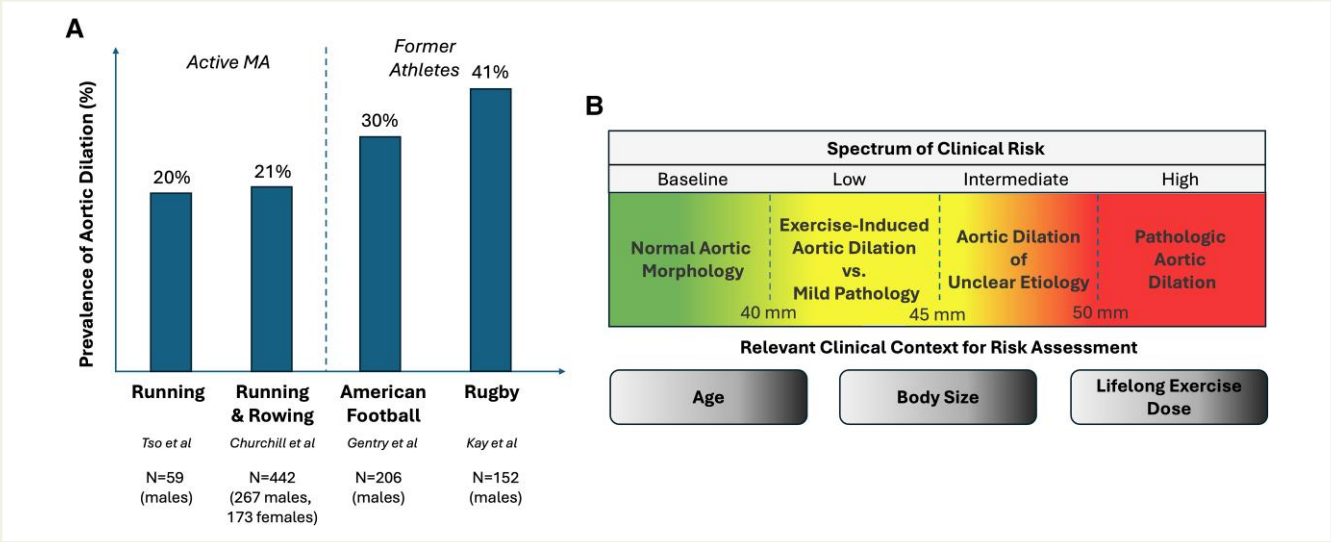


Figure 5 Prevalence of aortic dilatation (≥40 mm) in active Masters Athletes (MA)[15,82] and former elite strength athletes[80,81] (Panel *A*) and the spectrum of clinical risk among athletes with aortic dilatation (Panel *B*)

for aortic dilatation.[82] In contrast, recent studies found no impact of lifelong exercise characteristics on aortic dimensions in patients and athletes with BAV.[87,88]

Data on sport type and aortic dilatation are inconclusive. The marked increases in systolic blood pressure that occur during strength sports and resistance training have generated concerns about aortic complications.[89] In young athletes, however, multiple studies have shown endurance athletes tend to have larger aortic dimensions compared to those in power/strength sports.[75–78] Comparative studies of this sort among Masters athletes are lacking, with the exception of one analysis that found rowing (a mixed sport with power and endurance components) was associated with a higher prevalence of aortic dilatation vs running.[15] Thus, the contribution of sports overall and sport type specifically to aortic dilatation and aortic events in Masters athletes remains unclear and represents an important area of future research.

There are no data defining rates of progression among Masters athletes with aortic dilatation. Similarly, data delineating prognosis, specifically rates of acute aortic syndromes, are extremely limited, precluding any significant conclusion about the exact magnitude of risk. Return to sport after aortic surgery is also an important area of uncertainty. Very limited data (n = 21) suggest safety of return to endurance sport after elective surgery for BAV-associated aortic disease.[90] Finally, the risk of competitive sport participation after aneurysm repair or aortic dissection is uncertain and likely determined by surgical outcome, the presence of residual dissection and underlying disease. Participation decisions in this context should be addressed using SDM.

Masters athletes diagnosed with aortic dilatation (i.e. ≥ 40 mm for both male and female athletes, while taking body size into account) should undergo a comprehensive clinical evaluation, including assessment for features suggestive of heritable or syndromic aetiology,[91] and comprehensive family history. Genetic testing may be appropriate for those at higher risk of heritable disease (*Table 5*).[91] Masters athletes with heritable thoracic aortic disease require careful evaluation and specialist care, which is beyond the scope of this document.[17,91]

All Masters athletes with aortic dilatation should undergo cross-sectional imaging of the entire aorta at least once. Transthoracic echocardiography is advised to assess aortic valve morphology and function. Detailed guidance for imaging in youthful competitive athletes is available, but not specific for Masters athletes.[92,93] A customized imaging surveillance plan based on an individual athlete's diagnosis and aortic size, including side-by-side image comparison by an expert reviewer, should be developed and initiated 6–12 months after index evaluation.

Aortic dimensions relate to body size, and there is ongoing debate about how best to normalize aortic measurements. Various approaches for indexing aortic measurements have been proposed for non-athlete populations,[94–102] but current guidelines rely primarily on the use of unadjusted aortic sizes given their association with outcomes data.[91] The practical advice provided in this document aligns with this framework and does not propose separate aortic size thresholds for male and female Masters athletes, although incorporating body size into the overall risk assessment may be appropriate.

The clinical management of Masters athletes with aortic dilatation should focus on blood pressure management, exercise prescriptions, and surveillance imaging. Resting blood pressure should be measured using proper techniques,[103] and athletes should undergo ambulatory blood pressure monitoring with intermittent home surveillance and/or 24-h ambulatory monitoring. Angiotensin receptor blockers are common first-line agents, although high-quality supportive evidence is lacking in the absence of specific genetic aortopathies. While beta blocker use is common in general population patients with aortic dilatation, they are often poorly tolerated by Masters athletes. At present, there are no data to support treatment of exercise-induced hypertension if resting blood pressure is normal. Finally, with

Table 5 Clinical consensus statements for Masters athletes with aortic dilatation

#	Consideration	Strength of the advice
1	Masters athletes with aortic dilatation (≥40 mm, irrespective of sex but after consideration of body size) should undergo a comprehensive medical evaluation, including a multigenerational family history of aortic disease and physical examination to evaluate for phenotypic traits suggestive of heritable aortic disease.	
2	Masters athletes with aortic enlargement of 45–50 mm on echocardiography should have cross-sectional imaging (computed tomography or magnetic resonance imaging) visualizing the entire aorta performed at least once. A customized imaging surveillance plan should be developed and initiated 6–12 months after index evaluation.	
3	Genetic testing is reasonable for Masters athletes with confirmed aortic enlargement of ≥45 mm.	
4	Masters athletes with aortic dimensions meeting established surgical thresholds (≥50 mm) should be counselled to avoid competitive sports and high-intensity exercise training and be referred for consideration of the timing of surgical intervention.	
5	Masters athletes with aortic dilatation measuring 40–44 mm, without a heritable etiology, are generally considered to be at low risk. Participation in competitive sports and high-intensity exercise training is reasonable, provided that appropriate clinical and imaging surveillance is maintained.	
6	Masters athletes with aortic dilatation of 45–50 mm, without a heritable etiology, are more likely at intermediate risk and require individualized evaluation. SDM should acknowledge the uncertain risks of high-intensity resistance and endurance training or competition on progression of aortic enlargement and acute aortic syndromes.	
7	Masters athletes with a history of surgical repair following aortic dissection should be counseled regarding the health benefits of low-to-moderate intensity aerobic exercise.	
8	Data defining risks of resumption of high-intensity athletic training and competition after surgical repair for aortic dissection are limited. Risks may outweigh benefits thereby necessitating SDM for Masters athletes in this context.	
9	It is reasonable for Masters athletes with BAV aortopathy who have undergone surgical aneurysm repair of the aortic root and/or ascending aorta to return to participation in all training and sporting activity with appropriate clinical and imaging surveillance.	
10	Returning to full training and sport participation after aneurysm repair in athletes with a tricuspid valve and no heritable etiology may be appropriate using a SDM model.	

regard to surgical intervention of Masters athletes with aortic dilatation, standard guidelines for surgical referral should be followed.[91,104]

Limited data are available to guide decision-making about sport participation for Masters athletes with aortic dilatation. Therefore, SDM is required with the following considerations.[105] Further advice regarding sport participation for athletes with tricuspid or bicuspid aortic valve and without heritable aetiology is outlined in *Table 5*. Across all risk categories, clinical assessment should consider age, sex, and body size. Masters athletes with maximal aortic dimensions of 40–44 mm are typically considered low-risk, while those who meet established thresholds for surgical intervention are considered high-risk.[91,104] Athletes with aortic aneurysms of 45–50 mm are at intermediate risk and require individualized evaluation. Although outcome data for this intermediate risk population are lacking, intermediate risk Masters athletes should generally be advised to avoid high-intensity resistance activity and counselled that high-intensity endurance training/competition may confer risk of progressive dilatation and incident acute aortic syndromes compared to moderate intensity. Following initial evaluation, a comprehensive longitudinal follow-up plan including serial imaging, blood pressure monitoring, and family screening should be implemented.[91,104]

Myocardial fibrosis

Cardiac magnetic resonance imaging is emerging as a valuable tool in evaluating athletes with suspected cardiac disorders due to advanced tissue characterization, and detection of subtle myocardial abnormalities, including MF following administration of a gadolinium-based contrast agent and post-contrast imaging. MF refers to increased deposition of collagen within the extracellular matrix of the myocardium after injury, and the assessment of LGE may clarify the aetiology (i.e. ischaemic vs non-ischaemic) and severity (% of myocardium affected) of MF. Common examples of

Table 6 Selected studies comparing the prevalence of myocardial fibrosis in older competitive athletes. Studies are divided by those supporting a higher prevalence of myocardial fibrosis in competitive athletes vs those that are non-supportive

Study	Subjects	Sex and Age	Training History	Sport Disciplines	LGE Findings	Mapping Findings
SUPPORTIVE						
Ragab *et al*, 2023[108]	74 athletes 36 controls	75% male, 44 ± 8 years 25% female, 36 ± 7 years age- and sex-matched	10 h/week and ≥1 completed marathon race (self-reported) <3 h/week	100% marathon runners	LGE 11% athletes (63% RV insertion point; 25% non-ischemic; 12% ischemic) vs LGE 0% controls No difference males vs females; *P* = .14	↑ ECV in male runners compared to controls
Farooq *et al*, 2023[109]	50 athletes 26 controls	100% male, 56 (53–64) years age-matched	>10 h/week for >15 years Compete regularly <3 h/week	82% cyclists 18% triathletes	LGE 48% athletes vs LGE 15% controls, *P* = .005 (all non-ischemic)	No difference in ECV in athletes vs controls
Domenech-Ximenos *et al*, 2020[110]	93 athletes 72 controls	53% male, 36 ± 6 years (total cohort) sex-matched, 34 ± 4 years	12 h/week previous 5 years <3 h/week	100% triathletes	LGE 35% athletes vs 5% controls (male, *P* = .001) LGE 41% athletes vs 0% controls (female, *P* = .001) (all hinge point)	↑ECV in athletes with LGE + vs athletes with LGE- (data available in 30%)
Tahir *et al*, 2018[111]	83 athletes 36 controls	65% male, 44 ± 10 years 35% female, 42 ± 10 years Age- and sex-matched	>10 h/week Regular triathlons in the past 3 years <3 h/week	100% triathletes	LGE 17% athletes vs 0% controls (male, *P* = .052) (non-ischemic)	Native T1 significantly lower in triathletes vs controls (male and female) and no ECV difference
Wilson *et al*, 2011[112]	12 veteran endurance 20 veteran controls	100% male, 56 ± 6 years 100% male, 60 ± 5 years	35–52 years not specified	100% endurance	LGE 50% of veteran athletes (17% ischemic, 17% non-ischemic, 66% hinge point) LGE 0% in young and veteran controls	-
Breuckmann *et al*, 2009[113]	102 runners 102 controls	100% male, 57 ± 6 years Age- and sex-matched	completed ≥5 marathons in the past 3 years not specified	100% marathon runners	LGE 12% athletes vs 4% controls (*P* = .077) (42% ischemic 58% non-ischemic)	-

Continued

Table 6 *Continued*

Study	Subjects	Sex and Age	Training History	Sport Disciplines	LGE Findings	Mapping Findings
NON-SUPPORTIVE						
Andresen *et al*, 2024[114]	27 athletes 16 controls	100% male, 41 ± 9 years Age- and sex-matched	>24 MET/week for the past 12 years not specified	marathon, triathlon, cycling (elite/Olympic)	LGE 12% athletes vs 0% controls (P = .14) (non-ischemic)	No difference in ECV between athletes vs controls
Missenard *et al*, 2021[115]	33 athletes 18 controls	94% male, 47 ± 6 years Age- and sex-matched	Minimum 8 h/week in the past 15 years Untrained individuals among military forces	Running, triathlon, cycling	LGE 5% controls (inferior wall) vs 0% athletes (P = .33)	No difference in T1 mapping/ECV between athletes vs controls
Malek *et al*, 2019[116]	30 athletes 10 controls	100% male, 41 ± 7 years Age- and sex-matched	70 km/week running Healthcare personnel	100% ultramarathon runners	LGE 27% athletes vs 10% controls (P = .4) (62.5% RV insertion point, 37.5% non-ischemic)	No difference in ECV between athletes vs controls
Pujadas *et al*, 2018[117]	34 athletes 12 controls	100% male, 48 ± 7 years Age- and sex-matched	>10 years of training marathon time <3 h Untrained individuals	100% runners	LGE 9% athletes vs 0% controls (non-ischemic) no correlation between total training volume and LGE	No difference in T1 mapping/ECV between athletes vs controls
Abdullah *et al*, 2016[118]	21 athletes 71 controls	73% male, >65 years Age- and sex-matched	6–7 exercise sessions/ week, 25 years (number of controls based on training session/week): – 25 with 1 session –23 with 2–3 sessions –23 with 4 + sessions	marathon and triathlon	LGE 0% athletes vs 1.5% controls (hinge-point)	-
Bohm *et al*, 2016[119]	33 athletes of whom 16 former world-class athletes 33 controls	100% male, 30–60 years Age- and sex-matched	≥ 10 h/week ≥10 years ≤3 h/week	Running, triathlon, cycling	LGE 3% athletes vs 0% controls (non-ischemic)	-

Table 7 Clinical consensus statements for Masters athletes with myocardial fibrosis

#	Consideration	Strength of the advice
1	In Masters athletes with myocardial fibrosis identified after CMR with LGE, further clinical investigation(s) should be based on historical features, particularly the presence of arrhythmic symptoms and/or ventricular arrhythmias, presence of underlying cardiovascular risk factors, supportive findings on other diagnostic tests (e.g. 12-lead ECG), and the pattern and extent of LGE.	
2	Clinical treatment(s) for myocardial fibrosis in Masters athletes should be based on the underlying clinical diagnosis and clinical risk stratification to lower risk for sudden cardiac arrest/death with treatment of ventricular arrhythmias and/or reduce symptoms.	
3	Asymptomatic Masters athletes with isolated LGE by CMR at the right ventricular insertion/hinge point(s) do not require further evaluation and/or clinical risk stratification.	
4	Among asymptomatic Masters athletes with incidentally detected LGE detected by CMR in a pattern associated with sudden cardiac arrest/death risk (e.g. isolated non-ischaemic left ventricular LGE with a stria pattern), it is reasonable to proceed with additional clinical risk stratification with maximal effort and sport-specific exercise testing, ambulatory rhythm monitoring (including during training), and serial clinical surveillance to rule out complex ventricular arrhythmias.	
5	The determination of competitive sports and/or intense physical activity for Masters athletes with myocardial fibrosis should incorporate shared decision-making and consider the underlying clinical diagnosis, pattern and extent of LGE, and results of additional clinical risk stratification, particularly the presence of ventricular arrhythmias.	

cardiac injury that precipitate MF include myocardial ischaemia, inflammation, and haemodynamic overload.[106] However, the identification of isolated MF among Masters athletes presents a diagnostic conundrum, as it can be challenging to distinguish between benign adaptations to athletic training and early signs of cardiomyopathy. MF may also constitute a substrate for ventricular arrhythmias due to the interplay between increased catecholamines, haemodynamic stresses during exercise, electrolyte shifts, and heterogeneous conduction in regions of MF.[107]

The reported prevalence of MF as measured by LGE in Masters athletes ranges from 3% to 50% (*Table 6*).[108–119] Primary reasons for this variability include differences in both study design and the athletic populations studied which vary by age, sex (females underrepresented), sport, and prior intensity and duration of training. Additional study limitations include the paucity of subjects, selection bias, unknown risk factors (possible confounders), lack of appropriate controls, self-reported training histories, and unknown effects of performance enhancing and/or illicit drugs. At present, mechanisms linking intense exercise training with acquired MF are speculative but include inflammation, subclinical hypertension, demand-mediated myocardial ischaemia, and chronic ventricular volume overload.[16]

When MF is identified in a Masters athlete, it is important to consider the clinical context in which the CMR study was obtained and the presence of underlying CV risk factors (*Table 7*). All Masters athletes with significant MF should undergo evaluation for common forms cardiomyopathy that are typically associated with MF. The clinical history, pattern and extent of LGE, and pre-test probability of underlying disease inform prognosis,[120] should guide consideration of additional tests and risk stratification. For example, isolated LGE at the right ventricular insertion/hinge point(s) among asymptomatic Masters athletes is common, not associated with adverse outcomes, and should not require additional testing.[121,122] In contrast, more extensive LGE in association with complex ventricular arrhythmias originating from affected myocardium is highly suggestive of significant pathology and should be further evaluated. The prognosis of Masters athletes with MF is dictated by the pattern and extent of LGE and the underlying aetiology.

It is reasonable to recommend temporary sports restriction in a subset of Masters athletes with LGE based on the clinical history, underlying risk factors, data from other diagnostic tests, and the pattern and extent of LGE. Requisite risk stratification testing for Masters athletes with significant MF should include sport-specific, maximal effort exercise testing and extended duration ambulatory rhythm monitoring (including while training) to assess for the presence of clinically significant ventricular arrhythmias. Treatment for Masters athletes with MF, including pharmacotherapeutics and/or defibrillator placement, should be guided by the underlying diagnosis and the presence and severity of symptoms.

Exercise-induced arrhytmogenic cardiomyopathy

Masters athletes occasionally present with ventricular arrhythmias, often with a right ventricular predominance, which can lead to the discovery of marked asymmetric ventricular dilatation during clinical work-up without clear aetiology. This clinical scenario is most often seen in Masters athletes participating in endurance sporting disciplines that have a concomitant isometric component (i.e. cycling, triathlon and rowing).[46] This uncommon clinical phenotype, for which

Table 8 Clinical consensus statements for Masters athletes with unexplained cardiomyopathy

#	Consideration	Strength of the advice
1	Exercise-induced cardiomyopathy, a proposed heart muscle disease attributed to longstanding exercise training with no other apparent etiology, is a diagnosis of exclusion. To date, no formal diagnostic criteria have been established for this condition; however, its proposed features include disproportionate RV dilation, RV systolic dysfunction, and ventricular arrhythmias of RV origin.	
2	Masters athletes with an extensive endurance sports history presenting with unexplained cardiomyopathy, characterized by RV dilatation out of proportion to LV dilatation and ventricular arrhythmias of RV origin, require a comprehensive diagnostic evaluation that includes contrast-enhanced CMR, genetic testing, rhythm monitoring, and exercise testing to exclude other forms of cardiomyopathy.	
3	The role of an invasive diagnostic electrophysiology study in Masters athletes with unexplained cardiomyopathy and symptomatic ventricular arrhythmias is uncertain but may be appropriate to guide risk stratification and identify athletes who may benefit from ablation.	
4	Among Masters athletes with cardiomyopathy of unexplained etiology, shared decision-making is suggested to discuss management decisions on sports participation, prescribed detraining, ongoing evaluation and management of ventricular arrhythmias, risk stratification to evaluate the need for ICD implantation, and the frequency of serial imaging to monitor disease progression.	

reliable prevalence estimates are lacking and whose existence remains debated, is reported in male and female Masters athletes,[123] and has been termed 'exercise-induced arrhythmogenic cardiomyopathy' (ExI-ACM).[46,124–126]

Initial cases of ExI-ACM were thought to represent gene-elusive variants of arrhythmogenic right ventricular cardiomyopathy, as they exhibited a highly similar phenotype but rarely carried pathogenic gene variants.[123,127] The repetitive haemodynamic strain of pressure and volume load during exercise, which disproportionately affects the right ventricle,[46,128] was proposed as a contributing causal mechanism. One moderate-sized study among former elite endurance athletes did not show evidence of adverse remodelling.[119,129] Advances in cardiac genetics have recently demonstrated a polygenic contribution to extreme cardiac remodelling.[130] However, a direct link between a polygenic profile and clinically significant maladaptive remodelling has yet to be established. At present, the relative contributions of training load, genetic predisposition, and other unmeasured confounders (i.e. performance enhancing drugs, undiagnosed hypertension, subclinical myocarditis, etc.) to the development of a pathologic myocardial substrate remain uncertain.[131]

Data defining the prognosis of ExI-ACM are limited. An early case series of 46 high-level endurance athletes with complex ventricular arrhythmias revealed that 18 had a major arrhythmic event, including 9 athletes who died suddenly, over a median follow-up of 2 years.[47] This concerning adverse event rate occurred despite clinician-rendered advice to stop competitive sport and to undergo medical treatment, ablation, and/or implantable cardioverter-defibrillator (ICD) implantation. In this series, outcomes could not be predicted by the presence of symptoms, morphologic imaging data at the time of diagnosis, or the results of non-invasive arrhythmia evaluation. The only predictive factor of adverse events was inducibility of sustained ventricular tachycardia or ventricular fibrillation during invasive electrophysiological testing. This initial case series, accrued from three tertiary care sports cardiology centres, likely identified the most severe phenotypes with the worse prognoses.[47]

Given the ongoing uncertainties, diagnosing ExI-ACM remains challenging and is primarily a diagnosis of exclusion. Before making this determination, all other potential causes of structural cardiomyopathy and electrical disease must be first carefully considered. Key differential diagnoses to exclude are genetic arrhythmogenic cardiomyopathy,[132] non-dilated left ventricular cardiomyopathy, dilated cardiomyopathy, and other arrhythmogenic conditions such as sarcoidosis.[120] When evaluating athletes for suspected ExI-ACM, attention should focus on high-risk structural and functional features that may impact return-to-play decisions, since continued unrestricted exercise can facilitate progression of the pathogenic substrate.[123,133] These features include disproportionate right ventricular enlargement relative to left ventricular size, reduced right ventricular ejection fraction (typically <40% on CMR; values <45% may still be physiological in ~1 in 6 elite young athletes),[130] and impaired right ventricular free wall strain, with values less negative than −20% indicating dysfunction.[134] Right ventricular wall motion abnormalities (akinesia or dyskinesia) are never adaptive in athletes and should always be considered pathological,[135] though they are uncommon in ExI-ACM[136] and more characteristic of familial arrhythmogenic cardiomyopathy, where they represent a major diagnostic criterion.

The management of Masters athletes with suspected ExI-ACM is summarized in *Table 8* in alignment with the general ESC guidelines for cardiomyopathies.[120] Several observational studies suggest that exercise detraining may contribute to the partial reversal of cardiac structural changes and a reduction of ventricular arrhythmias.[25] Prescribed detraining for athletes with suspected ExI-ACM is a reasonable approach for those without sustained or life-threatening arrhythmias and should be coupled with serial re-evaluation to guide risk stratification and clinical management decisions.[25] Management of arrhythmias, whether by medication, ablation or ICD implantation

should conform to the latest evidence and recommendations on ventricular arrhythmias and the prevention of sudden cardiac death in patients with cardiomyopathies.[25,120,137] In athletes with an initial indication for ICD implantation, reassessment after a detraining period may be appropriate. The wearable cardioverter-defibrillator could be proposed as an interim solution in those judged to be at high risk, during exercise restriction.[137,138] Epicardial ablation may be effective in athletes with documented ventricular tachycardia and isolated subepicardial right ventricular outflow tract scar.[125] Finally, athletes with suspected ExI-ACM who develop right ventricular failure and/or secondary tricuspid regurgitation,[139] should be managed in accordance with contemporary heart failure guidelines.[140]

Future perspectives and research directions

Over the past two decades, evidence on CV abnormalities in Masters athletes has evolved, yet significant uncertainties persist. Best practices for managing those with CV risk or CVD lack strong evidence and rely on expert consensus and extrapolation from the general population—despite key differences. First, the 'exercise paradox', referring to increased acute cardiac risk during exercise even in highly fit individuals, remains imprecisely quantified in Masters athletes.[27] Additionally, the risk-benefit balance between CV risk factors such as coronary artery calcification and the protective effects of high cardiorespiratory fitness is unclear.[63,141] Finally, the mechanisms underlying potentially maladaptive cardiac phenotypes in Masters athletes remain incompletely understood.[9,50,142]

Addressing these uncertainties will require rigorously controlled, longitudinal studies. Future Masters athlete registries should prioritize diverse populations with balanced male and female representation and initiate outcomes tracking as early as possible in life. Comprehensive phenotyping, inclusive of serial clinical assessments, 12-lead ECG, advanced imaging, and laboratory assessments data, is essential. Accurate prospective and objective measurement of exercise dose (duration, intensity, and frequency) and sporting discipline should replace reliance on retrospective recall. In addition, factors unique to this population, such as dietary intake (i.e. nutritional supplements/diet during high-training volume periods and competition), and psychosocial stress should be examined alongside traditional CV risk assessments. Future studies should also take the use of performance enhancing and/or illicit drug use into account, as this is often unreported despite its known deleterious CV effects.[143] Notably, no studies have explored social determinants of health or potential racial disparities in health outcomes among Masters athletes, whereas sex-based disparities in the incidence of disease among Masters athletes remain understudied and incompletely understood, highlighting a critical gap for future research.[144] Finally, advancements in artificial intelligence and computational cardiology may further inform this work.

Longitudinal registry data are required to better understand the potentially maladaptive clinical findings in Masters athletes. Differentiation of stable coronary calcifications vs unstable mixed-morphology atherosclerotic plaque, including their underlying mechanisms and clinical outcomes, will improve clinical management. Similarly, controlled registry data will help clarify causal mechanisms and distinguish pathological from physiological adaptations in AF, subclinical MF, and aortic dilatation. The possibility of ExI-ACM also warrants further investigation. While credible prevalence estimates of this proposed pathology are lacking, it appears to be a rare condition affecting a small subset of Masters athletes. Identifying factors that determine host susceptibility, including but not limited to genetic influences, is needed. Emerging evidence suggests that up to ~50% of gene-negative arrhythmogenic cardiomyopathy cases may harbor previously unrecognized genetic mutations,[145] suggesting an important but still poorly understood genetic component in this pathologic phenotype.

To date, no clinical trials or randomized controlled trials have focused exclusively on athletes, including Masters athletes. Accordingly, expert consensus opinion, based largely on observational data and clinical experience, drive current practice patterns and may deviate from general population guidelines. For example, consideration of coronary revascularization has been recommended for Masters athletes with stable obstructive CAD in ESC Sports Cardiology guidelines and AHA/ACC scientific statements.[17,32] In addition, sinus rhythm maintenance for Masters athletes with AF, which increasingly involves first-line PVI, remains complex and incompletely understood. Key gaps include understanding the effects of detraining on AF burden, the risk of AF recurrence with resumed high-intensity training post-PVI, the long-term impact of PVI on exercise capacity, and the efficacy of emerging ablation techniques (e.g. pulsed field ablation) in highly active individuals. While conducting randomized controlled trials in Masters athletes presents challenges, such studies are essential to establishing data-driven best practices for this unique population facing the challenges of CVD.

Conclusions

The global population of Masters athletes continues to rise. While Masters athletes benefit from high cardiorespiratory fitness and reduced cardiac mortality, they often harbour traditional CV risk factors and increased risk of common forms of CV abnormalities. Despite recent advances in our understanding of CVD among Masters athletes, uncertainties remain. Cardiac care for Masters athletes should include comprehensive traditional CV risk factor assessment, lifestyle counselling, and medical therapy when appropriate (*Graphical Abstract*). Key considerations that should guide therapeutic interventions include symptom burden and their impact on quality of life, the Masters athletes' preferences and goals, and disease-specific prognosis on a case-by-case basis. SDM represents an essential approach in determining treatment strategies and exercise prescription. Future research is warranted to more clearly delineate the benefits and potential risks of high-dose exercise in Masters athletes with and without established CV abnormalities.

Supplementary data

Supplementary data are not available at *European Heart Journal* online.

Declarations

Disclosure of Interest

Nothing to declare.

Data Availability

No data were generated or analysed for or in support of this paper.

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