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Review article

Exercise in cardiomyopathies: updated evaluation, risk stratification, and clinical prescription

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ABSTRACT

Cardiomyopathies comprise a heterogeneous group of myocardial disorders that may increase the risk of potentially life-threatening arrhythmias, particularly during physical activity. Historically, this risk prompted conservative recommendations, often leading to broad restrictions on exercise participation. However, advances in the understanding of pathophysiological mechanisms, coupled with improved tools for phenotypic assessment, risk stratification, and genetic evaluation, have enabled a more nuanced and individualized approach. This narrative review provides an updated overview of the relationship between exercise and cardiomyopathies, emphasizing clinical, functional, and imaging markers associated with elevated arrhythmic risk. It also summarizes key criteria that guide decision-making regarding which patients may safely engage in low-, moderate-, or even high-intensity physical activity. The importance of comprehensive clinical evaluation, shared decision-making, and longitudinal follow-up is highlighted, as these elements allow exercise prescriptions to be tailored to the specific characteristics and evolving risk profile of each patient. Based on current evidence, exercise should no longer be viewed as a universal contraindication; instead, it may serve as a therapeutic adjunct in appropriately selected and well-stratified individuals.

Keywords: Cardiomyopathies; Exercise; Death, Sudden, Cardiac; Risk Assessment (Source: MeSH-NLM).

RESUMEN

Ejercicio en miocardiopatías: evaluación, estratificación del riesgo y prescripción clínica actualizada

Las miocardiopatías constituyen un grupo diverso de enfermedades del músculo cardíaco que pueden aumentar el riesgo de arritmias potencialmente letales, especialmente durante el ejercicio. Históricamente, este riesgo motivó recomendaciones restrictivas, con una tendencia a limitar de forma generalizada la actividad física en estos pacientes. Sin embargo, los avances en la comprensión de los mecanismos fisiopatológicos, la evaluación del fenotipo estructural y eléctrico, y la incorporación de herramientas modernas de estratificación del riesgo y genética han permitido una aproximación más precisa y personalizada. Esta revisión presenta una actualización integral sobre la relación entre ejercicio y miocardiopatías, con énfasis en la identificación de factores clínicos, funcionales e imagenológicos asociados a un mayor riesgo arrítmico. Asimismo, sintetiza los criterios que permiten determinar qué pacientes pueden realizar actividad física de baja, moderada o incluso alta intensidad de manera segura. Se destaca la importancia de una evaluación clínica amplia, la toma de decisiones compartida y el seguimiento longitudinal para adaptar la prescripción de ejercicio a las características individuales de cada persona. Con base en la evidencia más reciente, el ejercicio deja de considerarse una contraindicación universal y se propone como una herramienta terapéutica potencialmente beneficiosa en pacientes adecuadamente seleccionados y estratificados.

Palabras clave: Cardiomiopatías; Ejercicio Físico; Muerte Súbita Cardíaca; Estratificación del Riesgo (Fuente: DeCS-BIREME).

Introduction

Regular physical activity (PA) provides widely recognised cardiovascular health benefits, including improvements in functional capacity, autonomic modulation, and quality of life [1]. However, in people with structural cardiovascular diseases, such as cardiomyopathies, exercise may be associated with an increased risk of ventricular arrhythmias and sudden cardiac death (SCD), particularly during vigorous exertion. This scenario has historically led to restrictive recommendations, many of which have been based more on caution than on robust evidence.

PA is defined as any bodily movement produced by skeletal muscles that results in energy expenditure above resting levels, including occupational, domestic, recreational, or transport-related activities [2]. By contrast, physical exercise is a specific subcategory of PA, characterised by being planned, structured, repetitive, and aimed at improving or maintaining one or more components of physical fitness. This conceptual distinction is particularly relevant in the context of cardiomyopathies, where not all PA entails the same haemodynamic, autonomic, or arrhythmic load, and where prescription should clearly differentiate between daily activity and systematic exercise [2,3].

Over the past decade, advances in cardiac imaging, phenotypic characterisation, genetics, and modern risk-stratification tools have improved understanding of the underlying arrhythmic mechanisms [2,3]. This has supported a more precise approach, in which exercise prescription is tailored to the individual characteristics of the patient and to the dynamic evolution of their risk. In this context, shared decision-making is a fundamental component, encouraging patients to participate actively in understanding their condition and defining a safe level of PA [4].

Despite this progress, many health-care professionals report uncertainty when making exercise recommendations for patients with cardiomyopathies, owing to the clinical heterogeneity of these conditions and the limited availability of evidence-based guidance. The aim of this review is to synthesise current evidence on assessment, risk stratification, and exercise prescription in the main cardiomyopathies, with the aim of providing a practical and updated framework for clinical decision-making.

Clinical assessment and risk stratification

Exercise prescription in patients with cardiomyopathies should be based on a comprehensive clinical assessment that allows training safety to be determined and intensity to be individually tailored. The European Society of Cardiology (ESC) guidelines, both the 2020 version [3] and the 2023 update [4], recommend systematic assessment before starting any PA programme, with the aim of maximising benefits and minimising arrhythmic risk.

Assessment begins with a detailed clinical history, aimed at identifying warning symptoms such as unexplained syncope, chest pain, dyspnoea, palpitations, or a history of cardiac arrest [5]. Comorbidities that limit functional capacity should also be considered, as well as a family history of SCD or premature cardiovascular disease, given their value as indicators of genetic risk.

Quantification of usual PA level is also relevant, as it provides a reference point for adjusting exercise prescription according to diagnosis and individual tolerance [6,7].

Structural and electrical cardiac assessment requires complementary investigations:

- Electrocardiogram (ECG): useful for detecting active arrhythmias or conduction abnormalities [4,5].
- Transthoracic echocardiography: an essential tool for assessing chamber size, ventricular function, and the presence of obstruction or valvular dysfunction [2,4].
- Exercise testing and cardiopulmonary exercise testing: allow identification of exercise-induced arrhythmias, assessment of the haemodynamic response, and determination of physiological variables such as peak VO2 [4].
- Ambulatory ECG monitoring (Holter ≥48 h): increases sensitivity for arrhythmia detection and should include a usual exercise session whenever possible [1,4].
- Cardiac magnetic resonance imaging: provides key information on myocardial structure and tissue characterisation, especially the presence and extent of fibrosis through late gadolinium enhancement (LGE), a marker associated with increased risk of SCD [3,4].
- Coronary CT angiography: considered in specific scenarios, according to recent European and Italian guideline recommendations [4,8].

After integrating clinical and imaging findings, formal risk stratification is performed, classifying patients into low-, moderate-, or high-risk categories. This classification determines the permitted exercise intensity, the appropriate setting for exercise, and the frequency of clinical follow-up.

Guidelines suggest reassessment at least annually. However, the onset of new symptoms, phenotypic changes, or complex arrhythmias justifies earlier assessment, particularly in individuals with high-risk features such as extensive fibrosis, ventricular dysfunction, or a history of arrhythmias **(Table 1)**.

Hypertrophic cardiomyopathy (HCM)

Hypertrophic cardiomyopathy (HCM) is the most prevalent cardiomyopathy and one of the conditions that has historically

Table 1. Decision-making algorithm for exercise prescription in cardiomyopathies.

Dominio	Elementos clave	Utilidad clínica
Clinical history	- Previous cardiac arrest - Unexplained syncope - Chest pain, dyspnoea, or palpitations - Limiting comorbidities - Family history of SCD or premature cardiovascular disease	Initial identification of symptoms or history associated with arrhythmic risk.
Baseline physical activity level	- Usual type of exercise - Intensity and frequency - Sports history	Defines the starting point for prescription and progression of training load.
Electrocardiogram (ECG)	- Active arrhythmias - Conduction or repolarisation abnormalities	Rapid detection of relevant electrical abnormalities.
Transthoracic echocardiography	- Chamber size - Systolic and diastolic function - Outflow tract obstruction	Essential structural assessment for diagnosis and risk stratification.
Exercise testing/ cardiopulmonary exercise testing	- Induced arrhythmias - heart-rate and blood-pressure response - peak VO2 and ventilatory efficiency	Determines functional capacity and arrhythmic risk during exercise.
Ambulatory ECG monitoring (≥48 h)	- Premature ventricular contractions or VT/NSVT - Arrhythmias during usual activity - Assessment during an exercise session	Higher sensitivity for detecting arrhythmias, especially ventricular arrhythmias.
Cardiac magnetic resonance imaging (CMR)	- Fibrosis (LGE) - Detailed morphology and function	Identification of arrhythmic substrates and advanced risk stratification.
Other investigations	- Coronary CT angiography when indicated - Device interrogation	Complement the assessment according to patient characteristics.

HR: heart rate. BP: blood pressure. Peak VO_2: peak oxygen uptake. VT/NSVT: ventricular tachycardia/non-sustained ventricular tachycardia. SCD: sudden cardiac death. LGE: late gadolinium enhancement. CMR: cardiac magnetic resonance imaging. Synthesis based on the 2020-2023 ESC guidelines, 2020 AHA/ACC guidelines, 2024 COCIS consensus, and recent specialised reviews on cardiomyopathies, risk stratification, and sports participation.

raised the greatest concern regarding exercise participation, owing to its association with SCD during exertion. The disease is characterised by left ventricular hypertrophy (LVH) not explained by haemodynamic overload and may or may not be associated with pathogenic variants in sarcomeric genes [8,9]. Its estimated prevalence ranges from 1 in 200 to 1 in 500 people [10], implying that a substantial number of physically active individuals may receive this diagnosis during sports evaluations or routine assessments.

Diagnosis is based on a diastolic wall thickness >15 mm in the absence of secondary causes [4]. However, the exercise-related arrhythmic risk has probably been overestimated. Many individuals diagnosed after years of PA have no symptoms or adverse events [1], prompting a reassessment of traditional recommendations.

HCM has a broad phenotypic spectrum: one-third of patients have left ventricular outflow tract (LVOT) obstruction at rest, another third only with provocation, and the remainder

have non-obstructive disease. Although up to 40% may experience adverse events such as SCD, heart failure, or atrial fibrillation [10], overall annual mortality is less than 1% with appropriate treatment [11]. From an exercise perspective, the key issue is to identify factors that modulate arrhythmic risk during exertion.

The main mechanisms of SCD include myocyte hypertrophy, tissue disarray, fibrosis, and abnormalities of the intramural arteries, which promote ischaemia and ventricular arrhythmias [10]. Factors that increase risk during exercise include unexplained syncope [12], extreme hypertrophy ≥30 mm [13], apical aneurysm [14], and extensive myocardial fibrosis detected as LGE [15]. Fibrosis burden is considered limited when LGE involves less than 15-20% of left ventricular mass. Values above this threshold have been associated with increased arrhythmic risk and should influence restriction of vigorous exercise [2,12,14].

Contemporary evidence has substantially changed the traditionally restrictive view of exercise. Moderate exercise programmes have been shown to improve functional capacity and quality of life without increasing adverse events [16], with possible benefits for mortality [17]. The LIVE-HCM study, which included 1,660 participants, found no differences in cardiac events between those who engaged in vigorous exercise and those who did not [10]. From a physiological standpoint, vigorous exercise is defined as activity performed at 70-85% of heart-rate reserve (HRR), approximately equivalent to 77-95% of estimated maximum heart rate. In metabolic terms, it corresponds to intensities above 6 METs, generally above the second ventilatory threshold, and is associated with a perceived exertion rating ≥15 on the Borg scale [6-20]. This definition allows the level of cardiovascular demand to be objectively assessed and is essential for defining recommendations in patients with cardiomyopathies [2,3,6]. Randomised trials have also shown that high-intensity interventions can improve fitness without increasing risk [18]. Similarly, appropriately evaluated competitive athletes with HCM do not show an increase in complications or phenotypic deterioration during follow-up [19,20].

Risk stratification is essential for defining sports eligibility and exercise prescription. The Italian guidelines [21] establish criteria to identify low-risk patients who may participate in structured PA and, in specialised settings, even competitive sports. Exclusion criteria include a history of cardiac arrest or sustained ventricular tachycardia, exercise-induced symptoms, family history of SCD before the age of 40 years, significant hypertrophy, severe left atrial dilatation, ejection fraction <50%, LVOT gradient ≥30 mmHg, complex arrhythmias, abnormal blood pressure response, LGE >15%, apical aneurysm, and an estimated 5-year SCD risk >4%.

In genotype-positive, phenotype-negative individuals (G+, P–), PA does not appear to accelerate transition to overt disease [22], and in LIVE-HCM, none experienced adverse events [10]. Although post-mortem studies have reported SCD in a small percentage of this group [18], in the absence of high-risk markers, vigorous exercise is permitted with regular follow-up.

Follow-up should be performed annually, or more frequently in young patients, men, Black individuals [23], and those with significant LGE, ventricular aneurysms, or systolic dysfunction. This monitoring is key to adjusting exercise prescription according to clinical evolution.

Resistance training is an essential component of cardiovascular exercise programmes and should be considered in patients with cardiomyopathies under specific safety criteria. Its prescription should follow the FITT principle: frequency, intensity, type, time/volume, and progression. A frequency of 2-3 sessions per week on non-consecutive days is recommended. Intensity should be set at 40-60% of one-repetition maximum (1RM), avoiding loads above 70% 1RM and maximal isometric efforts. Controlled breathing should be emphasised, avoiding the Valsalva manoeuvre because of its potential acute haemodynamic impact [1-3,7,10].

Regarding volume, 1-3 sets of 10-15 repetitions per muscle group are suggested, prioritising dynamic multi-joint exercises. Progression should be gradual, with 5-10% increases in load every 2-4 weeks, according to clinical tolerance and absence of exercise-induced arrhythmias. In patients with higher arrhythmic risk or significant ventricular dysfunction, defined as those with left ventricular ejection fraction <45-50%, documented non-sustained or sustained ventricular tachycardia, unexplained syncope of probable arrhythmic origin, high premature ventricular contraction burden (>500-1000/24 h), extensive myocardial fibrosis on cardiac magnetic resonance imaging (LGE ≥15-20%), abnormal blood pressure response to exercise, or carriers of mutations associated with aggressive arrhythmic phenotypes, such as LMNA, FLNC, DSP, or RBM20, resistance training should be kept at the lower intensity range, approximately 30-40% of 1RM, with 1-2 sets of 12-15 repetitions. Training to muscular failure should be avoided, controlled movements should be prioritised, and periodic clinical monitoring should be undertaken [2,3,8,14]. In these cases, the goal is not to develop maximal strength, but to maintain functional and metabolic capacity while minimising acute haemodynamic stress and excessive sympathetic activation; training should be performed under specialised supervision **(Table 2)**.

Dilated cardiomyopathy (DCM)

Dilated cardiomyopathy (DCM) is one of the cardiomyopathies with the greatest clinical relevance in relation to exercise participation, owing to variability in its presentation, its arrhythmic potential, and the heterogeneity of its response to training. It is defined by the presence of left ventricular or biventricular dilatation, or systolic dysfunction in the absence of haemodynamic overload or significant coronary artery disease, although this definition may be limited because some

patients present with hypokinesia without overt dilatation, which may represent an early stage of the disease (24). Therefore, assessment should integrate both morphological and functional parameters, particularly when determining exercise safety.

The causes of DCM include genetic and acquired aetiologies. The latter include viral myocarditis, chronic alcohol or cocaine use, cardiotoxicity from anthracyclines or mediastinal radiotherapy, endocrinopathies such as hypothyroidism or uncontrolled diabetes, peripartum cardiomyopathy, tachycardia-induced cardiomyopathy, and autoimmune diseases such as lupus or sarcoidosis [24]. In addition, 5-15% of secondary cases harbour pathogenic mutations, reinforcing the interaction between genetic predisposition and environmental factors. Diagnostic assessment should be multidimensional, integrating a focused clinical history, echocardiography, cardiac magnetic resonance imaging with LGE to document fibrosis, genetic testing in familial or idiopathic cases, and functional assessment through exercise testing or cardiopulmonary exercise testing [24]. This process is essential for arrhythmic risk stratification and for guiding exercise prescription.

In athletes, distinguishing DCM from physiological remodelling of the "athlete's heart" represents a considerable challenge, because both may show left ventricular dilatation. However, the athlete's heart is characterised by sinus bradycardia, normal or supranormal systolic function, absence of symptoms, and reversibility after a period of detraining, features that help differentiate it from true DCM [25]. Exercise echocardiography to assess contractile reserve, cardiac magnetic resonance imaging to exclude fibrosis, genetic testing when familial disease is suspected, and prolonged rhythm monitoring to detect arrhythmias are particularly useful for this purpose [25]. This distinction is crucial to avoid unnecessary restrictions or, conversely, exposure to vigorous exercise in the presence of true cardiomyopathy.

Physical exercise is an effective therapeutic tool in DCM, with demonstrated positive effects on functional capacity, quality of life, and ventricular function [26]. However, previous recommendations were historically conservative, as DCM was considered an important cause of sport-related SCD [27]. The annual risk of SCD is estimated at 1-2%, with higher values among carriers of mutations associated with aggressive arrhythmic phenotypes, such as variants in LMNA, RBM20, or FLNC [28]. This underscores the need for precise risk stratification before prescribing exercise.

High-risk factors include a history of syncope, family history of SCD before 40 years of age, the presence of atrial fibrillation, paroxysmal supraventricular tachycardia or polymorphic premature ventricular contractions, non-sustained ventricular tachycardia (≥3 beats at >120 bpm), ejection fraction <45%, segmental akinesia or dyskinesia, an abnormal exercise response with insufficient increase in LVEF (<15%) or systolic blood pressure increase <20 mmHg,

Table 2. Functional and structural risk stratification in hypertrophic cardiomyopathy

Variable	Low risk	Intermediate risk	High risk
Symptoms and medical history	Asymptomatic	Symptoms attributable to HCM without a clear relationship to exercise	History of cardiac arrest, unexplained syncope, or exercise-induced symptoms
ESC 5-year risk score	<4%	4–6%	>6%
Left ventricular outflow tract (LVOT) gradient	≤30 mmHg at rest or during exercise	30-49 mmHg at rest or during exercise	≥50 mmHg at rest or during exercise
Blood-pressure response to exercise	Normal	Attenuated increase in SBP (<20 mmHg)	Fall in blood pressure
Exercise-induced arrhythmias	No arrhythmias	Premature ventricular contractions (PVCs) during exercise	Non-sustained ventricular tachycardia (NSVT) or exercise-induced ventricular tachycardia
Left ventricular function (LVEF)	≥55%	50–55%	<50%
Cardiac magnetic resonance imaging (CMR)	No late gadolinium enhancement (LGE) or left ventricular aneurysm	LGE <20%	LGE ≥20% or presence of left ventricular aneurysm

HCM: hypertrophic cardiomyopathy. LV: left ventricle. LVOT: left ventricular outflow tract. BP: blood pressure. SBP: systolic blood pressure. PVCs: premature ventricular contractions. VT/NSVT: ventricular tachycardia/non-sustained ventricular tachycardia. LVEF: left ventricular ejection fraction. CMR: cardiac magnetic resonance imaging. LGE: late gadolinium enhancement.
The cut-off points used, 5-year risk, percentage of LGE, outflow tract gradient, blood-pressure response, and LVEF, derive from validated HCM risk-stratification models and should be interpreted in an integrated manner within the clinical context.
Information adapted from the 2020 ESC guidelines, 2020 AHA/ACC guidelines, 2024 COCIS consensus, and recent studies on arrhythmic risk stratification in HCM.

extensive fibrosis on cardiac magnetic resonance imaging, and high-risk pathogenic mutations such as LMNA, SCN5A, PLN, TMEM43, FLNC, RBM20, or DSP [2]. In this context, structural, electrical, functional, and genetic assessments become essential to determine whether a patient can engage in vigorous or competitive exercise.

In genotype-positive, phenotype-negative individuals (G+, P–), vigorous exercise or competitive sport is generally considered safe, except in mutations associated with high arrhythmic risk, especially LMNA or FLNC, in whom high-intensity activity should be avoided [1,3]. For most patients with DCM, low-to-moderate intensity exercise is recommended, provided that there are no limiting symptoms or exercise-induced arrhythmias [1,3]. In asymptomatic individuals with stable ventricular function and no high-risk markers, vigorous activity or participation in competitive sports may even be allowed in specialised centres [2].

Follow-up should be performed at least once a year, with earlier reassessment if any symptomatic or phenotypic change occurs. Patients with high-risk factors require closer monitoring to adjust exercise prescription appropriately and minimise the risk of arrhythmic events [1] **(Table 3)**.

Arrhythmogenic cardiomyopathy (ACM)

Arrhythmogenic cardiomyopathy (ACM) is an inherited disease characterised by abnormalities in desmosomal proteins that compromise adhesion between myocytes, promoting progressive cell loss and replacement by fibrous and adipose tissue [29,30]. These abnormalities create an electrically unstable substrate that facilitates the development of re-entrant ventricular tachycardias [31,32] and, to a lesser extent, deterioration of ventricular function. Although initially described as a predominantly right ventricular disease, contemporary evidence shows that biventricular involvement is frequent and that, in some cases, the left ventricle may be the predominantly affected chamber [29,30]. Clinical presentation ranges from subclinical phenotypes to malignant

Table 3. Risk stratification in dilated cardiomyopathy.

Variable	Bajo riesgo	Riesgo intermedio	Alto riesgo
Symptoms and clinical history	Asymptomatic	Mild symptoms without a clear relationship to exercise	Syncope, presyncope, or exercise-induced symptoms
Family history of SCD	No history	Uncertain or unrelated history	SCD in a first-degree relative aged <40 years
Arrhythmias (Holter/exercise)	No significant arrhythmias	Frequent PVCs or PSVT	Non-sustained VT (≥3 beats at >120 bpm) or polymorphic ventricular arrhythmias
Left ventricular function (LVEF)	≥50%	45–49%	<45%
Segmental LV wall motion	Normal	Mild hypokinesia	Segmental akinesia or dyskinesia
Exercise response (echocardiography or cardiopulmonary exercise testing)	Increase in LVEF ≥15% and normal BP response	Borderline increase in LVEF (10–15%) or attenuated BP response	LVEF improvement <15% or SBP increase <20 mmHg during exercise
Fibrosis on cardiac MRI (LGE)	No LGE	Limited LGE	Extensive or diffuse LGE
Genetics	No high-risk mutations	Variants of uncertain significance	Pathogenic mutations in LMNA, SCN5A, PLN, TMEM43, FLNC, RBM20, or DSP, associated with high arrhythmic risk
Phenotypic status	Stable, with no progression	Partial stability or mild changes	Progressive phenotype or documented deterioration

LVEF: left ventricular ejection fraction. LV: left ventricle. SCD: sudden cardiac death. PVCs: premature ventricular contractions. PSVT: paroxysmal supraventricular tachycardia. VT: ventricular tachycardia. BP: blood pressure. SBP: systolic blood pressure. MRI: magnetic resonance imaging. LGE: late gadolinium enhancement. Risk categories are based on criteria used for arrhythmic and prognostic risk stratification in cardiomyopathies, integrating clinical, electrical, functional, structural, and genetic parameters. The thresholds mentioned, LVEF, phenotypic progression, exercise response, extent of LGE, and pathogenic variants in high-risk genes such as LMNA, SCN5A, PLN, TMEM43, FLNC, RBM20, and DSP, derive from international consensus statements and recent evidence.
Adapted from the 2022-2023 ESC guidelines, 2020 AHA/ACC guidelines, and contemporary literature on risk assessment in cardiomyopathies.

arrhythmic episodes, especially during so-called "hot phases", characterised by acute inflammation and myocyte necrosis, with features resembling myocarditis [33].

Exercise plays a major role in the clinical course of the disease. Several studies have shown that high-intensity PA accelerates phenotypic progression and increases the risk of ventricular arrhythmias, even in carriers of pathogenic mutations without evident clinical manifestations. In a cohort of athletes with SCD, 13% had arrhythmogenic right ventricular cardiomyopathy, and left ventricular fibrosis emerged as the strongest predictor of sudden death during exercise [34]. Reduction in exercise intensity has been associated with a significant decrease in arrhythmias and mortality [1]. In athletes with an implantable cardioverter-defibrillator, this condition was the only predictor of appropriate shocks during training or competition, reinforcing the link between vigorous exercise and electrical destabilisation [35].

Risk stratification requires a comprehensive assessment that considers structural, electrical, and genetic findings. Diagnosis is based on criteria that include morphofunctional abnormalities, tissue characterisation by cardiac magnetic resonance imaging or biopsy, documented ventricular arrhythmias, family history, and pathogenic genetic variants. The Padua criteria refined the 2010 Task Force criteria by incorporating specific parameters for left ventricular involvement [4], allowing previously underestimated phenotypes to be recognised. For the prediction of sustained ventricular arrhythmias, a specific externally validated calculator for predominantly right ventricular ACM is available and has shown greater predictive capacity than algorithms traditionally used to guide implantable cardioverter-defibrillator indication [36], including in athletic populations.

Because of the negative effect of vigorous exercise on arrhythmic burden and phenotypic progression, current recommendations are particularly restrictive. Although some individuals without a history of cardiac arrest, unexplained syncope, complex ventricular arrhythmias, or significant structural changes may participate in recreational low-to-moderate intensity exercise, competitive sport and high-intensity exercise are not recommended in any scenario, regardless of genotype or phenotype [2,37]. This recommendation also applies to carriers of pathogenic mutations without clinical expression, especially high-risk variants such as DSC2, DSG2, PKP2, desmoplakin, or TMEM43, given that arrhythmic events may precede the development of structural abnormalities [1].

In phenotypes with predominant left ventricular involvement, available evidence also shows a high risk of arrhythmic events associated with competitive sport, although data on structural progression are limited. Even so, there is expert consensus to apply equally restrictive recommendations to all phenotypic variants of the disease because of its unpredictable behaviour [38]. Follow-up should be annual, with immediate reassessment if symptoms develop, arrhythmias appear, or evidence of phenotypic progression emerges. Individuals with high-risk factors, including young carriers of high-impact mutations, require closer surveillance and careful adjustments to PA [1].

Clinical exercise prescription in cardiomyopathies

Exercise prescription in patients with cardiomyopathies should be based on a complete clinical assessment, precise identification of risk markers, and longitudinal follow-up that allows the intensity and volume of PA to be adjusted according to each patient's clinical course [38,39]. Although each condition has specific pathophysiological features, common principles can guide safe and effective prescription, as well as critical differences that determine the level of restriction or permissiveness regarding vigorous exercise and competitive sport [40].

In HCM, contemporary evidence suggests that many patients considered to be at low risk can participate in low-to-moderate intensity exercise without a significant increase in adverse events, and may even derive functional and psychological benefits [41,42]. Participation in vigorous exercise or competitive sport may be considered in asymptomatic individuals with adequate functional capacity, no exercise-induced arrhythmias, preserved ejection fraction, no significant left ventricular outflow tract obstruction, and limited fibrosis burden, provided that assessment is performed in specialised centres and within a shared decision-making framework. By contrast, the presence of extreme hypertrophy, unexplained syncope, complex ventricular arrhythmias, significant obstructive gradient, defined as a left ventricular outflow tract gradient of ≥30 mmHg at rest or ≥50 mmHg with provocation or during exercise, apical aneurysm, or extensive LGE should lead to restriction of vigorous exercise and prescription based on low-intensity activities. These activities include comfortable walking, stationary cycling below 50% of heart-rate reserve, gentle recreational swimming, joint mobility exercises, dynamic yoga without the Valsalva manoeuvre, and tai chi. Such activities are usually below 3 METs and below the first ventilatory threshold [1,3].

In DCM, exercise represents a therapeutic intervention with positive effects on functional capacity and ventricular remodelling, and is indicated in most patients. The recommended intensity depends on clinical stability, ejection fraction, presence of ventricular arrhythmias, haemodynamic response to exercise, and fibrosis burden on cardiac magnetic resonance imaging [44,45]. In individuals with preserved ventricular function, no symptoms, and no exercise-induced arrhythmias, gradual progression towards moderate and even

vigorous intensities may be allowed in controlled settings. By contrast, the presence of reduced ejection fraction (<45%), non-sustained ventricular tachycardia, abnormal blood pressure response, defined as an increase in systolic blood pressure of less than 20 mmHg from baseline, or a fall in blood pressure during exertion, which may reflect advanced ventricular dysfunction or inability to adequately increase cardiac output, extensive fibrosis, or pathogenic mutations associated with high risk should guide prescription towards a structured low-to-moderate intensity programme, avoiding maximal efforts and restricting participation in competitive sport [45,46]. In genotype-positive, phenotype-negative individuals, exercise intensity should be individualised according to the gene involved. In the absence of mutations associated with high arrhythmic risk, moderate-intensity exercise may be permitted (55-70% of heart-rate reserve). However, in carriers of pathogenic variants associated with aggressive arrhythmic phenotypes, such as LMNA, FLNC, DSP, or RBM20, intensity should be limited to 40-60% of heart-rate reserve and vigorous or competitive exercise should be avoided, even in the absence of overt structural dysfunction [43,45,46].

ACM represents the most restrictive scenario in terms of exercise. There is strong evidence that high-intensity PA accelerates phenotypic progression, increases electrical instability, and raises the risk of sudden death, even in mutation carriers without evident phenotypic expression. Therefore, PA should be limited to low or moderate levels, while vigorous exercise should be avoided and participation in competitive sport prohibited, regardless of phenotypic or genotypic status [47]. In patients without high-risk markers, without exercise-induced ventricular arrhythmias, without significant fibrosis, and with documented electrical stability on prolonged monitoring, recreational low-to-moderate intensity exercise may be considered. Recreational low-to-moderate intensity exercise refers to structured PA performed for health or recreational purposes, without competition, in which cardiovascular load remains within well-defined submaximal ranges. From a physiological perspective, low intensity corresponds to values below 40-50% of heart-rate reserve, approximately equivalent to <60% of estimated maximum heart rate, with an energy expenditure <3 METs and a rating of perceived exertion of 9-11 on the Borg scale [3,5]. Moderate intensity corresponds to 50-70% of heart-rate reserve, approximately 60-75% of maximum heart rate, with a metabolic range of 3-6 METs and a perceived exertion rating of 12-14 on the Borg scale [2,3]. In all scenarios, the presence of complex ventricular arrhythmias, unexplained syncope, more than 500 premature ventricular contractions in 24 hours, extensive LGE, or high-risk mutations requires restriction of exercise intensity and the establishment of a supervised plan [48,49] **(Table 4)**.

Exercise prescription in cardiomyopathies should therefore be grounded in individualised assessment, identification of the patient's risk stratum, and clinical stability **(Figure 1)**. Shared decision-making is indispensable to balance risks and benefits, especially in scenarios where evidence is emerging but not definitive. Regular clinical follow-up, ideally annually or more frequently in individuals with higher-risk markers, allows exercise intensity to be adjusted dynamically and timely interventions to be implemented in response to

Table 4. Exercise prescription intensities according to intensity level and type of cardiomyopathy.

Cardiomyopathy	Low intensity	Moderate intensity	Vigorous intensity
HCM	Permitted; 40–55% of heart-rate reserve; <3 METs; RPE 9–11; below VT1	Permitted only in low-risk patients; 55–70% of heart-rate reserve; 3–6 METs; RPE 12–14; between VT1 and approximately 10% below VT2	Only in patients classified as low risk and under supervision; 70–85% of heart-rate reserve; >6 METs; RPE ≥15; maximal efforts should be avoided
DCM	Permitted; 40–55% of heart-rate reserve; 2–3 METs; RPE 9–11; near or below VT1	Permitted; 55–75% of heart-rate reserve; 3–7 METs; RPE 12–15; between VT1 and VT2	Only in selected stable patients; 75–85% of heart-rate reserve; >6 METs; RPE ≥15; maximal efforts should be avoided
ACM	The only recommended intensity; <40–50% of heart-rate reserve; <3 METs; RPE 9–11; always below VT1; volume <15 MET-h/week	Highly restricted; 50–55% of heart-rate reserve; 3–4 METs; RPE 11–12; should not approach VT2	Prohibited; >70% of heart-rate reserve; >6 METs; RPE ≥15; any exercise close to VT2; competitive sport prohibited

HCM: hypertrophic cardiomyopathy. DCM: dilated cardiomyopathy. ACM: arrhythmogenic cardiomyopathy. Heart-rate reserve: HRR. METs: metabolic equivalents. RPE: rating of perceived exertion on the Borg scale. VT1/VT2: ventilatory thresholds 1 and 2.
Intensities are presented according to standardised physiological parameters, heart-rate reserve, METs, RPE, and relation to ventilatory thresholds, aligned with recommendations for patients with cardiomyopathies. Restrictions for ACM derive from the high arrhythmic risk associated with vigorous exercise.
Based on the 2020-2023 ESC guidelines, 2020 AHA/ACC guidelines, 2024 COCIS consensus, and recent reviews on exercise prescription in cardiomyopathies.

changes in phenotype, arrhythmic burden, or ventricular function.

Future research

Evidence on exercise in cardiomyopathies has advanced substantially over the past decade; however, important gaps remain and require systematic investigation. One priority is the development of prospective and randomised studies with adequate sample sizes to more precisely establish the effect of vigorous exercise in specific subgroups, particularly patients with hypertrophic and dilated cardiomyopathy in intermediate-risk strata. Cohorts assessing the long-term safety of exercise in genotype-positive, phenotype-negative individuals are also needed, as this group represents a growing number of people identified through family screening and expanded genetic testing.

In relation to ACM, future studies should clarify the exact threshold of exercise intensity and volume capable of accelerating phenotypic progression and increasing arrhythmic burden, as well as explore interventions that might mitigate these adverse effects. Integration of molecular biomarkers, advanced tissue characterisation using cardiac magnetic resonance imaging, and continuous arrhythmia monitoring with wearable devices could improve the early detection of relevant exercise-induced changes.

Another emerging field is the personalisation of exercise prescription through artificial intelligence tools and combined predictive models integrating genetic, structural, electrical, and functional information. Further research is also needed on supervised exercise programmes, telemedicine, remote monitoring, and adherence, particularly in settings with limited availability of specialised centres.

Finally, the development of guidelines based on robust evidence, beyond expert consensus, will depend on the methodological quality of future multicentre studies and the inclusion of diverse populations, including women, recreational athletes, and under-represented ethnic groups. These studies will support progress towards safer, more precise, and more personalised exercise prescription across the different cardiomyopathies.

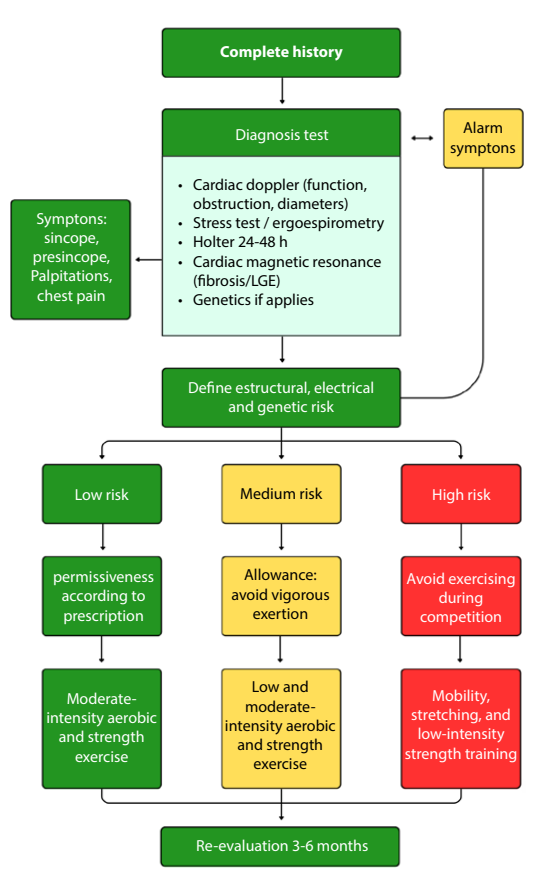

Figure 1. Decision-making algorithm for exercise prescription in cardiomyopathies.

Conclusions

Physical exercise is a valuable tool for many patients with cardiomyopathies, but its prescription requires individualised assessment and precise risk stratification. Recent evidence shows that historically restrictive recommendations are not applicable to all scenarios and that patients with hypertrophic or dilated cardiomyopathy who have been appropriately assessed and have no high-risk markers may benefit from low-to-moderate intensity PA and, in selected cases, vigorous exercise under specialised supervision. By contrast, ACM remains the condition most sensitive to intense exercise, justifying more conservative recommendations to prevent phenotypic progression and serious arrhythmic events. Shared decision-making, periodic clinical follow-up, and close monitoring in the presence of phenotypic changes are fundamental pillars to ensure safe participation. Overall, these considerations allow the benefits of exercise to be optimised while minimising risks, supporting progress towards a more precise, personalised, and evidence-based exercise prescription.

Author contributions

AGY: conceptualisation, methodology, analysis and interpretation, writing—original draft, supervision, and final approval of the manuscript. **MMS:** literature search, conceptualisation, methodology, data curation, writing—review and editing, and final approval of the manuscript. **MAS:** literature search, analysis and interpretation, writing—review and editing, visualisation (tables, figures, and diagrams), and final approval of the manuscript.

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