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STATE OF THE ART REVIEW
Heart failure and cardiomyopathies

Hypertrophic cardiomyopathy: changing the paradigm of exercise prescription and competitive sport participation

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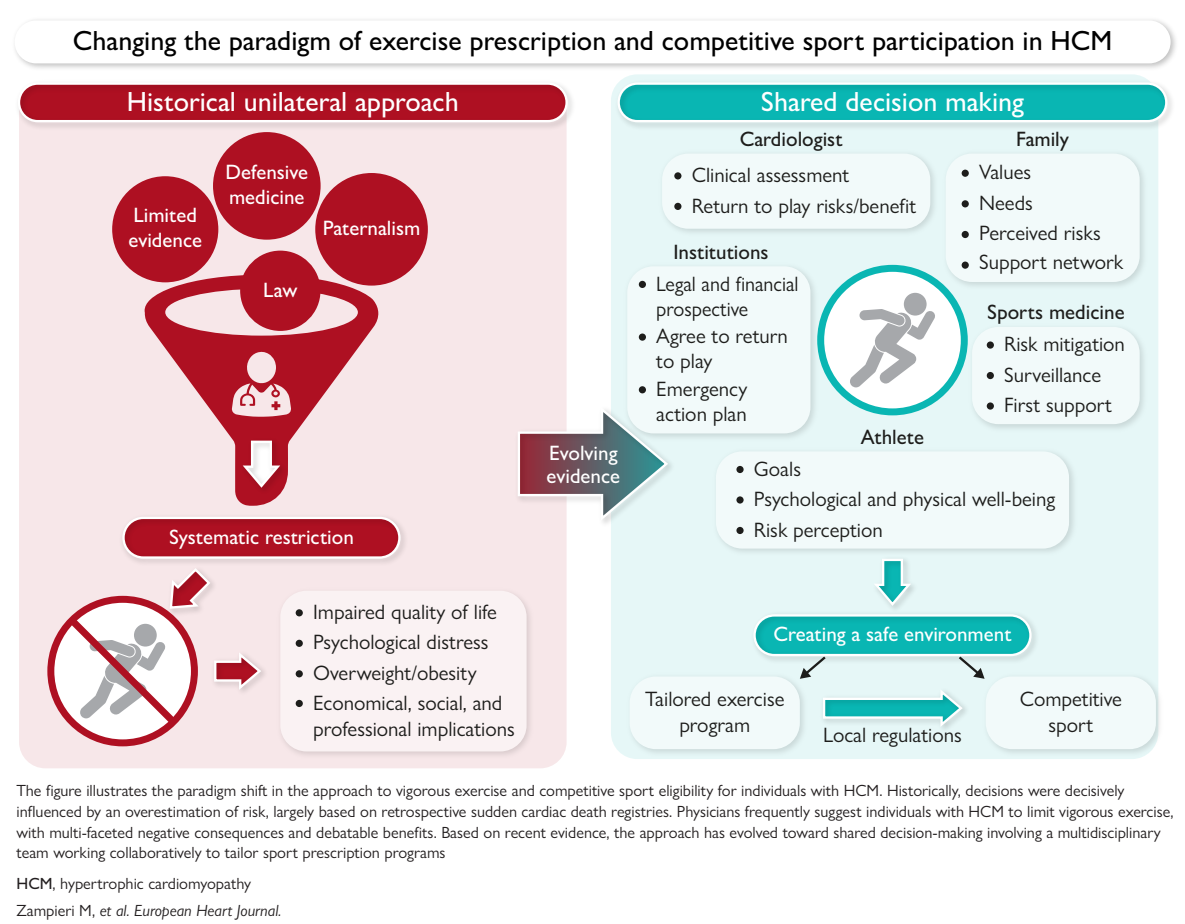
Graphical Abstract



The figure illustrates the paradigm shift in the approach to vigorous exercise and competitive sport eligibility for individuals with HCM. Historically, decisions were decisively influenced by an overestimation of risk, largely based on retrospective sudden cardiac death registries. Physicians frequently suggest individuals with HCM to limit vigorous exercise, with multi-faceted negative consequences and debatable benefits. Based on recent evidence, the approach has evolved toward shared decision-making involving a multidisciplinary team working collaboratively to tailor sport prescription programs

HCM, hypertrophic cardiomyopathy

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The figure illustrates the paradigm shift in the approach to vigorous exercise and competitive sport eligibility for individuals with hypertrophic cardiomyopathy (HCM). Historically, decisions were decisively influenced by an overestimation of risk, largely based on retrospective sudden cardiac death registries. Physicians often adopted a paternalistic approach, routinely restricting individuals with HCM from engaging in vigorous exercise, with multi-faceted negative consequences and debateable benefits. Based on recent, reassuring evidence, the approach has evolved towards shared decision-making, focusing on the individual's goals and well-being, with no signals raising safety concerns. This modern framework, based on individual empowerment, involves a multidisciplinary team working collaboratively to create a safe environment and tailored sports prescriptions programmes.

Abstract

Historically, individuals with hypertrophic cardiomyopathy (HCM) have been systematically excluded from vigorous exercise and competitive sports due to concerns about increased sudden cardiac death (SCD) risk. However, emerging evidence has challenged this paradigm. Observational studies and randomized trials demonstrate improvements in functional capacity, quality of life, and overall cardiovascular and psychological health with tailored exercise in HCM populations —including competitive sports in low-risk individuals—in the absence of safety signals. Low-risk individuals are defined by the lack of well-established adverse features such as a high European Society of Cardiology HCM SCD risk score, exercise-induced syncope, severe left ventricular outflow tract obstruction and ventricular arrhythmias. The concept that this low-risk subset can safely engage in competitive sports is becoming widely accepted internationally, although local differences exist in terms of regulations and clinical approaches. For individuals who do not meet low-risk criteria, evidence is more limited. Nevertheless, participation in competitive sports may still be considered on an individual basis, after appropriate risk mitigation strategies (including treatment of obstruction and ICD implantation when indicated). A contemporary approach to HCM should be based on individualized, goal-oriented exercise prescription, supported by multidisciplinary counselling and shared decision-making, recognizing the importance of aligning medical recommendations with individual safety, values, goals, and quality of life. In this review, we critically appraise the emerging evidence surrounding vigorous exercise and competitive sports in HCM and explore how novel data are reshaping clinical practice and international guidelines, setting the stage for a paradigm in the field.

Keywords Vigorous exercise • Competitive sport • Hypertrophic cardiomyopathy • Shared decision-making • Prescription

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiomyopathy, characterized by primary myocardial hypertrophy and increased propensity for ventricular arrhythmias, including rare life-threatening events at rest or during exercise.[1] Many individuals with HCM are asymptomatic or mildly symptomatic and maintain an active lifestyle, which they are reluctant to abandon following the diagnosis. However, the eligibility of individuals with HCM to safely engage in vigorous exercise and competitive sports is one of the most controversial and evolving issues in recent years. In the absence of robust data, clinical decisions have long relied on expert judgment and personal perception, leading to negative attitudes and generalized disqualification of HCM athletes. However, accumulating evidence suggests that sports participation, particularly when tailored to the individual profile, does not increase the baseline risk associated with the disease. Conversely, an imposed sedentary style is almost certain to produce harm.

In this review, we critically appraise the emerging evidence surrounding vigorous exercise and competitive sports in HCM and explore how novel data are reshaping clinical practice and international guidelines, setting the stage for a change of paradigm in the field.

Historical perspective

The impact of an HCM diagnosis on athletes' lives has long been a cause of concern for clinicians. As early as 1980, in the first autoptic study to address sudden cardiac death (SCD) in young athletes, BJ Maron reported HCM as the leading associated aetiology.[2] Of the 29 competitive athletes who died suddenly on the athletic pitch, HCM was deemed responsible in 14 (48%) cases.[2] This landmark study and subsequent iterations inextricably linked HCM to increased risk of SCD associated with competitive sports. In a large series of 1866 deaths in competitive athletes studied between 1980 and 2006, of which 1049 were cardiovascular deaths and 82% occurred during training or competition, the most common aetiology was again HCM, accounting for 36%.[3,4] Subsequent retrospective registries on autopsy studies have reinforced the perception of an association between HCM and SCD in young athletes[5] (*Table 1*), and multiple potential mechanisms were postulated.[29–32] Autopsy studies, however, have obvious selection bias, compared with systematic registries with pre-specified protocols, and their *post hoc* design has inevitably skewed findings towards the most severe spectrum of disease. In the case of HCM, the relatively high prevalence in the general population— compared with other causes of SCD in the young — inevitably impacts the absolute number of events compared with other conditions, thus overestimating relative risk both in the athletic and non-athletic setting. Recent studies based on larger autopsy registries, including adolescents and children, have reported a considerably lower proportion of HCM-related deaths, reclassifying HCM as an uncommon cause of SCD.[7–15,33–39]

Nevertheless, the legacy of these early studies remains. The clinical perception of a high risk in the context of vigorous exercise and competitive sports spread rapidly and became deeply entrenched, reinforced by widely publicized, anecdotal cases of elite athletes who died suddenly during high-profile competitions. Many Federations have since implemented screening programmes among professional athletes and the issue of pre-participation screening has dominated the scientific literature for more than two decades. The tenet that all individuals with HCM should avoid vigorous exercise and competitive sports participation was endorsed by scientific societies (*Figure 1*), starting in 1985 with the recommendations from the #16 Bethesda Conference on eligibility for competition,[40] later updated as Bethesda Conferences #26 in 1994[41] and #36 in 2005, which stated that '*Athletes with the unequivocal diagnosis of HCM should not participate in*

Table 1 Overview of studies investigating sudden cardiac death and physical activity in individuals with hypertrophic cardiomyopathy: data from sudden cardiac death registries, retrospective cohorts, prospective studies, and clinical trials

1st Author, Journal, year	Study design and population	Main findings	Key messages	Limitations
Sudden cardiac death registries				
Maron *et al.*, Circulation, 1980[2]	Retrospective autopsy registry and prospective collection of news reports; USA.	Among 29 sudden deaths in athletes, 14 had HCM, one WPW, and one anomalous coronary artery origin.	HCM was the most common cause of SCD in athletes.	Largely retrospective, autopsy-based. Diagnosis of HCM was post-mortem in all but one case. Small sample size.
Van Camp *et al.*, Med Sci Sports Exerc, 1995[5]	Retrospective review from National Center for Catastrophic Sport Injury Research (1983–1993); USA; 136 non-traumatic SCD in high school/college athletes.	HCM found in 51 cases (1 with concomitant WPW, 3 with concomitant coronary anomalies).	HCM and congenital coronary anomalies were the leading causes of SCD.	Autopsy-based, retrospective.
Corrado *et al.*, JAMA, 2006[6]	Observational; 42 386 athletes aged 12–35 screened in Italy over 23 years; comparison with unscreened non-athletes.	Pre-participation ECG-based screening led to 89% reduction in sudden cardiovascular death in athletes; no change in non-athletes. Decline mostly due to fewer deaths from cardiomyopathies.	Systematic ECG screening reduces mortality from cardiomyopathies in young athletes.	Observational. Regional data. Italian setting. Includes all cardiac deaths, not limited to HCM.
Holst *et al.*, Heart Rhythm, 2010[7]	Nationwide retrospective study; Denmark (2000–2006); death certificates of individuals aged 12–35.	15 sport-related SCDs; 1 due to possible HCM. Overall incidence: 1.21/100 000 athlete-years.	Sports-related SCD is rare and lower than in the general population.	Based on death certificates.
Maron *et al.*, JACC, 2014[3]	Mixed retrospective/prospective registry (2002–2011); NCAA and National Registry; USA.	Among 182 athlete SCDs, 21 had HCM. Overall cardiovascular mortality: 1.2/100 000 athlete-years.	Athletes had lower cardiovascular mortality than peers.	Not all deaths were exertional. Incomplete autopsy data. Media-reported and voluntarily submitted cases.
Harmon *et al.*, Circulation, 2015[8]	NCAA death database (2003–2013); USA; ~4.2 million athlete-years.	514 deaths; SCD incidence 1:53,703 athlete-years; 5 HCM cases.	Autopsy-negative findings most common; HCM was rare.	Autopsy quality and examiner expertise influenced cause attribution.
Maron *et al.*, Am J Med, 2016[4]	Prospective and retrospective. National Registry of Sudden Death in Athletes (1980–2011); USA.	2406 athlete deaths; 842 had confirmed cardiovascular causes. Overall trauma was the most common cause of death (488 individuals).	HCM was the leading cardiovascular cause of SCD (302 cases).	Media- and voluntarily reported. 214/2406 had no definitive cause; 464 had incomplete autopsies. Of cardiovascular deaths, 25% occurred at rest or during recreation activity.
Finocchiaro *et al.*, JACC, 2016[9]	Retrospective autopsy study; UK; 3684 SCD cases (1994–2014), 357 athletes.	Myocardial disease found in 40%. HCM in 23 cases, 10 occurred at rest.	Idiopathic LVH/fibrosis and ARVC were most common. HCM was a residual cause.	Retrospective, autopsy-based.
Harmon *et al.*, Mayo Clinic Proceedings, 2016[10]	NCAA deaths; athletes aged 14–18 from 7 US states; 6.97 million athlete-years.	104 SCA/SCD cases; 80% exertional. HCM in 7 cases.	SCA/SCD incidence: 1:44,832 in males, 1:237,510 in females. HCM represent a residual cause of SCD.	Variable autopsy standards; some ambiguous diagnoses.
Malhotra *et al.*, N Engl J Med, 2018[11]	Screening of 11 168 adolescent soccer players; UK; 1996–2016.	23 deaths over 10 ± 8 years. SCD incidence: 1/14 794 person-years. HCM in 3.	Most SCDs were due to cardiomyopathies not detected during screening.	Voluntary SCD reporting. No data on survivors. Elite adolescent athletes only.

Continued

Table 1 *Continued*

1st Author, Journal, year	Study design and population	Main findings	Key messages	Limitations
Morentin *et al.*, Forensic Sci Res, 2019[12]	Retrospective forensic autopsy review; Spain (2010–2017); individuals aged 1–35.	645 SCD cases; 75 sport-related; 24 HCM cases (8 sport-related).	One-third of sport-related SCDs were due to cardiomyopathy, mainly ARVC.	Autopsy-based. Only 5 sport-related deaths had pre-mortem diagnosis.
Weissler *et al.*, Circulation, 2019[13]	Retrospective; Ontario coroner database (2005–2016); ages 10–45.	44 definite, 3 probable, and 6 possible HCM-SCDs. 70% were undiagnosed during life. 64.8% occurred at rest.	HCM-related SCDs in the general population are rare and often non-exertional.	Some SCD cases may lack autopsy. High autopsy rate (94%) in Ontario.
Kwon *et al.*, Br J Sports Med, 2020[14]	Nationwide cohort; Korea; 7666 HCM patients from National Health Insurance Service database (2009–2016).	Higher activity was linked to reduced all-cause and cardiovascular mortality.	Moderate-to-vigorous activity was safe and beneficial in middle-aged HCM individuals.	Mean age ~59. Self-reported activity. No upper intensity threshold defined.
Finocchiaro *et al.*, JACC EP, 2023[15]	Retrospective autopsy study of 6860 SCD cases (1994–2020); UK; focus on ages 10–30.	264 HCM cases (4%); 66 were aged 10–30. Death during exertion in 26; at rest in 40.	Younger individuals (10–15) died mostly during exercise; older (16–30) at rest.	Small sample in younger group. No genetic testing. Most diagnoses post-mortem.
Retrospective cohort studies				
Dejgaard *et al.*, Int J Cardiol, 2018[16]	Cross-sectional; 187 subjects (49 ± 16 years); 121 with HCM, 66 genotype-positive/phenotype-negative; Norway.	Vigorous lifetime exercise increased LV mass within physiological range; no link with pathologic hypertrophy, arrhythmias, or earlier arrhythmic events.	Vigorous exercise was associated with favourable cardiac remodelling without increased arrhythmic risk.	Vigorous exercise was associated with favourable cardiac remodelling without increased arrhythmic risk.
Pelliccia *et al.*, Circulation, 2018[17]	Retrospective; 35 athletes with HCM (32 ± 13 years); 88% low ESC-SCD risk; Italy.	Over 9 years, 1 cardiac arrest occurred outside sport context. No difference in events between those who continued or discontinued sports.	Continuing or discontinuing sports did not significantly affect outcomes.	Small sample. Self-selection for continued exercise.
Pelliccia *et al.*, Br J Spors Med, 2020[18]	Retrospective; 88 HCM athletes (median age 31); 92% male; 98% Caucasian; Italy.	Over 7 years, 2 SCAs occurred in detrained individuals, outside of sport.	Low-risk HCM athletes continuing sport had no increased risk of major cardiac events.	Predominantly low-risk phenotype. Small sample. Self-selection bias.
Basu *et al.*, JACC, 2022[19]	Retrospective; 53 HCM athletes (39 ± 12 years), all low ESC-SCD risk; UK.	No deaths, sustained VT, or syncope over 4 ± 3 years of follow-up.	Low-risk HCM athletes continuing sport had excellent prognosis.	Overall low ESC-SCD risk. Small cohort.
Martinez *et al.*, JACC, 2023[20]	Multicenter retrospective; 76 elite athletes with genetic heart disease, including 40 with HCM; mean age 19 ± 5.	Return to play was associated with low, nonfatal event rates. 1 individual with HCM experienced ICD shock unrelated to sport.	Participation after shared decision-making was safe, even in elite athletes	Small sample. Most returned to sport voluntarily.
Hassanzada *et al.* Circ-Genom Precis Med, 2024[21]	Interview-based retrospective cohort; 188 MYBPC3 truncating variant carriers; median follow-up 8.8 years. Netherlands;	High-dynamic activity (>22 MET-h/week) was linked to increased arrhythmic risk; static activity showed no association.	Exercise was generally safe, but high-dynamic load may elevate arrhythmic risk in MYBPC3 carriers.	Not specific to HCM. Self-reported data. No ESC-SCD risk stratification.
Lee *et al.*, BMJ, 2025[22]	Retrospective; 75 SCD cases from 2619 HCM patients (2005–2023).	20% of SCDs occurred during high-intensity activity; these patients were younger (median 25 years). No links to traditional risk factors.	High-intensity activity-related SCD was associated with younger age but not traditional risk factors.	Retrospective, small sample.

Continued

Table 1 *Continued*

1st Author, Journal, year	Study design and population	Main findings	Key messages	Limitations
Prospective studies and trials				
Klempfner, Eur J Prev Cardiol, 2015[23]	Prospective; 20 HCM patients (62 ± 13 years), 65% NYHA III, enrolled in supervised rehab.	Exercise to 85% heart rate reserve improved exercise capacity and symptoms without major events	Supervised exercise improved function in symptomatic HCM without safety concerns.	Small sample. Adults only. No control group. No gas exchange measurements.
Saberi *et al.*, JAMA, 2017[24] (RESET-HCM)	RCT; 136 HCM patients (50 ± 13 years); 16 weeks moderate-intensity aerobic exercise vs usual activity.	Exercise improved capacity and self-perceived physical functioning. No major events.	Moderate-intensity aerobic exercise is safe and improves function.	Not powered for safety. No vigorous exercise. Adults only. Unblinded.
MacNamara, J Am Heart Assoc, 2023[25] (HIT-HCM)	RCT; 15 HCM patients (47 ± 8 years); moderate vs high-intensity training.	Both improved VO_2 peak. High-intensity increased LVEDV. No serious arrhythmias or adverse events.	Both exercise intensities were safe and improved fitness.	Small sample. Adults only. 1/3 with obstructive HCM. Not powered for safety.
Lampert, JAMA Cardiol., 2023[26] (LIVE-HCM)	Multicenter prospective; 1660 individuals (ages 8–60) with HCM or genotype-positive phenotype-negative; 3-year follow-up.	77 (4.6%) met composite endpoint. Vigorous exercisers had no increased event rates vs non-vigorous.	Vigorous exercise was not associated with increased mortality or arrhythmias.	Observational. Most participants from high-volume HCM centres.
Gudmundsdottir, Circulation, 2024[27]	RCT; 59 HCM patients (58 ± 12 years), NYHA I–III; moderate-intensity exercise vs usual care.	12-week supervised training reduced LV filling pressures and improved performance and quality of life. No major events.	Moderate-intensity exercise improved physiology and quality of life in non-obstructive HCM.	Small sample. Short intervention. Adults only. No vigorous activity. Not powered for safety.
Basu *et al.*, Eur Heart J, 2025 (SAFE-HCM)[28]	RCT; 80 HCM patients (48 ± 8 years); supervised high-intensity exercise vs usual care.	High-intensity exercise improved VO_2 peak, blood pressure, BMI, and psychological scores. No increase in arrhythmias or adverse events.	High-intensity exercise is feasible and effective in HCM patients under supervision.	Small sample. Adults only. Not powered for safety. Improvements mostly attenuated at 6 months.

ARVC, arrhythmogenic right ventricular cardiomyopathy; ESC, European Society of Cardiology; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter defibrillator; LV, eft ventricle; LVEDV, left ventricular end-diastolic volume; LVH, left ventricular hypertrophy; MET-h/week, metabolic equivalent task-hours per week; NCAA, National Collegiate Athletic Association; NYHA, New York Heart Association; RCT, randomized controlled trial; SCA, sudden cardiac arrest; SCD, sudden cardiac death; VO_2; peak, peak oxygen uptake; VT, ventricular tachycardia; WPW, Wolff-Parkinson-White syndrome.

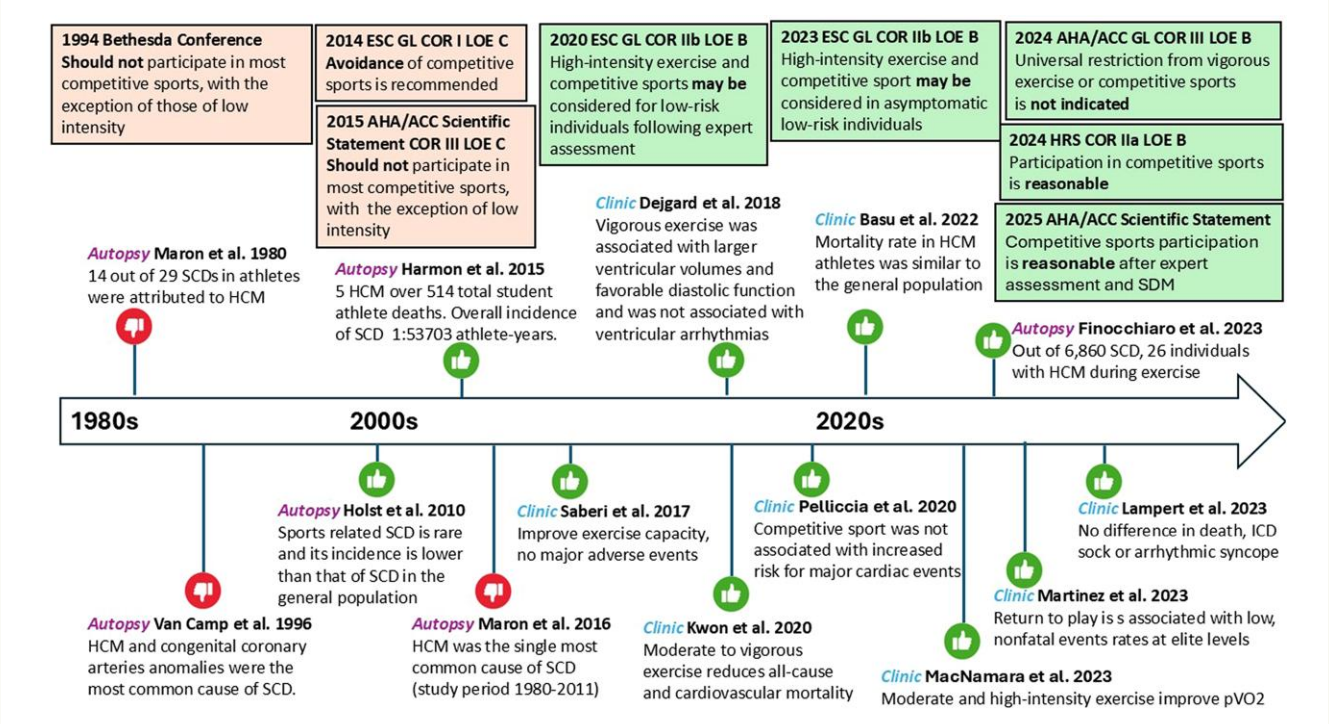

Figure 1 Timeline summarizing the evolution of recommendations and landmark studies on sport participation in individuals with hypertrophic cardiomyopathy (HCM). Red boxes represent key guidelines and statements discouraging vigorous exercise or competitive sport. Green boxes indicate more recent recommendations supporting participation following expert assessment. Red thumbs-down icons denote studies cautioning against sport in HCM. Green thumbs-up icons highlight studies supporting sport participation. Purple labels refer to autopsy-based studies; blue labels to clinical or cohort-based studies. AHA/ACC, American Heart Association/American College of Cardiology; ESC, European Society of Cardiology; COR, class of recommendation; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter defibrillator; LOE, level of evidence; SCD, sudden cardiac death; SDM, shared decision-making

most competitive sports'.[42] This recommendation was echoed by the European Society of Cardiology (ESC) recommendations in 2005.[43] As recently as in 2011, the ACCF/AHA guidelines for the diagnosis and treatment of HCM and the 2014 ESC guidelines on the diagnosis and management of HCM advised against participation in competitive sports and discouraged intense physical activity (*Figure 1*).[44,45] Such negative attitude was further fuelled at that time by a lack of reliable criteria for risk stratification with the inability to define truly 'low-risk' individuals.

Evolving evidence and paradigm shift

Following the widespread access to echocardiography in the 1990s, studies of non-selected HCM populations in the outpatient setting began to uncover a milder face of HCM, showing that the occurrence of SCD in the absence of major risk factors was as rare as 0.6% per year.[46] Advancements in the understanding of HCM clinical history have led to the development of effective, evidence-based risk stratification models for SCD.[1] Additionally, recognition of the essential role of physical activity in psychological and physiological well-being has driven the design and implementation of clinical studies aimed at enhancing the quality of available evidence. These studies, while still not definitive, have shed a consistently favourable light on the outcome for HCM individuals participating in personalized activity programmes, including vigorous exercise (*Table 1*).

Anecdotally, it has long been known that HCM may be compatible with sports. In 1994, BJ Maron described a cohort of 14 individuals (aged 30–66) who were incidentally diagnosed with HCM after participating in endurance sports such as marathons, swimming, and triathlons. Their athletic performance was notable, as 12 competed at a national or professional level, all remained asymptomatic, and none experienced disease progression or SCD over follow-up.[47]

In a retrospective study of 88 non-professional athletes, diagnosed with HCM between 1997 and 2017, all engaged in regular exercise [median 7 h/week (IQR: 6–14), >10 months/year), including competitive sports—contrary to the prevailing recommendations at the time. Over a median 7-year follow-up, no differences were observed in a composite outcome of symptoms, SCD, or cardiac arrest between those who continued to pursue vigorous exercise (≥3 sessions/week, ≥2 h/session, including high-intensity bouts) and those who detrained (<6 h/week). Notably, two cardiac arrests (0.3% per year) occurred among detrained athletes and outside the context of exercise. The cohort was generally low-risk [median ESC 5-year SCD risk score 2.2% (IQR: 1.7–3.0)], with only 11 participants (12.5%) classified as intermediate or high-risk.[18]

In a cross-sectional study of 121 individuals with HCM, of whom 44 (36%) engaged in vigorous sport, survival analysis showed no difference in ventricular arrhythmias between HCM athletes and non-athletes (log-rank $P = .36$). Being an athlete was not associated with ventricular arrhythmias adjusted for global longitudinal strain and maximal left ventricular wall thickness in multivariable analysis.[16]

Similarly, in a retrospective study of 53 athletes with low-risk HCM (mean ESC 5-year SCD risk score 1.9% ± 0.9%) who continued to participate in competitive sport, all remained symptom-free and none suffered syncope, sustained ventricular tachycardia or death at 4.5 years. Notably, 17% of the cohort comprised Black athletes who are perceived to be at higher risk, and half of the entire cohort were professional athletes.[19] In a cohort of 76 collegiate and professional athletes with genetic heart disease, including 40 (53%) with HCM, 91% elected to return to play after optimized guideline-directed management (including ICD implantation in 32%).[20] During the 7-year follow-up period, only 1 athlete (1.3%) experienced an exertional cardiac event, and there were no deaths.

In recent years, clinical trials and large prospective registries have increasingly supported the safety—and potential benefits—of moderate to vigorous intensity exercise in individuals with HCM (*Table 1*). The first systematic investigation came from a small, non-randomized study of symptomatic HCM patients enrolled in a cardiac rehabilitation programme, which demonstrated improved exercise capacity and reduced symptom burden. With no adverse events.[23] Two randomised controlled trials -including RESET-HCM—demonstrated that structured moderate-intensity exercise produced a significant improvement in peak oxygen consumption (pVO2), self-perceived physical limitation and quality of life in the trained groups compared with usual activity.[24,27] While neither was powered for safety, no serious adverse events or ventricular arrhythmias were reported.

The HIT-HCM trial further demonstrated that both moderate- and high-intensity training improve pVO2 to a comparable degree.[25] All participants were engaged in a moderate-intensity exercise regimen for 2 months before being randomized to continuing the moderate-intensity training or adding 1–2 supervised HIT sessions per week. Though the study was limited by the small sample size, it confirmed no increased risk of serious exercise-related arrhythmias or cardiac events. In the SAFE-HCM trial, individuals aged 16–60 were randomized to a high-intensity exercise programme or usual care. Individuals in the high-intensity exercise group improved their cardiorespiratory fitness, cardiovascular risk profile for atherosclerosis and psychological indices. Although arrhythmias were observed in the exercise group, they did not differ significantly from those in the control group.[28]

The multi-centre prospective LIVE-HCM registry followed 1660 individuals with HCM, comparing those engaged in vigorous (≥6 METs for ≥60 h/year; 42%) vs non-vigorous activity. After three years, there was no difference in the composite outcome of death, SCD, appropriate implantable cardioverter defibrillator (ICD) shock, or arrhythmic syncope (4.7% vs 4.6%; HR 1.01; 90% CI, 0.68–1.48; $P = .98$).[26] A subgroup of younger individuals (aged 14–22) was too small for meaningful analysis, but high school and varsity college athletes had a lower rate of adverse events than a similar aged, moderate-exercise/sedentary group [5.7/1000 person-years (95% CI, 0.8–40.8) vs 20.7/1000 person-years (95%CI, 9–46.2)].

Further reassurance is offered by the results of the ICD Sports Safety Registry,[48] which followed 372 athletes with ICDs (65 with HCM;13 [22%] engaged in high-level and competitive sports). Over a median follow-period of 31 months, there were no deaths or resuscitated arrhythmias during or following sports participation; one athlete received an appropriate ICD shock during practice.[48,49] Collectively, these data support the view that, when tailored to the individual's clinical profile, vigorous exercise, including competitive sport, does not appear to increase the baseline arrhythmic risk associated with HCM and suggest that absolute risk of exercise-related events is low (*Graphical abstract*).

The hidden price of inactivity

Individuals with HCM are prone to sedentary behaviour as a result of cardiac symptoms, perceived disability and fear of SCD, but also due to physicians discouraging exercise and providing vague advice, or no advice at all, in terms of recommended activities.[50] A national survey in the United States revealed that the time spent engaging in work or recreational physical activity was significantly less for individuals with HCM than for the general population, with most individuals reporting a purposeful reduction in activity after diagnosis.[51] In a study of 198 adults managed in a specialist HCM centre, 55% of individuals with HCM failed to meet the minimum recommendation of 150 min of moderate-intensity exercise per week when assessed by a dedicated questionnaire.[50] This proportion increased to 87% when assessed objectively with an accelerometer. A multi-centre cross-sectional study of children and teens with HCM reported that fewer than 10% met the recommendations for sixty minutes per day of moderate to vigorous physical activity, with 75% being under activity restriction from their cardiologist.[52]

Similar to the general population, individuals with HCM are susceptible to cardiovascular risk factors. Elevated body mass index (BMI) has been reported in over 70%, with half (35%) meeting the obesity criteria (BMI >30 kg/m^2).[50,53] Importantly, obese individuals with HCM have a two- to four-fold increased risk of developing heart failure with severe functional limitation [New York Heart Association (NYHA) functional class III/IV] compared with the non-obese, independent of other known determinants of heart failure symptoms.[53,54] Obesity has also been linked to increased left ventricular mass, proportional to the level of obesity, with obese HCM individuals being three times more likely to have a marked increase in left ventricular mass exceeding 120 g/m^2, compared with those of normal weight.[53]

Finally, obesity has been linked to a higher prevalence of provocable left ventricular outflow tract obstruction (LVOTO) and hypertension, which in turn contribute to elevated left ventricular mass.

A sedentary lifestyle promotes the development of risk factors for atherosclerosis, which is associated with excess mortality in individuals with HCM compared with the general population. In a cohort of 425 individuals with HCM, hypertension was present in 32% of White individuals and 58% of Black individuals and conferred a two-fold increased risk for the composite outcome of cardiovascular death, cardiac arrest or appropriate ICD therapy.[55] When HCM individuals develop coronary artery disease, their synergistic combination greatly enhances risk.[56,57]

Finally, the psychological impact of a diagnosis of HCM may adversely affect an individual's mental health, which in turn may influence prognosis and result in significant societal costs.[58] A negative psychological impact is amplified by the restriction of participation in vigorous exercise and competitive sports, particularly in athletic individuals for whom sports are a central part of their lives and livelihood. In a survey of largely recreational middle-aged exercisers with HCM, 54% found it difficult to adjust to exercise restrictions and many reported they found exercise recommendations vague and conflicting.[59] Exercise restrictions led to physical changes such as weight gain, as well as feelings of uncertainty and disability. Those with a history of higher levels of athleticism demonstrated greater psychological morbidity. Among 25 young competitive athletes recently diagnosed with a cardiovascular disease, including 5 with HCM, those who were previously engaged in high levels of competition and those who were frequently reminded of their underlying condition (daily medication, monitoring heart rate during activity) appeared at higher risk of psychological morbidity.[60]

Not surprisingly, highly motivated and trained HCM individuals often continue to engage in levels of physical activity that exceed restrictive recommendations, seeking to preserve the associated benefits. In a study exploring the motivations behind this behaviour, most participants cited improvements in mental and physical well-being, along with the importance of maintaining social connections, as key reasons for their continued activity.

Importantly, the negative impact of exercise restrictions on an individual's mental health may directly impact prognosis. Among 793 individuals with HCM, elevated anxiety levels were associated with three-fold increase in heart failure and 1.4-fold increased risk of SCD, and those with comorbid anxiety and depression had a four-fold higher risk of heart failure events and a 3.5-fold higher risk of SCD vs those with neither.[61]

Even in individuals with HCM who are encouraged to return to play or continue exercising after their diagnosis, psychological stressors are common and often under-recognized. These may include return-to-play anxiety, fear of ICD shocks during exertion, or maladaptive training behaviours driven by uncertainty or pressure to perform. Such factors may affect adherence to medical advice and contribute to emotional distress. Overall, despite the methodological limitations of individual studies, these findings underscore the importance of systematically identifying symptoms of anxiety and depression in individuals diagnosed with HCM, and to limit unnecessary lifestyle restrictions. Proactive psychological assessment and timely intervention—such as psychological counselling or other supportive measures—should be considered an integral part of comprehensive HCM care.[62]

Personalised risk evaluation and eligibility

A detailed, multidimensional evaluation of arrhythmic risk represents a cornerstone of clinical management in individuals with HCM and is key for determining the ideal level of activity. Indeed, reports in unselected HCM populations suggest an increased incidence of adverse events during vigorous exercise,[63] warranting caution in HCM individuals with distinct high-risk features such as severe LVOTO.[64] As the tools for risk stratification have become more refined, uncertainty inevitably remains when attempting to predict a rare event in a complex and dynamic biological system. Despite variations in stratification models between the ESC and AHA/ACC guidelines, key risk factors for SCD are similar and include: (i) previous episodes of cardiac arrest or sustained ventricular arrhythmias; (ii) younger age; (iii) family history of SCD or sustained ventricular arrhythmias attributable to HCM; (iv) severe left ventricular hypertrophy; (v) significant left atrial dilatation; (vi) detection of ventricular arrhythmias; (vii) history of recent arrhythmogenic syncope; (viii) presence of LVOTO; (ix) extensive fibrosis on cardiac magnetic resonance imaging; (x) left ventricular ejection fraction ≤50%; and (xii) presence of left ventricular apical aneurysm. The absence of all these features identifies individuals with HCM at low or very low risk.[1,65] In the presence of one or more of these features, different models have been proposed to aid exercise prescription and sport participation based on perceived risk.[66] However, common sense and clinical experience remain key in overcoming the many gaps in knowledge. Practical guidance for risk minimisation in HCM individuals willing to exercise are shown in *Table 2*.

The 2023 ESC guidelines recommend that vigorous exercise and competitive sport may be considered in asymptomatic low-risk individuals with morphologically mild HCM.[1] These recommendations have been adopted by several European countries and are reflected in national protocols for competitive sports eligibility.[67]

In contrast, the AHA/ACC guidelines do not specify exercise recommendations based on morphological criteria or SCD risk[21,65] and both the scientific statement from the AHA/ACC and from the Heart Rhythm Society (HRS) consider competitive sports participation reasonable in HCM after comprehensive expert assessment and informed shared decision-making.[21,64] The 2024 HRS consensus assigns a Class 2a recommendation ('reasonable') for return-to-play in athletes with HCM,[64] in contrast to the 2023 ESC guidelines, which offer a more cautious Class 2b recommendation ('may be considered').[1] This distinction reflects the HRS's interpretation of growing evidence of benefit balanced against the limited substantiation of risk.[64] A summary of U.S. and European perspectives on return-to-play in HCM is provided in the Supplementary Materials.

Of note, conventional SCD risk stratification models proposed by the AHA/ACC and ESC have not been validated for individuals engaging in vigorous exercise or competitive sports, raising concerns about their appropriateness in the athletes. The athletic status of those enrolled in the derivation and validation cohorts of these calculators is unknown, and individuals engaged in high-intensity sports were likely underrepresented—partly due to historical restrictions on athletic participation in HCM. In light of these considerations, decisions regarding vigorous exercise participation should be made within cardiomyopathy centres that possess the expertise to conduct a comprehensive, multidisciplinary evaluation, ensure fair discussion and provide close follow-up.

Finally, children and adolescents deserve particular consideration. On one hand, this age group has a critical need for regular physical activity to support healthy physical development, psychological well-being and social integration. On the other hand, HCM that presents early in life is often associated with a higher risk of arrhythmic events. While paediatric risk scores are available,[68,69] they do not incorporate intensity or type of physical activity and specific evidence in the literature remains limited and clinical decisions rely heavily on expert opinion.

Left ventricular outflow tract obstruction

A commonly encountered subgroup requiring consideration is athletes with obstructive HCM who self-report as asymptomatic. This subset presents a unique challenge, as current guideline-endorsed therapies are typically reserved for those with symptoms. However, in real-world practice, many individuals categorized as 'asymptomatic' may, in fact, experience functional limitation or exertional symptoms that are underestimated in daily life. In this context a comprehensive functional assessment—including cardiopulmonary exercise testing—plays a critical role in providing an objective evaluation. Furthermore, in truly asymptomatic individuals, adherence to standard precautionary principles for HCM during exercise (as summarized in *Table 2*) is recommended.

In the absence of symptoms or haemodynamic instability, consideration may still be given to pharmacological therapy to mitigate exertional LVOTO, when present. Surgical myectomy or alcohol septal ablation should be reserved for those with clear symptoms or objective functional limitation or haemodynamic instability (e.g. exertional syncope or hypotensive response to exercise), despite initial pharmacological therapy with beta-blockers or disopyramide. When performed in high-volume centres, these treatments are safe and provide radical

Table 2 Practical recommendations for individuals with hypertrophic cardiomyopathy

Before starting exercise or competitive sports	
Disease staging and risk stratification	Assess haemodynamics, cardiac function, and disease stage. Use conventional and novel risk factors for risk stratification, tailored counselling, and consideration of ICD implantation if indicated.
Clinical profile optimization • Optimize medical therapy as needed. Control exercise-induced LVOTO. • Manage arrhythmias and symptoms. • Avoid vasodilators and diuretics.	Ensure LVOTO control during exercise with beta-blockers and/or disopyramide. Consider septal reduction or myosin inhibitors in severe cases. Adjust beta-blocker dosage to prevent chronotropic incompetence, especially with myosin inhibitors. Temporarily discontinue exercise in individuals with concerning arrhythmias until evaluated and managed.
Promote healthy body weight and metabolic status	Rule out obstructive sleep apnoea. Prioritize dietary intervention and weight management before initiating vigorous activity in obese or detrained individuals.
Address comorbidities	Manage lipids, hypertension, smoking, and diabetes aggressively.
Functional assessment (cardiopulmonary exercise testing)	Assess baseline capacity, blood pressure response, and exercise-induced arrhythmias to guide individualized exercise prescription.
Exercise echocardiography	Evaluate for exercise-induced LVOTO and mitral regurgitation severity.
Ambulatory ECG monitoring during training	Detect arrhythmias; consider monitoring during a training session.
Emergency action plan	Encourage discussion with trainers, team-mates, and family about ensuring a safe environment. Recommend CPR training and access to an automated external defibrillator.
Exercise prescription	Tailor training to prior fitness level. Use a stepwise progression in sedentary individuals.
Athletes with an ICD	Programme ICD with long detection intervals and high-rate cut-offs to avoid inappropriate shocks. Establish an individualized critical heart rate threshold. Perform a maximal stress test for safety. Consider atrioventricular node-blocking agents for additional safety.
Performance-enhancing substances	Advise against anabolic steroids, growth hormone, and stimulants (e.g. caffeine-rich beverages), which may worsen LVOTO and increase sympathetic tone.
During exercise or competitive sports	
Avoid large meals pre-exercise	Postprandial hypotension may lead to dizziness, fatigue, or syncope, especially with LVOTO.
Hydration and electrolytes	Maintain adequate hydration before, during, and after exercise. Dehydration lowers preload and may exacerbate LVOTO and arrhythmias.
Setting selection	Prefer environments with established CPR protocols (e.g. gyms, athletic fields).
Supervision	Consider supervised sessions for previously sedentary individuals initiating training.
Environmental precautions	Avoid extreme temperatures: heat may cause hypotension and obstruction; cold increases blood pressure and myocardial oxygen demand. Discourage activities where syncope may be associated with significant harm (e.g. mountain climbing, underwater sports).
Manage isometric efforts	Intense static manoeuvres (i.e. weightlifting) may result in lightheadedness or syncope during Valsalva manoeuvre in those with LVOTO.
Warm-up and cool-down	Always include warm-up and cool-down phases. Cooling down helps prevent LVOTO-related symptoms.
Use wearables to monitor cardiovascular performance	Monitor progress, arrhythmias, and adherence to heart rate thresholds.
Follow-up	Annual comprehensive evaluation at a specialized center. Reassess fitness status and adapt recommendations accordingly.
Monitoring symptoms	Educate patients on warning symptoms. Instruct them to report any change in clinical status and refrain from exercise until cleared by a physician.

CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter defibrillator; LVOTO, left ventricular outflow tract obstruction.

and sustained symptomatic relief, demonstrated by marked improvement in exercise capacity and pVO2.[70] However, most symptomatic individuals with obstructive HCM fall within NYHA class II[71] and are thus not generally eligible for invasive treatment options, even though their symptom burden may interfere with an active lifestyle.

Following recent advances in pharmacological therapy, access to vigorous exercise may be offered today to an increasing number of individuals with obstructive HCM. The newly introduced cardiac myosin inhibitors (CMIs), mavacamten and aficamten, were developed to address excessive actin-myosin cross-bridge formation and

This summary table provides the core messages of the review

- Historical perception of risk: early data, notably from autopsy studies, linked athletes with HCM to SCD, leading to widespread restrictions on sports participation. However, these data suffer from many methodological limitations.
- Shifting evidence: multiple studies suggest that individuals with HCM who participate in vigorous exercise, including competitive athletes, do not have increased adverse cardiac events compared with less active individuals with HCM. Additionally, studies show improved cardiorespiratory fitness, reduced symptoms, better psychological well-being and quality of life in individuals with HCM who exercise regularly.
- Consequences of restriction: The majority of adults and children with HCM do not meet recommended physical activity guidelines, often due to physician-imposed exercise restrictions or vague, non-specific advice. This sedentary behaviour is associated with adverse outcomes, including increased rates of obesity (even in paediatric populations), greater left ventricular mass and LVOTO, heightened risk of heart failure and cardiovascular events, as well as significant psychological distress.
- Tailored exercise programmes are essential in HCM, exercise goals must be individualized, considering pre-diagnosis activity level, current fitness, age, personalized risk assessment, comorbidities and personal preferences.
- Vigorous exercise and competitive sports: the vast majority of HCM individuals should be encouraged to lead an active lifestyle, the low-risk subset defined by lack of well-established adverse features can safely engage in vigorous activities and in competitive sports.
- Shared decision-making has replaced the binary clearance model, promoting collaborative, informed choices involving athletes, families, and physicians. However, eligibility decisions are also shaped by national laws and sports body policies.

cardiac hypercontractility, resulting in reduced myocardial energy demands and improved diastolic function.[72–76] Cardiac myosin inhibitors demonstrated marked efficacy in reducing left ventricular outflow tract gradients, leading to improvements in exercise parameters, including pVO2, ventilatory efficiency (VE/VCO_2) and exercise duration in individuals with symptomatic obstructive HCM.[73,76,77] Notably, CMIs may be used as monotherapy in patients who do not tolerate beta-blockers or have chronotropic incompetence. Evidence from the FOREST-HCM trial suggests that, in individuals with favourable responses to CMIs, withdrawal of background therapy may be considered without compromising safety or efficacy, potentially improving functional capacity and minimizing polypharmacy.[78] Decisive evidence on this topic will be obtained by the ongoing MAPLE-HCM trial with aficamten. However, the negative effects of beta-blockers are not generalizable to athletic populations. In fact, low doses may be beneficial in certain settings. Therefore, any attempt to withdraw background therapy—particularly in athletic individuals—should be approached with caution while awaiting data specifically addressing this population.

As a general principle, when an individual with HCM experiences significant improvement in LVOTO following successful treatment—whether surgical, interventional, or pharmacologic—exercise prescription may be reconsidered in light of the updated haemodynamic and clinical profile.

Impact of the implantable cardioverter defibrillator on sport participation

While the ICD represents the only effective approach to prevent SCD in individuals at risk, the 2024 AHA/ACC HCM guidelines clearly recommend against implantation for the sole purpose of sports participation in the presence of a low-risk profile[65] as such an approach is likely to expose individuals to unnecessary device-related complications.[48] In ICD recipients, previous recommendations concerning sports participation have been restrictive,[43,79] despite lack of specific data, due to the assumption of an underlying high-risk substrate status. Data from the ICD Sports Registry, however, showed no ICD failures during competitive sport.[48] Accordingly, the 2024 HRS and 2025 AHA/ACC scientific statements consider competitive sports participation reasonable for athletes who have received an ICD for primary or secondary prevention, within a shared decision-making framework that takes into account the underlying diagnosis, comprehensive confirmation of rhythm stability, and the possibility of both appropriate and inappropriate device therapies.[21,64] Since ICD recipients, by definition, do not fit the criteria for low-risk HCM, there is no universal agreement on this liberal approach and more research is needed in the field.

Tailoring, prescribing, and monitoring exercise

By tailoring training programmes to individual needs and ensuring ongoing monitoring, clinicians can empower individuals with HCM to lead active and fulfilling lives while minimizing risks[80,81] (*Graphical abstract*). Following a comprehensive clinical evaluation, including a personal history of sports participation and ongoing training, exercise echocardiography and CPET, a well-structured training programme should align with the following principles:

(i) **Individualization.** Exercise regimens must be tailored to the individual's functional capacity, symptom burden, and risk profile. Trained individuals have a privileged starting point and may generally continue with vigorous activities, while individuals who have been sedentary, are overweight and/or have metabolic diseases, should start with moderate, rather than vigorous intensity activities. Such initial approach does not preclude participation in more intense activities over time. Individuals with high-risk features should receive more conservative recommendations and may require temporary discontinuation of training to optimize treatment. Implantable cardioverter defibrillator programming should be carefully adjusted, including setting higher rate detection zones, to minimize the risk of inappropriate shocks triggered by sinus tachycardia (*Table 2*). Although avoidance of contact sports is recommended to prevent device damage, participation may be possible with appropriate adjustments, including the use of protective equipment.

(ii) **Intensity.** Intensity can be determined using the Borg scale, percentage of maximum heart rate, heart rate reserve and peak VO_2, with calculation of the aerobic and anaerobic thresholds by CPET.[82,83] Use of these parameters in devising individualized exercise prescription should consider the impact of treatment, particularly beta-blockers.[84] In patients undergoing CPET, heart rates corresponding to individual ventilatory thresholds (*ventilatory threshold 1* marking onset of lactate accumulation; *ventilatory*

threshold 2 indicating the start of exercise-induced metabolic acidosis) can be used as an objective indicator of aerobic exercise intensity.[85] Notably, in most individuals, moderate, rather than vigorous, exercise will provide the greatest benefit when the objective is to promote weight loss, improve metabolic status and adopt a healthy lifestyle.

(iii) **Exercise setting.** Individuals with HCM should exercise in comfortable settings and should be advised about the risks of specific environmental conditions irrespective of exercise intensity, e.g. solo endurance activities in remote areas, mountain climbing or underwater sports (practical recommendations are provided in *Table 2*).

(iv) **Progression.** In untrained individuals aerobic training, focusing on moderate intensity, is effective in improving cardiorespiratory fitness and reducing cardiovascular risk. However, resistance training should also be encouraged targeting 40%–70% of a maximal repetition and starting gradually with 1–3 sets of 8–10 resistance exercises, performed at slow-moderate velocity, increasing weekly according to adaptation.[82] The frequency, duration and intensity of training sessions should increase progressively based on age, individual response, previous training, changes in medical therapy and clinical characteristics.[82]

(v) **Longitudinal monitoring.** individuals with HCM participating in vigorous exercise or competitive sports, re-evaluation is recommended on an annual basis, or more frequently when clinically required, including symptom review, resting and/or stress echocardiography, exercise testing -ideally CPET- and ambulatory arrhythmia monitoring performed during a typical training session. The *de novo* emergence of high-risk features—such as exercise-induced ventricular arrhythmias or significant LVOTO—should prompt a detailed re-assessment and renewed shared decision-making regarding ongoing sports participation. This structured and periodic approach allows for timely adjustments in management and helps ensure athlete safety.

(vi) **Wearable technology.** Wearable devices that track heart rate and rhythm during exercise can provide real-time feedback, enhancing safety and efficacy. Incorporating wearable technology can empower individuals with HCM to exercise safely while gaining valuable insights into their cardiovascular health. However, it is essential to recognize potential limitations of wearable devices and the need for professional counselling.

(vii) **Multidisciplinary collaboration.** Developing an exercise prescription for individuals with HCM often requires input from an expert multidisciplinary team. Even in large HCM centres, however, specific expertise in sports cardiology is not always available, and may require the external support of professionals such as sports medicine physicians and exercise physiologists. The multidisciplinary team should include psychological support, as anxiety or fear related to exercise is common in HCM and has been identified as a relevant cause of inactivity in this specific population.[50]

Conclusions: the role of shared decision-making

Systematic restriction of sports activity in HCM is no longer tenable in clinical practice. While no degree of clinical assessment can possibly guarantee a 'zero risk' scenario, contemporary evidence suggests that tailored exercise, including competitive sports practice, does not increase risks inherently associated with HCM, which are often low. The traditional, paternalistic model by which physicians unilaterally provide clearance or disqualification for sports participation is being supplanted by a more nuanced shared decision-making approach, i.e. a collaborative process that incorporates all relevant stakeholders—athletes, their families, institutions, cardiomyopathy experts and team physicians— once adequately informed. This approach enables a final decision based on personal preferences, perceived risks and consequences, physical and emotional development, professional aspirations, and economic or legal considerations.[86] Shared decision-making is firmly embedded in clinical guidelines and is regarded as a balanced, comprehensive approach, reducing decisional conflict,[87] although potential challenges and limitations exist.[86,88] While safety and SCD prevention remain the overarching aim, the potential harms of sports disqualification should be carefully weighed and addressed in the process.

Emerging evidence suggests that the implementation of shared decision-making within expert cardiomyopathy centres, coupled with appropriate risk stratification and follow-up, is associated with low incidence of adverse cardiac events. Furthermore, shared decision-making is already an integral part of management algorithms for ICD implantation, surgical vs pharmacological management of LVOTO and advanced heart failure treatment options.[1]

Despite considerable progress, major challenges remain. Specifically, the legal implications surrounding liability in scenarios where adverse events occur after an athlete is cleared to play may vary. Certain countries do not accept a shared decision approach, and eligibility determinations are shaped by the legal and policy frameworks established by the relevant governing bodies. This interplay between shared decision-making and regulatory frameworks renders eligibility decisions particularly complex and compromises international equity in return to play following a diagnosis of HCM. Thus, harmonising differing national approaches represents an important future goal.

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Supplementary data

Supplementary data are available at *European Heart Journal* online.

Declarations

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