Position Statement

Position Statement of the Argentine Federation of Cardiology's Exercise Cardiology Committee on sports activity in hypertrophic cardiomyopathy.

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ABSTRACT

Historically, individuals with hypertrophic cardiomyopathy (HCM) have been excluded from vigorous exercise and competitive sports due to the risk of sudden cardiac death (SCD), based on retrospective studies. Recent evidence has challenged this paradigm, demonstrating improvements in functional capacity, quality of life, cardiovascular and psychological health when competitive sports are included in low-risk individuals. For those who do not meet these criteria, evidence is more limited. However, participation in competitive sports can still be considered on an individual basis.

A contemporary approach to HCM should be based on individualized, goal-oriented exercise prescription, multidisciplinary intervention, and shared decision-making.

In this review, we critically appraise the evidence on vigorous exercise and competitive sports and explore how new data are reshaping clinical practice and international guidelines, laying the groundwork for a new paradigm in the field.

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most frequent genetic heart disease in young athletes, acknowledged as one of the main causes of sudden cardiac death (SCD) associated to exercise in this age group, although most of patients remain asymptomatic and with low risk of severe events.[1,2,3] It has traditionally been considered a contraindication for the practice of sports, particularly of high intensity and competitive, due to the risk of SCD. However, current evidence and recommendations have shown that most individuals with HCM present a stable clinical course and a low absolute yearly risk of arrhythmic events when evaluated, stratified and followed properly.[1,2]

The most recent guidelines of the American College of Cardiology (ACC), American Heart Association (AHA), Heart Rhythm Society (HRS) and other societies recommend that patients with HCM can benefit from recreational exercise of mild to moderate intensity, improving functional class and quality of life, not increasing the risk of arrhythmic events.[1,2] In competitive sports, in selected cases and after an expert evaluation, with a structured process of shared decision making (SDM), it may be reasonable to authorize participation at competitive level.[4,5] The classical restrictive approach has been replaced by an individualized model supported by a better understanding of phenotypical heterogeneity. The development of advanced tools of cardiovascular imaging, and the incorporation of the SDM concept, have made a significant contribution in the final advice for sports practice.[2,3]

INITIAL EVALUATION

A detailed and multidimensional evaluation of arrhythmic risk represents the cornerstone of clinical management in people with HCM, and is key to determine the ideal level of activity.

The evaluation of patients with HCM that wish to carry out physical activity or participate in sports should be comprehensive, systematic and made by physicians with experience in sports cardiology.

This initial evaluation should include: clinical history with particular emphasis on family and personal history of SCD, unexplained syncope, physical examination, 12-lead ECG, Holter monitoring, graded exercise testing (GXT) and/or cardiopulmonary exercise test (CPET), and cardiovascular imaging studies such as echocardiogram and cardiac MRI.

ECG

The extent of left ventricular hypertrophy (LVH) by imaging is not specific of HCM, and could be secondary to different pathologies. In this context, ECG is extremely useful to guide the differential diagnosis between sarcomere HCM and its phenocopies. Although Q waves with positive T waves in the same leads (Q/T mismatch) and giant T wave (0.10 mm) in the anterolateral leads support the diagnosis of sarcomere hypertrophic cardiomyopathy, short PR interval and/or ventricular preexcitation are frequent findings in deposit diseases, such as glycogen storage disease, Danon disease, PRLAG2 gene mutations and Anderson-Fabry

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disease, and can also be found in mitochondrial diseases.[6]

In HCM, numerous electrocardiographic anomalies are observed and these could be the only manifestation of disease in an early stage. These alterations are determined by extension, degree and distribution of LVH, as well as by the presence of fibrosis and/or necrosis of the cardiac muscle, and by the appearance of intraventricular conduction disorder.[7,8] Around 95% of patients with diagnosis of HCM present ECG alterations, with no pathognomonic signs. Likewise, normal ECG is present in 5% to 10% of cases, and is associated with less severe phenotypical forms and a favorable course; however, an abnormal ECG pattern does not predict future SCD events or appropriate ICD therapy.[9]

Abnormal Q waves are due to localized loss of electrical vectors associated to transmural fibrosis, and the result of the initial QRS vector due to abnormalities due to disproportionate hypertrophy of the interventricular septum and/or free baseline wall. Recent studies that investigate the correlation between the Q waves in ECG and increase in myocardial late gadolinium enhancement in MRI have obtained mismatching results.

Although increased QRS voltages are usually identified in young healthy individuals, this characteristic is usually associated with other anomalies in HCM, and just 2% can show isolated criteria of QRS voltage (Sokolow-Lyon index and Cornell voltage criteria). Complete bundle branch block is extremely rare to see, and in general is due to surgical history.

ST-T segment anomalies are usual in HCM, associated to T-wave inversion.

Inverted T-waves in inferior wall in absence of hypertrophy are identified in 6% of black athletes and in 2% of white ones. Inverted T waves in lateral wall should always be considered abnormal, and may precede the phenotypical manifestations of cardiomyopathy; therefore, clinical surveillance is fundamental.

QTc prolongation probably reflects the interaction of hypertrophy, fibrosis and left ventricular outflow tract obstruction and electrophysiological remodeling of cardiomyocytes.[7]

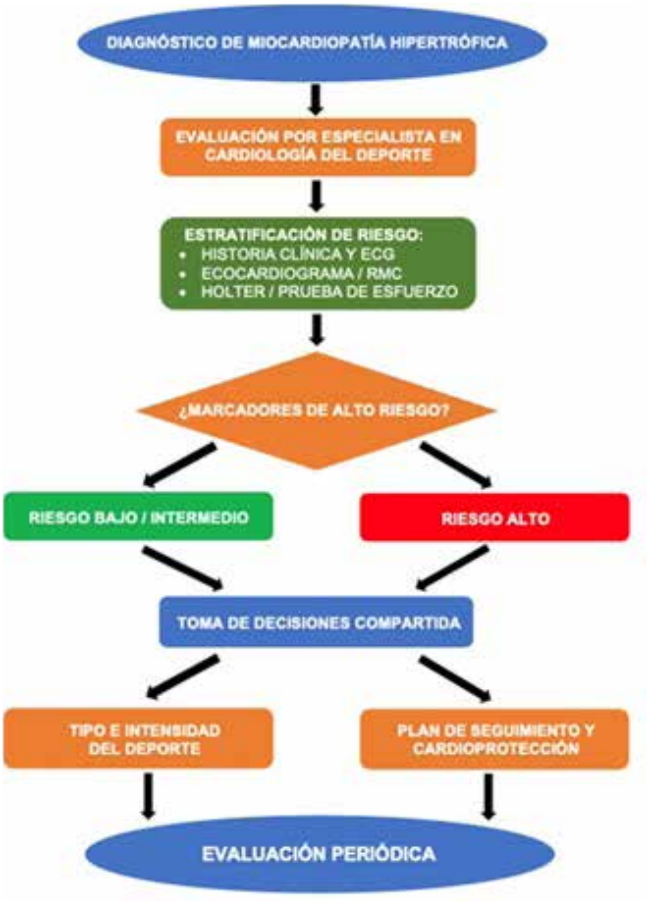

FIGURE 1.
FAC algorithm for evaluation and sports eligibility in hypertrophic cardiomyopathy
Description: confirmed diagnosis → comprehensive evaluation → risk stratification → identification of high risk markers → SDM → definition of sports and cardioprotected environment → longitudinal monitoring.
Based on 2024-2025 AHA/ACC, 2023 ESC Cardiomyopathy and 2020 ESC Sports Cardiology guidelines.

STRESS TEST

Stress test, with its different associated methodologies, is an essential component of noninvasive evaluation of HCM. It is involved in all diagnostic and prognostic algorithms of carriers of this disease.[10,11] It provides information on the severity and possible mechanism of functional limitation in case of left ventricular outflow tract obstruction, abnormal arterial response, chronotropic incompetence, arrhythmias, ischemia and/or reduction in heart rate reserve.[1]

The possibility of complications is extremely low, $<0.4\%$ in the series and its diagnostic contribution compensates the risk by far.[12] The stress test with echocardiography has been added to the standard clinical evaluation and the diagnostic armory in tests limited by symptoms. In symptomatic patients, without outflow gradient in rest, stress test with ultrasound is the most used method to prompt obstruction, which may predict the future development of heart failure symptoms and differentiate them from those without obstruction, with the logical therapeutic implications. On the other hand, there is no justification to perform it if patients have a resting gradient >50 mmHg.

We should be cautious when performing exercise tests in patients with cardioverter implantable defibrillators (CDI) to prevent unexpected shocks, whether appropriate or inappropriate.

Insufficient chronotropic response occurs in up to 25% of patients and it may indicate a low peak oxygen consumption.

Hypotensive or insufficient response to stress is defined as the inability to elevate pressure at least 20 mmHg during exercise peak over baseline or a hypotensive response. It could be a determinant factor in risk stratification, and some authors consider it as an indicator of poor outcome. It may appear in 20-40% of patients.[10] It lacks positive predictive value, but its negative predictive value is very useful to identify patients in low risk, who may restart sports practice in the presence of other low risk elements.[13]

As to myocardial ischemia assessment, stress test in HCM is associated to a considerable rate of false positives, due to the high prevalence of preexisting electrocardiographic anomalies. Although alterations have been proven by microcirculation disease.

Stress test associated to perfusion with radioisotopes is as conflictive, displaying low diagnostic specificity, but also with an elevated negative predictive value.[10]

Cardiopulmonary exercise test (CPET) allows to stratify risk in patients with non-conclusive symptoms or with etiological relationship, constituting a central tool for a comprehensive functional characterization of HCM, by allowing a dynamic evaluation of the interaction between heart rate, "systolic volume" and ventilatory efficiency, beyond resting findings.[11] Recent data show a significant correlation between oxygen pulse (O2P) and systolic volume in exercise, allowing to assess cardiac reserve in these patients. The combined identification of chronotropic incompetence (peak HR <80% of the expected value) and O2P reduction (<10% of the expected value), integrated in a new proposed classification, called RoMa, allowed to discriminate hemodynamic profiles with progressive deterioration of peak VO2, increase in the VE/VCO2 slope and less increase in systolic volume and cardiac output, regardless of ejection fraction or the obstructive gradient in rest.

From a practical view, CPET quantifies functional capacity, provides information on the limitation to exercise by differentiating predominantly chronotropic compromise, of systolic reserve or combined, which is particularly relevant for risk stratification, decision making on sports eligibility and therapeutic monitoring in HCM.[14]

Only 1.5% of people with hypertrophic cardiomyopathy (HCM) present a max VO2 exceeding the expected values, and no people have been detected with HCM whose max VO2 is 20% more than the expected maximum or 45 ml/kg/min (absolute value) in bicycle ergometer.[11]

In contrast, elite athletes with borderline left ventricular hypertrophy compared to athletes with borderline cardiomyopathy present max VO2 values in a 55 to 70 ml/kg/min range, exceeding the values expected in up to 50%. A VO2 of 50 ml/kg/min, or 20% of the maximum expected value, allows to completely differentiate both groups.[15]

Doppler echocardiography, including advanced techniques and stress echocardiography

Echocardiogram plays an essential role in diagnosis, prognostic assessment and decision making in patients with HCM. A clear sample of the value of transthoracic value is the fact that prognostic factors can be identified:

- Severe ventricular hypertrophy (maximum septal thickness ≥30 mm).[1,5]
- Left ventricular apical aneurysm.[1,5]
- Left ventricular systolic dysfunction (ejection fraction ≤50%).[1,5]
- Untreated significant left ventricular outflow tract obstruction.[5]
- Extensive myocardial fibrosis in cardiac MRI, defined as late gadolinium enhancement ≥15% of the left ventricular mass, associated to greater arrhythmic risk.[5]

The images obtained in rest and during stress provide complete and supplementary information, which allows to clarify the mechanisms responsible for the obstruction and the symptoms.

Transthoracic echocardiogram (TTE) constitutes the primary imaging modality in most patients, while transesphageal echocardiogram (TEE), MRI and cardiac CT provide supplementary information, particularly in selected patients when TTE is inconclusive. In comparison with cardiac MRI, echocardiogram does not diagnose 6% of HCM cases, underestimates maximum thickness in 20% and presents limitations in visualization of segmental apical, anterolateral or inferoseptal hypertrophy.

TTE should be performed as part of the initial evaluation of all patients with suspicion of HCM to establish a definitive diagnosis and, once confirmed, repeat every 1 to 2 years in the absence of clinical changes. It is, along with rest ECG, the main tool for screening first-degree relatives, both in the initial evaluation and in the periodical follow-up of genotype positive-phenotype negative individuals.

Speckle tracking techniques provide supplementary functional information, allowing to detect subclinical dysfunction and being associated to a greater load of fibrosis and arrhythmic risk.[16]

Myocardial longitudinal velocities and strain parameters (strain and strain rate), derived from tissue Doppler or speckle tracking, are usually reduced in the site of hypertrophy, as evidence of early systolic dysfunction, even before LVEF deterioration.

A decreased septal longitudinal and regional deformation (greater than -10%) is associated with greater susceptibility to ventricular arrhythmias, and may be altered before the development of septal thickness increase in relatives carriers of genetic variants.[17] A greater deterioration of longitudinal strain correlates with a greater degree of fibrosis and with the presence of ventricular arrhythmias.[18]

Stress echocardiography allows to identify dynamic gradients, inappropriate contractile response and elevation of pulmonary pressures induced by exercise, being key for clinical decisions in a context of sports.[19,20] These techniques should be interpreted in an integrated manner with conventional echocardiography and MRI.

GENETICS

Genetics plays an important role in the evaluation of sportspeople with HCM, as a supplementary diagnostic tool and as part of risk stratification.[5] The identification of pathogenic variants in sarcomere genes, mainly MYH7 and MYBPC, confirms the primary nature of hypertrophy and allows to differentiate HCM from the physiological remodeling of "athlete's heart" with more accuracy; particularly in borderline scenarios of mild septal hypertrophy.[21,22]

In the sports field, this differentiation has direct implications on eligibility and the type of activity allowed. Likewise, in genotype positive-phenotype negative individuals, a molecular finding demands a structured longitudinal monitoring, as high intensity exercise may influence penetrance and phenotypical expression in predisposed individuals. From a practical point of view, integrating genetic information with family history of sudden cardiac death, arrhythmic load, presence of fibrosis in MRI and hemodynamic response to exercise allows to make decisions individually and shared in regard to training and competition. Furthermore, the genetic study enables cascade familial screening, a particularly relevant aspect in families with physically active young people.[5]

RISK STRATIFICATION OF SUDDEN CARDIAC DEATH

Risk stratification should integrate clinical, electrophysiological (NSVT) cardiac imaging variables.

High risk markers include:

- Familial history of sudden cardiac death;
- Previous cardiac arrest;
- Sustained VT;
- Arrhythmic syncope;
- Severe hypertrophy (≥30 mm);
- Apical aneurysm;
- LVEF ≤50%;
- Untreated significant obstruction;
- Extensive fibrosis (≥15%);
- Repetitive non-sustained VT and high load of premature ventricular contractions.

In the presence of these markers, decisions should be cautious and guided by specialized teams.[1,2,3,4,5,23]

PARTICIPATION IN SPORTS

Mild to moderate recreational exercise should be recommended to all patients with HCM.

Participation in competitive sports should be evaluated by SDM, considering risk profile, type of sports, preferences of sportspeople and availability of cardioprotected environments.

For vigorous activities or competitive sports, participation should be considered reasonable in selected patients after an evaluation and prognostic stratification, under a model of SDM with Sports Cardiology experts; particularly in those with no high risk factors as those mentioned previously.

Evaluation should be comprehensive, including thorough clinical history, cardiac imaging tests (echocardiogram, MRI), stress test, and when indicated, genetic studies that may clarify specific situations, not just in sportspeople, but also in those who are not.[1,2,3,4,23,24,25]

In young sportspeople, it is essential to carry out yearly reevaluations, and a longitudinal monitoring, given the possible phenotypical progression and variable penetrance of the disease.

The specific recommendations for the practice of sports in young sportspeople with HCM can be summarized in the following:

- Recreational exercise of mild to moderate intensity recommended for all patients with HCM, as it improves functional class and quality of life, without increasing the risk of arrhythmic events.[1,3,24]
- Participation in competitive sports is reasonable in genotype positive by phenotype negative individuals; that is to say, carriers of pathogenic mutations with no evidence of ventricular hypertrophy or symptoms, after expert evaluation and regular follow-up.[1,4,23,26]
- In phenotype positive patients (with ventricular hypertrophy), the participation in competitive sports can be considered after an expert evaluation, SDM and risk management. This includes optimization of treatment, access to automated external defibrillators (AED) and establishment of an emergency plan in the sports environment. In cases of significant obstruction, it is recommended to treat the obstruction before returning to sports activity.[1,2,4,23]
- Universal restriction of vigorous physical activity is not recommended, and neither is the placement of an ICD just to allow the practice of sports. ICDs should be implanted only if patients meet standard criteria of high risk, regardless of the intention to play sports.[1,23,26]

There are limitations and uncertainty areas, particularly in children and teenagers, where prospective data is scarce and phenotypical penetrance may vary with age. Evidence about safety in competitive sports for patients in high risk is limited, so an individualized evaluation is required, as well as more studies in this group.[3,23]

In brief, the practice of sports in young people with hypertrophic cardiomyopathy should be individualized, based on risk stratification, periodical reevaluation and SDM, allowing participation in most cases, particularly in the absence of high risk factors and after an appropriate management of the disease.

CONSIDERATIONS IN REGARD TO THE TYPE OF SPORTS

There are differences according to the type of sports. The guidelines from the American College of Cardiology and the American Heart Association acknowledge that not all sports entail the same risk of young people with HCM.[1,4] Recommendations should adjust to the type of sports and the conditions in which they are carried out, contact, hydration state, altitude, etc., very common in adventure and ultra-endurance sports.

It is important to provide advice in terms of the sports supplements allowed and a safe use of them in the sports field, avoiding use of anabolic steroids, stimulants with caffeine, and all types of substances that stimulate the sympathetic system, due to the risk of arrhythmias and increase in obstructive gradients.

For this reason, in these aspects, specialists should know about types of competitions and the conditions in which the activity will be developed by the patient/sportsperson.

In summary, there are indeed differences in the recommendations for young sportspeople with HCM according to the type of sports they practice. High intensity sports, contact sports, or the risk of dehydration require more caution and an individualized assessment, while low intensity sports are usually safer. Participation should be based on risk stratification, the type of sports and what are the goals of the practice (competitive or recreational with low intensity). SDM with longitudinal follow-up, multidisciplinary suggestions and optimization of the treatment are key in the success of advising patients/sportspeople.[1,4,23,24]

CONCLUSIONS

Based on what was presented here, we can state that sports practice in patients with HCM should be individualized, based on risk stratification, periodical reevaluation and SDM, allowing participation in most cases, particularly in the absence of high risk factors and after an adequate management of the disease, including the indication for ICD in selected cases, having as priority a safe practice in cardioprotected environments, with staff trained in basic CPR and early access to defibrillators.

Physical activity and sports in HCM should be approached from a modern, individualized and non-restrictive view, based on risk stratification, periodical evaluation and SDM with the patient. Participation in sports is possible in most cases when evaluations and follow-up are made by specialists in Sports Cardiology.

BIBLIOGRAFIA

1. Ommen SR, Ho CY, Asif IM, et al. 2024 AHA/ACC/AMSSM/HRS/PACES/SCMR Guideline for the Management of Hypertrophic Cardiomyopathy: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol **2024**; 83: 2324 – 2405.
2. Zampieri M, Saberi S, D'Ascenzi F, et al. Hypertrophic cardiomyopathy: changing the paradigm of exercise prescription and competitive sport participation. Eur Heart J **2026**; 47: 902 – 915.
3. Bogle C, Colan SD, Miyamoto SD, et al. Treatment Strategies for Cardiomyopathy in Children: A Scientific Statement From the American Heart Association. Circulation **2023**; 148: 174 – 195.
4. Kim JH, Baggish AL, Levine BD, et al. Clinical Considerations for Competitive Sports Participation for Athletes With Cardiovascular Abnormalities: A Scientific Statement From the American Heart Association and American College of Cardiology. Circulation **2025**; 151: e716 – e761.
5. Arbelo E, Protonotarios A, Gimeno JR, et al. 2023 ESC Guidelines for the management of cardiomyopathies. Eur Heart J **2023**; 44: 3503 – 3626.
6. Rapezzi C, Arbustini E, Caforio ALP, et al. Diagnostic work-up in cardiomyopathies: bridging the gap between clinical phenotypes and final diagnosis. A position statement from the ESC Working Group on Myocardial and Pericardial Diseases. Eur Heart J **2013**; 34: 1448 – 1458.
7. Finocchiaro G, Sheikh N, Biagini E, et al. The electrocardiogram in the diagnosis and management of patients with hypertrophic cardiomyopathy. Heart Rhythm **2020**; 17: 142 –151.
8. Gersh BJ, Maron BJ, Bonow RO, et al. 2011 ACCF/AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation **2011**; 124: 2761 – 2796.
9. Salinas-Arce J, Gonzales-Luna AC, Cabrera-Saldaña M, et al. [Arrhythmias and Sudden Death Risk in Hypertrophic Cardiomyopathy]. Arch Peru Cardiol Cir Cardiovasc **2020**; 1: 94 – 104.
10. Rowin EJ, Maron BJ, Olivotto I, et al. Role of Exercise Testing in Hypertrophic Cardiomyopathy. JACC Cardiovasc Imaging **2017**; 10: 1374 – 1386.
11. Sharma S, Elliott P, Whyte G, et al. Utility of cardiopulmonary exercise in the assessment of clinical determinants of functional capacity in hypertrophic cardiomyopathy. Am J Cardiol **2000**; 86: 162 – 168.
12. Drinko JK, Nash PJ, Lever HM, et al. Safety of stress testing in patients with hypertrophic cardiomyopathy. Am J Cardiol **2004**; 93: 1443 – 1444,
13. Sharma S, Firoozi S, McKenna WJ. Value of exercise testing in assessing clinical state and prognosis in hypertrophic cardiomyopathy. Cardiol Rev **2001**; 9: 70 – 76.
14. Willixhofer R, Mapelli M, Baracchini N, et al. Cardiopulmonary exercise testing in hypertrophic cardiomyopathy: the role of reduced O2 pulse and chronotropic incompetence in myocardial adaptation. Eur J Prev Cardiol **2025**; :zwaf402.
15. Sharma S, Elliott PM, Whyte G, et al. Utility of metabolic exercise testing in distinguishing hypertrophic cardiomyopathy from physiologic left ventricular hypertrophy in athletes. J Am Coll Cardiol **2000**; 36: 864 – 870.
16. Nagueh SF, Phelan D, Abraham T, et al. Recommendations for Multimodality Cardiovascular Imaging of Patients with Hypertrophic Cardiomyopathy: An Update from the American Society of Echocardiography, in Collaboration with the American Society of Nuclear Cardiology, the Society for Cardiovascular Magnetic Resonance, and the Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr **2022**; 35: 533 – 569.
17. Thaman R, Gimeno JR, Murphy RT, et al. Prevalence and clinical significance of systolic impairment in hypertrophic cardiomyopathy. Heart **2005**; 91: 920 – 925.
18. Haland TF, Almaas VM, Hasselberg NE, et al. Strain echocardiography is related to fibrosis and ventricular arrhythmias in hypertrophic cardiomyopathy. Eur Heart J Cardiovasc Imaging **2016**; 17: 613 – 621.
19. Pelliccia A, Sharma S, Gati S, et al. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease. Eur Heart J **2021**; 42: 17 – 96.
20. Wolff S, Venegas P, Franco C, et al. Imágenes Cardíacas en Cardiología del Ejercicio: Distribuna Editorial Médica; **2025**. 1° Edición. ISBN:9786287673397.
21. Ho CY, Day SM, Ashley EA, et al. Genotype and Lifetime Burden of Disease in Hypertrophic Cardiomyopathy: Insights from the Sarcomeric Human Cardiomyopathy Registry (SHaRe). Circulation **2018**; 138: 1387 – 1398.
22. Sedaghat-Hamedani F, Kayvanpour E, Tugrul OF, et al. Clinical outcomes associated with sarcomere mutations in hypertrophic cardiomyopathy: a meta-analysis on 7675 individuals. Clin Res Cardiol **2018**; 107: 30 – 41.
23. Lampert R, Chung EH, Ackerman MJ, et al. 2024 HRS expert consensus statement on arrhythmias in the athlete: Evaluation, treatment, and return to play. Heart Rhythm **2024**; 21: e151 – e252.
24. Leggit JC, Whitaker D. Diagnosis and Management of Hypertrophic Cardiomyopathy: Updated Guidelines From the ACC/AHA. Am Fam Physician **2022**; 105: 207 – 209.
25. Miller EM, Brown E, Christian S, et al. Genetic testing and counseling for hypertrophic cardiomyopathy: An evidence-based practice resource of the National Society of Genetic Counselors. J Genet Couns **2025**; 34: e1993.
26. Maron BJ, Udelson JE, Bonow RO, et al. Eligibility and Disqualification Recommendations for Competitive Athletes with Cardiovascular Abnormalities: Task Force 3: Hypertrophic Cardiomyopathy, Arrhythmogenic Right Ventricular Cardiomyopathy and Other Cardiomyopathies, and Myocarditis: A Scientific Statement From the American Heart Association and American College of Cardiology. Circulation **2015**; 132: e273 – e280.