Unlocking “Pro” Secrets: Optimizing Echocardiographic Imaging in the Diagnosis and Management of Hypertrophic Cardiomyopathy

A Series of HCM Center of Excellence-Based Live and Live-Streamed Programs
Led by Regional Echocardiography Experts

Enduring Program on
www.academiccme.com

May 31, 2025 – May 30, 2026

Executive Summary

Needs Assessment, Gaps/Barriers. Hypertrophic cardiomyopathy (HCM) affects 1 in 200-500 people and leads to hypercontractility of the cardiac ventricles.[1] Cardiomyopathies, familial or sporadic, have become recognized as a leading source of cardiac morbidity and mortality. HCM, which is most often familial, is the leading cause of sudden death in young adults,[2] often after following an insidious clinical course. Early detection is likely beneficial for prevention of significant morbidity and mortality,[3] especially in high-risk patients such as those with a family history of sudden death and those with a history of unexplained syncope or non-sustained ventricular tachycardia, or with an incidentally discovered aortic flow murmur. While a multimodal imaging approach is often necessary to risk-stratify HCM and occasionally to establish the diagnosis, **echocardiography is the primary diagnostic test for this disease**.

The 2024 AHA/ACC/AMSSM/HRS/PACES/SCMR Guideline for the Management of Hypertrophic Cardiomyopathy state that "Echocardiography is the primary imaging modality in most patients . . . [to establish] the diagnosis (or excluding alternative diagnoses), evaluating the severity of the phenotype, and evaluate for concomitant structural and functional cardiac abnormalities (eg, systolic, diastolic, valvular function). Characterization of dynamic LVOTO, including the integral role of the mitral valve, is a key strength of echocardiography. Documentation of the maximal wall thickness, cardiac chamber dimensions, systolic function, and the presence of LV apical aneurysm all inform phenotype severity and SCD risk stratification."[4] The 2023 ESC guidelines for the management of cardiomyopathies state simply, "The noninvasive nature and widespread availability of echocardiography make it the main imaging tool, from initial diagnosis to follow-up."[5]

Early and definitive diagnosis of HCM is now more important than ever, given the regulatory approval of the first drug capable of manipulating the power output of cardiac muscle at the molecular level via a targeted small-molecule inhibitor of myosin (a cardiac myosin inhibitor, or CMI), thereby abrogating the elevated contractile force of the HCM heart, potentially improving symptoms and altering the course of disease progression.[6] Focused, highly competent echocardiography is required for the diagnosis of HCM, assessing progression of disease and impact of therapy, and to meet Risk Evaluation and Mitigation Strategies (REMS) requirements for the continued use of the approved CMI in patients with HCM.

AcademicCME therefore proposes an accredited live and live-streamed expert-led educational session on key elements of the HCM echocardiographic evaluation, including rationale, patient positioning and coaching, probe placement, obtaining key measurements accurately, and consistent reporting of findings. These two-hour programs will be jointly taught by an HCM expert cardiologist and that expert's designated most skilled and best instructor in echocardiography for HCM, both from a designated US HCM Center of Excellence. The targeted audience will be echocardiographers from the geographic region near that Center. The symposium will be presented live but will also be live-streamed, with optimized video technology to ensure the audience has clear and instructive views of both probe placement and imaging findings regardless of their viewing venue.

Over the past month, AcademicCME has canvassed a geographically dispersed sample of US HCM Centers of Excellence and has received uniformly positive and enthusiastic responses from potential faculty, including

confirmation of interest, of suitable facilities, and of a select echocardiographer to serve as lead faculty for his/her colleagues.

Target Audience and Audience Generation. The target audience for this program will be echocardiographers/cardiac sonographers who work in hospitals and clinics in the general geographic region of the "host" HCM Center of Excellence. Audience recruitment will be a joint effort of AcademicCME and the Center of Excellence faculty members, and will include e-mail blasts, direct mail, electronic bulletin board postings for echocardiographers, and word of mouth.

Curriculum Format. The program curriculum will be produced through a Steering Committee of three HCM specialist cardiologists and three echocardiographers. The content will be developed and produced exclusively to target the interests and educational needs of the target audience—echocardiographers/cardiac sonographers who image patients with a suspicion of or confirmed HCM. The program will focus on the critical components of the echocardiographic evaluation as recommended in the 2024 AHA/ACC/AMSSM/HRS/PACES/SCMR Guideline for the Management of Hypertrophic Cardiomyopathy.[4]

The 2.5-hour live presentations will consist of demonstration of techniques and tips aimed at ensuring the objectives of echocardiographic imaging in HCM are met. The echocardiographer faculty will demonstrate optimal image acquisition and the cardiologist faculty will explain the clinical importance of each metric. They will follow the curriculum developed by the Steering Committee but will provide their own additional insights and responses to learner questions.

Each 2.5-hour program will be video-archived and a single enduring version of the program will be developed that captures and collates the best demonstrations, didactics, and image capture from the series. This enduring program will be available on demand on academiccme.com and will be publicized to potential learners on other AcademicCME platforms including @cardiomet_ce, where AcademicCME's tweetorials on HCM have garnered a large and durable following.

Learning Objectives.

After completing the programs in this educational initiative, echocardiographer learners should have:

- A clear understanding of the structural manifestations of HCM, how they appear on echocardiography, and how progression or improvement of the disease is best tracked by imaging
- Operational confidence to obtain key images and measurements in the echocardiographic assessment of potential and known HCM
- A protocolized approach to obtaining the images and measures required in patient follow-up per the required REMS program for the currently approved CMI

Program Evaluation and Outcomes Reporting:

- **Symposium and enduring webcast.** Up to Moore's Level 5 subjective will be assessed for these activities. Learners will be required to complete a pre-test, intermediate polling questions, and a post-test, and an evaluation survey to measure knowledge and competency changes. To specifically measure change in understanding of cardiac imaging applications in the diagnosis and management of HCM, the pre-test survey, intermediate polling questions, post-test survey, and

follow-up survey will utilize knowledge questions and a self-reported practice evaluation. A 2-month follow up survey will be distributed to all webcast completers to evaluate retention of knowledge and implemented practice changes.

Communication Plan. Coordinated utilization and analysis of data collected from initial and follow-up assessments will provide a rich multi-dimensional report of outcomes of this educational activity. AcademicCME will provide the Sponsor with quarterly user reports, evaluations, and an educational impact summary tailored to both delivery platforms.

Innovation. Expert-led, intensive education in the priorities and subtleties of echocardiographic imaging in HCM that is targeted directly to the end-user is uncommon. In the proposed program, AcademicCME will harness the gravitas of HCM Centers of Excellence and the caliber of faculty these centers are optimally equipped to provide into a unique, highly accessible, interactive, comprehensive program.

Budget: The overall budget for the proposed program is $316,250, although this scalable to the number of programs executed. The detailed budget is provided in the attachment accounts for 8 live sessions and the enduring program.

Faculty: 6 steering committee members (3 cardiologists, 3 sonographers)
16 faculty members for 8 live sessions (8 cardiologists, 8 sonographers)

Activity Reach:
Live/live-streamed symposia (n=8):

- 600 anticipated learners
- 500 anticipated certificates

Enduring on-demand webcast:

- 1000 anticipated learners
- 700 anticipated certificates

Proposed Agenda (150 minutes, intended for a Saturday session):
The course will open with a rapid-fire assessment of several pre-recorded scans/images that illustrate key concepts in HCM imaging.

I. Structural Pathology and Pathophysiology of HCM and Visualization of Pathology with Echocardiography
II. Echocardiographic Techniques and Tips to Optimize:
    - Establishment of the diagnosis of HCM
    - Determination of the hypertrophy pattern and measurement of LV wall thickness
    - Assessment for the presence of LV apical hypertrophy and apical aneurysms
    - Assessment of LV systolic and diastolic function
    - Assessment of mitral valve structure, motion, and function, including detection of systolic anterior motion (SAM) of the valve
    - Assessment for diastolic dysfunction with parameters such as E/A ratio, deceleration time of E wave, tissue Doppler imaging (E' wave), and LA size

- Assessment of Global Longitudinal Strain (GLS) with speckle-tracking echocardiography
- Assessment of LVEF and presence and severity of LVOTO, determining peak LVOT gradient at rest and during provocation (e.g., Valsalva maneuver, exercise); use color Doppler and continuous-wave Doppler as needed
- Assessment for abnormalities in the aortic root and ascending aorta
- Ultrasound Enhancing Agents

III. When is TEE Needed?

IV. Quick Hits:

- The REMS Program for Mavacamten: Imaging Requirements (Focus on Systolic Dysfunction)
- CMR and Multimodal Imaging in HCM: A Brief Primer
- Is There a Role for Artificial Intelligence in HCM Imaging?

V. Q&A

Certified Credits:
A total of up to 2.5 contact hours and AMA PRA Category 1 Credits™ will be available for each learner, which is accepted by the American Registry of Diagnostic Medical Sonographers (ARDMS).

Experience and Capabilities

The leadership at AcademicCME has years of experience with the development and production of live and live-streamed symposia. From this experience, we have developed the following expertise:

- Selecting the most appropriate course chair and faculty to match the learning objectives, agenda, and learner audience.
- Managing the faculty to guide their activity for all aspects of the planning, development, and execution of the program.
- Implementing time-proven recruitment strategies to obtain the desired reach.
- Maintaining strict compliance with ACCME guidelines to avoid inappropriate bias.
- Providing opportunities for learner engagement through technology.
- Utilizing accurate outcomes measurements to assess the effectiveness of the symposium for each educational gap and learning objective.
- Addressing clinician skills in patient education, identifying barriers to treatment, and optimal use of the comprehensive care team.

Needs Assessment

Hypertrophic cardiomyopathy (HCM) affects 1 in 200-500 people and leads to hyper-contractility of the ventricles of the heart.[1] Cardiomyopathies, familial or sporadic, have become recognized as a leading source of cardiac morbidity and mortality. HCM is the leading cause of sudden death in young adults (Fig 1),[2] often taking an insidious clinical course. Early detection may be beneficial for prevention of significant morbidity and mortality in high-risk patients.[3]

Unfortunately, the clinical and genetic complexity of HCM and its manifold molecular mechanisms have hindered the development of effective treatment options.[7] Cardiomyopathies represent a heterogeneous group of diseases that negatively affect heart function.[8] Clinically, HCM is characterized by an asymmetric thickening of the ventricular walls and a decrease in the ventricular chamber size that occurs without a predisposing cause. In addition, fibrosis of the cardiac muscle and cardiomyocyte disarray are associated with the disease.[9] HCM is frequently accompanied by dynamic left ventricular outflow tract obstruction (LVOTO) and symptoms of dyspnea, angina, and syncope.[10] Palpitations, pre-syncope, and syncope, often due to recurrent non-sustained ventricular tachycardia, are among the cardinal clinical manifestations.[11] Arrhythmias are considered the main cause of mortality in HCM, accounting for significant morbidity and mortality worldwide.[3] Atrial fibrillation (AF) is the most important sustained arrhythmia in the HCM clinical spectrum, being sixfold more common in patients with HCM than in an otherwise age- and sex-matched general population.[12] Figure 1 illustrates the pathogenesis of AF in HCM.

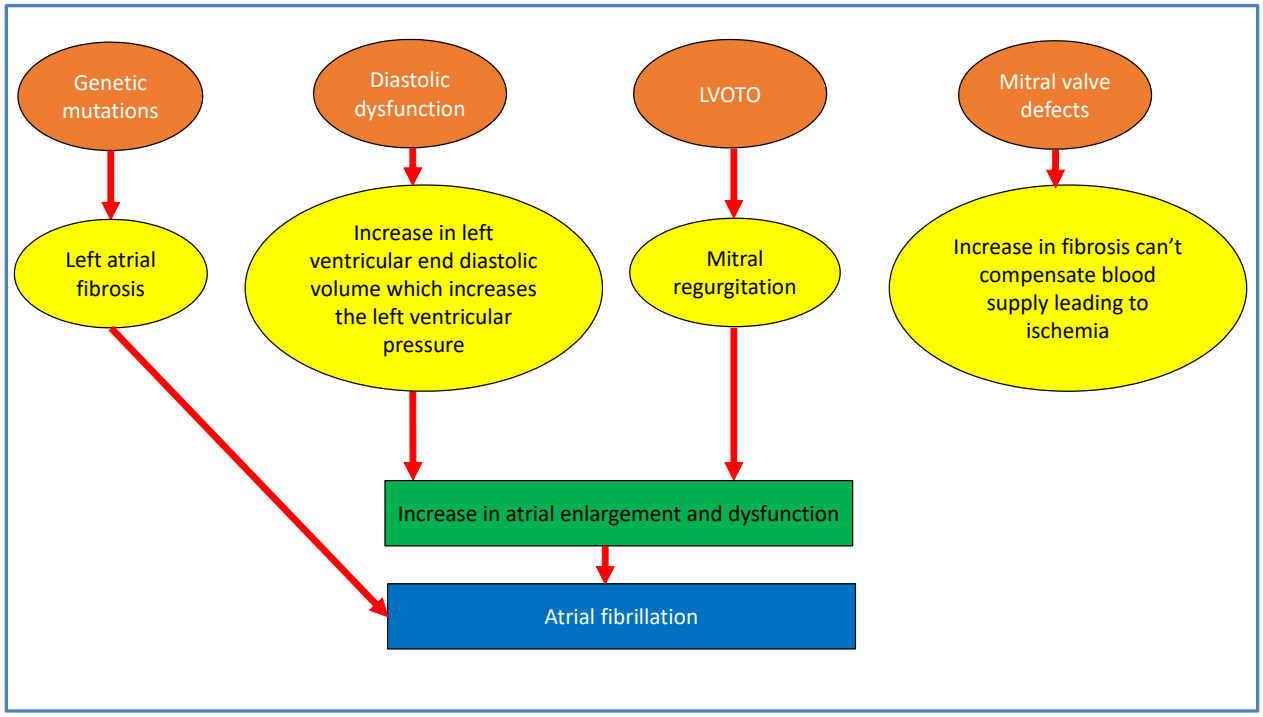


Figure 1. Pathogeneis of Atrial Fibrillation in Hypertrophic Cardiomyopathy[13]
LVOTO: left ventricular outflow tract obstruction

Pathophysiology of HCM

Mutations in genes that encode components of the sarcomere are well established as the cause of hypertrophic and dilated cardiomyopathies.[14] Increased myosin heavy chain binding and adenosine triphosphate (ATP) utilization lead to the hypercontractile sarcomere in HCM.[8] Hypertrophic cardiomyopathy is primarily caused by mutations in β-cardiac myosin and myosin-binding protein-C. Changes in the contractile parameters of myosin measured so far do not, however, explain the clinical hypercontractility caused by such mutations.[15] The first gene to be linked to HCM (*MYH7)* was identified in 1989, with its genetic repertoire expanded throughout the 1990s to include seven additional sarcomeric genes (*MYBPC3, TNNT2, TPM1, MYL2, MYL3, TNNI3, ACTC1*).[16]

Expanded access to genetic testing has facilitated identification of potential risk factors that appear through inheritance and manifest sometimes only in the advanced stages of the HCM.[8] Patients with HCM have genotypic and phenotypic variability. Indeed, a subgroup of these patients exhibits the HCM genotype but not the phenotype of left ventricular hypertrophy (LVH).[17] A shortcoming of current genetic testing for HCM is its failure to identify approximately 50% to 60% of patients with HCM. This is primarily because of the so-called “missing causal genes”. In large families in which the causal gene and its pathogenic mutation have not been identified, linkage analysis may be useful.[11] In small families or sporadic cases, either whole exome sequencing or the candidate gene approach may be required. Another limitation of genetic testing results from the great genetic heterogeneity of HCM (more than 1500 individual mutations have been described), which has prevented the genetic analysis from predicting the severity of hypertrophy or the risk of complications including SCD and heart failure.[11] However, the rare detection of multiple mutations (compound heterozygosity) by genetic testing has been associated with particularly severe hypertrophy.[11] Basic science in this disease continues to be focused on myosin structure–function in genetically engineered murine models.[18,19] However, newer alternative hypotheses to explain the basic determinants of HCM are emerging.[12] These proof-of-concept findings introduce personalized protein-protein interaction networks (reticulotypes) for characterizing patient-specific pathobiology, thereby offering a direct strategy for advancing precision medicine.[20]

Diagnosis of HCM: Focus on Imaging

Accurate diagnosis is vital for the successful management of patients with HCM. Mistakes and delays in diagnosis are common. Hypertrophic cardiomyopathy commonly "masquerades" as asthma, anxiety, mitral prolapse, and coronary artery disease. However, once properly diagnosed, patients with HCM can be effectively managed to improve both symptoms and survival.[21] Most genetically and clinically affected individuals probably remain undiagnosed, largely free from disease-related complications, although HCM may progress along 1 or more of its major disease pathways (ie, arrhythmic sudden death risk; progressive heart failure due to dynamic left ventricular outflow obstruction or due to systolic dysfunction in the absence of obstruction; or AF with risk of stroke).[22] Today, the diagnosis of HCM relies on the identification of increased left ventricular wall thickness on imaging.[23] Diagnosis is most often made using echocardiographic assessment of LVH, LVOT gradients, systolic and diastolic function, and mitral valve anatomy and function.

Comprehensive echocardiographic evaluations are recommended for the initial disease screening. As a first step for the diagnosis of HCM, careful assessment of LV wall thickness from the base to the apex is essential. LV wall thickness is recommended to be measured at end-diastole and preferably in parasternal short axis views, but it is important not to include RV trabeculations while measuring the septal wall thickness. The LV wall thickness measured at any site of the LV end-diastole is defined as maximal diastolic LV wall thickness.

The patterns of hypertrophy vary. Originally, Maron et al. proposed the following four-type classification based on the location of LV hypertrophy: type I, hypertrophy involving the basal anteroseptum; type II, hypertrophy involving the whole septum; type III, hypertrophy involving the whole septum and at least

one region among the anterior, posterior, or lateral wall; and type IV, other localization involving the posterior, apex, or lateral wall (Figure 1).[24] Although this classification is the most popular and clinically useful, the localization of the thickened wall changes depending on the cross section of the short axis view in echocardiography. Syed et al. proposed the following five-phenotype classification: type A, a predominant mid-septal convexity toward the LV cavity, with the cavity often having a crescent shape (reverse curvature septum HCM); type B, a generally ovoid LV cavity, with the septum being concave to the LV cavity and a prominent basal septal bulge (sigmoid septum HCM); type C, an overall straight septum that is neither predominantly convex nor concave toward the LV cavity (neutral septum HCM); type D, predominant apical distribution of hypertrophy (apical HCM); and type E, predominant hypertrophy at the mid-ventricular level (mid-ventricular HCM).[25] Helmy proposed yet another classification, but based on clinical presentation: pattern 1, septal hypertrophy alone; pattern 2, septum and adjacent segments' hypertrophy, but not apical hypertrophy; pattern 3, apical in combination with other LV segments' hypertrophy; and pattern 4, apical hypertrophy alone.[26] As these classifications can be used to guide treatment and also to assess prognosis, it is clear that imaging is key to early and ongoing evaluation of HCM.

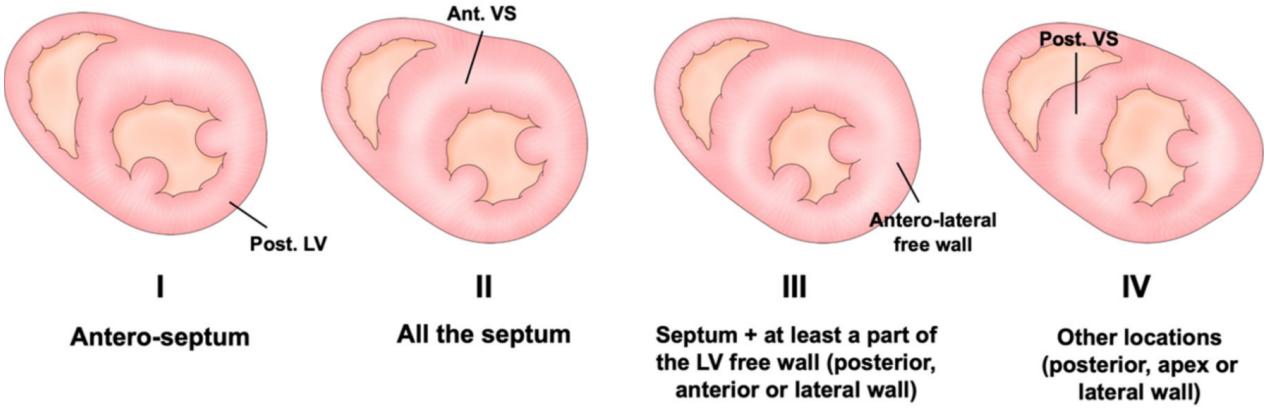


Figure 1: The classical four phenotypes of Maron's classification based on the location and degree of hypertrophy.

Optimizing Echocardiographic Imaging in HCM

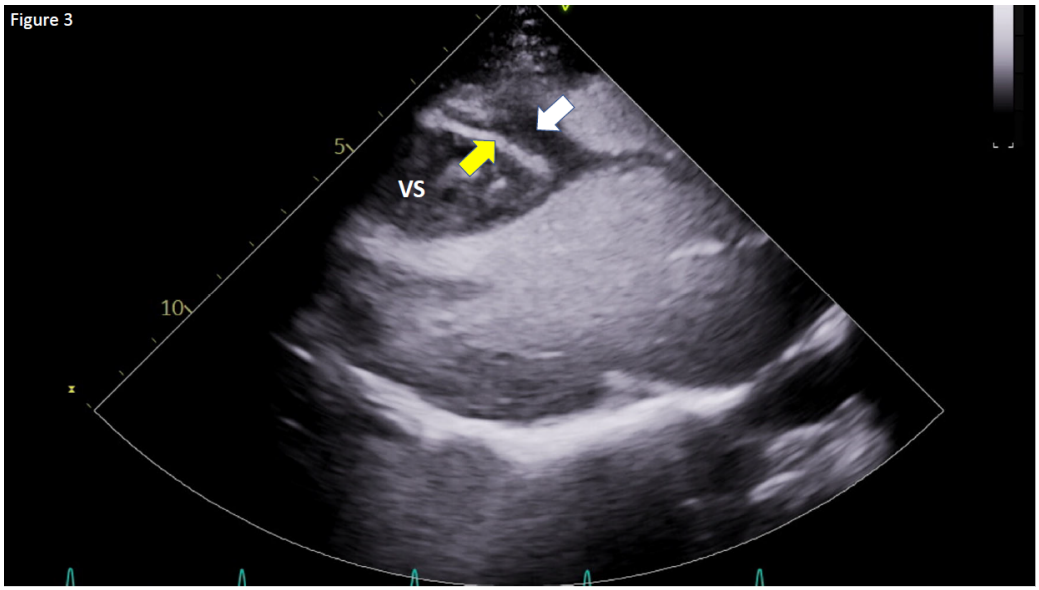
M mode: M mode echocardiography provides an "ice pick" sectional view of the heart at high frame rates.[27,28] In 1969, Dr Pravin Shah demonstrated the phenomenon of Systolic Anterior Motion of the mitral valve (SAM), followed a few years later by demonstration of asymmetric septal hypertrophy by M mode echocardiography, initially by Abbasi in 1972 and shortly thereafter by Henry in 1973.[29,30] Thus, M-mode echocardiography enabled the first non-invasive diagnosis of HCM. Septal and posterior walls that were visualized in this view allowed measurement of end-diastolic and end-systolic thickness. Others used M-mode to show the relationship between SAM and LVOT obstruction, and the absence or reduction of SAM after myectomy.

2D Echo: The introduction of 2D (B mode) echocardiography in the early 1980s allowed clinicians to gain a significantly wider perspective of cardiac anatomy in HCM.[24] Two-dimensional imaging allowed for complete visualization of all the morphologic and functional features of HCM, including ventricular wall hypertrophy, SAM, ventricular systolic function, and assessment of valves in the right ventricle. Subsequent introduction of color Doppler and spectral Doppler alerts for noninvasive quantification of LVOT gradients and qualitative and quantitative assessment of mitral regurgitation allowed finer discrimination of HCM. Doppler-derived LVOT gradients allowed clinicians to follow the severity of LVOT obstruction without the need for invasive cardiac catheterization.[31,32]

Stress echocardiography: Stress echocardiography as several advantages in the evaluation of a patient with HCM.[33,34] Earlier versions of the HCM guidelines suggested not exercising patients with high gradients. While complication rates are marginally higher in the HCM population compared to the general population undergoing stress testing, however, it appears safe and offers significant incremental information that is of value in the management of patients with HCM.[35,36] Stress echocardiography also allows the clinician to assess the exercise capacity of the patient, evaluate LVOT gradients under conditions of stress, and eventually evaluate for inducible ischemia. Since patients are mostly symptomatic with exertion, the availability of imaging during exercise is highly informative. Treadmill exercise is the most common form of exercise, but the supine bicycle is also used. Supine bicycle stress has the added advantage of being able to image the patient during the various stages of the exercise versus imaging after exercise has been terminated, as is the case with the treadmill stress.

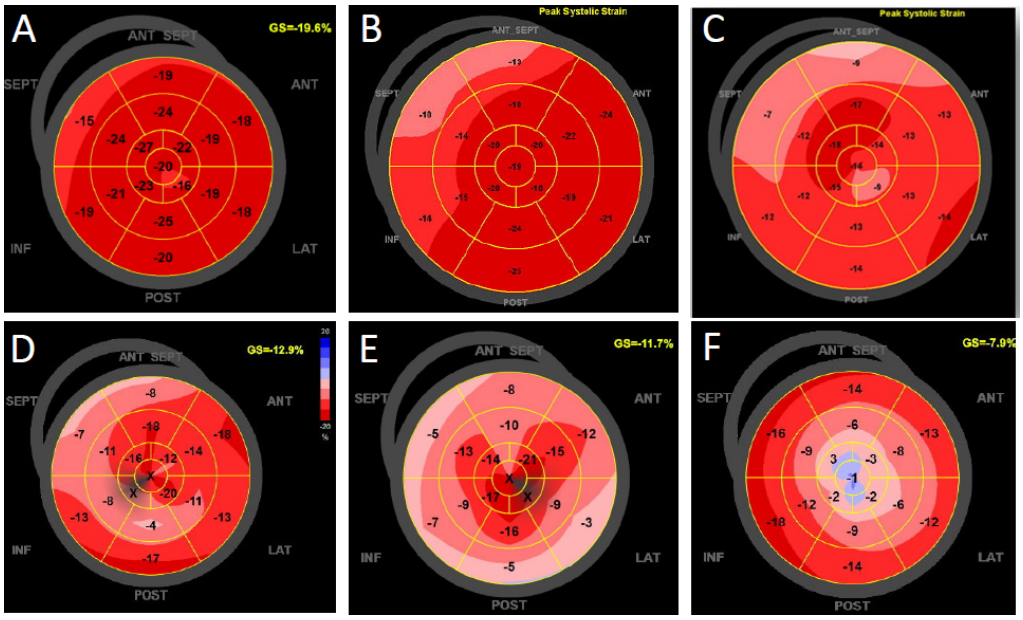
Ultrasound Enhancing Agents (UEA): Ultrasound enhancing agents or echo contrast agents have several potential uses in the evaluation of HCM patients. UEA are helpful to confirm septal wall thickness, ascertain if there are other distributions of hypertrophy (Figure 3), and are critical in evaluating apical pathologies including apical hypertrophy, apical aneurysm and detection of apical thrombus.

Figure 2: Two-dimensional echocardiographic image from the parasternal long axis view demonstrating the value of ultrasound enhancing agents for separating (yellow arrow) the true ventricular septum (VS) from a large moderator band (white arrow).



Strain echocardiography: HCM is a unique cardiomyopathy because regional and global left ventricular function are normal or hypernormal. Hence, myocardial function is not easily evaluated by the usual methodology of visual assessment. Strain is a sensitive, quantitative, and extensively validated measure of segmental/regional and global mechanics, accomplished by measuring rates and extent of segmental and global shortening and lengthening.[37] While there are regional patterns of strain abnormalities, most work has focused on measuring global longitudinal strain (GLS). There are several descriptions of patterns of strain abnormalities in various sub-types of HCM (Figure 4).[38]

Figure 4: Representative bull's-eye (polar) maps depicting left ventricular longitudinal strain using the 17-segment model. (A) normal pattern; (B) mildly reduced strain in the basal septal segments; (C) significantly reduced strain in the basal septal segments and extending into the anterior base; (D) reduce strain in the basal septal segments and extending into the mid inferior and inferolateral segments; (E) globally reduced strain with preserved strain predominantly at the apex and some mid segments; (F) reduced strain predominantly in the apical segments and extending into some of the mid-segments. Patterns of B-D represent varying degrees of septal involvement, he represents more diffuse basal and mid level involvement sparing the apex and F represents a classic apical pattern of HCM. Darker shades of prep are present with normal strength while lighter shades of red and blue represent abnormal strain.



Transesophageal echo: Transesophageal echo (TEE) offers the advantage of proximity and the lack of intervening structures, enabling high-quality images of the heart. TEE may be used in HCM when transthoracic images are of poor quality.[32] The primary advantages may be in assessing basal septal wall thickness, presence or absence of SAM, mitral and aortic valve abnormalities, papillary muscles and chordal structures, presence of a subaortic membrane/ring, and LVOT gradients. It may not be optimal in assessing the left ventricular apex in some patients, and LVOT gradients may be artifactually reduced due to hemodynamic state and use of sedation or anesthesia. TEE is also critical for either pre-operative planning and/or intraoperative assessment during surgical septal myectomy. Intraoperatively, TEE is used to assess the point of maximal septal wall thickening from the aortic valve and to ascertain the medial and lateral extent of hypertrophy. It also allows assessment for presence of hypertrophied, supernumerary, or apically-displaced papillary muscles and/or anomalous chords. Any of these morphologic features may also contribute to LVOT obstruction and may need to be addressed during surgery. TEE allows for assessment of primary mitral valve abnormalities and severity of mitral regurgitation prior to myectomy, and of presence and severity of residual mitral regurgitation post-myectomy. Lastly, it is used to assess presence of residual LVOT gradient after the patient is taken off bypass. This assessment may be done either in the resting state or after inducing a premature ventricular beat mechanically using a forceps, or after infusion of isoproterenol.

2024 AHA/ACC/AMSSM/HRS/PACES/SCMR Guideline for the Management of Hypertrophic Cardiomyopathy: Competencies in Echocardiography

The recently updated "American" guidelines highlight the importance and the utility of echocardiographic imaging in the diagnosis and management of HCM (Figure 5, below). While all aspects of these recommendations cannot be addressed in a single program, the key guidance from within this list is will be provided to meet the Learning Objectives of the proposed program. The essential role of echocardiography in HCM can be summarized as follows:

Echocardiography is a critical tool in the diagnosis and management of HCM due to its ability to provide detailed information about heart structure and function. Here are the key points highlighting its importance:

1. Diagnosis

Identification of Hypertrophy

- **Left Ventricular Hypertrophy (LVH):** Echocardiography can measure the thickness of the ventricular walls and septum, which is crucial for diagnosing HCM. Asymmetric septal hypertrophy is a hallmark of the condition.

Morphological Assessment

- **Assessment of Structural Abnormalities:** Echocardiography helps identify structural abnormalities such as mitral valve abnormalities, outflow tract obstruction, and papillary muscle displacement.

Evaluation of Systolic Function

- **Global and Regional Wall Motion:** Echocardiography assesses both global and regional systolic function, which can be abnormal in HCM.

2. Management

Risk Stratification

- **Outflow Tract Gradient Measurement:** Measuring the pressure gradient in the left ventricular outflow tract is essential for determining the severity of obstruction and guiding treatment decisions.

Monitoring Disease Progression

- **Serial Echocardiograms:** Regular echocardiographic evaluations help monitor the progression of HCM, including changes in ventricular wall thickness, cavity size, and outflow tract gradient.

Assessment of Complications

- **Detection of Left Atrial Enlargement:** Echocardiography can detect left atrial enlargement, which is associated with an increased risk of atrial fibrillation and other complications.
- **Identification of Diastolic Dysfunction:** Diastolic dysfunction is common in HCM and can be evaluated using Doppler echocardiography.

3. Guiding Treatment

Surgical and Interventional Planning

- **Myectomy and Alcohol Septal Ablation:** Echocardiography is used to plan and guide surgical procedures such as septal myectomy and non-surgical interventions like alcohol septal ablation.

Monitoring Therapy Effectiveness

- **Pharmacologic Therapy:** The effectiveness of medications aimed at reducing outflow tract obstruction and alleviating symptoms can be assessed through changes observed on echocardiography.

Device Therapy

- **Pacemakers and ICDs:** Echocardiography aids in the placement and monitoring of pacemakers and implantable cardioverter-defibrillators (ICDs) in patients at risk of sudden cardiac death.

4. Genetic and Family Screening

Family Screening

- **Identifying Asymptomatic Relatives:** Echocardiography is crucial in screening first-degree relatives of patients with HCM to identify those who may have the condition but are asymptomatic.

5. Non-Invasive and Accessible

Non-Invasive Nature

- **Safety and Accessibility:** Echocardiography is non-invasive and widely accessible, making it a safe and practical tool for repeated assessments without exposing patients to ionizing radiation.

In summary, echocardiography is indispensable in the comprehensive evaluation, management, and follow-up of patients with hypertrophic cardiomyopathy. It provides critical insights into the disease's anatomical and functional aspects, guides therapeutic decisions, and helps monitor disease progression and treatment outcomes.

(Note: Figure 5 is on following page)

Artificial Intelligence in HCM Imaging: A Growing Potential

The use and application of AI is a rapidly expanding field in all modalities of diagnostic imaging to improve consistency and reduce interobserver variability, including in the evaluation of HCM. Accurate echocardiographic interpretation is largely dependent on the subjective knowledge of the operator, and is burdened by the high dependence on the level of experience of the operator, to a greater extent than other imaging modalities such as computed tomography, nuclear imaging, and magnetic resonance imaging. AI technologies offer new opportunities for echocardiography to produce accurate, automated, and more consistent interpretations.[39]

AI may also supplement the role of echocardiography in distinguishing among HCM phenocopies. For example, a deep learning algorithm for the differential diagnosis of common left ventricular hypertrophy etiologies (hypertensive heart disease, HCM, and AL-cardiac amyloidosis) was constructed to classify the three diagnoses using five standard echo views (parasternal long-axis, parasternal short-axis, apical four-chamber, apical two-chamber, and apical three-chamber). The study population included a training (n = 620), a validation (n = 155), and a test cohort (n = 155). In the test cohort, the area under the curve (AUC) for the AI model was 0.962 for hypertensive heart disease, 0.982 for hypertrophic cardiomyopathy, and 0.996 for AL-cardiac amyloidosis. The overall diagnostic accuracy was significantly higher for the deep learning algorithm than for participating echocardiography specialists, therefore supporting that the use of AI can improve the diagnostic process in patients with left ventricular hypertrophy.[40]

The proposed program presents an interesting opportunity to initiate discussions about the potential role of AI in HCM-targeted cardiac sonography moving forward.

A complete list of the needs assessment references can be found in the Appendix.

Recommendations for Echocardiography Referenced studies that support the recommendations are summarized in the Online Data Supplement.		
COR	**LOE**	**Recommendations**
1	B-NR	1. In patients with suspected HCM, a transthoracic echocardiogram (TTE) is recommended in the initial evaluation.[1–6]
1	B-NR (children) C-LD (adults)	2. In patients with HCM who have no change in clinical status or events, repeat TTE is recommended every 1 to 2 years to assess the degree of myocardial hypertrophy, dynamic LVOTO, MR, and myocardial function (Figure 1).[7–14]
1	B-NR	3. For patients with HCM who experience a change in clinical status or a new clinical event, repeat TTE is recommended.[9,14–17]
1	B-NR	4. For patients with HCM and resting peak LVOT gradient <50 mm Hg, a TTE with provocative maneuvers is recommended.[18–21]
1	B-NR	5. For symptomatic patients with HCM who do not have a resting or provocable outflow tract peak gradient ≥50 mm Hg on TTE, exercise TTE is recommended for the detection and quantification of dynamic LVOTO.[20–25]
1	B-NR	6. For patients with HCM who are undergoing surgical septal myectomy, intraoperative transesophageal echocardiogram (TEE) is recommended to assess mitral valve anatomy and function and adequacy of septal myectomy.[26–29]
1	B-NR	7. For patients with HCM who are undergoing alcohol septal ablation, TTE or intraoperative TEE with intracoronary ultrasound-enhancing contrast injection of the candidate's septal perforator(s) is recommended.[30–34]
1	B-NR	8. For patients with HCM who have undergone SRT, TTE within 3 to 6 months after the procedure is recommended to evaluate the procedural results.[35–38]
1	B-NR	9. Screening: In first-degree relatives of patients with HCM, a TTE is recommended as part of initial family screening and periodic follow-up (Figure 1, Table 7).[3–5,7,14,32]
1	B-NR	10. Screening: In individuals who are genotype-positive, phenotype-negative, echocardiography is recommended at periodic intervals depending on age (1-2 years in children and adolescents, 3-5 years in adults) and change in clinical status (Figure 1, Table 7).[39–43]
2a	C-LD	11. For patients with HCM, TEE can be useful if TTE is inconclusive in clinical decision-making regarding medical therapy, and in situations such as planning for myectomy, exclusion of subaortic membrane or MR secondary to structural abnormalities of the mitral valve apparatus, or in the assessment of the feasibility of alcohol septal ablation.[26–29]
2a	B-NR	12. For patients with HCM in whom the diagnosis of apical HCM, apical aneurysm, or atypical patterns of hypertrophy is inconclusive on TTE, the use of an intravenous ultrasound-enhancing agent is reasonable, particularly if other imaging modalities such as CMR are not readily available or are contraindicated.[44,45]
2a	C-LD	13. For asymptomatic patients with HCM who do not have a resting or provocable outflow tract peak gradient ≥50 mm Hg on standard TTE, exercise TTE is reasonable for the detection and quantification of dynamic LVOTO.[15,19,20,22–25]

Figure 5. From reference 4.

Faculty Selection Process

Faculty will be selected based on the faculty's commitment to education and expertise. Any conflict of interest will be resolved through AcademicCME's conflict of resolution policies and procedures. We employ a rigorous process that identifies, vets, selects, and confirms faculty members to participate in our continuing medical education activities.

Potential faculty members are initially identified and screened based on the following criteria:

- relevant practice experience in the topic area
- current and past teaching positions
- familiarity with the activity topic
- published articles
- experience as a presenter for continuing medical education events

Assurance of Balanced, Unbiased, and Fair Content

This project is planned and will be executed in accordance with ACCME Essential Areas and Elements, including the Standards for Commercial Support. Potential conflicts of interest will be identified for all planning committee and faculty members and resolved through AcademicCME's conflict of interest resolution policy. All material will be independently peer reviewed. Educational materials will be developed in accordance with all regulations, guidelines, standards, and codes that apply to independent education, including — but not limited to — ACPE, OIG Compliance Program Guidance for Pharmaceutical Manufacturers and PhRMA Code on Interaction with Health Care Professionals

Accreditation

JOINT ACCREDITATION™
INTERPROFESSIONAL CONTINUING EDUCATION

In support of improving patient care, AcademicCME is jointly accredited by the Accreditation Council for Continuing Medical Education (ACCME), the Accreditation Council for Pharmacy Education (ACPE), and the American Nurses Credentialing Center (ANCC), to provide continuing education for the healthcare team. AcademicCME will apply to EACCME for European accreditation of this program. Learners' certificates will be customized by region to facilitate appropriate documentation of credit.

Standards for Commercial Support

AcademicCME complies with the ACCME Standards for Commercial Support and ensures that the following decisions are made free of the control of any commercial interest:

- Identification of needs

- Determination of educational learning objectives
- Selection and presentation of content
- Management of faculty conflict of interest on an individual basis
- Selection of educational method
- Evaluation of the activity

About AcademicCME

Dedicated to Improving Patient Outcomes

AcademicCME is a joint accredited healthcare solutions medical education company designed to impact practice behavior and thus, patient outcomes. AcademicCME leadership draws on decades of clinical experience of its staff healthcare providers in clinical practice and education which provides a unique understanding of the dynamics between healthcare team members behavior, patient care and practice based educational needs.

Focused on Bridging Knowledge Gaps

AcademicCME's focus is relaying the latest evidence-based research from clinical trials to the bedside. Its unique peer-to-peer relationship with top thought leaders and leading educational institutions allows it to keep a finger on the pulse of gaps in knowledge, competence, and performance in many therapeutic areas. The mission is to develop individualized continuing education initiatives by first assessing the needs of the healthcare providers, and then tailoring educational programming to specifically meet these needs. All work remains centered on the goal of improving patient outcomes.

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