The Use of Echocardiograms in Preparticipation Examinations

Caroline Lucas1; Deanna L. Kerkhof, MPH1; Jacilyn E. Briggs1; and Gianmichel D. Corrado, MD1,2,3,4

Abstract
Sudden cardiac death (SCD) is the leading cause of death during exercise in young athletes. Preparticipation physical examinations were developed to maintain the health and safety of athletes, including the prompt identification of those at risk for SCD. The use of medical history and physical examinations, electrocardiography, and echocardiography, or some combination thereof, is the source of continued debate. This article provides an overview of the etiology of SCD and reviews literature relating to preparticipation echocardiography, with a focus on its evolution, utility, and effectiveness. The limited echocardiogram is a potentially viable screening option yet to be thoroughly explored by experts and policymakers in the sports medicine community.

Introduction
Sudden cardiac death (SCD) is characterized as any unexpected death due to cardiac causes that occurs within 1 h of symptom onset (6,21,23). The incidence of SCD is estimated at 1 in 40,000 to 1 in 80,000 young athletes (≤35 yr of age) and is the leading cause of death during physical activity in this age group (18). The risk of SCD is higher in male athletes, black athletes, and basketball players. A recent review of the U.S. SCD registry found SCD to be five times more frequent in minority athletes than in white athletes (25). Additionally, more than 50% of the SCD caused by hypertrophic cardiomyopathy (HCM) occurred within minority populations (25). HCM and other structural cardiac abnormalities are responsible for most sudden deaths in autopsy-confirmed cases; however, Harmon et al. (17,25) found autopsy-negative sudden unexplained death (ANSUD) to be the leading cause of SCD in their 2015 review of National Collegiate Athletic Association (NCAA) athletes. Identifying athletes at risk for SCD through preparticipation physical examinations (PPE) could prevent the sudden unexpected loss of a seemingly fit and healthy young athlete.

The screening method used to identify athletes with an increased risk for SCD continues to be debated among the sports medicine community internationally. The American Heart Association (AHA) recommends a 14-element focused history and physical examination (H&P) despite research documenting its low sensitivity and specificity for detecting cardiovascular abnormalities (26). In Europe, a 12-lead electrocardiogram (ECG) supplements the traditional H&P. A 25-yr study evaluated the cost-effectiveness of screening ECGs in Italy and concluded the ECG “provided adequate sensitivity and specificity for detection of potentially lethal cardiomyopathy and arrhythmias” (8). The AHA has cited concerns about high false-positive rates with ECG screening, as well as inadequate infrastructure and access to care for socioeconomically disadvantaged athletes (1).

Government agencies continue to encourage the pursuit of a cost-effective solution to the cardiac screening dilemma (1). Refined ECG interpretation methods, which exclude normal findings consistent with cardiac changes in the athlete’s heart, have garnered significant attention and support. These methods — the Seattle Criteria (5,11) and the Refined Criteria (35,40) — have reduced false-positive results. Despite fewer false-positives, consensus groups assigned with the task of determining the appropriateness of ECG screening interpreted through revised methods maintain H&P as the standard for screening (12,16). PPE echocardiography (ECHO) is another potential screening option; however, it has not “been considered seriously as a primary cardiac imaging strategy or modality for large-scale universal PPE screening because of impracticality, cost, and interobserver variability” (24). Novel research investigates a new approach to PPE ECHO involving limited preparticipation ECHO performed by frontline providers (FLP) (i.e., physicians and physician assistants in internal medicine, family medicine, emergency medicine, and pediatrics). This approach may be a viable screening option that harbors the ECHO’s advantages for the clinical diagnosis of conditions, such as HCM, also while controlling costs (24,45).

1Northeastern University, Boston, MA; 2Division of Sports Medicine, Department of Orthopedics, Boston Children’s Hospital, Boston, MA; 3Harvard Medical School, Boston, MA; and 4The Micheli Center for Sports Injury Prevention, Waltham, MA

Address for correspondence: Gianmichel D. Corrado, MD, Boston Children’s Hospital, 319 Longwood Avenue, Boston, MA 02115; E-mail: Gianmichel.Corrado@childrens.harvard.edu.

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Focused ECHO. With POCU, FLP can perform time-sensitive assessments of cardiac structure and function. Studies have shown that cardiac features can be accurately identified and analyzed by non-specialists using portable ultrasound with minimal training. These features include: LV size, thickness, and function; valve calcification and regurgitation; presence of pericardial effusion; aortic root size; right ventricle (RV) size and function; and, coronary artery origin. Several cardiac conditions can be recognized by an FLP with portable ultrasound as an adjunct; although it is important for providers to recognize the limitations of portable ECHO by a FLP (PEFP).

Portable devices have lower-quality dynamic imaging and fewer capabilities than standard ultrasound devices or other imaging modalities such as cardiac MRI. Additionally, cardiologists are uniquely qualified to detect and diagnose cardiac abnormalities. If PEFP is unclear or suspicious, referral to a cardiologist is essential. Nevertheless, when limitations are acknowledged and respected, portable ultrasound can be a very useful tool for assessments and screenings.

Several studies have demonstrated the benefits of focused cardiac ECHO in frontline settings, including emergency departments and intensive care units; however, very few sports medicine providers use ultrasound for cardiac assessments. Typically, sports medicine clinicians use ultrasound for musculoskeletal diagnosis; although use can be extended beyond the musculoskeletal system. A handful of sports medicine physicians now use POCU for cardiac assessment during preparticipation examinations.

Utility of Preparticipation Echocardiography

PEFP can be used by sports medicine physicians to identify several structural and functional abnormalities indicative of conditions that can cause SCD in athletes. Its capabilities are particularly useful in cases where ECG patterns and/or H&P results are indefinite or “borderline” (5,11,40). The 2014 Refined Criteria group ECG variants into three categories: normal training variants, borderline variants, and training unrelated variants. Borderline variants include left and right atrial enlargement, left and right axis deviation, RV hypertrophy, and T-wave inversion in leads V1 to V4 (40). Physicians using the Refined Criteria have found it to be useful in reducing the number of false-positive ECGs in athlete populations (35,40). Although it is important to note that the number of false-positives from ECG screening is strongly influenced by physician experience (35). With PEFP, providers can validate or dismiss the presence of several abnormalities with direct visualization and further reduce the number of false-positives from ECG screening of athletes (15,39,47).

Additionally, PEFP can serve as a preventative measure for false-negatives. The adoption of athlete-specific criteria essentially dismisses ECG pattern abnormalities. For example, “normal” ECG patterns in athletes include a convex ST segment elevation with T-wave inversion in leads III, AVR, and V1. However, an ECG surface pattern with T-wave inversion also is seen in patients with arrhythmogenic right ventricular cardiomyopathy and HCM. With PEFP, providers can validate or dismiss the presence of such abnormalities with direct visualization (15,39,41,43,46).

Recent findings have shown that exercise-induced cardiac adaptation is not absolute (7,9,34,41,43,48). A recent study explored cardiac remodeling in professional Italian soccer players. Findings confirmed fluctuations in cardiac structure...
throughout the season, but found that LV mass changes were associated with changes in fat-free body mass (9). Beaudry et al. (4) examined determinants of cardiac remodeling in athletes. Their research found that intensity, duration, and frequency of training were the largest determinants of structural cardiac changes. This contrasts previously accepted research indicating that exercise-induced cardiac changes were largely dependent on the type of exercise (static or dynamic) (4). The aforementioned studies highlight the variance of cardiac adaptation in athletes, which manifest as variance in ECG examinations. It is important to consider these and similar studies when using athlete-specific ECG criteria for screening purposes. The merits of revised ECG criteria are being explored, but physicians must maintain that cardiac remodeling is not dichotomous; a grey area exists between pathology and physiology. It is a complex biological process that is affected by other environmental and genetic factors, and cardiac changes vary from athlete to athlete (4,9,41,43).

ECHO can appreciably help physicians with cardiac screening in athletes, particularly in cases where exercise-induced cardiac adaptations may be influenced by athlete size, race, or specialty or in cases where ECG patterns fall under borderline variant criteria (13,17,28,39,43,44,46).

The screening protocol followed by physicians differs from institution to institution. Yim et al. (45) used PEFP to obtain measurements useful for differentiating pathology from athlete physiology in borderline ECG variants (see Table). PEFP aims to augment the utility potential of the H&P and ECG with a direct visualization of the size, structure, and function of the heart; however, PEFP does not replace the traditional ECHO. If abnormalities on ECG and limited ECHO are indicative of a cardiac condition, a cardiac consultation and full ECHO are warranted (3,14,43). Other physicians use a standard transthoracic ECHO protocol to image and measure more cardiac features (see Table); however, for noncardiologists, accurately recognizing abnormalities with standard protocol takes a significant amount of training. Both limited and standard ECHO can image structural and functional abnormalities responsible for SCD, including the size of the atria and ventricles, thickness of the walls between chambers, and end-diastolic dimensions.

**Etiology of SCD**

There continues to be considerable debate as to what causes SCD in competitive athletes. Postmortem studies show distinct age-related differences in the etiologies attributed to

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**Table. Measurements obtained with limited and standard ECHO.**

<table>
<thead>
<tr>
<th>Standard ECHO measurements (EAE protocol) (11,48)</th>
<th>Limited ECHO measurements (PEFP) (1)</th>
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<tr>
<td><strong>LV Dimensions:</strong></td>
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<tr>
<td><strong>LV Internal Dimensions</strong></td>
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<tr>
<td>Diastolic Dimension (mm)</td>
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<td>Systolic Dimension (mm)</td>
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<tr>
<td><strong>LV Volumes (Biplane)</strong></td>
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<td>LV EDV (mL)</td>
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<td>LV ESV (mL)</td>
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<td><strong>LV Volumes Normalized by BSA</strong></td>
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<td>LV EDV (mL$^{-2}$)</td>
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<td>LV ESV (mL$^{-2}$)</td>
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<td>LV EF (biplane)</td>
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<tr>
<td>M-Mode Diameters (End Diastolic to End Systolic) or 2D&quot;Guided&quot;</td>
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<tr>
<td>Septum and Posterior Wall Thickness</td>
<td>AO root</td>
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<tr>
<td>LV Ejection Fraction: Volume-Based Quantitation Advisable</td>
<td>Asc AO</td>
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<td>LV Regional Wall Motion Abnormalities: From 1 (Normal) to 4 (Dyskinetic)</td>
<td>Ratio of IVSWd to LVPWd</td>
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<td>Left Atrium: at Least Two Orthogonal Diameters, Preferably Volume</td>
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<tr>
<td>RV: Size (Normal or Dilated)</td>
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<tr>
<td>RV Systolic Function: (Normal, Depressed: Mild, Moderate, Severe)</td>
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<td>Right Atrium: Size (Normal or Dilated)</td>
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<td>Aortic Root: Maximum Diameter at Sinus Level</td>
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<tr>
<td>Mitral Valvular Area Planimetry</td>
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</tbody>
</table>

*AO root, aortic root; Asc AO, ascending aorta; BSA, body surface area; EAE, European Association of Echocardiography; EDV, end-diastolic volume; ESV, end-systolic volume; IVSWd, interventricular septum at end diastole; LVPWd, left ventricular posterior wall end diastole.*
SCD in U.S. athletes. According to Myerburg et al. (12,17,41,43), the leading pathology of SCD in trained U.S. athletes older than 35 yr is athlosclerotic coronary artery disease (CAD), responsible for over 70% of SCD. Studies suggest that the mechanism of SCD in athletes with CAD is the disruption of large plaques triggered by intense physical activity and subsequent ischemia (43). Although the risk of SCD during exercise is transiently increased for individuals with CAD, the health benefits of habitual exercise are undeniable and far-reaching. Several studies have explored the relationship between habitual exercise and cardiovascular diseases, consistently demonstrating that habitual exercise serves as a protective measure from these diseases. In addition, these studies show that habitual exercise can treat CAD and other chronic diseases (1,16–18,27).

The leading cause of medical death in exceptionally fit NCAA athletes is SCD (1,16,17,19). The incidence estimates of SCD in the general young athlete population are highly variable, but a high risk of SCD in NCAA athletes is acknowledged. In young athletes, most SCD are caused by ventricular tachyarrhythmias and subsequent hemodynamic collapse. SCD that occur in young athletes are usually the consequence of an undiagnosed genetic or congenital cardiac condition. Intense exercise can trigger ventricular tachyarrhythmia in structurally abnormal or electrically unstable hearts, resulting in SCD (17,43).

Structural cardiac conditions known to cause SCD in young athletes include cardiomyopathies, congenital heart defects, conduction-related disorders, and acquired cardiac diseases. The most common cause of SCD in young athletes is debated, although studies indicate that cardiomyopathies are prevalent in NCAA athletes. Cardiomyopathies are cardiac conditions that enlarge, thicken, or stiffen the myocardium. Dilated and restrictive cardiomyopathy is rarely a cause of SCD in competitive athletes due to a low incidence in the general population and performance-limiting dyspnea and fatigue frequently associated with these conditions. Arrhythmogenic right ventricular cardiomyopathy (ARVC) and HCM are the most common cardiomyopathies in young athletes (12,17,18,41,43).

ARVC is a genetic cardiac condition with a prevalence of 1:5000 in the general population (39,41,43). Estimated incidence in athletes ranges from 2% to 10%. It is associated with fibrofatty replacement of myocardium in the RV. Progressive myocardial loss leads to myocyte death. Sports participation with ARVC increases the risk of ventricular tachyarrhythmia and progression of the disease. Individuals with this condition are advised not to participate in moderate- or high-intensity exercise. Specific functional, structural, and electrophysiological abnormalities are used to identify ARVC. ECG patterns indicative of ARVC include abnormal depolarization, epsilon waves, QRS widening, and inverted T waves on precordial leads. Abnormal function and volume of the RV on ECHO can also be used to confirm ARVC and help to distinguish from exercise-induced ventricular remodeling (43).

HCM has a higher prevalence in the general population than ARVC, with an estimated incidence rate of 1:500 in the general population (17,29,43). In a recent meta-analysis of U.S. studies from 1990 to 2014, HCM was found to be the cause of 10.3% SCD in young athletes; although, incidence estimates range from 8% to 15%. HCM is a genetic condition characterized by asymmetric LV hypertrophy at the expense of the LV cavity (4,7,17,29,34,43). Phenotypic expression of HCM is variable, with LV hypertrophy ranging from mild to severe. Most people with HCM are asymptomatic, but in some cases, chest pain, dyspnea, and syncope may be present. ECG are abnormal in up to 90% of patients, but the patterns are not specific. Certain ECG patterns common to HCM are conduction delay, T wave inversion, and pathological Q waves. Phenotypic features of HCM can be viewed on ECHO. As discussed above, these include symmetric hypertrophy, septal anterior movement, reduced LV cavity size, and reduced diastolic function (4,7,29,34).

Coronary artery anomalies (CAA) not associated with atherosclerosis are responsible for up to 17% of SCD in young athletes (17,29,43); although a study only on NCAA athlete SCD from 2003 to 2013 found CAA to be the cause of 11% of deaths. The most common type of CAA is anomalous origin of a coronary artery. CAA can occlude blood flow through the coronary arteries and result in subsequent ischemia (29,43). CAA frequently manifest as SCD in children or young athletes immediately after exercise. CAA can be detected with ECHO, although a proper diagnosis requires a coronary angiography, cardiac MRI, or cardiac CT.

Myocarditis is a prominent cause of SCD in young athletes. In NCAA athletes, myocarditis has been attributed to 9% of deaths (17); although, estimates of incidence rate in all young athletes range from 3% to 13% (27,43). It is an inflammation of the myocardium caused by allergens, drugs, and autoimmune reactions, although a viral infection is the most common cause of inflammation in young athletes. Inflammation of the myocardium can result in cardiac enlargement and interruption of electrical order in the heart. This can lead to ventricular fibrillation and subsequent SCD. Risk of SCD from myocarditis can be reduced if the condition is recognized before sports participation. Research shows exercise may worsen the inflammation and systemic infection. ECG patterns are typically abnormal in myocarditis, but ECG is not sensitive nor specific for this condition. ECG findings may contribute to the diagnosis and ECHO can be used to image enlargement abnormalities and to rule out noninflammatory causes, though a definitive diagnosis requires a biopsy.

Reports of SCD in young athletes with no morphological substrate range from 10% to 30% (3,17,27,29,43). These SCD also are referred to as autopsy negative. ANSUD are typically recognized as general cardiac arrhythmias, although some electrical conditions can be diagnosed postmortem through genetic testing. Conduction-related diseases are typically inherited. These diseases present as abnormal ECG patterns, but the heart appears structurally normal. The incidence estimates for these disorders range significantly due to the ambiguity of ANSUD. These conditions include long QT syndrome (LQTS), catecholaminergic polymorphic ventricular tachycardia (CPVT), Brugada syndrome (BrS), and Wolff-Parkinson-White (WPW) syndrome.

LQTS is an ion channelopathy with a prevalence of 1:2000 for the general population. This syndrome is a leading cause of conduction-related SCD and is characterized by a QTc interval longer than 460 ms (29,42,43). The long QT interval is a consequence of prolonged repolarization or depolarization of the myocardium, which can lead to arrhythmias, including ventricular fibrillation and torsades de pointes.
Repolarization abnormalities are due to mutations in several genes that encode for sodium and potassium channels. The risk of SCD in LQTS is dependent on the duration of QT interval prolongation. LQTS diagnosis is made based on long QT ECG patterns; however, additional blood tests are required to rule out secondary causes (29).

CPVT is another conduction-related condition known to cause SCD. CPVT is one of the most severe cardiac conditions. Undiagnosed CPVT has a mortality rate of 30% to 40% by age 40, though the prevalence is not established. Estimates of young athlete SCD from CPVT range from 5% to 20% (17, 25, 29, 43). This disorder affects calcium release and interferes with myocardial repolarization. As consequence, arrhythmias, including ventricular tachycardias and fibrillation, occur when catecholamines are released in response to emotional or physical stress. Diagnostic criteria for CPVT are a structurally normal heart and normal resting ECG, with exercise-induced tachycardia (29).

BrS is an inherited condition with an estimated prevalence in the United States of 1:10,000, though the prevalence is higher in Southeast Asian populations (17, 29, 43). BrS is caused by over 300 gene variants, which each encode different ion-channels. ECG abnormalities characteristic of BrS are right bundle branch block, ST segment elevation, and polymorphic ventricular tachycardia. Recent studies indicate that minor right ventricular abnormalities may be present with three-dimensional ECHO and cardiac MRI (29, 43).

WPW is another rare conduction disorder characterized by an extraneous electrical pathway between the atria and ventricles of the heart. Studies report a prevalence of 1:1000 individuals although it is responsible for up to 1% of SCD in athletes. Researchers suspect that this may be higher when the number of ANSUD is considered (17, 27, 29). Most individuals with WPW have normal heart anatomy, and WPW is diagnosed with ECG criteria. The most common clinical manifestation of WPW is supraventricular tachycardia. On ECG, WPW is characterized by a short PR interval <120 ms and delta wave (29, 43).

Effectiveness of PPE ECHO

ECHO has been shown to be an effective screening modality for the conditions discussed above, particularly when it is used as part of a multimodal approach (3, 9, 14, 21, 45). Specifically, ECHO increases PPE specificity and reduces the number of false positive ECG readings, often the consequence of similarities between HCM and athlete’s heart (14, 21, 45). Researchers have yet to come to a consensus on a singular PPE ECHO protocol that should be used. Irrespective of protocol, a multimodal imaging approach with ECHO can refute positive H&P and ECG tests. Additionally, other studies have found ECHO to be more sensitive than H&P and ECG alone. In a study of 3100 male soccer athletes, the referral rate for HCM was reduced by 33% and FLP obtained measurements statistically similar to those of formal ECHO. A reduced referral of 33% demonstrates that PPE ECHO could be a feasible, cost-effective method. This contrasts a major criticism of ECHO cost ineffectiveness cited by the AHA. The cost ineffectiveness of universal ECG screenings is well supported by research, but addition of ECHO as an adjunct to the current PPE protocol could significantly reduce the cost (14, 16, 21, 35, 40, 45). Additionally, PEFP also may be the most cost-effective modality for PPE cardiac screening based on time-driven, activity-based costing models recently introduced into health care delivery (19). Studies in resident and physician populations show training for FLP can be accomplished in as few as two (21). If ultrasound training of FLP continues to expand and improve, PEFP has the potential to become an even more effective screening modality.

Future Research

The AHA statement aims to produce the greatest good for the entire population, the Seattle Group strives to improve the efficacy of the ECG, and the Northeastern Group will continue to investigate the utility of incorporating PEFP. PEFP has shown that it can effectively identify structural cardiac abnormalities that are suggestive of potentially fatal cardiac abnormalities. Although, to date, only two studies have explored PEFP, and more research is needed to determine its utility and feasibility on a larger scale. In addition, future studies should continue to investigate new ultrasound and medical imaging technologies that could improve PEFP.

Machine-learning technology may further expand the use of ECHO as a screening and diagnostic modality (22, 32, 37). Thousands of data points are produced during ECHO imaging, but only a fraction of these are interpreted by clinicians for diagnostic purposes (37). With machine-learning techniques, these data points can be interpreted and analyzed for predictive accuracy of cardiac conditions. Data points with the highest predictive accuracy for detecting pathologies can then be used to develop cognitive machine-learning algorithms. These algorithms can be used during ECHO imaging as a diagnostic adjunct, by highlighting abnormalities for
physicians to investigate further. Cognitive computing machine learning has the potential to improve diagnostic accuracy for novice and non-specialist providers, reduce interoperator variability, and decrease medical costs (10).

The number of medical images and patient records available to physicians will continue to increase with the further development of cloud storage and open access data. High volumes of medical data can be used by research teams across the globe to create more accurate machine-learning algorithms. The integration of big data and cognitive machine learning would enable the development of precision phenotyping for cardiac conditions; these technologies could use cloud-stored data to compare a patient’s history and imaging results with data from thousands of patients who have similar medical history and cardiac imaging.

Obvious challenges exist for transitioning to big data and cognitive machine-learning, but these appear to be outnumbered by potential benefits. The AHA has expressed interest in big data and medical innovation for cardiovascular imaging with the recent development of a cloud-based precision medicine platform through Amazon Web Services (2). Big data and cognitive machine-learning have the potential to transform medicine by revolutionizing screening and diagnostic methods. Research should continue to explore PEFP, because the future of ECHO shows promise for improving cardiovascular health and preventing SCD.

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13. FIFA Pre-Competition Medical Assessment (PCMA), 2009.


