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ECHOCARDIOGRAPHY (JM GARDIN AND AH WALLER, SECTION EDITORS)

Preparticipation Cardiovascular Screening of Student-Athletes with Echocardiography: Ethical, Clinical, Economic, and Legal Considerations

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Abstract

Purpose of Review To identify whether the use of echocardiography is a viable approach for the screening of athletes for the prevention of sudden cardiac death when considering ethical, clinical, economic, and legal issues.

Recent Findings Ethical musings, echocardiographic findings, economic calculations, and legal analysis suggest that echocardiographic screening may reduce sudden cardiac death on the athletic field.

Summary Ethical, clinical, economic, and legal considerations suggest echocardiographic screening is a viable option to meet the societal goal to prevent athletic field sudden death.

Keywords Preparticipation screening · Echocardiography · Ethics · Economics · Liability · Sudden athlete cardiac death

Introduction

Sudden cardiac death (SCD) in a competitive athlete is a heartbreaking event. The sudden death of an athlete shocks the local community. The untimely athletic field sudden death of a young, robust athlete has led to community and national mandates for effective preparticipation screening programs to prevent sudden cardiac death. Presently, there is no proven preparticipation screening program to meet this mandate [1, 2]. Present-day screening has been flawed because it has focused on diagnosis rather than on the identification of specific variables known to quantify cardiovascular risk. The diagnosis of a disease does not quantify risk, and numerical associations do not delineate cause [3, 4]. The goal must be to identify a cause-and-effect relationship [5, 6].

Experts on both sides of the issue of echocardiographic preparticipation screening have debated the ethical, clinical, economic, and legal controversies surrounding the use of echocardiography to prevent SCD. The arguments against echocardiographic screening are limited human resources and the large financial investment in performing echocardiography. In this manuscript, there will be an exploration of the value for this screening weighing ethical, clinical, economic, and legal considerations.

This discussion will include the following: a physician's fiduciary responsibility to the student-athlete, the impact of intense physical activity on the morphological and physiologic changes of the myocardium to support augmented cardiac function, how changing myocyte function distinguishes between morphologically similar athletic heart and genetically acquired heart disease, how echocardiography is a cost-effective, validated imaging modality that is widely available and capable of simultaneously quantifying anatomic and physiologic features of athletes, and how Doppler echocardiography redefines the understanding of normal cardiac remodeling from latent and overt heart disease. Additionally, this article compares the cost of preparticipation screening to the dollar value of a statistical life (VSL) saved and discusses the complex liability issues

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related to cardiovascular preparticipation screening of student-athletes.

Ethical Considerations

Healthy competitive athletes may have unsuspected cardiovascular disease with the potential to cause SCD. This fact raises the query of what is the physicians' responsibility in preparticipation screening. The physician-patient (student-athlete) relationship is a member of a special class of legal relationships called *fiduciary relationships*. The physician is a fiduciary to the patient—athlete—and emanating from this relationship is the duty to always act in the best interest of the athlete.

The physician-patient relationship has its foundation in the theory that the physician is learned, skilled, and experienced in health, a subject in which the student-athlete has limited insight. Thus, the student-athlete must place confidence in the professional advice of the physician. The true essence of the fiduciary relationship is that the athlete's interests must be paramount. The fiduciary duty extends to all aspects of the physician-patient relationship. Understanding the factors that make the physician-patient relationship a fiduciary one will help physicians recognize potential violations [7].

In a competition, the athlete tests the cardiovascular system to the limit. The extremes of exertion occur in endurance and strength athletes, exposing the abnormal cardiovascular system to the potential for adverse health events and SCD. A physician fiduciary has the duty to identify the potential risks of intense physical exercise.

This fiduciary responsibility summons a strong ethical argument for implementing the highest quality-screening program for student-athletes. The occurrence of athletic field SCD during competition is felt to be ethically unacceptable if viable approaches to prevent these tragic deaths exist. It follows that reasonable preventive measures to detect previously unrecognized arrhythmias and congenital or acquired heart disease that may affect the health and longevity of the student-athlete are embodied in the ethical mandate. The detection of autosomal-dominant cardiac conditions may prevent adverse outcomes in the student-athlete and lead to evaluation of siblings and offspring who have a 50% chance of being affected. The physician's fiduciary responsibility to the student-athlete and a societal imperative to prevent athletic field sudden death ethically mandate the use of echocardiographic preparticipation screening.

The Physician Challenge: Distinguishing Athletic Heart from the Genetically Acquired Cardiovascular Diseases with Genetic Potential and Overt Risk for Adverse Cardiovascular Events

Cardiac Remodeling

Physician appreciation of the athletic heart's morphologic and physiologic variations is critical to understand when assessing student-athlete risk during intense competition [8]. Determining individualized risk in athletes requires an insight into the distinctions between the adaptive remodeling of an elite athlete and the maladaptive remodeling in genetically acquired heart disease. The typical morphological features of an athlete's heart are cardiac chamber enlargement, increased left ventricular (LV) mass, and aortic root dilation. The form and intensity of exercise will result in variable degrees of morphologic and physiologic remodeling [9, 10]. Cardiac morphology often vacillates between normal and abnormal during intense athletic training [11].

The elite athlete's cardiovascular physiology is dynamic. This dynamic range permits extreme athletic performance. The extreme physiologic changes associated with intense athleticism are related to LV relaxation and a fine-tuned physiologic and autonomic feedback system. The range of a resting heart rate < 40 beats/min to the peak exercise heart rates > 220 beats/min validates the impressive variability of the extremely conditioned cardiovascular system. A peak performance cardiac output may increase up to eightfold. These highly adaptive physiologic changes impact the adjoining ventricles, atria, and the aorta. This continuity between cardiac structures exposed to variations in volume and pressure results in adaptive and maladaptive changes in chamber physiology and morphology. The adjoining cardiac structures are impacted in elite athletes and in disease states [8].

Evolving myocyte changes over time allow an understanding of healthy (the athlete) and unhealthy (acquired heart disease) remodeling of the myocardium and provide insights into a distinction between health and disease. An athlete's diastolic function is typically supernormal, while individuals with heart disease experience early myocyte dysfunction evolving to LV diastolic and systolic dysfunction. This distinction is highlighted when comparing athletic heart and hypertrophic cardiomyopathy [12••].

In acquired heart disease, there is an early transition to abnormal myocyte function. This manifests as a reduction in mitral annular tissue velocity (depressed e′), followed by reduced strain, strain rate, and twist function and ultimately reduced global strain signifying fibrosis and cell death [13–15]. The acquired disease process is perceived

by appreciating the effects of multiple, related diastolic variables [16••, 17••].

A cardiovascular system may alter morphologically in a similar manner from athletic activity and an evolving disease state [18]. The solution to separating an athletic heart from a disease state lies in tests assessing causality [13, 14, 17••]. Causality is defined by abnormal myocyte function detected in the perturbation of diastolic variables.

The single best test to define cardiac morphology and physiology is echocardiography. Diastolic function is the crucial discriminator between health and disease. Assessing diastolic function is recommended as the most definitive way to categorize cardiovascular risk in athletes [19]. Diastolic function assessments are the best approach to identifying concealed disease in healthy, asymptomatic individuals [20, 21]. Athletes with genotype-positive and phenotype-negative expression of disease often have diastolic parameters indicating the pathophysiology associated with SCD. This diastolic data may allow for detection of an imminent life-threatening state before a tragic event [22–24], thus defining the degree of immediate risk and allowing for improved clinical decision-making [25, 26].

Specific Echocardiographic Features of Athletic Heart [27••]

The Left Ventricle

The LV remodels to variable degrees in athletes dependent upon the type (endurance versus strength) and intensity of exercise. The athletic heart characteristically has increased chamber dimensions and LV wall thickness [28]. These findings often mimic the echocardiographic features of diseases affecting the left ventricle. This conundrum is epitomized by the difficulty of distinguishing athletic heart and hypertrophic cardiomyopathy (HCM) (Fig. 1).

There is a difference in the cardiac remodeling of endurance and strength athletes. Elite endurance athletes typically exhibit increased wall thickness and moderate to severe chamber dilation, while strength-trained athletes exhibit an increase in LV wall thickness with no or mild chamber dilation. As expected, athletes that perform aerobic and strength training activities in their sport typically display a phenotype of mixed features of endurance and strength athletes [29]. The true phenotypic expression is frequently a mixture of pressure and volume overload of the myocardium [30–32]. The distinction between these phenotypic expressions and HCM is diastolic function.

Ultra-elite athletes may develop extreme remodeling of the LV resulting in LV end-diastolic dimensions > 60 mm. This extreme remodeling appears as a simulacrum of pathologic forms of dilated cardiomyopathy [32, 33]. The conundrum is the extreme remodeling in ultra-elite athletes may not regress with deconditioning. The dilated ventricle persists in 20% of retired elite athletes after 5 years. This raises the query of whether intense training may result in a phenotypic cardiomyopathy [34].

LV Diastolic Function

Athletic hearts do not exhibit diastolic dysfunction. Elite athletes have supernormal early diastolic filling (suction) manifested as an increased E wave velocity and a near absence of the A wave [35, 36]. Diastolic function variables are critical to distinguishing athletic heart from disease states [13, 14]. This distinction is highlighted with abnormal tissue Doppler echocardiography in gene-positive patients with HCM independent of phenotypic expression [15].

Diastolic function has a role in separating endurance athletes and strength athletes. Endurance athletes develop eccentric LV hypertrophy with elevated to supernormal diastolic function while strength athletes exhibit concentric hypertrophy with normal to mildly decreased diastolic function [37]. It has been noted that LV untwisting increases with intense exercise more than LV lengthening and expansion [38]. This enhanced LV untwisting rate may be the key to supernormal diastolic function as it improves early LV diastolic suction with almost complete LV filling during the E wave [39]. These speckle tracking echocardiographic parameters appear to be crucial to distinguishing adaptive and maladaptive remodeling in the difficult cases.

LV Systolic Function

LV systolic function is typically in the normal range among highly trained athletes. There are exceptions: elite cyclists may have LV ejection fractions lower than normal [40, 41], and highly conditioned, professional basketball players commonly have LV ejection fractions ranging from 45 to 50%, but cyclists and professional basketball players exhibit supernormal tissue Doppler peak systolic velocity (Sm), and normal systolic strain and strain rate measurements. The response of these athletes to stress testing is vigorous LV contractility. These additional measurements of LV systolic performance reveal normal LV systolic performance/function despite traditional resting parameters suggesting metrics below normative values.

It is critical to distinguish genetically driven heart disease with maladaptive remodeling from physiologic remodeling associated with intense physical training. Systolic strain and strain rate imaging of the LV have proven to be powerful in separating adaptive and maladaptive remodeling of the LV. Maladaptive remodeling results in a reduction of systolic strain and strain rate while with adaptive remodeling, these parameters are normal to supernormal and are laboratory-dependent [42]. Normal strain values include an integration

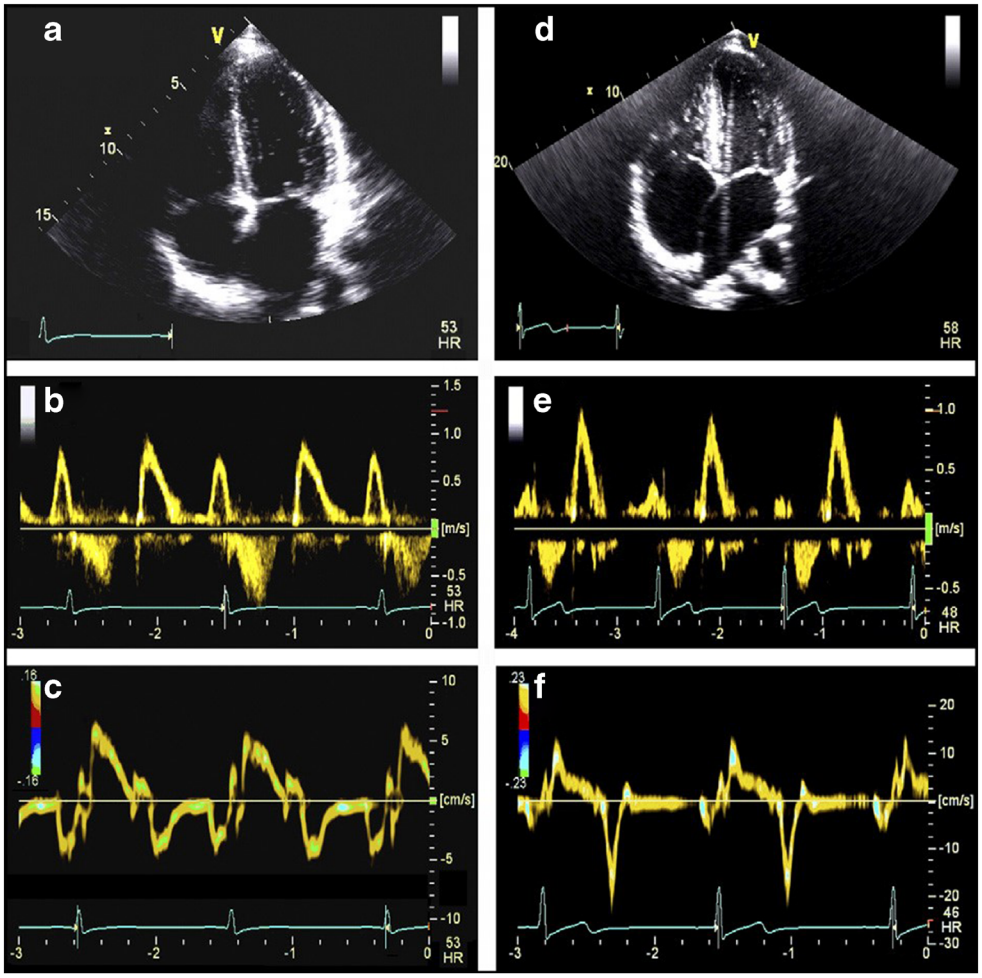

Fig. 1 Distinguishing features of HCM (maladaptive) from features of athletic heart (adaptive) using 2D echocardiography and Doppler parameters. **a** Apical 4-chamber image of a patient with hypertrophic cardiomyopathy exhibiting normal left ventricular dimension, increased left ventricular wall thickness, and dilation of the left atrium. **b** Mitral valve inflow Doppler reveals large atrial contribution in hypertrophic cardiomyopathy. **c** Tissue Doppler peak systolic velocity < 10 cm/s and early diastolic velocity (e′) < 5 cm/s in hypertrophic cardiomyopathy. **d** Athletic heart with an apical 4-chamber view revealing dilated left ventricle, increased left ventricular wall thickness, and increased left atrial volume. **e** Mitral inflow Doppler with prominent E wave and diminutive A wave. **f** Tissue Doppler peak systolic velocity > 10 cm/s and early diastolic velocity (e′) > 16 cm/s in athletic heart

of blood pressure, body surface area, left ventricular end-diastolic and end-systolic volumes, and ejection fraction. Additional parameters such as deformation mechanics, torsion, untwisting rate, and peak early diastolic untwisting rate are under investigation and may prove to be very important components of identifying early adaptive remodeling [43].

The Right Ventricle

Right ventricle (RV) phenotypic expression is very different in endurance- versus strength-trained athletes. The RV morphology in strength-trained athletes does not differ from that of sedentary controls. Alternatively, endurance athletes typically have increased RV wall thickness and RV cavity dilation when compared with sedentary controls [44]. The RV enlargement of endurance athletes often parallels the LV enlargement seen in endurance athletes [45–47]. There is the potential for the RV of an ultra-elite athlete to mimic arrhythmogenic RV dysplasia (cardiomyopathy).

RV function assessment is challenging and demanding because RV remodeling is variable across gender, race, and sports type. The parameters that appear most helpful in distinguishing endurance- and strength-trained athletes are RV inflow tract diameter, RV end-diastolic volume, and RV inflow velocity deceleration time. This was MRI data comparing elite soccer players and controls. This is an area that needs further investigation [48].

RV systolic function can be abnormal at rest in elite athletes, but there is a vigorous increase in RV systolic function with exercise, implying that the increased RV and right atrial sizes reflect physiologic remodeling [49]. Exercise testing may be necessary to evaluate true RV function as resting RV

contractility, strain, and strain rate often do not reflect true RV systolic function at rest in endurance-trained athletes [50–54]. Despite the heterogeneous morphologic remodeling seen in trained athletes, RV systolic and diastolic functions appear to remain normal. Often, this is proven through exercise testing revealing dynamic RV function when resting parameters appear depressed [48, 50, 55, 56].

The Aorta

The aorta of endurance athletes is exposed to increase volume, while the aorta of strength athletes is exposed to high pressure. The endurance athletes experience increased stroke volume frequently resulting in a larger aortic conduit. Endurance activity results in high-volume aortic flow with a modest increase in systolic blood pressure. The strength training results in normal volume aortic flow with a profound elevation of systolic blood pressure. There are conflicting data regarding aortic dimension in endurance and strength athletes: larger aortic root diameter among strength-trained athletes and refuting data reporting the largest aortic dimensions in endurance-trained athletes [57, 58]. The best available measurements in the literature on aortic morphology take into account aortic dimension and body surface area [59, 60].

The Atria

An increased left atrial volume index is normal in the athletic population. Intense athletic training results in remodeling of all cardiac chambers, including the left and right atria [61]. The typical endurance athlete has an increased LA volume index with normal LA pressure. Tissue Doppler and speckle tracking echocardiography are useful in evaluating LA function in athletes. Tissue Doppler assessment reveals supernormal diastolic function and speckle tracking reveals an increased LA reservoir and increased contractile function [62]. Assessment of LA myocardial deformation is critical to the evaluation of the LA with increased volume. LA myocardial deformation is impaired in patients with HCM compared with athletes and healthy controls [63].

Specific Features of Acquired Heart Disease

Causes of Sudden Cardiac Death

Sudden cardiac death is often due to an arrhythmia. There are two principal types of disease states that predispose people to complex arrhythmias: channelopathies and structural heart disease. Structural heart disease refers to non-coronary disease and includes valve disease, congenital heart disease, and cardiomyopathies. The nature of the triggers that initiate the arrhythmias varies by underlying disease state, pathophysiologic state, genetic determinants, and environmental factors [64]. People with cardiomyopathy, channelopathies, and structural heart disease frequently experience arrhythmia death [65–67].

Cardiomyopathies, channelopathies, and structural heart disease promote micro- and macro-vascular ischemia and replacement fibrosis, which cause electrical heterogeneity and induced arrhythmias [68, 69]. In athletes, a dormant "trigger" can suddenly initiate a life-threatening arrhythmia due to the physiologic stress of exercise. In vulnerable athletes, exercise changes LV compliance, rather than LV systolic function [70]. The distinction between a normal and abnormal "trigger" lies in the aggregated pathophysiologic profile of small groups of highly related features of diastolic function (E, A, E/A, e′, E/e′) [71] (as described below).

Echocardiographic Cardiac Morphologies Associated with SCD

Cardiomyopathies

Abnormal cardiovascular (CV) function can be effectively and reproducibly measured as a profile of small groups of highly related variables of diastolic function [71, 72]. This echocardiographic approach has been validated to have a very low incidence of false positive and false negative interpretations [63]. Typically, the diastolic parameters e′, E/e′ ratio, DT, and E/A ratio will allow a distinction between normal and abnormal myocyte function. These diastolic parameters are crucial in the early detection of myopathic processes because diastolic dysfunction precedes systolic dysfunction. Thus, diastolic dysfunction occurs early in the pathophysiologic continuum of maladaptive remodeling while structural and electrical changes occur later [73].

The prognostic importance of asymptomatic diastolic dysfunction is established [74]. For screening purposes, "cardiomyopathy" can theoretically describe almost any disease affecting the heart in which the heart muscle is physiologically and morphologically abnormal. Screening should be focused on prognostication of risk, and not detection, or characterization of a disease type. HCM is an archetypal candidate for pathophysiologic screening because standard testing does not distinguish it from athletic heart. HCM patients are typically asymptomatic, have nonspecific ECGs [75], and have a normal ejection fraction (EF), and morphological remodeling is variable and typically occurs late in the pathophysiologic cascade [76]. However, associated diastolic dysfunction can be detected and measured early in the HCM cascade, detecting pre-clinical HCM risk [15].

A screening exam should be designed to affirm the existence of abnormal physiologic profile and identify individuals with an abnormal risk profile, independent of morphology. The algebraic summation of a small aggregate of related data (E/A, DT, e′, E/e′) defines causality, which mirrors the status

of an individual's risk state in both positive and negative directions during serial screening, management, and follow-up [77]. Each cardiomyopathy disease (hypertrophic, restrictive, dilated, ischemic, hypertensive, arrhythmogenic right ventricular dysplasia, myocarditis, left ventricular noncompaction) has validated pathophysiologic features that can confirm a risk state. Averaged features have a very low incidence of false positive and false negative results [65].

Hypertensive Heart Disease

Screening blood pressure and increased LV wall thickness serve only as benchmarks that require physiologic classification with diastolic function assessment. The overlapping features of normal myocardial remodeling can mimic the features of HCM, hypertensive heart disease, and infiltrative diseases [78]. The goal of screening is to distinguish adaptive versus maladaptive remodeling. This is accomplished with diastolic function assessment independent of the disease state.

Ischemic Heart Disease

The risk of sudden death from coronary artery disease (CAD) is much greater among sports participants > 35 years and is greatest in the fifth decade. The incidence of fatal CAD is reported to be 0.7 per 100,000 people-years for athletes < 35 years of age and 13.7 per 100,000 persons-years for those ≥ 35 years of age [79]. The focus on the late-stage features of CAD (ECG abnormalities, angina, wall motion abnormality, and infarction) is too late and needs to be redirected to the more fundamental underlying subclinical diastolic and systolic dysfunctions that emerge earlier in the pathophysiologic cascade [80]. The objective of screening asymptomatic athletes is to disclose imminent pathophysiologic risk (occurrence of an event within 1 year, and not the presence of long-term risk; the Framingham Score 10-year risk profile) [81–83].

Channelopathies

Channelopathies are variably classified as a primary electrical disease. Channelopathies are often assumed to be the etiology in cases of undefined cause of athletic field death. The genetic ion channelopathies associated with sudden cardiac death include long QT, Brugada, short QT, catecholaminergic polymorphic ventricular tachycardia, and idiopathic ventricular fibrillation. These various forms of electrical disorders are not identifiable on echocardiography and require a molecular autopsy for identification. There is some speculation that the abnormal repolarization and rhythm disturbances may precipitate functional changes in the myocardium that may be detected through diastolic parameters [84].

Pulmonary Hypertension

The identification of significant pulmonary hypertension (PH) is unlikely in highly competitive athletes. Screening for PH prior to athletic competition in high school athletes is valuable as it may identify unrecognized PH and it is easily recognized with Doppler echocardiography. The pulmonary pressure can be estimated using several Doppler techniques including the tricuspid regurgitation velocity, the end-diastolic pulmonary regurgitation velocity, and the pulmonary flow acceleration time. These are screening measures and if values are elevated, there should be further investigation.

Coarctation of the Aorta

Coarctation of the aorta is infrequent. It should be excluded in every patient with bicuspid aortic valve (BAV). Coarctation of the aorta can be detected and quantified by a simple echo/Doppler signal in the abdominal aorta [85]. In the presence of coarctation, the overall aortic velocity is blunted, systolic upstroke is delayed, and there is continuous forward flow consistent with a diastolic and systolic gradient and/or collateral flow. Early diastolic reversal of flow is associated with a widely patent aortic arch. Elastic recoil of the ascending aorta causes the low velocity, late diastolic forward flow seen after the early diastolic reversal.

Anomalous Coronary Artery

Anomalous coronary arteries are a known cause of symptoms and sudden death in competitive athletes. The most life-threatening coronary anomaly is when the left or right coronary artery originates from the contralateral sinus of Valsalva and courses between the aorta and the pulmonary artery. Although the prevalence of coronary anomalies in the general population is debated, it has been reported to be the second most common abnormality associated with sudden death in athletes in Italy (~ 17%) [86, 87]. Echocardiography allows identification of these anomalies since the origin of the coronary arteries can be identified in 75–90% of young athletes [86]. If the origins are abnormal on echocardiography, further imaging with cardiac magnetic resonance is recommended. Echo assessment of the origin of the coronary arteries is an essential component of every athletic screening examination.

Aortic Aneurysm

There are three syndromic aortopathies that should be considered in screening of asymptomatic athletes: Marfan syndrome, bicuspid valve-associated aortopathy, and cystic medial necrosis [88, 89]. An aortic root dimension > 40 mm in a highly conditioned male athlete and > 34 mm in a highly conditioned female athlete is uncommon and should raise a concern for

aortic root pathology. Surveillance of the ascending aorta should be based upon root dimension and the patient's body surface area. Measurements should be leading edge to leading edge at the level of the annulus, sinuses of Valsalva, sinotubular junction, and ascending aorta [60, 90–92].

The presence of any aortic valve regurgitation in a young healthy athlete should alert the examiner to the possibility of aortic valve or aortic root pathology. These findings should result in life-long surveillance. The risk of rupture is related to an indexed relative size measure of the aortic root diameter to body surface area.

Valve Disease

Asymptomatic athletes with normal diastolic function and mild valvular disease typically will be eligible for physician-monitored athletic activities. Quantitative echocardiography/Doppler is a validated approach for evaluation and surveillance of both symptomatic and asymptomatic valve diseases [93]. A comprehensive diastolic physiologic profile is the best approach to determining imminent risk for adverse cardiovascular events even in patients with valve disease. Early echocardiographic evaluation of valve disease in the general population and the athletic population does not translate into reduced cardiovascular events or death [94, 95].

The Economics of Preparticipation Echocardiographic Screening—Is a Cost-Benefit Analysis Feasible?

Cost-Benefit Analysis [12••]

The present-day perception is that screening with echocardiography will yield excessive costs beyond the potential benefits. This is pertinent because we struggle as a society regarding resource allocation [96]. This mandates a comparison of the cost of screening to the dollar value of a statistical life (VSL) saved.

Cost of Screening

The cost of screening has been considered an obstacle to using echocardiography from a cost-benefit analysis standpoint. Standard echocardiography is expensive. There are approximately 460,000 college student-athletes participating in 24 sports at the National Collegiate Athletic Association according to NCAA.org statistics. The annual rate of cardiac death is approximately 1:4300 athletes. There are approximately ten NCAA collegiate athletic deaths each year [97, 98].

For calculation purpose, let us assume a screening echocardiogram costs $100.00 per college student-athlete resulting in a cost of $46 million per year. The screening echocardiogram would evaluate for morphological abnormalities and perform a detailed diastolic analysis. This approach to screening would have a high likelihood of identifying the majority of diseases with imminent risk of adverse cardiovascular events. The Achilles heel of this screening process is ion channelopathies.

What Is the Positive Dollar Value of Screening?

The benefit of preventing a fatality is measured by what is conventionally called the value of a statistical life (VSL). VSL is defined as the additional cost that individuals would be willing to bear for improvement in safety, i.e., reductions in risks that, in the aggregate, reduced the expected number of fatalities. There are mathematical and statistical formulas for calculating VSL for an additional year of statistical life (VSLY) [99]. To put teeth into this concept, consider a regulation (echocardiographic screening) that reduces risk by one in a 100,000 individuals. This is the equivalent of saving one statistical life.

The US Food and Drug Administration (USFDA) currently uses $8 million as its standard per estimated VSL [99]. This estimated value represents the value from birth to age 78 years. To identify a reasonable estimate for VSL for echocardiographic screening to prevent SCD, we extrapolated from the USFDA data. We annualized this data on VSL using a range of discount rates. The discount rates account for the fact that a dollar in the future is worth less than a dollar today; therefore, a dollar's worth of life in the future is worth less than a dollar worth of life today. Review of the literature reveals discount rates at zero, 3%, and 6% for VSL calculation, with 3% considered the benchmark [99, 100]. Using these discount rates, annualizing $8 million over 78 years yields VSLs of approximately $142,000.00, $ 278,000.00, and $520,000.00. Using each of these VSLY approximations, one can mathematically calculate the value of each life saved:

$$\text{Value of Life Saved} = \sum_{t=1} \text{VSLY}/(1+r)^t$$

This assumes a discount rate, r, and saved individual years (T) after the screening [99]. Assuming that screening occurs at age 18 years, T61, and utilizing the discount rates and corresponding VSLY estimates, this calculation yields values of each life saved ranging from $6.7 million to $8.3 million. More specifically, the formula yields:

- A value of $6.7 million assuming a VSLY of $142,000 and a discount rate of zero.
- It yields a value of $7.9 million assuming a VSLY of $278,000 and a discount rate of 3%.
- Finally, it yields a value of $8.3 million assuming a VSLY of $520,000 and a discount rate of 6%.

Therefore, if screening saves ten lives, the estimated benefit of screening is $67 million to $83 million. Thus, the cost-benefit analysis as provided with this prudent and reasonable screening model would favor screening from an economic perspective. The calculus from the lowest common denominator would suggest that screening will benefit society $21 million.

These calculations give a strong financial support and incentive to use screening echocardiography to prevent SCD in college student-athletes. The universities should finance the screening as they make profits from the sports activities on their campuses that could contribute to the cost of screening. This is a reasonable consideration given that athletes are performing for the university, and sports activities add to the overall experience of the entire student body who pay tuition to the university. It is a policy decision that deserves careful scrutiny [12••].

Liability Issues Surrounding Preparticipation Echocardiographic Screening

Liability Issues [12••]

The law requires physicians to use customary skill and medical care consistent with evidence-based best medical practice in evaluating athlete's fitness to participate in sports. The physician's fiduciary duty is to protect the athlete's interests and well-being. The present-day medical-legal construct holds that a high school or collegiate athlete may be withheld from competitive sports due to a cardiovascular abnormality to prevent medically unacceptable risk [101]. Preparticipation screening with echocardiography allows the identification of most cardiovascular abnormalities associated with increased risk.

A concern of many institutions is that a policy advocating echocardiographic screening as a standard of care for participation and disqualification may expand physician and institutional liability. The concern arises from the belief that screening would expand liability if an abnormality was present and not appreciated on echocardiography. This athlete would then be cleared to play competitive sports and be at risk for athletic field death. Retrospective review could then expose liability.

The alternative argument is that well-trained cardiologists would increase the detection cardiovascular conditions for which athletes are at risk. This detection would allow for disqualification of high-risk athletes. This detection of increased risk would decrease the incidence of SCD and therefore the risk of liability. Thus, the improved detection would reduce SCD and necessarily the potential for alleged negligence.

Viewed from a different lens, if we are not using screening echocardiography and an autopsy reveals an

Table 1 Examples of an educational institution's duty of care to the student-athlete [12••]

Case: Kleinknecht v. Gettysburg College (1993)	
Allegation	The deceased student-athlete's parents filed a wrongful death suit against Gettysburg College.
Legal lesson	Educational institutions have a special relationship with student-athletes and owe them a higher duty of care. Educational institutions have a duty to prepare an emergency medical response program for injured student-athletes that is responsible and respectful of the community standard of care.
Case: Davidson v. University of North Carolina at Chapel Hill (2001)	
Allegation	The university failed to supervise and educate student-athletes regarding safety of cheerleading.
Legal lesson	A special relationship existed between the university and the cheerleading team; thus, there was a duty to educate and supervise its cheerleading squad regarding safety. This finding extended the duty of care of a university to nonrecruited student-athletes.
Case: Kennedy v. Syracuse University (1995)	
Allegation	The university breached its duty to the student-athlete by not having a trainer present at all practices.
Legal lesson	The university, through its special relationship with student-athletes, had a duty to have a trainer present at all practices.
Case: Orr v. Brigham Young University (1994)	
Allegation	Failure to provide adequate medical care for a student-athlete who had a back injury in a university football game.
Legal lesson	Contrary to legal precedent, Orr found that a special relationship did not exist, thus negating the heightened duty of care.

echocardiographically detectable anomaly, does that prove negligence? The answer to these legal queries remains an enigma. The best solutions to these queries are the development of screening programs with well-trained cardiologists evaluating echocardiograms prior to competition. Detection of maladaptive remodeling and imminent risk would lower the incidence of SCD and the potential for alleged negligence.

Negligence Law and Duty

Discussion of liability issues invariably begins with the law of negligence. Negligence law provides that there must be a legal duty owed to a student-athlete before any discussion of liability can proceed. Courts typically maintain that student-athletes have a sufficient programmatic relationship with their educational institution to impose a duty of care on educational institutions and their physicians.

Case Law

Case law suggests that colleges owe all their student-athletes a duty in the scope of their athletic programs to take reasonable precautions against reasonably foreseeable harms [102, 103]. The national trend is to recognize a legal duty in the context of programmatic risk [104–108]. (Table 1).

Although the available case law and statutes limiting the scope of a physician and university's role in preparticipation screening are incomplete, basic principles of legal responsibility are well-established. Physicians must behave in accordance with a professional standard of care that is typically set by customary medical practice. We believe customary medical practice should include preparticipation screening echocardiography.

Conclusion

Echocardiographic screening is an emotionally charged debate between experts in the cardiology community. When there is intense negotiation between parties, shared interests are the "elixir of negotiation," the salve that will smooth over issues the parties vehemently debate. The major shared interest is prevention of SCD.

In our opinion, the argument for the use of echocardiographic screening prior to athletic participation to prevent SCD is compelling when ethical, clinical, economic, and legal issues are carefully considered. Its use meets the shared interest of all involved parties of saving lives by reducing sudden death on the athletic field. A paradigm shift of this magnitude will require collaboration among educational institution administrators and physicians devoted to screening based upon an in-depth knowledge of adaptive and maladaptive cardiac remodeling.

Compliance with Ethical Standards

Conflict of Interest Zachary R. Paterick and Timothy E. Paterick declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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