2018

Pre-Participation Physical Examination: Are Current Cardiovascular Recommendations Enough?

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PRE-PARTICIPATION PHYSICAL EXAMINATION: ARE CURRENT CARDIOVASCULAR RECOMMENDATIONS ENOUGH?

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Bachelor of Science, George Washington University 2004

A Scholarly Project

Submitted to the Graduate Faculty

Of the

University of North Dakota

In partial fulfillment of the requirements

For the degree of

Master of Science in Physician Assistant Studies

Grand Forks, North Dakota

May 2018

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Acknowledgements

I would like to acknowledge my family. Without you, I would not be here. Also, I would like to thank Jay Metzger for his sage counsel and guidance throughout this journey.

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Abstract

Inclusion of screening electrocardiograms (EKGs) during pre-participation physical examination has been a topic of debate for some time. There is unquestioned usefulness in the ability of a well-trained health care provider in using EKG to identify cardiac abnormalities. Further, there have been several contemporary EKG criteria published that increase specificity and sensitivity of detection of disease. However, these criteria do not account for athletes less than 14 years of age, and that demographic represents a large portion of patients seeking pre-participation screenings in the United States. The lack of research into pediatric cardiac remodeling secondary to activity, coupled with the significant overlap in normal pediatric EKG findings with adult pathological EKG findings create a difficult position for any health care provider. A literature review was performed to determine if screening EKGs are effective both medically and economically in athletes less than 14 years of age. Based on the review, the limitations of contemporary EKG criteria, the inability to prove cost effectiveness in the US healthcare model, and the lack of research into activity modulated pediatric cardiac remodeling should reinforce that the ACC/AHA checklist is an appropriate foundation for conducting pre-participation physical examination.

Keywords: pre-participation physical examination, sudden cardiac death, hypertrophic cardiomyopathy, electrocardiography, athletes, “Death, Cardiac, Sudden” [MeSH], “electrocardiography, screening” [MeSH], “cost-benefit analysis” [MeSH].

Pre-Participation Physical Examination: Are Current Cardiovascular Recommendations Enough?

Introduction

Pre-participation physical examinations are routinely conducted in the United States in an effort to protect young athletes. There are numerous estimates regarding the incidence of sudden cardiac death (SCD) in athletes, ranging from one in 40,000 to one in 80,000, and though SCD is a rare occurrence the multifactorial effects on the surrounding community cannot be denied. The majority of recommendations surrounding these physicals support gathering a personal and family history of cardiovascular disease as well as a physical examination. However, many of the underlying pathologies that lead to SCD produce subtle findings on exam and are difficult for even an experienced Primary Care Provider to detect (Finocchiaro, et al., 2016).

In patients requiring pre-participation physical examination, would EKG be an effective screening tool, both medically and economically, to identify conditions that may cause SCD in athletes younger than 14 years old? I conducted research into this question in PubMed, SPORTDiscus, and the Cochrane Library using key words, phrases, and MeSH terms: pre-participation physical examination, sudden cardiac death, hypertrophic cardiomyopathy, electrocardiography, athletes, “Death, Cardiac, Sudden” [MeSH], “electrocardiography, screening” [MeSH], and “cost-benefit analysis” [MeSH]. The research was then aligned into the following themes: pathophysiology, physical and EKG findings, contemporary EKG criteria, economic impact, and current recommendations. Electronic databases were searched with the date range of January 1990 to Oct 2017 for systematic reviews, meta-analyses, randomized controlled trials, and clinical reviews that were published in English. Articles were reviewed for relevance and included if they contained information on the etiology of sudden cardiac death in people younger than 35 years old. Articles that focused on atherosclerotic coronary artery disease as the cause of SCD death were excluded.

Pathophysiology

> SCD is defined as a sudden unexpected death due to cardiac causes, or a sudden death in a structurally normal heart that has no other explanation and a history consistent with cardiac-related death, that occurred within one hour of symptom onset in a person without known cardiac disease or an unwitnessed death occurring within 24 hours of the person having been alive and symptom free (Asif & Harmon, 2017, p. 269)

Generally, the leading causes of SCD are divided into two groups: structural malformations and channelopathies. In athletes younger than 35 years old, hypertrophic cardiomyopathy (HCM) accounts for 48% of SCD cases (Lisman, 2016) with several other structural anomalies and channelopathies accounting for the remaining 42%. The focus of this theme will be to give an overview of hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy.

Hypertrophic Cardiomyopathy

In 1957, Robert Donald Teare, an English pathologist at St. George's Hospital in London, reported the autopsy findings of eight patients with asymmetrical hypertrophy of the heart, seven of whom died suddenly (Liew, Vassiliou, Cooper, & Raphael, 2017). The disease was later named HCM. HCM is the most common inherited heart defect, occurring in 1 of 500 individuals through autosomal dominant inheritance (McCance & Huether, 2014) and generally considered the leading cause of sudden death in the young, with an incidence of sudden cardiac death of 0.5% to 1% per year (Liew et al., 2017). These mutations cause significant alteration to the contractile proteins in the sarcomeres of the myocardium. The most commonly involved genes are MYH7, MYBPC3, TNNT2 and TNNI3 (Varma & Neema, 2014) with MYH7 and MYBPC3 being responsible for up to 80% of cases. Figure 1 illustrates the sarcomere with established causal genes identified.

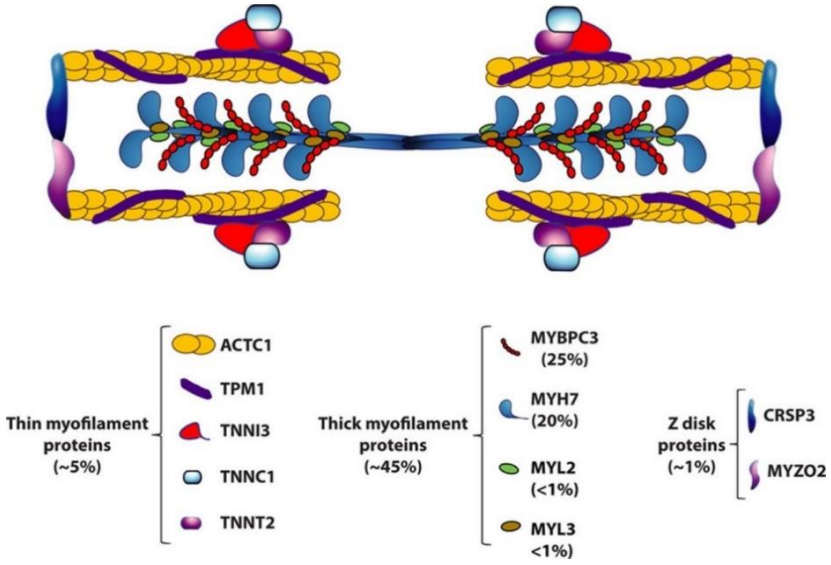

Figure 1. Hypertrophic cardiomyopathy (HCM) as a disease of sarcomere proteins. A schematic structure of a sarcomere composed of thick and thin filaments and Z discs is depicted along with its protein constituents involved in HCM. Established causal genes for HCM and their population frequencies are listed. From "Hypertrophic Cardiomyopathy: Genetics, Pathogenesis, Clinical Manifestations, Diagnosis, and Therapy", by A.J. Marian and E. Braunwald, 2017, *Circulation Research, 121*(7), p. 751. Copyright 2017 by American Heart Association, Inc. Reprinted with permission.

When the structure of the sarcomere is altered, the function of the sarcomere, specifically cardiac contraction, is altered as well. The exact mechanism of how mutations in sarcomere-related genes lead to hypertrophy is unknown, but these mutations lead to hypertrophy of heart muscle, myocardial disarray, and fibrosis (Varma & Neema, 2014).

Characteristically HCM will manifest in the ventricular septum, however, hypertrophy can be isolated to the left ventricular free wall, apex and anterolateral wall, and can be concentric in rare cases (Varma & Neema, 2014). As the thickness of the septal wall increases both dynamic and static issues arise. During systole, the hypertrophied septum can cause a left ventricular outflow obstruction, specifically with increased heart rate. Conversely, during diastole, ventricular relaxation and compliance are decreased which will significantly decrease intraventricular volume. Collectively, these events will lead to a decreased cardiac output.

Arrhythmogenic Right Ventricular Cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a primarily autosomal dominant disease whose prevalence is estimated to range from 1 case in 5000 persons in the general population to 1 in 2000 in some European countries (Corrado, Link, & Calkins, 2017). Mutated desmosomal proteins are central to the pathogenesis of ARVC. These proteins include desmoplakin (DSP), plakophilin 2 (PKP2), desmoglein 2 (DSG2), and desmocollin 2 (DSC2) (Corrado et al., 2017, p. 63). Desmosomes provide physical intercellular attachment points and are critical in preventing myocyte detachment and cellular death. Early in the disease, known as the concealed phase, the changes are discreet. As AVRC progresses to the overt phase, myocardial mass decreases and is replaced by fibrofatty tissue. This fibrofatty tissue does not possess the same electrical conductive properties as normal myocardium and is thought to contribute to the development of ventricular arrhythmias by slowing intraventricular conduction and acting as a substrate for arrhythmias through a scar-mediated macro-reentry mechanism, similar to that observed after myocardial infarction (Corrado et al., 2017, p. 63). It is possible that physical exercise could potentiate myocytic uncoupling and accelerate the progression of AVRC, so identification in the pre-participation physical examination becomes paramount.

Physical and EKG findings

Hypertrophic Cardiomyopathy

Physical examination may provide a practitioner with several clues suggesting HCM. Patients may initially be asymptomatic, but potentially may develop fatigue, peripheral edema, angina, and palpitations. Secondary to the dynamic nature of the left ventricular outflow obstruction, the carotid pulse should initially be brisk followed by a decrease and then a second increase. This phenomenon is known as bisferiens pulse and should assist a provider in differentiating HCM from other conditions with fixed left ventricular outflow obstructions such

as aortic stenosis. Additionally, the characteristic murmur associated with HCM is a crescendo-decrescendo murmur best appreciated at the upper left sternal border. Where most murmurs will decrease with the Valsalva maneuver, the murmur associated with HCM will increase, so the importance of dynamic testing in cardiac assessment cannot be overstated.

Ventricular hypertrophy may present with characteristic changes on EKG. In a normal EKG, the QRS complex in V1 will have a mostly negative deflection. Dubin (2016) outlines expected criteria for identifying ventricular hypertrophy on EKG:

Right ventricle

R wave greater than S wave in V1, but R wave gets progressively smaller from V1 to V6

S wave persists in V5 and V6, right axis deviation with slightly widened QRS complex

Rightward rotation in the horizontal plane.

Left ventricle

Depth (in mm) of S wave in V1 plus the height of R wave in V5 is greater than 35 mm

Left axis deviation with a wide QRS complex

Leftward rotation in the horizontal plane

Inverted T wave with gradual downward slope that returns rapidly to baseline

Arrhythmogenic Right Ventricular Cardiomyopathy

AVRC is most common in the second decade of life, and early disease may be asymptomatic, though the patient may still be at risk of SCD with increased activity. As the disease progresses, the patient may begin to experience light headedness, syncopal episodes and palpitations. In more advanced disease, signs of heart failure such as peripheral edema and shortness of breath begin to develop. There have been rare reports of an early systolic ejection murmur at the fourth intercostal space on the left, but usually there are no abnormal findings on physical examination early in the disease.

Up to ninety percent of patients with ARVC will demonstrate pathological features on EKG prior to symptom onset that include right bundle branch block (RBBB), premature ventricular contractions (PVCs) with left bundle branch block (LBBB) morphology, localized prolonged QRS complexes in the right precordial leads, and T wave inversion in the absence of RBBB in patients older than 12 years old (Leger et al., 2016, p. 6). ARVC may demonstrate RBBB and right precordial ST segment elevation on EKG similar to Brugada syndrome, but this represents a very small group of patients. RBBB can be identified in the right precordial leads by a widened QRS complex with an R and R' waveform. The depolarization of the individual ventricles is of normal duration; this waveform occurs because the asynchronous depolarization waves are superimposed on one another and the machine records this combined electrical activity as a widened QRS complex with two peaks (Dubin, 2016). In RBBB, the left ventricle will depolarize initially (R) followed by the right ventricle (R').

Contemporary EKG Criteria

Over the past decade, specific criteria have been developed to assist health care practitioners in evaluating EKGs in athletes. The first set of recommendations was released in 2005 by the European Society of Cardiology (ESC) and refined in 2010 to improve specificity secondary to the increasing number of sporting governing bodies undertaking per-participation cardiovascular screening reporting unacceptably high levels of false-positive EKGs arising from the overlap between physiological EKG patterns commonly observed in athletes and those suggestive of cardiac pathology (Riding, et al., 2015). In the report, Corrado et al. (2010) classified abnormalities of an athlete's EKG into one of two groups: common and training related EKG changes and uncommon and training-unrelated EKG changes. Table 1 further defines the differences between the groups.

Table 1. ESC classification of abnormalities of the athlete's electrocardiogram.

Group 1: common and training-related ECG changes	**Group 2: uncommon and training-unrelated ECG changes**
Sinus bradycardia	T-wave inversion
First-degree AV block	ST-segment depression
Incomplete RBBB	Pathological Q-waves
Early repolarization	Left atrial enlargement
Isolated QRS voltage criteria for left ventricular hypertrophy	Left-axis deviation/left anterior hemiblock
	Right-axis deviation/left posterior hemiblock
	Right ventricular hypertrophy
	Ventricular pre-excitation
	Complete LBBB or RBBB
	Long- or short-QT interval
	Brugada-like early repolarization

From "Recommendations for interpretation of 12-lead electrocardiogram in athletes" by D. Corrado, A. Pelliccia, H. Heidbuchel, S. Sharma, M. Link, C. Basso, A. Biffi, G. Buja, P. Delise, I. Gussac, A. Anastasakis, M. Borjesson, H.H. Bjornstad, F. Carre, A. Deligiannis, D. Dugmore, R. Fagard, J. Hoogsteen, KP Melwig, N. Panhuyzen-Goedkoop, E. Solberg, L. Vanheas, J. Drezner, N.A.M. Estes, S. Iliceto, B.J. Maron, R. Piedro, P.J. Schwartz, R. Stein, G. Thiene, P. Zeppilli, W.J. McKenna, 2010, *European Heart Journal*, *31*(2), p. 245.

The researchers provided recommendations for each of the group 1 and 2 changes based on a retrospective application of their criteria to 1,005 athlete's EKG from the previous decade. Previously, 40% of the athletes were diagnosed with abnormal EKGs. After repeat analysis and application of the 2010 ESC criteria, that number was lowered to 11%. Many studies have demonstrated that certain black ethnic populations, such as African, African-Caribbean, and Black Latin-American, continue to demonstrate a high prevalence of abnormal EKGs (approximately 20-40%) when using the 2010 ESC recommendations (Riding, et al., 2015)

Regular, intensive physical activity provokes cardiological adaptations that are commonly referred to as the athlete's heart. McClean et al. (2017) conducted a systematic review with meta-analysis of original research articles published in English. MEDLINE, PubMed, EMBASE, Web of Science, CINAHL, and SPORTDiscus were searched for relevant studies and a total of 14,278 male and female competitive athletes and 1688 non-athletes between 6 – 18 years of age were include in the review. The authors performed a risk of bias assessment using a self-developed 15 item check sheet. Articles that fell below 50% were excluded from the review. The researchers concluded that pediatric athletes had significantly longer PR intervals, and a significantly greater frequency of sinus bradycardia, first-degree atrioventricular block, and incomplete right bundle branch block than pediatric non-athletes. Voltage criteria for left ventricular hypertrophy was present in 35.2% (26.0 – 45.0, 95% CI, $p \leq 0.001$) of athletes when compared to pediatric non-athletes. Anterior (6.5% vs 5.7%), inferior (0.9% vs 0%) and lateral (0.2% vs 0%) T wave inversion was also more common in athletes than non-athletes. The review did reinforce previously known cardiac adaptations in adult athletes. New findings of the review included pediatric athletes being 13 times more likely to have a T wave inversion of ≥ 2 mm than non-athletes, pediatric athletes ≥ 14 years old are 16 times more likely to have inferolateral T wave inversion than athletes < 14 years old, black pediatric athletes

are up to 36 times more likely to have extended T wave inversion in V1 – V4 than Caucasian athletes, and left ventricular size was greater among athletes than non-athletes. A significant limitation of the study was the use of the European Society of Cardiology Recommendations which are not intended for use in athletes under 12 years old. Body mass index (BMI) was calculated from listed height and weight; this could be a potential source of error if the authors had considered linking BMI to incidence of HCM as it would be unknown if this was measured versus stated weight during the encounter. The review also illustrates the need for further research regarding cardiac remodeling secondary to physical training in the pediatric population.

In 2012, an international group of experts met in Seattle, WA and developed the Seattle Criteria for evaluation of EKGs in athletes and non-athletes between 14 and 35 years of age. The Seattle Criteria built upon the foundation of the 2010 ESC recommendations, and they expanded the lists of normal and abnormal EKG findings commonly associated with the athlete's heart. One group of contributors, Sharma, et al (1999), conducted an analysis of 1000 postpubertal junior elite athletes' 12 lead EKGs between April 1995 and November 1998. Forty-five percent of the athletes ($n = 450$, $p < 0.0001$) met or exceeded the voltage criteria for left ventricular hypertrophy based on the Sokolov voltage criterion compared to 23% non-athletes ($p < 0.0001$). Additionally, incomplete right bundle branch block was found in 29% ($p < 0.0001$) of athletes as opposed to 11% ($p < 0.0001$) of non-athletes. They concluded that their findings suggested that isolated instances of Sokolov voltage criterion for left ventricular hypertrophy, elevated ST segments, and peaked T waves are common variations in athletes and do not warrant cardiology evaluation in an otherwise healthy athlete without family history of cardiac channelopathies or structural issues. The study was limited in that only 1,000 EKGs were analyzed, and of that 988 came from white athletes and 73% were male. Additionally, of the 300 control group EKGs, 293 belonged to white subjects. In addressing the ESC recommendations' high rate of false

positive findings in athletes of African descent, the Seattle Criteria discussed early repolarization findings in black athletes, specifically a normal variant early repolarization pattern found in some black/African athletes, characterized by an elevated ST segment with upward convexity (dome shaped), followed by a negative T wave confined to leads V1-V4 (Drezner, et al., 2013). A significant limitation of the Seattle criteria is that they were developed with consideration of EKG interpretation in the context of an asymptomatic athlete age 14 to 35 years of age (Drezner, et al., 2013)

Shortly after the Seattle criteria were published, Sheikh et al. (2014) evaluated 1,208 black athletes and found that several isolated EKG patterns have a low diagnostic yield for cardiac disease, questioning their relevance as markers of pathology in athletes. Further, they observed that the previous research was mainly derived from white athletes and that the paucity of EKG interpretation criteria in black athletes was of concern, given that black athletes most frequently exhibit profound EKG alterations that overlap with primary cardiomyopathies, which magnifies their risk of an erroneous diagnosis (Sheikh, et al., 2014). The refined criteria proposed a third definition of abnormal EKGs that varied slightly from both the ESC recommendations and the Seattle criteria and are illustrated in table 2. The authors conducted a retrospective analysis of 5505 EKGs using the ESC recommendations, the Seattle criteria and the refined criteria. They found that application of the ESC recommendations resulted in 21.5% (n = 1183) of the EKGs being evaluated as abnormal. Application of the Seattle criteria yielded a 9.6% ($p < 0.001$) rate of abnormal EKGs. The refined criteria yielded only a 6.6% ($p < 0.001$) abnormal rate. The refined criteria also outperformed both the ESC recommendations and the Seattle criteria in reduction of abnormal EKGs in black athletes with a 71.5% and 37.5% reduction, respectively.

Table 2. ECG Parameters Used to Define Various ECG Abnormalities in the European Society of Cardiology Recommendations, Seattle Criteria, and Refined Criteria.

ECG Abnormality	European Society of Cardiology Recommendations	Seattle Criteria	Refined Criteria
Left atrial enlargement	Negative portion of the P wave in lead V1 ≥0.1 mV in depth and ≥40 ms in duration	Prolonged P wave duration of >120 ms in lead I or II with negative portion of the P wave ≥1 mm in depth and ≥40 ms in duration in lead V1	As ESC
Right atrial enlargement	P-wave amplitude ≥2.5 mm in lead II, III, or aVF	As ESC	As ESC
Left QRS axis deviation	−30° to −90°	As ESC	As ESC
Right QRS axis deviation	>115°	>120°	As ESC
Right ventricular hypertrophy	Sum of R wave in V1 and S wave in V5 or V6 ≥10.5 mm	Sum of R wave in V1 and S wave in V5 >10.5 mm and right axis deviation >120°	As ESC
Complete LBBB	QRS ≥120 ms, predominantly negative QRS complex in lead V1 (QS or rS), and upright monophasic R wave in leads I and V6	As ESC	As ESC
Complete RBBB	RSR′ pattern in anterior precordial leads with QRS duration ≥120 ms	Not relevant	As ESC
Intraventricular conduction delay	Any QRS duration >120 ms including RBBB and LBBB	Any QRS duration ≥140 ms or complete LBBB	As ESC
Pathological Q-wave	>4 mm deep in any lead except III, aVR	>3 mm deep or >40 ms duration in ≥2 leads except III and aVR	≥40 ms in duration or ≥25% of the height of the ensuing R wave
Significant T-wave inversion	≥2 mm in ≥2 adjacent leads (deep) or “minor” in ≥2 leads	>1 mm in depth in ≥2 leads V2−V6, II and aVF, or I and aVL (excludes III, aVR, and V1)	As Seattle
ST-segment depression	≥0.5 mm deep in ≥2 leads	As ESC	As ESC
Ventricular preexcitation	PR interval <120 ms with or without delta wave	PR interval <120 ms with delta wave	As Seattle criteria

From “Comparison of Electrocardiographic Criteria for the Detection of Cardiac Abnormalities in Elite Black and White Athletes”, by N. Sheikh, M. Papadakis, S. Ghani, A. Zaidi S. Gati, P.E. Adami, F. Carre F. Schnell, M. Wilson, P. Avila, W. McKenna, S. Sharma, 2014, *Circulation, 129*(16), p. 1640.

Economic Impact

The debate surrounding the need for routine EKG in conjunction with pre-participation exam has been ongoing for some time. High false positive rates and the cost of further specialist appointments are one of the greatest barriers to mainstream acceptance. The ESC recommendations for EKG screening stem from one Italian study. The study reported a marked decline in the incidence of SCD among athletes following the implementation of an Italian law mandating EKG screening (Corrado, Basso, Pavei, Michieli, & Thiene, 2006) but failed to address economic ramifications. Halkin, et al (2012) executed a cost-projection model using the same methodology as the 2006 Corrado et al. study, but applying US data concerning athletic activity and healthcare costs and projected their results 20 years into the future. They determined that between 2013 and 2023, 170 million high school and college athletes would require screening at an estimated cost of $51 to $69 billion. Over the course of those 20 years, they estimated that 4,813 lives would be saved through screening. The costs-per-lives saved would be in excess of $10 million. The authors recognized the limitation of using a single retrospective study as the focus of their research. They also acknowledge that their expenditure estimation most likely underestimates the actual costs of a mass EKG program secondary to expensive tests being used more liberally in the US, and that African American athletes represent a high percentage of total US athletes and have a higher prevalence of EKG abnormalities that would require additional tests.

Economic impact of application of contemporary EKG evaluation criteria has been studied and the results are promising. Dhutia et al. (2016) prospectively evaluated 4,295 athletes between 14 to 35 years old with history, physical examination, and EKG using the 2010 ESC recommendations. They applied the Seattle criteria and refined criteria retrospectively to the same study group. The need for further specialty referral was determined on the patient's

symptoms, family history, physical examination, or abnormal EKG findings. The study was based in the United Kingdom, and the initial subsidized cost of history, physical examination, and EKG for all 4,295 athletes in the study was $261,025 (approximately $61 per athlete). When the extended cost of specialty evaluation was factored in based on the ESC criteria, the cost increased to $110 per athlete screened, and $35,993 per cardiac condition associated with SCD. Application of the Seattle criteria reduced the same costs to $92 per athlete and $30,251 per SCD associated condition. The refined criteria outperformed both and further reduced cost to $87 per athlete and $28,510 per SCD associated condition. Limitations of the study included adopting a less conservative limit for abnormal QT intervals than outlined by the ESC recommendations when defining abnormal, but this was consistent with local clinical practice. The authors identified this and calculated that following the ESC recommendations would increase cost an additional $47 per athlete. They also identified that using subsidized amounts for the primary assessment and the relatively low cost of specialty care in the United Kingdom could reduce the utility of this study in countries with dissimilar health care models.

Current Recommendations

The American College of Cardiology (ACC) and the American Heart Association (AHA) have a combined recommendation that health care providers use a 14 item checklist when assessing otherwise healthy patients under 25 years old. Table 3 provides the details of the ACC/AHA 14 item checklist. The ACC and AHA do feel that EKG is a useful tool, however they do not recommend screening EKG during pre-participation examination in an otherwise healthy, asymptomatic individual without pertinent family history. Once a thorough personal and family history are gathered by the health care practitioner, the physical examination is conducted. Dynamic testing is recommended to determine if there is a left ventricular outflow obstruction.

Table 3. The 14 Element Cardiovascular Screening Checklist for Congenital and Genetic Heart Diseases

Personal History:

1. Chest pain/discomfort/tightness/pressure related to exertion
2. Unexplained syncope/near-syncope*
3. Excessive exertional and unexplained dyspnea/fatigue or palpitations associated with exercise.
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
6. Prior restriction from participation in sports
7. Prior testing for the heart, ordered by a physician

Family History:

8. Premature death (sudden and unexplained, or otherwise) before age 50 attributable to heart disease in $\geq$ 1 relative
9. Disability from heart disease in close relative <50 years of age
10. Hypertrophic or dilated cardiomyopathy, long-QT syndrome, or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of certain cardiac conditions in family members

Physical examination:

11. Heart murmur**
12. Femoral pulses to exclude aortic coarctation
13. Physical stigmata of Marfan syndrome
14. Brachial artery pressure (sitting position, preferably taken on both arms)

*****Judged not to be of neurocardiogenic (vasovagal) origin; of particular concern when occurring during or after physical exertion.

**Refers to heart murmurs judged likely to be organic and unlikely to be innocent; auscultation should be performed with the patient both in the supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction.

ACC/AHA Release Recommendations for Congenital and Genetic Heart Disease Screenings in Youth (2014). Retrieved from http://www.acc.org/latest-in-cardiology/articles/2014/09/15/14/24/acc-aha-release-recommendations-for-congenital-and-genetic-heart-disease-screenings-in-youth

Internationally, the recommendations and opinions regarding the pre-participation examinations differ slightly. The International Olympic Committee (IOC) has used the Lausanne Recommendations since 2004 which include a more thorough personal and family history assessment, specifically identifying 34 questions versus 10 in the ACC/AHA guidelines. The Lausanne recommendations also include 12 lead EKG after puberty every two years and these EKGs are interpreted using the ESC recommendations. When a patient presents with a positive personal or family history, positive EKG, or physical findings, that patient should be referred to a cardiologist for specialty evaluation.

Discussion

One of the goals of conducting a pre-participation physical examination in athletes it to identify conditions that may predispose them to SCD. While the ACC and AHA recommend against screening EKG in conjunction with pre-participation physical examination, the ESC and IOC have embraced it, and Italy has made it a legal requirement.

In patients requiring pre-participation physical examination, would EKG be an effective screening tool, both medically and economically, to identify conditions that may cause sudden cardiac death in athletes less than 14 years old?

When considering the signs and symptoms of HCM, patients may initially be asymptomatic, but could develop fatigue, peripheral edema, angina, and palpitations. Bisferiens pulse and laterally displaced point of maximal apical impulse may assist a health care provider in identifying HCM, and the characteristic crescendo-decrescendo murmur associated with HCM that increases with dynamic maneuvers should provide a key to diagnosis as well. However, these findings could be easily overlooked on exam and if found alone they are not considered pathognomic and further studies would be required to make a diagnosis of HCM. EKG could be helpful help in identification of ventricular hypertrophy. The Sokolov voltage criteria for LVH is

the industry standard. The S wave depth in V1 is added to the tallest R wave height in V5 or V6. If the sum is greater than 35 millimeters, then the voltage criteria for LVH is met however there are non-voltage criteria to consider as well. Increased R waves in V5 or V6 as well as left sided ST segment depression and T wave inversion must be present in order to diagnose LVH. This criteria is effective for diagnosis in adults, but both pediatric and athletic hearts have characteristics that differ from the adult heart. Because depolarization patterns change during childhood, repolarization is also affected and the T waves are almost always negative from V1 through V5 and become progressively more positive with growth from V5 to V1 (Leger et al., 2016). Additionally, Drezner et al. (2013) suggest that EKG QRS voltage may not be a reliable predictor of LVH secondary to the reliance of measuring the electrical activity of the heart by electrodes on the surface of the body, and that anything between the left ventricular myocardium and the electrodes will affect the voltage.

ARVC may be asymptomatic, though the patient may still be at risk of SCD with increased activity. As the disease progresses, the patient may begin to experience light headedness, syncopal episodes and palpitations. In more advanced disease, signs of heart failure like peripheral edema and shortness of breath begin to develop. Rarely, an early systolic ejection murmur can be auscultated at the fourth intercostal space on the left, but usually there are no abnormal findings on physical examination early in the disease. EKG changes in ARVC include right bundle branch block (RBBB), premature ventricular contractions (PVCs) with left bundle branch block (LBBB) morphology, localized prolonged QRS complexes in the right precordial leads, and T wave inversion in the absence of RBBB in patients older than 12 years old (Leger et al., 2016, p. 6). The Seattle criteria define incomplete RBBB as a normal finding in athletes. In prepubertal children, negative T waves in right precordial leads are physiological and should not be confused with a sign of ARVC (Leger et al., 2016).

The changes with the athlete's heart are well documented and the Seattle criteria integrate these changes into their definitions of common and training related changes. Unfortunately, the creators of the Seattle criteria admit that a significant limitation of the criteria is that they were developed with consideration of EKG interpretation in the context of an asymptomatic athlete age 14-35 (Drezner et al., 2013). Additionally, Drezner et al. (2013) state that the evaluation of EKG abnormalities is ideally performed in consultation with a specialist with knowledge and experience in athlete's heart and disorders associated with SCD in young athletes. While there are numerous studies on the athlete's heart, no current data exists on the physiological cardiac modifications related to exercise in children (Leger et al., 2016). Without further research into these physiologic pediatric changes, and given the known differences of the pediatric heart, a health care provider with limited understanding of overlapping pediatric EKG features who attempts to apply the Seattle criteria to athletes less than 14 years of age could unknowingly compromise patient safety.

The debate surrounding cost efficacy of routine EKG in conjunction with pre-participation exam is founded in the problem of high false positive rates and the cost of further specialist appointments increasing costs on an already heavily burdened health care system. The ESC recommendations for inclusion of EKG screening stem from one highly debated Italian study that claimed significant decline in incidence of SCD in athletes after Italian authorities enacted a law requiring screening EKG as part of the pre-participation physical exam. That study did not conduct a cost analysis. The work of Halkin et al (2012) showed that screening EKG is not cost effective in the US healthcare model. They determined that over a 20 year period, 170 million high school and college athletes would require screening at an estimated cost of $51 to $69 billion, with the understanding that their estimate was conservative based on trends in the US healthcare model and that African American athletes represent a high percentage of

total US athletes and have a higher prevalence of EKG abnormalities that would require additional tests. European studies support cost efficacy of screening EKG, but the data is not portable to the United States secondary to the European subsidized health care model.

Applicability to current practice

Pre-participation physical examination is not uncommon in the primary care provider's office. The purpose of the exam is to identify patients that may have increased risk of participation in sport. The low incidence of SCD in athletes is reassuring, but the deleterious effects of a child's death on a community are profound. Regardless of these emotions, the use of screening EKG by a primary care provider would significantly increase health care costs as well as increase inappropriate specialty referrals. Concerns for the physician when interpreting an athlete's EKG include both missing a dangerous cardiac condition and generating false-positive interpretations that cause needless further investigations, increased economic cost and potentially unnecessary activity restriction for the athlete (Drezner et al., 2013). Additionally, the EKG evaluation and the phenotypical manifestations of SCD pathologies in children differ importantly from the adult athlete, and therefore, EKG screening is likely to prove less effective as a pre-participation tool than in adult athletes (Leger et al, 2016). Further research surrounding activity modulated cardiac modifications in the pediatric population are needed prior to embracing any revised EKG criteria.

The ACC/AHA model has been embraced by all 50 states and school systems will not allow students to participate in athletics without a current exam. The checklist provides an appropriate foundation for pre-participation screening and if there are suspect findings in either personal history, family history, or physical exam, then specialist referral prior to participation is appropriate. Another aspect to consider is provider-directed risk assessment and stratification of proposed activity levels. As previously stated, schools require these physicals prior to

participation in any school activity. Exertional levels vary from activity to activity, and the cardiac stressors associated with chess club would vary considerably from that of long distance running. A common sense approach, paired with sound clinical decision making skills and adherence to the ACC/AHA 14 item checklist, is an effective means of executing pre-participation physical exam in in athletes less than 14 years of age.

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