

Cost and yield of adding electrocardiography to history and physical in screening Division I intercollegiate athletes: A 5-year experience

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BACKGROUND Electrocardiographic screening of intercollegiate athletes is controversial because the costs and yield are not well defined. Both the American Heart Association (AHA) and the European Society of Cardiology (ESC) have different criteria for screening, partly because the populations being screened are different.

OBJECTIVE The purpose of this study was to determine the cost and yield of a 5-year ECG screening program at a United States Division I college.

METHODS At the University of Virginia, all 1,473 competitive athletes over the course of 5 years were screened with history and physical and with ECGs using ESC guidelines with follow-up testing as dictated by clinical symptoms and ECG findings.

RESULTS History and physical alone uncovered five significant cardiac abnormalities. ECGs were abnormal in 275 (19%), resulting in 359 additional tests. Additional testing confirmed eight significant cardiac abnormalities that were not found by history and physical: 1 bicuspid aortic valve, 4 rapidly conducting accessory pathways, 1 long QT patient, 1 with frequent premature ventricular contractions and low ejection fraction, and 1 with frequent premature ventricular contractions but normal ejection fraction. No cases of hypertrophic cardiomyopathy were found. Total cost of

the program was US \$894,870. Cost of history and physical screening alone was \$343,725 or \$68,745 per finding. The marginal cost of adding ECG screening, including resulting tests and procedures, was US\$551,145 or US\$68,893 per additional finding.

CONCLUSION ECG screening of U.S. college athletes can uncover significant cardiac pathology not discovered by history and physical alone. Although ECG screening also results in many false positives resulting in additional tests, the overall cost per diagnosis of adding ECG screening is similar to that of history and physical screening alone.

KEYWORDS Athlete; Atrial fibrillation; Electrocardiography; Screening; Supraventricular tachycardia; Ventricular tachycardia; Wolff-Parkinson-White syndrome

ABBREVIATIONS AHA = American Heart Association; ARVC = arrhythmogenic right ventricular cardiomyopathy; ECG = electrocardiogram; EPS = electrophysiologic study; ESC = European Society of Cardiology; HCM = hypertrophic cardiomyopathy; LVH = left ventricular hypertrophy; MRI = magnetic resonance imaging; PVC = premature ventricular contraction

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Introduction

Preparticipation athletic screening with electrocardiograms (ECGs) is controversial. Whereas European Society of Cardiology (ESC) guidelines¹ recommend preparticipation ECGs in addition to history and physical in all competitive

athletes younger than 35 years, the American Heart Association (AHA)^{2,3} in the United States recommends only history and physical without ECG. The rationale for these two recommendations rests on disparate findings regarding cost and yield. One potential reason for this is that the United States is more ethnically diverse and thus may have a different prevalence of any given genetic condition. For example, in Italy, the incidence of arrhythmogenic right ventricular cardiomyopathy (ARVC) appears to be higher than in the United States.⁴ The U.S. population has grown more diverse, and few data from the modern era are available on the cost and yield of ECG screening in a U.S.

Presented in part at the Heart Rhythm Society Annual Meeting, Denver, Colorado, May 14, 2010. **Address reprint requests and correspondence:** Dr. Srijoy Mahapatra, P.O. Box 800158, University of Virginia, Charlottesville, Virginia 22908. E-mail address: Srijoyism@gmail.com. (Received November 14, 2010; accepted December 13, 2010.)

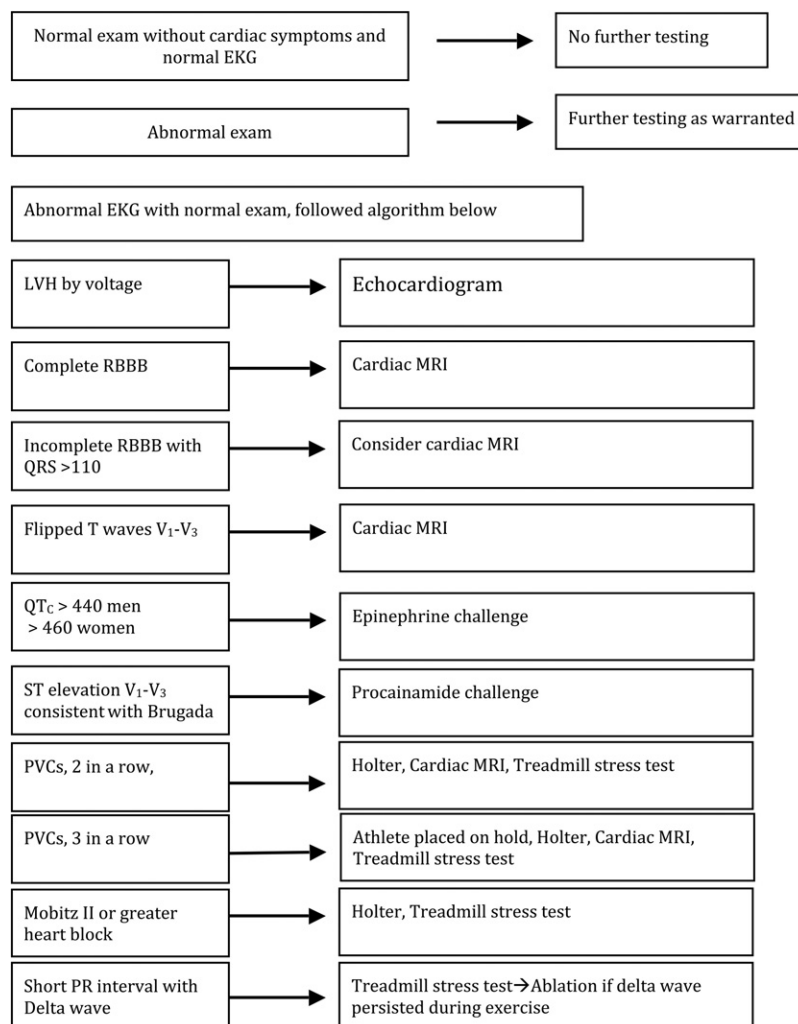


Figure 1 Testing algorithm depicting the electrocardiographic abnormality identified and the subsequent testing performed. LVH = left ventricular hypertrophy; MRI = magnetic resonance imaging; PVC = premature ventricular contraction; RBBB = right bundle branch block.

college population. The few studies that exist include fewer than 600 patients.⁵ At the University of Virginia, we have performed ECG screening in all 1,473 competitive athletes since 2005. We report the yield and cost of an ECG screening program in addition to history and physical in a National Collegiate Athletic Association Division I college athlete population, the most elite of the U.S. college athlete divisions.

Methods

From 2005 to 2010, all 1,473 National Collegiate Athletic Association Division I athletes regardless of sport underwent screening with history and physical and with ECG. The screening was a requirement that was disclosed to athletes and their parents prior to the athletes accepting an athletic position at the University of Virginia. The history and physical was performed by a team of physicians, including two internists with input from a cardiologist. Tests including echocardiograms were ordered as dictated by the history and physical.

An ECG was performed and reviewed by an internist and over-read by a cardiac electrophysiologist. A prospectively

defined protocol base was used to guide additional testing and athletic restrictions (Figure 1). The protocol directed additional testing, including transthoracic echocardiograms, magnetic resonance imaging (MRI), treadmill stress testing, and drug challenges. Abnormalities were defined as newly found conditions requiring invasive procedures, periodic follow-up testing, or exclusion from athletic participation.

On the screening ECG, left ventricular hypertrophy (LVH) criteria were taken from the ESC recommendations and included an R or S wave in a standard lead >2 mV, S wave in lead V_1 or V_2 >3 mV, or an R wave in V_5 or V_6 >3 mV.¹ If patients met these criteria, an echocardiogram was performed either at the University of Virginia or by the athlete's local physician to rule out hypertrophic cardiomyopathy (HCM). In order to differentiate HCM from athletic heart, two cardiologists with extensive experience in echocardiography examined echocardiograms to rule out left atrial dilation, abnormal diastolic function, and left ventricular thickness >12 mm.¹ If the patient met none of these criteria and had no history of exercise-induced syncope, the patient was declared not to have HCM. We did not detain

any athletes to see if LVH regressed. We categorized athletic intensity for the various sports screened using the scaling system as defined by the 36th Bethesda Conference⁶ for static component, but we did not take the dynamic component into account.

We defined incomplete right bundle branch block as an RsR' configuration in lead V₁ with QRS duration <120 ms. QT prolongation was defined as a corrected QT interval in any lead >460 ms in females and >440 ms in males. Short QT interval was defined as QT interval <330 ms.⁷

Although the University of Virginia paid for all history and physicals and ECGs, the athlete's insurance typically paid for the costs of imaging studies. As a result, if insurance companies refused MRI examinations, echocardiograms were done first, then MRI was done. Furthermore, some insurance companies would only pay for echocardiograms done locally, with images sent to the University of Virginia. All students were required to undergo the recommended tests to participate.

Cost analysis

Staff salaries and ECG machines were paid for by the University of Virginia. Using salaries with benefits, we calculated a 5-year personnel cost of \$192,000 for the history and physical screening program (estimated as 10% of the salary and benefits for each of two internists per year for 5 years based on standard University of Virginia quarterly effort reports). The 5-year personnel cost for ECG screening was \$176,100 (5% of additional salary for each internist, 2% per year of an electrophysiologist's salary, and \$40,000 for technicians for 5 years). The University of Virginia purchased two ECG machines for \$10,000 each. We assumed these machines depreciated at 10% per year, giving a 5-year cost of \$14,095.

Although most follow-up studies were paid for by insurance, these costs were included in our cost analysis. To estimate these costs, we used the average collection from private insurance companies at the University of Virginia for the athlete population. Reimbursement for an echocardiogram was \$900, MRI \$1,000, drug challenge \$1,200, Holter studies \$175, treadmill stress test \$300, and electrophysiologic study (EPS) \$25,000. We prospectively defined significant cardiac abnormality as any finding that required invasive therapy or interval follow-up testing (e.g., annual echocardiogram). We then took the total cost of the program (including follow-up tests) and divided it by the number of significant cardiac abnormalities to determine a cost per finding.

Statistical analysis

Statistical analysis using univariate analysis of variance was performed using PASW Statistics 18.0 (SPSS, Chicago, IL, USA). The study was approved by the University of Virginia Institutional Review Board.

Table 1 Athlete demographics

	Male	Female
Number	732	741
Age (years)	19 ± 2	19 ± 2
Body mass index	25 ± 3.9	22 ± 2.9
Systolic blood pressure (mmHg)	126 ± 12	115 ± 11
Diastolic blood pressure (mmHg)	75 ± 9	73 ± 22
Race		
Caucasian	488	561
African-American	153	70
Asian	13	17
Latino	13	20
Native American	1	3
Unreported	63	71
Sport intensity		
1	119	172
2	356	186
3	254	383

Sport intensity was defined as follows—1: low-intensity athletics: cheerleading, volleyball, softball, baseball; 2: intermediate-intensity athletics: track, field hockey, tennis, football, soccer; 3: high-intensity athletics: swimming, lacrosse, rowing.

Results

Athlete demographics

Demographic information of athletes screened is given in Table 1. Of the athletes, 741 (51%) were women. Racial distribution was white (71%), African-American (12.9%), Asian (2%), and Latino (2%). Ninety percent had no prior medical conditions, and 978 (66%) were taking no prescription medicines. Thirty athletes were taking medications (including Adderall) for attention deficit hyperactivity disorder.

Two athletes had undergone ablation procedures in the past, both for symptomatic accessory pathways.

Results of history and physical and subsequent testing

History and physical identified 87 athletes with potential cardiac complaints. Sixty-one of these athletes had undergone full cardiac workups (including echocardiogram) that were negative prior to arrival at the University of Virginia and did not undergo further testing at the University of Virginia. The other 26 athletes had a normal ECG and echocardiogram at the University of Virginia. Five of these athletes had symptomatic palpitations. Event recorders documented narrow complex tachycardia. All five athletes subsequently underwent EPS, which identified three patients with AV nodal reentrant tachycardia, one atrial tachycardia from near the coronary sinus, and one right inferior pulmonary vein tachycardia presenting with atrial fibrillation. All underwent successful ablation. None had a negative EPS. Thus, history and physical alone prompted 26 ECGs, 26 echocardiograms, and 5 EPS that led to the discovery of five potentially significant cardiac abnormalities.

ECG and follow-up test results

ECGs were abnormal in 443 (30%) athletes (Figure 2). The total number of abnormal findings exceeds the total number

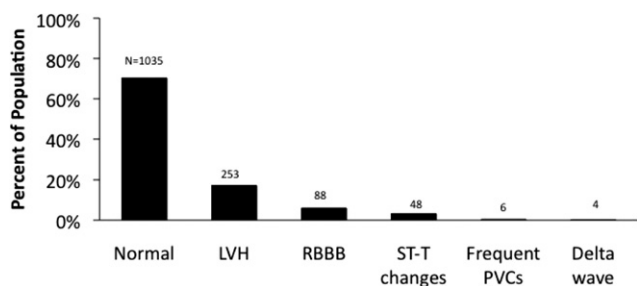


Figure 2 Distribution of abnormalities identified by ECG criteria. LVH = left ventricular hypertrophy; PVC = premature ventricular contraction; RBBB = right bundle branch block.

of abnormal ECGs, as some ECGs had multiple abnormal findings.

We identified 253 athletes with ECG criteria suggesting LVH. All received an echocardiogram (227 at our institution). No echocardiograms had evidence of HCM. However, one athlete was found to have a bicuspid aortic valve with mild aortic regurgitation.

We found no relationship between sport intensity, ethnic origin, and LVH findings on ECG, even though 43% of the athletes were performing high-intensity athletics (383 females and 254 males). Gender was the only factor that predicted LVH.

Incomplete right bundle branch block with inverted T waves in lead V₁, V₂, or V₃ occurred in 88 athletes, including 70 with QRS width >100 ms. We were aggressive in evaluating these ECG changes due to suggestions that T-wave inversions may be very sensitive for ARVC.⁸ A less aggressive approach may have reduced the number of MRIs and thus lowered cost at the slight potential risk of reducing sensitivity. As a result, 44 MRI examinations were performed. Thirty-six patients had echocardiography in lieu of MRI examinations due to insurance coverage or cost issues. The remaining eight patients had prior normal echocardiograms. No patient had evidence of ARVC on MRI, and no

right ventricular dilation was seen on echocardiography. No athletes were excluded from athletic participation based on T-wave inversion.

ECG identified four athletes with short PR intervals and delta waves. All were asymptomatic. All underwent exercise testing that showed exaggeration of the delta wave and thus underwent EPS. During EPS, all four pathways had refractory periods <250 ms and were successfully ablated given data suggesting that short refractory periods are associated with high risk for sudden death.⁹ Two also had inducible atrial fibrillation prior to but not after ablation. None were restricted after ablation.

Six athletes who had multiple premature ventricular contraction (PVCs) on ECG underwent exercise stress testing, Holter monitoring, and MRI. No student had symptoms. Four patients had fewer than 10% PVCs on 24-hour monitoring, normal MRI, and no increase in PVCs with exercise and were allowed to compete without restriction. One athlete had unifocal PVCs 28% of the day by Holter monitoring but had a reduction in PVCs with exercise, a normal ejection fraction, and normal MRI. The PVC had a right bundle branch block morphology with transition at V_{2/3} and was negative in inferior leads and positive in leads I and aVL, making it consistent with left ventricular apical/septal PVC. Because of a normal exercise test, this patient was cleared for athletics but will undergo annual echocardiography to follow the ejection fraction. The sixth patient had multifocal PVCs 48% of the day by Holter monitor, although PVCs decreased with stress testing and ejection fraction was normal by echocardiography. Due to the multifocal PVCs (Figure 3), this athlete was excluded from further athletic participation. Follow-up testing demonstrates that over the course of 1 year, ejection fraction has declined from 65% to 40%. MRI did not demonstrate findings consistent with arrhythmogenic right ventricular cardiomyopathy. This athlete remains excluded from athletic participation and is undergoing further evaluation.



Figure 3 ECG obtained at initial screening of an athlete subsequently found to have polymorphic premature ventricular contractions with exercise and, 1 year later, a reduced ejection fraction. This athlete was barred from further competition and was recommended to refrain from athletics.

A total of four athletes were found to have prolonged QT intervals. These athletes were subjected to epinephrine challenges.^{10,11} QT interval increased in one student and genetic screening confirmed long QT syndrome type 1. This athlete was excluded from further athletic participation. No athletes with short QT intervals were identified.

Three athletes who had coved ST segments on ECG underwent procainamide challenges. None of these studies were positive for Brugada syndrome.

In summary, ECG screening found eight significant cardiac findings, including 4 patients with asymptomatic pathways, 1 with frequent PVCs and low ejection fraction, 1 with frequent PVCs and normal ejection fraction (requiring annual echocardiography), 1 with long QT syndrome, and 1 with bicuspid aortic valve. Two of these students were excluded permanently from athletics.

Summary of screening findings

Overall, history and physical alone suggested arrhythmias in 5 patients that were confirmed on EPS, including 1 right inferior pulmonary vein tachycardia, 3 AVNRT, and 1 coronary sinus atrial tachycardia. Addition of ECG added 8 diagnoses, including 1 bicuspid aortic valve, 4 accessory pathways, 1 long QT syndrome, 1 frequent PVC with normal ejection fraction, and 1 frequent polymorphic PVCs with low ejection fraction. Two students were barred from athletic participation due to asymptomatic ECG findings.

Costs

Overall, the screening program cost our institution or insurance companies \$894,870 and found 13 significant cardiac findings, for a cost per finding of \$68,836.

The cost of the history and physical only program including 26 clinically indicated ECG (\$1,300), 3 Holter monitors (\$525), 26 echocardiograms (\$23,400), excluding those echocardiograms done prior to coming to the University of Virginia, salaries (\$192,000), and 5 EPS with ablations (\$125,000), was \$343,725. With this method alone, we found five abnormalities. Thus, the cost per significant cardiac finding of history and physical alone was \$68,745.

The marginal cost of adding ECGs to screening (including follow-up testing and ablations) was \$551,145, including 1,463 ECGs (cost of \$14,095 for an ECG machine and \$40,000 for technicians), 227 echocardiograms (\$204,500), 44 MRIs (\$44,000), 10 stress tests (\$3,000), 7 drug studies (\$8,400), 6 Holter monitors (\$1,050), 4 ablations (\$100,000), and physician cost (\$136,100). ECG identified 8 asymptomatic findings that required either therapy (4) or regular follow-up (4). Thus, the marginal cost of ECGs per diagnosis was \$68,893.

Discussion

In a 5-year experience, we demonstrated that adding ECGs to an athletic screening program discovers significant pathology in Division I college athletes at a cost per significant cardiac finding that is similar to that of history and physical alone. Although adding ECG to history and physical did

increase the cost, the yield increased proportionately. Thus, the overall program cost per diagnosis was similar to the cost per diagnosis for history and physical or ECG screening alone. This screening program required all athletes to be screened in order to participate in athletics.

Although other studies have been reported, our study differs in significant ways. First, our study is significantly larger with 1,473 patients as opposed to 510 in the largest previously reported study in American collegiate athletes. Second, Baggish et al⁵ evaluated history and physical compared to ECG and echocardiography in a blinded fashion and found that ECG and echocardiography identified more abnormalities than history and physical alone, but prompted more testing. Our report describes the costs and results of an active clinically oriented screening program rather than a research protocol. Thus, history and physical combined with ECG drove follow-up tests. Third, our study ran for a longer time period, which provided more follow-up. Fourth, our program only performed echocardiograms as guided by the ECG and history and physical, which may reduce costs. Fifth, because our program was funded internally or with insurance reimbursement, we were able to provide accurate costs. Finally, our population may represent a more elite population because 65% of our athletes were on athletic scholarship as opposed to studies done at another college that has no athletic scholarships.⁵

Our findings are different than results of a study by Baggish et al⁵ and are more consistent with a prior study on athletes who underwent both ECG and echocardiography in England. This study demonstrated very few athletes with echocardiographic and ECG findings suggestive of HCM using the Sokolow-Lyon criteria.¹² Only 0.09% of athletes in that cohort had both ECG and echocardiographic findings suggestive of HCM. However, how many in the English cohort had ECG criteria suggestive of LVH but echocardiographic findings that did not support the diagnosis is unclear.

We found no patients with HCM, in part perhaps because echocardiograms were not performed in all patients. However, given data that ECGs are highly sensitive for HCM,¹³ it is unlikely that HCM was missed. However, other reports indicate that HCM may develop over prolonged periods of time.¹⁴ To truly diagnose the specificity and sensitivity of the ESC criteria for disease in a U.S. athlete population would require echocardiography for all athletes and potentially could require serial echocardiography. Thus, the true sensitivity and specificity of ECG for cardiac diagnoses in this population remain unclear. Although echocardiography was not performed on all participating athletes, our practice reflects a more common, real world protocol because ECGs are more readily available than are echocardiograms.

We did find one patient with a bicuspid aortic valve who will require periodic echocardiograms. Furthermore, we discovered two patients with frequent PVCs (>10%), including one who developed a low ejection fraction, 4 with asymptomatic accessory pathways, and 1 with genotype-

confirmed long QT syndrome. Other studies have not identified these findings with ECG screening. Because all these findings are rare, it is possible that the differences are due to the small sample size of most studies. In that case, our study is important because it shows the different kinds of findings that can be found on screening ECGs.

In contrast to other studies, we excluded only 2 (0.14%) of 1,473 athletes, which is less than the 3 (0.60%) of 508 excluded by a smaller study.⁵ This difference may result because we provided aggressive electrophysiologic therapy to most patients with abnormalities. If this definitive therapy were not provided, we would have excluded 7 of 1,473 athletes based on ECG criteria alone and 11 of 1,473 based on ECG and symptoms, making the percentage excluded similar to that reported for other studies.

We identified a total of 13 abnormalities that required intervention. Of these, five were suggested by history and physical and were confirmed and treated by further testing. Eight asymptomatic abnormalities were suggested by ECG and confirmed with further testing.

The athlete with polymorphic PVCs and an initially normal heart was excluded from further athletic participation based on the 36th Bethesda conference,¹⁵ and continued participation of the athlete with monomorphic PVCs that declined with exercise is supported by the same guidelines.¹⁵ These guidelines also recommend that athletes with long QT syndrome type 1 may continue to participate in athletic activities, except for swimming. However, like many conditions in athletes, controversy exists regarding this population. NASPE guidelines from 2001 suggest restricting athletes with long QT syndrome type 1.¹⁶ Our patient would have been allowed to continue under the Bethesda guidelines because the QT interval was <480 ms but would have been barred from participation under the NASPE guidelines. We chose to exclude this patient after consulting two other centers and after discussion with the student.

Our testing protocol differs significantly from those previously described in the U.S. athlete population. Our results demonstrate that using the ESC criteria to evaluate U.S. athletes is likely to identify disease but also to generate many false-positive results. However, the bulk of the guidelines on ECG interpretation in this population are based on findings in Italian athletes whose genetic predispositions may be different from those of athletes in the United States. Thus, the yield of these criteria in a U.S. population is unclear. Our study suggests that the criteria produce a significant number of false-positive results but also a significant number of important diagnoses. Our protocol may need to be modified to account for the ECG criteria as described in a recent position paper by Corrado et al.¹⁷

Prior studies have compared LVH findings on ECG in athletes versus nonathletes, but we examined our data to determine whether sport intensity predicted ECG findings of LVH. We did not identify a relationship between ethnic

origin, sport intensity, and LVH findings. Only male gender was a significant predictor of LVH.

It is interesting that in our series of 1,473 young athletes screened by history and physical, family history, and ECG, we found no cases of HCM. Many series report an HCM incidence of 0.2%.¹³ Furthermore, the incidence in African-Americans is higher,¹⁸ and our population included 15% African-Americans. One possibility is that we missed some HCM patients because we did not perform echocardiograms in every student as did Baggish et al.⁵ However, the sensitivity of using these ECG criteria is thought to be as high as that of echocardiography.¹ Furthermore, we performed echocardiograms in 253 patients with LVH on ECG or with any history of syncope or a murmur consistent with HCM on examination and found no HCM. Because HCM often presents when patients are in their 40s¹⁹ and the mean age of our patients was 17 years, we may have missed HCM. Another possibility for having no HCM in our series is that HCM is simply rarer than suggested by other series.

The total cost of our screening program (including follow-up tests) was \$894,870 to diagnose 5 symptomatic and 8 asymptomatic findings, or \$68,836 per diagnosis. The cost of the history and physical program alone would have been \$68,745 per diagnosis. The cost of the ECG portion of the program was \$68,893 per finding. Thus, the costs per diagnosis associated with each strategy were similar. However, adding ECG screening excluded two asymptomatic patients with potentially lethal conditions and allowed us to definitively treat four arrhythmias that were potentially dangerous.⁹ Furthermore, we will continue annual echocardiography on one athlete due to frequent PVCs. It should be noted that the cost for the ECG with history and physical screening program was not \$68,745 + \$68,836 per finding. Instead, the total program cost \$68,836 per diagnosis. It should also be emphasized that our cost estimate was based on estimated salaries and on the average reimbursement for additional tests and not the nominal price. Our cost estimate does not account for treating complications related to invasive procedures because no complications were experienced. Although complications, such as groin hematoma, cardiac perforation, or heart block with resulting pacemaker, can occur with treatment for supraventricular tachycardia, the estimated rate is <1%.²⁰

Our one-time cost estimate per diagnosis is similar to the annual cost in 1993 dollars as treatment dialysis (\$46,000 per year) but is lower in annual cost than therapy for hyperlipidemia (\$154,000).²¹ However, our calculated costs are for both diagnosis and definitive therapy of most of the associated diagnoses. Furthermore, the per diagnosis cost for history and physical and ECG is very similar to the per diagnosis cost for history and physical alone, and screening with history and physical is accepted practice. Although the total cost of the ECG screening program was \$894,870, it appears to compare favorably to the cost of other programs, particularly given that the expenditure is for identifying and treating disease in a young population. It should be empha-

sized that much of our costs were borne voluntarily by the University and may not be feasible for the nation as a whole.

The ECG screening program found eight additional cardiac conditions that could have impacted a student's athletic career or health. Six of these were treatable. Thus, the cost of ECG screening may be justified, especially because the cost per finding for the history and physical alone strategy was similar to that for adding ECG, consistent with study by Wheeler et al.²² Furthermore, the bulk of the cost in the ECG-based screening process was full echocardiograms to exclude HCM in students whose ECG met LVH criteria. It is possible that using limited echocardiograms targeted to septal thickness may drop cost considerably. It is possible that more specific ECG criteria for HCM may reduce the need for echocardiograms. However, our study contained no HCM patients, so we cannot define what ECG criteria would maximize specificity without sacrificing sensitivity.

EPS were the most expensive test. However, this test was the most revealing and allowed for definitive therapy in the four patients who underwent ablation for a potentially life-threatening pathway.⁹ There were no negative EPS.

One concern of screening programs is the legal liability of disqualifying students. Our program was approved by general counsel. Furthermore, parents and athletes were informed that this program differed from screening programs at other U.S. universities prior to them accepting a position at the University of Virginia. Furthermore, in both disqualification cases, financial support of the athletes was maintained.

Study limitations

A previous analysis of athletic screening in the U.S. athlete population involved blinded analysis of history and physical, ECG, and echocardiography.⁵ Our study was not blinded, thus the results of history and physical may have affected the ECG reading. However, this reflects a real-world practice of using multiple pieces of information to guide care. We did not formally follow our athletes beyond college, and it is possible some developed HCM after college.

Our study looked at cost effectiveness as the cost of the total program divided by the total number of significant cardiac findings. However, because the likelihood that these findings would kill an athlete is generally low, we cannot provide any data on cost per life saved. However, our purpose was to compare a history and physical only program to a history and physical followed by ECG program. Neither program found a condition that was definitely fatal. In fact, the two most deadly conditions (low ejection fraction with frequent polymorphic PVCs and long QT syndrome) were found by ECG only with no symptoms. Thus, our data suggest that ECG plus history and physical is as cost effective as history and physical alone.

Conclusion

ECG screening of elite collegiate athletes increased the cost of screening due to false-positive ECGs but identified 8 cardiac abnormalities, 6 of which required intervention and 2 of which required discontinuation of athletic participation. The cost per diagnosis suggested by history and physical alone was similar to the cost per diagnosis identified by ECG.

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