

The 5-Minute Screening Echocardiogram for Athletes

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Background: Echocardiography is an accurate way to identify common cardiac abnormalities that lead to sudden death. We report a screening echocardiogram protocol incorporated into the routine athletic medical assessment for all incoming college freshman athletes.

Methods: A limited 2-dimensional echocardiogram was performed on athletes as part of a routine sports physical examination. The examination was performed by sonographers and senior cardiovascular medicine fellows and interpreted in real time by cardiologists using a 1-page checklist. No images were recorded.

Results: Of the 395 athletes representing 14 sports, 192 were female. The limited 2-dimensional echocardiogram took approximately 5 minutes per athlete. The majority of studies revealed normal findings (84%). A total of 55 had minor abnormalities not requiring follow-up. Five had abnormalities requiring a full echocardiogram and consultation with a cardiologist.

Conclusion: This study demonstrates that a rapid screening echocardiogram is feasible and can be incorporated into the routine athletic medical examination for incoming varsity athletes.

Keywords: Sudden cardiac death, Athlete screening, Preparticipation screening

Sudden death in a previously healthy athlete occurs rarely, but inevitably attracts significant attention because of the tragic magnitude of the event. When these events do occur, the question of how the episode could have been prevented is almost always raised. In the last 5 years, at the University of Wisconsin, Madison, Wis, two athletes have been prohibited from playing their sport because of hypertrophic cardiomyopathy (HOCM). One athlete collapsed during wind sprints, which lead to a cardiac workup, including an echocardiogram demonstrating typical findings of HOCM. The other athlete was found to have an outflow tract murmur suggestive of HOCM during his junior year physical examination, which led to an echocardiogram indicative of HOCM. Fortunately, neither of these individuals had sudden cardiac death as their presenting symptom, nevertheless, the diagnosis of HOCM was not made during their initial preparticipation examination.

Although guidelines for preparticipation cardiovascular screening for athletes exist, a uniform system to screen athletes is not in place in the United States.^{1,2} Screening of collegiate athletes is universally inadequate, with only 26% of 879 college athletic departments having preparticipation forms that inquire about at least 9 of the 12 elements recommended by the American Heart Association (AHA).³ In addition, only 56% of college athletes are asked about a family history of sudden death and only 33 (5%) institutions have examina-

tions conducted by physicians with formal cardiovascular training.³ Similar inadequacies are found among professional and high school athletic teams.^{4,5} As few schools currently screen athletes sufficiently with the history and physical examination, one may argue that a simple screen with an electrocardiogram (ECG) or echocardiogram may act as a safety net. Both time and cost constraints have been implicated as the reason these modalities have not been widely advised or implemented.² Currently, at the University of Wisconsin, Madison, Wis, 10 of the 12 elements recommended by the AHA are screened for with the two deficiencies being a personal history of fatigue or unexplained dyspnea. An ECG is not obtained as part of the screening protocol. Despite a thorough preparticipation history and physical examination, two athletes with HOCM were missed. Given our recent experience, we implemented a cardiovascular screening program with the use of echocardiograms in all incoming freshman athletes.

METHODS

From June 2005 to October 2006, a limited 2-dimensional (2D) echocardiogram (L2DE) was performed on athletes at our university as part of a routine sports physical. The examination was performed by sonographers and senior cardiovascular medicine fellows and interpreted in real time by a cardiologist using a 1-page checklist. Sonographers all had significant imaging experience and the fellows involved in the screening process had all met or exceeded level 2 COCATs requirements for echocardiography. No images were recorded and ECG leads were not placed on the patient. Any individual with an abnormal finding was referred to the adult echocardiography laboratory for a full study and referral to a cardiologist. Examinations were performed at one of 3 locations: one of the campus athletic facilities, an off-site cardiac rehabilitation and outpatient sports medicine center, or the adult echocardiography laboratory at the univer-

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sity hospital and clinics. Portable machines, including a General Electric Vivid i (General Electric Medical Systems, Milwaukee, WI), Acuson Cypress (Siemens Acuson, Mountain View, CA), and SonoSite Titan (SonoSite, Inc., Bothell, WA), were used at the remote locations. For the examinations performed in the adult echocardiography laboratory one of 3 regular systems was used: a Siemens Acuson Sequoia, General Electric Vivid 7, or Philips Sonos 5500.

The L2DE consisted of the parasternal long-axis, parasternal short-axis at the level of the aortic valve and the mid-left ventricle (LV), an apical 4-chamber, and an apical 5-chamber view. In the parasternal long-axis view, the ascending aorta was investigated for size and shape; if dilatation was suggested, measurements were performed. The aortic and mitral valves were imaged for morphology and motion, and color Doppler was performed to evaluate for regurgitation. The LV wall motion, size, and wall thickness were examined and measurements were carried out if LV hypertrophy was suggested. In the parasternal short-axis view, the aortic valve was again inspected for leaflet morphology and motion. The takeoff of the right coronary artery and left coronary artery were identified to determine whether position was normal. The pulmonic valve was identified and continuous wave Doppler was performed to evaluate forward flow velocity as a screen for pulmonic stenosis. In the midpapillary muscle level, the LV was evaluated for contractility, wall thickness, and asymmetry of wall thickness; again, if abnormalities were suggested, measurements were performed. In the apical 4-chamber view, the mitral and tricuspid valves were inspected for motion and morphology. Screening for regurgitation was done with color Doppler. If tricuspid regurgitation was present, an attempt to get a tricuspid regurgitant gradient as a screen for pulmonary hypertension was done. In this view, the right ventricle (RV) was scrutinized for size, function, and evidence of RV dysplasia. The LV was again evaluated for size and function. Finally, a 5-chamber view was obtained and forward flow across the aortic valve was interrogated with continuous wave Doppler and color Doppler for aortic insufficiency.

RESULTS

In all, 395 athletes representing 14 sports were screened. A total of 192 (49%) were female. The age ranged from 17 to 23 years with a mean age of 19 years. The L2DE took approximately 5 minutes per athlete. The origin of the left coronary artery was identified in 394 (99%) and the origin of the right in 380 (96%). The majority of studies (333 [84%]) revealed normal findings. In all, 55 (14%) had minor abnormalities such as mild tricuspid regurgitation, an upper normal-sized RV, or mild mitral regurgitation and did not require specific follow-up. The abnormalities identified can be found in Table. Other minor abnormalities included mitral valve prolapse without significant regurgitation, an atrial septal defect, and atrial septal aneurysms. No coronary abnormalities were identified. Five had abnormalities that required a full echocardiogram and a clinic visit with a cardiologist before participation. These abnormalities included: two bicuspid aortic valves—one with moderate aortic insufficiency, the other with mild; one pulmonic stenosis; one thickened mitral valve with mild mitral regurgitation; and one mild cardiomyopathy. The valvular disease was estimated with a combination of color Doppler parameters and velocities. Valve area calculations were not done on the screening echocardiograms. In the case of the mild cardiomyopathy, the ventricle was measured during the screening examination as the ventricle looked dilated. End-diastolic dimension was 57 mm and end-systolic dimension was 42 mm. The ejection

Table Abnormalities identified on screening echocardiogram

| Abnormality | N (%) |
|-------------------------|-----------|
| Mitral regurgitation | |
| Trivial | 66 (16.7) |
| Mild | 11 (2.8) |
| Aortic insufficiency | |
| Trivial | 11 (2.8) |
| Mild | 3 (0.8) |
| Moderate | 1 (0.3) |
| Tricuspid regurgitation | |
| Trivial | 38 (9.6) |
| Mild | 19 (4.8) |
| Pulmonic insufficiency | |
| Trivial | 8 (2.0) |
| Mild | 8 (2.0) |
| Moderate | 1 (0.3) |
| Pulmonic stenosis | |
| Moderate | 1 (0.3) |
| Mitral valve prolapse | 5 (1.3) |
| Atrial septal aneurysm | 2 (0.5) |
| Atrial septal defect | 1 (0.3) |
| Bicuspid aortic valve | 2 (0.5) |
| Mild cardiomyopathy | 1 (0.3) |
| RV mildly enlarged | 8 (2.0) |

RV, Right ventricular.

fraction was visually estimated to be 50%. The patient had a follow-up echocardiogram in the adult echocardiogram laboratory where ejection fraction was visually estimated at 55% and a quantified ejection fraction of 57%. The end-diastolic dimension was found to be 57 mm and end-systolic dimension was 38 mm. The patient was seen by a cardiologist (P. S. R.) and comparison was made to an echocardiogram performed at a different institution 2 years prior. The previous echocardiogram also showed minimal dilatation and an ejection fraction at the lower limits of normal. Given that the student had been vigorously training 2 years ago, whereas his current level of activity was significantly less because of recent athletic injury, the mild dilation and low normal ejection fraction were not likely caused by an athletic heart. In the 5 above-mentioned cases, the full echocardiogram and clinical evaluation confirmed the L2DE findings. No athletes were prohibited from participation in their sport as a result of the findings.

DISCUSSION

In North America the most common cause of sudden cardiac death in athletes is HOCM.⁶ One study that reviewed deaths in military recruits from 1977 through 2001 found that there were 126 sudden nontraumatic deaths and 86% of these were related to exercise.⁷ Half were a result of an identifiable cardiac abnormality, of those the most common abnormalities were anomalous coronary arteries, myocarditis, and HOCM.⁷ Echocardiography is an excellent way to identify the majority of the common abnormalities that lead to sudden cardiac death in young athletes. In our evaluation of almost 400 collegiate level athletes, we were able to identify the left main coronary artery in 99% of cases and the right coronary in 96%. This is comparable with a similar preparticipation evaluation of University of Maryland (College Park, MD) athletes where they identified 100% of left main coronary arteries by echocardiogram.⁸ Asymmetric septal hypertrophy, systolic anterior motion of the mitral valve, and outflow

tract obstruction—all features of HOCM—are readily identifiable by echocardiography. Other abnormalities, such as RV dysplasia, can also be identified using echocardiography although the diagnosis of RV dysplasia requires that ECG criteria also be present.

Although no significant cardiac abnormalities were identified in our study, we were able to demonstrate that a large number of athletes can be screened with echocardiograms in an efficient manner. Time and cost have been implicated as reasons for not widely adapting a preparticipation screening examination, which includes echocardiography. In our experience, the use of a limited, but thorough 2D echocardiogram took approximately 5 minutes to perform and interpret. Real-time interpretation of the images by an experienced reader allowed for an efficient turnover time. The examination and interpretation time did not vary substantially from the adult echocardiography laboratory to the off-site locations despite the use of portable machines at the off-site locations. Time and cost was saved by not using ECG leads. The screening program fell within the regular duties of the attending physicians, cardiovascular medicine fellows, and sonographers; therefore, additional liability insurance or reimbursement was not necessary. The hospital received \$35 per echocardiogram from the athletic department for the use of space and equipment. Similar experiences have been reported, with the cost of a limited screening echocardiogram to be between \$7.34 and \$15 using volunteer physicians and free equipment but paid technologists.^{9,10} Others have reported a cost of \$350 to \$400 for athletic screening echocardiograms.^{11,12} The variability is in the ability of the institution to recruit volunteer physicians, sonographers, and low-cost equipment.

The use of ECG screening has been recommended, as it is less costly.² In Italy, for example, all athletes have a screening ECG, which has been postulated as the reason that the number of sports-related sudden cardiac deaths has fallen.¹³ In Italy, the most common cause of sudden death among young athletes is arrhythmogenic RV cardiomyopathy.¹⁴ This is a fairly rare cause of sudden death in the United States and, therefore, their success with an ECG screening program may not translate to success in other countries where the spectrum of causes of sudden death is different.¹⁵ Others believe that using a L2DE as we report may be competitive, with respect to time and money investment, with a 12-lead ECG. It is estimated that a cost of \$74 for a L2DE would be comparable with that of 12-lead ECG screening.¹¹ In determining the cost of a 12-lead ECG, one must also take into account the high incidence of false-positive findings that may lead to further testing. One study of 1005 trained athletes found that 40% of their ECGs raised a suspicion for cardiac disease.¹⁶ One center has in fact stopped using screening 12-lead ECGs as the cost and time of the L2DE is comparable.¹⁰

Our experience using L2DE as a screening modality for preparticipation in collegiate level sports demonstrates that a large number of athletes can be efficiently and accurately screened for many of the common cardiac abnormalities that lead to sudden cardiac death. At a large university such as ours, this type of screening program is logistically easy because the university faculty practice on the same campus as the athletes. It may be more difficult for other programs where athletes are in a separate location. With more widespread use

of portable echocardiogram machines, similar screening of athletic programs may be feasible in a wide variety of locations.

REFERENCES

1. Maron BJ, Thompson PD, Puffer JC, McGrew CA, Strong WB, Douglas PS, et al. Cardiovascular preparticipation screening of competitive athletes: a statement for health professionals from the sudden death committee (clinical cardiology) and congenital cardiac defects committee (cardiovascular disease in the young), American Heart Association. *Circulation* 1996;94:850-6.
2. Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, Fromer M, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association task force and the European Society of Cardiology committee for practice guidelines (writing committee to develop guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death). *J Am Coll Cardiol* 2006;48:e247-346.
3. Pfister GC, Puffer JC, Maron BJ. Preparticipation cardiovascular screening for US collegiate student-athletes. *JAMA* 2000;283:1597-9.
4. Harris KM, Sponsel A, Hutter AM Jr, Maron BJ. Brief communication: cardiovascular screening practices of major North American professional sports teams. *Ann Intern Med* 2006;145:507-11.
5. Glover DW, Maron BJ. Profile of preparticipation cardiovascular screening for high school athletes. *JAMA* 1998;279:1817-9.
6. Maron BJ, Zipes DP. Introduction: eligibility recommendations for competitive athletes with cardiovascular abnormalities-general considerations. *J Am Coll Cardiol* 2005;45:1318-21.
7. Eckart RE, Scoville SL, Campbell CL, Shry EA, Stajduhar KC, Potter RN, et al. Sudden death in young adults: a 25-year review of autopsies in military recruits. *Ann Intern Med* 2004;141:829-34.
8. Maron BJ, Bodison SA, Wesley YE, Tucker E, Green KJ. Results of screening a large group of intercollegiate competitive athletes for cardiovascular disease. *J Am Coll Cardiol* 1987;10:1214-21.
9. Weidenbener EJ, Krauss MD, Waller BF, Taliencio CP. Incorporation of screening echocardiography in the preparticipation exam. *Clin J Sport Med* 1995;5:86-9.
10. Murray MJ, DeRuyter ML, Harrison BA. Opioids and benzodiazepines. *Crit Care Clin* 1995;11:849-73.
11. Fuller CM. Cost effectiveness analysis of screening of high school athletes for risk of sudden cardiac death. *Med Sci Sports Exerc* 2000;32:887-90.
12. Lewis JF, Maron BJ, Diggs JA, Spencer JE, Mehrotra PP, Curry CL. Preparticipation echocardiographic screening for cardiovascular disease in a large, predominantly black population of collegiate athletes. *Am J Cardiol* 1989;64:1029-33.
13. Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *JAMA* 2006;296:1593-601.
14. Corrado D, Basso C, Rizzoli G, Schiavon M, Thiene G. Does sports activity enhance the risk of sudden death in adolescents and young adults? *J Am Coll Cardiol* 2003;42:1959-63.
15. Thompson PD, Levine BD. Protecting athletes from sudden cardiac death. *JAMA* 2006;296:1648-50.
16. Pelliccia A, Maron BJ, Culasso F, Di Paolo FM, Spataro A, Biffi A, et al. Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation* 2000;102:278-84.