

although retrospective in nature, should not be overlooked, considering that amiodarone increases the defibrillation threshold of ICDs and may interfere with antitachycardia pacing by slowing the rate of ventricular tachycardia.<sup>4</sup> Therefore, until data from a prospective trial become available, antiarrhythmic therapy — amiodarone in particular — should not be considered preferable to catheter ablation in patients with ICDs who have repetitive ventricular arrhythmias.

Michele Coceani, M.D.

National Research Council Institute of Clinical Physiology  
56124 Pisa, Italy  
michecoc@ifc.cnr.it

1. Estes NA III. Ablation after ICD implantation — bridging the gap between promise and practice. *N Engl J Med* 2007; 357:2717-9.
2. Connolly SJ, Dorian P, Roberts RS, et al. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: the OPTIC Study: a randomized trial. *JAMA* 2006;295:165-71.
3. Worck R, Haarbo J, Thomsen PE. Electrophysiological study and 'slow' ventricular tachycardia predict appropriate therapy: results from a single-centre implantable cardiac defibrillator follow-up. *Europace* 2007;9:1048-53.
4. Goldschlager N, Epstein AE, Naccarelli GV, et al. A practical guide for clinicians who treat patients with amiodarone: 2007. *Heart Rhythm* 2007;4:1250-9. [Erratum, *Heart Rhythm* 2007;4:1590.]

## Athletes with Repolarization Abnormalities

**TO THE EDITOR:** Pelliccia et al. (Jan. 10 issue)<sup>1</sup> report long-term follow-up data on 81 athletes with deeply inverted T waves on electrocardiograms (ECGs), who had no apparent cardiac disease, selected from a cohort of 12,550 trained athletes. Among these 81 athletes, 5 (6%) ultimately proved to have cardiomyopathies, and 3 of the 5 had hypertrophic cardiomyopathy. We would like to reconsider the relation between deeply inverted T waves and hypertrophic cardiomyopathy in apparently healthy young persons, whether or not they are athletes. In our opinion, myocardial hypertrophy does not represent the true hypertrophic cardiomyopathy phenotype. Geisterfer-Lowrance et al.<sup>2</sup> have demonstrated that in a mouse model of familial hypertrophic cardiomyopathy, myocyte disarray precedes the development of hypertrophy, whereas ECG changes appear only when the disarray becomes evident. Clinical data support this finding.<sup>3</sup> Tissue Doppler and strain-rate imaging may be useful in preclinical diagnosis of the disease.<sup>4,5</sup> The presence of deeply inverted T waves on an ECG in an otherwise healthy young person may represent hypertrophic cardiomyopathy, regardless of anatomy, and probably should be considered a reason for disqualification from competitive sports.

Georgios K. Efthimiadis, M.D.  
Soulтана Meditskou, M.D.  
Georgios E. Parcharidis, M.D.

Aristotle University of Thessaloniki  
54636 Thessaloniki, Greece  
efthymos@med.auth.gr

1. Pelliccia A, Di Paolo FM, Quattrini FM, et al. Outcomes in athletes with marked ECG repolarization abnormalities. *N Engl J Med* 2008;358:152-61.
2. Geisterfer-Lowrance AA, Christe M, Conner DA, et al. A mouse model of familial hypertrophic cardiomyopathy. *Science* 1996; 272:731-4.
3. McKenna WJ, Stewart JT, Nihoyannopoulos P, McGinty F, Davies MJ. Hypertrophic cardiomyopathy without hypertrophy: two families with myocardial disarray in the absence of increased myocardial mass. *Br Heart J* 1990;63:287-90.
4. Ganame J, Mertens L, Eidem BW, et al. Regional myocardial deformation in children with hypertrophic cardiomyopathy: morphological and clinical correlations. *Eur Heart J* 2007;28: 2886-94.
5. Nagueh SF, Bachinski LL, Meyer D, et al. Tissue Doppler imaging consistently detects myocardial abnormalities in patients with hypertrophic cardiomyopathy and provides a novel means for an early diagnosis before and independently of hypertrophy. *Circulation* 2001;104:128-30.

**TO THE EDITOR:** Pelliccia et al. report two cardiac arrests, one of which was fatal, among 81 athletes with marked repolarization abnormalities but normal echocardiograms over a mean follow-up period of 9 years. These findings undermine the reassurance provided by a normal echocardiogram in the setting of ECG repolarization abnormalities and the concept of benign "athlete's heart." Pelliccia and his colleagues have previously advised screening with ECG before participation in athletic events because of the attendant risk of sudden death from cardiac causes in this population (1 in 100,000). Why then do they recommend continued annual surveillance rather than disqualification from competitive sports for this subgroup of athletes who have a risk of sudden death from cardiac causes or aborted cardiac arrest of 1 in 40? Surveillance echocardiography failed to identify the patient who died of right ventricular

cardiomyopathy, and the echocardiographic findings were not convincingly abnormal in the patient who received a diagnosis of hypertrophic cardiomyopathy after a cardiac arrest with a 13-mm septum. It appears unlikely that echocardiography will provide adequate surveillance in this subgroup of athletes, but if surveillance rather than disqualification is being considered, other methods such as magnetic resonance imaging may be required.

Joseph M. Galvin, F.R.C.P.I.

Connolly Hospital  
Dublin 15, Ireland  
joseph.galvin@ireland.com

**TO THE EDITOR:** We commend Pelliccia and colleagues for highlighting the importance of clinical surveillance in athletes with abnormal ECGs. However, the risk of cardiomyopathy in this cohort may be even higher than suggested.

Echocardiography is frequently suboptimal for identifying apical and right ventricular abnormalities; using supplementary techniques may therefore improve the diagnostic yield. Tissue Doppler studies can predict the development of hypertrophic cardiomyopathy in patients with subclinical disease.<sup>1</sup> Contrast echocardiography and cardiovascular magnetic resonance (CMR) imaging provide better visualization of apical abnormalities in noncompaction cardiomyopathy and apical hypertrophic cardiomyopathy<sup>2</sup> and of right ventricular thickening in hypertrophic cardiomyopathy.<sup>3</sup> Accurate identification of right ventricular enlargement and dysfunction is particularly important in arrhythmogenic right ventricular cardiomyopathy, and tissue characterization by CMR imaging may confirm fatty infiltration.<sup>4</sup> Contrast-enhanced CMR imaging may provide additional diagnostic and prognostic information in hypertrophic cardiomyopathy and dilated cardiomyopathy.<sup>2</sup>

Given the inherited component of many cardiomyopathies, a positive family history may also clinch the diagnosis, even when clinical manifestations are absent.

Therefore, family screening and additional imaging are prudent in young persons with ECG abnormalities in whom standard evaluation fails to yield a diagnosis.

Jayanth R. Arnold, B.M., B.Ch.  
Theodoros D. Karamitsos, M.D., Ph.D.  
Steffen E. Petersen, M.D., D.Phil.

University of Oxford  
Oxford OX3 9DU, United Kingdom  
ranjitarold@yahoo.co.uk

1. Nagueh SF, McFalls J, Meyer D, et al. Tissue Doppler imaging predicts the development of hypertrophic cardiomyopathy in subjects with subclinical disease. *Circulation* 2003;108:395-8.
2. Assomull RG, Pennell DJ, Prasad SK. Cardiovascular magnetic resonance in the evaluation of heart failure. *Heart* 2007;93:985-92.
3. Maron MS, Hauser TH, Dubrow E, et al. Right ventricular involvement in hypertrophic cardiomyopathy. *Am J Cardiol* 2007;100:1293-8.
4. Tandri H, Macedo R, Calkins H, et al. Role of magnetic resonance imaging in arrhythmogenic right ventricular dysplasia: insights from the North American Arrhythmogenic Right Ventricular Dysplasia (ARVD/C) study. *Am Heart J* 2008;155:147-53.

**THE AUTHORS REPLY:** We disagree with certain inferences that Efthimiadis et al. make from our data. First, there is compelling evidence, assembled over a period of 50 years, that the most consistent manifestation of the hypertrophic cardiomyopathy phenotype is left ventricular hypertrophy.<sup>1</sup> To argue otherwise is to unnecessarily inject confusion into the clinical assessment of this disease. However, it is also true, as shown in our study and several others, that other manifestations (including ECG alterations and left ventricular filling abnormalities) can precede the appearance of left ventricular hypertrophy in some young people who inherit a mutant gene for hypertrophic cardiomyopathy.

Second, and more important, it was not our intention to suggest that an abnormal ECG repolarization pattern should itself justify disqualification from competitive sports. This would be inconsistent with both the 36th Bethesda Conference<sup>2</sup> and the European Society of Cardiology<sup>3</sup> recommendations, given the uncommon occurrence of these ECG patterns in the vast athlete population, as well as the rarity with which these abnormalities predict future cardiac disease and events. However, we do suggest for such athletes a prudent strategy of systematic surveillance with echocardiography and probably CMR imaging.

Galvin and Arnold et al. have raised an issue similar to that raised by Efthimiadis et al., but with additional questions concerning the most

effective strategies for long-term assessment of athletes who may have a proclivity for delayed development of the hypertrophic cardiomyopathy phenotype or the arrhythmogenic right ventricular cardiomyopathy phenotype. This is an important point that deserves emphasis. For example, conventional echocardiographic imaging may be incapable of reliably confirming the diagnosis of hypertrophic cardiomyopathy in some patients. Indeed, CMR imaging may identify segmental hypertrophy in either the apex or the anterolateral left ventricular free wall<sup>4</sup> that is undetected by echocardiography. Furthermore, as indicated by Arnold et al., tissue characterization by CMR imaging may also identify fatty infiltration of the right ventricle, allowing for the diagnosis of arrhythmogenic right ventricular cardiomyopathy. Therefore, contemporary follow-up in this selected subgroup of trained athletes with marked repolarization changes on ECG should include both echocardiography and CMR imaging.

Antonio Pelliccia, M.D.

Institute of Sports Medicine and Science  
00197 Rome, Italy  
ant.pelliccia@libero.it

Barry J. Maron, M.D.

Minneapolis Heart Institute Foundation  
Minneapolis, MN 55407

1. Maron BJ, McKenna WJ, Danielson GK, et al. American College of Cardiology/European Society of Cardiology clinical expert consensus document on hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2003;42:1687-713.
2. Maron BJ, Zipes DP. 36th Bethesda Conference: eligibility recommendations for competitive athletes with cardiovascular abnormalities. *J Am Coll Cardiol* 2005;45:1312-75.
3. Pelliccia A, Fagard B, Bjørnstad HH, et al. Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J* 2005;26:1422-45.
4. Rickers C, Wilke NM, Jerosch-Herold M, et al. Utility of cardiac magnetic resonance imaging in the diagnosis of hypertrophic cardiomyopathy. *Circulation* 2005;112:855-61.

## Neurogenic Orthostatic Hypotension

**TO THE EDITOR:** In his Clinical Practice article on neurogenic orthostatic hypotension, Freeman (Feb. 7 issue)<sup>1</sup> notes that the definition of orthostatic hypotension encompasses a drop in blood pressure during the first 3 minutes of standing. He states that when evaluating a patient, however, the blood pressure should be measured with the patient in the supine position and at least 3 minutes after the patient stands up. These two statements appear to contradict each other.

Mark Joy, M.D.

Veterans Affairs New York Harbor Healthcare System  
Brooklyn, NY 11209  
mark.joy@va.gov

1. Freeman R. Neurogenic orthostatic hypotension. *N Engl J Med* 2008;358:615-24.

in systolic blood pressure of at least 20 mm Hg or a reduction in diastolic blood pressure of at least 10 mm Hg within 3 minutes of standing. In clinical practice, to diagnose orthostatic hypotension, it is recommended that blood pressure be measured with the patient in the supine position and at least once when the patient has been standing for 3 minutes. More severe orthostatic hypotension may be diagnosed by measuring blood pressure within 3 minutes after standing. Delayed orthostatic hypotension may be diagnosed by measuring blood pressure after more than 3 minutes of standing.

Roy Freeman, M.B., Ch.B.

Harvard Medical School  
Boston, MA 02115  
rfreeman@bidmc.harvard.edu

**THE AUTHOR REPLIES:** Orthostatic hypotension is defined by a consensus of experts as a reduction