With great interest I read the article ‘Reduced right ventricular ejection fraction in endurance athletes presenting with ventricular arrhythmias: a quantitative angiographic assessment’ by Ector et al.1 If invasive methods are employed, a thorough evaluation of the morphology and functions of the right ventricle (RV) may be difficult due to the complex geometry and anatomy. Examination of RV geometry in athletes poses an additional puzzle because of the gross changes of both ventricular shape and function associated with athletic training.2 Based on the current literature, the RV plays two major roles in cardiovascular function and prognosis: both good and bad.

In its deleterious role, dysfunction and pathologies of the RV lead to series cardiac arrhythmias and a poor prognosis.3 Studies have shown that the patient’s degree of ventricular geometric distortion correlates with RV or left ventricle (LV) pressure and volume overload, which often affects each other.4 It has also been shown that primary RV systolic dysfunction can lead to secondary changes in the systolic and diastolic function of the LV.4 Although the authors of this study presented ventricular arrhythmias and RV dysfunction, the authors did not test the effects of LV functions on RV functions by multivariate analysis. RV changes without evaluating any real time LV alterations this may lead to difficulties. Biffi et al.5 found that a reduction in the frequency of ventricular tachycardia after deconditioning and the absence of cardiac events in follow-up support the benign clinical nature of these rhythm disturbances as just another expression of athlete’s heart. But an important question that has not been asked is what prognostic role could ventricular arrhythmias play in athletes without cardiovascular abnormalities?

In its beneficial role, a better functional RV in the maintenance of endurance capacity and its function may contribute to enhanced endurance capacity via increased LV filling and performance.6 Both the pulmonary and the systemic circulation must show a performance change to meet a circulatory demand of extensive and prolonged physical exercise.6 Both the RV and the LV must alter its performance to meet the increased circulatory demands of exercise. RV adaptation to intense and prolonged physical exercise may be expected to increase the RV cavity dimensions, with a concomitant increase in RV contractile reserve.6,7 Interestingly, RV cavity diameters in endurance-training athletes were higher than in strength – this phrase/sentence also needs a numerical reference training athletes. It is proposed that volume overload exercise training (isometric type) causes a greater increase in the diameter of the RV cavity compared with volume overload exercise training (isometric type).7 Furthermore, D’Andrea et al.8 recently reported that there was a close association between LV end-diastolic diameter and peak early diastolic velocity of RV in the overall master athletes. In addition, early-diastolic right ventricular myocardial function was a powerful independent determinant of both LV stroke volume and of maximal workload achieved during physical effort.

In conclusion, although it is known that alterations of LV functions affect both the global functioning and prognosis, several important questions remain for athletes who have suspicious right ventricular dysfunction, including how should the prognosis be evaluated and what are the recommendations available?

References

Erdem Kasikcioglu
Department of Sports Medicine
Istanbul Faculty of Medicine
Istanbul University
Istanbul 34640
Turkey
Tel: +90 216 340 5316
fax: +90 216 340 5316
E-mail address: ekasikcioglu@yahoo.com

doi:10.1093/eurheartj/ehm496

Online publish-ahead-of-print 25 October 2007

Redefining cardiomyopathies: the role of cardiovascular magnetic resonance imaging

We read with great interest the article by Monserrat et al.1 published in a recent issue of European Heart Journal. The authors report on the E101K mutation in the alpha-cardiac actin gene that has previously been reported as a cause of apical hypertrophic cardiomyopathy (HCM) by the same group.2 They now hypothesize that this mutation may be present with the same or a different phenotype. Having screened 247 families with HCM, left ventricular non-compaction (LVNC), and dilated cardiomyopathy, the authors identified five index patients: one with LVNC and four with apical HCM. A total of 46 mutation carriers with an abnormal LV phenotype were identified; they were associated mainly with LVNC but also with apical HCM and septal defects. Inevitably, the question arises as to whether this represents an overlapping phenotype between apical HCM and LVNC.

Monserrat et al.1 admit the choice of imaging modality as one of their limitations, but we would like to elaborate on this point. In their study, they used transthoracic echocardiography without contrast to perform family screening and to characterize ventricular morphology. Conventional transthoracic echocardiography has inherent limitations, particularly in the visualization of the apex where hypertrophy or trabeculations may be seen associated with the E101K mutation. Cardiovascular magnetic resonance (CMR) is a powerful non-invasive imaging modality capable of assessing global and regional functions and performing tissue characterization with and without contrast administration.3 CMR can image in any plane, and the introduction of steady-state free