Cardiac adaptation to high-intensity exercise is well recognized and often termed the athlete’s heart. The characteristic features of athlete’s heart are increased left ventricular (LV) volume, increased LV wall thickness and mass, resting bradycardia, and ECG repolarization abnormalities. These changes are particularly associated with endurance training causing eccentric LV hypertrophy, whereas significant resistance training (eg, weight lifters) may lead to concentric LV hypertrophy. Although somewhat controversial, it is widely accepted that in the majority of athletes, these changes occur as a physiological response to exercise training; LV volumes and mass regress in subjects who stop training and decondition. However, there is an increasing number of reports in older veteran athletes that may suggest that some people who exercise intensely over many years develop myocardial fibrosis and have an increased risk of arrhythmias, particularly atrial fibrillation.

**See Article by McDiarmid et al**

Difficulties may also arise in differentiating athlete’s heart from pathological cardiac conditions that are associated with sudden death, such as dilated and hypertrophic cardiomyopathy (HCM), in certain situations. Many countries and professional sports teams have implemented preparticipation sports screening programs, and because of the high prevalence of ECG abnormalities, a significant minority of athletes are referred for echocardiographic imaging. Similarly, healthy athletes with atypical cardiac symptoms are more likely to be referred for cardiac investigations because of high frequency of repolarization abnormalities. Cardiac magnetic resonance imaging (CMR) may be particularly useful in such situations because of the superiority over echocardiography to detect hypertrophy in the lateral segments and apical HCM. Another major strength of CMR is the ability to characterize the myocardium, particularly the detection of replacement myocardial fibrosis with the technique of late-gadolinium enhancement that is seen in many patients with HCM and to a lesser extent in dilated cardiomyopathy. Late-gadolinium enhancement tends to represent a late stage of established fibrosis and has a limited role in detecting the preceding stage of increasing interstitial fibrosis. Native (before contrast) and post–contrast T1 mapping allow quantification of myocardial extracellular volume (ECV), which correlates with diffuse interstitial fibrosis and is highly robust and reproducible. In addition, native T1 mapping, which reflects the water content of tissues, is a useful imaging tool in many disease states, particularly the diagnosis and monitoring of patients with cardiac amyloidosis. More recently, native T1 mapping has been shown to potentially improve the ability of CMR to differentiate patients with early-stage dilated cardiomyopathy from physiological adaptation to exercise in veteran athletes with low-normal ejection fraction on echocardiography. Although extracellular expansion leads to higher ECV measurements, intracellular volume expansion leads to reduced ECV measurement, such as in Anderson–Fabry disease.

In this issue of *Circulation: Cardiovascular Imaging*, McDiarmid et al compared native T1 and ECV measurements in 30 younger, white male endurance athletes with 15 healthy age-matched controls. As expected, the athletes had higher LV volumes, mass, and similar ejection fractions to the controls. However, for the first time, they have also shown that the increase in LV mass is because of increased cellular mass, and there was a relative decrease in ECV (22.5%±2.6 versus 24.5%±2.2; P=0.02) compared with controls. There was also a small statistically significant reduction in native T1 time in the athletes compared with controls. Consistent with previously published studies, they also show a significant correlation between LV mass index, LV end-diastolic volume index, and VO_{2max}. The authors also present data showing that increasing degrees of fitness (measured objectively by VO_{2max}) increase linearly with myocardial hypertrophy and inversely with ECV in the athletic group. These reassuring findings are in keeping with previous longitudinal studies of athletes and consistent with the hypothesis that the observed changes are because of normal physiological adaptation and not pathological hypertrophy with associated interstitial fibrosis. The authors acknowledge that the results have not been confirmed histologically, which will always be problematic in healthy volunteers and athletes, but the ECV technique has been validated in various cardiac pathologies.

Another interesting aspect is whether ECV is correspondingly reduced in veteran endurance athletes. The previous work by Mordi et al, although showing a significant reduction in ECV between older athletes and patients with dilated cardiomyopathy, did not demonstrate a decrease in ECV.
compared with sedentary controls. This discrepancy between the results of current study does raise the intriguing possibility that increasing ECV levels may be able to identify athletes who have been exercising at a high level for many years and who are starting to develop interstitial fibrosis; before overt replacement fibrosis occurs, as detectable by late-gadolinium enhancement (present in 13% of elite and ≤50% [6/12] of life-long veteran or endurance athletes). Long-term cohort studies will be required to confirm this hypothesis. If confirmed, ECV measurement may allow the identification of pathogenic factors that are associated with the development of fibrosis in athletes and allow the assessment of interventions.

The immediate clinical relevance of these results is clear. ECV is a potential early-stage imaging biomarker for the diagnosis of HCM, which is traditionally one of the differential diagnoses of athlete’s heart, especially in younger athletes. This study shows that ECV is relatively reduced in athletes compared with the expanded intracellular compartment, which is the opposite of what is seen in patients with HCM, where myocyte disarray and interstitial fibrosis lead to an increase in ECV, reflecting the expansion of the extracellular compartment. This may be particularly useful in differentiating the grey cases of early-stage HCM from athlete’s heart as it has already been demonstrated that ECV measurements are higher in genotype-positive HCM patients without overt LV hypertrophy than in genotype-negative control patients (0.33±0.01 versus 0.27±0.01 in genotype-positive/LV hypertrophy–negative versus genotype-negative/LV hypertrophy–negative controls; P<0.001). This offers a clinically attractive diagnostic tool. Currently, when CMR is unable to differentiate between athlete’s heart and HCM, some experts advocate repeating CMR scanning after a period (often 3 months) of deconditioning to assess for regression of LV mass and LV wall thickness, potentially delaying diagnosis and removing an athlete from training for a protracted period of time.

The limitations of ECV measurement should, however, be borne in mind. ECV is a surrogate for interstitial fibrosis, but measures the entire extracellular space. For this reason, the normal range is in the order of 20–30%, when most healthy people will have little or no interstitial fibrosis. This means that ECV markedly overestimates the degree of fibrosis early on, and differentiating patients from aged-matched controls may not be possible, as we have shown in asymptomatic patients with moderate and severe aortic stenosis. Further work is also required to confirm that these findings are consistent in women, older athletes, those from sports other than running/cycling/triathlon, and ethnic backgrounds who may be more prone to adverse LV remodeling. Given that preparticipation screening for sports is controversial and the high cost of CMR, widespread use in this situation is not indicated. However, the addition of T1 mapping to standard contrast-enhanced CMR could be used in selected athletes who are identified with an abnormal ECG, and the echocardiogram is inconclusive.

Native T1 mapping and ECV quantification in particular are exciting developments that will allow us to better understand the physiological adaptations that occur in response to exercise training. Although further studies are clearly warranted, T1 mapping is a promising tool to allow the clinical detection and differentiation of subtle cardiomyopathies from the athlete’s heart.

Disclosures

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