**Cardiac Magnetic Resonance Imaging in Athletes: Acquiring the Bigger Picture.**

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Participation in regular intensive physical exercise is associated with substantial increases in cardiac dimensions which overlap with cardiomyopathy in some white male endurance athletes and some black athletes who engage in explosive sprint sports such as soccer and basketball. While echocardiography has traditionally been the principal imaging modality to describe the normal structure of the athlete’s heart, the exciting potential of cardiovascular magnetic resonance (CMR) imaging can further our understanding of cardiac adaptation in athletes and facilitate the differentiation between physiological increases in cardiac dimensions and morphologically mild forms of cardiomyopathy. Cardiovascular magnetic resonance imaging allows more precise and detailed characterization of specific aspects of cardiac structure, particularly the right ventricular inflow and outflow tract, left ventricular apex and the left ventricular free wall. Furthermore, CMR provides more accurate assessment of ventricular volume and ejection fraction and can detect myocardial inflammation and fibrosis with late gadolinium enhancement. More sophisticated sequences include stress perfusion for assessment of ischaemia, T2 mapping for myocardial edema and T1 mapping for interstitial fibrosis. Such techniques may be harnessed to aid our understanding of cardiac adaptation to exercise on a cellular level.

Current studies involving CMR in athletes are limited by relatively small numbers in homogeneous sporting disciplines, most likely due to the costs and time associated with performing high volumes of studies in ostensibly normal healthy individuals. Other studies have focused on athletes with marked repolarization abnormalities such as deep T wave inversion, where abnormal dimensions may represent cardiomyopathy. Indeed, CMR imaging in athletes with such repolarisation anomalies who have an apparently normal echocardiogram have revealed a diagnosis of cardiomyopathy in approximately 20% on the basis of its superiority for detecting myocardial scar. Not all individuals with cardiomyopathy however, reveal scar, particularly during the early stages of the disease, therefore reliance on absolute dimensions and cardiac function are crucial for confirming or refuting cardiomyopathy in athletes. 1–3 In this regard, there is a pressing need to provide normal values for cardiac dimensions in athletes engaging in a variety of sports who are otherwise considered free of cardiovascular disease especially when consideration is given to the implications of an erroneous diagnosis in this scenario.

In this issue, D’Ascenzi and colleagues4 report a timely meta-analysis on studies involving CMR in apparently healthy competitive athletes aged between 18 and 55 years old (mean age 31±8 years) to propose normal values for ventricular size and function in athletes. Twenty-seven studies involving 983 male athletes were assessed including 250 endurance athletes, 263 combined trained (endurance and strength) and 472 mixed athletes (arbitrary definition). There were not enough subjects to include purely strength trained athletes. The authors provide the 5th to 95th percentiles for absolute and indexed ventricular volumes and mass, and ejection fraction. As expected from a plethora of echocardiographic studies, athletes showed larger mean ventricular volumes compared to those derived from the general population. Similarly, endurance athletes had the greatest left ventricular volumes and mass.

Findings from this study corroborate with a smaller, single-centre study of 222 athletes (79 elite-level and 143 recreational), that reported higher indexed left ventricular volumes and mass among male endurance athletes compared with nonathletes.1

Athletes with the largest training volumes, irrespective of sporting discipline also had the largest right ventricular volumes. Mean right ventricular ejection fraction was lower than in the general population but not as low as the Task Force criteria for arrhythmogenic right ventricular cardiomyopathy. Right ventricular ejection fraction was lower at rest (52%) than in the general population was higher than the cut-off for values for Task Force criteria for arrhythmogenic right ventricular cardiomyopathy (ARVC). We assume that morphological assessment for regional wall motion abnormalities was normal. Consistent with the hypothesis that the right ventricle is subjected to a greater afterload during strenuous exercise, this study highlights that increases in right ventricular volumes were related to amount of training rather than sporting discipline; surprisingly, there was no effect on left ventricular volumes.

Given that 27 articles included in this meta-analysis yielded only 983 athletes, reflects the paucity of data on CMR studies in healthy athletes, nevertheless data from almost 1000 athletes represents a respectable number to provide essential information in a specific cohort of athletes but also highlight the many deficiencies of such a meta-analysis in a field with limited data. Specifically, these results are applicable only to male athletes mainly in their third to fifth decade of life. These results cannot therefore be extrapolated to adolescent athletes, who undergo more preparticipation cardiac screening that any other age group and one that is arguably the most vulnerable to sudden cardiac death.7 This study was based solely on white subjects, therefore these normative values are not applicable to black athletes, who demonstrate a greater degree of left ventricular hypertrophy than white athletes and pose the greatest diagnostic challenge when differentiating physiological left ventricular hypertrophy from morphologically mild hypertrophic cardiomyopathy.8 Although the risk of sudden cardiac death from cardiomyopathy in female athletes is at least 10-fold lower than in males, the exclusion of females is a missed opportunity to appreciate sex differences in cardiac adaptation to exercise. While conventional demographic factors such as sex and ethnicity should always be accounted for, the training volume in this study is difficult to quantify objectively, particularly with subjective definitions of “amateur”, “professional” and “competitive”.

In the real world, there are inconsistencies between image acquisition and analysis technique. Some of the studies in this meta-analysis did not specify which technique was used while others differed in terms inclusion of papillary muscle and trabeculations. Such variation will lead to discrepancies in left ventricular mass and volume calculations. Tissue characterization by gadolinium enhancement was not possible, mitigating the unique ability of CMR to detect incidental myocardial fibrosis in athletes beyond 40 years of age. Small studies reveal that major focal fibrosis is noted in up 11-12% master athletes and we believe this requires further investigation until more data is available. Paradoxically, the prevalence and significance of minor focal fibrosis at the right ventricular insertion point is less clear. Considering the burgeoning increase in mass participation in endurance events and parallel reports of a J shaped relationship between intensity and volume of exercise and mortality, CMR will play a key role in ascertaining the prevalence, distribution and significance of myocardial fibrosis in life long athletes.

Despite the limitations of this study, D’Ascenzi and colleagues have helped fill a void in the literature by providing normative data for adult male athletes. As far as pre-participation screening with CMR is concerned, there are both logistical and financial barriers. A study of 5,255 children aged 13 years old who volunteered to undergo a CMR resulted in 19% meeting criteria for left ventricular non-compaction.11 The dichotomy between gaining novel data and having the resources to cope with abnormal findings may well limit the widespread applicability of such techniques.

Despite the advantages for CMR imaging, echocardiography will remain the leading imaging modality for cardiac pre-participation for the foreseeable future, given the ease of availability and lower costs. Structural findings need to be interpreted in the context of the clinical picture that includes patient symptoms and a family history and electrocardiogram. With particular reference ot family history and repolarization anomalies, CMR holds a core role to confirm or refute structural markers of arrhythmic risk. In this regard, reference values are paramount in establishing what is considered normal.

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