The Right Ventricle of the Endurance Athlete: The Relationship between Morphology and Deformation

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Background: The aims of this study were to establish absolute ranges for right ventricular (RV) structural and functional parameters for endurance athletes and to establish any impact of body size. These data may help differentiate physiologic conditioning from arrhythmogenic RV cardiomyopathy.

Methods: A prospective observational study design was used, and standard two-dimensional echocardiography was performed on 102 endurance athletes, providing RV structural indices. A two-dimensional strain (ε) technique was used to provide indices of RV ε and strain rate. The association of RV chamber size to body surface area (BSA) and functional indices was examined by simple ratio scaling as well as adoption of the general, nonlinear allometric model.

Results: The values for RV inflow, outflow, length, and diastolic area were greater than published ''normal ranges'' in 57%, 40%, 69%, and 59% of the population, respectively, while 28% of the population had RV outflow tract values greater than the proposed ''major criteria'' for arrhythmogenic RV cardiomyopathy. Simple ratio scaling for all RV dimensions to BSA did not produce size independence, whereas scaling for BSA allometrically did. Strain and strain rate values were consistent with published normal ranges, and there is no evidence to suggest that scaling is required.

Conclusions: RV chamber dimensions are larger in endurance athletes than those described by ''normal ranges'' and frequently meet the major criteria for the diagnosis of arrhythmogenic RV cardiomyopathy. Functional assessment of RV ε may aid in this differential diagnosis. RV size is allometrically related to BSA and therefore scaling for population-specific *b* exponents is encouraged. (J Am Soc Echocardiogr 2012;25:263-71.)

Keywords: Right ventricle, Strain imaging, Athlete, Cardiomyopathy

It is well established that participation in long-term endurance exercise is associated with cardiac adaptation, including hypertrophy and dilatation of the left ventricle,[1] which can complicate the differential diagnosis of athlete's heart from those of both hypertrophic and dilated cardiomyopathy.[2] The impact of physiologic conditioning on right ventricular (RV) structure and function, and a similar diagnostic challenge with arrhythmogenic RV cardiomyopathy (ARVC), has received considerably less attention. It is thought that ARVC is implicated in 4% to 21% of all cardiac sudden deaths in the young (aged $<$ 35 years) athletic population.[2] Furthermore, an ARVC phenotype without genetic cause has been observed in a number of endurance athletes,[3,4] raising the possibility that forms of ARVC may be acquired through long-term intensive endurance exercise.

Echocardiography plays a vital role in the cardiovascular evaluation of athletes, in particular in differentiating between ARVC and normal physiologic RV enlargement.[5] However, data for RV dimensions in athletes are scant,[6] with even less available information relating to RV function in this group. This is likely due to inherent difficulties in providing accurate structural and functional indices because of complex RV geometry and excess trabeculation.[7] Despite these obstacles, the American Society of Echocardiography (ASE) published normal ranges for RV dimensions in 2005,[8] which were adopted by professional bodies such as the British Society of Echocardiography. A subsequent ASE guideline report provided a more comprehensive and substantial range of RV dimension values including myocardial deformation parameters to facilitate the identification of RV pathology, but these data were obtained from the sedentary population.[9] More recently, studies in athletes, albeit in small cohorts, have demonstrated that some athletes exhibit large RV dimensions.[6,10-12] Myocardial deformation studies within these groups have attempted to establish parameters that may readily differentiate cardiac physiology from ARVC in athletes with enlarged RV dimensions.[6,13] Although the relationship of cardiac

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Abbreviations

ARVC = Arrhythmogenic right ventricular cardiomyopathy

ASE = American Society of Echocardiography

BSA = Body surface area

CI = Confidence interval

ε = Strain

LA = Left atrial

LAd = Left atrial dimension

LV = Left ventricular

LVd = Left ventricular end-diastolic diameter

LVEDV = Left ventricular end-diastolic volume

RV = Right ventricular

RVDarea = Right ventricular end-diastolic area

RVI = Right ventricular inflow

RVL = Right ventricular length

RVOT = Right ventricular outflow tract

SR = Strain rate

SRA′ = Strain rate during late ventricular diastole

SRE′ = Strain rate during early ventricular diastole

SRS′ = Strain rate during ventricular systole

morphology to body size is well established,[14] neither the ASE nor the British Society of Echocardiography guidelines provide ranges for RV data indexed to body surface area (BSA).[8,9] Therefore, the exact nature of how RV morphology data relate to body size has not been studied previously. Similarly the relationship between RV size and myocardial deformation remains to be elucidated. In view of the importance of RV size and function in the echocardiographic assessment of endurance athletes in providing clear normative data and thus differentiating from ARVC, the primary aims of this study were threefold: (1) to provide a range of absolute values for RV dimensions in 102 endurance athletes and to assess the nature of the relationships between these variables and measures of body size, (2) to provide a range of data for global RV strain (ε) and strain rate (SR) indices and to establish the nature of any relationships between indices of RV morphology and deformation, and (3) to establish if any increase in RV size is consistent with increases in left ventricular (LV) and left atrial (LA) size and, furthermore, whether the left-sided chambers require scaling by the same factors and methods that are applicable to RV assessment.

METHODS

One hundred two endurance athletes (86 men, 16 women) with a broad age range (mean, 36 ± 11 years; range, 21–71 years) volunteered to participate in the study. We recruited elite, internationally competitive athletes via direct contact with athletes, coaches, and race organizers. All athletes were competing at the highest level within their chosen sports, including internationally renowned competitions. Height and body mass were measured using standard equipment; the mean height was 178 ± 8 cm (range, 156–193 cm), and the mean body mass was 71 ± 10 kg (range, 44–100 kg). BSA was calculated using the equation of Dubois and Dubois[15]: {[(height)$^{0.425}$ × (body mass)$^{0.725}$ × 71.84]/1,000}. The mean BSA was 2.02 ± 0.24 m^2 (range, 1.36–2.68 m^2). The mean heart rate was 55 ± 10 beats/min (range, 34–89 beats/min). All subjects were healthy and free from known cardiovascular disease and any early family history of cardiovascular disease and were not currently taking any form of prescribed medication. All athletes had no subjective evidence of ARVC, such as saccular outpouching or prominent moderator bands. All subjects were either endurance runners ($n = 73$) or endurance cyclists ($n = 29$) and were scanned during a period of "peak" conditioning. We adopted a definition of "endurance athlete" on the basis of training and competition history that clearly reflected prolonged but intermittent periods of cardiovascular work. Moreover, training must have been continuous for ≥2 years before this assessment, and the mean number of training hours per week over this period ranged from 8 to 24. The population was purposely heterogenic, with a wide age and body size range. All subjects provided written informed consent to participate, and the studies were granted ethics approval by the Ethics Committee of Liverpool John Moores University.

Echocardiographic Assessments

After a full explanation of procedures, participants lay supine for 5 min before the echocardiographic examination. Heart rate was taken from the electrocardiograph inherent to the ultrasound system. All echocardiographic images were acquired using a commercially available ultrasound system (Vivid I or Vivid 7; GE Vingmed Ultrasound AS, Horton, Norway) with a 1.5-MHz to 4-MHz phased-array transducer. All images from all individual studies were acquired by the same single experienced sonographer using the same standard protocol, with the subject lying in the left lateral decubitus position. Images were recorded to DVD in a raw Digital Imaging and Communications in Medicine format. Offline analysis was performed by the same sonographer using commercially available software (EchoPAC version 7.0; GE Vingmed Ultrasound AS), and a minimum of three cardiac cycles were averaged for all indices.

Standard two-dimensional echocardiographic parameters were obtained from parasternal and modified apical acoustic windows. All settings were optimized to obtain maximum signal-to-noise ratio and two-dimensional images to provide optimal endocardial delineation. In accordance with ASE guidelines, RV size was measured at end-diastole from the proximal RV outflow tract (RVOT) using a parasternal short-axis orientation at the level of the aortic valve and at the basal RV inflow (RVI) from a modified apical four-chamber orientation[9] (see Figure 1). RV length (RVL) was also measured from the RV apex to the tricuspid annulus. RV end-diastolic area (RVDarea) and end-systolic area were calculated by tracing around the endocardium from a modified apical four-chamber orientation, and RV fractional area change was calculated.

Color-coded Doppler tissue imaging was used to assess RV myocardial velocities. Standard color Doppler tissue imaging acquisition was undertaken as previously described,[16] with a temporal resolution > 150 frames/sec. Offline analysis allowed a 6 × 6 mm sample volume to be placed in the tricuspid annulus of the RV lateral wall, and peak velocities in systole (S′) and early diastole (E′) were measured.

Conventional two-dimensional assessments of LV and LA structure were made in accordance with ASE guidelines.[8] LV mass, LV end-diastolic volume (LVEDV), and LV end-diastolic diameter (LVd) were calculated for scaling purposes, while LV end-systolic volume was also calculated using Simpson's biplane methodology to enable the calculation of LV ejection fraction. LA size was presented as the standard anterior-posterior dimension (LAd), measured using a standard parasternal long-axis orientation, while LA volume at end-systole was measured using apical four-chamber and two-chamber orientations and Simpson's biplane methodology. To establish the extent of physiologic adaptation of the right ventricle relative to LV remodeling, the ratio of RVI to LV dimension in the apical four-chamber orientation was calculated.[9]