

# The Right Ventricle of the Elite High End Endurance Athlete Cannot Be Underestimated

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Numerous studies have described features of adaptive cardiac remodeling in athletes. There remains overlap, however, between many of the structural and functional features of the athletic heart and potentially dangerous cardiac conditions including arrhythmogenic right ventricular (RV) cardiomyopathy (ARVC) and hypertrophic cardiomyopathy. The use of newer echocardiographic techniques including tissue Doppler, 3-dimensional imaging, and strain and strain rate imaging has improved our ability to identify these potentially life threatening conditions. Although much of the prior work has focused on the left heart, the right heart has recently received more attention. A number of recent studies have demonstrated evidence of RV myocardial dysfunction associated with both acute and chronic intense physical exercise.<sup>1-3</sup>

This distinction between the athletic heart and ARVC is particularly important in athletes who present with cardiac complaints including chest pain, syncope, and palpitations. Although magnetic resonance imaging remains the gold standard for diagnosing ARVC, specific echocardiographic criteria have been established to distinguish ARVC from nonpathologic RV remodeling.<sup>4</sup>

In an effort to distinguish athletic remodeling from ARVC, Baucé *et al.*<sup>5</sup> compared clinical, electrocardiographic, and echocardiographic features of 40 ARVC patients, 40 athletes, and 40 control subjects. They found that RV enlargement was present in both athletes and patients with ARVC compared with controls. The patients with ARVC, however, exhibited RV outflow tract enlargement and reduced RV fractional shortening and ejection fractions compared with athletes. One feature commonly described in patients with ARVC, a thickened moderator band, was found with similar frequency in both patients with ARVC and athletes.

There has been recent concern regarding whether RV changes associated with prolonged, intense physical exertion lead to pathologic changes in RV structure and function. Although there is compelling evidence for the cardiovascular benefits of regular physical exercise, recent studies have shown that transient RV myocardial damage may occur during intense training regimes and prolonged exercise, especially in amateur participants.<sup>2,3,6</sup> An association between pronounced RV structural changes and cardiac events was demonstrated by Heidbüchel *et al.*<sup>7</sup> at the University of Leuven in Belgium. They demonstrated that mild RV dysfunction was common in athletes with complex ventricular arrhythmias, and a significant number resulted in sudden death. Heidbüchel *et al.* coined the term "exercised-induced RV cardiomyopathy" and subsequently demonstrated that endurance athletes with ventricular arrhythmias had lower RV ejection fractions as determined by the gold standard of ventriculography than athletes without ventricular arrhythmias.

We have known for years that habitual endurance exercise results in structural and functional cardiac changes, termed "athlete's heart syndrome." We know that all chambers of the heart undergo remodeling, which is believed to represent an adaptive response providing the means for enhanced cardiovascular performance during exercise. However the differentiation between long-term loading-induced RV remodeling and reduced RV contractility may have important therapeutic and prognostic implications. It has been suggested that some high-end endurance elite athletes may undergo structural and electrical remodeling that may create substrate arrhythmias, including life-threatening arrhythmias.<sup>7</sup>

The possibility of cardiac remodeling as a substrate for atrial fibrillation in athletes has been long questioned. Aziz *et al.*,<sup>8</sup> from Columbia University College of Physicians and Surgeons in New York, showed that RV dysfunction is a strong predictor for developing atrial fibrillation. This finding might be linked with studies by La Gerche *et al.*,<sup>9</sup> who showed that intense exercise induces an increase in RV end-systolic wall stress that exceeds left ventricular end-systolic wall stress in elite athletes, and this leads to greater RV enlargement and greater wall thickening, which may be a product of this disproportionate load excess.

Further studies by La Gerche *et al.*<sup>10</sup> showed a certain phenotype of ARVC, characterized by a high volume of intense, endurance sport practice and little or no evidence of a familial predisposition. This cohort may have a mild genetic risk or unrecognized gene, which could evoke an ARVC phenotype if combined with intense endurance exercise. This even further emphasizes the absolute necessity of a full and complete echocardiographic study to include each and every modality in these high-end elite athletes.

If one can assume that RV myocardial function must be preserved, if not enhanced, in well-trained athletes to enable greater augmentation of cardiac output during exercise, this would suggest that resting strain and strain rate are unreliable measures of RV function.

The current issue of *JASE* features two reports from studies that examined RV structural and functional parameters in athletes. La Gerche *et al.*<sup>11</sup> show that resting measures of RV function are a poor surrogate of RV functional reserve and that low strain and systolic strain rate measures obtained at rest should not be interpreted as a sign of subclinical myocardial damage. The fundamental message from La Gerche *et al.*'s report is that although RV function may be low normal at rest in athletes, there is abundant RV functional reserve demonstrated in both athletes and nonathletic individuals after provocation with exercise and that the increases in ventricular and atrial size reflect physiologic changes rather than subclinical myocardial damage.

Prior work by Dr. King and his colleagues demonstrated that the acceleration of the RV wall during the isovolumic period of contraction is maintained or enhanced in athletes, thus providing a more physiologically plausible summary of function.<sup>12</sup>

In a study examining left ventricular and RV structure and function in Olympic speed skaters, Wood and colleagues examined the RV structure and function of a group of Olympic speed skaters and found evidence of RV remodeling, including larger basal RV dimensions ( $38 \pm 5$  vs  $32 \pm 4$  mm,  $P = .001$ ) and attenuated resting RV systolic

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0894-7317/\$36.00

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doi:10.1016/j.echo.2012.01.018

function (RV area change  $35 \pm 13\%$  in athletes vs  $47 \pm 11\%$  in controls,  $P = .006$ ), as well as reduced RV systolic strain rate ( $1.9 \pm 0.5$  vs  $2.9 \pm 1.1 \text{ sec}^{-1}$ ,  $P < .001$ ).<sup>13</sup> Interestingly, however, the athletes demonstrated enhanced RV diastolic function at rest (E' velocity,  $13.5 \pm 3.6$  vs  $11.1 \pm 1.5 \text{ cm/sec}$ ,  $P = .016$ ). After an episode of intense skating, the athletes exhibited augmentation of RV systolic function with increased RV fractional area change (increasing to  $43 \pm 10\%$ ,  $P = .007$ ) and systolic strain rate ( $2.5 \pm 1.2 \text{ sec}^{-1}$  after exercise,  $P = .038$ ). These findings are similar to the enhanced RV systolic function after exercise described by La Gerche *et al.*<sup>11</sup> in this issue of *JASE*. This work suggests that RV diastolic parameters deserve further study in larger groups of endurance athletes both at rest and after exertion. It is likely that similar to the left ventricle, there is a close interaction between systolic and diastolic coupling in the right ventricle. Diastolic indices (tissue velocities, strain, and strain rate) may distinguish adaptive athletic remodeling from potentially pathologic RV conditions.

Another article in this issue of *JASE* also provides distinct but highly compelling insight into RV behavior in elite athletes. Oxborough *et al.*<sup>14</sup> establish absolute ranges for RV structural and functional parameters for elite athletes by allometrically scaling for body surface area (BSA). Isometric scaling is often used as a null hypothesis in scaling studies, with "deviations from isometry" considered evidence of physiologic factors forcing allometric growth. This study also showed that simple scaling for RV dimensions to BSA did not produce size independence, whereas scaling for BSA allometrically did. This report supports the development of algorithms for use in physicians' offices to allow for careful evaluation of RV parameters. The clear message provided by Oxborough *et al.* is that allometric scaling of RV chamber size to BSA ensures size independence and accuracy between subject comparisons. Allied to this was a lack of allometric association with peak strain and strain rates, and therefore there was no reason to scale these parameters.<sup>14</sup> All this evidence supports the need for an in-depth study of RV function when assessing high-end endurance elite athletes during echocardiographic screening. Further investigations into the RV diastolic parameters in this population should also be considered. This work also supports the use of newer echocardiographic modalities (strain, strain rate, tissue Doppler) when performing cardiovascular evaluations of athletes. Given the exercise-induced enhancement of RV function described by La Gerche *et al.*<sup>11</sup> in this issue of *JASE*, it seems prudent to incorporate exercise (routine stress testing or cardiopulmonary exercise testing) with echocardiographic imaging when abnormalities are noted on the baseline echocardiogram of an endurance athlete.

## CONCLUSION

The health benefits of moderate exercise are well established, but the health benefits of intense prolonged exercise are less certain. Echocardiographic parameters of RV systolic performance are well documented. Offline analysis of strain and strain rate parameters, myocardial velocities, and displacement measures using color-coded Doppler tissue imaging should be assessed on all examinations of elite athlete. Work described in this issue of *JASE* supports consideration of echocardiographic imaging both at rest and after exertion. These should be performed by accredited clinical physiologists in conjunction with cardiologists. We owe this type of care to every high-

endurance elite athlete who presents for cardiac assessment. Whether their presentation is related to new symptoms, abnormal electrocardiographic findings, or changes in their performance and perceived endurance, athletes deserve thorough evaluations. We must look to the right as well as the left before crossing the Rubicon of a diagnosis in these elite athletes. The studies by La Gerche *et al.*<sup>11</sup> and Oxborough *et al.*<sup>14</sup> have made this crossing a little less daunting.

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