Commentary

Exercise and the Female Heart

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ABSTRACT

Female participation in sport has increased sharply during the last few decades, and for the third straight Olympic Games, there were more women than men on the US roster for the 2020 Tokyo Games. Given this, an understanding of the differences between men and women with respect to exercise-induced cardiac remodeling is critical for those caring for female athletes. Recent studies have provided insight into female-specific cardiac remodeling and have enhanced our understanding on the upper limits of cardiac remodeling in female athletes and how these adaptations compare with sedentary females, male athletes, and cardiomyopathies. Female athletes display fewer signs of adaptive remodeling on ECG compared with male athletes. Structurally, male athletes have larger absolute cardiac dimensions, but female athletes have similar or larger chamber size when adjusted for body size. Female athletes have a lower incidence of sudden cardiac arrest or death compared with male athletes in the early competitive years (high school, college, and professional) and in the masters athlete years. In addition, female athletes are less likely to have coronary disease and atrial fibrillation compared with male athletes. Data on longevity indicate that female athletes live longer than their sedentary counterparts. Unlike men, there has been no convincing association of extreme exercise and cardiovascular disease in longer-term endurance female athletes. The underlying mechanisms of these sex-based differences are not very well understood, and future studies are warranted to better understand the mechanisms of cardiac adaptation in female athletes. (*Clin Ther.* 2022;44:41–49.) © 2021 Elsevier Inc.

Key words: athlete's heart, cardiovascular disease, exercise-induced cardiac remodeling, exercise physiology, sports cardiology, female athletes.

INTRODUCTION

Augmentation in cardiac pressure and volume during routine vigorous exercise can lead to the development of structural, functional, and electrical adaptive changes that collectively are known as exercise-induced cardiac remodeling (EICR) or the athlete's heart. These changes include, but are not limited to, increases in chamber cavity size and wall thickness, enhanced diastolic filling, bradycardia, and augmentation of stroke volume. The influence of the type of sport, classified by the peak static and dynamic components,[1] on cardiac remodeling has revealed that cardiac adaptations are sport specific.[2,3] Sports with a predominantly high static component, such as weightlifting and rock climbing, result in high cardiac pressure loads that typically result in concentric left ventricular hypertrophy (LVH) characterized by increased LV wall thickness (LVWT) without changes to the chamber size. Meanwhile, endurance running and other sports with a high dynamic component require sustained increased in cardiac output, and the EICR is characterized by 4 chamber dilation without significant changes to the LVWT consistent with eccentric LV remodeling.[4] Sports that have mixed physiologic attributes with both high-static and dynamic components, such as rowing and triathlon, result in 4-chamber dilation with balanced increased in LV size and LVWT or eccentric LVH. However, the degree of EICR is influenced by many factors, including sex, age, ethnicity, exercise exposure (which can be quantified as a dose using frequency, intensity, and duration), body size, and genetics, that also need to be considered when evaluating an athlete.

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Table I. Sex and ethnicity-based differences in ECG findings in athletes.

ECG Finding	All Female Athletes	White Female Athletes	Black Female Athletes	Male Athletes
Normal Training-Related Findings				
QRS voltage criteria for LVH*	8%–14%[14–16,58,59]	NA	NA	31%–64%[14–16,58,59]
Incomplete RBBB	3%–6%[15,58,59]	NA	NA	13%–24%[15,16,58,59]
Normal early repolarization	23%–29%[58,59]	NA	NA	36%–84%[15,16,58,59]
First degree AV block	3%–7%[15,58,59]	NA	NA	4%–10%[15,16,58,59]
Sinus bradycardia	50%–70%[58,59]	NA	NA	51%–64%[16,58,59]
Abnormal, Potentially Pathologic Findings				
Anterior TWI	4%–9%[14,15]	0%–2.1%[11,60,61]	14%[11]	0%–4%[14–16]
Inferior or lateral TWI	0%–2%[14,15]	2%[11]	NA	0%–5%[14,15]

AV = atrioventricular; LVH = left ventricular hypertrophy; NA = not applicable; RBBB = right bundle branch block; TWI = T-wave inversion.
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At the time of the ratification of Title IX in 1972, approximately 31,000 women were competing in college sports. For the 2017 to 2018 academic year, this number increased to 216,378 and accounted for 44% of the National Collegiate Athletic Association student athlete population.[5] For the third straight Olympic Games, there were more women than men on the US roster, with women accounting for 54% of athletes for the 2020 Tokyo Games.[6] Although much of the early data on EICR came from studies performed predominantly in men, the sharp increase in female sport participation during the last few decades has been followed by data on sex as an important biologic variable in EICR. Differences in anthropometric, biochemical, and physiologic profiles affect the female cardiovascular response to athletic training.[7] We highlight the sex-specific differences in cardiac remodeling, development of cardiovascular risk factors, and disease and mortality in response to exercise training.

ELECTRICAL ADAPTATIONS

Specific recommendations for the interpretation of the electrocardiogram (ECG) in athletes have been published and refined in attempts to maintain sensitivity but minimize false-positive results, with the most recent recommendations published in 2017.[8–10] Important differences exist between female and male athletes with respect to the prevalence of normal and abnormal ECG findings as defined by the recommendations (Table I). Male athletes have higher prevalence of normal training–related variants, which include elevated QRS voltage, longer QRS duration, longer PR intervals, and early repolarization, compared with female athletes. The higher prevalence of normal training–related ECG variations in males is not unexpected given that the ECG findings reflect structural cardiac remodeling and increased vagal tone and men have more pronounced structural remodeling than women. Abnormal ECG findings of inferior and lateral T-wave inversions (TWIs) are more common in male athletes, whereas anterior TWIs are more commonly seen in female athletes. Among female athletes, anterior TWIs and ST-segment elevation (11% vs 1%) seem to be more common in black compared to white female athletes.[11] A firm understanding of the expected ECG differences based on sex and ethnicity is important when caring for competitive athletes. It is our practice to repeat an ECG confirming lead placement in female athletes with TWIs in V1-V3 before pursuing further workup because breast tissue can lead to variability in chest lead placement.

STRUCTURAL ADAPTATIONS

LV Findings

Normal values for LV chamber size, thickness, and mass are smaller for women compared with men in the general population.[12] This pattern holds true for athletes; when compared with male athletes, female athletes have an approximately 10% smaller absolute

Table II. Relative magnitudes of cardiac remodeling in female compared with male athletes.

Measure	Total Female Athletes	White Female Athletes	Black Female Athletes
Left Ventricle			
LVEDD	⇓⇓	⇓	⇓⇓
LVEDD/BSA	⇑/⇔		
LVWT	⇓⇓	⇓⇓	⇓
LVM	⇓⇓	⇓⇓	⇓
LVM/BSA	⇓		
EF	⇔	⇔	⇔
Right Ventricle			
RVBD	⇓⇓	⇓	⇓⇓
RVBD/BSA	⇔/⇑	⇔/⇑	⇔/⇑
TAPSE	⇔	⇔	⇔

BSA = body surface area; EF = ejection fraction; LVEDD = left ventricular end-diastolic diameter; LVM = left ventricular mass; LVWT = left ventricular wall thickness; RVBD = right ventricular basal diameter; TAPSE = tricuspid annular plane systolic excursion; ⇔ = similar; ⇓ = decreased; ⇓⇓ = relatively larger decrease than ⇓.

LV chamber size, 10% to 20% smaller LVWT, and 20% to 55% smaller absolute LV mass, indicating the importance of understanding distinct sex-based differences in EICR (Table II).[13–16] These differences in LV remodeling between the sexes have been found as early as adolescence and extend through adulthood.[17] When LV cavity size is indexed for body surface area (BSA), the indexed LV dimensions are actually greater in female athletes when compared with male athletes.[14]

When compared with their sedentary counterparts, female athletes display remodeling of the LV with a 10% to 20% increase in LVWT and a 10% to 15% increase in ventricular size.[7,18] Significant LV remodeling can be seen in female athletes, with an upper LV end diastolic dimension in female athletes reported to be 61 to 66 mm.[6,14,18] EICR is also a progressive process that continues throughout an athletic career as noted by incremental, age-dependent increases in LV mass, LVWT, and LV chamber volumes in a study that included elite female soccer players.[16] Thus, absolute LV dimensions alone are not sensitive in distinguishing physiologic from pathologic findings.

Findings from a recent study on the cardiac structure and function in a cohort of 140 elite female basketball players (75% black) found increased cardiac dimensions (LV cavity size, LVWT, and LV mass) (Table III).[19] Approximately one fourth of athletes had LV dilation as defined by LV end diastolic dimension >5.2 cm by guidelines,[12] but most normalized when indexed to BSA. When comparing ethnic differences, white female athletes have a larger LV end diastolic dimension compared with black female athletes.[19] This study also provided important insight into LVH in female athletes. Although the range of LVWT was 0.7 to 1.4 cm, the mean (SD) was 9 (1) mm, suggesting that even some of the largest (mean BSA, 2.02 m^2) female professional athletes do not routinely exhibit significant LVH in response to training. Of the athletes with LVH, 69.6% had eccentric LVH. Data from the aforementioned study on professional soccers players indicated that elite female players (n = 122, 71% white) had a similar mean (SD) LVWT of 8.9 (1.2) mm with a maximum LVWT of 12 mm. Although significant LVH was not seen, a lesser degree of

Table III. Classification of specifically referenced sports.

Sport	Static Component	Dynamic Component
Basketball	Moderate	High
Soccer	Low	High
Triathlon	High	High
Long distance running	Low	High

remodeling was common, with 30% of females athletes having a LVWT above the guideline-defined normal range of 9 mm.[12,16]

Despite these changes in LV size, mass, and thickness, most female athletes have normal LV geometry.[14,15,19] The effect of sex and sport discpline on LV geometry was evaluated in a cohort of 1083 elite, white athletes (41% female).[14] Notable findings include that most athletes had normal LV geometry, all female athletes had a LVWT ≤12 mm, and for those engaged in dynamic sport, female athletes more frequently had eccentric hypertrophy compared with male athletes (22% vs 14%). Female athletes only rarely had concentric hypertrophy or remodeling when engaged in dynamic sport. Assessment of LV geometry using LV mass index and relative wall thickness can be helpful in differentiating EICR from pathologic LVH because use of absolute measurements of LV size, thickness, and mass is challenging because these measurements are influenced by sex, body size, and type of sport.

Ethnic differences in LV remodeling among female athletes have also been reported. A study on 240 elite black and 200 white female athletes found that black female athletes had greater LV mass and LVWT, contributing to greater LV hypertrophy.[18] Although 3% of black athletes had LVWT >11 mm, with a maximum of 13 mm, no white female athletes had LVWT >11 mm. No difference was found in LV ejection fraction, LV cavity size, or diastolic function. Although black female athletes have increased LVWT compared with white female athletes, LVWT >12 mm is still rare and should warrant further investigation for any female athlete.[11,12,19]

These studies enhance our understanding of the expected ranges of LV remodeling in female athletes, but they also highlight the importance of interpretating LV structure and function in female athletes in the context of all parameters that influence EICR. It has been suggested that males display more pronounced LVH than females given a prohypertrophic effect of testosterone and dihydrotestosterone on cardiac myocytes.[20] In addition, estrogens inhibit myocardial hypertrophy and promote peripheral vasodilation and afterload reduction.[21,22] Smaller LV dimensions in female athletes may be related to women having lower lean body mass, lower testosterone levels, a lower sympathetic adrenergic response to exercise with lower peak systolic blood pressure, stroke volume, and peak $\dot{V}o_2$.[7,23]

RV Findings

Right ventricular (RV) remodeling is most commonly seen in endurance athletes as a direct consequence of volume loading of the heart with dynamic exercise. RV enlargement is also part of the criteria for the diagnosis of arrhythmogenic RV cardiomyopathy.[24] Thus, physiologic RV enlargement in athletes must be differentiated from pathologic enlargement because vigorous exercise with underlying arrhythmogenic RV cardiomyopathy can accelerate disease progression, provoke ventricular arrhythmias, and increase mortality.[25]

Differences in RV remodeling between sexes are similar to those noted in the LV; namely, female athletes have larger RV size when compared with their sedentary counterparts, and a smaller absolute but larger indexed (BSA) RV size when compared with male athletes.[15,26–30] In addition, similar to the LV, RV functional parameters do not have any differences in relation to sex.[27,30] A longitudinal study following up 29 elite athletes (55% female) during 1 training season found that RV size increased during the season, which was seen with a concomitant increase in the LV size and stable RV function.[28] These findings support the theory that RV enlargement is a physiologic response to dynamic training. A study on ethnic differences in RV dilation revealed that black female athletes had significantly smaller absolute RV end diastolic dimension and area when compared with white female athletes.[26]

In a cross-sectional study of 1009 Olympic athletes (36% women), athletes who participated in endurance sport had the largest RV dimensions and the highest global RV function.[27] The mean (SD) and upper range of RV basal diameter for female athletes (all sports) was significantly lower when compared with male athletes (35.2 [4.8] mm [upper range, 44 mm] vs 40.6 [5.1] mm [upper range, 49 mm]) but unchanged when indexed to height. When compared with guideline-based normal values,[12] approximately one third of the entire cohort met the criteria for a dilated RV, and 16% and 41% fulfilled the major and minor criteria for the diagnosis of arrhythmogenic RV cardiomyopathy, respectively.[24] Importantly, RV enlargement was not an isolated finding in these athletes because there was a positive linear correlation between RV and LV size,

suggesting a balanced, symmetric ventricular cavity enlargement in athletes.[27] In this regard, a mean (SD) basal end-diastolic RV/LV diameter size ratio of 0.8 (0.1) in the long axis has been suggested as 1 tool to help differentiate physiologic from pathologic RV dilation.[27,31]

Atria

As previously noted, endurance sport requires sustained high levels of cardiac output. This volume load is felt by all 4 cardiac chambers such that left and right atrial remodeling commonly parallel changes seen in the ventricles. Although endurance athletes have the largest left atria (LA), LA enlargement is seen in all athletes regardless of sport type but to a lesser degree as the dynamic component decreases and the static component increases.[32] In a large metanalysis, LA diameter and LA volume indexed for BSA were 13% and 30%, respectively, larger in athletes (all sport types) compared with controls.[32] LA diameter was 2.3 mm greater in male compared with female athletes, but when indexed LA volumes were compared there was no difference between the sexes.[32] LA and RA enlargement were noted in 40.7% and 18.2%, respectively, of elite female basketball players.[19] Similar to the LA, the degree of RA remodeling is most prominent in endurance athletes.[27]

D'Ascenzi et al[33] longitudinally investigated the change in atrial size and function in 26 female athletes after a 16-week period of intensive training in mixed static and dynamic sports. The study revealed significantly increased absolute and indexed biatrial volumes in response to exercise training, highlighting a strong causal relationship between exercise and atrial remodeling. Before the training period, 95% of female athletes had a normal LA volume index and 5% had mildly dilated LA; however, after just 16 weeks of training, 30% had mild LA enlargement and 15% had moderate enlargement. Importantly, normal diastolic function and filling pressures were maintained. Remodeling of the RA was also noted, with no female athletes having dilated RA before training but 10% having RA enlargement after the training period.[33] Cardiac magnetic resonance imaging used to quantify cardiac volumes found larger atrial volumes in male and female athletes compared with controls, but when the atrial volume was normalized for total heart volume, no difference was found between athletes and controls, indicating a balanced atrial enlargement.[34] Atrial remodeling was found to be less pronounced in female endurance athletes when compared with males athletes. [34]

SUDDEN CARDIAC DEATH

On the basis of data collected during a period of 3 decades, the incidence of sudden cardiac death (SCD) in male competitive athletes was 6.5-fold that of SCD in their female counterparts.[35] Hypertrophic cardiomyopathy accounted for 11% of the female deaths compared with 39% of the male sudden deaths. Females died of coronary artery anomalies, arrhythmogenic RV cardiomyopathy, and long QT syndrome more commonly than males. Ethnic differences also exist, with the cardiovascular death rate in black/other minority athletes exceeding that of white athletes by almost 5-fold.[35] From a study of collegiate athletes, male athletes were >3 times more likely to die than female athletes, Division I athletes were more likely to die than Division III athletes and black athletes 3 times more than likely than any other race.[36] The most common cause of death in this series was in the setting of a structurally normal heart and likely attributable to primary arrhythmia syndromes, a finding supported by other recent studies.[36,37]

In a review of sudden deaths during US triathlons from 2006 to 2016, of the 135 deaths (mean age, 47 years), 85% were male, and the incidence of death or cardiac arrest was 0.74 per 100,000 in women, which was significantly lower compared with 2.40 per 100,000 in men.[38] The risk of cardiac arrest in marathon and half-marathons in the United States is 0.16 per 100,000 for women compared with 0.9 per 100,000 for men.[39]

Previously, the high incidence of SCD in males was attributed to a higher proportion of male participation in competitive sports. However, despite the increase in female participation during the past few decades, there has not been a parallel increase in the incidence of SCD for female athletes.[22] In addition, the cause of sudden cardiac arrest or death is typically influenced by age, with events in those >35 years of age typically caused by atherosclerotic disease and events in those <35 years of age usually caused by inherited cardiac diseases, such as cardiomyopathies and channelopathies. These studies imply that the reduced SCD risk in women is seen in both age groups. Findings of increased sympathetic tone and increased release of catecholamines in male athletes

may lead to deleterious effects in susceptible (those with underlying inherited syndromes or atherosclerotic disease) athletes.[40,41] The biochemical modification of arrhythmic risk, whether it be a harmful effect of increased levels of androgens in males or a protective effect of estrogen in females, need further evaluation.[22,42]

IMPACT OF EXERCISE ON CARDIOVASCULAR RISK FACTORS AND DISEASE

Routine moderate-intensity exercise has several health benefits, ultimately decreasing morbidity and improving survival. The effect of high-intensity interval training (HIIT), which involves short bouts of intense exercise interspersed with periods of rest or lower-intensity exercise, versus moderate-intensity continuous training on cardiovascular risk factors has also been studied. Although most studies have involved male participants, some studies have included women in small numbers. Both HIIT and moderate-intensity continuous training provide comparable reduction in the resting blood pressure in adults with established hypertension, but HIIT improves cardiorespiratory fitness to a greater magnitude and has been associated with improved endothelial function and decreased insulin resistance.[43] In addition, an important advantage HIIT is that the same exercise dose can be achieved compared with moderate-intensity continuous training in a shorter duration of time, making HIIT a good option for some with limited leisure time.

Some studies have suggested an association between long-term endurance exercise and development of coronary artery disease. One study of 152 masters athletes (46 female) found that most female athletes (78%) had a calcium score of zero, but there was no difference in the prevalence of coronary artery disease, coronary artery calcium scores, number of plaques, or plaque morphologic findings between female athletes and sedentary female controls.[44] A study of female marathon runners specifically found that they had minimal coronary artery calcium, lower coronary plaque prevalence (19 vs 50%), and a lower volume of calcified plaque compared with sedentary controls.[45] These findings differ from studies in male athletes that suggest that male athletes consistently have higher burden of coronary atherosclerosis compared with sedentary controls.[44,46] This finding suggests that the long-term effects of intense athletic training on the development of coronary atherosclerosis may be different in men and women.

Emerging data suggest that a small portion of masters endurance athletes have myocardial fibrosis when evaluated by cardiac magnetic resonance imaging. When located at the RV insertion point, the cause of fibrosis may reflect ventricular overload from long-term high-volume exercise training; however, in other locations, fibrosis attributable to occult coronary disease or prior myocarditis should be considered before suggesting findings are a result of exercise.[42] A recent study of male and female triathletes found evidence of late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging, suggestive of focal nonischemic myocardial fibrosis in 17% of male participants but none of the 29 female participants.[47] In a follow-up letter to the editor, it was suggested that more than 55% of the LGE noted in male participants could be explained by prior myocarditis and thus should not be considered a result of high-volume exercise.[48] A more recent study evaluated 93 endurance athletes (44 female) and found that 41% of the female and 35% of the male athletes had LGE, all located at the RV insertion point.[49] There was no LGE noted in the female controls. The peak exercise blood pressures was similar when comparing athletes with LGE with those without LGE, a findings that goes against a prior suggestion that LGE in athletes may be a result of a more robust blood pressure response to exercise.[47] Data on LGE in long-term endurance female athletes remain limited and require further investigation on the true prevalence, mechanism, and outcomes in this cohort.

The frequency of atrial fibrillation in endurance athletes is 2 to 10 times greater in high-intensity endurance athletes compared with their sedentary counterparts, and in men, vigorous physical activity has been consistently associated with an increased risk of atrial fibrillation.[50–52] Findings in female study participants have not been as consistent. A large prospective study suggested that moderate physical activity reduces the risk of atrial fibrillation compared with controls, whereas the risk of atrial fibrillation in vigorous physical activity is similar to controls in both male and female athletes.[53] Meanwhile, other studies have found continued reductions in atrial fibrillation risk in women exercising at the highest doses.[51,54] More study is required to better understand whether there is a true difference between the sexes

in the incidence of atrial fibrillation in high-volume endurance athletes.

LONG-TERM MORTALITY

A recent meta-analysis of >165,000 former athletes (15.6% female) investigated the nature of the exercise-longevity relationship. Female athletes had a 49% lower risk of all-cause mortality compared with the general population, and the female survival advantage (female life expectancy is 6–8 years longer than males at birth) persisted and may even be extended after a career in elite sport.[55] A retrospective cohort study of 8124 US athletes (2301 women) who competed in an Olympic Games from 1912 to 2012 and were followed up through 2015 found that former male and female Olympians live 5.1 years longer than their peers in the general population and female Olympian live longer than male Olympians.[56] This advantage is thought to be primarily conferred by lower risks of cardiovascular disease and cancer. Similar findings of improved longevity have also been reported in former French Olympians.[57] Future work should focus on long-term follow-up to establish benefits and/or consequences of long-term intense physical activity and assessment of sport-specific mortality in larger cohorts of female athletes.

CONCLUSIONS

Routine vigorous exercise training results in electrical, structural, and functional cardiac changes that collectively are known as EICR or the athlete's heart. Although sport-specific changes in EICR have been well recognized, more recent literature shows clear differences in the type and extent of EICR between men and women. Female athletes more commonly have anterior TWIs, whereas in male athletes, elevated QRS voltage and early repolarization are more common. Female athletes have significant cardiac remodeling compared with sedentary controls but rarely reach absolute LV dimensions or LVWT that overlap with dilated cardiomyopathy or hypertrophic cardiomyopathy. In addition, female athletes commonly have normal LV geometry. In this context, female athletes consistently have a reduced risk of sudden cardiac arrest or death, a decreased prevalence of cardiovascular disease, and improved longevity compared with male athletes and sedentary females. The underlying mechanisms of many of these differences are not very well understood. Given increasing participation of women in sports, future studies are warranted to better understand the mechanisms of cardiac adaptation in female athletes.

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DISCLOSURE

The authors have indicated that they have no conflicts of interest regarding the content of this article.

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