**The Complex Phenotype of the Athletes Heart: Implications for Pre-Participation Screening**

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**ABSTRACT**

Pre-participation screening is vital to exclude inherited cardiac conditions which have the potential to cause sudden cardiac death in seemingly healthy athletes. Recent research has questioned traditional theories of load-induced, dichotomous cardiac adaptation. We therefore considered whether a “one-size-fits-all” approach to screening can account for inter-individual differences brought about by sporting discipline, training volume, ethnicity, body size, sex and age.

**SUMMARY**

Considering inter-individual differences in key factors affecting adaptation of the athlete’s heart, facilitates better discernment of physiological and pathological adaptation.

**KEY POINTS**

* Sudden cardiac death caused by inherited conditions is most prevalent in young athletes, but can be reduced through effective screening programmes.
* Application of refined criteria may be appropriate for screening of African/Afro-Caribbean athletes, as (in contrast to Caucasian athletes) T-wave inversion, trabeculation and LV hypertrophy may represent physiological adaptation within this population.
* Sporting discipline and training volume form important factors in dictating morphology of the athlete’s heart. However, more well-controlled longitudinal examinations are required to develop accurate understanding of what constitutes normal/abnormal adaptation for a given sport and training volume.
* The “athlete’s heart” phenotype refers to a whole heart adaptation, rather than being restricted to the left ventricle. Future research should therefore seek to define athletic norms (using appropriate scaling methodologies) for the structure and function of the atria and right ventricle.

**KEY WORDS**

Athlete's Heart; Cardiac Screening; ECG; Echocardiography; Sudden Cardiac Death; Sports Cardiology

**INTRODUCTION**

It is well established that the athlete’s heart (AH) undergoes physiological adaptation in response to chronic exercise training, however much of the early literature focused on the left ventricle (LV) with little acknowledgement of right ventricular (RV), atrial, or functional adaptation. Interest in “whole” cardiac adaptation has significantly increased in the last 10 years, and original theories of load induced, dichotomous adaptations have been questioned [44]. The impact of other factors on cardiac phenotype have also been raised, including sporting discipline, training volume, ethnicity, body size, sex and age, on the magnitude and nature of adaptation (Figure 1) [6, 26, 38, 45, 48].

*[INSERT FIGURE 1 HERE]*

The reasons behind this growth of interest appear to have been driven by developments in imaging technology and high profile cardiac events which continue to occur in the seemingly healthiest of the population. Although contradictory schools of thought exist regarding the efficacy of pre-participation screening [24], there is a growing demand for sports physicians to undertake screening in order to exclude inherited cardiac diseases, which account for the majority of sudden cardiac death (SCD) cases in individuals under 35 years of age [11]. A comprehensive understanding of cardiac structure and function in a heterogeneous athletic population is therefore fundamental to facilitate identification of normal and abnormal features on the athlete’s electrocardiogram (ECG) and echocardiogram. This review aims to provide a balanced perspective on our current understanding of the athlete’s heart, with a focus on the impact of sporting discipline, training volume, ethnicity, body size, sex and age. We hypothesised that an individualised approach to diagnostic testing is required to differentiate between physiological and pathological cardiac adaptation in athletic populations.

**THE IMPACT OF SPORTING DISCIPLINE AND TRAINING VOLUME**

Although it is known that exercise training elicits physiological adaptation of the heart, our understanding of how specific training stimuli and volume are related to this adaptation is less clear. The seminal work of Morganroth *et al.* suggested endurance and strength based training elicit eccentric hypertrophy and concentric remodelling of the LV, respectively. More recent work has challenged this [43, 45], highlighting the need for a more comprehensive study of the AH phenotype, and greater clarity in the classification of sporting disciplines. Data are also now emerging regarding the effects of changes in training volume (duration x intensity) on adaptation of the AH.

*The 12-Lead Electrocardiogram*

Our knowledge of training-induced ECG changes is predominantly based upon large cohort studies, where little consideration has been given to sport specific cardiovascular demands. In view of this, Brosnan *et al*. [9] analysed the resting ECG of endurance athletes and non-endurance athletes using the 2010 European Society of Cardiology (ESC) guidelines (See table 1), which specify group 1 (common training related) and group 2 (abnormal non-training related) criteria (Table 1). A higher prevalence of both group 1 and group 2 changes were observed in endurance athletes compared to non-endurance athletes. The false positive rate of almost 30%, suggests the specificity of these criteria are relatively poor. In view of this, refined criteria have been developed which better-reflect normal “training-related” changes in the ECG [9, 42]. Brosnan *et al*. [9] reported a significant decrease in the false-positive rate through application of the Seattle criteria.

*[INSERT TABLE 1 HERE]*

*The Left Ventricle*

Many studies have attempted to quantify the magnitude of cardiac adaptation and to understand any relationship to the type of exercise stimulus. Recent work [45, 46] has challenged the traditional theory of dichotomous adaptations induced by endurance and resistance training. Whilst LV cavity dilatation was observed in endurance athletes, neither concentric remodelling nor concentric hypertrophy were discerned in resistance trained athletes [43, 45]. A more recent study has provided further support for this, describing a predominance of normal LV geometry in both endurance and resistance trained athletes [46]. It is unclear whether the differences between findings in these studies can be accounted for by improvements in imaging quality, or changes in athletes’ training methods over the last 40 years. The inclusion of more aerobic conditioning in strength trained athletes’ programmes, and more strength training in endurance athletes’ programmes may provide some explanation for the similar LV geometry between groups.

Longitudinal studies have demonstrated that increases in endurance training volume lead to progressive increases in LV cavity dimension and wall thickness, in a close relationship with development of fat-free mass (FFM) [5, 15, 43]. Spence *et al*. [43] described no structural changes to the LV, whereas Baggish *et al*. [5] observed concentric hypertrophy following 3-6 months of resistance training. These disparate findings may be explained by differences in study specific training volumes, training status of the participants, or imaging methodologies [44]. Further longitudinal studies are required to assess the impact of training volume on structural adaptation in both endurance and resistance training settings.

Previous research has highlighted decreased resting systolic function in endurance athletes, with up to 12% of this group presenting with an “abnormally” low ejection fraction (EF) [1]. This decrease in EF appears to be a consequence of an increased LV end-diastolic volume (EDV) (see table 2), resulting in the need for a lower contraction force to generate the necessary stroke volume. Previous large scale studies have observed no differences in regional or global peak longitudinal strain (ε) between endurance and resistance trained athletes [46]. Furthermore, no differences in peak longitudinal, circumferential, and rotational ε values have been reported between endurance athletes and sedentary controls [46]. However, superior ability to augment systolic function during exercise has previously been demonstrated in athletes [28]. Therefore, in-exercise assessment of LV systolic function provides a useful screening tool in athletes who present with decreased contractility, regardless of sporting discipline.

Increased training volume does not appear to be associated with changes in resting global systolic function [43]. Increases in resting radial, circumferential and longitudinal ε, along with increased LV torsion, have previously been described following 3 months of intense rowing training [5]. Furthermore, an altered base-to-apex gradient was identified in longitudinal and rotational ε patterns, whereby deformation increased most markedly at the apical level [5]. In contrast, Spence *et al*. [43] did not observe changes in longitudinal ε following 6 months of intensive, individualised and carefully supervised endurance or resistance training.

Assessment of diastolic function is complex, as conventional Doppler parameters are not directly related to overall volume, and are dependent upon atrial and LV pressures. It is therefore unsurprising that many studies observe no differences in peak E velocity between endurance trained, resistance trained, and sedentary individuals [45]. That aside, the atrial component, and E/A ratio are often significantly different in endurance athletes, indicative of enhanced early diastolic filling. This is supported by the increased early myocardial mitral tissue velocities (E’) displayed by endurance athletes [21]. It is thought untwisting of the LV plays an important role in lowering LV pressure, and enhancing early diastolic filling during exercise [48]. The reduction in peak left atrial (LA) longitudinal ε observed in high-dynamic athletes provides complimentary evidence for enhanced early diastolic filling, and a decreased atrial component [15].

Neither endurance nor resistance training appear to elicit changes in global diastolic function during training periods up to 6 months [5, 43]. Data pertaining to training volume induced adaptation of diastolic myocardial function is sparse and conflicting. Spence *et al*. [43] reported no change in early or late diastolic longitudinal strain rate (SR) following 6 months of endurance or strength training, whereas Weiner *et al*., [48] highlighted an increase in LV peak untwisting rate following 3 months of intense rowing training.

*The Right Ventricle*

As approximately 4-23% of all sudden cardiac death cases in athletes are due to arrythmogenic RV cardiomyopathy (ARVC), understanding the nature and magnitude of physiological training-induced RV remodelling is vital [11]. Increased RV cavity, inflow and outflow dimensions are observed in endurance athletes, compared to resistance athletes, who have similar chamber dimensions to sedentary individuals [13]. In addition to this, a large proportion of endurance athletes exceed “normal” values for RV inflow and outflow dimensions (57% and 40% respectively) [34]. The prevalence of abnormal RV/LV ratios within this population (66%) also provide evidence for disproportionate loading on the RV during endurance exercise [34].

The work of Spence *et al*. [43] provides further support for this phenomenon, as increased RV cavity dimensions were observed in participants who completed 6 months of endurance training, but not in those who completed resistance training. A study of longer duration (12 months) has also demonstrated a progressive increase in RV:LV ratio in response to high dynamic training, providing further support for disproportionate loading on the RV acting as a stimulus for more marked remodelling compared to the LV [3].

RV enlargement and an increased prevalence of T-wave inversion in endurance athletes, presents a challenge in the differential diagnosis from ARVC. It is therefore important to note that global function is maintained when assessed by tricuspid plane systolic excursion (TAPSE), RV fractional area change (RV FAC), RV myocardial tissue velocities, and peak RV global longitudinal ε [13, 34]. Decreased myocardial function at the base of the lateral RV wall has also been reported in some endurance athletes [28]. This appears to occur in combination with concomitant RV dilatation, generating ambiguity in the pre-participation screening setting [28]. Importantly, ε values are only impacted at a regional level and do not fall as low as those seen in the ARVC patients [28]. Furthermore, a normal physiological response to exercise is maintained, highlighting the potential diagnostic role of exercise echocardiography in ambiguous cases [28].

*The Atria*

Left atrial dilatation is commonly observed in patients with hypertrophic cardiomyopathy (HCM) [31], highlighting the importance of defining normal, training-induced adaptation. Bi-atrial dilatation and increased functional volume is observed in athletes from high-dynamic sporting disciplines [31]. In contrast, there are no structural differences between sedentary individuals and athletes from low-dynamic sporting disciplines [31]. Although cross-sectional studies suggest there are no differences in atrial longitudinal ε between high-dynamic, low-dynamic or sedentary groups [31], an 8 month assessment of athletes completing high dynamic training has demonstrated a progressive decrease in LA ε [14]. It is suggested that, in the presence of normal LV/RV diastolic function, atrial dilatation represents a normal physiological manifestation of the AH. Future research should seek to better characterise the impact of training volume, and detraining, on atrial structure and function.

*[INSERT TABLE 2 HERE]*

**THE IMPACT OF ETHNICITY**

*The 12-Lead Electrocardiogram*

Our knowledge of the athlete’s ECG is primarily based on studies in Caucasian athletes [9]. Recent studies have attempted to document the impact of some ethnicities. In a large study of elite athletes, Sheikh *et al.* [42], reported African/Afro-Caribbean athletes were more likely to present an abnormal ECG compared to their Caucasian counterparts (11.5% vs 5.3%). An earlier study found that T-wave inversion was present in 23% of African/Afro-Caribbean athletes (primarily in contiguous anterior leads), compared to only 3.7% of Caucasian athletes [34]. This T-wave inversion expression is in stark contrast to African/Afro-Caribbean HCM patients, who generally exhibit T wave inversions in the lateral leads (76.9%). T wave inversion extending into lateral leads therefore warrants investigation for the exclusion of pathology in all cases, irrespective of ethnicity [34]. African/Afro-Caribbean athletes also exhibit a higher prevalence of ST-segment elevation. Furthermore, T-wave inversion and convex profile ST-segment elevation were commonly found together in contiguous anterior leads and are likely to represent a physiological, training-induced characteristic of the African/Afro-Caribbean AH (see table 3). In addition, a higher prevalence of early repolarisation (ERP), RV hypertrophy, LA enlargement and RA enlargement were evident in African/Afro-Caribbean athletes.

The prevalence of training-related ECG changes appears to be lower in Arabic/Middle Eastern athletes compared to their Caucasian counterparts, whilst non-training related changes were similar between groups [38]. Based on this, it is recommended that current guidelines are relevant and appropriate in the pre-participation screening of these athletes [38]. It should also be noted however, that a small number of Arabic/Middle Eastern athletes can be expected to present T-wave inversion in contiguous leads, in combination with a convex profile ST-segment elevation (as previously highlighted in African/Afro-Caribbean athletes).

There is a lack of ECG data pertaining to South/East Asian athletes, although we are able to draw some information from a large cohort study (*n=* 18,497) characterising ECG findings in young Singaporean army recruits [33]. Seven percent of subjects exhibited an ECG abnormality. For those who received further assessment, the most common abnormality was increased R/S voltage, followed by right and left axis deviation, right bundle branch block and pathological Q wave expression. Accordingly, East/South Asian athletes are likely to present a similar prevalence of ECG changes to Caucasian and Arabic/Middle Eastern athletes.

*The Left Ventricle*

Similar to our understanding of ECG adaptation, our knowledge of the athlete’s LV is predominantly based on studies of Caucasian athletes [50]. These data have been used to discern “normal” limits for LV wall thickness in males and females. In a comparison between highly trained African/Afro-Caribbean and Caucasian male athletes, a higher proportion of African/Afro-Caribbean athletes presented LV wall thickness values >12 mm [6]. In addition, profound LV hypertrophy (≥15 mm) was demonstrated in a small number (3%) of African/Afro-Caribbean athletes, but not in any Caucasian athletes. Similarly, a higher prevalence of LV hypertrophy has been observed in female African/Afro-Caribbean athletes compared to Caucasian counterparts [36]. Interestingly, no differences have been observed in the LV wall thicknesses of African/Afro-Caribbean and Caucasian sedentary individuals, suggesting African/Afro-Caribbean individuals exhibit a more pronounced training response, rather than a pre-disposition to greater wall thicknesses [6]

Athletic training is associated with increased LV trabeculation, which may mimic LV non-compaction cardiomyopathy (LVNC). Increased trabeculation is more prevalent in African/Afro-Caribbean athletes, compared with Caucasian counterparts [18]. Gati *et al*. [18] also found more African/Afro-Caribbean athletes met two criteria for LVNC, than Caucasian athletes. Of these athletes, a higher proportion of African/Afro-Caribbean individuals also presented deep T-wave inversionand reduced LV EF (3.4% vs 0.5%) [18]. It should be noted however, that T wave inversion is generally expressed in anteroseptal leads in athletes fulfilling LVNC criteria, in stark contrast to LVNC patients where a greater prevalence of inferolateral T wave inversion is observed [18]. Although increased trabeculation appears to be a physiological process in both African/Afro-Caribbean and Caucasian ethnic groups, differentiation between physiological adaptation and LVNC appears to be more challenging in the African/Afro-Caribbean athletic population, with more athletes falling into the diagnostic “grey zone” [18].

Structural remodelling in Arabic/Middle Eastern athletes appears similar in nature to that of Caucasian athletes. Although the magnitude of adaptation appears to be smaller in Arabic/Middle Eastern athletes, differences in LV mass can be negated via scaling to BSA, suggesting this finding may simply express differences in body-size [38]. Furthermore, a similar prevalence of LV hypertrophy was presented by Arabic/Middle Eastern, African/Afro-Caribbean and Caucasian athletes [38]. Global measures of LV function appear to be consistent across Arabic/Middle Eastern, African/Afro-Caribbean and Caucasian athletes, with all groups presenting normal systolic function and diastolic filling.

Although data regarding LV adaptation in South/East Asian athletes is sparse, both male and female Chinese athletes appear to display a similar magnitude and prevalence of LV cavity dilation and hypertrophy to Caucasian counterparts [29]. Structural morphology has also been studied in a group of Japanese ultramarathon runners. Extreme LV dilatation (LVIDd ≥70 mm) was reported in 11.3% of their cohort, combined with LV wall thickness values up to 19 mm [32]. Due to the lack of data available for comparison, it is impossible to confirm whether these findings reflect an ethnically mediated physiological adaptation to ultra-endurance training, a unique training volume, pathologic expression, or simply weak measurement methodology, although the latter is most likely as these findings have not been reproduced in similar studies.

Global systolic and diastolic measures of function can be expected to fall within normal ranges for African/Afro-Caribbean, Arabic/Middle-Eastern, South/East Asian and Caucasian athletes [6, 29, 38]. Currently, there are no data pertaining to ethnically mediated adaptation in LV mechanics. Further study is this area is warranted.

*The Right Ventricle*

To the best of our knowledge, there is only one study which has assessed RV structure in African/Afro-Caribbean athletes, highlighting similar RV structural adaptation to Caucasian athletes [52]. Importantly, in the context of pre-participation screening, the combination of ECG and structural criteria for ARVC is more commonly met by African/Afro-Caribbean athletes compared to Caucasian athletes, creating a greater diagnostic challenge in this group. The lack of data for other ethnicities highlights the need for further work to establish the impact of ethnicity on RV structure and function.

*The Atria*

While there is no data pertaining to RA adaptation in African/Afro-Caribbean athletes, larger LA dimensions have been discerned in this group of athletes compared to Caucasian athletes [36]. The clinical/physiological consequences of this finding remain unclear. There are no data pertaining to other ethnic groups and therefore until further work is undertaken to establish ethnic variance our existing normal ranges should be applied to all.

[*INSERT TABLE 3 HERE*]

**THE IMPACT OF BODY SIZE**

Although athletic training is known to increase cardiac dimensions, inter-population comparison is challenging due to anthropometric differences. Indexing of cardiac dimensions aims to provide body-size and/or body-composition independent values, providing a better platform for comparison [20]. The many methods of indexing come with their own merits and flaws which may impact on interpretation. Simple ratio-metric scaling is the most common approach to scaling, whereby a cardiac measurement is indexed to a body size parameter (i.e. *y/x*). This method has been criticised as relationships between cardiac dimensions and body-size are rarely linear [20]. In contrast, allometric scaling methods produce a curvi-linear “line of best fit”, and come close to generating body-size independent values [20]. In order to scale allometrically, the size parameter should be indexed to the scaling factor raised to the power of the defined exponent (*y = axb*). Once determined, the resultant scaled index will not correlate with the original body size factor. The value of indexing is also reliant upon the body size variable selected (body mass, body surface area, height or fat free mass (FFM)).

*The Left Ventricle*

Height, body mass, and body surface area (BSA) represent the most common indexing parameters due to their ease of access. A number of large scale studies have sought to generate an appropriate *b* exponent to facilitate between-study comparison of LV mass [20]. Using height as an example, *b* exponent values generated by this work range from 1.97-3, reflecting differences in cohort age, sex, and physical fitness [20]. A similar range of *b* exponents have been described for indexing to body mass, highlighting the challenge of producing a “one size fits all” value. More recently, the efficacy of FFM as an indexing variable has become clear [14, 43, 49]. In order to determine FFM (fat mass subtracted from total body mass), firstly an individual’s body composition must be measured. This measurement may be carried out using skinfold callipers, dual energy x-ray absorptiometry (DEXA), or magnetic resistance imaging (MRI). DEXA, which uses two different x-ray intensities to differentiate between lean and fat body mass, is commonly used in the literature because of its greater accuracy compared to skinfold calliper measurements, and relative inexpensiveness compared to MRI.

Whalley *et al*. [49] found ratiometric scaling of LV mass and LVIDd to FFM provided a stronger correlation than BSA or height2.7. This method appears to overcome many of the limitations of extreme body anthropometry observed in athletes, as LV mass and FFM develop concurrently [14]. It should be noted however, that even in athletes displaying extreme anthropometry, physiological adaptation of the LV appears to be proportional to body size and remain within “normal” limits [37]. Scaling of LV structures to FFM therefore appears to be optimal, although use of BSA with a population specific *b* exponent will also generate acceptable, body-size independent values (see table 4).

*The Right Ventricle*

Data pertaining to scaling of RV structural parameters is limited, likely representing the difficulty of imaging the RV, and its challenging geometry [20]. Although a linear relationship between RVIDd and BSA has previously been described, this may have been fortuitous, as George *et al*. [20] found no significant linear relationships between RVIDd measurements and body mass, height or BSA. Body size independent measurements of RVOT, RVI and RV length are feasible however, using allometric scaling with population specific *b* exponents [34]. Use of these indexing methods may provide greater sensitivity in the identification of ARVC, when compared to conventional guidelines, which are commonly exceeded by athletes. Furthermore, Oxborough *et al*. [34] observed body-size independence in functional assessment of the RV (using absolute ε values).

*The Atria*

Relatively little research has been carried out regarding scaling of the LA, and no data pertaining to indexing of the RA are available. Like the LV, the LA appears to confirm to conventional geometrical similarity [20]. George *et al*., [20] observed a significant linear relationship between height and LAD using ratiometric scaling, suggesting this simple approach may be appropriate for body-size independent measurement. More recent work has questioned this, and found indexing of LAD to BSA with a population specific *b* exponent provided more valid, body-size independent values [22]. The efficacy of scaling LA volume to FFM has also been described, with a cohort specific *b* exponent of 0.7 being optimal for both male and female collegiate-level athletes [22].

*The Aorta*

Although this literature review is focused on athletic development of cardiac chambers, it is also important to acknowledge the impact of body size on adaptation of the aorta in the athlete’s heart. Particularly as a small number of SCD cases in athletes can be attributed to aortic dissection. Although correlations between aortic root dimension (at the sinus of Valsalva) and both BSA and height have previously been identified [8, 35, 37], investigation to exclude Marfan syndrome is warranted in males presenting dimensions >40 mm (or >34 mm in females), irrespective of extreme anthropometry [16, 37].

[*INSERT TABLE 4 HERE*]

**THE IMPACT OF SEX**

The relative scarcity of data defining “normal” athletic adaptation of all four cardiac chambers in female athletes presents a challenge to clinicians. Inter-sex differences in body-size, body composition and hormonal profile therefore present challenges in pre-participation screening [39]. While early research has consistently demonstrated smaller cardiac dimensions in female athletes (compared to males), recent work has sought to eliminate inter-sex differences in body-size and composition to isolate the influence of athletic training on the female AH [22].

*The 12-Lead Electrocardiogram*

In a comparison between elite female and male rowers, a similar prevalence of abnormal ECG findings were observed between sexes (3% and 4% respectively), but with profound differences in the prevalence of specific training-related changes [47]. A higher prevalence of early repolarisation pattern (ERP) was reported for male athletes compared to their female counterparts (76% vs 23%) [47]. Interestingly the increased overall prevalence of ERP in male athletes appears to be driven by a higher prevalence of anterior lead ERP, as the prevalence of lateral and/or inferior lead ERP was similar between males and females. Females are also far less likely to display isolated QRS voltage criteria for LV hypertrophy compared to their male counterparts (8% vs 51%) [47].

*The Left Ventricle*

Male athletes consistently display larger LV cavity dimensions and wall thicknesses compared to their female counterparts [22]. LV hypertrophy in female athletes (wall thickness >11mm) is extremely rare, compared to male athletes where a minority of athletes (2.5-5% prevalence in males) can be expected to present thicknesses >12mm [50]. Whether these differences can be accounted for by body-size, or whether a sex-specific difference in physiological remodelling exists is a contentious issue [22]. Rowland *et al*. [39] reported larger LV dimensions in male athletes compared to their female counterparts despite indexing for BSA, BSA0.5, and FFM-1 (derived from skin-fold measurements). These findings may represent weak scaling methodology as opposed to physiological differences however, as scaling of LV structural parameters to FFM (derived using dual energy x-ray absorptiometry) eliminates any meaningful inter-sex differences (see table 5) [22]. Remodelling of the LV in females appears to follow a similar pattern, time-scale and relative magnitude to that observed in male athletes [22].

Global systolic function is similar between male and female athletes, with differences in absolute LV SV being eliminated by scaling to FFM [22]. Although Giraldeau *et al*. [22] reported a slightly higher LV EF and global longitudinal ε in females compared to males (66% vs 63% and -22% vs -20.6% respectively), this did not translate into meaningful differences in LV SV index, or systolic longitudinal SR. Furthermore, Giraldeau *et al*., [22] observed a higher early diastolic longitudinal SR in female athletes compared to their male counterparts (1.81 %/s vs 1.56 %/s). Further examination in a large heterogeneous cohort is required to confirm these findings.

One potential confounding factor in the pre-participation screening of female athletes is the menstrual cycle, and possible influence of contraceptive methods. Fluctuations in oestrogen throughout the menstrual cycle are known to impact both central and peripheral cardiovascular factors, including blood volume and total peripheral resistance, yet disagreement exists as to whether these factors impact LV function [23]. Fuenmayor *et al*. [17] observed a significant difference in mitral valve E/A ratio between follicular and luteal phases, whereas George *et al*. [19] reported no meaningful differences in functional parameters between these time-points. Further examination into the impact of the menstrual cycle on LV function, including STE indices, will provide important insight for pre-participation screening.

*The Right Ventricle*

Much like the LV, larger RV structural dimensions are observed in male athletes compared to their female counterparts [22]. Giraldeau *et al*. [22] observed that inter-sex differences in chamber dimensions can be eliminated by indexing to FFM, suggesting differences in structural parameters can be accounted for by body-size [22].

Giraldeau *et al*. [22] observed a higher early diastolic longitudinal SR in the RV free wall in females compared to males, suggestive of slightly enhanced diastolic function at rest in female athletes. No other inter-sex differences were observed in STE derived functional indices. To the best of our knowledge, no research has been carried out to examine the impact of the menstrual cycle on RV function. This may represent an excellent research opportunity, as any effect of the menstrual cycle on LV function is likely to be magnified in the RV, due to disproportionate haemodynamic loading.

*The Atria*

Training-induced bi-atrial dilatation is observed in male and female athletes, with smaller absolute dimensions being displayed by female athletes [5, 22]. The relative magnitude of physiological adaptation in LA dimensions appears to be similar between male and female athletes [22]. There are insufficient data to comment on the inter-sex difference in RA adaptation. Like ventricular structures, inter-sex differences in LA and RA volume are eliminated when indexed to FFM, indicating a close relationship to body size [22].

[*INSERT TABLE 5 HERE*]

**THE IMPACT OF AGE**

In a large study (*n*= 1210) of SCD in the US general athletic population, Harmon *et al*., found the mean age of cases to be only 17 years [24]. This finding is supported by Italian, Israeli, Danish and Swedish groups, who described similar prevalence of SCD in young athletes [24]. The efficacy of cardiovascular screening in school-age athletes has previously been demonstrated by a reduction in sudden death rates from 1:28,000 to 1:250,000 following implementation of a screening programme [24], yet there is no consensus on how age should impact classification of normal/abnormal findings in athletic individuals. The growth of participation in competitive sport at masters and veteran levels has also increased the need for understanding of the impact of ageing upon cardiac adaptation.

*The 12-Lead Electrocardiogram*

Youth athletes (aged 10-15 years) can be expected to present fewer abnormal (10% vs 40%) and mildly abnormal (3-8% vs 19-36%) ECG traces compared to their senior counterparts [27]. This is likely a result of fewer cumulative training hours and may also be influenced by the higher levels of body fat and lower levels of sex hormones observed in this group [27]. Within the abnormal patterns presented by youths, there is a strikingly high prevalence of anterior T-wave inversion, raising concerns over ARVC [4]. However, T-wave inversion appears to be a feature of immaturity rather than pathology in this group, and a progressive decline in precordial T-wave prevalence is observed during adolescence (32.2% in 6-8 year olds compared to 3.3% in 16-18 year olds) [4] (see table 6). Development of refined criteria, which factor in the chronological age and anthropomorphic characteristics of young athletes may be appropriate to minimise the rate of false positive ARVC diagnoses [4]. The prevalence of LV hypertrophy (using isolated Sokolow criteria) is considerably lower in junior male athletes (15%) compared to senior male athletes (51%) [7]. The lack of data pertaining to training and non-training related ECG changes in veteran athletes, warrants further exploration.

*The Left Ventricle*

Development of an increased LV cavity size is generally observed in non-athletic males and females between birth and 30 years of age, followed by a progressive decline as age increases [26]. LV cavity enlargement and wall thickness are increased in junior athletes compared to age matched non-athletic controls, but are less pronounced compared to their senior counterparts due to a lack of physical maturity and fewer cumulative training hours [30, 40, 41]. Despite this, an LV cavity dimension >60 mm in the presence of diminished systolic or diastolic function represents an appropriate indicator for the investigation of dilated cardiomyopathy (DCM) in both adolescent and senior athletes [30]. Similarly, conventional guidelines which warrant investigation to exclude HCM (LV wall thickness >12 mm in males or >11 mm in females) are also applicable to the athletic adolescent population [40].

It is clear that a relationship between lifelong exercise “dose” and LV cavity size exists. Carrick-Ranson *et al*. [10] observed significantly higher LV EDV index values in master athletes, compared with those of age-matched sedentary individuals and casual exercisers. Furthermore, some structural adaptation initiated by athletic training may remain present more than ten years after cessation of participation [10]. It should be noted however, that sporting discipline appears to be a factor in the longevity of structural adaptation, as preservation of the AH phenotype has been discerned in retired wrestlers, but not in retired marathon runners (in an age-matched cohort).

Ageing does not appear to be associated with changes in global measures of systolic function in healthy non-athletic individuals [10]. Furthermore, no differences in EF are observed between sedentary young individuals, junior athletes, sedentary older individuals, and master athletes [30, 40]. A progressive decrease in peak longitudinal ε is observed throughout the lifespan of healthy, non-athletic individuals [26]. Whilst some evidence suggests exercise may have a protective effect on this decrease in LV longitudinal deformation [26], further clarification is needed. Although there does not appear to be an age-related change in peak rotational, circumferential or radial ε, a shift in the base-apex deformation gradient has been identified within these planes [26]. It therefore appears that systolic function is maintained with progressing age through increased action of the apical region of the LV, which compensates for decreased deformation at basal level [26].

Global diastolic function is comparable between junior athletes and age matched non-athletic individuals [30, 40]. Following maturation, a gradual decrease in the trans-mitral E/A ratio is observed, as a greater reliance is placed upon the atrial component of LV filling [26]. Although master athletes and sedentary age-matched individuals display similar contractile function, master endurance athletes display significantly greater ventricular compliance and decreased wall stress, resulting in LV pressure-volume relationships similar to those of young healthy individuals [2]. Future research should seek to clarify the impact of age on temporal diastolic deformation characteristics, in rotational, circumferential, and radial planes.

*The Right Ventricle*

As in the LV, development of RV cavity size is observed throughout adolescence in young athletes [20], likely reflecting an accumulation of training hours and physical maturation. At the other end of the spectrum, decreasing RV chamber area is observed in non-athletic, ageing individuals [25]. Whether life-long training has a protective effect on these decreases is unknown, and represents an opportunity for future research.

To the authors’ knowledge, there are no data available pertaining to global function of the adolescent athlete’s heart. Systolic function in ageing individuals is characterised by decreases in RV S’, and RV peak ε [12]. In addition, RV systolic reserve appears to decrease with advancing age, resulting in a reduced ability to augment RV deformation [12]. Diastolic function of the RV diminishes with advancing age in sedentary individuals. A decrease in trans-tricuspid E/A ratio is observed, along with decreased ability to augment diastolic function during exercise stress [12].

*The Atria*

Data regarding the impact of ageing on the athlete’s atrial structure and function are limited. It is clear however, that LA cavity size increases throughout adolescence in junior athletes, most likely as a function of cumulative training hours and physical maturation [20]. Life-long endurance athletes can also be expected to present significantly larger atria compared to sedentary age matched controls [51]. To the best of our knowledge, there are no data available regarding the impact of age on the RA. Future research which develops understanding of structural and functional adaptation of the atria with advancing age will therefore be highly valuable to practitioners.

*[INSERT TABLE 6 HERE]*

**CONCLUSION**

Many factors contribute to athletic cardiac adaptation, and as such the “AH phenotype” fulfils a broad spectrum of electrophysiological, structural and functional characteristics. These inter-individual training induced adaptations influence interpretation of both the ECG and echocardiogram in a pre-participation screening environment. The risk of false-negative diagnoses, or unnecessary disqualification of an athlete due to false-positive diagnoses, highlight the need to approach pre-participation screening with consideration for the individual. By quantifying the impact of training volume, sporting discipline, ethnicity, body size, sex and age on structural and functional components of the AH phenotype, clinicians will be better equipped to discern physiological and pathological adaptation, and reduce both false-negative and false-positive rates.

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**FIGURES AND TABLES**

*Table 1. Classification of ECG characteristics based on the 2010 ESC recommendations*

|  |  |
| --- | --- |
| ESC Classification of ECG Abnormalities in athletes | |
| Group 1 (training-related) | **Group 2 (training-unrelated)** |
| Sinus Bradycardia | T-wave inversions |
| First degree AV Block | ST-segment depression |
| Incomplete RBBB | Pathological Q-waves |
| Early Repolarisation | Left Atrial Enlargement |
| Isolated QRS voltage criteria for LVH | Left axis deviation / left anterior hemiblock |
|  | Right axis deviation / left posterior hemiblock |
|  | Right Ventricular Hypertrophy |
|  | Ventricular pre-excitation |
|  | Complete LBBB or RBBB |
|  | Long QT or short QT interval |
|  | Brugada-like early repolarisation |

*Table 2. Key points to consider regarding* ***SPORTING DISCIPLINE*** *for pre-participation screening*

|  |
| --- |
| Key points for pre-participation screening |
| Physiological eccentric hypertrophy of the LV is often presented by endurance athletes, however concentric hypertrophy and concentric remodelling is rare in any athlete, and warrants further investigation |
| Endurance athletes can be expected to present normal or superior diastolic function compared to the non-athletic population, and hence any deviation from normal function warrants further investigation |
| Athletic structural adaptation of the RV may mimic ARVC in some cases. Exercise -echocardiography should be used to ensure a normal physiological response to exercise |
| Extended periods of very high training volume are associated with development of ECG abnormalities, which mimic pathological adaptation. Detraining periods can be employed to discount pathology in this situation |
| Extended periods of moderately increased training volume are associated with increased LV mass, RV dilatation, and maintenance or improved resting global diastolic function |
| Severe acute increases in training volume are associated with significant decreases in systolic and diastolic myocardial function of the LV and RV. In the case whereby function remains diminished five days after the acute bout of exercise, further investigation is warranted |

*Table 3. Key points to consider* ***REGARDING ETHNICITY*** *for pre-participation screening*

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| --- |
| Key points for pre-participation screening |
| T-wave inversion, accompanied by a convex profile ST-elevation in leads V1-4 is common in African/Afro-Caribbean athletes, and is likely to represent a physiological component of the AH phenotype in this ethnic group |
| The prevalence of training-induced LV hypertrophy is greater in African/Afro-Caribbean athletes compared to their Caucasian, Middle Eastern and Arabic counterparts. Differentiation between physiological trabeculation of the LV, and LVNC is also more challenging in African/Afro-Caribbean athletes, compared to their Caucasian counterparts |
| Structural adaptation of the RV appears to be similar between African/Afro-Caribbean and Caucasian athletes |

*Table 4. Key points to consider* ***REGARDING BODY SIZE*** *for pre-participation screening*

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| --- |
| Key points for pre-participation screening |
| Allometric scaling (with generation of a population specific b exponent), is more effective than ratiometric scaling, in terms of generating body-size independence in structural parameters |
| Indexing of structural parameters to FFM (measured using DEXA), is superior to BSA, height and body mass. Indexing to BSA may be preferable however, due to its ease of access |
| Indexing of RV structural parameters may improve the sensitivity of ARVC diagnosis in athletes, who often present with extreme anthropometry |

*Table 5. Key points to consider* ***REGARDING GENDER*** *for pre-participation screening*

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| --- |
| Key points for pre-participation screening |
| Training-induced adaptation of the LV, RV and LA, appears to follow a similar pattern, time-scale, and relative magnitude between genders |
| Presentation of LV hypertrophy is rare in male athletes, and extremely rare in female athletes |
| Fluctuations in blood volume and hormonal profile during the menstrual cycle may impact on the interpretation of global diastolic function in the LV |

*Table 5. Key points to consider* ***REGARDING SEX*** *for pre-participation screening*

|  |
| --- |
| Key points for pre-participation screening |
| Training-induced adaptation of the LV, RV and LA, appears to follow a similar pattern, time-scale, and relative magnitude between genders |
| Presentation of LV hypertrophy is rare in male athletes, and extremely rare in female athletes |
| Fluctuations in blood volume and hormonal profile during the menstrual cycle may impact on the interpretation of global diastolic function in the LV |

*Table 6. Key points to consider* ***REGARDING AGEING*** *for pre-participation screening*

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| --- |
| Key points for pre-participation screening |
| Conventional guidelines which warrant investigation into DCM and HCM are also applicable to adolescent athletes |
| T-wave inversion is more prevalent in adolescent athletes compared to senior athletes, and can be expected to diminish throughout the maturation process |
| Ventricular compliance is maintained in masters athletes, preventing the decreases in diastolic function observed in sedentary age-matched individuals |

Figure 1. The multifactorial impact of cardiac adaptation in the athlete

