

Echocardiographic measurements of aortic root diameter (ARD) in collegiate football Athletes at pre-participation evaluation

Fred Reifsteck,¹ James R Clugston,² Stephen Carek,² Kimberly G Harmon,³ Breton Michael Asken,⁴ Michael C Dillon,⁵ Joan Street,⁶ Katherine M Edenfield^{6,2}

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¹University Health Center, University of Georgia Athletic Association, University of Georgia, Athens, Georgia, USA

²Community Health and Family Medicine, University of Florida, Gainesville, Florida, USA

³Family Medicine and Orthopaedics and Sports Medicine, University of Washington, Seattle, Washington, USA

⁴Clinical and Health Psychology, University of Florida, Gainesville, Florida, USA

⁵The Cardiac and Vascular Institute, Gainesville, Florida, USA

⁶Student Health Care Center, University of Florida, Gainesville, Florida, USA

Correspondence to

Dr Fred Reifsteck;
reifsteck@uhs.ufl.edu

ABSTRACT

Background Some remodelling of the aortic root may be expected to occur with exercise but can already vary due to different body sizes, compositions and genetic predispositions. Attributing the cause of borderline aortic root diameter (ARD) values to either physiological or pathological conditions in American college football athletes is difficult as there is very limited normal reference values in this population. Body surface area (BSA) specific norms are thought to be useful in other cardiac measurements of football athletes.

Methods A retrospective cohort review of pre-participation examination (PPE) transthoracic echocardiogram data from collegiate football athletes was performed. ARD was analysed by field position (linemen, n=137; non-linemen, n=238), race (black, n=216; white, n=158) and BSA for predictive value and associations. Values were compared with non-athlete norms, and collegiate football athlete-specific normal tables were created.

Results Only 2.7% of football athletes had ARD measurements above normal non-athlete reference values and the mean athlete ARD values were *lower* than non-athlete values. No athletes had an aortic root >40 mm or were disqualified due to underlying cardiac pathology. Univariate analyses indicated linemen position and increasing BSA was associated with larger values for ARD. BSA outperformed race in predicting ARD. Normal tables were created for ARD stratified by BSA group classification (low, average and high BSA). Proposed clinical cut-offs for normal and abnormal values are reported for raw echocardiograph metrics and their BSA indexed scores.

Conclusions Non-athlete reference values for ARD appear applicable for defining upper limits of normal for most collegiate football athletes. BSA-specific normal values may be helpful in interpreting results for athletes that exceed non-athlete norms.

INTRODUCTION

Participation in American style football (ASF) at the collegiate level requires a pre-participation evaluation (PPE). Institutions have traditionally performed history and physical examinations but many are now also

What are the new findings?

- ▶ Non-athlete reference values for aortic root diameter (ARD) may be applicable for defining upper limits of normal in most collegiate football players, as the mean athlete raw ARD and ARD/body surface area (BSA) index scores in our cohort were actually *lower* than previously published non-athlete values.
- ▶ BSA should be considered when interpreting ARD values at or exceeding the upper limits of normal in athletes, as we found it to have the most significant association.
- ▶ Previously defined values for ARD in an older and professional athlete population are larger than our study population and may not be applicable to college-aged football athletes.

including non-invasive cardiac testing such as ECG and echocardiogram.¹ PPEs aim to uncover potentially serious or life threatening health conditions, and history and physical alone are generally not sensitive enough for undetected cardiac conditions.² The use of these non-invasive tests may add to the sensitivity and specificity of the evaluation if appropriate normal reference values are available. Non-athlete norms may not apply to collegiate ASF (C-ASF) athletes, as non-pathological adaptation and remodelling of the heart frequently occur with athletic activity and these C-ASF athletes tend to have larger body surface area (BSA) than most people.

There are limited data regarding aortic root dimension of ASF players. Strength sports, such as ASF, which utilise static anaerobic exercise in certain positions, may increase the degree of aortic remodelling and dilation. The extent of aortic remodelling in athletes is not completely understood, especially for ASF players who typically have high training demands and increased BSA.

Historically, an aortic root diameter (ARD) >40 mm represents a cut-off for disqualification from high-intensity exercise and evaluation for underlying connective tissue disorders, such as Marfan's syndrome.³ It is well established that the evaluation of aortic root dimensions should consider age as well as anthropometric variables, such as height and BSA, in non-athletic populations.^{4 5} Pelliccia *et al* suggest that the most haemodynamically intense endurance disciplines, such as cycling and swimming, are associated with a mild increase in aortic dimension.⁶ However, these increases in aortic root dimensions are small and fall within the established limits for the general population. Additional studies have supported the use of BSA as a strong determinant of aortic dimensions.⁷⁻⁹ Sports with extremes of BSA and height, such as basketball and volleyball, have shown a higher prevalence of athletes with aortic roots >40 mm than most athletic populations but have demonstrated a plateau of aortic dimensions at the uppermost of height and BSA.^{10 11}

Understanding the sport-specific cardiac structural findings in ASF players is important to better utilise echocardiographic screening while avoiding unnecessary testing or disqualification of athletes.

We sought to contribute echocardiographic reference values for ARD at the sinus of Valsalva in C-ASF athletes at the time of their PPE. We examined this value due to its association with cardiac conditions which can predispose to sudden cardiac death such as Marfan's syndrome and aortic root aneurysm/dissection and the ability to view it easily with echocardiogram. We also sought to determine the applicability of current non-athlete echo norms to our sample.

METHODS

Transthoracic echocardiographic data from ASF athletes in the University of Florida Athletic Association Cardiac Databank collected between 2012 and 2017 was combined with similar, de-identified data from the University of Georgia collected between 2010 and 2015. All studies were performed as part of routine PPE at the time of athlete matriculation.

Most studies (>60%) were performed in mass by Athletic Heart, LLC¹² during screening days held at each institution. Athletes who missed these screening days had echocardiograms performed on a GE Vivid E9 echocardiography machine with an M5 cardiac probe at each institution's designated cardiology office. All echocardiograms were read by cardiologists at the athlete's institution. Twelve different cardiologists contributed to interpreting these studies with the breakdown of percentage read by each as follows: 49%, 35%, 9%, 2%, 2% and seven cardiologists each reading <1% of total studies. Routine values were measured from a parasternal long-axis view as per American Society of Echocardiography guidelines.¹³ Measurements were calculated initially by the echocardiographic technicians at the time of the study, with the

interpreting cardiologist making corrections as needed before the final results were reported.

Our overall sample included 375 male C-ASF athletes, of which 340 had ARD measurements. The 35 participants without ARD data did not differ from the other athletes included in the analyses on any demographic factor. Height and weight were measured at the time of PPE. Race and position played were noted for each subject. The race of the athletes was self-reported in the majority of cases but if unavailable it was determined by the investigators through review of publicly available photographic roster information.^{14 15} Subgroup analysis of race was confined to Black and White athletes, as there was only 1 athlete in the data set of another race. Position played was classified as linemen (offensive/defensive guard, offensive/defensive tackle, centre, defensive end or tight end) or non-linemen (running back, receiver, quarterback, linebacker, cornerback, safety, punter, kicker) and determined in most cases by athlete report at the time of their PPE or, if unavailable, by review of publicly available roster listings.^{14 15}

The primary outcome variable was ARD at the sinus of Valsalva, measured in mm. Predictor variables of interest included player position (position: dichotomised as linemen and non-linemen), race status (race: Black and White), heart rate (HR), left ventricular ejection fraction (LVEF) and BSA (m²) calculated as the square root of (height in cm×weight in kg)/3600).¹⁶ Previous studies have considered height and weight; however, since height and weight are used to calculate BSA and are thus strongly correlated, we elected to report only BSA. Given the homogeneity of ages in our sample (mean (SD) (range): 18.8 (1.0) (17–23) years), we did not assess for age effects.

Statistical analyses

All analyses were performed using the IBM SPSS statistical package, V.24. Independent samples t-tests were used to assess ARD differences based on race and position. For continuous predictor variables (BSA, HR and LVEF), Pearson bivariate correlations were used. Factors demonstrating a univariate association with a given outcome measure were considered for inclusion in a multiple linear regression.

A primary goal of this study was to determine if ARD normal reference values¹³ for non-athletes applied to our collegiate football athletes. First, we evaluated whether differences existed between the current study's healthy athlete sample and widely used normal values¹³ derived from non-athlete samples for ARD. Cohen's d was calculated as an indicator of the magnitude of the sample differences by extracting the mean, SD and associated sample size data from previously published non-athlete normal references.

We then further investigated demographic-specific influences on the current ARD values. If our prior analyses suggested that a given factor explained significant variance in ARD, then that factor would be used for

Table 1 General characteristics of studied population of collegiate football players at the time of PPE

Characteristic	Race			P value*	Position group		P value*
	N=340	Black (N=197)	White (N=142)		Non-linemen (N=214)	Linemen (N=126)	
		Mean (SD) (range)	Mean (SD) (range)		Mean (SD) (range)	Mean (SD) (range)	
Age, years	18.3 (1.0) (17–23)	18.2 (0.9) (17–21)	18.5 (1.2) (17–23)	0.002	18.4 (1.1) (17–23)	18.2 (0.9) (17–21)	0.049
Height, cm	186.7 (6.8) (167.6–201.0)	186.4 (6.5) (167.6–201.0)	187.1 (7.2) [170.2–200.7]	0.362	183.8 (6.1) (167.6–196.9)	191.6 (4.9) (179.7–201.0)	<0.001
Weight, kg	102.5 (30.0) (61.2–162.2)	103.3 (21.3) (66.9–162.2)	101.4 (20.6) (61.2–155.4)	0.400	90.7 (11.0) (61.2–125.9)	122.4 (18.7) (77.1–162.2)	<0.001
BSA, m ²	2.30 (0.26) (1.72–2.94)	2.30 (0.26) (1.84–2.93)	2.29 (0.26) (1.72–2.94)	0.554	2.15 (0.15) (1.72–2.56)	2.55 (0.21) (1.97–2.94)	<0.001
Heart rate, bpm	68.6 (11.5) (43–105)	67.6 (11.6) (43–105)	69.9 (11.4) (48–104)	0.076	67.2 (11.2) (43–99)	71.1 (11.9) (51–105)	0.004
LVEF	59.9 (5.1) (48–80)	59.7 (5.0) (50–80)	60.1 (5.3) (48–78)	0.440	59.8 (5.3) (50–80)	59.9 (4.8) (48–76)	0.938

The 340 participants is a subset of a larger sample (N=375) with additional echocardiograph measures. No demographic differences were noted between those with (N=340) and those without (N=35) available ARD data. Other missing data within the ARD sample (N): race (1), heart rate (2), LVEF (6).

*Demographic characteristic differences assessed using independent samples t-test.

ARD, aortic root diameter; LVEF, left ventricle ejection fraction; bpm, beats per minute.

creating more representative reference value tables. We established an a priori requirement of at least a medium effect size association (Cohen's $d > 0.5$ or $r > 0.3$) as our criteria for factor-specific reference value tables. We sought also to report our findings of ARD individually and indexed to BSA.

Patient and public involvement statement

This research was done without patient involvement. Patients were not invited to comment on the study design and were not consulted to develop patient relevant outcomes or interpret results. Patients were not invited to contribute to the writing or editing of this document for readability or accuracy.

RESULTS

Our sample with ARD measurements included 340 male C-ASF athletes (57% black, 62% non-linemen). Descriptive statistics are shown for demographic and primary predictor variables for the overall sample stratified by race and position (table 1). shows descriptive statistics for demographic and primary predictor variables for the overall sample stratified by race and position. As expected, linemen had significantly higher height, weight and BSA than non-linemen. ARD values showed a normal distribution (figure 1).

Determination of appropriateness of non-athlete norms

Comparison of the current sample's ARD values to non-athlete normal reference data¹³ revealed differences with medium or large effects. Our overall C-ASF sample had lower mean ARD values ($d = 1.38$, large effect) than non-athlete normal reference values. We had 10 athletes

(2.7%) with greater than normal values as defined in a non-athlete population, none of which had an ARD >40 mm.

Independent effects of race, position and BSA

Demographic-specific influences evaluated within our C-ASF study sample showed significant differences within position groups (table 2). No race differences in ARD were observed. For position, linemen exhibited significantly higher ARD ($d = 0.55$, medium effect) than non-linemen. As expected, BSA correlated very strongly

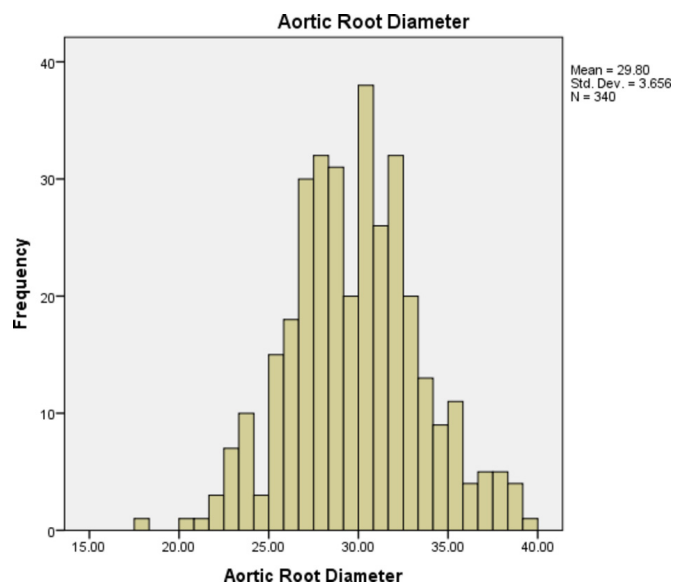


Figure 1 Histogram of aortic root diameter at the sinuses of Valsalva distribution in collegiate football athletes.

Table 2 Echocardiogram characteristics of collegiate football players

Echo metric (mm)	Race			Mean difference (95% CI)	Effect size Cohen's d	Position group		Mean difference (95% CI)	Effect size Cohen's d
	Overall	Black	White			Non-linemen	Linemen		
ARD	29.8 (3.7) (18.0–39.7)	29.8 (3.9) (18.0–39.7)	29.8 (3.3) (23.0–39.0)	0.02 (–0.8 to 0.7)	0.01	29.1 (3.4) (20.6–39.7)	31.0 (3.7) (18.0–39.0)	2.0 (1.1 to 2.8)	0.55*

Sample size (N) for ARD: overall (340), Black (197), White (142), non-linemen (214), linemen (126).

*P<0.001.

ARD, aortic root diameter.

with height ($r=0.736$, $p<0.001$) and weight ($r=0.991$, $p<0.001$); therefore, only BSA effects were further evaluated. Similar results with demographic associations were found when using BSA-indexed scores instead of raw scores (table 3).

Associations between echocardiogram values and continuous predictor variables (BSA, HR and LVEF) were explored. BSA correlated positively with ARD ($r=0.379$, $p<0.001$, medium effect). No associations between HR or LVEF and ARD were observed.

Only position and BSA (not race) were significantly associated with ARD. The χ^2 analyses indicated no association between race and position ($\chi^2(1, 375)=0.196$, $p=0.658$). However, position and BSA correlated very strongly ($r=0.750$, $p<0.001$, large effect), suggesting likely multicollinearity effects. The decision was made to stratify normal reference values by BSA rather than position. We based this decision on previous research highlighting the importance of BSA in cardiac measurement outcomes, and investigator consensus that BSA more appropriately characterises the 'risk' for differences in ARD than the position played by the participant (ie, a non-lineman with a high BSA is presumably at the same 'risk' as a lineman with equivalent BSA).

Reference values for collegiate football athletes stratified by BSA group

BSA was transformed into a categorical factor based on 1-SD cut-points in the BSA distribution, creating below average ($Z<-1$), average ($-1<Z<1$) and above average

($Z>1$) reference groups. Analyses of variance confirmed that the BSA groupings preserved the linear association between BSA and ARD ($F(2,337)=18.984$, $p<0.001$), with significant mean differences in the expected direction (below average<average<above average). Additional comparisons to non-athlete normal ranges were made using the BSA subgroups in our C-ASF athlete sample (combined linemen and non-linemen).

Tables 4 and 5^{4 13 17} show ranges for 'normal' and possible 'abnormal' cutoffs for each normal reference group, using both raw ARD values, as well as index scores calculated by dividing each participant's measurements by their BSA. Values were determined to be 'normal' if they fell before the 95th percentile in the distribution, or all values with a Z-score less than 1.6. 'Mildly abnormal', 'moderately abnormal', and 'severely abnormal' were defined by values falling in the 95th–98th percentile ($1.6<Z<2.0$), 98th–99th percentile ($2.0<Z<2.5$) and >99th percentile ($Z>2.5$), respectively.

Of note, despite using the BSA index score in the current sample, we still observed the same linear association between BSA group and ARD measurements ($p<0.001$), suggesting BSA group-specific normal reference values remain warranted even when using index scores that attempt to account for BSA differences. We once again compared our C-ASF sample to non-athlete normal reference data, this time using BSA-indexed scores. Non-athlete values were much higher than our overall football athlete sample for ARD/BSA ($d=2.18$,

Table 3 Echocardiogram characteristics of collegiate football players indexed to BSA

Echo metric/ BSA (mm/ m ²)	Race			Mean difference (95% CI)	Effect size Cohen's d	Position group		Mean difference (95% CI)	Effect size Cohen's d
	Overall	Black	White			Non-linemen	Linemen		
ARD	13.1 (1.7) (7.6–18.2)	13.0 (1.8) (7.6–18.2)	13.1 (1.6) (8.9–16.9)	0.11 (–0.48 to 0.25)	0.06	13.6 (1.6) (8.4–18.2)	12.3 (1.6) (7.6–16.3)	1.3 (0.97 to 1.66)	0.81*

Sample size (N) for ARD: overall (340), Black (197), White (142), non-linemen (214), linemen (126).

*P<0.001.

ARD, aortic root diameter; BSA, body surface area.

Table 4 ARD reference values by BSA group

Echo measure (mm)	Overall sample 1.72–2.94 m ²	BSA reference group			General population male norms ^{4 13 17}
		Below average 1.72–2.05 m ²	Average 2.06–2.57 m ²	Above average 2.58–2.94 m ²	
ARD					
Normal	<35.6	<32.4	<35.5	<37.9	<38.8
Mildly abnormal	35.6–37.1	32.4–33.5	35.5–36.9	37.9–39.4	38.8–40.0
Moderately abnormal	37.2–38.9	33.6–35.0	37.0–38.7	39.5–41.2	40.1–41.5
Severely abnormal	>38.9	>35.0	>38.7	>41.2	>41.5

Our definitions for BSA reference groups: mildly abnormal: 95–98th percentile or $1.6 < Z < 2.0$; moderately abnormal: 98–99th percentile or $2.0 < Z < 2.5$; severely abnormal: >99th percentile or $Z > 2.5$.

.ARD, aortic root diameter; BSA, body surface area.

large effect), which was consistent across the Below Average ($d=1.52$, large effect), Average ($d=2.17$), and Above Average BSA groups ($d=3.12$, large effect).

It is worth noting that *both* the raw ARD measurement and ARD/BSA index scores were found to be higher in non-athlete normal reference data.

DISCUSSION

We found that for C-ASF players' echocardiogram values at the time of PPE, using the non-athlete reference values¹³ for the upper limit of normal may still be applicable, even though they show a different normal distribution than non-athletes. The ARD distribution for our C-ASF athlete sample was *lower* than for non-athletes which was an unexpected finding. Since the medical concern is with *elevated* ARD values, the non-athlete upper limit of normal reference value (37 mm)¹³ may still be appropriate, while at values above that BSA should be considered in the interpretation.

Aortic root enlargement is of interest in athletes as it is associated with Marfan's syndrome and can lead to aortic dissection and sudden death. The Marfanoid habitus can mimic some beneficial athletic features including tall stature and long arms. Height, weight and specific type of sport participation have been shown to have the greatest impacts on ARD.¹⁸ Our data showed linemen exhibited significantly higher ARD than non-linemen. Previous studies have recommended ≥ 38 mm and ≥ 40 mm as cut-off values for ARD in non-athletes¹³ and athletes,¹⁸ respectively. In our sample of C-ASF athletes at the beginning of their collegiate careers, we found only 10 athletes (2.7%) had an aortic root of ≥ 38 mm and no athletes in this cohort had an aortic root of ≥ 40 mm. Therefore, the non-athlete upper limit of normal reference value >37 mm may be appropriate when interpreting ARD values. There was no evidence of aortic root adaptations to exercise that exceeded predefined norms in this C-ASF sample. A meta-analysis by Iskandar and Thompson demonstrated a weighted mean ARD of 31.6 mm (95% CI 30.2 to 33.1) at the sinus of Valsalva,¹⁹

compared with a diameter of 29.8 mm (95% CI 29.4 to 30.2) in our population.

It is possible that since the timing of our screening is prior to engaging in the intensity of training and conditioning associated with C-ASF, these values do not yet represent the possible adaptive changes specific to collegiate-level athletics. Progressive aortic dilation has been shown in a cohort of strength trained athletes as duration of training increases, with duration of training correlating significantly with aortic root size.⁸ Additionally, most studies evaluating aortic root dimensions are in athletes who are older than our college population.^{7 11 20} It is possible that aortic remodelling may occur as a product of prolonged, intense training in larger individuals. However, Pelliccia *et al* followed several athletes with dilated aortas >40 mm and without stigmata of Marfan's syndrome over several years after conclusion of their athletic careers and found that the largest increases in aortic dimension in athletes tends to occur in midlife as opposed to years with high volumes of training.⁶ A recent study of 3781 young athletes (age: 19 ± 5.9 years) had 5 ± 1.5 years longitudinal follow-up of those athletes with an enlarged aortic diameter and found no progressive enlargement of the aortic diameter during that time.²¹ Their cohort had a *larger* mean ARD compared with controls and did *not* include American-style football athletes.²¹ This discrepancy in findings regarding the aetiology of increased ARD in some athletes presents opportunity for further research to better understand the timing of aortic remodelling and determine if there are clinical consequences to progressive dilation.

Our findings of ARD values at the time of PPE being *lower* than non-athlete controls contrasts with our findings of significantly *larger* left ventricular end diastolic diameter (LVEDD) and interventricular septal diameter (IVSD) in the same population of collegiate football athletes.²² It is possible that the mostly muscular areas of the heart (including LVEDD and IVSD) have non-pathological training adaptations, while areas containing mostly connective tissue do not experience similar adaptations in adolescence.

Table 5 ARD indexed by BSA reference values by BSA group

Echo index measure (mm/m ²)	Overall sample 1.72–2.94 m ²	BSA reference group		General population male norms ^{4 13 17}
		Below average 1.72–2.05 m ²	Above average 2.58–2.94 m ²	
ARD/BSA				
Normal	<15.8	<16.8	<15.6	<20.2
Mildly abnormal	15.8–16.5	16.8–17.5	15.6–16.2	20.2–21.0
Moderately abnormal	16.6–17.3	17.6–18.3	16.3–16.9	21.1–22.0
Severely abnormal	>17.3	>18.3	>16.9	>22.0

Mildly abnormal: 95–98th percentile or $1.6 < Z < 2.0$.

Moderately abnormal: 98–99th percentile or $2.0 < Z < 2.5$.

Severely abnormal: >99th percentile or $Z > 2.5$.

ARD, aortic root diameter; BSA, body surface area.

CONCLUSIONS

The results of this study provide normal reference data for C-ASF players at the time of PPE for ARD at the sinuses of Valsalva. Our results suggest that non-athlete ARD abnormal cut-off of 37 mm may be applicable. Our analysis shows BSA to have the most significant association with ARD values. ARD values in C-ASF athletes approaching upper limits of non-athlete norms should be approached with caution and further evaluated as we did not find larger ARD to be typical in our studied C-ASF population.

Study limitations

Although the study was performed on a large sample of C-ASF athletes, it did not include athletes from races other than blacks and whites and may not be applicable to other races. Additionally, both institutions compete in NCAA Division I Football Bowl Subdivision and our results may not apply to other competition levels. The echocardiograms were obtained at the time of matriculation so do not represent adaptive changes seen from playing collegiate football. Therefore, our results may not apply to different time points in football participation including youth, end of college career or professional. Measurements of ARD were made at the Sinuses of Valsalva, which represents only one segment of potential dilation of the thoracic aorta. However, this site was chosen as it is the location of enlargement used in the diagnosis of Marfan's syndrome²³ which is the primary cause of aortic dilation in this population. The presence of bicuspid aortic valve was also not reported, which can also influence interpretation of aortic root dimensions. The echocardiogram measurements analysed were taken from those interpreted clinically and were not over-read by a single cardiologist for research purposes. Multiple cardiologists contributed to the interpretation of studies; however, three cardiologists read 93% of the echocardiograms. These factors may lead to some inherent variability in the measurements. Other limitations include the lack of a comparison group of non-athletes with similar biometrics to this C-ASF cohort, as well as the study's cross-sectional design which excluded follow-up for those athletes with cardiac variables at the outer edges of distribution, and the use of statistically derived abnormal cut-off values. Longitudinal studies are needed to look at athletes with aortic root dilations to see if their presence correlates with an increased cardiovascular risk over time.

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