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Research paper

Relationship between arm span to height ratio, aortic root diameter, and systolic blood pressure in collegiate athletes

Joshua Altman [a,*,1], Cecil A. Rambarat [b,1], Robert Hamburger [b], Osama Dasa [c], Michelle Dimza [b], Matthew Kelling [b], James R. Clugston [d], Eileen M. Handberg [b], Carl J. Pepine [b], Katherine M. Edenfield [d]

[a] University of Florida College of Medicine, Department of Emergency Medicine, Department of Orthopaedic Surgery and Sports Medicine, Gainesville, FL, United States of America
[b] University of Florida College of Medicine, Department of Medicine, Division of Cardiovascular Medicine, Gainesville, FL, United States of America
[c] University of Florida, College of Public Health and Health Professions, Gainesville, FL, United States of America
[d] University of Florida College of Medicine, Department of Community Health and Family Medicine, Gainesville, FL, United States of America

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ABSTRACT

Study objective: Sudden cardiac death is the most common cause of non-traumatic death in collegiate athletes. Marfan syndrome poses a risk for sudden cardiac death secondary to aortic root dilation leading to aortic dissection or rupture. Arm span to height ratio (ASHR) > 1.05 has been proposed as a screening tool for Marfan syndrome in pre-participation examinations (PPE) for collegiate athletes but limited data exists on the association between ASHR and aortic root diameter (ARD). This study examines the relationship between ASHR and ARD and assesses for predictors of ARD.
Design: Retrospective chart review.
Setting: National Collegiate Athletic Association Division I University.
Participants: 793 athletes across thirteen sports between 2012 and 2022 evaluated with PPE and screening echocardiogram.
Interventions: Not applicable.
Main outcome measures: (1) Relationships between ASHR, SBP, BSA, and ARD amongst all athletes as well as stratified by ASHR >1.05 or ≤1.05 using univariate analysis. (2) Predictors of ARD using multivariate analysis using linear regression.
Results: 143 athletes (18 %) had ASHRs > 1.05. Athletes with ASHR > 1.05 had higher ARD (2.99 cm) than athletes with ASHR ≤ 1.05 (2.85 cm). Weak correlations were noted between ASHR, ARD, and SBP. Multivariate analysis showed that BSA, male sex, and participation in swimming were predictors of ARD. ASHR was not predictive of ARD in regression analysis.
Conclusions: These findings showed a tendency towards higher ARD in athletes with ASHR >1.05 but this observation was not statistically significant in multivariate analysis.

1. Introduction

Sudden cardiac death is a rare but devastating event in athletics and the most common cause of non-traumatic death in collegiate athletes [1]. A risk factor for sudden cardiac death is Marfan syndrome, an autosomal dominant clinical syndrome caused by a mutation in the fibrillin-1 gene, leading to connective tissue disorder. Feared sequelae of Marfan syndrome in both athletes and nonathletes alike is the risk of aortic root dilation, occurring initially at the Sinus of Valsalva, which could lead to aortic dissection or aortic rupture [2]. There is an increased incidence of Marfan syndrome in athletes participating in certain sports, such as basketball or volleyball, due to the favorable advantage of

* Corresponding author at: University of Florida, Department of Emergency Medicine, 1329 SW 16th Street Suite 5270, Gainesville, FL 32610, United States of America.
E-mail address: jaltman7@ufl.edu (J. Altman).
[1] Drs. Altman and Rambarat contributed equally to this work and are first authors.

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increased height and arm span [3]. In an effort to identify athletes at risk for Marfan syndrome, the American Heart Association and the National Collegiate Athletic Association have included family history of Marfan syndrome and physical stigmata of Marfan syndrome in their recommended targeted 14-point medical history [1,4]. This has led to increased use of arm span to height ratio (ASHR) as a screening tool in pre-participation physical examinations, with normal values considered ≤1.05.

While the use of ASHR as one of several clinical screening tools for Marfan Syndrome has been recommended [4,5], there remains limited data on the association between ASHR and aortic root diameter (ARD), especially in competitive athletes. ARD is an important echocardiographic parameter since dilation may predispose to dissection or rupture with disastrous complications and risk of sudden cardiac death. Furthermore, while it has been established that elevated systolic blood pressure (SBP) is associated with increased incidence of aortic root dilation [6] there is limited data describing predictors of increased ARD in a cohort of National Collegiate Athletic Association (NCAA) Division I athletes.

2. Methods

2.1. Study population

A total of 793 NCAA Division I athletes from the University of Florida underwent a pre-participation physical evaluation which included routine personal and family history, American Heart Association 14 element cardiovascular screening recommendations, physical examination including arm span to height ratio (ASHR), electrocardiogram (ECG) and transthoracic echocardiogram (TTE). Athletes were included from the following sports: baseball, basketball, diving, football, golf, gymnastics, lacrosse, soccer, softball, swimming, tennis, track/cross country, and volleyball. De-identified, pre-participation physical evaluation athlete data collected between 2012 and 2022 was obtained from the University of Florida Athletic Association Cardiac Databank. One athlete was medically disqualified due to abnormal ARD found during screening, and none were known to have been excluded during their subsequent careers. Subgroup analyses based on sex and ASHR > 1.05 (abnormal ASHR) were performed.

2.2. Arm span to height ratio

Arm span was measured in inches using measuring tape with athletes standing upright with arms outstretched perpendicular to the body at maximum reach. The distance from the furthest point (usually middle finger) to the furthest point across the sternum (usually the contralateral middle finger) was recorded as the arm span. Arm span was divided by height measured in inches at the time of pre-participation evaluation to calculate the ASHR.

2.3. Echocardiograms

The majority of studies were performed *en masse* by Athletic Heart [7] during routine pre-participation physical evaluation. 784 of the 793 echocardiograms performed were read by one cardiologist (98.9 %) for clinical purposes. Athletes who missed initial screening days had TTEs performed on a GE Vivid E9 echocardiography machine with an M5 cardiac probe at the institution's designated cardiology office.

Echocardiography measurements were performed and calculated based on the American Society of Echocardiography (ASE) recommendations for chamber quantification in adults [8]. Aortic root measurements were performed from a parasternal long-axis view at the Sinuses of Valsalva in the moment of largest dimension using a leading-edge to leading-edge technique at end-diastole [9]. Valvular disease of moderate or greater severity was assessed based on recommendations of the ASE guidelines and standards for the echocardiographic assessment of valve stenosis and native valvular regurgitation [10,11].

2.4. Statistical analysis

Analyses were performed using the IBM SPSS V.28 statistical package (IBM, Armonk, NY). All data were summarized using descriptive statistics. Means and standard deviations were used to describe normally distributed variables while median and interquartile ranges were reported for all non-normally distributed variables. Between-group differences for ASHR ≤ or > 1.05 (abnormal ASHR) were assessed using independent-samples *t*-test for continuous variables and chi-square analysis for categorical variables. Univariate analysis with the Pearson's correlation coefficient test was used to measure the strength of the linear relationship between ASHR, ARD, BSA, and SBP. Multivariate analysis using linear regression was performed to test for correlation of ARD using age, BSA, race, sex, systolic BP, diastolic BP, ASHR >1.05, and sport as independent variables entered in a stepwise method. Scatter diagrams with regression lines were used to present these interrelationships. Statistical significance for main effects was defined a priori as unadjusted $p < 0.05$.

3. Results

A total of 793 athletes (43 % female) participating in 13 sports were included in the study. The median age was 18.6 ± 1.1 years. 29.4 % were Black, 67.2 % were White, 1.9 % Asian, and 1.5 % identified as Other. Baseline pre-participation physical evaluation findings stratified by sex are reported in Table 1. A total of 143 athletes (18 %) had ASHR > 1.05 and 650 athletes (82 %) had ASHR ≤ 1.05. Fig. 1 shows the distribution of athletes with ASHR > 1.05 by sport in males and females respectively. Of sports with >10 participants, only lacrosse had no athletes with ASHR >1.05. Of the female sports, gymnastics (23.3 %) and track/cross country (21.1 %) had the highest proportion of athletes with ASHR >1.05. For male sports, basketball (41.2 %), football (32.4 %), and swimming (25.9 %) had the highest proportion of athletes with ASHR >1.05. Fig. 2 shows the mean distribution of ARD by sport for male and female sports. For female sports, athletes participating in swimming had the highest ARD (2.75 cm ± 0.30), followed by basketball (2.72 cm ± 0.33) and volleyball (2.70 cm ± 0.23). For male sports, athletes participating in swimming were similarly shown to have the highest ARD (3.17 cm ± 0.27), followed by football (3.08 cm ± 0.35), tennis (3.07 cm ± 0.37), and baseball (3.05 cm ± 0.33).

Athletes with ASHR >1.05 were slightly older and had higher height, weight, BMI, BSA, SBP, DBP, and ARD (Table 2). There were increasing proportions of athletes with ASHR >1.05 with worsening degrees of hypertension as shown in Table 3. Of the athletes with normal screening blood pressure, 11.4 % has ASHR >1.05, while 24.1 % of athletes with stage 1 and 33.3 % of athletes with stage 2 hypertension had ASHR >1.05.

Results of univariate analysis revealed a weakly positive correlation between ASHR and ARD ($r = 0.19$, $p < 0.01$), SBP and ARD ($r = 0.31$, $p < 0.01$), and BSA and ARD ($r = 0.55$, $p < 0.01$). In the results of multivariate analysis, BSA ($\beta = 0.33$, $p < 0.01$), male sex ($\beta = 0.35$, $p <$

Table 1
Baseline characteristics of the study population.

	Male (n = 453)	Female (n = 340)	p-Value
Age	18.9 ± 1.1	19.2 ± 1.3	<0.001
Height (cm)	186 ± 8	170 ± 9	<0.001
Weight (kg)	90 ± 22	66 ± 13	<0.001
BMI	26.0 ± 5.2	22.9 ± 3.6	<0.001
BSA	2.1 ± 0.3	1.8 ± 0.2	<0.001
ASHR	1.03 ± 0.03	1.01 ± 0.04	<0.001
SBP (mm Hg)	128 ± 10	118 ± 11	<0.001
DBP (mm Hg)	73 ± 7	72 ± 8	0.005
ARD (cm)	3.06 ± 0.3	2.6 ± 0.3	<0.001

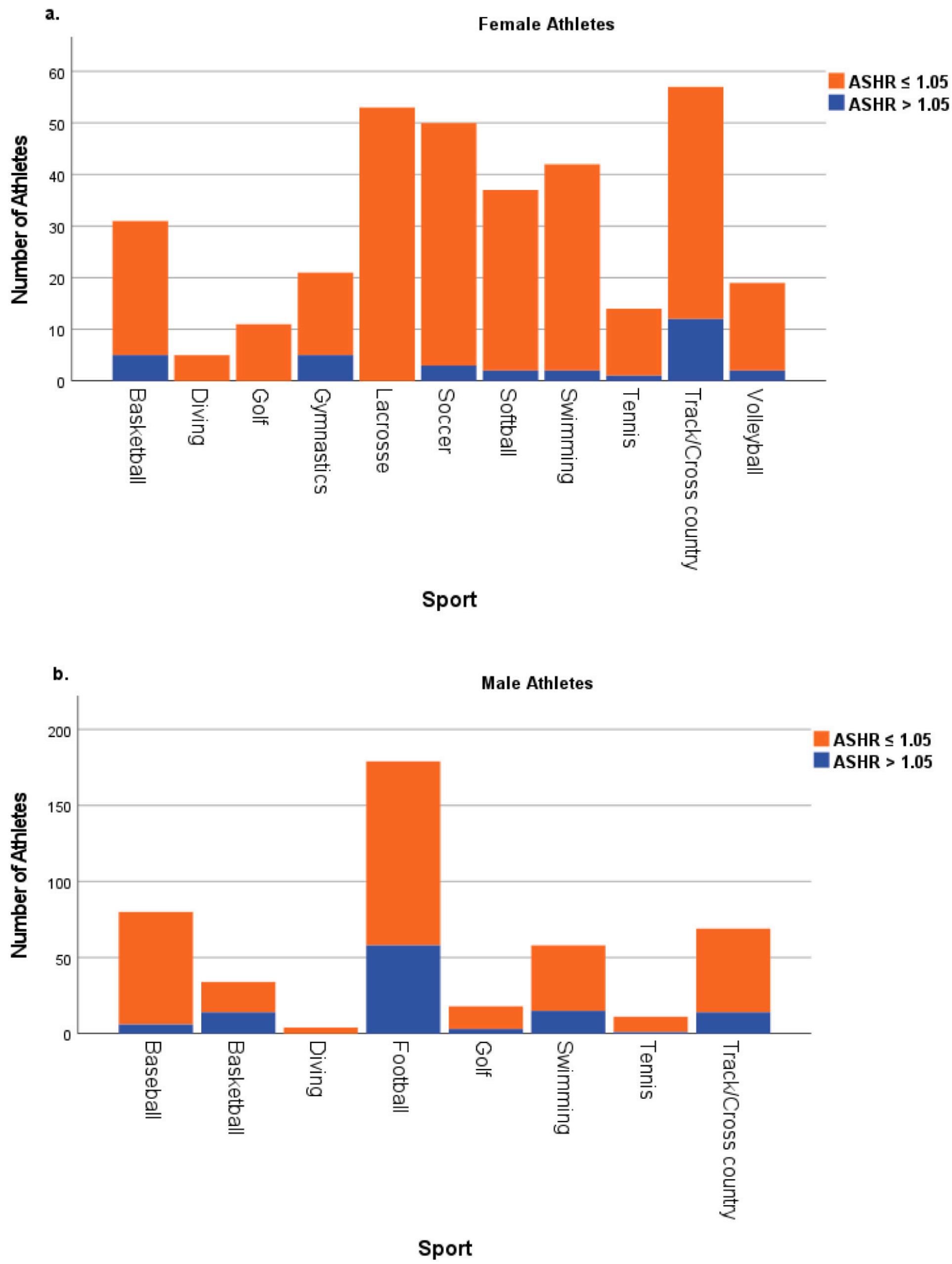

Fig. 1. Arm span to height ratios amongst respective sports by sex.

0.01), and swimming ($\beta = 0.15$, $p < 0.01$) significantly predicted ARD (Table 4). Age, ASHR, SBP, and diastolic blood pressure (DBP) did not significantly predict ARD.

4. Discussion

In many athletic sports, having an increased arm span to height ratio has been viewed as a favorable athletic build, as it offers advantages such as increased athletic prowess and reach [12]. This has been validated in studies with athletes who participate in basketball, volleyball, or boxing [12]. As such, there is a predilection for athletes with increased ASHR in certain sports. In our study, male athletes participating in sports where increased reach can be advantageous such as basketball and football were found to have the highest proportion of increased ASHR. Interestingly amongst female athletes, gymnastics and track/cross country had the highest proportion of increased ASHR despite these not typically being considered as sports where increased reach is advantageous [13].

ASHR has been validated as a screening tool for Marfan Syndrome. In the Ghent criteria for Marfan Syndrome, an ASHR > 1.05 has been noted as characteristic of individuals with a higher likelihood of Marfan Syndrome [5]. The frequency of Marfan Syndrome in the athletic population is not well defined; however, it is likely to be found more frequently in sports that favor increased ASHR, such as basketball and volleyball. The risk of sudden cardiac death in athletes related to Marfan Syndrome is estimated to be around 4–5 % [14,15]. As a result of this, the AHA has recommended that all athletes be screened at the pre-participation physical examination for family history of Marfan Syndrome and physical stigmata of Marfan Syndrome, such as increased ASHR [16].

Currently, however, few studies have specifically examined the relationship between ASHR and aortic root diameter. In a study evaluating the cardiovascular and musculoskeletal assessment of 70 volleyball athletes from the United States national volleyball team, 6 % of athletes had ASHR > 1.05. In the study, 8 % of male athletes and 6 % of

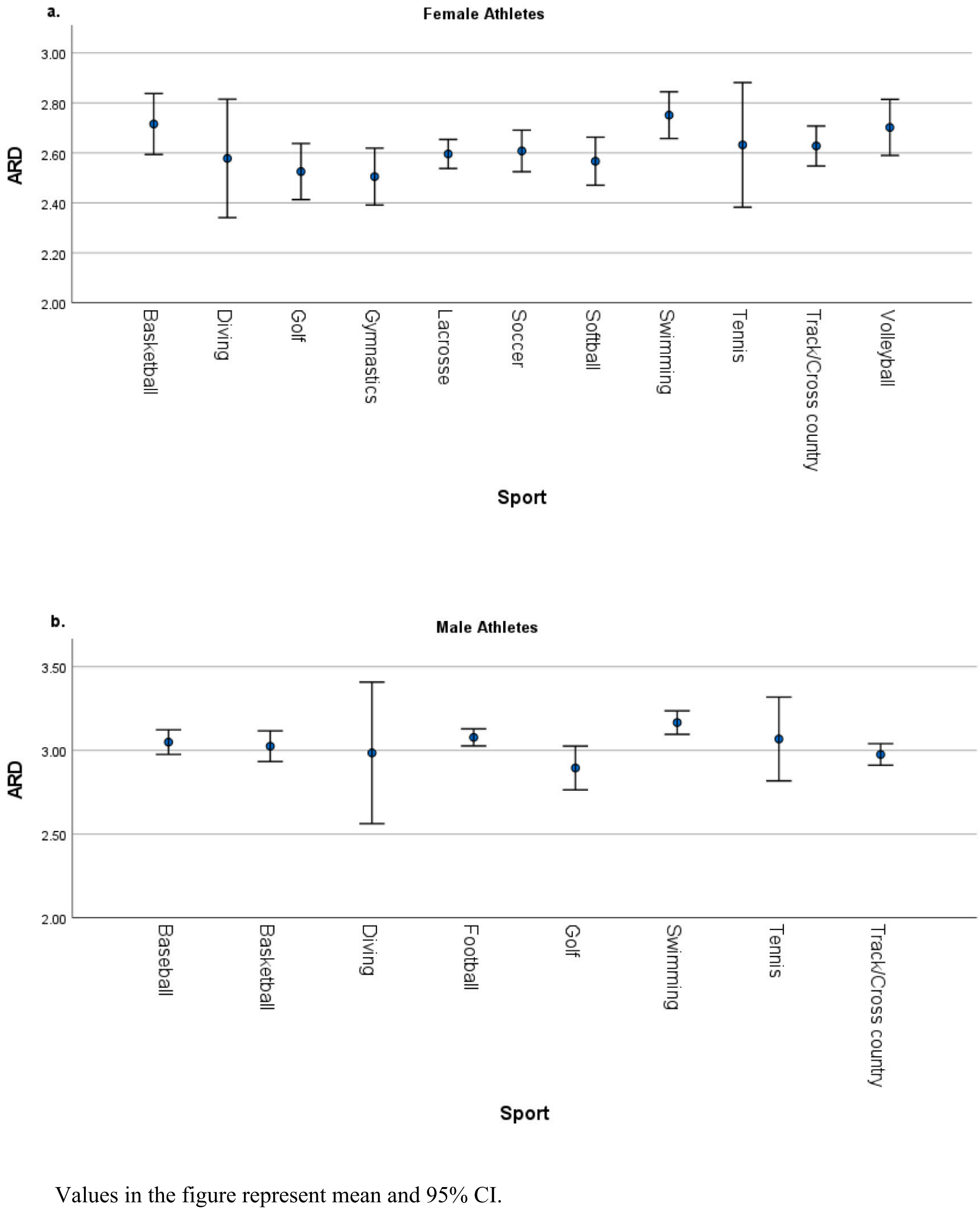

Fig. 2. Mean ARD by sport and sex.
Values in the figure represent mean and 95 % CI.

Table 2
Baseline characteristics of arm span to height ratio groups.

	ASHR ≤ 1.05 (n = 650)	ASHR > 1.05 (n = 143)	p-Value
Age (years)	19 (±1.2)	19.5 (±1.6)	<0.001
Height (cm)	178 (±11)	183 (±11)	<0.001
Weight (kg)	77 (±20)	91 (±26)	<0.001
BMI	24 (±4)	27 (±6)	<0.001
BSA	1.95 (±0.3)	2.14 (±0.3)	<0.001
SBP (mm Hg)	123 (±12)	128 (±11)	<0.001
DBP (mm Hg)	72 (±8)	74 (±8)	0.009
ARD (cm)	2.85 (±0.4)	2.99 (±0.4)	<0.001

Table 3
Presence of hypertension by ASHR.

Screening blood pressure	ASHR ≤ 1.05 n (%)	ASHR > 1.05 n (%)
Normal	256 (88.6)	33 (11.4)
Elevated	184 (83.3)	37 (16.7)
Stage 1	173 (75.9)	55 (24.1)
Stage 2	36 (66.7)	18 (33.3)

Normal = systolic blood pressure <120 mm Hg, diastolic < 80 mm Hg; elevated = systolic 120–129 mm Hg, diastolic <80 mm Hg; Stage 1 = Systolic 130–139 mm Hg, diastolic 80–89 mm Hg; Stage 2 = systolic ≥ 140 mm Hg, diastolic ≥ 90 mm Hg.

Table 4
Standardized linear regression coefficients for notable predictors of ARD.

Predictor	Standardized β-coefficient	p-Value
Sex[a]	0.35	<0.01
Age in years	0.07	0.10
BSA	0.33	<0.01
ASHR	−0.03	0.43
SBP	−0.005	0.88
DBP	0.013	0.17
Sport = Swimming	0.15	<0.01

Abbreviations: ARD, aortic root diameter; ASHR, arm span height ratio; BSA, body surface area; SBP, systolic blood pressure; DBP, diastolic blood pressure.
[a] Male sex compared to female.

female athletes had enlarged aortic root diameter [17]. The study did not go on to evaluate for the relationship between elevated aortic root diameter and ASHR. In another study evaluating the clinical significance of aortic root dilatation in over 2000 highly trained Italian athletes, the aortic root was found to be enlarged (>4.0 cm) in 1.3 % of males and 0.9 % of females (>3.4 cm) [18]. Of the athletes who had elevated aortic root diameter, one had elevated ASHR > 1.05. In our study, when compared as stratified cohorts based on normal or increased ASHR, collegiate athletes with elevated ASHR (ARD = 2.99 cm) were found to have a statistically significant increase in ARD compared to those with normal ASHR (ARD = 2.85 cm) but this difference is likely not of clinical significance. Moreover, although a weak positive correlation was seen amongst all athletes between ASHR and ARD using univariate regression, when accounting for potential confounding variables using a multivariate analysis amongst all athletes, no statistical association was found between ASHR and ARD. This suggests that increased ASHR as an isolated variable is not a reliable predictor of ARD.

When evaluating aortic root diameter, another possible contributing factor to aortic root dilation which should be considered is systolic blood pressure. Elevated systolic blood pressure has been previously shown to be possibly associated with increased aortic root diameter [6,19]. Mechanisms include exposure to repeated cyclic stretching, resulting in accelerated elastin breakdown and thinning of aortic media [1]. Similar to the relationship between ASHR and ARD, there is limited data detailing the relationship between systolic blood pressure and aortic root diameter in athletes. Our study also found a weak positive correlation between SBP and ARD with univariate correlation analysis which again was not observed to be statistically significant using a multivariate analysis.

Several studies have also demonstrated associations between anthropometric variables and ARD. Measurements such as height and BSA have previously been shown to have linear correlations and be reliable predictors of ARD [20] up to extremes of body size at which point aortic root diameter tends to plateau [21]. Published equations have been proposed to help predict the expected range of normal ARD measurements across different populations stratified by age and BSA [8]. Additional recent studies have shown that use of allometric scaling to account for the influence that body size has on aortic root remodeling very reliably predicts changes in aortic root diameter amongst competitive athletes [22]. In our study we were also able to show a relationship between BSA and aortic root diameter in both univariate and multivariate analysis.

Sport specific cardiovascular demand has also been shown to play a role in aortic root remodeling. Data derived from one large cohort of athletes previously showed that endurance sports that are the most hemodynamically intense (such as cycling, swimming, water polo, and basketball) are associated with mild but statistically significant increases in aortic root remodeling [23]. Additionally, power sports tend to be associated with increased systemic blood pressure [24] which has also been proposed to lead to aortic root remodeling in response to hemodynamic stress from increased cardiac output required to meet oxygen demand [23]. There is, however, conflicting data with regards to the effects of power sports on aortic remodeling with more recent studies showing trivial to no statistically significant correlation [23]. It has been proposed that aortic remodeling may be more dependent on the combination of prolonged volume and pressure overload associated with endurance training than the intermittent pressure overload associated with strength training [23]. Additional research in this area is likely needed to further elucidate the relationships of sport specific effects on aortic root remodeling and underlying physiologic mechanisms. In this study we found that swimming predicted increased ARD but did not find a statistically significant predictive value of other high endurance sports.

No athletes in this study were ultimately diagnosed with Marfan syndrome, however one athlete was disqualified due to abnormal ARD after extensive workup and consultation. The weak statistically significant univariate association we identified between ASHR and ARD was not observed using a multivariate analysis. While increased ASHR may reflect underlying athlete connective tissue properties, in the absence of other underlying risk factors or clinical features of Marfan syndrome, this does not appear to hold clinical value in predicting athletes with increased ARD. ASHR as a screening tool in American collegiate athletes on pre-participation physical examination should prompt further consideration for additional features of Marfan syndrome or other connective tissue disease, but in isolation does not appear to support further screening for ARD based on the results of this study. Since pre-participation TTE is not feasible on every collegiate athlete due to associated costs, predictors for aortic root dilation such sex, BSA and specific sport in addition to known or identified cardiovascular or connective tissue risk factors on PPE should be examined on an individual basis and used in decision making to screen for athletes who may have elevated ARD. These simple steps taken can potentially lead to the prevention of catastrophic outcomes. A review of the available literature also suggests that if elevated ARD is discovered on screening echocardiograms, closer follow-up of these athletes with subsequent echocardiograms during and post-athletic careers is valuable, as prior studies have noted a continued increase in aortic root diameter [17].

4.1. Limitations

Although this study included a relatively large cohort of athletes compared to similar studies, the number of athletes in specific sports may have limited detection of statistically significant differences. Small numbers of athletes in certain sport specific subgroups may be statistically underpowered for detecting predictive value of sport specific factors on ARD that may exist. Additionally, it is worth noting that these athletes were studied at the beginning of their college careers with the potential for extreme variability in training up to that point. Therefore, cardiac remodeling, including change in aortic root diameter that might be associated with the training of a collegiate career, had not yet occurred. At the time of this manuscript, we did not have long-term follow-up data on athletes.

5. Conclusion

Collegiate athletes with ASHR > 1.05 had a small statistically significant increase in average aortic root diameter compared with athletes with ASHR ≤ 1.05. There was a weak positive correlation between ARD and both ASHR and SBP that was not observed in a multivariate analysis. Predictors of increased ARD in collegiate athletes included sex, BSA and swimming. These findings suggest that increased ASHR and SBP in isolation measured during PPE may not have significant utility in predicting increased ARD in the absence of other predictors or risk factors and should be used in conjunction with other clinical factors to determine need for further echocardiographic screening.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.lfs.2021.120125.

References

[1] K.G. Harmon, I.M. Asif, J.J. Maleszewski, et al., Incidence, cause, and comparative frequency of sudden cardiac death in National Collegiate Athletic Association Athletes: a decade in review, Circulation 132 (1) (Jul 2015) 10–19.
[2] D.M. Milewicz, H.C. Dietz, D.C. Miller, Treatment of aortic disease in patients with marfan syndrome, Circulation 111 (11) (2005) e150–e157.
[3] N. Kinoshita, J. Mimura, C. Obayashi, F. Katsukawa, S. Onishi, H. Yamazaki, Aortic root dilatation among young competitive athletes: echocardiographic screening of 1929 athletes between 15 and 34 years of age, Am. Heart J. 139 (4) (Apr 2000) 723–728.
[4] B. Hainline, J.A. Drezner, A. Baggish, et al., Interassociation consensus statement on cardiovascular Care of College Student-Athletes, J. Am. Coll. Cardiol. 67 (25) (2016) 2981–2995.
[5] B. Loeys, H. Dietz, A. Braverman, B. Callewaert, J. De Backer, R. Devereux, Y. Hilhorst-Hofstee, G. Jondeau, L. Faivre, D. Milewicz, R. Pyeritz, P. Sponseller, P. Wordsworth, A. De Paepe, The revised Ghent nosology for the Marfan syndrome, J. Med. Genet. 47 (7) (July 2010) 476–485.
[6] M. Kim, M.J. Roman, M.C. Cavallini, J.E. Schwartz, T.G. Pickering, R.B. Devereux, Effect of hypertension on aortic root size and prevalence of aortic regurgitation, Hypertension 28 (1) (Jul 1996) 47–52.
[7] A. Heart. https://athletic-heart.com/, 2020 https://athletic-heart.com/.
[8] R.M. Lang, L.P. Badano, V. Mor-Avi, et al., Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the european Association of Cardiovascular Imaging, J. Am. Soc. Echocardiogr. 28 (1) (Jan 2015) 1–39.e14.
[9] C. Mitchell, P.S. Rahko, L.A. Blauwet, et al., Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American Society of Echocardiography, J. Am. Soc. Echocardiogr. 32 (1) (Jan 2019) 1–64.
[10] H. Baumgartner, J. Hung, J. Bermejo, et al., Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice, J. Am. Soc. Echocardiogr. 22 (1) (Jan 2009) 1–23, quiz 101–102.
[11] W.A. Zoghbi, D. Adams, R.O. Bonow, et al., Recommendations for noninvasive evaluation of native valvular regurgitation: a report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance, J. Am. Soc. Echocardiogr. 30 (4) (Apr 2017) 303–371.
[12] T. Monson, M. Brasil, L. Hlusko, Allometric variation in modern humans and the relationship between body proportions and elite athletic success, J. Anthropol. Sport Phys. Educ. 3 (2018) 3–8.
[13] K.T.L.J. Keskinen, P. Komi, Maximum velocity swimming: interrelationships of stroking characteristics, force production, and anthropometric variables, Scand. J. Sports Sci. 11 (2) (1989) 87–92.
[14] F.M. McCaffrey, D.S. Braden, W.B. Strong, Sudden cardiac death in young athletes. A review, Am. J. Dis. Child. 145 (2) (Feb 1991) 177–183.
[15] B.J. Maron, J. Shirani, L.C. Poliac, R. Mathenge, W.C. Roberts, F.O. Mueller, Sudden death in young competitive athletes. Clinical, demographic, and pathological profiles, JAMA 276 (3) (1996) 199–204.
[16] B.J. Maron, P.D. Thompson, M.J. Ackerman, et al., Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on nutrition, physical activity, and metabolism: endorsed by the American College of Cardiology Foundation, Circulation 115 (12) (2007), 1643-1455.
[17] C.K. Davis, D.A. Dyar, L.A. Vargas, P.D. Grossfeld, Cardiovascular and musculoskeletal assessment of elite US volleyball players, Clin. J. Sport Med. 25 (6) (Nov 2015) 546–550.
[18] A. Pelliccia, F.M. Di Paolo, E. De Blasiis, et al., Prevalence and clinical significance of aortic root dilation in highly trained competitive athletes, Circulation 122 (7) (Aug 2010) 698–706.
[19] S.M. Farasat, C.H. Morrell, A. Scuteri, et al., Do hypertensive individuals have enlarged aortic root diameters? Insights from studying the various subtypes of hypertension, Am. J. Hypertens. 21 (5) (May 2008) 558–563.
[20] E. Yim, Aortic root disease in athletes: aortic root dilation, anomalous coronary artery, bicuspid aortic valve, and Marfan's syndrome, Sports Med. 43 (2013) 721–732.
[21] A. Baggish, R. Battle, T. Beaver, W. Border, P. Douglas, C. Kramer, M. Martinez, J. Mercandetti, D. Phelan, T. Singh, R. Weiner, E. Williamson, Recommendations on the use of multimodality cardiovascular imaging in young adult competitive athletes: a report from the American Society of Echocardiography in collaboration with the Society of Cardiovascular Computed Tomography and the Society for Cardiovascular Magnetic Resonance, J. Am. Soc. Echocardiogr. 33 (5) (May 2020) 523–549.
[22] M. Abuli, G. Grazioli, M. Sanz de la Garza, S. Montserrat, B. Vidal, A. Doltra, G. Sarquella-Brugada, M. Bellver, R. Pi, D. Brotons, D. Oxborough, M. Sitges, Aortic root remodeling in competitive athletes, Eur. J. Prev. Cardiol. 27 (14) (Sep 2020) 1518–1526.
[23] A. Pelliccia, F. Di Paolo, F. Quattrini, Aortic root dilatation in athletic population, Prog. Cardiovasc. Dis. 54 (5) (March 2012) 432–437.
[24] V. Schweiger, D. Niederseer, C. Schmied, C. Attenhofer-Jost, S. Caselli, Athletes and hypertension, Curr. Cardiol. Rep. 23 (12) (Oct 2021) 176.