Original Investigation

Athletic Cardiac Remodeling in US Professional Basketball Players

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IMPORTANCE The incidence of sudden cardiac death is higher in US basketball players compared with other athlete groups. However, the recognition of the risk for sudden cardiac death among basketball players is challenging because little is known regarding athletic cardiac remodeling in these athletes or athletes of similarly increased size.

OBJECTIVE To perform a comprehensive cardiac structural analysis of National Basketball Association (NBA) professional athletes.

DESIGN, SETTING, AND PARTICIPANTS Echocardiographic observational study of NBA players on the active rosters for the 2013-2014 and 2014-2015 seasons was performed from December 16, 2013, to December 12, 2014. The policy of the NBA mandates annual preseason stress echocardiograms for each player. The NBA has sanctioned Columbia University Medical Center to conduct annual health and safety reviews of these echocardiograms. Data were analyzed from January to May 2015.

MAIN OUTCOMES AND MEASURES Cardiac variables assessed included left ventricular (LV) size, mass, wall thickness, and hypertrophy patterns and function; left atrial volume; and aortic root diameter. All dimensions were biometrically scaled.

RESULTS Of the 526 athletes included in the study, 406 (77.2%) were African American and 107 (20.3%) were white, with a mean (SD) age of 25.7 (4.3) years. Mean (SD) athlete height was 200.2 (8.8) cm; mean body surface area, 2.38 (0.19) m^2. Left ventricular size and mass in NBA athletes were proportional to body size, extending to the uppermost biometrics of the cohort. Left ventricular hypertrophy was present in 144 athletes (27.4%). African American athletes had increased LV wall thickness (unadjusted mean, 11.2 mm; 95% CI, 11.1-11.3 mm) and LV mass (unadjusted mean, 106.3 g/m^2; 95% CI, 104.6-108.0 g/m^2) compared with LV wall thickness (unadjusted mean, 10.5 mm; 95% CI, 10.3-10.7 mm; $P < .001$) and LV mass (unadjusted mean, 102.2 g/m^2; 95% CI, 99.0-105.4 g/m^2; $P = .029$) in white athletes. The maximal aortic root diameter in the cohort was 42 mm. Aortic root diameters reached a plateau at the uppermost biometric variables. Five athletes (1.0%) had an LV ejection fraction of less than 50%, and all ventricles augmented normally with exercise.

CONCLUSIONS AND RELEVANCE This study provides normative cardiac data for a group of athletes with greater anthropometry than any previously studied athlete group and for a group known to have elevated rates of sudden cardiac death. These data can be incorporated into clinical assessments for the primary prevention of cardiac emergencies in basketball players and the athletic community at large.

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The characterization of the athlete's heart, defined by the development of increased left ventricular (LV) end-diastolic diameter (LVEDD), LV wall thickness (LVWT), and LV mass (LVM),[1-3] is based on data collected from a pooled analysis of athletes engaged in multiple sports. Athletes from the United States are vastly underrepresented in published studies. The most popular sports within the United States and the demographics of US athletes are different from those of the European centers where most of the data on the athlete's heart have been collected. Basketball is a leading sport within the United States, with numerous athletes competing from the youth to the professional levels. A detailed understanding of normal and expected cardiac remodeling in US basketball players has significant clinical importance given that the incidence of sports-related sudden cardiac death (SCD) in the United States is highest among basketball players and that the most common cause of SCD in this population is hypertrophic cardiomyopathy (HCM).[4-7]

A recent study suggested that the incidence of SCD in National Collegiate Athletic Association Division 1 male basketball players is as high as 1:5200 per year.[8] The "gray zone" that differentiates the upper limit of LV hypertrophy (LVH) related to athletic remodeling and pathologic hypertrophy due to HCM varies depending on the reference sport and is incompletely defined in basketball players in particular.[9-12] Sudden cardiac death in athletes is not limited to HCM; for basketball players, for whom tall stature is advantageous, Marfan syndrome is another systemic disorder linked with SCD in athletes for whom the differentiation between normal athletic remodeling and pathologic change is of crucial importance. Aortic root sizes have been shown to be modestly dilated in athletes across several sports; in addition, aortic root size has a correlation with height, creating another gray zone in the cardiac evaluation of basketball players.[13-16] The level of uncertainty regarding reference values for all cardiac dimensions is high for elite US basketball players because, to our knowledge, no large-scale studies have provided comparative data for athletes of this size.

We completed a comprehensive echocardiographic analysis of 526 current professional athletes from the National Basketball Association (NBA). A detailed and systematic review of cardiac structure and function in this large group of athletes will provide an invaluable frame of reference to enhance player safety for the large group of US basketball players in training at all skill levels and in the athletic community at large.

Methods

Study Population

The policy of the NBA mandates that each athlete undergo an annual preseason stress echocardiogram as part of a preseason medical evaluation, in addition to the screening stress echocardiogram performed at entrance into the league. The NBA has sanctioned the Division of Cardiology at Columbia University Medical Center to conduct a player health and safety review of these echocardiograms on an annual basis. The 2013 and 2014 preseason echocardiograms performed by NBA team–affiliated physicians and the echocardiograms performed at the

2014 NBA predraft evaluation (Combine) were sent to Columbia University Medical Center for core laboratory analysis in digital format via a league-wide electronic medical records system. The 526 athletes included in this study represent the total number of athletes with echocardiograms available for review. Subgroup analysis by race/ethnicity was performed and confined to African American and white athletes for the purpose of complementing the existing and well-established data characterizing electrocardiographic differences in African American and white athletes[17-19] with echocardiographic data. Hispanic and Asian athletes were not included in the subgroup analyses given the small number of representative athletes (13 [2.5%]). The race/ethnicity of the athletes was determined by the investigators. The review and analysis of these echocardiograms for this study was approved by the NBA, the NBA Players Association, and the institutional review board of Columbia University Medical Center. Pursuant to approval from the institutional review board of Columbia University Medical Center, informed consent was not required for this study.

Echocardiograms

Data were collected from December 16, 2013, to December 12, 2014. Transthoracic echocardiograms were performed using commercially available systems. Interventricular septum and posterior wall thickness, LVEDD, left atrial (LA) anteroposterior diameter, and aortic root diameter at the sinus of Valsalva were measured from a parasternal long-axis view as per American Society of Echocardiography recommendations.[20] Left atrial volumes were calculated using the biplane area-length method and indexed for body surface area (BSA) (calculated as the square root of [height in centimeters × weight in kilograms]/3600). The LV ejection fraction (LVEF) was calculated using the biplane modified Simpson rule,[21] replaced by visual estimation for technically suboptimal images. The LV diastolic function was assessed using pulsed-wave Doppler sampling at the tips of the mitral leaflets in diastole (peak early velocity E) and tissue Doppler imaging of the lateral mitral anulus (peak early velocity E'). We calculated LVM with a validated method[20] and indexed LVM by BSA. We calculated LV relative wall thickness as (2 × posterior wall thickness)/LVEDD. Hypertrophy patterns were calculated and defined using established echocardiographic methods to measure LV volumes and concentricity.[22,23]

Reproducibility

A single reader (D.J.E.) analyzed all echocardiograms in this study. Reproducibility was assessed using 20 studies in which core dimensions were remeasured, and intraclass correlation coefficients were calculated for each measure.

Statistical Analysis

Data were analyzed from January 5 to May 9, 2015. Data are presented as means with 95% CIs or with SDs in the reporting of baseline characteristics. Differences between the mean baseline characteristics in African American and white athletes were assessed using the 2-tailed t test. For differences in LVH, the 2-tailed t test was used for the comparison of unadjusted means, and a linear regression model was used to calculate the adjusted means and their SEs after adjustment for age, BSA,

Table 1. General Characteristics of the Study Population

| Characteristic | All, Mean (SD) [Range] (N = 526) | Athlete Group, Mean (SD) | | P Value |
		African American (n = 406)	White (n = 107)	
Age, y	25.7 (4.3) [18-39]	25.4 (4.3)	26.3 (4.3)	.06
Height, cm	200.2 (8.8) [175-218]	198.9 (8.4)	205.0 (8.3)	<.001
Weight, kg	101.1 (12.3) [72.6-142.9]	100.0 (12.1)	105.5 (12.1)	<.001
BSA, m²ᵃ	2.38 (0.19) [1.94-2.85]	2.36 (0.18)	2.46 (0.18)	<.001
Heart rate, bpm	56 (10) [36-91]	56 (10)	57 (10)	.29
SBP, mm Hg	119.2 (12.0) [90-166]	119.2 (11.4)	119.7 (14.0)	.69
DBP, mm Hg	72.8 (8.6) [44-86]	73.0 (8.6)	71.5 (8.5)	.11

Abbreviations: BSA, body surface area; DBP, diastolic blood pressure; SBP, systolic blood pressure.

ᵃ Calculated as the square root of (height in centimeters × weight in kilograms)/3600.

Figure 1. Left Ventricular (LV) End-Diastolic Diameter (LVEDD) in National Basketball Association Athletes
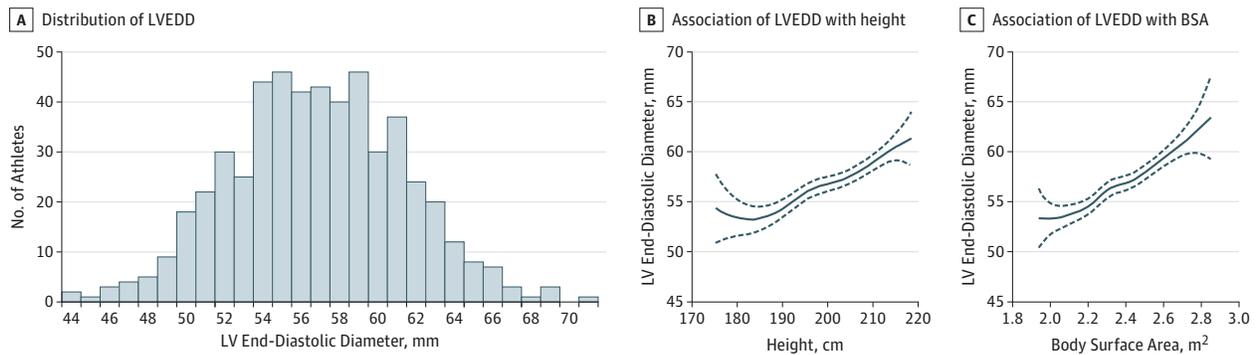
Results are shown for all 526 athletes included in the study. BSA indicates body surface area (calculated as the square root of [height in centimeters × weight in kilograms]/3600); solid lines, association of the 2 characteristics (B and C); and dotted lines, 95% CIs.

and blood pressure. The Fisher exact test was used in the analysis of hypertrophy patterns between African American and white athletes. The association between cardiac dimensions, height, and BSA was assessed by grouping means and generalized additive models using the gam function with 6 *df* and cubic splines (https://cran.r-project.org/web/packages/gam/gam.pdf). We generated fitted curves with 95% point-wise CIs. For all statistical analysis, a 2-tailed *P* value of less than 0.05 was considered significant. The analysis was performed in SAS (version 9.3; SAS Institute, Inc) and R (version 3.0.1; https://cran.r-project.org) software.

Results

Characterization of Study Population

Baseline characteristics of the study population are shown in **Table 1**. The 526 athletes ranged in age from 18 to 39 years (mean age, 25.7 [4.3] years); 406 (77.2%) were African American, 107 (20.3%) were white, 12 (2.3%) were Hispanic, and 1 (0.2%) was Asian. The mean height of the group was 200.2 (8.8) cm; mean weight, 101.1 (12.3) kg; and mean BSA, 2.38 (0.19) m². Blood pressures were normal in the athlete group. White athletes had increased BSA compared with African American athletes. We found no differences in age, heart rate, or blood pressure between African American and white athletes.
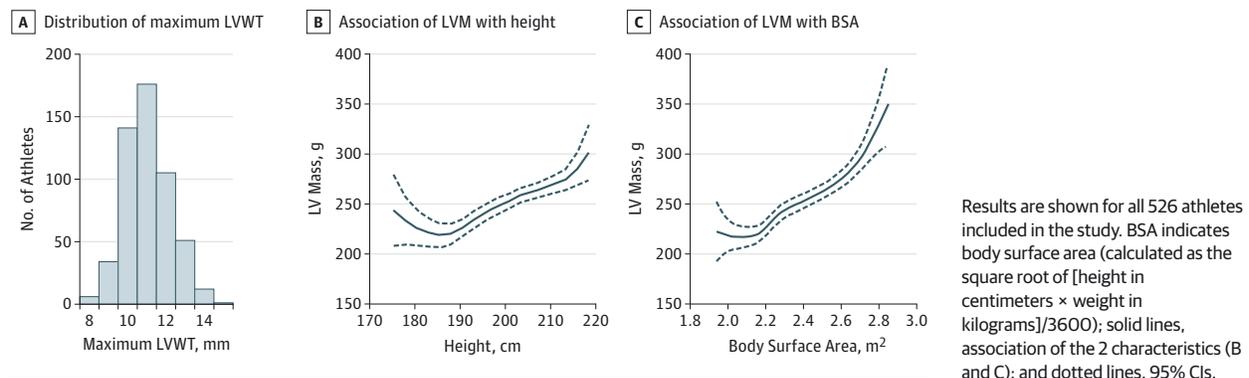
Cardiac Dimensions

LV Size and LVM

The distribution for LVEDD is shown in **Figure 1**A. The LVEDD ranged from 44 to 71 (mean, 56.8; 95% CI, 56.4-57.2) mm. Left ventricular cavity dilation (LVEDD, ≥59 mm)[20] was present in 192 athletes (36.5%), whereas 23 (4.4%) had substantial LV dilation (LVEDD, ≥65 mm). Although a significant proportion of NBA athletes had LV dilation, when LVEDD was indexed to height and BSA, LV cavity sizes were normal compared with reference male adults, even among the largest athletes,[20] with a mean LVEDD per unit of BSA of 2.40 (95% CI, 2.23-2.57) cm/m² vs reference adults (range, 2.3-3.0 cm/m²) and a mean LVEDD per unit of height of 2.84 (95% CI, 2.66-3.02) cm/m vs reference adults (range, 2.4-3.3 cm/m). We observed an approximately linear association between LVEDD and height and BSA that extended to the uppermost biometrics of the athlete group, demonstrated by the curves with 95% CIs in Figure 1B and C. Raw data for the distribution of LVEDD in the NBA cohort and mean LVEDDs associated with the span of height and BSA in the group is provided in eFigure 1 in the Supplement.

The distribution for maximal LVWT is shown in **Figure 2**A. The LVWT ranged from 8 to 15 (mean, 11.04; 95% CI, 10.94-11.14) mm, with 64 athletes (12.2%) having an LVWT of 13 mm or greater and 13 (2.5%) having an LVWT of 14 mm or greater. As we observed with LV cavity size, LVM was associated with height and BSA in an approximately linear fashion extending to the upper-

Figure 2. Left Ventricular Wall Thickness (LVWT) and Mass (LVM) in National Basketball Association Athletes

Results are shown for all 526 athletes included in the study. BSA indicates body surface area (calculated as the square root of [height in centimeters × weight in kilograms]/3600); solid lines, association of the 2 characteristics (B and C); and dotted lines, 95% CIs.

most biometrics of the group, as demonstrated by the curves with 95% CIs in Figure 2B and C. Raw data showing the mean LVMs associated with the span of height and BSA in the group are provided in eFigure 2 in the Supplement. The mean LVM index (LVMI) was 105.4 (95% CI, 103.9-106.9) g/m^2, and the mean relative wall thickness was 0.38 (95% CI, 0.37-0.39). Using the American Society of Echocardiography standards for LVH,[20] 144 athletes (27.4%) had LVH (LVMI, >115 g/m^2), whereas 29 (5.5%) had moderate LVH (LVMI, >132 g/m^2) and 9 (1.7%) had severe LVH (LVMI, ≥149 g/m^2). We further characterized the pattern of hypertrophy in these athletes using a 4-group classification[22,23] that incorporates LV volume and concentricity. Of the 134 African American and white athletes with LVH, the most predominant pattern of hypertrophy was concentric nondilated (64 [47.8%]), followed by eccentric dilated (37 [27.6%]), concentric dilated (26 [19.4%]), and eccentric nondilated (7 [5.2%]). We found significant increases in LVWT, LVMI, and relative wall thickness in African American compared with white athletes independently of age, BSA, and blood pressure. In addition, we observed a variation in the pattern of LVH between African American and white athletes. In those athletes with LVH, the proportion of concentric nondilated hypertrophy was substantially higher in African American athletes, whereas the eccentric dilated form of hypertrophy was more predominant in white athletes. These findings are demonstrated in **Table 2**.

LA Size

Left atrial diameter ranged from 26 to 49 (mean, 38.1; 95% CI, 37.8-38.4) mm. The mean LA volume index (LAVI) was 30.9 (95% CI, 30.4-31.4) mL/m^2. Left atrial enlargement in this athlete cohort was mild in 85 athletes (16.2%; LAVI, 35-41 mL/m^2), moderate in 23 (4.4%; LAVI, 42-48 mL/m^2), and severe in 7 (1.3%; LAVI, >48 mL/m^2).[20] In those athletes with increased LA volume, a corresponding increase in LVM compared with the athletes who had normal LA volumes was noted (LVMI, 110.4 [95% CI, 106.9-113.9] g/m^2 and 102.4 [95% CI, 100.5-104.3] g/m^2, respectively; P < .001). Left atrial volumes were similar in athletes with concentric and eccentric forms of LVH (LAVI, 33.7 [95% CI, 32.5-35.9] mL/m^2 and 35.3 [95% CI, 32.9-37.7] mL/m^2, respectively; P = .27).

Aortic Root Dimension

The distribution for aortic root diameter is shown in **Figure 3**A. Aortic root diameter ranged from 25 to 42 (mean, 33.7; 95% CI,

Table 2. Comparison of LVH in African American and White Athletes

| | Athlete Group | | |
	African American (n = 406)	White (n = 107)	P Value
Maximum mean LVWT, mm			
Unadjusted (95% CI)	11.2 (11.1-11.3)	10.5 (10.3-10.7)	<.001
Adjusted (95% CI)[a]	11.2 (11.1-11.4)	10.4 (10.2-10.6)	
Mean LVMI, g/m^2			
Unadjusted (95% CI)	106.3 (104.6-108.0)	102.2 (99.0-105.4)	.03
Adjusted (95% CI)[a]	106.5 (104.8-108.2)	101.7 (98.4-105.0)	
Mean RWT			
Unadjusted (95% CI)	0.39 (0.38-0.40)	0.35 (0.34-0.36)	<.001
Adjusted (95% CI)[a]	0.39 (0.38-0.40)	0.35 (0.34-0.36)	
LVH, No. (%)[b]			
Concentric nondilated	60 (53.1)	4 (19.0)	.004
Eccentric nondilated	7 (6.2)	0	.60
Concentric dilated	19 (16.8)	7 (33.3)	.13
Eccentric dilated	27 (23.9)	10 (47.6)	.03

Abbreviations: LVH, left ventricular (LV) hypertrophy; LVMI, LV mass index; LVWT, LV wall thickness; RWT, relative wall thickness.

[a] Linear regression was used to calculate adjusted means after adjustment for age, body surface area, and systolic and diastolic blood pressure.

[b] Pattern of hypertrophy is shown as percentages of African American and white athletes with subtypes of hypertrophy.

33.4-34.0) mm, with 24 athletes (4.6%) having an aortic root diameter of 40 mm or greater and 7 (1.3%) having the maximum measured aortic root diameter of 42 mm. Figure 3B and C show the curves with 95% CIs of aortic root diameters in the NBA athletes associated with height and BSA. Raw data for the distribution of aortic root diameter in the NBA cohort and means of aortic root diameter in relation to the span of height and BSA in the group is provided in eFigure 3 in the Supplement. We observed that at the uppermost ranges in body size, aortic root diameters reached a plateau. This finding is in contrast to our observations of LVM and cavity size, where these cardiac dimensions continued to rise proportionally as the athletes' biometrics increased.

We found no difference in age or blood pressure between athletes with the largest aortic root diameters (≥40 mm) and

Figure 3. Aortic Root Diameters at the Sinuses of Valsalva in National Basketball Association Athletes
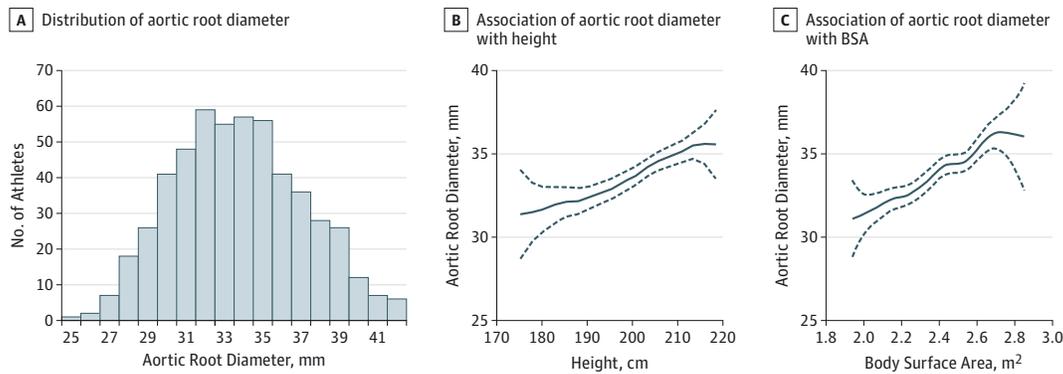
Results are shown for all 526 athletes included in the study. BSA indicates body surface area (calculated as the square root of [height in centimeters × weight in kilograms]/3600); solid lines, association of the 2 characteristics (B and C); and dotted lines, 95% CIs.

athletes with smaller aortic roots. Aortic root diameter indexed to the height and BSA in athletes with aortic roots of 40 mm or greater remained significantly higher in comparison with those of the other athletes (aortic root per unit of height, 0.197 [95% CI, 0.195-0.200] mm/cm and 0.167 [95% CI, 0.165-0.168] mm/cm, respectively; $P < .001$). Aortic root per unit of BSA was 16.1 (95% CI, 15.6-16.6) mm/m² for athletes with aortic roots of 40 mm or greater vs 14.1 (95% CI, 14.0-14.2) mm/m² for those of other athletes ($P < .001$), suggesting that body size was not the determining factor to explain the increased aortic root diameter in these athletes with the largest aortic roots. We also observed a higher proportion of mitral valve prolapse in the athletes with aortic root diameters of 40 mm or more (4 of 24 [17%]) compared with the athletes with smaller aortic roots (20 of 502 [4.0%]).

Left Ventricular Function

The mean LVEF (measured by the biplane modified Simpson rule, 81%; by visual estimation, 19%) was 59.6% (95% CI, 59.3%-59.9%) for this athlete group (range, 45%-72%). We found that 5 of the 526 athletes (1.0%) had an LVEF of less than 50%. All ventricles were observed by stress echocardiography to augment normally with exercise. Stroke volume was calculated in 247 of 526 athletes (47.0%), and the mean stroke volume was 111.3 (95% CI, 108.3-114.3) mL, which was increased in the NBA cohort compared with the mean in normal male individuals (73 [17] mL).[20] We observed that athletes with the lowest LVEFs (<50%) did not differ compared with the other athletes with respect to height, BSA, age, blood pressure, or heart rate. We observed that athletes with an LVEF of less than 50% had larger LVEDDs at 62.4 (95% CI, 59.8-65.0) mm vs 56.8 (95% CI, 56.4-57.2) mm ($P = .005$) and increased LVMI at 121.0 (95% CI, 98.7-143.3) g/m² vs 105.2 (95% CI, 103.8-106.7) g/m² ($P = .04$) than athletes with LVEF of greater than 50%.

Left ventricular diastolic function using pulsed-wave and tissue Doppler analysis was measured in 213 of 526 athletes (40.5%) and was normal in all participants (E/E′, 4.8; 95% CI, 4.6-5.0). No difference in diastolic function was observed between those athletes with increased LVM or increased LA volume vs those athletes with normal indices.

Reproducibility

For intraobserver variability, the intraclass correlation coefficients were 0.99 for LVEDD and 0.98 for aortic root diameter. The mean intraobserver difference was 0.6 mm for LVEDD and 0.7 mm for aortic root diameter.

Discussion

The distinction in an individual athlete between expected athletic cardiac remodeling and cardiac pathologic change that predisposes to SCD can be challenging, given the wide range of hemodynamic demands involved in training and competition in different sports and the varied baseline characteristics of athletes engaged in these sports. Because much of the literature on the structural features of the athlete's heart is derived from European centers where comprehensive cardiac screening of all elite athletes is performed, far less has been known about cardiac structure and function in elite US athletes, for whom these mandatory screening policies do not exist. In 2006, the NBA adopted a policy of annual screening of athletes with stress echocardiography to supplement the requirement that players undergo a screening stress echocardiogram before entrance into the league. The NBA is the only US professional sports league to develop and operate such a cardiac program. The large contingent of NBA athletes forming our study represent an athletic group with more ethnic diversity and significantly greater height and BSA than any athletic group that has been studied on this scale. We have included an eTable in the Supplement for reference that contains all measured raw and indexed cardiac variables for the NBA cohort.

We found that LV cavity sizes in NBA players were larger than in normal adults, but that LV size was proportional to body size. This finding is comparable to those of series of other athlete groups, including Olympic athletes followed up at the Institute of Sports Science in Rome, Italy,[24-26] smaller series of elite athletes in mixed sports,[2,27-29] and 1 cohort of National Football League athletes.[30] The LV cavity sizes were not as large as those reported in professional cyclists[31] in which LV dilation was more pronounced than in any other published athlete group. The results of our study reinforce the concept that scaling LV size to

body size is vitally important in the cardiac evaluation of basketball players, whose heights extend to 218 cm and BSAs to 2.8 m^2.

Our data show just a modest degree of LVH in NBA athletes, with 27.4% of athletes having an increased LVMI. The upper limit of absolute LVWT was 15 mm (in 1 athlete), and 12.2% of the athletes had a maximal LVWT of 13 to 14 mm. African American athletes were found to have increased indices of LVH compared with white athletes, as reported in smaller series of athletes in mixed sports,[32-34] but specifically we found that African American athletes had a substantially higher proportion of nondilated concentric hypertrophy, whereas the most predominant form of hypertrophy in white athletes was eccentric dilated hypertrophy. These data from the NBA cohort help to extend the understanding and characterization of athletic hypertrophy in US basketball athletes and provide further reference to assist in the recognition of HCM in basketball players, an important task given the guidance from the most recent American Heart Association–American College of Cardiology Task Force recommendations[35] to restrict individuals diagnosed with HCM from all but low-intensity competitive sports.

With respect to LV systolic function, we found that most NBA athletes had a normal LVEF, whereas only 5 athletes (1.0%) had an LVEF of less than 50%. No athlete had an LVEF of less than 45%. All athletes had normal augmentation of LV systolic function with exercise and no athlete was found to have diastolic dysfunction. With serial echocardiographic evaluations of the athletes with lower LVEF, in keeping with NBA protocol, we have found that LVEF has remained stable or increased over time. Reports of individuals with low normal to mildly reduced LVEF are present in other elite athlete groups, such as professional football players and cyclists,[30,31] but no similarly corroborating evidence has suggested that these individuals have an underlying cardiomyopathy.

Aortic root diameter was a cardiac dimension of particular importance in the evaluation of NBA athletes, given the extreme height of the average NBA player and the potential for overlap with individuals who have Marfan syndrome. The maximum aortic root diameter measured in the NBA cohort was 42 mm, and just 4.6% of athletes had an aortic root of 40 mm or greater. In fact, aortic root sizes were similar to those reported across several elite athlete groups,[14,15,36,37] despite the different anthropometry between NBA players and these other athletes. We observed that in relation to body size, aortic root diameters tapered in their increase and reached a plateau at the uppermost heights and BSAs. Because to date no large-scale comparative data have assessed aortic root dimensions for athletes of the size of the average NBA player (mean height, 200.2 cm; mean BSA, 2.38 m^2), these data have important implications in the evaluation of exceptionally large athletes and question the applicability in individuals with significantly increased biometrics of the traditional formula to estimate aortic root diameter

that assumes a linear association between aortic diameter and BSA.[13,20,38] These data support recent American Heart Association–American College of Cardiology Task Force recommendations[39] to surveil with serial imaging tall athletes with aortic root diameters of greater than 41 mm.

The results of this study are the first to date to provide large-scale normative data for US basketball players, an athletic group with greater anthropometry than any previously studied athlete group and shown to have elevated rates of SCD. As highlighted by the American College of Cardiology Sports and Exercise Cardiology Think Tank,[40] gaining an understanding of the actual cardiac structural data without having to extrapolate from data in other athlete groups is essential for reference for the health care professionals who treat the large number of US basketball players from the youth to professional levels. We hope that the results of this study will assist recognition of cardiac pathologic change and provide a framework to help avoid unnecessary exclusions of athletes from competition. We believe that these data have additional applicability to other sports that preselect for athletes with height, such as volleyball, rowing, and track and field.

In our study, we did not undertake a formal evaluation of right ventricular structure and function. Inherent difficulties in the evaluation of right ventricular function at the present time include a lack of uniformity in image acquisition of the right ventricle across multiple centers and evolving standards for objectively assessing right ventricular size and function. However, in a subset of 128 athletes among whom tissue Doppler velocity of the lateral tricuspid anulus was measured (S'), all athletes were found to have normal right ventricular function by this method (right ventricular S' = 13.9 cm/s; 95% CI, 13.5-14.3 cm/s), and no players were found to have pulmonary hypertension. Other limitations of our study include the lack of a comparison group of nonathletes with biometrics similar to the NBA cohort and the lack of follow-up data for those athletes with cardiac variables on the outer edges of the distributions. Follow-up data collection is ongoing to assess the temporal changes of the cardiac variables in the athletes we included for analysis.

Conclusions

Conclusions about an athlete's heart should not be based on echocardiographic measurements in isolation. Instead, such conclusions require placement in full medical context, including a complete medical evaluation with incorporation of all other pertinent clinical information. We hope that the present data will help to focus decision making and improve clinical acumen for the purpose of primary prevention of cardiac emergencies in US basketball players and in the athletic community at large.

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─────────────────── **Invited Commentary** ───────────────────

Cardiac Variables in Professional Basketball Players
Looking Closely at the *Normal Big Athlete* (NBA)

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Exercise-induced cardiac remodeling (EICR) is the process of structural and functional myocardial adaptation that develops in response to sustained exercise training. Exercise-induced cardiac remodeling is a complex process with numerous determinants, including the athlete's age, sex, ethnicity, genetics, and sport type.[1] Although EICR was first described more than a century ago,[2] it remains an area of active investigation for 2 basic reasons. First, the study of EICR continues to refine our understandings of adaptive human biology and the role of the cardiovascular system in human performance. Second, EICR provides critical data for the effective clinical management of athletes and highly active individuals. Differentiating EICR from the diseases associated with the risk for adverse events during athletic participation is among the most challenging and important tasks for the sports cardiologist. At the most basic level, differentiating adaptive physiologic development from occult pathologic change requires a comprehensive understanding of what constitutes *normal* for a given athlete subtype.

In this issue of *JAMA Cardiology*, Engel et al[3] from Columbia University Medical Center present echocardiographic data from a sizable cohort of professional basketball players. Data derived from National Basketball Association's (NBA's) compulsory annual preparticipation screening protocol, unique in the global sports world in that it includes stress echocardiography, was used to generate a robust normative database. The athletes included in this 2-year study were young (mean [SD] age, 25.4 [4.3] years), largely African American (406 of 526 [77.2%]), and men who were most notable for their height (mean [SD], 200.2 [8.8] cm). Key findings from this effort can be summarized as follows. Measurements of the left heart structure including left ventricular (LV) cavity size, LV wall thickness, and LV mass exceeded normal cut points proposed for use in the general population in a significant number of athletes. However, each of these LV metrics was directly proportional to body size and not significantly different from more sedentary populations after appropriate scaling. Second, LV hypertrophy (LVH), the hallmark of the athlete's heart, was observed in 144 of 526 NBA athletes (27.4%), with ethnicity identified as a key determinant of the prevalence and geometric pattern of LVH. Third, LV function, as assessed using ejection

fraction, was mildly depressed (<50%) in a small subset of players who were noted to have larger ventricles than athletes with higher ejection fractions. All athletes with mildly reduced ejection fractions had appropriate ejection fraction augmentation during exercise provocation. Finally and perhaps of greatest interest, aortic root diameter, despite the immense body size of these athletes, exceeded 40 mm in very few athletes (24 of 526 [4.6%]), and when dilation was observed, it was of a very small magnitude (maximally recorded dimension, 42 mm). Unlike LV variables, which retained a direct relationship with height across the measurement spectrums, aortic root diameter reached a plateau at a body height of approximately 212 cm. This important finding confirms the notion that only mild aortic dilation should be considered physiologic among athletes[4] and suggests that athletes at the extreme end of the height spectrum should not be expected to demonstrate proportionally marked aortic dilation.

A consortium of experts recently convened by the American College of Cardiology[5] identified the development of normative, sport-specific, athlete databases as a key area of scientific pursuit. The study by Engel et al[3] provides a crucial piece of this puzzle and as such, the authors are to be commended for their work. Their data underscore the need for physicians who care for competitive basketball players, and perhaps other tall athletes from alternative sporting disciplines, to consider body size during the interpretation of cardiac imaging data as a matter of routine. In addition, this study reminds us that a mildly reduced LV ejection fraction, particularly among athletes with marked eccentric LVH (concomitant wall thickening and chamber dilation), most often represents physiologic features and not disease. This finding is best explained by the concept that stroke volume, not ejection fraction, is physiologically regulated and that athletes with large ventricles simply need to eject a smaller percentage of end-diastolic volume under resting conditions. As nicely demonstrated in this study, augmentation of LV ejection fraction during exercise may be a useful tool for differentiating the low ejection fraction of health from that of mild myopathy.

Finally, this study enhances our understanding of the association between aortic size and athletic participation in perhaps the most clinically relevant population. Unlike ventricular size, which increases proportionally with body size, aortic