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Sleep-Disordered Breathing and Cardiovascular Correlates in College Football Players

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Abstract

This study sought to determine the cardiovascular physiologic correlates of sleep disordered breathing (SDB) among American-style football (ASF) participants using echocardiography, vascular applanation tonometry, and peripheral arterial tonometry. 40 collegiate ASF participants were analyzed at pre- and post-season time points with echocardiography and vascular applanation tonometry. WatchPAT[®] (inclusive of peripheral arterial tonometry) used to assess for SDB was then performed at the post-season time point. 22/40 (55%) ASF participants demonstrated SDB with an apnea-hypopnea index (pAHI) ≥ 5 . ASF participants with SDB were larger (109 ± 20 vs. 92 ± 14 kg, $P=0.004$) and more likely linemen position players (83% vs. 50%, $P=0.03$). Compared to those without SDB, ASF participants with SDB demonstrated relative impairments in left ventricular diastolic and vascular function as reflected by lower lateral e' (14 ± 3 vs. 17 ± 3 cm/s, $P=0.007$) and septal e' (11 ± 2 vs. 13 ± 2 cm/s, $P=0.009$) tissue velocities and higher pulse wave velocity (5.4 ± 0.9 vs. 4.8 ± 0.5 m/s, $P=0.02$). In the total cohort, there were significant positive correlations between pAHI and pulse wave velocity ($r=0.42$, $P=0.008$) and inverse correlations between pAHI and the averaged e' tissue velocities ($r=-0.42$, $P=0.01$). In conclusion, SDB is highly prevalent among youthful collegiate ASF participants and associated with relative impairments in cardiac and vascular function. Targeted efforts to identify youthful populations

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with SDB, including ASF participants, and implement SDB treatment algorithms represent important future clinical directives.

Keywords

sleep; American-style football; diastolic function; arterial stiffness; cardiac remodeling

INTRODUCTION

Increased rates of SDB have been reported among collegiate and professional American-style football (ASF) participants in the United States.^{1–3} In parallel, mounting data document associations between ASF participation and adverse cardiovascular health parameters including hypertension,⁴ arterial stiffening,⁵ and reduced LV function.^{6, 7} At present, the relations between SDB and underlying cardiovascular physiology among youthful ASF participants remain uncertain. We therefore sought to determine the cardiovascular physiologic correlates of SDB among ASF participants. We hypothesized that ASF participants with SDB would demonstrate relative decrements across clinically relevant parameters of cardiac and vascular function parameters compared to those without SDB. To address this hypothesis, we longitudinally studied collegiate ASF participants before and after a competitive ASF season to define sport-related cardiovascular changes and then measured sleep quality to examine relations between SDB and acquired cardiovascular structure and function.

METHODS

ASF participants representing two National Collegiate Athletic Association Division I programs [Georgia Institute of Technology (Atlanta, GA) and Furman University (Greenville, SC)] were recruited for this study. Anthropometrics, clinical characteristics, 2-D echocardiography, and vascular function measurements were performed longitudinally at two time points to define relevant physiologic changes associated with ASF participation. Time point one was the pre-season, representing the time of initial university matriculation (2014–15). Time point two was the immediate conclusion of the 2015 ASF season and represented the end of the freshman (6 months later) and sophomore ASF campaign (18 months later). While the inclusion of freshmen and sophomores led to differences in time between data points, analyses of post-season cardiac remodeling were performed to ensure similar measures of cardiac structure and function. The inclusion of second year ASF participants allowed for the assessment of SDB further into the collegiate ASF career. WatchPAT® data, inclusive of peripheral arterial tonometry (PAT), defining sleep quality were then collected at the post-season time point to facilitate analyses examining the associations between SDB and acquired ASF cardiovascular characteristics. The Emory Institutional Review Board approved all aspects of the study before initiation and subjects provided written informed consent.

Anthropometric and clinical data collected included age (years), height (cm), weight (kg), current medication use, personal/family history of hypertension, systolic and diastolic blood pressure [(mm Hg), measured using a manual aneroid sphygmomanometer and an

appropriately sized cuff], 2-D echocardiography, and vascular applanation tonometry. Participants were required to abstain from exercise for 24 hours prior data collection time points. Field position for each ASF participant was classified as either lineman (LM) or non-lineman (NLM) as previously proposed.⁸ Each participant was subject to performance-enhancing drug testing as per NCAA standards.

Arterial stiffness was measured using high fidelity applanation tonometry (SphygmoCor®, Atcor Medical, Australia), which records sequential high-quality pressure waveforms at peripheral pulse sites. Full details of tonometer technology and measurement algorithms have been previously detailed.⁹ Vascular function was characterized by pulse wave velocity (PWV), the gold standard index of arterial stiffness,⁹ and was measured by acquisition of pressure waveforms within the carotid and femoral arteries. The distance between carotid and femoral sites was estimated using the “foot-to-foot” method.¹⁰ PWV with <10% standard deviation was required for quality control.

Trans-thoracic echocardiography was performed using a commercially available system (Vivid-I, GE Healthcare, Milwaukee, WI) after 20 minutes of rest. 2-D, tissue-Doppler, and speckle-tracking imaging from standard parasternal and apical positions was performed by experienced sonographers. All data were stored digitally, and post-study offline data analysis (GE EchoPAC version 7) was performed (J.H.K). Definitions of normality for cardiac structure and function were adopted from the most recent guidelines.¹¹ LV ejection fraction, end-diastolic volume, and end-systolic volume were calculated using the modified biplane technique. LV mass was calculated using the area-length method. Relative wall thickness was calculated as: $[(2 \times \text{posterior wall thickness (mm)}) / \text{LV end-diastolic diameter (mm)}]$. Concentric LV hypertrophy was defined as a relative wall thickness of >0.42 with an LV mass index of $>102 \text{ g/m}^2$; concentric LV remodeling was defined as a relative wall thickness of >0.42 with an LV mass index of $\leq 102 \text{ g/m}^2$; and eccentric LV hypertrophy was defined as a relative wall thickness of ≤ 0.42 with an LV mass index of $>102 \text{ g/m}^2$.¹¹ Measurements were adjusted for body surface area when appropriate.¹² Assessment of regional myocardial function using speckle-tracking and tissue-Doppler imaging was performed. Longitudinal tissue velocities (e' , a' , and s') were measured from images at the lateral and septal mitral annulus. Global LV longitudinal strain was measured only in the apical four-chamber view.

PAT was used to measure sleep quality was performed using the WatchPAT-200® device (Itamar Medical Ltd., Caesarea, Israel), a validated surrogate for polysomnography.^{13–15} In brief, this is a forearm-mounted device with 2 finger probes, a pneumo-optical sensor measuring PAT and pulse oximeter.^{13, 14} Overnight, the device recorded the PAT signal, oxyhemoglobin saturation, and sleep-wake states.^{13, 14} Sleep-time detection was determined by total recording time minus invalid signals and wake time and internal algorithms were used to differentiate respiratory events.¹³ Studies were considered valid for those with total sleep time ≥ 3 hours. The apnea-hypopnea index (pAHI) was calculated as number of apneic and hypopneic events per hour of detected sleep and calculated based on oxygen-saturation data and indication of autonomic activation from PAT.¹³ Respiratory events detected from the PAT signal associated with an oxygen desaturation events of $\geq 3\%$ counted into the pAHI.¹³ All recorded data were stored at a sample rate of 100 Hz on an internal SD card and then downloaded off-line for further analysis and review by a board certified sleep specialist

blinded to participant demographics and characteristics (N.A.C.). pAHI of ≥ 5 and <15 events per hour of sleep was considered abnormal and equivalent to mild SDB.^{16, 17} pAHI of 15 but <30 events per hour was considered moderate SDB, and pAHI ≥ 30 was considered severe.^{16, 17}

Categorical variables are presented as percentages and continuous variables as mean $\pm$ standard deviation. Repeated measures were assessed with the paired *t*-test for normally distributed variables or the Wilcoxon signed-rank test for non-normally distributed variables. Comparison of post-season measures was assessed with a 2-sample *t*-test for normally distributed variables or the Wilcoxon rank-sum test for non-normally distributed variables. Comparisons between categorical variables were performed by the Chi-square test of homogeneity. Pearson correlation coefficients were determined for correlations between post-season measured pAHI (log transformed) and weight, PWV, and measures of LV diastolic function. Analyses were performed with SAS software (version 9.3, Cary, North Carolina). A P-value of ≤ 0.05 was considered significant.

RESULTS

Of 46 ASF participants initially recruited for this study, 40 (21 freshmen and 19 sophomores, 18.1 ± 0.5 years old) remained free of injury and submitted adequate WatchPAT[®] data for interpretation. The study cohort was evenly distributed in ethnicity (55% Caucasian / 45% African-American) and predominantly NLM by field position (65% vs. 35%). While no subject reported a personal history of hypertension or taking prescription cardiovascular medications, 40% disclosed a family history of hypertension. During the study period, ASF participants demonstrated increases in LV wall thickness, relative wall thickness, and LV mass index, a pattern consistent with concentric LV remodeling, and concomitant reductions in diastolic and vascular function (Table 1). In the separate analysis of freshman versus sophomore ASF participants, similar post-season cardiac and vascular changes were observed (Table 2). 55% of ASF participants demonstrated evidence of at least mild SDB (pAHI ≥ 5) with 3/40 (8%, 1 LM / 2 NLM) found to have severe SDB (pAHI >30). ASF participants with SDB were larger and more likely to be classified as LM (Table 3).

Concentric LV remodeling or hypertrophy was more common among ASF participants with SDB (9/22, 41%) than among those with normal sleep patterns (2/18, 11%; $P = 0.04$) at post season. In contrast, LV remodeling among ASF participants without SDB was characterized by balanced increases in LV wall thickness and chamber diameter with stable relative wall thickness (i.e. eccentric remodeling), which translated into eccentric LV hypertrophy in 8/18 participants (44%, Table 4). Compared to ASF participants without SDB, ASF participants with SDB demonstrated more pronounced longitudinal decrements in diastolic function, which translated to relative impairments in diastolic function at the post-season time point (Figure 1, Table 4). In regard to vascular function, post-season PWV was significantly increased in the SDB ASF group (Table 4) and was associated with a modest, but longitudinal increase in PWV from the pre-season (Figure 1).

Correlation analyses between pAHI and minimum O₂ saturation, weight, PWV, and LV diastolic tissue velocities were performed in the total cohort (Figure 2). There were significant positive correlations found between pAHI and weight ($P = 0.02$) and PWV ($P = 0.008$) as well as significant inverse correlations found between pAHI and minimum O₂ saturation ($P < 0.001$) and the averaged e' tissue velocities ($P = 0.01$). Upon removal of the 3 outlying subjects with pAHI > 30 , correlations between pAHI and weight ($r = 0.47$, $P = 0.004$), PWV ($r = 0.38$, $P = 0.02$), minimum O₂ saturation ($r = -0.48$, $P = 0.003$), and averaged e' ($r = -0.60$, $P = 0.002$) remained significant.

DISCUSSION

This study was designed to examine associations between SDB and cardiovascular phenotypes among healthy collegiate ASF participants. Key findings are as follows. Consistent with prior studies,^{4–7} we documented sub-clinical pathologic changes in numerous cardiovascular health metrics including blood pressure, LV diastolic function, and vascular stiffness among ASF athletes during relatively short time periods of ASF participation. Second, we observed a substantial burden of SDB, which was associated with factors including weight and the LM position. Finally, we detected significant correlation between SDB and relative impairments in vascular and LV diastolic function thereby suggesting a link between SDB and maladaptive ventriculo-arterial coupling. In aggregate, our data confirm that ASF participants are at risk for SDB and demonstrate that those with SDB harbor corollary sub-clinical cardiovascular pathology similar to that reported in older, more co-morbid members of the general population with SDB.^{18–21}

The association between SDB and adverse long-term cardiovascular outcomes, particularly hypertension, has been well established.^{22–26} However, associations between SDB and cardiovascular pathophysiologic correlates remain comparatively limited. One prior study documented increased LV hypertrophy and reduced diastolic function among 43 patients with OSA compared to controls.¹⁹ Of note, the OSA cohort in this study was comprised of older (55 ± 11 years) men with a substantial burden of hypertension ($153 \pm 25 / 88 \pm 17$ mm Hg) and tobacco use (28%).¹⁹ Comparable cross-sectional data exist but have limited generalizability to healthier individuals with SDB.^{27–29} The present study therefore adds to our broader understanding of the pathophysiology of SDB by demonstrating cardiovascular correlates of SDB in a cohort of healthy ASF athletes, who appear to be uniquely at risk for early life SDB.

Increased prevalence of early life hypertension, LV hypertrophy with reduced diastolic function,⁶ and reduced vascular function have been previously demonstrated among ASF participants.^{4, 5} In one analysis of 113 freshman collegiate ASF athletes, 60% developed at least pre-hypertension after one competitive ASF season with LM appearing to be at highest risk.⁴ In a separately assessed collegiate ASF cohort, increases in blood pressure coincided with arterial stiffness with both processes occurring more frequently when compared to non-athletic undergraduate controls.⁵ Despite these accumulating data documenting acquired maladaptive physiology among ASF participants, insights into causal mechanisms, particularly in regard to the role of SDB, remain speculative.

Findings from the current study begin to shed insight into this critical area of uncertainty by defining the following novel relationships between SDB and concomitant cardiovascular physiology among ASF participants. First, SDB prevalence estimates in this analysis (~55%), though derived from a relatively small cohort, were increased compared to prior reports (8% – 25%),^{1–3} thereby confirming that SDB is highly prevalent among ASF participants. Second, we found that increased weight as a result of ASF participation, particularly among LM, significantly correlated with the presence of SDB. This finding suggests that ASF-associated weight gain represents a primary risk factor for SDB and may be a useful screening metric for targeted assessment of at-risk athletes.^{1–5, 30} Finally, our data suggest that SDB represents a mechanistic factor underlying maladaptive ventriculo-arterial coupling and ASF-associated hypertension. Clarification of the differential impact of weight gain, SDB, and other vascular risk factors (ex. pharmacologic anti-inflammatory use, dietary sodium intake, oxidative stress, etc.) on pathologic cardiovascular ASF phenotypes represents an important area of future work.

There are clinical directives that arise from this study. Our data confirm that young ASF athletes are at risk for the development of early life subclinical cardiovascular disease including SDB. This finding underscores the need to prioritize cardiovascular risk assessments that include screening for SDB and initiate guideline-based lifestyle and pharmacotherapy when appropriate. In addition, our findings suggest that ASF participants with hypertension may benefit from diagnostic testing to assess for SDB. The association between SDB and pathologic ventriculo-arterial coupling coupled with the treatability of this condition may represent opportunities to improve long-term health outcomes. The current study sets the stage for future longitudinal analyses incorporating repeated measures PAT with cardiac and vascular function testing to establish the temporal relationships between SDB and ASF-related cardiovascular dysfunction.

Several limitations of this study are noteworthy. First, the overall limited subject numbers and the additional stratification of the study cohort by pAHI limited the power of the study and introduced the possibility of type II error. Second, although cardiac imaging and vascular function testing were performed longitudinally, WatchPAT[®] was implemented exclusively at the post-season time point. We are therefore unable to comment on changes in SDB prevalence and severity over time. Third, factors other than SDB that may drive ASF-acquired cardiovascular dysfunction were not addressed, thus making mechanistic interpretations speculative. Finally, the evaluation of ASF participants at alternative levels of competition and intensity, such as at the high school and professional level, were not included in the present study.

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References

1. Rice TB, Dunn RE, Lincoln AE, Tucker AM, Vogel RA, Heyer RA, Yates AP, Wilson PW, Pellmen EJ, Allen TW, Newman AB, Strollo PJ Jr, National Football League Subcommittee on

- Cardiovascular H. Sleep-disordered breathing in the National Football League. *Sleep*. 2010; 33:819–824. [PubMed: 20550023]
2. George CF, Kab V, Kab P, Villa JJ, Levy AM. Sleep and breathing in professional football players. *Sleep Med*. 2003; 4:317–325. [PubMed: 14592304]
 3. Dobrosielski DA, Nichols D, Ford J, Watts A, Wilder JN, Douglass-Burton T. Estimating the Prevalence of Sleep-Disordered Breathing Among Collegiate Football Players. *Respir Care*. 2016; 61:1144–1150. [PubMed: 26979096]
 4. Weiner RB, Wang F, Isaacs SK, Malhotra R, Berkstresser B, Kim JH, Hutter AM Jr, Picard MH, Wang TJ, Baggish AL. Blood pressure and left ventricular hypertrophy during American-style football participation. *Circulation*. 2013; 128:524–531. [PubMed: 23897848]
 5. Kim JH, Sher S, Wang F, Berkstresser B, Shoop JL, Galante A, Al Mheid I, Ghasemzadeh N, Hutter AM, Williams BR 3rd, Sperling LS, Weiner RB, Quyyumi AA, Baggish AL. Impact of American-style football participation on vascular function. *Am J Cardiol*. 2015; 115:262–267. [PubMed: 25465938]
 6. Baggish AL, Wang F, Weiner RB, Elinoff JM, Tournoux F, Boland A, Picard MH, Hutter AM Jr, Wood MJ. Training-specific Changes in Cardiac Structure and Function: A Prospective and Longitudinal Assessment of Competitive Athletes. *J Appl Physiol*. 2008; 104:1121–1128. [PubMed: 18096751]
 7. Lin J, Wang F, Weiner RB, DeLuca JR, Wasfy MM, Berkstresser B, Lewis GD, Hutter AM Jr, Picard MH, Baggish AL. Blood pressure and left ventricular remodeling among American-style football players. *JACC Cardiovasc Imaging*. 2016; 9:1367–1376. [PubMed: 27931524]
 8. Croft LB, Belanger A, Miller MA, Roberts A, Goldman ME. Comparison of National Football League linemen versus nonlinemen of left ventricular mass and left atrial size. *Am J Cardiol*. 2008; 102:343–347. [PubMed: 18638599]
 9. Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, Pannier B, Vlachopoulos C, Wilkinson I, Struijker-Boudier H, European Network for Non-invasive Investigation of Large A. Expert consensus document on arterial stiffness: methodological issues and clinical applications. *Eur Heart J*. 2006; 27:2588–2605. [PubMed: 17000623]
 10. Yasmin, Brown MJ. Similarities and differences between augmentation index and pulse wave velocity in the assessment of arterial stiffness. *QJM*. 1999; 92:595–600. [PubMed: 10627881]
 11. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, Lancellotti P, Muraru D, Picard MH, Rietzschel ER, Rudski L, Spencer KT, Tsang W, Voigt JU. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging*. 2015; 16:233–270. [PubMed: 25712077]
 12. Mosteller RD. Simplified calculation of body-surface area. *N Engl J Med*. 1987; 317:1098. [PubMed: 3657876]
 13. Zou D, Grote L, Peker Y, Lindblad U, Hedner J. Validation a portable monitoring device for sleep apnea diagnosis in a population based cohort using synchronized home polysomnography. *Sleep*. 2006; 29:367–374. [PubMed: 16553023]
 14. Bar A, Pillar G, Dvir I, Sheffy J, Schnall RP, Lavie P. Evaluation of a portable device based on peripheral arterial tone for unattended home sleep studies. *Chest*. 2003; 123:695–703. [PubMed: 12628865]
 15. Choi JH, Kim EJ, Kim YS, Choi J, Kim TH, Kwon SY, Lee HM, Lee SH, Shin C, Lee SH. Validation study of portable device for the diagnosis of obstructive sleep apnea according to the new AASM scoring criteria: Watch-PAT 100. *Acta Otolaryngol*. 2010; 130:838–843. [PubMed: 20082567]
 16. Epstein LJ, Kristo D, Strollo PJ Jr, Friedman N, Malhotra A, Patil SP, Ramar K, Rogers R, Schwab RJ, Weaver EM, Weinstein MD, Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep M. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med*. 2009; 5:263–276. [PubMed: 19960649]

17. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep*. 1999; 22:667–689. [PubMed: 10450601]
18. Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ, Stubbs R, Hla KM. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. *Sleep*. 2008; 31:1071–1078. [PubMed: 18714778]
19. Shivalkar B, Van de Heyning C, Kerremans M, Rinkevich D, Verbraecken J, De Backer W, Vrints C. Obstructive sleep apnea syndrome: more insights on structural and functional cardiac alterations, and the effects of treatment with continuous positive airway pressure. *J Am Coll Cardiol*. 2006; 47:1433–1439. [PubMed: 16580533]
20. Lisi E, Faini A, Bilo G, Lonati LM, Revera M, Salerno S, Giuli V, Lombardi C, Parati G. Diastolic dysfunction in controlled hypertensive patients with mild-moderate obstructive sleep apnea. *Int J Cardiol*. 2015; 187:686–692. [PubMed: 25910471]
21. Korcarz CE, Benca R, Barnett JH, Stein JH. Treatment of Obstructive Sleep Apnea in Young and Middle-Aged Adults: Effects of Positive Airway Pressure and Compliance on Arterial Stiffness, Endothelial Function, and Cardiac Hemodynamics. *J Am Heart Assoc*. 2016; 5:e002930. [PubMed: 27039928]
22. Garvey JF, Pengo MF, Drakatos P, Kent BD. Epidemiological aspects of obstructive sleep apnea. *J Thorac Dis*. 2015; 7:920–929. [PubMed: 26101650]
23. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. 1993; 328:1230–1235. [PubMed: 8464434]
24. Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA*. 2000; 284:3015–3021. [PubMed: 11122588]
25. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med*. 2000; 342:1378–1384. [PubMed: 10805822]
26. Nieto FJ, Young TB, Lind BK, Shahar E, Samet JM, Redline S, D'Agostino RB, Newman AB, Lebowitz MD, Pickering TG. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep Heart Health Study. *JAMA*. 2000; 283:1829–1836. [PubMed: 10770144]
27. Chami HA, Devereux RB, Gottdiener JS, Mehra R, Roman MJ, Benjamin EJ, Gottlieb DJ. Left ventricular morphology and systolic function in sleep-disordered breathing: the Sleep Heart Health Study. *Circulation*. 2008; 117:2599–2607. [PubMed: 18458174]
28. Tavit Y, Kanbay A, Sen N, Ulukavak Ciftci T, Abaci A, Yalcin MR, Kakturk O, Cengel A. The relationship between aortic stiffness and cardiac function in patients with obstructive sleep apnea, independently from systemic hypertension. *J Am Soc Echocardiogr*. 2007; 20:366–372. [PubMed: 17400115]
29. Jones A, Vennelle M, Connell M, McKillop G, Newby DE, Douglas NJ, Riha RL. Arterial stiffness and endothelial function in obstructive sleep apnoea/hypopnoea syndrome. *Sleep Med*. 2013; 14:428–432. [PubMed: 23462229]
30. Tucker AM, Vogel RA, Lincoln AE, Dunn RE, Ahrensfield DC, Allen TW, Castle LW, Heyer RA, Pellman EJ, Strollo PJ Jr, Wilson PW, Yates AP. Prevalence of cardiovascular disease risk factors among National Football League players. *JAMA*. 2009; 301:2111–2119. [PubMed: 19470988]

Figure 1.

Comparison of the pre- to post-season changes in cardiac function and vascular function in American-style football participants with (N = 22) and without (N = 18) evidence of sleep disordered breathing.

pAHI: WatchPAT[®] determined apnea-hypopnea index; **TDI:** tissue-Doppler imaging

*P < 0.05 for within group

Figure 2.

Correlation analyses between post-season apnea hypopnea index (log transformed) and post-season weight, vascular function, and cardiac diastolic function in the total (N = 40) American-style football participant cohort.

TDI: tissue-Doppler imaging

*Log transformed apnea-hypopnea index

Table 1

Longitudinal cardiac remodeling and vascular function differences in American-style football participants (N = 40)

Longitudinal ASF Data			
Variable	Pre-First Year	Post-Season	P-value
Weight (kg)	100 ± 20	102 ± 20	0.03
Systolic Blood Pressure (mm Hg)	133 ± 10	135 ± 11	0.42
Diastolic Blood Pressure (mm Hg)	72 ± 9	75 ± 10	0.01
Ventricular Septal Thickness (mm)	8.9 ± 1.1	9.7 ± 1.0	<0.001
Posterior Wall Thickness (mm)	9.3 ± 1.0	10.1 ± 1.0	<0.001
LV Internal Diameter End-Diastole / Body Surface Area (mm/m ²)	22.5 ± 2	22.6 ± 3	0.63
LV Mass / Body Surface Area (gm/m ²)	93.5 ± 12	101 ± 11	<0.001
Relative Wall Thickness	.37 ± .04	.39 ± .06	0.02
Ejection Fraction (%)	62 ± 5	59 ± 4	0.009
4-Chamber Global Longitudinal Strain (%)	19.6 ± 2	19 ± 2	0.15
Trans-Mitral E (cm/s)	89 ± 17	80 ± 15	0.003
Trans-Mitral A (cm/s)	44 ± 10	46 ± 10	0.41
Trans-Mitral E/A Ratio	2.1 ± 0.6	1.8 ± 0.4	0.008
Tissue-Doppler LV Lateral E' (cm/s)	20.4 ± 4	15.5 ± 3	<0.001
Tissue-Doppler LV Septal E' (cm/s)	13.1 ± 2	11.8 ± 2	0.004
Pulse Wave Velocity (m/s)	4.8 ± 0.7	5.1 ± 0.8	0.02

ASF: American-style football; **LV**: left ventricle

Table 2

Comparison of post-season cardiac structure, cardiac function, and vascular function measurements in freshmen and sophomore American-style football participants

Variable	Post-Season ASF Comparison (N = 40)		
	Freshman (N = 21)	Sophomore (N = 19)	P-value
Ventricular Septal Thickness (mm)	9.7 ± 0.9	9.6 ± 1.3	0.85
Posterior Wall Thickness (mm)	10.2 ± 1	10 ± 1.4	0.65
LV Internal Diameter End-Diastole / Body Surface Area (mm/m ²)	22.5 ± 2.7	22.9 ± 2.2	0.66
LV Mass / Body Surface Area (gm/m ²)	103 ± 13	99 ± 8	0.31
Relative Wall Thickness	.40 ± .05	.38 ± .06	0.26
Ejection Fraction (%)	60 ± 3	59 ± 4	0.38
4-Chamber Global Longitudinal Strain (%)	19.4 ± 2	17.8 ± 3	0.06
Trans-Mitral E (cm/s)	79 ± 15	82 ± 14	0.56
Trans-Mitral A (cm/s)	43 ± 9	50 ± 10	0.03
Trans-Mitral E/A Ratio	1.9 ± 0.4	1.7 ± 0.5	0.23
Tissue-Doppler LV Lateral E' (cm/s)	15.8 ± 3	15 ± 3	0.40
Tissue-Doppler LV Septal E' (cm/s)	12 ± 2	11.4 ± 2	0.44
Pulse Wave Velocity (m/s)	5 ± 0.8	5.2 ± 0.9	0.66

ASF: American-style football; LV: left ventricle

Table 3

Characteristics of American-style football participants with and without sleep disordered breathing

ASF Characteristics	Post-Season ASF Participants (N = 40)		P-value
	pAHI <5 (N = 18)	pAHI ≥ 5 (N = 22)	
Freshmen / Sophomore (%)	55 / 45	50 / 50	0.77
Age (years)	18.1 ± 0.5	18.1 ± 0.4	0.81
White	59%	50%	0.56
Black	41%	50%	
Non-Linemen / Linemen	50% / 50%	17% / 83%	0.03
Weight (kg)	92 ± 14	109 ± 20	0.004
Systolic Blood Pressure (mm Hg)	132 ± 11	137 ± 10	0.17
Diastolic Blood Pressure (mm Hg)	75 ± 8	76 ± 12	0.76
WatchPAT®			
Total Sleep Time (min)	324 ± 88	318 ± 89	0.82
pAHI	2.6 ± 1.1	11.1 ± 10	0.001
Maximum O ₂ Saturation (%)	99.7 ± 0.6	99.3 ± 0.9	0.11
Minimum O ₂ Saturation (%)	92.2 ± 2.9	89.2 ± 2.6	0.002
O ₂ Saturation (%)	7.5 ± 2.9	10 ± 2.9	0.008

pAHI: WatchPAT® determined apnea-hypopnea index; **ASF:** American-style football

Table 4

Post-season cardiac remodeling and vascular function differences in American-style football participants with and without sleep disordered breathing

Variable	Post-Season ASF Comparison (N = 40)		
	pAHI <5 (N = 18)	pAHI ≥ 5 (N = 22)	P-value
Interventricular Septum Thickness (mm)	9.3 ± 0.8 *	10 ± 1.2 *	0.09
Posterior Wall Thickness (mm)	9.6 ± 1.0 *	10.5 ± 1.2 *	0.03
LV Internal Diameter End-Diastole / Body Surface Area (mm/m ²)	24.2 ± 2 *	21.5 ± 2	<0.001
LV Mass / Body Surface Area (gm/m ²)	105 ± 11 *	99 ± 11 *	0.12
Relative Wall Thickness	.37 ± .04	.41 ± .06 *	0.04
Ejection Fraction (%)	59 ± 4	60 ± 4 †	0.85
4-Chamber Global Longitudinal Strain (%)	18.9 ± 2	19 ± 3	0.72
Trans-Mitral E (cm/s)	85 ± 11 †	77 ± 16 †	0.11
Trans-Mitral A (cm/s)	44 ± 8	47 ± 12	0.46
Trans-Mitral E/A Ratio	2 ± 0.5	1.7 ± 0.4 †	0.06
Tissue-Doppler LV Lateral E' (cm/s)	17 ± 3 †	14 ± 3 †	0.007
Tissue-Doppler LV Septal E' (cm/s)	13 ± 2	11 ± 2 †	0.009
Pulse Wave Velocity (m/s)	4.8 ± 0.5	5.4 ± 0.9 *	0.02

pAHI: WatchPAT[®] determined apnea-hypopnea index; ASF: American-style football; LV: left ventricle

* P<0.05 time point 1 to time point 2 longitudinal increase

† P<0.05 time point 1 to time point 2 longitudinal decrease