NIH Public Access
Author Manuscript
Am J Cardiol. Author manuscript; available in PMC 2014 May 08.

Published in final edited form as:
Am J Cardiol. 2013 June 1; 111(11): 1650–1654. doi:10.1016/j.amjcard.2013.02.008.

Functional Aerobic Capacity in Patients With Sleep-Disordered Breathing

Meghna P. Mansukhani, MBBS[a], **Thomas G. Allison, PhD, MPH**[b], **Francisco Lopez-Jimenez, MD, MS**[b], **Virend K. Somers, MD, PhD**[b], and **Sean M. Caples, DO**[a,c,*]

[a]Center for Sleep Medicine, Mayo Clinic, Minnesota

[b]Division of Cardiovascular Diseases, Mayo Clinic, Minnesota

[c]Division of Pulmonary and Critical Care Medicine, Mayo Clinic, Minnesota

Abstract

Few studies have examined exercise capacity or cardiovascular responses to maximal exercise testing and recovery in patients with sleep-disordered breathing (SDB), and results from these studies are conflicting. The objective of this cross-sectional study conducted at a tertiary referral center was to examine the association between SDB and exercise testing outcomes independent of body mass index (BMI) and other cardiopulmonary risk factors. Between January 1, 2005 and January 1, 2010, 1,424 adults underwent exercise testing and within 6 months before first-time diagnostic polysomnography. Subjects were categorized by apnea-hypopnea index (AHI) into 4 groups: <5, 5 to 14, 15 to 29, and ≥30. A logistic regression model incorporated age, gender, BMI, smoking, hypertension, diabetes, beta-blocker use, and cardiac and pulmonary disease as covariates. The primary variable of interest was functional aerobic capacity (FAC). Mean age was 56.4 ± 12.4 years; 75% were men. Mean BMI was 32.4 ± 7.1 kg/m^2, and mean AHI 19.5 ± 22.1 per hour. On multivariate analysis, AHI as a continuous variable showed a negative correlation with FAC (R^2adj = 0.30, p <0.001) and postexercise SBP (R^2adj = 0.23, p = 0.03), and positively correlated with resting and peak DBP (R^2adj = 0.09, p = 0.01 and R^2adj = 0.09, p = 0.04 respectively). When comparing patients with severe SDB (AHI ≥30) with those without SDB (AHI <5), FAC and heart rate recovery were significantly lower, and resting, peak, and postexercise DBP were higher in those with severe apnea (all p <0.05), after accounting for confounders. In conclusion, SDB severity was associated with reduced FAC and increased resting and peak DBP. Even after accounting for confounders, severe SDB was associated with attenuated FAC, impaired heart rate recovery, and higher resting, peak, and postexercise DBP.

We hypothesized that decreased exercise capacity is a possible consequence of the cardiopulmonary and deconditioning effects of sleep apnea. Few studies have examined exercise capacity and responses to maximal exercise testing and recovery in patients with

© 2013 Elsevier Inc. All rights reserved.

*Corresponding author: Tel: (507) 266-6880; fax: (507) 266-7772. caples.sean@mayo.edu (S.M. Caples).

Disclosures

See page 1653 for disclosure information.

The other authors have no disclosures.

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

sleep-disordered breathing (SDB). The limited available data are derived from small studies, and results are conflicting. Furthermore, in most previous studies, it is difficult to discern the effects of SDB independent of BMI and cardiopulmonary disease.[1] We aimed to conduct a cross-sectional study at our center to examine the association between SDB and exercise testing outcomes independent of body mass index (BMI) and other cardiopulmonary risk factors.

Methods

Patients who were referred to our center for comprehensive exercise testing between January 1, 2005 and January 1, 2010 were identified from the Cardiovascular Health Clinic (CVHC) database. Of these, subjects who underwent first-time diagnostic polysomnography (PSG) at the Center for Sleep Medicine at our facility within 6 months following the appropriate exercise test were identified using the relevant *International Classification of Diseases* (9th revision) procedure codes. Verification that this was the first diagnostic PSG and that the subject was treatment-naive was made by detailed review of the individual electronic medical records. Patients with amyloid or sarcoid heart disease; liver, kidney, or cardiac transplant; on dialysis; or with a history of previous lung resection were excluded.

Information on comorbidities and medications was collected by the clinician assessing the patient at the time of presentation to the CVHC for exercise testing. This was performed through patient interview and review of the electronic medical record, including results of other testing such as echocardiogram or pulmonary function tests where available. PSG measures collected by review of the individual sleep study reports were apnea-hypopnea index (AHI) and sleep efficiency. Patients were divided into 4 subgroups based on their AHI (<5, 5–14, 15–29, and ≥30). PSGs were manually scored by registered PSG technologists and reviewed by physicians board certified in sleep medicine, using standard American Academy of Sleep Medicine criteria.[2]

Most patients underwent a treadmill exercise test using the Bruce, Naughton, or modified Naughton protocols.[3] A minority had cycle ergometry. Exercise testing variables included predicted maximum exercise time, maximum exercise time achieved, predicted metabolic equivalents (METs), METs achieved, and functional aerobic capacity (FAC), the main outcome measure. FAC was calculated using a nomogram based on age, sex, baseline activity level and observed duration of exercise.[4] Data on resting heart rate (HR), peak exercise HR, 1-minute post–peak exercise HR, resting systolic blood pressure (SBP) and diastolic blood pressure (DBP), peak exercise SBP and DBP, and 3-minute post–peak exercise SBP and DBP were collected. Heart rate recovery (HRR), SBP, and DBP recovery were calculated as the difference or ratio between peak exercise and postexercise values, respectively. Where applicable, reason for termination of the test, electrocardiogram abnormality, grade of dyspnea or ventricular arrhythmia, and chest pain index were recorded. Most patients were referred for an exercise test to the CVHC for symptoms of fatigue, chest pain, and palpitations or for testing before prescribing exercise in sedentary individuals. Forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC) were measured if required depending on test indication (e.g., complex cardiovascular disease, risk stratification in heart failure, dyspnea on exertion, or in athletes).

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Covariates in the logistic regression analyses included age, gender, BMI (kg/m^2), history of current smoking, hypertension, diabetes mellitus, beta-blocker use, cardiac disease (including coronary artery disease, cardiomyopathy, heart failure, known arrhythmia, valvular heart disease, congenital heart disease), and pulmonary disease (chronic obstructive pulmonary disease, asthma, restrictive lung disease, previous pulmonary embolism or presence of pulmonary artery stenosis, other pulmonary disease).

The primary variable of interest assessed was FAC. Secondary variables of interest were HR, peak exercise HR, 1-minute post-peak exercise HR, HRR, resting SBP and DBP, peak exercise SBP and DBP, 3-minute post-peak exercise SBP and DBP, and SBP and DBP recovery, electrocardiogram abnormality, and grade of ventricular arrhythmia. Univariate and multivariate analyses were conducted using AHI as a continuous variable to assess for a dose-response relationship between severity of SDB and the variable of interest. In subsequent analyses, subjects with severe SDB were compared with those without SDB using logistic regression analyses accounting for the complete list of confounders described earlier. Statistical analyses were performed using JMP software version 8 (SAS Institute, Cary, North Carolina).

Results

Between January 1, 2005 and January 1, 2010, 41,310 exercise tests were performed at the CVHC; 3,901 subjects underwent a first-time diagnostic polysomnogram during this time frame, and of these 2,716 had an exercise test. Reviewing these tests, we found 1,463 patients who underwent a first time diagnostic PSG within 6 months following the exercise test. After exclusions, the number of subjects analyzed was 1,424. The mean age of the subjects was 56.4 years (SD 12.4), and 75% were men. Subjects with moderate or severe SDB tended to be older with a higher BMI, and a greater proportion were male, compared with those with mild or no SDB. The most common comorbidity was hypertension (Table 1).

One hundred seven patients had a cycle ergometer test. All other patients underwent a treadmill test, using the Bruce (n = 790) or Naughton or modified Naughton (n = 78) protocols, and 499 patients had an oxygen consumption test. The most common reason for termination was the development of fatigue or other symptoms (n = 1,325). FEV1 and FVC were measured at the time of the exercise test in 198 subjects. All other exercise test measures were available for the entire cohort (n = 1,425). Maximum exercise time and METs achieved negatively correlated with AHI (all ps <0.001) and decreased across AHI categories from no SDB to severe SDB.

Overall, the mean FAC of the cohort was 76.3% (SD 22.1) and showed a progressive decline across AHI categories from no SDB to severe SDB. Univariate analysis results using AHI as a continuous variable showed that AHI negatively correlated with FAC (adj. R^2 = 0.05, p <0.001), peak HR (adj. R^2 = 0.04, p <0.001) and HRR (adj. R^2 = 0.01, p <0.001). AHI showed a positive correlation with resting SBP (adj. R^2 = 0.002, p = 0.04) and peak DBP (adj. R^2 = 0.003, p = 0.04; Table 2). One-minute postexercise HR showed a negative correlation (adj. R^2 = 0.03, p <0.001) and 3-minute postexercise DBP showed a positive

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

correlation (adj. $R^2 = 0.03$, $p = 0.03$) with AHI, but resting HR, resting DBP, peak SBP, and SBP and DBP recovery were not significantly associated with AHI (all ps >0.05).

In the multivariate analysis, AHI as a continuous variable continued to be negatively correlated with FAC (adj. $R^2 = 0.30$, $p < 0.001$). AHI also showed a negative correlation with postexercise SBP (adj. $R^2 = 0.23$, $p = 0.03$) and a positive correlation with resting DBP (adj. $R^2 = 0.09$, $p = 0.01$) and peak DBP (adj. $R^2 = 0.09$, $p = 0.04$). After accounting for confounders, AHI positively correlated with FREIVA ($p = 0.04$), VT <30 and ≥30 seconds, maximum exercise time and METs achieved, as well as symptom-limited test/patient requesting termination (all ps <0.05).

When comparing subjects with severe SDB (AHI ≥30) with those with no SDB (AHI <5) in multivariate analyses, FAC (adj. $R^2 = 0.30$, $p = 0.004$) and HRR (adj. $R^2 = 0.07$, $p = 0.05$) were lower, and resting DBP (adj. $R^2 = 0.06$, $p = 0.004$), peak DBP (adj. $R^2 = 0.09$, $p = 0.008$), and post-exercise DBP (adj. $R^2 = 0.04$, $p = 0.03$) were higher in those with severe apnea.

Multiple logistic regression analyses were repeated, including use of calcium antagonist medications (n = 126), percentage body fat calculated from skin-fold thickness measurements at the mid-triceps, mid-axillary, and suprailiac regions (data available in 1,253 subjects, mean 28.07%, SD 6.45), and waist-to-hip body ratio (Table 2) as covariates; results for FAC were again similar (adj. $R^2 = 0.29$, $p < 0.001$). Subsequent multiple logistic regression analyses were conducted including (1) only those subjects with sleep efficiency (total sleep time/total recording time) ≥85% (n = 399) to increase accuracy of diagnosis of sleep disordered breathing, although it should be noted that sleep disordered breathing may itself affect sleep efficiency, (2) only those with a diagnosis of obstructive sleep apnea (n = 1,314), and (3) baseline exercise parameters along with the other covariates. FAC, resting, and peak DBP continued to be significantly associated with AHI in each of these analyses (results not shown).

Discussion

In what is, to our knowledge, the largest study to date exploring the relation between exercise testing and untreated SDB, we have shown an independent and highly significant association between SDB severity and reduced exercise capacity (adj. $R^2 = 0.30$, $p < 0.001$), as well as an independent association between SDB severity and increased peak DBP and exercise-related ventricular arrhythmias. We have also found that, compared with patients without SDB (AHI <5), those with severe SDB (AHI ≥30) have significantly reduced FAC and HRR and higher peak and post-exercise DBP after accounting for multiple possible confounders including obesity and cardiopulmonary disease.

There are several possible mechanisms through which SDB might contribute to abnormalities in exercise testing and recovery. Dysregulation of the blood pressure response to exercise may result from the repetitive cycles of hypoxemia and reoxygenation characteristic of SDB, which have been associated with endothelial dysfunction and autonomic instability. Arrhythmias are commonly encountered during PSG in patients with

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

SDB. Similar pathways could explain the propensity for ventricular ectopy noted on exercise testing. Impaired vagal activity, increased platelet aggregation, and/or insulin resistance may contribute to impairment of left ventricular function,[5] including diastolic dysfunction.[6] Reduced exercise capacity may indicate early cardiovascular dysfunction in patients with SDB. Other factors contributing to reduced exercise capacity may include impaired muscle metabolism,[7,8] sleep fragmentation or loss,[9-11] and physical deconditioning due to daytime fatigue and/or somnolence. Although measures of daytime sleepiness were not ascertained in our study, higher sleep efficiency on polysomnography was seen to positively correlate with FAC in univariate (adj. $R^2 = 0.03$, $p < 0.001$) and multivariate (adj. $R^2 = 0.31$, $p = 0.015$) analyses, suggesting that sleep fragmentation may play a role in impaired exercise capacity seen in subjects with SDB. Increased diastolic blood pressure with exercise may relate to sympathetic induced vasoconstriction,[12] endothelial dysfunction,[13] or a blunted response to beta 2 receptor stimulation,[14] mechanisms that have been shown to occur in patients with SDB. It is worth noting the limited accuracy of BP measurements during exercise testing; however, in our study, this association persisted even after accounting for a number of confounders. The large number of subjects analyzed in this study suggests that this is a reliable result.

FEV1 and FVC were not significantly associated with FAC or other exercise test outcomes. Measurements of metabolic expired gases were not available, and FEV1 and FVC were collected only on a small number of patients at the time of exercise testing because a full cardiopulmonary exercise test was not thought to be required in other subjects based on the indication for the study and only a few patients were noted to have preexisting pulmonary disease or previous abnormal pulmonary function tests at the time of presentation to the CVHC. Hence, it is possible but seems less likely that pulmonary factors such as low lung volumes may have accounted for the negative association with FAC in this cohort.

Existing literature describing exercise testing in the setting of SDB report conflicting results. Some studies have shown a decrease in exercise capacity in patients with SDB[7,14-19]; others have shown no differences.[20-22] The study by Grote et al showed impaired HRR in patients with SDB.[14] A few studies have shown an increased diastolic blood pressure response to exercise.[14,23,24] A recent small case-control study of young men failed to show a difference in peak cycle ergometric exercise blood pressure in patients with obstructive sleep apnea (OSA) diagnosed by portable monitoring compared with subjects without OSA.[25] The large size and careful methods of our study offer robust evidence that SDB is associated with decreased exercise capacity, increased blood pressure responses with exercise, and abnormal HRR. By limiting the analysis to exercise testing that preceded in-laboratory attended polysomnography by up to 6 months, we have eliminated the confounding effects of SDB treatment (i.e., positive airway pressure therapy) on exercise outcomes. Furthermore, by analyzing the data using AHI as a continuous variable, we found a dose effect of SDB on exercise outcomes.

Previous studies are largely case-control in design, with relatively small sample sizes. Few used attended, in-laboratory PSG to confirm the presence or absence of SDB. Inconsistencies in the definition of SDB (e.g., not accounting for hypopneas) were encountered and outcomes were not assessed by severity of SDB. Furthermore, many of

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

these studies examined selected and specific subpopulations such as lean subjects or young male patients. Other limitations include variations in exercise testing protocols and lack of adjustment for BMI or other confounders such as cardiac or pulmonary diseases that are known to affect exercise testing outcomes.

In contrast, we accounted for a comprehensive set of confounders in logistic regression analyses and used echocardiogram and pulmonary function testing data where available to diagnose cardiac and pulmonary disorders in the CVHC. We employed relatively uniform exercise testing protocols at a single large center. Data on exercise times, METs achieved, and cardiac exercise testing variables was available for the entire study population, minimizing any bias due to variable data collection. Subsequent analyses were performed that included only patients with OSA and that accounted for sleep efficiency, calcium antagonist use, as well as other exercise testing variables as additional covariates.

Limitations of our study include its cross-sectional nature, possible referral bias, and a predominantly Caucasian population. It is worth noting that although results of this study may not be representative of all patients with SDB because this was a referred sample, most patients who underwent PSG in our study did have an exercise test performed. It is possible for cardiopulmonary comorbidity to have developed in between the period of exercise test and PSG; however, this would have occurred in a small proportion of the cohort, if at all, and hence is unlikely to have significantly affected the overall results. Finally, even though we tried to account for visceral obesity and fat distribution in the analyses through waist-to-hip ratio and skin-fold thickness measurements in addition to BMI, there may be unknown confounders associated with obesity may not have been controlled for.

Acknowledgments

V.K.S. has served as a Consultant for ResMed, Respicardia, Sova Pharmaceuticals, Neupro, Johnson & Johnson, and Apnex Medical; has been a principal investigator or co-investigator on research grants funded by Respironics Foundation and Sorin Corporation; and is supported by Grant NIH R01 HL065176 from the National Institutes of Health, Bethesda, Maryland. SMC has received research support from Grant HL 99534 from the National Institutes of Health, Bethesda, Maryland; ResMed Foundation; Ventus Medical; and Restore Medical. This study does not involve the use of off-label or investigational products.

The study was approved by the institutional review board, and all subjects provided research consent. No external funding sources were used.

References

1. Aron A, Zedalis D, Gregg JM, Gwazdauskas FC, Herbert WG. Potential clinical use of cardiopulmonary exercise testing in obstructive sleep apnea hypopnea syndrome. Int J Cardiol. 2009; 132:176–186. [PubMed: 19042045]
2. The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications. Westchester: American Academy of Sleep Medicine; 2007.
3. Daida H, Allison TG, Johnson BD, Squires RW, Gau GT. Further increase in oxygen uptake during early active recovery following maximal exercise in chronic heart failure. Chest. 1996; 109:47–51. [PubMed: 8549215]
4. Bruce RA, Cooper MN, Gey GO, Fisher LD, Peterson DR. Variations in responses to maximal exercise in health and in cardiovascular disease. Angiology. 1973; 24:691–702. [PubMed: 4543577]

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

5. Parati G, Lombardi C, Narkiewicz K. Sleep apnea: epidemiology, pathophysiology, and relation to cardiovascular risk. Am J Physiol Regul Integr Comp Physiol. 2007; 293:R1671–R1683. [PubMed: 17652356]
6. Otto ME, Belohlavek M, Romero-Corral A, Gami AS, Gilman G, Svatikova A, Amin RS, Lopez-Jimenez F, Khandheria BK, Somers VK. Comparison of cardiac structural and functional changes in obese otherwise healthy adults with versus without obstructive sleep apnea. Am J Cardiol. 2007; 99:1298–1302. [PubMed: 17478161]
7. Vanuxem D, Badier M, Guillot C, Delpierre S, Jahjah F, Vanuxem P. Impairment of muscle energy metabolism in patients with sleep apnoea syndrome. Respir Med. 1997; 91:551–557. [PubMed: 9415356]
8. Bonanni E, Pasquali L, Manca ML, Maestri M, Prontera C, Fabbrini M, Berrettini S, Zucchelli G, Siciliano G, Murri L. Lactate production and catecholamine profile during aerobic exercise in normotensive OSAS patients. Sleep Med. 2004; 5:137–145. [PubMed: 15033133]
9. Mougin F, Simon-Rigaud ML, Davenne D, Renaud A, Garnier A, Kantelip JP, Magnin P. Effects of sleep disturbances on subsequent physical performance. Eur J Appl Physiol. 1991; 63:77–82.
10. Hong S, Dimsdale JE. Physical activity and perception of energy and fatigue in obstructive sleep apnea. Med Sci Sports Exerc. 2003; 35:1088–1092. [PubMed: 12840627]
11. Azboy O, Kaygisiz Z. Effects of sleep deprivation on cardiorespiratory functions of the runners and volleyball players during rest and exercise. Acta Physiol Hung. 2009; 96:29–36. [PubMed: 19264040]
12. Somers VK, Dyken ME, Mark AL, Abboud FM. Sympathetic-nerve activity during sleep in normal subjects. N Engl J Med. 1993; 328:303–307. [PubMed: 8419815]
13. Carlson JT, Hedner JA, Sellgren J, Elam M, Wallin BG. Depressed baroreflex sensitivity in patients with obstructive sleep apnea. Am J Respir Crit Care Med. 1996; 154:1490–1496. [PubMed: 8912770]
14. Grote L, Hedner J, Peter JH. The heart rate response to exercise is blunted in patients with sleep-related breathing disorder. Cardiology. 2004; 102:93–99. [PubMed: 15103179]
15. Lin CC, Hsieh WY, Chou CS, Liaw SF. Cardiopulmonary exercise testing in obstructive sleep apnea syndrome. Respir Physiol Neurobiol. 2006; 150:27–34. [PubMed: 16448931]
16. Guillermo LQ, Gal TJ, Mair EA. Does obstructive sleep apnea affect aerobic fitness? Ann Otol Rhinol Laryngol. 2006; 115:715–720. [PubMed: 17076091]
17. Vanhecke TE, Franklin BA, Ajluni SC, Sangal RB, McCullough PA. Cardiorespiratory fitness and sleep-related breathing disorders. Expert Rev Cardiovasc Ther. 2008; 6:745–758. [PubMed: 18510490]
18. Ucok K, Aycicek A, Sezer M, Genc A, Akkaya M, Caglar V, Fidan F, Unlu M. Aerobic and anaerobic exercise capacities in obstructive sleep apnea and associations with subcutaneous fat distributions. Lung. 2009; 187:29–36. [PubMed: 19023624]
19. Nanas S, Sakellariou D, Kapsimalakou S, Dimopoulos S, Tassiou A, Tasoulis A, Anastasiou-Nana M, Vagiakis E, Roussos C. Heart rate recovery and oxygen kinetics after exercise in obstructive sleep apnea syndrome. Clin Cardiol. 2010; 33:46–51. [PubMed: 20063292]
20. Alonso-Fernandez A, Garcia-Rio F, Arias MA, Mediano O, Pino JM, Martinez I, Villamor J. Obstructive sleep apnoea-hypoapnoea syndrome reversibly depresses cardiac response to exercise. Eur Heart J. 2006; 27:207–215. [PubMed: 16267074]
21. Maeder MT, Wolber T, Ammann P, Myers J, Brunner-La Rocca HP, Hack D, Riesen W, Rickli H. Cardiopulmonary exercise testing in mild heart failure: impact of the mode of exercise on established prognostic predictors. Cardiology. 2008; 110:135–141. [PubMed: 17975313]
22. Rizzi CF, Cintra F, Risso T, Pulz C, Tufik S, de Paola A, Poyares D. Exercise capacity and obstructive sleep apnea in lean subjects. Chest. 2010; 137:109–114. [PubMed: 19801583]
23. Tryfon S, Stanopoulos I, Dascalopoulou E, Argyropoulou P, Bouros D, Mavrofridis E. Sleep apnea syndrome and diastolic blood pressure elevation during exercise. Respiration. 2004; 71:499–504. [PubMed: 15467328]
24. Kaleth AS, Chittenden TW, Hawkins BJ, Hargens TA, Guill SG, Zedalis D, Gregg JM, Herbert WG. Unique cardiopulmonary exercise test responses in overweight middle-aged adults with obstructive sleep apnea. Sleep Med. 2007; 8:160–168. [PubMed: 17275399]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

25. Hargens TA, Guill SG, Zedalis D, Gregg JM, Nickols-Richardson SM, Herbert WG. Attenuated heart rate recovery following exercise testing in overweight young men with untreated obstructive sleep apnea. Sleep. 2008; 31:104–110. [PubMed: 18220083]

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

Am J Cardiol. Author manuscript; available in PMC 2014 May 08.

Table 1

Characteristics by AHI category

Characteristic Mean (SD)	AHI <5 (n = 339)	AHI 5–14 (n = 468)	AHI 15–29 (n = 311)	AHI ≥30 (n = 306)	All Subjects (n = 1,424)
Age (yrs)	50.8 (12.4)	56.4 (11.4)	58.4 (12.0)	60.4 (12.1)	56.4 (12.4)
Men	64%	73%	80%	86%	75%
BMI (kg/m^2)	30.7 (7.0)	32.1 (6.8)	32.6 (6.6)	34.4 (7.2)	32.4 (7.1)
Waist/hip ratio	0.92 (0.09)	0.96 (0.09)	0.97 (0.13)	0.99 (0.09)	0.96 (0.10) n = 1,393
Sleep efficiency (%)	76.2 (14.4)	74.1 (14.4)	72.6 (15.8)	69.3 (16.7)	73.3 (15.4)
AHI (events/h)	1.8 (1.2)	8.4 (2.8)	21.0 (4.3)	54.4 (22.0)	19.5 (22.1)
Beta blockers	19.2%	27.1%	30.9%	42.2%	29.3%
Coronary artery disease	11.8%	15.2%	18.3%	25.8%	17.3%
Valvular heart disease	2.1%	2.8%	4.8%	5.2%	3.6%
Arrhythmia	13.9%	19.0%	20.3%	25.8%	19.5%
Cardiomyopathy/heart failure	7.4%	11.8%	13.2%	20.3%	12.8%
Pulmonary disease	8.8%	8.8%	5.1%	7.8%	7.8%
Diabetes	9.7%	11.3%	11.5%	19.6%	12.9%
Hypertension	28.0%	37.8%	38.9%	49.7%	38.2%
Current smoker	1.2%	3.8%	1.6%	2.6%	1.9%
Pulmonary embolism/Pulmonary artery stenosis	0.9%	2.1%	1.0%	0.7%	0.8%
Congenital heart disease	3.8%	1.9%	2.9%	4.6%	3.2%

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

Table 2

Exercise testing outcomes by AHI category

Measure Mean (SD)	AHI <5	AHI 5–14	AHI 15–29	AHI ≥30	All Subjects	Estimate	p Value*
Functional aerobic capacity (%)	80.7 (21.0)	78.8 (22.2)	76.2 (22.3)	68.0 (20.5)	76.3 (22.1)	−0.231	<0.001
Resting heart rate (beats/min)	80 (15.3)	78.9 (39.6)	76.8 (13.7)	78.6 (14.1)	78.6 (25.5)	−0.007	0.81
Peak heart rate (beats/min)	155.2 (24.9)	144.6 (29.5)	142.6 (27.5)	136.1 (28.6)	144.9 (28.6)	−0.267	<0.001
Heart rate recovery (beats/min)	19.4 (17.0)	18.4 (20.4)	17.0 (18.7)	13.0 (10.3)	17.2 (17.6)	−0.098	<0.001
Resting SBP (mm Hg)	120.2 (17.8)	121.4 (17.6)	122.2 (17.8)	123.3 (18.6)	121.7 (17.9)	0.043	0.043
Resting DBP (mm Hg)	75.7 (11.4)	76.2 (11.5)	76.5 (10.9)	76.6 (11.1)	76.2 (11.2)	0.015	0.26
Peak SBP (mm Hg)	167.4 (29.3)	166.2 (30.1)	171.1 (32.2)	164.8 (34.1)	167.3 (31.3)	−0.032	0.39
Peak DBP (mm Hg)	68.9 (17.6)	70.4 (17.5)	71.7 (17.6)	72.8 (15.2)	70.9 (17.1)	0.042	0.038
Abnormal electrocardiogram	13.0%	20.2%	24.0%	24.5%	20.2%		0.002
Any ventricular ectopy	14.5%	16.7%	18.0%	22.8%	17.8%		0.038
Frequent or repetitive ventricular ectopy	3.2%	3.2%	2.9%	2.0%	2.9%		0.74

*Univariate analysis.