

COMMENTARY

OSA, Exercise, and the Military

Comment on Powell et al. Moderate to severe obstructive sleep apnea in military personnel is not associated with decreased exercise capacity. *J Clin Sleep Med*. 2019;15(6):823–829.

Aaron B. Holley, MD

Pulmonary/Sleep and Critical Care Medicine Service, Walter Reed National Military Medical Center, Bethesda, Maryland

This seems like a slam dunk to me. Given what we know about the physiologic effects from sleep deprivation, regardless of cause, both central and peripheral fatigue should occur with exertion.¹ All things being equal then, those without obstructive sleep apnea (OSA) should outperform those who have it. OSA also has specific effects on the respiratory response to gas-exchange perturbations² and on sympathetic tone³ that might affect exercise. All that said, the relationship between OSA and physiologic variables collected during cardiopulmonary exercise testing (CPET) remains unclear.

Using different study designs and varying definitions for hypopneas, investigators have reported no relationship between OSA and VO_2peak ,^{4–6} an unadjusted correlation between apnea-hypopnea index (AHI) and VO_2peak in women but not men,⁷ or an adjusted correlation between AHI and VO_2peak .⁸ Studies reporting differences in CPET variables with OSA have generally been small and unable to separate OSA effects from deconditioning (lack of fitness).^{9,10} Although one study found OSA affects gas-exchange variables,⁶ most studies have not.^{4,7–10}

In this issue of the *Journal of Clinical Sleep Medicine*, Powell et al took another shot at dissecting the relationship between OSA and exercise.¹¹ They used the STOPBANG questionnaire to screen a cohort of current (or former) active duty service members with dyspnea for OSA. All patients had spirometry, diffusion capacity for carbon monoxide (DLCO), and CPET performed. In univariate analysis, patients with OSA had virtually identical exercise response on CPET, with no differences in VO_2peak or respiratory variables. So how do we synthesize these findings with what we already know, and can we apply them clinically to our patients?

First, we must acknowledge that the body's response to exercise integrates feedback from multiple systems.¹² At the individual level, it is difficult to isolate the specific cause for exercise termination. In research we analyze CPET data in aggregate, masking the nuance that occurs at the individual level. Second, we have to accept that any paper examining outcomes on CPET has to rigidly control for baseline fitness level. Physical activity, and lack thereof, dramatically alters VO_2 and many of the other variables collected.^{13,14} Without knowing baseline activity levels, we'll never know whether OSA directly affects the response to exercise on CPET or is simply a marker for a sedentary lifestyle. The Powell study does not help us here.

I do believe we can use this study to draw general conclusions about OSA, exercise, and active duty service members. The absence of association on CPET despite the dramatic difference in AHI between those with and without OSA (32.7 versus 5.8 events/h) casts doubt on a relationship between OSA and exercise in these patients. They also found no difference in STOPBANG scores, consistent with data from our sleep center,¹⁵ or Epworth Sleepiness Scale (ESS) scores, despite the fact that ESS was significantly elevated in both groups. So in this paper, it does not seem that an OSA diagnosis (in the severe range) is related to exercise physiology or sleep-related symptoms.

Active duty service members with sleep complaints typically have mild OSA,^{15,16} which is poorly correlated with health outcomes and symptoms,¹⁷ and comorbid disease (insomnia and insufficient sleep syndrome)¹⁸ which could be the true driver of their symptoms. The Powell paper shows the AHI in this population is not related to sleep-related symptoms or exercise, so should we really expect benefits from normalizing it using positive airway pressure (PAP)? To some degree the question is moot, because their adherence is so poor.^{16,19}

Behavior change is incredibly hard, so when we ask this of our patients we need to be confident the “juice is worth the squeeze.” PAP use entails significant behavior change, and pushing it represents an opportunity cost. For the active duty service member with milder OSA, comorbid disease, and sleep and exercise complaints, the Powell paper tells us the AHI is less important. For these patients, we should stay away from PAP and tell them to increase total sleep time and get on an exercise program. That is enough behavior change to ask of anyone.

CITATION

Holley AB. OSA, exercise, and the military. *J Clin Sleep Med*. 2019;15(6):819–820.

REFERENCES

- Hirshkowitz M. Fatigue, sleepiness, and safety: definitions, assessment and methodology. *Sleep Med Clin*. 2013;8(2):183–189.

2. Younes M. Role of respiratory control mechanisms in the pathogenesis of obstructive sleep disorders. *J Appl Physiol* (1985). 2008;105(5):1389–1405.
3. Golbin J, Somers VK, Caples SM. Obstructive sleep apnea, cardiovascular disease, and pulmonary hypertension. *Proc Am Thorac Soc*. 2008;5(2):200–206.
4. Rizzi C, Cintra F, Mello-Fujita, et al. Does obstructive sleep apnea impair the cardiopulmonary response to exercise? *Sleep*. 2013;36(4):547–553.
5. Kaleth A, Chittenden TW, Hawkins BJ, et al. Unique cardiopulmonary exercise test responses in overweight middle-aged adults with obstructive sleep apnea. *Sleep Med*. 2007;8(2):160–168.
6. Hargens T, Guill SG, Aron A, et al. Altered ventilatory responses to exercise testing in young adult men with obstructive sleep apnea. *Respir Med*. 2009;103(7):1063–1069.
7. Cintra F, Poyares D, Rizzi CF, et al. Cardiorespiratory response to exercise in men and women with obstructive sleep apnea. *Sleep Med*. 2009;10(3):368–373.
8. Beitler JR, Awad KM, Bakker JP, et al. Obstructive sleep apnea is associated with impaired exercise capacity: a cross-sectional study. *J Clin Sleep Med*. 2014;10(11):1199–1204.
9. Lin CC, Hsieh WY, Chou CS, Liaw SF. Cardiopulmonary exercise testing in obstructive sleep apnea syndrome. *Respir Physiol Neurobiol*. 2006;150(1):27–34.
10. Vanhecke T, Franklin BA, Zalesin KC, et al. Cardiorespiratory fitness and obstructive sleep apnea syndrome in morbidly obese patients. *Chest*. 2008;134(3):539–545.
11. Powell TA, Mysliwiec V, Aden JK, Morris MJ. Moderate to severe obstructive sleep apnea in military personnel is not associated with decreased exercise capacity. *J Clin Sleep Med*. 2019;15(6):823–829.
12. Weisman I, Beck KC, Casaburi R, et al. ATS/ACCP statement on cardiopulmonary exercise testing. *Am J Respir Crit Care Med*. 2003;167(2):211–277.
13. Saltin B, Blomqvist G, Mitchell JH, Johnson RL Jr, Wildenthal K, Chapman CB. Response to exercise after bed rest and after training. *Circulation*. 1968;38(5 Suppl):VII1–78.
14. Eschenbacher WL, Mannina A. An algorithm for the interpretation of cardiopulmonary exercise tests. *Chest*. 1990;97(2):263–267.
15. McMahon MJ, Sheikh KL, Andrada TF, Holley AB. Using the STOPBANG questionnaire and other pre-test probability tools to predict OSA in younger, thinner patients referred to a sleep medicine clinic. *Sleep Breath*. 2017;21(4):869–876.
16. Holley AB, Londeree WA, Sheikh KL, et al. Zolpidem and eszopiclone pre-medication for PSG: Effects on staging, titration, and adherence. *Mil Med*. 2018;183(7–8):e251–e256.
17. Chowdhuri S, Quan SF, Almeida F, et al. An official American Thoracic Society research statement: impact of mild obstructive sleep apnea in adults. *Am J Respir Crit Care Med*. 2016;193(9):e37–e54.
18. Capaldi VF, Balkin TJ, Mysliwiec V. Optimizing sleep in the military: challenges and opportunities. *Chest*. 2019;155(1):215–226.
19. Hostler JM, Sheikh KL, Andrada TF, Khramtsov A, Holley PR, Holley AB. A mobile, web-based system can improve positive airway pressure adherence. *J Sleep Res*. 2017;26(2):139–146.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication May 13, 2019

Submitted in final revised form May 13, 2019

Accepted for publication May 13, 2019

Address correspondence to: LTC(P) Aaron B. Holley, MD, Pulmonary/Sleep and Critical Care Medicine, Walter Reed National Military Medical Center, 8901 Wisconsin Ave, Bethesda, MD 20889; Email: aholley9@gmail.com

DISCLOSURE STATEMENT

The author reports no conflicts of interest.