

## COMMENTARY

## Sense and Sensitivity: Obstructive Sleep Apnea, Morning Blood Pressure, and Occult Hypertension

Commentary on Mokros et al. Morning diastolic blood pressure may be independently associated with severity of obstructive sleep apnea in non-hypertensive patients: a cross-sectional study. *J Clin Sleep Med.* 2017;13(7):905–910.

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Observations have raised the suspicion of a relationship between obstructive sleep apnea (OSA) and systemic hypertension for many years. More than 30 years ago, Kales et al.<sup>1</sup> noted that patients with more severe OSA seemed to have higher blood pressure. Shepard<sup>2</sup> demonstrated an increase in systemic blood pressure (BP) with apnea termination via radial artery catheterization during polysomnography. Ten years later, Somers et al.<sup>3</sup> demonstrated that sympathetic activity increased along with an increase in systemic BP in OSA subjects during polysomnography, and that the increased sympathetic activity was present during wakefulness. This improved with continuous positive airway pressure. Suzuki et al. had earlier shown that treatment of OSA with continuous positive airway pressure could normalize hypertension.<sup>4</sup> Worsnop et al.<sup>5</sup> demonstrated that those with an apnea-hypopnea index (AHI) > 15 events/h had a significantly higher risk of having systemic hypertension in cross-sectional studies. Peng et al.<sup>6</sup> showed that rats subjected to 10 days of chronic intermittent hypoxia had long-term facilitation of the carotid body. This may lead to persistent reflex activation of sympathetic nerve activity and hypertension. Because of the confounding effect of obesity, it has been difficult to definitively confirm a causal relationship between OSA and hypertension, but the bulk of data and most pointedly the Wisconsin Sleep Cohort Study<sup>7</sup> results indicate a relationship. However, the “negative” results of the Sleep Heart Health Study<sup>8</sup> still need to be explained. It should be noted that in those with AHI > 30 events/h and BMI < 27 kg/m<sup>2</sup>, there was increased incident hypertension. One explanation for the difficulty in confirming and explaining the relationship between OSA and systemic hypertension may be in the flawed way in which the diagnosis of hypertension has been made: by measuring daytime, awake BP. We now know from the MAPEC<sup>9</sup> study that the only BP measurements that actually represent a prognostic marker of cardiovascular morbidity and mortality are those taken at night during sleep. This is such an important new development that it has caused the United States Preventive Services Task Force<sup>10</sup> to recommend that 24-hour ambulatory blood pressure monitoring (ABPM) should be the reference standard for confirming office BP screening, thus preventing the unnecessary treatment

of those misdiagnosed with hypertension by daytime measurements. Just as important, ABPM allows the diagnosis of masked hypertension in those who only have hypertension during sleep or in whom BP fails to lower during sleep (“non-dippers”).<sup>11</sup> Indeed Baguet et al.<sup>12</sup> used ABPM to study 130 patients with a new diagnosis of OSA and ABPM and found that 30% had masked hypertension (normal clinic BP with abnormal ABPM). They found no correlation between AHI and office BP, but a significant correlation between AHI and nocturnal diastolic BP as well as day-night diastolic BP decrease. When ABPM was used in the Wisconsin Sleep Cohort Study, they found dose-response increase in developing nondipping BP with severity of OSA.<sup>13</sup> Now Mokros et al.<sup>14</sup> have reported on the results of a retrospective study of BP taken by standard sphygmomanometry (1) at the first physician encounter, (2) the night of polysomnography, and (3) the morning after polysomnography in the sitting position. Subjects were not known to have hypertension. They found that diastolic BP, but not systolic BP, was higher in the morning after polysomnography, with a higher AHI in a linear regression model. In view of all we have learned from past investigations and new revelations about the importance of sleep BP measured by ABPM, rather than daytime office BP, it is intriguing to postulate whether a simple and inexpensive morning BP measurement might constitute an effective screening mechanism to indicate who warrants ABPM and a definitive diagnosis of sleep hypertension.

## CITATION

Castriotta RJ. Sense and sensitivity: obstructive sleep apnea, morning blood pressure, and occult hypertension. *J Clin Sleep Med.* 2017;13(7):861–862.

## REFERENCES

1. Kales A, Bixler EO, Cadieux RJ, et al. Sleep apnoea in a hypertensive population. *Lancet.* 1984;2(8410):1005–1008.

2. Shepherd JW Jr. Gas exchange and hemodynamics during sleep. *Med Clin North Am.* 1985;69(6):1243–1264.
3. Somers VK, Dyken ME, Clary MP, Abboud FM. Sympathetic neural mechanisms in obstructive sleep apnea. *J Clin Invest.* 1995;96(4):1897–1904.
4. Suzuki M, Otsuka K, Guilleminault C. Long-term nasal continuous positive airway pressure can normalize hypertension in obstructive sleep apnea patients. *Sleep.* 1993;16(6):545–549.
5. Worsnop CJ, Naughton MT, Barter CE, Morgan TO, Anderson AI, Pierce RJ. The prevalence of obstructive sleep apnea in hypertensives. *Am J Respir Crit Care Med.* 1998;157(1):111–115.
6. Peng YJ, Overholt JL, Kline D, Kumar GK, Prabhakar NR. Induction of sensory long-term facilitation in the carotid body by intermittent hypoxia: implications for recurrent apneas. *Proc Natl Acad Sci.* 2003;100(17):10073–10078.
7. 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(19):1378–1384.
8. O'Connor GT, Caffo B, Newman AB, et al. Prospective study of sleep-disordered breathing and hypertension: the Sleep Heart Health Study. *Am J Respir Crit Care Med.* 2009;179(12):1159–1164.
9. Hermida RC, Ayala DE, Mojón A, Fernández JR. Decreasing sleep-time blood pressure determined by ambulatory monitoring reduces cardiovascular risk. *J Am Coll Cardiol.* 2011;58(11):1165–1173.
10. Piper MA, Evans CV, Burda BU, Margolis KL, O'Connor E, Whitlock EP. Diagnostic and predictive accuracy of blood pressure screening methods with consideration of rescreening intervals: a systematic review for the U.S. Preventive Services Task Force. *Ann Intern Med.* 2015;162(3):192–204.
11. Hermida RC, Ayala DE, Smolensky MH, Fernández JR, Mojón A, Portaluppi F. Sleep-time blood pressure: unique sensitive prognostic marker of vascular risk and therapeutic target for prevention. *Sleep Med Rev.* 2017;33:17–27.
12. Baguet JP, Levy P, Barone-Rochette G, et al. Masked hypertension in obstructive sleep apnea syndrome. *J Hypertens.* 2008;26(5):885–892.
13. Hia KM, Young T, Peppard PE, Szklo-Coxe M, Stubbs M. Longitudinal association of sleep disordered breathing and non-dipping of nocturnal blood pressure in the Wisconsin Sleep Cohort Study. *Sleep.* 2008;31(6):795–800.
14. Mokros Ł, Kuczyński W, Franczak Ł, Białasiewicz P. Morning diastolic blood pressure may be independently associated with severity of obstructive sleep apnea in non-hypertensive patients: a cross-sectional study. *J Clin Sleep Med.* 2017;13(7):905–910.

## SUBMISSION & CORRESPONDENCE INFORMATION

**Submitted for publication June 1, 2017**

**Submitted in final revised form June 1, 2017**

**Accepted for publication June 9, 2017**

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## DISCLOSURE STATEMENT

Dr. Castriotta has served on the Genentech Speakers Bureau (for pulmonary fibrosis) and has received research support from Reata (for mitochondrial myopathy). He reports no conflicts of interest with the subject or content of this commentary.