Journal of the American Society of Hypertension 11(2) (2017) 122–129

Review Article

Sleep, insomnia, and hypertension: current findings and future directions

S. Justin Thomas, PhD[a,*] and David Calhoun, MD[b]

[a]*Department of Epidemiology, University of Alabama at Birmingham, Birmingham, AL, USA; and*
[b]*Department of Medicine, Division of Cardiovascular Disease, University of Alabama at Birmingham, Birmingham, AL, USA*

Manuscript received October 10, 2016 and accepted November 26, 2016

Abstract

Blood pressure (BP) varies over 24 hours. During normal sleep, BP typically decreases by 10% or more. Research suggests that disordered sleep, particularly sleep deprivation and obstructive sleep apnea, is associated with increased BP and risk of hypertension. Less is known about the relationship between insomnia and hypertension. Population-based studies have reported an association between insomnia symptoms and both prevalent and incident hypertension, particularly in the context of short sleep duration. Furthermore, a number of mechanisms have been proposed to explain the relationship between insomnia and hypertension. However, few studies have examined these proposed mechanisms, and even fewer clinical trials have been conducted to determine if improved sleep improves BP and/or reverses a nondipping BP pattern. Methodological concerns, particularly with respect to the diagnosis of insomnia, no doubt impact the strength of any observed association. Additionally, a large majority of studies have only examined the association between insomnia symptoms and clinic BP. Therefore, future research needs to focus on careful consideration of the diagnostic criteria for insomnia, as well as inclusion of either home BP or ambulatory BP monitoring. Finally, clinical trials aimed at improving the quality of sleep should be conducted to determine if improved sleep impacts 24-hour BP. J Am Soc Hypertens 2017;11(2):122–129.

Keywords: Ambulatory blood pressure monitoring; hypertension; insomnia.

Sleep and Blood Pressure

Blood pressure (BP) is known to vary over 24 hours and differs when measured inside versus outside the clinic setting. BP decreases by approximately 10%–20% during sleep, increases just prior to waking, and peaks shortly after waking (Figure 1).[1] The decrease in BP during sleep is termed ‘‘dipping,’’ and the increase in BP upon waking is termed ‘‘morning surge.’’[2] The mechanism underlying circadian variation in BP is attributed to circadian clock genes (ie, CLOCK, BMAL1, Per, and Cry) that orchestrate regulation of BP over 24 hours, in part via output from the suprachiasmatic nucleus to the sympathetic nervous system and hypothalamic–pituitary–adrenal (HPA) axis.[3] Knockout models of circadian clock genes result in a reduction and/or elimination of circadian variation in BP.[4–7] Furthermore, repeated dosing of melatonin, a hormone involved in the regulation of the sleep/wake cycle, has been shown to reduce systolic and diastolic BP (SBP and DBP, respectively) as well as improve nocturnal SBP and DBP dipping in patients with hypertension. The observed improvements in sleep and BP occurred independently of each other and were attributed to restoration of circadian function.[8] In addition to these endogenous mechanisms, cognitive and behavioral factors also play a role in governing BP levels and patterns.[9]

A nondipping BP pattern is defined as a nocturnal decline in SBP <10% from daytime SBP and is associated with increased risk for cardiovascular morbidity and mortality.[10–13] Although nondipping may occur for both SBP and

Conflict of interest: The authors have no conflicts of interest to disclose.

This work was supported by NIH grant R01 HL113004 and grant 15SFRN2390002 from the American Heart Association.

*Corresponding author: S. Justin Thomas, PhD, Department of Epidemiology, University of Alabama at Birmingham, LHL 410, 1720 2nd Ave. S., Birmingham, AL 35294-0013, USA. Tel: 205-996-0142; fax: 205-975-7058.

E-mail: sjthomas@uab.edu

1933-1711/$ - see front matter
http://dx.doi.org/10.1016/j.jash.2016.11.008

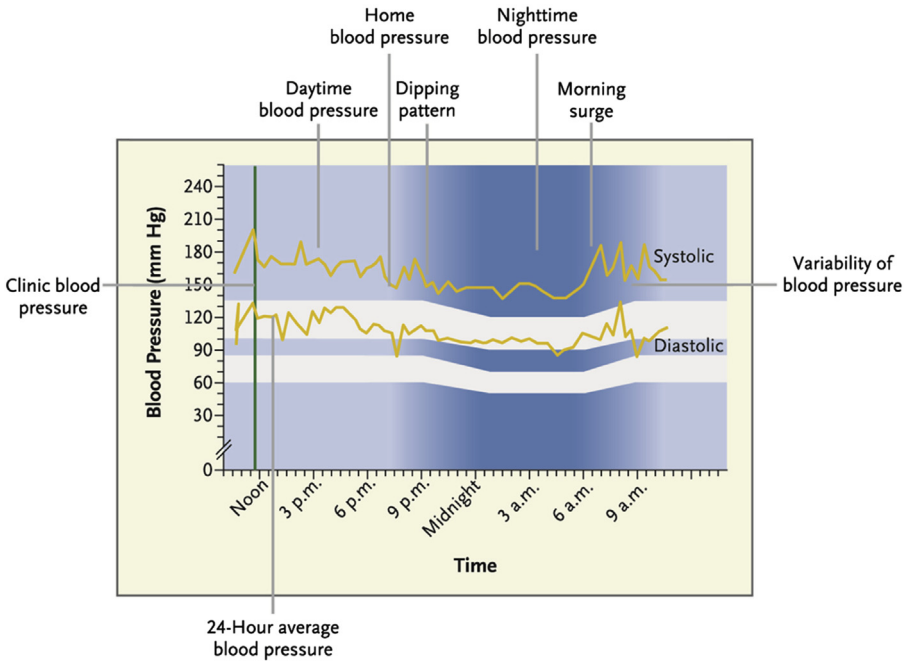

Figure 1. Systolic and diastolic blood pressure over 24 hours. Figure is from Pickering TG, Shimbo D, Haas D. Ambulatory blood-pressure monitoring. *N Engl J Med*. 2006; 354(22):2368–2374.

DBP, a nondipping BP pattern typically refers to SBP alone. An extreme form of nondipping is "reverse dipping," wherein SBP during sleep is higher than during wakefulness. The mechanisms underlying a nondipping BP pattern are not fully understood, although autonomic dysregulation[14,15] as well as alterations in the HPA system,[16] renin-angiotensin-aldosterone system,[17] and kidney function/sodium handling[18–20] have all been implicated.[21,22] The physiological underpinnings of a nondipping BP pattern may be mediated, in part, by the presence of sleep disorders such as obstructive sleep apnea (OSA). For example, untreated OSA has been associated with increased sympathetic activation that may, in turn, result in increased BP both during sleep and wakefulness.[23–25]

Nocturnal BP has been shown to be a better predictor of cardiovascular outcomes when compared with daytime BP.[26–28] Sleep disorders that affect nocturnal BP are associated with an increased risk for incident hypertension and cardiovascular morbidity and mortality. The relationship between OSA and hypertension is well documented.[29–31] However, less is known about the relationship between insomnia and hypertension. Lanfranchi et al[32] found that normotensive individuals with chronic insomnia had higher nighttime SBP and blunted day-to-night SBP compared with normotensive individuals without chronic insomnia. These increases in SBP and reduced SBP dipping were associated with increased cortical activation as measured by electroencephalography. The mechanisms underlying the association between insomnia and hypertension may be similar to those found in OSA.[33]

Sleep Duration and Hypertension

Short sleep duration is consistently associated with both prevalent and incident hypertension in young and middle-aged adults.[34–38] For example, an analysis of the Sleep Heart Health Study reported that a sleep duration of less than 6 hours was associated with prevalent hypertension.[34] Separately, an analysis of the National Health and Nutrition Examination Survey reported that a sleep duration of 5 hours or less was associated with an increased risk for incident hypertension.[34,35] The relationship between a short sleep duration and hypertension appears to be particularly strong for premenopausal women.[36,39] An analysis of the Sleep Heart Health Study also reported an association between short sleep duration and prevalent hypertension in older adults; however, other studies have reported that a short sleep duration is not associated with hypertension in this population.[37,40] In one of the studies that found no association, hypertension was defined as SBP/DBP $\geq$ 160/100 mmHg, thus likely attenuating any relationship that might have existed.[40]

Sleep Quality and Hypertension

Poor sleep quality, like short sleep duration, has been associated with both prevalent and incident hypertension.[41–44] Fiorentini et al[41] reported that hypertension was more prevalent in "poor sleepers" compared with "good sleepers" (87.1% vs. 35.1%, respectively) as measured by the Pittsburg Sleep Quality Index global score. Rod et al[42]