Background: Blood pressure (BP) dips at night during sleep in healthy individuals but in disturbed sleep, dipping is blunted. However, the impact of chronic insufficient sleep duration, with limited intermittent recovery sleep, on BP dipping is not known. The objective of this study was to examine, in a controlled experimental model, the influence of chronic sleep restriction on BP patterns at night and during the day.

Method: In a highly controlled 22-day in-hospital protocol, 45 healthy participants (age 32 ± 2 years; BMI 24 ± 1 kg/m²; 22 men and 23 women) were randomly assigned to one of two conditions: repeated sleep restriction (4 h of sleep/night from 0300 to 0700 h for three nights followed by recovery sleep of 8 h, repeated four times in succession) or a control group (8 h/night from 2300 to 0700 h).

Results: Beat-to-beat BP and polysomnography were recorded and revealed that sleep-associated DBP dipping was significantly blunted during all four blocks of sleep restriction (P = 0.002). Further, DBP was significantly increased for the whole day during the first, second, and fourth block of sleep restriction (all P < 0.01), and SBP was significantly increased for the whole day during the first block of sleep restriction.

Conclusion: Repeated exposure to significantly shortened sleep blunts sleep-associated BP dipping, despite intermittent catch-up sleep. Individuals frequently experiencing insufficient sleep may be at increased risk for hypertension due to repetitive blunting of sleep-associated BP dipping, and resultant elevations in average circadian BP.

Keywords: autonomic nervous system, blood pressure, circadian, blood pressure dipping, diurnal, heart rate, sleep deprivation, sodium excretion

Abbreviations: BL, baseline; BP, blood pressure; CRC, clinical research center; CVD, cardiovascular disease; HR, heart rate; N1, N2, N3, stages 1–3 in nonrapid eye movement sleep; Rec, recovery; REM, rapid eye movement; SE, sleep efficiency; TST, total sleep time

INTRODUCTION

Several lines of evidence suggest that insufficient sleep contributes to the development of cardiovascular disease (CVD) risk factors and stroke [1]. Longitudinal studies including at least 5 years of follow-up have reported significant associations between short sleep and elevated blood pressure (BP) [2,3], and experimental studies have reported elevated BP following acute total sleep deprivation [4,5] and partial sleep restriction [6].

Early studies using intra-arterial ambulatory BP monitoring found that nocturnal BP significantly decreases during sleep [7] and surges in response to morning awakening [8]. A recent systematic review for the US Preventive Services Task Force suggested that ambulatory BP measurement (noninvasively measured across day and night) is a better predictor of long-term cardiovascular outcomes than office BP and is a reference standard for evaluating/screening for high BP in adults [9]. The average BP decline from wake to nocturnal BP is 10–20%, which is known as normal dipping. Reduction in nocturnal BP dipping is a stronger predictor of cardiovascular adverse events than daytime BP, even after adjustment for other covariates [10]. Furthermore, poor sleep can blunt BP dipping [11], implicating sleep as a mechanism in BP homeostasis. However, there are no studies that have investigated the effects of chronic short sleep, free of sleep disorders, on BP.

Due to busy schedules, it is not unusual to cut back on sleep during the week and to catch up on weekends, or days off [12]. Whether or not such ‘recovery’ sleep on the weekend can compensate for the adverse effects of short sleep on BP dipping is unknown. The purpose of this study was to examine the influence of repeated blocks of sleep restriction on BP patterns at night and during the day. Blocks of sleep restriction consisted of three consecutive nights of short sleep followed by one recovery night of 8 h of sleep opportunity. We repeated this schedule pattern four times, producing four blocks of repeated exposure to sleep restriction. This model mimics the real-life sleep schedule of many people, with short sleep duration for consecutive nights followed by insufficient recovery sleep, before the pattern is repeated. We hypothesized that sleep...
plays an important role in BP regulation and predicted that sleep-associated nocturnal BP dipping would be blunted during sleep restriction and would fail to adapt to repetitive blocks of restricted sleep.

**METHODS**

**Participants**

The current study was approved by the Beth Israel Deaconess Medical Center Institutional Review Board, and all participants provided informed consent. Forty-five medically screened, healthy individuals (22 men and 23 women) between the ages of 21 and 53 years participated, and 43 completed. One man withdrew after study start because it was discovered that he was wearing a nicotine patch, one woman withdrew due to illness.

Participants underwent a complete medical history and physical screening, including blood chemistry. This screening was done to rule out participants with psychiatric, neurological, pain-related, immune disorders, diabetes mellitus, chronic renal disease, significant allergy, and CVD. Overnight sleep studies were performed to rule out sleep disorders (respiratory disturbance index of >5 events/h on polysomnographic sleep study, leg movements with arousal >10 events/h) before being enrolled for the in-hospital phase of the protocol. Participants were excluded from the study if they showed signs of acute or chronic disease.

All female participants reported regular menstrual cycles without significant discomfort during premenses/menses. Use of regular medications with the exception of birth control was exclusionary. Two weeks of actigraphic data along with sleep diaries were collected to ensure that the participants had a usual daily sleep duration between 7 and 9 h, sleep efficiency more than 80%, and habitual sleep periods beginning within 1 and 1.5 h of 2300 h (the scheduled normal bed time during the study).

**Experimental design**

The 22-day protocol (Fig. 1) was carried out in the clinical research center (CRC). Following 2 days of adaptation and 1 day of baseline recording with 8 h sleep opportunity from 2300 to 0700 h, participants were randomly assigned to an experimental group involving four blocks of sleep restriction or to an 8 h/night sleep control group. In the sleep restriction group, participants slept 4 h/night from 0300 to 0700 h for three consecutive nights and then were permitted a night of 8 h recovery sleep. This pattern was repeated four times; and when completed there were two additional nights of 8 h sleep scheduled. Participants remained in bed in semisupine position with dimmed light (<20 lx) between 2300 and 0300 h during sleep restriction, no food or water was allowed, with the aim to minimize differences between conditions, except for sleep opportunity. Each participant was accompanied by a research assistant who helped maintain their wakefulness and social interaction, as well as ensure data quality and protocol compliance. During the sleep deprivation hours between 2300 and 0300 h in the SR group, participants lost the ability to maintain wakefulness for only very short times (1–5 min total, per 1 h block), and this amount did not change across the four cycles (data not shown). The participants’ usual activities were talking, reading, writing, watching

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**FIGURE 1** Protocol summary. Gray bars indicate the scheduled sleep periods, and hatched bars with vertical lines represent the wake and sleep periods with intensive physiological monitoring (polysomnography, single channel ECG, and beat-to-beat blood pressure recordings). Following three nights of 8 h sleep, participants were randomly assigned to sleep restriction group (4 h group) or sleep control group (8 h group). In the 4 h group, participants slept 4 h (0300 to 0700 h) for three consecutive nights, followed by one night of 8 h sleep, the block repeated four times, with an additional two nights of 8 h sleep at the end. In the 8 h group, participants were permitted to sleep normally (2300 to 0700 h) throughout the study.
TV/movies, playing video/board games, and so on during their 22-day stay. In the sleep control group, sleep was permitted between 2300 and 0700 h throughout the 22-day stay.

On the morning following the first two nights of restricted sleep, participants were instrumented with beat-to-beat BP and polysomnography, and the equipment was removed the next morning after at least 30 min of wakefulness, resulting in up to approximately 24 h of continuous beat-to-beat BP recordings. Weighed diets (designed to maintain participant’s body weight, controlled for fats, carbohydrates, sodium, and potassium) were provided at standard times (breakfast at 0730 h, lunch at 1230 h, and supper at 1830 h) along with a beverage. Additional water intake was provided in hourly aliquots based on weight-adjusted norms and adjusted in the first two adaptation days to accommodate individual preference. Fluid intake and output were measured and tracked. Room temperature was maintained at participant’s preferred level during the day, and dropped by 2 °C at night. Outside of days when participants were instrumented for physiological recordings (recording days), they went on hourly walks to maintain their daily physical activity levels. In addition, three times per week they used the hospital fitness center.

**Measurements**

**Actigraph and sleep diary**

An Actiwatch (Actiwatch 2; Respironics Inc., Murrysville, Pennsylvania, USA) and a sleep diary were given to the participant after initial screening. Fourteen days of sleep data were collected to evaluate participants’ habitual bedtimes and sleep times according to inclusion criteria.

**Subjective ratings**

During wake time, participants rated their subjective sleepiness and a few other feeling-state parameters such as appetitive hunger and thirst, every 2 h on computerized visual analog scales. In the sleep restriction group, participants continued to rate their subjective sleepiness and other states, every 2 h during the sleep deprivation period (2300 to 0300 h) as well.

**Beat-to-beat blood pressure**

The arterial waveform in a finger of the nondominant hand was obtained beat-to-beat by digital photoplethysmography (Portapres system; Finapres Medical Systems; Amsterdam, Netherlands); this noninvasive method was chosen to minimize sleep disturbance. Two small finger cuffs were placed on the fingers (designated to maintain participant’s body weight, controlled for fats, carbohydrates, sodium, and potassium) were provided at standard times (breakfast at 0730 h, lunch at 1230 h, and supper at 1830 h) along with a beverage. Additional water intake was provided in hourly aliquots based on weight-adjusted norms and adjusted in the first two adaptation days to accommodate individual preference. Fluid intake and output were measured and tracked. Room temperature was maintained at participant’s preferred level during the day, and dropped by 2 °C at night. Outside of days when participants were instrumented for physiological recordings (recording days), they went on hourly walks to maintain their daily physical activity levels. In addition, three times per week they used the hospital fitness center.

**Polysomnographic recording**

Sleep was recorded using the Embla N7000 system (Medcare US, Buffalo, New York, USA) on the screening night to ensure that participants were free of sleep disorders (sleep apnea and restless legs syndrome). The Embla N7000 system was also used to collect sleep data on recording days during the study period (Fig. 1) and on an additional final night of sleep before leaving the study (data not shown). Sleep and wake periods were recorded and manually scored for sleep, according to standardized procedures [15]. ECG electrodes were placed beneath each clavicle.

**Urinary sodium excretion**

Urine samples were collected during the night period (2300 to 0700 h) on heavy recording days. The total urine volumes for night periods were recorded by the nurses. Urine sodium concentrations were measured by ion selective electrode in a Cobas 6000 chemistry analyzer from Roche Diagnostics (Indianapolis, Indiana, USA). Measurements were performed by a CLIA-certified clinical laboratory, and interassay coefficient of variation was 2.7%. For each participant, the sodium excretion value (mmol) was calculated as the concentration of sodium in the urine (mmol/l) multiplied by the urinary volume.

**Data analysis**

**Beat-to-beat blood pressure**

Beat-to-beat BP signals acquired by the Portapres system were fed directly into the Embla system, in which the data were transformed to European Data Format and exported to LabChart (LabChart 7; ADInstruments Co., Dunedin, New Zealand) for off-line analysis. Beat-to-beat BP recordings up to 24 h were successfully obtained from 43 participants. Eight participants were missing data on 1 day, five participants were missing data on 2 days and one participant was missing data on 3 days’ recordings (due to malfunctioning devices), this reflects approximately 8% missing data. Due to varied BP start time in the morning, 20-h BP data are presented from 1100 to 0700 h. Data for each participant were first averaged for each hour, and then the hourly average data entered into a mixed model analysis. Data are presented as estimated mixed model average SBP and DBP.

**Beat-to-beat heart rate**

Heart rate (HR) was acquired on the Embla N7000 system, data were transformed to the European data format and imported to LabChart for off-line analysis. Seven participants had one day (out of six heavy recording days), or less than 3%, of missing HR data due to technical problems.

**Nocturnal dip**

For direct comparison, nocturnal BP dipping was calculated using the first 4 h of the sleep period (i.e. 2300 to 0300 h for the 8 h sleep and 0300 to 0700 h for the 4 h sleep condition). The percentage change in first 4 h sleep BP was calculated as $100 \times (\text{wake BP} - \text{sleep BP})/\text{wake BP}$, the wake time data were analyzed from 1100 to 2300 h (12 h). Nocturnal dipping data were analyzed from 43 participants.
Results

Demographics

Forty-three healthy participants (21 men and 22 women) between the ages of 21 and 52 years completed the trial and were included in the analyses. BMI (kg/m²) averaged 24.1 ± 2 kg/m² for both groups, and similar groups (all ≥ 0.20). Participants in both control groups had almost identical habitual sleep duration (7.8 h for both groups) and sleep efficiency or percentage of time spent asleep after sleep began and before final awakening in the morning (83 and 85% for restricted and control condition respectively). Sleep duration and sleep efficiency were not correlated to daytime nor nighttime BP changes (data not shown). There were no differences between the sleep restriction and sleep control groups in terms of daily intake and output measures.

### Table 1. Daily water and sodium intake, night-time urinary sodium excretion, subjective sleepiness and sleep parameters

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (N = 22; 11 women)</th>
<th>Restriction (N = 21; 11 women)</th>
<th>Interaction P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water intake (24h) (ml)</td>
<td>2456 ± 109</td>
<td>2428 ± 109</td>
<td>0.77</td>
</tr>
<tr>
<td>Sodium intake (24h) (mg)</td>
<td>2462 ± 179</td>
<td>2519 ± 179</td>
<td>0.12</td>
</tr>
<tr>
<td>Night-time sodium excretion (mmol)</td>
<td>24 ± 3</td>
<td>21 ± 3</td>
<td>0.05</td>
</tr>
<tr>
<td>Daytime sleepiness (mmol)</td>
<td>20 ± 4</td>
<td>19 ± 4</td>
<td>0.13</td>
</tr>
<tr>
<td>Night-time sleepiness</td>
<td>20 ± 4</td>
<td>19 ± 4</td>
<td>0.32</td>
</tr>
<tr>
<td>TST (min)</td>
<td>389 ± 8</td>
<td>391 ± 8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>SE (%)</td>
<td>81 ± 2</td>
<td>82 ± 2</td>
<td>0.02</td>
</tr>
<tr>
<td>N1 (min)</td>
<td>35 ± 2</td>
<td>35 ± 2</td>
<td>0.13</td>
</tr>
<tr>
<td>N2 (min)</td>
<td>201 ± 7</td>
<td>201 ± 7</td>
<td>0.01</td>
</tr>
<tr>
<td>N3 (min)</td>
<td>72 ± 5</td>
<td>72 ± 5</td>
<td>0.02</td>
</tr>
<tr>
<td>REM (min)</td>
<td>30 ± 6</td>
<td>30 ± 6</td>
<td>0.24</td>
</tr>
</tbody>
</table>

N1, N2, N3, stage 1–3 in nonrapid eye movement sleep; REM, rapid eye movement sleep; TST, total sleep time.

All data were analyzed statistically using software IBM SPSS 22.0. An independent t test was used to compare group means with the appropriate error terms. Significant difference was considered significant at P value of 0.05 (two-tailed). We also considered the results as significant in cases that P value was < 0.01.
consumed protein, fats, carbohydrates (presented in both grams and percentages), daily potassium, or sodium.

**Hemodynamic parameters**

**Blood pressure dipping**

Figure 2 shows nocturnal sleep-associated BP dipping. The top panel summarizes the percentage dipping for SBP (a) and DBP (b) and indicates the blocks with significant blunting. SBP and DBP wake (hatched bars) and sleep (solid bars) periods are shown from baseline through the blocks of sleep restriction and recovery sleep (a2 and b2); and on the days for the sleep control condition (a3 and b3). There was a significant interaction effect (blocks × time) for DBP in the sleep restriction group ($P = 0.002$) and a trend for SBP ($P = 0.090$). DBP dipping was significantly attenuated during sleep restriction blocks compared with baseline, for every block of sleep restriction ($P < 0.05$ for each block compared with baseline). In the sleep control group, dipping was unchanged throughout the study (block × time interaction, $P = 0.18$).

**Daily averages**

Figure 3 depicts the average circadian SBP, DBP, and HR changes associated with sleep conditions. There was a significant interaction (condition × block, $P < 0.001$) for SBP, showing an overall elevation in sleep restriction compared with the control sleep condition. This elevation over baseline in SBP was seen in the first block of sleep restriction and decreased below baseline during recovery sleep.

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*FIGURE 2* SBP (a) and DBP (b) dipping in sleep restriction group (a1 and b1). The blood pressure dipping data plotted on the (a1) and (b1) were calculated from the means of blood pressure summarized in the bar graphs in the middle panels (a2 and b2). *Significantly different ($P < 0.05$) from baseline in the restriction group. Bar graph: wake (1100 to 2300 h) blood pressure (hatched bars) and first 4 h of sleep blood pressure (solid bars) at baseline, first block of interventions to fourth block and recovery in sleep restriction group (a2 and b2) and sleep control group (a3 and b3). The first 4 h of sleep SBP and DBP data were included (2300 to 0300 h for control and 0300 to 0700 h for the sleep-restricted condition). Mixed model analysis found a significant interaction (block × time, $P = 0.002$) effect in DBP and a trend in SBP (block × time, $P = 0.09$).
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FIGURE 3 Changes (delta) in daily average (20 h) SBP (a), DBP (b), and heart rate (c) in four blocks of sleep control/restriction and recovery compared with baseline. Mixed model analyses found significant interactions for each of these models (P < 0.01); individual block data are compared with baseline and significance (P < 0.05) indicated in the figure (1).

(indicated by asterisks in Fig. 3a). DBP (Fig. 3b) also showed a condition by block interaction (P = 0.001). In the sleep restriction group, DBP was significantly increased over baseline during the first, second, and fourth blocks of restriction (indicated by asterisks in Fig. 3b). Similarly, HR (Fig. 3c) showed a significant interaction (condition x block, P < 0.001) elevated over baseline in the first block, whereas in the control condition, HR stayed significantly lower than it had been at baseline, throughout the study days, for the remainder of the protocol.

Figure 4 depicts the hourly averaged DBP during baseline, four blocks, and recovery in the sleep restriction group. There was a significant interaction effect (between block and time of the day, P = 0.022) in the sleep restriction group. In sleep restriction, DBP maintained wake period levels during 4 h of sleep deprivation at night (between 2300 and 0300 h) compared with baseline and recovery. SBP showed similar changes (data not shown, block x time of the day interaction, P = 0.006).

Sleep electrophysiology

As expected, total sleep time was significantly decreased (condition x block interaction, P < 0.001) by the sleep restriction protocol, and sleep efficiency was increased (condition x block interaction, P = 0.022). There was no change in deep slow wave sleep (N3 in minutes), despite sleep restriction (condition x block interaction, P = 0.243), but there was a reduction in other stages of sleep and of wakefulness during sleep (Table 1).

Despite a substantial amount of sleep loss, wakefulness was well maintained during the vigil hours between 2300 and 0300 h in the sleep restriction group. Participants lost the ability to maintain wakefulness and lapsed into 30-s epochs of sleep, for only very short times (1–5 min total, per 1 h block), and this amount did not change across the four blocks (data not shown).

Subjective estimations of appetitive and feeling states

Subjective sleepiness ratings during the sleep deprivation night-time period were close to double the daytime ratings (Table 1). Daytime subjective sleepiness ratings (excluding nocturnal periods) significantly increased throughout the four blocks in the sleep restriction group, but not sleep control (condition x block interaction, P = 0.006). Subjective thirst and self-reported appetite for salty food as assessed during the first test of the morning following heavy recording days did not show any changes throughout the protocol in either group (data not shown).

DISCUSSION

This highly controlled physiological study on the effects of prolonged exposure to insufﬁcient sleep with only occasional opportunities to catch up on lost hours resulted in two important ﬁndings concerning the role of sleep in...
autonomic and cardiovascular regulation in healthy adults. First, chronic, repetitive sleep restriction to 4 h in the latter part of the night blunts the BP dipping that occurs during sleep, without signs of adaptation over time. Second, shortened sleep affects 24-h BP through relative elevations in sleep-associated values, without affecting daytime pressures. These findings suggest that one mechanism whereby insufficient sleep duration may lead to increased risk for CVDs is by blunting BP dipping.

Observational studies have examined the association between poor sleep and BP dipping and reported a significant correlation between lower sleep efficiency and blunted BP dipping [16–18]. In the current study, electrophysiologically defined sleep quality was excellent during restricted sleep, yet BP dipping was blunted. Sleep stages themselves are known to modulate BP. Direct recordings from human muscle sympathetic nerves have shown a pronounced decrease in firing from wake to slow wave sleep, accompanied by lowered BP and HR [19]. This is in contrast with rapid eye movement (REM) sleep, in which BP and HR are closer to levels seen in waking, and sympathetic nerve activity is at its daily high [19]. The fact that this study found reduced sleep-associated BP dipping, in spite of increased sleep efficiency, slow wave sleep (N3)/%, and REM%/ during sleep restriction, supports the hypothesis that sleep is an important mechanism of BP control.

As participants were closely monitored throughout the protocol, there was minimal lapsing into sleep during the deprivation periods. The only measurement that was differentially affected during the night, including subjective indices, was sodium excretion. Although increased sodium excretion has been reported in total sleep deprivation studies of 24 h of continuous wakefulness [20,21], this is the first to show this increase in chronic repeating sleep restriction, and it is of note that this change became significant only after repeated blocks of restricted sleep challenge.

Plasma renin activity is known to increase in non-REM, particularly slow wave sleep, and decrease in REM sleep [22,23], and patients with fragmented sleep due to sleep apnea have reduced BP dipping, increased nocturnal urinary output and urinary sodium excretion, increased atrial natriuretic peptide release, and decreased renin–angiotensin–aldosterone activity [24,25]. Although a sleep–renal connection has been established, this is the first study to show that repetitive exposure to insufficient sleep affects sodium excretion. As we controlled the sodium intake throughout the study, the increased sodium excretion may indicate measurable drops in total body sodium in response to repetitive sleep restriction. Further research is required to elucidate the relative involvement of sympathetic, atrial natriuretic peptide, and renin–angiotensin–aldosterone mechanisms.

The sustained blunted BP dipping reached significance in DBP and showed a trend in SBP, indicating a differential effect of repeated sleep restriction on total peripheral resistance and cardiac output. Previous studies reported endothelial dysfunction in response to sleep deprivation. Specifically, acute total sleep deprivation (i.e., 40 h) caused endothelial dysfunction by decreasing cutaneous vascular conductance even before the increase in sympathetic activity and SBP [26], and further, 8 days of partial sleep deprivation (i.e., 5.1 h/night) impaired flow-mediated dilation [27]. Thus, it can be speculated that both endothelial dysfunction and increased sympathetic activity-induced vasoconstriction would increase total peripheral resistance (primarily affecting DBP) and manifest before the increase in SBP in this model of repeated exposure to sleep restriction.

**Limitations**

There are three limitations to the current study that warrant discussion. One is the timing of the sleep period. The circadian placement of the short sleep period, during the latter half of the night when circadian influences favor REM sleep [28], resulted in higher amounts of REM sleep during the first 4 h of sleep in the restricted condition. Although slow wave sleep, which is known to facilitate nocturnal BP drop [19], was maintained in the sleep-restricted condition, we cannot generalize to other possible time placements of the 4-h sleep period. Further research is required to determine whether permitting sleep between 2300 and 0300 h for instance, instead of 0300 to 0700 h as was done in the current study, would affect BP dipping in the same way.

Another limitation is the age of the participants. The participants were adults in their early 30s, on average, an age when diastolic hypertension may emerge as a risk factor for subsequent development of conjoint SBP and DBP hypertension [29]. It is not clear how older adults would respond to exposures to repetitive sleep restriction.

Third, the problem associated with using Portapres is that the absolute values collected from arteries in the digits may not closely correspond to the arterial BP measured from the upper arm. However, our emphasis is on within-participant changes in response to manipulations of sleep schedule. For each participant stay, we used the same cuffs to minimize technical bias, wherever feasible. The Portapres produces less noise and tactile stimuli than ambulatory arm measurement devices [30] and was chosen for its minimal sleep disruption. The larger measurement sample size collected from Portapres also provided more accurate estimation of overall BP compared with the limited sample size from an ambulatory BP monitor.

**Perspective**

The current study is the first to investigate sleep-associated BP dipping in the context of repeated exposure to significantly shortened sleep in healthy participants with good quality sleep. The results of this study show that duration of sleep is important for regulating BP dipping and thereby, average 24-h BP. Results of this study show that sleep-associated BP dipping does not adapt to insufficient sleep taken in the latter half of the normal sleep period time.

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REFERENCES


Reviewers’ Summary Evaluations

Reviewer 1

In this study, the authors assessed the effects of sleep deprivation on nocturnal blood pressure dipping in a cohort of normotensive young to middle-aged subjects. In a carefully designed study, their findings enable separation of factors in sleep deprivation from other life style occurrences such as work stress that are frequently associated with high blood pressure and disturbed sleep patterns. While the study could have been more adequately powered, the authors clearly demonstrate the loss of dipping in the sleep-deprived group and consider possible mechanisms.

Reviewer 2

This is a small but well conducted study with a clever design. Sleep interruption was associated with changes in diastolic BP. Systolic BP changes were less clear cut but the findings may have implications for the diagnosis of hypertension in people with different sleep patterns.