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BRIEF COMMUNICATION

Effects of a Restricted Sleep Regimen on Ambulatory Blood Pressure Monitoring in Normotensive Subjects

P. Lusardi, A. Mugellini, P. Preti, A. Zoppi, G. Derosa, and R. Fogari

The influence of sleep deprivation during the first part of the night on 24-h ambulatory blood pressure monitoring (ABPM) was studied in 18 normotensive subjects. They underwent two ABPM, one week apart: during the first, they slept from 11 PM to 7 AM, and during the second, from 2 AM to 7 AM. The main differences were observed at dawn, before awakening, when SBP and DBP significantly decreased ($P < .01$) in the restricted sleep regimen, and during the morning after the recovery sleep, when SBP and HR significantly increased ($P < .05$). The explanation for these findings is not obvious. We suppose that the decrease in SBP and DBP at dawn might be due to a reorganization of the sleep phases in the restricted sleep regimen, whereas the increase in SBP and HR after awakening might be due to a greater sympathetic activation, as though sleep deprivation was a stressful condition. Am J Hypertens 1996;9:503–505

KEY WORDS: Sleep, blood pressure, heart rate, ambulatory blood pressure monitoring.

Many longitudinal and cross-sectional studies relate habitual sleep deprivation to premature follow-up mortality,[1–3] cardiovascular morbidity[4] and functional disability[5]: subjects sleeping less than 6 h per night have been reported to be at greater risk for such adverse outcomes when compared to subjects sleeping 8 h per night.[6]

Restricting sleep to 4 h a night is known to affect both the composition of sleep,[7] with a consequent reducing of the amount of rapid eye movement (REM) sleep, and blood pressure and heart rate regulation during sleep.[8] In fact, during non-REM phases, HR tends to slow and BP tends to lower as a result of a relative increase in parasympathetic activity, whereas REM sleep is associated with profound sympathetic activation which makes BP and HR return to levels similar to those during the awake state.

To date, no study has examinated the effects of sleep deprivation on BP and HR in the morning after the recovery sleep. The aim of this study is to evaluate the influence of sleep deprivation during the first part of the night on night and following morning BP in normotensive subjects.

SUBJECTS AND METHODS

Eighteen healthy normotensive volunteers (8 men and 10 women) aged 24–30 years (SBP = 125 ± 6 mm Hg, DBP = 75 ± 5 mm Hg), nonsmokers, without any family history of diabetes mellitus, with a regular sleep–wake schedule (about 8 sleep-hours per night) and no major sleep complaints were studied. They underwent a 24-h ambulatory blood pressure monitoring (ABPM) twice, 1 week apart: during the first one they had a full night sleep and during the second they were subjected to sleep deprivation during the first part of the night. All gave informed consent to participate in the study.

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From the Department of Internal Medicine and Therapeutics, University of Pavia, Pavia, Italy

Address correspondence and reprint requests to Paola Lusardi, Via Costanza 15, 20146 Milano, Italy.

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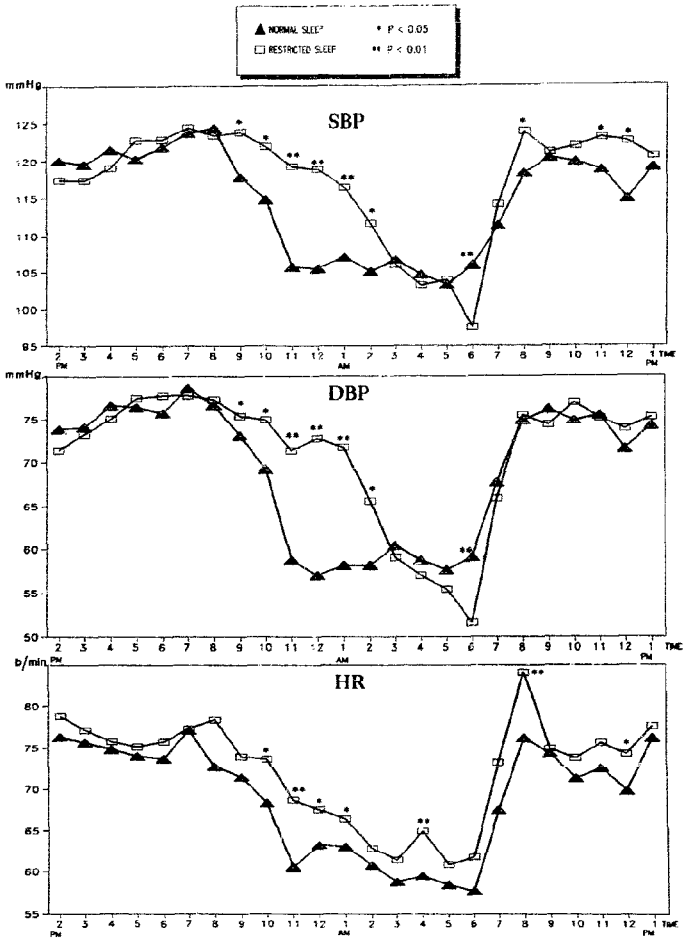

FIGURE 1. *Mean hourly values for systolic (SBP) and diastolic (DBP) blood pressure and heart rate (HR) in normotensive subjects during a regular sleep–wake schedule (—▲—) and, one week apart, a sleep deprivation schedule (—□—). * = P < .05 ** = P < .01).*

Twenty-four-hour ABPM was performed with SpaceLabs (Redmond, WA) 90207 device; it always started at 2 PM, and recordings were taken at 15-min intervals for the entire period. The first night the sleep period was scheduled from 11 PM to 7 AM; the second night, 1 week after, subjects were subjected to a restriction of sleep to the second half of the night (sleep-time from 2:30 AM to 7 AM, about 43% of the total amount). Subjects were not allowed to nap, drink caffeinated beverages or alcohol, or perform any heavy activity. Twenty-four hour recordings were excluded from analysis when more than 10% of all readings, or more than one reading per hour, were missing or incorrect.

The statistical analysis of the data was performed using the SAS system version 6.04. Analysis of variance and paired Student's *t* test were used, with $P < .05$ taken as statistically significant.

RESULTS

Results are shown in Figure 1. From 2 PM to 9 PM the mean values of SBP, DBP and HR showed no significant differences between the full night sleep control condition and the restricted sleep condition. From 9 PM to 2 AM SBP and DBP decreased in both conditions, but in the restricted sleep regimen BP was higher (SBP = +10 mm Hg; DBP = +10 mm Hg) and the fall was more gradual ($P < .05$ at 9 PM, 10 PM and 2 AM; $P <$

.01 at 11 PM, 12 PM and 1 AM); HR decreased in both conditions and maintained itself higher (+5 beats/min) in the restricted sleep regimen ($P < .05$ at 10 PM, 12 PM and 1 AM; $P < .01$ at 11 PM). From 2 AM to 5 AM, BP showed no differences between the two conditions; at 4 AM, HR was significantly higher ($P < .01$) in the restricted sleep condition. At 6 AM BP significantly decreased (SBP = −7 mm Hg and DBP = −4 mm Hg, $P < .01$) and HR did not change during sleep deprivation, whereas in the full night sleep condition, BP and HR began to increase. After awakening, at 7 AM, BP and HR quickly increased to reach the mean values of the daytime period at 8 AM, but, with regard to the restricted sleep regimen, SBP and HR were significantly higher during the morning (SBP = +4 mm Hg, $P < .05$ at 8, 11 and 12 AM; HR = 4 beats/min, $P < .05$ at 8 and $< .05$ at 12 AM). DBP showed no differences between the two conditions.

DISCUSSION

The results obtained in the present study confirm previous findings: the timing and the amplitude of the diurnal rhythm of BP is influenced by both extrinsic and intrinsic factors, but the effects of sleeping and waking phases are much greater,[10,11] as BP follows the sleep pattern. Sleep deprivation has no effects on the amplitude of the fall of nocturnal BP, except at dawn, before awakening, when SBP and DBP significantly decrease: this might be due to an increase in the deeper forms of non-REM sleep, in order to compensate for the loss of non-REM sleep when the total time available for sleep is reduced.[7]

The unforeseen data concern the greater increase in SBP and HR after awakening and during the subsequent morning in the restricted sleep regimen. We theorize that sleep deprivation represents a stressful condition, which has already been proven in rats subjected to REM sleep deprivation.[12] Stress promotes an increased synthesis of catecholamines through the activation of superior centres. Unlike noradrenaline, adrenaline shows its hemodynamic effects, such as a rise in SBP and HR, at relatively low concentrations, easily reachable during physiological conditions.

If these findings are confirmed, restricting sleep to the second part of the night could potentially be dangerous for patients with coronary and cerebrovascular disease.

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