Acute changes in cardiovascular function during the onset period of daytime sleep: comparison to lying awake and standing

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Zaregarizi M, Edwards B, George K, Harrison Y, Jones H, Atkinson G. Acute changes in cardiovascular function during the onset period of daytime sleep: comparison to lying awake and standing. J Appl Physiol 103: 1332–1338, 2007. First published July 19, 2007; doi:10.1152/japplphysiol.00474.2007.—The siesta habit is associated with a 37% reduction in coronary mortality, possibly because of reduced cardiovascular stress associated with daytime sleep. Whether the most important behavior is the daytime nap itself, a supine posture, or the expectancy of a nap is unknown. We present the first detailed description on healthy individuals of the acute changes in cardiovascular function during defined phases of the daytime sleep-onset period. These responses were compared with lying awake and standing. Following a night of restricted (4 h) sleep, nine healthy participants (aged 34 ± 5 yr) were allowed to sleep at 1400 for up to 1 h. Polysonomography was used to calculate three phases of daytime sleep onset: phase 1, a baseline period of relaxed wakefulness before lights out; phase 2, the period between lights out and onset of stage 1 sleep; and phase 3, the period between onsets of stages 1 and 2 sleep. Differences (means ± SD) in blood pressure, heart rate, and forearm cutaneous vascular conductance (CVC) between phases were analyzed. During the 9.7 ± 13.8 min of phase 2, systolic and diastolic blood pressure was 4.7 ± 4.5 and 3.6 ± 2.8 mmHg lower than baseline, whereas CVC was 9.5 ± 4.3% higher than baseline (P < 0.05). Subsequent changes in cardiovascular function during the sleep itself were trivial (P > 0.05). The above changes were not observed when subjects stood or laid supine in relaxed wakefulness for 1 h (P > 0.05). Our findings suggest that the period between lights out and sleep onset is associated with the largest acute reduction in blood pressure during one afternoon siesta.

Blood pressure (BP) and heart rate are generally lower at night in normotensive (24) and hypertensive (4) subjects. Data from studies involving 24-h ambulatory monitoring suggest that BP during a daytime nap decreases to similar levels as at night (8, 32). Unfortunately, detailed investigations on changes in cardiovascular function during the sleep-onset period have been undertaken mostly during the nighttime hours (11, 36). In the only relevant study we could locate, Rosansky et al. (29) examined the changes in BP in relation to encephalographic-determined stages of the daytime sleep-onset period. The greatest decline in BP was found to occur before the onset of sleep. All 33 participants in the study by Rosansky et al. were patients from a hypertension clinic, and over half these people had renal dysfunction. Moreover, information about the light-dark protocol was not provided by these researchers.

The practice of “siesta” involves a short nap or period of rest usually taken during the afternoon. Siesta is common in Mediterranean and Latin American countries, which also tend to have low mortality rates of coronary heart disease (16). Although the results of some epidemiological studies suggest that the siesta habit is associated with a slight increased risk of myocardial infarction (7, 17, 26), there have been problems in these studies with control of confounders such as age, diet, and level of physical activity (25). In a recent 12-yr prospective study, Bursztyn and Stessman (10) reported a mortality hazard ratio of 1.6 for those people who napped during the day. These authors ruled out the influences of age, sex, and self-rated health on their findings, although objective measures of diet and physical activity were not studied. The results of the most recent epidemiological study, involving 23,681 individuals and tight control of possible confounders, including diet and physical activity, suggest that adoption of the siesta habit is inversely associated with coronary mortality (25). In this study, individuals who occasionally napped in the afternoon showed a 12% lower coronary mortality and those who routinely took a siesta had a 37% lower coronary mortality than non-siesta takers. This relationship between the degree of siesta habit and coronary mortality was stronger in men than in women. Naska et al. (25) postulated that siesta could act to reduce coronary mortality by reducing “stress,” although the biological evidence for this claim was reported to be inadequate at present. Therefore, it would be illuminating to research any acute changes in cardiovascular-related stress that are associated with a bout of daytime sleep.

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have influenced BP through psychological mechanisms or via subtle changes in posture. Nevertheless, it was clear from the data of Carrington et al. that BP reduced substantially before nocturnal sleep. Carrington et al. postulated that the observed changes in BP in the presleep period could have been due to peripheral vasoconstriction, although relevant variables such as cutaneous vascular conductance (CVC) were not recorded in their study to verify this hypothesis. Therefore, to add to previous research, the primary aim of the present study was to describe the acute changes in BP, heart rate, and CVC during polysomnographic-defined phases of one daytime sleep-onset period. We also aimed to compare these acute changes with those observed while lying awake and standing for similar time periods during the day. We hypothesized that 1) a single daytime nap is associated with lower BPs compared with single bouts of lying awake and standing for similar periods of time, 2) this acute reduction occurs mostly in the presleep period of the nap, and 3) this presleep reduction in BP occurs in parallel with a presleep increase in peripheral vasoconstriction.

METHODS

Participants. Following an estimation of required sample size for our primary comparisons (see Statistical analysis), nine healthy and normotensive adults (8 men and 1 woman) volunteered for the study. Participants were aged 34 ± 5 yr and had a body mass index of 28.9 ± 4.9 kg/m² (means ± SD). Participants were healthy nonsmokers who had not undertaken any shift work or any transmeridian travel in the 2 wk before the study. Participants did not routinely adopt a siesta during their normal day-to-day activities and did not take any medication. They did not engage in intense physical exercise or recreational drug consumption and did not consume large amounts of caffeine (>350 mg/day) or alcohol (>5 standard drinks/wk) in the 24 h before each test session. They did not consume food at least 2 h before the daytime sleep. All participants reported no known personal or family history of a sleep, cardiovascular, or respiratory disorder. The female participant was predicted (from a simple questionnaire) not to be in the menses or ovulation phases of her menstrual cycle during data collection and did not take any oral contraceptives during the study period. The study took place in the winter and was approved by the University’s ethics committee, with all participants providing informed consent.

Experimental design. After familiarization with the laboratory environment and all equipment, each participant was required to attend the laboratory on three occasions according to the three conditions in the experiment: daytime sleep, resting awake and supine, and standing at rest. The order of experimental conditions was counterbalanced. To encourage daytime sleep, participants were instructed to sleep only for a 4-h period from 0200 to 0600 (±30 min) during the night preceding the nap. Importantly, participants followed this sleep protocol during all three trials. Wrist accelerometry data were monitored to ensure adherence to this schedule. The participants reported to the laboratory at 1200 (±30 min). They were then fitted with noninvasive equipment designed to record polysomnographic data, BP, heart rate, and CVC. At 1400 (±30 min), participants were allowed to sleep at their leisure, undisturbed for up to 1 h in a bed situated in our sleep laboratory. The temperature in this laboratory remained between 20 and 22°C. Throughout the presleep wakefulness period, participants remained in a supine position on a bed with their head slightly elevated. Reading, listening to music, or watching television was not allowed. Continuous recordings of BP, heart rate, and CVC were obtained beginning 5 min before lights out and for 1 h after this time.

The data recording period was divided into three phases, derived from the normal progression from wake to sleep and according to polysomnographic criteria described previously (11, 35). The polysomnography-based identification of the phases was completed by an experienced scorer through visual analysis of paper chart recordings. Phase 1 (baseline) consisted of a 5-min period of relaxed wakefulness with eyes open before lights out. Ambient light in this phase was ~200 lx. At the beginning of this phase, participants had already been in bed in a supine position for a minimum of 15 min. Phase 2 was defined as the length of time between the relaxed wakefulness after lights out and the first occurrence of 10 s of continuous α-activity. Immediately after turning off the lights, the experimenter left the sleep laboratory. Participants were left alone to close their eyes and fall asleep at their leisure. Phase 3 was the time between the first 10 s of continuous θ-activity and the first sleep spindle or K complex observed in the polysomnographic data. Phase 3 incorporated alternating periods of α- and θ-activity and was thus synonymous with stage 1 sleep. Cardiovascular function was analyzed as a function of the three phases within this sleep-onset period. The ambient light during phases 2 and 3 was <20 lx. In line with our focus on the daytime sleep-onset period, three rather than the five phases studied by Carrington et al. (11) were chosen, because our study involved a relatively short afternoon nap, which is characteristic of siesta rather than the protracted period of nocturnal sleep studied by Carrington et al. and other sleep researchers. Most participants did not sleep long enough for analysis of the stages of sleep (stages 3 and 4 and rapid eye movement sleep), which typically have longer latencies than participants are prepared to nap for during an afternoon in a typical siesta.

For the other two experimental trials (resting supine and resting standing), participants reported to the laboratory at the same time as above, and BP, heart rate, and CVC were measured for ~1 h. In the rest condition, the subjects were laid supine and awake on the bed for 1 h without reading, listening to music, or watching television. Participants were not allowed to close their eyes. The experimenter checked that the participant did not sleep but kept communication to a minimum. In the standing condition, participants stood for 1 h and were allowed to periodically move their feet to deter muscle cramps or blood pooling. The data collected in these two conditions were also separated into three phases defined for each participant from the polysomnographic data collected during the nap condition.

Procedures. Polysomnographic data were recorded using a Med-elec MR95 digital ambulatory recorder by a central (C3-A2) and occipital (O1-A2) electroencephalography, electrooculography (left and right outer canthi displaced vertically) and electromyography (submental), according to standardized procedures (28).

As in the study by Carrington et al. (11), BP was measured throughout using an arterial volume clamp method with a Portapres device (Portapres model 2, TNO Biomedical Instrumentation, Amsterdam, The Netherlands), which allows beat-to-beat finger arterial BP to be monitored continuously. This equipment has been found not to compromise sleep quality (37). Inflatable cuffs were placed around the index and middle fingers of the left hand, which was elevated to the level of the heart. Because peripheral arterial tone of the finger is affected by ambient temperature, the laboratory temperature was controlled between 20 and 22°C.

The continuous finger arterial pressure wave data were analyzed on a beat-to-beat basis, by the BeatScope pulse contour analysis software (TNO Biomedical Instrumentation). Blood pressure was obtained from the electrical integration of the continuous pressure signal (40). Comparisons of the Portapres and intra-arterial BP have shown that finger arterial pressure agrees well with central arterial pressure (18) and that BP can be determined reliably (15). The Portapres device also provided indirect measurements of stroke volume, heart rate, and cardiac output based on the same three-element model of aortic input impedance as for arterial BP (40). The pulse contour analysis performed by the software has been shown to be a reliable method to track changes in stroke volume (19, 27) and cardiac output (27, 34).
Cutaneous blood flow was measured using laser-Doppler flowmetry (Periflux System 5001, Jarfalla, Sweden). This technique is noninvasive and measures red blood cell flux. One laser-Doppler probe (Perimed, Suffolk, UK) was attached to the ventral aspect of the left forearm skin using an adhesive disk. The output from the flow-meter was relayed to a personal computer and displayed in real time using the associated recording software. The cutaneous blood flow data were converted from units of perfusion to units of CVC by calculating the ratio of laser-Doppler flow measured in perfusion units to mean arterial pressure (MAP).

Data reduction. An important aspect of our study was that the changes in cardiovascular function were expressed relative to polysomnographic-determined phases of the sleep-onset period. In phase 1 of the sleep-onset period, data were averaged into five 1-min periods. Obviously, the duration of phases 2 and 3 would vary between participants because of differences in entry time to the different sleep stages. Accordingly, these data were averaged into proportions of the total time spent in the phase according to the methods outlined by Carrington et al. (11). Consequently, the duration of each phase was divided into 10 equal segments, with 1 epoch representing 10% of the total time spent in the phase. All cardiovascular data in all three conditions were averaged according to these three defined phases of sleep-onset determined in the nap condition.

Statistical analysis. The primary outcome variables were systolic and diastolic BP. The primary comparison was the mean difference in BP between the baseline period and the phase 2 (lights out to onset of stage 1 sleep) period. Without any prior study exactly like ours, we hypothesized that this difference in systolic BP would be similar to the reduction of ~4 mmHg that was observed by Carrington et al. (11) between the same phases of nocturnal sleep and by Rosansky et al. (29), who studied the daytime sleep of diseased individuals. Therefore, it was estimated that at least eight subjects would be required for a difference of 4 mmHg to be deemed statistically significant assuming a standard deviation of differences (SD$_{diff}$) of ~5 mmHg, statistical power of 80%, and use of a one-tailed (in line with our directional hypotheses) paired t-test. In view of the fact that previous authors of related studies did not quote the within-subjects SD of the changes in BP, our delimitation of SD$_{diff}$ was based on the known reproducibility of our BP measurements (18).

A two-factor (condition $\times$ phase) repeated-measures general linear model was used to compare the changes in systolic BP, diastolic BP, heart rate, and CVC across the three-phase averages between the three conditions. A statistically significant interaction between condition and phase was followed up with simple main effects analysis (30) across the three sleep phases within each experimental condition. A significant effect of phase within each condition was followed up with pairwise multiple comparisons using the Bonferroni method of controlling type I error rate (2). All repeated-measures analyses were corrected for any violation of the sphericity assumption (1). All data were analyzed using version 12 of the Statistical Package for the Social Sciences. Data are presented in the text as means $\pm$ SD. Ninety percent confidence intervals (90% CI) were also calculated for the primary comparisons (33).

RESULTS

Durations of sleep-onset phases. The duration of the phase 1 baseline period leading up to lights off was set at 5 min. Participants had been lying supine for 15 min before this first phase. The durations (means $\pm$ SD) of phase 2 (lights out to onset of stage 1 sleep) and phase 3 (onset of stage 1 sleep to onset of stage 2 sleep) were 9.7 $\pm$ 13.8 and 5.9 $\pm$ 11.1 min, respectively.

Changes in BP and heart rate. Figure 1 presents the acute changes in BP and heart rate over the three phases of the sleep-onset period and in the three experimental conditions. Statistically significant interactions were found between condition and sleep-onset phase for systolic BP ($P = 0.009$), diastolic BP ($P < 0.0005$), and heart rate ($P = 0.03$). Simple main effects analysis showed that, in the resting-supine and standing conditions, there were no significant changes in systolic BP and heart rate over time ($P > 0.05$). Diastolic BP was found to increase over time in the standing condition ($P = 0.005$). Conversely, systolic and diastolic BP and heart rate showed significant reductions over the three phases of sleep onset in the nap condition. Pairwise comparisons between

Fig. 1. Mean changes in systolic blood pressure (BP), diastolic BP, and heart rate over the 3 study phases during the 3 experimental conditions (nap, lying awake, and standing). Phase 1 (P1), baseline; phase 2 (P2), lights off to onset of stage 1 sleep; phase 3 (P3), onset of stage 1 sleep to onset of stage 2 sleep. The data collected in the standing and lying awake conditions were expressed relative to the durations of these phases of the sleep-onset period in the nap condition. Statistically significant interactions between condition $\times$ phase were found for systolic and diastolic BP ($P < 0.05$).
phases in this condition are shown in Table 1. The largest reductions in systolic (90% CI = −1.0 to −8.5 mmHg) and diastolic (90% CI = −1.2 to −6.0 mmHg) BP were found between phases 1 and 2, i.e., in the relaxed wakefulness before the onset of stage 1 sleep (Fig. 1).

The changes in MAP within each phase of the nap condition were explored using separate repeated-measures general linear models together with trend analysis (Fig. 2). A significant linear reduction in MAP was found within phase 1 (P = 0.03). The mean rate of reduction in MAP within this phase was −0.58 mmHg/min. A significant quadratic trend was found for the changes in MAP in phase 2 (P = 0.05), indicating that BP was decreasing but stabilizing toward the end of this phase. Using the mean time spent in phase 2, the mean rate of reduction in MAP within this phase was calculated to be −0.27 mmHg/min. No significant changes in MAP were found within phase 3 (P = 0.84) of the sleep-onset period (Fig. 2). The rate of change in MAP within this phase was actually found to be slightly positive (0.20 mmHg/min).

BP was generally lowest in the nap condition. Over all three phases, systolic BP was 9.8 ± 9.3 mmHg lower and diastolic BP was 10.0 ± 6.9 mmHg lower during the nap condition compared with the standing condition (P < 0.05). During phase 3, systolic BP was 11.0 ± 13.9 mmHg lower and diastolic BP was 6.9 ± 5.7 mmHg lower during the nap compared with resting, supine (P < 0.05).

Changes in cardiac output, stroke volume, and CVC. Figure 3 presents the acute changes in cardiac output, stroke volume, and CVC measured over the three phases and three experimental conditions. No significant interaction was found between condition and sleep-onset phase for cardiac output (P = 0.81). The condition × phase interaction approached significance for stroke volume (P = 0.06). This variable remained relatively stable over time in the nap and resting-supine conditions but rose slightly over time in the standing condition (Fig. 3). There was also evidence (P = 0.09) of a condition × phase interaction in CVC. This variable was found to change over the sleep-onset phases only in the nap condition (P = 0.01). CVC

![Figure 2](http://jap.physiology.org/) Changes (means ± SD) in mean arterial pressure within the 3 study phases during the nap condition. The data collected in the standing and lying awake conditions were expressed relative to the durations of these phases of the sleep-onset period. Significant trends over time were found within phases 1 and 2 (P < 0.05) but not phase 3 (P = 0.84).

![Figure 3](http://jap.physiology.org/) Mean changes in cardiac output, stroke volume, and cutaneous vascular conductance (CVC) over the 3 study phases during the 3 experimental conditions (nap, lying awake, and standing). The data collected in the standing and lying awake conditions were expressed relative to the durations of these phases of the sleep-onset period in the nap condition.
increased by 9.5 ± 4.3% in phase 2 compared with baseline (P = 0.02). Once stage 1 sleep was initiated, no further significant change in CVC was found (Table 1).

DISCUSSION

In view of the recently reported inverse relationship between siesta and cardiovascular mortality (25), the present study was designed to compare the acute changes in cardiovascular function (BP, cardiac output, heart rate) over polysomnographic-defined phases of the daytime sleep-onset period with those associated with lying awake and standing. Before the present study, only nocturnal sleep had been investigated in this way with healthy subjects (11). Our aims were not to compare changes in cardiovascular function between nocturnal and daytime sleep nor between individuals who habitually siesta with those who do not. In the present study, MAP was found to be 5–10 mmHg lower during the sleep-onset period compared with lying awake or standing for similar durations. Importantly, most of this reduction in BP occurred between lights off and the onset of stage 1 sleep. CVC was found to increase by ~10% during this same presleep period.

Duration of daytime sleep phases. Following a night of prior sleep restriction (4 h), polysomnographic data showed that all participants fell asleep after lights-out at 1400. Like Carrington et al. (11), we found that the duration of phase 3 (5.9 min) of the sleep-onset period was shorter than the time spent in phase 2 (9.7 min). Nevertheless, these two durations found in the present study were ~3 min shorter than those reported by Carrington et al. during the nocturnal sleep-onset period. This increased sleep latency may be explained by the fact that our participants restricted their prior nocturnal sleep or by the fact that we did not allow participants to read, listen to music, or watch television during the sleep-onset period. Alternatively, this difference could be explained by individual differences in the propensity for sleep onset. Our participants were 13 yr older, on average, than those studied by Carrington et al., which may have been a factor in increasing sleep propensity. Like Carrington et al, we found large interindividual differences in the durations of sleep-onset phases; the standard deviation was often larger than the mean duration itself. This heterogeneity in sleep propensity is well known, and it was the reason we expressed all the changes in cardiovascular function relative to the time spent by each participant in each sleep stage. There were also large interindividual differences in sleep architecture following initiation of stage 2 sleep (the end of our phase 3 study period). Several subjects woke too soon after this period for us to analyze the changes in cardiovascular function during stages 3 and 4 of sleep and rapid eye movement sleep. We also did not examine, during the nap, the cardiovascular responses to being woken by the experimenter in the same way as Burgess et al. (6), because we could not guarantee attainment of stable sleep during the afternoon nap and thought that expectancy of being woken at a later stage might interfere with the participants ability to fall asleep.

Presleep reductions in BP. Although we did not compare directly the daytime sleep-onset period with that associated with nocturnal sleep, our observation that BP fell in the presleep period (Figs. 1 and 2) is consistent with the findings of Carrington et al. (11) and other researchers (13, 14) who studied the nocturnal sleep-onset period. Our data also agree with those of Rosansky et al. (29), who studied daytime sleep in hypertensive patients with renal failure but provided no information about the light-dark protocol. We found a clear reduction in BP after the time of lights out, when individuals were free to sleep at their leisure. This reduction was not apparent when participants spent a whole experimental trial lying awake but knowing that they were not allowed to sleep. Therefore, from a public health perspective, therefore, the most important aspects of the siesta habit for inducing an acute reduction in BP could be the expectancy of sleep itself. In terms of causal mechanisms, the effects of subtle changes in body posture should have been trivial in the present study, because our participants were not allowed to read, listen to music, or watch television. Rather, they remained in the same supine position during baseline and phase 2 of the sleep-onset period. It is also unlikely that darkness itself exerted a direct effect on cardiovascular activity, because we found no changes in cardiac output over the phases of the sleep-onset period.

Presleep changes in CVC. Carrington et al. (11) postulated that one explanation for the fall in BP before nocturnal sleep is through “peripheral vasodilation . . . in anticipation of sleep.” In our study on daytime sleep, we found increases in CVC in parallel with the reductions in BP during the period before onset of stage 1 sleep (Fig. 3). Peripheral vasodilatation has an endogenous circadian component, being greatest in the “heat-loss” period during the evening and before nocturnal sleep (39). Kawano et al. (21) and Casiglia et al. (12) reported that the nocturnal reduction in BP and heart rate was associated with an increase in forearm blood flow in normotensive subjects. Nevertheless, there is a large influence of sleep-wake behavior on this mechanism. Krauchi (23) presented a model based on the main hypothesis that all thermoregulatory effects that lead to an increase in the core-to-shell ratio (i.e., reduced shell, increased distal skin blood flow, and increased distal skin temperature) should lead to increased sleepiness and, as a consequence, to increased sleep propensity. In this model, there was a feedback loop from the sleep regulatory system to the thermoregulatory system via sleep-related behaviors (e.g., relaxation, lying down). It has been known for many years that, even when ambient temperature is fully controlled, relaxing in a supine posture leads to a decrease in cutaneous sympathetic nerve activity that can increase peripheral blood flow and, therefore, skin temperature in mammals and humans (5, 38). Nevertheless, there is evidence that most of this increase occurs between lights-off and onset of stage 2 sleep a long time after the subjects first adopted a supine position (22). The model provided by Krauchi (23) is consistent with our findings on changes in BP in the presleep period. Unfortunately, core body temperature was not measured in the present experiment, and so any relationships between BP, peripheral vasodilatation, and presleep reductions in body temperature cannot be examined.

Sleep-onset vs. sleep-wake periods. It is clear that BP was lower while resting supine and, especially, when daytime sleep was allowed than while standing (Fig. 1). It can be speculated that repeated bouts of this acute reduction in BP may be one explanation for the lower cardiovascular mortality in habitual siesta takers found by Naska et al. (25). Theirs was the first well-controlled study that resulted in an inverse relationship between the siesta habit and cardiovascular mortality. Although previous research groups found this relationship to be
positive (7, 17, 26), Naska et al. believed that these groups did not adequately control all confounding variables, including diet and physical activity. Nevertheless, Naska et al. still stressed the possibility that the waking period during siesta could lead to increases in the risk of coronary attack or death. It is known that sudden cardiac death is most common during the “surge” in BP that occurs during the morning hours after nocturnal sleep (3). BP and heart rate are also transiently increased following an afternoon nap and there are also hormonal changes that collectively have the potential to increase thrombogenic potential (9, 31). Recently, Jones et al. (20) reported that the BP reactivity to a given change in physical activity is highest in the morning after nocturnal sleep, although a secondary peak was also observed in the late afternoon. Future research work might examine the changes in cardiovascular function during the transition period covering the time of waking from an afternoon nap to becoming physically active again after siesta.

Study limitations. The results of the present study should be limited to daytime rather than nocturnal sleep as well as to people who do not routinely adopt the siesta habit. We aimed to compare the acute cardiovascular responses to a “one-off” afternoon nap with those associated with bouts of standing and lying awake in the afternoon. Twenty-four-hour ambulatory monitoring (without any associated polysomnographic data) has been employed to examine the BP changes in habitual siesta takers who have not necessarily had their nocturnal sleep disrupted (8, 32). For example, Stergiou et al. (32) collected ambulatory BP data from siesta takers who, on average, slept for 7.4 h during the night. Nevertheless, it would have been difficult for us to obtain polysomnographic data over a large number of 24-h periods to confirm whether our findings are maintained over a long period of time. Therefore in the present study, we needed to compromise between maximal external validity (i.e., allowing subjects complete freedom over their sleep-wake habits as in an ambulatory study) and the maximum depth of analysis within the daytime sleep-onset period (i.e., promotion via a nocturnal sleep restriction protocol of at least some daytime sleep in all subjects and frequent measurements of BP and CVC using laboratory-based equipment).

The present study was also administered in the winter. Siesta is probably more common in the summer months when people try to avoid peak ambient temperatures in the afternoon. The cardiovascular responses to a daytime nap may therefore be different following a few months of acclimatization to high ambient temperatures.

In conclusion, the results of the present study suggest that it is the change in BP during the period between lights off and onset of stage 1 sleep that contributes significantly to an acute reduction in BP during an afternoon nap. An acute increase in CVC was also found during this sleep-onset period. This acute reduction in BP may be one factor in explaining the lower coronary mortality that has been found recently in siesta takers, although more research work is needed to describe in similar detail the changes in cardiovascular function during the waking period of daytime sleep.

REFERENCES


