Effects of selective slow-wave sleep deprivation on nocturnal blood pressure dipping and daytime blood pressure regulation

Friedhelm Sayk, Christina Teckentrup, Christoph Becker, Dennis Heutling, Peter Wellhöner, Hendrik Lehnhert, and Christoph Dott

Effects of selective slow-wave sleep deprivation on nocturnal blood pressure dipping and daytime blood pressure regulation. Am J Physiol Regul Integr Comp Physiol 298: R191–R197, 2010. First published November 11, 2009; doi:10.1152/ajpregu.00368.2009.—Nocturnal blood pressure (BP) decline or “dipping” is an active, central, nervous system mechanism important for BP regulation during daytime. It is, however, not known whether the sleep process itself or, more specifically, slow-wave sleep (SWS) is important for normal dipping. Therefore, in the present study, healthy subjects (6 females, 5 males) were selectively deprived of SWS by EEG-guided acoustic arousals. Mean arterial BP (MAP) and heart rate (HR) were monitored during experimental nights and the following day. Additionally, nocturnal catecholamine excretion was determined, and morning baroreflex function was assessed by microneurographic measurements of muscle sympathetic nerve activity (MSNA) and heart rate variability (HRV). Data were compared with a crossover condition of undisturbed sleep.

Nocturnal BP dipping was successfully deprived leading to significantly attenuated mean arterial BP dipping during the first half (P < 0.05), but not during the rapid-eye-movement-dominated second half of total sleep; however, dipping still evolved even in the absence of SWS. No differences were found for nighttime catecholamine excretion. Moreover, daytime resting and ambulatory BP and HR were not altered, and morning MSNA and HRV did not differ significantly, indicating that baroreflex-mediated sympathoneural BP regulation was not affected by the preceding SWS deprivation. We conclude that in healthy humans the magnitude of nocturnal BP dipping is significantly affected by sleep depth. Deprivation of SWS during one night does not modulate the morning threshold and sensitivity of the vascular and cardiac baroreflex and does not alter ambulatory BP during daytime.

Nondipping; baroreflex; muscle sympathetic nerve activity

UNDISTURBED NOCTURNAL SLEEP is a prerequisite for health and well being. Particularly a proper nocturnal blood pressure decline commonly termed “dipping” is important for cardiovascular health. This process is not merely the consequence of physical inactivity but is actively governed by the central nervous system and mediated by an interplay between the autonomic nervous system and (neuro-)endocrine pathways, some of which are rather sleep dependent, while others are subject to circadian rhythms (1, 13, 34). Importantly, this sleep-associated blood pressure decline does not induce any sympathetic counter-regulation, which is in contrast to the vigorous baroreflex activation to a blood pressure decrease of the same degree in awake subjects.

The sleep-dependent modulation of blood pressure seems to result from the sleep stage-specific integration between cardiovascular reflexes and decreased central autonomic commands (7, 25). In fact, sympathetic nervous traffic to the vasculature continuously decreases with the progressive deepening of non-rapid-eye-movement (NREM) sleep (11, 27). Such decrease of vasoconstictive sympathetic activity to the muscle vascular bed combined with the decline of blood pressure indicates a downward resetting of sympathovagal baroreflex setpoints being most relevant during slow-wave sleep (SWS) (7, 25). Baroreflex mechanisms are increasingly recognized to be involved in the medium-term regulation of blood pressure in addition to their role in buffering acute blood pressure changes (17, 24). The proper resetting of the baroreflex threshold during NREM sleep is probably one key factor for the beneficial effects of sleep-related blood pressure dipping on cardiovascular health (15, 16) and was found to exert stabilizing effects for blood pressure regulation during the daytime period (22).

Sleep disturbances, in contrast, are linked to deficits in blood pressure regulation.

Although the importance of nocturnal blood pressure dipping and baroreflex resetting for cardiovascular health is well acknowledged, its basic physiology is incompletely understood (18, 28). According to observational studies, nocturnal blood pressure dipping seems to depend on the depth of sleep (14). Lack of SWS could attenuate dipping, and therefore inhibit mechanisms of baroreflex-mediated blood pressure resetting. Both fragmentation of sleep due to repetitive arousals as well as sleep deprivation result in nocturnal sympathoexcitation leading to a nondipping profile and increased blood pressure even during daytime (12, 16, 36). The latter has repeatedly been shown for conditions of sleep-disordered breathing and was hypothesized to reflect a pathophysiologic adaptation to the increased nocturnal sympathetic activity (2, 19). The understanding of the underlying mechanisms has lagged behind the identification of the statistical association.

The identification of pathophysiologic processes requires the prior investigation of sleep influences on cardiovascular control mechanisms in healthy individuals. Especially the mere contribution of SWS for the restoration of normotensive baroreflex function during daytime has not yet been defined. Therefore, in the present study we aimed at examining the question of whether selective SWS deprivation 1) alters nocturnal blood pressure dipping and 2) affects baroreflex regulation of daytime blood pressure in young normotensive subjects.
was incrementally infused at doses of 0.15 mg vasoactive drug method (22, 23, 29). In brief, sodium nitroprusside variability (HRV), and oscillometric blood pressure. Subsequently, Data sampling started with a resting period of 15 min Heidelberg, Germany). Computer disk for subsequent analysis (PowerLab; ADInstruments, curvical details and evidence that the recorded activity is of muscular postganglionic efferent muscle sympathetic nerveactivity (MSNA) access to the superficial peroneal nerve. Multiunit recordings of citatory effects of a filled bladder during the subsequent baroreflex testing nerve activity recordings. After awakening, subjects were asked awoken at that time. Subjects woke up spontaneously before 6:30 AM or were gently Subjects' preparation for baroreflex assessment/muscle sympathetic nerve activity recordings. After awakening, subjects were asked to void urine to collect samples for determination of the nighttime excretion of catecholamines and to avoid confounding sympathoexcitatory effects of a filled bladder during the subsequent baroreflex assessment (10). Subsequently, subjects were investigated in a comfortable supine position with one leg slightly elevated to allow easy access to the superficial peroneal nerve. Multunit recordings of postganglionic efferent muscle sympathetic nerve activity (MSNA) were obtained using intraneural Tungsten microelectrodes. Technical details and evidence that the recorded activity is of muscular sympathetic origin has been published previously (23). Analog curves of all parameters were digitized online and stored on a computer disk for subsequent analysis (PowerLab; ADInstruments, Heidelberg, Germany).

Experimental protocol for baroreceptor set point and sensitivity testing. Data sampling started with a resting period of 15 min serving for baseline recordings of MSNA, heart rate, heart rate variability (HRV), and oscillometric blood pressure. Subsequently, baroreflex modulation of MSNA and heart rate was studied by the vasoactive drug method (22, 23, 29). In brief, sodium nitroprusside was incrementally infused at doses of 0.15 mg·kg⁻¹·h⁻¹, and 0.55 mg·kg⁻¹·h⁻¹ followed by infusion of phenylephrine at doses of 0.09 mg·kg⁻¹·h⁻¹, 0.21 mg·kg⁻¹·h⁻¹, and 0.30 mg·kg⁻¹·h⁻¹. Each step was maintained for 5 min. Nitroprusside and phenylephrine periods were separated by a 15-min washout period.

Data Analysis and Statistics

Sleep recordings. Somnopolygraphical recordings were scored off-line according to the criteria of Rechtschaffen and Kales (21) and proposed supplements by two independent investigators (26). For each night, total sleep period (TSP), total sleep time (TST), and the percentage of time spent in different sleep stages [wakefulness after sleep onset (WASO); sleep stages 1 and 2; slow-wave sleep (SWS; stages 3 and 4); and rapid eye movement (REM; stage 5)] were determined with reference to the TSP (in min). TSP lasted from sleep onset, defined as the onset of the first stage 1 epoch, followed by stage 2 sleep, until final awakening, whereas TST excluded the time of intermittent wakefulness (TST = TSP – WASO). Because SWS predominantly occurs during the first half of nighttime sleep, while REM sleep dominates during the second half, sleep parameters were also calculated for the first and second half of total sleep separately.

Baroreflex testing. MSNA, heart rate, and blood pressure were evaluated during five artefact-free minutes of the initial 15-min baseline period and the last minute of each dosing step of the nitroprusside and phenylephrine infusion, respectively. Sympathetic bursts were visually identified by inspecting the mean voltage neurogram. An MSNA recording was considered suitable for analysis when the signal-to-noise ratio was > 3. MSNA was quantified with the aid of analytical software (Chart version 5.02; ADInstruments, Heidelberg, Germany), and expressed as the number of bursts per minute (burst frequency). Frequency domain measures of HRV were assessed during 5 min of the baseline period and during 5-min periods at maximal doses of nitroprusside or phenylephrine. Limits of low-frequency and high-frequency domains were defined according to task force standards and expressed as normalized units (31). All recordings were analyzed by the same observer who was unaware of the sleep condition.

Biochemistry. Urine catecholamines, metanephrines, normetaneph- rines, cortisol, and creatinine were measured according to routine laboratory methods.

Statistics. For statistical evaluation, the whole experimental period was divided into following periods: 1) period prior to sleep; 2) total sleep period, which was additionally subdivided into the first and second half of the total sleep period, 3) period immediately after awakening but before arising, including baroreflex testing; and 4) period after leaving the laboratory (ambulatory daytime). Individual values of MSNA, heart rate, HRV, and systolic and diastolic blood pressure, as well as biochemical parameters were averaged for each condition and expressed as means ± SE. Statistical analysis based on ANOVA with the repeated measures factor time and the group factor treatment (undisturbed sleep vs. SWS deprivation). When overall analysis defined significance, post hoc analysis was performed (SPSS for Windows). A Greenhouse-Geisser corrected P value of < 0.05 was considered significant.

RESULTS

Characteristics of Nighttime Sleep

The TSP was not affected by SWS deprivation. However, in accordance with the intended purpose of the protocol, sleep architecture was profoundly modulated. SWS was reduced to < 5% of total sleep (stage 3) compared with undisturbed sleep, and sleep stage 4 was not observed. The majority of acoustic stimuli reduced sleep depth to stages 1 or 2, and only in some episodes did the participants show α-EEG. Thus, SWS
Table 1. Sleep characteristics of both experimental conditions during the whole night and the first and second half of nighttime sleep separately in 11 normotensive subjects

<table>
<thead>
<tr>
<th></th>
<th>SWS Depivation</th>
<th>Control Sleep</th>
<th>P Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total sleep period, min</td>
<td>407.2 ± 3.0</td>
<td>412.1 ± 3.6</td>
<td>0.287</td>
</tr>
<tr>
<td>Total sleep time, min</td>
<td>379.5 ± 9.3</td>
<td>408.8 ± 4.8</td>
<td>0.017*</td>
</tr>
<tr>
<td>Stage 1, %</td>
<td>17.4 ± 2.6</td>
<td>6.9 ± 1.4</td>
<td>0.006*</td>
</tr>
<tr>
<td>Stage 2, %</td>
<td>53.4 ± 3.1</td>
<td>46.0 ± 2.8</td>
<td>0.005†</td>
</tr>
<tr>
<td>Stages 3 and 4, SWS, %</td>
<td>3.0 ± 0.9</td>
<td>23.7 ± 3.1</td>
<td>0.000‡</td>
</tr>
<tr>
<td>Stage 5, REM, %</td>
<td>16.6 ± 2.0</td>
<td>20.3 ± 1.4</td>
<td>0.109</td>
</tr>
<tr>
<td>Stage 0, WASO, %</td>
<td>6.9 ± 1.9</td>
<td>0.8 ± 0.3</td>
<td>0.009*</td>
</tr>
<tr>
<td>No. of acoustic stimuli</td>
<td>44.7 ± 12.3</td>
<td>(40; 28–110)</td>
<td></td>
</tr>
</tbody>
</table>

First half of sleep

| Stage 1, %             | 19.1 ± 3.1     | 6.6 ± 2.2     | 0.015*   |
| Stage 2, %             | 55.7 ± 4.2     | 42.0 ± 3.5    | 0.004‡   |
| Stages 3 and 4, SWS, % | 4.0 ± 1.3      | 34.6 ± 5.9    | 0.000‡   |
| Stage 5, REM, %        | 9.3 ± 2.2      | 13.6 ± 2.0    | 0.138    |
| Stage 0, WASO, %       | 9.7 ± 2.6      | 1.0 ± 0.5     | 0.010*   |
| No. of acoustic stimuli| 25.6 ± 7.3     | (23; 12–65)   |          |

Second half of sleep

| Stage 1, %             | 15.7 ± 2.7     | 7.5 ± 1.7     | 0.015*   |
| Stage 2, %             | 51.2 ± 2.6     | 50.6 ± 2.6    | 0.816    |
| Stages 3 and 4, SWS, % | 2.1 ± 1.0      | 11.0 ± 1.7    | 0.000‡   |
| Stage 5, REM, %        | 23.6 ± 2.7     | 27.8 ± 2.7    | 0.225    |
| Stage 0, WASO, %       | 4.1 ± 1.3      | 0.7 ± 0.3     | 0.017*   |
| No. of acoustic stimuli| 19.1 ± 5.6     | (17; 6–45)    |          |

Values are means ± SE and % of different sleep stages with reference to the total sleep period; SWS, slow-wave-sleep; REM, rapid-eye-movement; WASO, wake after sleep onset. Additionally, the number of acoustic stimuli applied to deprive SWS is reported (means ± SE, median, and range). *P < 0.05; †P < 0.005.

Table 2. Mean blood pressure and heart rate characteristics during different recording periods of the night and daytime in 11 normotensive subjects

<table>
<thead>
<tr>
<th>Recording Period</th>
<th>Mean Blood Pressure, mmHg</th>
<th>Heart Rate, min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SWS Depivation</td>
<td>Control Sleep</td>
</tr>
<tr>
<td>Prior to sleep</td>
<td>88.6 ± 2.3</td>
<td>87.4 ± 1.6</td>
</tr>
<tr>
<td>Total sleep period</td>
<td>79.5 ± 2.4</td>
<td>76.5 ± 2.4</td>
</tr>
<tr>
<td>First half</td>
<td>79.2 ± 2.4</td>
<td>76.3 ± 2.3</td>
</tr>
<tr>
<td>Second half</td>
<td>79.2 ± 2.4</td>
<td>76.2 ± 2.5</td>
</tr>
<tr>
<td>Morning baseline</td>
<td>83.2 ± 1.9</td>
<td>83.0 ± 2.5</td>
</tr>
<tr>
<td>Ambulatory daytime</td>
<td>90.0 ± 2.5</td>
<td>91.1 ± 2.2</td>
</tr>
</tbody>
</table>

Values are means ± SE. The period prior to sleep started ~1 h before sleep. First and second half of sleep consisted of 7 measurements each (at 30-min intervals). The baseline period contained 2 data points (followed by baroreflex challenge with vasoactive drugs; see supplemental table). Recordings during ambulatory daytime were continued until evening (~9 h, at 20-min intervals). *P < 0.05.

BLOOD PRESSURE EFFECTS OF SLOW-WAVE SLEEP DEPRIVATION

Morning Baroreflex Testing

During the baseline period of baroreflex testing blood pressure, MSNA, heart rate, and frequency domain indices of HRV did not differ between both sleeping conditions.

Nitroprusside infusion. The three incremental doses of the direct vasodilator nitroprusside caused a progressive decline in blood pressure together with a counterregulating increase in MSNA and heart rate. Absolute and relative changes of blood pressure and heart rate were similar in both treatment conditions, and HRV did not differ.

Phenylephrine infusion. Infusion of incremental doses of the α1-agonist phenylephrine resulted in a progressive elevation of mean blood pressure without any differences of absolute or net values between both sleeping conditions. Concomitantly, MSNA and heart rate were reflexively reduced without differences of absolute and relative decreases between both experimental conditions. Furthermore, HRV showed no differences (see supplemental table. Supplemental data for this article is

Reduction was accompanied by significant increases of sleep time spent in stages 1 and 2 and periods of intermittent WASO. The latter resulted in a significantly shorter TST of ~30 min. Proportionally, this difference was bigger during the first half of sleep. The difference in the stage 2 percentage of total sleep was confined to the first half of TSP, whereas differences of SWS, stage 1, and WASO were found during both parts. The percentage of REM sleep was not significantly affected by SWS deprivation (Table 1).

In conformity with previous studies of selective SWS deprivation (6, 32) the number of acoustic stimuli applied to deprive SWS varied considerably between subjects. In eight out of eleven individuals this number was higher during the first half of sleep compared with the second half. Taking all participants (n = 11) into account, this difference was near to significant (P = 0.065).

Blood pressure and heart rate profiles. Evening blood pressure or heart rate prior to the experimental sleep did not differ between both conditions (Table 2). Independently from the experimental intervention, the mean nocturnal blood pressure was decreased to <90% of daytime values of the control condition (P < 0.001 for the factor time and not significant for group factor treatment). Equally, heart rate was decreased during sleep compared with daytime (P < 0.001 for repeated-measures factor time) and did not show any differences between both sleeping conditions (not significant for group factor treatment; Fig. 1; Table 2). However, mean arterial blood pressure was significantly lower during undisturbed sleep compared with the SWS-deprived condition (P = 0.048). Further subanalysis of the first and second half of sleep revealed that SWS deprivation attenuated the decrease of mean blood pressure (P = 0.017) but not heart rate during the first half, while it had minor effects during the second part (P = 0.17). Morning blood pressure and heart rate, i.e., immediately after awakening and prior to any orthostatic challenge, did not differ between both conditions. Similarly, no differences of ambulatory blood pressure and heart rate values were observed after subjects had left the laboratory and resumed normal daytime activity.

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To further characterize the set point and sensitivity of the vascular branch of the baroreflex, MSNA was correlated to the corresponding blood pressure values at resting conditions (baseline) and during pharmacologic baroreceptor stimulation or deactivation, respectively, as demonstrated in Fig. 2. Both, set point and stimulus response curve were not altered after nocturnal SWS deprivation compared with the control condition. Correspondingly, cardiac baroreflex set point and sensitivity, defined by the correlation of heart rate and blood pressure, were not altered.

Urine analysis. Urine analysis did not reveal any differences regarding nocturnal adrenaline, noradrenaline, metanephrine, normetanephrine, cortisol, and creatinine excretion.

**DISCUSSION**

Sleep disturbances with reduced SWS have been associated with a blunted or absent blood pressure reduction from day to night, assigned as nondipping, and linked to arterial hypertension even during daytime. In our study, selective SWS deprivation significantly attenuated the sleep-related decrease in blood pressure. This effect was confined to the first half of nocturnal sleep, which is dominated by SWS. During the second half of the night, which shows fewer periods of SWS but is dominated by REM sleep, neither heart rate nor mean blood pressure was changed by SWS deprivation. Thus, our study indicates that disturbing the depth of NREM sleep affects blood pressure regulation during that period of nighttime sleep. This is well in accordance with a recent study of Loredo et al. (14) in normotensive subjects. In this study, which did not selectively disturb SWS but correlated sleep depth with the decline of blood pressure, deeper and less-fragmented sleep was positively correlated with stronger blood pressure dipping. Even during almost complete SWS deprivation in our study, however, the nocturnal blood pressure decreased to \( \approx 90\% \) of daytime values, and thus still met the definition criteria of normal dipping (13, 35). This fits with the observation of Carrington, Trinder, and colleagues (3, 33) who reported that the most pronounced decreases of blood pressure and heart rate occur in association with sleep onset, rather than anticipation of sleep onset or depth of sleep (5). Hence, it is sleep itself, independent from its depth, that mainly determines dipping. The extent of dipping, however, is slightly modulated by SWS occurrence.

In accordance with the study of Tiemeier et al. (32), we found that the selectively deprived SWS was compensated by lighter sleep stages (stage 1 and 2). The TSP, i.e., time from sleep onset until final awakening, did not differ significantly between both experimental conditions. However, TST was significantly shorter (approximately \(-30\) min) during the SWS-deprived night compared with control sleep, due to a higher percentage of intermittent wakefulness after sleep onset. This difference was more pronounced during the first part of the night, which together with the lack of SWS might contribute to the attenuated blood pressure dipping. It remains, how-
Slow-wave sleep deprivation has no effect on global sympathetic activity during the night as estimated by urinary catecholamine secretion. This again is well in accordance with Tiemeier (32) who demonstrated that selective SWS deprivation by acoustic stimuli has no influence on catecholamine excretion. Furthermore, we previously reported that NREM sleep itself, but not different stages of NREM sleep, modulated plasma catecholamine levels (9, 20). Obviously, the increase in periods of short arousal was insufficient to substantially alter overall urinary catecholamine secretion.

In contrast to the clear effects of SWS deprivation on nocturnal blood pressure dipping, morning blood pressure or heart rate after awakening were not altered, and sympathovagal balance of HRV as well as parameters of vascular baroreflex set point and sensitivity did not differ between both experimental conditions at supine rest. Moreover, no differences were found during ambulatory daytime after subjects had resumed their routine activities. Thus, in young healthy humans SWS deprivation together with a moderate diminution of nocturnal blood pressure dipping for one night seems not to be important for normotensive baroreflex function during daytime. Previously, total sleep deprivation of one night was reported to elicit a modest but significant increase in morning blood pressure (12, 16, 36), whereas MSNA decreased, and heart rate, forearm vascular resistance, and plasma catecholamines were not altered. This indicates that the blood pressure elevation following total sleep deprivation seems not to be mediated by sympathetic vasoconstriction or tachycardia, and, according to our present findings, to depend on the bare fact of whether the subjects slept at all rather than the quality of sleep.

Several pathologic conditions in humans affect both sleep quality and sympathetically mediated blood pressure regulation, i.e., sleep-related breathing disorders are characterized by sleep fragmentation with lack of nighttime blood pressure dipping and an increased sympathetic activity with hypertensive blood pressure levels prevailing even at normoxic wakefulness during daytime. On this background, one could hypothesize that the lack of SWS is an important contributor to this complex of symptoms. However, our study indicates that the bare effects of SWS deprivation are modest and do not affect parameters of sympathetic function during the following waking period. However, our single night intervention may underestimate the impact of chronic loss of SWS (4, 5) and therefore might not perfectly translate to patients suffering from intrinsic sleep pathologies. Recently, Tasali et al. (30) reported that selective SWS deprivation during three successive nights shifted cardiac sympathovagal balance toward higher sympathetic activity. This is in contrast with our present HRV findings after one night of SWS deprivation. Repetitive loss of SWS over longer periods might induce changes in cardiovascular regulation that are stronger and more sustained than in our study. Thus, arterial hypertension and sympathetic activation in SWS-deprived subjects only occur in chronic conditions and might be reinforced by phases of apnea and hypoxemia (2, 8, 19).

In conclusion, our study demonstrates that selective SWS deprivation significantly reduces nocturnal blood pressure dipping in young healthy normotensives. However, the almost complete absence of SWS resulting in attenuated dipping during the night has no adverse effects on resting or ambulatory blood pressure or heart rate during the subsequent day.
Blood pressure dipping during sleep is an active, central, nervously governed process, which results from the integration between the decrease of feed-forward autonomic commands and the sleep-stage dependent downward resetting of the baroreflex set point. Vasoconstrictive sympathetic activity is most reduced during SWS (11, 25, 27). This points to the possibility that this sleep stage has specific sympathoinhibitory properties, which is underlined by the fact that nondipping and chronic sleep disturbances with reduced SWS are linked to sympathetic overactivity, hypertension, and increased cardiovascular morbidity. Our study shows that short-term lack of SWS attenuates nocturnal blood pressure dipping but neither affects baroreflex function in the morning nor causes increased daytime blood pressure. Thus, our study challenges the concept that the central nervous processes coupled with the generation of SWS are of long-term importance for sympathetically mediated blood pressure regulation in healthy humans. Whether this finding can be transferred to long-term SWS deprivation deserves further investigation as well as the question of whether a disturbed sympathetic deactivation during SWS per se adds to the development of hypertension.

Perspectives and Significance

Blood pressure dipping during slow-wave sleep is an active, central, nervously governed process, which results from the integration between the decrease of feed-forward autonomic commands and the sleep-stage dependent downward resetting of the baroreflex set point. Vasoconstrictive sympathetic activity is most reduced during SWS (11, 25, 27). This points to the possibility that this sleep stage has specific sympathoinhibitory properties, which is underlined by the fact that nondipping and chronic sleep disturbances with reduced SWS are linked to sympathetic overactivity, hypertension, and increased cardiovascular morbidity. Our study shows that short-term lack of SWS attenuates nocturnal blood pressure dipping but neither affects baroreflex function in the morning nor causes increased daytime blood pressure. Thus, our study challenges the concept that the central nervous processes coupled with the generation of SWS are of long-term importance for sympathetically mediated blood pressure regulation in healthy humans. Whether this finding can be transferred to long-term SWS deprivation deserves further investigation as well as the question of whether a disturbed sympathetic deactivation during SWS per se adds to the development of hypertension.

GRANTS

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DISCLOSURES

All authors declare that there is no potential conflict of interest.

REFERENCES


