

Association Between Hospital-Induced Sleep Disruption and Short-Term Blood Pressure Variability in Hospitalized Patients

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ABSTRACT

Objective: To compare short-term blood pressure variability (BPV) between hospitalized patients with poor and good sleep quality, as assessed by the Richards-Campbell Sleep Questionnaire (RCSQ).

Background: Sleep disruption is common among hospitalized patients and may contribute to hemodynamic instability. Short-term blood pressure variability (BPV) is recognized as an independent predictor of cardiovascular risk. This study aimed to evaluate the association between sleep quality and short-term BPV in hospitalized patients using the Richards-Campbell Sleep Questionnaire (RCSQ).

Methodology: This cross-sectional analytical study was conducted at Gujranwala Medical College Teaching Hospital, Gujranwala, Pakistan, from June 2025 to August 2025. A total of 150 inpatients aged 18 years and above were enrolled. Sleep quality was measured using the Richards-Campbell Sleep Questionnaire (RCSQ), and patients were categorized into poor (RCSQ < 65) and good (RCSQ ≥ 65) sleep groups. Short-term BPV was calculated as the difference between maximum and minimum systolic and diastolic readings across three days (nine readings total). Data were analyzed using IBM SPSS Statistics 20. The Mann–Whitney U test was used to compare BPV between the two sleep quality groups.

Results: The mean age of participants was 45.3 ± 20.15 years, with 52% females. Poor sleep quality was found in 47.3% of patients. The Mann–Whitney U test revealed significantly higher BPV in the poor sleep group for both systolic (U = 2,179.5, $p = 0.017$) and diastolic (U = 2,242.5, $p = 0.030$) pressures.

Conclusion: Poor sleep quality is prevalent among inpatients and is significantly associated with increased short-term BPV. These findings highlight the importance of promoting adequate sleep to reduce cardiovascular instability in hospital settings.

Keywords: Sleep quality, blood pressure variability, inpatients, Richards-Campbell Sleep Questionnaire, cardiovascular risk.

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INTRODUCTION

Sleep is a critical physiological process essential for cardiovascular regulation and overall health. Disrupted

sleep has been linked with increased sympathetic activity, altered hormonal balance, and higher cardiovascular risk. In hospital environments, sleep is often compromised due to noise, pain, nocturnal interventions, and emotional distress.¹ Such disturbances may lead to fluctuations in blood pressure and increase short-term BPV, which is an established marker of cardiovascular morbidity and mortality.²

BPV reflects the magnitude of blood pressure fluctuations over time and can be measured over short-term (minutes to days) or long-term (days to months)

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intervals. Short-term BPV, in particular, captures autonomic nervous system influences and vascular reactivity.³ Elevated BPV is associated with endothelial dysfunction, increased arterial stiffness, and risk of cardiovascular events.⁴

Although sleep quality has been extensively studied in the general population, there is limited research assessing its relationship with short-term BPV in hospitalized patients a group inherently vulnerable to both sleep disturbance and hemodynamic instability. This study, therefore, aimed to investigate whether poor sleep quality is associated with increased short-term BPV among inpatients.

Furthermore, hospitalized patients frequently experience fragmented sleep patterns due to repeated nursing care, medication administration, monitoring procedures, and environmental disruptions during nighttime hours. These interruptions may impair normal circadian regulation of blood pressure, including the physiological nocturnal dipping pattern, thereby contributing to autonomic imbalance and increased cardiovascular stress.⁵ Previous evidence suggests that even short durations of sleep deprivation can lead to transient elevations in blood pressure and heightened sympathetic nervous system activity.⁶

Understanding the relationship between sleep disruption and BPV in admitted patients may have important clinical implications. Identifying poor sleep as a potentially modifiable factor associated with abnormal BPV could encourage the implementation of targeted interventions such as noise reduction strategies, optimized nighttime care schedules, and sleep-promoting protocols within hospital settings. Such measures may ultimately improve patient recovery and reduce cardiovascular complications during hospitalization.⁷

METHODOLOGY

STUDY DESIGN AND PARTICIPANTS

A cross-sectional analytical study was conducted among 150 inpatients admitted to Gujranwala Medical College Teaching Hospital between June 2025 and August 2025. This study was approved by the Institutional Review Board (IRB) of Gujranwala Medical College and Allied Teaching Hospitals, Gujranwala, Pakistan (IRB Approval No.: IRB-42/GMC; Date of Approval: 31 May 2025). All

participants provided written informed consent prior to enrollment. The study was conducted in accordance with the Declaration of Helsinki. Patients were eligible if they were aged 18 years or above, admitted for a minimum of three consecutive days, able to communicate and provide informed consent, and completed the Richards-Campbell Sleep Questionnaire (RCSQ) for three consecutive days with nine blood pressure readings (three per day). Exclusion criteria were defined to minimize confounding and selection bias, and included: patients aged below 18 years; those with incomplete RCSQ data or fewer than nine blood pressure readings; patients with a confirmed diagnosis of primary hypertension on active antihypertensive therapy (to exclude pre-existing BPV elevation); patients admitted to the intensive care unit or receiving mechanical ventilation; those with known obstructive sleep apnea or other diagnosed sleep disorders; patients receiving sedatives, opioids, or psychotropic medications that could independently affect sleep or blood pressure; those with severe cognitive impairment, psychiatric illness, or neurological conditions impairing questionnaire completion; patients with end-stage renal disease, heart failure (NYHA Class III–IV), or other conditions causing inherent hemodynamic instability; and those who did not provide written informed consent.⁸

Sleep quality was assessed using the RCSQ, which includes five domains: sleep depth, sleep latency, awakenings, returning to sleep, and overall quality. Each domain is rated on a visual analog scale. The daily RCSQ score was averaged over three days to yield an overall score. Patients were categorized as:

- *Poor Sleep*: RCSQ < 65
- *Good Sleep*: RCSQ ≥ 65
(cutoff based on Rollo et al., 2022).

Blood pressure was measured thrice daily for three days, totaling nine readings each for systolic (SBP) and diastolic (DBP) pressures. BPV was computed as the difference between the highest and lowest values during this period.

All analyses were conducted using IBM SPSS Statistics 20. Data normality was assessed using the Kolmogorov–Smirnov test. Because BPV variables were non-normally distributed, the non-parametric Mann–Whitney U test was used to compare short-term BPV between the poor and good sleep quality groups,

consistent with the study's primary objective of group comparison. A p -value < 0.05 was considered statistically significant. To address potential confounding, patients on antihypertensive medications, those with a prior history of sleep disorders, and those with hemodynamically unstable conditions were identified at baseline and their characteristics were recorded and reported separately in Table 1. Subgroup sensitivity analyses were not performed due to sample size constraints; however, these variables were noted as limitations and are recommended for adjustment in future studies.

Table 1: Baseline Characteristics of Participants

Variable	Value
Total participants	150
Mean age (years)	45.3 ± 20.15
Female gender	78 (52%)
Hypertension history	52 (34.7%)
On antihypertensive medication	42 (28%)
History of sleep disorder	50 (33.3%)

RESULTS

A total of 150 hospitalized patients were included in the study. Among them, 78 (52%) were female and 72 (48%) were male. The mean age of participants was 45.3 ± 20.15 years. Hypertension was reported in 52 patients (34.7%), while 42 participants (28%) were receiving antihypertensive medications at the time of admission. In addition, 50 patients (33.3%) reported a prior history of sleep-related disturbances.

Assessment using the RCSQ demonstrated that poor sleep quality was common among admitted patients. In Table 2 overall, 71 participants (47.3%) had poor sleep quality (RCSQ score < 65), whereas 79 participants (52.7%) had good sleep quality (RCSQ score ≥ 65). This finding highlights the high burden of sleep disruption within the hospital setting, where environmental noise, nighttime clinical interventions, pain, and anxiety frequently impair restorative sleep. Because apparently hospitals are designed on the principle that healing should occur beside alarms, corridor conversations, and doors slamming every twelve seconds. Figure 1: Pie chart showing prevalence of poor versus good sleep quality.

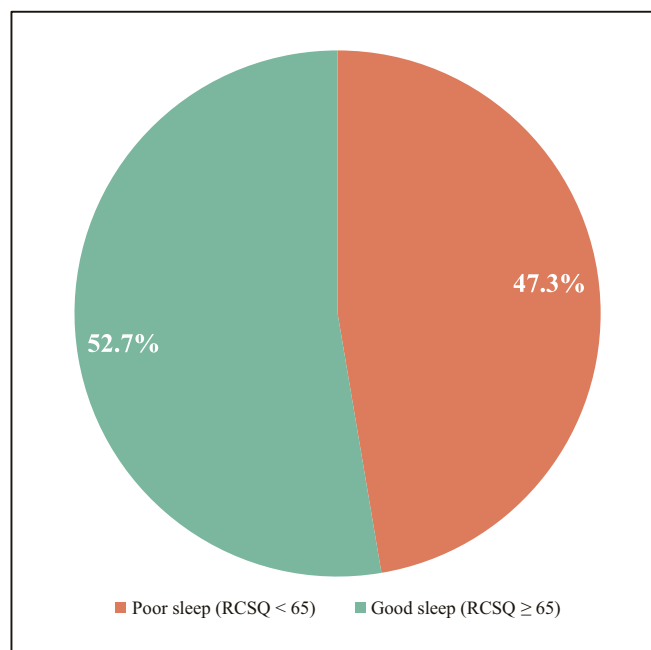


Figure 1. Distribution of sleep quality among 150 hospitalized patients measured by the Richards-Campbell Sleep Questionnaire (RCSQ). Patients scoring ≥ 65 were classified as good sleepers ($n = 79$, 52.7%), while those scoring < 65 were classified as poor sleepers ($n = 71$, 47.3%).

Table 2: Sleep Quality Distribution

Sleep Quality Category	Frequency (%)
Poor sleep (RCSQ < 65)	71 (47.3%)
Good sleep (RCSQ ≥ 65)	79 (52.7%)

The mean systolic blood pressure variability (SBPV) among participants was 34.34 ± 22.98 mmHg, while the mean diastolic blood pressure variability (DBPV) was 27.81 ± 19.72 mmHg. Normality testing using the Kolmogorov-Smirnov test revealed that both SBPV and DBPV were non-normally distributed ($p < 0.001$), supporting the use of non-parametric statistical methods for further analysis. Figure 2: Boxplot comparing systolic BP variability between poor and good sleep groups.

Comparison of blood pressure variability between patients with poor and good sleep quality was performed using the Mann-Whitney U test. Patients with poor sleep quality demonstrated significantly higher systolic BP variability compared to those with good sleep quality. The poor sleep group had a mean

rank of 84.30, whereas the good sleep group had a mean rank of 67.59 ($U = 2179.5$, $p = 0.017$).

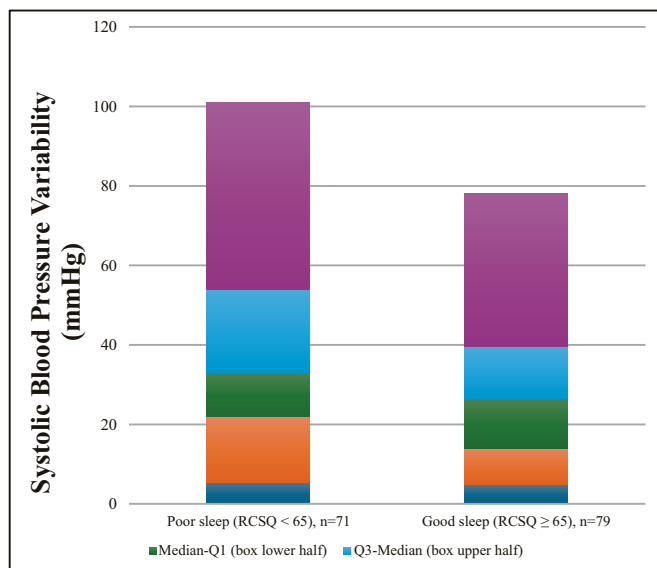


Figure 2. Box plot of systolic blood pressure variability (mmHg) stratified by sleep quality. The Poor Sleep group ($RCSQ < 65$, $n = 71$) demonstrated higher median variability and a wider interquartile range than the Good Sleep group ($RCSQ \geq 65$, $n = 79$). The difference was statistically significant (Mann-Whitney $U = 2179.5$, $p = 0.017$).

Table 3: Blood Pressure Variability and Sleep Quality

Variable	Poor Sleep Mean Rank	Good Sleep Mean Rank	U value	p-value
Systolic BP variability	84.30	67.59	2179.5	0.017
Diastolic BP variability	83.42	68.39	2242.5	0.030

Similarly, diastolic BP variability was significantly greater among patients with poor sleep quality. The mean rank for the poor sleep group was 83.42 compared to 68.39 in the good sleep group ($U = 2242.5$, $p = 0.030$). These findings indicate a statistically significant association between poor sleep quality and increased short-term blood pressure variability in hospitalized patients. Figure 3: Boxplot comparing diastolic BP variability between poor and good sleep groups.

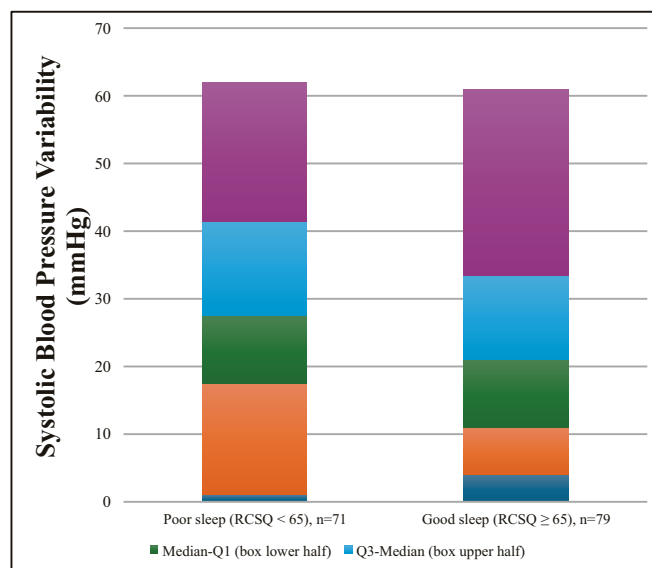


Figure 3. Box plot of diastolic blood pressure variability (mmHg) stratified by sleep quality. The Poor Sleep group ($RCSQ < 65$, $n = 71$) showed higher median diastolic variability compared to the Good Sleep group ($RCSQ \geq 65$, $n = 79$). The difference was statistically significant (Mann-Whitney $U = 2242.5$, $p = 0.030$).

DISCUSSION

This study demonstrated that poor sleep quality is highly prevalent among hospitalized patients, with nearly half of the participants reporting inadequate sleep based on RCSQ scores. More importantly, patients with poor sleep quality exhibited significantly higher short-term systolic and diastolic blood pressure variability (BPV) compared with those reporting good sleep. These findings support the hypothesis that disrupted sleep contributes to autonomic dysregulation and hemodynamic instability in hospitalized individuals. Since elevated BPV is increasingly recognized as an independent predictor of cardiovascular morbidity and mortality, the observed association highlights the potential clinical importance of sleep quality assessment during hospitalization. Our findings align with prior research linking inadequate sleep with elevated BPV and cardiovascular events. For example, it is found that partial sleep deprivation increased nocturnal sympathetic activity and morning BP spikes.⁹ Similarly, study demonstrated that hospitalized patients with poorer subjective sleep had significantly greater BP fluctuations.¹⁰ These studies collectively emphasize the physiological relevance of sleep quality in cardiovascular regulation.

Our results also align with findings from Calhoun and Harding, who emphasized that poor sleep quality and sleep deprivation contribute significantly to hypertension development through sympathetic nervous system overactivity and impaired nocturnal blood pressure dipping.¹¹ In hospitalized settings specifically, Delaney et al. highlighted those environmental disturbances such as alarms, nighttime nursing interventions, pain, and anxiety substantially impair patient sleep and may worsen cardiovascular stress responses.¹²

Several large epidemiological studies have further demonstrated that abnormal sleep patterns are associated with adverse cardiovascular outcomes. The Sleep Heart Health Study conducted by Gottlieb et al. found that reduced sleep duration and fragmented sleep were strongly linked with hypertension and cardiovascular disease risk.¹³ Moreover, research by Kario et al. established that increased short-term BPV is itself an independent risk factor for stroke, endothelial injury, and target organ damage.¹⁴ The current study expands upon these findings by specifically examining hospitalized patients, a population that remains underrepresented in sleep-BPV research despite being highly vulnerable to both sleep disruption and hemodynamic instability.

MECHANISTIC INSIGHTS

The association between poor sleep quality and elevated BPV may be explained by several physiological mechanisms. Sleep deprivation activates the sympathetic nervous system and stimulates hypothalamic-pituitary-adrenal axis activity, resulting in increased catecholamine and cortisol release.¹¹ These neurohormonal changes lead to transient elevations in blood pressure, impaired vascular relaxation, and reduced parasympathetic activity. Additionally, poor sleep disrupts the normal nocturnal dipping pattern of blood pressure, a phenomenon strongly associated with cardiovascular risk and endothelial dysfunction.¹⁴

Repeated sleep fragmentation may also promote systemic inflammation and oxidative stress, contributing to arterial stiffness and impaired vascular compliance. In hospitalized patients, this effect may be amplified by environmental stressors such as noise exposure, frequent nighttime awakenings, bright lighting, pain, and emotional distress. Humans somehow engineered buildings where recovery depends on resting while machines beep like

malfunctioning spacecraft and strangers wake patients every few hours to ask if they are sleeping well. Clinical architecture occasionally feels like satire.

CLINICAL IMPLICATIONS

The findings of this study underscore the importance of incorporating sleep assessment into routine inpatient care. Since poor sleep quality appears to be associated with increased BPV, identifying and addressing sleep disturbances may represent a simple yet valuable strategy to improve cardiovascular stability during hospitalization. Non-pharmacological interventions such as minimizing nighttime noise, reducing unnecessary overnight interruptions, optimizing lighting conditions, and clustering nursing activities may help improve sleep quality.

In addition, sleep-promoting interventions including relaxation techniques, cognitive behavioral approaches, and individualized medication scheduling could potentially reduce autonomic stress and stabilize blood pressure fluctuations. Hospitals may benefit from implementing structured sleep hygiene protocols as part of patient-centered care strategies. Future prospective and interventional studies are needed to determine whether improving sleep quality can directly reduce BPV and improve cardiovascular outcomes among hospitalized patients.

CONCLUSION

Poor sleep quality is highly prevalent among hospitalized patients and is significantly associated with increased short-term systolic and diastolic blood pressure variability. The findings of this study suggest that disrupted sleep may contribute to autonomic imbalance and cardiovascular instability during hospitalization, potentially increasing the risk of adverse clinical outcomes. Given that blood pressure variability is an established predictor of cardiovascular morbidity, sleep disturbance should not be viewed merely as a comfort-related issue but rather as an important clinical factor requiring routine assessment and management.

Improving sleep quality through targeted hospital-based interventions, including reduction of nighttime noise, minimization of unnecessary nocturnal interruptions, optimization of lighting, and incorporation of sleep hygiene practices, may help stabilize blood pressure fluctuations and enhance

patient recovery. Incorporating validated tools such as the RCSQ into routine nursing assessments could facilitate early identification of patients at risk of sleep-related cardiovascular instability.

Future longitudinal and interventional studies using objective sleep assessment methods such as actigraphy and polysomnography are recommended to further clarify the causal relationship between sleep disruption and blood pressure variability in hospitalized populations. Because apparently modern medicine can monitor electrolytes down to decimal points but still struggles with the revolutionary concept of letting sick people sleep through the night.

LIMITATIONS

- **Cross-sectional design:** Causality between poor sleep and BPV cannot be inferred.
- **Subjective measurement:** RCSQ is self-reported and may be influenced by perception or recall bias.
- **Confounders:** Factors like disease severity, sedative use, or emotional distress were not controlled.
- **Measurement consistency:** BP readings were obtained during clinical care rather than standardized research intervals.

ETHICAL APPROVAL

This study was approved by the Institutional Review Board (IRB) of Gujranwala Medical College and Allied Teaching Hospitals, Gujranwala, Pakistan (IRB Approval No.: IRB-42/GMC; Date of Approval: 31 May 2025). All participants provided written informed consent prior to enrollment. The study was conducted in accordance with the Declaration of Helsinki.

CONFLICT OF INTEREST

The authors declare no conflicts of interest. The research was conducted independently without any personal, financial, or professional influence on study design, analysis, or reporting.

FUNDING DISCLOSURE

No external funding was received. All research-related expenses were borne by the authors without financial support from any institution or organization.

AUTHORS' CONTRIBUTIONS

ZI: Study design, data collection, clinical evaluation of

patients.

FS: Data verification, statistical analysis.

MN: Data management, biochemical assays.

TN: Data collection.

MM: Biochemical assays.

MMA: Data collection and analysis.

MMu: Data management.

ZA: Literature review and manuscript drafting.

BT: Biochemical assays and data interpretation.

AS: Critical review of the manuscript.

AA: Review and Submit the manuscript.

All authors have read and agreed to the published version of the manuscript.

REFERENCES

1. Al Haddad N, Costanian C, Zibara V, Bilen Y, Kilani H, Tohme F, et al. The association between sleep disturbances and blood pressure variability: a review of the literature. *J Clin Sleep Med.* 2023 Aug 1;19(8):1533. doi:10.5664/JCSM.10566 PubMed PMID: 37078190.
2. Parati G, Ochoa JE, Lombardi C, Bilo G. Assessment and management of blood-pressure variability. *Nat Rev Cardiol.* 2013; 10(3):143–55. doi:10.1038/NRCARDIO.2013.1 PubMed PMID: 23399972.
3. Rollo E, Della Marca G, Scala I, Buccarella C, Rozera T, Vollono C, et al. Validation Study of the Richards-Campbell Sleep Questionnaire in Patients with Acute Stroke. *J Pers Med.* 2022 Sep 1;12(9). doi:10.3390/JPM12091473 PubMed PMID: 36143258.
4. Webb AJS, Mazzucco S, Li L, Rothwell PM. Prognostic significance of blood pressure variability on beat-to-beat monitoring after transient ischemic attack and stroke. *Stroke.* 2011; 42(9):1170–7. doi:10.1161/STROKEAHA.111.019107/-/DC1 PubMed PMID: 2229726.
5. Calhoun DA, Harding SM. Sleep and hypertension. *Chest.* 2010 Aug 1;138(2):434–43. doi:10.1378/CHEST.09-2954 PubMed PMID: 20682533.
6. Lusardi P, Zoppi A, Preti P, Pesce RM, Piazza E, Fogari R. Effects of insufficient sleep on

- blood pressure in hypertensive patients: a 24-h study. *Am J Hypertens*. 1999;12(1 Pt 1):63–8. doi:10.1016/S0895-7061(98)00200-3 PubMed PMID: 10075386.
7. Delaney LJ, Van Haren F, Lopez V. Sleeping on a problem: the impact of sleep disturbance on intensive care patients - a clinical review. *Ann Intensive Care*. 2015;5(1):3. doi:10.1186/S13613-015-0043-2 PubMed PMID: 25852963.
 8. Richards KC, O'Sullivan PS, Phillips RL. Measurement of sleep in critically ill patients. *J Nurs Meas*. 2000;8(2):131–44. doi:10.1891/1061-3749.8.2.131 PubMed PMID: 11227580.
 9. Tochikubo O, Ikeda A, Miyajima E, Ishii M. Effects of insufficient sleep on blood pressure monitored by a new multibiomedical recorder. *Hypertension*. 1996;27(6):1318–24. doi:10.1161/01.HYP.27.6.1318 PubMed PMID: 8641742.
 10. Stevens SL, Wood S, Koshiaris C, Law K, Glasziou P, Stevens RJ, et al. Blood pressure variability and cardiovascular disease: systematic review and meta-analysis. *BMJ*. 2016;354. doi:10.1136/BMJ.I4098 PubMed PMID: 27511067.
 11. Calhoun DA, Harding SM. Sleep and hypertension. *Chest*. 2010 Aug 1;138(2):434–43. doi:10.1378/CHEST.09-2954 PubMed PMID: 20682533.
 12. Delaney LJ, Van Haren F, Lopez V. Sleeping on a problem: the impact of sleep disturbance on intensive care patients - a clinical review. *Ann Intensive Care*. 2015;5(1):3. doi:10.1186/S13613-015-0043-2 PubMed PMID: 25852963.
 13. Gottlieb DJ, Redline S, Nieto FJ, Baldwin CM, Newman AB, Resnick HE, et al. Association of usual sleep duration with hypertension: the Sleep Heart Health Study. *Sleep*. 2006 Aug 1 ; 29 (8) : 1009 – 14 . doi:10.1093/SLEEP/29.8.1009 PubMed PMID: 16944668.
 14. Kario K, Pickering TG, Matsuo T, Hoshida S, Schwartz JE, Shimada K. Stroke prognosis and abnormal nocturnal blood pressure falls in older hypertensives. *Hypertension*. 2001 ; 38 (4) : 852 – 7 . doi:10.1161/HY1001.092640 PubMed PMID: 11641298.



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