Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 16 May 2026
Revised 12 June 2026
Accepted 04 July 2026

Natural Resources for Human Health 2026, Vol. 6 (4s): 263–275

KEYWORDS:
chronic sleep restriction; military personnel; ambulatory blood pressure monitoring; circadian blood pressure profile; arterial stiffness; central hemodynamics.

Early Cardiovascular Dysfunction in Military Personnel with Chronic Sleep Restriction: Integrated Evaluation of Ambulatory Blood Pressure and Arterial Stiffness

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ABSTRACT:

Background: Chronic sleep restriction and irregular duty schedules are common among military personnel and have been associated with increased cardiovascular risk. However, the earliest cardiovascular alterations preceding overt disease remain incompletely characterized. This study investigated whether chronic sleep disturbances are associated with abnormalities in ambulatory blood pressure regulation, circadian blood pressure profile, arterial stiffness, and central hemodynamics in active-duty military personnel.

Methods: This cross-sectional observational study included 1,327 active-duty male military personnel aged 19–46 years. Participants were classified into three groups according to objectively assessed sleep characteristics and duty schedules: adequate sleep (n=418), chronic sleep restriction (n=503), and disrupted duty schedule (n=406). Sleep was evaluated using 14-day wrist actigraphy, sleep diaries, the Pittsburgh Sleep Quality Index, the Epworth Sleepiness Scale, and STOP-BANG screening. Twenty-four-hour ambulatory blood pressure monitoring was performed to assess circadian blood pressure regulation, while arterial stiffness and central hemodynamics were evaluated using non-invasive pulse wave analysis.

Results: Participants with chronic sleep restriction and disrupted duty schedules demonstrated significantly higher 24-hour and nighttime systolic blood pressure than those with adequate sleep (all $P<0.001$). The prevalence of unfavorable circadian blood pressure phenotypes increased progressively from 26.3% in the adequate-sleep group to 52.7% and 71.7% in the chronic sleep restriction and disrupted duty schedule groups, respectively ($P<0.001$). Nocturnal systolic blood pressure decline decreased significantly across the groups (14.96±9.67%, 8.80±9.33%, and 4.82±9.26%; $P<0.001$), while systolic

Natural Resources for Human Health
eISSN: 2583-1194

blood pressure load increased correspondingly (P<0.001). In contrast, aortic pulse wave velocity, augmentation index, and central systolic blood pressure did not differ significantly among the study groups.

Conclusions: Chronic sleep restriction and disrupted duty schedules were associated with early impairment of circadian blood pressure regulation in military personnel, characterized by elevated nighttime systolic blood pressure, attenuation of physiological nocturnal blood pressure decline, and a higher prevalence of unfavorable circadian blood pressure phenotypes. These findings suggest that abnormalities in circadian blood pressure regulation may represent earlier manifestations of cardiovascular dysfunction than detectable changes in arterial stiffness or central hemodynamics. Integrating objective sleep assessment with ambulatory blood pressure monitoring may facilitate earlier cardiovascular risk stratification in military populations.

INTRODUCTION

Sleep disturbances have become a widespread occupational health problem with important cardiovascular implications. Persistent sleep deficiency and circadian disruption are increasingly recognized as independent determinants of cardiovascular risk, contributing to the development of hypertension, vascular dysfunction, and premature cardiovascular events [1–5]. These effects are especially relevant in occupations characterized by irregular work schedules, night duties, and prolonged operational demands, where adequate recovery sleep is frequently compromised [6–9].

Military personnel represent a particularly vulnerable population because routine service often involves repeated sleep restriction, rotating shifts, and sustained physical and psychological stress. Although these conditions may not immediately produce clinical cardiovascular disease, they can initiate early functional and vascular alterations that remain undetected by conventional clinical assessment [10–13].

Experimental, epidemiological, and clinical studies consistently demonstrate that chronic sleep disturbance adversely affects cardiovascular regulation. Insufficient sleep promotes sympathetic activation, endocrine dysregulation, endothelial dysfunction, oxidative stress, and vascular inflammation, thereby accelerating the development of hypertension and arterial remodeling [14–19].

Among the earliest manifestations of these changes are abnormalities in the circadian blood pressure profile and increased arterial stiffness. Loss of the normal nocturnal decline in blood pressure, elevated nighttime blood pressure, and increased pulse wave velocity are associated with subclinical target-organ damage and independently predict future cardiovascular events [20–29]. Furthermore, growing evidence indicates that sleep restriction and circadian misalignment may impair vascular elasticity even in young adults without overt cardiovascular disease, suggesting that vascular dysfunction precedes structural cardiovascular abnormalities [30–36].

Circadian regulation is essential for maintaining normal blood pressure variability over the 24-hour cycle. In healthy individuals, blood pressure decreases during sleep and rises before awakening. Chronic sleep restriction and irregular sleep–wake schedules disrupt this physiological rhythm, resulting in elevated nighttime blood pressure, loss of normal nocturnal dipping, increased blood pressure variability, and an exaggerated morning blood pressure surge [14,17,24,37,38].

These abnormalities are independently associated with target-organ damage and adverse cardiovascular outcomes, even in individuals without diagnosed hypertension. Ambulatory blood pressure monitoring is therefore considered the most informative method for detecting early circadian disturbances in blood pressure regulation that cannot be identified by conventional office measurements [23,25,26,28,29].

Arterial stiffness is an early marker of vascular damage and an independent predictor of cardiovascular events. Sleep deficiency promotes endothelial dysfunction, oxidative stress, chronic inflammation, and structural remodeling of the arterial wall, leading to progressive loss of vascular elasticity [18,20,30,33,39].

Although increased arterial stiffness has been reported in individuals with chronic sleep disturbances, evidence in military personnel remains limited. Furthermore, few studies have simultaneously evaluated arterial stiffness together with 24-hour

blood pressure characteristics, despite the complementary value of these parameters for identifying early vascular dysfunction before the development of clinically overt cardiovascular disease [11,28,34,35].

Military personnel are routinely exposed to occupational conditions that interfere with normal sleep, including night duties, rotating shifts, prolonged operations, intensive physical training, and sustained psychological stress. Recurrent sleep disruption under these conditions contributes to circadian misalignment and may adversely affect cardiovascular regulation even in young and otherwise healthy individuals [6,8–13,40]. Early identification of cardiovascular alterations in this population is therefore important not only for preventing future cardiovascular disease but also for maintaining operational readiness and long-term health.

Current research has largely established the relationship between sleep disturbances and cardiovascular morbidity, yet the earliest vascular consequences of chronic sleep restriction remain poorly characterized. In particular, the integrated evaluation of circadian blood pressure patterns and arterial stiffness in active-duty military personnel has been insufficiently investigated, limiting opportunities for the early identification of subclinical cardiovascular dysfunction before the development of clinically overt disease [41–44].

Furthermore, few studies have simultaneously assessed 24-hour ambulatory blood pressure characteristics and arterial stiffness in military populations, despite the complementary value of these methods for detecting subclinical vascular dysfunction [20,22,26,28,29].

The present study aimed to evaluate circadian blood pressure characteristics and arterial stiffness in military personnel with chronic sleep disturbances and to determine whether sleep disruption is associated with early vascular alterations before the onset of overt cardiovascular disease. The novelty of this study lies in the integrated assessment of ambulatory blood pressure monitoring and arterial stiffness in a large cohort of active-duty military personnel, providing evidence on early cardiovascular changes in a high-risk occupational population.

METHODS

Study Design

This cross-sectional observational study examined the association of chronic sleep restriction and disrupted duty schedules with circadian blood pressure regulation, arterial stiffness, and central hemodynamics in active-duty military personnel.

All assessments were performed using a standardized protocol under habitual military service conditions. The protocol included clinical and anthropometric evaluation, objective sleep monitoring, 24-hour ambulatory blood pressure monitoring, and non-invasive assessment of arterial stiffness and central hemodynamics.

Sleep exposure was determined before the analysis of cardiovascular outcomes. Participants were subsequently classified into three predefined groups according to objectively assessed sleep duration, sleep-timing regularity, and documented duty schedules.

Study Setting

The study was conducted in military medical units. Sleep characteristics were recorded during routine occupational activities, including daytime service, night duties, rotating shifts, and mixed duty schedules. Cardiovascular measurements were performed under standardized clinical conditions.

Participants

The study included 1,327 active-duty male military personnel aged 19–46 years. Duration of military service ranged from 1 to 24 years. Participants represented different military branches and duty schedules, allowing evaluation of sleep-related cardiovascular changes under routine service conditions.

Demographic characteristics, military service history, duty schedule, smoking status, stimulant consumption, physical activity, medication use, and relevant medical history were collected through structured interviews and standardized case-report forms.

Height and body weight were measured using standardized procedures.

Eligibility Criteria

Inclusion criteria

Participants were eligible when they met all of the following criteria:

- active-duty military service;
- male sex;
- age between 19 and 46 years;
- military service duration of at least 1 year;
- performance of routine duties during the monitoring period;
- completion of objective sleep assessment;
- availability of technically valid cardiovascular measurements;
- provision of written informed consent.

Exclusion criteria

Participants were excluded in the presence of:

- previously diagnosed cardiovascular disease;
- clinically significant cardiac arrhythmia;
- acute infectious or inflammatory disease;
- severe systemic, endocrine, neurological, or psychiatric disease that could affect sleep or cardiovascular function;
- regular use of medications substantially affecting blood pressure or vascular tone;
- clinically significant sleep-disordered breathing identified during additional screening;
- incomplete clinical data;
- technically inadequate sleep, ambulatory blood pressure, or vascular recordings.

Participants at increased risk of obstructive sleep apnea underwent overnight cardiorespiratory monitoring. Cases with clinically significant sleep-disordered breathing were excluded from analyses intended to evaluate the independent cardiovascular effects of chronic sleep restriction.

Group Allocation

Participants were allocated to three predefined groups using actigraphy-derived sleep duration, sleep-timing variability, sleep diaries, and documented duty schedules.

- **Group I: Adequate sleep** — mean nocturnal sleep duration of at least 7 hours and a relatively regular duty schedule.
- **Group II: Chronic sleep restriction** — mean nocturnal sleep duration below 7 hours with a relatively stable sleep–wake and duty schedule.
- **Group III: Disrupted duty schedule** — irregular, rotating, mixed, or night duties associated with substantial variability in sleep timing.

Validated questionnaires were used to characterize subjective sleep quality and daytime sleepiness but were not used as the sole basis for group allocation.

Table 1. Distribution of the study population

Study group	Definition	n	%
Group I	Adequate sleep	418	31.5
Group II	Chronic sleep restriction	503	37.9
Group III	Disrupted duty schedule	406	30.6
Total		**1,327**	**100.0**

Study population by sleep group

Group allocation was based on objective sleep characteristics and documented duty schedules.

Table 2. Baseline demographic characteristics

Characteristic	Adequate sleep (n=418)	Chronic sleep restriction (n=503)	Disrupted duty schedule (n=406)
Age, years	32.7 ± 8.1	32.2 ± 7.7	32.5 ± 8.0
Age range, years	19–46	19–46	19–46
Military service duration, years	12.6 ± 7.1	12.2 ± 7.1	12.9 ± 6.7
Service duration range, years	1–24	1–24	1–24

Data are presented as mean ± standard deviation or range.

Sleep characteristics were assessed using a multimodal approach combining wrist actigraphy, sleep diaries, and validated questionnaires. Participants underwent continuous wrist actigraphy (ActiGraph wGT3X-BT, ActiGraph LLC, USA) for 14 consecutive days during routine military service. Daily sleep diaries were used to verify bedtime, wake-up time, night duties, naps, and periods of device removal.

Objective sleep variables included mean nocturnal sleep duration, sleep efficiency, sleep onset latency, wake after sleep onset, and sleep fragmentation. Subjective sleep quality and daytime sleepiness were evaluated using the Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS), respectively. Obstructive sleep apnea was screened using the STOP-BANG questionnaire. Participants with suspected sleep-disordered breathing underwent overnight cardiorespiratory monitoring, and those with clinically significant sleep apnea were excluded.

Participants were classified according to objectively measured sleep duration and duty schedule into three groups: adequate sleep (≥7 h/night), chronic sleep restriction (<7 h/night), and disrupted duty schedule (rotating, mixed, or night duties associated with irregular sleep timing).

Table 3. Sleep assessment protocol

Assessment	Method	Main variables
Objective sleep	14-day wrist actigraphy	Sleep duration, efficiency, fragmentation
Sleep diary	Daily recording	Bedtime, wake-up time, night duties
Subjective sleep	PSQI, ESS	Sleep quality, daytime sleepiness
OSA screening	STOP-BANG ± overnight monitoring	Exclusion of significant sleep apnea

Ambulatory Blood Pressure Monitoring

Twenty-four-hour ambulatory blood pressure monitoring (ABPM) was performed using the BPLab® oscillometric system (Petr Telegin LLC, Nizhny Novgorod, Russian Federation), which has been validated according to international protocols.

The cuff was placed on the non-dominant arm after calibration against office blood pressure measurements. Blood pressure

was recorded every 15 minutes during daytime and **every** 30 minutes during nighttime. Daytime and nighttime periods were defined individually according to actigraphy and sleep diary records rather than fixed clock intervals.

Recordings were considered valid when monitoring lasted at least 22 hours and ≥70% of scheduled measurements were successfully obtained.

The following parameters were analyzed:

- 24-hour systolic and diastolic blood pressure;
- daytime systolic and diastolic blood pressure;
- nighttime systolic and diastolic blood pressure;
- nocturnal blood pressure decline;
- circadian blood pressure phenotype (dipper, non-dipper, extreme dipper, reverse dipper);
- morning blood pressure surge.

Arterial Stiffness and Central Hemodynamics

Arterial stiffness and central hemodynamic parameters were assessed non-invasively using the TensioMed Arteriograph® system (TensioMed Ltd., Budapest, Hungary).

Measurements were performed under standardized conditions after 10–15 minutes of rest in a quiet room. Participants refrained from smoking, caffeine intake, and vigorous physical activity for at least 2 hours before the examination.

Each measurement was repeated twice, and the average value was used for statistical analysis.

The primary vascular parameters included:

- aortic pulse wave velocity (PWVao);
- augmentation index (AIx);
- central systolic blood pressure (cSBP).

These parameters were used as markers of early vascular remodeling and subclinical cardiovascular dysfunction.

Table 5. Cardiovascular assessment protocol

Assessment	Device	Primary variables
Office blood pressure	OMRON M6 Comfort	SBP, DBP
24-h ABPM	BPLab®	24-h, daytime, nighttime BP, dipping status, morning surge
Arterial stiffness	TensioMed Arteriograph®	PWVao, AIx
Central hemodynamics	TensioMed Arteriograph®	Central SBP

Table 6. Main cardiovascular outcome measures

Outcome	Unit	Clinical significance
24-h SBP	mmHg	Overall blood pressure burden
24-h DBP	mmHg	Overall blood pressure burden
Nighttime SBP	mmHg	Nocturnal hypertension
Nighttime DBP	mmHg	Nocturnal hypertension

Nocturnal BP decline	%	Circadian BP regulation
Morning BP surge	mmHg	Morning cardiovascular risk
PWVao	m/s	Arterial stiffness
AIx	%	Wave reflection
Central SBP	mmHg	Central aortic pressure

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA).

Data distribution was assessed using the Shapiro–Wilk test. Normally distributed continuous variables are presented as mean ± standard deviation (SD), whereas non-normally distributed data are expressed as median (interquartile range [IQR]). Categorical variables are reported as frequencies and percentages.

Comparisons among the three study groups were performed using one-way analysis of variance (ANOVA) with Bonferroni post hoc correction or the Kruskal–Wallis test, as appropriate. Categorical variables were compared using the χ^2 test or Fisher's exact test.

Associations between sleep characteristics and cardiovascular parameters were evaluated using Pearson's or Spearman's correlation coefficients according to data distribution.

All statistical tests were two-sided, and a P-value <0.05 was considered statistically significant.

Table 7. Statistical methods used in the study

Analysis	Statistical method
Normality testing	Shapiro–Wilk test
Continuous variables	Mean ± SD or Median (IQR)
Comparison of three groups	One-way ANOVA / Kruskal–Wallis test
Post hoc analysis	Bonferroni correction
Categorical variables	χ^2 test / Fisher's exact test
Correlation analysis	Pearson or Spearman correlation
Statistical significance	Two-sided P <0.05

Ethics

The study was conducted in accordance with the principles of the Declaration of Helsinki and approved by the Institutional Ethics Committee (Approval No. IRB 853/063).

All participants received detailed information regarding the study procedures and provided written informed consent before enrollment. Participant confidentiality was ensured through anonymization of all study data before statistical analysis.

RESULTS

Twenty-four-hour ambulatory blood pressure monitoring and circadian blood pressure profile

Twenty-four-hour ambulatory blood pressure monitoring (ABPM) was performed in 1,327 military personnel classified into three study groups according to sleep characteristics and service schedule. The analysis included 24-hour, daytime, and

nighttime systolic and diastolic blood pressure, nocturnal blood pressure decline, circadian blood pressure profile, and systolic blood pressure (SBP) load.

Mean 24-hour SBP differed significantly among the study groups ($p<0.001$), whereas 24-hour DBP remained comparable ($p=0.353$). Participants with chronic sleep restriction and those with service schedule disturbances demonstrated higher average SBP than individuals with adequate sleep, while no significant difference was observed between the two sleep-disrupted groups (Table 8).

Table 8. Twenty-four-hour ambulatory blood pressure in the study groups

Variable	Adequate sleep (n=418)	Chronic sleep restriction (n=503)	Service schedule disturbance (n=406)	*p*
24-h SBP (mmHg)	123.30 ± 7.40	126.17 ± 6.96	126.46 ± 6.95	<0.001
24-h DBP (mmHg)	78.43 ± 4.61	78.82 ± 4.39	78.69 ± 4.39	0.353

Daytime SBP also showed statistically significant between-group differences ($p=0.030$), although the absolute differences were smaller than those observed for the 24-hour average. Daytime DBP did not differ significantly among the three groups ($p=0.355$) (Table 9).

Table 9. Daytime ambulatory blood pressure

Variable	Adequate sleep	Chronic sleep restriction	Service schedule disturbance	*p*
Daytime SBP (mmHg)	130.03 ± 10.37	130.21 ± 9.65	128.75 ± 9.62	0.030
Daytime DBP (mmHg)	81.44 ± 6.08	81.95 ± 5.89	81.93 ± 6.04	0.355

In contrast, the most pronounced differences were observed during the nighttime period. Nighttime SBP increased progressively across the study groups, reaching the highest values in military personnel with disturbed service schedules ($p<0.001$). Compared with the adequate sleep group, nighttime SBP was higher by 8.25 mmHg in the chronic sleep restriction group and by 12.04 mmHg in the service schedule disturbance group. Significant pairwise differences were identified between all three groups. Nighttime DBP remained similar across the study **population ($p=0.532$) (Table 10).**

Table 10. Nighttime ambulatory blood pressure

Variable	Adequate sleep	Chronic sleep restriction	Service schedule disturbance	*p*
Nighttime SBP (mmHg)	109.84 ± 8.18	118.09 ± 7.99	121.88 ± 7.82	<0.001
Nighttime DBP (mmHg)	72.42 ± 5.99	72.56 ± 6.07	72.19 ± 5.72	0.532

Progressive impairment of circadian blood pressure regulation was further demonstrated by the distribution of nocturnal blood pressure profiles (Table 11). The prevalence of the physiological dipper pattern decreased from 73.7% in the adequate sleep group to 47.3% in the chronic sleep restriction group and 28.3% among participants with disturbed service schedules. Conversely, the combined prevalence of non-dipper and reverse-dipper profiles increased from 26.3% to 52.7% and 71.7%, respectively ($p<0.001$). The magnitude of nocturnal SBP decline (DIP) also decreased significantly across the three groups, from 14.96 ± 9.67% in the adequate sleep group to 8.80 ± 9.33% and 4.82 ± 9.26%, respectively ($p<0.001$).

Table 11. Circadian blood pressure profile

Variable	Adequate sleep	Chronic sleep restriction	Service schedule disturbance	*p*
Dipper, n (%)	308 (73.7)	238 (47.3)	115 (28.3)	<0.001
Non-dipper, n (%)	85 (20.3)	183 (36.4)	173 (42.6)	
Reverse-dipper, n (%)	25 (6.0)	82 (16.3)	118 (29.1)	

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eISSN: 2583-1194

Unfavourable profile, n (%)	110 (26.3)	265 (52.7)	291 (71.7)	<0.001
Nocturnal SBP decline (DIP, %)	14.96 ± 9.67	8.80 ± 9.33	4.82 ± 9.26	<0.001

The proportion of ambulatory measurements exceeding systolic blood pressure thresholds also increased significantly across the study groups. SBP load reached 25.93 ± 15.59% in the adequate sleep group, increasing to 33.22 ± 15.68% among participants with chronic sleep restriction and 34.99 ± 15.15% in those with service schedule disturbances (p<0.001) (Figure 1).

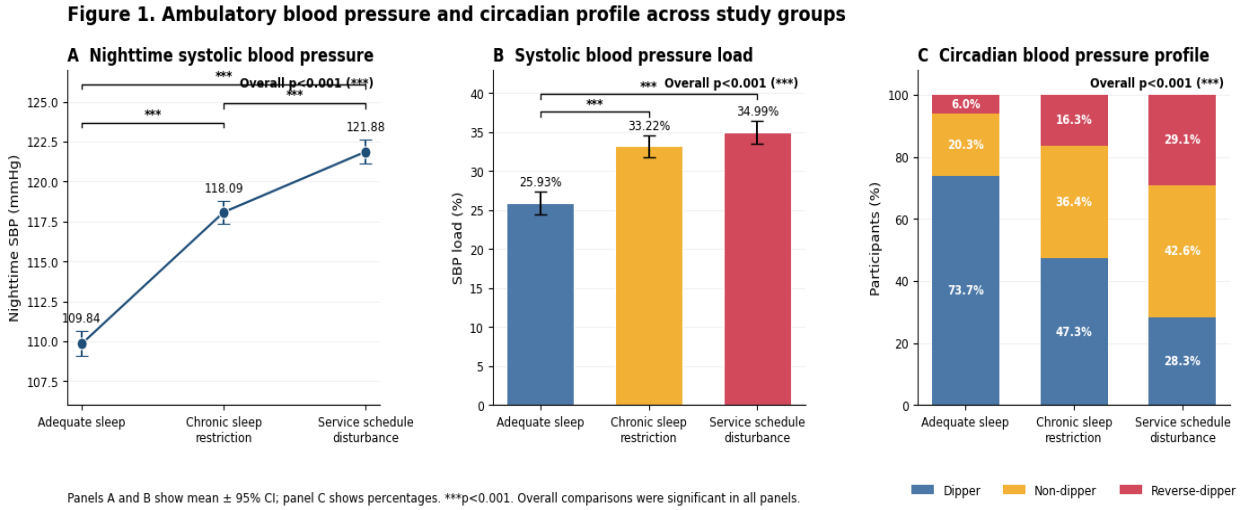

Figure 1. Ambulatory blood pressure and circadian profile across study groups.

(A) Nighttime systolic blood pressure presented as mean ± 95% confidence interval. (B) Systolic blood pressure load presented as mean ± 95% confidence interval. (C) Distribution of dipper, non-dipper, and reverse-dipper circadian blood pressure profiles. Overall between-group differences were statistically significant in all panels (***p<0.001)

Arterial Stiffness and Central Hemodynamics

Arterial stiffness and central hemodynamic parameters were comparable across the three study groups. Aortic pulse wave velocity (PWVao) showed no significant pairwise differences between participants with adequate sleep, chronic sleep restriction, and disrupted duty schedules; all adjusted pairwise *p* values were 1.000. Similarly, the augmentation index (AIx) did not differ between the adequate-sleep and chronic sleep-restriction groups (p=0.805), the adequate-sleep and disrupted-duty-schedule groups (p=1.000), or the two sleep-disrupted groups (p=1.000).

Central systolic blood pressure was also similar among the groups. No significant pairwise differences were observed for PWVao, AIx, or central systolic blood pressure after Bonferroni correction (all adjusted p>0.05) (Table 12**).**

Table 12. Arterial stiffness and central hemodynamic parameters across the study groups

Parameter	Adequate sleep (n=418)	Chronic sleep restriction (n=503)	Disrupted duty schedule (n=406)	Adjusted pairwise *p* values
PWVao, m/s	9.89 ± 1.12; 9.83 [9.06–10.59]	9.81 ± 1.09; 9.85 [9.09–10.54]	9.81 ± 1.01; 9.81 [9.14–10.52]	I–II: 1.000; I–III: 1.000; II–III: 1.000
AIx, %	28.38 ± 5.92; 28.60 [24.92–31.98]	28.07 ± 6.33; 28.00 [23.80–31.80]	28.03 ± 5.77; 28.50 [24.30–31.98]	I–II: 0.805; I–III: 1.000; II–III: 1.000
Central SBP, mmHg	123.35 ± 10.29; 123.30 [116.30–130.38]	122.07 ± 9.93; 121.90 [115.55–128.80]	122.21 ± 9.96; 122.40 [114.83–128.38]	I–II: 0.201; I–III: 0.201; II–III: 0.968

Data are presented as mean ± standard deviation and median [interquartile range]. AIx, augmentation index; central SBP, central systolic blood

pressure; PWVao, aortic pulse wave velocity. Pairwise probability values were adjusted for multiple comparisons.

The proportion of participants meeting the predefined vascular criterion was 66.0% in the adequate-sleep group, 62.0% in the chronic sleep-restriction group, and 67.5% in the disrupted-duty-schedule group. The corresponding 95% confidence intervals were 61.4–70.4%, 57.7–66.2%, and 62.8–71.9%, respectively. Pairwise comparisons were not statistically significant (p=0.469, p=0.711, and p=0.303, respectively).

DISCUSSIONS.

The present study demonstrated that chronic sleep restriction and disrupted duty schedules were associated with progressive alterations in ambulatory blood pressure regulation, particularly during the nighttime period. The most prominent findings included deterioration of the circadian blood pressure profile, attenuation of physiological nocturnal blood pressure decline, and an increased prevalence of non-dipper and reverse-dipper phenotypes, whereas arterial stiffness and central hemodynamic parameters remained largely comparable among the study groups. These observations suggest that disturbances in circadian blood pressure regulation may become detectable before measurable structural vascular changes develop.

Our findings are consistent with previous epidemiological and experimental studies demonstrating that insufficient sleep adversely affects ambulatory blood pressure regulation rather than office blood pressure measurements alone. Bertisch et al. reported that objectively short sleep duration was independently associated with an increased risk of incident hypertension, emphasizing the importance of nocturnal blood pressure assessment in individuals with chronic sleep restriction [45]. Similarly, Bilo et al. showed that nighttime blood pressure is more strongly associated with target-organ damage than conventional daytime measurements, highlighting its superior prognostic value [46]. These observations support the concept that alterations in nocturnal hemodynamics represent one of the earliest manifestations of cardiovascular dysregulation in sleep-deficient individuals.

The progressive shift from a physiological dipper pattern toward non-dipper and reverse-dipper phenotypes observed in the present study also agrees with accumulating evidence linking circadian blood pressure disruption to adverse cardiovascular outcomes. Björklund et al. demonstrated that elevated nighttime blood pressure is frequently observed in individuals with sleep-related breathing disorders and is associated with unfavorable metabolic and cardiovascular profiles [47]. More recently, Crowthers et al. emphasized that circadian disruption itself contributes to the development of hypertension through alterations in the normal temporal organization of blood pressure regulation, independent of traditional cardiovascular risk factors [48]. Collectively, these findings reinforce the growing evidence that impaired nocturnal blood pressure regulation represents an early cardiovascular consequence of chronic sleep deficiency and circadian misalignment rather than simply a secondary manifestation of established hypertension.

The observed alterations in nocturnal blood pressure regulation are biologically plausible and may primarily reflect disruption of normal circadian cardiovascular control. Chronic sleep restriction is associated with persistent sympathetic activation, reduced parasympathetic activity, dysregulation of the hypothalamic–pituitary–adrenal axis, and impaired nocturnal melatonin secretion, all of which contribute to attenuation of the physiological nighttime blood pressure decline [49,50]. In addition, circadian misalignment may impair endothelial function and vascular homeostasis, further promoting abnormal ambulatory blood pressure patterns without necessarily producing detectable changes in arterial stiffness during the early stages of cardiovascular dysfunction [51,52]. These mechanisms may explain why disturbances in circadian blood pressure regulation preceded measurable alterations in vascular structure in the present study.

From a clinical perspective, the present findings emphasize the importance of incorporating sleep assessment together with 24-hour ambulatory blood pressure monitoring into routine cardiovascular evaluation of military personnel exposed to chronic sleep restriction and irregular duty schedules. Because conventional office blood pressure measurements may fail to detect early circadian abnormalities, ABPM may improve identification of individuals at increased cardiovascular risk before the development of overt hypertension or structural vascular changes [53].

The strengths of this study include the large, well-characterized cohort of military personnel and the comprehensive assessment of both sleep characteristics and cardiovascular function using complementary objective and validated methods. In addition to ambulatory blood pressure monitoring, the study evaluated circadian blood pressure profile, arterial stiffness, and central hemodynamics, providing an integrated characterization of early cardiovascular alterations associated with chronic sleep restriction. This multidimensional approach enhances the robustness and clinical relevance of the findings.

Several limitations should be acknowledged when interpreting the present findings. First, the cross-sectional design precludes causal inference regarding the relationship between chronic sleep restriction and cardiovascular alterations. Second, the study population consisted exclusively of male military personnel from a single occupational setting, which may limit the generalizability of the findings to women, civilians, or other professional groups. Third, although multiple validated methods were used to characterize sleep and cardiovascular function, longitudinal follow-up was not performed to determine whether the observed abnormalities predict future hypertension or major cardiovascular events.

Future prospective studies should evaluate whether improving sleep duration and restoring circadian alignment can reverse abnormalities in ambulatory blood pressure regulation and reduce long-term cardiovascular risk. In addition, longer follow-up incorporating repeated assessments of vascular function and clinical cardiovascular outcomes would help clarify the temporal progression from functional circadian dysregulation to structural cardiovascular disease.

In conclusion, chronic sleep restriction and irregular duty schedules were associated with early disturbances in ambulatory blood pressure regulation, particularly impaired nocturnal blood pressure decline and an unfavorable circadian blood pressure profile. These abnormalities occurred despite largely preserved arterial stiffness and central hemodynamic parameters, suggesting that circadian blood pressure dysregulation may represent one of the earliest detectable manifestations of cardiovascular dysfunction in military personnel. Routine assessment of sleep and 24-hour ambulatory blood pressure monitoring may therefore facilitate earlier cardiovascular risk stratification in this high-risk occupational population.

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