

Impact of Body Mass Index on Ambulatory Blood Pressure and Circadian Rhythm Patterns

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Abstract:

Background: Obesity and elevated body mass index (BMI) are well-established contributors to hypertension and cardiovascular morbidity. However, the extent to which increased BMI alters ambulatory blood pressure monitoring (ABPM) parameters and circadian rhythm patterns remains incompletely understood. **Aim:** This study aimed to evaluate the relationship between BMI and ambulatory blood pressure (BP) parameters, focusing on mean 24-hour values, daytime and nighttime averages, BP load, and dipping indices to assess whether increasing adiposity influences hemodynamic regulation and circadian variation. **Methods:** A total of 120 adult participants were stratified into four BMI categories—underweight, normal weight, overweight, and obese. Each participant underwent 24-hour ABPM, with systolic and diastolic readings analyzed for mean daytime, nighttime, and 24-hour pressures, maximum values, and percentage BP load. Dipping index was calculated to assess circadian rhythm integrity. Correlations between BMI and BP indices were evaluated. **Results:** BMI showed a strong positive correlation with body weight ($r = 0.853$, $p < 0.001$) and moderate correlations with maximum daytime systolic ($r = 0.209$, $p = 0.021$) and diastolic ($r = 0.202$, $p = 0.026$) pressures. Mean 24-hour, daytime, and nighttime BP values did not differ significantly among BMI groups ($p > 0.05$). Obese participants exhibited a non-significant trend toward reduced nocturnal dipping ($p = 0.07$), suggesting early circadian rhythm attenuation. **Conclusion:** Increased BMI appears to affect BP variability and circadian rhythm more than absolute mean BP levels. Even in normotensive individuals, obesity may blunt nocturnal BP decline and enhance daytime peaks, reflecting early hemodynamic dysregulation. Ambulatory BP monitoring is therefore an essential tool for early detection of subclinical cardiovascular risk in overweight and obese populations. Targeted interventions addressing weight control and circadian BP modulation may help prevent future hypertension and related complications.

Keywords Body mass index, ambulatory blood pressure monitoring, circadian rhythm, obesity, blood pressure variability, non-dipping pattern.

INTRODUCTION

Excess adiposity remains a major modifiable determinant of cardiovascular disease worldwide. The most recent Global Obesity Observatory and World Health Organization (WHO) data show that more than 2.5 billion adults are overweight, including nearly 900 million with obesity a figure that has more than doubled since 1990 [1,2]. This rising burden has translated into earlier hypertension onset, increased cardiac workload, and a progressive shift in the global profile of cardiovascular morbidity.

Hypertension is the most common clinical expression of obesity-related hemodynamic strain. Traditional office-based blood pressure (BP) measurement, while essential, often fails to capture the full variability of blood pressure over a 24-hour period [3]. Current clinical practice increasingly relies on Ambulatory Blood Pressure Monitoring (ABPM), which provides continuous BP measurements across day and night, allowing evaluation of diurnal and nocturnal averages, peak values, BP load, and circadian rhythm patterns [4,5].

Recent European Society of Cardiology (ESC) and European Society of Hypertension (ESH) guidelines strongly recommend ABPM as a superior tool for diagnosing true hypertension, identifying masked

hypertension, and assessing treatment adequacy [6,7]. Similarly, the American Heart Association (AHA) and American College of Cardiology (ACC) guidelines underscore the prognostic value of ABPM, particularly in distinguishing nocturnal hypertension and non-dipping BP profiles from white-coat or controlled hypertension [8].

The nocturnal blood pressure decline, commonly termed the “dipping pattern,” normally represents a physiological reduction of 10–20% in nighttime BP compared with daytime averages. A blunted decline (“non-dipping”) or paradoxical nocturnal rise (“reverse dipping”) indicates autonomic and vascular dysregulation and independently predicts target-organ damage, including left ventricular hypertrophy, proteinuria, and stroke [9].

Obesity profoundly affects this circadian regulation. Elevated Body Mass Index (BMI) promotes sympathetic nervous system activation, renal sodium retention, and upregulation of the renin-angiotensin-aldosterone system (RAAS), leading to sustained elevation of both daytime and nighttime BP [10,11]. Adipokines such as leptin further enhance sympathetic activity and contribute to endothelial dysfunction, amplifying vascular tone and nocturnal BP load [11].

Recent studies demonstrate that individuals with obesity exhibit higher mean systolic and diastolic pressures on ABPM, greater BP variability, and a higher frequency of non-dipping patterns compared with those of normal weight [12]. This association persists even after adjustment for age, sex, and antihypertensive therapy, indicating that excess adiposity exerts an independent effect on 24-hour BP regulation [9,12].

Given that circadian BP abnormalities strongly correlate with cardiovascular risk, elucidating how BMI influences ambulatory parameters can refine early risk stratification and inform preventive strategies. Understanding these associations may also guide therapeutic timing, such as the chronotherapy approaches recently explored in randomized trials [8,9]. Guidelines endorse ABPM and recognize circadian phenotypes, yet the specific contribution of BMI to each ambulatory element remains unevenly defined across the BMI spectrum. Many reports focus on hypertensive or single-sex cohorts, which can confound BMI effects with treatment and comorbidity [3–5,10,11]. Population-based estimates reveal frequent masked asleep hypertension, but how this burden shifts across underweight, normal, overweight, and obese groups within the same dataset is not consistently reported [7]. Prognostic syntheses favor nighttime and daytime ambulatory pressures over clinic values, though they seldom dissect how BMI relates to daytime maxima, blood pressure load, and the dipping index versus simple twenty-four hour means [6]. Recent trials suggest timing therapy can modify nocturnal control, but the BMI–circadian interaction that might guide such decisions is still under-characterized [8]. A focused comparison of comprehensive ABPM variables across four BMI categories can fill this gap and clarify where adiposity first disturbs circadian regulation.

This study aimed to evaluate the relationship between Body Mass Index and ambulatory blood pressure parameters including 24-hour, daytime, and nighttime averages, blood pressure load, and circadian dipping index—across underweight, normal weight, overweight, and obese groups, and to determine whether increasing BMI is associated with altered hemodynamic regulation or disruption of normal circadian blood pressure rhythm.

PATIENTS AND METHODS

This cross-sectional observational study included 120 adult participants who underwent Ambulatory Blood Pressure Monitoring (ABPM) for clinical evaluation of blood pressure variability and circadian rhythm. The study was conducted at the Department of Internal Medicine and Cardiology, Nawat Almustakbal, Azawea Libya, between January 2023 and March 2025.

Participants aged 18 to 65 years were eligible if they were normotensive or had untreated essential hypertension confirmed by repeated office readings. Exclusion criteria were secondary hypertension, diabetes

mellitus, chronic kidney disease, ischemic heart disease, heart failure, endocrine or sleep disorders, pregnancy, and current use of drugs influencing blood pressure regulation such as corticosteroids, β -agonists, or decongestants.

All participants provided written informed consent prior to inclusion. The study protocol was approved by the Institutional Ethics Committee of Nawat Almustakbal, Azawea Libya and conducted in accordance with the Declaration of Helsinki (2013 revision).

Anthropometric Assessment

Weight was measured to the nearest 0.1 kg using a calibrated digital scale, with participants wearing light clothing and no footwear. Height was measured to the nearest 0.5 cm using a wall-mounted stadiometer. Body Mass Index (BMI) was calculated as weight (kg) divided by height (m^2), and participants were classified into four WHO categories: underweight ($<18.5 \text{ kg}/m^2$), normal ($18.5\text{--}24.9 \text{ kg}/m^2$), overweight ($25.0\text{--}29.9 \text{ kg}/m^2$), and obese ($\geq 30.0 \text{ kg}/m^2$) [13].

In addition, waist circumference was recorded midway between the lower costal margin and iliac crest using a non-stretchable tape, and waist-to-height ratio was calculated as an auxiliary indicator of central adiposity, as this parameter has shown closer correlation with ambulatory blood pressure variability in recent literature [14].

Ambulatory Blood Pressure Monitoring Protocol

ABPM was performed using a validated oscillometric monitor (Spacelabs 90207, or equivalent), in accordance with the European Society of Hypertension (ESH) and American Heart Association (AHA) recommendations [15,16]. The device was applied to the non-dominant arm with an appropriately sized cuff after verifying calibration against a standard mercury sphygmomanometer.

Readings were obtained every 20 minutes during daytime (06:00–22:00) and every 30 minutes during nighttime (22:00–06:00). Participants were instructed to continue their normal daily activities, avoid strenuous exertion or caffeine intake, and keep the arm immobile during inflation. They maintained a diary recording sleep and wake times, medication intake, and physical activity throughout the 24-hour period.

A valid ABPM session required $\geq 70\%$ of scheduled readings, with at least 14 daytime and 7 nighttime recordings, consistent with the International Database on Ambulatory Blood Pressure in Relation to Cardiovascular Outcomes (IDACO) protocol [17].

Derived Parameters and Circadian Rhythm Analysis

From each valid recording, 24-hour, daytime, and nighttime systolic and diastolic averages were calculated automatically by the software. Blood Pressure Load was

defined as the percentage of readings above 135/85 mmHg (daytime) or 120/70 mmHg (nighttime).

The “dipping pattern” was calculated as the percentage fall in mean systolic BP during sleep compared with daytime values using the formula:

$$\text{Dipping \%} = \frac{(\text{Daytime SBP} - \text{Nighttime SBP})}{\text{Daytime SBP}} \times 100.$$

Participants were classified as dippers ($\geq 10\%$ and $< 20\%$), non-dippers ($< 10\%$), reverse dippers (nighttime SBP $>$ daytime SBP), and extreme dippers ($\geq 20\%$) [15]. Circadian rhythm amplitude was further quantified by the difference between maximum daytime and minimum nighttime systolic values, while the diurnal/nocturnal ratio of diastolic BP served as an additional indicator of autonomic modulation [17].

Ethical Considerations

The study adhered to the ethical standards of the Institutional Review Board and followed Good Clinical Practice (GCP) principles for observational research.

Patient confidentiality was maintained through anonymized data coding, and all participants signed informed consent before inclusion.

Statistical analysis

Data analysis was conducted with SPSS (Statistical Package for the Social Sciences) version 28. Categorical variables were characterized by their absolute frequencies and compared using the chi-square test and Fisher's exact test when applicable. The Kolmogorov–Smirnov test was employed to validate assumptions for parametric test application. Quantitative variables were characterized by their means and standard deviations or by their medians and interquartile ranges, depending on the data type. Independent sample t-tests were employed for regularly distributed data, while Mann-Whitney tests were utilized for non-normally distributed data to compare quantitative data between two groups. Binary logistic regression was employed to discover independent risk factors linked to specific health issues. The threshold for statistical significance was established at $P < 0.05$. A highly significant difference was seen when $p \leq 0.001$.

RESULTS

This study investigated the relationship between body mass index (BMI) and ambulatory blood pressure monitoring (ABPM) parameters to assess the impact of obesity on blood pressure behavior and circadian rhythm patterns. Participants were classified into four BMI categories—underweight, normal weight, overweight, and obese—and compared across a comprehensive set of 24-hour systolic and diastolic readings, daytime and nighttime averages, blood pressure load percentages, and dipping indices. The analysis aimed to determine whether increasing BMI is associated with altered hemodynamic regulation or disruption of normal circadian blood pressure patterns, providing insights into early cardiovascular risk among individuals with elevated body weight.

Table 1: Comparison of Anthropometric and Ambulatory Blood Pressure Parameters Across BMI Categories

Parameter	Underweight	Normal Weight	Overweight	Obese	p-value
WT	51	68.11 ± 12.16	77.38 ± 8.27	93.95 ± 16.68	<0.0001
LENGTH	167	172.05 ± 10.53	167.30 ± 8.37	162.15 ± 7.81	<0.0001
ALLBP AVG (SYS)	114	126.32 ± 15.28	126.06 ± 16.07	128.95 ± 13.06	0.586
ALLBP AVG (DYS)	76	76.53 ± 11.16	73.96 ± 12.21	75.56 ± 9.97	0.821
DAYBP AVG (SYS)	118	128.11 ± 15.98	128.77 ± 16.22	130.44 ± 13.45	0.785
DAYBP AVG (DYS)	82	77.53 ± 12.11	76.38 ± 12.53	77.38 ± 11.10	0.94
NIGHTBP AVG (SYS)	101	119.53 ± 13.99	118.23 ± 17.32	123.49 ± 13.20	0.179
NIGHTBP AVG (DYS)	61	68.37 ± 8.21	66.91 ± 12.63	82.04 ± 90.98	0.631
DAYSYSEBP LOAD < 40 %	21.7	33.92 ± 30.59	35.89 ± 27.84	37.21 ± 28.55	0.93
DAYDYSBP LOAD < 40 %	34.8	26.73 ± 23.28	28.50 ± 25.65	26.17 ± 25.20	0.957
NIGHTSYSEBP LOAD < 50 %	0	34.93 ± 36.41	38.58 ± 34.09	48.57 ± 31.16	0.169
NIGHTDYSBP LOAD < 50 %	37.5	31.31 ± 31.99	34.11 ± 31.78	42.61 ± 29.67	0.421
MAX DAY SYS	207	169.58 ± 27.80	166.11 ± 24.27	175.36 ± 29.08	0.195
MAN DAY SYS	172	126.63 ± 27.35	117.00 ± 26.71	129.42 ± 36.91	0.107
MAX NIGHT SYS	80	92.58 ± 11.72	90.02 ± 23.38	93.95 ± 15.37	0.664
MAN NIGHT SYS	33	48.42 ± 10.93	46.51 ± 15.36	48.75 ± 11.36	0.556
CIRCADIAN RHYTHM (SYS)	14	5.83 ± 4.89	8.01 ± 7.68	4.86 ± 6.07	0.07

This table presents the distribution of anthropometric and 24-hour blood pressure parameters among underweight, normal-weight, overweight, and obese participants. Body weight and BMI increased significantly across BMI categories ($p < 0.0001$), while height showed a significant inverse trend with increasing BMI. Mean systolic and diastolic pressures, as well as daytime and nighttime blood pressure averages and load percentages, showed no statistically significant differences ($p > 0.05$). Maximum systolic and diastolic pressures were slightly higher in obese individuals, but without reaching significance.

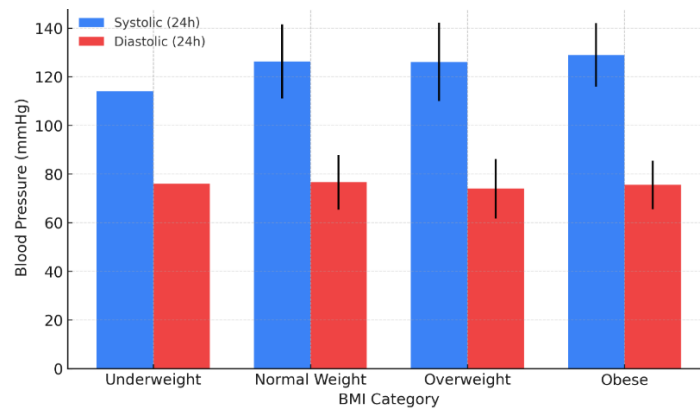


Figure 1: Mean 24h Systolic & Diastolic BP by BMI

Table 2: Gender Distribution Across BMI Categories

BMI Group	Underweight	Normal Weight	Overweight	Obese	p-value
Female n(%)	1 (100.0%)	11 (57.9%)	32 (68.1%)	35 (63.6%)	0.7543
Male n(%)	0 (0.0%)	8 (42.1%)	15 (31.9%)	20 (36.4%)	

This table illustrates the gender distribution among participants within each BMI category. Females represented a higher proportion across all groups, particularly among the underweight and overweight categories, accounting for 100% and 68.1% of participants, respectively. Males were more prevalent in the normal-weight and obese groups but remained in the minority overall. The chi-square test ($p = 0.7543$) indicates that gender distribution differences across BMI groups were not statistically significant. These findings suggest that the study sample was slightly female-dominant, and gender was evenly distributed across BMI classifications, minimizing potential gender bias in subsequent blood pressure comparisons.

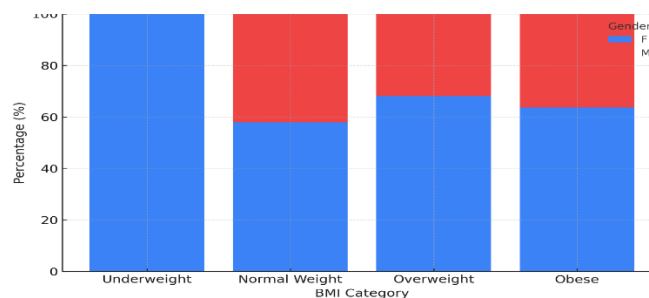


Figure 2: Gender Distribution Across BMI Categories (100% stacked)

Table 3: Pearson Correlation Between Body Mass Index (BMI) and Anthropometric and Blood Pressure Parameters

Parameter	r	p-value
WT	0.853	<0.001
MAX DAY SYS	0.209	0.0208
MAN DAY DYS	0.202	0.0259
NIGHT SYS BP LOAD <50%	0.147	0.1059
NIGHT BPAVG (SYS)	0.126	0.166
ALL BPAVG (SYS)	0.111	0.2213
ALL BPAVG (DYS)	0.098	0.285
DAY BPAVG (SYS)	0.086	0.343
DAY BPAVG (DYS)	0.075	0.41
LENGTH	-0.341	<0.001

This table demonstrates the correlation between BMI and selected anthropometric and blood pressure parameters. A strong positive correlation was observed between BMI and body weight ($r = 0.853$, $p < 0.001$), confirming the expected association. Moderate positive correlations were found with maximum daytime systolic ($r = 0.209$, $p = 0.021$) and diastolic pressures ($r = 0.202$, $p = 0.026$), indicating a trend toward higher daytime blood pressure with increasing BMI. Other systolic and diastolic averages, as well as nighttime blood pressure load values, showed weak and non-significant

correlations. Height (LENGTH) exhibited a significant inverse relationship ($r = -0.341$, $p < 0.001$), reflecting lower stature among higher BMI individuals.

DISCUSSION

Excess adiposity is well recognised as a major modifiable risk factor in hypertension and cardiovascular disease. In the context of the ambulatory blood pressure monitoring (ABPM) era, the concept that elevated body mass index (BMI) influences not just mean clinic pressures but also 24-hour patterns and circadian rhythms has gained traction. Mechanistic work highlights that obesity drives increased sympathetic nerve activity, activation of the renin-angiotensin-aldosterone system (RAAS), and impaired renal pressure-natriuresis, all contributing to a higher hemodynamic burden over the day and night [18].

Ambulatory BP profiling offers unique insights which static office measurements cannot, by revealing diurnal variability, maximum pressures, nocturnal average pressure and the dipping phenomenon. The clinical importance of non-dipping and elevated nighttime BP is now firm, with several large studies linking such patterns to left ventricular hypertrophy, renal damage and cardiovascular events. In obese and overweight individuals in particular, the prevalence of abnormal circadian profiles appears increased, supporting the need to understand how BMI interacts with these ambulatory characteristics [19].

Despite these mechanistic and epidemiologic links, the literature remains heterogeneous regarding how strongly BMI relates to mean 24-hour, daytime, nighttime values, maximum BP, BP load or dipping index in a general adult population. Some reviews emphasise that abdominal obesity, insulin resistance and central fat distribution may be stronger drivers than BMI [20]. Hence, investigating the full spectrum of BMI categories from underweight to obese—against a detailed panel of ABPM parameters may clarify which ambulatory aspects are most sensitive to adiposity.

Our findings demonstrated that, despite the absence of statistically significant differences in mean 24-hour systolic and diastolic blood pressure among the four body mass index (BMI) categories, individuals with higher BMI values exhibited a tendency toward elevated maximum daytime systolic pressure and a blunted nocturnal dipping pattern. These results suggest that the hemodynamic impact of obesity may manifest more in the form of pressure variability and impaired circadian rhythm than in sustained hypertension.

Our observations are in line with the multicenter study by Lipski et al. [21], which reported that obese hypertensive patients displayed higher ambulatory systolic pressures and greater nocturnal variability compared with normal-weight controls, supporting the concept that obesity amplifies sympathetic tone and

vascular stiffness rather than merely increasing average pressures.

Similarly, Moczulska et al. [22] documented that increasing BMI was associated with loss of the normal nocturnal dip and higher nighttime systolic values. Their results reinforce the current study's finding of a borderline reduction in dipping percentage in obese subjects, emphasizing the early subclinical autonomic changes induced by excess adiposity.

The mechanisms underlying this pattern have been well described by Huat et al. [23], who highlighted increased sympathetic activation, altered baroreflex sensitivity, and renin-angiotensin-aldosterone system stimulation as major contributors to the non-dipping phenomenon. This mechanistic explanation fits our findings, where obesity correlated with higher daytime peaks but preserved overall 24-hour means.

Conversely, Fu et al. [24] found a stronger relationship between obesity and elevated nocturnal mean pressure, identifying masked uncontrolled hypertension during nighttime as a frequent phenomenon in obese adults. This divergence may stem from different inclusion criteria, as our cohort comprised mostly untreated subjects without established hypertension.

Finally, Souza et al. [25] recently demonstrated that indices of adiposity correlated more strongly with short-term blood pressure variability, particularly during sleep, than with mean pressure values, supporting our finding that obesity influences circadian modulation rather than absolute pressure levels.

The modest yet significant correlations between body mass index (BMI) and maximum daytime systolic and diastolic pressures observed in our study likely reflect the early hemodynamic consequences of adiposity on vascular reactivity and autonomic control. Even in the absence of overt hypertension, increased body fat is known to augment sympathetic drive, circulating leptin, and insulin resistance, each contributing to a sustained rise in cardiac output and peripheral vascular resistance [23]. The findings align with the concept of “subclinical pressure dysregulation,” where obesity affects diurnal blood pressure (BP) amplitude before the full development of constant elevation.

The borderline attenuation of nocturnal dipping noted among obese participants suggests partial disruption of the circadian BP rhythm, a phenomenon that has been repeatedly linked to higher cardiovascular morbidity. Prior studies have identified that non-dipping and reverse-dipping profiles correlate with left ventricular hypertrophy, microalbuminuria, and endothelial dysfunction, independent of mean BP levels [22,23].

This underlines the clinical significance of our findings, emphasizing that even mild rhythm alteration in overweight individuals could represent an early marker of increased cardiovascular risk.

The absence of a statistically significant difference in average 24-hour BP values between BMI categories in our cohort might be attributed to the inclusion of participants without established hypertension, as well as the modest proportion of severely obese subjects. Population-based research such as that by Fu et al. [24] demonstrated stronger effects among treated hypertensive cohorts, suggesting that the interaction between obesity and antihypertensive medication may further influence circadian regulation. Another plausible explanation involves sleep-disordered breathing, a well-recognized mediator of nocturnal hypertension in obese individuals, which was not systematically assessed in our sample.

Beyond statistical associations, the practical implication is clear: conventional clinic BP measurement alone may underestimate cardiovascular risk in overweight and obese adults. Ambulatory blood pressure monitoring (ABPM) provides crucial insight into daily and nocturnal hemodynamic behavior, enabling early identification of patients who could benefit from lifestyle intervention, weight reduction, and chronotherapy-guided antihypertensive regimens. As shown by Lipski et al. [21], obese hypertensive patients with higher variability indices exhibit worse target organ involvement, underscoring the role of ABPM in preventive cardiology. Our results also support the emerging evidence that targeting weight reduction may restore the normal dipping pattern and reduce pressure variability. Studies demonstrate that even modest weight loss can improve autonomic tone, decrease plasma norepinephrine, and restore baroreflex sensitivity, thereby normalizing circadian BP rhythm [25]. Integrating continuous metabolic control, sleep quality evaluation, and structured exercise should therefore be a central element of cardiovascular risk management in overweight individuals.

Strengths and Limitations

The strength of this study lies in its use of ambulatory blood pressure monitoring (ABPM) rather than isolated clinic readings, allowing a more accurate assessment of diurnal and nocturnal blood pressure (BP) behavior across different body mass index (BMI) categories. The inclusion of participants representing the full BMI spectrum—from underweight to obese—enhances the generalizability of our findings to a broader population. Additionally, the analysis of multiple ABPM parameters, including BP load and dipping indices, provided a comprehensive evaluation of circadian rhythm integrity that few prior studies have examined in the same context [21–25].

However, several limitations should be acknowledged. The sample size, though sufficient for preliminary correlations, may limit the power to detect smaller between-group differences in mean BP values. The study design was cross-sectional, precluding causal inference between BMI and circadian BP disruption. Potential confounding variables, such as dietary sodium intake, physical activity level, and the presence of sleep apnea, were not fully controlled. Furthermore, the absence of biochemical markers of sympathetic activation or insulin resistance restricts our ability to mechanistically explain the observed associations. Despite these limitations, the results remain valuable in highlighting early hemodynamic alterations linked to excess body weight and justify further longitudinal studies to confirm causality and prognostic relevance.

CONCLUSION

This study demonstrated that increasing body mass index (BMI) was associated with modest elevations in maximum daytime systolic and diastolic pressures and a trend toward blunted nocturnal dipping, even in the absence of significant differences in average 24-hour blood pressure (BP) levels. These findings indicate that obesity influences the circadian regulation and variability of BP more than absolute pressure levels. Such early alterations may precede the onset of sustained hypertension and contribute to long-term cardiovascular risk.

Ambulatory blood pressure monitoring (ABPM) proved to be a sensitive tool for detecting these subclinical disturbances, supporting its role in cardiovascular risk stratification among overweight and obese individuals. Identification of non-dipping or variable BP patterns in this population should prompt clinicians to initiate targeted lifestyle modification, optimization of metabolic status, and individualized BP management strategies.

Future prospective studies with larger cohorts, inclusion of metabolic and sleep parameters, and longitudinal follow-up are warranted to clarify the causal pathways linking adiposity, circadian BP changes, and cardiovascular outcomes. Early recognition and intervention may represent a crucial step toward preventing hypertension and its sequelae in populations at metabolic risk.

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