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Research paper

Nur Fitriani[1], Syakib Bakri[1], Hasyim Kasim[1], Haerani Rasyid[1], Sitti Rabiul Zatalia[1], Nasrum Machmud[1], Ahkyar Albaar[1], Andi Makbul Aman[2], Pendrik Tendean[3], Faridin Pango[4], Ilham Jaya Pattelongi[5]

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Effect of obesity and blood pressure dipping status on left ventricular mass in normotensive young adults

[1]Nephrology Hypertension Division, Department of Internal Medicine, Faculty of Medicine Hasanuddin University, Makassar, Indonesia
[2]Endocrinology and Metabolic Disease Division, Department of Internal Medicine, Faculty of Medicine Hasanuddin University, Makassar, Indonesia
[3]Cardiovascular Division, Department of Internal Medicine, Faculty of Medicine Hasanuddin University, Makassar, Indonesia
[4]Rheumatology Division, Department of Internal Medicine, Faculty of Medicine Hasanuddin University, Makassar, Indonesia
[5]Department of Physiology, Faculty of Medicine Hasanuddin University, Makassar, Indonesia

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Abstract. *Despite the established links between obesity, blood pressure variability, and cardiovascular risks, the specific impact of the interaction between obesity and non-dipping blood pressure status on left ventricular mass (LVM) in normotensive individuals remains underexplored. Most studies have focused on hypertensive populations, leaving a critical gap in understanding how these factors contribute to cardiovascular changes in normotensive young adults. This study aims to address this gap by examining the combined effects of obesity and blood pressure dipping status on LVM in a normotensive cohort of young subjects.*
Methods: This cross-sectional study was conducted at Hasanuddin University Hospital and Central General Hospital (RSUP) Dr. Wahidin Sudirohusodo from March 2023, with a sample of 63 subjects divided into 32 dippers and 31 non-dippers. Interviews and ambulatory blood pressure monitoring (ABPM) were used to collect blood pressure data and history of hypertension. LVM measurements were obtained via echocardiography. Data were analyzed using SPSS version 25, with statistical significance set at $p<0.05$.
Results: LVM was significantly greater in males, obese individuals, and non-dippers compared to females, non-obese individuals, and dippers (178.2±141.6 g vs. 102.68±32.1 g; 156.3±124.3 g vs. 101.39±26.02 g; 150.5±118.6 g vs. 103.5±38.4 g with $p=0.029$, $p=0.026$, $p=0.037$, respectively). Both non-dipping status and obesity significantly affected LVM, with odds ratios of 4.27 and 3.31, respectively ($p<0.05$). Non-dipping status was the dominant factor affecting LVM, with the lowest risk observed in the dipping and non-obese group (OR=1.00).
Conclusion: Obesity and non-dipping blood pressure status, and their interaction, increase LVM. It is necessary to control obesity in young adults even if they are normotensive.
Keywords: *obesity, blood pressure dipping status, left ventricular mass.*

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Correspondence should be addressed to Nur Fitriani: lilyaae@gmail.com

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Нур Фітріані[1], Сякіб Бакрі[1], Хасим Касім[1], Хаерані Расід[1], Сітті Рабіул Заталія[1], Насрум Мачмуд[1], Ахкяр Альбаар[1], Анді Макбул Аман[2], Пендрік Тендіан[3], Фарідін Панго[4], Ільхам Джая Паттелонгі[5]

Вплив ожиріння та нічного зниження артеріального тиску на масу лівого шлуночка у молодих людей з нормотензією

[1]Відділення нефрологічної гіпертензії, кафедра внутрішньої медицини, медичний факультет Університету Хасануддіна, Макассар, Індонезія

[2]Відділення ендокринології та метаболічних захворювань, кафедра внутрішньої медицини, медичний факультет Університету Хасануддіна, Макассар, Індонезія

[3]Відділення серцево-судинних захворювань, кафедра внутрішньої медицини, медичний факультет Університету Хасануддіна, Макассар, Індонезія

[4]Відділення ревматології, кафедра внутрішньої медицини, медичний факультет Університету Хасануддіна, Макассар, Індонезія

[5]Кафедра фізіології, медичний факультет Університету Хасануддіна, Макассар, Індонезія

Резюме. *Незважаючи на встановлені зв'язки між ожирінням, варіабельністю артеріального тиску та серцево-судинними ризиками, конкретний вплив взаємодії між ожирінням і варіабельністю артеріального тиску на масу лівого шлуночка (МЛШ) у людей з нормальним тиском залишається недостатньо вивченим. Більшість досліджень зосереджено на групах пацієнтів з гіпертонією, залишаючи критичну прогалину в розумінні того, як ці фактори сприяють серцево-судинним змінам у молодих людей з нормальним тиском. Метою цього дослідження було вивчити вплив ожиріння та нічного зниження артеріального тиску на МЛШ у когорті молодих людей з нормотензією.*

Методи. Це поперечне дослідження було проведено в університетській лікарні Хасануддіна та Центральній загальній лікарні (RSUP) доктора Вахідіна Судірогусодо за участі 63 умовно-здорових суб'єктів. Учасники були стратифіковані за зниженням нічного артеріального тиску на 2 групи: діппер (n = 32) та нон-діппер (n = 31). Для збору даних щодо артеріального тиску використовували опитування та амбулаторний моніторинг артеріального тиску (АВРМ). Вимірювання МЛШ здійснювалися за допомогою ехокардіографії. Дані аналізували за допомогою SPSS версії 25, статистична значущість встановлювалась при p<0.05.

Результати. МЛШ була значно більшою у чоловіків (178.2 ± 141.6 г проти 102.68 ± 32.1 г; p = 0.029), осіб з ожирінням (156.3±124.3 г проти 101.39±26.02 г; p = 0.026) та нон-діпперів (150.5 ± 118.6 г проти 103.5 ±3 8.4 г; p = 0.037). Статус «нон-діппер» та ожиріння мали статистично значущий евект на МЛШ з відношенням шансів 4.27 і 3.31 відповідно (p < 0.05).

Висновки. Ожиріння, «нон-діппер» статус артеріального тиску та їх поєднання достовірно збільшують МЛШ, що свідчить про необхідність контролю маси тіла навіть у нормотензивних молодих людей.

Ключові слова: *ожиріння, спад артеріального тиску, маса лівого шлуночка.*

Introduction. Obesity is excessive fat accumulation due to an imbalance in energy intake (energy intake) with energy used for a long time. The World Health Organization (WHO) sets a body mass index (BMI) cutoff of > 25 for the obesity category in Asian adults Obesity is found in adults, adolescents, and children [1]. More than 1.4 billion adults are overweight and more than 500 million adults in the world are obese. Data from Laporan Nasional Riset Kesehatan Dasar (Riskesdas) 2008 in Indonesia shows that the prevalence of central obesity (waist circumference for men >90cm and women >80 cm) is 18.8%, whereas in 2013 it was 26.6%; BMI >25 kg/cm2 at age >18 years in 2007 was 13.9% and in 2013 it was 19.7% [2].

Nur Fitriani:
lilyaae@gmail.com

Obesity is associated with risk factors for hypertension where obese individuals continue to increase and become an epidemic, along with excessive eating patterns and lack of physical activity. Every 10% increase in body weight will increase blood pressure by 6.5 mmHg. Obesity is not only a risk factor for hypertension, but also a direct risk factor for damage to the kidneys, brain, and heart [3]. The Framingham Study showed that 78% of male hypertension sufferers and 65% of female hypertension sufferers were directly related to obesity. The risk of hypertension increases up to 2.6 times in obese men and 2.2 times in obese women compared to individuals with normal weight [4].

Hypertension (HT) is an important health problem because its prevalence is increasing with implications for increased morbidity and mortality. A collaborative meta-analysis study showed that research shows that blood pressure (BP) levels are associated with cardiovascular (CV) risk, where the higher the BP, the greater the risk. Starting from a systolic blood pressure (SBP) of 115 and a diastolic blood pressure (DBP) of 75 mmHg, if the BP increases more than 20 mmHg at the SBP level or 10 mmHg BP at the DBP level, it results in a twofold increase in CV risk [5].

Data from The Third National Health Nutrition and Examination Survey (NHANES III) in 2003 showed a significant linear relationship between increasing BMI, DBP, and pulse pressure in the American population [6]. The circadian pattern during the 24 hours in normotensive adults is characterized by a tendency for BP to be higher during the morning to midday and lower at night to early morning. Nighttime BP decreases from 10% to 20% of daytime BP. This variability is different for everyone, which can occur in normotensive subjects or hypertensive. BP variability is a physiological condition based on hormonal, environmental, and activity stimuli [7].

Hypertension is classified based on several categories. The American College of Cardiology (ACC) and the American Heart Association (AHA) set SBP >130 mmHg and DBP >80 mmHg as criteria for hypertension [8]. Meanwhile, the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC) SBP >140 mmHg and or DBP >90 mmHg as hypertension [9].

Increased BP and the presence of BP variability can be associated with increased cardiovascular risk and damage to target organs in the cardiovascular system [10-12], and the degree of damage to these target organs will increase as BP variability increases over 24 hours [12-14]. BP variability refers to changes in BP that can occur in the short term (24 hours) and long term, which is characterized by circadian variations in BP both during the day and at night. The pattern of decreasing BP at night is classified into 4 groups, namely dipping, non-dipping, riser (reverse dipping), and extreme dipping patterns [8]. Based on the ratio of decreasing SBP at night and during the day it can be categorized into reverse dipping (>1.0), non-dipping (0 .9-1.0), dipping (0.8-0.9), and extreme dipping (≤0.8) [7, 15].

A systematic review involving 23,856 hypertensive patients and 9641 normotensive subjects from Asia, Europe, and South America in 2011 found that BP variability at night was associated with an increased risk of cardiovascular mortality and morbidity in hypertensive patients and healthy individuals [16]. Mancia et al. in 2001 stated that subjects who experienced a decrease in BP of less than 10% at night showed more severe target organ damage and a higher incidence of cardiovascular disease when compared with subjects who experienced a decrease in BP of more than 10% at night [17].

Ambulatory blood pressure monitoring (ABPM) can assess circadian BP patterns, namely patterns in the morning, evening, and 24-hour average blood pressure which are not detected in regular blood pressure checks in the clinic. Population-based and clinical studies using ABPM show that nighttime blood pressure is a better predictor than daytime blood pressure. An increase in systolic blood pressure is associated with the incidence of left ventricular hypertrophy (LVH), which is a condition that is a predictor of CV events [18-20]. LVH is damage to the target organ of the heart with a higher prevalence in people with hypertension. An increase in left ventricular mass (LVM) is caused by the thickening of the left ventricular wall as a compensatory mechanism to minimize wall stress in response to increased blood pressure. LVM is one of the echocardiographic parameters used to diagnose LVH [21].

Overweight and obesity are additional cardiovascular risk factors that can lead to left ventricular remodeling. Several studies examining obese patients or research participants with metabolic syndrome found an unfavorable influence of obesity on left ventricular mechanics. Research conducted to examine the effect of the interaction between obesity and dipping and non-dipping status on LVM in HT subjects has been widely reported, but the effect on normotensive subjects is still very limited. This study is done to learn the effects of obesity and blood pressure dipping status on LVM in a normotensive cohort of young subjects.

Methods. This cross-sectional study was conducted at Hasanuddin University Hospital and the Integrated Heart Center of Central General Hospital (RSUP) Dr. Wahidin Sudirohusodo in Makassar from March 2023 to November 2023. The research was carried out after obtaining ethical approval from the Ethics Committee for Biomedical Research on Humans, Faculty of Medicine, Hasanuddin University, Makassar with approval number 923/UN4.6.4.5.31/PP36/2023. Informed consent was also obtained from participants for their participation in the study.

A total of 63 normotensive adult subjects, divided into 32 dippers and 31 non-dippers, were recruited through consecutive sampling. The inclusion criteria for participation were: (1) age range 25-40 years, (2) non-diabetic, and (3) willingness to participate in the study. Subjects were excluded if they had a history of hypertension, were pregnant, or had a history of congenital heart disease.

The research involved interviewing all subjects to gather information about their hypertension history, ensuring they met the inclusion and exclusion criteria, and obtaining informed consent. Subsequently, ABPM measurements were performed using a BTL-08 ABPM device by BTL Industries Ltd. The cuff was placed on the non-dominant arm, and blood pressure was recorded every 30-60 minutes. After 24 hours, the data were downloaded and processed using the Cardiopro application.

Daytime and nighttime average SBP and DBP were calculated based on ABPM readings. The IDACO criteria defined a complete ABPM period as having at least 10 daytime (10 AM–8 PM) and 5 nighttime (12 AM–6 AM) SBP and DBP measurements. Mean daytime and nighttime SBP and DBP were calculated by averaging the respective readings. The mean 24-hour blood pressure was determined by averaging all available BP measurements from ABPM. The nighttime-to-daytime SBP ratio was defined as mean nighttime SBP divided by mean daytime SBP.

Dipping patterns were categorized based on the nighttime to daytime SBP ratio: dipping pattern (≤0.90) and nondipping pattern (>0.90). Participants with a reverse dipping pattern (≥1.0) were included in the nondipping pattern category due to the small sample size (n=19).

Subjects were instructed to maintain normal daily activities, remain calm during measurements, avoid exercising or driving during pumping, press the drug button if engaging in excessive activity, and avoid medications that could affect blood pressure.

LVM measurements were obtained through transthoracic echocardiography at the Integrated Heart Center of Dr. RSUP. Wahidin Sudirohusodo Makassar, using the GE Vivid E95 device. Parameters assessed included left ventricular dimensions such as left ventricular end-diastolic diameter (LVEDD), interventricular septal thickness (IVSD), and posterior wall diameter (PWD).

SPSS version 25 (Armonk, NY: IBM Corp) was used for data analysis, employing descriptive methods and statistical tests. The descriptive method provided general information on the research sample, while the statistical method calculated mean values, standard deviation, and frequency distribution. Statistical tests utilized the student's t-test for normally distributed data and the Mann-Whitney U test for non-normally distributed data, comparing variations in blood pressure and left ventricular mass. Further analysis involved univariate and multivariate logistic regression to identify factors influencing LVM. The results, expressed as Odds Ratio (OR) with a 95% confidence interval (95% CI), will be presented in narrative form, accompanied by tables and figures. Statistical significance was determined at a p-value of <0.05.

Results. Among the study cohort, there were 20 men (31.7%) and 43 women (68.3%) aged 25-39, with a mean age of 30.63 + 3.04 years. Subject characteristics are presented in Table 1.

Table 1

Study subject characteristics

Variable	Descriptive statistics	
	n (%)	(Mean ± SD)
Sex Male Female	 20 (31,7) 43 (68,3)	
Age, year	63 (100)	30.63 ± 3.04
BMI Obese Non-Obese	 34 (54) 29 (46)	
BP variability Dipping Non-dipping	 32(50,8) 31(49,2)	
24 hours mean BP, mmHg		
SBP	63 (100)	117.9 ± 10.9
DBP	63 (100)	72.95 ± 8.5
Night-time BP, mmHg		
SBP	63 (100)	117.36 ± 13.5
DBP	63 (100)	72.5 ± 10.9
Daytime BP, mmHg		
SBP	63 (100)	117.3 ± 9.4
DBP	63 (100)	72.3 ± 8.3
LVM, gram	63 (100)	126.6 ± 90.03

The data revealed that 54% (n = 34) of the subjects were obese, while 46% (n = 29) were non-obese. Regarding blood pressure, 50.8% (n = 32) were classified as dipping, and 49.2% (n = 31) were non-dipping. Analysis of 24-hour blood pressure monitoring indicated that the average night-time SBP ranged

from 94-147 mmHg (mean 117.36 ± 13.5 mmHg), while the daytime SBP ranged from 96-146 mmHg (mean 117.3 ± 9.4 mmHg). The minimum SBP was 72-127 mmHg (mean 94.57 ± 11.25 mmHg), and the maximum SBP ranged from 120-178 mmHg (mean 139.13 ± 11.21 mmHg). The daytime DBP ranged from 56-91 mmHg (mean 72.3 ± 8.3 mmHg), the minimum DBP ranged from 36-86 mmHg (mean 57.24 ± 9.4 mmHg), and the maximum DBP ranged from 67-111 mmHg (mean 88.89 ± 9.8 mmHg). The LVM for all subjects ranged from 53.4-763 g (mean 126.67 ± 90.03 g).

Further analysis showed that LVM was significantly greater in males, obese individuals, and the non-dipping group compared to females, non-obese individuals, and the dipping group (Table 2).

Table 2

Comparison of LVM based on sex, obesity, and BP dipping status

Variable	n	LVM (Mean ± SD)	p-value
Sex			
Male	20	178.2 ± 141.6	0.029
Female	43	102.68 ± 32.1	
Obesity			
Obese	29	156.3 ± 124.3	0.026
Non-Obese	34	101.39 ± 26.02	
BP Variability			
Dipping	32	103.5 ± 38.4	0.037
Non-dipping	31	150.5 ± 118.6	

The impact of blood pressure dipping status and obesity on LVM was examined by categorizing LVM based on median values for males (147.02 g) and females (101.03 g). This allowed for multivariate analysis using logistic regression. Table 3 displays the results of the regression analysis, showing the relationship between gender, dipping status, obesity, and LVM category (> median and ≤ median).

Table 3

Univariate and multivariate logistic regression analysis factors influencing LVM

Variable	Univariate analysis			Multivariate Analysis		
	Crude OR	95%CI	p-value	Crude OR	95%CI	p-value
Sex (M/F)	0.955	0.33-2.76	1.000			
Obese (Yes/No)	3.07	1.09-8.61	0.057	3.31	1.09-10.02	0.004
Dipping (ND/D)	4.00	1.41- 11.44	0.007	4.27	1.42-12.86	0.010

R2=22.9%

As presented in Table 3, dipping status and obesity had an impact on LVM. Subsequent adjustments for LVM changes revealed that both non-dipping status and obesity significantly affected LVM, with odds ratios of 4.27 and 3.31 respectively ($p<0.05$).

Next, an analysis of the interaction effect of dipping status and obesity on left ventricular mass (> median and ≤ median) was carried out by grouping subjects based on a combination of blood pressure dipping status (dipping or non-dipping) and obesity (obese or non-obese). Table 4 reveals the impact of dipping status and obesity on changes in LVM.

Table 4

Association between dipping status and obesity with LVM

Category	LVM		OR	95% CI
	> median	≤ median		
	n (%)	n (%)		
Dipping non-obese	4(22.2)	14(77.8)	1.00	-
Dipping obese	7(46.7)	8(53.3)	3.06	0.68-13.79
Non-dipping non-obese	9(60.0)	6(40.0)	5.25	1.15-23.94
Non-dipping obese	12(80.0)	3(20.0)	14.00	2.60-75.41

LVM (> median and ≤ median)

As shown in Table 4, non-dipping is the dominant factor, with the lowest risk observed in the dipping+non-obese group (OR=1.00). The dipping+obese group has a higher risk (OR=3.05), followed by non-dipping+non-obese (OR=5.25), and the highest risk is found in the non-dipping+obese group (OR=14.00). Obesity alone does not pose a significant risk, but non-dipping BP status has a significant effect (OR=5.25, 95%CI=1.15-23.94). This risk increases to 14.00 when obese individuals have non-dipping status compared to those with dipping status without obesity.

Discussion. This study evaluated the impact of the interaction between obesity and BP dipping status on LVM in normotensive young adult subjects. This research was conducted on 63 adult subjects consisting of 20 men (31.7%) and 43 women (68.3%) with an age range of 25-39 years with a mean of (30.63+3.04) years. Obese subjects 34 (54%) and non-obese 29 (46%), dipping 32 (50.8%), non-dipping 31 (49.2%). The 24-hour BP monitoring using the ABPM method obtained a SBP range ranging from 96-148 mmHg (117.9+10.9 mmHg), and an average DBP between 57-87 mmHg (72.95+8.5 mmHg).

In this study, it was found that obesity influenced LVM. It was found that LVM was significantly greater in the obese group compared to the non-obese group (150.5+118.6 g vs 103.5+38.4 g). Previous studies reported the same results. Rider et al. in 2009 reported that obesity was significantly associated with increased LVM (126±27 in obese subjects compared to 90±20 g in non-obese subjects; p<0.001) [22], Avelar et al. in 2007 found in severely obese subjects 234±65 g compared to 160±38 g in non-obese [23] and linear regression analysis conducted by Maugeri et al. in 2019 found that increasing BMI was an independent predictor of increasing LVM [24]. This may be related to the interaction between systemic hypertension and pressure overload in obese individuals which is reported to produce an exponential effect on the prevalence of LVH. The effect of increasing BP on changes in LVM appears to be significantly greater in obese individuals compared to non-obese individuals.

Obesity leads to excessive adipose accumulation, resulting in increased blood volume and cardiac output. The increase in cardiac output is due to a rise in stroke volume, as heart rate remains unchanged with weight gain. This causes systemic vascular resistance to decrease, leading to left ventricular dilatation. Recent research has found TNF alpha receptors in heart muscle, explaining how obesity can cause LVH without hypertension. Increased secretion of TNF alpha by fat cells in obesity leads to its binding to myocardial receptors, triggering reactions such as myocyte apoptosis and fibrosis, ultimately resulting in LVH [25].

BP variability can be divided into several categories based on the ratio of night-time to daytime SBP, namely into the dipping category if the SBP ratio is ≤0.9, non-dipping if the SBP ratio is >0.9-1.0, and reverse dipping if the ratio SBP > 1.0 [26]. Based on the results of measuring the average daily BP for 24 hours, 32 subjects in this study (50.8%) showed BP variability with a dipping pattern, while the remaining 31 subjects (49.2%) showed a non-dipping pattern.

This study also found that non-dipping subjects had LVM values that were greater and statistically significant than dipping subjects (150.5+118.6 g vs 103.5+38.4 g; p=0.037) thus indicating the possibility of an influence from 24-hour VTD on changes in LVM in young adults in healthy conditions. Previous research also obtained similar results, including research from Radhakrishna et al. who reported a significantly greater mean LVM in non-dipping subjects when compared to dipping subjects (217.7±59.3 compared to 197.6±54 g; p=0.011) [26].

The cause of the increase in LVM is not fully understood, but it is believed to be linked to heightened blood pressure and various changes in blood vessels like increased tension, muscle cell growth, reduced artery elasticity, and increased aortic stiffness. As a result, the heart muscle is burdened with a sustained workload [27]. Research has shown that LVM increases in response to a higher heart workload due to disturbances in daily changes or vascular tone dysfunction (VTD) [27]. Hendriks et al. (2019) found that there is a direct relationship between high blood pressure (SBP) and increased LVM, with a 10 mmHg increase in SBP estimated to raise LVM by 4.01g [28].

This study found a link between VTD and changes in LVM, establishing the effectiveness of 24-hour BP monitoring in detecting night-time BP changes and

identifying early CV risk in healthy individuals without symptoms. This allows at-risk individuals to take prompt preventive actions, reducing future cardiovascular disease-related morbidity and mortality.

After categorizing changes in LVM based on gender, multivariate analysis showed that BP dipping status and obesity independently influenced LVM with a significant impact ($p<0.05$). The odds ratio (OR) for blood pressure dipping status was 4.27 and for obesity was 3.31. Another study by Kim et al. in 2017 also found that non-dipping status and obesity were independent factors contributing to increased left ventricular mass, with OR values of 2.134 and 3.694, respectively [29].

Further analysis was conducted to assess the risk of increased left ventricular mass based on dipping status and obesity. The reference group comprised non-obese subjects who experienced dipping (OR=1.00). The lowest risk increase (OR=3.06) was observed in obese subjects with dipping status. Non-obese subjects with non-dipping status exhibited a higher risk increase (OR=5.25), while the highest risk increase (OR=14.00) was seen in obese subjects with non-dipping status, compared to the reference group.

The analysis reveals that the combination of obesity and non-dipping status significantly increases the risk of LVM changes. This can be attributed to the impact of obesity and insulin resistance, which activate the renin-angiotensin-aldosterone system, sympathetic tone, and salt sensitivity. These factors ultimately lead to endothelial dysfunction, arterial stiffness, and elevated blood pressure [30]. Previous studies have also demonstrated a link between obesity and reduced nocturnal dipping. For instance, Cuspidi et al. (2013) reported a 15% lower prevalence of nocturnal systolic blood pressure reduction in obese individuals compared to non-obese individuals [31]. Similarly, Talalaj et al. (2023) found lower levels of nocturnal dipping in obese subjects [32], and Miazgowski et al. (2019) identified a significant correlation between visceral fat and nocturnal dipping levels [33].

This study shows that non-obese-non-dipping subjects are more at risk of experiencing LVM changes than obese-dipping subjects which shows that obesity does not significantly increase the risk of LVM changes without non-dipping status, and non-dipping status can have a more significant influence on LVM risk even though without being accompanied by obesity.

Previous research also found a similar interaction between obesity and blood pressure in causing left ventricular changes. Maugeri et al. in 2019 found that BMI and SBP were the main independent predictors of changes in LVM, and these two variables could interact with each other because changes in systolic blood pressure were proven to have a greater effect on changes in LVM in obese subjects ($\beta=0.195$; $p=0.033$) compared to non-obese ($\beta=0.134$; $p=0.048$) [6, 24]. Chobanian et al. in 2003 also found that the effect size of increasing BP in causing left ventricular remodeling appeared to be greater in obese subjects compared to non-obese [7, 34]. In contrast, Maugeri et al in 2019 did not find any significant differences in LVM between obese and non-obese subjects who did not experience an increase in BP [24], while research from Cleva et al. in 2018 [35], Zhang et al in 2014 [36], and Maugeri et al in 2019 [24] found that increasing BP was proven to increase the risk of changes in LVM without being affected by BMI.

The results of this study and several previous studies show that the interaction between obesity status and the occurrence of BP dipping at night has the potential to influence changes in LVM although obesity cannot trigger a significant pathological remodeling process of the left ventricle without being accompanied by changes in BP variations while changes in BP variations can influence LVM is not affected by obesity. Interestingly, our study found that subjects with non-dipping status or the absence of a decrease in BP at night in normotensive young adults have a greater role in causing an increase in LVM when compared with obesity.

Our study has several limitations. Considering at least physical activity levels that could influence the study results, a small sample size, cross-sectional design, and limitations specific to the measurement methodology for addressing. There is a need for further analysis regarding physical activity measured by MeT level (type of activity x amount minute of activity x day/week) (routine and exercise) as well as a larger study sample size to increase the power of statistical tests.

Conclusions. In conclusion, LVM was found to be significantly greater in obese subjects compared to non-obese subjects, in non-dipping subjects than in dipping subjects, and the presence of obesity in non-dipping subjects will further increase LVM. Overall, obesity and non-dipping BP status and their interaction increase LVM. It is necessary to control obesity in young adult subjects even though they are normotensive. Further observation study is needed in obese subjects regarding the risk of LVH.

Ethics Statement. The research was carried out after obtaining ethical approval from the Ethics Committee for Biomedical Research on Humans, Faculty of Medicine, Hasanuddin University, Makassar with approval number 923/UN4.6.4.5.31/PP36/2023. Informed consent was also obtained from participants for their participation in the study.

Competing interest. The authors declare no conflict of interest.

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Data availability. Data is available upon reasonable request.

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