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ORIGINAL RESEARCH

Nighttime Systolic Blood Pressure Determined by Pulse Transit Time Predicts Cardiac Events in Patients With Heart Failure

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BACKGROUND: The optimal systolic blood pressure (SBP) target for heart failure (HF) patients has not been established. Although the association between daytime SBP and prognosis has been reported in patients with HF, the prognostic impact of nighttime SBP remains unclear.

METHODS: We conducted continuous nighttime SBP measurements noninvasively using pulse transit time in 366 patients with HF (median age, 72 years; male sex, 195), and followed up for cardiac events (HF hospitalization or cardiac death). Average values of nighttime pulse transit time –based SBP were used in the present study. The patients were divided into tertile groups based on nighttime pulse transit time–based SBP: high-SBP group (median SBP, 136 mm Hg; n=122), middle-SBP group (median SBP, 117 mm Hg; n=122), and low-SBP group (median SBP, 100 mm Hg; n=122).

RESULTS: During a median follow-up period of 1083 days after nighttime pulse transit time–based SBP measurement, 71 patients experienced a cardiac event. Kaplan–Meier analysis showed the highest incidence of cardiac events in the low-SBP group. Multivariate Cox proportional hazard analysis also showed that the lowest SBP tertile was associated with a higher risk of cardiac events compared with the highest SBP tertile as reference (hazard ratio, 2.100 [95% CI, 1.121–3.933]; *P*=0.021).

CONCLUSIONS: Low nighttime SBP was associated with an increased cardiac event rate in patients with HF.

Key Words: arterial stiffness ■ cardiac function ■ heart failure ■ nighttime blood pressure ■ pulse transit time

See Editorial by Chow et al.

Hypertension is a major risk factor for cardiovascular disease, the leading cause of death worldwide, and has been reported to have a strong causal relationship with an increased risk of cardiovascular disease.[1–5] In patients with hypertension, the effectiveness of blood pressure (BP)-lowering therapy in preventing cardiovascular disease and reducing death is well documented.[6,7] Long-standing elevated BP causes structural and functional changes in the heart, such as ventricular hypertrophy and diastolic

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CLINICAL PERSPECTIVE

What Is New?

- We conducted the first investigation into the association between nighttime systolic blood pressure and cardiac events in patients with heart failure, using a novel approach based on pulse transit time.

What Are the Clinical Implications?

- This study found that low nighttime systolic blood pressure is associated with an increased risk of cardiac events in patients with heart failure.
- The present study may help establish the target nighttime blood pressure for future heart failure management.

Nonstandard Abbreviations and Acronyms

CAVI	cardio-ankle vascular index
PTT	pulse transit time

dysfunction, subsequently leading to heart failure (HF).[8] As the end-stage manifestation of most forms of cardiovascular disease, HF is associated with substantial morbidity and death and has a considerable impact on health economies.[9] Hence, proper BP control in individuals with hypertension is crucial for preventing disease progression to HF.[10]

However, treating hypertension in patients with HF is complicated and challenging due to the lack of an established optimal BP target for such patients.[11,12] In addition, there are inconsistencies in the research findings regarding daytime BP levels and cardiovascular events in patients with HF. While some studies have shown an association between elevated daytime BP levels and reduced mortality rates in patients with HF,[13,14] others have indicated a link with higher rates of stroke and bleeding.[15] Furthermore, the prognostic impact of nighttime BP remains unclear in patients with HF.

Recently, pulse transit time (PTT) has been applied with a nonlinear algorithm to monitor beat-to-beat BP, using finger plethysmography.[16] PTT refers to the travel time between 2 arterial sites within the same cardiac cycle.[17] There are reports that mention the limitations of this method in BP estimation[18,19]; however, it has also been reported that systolic blood pressure (SBP) values using the PTT-based method and those measured by a cuff show a high correlation.[16] This innovative application, PTT-based BP measurement, has been reported to have advantages over cuff-based methods, such as continuous and noninvasive recording. However, little is known about its clinical utility, and the relationship between cardiac events and nighttime BP has not yet been fully elucidated in patients with HF. In the present study, we aimed to investigate the prognostic impact of nighttime PTT-based BP in patients with HF.

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Participants and Ethical Statement

We enrolled 379 consecutive patients with HF admitted to Fukushima Medical University Hospital who had undergone overnight sleep studies along with PTT-based BP monitoring between April 2018 and March 2022. PTT measurements were performed on consecutive patients with HF admitted to our hospital, except for those who could not be monitored continuously at night due to intensive care needs, dementia, and so on. The tests were conducted in a stable condition without the administration of oxygen, catecholamines, mechanical circulatory support, and the like. HF was diagnosed by independent cardiologists following the clinical practice guidelines.[12,20–22] We excluded patients receiving maintenance hemodialysis (n=13). Clinical information, including demographic data, laboratory data, and echocardiographic data, was collected in routine clinical practice. We also collected bilateral cardio-ankle vascular index (CAVI) data[23] and used the average of both sides as an indicator of arterial stiffness. The patients were divided into 3 groups based on nighttime PTT-based SBP in accordance with previous study[24]: the high-SBP group (median SBP, 136 mm Hg; n=122), middle-SBP group (median SBP, 117 mm Hg; n=122), and low-SBP group (median SBP, 100 mm Hg; n=122). We compared the clinical characteristics and prognoses among the 3 groups. The research protocol was approved by the Research Ethics Committee of Fukushima Medical University (No. 823), and written informed consent was obtained from all participants. The study adhered to the principles outlined in the Declaration of Helsinki.[25]

PTT-Based BP Monitoring for Beat-to-Beat BP Measurement

Overnight sleep studies were conducted using a type 3 polygraph system (SOMNOtouch RESP, Fukuda Denshi Co., Ltd., Tokyo, Japan) in combination with an ECG to monitor PTT.[26,27] This study used data obtained from the patients with HF for screening of

sleep-disordered breathing. PTT was defined as the time interval between the R wave on the ECG and the arrival of the pulse wave at the fingertip, measured via plethysmography.[16] In this study, we used the time commonly referred to as *pulse transit time*. However, a more accurate term would be *pulse arrival time*, as it includes the preejection period.[18] We opted to use the term *PTT* because it is labeled as such in the commercialized polygraph system used for this study. Following this, PTT-based beat-to-beat BP was measured using a specific formula implemented in DOMINO Light software version 1.5.0 (Somnomedics, Randersacker, Germany), which uses a patented algorithm (11/364174 US 2006/0217616 A1, 7 374 542), and was continuously recorded as described in a previous study.[16] Nighttime BP was defined as the BP measured from 10:00 PM to 6:00 AM the following morning. If the patient went to bed after 10:00 PM or woke up before 6:00 AM on the basis of their medical records, BP data from the estimated sleep period were used. Nighttime PTT-based BP was expressed as an average of systolic, diastolic, and mean BPs, each calculated from the total of all continuously measured beat-to-beat BP data. Daytime BP was measured manually for calibration using a cuff-based method at approximately 3:30 to 4:30 PM, with the patient in the supine position. We also examined BP dipping patterns from day to night, and classified them into 4 types: dipper (a 10%–20% decrease in nighttime BP compared with daytime levels, indicating a physiological phenomenon); extreme dipper (>20% decrease); nondipper (<10% decrease); and reverse dipper (increase in nighttime BP compared with daytime levels).[28]

Follow-Up and Clinical End Points

All patients were followed up annually through a comprehensive review of medical records at Fukushima Medical University Hospital or referral hospitals, or via direct phone calls to the patients or their families. The primary end point was defined as cardiac events, namely, HF hospitalization or cardiac death, and the secondary end point as HF hospitalization. HF hospitalization was defined by symptoms and signs necessitating urgent therapy, leading to hospitalization. Cardiac death was defined as death resulting from acute myocardial infarction, ventricular fibrillation, HF, or sudden cardiac death.

Statistical Analysis

To test for normality, the Shapiro–Wilk test was used. The statistical significance of differences in continuous variables was analyzed using the Kruskal–Wallis test and Steel–Dwass post hoc test. Categorical variables were analyzed using the χ^2 test. Cardiac event rates were compared using the Kaplan–Meier analysis with log-rank test. Cox proportional hazard analysis was applied to assess the predictive ability of PTT-based SBP for cardiac events. Hazard ratio was further adjusted for 3 models: model 1 (adjusted for age and sex), model 2 (additionally adjusted for age, sex, and other important prognostic factors in patients with HF, namely, B-type natriuretic peptide, estimated glomerular filtration rate, hemoglobin, and left ventricular ejection fraction), and model 3 (additionally adjusted for age, sex, B-type natriuretic peptide, estimated glomerular filtration rate, hemoglobin, and left ventricular ejection fraction, as well as use of β blockers, renin–angiotensin system inhibitors, mineralocorticoid receptor antagonists, loop diuretics, calcium channel blockers, and sodium–glucose cotransporter 2 inhibitors). There were no missing data in the adjusting variables. Statistical significance was established at $P<0.05$, and data analyses were carried out using IBM SPSS Statistics version 29.0 (IBM, Armonk, NY), or EZR version 1.61 (Saitama Medical Center, Jichi Medical University, Saitama, Japan), which is a graphical user interface for R version 4.1.2 (R Foundation for Statistical Computing, Vienna, Austria).[29]

RESULTS

The clinical characteristics of the nighttime PTT-based SBP groups are summarized in Table 1. Among the 366 cases, 241 had a daytime SBP of <130 mm Hg and a daytime diastolic BP of <80 mm Hg. Among the 3 groups, there was no significant difference in BP dipping patterns. The low-SBP group was the youngest. The prevalence of comorbidities showed no differences among the 3 groups, except for hypertension. A higher proportion of patients in the low-SBP group were prescribed β blockers, mineralocorticoid receptor antagonists and loop diuretics, while a lower proportion were prescribed calcium channel blockers ($P<0.05$ for all). The proportion of patients taking any antihypertensive medications including β blockers, renin–angiotensin system inhibitors, mineralocorticoid receptor antagonists, loop diuretics, and calcium channel blockers, in the evening or at bedtime did not differ among the 3 groups. With respect to laboratory and echocardiographic parameters, the low-SBP group showed the highest B-type natriuretic peptide level, as well as the largest left ventricular end-systolic volume, with the lowest left ventricular ejection fraction and stroke volume. In contrast, there was no significant difference in left ventricular end-diastolic volume and CAVI.

During a median follow-up period of 1084 days after nighttime PTT-based BP measurement, 71 patients experienced cardiac events (63 HF hospitalizations and 8 cardiac deaths). Kaplan–Meier analysis (Figure) showed the highest incidence of cardiac events and HF hospitalization in the low-SBP group.

In the multivariate Cox proportional hazard analysis (Table 2) showed an association between a 10-mm Hg

Table 1. Comparisons of Patients With Low, Middle, and High Nighttime PTT-Based SBP (n=366)

	Highest-SBP tertile	Middle-SBP tertile	Lowest-SBP tertile	*P* value
Nighttime PTT-based SBP (mm Hg)	136.0 (131.0–146.0)	117.0 (112.0–121.0)*	100.0 (93.0–105.0)*†	<0.001
Nighttime PTT-based DBP (mmHg)	75.3±13.6	68.0±10.4*	62.5±8.51*†	<0.001
Nighttime PTT-based MBP (mm Hg)	95.5 (90.0–102.0)	85.0 (79.0–89.0)*	74.5 (70.0–79.0)*†	<0.001
Nighttime PTT-based pulse pressure (mm Hg)	63.0 (54.0–72.0)	49.0 (40.0–56.0)*	36.0 (29.0–42.0)*†	<0.001
Dipping pattern, n (%)				0.855
Dipper	1 (0.8)	2 (1.6)	0 (0)	
Extreme dipper	0 (0)	1 (0.8)	1 (0.8)	
Nondipper	77 (63.1)	77 (63.1)	83 (68.0)	
Reverse dipper	42 (34.4)	40 (32.8)	37 (30.3)	
Daytime SBP (mm Hg)	137.0 (130.0–144.0)	118.0 (112.0–122.0)*	102.0 (94.0–106.0) *†	<0.001
Daytime DBP (mm Hg)	73.6±13.1	67.9±10.2*	63.0±7.76*†	<0.001
Daytime MBP (mm Hg)	95.3 (88.0–100.7)	83.5 (80.0–89.3)*	75.8 (71.3–81.0)*†	<0.001
Daytime SBP<130 and DBP<80, n (%)	23 (18.9)	98 (80.3)*	120 (98.4)*†	<0.001
Age, y	74.5 (68.0–81.0)	73.0 (63.0–80.8)	68.0 (54.5–75.0)*†	<0.001
Male sex, n (%)	59 (48.4)	68 (55.7)	68 (55.7)	0.411
Body mass index, kg/m^2	23.8 (21.2–26.9)	23.6 (20.4–26.1)	23.0 (20.8–26.0)	0.403
Comorbidities				
Hypertension, n (%)	92 (75.4)	78 (63.9)	61 (50.0)	<0.001
Diabetes, n (%)	44 (36.1)	51 (41.8)	48 (39.3)	0.654
Dyslipidemia, n (%)	71 (58.2)	76 (62.3)	77 (63.1)	0.700
Atrial fibrillation, n (%)	38 (31.1)	44 (36.1)	49 (40.2)	0.339
Stroke, n (%)	16 (13.1)	19 (15.6)	11 (9.0)	0.296
COPD, n (%)	31 (25.4)	31 (25.4)	24 (19.7)	0.475
Smoking, n (%)	52 (42.6)	61 (50.0)	65 (53.3)	0.233
Chronic kidney disease, n (%)	73 (59.8)	77 (63.1)	67 (54.9)	0.423
Sleep disordered breathing, n (%)	103 (84.4)	96 (78.7)	88 (72.1)	0.065
Medication				
β blockers, n (%)	77 (63.1)	84 (68.9)	95 (77.9)	0.040
RAS inhibitors, n (%)	73 (59.8)	70 (57.4)	80 (65.6)	0.404
MRAs, n (%)	26 (21.3)	43 (35.2)	58 (47.5)	<0.001
Calcium channel blockers, n (%)	74 (60.7)	34 (27.9)	23 (18.9)	<0.001
Loop diuretics, n (%)	64 (52.5)	78 (63.9)	87 (71.3)	0.009
Evening dosing of any antihypertensive medications, n (%)	49 (40.2)	51 (41.8)	55 (45.1)	0.731
Bedtime dosing of any antihypertensive medications, n (%)	0 (0.0)	2 (1.6)	0 (0.0)	0.134
SGLT2 inhibitors, n (%)	6 (4.9)	15 (12.3)	15 (12.3)	0.082
Diabetic drugs, n (%)	26 (21.3)	30 (24.6)	33 (27.0)	0.577
Statins, n (%)	46 (37.7)	61 (50.0)	50 (41.0)	0.133
Laboratory and echocardiographic data				
B-type natriuretic peptide, pg/mL	198.6 (77.5–407.5)	220.7 (97.7–503.3)	366.6 (118.6–662.3)*	0.028
Estimated GFR, mL/min per 1.73 m^2	52.8 (38.9–65.3)	53.3 (40.9–65.2)	55.8 (43.2–69.9)	0.166
Hemoglobin, g/dL	12.6±2.14	12.9±2.19	13.0±1.97	0.321
Type of HF, n (%)				<0.001
HFrEF	21 (17.2)	32 (26.2)	57 (46.7)	
HFmrEF	15 (12.3)	25 (20.5)	12 (9.8)	
HFpEF	86 (70.5)	65 (53.3)	53 (43.3)	
Ischemic, n (%)	20 (16.4)	23 (18.9)	31 (25.4)	0.193

(*Continued*)

Table 1. Continued

	Highest-SBP tertile	Middle-SBP tertile	Lowest-SBP tertile	*P* value
Left ventricular ejection fraction, %	59.0 (45.3–65.0)	51.5 (37.0–64.0)	40.5 (29.0–60.0)*†	<0.001
Left ventricular end-diastolic volume, mL	110.0 (82.0–164.0)	111.0 (83.0–161.0)	121.5 (86.0–181.0)	0.404
Left ventricular end-systolic volume, mL	43.0 (30.0–80.0)	53.2 (31.0–100.5)	70.0 (36.6–124.7)*	0.006
Stroke volume, mL	59.0 (48.0–75.0)	55.0 (45.6–68.0)	48.5 (40.0–61.0)*	<0.001
Cardio-ankle vascular index	8.15 (6.90–9.50)	8.35 (7.20–9.55)	8.45 (6.90–9.40)	0.792

Continuous variables are expressed as mean±SD or as median (interquartile range). Categorical variables are expressed as frequency (percentage).

COPD indicates chronic obstructive pulmonary disease; DBP, diastolic blood pressure; GFR, glomerular filtration rate; HF, heart failure; HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; MBP, mean blood pressure; MRA, mineralocorticoid receptor antagonist; PTT, pulse transit time; RAS, renin–angiotensin system; SBP, systolic blood pressure; and SGLT2, sodium–glucose cotransporter 2.

*P<0.05 vs high-SBP group.

†P<0.05 vs middle-SBP group.

decrease in PTT-based SBP and an increased rate of cardiac events and HF hospitalization. Furthermore, the multivariate Cox proportional hazard analysis showed that the lowest-SBP tertile was associated with cardiac events and HF hospitalization compared with the highest-SBP tertile. In addition, as well as PTT-based nighttime SBP, 1-time daytime SBP was similarly associated with poor prognosis (a 10-mm Hg decrease in 1-time daytime SBP; hazard ratio 1.236 [95% CI, 1.073–1.413]; P=0.002).

Furthermore, Kaplan–Meier analysis and Cox proportional hazards analysis were performed by dividing patients into tertiles based on nighttime diastolic BP. While there was a tendency for poorer prognosis in the low–diastolic BP group, no significant difference was observed (Figure S1 and Table S1).

DISCUSSION

In the present study, we investigated, for the first time, the association between nighttime SBP and cardiac events in patients with HF using the novel PTT-driven approach. We found that a 10-mm Hg decrease in nighttime SBP was associated with an increased risk of both cardiac events and HF hospitalization in patients with HF. PTT-based BP measurements are superior to ambulatory BP monitoring because they do not require a cuff, thereby reducing patient discomfort and allowing for nighttime measurements without disturbing sleep. In addition, we demonstrated the associated factors of low BP, namely, low cardiac function (left ventricular end-systolic volume, stroke volume, and left ventricular ejection fraction determined by echocardiography) and similar arterial stiffness (determined by CAVI).

In patients with HF, the relationship between BP and prognosis has not been well understood.[11,12] Few studies have reported the association between SBP and prognosis, and most of the results were based on daytime BP rather than nighttime BP.[24] Seidu et al reported in a meta-analysis that a 10-mm Hg increase in daytime SBP or diastolic BP was associated with a reduced risk of several cardiovascular end points and all-cause death in patients with HF.[24] The findings from the

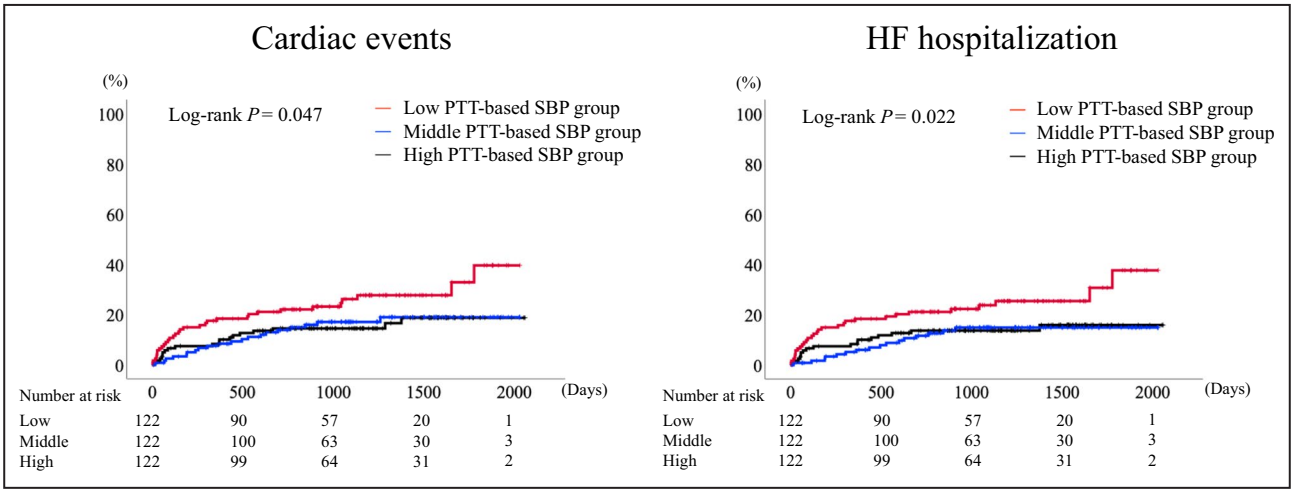

Figure. Kaplan–Meier curve for cardiac events and HF hospitalization rates stratified by nighttime PTT-based SBP tertiles.
HF indicates heart failure; PTT, pulse transit time; and SBP, systolic blood pressure.

Table 2. Association Between Nighttime PTT-Based SBP for Cardiac Events and HF Hospitalization: Cox Proportional Hazard Analyses

	Hazard ratio	95% CI	*P* value
Cardiac events (n=71/366)			
PTT-based SBP per 10-mm Hg decrease, unadjusted	1.187	1.041–1.356	0.013
PTT-based SBP per 10-mm Hg decrease, model 1	1.275	1.106–1.473	<0.001
PTT-based SBP per 10-mm Hg decrease, model 2	1.236	1.073–1.428	0.004
PTT-based SBP per 10-mm Hg decrease, model 3	1.224	1.051–1.428	0.008
Low-SBP tertiles (vs High SBP tertiles), unadjusted	1.814	1.028–3.200	0.040
Low-SBP tertiles (vs high-SBP tertiles), model 1	2.512	1.409–4.479	0.002
Low SBP tertiles (vs high-SBP tertiles), model 2	2.213	1.225–3.997	0.008
Low SBP tertiles (vs high-SBP tertiles), model 3	2.100	1.121–3.933	0.021
HF hospitalization (n=63/366)			
PTT-based SBP per 10-mm Hg decrease, unadjusted	1.211	1.051–1.413	0.007
PTT-based SBP per 10-mm Hg decrease, model 1	1.301	1.117–1.504	<0.001
PTT-based SBP per 10-mm Hg decrease, model 2	1.249	1.036–1.207	0.004
PTT-based SBP per 10-mm Hg decrease, model 3	1.262	1.062–1.489	0.007
Low-SBP tertiles (vs high-SBP tertiles), unadjusted	1.892	1.043–3.432	0.036
Low-SBP tertiles (vs high-SBP tertiles), model 1	2.566	1.402–4.698	0.002
Low-SBP tertiles (vs high-SBP tertiles), model 2	2.202	1.186–4.090	0.012
Low-SBP tertiles (vs high-SBP tertiles), model 3	2.084	1.073–4.047	0.030

Model 1: adjusted for age and sex. Model 2: adjusted for age, sex, B-type natriuretic peptide, estimated glomerular filtration rate, hemoglobin, and left ventricular ejection fraction. Model 3: adjusted for age, sex, B-type natriuretic peptide, estimated glomerular filtration rate, hemoglobin, left ventricular ejection fraction, β blockers, renin–angiotensin system inhibitors, mineralocorticoid receptor antagonists, loop diuretics, calcium channel blockers, and sodium–glucose cotransporter 2 inhibitors. HF indicates heart failure; PTT, pulse transit time; and SBP, systolic blood pressure.

KorAHF (Korean Acute Heart Failure) registry demonstrated a reverse J-curve relationship between daytime BP and outcomes in patients hospitalized for HF, showing an increased risk of death and HF hospitalization in those with both high and low BP levels.[30] In the present study, the lack of patients with HF with extremely low or high BP levels may have led to a linear relationship rather than a J-curve pattern.

The optimal BP target for patients with HF remains unclear due to the limited availability of high-quality evidence. While associations between BP and clinical outcomes have been demonstrated, the underlying mechanisms responsible for BP differences have not been fully investigated.[24] In the present study, we explored the mechanisms of BP changes that were assessed using echocardiographic data and CAVI. Since BP is determined by cardiac output and vascular resistance, a decrease in cardiac output can lead to a decrease in BP.[31] Hypertension increases collagen fiber production and accelerates elastin fiber degradation,[32] potentially causing arterial wall stiffness.[33] In addition, stiffened arteries are less responsive to BP changes, causing an increase in SBP and pulse pressure. While there are various indicators of arterial stiffness, CAVI is noninvasive and easy to measure.[23] In the present study, there was no difference in CAVI values between tertiles, suggesting that the low BP in the low-SBP group was not due to arterial stiffness, which might lead to vascular resistance. In patients with HF, cardiac output generally decreases as cardiac function declines.[31] Consequently, if the vascular resistance remains constant or does not increase sufficiently to compensate, BP drops as cardiac output diminishes. In patients with HF with low SBP, the probability of having advanced or end-stage HF is higher, often accompanied by reduced cardiac output and indicators of organ hypoperfusion.[14] As observed in this study, the low-SBP group had the lowest stroke volume and the smallest mean BP and pulse pressure, indicating the possibility of low perfusion. Low BP itself can lead to a poor prognosis due to circulatory insufficiency and inadequate organ perfusion. On the other hand, low BP may reflect the progression of HF, suggesting that low SBP could serve as a marker of impaired cardiac function.

Adverse events associated with nondipper and reverse dipper types have been reported in dipper pattern studies.[34–36] The present study found no significant differences in dipper patterns among the nighttime SBP tertiles, suggesting that low SBP does not necessarily reflect dipper patterns in patients with HF. In this study, low SBP based on 1-time daytime BP measurements was also associated with a poor prognosis. Nevertheless, we emphasize that the novelty of this study lies in its focus on nighttime BP, using noninvasive, continuous BP monitoring and investigating the

associations between nighttime BP and prognosis in patients with HF.

BP-lowering treatments must balance their associated adverse effects with the potential benefits. Medications with survival benefits remain effective even at lower BP thresholds.[37] There has been considerable debate about the optimal target BP,[38,39] making it difficult to establish a universal standard. Although US and European hypertension guidelines explicitly recommend a target daytime BP <130/80 mm Hg for patients with HF,[40,41] SBP <110 mm Hg may be associated with a poor prognosis,[42] and managing daytime SBP between 110 and 130 mm Hg is supported in the United States.[11] This recommendation is reinforced by observations from successful HF treatment trials, where SBP was typically reduced to this normal range using HF medications.[43] Evidence regarding the optimal nighttime BP in patients with HF is even more limited. The findings of the present study may help establish the target nighttime BP for future HF management.

As a preliminary investigation of patients with HF, this study has several limitations, including a small sample size at a single center. Furthermore, the study had a limited number of participants with either excessively high or low BP. Nighttime BP fluctuates under the influence of the autonomic nervous system, and disturbances in sleep quantity or quality are particularly suggested to contribute to the development and progression of nighttime hypertension.[44] The inpatient setting and sleep quality may have influenced nighttime SBP. The reproducibility of PTT-derived BP was not validated in this study. Unlike conventional nighttime BP assessed by ambulatory BP monitoring, PTT-derived BP is less widely used. Furthermore, the accuracy and reproducibility of PTT-based BP measurements should be investigated. The dipping pattern is ideally analyzed by comparing mean daytime and nighttime BP. However, in this study, cuff BP used for PTT calibration was provisionally used as daytime BP. Given the nature of the study, selection bias and potential unmeasured confounding factors should be acknowledged, particularly since the study enrolled patients who had undergone PTT monitoring in conjunction with an overnight sleep study. BP measurements were limited to overnight stays, with no continuous daytime monitoring. Changes in nighttime SBP during the follow-up period were not collected in this study, and follow-up BP data were missing, and this point could lead to potential bias. Additionally, we did not track changes in medications.

CONCLUSIONS

The present study demonstrated the relationship between low nighttime PTT-based SBP and cardiac events in patients with HF. A PTT-based BP measurement may be useful in assessing nighttime BP in patients with HF and could contribute to identifying those at increased risk of cardiac events.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Material

Table S1
Figure S1

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