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Prognostic impact of systolic blood pressure trajectory among patients hospitalised in an acute heart failure setting: insights from a real-world multinational cohort

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Summary

BACKGROUND: Systolic blood pressure is a prognostic marker in acute heart failure, but the prognostic implications of in-hospital changes in systolic blood pressure are unclear. We assessed the association between in-hospital systolic blood pressure changes and outcomes in a real-world, multinational cohort of acute heart failure patients.

METHODS: We analysed consecutive patients hospitalised for acute heart failure between 2005 and 2020 at two tertiary-care centres (CHUV, Switzerland; NCCIM, Kyrgyzstan) with available systolic blood pressure measurements at admission and discharge. Patients were classified into four systolic blood pressure trajectory categories: stable normal/low (systolic blood pressure consistently <140 mm Hg or minor increase, Δ<10), increasing (systolic blood pressure rose ≥10 mm Hg from <140 to ≥140 mm Hg), decreasing (systolic blood pressure dropped ≥10 mm Hg from ≥140 to <140 mm Hg), stable elevated (systolic blood pressure consistently ≥140 mm Hg or minor decrease, Δ<10). The primary outcome of the study was a composite of first heart failure hospitalisation or all-cause mortality, assessed over a 1-year follow-up period. The association between categories and the primary outcome was assessed with Cox models, adjusted for relevant covariates.

RESULTS: Among 1490 patients (80% Swiss, 56% male, age 75 ± 13 years), 621 experienced the primary outcome at 1 year. Compared to those with stable normal/low systolic blood pressure, patients with decreasing systolic blood pressure had a significantly lower risk of the primary outcome (adjusted HR: 0.81; 95% CI: 0.66–0.99; p = 0.040), with no significant differences for the other systolic blood pressure trajectories. Results remained consistent regardless of sex, age and left ventricular ejection fraction ($P_{interaction}$ for all >0.05).

CONCLUSION: In this real-world, multinational cohort of 1490 acute heart failure patients, in-hospital decline in systolic blood pressure was independently associated with improved outcomes in those with an elevated systolic blood pressure at admission.

Introduction

Despite recent significant advancements in the management of heart failure [1], hospitalisations for acute heart failure remain frequent and impose a considerable burden on healthcare systems. Acute heart failure represents the leading cause of unplanned hospital admissions among individuals aged 65 years and older, contributing substantially to high healthcare costs associated with these admissions [2–4].

Acute heart failure remains strongly associated with poor outcomes, including very high short-term readmission rates and high mortality. Every year, over one million patients are hospitalised with a primary diagnosis of acute heart failure in North America and Western Europe [4, 5].

High systolic blood pressure is a key risk factor of heart failure, both with reduced and preserved ejection fraction [6], and has emerged as an important prognostic marker in the context of acute heart failure [7]. Previous studies have suggested that, among patients hospitalised for acute heart failure, higher systolic blood pressure at the time of hospital admission may be linked to better outcomes compared to lower systolic blood pressure values [7, 8]. This contrasts with the ambulatory and chronic heart failure setting, where elevated systolic blood pressure has traditionally been associated with a poorer prognosis [1, 9, 10]. However, the prognostic significance of systolic blood pressure trajectory during hospitalisation remains less understood. Gaining a clearer understanding of how systolic blood pressure changes between hospital admission and discharge could help identify patients at higher risk of adverse clinical outcomes, who may benefit from closer monitoring.

Using data from a prospective, large, real-world, multiethnic cohort including close to 1500 patients in Switzerland and Kyrgyzstan, we aimed to examine the association between in-hospital systolic blood pressure changes and subsequent clinical outcomes.

Methods

Study design and population

Consecutive adult patients hospitalised for acute heart failure in the Cardiology and Internal Medicine Divisions of Lausanne University Hospital (CHUV) in Lausanne, Switzerland, and the National Center of Cardiology and Internal Medicine (NCCIM) in Bishkek, Kyrgyzstan, between 2005 and 2020, were included in the cohort. Both the CHUV and NCCIM are tertiary teaching hospitals offering comprehensive heart failure care, including ambulatory services, acute heart failure hospitalisation units, interventional procedures and device implantation.

Patients had a clinical diagnosis of acute heart failure according to heart failure guidelines in effect at the time of the study [11]. Only those with systolic blood pressure (SBP) measurements available at both admission (SBPa) and discharge (SPBd) were included in the present study. Exclusion criteria included patients who died during hospitalisation and those presenting with acute pulmonary embolism, acute myocardial ischaemia, severe valvular regurgitation or stenosis requiring percutaneous or surgical intervention, stress-induced cardiomyopathy, exacerbation of chronic obstructive pulmonary disease (COPD), complex congenital heart disease or recent cardiac surgery. Additional exclusions applied to patients with acute infectious, toxic or metabolic conditions, defined as acute non-cardiac conditions (such as acute intoxications or severe metabolic derangements, including diabetic ketoacidosis) that were considered the primary cause of haemodynamic instability or heart failure-like presentation, those undergoing dialysis, pregnant women or individuals with an estimated survival of less than one year due to comorbidities.

ABBREVIATIONS
ACE angiotensin-converting enzyme
AFF atrial fibrillation or flutter
ARB angiotensin receptor blocker
ARNI angiotensin receptor/neprilysin inhibitor
BMI body mass index
COPD chronic obstructive pulmonary disease
DBP diastolic blood pressure
EGFR estimated glomerular filtration rate
HBA1C haemoglobin A1C
HFH heart failure hospitalisation
ICD implantable cardioverter-defibrillator
LVEDD left ventricular end-diastolic diameter
LVEF left ventricular ejection fraction
LVMI left ventricular mass index
MRA mineralocorticoid receptor antagonist
NT-PROBNP N-terminal pro-B-type natriuretic peptide
SBPA systolic blood pressure at admission
SBPD systolic blood pressure at discharge

Echocardiography was performed or reviewed by board-certified cardiologists following applicable international guidelines [12]. Clinical variables, comorbidities, laboratory values, vital signs at admission and discharge, treatments and echocardiographic parameters were extracted from the electronic medical records of each centre using standardised case report forms. Mortality data were obtained from national death registries, and heart failure rehospitalisations were retrieved from each hospital's electronic health system. All data were then transferred into a centralised, deidentified research database. Quality control included double-checking of 20% of entries, yielding a 99.7% concordance rate [13]. Sodium-glucose cotransporter-2 inhibition was not yet recommended for heart failure management during the study period, and was therefore categorised as oral antidiabetic treatment. Information on 1-year all-cause mortality was extracted from national registries in Kyrgyzstan and Switzerland (accessed in December 2021). Data on hospitalisation for heart failure were obtained from the electronic medical records of each hospital.

This study was conducted in accordance with the Declaration of Helsinki and received approval from the corresponding local ethics committees (Bishkek: Local Ethics Committee 2019-7; Lausanne: CER-VD 2019-1158).

Systolic blood pressure measurement and categorisation

Blood pressure was measured using an automated device or a standard sphygmomanometer with an appropriately sized cuff, in both centres, at both admission and discharge. SBPa was defined as the first systolic blood pressure measurement available in the medical record after the start of hospitalisation, SBPd as the last systolic blood pressure measurement available before the end of hospitalisation.

Patients were categorised into four systolic blood pressure trajectory groups based on SBPa and SBPd values:

- Stable normal/low: both SBPa and SBPd <140 mm Hg, or SBPa <140 mm Hg and SBPd ≥140 mm Hg, with a difference (Δ) <10 mm Hg.
- Increasing: SBPa <140 mm Hg and SBPd ≥140 mm Hg, with a Δ ≥10 mm Hg.
- Decreasing: SBPa ≥140 mm Hg and SBPd <140 mm Hg, with a Δ ≥10 mm Hg.
- Stable elevated: both SBPa and SBPd ≥140 mm Hg, or SBPa ≥140 mm Hg and SBPd <140 mm Hg, with a Δ <10 mm Hg.

The threshold of 140 mm Hg for defining "high" and "normal/low" systolic blood pressure was chosen based on the standard definition of hypertension, applicable in both chronic and acute settings [14, 15]. The selection of a Δ of 10 mm Hg reflects the rationale that this level of change is clinically significant and meaningful in an acute setting.

Study outcomes

The primary outcome was a composite of first hospitalisation for heart failure or all-cause death occurring within one year after the index hospitalisation for heart failure. The secondary outcomes were the individual components of the composite, namely first hospitalisation for heart failure alone and all-cause death alone, assessed over the same follow-up period.

Statistical analyses

Data were reported as count (percentage) for categorical variables, mean (standard deviation) when distributed normally and median (interquartile range) for non-normally distributed variables. To compare global differences across the four groups, we used the chi-squared test for binary variables, analysis of variance for normally distributed continuous variables and the Kruskal–Wallis test for non-normally distributed continuous variables.

The association between systolic blood pressure trajectory categories and outcomes (all analysed as time to first events) was assessed using Cox proportional hazards models, with the stable normal/low category (SBPa and SBPd <140 mm Hg, Δ <10 mm Hg) serving as the reference. The follow-up period began at the date of discharge. Analyses were stratified by country (Kyrgyzstan or Switzerland), and were both unadjusted and adjusted for age, sex, body mass index (BMI), prior myocardial infarction, atrial fibrillation or flutter, prior hospitalisation for heart failure, hypertension, diabetes, left ventricular ejection fraction (LVEF), estimated glomerular filtration rate, length of hospital stay, use of beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB) and mineralocorticoid receptor antagonists (MRA).The selection of these covariates was guided by baseline differences between categories and clinical judgement. Interaction testing was conducted to evaluate the interaction between systolic blood pressure trajectory categories and sex, age groups (<78 years, ≥78 years, based on the median age in

the whole cohort) and LVEF (<40%, ≥40%) in relation to the primary outcome. We also assessed the relative change in the use of heart failure prognostic medication classes (beta-blockers, ACE inhibitors, angiotensin receptor blockers, mineralocorticoid receptor antagonists, angiotensin receptor/neprilysin inhibitor), as well as loop diuretics, between admission and discharge according to systolic blood pressure trajectory categories. The relative change for each medication class was calculated as: (proportion at discharge – proportion at admission) / proportion at admission. In sensitivity analyses, we further explored the prognostic impact of SBPa and SBPd individually, categorised as follows: <120 mm Hg, 120–139 mm Hg, 140–159 mm Hg and ≥160 mm Hg. Crude incidence rates of events were reported using the Kaplan–Meier method. We tested for violation of the proportional hazards assumption for all hazard ratios that were reported, via Schoenfeld residuals.

Additionally, we analysed the relationship between the primary outcome and continuous SBP change (defined as SBPd – SBPa), using Poisson regression to estimate incidence rates, and applying multivariable restricted cubic splines, with the numbers of knots selected to minimise the resulting Akaike information criteria (3–6 knots tested).

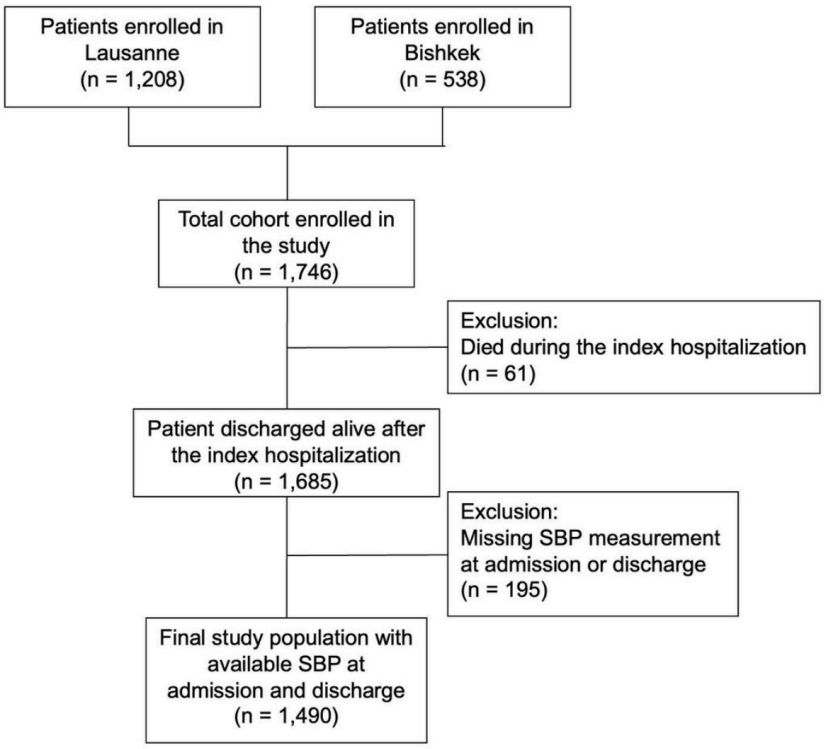
Finally, a multivariable logistic regression model with a forward stepwise approach was used to identify clinical, biological and echocardiographic parameters associated with the SBP trajectory category with the most favourable prognosis, including all relevant parameters at baseline in the model. To ensure comparability, continuous variables were standardised. Statistical analyses were performed using STATA, version 18 (StataCorp, College Station, TX, USA). A two-sided p-value <0.05 was considered statistically significant.

Results

Demographics and baseline characteristics

A total of 1746 patients were recruited, including 1208 (69.2%) in Switzerland. Among these, 61 patients (3.5%) died during the index hospitalisation and were excluded from the analyses. Among the remaining 1685 patients, systolic blood pressure measurements were available at both admission and discharge in 1490 patients (80% from Switzerland, 56% male, mean age: 75 ± 13 years). Of these, 757 (50.8%) had stable/low systolic blood pressure, 77 (5.2%) had increasing systolic blood pressure, 412 (27.7%) had decreasing systolic blood pressure and 244 (16.3%) had stable elevated systolic blood pressure (figure 1).

Figure 1: Patient selection flowchart. The flowchart illustrates the derivation of the final study population from patients hospitalised for acute heart failure at the two participating centres. "Patients enrolled" refers to patients meeting the clinical diagnosis of acute heart failure and included in the study databases at each centre. The flowchart details exclusions due to in-hospital death and missing systolic blood pressure measurements at admission and/or discharge, leading to the final analysis cohort.



The increasing systolic blood pressure category included more women (57.1%) and older patients (82.5 years) compared to other groups (p <0.001, table 1). Patients in this category also had a significantly higher prevalence of atrial fibrillation or flutter, lower glomerular filtration rates and lower haemoglobin levels. Patients with stable elevated systolic blood pressure were less likely to have a previous episode of hospitalisation for heart failure, but had a higher prevalence of hypertension and diabetes. N-terminal pro–B-type natriuretic peptide (NT-proBNP, only available in 55.8% of patients) was significantly higher in the stable normal/low systolic blood pressure group. No significant differences were observed across systolic blood pressure trajectory groups regarding BMI, prior myocardial infarction, COPD or tobacco use. In terms of echocardiographic parameters, patients with stable normal/low systolic blood pressure had significantly lower LVEF and larger LV end-diastolic diameters. Regarding treatments at admission, patients in the stable elevated systolic blood pressure group were more frequently prescribed beta-blockers, angiotensin receptor blockers, and insulin, while those in the stable normal/low systolic blood pressure group more often received mineralocorticoid receptor antagonists and loop diuretics.

Table 1: Baseline patient characteristics by systolic blood pressure (SBP) category. Results are expressed as n (%), mean ± standard deviation or median [interquartile range]. Groups were compared using the chi-squared test for categorical variables, analysis of variance (ANOVA) for normally distributed continuous variables and the Kruskal–Wallis test for non-normally distributed continuous variables.

Characteristics		Stable normal/low SBP (n = 757)	Increasing SBP (n = 77)	Decreasing SBP (n = 412)	Stable elevated SBP (n = 244)	p-value
Demographics and vitals	Age, years	73.1 ± 14.3	82.5 ± 8.7	76.3 ± 12.4	78.5 ± 10.1	<0.001
	Female sex	301 (39.8%)	44 (57.1%)	183 (44.4%)	128 (52.5%)	<0.001
	BMI, kg/m^2	27.4 ± 6.6	27.1 ± 6.5	28.1 ± 6.2	27.1 ± 5.7	0.23
	Bishkek cohort	188 (24.8%)	1 (1.3%)	92 (22.3%)	18 (7.4%)	<0.001
	SBPa, mm Hg	117.0 ± 14.2	120.6 ± 11.6	159.8 ± 17.3	162.4 ± 21.5	By design
	DBPa, mm Hg	71.8 ± 13.0	70.6 ± 15.0	90.7 ± 17.4	85.3 ± 16.9	<0.001
	Heart rate, bpm	93.4 ± 25.6	93.8 ± 26.3	95.1 ± 28.0	86.8 ± 23.7	<0.001
Comorbidities	Previous myocardial infarction	331 (43.7%)	32 (41.6%)	181 (43.9%)	112 (45.9%)	0.90
	AFF	444 (58.7%)	47 (61.0%)	224 (54.4%)	115 (47.1%)	0.011
	Prior heart failure hospitalisation	583 (77.0%)	52 (67.5%)	290 (70.4%)	155 (63.5%)	<0.001
	Hypertension	541 (71.5%)	70 (90.9%)	352 (85.4%)	230 (94.3%)	<0.001
	Diabetes	226 (29.9%)	29 (37.7%)	146 (35.4%)	106 (43.4%)	<0.001
	COPD	161 (21.3%)	14 (18.2%)	97 (23.5%)	49 (20.1%)	0.61
	Smoking	334 (44.1%)	33 (42.9%)	174 (42.2%)	112 (46.1%)	0.80
	Dyslipidaemia	386 (51.0%)	37 (48.1%)	234 (56.8%)	145 (59.7%)	0.040
	Haemoglobin, g/L	128.4 ± 22.9	117.0 ± 21.9	129.9 ± 22.7	121.6 ± 21.7	<0.001
	HbA1C, %	7.0 ± 1.5	7.1 ± 0.7	6.8 ± 1.4	6.9 ± 1.4	0.94
	NT-proBNP, pg/ml*	5543 [2361 – 11,060]	3586 [2005 – 9534]	4402 [2377 – 8886]	3530 [1785 – 9910]	0.048
	Creatinine, µmol/l	124.5 ± 63.4	130.2 ± 61.6	123.5 ± 88.1	143.2 ± 109.8	0.009
	eGFR, ml/min/1.73m^2	56.9 ± 22.7	48.2 ± 20.4	57.0 ± 21.6	52.1 ± 25.1	<0.001
Echocardiographic parameters	LVEF, %	41.0 ± 16.3	51.2 ± 13.6	45.2 ± 15.3	51.5 ± 14.0	<0.001
	LVEDD, mm	56.2 ± 11.1	49.8 ± 9.6	54.0 ± 10.0	50.7 ± 7.4	<0.001
	LVMi, g/m^2	119.1 ± 43.9	111.2 ± 33.7	123.2 ± 42.0	113.5 ± 32.0	0.031
Treatment	Statin	264 (34.9%)	30 (39.0%)	135 (32.8%)	98 (40.2%)	0.24
	Beta-blocker	351 (46.4%)	42 (54.5%)	187 (45.4%)	139 (57.0%)	0.011
	ACE inhibitor	224 (29.6%)	25 (32.5%)	129 (31.3%)	86 (35.2%)	0.42
	ARB	135 (17.8%)	23 (29.9%)	105 (25.5%)	67 (27.5%)	<0.001
	ARNi	3 (0.4%)	0 (0.0%)	3 (0.7%)	0 (0.0%)	0.50
	MRA	169 (22.3%)	5 (6.5%)	53 (12.9%)	24 (9.8%)	<0.001
	Loop diuretic	433 (57.2%)	43 (55.8%)	200 (48.5%)	124 (50.8%)	0.028
	Oral antidiabetic	125 (16.5%)	16 (20.8%)	56 (13.6%)	48 (19.7%)	0.15
	Insulin therapy	76 (10.0%)	13 (16.9%)	64 (15.5%)	51 (20.9%)	<0.001
	ICD	38 (5.0%)	1 (1.3%)	7 (1.7%)	4 (1.6%)	0.004
	Pacemaker	81 (10.7%)	8 (10.4%)	32 (7.8%)	21 (8.6%)	0.39

SBPa (systolic blood pressure at admission): first SBP measurement recorded after the start of the hospitalisation.
SBPd (systolic blood pressure at discharge): last SBP measurement documented before hospital discharge.
SBP trajectory categories: defined based on SBPa, SBPd and ΔSBP (stable normal/low, increasing, decreasing, stable elevated).
* Missing in 658 (44.2%) patients.

Association between systolic blood pressure trajectory categories and outcomes

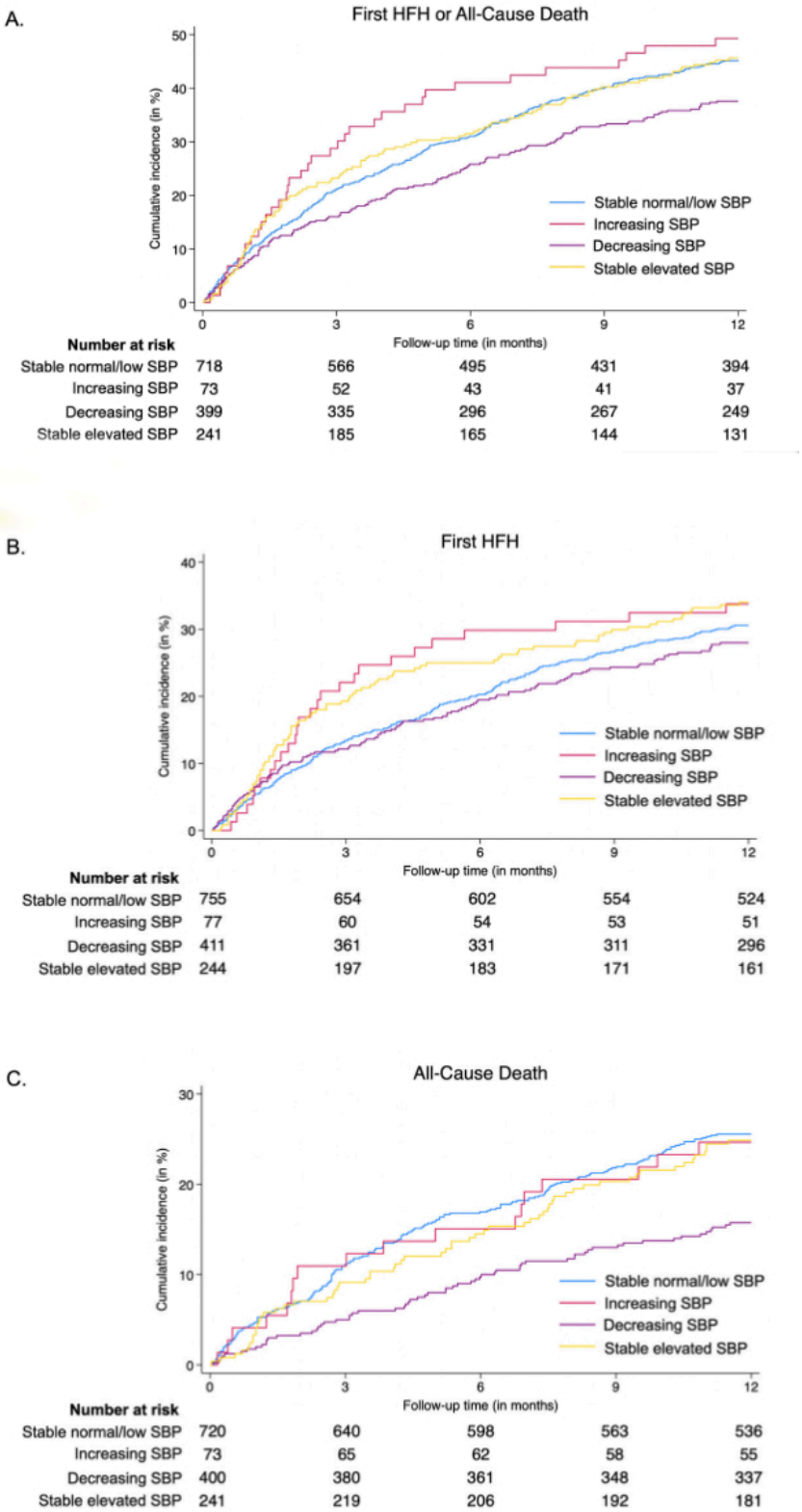
Over a 1-year follow-up period, 621 patients experienced a primary outcome event (table 2). Using the stable normal/low systolic blood pressure category as the reference (64.0 events per 100 patient-years [py]), the decreasing systolic blood pressure category was associated with a significantly lower risk for the primary outcome, both in non-adjusted (49.4 events per 100 py, HR: 0.78, 95% CI: 0.64–0.94; figure 2A) and adjusted (adjusted HR: 0.81, 95% CI: 0.66–0.99) analyses. No significant differences were observed for the other systolic blood pressure trajectory categories. These findings were consistent regardless of sex ($P_{interaction}$ = 0.91), age category (<78 and ≥78 years, $P_{interaction}$ = 0.09) and LVEF (<40% and ≥40%, $P_{interaction}$ = 0.22).

Table 2: Event counts, event rates and hazard ratios associated with categories of systolic blood pressure trajectory. All analyses are stratified by country (Switzerland or Kyrgyzstan). Comparisons across systolic blood pressure trajectory groups were performed using Cox proportional hazards regression.

Events		n (%)	Event rate (95% CI), per 100 patient-years	Unadjusted HR (95% CI)	Adjusted HR (95% CI), Model*
First heart failure hospitalisation or all-cause death	Stable normal/low SBP (n = 757)	325 (42.9%)	64.0 (57.5–71.4)	1 (reference)	1 (reference)
	Increasing SBP (n = 77)	36 (46.8%)	76.5 (55.2–106.0)	1.09 (0.77–1.54)	1.00 (0.70–1.43)
	Decreasing SBP (n = 412)	150 (36.4%)	49.4 (42.1–58.0)	0.78 (0.64–0.94)	0.81 (0.66–0.99)
	Stable elevated SBP (n = 244)	110 (45.1%)	65.5 (54.3–79.0)	0.97 (0.78–1.20)	0.96 (0.76–1.21)
First heart failure hospitalisation	Stable normal/low SBP (n = 757)	232 (30.6%)	38.1 (33.5–43.3)	1 (reference)	1 (reference)
	Increasing SBP (n = 77)	26 (33.8%)	45.1 (30.7–66.2)	1.11 (0.73–1.66)	1.03 (0.68–1.56)
	Decreasing SBP (n = 412)	115 (27.9%)	34.1 (28.4–41.0)	0.90 (0.72–1.13)	0.92 (0.73–1.16)
	Stable elevated SBP (n = 244)	83 (34.0%)	44.5 (35.8–59.1)	1.12 (0.87–1.44)	1.07 (0.82–1.40)
All-cause death	Stable normal/low SBP (n = 757)	184 (24.3%)	30.4 (26.3–35.1)	1 (reference)	1 (reference)
	Increasing SBP (n = 77)	18 (23.4%)	29.2 (18.4–46.3)	0.84 (0.52–1.37)	0.78 (0.47–1.28)
	Decreasing SBP (n = 412)	63 (15.3%)	17.3 (13.5–22.1)	0.56 (0.42–0.75)	0.58 (0.43–0.78)
	Stable elevated SBP (n = 244)	60 (24.6%)	29.0 (22.5–37.4)	0.86 (0.64–1.15)	0.94 (0.69–1.28)

*Adjusted for age, sex, BMI, prior myocardial infarction, atrial fibrillation or flutter, prior heart failure hospitalisation, hypertension, diabetes, left ventricular ejection fraction, estimated glomerular filtration rate, length of hospital stay, beta-blockers, ACE inhibitors, angiotensin receptor blockers, mineralocorticoid receptor antagonists.

Figure 2: Cumulative incidence of (A) the composite outcome of first heart failure hospitalisation or all-cause death, (B) first heart failure hospitalisation, (C) all-cause death according to categories of systolic blood pressure trajectory. Cumulative incidence curves were generated using the Kaplan–Meier method. Abbreviations: HFH: heart failure hospitalisation; SBP: systolic blood pressure.

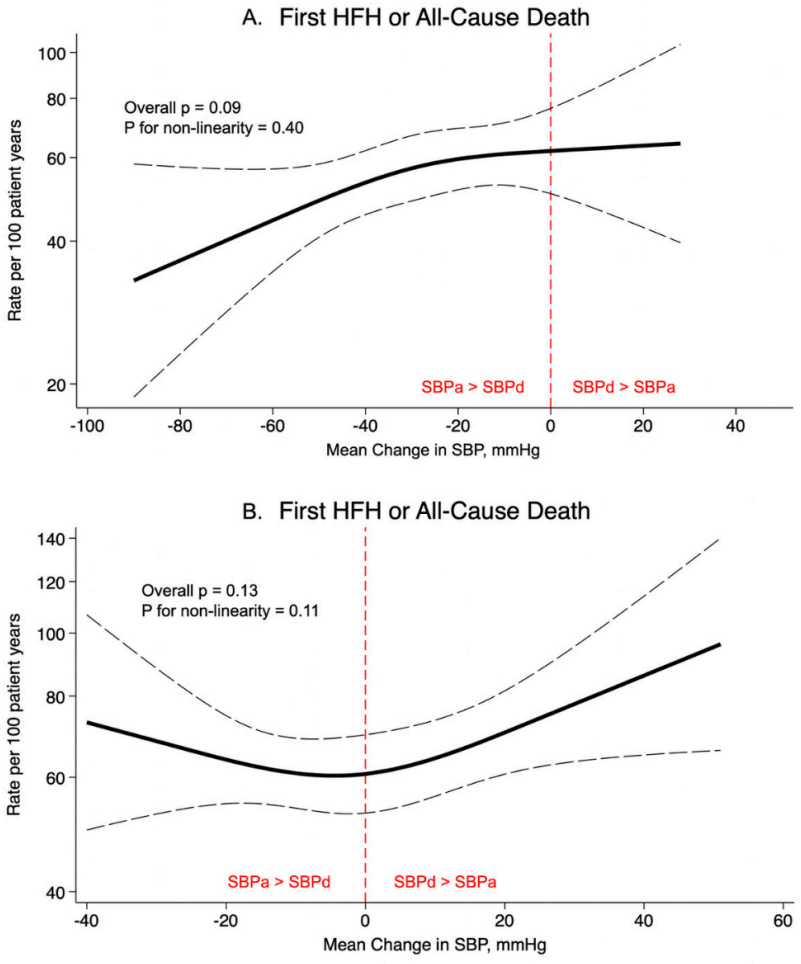


When analysing all-cause mortality and first hospitalisation for heart failure as separate outcomes, the decreasing systolic blood pressure category was associated with a significant reduction in the risk of all-cause mortality (adjusted HR: 0.58, 95% CI: 0.43–0.78; figure 2B), but there was no significant effect on the risk of first hospitalisation for heart failure (adjusted HR: 0.92, 95% CI: 0.73–1.16; figure 2C). No violations of the proportional hazards assumption for systolic blood pressure categories were identified for either the primary or secondary outcomes.

No significant association was observed between SBPa categories and the risk of the primary outcome (table S1 in the appendix). In contrast, patients in the lower SBPd category (SBPd <120 mm Hg) had a significantly higher risk of the primary outcome (adjusted HR: 1.27, 95% CI: 1.05–1.53) compared to those with an SBPd of 120–139 mm Hg (table S2 in the appendix).

When analysing systolic blood pressure changes between admission and discharge as a continuous variable, a decline in systolic blood pressure was associated with a lower rate of the primary outcome among patients with an SBPa ≥140 mm Hg (figure 3A). However, no significant association was observed between systolic blood pressure changes and event rates in patients with an SBPa <140 mm Hg (figure 3B).

Figure 3: Rate of first heart failure hospitalisation or all-cause death according to mean change in systolic blood pressure (SBP at discharge – SBP at admission) (A) among patients with a systolic blood pressure ≥140 mm Hg at admission, (B) among patients with a systolic blood pressure <140 mm Hg at admission. Event rates were estimated using Poisson regression models. The association between continuous systolic blood pressure change and the primary outcome was assessed using multivariable restricted cubic splines. Abbreviations: HFH: heart failure hospitalisation; SBP: systolic blood pressure; SBPa: systolic blood pressure at admission; SBPd: systolic blood pressure at discharge.



Relative changes in the use of prognostic heart failure medication classes and diuretics

Relative changes in the use of beta-blockers (+58.8%), ACE inhibitors (+52.7%), angiotensin receptor blockers (+4.7%), mineralocorticoid receptor antagonists (+112.5%) and loop diuretics (+76.1%) were greatest in the decreasing systolic blood pressure category compared to other systolic blood pressure trajectory categories (table 3). At discharge, the proportion of patients on these medications in the decreasing systolic blood pressure category was the highest or second highest among the four categories.

Table 3: Number, proportion and relative changes in heart failure prognostic medications by systolic blood pressure trajectory categories. Results are expressed as n (%) unless otherwise specified. The relative change for each medication class was calculated as: (proportion at discharge – proportion at admission) / proportion at admission.

Medication		Stable normal/low SBP (n = 757)	Increasing SBP (n = 77)	Decreasing SBP (n = 412)	Stable elevated SBP (n = 244)	p-value
Beta-blocker	At admission	351 (46.4%)	42 (54.5%)	187 (45.4%)	139 (57.0%)	0.011
	At discharge	510 (67.4%)	42 (54.5%)	297 (72.1%)	163 (66.8%)	0.020
	Relative change (in %)	+45.3%	0.0%	+58.8%	+17.2%	<0.001
ACE inhibitor	At admission	224 (29.6%)	25 (32.5%)	129 (31.3%)	86 (35.2%)	0.42
	At discharge	308 (40.7%)	32 (41.6%)	197 (47.8%)	121 (49.6%)	0.028
	Relative change (in %)	+37.5%	+28.0%	+52.7%	+40.9%	<0.001
Angiotensin receptor blocker	At admission	135 (17.8%)	23 (29.9%)	105 (25.5%)	67 (27.5%)	<0.001
	At discharge	107 (14.1%)	16 (20.8%)	110 (26.7%)	66 (27.0%)	<0.001
	Relative change (in %)	-20.8%	-30.4%	+4.7%	-1.8%	<0.001
Angiotensin receptor/neprilysin inhibitor	At admission	3 (0.4%)	0 (0.0%)	3 (0.7%)	0 (0.0%)	0.50
	At discharge	5 (0.7%)	0 (0.0%)	2 (0.5%)	0 (0.0%)	0.55
	Relative change (in %)	+75.0%	0.0%	-28.6%	0.0%	<0.001
Mineralocorticoid receptor antagonist	At admission	169 (22.3%)	5 (6.5%)	53 (12.9%)	24 (9.8%)	<0.001
	At discharge	334 (44.1%)	7 (9.1%)	113 (27.4%)	37 (15.2%)	<0.001
	Relative change (in %)	+97.8%	+40.0%	+112.5%	+55.1%	<0.001
Loop diuretic	At admission	433 (57.2%)	43 (55.8%)	200 (48.5%)	124 (50.8%)	0.028
	At discharge	664 (88.2%)	60 (78.9%)	352 (85.4%)	203 (83.2%)	0.049
	Relative change (in %)	+54.2%	+41.4%	+76.1%	+63.8%	<0.001

Factors associated with high systolic blood pressure at admission and subsequent decline during hospitalisation

Factors at baseline associated with the decreasing systolic blood pressure category are presented in table 4. They include a known comorbidity of hypertension at admission (OR: 1.61, 95% CI: 1.64–2.24), higher BMI (OR: 1.15 per 6.4 kg/m^2 change, 95% CI: 1.02–1.30), higher heart rate at admission (OR: 1.19 per 26.1 bpm, 95% CI: 1.06–1.34) and higher eGFR at admission (1.15 per 22.8 ml/min/1.73 m^2, 95% CI: 1.01–1.30).

Table 4: Factors associated with high systolic blood pressure at admission and subsequent decline during hospitalisation. Sample size: n = 1490. There were no missing data in any category. Odds ratios and 95% confidence intervals were obtained using a multivariable logistic regression model with forward stepwise selection.

Factor	Odds ratio	95% CI	p-value
Known hypertension	1.61	1.16–2.24	0.005
BMI, per 6.4 kg/m^2 (1 SD)	1.15	1.02–1.30	0.019
Heart rate, per 26.1 bpm (1 SD)	1.19	1.06–1.34	0.003
eGFR, per 22.8 ml/min/m^2 (1 SD)	1.15	1.01–1.30	0.034

BMI = body mass index; CI = confidence interval; eGFR = estimated glomerular filtration rate; SD = standard deviation.

Discussion

In this analysis of nearly 1500 patients hospitalised for acute heart failure, we found that: (1) In-hospital decline in systolic blood pressure was associated with improved outcomes in those with elevated SBPa. This result was primarily driven by a lower risk of all-cause mortality and was consistent irrespective of sex, LVEF and age. (2) No significant association was found between SBPa and the risk of the primary outcome, while lower SBPd was associated with a higher risk. (3) Relative changes in the use of heart failure prognostic medication classes and loop diuretics were greatest in the decreasing systolic blood pressure category compared to other systolic blood pressure trajectory categories. (4) Factors associated with the decreasing systolic blood pressure category included known hypertension, higher BMI, higher heart rate and higher eGFR at admission.

To the best of our knowledge, while many previous studies have examined the prognostic impact of systolic blood pressure in an acute heart failure setting [7, 8, 16], our study is the first to comprehensively assess the prognostic significance of the systolic blood pressure trajectory during hospitalisation. Despite being an observational analysis, the study is strengthened by the inclusion of a large, real-world, well-characterised, multinational cohort of acute heart failure patients and the detailed reporting of patient vital signs at both admission and discharge.

High systolic blood pressure in heart failure serves both as a marker and a mediator of cardiovascular risk. It is associated with increased afterload, arterial stiffness and impaired ventricular-vascular coupling, which exacerbate myocardial stress and adverse remodelling [6, 17, 18]. Among chronic heart failure patients with hypertension, lowering systolic blood pressure to a target range of 120–130 mm Hg may provide long-term benefits [1, 9]. In the acute setting, however, systolic blood pressure may have a different prognostic significance. Higher systolic blood pressure in acute heart failure has previously been shown to be associated with more favourable cardiovascular outcomes, possibly reflecting preserved cardiac output, a less advanced heart failure state and more stable haemodynamics [6]. These patients may be less prone to end-organ hypoperfusion or cardiogenic shock, and more likely to tolerate therapies like vasodilators or diuretics [2]. Furthermore, hospitalisations in this group may be more often due to hypertensive pulmonary oedema rather than chronic heart failure with systemic and neurohormonal activation [1]. High systolic blood pressure in acute heart failure may also facilitate the in-hospital initiation or up-titration of prognostic heart failure treatments, which has been shown to be associated with a better prognosis [19, 20].

Interestingly, systolic blood pressure at admission did not demonstrate any predictive value in our analyses. Patients with high systolic blood pressure at admission who achieved lower systolic blood pressure values during hospitalisation experienced better outcomes compared to those who remained hypertensive at discharge. This may be partly driven by the fact that a higher proportion of patients in the decreasing systolic blood pressure trajectory received heart failure prognostic medications and loop diuretics during hospitalisation, compared to other systolic blood pressure trajectory categories. It is well established that patients who respond to initiation or up-titration of heart failure therapy and achieve effective decongestion have improved prognoses in an acute heart failure setting [21]. Moreover, our results suggest that the trajectory of systolic blood pressure during hospitalisation appears to be a more reliable prognostic indicator than a single systolic blood pressure measurement at admission. The reason why the improved outcomes associated with decreasing systolic blood pressure were primarily driven by all-cause death (and not hospitalisation for heart failure) in our cohort remains uncertain and merits further investigation.

In contrast, low systolic blood pressure may signal more severe heart failure or a decompensated state, characterised by reduced stroke volume, lower cardiac output and peripheral organ dysfunction, both in a chronic and acute heart failure setting [22]. Consistent with this hypothesis, our findings show that better kidney function at admission (higher eGFR) was associated with a higher probability of being in the decreasing systolic blood pressure category. Additionally, higher BMI was also associated with this group, aligning with the “obesity paradox”, where obesity is postulated to confer a better prognosis in heart failure patients [23], although this concept is debated [24]. Notably, the better prognosis associated with the decreasing systolic blood pressure category remained robust after thorough adjustment (including eGFR and BMI) and was consistent across sex and heart failure subtypes (heart failure with reduced or preserved EF).

From a practical standpoint, our findings underscore the importance of paying close attention to systolic blood pressure trajectories in patients hospitalised for acute heart failure. Persistently low or high systolic blood pressure may reflect more severe or advanced heart failure, per-

sistent congestion or inadequate response to therapies, highlighting the need for closer monitoring and tailored management strategies. Further research is warranted to assess the effectiveness of incorporating in-hospital systolic blood pressure trajectories into clinical decision-making and its potential impact on patient outcomes.

Limitations

Our study has several potential limitations. First, as an observational study, causality cannot be established, and our findings should be interpreted as hypothesis-generating. Although we adjusted for multiple covariates, residual bias not accounted for in the analysis may still be present. Second, BP measurements were based on single readings, which are subject to variability; however, the reported values reflect standard clinical practice in hospitalised settings, where single measurements are often used. Third, only systolic blood pressure values at admission and discharge were available, limiting the ability to perform a more detailed longitudinal analysis of systolic blood pressure trajectories. Fourth, body weight at discharge was not systematically available, limiting the ability to assess decongestion using weight change as a surrogate during hospitalisation. Finally, the study spanned the period from 2005 to 2020 and included two centres from different countries, introducing the possibility of temporal and spatial heterogeneity in patient management and treatment practices.

Conclusions

In a real-world, multinational cohort of 1490 acute heart failure patients, a decline in systolic blood pressure during hospitalisation was independently associated with improved outcomes in those with elevated systolic blood pressure at admission, underscoring the potential prognostic significance of systolic blood pressure trajectories during the acute management of acute heart failure. This observation may, in part, be driven by the higher rates of initiation of heart failure prognostic medications in this group during hospitalisation. Further research is needed to explore the underlying pathophysiological mechanisms behind these findings and their practical implications.

Data sharing statement

Deidentified data may be made available from the corresponding author upon reasonable request.

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Potential competing interests

All authors have completed and submitted the International Committee of Medical Journal Editors form for disclosure of potential conflicts of interest. Dr Lu has received research grant support or served on advisory boards for Bayer, AstraZeneca, Boehringer Ingelheim, Cytokinetics and Abbott. The other authors do not have any disclosures to report in relation to this article.

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Appendix

Table S1: Event numbers, event rates, and Hazard Ratios associated with categories of systolic blood pressure at admission. All analyses are stratified by country (Switzerland or Kyrgyzstan).

First heart failure hospitalisation or all-cause death	Events, n (%)	Event rate (95% CI), per 100 patient-years	Unadjusted HR (95% CI)	Adjusted HR (95% CI), model*
SBPa <120 mmHg (n = 392)	177 (45.2)	70.9 (61.2–82.2)	1.20 (0.97–1.47)	1.19 (0.96–1.47)
SBPa 120–139 mmHg (n = 442)	184 (41.6)	60.3 (52.2–69.7)	1 (reference)	1 (reference)
SBPa 140–159 mmHg (n = 355)	139 (39.2)	54.1 (45.8–63.8)	0.89 (0.72–1.12)	0.88 (0.71–1.11)
SBPa ≥160 mmHg (n = 301)	121 (40.2)	56.5 (47.3–67.5)	0.93 (0.74–1.18)	1.00 (0.79–1.27)

*Adjusted for age, sex, BMI, prior myocardial infarction, atrial fibrillation or flutter, prior heart failure hospitalisation, hypertension, diabetes, left ventricular ejection fraction, estimated glomerular filtration rate, beta-blockers, ACE inhibitor, angiotensin receptor blocker, mineralocorticoid receptor antagonist.
CI = confidence interval; HR = Hazard Ratio; SBPa = systolic blood pressure at admission

Table S2: Event numbers, event rates, and Hazard Ratios associated with categories of systolic blood pressure at discharge. All analyses are stratified by country (Switzerland or Kyrgyzstan). Median follow-up time: 1 year.

First heart failure hospitalisation or all-cause death	Events, n (%)	Event rate (95% CI), per 100 patient-years	Unadjusted HR (95% CI)	Adjusted HR (95% CI), Model*
SBPd <120 mmHg (n = 629)	258 (41.0)	61.2 (54.2–69.1)	1.12 (0.93–1.34)	1.27 (1.05–1.53)
SBPd 120–139 mmHg (n = 548)	221 (40.3)	55.4 (48.6–63.2)	1 (reference)	1 (reference)
SBPd 140–159 mmHg (n = 243)	114 (46.9)	72.3 (60.2–86.9)	1.22 (0.97–1.53)	1.22 (0.97–1.54)
SBPd ≥160 mmHg (n = 70)	28 (40.0)	58.6 (40.4–84.8)	0.99 (0.67–1.47)	1.07 (0.71–1.60)

CI = confidence interval; HR = Hazard Ratio; SBPa = systolic blood pressure at admission