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Impact of Early Blood Pressure Reduction on Prognosis in ICU Patients with Severe Acute Hypertension: A Retrospective Study of the Medical Information Mart for Intensive Care IV (MIMIC-IV) Database

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Abstract

Severe acute elevations in blood pressure (BP) are common among intensive care unit (ICU) patients and are associated with adverse clinical outcomes. This study investigated the association between early BP dynamics following severe acute hypertension and 30-day mortality. In this retrospective cohort study using the Medical Information Mart for Intensive Care IV (MIMIC-IV) database, we included 4 895 ICU patients with severe acute hypertension, defined as at least one systolic blood pressure (SBP) measurement ≥ 180 mmHg. Restricted cubic spline (RCS) models were used to characterize the associations between 30-day mortality and four BP parameters measured 1 hour after the index episode: achieved systolic and diastolic BP (SBP-1h and DBP-1h) and their corresponding percent reductions (SBP-PR and DBP-PR). Survival analyses were conducted across predefined BP strata. The greatest reduction in BP occurred within the first hour after the index hypertensive episode. RCS analyses demonstrated U-shaped associations between 30-day mortality and both SBP-1h and SBP-PR, with nadirs at 156.76 mmHg for SBP-1h and 14.87% for SBP-PR. In multivariable Cox regression models, an SBP-1h of 140–180 mmHg or an SBP-PR of 5%-25% was associated with a significant survival benefit. Conversely, a DBP-1h < 70 mmHg or a DBP-PR > 27% was associated with an increased risk of mortality. Among ICU patients with severe acute hypertension, maintaining BP within an optimal range during the early treatment period is associated with improved prognosis. Excessive reductions in either SBP or DBP are associated with increased 30-day mortality, highlighting the importance of avoiding overaggressive BP lowering in this population.

INTRODUCTION

Marked and abrupt elevations in blood pressure (BP) are common and often unpredictable in hospitalized patients[1], and are associated with adverse clinical outcomes, particularly increased short-term mortality[2, 3]. This concern is especially relevant in the intensive care unit (ICU), where patients are hemodynamically unstable and frequently experience rapid BP fluctuations. Although substantial evidence and well-established guidelines support long-term BP targets for chronic hypertension[4–6], considerably less attention has been directed toward optimal short-term BP control in the acute setting.

In acute care, timely BP reduction is essential to prevent target organ damage (TOD), minimize the risk of hypertensive rebound, and allow safe clinical monitoring and therapeutic adjustment. For critically ill ICU patients, BP variability over short intervals is common, and determining an appropriate target range may be more clinically consequential than the selection of a specific antihypertensive agent[5, 7]. Moreover, the complexity of critical illness and the high burden of comorbidities necessitate individualized BP management strategies tailored to diverse clinical scenarios[8].

Previous studies have primarily examined BP elevations in postoperative states, intracranial hemorrhage, or acute ischemic stroke[9–11]. However, a universally applicable BP target for asymptomatic but markedly elevated BP in the ICU has not been established. Most available evidence supports gradual BP reduction under close monitoring[12, 13]. Current guidelines provide time-dependent BP targets, ranging from 1-hour

to 24-48-hour intervals, for hypertensive emergencies (HE), along with distinct recommendations for BP management in acute ischemic stroke. Nevertheless, these recommendations are not readily generalizable to critically ill ICU patients without overt acute TOD[4].

Importantly, guideline recommendations employ heterogeneous approaches, incorporating both percentage-based BP reductions and absolute BP thresholds, with a primary emphasis on systolic BP (SBP)In contrast, clinical studies often stratify patients according to achieved post-treatment BP values. This inconsistency highlights the need to clarify optimal BP targets in the early treatment phase. Specifically, it remains essential to define both the appropriate absolute BP levels and the magnitude of BP reduction within the critical first hour following severe acute hypertension.

METHODS

Data source and study population

Data were obtained from version 3.1 of the Medical Information Mart for Intensive Care IV (MIMIC-IV; https://mimic.mit.edu/), a large, publicly available, de-identified critical care database containing records from 94 458 ICU admissions between 2 008 and 2 022. Use of the database has been reviewed and approved by the Institutional Review Board of Beth Israel Deaconess Medical Center[14]. One author (certification ID: 665 79692) was granted credentialed access and performed the data extraction. We excluded ICU readmissions (*n* = 29 117), patients aged <18 or >100 years (*n* = 0), and ICU length of stay (LOS) < 24 hours (*n* = 13 362). Among the remaining 51 979 unique ICU admissions, all recorded BP measurements during the ICU stay were screened. A total of 6 713 patients experienced at least one episode of severe acute hypertension, defined as an index systolic blood pressure (SBP-index) ≥180 mmHg with at least one subsequent BP measurement documented within 1 hour. To reduce clinical heterogeneity, we further excluded patients with compelling indications for condition-specific BP management according to current guidelines, including hypertensive emergencies, pregnancy, intracranial hemorrhage, acute ischemic stroke, aortic dissection, and pheochromocytoma crisis. After applying these criteria, 4 895 patients comprised the final study cohort. The detailed selection process is shown in Figure 1. The study was conducted in accordance with the Declaration of Helsinki. Given the fully de-identified nature of the MIMIC-IV database, the requirement for informed consent was waived.

Data extraction and definitions

Baseline data were extracted using PostgreSQL (version 17.1.12). Retrieved variables included demographic characteristics, laboratory results, ICU severity scores, medication exposure, comorbidities, surgical history, and survival outcomes. For variables with <10% missing data, multiple imputation by chained equations was used to address missingness. All longitudinal BP measurements and their corresponding timestamps throughout the ICU stay were extracted to characterize BP trajectories. Compelling indications requiring specialized BP management, as well as baseline comorbidities, were identified using International Classification of Diseases, Ninth and Tenth Revision codes (Table S1).

BP data processing

BP measurements and timestamps were processed using Python (version 3.10.11). Each occurrence of SBP ≥180 mmHg was identified as an episode of severe acute hypertension. The index BP was defined as the earliest recorded BP at the onset of a qualifying episode (SBP-index and DBP-index). The achieved BP was defined as the last BP measurement recorded within the subsequent 1-hour window (SBP-1h and DBP-1h).

If no follow-up BP measurement was available within 1 hour of the initial episode, subsequent qualifying episodes were sequentially evaluated until an eligible episode with adequate follow-up data was identified.

The percentage reduction in BP was calculated as follows:

SBP-PR = [(SBP-index - SBP-1h) / SBP-index] × 100%

DBP-PR = [[(DBP-index - DBP-1h) / DBP-index] × 100%

Study endpoints

The primary endpoint was 30-day all-cause mortality. Follow-up began at the time of the index episode of severe acute hypertension.

Secondary endpoints included in-hospital acute clinical events indicative of TOD: Neurological dysfunction is defined as coma, indicated by a Glasgow Coma Scale score <915 .Renal dysfunction is defined as Stage 3 acute kidney injury (AKI) according to the Kidney Disease: Improving Global Outcomes criteria16.

Statistical analysis

All statistical analyses were conducted using R (version 4.5.0). Baseline characteristics were compared between 30-day survivors and non-survivors. Categorical variables are presented as counts and percentages and were compared using the chi-square test or Fisher's exact test, as appropriate. Continuous variables are expressed as medians with interquartile ranges and were compared using the Student's t test or Wilcoxon rank-sum test, depending on data distribution. Restricted cubic spline (RCS) models were used to assess and visualize potential non-linear associations between BP control parameters and 30-day all-cause mortality, with BP variables modeled as continuous measures. For SBP parameters, survival differences across clinically relevant strata were evaluated using Kaplan-Meier curves and compared with the log-rank test. Cox proportional hazards models were applied to estimate hazard ratios (HRs). For diastolic blood pressure (DBP) parameters, ROC curve analysis was performed to identify optimal thresholds for risk stratification, followed by survival analyses based on these cutoffs. Multivariable logistic regression models were used to examine the associations between BP control levels and secondary endpoints, including Stage 3 AKI and coma. Because the date of death in the

database is recorded in days, whereas the onset of severe acute hypertension is timestamped in hours, a small number of patients who died on the same calendar day as the index episode had calculated survival times of zero or slightly negative values. To meet the non-negative time requirement for survival analysis, these survival times were reassigned to 0.1 days in accordance with standard statistical practice. Cox regression results are reported as hazard ratios (HRs), and logistic regression results as odds ratios (ORs), both with 95% confidence intervals (CIs). A two-sided *P* value < 0.05 was considered statistically significant. Multivariable models were specified as follows:

Model 0: Unadjusted.

Model 1: Adjusted for sex, age, and ethnicity.

Model 2: Adjusted for Model 1 covariates plus comorbidities (myocardial infarction, heart failure, diabetes, renal disease, cerebrovascular disease, malignant tumor, peripheral vascular disease, chronic obstructive pulmonary disease (COPD), severe liver disease, rheumatic disease, pre-existing hypertension, and prior hypertension-related TOD), as well as sepsis, delirium, SBP-index, use of vasoactive agents, intravenous antihypertensive therapy, and timing of severe acute hypertension onset.

Unless otherwise specified, all reported results are based on Model 2.

RESULTS

Baseline characteristics and BP dynamics

During the 30-day follow-up period, 749 deaths occurred in the study cohort (364 females and 385 males). Compared with survivors, non-survivors were significantly older (73.39 vs. 67.15 years, *P* < 0.001) and had a higher burden of comorbidities, including myocardial infarction (18.83% vs. 13.84%, *P* < 0.001), congestive heart failure (28.97% vs. 23.32%, *P* = 0.001), cerebrovascular disease (21.09% vs. 14.47%, *P* < 0.001), COPD (28.70% vs. 25.08%, *P* = 0.04), liver disease (6.68% vs. 4.20%, *P* = 0.004), and malignant cancer (18.83% vs. 12.47%, *P* < 0.001).

During the ICU stay, non-survivors were more likely to develop Stage 3 AKI (46.06% vs. 23.56%, *P* < 0.001), sepsis (80.37% vs. 56.87%, *P* < 0.001), coma (17.49% vs. 7.86%, *P* < 0.001), and delirium (43.66% vs. 37.39%, *P* = 0.001). Accordingly, they required more intensive organ support, including continuous renal replacement therapy (15.35% vs. 6.08%, *P* < 0.001), mechanical ventilation (87.32% vs. 82.63%, *P* = 0.001), and vasoactive agents (54.07% vs. 31.21%, *P* < 0.001).

Notably, intravenous antihypertensive therapy was administered less frequently in non-survivors (52.60% vs. 67.75%, *P* < 0.001), and pre-existing hypertension was less prevalent in this group (72.36% vs. 79.88%, *P* < 0.001).

Regarding BP profiles, baseline SBP (131 vs. 141 mmHg, *P* < 0.001) and baseline DBP (69 vs. 72 mmHg, *P* < 0.001) were significantly lower among non-survivors. Similarly, mean SBP (129.22 vs. 136.90

mmHg, *P* < 0.001) and mean DBP (62.29 vs. 67.84 mmHg, *P* < 0.001) during the ICU stay were lower in the mortality group. In contrast, maximum SBP was slightly higher in non-survivors (196 vs. 192 mmHg, *P* < 0.001).

The onset of severe acute hypertension occurred significantly later in non-survivors compared with survivors (80.93 hours vs. 40.27 hours from ICU admission, *P* < 0.001). At the index episode, SBP was marginally higher (187 vs. 186 mmHg, *P* = 0.004), whereas DBP was slightly lower (86 vs. 87 mmHg, *P* = 0.002) in patients who died. One hour after the episode, achieved BP levels were consistently lower in non-survivors (SBP-1h: 147 vs. 155 mmHg; DBP-1h: 67 vs. 73 mmHg; both *P* < 0.001) (Table 1; Table S2).

The most pronounced BP reduction occurred within the first hour following the onset of severe acute hypertension. Median SBP decreased from 186 mmHg to 154 mmHg, and median DBP decreased from 87 mmHg to 72 mmHg (Figure 2A-B).

Association between BP Control and 30-day mortality

RCS analyses were performed to characterize the associations between BP control parameters and 30-day mortality. A significant U-shaped relationship was observed between mortality and both SBP-1h (Figure 3A) and SBP-PR (Figure 3C). The nadirs of the spline curves corresponded to an SBP-1h of approximately 156.76 mmHg and an SBP-PR of 14.87%, suggesting these values as potential optimal targets (Table 2).

In contrast, no clear U-shaped association was identified between mortality and either DBP-1h (Figure 3B) or DBP-PR (Figure 3D). Nevertheless, a consistent trend indicated that excessive reductions in DBP were associated with increased mortality risk, particularly at lower achieved DBP levels and higher reduction percentages.

Impact of SBP-1h and SBP-PR on mortality

To further evaluate prognostic implications, patients were stratified according to SBP-1h and SBP-PR categories (Tables 3 and 4). Using SBP-1h ≥180 mmHg as the reference group, patients whose SBP-1h was maintained between 140 and 180 mmHg demonstrated a significant survival advantage (Table S8). The most pronounced benefit was observed in patients with SBP-1h between 150 and 160 mmHg (HR 0.62, 95% CI 0.46-0.82, *P* < 0.001) and between 160 and 170 mmHg (HR 0.62, 95% CI 0.47-0.83, *P* = 0.001).

Similarly, when stratified by reduction magnitude, patients with no SBP reduction (SBP-PR ≤0%) served as the reference group. An SBP-PR between 5% and 25% was associated with a significantly lower risk of 30-day mortality (Table S9). Notably, an SBP-PR of 15-20% conferred the greatest survival benefit (HR 0.56, 95% CI 0.39-0.80, *P* = 0.002). Kaplan-Meier curves further illustrated improved survival among patients whose SBP was controlled within these optimal ranges (Figure 4A-B).

Impact of DBP-1h and DBP-PR on mortality

Patients were also stratified according to DBP-1h and DBP-PR. Compared with patients whose DBP-1h was ≥110 mmHg, those maintained within the 70-80 mmHg range experienced a significant survival benefit (HR 0.64, 95% CI 0.44-0.95, *P* = 0.03) (Tables S6-S7). However, no specific DBP-PR category demonstrated a statistically significant survival advantage in categorical analyses.

To further refine risk stratification, ROC curve analyses were performed (Figure S1; Table S3). The optimal prognostic thresholds were identified as 70 mmHg for DBP-1h and 27% for DBP-PR. Subsequent Kaplan-Meier analyses (Figure 4C-D) showed that patients with DBP-1h <70 mmHg or DBP-PR >27% had significantly higher mortality rates (P < 0.001). These associations were confirmed in multivariable Cox proportional hazards models (Tables S10-S11).

Association between BP targets and secondary endpoints

The effects of SBP control categories on secondary endpoints, coma and Stage 3 AKI, were further examined. RCS analyses demonstrated U-shaped relationships between both SBP-1h and SBP-PR and the incidence of these complications (Figures S2-S3; Table S4).

Multivariable logistic regression models were used to quantify the risks associated with excessive or insufficient BP reduction. Using SBP-1h 140-180 mmHg as the reference category, patients with SBP-1h <140 mmHg had a significantly increased risk of Stage 3 AKI (OR 1.19, 95% CI 1.02-1.40, *P* = 0.03) and a borderline higher risk of coma (OR 1.25, 95% CI 1.00-1.56, *P* = 0.05). Similarly, compared with an SBP-PR of 5%-25%, an SBP-PR >25% was associated with a higher risk of Stage 3 AKI (OR 1.23, 95% CI 1.04-1.45, *P* = 0.01) (Tables S12-S15).

For diastolic parameters, a DBP-1h <70 mmHg was associated with a significantly greater incidence of Stage 3 AKI compared with DBP-1h ≥70 mmHg (OR 1.18, 95% CI 1.02-1.36, *P* = 0.03). However, no significant association was observed between DBP categories and the occurrence of coma (Tables S16-S19). .

Associations between covariates and clinical outcomes

Associations between additional covariates and clinical outcomes are presented in Table S5. The multivariate analysis identified sepsis as a major independent predictor of 30-day mortality (HR 2.03, 95% CI 1.71-2.42, *P* < 0.001). Additionally, onset timing > 24h was associated with an increased risk of death (HR 1.44, 95% CI 1.23-1.68, *P* < 0.001). Conversely, the administration of intravenous antihypertensive therapy appeared to confer a survival benefit, significantly lowering the risk of mortality (HR 0.72, 95% CI 0.61-0.85, *P* < 0.001).

DISCUSSION

In this large real-world cohort of 4 895 critically ill patients from the MIMIC-IV database, we found that achieving BP within an optimal range during the first hour after a severe acute hypertensive episode was associated with a significant reduction in 30-day mortality. In contrast, excessive BP lowering was

associated not only with increased mortality but also with a higher incidence of acute complications, including coma and Stage 3 AKI. These associations remained robust after adjustment for key confounders, including demographic characteristics and comorbid conditions. Importantly, our findings consistently highlight the relevance of both systolic and diastolic parameters. Similar conclusions were obtained whether BP control was assessed using absolute achieved values (e.g., SBP-1h) or percentage reductions (e.g., SBP-PR). The spline analyses identified optimal targets of approximately 156.76 mmHg for SBP-1h and 14.87% for SBP-PR, with clinically meaningful benefit observed within broader ranges of 140–180 mmHg and 5%-25%, respectively. Notably, the adverse associations of excessive SBP reduction with coma and Stage 3 AKI were more pronounced than those observed with insufficient SBP reduction, suggesting the potential harm of overaggressive BP lowering in critically ill patients. With respect to diastolic BP, a DBP-1h < 70 mmHg or a DBP-PR > 27% was associated with increased 30-day mortality. These findings further emphasize the need to avoid excessive diastolic hypotension during early BP management, as diastolic perfusion pressure may be particularly critical for maintaining coronary and end-organ blood flow in vulnerable ICU populations.

The 2 025 guidelines from the American Heart Association recommend that in patients with HE, SBP should be reduced by no more than 25% within the first hour, followed by a target of approximately 160/100 mmHg over the subsequent 2–6 hours[4]. Similarly, the 2 024 guidelines from the European Society of Cardiology advise lowering SBP to < 180 mmHg within the first hour[6]. Although the clinical scenarios addressed in these guidelines differ from the population examined in our study, the numerical targets identified in our analysis are notably consistent with these recommendations. According to the 2 024 American Heart Association Scientific Statement, the patients included in our cohort would be classified as having hypertensive urgency (HU), or asymptomatic markedly elevated inpatient BP[17]. Current guidelines provide limited direction regarding specific BP reduction targets for this population, instead emphasizing the potential harm associated with overly aggressive BP lowering[4]. Despite HU being considerably more prevalent than HE[2], it has received substantially less investigative attention[17, 18]. Our findings help address this gap by providing empirical data to inform BP management in this clinically ambiguous “gray zone.” Moreover, guideline recommendations primarily focus on SBP, with relatively little emphasis on DBP. Some studies circumvent SBP-DBP discrepancies by using mean arterial pressure as a composite measure[19, 20]. In contrast, our study separately evaluated systolic and diastolic parameters and identified clinically relevant DBP thresholds. These results offer a foundation for future prospective studies and randomized controlled trials aimed at refining early BP targets in critically ill patients.

The adverse consequences of abrupt BP elevation are largely mediated by the risk of acute TOD. Compared with HU, HE carries a higher immediate mortality risk because TOD is already present[21, 22]. Nevertheless, both conditions share a common pathophysiological basis: a sudden increase in systemic vascular resistance. This elevation can induce microvascular injury, activate the renin-angiotensin-aldosterone system (RAAS), promote systemic vasoconstriction, and trigger pressure natriuresis with subsequent volume depletion, thereby establishing a feed-forward cycle that further aggravates

hypertension. Although autoregulatory mechanisms in vital organs typically buffer transient BP fluctuations, failure of these mechanisms may result in serious complications such as cerebral edema, intracranial hemorrhage, acute heart failure, and AKI[2, 18, 23–25]. Importantly, overly rapid pharmacologically induced BP reduction may be equally harmful[5, 26]. Prior studies have suggested that intravenous antihypertensive therapy in patients with severe acute hypertension may increase the risk of AKI[3]. In our cohort, the observed associations between excessive BP reduction and higher incidences of coma and Stage 3 AKI support this concern. These findings provide a plausible mechanistic explanation for the increased mortality observed in patients with overaggressive BP control. A key limitation of this study, however, is the inability to definitively determine the temporal sequence between BP elevation episodes and the onset of subsequent clinical events, which precludes firm conclusions regarding causality.

Management strategies for HE, as well as for specific high-risk conditions, including pregnancy-related hypertension, intracranial hemorrhage, acute ischemic stroke, aortic dissection, and pheochromocytoma crisis, have been extensively investigated and are supported by established clinical consensus[8, 28]. These populations differ substantially from the cohort examined in our study and may independently influence secondary outcomes such as AKI or neurological dysfunction. To minimize confounding, these patients were excluded from the present analysis. Just as HE encompasses heterogeneous patterns of organ injury and clinical trajectories[2, 21], HU likely represents a similarly heterogeneous entity. Future studies should therefore aim to refine BP targets according to precipitating factors, underlying etiologies, and specific patterns of TOD.

The critically ill population included in this study possesses distinctive clinical features. Unlike patients in the general ward or community setting, who often present to the emergency department with overt TOD, facilitating the diagnosis of HE and prompting symptom-driven intravenous antihypertensive therapy, the ICU environment allows for continuous and high-resolution hemodynamic monitoring. This enables earlier identification of severe acute hypertension, frequently before overt organ injury develops. However, management in the ICU is complicated by severe comorbid conditions, fluctuating hemodynamics, and often lower baseline MAP[20], making early recognition of progression to HE more challenging when relying solely on integrated clinical signs[29]. Importantly, our findings suggest that the timing of severe acute hypertension onset has prognostic significance. Mortality was substantially higher among patients whose hypertensive episodes occurred more than 24 hours after ICU admission. This temporal pattern resembles that observed with certain postoperative complications, in which delayed onset is often associated with worse outcomes[30]. A comparable analogy can be drawn to the distinction between community-acquired and hospital-acquired pneumonia[31], where timing reflects differences in underlying pathophysiology and risk profile. In severe acute hypertension, delayed onset may indicate a greater contribution from iatrogenic or in-hospital factors. Given that severe acute hypertension is influenced by diverse precipitating factors and prognostic determinants[23], optimal control strategies should be tailored accordingly. These areas represent critical directions for future investigation.

This study provides an overall characterization of intravenous antihypertensive use during episodes of severe acute hypertension. Importantly, our primary objective was to evaluate therapeutic BP targets rather than the specific pharmacologic agents used to achieve them. Although intravenous antihypertensive administration was documented, the precise temporal relationship between drug delivery and the onset of BP elevation could not be definitively established. In addition, we did not perform a direct comparison between oral and intravenous antihypertensive agents. This was partly due to inherent limitations of the database and, more importantly, because such comparisons were beyond the scope of the present investigation.

Numerous clinical studies, including several well-designed randomized controlled trials, have examined pharmacologic strategies for BP management[7, 9–11, 19, 32], particularly in conditions secondary to acute hypertension, such as intracranial hemorrhage[28]. However, substantial inter-patient heterogeneity complicates efforts to simultaneously standardize medication dosing and predefined BP targets. As a result, establishing a direct causal relationship between specific antihypertensive agents and clinical outcomes remains challenging[18]. In routine clinical practice, pharmacologic therapy must be individualized according to each patient's overall condition, comorbidity profile, and hemodynamic status[4, 13, 19]. These complexities highlight important directions for future research.

CONCLUSION

In this cohort of 4 895 critically ill patients with severe acute hypertension from the MIMIC-IV database, maintaining SBP-1h within 140–180 mmHg or achieving an SBP-PR of 5%-25% was associated with significantly improved clinical outcomes compared with persistently uncontrolled SBP. In contrast, an SBP-1h < 140 mmHg, SBP-PR > 25%, DBP-1h < 70 mmHg, or DBP-PR > 27% were independently associated with increased mortality and a higher incidence of adverse events, including coma and Stage 3 AKI. These findings suggest that moderate early BP reduction confers clinical benefit, whereas overly aggressive BP lowering may compromise end-organ perfusion and worsen prognosis in the ICU setting.

Declarations

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AUTHOR CONTRIBUTIONS

Yunqing Li, Weiwei Zhu took part in the study design, performed statistical analyses and drafted the manuscript. Yunqing Li, Yixin He and Weiqi Yang had the access permission to MIMIC-IV 3.0 and extracted data for the present study. Qishan Chen, Mei Yang, Weiguo Zhu and Li Zhang conceived of the study and revised the manuscript critically for important intellectual content.

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AVAILABILITY OF DATA AND MATERIALS

No datasets were generated or analysed during the current study.

Ethics approval and consent to participate

The data in the MIMIC-IV public database have passed the ethical review of the original study, and no further ethical review and informed consent is required.

CONSENT FOR PUBLICATION

All authors have consented to the publication of the paper.

COMPETING INTERESTS

The authors declare no competing interests.

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Tables

Table 1. Baseline characteristics and comorbidities by 30-day survival condition in critically ill patients with severe acute hypertension.

Variable	Overall (*n* = 4895)	Survivors (*n* = 4146)	Non-survivors (*n* = 749)	*P*
Demographics				
Male, *n*(%)	2 704 (55.24)	2 319 (55.93)	385 (51.40)	0.02
Age (year)	68.08 (56.90 - 79.01)	67.15 (56.43 - 78.01)	73.39 (61.73 - 83.00)	<0.001
White, *n*(%)	2 976 (60.80)	2 544 (61.36)	432 (57.68)	0.06
Medical History				
Myocardial infarction, *n*(%)	715 (14.61)	574 (13.84)	141 (18.83)	<0.001
Congestive heart failure, *n*(%)	1 184 (24.19)	967 (23.32)	217 (28.97)	0.001
Peripheral vascular disease, *n*(%)	575 (11.75)	486 (11.72)	89 (11.88)	0.90
Cerebrovascular disease, *n*(%)	758 (15.49)	600 (14.47)	158 (21.09)	<0.001
COPD, *n*(%)	1 255 (25.64)	1 040 (25.08)	215 (28.70)	0.04
Rheumatic disease, *n*(%)	189 (3.86)	153 (3.69)	36 (4.81)	0.15
Diabetes, *n*(%)	1 743 (35.61)	1 515 (36.54)	228 (30.44)	0.001
Renal disease, *n*(%)	1 209 (24.70)	1 025 (24.72)	184 (24.57)	0.96
Severe liver disease, *n*(%)	224 (4.58)	174 (4.20)	50 (6.68)	0.004
Malignant cancer, *n*(%)	658 (13.44)	517 (12.47)	141 (18.83)	<0.001
Pre-existing hypertension, *n*(%)	3 854 (78.73)	3 312 (79.88)	542 (72.36)	<0.001
Hypertensive TOD, *n*(%)	1 450 (29.62)	1 237 (29.84)	213 (28.44)	0.46
In-hospital Complications				
AKI, *n*(%)	3 939 (80.47)	3 263 (78.70)	676 (90.25)	<0.001
Stage 3 AKI, *n*(%)	1 322 (27.01)	977 (23.56)	345 (46.06)	<0.001
Sepsis, *n*(%)	2 960 (60.47)	2 358 (56.87)	602 (80.37)	<0.001
Severe coma, *n*(%)	457 (9.34)	326 (7.86)	131 (17.49)	<0.001
Delirium, *n*(%)	1 877 (38.35)	1 550 (37.39)	327 (43.66)	0.001
BP Parameters				
SBP-baseline (mmHg)	139 (118 - 160)	141 (119 - 161)	131 (110 - 154)	<0.001

Variable	Overall (*n* = 4895)	Survivors (*n* = 4146)	Non-survivors (*n* = 749)	*P*
DBP-baseline (mmHg)	71 (59 - 86)	72 (60 - 87)	69 (57 - 82)	<0.001
Timing of severe acute hypertension onset (hour)	50.66 (6.22 - 100.05)	40.27 (5.08 - 96.56)	80.93 (21 - 122.48)	<0.001
SBP-index (mmHg)	186 (182 - 193)	186 (182 - 192)	187 (182 - 194)	0.004
DBP-index (mmHg)	87 (75 - 101)	87 (75 - 101)	86 (71 - 100)	0.002
SBP-1h (mmHg)	154 (133 - 172)	155 (135 - 172)	147 (121 - 170)	<0.001
DBP-1h (mmHg)	72 (60 - 86)	73 (61 - 87)	67 (55 - 81)	<0.001
In-hospital mean SBP (mmHg)	135.75 (124.48 - 146.15)	136.90 (125.72 - 146.93)	129.22 (117.21 - 140.91)	<0.001
In-hospital mean DBP (mmHg)	67.01 (59.59 - 75.29)	67.84 (60.44 - 76.04)	62.29 (55.75 - 69.90)	<0.001
In-hospital max SBP (mmHg)	193 (185 - 205)	192 (185 - 204)	196 (187 - 210)	<0.001
In-hospital max DBP (mmHg)	117 (101 - 137)	117 (101 - 137)	114 (99 - 138)	0.10
Vital Signs				
Heart rate (bpm)	87 (73 - 102)	86 (73 - 102)	89 (74 - 104)	0.03
Temperature (℃)	36.78 (36.44 - 37.17)	36.78 (36.44 - 37.17)	36.67 (36.33 - 37.06)	<0.001
Respiratory rate (bpm)	19 (16 - 23.75)	19 (15 - 23)	20 (17 - 25)	<0.001
Treatments				
Ventilator use, *n*(%)	4 080 (83.35)	3 426 (82.63)	654 (87.32)	0.001
CRRT, *n*(%)	367 (7.50)	252 (6.08)	115 (15.35)	<0.001
Antihypertensive medications, *n*(%)	4 569 (93.34)	3 895 (93.95)	674 (89.99)	<0.001

Variable	Overall (*n* = 4895)	Survivors (*n* = 4146)	Non-survivors (*n* = 749)	*P*
Intravenous antihypertensive agents, *n*(%)	3 203 (65.43)	2 809 (67.75)	394 (52.60)	<0.001
Vasoactive agents, *n*(%)	1 699 (34.71)	1 294 (31.21)	405 (54.07)	<0.001
Hospital Stay (day)	10.02 (5.74 - 18.05)	10.33 (5.87 - 18.85)	8.86 (4.71 - 14.65)	<0.001

Abbreviations: SBP, Systolic Blood Pressure; SBP-index, Index Systolic Blood Pressure; SBP-1h, Achieved Systolic Blood Pressure at 1 hour; SBP-PR, Systolic Blood Pressure Percentage Reduction; DBP, Diastolic Blood Pressure; DBP-index, Index Diastolic Blood Pressure; DBP-1h, Achieved Diastolic Blood Pressure at 1 hour; DBP-PR, Diastolic Blood Pressure Percentage Reduction; COPD, Chronic Obstructive Pulmonary Disease; TOD, Target Organ Damage; HHD, Hypertensive Heart Disease; HKD, Hypertensive Kidney Disease; AKI, Acute Kidney Injury; CRRT, Continuous Renal Replacement Therapy; RAASi, Renin–Angiotensin–Aldosterone System Inhibitors; CCB, Calcium Channel Blockers; SNP, Sodium Nitroprusside; SCr, Serum Creatinine; WBC, White Blood Cell Count; Hb, Hemoglobin; LOS, Length of Stay.

Table 2. Identification of optimal blood pressure control targets within the first hour for 30-day mortality from Restricted Cubic Spline Analysis.

BP Parameters	Nadir Value
SBP-1h	156.76 mmHg
DBP-1h	71.63 mmHg
SBP-PR	14.87%
DBP-PR	/

Abbreviations: SBP-1h, Achieved Systolic Blood Pressure at 1 hour; SBP-PR, Systolic Blood Pressure Percentage Reduction; DBP, Diastolic Blood Pressure; DBP-1h, Achieved Diastolic Blood Pressure at 1 hour; DBP-PR, Diastolic Blood Pressure Percentage Reduction.

Table 3. Multivariate Cox proportional hazards analysis of achieved systolic blood pressure (SBP-1h) and 30-day mortality.

Model	SBP-1h	*n*	*n* of events	HR (95% CI)	*P*
Model 0	≥ 180 mmHg	710	114	1	Ref.
	170 mmHg - 180 mmHg	654	77	0.71 (0.54, 0.95)	0.02
	160 mmHg - 170 mmHg	738	86	0.71 (0.53, 0.93)	0.01
	150 mmHg - 160 mmHg	644	80	0.75 (0.57, 1.00)	0.05
	140 mmHg - 150 mmHg	577	74	0.79 (0.59, 1.05)	0.11
	130 mmHg - 140 mmHg	511	84	1.03 (0.77, 1.36)	0.86
	120 mmHg - 130 mmHg	327	56	1.08 (0.78, 1.49)	0.64
	< 120 mmHg	734	178	1.61 (1.27, 2.04)	<0.001
Model 1	≥ 180 mmHg	710	114	1	Ref.
	170 mmHg - 180 mmHg	654	77	0.71 (0.54, 0.95)	0.02
	160 mmHg - 170 mmHg	738	86	0.70 (0.53, 0.92)	0.01
	150 mmHg - 160 mmHg	644	80	0.76 (0.57, 1.01)	0.06
	140 mmHg - 150 mmHg	577	74	0.79 (0.59, 1.06)	0.11
	130 mmHg - 140 mmHg	511	84	1.04 (0.78, 1.38)	0.80
	120 mmHg - 130 mmHg	327	56	1.09 (0.79, 1.50)	0.59
	< 120 mmHg	734	178	1.65 (1.30, 2.08)	<0.001
Model 2	≥ 180 mmHg	710	114	1	Ref.
	170 mmHg - 180 mmHg	654	77	0.68 (0.51, 0.91)	0.01
	160 mmHg - 170 mmHg	738	86	0.62 (0.47, 0.83)	0.001
	150 mmHg - 160 mmHg	644	80	0.62 (0.46, 0.82)	<0.001
	140 mmHg - 150 mmHg	577	74	0.66 (0.49, 0.88)	0.01
	130 mmHg - 140 mmHg	511	84	0.77 (0.58, 1.03)	0.08
	120 mmHg - 130 mmHg	327	56	0.78 (0.56, 1.08)	0.13
	< 120 mmHg	734	178	1.05 (0.82, 1.35)	0.68

Relationship between diastolic blood pressure percentage reduction and 30-day mortality, with SBP-1h categorized into eight subgroups based on thresholds of 120mmHg, 130mmHg, 140mmHg, 150mmHg, 160mmHg, 170mmHg, and 180mmHg. The group with SBP-1h ≥ 180mmHg serves as the reference. In model 1, covariates included age, sex and race. In model 2, covariates included age, sex, race, comorbidities (myocardial infarction, heart failure, diabetes, renal disease, cerebrovascular disease, malignant tumor, peripheral vascular disease, chronic pulmonary disease (COPD), severe liver disease,

rheumatic disease, pre-existing hypertension, and prior TOD due to hypertension), sepsis, delirium, SBP-index, the use of vasoactive agents, intravenous antihypertensive therapy, and the timing of severe acute hypertension onset.

Abbreviations: BP, Blood Pressure; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; SBP-index, Index Systolic Blood Pressure; SBP-1h, Achieved Systolic Blood Pressure at 1 hour; HR, Hazard Ratio; CI, Confidence Interval; COPD, Chronic Obstructive Pulmonary Disease; TOD, Target Organ Damage.

Table 4. Multivariate Cox proportional hazards analysis of systolic blood pressure percentage reduction (SBP-RR) and 30-day mortality.

Model	SBP-PR	*n*	*n* of events	HR (95% CI)	*P*
Model 0	≤ 0%	372	55	1	Ref.
	0% - 5%	481	75	1.05 (0.74, 1.49)	0.77
	5% - 10%	676	75	0.73 (0.52, 1.03)	0.08
	10% - 15%	601	81	0.89 (0.63, 1.26)	0.52
	15% - 20%	604	66	0.72 (0.50, 1.03)	0.07
	20% - 25%	476	57	0.80 (0.55, 1.15)	0.23
	25% - 30%	436	72	1.13 (0.80, 1.60)	0.50
	> 30%	1 249	268	1.52 (1.14, 2.03)	0.005
Model 1	≤ 0%	372	55	1	Ref.
	0% - 5%	481	75	1.08 (0.76, 1.53)	0.67
	5% - 10%	676	75	0.72 (0.51, 1.02)	0.07
	10% - 15%	601	81	0.90 (0.64, 1.27)	0.56
	15% - 20%	604	66	0.73 (0.51, 1.04)	0.09
	20% - 25%	476	57	0.80 (0.55, 1.16)	0.24
	25% - 30%	436	72	1.16 (0.81, 1.64)	0.42
	> 30%	1 249	268	1.56 (1.17, 2.09)	0.003
Model 2	≤ 0%	372	55	1	Ref.
	0% - 5%	481	75	0.95 (0.67, 1.34)	0.76
	5% - 10%	676	75	0.60 (0.43, 0.86)	0.005
	10% - 15%	601	81	0.79 (0.56, 1.12)	0.18
	15% - 20%	604	66	0.56 (0.39, 0.80)	0.002
	20% - 25%	476	57	0.63 (0.43, 0.91)	0.02
	25% - 30%	436	72	0.82 (0.57, 1.17)	0.26
	> 30%	1 249	268	0.96 (0.71, 1.31)	0.80

Relationship between SBP-PR and 30-day mortality, with SBP-PR categorized into eight subgroups based on thresholds of 0, 5%, 10%, 15%, 20%, 25%, and 30%. The group with SBP-PR ≤ 0% serves as the reference. In model 1, covariates included age, sex and race. In model 2, covariates included age, sex, race, comorbidities (myocardial infarction, heart failure, diabetes, renal disease, cerebrovascular disease, malignant tumor, peripheral vascular disease, chronic pulmonary disease (COPD), severe liver disease, rheumatic disease, pre-existing hypertension, and prior TOD due to hypertension), sepsis, delirium, SBP-

index, the use of vasoactive agents, intravenous antihypertensive therapy, and the timing of severe acute hypertension onset.

Abbreviations: BP, Blood Pressure; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; SBP-index, Index Systolic Blood Pressure; RCS, Restricted Cubic Splines; DBP, Diastolic Blood Pressure; SBP-PR, Systolic Blood Pressure Percentage Reduction; AKI, Acute Kidney Injury; HR, Hazard Ratio; CI, Confidence Interval; COPD, Chronic Obstructive Pulmonary Disease; TOD, Target Organ Damage.

Figures

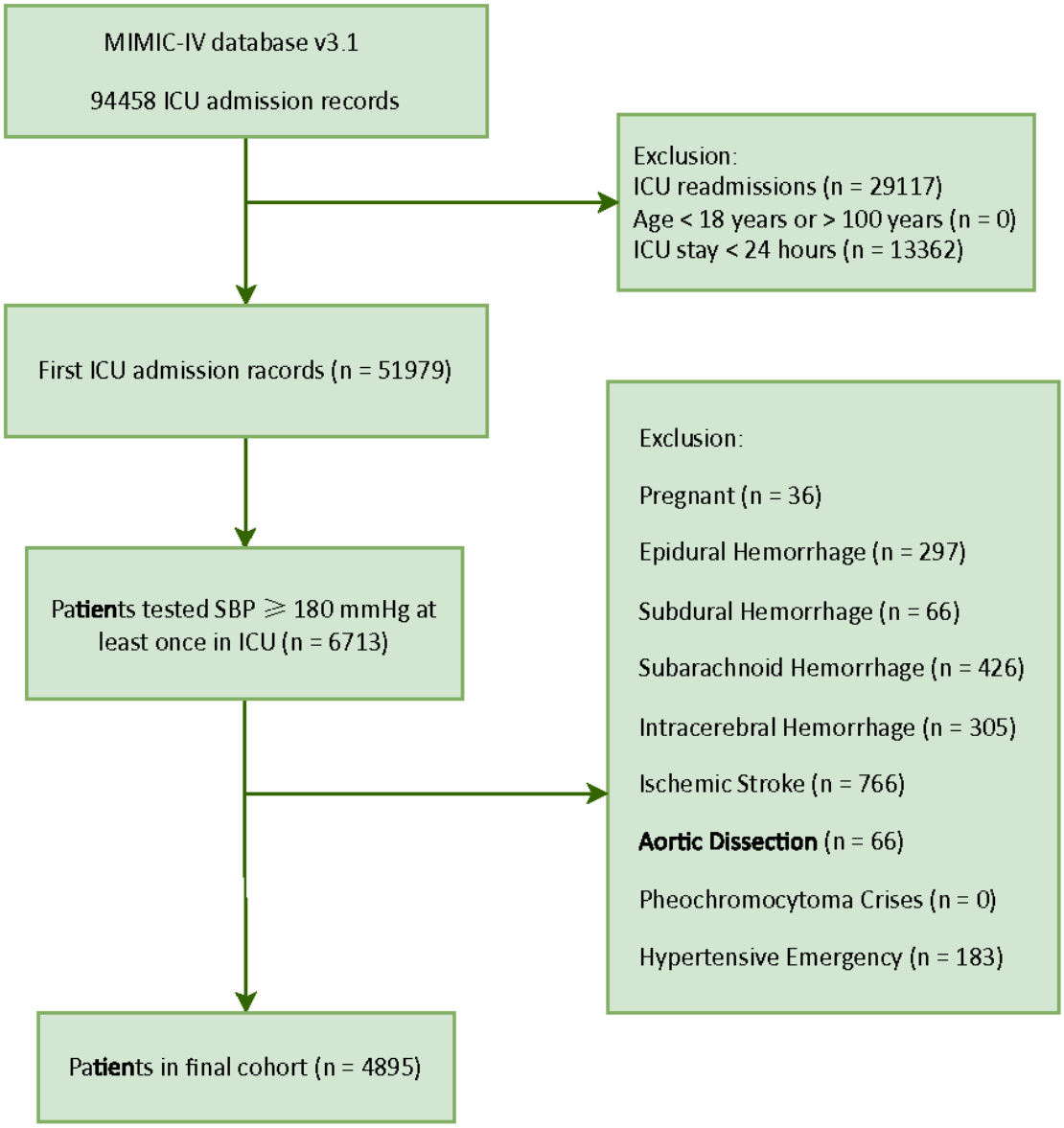


Figure 1

Flowchart of patient inclusion and exclusion from the MIMIC-IV database.

Abbreviations: MIMIC-IV, Medical Information Mart for Intensive Care-IV; ICU, intensive care unit; SBP, systolic blood pressure.

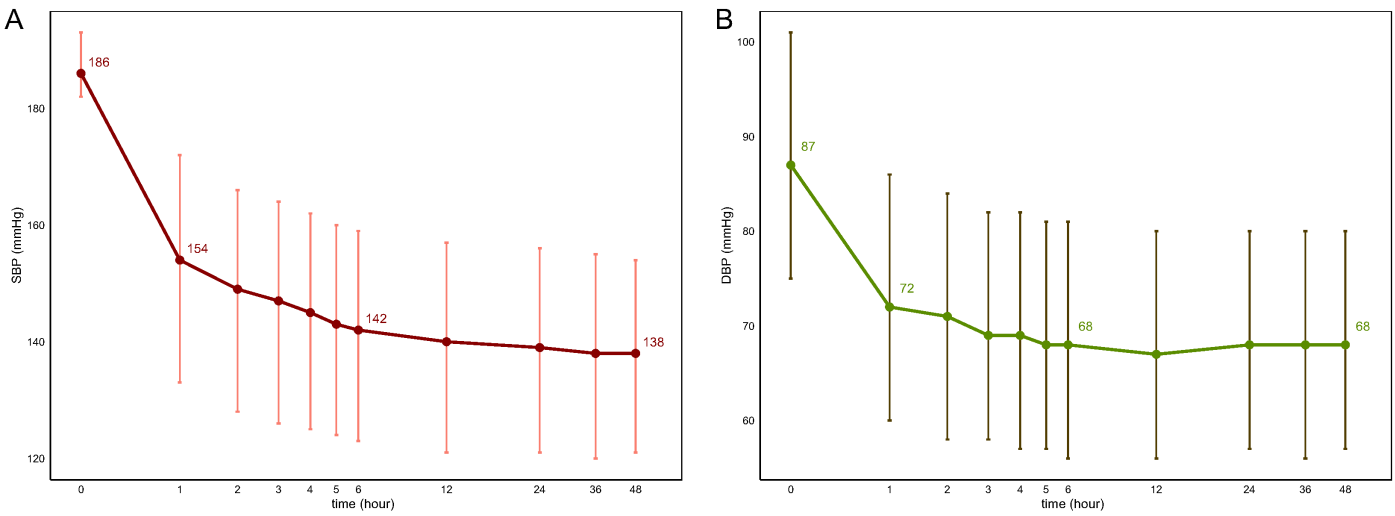


Figure 2

Dynamic changes in SBP and DBP within 48 hours after the initial onset of severe acute hypertension. (A) Trends of SBP over time; (B) Trends of DBP over time. Data points represent medians with interquartile ranges. The numerical values displayed near each marker indicate the median values.

Abbreviations: SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure

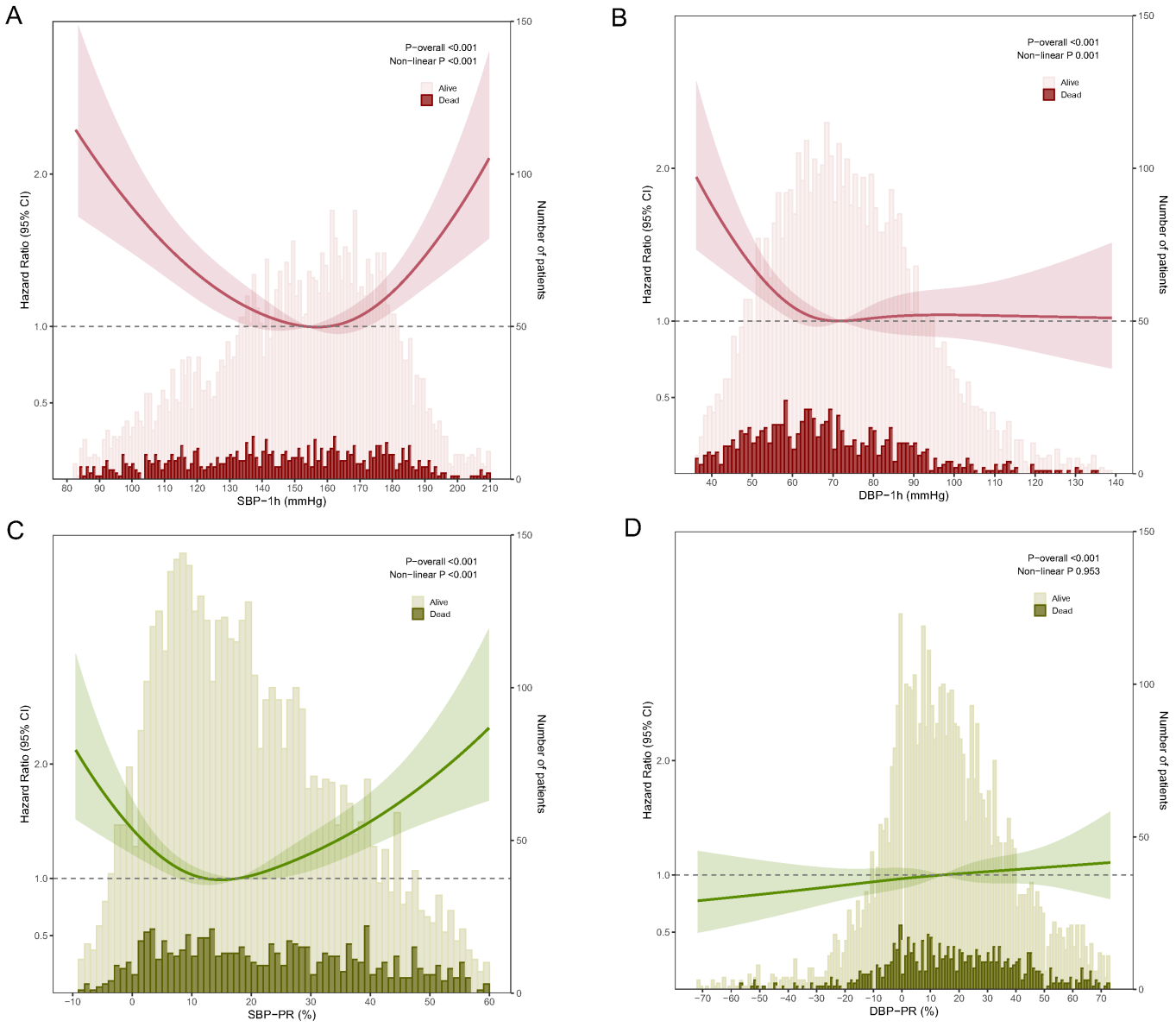


Figure 3

Restricted cubic spline (RCS) curves for blood pressure parameters and 30-day mortality. Non-linear associations between four blood pressure metrics—(A) SBP-1h, (B) DBP-1h, (C) SBP-PR, and (D) DBP-PR—and the risk of 30-day mortality, based on the multivariable-adjusted Cox proportional hazards model (Model 2). Solid lines representing estimated hazard ratios are overlaid on histograms displaying the distribution of the total patient population and mortality events. To ensure statistical stability and visual clarity, the graphical representation is restricted to the 1st to 99th percentile range of each independent variable, minimizing the influence of extreme outliers.

Abbreviations: RCS, Restricted Cubic Splines; DBP, Diastolic Blood Pressure; SBP-1h, Achieved Systolic Blood Pressure at 1 hour; DBP-1h, Achieved Diastolic Blood Pressure at 1 hour; SBP-PR, Systolic Blood

Pressure Percentage Reduction; DBP-PR, Diastolic Blood Pressure Percentage Reduction; HR, Hazard Ratio; CI, Confidence Interval.

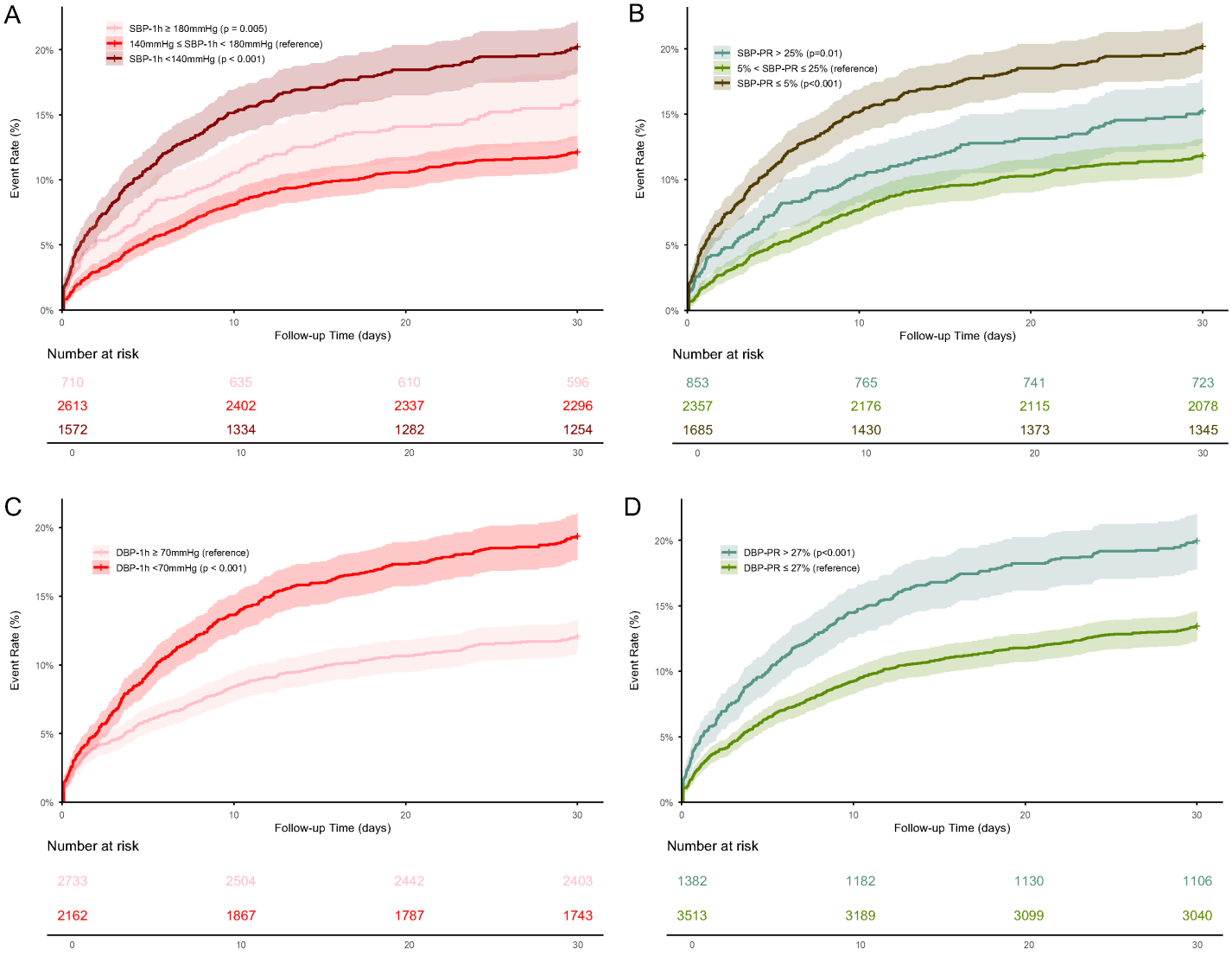


Figure 4

Kaplan-Meier curves stratified by blood pressure control targets.

Kaplan–Meier curves illustrating 30-day mortality probabilities across different blood pressure management categories. Panels (A) and (B) display systolic parameters, with SBP-1h and SBP-PR each stratified into three subgroups based on the primary study thresholds (140/180 mmHg and 5%/25%, respectively). Panels (C) and (D) display diastolic parameters, with DBP-1h and DBP-PR each stratified into two subgroups based on their respective binary thresholds (70 mmHg and 27%). Log-rank tests were performed to compare survival distributions among the groups.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryMaterial.pdf