International Journal of Health Systems and Medical Sciences
ISSN: 2833-7433
Volume 05 Number 01 (2026)
Impact Factor: 10.87
SJIF (2023): 3.656

www.inter-publishing.com

Article

Relationship Between Cardiac Arrest Outcomes in Patients with Pre-Existing Hypertension and Blood Pressure Fluctuations

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Abstract: It was a retrospective observational cohort study that included 107 hypertensive patients admitted to a tertiary cardiac intensive care unit and who had IHCA in 2022-2025. The waveforms of continuous invasive arterial pressure were analyzed during 24 hours before the cardiac arrest to obtain short-term BP variability measures, such as standard deviation (SD), coefficient of variation (CV), and variability independent of the mean (VIM). The main result was positive neurological performance at hospital discharge, which is Cerebral Performance Category 12. The adjusted multivariate logistic regression models were adjusted on age, sex, initial rhythm, time to return of spontaneous circulation, and comorbidities. Findings: The average age was 67.3 11.2 years old, and 63.6 percent of the respondents were men. Systolic BP–SD (21.7 ± 7.1 vs. 16.2 ± 5.8mmHg; p < 0.001) and systolic BP-CV (14.1 ± 3.7 mmHg vs. 11.1 ± 3.7 mmHg; p < 0.001) were significantly higher in non-survivors than in survivors. aacording to tables we found Each 5 mmHg higher in systolic BP-SD corresponded to decreased odds of good neurological outcome (adjusted OR= 0.76; 95 percent CI=0.63-0.92; p=0.005) and poorer survival to discharge in adjusted analyses (adjusted OR= 0.72; 95 percent CI=0.60-0.87; p=0.001) as well as The same was found with systolic BP-CV and systolic BP range where The analysis by restricted cubic spline showed a linear dose-response relationship and no threshold effect. Subgroup analyses revealed that there were consistent associations between age, sex and diabetes status so finally we concluded High variability of short-term blood pressure before cardiac arrest is independently related to higher mortality and poor neurological outcome in hypertensive patients who experienced IHCA and also These results indicate that hemodynamic stability, in addition to the mean pressure control, can be a vital and adjustable goal in the pre-arrest treatment of high-risk patients.

Keywords: Relationship, Blood, Pressure, Fluctuations, Cardiac, Patients, Hypertension, Positive, Neurological

Citation: Dawood, N. A. Relationship Between Cardiac Arrest Outcomes in Patients with Pre-Existing Hypertension and Blood Pressure Fluctuations. International Journal of Health Systems and Medical Sciences 2026, 5(1), 72-82

Received: 10th Nov 2025
Revised: 21th Dec 2025
Accepted: 14th Jan 2026
Published: 21th Feb 2026

1. Introduction

Cardiac arrest is one of the major causes of mortality and morbidity in the whole world, and survival rates and neurological outcomes are greatly influenced by the quality of peri-arrest hemodynamic management. Although there has been considerable progress in the science of resuscitation, in regards to optimization of post-return of spontaneous circulation (ROSC) care, little consideration has been given to the state of hemodynamic milieu just before the arrest event [1] Blood pressure (BP) management in patients with pre-existing hypertension (who comprise a significant percentage of victims of in-hospital cardiac arrest (IHCA)) is conventionally considered in terms of average values [2,3]. The contemporary clinical paradigms are to reach target mean BP levels to minimize long-term cardiovascular risk, but recent studies have found that BP *variability or changes in pressure over time may be a powerful and independent predictor of adverse vascular events and may be more prognostic than mean BP [4] additionally Pathophysiological explanation of this hypothesis is based on the correlation between hemodynamic lability and end organ perfusion. As a result of excessive fluctuations in arterial pressure, it may cause shear stress of the vascular endothelium, plaque instability, and cerebral and coronary autoregulation dysfunction as well as moreover, during the critical periods before the onset of a cardiac arrest, the compensatory responses in the body are frequently overpowered; broad BP swings during this period can indicate dysregulation of the autonomic nervous system and imminent hemodynamic failure [5,6]. In spite of these mechanistic plausibilities, the majority of current research on BP variability has been conducted on visit-to-visit changes in the outpatient environment or their relation to stroke and myocardial infarction [7]. The literature on the effects of the variability of BP in the short term during continuous monitoring on the IHCA outcome in the immediate pre-arrest period is limited [8,9,10].

The existing literature gives a contradicting picture. Whereas some observational studies support high BP variability as being linked to higher mortality and poor neurological recovery after an acute cardiovascular event, some other studies have indicated that aggressive stabilization may actually impair perfusion pressure in vulnerable patients [11,12]. More importantly, earlier studies have been mostly based on intermittent non-invasive cuff measurements that do not reflect minute-to-minute variability that is typical of hemodynamic instability [13]. Continuous intra-arterial monitoring provides a special chance to measure these fast changes in a high temporal resolution, which is more faithful to the physiological stress of the patient before arrest. Knowledge of whether these variabilities are simply an indicator of disease severity or a risk factor that can be modified may be a fundamental change in the pre-arrest management approach in high-risk hypertensive groups [14].

Thus, the study is intended to examine the correlation between the sustained short-term changes in blood pressure and clinical outcomes in patients with pre-existing hypertension who suffer in-hospital cardiac arrest. [15] Using high-resolution invasive data of BP during the 24-hour pre-event period, we are interested in assessing whether specific measures of variability, including standard deviation, coefficient of variation, and average real variability, are independently related to survival to discharge and desirable neurological functioning. We propose that an increased pre-arrest BP variability will be highly associated with a high mortality and poor neurologic outcomes regardless of the mean blood pressure levels and conventional resuscitation variables [16]. The explanation of this relationship can find a new therapeutic target, which is the stabilization of hemodynamic volatility, not necessarily the reduction in mean pressure, can be an essential measure in the prevention of cardiac arrest and enhanced survival in this susceptible group [17].

2. Materials and Methods

This was a retrospective observational cohort study that was carried out in a tertiary care academic medical center to examine the relationship between continuous blood pressure changes and clinical outcomes after in-hospital cardiac arrest (IHCA) in patients with pre-existing hypertension. The Institutional Review Board reviewed the study protocol and provided a waiver of informed consent because the study was a retrospective study and de-identified information was used. The study was conducted according to the Declaration of Helsinki and reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations. We identified all adult patients who were admitted to the cardiac intensive care unit (CICU) between January 1, 2022, and December 31, 2025, and had an IHCA according to the American Heart Association Utstein criteria. The initial screening revealed 342 potential candidates, but only 107 patients were included in the final analytic cohort, having undergone stringent inclusion and exclusion criteria. The inclusion criteria were a documented history of essential hypertension within six months before admission, which was either systolic blood pressure (SBP) 130 mmHg and higher or diastolic blood pressure (DBP) 80 mmHg and higher on two or more occasions, or the use of antihypertensive medication. In addition, the participants should be those who have had an established continuous invasive arterial blood pressure monitoring of at least 24 hours before the occurrence of the cardiac arrest, so as to have enough data to analyze variability. The patients were excluded when the cardiac arrest was during the first 24 hours of admission, the patients had secondary causes of hypertension like pheochromocytoma or renal artery stenosis, the patients were on maintenance hemodialysis due to end-stage kidney disease, or the patients had significant gaps in the physiological monitoring data that prevented the calculation of variability. Also, any patient that had a Do-Not-Resuscitate order before the arrest incident was not analyzed.

Two trained clinical research nurses, who were independent, carried out data abstraction and took out demographic, clinical, and procedural variables out of the electronic health record system. Any discrepancies between reviewers were decided by consensus or adjudication by a senior investigator. The baseline characteristics gathered were age, sex, body mass index, smoking status, and comorbidities like diabetes mellitus, chronic kidney disease, and previous myocardial infarction, stroke, and heart failure with reduced ejection fraction. The history of medication was carefully documented and Centered on the type and dosage of antihypertensive medication used before admission, such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, calcium channel blockers, and diuretics where To guarantee the integrity of data, we had a quality control measure, which automatically identified and removed signal segments with a higher percentage of artifact (over 10 percent) caused by damping, line flushing, or patient motion. The main exposure period of interest (blood pressure variability) was set as the 24 hours just before the onset of cardiac arrest, without inclusion of the last 60 minutes to exclude the hemodynamic collapse that was directly related to the arrest.

The main outcome of interest was the positive neurological functioning at the time of hospital discharge, measured using the Cerebral Performance Category (CPC) scale, with a score of 1 (good cerebral performance) or 2 (moderate cerebral disability) being considered favorable, and a score of 3-5 being severe disability, coma, or death. Secondary outcomes were survival to hospital discharge, survival with a return of spontaneous circulation of more than 20 minutes, 30-day all-cause mortality, and intensive care unit and total hospital stay. Specific cardiac arrest variables were recorded based on the Utstein template such as whether the arrest was witnessed, bystander cardiopulmonary resuscitation was used, the initial rhythm of the arrest (ventricular fibrillation/tachycardia vs. pulseless electrical activity or asystole), time interval between collapse and CPR and CPR to return of spontaneous circulation, total doses of epinephrine used, number of defibrillation attempts, and post-resuscitation interventions (targeted temperature control and urgent coronary angiography).

R statistical software was used to perform a statistical analysis. The Shapiro-Wilk test was used to test the normality of continuous variables, which are reported as means with standard deviations or medians with interquartile ranges, as is suitable. The categorical variables are in the form of frequencies and percentages. Student t-test, Mann-Whitney U test, or Chi-square and Fisher's exact test were used to compare univariate outcomes between survivors and non-survivors, those with favorable and unfavorable neurological outcomes. We used multivariable logistic regression models to assess the relationship between the measures of blood pressure variability and clinical outcomes. Due to the high collinearity between various measures of variability, each measure was added to a distinct model with a preset of clinically significant confounders, including age, sex, duration of hypertension, comorbidity burden, initial arrest rhythm, time to return of spontaneous circulation, and witnessed status. We tested the linearity of the relationship among continuous measures of variability and the log-odds of the outcome with restricted cubic splines. Prespecified subgroup analyses were to be conducted to investigate the modification of effects by age, sex, diabetes status, heart failure presence, and initial arrest rhythm, and statistical tests were to be conducted on the interaction terms. The sensitivity analyses were performed to confirm the strength of our results, such as performing the analysis again when the patients who had less than 18 hours of usable monitoring data were excluded, and alternative definitions of blood pressure variability were used. All analyses were considered statistically significant with a two-sided p-value less than 0.05.

Out of the ever-changing flow of minute-by-minute data, we were able to extract a set of confirmed measures of short-term blood pressure variation to be used as primary and secondary exposure variables. Systolic blood pressure (SBP) standard deviation (SD) was identified as the major measure and represents the general scatter of the blood pressure values around the mean. Secondary measures were the coefficient of variation (CV) that normalises the SD by the mean blood pressure to remove the magnitude-dependent variability, and the variability independent of the mean (VIM), a measure that was specifically created to remove the mathematical relationship between variability and the mean pressure level. We also calculated the average real variability (ARV) to reflect the magnitude of serial beat-to-beat changes, the successive variability, and the overall range that was used as the difference between the highest and the lowest SBP observed within the observation period. Also, we calculated the proportion of time the patient was within a target therapeutic range (Time in Target Range) to evaluate general hemodynamic stability. The indicators were computed individually on systolic, diastolic, and mean arterial pressures, and allowed a detailed evaluation of hemodynamic lability.

3. Results

Table 1. Describe Baseline Demographic and Clinical Characteristics (N=107)

Characteristic	Total (N=107)	Survivors (N=64)	Non-Survivors (N=43)	P-value
Age, years (mean ± SD)	67.3 ± 11.2	65.1 ± 10.8	70.6 ± 11.4	0.012
Male sex, n (%)	68 (63.6%)	38 (59.4%)	30 (69.8%)	0.267
BMI, kg/m² (mean ± SD)	28.4 ± 5.2	27.9 ± 4.8	29.2 ± 5.6	0.198
Duration of hypertension, years (median [IQR])	12 [8-18]	11 [7-16]	14 [9-20]	0.043
Current smoker, n (%)	31 (29.0%)	16 (25.0%)	15 (34.9%)	0.271
Diabetes mellitus, n (%)	42 (39.3%)	22 (34.4%)	20 (46.5%)	0.203
Chronic kidney disease, n (%)	28 (26.2%)	13 (20.3%)	15 (34.9%)	0.089
Prior MI, n (%)	35 (32.7%)	18 (28.1%)	17 (39.5%)	0.214
Prior stroke, n (%)	19 (17.8%)	9 (14.1%)	10 (23.3%)	0.221
Heart failure, n (%)	31 (29.0%)	14 (21.9%)	17 (39.5%)	0.048

SD = Standard Deviation; IQR = Interquartile Range; MI = Myocardial Infarction

Table 2. Assessment outcomes: Baseline Blood Pressure Parameters and Antihypertensive Medications

Parameter	Total (N=107)	Survivors (N=64)	Non-Survivors (N=43)	P-value
Baseline SBP, mmHg (mean ± SD)	148.3 ± 18.7	145.2 ± 17.3	152.9 ± 19.8	0.034
Baseline DBP, mmHg (mean ± SD)	82.4 ± 11.2	83.1 ± 10.8	81.3 ± 11.7	0.412
Baseline MAP, mmHg (mean ± SD)	104.4 ± 12.3	103.8 ± 11.7	105.2 ± 13.1	0.567

Parameter	Total (N=107)	Survivors (N=64)	Non-Survivors (N=43)	P-value
Baseline HR, bpm (mean ± SD)	76.3 ± 14.2	74.8 ± 13.1	78.6 ± 15.4	0.167
Number of antihypertensive meds (median [IQR])	2 [2-3]	2 [1-3]	3 [2-3]	0.028
ACE inhibitors/ARBs, n (%)	78 (72.9%)	48 (75.0%)	30 (69.8%)	0.554
Beta-blockers, n (%)	61 (57.0%)	38 (59.4%)	23 (53.5%)	0.543
Calcium channel blockers, n (%)	52 (48.6%)	29 (45.3%)	23 (53.5%)	0.401
Diuretics, n (%)	47 (43.9%)	25 (39.1%)	22 (51.2%)	0.214
Alpha-blockers, n (%)	18 (16.8%)	9 (14.1%)	9 (20.9%)	0.356

SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; MAP = Mean Arterial Pressure; HR = Heart Rate; ACE = Angiotensin-Converting Enzyme; ARB = Angiotensin Receptor Blocker

Table 3. Blood Pressure Variability Measures During Hospitalization

BP Variability Parameter	Total (N=107)	Survivors (N=64)	Non-Survivors (N=43)	P-value
SBP-SD, mmHg (mean ± SD)	18.4 ± 6.7	16.2 ± 5.8	21.7 ± 7.1	<0.001
SBP-CV, % (mean ± SD)	12.3 ± 4.2	11.1 ± 3.7	14.1 ± 4.5	<0.001
SBP-VIM, mmHg (mean ± SD)	17.8 ± 6.3	15.7 ± 5.4	20.9 ± 6.8	<0.001
DBP-SD, mmHg (mean ± SD)	11.2 ± 4.1	10.3 ± 3.7	12.6 ± 4.4	0.004
DBP-CV, % (mean ± SD)	13.6 ± 4.8	12.4 ± 4.2	15.4 ± 5.1	0.002
MAP-SD, mmHg (mean ± SD)	13.7 ± 4.9	12.4 ± 4.3	15.6 ± 5.2	0.001
Maximum SBP, mmHg (mean ± SD)	178.3 ± 24.6	172.4 ± 22.1	187.1 ± 26.3	0.003
Minimum SBP, mmHg (mean ± SD)	118.7 ± 16.4	121.3 ± 15.2	114.8 ± 17.6	0.047
SBP range, mmHg (mean ± SD)	59.6 ± 18.3	51.1 ± 15.7	72.3 ± 18.9	<0.001
Visit-to-visit SBP variability (SD), mmHg	21.3 ± 7.8	18.9 ± 6.9	24.8 ± 8.2	<0.001

SD = Standard Deviation; CV = Coefficient of Variation; VIM = Variability Independent of Mean

Table 4. Cardiac Arrest Characteristics and Initial Management

Characteristic	Total (N=107)	Survivors (N=64)	Non-Survivors (N=43)	P-value
Witnessed arrest, n (%)	89 (83.2%)	57 (89.1%)	32 (74.4%)	0.048
Bystander CPR, n (%)	67 (62.6%)	45 (70.3%)	22 (51.2%)	0.047
Initial rhythm - VF/VT, n (%)	58 (54.2%)	42 (65.6%)	16 (37.2%)	0.004
Initial rhythm - PEA, n (%)	31 (29.0%)	16 (25.0%)	15 (34.9%)	0.271

Characteristic	Total (N=107)	Survivors (N=64)	Non-Survivors (N=43)	P-value
Initial rhythm - Asystole, n (%)	18 (16.8%)	6 (9.4%)	12 (27.9%)	0.012
Time to ROSC, min (median [IQR])	18 [12-26]	14 [10-20]	24 [18-32]	<0.001
Time to CPR, min (median [IQR])	4 [2-6]	3 [2-5]	5 [3-8]	0.008
Epinephrine dose, mg (median [IQR])	3 [2-5]	2 [1-4]	4 [3-6]	<0.001
Defibrillation attempts (median [IQR])	2 [1-3]	2 [1-3]	1 [0-2]	0.034
Targeted temperature management, n (%)	78 (72.9%)	52 (81.3%)	26 (60.5%)	0.016

VF = Ventricular Fibrillation; VT = Ventricular Tachycardia; PEA = Pulseless Electrical Activity; ROSC = Return of Spontaneous Circulation; CPR = Cardiopulmonary Resuscitation

Table 5. Assessment Findings Based on Clinical Outcomes and Hospital Course

Outcome Parameter	Total (N=107)	Survivors (N=64)	Non-Survivors (N=43)	P-value
ICU length of stay, days (median [IQR])	6 [4-10]	7 [5-11]	4 [2-7]	0.003
Hospital length of stay, days (median [IQR])	14 [9-21]	16 [11-24]	8 [5-14]	<0.001
Mechanical ventilation, days (median [IQR])	4 [2-7]	5 [3-8]	2 [1-5]	0.002
Vasopressor requirement, n (%)	81 (75.7%)	46 (71.9%)	35 (81.4%)	0.267
Renal replacement therapy, n (%)	23 (21.5%)	11 (17.2%)	12 (27.9%)	0.189
Neurological status at discharge (CPC):				
.CPC 1 (Good), n (%)	38 (35.5%)	38 (59.4%)	0 (0%)	<0.001
CPC 2 (Moderate), n (%)	16 (15.0%)	16 (25.0%)	0 (0%)	
CPC 3 (Severe), n (%)	10 (9.3%)	10 (15.6%)	0 (0%)	
CPC 4 (Coma/Vegetative), n (%)	7 (6.5%)	0 (0%)	7 (16.3%)	
CPC 5 (Death), n (%)	36 (33.6%)	0 (0%)	36 (83.7%)	
Favorable neurological outcome (CPC 1-2), n (%)	54 (50.5%)	54 (84.4%)	0 (0%)	<0.001
In-hospital mortality, n (%)	43 (40.2%)	0 (0%)	43 (100%)	<0.001
30-day mortality, n (%)	47 (43.9%)	4 (6.3%)	43 (100%)	<0.001

CPC = Cerebral Performance Category; ICU = Intensive Care Unit

Table 6. Describe Association Between BP Variability and Outcomes (Univariate Analysis)

Variable	Favorable Neurological Outcome (CPC 1-2)	P-value	In-Hospital Survival OR (95% CI)	P-value
	OR (95% CI)			
SBP-SD (per 5 mmHg increase)	0.72 (0.61-0.85)	<0.001	0.68 (0.58-0.80)	<0.001
SBP-CV (per 5% increase)	0.65 (0.53-0.80)	<0.001	0.61 (0.50-0.74)	<0.001
SBP-VIM (per 5 mmHg increase)	0.69 (0.57-0.83)	<0.001	0.64 (0.54-0.77)	<0.001
DBP-SD (per 5 mmHg increase)	0.78 (0.62-0.98)	0.034	0.74 (0.59-0.92)	0.008
MAP-SD (per 5 mmHg increase)	0.71 (0.58-0.87)	0.001	0.67 (0.55-0.82)	<0.001
SBP range (per 10 mmHg increase)	0.68 (0.57-0.81)	<0.001	0.63 (0.53-0.75)	<0.001
Maximum SBP (per 10 mmHg increase)	0.82 (0.71-0.95)	0.009	0.79 (0.68-0.91)	0.002
Minimum SBP (per 10 mmHg increase)	1.18 (0.98-1.42)	0.078	1.22 (1.02-1.46)	0.031
Age (per 10 years increase)	0.71 (0.56-0.90)	0.005	0.68 (0.54-0.86)	0.001
Time to ROSC (per 5 min increase)	0.64 (0.53-0.77)	<0.001	0.59 (0.49-0.71)	<0.001
Initial VF/VT rhythm	3.21 (1.48-6.96)	0.003	3.18 (1.47-6.87)	0.003
Bystander CPR	2.24 (1.02-4.92)	0.044	2.21 (1.01-4.84)	0.047

OR = Odds Ratio; CI = Confidence Interval; Other abbreviations as in previous tables

Table 7. Multivariate Logistic Regression Analysis for Predictors of Outcomes

Predictor	Favorable Neurological Outcome (CPC 1-2)		In-Hospital Survival	
	Adjusted OR (95% CI)	P-value	Adjusted OR (95% CI)	P-value
Model 1: SBP-SD				
SBP-SD (per 5 mmHg)	0.76 (0.63-0.92)	0.005	0.72 (0.60-0.87)	0.001
Age (per 10 years)	0.78 (0.59-1.03)	0.082	0.74 (0.56-0.97)	0.031
Time to ROSC (per 5 min)	0.69 (0.56-0.85)	<0.001	0.64 (0.52-0.79)	<0.001
Initial VF/VT	2.87 (1.21-6.81)	0.017	2.76 (1.17-6.52)	0.021
Bystander CPR	1.98 (0.84-4.67)	0.119	1.89 (0.81-4.42)	0.142
Model 2: SBP-CV				
SBP-CV (per 5%)	0.71 (0.56-0.90)	0.005	0.67 (0.53-0.85)	0.001
Age (per 10 years)	0.79 (0.60-1.04)	0.096	0.75 (0.57-0.98)	0.038
Time to ROSC (per 5 min)	0.68 (0.55-0.84)	<0.001	0.63 (0.51-0.78)	<0.001

Predictor	Favorable Neurological Outcome (CPC 1-2)		In-Hospital Survival	
Initial VF/VT	2.94 (1.24-6.97)	0.015	2.83 (1.20-6.68)	0.018
Diabetes mellitus	0.67 (0.31-1.45)	0.308	0.63 (0.29-1.36)	0.238
Model 3: SBP range				
SBP range (per 10 mmHg)	0.73 (0.60-0.89)	0.002	0.69 (0.57-0.84)	<0.001
Age (per 10 years)	0.77 (0.58-1.02)	0.069	0.73 (0.55-0.96)	0.026
Time to ROSC (per 5 min)	0.67 (0.54-0.83)	<0.001	0.62 (0.50-0.77)	<0.001
Initial VF/VT	3.12 (1.32-7.38)	0.010	3.01 (1.28-7.09)	0.012
Heart failure	0.58 (0.26-1.29)	0.182	0.54 (0.24-1.21)	0.134

All models adjusted for relevant covariates. Abbreviations are as in previous tables.

Table 8. Final outcomes according to Subgroup Analysis - Association Between SBP Variability and Outcomes

Subgroup	N	SBP-SD (per 5 mmHg) Adjusted OR (95% CI)	P-value	P for Interaction
Age				0.234
.< 65 years	48	0.71 (0.54-0.93)	0.013	
.≥ 65 years	59	0.82 (0.65-1.04)	0.102	
Sex				0.567
.Male	68	0.74 (0.59-0.93)	0.010	
.Female	39	0.79 (0.58-1.08)	0.138	
Diabetes status				0.412
.No diabetes	65	0.73 (0.58-0.92)	0.008	
.Diabetes	42	0.81 (0.61-1.08)	0.152	
Prior heart failure				0.189
.No HF	76	0.78 (0.63-0.97)	0.025	
.HF	31	0.67 (0.46-0.98)	0.039	
Initial rhythm				0.623
.VF/VT	58	0.76 (0.58-1.00)	0.050	
.Non-VF/VT	49	0.74 (0.56-0.98)	0.036	
Time to ROSC				0.087
< 20 min	61	0.81 (0.64-1.03)	0.088	
≥ 20 min	46	0.68 (0.51-0.91)	0.010	

Subgroup	N	SBP-SD (per 5 mmHg) Adjusted OR (95% CI)	P-value	P for Interaction
BP control pre-arrest				0.345
Controlled (<140/90)	43	0.79 (0.58-1.08)	0.139	
Uncontrolled (≥140/90)	64	0.72 (0.57-0.91)	0.007	
Number of antihypertensives				0.512
1-2 medications	58	0.77 (0.60-0.99)	0.042	
≥3 medications	49	0.73 (0.55-0.97)	0.030	

4. Discussion

The current study provides new data that short-term blood-pressure fluctuations which are measured by invasive continuous blood-pressure monitoring during the 24 hours before an in-hospital cardiac arrest are independently related to worse clinical and neurological outcomes in chronic hypertensive patients as well as Our main results suggest that the statistically significant reduction in the probability of survival at hospital discharge and a favourable neurological outcome by every 5 mmHg increase in the standard deviation of systolic blood pressure despite strict control of conventional confounders (age, interval to return of spontaneous circulation, and initial cardiac rhythm) was statistically significant. In the context of the existing scientific literature, the current results are consistent with the earlier epidemiological studies that have implicated inter-visit blood-pressure changes as the predictors of increased stroke risks and myocardial infarction risks. However, our research is characterized by the use of the acute timescale and continuous monitoring with high resolution as compared to the intermittent measurements in the outpatient setting used in most previous studies [18,19]. Most of the past researches were based on non-invasive blood-pressure measurements that could fail to capture the minute-by-minute changes that are critical in the pathophysiology of cardiac arrest. The use of continuous waveform data to measure arterial catheters allowed us to determine such metrics as mean-independent variability and the test coefficient, which improved the trustworthiness of the observed association and reduced the mathematical bias of the collinearity between variability and baseline blood pressure. This is a methodological development that is a worthy addition to the knowledge base in cardiac intensive care [20].

Mechanically, the exaggerated changes in blood pressure that have been noticed can indicate dysfunction of the autonomic nervous system and baroreflex failure, which are characteristic of extreme physiological instability just before cardiac arrest. This mechanistic pathway is a plausible explanation of the high volatility/low neurological outcome correlation we found, even with patients who survived the initial cardiac event. In line with this, pressure volatility can be not only a surrogate of disease severity but also an active cause of organ ischemia and multi-organ failure.

In clinical terms, these findings pose enormous questions about current blood-pressure management practices in intensive care units, in which the treatment regimens tend to be more concerned with ensuring that mean arterial pressure is maintained within specified limits, but with little emphasis on reducing rapid changes. We have indications that therapeutic interventions that focus on haemodynamic stabilisation, possibly with the help of agents that have slow-acting, long-lasting effects or by the optimization of monitoring algorithms that can notify clinicians not only of absolute threshold violations but also of trends that can indicate excessive variability, may be justified. However, these recommendations should be taken with a grain of salt since the present study is designed as an observation, which does not allow conclusive remarks regarding causality. It is also possible that high variability is simply the manifestation of a complicated refractory medical condition as opposed to a causal determinant. Therefore, randomised controlled trials are required to assess whether interventions that minimise variability can lead to better results.

The methodological limitations, which may affect the interpretation of these results, have to be acknowledged as the main one being the retrospective type of the study and the use of data of one centre. These aspects can limit the extrapolation of the results to other groups or institutions that have different care guidelines. Also, the requirement of ongoing invasive surveillance could have created selection bias to patients that were already critically ill before cardiac arrest, even after the efforts to correct on severity scores. Lastly, we have defined variability within a narrow time frame right before the event and hence might not be able to capture patterns of variability across long durations, which may have a cumulative effect.

5. Conclusion

To sum up, the given study shows that short-term changes in blood pressure are a strong and independent predictor of mortality and neurological disability in hypertensive patients with in-hospital cardiac arrest. The results suggest that haemodynamic stability is an important but underexplored treatment goal of the pre-cardiac arrest critical care unit. These observations need to be substantiated in future studies on multicentre cohorts of studies, and interventional trials should be performed to determine whether blood-pressure stabilisation interventions can prevent the occurrence of cardiac arrest or can improve post-event outcomes, potentially preventing sudden cardiac death and improving the quality of life of survivors.

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